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The significance of emerging diseases in the tropical coral reef ecosystem

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Abstract: Novel pathologies of coral reef organisms, especially reef frame building scleractinian corals, have escalated during the decade between 1987 and 1997. These emerging diseases have appeared with progressively greater frequency and over wider distribution, and have revealed more diversified characteristics than ever before. The causes of most of these infections are not yet confirmed, but they evidence a gradual decline in the vital status of the coral reef ecosystem. As specific causes are identified for these afflictions, terminology will shift from non-specific descriptions, such as "white band", "white plague", "white pox", "yellow band" and "black band" diseases, to etiological and pathognomonic characterizations (e.g. aspergillosis and cyanobacteriosis). Stony corals are vulnerable to sedimentation, nutrient overloading, and chemical pollution from agricultural, urban, and domestic sources. They are incapable of relocation to other sites or of self-protection from cumulative effects of exposure to nitrates, phosphates, herbicides, pesticides, and raw sewage. In contrast to stresses attributed to warm water seasonal anomalies (e.g. coral reef bleaching), stresses imparted by pathogenic micro-organisms occur throughout the calendar year, fluctuate with changing temperature, and invariably result in tissue mortality. The coral has several mechanisms for defense. The epidermis, especially in tentacles of the coral polyp, contains nematocysts which are released in response to predators. The epidermal cells also possess cilia and a flagellary apparatus which are responsible for generating microcurrents in boundary water adjacent to the organism. These currents facilitate the entry of food into the coelenteron for digestion. Mesenterial filaments extend through the epidermis, sweep the surface of the colony, initiate digestion of food particles, and eventually return to the coelenteron. Both the epidermis and the gastrodermis contain mucocytes (or "immunocytes") which release a mucous secretion. That mucous blanket physically insulates the tissue from particulates or soluble toxins, and may also be bacteriostatic because of immunoglobulin (IgA). The recent emergence of diseases in corals may be interpreted as the consequence of (1) changing coastal ocean water quality favoring the proliferation, attachment and colonization of microbes, and (2) reduced efficiency of the coral's normal defenses. In order to appreciate these changes, research efforts to evaluate the microbial content of reef waters and to analyze the respective roles of mucus, cilia and flagella, and nematocysts of the corals are necessary. In this study, we have begun to detail the structural, physiological, chemical, and immunological attributes of the coral. Our analysis suggests that at least some of the emerging coral diseases may be explained by a decline in the capacity of coral colonies to mount effective protection against the increasing prevalence and varied invasive strategies of marine pathogens.

Key words: Coral diseases, emerging diseases, coral defense mechanisms.

The decades of the 1980s and 1990s have represented stressful times for coral reef organisms. During the 1980s mass coral reef bleaching was observed repeatedly throughout the tropical reef zone, in the Atlantic, Pacific and Indian Oceans, as well as in the Caribbean Sea (Goreau and Hayes 1994, Goreau *et al.* 1997, McGrath and Smith 1997). These episodes of coral reef bleaching coincided with the hottest period of the year in all sites observed (Goreau

et al. 1997). Moreover, the bleaching responses correlate precisely with extended periods of anomalously warm seas, perhaps combined with decreased wind intensity and increased light penetration into near shore waters.

In the 1990s, coral reef diseases have emerged to add to the stress upon reef communities (Cervino and Smith 1997; Antonius and Ballesteros 1997; Cervino *et al.* 1997; Goreau 1997). Some of these diseases have become epizootic in distribution. Significant numbers of colonies have been affected over a wide geographic range. Unfortunately, in contrast to the high potential for recovery of organisms from bleaching, many of the organisms afflicted by disease have died (Williams and Bunkley-Williams 1987, 1990). Mortality from coral reef diseases has the potential to be more detrimental to the survival of the coral reef than sedimentation, pollution, physical degradation, or all other threats combined.

In this review, we shall survey the defense mechanisms normally utilized by reef corals which are disabled in the course of illness. Any pathogen must physically penetrate or de-activate defenses in order to firmly establish its disease within the tissues of the reef organism. The recent emergence of multiple diseases and the rapid spread of these afflictions suggest both that the corals have lost the capacity to protect themselves and that the pathogens have adopted novel strategies or have acquired enhanced virulence in the coastal marine environment. It is possible that combined stresses resulting from repetitive episodes of bleaching, exposure to pollution, etc., might account for the increased incidence of diseases of reef organisms now being reported especially in the relatively confined Caribbean region.

The medical model of emerging diseases: Since 1980, numerous infectious diseases within human populations have increased in incidence, have assumed new hosts, and have appeared in a variety of new sites, paralleling our observations of the emergence of diseases in coral reef communities (Levins *et al.* 1994; Epstein 1995). The World Health Organization has documented over 30 emerging diseases afflicting human populations (LeDuc and Tikhomirov 1994). Moreover, this scourge of infectious diseases has continued to mount in new sites and to new hosts into the 1990s. Of concern in the public health field is the possibility that the changing profile of these diseases is being promoted by climate changes (Wilson *et al.* 1994). With extreme shifts in climate, the spread of diseases into new environments, onto new hosts, and/or among new susceptible populations has been documented. Changes in temperature ranges, rainfall, or drought conditions not only alter microbial distributions, but may shift the distribution or prevalence of intermediate vectors such as insects or rodents.

