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MICROBIAL PESTS: CORAL DISEASE IN THE WESTERN ATLANTIC

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ABSTRACT

Diseases of scleractinian corals have increased significantly over the last decade, affecting greater numbers of species around the world. Gross signs of coral disease are often observed as tissue loss on the skeleton, making differential diagnosis difficult. Using histopathological and ultrastructural techniques, coupled with microbiological analyses, the importance of microorganisms as pathogens in coral diseases is becoming more apparent. This paper addresses the ecology of pathogens on reefs, specifically bacteria and cyanobacteria that produce disease in scleractinian and alcyonarian corals. We review the nature of disease and the influence of adverse environmental conditions. An update is presented on research concerning the bacteria associated with black- and white-band diseases; observations are presented concerning other coral diseases in the western Atlantic that appear to be caused by bacteria. We conclude with suggestions for improving the recognition of coral diseases that include approaches for conducting research to identify bacterial pathogens and the role of environmental factors in the development of coral disease.

INTRODUCTION

Unprecedented decreases of cover by stony corals, altering both total abundance and species richness, have been documented during the last decade (Brown 1987; Ginsberg 1993; Hughes 1994; Sebens 1994). Increases have been reported in the incidence of physiological disorders, bleaching (loss of obligate symbiotic algae and/or algal pigments), and other diseases that cause loss of tissue. Accounts of declining reef quality from geographically-distant regions include the Florida Keys (Porter and Meier 1992, Zorpette 1995), Fiji (Littler and Littler 1996), Hawaii (Hunter and Evans 1995), the Red Sea (Antonius 1988), and the Great Barrier Reef (Dinsdale 1994; Glazebrook and Streiner 1994). These disorders have been observed primarily among the scleractinian corals, but other organisms in the reef ecosystem have been affected (Lessios et al. 1983; Hallock et al. 1993; Peters 1993).

Although diseases of corals have been reported from around the world, they have been documented most widely in the tropical western Atlantic (Peters 1993). Black-band and white-band diseases have been reported most frequently in this region, since the first observations were made in the early 1970s (Antonius 1977; Gladfelter et al. 1977). Subsequently, other maladies have been observed, including yellow-blotch disease on stony corals and red-band disease on octocorals. Most "band" diseases have been associated with microorganisms that appear to have a role in the development of lesions and the loss of coral tissue. Most coral diseases observed on reefs have not been thoroughly described. Their etiologies, including identification of the causative agent, pathogenesis, hosts affected, and interactions with anthropogenic stressors, are unknown. Knowledge of coral disease mechanisms, processes, and manifestation, and the role of microbial "pests" in development of these diseases might help to explain the striking increase in reef destruction.

Disease is any impairment of an organism's vital functions or systems, including interruption, cessation, proliferation, or other malfunction. Each organism has optimized behavioral responses, physiological status, and biochemical conditions that allow the constant regulation of its internal environment. The regulation of these responses allows an organism to maintain its health during exposure to stressors, including pathogens. As the intensity of a stressor or number of stressors to which the host is exposed increases, energy usage increases to counteract the impact, at the expense of growth and reproduction. When the organism can no longer maintain critical physiological and biochemical functions or reverse the condition, it succumbs to disease. The organism's competence to resist disease can be altered by environmental conditions, physiological status, nutrition, developmental stage, and genetic factors. Although the causal agent of a disease can be biotic or abiotic, both are closely related in disease manifestation; thus, most diseases are not caused by a single factor, and determining the primary cause can be difficult.

Biotic diseases are produced by parasites or pathogens. Potential infectious agents of corals include bacteria, viruses, protozoans, fungi, and macroparasites such as, helminths and arthropods. To date, no coral diseases are known to be caused by viruses. Abiotic diseases are structural and functional impairments to the organism that result from exposure to extreme physical and chemical conditions. Physical stress can be induced by changes in salinity, temperature, light intensity, radiation, sedimentation, oxygen, and water flow, as well as by direct injury to coral tissues. Abiotic diseases are noninfectious and cannot be spread by contact with an affected individual. Abiotic stressors can lead to greater susceptibility to disease by parasites and pathogens, and made more severe by the invasion of secondary pathogens. Secondary or opportunistic pathogens cause disease in a host whose defense mechanisms are compromised and who normally would not be infected by these microorganisms.

