Coral diseases: what is really known?

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Reports of new and emerging coral diseases have proliferated in recent years. Such coral diseases are often cited as contributing to coral reef decline. Many of these diseases, however, have been described solely on the basis of field characteristics, and in some instances there is disagreement as to whether an observed coral condition is actually a disease. A disease pathogen has been identified for only three coral diseases, and for only two of these has the pathogen been shown (in the laboratory) to be the disease agent. In one case, the same disease name has been used for several widely varying coral syndromes, whereas in another multiple disease names have been applied to symptoms that may be caused by a single disease. Despite the current confusion, rapid progress is being made.

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During the International Year of the Reef (1997) and continuing into the International Year of the Ocean (1998), much attention and activity have been focused on evaluating the current status of coral reefs[1–6]. The uniform conclusion of these multiple assessments is that coral reef ecosystems are degrading, and that this is most likely a combined result of global warming, ozone depletion, overfishing, eutrophication, poor land-use practices and other manifestations of human activities. All of these reports have emphasized that an increase in coral disease is contributing to reef degradation, a conclusion based largely on an observed increase in the numbers of diseased coral colonies. Recent rigorous studies of coral disease at both the mechanism and ecosystem levels are finally beginning to provide information that will define the role of coral disease in reef decline.

The first coral diseases

Investigations into the nature of coral diseases are relatively recent, with the first descriptive reports appearing in the 1970s of coral tissue degradation occurring on stony, reef-building corals (scleractinians). Two disease patterns were recognized in these initial studies, which were characterized by either a sharply demarcated interface between healthy and unhealthy coral tissue or a distinctive band (mm to cm wide). Each moved across coral colonies while completely destroying coral tissue. The first coral disease reported (in 1973) – 'black band' disease[7] – consisted of a dark band that was present between apparently healthy coral tissue and freshly exposed coral skeleton (Fig. 1). The second two coral diseases reported (in 1977) –'white band' disease of branching acroporid corals[8] and 'plague' (Fig. 2) of massive and plate-forming corals[9] – each appeared as a sharp boundary between coral tissue and exposed skeleton, with no apparent microbial biomass. All three diseases progressively destroyed coral tissue at rates of several millimeters per day.

Coral disease pathogens: early attempts at characterization

In the 1970s limited efforts were made to identify coral disease pathogens. These efforts routinely consisted of microscopic observations of diseased tissue, all of which revealed the presence of various bacteria. Heterotrophic and phototrophic (for black band disease) bacteria were observed, and both photosynthetic and heterotrophic bacteria were proposed as potential agents of coral disease[7,9–11]. There were no reports of attempts to apply the established methods of medical microbiology (Koch's postulates, Box 1) to coral diseases.

Research in the late 1970s and early 1980s followed the same descriptive approach. New results, however, included the first quantitative study of disease prevalence, and it was determined that white band disease was relentlessly eliminating the important reef-forming acroporid corals of shallow Caribbean waters[13]. This was the first case in which the potential severity of coral diseases was verified.

Further efforts were made to identify pathogens associated with specific diseases, and additional causative agents were proposed[14–16]. For black band disease, the postulated primary pathogens included one species of cyanobacteria *Oscillatoria submembranaceae*[16] (renamed *Phormidium corallyticum*[17]) and a marine fungus[18], in addition to the sulfate-reducing bacteria (presumed to be of the genus *Desulfovibrio*[11]) and sulfide-oxidizing bacteria (*Beggiatoa* spp.[10]) noted in the 1970s. Most of the studies included the observation that all of these microorganisms were present within black band. Black band disease was also newly reported to infect gorgonians[19,20] (soft corals), in addition to the scleractinian, or 'stony', corals targeted in all previous studies, and was for the first time observed on reefs of the Indo-Pacific[21] and the Red Sea[22], thus widely expanding the previously known range of black band disease (in the Caribbean and Western Atlantic).

A new, histological approach was applied to the study of white band disease by Peters *et al.*[23] in 1983. Their work revealed 'packets' of gram negative bacteria associated with white-band-diseased tissue (as well as healthy) from colonies of *Acropora palmata* at two sites in the Caribbean[23]. Within five years up to 95% of all colonies on these study reefs died. Laboratory culturing and isolation attempts yielded inconsistent results and no specific pathogen was postulated.

