Priorities for Effective Management of Coral Diseases

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SUMMARY

Diseases of scleractinian corals and associated species have proliferated in recent years, and they are now recognized as important phenomena capable of altering the structure and composition of coral reefs. Since the early 1990s there has been a concerted effort to characterize coral diseases, including the application of novel molecular tools to confirm identities of pathogens and understand mechanisms of host response and resistence. Most of the causative agents of emerging diseases, factors contributing to their occurrence and spread, and consequences on coral populations remain incompletely understood, however. A long-term, multi-disciplinary research and monitoring program for coral diseases is necessary to assist resource managers in identifying and responding to emerging coral diseases. These efforts should involve management-driven strategies that include 1) an early warning system to predict and identify disease outbreaks; 2) documentation of spatial distribution and temporal variations of coral diseases and other syndromes at local to global scales; 3) elucidation of relationships of environmental stressors, localized anthropogenic impacts, and widespread phenomena such as global warming and El Niño on coral health, disease, degradation and recovery; 4) development of standardized terminology for diseases and other syndromes through a characterization of the visual appearance, pathology and etiology, and the development of molecular probes and other tools to identify and verify diseases in the field; 5) identification of factors that facilitate the introduction, spread and transmission of pathogens; 6) research on the effects of disease on coral species and populations, associated species, and ecosystem structure and function; and 7) implementation of measures to mitigate disease impacts, including strategies that reduce anthropogenic stressors responsible for the proliferation or spread of diseases and the development of novel techniques to treat affected corals and improve habitat quality. Progress in coral disease research has been hindered by a lack of coordinated research, practical difficulties in correctly identifying diseases and characterizing their etiology and epizootiology, limited availability of funding, and limited awareness and recognition by policy makers and the public of the importance of coral diseases and their potential global implications. Given existing limitations in funding, disease research should be focused towards those efforts that have the greatest management benefits with applications on local to regional scales.

INTRODUCTION

Coral reef ecosystems are being degraded at an accelerated rate. The underlying causes of reef decline are diverse, and include pollution, sedimentation, fishing impacts, habitat destruction, invasive species, bleaching, disease, global climate change, and other factors. In recent years, disease outbreaks have caused widespread mortalities to scleractinian corals, gorgonians, sea urchins, reef fish, sponges, algae and associated coral reef organisms (Peters, 1997; Harvell et al., 2000; Williams and Bunkley-Williams, 2000). Unfortunately, characterization of the cause, prevalence, and consequences of most of these events is limited or non-existent. For instance, hundreds of studies have been published on coral diseases (Appendix III) since the first reported coral disease (Antonius, 1973), and yet the causative agent has been confirmed for only three diseases (Richardson, 1998). In addition, very limited quantitative data are available on the spatial and temporal distribution of coral diseases, synergistic and cumulative effects of environmental and anthropogenic stressors, and long-term consequences on the composition, structure and function of reef communities (Green and Bruckner, 2000).

Although quantitative field observations on coral diseases are restricted to few locations and relatively small numbers of corals, there is a growing consensus that coral diseases have proliferated in the 1990s (Santavy and Peters, 1997; Goreau et al., 1998; Hayes and Goreau, 1998; Richardson, 1998; Harvell et al., 2000; Williams and Bunkley Williams, 2000). In an examination of all information published on coral diseases between 1973-1999, diseases were reported in over 102 different coral species from 54 different nations, with a disproportionate number of records (66%) from the Wider Caribbean (Green and Bruckner, 2000). Currently, the majority of these reports are for black-band disease (BBD), white-band disease (WBD) and white plague. These diseases were first documented from the wider Caribbean in the 1970s, but they typically occurred at a low prevalence or in few locations (Antonius, 1977; Garrett and Ducklow, 1975; Dustan, 1977). Outbreaks of BBD and WBD were reported from numerous locations in the Caribbean in the 1980s and 1990s, and both are now circum-tropical in distribution (Antonius, 1987; Green and Bruckner, 2000). White plague was first identified as a significant infectious diseases in the Florida Keys in 1975, and it was still common in 1984 on 19 different reefs in the Key Largo region (Dustan, 1977; 1993). A more virulent form of white plague (plague type II) emerged in 1995 on reefs in the northern Florida Keys (Richardson et al., 1998). Additional outbreaks of white plague (plague type II and type III) were also observed in Florida and other locations around the Caribbean between 1999-2001 (Richardson and Aronson, in press). During the 1980s and 1990s, twenty five other diseases of scleractinian corals and gorgonians were reported from the western Atlantic (Table 1) and 14 from the IndoPacific (Table 2). Western Atlantic reefs have

Table 1. Diseases, syndromes and anomalies affecting scleractinian corals and gorgonians on coral reefs in the tropical western Atlantic. Data are compiled from publications and web sites on coral disease identified through 2001. Other names are used in the literature to describe similar disease signs but these have been combined under the following diseases: (1) BBD is also called black line disease; (2) WBD and white plague are also called white line disease; (3) WBD also used to describe massive corals in the Caribbean with signs similar to white plague; (4) YBD is also called yellow-band disease and yellow pox disease; (5) rapid wasting disease was renamed rapid wasting syndrome, and recently reported to be synonymous with parrotfish white spot biting; and (6) ridge mortality has been called damselfish-ridge denuding syndrome.

Condition	First observed	Known Range	Affected species	Source
Black-band disease (BBD)[1]	Belize; Florida; Puerto Rico	W. Atlantic 19 countries	faviids, agaricids gorgonians 22 species total	Antonius, 1972
White-band disease (WBD)[2]	St. Croix	USVI, BVI, Florida, Puerto Rico, Belize, Bonaire, Curacao Aruba, Jamaica, Bahamas, Tobago, Guadalupe, Nicaragua St. Lucia, Cuba	*A. palmata* *A. cervicornis* *A. prolifera*	Gladfelter, 1977
WBD type II	Bahamas	Bahamas	*A. cervicornis*	Richie and Smith, 1995
Shut-down reaction	Florida, Belize	Florida, Belize	massive corals, acroporids	Antonius, 1977
White plague [3]	Florida	Bahamas, Puerto Rico, Florida, USVI, BVI, Curacao, Bonaire, Jamaica, Bermuda, Tobago, Guadalupe, Nicaragua, St. Lucia	faviids, agaricids, mussids, poritids; 12 species	Dustan, 1977
Plague type II	Florida	PR, FL, USVI, Bonaire, Curacao, Mexico,	32 species	Zorpette, 1995
Plague type III	Florida	Caribbean	large massive corals (*M. faveolata, C. natans*)	Richardson and Aronson, 2001
Yellow-blotch disease (YBD)[4]	Florida	Florida, Mexico, Puerto Rico, Curacao, Bonaire, Panama, Jamaica, Honduras, Bahamas	*Montastraea annularis* complex; *C. natans*	Reeves, 1994
White pox	Florida	Bahamas	*A. palmata*	Porter, 1996
Patchy necrosis	Puerto Rico	Caribbean	*A. palmata*	Bruckner and Bruckner, 1997
Rapid wasting disease [5]	Bonaire	Caribbean	*Montastraea* spp., *C. natans*	Cervino et al., 1997
White spot syndrome	Caribbean		massive corals	Global Coral Reef Alliance web pages
Ridge mortality [6]	Flower Gardens	Caribbean	*C. natans,* *D. strigosa*	Abbott, 1979
Dark-spots disease (DSD)	Columbia	Caribbean	*S. siderea,* *S. radians*	Garzón-Ferreira, 1997

Table.1 continued. Diseases, syndromes and anomalies affecting scleractinian corals and gorgonians in the western Atlantic continued. ** Mortalities of sea fans were observed in the 1980s, but they may be caused by disease or other factors.

DSD- II	Caribbean		*S. intersepta; M. annularis; M. faveolata; M. cavernosa; C. natans; C. amaranthus; S. siderea*	Weil, 2001
Red-band disease (RBD) type I	Belize Puerto Rico		*Gorgonia*, *Agaricia*, *Colpophyllia, Mycetophyllia* and *Stephanocoenia*	Rützler et al., 1983 Santavy and Peters, 1997
RBD type II	Bahamas		*D. stigosa, M. annularis, M. cavernosa, P. astreoides* and *S. radians*	Richardson, 1992
Hyperplasia	Puerto Rico, USVI, Jamaica, Netherlands Antilles, Trinidad, Belize		massive corals	Loya et al., 1984
Neoplasia	Florida	Netherlands Antilles, Trinidad, Florida	*A. palmata*	Peters et al., 1986
Coccidium infection	USVI, Puerto Rico, Jamaica		*A. agaricites, D. cylindicus, D. strigosa, M. meandrites, M. cavernosa, P. astreoides, P. porites* 8 species	Upton and Peters, 1986
Stress-related necrosis				Peters, 1984
Blistering necrosis	Puerto Rico			1984
Ring disease	Bermuda	Honduras, Florida	*D. labyrinthiformis*	Weil, 2001
Finger coral denuding syndrome	?		*Porites*	Williams, 2000
Star coral polyp necrosis	?		*M. cavernosa*	Williams, 2001
Algal tumors	Bonaire, Trinidad	Caribbean	*Gorgonia* spp. *Pseudoplexaura; Plexaura*	Morse et al., 1977
Aspergillosis	Trinidad, Costa Rica, Columbia, Panama**	Carribean (at least 14 countries)	*Gorgonia* spp.	Nagelkerken et al., 1997
Fire coral fungal disease	Florida		*Millepora* spp.	TeStrake et al., 1988

Table 2. Diseases, syndromes, and anomalies reported to affect stony corals in the tropical Pacific ocean, Indian Ocean and Red Sea.

Condition	Location	Species affected	Source
Black-band disease	Saudi Arabia Tonga Fiji Australia United Arab Emirates Philippines Papua New Guinea Egypt India	*Favia, Favites, Acropora, Montipora, Pachyseris, Platygyra, Goniastrea, Leptoria, Turbinaria, Montastraea, Hydnophora* 18 species	Antonius, 1987 Chesher, 1984 Littler and Littler, 1995 Miller, 1996 Korrubel and Riegl, 1998 Fenner, 1998 Cervino, 1998
White-band disease	Saudi Arabia Fahl Island United Arab Emirates Philippines Egypt Oman Australia Guam India Papua New Guinea Mauritius	*Acropora, Goniastrea Platygyra, Hydnophora Leptoria, Favia Pocillopora, Stylophora Lobophyllia, Porites, Leptoseris, Echinopora, Mycedium, Podabacia, Monitopora, Symphyllia*, 33 species	Antonius, 1987 Coles, 1997 Korrubel and Riegl, 1998
Shutdown reaction	Saudi Arabia		Antonius, 1981
White blotch disease	Australia	*Acropora*	1992
Brown-band disease	Australia	*Acropora*	1994
Black aggressive band	Mauritius	*Acropora*	Antonius, 1995
Black overgrowing cyanobacteria	Indian Ocean	*Acropora, Pocillopora, Porites, Favia*	Antonius, 1995
Skeleton eroding band	Egypt	massive corals	Antonius, 1999
Pink Line disease	Papua New Guinea, Sri Lanka	*Porites*	Goreau/Cervino, coral list server
Yellow-band disease	Arabian Gulf	*Acropora, Porites, Cyphastrea, Turbinaria*	Korrubel and Riegl, 1998
Fungal infection	Adaman Islands, Indian Ocean	*Porites,*	Raghukumar et al
Porites ulcerative white spot disease	Philippines	*Porites*	Raymundo, 2000
Tumors	GBR	*Platygyra*	Loya et al. 1984
Pink-blue disease	Israel	*Acropora, Porites*	Red Sea Marine Park, 2001
Rapid Tissue Necrosis	Aquarium Corals	*Acropora, Pocillopora, Seriatopora*; 11 others	Borneman, 2001

become a "hot spot" for diseases, due to an increased virulence, spread, and host range of coral diseases, a recent emergence of new diseases, and the relatively small, enclosed and interconnected nature of the Caribbean basin.