It is increasingly evident that abiotic factors might influence the distribution and impact of biotic diseases in corals. Anthropogenic inputs enrich nutrients, increase sedimentation, and add pesticides and other toxic chemicals, enteric bacteria and viruses to coastal waters (Sindermann 1995). Additionally, agricultural and industrial contaminants are released into watersheds and discharged into the sea (McIvor et al. 1994). The discharge of these materials in the vicinity of reefs represent inputs of biotic and abiotic stressors to which corals might be exposed with the potential to overburden the reef ecosystem. Increased landscape modification, sewage outfalls, and injection well sites used for wastewater combine to facilitate the dissolution of carbonate sediments on offshore reefs, which might allow percolation of elevated nutrient loads in these areas (Tomascik and Sander 1987; Shinn 1993; Porter et al. 1994; Szmant and Forrester 1996). Whether these areas of enriched water correspond to areas of increased disease activity on corals has not been thoroughly investigated. It is recognized, however, that enriched nutrients, increased water temperatures, and exposure to high levels of UV radiation or toxicants cause mortality of corals under experimental conditions (Dustan and Halas 1987; Glynn *et al.* 1989; Glynn and D'Croz 1990; Williams and Bunkley-Williams 1990).

The first line of defense of the coral against biotic and abiotic materials is the production of mucus. Mucus enables coral to shed sediments, invading microorganisms, and other irritants from its surface. Many bacteria colonize the surface of coral and feed on the mucus in the absence of disease, representing a complex balance between a host and its commensal bacterial flora under normal conditions (Ducklow and Mitchell 1979; Pascal and Vacelet 1981; Paul et al. 1986; Vacelet and Thomassin 1991). The community of commensal bacteria associated with mucus varies depending on the species of coral and physiological status of the host (Ducklow and Mitchell 1979; Pascal and Vacelet 1981; Coffroth 1990; Santavy et al. 1992; Ritchie and Smith 1995a, 1995b). When mucus production increases after exposure to irritants, bacterial numbers also increase (Mitchell and Chet 1975; Rublee et al. 1980; Segel and Ducklow 1982). With a physical or environmental change, the equilibrium between the coral and its commensal bacteria in the mucus can be disturbed, causing substantive changes in the species composition of these bacteria (Santavy, 1995). For example, the ubiquitous marine bacterium, *Vibrio alginolyticus* outcompeted and dominated the natural bacterial communities in the mucus of stressed corals relative to bacterial communities found in the mucus of nonstressed corals (Ducklow and Mitchell, 1979). Acute coral mortality has been observed after excessive mucus production, leading to subsequent bacterial growth and tissue invasion following exposure of corals to stressors (Mitchell and Chet 1975; Knap et al. 1985).

The effects of temperature, dissolved organic carbon, and other factors, on the growth of bacteria in the mucus have the potential to increase their numbers and pathogenicity on reef corals. New species of bacteria dominating the mucus might stress the host, and induce greater susceptibility to diseases. Exposure of corals to oil induced tissue swelling, copious mucus production, bleaching, and

tissue loss resulting from bacterial infections and undescribed coral tissue loss (Loya and Rinkevich 1980; Segel and Ducklow 1982; Jackson et al. 1989). Field and laboratory studies have provided evidence that some bleaching in one species of coral is induced by a bacterium classified as a *Vibrio* (Kushmaro et al. 1996). Some bacteria can infect and degrade coral cells, causing more damage than exposure to the stressor alone (Mitchell and Chet 1975; Hodgson 1990). Hodgson (1990) speculated that physiological resistance to bacterial infection might be "the most important determinant of sedimentation tolerance of reef corals". Comprehensive studies to test these potential synergistic causes and effects in disease manifestation between biotic pathogens and abiotic stressors could significantly advance our understanding of cause and effect relationships.

