Proceedings of the 11th International Coral Reef Symposium, Ft. Lauderdale, Florida, 7-11 July 2008
Session number 7

White band syndromes in *Acropora cervicornis* off Broward County, Florida: Transmissibility and rates of skeletal extension and tissue loss

A.J. Smith[1], J.D. Thomas[1]

1) Nova Southeastern University, Oceanographic Center, 8000 N. Ocean Drive, Dania Beach, FL 33004

Abstract. The high latitude thickets of *Acropora cervicornis* off Broward County flourish despite the presence of natural and anthropogenic impacts. These populations provide a unique study opportunity which stands out against the disease stricken areas of the Florida Keys. This study uses time sequenced photographs to examine how *A. cervicornis* is coping with white band syndrome stressors. Variables monitored include healthy colony skeletal extension rates, diseased colony skeletal extension rates, and tissue loss. The transmissibility of the white band syndromes was examined through tissue grafting experiments. Skeletal extension rates of healthy and diseased fragments averaged 0.94 ± 0.4 cm mo^{-1} and 0.79 ± 0.4 cm mo^{-1} respectively. Mean linear tissue loss from disease signs was 1.2 ± 2.5 mm d^{-1} or 3.7 ± 7.5 cm mo^{-1}. In transmission experiments, 72.5% of all trials involving direct tissue contact resulted in low or no disease sign transmission. *A. cervicornis* thickets in Broward County are growing similar to other studies in Florida but faster than other areas of the Western Atlantic while tissue loss from disease is lower. White band syndromes are always present in Broward County, but the low prevalence and transmission of the syndrome seems to limit its affect on the thickets.

Key words: White band disease, *Acropora cervicornis*, growth rates, tissue loss, disease transmission

Introduction

Surveys in Broward County, FL have discovered several thickets of *Acropora cervicornis* (Thomas et al. 2000, Vargas-Angel et al. 2003). Despite the presence of abundant natural and anthropogenic impacts, the *A. cervicornis* thickets are exceptionally healthy. These thickets provide a unique site for studying white band syndrome effects on acroporid populations when compared to the disease-stricken populations of the majority of the Caribbean including the Florida Keys National Marine Sanctuary (Vargas-Angel et al. 2003).

Recent observation of *A. cervicornis* populations directly off Fort Lauderdale, Lauderdale-By-The-Sea, and Pompano beaches in Broward County and other locations have shown that white band disease seems more complex than previously believed. For this reason the term white band syndromes is being used to include all cases. Three patterns of tissue loss have been observed during this study. The first is a fast moving, clearly defined white band of tissue necrosis next to 3-7 cm of denuded skeleton followed by algal successional stages. The second is similar to the first but it is a slower moving, well defined white band of denuded skeleton, followed by no more than 1-2 cm of denuded skeleton, then algae. Both of these tissue loss patterns resemble previously described patterns referred to as white band Type I (Gladfelter 1982, Peters 1984). White band type II has been reported in the Bahamas with an area of bleached tissue preceding the necrotic tissue (Ritchie and Smith 1998). White band type II was not seen during this study. The third observed tissue loss pattern looks similar to White Pox observed on *Acropora palmata*. This condition has irregular areas of denuded skeleton, termed 'patchy necrosis,' which spreads rapidly, regularly killing large portions of colonies. Similar patterns of rapid tissue loss were reported around the time when the first observations of white band disease occurred (Bak and Criens 1981). *Serratia marcescens* has been found to be a cause of similar looking patchy necrosis in *A. palmata* (Patterson et al. 2002). Williams and Miller discuss similar tissue loss patterns in their study at White Bank off Key Largo and refer to it as 'rapid tissue loss' (Williams and Miller 2005). It is still unclear if or how this rapid tissue loss is linked to the white band diseases. The presence of both in Broward's *A. cervicornis* thickets provides a unique opportunity for study.

