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REPORT

Bleaching, disease and recovery in the threatened scleractinian coral *Acropora palmata* in St. John, US Virgin Islands: 2003–2010

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Abstract A long-term study of the scleractinian coral *Acropora palmata* in the US Virgin Islands (USVI) showed that diseases, particularly white pox, are limiting the recovery of this threatened species. Colonies of *A. palmata* in Haulover Bay, within Virgin Islands National Park, St. John, were examined monthly in situ for signs of disease and other stressors from January 2003 through December 2009. During the study, 89.9 % of the colonies ($n = 69$) exhibited disease, including white pox (87 %), white band (13 %), and unknown (9 %). Monthly disease prevalence ranged from 0 to 57 %, and disease was the most significant cause of complete colony mortality ($n = 17$). A positive correlation was found between water temperature and disease prevalence, but not incidence. Annual average disease prevalence and incidence remained constant during the study. Colonies generally showed an increase in the estimated amount of total living tissue from growth, but 25 (36.2 %) of the colonies died. *Acropora palmata* bleached in the USVI for the first time during the 2005 Caribbean bleaching event. Only one of the 23 colonies that bleached appeared to die directly from bleaching. In 2005, corals that bleached had greater disease prevalence than those that did not bleach. Just over half (52 %) of the colonies incurred some physical damage. Monitoring of fragments (broken branches) that were generated by physical damage through June 2007 showed that 46.1 % died and 28.4 % remained alive; the fragments that attached to the substrate survived longer than those that did not. Recent surveys showed an increase in the total number of colonies within the reef area, formed from both asexual and sexual reproduction. Genotype analysis of 48 of the originally monitored corals indicated that 43 grew from sexual recruits supporting the conclusion that both asexual and sexual reproduction are contributing to an increase in colony density at this site.

Keywords *Acropora palmata* · Population recovery · Coral disease · Coral bleaching · Water temperature

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Introduction

Coral diseases have become increasingly numerous and severe in the Caribbean in the last few decades affecting almost all species with particularly devastating effects on major reef-builders like *Acropora palmata*, *Montastraea annularis*, and *M. faveolata* (Croquer and Weil 2009a; Miller et al. 2009; Weil et al. 2009; Weil and Rogers 2011). Thirty years ago, *A. palmata* reefs helped define the shallow Caribbean seascape. With their complex branching morphology and large size (some reached several meters across), *A. palmata* colonies grew in dense, sometimes interlocking stands on reef crests and in the forereef zones of fringing and barrier reefs. *Acropora palmata* is one of the most important reef-building corals, providing complex

shelter for fish and numerous other species (e.g., Gladfelter 1982; Bruckner 2003). In 2006, this species was listed as threatened in the United States under the Endangered Species Act (Hogarth 2006) recognizing the significant population decline primarily from disease and hurricanes. White band disease, first described by Gladfelter (1982) from the US Virgin Islands (USVI), is thought to have caused extensive losses of *A. palmata* in the late 1970s, 1980s and 1990s throughout the Caribbean (Rogers 1985; Beets et al. 1986; Bythell and Sheppard 1993; Aronson and Precht 2001a, b; Bruckner 2003). In the Virgin Islands, a series of tropical storms and hurricanes in 1979, 1984, and 1989 caused further declines of this important reef-building species. Now many shallow zones in the USVI have "standing dead" *A. palmata* colonies interspersed with piles of storm-generated rubble. Living *A. palmata* colonies currently are growing on skeletons of long-dead colonies and on boulders and pavement near shore.

From 2000 to 2002, informal surveys around both St. John and Buck Island Reef National Monument, off St. Croix, indicated that *A. palmata* was becoming more abundant at least on some reefs, suggesting that recovery might be occurring (Rogers et al. 2003). In 2003, we began a systematic, long-term study to determine the factors that could influence the potential for *A. palmata* recovery in Haulover Bay, St. John (Rogers 2005).

At this time, white pox disease (also referred to as patchy necrosis and white patch) was starting to be recognized as a threat to *A. palmata* populations in Florida and Puerto Rico, but most of the information on this and other diseases came from infrequent and/or short-term surveys (see Bruckner 2003). A primary objective of this study was to conduct frequent (monthly) monitoring to explore the temporal dynamics of diseases, including white band, white pox and undescribed diseases affecting *A. palmata*. Because the study site was within a national park, it provided an opportunity to evaluate the potential for recovery in an area with relatively fewer stressors than other less-protected sites.

During this study, in 2005, sea-surface temperature in the Caribbean reached record highs, with the greatest anomalies in the USVI and Puerto Rico, and severe bleaching occurred (Wilkinson and Souter 2008; Eakin et al. 2010). This year was the hottest in the Northern Hemisphere since the advent of reliable records in 1880 (Wilkinson and Souter 2008). *Acropora palmata* bleached for the first time on record in the USVI (Woody et al. 2008).

The objectives of this long-term study were to determine: (1) the impact of bleaching and disease, (2) the changes in the population size and structure, and (3) the specific impacts of physical damage and fragmentation on the population of *A. palmata* in Haulover Bay, St. John, USVI.

Materials and methods

Study site

Monthly monitoring of individual *A. palmata* colonies was conducted from January 2003 through December 2009 on the fringing reef along the western shore of Haulover Bay, on the northeast coast of St. John, within Virgin Islands National Park (Fig. 1). Over 40 years ago, Kumpf and Randall (1961) stated that the reef in Haulover was "the best example of a typical fringing reef on St. John".

