The School for Field Studies - Center for Marine Resource Studies
South Caicos, Turks & Caicos Islands, British West Indies

Directed Research Paper

Are South Caicos *Acropora palmata* populations infected by diseases?

Spring, 2004

Jennifer A Lentz

ABSTRACT: *Acropora palmata* populations across the Caribbean are being decimated, with losses of reef building communities becoming all too common. While there are a plethora of stressors degrading the colonies, disease is one of the greatest areas for concern. At present very little is known about these devastating diseases, causing such high rates of tissue necrosis. Efforts to curb this degradation call for Caribbean wide reef assessments and monitoring. While visual surveys noted vast expanses of dead *A. palmata* stands, the living communities were remarkably healthy. Two diseases were found around South Caicos: an abnormal form of white band disease and two forms of white pox disease. The presence and rapid spread rates of white band disease (4.33 cm^2day^{-1} maximum) and white pox disease (WPDa 6.18 cm^2day^{-1} and WPDb 2.40 new pox per day maximum) show that reef-building communities around South Caicos may have quite a battle ahead of them. With 4 of the 5 study sites showing more than 80% of the *A. palmata* colonies were "healthy" (without disease), South Caicos is either more resistant than most Caribbean reefs, or is in the initial phase of decline. What ever the case may be the Turks and Caicos Islands prove to be an area in need of further study.

KEY WORDS: White Band Disease, White Pox Disease, Elkhorn coral, Turks and Caicos, Caribbean

INTRODUCTION

Recent comprehensive studies of coral cover show extensive region-wide declines throughout the Caribbean (Richardson 1998, Aronson and Precht 2001, Precht et al. 2002, Gardner et al. 2003). Over the past three decades the Caribbean has experienced losses of 95% or more of the once dominating *Acropora palmata* populations (Precht et al. 2002). Paleontological studies show Acroporids dominated coral reef communities throughout the Caribbean from the Pleistocene to the end of the 1970s, suggesting the present mortality rates is without precedent in the Holocene Epoch (Jackson 1992 and Mesolella 1967, cited in Aronson & Precht 2001).

Acroporids are known for developing the reef framework, as well as providing habitat critical to the support of diverse reef fish populations (Lirman 1999), and other organisms which contribute to the productivity and overall health of the reef (Precht et al. 2002). Rapid decline of *Acropora* cover over the past 30 years has led to the listing of *A. palmata* as a candidate for the List of Threatened Species under the Endangered Species Act (Precht et al. 2002).

While there are a plethora of causes for the degradation (including hurricane damage, snail predation, sedimentation, eutrophication, global warming, overfishing), studies show that fast spreading diseases are the primary cause of coral mortality, and coral degredation has been further amplified by the proliferation of new types of diseases in the 1990s (Richardson 1998, Harvell et al. 2001, Aronson & Precht 2001, Pretcht et al. 2002). In 1977 Gladfelter first reported white band disease (WBD) on *A. palmatas,* and until recently WBD was considered the dominant cause of *Acropora* mortality throughout the Caribbean (Gladfelter 1982, cited in Aronson & Precht 2001 and Rogers 2004). First documented in 1996, the highly contagious and rapidly spreading white pox disease (WPD) now rivals WBD as the leading cause of disease related *A. palmata* mortality (Patterson et al. 2002).

WBD is presumed to be a bacterial infection (Gladfelter 1982, cited in Aronson & Precht 2001), although to date no pathogen(s) have been identified (Aronson & Precht 2001, Richardson et al. 2001). Antonius (1981) posed that WBD may not be caused by a pathogen, but rather is a "shut-down-reaction" within the coral, a biochemical response to some type of coral trauma. The paleontologically unprecedented outbreaks of WBD have lead many to speculate anthropogenic stressors are at the heart of the disease outbreaks, but no direct evidence of this has yet been found (Gardner 2003, Aronson & Pretcht 2001, Richardson 1998). The cause of WPD, however, is known and is perhaps the first case

of a marine invertebrate pathogen caused by a bacterial infection stemming from human gut, *Serratia marcescens*, a common fecal enterobacterium (Patterson et al. 2002).

While there is debate over the causes of WBD, as well as the extent and severity of disease related mortality in Acroporids, increasingly studies are showing that virtually all areas of the Caribbean are at risk of degradation (Gardner et al. 2003). The Turks and Caicos Islands (TCI) currently have low human population pressures and reefs that are generally in good condition (Riegel et al. 2003). Within the TCI, South Caicos Island was selected in order to assess *Acropora* health in an area with relatively no tourism and very limited overall anthropogenic influences.

MATERIALS AND METHODS

Study Sites. Five survey sites were selected around the southern end of the South Caicos coast, according to where living *A. palmata* had been reportedly seen (Fig.1). For study site characteristics see Table 1.