Many studies including Atlantic and Gulf Rapid Reef Assessment surveys show that disease continues to be a considerable stressor on acroporid populations in the western Atlantic (Lang 2003, Sutherland et al. 2004, Weil 2004, Weil et al. 2006). Disease, low sexual recruitment, and anthropogenic impacts are to cause for the Caribbean acroporid corals to be listed as threatened on the Endangered Species Act (Anonymous 2005, Hogarth 2006) and listed as critically endangered under the International Union for the Conservation of Nature (IUCN) Red List criteria (Carpenter et al. 2008). This project was developed as part of a larger project investigating *A. cervicornis* thickets off Broward County, Florida for comparison to other western Atlantic acroporid populations. The primary goals of this project were to ascertain 1) healthy and diseased colony growth rates recorded in terms of linear skeletal extension not only for comparison to other populations but also to determine if there was a colony-wide influence from white band syndromes, 2) rates of tissue loss caused by white band syndromes recorded in terms of linear tissue loss, and 3) incidence of disease sign transmission from in-situ tissue grafting experiments confirmed via visual inspection.

Material and Methods

1) Skeletal Extension Rates – Colonies of *A. cervicornis* demonstrating no signs of white band syndromes and those with disease signs were identified for monitoring. On those colonies, branches with healthy tissue were marked 5-7 cm below the axial polyp with cable tied tags. Measurements from cable tie to branch tip were determined through photos calibrated with Coral Point Count with Excel extensions (CPCe) using the ruler photographed next to the branch (Kohler and Gill, 2006). All experiments were conducted at two different thickets concurrently from July through September 2007 and November 2007 through February 2008. Each site had 10 healthy and 10 diseased colonies tagged giving a total of 40 healthy fragments monitored and 40 diseased fragments monitored.

2) Tissue Loss Rates – Cable ties were placed near the tissue/skeleton interface on diseased colonies and used as a baseline from which progression of the disease front could be measured using CPCe in the same method described above. Linear movement of the tissue/skeleton interface was measured by calculating the difference between the cable tie and live tissue between measurements. This was monitored with the same method as above totaling 40 fragments.

To establish that the calibrated photo method is comparable to using calipers in-situ and an acceptable method for determining measurements, a small test was conducted. Five branches of *A. cervicornis* were tagged as they were in the experiment. Measurements were calculated using both methods. Caliper measurements were taken five times rotating around the axis of the branch. Photos were taken from five different angles. A two-sample paired t-Test was used to compare the two methods for each branch measured.

3) Transmissibility – Transmission of white band syndrome signs was observed with direct tissue contact via grafting experiments. There were four different trials 1) a diseased branch with a portion of an active lesion attached to a healthy colony, 2) a healthy branch attached to an active lesion on a diseased colony, 3) a healthy branch from a colony exhibiting disease signs attached to a healthy colony, and 4) a healthy branch from a healthy colony attached to a healthy colony. Branches were simply attached with cable tied tags and observed for disease sign transmission. Ten branches were used for each trial at each site giving a total of 160 fragments. Transmissibility was monitored through observation and photos. Each photographed tissue graft was assigned a classification based on the extent of transmission. The possibilities of transmission are outlined below (Fig. 1).

Classification	Explanation
None	No observed change in tissue color, integrity, or mucus output and no tissue loss on the 'healthy branch'.
Low	Margin of tissue contact showing whitening and possibly some tissue necrosis.
Moderate	'Healthy branch' clearly exhibiting tissue necrosis along grafting boundary.
Severe	Considerable tissue loss due to necrosis and most likely mortality of the transplanted fragment and host portion of the colony.

Figure 1: Chart outlining the four possible outcomes of a transmissibility experiment.

The following photos (Fig. 2-5) are examples of the possible transmission classifications.

