Award Number: NA12NOS4820073
Program Office: NOS Office of Ocean and Coastal Resource Management (OCRM)
Project Title: **Towards predicting coral disease patterns: quantifying coral responses to disease in the US Virgin Islands**
PI: Marilyn Brandt
Project Progress Report (Due 3/31/15)
Reporting period: 07/01/12 – 12/31/14

Project Abstract from Proposal:

Coral diseases are a major driver of coral decline within reef habitats of the U.S. Virgin Islands, including those within the protective boundaries of federal and territorial reserves. **The overall objective of this study is to identify consistent patterns in the responses of corals and coral populations to disease. Our central hypothesis is that corals respond predictably to conditions leading to diseased states at both physiological and ecological scales. The ultimate goal of this research is to use these patterns in order to develop predictive tools of disease incidence and impact.** We will apply techniques including gene expression profiling (transcriptomics) and histology to identify common responses of corals to disease across multiple diseases and species. Coral disease patterns at the population scale will also be investigated in response to variable levels of local and regional stressors through high frequency, spatially extensive surveys. This proposal will result in a greater understanding coral mortality processes driven by disease in the USVI, while also providing scientific training for a local graduate student. This information can be used to identify critical management interventions to prevent or mitigate the impacts of coral disease, directly addressing the CRCP objective of managing for resilience to climate change in the U.S. Virgin Islands. Results will be communicated to the scientific and management community through presentations at local venues and publications.

Summary of Project Results:

Results of this project have identified patterns of responses to coral disease that were unknown before and these responses can be used for future predictions of coral health states. This project contributed to the professional development of two graduate students, resulted in a PhD dissertation, a Masters thesis and one peer-reviewed publication thus far, contributed to six presentations at local, national and international conferences, and provided information to the USVI public and management communities concerning local issues of coral disease. We expect one additional peer-reviewed publication (currently in review) that used data from CTD casts collected during the project to result from the project, as well as three additional peer-reviewed publications (to be submitted) from the three complementary research aspects of the project that included: 1) Field sampling, 2) Expression profiling, and 3) Histological analysis.

Results of field sampling included quantification of white plague disease prevalence at a high temporal resolution across twelve sites. Results of disease surveys identified spatial patterns suggesting white plague disease is density dependent and transmissible at the colony-to-colony scale and possibly at the reef-to-reef scale. These preliminary findings were presented at the 2013 AMLC meeting and final results are scheduled to be presented at the 2015 AMLC meeting and concurrently submitted for publication.

Expression profiling was used to better understand the physiological response of corals to disease, and we applied *O. faveolata* and *A. palmata* cDNA microarrays to four diseases, White Plague (WPl) and Yellow Band Disease (YBD) affecting *O. faveolata* as well as White Band (WB) and White Pox (WPx) affecting *A. palmata*. Our results show dramatic core responses across diseases, as well as distinct disease-specific functional responses. Expression profile differences were noted across all diseases. Healthy and diseased samples show distinct profiles. Colonies exhibiting symptoms of YBD and WPl exhibited strong inflammatory responses as well as strong expression of genes involved in apoptotic responses. In *A. palmata*, WPx also induced a strong apoptotic response. All four diseases, WB, WPx, YBD, and WPl, exhibited increases in immune and defense responses as well as decreases in chemical defenses. These results are contained within the successfully defended doctoral dissertation of C. Closek and are in process of being submitted for peer-review.

Results of the histological analysis indicated that among WPl and YBD states, there was a consistent negative effect on coral reproduction due to disease. Zooxanthellate densities were also negatively affected by YBD but not WPl; however, healthy-appearing tissue on WPl affected colonies showed low zooxanthellae numbers, suggesting a possible whole colony response. Mucocytes were not visible in the original WPl samples; therefore additional samples were collected and processed. It is expected that results of analysis of these samples will be submitted for publication in the summer 2015.

