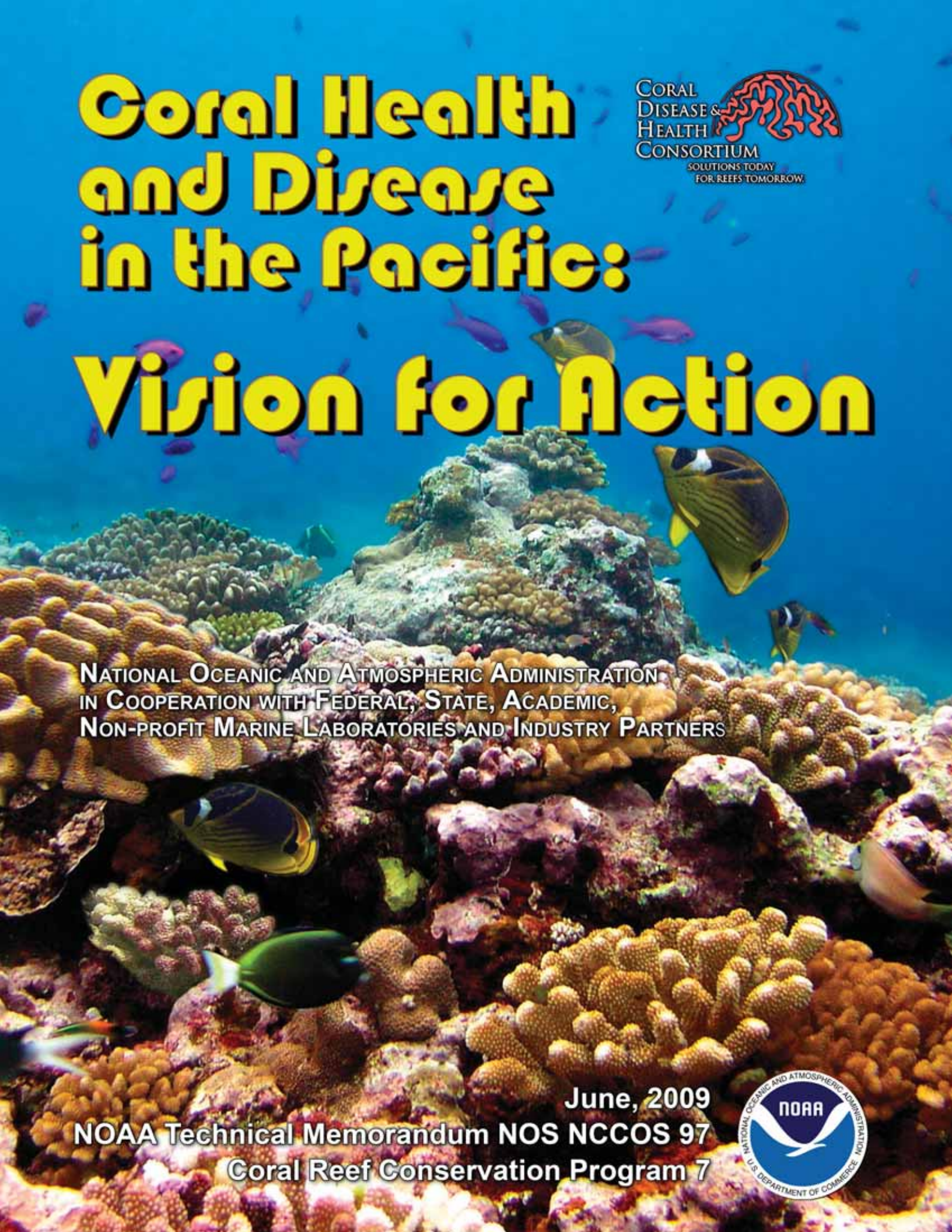
Coral Health and Disease in the Pacific:

Vision for Action



Coral Disease & Health Consortium
Solutions Today for Reefs Tomorrow

National Oceanic and Atmospheric Administration
in Cooperation with Federal, State, Academic,
Non-profit Marine Laboratories and Industry Partners

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United States Department of Commerce	National Oceanic and Atmospheric Administration	National Ocean Service
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EXECUTIVE SUMMARY

"We are studying arguably the most complicated ecosystem on the face of the Earth, and it is under serious threat…..We have an incredibly important message, if this ecosystem dies, if this ecosystem is otherwise perturbed to an extent that it cannot recover, not only does it spell potential disaster for this spaceship we call Earth, but there is no less than 80 emerging economies, nations that are entirely or nearly entirely dependent on coral reef ecosystems whether it be for the economy or for the subsistence." (Gary Ostrander, Vice Chancellor for Research at the University of Hawaii, opening remarks)

Shallow coral reefs in the IndoPacific contain the highest diversity of marine organisms in the world, with approximately 1500 described species of fish, over 500 species of scleractinian corals, and an estimated 1-10 million organisms yet to be characterized (Reaka-Kudla et al. 1994). These centers of marine biodiversity are facing significant, multiple threats to reef community and habitat structure and function, resulting in local to wide-scale regional damage. Wilkinson (2004) characterized the major pressures as including (1) global climate change, (2) diseases, plagues and invasive species, (3) direct human pressures, (4) poor governance and lack of political will, and (5) international action or inaction.

Signs that the natural plasticity of reef ecosystems has been exceeded in many areas from the effects of environmental (e.g., global climate change) and anthropogenic (e.g., land use, pollution) stressors is evidenced by the loss of 20% of the world's coral reefs (Wilkinson 2004). Predictions are that another 24% (Wilkinson 2006) are under imminent risk of collapse and an additional 26% are under a longer term threat from reduced fitness, disease outbreaks, and increased mortality. These predictions indicate that the current list of approximately 30-40 fatal diseases impacting corals will expand as will the frequency and extent of "coral bleaching" (Waddell 2005; Wilkinson 2004). Disease and corallivore outbreaks, in combination with multiple, concomitant human disturbances are compromising corals and coral reef communities to the point where their ability to rebound from natural disturbances is being lost.

Pacific reefs, in general, have been considered in good condition and most resource managers have no real concern about coral disease (regardless of whether the cause is an infectious agent or anthropogenic pollution); this 'good' condition status may only be a reflection of inadequate information for many areas. In fact, the U.S. state of coral reef ecosystems 2005 report (Waddell 2005) refutes this by showing an increase in disease reports throughout U.S. states, territories and freely associated states and documents a growing perception that coral disease may be a threat to Pacific reefs. Increased findings of coral disease from the World Bank Coral Disease Working Group (WBCDWG) and NOAA/USGS disease monitoring programs provide ample evidence that disease is present in Pacific reefs and may, in fact, be increasing. For example, in 2004, the WBCDWG recorded 12 syndromes at six survey sites; including four syndromes that had not been previously recorded (Waddell 2005). Some experts warn that Pacific coral reefs are on a trajectory of degradation similar to that experienced in the Caribbean Basin where coral reefs are decimated.

Though the proliferation of coral reef diseases is a sign of a sick ocean environment, this realization can also be used as an instrument of change. There is growing evidence that the increased severity and prevalence of these diseases is directly linked to human activities, such as pollution washing off the land, heat stress to corals, and through overfishing of organisms that can control macroalgae and pest species like corallivores. By developing an understanding of disease dynamics, causal links can be determined and factors driving these system failures can be identified. Developing such an understanding can move us toward the goal of *health management and preventative care* for coral reefs.

Recognizing the need for a strategic plan of action to combat and avert a possible health crisis for Pacific Reefs, the Coral Disease and Health Consortium (CDHC) convened a workshop to help organize and coordinate a U.S. scientific effort focused specifically on coral health issues in the Pacific. The goal was to develop an action plan that would enable regional scientific efforts to detect, identify, characterize, and manage coral diseases in the Pacific. This report documents the proceedings of this workshop: ***Coral Health and Disease in the Pacific: Vision for Action***. The goals of the workshop were to:

- Synthesize the state of knowledge of Pacific coral diseases;
- Discuss the concepts and principles of disease, their use in investigating causation and how this can be applied to corals;
- Characterize the difficulties in identifying, defining and managing disease in coral; and
- Develop a *Strategic Research Plan* that
  - identifies knowledge gaps that impede understanding coral disease mechanisms (i.e., pathology), and limit elucidation of causes, significance or control of coral disease (i.e., epidemiology);
  - recommends directed research and education to fill these knowledge gaps;
  - standardizes methods for investigating coral disease outbreaks considering both biotic and abiotic etiologies;
  - addresses issues relative to the management of coral reef resources; and
  - fosters collaboration among CDHC partners, stakeholders, key marine resource management agencies, and regional networks in the Pacific.

The workshop incorporated diverse viewpoints from experts representing biomedicine, coral disease, toxicology, and resource management. The opening day focused on presentations from 14 position papers (Appendix VI), to provide context and concepts for the break-out group discussions. These presentations covered key topics that included:

- ***What do we currently know about coral diseases in the Pacific?***
- ***What lessons have we learned from Caribbean disease outbreaks?***
- ***Diagnostic methods, systems biology and leveraging post-genomic technologies***
- ***Emerging diseases, disease outbreak investigations and ecological epidemiology***
- ***How to integrate science with social, economic and political values?***

The participants were then assigned to one of four groups: (1) Coral Cellular Physiology & Pathology; (2) Coral Toxicology & Ecological Epidemiology; (3) Pathology of

Infectious Disease; and (4) Preventing and Responding to Coral Disease in the Pacific Region: Management Perspectives. Each team was charged with identifying key impediments and making recommendations for strategic research priorities that comprise this *Strategic Plan of Action*.

The opening presentation provided a context for the workshop, identifying and discussing the features that set U. S. Pacific reefs apart from those in the Caribbean and other parts of the world. The U. S. Pacific and Atlantic reef areas have similar political histories and both were exploited through plantation agriculture. Recently, the economies of both regions are shifting from agriculture to tourism, and to a lesser extent fisheries, mining and logging. All of these activities have an impact on the region's coral reefs. The Pacific coral reefs have the highest biodiversity with at least 200 genera and 580 recognized species of coral. The hub of this diversity is located in Southeast Asia around Indonesia and the Philippines in portions of the Indian and Pacific Oceans referred to as the **'coral triangle'**. This diversity then diminishes from one island to the next across the Pacific to the east. The eastern Pacific (e.g., Pacific coast off central and South America) has the lowest coral diversity in the Pacific, followed by Hawaii. However, Hawaii has the highest regional endemicity, with an estimated 25% endemic organisms that inhabit these coral reefs. The region is also distinguished by resource management practices that are shaped by traditional cultural knowledge and practices that remain active in many of the Pacific islands today. These customs and tribal governance are unique in that U. S. Pacific Islanders perceive their natural resources as valuable and an integral part of their lives. In areas where these practices occur, their influences have successfully guided community-based management practices that contrast "western" ideas and National and regional management practices. The most obvious difference between the Caribbean and Pacific is the sheer area of coral reef habitat, the number of islands and atolls that exist in the Pacific, and the amount of open ocean between these islands, all of which greatly exceed those found in the Atlantic. This in itself creates a degree of isolation for many of the Islanders. However, this vastness and high biological diversity creates logistical, biological and cultural challenges to research as the Pacific Islander population is spread over numerous islands often at great distances, resulting in diluted scientific resources and insufficient personnel to monitor and combat any potential disease crisis in their reefs.

Our most comprehensive records (1972-2005) of coral disease are compiled in the Global Coral Disease Database (WCMC Wcmc 2006) developed through a partnership between NOAA and UNEPs World Conservation Monitoring Centre (WCMC). Dr. Andy Bruckner presented a report on the global diversity and distribution of coral diseases (Appendix VI). To date, this effort has documented reports of over 40 coral diseases from the western Atlantic, 28 from the Indo-Pacific and 5 from the Red Sea, and covers 63 countries. Over 150 species representing 39 genera have been observed with disease. In the Caribbean, this translates to 80% of all taxa (41 species of scleractinian, 8 gorgonians, 2 hydrozoans) being afflicted with disease. In the Indo-Pacific 97 species (approximately 17%) from 34 genera have been identified with disease and this is on the rise. These numbers reflect a 25% increase in genera and 45% increase in species number since 1999, with 7 new genera in the Indo-Pacific observed with disease over the last 5

years. Recent surveys conducted in strategic locations across the Indo-Pacific (Australia, the Philippines, American Samoa, Northwest Hawaiian Islands (NWHI) and elsewhere) illustrate the widespread, global distribution of coral diseases with prevalence varying from a low of 0.14% in American Samoa to 0.5% in the NWHI and highs of 10% along the Great Barrier Reef (GBR) to 14% in the Philippines (see white papers in Appendix VI of this report by Willis, Aeby and Work). In these areas, over the last five years, regions previously unaffected are reporting disease, while in other locations (i.e., GBR) the percentage of reefs affected by disease has increased, and several new disease manifestations have been reported since 2002 (Willis et al. 2004). Based on this and other information, it is reasonable to conclude that diseases in the Indo-Pacific are undergoing a rapid expansion in range and types of disease and ***now*** is the time to recognize the signs of a pending problem and take action.

Our understanding of coral diseases and thus ability to combat the declining health of our reefs is limited by our lack of understanding of the basic biology and physiology of coral hosts, and their responses and tolerances to changes in their environment. We are at the cross-roads---we can remain in the dark ages of medicine, as our understanding of coral disease has been described, or we can take advantage of the established principles of wildlife veterinary medicine and the technologies of a post-genomics era, apply them to coral health, and accelerate the evolution of this field….not only to determine the cause, but how to manage disease in the reef environment. The approach undertaken by the CDHC and strategies recommended by the workshop participants can help move the coral disease field into the 21st century, through implementation of wildlife and human medical approaches and tools. This requires enhanced funding, improved training and capacity building efforts, education initiatives, and development of new tools and information resources.

Recommendations:

- **Provide Competitive-based Grant Opportunities to Fill Knowledge Gaps.** Our ability to understand coral disease pathology is hampered by a limited knowledge of molecular and cellular physiological functions of corals. An understanding of these critical features of coral biology could be rapidly advanced by tapping into a knowledge-base and skills that exist in the wider research community, is just beginning to be applied to corals. Funding is a key impediment to filling these gaps. Partnerships with funding agencies (NSF, EPA NIEHS) to offer directed grant opportunities can provide the impetus needed to engage a broader research community in developing a knowledge-base of coral cellular physiology.

- **Adopt Model Coral Species for Research.** Identify representative reef building coral species from the Atlantic and Pacific that could be used in research studies to better characterize normal coral physiology and biological stress responses, then support culture facilities to propagate these corals (i.e., living stock collection), and make specimens readily available to researchers.

- **Adopt an *Ecological Epidemiology* Approach to Identify Risk Factors and Assess their Contribution to Coral Reef Degradation.** The principles and methodologies of epidemiology can be used to identify and quantify risk factors that impact coral health (e.g., toxins and pollutants that make corals more susceptible to disease) and quantify the contribution of the various factors leading to adverse health effects. Implementation requires developing standardized methods and tools to detect and track biological responses of corals which can focus diagnostic efforts, and help direct and prioritize management and research actions toward risk reduction.

- **Develop a Systematic Approach to Investigate and Study Diseases in Corals.**
  - Identify and recommend standardized approaches to systematically investigate coral diseases, including a system of nomenclature and terminology to describe diseases, survey approaches and laboratory techniques to provide compatibility among data.
  - Develop a protocol for responding to coral disease outbreaks, train regional and local teams in disease investigative methodologies, including documenting case histories, assessing the area and extent of an outbreak using appropriate survey techniques, sampling techniques for specific laboratory analysis, and implementing systematic investigations in response to unusual coral disease outbreaks and mortality events.
  - Develop a bioinformatics system to track outbreaks, synthesize case data to identify drivers in outbreaks and provide data in a format easily accessible to researchers and resource managers.

- **Manage Coral Reefs to Reduce Stressors that may make Corals more Vulnerable to Disease.** Managers often discount the study of coral disease because conceptually they believe disease is part of nature or even if causes of disease are identified, nothing can be done so why bother. However, management of disease in animal populations cannot occur in absence of information. Indeed, several tools are available to manage disease in human and animal populations (including wildlife), and these tools were developed precisely because targeted research identified the key interactions between agent(s), host, and their interactions with their environment that drive the occurrence of disease. Similar concepts also apply to corals. The key to managing coral health and mitigating disease impacts is not through stereotypic routes of medication, vaccination, and treatments, but rather by identifying causes of coral diseases, pathogenesis and factors that may modulate the resulting pathologies, including interactions with manageable anthropogenic and environmental stressors. The most controllable environmental factors are those associated with land-based sources of stressors; understanding disease dynamics can identify control points in a disease cycle that can also be used in management strategies. This will require researchers working collaboratively with key marine resource management agencies and regional networks in the Pacific such as the U.S. All Islands Coral Reef Initiative Coordinating Committee.

- **Create and Support Advanced Educational Opportunities.** There is a critical need to build scientific capacity in the field of coral pathology and disease management skills in reef resource management. These programs should include development of advanced degree programs in coral pathology, cellular physiology, toxicology or epidemiology as well as continuing education in specialty topics (e.g., disease identification for resource managers; disease investigation methods; environmental forensics) for professionals (i.e., resource managers).

- **Develop Guidance for the Proper Handling and Containment of Corals in Infectious Disease Experiments.** Most experimental studies involving corals have occurred under conditions that, in a medical setting, would be unacceptable. Typically, corals are placed in water tables, exposed to a suspect agent, and monitored for development of gross lesions whereupon the conclusion is made that agent 'A' caused disease 'B'. Critical oversights in such experiments include lack of environmental controls (e.g. use of water tables with little monitoring of what microorganisms go in and out of the system), lack of morphologic follow-up to confirm that a gross lesion is indeed due to the putative infectious agent being investigated, and lack of knowledge regarding the normal physiology and biota of the host being investigated. Other studies have been conducted in the field with no containment or control over the dispersal of the inoculum into the surrounding environment. These types of experimental studies are analogous to attempting to elucidate the cause of a farm animal disease by conducting studies in the barnyard. An important outcome of this workshop was the **recommendation by the participants to the CDHC to accept the following guidelines** for the care and handling of corals in experimental settings:
  - Field Challenges using agents grown in a laboratory setting should not be done. Just as we would not grow bacteria or viruses in the lab and infect livestock, wild animal populations or humans (vaccines being the one controlled exception), in open systems with no containment, nor should we do it in corals.
  - The export of laboratory reared coral back into the field is not currently recommended, until suitable tests are available for assuring these coral do not pose a threat to the wild populations.
  - The need for biosecurity and bio-containment guidelines for conducting laboratory challenge experiments with candidate infectious agents and toxicants is recognized and it is recommended that CDHC establish a steering committee to develop these guidelines that are consistent with existing guidelines for handling and containment of infectious agents in wildlife as well as protocols for hazardous materials handling.

- **Foster the Development of a Cohesive Coral Disease Research Community.** The goals outlined in this document can only be achieved through a cohesive group of people focused on common goals and a passion for healthy coral reefs. The participants of this workshop recommend that the CDHC provide a focus for cross-cutting priority research needs and a framework for interaction and collaboration among the coral disease research community.

OPENING REMARKS
(Transcribed from this Workshop's Opening Address presented by Gary K. Ostrander)

When I was thinking about my comments last night, since I do publish in the field of coral reef biology, I realized that I had a unique opportunity today to talk to the leadership in my scientific community. This is in addition to my responsibility as the Vice Chancellor to welcome you to Hawaii. This is significant to me in that you are a special group of scholars. You were all invited to this meeting because you are outstanding researchers and many of you represent and/or collaborate with top research groups and laboratories in the world.

I realized that if I actually had something significant to say, that it might impact you and in doing so it might extend to others in the field. So, my initial comment is as follows:

When are we going to get our act together as a research community?
At best, we're pathetic. If that puts you on the defensive or makes you uncomfortable, that's my intent. We are studying, arguably, the most complicated ecosystem on the face of the Earth and it is under serious threat. Yet, we are disproportionately under-funded, in terms of funding whether it's in the United States, at NSF or EPA, in Europe, Australia, etc. We are disproportionably under-represented in the top research journals: *Science, Nature, PNAS, Cell* and even in the second and third-tier research journals.

What's the problem, what are we doing wrong?
I tell my graduate students and staff that I don't have a problem if you come to me with your problems, but I do have a problem if you don't come to me with a solution or a starting point for a solution. So, I am going to hold myself to the same standard this morning and I'm going to offer the following for your consideration.

What are the causes of the problem that we currently face and what is a possible solution?
Are we stupid? I don't think so. I've met a lot of very smart people in this field. I didn't start out in this field--I'm a guest. I started my career in cancer biology. When I think of the solution and I think of the causes, I turn my attention to the zebrafish community. Does anybody in this room not know what a zebrafish is? Of course you do!

Twenty years ago, when I started working in fish cancer, nobody was working on zebrafish. In fact, I would argue that there was probably an order of magnitude, if not two orders of magnitude, more people working on coral reefs than on zebrafish. Yet in 20 short years, zebrafish have become recognized as a predominant model for developmental biology. They were on the cover of *Science* a few weeks ago. They continue to be in the top journals. If you sit on the panels at NIH, NSF and EPA, etc. in the United States, it's zebrafish work that's getting funded. They (i.e. the zebrafish community) did two things well. One is they asked important questions. I don't think that's a problem for us. But, secondarily and most important, they came together as a community very early on. They supported each other. Before there was an Internet, they put together the zebrafish handbook, the bible for zebrafish researchers. They went out of their way to make it easy for people to join the community, to work in the community. If someone had a line of zebrafish, if someone had a cell line, you could ask them for it, they would FedEx it to you and they would pay the shipping, and then they'd call you to find out if you had questions or needed help. When you sat on a NIH Study Section or an NSF panel and a zebrafish grant would come through, it would get the "regular" reviews from everybody. However, from the fish people, whether they supported the grant or not, they supported the particular application of fish, they supported the individual, and they were an advocate for the type of work. Zebrafish scientists joined the boards in the major societies in North America and throughout the rest of the

world as well as journal editorial boards. And though they could be just as critical and just as scathing, and just as nasty as we are to our colleagues, they didn't do that. When they were critical, they were critical in a positive and a productive way. Look at where it's gotten them.

Sadly, this is not the case for the coral reef community. I am on the Editorial Board for *Aquatic Toxicology*. When's the last time you saw a coral reef paper in *Aquatic Toxicology*? It's a top journal, there's lots of coral work in aquatic toxicology. It's amazing, when you send a coral paper out to someone who is not a coral biologist, you get a reasonable review, when you send it out to a coral biologist, you usually get a pretty scathing review, because everybody's defending their own territories. I see this when I sit on study sections at EPA or NSF. When I was in the fish community, you wanted a fish person to review your stuff, not because they would automatically approve it. No, you wanted them because you would get a constructive review if they didn't.

In my brief time in the coral reef community, I have learned that I don't want coral biologists reviewing my work. I am willing to take my chances with competent reviewers who don't know anything about coral biology.

Five years ago, approximately, Cheryl convened the first of these workshops. They have tremendous potential to help the coral community. Out of that workshop came the idea: could we sequence the coral genome? So, Craig Downs, Craig Venter, Claire Fraser, Steven Salzsberg and I got together to write a 'white paper'. The middle three individuals are some fairly significant names in the human genome community. Let me tell you a little bit about that effort and where it got us. The first question we had to address was which species do we sequence? That created quite a bit of controversy in the coral reef community. In the end, *Porites lobata* was suggested. And, parenthetically, I will tell you that was not my first choice even though I was leading the effort to write the white paper. Once the species was selected it was necessary to get letters of support. NHGRI had mandated that we be able to demonstrate that the community was going to rally around the organism selected and would actually use it because of the high costs of sequencing a genome. I'm not going to name names, but I have to tell you that I was really disappointed that when I went to colleagues who had "lost" as they viewed it, in their efforts to get "their species" sequenced, that they didn't provide letters of support, even when I emailed them a couple times.

We ended up submitting an application with 45 letters, and it was a good application. However, I was further disappointed that after it was submitted to NHGRI, some of our colleagues, hopefully nobody in this room, took it upon themselves to go to members of the panel to lobby for their own species and to disparage the rest of us and the species that had been selected. This did not send a good message to the NIH. They eventually came back to us with their decision: they said they would support pilot sequencing on three species, which they did. Ironically, one of the species they came back to us with was not even among the final three we had selected ourselves. Obviously, more politics was involved. At one point, somebody from the coral community went to the officials overseeing the website that runs the listserv we were using to solicit letters of support for the effort and wanted everything taken down because they presupposed members of our team were using this effort to patent sequences to make drugs. That is, we were doing this as a money making venture. Nobody came to ask me if that was the intent. No one came to me and said, 'Hey, we heard this rumor, is this true.' No one ever talked to us about it, they just went around us. Clearly, it was not true and it was not even possible if you understood the NHGRI program.

In the end, NHGRI provided funds and we did pilot sequencing on three coral species. I am told they are still planning to select one of them for full sequencing. However, I am also told it is not a

priority right now. What's I have heard through back-channels is that there's concern on the part of NHGRI as to whether our community is going to embrace whatever species that was selected and whether it would be used. Some people mistakenly assumed that if it was not their species that was sequenced, that it would have no value to their research. Now whether they are just ignorant of RT-PCR or some of the other technologies, or whether they are just being selfish, I don't know, but it's a real problem for our community.

So let me conclude. I challenge you, all of you, to take the first steps to create a more cohesive community, a community that works together. People will follow by example, it's been done before. We have an incredibly important message, if this ecosystem dies, if this ecosystem is otherwise perturbed to an extent that it cannot recover it spells disaster not only for this spaceship we call Earth, but for more than 80 emerging economies, nations that are entirely or nearly entirely dependent on coral reef ecosystems for their existence. This is an incredible opportunity, an incredible moment. One of our colleagues posted something on the coral list-serve that said in part '….the problem with the coral reef community is that they eat their young.' It's a nice analogy, I think its time we do something to reverse it. So with that I will conclude my welcome from the University of Hawaii.

Thank you for your time."

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June 19, 2006

PREFACE

Over the past three decades, coral reefs worldwide have experienced major changes in structure and function due to numerous anthropogenic stresses and natural factors. In particular, the prevalence and severity of coral diseases and the diminishing health condition among corals have contributed to unprecedented declines in live coral cover and altered the productivity of coral reef ecosystems. The Caribbean is referred to as a "hot spot" for diseases due to a rapid emergence and high virulence of new diseases, an increasing geographic distribution and wider host ranges of known diseases, and an increased frequency of epizootic events. The number of diseases and their distribution across the Indo-Pacific also appears to be on the rise. Increased anthropogenic stress, overfishing, changing environmental conditions associated with global climate change, and the synergistic effect of multiple stressors have been implicated as significant factors contributing to escalating disease levels. However, our ability to address the recent increases in coral disease is hampered by a paucity of relevant epizootiological data, an incomplete understanding of the mechanisms responsible for the diseases and their consequences, and few diagnostic tools to help managers evaluate and mange diseases. Responding to this growing threat requires improved scientific understanding and tools to: (1) detect and assess trends in coral diseases at scales relevant to scientific investigation and policy development; (2) determine the causes and consequences of increasing disease frequency and distribution; and (3) evaluate possible management options to mitigate the effects of disease on coral reef ecosystems and their users.

In 1998 the United States government issued Executive Order 13089 on coral reef protection. This Order called for the creation of the U.S. Coral Reef Task Force (US CRTF 2008) to develop, in partnership with federal agencies whose actions affect U.S. coral reef ecosystems, measures needed to understand, manage and restore coral reef ecosystem, with emphasis on reduction of impacts from pollution, sedimentation and fishing. The CRTF developed the National Action Plan to Conserve Coral Reefs (March 2000), which outlines 13 major themes focused on improving our understanding of reefs and quickly addressing human impacts to these ecosystems. This Plan, together with the National Strategy and the Coral Reef Conservation Act of 2000 has outlined a realistic strategy to improve the condition and health of coral reefs, and has helped focus our conservation efforts, especially in waters of the United States, our territories and commonwealths and the Freely Associated States. One of the key initiatives of the CRTF was the creation of the Coral Disease and Health Consortium (CDHC), focused specifically on coral health issues, with emphasis on the diagnosis and etiology of coral diseases and bleaching. The CDHC is a network of field and laboratory scientists, coral reef managers, and agency representatives devoted to understanding coral health and disease. Currently over **150 partners,** including three federal agencies (EPA, DOI, NOAA), academia, non-profit groups and industry are working *to understand and address the effects of natural and anthropogenic stressors on corals in order to contribute to the preservation and protection of coral reef ecosystems.*

In January 2002 the CDHC convened its first official meeting where recommendations to address the major gaps in coral disease and health research were identified and are detailed in *Coral Disease and Health: a National Research Plan* (Woodley et al. 2003). The major needs include:

- Establishing standard terminology, methodology and protocols;
- Expanding knowledge in basic coral physiology, biology and disease etiology;
- Developing model coral species; and
- Developing a centralized data/knowledge system, website, repository and core diagnostic facilities.

The CDHC working closely with partners have focused on five main activities. These include:

- Developing standardized procedures based on medical principles that clearly define the terminology, pathology and diagnostic criteria;
- Developing diagnostic tools to assist researchers in identifying coral stressors;
- Applying advanced technologies in functional genomics, proteomics and systems biology to expand our knowledge in coral health and disease dynamics;
- Providing local response capabilities to carry out formal disease investigations;
- Establishing culture facilities to maintain reef organisms for research.

The CDHC in cooperation with the research and management community has worked to 1) strengthen multidisciplinary collaborations and provide training for scientists and managers, 2) develop diagnostic capabilities (e.g., IRCP), 3) establish culture facilities to propagate model coral species for use in research and 4) develop educational materials, databases and web-based tools for scientists, managers and the public.

The CDHC recognized the need to improve collaboration among our U.S. Pacific and international colleagues. In June, 2006 experts from multiple disciplines were brought together in Honolulu, Hawaii to help chart a course for coral health and disease activities in the Pacific and Indo-Pacific. The intent of this meeting was to generate a strategic plan that addresses 1) research needs to help understand etiologies, epidemiology and ecology of Pacific coral diseases; 2) management needs in the context of identifying innovative strategies for disease management on coral reefs and 3) outreach and education needs to combat the spread of coral disease through novel strategies that engage the public and political sectors and enhance partnership with the CDHC. During the working group deliberations, participants identified five key areas for the CDHC to assist in organizing and coordinating scientific resources in:

- establishing diagnostic criteria and diagnostic tool development
- conducting mechanism-based research on coral health and disease
- leading outbreak investigations, training efforts, and epizootiological studies
- providing training and advanced continuing-education opportunities
- developing web-based communication and database tools

INTRODUCTION

Over the last 25 years there has been a worldwide increase in reports of disease affecting coral reef organisms, with the Caribbean basin emerging as a hot spot for diseases. The first documented Caribbean-wide epizootic was the mass mortality of the keystone herbivore, the long-spined black urchin (*Diadema antillarum)* (Lessios et al. 1984); this was followed by fungal infections that devastated seagrass (*Thalassia testudinum*) (Roblee 1991) populations in Florida Bay. Outbreaks of white band disease (WBD) were first reported from the US Virgin Islands in the late 1970s (Gladfelter et al. 1977). The disease spread throughout the region during the 1980s and over the next decade WBD contributed to the near elimination of *Acropora palmata* and *A. cervicornis* (Aronson and Precht 2001). The regional pattern of decline from these stressors is alarming, with coral cover decreasing from an average of 50% in 1977 to 5-10% in 2006 (Harvell et al. 2007; Miller et al. 2006; Waddell 2005).

In the Pacific Ocean, the threat of coral diseases has been thought to be relatively minor due to the spatial vastness of the region. Recent studies, however, indicate an escalating abundance and prevalence of disease throughout the Pacific (Harvell et al. 2007; Waddell 2005; Willis et al. 2004). While diseases affecting corals were first reported in the Indo-Pacific and Red Sea in the late 1970s (black band disease (BBD) and WBD), it is only in the last five years that coral disease survey efforts have increased throughout the region. These data are providing key findings that the number and distribution of diseases across the Indo-Pacific is on the rise. Scientists working in Australia, Philippines, Palau, Africa, American Samoa, the Red Sea and other locations have detected some of the more common and infectious diseases seen in the Caribbean, and have also discovered several diseases unique to each region. Recent data also suggest diseases may play a more important role in structuring Indo-Pacific reef communities than previously thought. These alarming trends emphasize the need for a comprehensive and collaborative research program to better understand biotic and abiotic diseases affecting Pacific coral reefs, and relationships between anthropogenic and environmental factors and their effects on coral health.

As evidenced by the loss of Caribbean acroporids and concurrent impacts on associated coral reef species, coral diseases have the potential to alter reef community structure and function. Several diseases are playing an increasingly important role in controlling coral population size, diversity and demographic characteristics. This decline in the health and living cover of reef building corals has created an urgent need for trans-disciplinary studies to understand mechanisms governing coral health and disease processes. However, much of the work on coral diseases to date has focused on the morphological characteristics of diseases and the search for specific pathogens that can be implicated in disease causation, without detailed investigation of the underlying cellular and structural characteristics. Furthermore, environmental and anthropogenic stressors (e.g., degraded water quality and climate change) have been cited as potential factors causing coral disease and mortality, yet few studies have adequately characterized causal links between disease and specific environmental stressors (Turgeon et al. 2002; Wilkinson 2002; Wilkinson 2004).

As an initial step to identify and address coral health and disease needs in the Pacific, the **CDHC Pacific Workshop: *Vision for Action*** was convened in Honolulu, Hawaii in June 2006. The goals of this meeting were to synthesize the state of knowledge of Pacific coral diseases and develop a strategic research plan that:

- identifies standardized methodologies for diagnosing coral disease in the Pacific
- identifies information gaps
- recommends strategic research for understanding coral disease etiologies
- addresses issues relevant to the management of coral reef resources and diseases
- fosters collaboration among CDHC partners and key stakeholders.

Workshop participants included recognized experts in biology, ecology, pathology, coral disease, molecular biology, cellular physiology, environmental microbiology, toxicology, veterinary medicine as well as the coral reef management community. These individuals were selected from academia, state and public health services, the biotechnology industry, U.S. government agencies and non-profit institutions, with representatives from Israel, the Indo-Pacific, western Pacific and Atlantic regions.

Several workshop participants were asked to develop position papers (Appendix VI). These included a variety of topics such as: 1) issues unique to Pacific coral reefs and coral health; 2) the current knowledge of coral disease in the Pacific; 3) lessons learned in the Caribbean that could be applied to Pacific efforts; and 4) key areas for the CDHC to assist in organizing and helping coordinate efforts in the Pacific. These papers were distributed to all participants prior to the workshop.

The authors of the position papers presented summaries of their reports during the plenary session on the first day. A number of complex biological and social issues unique to the Pacific were discussed including geographic expanse (i.e., a large number of islands and reefs), higher biodiversity, historical and cultural importance of reefs, the value of traditional knowledge, and considerations regarding traditional and local community ownership of reef resources. A brief background on the CDHC helped clarify the vision of the consortium, provided highlights of ongoing efforts in the Caribbean, and outlined possible opportunities for cross-disciplinary collaborations and training for scientists and managers in the Pacific. This was followed by a historical perspective of coral disease research in the wider Caribbean, including the current state of knowledge of Caribbean diseases, and what was missed during the emergence of diseases. Future considerations for Pacific efforts were also discussed, to help avoid some of the confusion that has arisen in the

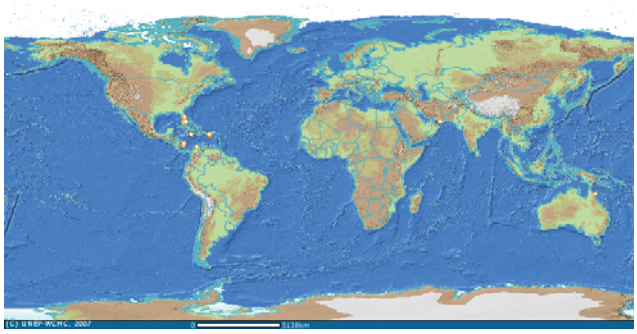

Figure I.1a Global distribution of Coral Diseases Reported in 1984.

Caribbean, and to better understand emerging infections and the drivers in disease outbreaks. This was followed by a series of presentations on the current knowledge of coral diseases in the Pacific, including the global distribution of diseases (Figs. I.1a and I.1b) and recent observations from Hawaii and NWHI, U.S. Territories and Freely Associated States, and the Western Pacific. The remainder of the session included discussions on diagnostic methods, disease outbreak investigations, and application of new tools and technologies for disease research, drawing heavily on approaches that have worked well in other wildlife veterinary and animal health programs.

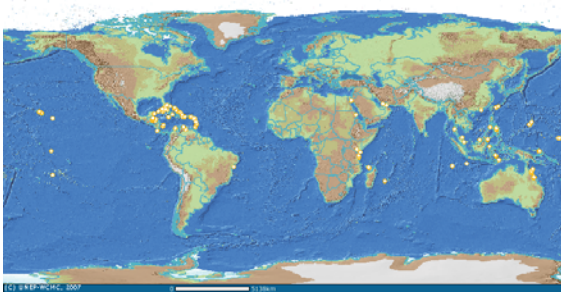

Figure I.1b Global Distribution of Coral Diseases Reported in 2004.

Four working groups were convened on the second day to 1) compile and synthesize available knowledge of coral disease for the Pacific and Indo-Pacific regions; 2) identify gaps in knowledge and technology; and 3) recommend strategic actions to improve our ability to respond to emerging infections in the Pacific:

- **Physiology & Pathology Working Group** (PPWG) was tasked with identifying what knowledge is needed regarding coral physiology to advance understanding and capabilities in coral pathology.
- **Toxicology & Ecological Epidemiology Working Group** (TEEWG) was tasked with identifying knowledge and needs related to toxicological impacts on coral, interactions with infectious agents and how to discriminate among them and the ecology of diseases.
- **Pathology of Infectious Disease Working Group** (PDWG) was tasked with identifying diagnostic approaches needed to move the coral disease field forward, including toxicology, microbiology, parasitology, pathology and diagnostic criteria to characterize the pathology associated with various infectious diseases.
- **Preventing and Responding to Coral Disease in the Pacific Region: Management Perspectives Working Group** (MWG) was tasked with evaluating management options that currently exist for diseases in corals and identifying existing tools and new approaches that can assist managers in the Pacific region in preventing and/or responding to coral disease.

Each of the working groups developed a series of goals, objectives and recommendations to address the identified tasks. These were presented to all participants in a plenary session at the end of the workshop, and are compiled in the following sections of these workshop proceedings. The participants also identified a number deliverables that will be produced as additional outputs of the workshop, including a series of resource manuals on 1) systematic approaches to disease investigations, 2) standardized laboratory methodologies, 3) diagnostic criteria and identification tools for Pacific diseases, and 4) culture facilities and husbandry approaches for a model species for coral research.

The four working groups identified numerous recommendations for CDHC activities related to coral disease research, monitoring, assessment, management, and communication to address coral disease and health in the Pacific. Specific objectives presented by each working group include:

- Identify tools and technologies, including available biomarkers, baseline assays, and targeted cellular research on coral physiology, to determine the health status of a coral and as a basis to understand coral pathology.
- Incorporate ecological epidemiology concepts into coral disease studies to identify predictors (ecological risk assessment model) for changes in coral health and ecosystem condition, quantify the strength of those associations, and focus diagnostic efforts toward identifying etiology.
- Develop diagnostic criteria and identification tools for Pacific diseases, including approaches to determine the suspected etiology of infectious disease in corals.
- Support the development of a Coral Disease Manager's Guide that includes standard nomenclature, survey protocols, outbreak response protocols, guidelines for research involving live organisms including biosecurity protocols, options for management responses, and resource materials available for managers.
- Compile a training manual on existing laboratory methods to investigate coral diseases.
- Develop a Manual for Selection and Husbandry of Model Species for Laboratory Investigations.
- Develop and implement coral disease monitoring programs for the Pacific, incorporating epizootiological surveys with assessment of relevant environmental parameters.

A. PHYSIOLOGY AND PATHOLOGY

Advancing Knowledge and Capabilities to Understand Coral Physiology and Pathology

Background

Signs abound in many areas that the natural plasticity of reef ecosystems to successfully respond to environmental (e.g., global climate change) and anthropogenic (e.g., land use, pollution) stressors has been exceeded. Vulnerable habitat conditions overlaid with multiple, concomitant stressors have compromised many coral communities to the point where their ability to rebound from natural disturbances is being lost. This is evidenced by the 27% loss of the world's coral reefs (Wilkinson 2002) and by predictions that estimate another 30% will be lost or impaired in less than 25 years. These predictions indicate that the current list of approximately 30-40 fatal diseases impacting corals will expand as will the frequency and extent of "coral bleaching" (Wilkinson 2002) resulting in effects ranging from reduced fitness, to community shifts, and ultimately to destruction of reef physical and biological functioning as we know it today.

Faced with many degrading environments over the planet, coral reefs are one of the key sentinels of ocean health and can serve as an indicator that links ocean and human health. Elevated disease levels among coral reefs serve as a sign of a sick ocean environment. With escalating disease reports throughout Pacific coral reefs, and predictions that point to a fate similar to that of Caribbean reefs, coral research and management are at a crossroads. This realization can instigate despair reflecting a hopeless inevitable fate that nothing can be done for reefs, or conversely be used as an instrument of change. It is our position that by developing an understanding of disease etiologies, causal links can be determined and factors driving these system failures can be identified. Developing such an understanding can move us from a triage mentality toward the ideal goal of *health management and preventative care* for coral reefs.

We are handicapped, however, in achieving preventative health care for coral reefs, in large part, because of a weak foundation in the basic sciences (e.g., biochemistry, cell biology, genetics, organismal and cellular physiology) of coral biology and the tools to enable rapid advancements. This has resulted in a fragmented research community, a menagerie of observations describing various coral afflictions with little coherence in how to make precise, defined observations in ways that promote comparative analysis, and almost no ability to discern mechanisms of disease.

The ability to successfully manage for healthy coral reefs depends on the inroads that are made into understanding the causes and effects of disease on coral vitality, i.e., coral pathology. Pathology however is rooted in the basic sciences of anatomy, physiology, microbiology, immunology, biochemistry and cell-molecular biology while integrating basic science with clinical applications. The very nature of pathology is predicated on the ability to discriminate between biological structures and functions occurring within a normal range and alterations resulting from disease processes. The depth at which we are

able to understand the normal structure and functions that govern corals at the colony level, individual, cellular and biochemical levels (i.e., their physiology) will dictate the speed and degree to which advancement is made in combating the spread of disease and ultimately proactively managing with the goal of healthy reefs.

Challenges and Recommendations

The Physiology & Pathology Working Group (PPWG) was tasked with identifying the information needed to advance knowledge and capabilities in coral physiology to better understand coral pathology. Thus providing a means to identify strategies to stop further reef degradation and create suitable conditions for natural restorative processes to take hold and flourish.

Coral biologists are challenged today with overcoming a void in information related to the functional processes of coral at cellular and organismal levels and the normal ranges in the functional parameters that define a healthy status, i.e., physiology. An adequate understanding of normal coral physiology and biochemistry is a prerequisite for building a sufficient foundation to competently study pathological conditions of corals. Understanding coral physiology and pathology requires defining the role of functional components at the cellular, systems, and organismal levels however the relationship between specific physiological sub-system (e.g., digestive, energy metabolism, nervous, reproductive, etc) processes, their regulation and the function of the whole animal has yet to be demonstrated for most coral species. This complicates discerning when or how normal biochemical/physiological processes have been disrupted to the extent that normal tolerance ranges of disease agents have been exceeded resulting in a pathogenic condition with lasting detrimental effects. Only when a full understanding of normal coral functions, as influenced by specific stressors, is achieved will the clinical manifestations of a specific disease be understood.

The pathogenesis and the etiology of a specific coral disease is partially known for only a few of the diseases described in the literature. Clinical manifestations have been described using a broad spectrum of biological/medical/veterinary terms that have been haphazardously applied to coral disease. The resulting nomenclature has painted a confusing picture that has led to misidentifying one syndrome for another. Another challenge for the coral disease community is to recognize the state of confusion within the field that can only be rectified by adopting standardized nomenclature and methodologies that will support exchange of information and ideas among coral disease investigators as well as with cross-disciplinary colleagues in fields not traditionally involved in coral disease research.

Pathogenesis: the pathologic, physiologic, or biochemical mechanism resulting in the development of a disease or morbid process

Etiology: the science and study of the causes of disease and their mode of operation

Clinical manifestations: gross morphological observations of corals

Compared to other wildlife diseases, coral disease research is in its infancy. Only recently have coral disease researchers begun applying technologies and methodologies routinely used in human and wildlife clinical and diagnostic medicine and pathology. Epidemiology is virtually nonexistent in the field. A growing number of scientists have begun applying biomedical approaches and adapting molecular biology tools in an effort to understand and characterize healthy corals and their responses when exposed to different stresses. These efforts show us the potential for understanding coral pathology and mechanisms for disease, and how a firmer grasp on this type of information can contribute to developing predictive indicators of adverse change in community health. However, there is a vast need to engage other researchers in the various aspects of coral health and disease. Persuading established researchers, in fields not traditionally part of marine science, to incorporate coral in their investigations or attracting new researchers to this field is difficult. The main challenges limiting progress in this arena include: 1) the availability of funding to conduct research on coral functional biology; 2) lack of a readily available source of research models; 3) few trained experts able to conduct the necessary research; and 4) lack of standardized field and laboratory approaches, including diagnostic criteria.

There is a critical need to equip scientists involved in coral research with the knowledge and skills to meet the challenges of health assessment and management. First and foremost, addressing information gaps on the functional biology of corals and their disease processes will require a broad integration of relevant disciplines that include health specialties (i.e., veterinary and medical science, pathology, medical microbiology, toxicology, epidemiology), marine scientists (i.e., wildlife and marine ecologists, marine biologists, oceanographers), basic scientists (i.e., biochemistry, cell physiology, microbiology, toxicology) and those who help interface with the public and politicians (i.e., resource managers, sociologists, economists). It is imperative to develop and provide advanced cross-disciplinary educational opportunities to encourage and equip the next generation of scientists to meet the challenges of coral reef health issues.

In addition to the strong support for strategic research in cellular physiology and funding routes pursued through directed funding by NOAA (i.e., grants program) and partnerships with NSF, EPA and other funding agencies, the PPWG recognized that a significant challenge to achieving success and a key underpinning is access to a valid research model (i.e., defined species, cell lines and zooxanthellae cultures). Agreement on selection of the model presents a significant challenge as many criteria need to be considered such as species range, growth forms, taxa with varying susceptibility to disease and bleaching, and known, reproducible genotypes. The PPWG also recognized the need to identify risk factors and preventative steps to reduce risks associated research activities (e.g., transport and introduction of pathogens via dive gear and tools, containment mechanisms to prevent the spread of coral disease under investigation in field and laboratory settings). Therefore, the PPWG devoted most of their time and effort in setting criteria for defining and selecting a model species for cellular physiological research which included identifying key information needs to support successful husbandry of the research animal model and identifying an initial set of candidate parameters to consider in delineating a normal physiological condition.

In the following section the PPWG identified six Strategic Objectives and associated Recommendations on practical approaches that can help address major gaps in the understanding of coral physiology and pathology. Achieving this goal will require instituting standard nomenclature to facilitate clear exchange of research and field observations, coral research models (species & cell lines) to elucidate physiological functions and morphological changes, and establishing standard culture conditions for consistency in use of model systems.

Strategic Objective A.1 **- Obtain strategic information needs in coral functional biology (e.g., cellular physiology, immunology, genetics, biochemistry)**.

Recommendation A.1.1: Provide targeted merit-based competitive grant opportunities to address knowledge gaps in the basic functional biology of corals through various Grants Programs offered by NOAA, NSF, EPA, NIEHS as well as private foundations.

There are limited sources of funding to conduct the research necessary to define physiological parameters and their natural variations in healthy coral. Most coral disease and health related funding has been targeted towards field monitoring that incorporates identification of gross lesions on coral to determine prevalence and incidence rates; microbiology to identify causative agents; histopathology to describe microscopic lesions; and a few biochemical and toxicological studies to measure responses of corals to various stressors, while few funding sources are specifically directed towards coral functional biology. The PPWG recommends establishing partnerships among granting agencies to develop a targeted RFP to support long term research and multi-investigator teams to determine baseline measures of coral health at the genetic, molecular, cellular, tissue and whole organismal level. This should include efforts to actively seek partnerships among the broader research community as a means of infusing new ideas and technologies from areas not traditionally considered as relevant to marine biology issues.

The PPWG identified five key information gaps that need to be addressed if the research and management community hopes to improve our understanding of coral pathology:

- Determine relationships between normal physiology and alterations caused by disease processes;
- Determine relationships between function at sub-system levels and functions at the whole organism and system levels;
- Elucidate how disruptions of normal physiological processes lead to pathologic processes;
- Determine the etiology, pathogenesis, and clinical manifestations of specific disease processes; and
- Predict clinical manifestations and appropriate treatment options for defined medical diagnoses.

By expanding research activities in areas of cellular physiology, genomics and proteomics, the research community will be better able to define nominal ranges of diagnostic parameters in healthy coral under normal spatial, temporal conditions and identify normal species-specific differences as a differential to recognize compromised health states. Through implementation of the recommendations put forward by this group, we can better characterize the complex mechanisms and factors underlying increases in bleaching events and coral disease outbreaks, as well as how human activities influence these processes. Understanding the mechanisms that confer resistance and susceptibility to disease, and deciphering the interactions between disease and environmental parameters will also provide the necessary information to support innovative development of diagnostic tools for rapid assessment of health and predictive capabilities of changes in health before disease signs manifest.

***Strategic Objective A.2* – Identify laboratory model(s) for coral research to enable rapid advances in our knowledge by focusing on fundamental biological concepts that are broadly applicable.**

Model species have been the key to rapid advances in disciplines such as developmental biology, genetics, toxicology, immunology, biochemistry and medicine. Model species have been developed in a number of taxa. Examples include *E. coli*, lambda phage, *Drosophila*, and *C. elegans* that have been instrumental in stimulating progress in our understanding of genetics and molecular biology. Selective breeding of species such as the brown rat and the common house mouse have produced white lab rats and mice that have been the workhorse of modern medicine. *Arabidopsis thaliana* (or Thale cress), *Medicago truncatula* (legume) and rice are three plant model species that have been essential for developing our understanding of the genetic and physiological bases responsible for fundamental biological functions that affect crop performance. Developmental genetics and cell biology have benefited enormously from studies of a non-mammalian vertebrate model, the zebrafish. Since its first recognition in the early 1970s, the zebrafish research community undertook several activities to promote uniform research conditions and open-exchange of information. Early on this included developing a manual for raising zebrafish for experiments and making it freely available and widely distributed among the research community. More recently, this free exchange of information has expanded to website resources and an enlarged zebrafish manual (see the following website for more information: http://zfin.org/zf_info/zfbook/zfbk.html), followed by the adoption of standard criterion for laboratory use of zebrafish. The website provides a large variety of resources in support of the zebrafish model, including products and supplies, gene collection, sequencing project, microarrays, funding opportunities, meeting information, and all types of document resources. The model is now listed on the NIH webpage for model organisms (Nih 2007) as one of eight non-mammalian models for biomedical research.

Our search for knowledge to date for hexacorals and octocorals has not been focused on a ‘model species’, but rather often represents the species readily available to a particular researcher. This has resulted in disparate studies involving hundreds of species or

subspecies, thus limiting the ability to compare data between studies and species. The PPWG recognized that all corals and their diseases are not the same, but an understanding of coral physiology requires focused development of one or two laboratory models that are most representative across scleractinian corals. There will always be a need to develop alternate models for specific diseases, but understanding basic coral physiology and the changes in these functions that result in disease will benefit from focused work on a few models.

Recommendation A.2.1 Establish criteria and select model species to focus basic coral physiological research.

Several suggestions for a cnidarian 'model species' have been published in the peer-reviewed literature, but only a few have recommended a scleractinian species. A brief summary of several recommended species and the disciplines for which they are most applicable are described in Appendix II and III. This literature review served to establish the currently available cnidarian models as well as to provide suggestions as to which criteria would be important in selecting a scleractinian model species for health and disease research.

Based on a review of the literature and the available expertise among working group members a list of criteria was developed for selecting a laboratory model for scleractinian coral physiology (see inset). The six possible candidates for the Indo-Pacific coral models identified by the PPWG are *Pocillopora damicornis, Stylophora pistillata, Porites rus, Galaxea fascicularis, Fungia scutaria, and Acropora formosa.* Each of these species has a different set of characteristics that make it a suitable candidate for a laboratory model for coral physiology studies. A brief rationale from published information is provided for each of these six species below.

Criteria for Selecting a Laboratory Model

1. Easy **adaptation** to long term captive rearing in closed, recirculation systems
2. Possible to provide many replicates through **fragmentation**
3. Widespread, geographical **distribution** in the Indo-Pacific
4. Reasonably **common**
5. Exhibits differences in **susceptibility** and **resistance** to disease
6. Representative of different **habitat** types (e.g., shallow water back reef and deeper water species)
7. Potential for **sexual reproduction** in captivity
8. Relatively **rapid** rates of **growth**
9. Branching and boulder growth **forms**

Pocillopora damicornis (aka, lace coral, cauliflower coral, bird's nest coral) is often referred to as the laboratory "white mouse" by coral biologists (Fig A.1). It is a major reef building coral widely distributed throughout the Indo-Pacific and Red Sea, and occurs in all shallow water habitats. It is affected by bleaching and disease worldwide, and has often served as an experimental subject for studies on coral physiology and reproduction. Its reproductive cycle is well described (Miller and Ayre 2004; Permata et

al. 2000; Richmond 1987; Sherman et al. 2006; Stoddart 1983; Ward 1992; Ward 1995; Whitaker 2006) and includes sexually produced planulae that are brooded to a fully developed Halcampoides-stage (Harrigan 1972). It is easily grown in a laboratory setting from fragments as well as larvae, and can be induced to produce planula year round by altering the night irradiance (Jokiel et al. 1985). The cryopreservation of P. damicornis larvae was reported by Hagedorn and colleagues (2006b). Its skeletal morphology, biochemical character and biomineralization process have been described (Brown et al. 1983; Domart-Coulon et al. 2004; Holden and Davis 2006; Letissier 1988; Tissier 1988; Vandermeulen 1975; Vandermeulen and Watabe 1973; Wainwright 1963). Conditions of stress and disease have been studied, including temperature extremes, bleaching, physical damage, sediment loading, ammonium enrichment, and infection by *Vibrio coralliilyticus* (Ben-Haim and Rosenberg 2002; Ben-Haim et al. 2003a; Ben-Haim et al. 2003b; D'croz and Mate 2004; Muller-Parker et al. 1994; Te 1992; Ward 1995). *P. damicornis* is susceptible to black-band disease (Dinsdale 2002; Willis et al. 2004), brown band disease (Willis et al. 2004), bacterial bleaching (Ben-Haim and Rosenberg 2002; Ben-Haim et al. 2003a; Ben-Haim et al. 2003b; Rosenberg and Ben-Haim 2002), mycelial fungal infections (Raghukumar and Raghukumar 1991) rapid tissue necrosis (RTN) (Luna et al. 2007), white syndrome (Willis et al. 2004) and skeletal eroding band disease (SEB) (Page and Willis 2008; Page et al. 2006; Willis et al. 2004). Primary cell cultures have been generated from *P. damicornis* and were demonstrated to produce aragonite crystals in adherent multicellular isolates (Domart-Coulon et al. 2001). Information is also available on growth characteristics following fragmentation, phylogenetic and symbiotic relationships, as well as histology and morphology. Recently the entire mitochondrial genome of *P. damicornis* was sequenced in a study designed to elucidate phylogenetically unique features of the family Pocilloporidae (Chen et al. 2008).

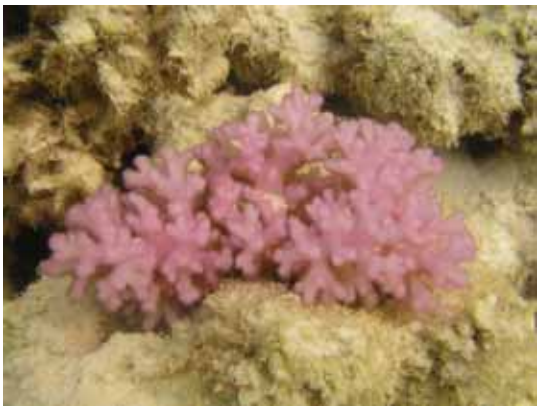

Figure A.1 ***Pocillopora damicornis*, photo by Greta Aeby.**

Stylophora pistillata (aka, false finger coral, cauliflower coral) is another well characterized scleractinian coral (Fig. A.2), whose widespread geographic distribution in the Indo-Pacific and commonality in shallow-water reef fringes make it an ideal candidate for a model species. It is a brooding species whose reproductive seasonality and lunar periodicity have been well described (Guest et al. 2005a; Guest et al. 2005b; Hall and Hughes 1996; Zakai et al. 2006). The larvae are easily induced to metamorphose in the laboratory (Baird and Morse 2004). *S. pistillata* has been used for many years as a key species for coral research in many fields, including coral biology, ecology, physiology, biochemistry, geochemistry, immunology, evolution, paleoecology, and biogeography (Loya, unpublished). Various features of the *S. pistillata* morphology

(Baird and Babcock 2000; Muscatine et al. 1997), physiology (Rinkevich and Loya 1986) and biochemistry (Dove et al. 2001; Dove et al. 1995; Richier et al. 2003; Rinkevich and Loya 1983; Tom et al. 1999; Zoccola et al. 2004; Zoccola et al. 1999) have been described, including characterization of the calcification process (Furla et al. 2000; Gattuso et al. 2000; Mass et al. 2007; Moya et al. 2006; Puverel et al. 2007; Puverel et al. 2005; Raz-Bahat et al. 2006) and dietary requirements (Houlbreque and Ferrier-Pages 2009; Houlbreque et al. 2003; Houlbrèque et al. 2004). The species has also been used in population level studies such as regional variations in population structure and dynamics, life history strategy, growth and regulation of populations, regeneration, competitive networks and reproductive strategy. Major contributions have been made on coral physiology, including insight into the symbiotic relationship between the coral host and its zooxanthellae, such as environmental effects on photosynthesis (Bhagooli and Hidaka 2003; Bhagooli and Hidaka 2004a; Ferrier-Pages et al. 2000), respiration (Hill and Ralph 2008; Leletkin 2005; Reynaud-Vaganay et al. 2001) and calcification mechanisms (Tambutte et al. 1996; Tambutte et al. 2007), energy budgets, carbon partitioning and utilization (Houlbrèque et al. 2004; Muscatine 1984; Reynaud et al. 2004), adaptive mechanisms of algal regulation and causes and effects of coral bleaching (Hoegh-Guldberg et al. 1987; Hueerkamp et al. 2001; Jones et al. 1999). The species has also been used to better understand obligatory, mutualistic or parasitic relationships and effects of marine pollution (crude oil, sewage and phosphates) at the population, individual and cellular levels (Loya et al. 2004; Rinkevich and Loya 1979; Walker and Ormond 1982). Recently the entire mitochondrial genome of *S. pistillata* was sequenced in a study designed to elucidate phylogenetically unique features of the family Pocilloporidae (Chen et al. 2008). *S. pistillata* is susceptible to Black-Band Disease (Dinsdale 2002; Willis et al. 2004), skeletal eroding band disease (SEB) (Page and Willis 2008; Willis et al. 2004) and Acroporid white syndrome (Roff et al. 2008; Willis et al. 2004).

Figure A.2 ***Stylophora pistillata*****, photo by Andy Bruckner.**

Porites rus (aka, plate and pillar coral) can form submassive, laminar branching, columnar structures, commonly over 5 meters across (Fig. A.3). It occurs throughout shallow reef environments in a wide variety of habitat types, where it may

Figure A.3 ***Porites rus*****, photo by Greta Aeby**

be the dominant coral. Kolinski and Cox (2003) reviewed the modes and timing of gamete and planula release for Hawaiian scleractinian corals; they listed, but failed to provide information on *P. rus.* This coral exhibits high survivorship when exposed to anthropogenic stressors such as pollution and elevated temperatures and is fairly resistant to bleaching (Yap 2004; Yap and Molina 2003). It can rapidly colonize areas after disturbance, exhibits relatively rapid growth rates and can be readily propagated from fragments (Dizon and Yap 2006a). The Tahitian *P. rus* was shown to produce four MAAs (mycosporine-like amino acids) (Teai et al. 1997). The sea-floor spectral reflectance (R) is a characteristic utilized in remote sensing; *P. rus* exhibits a spectral reflectance pattern consistent with "blue" corals (Hochberg et al. 2004). *Porites rus* from Guam was shown to harbor the "C" phylo-type of *Symbiodinium-like* (Rodriguez-Lanetty 2003). Although the response was not real strong, *P. rus* did show antimicrobial activity against cyanobacteria in a study conducted by Koh (1997). *P. rus* was reported in 2003 as one of the six branching species of coral in the Indo-Pacific that exhibited signs of PUWS (Porites ulcerative white spot disease) (Raymundo et al. 2005; Raymundo et al. 2003). In addition, Work and co-workers (white paper, this report page 189) reported discoloration as a result of a sponge infestation in *P. rus* in American Samoa.

Galaxea fascicularis (aka, tooth coral, moon coral, galaxy coral) is a hermatypic coral with a gonochoric (distinct sexes) breeding system (Fig. A.4). *G. fascicularis* has been grown successfully in culture with a >200% weight increase over a 37 month period (Carlson 1999); although coral extension rates and calcification rates in some aquarium systems are close to those reported for natural reefs, anomalies have been observed such as decreased skeletal density and unusual changes in colony morphology (Clode and Marshall 2003b). This species can be difficult to maintain in an aquaria due to high light requirements and a high susceptibility to infections (brown jelly). Various features of the *G. fascicularis* morphology, physiology and biochemistry have been described in the literature: characterization of the mucus (Fung and Ding 1998; Fung et al. 1997); characterization of a GFP-like protein (Karasawa et al. 2003); calcification processes (Al-Horani et al. 2007; Al-Horani 2005; Al-Horani et al. 2005a; Al-Horani et al. 2005b; Clode and Marshall 2002; Marshall and Clode 2004a; Marshall and Clode 2004b; Marshall et al. 2007); corallite morphology (Crabbe and Smith 2006); stress studies (Bhagooli and Hidaka 2003; Bhagooli and Hidaka 2004b; Philipp and Fabricius 2003); egg proteins (Hayakawa et al. 2006; Hayakawa et al. 2005); skeletal matrix (Clode and Marshall 2003a; Fukuda et al. 2003); various genomic studies (Fukuda et al. 2002; Watanabe et al. 2005); characterization of their algal symbionts (Huang et al. 2006; Watanabe et al. 2006); dietary requirements (Houlbrèque et al. 2004); and micosporine-like amino acid (MAA) abundance

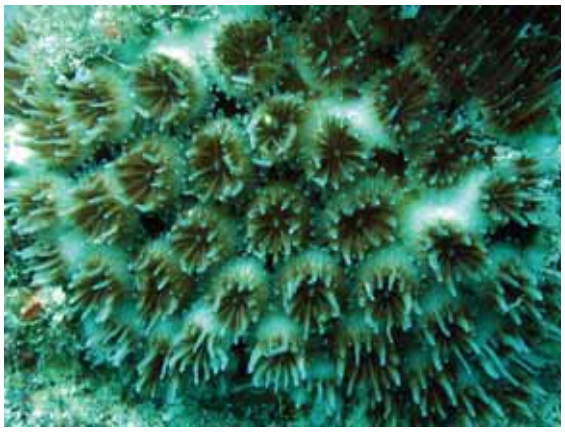

Figure A.4 *Galaxea fascicularis*, photo by Andy Bruckner.

(Yakovleva and Hidaka 2004). In an electrophoretic analysis, four soluble egg proteins were present in high abundance in the female egg, but were not found in the pseudo-eggs of functional males (Hayakawa et al. 2005). Gene expression, studied at the transcriptional level, was compared between female and functional male colonies. One of the vitellogenin-like proteins, GfEIP-4 protein, was cloned, sequenced and found to be expressed in both female functional eggs as well as male pseudo-eggs (Hayakawa et al. 2007). Although specific reports of disease in *G. fascicularis* have not been made, Winkler and colleagues (Winkler et al. 2004) reported SEB disease in coral reefs of Aqaba in the Red Sea including *Galaxea* sp. In addition, Work and co-workers (White paper, this report page 189) reported discoloration in *Galaxea* sp. in American Samoa.

Fungia scutaria (aka, mushroom coral) is the most common mushroom coral of the Indo-Pacific (Fig. A.5). It is free living and easy to collect. The species has separate sexes and releases eggs and sperm in the late afternoon, one or two days after a full moon. The larvae are azooxanthellate for 24 hours after fertilization and the process for establishing symbiosis can be observed without confounding background (Wood-Charlson et al. 2006). Krupp (1983) reported spawning to occur between 1700 and 1900 hours, 1-4 days following the full moon with only one short spawning event per lunar cycle. Krupp also reported that the oral pit formed by 24h and that the mouth became clearly visible by 39h; ingestion of zooxanthellae was not observed, but in a few days the planulae possessed zooxanthellae. *F. scutaria* also reproduce asexually (Krupp et al. 1993) and can regenerate from polyp stalks or from septal fragments (Krupp et al. 1996a). Their host-algal interactions have been studied, including means of infection and localization in tissues (Rodriguez-Lanetty et al. 2004; Rodriguez-Lanetty et al. 2006; Schwarz et al. 1999; Weis et al. 2001). Their sperm and planulae have been cryopreserved (Hagedorn et al. 2006a; Hagedorn et al. 2006b). The toxicity of the pesticide chlorpyrifos was tested against *F. scutaria's* gametes and planulae (Krupp et al. 1996b). From the examination of mucus samples for C,N,P composition (Krupp 1982) and their immunochemical nature (Krupp 1985), it was deduced that one of the components is sulfated acid polysaccharide and that the mucus was predominantly carbohydrate composition with some protein and of low nutritional quality. Although specific reports of disease in *F. scutaria* have not been made, Winkler and colleagues (Winkler et al. 2004) reported SEB disease in coral reefs of Aqaba in the Red Sea including *Fungia* sp.

Figure A.5 *Fungia scutaria*, photo by Thierry Work.

Acropora formosa (Dana 1846) (aka, staghorn coral) is a common branching coral that forms large thickets in shallow water on reef slopes, fringes, and lagoons (Fig. A.6); the species often coexists with other acroporids such as *A. nobilis* and *A. grandis* and is widely distributed throughout the Red Sea, the Pacific and Indian Oceans. This species reproduces sexually and can also be easily propagated from fragments (Okubo et al. 2005) but successful sexual reproduction following fragmentation is dependent upon the fragment size and the stage of oocyte development during fragmentation (Okubo et al. 2007). Staghorn-type *Acropora* sp. grows rapidly making it an ideal candidate for captive breeding/propagation. *A. formosa* calcification involves active Ca^{++} transport (Chalker 1976) and is impacted by ambient seawater temperature (Crossland 1984) with temperature exceeding light in determining growth and survival on a high-latitude reef. The development of scleractinian larvae from gamete-spawning coral, including *A. formosa*, was described in detail by Babcock and Heyward (1986) and in great detail for 5 related acroporid species (Okubo and Motokawa 2007). There are numerous papers describing the zooxanthellae associated with *A. formosa*: characterizing their DNA (Huang et al. 2006); describing their phosphate uptake (Jackson and Yellowlees 1990) and metabolism (Jackson et al. 1989); examining their potential for ammonia metabolism (Dudler and Miller 1988); and describing their turnover during bleaching (Jones and Yellowlees 1997). Toxic reactions to external chemicals both natural and anthropogenic have been reported: defensive chemicals exuded by soft corals can cause expulsion of *A. formosa*'s algae, followed by release of nematocysts and eventual tissue necrosis and death (Aceret et al. 1995) while herbicides have been shown to decrease the effectiveness of dinoflagellate photosynthesis by inhibiting the Photosystem II system (Jones and Kerswell 2003; Jones et al. 2003). *A. formosa* is susceptible to disease including Skeletal Eroding Band Disease (Page and Willis 2008; Riegl and Antonius 2003; Willis et al. 2004), brown band disease (Willis et al. 2004; Yarden et al. 2007), white syndrome (Willis et al. 2004), black-band disease (Willis et al. 2004) and skeletal anomalies (Peters et al. 1986; Sutherland et al. 2004; Work et al. 2008a). Photoprotection of *A. formosa* may be provided by both mycosporine-like amino acids, UV-absorbing compounds (Dunlap and Chalker 1986; Dunlap and Shick 1998) and by photopigments, one being a unique blue pigment in *A. formosa,* produced by the coral host (Dove et al. 2001; Dove et al. 1995). Another protective barrier, mucus, has been described in detail for *A. formosa*; the composition was reported to be polymers of proteoglycan crosslinked

Figure A.6 ***Acropora formosa*****, photo by Greta Aeby.**

by disulfides (Richards et al. 1983). Further characterization of the mucus side-chains indicated that they were sulfated oligosaccharides terminating with a mannitol (Meikle et al. 1987). In a comparative study, Meikle and colleagues found that there is not a common mucus structure for 6 different species of coral, rather the mucus was either dominated by unique protein or carbohydrate polymers (Meikle et al. 1988). Although complete genome information is not available for *A. formosa,* numerous studies have reported both genomic and mitochondrial DNA sequences.

Acropora millepora (a staghorn-type coral, no common name) is a hermatypic scleractinian coral with a digitate to branching growth form (Fig. A.7). It has a broad geographic distribution in the Indo-Pacific, found mainly in shallow water on reef slopes, fringes, and lagoons. *A. millepora* is a broadcast spawner that release eggs and sperm bundles once a year. Their embryology has been described in detail and the molecular cues have been investigated in parallel (Ball et al. 2002). They acquire their zooxanthellae during the planula larval stage (Van Oppen 2001). Ralph and co-workers demonstrated strong down-regulation of photosynthesis under conditions of high irradiance and observed little photoinhibitory damage to photosystem II (Ralph et al. 2002). High sea-surface temperatures lead to thermal stress in *A. millepora* with rapid bleaching, followed by recovery (with high retention of reproductive capacity) or death within 14 weeks; this in contrast to *Platygyra* sp. that resisted bleaching longer and took longer to recover (with loss of reproductive capacity) or die (Baird and Marshall 2002). Recently it was shown that there are shifts in the composition of the 'coral-associated bacterial assemblages' sampled from colonies of *A. millepora* during a natural bleaching event (Bourne et al. 2008a). The herbicide diuron did not inhibit fertilization in *A. millepora* but it did significantly impact metamorphosis in symbiont free larvae of this species (Negri et al. 2005). Other toxics, tributyltin, copper (Negri and Heyward 2001) and petroleum products (Negri and Heyward 2000), all inhibit *A. millepora* fertilization and metamorphosis to some extent. *A. millepora* is susceptible to black band disease (BBD) (Dinsdale 2002; Willis et al. 2004), skeletal eroding band disease SEB (Page and Willis 2008; Willis et al. 2004) and potentially skeletal growth anomalies (Work et al. 2008a). The innate immune capacity of *A. millepora* was characterized by scanning available EST and genomic resources; neither the Toll/TLR or the complement pathways were identified, but the presence of complement C3 and several MAC/PF are encouraging (Miller et al. 2007). Recently, the isolation and characterization of a mannose-binding lectin was reported; this pattern recognition protein binds bacterial pathogens as well as the coral symbiont

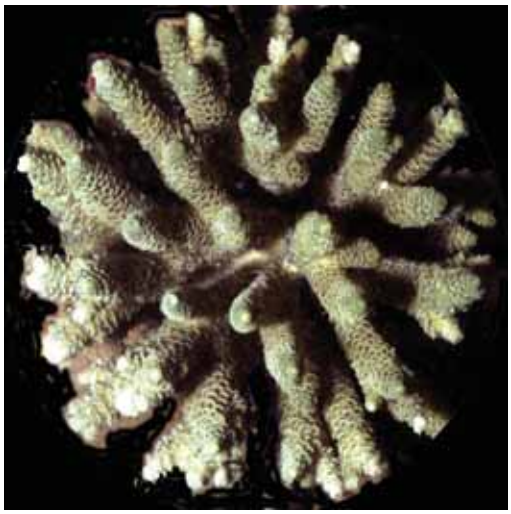

Figure A.7 ***Acropora millepora*****, photo by Andy Bruckner.**

Symbiodinium (Kvennefors et al. 2008). There are over 10,000 EST sequences listed in Genbank for *A. millepora* (Ball et al. 2002; Miller et al. 2007; Technau et al. 2005).

The working group submitted two candidate models for the Indo-Pacific region during the plenary discussion, ***P. damicornis*** and ***S. pistillata***. After additional discussion in the plenary session, the following two species were adopted as the recommendation of the Workshop, ***P. damicornis*** and ***A. millepora***

Strategic Objective A.3: Establish culture facilities to propagate corals, coral tissue and zooxanthellae cell cultures from the model species to augment laboratory studies.

Recommendation A.3.1: Determine the requirements and methodologies for establishing *in vitro* tissue culture and cell lines.

Coral tissue culture or zooxanthellae cell lines are not commercially available to researchers currently. The special requirements needed to establish and maintain coral cell cultures have not been fully elucidated and published such that this procedure is widely available and those reported successes of coral cell culture report only short term viability as primary cultures. There are however, private collections of zooxanthellae cell cultures, but only a handful of researchers are able to grow and maintain these cultures. The PPWG recommended the CDHC contact these investigators to assist in the development of a manual detailing the special requirements for establishing these cultures, and identify possible facilities that could maintain living cultures and provide them to researchers for a nominal cost, such as has been done in the harmful algal bloom (HAB's) community.

Several studies have undertaken the challenge to develop tissue/cell cultures of coral species. The importance of the presence of the extracellular matrix (ECM) was demonstrated by Schmid and colleagues (Schmid et al. 1999) as well as others (Dizon and Yap 2006a; Dizon and Yap 2006b; Lewis et al. 2006; Okubo et al. 2005; Permata and Hidaka 2005; Raymundo and Maypa 2004; Yap 2004). They noted that "when cultured, most cnidarian cells survive only when attached to ECM substrates; they rarely divide and die within short times." A review of the obstacles, approaches and improvements in culturing was published in 1999 (Schmid et al.) and updated in 2005 (Rinkevich). Moderate success was achieved in developing short-term primary cultures by several groups. Kopecky and Ostrander (1999) successfully cultured multicellular endothelial isolates from branching scleractinian coral (*Acropora micropthalma* and *P. damico*rnis) that survived in primary culture for 300h. In addition, five other species were successfully cultured: *Montipora digitata*, *S. pistillata*, and *Seriatopora hystri*x with *Porites sp.* less successfully. In a study conducted by Domart-Coulon et al. (2001) cells of apical coral colony fragments (*P. damicornis*) were isolated by spontaneous *in vitro* dissociation. Single dissociated cell types were separated by density in a discontinuous Percoll gradient. Primary cell cultures displayed a transient increase in alkaline phosphatase (ALP) activity, to the level observed in intact corals. Unique to this study was the demonstration of aragonite 'precipitation'. Continuous cell cultures of four

species of Octocorallia were reported by Frank et al. (2001) as well as a method to produce primary cell cultures for 10 cnidarian species including three Hexacorallia (*S. pistillata, Porites lutea, F. favus*). The primary cell cultures underwent cell proliferation within 2-3 weeks, and produced a collagenase soluble gelatinous matrix on the bottom of the wells. In another study focused on whole tissue isolation, soft tissue detachment from the skeleton of two branching coral species (*S. pistillata* and *P. damicornis*) yielded viable tissue capable of 70% survival for 3 days (Frank et al. 1994); these tissue pieces (containing whole polyps) quickly lost their morphology in dilute cell culture media, i.e., radial symmetry and oral-aboral polarity both were lost. After two days, in high glucose media, the tissue isolates dissociated layer by layer into individual cells, spreading in a circular outgrowth.

Recommendation A.3.2: Create the infrastructure and community-base to make experimental animals accessible and explore mechanisms to provide coral fragments from model species and in vitro tissue culture or cell lines to research community.

There are several groups interested in providing coral fragments or nubbins for coral research. The University of Miami's hatchery facility has been conducting feasibility studies with the goal of expanding their current culture capabilities (i.e., providing aplysia for neurological research) to coral production. However the major impediments, to establishing living stock collections of coral specimens of well defined genetic lines, are lack of funding and permit issues.

The PPWG identified one possible funding source to develop a coral culture facility. 'Living Stock Collections' is a potential source of short-term (36 months) support with the goal of developing innovative handling of stocks or well designed improvements in handling stocks. This is funded by the U.S. National Science Foundation (http://www.nsf.gov/pubs/2006/nsf06574/nsf06574.htm). There are only about 2-4 awards per year and the anticipated funding is limited with $1,000,000 available for all awards (new and renewed). Limitations of this program are: limited support for storage and distribution; no funds to conduct research beyond normal and appropriate curatorial efforts; and, no direct support for development of new reagents. Due to the short-term nature and limited support of this source of funding it is apparent that other resources must be explored.

To implement this recommendation, it will be necessary to obtain sustained government funding and/or assemble a team to actively identify benefactors from the private sector and secure long term funding until such a facility(ies) can be self-sustaining.