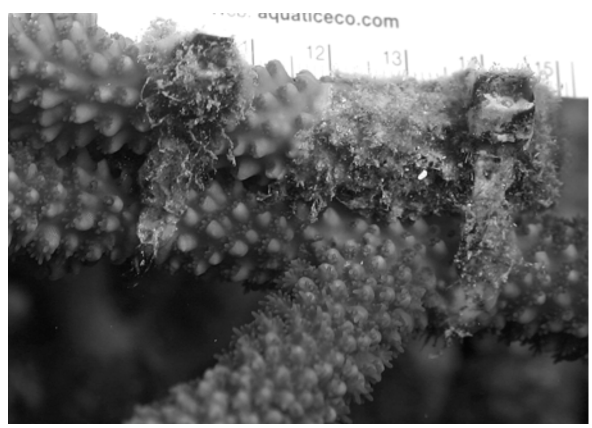

Figure 2: Two months after grafting, a host branch exhibits no transmission while the white band on the transplanted branch has ceased moving and the exposed skeleton was colonized by algae.

Figure 3: Nine days after grafting, a host branch exhibits low transmission while the transplant still has some healthy tissue.

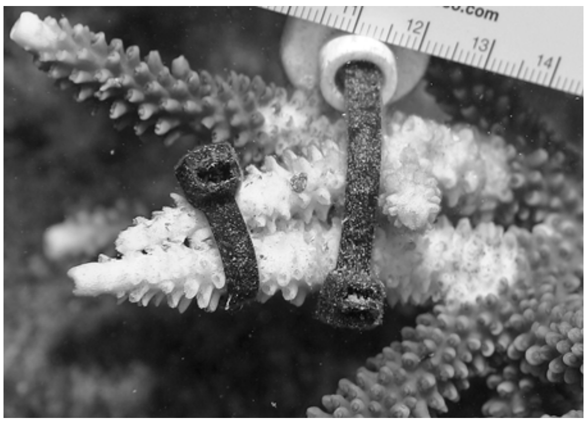

Figure 4: Nine days after grafting, a host branch is demonstrating moderate transmission while the transplant has lost almost all tissue.

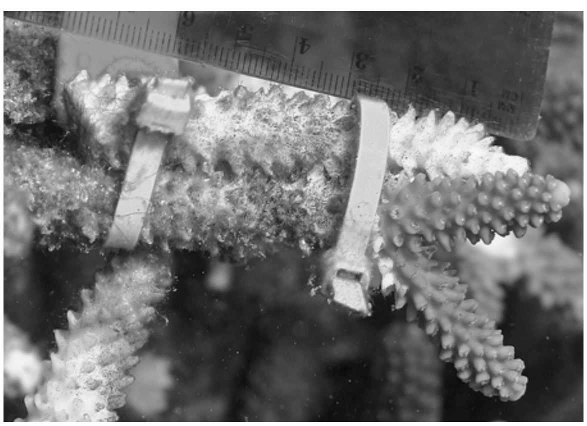

Figure 5: Fourteen days after grafting, a transplanted branch demonstrating severe transmission with no live tissue and the host branch is left with a small amount of tissue on the tip.

Results

The following chart (Fig. 6) lists the rates of healthy and diseased skeletal extension and tissue loss recorded during this experiment.

Measurement	Calculated Means
Healthy Skeletal Extension Rates	0.94 ± 0.4 cm mo^{-1}.
Diseased Skeletal Extension Rates	0.79 ± 0.4 cm mo^{-1}
Diseased Tissue Loss (linear)	1.2 ± 2.5 mm d^{-1} or 3.7 ± 7.5 cm mo^{-1}

Figure 6: Measurements reported as (mean ± SD).

A two sample paired t-test to compare healthy and diseased colony growth rates was conducted, with $\alpha=0.05$, $p=0.37$. This leads to the conclusion that there is no difference between the two skeletal extension rate means.

The comparison of five calibrated photos and five caliper measurements each of five different branches was analyzed using a paired t-test for sample means. With $\alpha=0.05$, the resulting p-values were 0.98, 0.82, 0.31, 0.61, and 0.64. All tests conclude that there is no difference between the two measurement means.

The following charts (Fig. 7-10) list the classifications of tissue grafting experiments in terms of percentage at each location at the termination of each experiment. The summer run was July through September. The winter run was November through February.