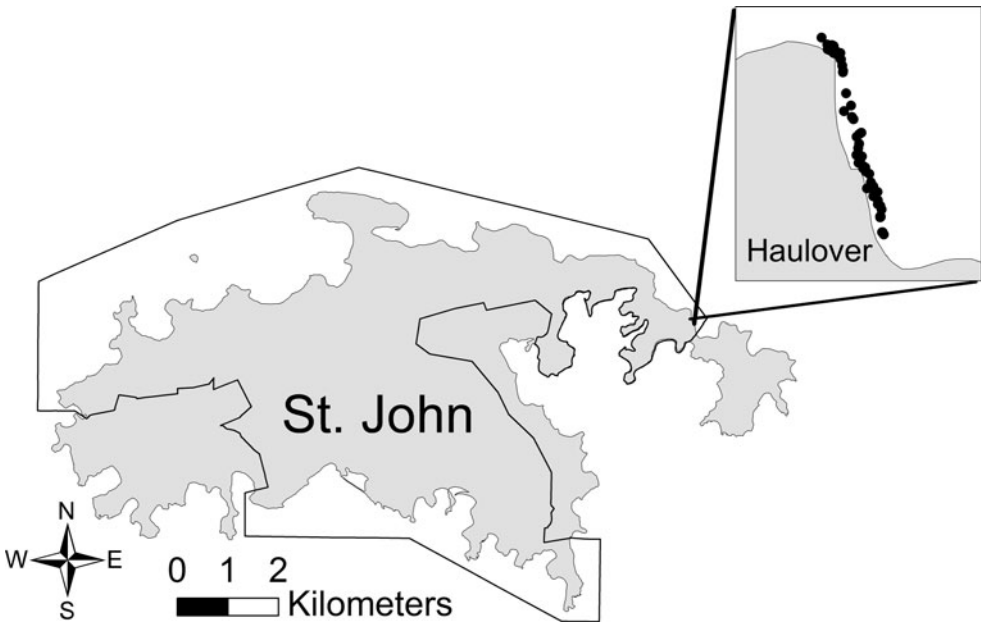

Fig. 1 Image of St. John, USVI, with an *inset* showing Haulover Bay and the location of monitored colonies of *A. palmata* (*black dots*). The *black line* shows the boundary of Virgin Islands National Park

In 1984, Beets et al. (1986) described the upper forereef zone here as containing "the best existing *A. palmata* stands within park boundaries". They estimated a total of 40–60 % living coral cover, with *A. palmata* being the most abundant species. No white band disease was seen, although the presence of standing dead colonies noted in their report suggested earlier presence of disease. The deeper reef zone (to a maximum depth of ca. 15 m) is dominated by *Montastraea annularis* and *M. faveolata*.

The boundary of Virgin Islands National Park passes through Haulover Bay, and the study site is within the park. The watershed directly above the study site is undeveloped although there is a paved road at the top. The site does not appear to be affected by any major local, human-related stressors, but sediments or other contaminants could reach the site from development of residential lots on the eastern shore of the bay (0.3–0.5 km away) or from upstream sources in the British Virgin Islands. Haulover is a popular snorkeling site but receives less recreational use than many other bays around St. John.

Sampling design

On 29 January 2003, an initial survey was conducted to locate every *A. palmata* colony within the shallow reef zone (area = 17,627 m^2) to a depth of about 5 m (the maximum depth of living colonies of this species at this location). A total of 69 colonies growing primarily on dead colonies of *A. palmata* were labeled with numbered plastic tags attached with plastic cable ties to nearby substrate. GPS coordinates were recorded for each colony to facilitate relocation.

Colonies were monitored approximately once a month for over 7 years. Monitoring involved photographing and documenting evidence of predation by snails, fish and fireworms; physical damage (specifically, breakage from humans or natural causes); urchin grazing; sediment abrasion; bleaching; and diseases. Although bleaching is technically a disease, it reflects loss of zooxanthellae and/or zooxanthellar pigments and is distinguished here from the other diseases observed on *A. palmata* in this study that are characterized by initial loss of tissue (Rogers 2010). The criteria used to differentiate among these stressors were consistent with those in Work and Aeby (2006), Williams et al. (2006) and Raymundo et al. (2008). The number of colonies monitored during each survey differed at times because the seas were too rough to locate some corals growing near shore in very shallow water and because some colonies died over time. Snail and fish predation affected ca. 77 % and ca. 60 % of the colonies over the course of the study, but tissue loss was generally minor. Fish bites typically were <1 cm^2 in diameter and usually healed between surveys. Sediment abrasion associated with heavy wave activity was noted near the base of a few colonies. In this paper, we focus on diseases and physical damage, as these stressors caused the most coral mortality.

Each month, disease prevalence was quantified as the proportion of individual colonies showing signs of disease from the entire surveyed population, while disease incidence was recorded as the number of new disease cases observed each month divided by the number of colonies in the population. A new disease case was defined as a coral colony showing signs of recent mortality caused by disease that was not showing these signs the prior month. (Over the course of several years, many of the individual corals repeatedly exhibited new signs of disease, contributing to these incidence calculations.) White band and white pox disease are relatively well-described and seem more distinctive than some of the other "white syndromes", but differentiating among these in the field remains difficult. Identification of several of the coral diseases in the field, and differentiation of these diseases from some of the other stressors such as predation, will continue to be problematic until effective diagnostic tools and better characterizations of the various diseases are available (Raymundo et al. 2008; Rogers 2010). Our own observations over several years contributed to more effective identification of diseases. Because our surveys were so frequent, we were often able to use sequential photographs to help to confirm our identifications. White band disease was recorded when distinctive bands with exposed coral skeleton separating living from dead coral were observed (Gladfelter 1982). The band typically progresses slowly from the base of the colony towards the ends of the branches (Fig. 2a). White pox was recorded when there were lesions (usually irregularly-shaped) that were often completely surrounded by apparently healthy tissue (Fig. 2b). If the gross signs of disease did not closely resemble either white band or white pox, e.g., there were particularly large areas of skeleton exposed by recent tissue loss rather than relatively small exposed patches or bands, the lesions were recorded as "unknown disease" (Fig. 2c).

We were conservative when recording diseases. Some cases of predation by snails or fish might be confused with the "white diseases". Fish predation generally results in scrapes with the removal of corallites or in regular, round patches without skeletal abrasion or damage. Damselfish territories exhibit distinctive "chimneys". Snail predation could perhaps be confused with white band disease if snails are not evident, but white band disease has a relatively uniform band separating live tissue from newly exposed skeleton rather than the more ragged or scalloped edges associated with predation by snails. White band was confirmed when the band of recent mortality advanced evenly over the coral branch for longer than one month.

