Coral Reef Diseases in the Atlantic-Caribbean

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1 Introduction

Coral reefs are the jewels of the tropical oceans. They boast the highest diversity of all marine ecosystems, aid in the development and protection of other important, productive coastal marine communities, and have provided millions of people with food, building materials, protection from storms, recreation and social stability over thousands of years, and more recently, income, active pharmacological compounds and other benefits. These communities have been deteriorating rapidly in recent times. The continuous emergence of coral reef diseases and increase in bleaching events caused in part by high water temperatures among other factors underscore the need for intensive assessments of their ecological status and causes and their impact on coral reefs.

In the last few decades, coral reefs around the world have experienced significant declines with changes in composition, structure, and function attributable to one or more natural and anthropogenic interacting factors (Harvell et al. 1999, 2005, 2007; Hoegh-Guldberg 1999; Ostrander et al. 2000; Hayes et al. 2001; Jackson et al. 2001; Gardner et al. 2003; Hughes et al. 2003; Pandolfi et al. 2003; Weil et al. 2003; Willis et al. 2004; Weil 2004; Sutherland et al. 2004; Wilkinson 2006; Rogers and Miller 2006; Hoegh-Guldberg et al. 2007; Lesser et al. 2007; Rogers et al. 2008a, b; Miller et al. 2009; Cróquer and Weil 2009b). The effect of each factor, or combination of factors, varies within regions and across time. A recent report indicated that 32% of zooxanthellate scleractinian corals face an elevated risk of extinction due mainly to bleaching and disease that seem positively and significantly correlated with elevated sea water temperature and further exacerbated by local anthropogenic stressors (Carpenter et al. 2008).

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A significant die-off of acroporids and other corals in the Florida Keys and the Dry Tortugas occurred during severe cold weather in the winter of 1977–1978 (Roberts et al. 1982). Some presumably minor restricted disease outbreaks that occurred in the 1970s in the Florida Keys and the Virgin Islands were followed by two apparently concurrent biotic, wide-geographic epizootic events during the late 1970s and early 1980s. [An epizootic or disease outbreak is defined as "an unexpected increase in disease or mortality in a time or place where it does not normally occur or at a frequency greater than previously observed" (Wobeser 1994; Work et al. 2008a, b; Woodley et al. 2008)]. The white band disease (WBD) outbreak affected *Acropora palmata* and *A. cervicornis*, two of the most abundant and important reef-building corals in the region (Gladfelter 1982), and an unknown pathogen produced the mass-mortality of the black sea urchin *Diadema antillarum*, an important and abundant species in all tropical and subtropical shallow marine habitats of the western Atlantic and Caribbean (Lessios et al. 1984a, b). Coral and sea urchin populations experienced over 90% mortalities over their geographic range, resulting in significant losses in genetic diversity, coral cover, and spatial heterogeneity of coral reefs across the Caribbean.

Almost 30 years after these events, the affected coral and urchin species have not recovered to their former densities and populations structures in reefs off Puerto Rico (Weil et al. 2003, 2005). Furthermore, hurricanes, storms, and two major widespread bleaching events in 1998 and 2005 led to further localized mortalities of surviving acroporids and other major reef-building species (Miller et al. 2006, 2009; Wilkinson and Souter 2008; McClanahan et al. 2009; Cróquer and Weil 2009a).

Disease is considered here as "any impairment to health resulting in physiological dysfunction," involving an interaction between a host, an agent i.e., pathogen, environment, genetics, and the environment (Martin et al. 1987; Wobeser 1994). These three components must interact in a precise way for disease to occur. This definition includes both non-infectious (produced by genetic mutations, malnutrition, and/or environmental factors), and infectious diseases (produced by pathogens). The host is the organism affected by

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the disease (e.g., coral, octocoral), the agent(s) is/are the factor(s) that directly or indirectly cause(s) disease. Infectious agents are capable of causing infection and may be transmissible between hosts (Stedman 1976; Wobeser 2006). The environment is considered to be the third factor of the disease triad and provides the stage where host–agent interactions occur (Wobeser 2006; Work et al. 2008a, b).

In a recent controversial report, Lesser et al. (2007) emphasized the importance of the environmental drivers causing disease outbreaks and questioned the generalized conclusion that diseases of corals are caused by a primary pathogen and are infectious in nature. They suggested that coral diseases are most often a secondary phenomenon caused by opportunistic pathogens after physiological stress produced by changing environmental conditions. Although it is important to understand the role of environmental co-factors, which in some cases could render corals more susceptible to disease, it is important to exercise a balanced approach that would increase our understanding of the interactions among host, agent, and environment (Work et al. 2008b). By definition, "if an organism develops an infectious disease, then there has to have been some breakdown in host defenses to allow pathogens to establish. By this logic, all diseases (e.g., common cold, TB, AIDS) are opportunist. The distinction that needs to be made (and that we can make in other animals but not corals yet) is whether we can measure or quantify the decrease in host response prior to development of disease, and thus make the case that this animal had a quantifiable suppression in immune status before development of disease" (T. Work, personal communication 2010; Work et al. 2008a).

