III. HISTORICAL PERSPECTIVE--LESSONS LEARNED

PROGRESS IN UNDERSTANDING CORAL DISEASES IN THE CARIBBEAN

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ABSTRACT

Coral disease research in the Caribbean initiated in 1972 with the discovery of black band disease (BDD) by Dr. Arnfred Antonius. Since this time, there has been an expansion in the number of researchers working in the Caribbean, including studies to document the prevalence and incidence across large spatial scales and at increased temporal frequencies, evaluation of the linkages between disease and environmental drivers, identification of disease vectors, and laboratory studies to characterize causes, physiological responses, histological changes, defense mechanisms and mechanisms of resistance and susceptibility. Since 1998, the Caribbean has emerged as a "hot spot" for coral diseases due to the large number of named diseases, their wide distribution, expanding host ranges, and increasing abundance and severity, with over 30 diseases now reported from this region. Localized epizootics of three diseases (BBD, WBD, WP-I) were first documented in the 1970s; one of these (WBD) expanded throughout the Caribbean to become the most significant factor in the region-wide decline of acroporids. Five diseases (WP-II, YBD, WPX, DSD and ASP) emerged in the mid 1990s, and have expanded their geographic distribution and host ranges over the last ten years with several of the diseases causing substantial coral mortality since the late 1990s. By 2005, at least 41 scleractinian corals, 8 gorgonians and two hydrozoans were observed with one or more diseases. The most abundant and important group of corals found on Caribbean reefs today (*M. annularis* complex) is susceptible to at least 8 different diseases, and individual colonies may show signs of 2-3 diseases at the same time. The average prevalence of coral diseases at the community level is generally low, although it is highly variable between and within sites, during different years, and seasonally. Disease outbreaks have affected up to 91% of certain susceptible populations in localized areas, and often (but not always) exhibit a clumped distribution. Disease prevalence and severity is generally greater during warm water periods, and recent disease outbreaks have been associated with mass bleaching events. Over the last five years, there has been an increase in the numbers of studies that have reported a correlation between disease and environmental factors, including higher prevalence rates and greater rates of spread in areas affected by nutrients, sediments and other pollutants. Causative agents have been identified for relatively few diseases, three of which (WP-II, ASP and WPX) have been verified through application of Koch's postulate. In these and other diseases, complex microbial communities have been identified using new molecular techniques, including

biota on diseased tissue that is absent from control samples and suites of microorganisms that differ from those identified using traditional (microscopy and culture) techniques. Furthermore, pathogens identified using traditional microbiological approaches are no longer infective, including the proposed causative agent for white plague type II, suggesting 1) the pathogens may have lost their virulence and/or corals have gained immunity, or 2) causation was determined based on a relatively small number of corals from a single location/event and other microbial agents are capable of causing similar signs. In addition to key advances in understanding the coral holobiont, and how microbial communities associated with coral tissue and coral mucus change during periods of stress, some advances have been made in identifying possible vectors of disease, including linkages between a coral eating snail (*Coralliophila abbreviata*) and a white syndrome that affects acroporids as well as three spot damselfish and BBD. Efforts have been made to mitigate disease, through removal of the microbial community, antibiotic treatments, use of putty and/or clay to cover the affected area, and addition of urchins to reduce algal abundance, however these exhibited only limited success and they do not appear to be feasible treatments on a larger scale. One of the major limitations in advancing our understanding of diseases has been the lack of standardized nomenclature and diagnostic criteria for diseases, which has resulted in a proliferation of names and the identification of new presumed diseases that later have been shown to be caused by other factors. Some of the key needs for the Caribbean include: 1) greater geographic coverage and more frequent surveys to characterize prevalence and incidence; 2) more emphasis on population dynamics and impacts, including size structure of diseased and healthy corals, extent of partial and whole colony mortality and impact to individual corals and coral populations; 3) concurrent monitoring and assessment of environmental factors; 4) revision of existing disease nomenclature and adoption of standardized terminology and diagnostics; 5) application of traditional culture and histopathology techniques in combination with new molecular tools to characterize causative agents and sources of pathogens and development of molecular probes to facilitate screening of corals; and 6) more emphasis on cellular diagnostics, including biomarker characterization, to assess stress levels in corals and underlying causes; and 7) a coordinated rapid response program to address coral disease outbreaks and unusual mortality events.

Introduction

Until the late 1970s, benthic substrates on Caribbean reefs were occupied primarily by reef-building corals, turf algae, coralline algae, and other benthic invertebrates (sponges). Coral reefs exhibited a generalized zonation pattern with elkhorn coral (*Acropora palmata*) forming large, monospecific stands in the reef crest and shallow fore reef (0-5 m depth); stands of staghorn coral (*A. cervicornis*) at intermediate depths (5-25 m depth) on wave exposed reefs and in shallow, protected environments; massive corals (dominated by *Montastraea annularis* complex) throughout the fore reef (5-30 m depth) and in back reef and lagoonal areas; and plating agaricids near the base of the reef (20-40 m depth) (Goreau, 1959; Adey, 1978). Caribbean reefs have experienced significant losses in living coral cover over the last three decades and "classic" zonation patterns have disappeared from many locations (Gardner et al., 2003). As corals die, exposed benthic substrates are monopolized by fleshy macroalgae, encrusting and bioeroding sponges, and other organisms. These "pest" species are outcompeting and

overgrowing corals, and may prevent new recruitment and regrowth of damaged corals (Hughes, 1994; Aronson and Precht, 2001; Weil 2004).

Coral diseases were first described in the western Atlantic almost 35 years ago (Antonius, 1973), but it wasn't until about ten years ago that diseases were identified as a significant factor accelerating the deterioration of coral reefs (Epstein et al., 1998; Harvell et al., 1999; Green and Bruckner, 2000; Sutherland et al., 2004; Weil, 2004). Black band disease (BBD; Antonius, 1973; Garrett and Ducklow, 1975; Antonius, 1973), white band disease (WBD; Gladfelter et al., 1977) and white plague (WP type I; Dustan, 1977) were first observed in the 1970s from reefs of Belize, Florida, Bermuda, Puerto Rico and the USVI and have become chronic afflictions of important reef-building corals. Although generally low in prevalence and patchy in distribution, these diseases have persisted on the same reefs for many years, and have spread throughout the western Atlantic infecting a growing number of host species (Gladfelter, 1982; Rützler et al., 1983; Dustan, 1987; Peters, 1984; Edmunds, 1991; Kuta and Richardson, 1996; Aronson and Precht, 1997; Bruckner et al., 1997). The earliest report of significant coral mortality from disease was from the Florida Keys (USA), where an outbreak of WP spread through *Mycetophyllia* spp. and *Colpophyllia* spp. populations, and was predicted to cause the disappearance of *M. ferox* from some locations (Dustan, 1977). Ten years later a second outbreak of WP affected *M. annularis* and 11 other species. Large numbers intact dead skeletons of *M. ferox* and other species were found on the fore reef, although numerous healthy, unaffected colonies were still apparent (Dustan and Halas, 1987), highlighting extensive losses of corals from disease as well as the resilience of these species during the 1980s. WBD played a dominant role in the precipitous (90-98%) decline of *A. cervicornis* and *A. palmata* populations during the 1970s and 1980s (Bruckner, 2002; Aronson and Precht, 2001; Gardner et al., 2003). It is the only disease to date that has caused major changes in composition and structure of reefs over large areas of the Caribbean (Williams et al., 1999; Green and Bruckner, 2000).

Since the mid 1990s, there has been a rapid proliferation of diseases, including a recent emergence of new syndromes (Sutherland et al., 2004; Weil, 2004; Weil et al., 2006). Over 30 diseases have been reported from the Caribbean (Table 1). Some of these affect a single species in specific localities, while others have a widespread geographic distributions and wide host ranges (Weil, 2004). Epizootic events have been associated with six diseases [WP-II, BBD, WBD-I, yellow band disease (YBD), white pox (WPX) dark spot disease (DSD) and Aspergillosis (ASP)], and three diseases (WP-II, YBD and ASP) are currently causing extensive mortality throughout the region (Bruckner, 2002; Weil, 2004). In addition to the region-wide decline of acroporids, there has also been a notable degradation of massive reef-framework corals (in particular the *Montastraea annularis* complex). Declining health of *M. annularis* (complex) has been associated with WP-II epizootics of increasing severity, bleaching events (1995, 1998, 2005), YBD, BBD, DSD, and parrotfish predation (Cervino et al., 1997; Bruckner and Bruckner, 2000; Nugues, 2002; Miller et al., 2003; Jordán-Dahlgren and Rodríguez-Martínez; Weil, 2004; Bruckner and Bruckner, 2006). The recent emergence of diseases in the wider Caribbean appears to be an unprecedented event over a millennial time scale (i.e. >3800 yr) (Aronson and Precht, 2001).

While disease has undoubtedly played a major role in shaping the structure and ecology of Caribbean reefs over the past few decades, very little is known about many of

the fundamental aspects of coral diseases, such as their causes, how diseases are transmitted, factors conferring resistance and resilience, and the long-term effects of diseases on coral populations and coral reef ecosystems (Woodley et al., 2003). Researchers have made significant advances in coral disease research through the application of new laboratory tools and more detailed epizootiological studies (e.g., Edmunds, 1991; Bythell et al., 1993; Carlton and Richardson, 1995; Kuta and Richardson, 1996; Bruckner et al., 1997; Richardson et al., 1998; Bruckner, 2002; Downs et al., 2005). Unfortunately, large gaps remain in our understanding of coral disease, and few strategies and tools have been provided to resource managers to assist in the management of diseases and mitigation of disease impacts. Due to a growing number of disease reports, and an absence of standardized criteria for naming diseases, much confusion surrounds many of the newly emerging diseases. In some cases, scientists do not have the appropriate diagnostic tools to characterize disease outbreaks, and resulting analyses may be inconclusive or incomplete. Disease studies are being undertaken without the use of standardized investigative methodology, making it difficult to consistently characterize these events and draw comparisons between disease outbreaks in different locations. Other factors limiting progress include 1) a lack of standardized nomenclature and diagnostic tools, 2) conflicting reports on causative agents and their sources, 3) insufficient data to conclusively identify linkages between disease and environmental stressors. Finally, standard operating procedures for sampling, approaches to prevent contamination/dispersal of diseases, and other strategies to minimize environmental impacts have not been widely applied in the Caribbean.

This paper reviews recent progress in coral disease research and summarizes our current understanding of the major diseases that have impacted Caribbean coral reefs over the last several decades. Topics discussed include 1) number and variety of diseases, 2) host ranges and geographic distribution, 3) prevalence and impact, 4) causes, and 5) role of environmental factors. An effort is made to identify gaps in knowledge, factors hindering progress, and contentious issues surrounding specific diseases from the western Atlantic. It is hoped that the information and lessons learned in the Caribbean can be applied to Pacific coral disease studies.

How many diseases are there?

Although over 30 coral diseases have been reported from the wider Caribbean since 1972 (Appendix I and II), only a handful of these syndromes (e.g., BBD, WBD, WP, YBD, DSD, WPX, ASP, and various skeletal abnormalities) have been observed throughout much of the Caribbean and certain aspects of their etiology and ecology have been characterized. Much confusion surrounds many of the other described syndromes, and few data are available on their distribution and abundance, impact, or cause. Several diseases have been subdivided into different "types" based on highly variable features (e.g., a zone of bleached tissue; differences in rates of movement, species affected). These characteristics often differ temporally and spatially, and may be unreliable, unless affected corals are tagged and followed over time. Syndromes have also been identified with limited etiological and ecological observations. These may lack a unique description of gross signs, disease signs differ among publications, or conditions were named on the basis of a single observation (Appendix II). Unsubstantiated causes or

agents have been proposed for several new syndromes, and information on prevalence, patterns of spread, or evidence of tissue mortality may be lacking. One of the difficulties in standardizing nomenclature used for coral diseases is that disease signs manifest on corals in a limited number of ways, but they may be caused by different pathogens or unrelated abiotic factors.

The major syndromes reported in the literature from the wider Caribbean are combined here into six major categories, based on similarities of gross field signs (Appendix I). This includes: white syndromes, cyanobacterial mat diseases, tissue discoloration, abnormal skeletal development, skeletal damage, and gorgonian syndromes.

White syndromes

There has been a proliferation of names for coral diseases with virtually identical visible signs that reflect the pattern of loss of coral tissue and exposure of skeleton. All of these syndromes are characterized by a sharp, distinct line between apparently healthy coral tissue and freshly exposed skeleton, with no obvious microbial mat present at the disease interface. These conditions have been differentiated based on 1) species affected; 2) presence of a zone of bleached tissue at the disease boundary; 3) rates of tissue loss; 4) location of lesion on colony surface; and 5) patterns of spread. Without microbial analysis of these diseases (or use of a molecular probe to confirm proposed causative agents), it is difficult to verify that WBD, various forms of WP, and other white syndromes are in fact distinct diseases, since disease signs are so similar (Appendix III; Bythell et al., 2004).

Many studies have used various terms interchangeably to describe white syndromes, making it difficult to characterize regional patterns of disease occurrence. For example, Antonius (1977; 1981) identified 11 species of massive and plating species with WBD, while other researchers report this as WP (Dustan, 1977), and only use the term WBD for *Acropora* spp. (Aronson and Precht, 2001). Dustan (1977) suggested that WP represents a suite of diseases that result in the death of coral tissue, but he does not provide a detailed diagnosis of the macroscopic field signs. He characterized WP as "*lesions on the colony that expanded at a rate of a few mm per day and often resulted in whole colony mortality"*, but presents little information on location of lesions or patterns of progression. An outbreak of a disease with signs that are similar to Dustan's "plague" (Dustan, 1977) was observed in 1995 on the same reefs (Richardson et al., 1998a). This condition was designated WP type II because of 1) a faster rate of progression (up to 2 cm/day); 2) highest prevalence on *Dichocoenia stokesi*, a species unaffected during the original WP outbreaks; and 3) a unique pattern of tissue loss. WP-II progresses from the entire base of the colony to the apex, while WP- I lesions occur more variably across the colony (Richardson et al., 1998b).