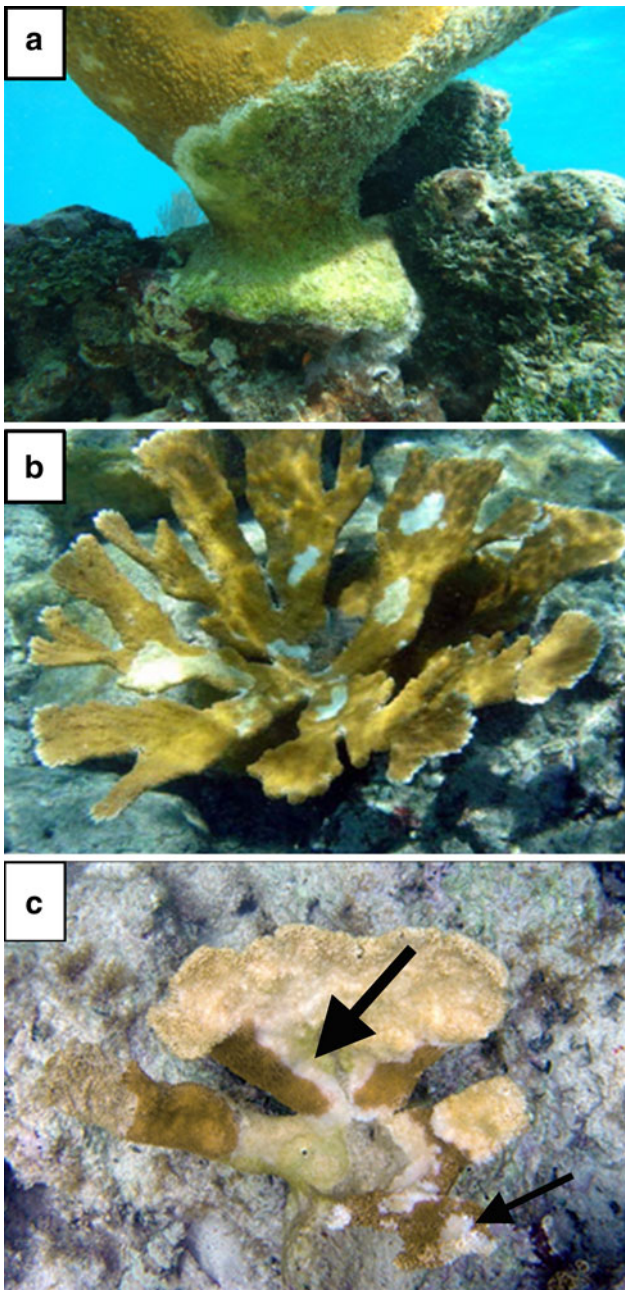

Fig. 2 *A. palmata* colonies showing signs consistent with **a** white band disease, **b** white pox disease, and **c** multiple diseases on a single colony, including undescribed disease (*large black arrow*) on the main part of the colony and white pox disease on the lowest *right* branch (*small black arrow*)

When conducting statistical analyses, and using disease prevalence or disease incidence as the dependent variable, colonies exhibiting white band, white pox and unknown disease were combined. The size of white pox induced lesions was also measured from 2005 to 2006 to compare the size of lesions on bleached and unbleached colonies. Lesions were photographed with a ruler held adjacent to the area of recent mortality and the surface area was calculated using ImageJ software (http://rsbweb.nih.gov/ij/index.html).

Physical damage, primarily broken branches, was also recorded. Some of the damage was associated with snorkelers, some with heavy swells; in a few cases, monofilament fishing line was found entangled in damaged corals. The fate (re-attachment and survival) of coral fragments was followed from February 2003 to June 2007. A fragment was defined as a broken branch found on the substrate, sometimes near a parent or donor colony which was identified from photographs and from the presence of the lesion formed from the loss of the branch. Surveyors recorded whether the fragment was attached to the substrate, and identified any cause of recent mortality (i.e., predation, disease, abrasion). When fragments attached to the substrate they were referred to as 'reattached fragments' to differentiate these corals from original colonies identified at the beginning of the study. Only fragments formed at least 6 months before the end of the fragment study (June 2007) were included in the analysis.

Once a year from 2003 to 2007, the volume of living tissue from each colony was estimated from three measurements (height, width, and length), to determine if the colony size was changing. Four different size classes were recognized (I: $\leq$1,000 cm^3, II: >1,000 and $\leq$15,625 cm^3; III: >15,625 and $\leq$125,000 cm^3, IV: >125,000 cm^3). These measurements were taken again in July 2010. Additionally, the total number of colonies within the same reef area that was surveyed in 2003 was again recorded in 2010 to determine if the number of colonies had increased.

Given the large size and topographical complexity of the study zone, it was not possible to do accurate surveys for new *A. palmata* recruits within the entire area each month. Given the length of this study and the rapid growth rate of this species, any major recruitment event would have been evident. New colonies that were recorded in 2010 (but that did not arise from fragments) were noted and identified as sexual recruits. It can be impossible to differentiate sexual recruits from remnant patches of *A. palmata* growing on partly dead *A. palmata* colonies (Miller et al. 2007). However, when small colonies are found growing on bare rock, as is the case in many shallow nearshore areas around St. John, they are clearly recruits. Furthermore, we were following fragments created from all original colonies so new colonies that became evident were most likely formed from sexual recruitment.

Seawater temperature data were recorded every 2 h using a Hobo thermistor placed midway along the study zone at a depth of approximately 2 m.

Coral genotype and zooxanthellae analysis

Genotypic diversity refers to the number of genetic individuals and is a reflection of "the relative contribution of

sexual and asexual reproduction in a population" (*Acropora* Biological Review Team (2005)—p. 10). In April 2005, small samples of 48 colonies (out of 56 of the original 69 still alive at this time) were placed in seawater in small vials and then returned to the boat where the sea water was replaced with 95 % ethanol at 4 °C. Samples were kept cold until shipped to Dr. Iliana Baums's laboratory for genotype analysis as described in Baums et al. (2005). Portions of these samples were later shipped to Bane Schill (USGS) for the analysis of zooxanthellae clades (Ririe et al. 1997; van Oppen et al. 2005).

Statistical analyses

Cross-correlation analysis was used to determine if there was synchronicity between average monthly water temperature and monthly (1) disease prevalence and (2) disease incidence over the 7 year study (2003–2009). Synchronous peaks (0 monthly time lag) in the cross-correlation analysis with a positive correlation coefficient were considered to indicate positive synchronicity between the two variables. Comparisons between disease prevalence on bleached and unbleached colonies during the 2005 bleaching event were conducted with a Student's *t* test using months as replicates. The assumptions of normality and homoscedasticity were tested and passed using the Shapiro–Wilk test and Levene test, respectively. A Mann–Whitney *U* test was used to compare the average area of disease-induced lesions on bleached and unbleached colonies during the bleaching event.