Project Timeline and Milestones from Proposal

Activities	J	A	S	O	N	D	J	F	M	A	M	J	J	A	S	O	N	D
Project preparation	x	x																
Disease surveys		x	x	x	x	x	x											
Tissue collections			x	x	x													
Histology processing					x	x	x	x	x	x	x							
Survey data analysis								x	x	x	x	x						
Expression profiling											x	x	x	x				
Histology data analysis											x	x	x	x	x	x		
Profiling data analysis														x	x	x		
Data integration, publication preparation																x	x	x

Extension Period: January 1, 2014 – December 31, 2014

Activities	J	F	M	A	M	J	J	A	S	O	N	D
PI Brandt maternity leave		x	x	x	x							
Survey data analysis finalization	x					x	x					
Histology processing (UVI – RA)	x	x	x	x	x							
Histology data analysis						x	x	x	x			
Expression profiling finalization	x	x	x	x								
Profiling data analysis					x	x	x	x	x			
Data integration, publication preparation								x	x	x	x	x

Actions completed during the project under the Timeline "Activities" heading

Disease surveys: Disease surveys were completed in August, September, October, November, and December of 2012 and in January, February, March, April and May of 2013. Videos from surveys were processed by one observer and verified independently by a second observer. Analysis of field survey data collected during this project was completed and is currently being combined with results of surveys completed subsequent to May 2013 for presentation at the 2015 AMLC meeting and for publication.

Tissue collections: Collin Closek traveled to St. Thomas to perform tissue collections with Dr. Brandt and M. Sevier between June 5th and 13th, 2013. Collections of samples occurred at three sites. These samples were processed for both histology and expression profiling as detailed below. Additional samples were collected in May 2014 as discussed below.

Histology processing: All tissues for histology collected in 2013 were shipped to Abigail Renegar, director of the histological laboratory at Nova Southeastern University, for the development of histology slides which were received in November 2013. Ms. Moriah Sevier completed the histology Nova Southeastern University workshop hosted by Dr. Esther Peters July 22-26, 2013. At this workshop, entitled "Understanding Corals from the Inside Out: Comparative Histopathology," she received training in the analysis of histological slides. Processing of the slides by Ms. Sevier began in November 2013 and continued through 2014. When it was apparent that coral mucocytes were not visible in WP samples, additional samples were collected in May 2014 and were shipped for processing in November 2014 when the NCRI lab was ready to receive them. These samples were processed by NCRI and returned to UVI for analysis in January 2015. Due to a family illness, the UVI graduate RA could not complete the analysis of these additional slides. However, the final results are expected in summer 2015 when the combined histopathology results will be submitted for publication.

Expression profiling data analysis: All samples collected in 2013 were processed immediately and then sent out for sequencing in 2014. Data were analyzed Collin Closek, the UC-Merced PhD student, and presented as part of his doctoral dissertation defense. Findings were incorporated into his dissertation and have been documented in a manuscript which is currently circulating among authors prior to submission to a peer-reviewed journal. The abstract of this manuscript is presented under findings.

Findings:

Hypothesis 1: Patterns of coral disease incidence

Reef-scale white plague disease trends

Disease prevalence data at all Rapid Status Assessment (RaStA) sites (Table 1) were tested for effects of site and time using a Repeated-Measures ANOVA (RM-ANOVA). Disease prevalence was found to significantly vary through time (RM-ANOVA, $F = 17.7$, $p < 0.05$), by site ($F = 16.17$, $p < 0.05$), and there was a significant interaction between site and time ($F = 2.87$, $p < 0.05$), indicating that disease patterns through time were not consistent among sites. Disease prevalence was significantly higher and more consistently observed at deeper, shelf-edge sites versus shallow, nearshore sites. Mid-depth sites were variable in how much disease was seen and

how consistently; Seahorse showed consistent but low (< 0.5%) levels of disease, South Capella showed consistent but occasionally elevated (> 1%) levels of disease, and East French Cap only had disease in November 2012.

A non-parametric Spearman's ρ correlation analysis performed to test relationships between WPl disease prevalence and physical characteristics collected using CTD casts also revealed a positive correlation between disease and increasing depth ($\rho = 0.52$, $p < 0.0001$). Disease was also positively correlated with salinity ($\rho = 0.29$, $p = 0.0068$) and negatively correlated with turbidity ($\rho = -0.39$, $p = 0.0003$). These results were surprising, as we expected to see higher disease in more turbid, nearshore environments. Instead, we observed higher levels of disease on deep, offshore reefs where there were no indications of inputs from land such as high turbidity or low salinity.

