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Rapid spread of diseases in Caribbean coral reefs

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Abstract: The variety and frequency of diseased coral reef organisms has increased across the Caribbean during the last 10 years. Black band, white band, white pox, and white plague diseases have become more widespread. Yellow band disease now affects a major portion of *Montastrea annularis* across the Caribbean. Mortality of Yellow band disease-affected tissues was observed to increase in the southern Caribbean during early 1997, with affected tissue turning white and dying. During the past two years a major fraction of encrusting red calcareous *Porolithon* algae died across the Caribbean. This disease, also found in the Indian Ocean, lacks the orange band characteristic of Pacific coralline lethal orange disease. A millimeter thick white circular rim is seen between live tissue and dead skeleton. Numerous other emerging diseases have attacked many other organisms in this period, including gorgonians, sponges, and echinoderms. Many of those now known have not yet been described. Generally, most diseases affect one or few species (except white plague and black band diseases), and their spatial and temporal distributions appear unrelated to bleaching, pollution (except black band disease), sedimentation, or grazing. This paper brings together observations made separately by many researchers over a large area and long period, in order to evaluate the geographic and historical distributions of each disease. Photographic images are included which show diagnostic field criteria for recognizing each disease.

Key words: Disease syndromes, Caribbean, corals, sponges, gorgonians, coraline algae.

An alarming number of diseases is now being reported from coral reefs, especially in the Caribbean region. It is essential to be precise about observations categorized as diseases of coral reef organisms. All of the afflictions presented in this paper as diseases are likely to be infectious in etiology and are likely to involve characteristic signs of progressively spreading necrosis. While for some the infectious pathogenic organism has been identified,

for others the infectious organisms are not yet confirmed. Searches are underway to confirm the latter, but either the specific organism has not yet been identified or Koch's postulates have not all been satisfied to establish responsibility for the disease.

We have restricted our attention to those afflictions which result in the loss of tissue viability and which may be caused by a pathogenic and transmissible microbe or by a combination of microbes acting in concert to produce the disease. Where several agents are involved in the appearance of the disease, this combination has been referred to as a microbial consortium (Richardson *et al.* 1996).

Several agents producing necrosis in coral reef organisms are often confused with diseases but should not be so identified. We distinguish from infectious diseases the genetic mutations that result in growth form abnormalities (genetic diseases) and the physiological responses to extreme or anomalous conditions (stress responses). Our attention has focused on infectious diseases rather than on these other problems because of the preponderance of infections and their escalation as contributions to mortality and to rapidly changing community admixtures in the tropical reef.

Genetic diseases include unusual growth patterns resembling, tumors, neoplasms, or galls, which have been analogized to cancers. They are typically expressed as nodular growths on colonial surfaces, or as distinct areas with different patterns, arrangements, or sizes of polyps than surrounding zones. The cause of these abnormal growths is still unknown, but it is possible that they are the result of somatic (non-germ line) cell mutations, presumably in regulatory genes. They are most common in the *Acropora* and in the *Diploria.* They have been known since the 1950s and are reviewed in an excellent paper by Peters and coworkers (1986). No pathogen has ever been found to account for these growths and these abnormalities have not noticeably increased since the 1950's. A change in their abundance would be important, and would suggest increasing rates of mutation.

One of the most recent examples of a response to environmental stress is coral reef bleaching, which has sometimes been misinterpreted as a disease. In contrast, coral bleaching is a general stress response resulting from the fact that the multi-dimensional (Hutchinsonian) niche of environmental conditions required for stable symbiosis is smaller than that of either one or both of the partners. Bleaching has long been known to be triggered experimentally by elevated temperature, salinity, or illumination, excessive sedimentation or turbidity, or chemical pollution (Williams and Williams 1990). Bleaching may also be a stress response to bacterial infection (Kushmaro *et al.* 1996). Natural mass coral reef bleaching in the field has been found to accompany periods of unusually high temperature exposure (Goreau *et al.* 1992). However, once triggered by temperature, high light levels, hyposalinity, sedimentation, or shading may accentuate the response. Although bleaching is clearly a physiological response to extrinsic stress, in most cases bleaching responses are episodic, and predictably follow exposures of the reef organisms to environmental extremes. There is no evidence to indicate that bleaching is infectious. Bleaching is a physiological modulation from which recovery is possible, provided the precipitating stress is not protracted, extremely severe, or accompanied by other sources of stress.

There are other contributors to coral mortality. A major cause of coral death is competitive overgrowth by other organisms, such as sponges, algae, or other corals. These overgrowths have accelerated in many sites around the Caribbean in recent years. Large numbers of corals have been killed by algal overgrowth, by overgrowth from several different sponge species, and by overgrowth by didemnid ascidians. Observers have described these interactions as diseases because they systematically kill the coral, but they are more attributable to immunological factors, not to pathogens, despite their alarming and recent spread. These competitive losses may be influenced by environmental perturbations that reduce the efficiency of affected organisms to defend them-

selves. That efficiency would be compromised by increased levels of nutrients in the coastal zone that favor growth of benthic algae (eutrophication), and promote increases in phytoplankton and the zooplankton. The latter are the food supply for the ascidians. An increase in bacteria and suspended particulates, derived from land based sources, results in increased organic loading of the water column, and provides food for sponges. The overgrowth of corals by sponges which attack from inside the skeleton resembles disease because it proceeds progressively across the entire colony.

Predation on corals by starfish, fish, gastropods, or polychaetes may contribute to tissue death and colonial destruction, especially when severe. Fish bites, grazing by sea urchins, and predation by other marine organisms do sometimes kill corals and other sessile invertebrates, but these agents leave behind characteristic scars. The lesions do not spread and do not generally introduce infection or produce inflammation. Some of these bites could offer a portal of entry for pathogens carried freely in the water column or harbored in oral secretions of the fish (Antonius 1981).