Results

Mortality from bleaching and disease

White pox disease was the most frequent cause of mortality during the 7 years of monitoring. Over the 84-month long study, 62 (89.9 %) of the colonies exhibited signs of disease. Of those 62 colonies, all had disease signs similar to white pox, while 10 colonies (14.5 %) also showed signs consistent with white band disease. Five colonies showed signs of white band and white pox simultaneously. Fifteen colonies (24.6 %) had signs that were not characteristic of white pox or white band.

Only seven colonies showed no signs of disease during the study. Two of these died (one in 2003, another in 2005) from unknown causes. Two colonies showed no signs of disease, but produced fragments that did. Three colonies which remained alive throughout the study never showed signs of disease and were of distinct genotypes (i.e., were not clones, see below).

Annual average disease prevalence and disease incidence remained relatively constant throughout the study (Fig. 3). Disease prevalence was approximately 12.7 % (±0.1 SE) over the length of the study and disease incidence averaged 7.4 % (±0.7 SE). Seasonal trends in disease prevalence and incidence were observed with two annual peaks, one when water temperature was highest from September to November and another (smaller one) from April to June (Fig. 4).

Disease prevalence ranged from 0 to 57 % throughout the study (Fig. 5). Disease was highest in November 2003 (38 %), October 2004 (50 %), and November 2009 (57 %). The average duration of disease on each colony was approximately 1.7 months, which appeared constant throughout the study. Cross-correlation analysis showed there was significant synchronicity with 0 time lags between disease prevalence and average water temperature (0 time lag: $r = 0.35$, $p < 0.05$; Fig. 5), but no significant synchronicity between disease incidence and average water temperature was observed (cross-correlation coefficients <0.24, $p > 0.05$).

Bleaching was observed only in 2005 and associated with increases in seawater temperature (Fig. 5). Seawater temperature in Haulover ranged from 24.9 °C (Feb. 9, 2005) to 31.4 °C (September 12, 2005). In 2005, the maximum daily temperature exceeded 30 °C on 65 days, including 44 consecutive days. The highest temperatures occurred from August through October 2005 (Fig. 5).

When bleaching was first observed at Haulover in September 2005, 53 of the initial 69 colonies remained. Of the 23 (43 %) of these that bleached, 9 partially died and 2 suffered complete mortality (one from bleaching and one from 'unknown disease'). Twelve of the bleached colonies recovered completely with no signs of mortality throughout the event. Six colonies died during this time (June to December 2005) although they did not appear bleached. Three died from disease, while the other three died from unknown causes. If the colony survived the event then complete pigment recovery occurred by January 2006.

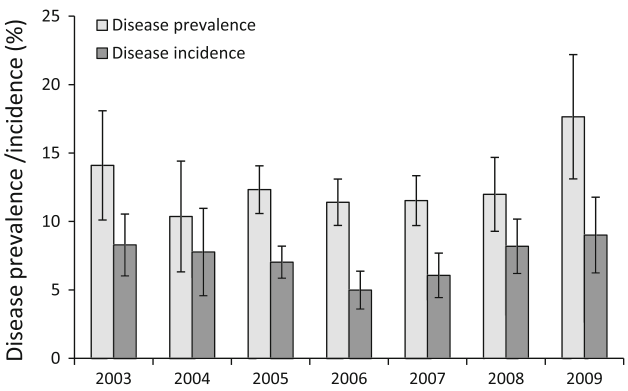

Fig. 3 Annual average disease incidence and prevalence on *A. palmata* colonies in Haulover Bay from 2003 to 2009. *Error bars* denote standard error of the mean

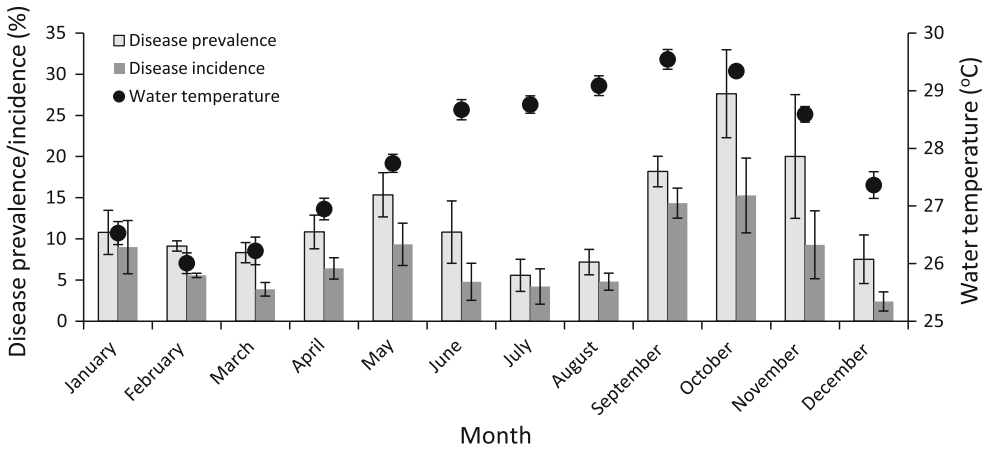

Fig. 4 Monthly average seawater temperature and disease incidence and prevalence on *A. palmata* colonies in Haulover Bay from 2003 to 2009. *Error bars* denote standard error of the mean