Richardson et al. (2001) also reported a third type of plague, *WP type III*, which was characterized by more rapid rates of progression (up to 10 cm/day) and a different pattern of tissue loss. Unlike WP-II, lesions start in the center of colonies and radiate out, and only the largest colonies of *M. annularis* (complex) and *Colpophyllia natans* are affected. White plague has also been used to describe tissue loss characterized by random patches of denuded skeleton that extend sporadically and do not give rise to a graded

algal community (Bythell et al., 2002). Peters (1984) coined the term "stress-related necrosis" for another similar condition characterized by sloughing of degenerating tissue, with no obvious discernable pathogens at the disease line.

Since the mid 1990s, several white syndromes with unique diagnostic features have been reported on A. *palmata,* including WPX, patchy necrosis, necrotic patch syndrome, and other unnamed conditions (Ritchie and Smith, 1998; Bruckner and Bruckner, 1996; Patterson et al., 2002; Sutherland et al., 2004; Jordán-Dahlgren and Rodríguez-Martínez, 2004). While some authors have suggested that these syndromes are synonymous (Sutherland and Ritchie, 2004; Sutherland et al., 2004), a pathogen has been identified only for colonies identified with WPX in the FKNMS (Sutherland et al., 2002), and descriptions and photographs of these conditions are highly variable. In some cases, WPX has been described as circular, dime-sized lesions, while other descriptions suggest these are more irregular in shape. In addition, Weil (2003, 2004) reported "patchy necrosis" on *A. palmata* colonies during doldrums-like conditions, and later indicated that these were associated with parrotfish and sea cucumber fecal matter.

Only two diseases have been identified in *A. cervicornis*, WBD type I and WBD type II. Published descriptions of WBD from *A. cervicornis* have far less detail on the pattern and rates of progression than reports of WBD on *A. palmata*. For example, WBD- II can be confused with both bleaching and WBD-I. Affected colonies have a receding margin that progresses at a faster rate than WBD-I, and tissue loss is preceded by a band of bleached tissue up to 20 cm in width. The bleaching margin may arrest, however, allowing the "peeling" margin to catch up to the pigmented tissue (Ritchie and Smith, 1998). Without the presence of a bleached margin, the disease is indistinguishable from WBD type I. Williams and Miller (2005) reported an outbreak of disease affecting *A. cervicornis* in Florida. Unlike WBD, tissue loss was characterized by rapid tissue sloughing from multifocal lesions and no bleaching was noted. They termed this condition *rapid tissue loss*.

Further complicating distinctions between various white syndromes, scars from predation can be difficult to differentiate from diseases, especially when predators are cryptic (e.g., *Coralliophila abbreviata*) or nocturnal (*Hermodice carunculata*). Predators frequently feed on degrading tissue associated with disease lesions; fireworms and corallivorous snails often occur on colonies with BBD, WP, WBD and other white syndromes, and they may also serve as vectors for disease (Bruckner, 2002; 2003; Williams and Miller, 2005).

Cyanobacterial mat diseases

A number of diseases are associated with cyanobacteria. These often exhibit a similar identifiable group of signs on the coral and consistent anatomical alterations that are visible in the field, making it difficult to separate these conditions without laboratory confirmation of the specific cyanobacteria present. Affected colonies have a distinctive visible microbial assemblage that forms an advancing band or mat, separating denuded coral skeleton from living tissue. The mat is usually dominated by one or several cyanobacterial species, although the species of the dominant cyanobacterium may vary between large geographic regions, even in the same presumed disease (Cooney et al. 2002; Frias-Lopez et al. 2002, 2003). The mat may appear black, brown or reddish depending on light levels, the species of cyanobacterium, and its complement of

photosynthesis pigments. This includes diseases referred to as BBD and red band disease (RBD type I and RBD-II; Appendix I; III).

Tissue discoloration

Corals exhibit wide variations in color depending on species, genotypes, clade of zooxanthellae and/or type and concentration of algal pigments, and in response to physical and environmental factors. Individual colonies may show changes in coloration due to a partial loss of zooxanthellae or their photosynthetic pigments (bleaching), host responses to irritants and injuries, or from coral diseases.

YBD is characterized by lightening of tissue. Affected colonies have small circular blotches of pale yellow tissue surrounded by normally pigmented tissue. These lesions expand in size over time, with central areas dying and becoming colonized by algae. YBD lesions can be confused with bleaching, and during bleaching events it may be difficult to determine which corals are affected by YBD (Cervino et al., 2001).

DSD is characterized by darker than normal coloration. Colonies may have one or more small, round spots or patches of darkened tissue (and discolored skeleton in some cases) that grow in size over time. The spots may be associated with a depression in the coral surface, and spots may expand into a ring surrounding dead coral. Weil (2004) recently divided DSD into DSD type I and DSD-II, and also identified three other similar syndromes, dark band disease (DBD), purple band syndrome (PBS) and tissue necrosis. DSD-II is similar to DSD-I, except the "spots" were larger and a thin, necrotic tissue line was apparent at the margin. PBS differs from DSD in that colonies of *S. siderea* have a band of discolored tissue that advances from the outer margin to the inside. It is unknown whether these are different diseases, or are later stages in the progression of DSD, as a causative agent has not been identified and few studies have followed the progression of DSD lesions over time. Some researchers consider these conditions the same as DSD (Gil-Agudelo et al., 2004; Appendix I and III).

Abnormal skeletal growths

Coral colonies often exhibit distinct circumscribed lesions on the surface of a coral, composed of the corals tissue and skeleton. These structures are typically raised spherical to irregular masses that project above the surrounding corallum. They can be subdivided into three categories on scleractinian corals: a) a proliferation of all cell types that may be atrophied or normal in appearance (gigantism, area of accelerated growth, hyperplasm, growth anomaly); b) white, globular masses with few discernable polyp structures and a reduction or absence of zooxanthellae (tumor, neoplasm, calicoblastic epithelioma); and c) chaotic polyp development (Peters et al., 1986; Appendix I and Table 1). Hyperplasms do not appear to cause significant damage to the colonies, while neoplasms damage affected areas, leaves them more susceptible to invasion by boring organism, and destroys normal polyps and their functions (e.g., reproduction).

Skeletal damage or erosion

Damage to scleractinian corals associated with the disruption of septa or calices, or complete loss of the upper skeletal layers may be the result of physical injuries (e.g., abrasions during storms, anchor damage, fin damage), various biotic interactions (predation by fishes, sponge bioerosion, aggressive interactions among corals) and certain

coral diseases. Loss of corallites has been reported for WPX lesions, but the skeleton remains intact (Sutherland, 2002). Skeletal damage has also been reported in WBD-II (Ritchie and Smith, 1998). In skeletal eroding band (SEB), ciliates create a distinct black band adjacent to living tissue. The ciliates secrete a lorica (their "house"), which is embedded in the coral's skeleton and can completely destroy the surface layer of the skeleton (Antonius, 1999). The ciliates form a distinct black to grey band at the margin between exposed skeleton and live tissue, which may be confused with BBD.

Rapid wasting disease (RWD) and ridge mortality disease (RMD) were reported as coral diseases (Abbott, 1979; Cervino et al., 1997; Goreau et al., 1998), but have since been found to be associated with predation by fishes and the formation of territorial algal lawns (Bruckner and Bruckner; 1998; 2000; Borneman, 2005). RWD was characterized as irregularly-shaped white lesions denuded of tissue with the uppermost layers of the skeleton etched away; the exposed limestone was unusually brittle and crumbly (Cervino et al., 1997). Filamentous fungal hyphae covered and were invading epidermal cells, and were proposed as the causative agent. Detailed visual and photographic observations and experimental manipulations demonstrated that RWD is caused by focused biting by the stoplight parrotfish *Sparisoma viride* (Bruckner and Bruckner, 2000), a phenomena that was documented over 100 years ago. The RWD researchers recently identified a fungus in the mouth and fecal matter of *S. viride*, and proposed that parrotfish were a vector for RWD (Richardson, 2000). While linkages between a fish and a fungal pathogen have not been conclusively verified, the major damage to affected colonies has been shown to result directly from predation: 1) lesions advance only during daylight; 2) no further tissue or skeletal loss occurs once the parrotfish were excluded; and 3) lesions rapidly heal in absence of further biting (Bruckner and Bruckner, 2000).

Ridge mortality disease is associated with the loss of tissue and skeletal structures along elevated ridges of brain corals, with tissue remaining in the valleys (Abbott, 1979). Lesions initiate at a single point within the colony surface (or at the margin) and expand outward, following the meanders of the colony. The ridges typically are not completely destroyed; skeletal damage is largely restricted to the loss of septa. This condition is associated with the development and expansion of *Stegastes planifrons* algal lawns (Bruckner, 2002, 2003; Borneman, 2005). However, it is unclear whether fish bites are the sole cause of tissue loss. Biting may cause a stress response that triggers tissue sloughing, or the fish may introduce a pathogen that causes advancing tissue loss. Interestingly, only ridges are affected, and tissue remains in the valleys (and around polyp mouths) until it is overwhelmed by algae. Similar biting by damselfish can create multiple focal lesions that affect individual circular polyps (on *M. annularis* and *S. siderea*), or result in the development of "chimneys" in acroporids (as the coral attempts to contain the algae).

Gorgonian syndromes

Gorgonians have been observed with BBD, RBD, Aspergillosis, predation by gastropods and polychaetes, tumors, and other conditions (Morse et al., 1977; Rützler et al., 1983; Nagelkerken et al., 1997). Abnormal growths often develop on the branches of gorgonian corals (algal tumor, algal gall, or nodules) in response to endolithic algae, fungi and other epibionts (Morse et al., 1977). These are hard concretions of fibers of gorgonin that form spherical or irregular, but may be irregular shaped masses. They are

located predominantly at the axial bases of the colony, but often extend the overall length of the colony.

Aspergillosis is an extremely virulent fungal disease that affects sea fans and branching gorgonians, causing tissue loss and destruction of the skeleton (Nagelkerken et al., 1997). ASP is characterized by degradation and recession of coenenchyme, purpling of adjacent tissue, production of galls, and secondary colonization of exposed axial skeleton. Field identification of ASP may be difficult, as similar lesions, purpling of tissue and nodules, and skeletal and tissue loss occur in response to predation, abrasions, algal interactions and other factors. Confirmation of ASP requires identification of *A. sydowii*, which may not be visible without microscopy.

What corals are affected by disease and what are the impacts?

Records in the Global Coral Disease Database (GCDD) show that 33 species of stony corals, 8 gorgonians and two hydrozoans are affected by at least one disease. This list may not be comprehensive, as Weil (2004) indicates that at least 41 scleractinian corals have been affected by diseases. Some of these differences may be related to the taxonomy used (i.e. whether species are combined or split), or may reflect unpublished observations. Some conditions show geographic variability in occurrence, and susceptibility may vary among species for the different "types" reported (Richardson and Aronson, 2002; Weil 2004). *Montastraea annularis* (complex) is currently most severely impacted by coral diseases, being susceptible to at least eight syndromes (Weil, 2004), with single colonies showing signs of two or more diseases simultaneously (Bruckner and Bruckner, 2006). White plague (type I and II), the most virulent disease observed to date, affects 39 scleractinian corals (Weil, 2004). BBD has been reported on 25 scleractinian corals, 6 branching gorgonians and sea fans (Green and Bruckner, 2000). YBD primarily affects *M. annularis* complex, although other faviids, *A. agaricites*, and *P. astreoides* are reported with this condition (Gil-Agudelo et al., 2004). The various dark spot/band diseases (Appendix I) collectively affect 14 species (Gil-Agudelo et al., 2004; Weil, 2004). Skeletal anomalies affect at least 16 Caribbean scleractinian corals, one hydrozoan and five gorgonians, with acroporids being most susceptible to neoplasms, and faviids commonly exhibiting hyperplasms (Sutherland et al., 2004). Susceptibility of the major genera of reef building corals to the 7 most significant scleractinian coral diseases are shown in Fig. 1.