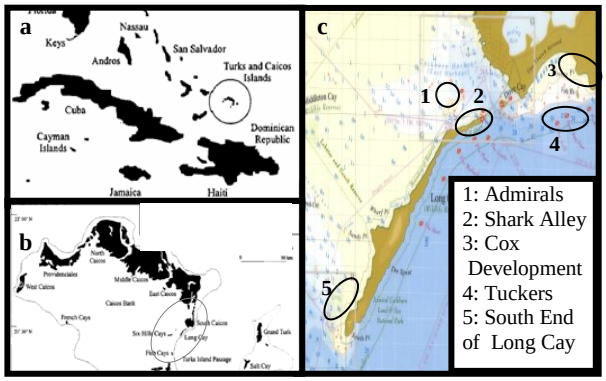


Figure 1. (a) Location of the Turks and Caicos Islands, (b) South Caicos Island, and (c) Data collection sites, located off the southern coast of South Caicos (modified from Béné & Tewfik 2001).

Table 1. Physical profile of the 5 data collection sites

Site	Reef type	Mean depth (±SD)
Admirals	Patch	0.75 m (± 0.35)
Cox Development	Fringing	0.92 m (± 0.04)
Shark Alley	Fringing	1.62 m (± 0.07)
South End Long Cay	Patch	0.74 m (± 0.06)
Tuckers	Fringing	1.54 m (± 0.12)

Sampling techniques. Before collecting data, each colony was given an identification number and marked by a duct tape label nailed into the underlying substrate without affecting the coral colony. The height, width, and length of the corals were measured, as well as the depth of each colony. The

percentages of total dead and recent dead coral were visually estimated. Percent total dead coral includes recent and old mortality; a recently dead coral can be identified by its bright white color and lack of algal overgrowth, whereas old mortality lacks the brightness and is often covered in algae. After all the colonies were tagged and measured at each site the total area of the site was measured in order to find the *A. palmata* density (colonies per ha) for each site.

Each tagged coral was carefully inspected for either WBD or WPD. Each WBD and WPD occurrence was tagged with a duct-tape number and a zip-tie as close to the disease as possible, when possible the zip tie was placed on old mortality. Calipers were used to measure the width and height of the disease. In order to distinguish which direction the disease was spreading the distance from the closest part of disease to the zip-tie was also measured. The sites were revisited 2-3 times over the course of 2 months in order to measure disease progress to calculate the rate the diseases are killing off the living *A. palmata* tissue.

Photo documentation. In addition to the measurements, digital photos were taken of the disease incidences on the tagged corals. Depending on the size and accessibility, either a wooden T-bar or a plastic caliper was held up next to the disease for scale. When the number of pox were too high to be tagged and measured individually, the total number of pox were counted. Disease spread rates will be based on the addition of new pox as well as changes in the sizes of old pox (found through photo comparison).

Data Analysis. A chi-squared test was run comparing the expected values (from a 2004 USVI study) to the actual values of the percent health, WBD, and WPD of each reef (Rogers 2004). STATISTICA 1997 was used for all of the statistical analyses. Levene's Homogeneity and the Normal Plot of Within Cell Residuals Normality were used to determine if there was a significant difference between the rate of disease spread between sites. If the data was found to be both normal and

homogeneous then a 1-way Anova significant difference test and Tukey Honest Significant Difference Tests (HSD) to determine where significant difference lies (if one was found in Anova) were used. When the data was not homogenous or normal the non-parametric Mann-Whitney U test was run. A Spearman Rank Order Correlation test was used to see if there was a correlation between the rate of disease spread and colony depth.

RESULTS

***A. palmata* abundance.** During surveys done between March 9th and April 19th 2004 tagged 267 *A. palmata* on the 5 reefs around South Caicos were tagged. The distribution of *A. palmata* colonies varied greatly between sites: Admirals had the lowest number, with only 2 tagged colonies and Cox Development had the most with 108 tagged colonies. Reef densities ranged from 19.85 to 129.19 colonies/ha, due to bad weather the density of South End Long Cay was not able to be measured.

Disease Distribution. Visual surveys of the tagged colonies showed that 7.9 % of the living *A. palmata* colonies at the 5 sites were diseased, with 0.7 % WBD, 7.1 % WPD. Over the course of the study 2 types of WPD were found, which are distinguished by the way they spread throughout a colony. "WPDa" spreads by means of increased mass; whereas, "WPDb" spreads by infecting the coral with new pox. Over all the incidence of the 2 types of WPD was very close, with WPDb accounting for 52.6 % of the WPD and WPDa 47.4%. Visual surveys showed that WPDb tended to be found on colonies within close proximity to other WPDb colonies; this trend was not nearly as strong with WPDa.