Studying diseases in the marine environment has proven to be challenging. For example, it is difficult to collect samples without contamination and variability in sample collection methods may confound comparative results. Additionally, most marine bacteria, possibly including pathogens, are difficult to culture or are unable to be cultured today, making their identification, laboratory manipulation, and testing of Koch's postulates difficult. Even if a putative pathogen is identified, testing which environmental variable or driver is responsible for its emergence is extremely difficult. Furthermore, recent evidence indicates that bacterial and fungal communities living in association with coral tissues are highly dynamic and different bacteria and fungi may produce similar physiological responses (i.e., disease signs) (Ritchie 2006; Voss et al. 2007; Toledo-Hernandez et al. 2008; Sunagawa et al. 2009).

Corals are "ecological communities" (holobionts), harboring high diversities and abundances of bacteria, zooxanthellae, endolithic algae, fungi, and other boring invertebrates interacting in complex ways (Knowlton and Rohwer 2003; Ritchie 2006; Kimes et al. 2010). Changes in environmental conditions would presumably affect this physiological equilibrium by changing the resident microbial community, which could enhance susceptibility to infectious agents and/or weakening of the host immune system, which could render corals more susceptible to infection, or loss of zooxanthellae (Harvell et al. 1999, 2002, 2007; Ritchie 2006; Mydlarz et al. 2009; Thurber et al. 2009). The compromised-host hypothesis suggests that rising ocean temperatures may increase the number and prevalence of coral diseases by making corals more susceptible to ubiquitous pathogens or by causing shifts in microbial communities making some of them pathogenic (Rosenberg and Ben-Haim 2002).

Few quantitative studies have attempted to relate the emergence, prevalence, and incidence of coral reef diseases with deterioration/change in environmental quality. This requires either large spatial and/or long temporal scales to produce reliable results. Short-term studies, however, have established significant correlations between increasing sea water temperatures and increases in prevalence of white syndrome (WS) and black band disease (BBD) in the Great Barrier Reef (GBR), and prevalence and virulence of Caribbean yellow band disease (YBD) and white patches (Boyett et al. 2007; Bruno et al. 2007; Weil 2008; Harvell et al. 2009; Muller et al. 2008; Weil et al., in press).

Very little is known about the composition and dynamics of the natural microbial communities living in association with most reef organisms (but see Rohwer et al. 2001, 2002; Rosenberg 2004; Ritchie 2006; Rosenberg et al. 2007; Lesser et al. 2007; Sunagawa et al. 2009; Thurber et al. 2009). A clear determination of causality, therefore, is difficult to accomplish. This would require controlled experiments, which is a problem when moving holobiont colonies or fragments from the field into laboratory conditions. Natural changes in composition of bacterial populations may follow the initial infection by a disease-causing agent. Other bacteria could become dominant and pathogenic producing similar signs (Bourne et al. 2007; Voss et al. 2007; Toledo-Hernandez et al. 2008; Sunagawa et al. 2009). The fact that some coral epizootic events occurred over large geographic scales within short periods of time, or simultaneously, suggests a response of already present bacteria (or other pathogens) to similar changes in environmental conditions favoring the infectious disease outbreak. We are currently unable to fully explain the source(s) and sudden emergence of the majority of diseases and/or the outbreaks in coral reef organisms.

Fig. 1 (continued) aspergillosis and red band disease in *G. ventalina* (**j**, **k**), other compromised health conditions in *E. caribbaeorum* (**i**) and *P. nutans* (**l**), and purple spots produced by an unknown protozoan (Labyrinthulomycote) in *G. ventalina* (**m**). Disease conditions in the hydrocoral *M. complanata* (**n**), the vase sponge *X. muta* (**o**), the tubular sponge *C. vaginalis* (**p**) and the zoanthid *P. caribbaeorum* (**q**). Caribbean coralline lethal orange disease (**r**) and crustose coralline white band disease in *N. accretum* (**s**) (Photos E. Weil)