It is now recognized that coral diseases have played an important role in the loss of coral cover over the last two decades. For instance, there is good evidence that mortality from WBD has modified the structure and composition of reefs in the wider Caribbean by removing two of the most common and locally abundant species. However, environmental perturbations and anthropogenic stressors that may have triggered disease outbreaks or contributed to their spread and severity are not fully understood. In addition, our ability to predict the long-term impact of coral diseases and other emerging syndromes is hindered by an incomplete understanding of 1) ecological processes and interactions at the species, population and community level; 2) connectivity among reefs and the importance of regional and local water movement patterns as vectors for dispersal of (coral) propagules and water-borne pathogens; and 3) factors that influence the resilience of coral reef ecosystems and their ability to persist under increasing environmental and anthropogenic impacts.

While much research is needed to achieve a better understanding ecological and oceanographic processes affecting coral reef ecosystems and responses of these ecosystems to chronic impacts, there is also an urgency to understand the causes of diseases and their consequences on coral populations. Regional and global coral reef monitoring programs [ReefCheck, Caribbean Coastal Marine Productivity (CARICOMP), Atlantic and Gulf Rapid Reef Assessment (AGRRA)] are providing valuable information on reef condition and threats. Some of these programs also include methodologies for disease surveys (Appendix I). However, there remains a need for targeted studies on coral disease etiology and epizootiology[1], to identify and characterize disease-causing microorganisms, and to understand processes and stressors that may trigger outbreaks of disease and exacerbate their impacts. In addition, resource managers need effective tools to control disease epizootics and reduce coral mortality.

The purpose of this paper is to summarize the type of information needed by resource managers to improve their ability to effectively manage coral diseases, and to discuss impediments hindering progress in coral disease research. Specific examples from the literature highlight some of the existing limitations of available data. The focus of this paper is on rapid ecological assessments, long-term monitoring, and targeted research to address gaps in the understanding of coral diseases, with a

[1]Epizootiology is the science that deals with outbreaks of animal diseases, including the prevalence and variability at local, regional and temporal scales, factors involved in the occurrence and spread, and the effect on host populations. It is the equivalent of human epidemiology.

discussion of possible strategies to link coral disease research with management objectives. Descriptions of regional coral disease monitoring protocols and techniques utilized by scientists illustrate the existing methodologies used to characterize coral disease epizootiology (Appendix I). Priorities for management of diseases identified by the Florida Keys National Marine Sanctuary in their draft science plan are also summarized (Appendix II).

Key priorities for effective management of coral reef diseases

1. Early warning systems for coral disease epizootics

The problem: Coral bleaching and disease outbreaks may be linked to unusual environmental events. Yet, it is difficult to reliably predict their occurrence or potential impacts, or to separate the relationships of these events with coral diseases from natural variations in disease occurrence.

The health of coral reef ecosystems is closely linked to physical properties of the environment. Certain global or regional-scale disturbances such as coral bleaching occur during periods of elevated water temperature, high UV radiation, excessive sedimentation and similar stressful events. Such events have been numerous in the Florida Keys (FKNMS) and some of these have been followed one to two years later by the appearance of a disease outbreak (Causey, pers. comm). However, the relative importance of large-scale phenomena such as global warming and El Niño as a cause of coral mortality, and how much these phenomena affect the prevalence and severity of coral diseases are unknown. By understanding the synchronization of various stressful events and the occurrence of coral diseases, managers may be able to predict potential threats in a timely manner and develop strategies to lessen impacts. This information may also assist in interpreting changes in reef condition that occurs in response to these events, and separate these from natural variations. Unfortunately, the wide distribution and remote location of many coral reefs presents challenges for *in situ* monitoring of unusual environmental events and their relationships with bleaching and disease outbreaks.

2 Detailed studies on the spatial distribution and temporal variations of coral disease outbreaks and other syndromes

The problem: Only limited quantitative information exists on the prevalence and incidence of diseases. Existing data may not be representative over large geographic or temporal scales, or at local scales, due to small sample sizes of corals, reefs, and/or depths and infrequent sampling.

A summary of all *in situ* observations (up to 1999) on the prevalence, range of species affected, global geographic distribution, and mortality for the "major" diseases has been compiled by Green and Bruckner (2000). This review highlights the large geographic scale over which diseases have been reported, but many of the records represent qualitative observations only. Currently, detailed quantitative data on the prevalence of diseases are available only for a few locations (e.g. Florida) and diseases (primarily BBD, WBD and white plague). There have been a few recent regional studies to evaluate the health of coral reefs and the occurrence of diseases. This includes the Atlantic and Gulf Rapid Reef Assessment (AGRRA) surveys conducted between 1998-2000 in 22 countries (Kramer et al., *in press*), and a CARICOMP survey of 19 reef sites from 6 widespread geographic localities (Weil, *in press*).

A. White-band disease (WBD)

White-band disease has been reported from most countries in the Caribbean, and it is believed to be the major factor responsible for the regional decline of *Acropora palmata* and *A. cervicornis* (Aronson and Precht, 2001). However, quantitative data on disease prevalence and incidence are available for few locations, and these show large local, regional, and seasonal variations (Table 3). In addition, long-term monitoring of WBD on relevant temporal scales was conducted only in the USVI (Gladfelter et al., 1977; Gladfelter, 1982; Gladfelter, 1991). WBD was observed on acroporids in many other locations, and extensive mortality occurred at some later date, but conclusive proof to verify that WBD was the leading (or only) source of mortality is not available. Localized losses of these species have been attributed to other factors (storms, predation, bleaching etc.). However, the importance of these factors versus WBD, or their relationships with WBD have not been fully characterized.

Table 3. An example of the variation in prevalence of white-band disease reported during the 1980s.

Prevalence of WBD	Location	Author
2-5%; up to 40%	Florida, USVI, Belize	Antonius, 1981
5-26%	British Virgin Islands	Davis et al., 1986
1-2%; up to 64%	U.S. Virgin Islands	Gladfelter et al., 1977
20-33%	La Parguera, Puerto Rico	Goenaga and Boulon, 1992
> 80%	Jamaica; Netherlands Antilles	Rogers, 1985

B. White plague

Studies from the Florida Keys suggest that white plague may have been a significant factor contributing to the declines in live coral cover over the last 20 years (Dustan, 1977; Dustan and Halas, 1987; Richardson et al., 1998). Plague type II has been one of the most severe disease events in terms of the number of coral species affected, the rates of tissue loss, mortality rates, and the rapid spread along the Florida reef tract (Richardson and Aronson, in press). However, it is difficult to evaluate long-term impact on coral populations because the disease has not been monitored at frequent intervals since it was first reported (Table 4). The value of frequent monitoring to accurately track the spread of a coral disease and evaluate its effect on coral populations is illustrated by a study from the USVI where researchers monitored coral populations monthly for about 4.5 years. They identified frequencies of plague type II that ranged from 3-58%, with new infections observed each month and an absence of a seasonal correlation in prevalence (Miller et al., in press).

The examples presented in Table 3 and 4 illustrate the high percentage of stressed, diseased and dying corals identified in surveys, but these studies do not distinguish between cause and effect, sources and/or causes of mortality. For instance, Dustan and Halas (1987) identified numerous dead corals in their transects which may have died from white plague, BBD, sediment-related necrosis or other factors.

Table 4. Reports of white plague from the Florida Keys.

Species	Description of event	Location	Author
M. ferox, M. lamarckiana, C. natans, M. annularis, P. astreoides, S. michelini	1) Outbreak observed in 1975; 2) Highest prevalence in M. ferox (24-73%); 3) Spread of up to 3.1 mm/day in *M. ferox*; 4) Mortality of 20-30% of *M. ferox* in one year.	Carysfort, Elbow, French, Key Largo Dry Rocks, Molasses	Dustan, 1977
Species with plague not identified	1) Corals at 10-21 m declined in abundance between 1974-1982; 2) Skeletons of *M. ferox, M. lamarckiana, S. hyades* and *C. natans* recorded along transects; 3) BBD, white plague and sediment-related tissue necrosis prevalent.	Carysfort	Dustan and Halas, 1987
Species with plague not identified	1) 362 out of 9800 (3.69%) colonies with white plague; 2) Prevalence same on all reefs (no "hot spots").	19 reefs in Key Largo	Dustan, 1993
A. agaricites, A. lamarcki, C. natans, D. cylindricus, D. stokessi, D. labyrinthiformis, D. strigosa, E. fastigiata, M. decactis, M. mirabilis, M. areolata, M. meandrites, M. annularis, M. cavernosa, S. siderea, S. bournoni, S. michelinii	1) Outbreaks of Plague type II from 1995-1997; 2) Plague type II spread throughout the Florida Reef Tract; 3) Epizootics confined to the middle keys in 1995, southern keys and the Dry Tortugas in 1996, and reefs north of Miami in 1997; 4) Up to 38% mortality of one species; 5) Seasonal occurrence (June-October)	23 reefs over 200 km reef tract	Richardson et al., 1998
C. natans, M. faveolata	Outbreaks of plague type III reported from Florida in 1999		Richardson and Aronson, in press

Some of the possible pitfalls associated with a limited number of surveys within and among reefs and surveys conducted infrequently are:

- Diseases often occur at a low frequency, but they may be clumped in occurrence. Surveys that are not representative, or are too few number, may miss localized aggregations of diseased corals (underestimating diseases) or they may focus on localized areas affected by multiple infections (overestimating disease prevalence).

- Diseases often affect individual hosts for a relatively short time, with new infections appearing on surrounding corals at frequent intervals. Surveys conducted infrequently are unable to accurately assess the duration of individual infections and their role in coral mortality.

- Some diseases exhibit seasonal variations in abundance or occurrence. Surveys conducted during certain times of the year may incorrectly estimate disease prevalence or incidence, and the impact of diseases.

- Coral mortality may be caused by numerous factors, in addition to disease. Infrequent surveys may overestimate the role of disease in coral mortality, by not identifying other causes of mortality and their relative importance.

3. Relationships between coral diseases and environmental factors, anthropogenic stresses and natural disturbances

The problem: The recent emergence of diseases is often presumed to be related to habitat degradation, increased human impacts, or poor water quality, but linkages with specific factors have not been conclusively determined. Reef managers needs to recognize and partition various threats that may affect the occurrence, prevalence or severity of diseases so their sources can be identified, and possibly reduced through better management practices.

Although it has been difficult to identify the cause of most new diseases, the sudden emergence may be related to deteriorating and changing environmental conditions (Santavy and Peters, 1997). For example, eutrophication may be an important source of stress that compromises a coral's immune system and decreases the resistance to disease, and it may also promote proliferation of pathogens, allowing new diseases to emerge (Hayes and Goreau, 1998). Green and Bruckner (2000) mapped the spatial distribution of coral diseases, and noted that 97% of all locations in the Caribbean affected by coral diseases correspond to areas where human activities are expected to have medium to high impacts, based on the Reefs at Risk[2] analysis.

[2] *Reefs at Risk (1998)* presents an indicator of potential threats from coastal development, overexploitation and destructive fishing, impact of inland pollution and erosion, and marine pollution based on a series of distance relationships correlating mapped locations of human activity. It also considers population demography, rainfall, and topography of associated land masses.