Table 1. Existing descriptions of gross signs of the primary Caribbean diseases

Syndrome	Diagnostics	Reference
WP type I	Lesions that expand at a rate of a few mm/day and often result in colony mortality. Lesions occur more variably across the colony surface including the edges and sides. Edges of lesions show a sharp boundary between apparently healthy tissue and freshly exposed skeleton with no build up of microorganisms or necrotic tissue visible to the naked eye. Mean rate of loss= 3 mm/day	Dustan 1977
WP type II	Freshly exposed coral skeleton with a sharp line between skeleton and apparently healthy coral tissue. No evident microbial community; a narrow (2-3 mm) zone of bleached tissue at the disease line. No skeletal damage. Similar to type I but faster progression and lesions always start at the base of the colony and advances to the apex. Max loss=2cm/day	Richardson et al., 1998
WP Type III	Lesions start in the center of the colony and expand outward. Tissue loss occurs as large patches on the sides of large (>2m) colonies. Rates of loss can exceed 10 cm/day	Richardson et al., 2001
WBD	A distinct band of white exposed skeleton separates live tissue and algal colonized dead coral. Tissue adjacent to lesion may appear healthy or form a narrow band of disintegrating coral tissue that is peeling off the skeleton. Tissue mortality starts near the base of a colony and where branches furcated, advancing towards branch tips at a rate of 1-21 mm/day (mean = 5.5 mm/day)[2]; it sometimes but not always encircles the entire branch. .No skeletal damage. The width of exposed skeleton varies depending on spreading rates, with older areas becoming progressively colonized by filamentous, turf, macro and coralline algae.	Gladfelter, 1982 [2]Davis et al., 1986
WBD II	A distinct band of white exposed skeleton separates live tissue and algal colonized dead coral. The lesion boundary is preceded by a 2-20 cm band of bleached tissue. The advancing lesion may "catch up" to the bleached margin, making the disease indistinguishable from WBD I. The disease progresses from the base to the branch tips, but can also progress from branch tips towards the base. Some dissolution of the skeleton may occur. Advance of up to 10 cm per day.	Ritchie and Smith,1998
WPX	Irregularly shaped distinct white patches devoid of coral tissue. Most lesions are small but can be > 80 cm^2 and can develop simultaneously on all surfaces of the coral colony. Lesions can merge, resulting in tissue loss that spans the entire colony. The denuded coral skeleton remains intact, but loss of corallites is common. Lesions enlarge along the perimeter at a rate of 2.5 (max= 10.5) cm^2/d	Patterson et al., 2002; Sutherland and Ritchie, 2004
BBD	Black mat/band on the surface of the coral that separates healthy tissue and white, tissue-denuded skeleton. The band consists of a microbial community (black, chocolate brown or reddish rust colored with white filaments) a few mms to cms in width, and 1 mm thick. The width of exposed skeleton varies with spreading rates, with older areas progressively colonized by algae. BBD progresses from single point (at the margin of the colony, or within the colony surface at the interface between previously killed skeleton and live tissue) and radiates outward in a circular or semicircular pattern The microbial consortium functions together to generate and maintain an environment of anoxia adjacent to living coral tissue, possibly causing tissue necrosis through a lack of oxygen and exposure to hydrogen sulfide. Tissue loss of up to 1 cm/day (mean=3 mm/day)	Rützler and Santavy 1983, Edmunds 1991, Richardson et al. 1997; Bruckner et al., 1997.
RBD-II	Small (2-3 cm) lesions on scleractinian coral colonies. Cyanobacterial filaments form a loose biofilm, or matrix, that is spread out over the lesion and onto adjacent healthy coral tissue during the day. At night, the filaments contract to form a tightly compacted band less than 1 mm wide that is closely associated with the edges of the lesion at the interface with apparently healthy coral tissue. Tissue loss= 1 mm/day	Richardson, 1992
RBD-I	A band or mat of filamentous cyanobacteria, 0.5-2.5 cm wide, that separate coral skeleton from live tissue; the band moves typically from the base to the tips.	Rützler et al., 1983

Syndrome	Diagnostics	Reference
YBD	Small circular area (s) of translucent light yellow tissue surrounded by fully pigmented tissue, or a narrow band of pale tissue at the colony margin that slowly expands in size. Tissue first affected (e.g., in the center of the blotch) gradually darkens and dies and these areas become colonized by algae. Recently exposed white skeleton may be absent or confined to small (<2 cm) irregular patches within the yellow band. Lesions expand 0.3-2 cm per month; multiple lesions appear on individual corals; these coalesce and continue spreading. Colonies show YBD signs for multiple years.	Bruckner and Bruckner, 2006
DSD	Small, round, dark spots that grow in size over time. Affected tissue may be depressed relative to the rest of the coral. Spots may expand into a ring surrounding dead coral[1] Rates of tissue loss for *S. siderea* were 0.51 cm^2/month and 1.33 cm^2/month for *M. annularis*[2]	[1]Garzón-Ferreira and Gil-Agudelo, 1998;
DSD-II	Similar to DSD-I except the "spots" were larger and may cover 90 % of the colony; Dark but healthy looking tissue up to 45 cm (diameter) associated with depressed skeletal areas. Darkened tissue eventually dies. Dead areas are usually associated with thin, necrotic tissue at the margin. The syndrome advances slowly but faster than DSS-I.	Weil 2004
DBS	Round or elongated bands of pale to dark, live tissue that is sometimes associated with depressed skeletal areas in the corallum. Starts as spots and develops into wide dark bands (1 – 2 cm) of ill-looking tissue. The bands advance from areas in the center or side of the colony towards the edge of the colony in most cases. In *S. siderea*, the syndrome advances from the edge to the center of the colony, or from one side to the other. Rates of advance are faster than those in DSS-I and DSS-II.	Weil 2004
PBS	Colonies have several purple spots over the surface, or a purple band that develops at the edge and moves to the center, leaving clean skeleton behind that is quickly colonized by turf algae. The width of the purple band is variable, but generally over 1 cm. The rate of advance is approx. 1 cm/month	Weil 2004
Tissue Necrosis	Similar to PBS, except colonies lack spots, band is wider and more irregular and tissue looks necrotic and peels off skeleton	Weil 2004
Calicoblastic Neoplasm	Raised (up to 1 cm) whitened, irregularly shaped lumps on upper or lower branch surfaces in any region of the coral colony, sometimes extending from one side of the branch to the other. Normal polyps absent from the center of the mass. Coenosteal skeletal material spreading upward between polyps at the margins, or over polyps toward the middle; coral tissue at the edges appears slightly swollen, ruffled, and lifted above the skeleton. Mean growth rate of 0.12 mm/d	Peters et al., 1986
Hyperplasm	Protuberant circular to ovoid lesions with enlarged skeletal elements relative to adjacent surfaces. Normal tissue features such as polyps and tentacles are present but enlarged. Pattern of the polyps, valleys, ridges may differ from the rest of the colony but tissue is usually similar in coloration to the rest of the colony.	Peters et al., 1986

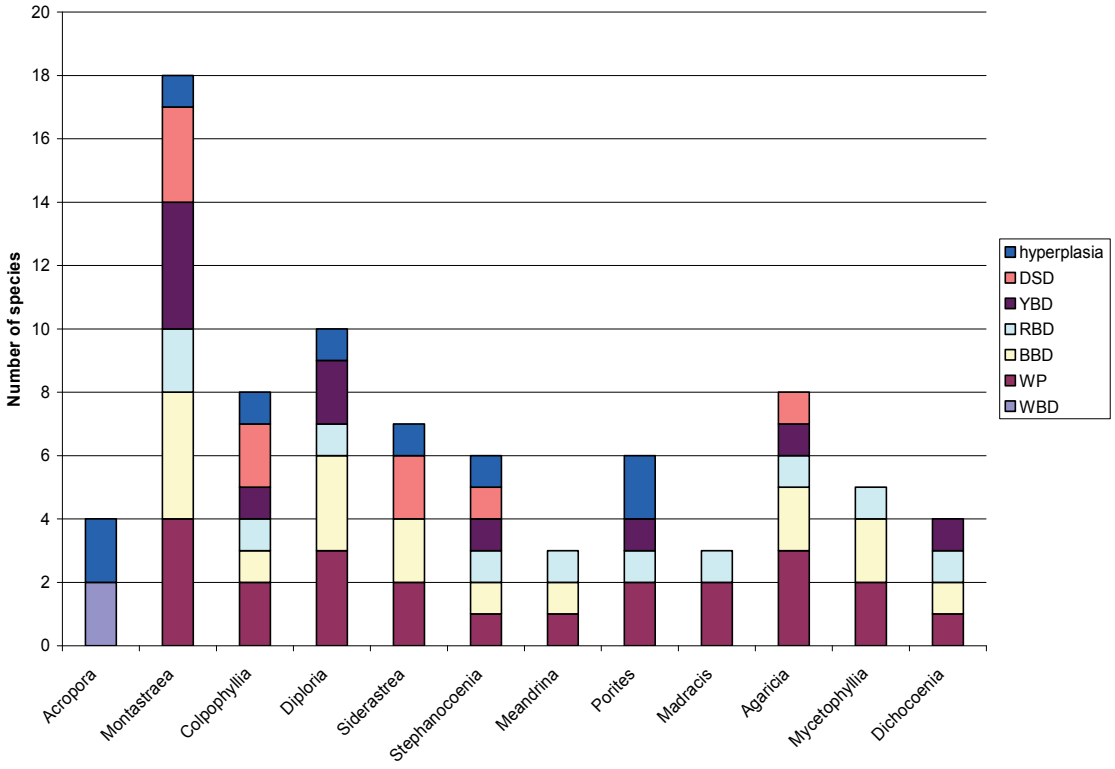

Fig. 1. Number of diseases affecting different genera of western Atlantic scleractinian corals.

The average prevalence of coral diseases at the community level is generally low, although it is highly variable between and within sites, during different years, and seasonally. Outbreaks of BBD, WP and WBD were identified during the 1970s and 1980s in Florida, Belize, Jamaica, USVI, Netherlands Antilles (Dustan, 1977; Rützler et al., 1983; Peters 1984; Rogers, 1985). In some locations, these diseases affected 50-80% or more of the corals, although outbreaks were largely confined to specific reefs or zones (e.g., the palmata zone). Over the last ten years, there has been an increase in monitoring efforts targeted at disease, and many of these report an increase in the prevalence and incidence of disease throughout the region over time, including outbreaks of WP-II, YBD, WPX and other emerging acroporids syndromes (Porter et al., 2001; Nugues, 2002; Miller et al., 2003; Weil, 2004; Bruckner and Bruckner, 2006). In one of the first Caribbean-wide surveys, prevalence rates in 9 locations ranged from 0.9% in Bermuda to 16% in Jamaica during 1999, with a higher number of diseased gorgonians (compared to scleractinian corals) and a significant increase in prevalence between 1999-2001 in Bermuda (4.3% in 2001), Puerto Rico (from 2.1% in1999 to 6.6% in 2001) and Venezuela (3% to 5.8%) (Weil, 2004). In contrast, more recent studies near Lee Stocking Island, Bahamas found very low levels of disease (<0.7% in 2002 and 2003; Voss and Richardson, 2006).

White band disease and other syndromes affecting acroporids

WBD was first documented on reefs around St. Croix, USVI, and later throughout the Caribbean. During the 1970s and 1980s, the prevalence of WBD varied from 1-2% to 26% in the British Virgin Islands (Davis et al., 1986), 33% in Parguera, Puerto Rico (Goenaga and Boulon, 1992), 40% in Florida and Belize (Antonius, 1981), 64% in the USVI (Gladfelter et al., 1977) and as high as 80% in Jamaica and the Netherlands Antilles (Rogers, 1985). Epizootics of WBD significantly reduced populations of acroporids throughout the region.

Although WBD was reported from 9 countries in the last ten years, most observations of WBD on *A. palmata* are for isolated colonies, with only one outbreak reported (Mona Island, Puerto Rico; Bruckner and Bruckner, 2005). In contrast, recent outbreaks of a more virulent disease (possibly a form of WBD, WBD-II, or some other syndrome) have been noted among *A. cervicornis* in Puerto Rico, Bahamas, Florida and other locations (Weil, 2004; Williams and Miller, 2005). Other conditions (WPX and other emerging syndromes) are also being observed more frequently on A. *palmata* since 1994. These are causing much more rapid rates of tissue loss (cm/day) than that reported for WBD in the 1970s and 1980s (5 mm/day). For instance, WPX lesions expand radially at their perimeters at an average rate of 2.5 cm^2/day and individual lesions may be greater than 80 cm^2 in area; lesions can develop simultaneously on all surfaces, and individual lesions often coalesce, resulting in tissue loss that spans the entire colony (Sutherland and Ritchie, 2004). In the Florida Keys populations of *A. palmata* sustained losses averaging 88% between 1996-2002, which was attributed primarily to WPX (Porter et al., 2001; Sutherland et al., 2004).

Black band disease

BBD is widely distributed throughout the Caribbean, with reports from 25 countries. BBD generally affects a low percentage of corals (<1%) at the community

level (Edmunds, 1991; Kuta and Richardson, 1996), but it occurs in most reef environments, and localized epizootics have been observed in the USVI, Jamaica, Florida and Puerto Rico (Peters, 1984; Bruckner and Bruckner, 1997; Bruckner, 1999 Bruckner, 2002). The disease may exhibit a clumped distribution (Kuta and Richardson, 1996; Bruckner et al., 1997), affecting up to 10 corals within a 2 m radius area (Peters, 1984). A greater percent of the corals may be affected by BBD in areas with high coral cover, and in habitats with a high density of colonies or dominance by susceptible species. For example, prevalence of BBD in St. John was low at the community level (0.2%), but much higher at the species level (5.5% of the *D. strigosa*); these sites had a high density of colonies, but susceptible species accounted for only 24% of the total coral composition (Edmunds, 1991). Florida reefs examined by Kuta and Richardson (1996) had a low density of corals (0.15 colonies/m^2) and relatively few infections (0.72%). Other sites examined in the Florida Keys ten years earlier (when they had 50-60% cover) had a mean prevalence of BBD of 6% (Dustan, 1987). Bruckner and Bruckner (1997) observed a maximum prevalence of 1.2% in Jamaica, although 5.2% of the corals became infected over 20 months. These sites had an intermediate density of corals (0.9 colonies/m^2), although over 90% of the corals were susceptible to BBD (Bruckner et al., 1997).

BBD typically advances at rates of about 3 mm/day (Rützler et al., 1983), and occasionally increases to a maximum of 1 cm/day (Antonius, 1981). Considerable variation in spreading rates is observed over the duration of individual infections (Rützler et al., 1983) and also between species, depths, seasons and locations (Bruckner, 2002). BBD occurs year round on tropical Caribbean reefs, while infections often disappear in winter months in Florida and other northern reefs, when temperatures decline below 20ºC. BBD can kill small (<50 cm^2) corals in several days while larger corals experience partial mortality before signs of BBD disappear (Bruckner, 2002). However, BBD may reappear later that season or the following year, and individual colonies can be affected by BBD for multiple years (Feingold, 1988; Kuta and Richardson, 1997; Bruckner and Bruckner, 1997). While BBD does not appear to have caused large die-offs of important reef-building corals, individual colonies lose substantial amounts of tissue that may affect their reproductive potential or their ability to resist other stresses (Edmunds, 1991; 2000). Kuta and Richardson (1997) noted that corals continue to lose tissue after signs of BBD disappear. In the USVI, 28 colonies identified with BBD in 1988 were tagged and followed for ten years; 50% of these were subsequently killed during a hurricane or died of unknown causes, 36% died from BBD, and 14% were still alive 10 years later (Edmunds, 2000). Regrowth of corals affected by BBD has been observed (especially in *D. strigosa*), although larger lesions in *M. annularis* and other species fail to completely regenerate (Bruckner, 2002; Weil, 2004).