RESULTS

Diseases of Scleractinian corals

Black band disease (BBD): The first reported coral disease in the Caribbean was presented by Antonius (1973). He found rare cases of a pathology which he called BBD. The coral tissue was observed to be consumed by a steadily advancing dark colored slimy mat, leaving white skeleton behind. The thickness and pigmentation of the black band can vary from a few mm. to several cm. wide, and range in color from black to purple or red (Figs. 1-4). Subsequent work identified the cyanobacteria, *Phormidium_corallyticum*, as the primary pigmented organism in this mat (Richardson *et al.* 1997a). During early observations of BBD, prominent *Beggiatoa* spp. mats are known to be one of the pathogens found within the black band, and in some cases these can be observed macroscopically (Garrett and Ducklow 1975).

More recently the mat has been shown to be a complex microbial community. The cyanobacterium forms a gel-like matrix of filaments in which the other organisms live, while generating large amounts of oxygen in the upper layers. The mat also serves as a barrier to the diffusion of oxygen, thereby maintaining anoxic conditions at the base of the mat in the day time, and throughout the mat at night (Richardson *et al.* 1997a). Some initial damage to tissue, of non-specific origin, creates a local wound which is attacked by heterotrophic bacteria. Biochemical decomposition creates locally anoxic conditions, which are utilized by heterotrophic anaerobic sulfate-reducing bacteria such as *Thiobacilli*, which use sulfate ions in sea water as terminal electron acceptors, generating high local concentrations of hydrogen sulfide, causing further death of coral tissue. The hydrogen sulfide is utilized by the microaerophilic autotrophic bacterium *Beggiatoa*, which derives metabolic energy by oxidizing sulfide with oxygen. *Beggiatoa* glides to position itself along the redox gradient between hydrogen sulfide diffusing from below and oxygen diffusing from above, and migrates vertically on a diurnal cycle. *Beggiatoa* forms crystals of elemental sulfur, which are incorporated into the cytoplasm and impart a white color. Under high light conditions the amount of oxygen produced by the photosynthetic cyanobacteria forces the *Beggiatoa* to migrate deep into the layer, but at night when no oxygen is produced and when the whole mat becomes oxygen consuming, it can migrate to the surface. BBD often first appear in areas which are regarded as the more polluted sites in most islands, and spreads radially outward from these sites. Its abundance mimics the distribution of human influenced areas, with the largest impacts in areas near sewage outflows from land or areas of high turbidity from eroded soil. BBD has become steadily more common and widespread with time. It is most common in shallow water, and is now seen everywhere in the Caribbean (Bruckner *et al.* 1997).

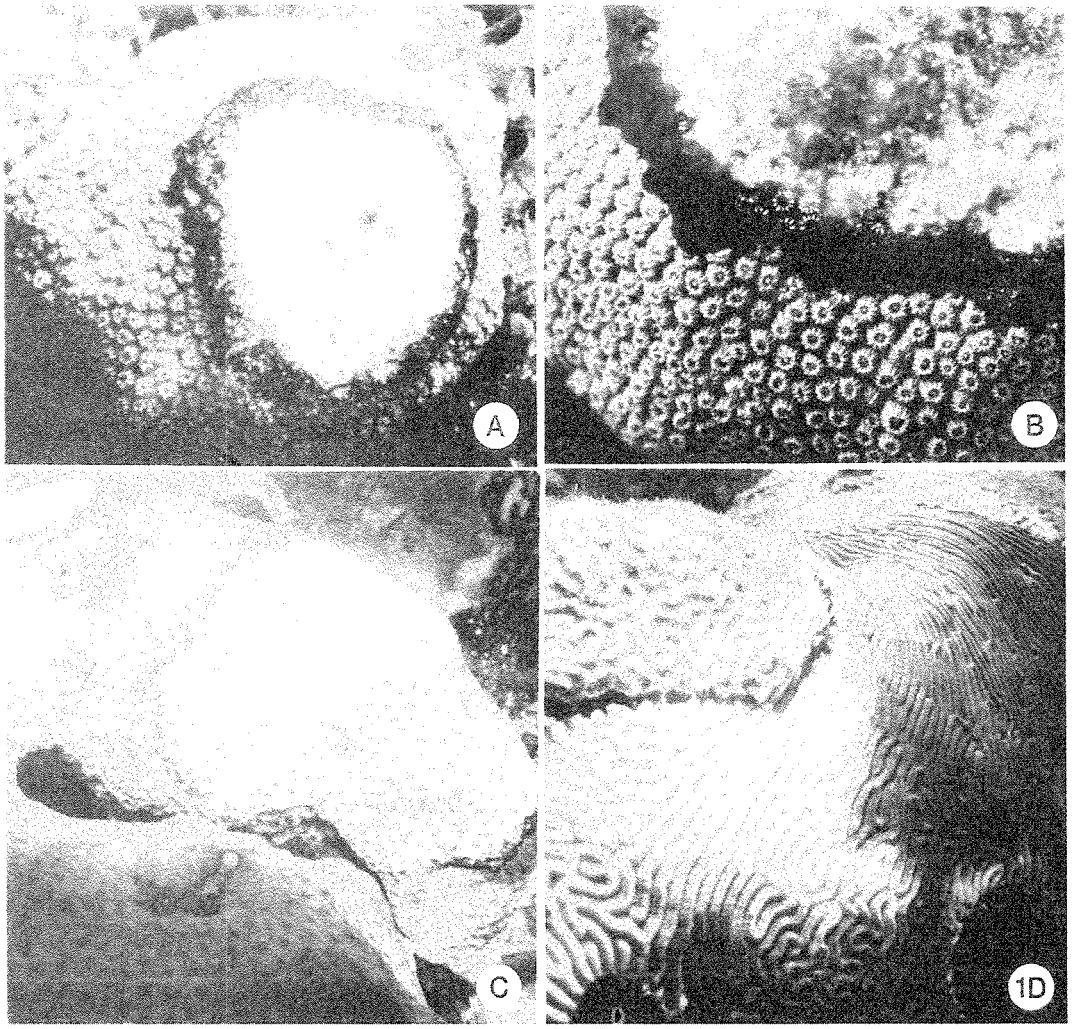

Fig. 1. A. Black band disease on *Montastrea annularis*. (Photo by R. Hayes). B. Black band disease on *M. annularis* (photo by J. Cervino). C. Black band disease on *Zooanthus*. (photo by M. Hayes). D. Black band disease and bleaching on *Diploria labyrinthiformis* (photo by M. Hayes).