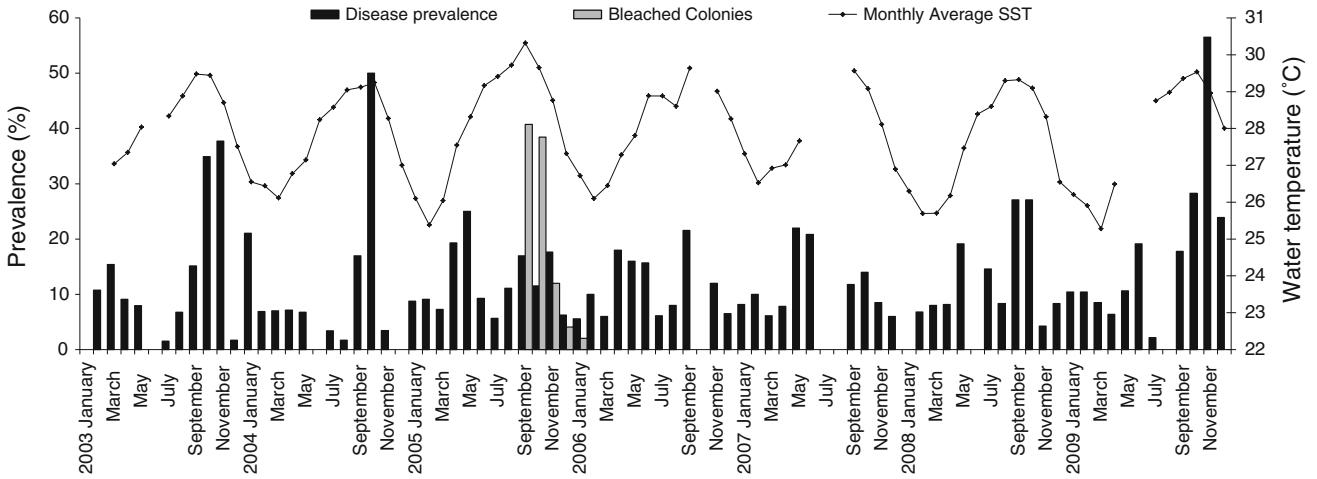

Fig. 5 Prevalence of disease and bleaching throughout the study versus average monthly water temperatures. Gaps in the temperature *line graph* indicate times with missing data

Corals that bleached had greater disease prevalence during and after the bleaching event (from August 2005 to April 2006) than those corals that did not show signs of thermal stress ($t_{(0.05,16)} = 2.578$, $p = 0.02$; Fig. 6). However, a comparison between the average size of white-pox lesions on bleached and unbleached colonies showed no significant difference ($Z_{(0.05,n1\ =\ 9,n2\ =\ 9)} = -1.15$, $p = 0.258$).

By December 2009, 25 (36.2 %) of the colonies had died. Disease was a major contributor to the death of 17 colonies, whereas only one colony died directly from bleaching. Seven colonies died from unknown causes. Of the colonies that died, 12 were in size class I, 10 were in size class II, and 3 were in size class III. The frequency of complete mortality changed over time with the highest number of deaths occurring during 2005, the year of the bleaching event (Fig. 7). Although only one colony died directly from bleaching, 8 out of the 9 colonies that died in 2005 did so during the bleaching event (July to December). The time periods with the greatest loss of entire colonies (primarily from disease) were fall 2003/winter 2004 (10.1 %) and fall 2005/winter 2006 (15.5 %) during the bleaching event.

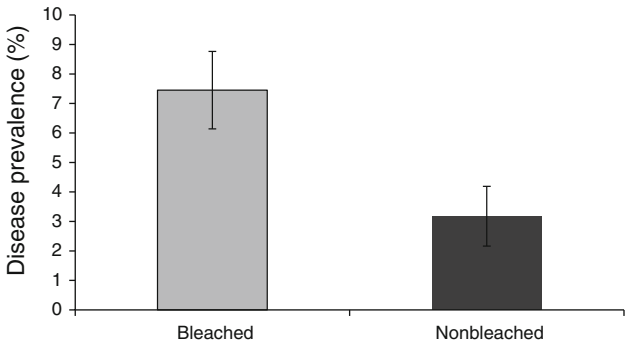

Fig. 6 Average disease prevalence on *A. palmata* colonies that bleached and those that did not bleach in 2005. Prevalence values for the comparison were taken from August 2005 through April 2006. *Error bars* represent the standard error of the mean

Increase in colony size and number of colonies

At the beginning of the study, the smallest colony was 48 cm^3 and the largest was 1,949,900 cm^3. By 2010 the smallest colony was 100 cm^3, whereas the largest colony was 2,830,500 cm^3. The average volume of the colonies over the course of the study increased every year (Fig. 8). This is an underestimate as it is a measurement of the living tissue on the main colony only and does not include the fragments. Growing colonies predominated each year except during the interval that included the 2005 bleaching event (2005/2006) where more colonies decreased in size than increased (decreased: $n = 29$, increased: $n = 27$; Fig. 9).

Over the years, the number of colonies within the study area increased. The initial 69 colonies contributed to many new colonies through fragmentation (see below), while additional new colonies were also discovered. These colonies were attributed to sexual recruitment since no fragments from the previously existing colonies were identified within the area prior to their appearance. When the total number of colonies within the reef area was surveyed in 2010, the number of individuals had increased to 213. Of the original colonies tagged in 2003, 44 were still alive. Approximately 139 were fragments or reattached fragments from the original colonies, and 30 were identified as colonies likely from sexual recruits.

Physical damage and fate of fragments

Physical damage was frequently observed (Fig. 10) with 2006 and 2008 having the greatest prevalence. The number of broken branches tended to peak in the late winter/early spring, likely reflecting damage associated with northerly swells during that time of year. Just over half of the colonies were damaged by snorkelers, fishing line, or heavy swells.

Of the 141 fragments that were monitored, 44 % survived for 6 months or more. By the end of the fragmentation study in June 2007, 46.1 % had died, 28.4 % were still alive and the fate of the remainder could not be determined. Only 32.6 % reattached to the substrate at some point. However, by the end of the study, these reattached fragments had survived for an average of 19.2 months while those that did not reattach to the substrate survived for only 5.4 months. Over half (53.7 %) of the unattached and 30.4 % of the reattached fragments suffered complete mortality. Fragment mortality (or loss)

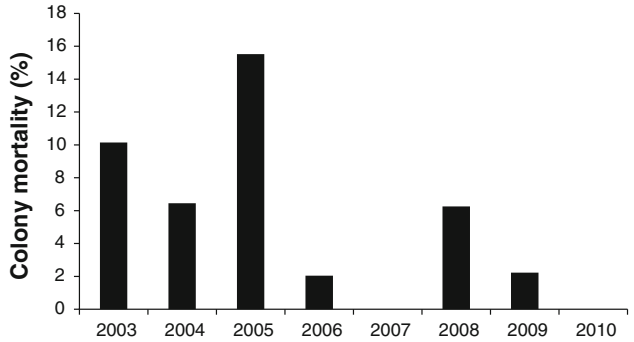

Fig. 7 Proportion of *A. palmata* colonies that died annually in Haulover Bay from 2003 to 2009