BACTERIAL DISEASES OF WESTERN ATLANTIC CORALS

Significant coral mortality has been reported around the world from black-band disease, white-band disease, and red-band disease, which have been associated with cyanobacteria and bacterial infections. Microbiological and molecular techniques are being applied to increase our understanding of the role of microbial "pests" in the development of these diseases. Additional observations on western Atlantic reefs suggest that microorganisms are important contributors to other types of coral tissue loss.

Black-Band Disease

Black-band disease (BBD) was the first disease reported to affect scleractinian corals. It was discovered in the 1970s on reefs off Belize, Bermuda, and the Florida Keys (Antonius 1973; Garrett and Ducklow 1975) and found throughout the western Atlantic and Caribbean basin in the 1980s (Peters 1993). Subsequently, cases of BBD have been reported off the Phillippines (Antonius 1985), in the Red Sea (Antonius 1988), off Fiji (Littler and Littler 1996), and on the Great Barrier Reef (Dinsdale 1994; Glazebrook and Streiner 1994; Miller 1996). The diseased tissue interface appears as a black band, a few mm to a few cm in width, with a clean, tissue-depleted skeleton next to apparently healthy tissue (Fig. 1). BBD affects primarily faviid corals in the Western Atlantic and is resisted by most other Scleractinia including the acroporids (Rützler et al. 1983). Recently, infections on acroporids on the Great Barrier Reef were reported (Miller 1996). This disease has also been documented on milleporid hydrocorals, gorgonians, and other families of Scleractinia (Rützler et al. 1983; Antonius 1985,1988; Feingold 1988; Glazebrook and Streiner 1994; Miller 1996). The disease proceeds at a rapid rate, with the band destroying several cm^2 tissue/day (Rützler et al. 1983).

It is unknown how the disease is originally established, although corals with physical injury or sloughing tissue appear most susceptible (Antonius 1985). BBD is usually found only on scattered coral colonies on reefs (Garrett and Ducklow 1975; Edmunds 1991), but extensive outbreaks also have been reported in the Caribbean (Peters 1993). A clumped distribution has been found in the Florida Keys, suggesting the disease is infectious (Kuta and Richardson 1994). Several factors appear to promote BBD infections; they include increased temperatures, nutrient loading, sedimentation, and water current patterns (Antonius 1985; Peters 1993; Littler and Littler 1996; Bruckner et al. in press).

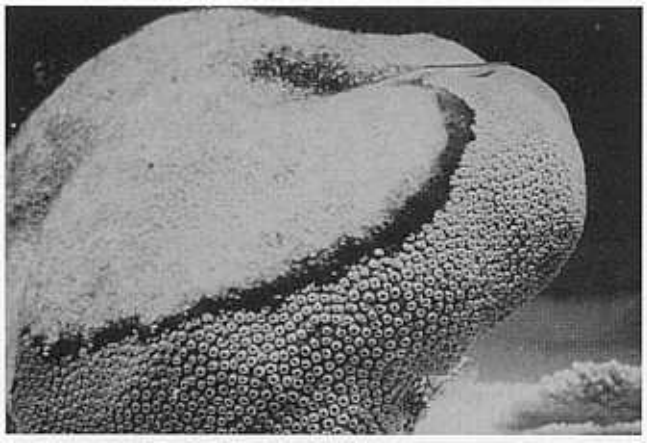

Fig. 1: Black-band disease affecting *Diplori a strigosa*.