Dustan's report[9] on plague in 1977 documented tissue loss rates of up to 3.1 mm per day, with mortality of individual colonies occurring within four months. Microscopic observation revealed the presence of gram-negative unicellular and flexi-bacteria in diseased tissue, but no attempts were made to culture or isolate potential pathogens.

Two additional coral afflictions were also reported in the 1980s. The first was an extremely rapid pattern of tissue loss on scleractinian corals, which affected an entire colony at once (termed 'shut down reaction')[16]. Although no microbiological studies were conducted, exposure of healthy corals to sloughed-off, necrotic coral tissue elicited the same symptoms. The second report described tissue hyperplasia of gorgonian corals associated with nodules that contained the filamentous green alga *Entocladia endozoica*[24].

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Neither tissue degradation nor colony mortality occurred, however, and infected gorgonians recovered by encapsulating affected areas[24]. Currently, this syndrome is not considered to be a disease (W.M. Goldberg, pers. commun.).

Therefore, by 1984, four tissue-degrading coral diseases (plus the algal nodules of gorgonians) had been studied at a descriptive level: black band, white band, plague and shut down reaction. Of the four, the identity of only one primary causative pathogen had been proposed – the cyanobacterium *P. corallyticum* found in black band disease[17]. This was proposed even though there was no success in obtaining an axenic (bacteria-free) culture and the disease state could only be initiated by inoculation using the cyanobacterium with other associated black-band-disease microbes. Despite the fact that Koch's postulates were not fulfilled (Box 1), it was accepted for many years that the black-band disease agent was known.

