CORAL DISEASE, ENVIRONMENTAL DRIVERS, AND THE BALANCE BETWEEN CORAL AND MICROBIAL ASSOCIATES

BY THE CORAL DISEASE WORKING GROUP OF THE GLOBAL ENVIRONMENTAL FACILITY CORAL REEF TARGETED RESEARCH PROGRAM

ACROSS THE GLOBE, we are witnessing the decline of coral reef ecosystems. One relatively new factor contributing to this decline is the outbreak of destructive infectious diseases, especially on Caribbean reefs. As the Coral Disease Working Group of the Coral Reef Targeted Research Program, our research focuses on four priorities: (1) assessing the global prevalence of coral disease, (2) investigating the environmental drivers of disease, (3) identifying the pathogens that cause disease, and (4) evaluating the coral's ability to resist disease. Monitoring has revealed new coral-disease syndromes at each of four Global Environmental Fund Centers of Excellence: the Caribbean, the Philippines, Australia, and East Africa. Over the last 20 years, drastic (> 50 percent) loss of coral cover has occurred on the Yucatán Peninsula, even in pristine areas.

Global surveys have revealed significant levels of disease and disease outbreaks occurring not only in the Caribbean "hotspots," but also in sites throughout the Pacific and Indian Oceans. By monitoring coral disease, we will create a baseline and long-term data set that can be used to test specific hypotheses about how climate and anthropogenic drivers, such as decreasing water quality, threaten coral reef sustainability. One such hypothesis is that high-temperature anomalies drive outbreaks of disease by hindering the coral's ability to fight infection and by increasing the pathogens' virulence. We observed recurrent outbreaks following the warm summer months of two of the most damaging diseases in the Caribbean. In addition, we found that coral disease in the Great Barrier Reef correlated with warm temperature anomalies. In the Caribbean and Mediterranean Seas, virulence of known coral pathogens and the normal coral flora changed during high-temperature periods. Other stresses such as high nutrients and sedimentation may similarly alter the balance between the coral and its resident microbial flora.

INTRODUCTION

Over the past few decades, coral reef communities around the world have been deteriorating due to a combination of natural and anthropogenic factors (Harvell et al., 1999; Harvell, 2004; Hughes et al., 2003). Coral damage can be caused both by abiotic factors (e.g., temperature stress, sedimentation, toxic chemicals, nutrient imbalance, ultraviolet radiation) and biotic factors (e.g., predation, overgrowth of algae, infectious disease). These factors, acting alone or in synergy, have led to a reduction in coral cover (Green and Bruckner, 2000; Richardson and Aronson, 2002; Hughes et al., 2003). Infectious disease in coral, observed in the field as lesions or distinct bands of tissue loss, can be caused by bacteria, viruses, protozoa, or fungi. In addition to the loss of coral tissue, disease can cause significant changes in reproduction rates, growth rates, community structure, species diversity, and abundance of reef-associated organisms (Loya et al., 2001). While an unprecedented increase in coral disease has been well documented in the Caribbean (Porter et al., 2001; Weil et al., 2002; Weil, 2004; Weil et al., 2006), much less is known about the status of disease throughout the Indo-Pacific. However, preliminary surveys in Australia (Willis et al., 2004), the Philippines (Raymundo et al., 2004), Palau (Cathie Page and others, James Cook University, *pers. comm.*, December 2006), and East Africa (McClanahan et al., 2004; Ernesto Weil, University of Puerto Rico, *pers. comm.*, December 2006) revealed significant and damaging new diseases in all locations surveyed.

What has prompted this emergence of coral disease? Current research suggests that climate warming is an important factor (Harvell et al., 2002; Selig et al., 2006). Tropical reef-building corals are generally found between the Tropic of Cancer (23.5°N) and the Tropic of Capricorn (23.5°S). Because they have a narrow range of thermal tolerance (between 18° and 30°C), they are extremely susceptible to temperature stress. It is well known that corals "bleach" (lose their symbiotic zooxanthellae) at high, stressful temperatures. The coral bleaching observed worldwide following the 1998 El Niño was the most massive and devastating recorded up to that point (Hoegh-Guldberg, 1999), only to be exceeded by another bleaching event in Australia in 2002. The fall of 2005 brought devastating bleaching to the Caribbean, caused by the largest warm thermal anomaly in 100 years (Mark Eakin, National Oceanic and Atmospheric Administration, *pers. comm.*, December 2006). The Caribbean thermal anomaly of 2005 was immediately followed by outbreaks of white plague and yellow blotch (Miller et al., 2006).

Our working hypothesis is that, in some cases, the death of coral during hot thermal anomalies is facilitated by opportunistic infectious pathogens whose virulence is enhanced by increased temperatures. Changing environmental conditions could also influence disease by altering host/pathogen interactions. Increased temperatures could affect basic biological and physiological properties of corals, particularly their ability to fight infection, thus influencing the balance between potential pathogen and host (Rosenberg and Ben-Haim, 2002). In addition, the pathogens themselves could become more virulent at higher temperatures (Ben Haim et al., 2003a, 2003b). This effect is particularly challenging to study because of the complexity of the coral holobiont—the coral polyp, which co-exists in a mutualistic relationship with unicellular algae, zooxanthellae, and a surface mucopolysaccharide layer (SML). The SML contains a complex microbial community that responds to changes in the environment in ways that we are just now beginning to appreciate (Azam and Worden, 2004; Klaus et al., 2005). The normal microbial flora within the mucus layer may protect the coral against pathogen invasion; disturbances in this normal flora could lead to disease (Ritchie, 2006). The massive introduction of non-indigenous pathogens, as is often

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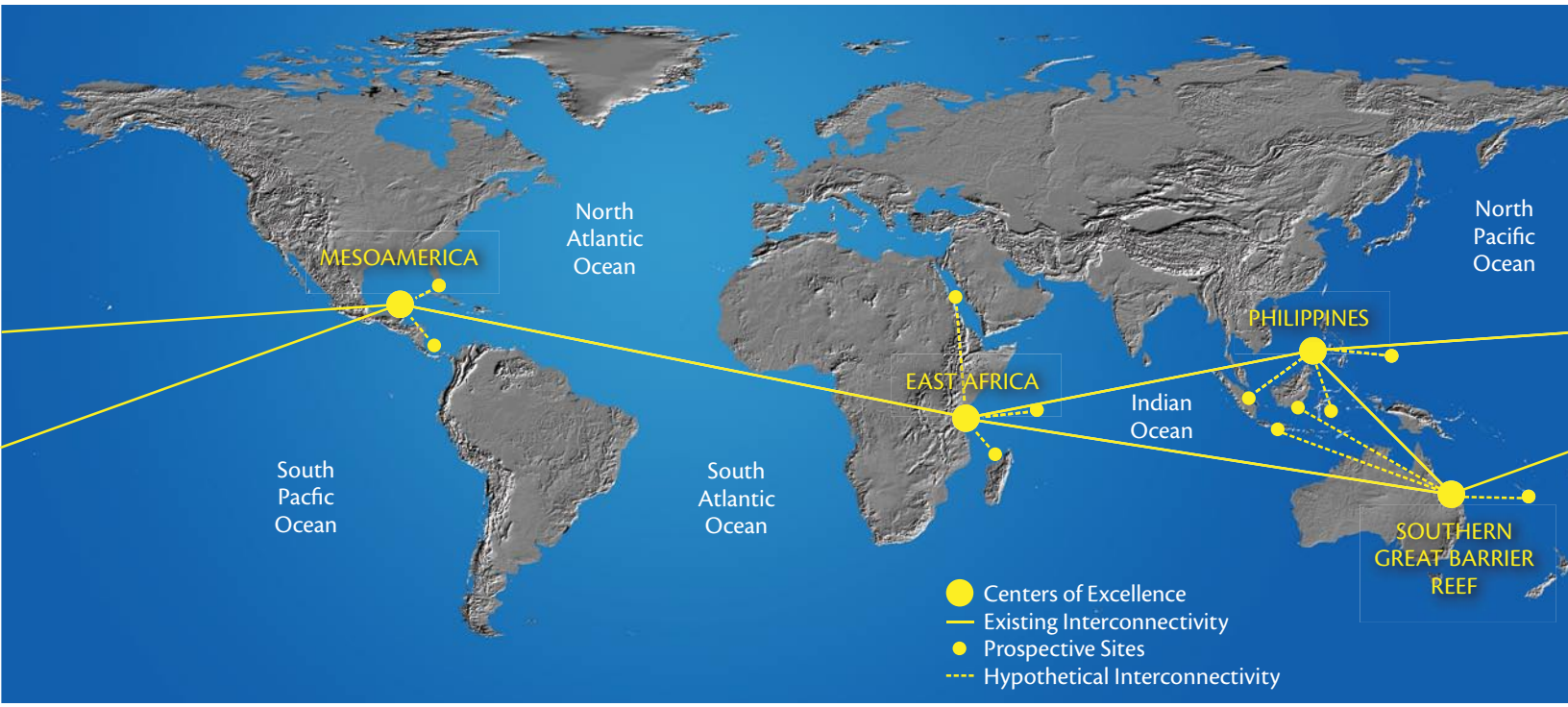

Figure 1. Map showing the Centers of Excellence for the World Bank/GEF Coral Reef Targeted Research project.

seen with aquaculture and ballast-water release, could also disturb the microbial community (Harvell et al., 2004).

Pollutants and other anthropogenic stressors could potentially impact any component of the holobiont, causing a disruption in the symbiosis and a concomitant loss of health. This loss of health could translate into a breakdown in host resistance and a potential elevation of disease severity or rate of infection. Sedimentation could alter the microbial community within the surface mucous layer of the coral holobiont. Nutrient loading could enhance both algal and pathogen growth (Bruno et al., 2003; Smith et al., 2006; Kuntz et al., 2005).

This paper details the priorities of a World Bank/Global Environment Facility initiative, the Coral Reef Targeted Research (CRTR) and Capacity Building for Management Program (for more information, go to http://www.gefcoral.org). As the Coral Disease Working Group within this project, the goals of our program are to fill critical information gaps about coral reef disease, build capacity to study and monitor disease internationally, and help develop solutions for managing and conserving reef ecosystems. We describe here the cooperative research effort being guided by our international team of microbiologists, ecologists, and physiologists toward these ends. Working out of four Centers of Excellence, our research priorities include assessing the global prevalence of coral disease, investigating the environmental drivers of disease, identifying the pathogens that cause disease, and understanding the coral's ability to resist disease. We are testing specific hypotheses about climate and anthropogenic changes that threaten coral reef sustainability. By building the capacity to manage these ecosystems, we hope to enhance reef resilience and recovery, worldwide.