The black band is composed of a consortium of microorganisms including the cyanobacteria *Phormidium corallyticum* and *Spirulina* sp., sulfate-reducing bacteria, the sulfur-oxidizing bacterium *Beggiatoa*, ciliates, and heterotrophic bacteria (Rützler and Santavy 1983). The dominant organism in BBD is the cyanobacterium *P. corallyticum* (Rützler and Santavy 1983) which forms a miniature microbial algal mat. Examinations of the mat using oxygen and sulfide microelectrodes have demonstrated very steep oxygen and sulfide gradients. The gradients correspond to the vertical migration of the cyanobacterial filaments in the band following a diurnal cycle related to oxygen production by photosynthesis (Carlton and Richardson 1995). The presence of H_2S in the anoxic zone probably induces necrosis of the coral tissue. The bacteria consume organic compounds released as the cells lyse, leaving behind bare skeleton. The compounds released from the necrotic tissue could enable heterotrophic growth of *P. corallyticum* and the microbial community (Taylor 1983).

Previously, *P. corallyticum* had been found associated only with the coral disease, never reported in the free-living form. It has a very distinct trichome morphology associated with the disease and in culture, allowing easy distinction from other filamentous cyanobacteria. Direct sequence comparisons of the 16S rRNA have verified *P. corallyticum* to be a distinct species (Santavy et al. 1995b). Species-specific oligonucleotide probes were used to examine diseased corals and environmental samples from adjacent areas in the Bahamas, Puerto Rico, and the Florida Keys. A very large black algal mat was found in the Bahamas overlaying the sand; closer examination confirmed the characteristic trichome morphology of *P. corallyticum*. Oligonucleotide probing using a fluorescence *in situ* hybridization technique confirmed the cyanobacterial filaments to be *P. corallyticum* (Santavy et al. 1996). Further studies will increase our knowledge about distribution of this cyanobacterium in the noninfectious state. Eventually, other samples from distant locations can be probed to determine whether *P. corallyticum* is associated with BBD worldwide or whether other cyanobacterial species can colonize compromised corals and produce BBD.

Red-Band Disease

Cyanobacteria have also been associated with diseases of the Octocorallia, especially the gorgonians. Red-band disease (RBD) was first noticed as a variant of BBD infecting *Gorgonia ventalina* near Carrie Bow Cay, Belize (Rützler et al. 1983). The term RBD was used to describe a brick red cyanobacterial mat resembling BBD on scleractinian corals from the Bahamas (Richardson 1993). The disease consists of a reddish-brown to brown-black band about one cm wide separating healthy gorgonian tissue from a dead portion of the axial skeleton (Fig. 2). RBD has been observed on the west coast of Puerto Rico infecting *Gorgonia ventalina* (A. Bruckner, pers. comm.) and in the Florida Keys. Video transect data have indicated a disease resembling RBD infecting many sea fan colonies near Key West (C. Quirolo, pers. comm.). Reports of a "brown band" infecting *Acropora formosa*, from the Great Barrier Reef could be RBD, it has affected 20 coral species in five scleractinian families and is reported to be different from BBD (Dinsdale 1994). Sea fans with "bands" experiencing mass mortalities have been reported from many other locations in the Caribbean (Peters 1993).

The identity of the primary cyanobacteria comprising the "band" of RBD are unclear, with different species proposed from distant locations. Closer examination of RBD by Rützler and coworkers (1983), revealed that *Phormidium corallyticum* was not present. *Schizothrix mexicana* and *S. calcicola* were the dominant cyanobacteria, a finding based on trichome morphology. The dominant cyanobacteria were two species of *Oscillatoria* in RBD found on scleractinian corals from the Bahamas (Richardson 1993). Subsequent investigation in Puerto Rico has confirmed the red band obtained from diseased gorgonians to be composed primarily of two species of cyanobacteria. Each cyanobacterium has trichome morphology and 16S rRNA sequences different from each other and *P. corallyticum* (Santavy et al. 1996). Additional research is needed to identify the pathogenic agent(s) of the disease(s) appearing as RBD on sea fans. RBD is analogous to BBD in its formation of a microbial mat, and the consortium of microorganisms is reported to include other cyanobacteria, the sulfur-oxidizing bacterium *Beggiatoa*, numerous heterotrophic bacteria, and the nematode *Araeolaimus* sp. The migration of the "band" in BBD was much faster than in RBD, with the movement of the red "band" occurring only during the day and the black "band" moving at night and day times (Richardson 1993). Different migration patterns of the cyanobacteria associated with RBD were observed as compared to *P. corallyticum* on scleractinian corals (Richardson 1993). A diel response of the band motility was observed with RBD, with the cyanobacterial filaments spreading outward in a diffuse network over the coral tissue during the day and consolidation of the band into a compact line at the

