Project Summary:

The primary goals of this project were to increase our understanding of the coral disease pathogens associated with black band disease and white plague type II, and to document the short and long term effects of disease outbreaks of each type on reefs of the Florida Keys. The project included both laboratory and field research which were complementary to each other. The project period was extended an additional year due to hurricane activity which shortened the field season in 1999.

The project focused on two coral diseases, both of which are active on reefs of the Northern Florida Keys. These are black band disease and plague type II. The field activity spanned a length of 40 km of reef tract, and included both field manipulative experiments and repeat monitoring.

Significant Results:

Significant results are summarized below. Many of these results have been published in peer-reviewed journals and presented at conferences and other venues (listed at the end of the report as deliverables). Several more papers will be forthcoming.

Black band disease:

The research supported by this project on black band disease is a continuation of ongoing work in Richardson’s lab to understand the etiology, epizootiology, and pathogenesis of this coral disease. Several new findings arose from the supported research:

a. We previously demonstrated that black band disease is composed of a complex microbial consortium that functions like a laminated microbial mat such as those found in illuminated, sulfide rich environments. New results from this project support the finding that light intensity is important in causing the black band microbial consortium to form a compact assemblage that characterizes the disease state. These findings were based on results of both in situ and laboratory studies on the effect of manipulated (and natural) light intensity on the motility patterns of two of the black band consortium members (Phormidium corallyticum and Beggiatoa). It was determined that the black band cyanobacterium (P. corallyticum) is very low light tolerant, thus forms the dense mat system which constitutes black band. These results agree with previous results on the photosynthetic capabilities of this organism. One new paper is in review on these findings (Viehrnan and Richardson).

b. The disease dynamics of the two coral diseases are very different. In the case of black band disease, three patterns have been recorded within our 10 year data base: a) seasonal, occurring between late April and late October (most common); b) no incidence for an entire year (two years); c) first yearly case observed well past normal (late October observed in 2000). Continued monitoring has revealed very slow colonization by scleractinian corals of the exposed surface of coral colonies partially killed by black band. Our earlier results, following study colonies for five years, had documented colonization primarily
by turf algae, macroalgae, and soft (octo-) corallians. The new data suggest that scleractinian recolonization occurs after 5 to 8 years. The original coral colonies at times exhibit extremely slow tissue regrowth, but in no case has a colony completely recovered.

c. One aspect of the project was not successfully completed. This was the artificial induction of black band disease on the reef by spiking reservoir populations with nutrients. The first attempt (1998) failed because our nutrient dosing apparatus was stolen. We had identified reservoir environments (biofilms of the major black band disease microorganisms in sediment patches on healthy black band disease susceptible corals) and had begun monitoring oxygen profiles of both nutrient-dosed (experimental) and non-dosed (control) patches using in situ oxygen microelectrodes. The dosing apparatus was stolen after four days; at this time weather prevented us from reinstalling the apparatus. The next year (1999) we planned to repeat the study on a less visited reef (Algae reef) – after extensive searching, however, we found no reservoir populations. The third (funding extended) year (2000) again we found no reservoir patches, on either Algae reef or our original study site (Horseshoe). In this year, no black band was observed on any of these reefs until October.

Plague type II:
The research supported by this project on plague type II is also a continuation of ongoing work in Richardson’s lab to understand this very different coral disease. Several new findings arose from the supported research.

a. One of the proposed tasks of the proposed work was to document that a pathogen isolated from the disease was the plague pathogen. This was successfully accomplished and published (Richardson et al., 1998). The plague pathogen isolated from these reefs is potentially a new genus of bacterium. As a collaborative project, we have assessed pathogens isolated from plague outbreaks in St. John, USVI (as well as repeated outbreaks in Florida) and determined that the same pathogen is responsible.

b. Plague, although extremely virulent and responsible for killing hundreds of small colonies during outbreaks, has much less effect on the Florida reefs than the less virulent black band. We have documented much recovery (regrowth of tissue in partly killed colonies) and high rates of recruitment of the most susceptible species (Dichocoenia stokesi). This work will be written up and submitted for peer-review and publication this year.

c. Both the plague pathogen and the black band cyanobacterium have growth temperature optima of 30°C or above. Nutrient data have shown a positive correlation of black band with elevated nitrite and lowered salinity.

Summary: In summary, we have accomplished the goals of the project with the exception of the black band initiation experiment. As listed below, we have generated 6 peer-reviewed papers, with a 7th in review and several to be written. We have 10 published abstracts (with associated presentations) of this work at national and international conferences. Additionally, several highly prestigious invitations were made and accepted to present this research. As noted below, such presentations included one at the National Academy of Sciences (with an associated invited peer reviewed paper) and one on Capital Hill in Washington DC at the prestigious U.S. Global Change Program monthly seminar series.

Deliverables:

1. Peer-reviewed publications:


2. Invited seminars, presentations at workshops, etc. (specific to this research)

1.) May, 1998. Invited speaker/participant, CariCOMP workshop on coral disease pathology and monitoring, Trinidad and Tobago

2.) November, 1998. Invited speaker, Duke University Marine Lab seminar series, Beaufort NC

3.) March, 1999. Invited speaker, University of Florida Department of Microbiology seminar series, Gainesville, FL


6.) April, 2000. Invited plenary speaker, 25th Annual Eastern Fish Health Workshop (Special Symposium on Coral Diseases), Plymouth, Mass

3. Talks at national and international meetings with associated abstracts:


March 15, 2001

Dr. Bill Kruczynski
US EPA
5550 Overseas Highway – Main House
Marathon, Florida 33050

Dear Dr. Kruczynski:

Enclosed is my final report for the Special Project you funded on coral diseases. The project end date was 12/31/00. According to my grant paperwork this report is due on 3/31/01.

We have generated quite a few deliverables, noted in the report, and have several backlog papers to write up. If you wish, I’ll let you know as these become published.

Thanks very much for funding this work, and especially thank you for the extension.

Best regards,

[Signature]

Laurie Richardson
Associate Professor of Biology