Along gradientes towards muddy mats grow at the rop surface (Goreau & Goreau, un...)

White band disease: In 1977 the second epizootic named WBD which caused massive mortality of elkhorn corals *Acropora palmata* on the reefs of Buck Island and Tague Bay, St. Croix (Gladfelter 1992). It is now apparent that this disease progressively destroyed the overwhelming bulk of all Caribbean *Acropora* from the base up in the late 1970s and early 1980s, but no detailed observations were made at the time of its spread. Hence, its timing is only approximately determined in retrospect by interviews with divers and fishermen. WBD is now epizootic across the Caribbean and while there has been some new recruitment of affected species at some locations, they are often rapidly infected by WBD, therefore limiting the amount of these species to no more than a few percent of former prevalence. WBD is slow acting (few mm/day), characterized by tissue sloughing off skeleton. No pronounced bleach-

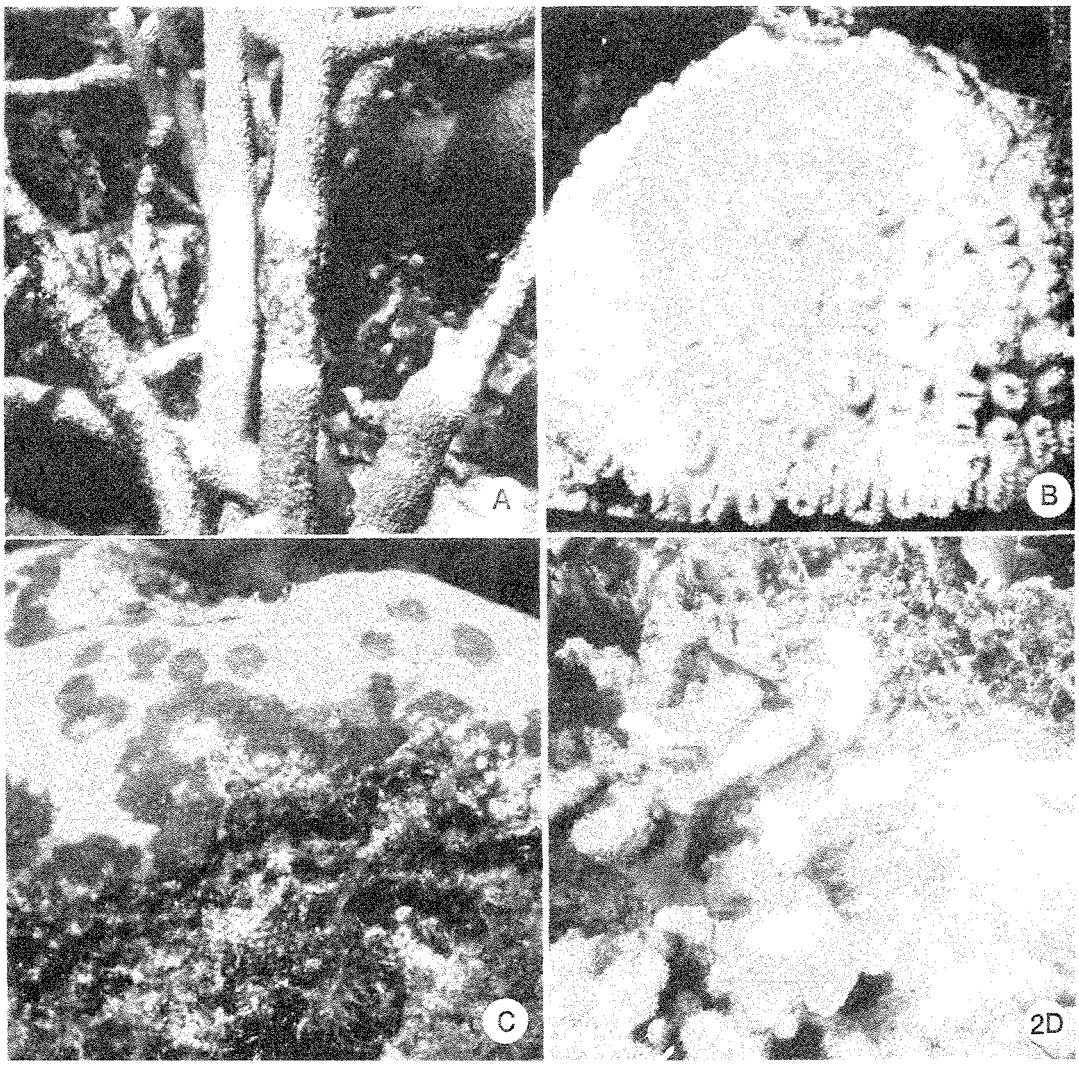

Fig. 2. A. White band disease on *Acropora cervicornis*. (photo by J. Cervino). B. Coral plague disease on *Dichocoenia stokesil* (photo by L. Richardson). C. Dark spot disease on *Siderastrea siderea* (photo by J. Cervino). D. White pox disease on *A. palmata* (photo by M. Hayes).

ing is evident, and usually starts at the base working its way upward. Reports on histopathological examinations of coral tissues from *A. palmata* and *A. cervicornis* show gram negative bacteria in affected tissue (Peters *et al.*, 1983). These are found in bodies surrounded by calicoblastic epidermis. Other studies have not found these bacterial aggregates in colonies showing signs of white band disease, so their role in tissue loss remains uncourtain (Santavy & Peters, 1997).