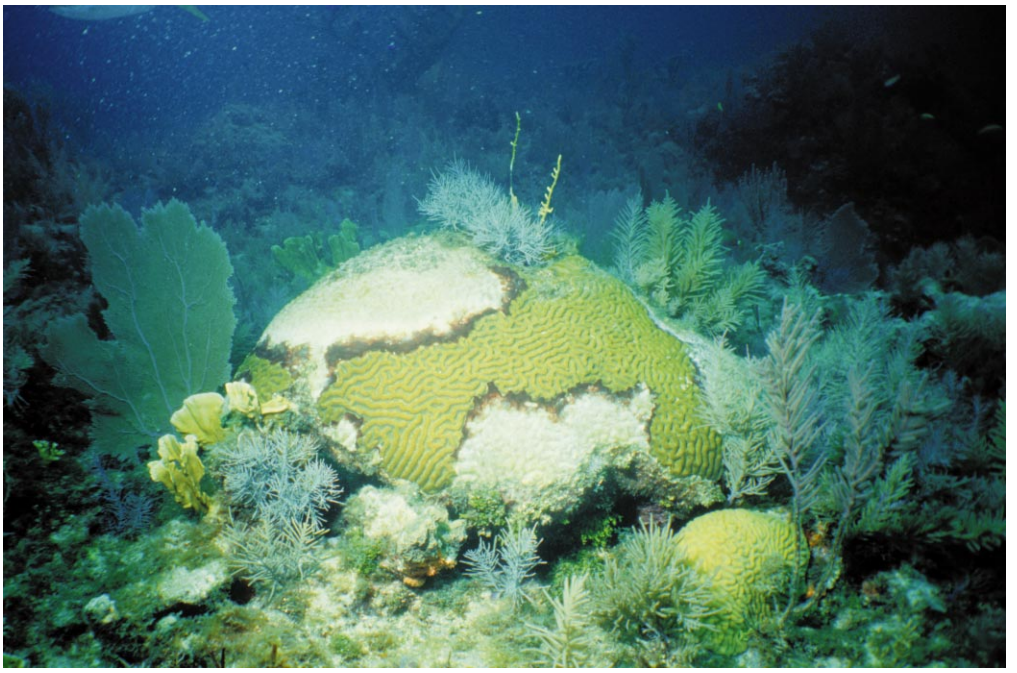

Fig. 1. Black band disease on *Colpophyllia natans*. Two active infections are present, revealing the characteristic dark band separating healthy coral tissue and exposed coral skeleton (exposed as the result of coral tissue degradation during the disease process). The band is dark because of high concentrations of the red pigment phycoerythrin, a light-harvesting pigment found in the dominant member of the black band community (the cyanobacterium *Phormidium corallyticum*). The disease is found on reefs world-wide and targets nonacroporid scleractinian corals. Disease progression is at a rate of 0.3–1 cm per day, and can kill a coral colony over several months. The sulfide-oxidizing bacterium *Beggiatoa* spp., another member of the black band community, is often present on the band surface as sulfide accumulates within the band. Populations of this genus appear white, the result of intracellular accumulation of highly refractile elemental sulfur (a by-product of the sulfide oxidation). The black band community is now known to consist of a microbial community similar to those found in other illuminated aquatic environments that contain steep gradients of oxygen and sulfide.

Characterization of coral diseases in the 1990s

Research progress has accelerated in the 1990s as investigators have become interested in specific aspects of coral disease etiology.

White band disease. Recently, Ritchie and Smith[25] have demonstrated that there are two patterns of tissue-loss associated with white band disease, which they have termed white band type I and white band type II. Type I exhibits a disease line of active tissue necrosis, whereas type II has a variable zone between active tissue death and exposed coral skeleton, where coral tissue can be bleached but not necrotic. Differentiation between the two types requires close scrutiny over time, because the bleached tissue area of type II at times 'catches up' with the necrotic zone, thus appearing identical to type I.

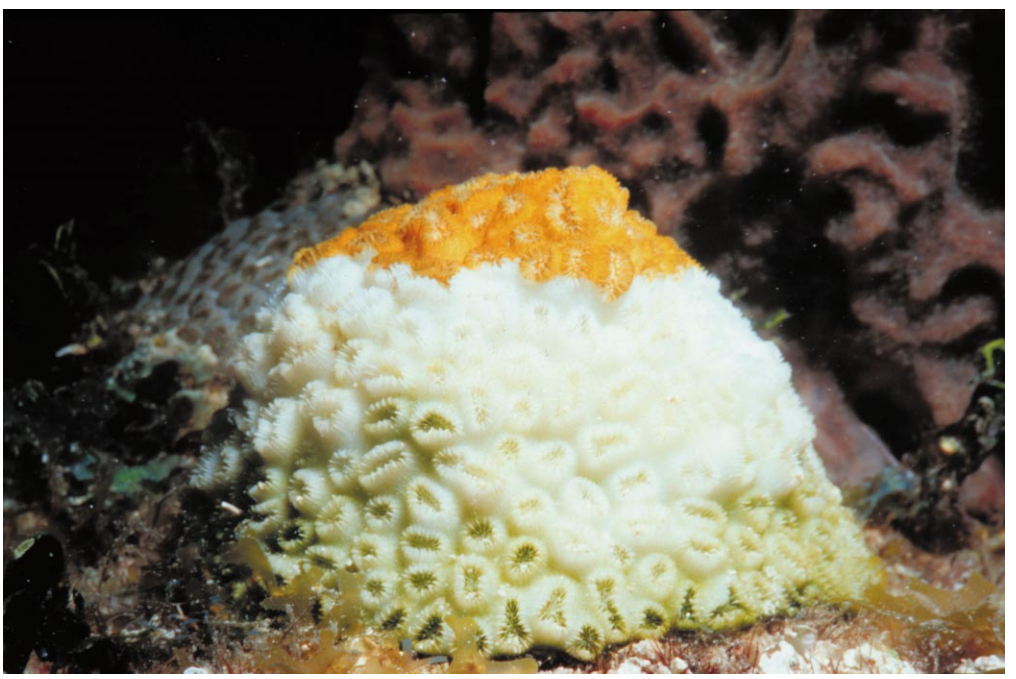

Fig. 2. Plague on *Dichocoenia stokesi*. Type II is shown here. This coral is the most susceptible of 17 affected coral species of the Florida reef tract (USA). Tissue loss occurs at rates of up to 2 cm per day, and because it affects small coral colonies (normally <10 cm in diameter) it routinely kills entire colonies in two to three days. Plague type II is the most virulent and destructive scleractinian coral disease yet documented. Long-term effects on the reef are unknown. Epizootics of plague have occurred on Florida reefs in the 1970s, 1980s and 1990s and were recently reported in the Caribbean in the 1990s.