Examples of emerging infectious diseases of humans include Leptospirosis, a bacterial disease characterized by fever and internal bleeding. This disease affected over 2500 individuals in Nicaragua in October, 1995. Also included is Equine encephalitis, a viral disease that infected over 3,000 people in Columbia and resulted in inflammatory swelling of the brains of these patients. Cholera, a bacterial disease which had not been recorded for over 100 years in Latin America, appeared in South and Central America during 1991-92 (Epstein 1993; Epstein *et al.* 1993; Colwell 1996). Peru recorded over 300,000 cases, with additional cases in Equador, Colombia, Brazil, Mexico, Venezuela, El Salvador, and Honduras. Diarrhea and extreme loss of fluids and electrolytes are common signs of cholera and over 1.0% of patients afflicted die. Other diseases/pathogens on the WHO list are Dengue fever (especially the hemorrhage form), Ebola virus, Cryptosporidium, Hanta virus, Onchocerciasis, Human immuno-deficiency virus (HIV), Legionnaries' disease, Malaria, Hepatitis, and multidrug-resistant Tuberculosis.

There is a possibility that latent forms of pathogenic micro-organisms may be transported into the coastal ocean along with soil residues during fresh water runoffs (Epstein *et al.* 1994). These microbes may shift distribution from terrestrial sites into riverine systems and from there into coastal oceans and into

TABLE 1

Selected emerging diseases affecting coral reef organisms of Florida and the Caribbean

Disease	Host	Pathogen	Reference
Black band	Many coral species	*Phormidium corallyticum* and consortium incl. *Beggiatoa* sp.	Antonius 1973 Richardson *et al.* 1997 Carlton and Richardson 1995 Kuta and Richardson 1996
Coralline algae lethality	*Porolithon* sp.	bacterium ?	Littler and Littler 1995 Goreau unpublished
Dark blotch	*Montastrea cavernosa*	?	Peters 1997
White pox	*Acropora palmata*	?	Porter and Meier 1992
Red band	*Siderastrea* sp. *Porites* sp. *Montastrea* sp. *Diploria* sp.	*Oscillatoria* sp.	Richardson 1992
Sea fan aspergillosis	*Gorgonia ventalina* and *G. flabellum*	*Aspergillus* sp.	Nagelkerken *et al.* 1996 Smith and Ritchie 1997
White band	*Acropora palmata* and *A. cervicornis*	*Vibrio* sp.?	Peters 1983 Dustan 1977 Ritchie and Smith 1995
Rapid wasting	*Montastrea* sp. *Colpophyllia* sp.	fungus	Cervino *et al.* 1997Cervino and Smith 1997
Coral plague	*Acropora* sp. *Dichocoenia stokesi*	? *Sphingomonas sp.*	Dustan 1977, Peters 1983 Richardson *et al.* 1997
Yellow band	*Montastrea* sp.	?	Quirolo, unpublished

contiguous ecosystems, such as mangroves, sea grass beds and coral reefs.

Emerging caribbean coral diseases: In Table 1, a summary of the various recently documented or newly described diseases reported in coral reef organisms is shown. Each of these diseases has now been reported in the literature, as indicated, and may be further distributed than was appreciated at the time of initial documentation. The genera affected as well as the reef tracts involved have not been fully determined. Based upon information currently available concerning the spread of these diseases, most if not all of them are infectious and are probably transmitted by a pathogen or combination of pathogens, as yet unidentified.

Black band disease was one of the first afflictions of stony corals described (Antonius 1973) and many documentations of Black band disease now exist in the literature (e.g. Richardson and Carlson 1993; Kuta and Richardson 1996). Black band disease is now recognized as epizootic in the Caribbean. Richardson and co-workers (Carlton and Richardson 1995; Richardson 1996; Richardson 1997; Richardson *et al.*, 1997) have described the consortium of organisms thought responsible for black band. The cyanobacterium, *P. corallyticum*, is the blue-green algal component, but the sulfite-oxidizing bacterium, *Beggiatoa* spp., the sulfate-reducing bacterium, *Desulfovibrio* spp., and other bacteria and fungi are essential components of the consortium (Richardson 1996, 1997).

White band disease, originally reported by Gladfelter *et al.* (1977), is now also widespread throughout the Caribbean (Ritchie and Smith

1997). This affliction was originally described by Dustan (1977) and by Peters *et al.* (1983). Plague also is widespread and affects several coral species. Recent data indicate that plague is attributable to the bacterial genus, *Sphingomonas* (Richardson *et al.*, 1997). Red band is another cyano-bacterial infection of corals. This disease is caused by *Oscillatoria* spp., according to Richardson (1992). Yellow band disease was initially recognized by Quirolo (1994, unpublished) and is commonly reported from many sites in the Caribbean region.