Porter and Tougas (2001) presented a coral disease stress model that links environmental quality with incidence of disease. In this model, a variety of stresses (elevated temperature, increased storm frequency, elevated nutrients and toxins and decreased water clarity) are predicted to suppress the immune and disease defense systems in corals. The consequences of reduced health include increases in the number of pathogenic organisms, susceptible species, locations affected by disease, and mortality rates. The authors suggest that the model is applicable to the Florida Keys, where a dramatic increase in the number of diseases, the number of species affected, and the rates of coral mortality were recorded in 40 sites between 1996-1998.

Previous studies quantifying the spatial and temporal distribution and abundance of BBD illustrate the widespread distribution of the disease, but infections exhibit significant variations in prevalence and dispersion (Table 5). BBD occurs on most reefs at a low abundance, although infections may be aggregated, and outbreaks have been reported from numerous locations. Goreau et al. (1998) reports that BBD often first appears 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 from eroded soil. 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. Antonius (1998) identified BBD on reefs in the Red Sea located in close proximity to industrialized areas, with a notable absence of the disease in other locations. 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.,1997). The incidence of BBD was not correlated with run-off in the most commonly affected species (*M. annularis*; *M. cavernosa* and *Diploria* spp.). However, one species that is thought to be highly resistant to BBD (*S. siderea*) exhibited few infections prior to the rainfall event, with a dramatic increase in bleaching, stress-related necrosis and BBD in the second year, corresponding to periods of high rainfall and run-off. Richardson (1995) compiled information on BBD incidence versus nutrients, temperature, light, turbidity, salinity, coral cover and coral diversity for 200 sites in the Florida Keys. This study may further clarify the role of environmental quality; however, at least initially, she noted that there is no clear correlation.

An extensive, multi-year study conducted in Puerto Rico by Bruckner (1999) suggests that a negative relationship may exist between BBD prevalence and turbidity or elevated nutrients. The lowest prevalence of BBD overall was found near Mayaguez and Ponce, which are some of the most polluted and turbid sites in Puerto Rico due to high sedimentation and nutrification associated with river discharge

and direct input of untreated sewage. In addition, the incidence of BBD on a fringing reef off the west coast (Rincon) 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. Furthermore, high turbidity was one of the major factors limiting the spatial distribution of BBD in La Parguera. Infections were restricted to shallow water 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 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 or agriculture (there is a small DNER facility and camping is permitted) and reefs are unaffected by river discharge (Bruckner, 1999).

The role of physical factors (temperature, light, wave action), anthropogenic stressors (nutrients, sediments, pollutants) and other environmental stressors must be carefully evaluated before concrete conclusions of relationships with disease can be made. Most coastal environments are affected by a suite of anthropogenic inputs, making it difficult to identify any one specific cause associated with deteriorating health or an increased prevalence of disease. Differences in prevalence, severity or impact of diseases may be related to specific anthropogenic stressors, other biotic or abiotic parameters or multiple factors. For instance, habitat characteristics and composition including the cover and abundance of susceptible corals, the amount of macroalgae, or presence or absence of certain key indicator species such as *Diadema* may influence coral diseases (Table 5).

Table 5. A comparison of published quantitative studies on black-band disease from the Caribbean.

Location	Survey information	BBD prevalence	Habitat and environmental characters	Author
USVI	22 sites on 7 reefs; 9204 corals; 4 surveys in 1 year.	0.2% of all corals; 5.5% of *D. strigosa*; random dispersion; BBD declined in November.	high density of corals (1.3 colonies/m^2) but only 24% of all corals in study area were susceptible to BBD; undeveloped watershed.	Edmunds, 1991
Florida	19 reefs; 9800 corals; single survey.	6%; same prevalence on all reefs.	surveys in 1980s; higher coral cover and abundance than in the 1990s.	Dustan, 1993
Florida	10 sites on 3 reefs; 1397 corals; 3 surveys in 1 year.	0.72%; clumped distribution; seasonal occurrence, but some corals infected year-round.	low density of corals (0.15 colonies/m^2); large annual decline in temperature; possible water quality issues.	Kuta and Richardson, 1996
Jamaica	20 sites on 4 reefs; 5564 corals; bi monthly surveys for 20 months.	5.2% total over 20 months; max 1.2% at one time; clumped distribution; new infections during each survey; no decline in winter, except in *M. cavernosa*.	>90% of the corals in sites were susceptible to BBD; moderate density of corals (0.9 colonies/m^2); seasonal periods of high rainfall runoff.	Bruckner et al., 1997

4. Standardize nomenclature to describe, identify and differentiate diseases and other syndromes

The problem: Inconsistent terminology for coral diseases, absence of standard requirements for reporting of new diseases, and lack of field identification tools hinders our ability to obtain accurate information on the prevalence and impact of diseases.

There is considerable disagreement among coral disease specialists about many of the fundamental aspects of the newly emerging diseases. This includes a profusion of names describing so-called "new diseases" which may represent coral syndromes caused by closely related pathogens, predators, or other factors (Goreau et al., 1997; Bruckner and Bruckner, 1998, Richardson et al., 1998). In the majority of cases, researchers have assigned names to coral syndromes based on the appearance of the microbial community (BBD), the color of the affected tissue (YBD), or the pattern of exposure of coral skeleton (WBD). In a recent review of coral disease literature, Green and Bruckner (2000) noted that 29 different "disease states" had been reported. In some cases, a variety of similar names were used to describe different diseases; in others, different names were used by separate researchers to describe apparently similar disease signs. For instance, the term white-band disease (which occurs only in *Acropora* spp. in the Caribbean) has been used to describe similar signs in massive and plating corals that are reported elsewhere as white plague (Table 1). Also, similar gross signs of coral abnormalities that are classified as the same disease have been later shown to have different histological characteristics (Santavy and Peters, 1997). There has also been an uncertainty as to whether what is being described as a coral disease is actually a biotic or abiotic disease, or whether it is caused by other factors. This includes rapid wasting disease (later renamed rapid wasting syndrome), which has been shown to be the result of focused biting by *Sparisoma viride* (Bruckner and Bruckner, in press), a phenomena that was documented over 100 years ago. Another example is ridge mortality disease (Abbott, 1979), a condition that affects brain corals and other species. This condition is always associated with *Stegastes planifrons* predation and the development of algal lawns (Humann and Deloach, 2001), and has been termed damselfish ridge-denuding syndrome by other authors (Williams and Bunkley-Williams, 2000).

In the last five years there have been numerous "review articles" on coral diseases (Cervino and Smith, 1997; Cervino and Smith, 1998; Santavy and Peters, 1997; Peters, 1997; Bruckner and Bruckner, 1997c; Richardson, 1998; Goreau et al., 1998; Hayes and Goreau, 1998; Harvell, et al., 2000; Williams and Bunkley-Williams, 2000; Borneman, 2001; Richardson and Aronson, in press). Many of these provide descriptions and lists of reported diseases, but the summaries often lack a thorough review of existing literature, and may be heavily biased towards work by one or a few researchers. In addition, some of these articles have been published without a thorough and unbiased peer-review. In some cases,

these articles have discounted many of the presumed diseases, while they identify additional "new" syndromes without any supporting quantitative field or laboratory studies. In other cases, even after considerable supporting evidence is available that discounts a presumed disease, references to this condition continue to appear in scientific literature and popular articles. Along with the continued controversy regarding many of the presumed new diseases, many of the articles draw conclusions on the recent spread, prevalence, associated factors, or potential impact based on qualitative information, in absence of sound quantitative science. Without a coordinated and cooperative effort among all researchers working on coral diseases, this misinformation is likely to continue to proliferate, severely affecting our ability to respond to a growing coral reef crisis.

5. Factors that facilitate the occurrence, spread and transmission of pathogens

The problem: The source of emerging diseases and their mode of spread and transmission is unknown.

It is currently unclear whether the recent emergence of diseases is associated with the introduction of pathogenic organisms from terrestrial environments, or whether disease-causing microbes have always been present but have only recently become pathogenic due to deteriorating environmental conditions and/or reduced host resistance. A critical area of research is the identification of disease sources and vectors, and natural reservoirs for pathogens. Possible sources include the introduction of novel pathogens via aerosols (e.g., African dust), run-off, sewage, vessel discharge, the introduction of exotic species as well as the movement of water masses as a vector for dispersal (NCORE, 2001).

Black-band disease now occurs in most reef environments in the western Atlantic. In many locations BBD increases seasonally in summer and fall, with infections becoming rare or absent during winter when water temperatures decline (Kuta and Richardson, 1996). Factors responsible for the reappearance the following year are not completely understood, but the occurrence of the dominant microorganism *(Phormidium corallyticum)* outside of the black band association has been reported. During surveys in Belize, Florida and the Bahamas, Taylor (1983) reported the occurrence of *P. corallyticum* from shaded areas under reef limestone not in association with living corals. Richardson (1997) identified non-pathogenic biofilms that contained *P. corallyticum* and other cyanobacteria in sediment patches on the surface of healthy, BBD-susceptible corals. However, none of these developed into active BBD after 12-17 months. Santavy and Peters (1997) also identified algal mats that contained *P. corallyticum* filaments at the base of a reef in the Bahamas.

Although *P. corallyticum* was not identified in plankton tows, injured colonies have become infected with BBD when placed downstream from an infected colony, suggesting that the disease is transmitted in the water column (Rützler and Santavy, 1983; Antonius, 1985). In Jamaica, a BBD epizootic was observed at the eastern end of a reef system and it progressed to the west over 20 months, in the direction of the major water current (Bruckner et al.,1997). In this study, portions of the BBD mat became dislodged during periods of high wave action, and floated towards other corals down current. Although the dislodged BBD mat was not observed to land on and infect a new host, a high number of new infections were identified 1 to 3 weeks after periods of high wave action (Bruckner et al., 1997). While water motion provides a vector for transmission of BBD, under extreme wave conditions, water turbulence may actually limit BBD progress and prevent new infections from becoming established. On exposed shallow (1-2 m) reefs on the north coast of Puerto Rico, a high incidence of BBD has been observed during spring and summer, coinciding with periods of low to moderate wave surge. In fall, although temperatures are approaching their maximum (29°C), BBD virtually disappears from exposed reefs; the decline corresponds to the onset of fall and winter storm waves. In certain protected locations, BBD reaches its maximum annual abundance during this period (Bruckner, 1999).

There is the possibility that pathogenic microorganisms may be transported into coastal environments along with soil during periods of run-off (Epstein et al., 1994). *Aspergillus sydowii*, a fungus identified as the cause of Aspergillosis in sea fans, is reported to be of terrestrial origin. One hypothesis is that *A. sydowii* was transported to coral reef environments via run-off; however, fungal isolates from the terrestrial environment are not pathogenic to corals. An alternate hypothesis is that *A. sydowii* was transported to Caribbean reefs in clouds of dust from western Africa (Weir et al in press).

6. Effects of disease on population dynamics, community structure and ecosystem function, including impacts on associated species

The problem: Long-term consequences of diseases on coral reefs are largely unknown. Managers need information on the fates of corals, the resilience of coral populations, potential effects on associated species, and long-term impacts on coral reef composition, structure and function.

Hayes and Goreau (1998) concluded that mortality from coral reef diseases has the potential to be more detrimental to the survival of coral reefs than sedimentation, pollution, physical degradation, or all other threats combined. It is clear that coral diseases can have a major impact on the structure and composition of coral reef ecosystems, as evidenced by the regional spread of WBD and devastating levels of mortality to acroporid populations. However, the fates of individual colonies, the resilience of

coral populations, and the long-term effects on associated species and the ecosystem are a complex interaction of multiple factors. For instance:

- The degree of threat is likely to vary among diseases and affected species, with additional seasonal and spatial variations and possible synergistic impacts from other disturbances.
- The role of coral disease as a community structuring force depends on the magnitude, duration and frequency of reappearance of the disease, and the recovery time of the system.
- The recovery time depends on the life history strategies of the impacted species.
- The impact from disease varies depending on the overall condition of the reef, including the cover and composition of reef building corals, the presence and abundance of critical indicator species (e.g. *Diadema*), and the degree of physical, environmental and anthropogenic stressors.