1. GLOBAL PREVALENCE

The CRTR program's four Centers of Excellence are located in Meso-America, Australia, East Africa, and Philippines/Southeast Asia (Figure 1). Working from these centers as well as other localities in each region, we are assessing the global range, prevalence, and impact of coral diseases. We standardized protocols for conducting coral and disease surveys in coordinated teams that allow comparison of disease levels in highly diverse reefs such as those in the Indo-Pacific with those in the Caribbean. Although

the Indo-Pacific has far and above the highest coral diversity (Figure 2), the most reports of disease come from the Caribbean. Overall, prevalence of all coral diseases combined within a region ranges from lows of less than 5 percent in Australia, Palau, and E. Africa, and 8 percent in the Philippines (Weil et al., 2002; Willis et al., 2004; Raymundo et al., 2005; Page et al., 2006; E. Weil and A. Croquer, University of Puerto Rico, *pers. comm.*, December 2006) to a high of up to 20 percent on the Yucatán and at other Caribbean localities (Jordán-Dalgren et al., 2005; Weil et al., 2006; Ward et al., 2006). What prevalence does not reveal is the dynamics of disease outbreaks that have been recorded sporadically in all regions, but most regularly in the Caribbean.

Meso-America: Caribbean Basin

The Caribbean has historically been dubbed a disease "hotspot" because of the fast emergence, high prevalence, wide geographic distribution, and virulence of coral reef diseases there. Although only 8 percent of all coral reefs (by area) are found in the Caribbean (Spalding and Greenfell, 1997), over 70 percent of all disease/syndrome reports come from

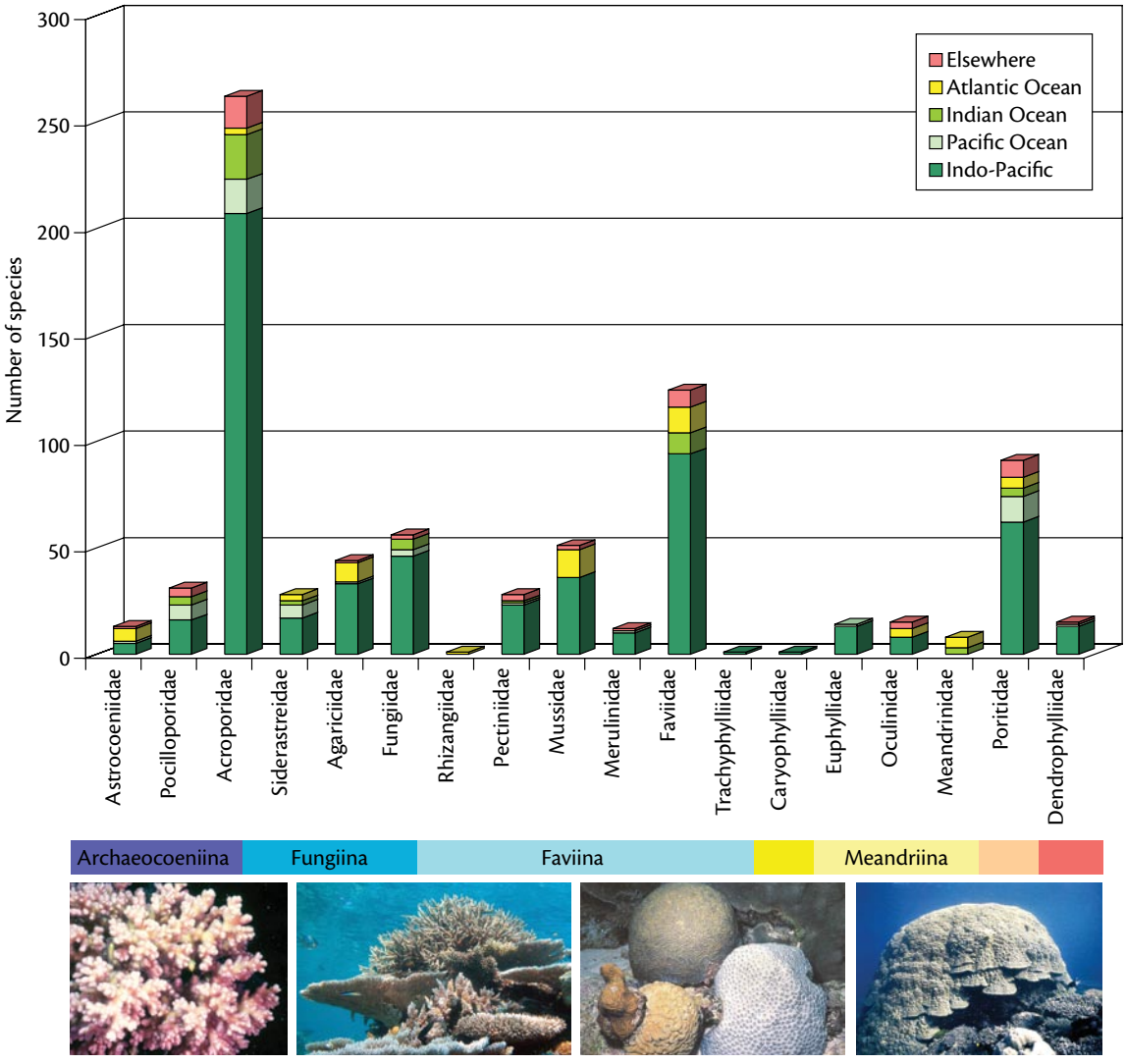

Figure 2. Species richness of reef corals within families (x-axis) and suborders (colored bar below x-axis) and their distributions across reef regions (colored portions within each histogram). Insets illustrate species within the most abundant and typically dominant coral families (from left to right: Pocilloporidae, Acroporidae, Faviidae, and Poritidae). Unlabeled suborders are: Caryophylliina (yellow), Poritiina (light orange), Dendrophyllina (dark orange). *Photos by J. Veron and B. Willis*

this region. While past bleaching events in the Caribbean did not produce high mortality rates like those reported for the Indo-Pacific (McClanahan, 2004), this rate may be changing. The 2005 bleaching event was the worst recorded and produced significant mortalities in some scleractinian (hard-coral) populations in several reef localities. Today, diseases of corals and other keystone species such as urchins have resulted in significant losses in coral cover, biodiversity, and habitat of many Caribbean coral reefs (Lessios et al., 1984; Hughes, 1994; Aronson and Precht, 2001; Weil 2004).

The first Caribbean coral-related diseases were reported in the early 1970s (Antonius, 1973; Garret and Ducklow, 1975). There are now about 20 reported diseases affecting 45 (that is, 75 percent) of zooxanthellate coral species, ten octocorals (soft coral), nine sponges, one zoanthid, and two crustose coralline algae in the region (Table 1). Potential pathogens have only been identified for seven of the commonly found coral diseases and Koch's postulates have only been fulfilled for five of these (Figure 3; see following section titled "Pathogens"). Several other common and highly viru-

Table 1. Some of the most commonly found coral diseases.

Disease	Acronym	Pathogen	# of Species Infected	
			COR	OCT
CARIBBEAN				
Black band	BBD	*P. corallyticum, Desulfovibrio, Beggiatoa* sp	19	6
White band I	WBD-I	Gram (-) bacterium	2	
White band II[2]	WBD-II	*Vibrio carchariae*	2	
White plague I	WP-I	Gram (-) bacterium	12	
White plague II[2]	WP-II	*Aurantimonas coralicida*	41	
Aspergillosis[2]	ASP	*Aspergillus sydowii*		10
White pox[2]	WPX	*Serratia marcescens*	1	
Growth Anomalies[1]	TUM	*A. endozoica* (algae) and other causes	7	5
Red band	RBD	*Oscillatoria* sp. and other cyanobacteria	13	1
Yellow blotch	YBS	*Vibrio* sp ?	11	
Dark spots I	DSS-I	*Vibrio* sp ?	10	
Dark bands	DBS-II	?	8	
INDO-PACIFIC-MEDITERRANEAN				
Porites trematodiasis	PTR	*Podocotyloides stenometra*	4	
Skeletal eroding band	SEB	*Halofolliculina corallasia*	2	
Brown band	BrB	New species of ciliate—not described	2	
Porites ulcerative white spots	PUWS	*Vibrio* sp	3	
Bacterial bleaching[2]	BBL	*Vibrio shiloi*	1	
Bacterial bleaching[2]	BBL	*Vibrio coralliilyticus*	1	
White-plague	WP	*Thalassomonas loyona*	5	

[1]Growth anomalies include hyperplasias and algal tumors.

[2]Koch's postulates fulfilled.

Presented here are some of the most commonly found coral diseases with their names, Acronym, Pathogen (if known), and Number of taxa affected for "hard" or scleractinian coral (COR) and "soft" or octocoral (OCT). Each disease is generally named for its symptoms. This informal classification system has caused some confusion in the literature, as the nomenclature is not yet standardized (adapted from Weil et al., 2006).