Disease prevalence among the sites varied greatly (Fig. 2). 50% of Admirals *A. palmata* had WPDa. Tuckers had the next highest disease occurrence with 17.9% of the 56 tagged corals exhibiting signs of WPDb. However, this percentage may be even higher since the whole *A. palmata* population

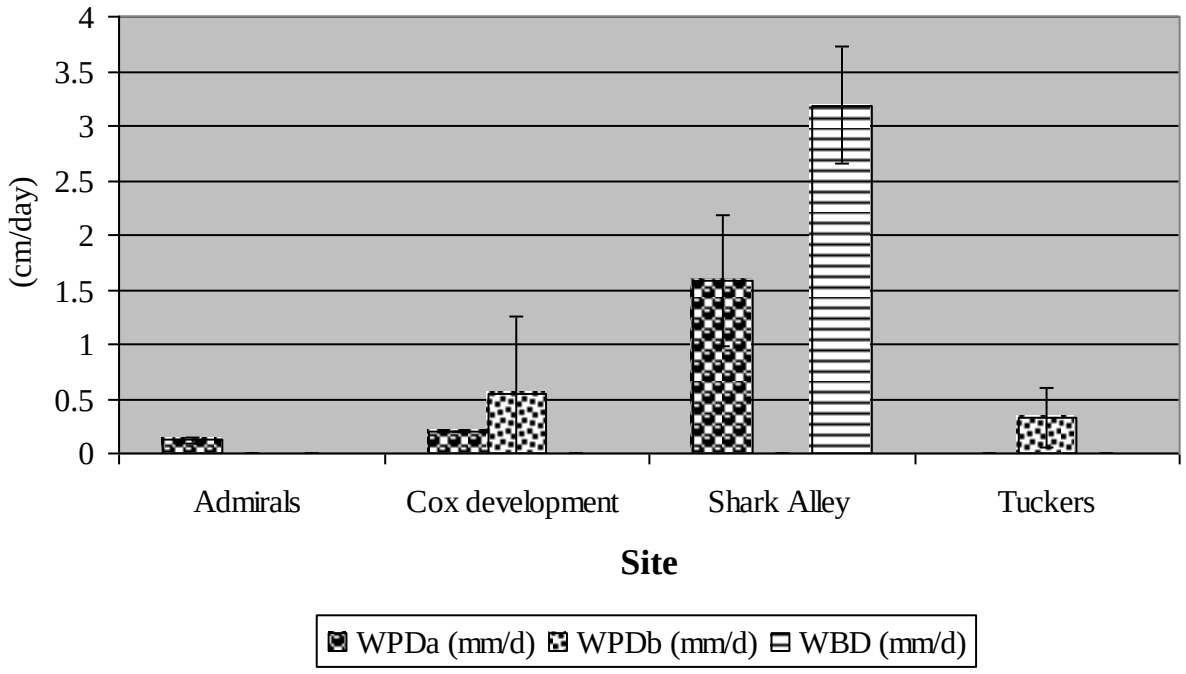


Fig. 2. Disease distribution per site

of the site was not tagged due to time restraints and weather conflicts. Shark Alley and Cox Development had lower disease incidences, 11.6% and 6.6% respectively, but their disease profiles (physical distribution, type of disease and rate of spread) varied greatly. South end Long Cay had no diseased colonies.

The community profile from the USVI found 36.36% of the *A. palmatas* were healthy, 59.09% had WPD, and 4.55% had WBD (Rogers 2004). A chi-squared test found the community profiles of each of our 5 sites to be significantly different from the USVI study. Admirals had the most similar profile to the USVI. However, the sample size of Admirals was very small with only 2 colonies.

White band disease was found only at Shark Alley on two colonies within relatively close proximity to each other; both instances of WBD exhibited atypical spread patterns. On one colony the band appeared to have initially followed a normal spread pattern; however, once the end of the second branch was reached the disease appears to have spread to the tip of a 3rd branch. A nearby colony

(within 10m) also showed this atypical spread pattern, starting at the tip of the branch and working its way down the branch (Fig. 3).

