

# **Coral Disease, Environmental Drivers, and the Balance Between Coral and Microbial Associates (Summary adapted from full article published in *Oceanography*, Vol.20, No 1, March 2007)**

## **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 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 (<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.

## **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 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;

Recent research also shows that some of these invertebrates can actively respond to the infections and that coral shows 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 unravelling of the roles of SML, zooxanthellae, and coral tissue in orchestrating defences against microbes. New advances in enhancing coral immunity are also emerging through the designing of microbial defence 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 defence, 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 Marine Protected Areas, increases resilience of coral reef ecosystems to regional-scale, water-borne pathogens such as the ones that have caused mass mortalities in the 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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