Recommendation A.3.3: Identify culture parameters that support normal physiologic condition and normal growth of the coral host model species.

The PPWG recommended the development of informational resources on coral husbandry, with emphasis on selected model species. These resources would describe optimal environmental parameters for culture facilities (e.g., light levels, water quality, food sources); identify potential stressors that may affect the system; develop protocols and methodologies for propagating corals through asexual and sexual reproduction with enhanced growth rates; and develop protocols for treating known diseases that affect aquarium invertebrates:

- Define tolerance ranges and optimum culture conditions (temperature, salinity, light intensity-wavelength, saltwater composition, density). Some information is presented in (Delbeek and Sprung 1994; Sprung and Delbeek 1997).
- Describe culture induced conditions, such as excess worms, macroalgae, parasites, and diseases, and provide protocols for treatment. See Borneman (2001) for more information.
- Define conditions required for sexual reproduction; provide protocols. See (Delbeek and Sprung 1994; Hagedorn et al. 2006a; Hagedorn et al. 2006b; Sprung and Delbeek 1997).
- Describe methods to promote optimum growth following fragmentation of coral. See (Ayre et al. 1997; Dizon and Yap 2006a; Okubo et al. 2005; Tarrant et al. 2004; Tsounis et al. 2006).

To implement this recommendation for developing these information products, the CDHC will need to identify experts in this field and assemble a team to draft the informational resources described above.

Recommendation A.3.4: Provide support to researchers in the optimal care and handling of the organisms by providing standard protocols for culturing the experimental animals through development of a manual for coral model laboratory organism.

The PPWG suggested that this manual could be patterned after the Zebrafish Manual. Once developed it should be published in hard copy and made available on the NOAA CDHC and CoRIS webpages. The group suggested the manual should include the following sections:

a. Introduction
b. Background [information on basic biology and biochemistry, nutritional requirements, physiological systems, reproductive characteristics, symbiotic relationships (algal and bacterial), susceptibility to stressors and genetics]
c. Distribution
d. Morphology
e. Developmental Biology
f. Molecular Biology (EST libraries, known gene sequences, phylogenetic relationships)
g. Husbandry

h. Availability and Processes for obtaining organism
i. Shipping instructions
j. Biosecurity
k. Permits, etc.
l. Histology: protocols and photographs
m. Standardized procedures for research studies
n. Research tools available (primers, specific antibodies, histological stains, etc.)

Creation of this manual is a team effort and will require experts to be identified who are willing to collaborate on the development of this manual. The CDHC could facilitate the team interactions by providing meeting logistics and publishing the manual after final peer-review.

Strategic Objective A.4: Determine key physiological parameters that typify a normal or healthy condition for the model coral species.

Recommendation A.4.1: Define a suite of physiological parameters that represents gross, cellular and subcellular levels of biological function and establish normal ranges for each parameter as criteria for determining health status of the model species.

The PPWG considered a number of possible assays that could be used to assess health status that include assays to assess photosynthetic potential, biochemical and cellular responses of coral to various stressors, regeneration of tissue, growth and reproduction. The group recognized that many of the specialized needs (e.g., immune function, cellular diagnostics, cell type and functional probes), would require a focused effort to develop the necessary assays. One approach to begin defining the physiological parameters suggested was to summarize methodologies and assays currently available as a resource manual for the model species. The initial list of factors to consider is as follows:

1. PAM fluorometer measurements, range and conditions, and standardized parameters for making the measurement;
2. Calcification rates under standard growing conditions;
3. Histology and morphology descriptions, and protocols for preparation of samples for light microscopy, SEM, TEM;
4. Microbial communities (culture-independent vs culture-dependent methods) under standard growing conditions.
5. Zooxanthellae symbionts characterized with respect to number, mitotic index, chlorophyll levels, and genotype;
6. Lesion development and regeneration;
7. Response to nutrient levels;
8. Developmental biology; and
9. Identification of cellular parameters useful in diagnosing environmental stressors and disease

The group recognized that much of the information proposed for inclusion in the resource described above may not be available, depending on the model species of interest. They identified seven key parameters that should be further explored in detail:

- Determine the specific ratio of protein-carbohydrate-fat in diet to support maximal growth.
- Determine nutritional requirements of the host, specifically any essential compounds, elements, vitamins, or trace elements required for successful metabolic homeostasis (i.e., nutrients provided by symbionts, mutualistic bacteria, etc that cannot be synthesized by the coral host) (Grover et al. 2002; Houlbreque and Ferrier-Pages 2008; Mills and Sebens 2004; Muscatine 1973; Muscatine and Hand 1958).
- Define the basics of the calcification process and required conditions for maximal growth. (Abramovitch-Gottlib et al. 2002; Al-Horani et al. 2005a; Elahi and Edmunds 2007; Fine and Tchernov 2007; Gattuso et al. 2000).
- Define the requirements to support normal reproduction and describe the developmental biology (Abramovitch-Gottlib et al. 2002; Lewis et al. 2006).
- Define specific metabolite levels or enzymatic activities describing critical cellular and tissue function and characteristics of microbial communities (TRFLP versus culture) under standard growing conditions. (Achermann 1980; Ball et al. 2002; De Jong et al. 2006; Gajewski et al. 1996; Holland 2004; Kopecky and Ostrander 1999; Miller and Harrison 1990; Seipel and Schmid 2006; Torras and Gonzalez-Crespo 2005; Watson and Mire 1999).

Recommendation A.4.2: Define markers of disease, both from a structural (e.g., histology) and functional (e.g., clinical diagnostic assays) perspective, that establish criteria for determining abnormal health condition in the coral host model species.

The PPWG identified four key research needs that could fill gaps in our understanding of changes in coral health, including morphological characterization using histology and electron microscopy; identification of cellular diagnostic parameters; characterization of patterns of lesion regeneration; and responses of the coral and associated symbionts to human and natural stressors:

- Reference materials (e.g., an atlas of coral tissue samples) with histological and morphological descriptions and photographs of healthy, stressed and diseased conditions in representative Pacific coral species. The Atlas should include protocols for preparation of samples for light microscopy, SEM, and TEM. (Ainsworth et al. 2006; Bourne et al. 2008a; Bourne et al. 2008b; Breitbart et al. 2005; Bythell et al. 2002; Gil-Agudelo et al. 2006; Klaus et al. 2007; Rosenberg et al. 2007; Work and Aeby 2006; Work et al. 2008a; Yokouchi et al. 2006).
- Characterize and describe cellular biomarkers for disease and develop a manual on assay and/or test protocols. (Downs and Downs 2007; Downs et al. 2005a; Downs et al. 2005b; Mc Clanahan et al. 2004; Peters 1984a; Peters 1984b; Peters 2001; Work and Aeby 2006; Work and Rameyer 2005).

- Understanding of conditions that support regeneration following lesion development (Hall 1997; Hall 2001; Henry and Hart 2005; Kramarsky-Winter 2004; Kramarsky-Winter and Loya 2000; Titlyanov et al. 2007).
- Characteristics of the response of the holobiont (coral host, zooxanthellae symbionts, and microbial community) to stressors (Branton et al. 1999; Hashimoto 2005; Lejeusne et al. 2006; Mc Dougall et al. 2006; Mitchelmore et al. 2007; Readman et al. 1996; Rougee et al. 2006).

Strategic Objective A.5: Create and support advanced educational opportunities.

Recommendation A.5.1: Develop an advanced degree program in coral pathology, offer continuing education in specialty topics for professionals and support fellowships for career development or cross-specialty training.

There is a critical need to build scientific capacity in the field of coral pathology and offer a health management perspective in resource management (Mullen et al. 2004; Sutherland et al. 2004; Woodley et al. 2008; Woodley et al. 2007; Work and Rameyer 2005). This will require a broad integration of relevant disciplines that assimilate expertise, tools and information from the coral research community as well as human, veterinary and wildlife scientists (e.g., pathologists, microbiologists, ecologists, cell physiologists). It is imperative to develop and provide advanced cross-disciplinary educational opportunities to encourage and equip the next generation of scientists to meet the challenges of coral reef health issues. This could include continuing education courses for professional development in histology/histopathology, environmental forensics, ecotoxicology, risk assessment and other disciplines. It should also include opportunities for advanced education such as a Master's program in coral pathology and graduate courses in cnidarian cell biology, histology and physiology.

Strategic Objective A.6: Organize a system of methodologies to investigate coral disease.

Recommendation A.6.1: Provide conceptual approaches to support sound science as coral biology merges with the field of medicine to understand disease causes and mechanisms that include guidance for the proper handling and containment regimes for laboratory and field experiments.

The PPWG discussed the concerns associated with the potential transfer of pathogenic organisms between locations and the lack of national or international guidelines for cleaning and disinfecting methods for vessels, equipment and divers that can prevent the transmission and/or introduction of pathogens to new hosts or locations. The potential for transmission may be elevated by researchers in direct contact with diseased corals or on reefs with disease outbreaks especially through 1) transfection experiments involving the removal of diseased tissue and transplantation to other presumably healthy hosts; 2) transfer of dive gear and tools that have not been decontaminated from a reef with a

disease outbreak to a neighboring reefs, or even to other locations within the same or different oceans; and 3) research to identify a causative agent via infection experiments conducted *in situ*. Other concerns include potential human health issues arising from the handling of infectious agents either in the field or laboratory.

A variety of groups have begun to develop protocols to protect against the introduction of pathogens or spread of disease including health certification for corals raised in laboratory settings and subsequently transplanted onto coral reefs, as well as protocols for cleaning and disinfecting vessels, dive gear and equipment prior to the transport between locations. Medical and veterinary containment measures may also be easily applied to potentially infectious disease outbreaks in the aquatic environment. It is recommended that a working group consider the available information and propose SOPs; an external review by a recognized authority of the SOPs is suggested to validate the process.

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B. TOXICOLOGY & ECOLOGICAL EPIDEMIOLOGY

Identifying the Current State of Knowledge and Knowledge-gaps for Toxicological and Infectious Impacts on Coral using Ecological Epidemiology

Background

The deterioration of many coral reef ecosystems worldwide is a clear example of not only the effects global environmental damage can have on our oceans' health, but also damage from local sources of pollution. This damage is multi-factorial as are its consequences. Since the 1970's, mounting evidence has built a convincing argument that human activities are a prominent cause (e.g., coastal urban and industrial development, agricultural runoff, sedimentation, over-harvesting, marine pollution, disease and climate change) (Bellwood et al. 2004; Bryant et al. 1998; Risk 1999; Turgeon et al. 2002; Walker and Ormond 1982). Anthropogenic factors (i.e., physical, chemical and biological) can be exacerbated by natural factors (e.g., *climate:* water temperature, UV, weather pattern changes, volcanic/tectonic activity; *biological:* nutrient cycling, bioerosion, infectious disease) resulting in adverse health effects collectively recognized as disease (Wobeser 1981).

Reef species experiencing persistent environmental disturbances (e.g., coastal development and land-based pollution) may respond with acute mortality, resulting in rapid loss of diversity and abundance; but may also display non-acute, sub-lethal effects. These effects often present as increased incidence of disease (i.e., gross lesions), reduced growth, diminished reproductive effort and recruitment, and ultimately reef systems can cascade into irreversible deterioration (CRMP 2001; Downs et al. 2005c; Hoegh-Guldberg 1999; Knowlton 2001; Nystrom et al. 2000; Patterson et al. 2002; Porter and Tougas 2001; Richmond 1993). On a global basis, attempts to arrest overall coral reef decline have failed with reef degradation continuing (Bellwood et al. 2004; Jameson et al. 2002; Wilkinson 2002).

Why are we failing to stop the declines? How can we change this?

An examination of coral reef health assessments conducted over the last 30 years show detailed descriptions at the population and community levels in terms of coral cover, diversity and population dynamics of other reef species (usually fish abundance and diversity) but with little change in methodology (Downs et al. 2005c). Though necessary, these well-defined descriptions are not sufficient to answer *why* or *what* to do about the continuing decline of reef condition. Similarly, contaminant chemistry programs that detail the array of chemicals found at a site cannot answer whether these contaminants are benign or causal in disrupting coral health. A better understanding of the root cause of reef decline is necessary if mitigation decisions are to be successful. This requires integrating descriptive data with efforts to elucidate mechanisms of action and causal analyses to determine if there is an association between a biological response and a putative stressor, the nature of that association (e.g., impairment) (Boehm et al. 1995a;

Boehm et al. 1995b; Downs et al. 2005c; EPA 2000; Suter 2006) and in turn determine the associated ecological risk for better informed management options.

By its very nature, toxicology is an integrative science that is designed to uncover fundamental mechanisms of action governing chemical effects on biological systems. Drawing from the basic disciplines of molecular biology, biochemistry and physiology, toxicological principles and methods can be applied to subcellular systems and extended to ecosystems by evaluating ecological effects of chemicals or ecotoxicology (Hahn and Stegeman 1999; Suter 1993). With only a few studies recently published, toxicology and its relationship to infectious disease is only beginning to be applied to coral. It is however a critical underpinning for developing sound evidence that provides causal links between stressors and their biological effect(s) on corals and reef systems.

By merging toxicology, causal analysis and risk assessment information with measures of health condition (e.g., pathology, and health assessment), epidemiological methods can be used to understand disease incidence, distribution and causes while identifying and characterizing risk factors (predictors) that drive its occurrence, regardless of the root causation (biotic or abiotic). While classical epidemiology explores the statistical relationships between disease agents (both infectious and non-infectious), a related field, ecological epidemiology views disease as a result of the ecological interactions among populations of hosts and parasites (pathogens) and is concerned with the identification of critical parameters (e.g. the incubation period or latency) as well as the chemical and physical nature of the environment and how each contributes to the health of the organisms within the particular ecosystem (Cormier 2006; Suter 2006). Since most disease is multi-factorial, identification of risk factors for coral health can direct and prioritize management strategies toward risk reduction without requiring knowledge of specific etiologies.

Challenges and Recommendations:
The ultimate challenge is to move from a triage approach to coral reef decline to a state of knowledge where causal links can be determined and factors driving these system failures can be identified. This can then support ecological risk assessments that lead to the formulation of risk reduction strategies and mitigation actions. Developing this understanding can move us toward the ideal **goal of *health management and preventative care* for coral reefs.** To achieve a position of coral health management, however, will require recognizing that we currently lack the understanding and the ability to mitigate the problem and current approaches to environmental assessments for corals are not effective. The necessity for a change in the paradigm and approaches that currently dictate how the welfare of coral reefs is assessed must also be recognized. This requires a new approach to the science, new assessments and methodologies and a different focus of effort.

To effectively protect coral reef resources, resource managers need sound information that can clearly 1) characterize baseline health of coral reef communities, 2) demonstrate resource injury and determine its extent, 3) forensically link causal factors to the injured resource, and 4) routinely and consistently evaluate effectiveness of the management

response and thus, enhance resource protection (Boehm et al. 1995a; Boehm et al. 1995b). A mechanistic understanding of modes of action, susceptibility differences among species, interaction between chemicals and environmental variables (e.g., temperature, salinity, light, pressure), and tools that allow monitoring for exposures and effects will enable causal and risk analyses to be used for coral reef assessments (Hahn and Stegeman 1999). Obviously not all human activities that cause environmental damage can be eliminated, however by adopting an environmental risk assessment strategy, decision-making can be improved to better protect coral reef resources by characterizing risks and quantifying them. Thus risk assessments enable prioritizing actions and provide quantitative measures for evaluating management actions and their consequences. While risk assessment is a process that assigns probabilities to adverse effects of human activities or natural damaging events, it does not address health assessment which is concerned with determining the occurrence and causes of impairments of non-human populations and communities, a field known as ecological epidemiology (Cormier 2006; Suter 2006). Thus integrating ecological epidemiology (biological assessment and causal analyses) with risk assessment (risk models that link alternative decisions to future conditions) provides a systematic means to improve understanding of the causal chain of events and the factors involved for informed management decisions (Suter 2006).

The Toxicology and Ecological Epidemiology working group (TEEWG) recognized the need to be able to *detect* change in coral health at the ecosystem, community and individual level *before* the system is damaged. Detecting change however requires establishing a baseline of health and disease indicators using standardized and accepted methodologies. The Group also emphasized that in order to determine the significance of the impacts that toxicants or pathogens have on coral ecosystems there is a greater need to track *biological responses* (i.e., health changes) than to measure the presence/absence of toxicants. The ability to discern biological consequences (direct and isolated effects) of toxicants will rely on the availability of laboratory studies. The integration of this process would call for adopting an epidemiological approach and then integrating it with ecological risk assessments for improving coral health and disease management options.

As a result of their deliberations, TEEWG recommended a systematic approach to begin the process (Fig. B.1). The first step is to adopt specific health indicators in field research and monitoring efforts to be able to detect change (i.e., condition assessment)(Cormier and Suter 2008) in coral health at the ecosystem, community, and individual organism levels; 2) conduct surveillance to determine baselines for health indicators and detect change resulting in impairment; 3) identify probable causes for impairment and (i.e., causal pathway analysis); 4) identify and assess risk factors as predictors of health effects (i.e., ecological risk assessment); 5) implement risk management decisions (i.e., management assessment); and 6) conduct outcome assessments to evaluate the success of the management decisions. The output of the Group provides a framework to move forward and a start at populating this framework with a) a list of predictors and outcomes; b) identification of data gaps and resources; c) a list of recommendations to enhance field monitoring efforts; d) a draft list of data variables and a standardized formaat for recording information; and e) specific recommendations to move forward.

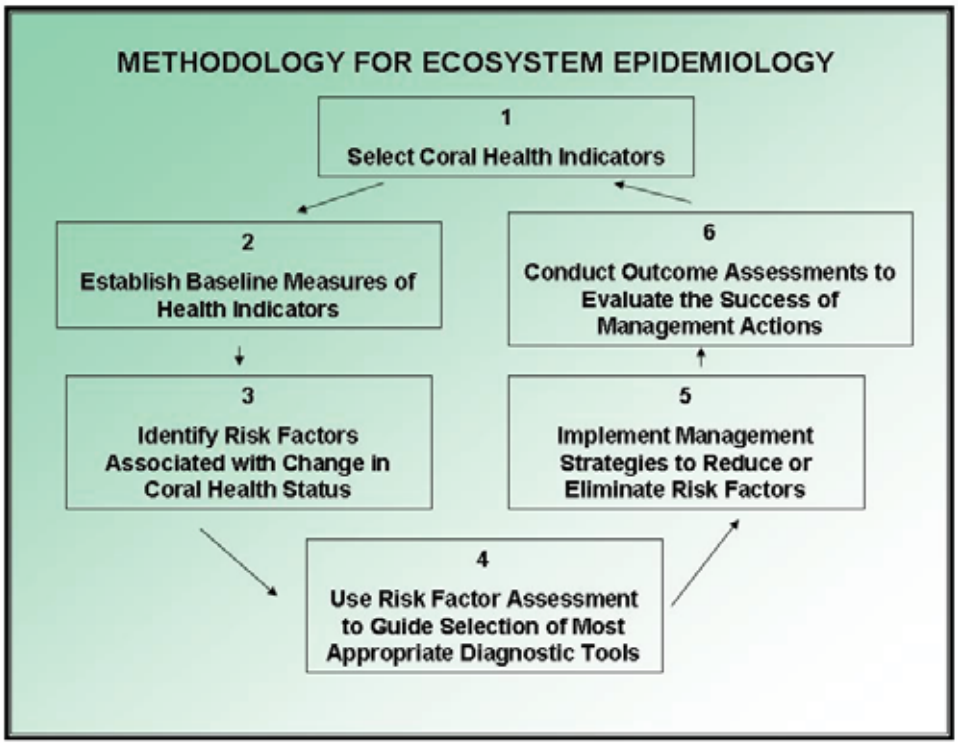

Figure B.1 Methodology for Ecosystem Epidemiology. A six step process that identifies Indicators and Risk Factors for Causal Assessment; Implements Management Strategy and Evaluates the effectiveness of management action.

B. Overall Strategic Objective: **Improve understanding of the causal links involved in coral reef decline to better inform decision makers for health management of reefs.**

B. Overall Recommendation: Adopt a formal environmental assessment framework that integrates ecological epidemiology with ecological risk assessment to provide decision makers with a coherent and consistent description of risks associated with management options that is transparent, reproducible and defensible.

In the following sections, the TEEWG identifies a series of key steps in the application of an integrated environmental assessment framework based on ecological epidemiology, that can help improve the detection, identification, and remediation (cure/management) of coral reef diseases and increase our understanding of the incidence, distribution and causes of harmful effects of chemical, physical, or biological agents (i.e., ecological epidemiology)(Suter 1993) on coral reef communities. The methodology involves:

1. Development and implementation of an ecological monitoring program to characterize coral community structure and function (Objective 1)
2. Establishment of baseline risk factors present at the site (Objective 2)

3. Use of epidemiology to identify potential risk factors associated with change (e.g., toxin, emerging disease) if a health change is detected, (Objective 3)
4. Use of these data to choose the most appropriate diagnostic tools to assess etiology, recognizing that the cause is likely multi-factorial (Objective 4)
5. Implementation of practical management strategies with the objective of reducing or eliminating risk factors associated with coral disease (Objective 5)
6. Conduct outcome assessments to evaluate the success of the management decisions (Objective 6)

Strategic Objective B.1 Identify and measure indicators of coral health and disease (morbidity) at the following levels: ecosystem, community, and individual for assessing condition and detecting impairments.

Recommendation B.1.1: Adopt a unified list of indicators of coral health and disease.

Morbidity – the relative incidence of disease

Bioindicator / Biomarker – a distinctive biological or biologically derived indicator (as a metabolite) of a process, event, or condition (as aging, disease, or oil formation)

Indicators of health and disease are often referred to as bioindicators or biomarkers. There are three general classes defined as biomarkers of exposure, effect or susceptibility. Changes in these biomarkers are used to identify delayed or sublethal effects in individuals that survive an initial exposure to an adverse event. They can reveal exposures that result in compromised health as well as help define causal linkages and risk of adverse health effects. The most crucial characteristic of a health indicator is that it not only can detect biological changes but has diagnostic value in determining the nature of the change in association with a given stressor(s). Health indicators can range from remote satellite imagery to subcellular biochemical or cellular physiological endpoints. Integrating across levels of biological organization from cellular parameters to higher levels can help develop mechanistic profiles for certain cellular functions and disease states, and contribute to a suite of indicators for overall performance. The behavior of these indicators and the identification and quantification of pattern changes provides a basis for defining health status (i.e., diagnosis) and providing a prognosis.

An initial list of biological indicators at the ecosystem, community and individual level is presented in Table B.1, and examples of the type of information they may produce.

Table B.1 CORAL HEALTH/DISEASE INDICATORS

	Indicator	Comments
ECOSYSTEM		
	Ecosystem structure and function	
	Calcification balance	
	Trophic level interactions	
	Ecosystem metabolism	
	Bioturbation	
	Changes in algal community composition and cover (abundance/biomass)	Increases in certain types of algae are indicative of impacts associated with increased nutrients and/or removal of herbivores.
	Bioindicators, e.g., foraminifora	
COMMUNITY		
	Species diversity and abundance	Identify organisms of special interest and their abundance: species of high commercial value, species of ecological importance, or pest species such as corallivores
	Bioerosion	
	Bleaching event	Extent of bleaching, species or genera affected,
	Rugosity/topographic analysis	Change on reef structure (topography or structural complexity) over time. Especially relevant for communities dominated by branching corals that may be detached and flattened during storms
	Bioindicators/symbionts: e.g. Butterfly fish (prevalence, feeding rates); stomatopods; sea cucumbers;	
	Catch per unit effort for reef fish.	

Table B.1 CORAL HEALTH/DISEASE INDICATORS (Cont')

	Indicator	Comments
POPULATION		
	Age (size)	Representation of different size classes
	Recruitment	Diversity and abundance of coral recruits, an indication of the potential for recovery and whether a reef is a source or sink.
	Intraspecific genetic diversity	
INDIVIDUAL		
	Bleaching	Amount of colony surface bleached, duration of bleaching, patterns of recovery and/or partial or total mortality
	Respiration/individual metabolism	
	Change in immune response	Susceptibility to disease
	Coral morphology	Growth form may be an alternate measure of species diversity
	Zooxanthellae content	
	Skeletal density/skeletal strength	
	Density banding.	
	Depth of tissue penetration	
	Abnormal growth/hyperplasia.	
	Growth/division rate of zooxanthellae vs coral growth rate. Growth rate; ratio; photosynthesis/respiration; proteins in PCP complex; photosynthetic efficiency	
	Autotrophic/heterotropic status of coral: C^{13} analysis, retrospective	
	Fecundity, gamete production. Changes in life history stages.	

Strategic Objective B.2: Establish a baseline of health and disease indicators

Recommendation B.2.1: Implement targeted surveillance programs for monitoring coral health and detecting biological change to develop a condition assessment.

The TEEWG identified examples (Table B.2 and Table B.3) of parameters, tools, and data that could provide detailed information on the structure, composition, functioning and health of the community. All of the variables identified may not be relevant to every region/location. For each location a detailed review of existing monitoring efforts, available baseline information and known threats should be undertaken to establish core baseline data variables. The TEEWG pointed out that the most prominent indicators in use today are associated with mortality and therefore identified a substantial need for more indicators of coral morbidity (rather than mortality). Examples include lesion regeneration rates, molecular indicators of stress, measures of genetic integrity, cellular physiological parameters indicative of immune status, detoxification, metabolism and various cellular and tissue-level processes. As new indicators for detecting biological change are identified, adopting a variable should be based on the criteria highlighted in the inset.

Criteria for Selecting a Biomarker

- Relevant
- Measurable
- Easy to collect
- Cost-worthy
- Reliable & valid (trustworthy)
- Amenable to standardized collection protocols
- Comparable

Table B.2 INDICATORS OF CORAL HEALTH & DISEASE: FIELD OBSERVATIONS: Variables are ranked in terms of difficulty in acquiring information and reliability of measurements with the minimum recommended frequency of monitoring. Difficulty of collecting: 1 = no training, no extra cost, less than 10 minutes during a survey; 5 = augmented funding, years of training, high cost, long time to collect). Reliability: is there an accepted protocol in place or standardized protocol that would be accepted? 1 = low trust in data; 5 = high trust, given that consortium has come up with an accepted protocol and data are collected using this protocol.

Variable	Difficulty	Reliability	Minimum Required Frequency of Monitoring
Species diversity	3	5	Annually
Trophic level interactions	2	3	Annually
Benthic community analysis	2	5	Annually
Remote sensing data	4.5	4	Annually
Population demographics • Age/size • Recruitment	 2 2	 2 5	Every 2 years
Bioerosion	1	5	Annually
Bioturbation (high, med, low: categorical designation)	1	5	Annually
Fish indicators: catch/unit effort; transects "fish counts"	2	5	1-4 times/year.
Bioindicators: sea cucumbers, butterfly fish.	1	3	At least annually
Algal blooms	1	5	Event driven/choice.
Rugosity	1	5	Every 2 years
Algal cover	2	5	Quarterly
Benthic algal community	2	5	Seasonal; 4 times a year.
Bleaching	1	4	Choice/ annual
Abnormal tissue growth	1	2.5	Annual
Fecundity assessment: Gamete presence/pigmentation	1	3.5	Seasonal
Human activity--use observations to guide further analysis.			
Natural: provide a list of potential risk factors.			

Table B.3 INDICATORS OF CORAL HEALTH & DISEASE: Laboratory Data (Definitions same as Table 2.2)

Variable	Difficulty	Reliability	Required frequency of monitoring
Calcification balance	3	5	Once
Life history stages; bioassays on different stages; controlled laboratory exposures	4	4	Most spawning events are annual.
Bioerosion--^{15}N isotope analysis.	1	5 (data) 3.5 (interpretation)	Once
Bioindicators: foraminifora	2	5	Annually; event driven
Algal blooms: algae characterization	2	5	Event driven/choice
Skeletal density.	1	5	once; opportunistic/event driven
Density banding	2	5	Once
Fecundity, gamete analysis (quantify, viability, fertilization study) Gamete histopathology	4	Annually or seasonally	
Sediment analysis: size, petrographic	1	4	Once
Water quality: pH, turbidity, salinity, temperature, wave action, e.g., routine YSI measurements.	1	4	Routinely
Tissue depth	ND	ND	RESEARCH NEED
Zooxanthellae: growth rates, ratio, photosynthesis/respiration; PCP complex. Normals yet to be established.	ND	ND	RESEARCH NEED
Autotrophic/heterotrophic balance. ^{13}C	Requires large sample size		RESEARCH NEED
Intraspecific genetic diversity	ND	ND	RESEARCH NEED
Contaminant chemical analyses: persistent organic pollutants (POPs). Water, tissue, skeletal.	1-5 metals: 1 persistent organics: 5	Variation--established methodology for some, not others.	RESEARCH NEED Testing based on probability; not shotgun contaminant analysis. Tends to be patchy in time and space.

Strategic Objective 3: Identify risk factors associated with a change in coral health status.

Recommendation 3.1 3.1: Establish site specific risk factors that may affect the location of interest and incorporate these into research and monitoring programs.

The TEEWG identified an initial list of possible risk factors (Table B.4) that may be associated with coral disease outbreaks. All categories of risk factors are not applicable to all situations. Potential risk factors must be measurable and quantifiable to allow detection of associations.

Many of the risk factors (i.e., causal factors) are anthropogenic in nature and affect water quality either from land-based sources of pollution or groundwater discharges. As these predictors of coral disease are more specifically characterized, the TEEWG identified types of anthropogenic and natural risk factors to consider in developing research and monitoring programs. These risk factors include:

- Anthropogenic (human activity)
  - Agricultural
  - Manufacturers / Industrial
  - Aquaculture
  - Fishing
  - Residential Activities
  - Recreational Activities
- Natural (general environmental)
  - Pathogens
  - Climate
  - Water Quality (temperature, salinity, turbidity, etc)

Strategic Objective B.4: Use risk factor assessments to choose the most appropriate diagnostic tools.

Recommendation B.4.1: Standardize methodologies for all variables.

The cause of most coral diseases are likely multi-factorial and investigations of these factors require a trans-disciplinary approach, drawing on many types of information to develop quantitative comparisons among groups and various factors. Adopting an Integrated Environmental Assessment (IEA) provides a logical, defensible and systematic approach to understand the complexities of disease. It blends concepts and methodologies of ecological epidemiology (i.e., biological assessment and causal analyses) with risk assessment (i.e., risk models that link alternative decisions to future conditions) to provide a systematic means to better identify causal factors and their path from source to impairment. A deliberate environmental assessment will provide a

Table B.4 IDENTIFICATION OF RISK FACTORS ASSOCIATED WITH A CHANGE IN CORAL HEALTH STATUS

PREDICTORS OF CORAL DISEASE (Risk Factors)	Measurement
Agriculture	
Sediment runoff	Measure: turbidity, sediment traps, YSI [*sediment--can be held by fleshy algae, re-release of metals/toxicants. Both measure of stress on reef and recorder of historical stress. Lead to causation.]*
Pesticides, herbicides	Measuare: quantify products. Separate water soluble and lipophilic--divide out risk, which associated with which disease processes. Also consider effects on different life history stages.
Fertilizer	Measure: Nitrogen, Phosphorus, Sulfur, heavy metals. Can fingerprint nitrogen (isotope suite); can tell fecal waste from industrial waste. Chlorophyll a: best measure of nutrient availability in the water
Salinity changes due to freshwater runoff	Measure: salinity
Animal waste	Measure: coliforms (total enterococci), antibiotics or other pharmaceuticals
Industrial/manufacturing:	
Oil/hard mineral extraction.	Measure: industrial solvents, processing waste, petroleum products, Inorganic waste: easy to monitor and use as diagnostic fingerprint (e.g. vanadium) Evolution of chemicals used in manufacturing. Testing done does not look at effect on coral reef organisms.
Mining coral, beach sand (destructive, but not toxic).	
Ore transport: potent source of nasty things. Measure: easy to monitor/identify--e.g. mussel shells.	
Perchlorates--present everywhere, in high levels in corals.	
Landfill runoff	
Mining tailings (sediment waste produced by mining).	
Marine transport/maritime activities	
Anti-fouling paints, copper used (processing waste). Diagnostic indicator of a certain type of activity	
Ballast water	
Oil spills/grounding	
Military activities	
Historical artifacts: bombs, sunken ships,	
Fishing activities	Measure: recreational, commercial, cultural, economic: fleshy algae dominate if consumers removed
Destructive fishing practices--dynamite fishing, may predispose to ciguatera, etc	
Overharvesting of large predators; effects on the food chain.	

Table B.4 IDENTIFICATION OF RISK FACTORS (Con't)

PREDICTORS OF CORAL DISEASE (Risk Factors)	Measurement
Mariculture/aquaculture	
Shrimp ponds: a major source of toxic materials, shoreline modification process. loss of mangrove ecosystem, biological filters. (Demonstrated human health impacts in Indonesia with shrimp ponds.)	Measure: presence or absence of aquaculture, distance, species cultured, type of aquaculture, pathogens
Escape of invasive species, predation	
Pond vs. cage: different downstream epidemiological implications	
Introduced pathogens	
Pharmaceutical/personal care products	
Endocrine disrupters	
Residential activity	
Household pesticide use: contributes to non-point source pollution.	
Chemical; pharmaceutical/personal care products	
Sewage	Organic: **Measure** coliforms (E.coli not a good measure in seawater) total enterococci; estrogen. Inorganic: **Measure** stable isotopes of nitrogen, caffeine--measures of human plumes
Polybromated diphenylethers: plasticizers, persistent. Industrial chemical, but also residential.	**Measure**: prevalence in water; if associated, look for primary source.
Recreational activity: water quality vs. direct impacts	
Water quality	
Hotel industry / golf courses / residential--sewage management.	Measure: Concentration of visitors/area as a risk factor for coral health.
Jet skis/waterskiing; pollution. Only burn about 80% of fuel.	
Divers as vectors of disease	
Direct impacts	
Water sports: anchoring dive boats, stepping on corals	Measure: number of snorkelers/divers in the area.
Jet skis: compression waves, effect of vibrations on fish.	Measure: compare management practices e.g. Palau vs. elsewhere. Presence of mooring buoys, controlled access. Jet ski rentals.
Anchor damage	
Invasive species--any maritime transport could vector. People or equipment. Algae, diseases, invasive species	

Table B.4 IDENTIFICATION OF RISK FACTORS (Con't)

PREDICTORS OF CORAL DISEASE (Risk Factors)	Measurement
Long range transport:	
Maritime: Ballast water	
Commercial: Transport of materials: e.g. sand	
Humans as fomites: wetsuit transport of disease	
Socioeconomical; ethics/values	
NATURAL	Consider with all risk factors that there are synergistic effects/multiple factors involved Measure: look at geological record for baseline. Case control: what could be the control time or location (place or time in which risk factor did not exist.)
Baseline processes	
Bioerosion	
Disease	
Climate:	
Water temperature	
UV	
Change in weather patterns	
Ocean level related to die-offs.	
Sea level fluctuations in geological time: big killers of reefs.	
Volcanoes/tectonic activity/volcanic ash	
Long range transport: Atmospheric transport--African dust	
Substratum/water quality Affected by runoff and sedimentation; heavy rainstorms, events occur naturally.	
Community	
Natural pathogens part of coral reef system	
Crown of thorn starfish, corallivorous gastropods and fishes	
HABs: cause of anoxia, coral damage. Natural vs. anthropogenic?	
Population	
Intraspecific variation/protection against population demise.	

quantitative basis for informed management decisions (Suter 2006). While relatively few diagnostic tools are available for corals, tools and approaches routinely applied to the study of other wildlife and human diseases are available to adapt for the study of coral diseases. These tools should be evaluated and tested on corals, with the goal of their application in a routine, standardized manner to Pacific coral disease and health studies. The TEEWG identified key actions that can help achieve standardized methodologies and integrate them into standard practices in the field of coral reef health assessments:

- Solicit standardized protocols from subject matter experts
- Publish selected protocols in peer-reviewed literature and central handbook (hard copies and web-based)
- Provide training for standardized protocols
- Educate users in the importance of standardized data to participants

Recommendation B.4.2: Develop and pilot a plug-and-play database

Standardized methods and protocols will help provide uniformity in data reporting and facilitate analyses and interpretation. However, the available data currently resides in a variety of databases and there is no integrated or centralized portal available to support the organization, analysis or interpretation of data that may be obtained through the IEAs outlined in Recommendation 4.1. The TEEWG recognizes that it is imperative to synchronize data from institutions to central location that is accessible, and is also equipped with computational tools to interrogate the data, conduct analyses and synthesize data into usable information for management decisions. To address this recommendation will require the creation of a sub-committee to develop such a database and agency support to house and maintain the database and develop analytical tools for end users. The TEEWG also pointed out that communication with participants and key stakeholders is critical and could be facilitated by providing an annual summary report, a valuable communication tool.

Recommendation B.4.3: Capacity building

The approach outlined by the TEEWG is not commonly used in the coral reef research and assessment community, yet it provides a valuable new thinking process for problem solving that logically organizes information, develops causal pathway models and builds weight of evidence arguments. This provides a transparent course of action to develop compelling information for causation and causal links that is vital for management decisions and selection of appropriate management actions. To successfully implement this integrated approach to environmental assessment will require education for the users. To this end, the TEEWG identified 7 key actions:

- Identify and acquire personnel (empower local resources & use traditional knowledge)
- Conduct training courses with subject matter experts
- Establish local infrastructure
- Ensure open communication / training among data collectors
- Address data sharing concerns regarding publication

- Assess potential reporting requirements with federal funds
- Consider using data routinely to:
  - Communicate with politicians, managers & legislators
  - Conduct long distance diagnostics with remote subject matter experts
  - Enable evidence-driven decisions
  - Identify risk factors (anthropogenic & natural)
  - Assess response to policy changes and other mitigation strategies

Strategic Objective B.5: Implement management strategies with the objective of reducing or eliminating risk factors associated with coral disease.

Recommendation B.5.1: Adopt an adaptive management approach whereby specific risk factors of concern are reduced or eliminated in certain areas.

To achieve a position of proactive coral health management requires being equipped to recognize new and reemerging infectious as well as non-infectious disease conditions, and understand the factors involved in disease emergence, prevention, and elimination. This requires:

- Adopting a methodology appropriate for assimilating and synthesizing numerous and diverse data points that encompasses the ability to detect chemical, physical and biological impairments; identify sources and pathways leading to the impairment; predictive capabilities to estimate risks (e.g., societal, economic, environmental) for different management options; and a means to evaluate the success of the management decisions.
- Providing training courses for equipping individuals to conduct risk analysis and ecological epidemiology and translate these analyses for decision making.

As Pacific Coral Reef Management evolves, it is critical to acknowledge, embrace and incorporate the traditional system of resource management into each of the steps in the process. A wealth of knowledge and success is espoused in these traditional methods that need to be incorporated into any contemporary coral reef management regime. Pacific Islanders are in tune with their local environment and are keenly aware of indicators of a healthy ecosystem as well as those that strike an alarm of impairment. Because of this knowledge and inherent value and respect this culture brings to coral reef management, it is important that it play a prominent role in developing a surveillance system to work with contemporary scholastic knowledge to understand and identify causes of ecosystem impairment and solutions. The Pacific Islander culture also provides a vital quality: once a problem is recognized they take local ownership and action to attain the solution, quickly before further harm is done to their resource. Given the vast area of Pacific coral reefs, and the limited capacity per area, training and capacity building efforts should empower local resources and take advantage of traditional knowledge.

Recommendation B.5.2: Identify a central facility to compile and share information in a timely manner with researchers, managers and other stakeholders, and to train local responders in risk factor assessments.

When conducting condition assessments, causal analyses, risk assessment or epidemiological investigations it is important to summarize the investigation in a report that includes the reason for the investigation; general summary characterizing the investigation, the clinical descriptions, results and possible source and conclusions on the nature of the disease, source of outbreak and method of transmission and any possible recommendations for control or management. These reports should be provided to relevant resource managers, researchers participating in the assessment, key stakeholders, and other decision makers in a timely manner to allow implementation of management responses, as necessary, as soon as possible after identification of the event. This will be best achieved through:

- Centralized facilities and web-accessible databases to compile, analyze and share data and information in a timely manner;
- Involving experts capable of conducting detailed analysis of these data, including local participants, with the goal of developing a hypothesis to explain the most likely cause, source and risk of distribution of the cases and suggest tools and strategies to mitigate the disease and or its impacts.

Because many Pacific communities still utilize traditional management systems it is important to ensure local ownership of the problem/solution and encourage local participation at every stage of the process while reaching resolution of the problem.

Strategic Objective B.6: Conduct outcome assessments to evaluate the success of the management decisions.

Recommendation B.6.1: Institute performance measures appropriate for evaluating the success or weakness of each component of the environmental assessment process, decisions and actions.

Once a problem has been detected, Resource Managers attempt to determine causes and evaluate solution options. Although their decisions are based on the 'best available science', it is essential to have a means to evaluate the performance of their actions, detect inadequacy in the evidence (i.e., science) used as a basis for their decisions or determine whether the action was effective. This may be accomplished by comparisons to similar areas without management intervention or through monitoring and surveillance to determine whether changes have occurred compared to baselines. This evaluation is key to identifying knowledge gaps and directing research and monitoring activities strategically in support of a successful adaptive management process.

Figure B.2 Integrated Framework for Environmental Assessment. *Adapted from Cormier & Suter 2008.*

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C. PATHOLOGY OF DISEASE

Identifying Diagnostic Tools Necessary to Adequately Characterize the Pathology Associated with Coral Disease

Background

Over the past three decades, coral reefs worldwide have experienced significant losses in living coral cover and changes in the structure and function of these communities. Infectious diseases have been recognized as a prominent cause of mortality in scleractinian corals in the western Atlantic since the 1980's, but until recently there were few reports of coral disease from the Indo-Pacific region. Current efforts to systematically assess the types and prevalence of coral disease in the Indo-Pacific suggest that coral disease also occurs commonly on Indo-Pacific reefs, and these diseases may have a greater role in structuring coral communities in the region than previously thought. Unfortunately, few diseases affecting Indo-Pacific corals have been adequately characterized, quantitative data on the spatial and temporal variability of diseases and their impacts are lacking for most locations, and linkages between environmental parameters and diseases affecting Indo-Pacific corals are unknown.

'Disease' is a word with many different connotations, depending on one's particular perspective or experiences. Coral biologists use disease almost exclusively to describe gross changes in a coral's appearance and usually assume that a disease is due to an infectious agent. Disease however, includes *"any impairment that interferes with or modifies the performance of normal functions, including responses to environmental factors such as nutrition, toxicants, and climate; infectious agents; inherent or congenital defects; or combinations of these factors"* (Wobeser 1981). Therefore, as in other animal diseases, it is imperative that an unbiased approach is used when investigating coral disease.

Literally speaking pathology is the study (*logos*) of suffering (*pathos*); practically, it involves studying the structural and functional changes in cells, tissues and organs that define disease processes. There are four aspects that are investigated to understand disease processes: *etiology, pathogenesis, morphologic changes, and clinical significance* (Kumar et al. 2005). It is in this context that adopting concepts and principles of pathology will help to organize our observations and direct conclusions about coral disease in a rigorous and organized manner. Two recent publications address these issues for coral pathology (Work and Aeby 2006; Work et al. 2008b).

Etiology: its cause

Pathogenesis: mechanisms of disease development; sequence of events in response to etiologic agent

Morphologic changes: structural alterations in cells and organs

Clinical significance: functional consequences of the changes

Challenges and Recommendations:

The *Pathology of Diseases Working Group* (PDWG) was charged with identifying gaps in knowledge, research needs, and potential approaches that could be used to address pathology, pathogenesis and etiology of diseases in animals applicable to corals with appropriate modifications. Given the limited knowledge of the cause of many diseases in corals and the lack of uniformly applied methods to investigate disease, this topic was considered critical and timely.

The challenge for coral pathology is to develop approaches, procedural guidelines, and analytical methodologies that take advantage of the advances made in the study of the pathology and pathogenesis of disease in humans and other animals. There was general agreement within the group that the study of diseases in corals suffers from a lack of systematic investigation, particularly in regards to establishing case definitions and arriving at causality of disease. A case definition encompasses all the factors that define a particular disease, and can serve as a standardized point of reference for tracing the disease across different populations or geographic areas. Case definitions can change as new data on the particular disease appear. As a starting point, case definitions for newly described coral diseases should include good morphologic descriptions encompassing gross and microscopic pathology. More complete case definitions will include information about the causative agent and the pathogenesis of the disease. Many of the methods currently used to characterize diseases in terrestrial and other marine animals are applicable to corals.

For diseases that are novel or previously undescribed, carefully controlled laboratory studies can help elucidate the pathogenesis and cause of the disease. This includes studies evaluating host-agent interactions such as exposure of corals to suspected infectious or non-infectious agents in attempt to replicate clinical signs observed in the field. In cases where an etiologic agent is suspected to be necessary and sufficient to cause disease, this can be demonstrated through the use of Koch's postulates, where the animal is exposed to the isolated infectious agent, clinical signs reproduced, and the agent re-isolated from the animal (Work et al. 2008b). Unfortunately, Koch's postulates have often been applied to coral diseases based on identification of external characteristics (e.g., disease signs) without more detailed investigation of underlying cellular and structural characteristics of the experimentally reproduced lesion. These efforts have failed to distinguish between *primary* and *opportunistic* pathogens and have served only to enhance confusion in the literature. In addition, many diseases are complex making them difficult to study using Koch's postulates alone. Furthermore, many marine microorganisms are not culturable in laboratory settings thereby complicating their experimental manipulation. Through application of culture-independent methods, the presence of multiple disease-associated pathogens may be identified. In addition to traditional methods of morphological pathology (e.g. histopathology) and culture methods, the application of genomic, proteomic, and metabolomic-based approaches may be necessary to understand the pathology, pathogenesis and etiology of coral diseases.

General Recommendation: Develop key questions that might be asked in regards to a disease outbreak in corals.

At the outset, there was a consensus that great confusion existed on nomenclature of gross lesions in corals and that a good morphologic description provided a foundation for describing any disease. This led to considerable digression and discussion, however, in the end, the group decided on a decision tree that would give a broad outline on the process of disease investigations. In addition, three products were identified that were judged critical to sorting out existing knowledge gaps regarding disease in corals. These products included:

Key questions related to a disease outbreak

1. How do you describe the disease?
2. Does it have a significant demographic effect?
3. Does it move rapidly?
4. Do corals recover?
5. Do you know what causes it?
6. Does it correlate with environmental factors?

- Field identification cards for major lesions in corals from the Pacific.
- A summary of existing approaches to coral disease diagnostics.
- An approach to arrive at the suspected etiology of infectious disease in corals.

Strategic Objective C.1: Develop a decision tree for standardized investigation of coral diseases.

Recommendation C.1.1: The disease investigation process should follow a standard course of events.

An unusual mortality or morbidity event is signaled via presence of dead or dying corals (field signs) (Fig. C.1). Recognition of the event is followed by a systematic description of lesions (morphology) in affected corals leading to a decision point. Either a management decision is made (Management) based on field signs and gross morphology (e.g. continue observing, implement intervention, do nothing) or samples are taken for further laboratory diagnostics (Sample). The focus of laboratory diagnostics is to arrive at the cause of a lesion or to begin building a database of information that will add to the foundation of the case definition (morphology). If a causal agent is identified (or as information is accrued from the laboratory), this information is fed back to management. This communication has several purposes. First, it promotes "buy in" to the disease investigation on the part of management. Second, sharing of laboratory information with managers provides a forum for generation of further hypotheses and further sampling to arrive at cause of disease. Finally, if an etiology is identified, input from managers is critical in helping elucidate the ecology of the agent so that the disease can be effectively mitigated or potentially stamped out.

Strategic Objective C.2: Develop a field guide of common lesions observed in corals from the Indo-Pacific.

Recommendation C.2.1: A simple field guide to common lesions of corals in the Indo-Pacific should be developed, based on the approach identified for western Atlantic corals. This guide would include: accepted common name of lesion, morphologic description and representative photos (distance and macro).

Over the last 35 years coral reef researchers have identified and named over 50 diseases in scleractinian corals through field monitoring programs and targeted coral disease

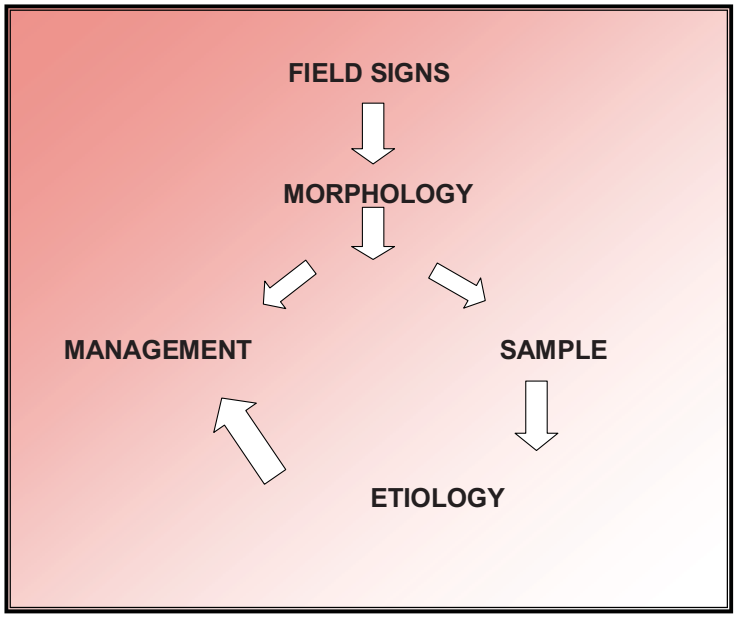

Fig. C.1 Disease Outbreak Response

research projects (Bruckner 2007; Green and Bruckner 2000; Weil 2004). While these observations have increased the visibility of coral diseases and have led to the recognition of the importance of coral diseases as a community structuring agent, the lack of a standardized approach to describe diseases has caused much confusion thereby limiting our ability to apply management tools in order to prevent disease occurrence and spread. Currently, coral disease is typically diagnosed in the field by identifying lesions, with comparative observations by different researchers and in different regions relying primarily on available photographs of gross signs and general descriptions based on the locations of lesions, the color of affected tissue or exposed skeleton, species affected, and rates of mortality. Unfortunately, this has led to a profusion of new names, including the use of different terminology to describe presumably similar gross field signs and similar terminology for syndromes observed in different ocean basins that have vastly different signs. Part of the problem has been that those describing coral diseases often infer causation based on gross appearance alone, however, the determination of causation is

something done more appropriately in a laboratory setting. Some progress has been made towards standardizing nomenclature of coral lesions (Work and Aeby 2006). More recently, the CDHC convened a workshop to establish diagnostic criteria (including nomenclature and case definitions) for coral syndromes affecting western Atlantic corals. Through this workshop, the CDHC developed a three-tiered approach to identify and differentiate coral diseases (Raymundo et al. 2008; Work and Aeby 2006)

Strategic Objective C.3: Review of existing laboratory methods to investigate coral diseases.

Recommendation C.3.1: Develop a white paper on investigative processes applied to coral diseases.

This review should include detailed information on pathology, microbiology (including bacteriology, virology, mycology and protozoology), toxicology, genomics/proteonomics and parasitology.

A variety of methods exist to investigate various aspects of coral disease, however, whether these methods are sufficiently standardized or adequate as currently applied remains questionable. The group recommended that a comprehensive literature search be implemented to review what methods have been used to investigate diseases of corals (from sampling to analysis), their limitations, and their potential.

Strategic Objective C.4: Identify a standardized approach to elucidate etiology of disease in corals.

Recommendation C.4.1: Assemble a model approach that could be used to determine whether a particular etiologic agent would have high probability of being associated with (or causal of) a lesion.

The model approach is based on answering certain critical questions:

Can a potential etiology be consistently visually associated with the lesion? For example, in some cases, an etiologic agent (bacterium, fungus, parasite or virus) is visibly associated with cellular damage either at the light or electron microscope level. Strong presumptions of causality can be inferred in cases where such findings are consistently associated with lesions.

Is the lesion transmissible? The group judged that transmission experiments could be done in the field (and also the lab) under the following conditions:

- These are limited to a restricted geographic area (currently unspecified but probably carried out in the immediate area such as a 0.5 km radius).
- That healthy fragments be attached to diseased colonies (and not vice versa).
- That appropriate controls be run for all transmission experiments.

[**Editor's note:** In the final plenary session of the Workshop, the participants of all the working groups discussed field transmission experiments and decided they should be put

on hold until specific guidelines could be developed. See the OPINION paper by Cheryl Woodley in Appendix VI.]

The illustration below (Fig. C.2) shows the general concept in determining the nature of a communicable agent. If field or laboratory trials indicate the lesion to be communicable, subsequent experiments are moved into the laboratory. There, tissues from the diseased corals are extracted (methods vary), filtered or unfiltered extracts are inoculated onto susceptible colonies, and those observed for development of lesions. If lesions are reproduced using 0.1µm filterable extracts, it is assumed that causative agent is subcellular element (virus, protein, nucleic acid or chemical). If lesions are reproduced using non-filtered extracts, it is assumed that causative agent is cellular (e.g. bacteria, parasite). A more comprehensive schema and approach is available elsewhere (Work et al. 2008b)

For non-filterable agents, clues can sometimes be gained by visual association (e.g., light or electron microscopy) as to its identity. In many cases, however, there are too few organisms to visualize effectively, and attempts must be made to culture. Although many bacteria in the marine environment are not culturable, attempts should be made to rule out culturable bacteria by using a variety of selective and non-selective media (including anaerobic conditions) to compare flora between sick and healthy individuals in efforts to target potential organisms in trials to fulfill Koch's postulates. Genomic approaches can also be used to compare sick and healthy corals; however, because of the large number and variety of organisms detected using these methods, large samples sizes may be necessary and this approach, though helpful in generating hypothesis, rarely gives a definitive cause of the lesion. Methods to detect culture-independent flora associated with corals are still under development and will continue to develop. In addition, there is a need to develop primers to detect bacteria associated with corals.

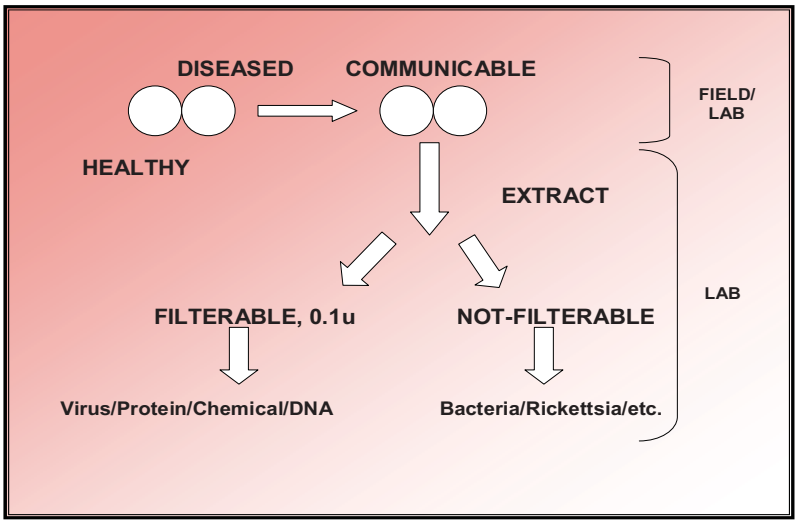

Fig. C.2 Scheme for Determining the Nature of a Communicable Disease

Standard methods exist to identify filterable agents such as viruses. These include electron microscopy, sucrose density gradients (physical separation based on density), degenerate primers and PCR, and susceptibility to chloroform or ether (to assess whether enveloped or unenveloped particles are present). A major limitation to the study of virology in corals is the current lack of laboratory cell culture systems (see Recommendation 3.2 in the PDWG section indicating the need for coral cell lines). Standard methods also exist to identify filterable agents that are not viruses. Extracts can be treated with chloroform and the aqueous and lipid soluble phase assayed for effect (lesion) in corals. Such methods coupled with gas chromatography-mass spectrometry can help identify compounds (chemicals) that may be associated with presence of lesions.

Strategic Objective C.5: Build critical scientific capacity in the field of coral pathology and offer a health management perspective in resource management.

Recommendation C.5.1: Create and Support Advanced Educational Opportunities.

Strategic Objective C.6: Improve capacity to Manage Coral Disease Outbreaks.

Recommendation C.6.1: Establish a Coral Disease Outbreak and Unusual Mortality Response Program.

A response program should be developed that involves a National Center that provides guidance in responding to disease outbreaks and serves as a repository for information, regional coordinators and local responders. The National Center should organize training programs for Response Teams in strategic Pacific and Caribbean locations and whenever possible should assist in the investigation of coral disease outbreaks, facilitate processing of samples, and ensure relevant results and recommendations are provided to resource managers, participants and stakeholders in a timely manner. The National Center should also develop, with input from experts, a manual with a set of tools and procedures for investigating coral disease.

Pathology of Disease Working Group Members:

Thierry Work (Chair) – USGS, Honolulu, HI
Julie Higgins (Recorder) – NOAA NOS, Charleston, SC
Andrew Bruckner – NOAA NMFS, Silver Spring, MD
Dave Jessup - California Dept. Fish and Game, Santa Cruz, CA
Robert Jonas – George Mason University, Fairfax, VA
Ariel Kushmaro- Ben-Gurion University, Be'er-Sheva, Israel
Emmett Shotts – USGS (retired), Cleveland, GA
Pamela Parnell - Professional Veterinary Pathology Services, Columbia, SC
Meir Sussman - James Cook University, Townsville, Australia
Bette Willis - James Cook University, Townsville, Australia

D. PREVENTING AND RESPONDING TO CORAL DISEASE IN THE PACIFIC REGION: MANAGEMENT PERSPECTIVES

Background

Coral reefs are biologically diverse ecosystems that provide numerous economic and social benefits including sources of revenue and jobs as well as a variety of ecosystem services such as shoreline protection, recreation and ecotourism. In addition, they provide biomedicals, minerals, chemicals, food, curios and ornamentals, and building materials to over 100 developing and developed countries. Impacts associated with landscape changes that introduce sediments and pollutants (e.g., agriculture, industry, coastal development and physical alteration of habitats) and over-exploitation of coral reef resources are among the most pervasive localized stressors. Furthermore, localized impacts are being compounded by threats from global climate change such as increased sea water temperatures, elevated UV radiation and changes in ocean acidity. While we already know these ecosystems are easily damaged, we are only beginning to understand what can be done to prevent continued degradation. The complexity of these ecosystems, along with a growing list of human activities and demands placed on them by multiple user groups, is an enormous challenge for managers, who must find a balance between protection and continued use.

One of the most widely recognized management systems for long term sustainable use of coastal resources involves integrated coastal zone management (ICZM). To achieve ICZM, concurrent steps are undertaken that address anthropogenic threats to coastal watersheds, including implementation of coastal development policies, measures to reduce industrial discharges, environmentally friendly agricultural practices, and sewage treatment measures. To be successful, these measures must be applied in concert with other actions to ensure sustainable commercial and recreational fishing and tourism. For example, typical efforts to mitigate land-based sources of pollution have focused on tertiary treatment of wastes, regulated use of fertilizers and pesticides, controlling nutrient loss and sediment run-off by replanting native coastal vegetation, and environmentally friendly development, dredging and beach renourishment practices. An excellent example of successful coral reef ecosystem management is provided by the Great Barrier Reef Marine Park, where multi-use zoning limits or prohibits specific activities. Fishing, curio collecting and tourism are permitted in certain areas by designating different intensities of use through the establishment of habitat protection zones (e.g., MPAs), National Parks, preservation areas, and general use zones. Together, these steps can help address localized human impacts to reefs; however, successful implementation requires strong government, industry and community support and participation, a strong lead agency, and sufficient capacity in planning, monitoring, education and enforcement.

General efforts to mitigate anthropogenic stressors are likely to reduce pressures enabling coral reef ecosystems to better tolerate natural stresses and more resilient to climate change and bleaching. However, biodiversity at genetic, species and ecosystem levels differs among locations and localized impacts to coral reefs are often site specific and may influence a coral reef's ability to resist disease and/or recover from impacts. These factors require consideration when implementing management strategies. Furthermore, the state of knowledge regarding diseases and disease impacts is highly variable between Pacific and Atlantic regions, and between local jurisdictions. It is likely that additional targeted management measures need to be derived specifically for each jurisdiction, territory, state or country to identify, understand, and respond appropriately to disease events on their reefs. These measures could be identified through an assessment of multiple factors such as the types of reefs and their distribution, biodiversity, social and economic uses of reef resources, existing human and environmental stressors and current state of knowledge of these ecosystems. Management needs for coral diseases should initially focus on 1) building infrastructure and capacity to proactively respond to disease outbreaks; 2) increasing public awareness about diseases and their potential impacts; and 3) collaboration between managers and scientists to fill critical gaps in our understanding of disease in the Pacific. In addition to key proactive management responsibilities, reactive measures geared towards addressing impacts and restoring degraded coral reef ecosystems are also a critical responsibility of the management community.

What do we need from the managers?

1. **Better defined response process**
2. **Monitor the response**
3. **Quarantine the reef or eliminate certain activities**
4. **Restrict or modify activities that may be problematic**
5. **Policy and regulation changes**
6. **Possible depopulation of the reef**
7. **Treatment**
8. **Prevention**
9. **Community outreach**

State of coral disease understanding and management in the Pacific

Coral diseases have been reported on 39 genera and 148 species from 63 countries. The vast majority of all observations to date (86%) are from the wider Caribbean, with only 14% of the records from the Red Sea and Indo-Pacific. Coral diseases (BBD and WBD) were first reported from the Indo-Pacific and Red Sea during the late 1970's and 1980's by a single researcher (Antonius 1977; Antonius 1982; Antonius 1985) working in three countries (Philippines, Egypt and Saudi Arabia). By 1994, diseases had only been reported from six countries, including several new conditions first observed on reefs in Australia. Indo-Pacific diseases appear to be exhibiting a rapid expansion in range and in the types of disease since 2000. For instance, recent surveys conducted in Australia (Willis et al. 2004), western Indian Ocean (Mc Clanahan et al. 2004), Philippines (Raymundo et al. 2005; Raymundo et al. 2003), Red Sea (Loya et al. 2004), Palau (Sussman et al. 2006), Hawaii (Aeby 2005; Aeby 2007) and American Samoa (Work and

Rameyer 2005) illustrate the widespread, global distribution of coral diseases. Through annual and semi-annual monitoring programs, researchers are also identifying coral diseases on a greater number of reefs and species, and at higher levels since the late 1990's, suggesting that diseases have become more prevalent in the Indo-Pacific over the last five years (Kaczmarsky 2006; Raymundo et al. 2003; Willis et al. 2004). This includes reports from new regions that were previously presumed to be unaffected (South Africa and Solitary Islands, Australia), a higher percentage of reefs with disease and recent increases in disease incidence in certain locations (e.g., Great Barrier Reef in Australia), and an emergence of several new conditions.

Based on lessons learned from dealing with disease and the devastating effect disease has had in the Caribbean, coordinated and strategic preventative measures, with a focus on maintaining overall ecosystem health, need to be taken ***now*** in the Pacific Region. Managers need to be engaged with the scientific community 1) to better direct and assist with research efforts, 2) to identify possible options for responses to disease outbreaks, and 3) to identify realistic management strategies for Pacific coral reefs. While efforts to document diseases has certainly increased in the Indo-Pacific, the numbers of trained experts and the numbers of jurisdictions with routine coral disease monitoring programs remains very low. Furthermore, few research activities are directed towards an understanding of causative agents, sources of pathogens, linkages with environmental stressors, monitoring of the impacts of diseases on the physiology/biology of affected corals, or the role of disease in structuring coral reef communities. Some of these limitations may be overcome through educational programs targeted towards graduate students and researchers, and development of centers of excellence in Pacific jurisdictions with the necessary staff, infrastructure and training to process samples and identify and develop specific tools and informational materials directed at coral diseases.

Approaches undertaken to manage or mitigate coral diseases have been limited in scope and the effectiveness of these measures is not fully understood. For instance, massive corals affected by black-band disease have been "treated" by aspirating the microbial band and covering the affected area with clay or underwater epoxy, while antiobiotics have been successfully applied to diseased corals in aquarium environments. Pilot experiments involving the removal of corallivores (e.g. crown of thorns starfish and corallivorous gastropods) have been undertaken to reduce predation pressure on corals, secondarily eliminating potential vectors of disease. Reintroduction of the herbivore, *Diadema antillarum*, is being undertaken in parts of the Caribbean to stem increases in macroalgae, which may also improve the health of corals, thereby indirectly reducing the likelihood of disease. Researchers are also attempting to identify disease resistant clones of certain species of corals, with the goal of propagating and transplanting these into degraded areas. In 2003, the Florida Keys National Marine Sanctuary (FKNMS) closed a portion of the reef to recreational divers in attempt to prevent transmission and spread of a disease affecting *A. cervicornis* (Federal Register 2003). Other efforts have focused on improving resilience of reefs, such as the implementation of no-take MPAs and reduction in the discharge of certain land-based stressors to specific locations; these measures have not been implemented as a strategy to mitigate disease, but they may indirectly reduce morbidity and enhance the health and resistance of corals.

Challenges and Recommendations

There are numerous factors that have hindered recognition by the management community of the importance of diseases and the need for management actions directed towards an improved understanding of diseases, surveillance of the occurrence, distribution and impact, and responsive (proactive and reactive) actions to address diseases. This includes the existence of very limited basic knowledge on locations and species affected by diseases, numbers of different diseases and their abundance, causes, and links to other anthropogenic and natural stressors. Moreover, few studies from the Pacific have quantified the extent to which disease has or could contribute to overall reef decline.

The general lack of knowledge on Pacific coral diseases severely limits our ability to gauge the severity of the problem. In light of current and future funding limitations and a paucity of information on diseases, managers may be reluctant to direct their limited available resources towards implementing proactive measures to address disease and in so doing, fail to protect unimpacted coral reefs from possible disease outbreaks. We need to develop a dialogue with managers that will communicate the urgency to prevent Pacific coral reefs from being thrust on the same trajectory as their Caribbean counterparts; improving our understanding of diseases through strategic research and surveillance as well as developing and implementing proactive conservation measures can help avert this impending threat to Pacific reefs. Engaging stakeholders, and raising their awareness to the benefits of prevention rather than treatment is not only cost-effective but more likely to be successful than efforts to treat diseases and/or restore reefs after diseases have degraded coral reef habitats.

The goal for the managing coral disease should focus on the maintenance or improvement of coral ecosystem health, using a comprehensive ecosystem-based approach through implementation of adaptive management practices. In general, coordination and communication among research scientists and managers can be facilitated with an inter-disciplinary approach that brings scientists and managers together to work closely to address disease, using a single ecosystem approach to science and management that exemplifies the land-sea connection since many potential stressors are believed to be land-based. This includes support for:

What do managers need to know with regards to disease outbreaks?

1. **What is it?**
2. **What is affected?**
3. **Location of infection within the reef, and where is the reef?**
4. **Time frame, seasonality?**
5. **What are the population impacts?**
6. **Is it transmissible?**
7. **What causes the disease?**
8. **What should be done?**
9. **How widespread is the disease in neighboring areas?**

a. **Monitoring and assessment** of the current state of coral reefs and condition of important reef building corals. Current efforts to monitor disease are minimal, and typically include attempts to gather baseline data or opportunistic reporting of disease signs noticed during field research or other routine monitoring. There is a need for adopting standardized disease protocols to ensure signs and stages of disease are reported consistently as well as uniform reporting guidelines to ensure the information is being communicated to the correct management agencies. Institution of these standardized procedures would enhance the opportunity to obtain the funding required to support long-term monitoring efforts.

b. **Research geared towards an improved understanding of potential stressors, causes and sources of disease** such as identification of specific vectors, sources of pathogens, and measurable indicators of change in the health status of corals (e.g., specific biomarker expression). Existing efforts have been primarily directed towards counts of corals with and without specific disease signs, with few studies focused on understanding physiological changes in coral health before the coral manifests visible signs of mortality. Data pinpointing disease sources are also lacking, making it difficult to convince managers, politicians and the public to care about and seek management alternatives to address coral disease.

In other disciplines, such as in most veterinary practices and management of wild animal (terrestrial) populations, cost-benefits of proactive and precautionary management measures have been fruitful. Some of the major actions that have improved animal health without actually treating a disease have included addressing contaminated sources of water, good cleanliness practices**.** Management of human activities is likely to be the key to improve the health of coral reefs, taking into account social systems and considerations of the regulatory/legislative framework, and whether managers are able to be proactive.

Our ability to characterize and address coral disease in the Pacific is hampered by a paucity of spatially and temporally relevant epizootiological data, an incomplete understanding of underlying mechanisms responsible for the occurrence, spread and impact of diseases, and limited technical information and few diagnostic tools to help managers evaluate, track, predict or mitigate diseases. In an attempt to identify specific management needs that can help address coral disease on Pacific coral reefs, the Management Working Group (MWG) identified a series of broad strategic objectives and accompanying recommendations for actions to achieve these objectives.

Vision: To understand and manage impacts to reef ecosystems from climate change, bleaching and disease for increased resistance and resilience by:

a. Understanding the types of diseases present and their distribution;
b. Monitoring the prevalence, incidence and impacts of disease with emphasis on stakeholder participation in monitoring and reporting of bleaching and disease;
c. Determining existing legal mandates and identifying new authorities as necessary to address priority gaps and research needs for diseases and human impacts known to affect the health of corals and other reef organisms;

d. Identifying and mitigating manageable factors that exacerbate the occurrence of diseases and testing the effectiveness of these measures by employing an adaptive management approach;
e. Increasing public awareness regarding diseases;
f. Improving policy support to address diseases and enhance communication among managers, scientists, and policy makers;
g. Implementing training and capacity building programs for managers, graduate students, scientists and other stakeholders with the goal of improving research and management capacity directed towards disease; and
h. Developing tools and technologies to respond to and mitigate diseases and their impacts.

General Recommendations

- **Address management needs for coral disease outbreaks in the U.S. Pacific through the U.S. Coral Reef Task Force Local Action Strategy Process**

The U.S. Coral Reef Task Force (US CRTF 2008) developed a National Action Plan in 2000[1] to improve our understanding of coral reefs and implement actions to mitigate human impacts to these ecosystems. As part of this plan, Local Action Strategies (LAS) were developed in partnership with the U.S. All Islands Coral Reef Committee during the fall of 2002 to help increase and link the goals and objectives of the National Action Plan to Conserve Coral Reefs (U.S. Coral Reef Task Force 2000) with priorities and actions that are relevant for particular areas. The LAS are locally driven, short-range roadmaps for collaborative and cooperative efforts among federal, state, territory, and non-governmental partners to identify and implement priority projects that reduce key threats to valuable coral reef ecosystems in each region. Together, the LAS from the seven U.S. coral jurisdictions (American Samoa, the Commonwealth of the Northern Mariana Islands (CNMI), Florida, Guam, Hawaii, Puerto Rico, and the U.S. Virgin Islands) have identified projects to address five priority threats to coral reef ecosystems: land-based sources of pollution; overfishing; recreational overuse and misuse; lack of public awareness; and climate change, coral bleaching, and disease. Hawaii and Guam are the first jurisdictions to complete a LAS for coral disease and bleaching. There is a key need for other U.S. Pacific jurisdictions to create LAS that identify key activities, partners and funding needed to tackle coral disease-related issues throughout the region.

[1] The US Coral Reef Task Force National Action Plan was the first national blueprint for US action to address the loss and degradation of US and international coral reef ecosystems. Based on input from government and non-government organizations, scientists, resource managers and other. http://coralreef.gov/

- **Unify the coral disease research community with emphasis on efforts to bring together the World Bank Coral Disease Working Group (DWG) and the CDHC**.

The World Bank Coral Disease Working Group (DWG) has identified a number of needs and activities to address coral diseases globally, many of which overlap with priorities outlined in the CDHC National Research Plan (Woodley et al. 2003). For example, both groups recognize the need for standardized methodologies, nomenclature and diagnostics to improve the comparability of coral disease reports across jurisdictions and among different researchers. One of the most cost-effective ways to address gaps in knowledge and to facilitate the development of tools, technologies and informational products that can help resource managers respond to disease is through an enhanced collaborative partnership between the CDHC and DWG. This is a key step to help advance the field of coral disease research and ensure accurate and comparable results of coral disease research efforts.

- **Develop web-accessible database and informational resources for Pacific coral diseases**

In the coral reef arena there is often a period of several years between conducting a research project and publication of its findings, with limited communication of pertinent results to the management community by researchers. Furthermore, these findings are most often published in peer-reviewed journals that may be highly technical in nature, with inclusion of few management options in response to the findings. Managers need to have information presented in a manner that will enhance:

a. Recognition of the need for management actions in response to coral diseases;
b. Understanding the risks posed by diseases and risks associated with lack of management actions;
c. The ability to determine the state of their coral reefs, including baseline levels of diseases, changes in disease prevalence or linkages between disease occurrence and manageable human impacts; and
d. A comparison of what is happening in waters under their jurisdiction to surrounding areas, including status, trends, types and benefits of proposed and implemented management actions.

- **Develop a manager's guide for coral disease**

The MWG requested that the CDHC develop a guide that brings together the latest scientific knowledge and management experience to assist managers in responding effectively to coral disease outbreaks. This guide might be modeled after the Reef Manager's Guide to Coral Bleaching (Marshall and Schuttenberg 2006) and should include: a) survey/assessment protocols; b) outbreak response protocol; c) protocols for post-disease response actions; d) guidelines for research involving live organisms including safety and biohazard containment strategies; e) disease identification guides and standardized nomenclature and diagnostics; f) possible management responses to reduce the occurrence of disease and control spread; and g) resource materials for managers.

Strategic Objective D.1: Enhance the state of knowledge of coral diseases.

Recommendation D.1.1: Conduct baseline surveys on coral diseases throughout the region.

The accurate and thorough documentation of the type, prevalence and geographic distribution of diseases currently present in each jurisdiction is fundamental to attempting to effectively manage these diseases. As a first step, baseline surveys are needed at relevant spatial scales (e.g., different depths, habitats, reef types, and at varying distances from land within each jurisdiction) to identify what diseases are present now, how common they are and what coral species and/or other marine organisms are being affected. These surveys could be easily incorporated into existing survey efforts that examine the community structure and cover of corals, other benthic invertebrates, commercially and ecologically important mobile invertebrates (e.g., lobsters and crabs), and coral reef dependent fishes.

At minimum, efforts to characterize the baseline prevalence of diseases should include parameters that address:

- Coral species diversity at lowest possible taxonomic resolution (e.g., genus or preferably species level data)
- Coral community structure including size class and other population parameters
- Coral cover and colony condition
- Abundance of diseases and presence of possible disease vectors (e.g., gastropods, crown of thorns, fireworms)

In order to collect comparable data, the group recommended that coral disease surveys be conducted using standardized methodologies, disease nomenclature and forms.

Strategic Objective D.2: Develop and implement proactive management strategies.

Recommendation D.2.1: Develop a disease monitoring program for Pacific reef areas or integrate disease into an existing monitoring program.

Long-term monitoring of coral disease using standardized techniques is essential to detect and assess changes in disease prevalence, types, and organisms affected and to provide regular, up-to-date information to managers. Most areas in the Pacific with established monitoring programs do not include disease monitoring in their protocols. Furthermore, in areas where disease monitoring is occurring, survey approaches are highly variable, allowing only limited comparisons between programs and jurisdictions. The development of a disease monitoring program capable of assessing changes and trends in reef ecosystem health is recommended. This program should be appropriate for the specific reef areas of each jurisdiction and record comprehensive data exceeding current efforts at presence/absence of disease. It is further recommended that the effort be integrated into

existing monitoring programs whenever possible. Regular long-term monitoring, that includes disease surveys along with surveillance of high priority environmental stressors, can potentially be used strategically to identify and address emerging threats to specific areas (anthropogenic and natural threats).