Coral cover was higher in the offshore, shelf edge sites, therefore disease prevalence recorded in RaStA transects was compared with coral cover recorded in annual monitoring of sites through the Territorial Coral Reef Monitoring Program (TCRMP). Surprisingly, no significant relationship was found between average disease prevalence recorded in RaStA video transects and coral cover recorded in TCRMP transects (Linear regression: $F = 2.61$, $p = 0.15$, $R^2 = 0.22$). However, > 77% of disease cases in the TCRMP database were recorded on the species *Orbicella faveolata* (formerly *Montastraea faveolata*) and *O. franksi* (formerly *M. franksi*), and these two species comprise > 60% of coral cover at these sites. Average disease prevalence was therefore compared with the percent cover of these two species and a significant positive relationship was found (Linear regression: $F = 12.0$, $p < 0.05$, $R^2 = 0.57$).

Table 1. Characteristics of RaStA sites including 1) coral cover, 2) depth, 3) reef area, and 4) distance to nearest reef. 1) Coral cover measured in TCRMP transects. H – High coral cover, L – Low coral cover, "~" estimated because no TCRMP data. 2) Depth measured in RaStA surveys. S – Shallow, M – Mid-depth, D – Deep. 3) Reef area measured in GIS. S – Small, L – Large. 4) Distance to nearest reef measured in GIS. N – Near, F – Far. *Indicates TCRMP long-term monitoring site.

Site	Coral Cover (%) H > 20 | L < 20		Depth (m) S < 15 | M 15-30 | D > 30		Reef Area (hectares) S < 100 | L > 100		Dist. to nearest reef (m) N < = 100 | F > 100	
Brewers Bay*	31.6	H	7	S	4	S	25	N
Cas Cay		~H	13	S	27	S	700	F
College Shoal*	33.7	H	31	D	1000	L	1	N
E. French Cap		~L	19	M	150	L	1500	F
Flat Cay*	18.3	L	13	S	1	S	200	F
Grammanik*	33	H	38	D	28	S	100	N
Hind Bank*	25.2	H	39	D	48	S	100	N
Meri Shoal*	32.6	H	29	M	175	L	820	F
Perseverance*	12.6	L	10	S	5	S	60	N
S166	13	L	42	D	1100	L	1	N
S. Capella*	15	L	22	M	60	S	300	F
Seahorse*	17.3	L	18	M	1	S	750	F

WP prevalence was significantly positively correlated with contiguous reef size measured in GIS ($r = 0.76$, $p < 0.05$) (Table 1) and a significant positive correlation ($r = 0.44$, $p < 0.05$) also existed between the linear distance between sites (as measured in GIS) and the difference in

average disease prevalences between sites in RaStA surveys (i.e., as distance between sites decreased, the temporal pattern of disease prevalence became more similar). These results suggest that components of intra-reef connectivity including distance and metapopulation size influence disease prevalence. While this evidence is suggestive, connectivity as predicted by water currents, and not linear distance, may better explain disease patterns.

There was no overall relationship between WPl disease prevalence and temperature ($\rho = -0.12$, $p = 0.2766$), which was also surprising as many coral diseases have been found to have significant relationships with temperature (Miller and Richardson 2014). However, temperature differed significantly among sites and at seven sites we noted that peak disease corresponded with peak seasonal temperatures. When we performed site specific analyses, disease was positively correlated with temperature at three sites: a shallow site ($r = 0.89$, $p < 0.05$), a mid-depth site (($r = 0.65$, $p < 0.05$), and an MCE site ($r = 0.75$, $p < 0.05$). This suggests that temperature may play a role in disease but that it is not the primary factor influencing disease incidence.

Within-reef white plague disease trends

Diseased colonies seen in video transects were analyzed for spatial distributions within sites using the time stamps of each diseased colony within a video transect. For all transects, the distance between every diseased colony and its closest other diseased colony (i.e., nearest neighbor) on a transect was measured in seconds (e.g., the distance between a disease colony identified at minute 2:00 and a diseased colony identified at minute 2:10 was measured as 10 seconds). These "distances" were then summarized in frequency distributions as the average proportion of diseased neighbor colonies found within 30 second time intervals. Patterns of diseased colony distribution were similar across sites, with the majority of neighbor diseased colonies being found within 1 minute of a selected colony (Figure 1). These patterns suggest a clumped pattern of disease colonies, potentially indicating disease transmission among colonies.