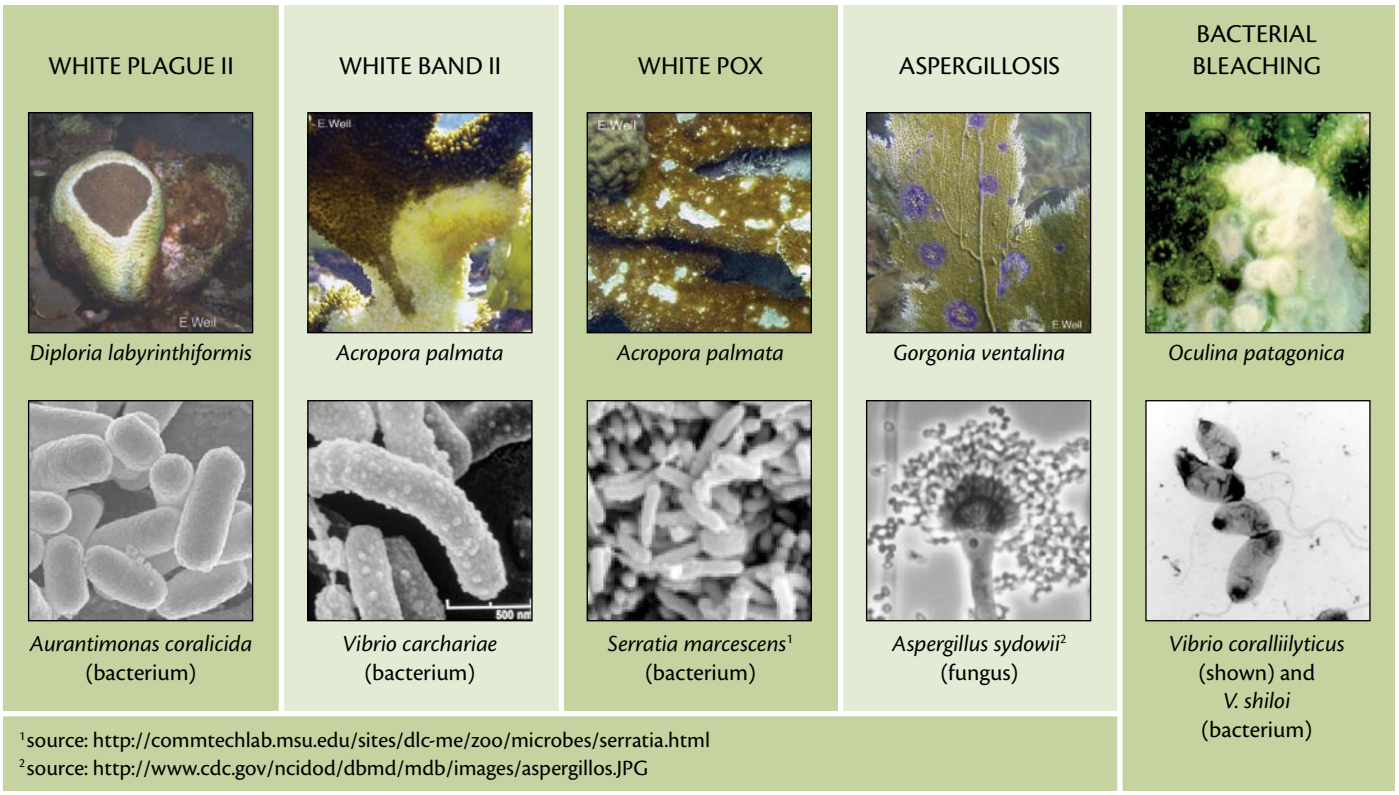

Figure 3. The five coral diseases for which Koch's postulates have been fulfilled, showing disease, host coral, and microbial pathogen. The classic way to prove a microorganism causes disease is to satisfy Koch's postulates. Briefly, a microorganism must be isolated from a diseased individual. That isolate is then used to infect a healthy individual. The same disease must develop, and the same organism must be isolated from the new infection.

lent syndromes, such as yellow blotch (Figure 4A), dark band (Figure 4D), white blotch, and tissue necrosis (Figure 4E–F) have become more prevalent and widespread in recent years, posing an increasing threat to coral and octocoral populations (Gil-Agudelo et al., 2004; Cervino et al., 2004; Smith and Weil, 2004). Furthermore, extensive surveys in many coral reefs around the wider Caribbean revealed a suite of new syndromes and problems that produce tissue necrosis and/or colony mortalities in other important components of coral reef communities such as hydrocorals, sponges, zoanthids, and calcareous-crustose algae (Figure 4J–O).

Surveys conducted from 1999 through 2004 on more than 40 reef sites in over ten geographic locations in the wider Caribbean (Weil et al., 2002; Weil, 2004; Smith and Weil, 2004) revealed several patterns. (1) Disease prevalence increases from north to south in the Caribbean region and is highly variable both spatially and temporarily. (2) Most virulent infectious diseases (white plague, yellow blotch, and white band) have a widespread distribution and are significantly impacting the ten most important reef-building species around their geographic distribution in the wider Caribbean, potentially affecting Caribbean coral reef resilience. (3) Most outbreaks occur during the warmest season of the year and produce significant loss of coral cover. (4) Different diseases affect their hosts differently over their geographic distribution. (5) Prevalence of colonies with multiple diseases/syndromes is increasing. (6) The newly described ciliate disease in the Caribbean (Croquer et al., 2006) is expanding geographically, infecting not only diseased colonies, but healthy colonies as well (Miller et al., 2006; Weil et al., 2006; Aldo Croquer and Ernesto Weil, University of Puerto Rico, *pers. comm.*, December 2006).

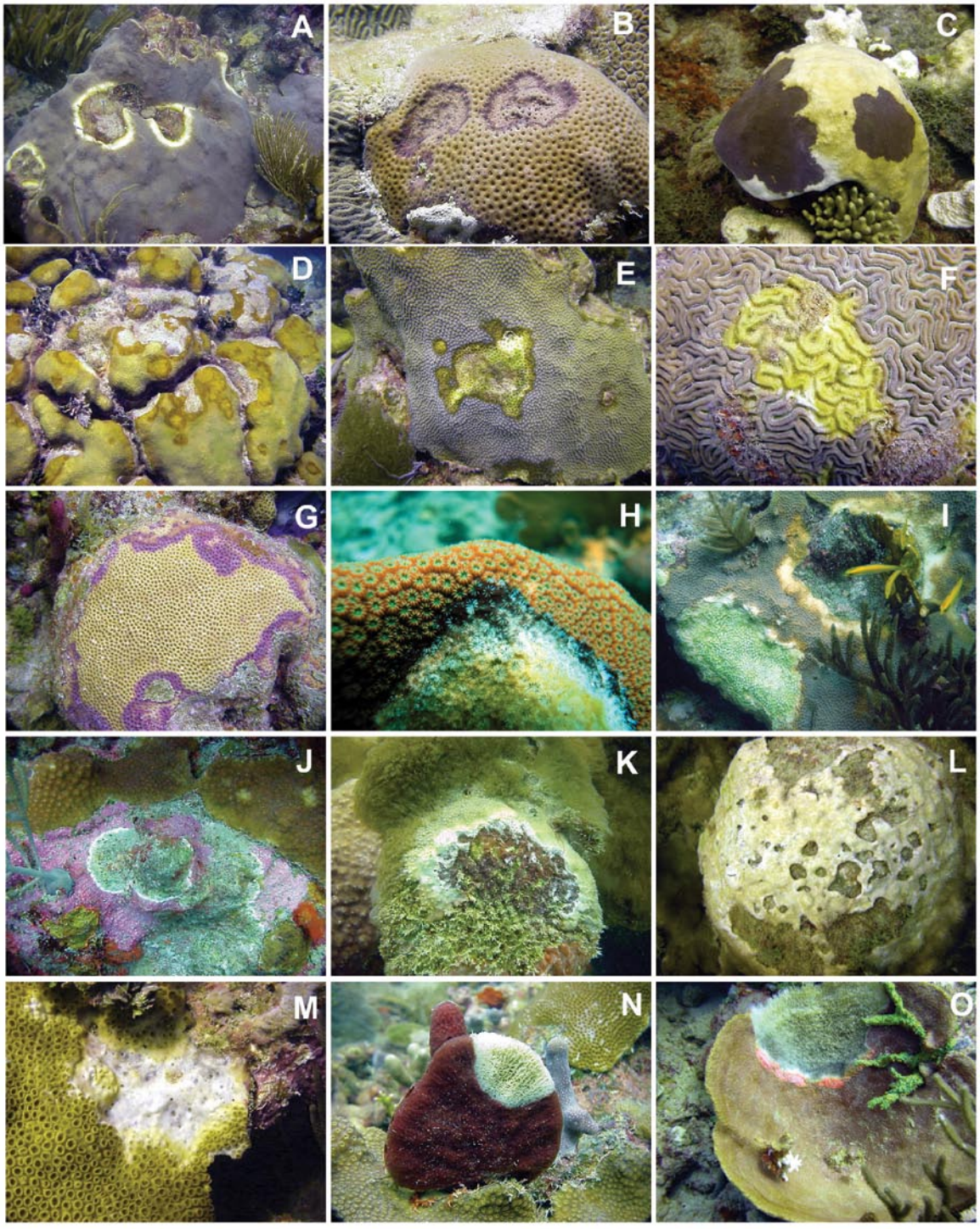

Figure 4. Common diseases affecting corals and other coral reef organisms in the Caribbean. (A) yellow blotch; (B and C) dark spots; (D) dark band; (E and F) necrotic tissue; (G) purple band; (H) dark line of ciliates (*Halofoliculina* sp) in *Montastraea*; (I) colony with multiple diseases; (J) Coralline white band in crustose alga; (K and L) necrotic tissue in crustose octocorals; (M) a zoanthid; and (N and O) sponges. *Photos by E. Weil*

Meso-America: Yucatán Coast

The Meso-American Reef consists of barrier-like reefs that border the Yucatán coastline from the Mexican Caribbean to the coast of Honduras. Using SCUBA, baseline surveys were conducted in 1985 using a standard sampling protocol. This same protocol was used to resurvey the sites in 2005. Comparison of total coral cover from these two surveys revealed a drastic decrease in coral coverage over the 20-year period, particularly in the reefs in the Biosphere Preserve (the Mahahual site—see Figure 5).

While the decline in coral cover may have different causes, the most likely are hurricanes and disease outbreaks. In the early 1980s, there were massive die-offs of acroporid (staghorn) corals due to white band disease and the sea urchin *Diadema antillarum* (Lessios et al., 1984). Then, in 1988, the northern Mexican Caribbean reefs (near Puerto Morelos) were severely impacted by Hurricane Gilbert (Class V), which destroyed most of the remaining *Acropora* stands. The central-section reefs of El Uvero and Akumal were damaged to a variable degree by Hurricane Roxanne (Class III) in 1995, but by then, most acroporids had died due to another outbreak of white band disease. The southern reefs of Mahahual and Xcalac were again affected by the sea swell generated by Hurricane Mitch (Class IV) in 1998. Recovery from the damage was patchy, but noticeable (Jordán-Dahlgren and Rodríguez-Martínez, 1998). By 2000, a variety of diseases were affecting many species, but only yellow blotch became an important disease on *Montastraea* hosts.

While the hurricanes may have had an impact on overall community structure, diseases seem to be the main factor in the declining cover of scleractinian corals in the surveyed areas. Data on diseases from ten sites clustered in the central Yucatán, adjacent to Akumal, reveal that the most abundant coral taxa (*Montastraea)* had the highest disease prevalence (Figures 6 and 7). In addition, some coral species are facing challenges from multiple pathogens. For example, white band disease severely reduced the extent of *Acropora palmata* coral cover,

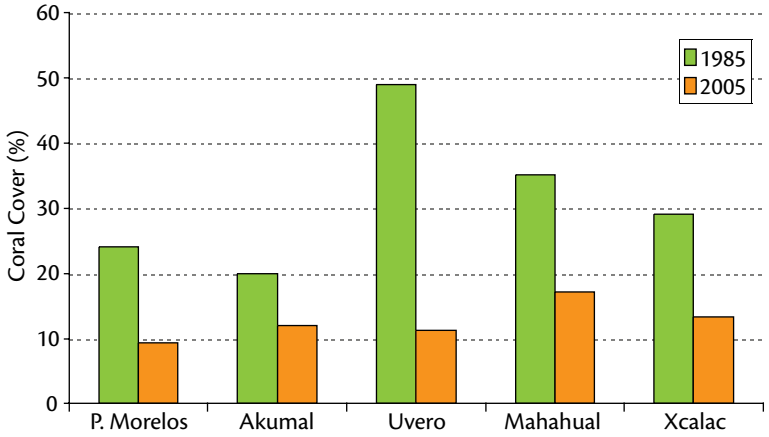

Figure 5. Coral cover surveys conducted in 1985 and 2005 on the Mexican Yucatán show a significant decrease in overall coral cover over the 20-year period.