The MWG identified some of the basic information that should be included in the disease monitoring program and recommended four documents that could serve as a starting point for the development of an integrated, Pacific-wide disease assessment and surveillance effort:

- IOC/UNESCO Coral Reef Targeted Research & Capacity Building for Management (CRTR) Program; Coral Disease Working Group assessment protocol
- A Reef Manager's Guide to Coral Bleaching (Marshall and Schuttenberg 2006)
- CDHC's Field Manual for Investigating Coral Disease Outbreaks (Woodley et al. 2008)
- Priorities for Effective Management of Coral Diseases (Bruckner 2002)

Recommendation D.2.2: Identify potential stressors that may influence susceptibility or resistance to disease and the potential to recover following disease outbreaks

Ecosystem condition, including biological attributes such as coral cover, condition and biodiversity, other ecosystem parameters (e.g., abundance diversity and structure of associated fish and invertebrate communities), and environmental attributes such as water quality, influence the resistance of corals and resilience of coral reef ecosystems. Variations in the local environments, including unusual exposure to heat stress, excessive sedimentation and nutrient loading, can play an important role in triggering coral disease outbreaks by increasing the susceptibility of corals to disease and potentially increasing the virulence of coral pathogens. Environmental stressors, along with other factors such as connectivity can also influence the ability of corals to recover from disease as well as the ability of degraded reef ecosystems to recover through recruitment. *While managers can do little to address increasing sea water temperatures and other stressors associated with global climate change, it is possible to manage and mitigate local or regional human impacts such as unsustainable removal of keystone species (through fishing and other activities), excessive input of pollutants and sediments, boat anchoring and other physical impacts to reef ecosystems, and marine pollution associated with recreational and commercial vessels.*

There is a growing body of evidence linking environmental stressors to coral disease outbreaks. However, few programs are conducting detailed monitoring of water quality in concert with disease studies, and few attempts have been made to tease out relationships between specific stressors and occurrence of disease, or the threshold of these stressors that will trigger a change in the health of corals and/or manifestation of disease signs. Through concurrent water quality monitoring, it may be possible to statistically compare disease abundance at single time points with the concentrations of specific stressors, as well as relationships between changes in input of stressors (e.g., during periods of high rainfall vs low rainfall) and the incidence of disease. For sites

known to be affected by specific contaminants or environmental stressors, it may also be possible to identify specific physiological/biochemical responses of the coral host that can be used as an indicator for that parameter. The MWG proposed a number of actions that could help to elucidate the responses of corals to various stressors and ultimately identify those stressors that can be managed to reduce disease occurrence:

- Identify stressors in specific area(s) of concern (i.e., water quality: content of nutrients, suspended sediments, agricultural or industrial chemicals, pharmaceuticals, secondary petroleum products, temperature, recreational uses) and characterize their effect and impact on corals and coral reef ecosystems. As a first step to identify possible stressors that may exacerbate diseases, monitoring programs could be established along a gradient including reefs adjacent to a known impact (e.g., adjacent to a sewage outfall) and sites varying distances upstream and downstream. If adverse effects were detected, this could be supplemented with more detailed studies on responses of individual corals to those stressors (i.e., bioaccumulations targeted biomarkers analyses and ecotoxicological assays).
- Identify site-specific stressors of major concern and determine "threshold for action" based on clearly defined acceptable/unacceptable percent change.
  - Metrics or parameters should be selected that are appropriate for the local area and acceptable/unacceptable percent change should be defined.
  - Natural fluctuations should be considered when selecting metrics.
- Scientists and managers should be encouraged to work together in developing general guidelines to reduce specific stressors of concern. A guidebook for "best management practices" (BMP) for addressing key environmental stressors should be created and encompass alternative management practices such as limiting development to specific low-impact places, reducing recreation in sensitive areas, and offering other protocols for addressing specific problems in a given area. In areas where these BMP guidelines or manuals already exist, scientists and managers should work to reference, communicate and apply this information in management activities.

Recommendation D.2.3: Develop disease education and outreach materials and incorporate these as components of existing educational programs (knowledge, attitude, behavior).

Most communities know that their coral reef resources are steadily being depleted but often they do not understand why. Local citizens in the Pacific Region are generally unaware of the presence of coral disease and therefore the potential impacts that disease could have on the coral ecosystem. This lack of understanding poses a challenge to the coral disease research community in that they may have difficulty in convincing the public, politicians and managers that they should care about coral diseases and consider preventative actions to address coral diseases and their impacts.

Educational efforts should include some very basic messages about coral disease and why they should care about disease, with a strong emphasis on encouraging stakeholders to take actions designed to improve overall coral ecosystem health. Because governments

usually do not have sufficient resources to enforce regulations effectively, education and awareness programs should target all stakeholders, emphasizing the need for local citizens to take ownership of the resources, including steps to address harmful activities they are responsible for, and to promote community based management. An essential component of education is actual participation, including involvement in assessing and monitoring status and trends of coral reefs and diseases.

Four very basic messages for local citizens and tourists were identified by the working group:

- **Diseases can and do kill coral reefs. Death of these keystone species has caused major shifts in community structure in some locations which results in losses of valuable ecosystem function and services.**
- **Corals are living animals that often have algal symbionts. They are susceptible to disease, as all animals are, and therefore can get sick and die.**
- **Exposure to stressors can make corals more vulnerable to disease; the stressors include physical damage, land-based pollution and overfishing. Many of these stressors can be reduced or eliminated through adoption of best management practices.**
- **Disease can kill reef organisms, including corals, and in some locations has caused major shifts in community structure and concurrent losses of ecosystem function.**

Additionally, specific educational materials should be developed for targeted agency administrators, policy-makers, managers and legislators that:

- Improve their general understanding of coral biology and factors as well as consequences associated with declining health of corals
- Provide detailed information on:
  - Interconnected relationships between corals and manageable environmental stressors,
  - Effects of these stressors on coral organisms and reef ecosystems,
  - Importance of understanding when and why changes to reefs and ecosystems are occurring, and
  - Rationale for taking action before visible signs of disease appear (e.g., tissue loss).
- Identify consequences of non-action in addressing disease situations, including economic ramifications of coral mortality and reef degradation.
- Communicate lessons learned from other reef areas – including regulations, legislation issues, and management responses that helped mitigate disease and/or improve the resistance and resilience of corals and associated organisms.
- Encourage local stakeholder advocacy to decision-makers.

Recommendation D.2.4: Identify management actions needed to reduce other stressors that may make corals more vulnerable to disease

A variety of natural and anthropogenic factors place substantial stress on reef building corals long before any visible signs of disease appear. These can include:

a. Contaminants and pollutants associated with degraded water quality that directly affect the growth, reproduction and ability of the coral to resist pathogens, such as certain chemical contaminants;
b. Excessive growth of macroalgae and cyanobacteria that affect the long term survival of the coral and future recruitment potential (due to top-down factors such as loss of key herbivores or bottom up factors such as increased nutrients);
c. Injuries to corals caused by physical impacts associated with ship groundings, anchoring and diver contact that provide an entry point for a pathogen;
d. Population explosions of coral predators such as crown of thorns sea stars and corallivorous snails which may serve as vectors for disease;
e. Increases in temperature and UV radiation as a result of climate change that affect the resistance of corals; and
f. Direct introduction of pathogens through run-off, discharge of human sewage, atmospheric deposition, subsequent transport to reef ecosystems via water circulation as well as ship traffic (hull microbial communities and bilge water).

Reefs are likely to be affected by several of these factors simultaneously, making it extremely difficult to tease out the importance of any specific factor(s) or a critical threshold for individual factors in terms of the relationship with coral disease. Furthermore some of these factors (e.g., temperature change) may be out of direct control by managers. However, management efforts geared towards reducing specific human impacts negatively affecting a given reef system (i.e., nutrient loading, sedimentation, overfishing, recreational and other human activities) may increase the local survivorship of corals and the resilience of reef ecosystems, thereby improve their resistance to infections and recovery following disease outbreaks. Therefore, it is crucial that initial management actions target efforts to reduce known land-based stressors by implementing best-management practices.

Recommendation D.2.5: Develop and implement training modules for coral disease and health surveillance methodologies, field and laboratory research, and potential management actions in the Pacific Islands

There is a critical need to develop and deliver training programs for multiple audiences covering a variety of topics related to coral health and disease in the Pacific Region. Effective training programs are needed to identify various coral disease research methods and to promote adoption of standardized methodologies across the region to allow comparative analysis of data to reveal regional patterns and trends, and allow comparisons among locations affected by different stressors. The WG suggested that the CDHC lead these efforts with assistance from the U.S. All-Islands Coral Reef Coordinating Committee[2].

[2] The US All Islands Coordinating Committee is a collaboration of marine resource managers working together with federal agencies to strengthen the conservation and protection of coral reef ecosystems in the United States. http://allislandscorals.org/

Key training areas identified by the WG include:

a. Basic survey methodologies for coral disease;
b. Disease outbreak incidence response protocols;
c. Disease identification and standardized nomenclature (The workgroup suggests creating "disease-cards" that depict and describe distinctive disease signs, and diagnostic criteria to distinguish these signs, along with general information on coral species that are typically affected, reef areas where the disease may be observed, and how/where to report disease sightings);
d. Advanced curriculum on coral disease-related topics with emphasis on field and laboratory investigation, including surveillance and sampling procedures, coral histology and physiology studies, disease epidemiology, and molecular methods including biomarker and toxicology studies;
e. Management practices and options for coral disease prevention and outbreak response. Coral and natural resource managers in the Pacific would tremendously benefit from training on recommended management alternatives and lessons learned from Caribbean experiences, including the "A Reef Manager's Guide to Coral Bleaching", and management efforts in Australia. Instruction and guidance is also needed to assist agency personnel with determining whether the existing infrastructure in each jurisdiction is appropriate for managing disease events. This would include an assessment and evaluation of existing agency mandates and legislation, current regulatory processes and enforcement capabilities.

Strategic Objective D.3: Develop a management program to respond to disease outbreaks.

Recommendation D.3.1: Evaluate local agency mandates and existing legislation, regulations and legal framework for addressing disease

Many aspects of coral reef research and monitoring, responses to unusual events or emerging issues, and proactive management actions can be delayed or obstructed due to lack of existing authorities to conduct an activity, complications with a permit process or policy documents with unclear provisions for disease-related activities. Existing mechanisms for permitting and implementing various coral reef activities, especially those directed at coral disease research and management should be evaluated to determine whether they are adequate and allow timely implementation of actions. Managers can participate by assisting responders with specific permitting processes and permissions and regulatory responsibilities that are required to allow rapid responses in the event of disease outbreaks. If existing permits or policies are insufficient, action can be taken to work with administrators, legislators, and local authorities to establish authority for timely and thorough responses to a disease outbreak to allow experts to conduct surveillance efforts, collect and transport appropriate specimens quickly, and take appropriate emergency response measures. This may include the establishment of a Memorandum of Agreement between responsible agencies.

Recommendation D.3.2: Develop local and regional infrastructure to respond to disease outbreaks and unusual mortality events.

While the MWG recognized the need for and benefits of proactive management responses for diseases, they also acknowledged the likelihood of future disease outbreaks and the need to obtain timely information on the occurrence, extent, cause and impact of these events. The MWG also recognized the advantages of an organized, systematic approach to create diagnostic case definitions of the disease (e.g., What is it?), identify risk factors (Where did it come from? How is it spreading?), formulate possible measures to control and manage the outbreak, and predict the consequences under various scenarios. Currently, one of the largest limitations in our ability to respond is a lack of appropriate infrastructure, including:

- People capable of identifying and reporting unusual outbreaks when they first occur;
- Standardized surveillance and sampling protocols;
- Trained response teams;
- Capacity (e.g., boats, supplies, diagnostic laboratories) to respond in an effective and timely manner; and
- Existing system for reporting observations.

The MWG was supportive of the CDHC's proposed Incident Command System for responding to disease outbreaks. For this effort to be successful the MWG acknowledged the importance of outlining the desired response process, assigning roles and responsibilities, creating necessary response protocols, databases and communication mechanisms, and identifying gaps in permitting procedures and funding sources. The MWG recommended the following infrastructure elements:

a. Develop a local response protocol following the CDHC basic response framework;
b. Create local **"Eyes of the Reef"** initiatives including public education on various aspects of response such as disease identification, reporting, and volunteer monitoring (divers, reef check, NGO's, academics);
c. Create a response team with defined roles and responsibilities and identify training, equipment and permissions needed to be a responder;
d. Define the communication structure between the response team and coral reef managers;
e. Set up a central system and/or database for reporting observations and data;
f. Explore mechanisms for setting up permits for emergency response and mitigation to include standard permits for defined coral disease responses; and
g. Identify additional funding sources needed for a fully functional response system (i.e., response activities, communications, data analysis).

Additionally, based on a cursory evaluation of agency mandates, legislation and regulatory processes there may be a need to revise or create clear policy guidelines that will allow for immediate decision making and response activities in the event of a disease incident. We recommend that proactive steps be taken, if needed, to develop appropriate policy statements and Memorandums of Agreement between local agencies that would

allow immediate resource-based decision making to implement timely response activities and protective measures, such as temporary closures or activity restrictions in critical areas.

The CDHC model response protocol (Woodley et al. 2008) follows a tiered approach for responding which allows the level of response to be determined based on an assessment interview of the original observer of the disease outbreak (Level I response).

If a Level II or Level III response is deemed appropriate, the MWG identified the need for timely reporting of findings to managers to allow implementation of possible management actions in a timely manner. This includes a recognized need for follow-up surveys to determine impacts on affected corals, fisheries resources and the ecosystem in general. The MWG identified the importance of adopting standardized monitoring protocols and ensuring sufficient human and financial capacity to support post-incident monitoring before the disease event occurs. Moreover, a communication plan should be outlined to ensure that critical post-incident monitoring information reaches coral managers in a timely manner so suitable management decisions can be made and corrective or protective actions can be implemented.

Recommendation D.3.3: Identify ecological and economic cost and benefits of various management actions in response to disease outbreaks.

In response to a disease outbreak or incident specific management actions may be called for to control the potential exchange of disease vectors to other reef areas during outbreaks, to minimize long-term damage to the ecosystem and to allow for enhanced recovery of the affected reef.

Recommended actions to be considered by managers to enhance reef recovery:

- **Modify MPA boundaries or zoning**
- **Temporary closures, activity restrictions**
- **Containment of affected area and adjacent reef area**
- **Decrease or limit adjacent land use and development for specified times**
- **Use the permit process to control field activities related to coral disease**

Strategic Objective D.4: Identify priority coral disease and health research needs to aid in management.

The MWG also recognized the need for enhanced collaboration with and support for local scientists conducting coral reef research with emphasis on encouraging activities that will lead to increased understanding of coral disease and disease processes.

Recommendation D.4.1: Create a CDHC Pacific Research Plan that emphasizes regional disease priorities.

The MWG identified the need for additional meetings involving both scientists and managers to:

- More thoroughly review existing knowledge of coral diseases in the Pacific;
- Identify specific critical gaps hindering effective responses to disease outbreaks and management actions to mitigate diseases; and
- Identify a strategy to address these information gaps.

As a starting point, the Strategic Objectives originally laid out in the 2003 CDHC National Research Plan (Woodley et al. 2003) should be revisited and reviewed to identify outstanding research priorities that apply nation-wide and identify an approach to fill these gaps. Eight regional research needs were identified by the MWG which will ultimately provide essential information to make better management decisions for preventing, responding to and managing disease outbreaks in the Pacific include:

- Conduct economic valuations of coral reef ecosystems and associated resources where valuations have not been completed yet.
- Explore the potential to incorporate disease factors into modeling.
- Investigate how Pacific island cultural and social practices enhance or detract from management efforts and how are they can be better incorporated into management strategies.
- Conduct targeted disease transmission experiments under controlled conditions.
- Develop research projects to explore potential treatments and possible cascading effects of disease.
- Conduct research aimed at developing feasible and effective recommendations for action and mitigation strategies and establishing thresholds for action.
- Establish links between ecosystem health and organism health in coral reef ecosystems.
- Encourage interdisciplinary research following a watershed approach to link land-based stressors to coral disease.

Strategic Objective D.5: Environmental and Human Health Safety Issues.

Inherent in disease studies is some measure of risk to human and environmental health. Those working with diseased organisms and putative infectious agents must recognize that a potential exists for humans to become infected (though most likely a small risk) or they and/or their equipment may serve as inadvertent vectors to other corals and to other

locations. Just because coral disease occurs in an aquatic environment, does not make it completely safe to study and without risk. There are common practices that medical science adheres to when dealing with potentially infectious disease outbreaks (of known or unknown origin) or toxic events and are applicable, regardless of whether the focus is human disease or coral disease. Preventative containment measures are a logical option to mediate risk. Containment measures are relatively easy to apply in the aquatic environment and therefore should be included as part of each response activity particularly to limit the possible spread of infectious agents. The three recommendations listed below target important areas where managers can assist in coral disease management.

Recommendation D.5.1: Create an awareness of the dangers of outplanting aquaria raised corals.

There are significant issues related to placing captive animals into the wild. If not closely scrutinized catastrophic consequences can occur, even with the best of intentions. Though restoration efforts are important considerations for reef management in certain locations, it is also important to conduct a hazards analysis to avoid bringing modified organisms (through captive conditions) into the wild that may present an unacceptable risk to other wild species within the ecosystem. To fully explore the benefits and dangers will require a focus group to evaluate this issue and provide recommendations.

Recommendation D.5.2: Develop requirements for containment measures needed for conducting disease transmission studies

Bio-safety and bio-containment are critical issues when conducting disease research. Veterinary research facilities for aquatic and terrestrial animals have rigorous guidelines for handling sick and diseased organisms, as do domestic (e.g., U.S. Department of Agriculture) and international groups (OIE, World Organization for Animal Health). The guidelines and practices of these groups should function as a role model for adopting similar guidelines tailored for coral disease research. Development of these guidelines will require a team of coral disease researchers to interface with experts and practitioners of animal health, bio-security and bio-containment to develop methods appropriate for tropical marine systems.

Recommendation D.5.3: Develop recommended methods for decontamination of dive gear.

This is a specific project that can have wide spread management applications and is an obvious follow on to Recommendation 5.1. Determining the risk associated with dive gear in transmitting disease to either humans or marine organisms is vital to provide a basis for risk management options. Once the risk level is determined it is important to provide a means of decontamination that is safe and effective for both the user and the environment. Results of such a focused study can provide an unambiguous tool to help manage coral disease on a local level.

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E. 'WHITE PAPERS'

The opening day of the workshop focused on presentations derived from 14 position papers, to provide context and concepts for the break-out group discussions. The presentations, included in this section, covered key topics:

- *What do we currently know about coral diseases in the Pacific?*
- *What lessons have we learned from Caribbean disease outbreaks?*
- *Diagnostic methods, systems biology and leveraging post-genomic technologies*
- *Emerging diseases, disease outbreak investigations and ecological epidemiology*
- *How to integrate science with social, economic and political values?*

I. INTRODUCTION—SETTING THE STAGE

CORAL DISEASE AND HEALTH CONSORTIUM (CDHC)

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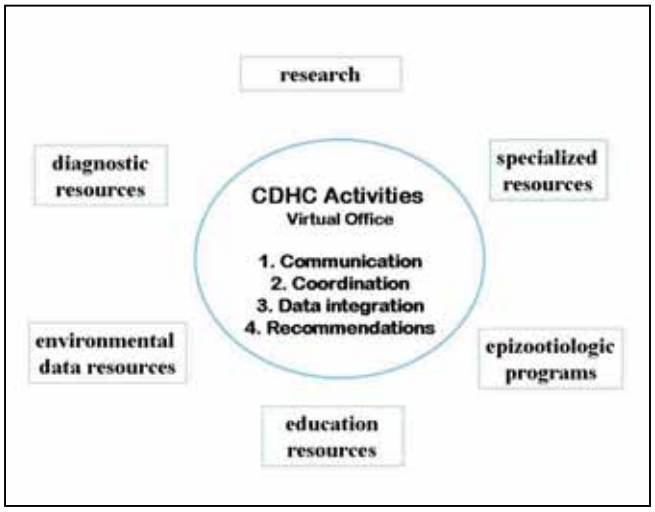
CDHC - VISION

"To understand and address the effects of natural and anthropogenic stressors on corals in order to contribute to the preservation and protection of coral reef ecosystems."

CDHC - WHO ARE WE?

The Coral Disease and Health Consortium (CDHC) was created in 2002, in response to the U.S. Coral Reef Task Force's (USCRTF) National Action Plan to Conserve Coral Reefs (United States Coral Reef Task Force 2000). Our goal is to provide coastal and ocean managers with scientific understanding and tools to help protect healthy coral reef ecosystems and restore degraded ones. The CDHC is a network of field and laboratory scientists, coral reef managers, and agency representatives devoted to understanding coral health and disease. It is extensive, highly collaborative, and completely voluntary. Currently over **150 partners,** from federal agencies, EPA, DOI, NOAA along with academia, non-profit and industry, contribute their time and expertise to the CDHC, while the organizational infrastructure is supported by the congressionally funded NOAA's Coral Reef Conservation Program.



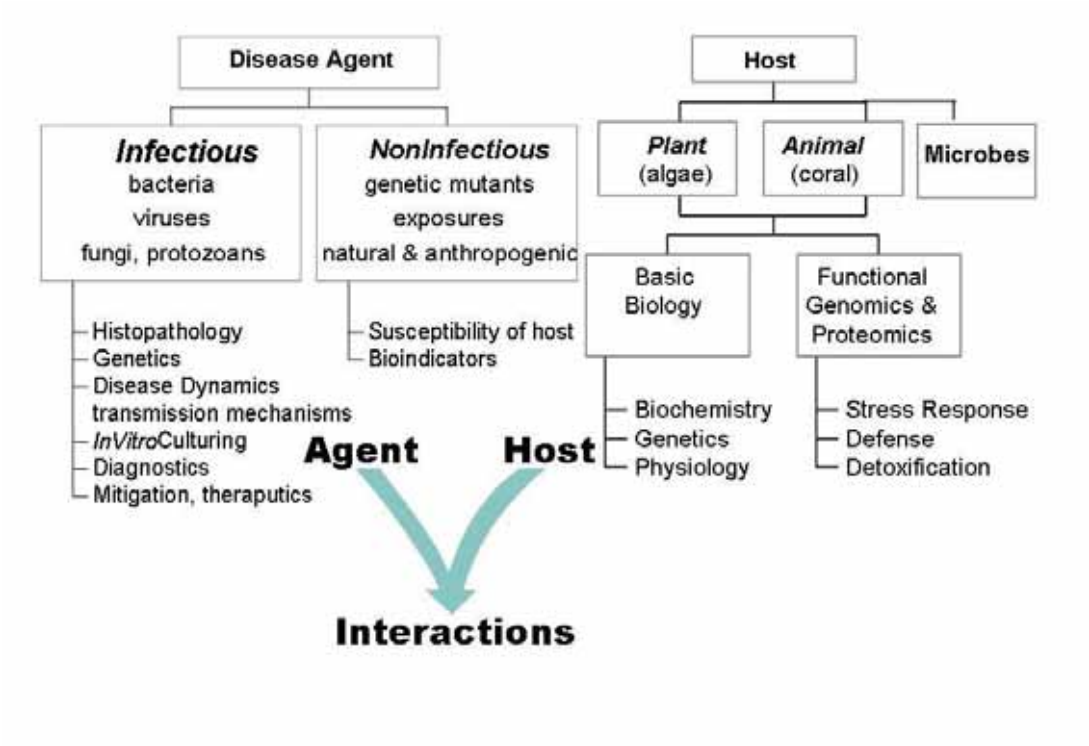
The commitment to share information, ideas, and common goals led to the development of a national research plan, *Coral Disease and Health: A National Research Plan* (Woodley et al. 2003), that has inspired many to seek funding and devote new resources to the study and amelioration of coral disease. Members of the CDHC come from a variety of backgrounds, but all have a common commitment to share information, ideas, and common goals to further the study of coral

disease and in so doing identify ways to better manage coral disease. We encourage participation of anyone sharing our vision and goals.

CDHC - WHY?

Recent reviews have documented an explosion in the incidence of disease, particularly in the Caribbean. The first disease report occurred in 1965 related to skeletal anomalies (Squires 1965), with the next report coming 8 years later by Antonius (1973), from these reports through the early 1990s, only four diseases had been recognized: skeletal anomalies, Black band, White plague Type I, and Shutdown reaction (Sutherland et al. 2004). Since the early 1990s, monitoring programs in the Florida Keys have documented a sharp increase **in the number** and **prevalence** (the ratio, for a given time period of the number of occurrences of a disease or event to the number of units at risk in the population) of coral diseases. Reports from the Indo-Pacific suggest an emerging crisis in coral disease as monitoring efforts are able to explore new areas.

The picture of coral disease has expanded from the simple perception of an infectious disease agent to a plethora of possible interactions with a variety of possible agents attacking not just the coral animal, but an intricate group of organisms consisting of plant, animal and microbial associates. The complexity of this growing disease problem made it



more difficult to design management regimes and increased significantly the need for cross-disciplinary tools to combat the problem (see diagram below).

Realizing these were complex and complicated issues, we recognized that our lack of understanding of the underlying mechanisms of coral pathologies was inhibiting our ability to manage the growing number of coral health problems. Also, to improve our ability to identify the factors responsible for coral health decline and increased disease incidence would require embracing a *new paradigm of scientific investigation* that incorporates new methods and new technologies able to help elucidate mechanisms that link cause and effect relationships so the field could move from just descriptive science into mechanistic science.

There was an obvious need to unify the coral disease community, build scientific skills and capacity, and provide standardization in investigative nomenclature, methodologies and technologies in order to competently communicate and interact with other main stream disease fields (e.g., pathology, cell biology, physiology, infectious disease, toxicology, medicine). In response, the CDHC was organized in 2002 when 50 experts from various disciplines and perspectives from science to management, met and developed what we now refer to as the *Coral Disease and Health: A National Research Plan* (Woodley et al. 2003). This document provided an integrated roadmap that began tying these ideas together. This document outlined gaps in our knowledge and recommended research directions needed to support this new paradigm. Four major themes with accompanying strategic objectives were identified: **Biology (6), Disease Identification and Disease Investigation (4), Disease Diagnostics (5) and Environmental Factors Affecting Susceptibility and Infectivity (11).** The 26 recommendations encompassed 9 topic areas: **Nomenclature, Model System(s), Field Assessment of Coral Reef Condition, Microbiology, Toxicology, Histopathology, Molecular, Bioinformatics, and Advanced Education and Outreach.**

CDHC: WHAT ARE WE DOING?

Research

Information is limited on the physiological parameters that define healthy coral and even less on coral pathology. Our challenge is to apply advanced technologies in functional genomics, proteomics, toxicology, and systems biology to expand our knowledge to understand and recognize coral health and elucidate disease dynamics. The knowledge gained from this research approach is positioning us to move aggressively toward characterizing the processes that control ecological connectivity among reefs and discover critical control points for management strategies. The first step is to establish and make available tools that can support discovery and applied research. For example, CDHC efforts have helped establish transcriptomic resources from expressed sequence tag (EST) cDNA projects with over 30,000 coral EST sequences publically available from five species: *Montastraea annularis, Oculina varicosa*, *Porites astreoides, Acropora palmata and A. millipora.* There are also over 28,000 ribosomal gene sequences cloned from coral-associated bacteria available to assist in microbial diversity

and pathogen research efforts. This type of information is vital and basic to developing an understanding for how an organism responds to its environment, is key to developing diagnostic tools to assess coral health and lays the foundation for identifying critical control points and viable management options.

Diagnostic Resources

There is limited application of medical/veterinary knowledge or protocols to the study of coral health and disease, resulting in ambiguous and often misleading communication of findings. Compounded by inadequate diagnostic tools and insufficient application of diagnostic procedures, the challenge is to develop standardized procedures based on medical principles that clearly define terminology, pathology and diagnostic criteria.

Education

Experts in coral biology, pathology and veterinary science are developing resources and web-enabled tools for use in recognizing gross signs of disease and in clinical diagnostic pathology as well as developing case definitions for selected coral syndromes. The web-tool will be used to guide investigators in the diagnostic process. Additional modules are planned that will include virtual slide technologies for distance learning coral histology and histopathology, consultation with experts on disease cases, and continuing education through regular 'grand round' web meetings.

Diagnostic Tools

Consortium members have achieved significant advances in diagnostic assay development that assist researchers in identifying coral stressors. Examples of new techniques include:

- DNA probe for the White Plague agent – Dr. Laurie Richardson, Florida Atlantic Univ.
- DNA sequence analysis for the White Pox agent *Serratia marcescens* (newly designated 'White Pox Serratiosis' when the presence of *S. marcescens* is confirmed) - Dr. Kathryn Sutherland, Rollins College, Winter Park FL
- Coral immuno-competence (IMCOMP) assay to assess the presence of antimicrobial agents within coral tissue by using a modified bacterial viability assay – Dr. Craig Downs, Haereticus Environmental Laboratory
- PCR-screening test for recognized pathogens – Dr. Shawn Polson, Univ. Delaware & NOAA NOS Charleston, SC
- DNA Abasic site lesions – NOAA NOS Charleston, SC
- Various toxicity tests are being adapted or modified to address development, mutagenesis, and cellular pathologies associated with toxicant exposures.

Specialized Resources

Several specialized resources that help build capacity and provide outreach and educational opportunities are being made available by members of the CDHC. These include:

- International Registry of Coral Pathology (IRCP) supported by NOAA, Oxford, MD, is a research tool and resource of voucher materials for the coral research community. Submission, holdings and acquisitions are located at http://www.chbr.noaa.gov/InternationalRegistry.html. For more information contact Dr. Shawn McLaughlin, shawn.mclaughlin@noaa.gov
- Annotated cnidarian bibliography containing >5000 references and abstracts available as an ENDNOTE™ library or on CD, is supported by a complete set of reprints and is accessible on an individual basis on site in Charleston, SC. Contact Dr. Sylvia Galloway, sylvia.galloway@noaa.gov for more information.
- CDHC Website and Listserve – Supported by NOAA's Coral Health and Monitoring Program at the Atlantic Oceanographic and Meteorological Laboratory in Miami, FL. http://coral.aoml.noaa.gov/mailman/listinfo/ http://www.coral.noaa.gov/coral_disease/

CDHC – VISION FOR ACTION - WHY ARE WE HERE AT THIS MEETING?

The overarching goal for us is to "**Promote the effective detection, identification and management of coral reef diseases".** To do this a plan of action is needed that will ***'Chart a course for coral health and disease in the Pacific and Indo-Pacific'.*** We have convened this meeting to:

- Synthesize the state of knowledge of Pacific coral diseases
- Develop a strategic plan to:
- Identify research needs to help understand etiologies, epidemiology and ecology of coral diseases
- Identify innovative strategies for disease management on coral reefs
- Identify novel strategies to engage public and political sectors in partnering with us to combat the spread of coral disease

Disease

"any impairment that interferes with or modifies the performance of normal functions, including responses to environmental factors such as nutrition, toxicants, and climate; infectious agents; inherent or congenital defects, or combinations of these factors" Wobeser 1981.

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STUDYING CORAL DISEASES; UNDERSTANDING THE NORM

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Coral disease incidence has been on the rise for the past thirty years. Within that time frame approximately 30 new syndromes and diseases have been identified. To date, only few diseases or syndromes have been characterized in terms of causality and even fewer have been characterized in terms of the physiological effect on the coral.

The coral holobiont may be considered a "super-organism" composed of the coral host, its algal symbionts, and accompanying microorganisms. Since disease is defined as "any deviance from normal physiological function of an organism" it then becomes no trivial task to ascertain the physiological norm of this complex "super-organism".

In corals, disease signs and symptoms are usually classified by superficial signs, such as color change, tissue loss pattern, or changes in gross colony architecture. This simplistic classification is due to the inadequacy of information available describing basic biological and physiological processes that could provide the baseline for comparison. For example, information pertaining to cellular processes responsible for coral calcification and growth is still rudimentary. This is also true for information pertaining to biochemical regulation of coral reproduction. Similarly though mechanisms of tissue repair and regeneration have been studied at the organismal level, only recently have they begun to be assessed at cellular and molecular levels. Since these are all vital biological processes without which corals could not survive, understanding them is crucial to our perception of normal coral physiology. In turn understanding the norm will allow for a proper diagnosis of deviation from it.

The use of "the diagnostic method" borrowed from the world of medicine may prove useful in elucidating disease processes. This method incorporates performing a "clinical" examination that includes historical and current information about the coral in order to determine its state and provide a diagnostic interpretation. The purpose of this examination is to detect overt changes in carefully chosen assessment end-points with known reference values. This will only really be possible though, once basic reference values have been established. The elucidation of physiological regulatory pathways will improve our understanding of how the coral holobiont responds to stress and will assist in formulating standards for proof of cause-and-effect relations and provide information on how environmental change could affect host-pathogen relations and immune defenses.

It is clear that assessment of coral health should be not be carried out only on a single level of biological organization, but should be evaluated across a hierarchy of organization including molecular, biochemical, cellular and tissue-level and whole organism phenomena, and include population metrics as well. Once these parameters are evaluated as a whole, we will be much better equipped to properly diagnose and mitigate coral disease.

II. GLOBAL PERSPECTIVE OF CORAL DISEASE

THE GLOBAL PERSPECTIVE OF INCIDENCE AND PREVALENCE OF CORAL DISEASES

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ABSTRACT

Diseases occur globally in most coral reef habitats whether near human population centers or remotely offshore. They generally affect a low proportion of the susceptible species, although localized outbreaks have produced significant mortalities to scleractinian corals, gorgonians, sea urchins, reef fish, sponges, algae and other coral reef organisms (Peters, 1993; Harvell et al., 1999; Williams and Bunkley-Williams, 2000). There are now over 30 named diseases in the Caribbean basin affecting 45 zooxanthellate scleractinian corals, three hydrozoan corals, ten octocorals, two zoanthids, nine sponges and two crustose coralline algae (Green and Bruckner, 2000; Weil et al., 2006), and at least seven major diseases from the IndoPacific, along with about 30 additional conditions that are associated with compromised health in scleractinian corals. While an apparently unprecedented increase in disease occurred in the Caribbean since the 1980s, much less is known about the status of disease in the IndoPacific and Red Sea. Surveys over the last decade in Australia, Palau, East Africa, the Philippines and other locations have revealed new diseases, suggesting a rapid emergence of disease, or at least a realization of their presence, throughout the Indo-Pacific. Between 1972 and 2005 coral diseases were reported on 39 genera and 148 species worldwide, with observations in 63 countries. Although Pacific reefs have a higher diversity of reef-building corals than the Atlantic and harbor 92% of the world's coral reefs (Spalding and Greenfell, 1997), only 14% of the global observations of coral disease were from the Indo-Pacific during this period (Green and Bruckner, 2000, Sutherland et al., 2004, GCDD, 2007), and 58% of all coral disease records are for BBD, WBD and WP. The Caribbean has historically been referred to as a "hotspot" for disease, largely because of the rapid emergence, high prevalence, wide distribution, large numbers of host species, and virulence of diseases in this region.

There are eight major diseases (BBD, WP, WBD, YBD, DSD, WPX, ASP and tumours) that have been reported from throughout the western Atlantic along with another 32 conditions (including different "types" of the major diseases) that have been reported since 1972. WBD, BBD and WP were first reported from the Caribbean in the 1970s from a small number of countries, with observations expanding to new locations during the 1980s including reports of WBD from about half of the Caribbean nations. During this period, BBD and WP caused localized mortality, while WBD contributed to a

regional decline of *Acropora*. Reports of WBD decreased dramatically during the 1990s, and then increased again since 2000, with most reports on *A. cervicornis*. Reports of WP and BBD have also escalated since 1998, with recent observations from 24 countries. While low level chronic infections of BBD have been observed on the same reef for years to decades, WP prevalence has dramatically increased since 1998, with outbreaks occurring over an expanding range. Many other new diseases have been reported on western Atlantic reefs since the mid 1990s, including four (DSD, YBD, WPX and ASP) that are widely distributed and four (YBD, WPX, WP-II and ASP) that are causing substantial coral mortality. Close to 80% of all western Atlantic corals are affected by diseases (41 species of scleractinian corals, 8 gorgonians, 2 hydrozoans), with some corals (especially *M. annularis* complex) being susceptible to as many as 8 diseases and corals showing signs of 2-3 diseases at one time. During the 1970s-early 1990s acroporids were most severely impacted by disease, while massive and plating corals, and in particular the *M. annularis* complex, are being affected more severly today. WP is the most virulent disease and has the widest host range.

Coral diseases were first reported from the IndoPacific and Red Sea in the late 1970s. Most observations during the 1970s and 1980s were for BBD and WBD by a single researcher working in three countries (Philippines, Egypt and Saudi Arabia), along with additional reports of abnormal skeletal development (tumors). By 1994, diseases had only been reported from six countries, including several new conditions first observed on reefs in Australia. In the mid to late 1990s, several new diseases emerged (YBD, SEB, PUWS), but these and other diseases were restricted to a few countries. IndoPacific diseases appear to be exhibiting a rapid expansion in range and in the types of disease since 2000. This includes reports from new regions that were previously unaffected (South Africa, Solitary Islands), a higher percentage of reefs in certain locations (e.g., Great Barrier Reef Australia) with diseases, an increasing incidence of diseases, and an emergence of several new conditions (fungal disease, WS, BrBD, Pink Line). Fast growing corals in the family acroporidae and pocilloporidae in the IndoPacific are affected by the largest number of diseases and are observed with disease more frequently than all other species.

Introduction

Coral reefs have experienced unprecedented losses of live coral cover from anthropogenic and natural stressors during the last three decades (Byrant et al., 1998; Jackson, 2001; Pandolfi et al., 2003). Coral diseases are one of the major factors responsible for this decline, especially in the wider Caribbean (Harvell et al., 1999; Aronson et al., 2003; Gardner et al., 2003). The Caribbean has been referred to as a "hot spot" for coral diseases, due to the rapid spread, wide distribution, expanding host ranges, and increased virulence of these diseases (Rosenberg and Loya, 2004; Weil, 2006). In addition to black band disease (BBD), white plague (WP) and white band disease (WBD), which have persisted on Caribbean reefs since the 1970s, there has been a recent emergence of diseases with new types of pathologies and elevated rates of tissue mortality (Richardson and Aronson, 2002; Weil, 2004).

By the late 1990s, diseases had been observed on 102 coral species in 54 different nations, with 27 diseases reported from the Caribbean and 13 from the Indo-Pacific and Red Sea (Green and Bruckner, 1999). Over 66% of these reports were for BBD, WBD

and WP in the western Atlantic (Green and Bruckner, 2000; Sutherland et al., 2004). Although western Atlantic reefs exhibit a low diversity of reef-building corals relative to the IndoPacific, and they constitute only 8% of the world's coral reefs (Spalding and Grenfell, 1997), this region hosted a disproportionate number of diseases and affected corals (>80%) (Sutherland et al., 2004).

Although coral diseases were reported from the Indo-Pacific in the 1980s (Antonius, 1985), the vast majority of these observations were made by one researcher in the Red Sea and Gulf of Arabia (Antonius, 1985; 1987; 1988). Recent surveys conducted in Australia (Willis et al., 2004), western Indian Ocean (McClanahan, 2004), Philippines (Raymundo et al., 2004), and Red Sea (Loya et al., 2004) illustrate the widespread, global distribution of coral diseases. Through annual and semi-annual monitoring programs on the Great Barrier Reef Australia, the Philippines, and other locations, researchers are identifying coral diseases on a greater number of reefs and species, and disease incidence appears to have increased since the late 1990s, suggesting that diseases have become more prevalent in the IndoPacific over the last five years (Raymundo et al., 2003; Willis et al., 2004; Kaczmarsky, 2006). However, it is difficult to determine "baseline" levels of coral diseases, and conclusively state that diseases are increasing, as the proliferation of reports at least partially reflects an increased monitoring effort.

Increases in the types of diseases and their abundance and severity may be at least partially related to an overall deterioration of the marine environment due to human stressors (e.g., land-based pollutants), climate warming, and other changing environmental conditions (Harvell et al., 2002; Kuta and Richardson, 2002; Garrison et al., 2003; Kaczmarsky et al., 2005). Sediment, sewage, toxic chemicals and other pollutants may facilitate disease outbreaks by introducing opportunistic pathogens, increasing pathogen virulence, and reducing host resistance (Antonius, 1977, Ducklow and Mitchell, 1979; Peters, 1984; Peters, 1993). However, reefs removed from direct anthropogenic inputs are also being impacted by disease (Santavy and Peters, 1997; Weil, 2004; Bruckner and Bruckner, 2006) highlighting potential associations between disease and elevated temperatures, light levels and other manifestations of global climate change including coral bleaching. Corals live close to their thermal tolerance limits, and a 1-2°C increase in SST is sufficient to induce coral bleaching. Recent outbreaks of WP in the eastern Caribbean following the 2005 bleaching event (Miller et al., 2006; Weil et al., 2006), provide additional evidence that bleached corals have a higher susceptibility to other diseases.

The Global Coral Disease Database

To begin gathering more comprehensive data on the global distribution and abundance of coral diseases, and quantify relationships between coral disease and various environmental stressors, NOAA Fisheries worked with the United Nations Environmental Program's World Conservation Monitoring Centre (WCMC) to develop a **Global Coral Disease Database** (GCDD)[4] . The GCDD is a web-accessible GIS database that compiles records of disease observations and tracks their spread over time, by geo-referencing disease locations and plotting their occurrences onto WCMC coral reef

[4] http://development.unep-wcmc.org/GIS/Coraldis/index.cfm

distributional maps. The GCDD includes a new online mapping tool (prototype IMAPS tool) that enables users to search and plot data by disease name and year, with zoom capabilities and full information sheet for each line of data. For each disease, information can be obtained on its global and regional occurrence and abundance, affected locations (e.g., country, reef, latitude and longitude) and species, and any available site-specific data on prevalence, incidence, and extent of mortality. A summary of all *in situ* observations on the prevalence, range of species affected, global geographic distribution, and mortality for reported coral diseases up to 1999 (2076 records of coral disease from 155 references) are included in the first iteration of the GCDD. The second version of the GCDD includes over 7100 data points compiled from information available through December, 2005, including peer-reviewed literature, grey literature, regional monitoring data from AGRRA (Atlantic and Gulf Rapid Reef Assessment surveys conducted between 1998-2000 in 22 countries; Lang, 2003), CARICOMP (survey of 19 reef sites from 6 countries in the Caribbean; Weil, 2004), Reef Check, and other programs, and reports submitted by researchers. These datasets reflect wider spatial coverage of disease surveys, repeat surveys, and increases in the types of diseases and species affected.

Global diversity and distribution of coral diseases

The GCDD contains records for over 40 coral diseases from the western Atlantic, 28 from the IndoPacific and 5 from the Red Sea that were reported between 1972 and 2005 (Table 1-4). Five coral diseases [BBD, WBD, WP, red band disease (RBD) and shut down reaction (SDR)] were first observed in the western Atlantic 20-30 years ago and three of these (BBD, WBD, SDR) were also reported from the Red Sea and IndoPacific during the 1980s (Antonius, 1977, 1981, 1985). Five other diseases [WP type II, white pox (WPX), yellow band disease (YBD), dark spots disease (DSD) and Aspergillosis (ASP)] first emerged on Caribbean reefs in the 1990s; all of these diseases (with the possible exception of DSD), have caused significant localized mortality and they represent continuing major threats to western Atlantic coral reefs. More recently, five IndoPacific diseases [white syndrome (WS), YBD, fungal syndrome, Porites ulcerative white spot disease (PUWS)] are causing substantial localized mortality and the prevalence of two of these (WS and PUWS) appears to be increasing. In addition to diseases that are presumed to be caused by bacteria, fungi and cyanobacteria, several conditions with rapidly expanding ranges [skeletal eroding band (SEB) and brown band disease(BrBD)] are being caused by ciliates and one disease observed so far only in Hawaii results from infection by a trematode (*Plagioporus*). Skeletal anomalies (tumors, hyperplasia, neoplasia, calicoblastic epitheliomas) have been reported from the Atlantic, Pacific and Indian Oceans and the Red Sea since at least 1965 (Squires, 1965), but few data are available on prevalence or impact. Some conditions are visible only with microscopy (e.g., coccidian infections, nematopsis spores). Most of the other conditions have been observed infrequently or are confined to localized areas.

A lot of confusion has been created by many reports of new diseases over the last ten years. There are also at least 19 other diseases that have been assigned on the basis of a few or single observations. These include 1) conditions presumed to be caused by a pathogen but later shown to result from predation; 2) conditions that lack details on gross signs or photographic documentation, or evidence of coral tissue destruction; 3) terminology that has been used interchangeably to describe similar signs, such as the

various white syndromes; and 4) similar conditions identified in the Caribbean that have been split into two or more syndromes (e.g., "Type 1" and "Type II"), based on rates or patterns of disease spread or species affected. Because of the difficulty in verifying which "type" of disease is present based on single observations (e.g., initial signs of infection may look different than later stages and rates of spread may vary over the duration of the infection), many researchers do not differentiate between types, or they use a different name overall (e.g., "plague-like"). Examples from the Caribbean include 1) white plague type I, II, II (Richardson and Aronson, 2002); 2) WBD type I and II; 3) DSD type I, II, dark band syndrome, purple band syndrome and tissue necrosis (Weil, 2004); and 4) white pox, patchy necrosis and necrotic patch syndrome.

1. White syndromes

There is a proliferation of names for coral diseases that are characterized by white lesions with a sharp, distinct line between apparently healthy tissue and exposed skeleton and an absence of an obvious microbial community at the disease line. These have been separated based on the identification of variable features such as 1) a zone of bleached tissue that may or may not be present used to differentiate WBD type I from WBD-II, differences in the rates of tissue loss and patterns of spread in WP type I, WP-II and WP-III, or differences in affected species (WP versus WBD). Antonius (1977, 1981) and other colleagues reported WBD on acroporids and other massive and plating corals in the western Atlantic, as well as corals in the IndoPacific. Other researchers from the Caribbean report WBD on *Acropora* and refer to similar signs in other host species as WP (Dustan, 1977; Richardson et al., 1998).

Table 1. Diseases, syndromes, abnormal tissue conditions, and parasitic infestations of scleractinian corals and gorgonians on coral reefs in the tropical western Atlantic.

condition	geographic range	host species	source
Black band disease (BBD)	W. Atlantic, 25 countries	26 scleractinians, 1hydrozoan, 6 gorgonians: faviids, *Agaricia*, *Siderastrea, Meandrina*; *A. palmata*[2], *P. astreoides*, *P. porites*[3], *Madracis mirabilis*, *M. decactis*[4]	Antonius, 1972 [2]Garzon-Ferreira *et al.*. 2001; [3]G. Smith[4]Sutherland et al., 2004
White band disease (WBD)	Caribbean, 27 countries	*A. palmata, A. cervicornis*	[1]Gladfelter et al., 1977
WBD type II	Bahamas, Puerto Rico	*A. cervicornis*	Richie and Smith, 1995; Weil, 2006
White pox (WPX)	Bahamas, Florida, Cuba Puerto Rico, Jamaica	*A. palmata* Synonyms: Patchy necrosis[2] Necrotic patch syndrome[3]	Porter, 1996 Patterson et al., 2002; Bruckner and Bruckner, 1997[2] Jordan-Dahlgren and Rodríguez-Martinez, 2004[3]
Plague (WP)	20 countries	31 species	GCDD records
WP type I	Florida and Bahamas	*Mycetophyllia*, *Montastraea, Colpophyllia, Agaricia*, *Mussa, Stephanocoenia, Porites*; 12 species	Dustan, 1977; 1984
WP type II	Bermuda, Bonaire, Colombia, Florida, Jamaica, Mexico, USVI, Puerto Rico, Venezuela	*D. stokesi* and 17 other species[1] 41 species[2]	Richardson et al., 1998 [2]Weil et al., 2006
WP type III	Florida	large corals (*M. faveolata, C. natans*)	Richardson, 2000
Yellow band disease (YBD)	12 countries	*M. annularis* complex; *M. cavernosa; C. natans* and other faviids *; P. astreoides; A. agaricites*	Reeves, 1994; Cervino et al., 2001; Bruckner and Bruckner, 2006
Dark-spots disease (DSD)	Caribbean	*M. annularis, S. siderea, S. radians*, *S. intersepta;* [1] also *M. franksi, M. faveolata and M. cavernosa*[2]	Garzón-Ferreira and Gil-Agudelo, 1998; [2]Garzon-Ferreira *et al.*. 2001
DSD- II	Bermuda, Bonaire, Colombia, Puerto Rico, Venezuela	*S. intersepta, M. annularis, M. faveolata, M. cavernosa, C. natans, C. amaranthus, S. siderea*	Weil et al., 2002; Weil, 2004; Weil, 2006
Dark band syndrome (DBS)	Puerto Rico, Mexico	*M. annularis, M. faveolata*	Weil, 2002;2004
Purple band syndrome (PBS)	Grenada, Venezuela	*S. siderea, S. intersepta*	Weil, 2004
Tissue necrosis	Puerto Rico	*M. faveolata*	Weil, 2004

Table.1a. Syndromes reported for scleractinian corals and gorgonians in the tropical western Atlantic (continued).			
Red band disease (RBD) type I	Bahamas, Belize, Bonaire, Colombia, Costa Rica, Curaçao, Dominica, Puerto Rico, Jamaica, Mexico, Turks and Caicos, Florida	11 species: *Gorgonia*, *Agaricia*, *Colpophyllia, Mycetophyllia, Diploria, Stephanocoenia Millepora, Meandrina, Montastraea, Porites, Siderastrea.*	Rützler et al., 1983; Santavy and Peters, 1997
RBD type II	Bahamas, Mexico	*D. strigosa, M. annularis, M. cavernosa, P. astreoides, S. radians*	Richardson, 1992
Mottling syndrome	Flower Gardens GOM	*C. natans*	Borneman, 2005
Pale ring syndrome	Flower Gardens GOM	*Montastraea, Colpophyllia, Diploria*	Borneman, 2005
Light patch syndrome	Flower Gardens GOM	*D. strigosa*	Borneman, 2005
Hyperplasia (accelerated growth)	Bermuda, Puerto Rico, USVI, Jamaica, Netherlands Antilles, Trinidad, Belize, Brazil	12 species: *Porites, Favia, Diploria, Montastraea, Stephanocoenia, Acropora, Siderastrea, Colpophyllia.*	Loya et al., 1984
Calicoblastic Neoplasm	Florida, Bonaire, Puerto Rico, Trinidad, Mexico	*A. palmata*	Peters et al., 1986
Folliculinid ciliates (SEB)	Venezuela	10 species	Croquer et al., 2006
Shut-down reaction	Belize, Florida	massive corals, acroporids	Antonius, 1977
Coccidiosis	Jamaica, Puerto Rico, USVI	*A. agaricites, D. cylindicus, D. strigosa, M. meandrites, M. cavernosa, P. astreoides, P. porites*	Upton and Peters, 1986
Nematopsis spores	USVI	*Porites* spp	Peters, 1984
Stress-related necrosis	Puerto Rico	Multiple species	Peters, 1984
Blistering necrosis	Puerto Rico, USVI	*S.siderea D. strigosa, D. labyrinthiformis M.annularis, P.astreoides, S. intersepta, A. agaricites*	Peters, 1984
Ring disease	Bermuda, Florida, Honduras	*D. labyrinthiformis*	Weil, 2001
Algal tumors	Bonaire, Trinidad, Florida	*Gorgonia Pseudoplexaura Plexaura*	Morse et al., 1977
Aspergillosis	18 countries	*Gorgonia* spp.	Nagelkerken et al., 1997
Fire coral fungal disease	Florida	*Millepora* spp.	TeStrake et al., 1988
Epizoism	Florida and Belize	*Acropora, P. porites*	Antonius, 1998
Epizootic Cyanobacteria	Florida	*Briareum asbestinum*	Harvell et al, 2001

In a review article, Sutherland et al. (2004) suggests these all represent a single disease which she refers to as "white plague like", however the term white plague has not been reported in the IndoPacific. In contrast recent IndoPacific studies are reporting a disease with signs that are similar to WBD as white syndrome (WS) (Willis et al., 2004). To avoid confusion, the white diseases are grouped here as 1) **WBD** for Caribbean acroporids; 2) **white pox** (WPX) for acroporids reported with WPX, patchy necrosis or necrotic patch syndrome; 3) **white plague** (WP type I or WP-II) for all non acroporids corals in the western Atlantic with signs similar to WBD; and 3) **white syndrome** for cases identified as WBD, white syndrome, white plague, or plague-like from the Red Sea and IndoPacific

White band disease (WBD) was first observed in the mid 1970s in St. Croix, USVI among *A. palmata* populations (Gladfelter et al., 1977). It subsequently spread throughout the Caribbean where it affected *A. palmata* and *A. cervicornis*, with reports of WBD from 27 countries during the 1980s. WBD has been reported much less frequently during the last decade; isolated cases of WBD were identified among *A. palmata* populations in 5 countries (Jamaica, Mexico, Cuba, Caymans and Bahamas) with an outbreak observed in a single location that spread throughout a population off Mona Island, Puerto Rico between 2003-2005 (Bruckner, 2005). Conversely, recent outbreaks of WBD on *A. cervicornis* populations appear to be more prevalent over the last decade. This condition may represent a new syndrome (it has also been referred to as WBD-II by Weil, 2004 and rapid tissue loss by Williams and Miller, 2005), as rates of tissue loss are much more rapid than that reported for WBD and patterns of tissue loss were more irregular (Williams and Miller, 2005).

White pox (WPX) was first observed in Puerto Rico in 1994 (called patchy necrosis (PN); Bruckner and Bruckner, 1996) and in Florida in 1996 (Patterson et al., 2002). WPX has also been reported from the USVI and Puerto Rico, with reports for PN from the Bahamas, Cuba, Puerto Rico, Jamaica and necrotic patch syndrome from Mexico. WPX is believed to have caused losses of 88% of the remaining acroporids in the Florida Keys between 1996-2002 (Porter et al., 2001; Sutherland et al., 2004).

White plague has been reported from 20 countries in the Caribbean, with few reports specifically identifying this as Type I or Type II . **WP (type I**) was first observed in 1975 on reefs off Key Largo Florida among six species, with the highest prevalence in *Mycetophyllia* spp. and *C. natans* (Dustan, 1977). It was still prevalent throughout the Key Largo region ten years later, although *M. annularis* (complex) colonies were affected most severely, along with 11 other species (Dustan, 1987). Since this time, WP-I has only been reported from the Bahamas and Puerto Rico. A condition with similar signs, but more rapid rates of tissue loss and a wider host range emerged on these reefs in 1995 (**WP type II**). The most susceptible species (*D. stokesi*) was unaffected during WP outbreaks in the 1970s and 1980s; it was also observed on 17 other species in Florida, including 8 (*M. annularis, M. cavernosa, M. faveolata, S. siderea, A. agaricites, C. natans, D. labyrinthiformis, S. intersepta*) reported during earlier WP outbreaks (Richardson et al., 1998). WP type II has been reported from 9 countries, with infections documented on 41 species (Weil et al., 2006). A separate condition termed WP Type III (based on rates of spread of up to 10 cm/day) was reported to affect the largest massive corals including *Montastraea* spp. and *C. natans* (Richardson and Aronson, 2001); it is unclear whether this is distinct from WP-II and epizootiological data are currently

unavailable. Outbreaks of WP have been reported more frequently since 2000, including offshore locations and deeper reefs (e.g., Sherwood Forest, Dry Tortugas; St Croix; La Parguera PR Shelf Edge). A similar condition has also been observed in a remote location (Flower Gardens, Texas) that was not previously affected (Hickerson, 2005).

White syndrome was first reported from the Red Sea in 1996 and Australia in 2001. This may be the same as WBD, which was first documented in the IndoPacific in the 1980s as many of the same species are affected and patterns of tissue loss are similar. Antonius (1981; 1985) reported WBD in the Red Sea on 17 genera and 31 species of corals, including 11 acroporids (Egypt, Saudi Arabia, United Arab Emirates) and 22 species in the Philippines, including two new genera (*Montipora* and *Podabacia*). Additional cases reported over the last ten years in Australia, Guam, Oman, India, Malaysia and the Philippines (Coles, 1994; Riegl, 2002; Jeyabaskaran and Raghukumar, 2004). Willis et al. (2004) observed a 20 fold increase in the number of corals affected by white syndrome between 1998 and 2003, with the greatest increases on outer reefs. In addition, infections spread from 75% of the regions and 45% of the reefs in 1998 to all regions and 89% of the reefs by 2003 (Willis et al., 2004). A disease that is similar to white syndrome and white plague was reported in a subtropical location (Solitary Islands) off Australia. Six coral genera were affected, with new observations for *Turbinaria* (2 species). Disease incidence in the Solitary Islands varied throughout the year but was lowest in March (6.2%) and highest in June (13.6%) (Dalton and Smith, 2006).

***Porites* ulcerative white spot syndrome** (PUWS) was first observed in 1996 in the Philippines, where it caused discrete bleached round lesions that may result in ulcerations that coalesce and cause tissue loss and colony mortality (Raymundo et al., 2003). This disease affected >20% of the *Porites* colonies on 8 out of 10 reefs examined in the 1990s (Raymundo et al., 2003). More recently up to 40% of the colonies were affected at sites near a populated city (Dumaguete), with prevalence declining with increasing distance from the city. The incidence of PUWS also increased between March and August, 2003, as water temperatures became elevated. In this study, PUWS was identified to affect 6 branching species of *Porites* and one massive species (Kaczmarsky, 2006).

Shutdown reaction (SDR) has been reported in the Caribbean and Red Sea, with a single report from Tonga (Chesher, 1985; Antonius, 1988). No information is available on the prevalence of this condition. There also was a single report of **white blotch disease** in Australia.

2. Cyanobacterial mat diseases

Interactions between cyanobacteria and corals have been documented throughout the Caribbean, and on reefs of Guam, Micronesia, NWHI and other locations, and cyanobacterial blooms are believed to be becoming more frequent (Thacker and Paul, 2001; Kuffner and Paul, 2004). A number of cyanobacteria have been identified as the primary causative agent of coral diseases (e.g., BBD and RBD) while others that form mats on the substrate and may smother corals and other organisms (e.g., *Schizothrix*).

Table 2. Various white syndromes reported to affect stony corals in the tropical Pacific Ocean, Indian Ocean and Red Sea.

Condition	Location and Species affected	Description	Source
White band disease	Australia, Egypt, Guam, India, Mauritius, Oman, Papua New Guinea, Philippines, Saudi Arabia, United Arab Emirates 18 genera; 37 species	A distinct band of white, recently exposed skeleton between healthy tissue and algal colonized skeleton. The white band forms a moving front that advances a few mm per day. It may be triggered by contact to cyanobacteria[1]	Antonius, 1981; 1985; 1987; 1995[1] Coles, 1994; Korrubel and Riegl, 1998; Baird, 2000; Riegl, 2002
White syndrome	Egypt[2], Australia, Solitary Islands, [4] 38 species[1, 2, 3] *Turbinaria, Acropora, Goniastrea, Pocillopora, Stylophora* and *Porites*[4]	A distinct band of white, recently exposed skeleton between apparently healthy tissue and algal colonized skeleton that advances several mm/day	[1]Willis et al., 205; [2]Riegl, 1998 [3]AIMS archives [4]Dalton and Smith, 2006
Shutdown reaction (SDR)	Saudi Arabia, Egypt, Tonga *Acropora*	Complete and sudden disintegration of coral tissue, starting at the margin of an injury. Coenosarc sloughs off skeleton in thick strands or blobs at rates of 10 cm/hr	Antonius, 1988 Chesher, 1984
Porites ulcerative white spot disease	Philippines *Porites: 7 species*	Ovoid bleached lesions, 3-5 mm diameter, affecting 3-4 polyps and surrounding coenosteum; discrete margin between bleached and apparently healthy tissue. Bleaching is followed by tissue mortality. Recovery observed in small lesions; larger lesions may coalesce and kill the coral	Raymundo et al., 2003; Kaczmarsky, 2006
White blotch disease	Australia *Acropora*	White blotches associated with infestations of polychaetes	Dinsdale, 1994

Black band disease (BBD) was first described in 1972 from reefs off Belize, Puerto Rico, Florida and Bermuda (Antonius, 1973). BBD is now known to occur in at least 25 countries in the western Atlantic, and 11 countries in the Red Sea and Indo-Pacific, although cyanobacteria differ depending on location (Cooney et al. 2002, Frias-Lopez et al. 2002, 2003). In the western Atlantic, BBD has been reported on 26 scleractinian corals, one hydrozoan coral (*M. complenata*) and six gorgonians (Rützler et al. 1983; Feingold 1988; Green and Bruckner 2000). Faviid corals are most susceptible, although infections are frequently observed on *S. siderea*, and occasionally on *Agaricia* spp, *Mycetophyllia* spp., and *M. meandrites*. Caribbean colonies of *Porites* spp. and *Acropora* spp. were thought to be resistant to BBD, although BBD was reported on *A. palmata* in Colombia (Garzon-Ferriera et al., 2001), *P. astreoides* in Cuba (AGRRA database), Bermuda (Garret and Ducklow, 1975) and Mexico (Ryan Walker, Coral Cay Conservation, pers. Comm.), and *P. porites* in the Bahamas and Jamaica (AGRRA database). Several cases of BBD have also been observed on *Solenastrea bournoni* in Mexico (Ryan Walker). Sutherland et al. (2004) also reports BBD on *Madracis mirabilis* and *M. decactis*, although the source of these records is not provided.

In the IndoPacific and Red Sea, BBD has been observed on 19 genera and 49 species, most commonly on faviids and acroporids (Antonius 1988; Miller 1996; Green and Bruckner 2000; Dinsdale 2002; Sutherland et al., 2004). BBD was observed on 19% of 91 reefs examined in 1993/94 (Miller, 1996). More recent surveys show that BBD is widely distributed throughout the GBR Australia, but prevalence remained very low (04-0.47 colonies per reef on any given year) between 1998 and 2003 (Willis et al., 2004). Kaczmarsky (2006) recently identified an outbreak of BBD affecting close to 8% of the corals at one site in the Philippines; these observations include 5 new hosts for BBD in the Philippines and one new genera overall (*Coscinaraea columna*).

Red band disease (RBD type I and RBD-II) are only known from the Caribbean, although another disease termed "red band" has also been reported from Palau on *Pachyseris* spp. (Harvell et al., 2004). RBD- I was first identified on gorgonians in Belize in the 1980s, and has since been reported from 12 countries in the Caribbean, with records from 10 scleractinian corals, *Millepora* spp., and the sea fan *G. ventalina* (Rutzler et al., 1983). RBD-II has only been observed in a single location in the Bahamas (Richardson, 1993).

Several other cyanobacterial diseases have also been reported from the IndoPacific. Black overgrowing cyanobacteria is associated with at least five cyanobacteria that overgrow *Acropora, Favia, Pocillopora* and *Porites* on reefs in the Indian Ocean (Antonius, 1995). Black aggressive band has been observed in a single location on branching acroporids (Antonius, 1995), while brown band disease has only been recorded on *A. formosa* on the GBR Australia (Dinesdale, 1994; Antonius, 1999). Pink line syndrome, reported on *Porites compressa* and *P. lutea* in the Indian Ocean, has also been associated with a cyanobacteria (*Phormidium valderianum*) that is thought to induce pink coloration in affected coral tissue (Ravindran & Raghukumar, 2002).

3. Color change

In many reported syndromes, color change is an important diagnostic feature used to identify and differentiate diseases. Color change may include darker than normal tissue, lighter tissues, or a change in color such as the appearance of purple or pink spots or bands. Three syndromes associated with lightening of tissue have been reported from the Flower Gardens (Texas). This includes: 1) **light patch syndrome** which is observed in *D. strigosa* and is associated with variably sized, solid patches of uniformly contrasting paler tissue on normally pigmented colonies; 2) **pale ring syndrome**, which causes a variably wide ring that occurs singly or in multiples on the corallum surface in *M. faveolata*, *M. cavernosa*, *C. natans* and *D. strigosa*; and 3) **mottling syndrome** in *C. natans*, in which the tissue appears mottled as a result of total to near-total bleaching associated with a focal lesion, with spotty bleaching occurring in a wide band that grades towards apparently healthy tissue (Borneman, 2005).

Table 3. Diseases associated with cyanobacteria reported to affect stony corals in the tropical Pacific Ocean, Indian Ocean and Red Sea.

Condition	Location and Species affected	Description	Source
Black band disease	Australia, Egypt, Fiji, India, Jordan, Papua New Guinea, Philippines, Saudi Arabia, Tonga, South Africa, CNMI, Palau 19 genera, 49 species; Pocillopora and Acropora most frequently affected	A darkly pigmented mat/band 1-30 cm wide on the surface of the coral that separates healthy tissue from recently denuded white skeleton.	Antonius, 1987; Chesher, 1985; Glazebrook and Steiner, 1994; Littler and Littler, 1996; Miller, 1996; Korrubel and Riegl, 1998; Fenner, 1998; Cervino, 1998; Jordan and Samways, 2001; Dinesdale, 2002, Willis et al., 2004
Brown band disease	Australia *Acropora Formosa*	Different condition from above associated with cyanobacteria	Dinsdale, 1994
Black aggressive band	Mauritius *Acropora* (staghorn coral)	Resembles BBD but the band material is thinner and appears grey rather than black; possibly caused by a cyanobacteria (*Spirulina*) or a spirochete	Antonius, 1995a
Black overgrowing cyanobacteria	Indian Ocean, Mauritius *Acropora, Favia Pocillopora, Porites,*	Cyanobacteria (*Calothrix, Hormothamnium, Lyngbia, Phormidium, Spirula*) cover coral tissue and progressively overgrow it; may penetrates and erodes skeleton	Antonius, 1995a
Red band disease	Palau *Pachyseris speciosa* and *Porites* spp.	A reddish band on the surface of the coral that separates healthy tissue from recently denuded white skeleton.	Harvell et al., 2004; Sussman et al., 2006
Pink line disease /syndrome	Papua New Guinea, Sri Lanka, Kavaratti Island, Indian Ocean *Porites compressa, P. lutea*	Band of pink pigmented tissue separating recently killed skeleton and normal tissue; it may begin as a small ring and progress outward. Associated with a cyanobacteria.	Ravindran et al., 2001; Goreau/Cervino, coral list server

Two distinct conditions have been reported as **yellow band disease**. In the Caribbean, YBD (also referred to as yellow blotch disease) was first reported from Florida in 1994 (Reef Relief), and subsequently observed in 24 countries throughout the Caribbean. It primarily affects *M. annularis* complex and 4 other massive faviids corals, *A. agaricites,* and *P. astreoides*. This disease has been reported at an unusually high prevalence in a number of countries (18-91%) including Puerto Rico, Mexico, Curacao, Bonaire, Grenada, Panama, and USVI (Cervino et al., 2001; Jordan-Dahlgren and Rodriguez-Martinez, 2004; Bruckner and Bruckner, 2006). **Yellow band disease** was first reported in 1995 from the Arabian Gulf off Dubai (United Arab Emirates) and in 1999 and 2003 off Fahr Island and Kish Island (Iran). It affects *Turbinaria, Porites, Cyphastrea* and *Acropora* (Korrubel and Riegl, 1998).

Dark spots disease was first observed in Colombia in the mid 1990s, but has since been reported from 15 other countries in the Caribbean (Garzon-Ferriera et al., 2001; Weil 2004). It is most commonly observed on the genera *Stephanocoenia, Siderastrea* and *Montastraea*, although similar signs are observed on 6 other species. Colonies are characterized by darkly pigmented spots or bands within the tissue, and occasionally extending into the skeleton, with depressed skeletal features observed in *Stephanocoenia*. Over time these dark spots may increase in size, or the center of the spot may die and dark tissue may expand into a band or ring that slowly migrates outward. Weil (2002; 2004) reported three additional syndromes that are similar in appearance to DSD (DSD-II, dark band disease, purple band disease and tissue necrosis). These syndromes could be related, or are a different stage in the progression of dark spots disease (Weil, 2004).

4. Other conditions

Two conditions have been reported in the GCDD that are associated with ciliates**, brown band disease (BrBD)** and **skeletal eroding band (SEB).** BrBD has only been reported from the GBR, Australia, where it affected a low proportion of corals (0.3%), including acroporids, pocilloporids and faviids (Willis et al., 2004). SEB has been observed in the Red Sea, Indian Ocean and Pacific (5 countries) on 21 genera of corals (Reigl and Antonius, 2003; Willis et al., 2004). In Australia, SEB was the dominant disease affecting acroporids and pocilloporids, with a 20 fold increase during summer (Willis et al., 2004). A similar condition (folliculinid ciliates) was recently reported from the Caribbean (Venezuela) on 10 species of coral (Croquer et al., 2006).

Tumors (including calicoblastic neoplasms, hyperplasias, abnormal growth) are among the most widely reported condition affecting corals with the first observations over 40 years ago (Squires, 1965), and subsequent reports from 15 countries in the IndoPacific and 13 countries in the Caribbean. Neoplasia has been reported most frequently on *Acropora*, with reports from the Caribbean, Philippines, Guam, Hawaii, and Oman.

Conclusions

1. Diseases occur globally, in most reef habitats and in most locations including reefs near human population centers and remote offshore locations. Although most of the reports available prior to 1998 were from areas that had a medium to high level of human impact, reports of disease from remote locations has escalated, and in some cases offshore locations are exhibiting the most dramatic increases in diseases incidence and mortality.
2. Diseases have been observed in 63 countries, a 17% increase since 1999. This includes increasing numbers of observations of disease in the IndoPacific, along with a number of new diseases and increasing prevalence of these diseases, but Caribbean reefs are still disproportionately affected by disease.
3. There are six major diseases of concern in the Caribbean (BBD, WBD, WP, YBD, ASP, WPX) that have caused substantial coral mortality since their discovery; two of these remain a major threat to acroporids (WBD, WPX), one is impacting a growing number of gorgonian species (ASP), and three (WP, YBD, BBD) are of major concern to *M. annularis* (complex) and other species. Two other conditions appear to be widespread (DSD and SEB), but are causing slow rates of mortality at this time.
4. The disease of most concern on IndoPacific reefs is white syndrome, which is having the largest impacts on acroporids throughout the region; PUWS is a growing threat to *Porites*, but at this time it appears to be restricted to the Philippines. Most other newly emerging IndoPacific diseases have caused localized mortality and appear to have a limited distribution.
5. Although a greater number of corals have been identified with disease in the IndoPacific (34 genera and 97 species), a higher percentage of coral species (close to 80% of all taxa; 41 species of scleractinian corals, 8 gorgonians, 2 hydrozoans) are affected by diseases in the Caribbean. This represents a 25% increase in number of genera and 45% increase in number of species and includes 7 new genera identified with disease in the IndoPacific since 1999.
6. Rapidly growing corals in the family acroporidae and pocilloporidae in the IndoPacific are affected by the largest number of diseases and are observed with disease more frequently than all other species, while the *M. annularis* complex is being affected most severely in the Western Atlantic.
7. Tumors (hyperplasia, neoplasia etc.) are the oldest known afflictions of corals, and are found on most corals in most locations, but their impacts appear to be minimal at this time.

Table 4. Other Diseases, syndromes, and anomalies reported to affect stony corals in the tropical Pacific Ocean, Indian Ocean and Red Sea.

Condition	Location and Species affected	Description	Source
Yellow band disease	United Arab Emirates; Arabian Gulf; Iran[2] 4 genera, 12 species	A broad band of denuded skeleton, yellow in color, adjacent to decaying and sloughing tissue; the band advances 9-20 mm/week.	Korrubel and Riegl, 1998 [2]Maghsoudlou, and Eghtesadi, 2004
Brown band disease	Australia *Acropora Formosa*	A brown band of variable width flanked by healthy tissue at6 the advancing front and exposed white skeleton at the trailing edge. The band moves in both directions along the branch, destroying coral tissue. Dense populations of ciliates, packed with zooxanthellae from coral cause brown coloration.	Willis et al., 2004
Skeleton eroding band (SEB)	Egypt, Jordan[1] PNG, Mauritius[2], Australia [2] 21 genera ; *Cyphastrea chalcidicum*, acroporids[2] 13 genera scleractinian, 1 hydrozoan in Australia [3]	Masses of black loricae of *Halofolliculina corallasia*, a colonial heterotrich ciliate, that forms a front separating live tissue from a white zone; the front advances like BBD, causing tissue loss and skeletal damage.	[1]Antonius, 1999; Winkler et al., 2004 [2]Riegl and Antonius, 2003 [3]Willis et al., 2004
Plagioporus	Hawaii *Porites compresssa, P. lobata*	Metacercaria of the digenetic trematode encyst in elevated nodules, causing enlarged pink polyps. Cyst wall is secreted by parasite, produces distortions of gastrovascular cavity and cellular alterations within tentacles	Aeby, 1991
Patchy necrosis	Adaman Islands, Indian Ocean *Porites,Goniastrea Goniopora, Montipora, Favia, Goniastrea* and *Pocillopora*	Hyphomycetous fungus associated with necrotic patches. Top layer of necrotic patches consists of epilithic algae, followed by a thin black zone of fungal growth, a green band containing shell-boring algae and a dense black fungal layer at the base	Raghukumar and Raghukumar, 1991; Ravindran et al., 2001
Fungal syndrome	East African Coast *Astreopora, Montipora, Echinopora, Acropora, Goniopora, Platygyra, massive Porites, Pocillopra, Goniastrea Hydnophora, Cyphastrea*	Corals develop ashy dull color and brittle or weak skeleton. Corals become covered in mucus, which traps debris. Once this clears, a white calcareous dust is left on the surface and a black layer forms underneath; death occurs in about two weeks	McClanahan et al., 2004

Table 4 (continued). Other Diseases, syndromes, and anomalies reported to affect stony corals in the tropical Pacific Ocean, Indian Ocean and Red Sea.

Condition	Location and Species affected	Description	Source
Hyperplasia (and other reports of tumors)	Australia, Hawaii, Guam, Palau, Enewatak, French Polynesia, New Caledonia, Maldives, Micronesia, Marshall Islands, Japan, Oman, China, Philippines *Pocillopora, Pavona, Fungia, Madrepora, Montipora, Platygyra*	Irregular growths on colonies reported as tumors and hyperplasms associated with a proliferation of cell types (normal in appearance, but larger in size)	Loya et al. 1984; Peters et al., 1986 ; Glazebrook and Steiner, 1994;Yamashiro et al., 2000
Neoplasia	CNMI, Oman Acropora	calicoblastic epitheliomas, neoplasms associated with a proliferation of cell types and white globular masses of skeleton with few discernable polyp structures.	Cheney, 1975; Coles and Seapy, 1998
Stress related necrosis	Hawaii *Porites lobata*	Gram negative bacterial aggregates in gastrodermal cells of tentacles. Tissues exhibit lysed nuclei and cell death	Hunter,
Pink-blue disease	Israel, India, Lackshadweep Islands *Acropora, Porites*	Pink to blue coloration adjacent to lesions	Red Sea Marine Park, 2001; Ravindran et al., 2001
Black necrosing syndrome	Australia Gorgonians, *Isis hippuris*	Black necrotic patches appearing on 10% of the population on one reef	Morrison Gardiner, 2001; Willis et al., 2004
Vibrionic Bleaching	Mediterranean, Israel, Tanzania *Oculina pategonica; Pocillopora*		Rosenberg, 2002
Atramentous necrosis	Florence Bay and Bright Point, Australia *Montipora aequituberculata*		Jones et al., 2004
Yellowing disease	Sodwana, South Africa *Favia pentagona* and *Lobophytum*		Jordan and Samways, 2001
Red plague syndrome	Kavaratti Island, India *Montipora* spp. and *Porites* spp.		Jeyabaskaran and Raghukumar, 2004

Fig. 1. Five major scleractinian coral diseases reported for the wider Caribbean compiled in the GCDD. Reports of syndromes with different "types" (e.g., WP type I and WP type II) have been pooled.

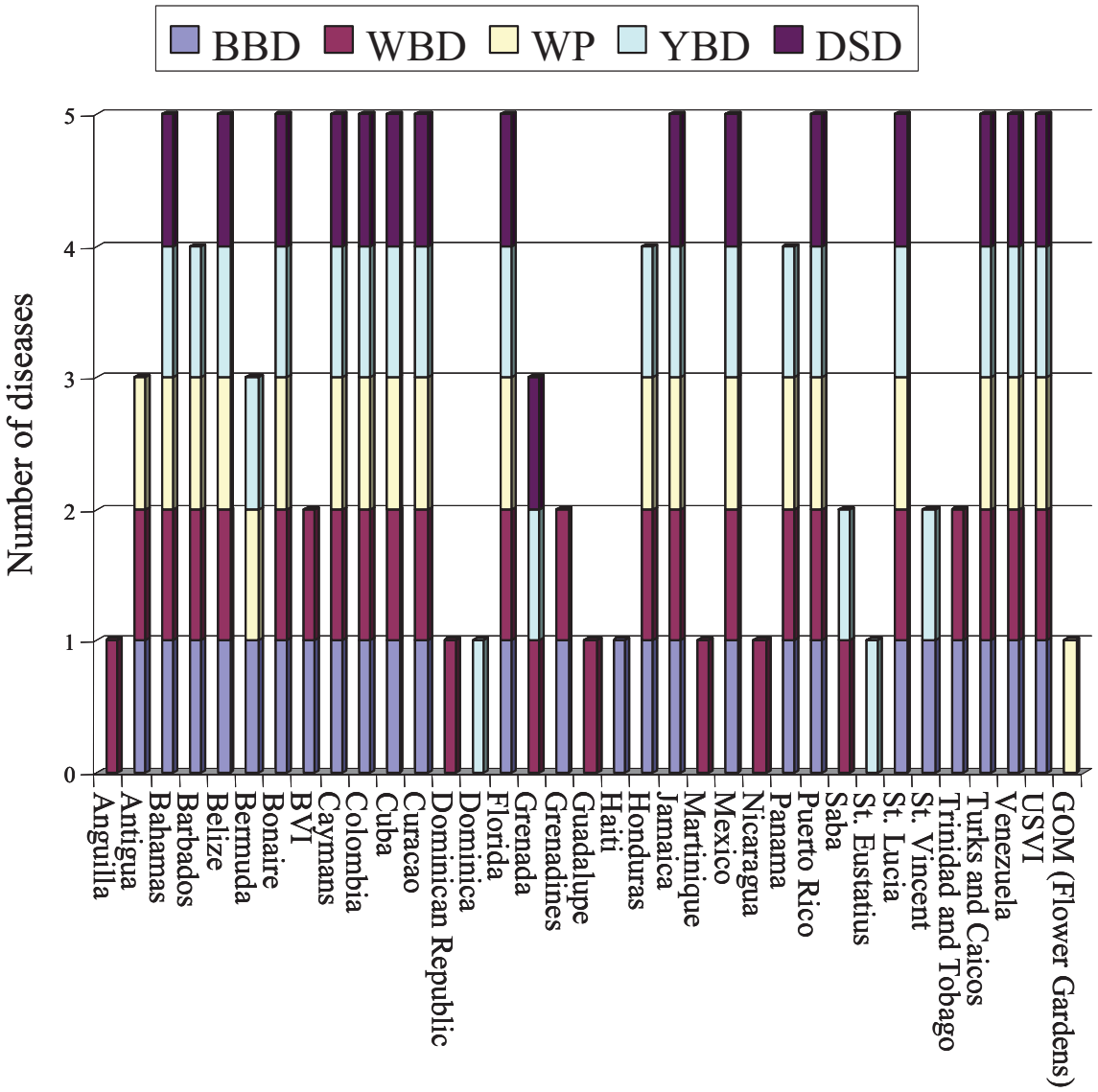


Fig. 2 Susceptibility of scleractinian corals to seven major syndromes observed in the Indo Pacific.