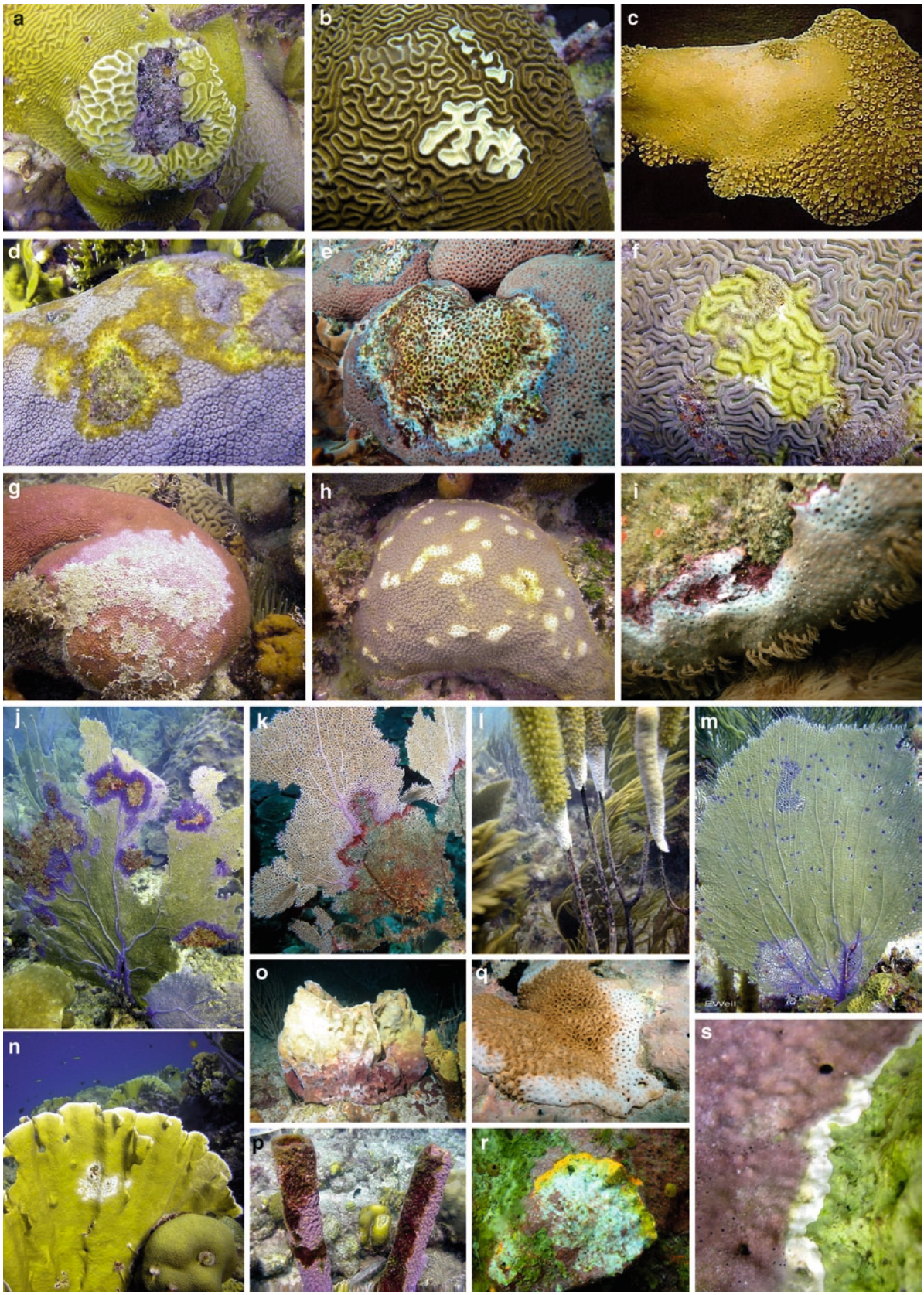

Fig. 1 Photographs of other conditions affecting Caribbean corals and other reef organisms. Growth anomalies in *D. strigosa* (**a**, **b**) (hyperplasia) and *A. palmata* (**c**) (neoplasia) (photo courtesy of E. Peters), pigmentation responses from unknown conditions producing tissue mortality in *M. faveolata* (**d**), *S. siderea* (**e**) and *D. labyrinthiformis* (**f**), white syndromes in *S. siderea* (**g**) and *M. faveolata* (**h**). Diseases in other Cnidarians include

The goal of this chapter is to present a historical perspective of diseases in coral reefs and a summary of the current distribution and status of coral diseases in the Atlantic-Caribbean along with recommendations for future research. There are several other important biological members of the coral reef community such as hydrocorals, sponges, zoanthids, sea urchins, and crustose coralline algae (CCA) that are affected by diseases (see Fig. 1) that will not be discussed in this chapter for lack of space. They will be discussed in a future publication. We have adopted the coral reef disease nomenclature recently updated in Work et al. (2008a), Raymundo et al. (2008), Beeden et al. (2008), and Weil and Hooten (2008b).