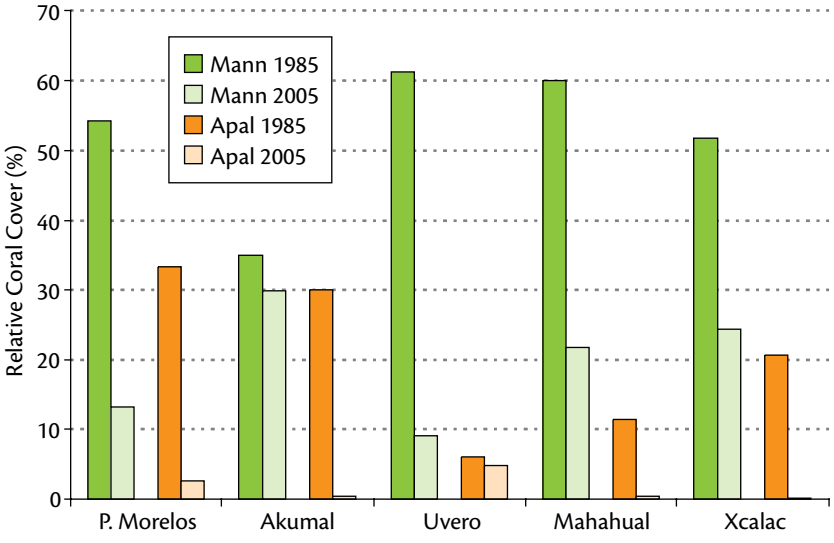

Figure 6. Coral cover surveys conducted in 1985 and 2005 on the Mexican Yucatán show a decrease in key reef building species. Mann = *Montastraea annularis* species complex. Apal = *Acropora palmata*.

but current declines of *A. cervicornis* may also be the result of this disease. Similarly, yellow blotch on *Montastraea* spp. has caused large mortalities (Jordán-Dahlgren et al., 2005), and this effect is now further enhanced by a white plague outbreak on the remaining colonies.

Although Figures 5 and 6 show moderate disease levels from 2005, surveys in 2006—after the hurricane and bleaching events of 2005—revealed an order of magnitude increase (Eric Jordan, Universidad Nacional Autónoma de México, *pers. comm.*, September 2006).

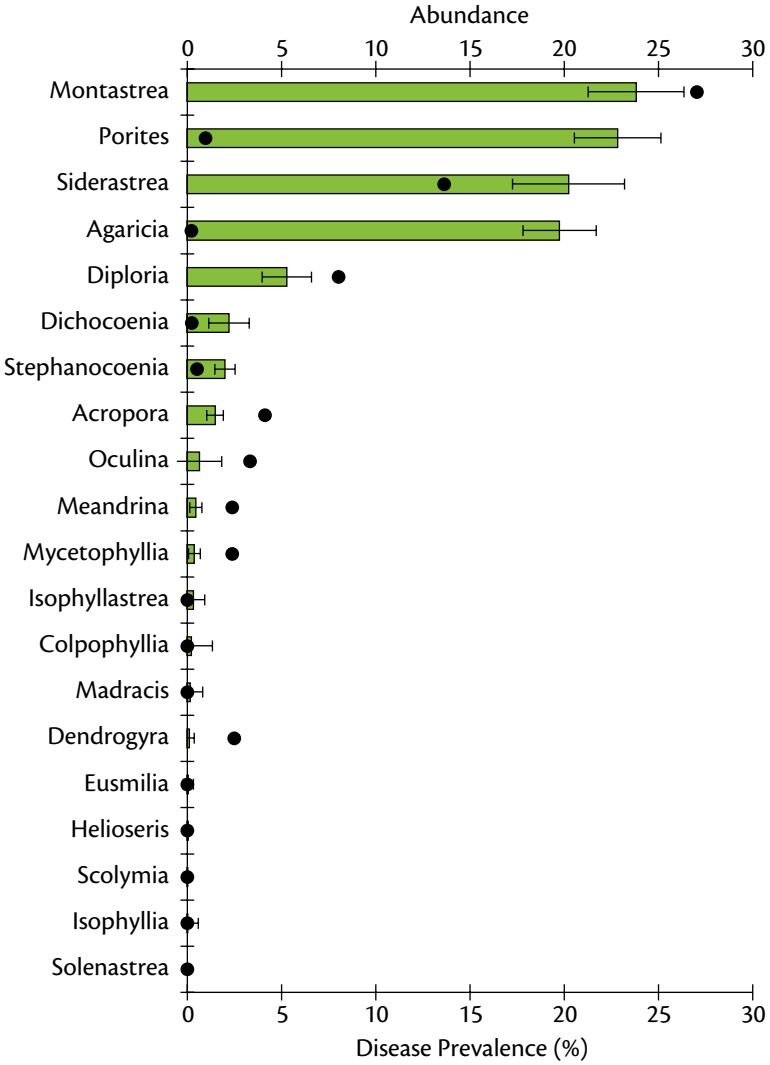

Figure 7. The abundance of major coral genera (bar) and prevalence of disease (circle) in the Yucatán (2004). Note that some of the most important reef-building coral also have the highest prevalence of disease. *Replotted from Ward et al. (2006)*

Australia: Great Barrier Reef

Until recently, it was assumed that disease has had little impact on the population dynamics and structure of coral assemblages on the Great Barrier Reef (GBR). The GBR is the largest coral reef system in the world, stretching 2300 km along Australia's northeast coast and comprised of more than 2800 reefs. Considered among the healthiest and most pristine, the majority of GBR reefs are located between 20 and 150 km offshore and adjacent to either unpopulated coastlines or generally low-density urban development. The entire GBR was accorded Marine Park status in 1975, and the area of highly protected zones elevated from 4.5 to 33 percent of the Marine Park in 2004. Prior to 2000, only two studies had focused on coral disease in the region, one on black band disease (Dinsdale, 2002) and the other on skeletal eroding band (Antonius, 1999; Antonius and Lipscomb, 2001). However, dramatic increases in the abundance of white syndrome (described below) on a number of reefs in 2002–2003 (Willis et al., 2004) heralded an increasing awareness of coral disease on the GBR.

Quantitative surveys between 2002 and 2006 revealed generally low (< 5 percent) disease prevalence on reefs surveyed in the northern, central, and southern regions of the GBR (Willis et al., 2004; Bette Willis and Cathie Page, James Cook University, *pers. comm.*, December 2006). The surveys sampled a range of habitats and reef types along north-south gradients more than 2000 km in length and cross-shelf (east-west) gradients ranging up to 100 km from the coast. Overall, seven disease types have been recorded: black band

disease, skeletal eroding band, white syndrome, brown band disease, growth anomalies, atramentous necrosis, and cyanobacterial syndromes (other than black band disease). Detection of some of the more common and infectious Caribbean diseases (black band disease and potentially some of the white diseases), in combination with discovery of diseases unique to the region such as brown band disease (Willis et al., 2004), suggest that coral diseases are common on Indo-Pacific reefs and may have a greater role in structuring coral communities in the region than previously thought.

All seven of the coral diseases detected are widespread throughout the GBR. Black band disease (Figure 8A) occurs on more than 70 percent of reefs surveyed (19 in all) and in all three sectors, although its prevalence is typically low (~ 0.1 percent of scleractinian corals) (Page et al., 2006). It has infected at least 32 coral species in 10 families on the GBR, with branching pocilloporid and acroporid corals being important hosts (Willis et al., 2004). Skeletal eroding band (Figure 8B), caused by the protozoan *Halofolliculina corallasia,* is the most common disease recorded in surveys. The ciliate erodes both coral tissue and skeleton as it produces a black test (Antonius, 1999); it affects at least 31 species in six coral families on the GBR (Willis et al., 2004). Although initially thought to be restricted to the Indo-Pacific, records of a new species of *Halofolliculina,* causing similar signs on corals from six families in the Caribbean (Croquer et al., 2006), now suggest that halofolliculinid infections are global in distribution.

Cases of white syndrome (Figure 8C) reached peak abundance in 2002–2003, concurrent with the most severe bleaching event so far recorded on the GBR, but have since declined to low levels in all regions (Bette Willis and Cathie Page, James Cook University, *pers. comm.*, December 2006). The twentyfold increase in white syndrome on some outer-shelf reefs in the northern and southern sectors between 1998 and 2003 suggests that the prevalence of white syndrome may be correlated with elevated temperatures, but possibly only when host densities are high (Selig et al., 2006). White syndrome is a collective term for conditions producing white signs associated with tissue loss on Indo-Pacific corals; it is used to describe progressive exposure of skeleton in white bands behind receding tissue fronts (Willis et al., 2004). The role of secondary pathogens, such as ciliates (see brown band disease description below) in escalating rates of tissue loss requires further investigation. White syndrome has been recorded to affect 17 coral species in four families on the GBR, with species of *Acropora* being important hosts (Willis et al., 2004).

Brown band disease (Figure 8D) is a new syndrome and has been recorded on corals in all three sectors of the GBR (Willis et al., 2004). The distinctive macroscopic field sign is a brown zone of variable width flanked by healthy tissue on one side and exposed white skeleton on the other as the band advances over the surface of the colony. Dense populations of an unidentified ciliate, packed with zooxanthellae from engulfed coral tissue, cause the brown coloration of the band. At high densities, the ciliates can cause rapid tissue loss. Brown band disease has been reported on 16 species from three families on the GBR, with acroporid corals being important hosts (Willis et al., 2004).