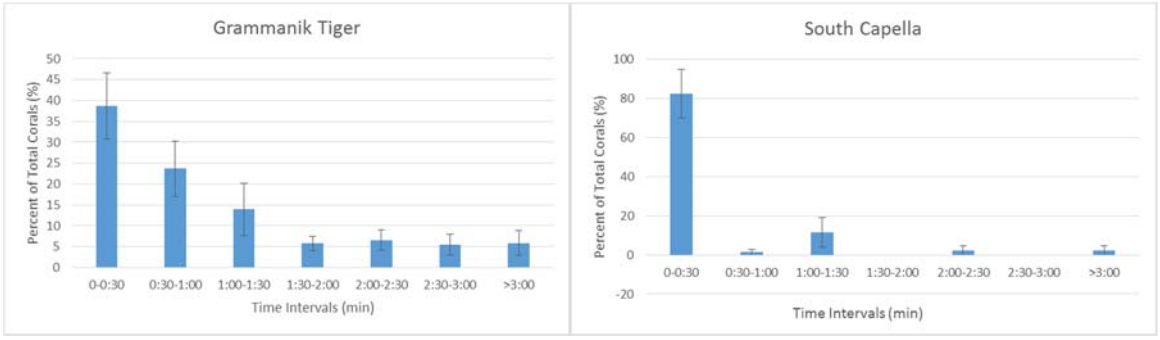

Figure 1. Spatial distribution of diseased colonies within video transects at two Rapid Status Assessment sites. Spatial distribution presented as the average proportion of diseased neighbor colonies found in that time category. Left) Spatial distribution of diseased colonies at a deep site (135 fsw), Right) Spatial distribution of diseased colonies at mid-depth site (80 fsw).

Summary Hypothesis 1: Our original hypothesis stated that *the spatial and temporal patterns of coral disease incidence follow spatial and temporal patterns of exposure to local stressors, primarily sedimentation*. Results of our surveys of WPl disease across multiple sites through

time indicated that this was not the case. Instead, our results suggest that WPl may be a density dependent disease that shows indications of transmission among colonies at the colony-to-colony scale and possibly the reef-to-reef scale. WPl was highest at deep high coral cover sites where *Orbicella* was the dominant species group. Sites located in nearshore turbid environments showed the lowest amount of disease, however these sites also tended to have the lowest coral cover. Recommendations for future studies would be to expand the number of shallow, high coral cover sites that are surveyed monthly for disease to include high coral cover sites in impacted versus unimpacted watersheds. However, due to pressures on shallow nearshore reefs and increasing development on the USVI it may be difficult to find such high coral cover reefs remaining.

Hypothesis 2: Overlap of gene expression profiles and physiological biomarkers

Histopathology results

Processed histology slides were assessed for reproductive indices including # of oocytes and # of spermaries present. No spermaries were identified in any slides. The number of oocytes varied significantly by health state (Non-parametric Kruskal-Wallis test: $\chi^2 = 30.37$, $p < 0.0001$). Diseased tissues showed significantly lower numbers of oocytes versus tissue collected from healthy colonies and tissue collected from apparently healthy tissue on diseased colonies (Figure 2). In addition, there was no significant difference between diseased states.

Slides were additionally analyzed for number of polyps and zooxanthellae density. Number of polyps did not differ among coral samples of different health status. Average zooxanthellae density varied significantly among coral health status (Kruskal-Wallis test: $\chi^2 = 138.61$, $p < 0.0001$). As expected, zooxanthellae densities were significantly lower in yellow band diseased tissue versus all other health status tissue (Figure 3). Interestingly, average zooxanthellae densities were significantly lower in healthy tissue on white plague diseased coral versus the other health status categories, although it was still significantly greater than yellow band diseased tissue (Figure 3). This suggests the possibility of a whole colony reaction to the disease.

Mucocytes were not visible in original WP samples. Therefore, additional WP samples were processed in order to specifically examine mucocyte density. These slides were received in January 2015; however analysis of slides was postponed due to a family illness of M. Sevier, the UVI graduate research assistant trained in histology. PI Brandt has acquired professional development funds to travel to NCRI and collaborate with a pathologist on the analysis of the remaining slides if M. Sevier continues to be unavailable.