interface between the live tissue and denuded coral skeleton at night (Richardson 1993). RBD probably creates a microbial microenvironment with oxygen and sulfur dynamics analogous to those in BBD (Carlton and Richardson 1995).

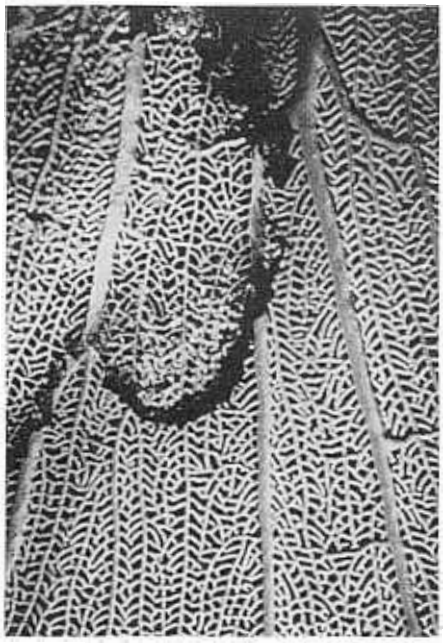

Fig. 2: *Gorgonia ventalina* affected with red-band disease, note that the axial skeleton remains devoid of purple tissue.

White-Band Disease

White-band disease (WBD) affecting acroporid corals was first reported in the 1970s from St. Croix, U.S. Virgin Islands (Gladfelter et al. 1977). Signs of tissue sloughing similar to that of WBD were reported on coral throughout the Caribbean Sea, including the Florida Keys (Dustan 1977; Antonius 1981a); Puerto Rico and the US Virgin Islands (Gladfelter 1982; Peters 1984; Davis et al. 1986; Bythell and Sheppard 1993); Antigua, St. Martin, Curacao, Nicaragua, and Panama (Gladfelter 1982); Tobago (Laydoo 1984; Davis et al. 1986); the Bahamas (Busch 1986; Ritchie and Smith 1995b); and Bermuda (Antonius 1981a). Its distribution has been extended to other oceans, with reports of WBD from the Red Sea (Antonius 1988); the Philippines in the Pacific (Antonius 1985); the Gulf of Oman off the Arabian Sea (Coles 1994) and the Great Barrier Reef (pers. comm. John Thompson). WBD has been reported to affect *Diploria strigosa, Montastraea annularis, Porites* spp., *Agaricia* spp., *Mycetophyllia* spp., and *Colpophyllia natans*, among other species, in the Western Atlantic (Dustan 1977; Antonius 1981b; Peters 1984). In the Pacific Ocean and Red Sea, the most susceptible species were members of the families Acroporidae and Pocilloporidae, although WBD has also been reported from six other scleractinian families, with most genera in the Faviidae (Antonius 1985; Coles 1994). When faviid corals are affected, recovery is more frequent than in the more susceptible Acroporidae where mortality is more prevalent (Antonius 1985).