Growth anomalies (Figure 8E) have been found primarily on species of *Acropora* in disease surveys on the GBR, although they also affect species of *Montipora* and *Porites* (Willis et al., 2004). Reports of coral tumors growing on 18–24 percent of *Platygyra pini* and *P. sinensis*' populations on Magnetic Island in the central GBR (Loya et al., 1984) indicate that they have affected corals on the GBR for more than two decades and may be moderately prevalent on local scales. Atramentous

...innovative microbiological approaches to coral defense coupled with improved molecular diagnostics of pathogenic microorganisms and attempts to approach coral resistance with genomics tools, are emerging areas in the study of coral disease.

necrosis (Figure 8F) has primarily been recorded on a *Montipora* species at Magnetic Island (Jones et al., 2004), although it has also recently been recorded in disease surveys in the northern and southern regions of the GBR (Bette Willis and Cathie Page, James Cook University, *pers. comm.*, December 2006). Cyanobacterial syndromes (other than black band disease) (Figure 8G) affect a range of corals in at least five families, but acroporids and pocilloporids are primary hosts (Bette Willis and Cathie Page, James Cook University, *pers. comm.*, December 2006). A number of other macroscopic field signs are classified as indicators of compromised coral health, including pigmentation response (pink or purple tissue pigmentation adjacent to sites of competitive interactions and lesions), algal overgrowth (algal filaments growing directly on live coral tissue), and unusual bleaching patterns (e.g., distinct and unusual patches, spots, and stripes of bleached tissue that differ from typical patterns of whole colony bleaching or paling seen during thermal anomalies) (Willis et al., 2004).

Very little is known about pathogens or abiotic triggers associated with coral disease on the GBR. Of the seven disease types described, two are associated with cyanobacterial infections (black band disease, other cyanobacterial syndromes) and two with protozoan infections (skeletal eroding band, brown band disease). White syndrome and atramentous necrosis have been associated with *Vibrio* infections (Meir Sussman, James Cook University, and David Bourne, Australian Institute of Marine Science, *pers. comm.*, September 2006). Carbon-14 studies near boundaries of lesions on tabular

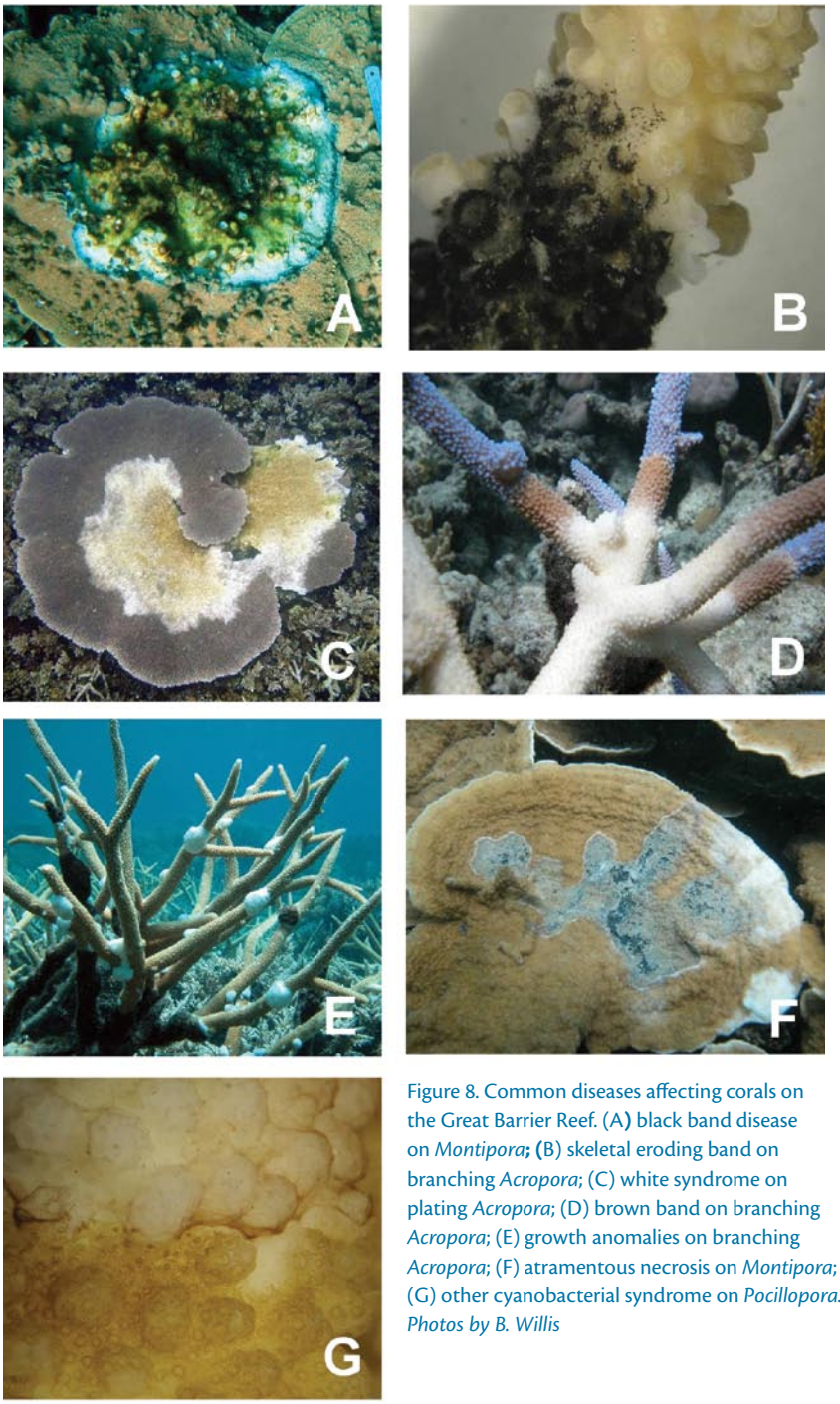

Figure 8. Common diseases affecting corals on the Great Barrier Reef. (A) black band disease on *Montipora*; (B) skeletal eroding band on branching *Acropora*; (C) white syndrome on plating *Acropora*; (D) brown band on branching *Acropora*; (E) growth anomalies on branching *Acropora*; (F) atramentous necrosis on *Montipora*; (G) other cyanobacterial syndrome on *Pocillopora*. *Photos by B. Willis*

Acropora colonies suggest that photoassimilates are preferentially translocated away from lesions in an apparent shutdown reaction, potentially as a result of abiotic factors or pathogens triggering an apoptotic pathway in the host (Roff et al., 2006). Clearly, further studies of coral pathogens would significantly enhance current understanding of coral diseases on the GBR and increase the potential for mitigating their impacts.

Philippines/Southeast Asia

Philippine coral reefs comprise the second largest reef area in Southeast Asia, covering an estimated 26,000 km^2. They are among the most diverse reefs known, with over 500 species of scleractinian corals recorded (Veron, 2000). However, these reefs are also among the most stressed in the world, facing multiple threats that include bleaching, overfishing, destructive fishing, siltation, and outright destruction for coastal development. Due to a human population growth rate of 2.3 percent per year, 98 percent of Philippine reefs are considered to be under medium-to-high risk from these anthropogenic impacts (Burke et al., 2002). In an attempt to address the rapid loss of these highly productive systems, the Philippine government has enacted a number of laws over the past decade to manage remaining reefs, largely through the establishment of Marine Protected Areas (MPAs). The end result of this legislation has been the designation of approximately 500 MPAs throughout the country, one of the largest MPA networks currently in place (Aliño et al., 2000). Therefore, the diverse Philippine reef system contains both highly impacted and well-managed reefs. Coral disease has recently been added to the list of stressors these reefs face. With the current effort to determine the nature of linkages between anthropogenic drivers and disease progression and infection rates, and the compelling evidence in support of such a link, it is likely that coral diseases will become a major source of mortality on many Philippine reefs already stressed.

Antonius (1985) was the first to record diseases affecting Philippine reefs. Black band disease, while found in eight different species of primarily faviids, was relatively uncommon. Almost 20 years later, combined surveys and monitoring efforts have revealed a number of diseases affecting a broad range of host species. Mean total disease prevalence, established from surveys of eight reefs in two regions in 2003, was 8 percent (Raymundo et al., 2005). The two consistently most prevalent diseases affecting Philippine reefs are *Porites* ulcerative white spot disease (PUWS) and a growth anomaly affecting massive *Porites* (Raymundo et al., 2003; Raymundo et al., 2005; Kaczmarsky, 2006) (Figure 9). PUWS affects at least 14 different species, with some localized areas having a prevalence of 72 percent of host species (Raymundo et al., 2005). Growth anomalies, likewise, show localized areas of prevalence as high as 39 percent of massive *Porites* (Kaczmarsky, 2006). Black band disease was recorded in very low prevalence in 2002–2003 (Kaczmarsky, 2006) and in 2006 (Laurie Raymundo, University of Guam, *pers. comm.*, December 2006). In addition, skeletal eroding band and white syndrome (as described above on the GBR) have been found on a variety of genera (Kaczmarksy, 2006). In 2006, coralline lethal orange disease, affecting crustose coralline algae, was also recorded for the first time in Philippine reefs, in low prevalence (Laurie Raymundo, University of Guam, *pers. comm.*, December 2006).

A putative causal agent has been determined for black band disease from Palau corals: an unidentified cyanobacterium identical to that associated with black band disease in Caribbean corals (Sussman et al., 2006). While it has not been determined if the Philippine black band disease infections contain the same cyanobacterial consortium, infected corals from Palau and the Philippines show strong similarities at both gross and microscopic levels. A causal agent for skeletal eroding band affecting GBR corals has also been determined (Table 1), though, again, it is not known if the ciliate causing skeletal eroding band in the Philippines is the same species. A putative causal agent for PUWS appears to be an undescribed species of *Vibrio*, which is currently under investigation. Many of these diseases appear to target the dominant reef-building genus *Porites* (Raymundo et al., 2005; Kaczmarsky, 2006). Preliminary data suggest a link between disease prevalence and proximity to human population centers (Kaczmarsky, 2006). These results suggest the potential for long-term impacts on reef communities.