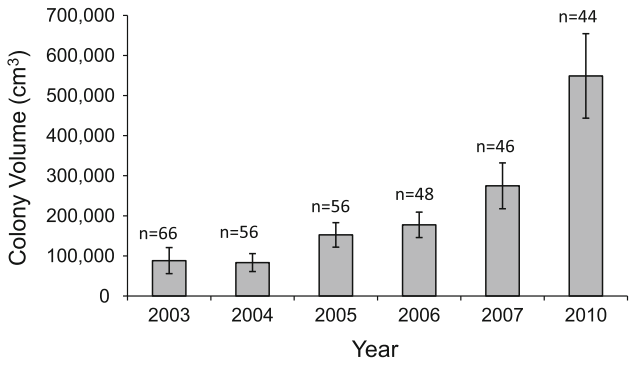

Fig. 8 Average volume of living tissue on monitored colonies of *A. palmata* still alive at time of measurement in Haulover Bay from 2003 to 2010. *Error bars* denote standard error of the mean

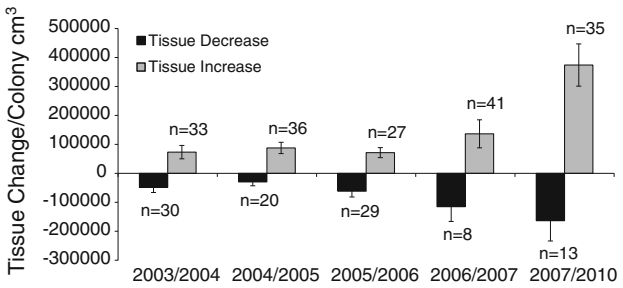

Fig. 9 Average change in volume of living tissue on monitored colonies of *A. palmata* in Haulover Bay between measurement dates between 2003 and 2010. *Error bars* denote standard error of the mean

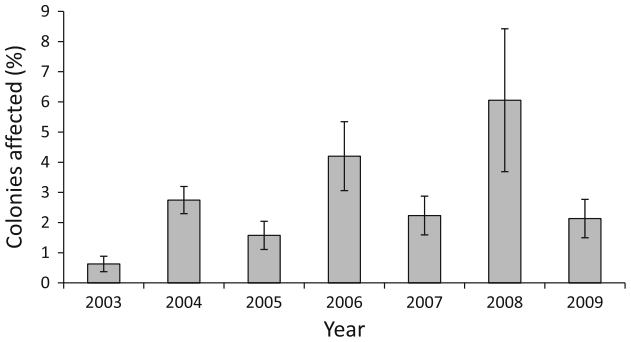

Fig. 10 Average percent of colonies with physical damage each year of the study. *Error bars* denote standard error of the mean

was from disease (29.2 %) disappearance (12.3 %), unknown causes (64.6 %) and bleaching (1.5 %).

Genotypic analyses of coral hosts and zooxanthellae

Forty-three of 48 corals had different genotypes, meaning that they grew from sexual recruits or from fragments of sexual recruits. Two genotypically distinct corals growing adjacent to each other exhibited different responses with only one of the colonies bleaching in 2005 (Fig. 11). All 48 of the *A. palmata* colonies that were examined, with one exception, had zooxanthellae in Clade A. (The clade for the remaining sample was not identified but it was not Clade A). Two colonies with the same host genotype showed similar morphology and growth patterns throughout the study (Fig. 12).

Discussion

Disease, bleaching, and water temperature

The greatest mortality of any Caribbean coral was the decimation of *A. palmata* throughout the region beginning in the 1970s and continuing for the next one to two decades, apparently from white band disease. (Few rigorous or long-term studies documented or quantified these losses and the specific causative disease or diseases are not known. An exception is the study by Gladfelter (1982) which includes clear descriptions, photographs, and rates of disease advance.) More recent studies, including this one from Haulover Bay, and others (Mayor et al. 2006) indicate that white band disease, although more virulent, is less prevalent than white pox and other diseases affecting this species. At Haulover, the single greatest cause of partial or complete coral mortality was disease, primarily white pox. White band disease was only seen on 10 colonies, only one of which died from the disease.

Fig. 11 Image of the different response to the 2005 thermal anomaly for two genetically distinct *A. palmata* individuals growing right next to each other. Genet on the *right* is bleached, whereas the genet on the *left*, displaying signs consistent with white pox, is not

Often white band disease progresses steadily across a colony, eventually killing it. In contrast, white pox, which affected 87 % of the colonies monitored in Haulover, often stops advancing, and coral tissue can regenerate over skeletal patches exposed by disease in less than a few months. Because white pox is less virulent than white band, *A. palmata* populations may have a better chance of persisting. Disease prevalence throughout this study varied greatly, ranging from 0 to 57 %. In Hawksnest Bay, off St. John's northwest coast, disease prevalence ranged from 0 to 32.6 % from May 2004 to December 2006 suggesting that the wide range of white pox prevalence may be similar among sites (Muller et al. 2008).

These wide ranges in prevalence and short duration of disease signs ($\sim$1.7 months) show that one-time surveys of prevalence can give a very misleading picture of the role of disease in determining the status of coral populations (Rogers 2010). The disease prevalence values observed in Haulover are generally higher than those reported for *A. palmata* on other St. John reefs (Rogers et al. 2008) and in other locations (e.g., Ward et al. 2006; Vega-Sequeda et al. 2008) and for diseases affecting other coral species (e.g., Ward et al. 2006; Croquer and Weil 2009a).

Disease prevalence was synchronous with water temperature with a 0 month time lag, indicating that water temperature influenced disease prevalence. The positive correlation coefficient between these two variables ($r = 0.34$) shows this association is positive. However the coefficient was also relatively small, suggesting that other parameters are likely influencing disease prevalence on *A. palmata*.

Annual average disease incidence remained constant throughout the study. Disease incidence over the course of this long-term study reflected multiple visible cases of disease on the same corals. As in any field survey, corals that appeared to be without disease could actually be infected and particular environmental conditions, such as warmer temperatures, might be required for the disease to manifest itself. For this reason, disease prevalence may be a more reliable indicator of the status of the population and its response to the environment.

Bleached corals had significantly higher disease prevalence than unbleached. Muller et al. (2008) found a positive linear relationship between disease prevalence and water temperature at Hawksnest Bay, but only for 2005 when sea water temperatures were the highest and when bleaching occurred.