The disease is characterized by tissue peeling off the skeleton and sloughing away, leaving a "band" of white, denuded skeleton several cm wide next to apparently healthy tissue (Fig.3). The exposed skeleton is colonized eventually by filamentous algae and rarely is recovery observed. In comparison to BBD, there are no visual signs of pathogens or parasites at the sloughing tissue interface, although bacteria, ciliates, nematodes, and microcrustaceans can be found on the freshly-denuded skeleton (Gladfelter et al. 1977; Antonius 1981a, 1985). The disease begins at the colony base moving upward towards the tips, destroying entire colonies in the most extreme cases. On *Acropora cervicornis* colonies, tissue necrosis also can begin in the middle of the branches. In contrast, predator damage by fish grazing, gastropods such as *Corallophilia*, or fireworms is characterized by random white patches, algal patches, or tissue destruction originating from the tips of the branches (Ott and Lewis 1972; Brawley and Adey 1982). The advancement of WBD can be up to 4-5 mm/day, much greater than the growth of regenerating areas, which is approximately 0.3mm/day, and the growth of recruits at approximately 0.1-0.2 mm/day (Davis et al. 1986).

The etiology of WBD is uncertain, with ambiguous reports confusing our present understanding. Aggregates of bacteria were reported in acroporid colonies affected with WBD from St. Croix, Bonaire, and the Netherland Antilles (Peters et al. 1983). Bacterial aggregates were found also in apparently healthy colonies at St. Croix, but within five years up to 95 percent of the *Acorpora palmata* colonies had died (Peters 1984). Bacterial aggregates have been found in acroporids without signs of WBD from other western Atlantic locations (Peters, unpub. observ.), but the role of the bacteria in the development of the disease is still unclear. Although the number of bacterial aggregates in grossly diseased and healthy colonies overlapped, the counts of bacterial aggregates per area of tissue were significantly higher in the diseased colonies from St. Croix (Peters 1984). Diseased colonies from the Indo-Pacific region and the Red Sea have not been examined histologically; therefore, it is unclear whether the bacterial aggregates are present.

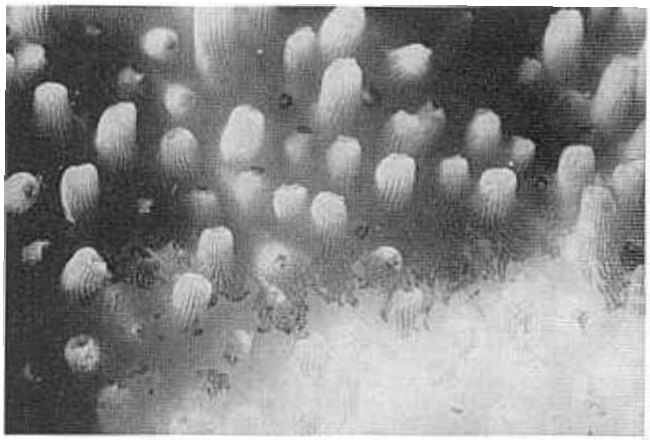

Fig. 3: White-band disease on *Acropora palmata*.

A study at Lee Stocking Island, Bahamas, conducted by the authors, examined a patch of *Acropora cervicornis* affected by WBD. Many colonies had signs of WBD in November 1993, with notable recovery occurring over the next 10 months. In March 1994, few colonies had any recent WBD tissue destruction as indicated by bare skeleton, and in September, algae had overgrown the exposed skeleton. Many colonies appeared to have recovered from WBD observed in November 1993, but about one-third of the colonies were observed to have pale pigmentation in March. In September 1994, few colonies displayed WBD signs or paling, but most colonies had recovered from the WBD. No recent tissue loss and normal tissue pigmentation were observed. Recovery from an outbreak of WBD on acroporids has not been reported previously (Santavy et al. 1995a).

Histological and ultrastructural studies revealed the presence of bacterial aggregates in the Lee Stocking Island samples. The relationship between the coral fitness status and the number of bacterial aggregates was used to determine their relationship to the disease process examined in colonies collected in November 1993, March 1994, and September 1994. Most of the WBD-affected colonies were associated with the highest number of bacterial aggregates; significantly fewer bacterial aggregates were found in colonies with pale pigmentation. The apparently healthy colonies possessed the lowest number of bacterial aggregates. Bacterial aggregates were most numerous in WBD-afflicted colonies at the tissue-skeletal interface, and these decreased significantly with increasing distance from the diseased tissue margin. The greatest numbers of bacterial aggregates were associated with the base of the colony, with fewer numbers of bacterial aggregates in the mid-branch section, and the fewest numbers of bacterial aggregates were in the branch tips (Santavy et al. 1995a). The relationship between the disease signs and the zonation of the bacterial aggregates within the tissue suggests that the bacteria have a role in manifestation of WBD.