Another form of white band was recognized in the early 1990s as being distinct from the previously recognized variety, now named white band I and described as WBD, Type II (Bythell and Sheppard 1993). WBD I is distinguished by a narrow white band, no more than a few millimeters across, which divides dead skeleton from peeling tissues, affecting both *A. palmata* and *A. cervicornis*. WBII in contrast shows a wide band of bleached tissue, often 5-10 cm wide, bordering receding normal tissue,

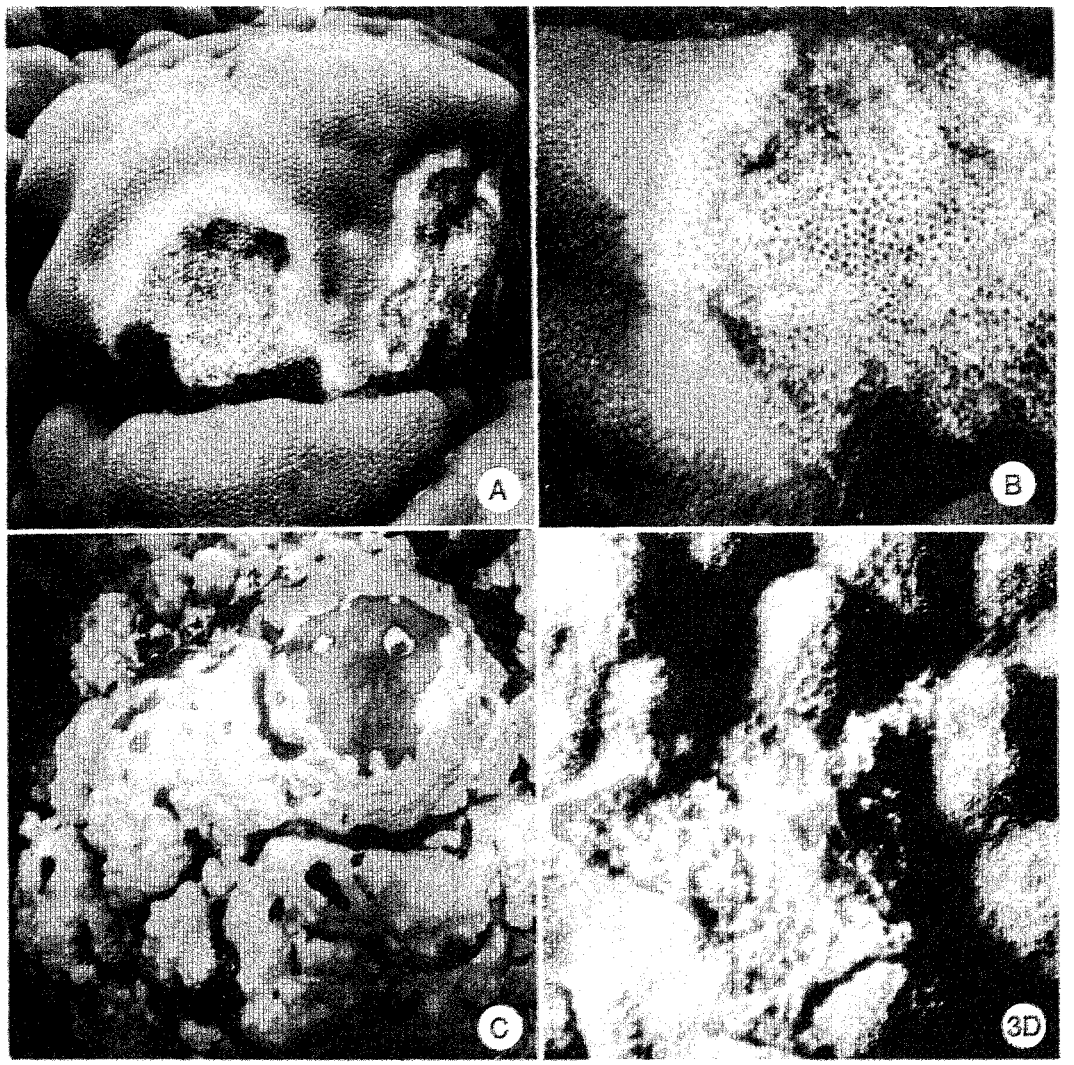

Fig. 3. A. Yellow band disease on *Montastrea annularis* (photo by J. Cervino). B. Yellow band disease on *M. annularis* (photo by J. Cervino). C. Red band disease on bleached *M. annularis* (photo by M. Hayes). D. Red band disease on *M. annularis* (photo by L. Richardson).

affecting Acroporid species, most commonly *A. cervicornis* (Ritchie and Smith, 1997 - 1998). Different types of bacteria, which have not yet been identified, are found in each type of white band. WBI and WBII are both widely distributed all around the Caribbean, and affect both *Acropora cervicornis* and *Acropora palmata.* (Fig. 1).

Red band disease (RBD): In 1983, RBD, was described to affect *Gorgonia ventalina* near Carrie Bow Cay, Belize (Rutzler and Santavy 1983). RBD is associated with a cyanobacterium of the group *Oscillatoria* spp. (Richardson 1992). The term RBD was originally used to describe a mat of red cyanobacteria resembling BBD from the Bahamas (Richardson 1992). RBD has been observed on the west coast of Puerto Rico infecting *G. Ventalina*, and in the Florida Keys. The primary cyanobacteria present in the red band are not known, with different species thought to be

responsible in various locations (Santavy and Peters, 1996). Examinations conducted by Rutzler and coworkers (1983) reveal that *Phormidium corallyticum* was not present. *Schizothrix mexicana* and *S. calciola* were the domninant species found. Dominant species of cyanobacteria *Oscillatoria* are found in samples collected in the Bahamas (Richardson 1992). Red band samples from diseased gorgonians in Puerto Rico confirm two species of cyanobacteria. RBD is similar to BBD in its development of a consortium of microbial communities within the mat. They include other cyanobacteria, the sulfur-oxidizing bacterium (*Beggiatoa*), heterotrophic bacteria, and the nematode, *Araeolaimus* (Figs. 3-4).

Yellow band disease (YBD): YBD was first documented in 1990, associated with bleached corals in the Cayman Islands (Hayes and Bush 1990). Its abundance steadily spread throughout the reef and affected coral tissue slowly died. At the time the pattern was first noticed the corals were recovering from a bleaching event, and it was interpreted to be a variety of stress response or a stage in the recovery from bleaching. In subsequent years, YBD has continued to appear independent from bleaching. Quirolo (1995, unpublished) noticed its prevalence at sites near Key West, Florida and began using the term YBD. It has now been confirmed in many Caribbean sites and is an important predecessor to mortality of the reef builder, *M. annularis*, in much of the region (Figs. 3A and 3B).