Rapid wasting disease is the most recent of the afflictions of stony corals. Discovered within the last year in the Netherlands Antilles, Rapid wasting disease has been closely followed and reported by Cervino and coworkers (1997). This disease has been observed primarily on *M. annularis* and *C. natans*. It spreads very quickly over those colonies affected and leads not only to death of the coral tissues but also to erosion of the coral skeleton.

Several other coral reef infections are also recognized, including sea fan disease, now attributed to a species of the soil fungus, *Aspergillus* spp. (Ritchie and Smith 1996; Smith *et al.* 1997). Porter and Meier (1992) have reported another coral affliction, White pox, in the Florida keys. Recognition of as well as definitive descriptions for most of these concerns were not available before 1990. All of these problems in the coral reef community deserve further scrutiny and definition in order to complete our understanding as to the genera and species most affected, the range of influence both within the reef tract and among tracts throughout the region, the rate of spread of the disease, and the prognosis for affected colonies.

However, the most important information outstanding about all of these concerns the etiology of the diseases and their differential diagnostic features. It is important that researchers continue to analyze these diseases to determine if in fact they are microbial diseases, as opposed to stress responses or genetic variations. For example, if no pathogen is identified, Yellow band disease might better be classified as a stress response similar to coral bleaching. Also, several contributing microbes might be required to produce the pathology. Richardson and co-workers (1997) have found that Black band disease depends upon a specific admixture (consortium) of microbes. The circumstances under which microbial consortia or single micro-organisms display pathogenicity toward hosts in the reef community require elucidation. As researchers establish which virus, bacterium, fungus, or which specific combination of pathogens cause observed infections, those diseases should be re-named to indicate the specific organisms implicated as etiological factors.

Coral defense mechanisms: A complete appreciation of emerging diseases in reef corals requires an understanding of the defenses which corals normally utilize to protect themselves against disease. Basically, there are four lines of defense at the tissue-environment interface of the scleractinian coral. These are: (1) mucus, (2) mesenterial filaments, (3) cilia, and (4) nematocysts. Instead of the possibilities that any one microbe may have mutated into a pathogen or that a new combination of microbes accounts for pathogenicity, it is also possible that the defense mechanisms which the coral normally depends upon for protection may have become exhausted or impaired, thus allowing latent or subliminal (sub-threshold) endemic infections to erupt and spread.

Mucus production by the coral is an important and under-appreciated aspect of coral defense (Jackowska 1966; Hillman 1978; Jackowska *et al.* 1978; Santavy and Peters 1997). The consistency, quantity and chemical composition of mucus varies according to the physiological status of the organism. The mucous layer varies in thickness, but is easily identified in microscopic sections (Fig. 1A). With inflammation, the discharge of mucus increases and the blanket thickens to protect the epithelium. This mucous blanket stains positively with Periodic acid-Schiff reagent, is basophilic, and is metachromatic.