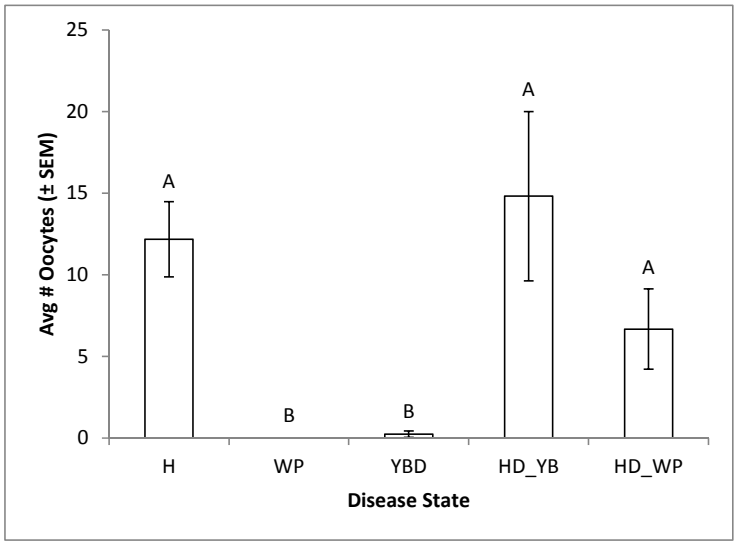

Figure 2: Average number of oocytes in tissue sections of different disease states. "H" = healthy (n = 10), "WP" = white plague diseased (n = 6), "YBD" = yellow band diseased (n = 7), "HD_YB" = healthy tissue on a yellow band diseased colony (n = 7), "HD_WP" = healthy tissue on a white plague diseased colony (n = 2). Letters indicate significant groups as determined by pair-wise non-parametric Wilcoxon tests.

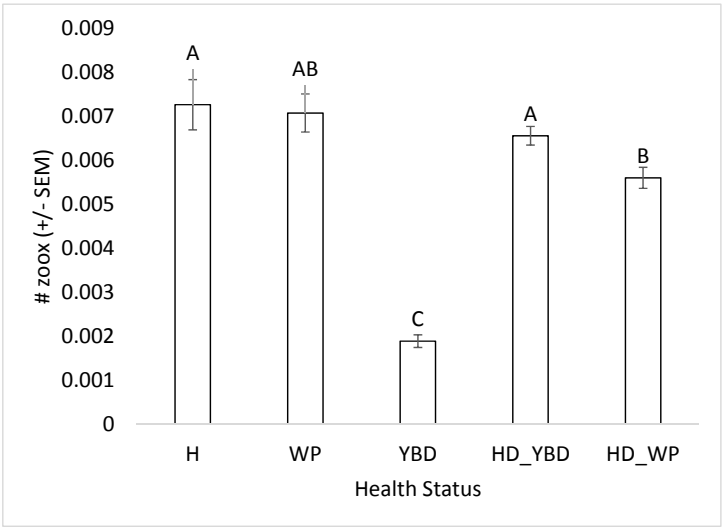

Figure 3. Average zooxanthellae density in tissue sections of different disease states. Health status abbreviations and sample sizes as in Figure 1. Letters indicate significant groups as determined by pair-wise non-parametric Wilcoxon tests.

Gene expression profiles

Abstract of manuscript to be submitted for publication - Only a few decades ago, *Orbicella* spp. and *Acropora palmata* were some of the most abundant corals in the shallow reefs of the Caribbean. These species are major reef building corals that contribute to the reef structure and provide many ecosystem services. Increasing environmental and anthropogenic stressors, such as

high temperatures and nutrient effluents, have intensified coral disease outbreaks. Disease has ultimately contributed to significant die-offs of these important species throughout the region. Using a transcriptomic approach, to better understand the physiological response of corals to disease, we investigated the following questions: (1) How do *O. faveolata* and *A. palmata* respond to more common diseases? (2) Which genes exhibit enhanced expression under poor health conditions? (3) Do core responses and/or disease-specific responses exist? To examine these questions, we applied *O. faveolata* and *A. palmata* cDNA microarrays to four diseases, White Plague (WPl) and Yellow Band Disease (YBD) affecting *O. faveolata* as well as White Band (WB) and White Pox (WPx) affecting *A. palmata*, in the US Virgin Islands.