Dark spot disease (DSD): DSD is another new coral affliction which appeared during the late 1990s. This affliction includes a characteristic dark band along the edge of tissue which slowly dies back. In early stages it can appear as dark spots in the middle of normal tissue, which expand into a ring surrounding dead coral. This disease is most common on *Siderastrea siderea*, on which it assumes several hues, including purple, maroon, and brown. Some examples are shown in figure 2C. This affects a major portion of all colonies of this species at reefs Caribbean-wide.

White pox disease (WPD): White pox, which causes round white spots or patches on *Acropora palmata*, was first reported around 1995 in the Florida Keys (Porter and Meier 1992). It has spread rapidly since then, and is now seen at sites throughout most of the Caribbean. White pox is differentiated from WBD because it starts as small, round, expanding white patches, bare of coral tissue, in the middle of normal colored tissue, on both upper and lower surfaces (Fig. 2D), whereas WBD normally proceeds upward from the base (Fig. 2A). Coral colonies affected commonly are covered with many fairly uniform white circular patches, up to 10 cm. across. These may be abundantly distributed on one colony while adjacent ones have few or none.

Coral (white) plague: Another coral disease, termed the Plague, was identified in the Florida Keys (Dustan 1992), affecting non-Acroporid corals, especially *Colpophyllia* and *Mycetophyllia*. This progressed very slowly, and appeared to be infectious, but there is little or no information available on this phenomenon since then, and it appears to be difficult to relate it to any known cause or subsequent disease observations. Since then a faster acting (cm/day) type plague has been affecting 18 species of non-Acroporid corals in the Florida Keys, where there have been seasonal attacks (Richardson *et al.* 1997b). It appears similar to the earlier version, but differs with respect to rate of spread. This latest form of Plague spreads across a colony extremely quickly, while the earlier-reported version progresses much slower. This disease spreads exceptionally rapidly, killing rounded coral colonies from the base upwards, at a rate of cm. per day, and affecting most coral species in the Florida Keys patch reefs. (Fig. 2B).

Rapid wasting syndrome (RWS): RWS attacks many morphotypes of *Montastrea annularis* and *Colpophyllia natans*. A fungus and a ciliate are found in microscopic specimens in close association with diseased tissues (Cervino *et al.* 1998). These potential

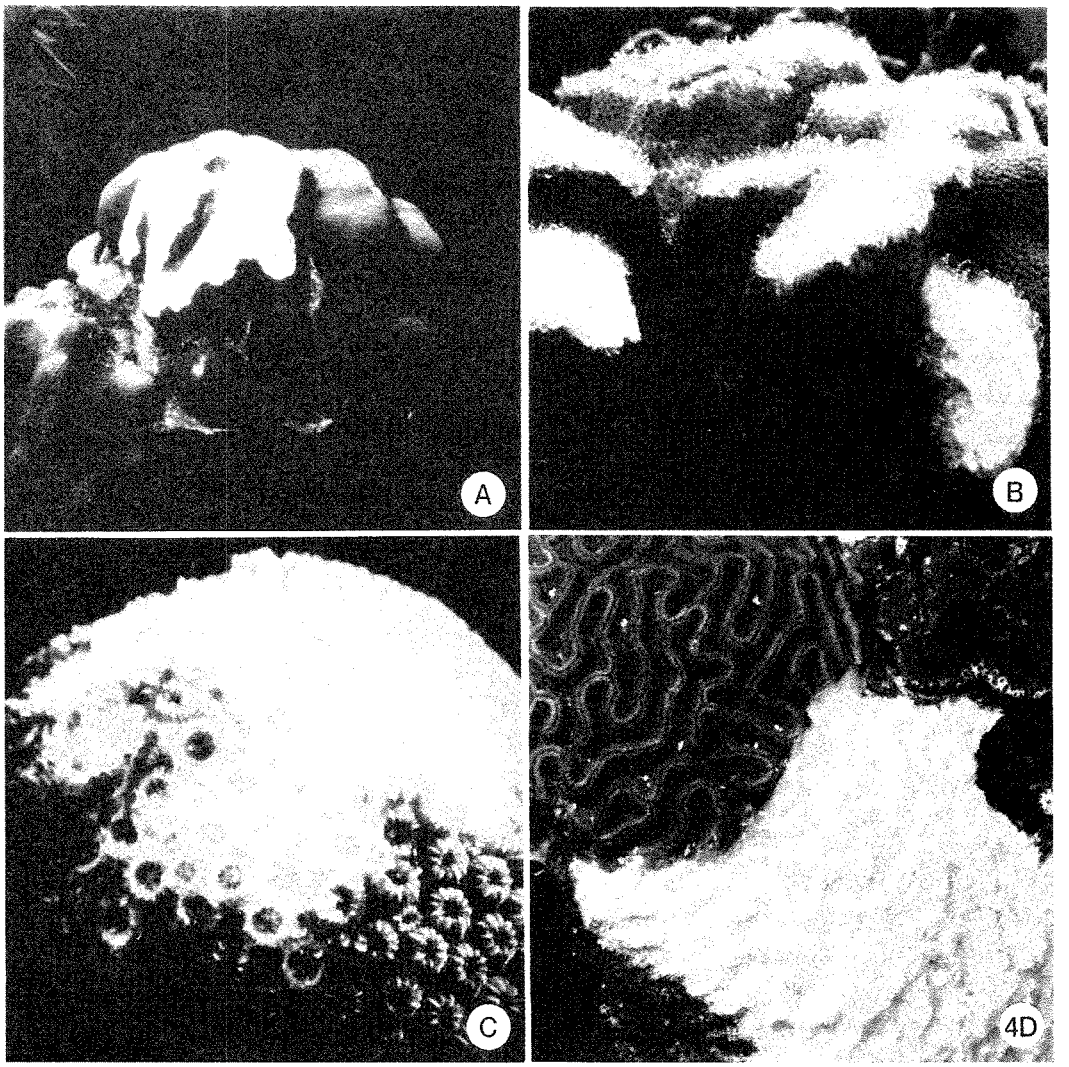

Fig. 4. A. Rapid wasting syndrome on *Montastrea annularis* (photo by J. Cervino). B. Rapid wasting syndrome on *M. annularis* (photo by J. Cervino). C. Rapid wasting syndrome on *M. annularis* (photo by J. Cervino). D. Rapid wasting syndrome on *Colpophyllia natans* (photo by J. Cervino).