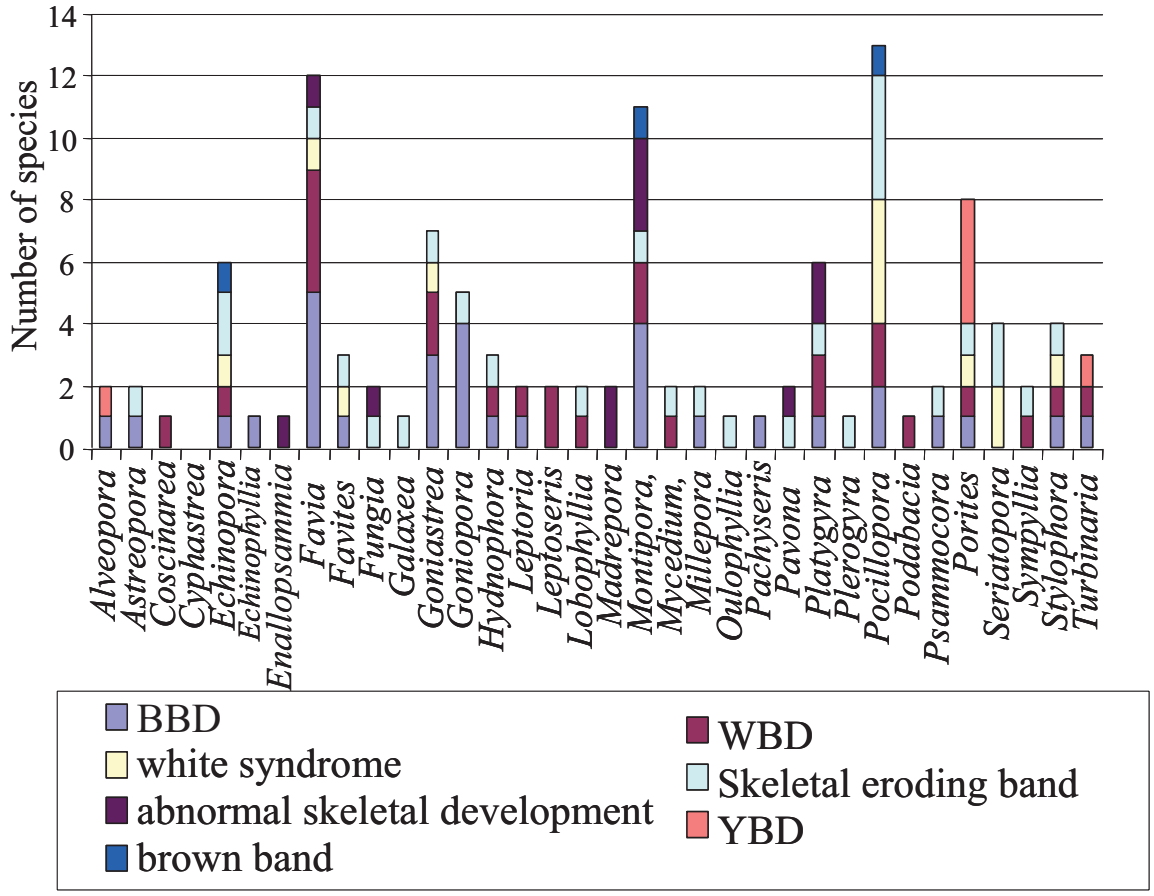


Fig. 3. Number of diseases observed on IndoPacific Reefs. Only those conditions reported in peer-reviewed publications are included

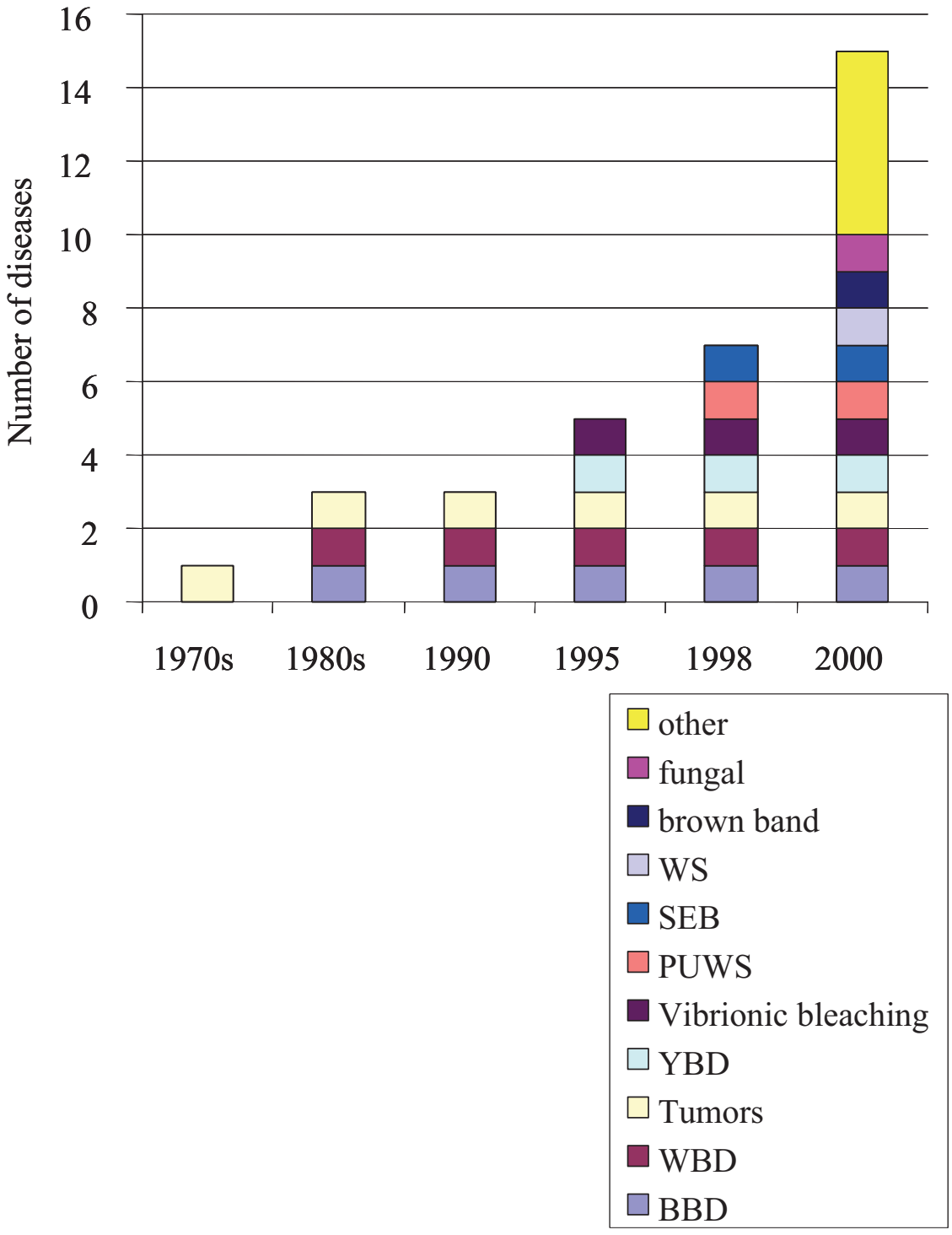
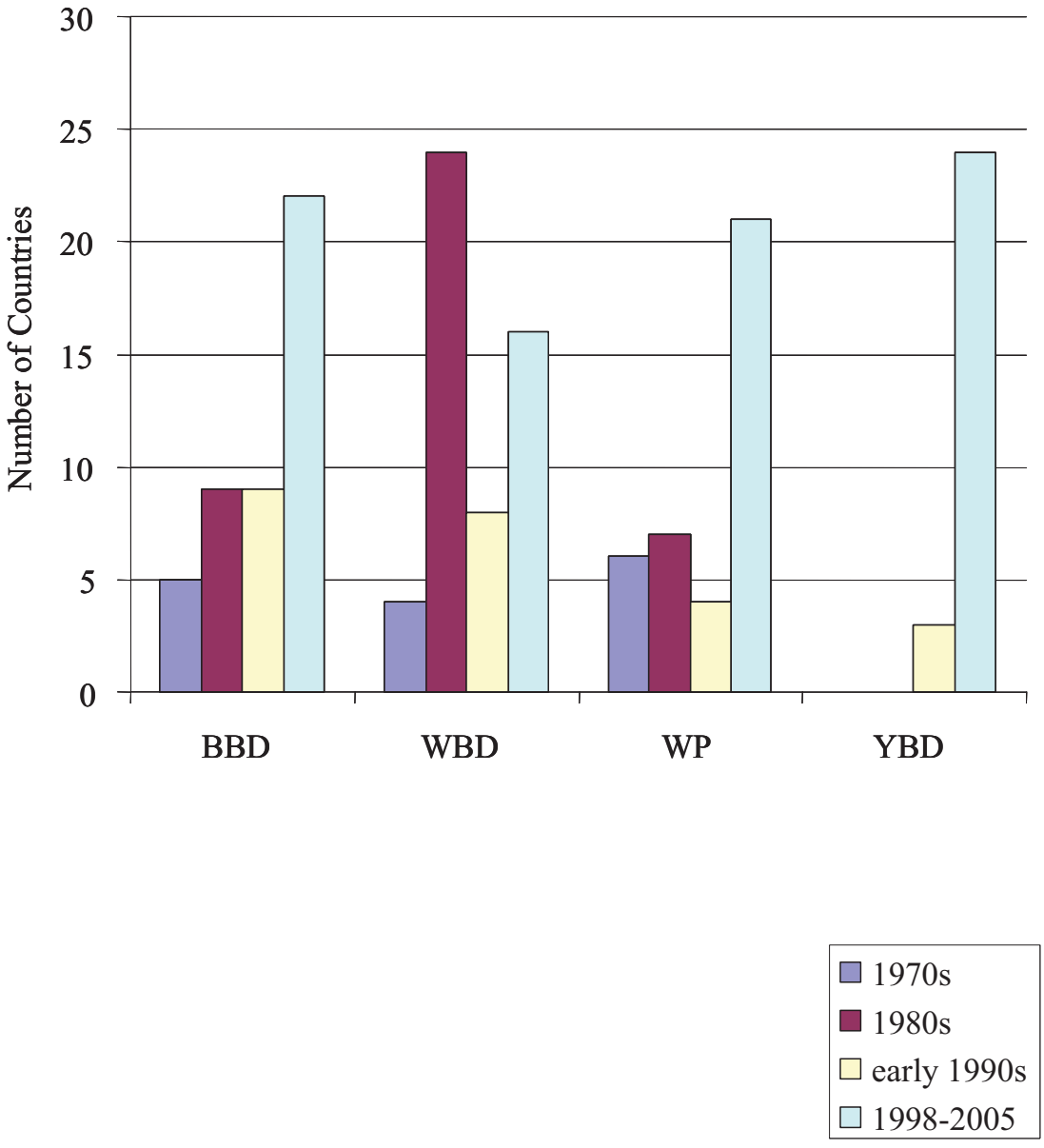


Fig. 4. Number of countries reported with BBD, WBD, WP and YBD during the 1970s, 1980s, 1990s and today



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WORLD BANK PROJECT: CORAL DISEASE WORKING GROUP

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CRTR Coral Disease Working Group

(excerpted from the CRTR webpage - http://www.gefcoral.org/**)**

Coral reefs are under increasing stress from a number of causes, including climate warming, poor water quality and over fishing. Disease outbreaks cause not only coral loss, but they can result in significant changes in community structure, species diversity and reef-associated organisms. Coral diseases potentially impact both well-managed and unmanaged reefs indiscriminately. However, strategies for dealing with disease outbreaks are currently non-existent. The increasing frequency with which diseases influence and alter reef communities means they must be considered and incorporated into management plans.

Background:

The CRTR Program is a partnership between the Global Environment Facility, the World Bank, The University of Queensland (Australia), the United States National Oceanic and Atmospheric Administration (NOAA), and approximately 50 research institutes and other third parties around the world. The CRTR Coral Disease Working Group's research will provide us with a greater understanding of the ways in which coral diseases can alter reef function and the conditions under which outbreaks may occur.

Global impact of coral disease

Coral disease stands out as a primary factor in the deterioration of many coral reefs worldwide, with preliminary surveys indicating that significant and damaging new diseases are now beginning to appear in all reef regions. The CRTR Program Disease Working Group is conducting a global coral disease census across 24 high priority sites. This major assessment is designed to catalogue syndromes for the first time, and to reveal whether disease outbreaks are correlated with climate warming anomalies. In each location the impact and prevalence of coral disease is being measured.

Global warming and anthropogenic inputs

Increases in disease following warming events may be because corals have lower ability to fight disease while under temperature stress, or because bacteria are more virulent. While connections between poor water quality (nutrient loading and sedimentation) and disease are of increasing concern, evidence of direct links and synergistic effects is limited. The CRTR Program Disease Working Group is measuring nitrogen and sediment loading at key research sites. The team will use molecular and enzymatic techniques to assess differences in microbial communities - in coral mucus, water and sediment

between sites with different loadings, and between healthy and bleached corals. There will also be an evaluation of climate and anthropogenic influences on changes within microbial communities.

The causes, reservoirs and vectors of corals disease

Current research on disease reservoirs and vectors is hampered by a lack of knowledge of the pathogens causing the majority of coral diseases. To date there are only five coral diseases for which the microbial cause is known. The Disease Working Group is developing a suite of techniques to facilitate the identification of pathogens in coral. Because only a small percentage of bacteria in nature are culturable, the identity and source of pathogens will be confirmed using various molecular fingerprinting techniques. Eventually a micro-array chip of global coral disease will be developed.

Coral resistance to disease

The microbial communities associated with coral are very complex, existing both inside the coral animal and in the surface mucous layers (SML). These normal communities protect the coral from disease. When the community structure changes, corals may become more susceptible to disease. Both bleaching and disease appear to change the microbial community profiles in the SML. The goal of the Disease Working Group's immunological work is to develop assays to determine general antimicrobial activity. Once resistant compounds are identified, they will be incorporated into a chip of biomarkers for stress. Field sampling will eventually allow the team to quantify and estimate the response of corals to different experimental treatments of enhanced nutrients and temperature, and map the spatial extent and variation of disease resistance in the field.

Our Research

Research Activities:

Over the last 20 years, unprecedented increases in disease on coral reefs have contributed significantly to coral reef degradation. Disease-related damage of coral reefs has been well documented in the Caribbean, but recent observations of coral disease in other regions of the world are just beginning. Disease occurrence in these other regions may be a potential harbinger for further outbreaks and impacts associated with increasing climate warming. The Disease Working Group is targeting investigations to address the causes of this rapid emergence of coral disease, to understand the impacts of the problem and to develop tools and responses that can be used for management.

Research Update:

The Disease Working Group has answered many pressing questions including which disease syndromes are infectious; which reef regions surveyed as part of the the CRTR Program have the greatest prevalence of coral disease; which Centres of Excellence would be the most suitable for identifying local factors that might impact on disease; and whether ocean warming affects coral disease levels.

Impact of fish farms
As part of its study of the impact of local environmental factors on coral health, the Group has found that the fish pens in Bolinao Bay (Philippines) have a strong influence on bacterial communities, nutrient input, primary production and the patterns of energy and carbon flux in the surrounding waters. Researchers are working to identity specific bacteria from fish farms that reside on the surface of reef corals, and whether aquaculture plays a role as an incubator, conveyor and facilitator of disease into natural populations.

Disease in a warming ocean
The Group has made significant discoveries in the Caribbean and Great Barrier Reef region in Australia regarding the potential impacts of heat stress, associated with climate warming events, on the outbreak of coral disease. In collaboration with the Remote Sensing Working Group, it is developing new models to predict disease outbreaks using satellite monitoring data. The models use predicted sea temperature data and can identify the potential efficacy of various management strategies for future scenarios.

Other causes of coral disease
The Group continues to survey the prevalence of coral disease in Caribbean, Yucatan and Australian coral reefs, and is making progress in determining agents that cause coral disease such as skeletal eroding band, brown band and white syndrome.

Tools for Management:
The Disease Working Group has developed important new tools for coral reef managers and researchers across the Western Atlantic and the Indo-Pacific to identify and address coral disease – the *Coral Disease Handbook: Guidelines for Assessment, Monitoring and Management* and two sets of underwater identification cards. These were launched at the 11th International Coral Reef Symposium in July 2008.

Handbook
Designed for reef managers by international experts in coral disease, the Handbook outlines procedures for describing indicators, measuring impacts, monitoring outbreaks, assessing causes, and managing reefs to minimize losses due to disease. This handbook helps managers not only to document and manage disease on their reefs, but also enables them to contribute to our scientific understanding of this grave and increasing threat.

Underwater Cards Caribbean
These Underwater Cards for assessing the health of coral reefs have been designed so that scientific, professional and recreational divers can all assist with gathering information on the occurrence of coral reef diseases in the Caribbean. These cards will assist in the identification and monitoring of diseases in Caribbean coral and other reef organisms.

Underwater Cards Indo-Pacific
These Underwater Cards for assessing the health of coral reefs have been designed so that scientific, professional and recreational divers can all assist with gathering information on the occurrence of coral reef diseases in the Indo-Pacific. These cards will assist in the identification and monitoring of diseases in Indo-Pacific corals and other reef organisms.

Who we are

Working Group Members:

Working Group members bring international expertise and experience to this targeted research: C. Drew Harvell (Chair), Garriet W. Smith (Co-Chair, Microbiology), Bette Willis (Co-Chair, Ecology), Farooq Azam, Eric Jordan Dahlgren, Eugene Rosenberg, Ernesto Weil, Laurie Raymundo.

Project Partners:

Working Group partners bring capacity to this research endeavour:
Section of Ecology and Evolutionary Biology, Cornell University, USA;
Department of Biology and Geology, University of South Carolina-Aiken, USA;
Scripps Institution of Oceanography, University of Southern California, USA;
Unidad Académica de Sistemas Arrecifiales, Instituto de Ciencias del Mar y Limnologia, Universidad Nacional Autónoma de México, Mexico;
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III. HISTORICAL PERSPECTIVE--LESSONS LEARNED

PROGRESS IN UNDERSTANDING CORAL DISEASES IN THE CARIBBEAN

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ABSTRACT

Coral disease research in the Caribbean initiated in 1972 with the discovery of black band disease (BDD) by Dr. Arnfred Antonius. Since this time, there has been an expansion in the number of researchers working in the Caribbean, including studies to document the prevalence and incidence across large spatial scales and at increased temporal frequencies, evaluation of the linkages between disease and environmental drivers, identification of disease vectors, and laboratory studies to characterize causes, physiological responses, histological changes, defense mechanisms and mechanisms of resistance and susceptibility. Since 1998, the Caribbean has emerged as a "hot spot" for coral diseases due to the large number of named diseases, their wide distribution, expanding host ranges, and increasing abundance and severity, with over 30 diseases now reported from this region. Localized epizootics of three diseases (BBD, WBD, WP-I) were first documented in the 1970s; one of these (WBD) expanded throughout the Caribbean to become the most significant factor in the region-wide decline of acroporids. Five diseases (WP-II, YBD, WPX, DSD and ASP) emerged in the mid 1990s, and have expanded their geographic distribution and host ranges over the last ten years with several of the diseases causing substantial coral mortality since the late 1990s. By 2005, at least 41 scleractinian corals, 8 gorgonians and two hydrozoans were observed with one or more diseases. The most abundant and important group of corals found on Caribbean reefs today (*M. annularis* complex) is susceptible to at least 8 different diseases, and individual colonies may show signs of 2-3 diseases at the same time. The average prevalence of coral diseases at the community level is generally low, although it is highly variable between and within sites, during different years, and seasonally. Disease outbreaks have affected up to 91% of certain susceptible populations in localized areas, and often (but not always) exhibit a clumped distribution. Disease prevalence and severity is generally greater during warm water periods, and recent disease outbreaks have been associated with mass bleaching events. Over the last five years, there has been an increase in the numbers of studies that have reported a correlation between disease and environmental factors, including higher prevalence rates and greater rates of spread in areas affected by nutrients, sediments and other pollutants. Causative agents have been identified for relatively few diseases, three of which (WP-II, ASP and WPX) have been verified through application of Koch's postulate. In these and other diseases, complex microbial communities have been identified using new molecular techniques, including

biota on diseased tissue that is absent from control samples and suites of microorganisms that differ from those identified using traditional (microscopy and culture) techniques. Furthermore, pathogens identified using traditional microbiological approaches are no longer infective, including the proposed causative agent for white plague type II, suggesting 1) the pathogens may have lost their virulence and/or corals have gained immunity, or 2) causation was determined based on a relatively small number of corals from a single location/event and other microbial agents are capable of causing similar signs. In addition to key advances in understanding the coral holobiont, and how microbial communities associated with coral tissue and coral mucus change during periods of stress, some advances have been made in identifying possible vectors of disease, including linkages between a coral eating snail (*Coralliophila abbreviata*) and a white syndrome that affects acroporids as well as three spot damselfish and BBD. Efforts have been made to mitigate disease, through removal of the microbial community, antibiotic treatments, use of putty and/or clay to cover the affected area, and addition of urchins to reduce algal abundance, however these exhibited only limited success and they do not appear to be feasible treatments on a larger scale. One of the major limitations in advancing our understanding of diseases has been the lack of standardized nomenclature and diagnostic criteria for diseases, which has resulted in a proliferation of names and the identification of new presumed diseases that later have been shown to be caused by other factors. Some of the key needs for the Caribbean include: 1) greater geographic coverage and more frequent surveys to characterize prevalence and incidence; 2) more emphasis on population dynamics and impacts, including size structure of diseased and healthy corals, extent of partial and whole colony mortality and impact to individual corals and coral populations; 3) concurrent monitoring and assessment of environmental factors; 4) revision of existing disease nomenclature and adoption of standardized terminology and diagnostics; 5) application of traditional culture and histopathology techniques in combination with new molecular tools to characterize causative agents and sources of pathogens and development of molecular probes to facilitate screening of corals; and 6) more emphasis on cellular diagnostics, including biomarker characterization, to assess stress levels in corals and underlying causes; and 7) a coordinated rapid response program to address coral disease outbreaks and unusual mortality events.

Introduction

Until the late 1970s, benthic substrates on Caribbean reefs were occupied primarily by reef-building corals, turf algae, coralline algae, and other benthic invertebrates (sponges). Coral reefs exhibited a generalized zonation pattern with elkhorn coral (*Acropora palmata*) forming large, monospecific stands in the reef crest and shallow fore reef (0-5 m depth); stands of staghorn coral (*A. cervicornis*) at intermediate depths (5-25 m depth) on wave exposed reefs and in shallow, protected environments; massive corals (dominated by *Montastraea annularis* complex) throughout the fore reef (5-30 m depth) and in back reef and lagoonal areas; and plating agaricids near the base of the reef (20-40 m depth) (Goreau, 1959; Adey, 1978). Caribbean reefs have experienced significant losses in living coral cover over the last three decades and "classic" zonation patterns have disappeared from many locations (Gardner et al., 2003). As corals die, exposed benthic substrates are monopolized by fleshy macroalgae, encrusting and bioeroding sponges, and other organisms. These "pest" species are outcompeting and

overgrowing corals, and may prevent new recruitment and regrowth of damaged corals (Hughes, 1994; Aronson and Precht, 2001; Weil 2004).

Coral diseases were first described in the western Atlantic almost 35 years ago (Antonius, 1973), but it wasn't until about ten years ago that diseases were identified as a significant factor accelerating the deterioration of coral reefs (Epstein et al., 1998; Harvell et al., 1999; Green and Bruckner, 2000; Sutherland et al., 2004; Weil, 2004). Black band disease (BBD; Antonius, 1973; Garrett and Ducklow, 1975; Antonius, 1973), white band disease (WBD; Gladfelter et al., 1977) and white plague (WP type I; Dustan, 1977) were first observed in the 1970s from reefs of Belize, Florida, Bermuda, Puerto Rico and the USVI and have become chronic afflictions of important reef-building corals. Although generally low in prevalence and patchy in distribution, these diseases have persisted on the same reefs for many years, and have spread throughout the western Atlantic infecting a growing number of host species (Gladfelter, 1982; Rützler et al., 1983; Dustan, 1987; Peters, 1984; Edmunds, 1991; Kuta and Richardson, 1996; Aronson and Precht, 1997; Bruckner et al., 1997). The earliest report of significant coral mortality from disease was from the Florida Keys (USA), where an outbreak of WP spread through *Mycetophyllia* spp. and *Colpophyllia* spp. populations, and was predicted to cause the disappearance of *M. ferox* from some locations (Dustan, 1977). Ten years later a second outbreak of WP affected *M. annularis* and 11 other species. Large numbers intact dead skeletons of *M. ferox* and other species were found on the fore reef, although numerous healthy, unaffected colonies were still apparent (Dustan and Halas, 1987), highlighting extensive losses of corals from disease as well as the resilience of these species during the 1980s. WBD played a dominant role in the precipitous (90-98%) decline of *A. cervicornis* and *A. palmata* populations during the 1970s and 1980s (Bruckner, 2002; Aronson and Precht, 2001; Gardner et al., 2003). It is the only disease to date that has caused major changes in composition and structure of reefs over large areas of the Caribbean (Williams et al., 1999; Green and Bruckner, 2000).

Since the mid 1990s, there has been a rapid proliferation of diseases, including a recent emergence of new syndromes (Sutherland et al., 2004; Weil, 2004; Weil et al., 2006). Over 30 diseases have been reported from the Caribbean (Table 1). Some of these affect a single species in specific localities, while others have a widespread geographic distributions and wide host ranges (Weil, 2004). Epizootic events have been associated with six diseases [WP-II, BBD, WBD-I, yellow band disease (YBD), white pox (WPX) dark spot disease (DSD) and Aspergillosis (ASP)], and three diseases (WP-II, YBD and ASP) are currently causing extensive mortality throughout the region (Bruckner, 2002; Weil, 2004). In addition to the region-wide decline of acroporids, there has also been a notable degradation of massive reef-framework corals (in particular the *Montastraea annularis* complex). Declining health of *M. annularis* (complex) has been associated with WP-II epizootics of increasing severity, bleaching events (1995, 1998, 2005), YBD, BBD, DSD, and parrotfish predation (Cervino et al., 1997; Bruckner and Bruckner, 2000; Nugues, 2002; Miller et al., 2003; Jordán-Dahlgren and Rodríguez-Martínez; Weil, 2004; Bruckner and Bruckner, 2006). The recent emergence of diseases in the wider Caribbean appears to be an unprecedented event over a millennial time scale (i.e. >3800 yr) (Aronson and Precht, 2001).

While disease has undoubtedly played a major role in shaping the structure and ecology of Caribbean reefs over the past few decades, very little is known about many of

the fundamental aspects of coral diseases, such as their causes, how diseases are transmitted, factors conferring resistance and resilience, and the long-term effects of diseases on coral populations and coral reef ecosystems (Woodley et al., 2003). Researchers have made significant advances in coral disease research through the application of new laboratory tools and more detailed epizootiological studies (e.g., Edmunds, 1991; Bythell et al., 1993; Carlton and Richardson, 1995; Kuta and Richardson, 1996; Bruckner et al., 1997; Richardson et al., 1998; Bruckner, 2002; Downs et al., 2005). Unfortunately, large gaps remain in our understanding of coral disease, and few strategies and tools have been provided to resource managers to assist in the management of diseases and mitigation of disease impacts. Due to a growing number of disease reports, and an absence of standardized criteria for naming diseases, much confusion surrounds many of the newly emerging diseases. In some cases, scientists do not have the appropriate diagnostic tools to characterize disease outbreaks, and resulting analyses may be inconclusive or incomplete. Disease studies are being undertaken without the use of standardized investigative methodology, making it difficult to consistently characterize these events and draw comparisons between disease outbreaks in different locations. Other factors limiting progress include 1) a lack of standardized nomenclature and diagnostic tools, 2) conflicting reports on causative agents and their sources, 3) insufficient data to conclusively identify linkages between disease and environmental stressors. Finally, standard operating procedures for sampling, approaches to prevent contamination/dispersal of diseases, and other strategies to minimize environmental impacts have not been widely applied in the Caribbean.

This paper reviews recent progress in coral disease research and summarizes our current understanding of the major diseases that have impacted Caribbean coral reefs over the last several decades. Topics discussed include 1) number and variety of diseases, 2) host ranges and geographic distribution, 3) prevalence and impact, 4) causes, and 5) role of environmental factors. An effort is made to identify gaps in knowledge, factors hindering progress, and contentious issues surrounding specific diseases from the western Atlantic. It is hoped that the information and lessons learned in the Caribbean can be applied to Pacific coral disease studies.

How many diseases are there?

Although over 30 coral diseases have been reported from the wider Caribbean since 1972 (Appendix I and II), only a handful of these syndromes (e.g., BBD, WBD, WP, YBD, DSD, WPX, ASP, and various skeletal abnormalities) have been observed throughout much of the Caribbean and certain aspects of their etiology and ecology have been characterized. Much confusion surrounds many of the other described syndromes, and few data are available on their distribution and abundance, impact, or cause. Several diseases have been subdivided into different "types" based on highly variable features (e.g., a zone of bleached tissue; differences in rates of movement, species affected). These characteristics often differ temporally and spatially, and may be unreliable, unless affected corals are tagged and followed over time. Syndromes have also been identified with limited etiological and ecological observations. These may lack a unique description of gross signs, disease signs differ among publications, or conditions were named on the basis of a single observation (Appendix II). Unsubstantiated causes or

agents have been proposed for several new syndromes, and information on prevalence, patterns of spread, or evidence of tissue mortality may be lacking. One of the difficulties in standardizing nomenclature used for coral diseases is that disease signs manifest on corals in a limited number of ways, but they may be caused by different pathogens or unrelated abiotic factors.

The major syndromes reported in the literature from the wider Caribbean are combined here into six major categories, based on similarities of gross field signs (Appendix I). This includes: white syndromes, cyanobacterial mat diseases, tissue discoloration, abnormal skeletal development, skeletal damage, and gorgonian syndromes.

White syndromes

There has been a proliferation of names for coral diseases with virtually identical visible signs that reflect the pattern of loss of coral tissue and exposure of skeleton. All of these syndromes are characterized by a sharp, distinct line between apparently healthy coral tissue and freshly exposed skeleton, with no obvious microbial mat present at the disease interface. These conditions have been differentiated based on 1) species affected; 2) presence of a zone of bleached tissue at the disease boundary; 3) rates of tissue loss; 4) location of lesion on colony surface; and 5) patterns of spread. Without microbial analysis of these diseases (or use of a molecular probe to confirm proposed causative agents), it is difficult to verify that WBD, various forms of WP, and other white syndromes are in fact distinct diseases, since disease signs are so similar (Appendix III; Bythell et al., 2004).

Many studies have used various terms interchangeably to describe white syndromes, making it difficult to characterize regional patterns of disease occurrence. For example, Antonius (1977; 1981) identified 11 species of massive and plating species with WBD, while other researchers report this as WP (Dustan, 1977), and only use the term WBD for *Acropora* spp. (Aronson and Precht, 2001). Dustan (1977) suggested that WP represents a suite of diseases that result in the death of coral tissue, but he does not provide a detailed diagnosis of the macroscopic field signs. He characterized WP as "*lesions on the colony that expanded at a rate of a few mm per day and often resulted in whole colony mortality"*, but presents little information on location of lesions or patterns of progression. An outbreak of a disease with signs that are similar to Dustan's "plague" (Dustan, 1977) was observed in 1995 on the same reefs (Richardson et al., 1998a). This condition was designated WP type II because of 1) a faster rate of progression (up to 2 cm/day); 2) highest prevalence on *Dichocoenia stokesi*, a species unaffected during the original WP outbreaks; and 3) a unique pattern of tissue loss. WP-II progresses from the entire base of the colony to the apex, while WP- I lesions occur more variably across the colony (Richardson et al., 1998b).

Richardson et al. (2001) also reported a third type of plague, *WP type III*, which was characterized by more rapid rates of progression (up to 10 cm/day) and a different pattern of tissue loss. Unlike WP-II, lesions start in the center of colonies and radiate out, and only the largest colonies of *M. annularis* (complex) and *Colpophyllia natans* are affected. White plague has also been used to describe tissue loss characterized by random patches of denuded skeleton that extend sporadically and do not give rise to a graded

algal community (Bythell et al., 2002). Peters (1984) coined the term "stress-related necrosis" for another similar condition characterized by sloughing of degenerating tissue, with no obvious discernable pathogens at the disease line.

Since the mid 1990s, several white syndromes with unique diagnostic features have been reported on A. *palmata,* including WPX, patchy necrosis, necrotic patch syndrome, and other unnamed conditions (Ritchie and Smith, 1998; Bruckner and Bruckner, 1996; Patterson et al., 2002; Sutherland et al., 2004; Jordán-Dahlgren and Rodríguez-Martínez, 2004). While some authors have suggested that these syndromes are synonymous (Sutherland and Ritchie, 2004; Sutherland et al., 2004), a pathogen has been identified only for colonies identified with WPX in the FKNMS (Sutherland et al., 2002), and descriptions and photographs of these conditions are highly variable. In some cases, WPX has been described as circular, dime-sized lesions, while other descriptions suggest these are more irregular in shape. In addition, Weil (2003, 2004) reported "patchy necrosis" on *A. palmata* colonies during doldrums-like conditions, and later indicated that these were associated with parrotfish and sea cucumber fecal matter.

Only two diseases have been identified in *A. cervicornis*, WBD type I and WBD type II. Published descriptions of WBD from *A. cervicornis* have far less detail on the pattern and rates of progression than reports of WBD on *A. palmata*. For example, WBD- II can be confused with both bleaching and WBD-I. Affected colonies have a receding margin that progresses at a faster rate than WBD-I, and tissue loss is preceded by a band of bleached tissue up to 20 cm in width. The bleaching margin may arrest, however, allowing the "peeling" margin to catch up to the pigmented tissue (Ritchie and Smith, 1998). Without the presence of a bleached margin, the disease is indistinguishable from WBD type I. Williams and Miller (2005) reported an outbreak of disease affecting *A. cervicornis* in Florida. Unlike WBD, tissue loss was characterized by rapid tissue sloughing from multifocal lesions and no bleaching was noted. They termed this condition *rapid tissue loss*.

Further complicating distinctions between various white syndromes, scars from predation can be difficult to differentiate from diseases, especially when predators are cryptic (e.g., *Coralliophila abbreviata*) or nocturnal (*Hermodice carunculata*). Predators frequently feed on degrading tissue associated with disease lesions; fireworms and corallivorous snails often occur on colonies with BBD, WP, WBD and other white syndromes, and they may also serve as vectors for disease (Bruckner, 2002; 2003; Williams and Miller, 2005).

Cyanobacterial mat diseases

A number of diseases are associated with cyanobacteria. These often exhibit a similar identifiable group of signs on the coral and consistent anatomical alterations that are visible in the field, making it difficult to separate these conditions without laboratory confirmation of the specific cyanobacteria present. Affected colonies have a distinctive visible microbial assemblage that forms an advancing band or mat, separating denuded coral skeleton from living tissue. The mat is usually dominated by one or several cyanobacterial species, although the species of the dominant cyanobacterium may vary between large geographic regions, even in the same presumed disease (Cooney et al. 2002; Frias-Lopez et al. 2002, 2003). The mat may appear black, brown or reddish depending on light levels, the species of cyanobacterium, and its complement of

photosynthesis pigments. This includes diseases referred to as BBD and red band disease (RBD type I and RBD-II; Appendix I; III).

Tissue discoloration

Corals exhibit wide variations in color depending on species, genotypes, clade of zooxanthellae and/or type and concentration of algal pigments, and in response to physical and environmental factors. Individual colonies may show changes in coloration due to a partial loss of zooxanthellae or their photosynthetic pigments (bleaching), host responses to irritants and injuries, or from coral diseases.

YBD is characterized by lightening of tissue. Affected colonies have small circular blotches of pale yellow tissue surrounded by normally pigmented tissue. These lesions expand in size over time, with central areas dying and becoming colonized by algae. YBD lesions can be confused with bleaching, and during bleaching events it may be difficult to determine which corals are affected by YBD (Cervino et al., 2001).

DSD is characterized by darker than normal coloration. Colonies may have one or more small, round spots or patches of darkened tissue (and discolored skeleton in some cases) that grow in size over time. The spots may be associated with a depression in the coral surface, and spots may expand into a ring surrounding dead coral. Weil (2004) recently divided DSD into DSD type I and DSD-II, and also identified three other similar syndromes, dark band disease (DBD), purple band syndrome (PBS) and tissue necrosis. DSD-II is similar to DSD-I, except the "spots" were larger and a thin, necrotic tissue line was apparent at the margin. PBS differs from DSD in that colonies of *S. siderea* have a band of discolored tissue that advances from the outer margin to the inside. It is unknown whether these are different diseases, or are later stages in the progression of DSD, as a causative agent has not been identified and few studies have followed the progression of DSD lesions over time. Some researchers consider these conditions the same as DSD (Gil-Agudelo et al., 2004; Appendix I and III).

Abnormal skeletal growths

Coral colonies often exhibit distinct circumscribed lesions on the surface of a coral, composed of the corals tissue and skeleton. These structures are typically raised spherical to irregular masses that project above the surrounding corallum. They can be subdivided into three categories on scleractinian corals: a) a proliferation of all cell types that may be atrophied or normal in appearance (gigantism, area of accelerated growth, hyperplasm, growth anomaly); b) white, globular masses with few discernable polyp structures and a reduction or absence of zooxanthellae (tumor, neoplasm, calicoblastic epithelioma); and c) chaotic polyp development (Peters et al., 1986; Appendix I and Table 1). Hyperplasms do not appear to cause significant damage to the colonies, while neoplasms damage affected areas, leaves them more susceptible to invasion by boring organism, and destroys normal polyps and their functions (e.g., reproduction).

Skeletal damage or erosion

Damage to scleractinian corals associated with the disruption of septa or calices, or complete loss of the upper skeletal layers may be the result of physical injuries (e.g., abrasions during storms, anchor damage, fin damage), various biotic interactions (predation by fishes, sponge bioerosion, aggressive interactions among corals) and certain

coral diseases. Loss of corallites has been reported for WPX lesions, but the skeleton remains intact (Sutherland, 2002). Skeletal damage has also been reported in WBD-II (Ritchie and Smith, 1998). In skeletal eroding band (SEB), ciliates create a distinct black band adjacent to living tissue. The ciliates secrete a lorica (their "house"), which is embedded in the coral's skeleton and can completely destroy the surface layer of the skeleton (Antonius, 1999). The ciliates form a distinct black to grey band at the margin between exposed skeleton and live tissue, which may be confused with BBD.

Rapid wasting disease (RWD) and ridge mortality disease (RMD) were reported as coral diseases (Abbott, 1979; Cervino et al., 1997; Goreau et al., 1998), but have since been found to be associated with predation by fishes and the formation of territorial algal lawns (Bruckner and Bruckner; 1998; 2000; Borneman, 2005). RWD was characterized as irregularly-shaped white lesions denuded of tissue with the uppermost layers of the skeleton etched away; the exposed limestone was unusually brittle and crumbly (Cervino et al., 1997). Filamentous fungal hyphae covered and were invading epidermal cells, and were proposed as the causative agent. Detailed visual and photographic observations and experimental manipulations demonstrated that RWD is caused by focused biting by the stoplight parrotfish *Sparisoma viride* (Bruckner and Bruckner, 2000), a phenomena that was documented over 100 years ago. The RWD researchers recently identified a fungus in the mouth and fecal matter of *S. viride*, and proposed that parrotfish were a vector for RWD (Richardson, 2000). While linkages between a fish and a fungal pathogen have not been conclusively verified, the major damage to affected colonies has been shown to result directly from predation: 1) lesions advance only during daylight; 2) no further tissue or skeletal loss occurs once the parrotfish were excluded; and 3) lesions rapidly heal in absence of further biting (Bruckner and Bruckner, 2000).

Ridge mortality disease is associated with the loss of tissue and skeletal structures along elevated ridges of brain corals, with tissue remaining in the valleys (Abbott, 1979). Lesions initiate at a single point within the colony surface (or at the margin) and expand outward, following the meanders of the colony. The ridges typically are not completely destroyed; skeletal damage is largely restricted to the loss of septa. This condition is associated with the development and expansion of *Stegastes planifrons* algal lawns (Bruckner, 2002, 2003; Borneman, 2005). However, it is unclear whether fish bites are the sole cause of tissue loss. Biting may cause a stress response that triggers tissue sloughing, or the fish may introduce a pathogen that causes advancing tissue loss. Interestingly, only ridges are affected, and tissue remains in the valleys (and around polyp mouths) until it is overwhelmed by algae. Similar biting by damselfish can create multiple focal lesions that affect individual circular polyps (on *M. annularis* and *S. siderea*), or result in the development of "chimneys" in acroporids (as the coral attempts to contain the algae).

Gorgonian syndromes

Gorgonians have been observed with BBD, RBD, Aspergillosis, predation by gastropods and polychaetes, tumors, and other conditions (Morse et al., 1977; Rützler et al., 1983; Nagelkerken et al., 1997). Abnormal growths often develop on the branches of gorgonian corals (algal tumor, algal gall, or nodules) in response to endolithic algae, fungi and other epibionts (Morse et al., 1977). These are hard concretions of fibers of gorgonin that form spherical or irregular, but may be irregular shaped masses. They are

located predominantly at the axial bases of the colony, but often extend the overall length of the colony.

Aspergillosis is an extremely virulent fungal disease that affects sea fans and branching gorgonians, causing tissue loss and destruction of the skeleton (Nagelkerken et al., 1997). ASP is characterized by degradation and recession of coenenchyme, purpling of adjacent tissue, production of galls, and secondary colonization of exposed axial skeleton. Field identification of ASP may be difficult, as similar lesions, purpling of tissue and nodules, and skeletal and tissue loss occur in response to predation, abrasions, algal interactions and other factors. Confirmation of ASP requires identification of *A. sydowii*, which may not be visible without microscopy.

What corals are affected by disease and what are the impacts?

Records in the Global Coral Disease Database (GCDD) show that 33 species of stony corals, 8 gorgonians and two hydrozoans are affected by at least one disease. This list may not be comprehensive, as Weil (2004) indicates that at least 41 scleractinian corals have been affected by diseases. Some of these differences may be related to the taxonomy used (i.e. whether species are combined or split), or may reflect unpublished observations. Some conditions show geographic variability in occurrence, and susceptibility may vary among species for the different "types" reported (Richardson and Aronson, 2002; Weil 2004). *Montastraea annularis* (complex) is currently most severely impacted by coral diseases, being susceptible to at least eight syndromes (Weil, 2004), with single colonies showing signs of two or more diseases simultaneously (Bruckner and Bruckner, 2006). White plague (type I and II), the most virulent disease observed to date, affects 39 scleractinian corals (Weil, 2004). BBD has been reported on 25 scleractinian corals, 6 branching gorgonians and sea fans (Green and Bruckner, 2000). YBD primarily affects *M. annularis* complex, although other faviids, *A. agaricites*, and *P. astreoides* are reported with this condition (Gil-Agudelo et al., 2004). The various dark spot/band diseases (Appendix I) collectively affect 14 species (Gil-Agudelo et al., 2004; Weil, 2004). Skeletal anomalies affect at least 16 Caribbean scleractinian corals, one hydrozoan and five gorgonians, with acroporids being most susceptible to neoplasms, and faviids commonly exhibiting hyperplasms (Sutherland et al., 2004). Susceptibility of the major genera of reef building corals to the 7 most significant scleractinian coral diseases are shown in Fig. 1.

Table 1. Existing descriptions of gross signs of the primary Caribbean diseases

Syndrome	Diagnostics	Reference
WP type I	Lesions that expand at a rate of a few mm/day and often result in colony mortality. Lesions occur more variably across the colony surface including the edges and sides. Edges of lesions show a sharp boundary between apparently healthy tissue and freshly exposed skeleton with no build up of microorganisms or necrotic tissue visible to the naked eye. Mean rate of loss= 3 mm/day	Dustan 1977
WP type II	Freshly exposed coral skeleton with a sharp line between skeleton and apparently healthy coral tissue. No evident microbial community; a narrow (2-3 mm) zone of bleached tissue at the disease line. No skeletal damage. Similar to type I but faster progression and lesions always start at the base of the colony and advances to the apex. Max loss=2cm/day	Richardson et al., 1998
WP Type III	Lesions start in the center of the colony and expand outward. Tissue loss occurs as large patches on the sides of large (>2m) colonies. Rates of loss can exceed 10 cm/day	Richardson et al., 2001
WBD	A distinct band of white exposed skeleton separates live tissue and algal colonized dead coral. Tissue adjacent to lesion may appear healthy or form a narrow band of disintegrating coral tissue that is peeling off the skeleton. Tissue mortality starts near the base of a colony and where branches furcated, advancing towards branch tips at a rate of 1-21 mm/day (mean = 5.5 mm/day)[2]; it sometimes but not always encircles the entire branch. .No skeletal damage. The width of exposed skeleton varies depending on spreading rates, with older areas becoming progressively colonized by filamentous, turf, macro and coralline algae.	Gladfelter, 1982 [2]Davis et al., 1986
WBD II	A distinct band of white exposed skeleton separates live tissue and algal colonized dead coral. The lesion boundary is preceded by a 2-20 cm band of bleached tissue. The advancing lesion may "catch up" to the bleached margin, making the disease indistinguishable from WBD I. The disease progresses from the base to the branch tips, but can also progress from branch tips towards the base. Some dissolution of the skeleton may occur. Advance of up to 10 cm per day.	Ritchie and Smith,1998
WPX	Irregularly shaped distinct white patches devoid of coral tissue. Most lesions are small but can be > 80 cm^2 and can develop simultaneously on all surfaces of the coral colony. Lesions can merge, resulting in tissue loss that spans the entire colony. The denuded coral skeleton remains intact, but loss of corallites is common. Lesions enlarge along the perimeter at a rate of 2.5 (max= 10.5) cm^2/d	Patterson et al., 2002; Sutherland and Ritchie, 2004
BBD	Black mat/band on the surface of the coral that separates healthy tissue and white, tissue-denuded skeleton. The band consists of a microbial community (black, chocolate brown or reddish rust colored with white filaments) a few mms to cms in width, and 1 mm thick. The width of exposed skeleton varies with spreading rates, with older areas progressively colonized by algae. BBD progresses from single point (at the margin of the colony, or within the colony surface at the interface between previously killed skeleton and live tissue) and radiates outward in a circular or semicircular pattern The microbial consortium functions together to generate and maintain an environment of anoxia adjacent to living coral tissue, possibly causing tissue necrosis through a lack of oxygen and exposure to hydrogen sulfide. Tissue loss of up to 1 cm/day (mean=3 mm/day)	Rützler and Santavy 1983, Edmunds 1991, Richardson et al. 1997; Bruckner et al., 1997.
RBD-II	Small (2-3 cm) lesions on scleractinian coral colonies. Cyanobacterial filaments form a loose biofilm, or matrix, that is spread out over the lesion and onto adjacent healthy coral tissue during the day. At night, the filaments contract to form a tightly compacted band less than 1 mm wide that is closely associated with the edges of the lesion at the interface with apparently healthy coral tissue. Tissue loss= 1 mm/day	Richardson, 1992
RBD-I	A band or mat of filamentous cyanobacteria, 0.5-2.5 cm wide, that separate coral skeleton from live tissue; the band moves typically from the base to the tips.	Rützler et al., 1983

Syndrome	Diagnostics	Reference
YBD	Small circular area (s) of translucent light yellow tissue surrounded by fully pigmented tissue, or a narrow band of pale tissue at the colony margin that slowly expands in size. Tissue first affected (e.g., in the center of the blotch) gradually darkens and dies and these areas become colonized by algae. Recently exposed white skeleton may be absent or confined to small (<2 cm) irregular patches within the yellow band. Lesions expand 0.3-2 cm per month; multiple lesions appear on individual corals; these coalesce and continue spreading. Colonies show YBD signs for multiple years.	Bruckner and Bruckner, 2006
DSD	Small, round, dark spots that grow in size over time. Affected tissue may be depressed relative to the rest of the coral. Spots may expand into a ring surrounding dead coral[1] Rates of tissue loss for *S. siderea* were 0.51 cm^2/month and 1.33 cm^2/month for *M. annularis*[2]	[1]Garzón-Ferreira and Gil-Agudelo, 1998;
DSD-II	Similar to DSD-I except the "spots" were larger and may cover 90 % of the colony; Dark but healthy looking tissue up to 45 cm (diameter) associated with depressed skeletal areas. Darkened tissue eventually dies. Dead areas are usually associated with thin, necrotic tissue at the margin. The syndrome advances slowly but faster than DSS-I.	Weil 2004
DBS	Round or elongated bands of pale to dark, live tissue that is sometimes associated with depressed skeletal areas in the corallum. Starts as spots and develops into wide dark bands (1 – 2 cm) of ill-looking tissue. The bands advance from areas in the center or side of the colony towards the edge of the colony in most cases. In *S. siderea*, the syndrome advances from the edge to the center of the colony, or from one side to the other. Rates of advance are faster than those in DSS-I and DSS-II.	Weil 2004
PBS	Colonies have several purple spots over the surface, or a purple band that develops at the edge and moves to the center, leaving clean skeleton behind that is quickly colonized by turf algae. The width of the purple band is variable, but generally over 1 cm. The rate of advance is approx. 1 cm/month	Weil 2004
Tissue Necrosis	Similar to PBS, except colonies lack spots, band is wider and more irregular and tissue looks necrotic and peels off skeleton	Weil 2004
Calicoblastic Neoplasm	Raised (up to 1 cm) whitened, irregularly shaped lumps on upper or lower branch surfaces in any region of the coral colony, sometimes extending from one side of the branch to the other. Normal polyps absent from the center of the mass. Coenosteal skeletal material spreading upward between polyps at the margins, or over polyps toward the middle; coral tissue at the edges appears slightly swollen, ruffled, and lifted above the skeleton. Mean growth rate of 0.12 mm/d	Peters et al., 1986
Hyperplasm	Protuberant circular to ovoid lesions with enlarged skeletal elements relative to adjacent surfaces. Normal tissue features such as polyps and tentacles are present but enlarged. Pattern of the polyps, valleys, ridges may differ from the rest of the colony but tissue is usually similar in coloration to the rest of the colony.	Peters et al., 1986

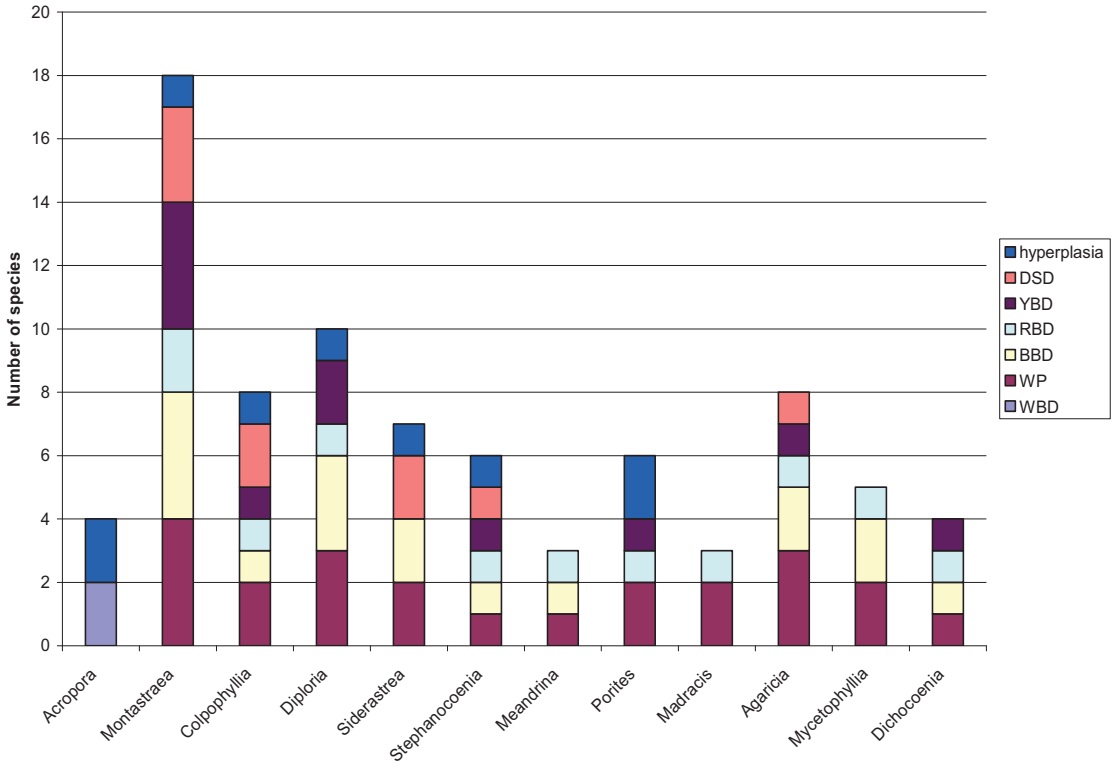

Fig. 1. Number of diseases affecting different genera of western Atlantic scleractinian corals.

The average prevalence of coral diseases at the community level is generally low, although it is highly variable between and within sites, during different years, and seasonally. Outbreaks of BBD, WP and WBD were identified during the 1970s and 1980s in Florida, Belize, Jamaica, USVI, Netherlands Antilles (Dustan, 1977; Rützler et al., 1983; Peters 1984; Rogers, 1985). In some locations, these diseases affected 50-80% or more of the corals, although outbreaks were largely confined to specific reefs or zones (e.g., the palmata zone). Over the last ten years, there has been an increase in monitoring efforts targeted at disease, and many of these report an increase in the prevalence and incidence of disease throughout the region over time, including outbreaks of WP-II, YBD, WPX and other emerging acroporids syndromes (Porter et al., 2001; Nugues, 2002; Miller et al., 2003; Weil, 2004; Bruckner and Bruckner, 2006). In one of the first Caribbean-wide surveys, prevalence rates in 9 locations ranged from 0.9% in Bermuda to 16% in Jamaica during 1999, with a higher number of diseased gorgonians (compared to scleractinian corals) and a significant increase in prevalence between 1999-2001 in Bermuda (4.3% in 2001), Puerto Rico (from 2.1% in1999 to 6.6% in 2001) and Venezuela (3% to 5.8%) (Weil, 2004). In contrast, more recent studies near Lee Stocking Island, Bahamas found very low levels of disease (<0.7% in 2002 and 2003; Voss and Richardson, 2006).

White band disease and other syndromes affecting acroporids

WBD was first documented on reefs around St. Croix, USVI, and later throughout the Caribbean. During the 1970s and 1980s, the prevalence of WBD varied from 1-2% to 26% in the British Virgin Islands (Davis et al., 1986), 33% in Parguera, Puerto Rico (Goenaga and Boulon, 1992), 40% in Florida and Belize (Antonius, 1981), 64% in the USVI (Gladfelter et al., 1977) and as high as 80% in Jamaica and the Netherlands Antilles (Rogers, 1985). Epizootics of WBD significantly reduced populations of acroporids throughout the region.

Although WBD was reported from 9 countries in the last ten years, most observations of WBD on *A. palmata* are for isolated colonies, with only one outbreak reported (Mona Island, Puerto Rico; Bruckner and Bruckner, 2005). In contrast, recent outbreaks of a more virulent disease (possibly a form of WBD, WBD-II, or some other syndrome) have been noted among *A. cervicornis* in Puerto Rico, Bahamas, Florida and other locations (Weil, 2004; Williams and Miller, 2005). Other conditions (WPX and other emerging syndromes) are also being observed more frequently on A. *palmata* since 1994. These are causing much more rapid rates of tissue loss (cm/day) than that reported for WBD in the 1970s and 1980s (5 mm/day). For instance, WPX lesions expand radially at their perimeters at an average rate of 2.5 cm^2/day and individual lesions may be greater than 80 cm^2 in area; lesions can develop simultaneously on all surfaces, and individual lesions often coalesce, resulting in tissue loss that spans the entire colony (Sutherland and Ritchie, 2004). In the Florida Keys populations of *A. palmata* sustained losses averaging 88% between 1996-2002, which was attributed primarily to WPX (Porter et al., 2001; Sutherland et al., 2004).

Black band disease

BBD is widely distributed throughout the Caribbean, with reports from 25 countries. BBD generally affects a low percentage of corals (<1%) at the community

level (Edmunds, 1991; Kuta and Richardson, 1996), but it occurs in most reef environments, and localized epizootics have been observed in the USVI, Jamaica, Florida and Puerto Rico (Peters, 1984; Bruckner and Bruckner, 1997; Bruckner, 1999 Bruckner, 2002). The disease may exhibit a clumped distribution (Kuta and Richardson, 1996; Bruckner et al., 1997), affecting up to 10 corals within a 2 m radius area (Peters, 1984). A greater percent of the corals may be affected by BBD in areas with high coral cover, and in habitats with a high density of colonies or dominance by susceptible species. For example, prevalence of BBD in St. John was low at the community level (0.2%), but much higher at the species level (5.5% of the *D. strigosa*); these sites had a high density of colonies, but susceptible species accounted for only 24% of the total coral composition (Edmunds, 1991). Florida reefs examined by Kuta and Richardson (1996) had a low density of corals (0.15 colonies/m^2) and relatively few infections (0.72%). Other sites examined in the Florida Keys ten years earlier (when they had 50-60% cover) had a mean prevalence of BBD of 6% (Dustan, 1987). Bruckner and Bruckner (1997) observed a maximum prevalence of 1.2% in Jamaica, although 5.2% of the corals became infected over 20 months. These sites had an intermediate density of corals (0.9 colonies/m^2), although over 90% of the corals were susceptible to BBD (Bruckner et al., 1997).

BBD typically advances at rates of about 3 mm/day (Rützler et al., 1983), and occasionally increases to a maximum of 1 cm/day (Antonius, 1981). Considerable variation in spreading rates is observed over the duration of individual infections (Rützler et al., 1983) and also between species, depths, seasons and locations (Bruckner, 2002). BBD occurs year round on tropical Caribbean reefs, while infections often disappear in winter months in Florida and other northern reefs, when temperatures decline below 20ºC. BBD can kill small (<50 cm^2) corals in several days while larger corals experience partial mortality before signs of BBD disappear (Bruckner, 2002). However, BBD may reappear later that season or the following year, and individual colonies can be affected by BBD for multiple years (Feingold, 1988; Kuta and Richardson, 1997; Bruckner and Bruckner, 1997). While BBD does not appear to have caused large die-offs of important reef-building corals, individual colonies lose substantial amounts of tissue that may affect their reproductive potential or their ability to resist other stresses (Edmunds, 1991; 2000). Kuta and Richardson (1997) noted that corals continue to lose tissue after signs of BBD disappear. In the USVI, 28 colonies identified with BBD in 1988 were tagged and followed for ten years; 50% of these were subsequently killed during a hurricane or died of unknown causes, 36% died from BBD, and 14% were still alive 10 years later (Edmunds, 2000). Regrowth of corals affected by BBD has been observed (especially in *D. strigosa*), although larger lesions in *M. annularis* and other species fail to completely regenerate (Bruckner, 2002; Weil, 2004).

White plague

White plague was first observed in Key Largo, Florida in the 1975, where it affected up to 7% of *S. siderea* colonies and 24-73% of *M. ferox* colonies at Carysfort Reef (Dustan, 1977). WP was still prevalent in 1984 on Carysfort Reef, and was also documented throughout the entire Key Largo region (inner, middle and outer reefs) at a mean prevalence of about 3.7% (Dustan, 1993). WP caused rates of tissue loss of up to 3.1 mm/day, and was estimated to have killed 20-30% of *M. ferox* population (Dustan 1977, 1987).

A more virulent form of WP (WP-II) was observed on reefs of the middle and northern Florida Keys in 1995. It initially affected 9-38% of *D. stokesi* colonies, and subsequently spread to 16 other species (Richardson et al., 1998a). The epizootic recurred in 1996 in the lower Keys and Dry Tortugas (although sites with outbreaks in 1995 had few infections in 1996) and in 1997 emerged on reefs north of the Florida Keys reef tract (Richardson et al . 1998b).

WP (type I and type II) has continued to spread throughout the region over the last 10 years, with reports from 21 countries. In La Parguera, Puerto Rico, WP first appeared in 1996 on inner middle and shelf edge reefs; it affected 5 genera, but was most severe on one inner reef where 47% of the *D. labyrinthiformis* colonies contracted WP between August and December (Bruckner and Bruckner, 1997). Subsequent outbreaks were observed in Puerto Rico in 1998, 2000, 2002 and 2006 (Bruckner, pers. Obs., Weil 2004; Hernandez, coral list posting).

WP-II first appeared on reefs around St. John, USVI in 1997, and has been since observed on most back reef (< 3m depth), fore reef slope (6-12 m) and deeper offshore habitats (24-32 m). It primarily affects *M. annularis* (over 90% of infections) but also *M. cavernosa*, *C. natans*, *S. siderea* and *Diploria* spp. are susceptible (Miller et al., 2003). New infections were observed every month over 4 years at Tektite Reef, although the incidence varied substantially (3-58%) over the duration of the study and was highest during the first year (Miller et al., 2003). An outbreak occurred on these and other reefs in St. John following the 2005 bleaching event (J. Miller, unpubl obs).

An outbreak of WP was also first observed on reefs near Soufriere, St. Lucia in July 1997 (Nugues, 2002). Eleven percent (range=7-14%) of the six dominant coral species were infected with WP on three reefs in March 1998. Over 8 months, WP was estimated to have killed 6.6% of the living coral tissue at the most affected site, with most of the tissue loss occurring on the two dominant species (*M. faveolata* and *C. natans*) (Nugues, 2002).

Yellow band disease

YBD was first reported from the Florida Keys in 1994 (Cervino et al., 2001), with subsequent observations from 24 countries. The incidence of YBD increased between 1996-2000 in Colombia, Mexico, the Netherland Antilles, Panama, Puerto Rico, Grenada, St. John, Turks and Caicos, USVI and Venezuela, where 18-91% of *M. annularis* (complex) colonies were affected (Santavy et al 1999; Cervino et al 2001; Bruckner and Bruckner 2003; Gil-Agudelo et al. 2004; Jordán-Dahlgren and Rodríguez-Martínez 2004; Bruckner and Bruckner, 2006). YBD currently appears to be most abundant in remote, offshore locations removed from human population centers (Bruckner and Bruckner, 2003; 2006; Weil, 2004).

Rates of mortality from YBD are generally slow (5-15 cm/yr), although colonies with single YBD lesions become infected in multiple locations, and infections can persist for over 5 years (Bruckner and Bruckner, 2006). In both Curacao and Puerto Rico, YBD appeared to preferentially target the larger corals in the population; over several years, live tissue area is progressively reduced in size, with infections persisting until the coral dies (Bruckner and Bruckner, 2006; Bruckner and Bruckner, in press).

Dark spots disease

DSD was first noticed in Colombian reefs in 1992 during a bleaching event (Solano et al., 1993), with the first quantitative study conducted in 1997 (Garzón-Ferriera and Gil, 1998). In this study, DSD affected > 16% of six species (over 1545 colonies); the two most abundant species (*M. annularis* and *S. siderea)* had the highest number of infections (Gil-Agudelo and Garzón-Ferreira, 2001). Cervino et al., (2001) reported prevalence rates of 42-56% for *S. intersepta* and *S. siderea* in Bonaire, Turks and Caicos, and Grenada. Gochfeld et al. (2006) reported a mean prevalence of 31.5% on St. Thomas, USVI, 50.3% on Culebra, Puerto Rico, and up to 80% in the Bahamas for *S. siderea*, with the highest incidence during August and sudden declines each year in October.

Spreading rates of DSD are generally low. In Colombia, loss was 0.51 cm^2/day for *S. siderea* and 1.33 cm^2/month for *M. annularis*. Recovery of lesions was not observed, and signs of the syndrome persisted for several years (Garcés-Baquero, 2000). Cervino et al., (2001) reported rates of tissue die back of 3.99 cm/month for 2 colonies of *S. siderea*. In the Bahamas, Gochfeld et al., (2006) did not observe any colony mortality from DSD over two years; small areas of necrosis were noted, but these areas regenerated relatively rapidly. In addition, dark spots disappeared from affected colonies in October, but they also often reappeared the following year in the same or different location (Gochfeld et al., 2006).

Aspergillosis

Aspergillosis is thought to have emerged in the 1980s along the coast of Costa Rica, Panama and Trinidad (Guzman and Cortes, 1984, Garzón-Ferreira and Zea, 1992, Laydoo, 1983), where it caused localized mass mortalities. Sea fans showing similar signs to the 1980s epizootics were reported from 22 locations throughout the Caribbean in 1995 (Nagelkerken et al., 1997). In the Bahamas, disease incidence and virulence increased between 1995-1998 (Smith and Weil, 2004). By 1999, Aspergillosis was identified on branching gorgonians (*Pseudoterigorgia*, *Plexaura*, *Pseudoplexaura* and *Plexaurella* spp.) in Puerto Rico and the Bahamas. Over the last 5 years ASP infections have been observed in 17 countries.

Prevalence of ASP appears to be greater in protected sites and in deeper areas on exposed reefs (Nagelkerken, 1997). In the Florida Keys, 31% of sea fans were affected in August 1997; declining numbers of infections were observed over the next seven years in all sites, except for three localized outbreaks during the summers of 1998, 2000 and 2002 (Kim and Harvell, 2004). Aspergillosis does not appear to have impacted the abundance of sea fans in Florida, although partial and complete mortality altered the size-class structure of the population by removing the large colonies (Kim and Harvell, 2004).

Table 2. Prevalence, incidence and impact of the major western Atlantic coral diseases.

Disease	Spatial distribution and prevalence	Temporal/ Spatial variations	Impact
WP	7% *of S. siderea*, 24-73% of *M. ferox* in Florida[1]; 20.1% of *D. stokesi* colonies in Florida[2] 11% of all species, 19% *M. faveolata* and 13% *C.natans* in St. Lucia[3]	Highest prevalence in late summer and early fall at temps of 29-30° C [1] In St. Croix, prevalence of 3.1% in clean site and 11.4% in polluted site[5]	20-30% mortality of *M. ferox* populations[1] 0.1-5% tissue loss over 26 months in the USVI during the 1980s [4] 9.4% of affected *D. stokesi* colonies died in 2 months[5]
WBD	2-5%; up to 40% in Florida, USVI, Belize[1]; 5-26% in BVI[2]; up to 64% in USVI[3]; 20-33% in Puerto Rico[4]		Contributed to a regional decline of Caribbean acroporids
WPX	Contagious; Clumped distribution[1], up to 100% of colonies may be affected[2] 35-73% affected in PR[3]	Greatest rate of tissue loss during warm water[1]	Rapid spread within and between reefs during the 1990s; killed 50-80% of elkhorn coral in certain areas in FKNMS[1]Average loss of tissue over 10 days was 17%[3]
BBD	Up to 8% of gorgonians at one time, 13.8% of all colonies over 26 months in Florida[1] 0.2% of all corals; 5.5% of *D. strigosa*[2] 5.2% total over 20 months; max 1.2% at one time[3]	Highest prevalence in summer in shallow locations; disappears in winter in Florida[1] and when temps drop below 27.5 C in the Bahamas[5] Temperature and light affect growth and spreading rates of BBD[3] In USVI higher prevalence (1% vs. 2.7%) in a polluted site [4] Sites with BBD had higher sedimentation rates[5]	Mean rate if tissue loss is 3 mm/day; in St. Croix, rates were 1.45 mm/day[4] 58% of *D. strigosa* colonies lost >75% of their tissue; overall loss of 3.9% of *D. strigosa* tissue per year[2]
YBD	Up to 12.5% *M. annularis* and 7.8% *D. strigosa* affected in Colombia in 1999[1] 35% of *M. annularis* in Mexico during 2001[2] up to 52% in Puerto Rico[3]	Higher spreading rates in summer. Sites affected between 1997-2002 shows a declining trend of new infections; older infections have persisted and new areas are being impacted.	Colonies affected with YBD in 1999 and 2000 lost 60% of their tissue by 2003 and most were still affected by YBD[3]
DSD	16% of six species; [1]; 42-56% of *S. intersepta* and *S. siderea*[2] Incidence on *S. siderea* varied from 81% on the deepest reef in May 2002 to 67% in a shallow site in Jan 2003[3]	Highest incidence July-Oct.; more infections in shallow water [1] In the Bahamas dramatic decrease in October, unrelated to temperature and depth [3]	Tissue loss of 4 cm/month in *S. siderea*[2] No significant loss over two years; small lesions recover[3]
ASP	39% of sea fans in Caribbean in 1995-1996[1]; 31% in Florida in 1997, declining to 5.9% by August 2003[2] In Mexico prevalence declined from 12.9% in 2002 to 5.3% in 2004	Prevalence higher in protected sites and deep water [1]	Sea fan density has remain constant; Keys-wide reduction in height of sea fans (40 cm – 26 cm) and >50% of the tissue area over six years [2]

WP: [1]Dustan, 1977; [2]Richardson, 1995; [3]Nugues, 2002; [4]Bythell et al.,1993; [5] Kaczmarsky et al., 2005.
WBD: [1]Antonius, 1981 [2]Davis et al., 1986 [3]Gladfelter et al., 1977 [4]Goenaga and Boulon, 1992 [5]Rogers, 1985
WPX: [1]Patterson et al., 2002; [2]Sutherland and Ritchie, 2004 [3]Weil, 2004 [4] Kaczmarsky et al., 2005.
BBD: [1] Feingold, 1988. [2]Edmunds, 1991; [3]Rützler et al., 1983 ; [4] Bruckner et al., 1997
YBD: Cervino et al., 2001 Gil-Agudelo et al., 2004; Bruckner and Bruckner 2003; Bruckner and Bruckner, 2006
DSD: [1]Gil-Agudelo and Garzón-Ferreira, 2001; [2]Cervino et al., 2001[3] Gochfeld et al., 2006
ASP: [1]Nagelkerkin et al., 1997; [2]Kim and Harvell, 2004

What is causing these diseases and where are they coming from?

Proving disease causation has been one of the largest challenges in coral disease research. Because diseases manifest on corals in a limited number ways, corals that exhibit specific disease signs in the field may be affected by a variety of pathogens that differ spatially or temporally (e.g., the pathogen for WP-II may differ depending on the location, affected species, or other factors). To date, causative agents have been identified for four of the most common diseases found on Caribbean reefs (BBD, WP-II, ASP, and WPX). Three of these (WP-II, ASP, WPX) were verified through inoculation experiments with cultured bacterial isolates (through fulfillment of Koch's postulate), while the cause of BBD was identified using microscopy. In other diseases (YBD, DSD, WBD-II) screening of microbial communities of healthy and diseased tissue (and mucus layer) using traditional culture methods illustrates a high diversity of microorganisms, along with several bacteria (especially *Vibrio* spp.) that appear to be more prevalent in diseased samples (Cervino et al., 2001; Gil-Agudelo et al., 2004; Weil, 2006) . Molecular studies (16S and 18S rRNA/DNA gene sequence amplification of microbial communities) have identified complex multi-species microbial communities in corals (including microorganism that may be unculturable) that appear to vary spatially, seasonally, between species, and also between diseased and apparently healthy parts of the colony (Rohwer et al., 2002; Pantos et al., 2003; Pantos and Bythell, 2006). In many cases (e.g., WP-II, BBD and WBD) these molecular studies have identified a different suite of organisms as potential causative agents than that observed in earlier studies.

Black band disease

The causative agent of BBD was originally described as the cyanobacteria *Oscillatoria submembranaceae* and then *Phormidium corallyticum* based exclusively on filament morphology, pigmentation and motility determined using light microscopy (Antonius, 1981; Rützler et al., 1983). Other heterotrophic bacteria (Garrett and Ducklow, 1975), the sulfide oxidizing bacterium *Beggiatoa* spp. (Ducklow and Mitchell, 1979) and marine fungi (Ramos-Flores, 1983) have also been suggested as the primary pathogen. Richardson and colleagues described BBD as a consortium of microorganisms dominated by a gliding filamentous cyanobacteria (*P. corallyticum*) that functions together with sulfur oxidizing bacteria (*Beggiatoa* spp.) and sulfur reducing bacteria (*Desulfovibrio* spp.) to produce anoxia and high levels of sulfide adjacent to the coral, conditions that are lethal to the coral (Carlton and Richardson, 1995; Viehman et al., 2006). More recent work using 16S rRNA gene sequencing identified a complex and variable assemblage of heterotrophic organisms that includes over 500 species of bacteria as well as cyanobacteria (Cooney et al. 2002, Frias-Lopez et al. 2002, 2003). These molecular studies identified anomalies in the identification of the cyanobacteria: three unique taxa of cyanobacteria have been isolated, with differences noted between the Caribbean and IndoPacific. Interestingly, *P. corallyticum* was not detected in the clone libraries or evident in the DGGEs (Cooney et al. 2002, Frias-Lopez et al. 2002, 2003).

White plague

The disease pathogen for WP II was identified as a gram negative α-proteobacterium (a new species of *Sphingomonas* later renamed *Aurantimonas*

coralicida; Denner et al., 2003) based on culture of a bacterial isolate obtained from a single diseased *D. stokesi* colony and subsequent inoculation on two healthy colonies of *D. stokesi* (Richardson et al., 1998a). This same microbial pathogen was subsequently reisolated from another affected colony at a later date. Although both strains are in culture, apparently healthy colonies no longer appear to be susceptible to either the original or newly isolated strains of the pathogen. This suggests that bacteria may lose virulence when in culture or *A. coralicida* is not in fact the causative agent. Another study examining a plague-like disease on *M. annularis* colonies from the USVI and Barbados (using bacterial 16s rDNA genes) identified a high diversity of bacteria in diseased samples, and differences between diseased and healthy tissue (Pantos et al., 2003). While *Sphingomonas* spp. was detected in healthy corals and control areas on diseased colonies, it was absent from diseased areas. Instead, an α-proteobacterium most closely related to the causative agent of juvenile oyster disease was present in diseased tissues, but consistently absent from healthy tissue (Pantos et al., 2003).

A fluorescent probe specific for *A. coralicida* has tested positive on colonies with signs of WP-I and WP-II in a number of locations including the USVI, Puerto Rico (Miller et al., 2003; Weil, 2004), suggesting that this bacterium is widely distributed throughout the region.

White band disease

A causative agent for WBD type I has not been identified. Using histology, Peters et al., (1983) identified gram negative rod-shaped bacterial aggregates in the calicoblastic epidermal tissue of *A. palmata* colonies with WBD from the USVI and Bonaire. These bacterial bodies were also found in acroporids without signs of WBD, although the counts of bacteria per area were significantly less (Peters, 1984). Santavy et al., (1995) found bacterial aggregates in some but not all colonies of *A. cervicornis* with WBD on one reef in the Bahamas, while corals on a neighboring reef did not contain bacterial aggregates (Santavy and Peters, 1997). Bacteria were absent from WBD-affected *A. cervicornis* colonies in Florida (Kozlowski, 1996).

Studies of the bacterial community of WBD-II has concentrated on the surface mucopolysaccharide layer and the use of preferential carbon utilization methods. A bacterium most closely related to *Vibrio carchariae* was identified as a possible cause, and these were found to increase in number with the onset of disease WBD II (Ritchie and Smith, 1995).

Microbial communities identified using 16s RNA techniques were found to differ substantially (only 10% similarity) between healthy and WBD *A. palmata* colonies. Healthy and diseased tissues both contained similar proportions of four predominant bacterial groups, although planctomycetes, cyanobacteria and Cytophaga-Flexibacter-Bacteroides group were found only in diseased samples (Pantos and Bythell, 2006).

Yellow band disease

The causative agent for YBD is unknown. Cervino et al. (2001) proposed that YBD is a zooxanthellae disease which can kill coral by damage produced by the symbiont, based on a finding of a reduction of the number of zooxanthellae (41 to 97%) and reduction in the mitotic index of zooxanthellae (2.5% to 0%). Several bacterial

strains metabolically related to the genus *Vibrio* have been found in the mucus associated with diseased tissue (Gil-Agudelo et al., 2004).