A. Fates of infected corals

Coral diseases can cause rapid rates of tissue mortality, but spreading rates are highly variable depending on the disease, affected species, location and season (Table 6). Diseases may produce mortality of affected corals over varying periods of time, although diseases often cause only partial mortality (Bruckner and Bruckner, 1997). While corals have the potential for recovery once the insult is eliminated, the reduced size of affected colonies may affect growth, reproduction, resistance to injury and other diseases, and competitive abilities of the surviving polyps (Edmunds, 1991). Colonies affected by disease are vulnerable to reinfection, infection by other diseases or subsequent attacks by predators (Kuta and Richardson, 1996; Bruckner and Bruckner, 1997; Antonius and Riegl, 1997; 1998). For instance, Bruckner and Bruckner (1997) reported a recurrence of BBD later in the season or in subsequent years (1995-1996) in 39 corals (11.4%) identified with BBD in 1992 or 1993.

B. Effects of disease on coral populations

Coral diseases, once they invade a reef system, may become a chronic feature of that system which undergo periodic escalations in abundance and virulence (Bruckner, 1999; Richardson and Aronson, in press). From an evolutionary standpoint it is not beneficial for a pathogen to eliminate all of its host species. A coral species may occasionally suffer periods of high mortality from disease, however survivors are likely to persist by becoming resistant, the pathogen may undergo a decrease in virulence, or disease may occur on a wider range of host species or over a wider distribution. For instance, Dustan (1977) observed an epizootic of white plague in 1975 that was predicted to result in the disappearance of *Mycetophyllia ferox* from Carysfort Reef in the Florida Keys. This species still occurred on this reef in 1984, and these were not observed with white plague, suggesting a possible resistence to the pathogen

Table 6. Spreading rates of black-band disease and the extent of mortality attributed to infections.

Affected species	linear spread	Tissue loss (observation period)	Total loss/season	Location & Source
M. annularis (n=2) *D. strigosa* (n=1)	0.3-6.2 (mm/day) 2.4-4.0 (mm/day)	64-746 cm^2 (41 days)	1509-4820 cm^2	Belize; Rutzler et al., 1983
D. strigosa (n=12)	no data		7 colonies lost >75% live tissue; 3 lost 50-74%; 2 lost <25%	USVI; Edmunds, 1991
S. siderea (n=93) *M. cavernosa* (n=48)	no data	1810 cm^2 (2 years) 1630 cm^2 (2 years) Small colonies (mean=239 cm^2 died in 1-8 months.	Large colonies of Ss lost 50% live tissue in 2-3 years; large colonies of Mc lost 68% of live tissue.	Jamaica; Bruckner and Bruckner, 1997
M. annularis *D. strigosa*	up to 1 cm/day			Antonius, 1981
M. annularis *M. faveolata* *M. franksi* *M. cavernosa,* *D. strigosa* *D. clivosa* *C. natans* *S. siderea* (n=210)	0.5-8.1mm/day Variations among species, reefs and depths. Slowest spread among *S. siderea* in deep water and fastest among *M. annularis* in shallow water.	1.3-100 cm^2 /day 107-1329 cm^2 total Mean duration of BBD= 46-220 days.	Mortality rates depended on species, reef and depth and also size of coral, length of BBD and duration of BBD. Mean loss (pooled by species) ranged from 18%-96%.	Puerto Rico; Bruckner, 1999
M. faveolata (n=9) *D. strigosa* (n=51)	4.1 mm/day 2.0 mm/day			Honduras; Griffin, 1998

(Dustan, 1987). In contrast, *M. faveolata* colonies showed little signs of tissue necrosis in 1975, while many were infected with white plague in 1985 (Dustan, 1987). More recently, Richardson et al (1998) reported an outbreak of white plague in the Florida Keys which initially targeted *Dichocoenia stokessi*, a species that was not affected in the 1970s or 1980s. Between 1995-1997, white plague spread among 17 coral species; greater observed rates of tissue loss suggest involvement of a more virulent pathogen than that reported by Dustan (Richardson, 1998). The outbreak was one of the most severe disease events in terms of diversity of species affected, mortality rates, and rapid rate of spread throughout the reef tract, but it had minimal long-term impacts, disease incidence was greatly reduced in these sites in subsequent years, and the pathogen identified as the cause no longer appear to induce disease signs (Richardson and Aronson, in press). Another outbreak (Plague type III) was observed in 1999 on the same reefs; rates of tissue loss exceeded that reported for both plague type II and plague type I, and large faviid corals appeared to be most susceptible (Richardson and Aronson, in press).

C. Effects of diseases on the ecosystem

When diseases have particularly severe impact on populations of a single species, the effects can extend over the entire ecosystem. Diseases may exert a major control on biodiversity by eliminating certain highly susceptible species. These losses may also reduce the capacity for the ecosystem to sustain associated species. Until about 1980, more than 95% of the total coral coverage in the reef crest and shallow fore reef consisted of *A. palmata* and *A. cervicornis*, with colonies forming extensive high relief thickets (Gladfelter et al., 1977). Populations throughout the Caribbean were reduced to unprecedented levels during the 1980s and 1990s (Aronson and Precht, 2001). The combination of coral mortality, declining herbivory (associated with the *Diadema* die-off and overfishing of parrotfish and surgeonfishes) and possibly elevated nutrients has precipitated a phase shift from coral to macroalgal dominance (Hughes, 1994; Aronson and Precht, 2000). In many locations, *A. palmata* colonies suffered near total mortality, but erect skeletons remained in growth position, continuing to provide high-relief habitat. However, exposed skeletal surfaces were rapidly colonized by invertebrates and algae and skeletons have become bioeroded, dislodged and removed during storms. The collapse of acroporid skeletons is associated with the elimination of high relief, three-dimensional topography. Long-term consequences may include loss of critical habitat and refuge for a wide number of associated species, reduced rates of reef accretion, and reduced shoreline protection from tropical storm waves.

7. Measures to mitigate disease impacts

The problem: Resource managers need tools to combat disease epizootics, prevent future outbreaks, and reduce the time needed for recovery of affected reefs and species

In the past two decades numerous reef mitigation and restoration projects have been undertaken in response to coral mortality, in attempt to offset these losses. While the majority of these projects have been undertaken in response to direct human impacts (e.g. ship groundings), limited restoration efforts have been conducted to rehabilitate areas degraded by coral mining, blast fishing and other human impacts. A variety of treatments have also been attempted for corals with disease, including application of antibiotics, removal of the microbial community, shading, and biological controls (Peters, 1997; McGrath, 1998; Bruckner, 1999; Franklin, 1998; Borneman, 2001). These have varying success, but experiments have been conducted on a very small scale and often only limited efforts have been made to determine the long-term benefits. As an alternative to direct treatment of diseased corals, novel biological restoration approaches are being applied in Florida and other locations. These approaches are designed to enhance recruitment of corals, reduce coral mortality, and improve habitat quality, and

include the reintroduction of *Diadema antillarum* as a tool to reduce macroalgae and induced settlement of coral larvae (Miller, unpubl data).

In response to an outbreak of BBD in the Florida Keys in the 1980s, Harold Hudson attempted to treat colonies through aspiration of the BBD using a boat-operated vacuum pump, followed by application of modeling clay over the affected area to prevent reinfection. Unfortunately, colonies were not tagged or followed to determine their fates. In 1998, Hudson and Franklin repeated their initial experiments in response to another outbreak of BBD. An experimental approach to treat BBD corals was undertaken by Bruckner (1999) in Puerto Rico. The experimental design included 1) removal of the BBD using a large hypodermic syringe to aspirate the BBD . In these experiments, the affected area was unmanipulated, covered with modeling clay, or covered with underwater putty; 2) application of underwater putty directly over the BBD; 3) shading using various grades of sun screen; and 4) addition of *Diadema antillarum* to cages containing BBD-infected corals. All treatments were effective to varying degrees, with the highest success (>95%) achieved with the use of underwater putty (Bruckner and Bruckner, 1998d). Limited attempts have also been undertaken to treat corals with YBD (Bruckner, unpubl. data) and white plague (Miller, et al., in press).

Putty proved to be a highly efficacious treatment which prevented further destruction of century-old colonies, and may be most suitable when applied on a small scale for those colonies most important to the persistence of the reef (e.g., the largest colonies of slow-growing massive species). However, it may be impractical to treat a large number of corals due to the time required to manipulate infections and the expense associated. Because BBD is spread by water motion, care must also be taken to remove the bands without liberating filaments into the water column, which could result in additional infections downstream.. There are a few other limitations associated with treatments discussed for BBD: 1) aspiration of BBD followed by application of clay may be unsuccessful because any remaining *Phormidium* filaments are capable of emerging from within clay; 2) shading may cause coral bleaching; 3) *Diadema* may injure coral tissue when grazing pressure exceeds available algal resources.

Hayes and Goreau (1998) recommend rapid isolation and removal of diseased individuals or colonies to provide protection for other reef constituents against the spread of disease. However, this approach is likely to have minimal benefits, as the removal of an infected host may actually promote dispersal and spread of the pathogen, and it may result in considerable habitat damage. Furthermore, low background levels of disease may be a normal occurrence on most reefs that require minimal management interventions, while a disease epizootic is of much greater concern and yet it would be very impractical to remove all corals affected during a disease epizootic.

Proposed strategies to address management needs for coral diseases

1. Early warning systems for coral disease epizootics

Remote sensing of coral reefs, using high resolution satellite sensors, airborne LIDAR and hyperspectral coverage, and acoustic mapping methods are important tools to characterize ecosystem types and features, classify human use patterns, and determine sea surface temperature, water mass movements, light exposure and other physical properties. Satellite remote sensing measurements (ocean color, surface winds and insolation data) are being used in NOAA's Coral Reef Watch Program to conduct thermal bleaching surveillance of coral reefs world-wide. In this program, oceanographic data are integrated with a GIS spatial database to produce high resolution "HotSpot" (bleaching anomaly) charts available on the world wide web[3]. Also, a series of moored buoys are being deployed as part of a Coral Reef Early Warning System (CREWS) in the NWHI, U.S. Pacific, Florida, the Bahamas and other locations. These permanent oceanographic stations provide detailed *in situ* measurements of a variety of water quality parameters including water and air temperature, light (PAR and UV), nutrients, wind speed and direction, and water currents. Through the Coral Health and Monitoring Program (CHAMP) coral-list server, NOAA regularly announces sites that are affected by high sea water temperatures as a warning for potential bleaching. Researchers are then able to verify the onset of bleaching, and subsequently monitor impacts associated with the event. For instance, these data demonstrated accurate capabilities for detection of El-Niño induced coral reef bleaching in 1998. Some coral diseases become more prevalent during warm-water periods, and "HotSpot" data may also prove useful in understanding relationships between coral bleaching and disease. It is important to link oceanographic data with rapid assessments for disease. Real-time information on environmental stressors could provide valuable insight into identifying indicators of reef health, and factors that trigger disease outbreaks.

2. Detailed studies on the spatial distribution and temporal variations of coral disease outbreaks and other syndromes

Specific monitoring programs need to be developed to provide information on disease occurrence and progression at a variety of temporal and spatial scales using standardized techniques suited to answer specific questions on coral diseases. Because infections often occur at a low frequency and in a clumped

[3]NOAA/NESDIS Coral Reef Watch incorporates the Coral Reef Early Warning System (CREWS) and OAR/AOML international coral reef information system. This information is available through the Coral Health and Monitoring Program (CHAMP) Web Page: http://www.coral.noaa.gov.

distribution, surveys must encompass large areas to quantify the distribution and abundance of infected colonies (Edmunds, 1991). Surveys must also be conducted relatively frequently to identify temporal variability, quantify synergistic impacts, and distinguish mortality from disease from mortality caused by other factors.