East Africa

The corals reefs of East Africa range from the coast of South Africa to Somalia. Encompassing approximately 7000 km^2, these highly diverse reefs are home to over 300 coral species. There are four major reef systems: isolated

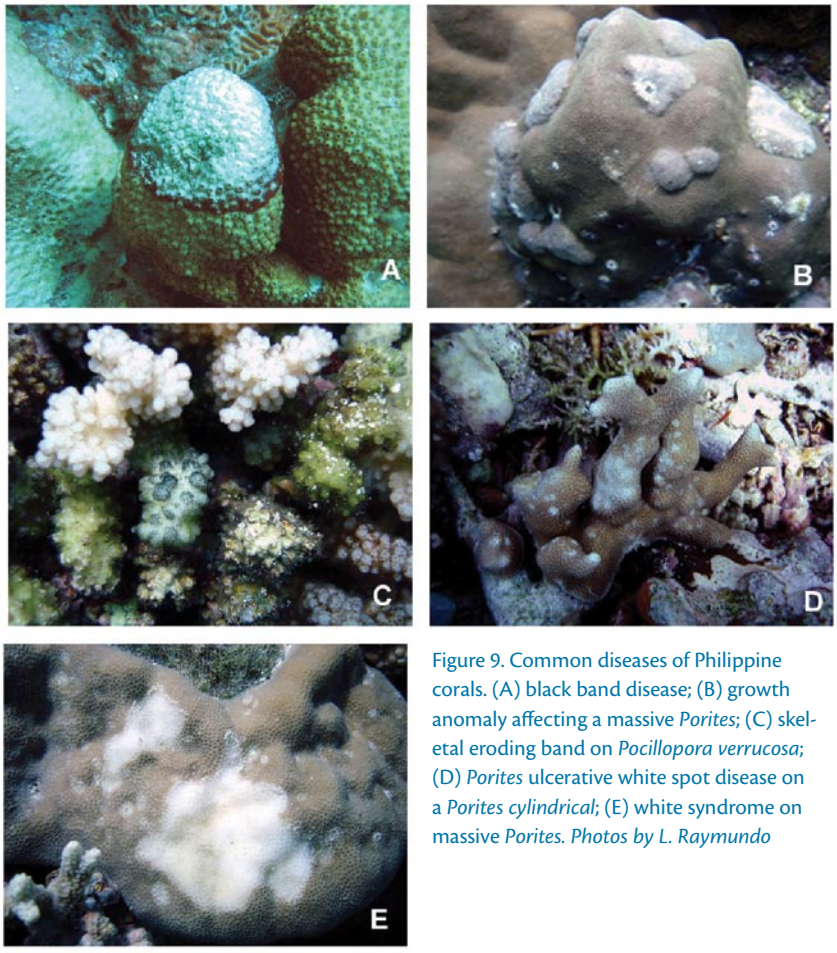

Figure 9. Common diseases of Philippine corals. (A) black band disease; (B) growth anomaly affecting a massive *Porites*; (C) skeletal eroding band on *Pocillopora verrucosa*; (D) *Porites* ulcerative white spot disease on a *Porites cylindrical*; (E) white syndrome on massive *Porites*. *Photos by L. Raymundo*

reefs along the coast of South Africa to Mozambique, a barrier and island reef system near Tanzania, fringe reefs off southern Kenya, and patchy reefs to the north (Obura et al., 2004).

There have been few studies on coral disease in this area; thus, infectious disease outbreaks have not been commonly reported. Bacteria-induced bleaching was found in Zanzibar (Ben-Haim and Rosenberg, 2002). Black band, white band, and yellow band diseases were reported in isolated outbreaks (McClanahan, 2004). More recently, a limited outbreak of a newly described white syndrome occurred off the Kenya coast. This outbreak, associated with an infection of fungal hyphae, almost eliminated *Montipora* from affected Kenyan reefs (McClanahan et al., 2004). Observations in Zanzibar and Kenya revealed low levels of PUWS, brown band, white syndromes, growth anomalies, and tissue necrosis affecting corals, octocorals, and sponges in several reef localities off the coast of Zanzibar and Kenya in 2005 (Figure 10; Ernesto Weil, University of Puerto Rico, *pers. comm.*, December 2006).

One of the most destructive forces on coral reefs in the western Indian Ocean has been coral bleaching. High water temperatures associated with the 1998 El Niño Southern Oscillation caused a widespread bleaching event that resulted in 50 percent mortality in some areas (McClanahan, 2004). Other serious but more regional bleaching events occurred in 2003 and 2005. Recent studies show that recovery has occurred in some reefs, while others suffered serious bio-erosion due to the destruction of the underlying reef framework (Obura, 2005).

While these reefs have historically been considered relatively isolated and pristine, rapid human population growth has been driving a decrease in water quality (due to nutrients and sedimentation), and an increase in destructive fishing methods (Obura, 2005). Working with local and international nongovernmental organizations, many countries have set up MPAs to try to alleviate and prevent reef destruction. The national coral reef task forces have implemented monitoring programs that observe general reef status and diversity (Obura, 2005). It is critical that these programs begin to look for coral disease and identify outbreaks in what has been a highly understudied area. To this end, we held a coral disease workshop in April 2006 at the Center of Excellence in Zanzibar for regional scientists and government personnel to train in microbiology and help foster local monitoring and reporting of any outbreaks in the region.

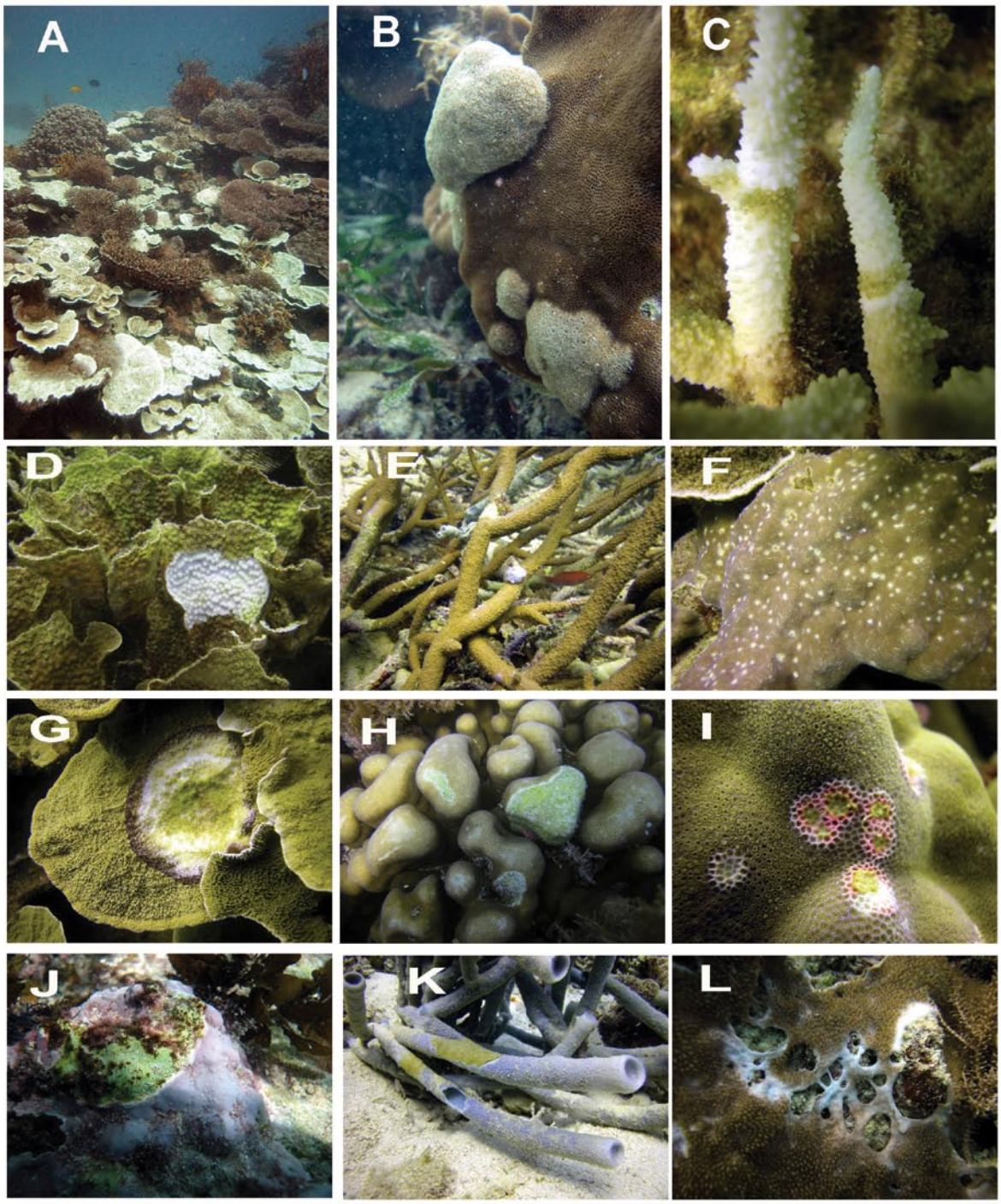

Figure 10. Most common diseases and syndromes in East Africa. White syndrome affecting *Montipora* (A) and *Echinopora* (D) colonies in Zanzibar. Growth anomaly (puffy syndrome) on massive *Porites* in Kenya (B) and on *Acropora* in Zanzibar (E). *Acropora* with brown band disease (ciliates) (C). *Porites* ulcerative white spots (PUWS) on massive *Porites* (F) and black band on *Montipora* (G) in Zanzibar. Compromised tissue responses in *Porites* (necrotic tissue) (H) and pigmentation response in *Porites* (I). Other important reef organisms affected included crustose coralline algae with white band-type syndrome (J), tube sponges with necrotic areas (K), and crustose octocorals with necrotic areas (L). *Photos by E. Weil*

2. ENVIRONMENTAL DRIVERS

Temperature

One of our research goals is to investigate the relationship between disease outbreaks and ocean-warming anomalies. Our hypothesis is that coral disease is enhanced by ocean warming. There is evidence for this relationship in the mass mortality of the gorgonian coral *Briareum asbestinum* following the 1998 El Niño event (Harvell et al., 2001). An increase in disease following warming events may occur because corals are less able to fight disease while under temperature stress, or because pathogens are more virulent at higher temperatures. In at least three cases *(Aspergillus sydowii, Vibrio shiloi,* and *Vibrio coralliilyticus*), pathogen growth and/or virulence factors increase to an optimal temperature (Israely et al., 2001; Banin et al., 2000; Alker et al., 2001; Ben-Haim et al, 2003a, b).

Seasonal patterns in disease prevalence in the northeastern Caribbean provide further support for a link between warming ocean waters and disease outbreaks. Recurrent outbreaks of the two most virulent and damaging diseases, white plague and yellow blotch, developed during the summer and fall seasons (highest water temperatures) of the past four years on Puerto Rican reefs (Ernesto Weil and Edwin Hernandez-Delgado, University of Puerto Rico, *pers. comm.*, December 2006) and in the US Virgin Islands (Miller et al., 2006; Rogers and Miller, 2006). Immediately following the peak of the 2005 bleaching event, the most devastating recorded in the northeastern Caribbean, outbreaks of white plague and yellow blotch were even more extensive in these areas.