Although it is logical to conclude that thermally stressed scleractinian colonies are more likely to develop disease

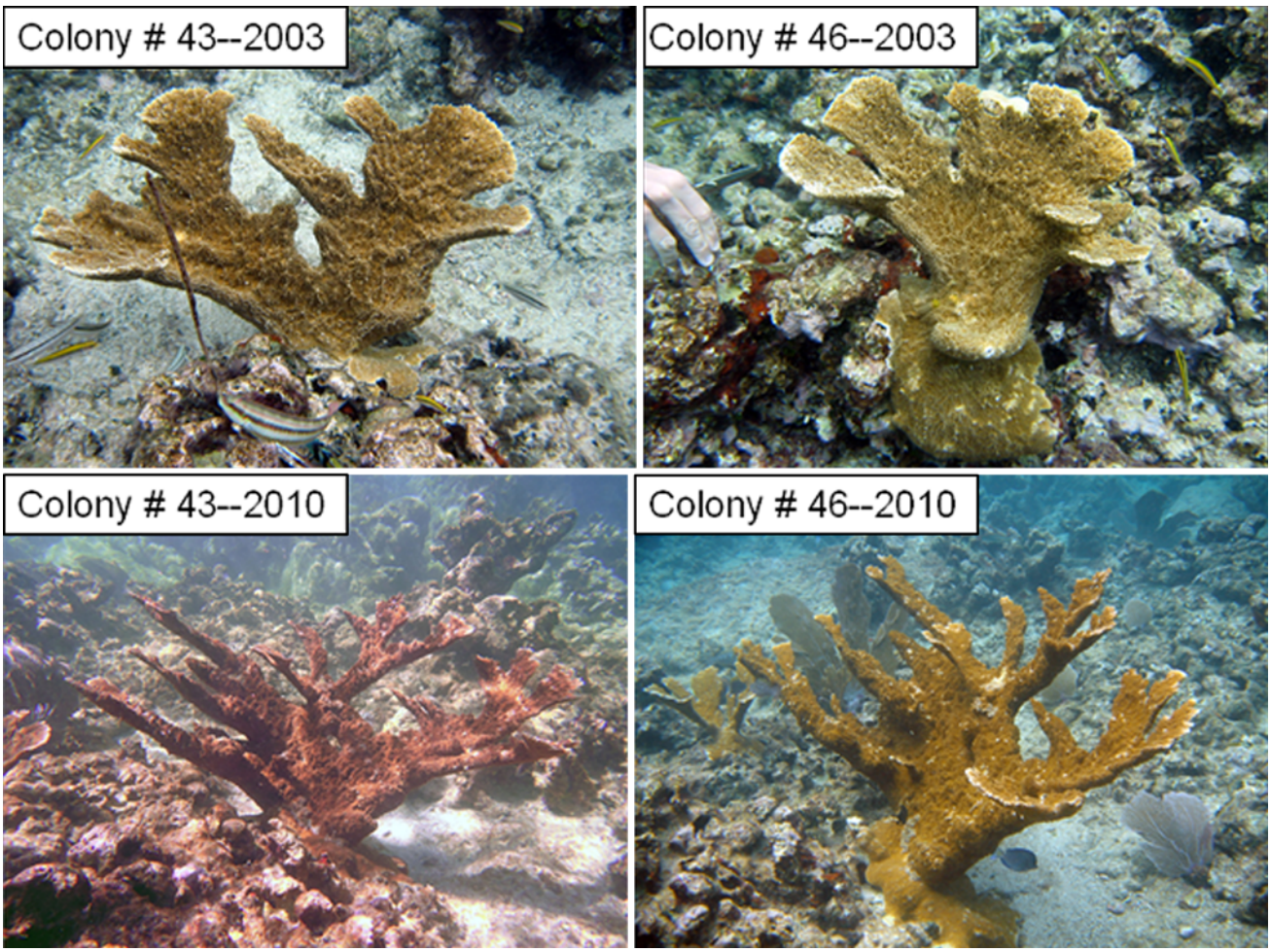

Fig. 12 Image of similar growth patterns of two genetic clones of *A. palmata* in 2003 and 2010

(in addition to bleaching) (e.g., Harvell et al. 2009), very few published studies clearly and conclusively show this relationship (Rogers et al. 2009a). Some of these studies involve only one disease (e.g., black band), a small geographic area, and/or a small number of coral colonies (Edmunds 1991; Kuta and Richardson 2002; Muller et al. 2008; Weil et al. 2009). An exception is the study by Croquer and Weil (2009b) who examined the link between bleaching intensity in 2005 and disease prevalence in 2006 on reefs from Bermuda, Puerto Rico, Grand Cayman, Panama, Curacao, and Grenada. They found a significant correlation between percent of bleached corals and prevalence of coral disease 1 year later. However, they noted that not all of their study sites that experienced severe bleaching also showed increases in disease prevalence. In another study over a large spatial scale, Bruno et al. (2007) showed a correlation between high temperature and "white syndrome" (but not other diseases) based on data from 6 years of annual monitoring at 48 Great Barrier Reef sites (see also Willis et al. 2004). Based on essentially the same dataset, Selig et al. (2006) showed a positive relationship between thermal stress and disease. However, thermal stress, defined by Selig et al. (2006) as Weekly Sea Surface Temperature Anomalies, was not correlated with bleaching severity. Some of the inshore reefs had the most severe bleaching while the outer Capricorn reefs had the highest thermal stress and disease prevalence (see Berkelmans et al. 2004).

Disease prevalence at Haulover showed a weak association with temperature, and bleached corals had significantly higher disease prevalence than unbleached. Of course, correlation is not the same as causation, and more research is needed to determine the relationship among high temperatures, bleaching and disease. The positive association between water temperature and disease prevalence may have resulted from pathogen proliferation during high water temperature or because compromised coral hosts became more susceptible to pathogenic infection. Further research is essential to determine the mechanism driving this association. Bleaching does not always result in mortality and it does not always precede disease. In addition, the time interval between bleaching and disease outbreaks requires further consideration. The few studies examining

the links between bleaching and disease have been done over different time frames. In the study by Croquer and Weil (2009b) described above, surveys were about 1 year apart. If the surveys had been done just 6 months apart, an even stronger correlation might have been found between bleaching and subsequent disease. Specifically, prevalence at some sites could have been higher than months later after more corals present in the initial survey had completely died.