pathogens are not yet identified. Infected tissues may be subsequently attacked by parrotfish (Cervino *et al.* 1998), which cause deep excavation of the skeleton (Bruckner and Bruckner 1998) spreading up to several centimeters per day. The syndrome was first reported in Bonaire, Netherlands Antilles (Bak and De Meyer 1996, unpublished), but now appears to have a distribution throughout the Caribbean (Cervino *et al.* 1997). Figures 4A-4D show cases where RWS has attacked the tops of colonies, but then stops, is overgrown by algae, and new centers of infection appear nearby which attack from the lower edge, and spread laterally. Figure 4D shows RWS on *Colpophyllia natans*, on which the erosion of the skeleton is up to 2 cm deep, about 10 times that seen on *M. annularis* (Cervino and Smith 1997).

Diseases of other reef organisms

Sponge disease: Sponge disease takes the form of white bordered dead holes in the sides of barrel, tube, and encrusting sponges. The disease was not noted before 1996 when it seen to spread rapidly in the Belize Barrier Reef Tract, killing many large barrel sponges, *Xestospongia muta* (Paz, 1997, unpublished). The largest barrel sponge in Curacao has been steadily killed off during the first half of this year (Nagelkerken, 1997, unpublished). A different slit barrel sponge is widely affected in Tobago, and several species of tube and encrusting sponges are affected in Portobelo, Panama. All these cases have a similar appearance, with the necrotic edge appearing white (Fig. 5C). Samples have been taken for microbial culture, but pathogens have not been identified.

Coralline algal disease: Coralline algal diseases are also new and rapidly expanding. Orange-yellow growths of an unidentified bacteria attacking *Porolithon* crusts with expanding circles of orange rimmed (about 1.0 cm wide) dead skeleton have been reported from the Pacific (Littler and Littler 1994). This affliction was termed coralline algae lethal disease or "CLOD".

In February 1996 a new condition began to kill *Porolithon* in Jamaica, starting at the water line as small spots which expanded in size, frequency, and depth distribution, killing about half of all Porolithon at sites on the west, east, and south of the Island within 6 months. This disease differs from CLOD in that no broad orange band is present. Instead a thin white rim, about 1 to 2 mm wide, expands into pink *Porolithon*, with a green overgrowth composed of filamentous green algae. Figure 5D shows this condition. This disease appears to have killed between a quarter to three quarters of *Porolithon* recently at many Caribbean sites. Microscopic examination shows that the white band is soft and can be gouged out with a needle, and the white rim spreads into normal *Porolithon*, followed by ramifying branched filamentous green algae and fungi . In all locations where it occurs the disease is easy to identify when it is spreading rapidly and marked by clear expanding white-rimmed green circles, but becomes progressively more difficult as these halos merge, resulting in a green and white speckled surface with residual pink patches. These are now so ubiquitous where the encrusting red alga was prevalent that its loss seems normal. Much or most of it has vanished recently in an almost unnoticed fashion, leaving behind an unhealthy mottled fuzzy green surface which was once hard, smooth, and pink. The identities of a pathogen and of the filamentous algae replacing them are still unknown.

Sea fan disease (*Aspergillosis*): Since the 1980's a number of reef organisms other than stony corals have shown increasing signs of disease. Sea fan mass mortalities have occurred in various locations in the Caribbean (Laydoo 1983; Guzmán and Cortés 1984; Garzon-Ferreira and Zea 1992; Diaz *et al.* 1995), which appear to have been epizootic occurrences very similar in appearance to the pan-Caribbean sea fan disease outbeak which took place since 1995 (Nagelkerken *et al.* 1996), which was identified as an *Aspergillus* spp. fungus (Smith *et al.* 1996). (Figs. 5A and 5B).

Sea urchin disease: In 1983-84, a dramatic mass mortality wiped out most of the echinoid, *Diadema antillarum*, throughout the western tropical Atlantic and Caribbean (Lessios *et al.* 1984), spreading from Panama eastward, northward up the Lesser Antilles, and westward along the Greater Antilles. This event killed 90-95% of this environmentally significant marine invertebrate in the Caribbean (Williams *et al.* 1991). This species has recovered patchily since then, returning to near pre-1983 densities in some areas and not at all in others, and has been affected by at least two later mass mortalities (Williams *et al.* 1986, Williams and Bunkley-Williams 1996). The echinoid, *Tripneustes ventricosus*, underwent a severe die-off during 1995, with at least 95% mortality by visual estimates (Williams *et al.* 1996). Urchin mortalities in 1997 affected Antigua in