Box 1. Koch's postulates for demonstrating the identity of a pathogenic microorganism

Disease related research in other areas of scientific endeavor always includes strict attention to the fulfillment of Koch's postulates (a procedure set forth by Robert Koch in the 1870s) by which a presumed disease pathogen is demonstrated to be the cause of a disease[12]. To demonstrate unequivocally the identity of a pathogenic microorganism, the following must be carried out:

- The microorganism must be documented as always being found associated with a particular disease.
- The microorganism must be isolated from the disease state and grown in pure culture under laboratory conditions.
- The pure culture of the microorganism must produce the disease when inoculated into or onto a healthy animal.
- The microorganism must be re-isolated from the newly diseased animal and identified as the same microorganism as the presumptive pathogen.

Satisfaction of Koch's postulates when the host is a coral is challenging for several reasons. First, duplication of the normal reef environment in laboratory aquaria is difficult, especially in terms of water movement (currents vs. aeration) and microorganisms present in the water column. Second, the natural mode of infection is not known for any coral disease. Consequently, inoculation by syringe or after wounding the host tissue could be as unnatural as exposure to concentrated suspensions of pathogen in aquarium water or placement of colonies on pathogen-inoculated plates. Finally, it is difficult to prove the re-isolation of the pathogen by sampling the newly diseased experimental coral. Because some diseases are present on the surface of coral tissue, and experimental inoculation usually involves inoculation of the aquarium environment, recovery of the test microorganism could be compromised by the presence of contaminated aquarium water.

Both type I and type II affect two species of *Acropora* (*palmata* and *cervicornis*; G. Smith, pers. commun.). Although type I is widespread throughout reefs of the Western Atlantic[26], type II has only been observed in the Bahamas[25].

Working with white band type II, Ritchie and Smith were the first to apply a commercially available (Biolog Inc., Hayward, CA, USA) diagnostic assay that determines metabolic (carbon-utilization) patterns to the study of bacteria associated with diseased versus nondiseased corals[27,28]. They supplemented the metabolic approach with genetic sequence analyses to identify disease microorganisms. Using this method, they have determined that white band type II, although associated with an assortment of gram-negative bacteria, always includes the bacterium *Vibrio charcharii*[25,27,28]. However, satisfaction of Koch's postulates still remains incomplete.

During the 1990s, Santavy and Peters concentrated on histological studies of the bacterial packets associated with white band type I. They have sampled and analysed white band diseased colonies of *A. cervicornis* on reefs from three regions (the Bahamas, the Florida Keys and Puerto Rico)[29], but have only found bacterial packets within diseased tissue from samples collected at one site in the Bahamas.

Plague. Further investigations by Dustan[30] and Richardson *et al.*[31,32] of plague on reefs of the Florida Keys have revealed that there are also two etiologies associated with this disease. In the late 1980s Dustan reported the recurrence of plague on Florida reefs and introduced the term 'white plague', which he used synonomously with plague[30]. He noted that, in contrast to his 1977 report[9], different coral species were susceptible. In 1995 Richardson *et al.*[31,32] documented a dramatic plague epizootic that also occurred on reefs of the northern Florida Keys, which rapidly spread to infect 17 of the 43 species of scleractinian corals present. Mortality rates of up to 38% of the most susceptible species occurred within periods as short as 11 weeks[31]. Infected coral colonies appeared to have the same symptoms as those revealed in Dustan's reports on the 1977 and 1980s plague epizootics. The 1990s plague (which we termed plague type II, Ref. 32) spread, from 1995 to 1997, to affect >400 km of the Florida Reef Tract[31], covering a range both north and south of Dustan's study site.

The most important characteristics that distinguish the plague outbreaks of the 1990s from Dustan's reports of this disease in the 1970s and 1980s (referred to as plague type I, Ref. 32) are the rate of tissue destruction and the coral species affected. In the latter outbreaks, coral tissue degradation occurred at rates much greater than 3 mm per day (up to 2 cm per day), and resulted in colony death in a matter of days as opposed to months[30,31]. Moreover, in the 1990s, *Dichocoenia stokesi* was the species most severely affected, whereas it was not reported as susceptible in the earlier plague events. Although Dustan's report on plague in the 1980s did not include any further observations of associated microorganisms, a pathogen was isolated from the 1995 plague epizootic[31]. This disease was found to be caused by a single, gram-negative bacterium, identified as a new species of the genus *Sphingomonas*[31]. Pure cultures of this bacterial isolate were shown to readily initiate disease activity in healthy corals in the laboratory, thus satisfying the procedures of Koch's postulates[32].