Our results show dramatic core responses across diseases, as well as distinct disease-specific functional responses. Expression profile differences were noted across all diseases. Healthy and diseased samples show distinct profiles. Colonies exhibiting symptoms of YBD and WPl exhibited strong inflammatory responses as well as strong expression of genes involved in apoptotic responses. In *A. palmata*, WPx also induced a strong apoptotic response. All four diseases, WB, WPx, YBD, and WPl, exhibited increases in immune and defense responses as well as decreases in chemical defenses. In this study, a transcriptomic approach allowed us to distinguish between differences in the responses of hosts under varying diseases while also revealing common responses across diseases. This multi-species, multi-disease perspective provides a simultaneous snapshot of host response and illustrates the complexity of disease dynamics in the reef.

Summary Hypothesis 2: Our original hypothesis stated that *significant overlap of expression profiles and physiological biomarkers exists among diseases and is consistently different from that of controls*. Our histological findings support our hypothesis; specifically we found that there was significant overlap of reproductive indicators between the WPl and YBD samples and that the diseased samples were consistently different from controls. Our expression profiling results also support our hypothesis and showed that all four diseases tested (WPl, YBD, WB and WPx) exhibited increases in immune and defense responses and that expression profiles were distinctly different between diseased and control states.

Project Outputs

Manuscripts published:

1. Closek CJ, S Sunagawa, MK DeSalvo, YM Piceno, TZ DeSantis, EL Brodie, MX Weber, CR Voolstra, GL Andersen and M Medina. 2014. Coral transcriptome and bacterial community profiles reveal distinct yellow band disease states in Orbicella faveolata. ISME J (2014) 1-12. *This manuscript was a component of C. Closek's dissertation work.*

Manuscripts in review

1. Smith TB, J Gyory, ME Brandt, J Miller, J Jossart, and RS Nemeth. Mesophotic coral reefs are unlikely climate change refugia. Under review at Nature Climate Change. *This manuscript included analyses of CTD casts made with CRCP funds.*

Manuscripts in preparation

1. Brandt ME, TB Smith, M Sevier and E Clemens. *Spatial and temporal variability of white plague disease on Caribbean reef systems*. To be submitted to Coral Reefs.

2. Closek C, AV Magana, BSK Kamel, EM Diaz-Almeyda, ME Brandt, and M Medina. *Comparative analysis of coral transcriptomes across four diseases.* To be submitted to ISME J.
3. Brandt ME and M Sevier. *Histological responses of corals to multiple diseases*. To be submitted to Diseases of Aquatic Organisms.

Conference presentations:

1. Brandt ME, E Clemens, TB Smith and J Gyory. *Spatial and temporal variability in white plague disease in Caribbean reef systems.* Oral presentation at the Association of the Marine Laboratories of the Caribbean (AMLC) Meeting, Discovery Bay, Jamaica, 17-21 June 2013.
2. Closek C, M Brandt, and M Medina. *Coral gene response across four Caribbean diseases*. Poster presentation at the Ecology and Evolution of Infectious Diseases Meeting, Penn State University, 20-24 May 2013.
3. Brandt M, T Smith and E Clemens. *The ecology and etiology of a coral killer.* Oral presentation at the annual Virgin Islands Experimental Program to Stimulate Competitive Research (EPSCoR) conference, 12 May 2014, St. Thomas, VI.
4. Smith TB, Gyory J, Brandt M, Brandtneris V, Jossart J, and D Holstein. *Mesophotic coral ecosystems in the Caribbean and eastern Pacific and their potential as climate change refugia*. Oral presentation at the International Mesophotic Workshop – Eilat 2014, 26-31 October 2014.
    - *Co-PI Smith included data from video transects and CTD profiles collected under this project in this talk.*
5. Sevier M, A Primack, T Smith, and M Brandt. Relationships between terrestrial inputs and *Gorgonia ventalina* aspergillosis. Oral presentation at the Western Society of Naturalists Meeting, Tacoma WA, November 13-16, 2014.
    - *The UVI RA, Moriah Sevier, included data analyzed using her histopathology training received under this project in this talk.*
6. Brandt M, TB Smith, E Clemens, and M Sevier. *Disease in the Deep: Coral white plague in Mesophotic coral ecosystems*. Accepted to give oral presentation at the Association of the Marine Laboratories of the Caribbean Meeting, 18-22 May 2015, CARMABI, Curacao.