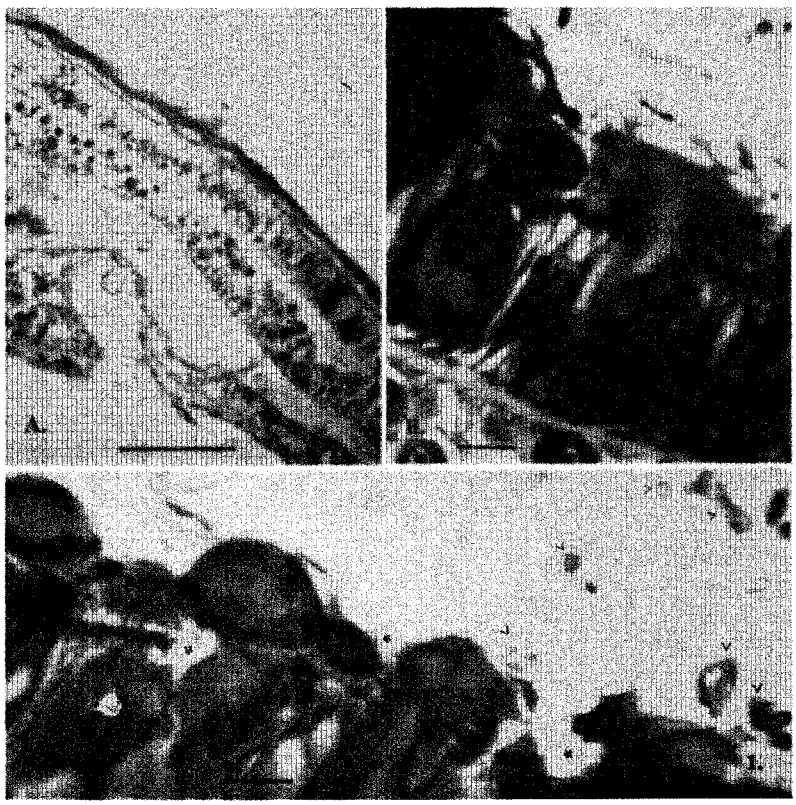


Fig. 1. (A) Light micrographic section of *Montastrea annularis* showing the coral epidermis, mesoglea, and gastrodermis, with coelenteron and calicoblast. The aragonitic skeleton has been removed, but normally would appear in the lower left portion of the field. Note the intact and continuous thin mucus layer over the epidermis in the right upper field. Marker = 0.1mm.; (B) Light micrographic section of *M. annularis* showing the appearance of the mucus layer in Rapid wasting disease. The mesoglea separates the epidermis from the zooxanthellate gastrodermis. Marker = 0.01mm.; (C) Another section of *M. annularis* with Rapid wasting disease, showing fungal hyphae (^) adjacent to the epidermis. Marker = 0.01mm. In both (B) and (C) the mucous layer, instead of being integrated into a continuous thin film over the coral epidermis, is eroded and unconsolidated, appearing as mucus pitting (*) and as individual globules. These abnormal features indicate the breakdown of the usually protective mucous barrier over the coral epithelium. Plastic embedded sections stained with Toluidine Blue 10B.

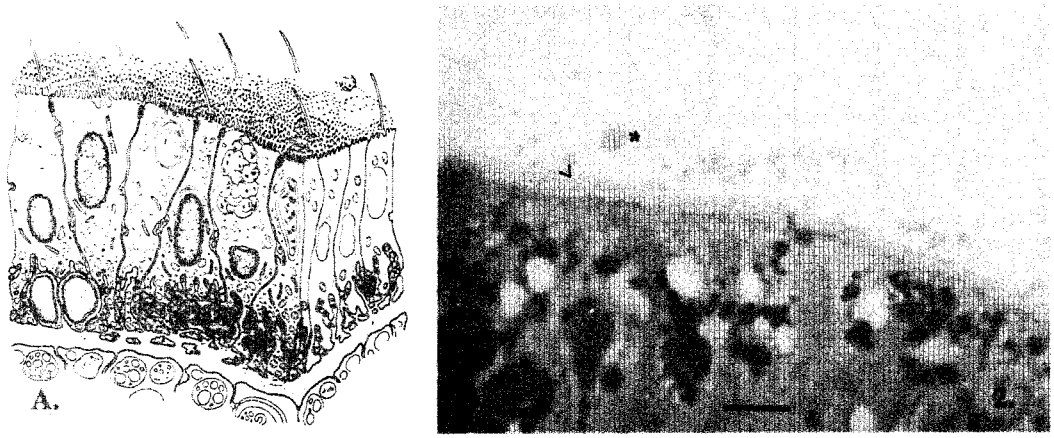


Fig. 2. (A) Diagram of coral planula of *Porites porites* showing microvilli and flagellae of the epidermis. Original diagram by R. Hayes (Hayes and Goreau, 1977); (B) Section of *P. porites* planular epidermis stained with potassium permanganate to show microvilli (^) and flagellae (*). Marker = 0.01mm.

Mucous secretions of the coral are lubricating, protective and osmo-regulatory. When released in response to inflammation, the mucins may impart immunological properties. A secretory molecule in mucin, immunoglobulin A, is known to be produced in Cnidarians (Tomasi and Grey 1972). A large molecular weight unit (ca. 300kDa), IgA dimers bind to antigenic molecules, providing immunological protection against toxins (Tomasi and Plaut 1985). Also, mucus from tropical marine organisms is reported to contain molecules of pharmacological and antibiotic significance (Jakowska 1977). Although evidence exists to suggest that the mucous layer of the coral epidermis is altered both in quantity as well as quality after bleaching stress or following infection, the molecular chemistry of coral mucus is incompletely appreciated and deserves further research to establish its role in disease.

If the coral mucus were to break down, either by erosion or fragmentation, the usual physical as well as chemical protections offered by the discharge would be lost. When this mucous barrier is disrupted, pathogens are able to colonize the epithelial surface as well as to invade the tissue and/or release toxic secretions into deeper tissues. The more virulent the pathogen, the more rapid the attachment, proliferation and toxic influences upon host tissues. Loss of the integrity of a mucous covering allows a pathogen to evade one of the most effective defense mechanisms of the coral (Santavy and Peters, 1996).

From histological images of sectioned coral tissues affected by Rapid wasting disease, it is clear that the mucous blanket of the coral does not form a complete barrier to infection. Figs. 1B and 1C show the appearance of the epidermal mucus in this disease. Not only does the mucus appear to be pitted or dissolved, but discharges from individual mucocytes have failed to coalesce into an intact layer. Failure of the mucus to coalesce into an integrated sheet exposes epithelial cell surfaces directly to potential pathogens. Acidic discharges from the fungal mycelium may be disrupting the consolidation of the mucous layer, allowing fungal hyphae to penetrate through the mucus to reach direct contact with the epithelial surface. In succession, the epidermal cells are dissociated, the mesoglea is dissolved, and the gastrodermal cells are released from their organization into an epithelium. Finally, there is evidence that the aragonitic skeleton of the coral is etched as the progress of this infection spreads across the colonial surface to the calcifying basal epidermis (calicoblast). The failure of the mucous blanket to envelope the coral surface suggests that its molecular chemistry is modified, its physical properties are altered, and its effectiveness as a barrier to disease is lost. Identification of a fungal component associated with Rapid wasting disease of corals emphasizes the significance of mucus in this marine pathology. By comparison, opportunistic fungi are also a component of HIV infection. Fungal illnesses in humans tend to occur in unusually warm, moist environments. Epidermal injuries or loss of mucosal integrity offer a portal of entry for pathogens to enter vulnerable areas of the human body (e.g. oral cavity, respiratory tract, urogenital tracts).