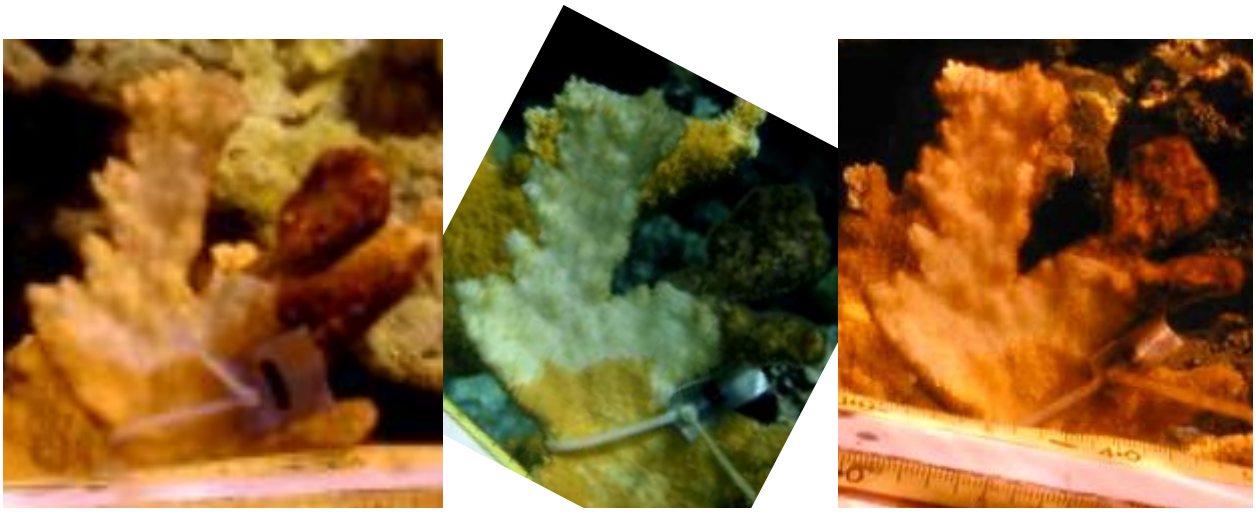


Fig. 3. Shark Alley colony 7 exhibited atypical WBD branch-tip to base spread over a 16 day period. From left to right, pictures taken on March 24th, March 30th, and April 9th 2004.

Rate of spread. Due to the difference in method of disease proliferation the rates of spread for WPDa and WPDb could not be compared. However, WBD can be compared to WPDa because they both spread by means of expansion (Fig. 4). Physical measurements showed WPDa lesions could increase in area up to 6.18 cm^2day^{-1}, with a mean rate of lesion expansion of 0.80 cm^2day^{-1} (±0.56 SD). There was no significant difference between the rates of WPDa expansion for Admirals and Shark Alley (Mann-Whitney U, u = 12, z = -1.58, p = 0.11), there were not enough measurements to include other sites in the comparison. While WBD showed a higher mean rate of tissue loss than WPDa, 2.80cm^2day^{-1} (±0.76 SD), and was capable of increasing in area up to 4.33 cm^2day^{-1}; there was no significant difference between the spread rates of WBD and WPDa (Mann-Whitney U, u = 15, z = 0.51, p = 0.13). WPDb showed as many as 2.40 new pox per day, with a mean rate of expansion of

0.62 newly developed pox day^{-1}(±0.41 SD). There was no significant difference between the spread rates of WPDb between sites (Mann-Whitney U, u = 18, z = 0.77, p = 0.44).

The Spearman Rank Order Correlation test was used to test for a correlation between the rates of WPA spread per colony and depth; a correlation was not done for WBD because the sample size was too small. The test found a weak correlation (45%) for both types of pox (WPDa: r = 2.29, p = 0.03; WPDb: r = 1.73, p = 0.10).

Photographic analysis. Photographic disease spread analysis was only possible for one colony. Analysis of pictures taken on 3 separate occasions show a mean rate of disease spread per day of 2.23 cm^2day^{-1} (±1.77 SD). When compared to the caliper field-measurement data, the photographic analysis yielded a 0.21 cm^2day^{-1} higher mean spread rate than the caliper measurements.

DISCUSSION

***A. palmata* abundance & disease tendencies**. The distributional results of our study conquered with a 1999 AGRRA study of the TCI, finding the highest *A. palmata* densities on the Eastern facing sides of the reefs (Riegel et al. 2003). With out tagging all of the *A. palmata* colonies it is not possible to have truly representative data on the health of the colonies. Due to persistently bad weather only 2 of the 5 sites were mapped completely (Admirals and Shark Alley); however, while the presence of WBD or WPDa-b were noted on all the Shark Alley Colonies time constraints made it impossible to measure the rate of spread on all of the colonies. Only 15 colonies were tagged at the South End of Long Cay, none of which were disease; however, it is possible that the Acroporids reportedly seen closer to the southern tip of the cay are more diseased. While the northern face of the Tuckers reef is mapped completely, the A. palmata colonies on the southern side were not found until the end of the study and were far too abundant to be completely mapped. The same is true of Cox

Development; the western side of the reef was mapped completely but the eastern side was found too late in the study to be to fully mapped. With the presence of WPDb at both Tuckers and Cox Development, it is especially important that future studies go back to these sites and finish mapping the distribution of the colonies

Across the board the South Caicos reefs are considerably healthier than the USVI reefs. This conquers with the AGRRA study which reported the TCI reefs lived up to their reputation of being pristine; however, both the South Caicos the AGRRA study found standing dead *A. palmata* colonies were far more abundant than living colonies (Riegel et al. 2003). The health of the South Caicos reefs is most likely caused by the relatively low population pressures of humans in the area. As in the USVI, WBD incidence was far less common than WPD.