Although we distinguished plague type II from plague type I in the 1990s (Ref. 32), there may have been a previous outbreak. Comparison of the symptoms of plague type II with a description by Antonius[21] of 'white band disease' on nonacroporid corals in the Indo-Pacific in the 1980s reveals strong similarities, which suggest that this could also have been plague type II. Plague type II has recently been observed on reefs of Puerto Rico[2].

Black band disease. Advances have also been made in understanding the etiology of black band disease. Carlton and Richardson used microelectrode techniques to demonstrate that the suite of microorganisms found in black band disease (most of which were individually proposed as the primary pathogen) function together to maintain a distinctive microbial consortium that is directly analogous to the well studied microbial mats found in many illuminated sulfide-rich benthic aquatic environments[33,34]. Within the 1 mm thick band, steep vertical microgradients of oxygen and sulfide were observed, which are generated and maintained by the metabolism of the microbiota associated with the band (in the same manner as other microbial mat communities)[35]. Within the black band microcosm, sulfidogenesis in the anoxic microenvironment present at the base of the band resulted in the accumulation of up to 800 μM sulfide[33]. It was experimentally determined[34] that exposure of coral to this chemical microenvironment killed coral tissue. This is the only study to demonstrate the mechanism of coral tissue death by a coral disease.

Regional impacts of coral disease

The expansion of research efforts in the 1990s has also included the first statistical analyses of quantitative, regional-scale surveys of coral disease abundance and distribution. The distribution of black band disease was found to be random in the USA Virgin Islands[36], but clumped on reefs of the Florida Keys[37] and Jamaica[38]. Several studies have investigated the effect of the incidence of coral disease on reef structure and function. Two studies tested whether black band disease could open up new substrate

for coral colonization[36,39] – a potentially positive role. In both studies, however, the colonization of coral skeletal surfaces exposed as a result of black-band tissue degradation was dominated by algae and octocorallian coral species. Only one of 32 corals followed for periods of 25–60 months exhibited colonization by (a single) reef-building scleractinian coral.

An extensive paleological study documented the complete restructuring of a shallow reef community in Belize by white band disease[40]. The disease resulted in a shift in domination by canopy forming acroporid corals (from 70% cover to 0%) to small (i.e. <10 cm diameter), 'lettuce' corals of the genus *Agaricia* (from 10–>50% cover). Geological analyses of the sedimentary record (from trenches and cores) showed that this event, which happened between 1986 and 1995, had not occurred in the previous 4000 years (3835±100 years)[40], providing the first test of the hypothesis that current damage from diseases is significantly affecting reefs on a geological as well as a regional scale. Although stands of *A. palmata* and *A. cervicornis* were reported as predominate in the Caribbean up until the 1970s, such stands have now virtually disappeared[4,5,13,26,40].

Coral disease emergence in the 1990s

There have been many reports of new coral diseases in the 1990s. These include red band disease, yellow band disease, yellow blotch, dark spot disease, white pox, sea fan disease and rapid wasting disease. The emergence of these diseases was broadcast in the popular literature[1–3], on coral-reef web-sites and on coral-reef related Internet servers as anecdotal observations (Refs 4 and 5 provide a summary). For most of these diseases, supporting data were limited to photographs of afflicted coral colonies. In many cases, it is not clear that what is being shown is actually a disease[5]. The status of these new diseases is extremely confusing (Box 2).