An effective program can include rapid assessments conducted on a regional scale to elucidate large-scale processes such as the transmission of regional-scale water-borne pathogens, and to serve as an early warning system to identify and track disease outbreaks before irreparable harm occurs. Targeted monitoring can provide information on a particular disease, and intensive seasonal or weekly monitoring of specific reefs can help managers understand the effect of a particular disease at a local scale, or the role of a localized stressor on disease processes and impacts. Monitoring of individual colonies affected by disease is also necessary to document patterns of spread, rates of tissue destruction, impact of diseases at a colony or population level, and relationships of disease occurrence with other natural or anthropogenic impacts.

A variety of approaches have been utilized to identify the prevalence (number of infected corals divided by the number of corals examined) and incidence (number of new infections occurring in a population over a given time divided by the number of uninfected individuals at the beginning of the time period) of coral diseases, including quantitative, semi-quantitative and qualitative surveys (Table 7; Appendix I). Many monitoring programs rely on repeated sampling of permanently marked transects, quadrats, or radial study sites. Detailed monitoring of particular reefs, through the establishment of permanent sites, can provide information on disease incidence. In addition, long-term monitoring of individual tagged colonies affected by disease can provide detailed information on the rate of spread and amount of mortality sustained from an infection, the duration of infections, frequency of reinfection, and effect at the species level.

Monitoring programs that involves the reexamination of permanent sites may be impractical for sampling more than a few sites or reefs, and may underestimate or overestimate the prevalence or potential impacts. Because diseases often occur at a low frequency or they exhibit a clumped distribution, a large number of transects may be required within each reef to accurately estimate disease prevalence. To obtain a more realistic measure of disease prevalence overall at a larger (country-wide or regional) scale, and to determine factors responsible for the proliferation and spread of diseases, a repeated measures approach that utilizes multiple random belt transects positioned throughout each reef environment is necessary.

Specifically, a monitoring program implemented for coral diseases at the community level is needed to obtain information on:

- The type of disease/syndrome, coral species affected, and variability among species.
- The prevalence and incidence of diseases, the duration of a disease outbreak or bleaching event, and the consequences to affected species.
- Physical parameters (depth, water clarity, temperature etc.), biological factors (predators, algal abundance, etc.), environmental stressors and anthropogenic impacts associated with the outbreak.
- Local, regional and global distribution of diseases and temporal and spatial variations.

Table 7. Monitoring and assessment techniques used for coral diseases.

Monitoring Approach	*Condition examined.* Description of technique	Author
Timed swims	*All syndromes.* Diver swims for 30 minutes along a single depth gradient and examines all corals in a band approximately 2 m wide recording the type of disease, species and number of infections. Disease occurrence is categorized as rare (1-3 cases), moderate (4-12 cases), frequent (13-25 cases), abundant (26-50 cases), epidemic (51-100 cases) or catastrophic (greater than 100 cases).	Antonius, 1995
Radial sites	*Prevalence and incidence of BBD;* Total number of infected and uninfected colonies of each species present are recorded within circular area (10 m radius; 314 m^2).	Edmunds, 1991; Kuta and Richardson, 1996; Bruckner et al., 1997;
Radial belt transect	*All diseases on stony corals and gorgonians.* Total number of infected and uninfected colonies of each species present are recorded within the outer 8- to 10-m of an arc radius (circular area, 10 m radius; 314 m^2).	Santavy et al., 2001
Linear transects	*A. Changes in composition, size and cover.* Twenty 25 m transects on Carysfort Reef from 0.3 to 21 m depth. Permanent transects were extended parallel to another and subdivide an area approximately 25 m X 300 m. Mean colony size (length under the transect line) and distance between colonies were recorded; colonies were inspected for signs of mortality (infections, physically damaged areas, sediment covered tissue or other blemish). *B. Condition of stony corals.* Minimum of ten 10 m transects per reef/depth; surveys at 3 m and 10 m depth; record species, size, percent recent and old mortality of all colonies that touch the transect line; examine whole colony for diseases or other source of mortality.	A. Dustan, 1985 B. AGRRA, 1998
Belt transects	*A. Sea fan disease.* One or two 4 m X 12.5-50 m transects per country. *B: All diseases; corals and gorgonians.* Ten 10 X 2 m belt transects (level 1) and three 20 m X 2 m belt transects per depth minimum 9 transects at each site (level 2). *C. Reef condition.* Three 20 m X 5 m transects per reef; divers note presence and proposed type of disease/ affected species, but do not collect quantitative information.	A. Nagelkerken et al., 1997; B. CARICOMP (Appendix I) C. Reef Check
Quadrats	*A. BBD in Gorgonians*. Prevalence: three 100 X 250 m quadrats; incidence: one 10 X 10 m quadrat; one reef B. *Sources of mortality*. Photostations 2 X 2.25 m; 2 per reef; 2 reefs to identify sources and amount of mortality or time; complemented with permanent chain transects extended over 25 m to measure changes in percent coral cover, diversity, and colony size. C. *Coral reef monitoring.* 160 stations, each 2 m X 22 m; sampled 40 sites in the FKNMS. 15 minute survey of each quadrat to complete a species inventory, identify species that are affected with bleaching or disease and record the type of disease. No data collected on disease prevalence. Also run video transects. Sites reexamined once each year from 1996-2000.	A. Feingold, 1988; B. Porter, 1992 C. Jaap et al., 2000

3. Relationships between coral diseases and environmental factors, anthropogenic stresses and natural disturbances

If there is a link between anthropogenic influences and the prevalence of coral diseases, disease occurrence is unlikely to be randomly distributed at regional scales. An understanding of the global epizootiology of diseases constitutes a first step in examining relationships between human impacts and the occurrence of coral diseases. A comprehensive database needs to be developed linking all accurate field reports by trained observers as well as observations and photographs from amateur observers to understand the impacts of coral reef diseases on a global scale (Goreau, 1998). The Global Coral Disease Database[4], developed by NMFS in conjunction with UNEPs World Conservation Monitoring Centre provides a first attempt at analyzing the global distribution of diseases (Green and Bruckner, 2000). This web-accessible database provides information on the reported types of disease, species affected and global distribution of each disease. Disease occurrence can be mapped by particular disease, location or year. Currently, the database contains all published records identified through 2000, as well as volunteer disease reports; a form is available for reporting disease data electronically. The database is being expanded to include new regional monitoring data from AGRRA, CARICOMP and other sources, with a separate category for volunteer diver reports of disease (non-experts). Furthermore, additional layers need to be added to the GIS database that maps environmental and anthropogenic parameters. This information will facilitate interpretation of disease epizootics and causative factors involved in the development of these outbreaks.

In addition, targeted research that combines both laboratory and field methods is needed to understand the influence of various stressors on coral diseases, bleaching and mortality. These efforts should focus on establishing cause-effect thresholds of anthropogenic pollutants and environmental stressors, and capabilities for providing early warnings of disease outbreaks. For example this could include studies on:

- Mechanisms and factors that increase/decrease host susceptibility to disease.
- Cellular, mechanical, chemical and behavioral mechanisms that impart host resistence and host defense to diseases, and the ability of a host to counteract disease agents.
- Genetic adaptations and resistence of specific clones.
- Identification of biomarkers for stress to corals resulting from anthropogenic impacts or environmental perturbations.

[4]http://www.unep-wcmc.org/marine/coraldis/home.htm

4. Standardize nomenclature to describe, identify and differentiate diseases and other syndromes

A method to categorize disease terminology and standardize the list of recognized diseases is essential in attempting to assess the global implications of disease. Classifying the various visual categories of recent mortality as well as deviations from normal coloration or morphology is necessary to correlate damage with causes (Santavy and Peters, 1997). Field observations must be combined with techniques from biochemistry, histopathology, and microbiology to determine whether diseases are microbial or abiotic in nature, to identify and differentiate disease-causing microorganisms, megafauna (corallivores) and environmental stressors associated with each condition, and to develop molecular tools to validate field identification of diseases that are difficult to visually identify (e.g., Aspergillosis).

As a first step, the list of all known and presumed coral diseases (Table 1 and 2) should be evaluated for its validity. Accepted terminology should reflect the first name assigned to a condition, and terminology to describe presumed "new diseases" should be discarded if the condition was previously described. For instance, the condition "rapid wasting syndrome" described in 1998 was called "parrotfish white spot biting" in literature from the 1980s. Separate terminology should also be used for syndromes that visually differ to avoid confusion. This includes "yellow-band disease" which was first described in acroporids in the Arabian Gulf, and later used to describe an unrelated condition in faviid corals (primarily *M. annularis* complex) from the western Atlantic. Santavy and Peters (2000) recommended the use of yellow-blotch disease for the condition affecting *M. annularis*, thereby differentiating it from the yellow-band disease affecting Pacific acroporids.

Disease nomenclature may evolve from descriptive terminology to etiological and pathognomonic characterizations (e.g., Aspergillosis) (Hayes and Goreau, 1998). Hayes and Goreau (1998) suggest that conditions where no pathogen is identified may be better classified as a stress response, while pathogenic diseases should be renamed to indicate the specific organisms implicated as the causative agent. However, several contributing microbes may be required to produce a disease sign, such as that observed in BBD, making a single name (e.g., cyanobacteriosis) impractical or incorrect. In other cases, the proposed pathogen may not be the initial cause, but a secondary invader. Conclusive demonstration of the causative agent (and verification according to Koch's postulate) has proven difficult, and positive identification of the cause of some diseases may not be possible in immediate future. 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 plague may differ depending on the location, affected species, or other factors).

There are several sources of photographs and descriptions of coral diseases that can assist in developing a list of recognized coral diseases and descriptions to allow field identification of these diseases. These include 1) a coral diseases web site by McCarty and Peters[5] ; 2) underwater coral disease and predator identification cards by Bruckner and Bruckner designed to help differentiate signs of disease from predation (NOAA, 1999); and 3) a recent chapter *Coral health and mortality: recognizing signs of disease and predation* in Reef Coral Identification, Florida, Caribbean, Bahamas 2nd edition (Bruckner, 2001), which contains extensive descriptions and photographs of diseases, signs of predation, and coral overgrowth. A coral disease identification CD is being developed by NMFS and UNEPs-WCMC for distribution to dive shops throughout the Caribbean to complement volunteer diver disease identification programs. One additional source of information under development is a comprehensive coral disease website within the CHAMP database [6] designed to provide help in disease identification and research approaches for the laymen, scientists, managers, and disease specialists. This will contain an identification key for diseases, high resolution photographs showing diseased corals, tissue sections, and electron micrographs, a Frequently Asked Questions (FAQ) list, a glossary, contact information for coral disease researchers, a list of publications on disease, and PDF files of relevant publications.

5. Factors that facilitate the occurrence, spread and transmission of pathogens

A multi-disciplinary effort is necessary to conduct controlled experiments that evaluate possible sources of pathogens, identify mechanisms of dispersal and spread and develop strategies to reduce the potential for introduction and spread of pathogenic organisms. This includes:

- Evaluation of the role of water circulation as a vector for dispersal of pathogenic microorganisms.
- Examination of microorganisms transported in African dust, through species introductions, from discharge of ballast water, and other possible external sources of pathogens.
- Examination of microbial populations found in sediment adjacent to corals and microbiota associated with coral mucus.
- Evaluation of other possible sources of pathogens, including algal and cyanobacterial mats that occur in benthic habitats outside of disease associations.
- Identification of conditions and factors that promote the proliferation of pathogenic organisms and/or affect their virulence.