Mesenterial filaments are duplications of the gastrodermal epithelium of the coral which protrude through the oral aperture and also through the epidermis. These filaments sweep across the surface of the parent coral colony and also extend beyond the border of the colony to adjacent organisms (e.g. sponges, bryozoans, and calcareous algae) or to other coral colonies. This activity may be more pronounced at night or during periods of low tidal flow. The mesenterial filaments are important for protecting the coral against invasion and overgrowth by neighboring organisms such as sponges, calcareous algae and other corals. However, these digestive enzyme-releasing filaments also would retard the colonization of microbial agents that might settle onto the surface mucous blanket or that might be ingested into the coral gastrocoele. Proteolysis of microbial membranes would effectively control the proliferation of microbial flora and also might provide a food source for the coral.

The epidermal cells of the coral possess cilia on their external border. Several hundred cilia may occupy the surface of the cell, extending its surface area and providing for water movement along the cell surface. These cilia are fingerlike projections of the cell membrane (Figs. 2A and 2B). They are bound together by an extracellular glycocalyx and by mucous strands. A filamentous core fills the axis of the cilium and by anchoring into a terminal web of filaments in the apical cytoplasm, motility of the ciliary assemblage is possible. These cilia generate micro-currents in boundary water around the coral, thereby mobilizing small particles and dissolved substances that might approach the surface of the cell. Interspersed among the cilia and tethered into functional union with them are contractile flagellae which contribute to the movement of water adjacent to the coral (Hayes and Goreau 1977). The movement of the ciliary assembly, enhanced by whip-like contractions of flagella, requires energy through the utilization of ATP. If energy resources of the coral were depleted by prior episodes of bleaching and nutrient deprivation, another of the normal defenses of the coral would be rendered ineffectual.

The nematocyst is the product of the nematocyte or cnidocyte which is a cellular constituent of the coral epidermis. These cells are particularly concentrated within the tentacles of the coral and include a coiled stinging organelle within a membrane bound vesicle (Fig. 3A). The nematocyst release is triggered by tactile stimuli and is most effective as a defense against large particulates. This barbed structure is particularly effective for penetration of predators. Ciliates and other copepods contacting the coral surface stimulate the release of the stinging structure which physically impales the foreign object (Fig. 3B). Were this mechanism inactivated through insensitivity there would be loss of another essential defense of the coral.

The breakdown of one, a combination of two or more, or all of these multiple defense mechanisms accompanies each of the pathologies which are now being reported in reef corals. Although the microscopic sequence of events has not been documented for most coral diseases, additional research would most likely extend our understanding of the mechanism of action of putative pathogens and would reveal the capacity of corals to recover from these infections.

Comparison of emerging coral reef and human diseases: The concept of resurgent or emerging diseases in coral reef organisms is based upon the realization that infections are newly appearing or are rapidly increasing in incidence or expanding in geographical range. There are basically three ways an infectious disease might be considered emergent: (1) because it is a new appearance, (2) because it had appeared at an earlier time infrequently or without recognition, but had recently increased in frequency and potential significance as a local occurrence, or (3) because it had been recognized as a disease earlier but had recently appeared to expand into other reef zones, into remote reef tracts, or onto new hosts.

Appreciation of emergent disease problems in the tropical reef community requires confirmation that each of these diseases is properly ascribed either to a single marine pathogen (*e.g.* aspergillosis, or sea fan disease) or to a consortium of microbial organisms (*e.g.* cyanobacteriosis, blue-green algae complexed with sulfur-reducing bacteria and with nitrifying bacteria as in black band). Koch's postulates must be confirmed in order to differentiate the opportunistic organisms which colonize a diseased tissue from those which represent the etiological agents for the disease. That is, an organism must be isolated from a site of infection, cultured in axenic form, re-infect an uninfected specimen, and be recoverable and identified from the infection site. The satisfaction of these criteria not only establishes the infectious diseases as a disease, but allows us to dispense with descriptive terminology by ascribing labels that are diagnostic for the pathology.