White plague

White plague was first observed in Key Largo, Florida in the 1975, where it affected up to 7% of *S. siderea* colonies and 24-73% of *M. ferox* colonies at Carysfort Reef (Dustan, 1977). WP was still prevalent in 1984 on Carysfort Reef, and was also documented throughout the entire Key Largo region (inner, middle and outer reefs) at a mean prevalence of about 3.7% (Dustan, 1993). WP caused rates of tissue loss of up to 3.1 mm/day, and was estimated to have killed 20-30% of *M. ferox* population (Dustan 1977, 1987).

A more virulent form of WP (WP-II) was observed on reefs of the middle and northern Florida Keys in 1995. It initially affected 9-38% of *D. stokesi* colonies, and subsequently spread to 16 other species (Richardson et al., 1998a). The epizootic recurred in 1996 in the lower Keys and Dry Tortugas (although sites with outbreaks in 1995 had few infections in 1996) and in 1997 emerged on reefs north of the Florida Keys reef tract (Richardson et al . 1998b).

WP (type I and type II) has continued to spread throughout the region over the last 10 years, with reports from 21 countries. In La Parguera, Puerto Rico, WP first appeared in 1996 on inner middle and shelf edge reefs; it affected 5 genera, but was most severe on one inner reef where 47% of the *D. labyrinthiformis* colonies contracted WP between August and December (Bruckner and Bruckner, 1997). Subsequent outbreaks were observed in Puerto Rico in 1998, 2000, 2002 and 2006 (Bruckner, pers. Obs., Weil 2004; Hernandez, coral list posting).

WP-II first appeared on reefs around St. John, USVI in 1997, and has been since observed on most back reef (< 3m depth), fore reef slope (6-12 m) and deeper offshore habitats (24-32 m). It primarily affects *M. annularis* (over 90% of infections) but also *M. cavernosa*, *C. natans*, *S. siderea* and *Diploria* spp. are susceptible (Miller et al., 2003). New infections were observed every month over 4 years at Tektite Reef, although the incidence varied substantially (3-58%) over the duration of the study and was highest during the first year (Miller et al., 2003). An outbreak occurred on these and other reefs in St. John following the 2005 bleaching event (J. Miller, unpubl obs).

An outbreak of WP was also first observed on reefs near Soufriere, St. Lucia in July 1997 (Nugues, 2002). Eleven percent (range=7-14%) of the six dominant coral species were infected with WP on three reefs in March 1998. Over 8 months, WP was estimated to have killed 6.6% of the living coral tissue at the most affected site, with most of the tissue loss occurring on the two dominant species (*M. faveolata* and *C. natans*) (Nugues, 2002).

Yellow band disease

YBD was first reported from the Florida Keys in 1994 (Cervino et al., 2001), with subsequent observations from 24 countries. The incidence of YBD increased between 1996-2000 in Colombia, Mexico, the Netherland Antilles, Panama, Puerto Rico, Grenada, St. John, Turks and Caicos, USVI and Venezuela, where 18-91% of *M. annularis* (complex) colonies were affected (Santavy et al 1999; Cervino et al 2001; Bruckner and Bruckner 2003; Gil-Agudelo et al. 2004; Jordán-Dahlgren and Rodríguez-Martínez 2004; Bruckner and Bruckner, 2006). YBD currently appears to be most abundant in remote, offshore locations removed from human population centers (Bruckner and Bruckner, 2003; 2006; Weil, 2004).

Rates of mortality from YBD are generally slow (5-15 cm/yr), although colonies with single YBD lesions become infected in multiple locations, and infections can persist for over 5 years (Bruckner and Bruckner, 2006). In both Curacao and Puerto Rico, YBD appeared to preferentially target the larger corals in the population; over several years, live tissue area is progressively reduced in size, with infections persisting until the coral dies (Bruckner and Bruckner, 2006; Bruckner and Bruckner, in press).

Dark spots disease

DSD was first noticed in Colombian reefs in 1992 during a bleaching event (Solano et al., 1993), with the first quantitative study conducted in 1997 (Garzón-Ferriera and Gil, 1998). In this study, DSD affected > 16% of six species (over 1545 colonies); the two most abundant species (*M. annularis* and *S. siderea)* had the highest number of infections (Gil-Agudelo and Garzón-Ferreira, 2001). Cervino et al., (2001) reported prevalence rates of 42-56% for *S. intersepta* and *S. siderea* in Bonaire, Turks and Caicos, and Grenada. Gochfeld et al. (2006) reported a mean prevalence of 31.5% on St. Thomas, USVI, 50.3% on Culebra, Puerto Rico, and up to 80% in the Bahamas for *S. siderea*, with the highest incidence during August and sudden declines each year in October.

Spreading rates of DSD are generally low. In Colombia, loss was 0.51 cm^2/day for *S. siderea* and 1.33 cm^2/month for *M. annularis*. Recovery of lesions was not observed, and signs of the syndrome persisted for several years (Garcés-Baquero, 2000). Cervino et al., (2001) reported rates of tissue die back of 3.99 cm/month for 2 colonies of *S. siderea*. In the Bahamas, Gochfeld et al., (2006) did not observe any colony mortality from DSD over two years; small areas of necrosis were noted, but these areas regenerated relatively rapidly. In addition, dark spots disappeared from affected colonies in October, but they also often reappeared the following year in the same or different location (Gochfeld et al., 2006).

Aspergillosis

Aspergillosis is thought to have emerged in the 1980s along the coast of Costa Rica, Panama and Trinidad (Guzman and Cortes, 1984, Garzón-Ferreira and Zea, 1992, Laydoo, 1983), where it caused localized mass mortalities. Sea fans showing similar signs to the 1980s epizootics were reported from 22 locations throughout the Caribbean in 1995 (Nagelkerken et al., 1997). In the Bahamas, disease incidence and virulence increased between 1995-1998 (Smith and Weil, 2004). By 1999, Aspergillosis was identified on branching gorgonians (*Pseudoterigorgia*, *Plexaura*, *Pseudoplexaura* and *Plexaurella* spp.) in Puerto Rico and the Bahamas. Over the last 5 years ASP infections have been observed in 17 countries.

Prevalence of ASP appears to be greater in protected sites and in deeper areas on exposed reefs (Nagelkerken, 1997). In the Florida Keys, 31% of sea fans were affected in August 1997; declining numbers of infections were observed over the next seven years in all sites, except for three localized outbreaks during the summers of 1998, 2000 and 2002 (Kim and Harvell, 2004). Aspergillosis does not appear to have impacted the abundance of sea fans in Florida, although partial and complete mortality altered the size-class structure of the population by removing the large colonies (Kim and Harvell, 2004).

Table 2. Prevalence, incidence and impact of the major western Atlantic coral diseases.

Disease	Spatial distribution and prevalence	Temporal/ Spatial variations	Impact
WP	7% *of S. siderea*, 24-73% of *M. ferox* in Florida[1]; 20.1% of *D. stokesi* colonies in Florida[2] 11% of all species, 19% *M. faveolata* and 13% *C.natans* in St. Lucia[3]	Highest prevalence in late summer and early fall at temps of 29-30° C [1] In St. Croix, prevalence of 3.1% in clean site and 11.4% in polluted site[5]	20-30% mortality of *M. ferox* populations[1] 0.1-5% tissue loss over 26 months in the USVI during the 1980s [4] 9.4% of affected *D. stokesi* colonies died in 2 months[5]
WBD	2-5%; up to 40% in Florida, USVI, Belize[1]; 5-26% in BVI[2]; up to 64% in USVI[3]; 20-33% in Puerto Rico[4]		Contributed to a regional decline of Caribbean acroporids
WPX	Contagious; Clumped distribution[1], up to 100% of colonies may be affected[2] 35-73% affected in PR[3]	Greatest rate of tissue loss during warm water[1]	Rapid spread within and between reefs during the 1990s; killed 50-80% of elkhorn coral in certain areas in FKNMS[1]Average loss of tissue over 10 days was 17%[3]
BBD	Up to 8% of gorgonians at one time, 13.8% of all colonies over 26 months in Florida[1] 0.2% of all corals; 5.5% of *D. strigosa*[2] 5.2% total over 20 months; max 1.2% at one time[3]	Highest prevalence in summer in shallow locations; disappears in winter in Florida[1] and when temps drop below 27.5 C in the Bahamas[5] Temperature and light affect growth and spreading rates of BBD[3] In USVI higher prevalence (1% vs. 2.7%) in a polluted site [4] Sites with BBD had higher sedimentation rates[5]	Mean rate if tissue loss is 3 mm/day; in St. Croix, rates were 1.45 mm/day[4] 58% of *D. strigosa* colonies lost >75% of their tissue; overall loss of 3.9% of *D. strigosa* tissue per year[2]
YBD	Up to 12.5% *M. annularis* and 7.8% *D. strigosa* affected in Colombia in 1999[1] 35% of *M. annularis* in Mexico during 2001[2] up to 52% in Puerto Rico[3]	Higher spreading rates in summer. Sites affected between 1997-2002 shows a declining trend of new infections; older infections have persisted and new areas are being impacted.	Colonies affected with YBD in 1999 and 2000 lost 60% of their tissue by 2003 and most were still affected by YBD[3]
DSD	16% of six species; [1]; 42-56% of *S. intersepta* and *S. siderea*[2] Incidence on *S. siderea* varied from 81% on the deepest reef in May 2002 to 67% in a shallow site in Jan 2003[3]	Highest incidence July-Oct.; more infections in shallow water [1] In the Bahamas dramatic decrease in October, unrelated to temperature and depth [3]	Tissue loss of 4 cm/month in *S. siderea*[2] No significant loss over two years; small lesions recover[3]
ASP	39% of sea fans in Caribbean in 1995-1996[1]; 31% in Florida in 1997, declining to 5.9% by August 2003[2] In Mexico prevalence declined from 12.9% in 2002 to 5.3% in 2004	Prevalence higher in protected sites and deep water [1]	Sea fan density has remain constant; Keys-wide reduction in height of sea fans (40 cm – 26 cm) and >50% of the tissue area over six years [2]

WP: [1]Dustan, 1977; [2]Richardson, 1995; [3]Nugues, 2002; [4]Bythell et al.,1993; [5] Kaczmarsky et al., 2005.
WBD: [1]Antonius, 1981 [2]Davis et al., 1986 [3]Gladfelter et al., 1977 [4]Goenaga and Boulon, 1992 [5]Rogers, 1985
WPX: [1]Patterson et al., 2002; [2]Sutherland and Ritchie, 2004 [3]Weil, 2004 [4] Kaczmarsky et al., 2005.
BBD: [1] Feingold, 1988. [2]Edmunds, 1991; [3]Rützler et al., 1983 ; [4] Bruckner et al., 1997
YBD: Cervino et al., 2001 Gil-Agudelo et al., 2004; Bruckner and Bruckner 2003; Bruckner and Bruckner, 2006
DSD: [1]Gil-Agudelo and Garzón-Ferreira, 2001; [2]Cervino et al., 2001[3] Gochfeld et al., 2006
ASP: [1]Nagelkerkin et al., 1997; [2]Kim and Harvell, 2004

What is causing these diseases and where are they coming from?

Proving disease causation has been one of the largest challenges in coral disease research. Because diseases manifest on corals in a limited number ways, corals that exhibit specific disease signs in the field may be affected by a variety of pathogens that differ spatially or temporally (e.g., the pathogen for WP-II may differ depending on the location, affected species, or other factors). To date, causative agents have been identified for four of the most common diseases found on Caribbean reefs (BBD, WP-II, ASP, and WPX). Three of these (WP-II, ASP, WPX) were verified through inoculation experiments with cultured bacterial isolates (through fulfillment of Koch's postulate), while the cause of BBD was identified using microscopy. In other diseases (YBD, DSD, WBD-II) screening of microbial communities of healthy and diseased tissue (and mucus layer) using traditional culture methods illustrates a high diversity of microorganisms, along with several bacteria (especially *Vibrio* spp.) that appear to be more prevalent in diseased samples (Cervino et al., 2001; Gil-Agudelo et al., 2004; Weil, 2006) . Molecular studies (16S and 18S rRNA/DNA gene sequence amplification of microbial communities) have identified complex multi-species microbial communities in corals (including microorganism that may be unculturable) that appear to vary spatially, seasonally, between species, and also between diseased and apparently healthy parts of the colony (Rohwer et al., 2002; Pantos et al., 2003; Pantos and Bythell, 2006). In many cases (e.g., WP-II, BBD and WBD) these molecular studies have identified a different suite of organisms as potential causative agents than that observed in earlier studies.

Black band disease

The causative agent of BBD was originally described as the cyanobacteria *Oscillatoria submembranaceae* and then *Phormidium corallyticum* based exclusively on filament morphology, pigmentation and motility determined using light microscopy (Antonius, 1981; Rützler et al., 1983). Other heterotrophic bacteria (Garrett and Ducklow, 1975), the sulfide oxidizing bacterium *Beggiatoa* spp. (Ducklow and Mitchell, 1979) and marine fungi (Ramos-Flores, 1983) have also been suggested as the primary pathogen. Richardson and colleagues described BBD as a consortium of microorganisms dominated by a gliding filamentous cyanobacteria (*P. corallyticum*) that functions together with sulfur oxidizing bacteria (*Beggiatoa* spp.) and sulfur reducing bacteria (*Desulfovibrio* spp.) to produce anoxia and high levels of sulfide adjacent to the coral, conditions that are lethal to the coral (Carlton and Richardson, 1995; Viehman et al., 2006). More recent work using 16S rRNA gene sequencing identified a complex and variable assemblage of heterotrophic organisms that includes over 500 species of bacteria as well as cyanobacteria (Cooney et al. 2002, Frias-Lopez et al. 2002, 2003). These molecular studies identified anomalies in the identification of the cyanobacteria: three unique taxa of cyanobacteria have been isolated, with differences noted between the Caribbean and IndoPacific. Interestingly, *P. corallyticum* was not detected in the clone libraries or evident in the DGGEs (Cooney et al. 2002, Frias-Lopez et al. 2002, 2003).