Of a total of 467 *A. palmata* corals from Haulover and three other sites around St. John that were being monitored monthly in 2005, 48 % bleached, 13 % bleached and died partially, and 8 % bleached and died completely by July 2006 (Rogers et al. 2008). Of the corals that died, bleaching seemed to be followed directly by mortality without the return of normal coloration. In contrast, at deeper long-term study sites, colonies of nearly all species bleached dramatically in October 2005 (over 90 % of the total coral cover), and then began to regain their normal coloration after several days of overcast, rainy conditions in November. By December they began to succumb to white plague disease which persisted for several months and caused over 60 % loss in coral cover (Miller et al. 2006, 2009). By October 2006, 1 year after the peak in the bleaching event, the white plague disease outbreak had ended. Therefore, annual monitoring would have completely missed this disease outbreak.

No definite links between disease and any anthropogenic factor are evident for corals on reefs around St. John. Samples from *A. palmata* colonies in Haulover and Hawksnest Bays with white pox signs did not show a conclusive link with *Serratia marcescens* (Polson et al. 2009; May et al. 2010), the bacterium associated with this disease and linked with human sewage in Florida (Patterson et al. 2002; Sutherland et al. 2010).

Fate of fragments

Fragmentation is a primary mode of reproduction for *A. palmata* (Highsmith 1982), and the fate of fragments (mortality, fusion to the substrate and growth, etc.) may provide a good indication of the potential for new and old coral colonies to survive and grow in a particular reef zone. If a fragment does not land on or attach to suitable substrate, survivorship may be low. Fragments of *A. palmata* may be more likely to survive in the USVI than in Florida (see Williams et al. 2008). In Haulover, 44 % of the fragments survived for 6 or more months. Indeed, our data show that most of the new corals recorded in 2010 were formed from fragmentation. Williams et al. (2008) documented significant loss of *A. palmata* in the upper Florida Keys from April 2004 through April 2007 primarily from hurricanes and noted that over 70 % of the fragments associated with Hurricane Dennis were either dying or dead within just 2–3 weeks of the storm.

Genotypes and disease resistance

Further research, using more genetic markers, may eventually reveal some genetic basis for differential susceptibility of corals to bleaching and disease. Vollmer and Kline (2008) state that 3 out of 49 genotypes of *Acropora cervicornis* appeared to be resistant to white band disease. Almost all of the *A. palmata* colonies monitored in Haulover had disease at one time during the study, although most were of different genotypes (i.e., not clones). Interestingly, three corals that survived until the end of the study never showed signs of disease. Possibly the two adjacent corals shown in Fig. 11, which were two distinct genotypes, had different "fates" (one bleached and one not) because they had different host or zooxanthellae genotypes.

Signs of "recovery"

No published papers to our knowledge document significant recovery of any *A. palmata* population in the Caribbean. The best evidence of recovery would come from multi-year studies showing all of the following: an increase in the overall amount of living tissue of this species, growth of existing colonies, and an increase in the number of small corals arising from sexual recruitment.

Currently we lack sufficient quantitative data for the wider Caribbean to state whether recovery is occurring or to provide a baseline for future evaluation. Information on the historic and current distribution and abundance of *A. palmata* comes from Bruckner (2003), Aronson and Precht (2001a), and the *Acropora* Biological Status Review Team (2005). The Biological Status Review (2005) has scattered records of the presence of *A. palmata* but few reports on its status and distribution since 2000.

A few studies have shown increases in the number of colonies but none has shown recovery to abundances/densities similar to the 1970s (Macintyre and Toscano 2007; Zubillaga et al. 2008). Some investigators report no living *A. palmata* where there used to be substantial populations (Macintyre et al. 2007; for Barbados), while some have documented little change or a decrease (e.g., Garzon-Ferreira et al. 2004; Vega-Sequeda et al. 2008; Rodriguez-Ramirez et al. 2010, for reefs in Colombia).

Existing data on the distribution and trends in abundance of *A. palmata* in the wider Caribbean support the listing of *A. palmata* as a threatened species—i.e., the species is not presently likely to become extinct but is facing possible extinction in the future. The status of the *A. palmata* population in Haulover Bay, which is within the boundary

of a national park and not subjected to excessive sewage, sedimentation or other stressors as far as we know, may be representative of the best case for this species. Here increases in the number and average size of the colonies and evidence of sexual recruitment suggest the possibility of recovery, but several stressors continue to threaten *A. palmata*. Bleaching episodes are predicted to become more frequent and tissue-loss diseases may become even more prevalent. Possibly acroporids will have less ability to deal with bleaching because they do not have better-adapted zooxanthellae clades (e.g., Stat and Gates 2011). Scleractinian coral diseases, including those that affect the acroporids, are widespread throughout the Caribbean (Weil and Rogers 2011), even occurring in relatively remote locations (Miller and Williams 2007). Much is uncertain about how the acroporids and other corals, and entire reefs, will respond to ocean acidification (Kleypas 2007; Bak et al. 2009). Future storms, also expected to increase in frequency with climate change, will cause more fragmentation, and if they are frequent enough, could hinder recolonization. For example, in August 2011, Tropical Storm Irene caused fracturing of half of the colonies in Haulover. A 2008 study showed that damage from waves was correlated with greater disease prevalence on *A. palmata* colonies in Hawksnest (A. Bright, personal communication). Following several hurricanes in Florida in 2005, Williams et al. (2008) saw substantial loss of *A. palmata* from physical damage and diseases.

Although *A. palmata* colonies will continue to face stressors in the future, this fast-growing species may have a greater likelihood of recovering than the massive corals (*Montastraea annularis* complex, *Diploria* spp., *Colpophyllia natans*) that declined dramatically in the USVI because of an outbreak of disease following the severe bleaching in 2005 (Miller et al. 2009; Rogers et al. 2009b).

Recovery of *A. palmata* in Haulover Bay will be a slow, uncertain process, if it happens at all. Disease prevalence at Haulover Bay remains high, even though Sutherland et al. (2010) noted that white pox prevalence had declined in Florida. White band continues to affect at least some *A. palmata* populations in the Caribbean (Mayor et al. 2006). Even *A. palmata* colonies at relatively remote areas like uninhabited Navassa Island have disease (Miller and Williams 2007). However, increases in abundance and size of surviving colonies at Haulover Bay provide some hope for potential population growth.

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