[5]http://ourworld.compuserve.com/homepages/mccarty_and_peters/coraldis.htm
[6]http://www.coral.noaa.gov/

6. Effects of disease on population dynamics, community structure and ecosystem function, including impacts on associated species

Long-term monitoring of individual colonies and species affected by disease should be conducted to determine their fates, factors that may increase or decrease the amount of mortality and their potential for recovery or recolonization, and the effects of coral mortality on associated species and coral reef community structure and function. Relevant studies include:

- Monitoring of individual colonies to determine the rate of tissue loss, extent of mortality, effect on physiological processes such as growth and reproduction (for corals that experience partial mortality), differences among and within diseases, coral species, seasons, depths and locations, and synergistic effects of other stressors.

- Evaluation of the fates of coral skeleton exposed by diseases, including the potential for corals to regrow over the affected areas, extent of colonization by algae and invertebrates (especially organisms that bioerode coral skeletons), and the extent of coral recruitment.

- Examination of the importance of reef-building coral species for associated species and in the composition, structure and function of reef communities, and the role of diseases in structuring communities and influencing relationships among predators, competitors and other organisms.

7. Measures to mitigate disease impacts

The ability for resource managers to effectively address coral disease epizootics and mitigate their impacts relies on an understanding of the causes, species affected, spatial and temporal incidence, and synergistic effects of climate change and human impacts. Research should focus on the development of techniques to prevent the occurrence of diseases, cure diseases and minimize the effects of disease. This includes:

- Strategies to reduce relevant stressors that are correlated with the occurrence, virulence or spread of diseases.

- Novel biological, ecological and physical techniques to improve the habitat quality and water quality, and potential re-introductions of ecologically important species (that were formerly abundant but have declined due to human pressures or other disturbances) including predators, competitors or other organisms that may help counteract the disease-causing agent.

- Treatments for corals with disease, including efforts to reduce host contact with pathogens and increase host defense.

- Restoration approaches to enhance coral recruitment and introduce disease-resistant clones of species impacted by disease that are unlikely to recover naturally in the short term. All efforts to mitigate diseases should involve hypothesis-driven experiments to determine the effectiveness of these approaches.

CONCLUSION

Most managers and policy makers are aware of the continued, rapid degradation of coral reefs, and they recognize that many of the stressors responsible for these changes are increasing in severity. Coral diseases are receiving increased attention by the media as an important source of coral mortality, but several factors hinder the development and implementation of strategies necessary for rapid response to diseases and management of disease-related impacts. This includes difficulties in obtaining funding for monitoring and targeted research needed to address many unanswered questions regarding diseases. It is also apparent that there is a lack of coordination among research scientists which must be overcome. 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 between diseases and other biotic and abiotic factors. To achieve an understanding of diseases at local to global scales requires considerable investment of time and effort to survey large areas of reef and to conduct these surveys at frequencies that are sufficient to document the duration of infections, seasonal patterns or chronic effects. These studies must be combined with research to elucidate relationships with environmental and anthropogenic stressors, which may help explain differences observed at regional to local scales and to identify approaches to mitigate disease impacts.

The policy and decision-making processes necessary for effective conservation of reef ecosystems, including the development of appropriate actions to manage coral diseases, are influenced by equally complex socioeconomic and political systems. There is a limited awareness and recognition of the potential global implications of disease by policy makers and the public who may have the tendency to overlook diseases, partly because they are not charismatic. Important decisions that must be addressed, such as the regulation of coastal development or implementation of strategies to control land-based run-off must occur over large spatial scales and be in effect for long time periods. The science in support of these decisions is often conducted on much smaller spatial and temporal scales, it may involve highly specialized research with results that are difficult to apply directly to policy decisions, or the science may be completely lacking. Given limitations of existing funding, coral disease research should be targeted towards efforts with the greatest management benefits that can be applied over large spatial scales and also are relevant to mitigate diseases and their impacts at the level of individual reefs or countries.

APPENDIX I: Monitoring and assessment approaches

I. Atlantic and Gulf Rapid Reef Assessment Protocol

The following description represents the benthic survey method for corals. The assessment technique involves 10 m transects placed along depth gradients at haphazard, non-overlapping intervals. The condition of corals that touch the transect are assessed. The full protocol can be accessed at: http://coral.aoml.noaa.gov/agra/method.htm

The AGRRA method is focused on assessing the condition of the principal scleractinian and hydrozoan corals that contribute most to the three-dimensional structure and complexity of reefs. The vitality of these corals responsible for the construction and maintenance of reef framework is important for the long-term persistence of a coral reef (Dustan 1987, Done 1997). The AGRRA method assesses overall coral cover, and for each coral, assesses the amount of partial coral mortality (both recent and old), size and height, incidence of bleaching/diseases, causes of mortality, and number of damselfish.

AGRRA makes a further distinction of mortality into partial mortality that includes both "recently" dead and "old" dead. "Recently dead" is defined as any non-living parts of the coral in which the corallite structures are white and either still intact or covered over by a thin layer of filamentous algae or mud. In contrast, "old dead" is defined as any non-living parts of the coral in which the corallite structures are either gone or covered over by organisms that are not easily removed (e.g., certain algae and invertebrates), even if the outline of the dead corallites is still visible below (e.g., certain zooxanthella-containing boring sponges that overgrow their substrates).

Causes of partial mortality include grazing (e.g., parrotfish bites), extended bleaching, sedimentation, algal overgrowth, disease (e.g., black band), corallivores, etc. In order to regenerate injured tissue, corals must reallocate energy that would otherwise be used for energy storage, growth or reproduction. It is fairly common for corals to experience partial mortality and to regenerate these areas if the lesions are not too large, relative to their remaining live tissues. Most partial mortality is small in size, but above certain sizes, which vary with species and the kind of injury that has been received, dead skeletal areas tend to become overgrown or eroded by algae or other bioeroding organisms (Meesters et al. 1996). In cases of large lesions, corals often will put energy into marginal growth instead of regenerating new tissue over the dead area.

By quantifying the amount of recent mortality, you sum the extent of any damage and obtain information on whether the corals are likely to recover. Several studies have shown that the extent and amount of partial mortality is related to differences in size, morphology, species identity, and differences in the spatial and temporal distribution of predators. Normally, small corals often have either no injuries or total mortality, while larger corals are likely to survive partial mortality (Hughes and Jackson 1980, Babcock 1985, Meesters et al. 1996). The AGRRA data can be used to calculate size frequency distributions as well as mortality patterns related to size and, when sample sizes are large enough, species identity. This allows the added benefit of being able to examine coral size-frequency statistics to assess a population structure (Bak and Meesters 1998) and interpret levels of mortality. Although there are studies showing the importance of size frequency distributions in population dynamics (e.g., Hughes and Jackson 1980, Done 1987, Bak and Meesters 1998), additional research is required to determine "normal" size frequency distributions for major reef building species in various habitats.

Areas of recent mortality and adjacent coral tissues should be examined closely to determine if calices are disrupted or damaged (fish bites), if corallivores are present (e.g. gastropods or polychaetes), if polipary structures are abnormally large or tumorous (hyperplasm or neoplasm), or if a disease condition exists. Diseases are recorded based on color, including black (black-band), white (white-band or other white-type diseases in acroporids; white plague in massive corals), yellow (yellow-blotch) or red (red-band). White areas must be examined closely to distinguish areas without living tissue (e.g. exposed skeleton caused by disease or predation) from bleached tissue. Dark-spot disease also has a mottled appearance, however it is manifested as irregular patches that are darker than the normal pigmentation and these areas may be slightly depressed. Predation can often be differentiated from disease (and bleaching) by examining tissues at the interface of white, exposed skeleton - diseased tissue will appear irregular and may be sloughing off the skeleton. Elevated "chimneys" on Acropora spp. and "ridge mortality" on brain corals are induced by repeated three spot damselfish (Stegastes planifrons) bites.

1.At each SITE, haphazardly lay the 10-m transect line just above the reef surface in a direction that is parallel to the long axis of the reef. Make sure the line is taut.

Note: Be sure to avoid or cross any other transects that are being set by your companions, and stay away from the edges of the reef. Also try to avoid areas with abrupt changes in slope, deep grooves, large patches of sand or unconsolidated coral rubble. You want to place the transect in areas where corals are likely to grow. Unusual reef features should only be included to the extent appropriate to their relative abundance at the site.

2. Approximate live coral cover by swimming along the transect line with your 1 m measuring device and roughly estimating how many meters of the line is underlain by living coral (to the nearest 10 cm). Be sure to include all live stony corals below the line, regardless of size or species density (but remember do not include dead skeleton). Record this total. If the reefs are too small for you to avoid sandy patches, record how much of the transect line crosses sand, to allow a later calculation of the number of coral heads encountered/m of reef surface.

3. Swim towards the other end of the transect and stop at the first coral head, cluster, or thicket (or a portion) of the appropriate species that is located directly beneath the transect line, is at least 10 cm wide, and which is in original growth position. For a colony that has fallen or been knocked over, only assess it if it has become reattached to the substratum or is too large to move. If the colony is loose and rolling around, then skip it. For each coral surveyed, record each of the
following:
A. Name (genus and species)

B. Record the water depth at the top of the corals at the beginning and end of each transect. In cases where bottom topography is very irregular, or the size of the individual corals is very variable, record the water depth at the top of each coral beneath the transect line at any major change in depth (>1m).

C. Identify the colony's boundaries based on connective or common skeleton, connective living tissue, polyp size, and polyp color. Using a measuring device, measure to the nearest cm, its maximum projected diameter (live + dead areas) in plan view and maximum height (live + dead areas) from the base of the colony' substratum, not the reef's substratum or ocean floor. The diameter should be measured perpendicular to the axis of growth. The height should be measured parallel to the axis of growth. Plan view is assessed from an angle that is parallel to the axis of growth.

Note 1: Some surveyors may want to measure all sizes of corals (even <10 cm max. diameter) on reefs on which large colonies are scare. They are encouraged to do so, but only data based on corals greater than 10 cm will be directly comparable to other AGRRA results). In previous years we suggested measuring corals >25 cm, but based on consensual agreement we have changed it to >10cm.

D. Estimate the percent (%) of the coral that is "recently dead" and the % of the coral that is **"Long dead"** as viewed from above in "plan" or "map" view. Plan view is assessed from an angle that is parallel to the axis of growth (be prepared to tilt your head to find the axis of growth and establish the proper plan view).

"Recently dead" is defined as any non-living parts of the coral in which the corallite structures are white and either still intact or covered over by a layer of algae or fine mud. For recent mortality, there are several "stages" of recent

Very recent = white intact skeleton is still visible (dead w/in 1 month or less)
Recent = corallite may be covered by thin turf algae or sediment (up to 6 months)
Older recent = corallite structure may be slightly eroded or covered but can still identify to genera (< 1-2 yrs), unless covered by clionid sponge

E. Combine all of these recent mortality categories into RECENT. In some cases circular or oblong lesions or excavations caused by fish biting may result in destruction of the corallite. If fish bites are identifiable and constitute part of the mortality, consider it as recent mortality.

Note 1: "Long dead" is defined as any non-living parts of the coral in which the corallite structures are either gone or covered over by organisms that are not easily removed (certain algae and invertebrates). If it is entirely "long dead", indicate this on your data sheet as 100% "long dead", as long as you can identify it to generic level based on morphology (e.g., Acropora palmata) or skeleton (e.g., Diploria sp.).