There are significant contrasts between the medical model of disease and the emergence of infectious diseases in the coral reef community. First, the human model is confined to a single

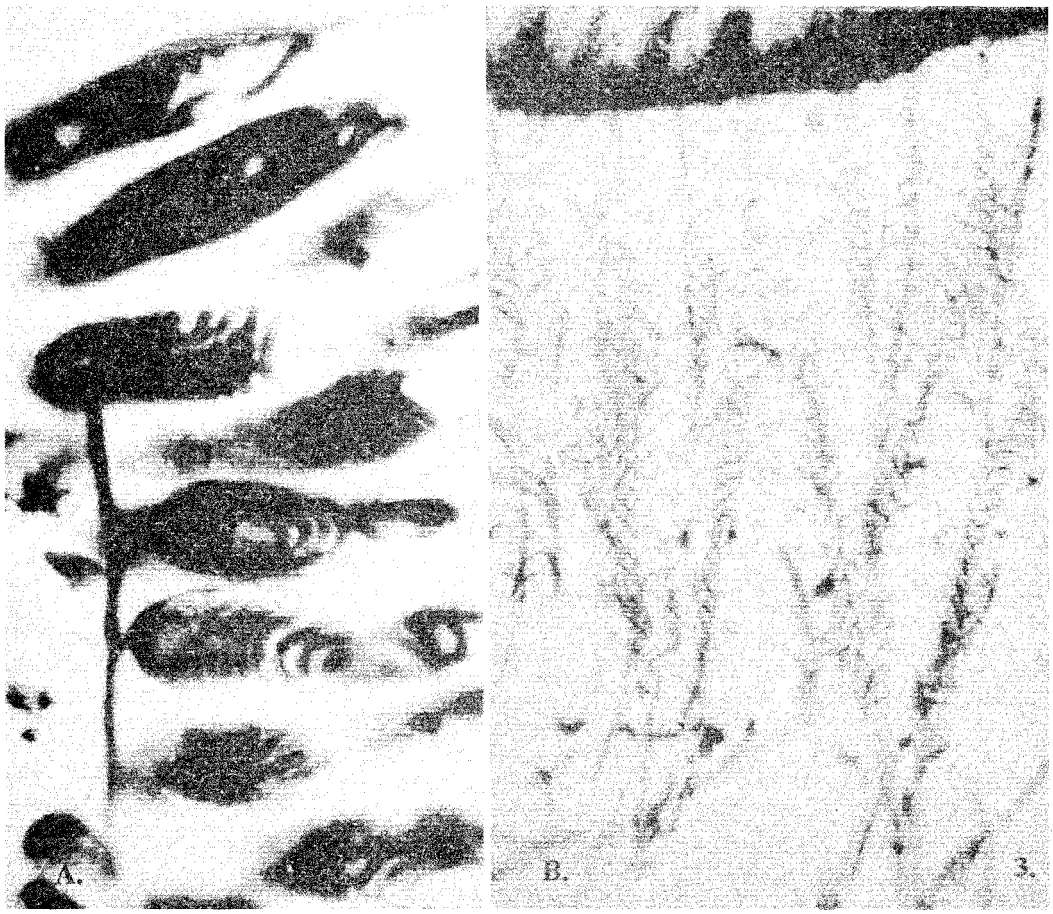


Fig. 3. Coral nematocysts in situ. (A) Light micrograph showing en face view of nematocysts in whole mount of coral tissues; (B) Barbed nematocysts released from vesicles in the coral epidermis. Photos by T.F. Goreau.

species, *Homo sapiens*. Regardless of who the person is or where the person is located, a disease presents similarly and is easy to identify by trained personnel.

When epidemics appear, they are easy to recognize because all affected individuals share the same signs and symptoms. The coral disease picture is complicated by virtue of the multiplicity of genera and species likely to be affected by the same microbe. The appearance of the disease in the coral reef environment may also vary according to depth as the morphology of the colonial organism varies. The possibility that a single disease appears in many forms leads to misinterpretation, duplicity, and confusion. The marine disease must be identified by objective "signs" or appearances only, since "symptoms" or subjective feelings of discomfort are impossible to ascertain.

The sea can restore itself over time, but just as with the human model, recovery or restoration may be protracted in the event of an epizootic which presents an overwhelming burden of pollutants or pathogens. During the critical period of an epizootic, many marine organisms succumb to the influences of the cumulative stress and assorted pathologies. Significant mortality within the coral reef population follows such events. In the coral reef community, high levels of selective mortality imbalance those inter-relationships upon which the integrity of the ecosystem depends.

Secondly, medical expertise is available within a large pool of health care professionals, all of whom are trained to recognize diseases. The medical professionals responsible for identification and differential diagnosis offer reliability to any database developed. In contrast, coral reef researchers vary in their awareness and ability to identify coral diseases. Not only must training of marine scientists establish this skill, but textbooks and other resources must be updated to document the critical information required by the professional.