Diseased X Healthy				
Classification:	None	Low	Moderate	Severe
Scooter (S)	70%	10%	0%	20%
Oakland (S)	10%	0%	0%	90%
Scooter (W)	30%	10%	30%	30%
Oakland (W)	30%	60%	10%	0%

Figure 7: Transmission classifications for diseased branches attached to a healthy colony host. (S) = Summer (W) = Winter.

Healthy X Diseased				
Classification:	None	Low	Moderate	Severe
Scooter (S)	10%	0%	10%	80%
Oakland (S)	0%	0%	0%	100%
Scooter (W)	0%	10%	30%	60%
Oakland (W)	30%	20%	10%	40%

Figure 8: Transmission classifications for healthy branches attached to a diseased colony host.

Healthy from Diseased X Healthy				
Classification:	None	Low	Moderate	Severe
Scooter (S)	90%	10%	0%	0%
Oakland (S)	100%	0%	0%	0%
Scooter (W)	100%	0%	0%	0%
Oakland (W)	90%	0%	10%	0%

Figure 9: Transmission classifications for a healthy branch from a colony exhibiting disease signs attached to a healthy colony host.

Healthy from Healthy X Healthy				
Classification:	None	Low	Moderate	Severe
Scooter (S)	80%	0%	0%	20%
Oakland (S)	100%	0%	0%	0%
Scooter (W)	100%	0%	0%	0%
Oakland (W)	100%	0%	0%	0%

Figure 10: Transmission classifications for a healthy branch from a healthy colony attached to a healthy host.

Discussion

The table below (Fig. 11) lists annual growth rates of *A. cervicornis* recorded in several locations in the western Atlantic.

Growth Rate (cm y^{-1})	**Location**	**Source**
4	Dry Tortugas	(Vaughn 1915)
10.9	Key Largo, FL	(Shinn 1966)
11.5	Eastern Sambo, FL	(Japp 1974)
10	Key Largo, Florida	(Shinn 1976)
7.1	U.S. Virgin Islands	(Gladfelter et al. 1978)
3 to 4	Exuma, Bahamas	(Becker and Miller 2001)

Figure 11: Adapted from Atlantic Acropora Status Review (*Acropora* Biological Review Team 2005).

After converting this study's skeletal extension rates, healthy colonies have an average growth rate of 11.3 cm yr^{-1} and diseased colonies have an average growth rate of 9.5 cm yr^{-1}. These rates are within the same range of other Floridian *A. cervicornis* populations. Analyzing the data with a two sample paired t-test concludes that there is no statistically significant difference between the skeletal extension rate means of healthy or diseased colonies. This demonstrates that white band syndromes only impact the health of the coral at the location of the lesions and does not affect the rest of the colony's growth.

Not many studies have reported disease progression in terms of linear tissue loss. Williams and Miller (2005) discuss patterns of tissue loss similar to those observed in this study. This method of tissue loss termed 'rapid tissue loss' was seen during the study however, every effort was made to select corals that did not demonstrate rapid tissue loss. Corals that exhibited tissue loss patterns conforming to previous descriptions of white band syndrome type I were selected for this study. This study found mean tissue loss to be 0.12 ± 0.25 cm d^{-1}. Williams and Miller observed rates of 4 cm d^{-1} (Williams and Miller 2005) while other studies reported maximum rates of 2 cm d^{-1} (Antonius 1981, Gladfelter 1982, Peters et al. 1983). It seems that other factors must play a role in tissue loss rates such as temperature, season, nutrients, feeding scars, and recent branch breakage.

Transmission experiments involved four different tests. When a diseased branch was attached to a healthy colony the results were mixed. One site had 90% severe transmission. This ended up being an odd response. In all other treatments, the majority of transmission was not severe. It appears that some colonies may have the ability to resist transmission of the disease. Certain genotypes have been proven to be resistant to white band syndromes (Vollmer and Kline 2008). However, since there was some transmission, it appears there must be some sort of tranfer of causative agent or pathogen.