Note 2: In some cases, a coral may be partially or completely overgrown by the brown encrusting sponge, Cliona sp. At a quick glance, it may look like live coral tissue, but if you look closely you may observe the in/ex- current holes of the sponge and sponge tissue and will not observe live coral polyps. If you can see the coral skeletal structure underneath and are able to identify it to genus (e.g., Diploria and Montastraea), this should be considered old mortality and you should note Cliona overgrowth in comments.

Note 3: If a coral like columnar M. annularis or Dendrogyra has been knocked over, has either reattached to the substrate or is too large to move, has started to regrow towards the water's surface, it now has a different diameter and height because of its new growth direction. You should measure diameter, height, and mortality in the correct plan view along the new axis of growth. Only measure the recently "live" part of the M. annularis when estimating mortality and not the old "base" because this will skew old mortality.

Quickly scan over the surviving portions of the ENTIRE coral colony and note if there are any DISEASES and/or BLEACHED tissues present. Characterize any DISEASES by the following color categories:

BB = Black band
WB = White band
WS = White spots, patches or pox
WP = White plague
YB = Yellow blotch (sometimes called yellow band)
RB = Red band
UK = Unknown

II. CORAL-OCTOCORAL DISEASE SURVEY - PROTOCOL LEVEL-2 CARICOMP. Modified by E.Weil June of 2000.

OBJECTIVES:

To assess the occurrence (incidence at population and community levels) and local/geographic distribution of coral and octocoral diseases across the wider Caribbean region.

More specifically : 1- To quantify relative frequencies of infected colonies in each coral species affected within and across localities. 2- To quantify relative frequencies of the different diseases within and across species and within and across localities. 3- To determine if there is a correlation between frequencies of disease corals with depth, and distance to anthropogenic affected areas.

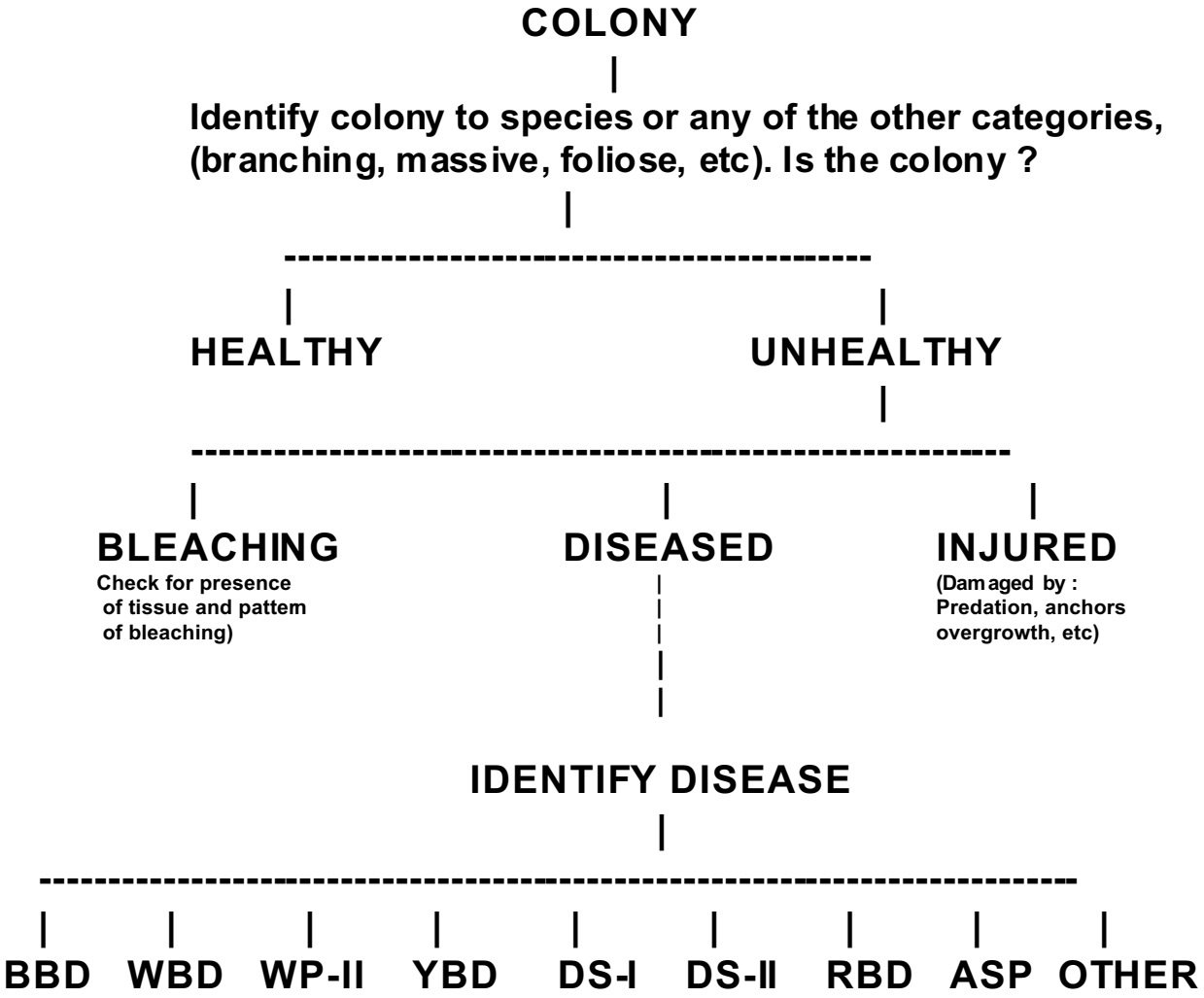
LEVEL- 1 - PROTOCOL:

Protocol will work with many different sampling methods (quadrats, chain transects, belt transects, etc.). This is to be carried out at the two reef sites of the CARICOMP reef monitoring using the chain. The survey is organized in a hierarchical way that will facilitate the decision-making process. A set of bilingual-underwater-laminated cards (Bruckner & Bruckner & Weil) was selected to be distributed amongst all site directors. This ID cards are to be used as a the ONLY guide to identify the different diseases and other causes of coral mortality. This will serve as a tool to standardize the identification of the different diseases and causes of mortality. There are ten, 10 m long chain sections per locality. Survey should be conducted over a belt transect of two meters wide (one meter on each side of the chain) by ten meter long (20 m²), for a total of 200 m5 per locality at one depth interval. All coral and octocoral colonies within this area should be checked and counted. The sample unit is one colony (ramet). Each coral colony should be identified to species (or genus at least) and/or to either one of the five colony form descriptors specified in the CARICOMP manual (branching, crustose, massive, foliose or milleporid). Octocorals should be ID at least at the genera level, or using the four cathegories (Ros, fan, feather, whip) in the manual.

The first discrimination-category for each colony is if it is healthy or unhealthy. Healthy colonies are those without signs of disease, recent injury and/or bleaching. Unhealthy colonies are those with any signs of disease, recent, open injuries and/or bleaching. A non-healthy colony will then be put into one of three categories, bleached, injured or diseased. If a colony has a disease and any of the other two signs (bleaching, injuries) it will be included in the disease category. Notes about the other symptoms taken. Bleaching should be verified by looking for the presence of live tissue (but decolored) and at the pattern of bleaching (usually not a band-like pattern like most Awhite-type" diseases; e.g. **pale, blotchy or white**). Injured colonies include those with damage from predation (often with skeletal damage), anchors or other physical damage, and overgrowth. If you can identify the type of damage (small lesions like individual bite marks or large excavations such a anchor damage) please do so. If the colony is being overgrown by other organisms (algae, zoanthid, sponge, hydroid, tunicates, etc.) it is important to note which organism (s) are overgrowing it. Colonies with old dead areas covered with algae but with healthy looking tissues fall into the healthy category.

A diseased colony will then be carefully observed to identify the disease that is affecting it. Observations should be compared with the information in the Q-cards to put the colony within any of the following 9 categories (see below and the flow diagram):

1- Black-Band Disease (BBD)	**Multispecific**
2- White-Band Disease (WBD)	**Types I and II (only in acroporids for the moment)**
3- White Plague-II (WP-II)	**Multispecific**
4- Yellow-Blotch Disease (YBD)	**Only described in species of *Montastraea* but also reported in other favid species.**
5- Dark Spots Disease I (DS-I)	**Small, dark areas with no apparent tissue mortality. Common in *Siderastrea spp.***
6- Dark Spots Disease II (DS-II)	**Large dark areas, larger than the DSD-I; common in *M. annularis* and *S. intersepta*.**
7- Red Band Disease (RBD)	**Careful here since BBD can be seen as red bands too. RBD has been reported for *Gorgonia* spp and Agaricids in the Caribbean.**
8- Aspergillosis (ASP)	***Gorgonia ventalina, G. flabellum, P. americana;* other octocorals (*Plexaura flexuosa*).**
9- White POX (WPX)	**Round or irregular white areas on surface of Acroporids.**
9- Other -	**this category includes all other "unconfirmed-pathogen-produced" diseases (tumors, hyperplasia, white pox, and all the "blotches").**



LEVEL 2 PROTOCOL - CORAL DISEASES .

The purpose of this activity is to assess the incidence of diseases in each coral and octocoral species at different depth intervals (habitats) and in at least two different reef sites per locality. The diseases will be identified following the protocol level-1 described above. The field experimental design is as follows :

1- At least two separated (more than 5 km) reef sites should be selected at each geographic locality (CARICOMP chapter), and preferably, one near and one far from anthropogenic-impacted areas. A preliminary qualitative, visual assessment of current levels of disease incidence and depth distribution within the two different reef localities should be done to determine if coral diseases are common or rare.

2- A quantitative assessment will follow using a modified protocol from the standard CARICOMP coral survey. Three depth intervals would be selected in those reefs with a depth profile to at least 45 feet deep. The depth intervals are: 0-15 feet; 16-35 feet and 35-50 feet, so major habitats would be surveyed (shallow, branching *Acropora* zone and boulder-crustose platform zone; intermediate depth *Montastraea* zone - massive, boulders and platy spp.; and the deeper more diverse zone - foliose, massive and platy spp). Re-bars or other permanent means of marking the transect position should be hammered into the substrate for future monitoring.

In each depth interval, at least three 20 meter long x 2 m (40 m²) wide band-transects should be surveyed (a minimum of 9 transects/reef, better if 12 transect per reef are surveyed). Two sets of 6 band-transect each should be separated at least 100 m. In each depth interval a set of two band-transects could be done using a 50 m long, plastic tape. Once the tape is positioned, two 20 x 2 m bands separated by a 10 m interval will be surveyed (from 0 to 20 m and 30 to 50 m). A one m long pvc section, marked in cm (100 cm) will be moved along each side of the transect line and every colony of coral and of *Gorgonia* spp. will be surveyed using the protocol level 1 described above.

Once a colony is found to have a disease ,two diameters (maximum and minimum) will be measured with the pvc stick to estimate surface area. ***ONLY disease colonies (corals and Gorgonia spp) will be measured***,

Separation between the consecutive band transects (at same depth interval) should be at least 10 m. If the reef areas do not have a depth profile or slope, or are such that only two depth intervals can be selected, the minimum of 12 band-transects should be then spatially distributed within the area. All coral colonies should be recorded to species (or genus at least) level.

Confusion of what an individual colony or ramet is will probably arise when branching, columnar or massive species that have suffered fission (fragmentation and/or partial mortality) are encountered. For the purposes of this protocol, a ramet is any colony that is spatially separated from any other colonies of the same species. On some occasions however, it is clear for some fragmented colonies, that in the near past, there was a physiological and physical connection between two or more ramets (i.e. in massive colonies suffering partial mortality or broken branches nearby the mother ramet of ramose species). In these situations, instead of counting 2000 small fragments (i.e. *P. porites* or *M. mirabilis*) one should use common sense and spatially delimit the spread of the individual genet (in the many ramets) and count it as a single ramet (genet).