White band disease was only found at one of the 5 sites and on only two colonies. Both colonies were in close proximity to each other which might suggest that their incidence is related. The progression of the band between branches on one of the two infected colonies also suggests the disease may be contagious; although the high Damsel fish grazing and snail predation on colony 7 may also indicate that the WBD is a "shut-down" reaction from the stress (Antonius 1981, Richardson 1998, Gladfelter 1982). To date there are no instances of WBD on *A. palmata* spreading from the tip of the branch to the base, this is only found in WBD Type II which has only been found on *A. cervicornis* (Rogers 2004, Ritchie & Smith 1998). While the mean rate of spread per day (2.80 cm^2day^{-1}, ± 0.81 SD) of WBD on the 2 colonies is close to the maximum rate of expansion (2.06 cm^2day^{-1}) documented in 1986 (Davis et al. 1986 cited in Rogers 2004); the maximum rate of spread found at Shark Alley (5.47 cm^2day^{-1}) was much higher than other documented rates (Davis et al. 1986 and Gladfelter 1982 cited in Rogers 2004). The atypical method of progression may be a factor in the increased spread rate.

Visual distribution maps of WPDa and WPDb incidences within and between reefs conquered with Patterson's (2001) study which found WPD was highly contagious within and among reefs, adhering closely to the nearest-neighbor contagion model. White pox was only found on the 4 reefs that were within close proximity of each other; the site that was the most remote had no diseases present. This shows a very high frequency of between reef pox spread. Of the individual types of pox, WPDa does not appear to have a high within reef rate of transmittance; whereas, WPDb is found exclusively in colony clusters, thus exhibiting a very high rate of dispersion.

The spread rates for WPDa can be compared to Patterson's study of WPD in the Florida Keys, keeping in mind that the rates are not representative of the total pox around South Caicos (Patterson et al. 2002). With a third the sample size our results showed a 1.7 cm^2day^{-1} difference from the Florida study in mean rate of lesion expansion. Because the area coverage of WPDb was not measured it was not possible to correlate Patterson's large-fast growing pox to WPDa and his small-slow growing pox to WPDb. Our study found high variability in tissue loss between colonies, which conquered with Patterson's findings; our study also found high variability within colonies.

Photographic Analysis. A comparison between the caliper and photographic measured rates of WBD spread on one colony found that there was not a large difference between the two data collection methods. Therefore, while the photo analysis is more precise, the caliper measurements are not inaccurate.

Local Stressors. While at present the TCI reefs remain relatively pristine with few human induced negative impacts, there are stressors present which put the colonies at risk of incurring the rapidly increasing mortality rates which are becoming all too common across the Caribbean (Gardner et al. 2003). Increasingly studies are showing acroporid diseases occur in the end stages of stress induced synergistic effects (Harvell et al. 2001, Richardson 1998, Antonius 1981). It is believed that

stressed corals have weakened defense mechanisms and are therefore more susceptible and vulnerable to diseases (Gardner et al. 2003, Aronson & Precht 2001, Harvell et al. 2001, Richardson 1998). Natural occurring stressors include snail predation, weather, climate change, and temperature induced bleaching.

Snail predation is a common natural stressor of *A. palmata* colonies; stressed corals' high vulnerability to disease thus makes a link between snail predation and disease incidence probable (Antonius & Riegl 1997 and Bruckner et al. 1997 cited in Aronson & Pretch 2001). However, our study found no significant link between the two, which correlates to other studies which, to date, have yet to establish a firm connection between the two (Aronson & Pretch 2001).

While hurricanes have been responsible for extensive Caribbean coral mortalities (Aronson & Pretcht 2001), having only been directly hit twice in the past century hurricanes are most likely not the primary cause for coral mortality in South Caicos.

Other, more global, natural stressors such as global warming have also been linked to acroporid degradation, through incidences of bleaching and increased water temperature (Gardner 2003, Patterson 2002, Aronson & Precht 2001, Harvel et al. 2001, Richardson 1998, Antonius 1981). Patterson (2002) reports a causal linkage between bleaching and disease related mortality due to increased opportunistic infections. Patterson further speculates that the increased frequency and intensity of future bleaching events, due to global warming, may lengthen the disease season.

Relatively low anthropogenic stressors in the TCI most likely play a large role in the overall health of the *A. palmata* populations. With almost no tourism market and low population pressures, the greatest anthropogenic stressor of South Caicos is overfishing. However, increased pressure to cater to the tourism market has led to the present construction of 2 hotels and one all-inclusive resort on the island, thus putting South Caicos at risk of increased human-related stressors in the near future.

Located along the bank of one of the hotels currently under construction, the Cox development site is at immediate risk of anthropogenic stress. Current high sedimentation and pollution (nets) found on the WPDb infected colonies suggests that the synergistic degradation process has already begun at this site.