White plague

The disease pathogen for WP II was identified as a gram negative α-proteobacterium (a new species of *Sphingomonas* later renamed *Aurantimonas*

coralicida; Denner et al., 2003) based on culture of a bacterial isolate obtained from a single diseased *D. stokesi* colony and subsequent inoculation on two healthy colonies of *D. stokesi* (Richardson et al., 1998a). This same microbial pathogen was subsequently reisolated from another affected colony at a later date. Although both strains are in culture, apparently healthy colonies no longer appear to be susceptible to either the original or newly isolated strains of the pathogen. This suggests that bacteria may lose virulence when in culture or *A. coralicida* is not in fact the causative agent. Another study examining a plague-like disease on *M. annularis* colonies from the USVI and Barbados (using bacterial 16s rDNA genes) identified a high diversity of bacteria in diseased samples, and differences between diseased and healthy tissue (Pantos et al., 2003). While *Sphingomonas* spp. was detected in healthy corals and control areas on diseased colonies, it was absent from diseased areas. Instead, an α-proteobacterium most closely related to the causative agent of juvenile oyster disease was present in diseased tissues, but consistently absent from healthy tissue (Pantos et al., 2003).

A fluorescent probe specific for *A. coralicida* has tested positive on colonies with signs of WP-I and WP-II in a number of locations including the USVI, Puerto Rico (Miller et al., 2003; Weil, 2004), suggesting that this bacterium is widely distributed throughout the region.

White band disease

A causative agent for WBD type I has not been identified. Using histology, Peters et al., (1983) identified gram negative rod-shaped bacterial aggregates in the calicoblastic epidermal tissue of *A. palmata* colonies with WBD from the USVI and Bonaire. These bacterial bodies were also found in acroporids without signs of WBD, although the counts of bacteria per area were significantly less (Peters, 1984). Santavy et al., (1995) found bacterial aggregates in some but not all colonies of *A. cervicornis* with WBD on one reef in the Bahamas, while corals on a neighboring reef did not contain bacterial aggregates (Santavy and Peters, 1997). Bacteria were absent from WBD-affected *A. cervicornis* colonies in Florida (Kozlowski, 1996).

Studies of the bacterial community of WBD-II has concentrated on the surface mucopolysaccharide layer and the use of preferential carbon utilization methods. A bacterium most closely related to *Vibrio carchariae* was identified as a possible cause, and these were found to increase in number with the onset of disease WBD II (Ritchie and Smith, 1995).

Microbial communities identified using 16s RNA techniques were found to differ substantially (only 10% similarity) between healthy and WBD *A. palmata* colonies. Healthy and diseased tissues both contained similar proportions of four predominant bacterial groups, although planctomycetes, cyanobacteria and Cytophaga-Flexibacter-Bacteroides group were found only in diseased samples (Pantos and Bythell, 2006).

Yellow band disease

The causative agent for YBD is unknown. Cervino et al. (2001) proposed that YBD is a zooxanthellae disease which can kill coral by damage produced by the symbiont, based on a finding of a reduction of the number of zooxanthellae (41 to 97%) and reduction in the mitotic index of zooxanthellae (2.5% to 0%). Several bacterial

strains metabolically related to the genus *Vibrio* have been found in the mucus associated with diseased tissue (Gil-Agudelo et al., 2004).

Dark spots disease

The causative agent for DSD is not known. Differences in the structure of the microbial community from the mucus of healthy and diseased *M. annularis* and *S. siderea* colonies were identified based on the metabolic profile of culturable bacteria (Gil-Agudelo et al., 2004). A total of 17 groups of bacteria were identified, 16 of which were found in both diseased and healthy tissue, and one (*V. carchariae*) that was only observed in diseased samples. Healthy colonies inoculated with this bacterium failed to manifest signs of DSD (Gil-Agudelo et al., 2004).

Aspergillosis

Perhaps one of the most comprehensive etiologic studies of a coral disease in the Caribbean involves Aspergillosis. The fungus *Aspergillus sydowii* was identified as the cause of this disease through the use of transfection experiments, fungal cultures, morphologic and metabolic characteristics and 18s rDNA gene sequencing (Smith et al., 1998; Alker et al., 2001). *A sydowii* has been isolated from benthic environments near the Orinoco River, and pathogenic strains have also been collected in the USVI during African dust events (Smith and Weil, 2004).

Table 3. Causative agents and associated microorganisms reported for western Atlantic coral diseases.

Disease	Reported causes and associated organisms
WBD	unknown ; gram negative rod-shaped bacterial aggregates in the calicoblastic epidermal tissue[1]; blue green algae, ciliates, turbellarian flatworms, copepods, amphipods, nematodes [2]
WBD II	*Vibrio harveyi/carchariae*[a]
WPX	*Serratia marcescens*[a] , a common gram-negative bacterium classified as a coliform and a member of the Enterobacteriaceae family; fish feces proposed as a vector[2]
WP I	Unknown, Gram negative bacteria
WP II	*Aurantimonas coralicida*[a] , an obligately aerobic,polarly flagellated gram negative bacterium[1]; α-proteobacterium closely related to the causative agent of juvenile oyster disease[2]
BBD	*Oscillatoria submembranopora*[1] The cyanobacteria *Phormidium corallyticum*[2] in association with sulfate reducing bacterium *Desulfovibrio* spp, and sulfide oxidizing bacterium *Beggiatoa*[2] *Oscillatoria* spp and *Trichodesmium tenue*[3] Other cyanobacteria (*Oscillatoria, Spirulina, Lyngbya, Arthrospira* and other *Phormidium* species), pennate diatoms, ciliates, flagellates, and marine fungi occur in the band
RBD-I	Cyanobacteria *Schizothrix mexicana* and *S. calcicola*
RBD-II	Cyanobacteria; Two species of *Oscillatoria* characterized by filaments that are wider than they are long; filaments have two rounded tips
YBD	Undescribed *Vibrio*
DSD	Unknown; Over 250 bacteria were isolated from mucus of healthy and diseased *M. annularis* and *S. siderea* colonies; A bacterium most closely related to *V. carchariae* was isolated from diseased corals.
Tumors	Unknown. May be mutations of the genome or programmative changes in gene expression of the coral cells. The role of environmental parameters (e.g., UV radiation) has not been determined.
ASP[a]	*Aspergillus sydowii*[1] *A. terreus, A. niger* and *A. flavus*[2]

WBD: [1]Peters et al.,1983 [2]Gladfelter et al., 1977 WBD II: Ritchie and Smith, 1998; Weil, 2006
WPX: [1]Patterson et al., 2002. [2] Weil, 2004
WP: Dustan, 1977
WP-II: [1]Denner et al., 2003 [2]Pantos et al., 2003
BBD: [1]Antonius, 1973; [2]Rutzler and Santavy, 1983; Richardson et al., 1995; [3]Cooney et al. 2002, Frias-Lopez et al. 2002, 2003
RBD-I: Santavy and Peters, 1997
RBD-II Richardson, 1993
YBD: [1]Cervino et al., 2004; [2]Gil-Agudelo et al., 2004
DSD : [1]Gil-Agudelo et al., 2004 [2]Jordan-Dahlgren and Rodríguez-Martínez, 2004,
ASP : [1]Smith et al. 1996, Geiser et al. 1998, Alker et al. 2001 [2]Toledo-Hernandez et al., 2004

[a]Causative agent identified through fulfillment of Koch's postulate

Where are these pathogens coming from?

External sources of pathogens that have been proposed include terrestrial runoff (*Aspergillus*), sewage (*Serratia marcescens*), and African dust events (*Aspergillus*). Pathogens may also already be present in the marine environment. Reservoirs of *P. corallyticum* (along with other cyanobacteria) occurred as biofilms on the surface of sediment patches present in depressions on healthy *M. annularis*, *M. cavernosa* and *C. natans* colonies (Richardson, 1996). These may be dispersed via movement of water masses, especially during storms (Bruckner and Bruckner, 1997), and transmitted through various vectors like damselfish, parrotfish, fireworms and coral-eating snails (Williams and Miller, 2005; Aeby and Santavy, 2006).

Bacteria also occur on corals in a non-infectious state. Coral mucus is a rich protein-carbohydrate complex that harbors a diverse community of bacteria and other microbiota, and these communities are known to be distinct from the surrounding water (Rohwer and Kelley, 2004). Bacterial diversity varies between healthy corals, healthy parts of diseased corals, and diseased tissue (Rohwer et al., 2002; Pantos et al., 2003). Environmental changes can affect the physiological equilibrium between bacteria associated with the corals and their hosts, or stimulate the growth of other bacteria (Pantos et al., 2003). The coral-microbe relationship can be disrupted by nutrient and organic carbon loading by overstimulating the growth of these microbes, which may result in coral mortality (Kuntz et al., 2005). Under stressful conditions one or more of these microbes may become virulent or affect the resistance of the host, and subsequently trigger onset of an infectious disease.

Are disease outbreaks associated with changing environmental conditions?

Diseases may be infectious (produced by parasites and pathogens) or non-infectious (genetic mutations, produced by environmental factors). The frequency and severity of infectious diseases may affected by changing environmental conditions (elevated SST, declining water quality), human induced alterations of the marine environment (e.g., input of land-based pollutants; dredging, coastal development), and hurricanes and other natural disturbances. Increased temperatures may cause physiological stress and/or trigger the development of pathogenic agents that otherwise would remain non virulent.

Increased abundance and virulence of at least five diseases (BBD, WPX, DSD, ASP and YBD) has been associated with elevated seawater temperatures, with declines in these conditions reported during winter months (Kuta and Richardson, 2002, Alker et al., 2001; Patterson et al., 2002, Gil-Agudelo and Garzón-Ferreira, 1999 and Weil, 2004). Disease outbreaks may also be more severe during or immediately following bleaching events due to a lower resistance of host corals. Widespread and severe outbreaks of WP-II were observed in Puerto Rico, USVI and the eastern Caribbean following the 2005 Caribbean bleaching events (J. Miller, coral list posting; Weil, 2006). Other natural factors, such as habitat characteristics, composition, cover and abundance of susceptible corals, the amount of macroalgae, and presence/absence of certain key indicator species such as *Diadema* may also influence the occurrence and severity of coral diseases. Patterns of disease distribution obtained from the Global Coral Disease Database have shown that 97% of the areas affected by disease in the Caribbean prior to 2000 correlate

to areas where human activities have medium to high impact (Green and Bruckner, 2000). Despite the contention that deteriorating water quality associated with land-based inputs of pollutants and sediments and other human impacts is linked to disease outbreaks, there is minimal quantitative data to support this hypothesis, and links to specific disturbances are unclear (Bruckner, 2002).

Black band disease

Goreau et al. (1998) reported that BBD often first appeared in polluted areas and infections spread radially outward. They suggest that the abundance of BBD mimics the distribution of human influenced areas, with the largest impacts near sewage outflows and areas of high turbidity. Peters (1993) also noted that BBD prevalence is related to adverse environmental conditions, including warmer than normal temperatures, nutrient loading, increased sedimentation and turbidity, predation, and toxics. In Jamaica, the incidence of BBD progressively increased over 19 months, with the largest increase during or just after a period of unusual rainfall and run-off (Bruckner et al., 1997a). In this study, one species that is generally resistant to BBD (*S. siderea*) exhibited few infections prior to the rainfall event, with a dramatic increase in bleaching, WP and BBD in the second year of the study, corresponding to periods of high rainfall and run-off (Bruckner, et al., 1997).

An extensive, multi-year study evaluating BBD incidence on reefs off southern and western Puerto Rico failed to identify direct relationships between BBD prevalence and poor water quality (Bruckner, 1999). The lowest prevalence of BBD overall was found near Mayaguez and Ponce, which are the most polluted and turbid sites in Puerto Rico due to high sedimentation and nutrification associated with river discharge, agricultural runoff, and direct input of untreated sewage. On a fringing reef off the west coast (Rincon), BBD incidence was highest in spring (May-June) when water clarity was high, with infections disappearing during the rainy period (July-August) when run-off increased and visibility declined, even though temperatures were approaching their annual maxima. High turbidity also appears to limit the spatial distribution of BBD in southwest Puerto Rico (La Parguera). Infections were restricted to shallow water (<8 m depth) on turbid inshore reefs, even though species susceptible to BBD occurred in shallow and deep water, while BBD occurred to depths of 30 m on offshore shelf edge reefs with high water clarity. The disease was also common in remote locations around Mona Island, which is 70 km from the mainland of Puerto Rico and lacks permanent inhabitants, industry, agriculture or river discharge (Bruckner, 1999).

In contrast to Bruckner (1999), Voss and Richardson (2006) reported higher sedimentation rates on sites with BBD in the Bahamas. Some of the differences between this study and Bruckner (1999) may be related to the scale of inputs: sites near LSI Bahamas described as having high rates of sedimentation have relatively low levels of sedimentation and high water clarity, when compared to coastal reefs near Puerto Rico. Kaczmarsky et al., (2005) also observed a significantly higher prevalence of BBD in a site off St. Croix, USVI exposed to sewage discharge, as compared to an ecologically similar location upstream from the pollution effluent.