The aggregates, up to 40 µm in diameter, contain numerous bacteria in the calicoblastic epidermis lining the gastrovascular canals (Peters et al. 1983). Examination of the bacterial aggregates by electron microscopy revealed

that each bacterial aggregate was separated from the adjacent coral cells by a bilayered membrane of uncertain origin (Santavy et al. 1995a). The bacterium contained within the aggregates appeared as bundles of filaments, possessed a typical Gram-negative wall, and appeared to be either unicellular in the state of division or multicellular as evidenced by the septal formation between elongated cells (Santavy et al. 1995a).

An enigma that remains to be resolved is the inconsistency of bacterial aggregates associated with *Acropora cervicornis* and *A. palmata* with gross signs of WBD. Bacterial aggregates were not found in all corals with degenerating tissue from other locations examined by the authors. Bacterial aggregates were found in tissues of *A. cervicornis* affected with WBD from Rainbow Gardens Reef in the vicinity of Lee Stocking Island, Bahamas. But WBD-affected *A. cervicornis* did not contain any bacterial aggregates at the proximal reef location, Norman's Pond Reef, and at South Carysfort Reef in the Florida Keys (Kozlowski 1996). Furthermore, histopathological analysis of *A. palmata* from Puerto Rico with putative WBD did not contain these bacterial aggregates, but did contain other potentially pathogenic organisms and tissue deterioration. The histopathology of the corals from Puerto Rico appeared very different from that observed in *A. cervicornis* from Rainbow Gardens (R. Bruckner and D.L. Santavy, unpublished data). The absence of any obvious infectious agent or bacterial aggregates in these corals with similar gross characteristics of WBD, suggests that either visible signs of this disease do not represent the same disease, or that WBD is caused primarily by other factors.

Colonies could possess similar external or gross disease signs but might be afflicted with different pathogens or stressed by abiotic factors. Perhaps this explanation is applicable to WBD, a term that generally has been applied to all species of corals in which tissue is sloughing from the skeleton. "White plague" was transmitted by inoculating material from the affected tissue interface onto the surface of another coral that had been intentionally injured (Dustan 1977). Only material obtained from diseased *Mycetophyllia ferox, M. lamarkiana,* and *Colpophyllia natans* was able to produce infection in healthy corals of the same species. Bacteria were observed in the inoculant; however, no microbiological studies were performed to isolate and characterize potential bacterial pathogens. White plague obtained from the previous species did not affect *M. annularis, Porites astreoides,* or *Stephanocoenia michelini*. It was hypothesized that the appearance of similar signs of disease in the latter species might be due to another agent, or those species were resistant to the inoculum (Dustan 1977). In contrast, xenografts of affected and healthy coral species did not transfer WBD to healthy colonies (Antonius 1981a, b). Thus, the term "stress-related necrosis" was proposed to describe the condition in which degenerative changes in cell structure are observed in the absence of obvious pathogens, after histopathological observations (Peters 1984). Ritchie and Smith (1995a,b) have examined bacteria from the coral surface mucus layer in another WBD-like disease affecting *A. cervicornis* from San Salvador, Bahamas.