Student Progress and outputs:

- Moriah Sevier, the UVI MMES student supported by the project under the advisement of PI Brandt, was originally scheduled to defend her Masters thesis and graduate by May 2015, however she has taken a leave of absence for the spring 2015 semester due to family health issues. Her planned graduation is now scheduled for December 2015.
- Collin Closek, the UC-Merced Ph.D. student supported by the project under the advisement of Co-PI Medina, successfully defended his Ph.D. on 8 May 2014 date and graduated from UC-Merced 20 December, 2014. He has since accepted a post-doctoral fellow position at the School of Aquatic and Fishery Sciences at the University of Washington. Title of dissertation: "The Good, the Bad, and the Ugly: Assessing Reef Health and Coral Diseases through Associated Microbes and Host Response" (https://etda.libraries.psu.edu/paper/23494/).

Information dissemination and Community Outreach:

- **Earth Day 2013**: Distribution of research information took place at the Earth Day Celebration on 20 April 2013 at Coral World on St. Thomas.
- **Science Seminar Series at Reef Fest 2013:** On November 23, 2013, the UVI Center for Marine and Environmental Studies in cooperation with Coral World hosted "Reef Fest 2013; a Celebration of the Reef" at Coral World on St. Thomas, USVI. Over 300 people attended the event. A "Science Café Seminar Series" was featured during the event and included informal interactive talks about the health of the reef (Figure 4). PI Brandt presented at 11am: "MICROBES: The Scariest Monsters on Coral Reefs?" This interactive seminar included information on coral health and disease, a description of the CRCP funded project, and an interactive "Name that Disease" game. Co-PI Smith presented at 12pm: "Coral Reef Change and Ciguatera Fish Poisoning in the USVI." This talk included data from the VI Territorial Coral Reef Monitoring Program and data collected as part of this CRCP project. Overall, approximately 100 people attended the seminars.
- **Science Seminar Series at Reef Fest 2014**: On November 9, 2014, the UVI Center for Marine and Environmental Studies in cooperation with Coral World hosted "Reef Fest 2014; a Celebration of the Reef" at Coral World on St. Thomas, USVI. This year, attendance was more than twice that of Reef Fest 2013; over 800 people attended the event. A "Science Café Seminar Series" was again featured during the event and included informal interactive talks about the health of the reef (Figure 3). PI Brandt presented at 12pm an updated version of "MICROBES: The Scariest Monsters on Coral Reefs?" This interactive seminar included information on coral health and disease, a description of the CRCP funded project, and an interactive "Name that Disease" game.
- **Fact Sheet:** A coral health and disease one page fact sheet specific to the US Virgin Islands (Figure 5) was designed by PI Brandt and the graduate RA for dissemination in May of 2014 (*note this component was inadvertently excluded from progress reports 4 and 5). Fact Sheets were printed and distributed at the Virgin Islands Experimental Program to Stimulate Competitive Research (EPSCoR) conference, 12 May 2014, St. Thomas, VI which was attended by > 50 people from the local scientific and management community. The fact sheet was additionally distributed at Reef Fest 2014.

Figure 4. Left: PI Dr. Marilyn Brandt presenting the seminar "MICROBES: The Scariest Monsters on Coral Reefs" at 2013 Reef Fest. Right: Co-PI Dr. Tyler Smith presenting the seminar: "Coral Reef Change and Ciguatera Fish Poisoning in the USVI."

Other Outputs

- CTD profiles collected under this project were used in UVI Graduate Student Jonathan Jossart's Masters thesis project. Mr. Jossart successfully defended his thesis entitled "Modeling a Yellowfin Grouper (*Mycteroperca venenosa*) Spawning Aggregation with Passive Acoustic Telemetry on the Grammanik Bank, St. Thomas, US Virgin Islands" in November 2014, and he graduated in December 2014.
- Disease prevalence values from video surveys taken in the latter half of 2012 were used as validation data for the Masters thesis project of Elizabeth Clemens. Ms. Clemens successfully defended her thesis entitled "Investigating the Transmissibility of the Coral Disease, White Plague" in April 2013 and graduated in May of 2013.

Figure 5. Coral Health and Disease Fact Sheet for Distribution to Management Community.