White plague

WP is reported to be seasonal on some northern reefs (Bahamas and Florida), while infections occur year round in USVI, Puerto Rico, Curacao and other locations (Richardson et al., 1998a; Miller et al., 2003; Weil, 2004; Bruckner and Bruckner, in press) and outbreaks have been observed during the coldest time of the year in the Flower Gardens (Hickerson, coral list posting) and in St. John (J. Miller, pers. Comm.). In St. Croix, USVI, Kaczmarsky et al. (2005) observed a much higher prevalence of WP (11.4% in a site affected by sewage discharge, when compared to a site located upstream from the effluent (3.1%).

White band disease

Few data are available to verify the role of environmental factors on WBD prevalence or severity. Outbreaks of WBD have been reported from throughout the region which has spread through acroporids reefs in both nearshore areas impacted by human settlement as well as remote locations and protected watersheds.

White pox

The prevalence and rates of tissue mortality are greater during warm water months (Sutherland et al., 2002). Sewage effluent is the proposed source of the WPX pathogen (Patterson et al., 2002), but no information is available on the prevalence and/or severity of WPX in polluted versus unpolluted sites.

Yellow band disease

YBD progresses more rapidly during warm water periods (Gil Agudelo et al. 2004). Although nutrient enrichment has been shown to increase the rate of tissue loss from YBD (Bruno et al., 2003), YBD is currently most abundant in remote locations or reefs subjected to low levels of human impact (Bruckner and Bruckner, 2006).

Dark Spots Disease

DSD was found to be more prevalent when water temperatures are over 28ºC and in shallow (<10 m depth) reef habitats in Colombia (Gil-Agudelo et al., 2004). In contrast, Gochfeld et al. (2006) did not find a correlation between DSD and water temperature; infections dramatically declined each year in October (which is just after the warmest time of year) and new infections emerged beginning in January of each year (close to the coldest water temperatures of the year). There also was no relationship with depth and DSD prevalence in the Bahamas. In 2002, the highest prevalence was observed at the deepest site (81.25%), while the highest prevalence was in a shallow (<5 m) site (67%) in January, 2003 (Gochfeld et al., 2006). Nutrient enrichment also did not appear to affect the prevalence or severity of dark spots disease in laboratory studies, although increased nutrients did induce bleaching (Gochfeld et al., 2006).

Aspergillosis

Aspergillus sydowii exhibits maximal growth at 30°C and is less affected by the hosts defenses at 30ºC than at 25ºC (Kim and Harvell, 2004). Nutrient enrichment (Nitrates) was shown to increase the progression of Aspergillosis (Bruno et al., 2003).

What have we learned from Caribbean coral disease research?

Research on coral diseases requires an approach that combines ecological monitoring with biochemistry, molecular biology, histology, toxicology, physical oceanography, ecology, taxonomy and other laboratory and field methods. An interdisciplinary approach is necessary to identify, differentiate and characterize coral diseases and their consequences, and understand relationships among diseases and other biotic and abiotic factors.

- **Epizootiology:** To understand the spatial extent of diseases at local to global scales, large areas of reef must be examined at the same time. Surveys must also be conducted at frequencies that are sufficient to document the duration of the condition, and identify seasonal patterns or chronic effects. Monitoring programs should include size measurements and colony condition (amount of recent and old mortality), and follow individual colonies over time to determine the severity of disease and potential population level impacts. Efforts should be made to standardize monitoring approaches.
- **Relationships between disease outbreaks and environmental factors:** Epizootiological studies must be combined with an examination of climate parameters (e.g., temperature and light levels), water quality measures (levels and types of nutrients and contaminants, turbidity, and rates of sedimentation), and impacts of other natural disturbances (e.g., predator outbreaks and hurricanes).
- **Rapid response program:** A coordinated rapid response to disease outbreaks can allow for the timely recognition, characterization, and reporting of disease outbreaks. This information is necessary so managers can 1) quickly direct resources to additional studies that are needed to identify appropriate management responses; 2) identify possible responses to control or mitigate the outbreak and 3) educate local and regional stakeholders on the condition of the corals.
- **Coral disease diagnostics and nomenclature**. There is a need to review and refine existing terminology and develop an approach for naming new diseases to reduce confusion. Disease nomenclature must include descriptive terminology of the gross signs (visible by an unaided eye, underwater) that could be applied by all scientists conducting epizootiological studies. Lesions should first be categorized as tissue loss, growth anomaly, and/or change in coloration. For each lesion, relevant information on the distribution of the lesions (e.g., focal or multifocal), location on colony, lesion shape, relief, texture, color, and size, and structures affected (e.g. tissue, individual polyps, coenosarc, skeleton) should be included in the morphologic diagnosis. By following affected colonies over time, more detailed information on patterns and rate of spread and extent of tissue loss can be compiled. This field terminology can be further refined once a causative agent is identified.
- **Identification of the causative agents** Molecular approaches should be combined with traditional culture techniques to identify and verify coral associated microbes, including spatial and temporal fluctuations, variations among species, and differences between healthy and diseased corals. Screening must include larger sample sizes (especially when isolating a putative pathogen and testing infectivity of proposed pathogens), multiple species (if they show similar signs), and multiple locations. There is an urgent need for molecular probes that will allow rapid screening of corals.

- **Application of new approaches and tools.** More emphasis needs to be placed on understanding processes and factors that may improve the resistance and resilience of the coral hosts, such as antimicrobial activities, immune responses, and regeneration processes**.** Efforts should also include a cellular diagnostics approach to identify stress and its underlying causes, and identify biomarkers that can characterize the condition of the coral and normal ranges of these biomarkers.

Appendix 1. Diseases reported to affect scleractinian corals and gorgonians on coral reefs in the tropical western Atlantic. The terminology highlighted in bold represents the proposed nomenclature identified at the 2004 CDHC workshop in Madison, Wisconsin.

Condition	Synonyms	Host range	Source
White syndromes			
White band disease (WBD)	White line disease; white death; white plague	*Acropora palmata* *A. cervicornis*	Gladfelter et al., 1977
WBD type II	White band disease	*A. cervicornis*	Ritchie and Smith, 1995; Weil 2004
Plague type I	White plague, white band disease; plague-like[2] Stress-related necrosis[3]	12 species of massive and plating corals	Dustan, 1977; [2]Pantos et al., 2003 [3]Peters, 1984
Plague type II	White plague, white band disease, white line disease	*D. stokesi;* 40 other species of plating and massive corals	Richardson et al., 1995; Weil, 2006
Plague type III	**Plague type II**	large massive corals (*M. faveolata, C. natans*)	Richardson and Aronson, 2001
Shut-down reaction (SDR)	Rapid tissue necrosis (in aquaria)	massive corals, acroporids	Antonius, 1977
White pox (WPX)	**White patch disease; acroporid serratiosis; white pox serratiosis**, patchy necrosis	*A. palmata*	Porter, 1996; Patterson et al., 2002; Sutherland and Ritchie, 2004
Patchy necrosis[1]	**White patch disease**; white pox, necrotic patch syndrome[2]	*A. palmata*	[1]Bruckner and Bruckner, 1997; [2]Rodríguez-Martínez et al., 2001
Cyanobacterial mat diseases			
Black band disease (BBD)	Black line disease	24 scleractinian corals, 1 hydrozoan, 6 gorgonians	Antonius, 1973
Red band disease type I (RBD)	Red band disease	*Gorgonia*, *Colpophyllia, Agaricia*, *Mycetophyllia Stephanocoenia*	Rützler et al., 1983 Santavy and Peters, 1997
RBD type II	Red band disease	*D. stigosa, M. annularis, M. cavernosa S. radians, P. astreoides*	Richardson, 1992

Condition	Synonyms	Host range	Source
Tissue Discoloration			
Yellow band disease (YBD)	Yellow blotch disease; ring bleaching[1], yellow pox disease[2]; yellow band syndrome[3]	*M. annularis complex,* other faviids *; A. agaricites* [4] ; *P. astreoides*[5]	Reeves, 1994;[1]Dustan 1977; [2]Garriet Smith; [3]Foley et al., 2004; [4]Gil Agudelo et al., 2004; [5] Sutherland et al., 2004
Dark spots disease (DSD)	Dark spot disease, dark spot syndrome, Ring disease [2]	*M. annularis* (complex), *S. siderea, S. intersepta* and *Agaricia agaricites*	Gil-Agudelo and Garzón-Ferreira, 2001; [2]Agudelo et al., 2004; Gochfeld et al., 2006
Dark spots disease Type II (DSD- II)	Dark spots disease	*S. intersepta; M. annularis; M. faveolata; M. cavernosa; C. natans; C. amaranthus; S. siderea*	Weil, 2004
Dark band syndrome (DBS)	Dark spots disease	*M. annularis; M. faveolata*	Weil, 2004
Purple band syndrome (PBS)	Dark band syndrome	*S. siderea, S. intersepta*	Weil 2004
Tissue necrosis	Dark spots disease	*M. faveolata*	Weil 2004
Mottling syndrome	bleaching	*C. natans*	Borneman, 2005
Pale ring syndrome	bleaching	*Montastraea, Colpophyllia, Diploria*	Borneman, 2005
Light patch syndrome	bleaching	*D. strigosa*	Borneman, 2005
Bleaching	Blanching	All zooxanthellate corals	
Abnormal growth			
Hyperplasia	**Growth anomaly**, tumors, Gigantism, area of accelerated growth, chaotic polyp development	*Diploria*, *Colpophyllia*, *Montastraea*, *Agaricia*, *Porites, Dichocoenia, Madracis*	Loya et al., 1984
Calicoblastic epithelioma	Growth anomaly, tumor, neoplasm,	*A. palmata*	Peters et al., 1986
Algal tumors	Tumor-like growth, tumor, algal tumor, gorgonin pearl, nodule, galls	*Gorgonia* spp. *Pseudoplexaura; Plexaura*	Morse et al., 1977
Skeletal damage			
Skeletal eroding band	Follicullinid ciliates	10 species: *Dichocoenia, Montastraea, Acropora,*	Croquer et al., 2006; Weil et al., 2006
Rapid wasting disease (RWD)[1] (Rapid wasting syndrome)	parrotfish white spot biting[2]; **parrotfish spot biting; parrotfish focused biting**[3] Rhodotorulosis [4]	*Montastraea* spp., *C. natans*	[1]Cervino et al., 1997; [2]Bruggeman et al., 1994 [3] Bruckner and Bruckner, 2000; [4] Richardson, 2000
Ridge mortality disease (RMD)	Damselfish ridge denuding syndrome[2]	*C. natans, D. strigosa*	Abbott, 1979, [1]Zimmerman 1994 [2]Williams et al., 2000
Tissue and skeletal loss, discoloration of tissue and abnormal growth			
Aspergillosis	Sea fan disease	*Gorgonia* spp.	Nagelkerken et al., 1997

Appendix II. Other abnormal conditions observed infrequently in scleractinian corals and gorgonians in the tropical western Atlantic. Some of these require histological analysis for confirmation (Coccidiosis and Nematopsis spores), while others are unconfirmed syndromes.

Syndrome	**Synonyms**	***Host species***	**Description**	
Coccidiosis	Coccidian infection	8 species: *Agaricia, Dendrogyra, Diploria, Montastraea, Meandrites, Porites*	Parasite infection: Oocysts found in mesentarial filaments; causes loss of zooxanthellae, patchy bleaching and tissue necrosis	Upton and Peters, 1986
Nematopsis spores	Sporozoan (protozoan) infection	*Porites* spp.	Thick walled ovoid ovoid capsules in calicoblastic epithelium	Peters, 1984
Ring syndrome	hyperplasia	*D. labyrinthiformis*	Fast growth of tissue and skeleton at ridge areas produces high, pale and thin ridges over the colony. Tissue inside ridges slowly dies	Weil, 2004
Fire coral fungal disease		*Millepora* spp.	Associated with bleaching	TeStrake et al., 1988
Thin dark line	Blistering necrosis [2]	8 species: *Diploria. Montastraea, Porites, Siderastrea*	Thin dark line at the boundary of living tissue that advances <1 cm/year	Jordan-Dahlgren and Rodriguez-Martinez, 2004; [2]Peters, 1984
White spot syndrome	Spot biting	massive corals	Predation by parrotfish referred to as "spot biting [1]"	Global Coral Reef Alliance ; [1]Bruckner and Bruckner., 2002
Star coral polyp necrosis (SCPN)		*M. cavernosa*	No further information presented	Williams and Bunkley-Williams, 2000

References

Abbott, R.E. 1979. Ecological processes affecting the reef coral population at the East Flower Garden Bank, northwest Gulf of Mexico. Ph.D. thesis. Texas A&M Univ., 154 pp.

Adey, W.H. 1978. Coral reef morphogenesis: A multidimensional model. Science. 202:831-837.

Aeby, G.S., and D.L. Santavy. 2006. Factors affecting susceptibility of the coral *Montastraea faveolata* to black-band disease. Mar. Ecol. Prog. Ser. 318:103-110.

Alker, A.P., G.W. Smith, and K. Kim. 2001. Characterization of *Aspergillus sydowii* (Thom et Church), a fungal pathogen of Caribbean sea fan corals. Hydrobiologia. 460:105-111.

Antonius, A. 1973. New observations on coral destruction in reefs. Assoc. Isl. Mar. Lab. Carib., Univ. Puerto Rico. Mayaguez. Abstr. 10:3.

Antonius, A. 1977. Coral mortality in reefs: a problem for science and management. Proc. Third Intern. Coral Reef Symp. 617-623.

Antonius, A. 1981a. Coral Reef Pathology: a review. Proc. Fourth Intern. Coral Reef Symp. 2:3-6.

Antonius, A. 1981b. The "band" diseases in coral reefs. Proc. Fourth Intern. Coral Reef Symp. 2:7-14

Antonius, A. 1999. *Halofolliculina corallasia*, a new coral-killing ciliate on Indo-Pacific reefs Coral Reefs 18:30.

Aronson, R.B., and W.F. Precht. 1997. Stasis, biological disturbance, and community structure of a Holocene coral reef. Paleobiology. 23 (3):326-346.