During the summer of 1995, a new disease emerged in the Florida Keys. It began affecting only *Dichocoenia stokesii*, but by the end of the season, it was reported to affect over a dozen different coral species (Zorpette 1995). Superficially, signs of tissue loss were similar to WBD described from other areas, but the rate of coral tissue loss was greater. Rapid tissue necrosis was observed on massive, branching, and encrusting corals, with sloughing tissue progressing at a rate of over a cm per day, much faster than any other reports of WBD, white death, or white plague. The disease destroyed small colonies in a matter of weeks. The extremely rapid rate of tissue loss and ability to cause mortalities in many species of corals make this condition of particular concern, not only for the Keys, but also for the rest of the tropical western Atlantic. The etiology of the disease is under investigation.

Yellow-Blotch Disease

Yellow-blotch disease (YBD) is a condition that affects massive heads of *Montastraea faveolata*, often found on colonies up to 7 m (height). It was first noted by C. Quirolo (Reef Relief, Key West) in the lower Florida Keys in 1994. YBD is described as a necrotic event where concentric tissue margins have a pale yellowish-colored border several cm wide (Fig. 4). Inward from the tissue margin, a bleached denuded skeleton remains and the pale yellow tissue border gradually becomes apparently healthy tissue. Monitoring efforts have recorded an increased incidence of this disease, accompanied by alarming coral mortality on reefs off Key West (C. Quirolo, unpublished data). Histological investigations reveal predictable degenerative changes in the tissues and cells in the affected areas and the presence of pockets of unusual crystalline-like material in the gastric cavity. Examination of the tissue with electron microscopy and additional planned studies will also help to determine if bacteria play a major role in this deposition and its relationship to the disease state.

FACTORS TO BE CONSIDERED IN DISEASE STUDIES

Our understanding of how changes in environmental conditions can influence coral diseases is limited, and the causal agents currently are undescribed for most coral diseases. Many times similar gross signs of coral abnormalities are classified as the same disease by those making observations in the field. After histopathological evaluation of these diseased corals, it is apparent that there are different pathologies associated with the same gross signs. Therefore, many different histological conditions are being classified into few described diseases. Consequently, many of the abnormalities are not adequately characterized and cannot be distinguished by untrained observers. Closer observation of coral diseases indicate that a single disease state might actually be caused by multiple biotic or abiotic factors; the unhealthy condition of a coral can be manifested in a limited number of ways. Subtle variations in gross appearance and species afflicted by particular disease signs are indicative of different diseases with different etiologies (e.g.,WBD vs. stress-related necrosis vs. white plague or temperature-related bleaching vs. UV-related bleaching vs. microparasites) (Brown 1990; Peters 1993).

Fig. 4: Yellow-blotch disease on *Montastraea faveolata*.

Localized epizootics or abnormal conditions often are first reported by coral reef ecologists or recreational divers. Diagnosis of the disease could be greatly facilitated if appropriate data are gathered and careful observations and collections are provided to coral disease researchers. Detailed information recorded at the time of collection or initial observation will aid in elucidating causative factors and provide guidance for applying proper collection and optimized preservation methods.

Classifying the various visual categories of dead tissue areas on corals in the field, as well as deviations from normal coloration or morphology, is a necessary exercise to correlate damage with causes. Figure 5 presents the minimum information that should be collected in monitoring efforts or single observations on the condition of coral colonies. It is preferable to photograph the affected colony, to provide a close-up of the affected tissue, with a scale and recorded observations. Additional information should include basic observations on other biotic and abiotic factors that might be responsible for disease manifestation. This exercise could provide data to focus research on poorly understood coral diseases, eliminating studies that might be redundant or that might not yield pertinent information. It might identify outbreaks of new diseases early in their development.

We propose that collection of these data be considered for incorporation into the worldwide monitoring program to be conducted for the "International Year of the Reef". These efforts would aid in evaluating specific hypotheses regarding the loss of coral tissue from reefs. Epidemiology alone will not explain why diseases occur on reefs. Multidisciplinary studies incorporating techniques from biochemistry, ecology, histopathology, microbiology, physical oceanography, physiology, toxicology, virology, and others will be necessary. Field and experimental data will be required on the effects of exposure to physical, chemical, and biological stressors to identify the etiologic agent(s) of tissue loss in corals.

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