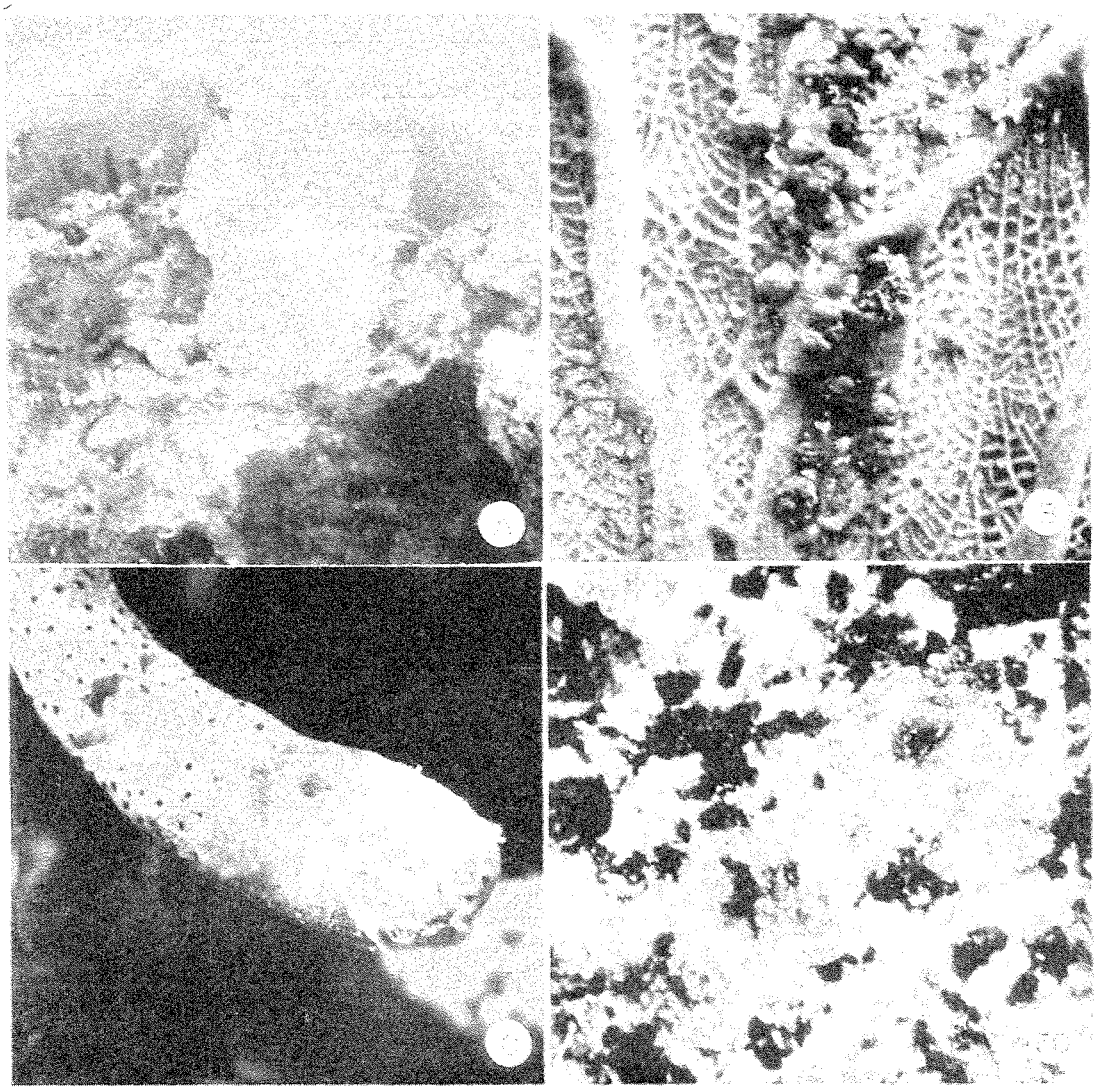

Fig. 5. A. *Aspergillosis* on sea fan, indicating the clear line of demarkation between normal and diseased tissues (photo by M. Hayes). B. *Aspergillosis* on sea fan, showing the galls at the diseased tissue boundary (photo by J. Cervino). C. Sponge disease on tube sponge (photo by J. Cervino). D. Coralline algal lethal disease on *Porolithon* (photo by J. Cervino)

June, Puerto Rico in July, and Jamaica between August and September. Other die-offs affected the urchins *Plagiobrissus grandis* during 1996 in Aruba and *Meoma ventricosa* during 1997 in Curacao (Nagelkerken *et al.* unpublished). No pathogenic agent has yet been identified for any of the echinoid mortalities, despite searches for them, but pathogens are thought likely to exist and probably not yet found due to difficulty of establishing correct culture conditions.

Pelagic species (turtle, fish and marine mammal) diseases: Mobile fauna have also been afflicted with diseases throughout the Caribbean region (Williams and Williams 1987). There have been repeated incidents of fish kills caused by fungi (Williams, unpublished). Turtles have shown a great increase in superficial tumors (fibropapillomas), and there have been episodic unexplained marine mammal deaths (Williams, unpublished). Currently,

there is an outbreak of Slime blotch disease of reef fish in South Florida and in Puerto Rico. This disease is thought to be caused by the protozoan parasite, *Brooklynella hostilis*, and a mixture of other pathogens (Williams and Bunkley-Williams, unpublished).

DISCUSSION

In this survey we have focused on infectious diseases of coral reef organisms with clear and recognizable syndromes, resulting in the progressive death of tissue on the surface of the colony. A common feature of these infections is that they are transmissible, spreading rapidly in distribution and extent. There are many cases of coral death of uncertain origin which may never be clearly attributable to a disease (Santavy and Peters 1997). If dying specimens have not shown recognizable and consistent disease-like signs, such as progressive spreading across a coral, we consider them of unknown origin. We suspect many of these may be due to physical damage by sources such as human contact, anchors, dredging, boat impacts, destructive fishing methods, large marine animals, storms, episodic sediment loading, or toxic exposures to soluble pollutants. Once these causative agents have intervened, they become virtually impossible to verify. However, physical damage to a coral colony may facilitate a disease process in the presence of viable pathogens (Antonius 1981).

In an attempt to organize and systematize the descriptions of the many diseases now being recognized in coral reef communities, we have noted several features which appear to be characteristic of these afflictions. For example, we note that most coral reef diseases affect only a few species, ranging between one and fewer than half a dozen in most cases. Often the species affected are similar in morphotype, and sometimes they are cogeners. The major exceptions are BBD and white plague which affect multiple species of scleractinian corals.

Also, most of these diseases have no obvious spatial or temporal correlation with pollution, with the major exception of BBD. This disease is most common in polluted shallow waters, and has spread in time and space as human development spread along marine coastlines. However environmental stresses to coral reefs, such as abnormal shifts in temperature, salinity, water clarity, oxygen, or pH, sedimentation, eutrophication, physical damage, or grazing, can increase mucus production. Increased stress from anthropogenic or natural disturbances could be raising the corals' metabolic activity. This causes the corals to expend energy, triggering their mucociliary system to produce excess mucus. The coral surface area then increases, which increases the chances of bacterial invasion (Peters 1984).