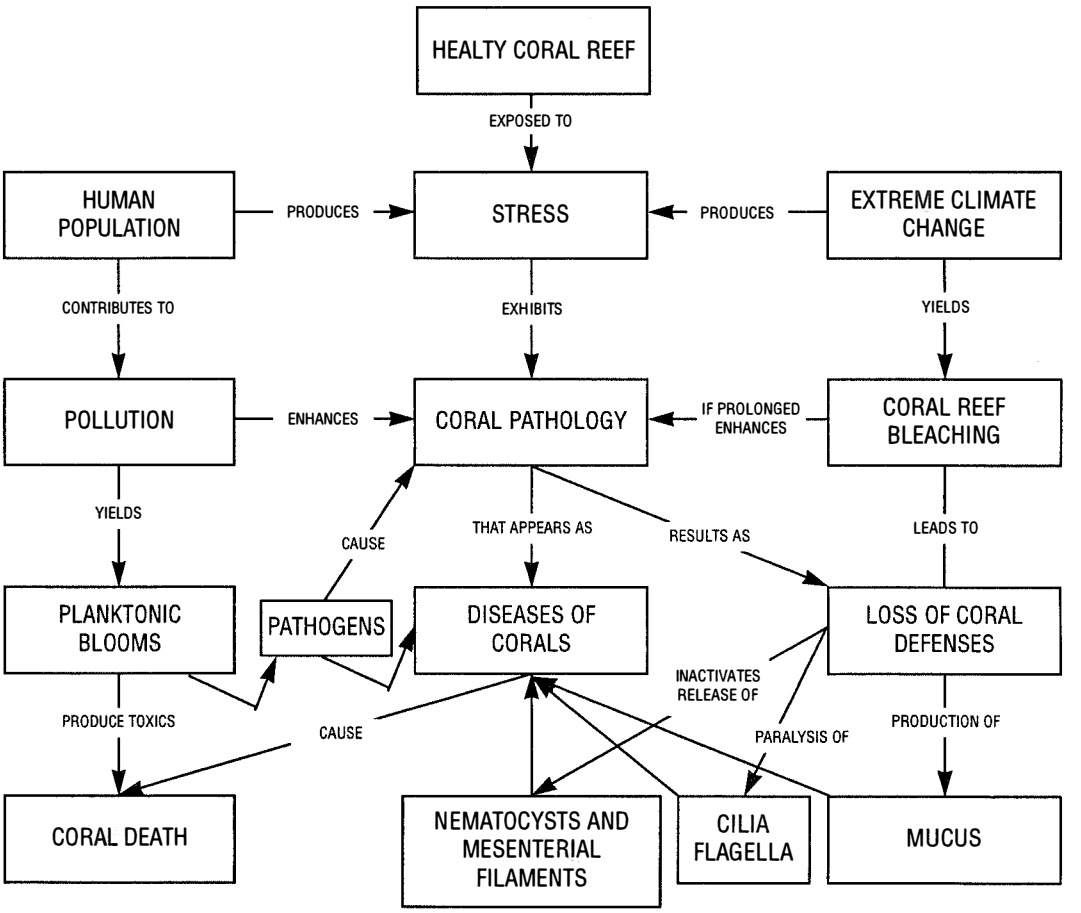
Third, the medical information network is vast and frequently utilized by physicians and public health professionals. Rapid reporting and comparisons are available to track the outbreak of diseases. The Center for Disease Control and Prevention is available to mobilize expert teams throughout the world without delay (Bryan et al. 1994). The WHO offers real time data exchange and periodic alerts to all personnel (leDuc and Tikhomirov 1994). The coral reef researcher often works alone in remote areas and with inadequate resources for accessing other data than are locally available. Electronic communication lines are improving among reef scientists. These are not regionally reliable in real time and are not utilized either as repositories of information or sources of comparative data. Electronic information transfer is now beginning to link the global marine scientific community, but it needs to become an active tool for real time communication of local or regional observations.

Fourth, the critical nature of epidemics of human disease has led to an attitude of open transfer and exchange of information. In the academic environment, including coral reef research, information is considered proprietary until published. After all, research grant awards, institutional contract renewal, and academic promotion and tenure are all dependent upon being the principal investigator or preparing the primary reference on an important topic in the field of marine sciences. This culture of possessiveness about observational research data must be radically changed before a new liberal, free exchange of data on coral reef diseases emerges. Individual research findings must be shared rapidly and reliably in order that a global perspective of diseases and stress responses might be appreciated. This is necessary to facilitate the flow of data about threats to the coral reef environment and for prompt documentation of environmental degradation, regardless of the source of such insults.

In essence, there is one global ocean and containment of disease within the marine environment is not easily attained. Human disease control may be effectively managed by applying containment strategies at the continental, national, regional or local level. Such geographic boundaries are not easily drawn for oceanic environments. A problem which develops in one coastal zone will rapidly progress to encompass all portions of that zone. Upstream or downstream effects will be transferred rapidly by water movement. Therefore, progression from a local to an epizootic phenomenon occurs very rapidly and completely. A concept map of the local conversion of a healthy coral reef into a stressed, diseased or dead reef is presented in Table 2. According to this conceptual map, major stresses upon the coral reef ecosystem emanate from human activity and from climate change. Human activities are most likely to pollute the reef through the addition of nutrients or chemicals to the water column; climate changes, such as global warming, induce stress responses which, among other things, may uncouple symbiotic relationships among reef organisms (bleaching response). Pollutants in the environment shift growth and reproductive dynamics on the reef, favoring algal overgrowth and microbial proliferation (blooms). Following bleaching, the reef coral is unable to calcify a skeleton, to sexually reproduce, to obtain adequate nourishment, and to exhibit normal energetics related to metabolism and self sustainment. These losses become more critical with prolongation of bleaching and may contribute to the loss of normal mechanisms of defense against predation. Disease in coral reef organisms is the expression of both a breakdown in water quality and a deterioration in available protective strategies. The loss of nor-