On the GBR, seasonal patterns in coral disease show dramatic increases in prevalence between winter and summer surveys in all major families of coral (Willis et al., 2004). For example, disease increased fifteenfold in acroporids, twelvefold in faviids, and doubled in pocilloporids in summer surveys. Prevalence of three coral diseases increased significantly in summer surveys, with skeletal eroding band increasing more than twofold, black band and other cyanobacterial infections more than threefold, and white syndrome more than fiftyfold.

To investigate whether coral disease was correlated with warm-temperature anomalies, we used disease-prevalence surveys spanning 500 km of a latitudinal gradient along the GBR (Selig et al., 2006). In 1998, the Australian Institute of Marine Science's Long-Term Monitoring Program began to systematically monitor white syndrome, which affects more than 15 coral species, including the dominant plating acroporids. Using SCUBA, divers conducted annual coral disease surveys on 47 reefs from 1998 to 2004 to quantify the number of cases of white syndrome. Using a weekly 4-km data set of temperature values derived from the US National Oceanic and Atmospheric Administration Advanced Very High Resolution Radiometer (AVHRR) Pathfinder (a radiation-detection imager that can determine surface temperature), we detected a highly significant relationship between the frequency of warm temperature anomalies and the incidence of white syndrome, indicating a relationship between temperature and disease. Interestingly, this relationship also depends on a high degree of coral cover, as would be expected for transmission of an infectious agent (Bruno et al., in press).

Water Quality

Scientists generally agree that environmental stress can impact coral health. As human populations continue to increase, nutrients, terrigenous silt, pollutants, and even pathogens themselves can be released into nearshore benthic communities (Harvell et al., 2004). The detrimental effects of such inputs may vary between species and at different life-history stages within species, and may be affected by the nature and timing of delivery. Effects of environmental stress on development, growth, reproduction,

If habitat deterioration and climate warming continue at the same rates, we are faced with unprecedented challenges in managing coral reef communities.

and survival have been demonstrated in a variety of benthic nearshore taxa. And while the link between anthropogenic stress and disease susceptibility is currently poorly understood, our hypothesis is that coral disease is facilitated by a decrease in water quality, particularly due to eutrophication and sedimentation (Bruno et al., 2003).

Eutrophication poses a number of threats, including enhanced disease progression. Although corals are known to grow in high-nutrient water (Atkinson et al., 1995), recent evidence suggests a synergistic relationship between elevated nutrients and disease. High-nutrient levels (nitrogen and phosphorus) were associated with accelerated tissue loss in both yellow band- and *Aspergillosis*-infected corals in field manipulations (Bruno et al., 2003), and in black band-affected corals (Voss and Richardson, 2006), although high-nutrient levels alone were not associated with increased tissue loss in healthy corals. This observation is consistent with the findings of Kuntz et al. (2005) that there is rapid tissue sloughing in healthy corals exposed to elevated carbon sources, but little effect from elevated nitrogen and phosphorus. Thus, corals may seem to thrive under high-nutrient conditions, but the combination of an active infection and elevated nutrients increases disease-progression rates. It is unclear whether this effect is due to an impact on host resistance or a positive effect on pathogen growth or virulence.

Siltation offers yet another challenge to host disease resistance. The impacts of terrigenous sedimentation on nearshore communities are visible and well documented; corals inhabiting silted reefs are often observed to possess large patches of dead, exposed skeleton bordered by apparently receding margins of healthy tissue. While coral tissue mortality was previously assumed to be the result of direct smothering, microbial agents may also be implicated. Early work by Hodgson (1990) identified silt-associated bacteria as a possible cause for necrosis in sediment-damaged corals, as antibiotic-treated water reduced the amount of tissue damage in experimentally silted corals. More recently, opportunistic terrestrial pathogens (the soil fungus *Aspergillus sydowii* and the human enterobacterium *Serratia marcescens)* have been demonstrated as causal agents for two diseases currently impacting dominant corals in the Caribbean (Geiser et al., 1998; Patterson et al., 2002). Eolian transport of dust from an expanding Sahara desert has been hypothesized as a source of *Aspergillus* spores (Garrison et al., 2003), suggesting a mechanism that at least partially explains development of the Caribbean basin as a global disease "hotspot." Thus, terrigenous inputs may not only be a cause of physical stress for shallow, benthic organisms such as corals, but may also act as a pathogen reservoir.

This evidence suggests that anthropogenic stressors are linked with disease severity in complex ways. It is important to establish and quantify such linkages, as understanding these factors may make it possible to mitigate stressors via improved reef management and land-use practices. The challenge lies in demonstrating these linkages in the complex system of diverse stressors acting upon the coral holobiont.

3. PATHOGENS

Unfortunately, the identities of most coral pathogens are not known. The classic way to prove a particular microorganism causes disease is to prove Koch's postulates (see Figure 3). The five diseases for which the microbial cause has been established via Koch's Postulates include white band II (Ritchie and Smith, 1998), white plague type II (Richardson et al., 1998), aspergillosis (Smith et al., 1998; Nagelkerken et al., 1997; Geiser et al., 1998), white pox (Patterson et al., 2002), and bacterial bleaching of *Oculina patagonica* by *Vibrio shiloii* (Kushamaro et al., 1997) and of *P. damicornis* by *Vibrio coralliilyticus* (Ben Haim et al., 2003b). Some diseases seem to be caused by a single organism while others appear to be caused by complex consortia of microbes. For example, black band disease, found throughout the Caribbean and the Indo-Pacific, appears to contain at least 50 different bacterial types (Sekar et al., 2006).

This complex relationship of microbes that constitutes many coral diseases makes a definitive comparison of disease with similar symptoms difficult. Without knowing what to look for, it is extremely difficult to follow these pathogens through the environment to determine their reservoirs and modes of transmission. In addition, because diseases are often identified by their symptoms alone, there has been confusion over whether certain reported diseases were the same or different. Nevertheless, our knowledge of their pathology (isolation and identification of the pathogen), etiology (symptoms and relationships between the host and pathogen), and epizootiology (e.g., geographic distri-

CASE STUDY: BACTERIAL BLEACHING OF CORALS

On a global scale, coral bleaching is the most devastating coral disease. Coral bleaching is the disruption of the symbiosis between the coral animal and intracellular dinoflagellate algae, commonly known as zooxanthellae. As a result of the degeneration and/or expulsion of zooxanthellae from the coral host, the white skeleton becomes visible through the transparent coral tissue, giving the organism a "bleached" white appearance. Bleaching is fatal to the coral unless the symbiotic relationship is reestablished.

Studies over the last 20 years have indicated a correlation between "higher than normal" seawater temperature and coral bleaching (reviewed by Jokiel, 2004). The most widely accepted hypothesis to explain this correlation is that photo-inhibition and damage to the photosynthetic apparatus of the zooxanthellae cannot be repaired at elevated temperatures (reviewed by Stambler and Dubinsky, 2005). Studies showing that some bacterial pathogens become more virulent at higher temperatures (Rosenberg and Falkovitz 2004) raise questions about the potential contribution of bacterial diseases to mass bleaching events.

Two cases of bacterial bleaching of corals have been well documented: bleaching of *Oculina patagonica* in the Mediterranean Sea by *Vibrio shiloi* (Kushmaro et al., 1996, 1997) and bleaching of *Pocillopora damicornis* in the Indian Ocean and Red Sea by *Vibrio coralliilyticus* (Ben Haim et al., 2003 a, b). The *V. shiloi/O. patagonica* system has been studied in considerable detail. The bacterium shows chemotaxis to its coral host (Banin et al., 2001a) and then binds to a β-galactoside receptor in the coral mucus (Toren et al., 1998). It then penetrates into the epidermal layer of the coral (Banin et al. 2000), where it multiplies intracellularly to cell densities of over 10^8 cells per cm^3. *Vibrio shiloi* produces a proline-rich peptide called Toxin P, which causes a rapid decrease in the photosynthetic quantum yield of zooxanthellae (Banin et al., 2001b). Several of the virulence factors essential for a successful infection of *O. patagonica* by *V. shiloi* are synthesized at elevated summer seawater temperatures. These factors include (1) a protein on the bacterial cell surface that recognizes a receptor in the coral mucus (Toren et al., 1998; Banin et al., 2001a); (2) superoxide dismutase, which allows the bacteria to survive in the oxygen-rich coral tissue (Banin et al., 2003); (3) Toxin P, which binds to zooxanthellae membranes and inhibits photosynthesis (Banin et al., 2001b); and (4) enzymes that lyse zooxanthellae (Ben-Haim et al., 1999).

Knowledge of reservoirs and modes of transmission has proven useful in the past for developing technologies for controlling the spread of disease. Using fluorescence in situ hybridization with a *V. shiloi*-specific deoxyoligonucleotide probe, it was found that the marine fireworm *Hermodice carunculata* is a winter reservoir for *V. shiloi* (Sussman et al., 2003). Worms taken directly from the sea during the winter contained approximately 10^8 *V. shiloi* per worm. Worms carrying the pathogen could serve as vectors for transmission of the disease, as they feed on coral tissue during the summer.

How general is bacterial bleaching of corals? Several investigators have reported the patchy spatial distribution and spreading nature of coral bleaching (e.g., Jokiel and Coles, 1990; Edmunds, 1994). Patchy distribution and spreading are highly symptomatic of infectious disease. Clearly, more microbiological research is necessary during a mass-bleaching event to test the bacterial hypothesis of coral bleaching.

butions, environmental factors, host ranges, prevalence, vectors, reservoirs, and spatial and temporal variability) is limited. Disease reservoirs have only been identified for black band disease (biofilms in reef sediments were found to contain non-pathogenic aggregates of the black band community) (Carlton and Richardson, 1995), and possibly for aspergillosis (atmospheric African dust has been suggested to contains spores of the fungus *Aspergillus sydowii*) (Shinn et al., 2000). The only coral-disease vectors identified are the fireworm *Hermodice carunculata*, whose gut has been found to harbor *Vibrio shiloi* (the pathogen inducing bacterial bleaching in a Mediterranean coral) (Sussman et al., 2003), and damselfish, which harbor one life-history stage of a digenean (trematode) that infects *Porites* (Aeby and Santavy, 2006).

4. DISEASE RESISTANCE

Microbial Surface Mucous Layers: A Barrier to Disease

While all corals secrete a layer of mucus over their surface (SML), we do not understand much about its production, composition, or function within the holobiont. Most of the carbon that makes up the SML originates from the symbiotic zooxanthellae (Patton et al., 1977), but is secreted by coral epidermal mucus cells as an insoluble, hydrated, glycoprotein that forms a gel-like layer over the coral surface (Ducklow and Mitchell 1979; Meikle et al., 1988). The thickness of the SML can vary from less than one millimeter in some scleractinians, to a few centimeters in some gorgonians. The chemical composition of the SML from different coral species varies qualitatively and quantitatively (Meikle et al., 1988). It is not known if the normal differences in the chemical composition of the SML are due to variations in the zooxanthellae or variations in the metabolism of the coral host.