Dark spots disease

The causative agent for DSD is not known. Differences in the structure of the microbial community from the mucus of healthy and diseased *M. annularis* and *S. siderea* colonies were identified based on the metabolic profile of culturable bacteria (Gil-Agudelo et al., 2004). A total of 17 groups of bacteria were identified, 16 of which were found in both diseased and healthy tissue, and one (*V. carchariae*) that was only observed in diseased samples. Healthy colonies inoculated with this bacterium failed to manifest signs of DSD (Gil-Agudelo et al., 2004).

Aspergillosis

Perhaps one of the most comprehensive etiologic studies of a coral disease in the Caribbean involves Aspergillosis. The fungus *Aspergillus sydowii* was identified as the cause of this disease through the use of transfection experiments, fungal cultures, morphologic and metabolic characteristics and 18s rDNA gene sequencing (Smith et al., 1998; Alker et al., 2001). *A sydowii* has been isolated from benthic environments near the Orinoco River, and pathogenic strains have also been collected in the USVI during African dust events (Smith and Weil, 2004).

Table 3. Causative agents and associated microorganisms reported for western Atlantic coral diseases.

Disease	Reported causes and associated organisms
WBD	unknown ; gram negative rod-shaped bacterial aggregates in the calicoblastic epidermal tissue[1]; blue green algae, ciliates, turbellarian flatworms, copepods, amphipods, nematodes [2]
WBD II	*Vibrio harveyi/carchariae*[a]
WPX	*Serratia marcescens*[a] , a common gram-negative bacterium classified as a coliform and a member of the Enterobacteriaceae family; fish feces proposed as a vector[2]
WP I	Unknown, Gram negative bacteria
WP II	*Aurantimonas coralicida*[a] , an obligately aerobic,polarly flagellated gram negative bacterium[1]; α-proteobacterium closely related to the causative agent of juvenile oyster disease[2]
BBD	*Oscillatoria submembranopora*[1] The cyanobacteria *Phormidium corallyticum*[2] in association with sulfate reducing bacterium *Desulfovibrio* spp, and sulfide oxidizing bacterium *Beggiatoa*[2] *Oscillatoria* spp and *Trichodesmium tenue*[3] Other cyanobacteria (*Oscillatoria, Spirulina, Lyngbya, Arthrospira* and other *Phormidium* species), pennate diatoms, ciliates, flagellates, and marine fungi occur in the band
RBD-I	Cyanobacteria *Schizothrix mexicana* and *S. calcicola*
RBD-II	Cyanobacteria; Two species of *Oscillatoria* characterized by filaments that are wider than they are long; filaments have two rounded tips
YBD	Undescribed *Vibrio*
DSD	Unknown; Over 250 bacteria were isolated from mucus of healthy and diseased *M. annularis* and *S. siderea* colonies; A bacterium most closely related to *V. carchariae* was isolated from diseased corals.
Tumors	Unknown. May be mutations of the genome or programmative changes in gene expression of the coral cells. The role of environmental parameters (e.g., UV radiation) has not been determined.
ASP[a]	*Aspergillus sydowii*[1] *A. terreus, A. niger* and *A. flavus*[2]

WBD: [1]Peters et al.,1983 [2]Gladfelter et al., 1977 WBD II: Ritchie and Smith, 1998; Weil, 2006
WPX: [1]Patterson et al., 2002. [2] Weil, 2004
WP: Dustan, 1977
WP-II: [1]Denner et al., 2003 [2]Pantos et al., 2003
BBD: [1]Antonius, 1973; [2]Rutzler and Santavy, 1983; Richardson et al., 1995; [3]Cooney et al. 2002, Frias-Lopez et al. 2002, 2003
RBD-I: Santavy and Peters, 1997
RBD-II Richardson, 1993
YBD: [1]Cervino et al., 2004; [2]Gil-Agudelo et al., 2004
DSD : [1]Gil-Agudelo et al., 2004 [2]Jordan-Dahlgren and Rodríguez-Martínez, 2004,
ASP : [1]Smith et al. 1996, Geiser et al. 1998, Alker et al. 2001 [2]Toledo-Hernandez et al., 2004

[a]Causative agent identified through fulfillment of Koch's postulate

Where are these pathogens coming from?

External sources of pathogens that have been proposed include terrestrial runoff (*Aspergillus*), sewage (*Serratia marcescens*), and African dust events (*Aspergillus*). Pathogens may also already be present in the marine environment. Reservoirs of *P. corallyticum* (along with other cyanobacteria) occurred as biofilms on the surface of sediment patches present in depressions on healthy *M. annularis*, *M. cavernosa* and *C. natans* colonies (Richardson, 1996). These may be dispersed via movement of water masses, especially during storms (Bruckner and Bruckner, 1997), and transmitted through various vectors like damselfish, parrotfish, fireworms and coral-eating snails (Williams and Miller, 2005; Aeby and Santavy, 2006).

Bacteria also occur on corals in a non-infectious state. Coral mucus is a rich protein-carbohydrate complex that harbors a diverse community of bacteria and other microbiota, and these communities are known to be distinct from the surrounding water (Rohwer and Kelley, 2004). Bacterial diversity varies between healthy corals, healthy parts of diseased corals, and diseased tissue (Rohwer et al., 2002; Pantos et al., 2003). Environmental changes can affect the physiological equilibrium between bacteria associated with the corals and their hosts, or stimulate the growth of other bacteria (Pantos et al., 2003). The coral-microbe relationship can be disrupted by nutrient and organic carbon loading by overstimulating the growth of these microbes, which may result in coral mortality (Kuntz et al., 2005). Under stressful conditions one or more of these microbes may become virulent or affect the resistance of the host, and subsequently trigger onset of an infectious disease.

Are disease outbreaks associated with changing environmental conditions?

Diseases may be infectious (produced by parasites and pathogens) or non-infectious (genetic mutations, produced by environmental factors). The frequency and severity of infectious diseases may affected by changing environmental conditions (elevated SST, declining water quality), human induced alterations of the marine environment (e.g., input of land-based pollutants; dredging, coastal development), and hurricanes and other natural disturbances. Increased temperatures may cause physiological stress and/or trigger the development of pathogenic agents that otherwise would remain non virulent.

Increased abundance and virulence of at least five diseases (BBD, WPX, DSD, ASP and YBD) has been associated with elevated seawater temperatures, with declines in these conditions reported during winter months (Kuta and Richardson, 2002, Alker et al., 2001; Patterson et al., 2002, Gil-Agudelo and Garzón-Ferreira, 1999 and Weil, 2004). Disease outbreaks may also be more severe during or immediately following bleaching events due to a lower resistance of host corals. Widespread and severe outbreaks of WP-II were observed in Puerto Rico, USVI and the eastern Caribbean following the 2005 Caribbean bleaching events (J. Miller, coral list posting; Weil, 2006). Other natural factors, such as habitat characteristics, composition, cover and abundance of susceptible corals, the amount of macroalgae, and presence/absence of certain key indicator species such as *Diadema* may also influence the occurrence and severity of coral diseases. Patterns of disease distribution obtained from the Global Coral Disease Database have shown that 97% of the areas affected by disease in the Caribbean prior to 2000 correlate

to areas where human activities have medium to high impact (Green and Bruckner, 2000). Despite the contention that deteriorating water quality associated with land-based inputs of pollutants and sediments and other human impacts is linked to disease outbreaks, there is minimal quantitative data to support this hypothesis, and links to specific disturbances are unclear (Bruckner, 2002).

Black band disease

Goreau et al. (1998) reported that BBD often first appeared in polluted areas and infections spread radially outward. They suggest that the abundance of BBD mimics the distribution of human influenced areas, with the largest impacts near sewage outflows and areas of high turbidity. Peters (1993) also noted that BBD prevalence is related to adverse environmental conditions, including warmer than normal temperatures, nutrient loading, increased sedimentation and turbidity, predation, and toxics. In Jamaica, the incidence of BBD progressively increased over 19 months, with the largest increase during or just after a period of unusual rainfall and run-off (Bruckner et al., 1997a). In this study, one species that is generally resistant to BBD (*S. siderea*) exhibited few infections prior to the rainfall event, with a dramatic increase in bleaching, WP and BBD in the second year of the study, corresponding to periods of high rainfall and run-off (Bruckner, et al., 1997).

An extensive, multi-year study evaluating BBD incidence on reefs off southern and western Puerto Rico failed to identify direct relationships between BBD prevalence and poor water quality (Bruckner, 1999). The lowest prevalence of BBD overall was found near Mayaguez and Ponce, which are the most polluted and turbid sites in Puerto Rico due to high sedimentation and nutrification associated with river discharge, agricultural runoff, and direct input of untreated sewage. On a fringing reef off the west coast (Rincon), BBD incidence was highest in spring (May-June) when water clarity was high, with infections disappearing during the rainy period (July-August) when run-off increased and visibility declined, even though temperatures were approaching their annual maxima. High turbidity also appears to limit the spatial distribution of BBD in southwest Puerto Rico (La Parguera). Infections were restricted to shallow water (<8 m depth) on turbid inshore reefs, even though species susceptible to BBD occurred in shallow and deep water, while BBD occurred to depths of 30 m on offshore shelf edge reefs with high water clarity. The disease was also common in remote locations around Mona Island, which is 70 km from the mainland of Puerto Rico and lacks permanent inhabitants, industry, agriculture or river discharge (Bruckner, 1999).

In contrast to Bruckner (1999), Voss and Richardson (2006) reported higher sedimentation rates on sites with BBD in the Bahamas. Some of the differences between this study and Bruckner (1999) may be related to the scale of inputs: sites near LSI Bahamas described as having high rates of sedimentation have relatively low levels of sedimentation and high water clarity, when compared to coastal reefs near Puerto Rico. Kaczmarsky et al., (2005) also observed a significantly higher prevalence of BBD in a site off St. Croix, USVI exposed to sewage discharge, as compared to an ecologically similar location upstream from the pollution effluent.

White plague

WP is reported to be seasonal on some northern reefs (Bahamas and Florida), while infections occur year round in USVI, Puerto Rico, Curacao and other locations (Richardson et al., 1998a; Miller et al., 2003; Weil, 2004; Bruckner and Bruckner, in press) and outbreaks have been observed during the coldest time of the year in the Flower Gardens (Hickerson, coral list posting) and in St. John (J. Miller, pers. Comm.). In St. Croix, USVI, Kaczmarsky et al. (2005) observed a much higher prevalence of WP (11.4% in a site affected by sewage discharge, when compared to a site located upstream from the effluent (3.1%).

White band disease

Few data are available to verify the role of environmental factors on WBD prevalence or severity. Outbreaks of WBD have been reported from throughout the region which has spread through acroporids reefs in both nearshore areas impacted by human settlement as well as remote locations and protected watersheds.

White pox

The prevalence and rates of tissue mortality are greater during warm water months (Sutherland et al., 2002). Sewage effluent is the proposed source of the WPX pathogen (Patterson et al., 2002), but no information is available on the prevalence and/or severity of WPX in polluted versus unpolluted sites.

Yellow band disease

YBD progresses more rapidly during warm water periods (Gil Agudelo et al. 2004). Although nutrient enrichment has been shown to increase the rate of tissue loss from YBD (Bruno et al., 2003), YBD is currently most abundant in remote locations or reefs subjected to low levels of human impact (Bruckner and Bruckner, 2006).

Dark Spots Disease

DSD was found to be more prevalent when water temperatures are over 28ºC and in shallow (<10 m depth) reef habitats in Colombia (Gil-Agudelo et al., 2004). In contrast, Gochfeld et al. (2006) did not find a correlation between DSD and water temperature; infections dramatically declined each year in October (which is just after the warmest time of year) and new infections emerged beginning in January of each year (close to the coldest water temperatures of the year). There also was no relationship with depth and DSD prevalence in the Bahamas. In 2002, the highest prevalence was observed at the deepest site (81.25%), while the highest prevalence was in a shallow (<5 m) site (67%) in January, 2003 (Gochfeld et al., 2006). Nutrient enrichment also did not appear to affect the prevalence or severity of dark spots disease in laboratory studies, although increased nutrients did induce bleaching (Gochfeld et al., 2006).

Aspergillosis

Aspergillus sydowii exhibits maximal growth at 30°C and is less affected by the hosts defenses at 30ºC than at 25ºC (Kim and Harvell, 2004). Nutrient enrichment (Nitrates) was shown to increase the progression of Aspergillosis (Bruno et al., 2003).

What have we learned from Caribbean coral disease research?

Research on coral diseases requires an approach that combines ecological monitoring with biochemistry, molecular biology, histology, toxicology, physical oceanography, ecology, taxonomy and other laboratory and field methods. An interdisciplinary approach is necessary to identify, differentiate and characterize coral diseases and their consequences, and understand relationships among diseases and other biotic and abiotic factors.

- **Epizootiology:** To understand the spatial extent of diseases at local to global scales, large areas of reef must be examined at the same time. Surveys must also be conducted at frequencies that are sufficient to document the duration of the condition, and identify seasonal patterns or chronic effects. Monitoring programs should include size measurements and colony condition (amount of recent and old mortality), and follow individual colonies over time to determine the severity of disease and potential population level impacts. Efforts should be made to standardize monitoring approaches.
- **Relationships between disease outbreaks and environmental factors:** Epizootiological studies must be combined with an examination of climate parameters (e.g., temperature and light levels), water quality measures (levels and types of nutrients and contaminants, turbidity, and rates of sedimentation), and impacts of other natural disturbances (e.g., predator outbreaks and hurricanes).
- **Rapid response program:** A coordinated rapid response to disease outbreaks can allow for the timely recognition, characterization, and reporting of disease outbreaks. This information is necessary so managers can 1) quickly direct resources to additional studies that are needed to identify appropriate management responses; 2) identify possible responses to control or mitigate the outbreak and 3) educate local and regional stakeholders on the condition of the corals.
- **Coral disease diagnostics and nomenclature**. There is a need to review and refine existing terminology and develop an approach for naming new diseases to reduce confusion. Disease nomenclature must include descriptive terminology of the gross signs (visible by an unaided eye, underwater) that could be applied by all scientists conducting epizootiological studies. Lesions should first be categorized as tissue loss, growth anomaly, and/or change in coloration. For each lesion, relevant information on the distribution of the lesions (e.g., focal or multifocal), location on colony, lesion shape, relief, texture, color, and size, and structures affected (e.g. tissue, individual polyps, coenosarc, skeleton) should be included in the morphologic diagnosis. By following affected colonies over time, more detailed information on patterns and rate of spread and extent of tissue loss can be compiled. This field terminology can be further refined once a causative agent is identified.
- **Identification of the causative agents** Molecular approaches should be combined with traditional culture techniques to identify and verify coral associated microbes, including spatial and temporal fluctuations, variations among species, and differences between healthy and diseased corals. Screening must include larger sample sizes (especially when isolating a putative pathogen and testing infectivity of proposed pathogens), multiple species (if they show similar signs), and multiple locations. There is an urgent need for molecular probes that will allow rapid screening of corals.

- **Application of new approaches and tools.** More emphasis needs to be placed on understanding processes and factors that may improve the resistance and resilience of the coral hosts, such as antimicrobial activities, immune responses, and regeneration processes**.** Efforts should also include a cellular diagnostics approach to identify stress and its underlying causes, and identify biomarkers that can characterize the condition of the coral and normal ranges of these biomarkers.

Appendix 1. Diseases reported to affect scleractinian corals and gorgonians on coral reefs in the tropical western Atlantic. The terminology highlighted in bold represents the proposed nomenclature identified at the 2004 CDHC workshop in Madison, Wisconsin.

Condition	Synonyms	Host range	Source
White syndromes			
White band disease (WBD)	White line disease; white death; white plague	*Acropora palmata* *A. cervicornis*	Gladfelter et al., 1977
WBD type II	White band disease	*A. cervicornis*	Ritchie and Smith, 1995; Weil 2004
Plague type I	White plague, white band disease; plague-like[2] Stress-related necrosis[3]	12 species of massive and plating corals	Dustan, 1977; [2]Pantos et al., 2003 [3]Peters, 1984
Plague type II	White plague, white band disease, white line disease	*D. stokesi;* 40 other species of plating and massive corals	Richardson et al., 1995; Weil, 2006
Plague type III	**Plague type II**	large massive corals (*M. faveolata, C. natans*)	Richardson and Aronson, 2001
Shut-down reaction (SDR)	Rapid tissue necrosis (in aquaria)	massive corals, acroporids	Antonius, 1977
White pox (WPX)	**White patch disease; acroporid serratiosis; white pox serratiosis**, patchy necrosis	*A. palmata*	Porter, 1996; Patterson et al., 2002; Sutherland and Ritchie, 2004
Patchy necrosis[1]	**White patch disease**; white pox, necrotic patch syndrome[2]	*A. palmata*	[1]Bruckner and Bruckner, 1997; [2]Rodríguez-Martínez et al., 2001
Cyanobacterial mat diseases			
Black band disease (BBD)	Black line disease	24 scleractinian corals, 1 hydrozoan, 6 gorgonians	Antonius, 1973
Red band disease type I (RBD)	Red band disease	*Gorgonia*, *Colpophyllia, Agaricia*, *Mycetophyllia Stephanocoenia*	Rützler et al., 1983 Santavy and Peters, 1997
RBD type II	Red band disease	*D. stigosa, M. annularis, M. cavernosa S. radians, P. astreoides*	Richardson, 1992

Condition	Synonyms	Host range	Source
Tissue Discoloration			
Yellow band disease (YBD)	Yellow blotch disease; ring bleaching[1], yellow pox disease[2]; yellow band syndrome[3]	*M. annularis complex,* other faviids *; A. agaricites* [4] ; *P. astreoides*[5]	Reeves, 1994;[1]Dustan 1977; [2]Garriet Smith; [3]Foley et al., 2004; [4]Gil Agudelo et al., 2004; [5] Sutherland et al., 2004
Dark spots disease (DSD)	Dark spot disease, dark spot syndrome, Ring disease [2]	*M. annularis* (complex), *S. siderea, S. intersepta* and *Agaricia agaricites*	Gil-Agudelo and Garzón-Ferreira, 2001; [2]Agudelo et al., 2004; Gochfeld et al., 2006
Dark spots disease Type II (DSD- II)	Dark spots disease	*S. intersepta; M. annularis; M. faveolata; M. cavernosa; C. natans; C. amaranthus; S. siderea*	Weil, 2004
Dark band syndrome (DBS)	Dark spots disease	*M. annularis; M. faveolata*	Weil, 2004
Purple band syndrome (PBS)	Dark band syndrome	*S. siderea, S. intersepta*	Weil 2004
Tissue necrosis	Dark spots disease	*M. faveolata*	Weil 2004
Mottling syndrome	bleaching	*C. natans*	Borneman, 2005
Pale ring syndrome	bleaching	*Montastraea, Colpophyllia, Diploria*	Borneman, 2005
Light patch syndrome	bleaching	*D. strigosa*	Borneman, 2005
Bleaching	Blanching	All zooxanthellate corals	
Abnormal growth			
Hyperplasia	**Growth anomaly**, tumors, Gigantism, area of accelerated growth, chaotic polyp development	*Diploria*, *Colpophyllia*, *Montastraea*, *Agaricia*, *Porites, Dichocoenia, Madracis*	Loya et al., 1984
Calicoblastic epithelioma	Growth anomaly, tumor, neoplasm,	*A. palmata*	Peters et al., 1986
Algal tumors	Tumor-like growth, tumor, algal tumor, gorgonin pearl, nodule, galls	*Gorgonia* spp. *Pseudoplexaura; Plexaura*	Morse et al., 1977
Skeletal damage			
Skeletal eroding band	Follicullinid ciliates	10 species: *Dichocoenia, Montastraea, Acropora,*	Croquer et al., 2006; Weil et al., 2006
Rapid wasting disease (RWD)[1] (Rapid wasting syndrome)	parrotfish white spot biting[2]; **parrotfish spot biting; parrotfish focused biting**[3] Rhodotorulosis [4]	*Montastraea* spp., *C. natans*	[1]Cervino et al., 1997; [2]Bruggeman et al., 1994 [3] Bruckner and Bruckner, 2000; [4] Richardson, 2000
Ridge mortality disease (RMD)	Damselfish ridge denuding syndrome[2]	*C. natans, D. strigosa*	Abbott, 1979, [1]Zimmerman 1994 [2]Williams et al., 2000
Tissue and skeletal loss, discoloration of tissue and abnormal growth			
Aspergillosis	Sea fan disease	*Gorgonia* spp.	Nagelkerken et al., 1997

Appendix II. Other abnormal conditions observed infrequently in scleractinian corals and gorgonians in the tropical western Atlantic. Some of these require histological analysis for confirmation (Coccidiosis and Nematopsis spores), while others are unconfirmed syndromes.

Syndrome	**Synonyms**	***Host species***	**Description**	
Coccidiosis	Coccidian infection	8 species: *Agaricia, Dendrogyra, Diploria, Montastraea, Meandrites, Porites*	Parasite infection: Oocysts found in mesentarial filaments; causes loss of zooxanthellae, patchy bleaching and tissue necrosis	Upton and Peters, 1986
Nematopsis spores	Sporozoan (protozoan) infection	*Porites* spp.	Thick walled ovoid ovoid capsules in calicoblastic epithelium	Peters, 1984
Ring syndrome	hyperplasia	*D. labyrinthiformis*	Fast growth of tissue and skeleton at ridge areas produces high, pale and thin ridges over the colony. Tissue inside ridges slowly dies	Weil, 2004
Fire coral fungal disease		*Millepora* spp.	Associated with bleaching	TeStrake et al., 1988
Thin dark line	Blistering necrosis [2]	8 species: *Diploria. Montastraea, Porites, Siderastrea*	Thin dark line at the boundary of living tissue that advances <1 cm/year	Jordan-Dahlgren and Rodriguez-Martinez, 2004; [2]Peters, 1984
White spot syndrome	Spot biting	massive corals	Predation by parrotfish referred to as "spot biting [1]"	Global Coral Reef Alliance ; [1]Bruckner and Bruckner., 2002
Star coral polyp necrosis (SCPN)		*M. cavernosa*	No further information presented	Williams and Bunkley-Williams, 2000

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IV. STATE OF KNOWLEDGE IN THE PACIFIC—WHAT DO WE KNOW AND WHAT HAVE WE LEARNED?

OVERVIEW OF ISSUES UNIQUE TO THE PACIFIC: BIOLOGICAL & SOCIAL PERSPECTIVES

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Pacific Islands

The term "Pacific Islands" in the context of this paper arbitrarily refers to those tropical islands of the central and western Pacific Ocean which support shallow hermatypic coral reefs, but excluding the Hawaiian Archipelago, which is covered in other papers. The tropical Pacific Island nations and territories all support coral reefs and, no doubt, harbor coral diseases, although these have not been scientifically documented in many of the islands. In fact, as part of the U.S. National Action Plan to Conserve Coral Reefs, surveys in 2002 and 2004 of coral reef academic scientists, resource managers, government agencies and NGOs recorded that in the U.S. Pacific islands they perceived "no threat" from coral disease, although American Samoa registered an increase to perception of "moderate threat" in the 2004 survey (Waddell, 2005). This lack of concern partially reflects a lack of information on the status of diseases in many islands. Wilkinson (2004, p. 405) notes that in American Samoa and Micronesia "Coral bleaching and disease were either rare or undocumented in 1994, but are now clearly evident and considered a serious threat to many reefs in the region."

The Pacific island coral reefs range from veneers on newly emergent volcanic islands, to platform-like fringing reefs, to barrier reefs with lagoons, to atolls, and include non-emergent isolated banks.

The islands associated with the reefs vary from large high islands, such as in Fiji and New Caledonia, to small low atoll islands rising less than two meters above sea level, as in the entire nations of Tuvalu and the Republic of the Marshall Islands. Although the availability of land resources has limited human settlement and development in these islands, even some of the small atoll areas have been inhabited continuously for thousands of years. The long term and current native residents of the islands are generally defined as Polynesian, Melanesian and Micronesian, based upon their ancestry, culture and languages and their geographic association within these island archipelagoes.

Similarities to Atlantic Areas

Before European contact, the Atlantic and Pacific coastal residents and islanders (except for those far in the interior of high islands), depended on coral reef resources for survival and managed these resources in a sustainable way. But Western development brought major changes to land use, which impacted on surrounding reefs. As in the Western Atlantic area of coral reefs, the Pacific Islands have had similar political histories of colonialism and plantation agriculture exploitation over the last few centuries. Both areas were colonized by European nations from the Sixteenth Century to the present, although most island states have become independent countries in the decades since World War II.

During the early periods of colonization, diseases, warfare and forced relocation decimated local populations. During the last century, populations have re-established or been replaced by immigrants and are in many cases exceeding the traditional self-sustainable carrying capacities. Economic development bases have been shifting from plantation monoculture agriculture to tourism and, to a limited degree, mining, fisheries, and logging (which is typically not sustainable). Impacts of related coastal pollution are similar world-wide.

Atlantic and Pacific reefs are influenced by similar weathers patterns with the same water temperature ranges, variable annual precipitation from very dry to some of the wettest places on earth and frequent violent tropical storms (hurricanes, typhoons or cyclones). Trans-oceanic wind-driven continental dust may be reaching parts of the North Pacific from Asia, but this is not as apparent as African wind driven pollutants entering the Caribbean.

Differences between the Pacific and Atlantic

Island and Reef Numbers:
Although both oceans have atolls and barrier reefs, the numbers of islands and atolls and the lengths and areas of their reefs are much greater in the Pacific (Wilkinson, 2004). Although they are relatively small, the islands of Micronesia alone number over one thousand. Compared to the few atolls in the Caribbean, the Pacific has over fifty. The barrier reefs of Fiji, New Caledonia, Papua New Guinea and other Pacific islands together exceed the extent of the Mesoamerican Barrier Reef System, without considering the world's largest barrier reef in Australia.

Ocean Size:
Distances across the vast Pacific and between its far-spread reefs dwarf those of the Caribbean. The entire Caribbean, if it could be moved over the Pacific, would be lost between Hawaii and Guam without even overlapping the larger and broader tropical Pacific waters south of the equator.

Fisheries:
These great expanses of the Pacific support industrial fisheries fleets supplying over one third of the annual worldwide catch of tropical tuna. And the artisanal and subsistence catches of reef fishes continue to supply the basic protein needs of hundreds of thousands of Pacific islanders.

Biodiversity:
The greatest marine bio-diversity lies in the western Pacific. This diversity diminishes from island to island, crossing the Pacific to the east. For example, the corals of the Hawaii Islands have been intensively studied and found to have similar species numbers to the Caribbean (approximately 60 and 2 massive soft corals) (Waddell, 2005). But the Hawaiian Island Archipelago is isolated from the central triangle of highest marine diversity among western Melanesia, Indonesia and the Philippines (Carpenter and Springer, 2005). The Marianas including Guam, also fairly well studied, lie closer to this center, and typical of many Pacific islands, have recorded over 400 species in 108 genera and 21 families of hard corals and over 30 species of massive soft corals (Paulay, 2003). Higher diversity progresses towards this center through Fiji, Palau and the Great Barrier Reef of Australia.

Traditional Knowledge:
The ancient pre-colonial knowledge of coral reef resources, which must have resided with the aboriginal Caribbean residents, apparently has been lost through most of those islands. But the descendents of Pacific island fishermen have retained much of their ancestors' knowledge of their reefs, which has been accumulated and passed down over thousands of years. The sustainable management practices for reef resources that evolved over these thousands of years remain active in some of the islands, although western ideas and their misplaced or unsuccessful management approaches have replaced traditional controls in many islands. Traditional knowledge retained by Palauan fishermen has been partially recorded in the book "Words of the Lagoon" compiled by Robert Johannes (1981), after residing in Palauan villages for over a year to seek out this knowledge. This gives an example of the actual scientific knowledge base that exists among island fisherfolk. Although their knowledge is usually focused on edible or harvestable species, it includes ecological information that can relate directly to coral health.

Many traditions regarding coral reefs still strongly influence the daily lives of islanders and must be recognized and respected by scientists wanting to study the reefs. For example, in many islands, submerged reef resources are privately owned or are strictly managed by chiefs or families (Gawel, 1984). This ownership and control is not simple in any case and involves complicated overlapping and changing rights to use various resources. These systems have succeeded in managing many of the key reef resources for centuries, but are being eroded often because of modern aspirations for commercial development of marine and coastal resources. Entry to some reef areas or taking of scientific samples may sometimes not be allowed. Even taking of a dead coral or piece of rock may be taboo at times in certain places. Gender distinctions separating how men and women relate to coral reef activities strongly persist in many islands, especially

related to subsistence uses. And diving on Sunday may be offensive or prohibited in some islands due to European derived religious beliefs, which are treated as strong tradition in many islands. Consequences of violating traditional laws can be very severe in certain island communities. Value of conservation or research efforts to the traditional users and to the health of the reefs may not always be recognized in decisions by traditional leaders. But their authority can remain unchallenged because of broader aspects of custom and traditional life.

War:
The impacts of World War II and pre-war activities in the Pacific islands contrast with that war's impacts on Atlantic reefs. American, European colonial and Japanese harbors and fleets were developed and destroyed across the coral reef islands. Serious damage was caused and, although most coral reefs have recovered over the last sixty tears, millions of pounds of explosives, fuel and toxic chemicals remain in the ships sunk in coral reef areas. An unfortunate result of war practices is the use of old recycled explosives to illegally harvest fishes by blasting coral reef areas.

Current Political Ties:
Although Guam and parts of Micronesia had been Spanish colonies for hundreds of years, and subsequently these islands (excluding Guam) were German and then Japanese owned, their ties with these mother countries have been greatly over-shadowed by the American influence since WWII. This has led to English being the most widespread language of Micronesia. South of the equator, France has retained three island groups within its nation and Australia and New Zealand have replaced former European nations as having the greatest alliances with the former colonies. (Note: the three Tongan archipelagoes had never been colonized and retain their traditional king.) The recently independent Pacific island countries are members of the United Nations and have attracted assistance and attention from East Asian neighbors including Japan, China, Taiwan and South Korea (Crocombe, 2001).

Authorities for Management:
Under modern laws in force in the Pacific islands, regulations and permit systems to manage marine resources uses and activities that impact coral reefs exist at national levels. For example, taking of endangered species like turtles may be prohibited and dredging for a new reef passage or harbor may require environmental impact assessments and permits with conditions. But these legal controls apply mostly to centers of population where more damaging impacts to the reefs tend to occur (Birkeland, 1997). Fortunately, the more remote reef areas remain more pristine although modern legal controls are less recognized by outer island populations. Traditional and local community controls continue to function more in areas of low development and population pressure. Even in national centers and developed areas, the approach of involving communities in coral reef conservation is proving to be the best approach. The U.S. Coral Reef Task Force is pursuing support of "Local Action Strategies" to locally develop strategies and projects at more of a community level for coral reef conservation, to be implemented with federal funding (Wilkinson, 2004). One of the nationally applied LAS issue areas is Coral Bleaching and Disease (Waddell, 2005). Those islands

not under the U.S. flag have other sources of support for coral reef conservation such as the Global Environment Facility and non-governmental organizations such as TNC.

Scientific Resources in the Pacific

A number of centers of coral reef scientific expertise have developed in the Pacific and new ones are arising. The following tables list some of the major academic, research and data centers in the tropical Pacific, excluding many from Hawaii and Australia.

Universities and Colleges:

- University of the South Pacific
- University of French Polynesia
- University of New Caledonia
- University of Papua New Guinea
- Community College of Micronesia
- College of the Northern Marianas
- College of the Marshall Islands
- Palau Community College
- University of Guam

Regional Institutions and Centers:

- Secretariat of the Pacific Regional Environmental Programme (SPREP)
- South Pacific Applied Geoscience Commission (SOPAC)
- Secretariat of the Pacific Community (SPC)
- Coral Reef Initiative for the South Pacific (CRISP)
- University of the South Pacific (USP)
- University of Guam (UOG)
- US Coral Reef Task Force: All Islands Group
- Marine Resources Pacific Consortium (MAREPAC)
  - American Samoa
  - Republic of the Marshall Islands
  - Federated States of Micronesia
  - Republic of Palau (Palau International Coral Reef Center)
  - Commonwealth of the Northern Marianas
  - University of Guam

Global and Regional Research and Data Organizations:

- Australian Institute for Marine Sciences
- The Global Coral Reef Monitoring Network
- ReefBase
- Reef Check
- Hawaii Institute of Marine Biology, University of Hawaii
- Institute of Marine Resources, University of the South Pacific
- University of Guam Marine Lab
- Palau International Coral Reef Center
- CRIOBE Research Center Moorea

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BASELINE LEVELS OF CORAL DISEASE IN THE NORTHWESTERN HAWAIIAN ISLANDS

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ABSTRACT

There has been a worldwide increase in the reports of diseases affecting marine organisms. In the Caribbean, mass mortalities among organisms in reef ecosystems have resulted in major shifts in community structure. However, our ability to fully understand recent disease outbreaks is hampered by the paucity of baseline and epidemiological information on the normal disease levels in the ocean. The Northwestern Hawaiian Islands (NWHI) is considered one of the last relatively pristine coral reef ecosystems remaining in the world. As such, it provides the unique opportunity to document the normal levels of disease in a coral reef system exposed to limited human influence.

In July 2003, baseline surveys were conducted at 73sites throughout the NWHI to quantify and characterize coral disease. Ten disease states were documented with the most common disease found to be *Porites* trematodiasis. This disease was widespread and is known to exclusively affect *Porites* sp. coral. Numerous other conditions were observed but at much lower levels of occurrence. Numbers of colonies affected by *Porites* trematodiasis were not enumerated but other types of conditions were counted with the average prevalence of disease estimated at 0.5%. Several of the observed disease states were distinct from what has been described from other coral reef systems. Coral genera exhibited differences in types of syndromes and prevalence of disease. Pocilloporids, common corals on the reefs of the NWHI, were comparatively resistant to disease. In contrast, acroporids showed the greatest damage from disease and the highest estimated prevalence of disease.

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Report 2: French Frigate Shoals Reef Health Survey, included in this document below.

Report 2. *French Frigate Shoals Reef Health Survey*

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INTRODUCTION

French Frigate Shoals (FFS) is one of the refugia comprising the Northwest Hawaiian Islands National Wildlife Refuge (NWHINWR). French Frigate Shoals was discovered by La Perouse in the late 18th century; however, the atoll was most notable as a naval air station during World War II when the US Navy dredged Tern Island into an airstrip, and the US Coast guard established a LORAN station on East Island. After the war, the LORAN station was moved to Tern Island where it remained until the Coast Guard vacated in 1979. Since then, the US Fish and Wildlife Service (USFWS) has managed Tern Island-FFS as a wildlife refuge with a full time staff presence (USFWS, 2001).

French Frigate Shoals consists of a large (31 nm) fringing reef partially enclosing a lagoon. A basalt pinnacle (La Perouse Pinnacle) arises approximately halfway between the two ends of the arcs of the fringing reefs. Tern Island is situated at the northern end of the lagoon and is surrounded by a dredged ship channel. The lagoon becomes progressively shallower from west to east and harbors a variety of marine life including corals, fish, marine mammals, and sea turtles (Amerson 1971). In 2000, an interagency survey of the northwestern Hawaiian Islands was done to document the fauna and flora in FFS (Maragos and Gulko, 2002). During that survey, 38 stations were examined, and 41 species of stony corals were documented, the most of any of the NW Hawaiian islands (Maragos and Gulko 2002). In some of these stations, corals with abnormalities were observed. The present study aimed to expand on the 2000 survey to evaluate the lesions in areas where they were documented.

METHODS

Survey areas:

Because of the ocean conditions, patchy distibution of corals, and lack of water clarity, all surveys were done using SCUBA. Survey locations were chosen based on observations of Jim Maragos during the 2001 NOWRAMP surveys (Fig. 1). Specifically, he had noted bulbous white tumors in *Montipora patula* on site R31 and *Porites lobata* with dead lesions in sites R33 and 39. Emphasis was placed on these locations and those areas where coral cover was substantial (>40%) and that were accessible by boat.

Corals were photographed using a Nikonos V underwater camera with a 20 mm lens and twin Ikelite 50 strobes or a digital camera in an underwater housing. Close-up photos were taken with a Nikonos V camera with a single Ikelite 50 strobe and a 2:1 extension tube. Coral samples were taken using bone shears, or hammer and chisel, and placed into labeled plastic bags in seawater. Gross lesions were broadly characterized as growth anomaly, tissue necrosis, algal infiltration, trauma, or a combination thereof.

Corals were preserved in Helleys fixative (Barszcz and Yevich, 1975) with added salt and allowed to fix for 24 hr. The fixative was decanted and the coral rinsed with fresh water once every 12 hr for 24 hr. Subsequently, coral was stored in 70% ethanol, decalcified

with Cal-ex II (Fisher Scientific), placed in cassettes, processed for paraffin embedding, trimmed at 5 um, and stained with hematoxylin and eosin. Slides were examined using light microscopy at magnifications ranging from 20-1000X. Normal histology of species of corals not yet in USGS-Hawaii Field Station archives was described. Lesions were classified as neoplasia, algal infiltration, necrosis, inflammation, atrophy or parasites.

RESULTS

For some of our survey, we were limited to doing shore dives from Tern Island because of wind conditions that rendered small boat operations in the lagoon unsafe. A review of available wind speed data for 1989-93 and 2000-1 revealed that the best times to conduct open water operations on Tern Island was late March-early April, late May-early June, late July mid-August, and early September-mid October (Fig. 2). We did a total of 16 dives half of which were shore dives from 18-28 March, 2002. Coral cover was generally patchy throughout all dive sites but seemed most sparse around the ship channel and most rich near La Perouse Pinnacle.

Lesions

Of 44 coral samples examined, 34 had lesions. Of the samples with lesions, algal infiltration of coral tissue was the predominant diagnosis (26) followed by bleaching (4), tumors (2), and parasites (1). The remainder of corals was healthy individuals used for reference purposes.

Algae-coral interactions:

Invasion of coral tissue by marine algae was seen in *Acropora cytherea, Porites lobata, P. evarmanni, P. compressa, Leptastrea purpurea, Montipora capitata, and Pavona duerdeni*. In *P. duerdeni* and *L. purpurea*, algal infiltrates had a distinct border with presence of macro-algae within the algal mats (Figs. 3A-B). Algal infiltrates in *A. cytherea* were characterized by ill-defined discolored areas with occasional greenish or yellow tinge or complete bleaching (Figs. 3C-D). In some instances, algal infiltrates were accompanied by exuberant skeletal growth (Figs. 3E-F). In *P. lobata* and *P. evarmanni*, algal infiltrates had less distinct borders, and coral tissues adjacent to infiltrates were bleached or tinged with pink coloration (Figs. 3G-H). Gross lesions of algal infiltration in *M. capitata* consisted of firm, smooth to rugose raised areas (Figs. 4A-D).

On microscopy, a mixed assemblage of algal organisms infiltrated coral tissue. In *A. cytherea*, reaction to algal infiltration was much less marked and generally characterized by focal tissue death and focal thickening of the gastrodermis of the gastrovascular canal near the algal infiltrates (Figs. 4E-F). In *Porites* sp. and *P. duerdeni*, within gastrovascular canals, gastrodermal cells formed rosettes and clumps among algal filaments. In many instances, there was localized thickening of calicoblast layer adjacent to algae. Clumps of sloughing and necrotic gastrodermal cells appeared constricted by

linear bands of unidentified granular grey material. In many cases, there were clumps of necrotic debris associated with algae (Figs 4G-H; 5A-D). In *Montipora* sp., algal infiltrates were often accompanied by necrosis and a prominent cellular response comprising calicoblast and pigment cells (Figs. 5E-H).

Parasites:

The only instance of parasitism was in *P. lobata*. Grossly parasites were manifested by multiple small bumps scattered throughout the surface (Figs. 6A-B). On histology, these foci were characterized by necrosis of epithelium (Fig. 6C). Within the gastrovascular canal network, there was erosion of epithelium and proliferation of calicoblast and pigment cells around putative coccidia (Figs. 6C-F). As the lesion progressed, it appeared that these coccidia became encapsulated leading to eventual loss of sporozoites (Figs. 6G-H). In many cases, parasite-induced lesions were accompanied by algal infiltrates (Fig. 6H).

Tumors:

Tumors were seen only in *A. cytherea*. One type of tumor was cauliflower-like and had focal distribution on the coral (Fig. 7A). Another type of tumor was also localized but appeared vermiform (Fig. 7B). On histology, both tumors revealed a marked disorganized proliferation of gastrodermal cells with no mitotic figures and no necrosis (Figs. 7C-D).

Miscellaneous:

Other lesions included the burrows of the skeleton-dwelling symbiotic crab *Pseudocryptochirus kahe* McCain and Coles (1979) (Fig. 7E) and bleaching (Fig. 7F) in *P. eydouxi* and trauma of *P. evermani* due to fish bites. Bleaching of *P. eydouxi* was characterized by lack of pigmentation of tissues, and on histology, bleached tissue was markedly atrophied with loss of mesoglea and zooxanthellae (Fig. 7H). Lesions caused by *P. kahe* consisted of distinct holes within coral skeleton surrounded by hyperpigmented tissue. On microscopy, each hole contained a single crab surrounded by mats of algae. Fish bites in *P. evarmanni* consisted of localized areas of tissue ablation revealing white skeleton beneath.

Normal histology:

Palythoa tuberculosa (Figs. 8A-D)

Colonies consisted of closely apposed large polyps encrusting on the substratum. Coenosarc epithelium was composed of columnar cells mixed with holotrichous isorhizas, clusters of eosinophilic granular cells, scarce zooxanthella, and large vacuoles. Tentacle epithelium consists of closely apposed columnar cells mixed with spirocysts, granular pigment cells, and zooxanthella. Deeper down, near the mesoglea, there was a mesh-like filigree of cells and delicate filaments. Pharyngeal epithelium was composed

of closely apposed ciliated columnar cells with aggregates of granular eosinophilic and brown pigment cells at the base.

Tentacle gastrodemis was composed of cuboidal cells replete with zooxanthellae. Gastrovascular canals course through the large mesoglea in a haphazard manner and are lined by similar cells as those found in tentacles. Mesenteric filaments was composed of a cnidoglandular cap consisting of closely apposed columnar cells, eosinophilic granular cells, few holotrichous isorhizas, and a base consisting of columnar cells mixed with large numbers of zooxanthella and granular eosinophilic cells.

Mesoglea consisted of a large network of connective tissue enveloping a mesh of skeleton. Gastrovascular canals course through the mesoglea that forms the structural matrix. Several different types of cells were noted within the mesoglea including eosinophilic granular cells, stellate cells with basophilic cytoplasm, denegerating zooxanthella, and small amphophilic cells with small dark granules in a clear cytoplasm. At the base where the zooxanthid contacted the substrate, the mesoglea was composed of eosinophilic debris mixed with branching septated organisms.

Trabeculae of skeleton coursed through mesoglea. Calicoblast layer was almost not discernable and consisted of a single layer of eosinophilic granular cells. Occasional hyphae and filamentous organisms were seen in the skeletal space.

Cyphastrea ocellina (Figs. 8E-F)

Large brown encrusting colony with large calices. Coenosarc epithelium consisted of columnar cells mixed with vacuoles and pigment cells. Epithelium of polyps consisted of closely apposed columnar cells mixed with vacuoles and batteries of spirocysts. Pharynx epithelium consisted of closely apposed ciliated columnar cells. Mesoglea of coenosarc was thin and somewhat thickened with mesogleal pleats within mesenteric filaments.

Gastrodermis of coenosarc was vacuolated and contained moderate numbers of zooxanthella and granular brown pigment cells. Gastrodermis of polyps was massively distended with zooxanthella. Gastrodermis of gastrovascular canals contained granular brown pigment cells. Cnidoglandular cap of mesenteric filaments consisted of closely apposed columnar cells with spirocysts. Occasional planula were noted within gastrovascular canals. Calicoblast consisted of single squamous layer of cells.

Leptastrea purpurea

Coral was encrusting, brown, and has large contiguous calices reminiscent of brain coral. Epithelium consisted of columnar ciliated cells underlaid focally by larger supporting cells. Spirocysts were scattered throughout although these were clumped and more numerous within tentacles. Occasional gray vacuolar cells are noted. Pharyngeal epithelium consists of closely apposed columnar ciliated cells mixed with larger dark globular cells.

The mesoglea formed an arching structure that was overlaid externally by epithelium and internally by gastrodermis forming a single layer canal network. Mesogleal pleats were prominent. The gatrodermis underlying epithelium was composed of pseudostratified columnar epithelium containing numerous zooxanthellae. Mesenteric filaments contained typical closely clumped columnar cells with prominent eosinophilic granular cells at cnidoglandular cap. Some mesenteric filaments contained batteries of holotrichous isorhizas. Gastrodermis of gastrovascular canal contained numerous mucous and pigment cells. The calicoblast consisted of a single layer of squamous cells that were focally hyperplastic.

DISCUSSION

Algal-coral interactions made up the preponderance of lesions encountered in corals at FFS. It is likely that some of the necrotic lesions in Porites sp. and the tumor-like lesions in Montipora observed by Maragos during the 2000 NOWRAMP survey were due to invasion of coral tissue by algae. Insufficient time and weather conditions precluded our doing manta-tows to assess spatial distribution of lesions. As such, we were unable to determine if certain sites had higher incidence of coral-algal interactions than others. However, cursory observations did not indicate a particular site having unusual numbers of lesions.

Different species of corals appeared to respond to algal invasion differently. All species responded by increasing thickness of gastrodermis and calicoblast layers adjacent to algae. Peters (1984) described blistering necrosis of cells in Caribbean corals infiltrated by algae. The appearance of sloughing rosettes of gastrodermis appeared to be a response limited to *Porites* sp. and *P. duerdeni*. Exuberant growth of skeleton in response to algae was characteristic of *Montipora* sp. giving them a gross appearance of a tumor-like growth. Although similar responses were seen in some *A. cytherea,* this was far less common. A similar manifestation to coral invasion was seen in Montipora from Johnston Atoll (Work et al., 2001). *Montipora* sp. were one of the few species that also showed microscopic evidence of a distinct cellular inflammatory response to algal invasion. Most studies of coral immunity have involved grafting experiments (Jokiel and Bigger 1994, Hildeman et al. 1975). Microscopic evidence of inflammation in corals is a rarely documented phenomenon that merits further study. Likewise, the algal assemblage infiltrating coral tissues appeared to be a mix of different species. There is a need to elucidate what species of algae are associated with these lesions.

The manifestation of parasitism in *P.lobata* was markedly different than that observed in the main Hawaiian Islands. In the latter case, the parasite was a trematode that is common in the main Hawaiian Islands (Aeby 1991). In this study, the parasites were compatible in morphology to coccidia and were similar in appearance to *Gemmocystis* sp. recorded by Upton and Peters (1986) in *Porites* sp., *Montastrea* sp., *Diploria* sp. and *Meandrina* sp. from the Caribbean. The coccidia seen here contained a single sporozoite, and the coral appeared to mount a response to these parasites via proliferation of calicoblast cells. In later infections, there appeared to be encapsulation of coccidia

suggesting a possible mechanism of parasite clearance by the coral. Other parasites that have been documented from corals elsewhere in the Caribbean include ciliates, nematodes, and amoeba (Peters 1984).

The tumors in *A. cytherea* were both gastrodermomas based on tissue morphology and location of cell proliferation. Criteria used to define these lesions as tumors were similar to those used by Work et al. (2001). One type of tumor was identical to that seen in *A. cytherea* from Johnston Atoll and classifed as type 1 tumor (Work et al., 2001). The second type of tumor was also a gastrodermoma but manifested as a vermiform rather than a cauliflower-type growth. Two coral heads were seen with a type 1 tumor whereas only one coral was noted with the vermiform tumor. The paucity of tumors on *A. cytherea* suggests that they did not appear to pose a major threat to this species.

Remaining lesions were incidental findings. The crab-induced lesions in *P. eydouxi* were similar to those observed on the main Hawaiian Islands (Work and Rameyer, 2001). The numbers of corals affected with this lesion appeared small, and interestingly, corals that were infested with crabs appeared to have high numbers with tissue that appeared paler than normal. The crab-induced lesions are common on thick-branched *Pocillopora* corals in the Hawaiian Islands (McCain and Coles, 1979) and Johnston Atoll (Work et al., 2001). Fish bites in *Porites* sp. were also similar to those observed in the island of Hawaii.

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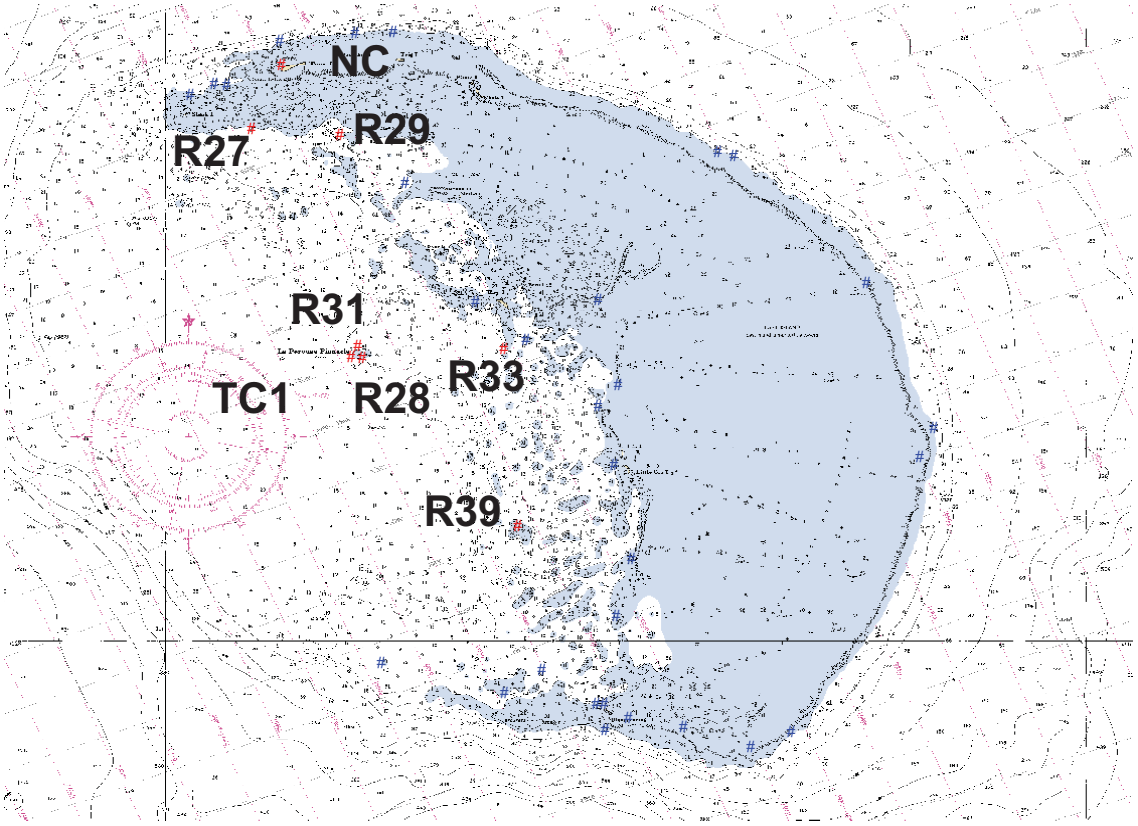

Figure 1. NOWRAMP 2001 survey locations for FFS. Survey sites for this study in red

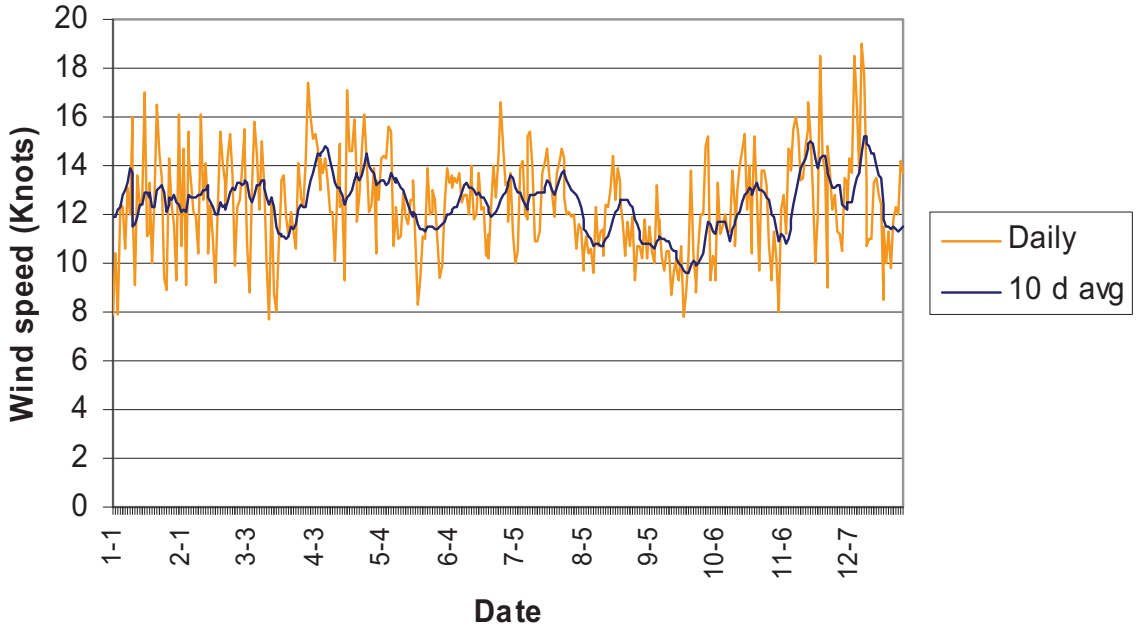

Figure 2. Daily average wind speed (orange) and 10 day running average wind speed for 1989-93; 2000-11, FFS.

Figure 3. ***P. duerdeni.*** **Note clear demarcation between normal tissue (right) and alga (left) (A).** ***L. purpurea.*** **Note clearly defined patches of algal growth (arrow) (B).** ***A. cytherea*** **(C-F). Note algal infiltration with bleaching (C) and pigmentation (D). Note algal growth (arrow) accompanied by skeletal proliferation (E). Close up of C (F).** ***P. lobata*** **(G-H). Note ill defined areas of depigmentation (G) and raised pigmented areas on edge of lesion (H).**

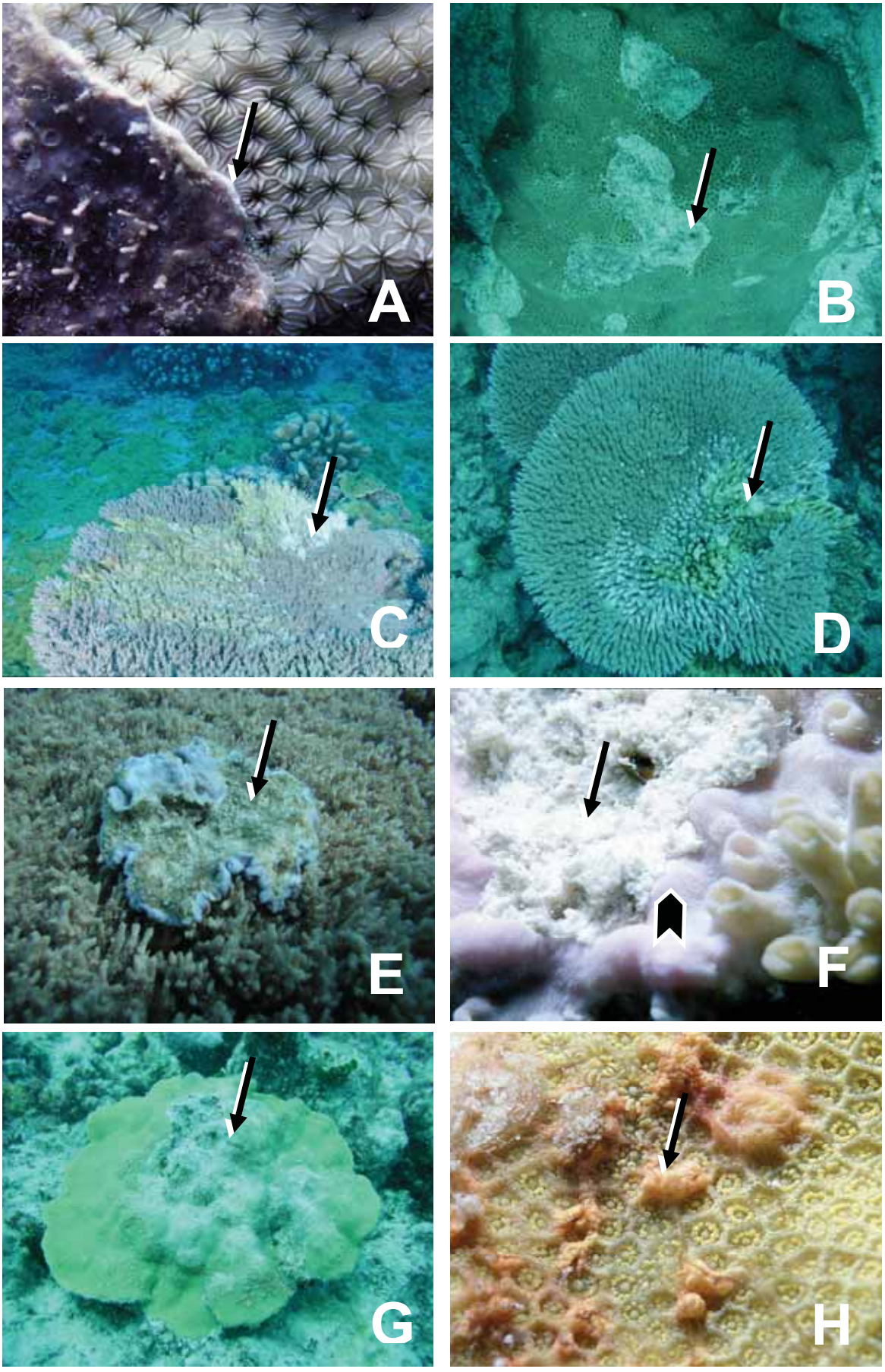
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Figure 4. ***M. capitata*****, note growth anomalies (arrows) (A-D*). *A. cytherea* (E-F). Note focus of necrotic tissue (arrow) and algal filaments (arrowhead), bar=50 µm (E). Hyperplastic gastrodermis (arrow) and algal filaments (arrowhead), bar=100 µm (F). *P. lobata* (G-H). Polyp being invaded by algae (arrowhead), bar=100 µm (G). Sloughing gastrodermal cells forming rosettes (arrowhead), bar=50 µm (H).**

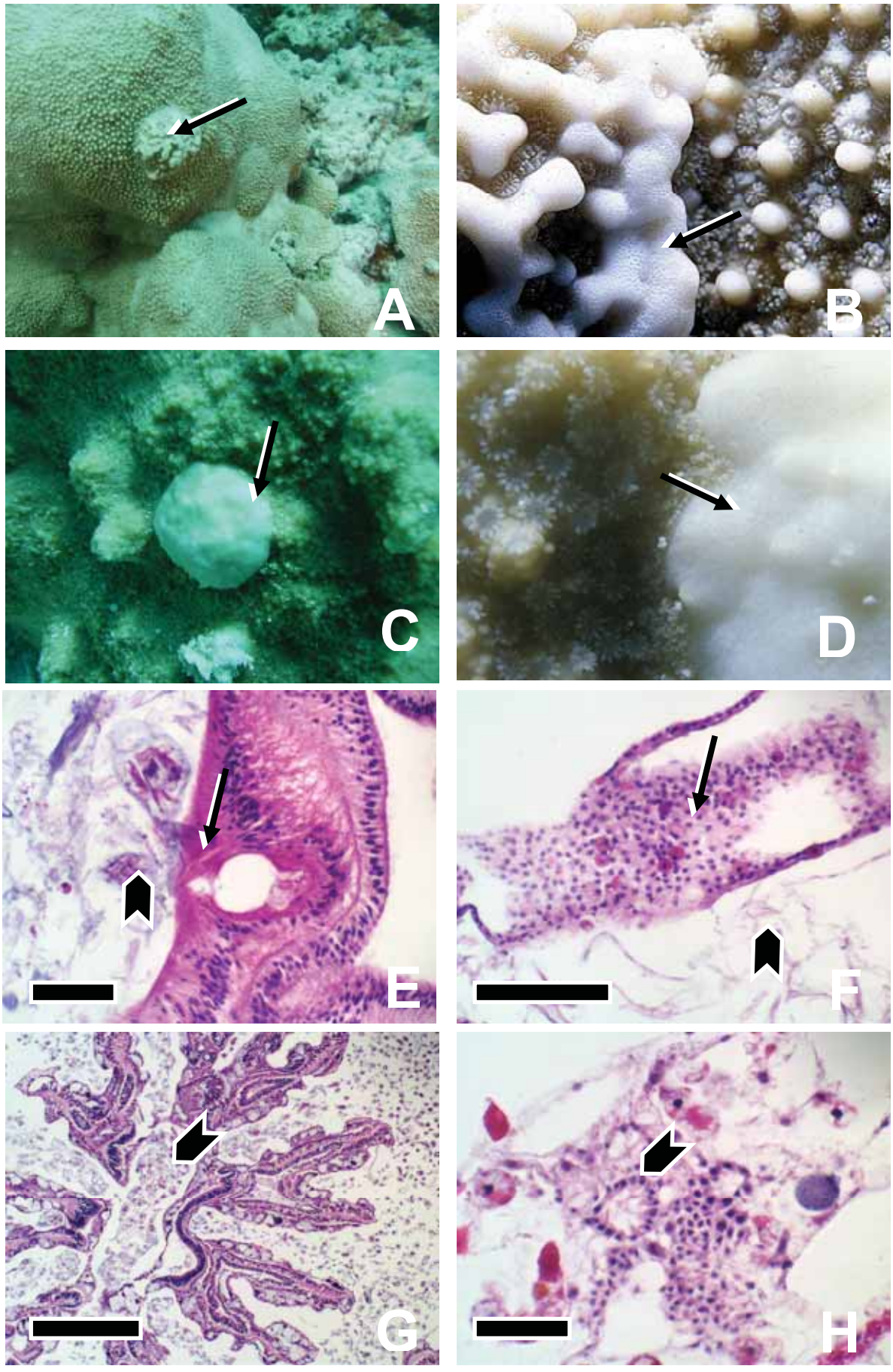
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Figure 5. ***P. lobata*** **(A-D). Note clump of alga surrounded by red capsule (arrow) and reactive calicoblast cells (arrowhead), bar=50 µm (A). Note alga (arrowhead) and reactive calicoblast (arrow), bar=100 µm (B). Clump of necrotic gastrodermal cells among algal filaments. Note bands of grey material (arrow), bar=100 µm (C). Close of of band of grey material (arrow) and algal filament (arrowhead), bar=100 µm (D). M. capitata (E-H). Note thickened gastrodermis in area of algal infiltration (arrowhead), bar=200 µm (E). Note sparse algal infiltrates in skeletal matrix (arrowhead) overlaid by thin squamous membrane (arrow), bar=200 µm (F). Note thickened gastrodermis in gastrovascular canal (arrowhead) and granular red inflammatory cells (arrow), bar=50 µm (G). Granular red cells (arrow) among thickened gastrodermal cells and algal filaments (arrowhead), bar= 50 µm (H).**

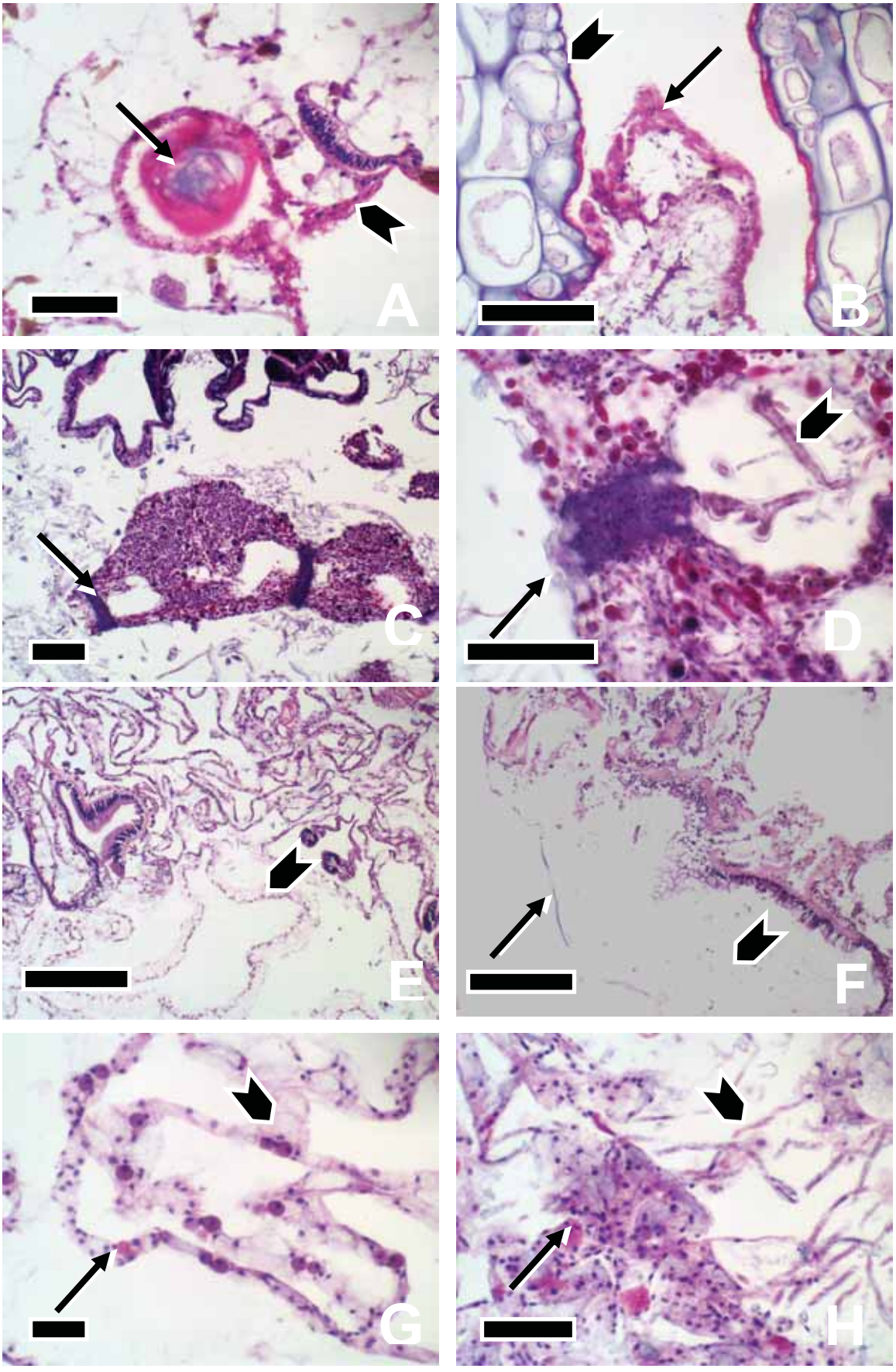
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Figure 6. ***P. lobata*** **(A-H). Coral with focal lesions pan view (A) and macro (B). Coenosarc epithelium. Note necrosis (arrow), bar=100 µm (C). Reactive calicoblast (arrowhead) and pigment cells (arrow), bar=10 µm (D). Clump of gastrodermal cells, loose zooxanthella and pigment cells (arrow), bar=20 µm (E). Coccidia containing single sporozoite (arrow) surrounded by reactive calicoblast cells, bar=20 µm (F). Similar coccidian as in F (arrow) surrounded by laminar capsule (arrowhead), bar=20 µm (G). Encapsulated coccidian minus sporozoites. Note algal filaments (double arrow), bar=20 µm (H).**

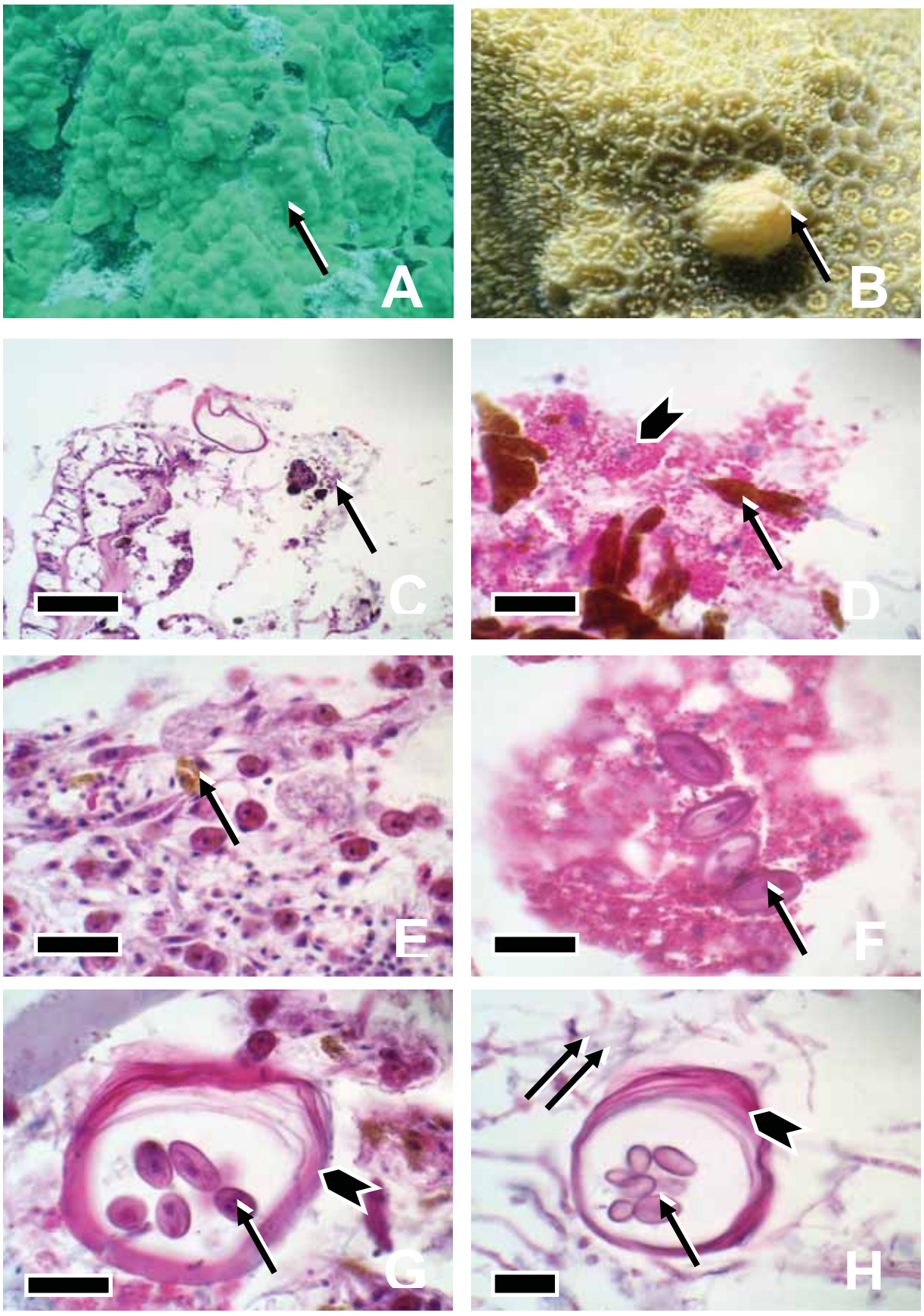
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Figure 7. ***A. cytherea*** **(A-D). Type 1 tumor (A) and vermiform tumor (B). Note proliferation of gastrodermis, bar=100 µm (C-D).** ***P.eydouxi*** **(E-H). Note holes with pigmented rim of tissue (arrow) (E). Note areas of bleaching (arrow) (F). Normal epithelium (arrow) and gastrodermis (arrowhead), bar=50 µm (G) and atrophied epithelium (arrow) and gastrodermis (arrowhead), bar=50 µm (H).**

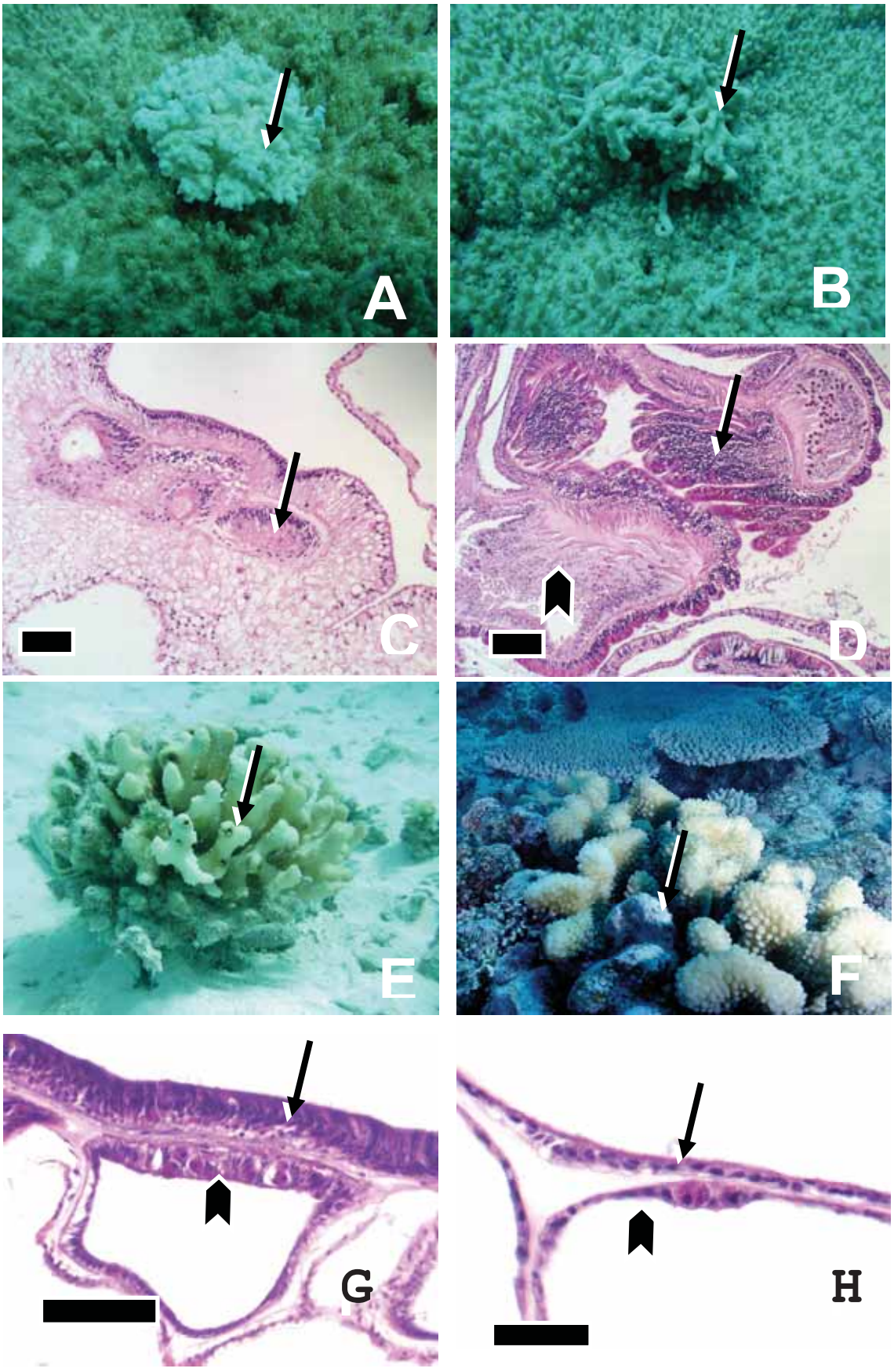
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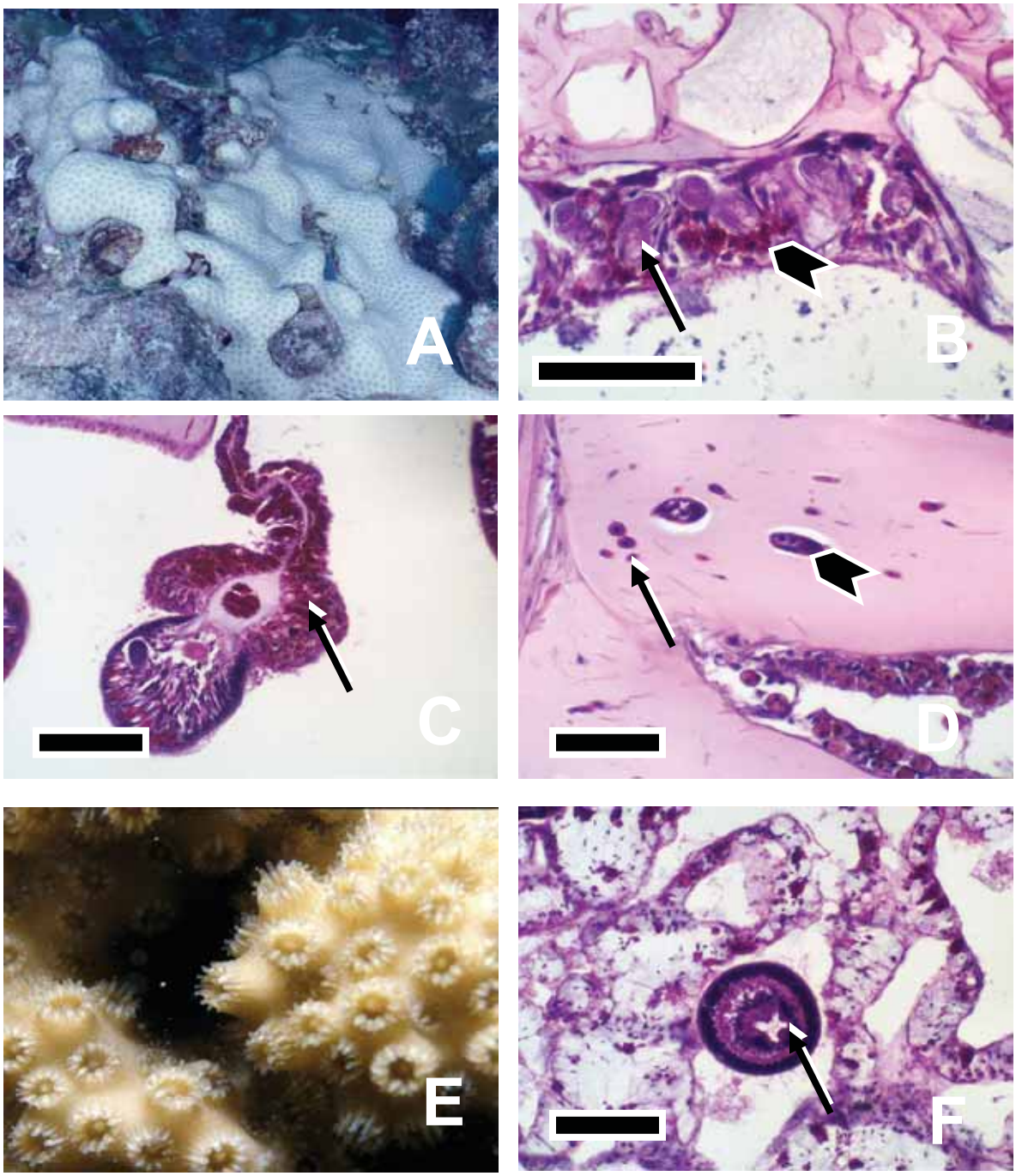
Figure 8. Blue-gray zooanthid (A-D). Epithelium (bottom) and mesoglea (top). Note holotrichous isorhizas (arrow) and red granular cells (arrowhead) in epithelium, bar=100 µm (B). Mesenteric filament. Note the base replete with zooxanthella and red granular cells (arrow), bar=100 µm (C). Mesoglea. Note gastrovascular canal (arrowhead) and mesogleal cells (arrow), bar=100 µm (D). Cyphastrea ocellina (E-F). Note planula (arrow) in gastrovascular canal, bar=100 µm (F).