The study is aimed at comparing the incidence of coral diseases between different reefs within a given location (Cuba, Panama, Venezuela, etc.) and across locations in the wider Caribbean. It is fundamental to the proposed experimental design to insure the sampling of large reef areas, in order to estimate current levels of disease incidence at a suitable spatial scale. Time window to conduct the surveys is between July and October. Frequency of surveys will depend on each individual laboratory=s logistics, time and personnel. A template (Lotus 123, Quatro or excel) to input data into the computer is available from E.Weil. If you decide to carry on this protocol, please contact Dr. E. Weil at eweil@caribe.net.

Materials and Methods utilized by Santavy et al., 2001 for coral disease surveys conducted in the Florida Keys.

General Approach

The SCUBA-based field study was developed and implemented over a two-year period in the south Florida region. Survey areas were selected in the Lower Florida Keys in the vicinity of Key West, the New Grounds, and the Dry Tortugas (Fig. 1). Zones that contained hard coral bottom were demarcated within each geographic area. These coral reef zones were located using a prototype of the Florida Marine Research Institute (FMRI) Benthic Habitats Map of the Florida Keys (FMRI, 1998). Potential stations for the pilot surveys were selected using a stratified random design, within the three regional areas. Individual stations were chosen by placing a random grid pattern that incorporated a hexagonal overlay over the individual coral reef zones contained within each geographic area (Summers et al., 1995). Surveyors went to randomly selected locations and assessed their suitability for sampling. If the location had sufficient coral coverage (>5%), the site was surveyed; if it was not suitable, the next location on the list was assessed for sampling suitability. Twenty-one stations were surveyed in the 1997 spring pilot survey.

Survey periods targeted late spring, the time when coral diseases are believed to emerge, and late summer, the time when coral diseases are believed to be most prevalent. The 1997 spring and summer pilot surveys were conducted during 1 to 8 June 1997 and 6 to14 September 1997. Stations assessed as suitable during the spring pilot could not be permanently established at that time, but permission was granted for permanent installation of stakes during the summer pilot. In September 1997, stations assessed during the spring pilot were relocated using GPS coordinates, and permanent sites were established by installing stakes to be used for future surveys in 20 of these stations (1 being omitted at New Grounds). At the same time, 6 new stations were added in the Key West and Dry Tortugas areas.

Survey strategy

Based on results from the 1997 pilot survey, reef type was added as a stratum to the 1998 sampling design (see Results). For the 1998 surveys, 6 additional stations were selected to balance the sample design across two strata: (1) three geographic areas established in the pilot surveys and (2) three reef types. The three reef types—fore reef, back reef, and transitional reef, as defined in the Florida Keys National Marine Sanctuary (FKNMS) Management Document (Dobbin, 1983; Jaap, 1984)—were used for comparison among the areas. However, not all areas contained all reef types; for example, only deep transitional reefs were found in the New Grounds area. The 1998 spring survey was conducted from 25 May to 1 June 1998 and the 1998 summer survey from 2 to 11 September 1998. Thirty-two stations were assessed in the spring and summer of 1998. A power analysis was performed on the data acquired from both spring and summer 1998 surveys to determine the optimal number of sampling stations and the appropriate α level for data analysis (Sokal & Rohlf, 1981).

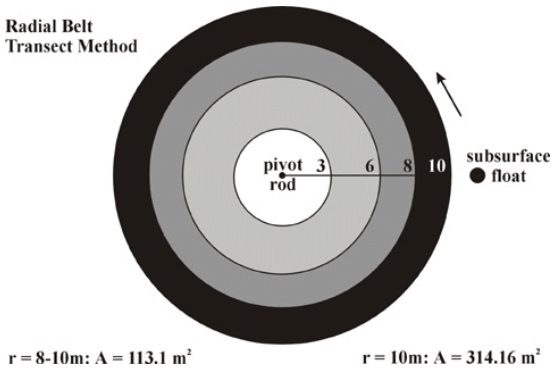
Survey methodology

All surveys were conducted using a radial arc transect method developed for this study. SCUBA was used on deeper reefs and snorkel was used on shallow back reefs. A stainless steel rod was positioned by driving it into the calcareous substratum for temporary sites (1997 spring pilot) or by permanently affixing a 12" stainless steel pipe (all other surveys) at the designated site with underwater epoxy (Gunnebo® Liquid Roc 500, Gunnebo Fastening Corporation 800-336-1640). Site coordinates were determined by GPS technology during 1997, then by Differential-GPS (D-GPS) during 1998 surveys as it became available in our study region, improving our ability to easily locate stations.

Surface maps with triangulation bearings and maps of underwater structures were generated, and a subsurface 3" buoy was used to mark each station to enable us to return to each underwater stake.

The survey procedure required inserting a 6-foot pole into the stainless steel pipe. The pole had 2 adjustable collets with a carabineer and a snap shackle on one end of the pole. A 12-m Kevlar™ fishing line contained within a plastic housing reel (fly-fishing reel) was fastened to the snap shackle on the pole. The line was marked every meter and unreeled to desired lengths during the survey. Small fluorescent tags attached to the line were used to mark the 2-m wide transect area under the line. A line tender held the line taut above the reef structures and slowly moved the line in an arc around the fixed central stake, allowing time for the surveyors to record their data (Fig 2). Two surveyors swam in concentric circles directly over the line, one recording the number of colonies of each coral species and the other recording the number of colonies of each species that displayed signs of a specific disease. The surveyors counted colonies larger than 10 cm that fell directly below each 2-m segment of the line, providing more than half of their area occurred within the segment. The originating point of the arc was marked with a weighted subsurface buoy to alert the line tender when an entire arc segment had been completed.

During 1997, the surveys were conducted within the entire 10-m radius, in multiple radial increments that enlarged the arc by 2 m for each complete circle. For example, the first arc segment included the 0- to 2-m increment, the second arc segment included the 2- to 4-m segment, and so on until the entire 10-m radius of the arc had been completed, circumscribing a total area of 314m^2. A species area curve was constructed to compare the June 1997 mean percent coral disease (MPCD) for increasing increments of the radial arc areas within the arc transect. Analysis of variance was used to determine whether the MPCD could be estimated using a portion of the arc, instead of the entire 10-m radius. Individual MPCD for each arc segment (0-2, then 2-4, and so on for each segment of 10-m radius) and cumulative MPCD for the cumulative areas of the arc segments (0-2, then 0-4, and so on for the entire 10-m) were used for the analysis, with arc area used as the class variable. This investigation determined that an area of 113m^2, within the 8- to 10-m segment was sufficient for a reliable estimate of total MPCD (see Results). Therefore, during 1998 surveys only the 8- to 10-m segment of the arc radius was used for assessment.



Coral species and coral disease identification

Ten disease conditions affecting 16 species of scleractinian corals and gorgonian sea fans were enumerated (Table 1). Three species of coral contained within the *Montastraea annularis* complex (Weil & Knowlton, 1994) were combined as a single category for data analysis, because discrepancies in identification were noted among some of the surveyors. Two gorgonian species, *Gorgonia flabellum* and *Gorgonia ventalina,* were combined as *Gorgonia* spp. All diseases were scored only for colonies containing active lesions; diseases were not scored if mortality had occurred recently and the cause of

death was not apparent. Signs used to distinguish most coral diseases have been detailed elsewhere (Table 1) (McCarty & Peters, 1998; Santavy & Peters, 1997). Similar conditions described in the literature as patchy necrosis disease (Bruckner & Bruckner, 1997) and white pox (Holden, 1996), might be the same disease. We could not distinguish between the two conditions based on Bruckner & Bruckner's (1997) mention of patchy necrosis or Holden's (1996) mention of white pox; therefore we used the term patchy necrosis disease/white pox to describe the lesions found on *Acropora palmata* colonies that were not white-band disease or predation. We did not distinguish the differences between white plague type 1 and 2, since the primary difference in distinguishing them in the literature is dependent on the rate of progression (Dustan, 1977; Richardson et al., 1998a, 1998b). This could not be determined in our surveys; therefore, we identified these conditions only as white plague in our surveys. Finally, there is some uncertainty in identifying aspergillosis (Kim et al., 1997; Nagelkerken et al., 1997a, 1997b; Smith et al., 1996). For our surveys, this disease was scored if one of the following conditions were met: white fungal-like filaments with active lesions (tissue loss) and major skeletal damage, or white fungal-like filaments with active lesions showing coenenchyme purpling, or white fungal-like filaments with active lesions and purple galls in the vicinity of the diseased area. There are some inconsistencies in the literature concerning the signs of this disease, therefore, one might prefer to refer to the condition simply as sea fan disease.

Quality assurance

A rigorous quality assurance (QA) plan was adopted to quantify surveyor error and minimize data processing errors. The data collection protocols required training to improve identification skills and familiarize participants with the survey procedures, including the completion of standardized data forms. Scientific experts assessed the coral species and diseases in the 1997 surveys. Coral surveyors in 1998 were evaluated by expert coral taxonomists and expert coral pathologists for their ability to identify coral species and to classify coral conditions as either healthy, affected by a specific disease, bleached, or physically damaged. Only those individuals who had successfully passed a test (scoring 90% similarity or greater using the experts as truth) were employed to collect data for the surveys. To evaluate inter-surveyor error, we had multiple surveyors take repeated counts of coral colonies by species and of coral disease types by species. To evaluate intra-surveyor error, we had each surveyor count species and disease types multiple times at a single station. The QA plan included procedures for several levels of data verification, including checks made in the field, duplicate surveys, and independent validation of all electronically entered data.

Data analysis

Data were recorded on standardized data sheets photocopied on Dura/Copy™ (J.L. Darling Corp., Tacoma, WA.) underwater paper. All data were entered into a computerized database using a PerForm Pro™-generated template, exported into MS Excel® worksheets, and used to create SAS data sets. Electronic data quality was confirmed twice by someone other than the original data recorder. The parameter of interest was mean percent coral disease (MPCD) (i.e., number of affected colonies per total number of colonies) per unit area. Data were analyzed using one-way ANOVA for unbalanced design in the 1997 pilot surveys and for balanced design in the 1998 surveys. The assumptions for ANOVA were tested and met, including independence, homogeneity of variances, and normality. The class variables or strata used included geographical areas (Key West, New Grounds, or Dry Tortugas) and reef types (back, fore, or transitional reef). Statistical significance for type I error was designated as α=0.05 level. Tukey's Studentized Range Test (HSD) was used for means separation at the α =0.05 level. A power analysis was performed to determine appropriate type 1 and 2 errors, and statistical power to be employed for future analyses. All analyses for the study were performed using SAS Version 6.12 (Statistical Analysis System Institute Inc., Cary, N.C., 1989-1996).

Appendix II. Priorities for research on coral diseases linked to management objectives in Florida. Developed by Brian Keller and Billy Causey.

1. Direct and indirect causes of coral decline in the Florida Keys, with emphasis on cause and effect. Process-oriented research is required to separate natural variation in community composition from anthropogenic changes. We know that disease contributes to the decline, but we do not know its relative importance and how much forces such as global warming are adding to the problem.

2. Effects of coral bleaching on coral survival. There is a general relationship between severity of bleaching events and coral mortality. However, more information is needed on intra- and interspecific differences in sensitivity to bleaching, habitat-related differences, and synergistic effects, including possible genetic factors.

3. Coral pathology and epizoology with an emphasis on cause and effect. More information is needed on coral immune systems and how they may be compromised by environmental stresses, whether a number of purported coral diseases meet Koch's criteria, how diseases spread, and whether there are ways to prevent coral diseases.

4. Relationships between water quality and the incidence of diseases. There are claims that elevated nutrient concentrations are associated with black-band disease.

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