When a small piece of a healthy branch was attached to the lesion of a diseased colony the results were consistent. The majority of the transplants had severe transmission. There were some branches that did not transfer the disease signs (10% in the summer at one site and 30% in the winter at the other site). This supports studies that have shown that injuries such as a feeding scar from corallivorous snails or a fireworm (*Hermodice carunculata*) or in this case branch breakage can be a precursor to disease (Williams and Miller 2005, Sussman et al. 2003).

When a healthy branch from a diseased colony was transplanted to a healthy host the results were consistent. 90-100% of the trials showed no transmission of disease signs. This implies that a pathogen or causative agent is most likely present on healthy portions of a colony but are more concentrated on areas of active tissue loss caused by the white band syndromes.

When a healthy branch from a healthy colony was transplanted to a healthy colony the results were consistent. 100% no transmission was observed in all trials except one which had 20% severe. This further supports the idea from the previous paragraph that a causative agent is present on healthy portions of the colony.

During the transmission experiments, despite the effort to choose only those branches exhibiting disease signs resembling white band disease type I, when a transmission experiment lead to a transfer of disease signs, some branches demonstrated disease signs of white band type I and others the rapid tissue loss discussed earlier. Although each transmission experiment had differing results, 72.5% of all the tissue grafts had low or no transfer of disease signs.

When comparing methods using calipers or calibrated photos for measuring a branch of *A. cervicornis*, all paired t-tests for sample means resulted in p-values that conclude there is no statistically significant difference in the two methods means. The photo method was chosen to be less invasive. The only contact with the coral is the installation of the tag. If the tag is connected firmly with a cable tie gun, it will prevent tag movement and tissue irritation, the fragments can still thrive. Several tagged fragments started to overgrow their tags in just a few months.

In summary, the skeletal extension rates in Broward County are similar to previously recorded rates in Florida. These rates however, seem to be significantly higher than other areas of the western Atlantic. Tissue loss rates due to white band syndromes are faster than corals are growing but less than previously reported rates from the Florida Keys and other locations.

Transmissibility of white band syndromes appears to be more severe when involving small fragments but transmission is not always severe, supporting studies that have shown that tissue damage can be a precursor to disease. A significant portion of tissue grafts did not demonstrate disease signs transfer when directly attached to a diseased colony supporting other findings that colonies can be resistant. Future research such as genotyping of Broward's thickets could prove interesting regarding colony resistance. Using the calibrated photo method will allow future studies to make consecutive measurements with minimal interference with the normal growth and health of *A. cervicornis* colonies. When compared to other western Atlantic populations this study demonstrates that the *A. cervicornis* thickets in Broward County are healthy and growing faster. Despite the constant presence of white band syndromes, the low incidence and low transmission of disease signs seems to play a significant role in the good health of these populations. There is still much to be understood of the white band syndromes and the thickets of Broward County provide an excellent study site for such research.

Acknowledgement

Thank you to Dr. Kiho Kim, Dr. Drew Harvell, and Dr.Cathie Page for allowing me to present this research at the 11th ICRS. It was a great opportunity. Thank you to my advisors Dr. Jim Thomas, Dr. Esther Peters, Dr. Bernardo Vargas-Angel, and Dr. Pat Blackwelder for their time and patience to see this project through to the end. Thanks to all my volunteers: Erin Hodel, Joe Penta, Jen Hester, Dave Portnoy, Kate Koontz, Mike Lehman, and Liam Murphy. I couldn't have done it without you offering your time. Thanks to Kyle Miller and Lisa Gregg for all their help in obtaining the proper permits for this study (SAL 07SRP-606). Also, thanks to Lance Robinson, Dr. Dave Gilliam, and Dr. Dick Dodge for helping facilitate this project.

References

Acropora Biological Review Team (2005) Atlantic *Acropora* Status Review Document. Report to National Marine Fisheries Service, Southeast Regional Office. March 3, 2005. 152 p + App.