CURRENT KNOWLEDGE OF DISEASES IN US TERRITORIES/FREELY ASSOC. STATES

Thierry Work

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Report 1: CORAL AND CRUSTOSE CORALLINE ALGAE DISEASE ON THE REEFS OF AMERICAN SAMOA
Greta Aeby, Thierry Work, Eva Didonato

Report 2: JOHNSTON ATOLL REEF HEALTH SURVEY
Thierry M. Work, Steve L. Coles, Robert A. Rameyer

Report 1. CORAL AND CRUSTOSE CORALLINE ALGAE DISEASE ON THE REEFS OF AMERICAN SAMOA

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Thierry Work, USGS, National Wildlife Health Center, Hawaii Field Station
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Submitted October, 2005

INTRODUCTION

The world's coral reefs are in serious decline (Wilkinson 2004). The effects of overfishing and pollution from agriculture and land development have been a major force accelerating decreases in abundance of coral reef species (Hughes et al. 2003, Pandolfi et al., 2003). With increased human populations the scale of human impacts on reefs has grown exponentially. Within American Samoa, alone, the population has risen 22% in the last ten years (Turgeon et al., 2002) and thus so has the potential for damage to the near shore resources. Compounding these anthropogenic stressors are the impacts of global climate change which is predicted to result in more frequent bleaching episodes and higher levels of disease (Hughes et al., 2003). As such, reef managers are faced with the challenge of developing strategies to maintain these reefs in the face of these changing conditions and it becomes clear that research in support of management is urgently needed.

The reefs of American Samoa support more than 200 species of corals and their conditions have been affected by both natural disturbances (crown-of-thorns starfish invasion, hurricanes and mass bleaching events) and human-induced impacts (pollution and over fishing) (Turgeon et al., 2002). The reefs of American Samoa suffered mass bleaching in 1994 (Birkeland et al., 2000) with reports of bleaching also occurring in both 2002 and 2003 (Peter Craig, pers. comm.). Coral disease has been reported from these reefs (Work & Rameyer 2002) but no surveys to quantify disease on a spatial scale had been conducted. An important component of monitoring the health of reefs is to have baseline 'before' data with which to compare 'after' conditions (Porter et al. 2001; Santavy et al. 2001). Unfortunately, investigations on the role of disease in animal populations are often done only after problems are noticed and out of control. However, American Samoa is in the unique position of not having suffered major catastrophic declines of reefs due to disease. Furthermore, significant effort is planned by American Samoa Department of Marine and Wildlife Resources to conduct monitoring programs on reefs throughout Manua and Tutuila. As such, American Samoa is well placed to develop a baseline assessment of the health of their reefs. The objectives of this study were to: 1) document the baseline levels of bleaching and disease in the major genera of corals and coralline algae; 2) compare incidence of disease in coral and coralline algae across a gradient of levels of reduced water quality based on watershed population; 3) systematically describe gross and microscopic morphology of lesions in reef corals and develop a standardized nomenclature for identifying and designating coral disease.

METHODS

The first task of the proposed research was to conduct a baseline assessment of the abundance and distribution of bleached and diseased corals and crustose coralline algae at seven sites in American Samoa (Vatia (National park), Tafeu Cove (National park), Fagaitua, Faga'alu, Fagatele Bay (National marine sanctuary), Leone and Maloata (see Table 1, GPS coordinates, and Fig. 1, map).

Figure 1. Map showing the seven sites surveyed for bleaching and disease in corals and crustose coralline algae in June, 2004.

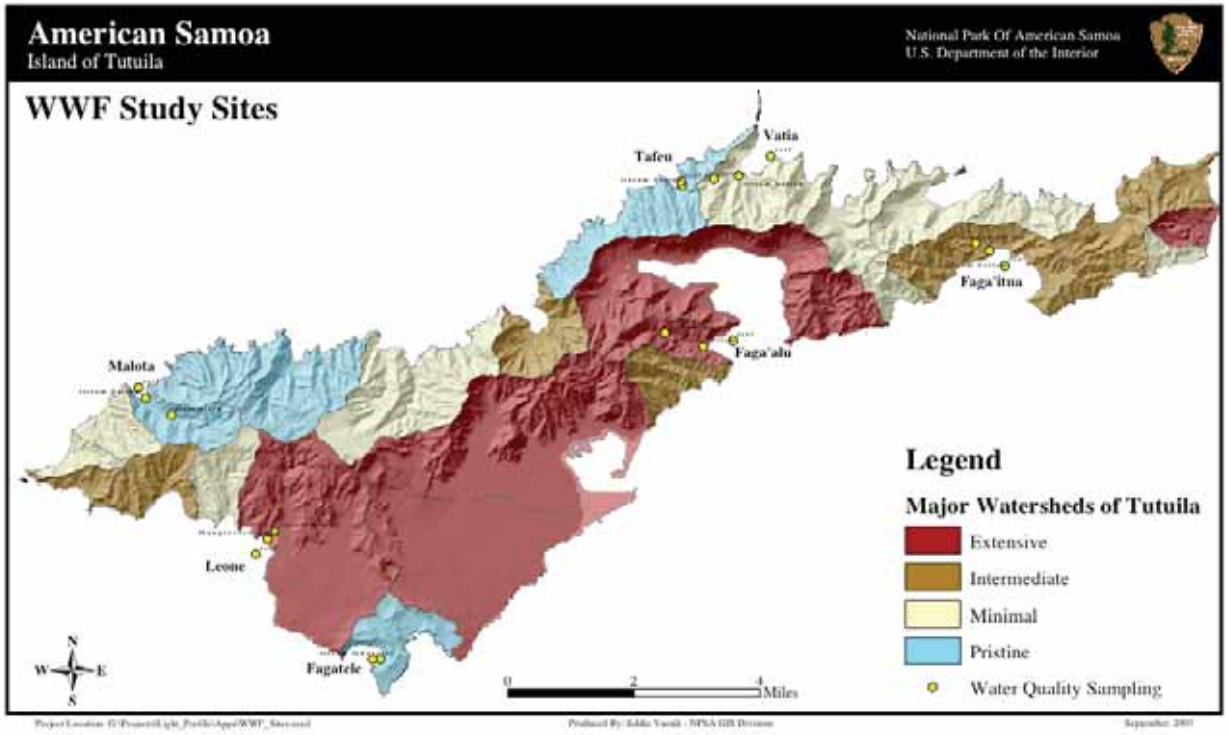


Table 1. Coordinates of sites surveyed for bleaching and disease of coral and coralline algae on Tutuila in June, 2004. Coordinates were taken by GPS unit onboard boat at start of transect 1

Site	Date	Depth (ft)	Latitude (S)	Longitude (W)	Notes
Fagaitua1	6/8/04	27	14 16.342'	170 36.728'	fringing reef off Fagaitua village- WWF site
Fagaitua 2	6/8/04	22	14 17.005'	170 36.393'	fringing reef off Alofau village
Fagatele Bay 1	6/9/04	18	14 21.944'	170 45.736'	Fagatele Bay National Marine Sanctuary-WWF site
Fagatele Bay 2	6/9/04	22	14 21.817'	170 45.708'	other side of the Bay from WWF
Tafeu 1	6/10/04	20	14 15.142'	170 41.338'	fringing reef- WWF site
Tafeu 2	6/10/04	21	14 15.158'	170 41.498'	other side of the embayment from WWF
Vatia 1	6/14/04	20	14 14.774'	170 40.076'	fringing reef- WWF site
Vatia 2	6/14/04	26	14 14.704'	170 40.262'	fringing reef other side of the embayment from WWF
Leone	6/15/04	32	14 20.592	170 47.328	fringing reef- WWF site
Maloata	6/15/04	25	n/a	n/a	fringing reef- WWF site
Faga'alu	6/16/04	19	n/a	n/a	fringing reef- WWF site

A project funded by the World Wildlife Federation (WWF) has been documenting water quality parameters at these sites, providing a gradient of sites with presumed anthropogenic impacts (Lara Hansen, personal communication). We documented baseline levels of coral bleaching and disease at each of the sites using two 25 m x 2 m belt transects with visual counts (total 100 m^2 area of reef). The two transect lines were laid end to end along depth contours (20-30 ft) separated by approximately 5 meters. A team of two divers swam along the transect, with one diver identifying and enumerating coral colonies, while the other diver recorded incidence of bleaching and disease. Corals were identified to the genus level and assigned to one of seven size classes (0-5cm, 6-10cm, 11-20cm, 21-40cm, 41-80cm, 81-160cm and > 160cm). These size classes and protocols were adapted from Mundy (1996) who used them for broad-scale surveys in American Samoa. They have also been used to examine coral community structure in the NWHI (Maragos et al., 2004). Diseased corals and coralline algae were photographed and a general description of the condition was recorded. Samples of diseased coral (and healthy portions for controls) were collected for laboratory investigations using standard histopathological techniques. Substrate characteristics were documented by line-intercept method whereby the substratum underlying the tape measure was recorded at 10 cm intervals. At some of the sites (Vatia, Tafeu, Fagatele, Faga'itua) a 2nd station was surveyed as described above. Surveys from both stations were combined at these sites for analysis. Surveys were conducted June, 8-16, 2004.

Another task of the proposed research was to systematically describe gross and microscopic morphology of lesions in corals and crustose coralline algae and develop a standardized nomenclature for identifying and designating disease. For characterization of gross lesions, corals with abnormalities were photographed with a digital camera (Olympus C5050) contained in an Ikelite® housing, and attached to an Ikelite® 50 substrobe. Lesions were photographed from two aspects. A pan photograph encompassed the larger colony to gauge the extent of the lesion. Close-up photos were done of the same lesion to gain detail on polyp morphology. The following data were recorded: date, location, and depth of collection.

Grossly, lesions were classified by distribution as focal, locally extensive, or diffuse. Lesions were further classified as tissue loss, discoloration, or growth anomaly.

Tissue loss included those cases where tissue was missing leaving exposed skeleton. In such cases, we recorded if the skeleton was intact or damaged (eroded) and characterized as acute, subacute, and chronic. Acute tissue loss was those cases where skeleton was bare (eroded or intact). Subacute tissue loss included cases of algal growth on skeleton separated from intact tissue by bare skeleton. Chronic tissue losses were those cases where skeleton was completely covered by algae or sediment. Discoloration included those lesions where tissues were abnormally colored and was further subdivided into bleaching and non-bleaching discoloration. Growth anomaly included those lesions exhibiting anomalous growth of coral skeleton.

For histopathology, sections of corals were fixed in Z-Fix (Anatech Ltd.) according to manufacturer instructions. Briefly, Z-fix was diluted 1:5 with seawater and placed in 100 cc plastic jars. Coral were placed in the fixative and allowed to fix for at least 24 h. Corals were decalcified in dilute formic acid/formaldehyde solution (CalExII, Fisher Scientific) until the skeleton was completely dissolved. Tissues were dehydrated in alcohol series, embedded in paraffin, sectioned at 5 μm, placed on microscope slides and stained with hematoxylin and eosin. Special stains were used as appropriate to identify fungi, bacteria, algal filaments, or protozoa. Tissues were examined using light microscopy at magnifications ranging from 40X-1000X.

On histology, lesions were classified as depletion of zooxanthella, atrophy, uncomplicated necrosis, necrosis associated with fungi, algae, protozoa, or metazoa, and hyperplasia of gastrovascular canals. Depletion of zooxanthella included cases where gastrodermis was depleted of zooxanthella. Uncomplicated necrosis included evidence of cytoplasmic fragmentation and hypereosinophilia, pyknosis, or karyorrhexis. Organisms were classified as algae if they were filamentous and stained negative with silver or if cell walls were present or fungi if they stained positive with silver (Prophet et al. 1992).

RESULTS

Surveys

Coral community structure

Based on colony counts within transects, the four dominant coral genera on the reefs of Tutuila were *Montipora* (39.9% of the total colonies), *Galaxea* (12.9%), *Pocillopora* (11.4%) and *Acropora* (10.1%). Coral community structure varied among the seven sites as did the coral cover (Table 2).

Table 2. Coral community structure at seven sites around Tutuila surveyed for bleaching and disease in June, 2004. Percent coral cover was estimated using line-intercept. Coral community was calculated from colony counts along belt transects. Data show the percentage of colony counts along the transect represented by each coral genera. Dominant coral genera shown in bold for each site.

	Fagatele	**Maloata**	**Tafeu**	**Vatia**	**Faga'itua**	**Leone**	**Faga'alu**
coral community (%)							
Acropora	8.7	4.6	3.1	**19.7**	10.7	15.3	10.6
Astreopora	0	0.57	0.77	0.24	0.39	0	0
Coscinaraea	0	0.57	2.3	0.72	0.65	0.4	0
Cyphastrea	0	0	0	0	0.13	0	0
Diploastrea	0	0	0	0	0	0	0
Echinophyllia/Oxypora	0.25	0	0	0	0.65	0	0
Echinopora	0.13	0	0	0	0.91	0	0
Favia/Favites	4.9	4.6	3.8	3.6	1.7	1.2	0.33
Fungiidae	1.6	0.57	0.38	2.2	0.78	0	0.66
Galaxea	**38.6**	9.7	1.2	1.9	10.5	0	0
Goniastrea	0.5	0	0	0.24	2.2	0	0
Goniopora/Alveopora	0.13	0	0	0.24	0	0.4	0
Hydnophora	0	0	0.19	0	0.52	0	0
Leptastrea	0.38	2.3	11.2	0.48	0.65	1.2	0.33
Leptoseris/Pachyseris/Coe	0.63	0	0	0	0	0	0
Lobophyllia	0.25	0	0	0.24	3.8	0	0
Merulina/Scapophyllia	0	0	0	0	0	0.8	0
Millepora	0	0	0	0.24	0	0	0
Montastrea	0	4.6	0.77	0.96	0.26	0.4	1.3
Montipora	20.8	**39.4**	**51.9**	41	**39.7**	**70.7**	**46.4**
Pavona	2.4	10.3	14	13.4	11.2	2.4	5.6
Pocillopora	7.8	21.7	4	9.4	12.9	3.6	33.4
Porites	12.9	1.1	6.3	5.5	2.5	3.6	0.66
Psammocora	0	0	0	0	0	0	0.66
avg. coral cover (%)	46.3	22.8	45.7	48.2	28.4	40.9	32.6

Overall occurrence of bleaching and disease

Within belt transects an estimated 16,824 coral colonies from 24 different genera were examined for bleaching and disease. Bleaching was found at very low levels affecting less than one percent of the overall colonies. Six different coral disease states as well as a number of lesions not associated with disease were documented from four coral genera on the reefs of Tutuila (Appendix I). Three diseases affected *Acropora*: *Acropora* white syndrome, *Acropora* ciliate disease, and *Acropora* growth anomalies. *Montipora* was affected by one disease: *Montipora* growth anomalies. *Porites* was affected by one disease: *Porites* tissue loss syndrome. *Lobophyllia* was affected by one disease: *Lobophyllia* tissue loss syndrome. Coral disease was found at all seven sites but the overall proportion of colonies examined that had signs of disease (prevalence) was low (0.143%) (range = 0.029-0.40%). The crustose coralline algae (CCA) disease,

coralline lethal orange disease (CLOD) was found at 4 of the 7 sites (57% of the sites). The number of CLOD infections per m^2 of CCA ranged from 0 to 0.24 (Table 3).

Distribution and prevalence of each disease state

Distribution of the different coral diseases varied (Table 3). *Acropora* white syndrome was found to be the most widespread disease occurring at 5 of the 7 sites (71.4%). *Acropora* growth anomalies occurred at 4 of the 7 sites (57.1%) with the other diseases only occurring at one site each (14.3%). The proportion of coral colonies affected by each disease (prevalence) also varied with both *Acropora* white syndrome and growth anomalies being the highest (0.624% each). Prevalence of the other diseases was lower; Acropora ciliate disease (0.07%), *Porites* tissue loss syndrome (0.095%), and *Montipora* growth anomalies (0.031%). Prevalence of *Lobophyllia* tissue loss syndrome was not calculated as the diseased colony was not found within the transect area.

Prevalence of disease differed among the affected genera with *Acropora* having the highest overall prevalence (1.2%) compared to the other genera; *Montipora* (0.031%), *Porites* (0.095%).

Relationship between disease and watershed usage

The seven sites were originally selected based on a population gradient within each watershed used as an indicator of anthropogenic stress. The number of different diseases present within each site varied as did the overall prevalence of disease (Table 3). However, no patterns emerged suggesting that disease levels were directly related to anthropogenic watershed stress.

Table 3. Distribution and prevalence of different coral diseases and density of coralline algae diseases around Tutuila in June, 2004. Coral disease prevalence=(# diseased colonies/# colonies examined)*100
Coralline lethal orange disease (CLOD) density =# CLOD infections/est. m^2 of CCA at site
X* Disease present at site but prevalence not calculated as affected colony was not within belt transect
Human usage rating based on watershed populations.

human usage	pristine	pristine	pristine	minimal	intermediate	extensive	extensive
site	**Fagatele**	**Maloata**	**Tafeu**	**Vatia**	**Faga'itua**	**Leone**	**Faga'alu**
Acropora white syndrome	0.24		2.08	1.08	0.54		0.52
Acropora ciliate disease				0.27			
Acropora growth anomalies	X*	4.2		1.6		5.3	
Montipora growth anomalies		0.48					
Porites tissue loss syndrome	0.16						
Lobophyllia tissue loss syndrome					X*		
# CLOD/m^2 CCA	0.24	0.02	0	0.009	0	0.004	0
overall coral disease prevalence	0.031	0.286	0.032	0.266	0.029	0.402	0.055

Histology (gross and microscopic findings)

We examined tissue specimens from 8 sites comprising 67 samples from 59 colonies comprising at least 20 species of corals (Table 4). Of these, 10 were from apparently normal coral and the remainder from lesions. The most common gross lesions included tissue loss and growth anomalies, and for these, *Acropora* sp. and *Montipora* sp. were over-represented (Table 5).

Table 4. Number of coral samples collected for histopathology at various sites around Tutuila, June 2004.

Species	Faga'itua2	Faga'alu	Fagatele	Faga'itua1	Leone	Malato	Tafeu1	Vatia1	Total
Acropora abrotenoides				1				2	3
A. cytherea			1					7	8
Acropora sp.					1				1
plate *Acropora*		1					2	7	10
corymbose *Acropora*		1			3			1	5
encrusting *Acropora* sp.	2		1			1			4
Astreopora sp.							1		1
Favia sp.			1	1			2		4
Galaxea sp.			2						2
Goniastrea sp.	1								1
Leptoria phrygia			1						1
Lobophyllia corymbosa			2						2
Lobophyllia sp.	2		2						4
massive *Porites* sp.	1						2		3
Porites rus			2						2
Montastrea sp.							1		1
Montipora sp.	1	1	2	3	1	1	1	1	11
Pocillopora eydouxi				2					2
Pocillopora meandrina								1	1
Pavona sp.				1					1
Total	**7**	**3**	**14**	**8**	**5**	**2**	**9**	**19**	**67**

Table 5. Gross lesions found in various genera of corals in Tutuila, June 2004.

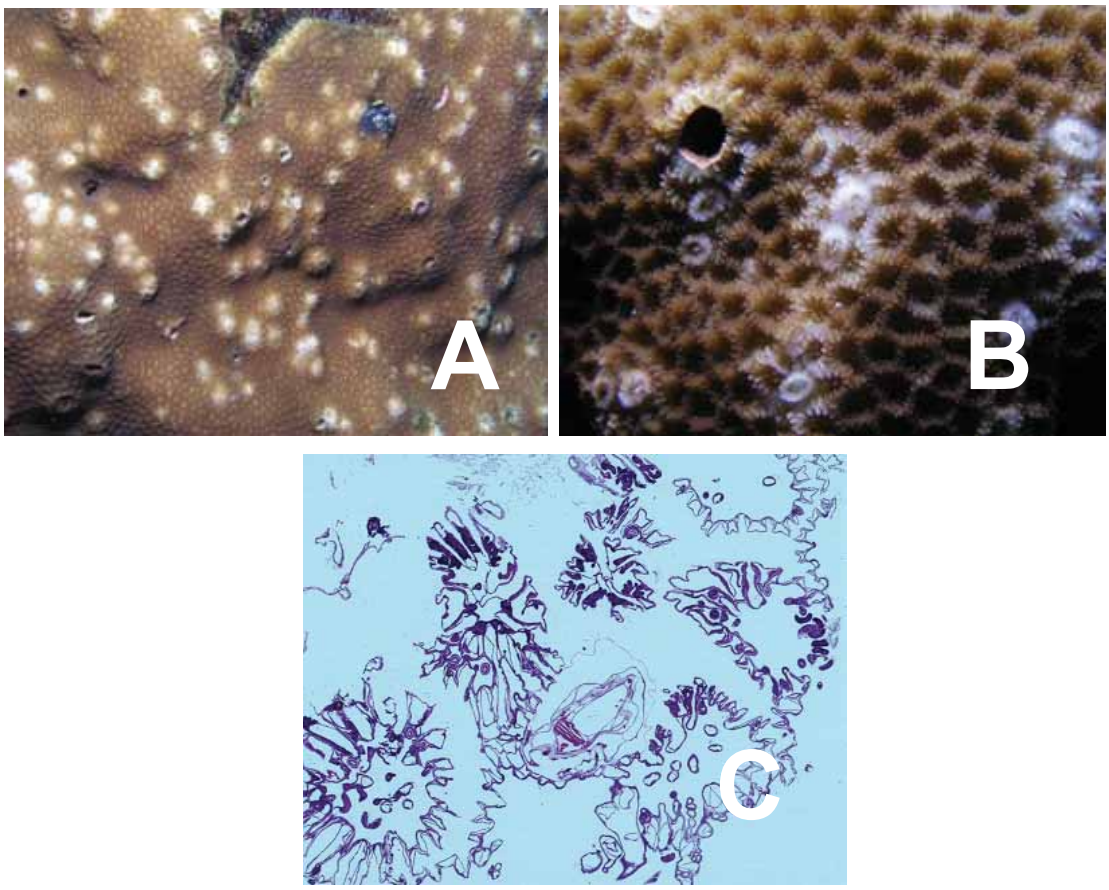
Genus	Discoloration	Growth anomaly	Tissue Loss	Normal	Total
Acropora	1	11	14	5	31
Astreopora	1				1
Favia	4				4
Galaxea	1			1	2
Goniastrea			1		1
Leptoria	1				1
Lobophyllia	1		2	3	6
Montastrea	1				1
Montipora		3	8		11
Pavona			1		1
Pocillopora	1		1	1	3
Porites	4		1		5
Total	15	14	28	10	67

Tissue loss:

The following patterns were seen for tissue loss:

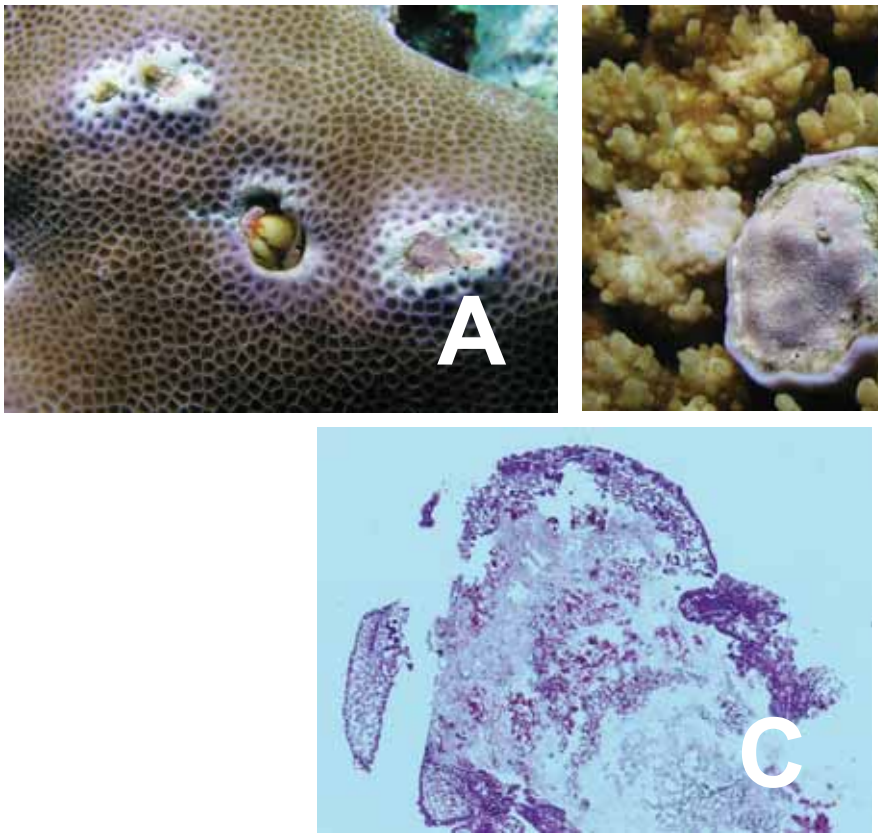
Barnacle infestation: This was seen in *Goniastrea* only and was characterized by small white foci encompassing approximately the diameter of one polyp (Fig. 2A). The center of the area contained a barnacle with bleaching of tissues immediately around the barnacle (Fig. 2B). Microscopy revealed a crustacean surrounded by normal coral tissue (Fig. 2C). In some cases, a mix of algae and sponges infiltrated into the coral tissue leading to cell fragmentation and necrosis.

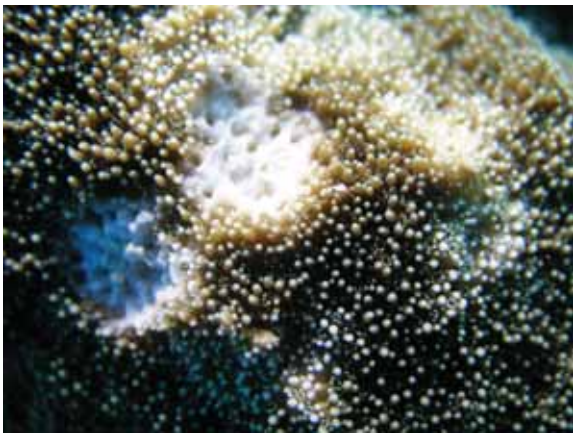
Figure 2. A) *Goniastrea* sp. with barnacles. B) Note barnacles (arrowhead) surrounded by bleached tissue. C) Photomicrograph of barnacle (arrowhead) among coral tissue.



Chronic tissue loss and excessive skeletal growth: This was seen in massive *Porites* sp. and *Acropora cytherea* and was characterized by a focus of coralline or turf algae surrounded by exuberant skeletal growth. On microscopy, these lesions manifested as full thickness necrosis of coral tissue associated with marine algae with some depletion of zooxanthella (Fig. 3).

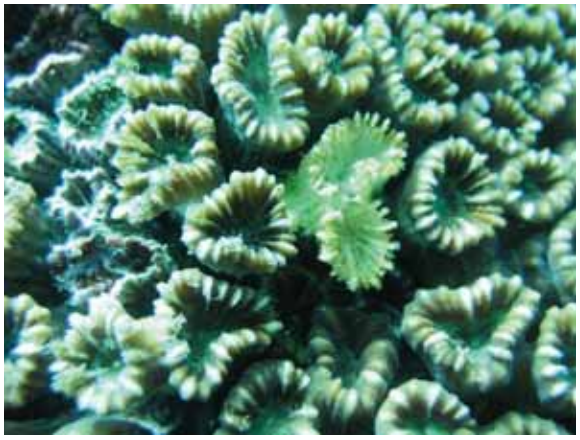
Fig. 3. A) Massive *Porites*. B) *Acropora cytherea*. A-B) note central area of coralline algae infiltration with exuberant skeletal growth surrounding lesion. C) Photomicrograph of E. Note, mass of algae and sponges and clumps of necrotic coral tissue.





Fish bites: This was manifested by well-circumscribed localized loss of tissue accompanied by bare eroded skeleton (Fig. 4). On histology, these lesions were characterized by fragmentation of coral tissue with depletion of zooxanthella from gastrodermis.

Figure 4. *Montipora* sp. fish bite. Note characteristic erosion of skeleton and loss of tissue.



Subacute tissue loss: This was manifested by complete tissue loss of single polyps revealing bare intact skeleton completely covered with algae. Only *Lobophyllia* sp. was sampled with this lesion (Figure 5).

Figure 5. *Lobophyllia* tissue loss

Acute tissue loss: This was manifested by a well-circumscribed diffuse area of tissue loss revealing intact white skeleton. The tissue loss usually encompassed the edge of the colony. On histology, the major changes seen included no lesions, tissue fragmentation, or hypertrophy of calicoblastic epithelium. In some cases, this tissue loss was associated with presence of corallivorous snails. Tissue loss in *P. rus* was attributed, on microscopy, to infestation with sponges associated with necrosis. (Fig. 6).

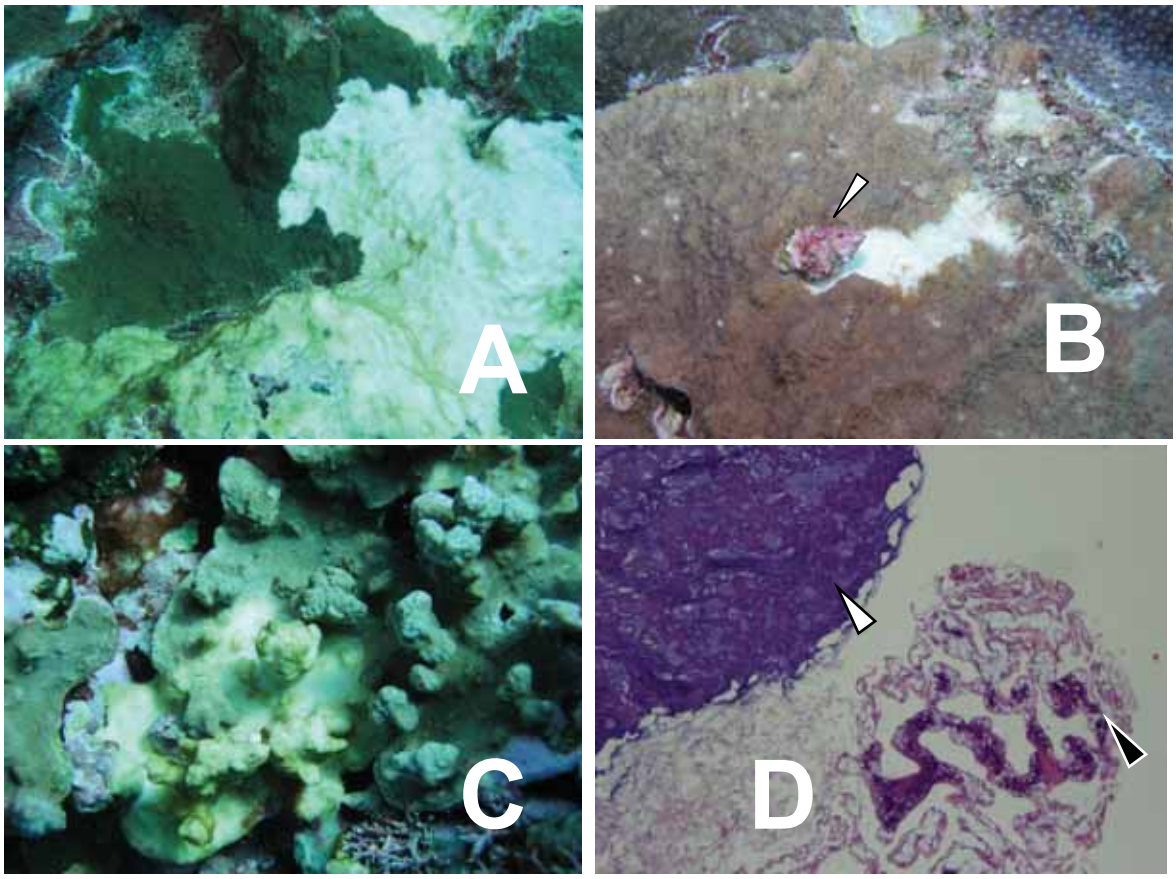


Figure 6. A-B) Acute tissue loss in encrusting *Montipora* sp., note bare white skeleton bereft of tissue and lesion predominantly on the edge of the colony. B) Snail (arrowhead) associated with acute tissue loss in *Montipora* sp. C) *P. rus* acute tissue loss. D) Photomicrograph of D. Note invasion of coral tissue with sponge (white arrowhead) and necrosis of tissue (black arrowhead).

Acute to subacute tissue loss: This was manifested by well circumscribed diffuse areas of tissue loss revealing intact skeleton with presence of recent algal growth in the center and bare intact white skeleton at the interface between tissue and algae. This was found mainly in plating *Acropora*. On microscopy, findings ranged from simple uncomplicated tissue loss to infection with microparasites (ciliates) (Fig. 7).

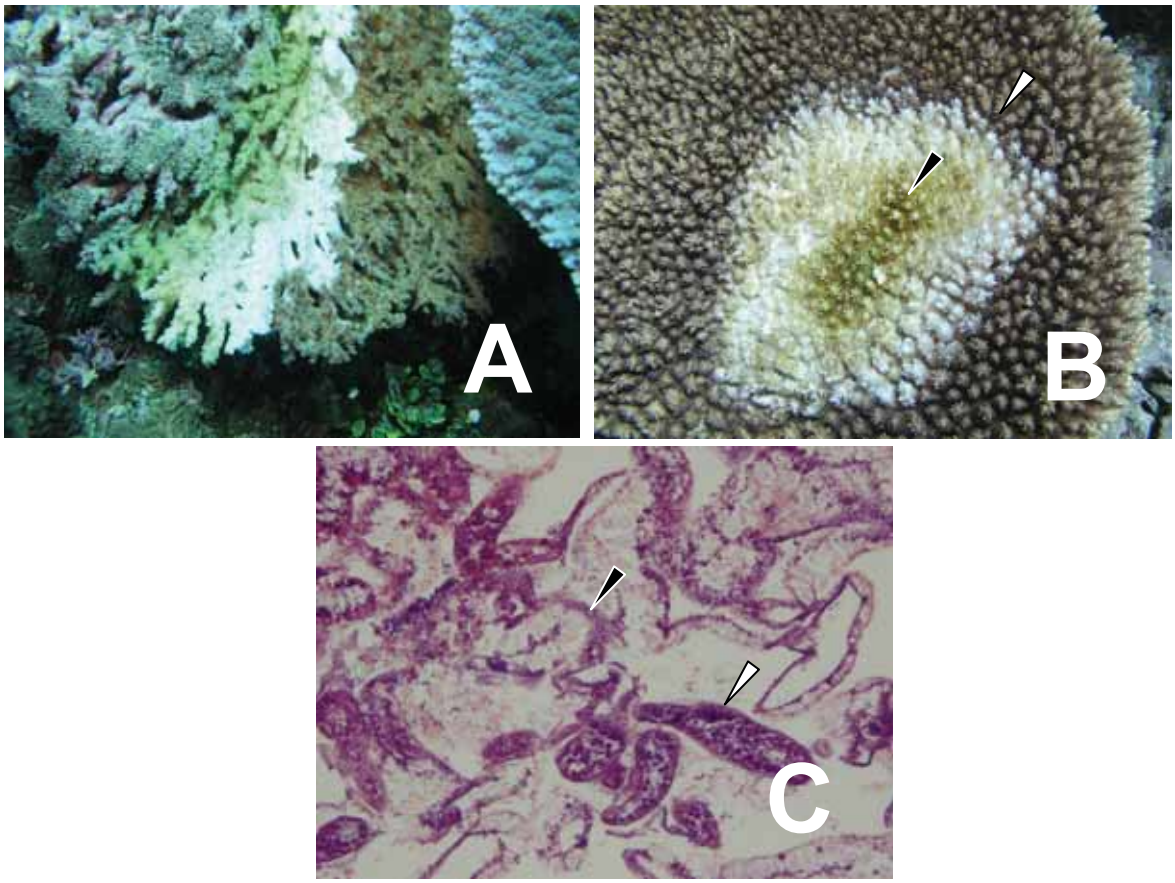


Figure 7. A-B) Plating *Acropora* sp. with subacute tissue loss. A) note distinct band of white skeleton bereft of algae separting intact tissue and skeleton covered by algae. B) Note presence of algae covered intact skeleton (black arrowhead) separated from intact tissue by intact skeleton bereft of algae (white arrowhead). C) Photomicrograph of B, *Acropora* with ciliate infection. Note invasion of ciliates (white arrowhead) associated with necrotic tissue (black arrowhead).

Discoloration:

Bleaching: We saw 10 cases of bleaching in 7 genera of corals. Grossly, bleaching was characterized by a diffuse white discoloration. Bleaching in corals is typically attributed to loss of symbiotic zooxanthellae from coral tissues, and on microscopic examination, this was seen in all cases. In 3 cases, additional microscopic lesions were seen including necrosis of tissue associated with infiltrates of sponges or algae. In those cases, the sponge/algae complex was seen invading tissue from below and overlying epidermis and gastrodermis were intact but atrophied and bereft of zooxanthella (Fig 8).

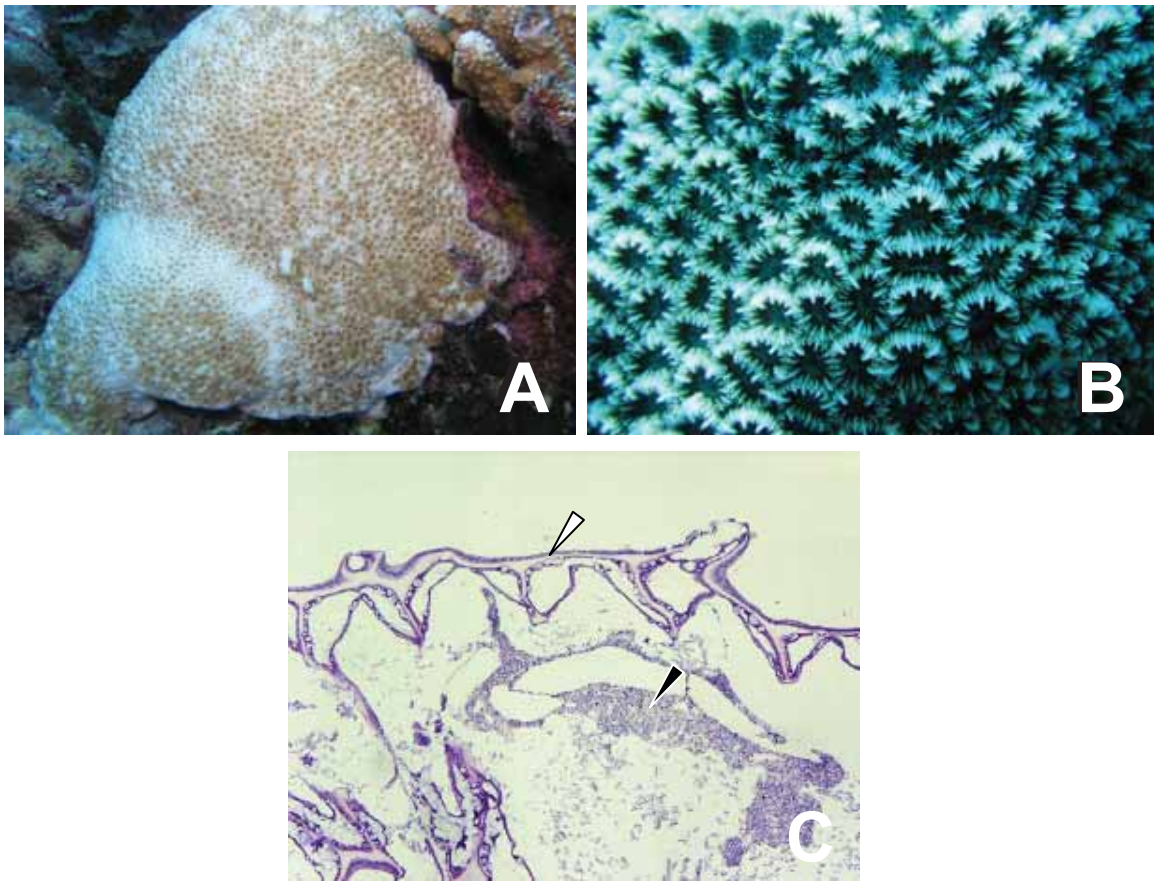


Figure 8. A) *Astreopora* sp. with diffuse bleaching. B) *Favia* sp. with diffuse bleaching. C) Photomicrograph of B. Note infiltration of sponges and algae (black arrow) below the intact epidermis (white arrowhead) with gastrodermis bereft of zooxanthella.

Non-bleaching discoloration: Other cases of discoloration did not fit the bleaching pattern. Discoloration in *Favia/Favites* was, on microscopy, attributed to mucus sheathing and not considered abnormal. No microscopic lesions associated with discoloration were seen in *Lobophyllia* (Fig. 9).

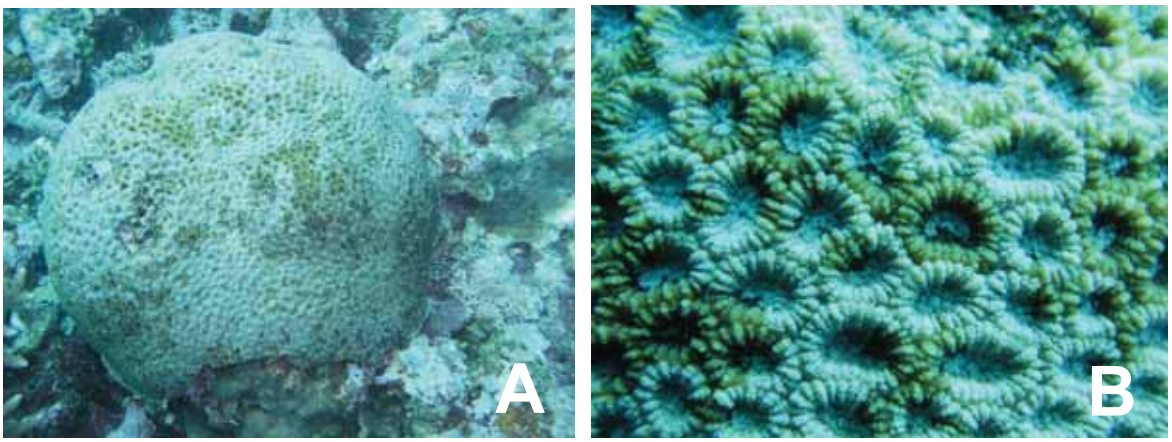
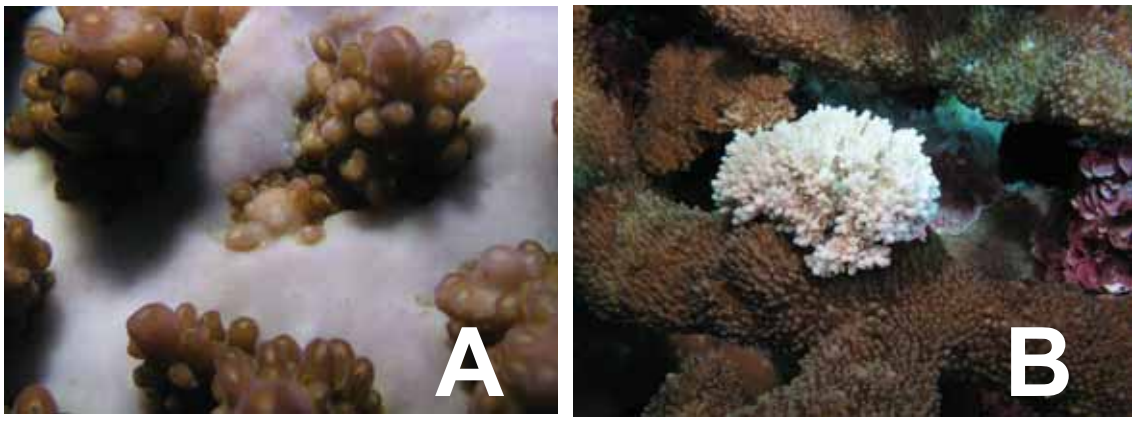


Figure 9. A-B) Mucus sheathing in *Favia* sp., note well-defined areas of discoloration.

Growth Anomaly

Acropora spp. were the most common genera see with this lesion although *Montipora* spp., also had growth anomalies. These ranged from smooth to more rugose and cauliflower shapes and was found in branching, corymbose, and plating *Acropora (*Figure 10*)*. On microscopy, the most common finding was hyperplasia of gastrovascular canals; however, there were not the classic hallmarks of cancer as seen in other animals making it difficult to conclude that these are true neoplasia *sensu stricto.* In two cases, growth anomalies were, on microscopy, associated with necrosis of tissues associated with marine algae.



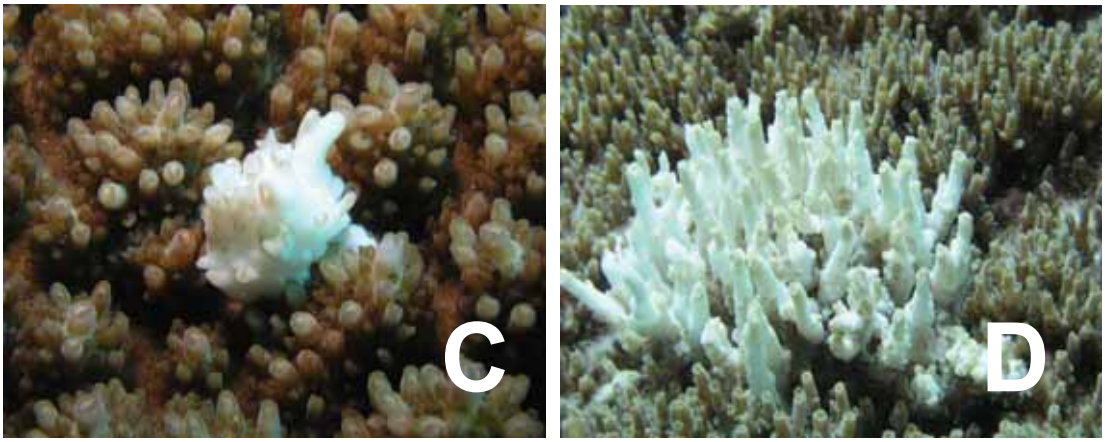


Figure 10. Growth anomalies in Acropora species. A) Corymbose colonies; B) Branching colonies; C-D) plating colonies.

Summary of Survey Findings

1. The overall prevalence of coral disease was found to be low (0.143%) as compared to other regions in the Indo-Pacific. Overall disease prevalence in the Northwestern Hawaiian Islands was found to be 0.5% (Aeby, in press). Willis et al. (2004) surveyed eight sites along the Great Barrier Reef (GBR) and found the prevalence of disease in hard corals to range from 7.2-10.7%. Raymundo et al. (in press) surveyed eight sites in the Philippines and reported an overall prevalence of disease of 14.2%.
2. Six coral disease states and one disease of CCA were documented from the reefs of Tutuila. Bleaching was found at low levels.
3. Five of the six coral disease states have been reported from other areas of the Indo-Pacific. *Porites* tissue loss syndrome is reported from the NWHI (Aeby, in press) Australia (Willis et al., 2004) and the Philippines (Raymundo et al., in press). *Acropora* white syndrome is reported from the NWHI (Aeby, in press) and Australia (Willis et al., 2004). Growth anomalies in both *Acropora* and *Montipora* have been recorded from Australia (Willis et al., 2004), Johnston Atoll (Work et al., 2001) and Okinawa (Yamashiro et al., 2000, 2001; Yamashiro 2004). *Lobophyllia* tissue loss syndrome has not yet been reported elsewhere. It must be noted that there are regional differences in names assigned each set of field disease signs but through the efforts of the Coral Disease and Health Consortium (www.coral.noaa.gov/coral_disease/cdhc.shtml) this nomenclature problem will eventually be resolved. It should also be noted that any similarities in field signs of disease between regions does not necessarily imply the diseases have the same etiology.
4. After histopathological analysis it was found that a number of coral lesions found during surveys were not associated with infectious agents or underlying pathologic process. These include lesions due to predation, barnacle infestation and mucous sheathing. This confirms the critical component histology plays in understanding disease processes.

5. There were differences in prevalence of disease among coral genera with *Acropora* having the highest prevalence. *Acropora* comprised only 10.1% of the overall coral community along the transects yet showed the highest overall prevalence of disease (1.2%). In contrast, *Montipora* comprised 40% of the coral community but had a disease prevalence of 0.031%. This suggests that there may differences in disease susceptibility among coral genera and that pathogens do not necessarily affect the most common or abundant corals. *Acropora* have also been found to have the highest levels of disease in the NWHI (Aeby, in press). The sites surveyed had differences in coral community which would be a factor in what diseases and what levels of disease would be found at a particular reef.
6. No pattern emerged suggesting that disease levels were directly related to anthropogenic watershed stress.

Summary of Histological Findings

1. Some cases of acute tissue loss are probably due to snail predation. We opted to be conservative and to include only those cases where snails were visible, however, this is probably an underestimate. Lesions presumably attributable to snails were generally peripheral on encrusting colonies and this pattern was more prevalent in certain sites versus others. Quantifying populations of corallivores in conjunction with measurement of lesions would be helpful in evaluating their effects on reefs.
2. Certain gross lesions have clear causes. For example, patchy tissue loss and discoloration with presence of a crustacean in the center of the lesion is indicative of barnacle infestations. Likewise, focal erosion of tissues and skeleton are indicative of fish bites. Determining the species of barnacles affected and the types of bites produced by particular fish may be useful in the future.
3. Not all discolorations are disease processes. In the case of faviids, mottled discoloration is probably indicative of mucus shedding.
4. Growth anomalies are more common in acroporids. Determining how fast these grow and what effect they have on the health of the corals should be the focus of future investigations.
5. There is a need to refine the description of gross lesions in corals in order to better understand whether certain gross lesions can be related to microscopic findings in a more consistent manner.

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Appendix I. Summary of coral lesions found in American Samoa in June 2004.

A. CORAL DISEASES

Acropora ciliate disease

Histology: ciliates associated with necrotic tissue

Location: Vatia

Frequency of occurrence (# sites w/ disease/tot sites surveyed): 9.1%

Acropora white syndrome

Histology: subacute tissue loss and necrotic tissue consistent with disease

Location: Fagatele, Tafeu, Vatia, Faga'itua, Faga'alu

Frequency of occurrence: 45.5%

Acropora Growth Anomalies

Histology: hyperplasia (overgrowth) of gastrovascular canals.

Location: Fagatele, Maloata, Vatia, Leone

Frequency of occurrence: 27.3%

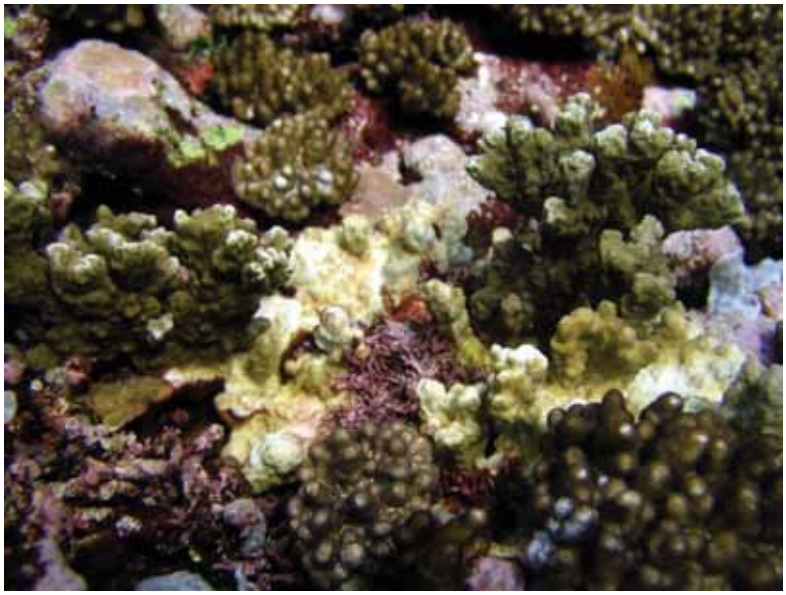
Montipora growth anomalies

Histology: hyperplasia (overgrowth) of gastrovascular canals.

Location: Maloata

Frequency of occurrence: 9.1%

Porites tissue loss syndrome

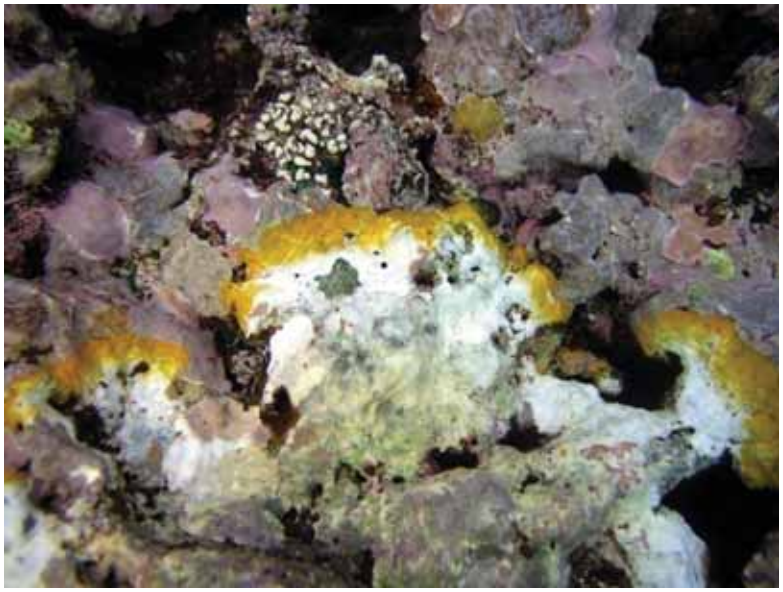


Histology: tissue necrosis and sponge invasion
Location: Fagatele
Frequency of occurrence: 9.1%

Lobophyllia tissue loss syndrome

Histology: chronic tissue loss
Location: Fagaitua
Frequency of occurrence: 9.1%

Coralline lethal orange disease (CLOD)



Location: Fagatele Bay, Maloata, Vatia, Leone

Frequency of occurrence: 45.5%

Report 2. JOHNSTON ATOLL REEF HEALTH SURVEY

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INTRODUCTION

Johnston Atoll is an overlay U. S. Fish and Wildlife Service (USFWS) National Wildlife Refuge located ~1300 km southwest of Honolulu, Hawaii. Johnston Atoll is not part of the Hawaiian Islands, but is more closely associated with a subsurface mountain range called the Marcus-Necker Rise (Amerson and Shelton, 1976). The atoll was discovered in 1796, used in the early 19th century for guano extraction, and during WWII, the US Navy used it for aerial and submarine operations. Subsequent activities included establishment of LORAN towers by the US Coast Guard, and use of the atoll for atomic weapons tests (Amerson and Shelton, 1976). Since 1976, the USFWS and the Defense Nuclear Agency have an agreement whereby USFWS manages the natural resources of the atoll. In 1985, a chemical weapons disposal plant (Johnston Atoll Chemical Agents Disposal System) was built on the Western end of the main island (Johnston). This plant is soon to be decommissioned, and Johnston Atoll will revert back to the USFWS in ~2003.

Johnston Atoll is composed of a fringing reef surrounding 4 islands, two of which (Haukau and Hikinau) are man made (Fig. 1). The lagoon becomes progressively deeper from northwest to southeast and supports a variety of corals, reef fish, sea turtles, and seabirds (see Coles et al., 2001, Appendix A for full listing of recent and previous reports). Compared to other atolls and reef environments, the marine fauna of Johston Atoll is depauperate by species, although coral coverage is high. The dominant species of corals include *Acropora cytherea, A. humilis, A. valida, Montipora patula, Pocillopora eydouxi* and *P. meandrina.* Maragos and Jokiel (1986) classified the lagoon into 4 area with the northwest portion harboring the greatest abundance of corals. During and after WWII, much of the coral in that area was destroyed during extensive dredging and filling of the reef and lagoon (Brock et al 1965, 1966).

Much work has been done to identify the identity and extent of organisms at Johnston Atoll (see Coles et al., 2001 for review). However, relatively little effort has been spent looking at health parameters of marine fauna. Cohen et al. (1997) observed bleaching of corals, and Coles et al. (2001) observed growth anomalies in *Acropora cytherea*. The USFWS sponsored the USGS National Wildlife Health Center to further investigate growth anomalies of corals in Johnston Atoll. From 29 March to 2 April, 2001, a team from the US Geological Survey Hawaii Field Station (Work, Rameyer) and the Bishop Museum (Coles) surveyed coral reefs at Johnston Atoll for lesions. The trip had the following objectives:

1) Conduct manta tow and spot surveys for lesions in dominant coral species in the northern lagoon and sites off Johnston and East islands.
2) Describe gross and microscopic anatomy of lesions in corals.
3) Obtain pigment profiles of the dominant coral (*Acropora cytherea*).

METHODS

Survey areas:

Broad-scale manta tow surveys were done using flat-bottom boats with an outboard motor focusing on the northwestern portion of the lagoon because it had the highest density of coral (Maragos and Jokiel, 1997), and because it encompassed the area where growth anomalies in *A. cytherea* were previously noted by Coles et al. (2001).

Six locations were selected for spot dives. Site 1 was where Coles et al. (2001) observed growth abnormalities in *Acropora* cytherea. Site 2 (Agent Orange) was immediately offshore the northwestern portion of Johnston Island where the herbicide Agent Orange had been stored during the Vietnam war. Site 3 (Mt. Pluto) was directly offshore the middle of north Johnston Island where an aborted launch of a nuclear warhead caused localized contamination with plutonium during the 1960s. Site 4 (Burn pit) was north of Eastern Island and was adjacent to an old burn pit where plastics and other materials were incinerated in open air. Sites 5 and 6 (Signal Tower and Donovan's reef, respectively) were "control" areas (Fig. 1).

Manta tows were done according to methods of English et al. (1994). Briefly, a diver was towed at low speeds ca. 30-40 feet behind a boat for 2 minute intervals at the end of which the diver recorded the bottom type (rubble, sand, coral), estimated percent coral cover by category (0-10%, 10-25%, 26-50%, >50%), and dominant coral genera (*Acropora* sp., *Montipora* sp. or *Pocillopora* sp.). For *Acropora* sp., the diver categorized lesions into 5 groups (see results). After each two-minute manta tow interval, the diver recorded the number of each type of lesion. Manta tows proceeded perpendicular to the long axis of Johnston Island and coordinates of way points were collected at the start and end of every 2 minute tow using a Garmin GPS 12 unit. Spot dives were done using SCUBA in Sites 1, 3, 5 and 6 and snorkel for the remaining sites. For areas 2 and 4, we snorkeled a network of parallel lines close to shore. Scuba surveys consisted of swimming in a haphazard pattern looking for lesions.

Corals were photographed using a Nikonos V underwater camera with a 20 mm lens and twin Ikelite 50 strobes or a digital camera in an underwater housing. Close-up photos were taken with a Nikonos V camera with a single Ikelite 50 strobe and a 2:1 extension tube. Coral samples were taken using bone shears, or hammer and chisel, and placed into labeled plastic bags in seawater. Corals were preserved in Helleys fixative (Barszcz and Yevich, 1975) with added salt and allowed to fix for 24 hr. The fixative was decanted and the coral rinsed with fresh water once every 12 hr for 24 hr. Subsequently coral was stored in 70% ethanol, decalcified with Cal-ex II (Fisher Scientific), placed in cassettes, processed for paraffin embedding, trimmed at 5 um, and stained with hematoxylin and eosin. Slides were examined using light microscopy at magnifications ranging from 20-1000X.

Twenty fragments of normal *A. cytherea* were collected for pigment analyses. Coral fragments were weighed (nearest 0.001 g), placed in 50 ml of 100% methanol (n=10) or 0.6M phosphate buffer pH 8 (n=10), and extracted in the dark at 4C for 24 hr. The supernatant was decanted, centrifuged (14000g) for 5 minutes, and stored at –190C. The extract was filtered using 0.45 μ syringe filters and scanned from 200-700 nm using a Spectronic Genesys 8 Spectrometer. Methanol extracts were for chlorophyll pigments, and phosphate buffer extracts for pigments insoluble in methanol.

RESULTS

Surveys

Coral cover seemed uniform over most of the lagoon but appeared denser away from the dredge channel (Fig. 2). Coral cover was relatively sparse east of Johnston Island. *Acropora* (*mostly A. cytherea*) was more dominant towards the reef edge, and *Montipora* sp. was more

dominant near the ship channel. *Pocillopora* sp. appeared more numerous east of Johnston Island (Fig. 3.).

Normal morphology

Acropora cytherea

Normal *A. cytherea* colonies formed wide flat tables (Fig. 4A) with small branches containing numerous tan-brown to tan-pink exsert axial corallites (Fig. 4B). On microscopy, the epithelium overlying was composed of an intact continuous layer of columnar cells with basally located nuclei. These were interspersed with occasional intracytoplasmic basophilic granular cells, mucus cells, and rare isolated spirocysts (a type of nematocyst) (Fig. 4C). Epithelium overlying tentacles contained numerous batteries of spirocysts mixed with barely visible holotrichous isorhizas. Spirocysts became less numerous and eventually absent in the stomodeum, the epithelium of which was lined by closely apposed ciliated columnar epithelium. Mesoglea was uniformly thin except near the base of tentacles where it thickened and contained prominent mesogleal pleats; within the gastrovascular canal, mesoglea was not discernable.

Coenosarc gastrodermis was composed of columnar and mucus cells mixed with focally aggregated zooxanthella. In tentacles, gastrodermis was thickened and contained clusters of zooxanthella. Within the gastrovascular canal, gastrodermis was focally hyperplastic and contained rare zooxanthella. The gastrodermis was either closely apposed to calicoblast or contained prominent mucus cells, giving it a foamy appearance. Mesenteric filaments were of two types. One consisted of densely packed columnar cells with closely apposed nuclei just below the cnidoglandular cap, giving the filament a distinct pattern (Fig. 4D). Other filaments contained prominent mesogleal pleats, scattered eosinophilic granular cells, and mucous cells giving them a moth-eaten appearance. Occasional filaments at the base of the branch contained ovarian tissue. Cnidoglandular caps of all filaments contained eosinophilic granular cells. The calicoblast was uniformly cuboidal to squamous, closely apposed to gastrodermis, and contained scattered desmoid processes.

Montipora patula

Colonies were plate-like to encrusting, with purple polyps interspersed among a network of yellow trabeculae (Fig. 4E). Epithelium overlying coenosarc was composed of a single layer of columnar cells with basal nuclei and interspersed with occasional mucus cells. Epithelium overlying tentacles contained localized batteries of spirocysts (Fig. 4F), which gave way to small clusters of eosinophilic granular cells in the stomodeum.

The mesoglea was barely visible in the coenosarc and tentacles, but became more prominent along with mesogleal pleats within gastrovascular canals at the base of polyps. Occasional eosinophilic granulocytes characterized by cells with an eccentric nucleus and distended by intracytoplasmic accumulations of brightly eosinophilic granules were seen within the mesoglea of the gastrovascular canal.

The coenosarc gastrodermis was cuboidal, contained numerous zooxanthella, and was closely apposed to squamous calicoblast layer. Within gastrovascular canals, gastrodermal cells were cuboidal or focally hyperplastic and apposed to squamous calicoblastic cells. The gastrodermis contained scattered zooxanthellae. Mesenteric filaments deep within canals contained batteries of macrobasic mastigophores (a type of

nematocyst). Other mesenteric filaments were composed of closely apposed columnar cells mixed with mucus cells and eosinophilic granular cells. Occasional ova were noted within filaments. The skeleton contained mats of gray to amphophilic branching filaments (probable endolithic algae).

Montipora capitata

Colonies were placoid to branching, brown, with a verrucose surface containing polyps interspersed among a network of trabeculae. Epithelium overlying coenosarc was simple columnar interspersed with occasional clear mucus cells. Epithelium overlying tentacles was pseudostratified columnar with numerous large glandular cells giving it a vacuolated appearance. Batteries of spirocysts were noted near the base of tentacles, and these disappeared in the stomodeum and were replaced by closely packed columnar cells with a ciliated surface. Deeper into the stomodeum, there were clumps of eosinophilic granular cells near the central lumen. Mesoglea was uniformly thin and bereft of cells except near base of polyps or near mesenteric filaments where mesogleal pleats were noted.

The coenosarc gastrodermis was simple to pseudostratified cuboidal and contained few zooxanthellae. Zooxanthellae were much more numerous within gastrodermis of tentacles. Within gastrovascular canals, gastrodermis was squamous and focally hyperplastic with few zooxanthella and occasional ovarian tissue. Mesenteric filaments were composed of closely apposed columnar cells interspersed with aggregates of red granular cells at cnidoglandular caps, and occasional mucus cells giving the filament a moth-eaten appearance. Batteries of macrobasic mastigophores were occasionally noted within the gastrodermis. Planulae, characterized by three layers of cells (ciliated columnar cells, mesoglea, and gastrodermis) surrounding a lumen were sometimes seen in the gastrovascular canal. Calicoblastic epithelium was uniformly squamous.

Pocillopora eydouxi

Colonies were composed of tan to cream upright flattened branches with a verrucous surface containing haphazardly arranged corallites (Fig. 7 C-D). Coenosarc epithelium was composed of a single layer of ciliated columnar cells. Over the tentacles, columnar cells were prominent and mixed with batteries of spirocysts, which disappeared within the stomodeum and were replaced by closely apposed ciliated columnar cells. Coenosarc mesoglea was thin and not discernable but widened and contained mesogleal pleats, particularly around polyps and within tentacles.

Gastrodermis was squamous to cuboidal and contained focally aggregated zooxanthellae, which were more numerous within tentacles. Gastrovascular canals formed a single layered parallel network below the external epithelium. Mesenteric filaments consisted of closely apposed columnar cells mixed with eosinophilic granular and mucus cells with rare zooxanthella. The calicoblast was squamous with focal hypertrophy where cells took on a columnar appearance.

Lesions:

Any anomaly in gross skeletal or tissue morphology was classified as a lesion. Lesions in *A. cytherea* were categorized into five groups (Fig. 5): purple bleaching, yellow bleaching, brown blotch, growth anomalies, and brown band. Purple bleaching (Fig. 5A-B) referred to areas of coral that were bleached with a raised light to dark purple margin, giving the entire lesion a bluish hue. This lesion usually surrounded a variably

sized central area of dead coral overgrown by algae. Polyps near the edge of these lesions appeared atrophied or entirely missing. Yellow bleachings (Fig. 5C-D) designated dwell-defined areas of bleached coral with a yellow hue and no distinct raised margin. These areas were separated from live coral and often had incipient algal growth on coral tissue. Brown blotch corresponded to single well-defined circular dark brown lesions on coral tables, usually near the middle of the plate, and was characterized by a film of brown mucoid material surrounded by apparently normal coral tissue. Growth anomalies (Figs. 5E, 6, 7A-B) included any abnormal growths of the coral skeleton. Brown bands (Fig. 5F) were slice-shaped areas of dead coral overgrown with algae demarcated from adjacent normal coral by a distinct band of bleached tissue

Growth anomalies in *A. cytherea* were of 3 types: Grossly, type 1 (Figs. 5E, 6A-B) growths were focal to coalescing, white to pink, and rugose, and tissue covering these masses was smooth and bereft of polyps. On histology (Fig. 6C), coenosarc epithelium was markedly hyperplastic and characterized by numerous pseudostratified columnar cells. Polyps were absent or manifested by rare batteries of spirocysts. The mesoglea was enlarged, and gastrodermal cells adjacent to epithelium were markedly hyperplastic to anaplastic, pleomorphic, and characterized by stellate nuclei, and widened intercellular spaces. Gastrodermis of gastrovascular canals was focally hyperplastic with clumps of proliferating cells free or projecting within the lumen of canals. Based on these characteristics, this lesion was considered neoplastic and classified as a gastrodermal neoplasm. Zooxanthellae were rarely seen. In some instances, calicoblast cells appeared enlarged and hypereosinophilic with swollen nuclei. Rare mesenteric filaments containing macrobasic mastigophores were noted.

Type 2 growths (Fig. 6D-E) were focal to coalescing, pink, with numerous cylindrical tubercles with an apical indentation. Tissue overlying these growths was generally bereft of polyps save for occasional single tentacles. On histology, (Fig. 6F) there was marked hyperplasia of coenosarc epithelium with cleft formation and thickening of mesoglea. Coenosarc gastrodermis was composed of simple columnar cells and that of gastrovascular canals was cuboidal and focally hyperplastic. Zooxanthella and mesenteric filaments were rarely seen. Calicoblast layer was uniformly squamous.

Type 3 growths (Fig. 7A) consisted of smooth, well-defined, sessile white firm growth arising from normal skeleton. Tissue overlying these growths lacked zooxanthella. On histology, tumor tissue was characterized by a markedly hyperplastic epithelium bereft of polyps. Coenosarc gastrodermis was largely bereft of zooxanthella and focally hyperplastic with stellate nuclei. Within gastrovascular canal, gastrodermal cells, particularly those associated with mesenteric filaments, were hyperplastic, fibroblast-like and pleomorphic forming papillary projections into the gastrodermal cavity. Myonemes within occasional mesenteric filaments were enlarged and occasionally diffusely necrotic. Diffusely, calicoblast was squamous and focally hyperplastic. Based on pleomorphic appearance of gastrodermal cells and cellular necrosis, these growths were classified as gastrodermal neoplasms.

Pocillopora eydouxi

Grossly, lesions were characterized by exuberant growth of skeleton overlaid by apparently normal tissue (Figs. 7D-E). These were seen in one location on the north lagoon and Site 3. On histology, no significant lesions were noted save for markedly

atrophied epithelium and absence of polyps and zooxanthella within coenosarc. One normal appearing colony from the northern lagoon that contained variably sized, well defined, round aggregates of deeply basophilic filamentous organisms (probable bacteria) within tentacular epithelium (Fig. 7F).

Montipora patula, Montipora capitata

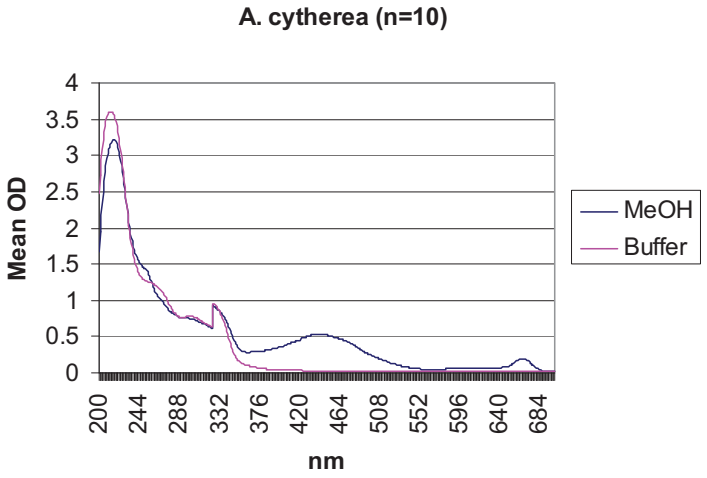
Grossly, lesions were characterized by single to coalescing well-defined firm smooth to rugose white nodules ranging in size from ca. 0.5-5 cm diameter. In some instances, the center of the lesion was ulcerated and colonized by filamentous algae. These lesions were seen sites 1-4 and did not appear particularly numerous at any one site (Fig. 8A-B, D-E).

On histology, large mats of variably sized eosinophilic filamentous organisms displaced coral tissue. Epithelium of adjacent coral was cuboidal to simple columnar, and gastrodermal cells were hyperchromatic with shrunken cytoplasm. The mixed population of filamentous branching structures was largely limited to the skeleton and ranged in invasiveness from localized penetration into gastrovascular canals to complete invasion and effacement of tissue architecture. In some cases, the filamentous material encompassed fragments of degenerating gastrodermis. In other cases, these nodules were composed of granular gray material overlaid by cells with cell walls (plant material) (Fig. 8C). The center of the nodule occasionally contained an invertebrate (metazoan) of unknown identity (Fig. 8F).

Distribution:

Lesions (all types) in *Acropora* cytherea were more numerous in the areas where this species was dominant (Fig. 9). Purple bleaching, yellow bleaching, brown band, growth anomalies, and brown blotch made up 57%, 38%, 2%, 2%, and 0.5%, respectively of the total (555) number of lesions seen. There did not appear to be a pattern to the distribution of the major types of lesions (Figs. 10-12). In addition to manta tow survey areas, purple and yellow bleaching was seen during spot dives in sites 1-5 and growth anomalies were noted in sites 1 and 2 (Fig. 1)

Pigment profiles



Peaks at ~230, 290, and 325 nm were seen for both methanol and buffer soluble pigments while peaks at 441 and 665 (Chlorophyll) were seen in methanol extracts only.

DISCUSSION

Distribution of corals and lesions

Distribution of corals in our survey area appeared similar to that observed by Maragos and Jokiel (1986). Although a variety of lesions were seen in different coral species, no distinct distributional pattern was recognized. The distribution and extent of lesions in *A. cytherea* was a function of coral abundance, and the most widespread lesion (purple bleaching), appeared to be a normal process ofsenescence, because this lesion was usually located adjacent to dead coral near the center of the plates. This area of dead coral seemed to expand as the plates became larger, whereupon they would collapse. Diffuse purple bleaching was noted on the underside of many collapsed colonies. Yellow bleaching was associated with incipient algal overgrowth and was noted wherever *A. cytherea* was present, including deep (80 ft.) habitats such as Donovan's reef. Contrary to expectations, skeletal growth anomalies in *A. cytherea* and other species of corals did not appear concentrated in areas contaminated with tumor-inducing compounds such as organohalogens (Site 2) or plutonium (Site 3). Although rare growth anomalies in *A. cytherea* were seen in Site 2, coral in the general area appeared particularly healthy.

Growth anomalies were noted in 4 species of corals (*A. cytherea, P. eydouxi, M. patula, and M. capitata*), however, in only two instance were these growth considered neoplastic (cancerous). Documented instances of neoplasms in corals are rare, and those that are documented mostly involve description of skeletal growth anomalies in absence of histology (Peters et al., 1986). In the most complete description of growth anomalies in a coral, Peters et al. (1986) concluded that growths in *A. palmata* were calicoblastic epitheliomas based on locally invasive skeletal protuberances with associated proliferation of calicoblast and associated tissues. In vertebrates, neoplasms are defined by uncontrolled proliferation of tissue with cells that exhibit abnormal morphology including nuclear or cytoplasmic polymorphism and, on occasion, mitotic figures and tissue necrosis (indicating rapid cell division) (Cheville, 1988). This is opposed to hyperplasia where cell growth is excessive but controlled, and where cell morphology is not abnormal. The definition of neoplasias in invertebrates is less clear, and some cases of neoplasia in invertebrates could also be interpreted as hyperplasia (Sparks, 1985).