Aronson, R.B., and W.F. Precht. 2001. White-band disease and the changing face of Caribbean coral reefs. Hydrobiologia, 460:25-38

Bak R.P.M., and S.R. Criens, 1981. Survival after fragmentation of colonies of *Madracis mirabilis, Acropora palmata* and *A. cervicornis* (Scleractinia) and the subsequent impact of a coral *disease.* Proc. Fourth Intern. Coral Reef Symp. 2:221-227.

Bak, R.P.M. 1983. Neoplasia, regeneration and growth in the reef-building coral *Acropora palmata.* Mar. Biol. 77: 221-227.

Borneman, E.H. 2005. Pathologies affecting reef corals at the Flower Garden Banks, northwest Gulf of Mexico.

Bruckner, A.W., and R.J. Bruckner. 1997a. The persistence of black-band disease in Jamaica: impact on community structure. Proc. Eighth Intern. Coral Reef Symp. 1: 601-606.

Bruckner, A.W., and R.J. Bruckner. 1997b. Outbreak of coral disease in Puerto Rico. Coral Reefs. 16:260.

Bruckner, A.W., and R.J. Bruckner. 1998b. Rapid-wasting disease: pathogen or predator? Science. 279:2023-2025.

Bruckner, A.W., R.J. Bruckner, and E.H. Williams. 1997b. Spread of a black-band disease epizootic through the coral reef system in St Ann's Bay, Jamaica. Bull Mar. Sci. 61:919-928.

Bruckner, A.W. 2002. Priorities for effective management of coral diseases NOAA Technical Memorandum NMFS-OPR-22. 54 pp.

Bruckner, A.W., and Bruckner, R.J. 2000. Coral predation by *Sparisoma viride* and lack of relationship with coral disease Proc. 9th Intern Coral Reef Symp.

2: 1245-1249.
Bruckner, A.W. 2003. Field guide to western Atlantic coral diseases and other causes of mortality. CD-ROM. UNEP-WCMC.
Bruckner, A.W., and R.J. Bruckner . 2003. Condition of coral reefs off less developed coastlines of Curaçao (stony corals and algae). Atoll Research Bull. 496:370-393.
Bruckner, A.W., and Bruckner, R.J. 2006. Consequences of yellow band disease (YBD) on *Montastraea annularis* (species complex) populations on remote reefs off Mona Island, Puerto *Rico* . Diseases Aquat. Org. 69: 67-73.
Bruggemann J., M. van Oppen, and A. Breeman. 1994. Foraging by the stoplight parrotfish, *Sparisoma viride*. I. Intake and assimilation of food, protein and energy. Mar. Ecol. Prog. Ser. 106:41–55.
Bruno J.F., L.E. Petes, C.D. Harvell, et al., 2003. Nutrient enrichment can increase the severity of coral diseases. Ecol. Lett. 6 :1056-1061.
Bythell J., and C. Sheppard. 1993. Mass mortality of Caribbean shallow corals. Mar. Poll. Bull. 26: 296-297.
Bythell, J.C., E.H. Gladfelter and M. Bythell. 1993. Chronic and catastrophic natural mortality of three common Caribbean reef corals. Coral Reefs. 12:143-152.
Bythell, J.C., M.R. Barer, R.P. Cooney, J.R. Guest, A.G. O'Donnell, O. Pantos, and M.D.A. Le Tissier. 2002. Histopathological methods for the investigation of microbial communities associated with disease lesions in reef corals. Letters in Applied Microbiol. 34:359–364.
Bythell, J., O Pantos and L. Richardson. 2004. White plague, white band and other "white" diseases, *In* E. Rosenberg and Y. Loya [eds.], Coral Health and Disease. Springer-Verlag 351-366.
Carlton, R.G., and L.L. Richardson. 1995. Oxygen and sulfide dynamics in a horizontally migrating cyanobacterial mat: Black band disease of corals. FEMS Microbiol. Ecol. 18:155-162.
Cervino, J.M., T.J. Goreau, M. Goreau, G. Smith, and 11 others. 1997. Rapid spread of diseases in Caribbean coral reefs. Proc Assoc. Isl. Mar. Lab. Caribb. 28:43.
Cervino, J., T.J.Goreau, I. Nagelkerken, G.W. Smith, and R. Hayes. 2001. Yellow band and dark spot syndromes in Caribbean corals: Distribution, rate of spread, cytology, and effects on abundance and division rate of zooxanthellae. Hydrobiologica. 460: 53-63.
Cervino, J.M., R. Hayes, T.J. Goreau, and G.W. Smith. 2004. Zooanthellae regulation in Yellow Blotch/Band and other coral diseases contrasted with temperature related bleaching: In situ destruction vs explusion. Symbiosis. 37:63-85.
Cervino, J.M., R. Hayes, S.W. Polson, T.J. Goreau, R.J. Martinez, and G.W. Smith. 2004. Relationship of *Vibrio* species infection and elevated temperatures to yellow blotch/band disease in Caribbean corals. Appl. Environ. Microbiol. 70(11):6855-6864.
Cooney, R.P., O. Pantos, M.D.A.L. Tissier, M.R. Barer, A.G. Ódonnell, and J.C. Bythell. 2002. Characterization of the bacterial consortium associated with black band disease in coral using molecular microbiological techniques. Environ. Microbiol. 4:401-413.
Croquer, A., E. Weil, and D. Bone. 2004. Temporal variation in tissue mortality of *Montastrea faveolata* with yellow blotch syndrome at Morrocy National Park, Venezuela. Abstract, 10th ICRS, Okinawa.

Croquer, A., C. Bastidas and L. Lipscomb. 2006. Folliculinid ciliates : a new threat to Caribbean corals ? Diseases Aquat. Org. 69:75-78.
Davis M., Gladfelter E., Lund H., Anderson M. 1986. Geographic range and research plan for monitoring white band disease. Biosphere Reserve Research Report No. 6. National Park Service. 28 pp.
Denner, E.B.M., G.W. Smith, J.H. Busse, P. Schumann, T.Narzt, S. Polson, W. Lubitz, and L.L. Richardson. 2003. *Aurantimonas coralicida* gen. nov., sp nov., the causative agent of white plague type II on Caribbean scleractinian corals. Int. J. Syst. Evol. Micr. 53: 1115-1122.
Downs, C.A. 2005. Cellular diagnostics and its application to aquatic and marine toxicology. pp.301-313 *In:* G. Ostrander [ed.], Techniques in Aquatic Toxicology, Vol 2. CRC Press, Inc., Boca Raton.
Ducklow, H.W., and R. Mitchell. 1979. Observations on naturally and artificially diseased tropical corals:scanning electron microscope study. Microb. Ecol. 5:215-223.
Dustan, P., and J.C. Halas. 1987. Changes in the reef-coral community of Carysfort Reef, Key Largo, Florida: 1974 to 1982. Coral Reefs. 6:91-106.
Dustan, P. 1977. Vitality of reef coral populations off Key Largo, Florida: recruitment and mortality. Environ. Geol. 2:51-58.
Dustan, P 1987. Preliminary observations on the vitality of reef corals in San Salvador, Bahamas. P. 57-65 *In* H.A. Curran [ed.], Proc. Third Symp. Geol. Of the Bahamas.
Dustan, P 1993. Developing methods for assessing coral reef vitality: a tale of two scales. Global Aspects of Coral Reefs. pp.M8-M14.
Edmunds, P.J. 1991. Extent and effect of black band disease on a Caribbean reef. Coral Reefs. 10:161-165.
Edmunds, P.J. 2000. Recruitment of scleractinians onto the skeletons of corals killed by black band disease. Coral Reefs. 19: 69-74.
Epstein, P.R. et al. 1998. Marine ecosystems: Emerging diseases as indicators of change: Health of the oceans from Labrador to Venezuela. Year of the Oceans Special Report. Cent. Health Global Environ, Harvard Medical School. 85 pp.
Feingold, J.S. 1988. Ecological studies of a cyanobacterial infection on the Caribbean sea plume *Pseudopterogorgia acerosa* (Coelenterata: Octocorallia*)*. Proc. Sixth Intern. Coral Reef Symp. 3:157-162.
Foley, J., S. Sokolow, E. Girvetz, and P. Foley. 2004. Spatial epizootiology of yellow band syndrome in *Montastrea* spp. coral in the Eastern Yucatan Peninsula, Mexico. Poster at the 10th International Coral Reef Symposium, Okinawa
Foley, J.E., et al. (2005). Spatial epidemiology of Caribbean yellow band syndrome in *Montastrea spp*.coral in the eastern Yucatan, Mexico. Hydrobiologia. 548:33-40.
Frias-Lopez, J., A. L. Zerkle, G.T. Bonhey, and B.W. Fouke. 2002. Partitioning of bacterial communities between seawater and healthy, black band diseased, and dead coral surfaces. Appl. and Environ. Microbiol. 68:2214-2228.
Frias-Lopez, J., G. T. Bonheyo, Q. Jin, and B.W. Fouke. 2003. Cyanobacteria associated with coral black band disease in Caribbean and Indo-Pacific reefs. Appl. and Environ. Microbiol. 69:2409-2413.
Garcés-Baquero, A.M. 2000. Desarrollo de la Enfermedad de Lunares Oscuros en los Corales Pétreos *Montastraea annularis* y *Siderastrea siderea* en la Bahía de Gayraca,

Caribe Colombiano. Ph.D. Thesis. Fundación Universidad de Bogotá Jorge Tadeo Lozano.
Gardner, T.A., I.M. Cote, J.A. Gill, A. Grant, and A.R.Watkinson. 2003. Long-term region-wide declines in Caribbean corals. Science. 301:958-960.
Garrett, P., and H. Ducklow. 1975. Coral diseases in Bermuda. Nature. 523:349-350.
Garrison et al., 2003. African and Asian dust: from desert soils to coral reefs. BioScience. 53: 469-480.
Garzon-Ferreira, J., and Kielman. 1993. Extensive mortality of corals in the Colombian Caribbean during the last two decades. Proc. Colloquim Global Aspects of Coral Reefs. P. 247-253.
Garzón-Ferreira, J., D.L. Gil-Agudelo, L.M. Barrios, and S. Zea. 2001. Stony coral diseases observed in southwestern Caribbean reefs. Hydrobiologia. 460:65-69.
Geiser, D.M., J. Taylor, K. Ritchie, and G. Smith. 1998. Cause of sea fan death in the West Indies. Nature. 394:137-138.
Gil-Agudelo, D.L., and J. Garzon-Ferreira. 1999. Seasonal variation of the dark spots disease in the Colombian Caribbean. Int. Conf. on Scientific Aspects of Coral Reef Assessment, Monitoring and Restoration. Abstract, pp.91.
Gil-Agudelo, D.L., G.W. Smith, J. Garzon-Ferreira, E. Weil, and D. Petersen. 2004. Dark spot disease and yellow band disease, two poorly known coral disease with high incidence in Caribbean reefs. Pp. 337-349 *In*, E. Rosenberg and Y. Loya [eds.], Coral Health and Disease.
Gladfelter, W.B., E.H. Gladfelter, R.K. Monahan, J.C. Ogden, and R.D. Dill.1977. Environmental studies of Buck Island Reef National Monument, St. Croix, USVI. National Park Service Rept. 140 pp.
Gladfelter, W.B. 1982. White-band disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. Bull. Mar. Sci. 32:639-643.
Gladfelter, W.B. 1990. Population structure of *Acropora palmata* on the windward fore reef, Buck Island National Monument, St Croix, U.S. Virgin Islands. U.S. Department of the Interior, National Park Service, U.S. Virgin Islands. 172pp.
Gochfeld, D.J, J.B. Olson and M. Slattery. 2006. Colony versus population variation in susceptibility and resistance to dark spot syndrome in the Caribbean coral *Siderastrea siderea.* Diseases Aquat. Org. 69:53-65.
Goenaga, C. and Boulon. 1992. The state of Puerto Rican corals: an aid to managers. Unpublished report submitted to the Caribbean Fisheries Management Council. 73 pp.
Goreau, T.F. 1959. The ecology of Jamaican coral reefs. I. Species composition and zonation. Ecology. 40(1):67-90.
Goreau, T. J., J. Cervino, M. Goreau, R. L. Hayes, et al. 1998. Rapid spread of disease in Caribbean coral reefs. Revista de Biología Tropical. 46:157-171.
Green, E., and A. W. Bruckner. 2000. The significance of coral disease epizootiology for coral reef conservation. Biological Conservation. 96:347-361.
Guzman, H., and Cortes. 1984a. Mass death of *Gorgonia flabellum* L. (Octocorallia: Gorgonidae) on the Caribbean coast of Costa Rica. Revista Biological Tropical. 32:305-308.
Harvell, C. D., K. Kim, J. M. Burkholder, R. R. Colwell, P. R. Epstein, D. J. Grimes, E. E. Hofmann, E. K. Lipp, A. D. M. E. Osterhaus, R. M. Overstreet, J. W. Porter, G. W.