Limitations. Our data collection was inhibited by two main factors: weather and camera malfunctions. According to South Caicos inhabitants spring 2004 had abnormally strong and prolonged weather systems. Strong winds and storm induced wave action severely limited the number of data collection days. On days when the boats were able to make it out, strong waves and tidal variation made taking measurements very slow and difficult, photo-documentation nearly impossible. Most of the sites visited were exposed and there were probabilities of coral injury in rough sea. In many cases coral diseases could not be re-measured because the low tides made it impossible to reach the tagged coral. We also experienced camera malfunctions resulting in the loss of photographic data from certain collection days.

In conclusion, while the present condition of *Acropora palmata* populations in South Caicos is good compared to other Caribbean islands, the presence of white pox disease is cause for concern. Since very little is known about the causes of WBD, WPDa, and WPDb it is very difficult to prevent colonies from catching them. Natural reef stressors are very difficult to prevent. However, the reefs can be protected from increased anthropogenic stress. Limiting the influence of colony stressors will likely prove to be the keystone in saving the *A. palmata* from the extinction that they are currently facing throughout the Caribbean.

Acknowledgements. I wish to thank Chris Schelton for all of her help and guidance, Caroline Rogers for her help in identifying diseases, and The School for Field Studies (SFS) Center for Marine Resources, South Caicos Island, for their financial and logistical support.

LITERATURE CITED

Antonius A (1981) The "band" diseases in coral reefs. Proc 4th Intl Coral Reef Symp, 2:7-13

Aronson RB, Precht FP (2001) White-band disease and the changing face of Caribbean coral reefs. Hydrobiologia 460:25-38

Done TJ (1992) Phase-shifts in coral communities and their ecological significance. Hydrobiologia 247:121-132

Gardner TA, Côté IM, Gill JA, Grant A, Watkinson AR (2003) Long-term region-wide declines in Caribbean corals. Science 301:958-960

Harvell D, Kim K, Quirolo C, Weir J, Smith G (2001) Coral bleaching and disease: contributors to 1998 mass mortality in *Briareum asbestinum* (Octocorallia, Gorgonacea). Hydrobiologia 460:97-104

Lirman, D (1999) Reef fish communities associated with *Acropora palmate*: relationships to benthic attributes. Bull Mar Sci 65(1):235-252

Riegel B, Manfrino C, Hermoyian C, Brandi M, Hoshing K (2003) Assessment of the coral reefs of the Turks and Caicos Islands (part1: stony corals and algae). In: Lang JC (2003) Status of Coral Reefs in the Western Atlantic results of Initial Surveys, Atlantic and Gulf Rapid Reef Assessment (AGRRA) Program. Atoll Res Bull 460-480

Patterson KL, Porter JW, Ritchie KB, Polson SW, Muller E, Peters EC, Santavy DL, Smith GW (2002) The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acropora palmata.* PNAS 99(13):8725-8730

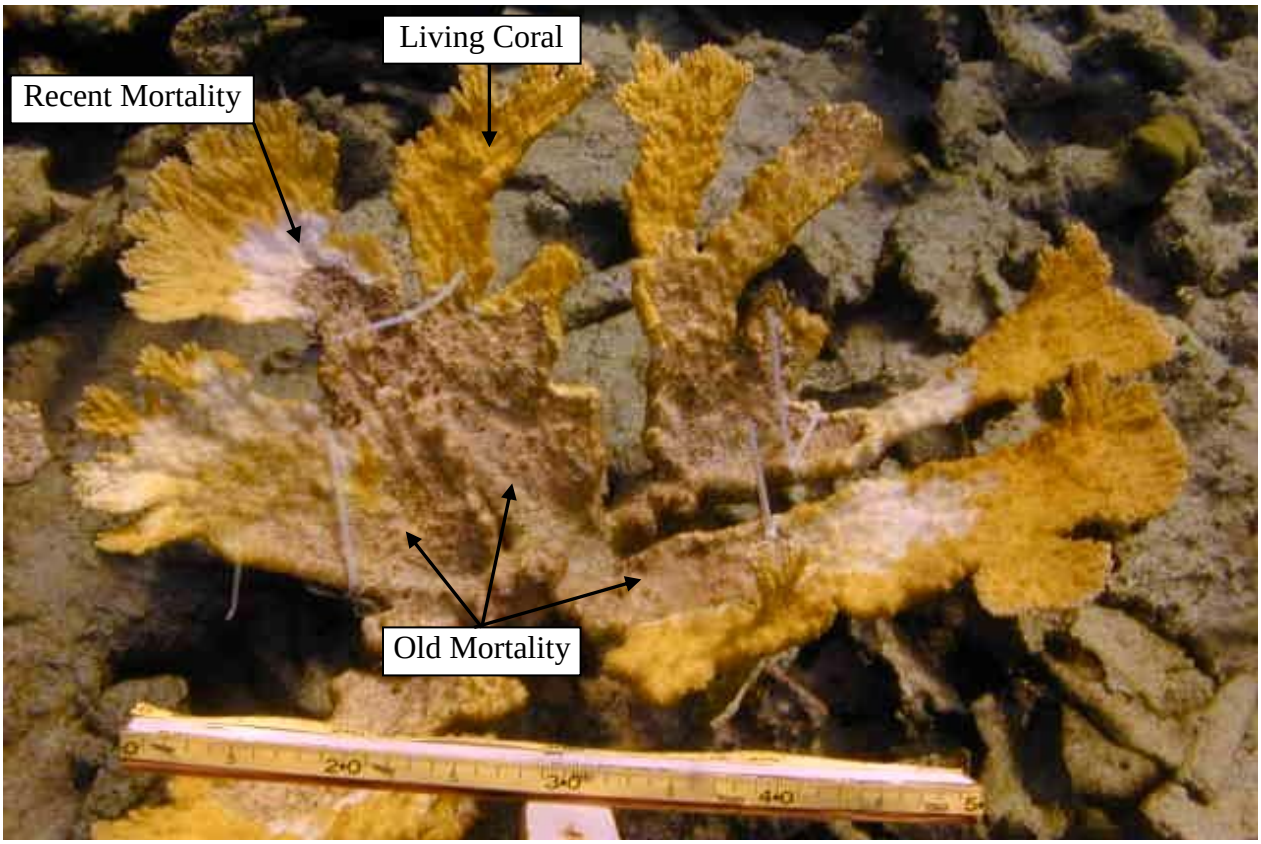
Precht WF, Bruckner AW, Aronson RB, Bruckner RJ (2002) Endangered acroporid corals of the Caribbean. Coral Reefs 21:41-42