Only one of the recently emerging new coral diseases has been systematically characterized. Aspergillosis of sea fans (gorgonian corals) rapidly swept through reefs of the Caribbean and the Florida Keys in 1995 and 1996, resulting in mass mortalities as a result of tissue-degrading lesions[42,43]. A team of investigators, using both laboratory and field techniques, showed that the lesions were caused by the terrestrial fungus *Aspergillus sydowii* (proven in laboratory experiments that fulfilled Koch's postulates)[42,44,45], and that disease incidence was correlated with water depth and protection from wave exposure[46]. The disease still persists throughout the western Atlantic. These investigators have postulated[46] that an unexplained, but well documented, mass mortality of sea fans that occurred throughout the Caribbean during the 1980s was an earlier epizootic of the same disease. This conjecture is based on photographs of diseased sea fans from the 1980s event that reveal the same lesions now known to be caused by *A. sydowii*. The effect of this extensive sea-fan mortality on the reef ecosystem is not known.

Results of studies of individual coral diseases

A summary of what is currently known about coral diseases (including only peer-reviewed literature that contains original data) is presented in Table 1. The main conclusions are as follows:

• There are currently only four diseases for which both coral tissue destruction leading to mortality, and the presence of a consistent, characteristic microorganism (or microbial consortium) associated with the disease are known. These are apergillosis, black band disease, white band disease type II and plague type II. This is in contrast to the 13 individual coral diseases put forth by various investigators.

• Only three diseases (aspergillosis, black band disease and plague type II) have an associated microorganism (or microbial consortium) that has been demonstrated to be the disease pathogen.

• The mechanism of coral tissue death is known only for black band disease.

• Only white band disease has been shown to restructure a reef on a regional scale.

Most coral diseases, including new ones and some that were first described in the 1970s and 1980s, have been only partially characterized[4,5,14,15]. These include white band type I, plague type I, shut down reaction, red band disease, yellow band disease, yellow blotch, rapid wasting disease, dark spot disease and white pox. No pathogens have been identified for any of these diseases, and confusion is prevalent. Despite this, many of these syndromes are currently included in monitoring programs designed to evaluate coral reef health.

Box 2. Incompletely characterized new diseases: a case of confusion

The status of many of the newly reported coral diseases is obscure. Two examples are presented that illustrate some of this current confusion. In both cases, the pathogens have not been isolated.

• There are numerous conflicting reports concerning 'red band disease'. In terms of chronology, Santavy and Peters[29] stated that red band disease was first reported in 1983 (Ref. 17) as a variant of black band disease found on gorgonians. The 1983 paper, however, did not mention the term 'red band' and referred to an observation of nonblack-band-disease cyanobacteria (two species) on gorgonian colonies, which they termed 'suspected gorgonian black band disease'. No tissue degradation or band migration rates were reported. In 1992 Richardson published a descriptive study of a 'red band' disease observed during one field trip to the Bahamas[41]. This (nonpeer-reviewed) report documented the unique motility pattern of a red population of cyanobacteria (again two species) observed on scleractinian corals (but not gorgonians). The red populations were associated with a disease line similar to those of other coral line-diseases but were unique in that filaments formed a dense, narrow line between healthy tissue and coral skeleton only at night, and spread out in a thin mat over both healthy tissue and exposed skeleton during the day. Coral tissue loss reached a peak of 1 mm per day. Culture attempts were unsuccessful, and a return to the same area the following year did not reveal any disease activity. This version of red band disease has been observed on scleractinian corals in the Caribbean[4] (and by J. Lang, pers. commun.). Photographs of another 'red band' disease on gorgonians have appeared that are actually a stage of aspergillosis disease of sea fans (see text), in which the sea fan tissue turns dark purple[42]. Yet another 'red band' disease was displayed as a representative image on a coral disease web site. In this case, the identifying image is clearly black band disease. All four of these versions of red band disease can be found on underwater coral disease identification cards and Internet sites put together by different coral-reef scientists in the absence of peer-reviewed, substantive coral disease characterizations.

• Different names are being given to different successive stages of the same disease. At a recent workshop on coral pathology in the Caribbean [Coastal Pathologies Workshop, CARICOMP (Ref. 6) annual site director's meeting], several coral disease investigators observed the same area of reef at the same time. At the site, the same coral colonies exhibited gradations of light to dark anomalies in pigmentation. One investigator, who noted that these areas are often initially dark green, is currently calling two patterns observed within the relatively lighter pigmented areas yellow blotch and yellow band disease. A second investigator said that his group termed the dark green areas on the same colonies dark spot disease. A third stated that he was aware of another group of investigators who believed the dark areas to be the initial stages of rapid wasting disease (also called rapid wasting syndrome); and yet another group believes that the lesions of rapid wasting syndrome are actually parrot fish bites, and that such an afflicted coral has 'parrot fish bite syndrome' or 'parrot fish white spot biting'. The last two have been a subject of active debate both on the Internet and in letters to the editors of journals (including *Science*).