However, BBD can now be found in most places, although it seems to be more evident in polluted waters. This disease is world-wide in scope (Antonius 1997; Bruckner and Williams 1996). Caribbean coral diseases other than BBD are abundant in both polluted and unpolluted environments. More studies are needed to determine whether disease abundance correlates with pollution gradients. However, at this time pollution appears to be a minor determinant for most diseases. For example, coral diseases are roughly equally common in Curacao, a densely populated island with a large petrochemical industry, as in Bonaire, an island with no industry except salt, less than a tenth as many people as Curacao, and one of the most protected and managed Caribbean marine parks. Most of the diseases, except for black band and sponge diseases, are not notably more prevalent in eutrophic or sediment affected sites in Panama, Grenada, or Tobago. Most of the species afflicted by these diseases have or are approaching a pan-Caribbean distribution. Only white plague II and RWS appear to be more prevalent in few locations. This may be a reflection of the early stage of identification, rather than an indication of any spatial limitation upon the affliction.

Most of these diseases were not recognized until they had spread so widely that it was too late to identify source regions or the rates of spread. This is more a function of the rapidity

of spread than of lack of notice by marine scientists and divers. We were fortunate that Plague and RWS were identified early enough that their spread could be determined through early alerts and specific attention to site surveys.

The causative pathogens for many of these diseases are unknown, because either microbial isolates have not yet been cultured, or because they have not yet been identified, characterized, and tested using Koch's postulates. We suspect that almost all the putative diseases described here may eventually be found to have a suite of pathogens, whose relationship to each other and pathogenic and normal environments remains to be determined, along with their mode of action and spread. However, the pathogenic agents must be separated from the opportunistic agents which move in to devour necrotic tissue or to take advantage of newly exposed substrate for attachment. It is clear that there is little that can be done to stop the spread of these diseases until we identify the causative agents, how they act, where they came from, and how they are transported. This will require extensive field analyses, aquarium experimentation, and historical reconstruction of environmental perameters which may have liberated or redistributed pathogens into the coastal marine environment.

There are several possibilities to explain the resurgence of disease in the coral reef communities of the Caribbean within recent years. One thing we know is that this pathogenicity has not been expressed for very long. These diseases are not normal events, and they have increased steadily in their abundance and distribution. All of these tropical marine diseases appear kill hosts which had reached large sizes and ages.

The pathogens for these diseases may have been present all along and were not pathogenic because organisms were able to ward them off, but increased stress has lowered their resistance mechanisms, causing them to succumb to stresses which were hitherto non-lethal. If so, the pathogens should also be common in unaffected organisms.

Virulence or evolution of the pathogen may have resulted from the cumulative effect of resistance to environmental toxins, or elevated mutations caused by increased concentration of human-generated mutagenic chemicals.

Global climate change may have increased the abundance or spread of pathogens. This could explain those diseases which appeared following bleaching events. The problem is that many of them mimic bleaching and appear to follow bleaching only because they were first recognized only when bleaching faded while diseases advanced. Many diseases have appeared at times and locations where there was no bleaching, and their spread appears independent of bleaching.

Diseases can be spread by aerosols, but most known pathogens are far more easily spread by water transmission. Many reef areas are subjected to very high erosional sediment deposition, and this can kill corals without causing algae overgrowth, diseases, or mass bleaching. The uncontrolled experiments created when reefs are killed by large amounts of water transported soil near deforested and rapidly eroding hilly coasts suggest that further additions of small amounts of airborne soil are likely to have only minor impacts on bleaching, eutrophication, and disease. The exception would have to be a severely virulent pathogen capable of better survival on dry soil subjected to high ultraviolet light levels than in a low light water medium. Nevertheless, these possibilities are not mutually exclusive, and few can be rejected outright at the present time.

Finally, a comprehensive data base needs to be put together linking all accurate field reports by trained observers as well as observations with photographs and video from amateur observers. Efforts to assure the collaboration of investigators working in different reef tracts of the Caribbean region are essential for acquisition of a comprehensive understanding of the impacts of coral reef diseases in large marine ecosystems. The broader our collaborative scientific groups are, the better we will be able to appreciate the incidence and impact of diseases within marine regions on a large scale. Only with progress on all four fronts (e.g., monitor-

ing, field sampling, laboratory experimentation, and construction of a database) in a simultaneous and integrated fashion will there be any hope to learn enough about the new diseases that there may be the possibility of devising steps to combat them. Until then no useful policy advice can be given regarding how to avoid or minimize damage to coral reef ecosystems.

The spread of coral reef diseases has become so commonplace, and with such intensity, that they have become the major cause of accelerating coral mortality in many locations and are likely to become far more prevalent in coming years. Immediate large scale research support to investigate these problems is essential for us to learn enough to make a difference before mortalities become catastrophic.

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RESUMEN

La variedad y frecuencia de organismos arrecifales enfermos ha crecido a través del Caribe durante los últimos 10 años. Las enfermedades de banda negra, banda blanca, punto blanco y plaga blanca se han esparcido más. La enfermedad de banda amarilla ahora afecta una mayor porción de *Montastrea annularis* a través del Caribe. El blanqueamiento y mortalidad de los tejidos afectados por la enfermedad de banda amarilla creció en el sur del Caribe a principios de 1997. Durante los dos años pasados, una mayor fracción de la alga roja calcárea *Porolithon* se murió a través del Caribe. Esta enfermedad, también encontrada en el oceáno Indico, no presenta la banda anaranjada característica de la enfermedad coralina letal naranja del Pacífico. Un borde blanco circular de un milímetro de grueso se observa entre el tejido vivo y el esqueleto muerto. Muchas otras enfermedades han emergido y atacado muchos otros organismos en este período incluyendo gorgónidos, esponjas y equinodermos. Muchas de estas ahora conocidas todavía no han sido descritas. Generalmente, la mayoría de enfermedades afectan una o pocas especies (excepto la plaga blanca y la banda negra), y sus distribuciones espacial y temporales no presentan relación alguna con el blanqueamiento, la contaminación (excepto la banda negra), la sedimentación ni el herbivorismo. Este manuscrito recopila observaciones realizadas independientemente por muchos investigadores a lo largo de un área extensa y un largo período de tiempo, con el propósito de evaluar la distribución geográfica e histórica de cada enfermedad. Se incluyen imágenes fotográficas para mostrar el criterio utilizado en el campo para reconocer cada enfermedad.

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