In this study, growth anomalies needed to fulfill two criteria to warrant a diagnosis of neoplasia: 1) abnormal and excessive growth of skeleton and 2) cellular proliferation accompanied by cell anaplasia and polymorphism. In *A. cytherea*, only two growth anomalies (Type 1 and 3, Figs. 11 A-B) were neoplastic. The other growth anomalies (Type 2) were classified as hyperplasia. No organisms were seen in tissue associated with growth anomalies in *A. cytherea*, and the cause of such lesions remains unknown. Interestingly, tumors that appeared different in gross appearance (Types 1 and 3) were both considered neoplasm of the same tissue type (gastrodermis). Tumors similar in appearance to Type 3 growths were seen in a tabular *Acropora* from a shallow site in an embayment in Oman and were classified as calicoblastic neoplasms (Coles and

Seapy 1998). Given the simple anatomic plan of corals (4 tissue layers), it is perhaps not surprising that the same tissue type would give rise to different tumor morphs. There is, however, a clear need for continued refinement of classification of coral neoplasms. Future investigations might focus on molecular markers for different tissue types in corals to better differentiate the tissue origin of tumors. Given the uncertain state of classification of coral neoplasia, speculation as to potential causes of these lesions seems unwarranted. In higher organisms, causes of neoplasia typically include viruses, ongoing damage to DNA from chronic trauma or senescence, and certain environmental contaminants.

Skeletal growth anomalies in *Montipora* sp. were responses to foreign agents including fungi, algae, and intra-skeletal metazoans (invertebrates). The coral skeleton harbors a variety of metazoans, filamentous algae, and fungi, and in most cases, these do not invade coral tissue. Le Campion-Alsumard et al. (1995) demonstrated that invasion of tissue by these organism is kept in check by a continuous process of mineralization. In some cases, however, this process fails and algae and fungi colonize live tissue. Interestingly, tissue reaction (necrosis) was minimal in spite of invasion of the gastrovascular canal by algae. Colonies of bacteria were seen in tissue associated with skeletal anomalies in *P. eydouxi* , however, this was not consistent. Thus, the cause of such anomalies in this species remains unknown.

In the pigment profiles for A. cytherea, peaks in the methanol extracts at 441 and 665 nm corresponded to chlorophyll (Dustan, 1979). Various coral proteins (UV pigments)were probably responsible for the peaks below 400 nm (Dove et al. 1995). Fluorescent pigments have been shown to be protective against sunlight for several species of Australian corals (Salih et al. 1998; 2000) and determination of fluorescent pigment properties may prove useful in future investigations of coral health. This pigment profile should provide a baseline to investigate future events of bleaching or pigment abnormalities in *A. cytherea*.

RECOMMENDATIONS

1. There needs to be a better method to quantify lesions on reefs during broad scale surveys. Surveys of lesions are complicated by the three dimensional nature of coral reefs and the intermixing of species. Ideally, it would be useful to have percent coverage of different species. Surveys would be targeted in such a way as to calculate percent of area of coral x covered by lesion y. Possible tools to consider for this include quadrats or video transects.
2. Pigment profiles of corals with lesions, particularly growth anomalies, should be evaluated to further define whether pigments or their absence plays a role in genesis of these anomalies.
3. Consider measuring growth anomalies using manual methods or photogrametry and following growth through time (months, years) to determine whether or not they are growing uncontrollably or remain unchanged.
4. Additional criteria are needed to determine exactly what constitutes neoplasia in corals.

5. Most efforts on this trip focused on growth anomalies. It would be useful to examine other lesions in *Acropora* (brown blotch, yellow bleach, purple bleach, brown band) in more detail to evaluate potential causative factors.

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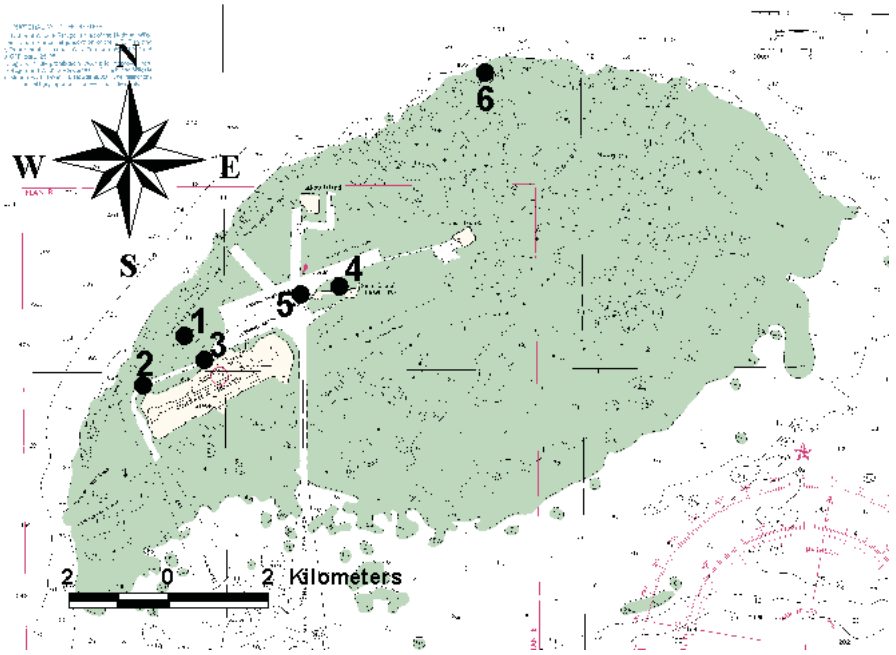

Figure 1. Johnston Atoll: Location of spot dives (see methods for details).

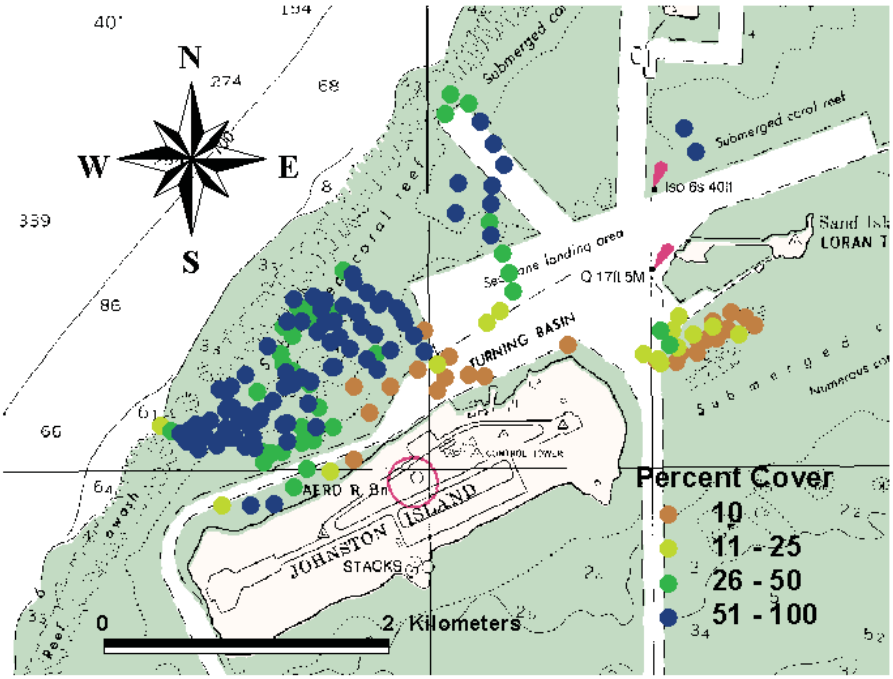

Figure 2. Percent coral cover observed during manta tows of North lagoon, Johnston Atoll.

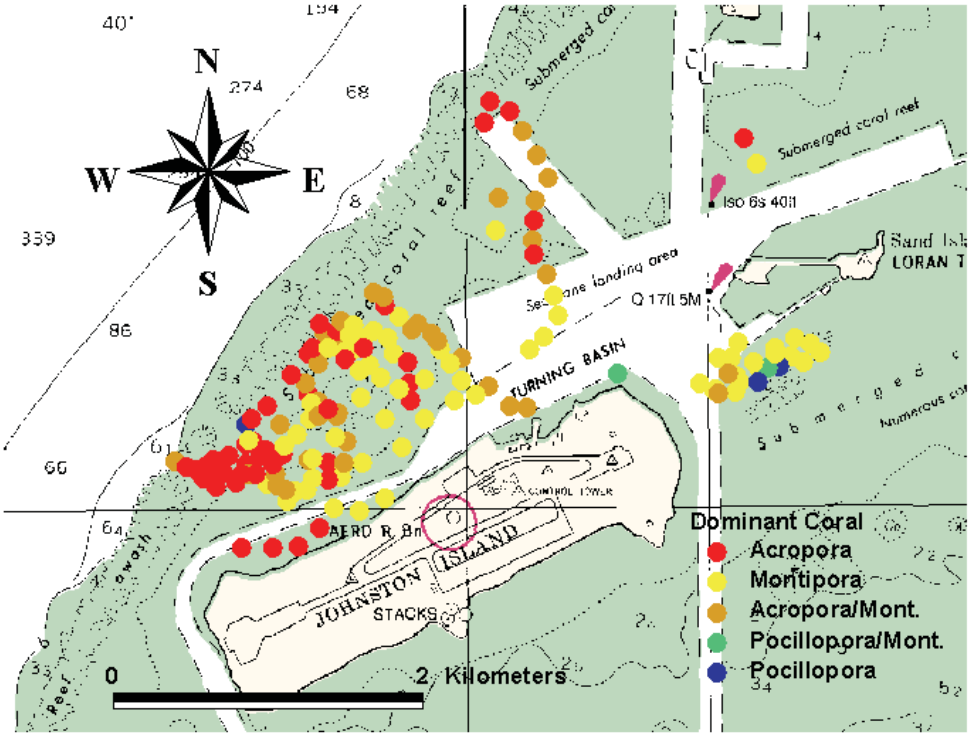

Figure 3: Dominant species of corals seen during manta tows of north lagoon.

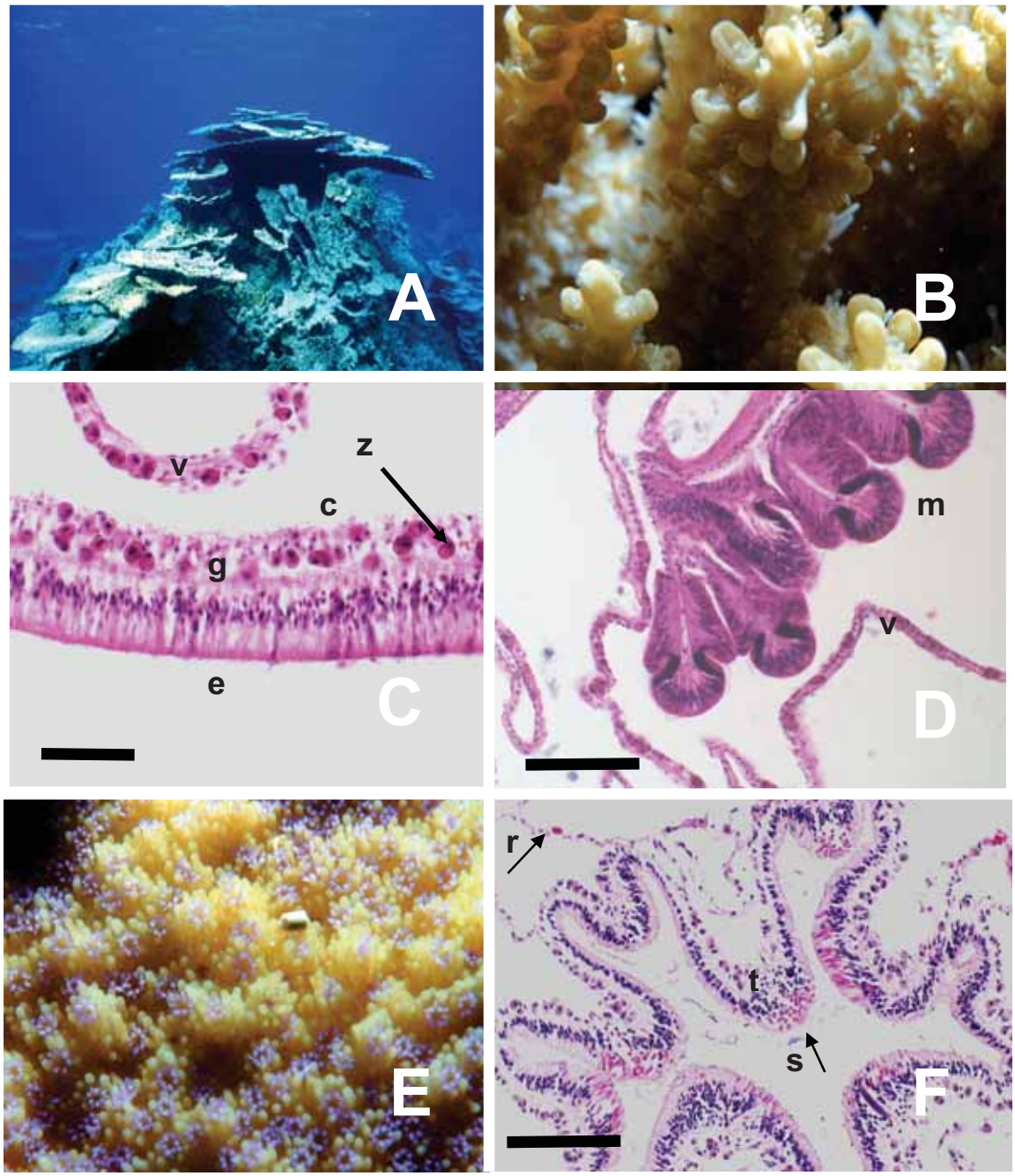

Figure 4. A-D) *A. Cytherea*; C) Coenosarc, bar= 50 µm; D) Gastrovascular canal (v) and mesenteric filaments (m), bar=100 µm; E-F) *M. Patula*; F) Polyp, bar= 100 µm. Epithelium (e), gastrodermis (g), calicoblast (c), zooxanthella (z), mesenteric filaments (m), tentacle (t), spirocysts (s), eosinophilic granulocyte (r).

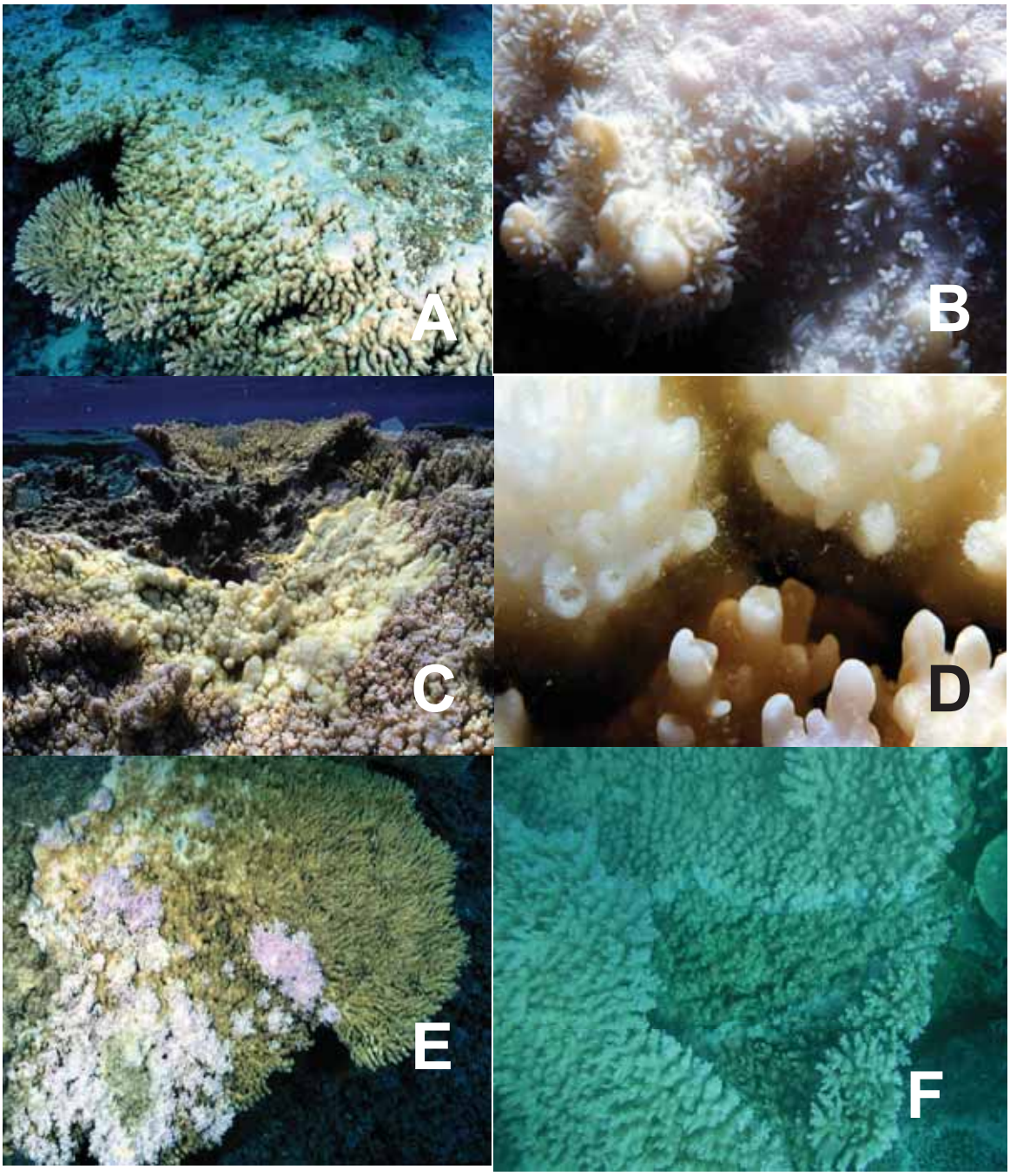

Figure 5. ***A. cytherea.*** **A) Purple bleaching; B); Purple bleaching-note atrophied polyps; C) Yellow bleaching; D) Yellow bleaching-note algal filaments; E) Growth anomalies; F) Brown band.**

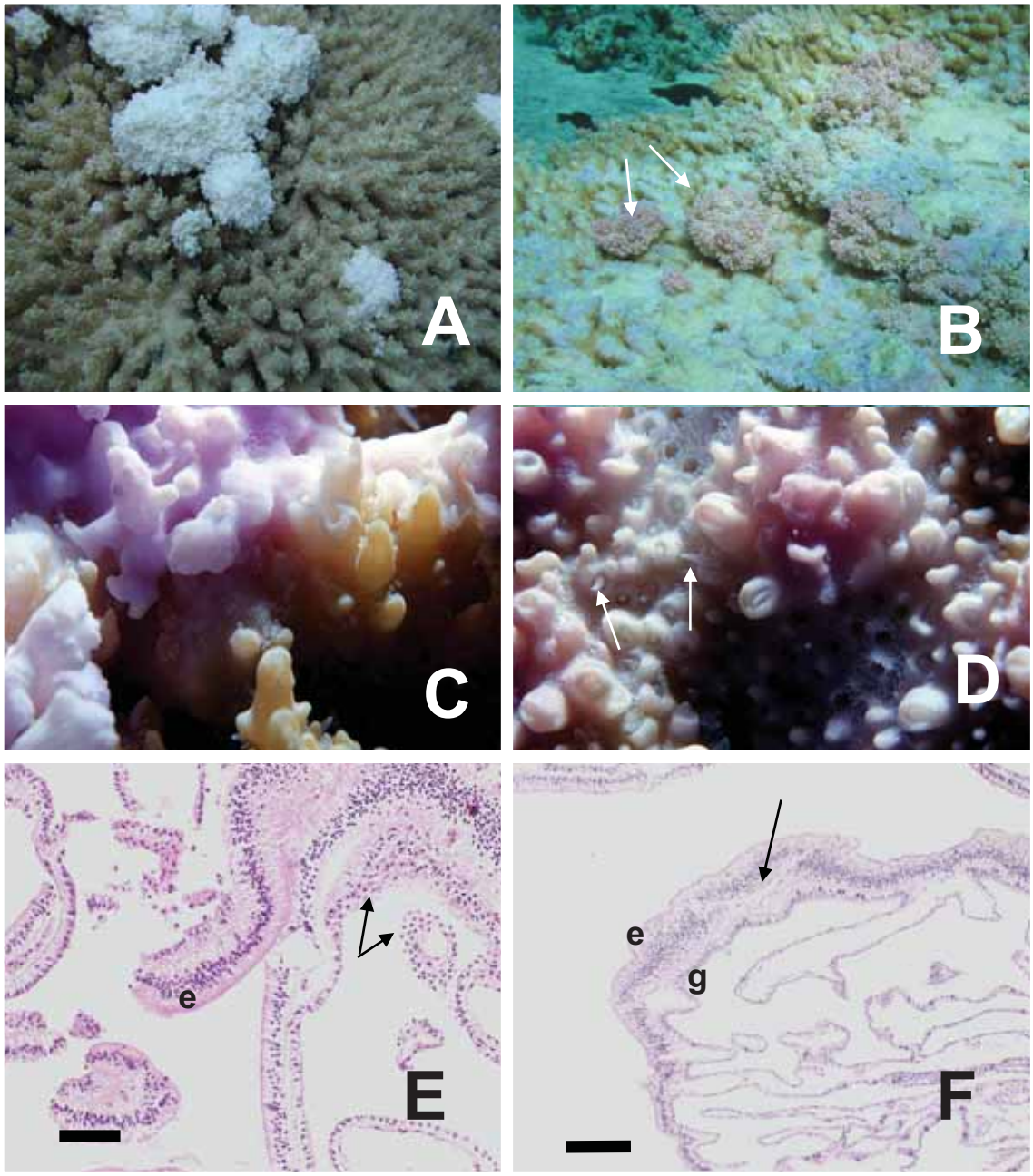

Figure 6. ***A. cytherea.*** **A-B) Type 1 growths, note absence of polyps; C) Note markedly hyperplastic epithelium and pleomorphic and hyperplastic gastrodermis (arrows), bar = 50 µm. D) type 2 growths (arrows); E) note malformed polyps with single tentacle (arrows); F) Note markedly hyperplastic epithelium with cleft formation (arrow) bar=200 µm. Epithelium (e), gastrodermis (g).**

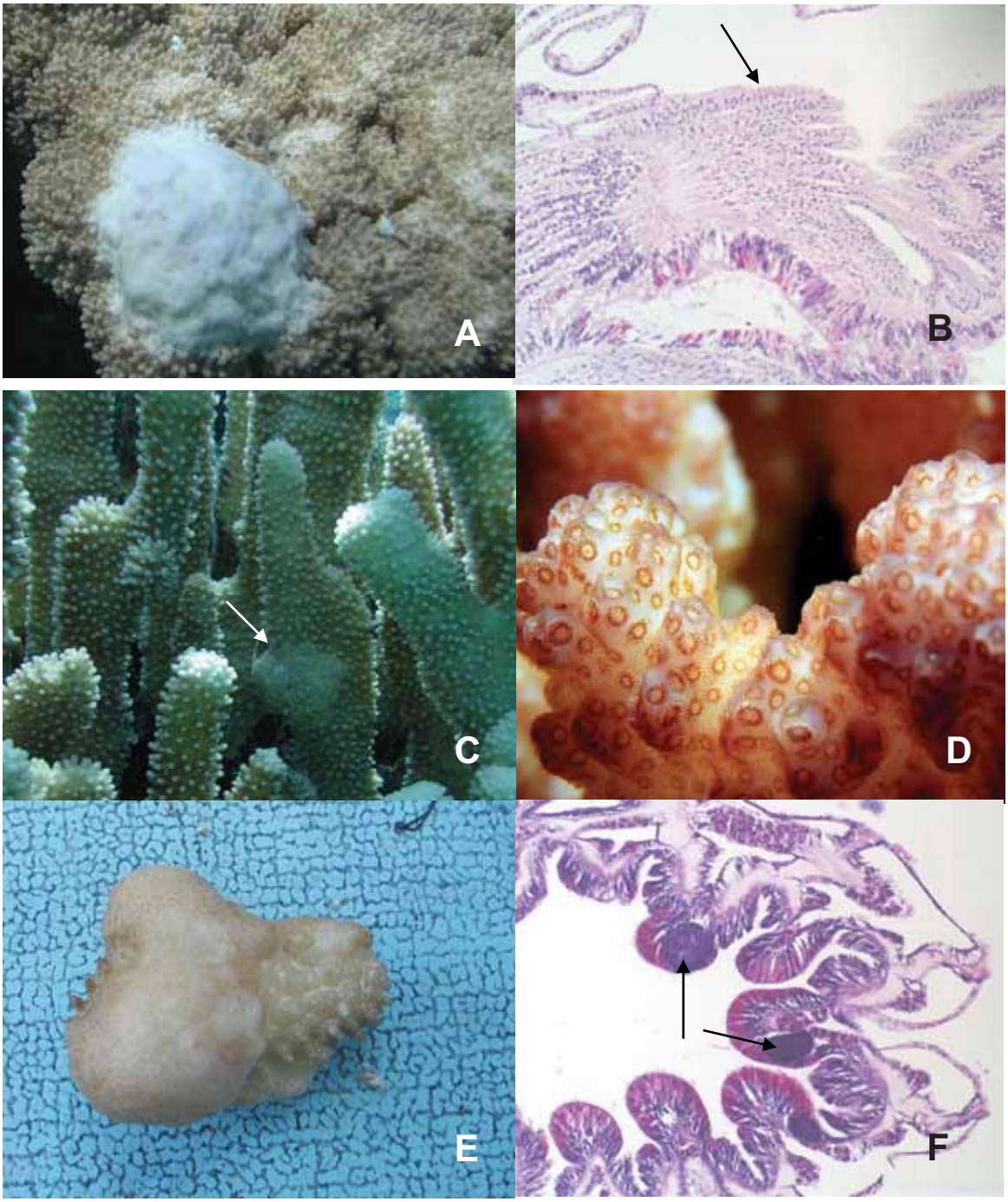

Figure 7. A-B) *A. cytherea*; D-F), *P. eydouxi*; A) Type 3 growth; B) Note marked pleomorphism of gastrodermal cells in tumor tissue (arrow), bar= 100 µm. C, E) growth anomalies (arrow) and normal coral (D); F) Polyp of normal appearing P. eydouxi, note bacterial colonies in epithelium (arrows) bar=50 µm.

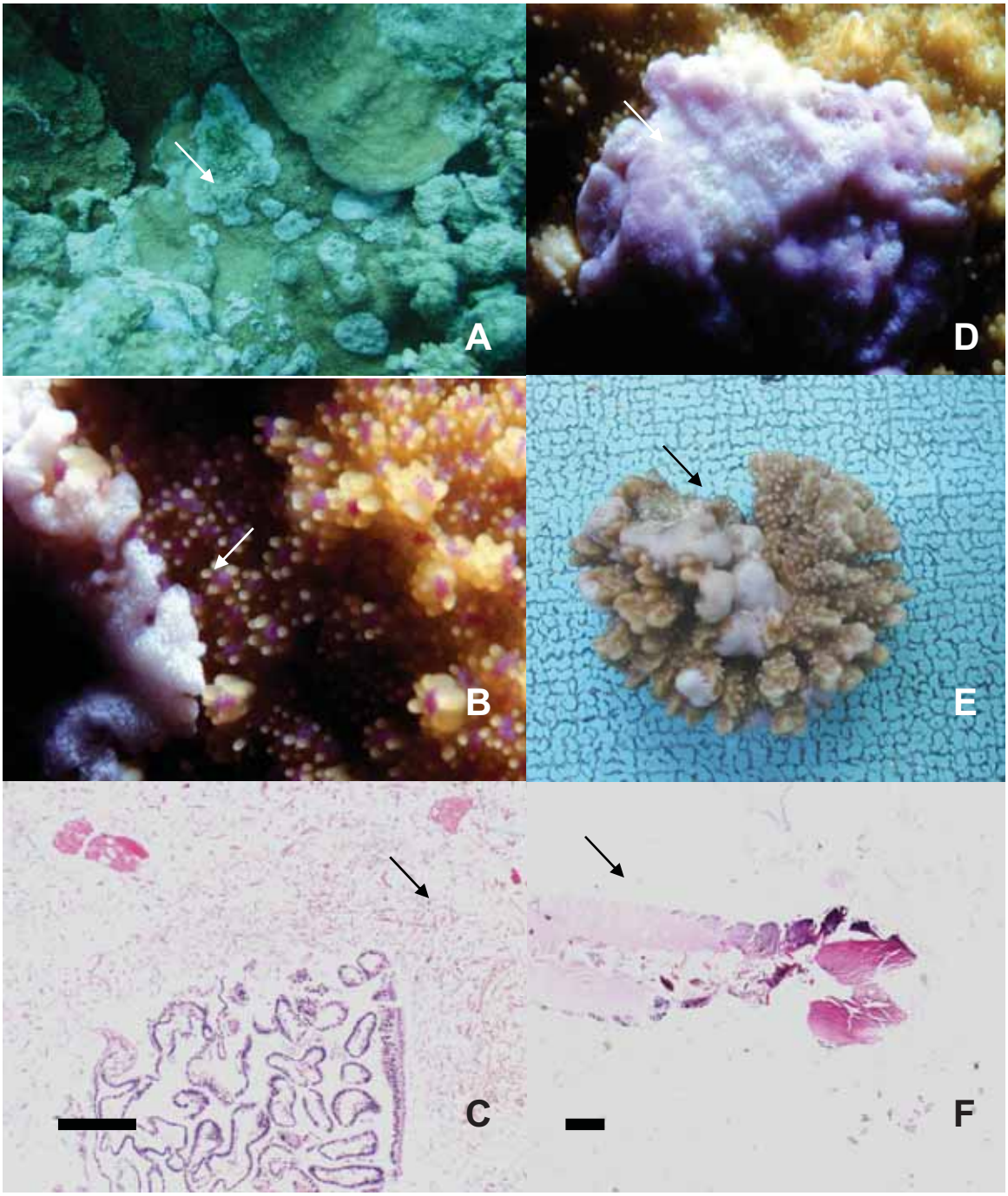

Figure 8. A-C) *M. patula*, D-F) *M. capitata*; A-B) growth anomalies (arrow); C) note invasion of skeleton and tissue with filamentous organisms (algae and fungi) arrow, bar= 200 µm; D-E) growth anomalies; F) note cross section of metazoan (arrow), bar=200 µm.

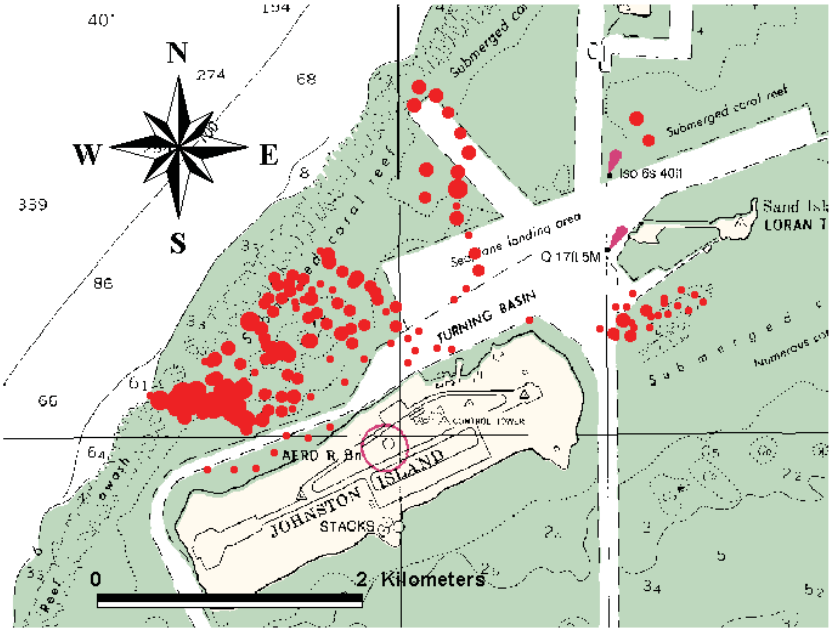

Figure 9. Number of lesions in *A. cytherea* observed during each manta tow interval.

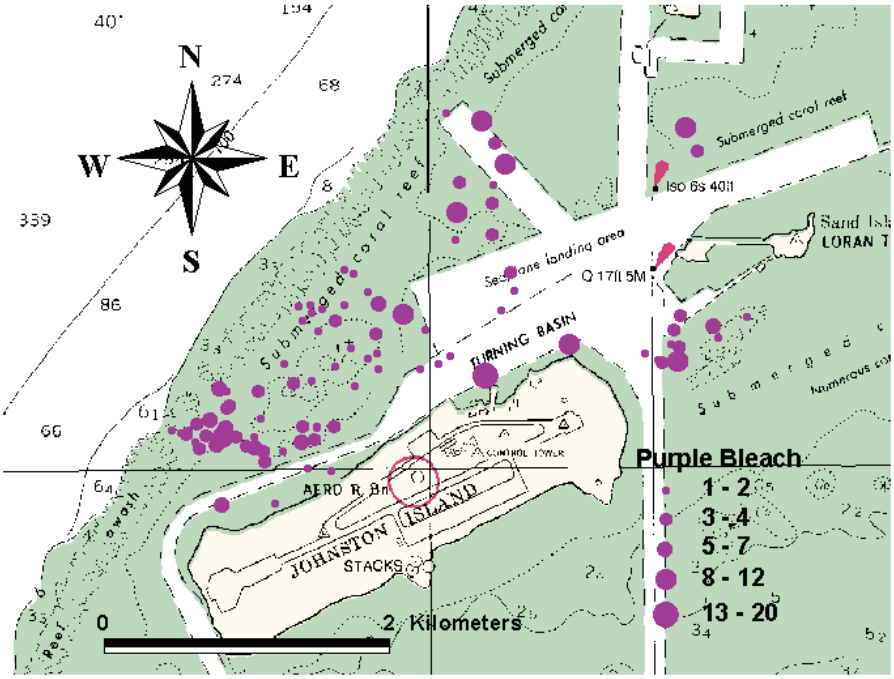

Figure 10. Number of instances of purple bleaching during each manta tow interval.

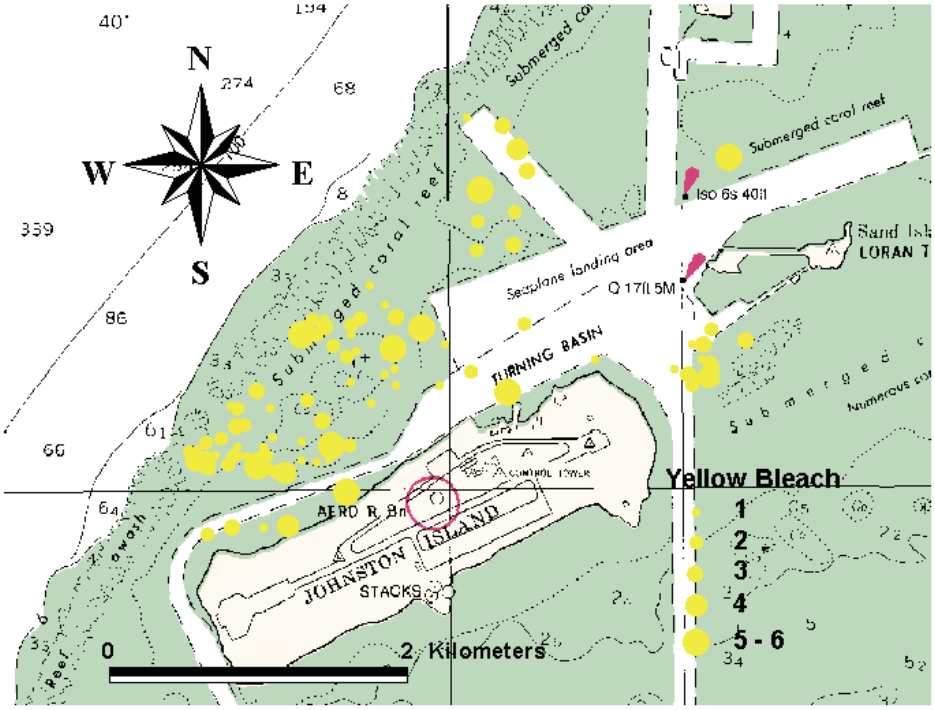

Figure 11. Number of instances of yellow bleaching seen during each manta tow interval

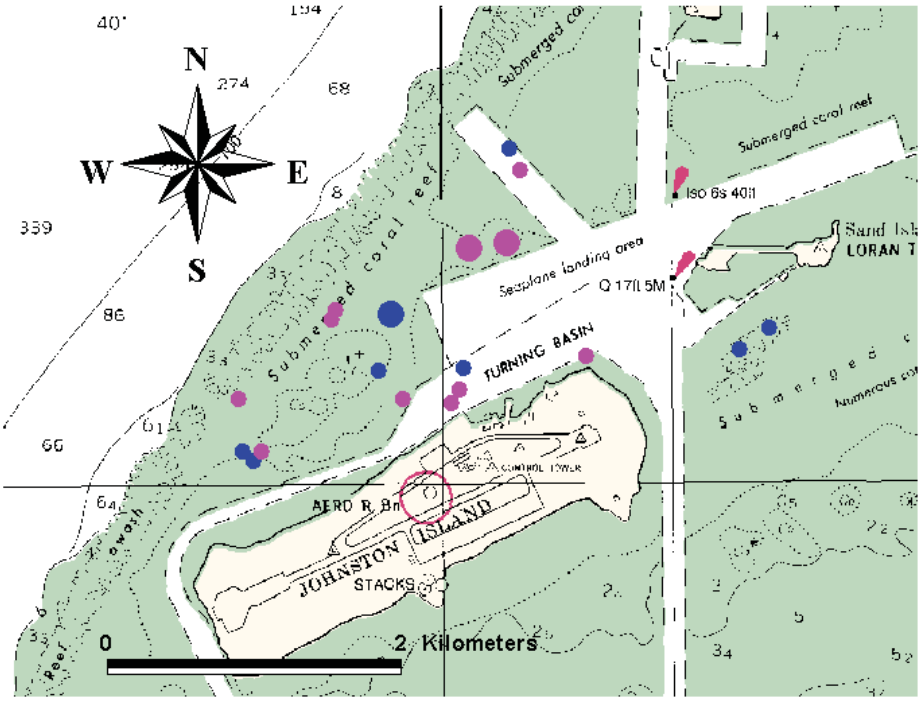

Fig 12. Number of instances of aberrant growth (blue) or brown band (violet) seen during each manta tow interval. Large dots=2, small dots=1.

CORAL DISEASE ON THE GREAT BARRIER REEF AND IN THE WESTERN PACIFIC

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Background

Until recently, it was assumed that disease has had little impact on the population dynamics or community structure of coral assemblages on the Great Barrier Reef (GBR). However, prior to 2000 there were only two studies of coral disease in the region, one focused on black band disease (BBD; Dinsdale 2002) and the other on skeletal eroding band (SEB; Antonius 1999; Antonius and Lipscomb 2001), both undertaken at Lizard Island in the northern sector of the GBR. Further anecdotal reports of BBD (Miller 1996) and a white disease (Baird 2000), plus increasing abundance of white syndrome (described below) detected in a Long Term Monitoring Program begun in the early 1990's by the Australian Institute of Marine Sciences (AIMS) (Willis et al. 2004), highlighted the need for a more in-depth study of coral disease on the GBR.

As an initiative of the GEF Coral Disease Working Group (DWG), surveys of coral disease were commenced in 2002 to more systematically assess the types and prevalence of coral disease on the GBR (Willis et al. 2004). Detection of some of the more common and infectious Caribbean diseases, in combination with discovery of diseases unique to the region (brown band disease: BrBD; Willis et al. 2004), suggested that coral disease occurs commonly on Indo-Pacific reefs and may have a greater role in structuring coral communities in the region than previously thought. Accordingly, a 7 year program to assess the ecological significance of coral disease on the GBR has been funded by the Australian Research Council (ARC). Results summarized below describe progress 2.5 years into the program. In collaboration with the GEF DWG, survey protocols and knowledge gained on the GBR have been applied to reefs throughout the Western Indo-Pacific, including Palau, the Philippines, Marshall Islands, Papua New Guinea and Indonesia, plus imminently Zanzibar, Tanzania, Kenya, Mauritius, Madagascar, Seychelles and the Comoros as a consequence of a recent GEF Workshop on Coral Disease for local reef scientists and marine park managers (Zanzibar, April 2006).

Survey design, transect protocols and targeted disease studies

Given the lack of baseline knowledge of coral disease on the GBR and the vastness of a reef system spanning north-south gradients more than 2000 km in length and cross-shelf (east-west) gradients in terrestrial influences up to 100 km across the

continental shelf, the region was divided into 3 sectors (northern, central and southern GBR) to optimize sampling a range of habitats and reef types. The sampling design comprises 3 belt and line intercept transects, at each of 2 sites, on each of 3 replicate reefs, in surveys at each of 3 cross-shelf positions (inner-, mid- and outer-reef positions) in the northern and central sectors, and at 1 cross-shelf (outer-reef) position in the southern sector. Transects are surveyed in summer, on the upper reef slope (typically 4-6 metres) where species diversity tends to be highest. In total, 19 reefs have been surveyed annually since 2004 and 21 reefs since the full design was inaugurated in 2005. Surveys of 2 reefs in the northern sector in both summer and winter since 2002-03 have shown a clear pattern of increased disease prevalence in summer (Willis et al. 2004).

The survey protocol developed to cope with species-rich, high-cover coral communities characteristic of Indo-Pacific reefs is based on a 20m x 2m belt transect combined with a 20m line intercept transect (LIT) to produce a concurrent estimate of percent coral cover. Belt transects of this size represent an efficient compromise between the need to survey a representative proportion of the species diversity and time constraints imposed by SCUBA surveys. Three replicate belt and LIT transects are typically completed within 1-2 dives (depending on percent cover) by a team of 2-3 experienced divers. All colonies within the belt are examined, recorded as healthy, diseased or showing signs of compromised health (see final section below) based on macroscopic field signs, and identified to genus (plus growth form for the genus *Acropora*) or family for less common groups. Diseased colonies are identified to species, photographed, and samples collected for microscopic examination and histological investigation as appropriate. In selected cases, typically when disease outbreaks are encountered, samples for microbiological and molecular studies are collected to isolate and identify pathogens. As evidence identifying the most common and virulent disease types has accumulated, more in-depth studies of selected disease types (SEB, WS, BBD, BrBD, AtN) have been initiated to quantify rates of progression across colonies, impacts on coral growth and reproduction, spread throughout populations and the effect of elevated temperature on these rates.

Broad ecological surveys inform detailed studies of lesions, pathogens and population impacts

The focus of the ARC-funded program is changing from its current emphasis on broad ecological surveys to more in-depth manipulative, microbiological and molecular studies of selected disease types as the research progresses. The objective is to use ecological surveys to provide the context for selection of disease types for more focused research. his overarching research plan recognizes the need to start with imperfect tools, i.e. observations of macroscopic disease signs underwater, as the first step in progressing research on under-studied Indo-Pacific coral diseases. Through extensive field surveys at diverse sites and through time, experienced coral biologists are well-placed to make informed decisions about the most prevalent and/or virulent disease types that should be targeted for further study. To carry this research to the next level, collaboration with biomedical histologists, microbiologists and molecular biologists have been developed. As knowledge of disease types is refined, diagnostic tools can be developed that will, in turn, refine ecological surveys. The importance of maintaining long-term monitoring programs cannot be over-stated, as these are the key to determining whether disease

incidence is changing through time and for developing hypotheses in relation to potential drivers.

In recognition of the early stage of Indo-Pacific coral disease research, we advocate the use of this integrated, collaborative approach to advance current understanding of coral diseases in the region. It is hoped that this meeting will be instrumental in developing further collaborations and a unified approach among research teams throughout the Indo-Pacific to strengthen and coherently build current understanding of coral disease in the region.

Recognizing and standardizing the naming of Indo-Pacific disease types

The issue of naming diseases when little is known apart from macroscopic and microscopic signs of disease is problematic. We support schemes under development to apply a structured approach to the description of gross lesions and the naming Indo-Pacific coral diseases (T. Work, G. Aeby, pers. comm.), with one additional pragmatic consideration. Whilst it would be ideal to incorporate the host species or genus of coral into each disease name, this approach becomes cumbersome when dealing with the more than 580 species and 200 genera of Indo-Pacific corals. An approach whereby disease names, at least initially, are applied to all corals exhibiting the same signs, avoids an unwieldy system for field surveys and researcher training purposes. Recording the names of coral species plus detailed descriptions for all records of disease retains species-level information in the event that diseases are later found to be specific to coral species. Refinement of disease names as histological, microbiological and molecular studies link pathogens to macroscopic field signs provides the foundation for an iterative approach to the development of definitive disease names. Figure 1 shows results for surveys of disease prevalence in the central and northern GBR using the above naming protocol. It is based on recognition of the following disease types in surveys on the GBR. All disease types have been detected in surveys on other western Indo-Pacific reefs.

Black Band Disease **(BBD):** BBD is widespread throughout the GBR, occurring on more than 70% of reefs surveyed (n=19) and in all 3 sectors, although its prevalence is typically low (~0.1% of scleractinian corals) (Page and Willis 2006). It has been recorded on at least 32 coral species in 10 families, with branching pocilloporid and acroporid corals being important hosts on the GBR (Willis et al. 2004). On reefs in Palau, a reddish band of cyanobacteria on *Pachyseris speciosa* and *Porites* sp. has been identified as having the same ribotype as cyanobacteria producing macroscopic signs of BBD on *Montipora* sp. (Sussman et al. 2006). Further evidence is required before the potential status of red band as a separate syndrome can be assessed (Sussman et al. 2006).

Skeletal Eroding Band **(SEB):** SEB, caused by the protozoan, *Halofolliculina corallasia,* erodes the tissue and skeleton of corals as it produces a black lorica or test (Antonius 1999). Clusters of ciliates along the tissue-skeleton interface produce a black band similar in appearance to black band disease, but, unlike the uniformly white skeleton exposed as BBD advances, the skeleton behind the advancing SEB is speckled with the remains of empty black loricae (Antonius and Lipscomb 2001). Progression of SEB can be relatively slow, approximately 1 mm per week, further distinguishing it from BBD, but it may also advance at rates up to 1 mm per day, comparable to BBD (Antonius

and Lipscomb 2001). SEB affects at least 31 species of corals in 6 families on the GBR. Recent records of a different species of *Halofolliculina* causing similar signs on 25 corals in 6 families from the Caribbean suggest that halofolliculinid infections affect corals on reefs globally (Croquer et al. 2006).

White Syndrome **(WS):** WS is a collective term for conditions producing white signs on corals from the GBR and Indo-Pacific reefs (Willis et al. 2004). Given the difficulty of consistently identifying features such as the variable zone of bleached tissue that distinguishes white band II (WBII) from white band I (WBI) or differences in the rates of movement that distinguish the faster moving white plague II (WPII) from white plague I (WPI; reviewed in Richardson 1998), we have elected to use the term white syndrome to describe conditions resulting in progressive loss of tissues to expose skeleton in white bands behind a moving front of tissue loss (Willis et al. 2004). A band of white bleached tissue may be present at the tissue-skeleton interface. The role of potentially secondary pathogens, like ciliates (see brown band description below), in possibly obscuring bleached zones as rates of tissue loss escalate, requires further investigation. ^{14}C studies near lesion boundaries on tabular *Acropora's* in the southern GBR suggest that photoassimilates are preferentially translocated away from lesions in an apparent shut-down reaction, potentially as a result of abiotic factors or pathogens triggering an apoptotic reaction in the host (Roff et al. 2006).

In addition to WBI/II and WPI/II, white syndrome could potentially encompass white pox (Patterson et al. 2002) and even shut down reaction (Antonius 1977). However, WS is distinguished from feeding scars by the narrow width of the zone of recently exposed, white skeleton and the relatively regular appearance of the tissue front. These features are in contrast to the wide zone of white skeleton commonly exposed following *Acanthaster planci* predation and the scalloped or wavy tissue front produced by *Drupella* spp. Determining the relationship(s) between the Caribbean white diseases and WS and applying the appropriate name(s) will not be possible until potential pathogens are isolated and compared to those producing white symptoms in Caribbean corals (Sussman et al. in prep.).

WS has been recorded for 17 species of corals in 4 families on the GBR, with species of *Acropora* being important hosts (Willis et al. 2004). Dramatic increases in abundance of WS on the GBR, by up to 20-fold on some outer-shelf reefs in the northern and southern sectors in 2002/03, suggest that the prevalence of WS may be increased by elevated temperatures when host densities are high (Selig et al. in press). Reports of a more than 50% increase in the prevalence of WS (from 8.55% to 13.58%) at a sub-tropical reef south of the GBR in 3 months following the summer of 2003 (Dalton and Smith 2006) provide further evidence of a correlation between aggregated distributions and high densities of hosts and WS prevalence.

Brown Band Disease **(BrBD):** BrBD is a new syndrome and has been recorded on corals in all 3 sectors of the GBR (Willis et al. 2004, unpubl. data). The distinctive macroscopic field symptom is a brown zone of variable width, flanked by healthy tissue at the advancing front and exposed white skeleton at the trailing edge as the band progresses over the surface of the colony. There is often a white zone between the healthy tissue and brown band, which may comprise bleached tissue and/or denuded skeleton. Dense

populations of ciliates, packed with zooxanthellae from engulfed coral tissue, cause the brown coloration of the band. As densities of ciliates decrease, the zone becomes lighter and may appear white at very low ciliate densities. In these latter cases, the condition would be assigned to the WS category based solely on field observations. The possibility that BrBD ciliates represent a secondary infection following tissue necrosis induced by a primary pathogen requires further investigation. At high densities, however, the ciliates become the primary agent of tissue loss. BrBD has been reported on 16 species from 3 families on the GBR, with acroporid corals being important hosts (Willis et al. 2004).

Note that an earlier report of a brown band on a colony of *Acropora formosa* (Dinsdale 1994) referred to a different, but unknown syndrome, and has subsequently been mistakenly quoted as affecting 20 coral species on the GBR (Santavy and Peters 1997; Borneman 2001). While it is possible that the unknown syndrome was caused by a cyanobacterium similar to the one causing red-band disease in the Caribbean as suggested by Santavy and Peters (1997), in the absence of the specimen it is not useful to speculate further about this isolated observation; it is not to be considered a record of BrB as described here.

Coral Tumors: Hyperplasia's, manifesting as raised masses projecting about 4.5 cm above the surface of the colony, were reported to affect 18-24% of populations of *Platygyra pini* and *P. sinensis* on Magnetic Island, central GBR (Loya et al. 1984). Tumors were associated with increased growth rates of polyps and a general proliferation of all cell types, some atrophied and others normal, but in all cases macroscopic polyp structures were discernible and tissues remained pigmented (Loya et al. 1984). Bleached neoplasms, manifesting as white, globular masses of skeleton raised above the surface of the colony with few discernible polyp structures, are most common on acroporid corals on the GBR (Willis et al. 2004). Bleached neoplasms, mainly on corals in the family Acroporidae, have been reported from throughout the Indo-Pacific, i.e. from Guam and Enewetak (Cheney 1975), French Polynesia (Le Champion-Alsumard et al. 1995), Japan (Yamashiro et al. 2001) and the Gulf of Oman (Coles and Seapy 1998).

Porites ulcerative white spots **(PUWS)*:*** PUWS is characterized by discrete bleached round foci, 3-5mm in diameter, that may either regress or progress to full tissue ulcerations that coalesce, occasionally resulting in colony mortality (Raymundo et al. 2003). Definitive cases have not yet been identified from the GBR.

Atramentous Necrosis **(AtN):** AtN is characterized by spreading lesions of blackened, dead tissues and has primarily been recorded on a *Montipora* species in the central GBR (Jones et al. 2004). Further observations indicate that there are 4 stages in the progression of the disease (Anthony et al. in prep). The first stage involves multi-focal areas of bleached tissue, 1-2 cm in diameter, often in depressions on the colony surface. In phase 2, the bleached tissue degenerates, leaving an area of bare, white skeleton. In phase 3, areas of bare skeleton are covered with a white, anoxic bacterial film. In phase 4, a black deposit accumulates under the white film, giving the lesion a grayish appearance (the stage described by Jones et al. 2004).

Cyanobacterial Syndromes (other than BBD): These include cases of unidentified cyanobacteria that appear distinct, both in colour and morphological dimensions, from BBD-associated cyanobacterial filaments. Cyanobacteria aggregate along fronts at the interface between exposed skeleton and tissue and are associated with tissue mortality (Willis et al. 20004).

Gorgonian Black Necrosing Syndrome: Little is known about gorgonian diseases on the GBR. The only study of GBR gorgonians to date reports that 10% of populations of *Isis hippuris* on Davies Reef were infected with a fungal disease that manifested as black necrotic areas and led to loss of both tissues and skeleton (Morrison-Gardiner 2001). Although two species of *Penicillium* isolated from infected gorgonians were able to infect healthy colonies of *I. hippuris* and *Pinnigorgia* sp., and could be re-isolated, they did not produce the typical symptoms of the disease (Morrison-Gardiner 2001). Black necrotic patches have also been observed on gorgonians at Lizard Island and have been referred to as black necrosing syndrome (Willis et al. 2004).

Indicators of Compromised Health

Pigmentation responses: Species of *Porites,* in particular, appear to respond to a variety of competitive, invasive and parasitic challenges by producing pink or purple pigmentation in coenosarc and polyps adjacent to sites of competitive interactions and lesions. Hence pink lines, rings, patches or spots are often visible in coral tissue bordering the margins of competing or boring organisms (Willis et al. 2004). The pigmentation appears to be part of a generalized response mounted by the coral to contain invading or competing organisms such as cyanobacteria (Ravindran and Raghukumar 2002), polychaetes, molluscs, and the intermediate metacercariae stage of the digenetic trematode, *Podocotyloides stenometra* (Aeby 1998). Although most commonly observed on *Porites,* pigmentation responses have been observed on most genera.

Algal overgrowth*:* Algal filaments growing directly on live coral tissue may result in small areas of bleaching and subsequent coral mortality or sediment may accumulate under the algae leading to small areas of bleaching and subsequent tissue loss (Willis et al. 2004). Such cases are differentiated from coral-algal competitive interactions where only occasional contact is made between the coral and algae. On reefs in the central GBR, examples that appear to cross the boundary between competitive interactions and disease include overgrowth of coral by (1) the filamentous algae, *Coralliophila hurysmansii* causing tissue swelling, and (2) by *Anotrichium tenue*, which traps mucus, sediments and possibly microbes damaging the underlying tissues (McCook et al. 2001). It is unclear at this stage whether some other stress or pathogen has previously weakened the corals' resistance allowing algae to invade their tissues; hence such cases are categorized as an indication of compromised health (Willis et al. 2004).

***Unusual bleaching patterns**:* Distinct and unusual patches, spots, stripes etc. of bleached tissue differ from typical patterns of whole colony bleaching or paling seen during thermal anomalies. The causes of these unusual bleaching patterns are unknown and it is unclear whether they are caused by specific stressors or pathogens, or if they represent more generalized stress responses. At present they are recorded as another indicator of compromised health.

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V. PATHOLOGY AND EPIDEMIOLOGY

DISEASE AND THE DIAGNOSTIC PROCESS

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It often is difficult to define health and disease, or to determine where one ends and the other begins. Definitions for these in humans are usually inappropriate for wild species, and death is a rather extreme endpoint to define disease in any species. One way of defining disease is on the basis of impairment of function. This allows consideration of effects on growth, behavior, reproduction, defense and survival as disease. Mild dysfunction, for which the organism can compensate, may fall within relative health, while more severe dysfunction represents disease. Dysfunction can be related to ecological fitness, and the concept can be extended to population effects.

Disease, regardless of cause, begins with injury to individual cells. However, it is the reaction by the organism to cellular injury that results in the dysfunction that we recognize as disease. **Disease is not synonymous with infection or exposure to an agent**; an organism may be exposed or infected but if there is no reaction there will be no disease.

Each organism has only a limited numbers of ways in which it can react to injury. Certain agents elicit a distinct pattern of reaction, but more than one agent may produce the same reaction. Diagnosis is the process of defining those features that distinguish a particular process from all others, i.e. distinguishing disease caused by agent A from that caused by agent B. **Recognition of the pattern of reaction to injury is the basic feature for making a diagnosis in the case of disease**.

The first step in the diagnostic process is to form a working description or **case definition** of the condition. This definition represents the state of knowledge at the time; it is usually crude at the outset, and it becomes progressively refined as information is collected. It is not necessary to know the cause to make a case definition. A critical part of the case definition is characterization of the reaction of the organism to injury. This is the purview of the diagnostic pathologist. While there may be very few "coral pathologists", there is no reason why description and analysis of the reaction to injury should be fundamentally different in coral than in other organisms. **The case definition is the touchstone** ("*a criterion for the quality of a thing*") against which all subsequent portions of an investigation must be tested. For instance, if a condition in another location has a different reaction pattern, or a putative cause results in a different pattern of reaction, one should suspect that the conditions are not the same entity.

A small proportion of diseases are caused by a single agent that is both necessary and sufficient in itself to cause a clearly defined disease. A set of rules (Koch's Postulates) often can be used to establish a cause–effect relationship in this type of disease. However, the great majority of diseases, in all species, are of more complex causation. Agents may cause disease under certain conditions but not under others, multiple agents may be required to produce disease, or several agents may independently cause similar disease. In many diseases there is a complex web of causation that may involve many inter-related factors. Disease must be considered in the context in which it occurs. Koch's Postulates are generally inadequate for establishing cause-effect relationships, and other criteria that include epidemiological information are more appropriate.

Investigation of disease involves answering five basic questions: Who? Where? When? What? and Why? It is impossible to predict in advance which disciplines or diagnostic techniques will be required to solve a problem and no one discipline is omnipotent. However, field and laboratory studies must be related back to the case definition (i.e., Are we looking at the same disease?) and the results should be used to refine the definition of the touchstone.

EVOLUTIONARY ECOLOGY AND DISEASE EMERGENCE: THE BIG PICTURE

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ABSTRACT: Understanding factors responsible for reemergence of diseases believed to have been controlled and outbreaks of previously unknown infectious diseases is one of the most difficult scientific problems facing society today. Significant knowledge gaps exist for even the most studied emerging infectious diseases. Coupled with failures in the response to the resurgence of infectious diseases, this lack of information is embedded in a simplistic view of pathogens and disconnected from a social and ecological context, and assumes a linear response of pathogens to environmental change. In fact, the natural reservoirs and transmission rates of most emerging infectious diseases primarily are affected by environmental factors, such as seasonality or meteorological events, typically producing nonlinear responses that are inherently unpredictable. A more realistic view of emerging infectious diseases requires a holistic perspective that incorporates social as well as physical, chemical, and biological dimensions of our planet's systems. The notion of biocomplexity captures this depth and richness, and most importantly, the interactions of human and natural systems. This article provides a brief review and a synthesis of interdisciplinary approaches and insights employing the biocomplexity paradigm and offers a social–ecological approach for addressing and garnering an improved understanding of emerging infectious diseases. Drawing on findings from studies of cholera and other examples of emerging waterborne, zoonotic, and vectorborne diseases, a ''blueprint'' for the proposed interdisciplinary research framework is offered which integrates biological processes from the molecular level to that of communities and regional systems, incorporating public health infrastructure and climate aspects.

EMERGING INFECTIOUS DISEASES

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1.0 Background

Newly emerging and re-emerging infectious diseases have been of increasing concern over the past 20 years (1-3). Global transportation of people, animals, and food supplies; increased interactions between wildlife, domestic animals and people; high-concentration populations; and the increase in the number people with compromised immune systems (AIDS/HIV and growing elderly populations) have all been identified as risk factors for emerging infectious diseases. This paper summarizes what has been learned from emerging infectious diseases in human and non-human animal populations; and provides preliminary recommendations for responding to emerging infectious diseases in Pacific coral reefs.

2.0 Unique profiles of emerging diseases

Emerging infectious diseases, by their nature, affect populations differently than non-emerging diseases. These differences may be considered when developing a strategy to prevent or respond to a population health event involving an emerging disease. The emergence of infectious diseases is most often due to 1) new pathogens, 2) changed indigenous pathogens, or 3) a compromised animal population. When a newly emerging infectious disease is introduced to a population, a large percentage of animals (if not all) may be immunologically naïve to the pathogen. As such, this pathogen is likely to cause rapid morbidity and/or mortality throughout the entire population. The rapid spread of the coronavirus causing Severe Acute Respiratory Syndrome (SARS) in China and other countries in 2003 demonstrates what can happen when a new disease affects an immunologically naïve human population (4). Newly emerging diseases may also affect multiple animal species concurrently; for example, during the Ebola virus outbreak in the Congo Republic in 2003, mortalities were reported in both gorilla and human populations (5); as another example, West Nile virus continues to cause concurrent morbidity and mortality in bird and human populations (6). Compared to newly emerging infectious diseases, re-emerging infectious diseases often occur due 1) a change in underlying factors within a population, or 2) acquired resistance of a pathogen to treatment. Both tuberculosis and toxoplasmosis became emerging infectious diseases due to the increased number of people with compromised immune systems from the AIDS/HIV pandemic. Methicillin-resistant *Staphylococcus aureus* (MRSA) emerged as a pathogen of concern due to its acquired resistance to multiple antibiotics. As such, re-emergence of a previously ‘quiet’ pathogen may indicate a more susceptible animal population or a new ability for the pathogen to resist natural or medical treatments.

For the reasons outlined above, emerging infectious pathogens are more likely to cause more severe disease in larger percentages of animal populations compared to established infectious diseases. Additionally, re-emerging diseases may indicate an immunocompromised population or an old pathogen with 'new tricks' to overcome natural or medical treatments.

Challenges of emerging diseases

There are four primary challenges to addressing the emerging disease issue. The first is the need to assess whether a disease is truly emerging or if case numbers are increasing simply due to improved detection and reporting capabilities. The second challenge is detection and characterization of a novel pathogen; if a pathogen is truly novel, identifying the appropriate diagnostic tools for detection can be difficult. Third, initial treatments for emerging diseases may be limited to supportive care and quarantine until the pathogen can be found; if the pathogen is a virus, treatment options will be limited, and a vaccine will not be readily available to prevent a pandemic. Finally, even if a disease is determined to be emerging, the etiological agent is found, and a treatment is identified, there remains a need to assess whether or not the emerging disease is a truly primary disease or if it is secondary to an underlying factor in the animal population. Responses to these challenges are outlined below.

3.0 Addressing the challenges of emerging diseases

Public and animal health agencies throughout the world have implemented targeted mitigation strategies to address the challenges of emerging diseases. Below are five activities that are commonly implemented to detect, track, prevent, and respond to emerging diseases. Any combination of these actions, if not already implemented, may be considered to help protect global and regional coral reef populations.

3.1 Determine the baseline for population health. In order to determine if a disease is truly emerging, there is a need to determine the baseline for a population's health. This baseline provides a statistical means of assessing significant differences in populations before, during, and after a potentially emerging disease. Determination of a population health baseline requires long-term collection of standardized health metrics in a population.

3.2 Establish a standardized disease surveillance system that includes both pathogen-specific and syndromic surveillance. The surveillance system should routinely collect and report standardized data related to known diseases of concern. Additionally, it should enable detection and reporting of an emerging disease event in which the cause (etiology) is unknown; many countries use syndromic surveillance (e.g., incidence of respiratory illness or skin lesions) to detect and track emerging diseases. International, centralized surveillance systems are better at detecting emerging diseases compared to multiple, fragmented surveillance systems.

3.3 Implement a robust disease diagnostics program. The more quickly the definitive diagnosis of an emerging disease can be acquired, the better chance

one has to target appropriate mitigation strategies to prevent a catastrophic event. Use of molecular diagnostics (e.g., polymerase chain reaction) has greatly enhanced the ability to rapidly identify and characterize infectious pathogens from clinical samples.

3.4 Conduct formal epidemiological risk assessments. Using the standardized health and disease data collected through a central surveillance system, formal epidemiological risk assessments can be conducted to 1) determine risk factors for an emerging disease, 2) identify appropriate mitigation strategies for specific disease events, and 3) assess the effectiveness of mitigation strategies on population health. These assessments can help determine if a disease is emerging due to a novel pathogen or a compromised animal population.

3.5 Develop a general emergency response plan. In the event that an emerging disease leads to an epizootic of high mortality, an emergency response plan may help to minimize the global impact of the event. Response plans may be more useful if emerging disease events are categorized (e.g., mild, moderate, severe) with corresponding response plans.

4.0 Potential action items for discussion

- Determine standardized health metrics that can be routinely collected and reported for coral populations
- Consider where a centralized coral health & disease surveillance system may reside
- Implement a robust molecular diagnostic program based upon polymerase chain reaction (PCR)
- Determine study questions for future epidemiologic risk assessments
- Create emerging disease event categories (e.g., Code Red, Yellow, Green) with corresponding response plans

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WILDLIFE DISEASE INVESTIGATIONS 101

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ABSTRACT

Worldwide we are seeing an increased interest in the ecology of disease in wild plants and animals and a concern over whether anthropogenic environmental changes are significantly influencing disease to the detriment of important species. This seems to be occurring across ecosystem boundaries (ie. in cloud forests, woodlands, savannas, deserts, lakes, rivers, and oceans) and with a wide variety of prominent examples (ie. distemper and TB in African lions, chytrid fungus in many species of amphibians, CWD in deer and elk, mycoplasmosis in tortoise, several viruses and whirling disease in salmonids, toxoplasmosis in sea otters, morbiliviruses in seals and dolphins, rickettsial wasting disease in abalone). Although none of these are the subject of this workshop on Coral Disease and Health; the problems, field methods, concepts and lessons learned from investigations of other wildlife diseases were deemed to be useful as an introduction. This paper will provide descriptions of actual wildlife disease investigations in several wildlife species that illustrate the logistical and physical challenges of trying to determine what causes disease process, how basic field information can be refined and approaches refined and rudimentary methods for management developed. Much of the information is drawn from the author's experience, various sources in the wildlife disease and health literature, and Wobeser, 1994.

INTRODUCTION: Investigations of wildlife diseases are not really new, they have been conducted for at least a century in North America and longer in Europe and Africa. However, the frequency of investigations, their complexity and importance attached to this area of research and service has increased greatly, particularly in the last decade. Wildlife disease investigations are conducted by State, Federal and tribal governments as part of their stewardship responsibilities for wildlife, by universities and institutes as part of academic or teaching responsibilities, by NGO's and conservation groups attempting to foster species recovery or health, and by cooperatives which are usually hybrids of the proceeding 3 institutional types. Examples of the government agencies might include USFWS and USGS under Department of Interior, NOAA-NMFS and NOS under Commerce, USDA-WS and USDA-APHIS under Agriculture; the Fish and Wildlife Agencies of the 50 States and the Canadian provinces; and some of the larger native American tribes such the Navaho and Yakima. A number of universities in North America have been involved in wildlife health and disease research and many of the first host/agent case descriptions, recognitions of environmental influence on disease and recognition parasite life cycles come out of academic research. In the last few decades nongovernmental organizations like the Wistar, Scripps and Hubbs Institutes, Wildlife Conservation Society, the Morris Animal Foundation and others have funded and supported wildlife disease research. Several large cooperative efforts, notably the

Southeastern Wildlife Disease Cooperative and the Canadian Wildlife Health Cooperative have successfully combined university, government and other resources, mandates and personnel to provide wildlife health research and services.

Scenario 1: In February of 1995 a California wildlife veterinarian got a call from the manager of Grizzly Island wildlife area in the Suisun Marsh just north of San Francisco concerning tule elk (*Cervus elaphus nannodes*) dying. These elk, a subspecies that had adapted to the swamps and fens of the central valley and coastal areas of California, had been hunted to near extinction in the 19th century and only remnant populations survived into the mid 20th century. In the 1970's and 1980's groups of tule elk were captured and translocated to a number of State, Federal and privately owned properties. The elk had done very well at this refuge and had surpassed 120 animals on 500 hectares. Several elk had been found down and others were dead. The downed animals appeared to be seizuring, paddling and those that had recovered appeared drunk and disoriented. In all it was estimated that at least 10-12 of the elk were affected and assistance was requested.

Analysis: This is a State managed species, on a State refuge, with the contacted person being a State Game and Fish Veterinarian responsible for health and disease in living and dead free-ranging species. No jurisdiction crossed, no permits or plans needed, no significant permission to request either for work on dead animals, handling or "take" of sick. It is an acute unexpected, previously undescribed event or phenomenon, a significant proportion of the population is affected, and immediate response is probably appropriate, although research, planning and sampling gear preparation are limited.

Response: Load up the necropsy kit, a rifle, sampling gear, immobilization equipment, leave the wife a note, (that's something people did before cell phones) and hit the road.

Findings: Eleven dead animals were located, all of them very fresh having died within the last day or two. All were yearlings, 6 male, 5 female. Several had been paddling around for several hours working up the ground around them, no external lesions of signs other than minor contusions and abrasions noted. Several live animals including at least one adult male were seen showing signs of incoordination, stiff high gait, opisthotonus (neck arched and head held high). Two live elk were down and could not rise. Treatment with steroids, antibiotics (penicillin) and atropine were tried with no results.

Postmortem examinations were done on 5 of the dead elk.

Dead elk were in fair to good body condition, no significant lesions were noted in the eyes, ears, nose, or mouth. Brains were not examined in the field. The lungs, heart, liver, kidneys, spleen, adrenals, reproductive organs, intestinal tract also appeared normal. The rumen contained bright green, somewhat frothy contents and some pale carrot like tubers. The smell of the rumen contents was unusual.

There had been many days of fog and rain and most grasses and forbs were buried under a think thatch of dead grasses. The primary green plant available was poison hemlock (*Conium maculatum*) and areas of heavy grazing and pawing to unearth roots were evident. Animals with CNS signs were actually seen returning to hemlock patches to graze. The roots of the hemlock looked very similar to those seen in the rumen of dead

animals, the leaves were the same bright green and the smell of roots and leaves were similar to that unusual small coming from the rumen. Diagnosis of acute hemlock poisoning was confirmed by isolation of conine toxin from rumen contents.

The following **management actions were recommended**: provide attractive grain and alfalfa hay feed it areas away from hemlock. Spray hemlock patches with carrot oil and disk it under ASAP, set up zone guns to scare elk away from hemlock areas where they were eating it, provide more diverse feed, reduce the numbers of elk on the refuge.

Scenario 2: In September of 2002 SCWDS gets a call about white-tailed deer (*Odocoileus virginianus*) that are dying around campgrounds in Great Smokey Mountains National Park. The person calling was camping there and describes depressed animals with foam coming from their mouths and bleeding from the rectums. They had described the deer to a NPS Ranger who didn't seem too excited about it and the person, a citizen of Athens, GA., was aware that SCWDS does a lot of wildlife health work all around the southeastern USA.

Analysis: SCWDS works with NPS and a call to offer diagnostic assistance and to discuss the need to deal with park visitor concerns is warranted. The description sounds a lot like EHD or bluetongue, an endemic orbiviral disease of deer, which is fairly common in the summer and fall but could be the start of a die off. Call the chief ranger of Great Smokey Mtns., it's their call. They may want to collect and ship the animals or take samples as they have been part of previous sampling programs.

Response: NPS personnel took blood samples from fresh dead deer and sent them to SCWDS. SCWDS personnel visited the park a week later and collected 4 deer, and examined 2 of which had died recently.

Findings: EHD 1 virus was isolated from tissues of one deer examined by SCWDS and blood of 1 animal sampled by NPS. Lesions in the 2 dead deer were compatible with hemorrhagic disease, these were confirmed by histopathology. Serology showed high antibody titers to EHD 1 and BT 10 viruses.

Comment: Although the initial observations (deer dying near campgrounds, frothing at the mouth and bleeding) were somewhat alarming, the syndrome is relatively common in the southeastern USA, particularly in the late summer and fall. The disease is a relatively natural process little influenced by human activities, but the epidemiology is followed closely by SCWDS in hopes of identifying predictors. Few if any actions are recommended for management despite the fact that dieoffs can involve many animals.

The above scenarios illustrate some reasons why wildlife disease investigations are done. Basic reasons for studying any disease (from Wobeser 1994) are to:

1) determine its nature and cause,
2) to determine the effects on individuals, populations and ecosystems (to assess its significance),
3) to identify methods to prevent, control or reduce the disease or its effects.

In addition, **with regard to wild species** the reasons may also include:
1) curiosity about the disease as a biological phenomenon,
2) concern over its impact on wild populations and ecosystem integrity,
3) public concern over highly visible die offs or unsightly conditions,
4) concern that disease in a wild species may be transmissible to humans or domestic species, and
5) concern that diseases in wild species are indicators of undesirable changes in the environment.

Scenario 3: In April of 1989 California bighorn sheep (*Ovis canadensis californiana*) in the Warner Mountains of the northeastern corner of California are reported to be dying, only few living animals and several bodies were spotted in a recent aerial survey. Mule deer (*Odocoileus hemionus*) in the area are also reported to be dying in large numbers in the valley bottom adjacent to a north-south highway and valley ranch lands.

Background: This bighorn population had started from a nucleus 12 animals released into a vast mountain range 10 years previously and was thought to now number approximately 70 individuals. Only about 600 of this subspecies live in California and they are State listed as "threatened". Bighorn sheep have a history of dying in large numbers due to fibrinopurulent (*Pasteurella*) broncho- pneumonia. Previous dieoffs in other locations have resulted in local extinctions. There are serious conservation and political implications (previous dieoffs have been associated with contact between domestic sheep and bighorn). The area is vast and steep, it is a late winter and only the valley areas thaw during sunny days.

Analysis: Investigation needs to proceed immediately as decomposition and scavenging will obliterate destroy all evidence quickly. The entire heard of bighorn could be in jeopardy. The deer and bighorn dieoffs may be linked or entirely separate events.

Response: Load up the necropsy kit, a rifle, sampling gear, arrange for a helicopter that can do both survey and capture (net-gunning) for sampling, arrange for the ex-wife to care for your dog (divorce is one hazard of lots of field work). Discuss the situation in detail with the local biologist, make arrangements to meet with ranchers in the area who may be cooperative and who control ground access.

Findings: Many deer carcasses in various states of preservation (those that die in the shadows have probably been frozen for weeks). Quick field postmortem examinations are done on 5. All the deer (about 10 observed grossly, 5 necropsies) are extremely emaciated. None show significant parasite loads, evidence of infectious disease processes including but not limited to respiratory disease, gastrointestinal disease, systemic lymphoid or hemorrhagic disease. Stomach contents are minimal but often straw or old alfalfa from hay bales. No significant natural browse or forage is available due to prolonged cold winter. General impression is one of starvation/malnutrition.

While doing post mortem examinations of deer at one of the cooperating ranches, the rancher mentioned that he had lost a number of sheep that he grazed in the adjacent range and had only recently found them as they were driven down by harsh weather. He had killed them and allowed access to the reasonably fresh carcass of one. Histological

examination by a state diagnostic lab pathologist revealed chronic bronchitis and low grade bacterial pneumonia. Cultures grew no bacteria of note.

Relatively few bighorn sheep carcasses are found and these have been dead for weeks and are badly scavenged. There are some sites of subcutaneous fat and bone marrow fat is evident when long bones are broken. Mats of yellow fibrin and black discoloration of the pleural lining suggest and portions of one lung suggest fibrinopurulent bacterial pneumonia. A small herd (3) feral goats are spotted in the area and one live bighorn ram is seen running and behaving in ways that suggest it is reasonably healthy. It was captured and sampled (blood, feces, nasal and tonsular swabs), tagged and collared and released. These samples reveal no lungworm (*Protostrongylus spp*.), no evidence of systemic infection (from CBC and chem.), no significant pathogenic bacteria isolated. The three feral goats were shot and postmortem examination was unremarkable. A *Pasteurella multocida* was isolated from tonsil of two animals.

Summary: The case described is not unusual in wildlife mortality events. Time, space, terrain, weather and other factors make it very hard to establish a cause of death or to sort out potential causes from coincidental events. The bighorn sheep dieoff pattern is typical of that seen in many western states when bighorn have contact with domestic sheep or goats that may carry pathogenic *Pasteurella* or *Manheimia* bacteria in their upper respiratory tracts. A few shreds of evidence suggest that either the feral goats or the feral sheep may have been involved, as both were in the same general area as the bighorn and both had some evidence of some potentially virulent bacterial respiratory flora, but certainly no cause and effect conclusions can be drawn. Under some circumstances bighorn may develop bacterial pneumonia without contact with domestic sheep or goats. A very few sightings of bighorn sheep in the range persisted for a few years, but all lambs born died before weaned and the herd slipped into extinction within 3 years of the outbreak. The deer dieoff appeared to have nothing to do with the bighorn dieoff but indicated harsh weather conditions that may have impacted other species as well. Without solid evidence on which to base land use policy decisions, enforcement actions, and wildlife management programs it is impossible to manage disease related conflicts between land and resource users.

Understanding causation is extremely important: In its simplest form disease may be seen a single agent infecting one species of hosts, relatively uninfluenced by the environment, with clear and decisive outcomes (recovery or death). Clear biological, pathological and pathogen isolation information clarifying the role of host(s), agent and environment make understanding causation much easier. Unfortunately, those kind of simple, straight forward situations are not common.