Richardson LL (1998) Coral diseases: what is really known? TREE 13(11):438-443

Ritchie KB, Smith GW (1998) Type II white-band disease. Rev Biol Trop 46:199-203

Rogers C (2004) White Band Disease: What we do and do not know. Coral Disease and Health Consortium Workshop. Florida

APENDIX



The above photo shows how to differentiate between living and dead (new and old mortality) *Acropora palmate*. The photo is of snail predation on colony #20 at Shark Alley on March 24th, 2004. In cases like colony 20, snail predation can be confused with WDB, it is therefore very important to check for the presence of snails before classifying the cause of coral degradation.

White Band Disease (WBD)

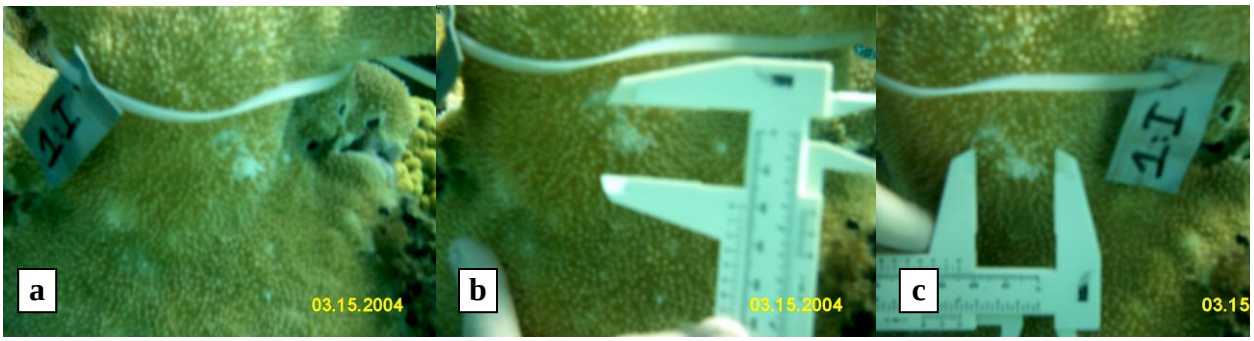
White band of recently dead tissue that works its way up the coral from the base (trunk) to the tips of the branches. The cause of WBD is not yet known. It is speculated to be either a "shut-down reaction" resulting from trauma, or caused by either bacterial pathogens. (Antonius 1981; photo by Halas*)