Table 1. Summary of the current state of knowledge of coral diseases[a]

Parameter	Described for	Refs
Documentation of tissue destruction and/or mortality[b]	Black band	7,16
	White band type I	8
	White band type II	25
	Plague type I	9
	Plague type II	32
	Aspergillosis	42,46
Identification of a pathogen[c]	Black band	10,11,16–18, 33,34
	Plague type II	31,32
	Aspergillosis	42,44
Identification of a consistent associated microorganism[d]	White band type II	25,28
Fulfillment of Koch's postulates	Aspergillosis	42,44
	Plague type II	31,32
Mechanism of tissue mortality	Black band	34
Identification of the pathogen reservoir	Black band	48
	Aspergillosis	44
Long-term effects of disease outbreaks on reef community	White band	40
Correlation with environmental degradation	Black band	16
Cue that controls disease-line migration over healthy coral	None	
Mechanism of disease transmission	None	
Identification of what triggers the disease	None	
Mechanism of host resistance	None	

[a]This list includes complete citations of references that are peer-reviewed and are primary reports that present definitive results. Diseases for which none of the parameters listed has been documented in peer-reviewed articles (with the exception of summaries of 'new' diseases) are yellow band disease, yellow blotch, red band disease, white pox, rapid wasting disease and dark spot disease.

[b]Various terms, not listed in the table, appeared in the early literature before the definitive studies were conducted that characterized these diseases. Thus the terms 'stress related necrosis', 'white line' disease, and 'white death' have all been applied to afflicted corals that would now be characterized as plague or white band types I or II.

[c]It is now known that black band disease consists of a microbial consortium. All primary references documenting individual members of the consortium, as well as defining the functional nature of the consortium, are listed.

[d]The packets of gram negative bacteria found in the diseased tissue of white band type I have also been found in some healthy acroporids, and some colonies with white band disease symptoms do not have the packets[23,29]. Because of this discrepancy, white band type I is not included in this category.

Correlation of coral disease with environmental degradation

One of the most important aspects of coral disease, especially in relation to overall reef degradation, is the effect of anthropogenic influence. Unfortunately, this is one of the least understood areas. Although several investigators have postulated that disease incidence might be associated with elevated nutrients, it has only been Antonius[16] who has presented data showing a correlation between black band (but not white band) disease incidence in polluted (i.e. nutrient rich) waters. In contrast, there are recent sightings of black band (and other) diseases in remote locations. Bruckner *et al.*[47] have revealed both an increase in the incidence of black band disease and a pattern of disease spread that is associated with high levels of terrestrial runoff; however, no nutrient data were presented. This is an area that demands focused research.

Prospects for the future

The ongoing characterization of coral diseases has yielded several important clues about their nature. Consequently, the natural reservoirs of two coral-disease pathogens are now known. *Aspergillus sydowii*, the pathogen of aspergillosis of sea fans, was recently cultured from both marine-coastal and oceanic zones[44], demonstrating a reservoir in the water column. A reservoir of the black band disease community has been found in sediment-filled depressions on healthy (but black band disease susceptible) coral colonies[48]. We now know the identity of at least some coral disease pathogens, which is the first step in determining possible treatment or prevention.

Current research by many of the investigators cited in this review is focusing on new areas, such as discerning mechanisms of aspergillosis resistance in sea fans, applying molecular probes to confirm identities of pathogens in outbreaks in different regions, and experimental manipulations to trigger disease activity from reservoir populations. Moreover, much current research is aimed at determining the relationship, if any, between increased nutrients (eutrophication) and coral disease.

The continuation of the rigorous research efforts of recent years, specifically those that go beyond descriptive studies, is of critical importance for a complete understanding of coral diseases. A word of caution, however: until a pathogen has been identified for each of the uncharacterized coral 'diseases' (including fulfillment of Koch's postulates), these syndromes should be clearly identified as potential disease states and not coral diseases[5].

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