(From Wobeser 1994) With the discovery of microbial pathogens at the turn of the 20th century, human and veterinary medicine was concerned with identification of specific agents responsible for acute infectious diseases. A set of rules (Koch's postulates) were developed for establishing cause and effect relationships that were generally widely accepted. These were:

1) the agent must be shown to be present in every case of disease through isolation in pure culture,
2) the agent must not be found in cases of other diseases,
3) the agent must be capable of experimentally reproducing the disease, and
4) the agent must be recovered from the experimental host.

But, Robert Koch is dead and our simple concepts of disease have become considerably more complex and encompassing of a much wider array of processes. Perhaps the broadest view yet is that one can see whole ecosystems as "healthy" or "unhealthy" and perhaps identify the reasons why.

A broader set of criteria for establishing causation, reflecting the multifactorial nature of most disease (adapted from Kelly, Thompson and Evans (1986)) is:

1) the hypothesized cause should be distributed in the population or in nature in the same manner as the disease,
2) the occurrence of the disease should be significantly greater in those exposed to the hypothesized cause than in those not exposed,
3) exposure to the hypothesized cause should be more frequent among those with disease than those without , if risk factors are constant,
4) disease should temporally follow exposure to the cause
5) higher doses or longer exposure to the cause should increase disease occurrence
6) for many diseases a spectrum of host responses along a biological gradient from mild to severe should follow exposure
7) other explanations and associations should be eliminated
8) the association between cause and disease should be evident in various populations studied by different methods
9) elimination or modification of exposure to the cause should decrease occurrence of the disease
10) prevention of exposure or modification of the host response (as by vaccination) should decrease or eliminate the disease
11) disease should occur more frequently in experimentally exposed animals than in controls, and
12) all the relationships and findings should make biological sense.

Scenario 4: When diseases occur regularly and are a serious threat to the survival of a species the efforts put into diagnosis and management may be larger and more prolonged than outbreak investigations. Biologists have been studying the diseases and causes of death in southern sea otters for over 35 years and for the last 14 years professional veterinary postmortem examinations have been done on all essentially all fresh dead (from 40-100 animals per year). This effort is seen as vital to sea otter recovery and it has allowed the clear description of a number of previously unrecognized or underappreciated disease processes. With an extensive dataset for comparison, mortality events that exceed average can be recognized and quantified and compared to previous years and events. The goal of these efforts is to identify relationships and associations that might lead to improvements in management that benefit the effected populations.

In the spring of 2003 large numbers of dead southern sea otters were picked up along the California coast. Record or near record carcass pickups occurred for several months and an unusual mortality event was declared by USFWS and NOAA. By the end of 2003 the number of sea otters recovered exceeded any previous year, even when numbers were indexed to population growth. No unusual spatial or temporal clusters of mortality were evident. The causes of death found by pathologists were not "unusual" in the sense that the agents and causes were similar in type and proportion to previous years, just greater, in magnitude with the exception that some animals had evidence of domoic acid (amnesic shellfish poisoning) intoxication. In the end it was felt that DA was an additive mortality factor that was behind the record losses.

In the spring of 2004 another dieoff of even greater magnitude occurred. This time the mortalities were clustered in both space and time, occurring around Morro Bay, CA in mid-April to mid-May. Eventually nearly 50 otters, some of which were initially recovered while still alive were collected. All live animals showed severe central nervous system signs including tremors, coma and seizures, and all but one died within a day of stranding. The majority of dead and dying animals had severe generalized lympadenopathy (swollen lymph nodes), multiorgan congestion, pericardial effusion, cardiac mottling and spleenomegaly. Serology suggested some otters had antibodies to *Toxoplasma gondii* but that many more had very high antibody titers to *Sarcocystis neurona*, both of which are protozoal parasites known to kill sea otters. H&E stained sections of effected otters showed severe meningoencephalitis (infection of the brain) classic rosette formations of schizonts typical of *S. neurona* and immunohistochemistry stains were positive. Although it had been known that *S. neurona* could and would kill sea otters previously, cases had been few in number, sporadic and with no particular spatial or temporal pattern that might implicate a source or cause.

The above should illustrate the importance of establishing a case description: (From Wobeser 1994) Identifying and defining a disease or disease process: Defining the cause or nature of a disease, or formulating a working hypothesis should be a very early step in every investigation. This is equivalent to a clinician arriving at a tentative diagnosis after examination of a patient (the "what" questions). This definition, often called a "case description" or "case definition" can be dynamic and is very likely to be modified by subsequent information and/or field work. The patterns of temporal and spatial disease occurrence (the "when" and "where" questions) are very important in understanding disease/health conditions in wild species. The disease or disease process is further defined by answering the "who" is questions by defining the population and parameters affected and the "why" question by defining the consistent cycle of the disease (pathogenesis) in the host and the causation.

SUMMARY: Although it has been customary to believe that wild animals are generally very healthy and that health is maintained by natural selection, we are beginning to understand that this is perhaps a very simplistic notion. More importantly there are few places on earth where human activities have not upset whatever natural balance existed and/or where human induced changes are not associated with disease in wild animals, wildlife populations and their ecosystems. Although wild animal populations and ecosystems have the ability to respond and to heal themselves, we must understand and in

many cases correct or mitigate the conditions that caused unbalance and/or disease if healthy populations and the be restored.

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VI. COMMUNICATION TO MAKE A DIFFERENCE

SCIENTISTS, THE PUBLIC AND THE POLITICIANS: HOW DO WE CONNECT FOR REEF'S SAKE?

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Two Examples

1 - Coastal Land Use and Policy

2 - Integrating Science & Values to Inform Ecosystem Management

First Example - Coastal Growth and Sustainability

South Carolina's population expected to increase by 24% by the year 2030 according to U.S. Census Bureau.

Rusk and many others have shown that corresponding land development occurs at much higher percentages.

Tools are needed to help policy and decision makers understand patterns of urban growth and potential impacts - economic, social/cultural, and ecological.

Modeling Land Use Change and Urban Growth

Transportation Models - TRANUS - (Johnston and Shabazian)

Deterministic Land Use Models - SACMET, UPLAN (Sui)

Cellular Automaton Models - SLEUTH (Jantz & Goetz, Clarke)

Rule-based Models (Pijanowski)

Logistic Regression Models - CUF (Landis)

Charleston, SC Region Growth Prediction Summary

Under the current modeling scenario, there are two assumptions involved. The ratio of overall urban land use change (255%) to overall population growth (41%) from 1973 to 1994 occurred at a ratio of about 6:1. Secondly, it is assumed that population for the three county area will grow to 795,879 by the year 2030 as predicted by projections of the

BCD COG compiled with information from the U.S. Census Bureau, SC Department of Commerce and the BCD COG. The predicted urban growth mainly takes the pattern of urban sprawl and by the year 2030 consumes 868 square miles within the BCD area. If the current growth trends continue and the predictions hold true, the future urban growth will sprawl considerably outward from the current urban boundaries. This has several significant economic, environmental, and social implications in policy-making and urban planning.

Regional Policy Considerations

Local - majority of land use decisions made at this level - municipalities must work closer with developers to balance growth and fiscal responsibilities - work with other municipalities to coordinate growth impacts.

County and State - policies affect how growth spreads into rural areas - have a certain amount of regulatory authority which must be carefully used to protect environment and influence developer decisions.

Federal - many programs indirectly influence development (DOT, NFIP, EPA) - can help by providing expertise to state and local entities through research/information as well as direct funding (community grants, etc.).

Planning and management - NOAA scientists utilizing growth predictions through LU-CES program. SCDNR officials incorporating growth models into coastal habitat management. SCDHEC officials considered growth models in Coastal Futures planning. TNC, SCCCL use models for open space planning.

Second Example - Integrating Science & Values to Inform Ecosystem Management

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Ecosystem Management Challenge
Management of ecosystems involves:
Factual uncertainty
Value saliency
Social controversy
High stakes
Distrust
How should decisions be made in such contexts? How can both facts and values be accommodated?

Four points to consider:

- An “Ecoplex” conceptual framework that sustainably links natural and social systems
- An “analysis and deliberation” protocol that recursively integrates science and values in ecosystem management decision-making
- A trust-based guide to stakeholder participation in ecosystem management
- Summary

Part I - Ecoplex and Sustainability

- The Ecoplex framework is a conceptual aid to understanding sustainable ecosystem mgt
- Sustainability Definition
- *Development that is environmentally, economically, and socially sustainable*
- Sustainable development requires balanced conversion of resources to improve quality of life (welfare)

Sustainable Capital Conversion

- Conversion of natural and human capital (resources) to economic and social capital (welfare), and vice versa
    - Resources *Conversion* Welfare
- Natural Capital + Human Capital Economic Capital + Social Capital
- Legend:
- Natural capital = ecosystem goods and services
- Human capital = labor, intelligence, technology
- Economic capital = wealth (currency, property, investments)
- Social capital = order, stability, fairness (social networks, security, trust, justice, laws)

Coupling of Social & Natural Systems

- Sustainability (a balanced conversion of resources to welfare) requires a carefully integrated coupling of social and natural systems
- We have developed a framework that provides a conceptualization of how this coupling can be accomplished

Part II - Analysis and Deliberation

- A&D protocol was proposed by National Research Council in its 1996 report, *Understanding Risk: Informing Decisions in a Democratic Society*
- A&D is an alternative to the 1983 NRC protocol that envisioned a top-down process of scientific impact assessment followed by a political process of impact management
- 1983 approach has been criticized for failing to recognize the importance of involving stakeholders in framing analyses to inform eco-mgt decisions

- Recursive relationship between analysis and deliberation

- Analysis is used to gather information about the social and natural systems to inform decision-making
- "Getting the science right"
- Deliberation is used to frame analysis and to make ecosystem management decisions
- "Getting the right science"

A&D in Ecosystem Management

- The 1983 protocol dictated that scientists alone define ecosystem management problems and decide what analyses are pertinent and whether fixes are necessary
- The A&D protocol places analysis in the service of deliberation and provides an opportunity for deliberants to help frame analysis
- However, the intensity of A&D should vary with context – specifically trust

Part III - Relationship of Trust to Participation

- Trust: the willingness to accept the risk of deferring to the judgments of others based on judgments of expertise and value similarity
- High trust: deference
- Low trust: vigilance
- The participation strategy that is most appropriate depends on stakeholders' trust of other policy actors
- Policy actors include experts, fellow stakeholders, and government decision-makers

Expert Trust

- Stakeholders' expert trust judgments based on:
- Perceived expertise
- Factual certainty and salience
- Subject matter and analytical competence
- Objective (unbiased) interpretation
- Perceived value similarity
- Responsiveness to stakeholder concerns (framing), caring attitude, openness, honesty, and forthrightness
- High expert trust: evidentiary participation
- Low expert trust: constitutive participation

Social Trust (of stakeholders)

- Stakeholders' social trust judgments based on:
- Perceived expertise
- Familiarity
- Perceived value similarity
- History of social interaction
- Conformance to dominant culture and traditions
- Civic mindedness
- High stakeholder trust: cooperative participation

- Low stakeholder trust: defensive participation

Government Trust

- Stakeholders' government trust judgments based on:
- Perceived expertise
- Technical competence
- Perceived value similarity
- Fiduciary responsibility
- High government trust: trustee (leader) role
- Low government trust: delegate (follower) role

Part IV - Summary

- Ecosystem management policy must recognize the relationship between natural & social systems
- We endorse sustainability as a guiding principle in this relationship
- Analysis and deliberation is the preferred mechanism by which to integrate facts and values
- The intensity of analysis and deliberation depends on the level of trust that stakeholders have of other policy actors ("one size does not fit all")

VII. TECHNOLOGIES FOR THE FUTURE OF CORAL HEALTH

POTENTIAL TECHNOLOGICAL DEVELOPMENTS FOR CORAL DISEASE MONITORING

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The Alliance for Coastal Technologies (ACT) is a NOAA-funded partnership of research institutions, state and regional resource managers, and private sector companies interested in developing and applying technologies for monitoring and studying coastal environments. The long term goal of ACT is to be a national resource for facilitating the transition of sensor technologies to routine use in monitoring and studying coastal environments. ACT was established to serve as a comprehensive information clearinghouse on technology performance, a forum for capacity building, and an unbiased, third-party testbed for evaluating coastal sensor technologies. ACT strives to provide products such that the coastal observing community is able to identify and select technologies that are appropriate for their needs and capacities, technology developers have tools for trend identification and targeted marketing, and that the latest, innovative, and most effective technologies are continuously integrated into observing capabilities.

ACT is organized to ensure geographic and sector involvement. The program is headquartered at the University of Maryland's Chesapeake Biological Laboratory, and there are currently eight ACT Partner institutions around the country with coastal technology expertise that represent a broad range of environmental conditions for testing. The ACT Stakeholder Council is comprised of resource managers and industry representatives who ensure that ACT focuses on service-oriented activities. Regional chapter Alliance Members provide advice to ACT and are kept abreast of ACT activities.

The Hawaii-Pacific Partnership is housed at the University of Hawaii, Hawaii Institute of Marine Biology. This relatively new partnership is striving to assess the needs of the region before full-scale implementation throughout the entire region. More information on current ACT activities throughout the nation can be found at www.act-us.info.

Preliminary ACT assessments revealed that coral disease monitoring is emerging as a critical issue in Hawaii and the broader Pacific. Current monitoring programs are often limited by the number of trained professionals who can do in situ assessments of coral disease status. In situ assessments require skilled personnel and are time consuming.

Technological advancements have allowed many types of coastal monitoring programs to increase precision and efficiency, and to decrease the number of people, training of those people, and overall program expense. Tools can increase the number of lesser-trained personnel who can be involved in a program, often enabling community participation, and can decrease the number of experts who have to be involved in the entire monitoring effort. This is especially true for a parameter like coral disease which requires a very high

skill level for in situ determination. Monitoring technologies tend to increase precision, allowing measurements to be taken by multiple individuals with reduced observer error.

Accuracy may be increased or decreased by switching to or adding in technology-based monitoring, so program managers should be aware of the level of accuracy needed to meet their goals, such as to establish a baseline or temporal trend, or to trigger management actions. Program managers should also consider what factors are limiting to them in developing a monitoring program: number of people, training level of people, operating budget, capital expense budget, equipment, analysis abilities, etc. Evaluating these limits can assist in the determination of what tools are best for their situation.

ACT Hawaii-Pacific is not aware of any existing technologies that have been specifically designed for coral disease monitoring. Many tools used in other coastal monitoring programs may be useful to coral disease monitoring, and many existing technologies used in diverse applications may be adapted for specific coral disease use. The best strategy is to build upon existing technologies and identify what adaptations are necessary for coral disease monitoring needs.

The first four things that coral disease experts and managers should work together to identify are 1) the best set of parameters to be measured, 2) sampling frequency, and 3) preferred method of deployment (in-situ mooring, hand-held, etc.), and 4) level of accuracy required. For the parameters that are proxies or indicators for disease, e.g. a water quality parameter, determine if an existing tool is able to sample at your required frequency, deployment type, and accuracy. A comprehensive database of existing sensors can be found at www.act-us.info.

For parameters that do not have adequate existing sensors, the next phase is to determine if any sensors exist that could be easily adapted to coral disease monitoring needs. If a tool already exists for the parameter, but the frequency, deployment type, or accuracy is not satisfactory, scientists and managers can collectively voice their needs to industry and ask for a modification. ACT was created to be a liaison between these communities and can assist in these discussions and negotiations.

If no sensors exist for a chosen parameter, an analysis of tools used in related fields may identify places to start. For example, if a manager needs to be able to spot-check for the presence of a particular bacterium, one can ask, what other fields are interested in spot-checking bacteria levels? Public health and bioterrorism experts come to mind. Perhaps tools that have been developed in those fields could be adapted by changing the bacterium of interest. Once a similar sensor is identified, the process of negotiating with industry begins.

This likelihood of success for convincing a company to modify a tool or create a new one depends on the collective finances available to purchase the tool. If only a few individuals would be able to purchase the tool, it is not likely that industry could justify the expense. This is one key reason that the global coral disease monitoring community would benefit from standardized protocols. If, however, this approach does not work, a

researcher may be able to create a tool and produce it on small scale using a federal grant for technology development. Several of these opportunities exist.

Consideration should be given not only to identifying the correct sensors, but also to data analysis protocols/tools and personnel training. ACT Hawaii-Pacific is poised to assist will all of these questions and hopes to facilitate some of the necessary discussions.

LEVERAGING POST-GENOMIC TOOLS AND SYSTEMS BIOLOGY APPROACHES TO ACCELERATE THE UNDERSTANDING OF CORAL DISEASE AND EFFECTIVELY MONITOR THE HEALTH OF TROPICAL REEF ECOSYSTEMS

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Biotechnology can no longer be considered a new scientific discipline; molecular and genomic innovations have progressed dramatically and continue to advance at unprecedented rates. No longer is there simply the promise of how these new technologies will one day help solve biological problems; already there exists many tangible examples of biotechnological success stories in fields ranging from aquaculture to transgenic plants, from fermentation sciences to animal health, and from biocatalysis to solutions for alternative energy.

Moreover, the development and refinement of molecular methods is beginning to impact our understanding of basic biological processes, including coral bleaching and the health of tropical reef ecosystems. In the context of genomics, DNA sequencing costs have plummeted more than three orders of magnitude resulting in the availability of complete genome sequences for over 300 microorganisms and representative genome sequences available for most eukaryote lineages. Furthermore, the dramatic reduction in sequencing costs has catalyzed the development of a new method which involves direct sequencing of uncultured microbial communities and organismal consortia. This environmental sequencing strategy, also known as metagenomics, utilizes genomic principles to glean information and understanding from such complex biological processes as symbioses and host:pathogen relationships. Metagenomics can also be used to evaluate and quantify the metabolic potential of a given environment and even serve to identify nucleic acid, protein and small molecule-based probes which can monitor spatial and temporal metabolic changes within a defined ecosystem.

In addition and complementary to genomics, improvements and recent progress in proteomics and metabolomics now enable one to pose scientific questions which utilize genomic and metagenomic sequence data sets as reagents to help understand and unravel the physiological and biological consequences of an ecological perturbation, such as coral bleaching. Collectively, these strategies make use of extremely sophisticated molecular tools to revisit the age old concept of *gestalt* biology, which views biological systems as complex, multi-organism, complete ecosystems which respond cooperatively to given environmental circumstances. Improvements and advancements of these genomic and systems biology tools will be reviewed and the ramifications toward understanding of coral disease and monitoring ecosystem health will be discussed.

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We would like to thank Ms. Athline Clark for her opening remarks that introduced us to the customs and traditions of the Pacific Islands which helped us understand a bit of the culture, their dependence on, yet love for coral reef ecosystems and their unique approach to stewardship of this resource.

We are grateful to Dr. Gary Ostrander for his time and welcome presentation. His candid insightful remarks called each of us in the coral disease community to action, action for change and action to work together as a cohesive research community that rallies around common goals and collaborating on common objectives. This moving presentation was transcribed and is the Forward to this document.

Background information that created a context for the workshop participants was generously provided by a series of 16 white papers and oral presentations.

Mr. Mike Gawel provided a context for the unique nature of the Pacific culture and biology in his presentation titled: *Overview of issues unique to the Pacific Biological & Social perspectives.*

Dr. Cheryl Woodley then provided an introduction for the Coral Disease and Health Consortium and shared some of the CDHC's progress over the last 4 years in her talk titled: *Introduction to the CDHC and Progress on the National Plan.*

Dr. Esti Winter provided introductory remarks that helped define the topic and scope for coral disease with her presentation titled: *Definition of Disease and Various Presentations of Disease in Coral.*

In his two white papers, Dr. Andy Bruckner provided insights from experiences with Caribbean coral diseases and a perspective of the global pervasiveness of coral disease. His presentations were titled: *Lessons learned in the Caribbean (Historical perspective)* and *Global perspective of Incidence & Prevalence of Coral Disease.*

Dr. Bette Willis updated the group on the international coral disease work of the Coral Reef Targeted Research & Capacity Building for Management Program, a partnership between the Global Environmental Facility, the World Bank, The University of Queensland, NOAA, research institutes and third-parties globally and she also provided a

report specifically addressing coral disease in the Western Pacific with her presentation titled: *Current knowledge of diseases in Western Pacific.*

Dr. Greta Aeby began focusing on the Pacific issues with her white paper and presentation: *Current knowledge of diseases in Hawaii & Northwest Hawaiian Islands.*

Dr. Thierry Work is a veterinary diagnostician that has worked on coral disease throughout the Pacific. His presentation and white papers were titled: *Current knowledge of diseases in U.S. Territories and Freely Assoc States.*

Dr. Gary Wobeser, a prominent wildlife veterinarian and professor of wildlife diseases provided the participants with a basic primer outlining fundamentals that need to be considered when studying diseases in wildlife. His presentation was entitled: *Basic Concepts in Diseases of Wildlife: The Diagnostic Method.*

Dr. Dave Jessup helped provide some practical guidelines responding to outbreaks of disease in the wild with his presentation: *Disease Outbreak Investigation: The Process - What elements are critical?*

Dr. Stephanie Venn-Watson is a veterinarian and wildlife epidemiologist. She shared with the group insights into preparing for diseases in corals with her talk: *Emerging Disease – How do we identify it?*

Dr. Bruce Wilcox is a leader in exploring human-ecosystem interactions as it relates to human health and environmental change. He provided context for coral disease and as an emerging issue with ocean health in his presentation: *Evolutionary Ecology, and Disease Emergence: The Big Picture*.

Dr. Jeffery Allen explored the inter-relatedness of good science with politicians and the public and the importance of these interactions to actually identify and make changes to save reefs in his presentation: *Scientists, the Public and the Politicians: How do we connect for reef's sake?*

Ms Melissa Bos shared with the group, advances in marine science technologies in her presentation: *Technologies for Disease Monitoring and Assessment* and asked the group for specific needs that with improved technology the field of coral disease research would be enhanced.

Mr. Eric Mathur presented a stimulating talk titled: *Technologies for Diagnostics: Leveraging Post Genomic Tools and Systems Biology to Accelerate the Understanding of Coral Disease and Effectively Monitor Tropical Reef Ecosystems,* in which he showed the power and possibilities provided with post-genomic information and new platforms for that information that could directly impact coral health and disease research.

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Appendix I. Meeting Agenda

CORAL HEALTH AND DISEASE IN THE PACIFIC: VISION FOR ACTION

AGENDA

Monday June 19, 2006
Sheraton Moana Surfrider
Ballroom I

8:00 Call Meeting to Order - Opening Ceremony & OLI
Athline Clark

8:15 Welcome Address
Athline Clark
Gary Ostrander

8:40 Overview of Pacific Issues: Biological & Social Perspectives
Mike Gawel

9:00 Introduction to the CDHC and progress on the National Plan
Cheryl Woodley

9:15 Definition of Coral Disease and Its Various Presentations
Esti Winter

9:45 Lessons learned in the Caribbean (Historical perspective)
Andy Bruckner

10:00 BREAK

10: 20 Global perspective of Incidence & Prevalence of Coral Disease
Andy Bruckner

10:45 World Bank Project: Coral Disease Working Group
Bette Willis

11:00 Current knowledge of diseases in Hawaii & Northwest Hawaiian Islands
Greta Aeby

11:20 Current knowledge of diseases in US Territories/Freely Assoc States
Thierry Work

11:40 Current knowledge of diseases in Western Pacific
Bette Willis

12 NOON LUNCH – *Parlor Room*

1:15 Basic Concepts in Diseases of Wildlife: The Diagnostic Method
Gary Wobeser

1:45 Emerging Disease – How do we identify it?
Stephanie Venn-Watson

2:15 Leveraging Post Genomic Tools and Systems Biology to Accelerate the Understanding of Coral Disease and Effectively Monitor Tropical Reef Ecosystems
Eric Mathur

2:45 Disease Outbreak Investigation: The Process - What elements are critical?
Dave Jessup

3:15 BREAK

3:45 Evolutionary Ecology, and Disease Emergence: The Big Picture
Bruce Wilcox

4:15 Scientists, the Public and the Politicians: How do we connect for reef's sake?
Jeffery Allen

4:45 Technologies for Disease Monitoring and Assessment
Melissa Bos

5:15 Adjourn for the day

6:00-8:00 Reception – Waikiki Aquarium

Many thanks to the Alliance for Coastal Technologies and Kahaled bin Sultan-Living Oceans Foundation for hosting this event

Tuesday, June 20, 2006

8:00-12:30 Breakout Groups I-IV

Group I – Coral Cellular Physiology & Pathology
Ship's Tavern

Group II – Coral Toxicology & Ecological Epidemiology
Ship's Tavern – Captain's Quarters

Group III – Pathology of Infectious Disease
Board Room

Group IV - Preventing and Responding to Coral Disease in the Pacific Region: Management Perspectives
Admirals Room

12:30-1:30 LUNCH – Ballroom I

2:00-5:00 Resume Breakout Group Deliberations

5:30 Adjourn for the Day

6:30 Reception – Terrace Moana Surfrider

7:30 Dinner – Grand Salon Moana Surfrider

Wednesday, June 21, 2006

8:00 General Session – Ballroom I
Group I and II Preliminary Report & Discussion

10:00 Break

10:20 General Session – Ballroom I
Group III and IV Preliminary Report & Discussion

11:30 Open Discussion on Needs for Technological Advances
Melissa Bos

12:00 – 1:00 **Lunch – Parlor Room**

1:30 – 5:00 Finalize Breakout Group Reports

Dinner on Your Own

Thursday, June 22, 2006

8:00 General Session – Parlor Room
Final Presentations of Breakout Groups

12 NOON Workshop Adjourned

Appendix II. Previously recommended cnidarian 'model species' and their justification from peer-reviewed literature.

Anthozoans

Plesiastrea versipora was recommended by (Ritchie et al. 1997): "*Plesiastrea versipora* (Lamarck, 1816) is a hardy scleractinian coral that can be maintained for long periods in the laboratory even without feeding. After removal of tissue, it shows considerable powers of regeneration and, following recovery, can be reused in later experiments. The rates of respiration, photosynthesis and translocation of photosynthate from algae to animal recover to normal levels and the regenerated animal tissue has host release factor activity. We have also shown that small pieces broken off the colony will survive and grow slowly to form clones of the parent colony."

Acropora spp. was recommended by Miller and Ball (2000): "The diploblastic Cnidaria form one of the most ancient metazoan phyla and thus provide a useful outgroup for comparative studies of the molecular control of development in the more complex, and more often studied, triploblasts. Among cnidarians, the reef building coral *Acropora* is a particularly appropriate choice for study. *Acropora* belongs to the Anthozoa, which several lines of evidence now indicate is the basal class within the phylum Cnidaria, and has the practical advantages that its reproduction is predictable, external and accessible and that the base content of its genome is not strongly biased. The *Acropora* system has already provided insights into ancestral linkages of homeobox genes and the evolution of the Pax genes, and has the potential to provide further new perspectives on the age, role in development, and evolution of these and other gene families."

Frank et al. (2001) also recommended *Acropora* as a model scleractinian species: "Scleractinians are of great value in various ecological studies, in particular those related to bleaching, global warmth, CO_2 household, etc. In addition, they may serve as a model system for certain questions in evolutionary developmental biology, given their basal position within the Cnidaria. Scleractinians are also good model organisms to study biomineralization. However, the cultivation of reef corals outside their natural habitats (which is inaccessible for most researchers outside the tropics) is very difficult. The generation time of corals is measured in years and their growth rate is extremely low. Embryos are available only a few days a year in *Acropora*, or a few months in other genera. Finally, reef corals are all protected by international law and the exchange of samples between laboratories is likely to be associated with legal problems."

The anemone *Nematostella vectensis* was recommended by Darling et al. (2005): *N. vectensis* is a gonochoric anemone that has been cultured through its entire life cycle (Fautin 2002; Hand and Uhlinger 1992). "In recent years, a handful of model systems from the basal metazoan phylum Cnidaria have emerged to challenge long-held views on the evolution of animal complexity. The most-recent, and in many ways most-promising addition to this group is the starlet sea anemone, *Nematostella vectensis*. The remarkable amenability of this species to laboratory manipulation has already made it a productive system for exploring cnidarian development, and a proliferation of molecular and

genomic tools, including the currently ongoing *Nematostella* genome project, further enhances the promise of this species. In addition, the facility with which *Nematostella* populations can be investigated within their natural ecological context suggests that this model may be profitably expanded to address important questions in molecular and evolutionary ecology. In this review, we explore the traits that make *Nematostella* exceptionally attractive as a model organism, summarize recent research demonstrating the utility of *Nematostella* in several different contexts, and highlight a number of developments likely to further increase that utility in the near future."

Hydrozoans

Frank et al. (2001) recommended *Hydractinia echinata* and *H. symbiolongicarpus*: "The Cnidaria represent the most ancient eumetazoan phylum. Members of this group possess typical animal cells and tissues such as sensory cells, nerve cells, muscle cells and epithelia. Due to their unique phylogenetic position, cnidarians have traditionally been used as a reference group in various comparative studies. We propose the colonial marine hydroid, *Hydractinia*, as a convenient, versatile platform for basic and applied research in developmental biology, reproduction, immunology, environmental studies and more. In addition to being a typical cnidarian representative, *Hydractinia* offers many practical and theoretical advantages: studies that are feasible in Hydra like regeneration, pattern regulation, and cell renewal from stem cells, can be supplemented by genetic analyses and classical embryology in *Hydractinia*. Metamorphosis of the planula larva of *Hydractinia* can be used as a model for cell activation and communication and the presence of a genetically controlled allorecognition system makes it a suitable model for comparative immunology. Most importantly, *Hydractinia* may be manipulated at most aspects of its (short) life cycle. It has already been the subject of many studies in various disciplines, some of which are discussed in this essay."

Day and Lenhoff (1981), Koizumi (2002) and Shimizu and Fujisawa (2003) also recommended *Hydra* as a model animal of cnidarians:

- As a model of developmental neurobiology (Koizumi 2002): "Hydra belongs to the class Hydrozoa in the phylum Cnidaria. Hydra, is a model animal, who's cellular and developmental data are the most abundant among cnidarians. The hydra nerve net is a mosaic of neural subsets expressing a specific neural phenotype. The developmental dynamics of the nerve cells are unique. Neurons are produced continuously by differentiation from interstitial multi-potent stem cells. These neurons are continuously displaced outwards along with epithelial cells and are sloughed off at the extremities. However, the spatial distribution of each neural subset is maintained. Mechanisms related to these phenomena, i.e., the position-dependent changes in neural phenotypes, are proposed… By large-scale screening of peptide signal molecules, peptide molecules related to nerve-cell differentiation have been identified... The neurons in the nerve ring show little turnover, although nerve cells in all other regions turn over continuously. These associations and quiet dynamics lead me to think that the nerve ring has features similar to those of the central nervous system in higher animals."

- As a classical model in evolutionary developmental biology (Hemmrich et al. 2007): *Hydra* phylogenetic relationships "reveal fundamental principles that underlie development, differentiation, regeneration and also symbiosis."
- As receptor-based models with hysteresis for pattern formation (Marciniak-Czochra 2006): "The properties of the model demonstrate a range of stationary and oscillatory spatially heterogeneous patterns, arising from multiple spatially homogeneous steady states and switches in the production rates" of diffusible biochemical molecules.
- As a model of stem cell morphogenesis (Wittlieb et al. 2006): "Transgenic *Hydra* allow *in vivo* tracking of individual stem cells during morphogenesis."
- As an evolutionarily conserved model system for regeneration (Holstein et al. 2003): "They (*Hydra*) can regenerate any amputated head or foot, and when dissociated into single cells, even intact animals will regenerate from reaggregates. This extensive regeneration capacity is mediated by epithelial stem cells, and it is based on the restoration of a signaling center, i.e., an organizer. Organizers secrete growth factors that act as long-range regulators in axis formation and cell differentiation."
- As a model of heart formation (Shimizu and Fujisawa 2003): The "peduncle of *Hydra* and the heart of higher organisms share a common ancestral origin. The heart is assumed to have evolved as the organ for pumping blood. Here we report a pumping phenomenon in Hydra, a member of the phylum Cnidaria. We find that the peduncle, lower quarter of the body column, stores most of the gastrovascular fluid when the animal is an elongate form. Upon contraction of the polyp, the peduncle contracts and transfers the fluid into the rest of the cavity. We also find that Hydra RFamide III, a homolog of cardioexcitatory RFamide neuropeptides in higher organisms, elevates this transfer activity. Further, CnNk-2, a homolog of a cardiomuscular tissue marker Nkx-2.5, is expressed in the endodermal tissue of the peduncle. These observations indicate that the transfer of fluid by the peduncle has a similar neurological and genetic basis to the pumping of blood by the heart, suggesting that the Hydra peduncle and the heart of higher organisms share a common ancestral origin."
- As a model for investigating epithelial cell--basement membrane interactions (Day and Lenhoff 1981): "*Hydra* mesoglea served as a suitable substrate for the attachment and spreading of hydra cells *in vitro*, irrespective of the species tested."

Appendix III. Coral Model Species Supplementary Information

The CoralZoo Project (for further information see the following web site: http://www.ist-world.org/ProjectDetails.aspx?ProjectId=cd19d34b169247f4a3e907f1a178b772) claims to be the first comprehensive approach that makes use of molecular biology, mathematical, toxicological and nutritional tools for the development of unique breeding protocol for corals in captivity. The goal is to enable the SMEs to establish large stocks of coral colonies (the asexual approach) that represent a high genetic variability (the sexual approach) and exhibit natural growth forms.

In order to achieve the main deliverables, research will focus on the following topics:

(1) sexual and asexual breeding of corals in captivity, including breeding and feeding techniques and induction of natural coral colony morphogenesis

(2) coral husbandry: development of generic bioassays to evaluate biotic and abiotic husbandry parameters and to monitor coral health, elaboration of methods for identification and treatment of coral diseases and optimization of transport and acclimation procedures.

The consortium members bring complementary expertise to the project. Researchers at Wageningen University have experience in water recirculation systems and the culturing of marine organisms. Microbiologists specializing in coral diseases are based at the Italian Consortium for Marine Sciences, while coral biologists at the Israel Oceanography and Limnology Research Institute are primarily investigating nutritional aspects. Finally, a group at the Technical University of Dresden is modeling the data obtained from the other research partners, to predict how corals will perform under particular aquarium conditions. The academic competences are complemented by the applied aquarist's research skills of the SME partners.

Appendix IV. Biographical Sketches of Workshop Participants

Greta Aeby

Greta is a coral biologist at the Hawaii Institute of Marine Biology (HIMB). She obtained her Ph.D. at the University of Hawaii where she studied the evolution and ecology of the coral disease, Porites trematodiasis. She then completed post-doctoral training at the University of West Florida examining factors affecting the susceptibility of coral to black band disease. She returned to Hawaii and has been investigating coral and fish disease in the main and northwestern Hawaiian Islands as well as in other areas of the Indo-Pacific.

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Jeff Allen

Jeff received his BS degree in Wildlife Biology from Michigan State University and his MS degree in Geography from the University of South Carolina. He received a Ph.D. in Policy Studies from Clemson University with an emphasis in natural resources policy. Prior to coming to the Thurmond Institute, he worked for the S.C. Wildlife and Marine Resources Department as a cartographic database manager; Clemson University and the National Park Service as an outdoor recreation planner for military installations across the U.S.; and the Regional Resources Development Institute at Clemson University as its program administrator. Currently, Jeff is the Director of the South Carolina Water Resources Center as well as Research Coordinator at the Strom Thurmond Institute of Government and Public Affairs (STI) at Clemson University. He oversees all projects within the Water Center (SCWRC), Regional Development Group (RDG), and Decision and Communication Technologies Group (DCTG). His work with the SCWRC involves administering grant money from USGS, coordinating water research with a national network of water research institutes and identifying and pursuing critical water research needs for South Carolina. His duties associated with other research groups include facilitating graduate student and faculty research, supervising STI's involvement with regional development issues, and initiating new endeavors that blend the needs and expertise of STI and various academic departments. Jeff has been actively involved in community development projects as well as natural resource policy and coastal research issues. Recent projects have included developing spatial models for predicting urban growth patterns as well as collaborating with a private company (SpectroTech) to bring hyperspectral remote sensing technology to Clemson University. Additionally, Jeff's work includes project design and administration of research within the Institute's Spatial Analysis Laboratory. This facility houses computers used for geographic information systems research and remote sensing image analysis. Projects within the facility focus on providing better spatial information to decision-makers and stake-holders regarding South Carolina and the Southeast.

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Melissa Bos

Melissa joined the Alliance for Coastal Technologies several months ago as the Hawaii-Pacific Coordinator based at the Hawaii Institute of Marine Biology. Prior to this, Melissa was the Coral Reef Specialist for the Hawaii Division of Aquatic Resources where she worked on six local action strategies for coral reef management in the Main Hawaiian Islands and with the US All Islands Group of the US Coral Reef Task Force. Nutrient uptake of coral reef communities under varying flow conditions was the topic of Melissa's MS thesis at the University of Hawaii Oceanography Department. Melissa worked as an environmental consultant in Hawaii and Texas after she received a BS in Chemistry and Marine Science from the University of Miami.

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Andy Bruckner

Andy is a coral reef ecologist with the NOAA Fisheries Office of Habitat Conservation. He received his MS in marine biology from Northeastern University, Boston MA in 1988, and his Ph.D. from the University of Puerto Rico in 1999. His Ph.D. dissertation involved a study on the occurrence, impact and treatment of black-band disease. During the 1990s he devoted much of his time to Caribbean coral reef research, focusing on the effects of coral predators and coral diseases on the survival of important reef-building corals. Andy has also been involved in the development, implementation and training in coral health monitoring protocols.

Most recently, Andy works on the NOAA Coral Reef Conservation Program and the U.S. Coral Reef Task Force on international and domestic coral reef conservation activities. Through the Coral Disease and Health Consortium (CDHC) he has been working with partners to develop diagnostic criteria for coral diseases, implement a rapid response protocol to address coral disease outbreaks; and improve our understanding of the global distribution and abundance of coral diseases and relationships with environmental factors. He recently developed a coral disease identification CD for western Atlantic reefs and partnered with UNEPs World Conservation Monitoring Center to implement the Global Coral Disease Database. He also continues his research on coral diseases and predators in Bonaire, Curaçao, Puerto Rico, Jamaica and the Flower Gardens. His recent efforts on the international trade in coral reef species include analyses of the volume, sources and types of coral reef species collected for marine aquaria and curios; an identification guides for corals in trade; assistance to developing countries in the development of sustainable management guidelines for ornamental coral reef fisheries; use of CITES Appendix II listings to prevent unsustainable trade in seahorses, humphead wrasse, corals, and other species on of CITES; the development of conservation strategies for sea cucumbers, and collection and mariculture guidelines for stony corals. In addition to his coral reef research, Andy manages two parts of the NOAA coral grants program and helps coordinate NOAAs Coral Reef Conservation Programs coral reef research, monitoring and management efforts. Recent awards include a 2004 Presidential Early Career Award for Scientists and Engineers (PECASE), and a 2003 NOAA Administrators Award.

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Athline Clark

Athline was one of the first to merge science and policy together in Hawaii during her Master's Thesis in Urban and Regional Planning at the UH Manoa. She now uses her training and experience as a Special Projects Program Manager for the Hawaii Division of Aquatic Resources. In this role she is the Hawaii Point of Contact to the US Coral Reef Task Force and oversees all the projects and programs that Hawaii has initiated in response to the Task Force. This includes the creation of six Local Action Strategies to address threats to coral reefs. She is also the State lead for the Northwestern Hawaiian Islands Sanctuary designation process.

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Craig Downs

Craig received his B.A in philosophy and a B.S. in biological science from Hiram College and his M.Sc. from Syracuse University, examining the relationship of biochemical/biophysical properties of photosynthesis to physiological/ecological phenomena. He is the founder and Executive Director of Haereticus Environmental Laboratory, a tax-exempt, non-profit organization whose purpose is to build scientific and technological capacities to address environmental health concerns. This concept was inspired during his time as Chief Executive Officer of the for-profit company, EnVirtue Biotechnologies, Inc., which provided financial and technological services *pro bono* to organizations, communities, and governments whose environmental issues could, in part, be resolved by the technologies provided by EnVirtue. Craig has almost 40 peer-reviewed scientific publications and leads scientific research and environmental assessment efforts at a number of universities, including University of Hawaii and Tel Aviv University (Israel). Craig founded Haereticus in 2004, and to date, and has built numerous partnerships and collaborations with U.S. and international national, state, and city agencies, non-governmental organizations, and academic institutions throughout the world (Micronesia, Polynesia, Central America, Caribbean Islands, New Zealand, the Middle East and the Arctic). He has helped establish or expand several environmental laboratories. Craig has taught accredited courses in the fields of environmental risk assessment, ecotoxicology, cellular diagnostics, and biochemistry and as part of Haereticus' educational effort, trains NGOs and governments in the area of environmental forensics and monitoring. He also remains active in the biotechnology industry, working with industry and governments to establish new biotech/economic zones in a number of countries, as well as encouraging new biotechnology start-up companies.

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Sylvia Galloway

Sylvia is a senior research scientist for NOAA working on assignment with the Marine Biomedical and Environmental Sciences Program, Medical University of SC. She received a BS in Foods and Nutrition from Syracuse University, a MS in Chemistry from

SUNY College of Environmental Science & Forestry and her Ph.D. in biochemistry Medical University of South Carolina. Her research focus is in the biochemistry of marine animals with particular reference to coral health/disease and to forensics issues. Currently she is studying molecular indicators of coral disease in relationship to environmental and anthropogenic stressors utilizing a genomic/proteomic approach. Past areas of research have included: (1) the measurement of environmental contaminants in marine mammal and sea turtle tissues, with special emphasis on the relationship of environmental contaminants to disease and death in these species; (2) the study of the metabolism of contaminant metals with special emphasis on the interaction of Se and CH_3Hg; (3) the use of species identification techniques (including PAG-IEF, marine fatty acid analysis, DNA sequencing and RFLP) for the forensic identification of unknowns in law enforcement cases related to managed or protected marine species; (4) marine biotoxin assessment as related to human consumers of marine fishery products; particular emphasis on program management at the national level.

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Mike Gawel

Mike is the Senior Planner of Guam Environmental Protection Agency and former Administrator of the Guam Coastal Management Program, has worked with coral reef resources and environmental management issues while residing in the tropical Pacific Islands since 1969. After graduating in Biology from Yale in 1968 and studying Ecology at the Yale School of Forestry and Environmental Studies, he served in the Peace Corps in Fiji as a researcher and lecturer at the University of the South Pacific. He moved to Guam in 1973, where he completed a M.Sc. on coral taxonomy and was able to discover and describe coral species and coral reef fish species new to science. He has worked as an environmental planner and Chief Planner in Guam and other islands of Micronesia and Chief of Marine Resources in the Federated States of Micronesia, where he was married in Chuuk and his son and daughter were born in Pohnpei. During the 1980's he studied at the East West Center and University of Hawaii's Urban and Regional Planning Program. In 1989 he was hired as an evaluator of the US AID World-Wide Coastal Resources Management Project in Sri Lanka, Thailand and Ecuador. He has documented impacts of disastrous typhoons, *Acanthaster planci* outbreaks, and ship groundings on coral reefs and has worked on coral reef conservation and management planning and the development of coral reef assessment and monitoring plans for Guam.

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Marion Henry

Marion represents The Federated States of Micronesia (FSM) which are Kosrae, Pohnpei, Chuuk (formerly Truk) and Yap, stretching over a vast expanse of Pacific Ocean just north of the equator. Geographically, these four small states are part of the Caroline Islands, consisting between them of over 600 islands, of which only 65 are inhabited. Marion is the Assistant Secretary for the Division of Resource Management & Development in the Department of Economic Affairs. The Division responsibilities

include agriculture, marine resources, tourism, and environment. He has been at this position for one year after serving as the Deputy Assistant Secretary responsible for marine resources for one year. He was Vice President of the National Fisheries Corporation for six year, prior to that served as Director of the Department of Resources and Development for the Government of Chuuk State after serving as the Deputy Director for 4 years. Prior to that Marion was Chief of Marine Resources for Chuuk State, serving in that capacity for more than five years.

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Julie Higgins

Julie received her B.S. in Ecology and Evolutionary Biology and her Master's in Microbiology from the University of Tennessee. Her Master's research investigated viral dynamics in high-nutrient, low-chlorophyll marine surface waters. Last year, Julie accepted a position with Dr. Cheryl Woodley's lab, where she is pursuing molecular approaches to studying marine diseases. Her current projects involve using PCR and DNA sequencing to conduct surveillance screening of Caribbean corals for known and suspected coral pathogens. Julie also just received her working diver certification from NOAA's Diving Center in Seattle, WA.

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Dave Jessup

David Jessup was the first wildlife veterinarian hired by the California Department of Fish and Game. He spent 15 years working on terrestrial species including elk, deer, bighorn sheep, wild pigs, bear, cougar and waterfowl, primarily their diseases, anesthesia, capture and translocation. He has spent the last 15 years working on the health and welfare of marine mammals and birds and marine ecosystems. Dave has authored or co-authored over 230 scientific and popular publications, book chapters or monographs from 1975-2006. He has also worked in Mexico, India and Africa on wildlife health and conservation problems. Dave currently supervises the Marine Wildlife Health and Research Center in Santa Cruz, CA for the OSPR division of CDFG.

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Bob Jonas

Bob received his Ph.D. in Environmental Microbiology (1981) from University of North Carolina, Dept. of Environmental Sciences and Engineering as well as a Master's of Science in Public Health also from UNC. He is Associate Professor of Environmental Science and Public Policy at George Mason University and former Director of Graduate Programs in Environmental Science and Public Policy. Bob's research focuses on microbial responses in stressed ecosystems (e.g. the Chesapeake Bay, coral disease). Along with Dr. Esther Peters he has taught a course in Diseases of Corals and Other Marine Organisms through Mote Marine Laboratory each summer since 1996. In a

collaboration between the College of the Bahamas and George Mason University he has been investigating white plague type II as well as black-band disease affecting corals along the barrier reef east of Andros Island. Currently he and his collaborators (Drs. Gillevet and Peters) (and of course the graduate students) are working on white plague (or white plague-like disease) from reefs around Lee Stocking Island, The Bahamas, and St Croix, USVI and a white syndrome (white plague-like) at the Flower Garden Banks National Marine Sanctuary.

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Eugene Joseph

After graduating from the College of Micronesia – Federated States of Micronesia (COM-FSM) in 1999 with an Associate's Degree in Marine Biology, Eugene was already motivated to work on protecting Pohnpei's coral reef, so in early 2001, he started working with the Conservation Society of Pohnpei (CSP). CSP Marine Program combines elements of traditional marine resource management with modern science to empower local communities to protect Pohnpei's marine biodiversity. Currently, the program's main focuses are MPA establishment, management and networking, spawning and aggregations, fish and coral monitoring, and income generating activities for MPA communities. Long before Eugene started at CSP, Pohnpei State Division of Marine Resources (DMR) has been exercising marine biophysical monitoring and data collection. However, there was no monitoring plan in place to keep the program running. Fortunately, a biophysical monitoring plan was successfully established in 2004 through a firm partnership between Palau International Coral Reef Center (PICRC), The Nature Conservancy (TNC), Pohnpei Marine Development and CSP. Two monitoring protocols were developed in the plan; a simple community based monitoring exercise and a more rigorous/science-based program to help monitor the changes in fish populations and coral reef cover and health. Eugene is currently a Marine Program Manager and as the monitoring team leader, aims to continue improving CSP marine program by enhancing project partnership both nationally and internationally. He is representing the Conservation Society of Pohnpei, Federated States of Micronesia at this workshop.

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Esti Kramarsky-Winter

Esti received her Ph.D. from Tel Aviv University. She conducted her studies in the laboratory of Professor Yossi Loya where she studied strategies in the survival of corals from stressed environments, with an emphasis on reproduction and regeneration. She has continued her work in corals, merging coral ecology with cellular physiology and microscopic anatomy and physiology. Her interests lie in understanding processes of coral tissue repair particularly as they pertain to the ability to resist and recover from disease. In addition, in collaboration with Dr. Ariel Kushmaro of Ben Gurion University, she is currently investigating the role that symbiotic microorganisms (bacteria and protists) may have in coral holobiont physiology. Esti is a leader in light and electron

microscopy of corals and is an active participant with other CDHC members in developing the field of coral pathology.

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Jennifer Kozlowski

Jennifer is a Coral Reef Program Specialist for NOAA's National Ocean Service Coastal Programs Division where she directly works with the state of Florida on their Local Action Strategy activities and is the Program Officer for Florida's annual Coral Reef Management Grant. She also serves as the lead for her office on coastal watershed management, land-based pollution, and coral disease-related issues in the states and Territories with coral resources and the U.S. Freely Associated States. Prior to joining NOAA, she worked with the EPA Chesapeake Bay Program as a coordinator of their Community-based Watershed Initiative. Jennifer has significant field experience during her Master's thesis where she studied white and black band diseases in the Florida Keys and Bahamas, she then spent 6 years conducting pharmaceutical research and development, and analytical development for both veterinary and pediatric vaccines. Among her other interests Jennifer volunteered at the University of Maryland Center for Marine Biotechnology investigating microbial communities in marine sponges as possible sources of medically important anti-viral, anti-bacterial or anti-cancer compounds, and worked in a research lab at Aberdeen Proving grounds developing a molecular-based rapid test methodology for detecting waterborne pathogens in drinking water sources.

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Ariel Kushmaro

Ariel is the head of the Environmental Biotechnology lab at Ben-Gurion University, Israel. Research activity includes investigation of the diversity, and distribution of microorganisms through approaches based on molecular (e.g. 16S rRNA analysis, FISH and DGGE) and novel culturing techniques. In addition his research work is aimed at understanding the structure and function of microbial communities and their dynamics with regard to the environment. Research projects include; coral diseases (e.g. bacterial bleaching and black band disease), novel antibiotics from cultured and uncultured microorganisms, characterizing microorganisms from coral surfaces and their potential roles in the marine ecosystem.

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Jo-Ann Leong

Jo-Ann is Director of the Hawaii Institute of Marine Biology at Coconut Island. She received her Ph.D. in Microbiology from the University of California, School of Medicine and her A.B. in Zoology at the University of California at Berkeley. Her research interests are in molecular virology, vaccine development and phylogeography. Viruses that infect aquatic organisms are important disease pathogens and their impact on aquacultured species such as salmon and trout can be devastating. These viruses, particularly infectious hematopoietic necrosis virus (IHNV), a rhabdovirus, and infectious pancreatic necrosis virus (IPNV), a birnavirus, are so lethal that 90% of salmon production at a hatchery can be lost to these diseases. Her laboratory is developing vaccines and other treatments to control these diseases in fish. After her arrival in Hawaii, Jo-Ann has become intrigued by the devastating diseases in corals and tropical fish and expanded her research into these research arenas. Jo-Ann helped in the formulation of the current CDHC National Research Plan and has been an active member in the Consortium since 2001.

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Qing Xiao Li

Qing is a professor in the Department of Molecular Biosciences and Bioengineering, University of Hawaii at Manoa. He is also the director of the Pesticide Residue Research Laboratory at UH Manoa. Qing received his Ph.D. from the University of California at Davis, California, in 1990, and his post-doctoral training in the University of California at Berkeley from 1991 to 1994. His current research is in the areas of analytical and environmental chemistry; marine pollution and toxicology, immunochemistry and antibody-based assays for environmental applications, phyto-remediation, bioremediation, microbial degradation, environmental metabolomics and proteomics. He has more than 100 peer-reviewed publications in these research areas and is interested in effects of pollutants on coral, particularly persistent organic pollutants.

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Kay Marano-Briggs

Kay received her Ph.D. from George Mason University where she studied the highly sulfidic Bahamian Blue Holes, characterizing the chemistry of this extreme environment and identifying novel microbial species. Kay is currently employed by the US Geological Survey and coordinates their biological research in the international arena. As an Affiliate Professor in the Department of Environmental Science and Policy at George Mason University, Kay collaborates with several GMU faculty on applied research topics related to coral disease and the marine environment. Her current research interest addresses the question of whether diver wetsuits can serve as a possible vector of disease, both for coral and humans. Kay has two daughters and five Thoroughbred horses, all of which necessitate her continued employment.

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Eric Mathur

In May 2006, Eric accepted a position as a scientific consultant (Animales, LLC) for the J. Craig Venter Institute and Synthetic Genomics, Inc. In addition, earlier this year, he became a delegate of the EO Wilson Biodiversity Foundation. Prior to these recent positions, he was the first employee and one of the scientific founders of Diversa Corporation. During his 12 year tenure at Diversa, he assembled the research organization, was instrumental in the development of their intellectual property portfolio and one of the lead technical scientists for business development. At various times he was responsible and had direct oversight for most of the research and development organization, including enzyme discovery, gene evolution, gene expression, cell engineering, sequencing, bioinformatics, subcloning and manufacturing. He eventually served as Vice President of Scientific Affairs and Molecular Diversity at Diversa Corporation. His Scientific Affairs responsibilities included external scientific spokesperson for Diversa Corporation, public relations officer and of Diversa's Biodiversity Access Program involving management of relationships with over 20 Countries and Institutions. Eric is the principle investigator on four separate DOE Initiatives for Proliferation Prevention (IPP) grants and has directed research in five former bioweapons laboratories in Russia. He also had oversight and directed Diversa's Molecular Diversity Program, which involved recovery and isolation of environmental nucleic acids, metagenomics, high throughput microbial cultivation and sequence-based gene and pathway discovery. Prior to Diversa, Eric was a founding Senior Scientist at Stratagene Cloning System, a La Jolla based biotechnology company where he was responsible for the discovery of Pfu DNA polymerase, Stratagene's current largest selling product. Eric also performed research at Scripps Research Foundation and at the University of California, where he received his BS degree in Biology with Highest Honors in 1977. Eric is an National Fellow of the Explorer's Club, he sits on the Scientific Advisory Boards of Synthetic Genomics, Inc., The Explorer's Club, Department of Energy's Joint Genome Institute, the Monterrey Biotechnology Institute and the Thermal Biology Institute at Montana State University; he has been a member of the SETI Life in the Universe Working Group, the PREVCON (Prevention of Forward Contamination of Mars) Space Studies Board of the National Academy of Science and served on the Sloan Foundation's International Census For Marine Microorganisms Technology Working Group. Additionally, Eric is an adjunct professor at the University of Hawaii's Institute of Marine Biotechnology and holds Visiting Distinguished Scientist positions at both the Institute of Thermal Biology at Montana State University and the International Center for Insect Physiology and Ecology in Kenya; sits on the Queensland North America Biotechnology Council, Cal State San Marcos Biotechnology Advisory Council, and is an Editor of the journal, *Extremophiles*. Eric has published over 60 scientific papers, is named inventor on more than 25 issued US patents and has been invited to present over 100 scientific lectures.

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Amanda McLenon

Amanda is currently working with the CDHC as an Educational Coordinator, developing training materials for conducting Coral Disease Outbreak Investigations. As a recent "transplant" from Ann Arbor, MI she will begin the College of Charleston, SC graduate program in Marine Biology in the fall. She has seven years of professional experience as a high school biology teacher, specializing in science literacy. Her education to date includes a Bachelor of Science Education in biology and chemistry from the University of Michigan, and a Master of Arts in Teaching from Marygrove College. Her goal in returning to graduate school is to gain significant laboratory and field experience. Ultimately, she wants to use her strong education background to help change global perceptions of the ocean and its importance. Her broad interests include coral ecology and disease, anthropogenic influences on marine ecosystems, macroalgae, and symbiotic relationships.

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Margaret Miller

Margaret is an Ecologist with the NOAA Fisheries' Southeast Science Center. She received an undergraduate degree from Indiana University and a doctorate in marine ecology from the University of North Carolina (Chapel Hill). Her dissertation involved ecological studies of non-reef building coral, Oculina spp, off North Carolina and factors that determined their growth and distribution. She then moved south to examine some "real" corals, in a three years post-doctoral position with the University of Miami. She began work for NOAA-Fisheries in 1997 as the sole benthic ecologist at the Miami Lab and has served as a foundation for its growing coral reef program. She is an active field researcher and diver. Her involvement with coral disease was forced upon her in the course of monitoring activities focused on *Acropora palmata* and *A. cervicornis* in the Florida Keys as massive die-offs were observed. Her disease-related research has mainly involved field assessment and transmission experiments and collaborating with the CDHC in outbreak investigation and other sample collections. Her additional current research foci include coral early life history, population studies of threatened elkhorn and staghorn corals and their threats, and area assessments of reef and fisheries status around remote Navassa Island. She lives with her husband and 2-year old son in Miami.

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Gary Ostrander

Gary is the Vice Chancellor for Research and Graduate Education at the University of Hawai‘i at Manoa. He received his Ph.D. from the University of Washington and completed his postdoctoral training in the Department of Pathology at the University of Washington Medical School in biochemical oncology. In 1990 he joined the Department of Zoology at Oklahoma State University as an Assistant professor and was promoted and tenured in 1993. He assumed the role of Directory of the Environmental Institute and Associate Dean of the Graduate College in 1995. Gary joined the faculty at Johns

Hopkins University in 1996. At the time of his departure in 2004 he was the Associate Provost for Research and Chair of the Graduate Board. While at Hopkins he held academic appointments in the Department of Biology and the Department of Comparative Medicine. Gary has authored over 80 technical papers and book chapters, edited 4 books and written a field guide. His primary research interest has been elucidating mechanisms of chemical carcinogens for which he has employed aquatic, rodent and human models. A second aspect of his work has involved both laboratory and field studies focused on understanding world-wide deterioration of coral reef ecosystems.

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Pam Parnell

Pam is a veterinary pathologist, and also serves as Director of the Clemson Veterinary Diagnostic Center in Columbia, South Carolina. She received her BS in Biology from Wofford College in 1983, Doctor of Veterinary Medicine (1987) and Doctor of Philosophy in Veterinary Pathology (1993) from the University of Georgia where she also completed her residency in anatomic veterinary pathology. She is a Diplomat of the American College of Veterinary Pathologists (1995) and has 13 years experience in diagnostic veterinary pathology. Research interests have included isolation and characterization of tumor growth factors, and more recently, viral diseases of shrimp and effects of environmental estrogens on development of mammary tumors in mice. She has participated in the CDHC since 2002.

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Esther Peters

Esther received her Ph.D. in biological oceanography from the University of Rhode Island, followed by a Postdoc where she studied at the National Museum of Natural History and the Registry of Tumors in Lower Animals. She is an internationally recognized expert on coral reefs and diseases of coral reef organisms, contributing chapters to books (e.g., *Life and Death of Coral Reefs*) and journal articles, including a review (with others) on "Ecotoxicology of Tropical Marine Ecosystems" (*Environ. Toxicol. Chem.* 16(1):12-40). Her research includes the effects of exposures to biological, chemical, and physical environmental stressors on a variety of invertebrates and fishes, comparative histopathology, and risk assessment. Her experience with examining the distribution of diseased corals and histopathological examinations of diseased corals includes the Florida Keys, northern and western Caribbean, Gulf of Mexico, and Hawaii. Esther is also an adjunct scientist, Mote Marine Laboratory; adjunct professor, where she has been teaching the course "Diseases of Corals and Other Reef Organisms" at the Tropical Research Laboratory, Summerland Key, Florida, since 1997. She also holds adjunct positions at Nova Southeastern University, Oceanographic Center, collaborating with scientists at the National Coral Reef Institute there; and an affiliate professor, Department of Environmental Science and Policy, George Mason University. Esther's involvement with coral issues also includes participating in the interagency Coral Disease and Health Consortium, U.S. Coral Reef Task Force, and the

Technical Advisory Committee on Land-Based Sources of Pollution for the Southeast Florida Coral Reef Initiative. In addition to all of these activities she her 'day job' is as an Environmental Scientist VI (toxicology and pathobiology) and Quality Assurance Manager in the Fairfax, Virginia, office of Tetra Tech, Inc.

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Bob Richmond

Bob is presently a Research Professor at the University of Hawaii's Kewalo Marine Laboratory. He previously served as a Professor of Marine Biology at the University of Guam Marine Laboratory for 18 years, and as Director of the Marine Lab from 1988 - 1991. He received a B.S. in Biology/Geology with High Distinction from the University of Rochester in 1976, an M.S. in Marine Environmental Sciences from the Marine Sciences Research Center, SUNY at Stony Brook, in 1982 and a Ph.D. in Biological Sciences from the Dept. of Ecology and Evolution, SUNY at Stony Brook, in 1983. Following completion of his graduate work, he received two postdoctoral fellowships, from the Smithsonian Institution and the Smithsonian Tropical Research Institute in Panama. He has spent most of his professional career studying coral reef ecosystems in both the Caribbean and the Pacific. He began his studies in the U.S. Virgin Islands and the Grenadines, and since 1980, has worked extensively throughout the Pacific Islands, from Hawaii to Palau. He served as Chief Scientist for a World Conservation Union-sponsored mission to the Galapagos Islands, and has assisted numerous Pacific Islands in dealing with marine resource and related conservation issues. He is the scientific advisor to the All-Islands Committee of the U.S. Coral Reef Task Force, and served as a Council Member for the International Society for Reef Studies. He was awarded an Aldo Leopold Fellowship in Environmental Leadership in 2004, and a Pew Fellowship in Marine Conservation in 2006. He works closely with island community-based organizations, traditional leaders and stakeholders, and has trained over 35 Pacific Islanders in his laboratory over the years. His present interests include coral reef ecology, marine conservation biology, ecotoxicology and integration of traditional management systems with modern approaches to resource use and protection.

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Mike Risk

Mike received his BSc (Hon. Geology) U Toronto, MSc Sedimentology U Western Ontario, and Ph.D. Marine Biology, with Gerry Bakus. Mike specialises in assessing impacts on reefs of land-based sources of pollution, and in climate change as recorded in skeletons of shallow and deep-water corals. He has worked in 40 countries, lived in seven-speaks five languages equally badly, including English.

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Emmett Shotts

Emmett received a BS in Bacteriology, in 1952, from Alabama; CMT, Medical Technology in 1953, from the Medical College of Alabama; MS, Medical Microbiology, in 1958, from Georgia and a PhD, Medical Microbiology in 1966, from Georgia. His specialty certifications include, Medical Technologist (ASCP); Specialist Microbiologist (Public Health/Medical), National Registry Microbiology; Diplomate, American College of Veterinary Microbiology (ACVM); Diplomate, American Veterinary Epidemiology Society (AVES); and a fellow, American Academy of Microbiology (AAM). Professionally, Emmett is active in the American Society for Microbiology (Clinical Section); American Fisheries Society (AFS; Fish Health Section); Wildlife Disease Assoc; International Assoc. Aquatic Animal Medicine; European Assoc. Fish Pathology; Epidemic Intelligence Service, Alumni Assoc; American Veterinary Medical Assoc (Affiliate); Editorial Board of Journal of Fish Diseases. During his career which has spanned some 45 years, he has received much recognition including the Snieszko Distinguished Service Award (AFS), the Distinguished Service Award (Wildlife Disease Assoc) and a Creative Research Medal from the University of Georgia. He has contributed over 250 manuscripts to Scientific Community in the form of articles, book chapters and published Abstracts, has two patents and made over 180 presentations. His research interests and expertise is varied and includes: Microbial Diseases of aquatic and terrestrial animals as well as humans, Zoonoses, Mechanisms of Pathogenesis, Molecular Technology, Mycology, Epidemiology, Ecology, Automated Systems, Antimicrobics, and Microbial Media development. His career has included affiliations and positions at Fort Detrick, MD. US Army (1954-56); Southeastern Wildlife Disease Study, Athens, GA (1956-59); Epidemic Intelligence Service officer, CDC; Asst. to the Chief, Veterinary Public Health Laboratory, CDC; Asst. to the Chief, National Rabies Investigations Laboratory, CDC, Atlanta (1959-64); Asst. Prof., Department of Pathology; Assist. Professor, Department of Medical Microbiology and Parasitology, Assoc. Prof., Prof., Prof. Emeritus, College of Veterinary Medicine, University of Georgia, Athens, GA (1966-97); and Director, National Fish Health Research Laboratory, Leetown Science Center USGS/DOI (1997-2000). During his career his research has carried him to Germany, England, Scotland, Ireland, Japan, Taiwan and Russia.

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Katie Siegler

Katie works for the Division of Aquatic Resources (DLNR-DAR) as the NOAA Coral Reef Fellow for the State of Hawaii. She is working with Greta Aeby on finalizing the Climate Change and Marine Disease Local Action Strategy. She has a Master's Degree in Environmental Science and is interested in pursuing further graduate studies in marine disease.

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Meir Sussman

Meir recently completed his Ph.D. under the mentorship of Drs. Bette Willis (James Cook University) and David Bourne (Australian Institute of Marine Science). His Ph.D. research investigated coral diseases on the GBR and in the Indo-Pacific where he isolated, cultured, and identified causative agents for 4 coral diseases. Meir then identified specific proteins and their gene transcripts, actively involved in these diseases that are part of bacterial virulence mechanisms expressed during coral infections. He plans to continue this line of research with the goal of developing management-applicable diagnostic tools for coral diseases.

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Mac Terzich

"Dr. Mac" is a Board certified veterinarian (Diplomate ACPV) that operates Pacific East Aquaculture, Inc., one of the nation's largest marine ornamentals captive coral propagation facilities, located on Maryland's Eastern shore in Mardela Springs. Pacific East Aquaculture is a Maryland State inspected and licensed coral aquaculture facility with the capacity to propagate and grow thousands of corals, and marine fishes and invertebrates. Mac is also active in sponsoring, setting up, and maintaining coral propagation projects in the Solomon Islands, and sponsoring research with local universities in coral health and captive propagation of marine ornamentals.

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Katherine Tucker-Mohl

Katherine received her degree from the University of Pennsylvania, School of Veterinary Medicine. She received a Veterinary Medicine Fellowship to work with Dr. Patrick Hart, Biological Resources Division - Pacific Island Ecosystems Research Center on *"Avian malaria in Hawaii: evaluating prevalence of infection and host resistance in the Common Amakihi"*. Her study seeks to build on recent observations that certain species of native birds have returned to lowland habitats and have established successful populations, despite high levels of chronic malarial infections. Research will compare the prevalence of avian malaria in two previously unstudied lowland populations of Common Amakihi near Kona, on the island of Hawaii, with the ultimate goal of improving conservation programs for, and the health of, endangered Hawaiian forest birds. She has recently accepted an internship with Dr. Thierry Work where she will experience treating and studying a variety of diseases in tropical organisms.

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Bernardo Vargas

Bernardo works for NOAA's Coral Reef Ecosystem Division in Hawaii, leading efforts to design and develop methods for identifying and characterizing coral disease in shallow-water coral reefs ecosystems of the U.S. Pacific Islands. He holds a Ph. D. from the University of Miami, Rosenstiel School of Marine and Atmospheric Science. Bernardo's research interests include coral reef community dynamics, with special emphasis on coral health status, disease incidence, prevalence, and development of diagnostic criteria based on field observations and histopathological examinations.

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Steven Victor

Steven is currently Head of the Research Department at the Palau International Coral Reef Center (PICRC) and has been a member of the research team since 2002. His main research interest is determining the effects of anthropogenic disturbances on coastal and marine resources, particularly coral reef habitats. In an effort to protect Palau's natural resources, Steven has worked with local partners such as The Nature Conservancy, Palau Conservation Society, local government agencies and international partners such as NOAA, the World Bank Targeted Research Group on Remote Sensing, Coral Disease, and Remediation, and Restoration Group, and Scientists from Australian Institute of Marine Science and University of Hawaii to understand how to best minimize human impacts on coral reef resources. Steven completed both his undergraduate and graduate degrees in Biology at the University of Guam. He continues to do research on mangroves, seagrasses and coral reefs in Palau and Micronesia.

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Bruce Wilcox

Bruce is a Professor of Tropical Medicine, Medical Microbiology and Pharmacology, at the John A. Burns School of Medicine, University of Hawaii. He is also the Director of the Asia-Pacific Center for Infectious Disease Ecology at the Asia-Pacific Institute for Tropical Medicine and Infectious Diseases. His fields of study were population biology, ecology and evolutionary biology and biology during his academic career at U. of California, San Diego and Yale. In addition, he has served in leadership positions in numerous Public Health organizations and as the founding Editor-in-chief of EcoHealth. He is an active teacher and researcher. Bruce's interest in emerging infectious disease, ecosystem management and disease ecology has recently synthesized into a research focus on social-ecological systems and the relation with emerging infectious disease.

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Dana Williams

Dana earned her doctorate degree in 2002, at the University of South Florida working with Dr. Pam Hallock-Muller on the population dynamics of a reef foraminifer. She then began post-doctoral research at the University of Miami Rosenstiel School (RSMAS) working with Dr. Margaret Miller (National Marine Fisheries Service) on acroporid coral populations in the Florida Keys. In 2003 Dana's discovery of an atypical disease outbreak in *Acropora cervicornis* in the Florida Keys led to her involvement in the CDHC and its first coordinated response and investigation of a coral disease outbreak. Since this outbreak investigation, Dana has continued to contribute her time and expertise toward refining the field sampling protocols for the CDHC's overall coral disease outbreak investigation response plan. Currently Dana is an assistant scientist at RSMAS working on *Acropora* spp. population monitoring and restoration methods in the Florida Keys.

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Bette Willis

Bette is a Professor at the ARC Centre of Excellence for Coral Reef Studies and an Associate Professor in the School of Marine Biology and Aquaculture at James Cook University (Townsville, Australia). Her research explores questions relating to the biology and ecology of scleractinian corals. Current research focuses firstly on the prevalence and ecological significance of coral disease on the Great Barrier Reef, and secondly on the implications of flexibility in algal endosymbiosis for the physiology of the coral host, particularly in relation to thermal tolerance and climate change. She is a Co-Chair of the GEF Working Group on Coral Disease (Targeted Research and Capacity Building for Management program), a primary goal of which is a global assessment into the causes, origins and impacts of coral disease worldwide.

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Wendy Wiltse

Wendy is an environmental scientist with US EPA Region 9 in Honolulu where she works on coral reef, wetlands, and water quality management. Wendy has worked on a range of EPA's water quality management programs, coordinated a watershed management project on Maui, and is active in Hawaii's Local Action Strategy to address land-based pollution threats to coral reefs. She's worked in Hawaii for over 12 years. Wendy has a Ph.D. in Marine Ecology from the University of Massachusetts.

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Gary Wobeser

Gary is with the Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, Canada. He was raised on a mixed farm in Saskatchewan and trained initially as a wildlife biologist (BSA, MSc) and then entered Veterinary Medicine (DVM) and specialized as a veterinary pathologist (my PhD dealt with aquatic mercury poisoning in fish and mink). He has been a faculty member of the Department of Veterinary Pathology since 1974 and served a stint as Head of the Department. Gary was founding co-director of the Canadian Cooperative Wildlife Health Centre, which is a cooperative enterprise among Canada's four veterinary colleges, and serves all federal and provincial wildlife resource agencies. He teaches veterinary pathology at the undergraduate and graduate level, and wildlife diseases at the graduate level. He also does diagnostic pathology for both domestic and wild species. Gary's research interest includes disease of all types in free-living animals and he has worked at one time or another with many different diseases in mammals, birds and a few in fish. He has published three books on the subject: Diseases of Wild Waterfowl (in its second edition), Investigation and Management of Disease in Wild Animals (currently being revised), and Essentials of Disease in Wild Animals (published in 2006).

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Stephanie Venn-Watson

Stephanie Venn-Watson, D.V.M., M.P.H. received her veterinary degree from Tufts University School of Veterinary Medicine (1999) and public health degree from Emory University Rollins School of Public Health (2000). During 1999-2001, Stephanie was a veterinary epidemiologist at the Centers for Disease Control and Prevention (CDC) and served as Project Director for the World Health Organization's Global Salmonella Surveillance System (WHO Global Salm-Surv). Stephanie completed a National Research Council Associateship at the U.S. Navy Marine Mammal Program (MMP) during 2001-2002. She has been a civil servant veterinary epidemiologist at the MMP since 2003. Stephanie's primary responsibilities are epidemiological investigations; marine mammal infectious diseases; oversight of population health and database development; and coordination of clinical research.

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Cheryl Woodley

Cheryl received her Ph.D. in Molecular and Cellular Biology and Pathobiology from the Medical University of South Carolina. Since 1992, she has served as a research scientist with NOAA's Center for Coastal Environmental Health and Biomolecular Research in Charleston, SC and also holds graduate faculty positions at the Medical University of South Carolina and the College of Charleston. Over the past five years, her expertise in virology, molecular biology and biochemistry has been used in adapting biomedical concepts and technologies to investigate the effects of natural and anthropogenic stressors on coral heath. Recently her laboratory joined a multi-investigator, multi-agency Marine Genomics team at the Hollings Marine Laboratory, providing access to state of the art genomic and proteomic technologies to identify diagnostic indicators useful in determining the health status of coral reef organisms. In 2002, she helped organize and establish the Coral Disease and Health Consortium (CDHC) in response to the US Coral Reef Task Force's National Action Plan to Conserve Coral Reefs. The mission of the CDHC is "to understand and address the effects of natural and anthropogenic stressors on corals in order to contribute to the preservation and protection of coral reef ecosystems". Cheryl currently serves as Coordinator of the CDHC and together with over 50 CDHC members, contributing their time and expertise, they work to unify the coral health and disease research community, identify research priorities, develop innovative technologies and encourage a new generation of coral researchers through education and outreach.

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Thierry Work

Thierry is project leader for the Honolulu Field Station of the National Wildlife Health Center (USGS). He has degrees in entomology, veterinary medicine, and preventive veterinary medicine. His interests include diseases of free ranging terrestrial and marine wildlife. Current topics of investigations include diseases of reef fish, corals, and marine turtles.

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Appendix V. List of Participants

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Appendix VI.

OPINION PAPER:

Transmission Experiments in the Field: Ethics, the Law, the Science

by Cheryl M. Woodley

"Wildlife disease practitioners determine transmissibility in the field through observational studies, not field manipulations. The most valid contribution of field studies is to provide detailed observations over time; specifically for coral lesions by describing them in very precise detail, determining by the use of specific and discriminating characteristics whether the lesions are the same or different among affected individual colonies and by recording the movements of the lesion border …noting either expansion or contraction."

There are three major factors that argue <u>*against*</u> conducting field activities, presumed to be valid transfection or transmission experiments *in situ,* in a quest to determine whether a diseased coral has an infective agent associated with visible lesions.

The first is an ethical issue. This practice is not condoned in human or veterinary medicine and is in direct opposition to the philosophy of conservation medicine, 'to do no harm'. There is a fundamental ethical question related to propagating disease in a 'wild' population, either within or between populations. When this manipulation is carried out, it in essence establishes another focus or node of infection, if the lesion is infectious. For example, no veterinarian would think of putting an oyster with *Perkinsus* into a 'healthy'oyster bed nor place a sick deer or bird with their herd/flock in the wild to see if members of the herd/flock got sick. A good scientific test of infectivity requires a valid statistical design requiring multiple 'nodes of infection' thus deliberately spreading the disease in an uncontrolled environment. The biologist/scientist unwittingly becomes a vector, a totally unacceptable situation.

The second factor is of a legal nature. Taking into consideration the status of coral reefs, and the fact that several species are already listed as threatened, either under the ESA or the IUCN's Red List, the transfection manipulation described presents a significant risk of further endangering the reef. This can be interpreted as a deliberate natural resource damaging action, and for Acroporids, a violation of the Endangered Species Act within U.S. jurisdiction. The IUCN (International Union for the Conservation of Nature and Natural Resources)/ The World Conservation Union has guidelines on practices related to management and wildlife conservation issues. None directly address coral or disease transmission in the wild, however the IUCN Position Statement on Translocation of Living Organisms (http://www.iucn.org/themes/ssc/publications/policy/transe.htm) points out that any movement of organisms (which includes their microbiota) needs to be screened for

disease and those with disease should not be moved. They further describe penalties that may be assessed: "Deliberate introductions without a permit as well as negligence resulting in the escape or introduction of species harmful to the environment should be considered criminal offences and punished accordingly. The author of a deliberate introduction without a permit or the person responsible for an introduction by negligence should be legally liable for the damage incurred and should in particular bear the costs of eradication measures and of habitat restoration where required."

The third factor is scientific considerations. The experimental design is fundamentally flawed when conducting a transmission manipulation within the same location/population where the disease is observed. This violates the principle of using cohorts in infectious disease studies. There are a number of confounding variables that make any results obtained from this type of study inconclusive and invalid. These include the fact that it is not known:

1) if the test subjects are already infected and not yet presenting with gross disease signs;

2) if disease signs appear in the test group whether they are the result of an infectious agent as opposed to a toxin or leachate; and

3) if disease signs appear in the test group whether they are caused by cell signaling molecules, chemical compounds released from dying tissue or proteases that propagate death (necrosis) from dying tissue.

In other words, the field studies will not conclusively determine whether an infective agent is present or not. Further, a valid statistical design would require exposing at least 9 individuals from a naïve population to determine infectivity----further deliberately spreading disease.

Ethical, legal and scientifically sound deliberate disease exposure studies should be conducted under containment regimes. A laboratory controlled population that consistently has presented with no signs of disease (naïve population) should be used as the test subject for exposure to diseased tissue. To meet criteria of evidence, the agent (infectious, chemical or toxin) should be isolated and characterized and then used to expose the naïve population to determine if the agent does elicit the same disease signs as observed in the field.

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