TABLE 2

Concept map showing inter-linking pathways for events or factors that would convert a healthy coral community into a stressed, diseased, and dying reef



mal protections imparted by the activity of cilia and mesenterial filaments as well as the physico-chemical changes in mucoid secretions establishes both necessary and sufficient conditions for the expression of the various diseases observed. The ultimate outcome of disease within the community is the death of the affected organisms.

Coral reef diseases must be identified by etiology and differentiated by specific features or signs. The pathognomonics and prognoses for each disease must be determined. Each disease must be described by incidence, including genera and species affected, geographical distribution, and zonation across the reef and within surrounding waters. In addition to these descriptive refinements of coral reef pathology, essential complementary efforts must include:

1. **Research** directed toward elucidation of the specific causes of the malady and options for controlling the spread of diseases.
2. **Management strategies** developed for rapid isolation and removal of diseased individuals or colonies to provide protection for other reef constituents against the spread of disease.
3. **Monitoring** directed toward determining incidence and distribution of each disease.

Finally, preventive measures should be emphasized in the education of stakeholders who utilize or depend upon tropical coastal resources so as to avert the appearance of disease within the reef community.

4. **Preventive measures** developed to minimize the spread of pathology within the coral reef community.
5. **Further research commitments** enhanced to promote our understanding of causal factors that stress coastal tropical marine ecosystems and that offer advantages to potential pathogens which spread disease into coral reef environments. Research funding must be prioritized for this effort and rapid, liberal, and global exchange of data must become readily accessible.

RESUMEN

Patologías nuevas de organismos arrecifales, especialmente de corales constructores de arrecifes, han escalado durante la década entre 1987 y 1997. Estas enfermedades emergentes han aparecido con progresiva mayor frecuencia, distribuciones más amplias, y con características más diversas que antes. La causa de la mayoría de estas infecciones no se ha confirmado todavía, pero han resultado en una degradación gradual de los ecosistemas arrecifales. Conforme se identifican causas específicas de estas enfermedades, la terminología va a cambiar de descripciones no-específicas, t les como "banda blanca", "plaga blanca", "lunares blancos", "banda amarilla" y "banda negra", a caracterizaciones etiológicas y patognomónicas (e.g. aspergillosis y cianobacteriosis). Los corales pétreos son vulnerables a la sedimentación, altas cargas de nutrimentos y contaminación química de fuentes agrícolas, urbanas y domésticas. No pueden desplazarse a otro lugar o protegerse de efectos acumulativos por exposición a nitratos, fosfatos, hierbicidas, plaguicidas y aguas negras. En contraste a impactos atribuidos a anomalías estacionales de aguas calientes (e.g. blanqueamiento de corales), estreses impartidos por microorganismos patógenos ocurre todo el año, fluctuando con los cambios en temperatura, e invariablemente resultando en la muerte de tejido. Los corales tienen varios mecanismos de defensa. La epidermis, especialmente en los tentáculos, contienen nematocistos que son liberados en respuesta a depredadores. Las células epidérmicas también poseen cilios y un aparato flagelar que son responsables de generar microcorrientes en las aguas adyacentes del animal. Estas corrientes facilitan la entrada de alimento al celenterón para su digestión. Filamentos mesentéricos se extienden a través de la epidermis limpiando la superficie de la colonia, iniciando la digestión de partículas alimentarias y eventualmente regresando al celenterón. Tanto la epidermis como la gastrodermis contienen mucocitos (o immunocitos) que liberan una secreción mucosa. Esa sábana mucosa físicamente aisla el tejido de partículas o toxinas solubles, y puede ser bacteriostático por la presencia de immunoglobulina (IgA). La aparición reciente de enfermedades en corales se puede interpretar como la consecuencia de (1) ca bios en la calidad de las aguas costeras favoreciendo al proliferación, asentamiento y colonización de microbios, y (2) reducción en la eficiencia de defensa de los corales. Para poder apreciar estos cambios, esfuerzos de investigación deben evaluar el contenido microbial de las aguas arrecifales y analizar los roles respectivos del mucus, cilios y flagelos, y nematocistos de corales. En este estudio, hemos empezado a detallar la estructura, fisiología, química y atributos immunológicos de los corales. Nuestro análisis sugiere que al menos algunas de las enfermedades emergentes se pueden explicar por una caída en la capacidad de las colonias de coral de montar protecciones efectivas contra la prevalencia creciente y variada de estrategias invasivas de p tógenos marinos.

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