Anonymous (2005) Endangered and threatened species: proposed threatened status for elkhorn coral and staghorn coral. Federal Register. 2005;70:24359–24365

Antonius A (1981) The 'band' diseases in coral reefs. Proc 4th Int Coral Reef Sym 2:7–14

Bak RPM, Criens SR (1981) Survival after fragmentation of colonies of *Madracis mirabilis*, *Acropora palmata* and *A.cervicornis* (Scleractinia) and the subsequent impact of a coral disease. Proc 4th Int Coral Reef Sym 2:221–227

Becker LC, Mueller E (2001) The culture, transplantation, and storage of *Montastraea faveolata, Acropora cervicornis*, and *Acropora palmata*: what have we learned so far. Bull Mar Sci 69:881-896

Carpenter KE, Abrar M, Aeby G, Aronson RB, Banks S, et al. (2008) One-Third of Reef-Building Corals Face Elevated Extinction Risk from Climate Change and Local Impacts. Science 321(5888):560-563

Gladfelter EH, Monahan RK, Gladfelter WB (1978) Growth rates of five reef-building corals in the northeastern Caribbean. Bull Mar Sci 28:728-734

Gladfelter WB (1982) White-band disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. Bull Mar Sci 32:639–643

Hogarth, WT (2006) Endangered and threatened species: final listing determinations for the Elkhorn Coral and Staghorn Coral. Federal Register, pgs. 26852–26861

Jaap WC (1974) Scleractinian growth rate studies. Proc Fl Keys Coral Reef Wrkshp. FL Dept Nat Res Coastal Coordinating Council, p 17

Lamberts AE (1974) Measurements of alizarin deposited by coral. Proc 2nd Int Coral Reef Sym 2:241-244

Lamberts AE (1978) Coral growth: alizarin method. in Stoddart DR, Johannes RE (eds) Coral reefs: research methods. UNESCO, Monogr Oceanogr Methodol 5:523-527

Lang JC (2003) Status of coral reef in the western Atlantic: Results of initial surveys, Atlantic and Gulf rapid reef assessment (AGRRA) Program. Atoll Res Bull 496

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

Peters EC (1984) A survey of cellular reactions to environmental tress and disease in Caribbean scleractinians corals. Helgol Meeresunters 37:113–137

Peters EC, Yevich PP, Oprandy JJ (1983) Possible causal agent of 'white band disease' in Caribbean acroporid corals. J Invertebr Pathol 41:394–396

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

Shinn EA (1966) Coral growth-rate: An environmental indicator. J Paleont 40:233-240

Shinn EA (1976) Coral reef recovery in Florida and the Persian Gulf. Environ Geol 1:241-254

Sussman M, Loya Y, Fine M, Rosenberg E (2003) The marine fireworm *Hermodice carunculata* is a winter reservoir and spring–summer vector for the coral-bleaching pathogen *Vibrio shiloi*. Environ Microbiol 5:250–256

Sutherland KP, Porter JW, Torres C (2004) Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar Ecol Progr Ser 266:273-302

Thomas JD, Dodge RE, Gilliam DS (2000) Occurrence of staghorn coral (*Acropora cervicornis*) outcrops at high latitudes in near shore waters off Fort Lauderdale, FL, USA. In: Proc 9th Int Coral Reef Sym, Bali, Indonesia. Abstr:86

Vargas-Angel B, Thomas JD, Hoke SM (2003) High-latitude *Acropora cervicornis* thickets off Fort Lauderdale, Florida, USA. Coral Reefs 22:465-473

Vaughan TW (1915) The geological significance of the growth rate of the Floridian and Bahamian shoal-water corals. J. Wash. Acad. Sci. 5:591-600

Vollmer SV, Kline DI (2008) Natural Disease Resistance in Threatened Staghorn Corals. PLoS ONE 3(11): e3718

Weil E (2004) Coral reef diseases in the wider Caribbean. in E. Rosenberg & Y. Loya (eds.) Coral Health and Disease. Springer, Berlin, pp 35-68

Weil E, Smith G, Gil-Agudelo D (2006) Status and progress in coral reef disease research. Dis Aquat Org 69:1-7

Williams DE, Miller MW (2005) Coral disease outbreak: pattern, prevalence and transmission in Acropora cervicornis. Mar Ecol Progr Ser 301:119-128