In contrast to the relatively nutrient-poor environment of the open water, the coral SML has a high concentration of organic compounds. As such, it hosts a dense, complex community of microorganisms that differ significantly from the microorganisms present in the open water. A number of spatial models have been proposed for the SML and the microbial communities living there (see Brown and Bythell, 2005). One model suggests that the spatial stratification of various organic and inorganic nutrients within the mucous layer results in the development of a specialized symbiotic microbial community (Figure 11), not unlike those found in microbial mats (Ritchie and Smith, 2004). It appears that certain bacteria may be characteristic of specific coral species (Rowher and Kelly, 2004). Although microbial communities may vary from coral species to coral species, their metabolic activities are likely to be similar.

Just as the normal microbial flora of humans protects us from infection, it is likely that the normal microbiota associated with the surface layer of corals protects the coral from invading microbes. Ritchie (2006) found that mucus from a healthy coral was able to inhibit the growth of other bacteria by tenfold. In addition, the competition of the normal

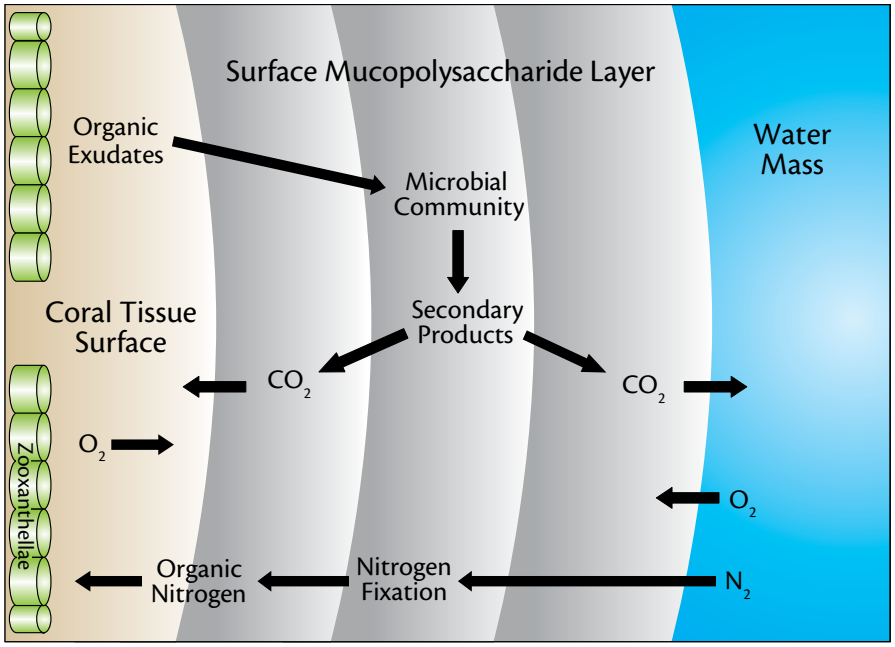

Figure 11. This model of coral surface mucopolysaccharide layer shows the movement of nutrients through the layer of slime that coats the surface of coral. Organic carbon from the zooxanthellae help feed the complex community of microorganisms within the slime layer. These microbes most likely provide a critical layer of protection for corals against infection.

flora for nutrients could prevent other potential pathogens from becoming established. Changes in the carbon and nitrogen pools (due to changes in the normal coral holobiont physiology, i.e., disease or stress) could result in changes in the SML microbial communities.

We are investigating the hypothesis that environmental factors can alter the SML microbial community. Studies show that shifts in heterotrophic microbial populations within the SML occur when corals are stressed, either due to disease or during bleaching (Ritchie and Smith, 1995a, b; Frias-Lopez et al., 2002; Koren and Rosenberg, 2006; Gil-Agudelo et al., 2006). Because qualitative changes have been reported in coral mucus during bleaching, the change in community may be a response to changes in available carbon sources. It appears that as corals recover from bleaching, their specific microbial populations also recover. McGrath and Smith (1999) showed that *Vibrio* sp. populations tend to increase during bleaching but return to previous levels during recovery, while populations of *Pseudomonas* sp. decrease during bleaching, but also return to previous levels during recovery.

As the microbial communities change, so do their physiological functions, including the production of anti-microbial compounds and the establishment of co-metabolic relationships, both among the microbes and between zooxanthellae and coral animals. Ritchie (2006) recently showed loss of antibiotic activity from coral mucus of *Acropora palmata* during a prolonged bleaching event. Thus, changes in the normal microbial communities may ultimately result in the development of disease.

Coral Immunity and the Effects of Environmental Stress

While lab studies of model organisms such as *Drosophila* provide a basis for understanding invertebrate innate (non-specific) immunity, we have little understanding about nonmodel organisms like corals and about the interactions between host immunity and pathogenesis in nature. Our work (Harvell) is focused on understanding the primary elements of immunity in a gorgonian coral-fungal pathosystem. A primary line of gorgonian defense against pathogens is the circulating amoebocytes, which encapsulate invaders (Mullen et al., 2004; Mydlarz et al., 2006) and are induced in large numbers during infections (Laura Mydlarz, University of Texas, *pers. comm.*, December 2006). In addition, prophenoloxidase is activated to catalyze melanin deposition, as well as other downstream reactions (Mydlarz et al., 2006). In infected gorgonians, melanin builds up as a barrier to advancing fungal hyphae, thus preventing its spread (Petes et al., 2003). This melanin buildup results in visible dark purple halos that are often associated with fungal infections like *Aspergillus*. Other fast-acting enzymes such as peroxidase (Mydlarz and Harvell, 2006) and chitinases (Douglas et al., 2006) play a role in defense.

In addition, corals make more slowly developed anti-microbial chemicals (Mullen et al., 2004; Geffen and Rosenberg, 2005), which have been detected in a number of gorgonian cell extracts (Kim et al., 2000a). Antimicrobials are also produced by SML-associated microorganisms, as some specific anti-microbial agents have been identified as products of resident bacteria in the mucus (Ritchie, 2006). Thus, we continue to embrace a holistic approach to resistance of the holobiont, while at the same time working through the various mechanistic elements to come to an even rudimentary understanding of how corals resist pathogenic infections.

> We are still far away from any miracle "vaccine" or remediation protocol against any of the current coral reef diseases.

There is evidence that heat-stressed corals are more susceptible to disease, although it is not clear whether warmer temperatures inhibit coral defenses by altering the immune response or because of bleaching, or whether temperature enhances the virulence of pathogens. Efforts to unveil this link between temperature stress and coral disease require careful experimentation with host immune responses and with pathogen virulence and infectivity. Pathogen response to increased temperatures may be a key element in the dynamics of coral diseases. For example, *Aspergillus sydowii,* the fungal pathogen of the sea fan dis-

ease aspergillosis, grows at a faster rate at higher temperatures (Alker et al., 2001), and the bacterial pathogen of hard corals, *Vibrio coralliilyticus* (Ben-Haim et al., 2003a), produces more lytic proteins when grown at elevated temperatures, which increases its virulence. Adhesion ability, a critical virulence factor in the causative agent in coral bleaching (*Vibrio shiloi*) is also temperature-sensitive (Toren et al., 1998). In addition to adhesion, production of anti-algal toxins and superoxide dismutase (which detoxifies oxygen radicals) are also temperature-dependent virulence factors that seem to be induced in *V. shiloi* by elevated seawater temperatures (Banin et al., 2003).

DISCUSSION AND CONCLUSION

After 20 years of research, we are still unable to explain the source or sudden emergence of the majority of disease syndromes in coral reefs. Warm-temperature anomalies may facilitate the emergence and spread of pathogens or spread of other stressful agents that could affect the natural resistance (i.e., the "physiological equilibrium" between coral hosts and their natural flora), or could stimulate other bacteria living in reef sediments into becoming virulent. Very little is known about the composition and dynamics of the natural microbial communities living in association with most corals, but recent findings reveal an impressive diversity of microbial communities. They range from single fungal or bacterial species to loosely or tightly structured bacterial consortia that include a wide variety of phototrophic and heterotrophic bacterial species with a wide range of metabolic modes and micro-niche characteristics (Koren and Rosenberg, 2006; Rohwer et al., 2001; Richardson et al., 2001; Kellog, 2004).

Recent research also shows that some of these invertebrates can actively respond to the infections. Recent research summarized in coral immunity shows the dynamic of an active immune response to microbial infections. More focus on understanding active mechanisms of holobiont resistance, both in the SML and in tissue of the coral, may suggest approaches to buffering immunity. The complex, symbiotic nature of the coral holobiont offers one of the greatest challenges in invertebrate immunity, requiring an unraveling of the roles of SML, zooxanthellae, and coral tissue in orchestrating defenses against microbes. New advances in enhancing coral immunity are also emerging through the designing of microbial defense systems, such as phage therapy. Phage therapy of corals was shown by isolating from nature phage viruses that consume pathogenic bacteria and resulted in non-diseased corals (Efrony et al., 2006). These innovative microbiological approaches to coral defense, coupled with improved molecular diagnostics of pathogenic microorganisms and attempts to approach coral resistance with genomics tools, are emerging areas in the study of coral disease.

If habitat deterioration and climate warming continue at the same rates, we are faced with unprecedented challenges in managing coral reef communities. We are still far away from any miracle "vaccine" or remediation protocol against any of the current coral reef diseases. Terrestrial disease managers use tools that include quarantine, culling, and vaccination, which are not practical in ocean systems. The fact that other keystone members of the reef community are also being affected by new syndromes complicates the picture even more. Marine pathogens can move faster and for longer distances than ever before due to human activities such as commercial and military shipping and the transport of marine species for aquaculture and the aquarium trade (McCallum et al., 2003). One major question is whether our current management tool, the establishment of MPAs, increases resilience of coral reef ecosystems to regional-scale, water-borne pathogens such as the ones that have caused mass mortalities in the

The complex symbiotic nature of the coral holobiont offers one of the greatest challenges in invertebrate immunity, requiring an unraveling of the roles of SML, zooxanthellae, and coral tissue in orchestrating defenses against microbes.

Caribbean (McCallum et al., 2004). Currently, the only viable management option is to trace the origin of coral disease and attempt to shut off any known inputs. It is unrealistic to think that we can restore a 1000-year-old coral reef without restoring the original environmental conditions. Without a concerted effort among researchers, governments, and all stakeholders, the future of tropical coral communities is in jeopardy.

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