Smith, and G. R. Vasta. 1999. Emerging Marine Diseases - Climate links and anthropogenic factors. Science. 285:1505-1510.
Harvell, D., Kim, K., Quirolo, C., Weir, J., and Smith, G. 2001. Coral bleaching and disease: contributors to 1998 mass mortality in *Briareum asbestinum* (Octocorallia, Gorgonacea). Hydrobiologica. 460: 97-104.
Hayes, R.L., and N.I. Goreau. 1998. The significance of emerging diseases in the tropical coral reef ecosystem. Revista de Biologica Tropical. 46:173-185.
Hughes, TP. 1994. Catastrophes, phase shifts, and large scale degradation of a Caribbean coral reef. Science. 265: 1547-1551
Jordan-Dahgren, E., and R.E. Rodriguez-Martinez. 2004. Coral diseases in the Gulf of Mexico reefs. pp. *In*: E. Rosenberg and Y. Loya [eds.], Coral Health and Disease. Springer-Verlag, Berlin.
Kaczmarsky, L.T., M. Draud, and E.H. Williams. 2005. Is there a relationship between proximity to sewage effluent and the prevalence of coral disease.Carib. Jour. Sci. 41:124-137.
Kim, K., and C.D. Harvell. 2002. Aspergillosis of sea fan corals: disease dynamics in the Florida Keys. pp. 813–824 *In*: Porter, J.W. & Porter, K.G. [eds.], The Everglades, Florida Bay, and Coral Reefs of the Florida Keys: An Ecosystem Sourcebook. CRC Press, Boca Raton.
Kim, K., and C.D. Harvell. 2004. The rise and fall of a six-year coral-fungal epizootic. American Naturalist. 164:S52-S63.
Knowlton, N. et al., 1981. Evidence for delayed mortality in hurricane-damaged Jamaican staghorn corals. Nature. 294:251-252.
Kozlowski, J. 1996. Investigations of the occurrence and distribution of bacterial aggregates in *Acropora cervicornis* from reefs in the Bahamas and the Florida Keys. M.S. Thesis, University of Maryland Eastern Shore, Princess Anne, MD.
Kuntz, N.M., D.I. Kline, S.A. Sandin, and F. Rohwer. 2005. Pathologies and mortality rates caused by carbon and nutrient stressors in three Caribbean coral species. Mar. Ecol. Prog. Ser. 294:173-180.
Kuta, K.G., and L.L. Richardson. 1996. Abundance and distribution of black band disease on coral reefs in the northern Florida Keys. Coral Reefs. 15:219-223.
Kuta, K.G. and L.L. Richardson. 1997. Black band disease and the fate of diseased coral colonies in the Florida Keys. Proc. Eighth Intern. Coral Reef Symp. 1:575-578.
Kuta, K.G. and L.L. Richardson. 2002. Ecological aspects of black band disease of corals: relationships between disease incidence and environmental factors. Coral Reefs. 21: 393-398.
Laydoo, 1983b. Recent mass mortality of gorgonians in Trinidad. Unpublished report presented at the 17th AMLC meeting. 12pp.
Laydoo, 1984. Inference of a white-band epidemic in the elkhorn coral, *A. palmata*, populations in Tobago, West Indies. Proc. Assoc. Island Mar. Labs. Carib. 18:12.
Lipp et al., 2002. Preliminary evidence for human fecal contamination in corals of the Florida Keys, USA. Mar. Poll. Bull. 44: 666-970
Miller, J., C. Rogers, and R. Waara. 2003. Monitoring the coral disease, plague type II, on coral reefs in St. John, U.S. Virgin Islands. Revista de Biologia Tropical. 51: 47-55.

Morse, D.E., A.N.C. Morse, and H. Duncan. 1977. Algal "tumours" in the Caribbean sea-fan, *Gorgonia ventalina*. Proc. Third Intern. Coral Reef Symp. 1:623-629.

Morse, D.E., A. Morse, H. Duncan, and R.K. Trench. 1981. Algal tumours in the Caribbean Octocorallian*, Gorgonia ventalina*: II. Biochemical characterization of the algae, and first epidemiological observations. Bull. Mar. Sci. 31:399-409.

Muller, E.M., C.S. Rogers, and R. Van Woesik. 2004. Lesion formation on *Acropora palmata* and the coral's ability to heal. Abstract, 10th ICRS, Okinawa.

Nagelkerken, I., A.W. Buchan, G. Smith, K. Bonair, P.Bush, J. Garzon-Ferreira, L. Botero, P. Gayle, C.D. Harvell, C. Heberer, K. Kim, C. Petrovic, L. Pots, and P. Yoshioka. 1997a. Widespread disease in Caribbean sea fans: I. Spreading and general characteristics. Proc Eighth Intern. Coral Reef Symp. 1:679-682.

Nagelkerken, I., A.W. Buchan, G. Smith, K. Bonair, P.Bush, J. Garzon-Ferreira, L. Botero, P. Gayle, C.D. Harvell, C. Heberer, K. Kim, C. Petrovic, L. Pots, and P. Yoshioka. 1997b. Widespread disease in Caribbean sea fans: II. Patterns of infection and tissue loss. Mar. Ecol. Prog. Ser. 160:255-263

Nugues, M.M. 2002. Impact of a coral disease outbreak on coral communities in St. Lucia: What and how much has been lost? Mar. Ecol. Prog. Ser. 229:61-71.

Pandolfi et al., 2003. Global trajectories of the long-term decline of coral reef ecosystems. Science. 301: 955-958.

Pantos, O., R.P. Cooney, M.D.A. Le Tissier, M.R. Barer, A.G. O'Donnell, and J.C. Bythell. 2003. The bacterial ecology of a plague-like disease affecting the Caribbean coral *Montastrea annularis.* Environ. Microbiol. 5(5):370-382.

Pantos, O. , and J.C. Bythell. 2006. Bacterial community structure associated with white band disease in the elkhorn coral *Acropora palmata* determined using culture-independent 16S rRNA techniques. Dis. Aquat. Org. 69:79–88.

Patterson, K.L., J.W. Porter, K.B. Ritchie, S.W. Polson, E. Mueller, E.C. Peters, D.L. Santavy, and G.W. Smith. 2002. The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, Acropora palmata. Proc. Natl. Acad. Sci. (USA). 99:8725-8730.

Peters, E.C., P.P. Yevich, and J.J. Oprandy. 1983. Possible causal agent of white band disease. Journal Invert. Path. 41:394-396.

Peters E.C., J.C. Halas, and H.B. McCarty. 1986. Calicoblastic neoplasms in *Acropora palmata,* with a review of reports on anomalies of growth and form in corals. Journal Nat. Cancer Inst. 76:895-912.

Peters, E.C. 1984. A survey of cellular reactions to environmental stress and disease in Caribbean scleractinian corals. Helgol Wiss Meeresunters. 37:113-137.

Porter, J.W., P. Dustan, W.C. Jaap, K.L. Patterson, V. Kosmynin, O.W. Meier, M.E. Patterson, and M. Parsons. 2001. Patterns of spread of coral disease in the Florida Keys. Hydrobiologia. 460:1–24.

Ramos-Flores, T. 1983. Lower marine fungus associated with black line disease in star corals *(Montastrea annularis,* E. & S.). Biol. Bull. 165:429-435.

Reeves, L. 1994. Newly discovered: Yellow band disease strikes Keys Reefs. Underwater USA. 11 (8):16.

Richardson, L.L., and R.G. Carlton, 1993. Behavioural and chemical aspects of black-band disease of corals: an in situ field and laboratory study. Proc. Amer. Academy Underwater Sci. 13th Ann. Scientific Diving Symp., pp.107-116

Richardson, L.L., and K.G. Kuta, 2003. Ecological physiology of the black band disease cyanobacterium *Phormidium corallyticum.* FEMS Microbiol. Ecol. 43: 287-298.
Richardson, L.L., K.G. Kuta, S. Schnell, and R.G. Carlton. 1997. Ecology of the black band disease microbial consortium. Proc. Eighth Intern. Coral Reef Symp. 1:597-600.
Richardson, L.L., G.W. Smith, K.B. Ritchie, and R.G. Carlton. 2001. Integrating microbiological microsensor molecular and physiological techniques in the study of coral disease pathogenesis. Hydrobiologia. 460:71–89.
Richardson, L.L. 1992. Red band disease: a new cyanobacterial infestation of corals. Proc. Tenth Ann. Amer. Acad. Underwater Sci. 153-160.
Richardson, L.L., W. M. Goldberg, K. G. Kuta, R. B. Aronson, G. W. Smith, K. B. Ritchie, J. C. Halas, J. S. Feingold, and S. L. Miller. 1998a. Florida's mystery coral-killer identified. Nature. 392:557-558
Richardson, L.L. 1996. Horizontal and vertical migration patterns of *Phormidium corallyticum* and *Beggiatoa* spp. associated with black-band disease of corals. Microb. Ecol. 32:323-335.
Richardson, L.L.1997. Occurrence of the black band disease cyanobacterium on healthy corals of the Florida Keys. Bull. Mar. Sci. 61:485-490.
Richardson, L. 2000. Coral Epidemiology. In: Opportunities for environmental application of marine biotechnology. Proc. Oct 5-6 Workshop. Board on Biology, National Research Council, National Academy Press.
Richardson, L.L., and R.A. Aronson. 2002. Infectious diseases of reef corals. Proc. Ninth Intern. Coral Reef Symp. 2: 1225-1230.
Ritchie, K.B., and G.W. Smith. 1995. Preferential carbon utilization by surface bacterial communiites from water mass, normal, and white-band diseased *Acropora cervicornis.* Mol. Mar. Bio. and Biotech. 4:345-352.
Ritchie, K.B., and G.W. Smith. 1998. Type II white-band disease. Revista Biologica Tropical. 46:199-203.
Rodríguez-Martínez, R. E., A.T. Banaszak, and E. Jordán-Dahlgren. 2001. Necrotic patches affect *Acropora palmata* (Scleractinia: Acroporidae) in the Mexican Caribbean. Diseases Aquat. Org. 47:229-234.
Rogers, C. 1985. Degradation of Caribbean and Western Atlantic coral reefs and decline of associated fisheries. Proc. Fifth Intern. Coral Reef Symp. 6:491-496.
Rohwer, F., V. Seguritan, F. Azam, and N.Knowlton. 2002. Diversity and distribution of coral-associated bacteria. Mar Ecol Prog Ser. 243: 1–10.
Rutzler, K., and D.L. Santavy. 1983. The black band disease of Atlantic reef corals. I. Description of the cyanophyte pathogen. PSZNI: Mar. Ecol. 4:301-319.
Rutzler, K., D.L. Santavy, and A. Antonius. 1983. The black band disease of Atlantic reef corals. III. Distribution, Ecology, and Development. PSZNI: Mar. Ecol. 4:329-258.
Santavy, D.L., and E. Peters. 1997. Microbial pests: coral disease in the Western Atlantic. Proc. Eighth Intern. Coral Reef Symp. 1:607-612.
Santavy, D.L. et al., 1995a. Characterisation of the bacterium suspected in the incidence of white band disease. 95th General Meeting of the ASM. Abstract, p.332.
Santavy, D.L., Peters, E.C., Quirolo, C., Porter, J.W., and Bianchi, C.N. 1999. Yellow-blotch disease outbreak on reefs of the San Blas Islands, Panama. Coral Reefs. 18:97.

Smith, G.W., C.D. Harvell, and K. Kim. 1998. Response of sea fans to infection with *Aspergillus* sp. (fungi). Revista Biologica Tropical. 46:205-208.

Smith, G.W., and E. Weil. 2004. Aspergillosis in gorgonians. Pp 279-288 *In:* Rosenberg E, Loya Y [eds.], Coral Health and Diseases. Springer-Verlag, New York.

Sutherland, K., J. Porter and C. Torres, 2004. Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar. Ecol. Prog. Ser. 266: 273-302.

Sutherland, K.P., and K.B. Ritchie. 2004. White pox disease of the Caribbean elkhorn coral *Acropora palmata.* pp *In:* Rosenberg E, Loya Y [eds.], Coral Health and Diseases. Springer-Verlag, New York.

TeStrake, D., W.C. Jaap, E. Truby, and R. Reese. 1988. Fungal filaments in Millepora complenata Lamarck, 1815 (Cnidaria:Hydrozoa) after mass expulsion of zooxanthellae. Biol. Sci. 5:184-188.

Toldeo-Hernandez, C., P. Bayman, and A. Sabat. 2004. Fungal infection in the Caribbean sea fans (Gorgonia sp.). Proc 10th Intern. Coral Reef Symp. Abstract. P.167.

Upton, S.J., and E.C. Peters. 1986. A new and unusual species of coccidium Apicomplexa: Agamococcidiorida from Caribbean scleractinian corals. J. Invert. Path. 47:184-93.

Viehman, T.S., and L.L. Richardson. 2002. Motility patterns of *Beggiatoa* and *Phormidium corallyticum* in black-band disease. Proc 9th Intern. Coral Reef Symp. 2:1251-1255.

Voss, J.D. and L.L. Richardson. 2006. Coral diseases near Lee Stocking Island, Bahamas: patterns and potential drivers. Diseases Aquat. Org. 69:33–40.

Weil, E. 2004. Coral reef diseases in the wider Caribbean. pp. 35-68 *In:* Rosenberg E, Loya Y [eds.], Coral Health and Diseases. Springer-Verlag, New York.

Weil, E., I. Urreizieta, and J. Garzon-Ferreira. 2000. Geographic variability in the incidence of coral and octocoral diseases in the wider Caribbean. Proc 9th Intern. Coral Reef Symp. 2: 23-27.

Weil, E., G.W. Smith, and D.L. Gil-Agudelo. 2006. Status and progress in coral reef disease research. Diseases Aquat. Org. 69:1-7.

Williams, E.H., Jr., P.J. Bartels, and L. Bunkley-Williams. 1999. Predicted disappearance of coral-reef ramparts: A direct result of major ecological disturbances. Global Change Biol. 5:839-845.

Williams, E.H., and L. Bunkley-Williams. 2000. Marine Major Ecological Disturbances of the Caribbean. Infect. Dis. Rev. 2:110-127.

Williams, D.E., and M.W. Miller. 2005. Coral disease outbreak: pattern, prevalence and transmission in *Acropora cervicornis*. Mar. Ecol. Prog. Ser. 301: 119-128.

Woodley, C.M., A.W. Bruckner, S.B. Galloway, S.M. McLaughlin, C.A. Downs, J.E. Fauth, E.B. Shotts, and K.L. Lidie. 2003. Coral Disease and Health: A National Research Plan. National Oceanic and Atmospheric Administration, Silver Spring, MD. 72 pp.

Zimmerman, R. 1994. What is ridge mortality? Hammerhead Chronicles. Winter/Spring Newsletter of the Flower Gardens Fund of the Gulf of Mexico Foundation. pp3.