*http://ourworld.compuserve.com/homepages/mccarty_andpeters/coral/wbdpage.htm

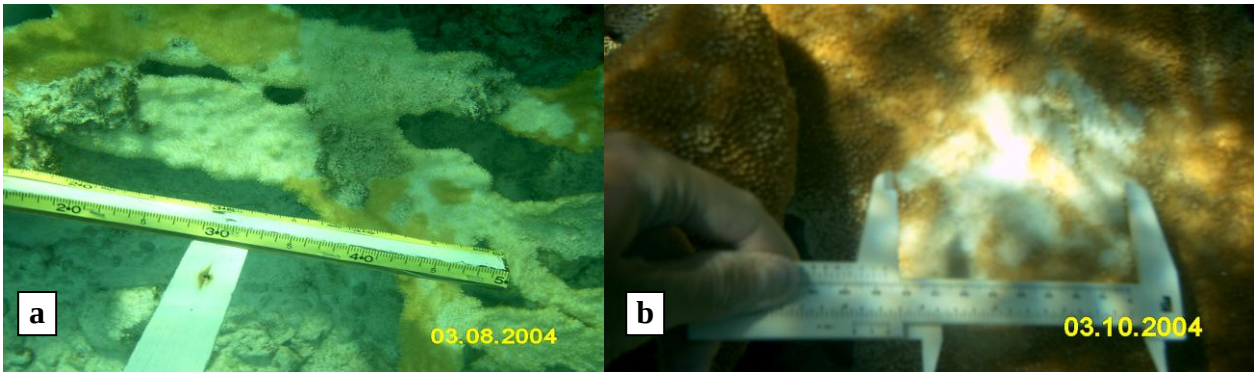
White Pox Disease (WPDa & WPDb)

Irregularly shaped white spots or patches that spread rapidly and sporadically throughout coral colonies. The pox are highly contagious between neighboring colonies and are thought to be caused by *Serratia marcescens*, a common human fecal enterobacterium. (Patterson et al. 2002)

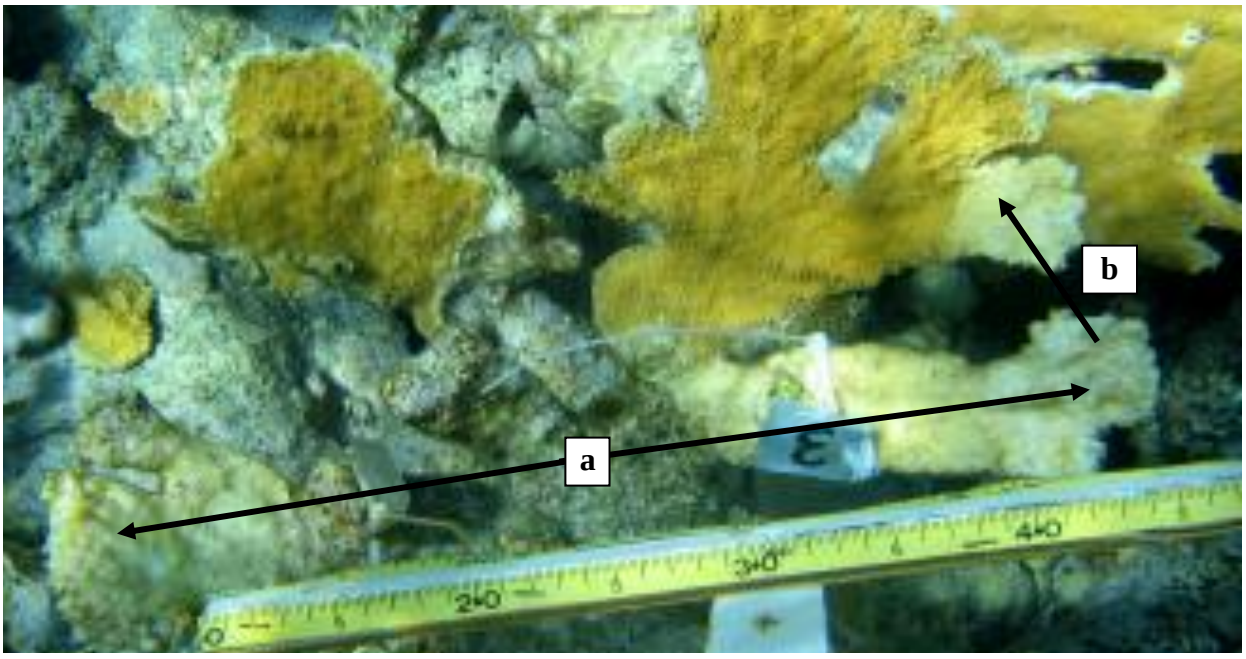
WPDa WPDb

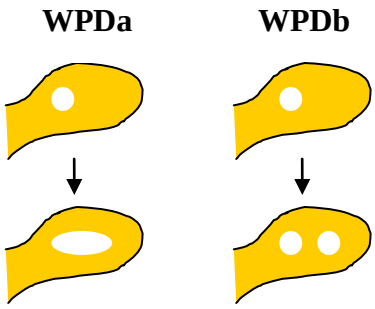


(a) How disease is marked and tagged (b) and (c) Demonstrate how height and width, respectively, were measured. The photo is of white pox disease taken on March 15th, 2004 at Admirals.



(a) A wooden, 50cm long T-bar used for large scale disease measurements or to measure disease that is difficult to access. (b) Smaller, more precise measurements are done using a plastic caliper.





Distinguishing WPDa from WPDb by method of disease spread.