Coral diseases: what is really known?

Laurie L. Richardson

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.

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. There were no reports of attempts to apply the established methods of medical microbiology (Koch’s postulates, Box 1) to coral diseases.

During the International Year of the Reef (1997) and continuing into the International Year of the Ocean (1998), much attention and activity has been focused on evaluating the current status of coral reefs. The uniform conclusion of these and previous observations 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 — 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 corals8 and ‘plague’ (Fig. 2) — revealed the presence of various bacteria. Heterotrophic microscopic observations of diseased tissue, all of which included the observation that all of these microorganisms were present within black band. Black band disease was also newly reported to infect gorgonians19,20 (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-Pacific21 and the Red Sea22, 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 Caribbean23. 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 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 filamentous 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 one time (termed ‘shut down reaction’). Although no microbiological studies were conducted, exposure of healthy corals to sloughed-off, necrotic coral tissue elicited the same symptoms. The second was a disease described as hyperplasia of gorgonian corals associated with nodules that contained the filamentous green alga Enteridiad endobius24.
Neither tissue degradation nor colony mortality occurred, however, and infected gorgonians recovered by encapsulating affected areas\textsuperscript{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, plaque and shut down reaction. Of the four, the identity of only one primary causative pathogen had been proposed – the cyanobacterium \textit{P. corallyticum} found in black band disease\textsuperscript{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.

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\textsuperscript{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.
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. To demonstrate unequivocally the identity of a pathogenic microorganism, the following must be carried out:

1. The microorganism must be demonstrable as always being found associated with a particular disease.
2. The microorganism must be isolated from the disease state and grown in pure culture under laboratory conditions.
3. The pure culture of the microorganism must produce the disease when inoculated into or onto a healthy animal.
4. The microorganism must be re-isolated from the newly diseased animal and identified as the same pathogen as the presumptive pathogen.
5. The pure culture of the microorganism must produce the disease when inoculated into or onto a healthy animal.
6. The microorganism must be isolated from the disease state and grown in pure culture under laboratory conditions.
7. The pure culture of the microorganism must produce the disease when inoculated into or onto a healthy animal.
8. The microorganism must be isolated from the disease state and grown in pure culture under laboratory conditions.
9. The pure culture of the microorganism must produce the disease when inoculated into or onto a healthy animal.
10. The microorganism must be isolated from the disease state and grown in pure culture under laboratory conditions.

Both type I and type II affect two species of Acropora (palmata and cervicornis). Smith et al.27,28. Although type I is widespread throughout reefs of the Western Atlantic26, type II has only been observed in the Bahamas26.

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 microbial carbon-utilization patterns to the study of bacteria associated with diseased versus nondiseased coral27,28. 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 charcharii30,31,32. However, satisfaction of Koch’s postulates still remains incomplete.

During the 1990s, Carlton et al.33,34 observed the expansion of research efforts in the 1990s has revealed that there are also two etiologies associated with this disease. In the late 1980s Dustan reported the re-emergence of plague on Florida reefs and introduced the term ‘white plague’, which he used synonymously with plague35. He noted that, in contrast to his 1977 report36, different coral species were susceptible. In 1995 Richardson et al.31,32 documented a dramatic plague epidemic 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 weeks37. Infected coral colonies appeared to have the same symptoms as those revealed in Dustan’s reports of the plague outbreaks of the 1980s from Dustan’s reports of this disease in the 1970s and 1980s (referred to as plague type I, Ref. 32) spread, from 1995 to 1997, to affect ~800 km of the Florida Reef Tract38, covering a range both north and south of Dustan’s study site.

The most important characteristics that distinguish the plague outbreaks of the 1980s 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 months39. Moreover, in the plague outbreaks of 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 reported in the 1980s did not include any further observations of associated microorganisms, a pathogen was isolated from the 1995 plague outbreak32. This disease was found to be caused by a single, gram-negative bacterium, identified as a new species of the genus Sphingomonas37. 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 postulates32.

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 Antonius27 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 Rico32.

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 environments31,32,33. 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 microbiont associated with the band (in the same manner as other microbial mat communities33). Within the black band micro-environment, sulfidogenesis in the crude extracellular environment present at the base of the band resulted in the accumulation of up to 800 μM sulfide33. It was experimentally determined19 that exposure of coral to this chemical micro- environment 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 disturbing. In 1995 the disease was to be random in the USA Virgin Islands40, but clumped on reefs of the Florida Keys and Jamaica41. 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—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 oococarall 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 paleoecological study documented the complete restructuring of a shallow reef community in Belize by white band disease. The disease resulted in a shift in domination by canopy-forming acroporid corals (from 70% cover to 3%) to a small (i.e. <10 cm diameter) ‘lettle’ or ‘lettle’ 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 (3X25–100 years), providing the first test of the hypothesis of a 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, these stands have now virtually disappeared.

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, on coral-reef websites and on coral-reef related Internet servers as anecdotal observations (Relis 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. 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. 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) and that disease incidence was correlated with water depth and protection from wave exposure. The disease still persists throughout the western Atlantic. These investigators have postulated 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 1986s 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. These include white band disease 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.
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 Antonius14 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.12 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 syzyrini, the pathogen of aspergillosis of sea fans, was recently cultured from both marine-coastal and oceanic zones,15 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.25 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 identity 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.16

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