2 Historical Perspective

Diseases of coral reef organisms have likely been around for millions of years and may have produced significant population mortalities in the past, but this cannot be confirmed in the fossil record. The emergence of coral reef diseases in the Caribbean in the past few decades appears to be unprecedented in the geological record. Limited paleontological evidence suggests that the white band disease (WBD) outbreak in the late 1970s, which killed acroporid corals throughout their geographic distribution, was unparalleled on a timescale of at least three millennia (Aronson and Precht 2001a, b). Moreover, in recent years, large colonies (many over 500 years old) of the other main reef-building species in the region have succumbed to single virulent diseases such as white plague disease (WPD) and Caribbean yellow band disease (YBD), or the combination of these and bleaching in short periods of time, further indicating that this seems to be a recent and expanding problem (Weil et al. 2006; Bruckner and Hill 2009; Weil et al. in press). However, a hiatus in *A. palmata* accretion occurred 3,000 years ago and again 800 years ago over a wide geographic area. "Understanding the causes of such large-scale community shifts provides both opportunities and challenges with respect to unraveling both natural and anthropogenic change" (Hubbard et al. 2008).

2.1 Black Band Disease

The first scleractinian infectious disease reported in the Caribbean was black band disease (BBD). It was first observed in Belize, Bermuda, and Florida in the early 1970s (Antonius 1973; Garrett and Ducklow 1975), but has since been found throughout the wider Caribbean and the Indo-Pacific (Antonius 1985; Willis et al. 2004; Galloway et al. 2007). It is characterized by a dark bacterial mat of varying composition forming a band separating healthy-looking tissue from the clean skeletal matrix (Table 1, Fig. 2a, b). Little etiological work was done in the early days but information on host range, mortality rates, and depth distribution was provided. It is the better-known coral disease with a significant number of studies expanding pathogeneses, etiology, and epizootiology (Rützler and Santavy 1983; Rützler et al. 1983; Richardson 1997; Aeby and Santavy 2006; Richardson et al. 2007), and including recent debates concerning the variability of the microbial community composition (Voss et al. 2007).

2.2 White Plague Diseases

The first report of a disease outbreak producing significant coral mortalities occurred in Florida in 1975 and was referred to as white plague type I. (WPD-I). It mainly affected the plating coral *Mycetophyllia ferox* (Dustan 1977) leading to fears of its disappearing from some areas in the Florida Keys. During a second WPD-I epizootic event, colonies of *M. ferox* were unaffected whereas massive *Montastraea* were affected (Dustan 1999), suggesting resistant *Mycetophyllia* colonies or a different causative agent. A third and more virulent outbreak of WPD occurred in the Florida Keys in 1995 mainly affecting *Dichocoenia stokesi* and 16 additional species over the next 2 years (Richardson 1998; Richardson et al. 1998a) (Table 1, Fig. 2e, f). In this case, *Aurantimonas coralicida* was isolated from WPD lesions and the disease termed white plague disease type-II (WPD-II) (Richardson et al. 1998b; Miller et al. 2001; Denner et al. 2003). In the late 1990s and early 2000s, a fourth more virulent epizootic was termed WPD-III and affected mostly *Montastraea* spp in Florida, the Virgin Islands, Puerto Rico, and Venezuela (Richardson and Aronson 2002; Weil 2002; Croquer et al. 2005). However, the disease agent was not identified (Richardson and Aronson 2002) and the term WPD-III was discarded.

Another condition called shut down reaction (SDR) in which tissue sloughed off quickly from coral colonies was described in the mid 1970s (Antonius 1977), but was never further studied and there have been no reports of this condition in the region in the last 2 decades.

2.3 White Band Disease and Diadema

Two years after the initial white plague epizootic, an outbreak of a disease with similar signs but affecting only acroporids called white band disease type I (WBD-I) devastated high proportions of *A. palmata* and *A. cervicornis* (Fig. 2c, d) pop-

Table 1 Common coral reef diseases in the western Atlantic and acronym (ACR), year reported/observed (year), pathogen/agent (P/A) identified = Y, No = N, number of taxa showing disease signs in corals (CO), octocorals (OC), hydrocorals (HY), sponges (SP), zoanthids (ZO) and crustose coralline algae (CCA) (number in parenthesis are Brasilian spp), depth distribution (DE), average community prevalence (PR), tissue mortality rate (TM), and their geographic distribution (GD) (WA = western Atlantic, WC = wider Caribbean, VI = Virgin Islands, FL = Florida, BE = Bermuda, CA = Caribbean, BA = Bahamas. ME = Mexico, PR = Puerto Rico), Updated from Weil et al. (2006) and other sources

Disease	ACR	Year	P/A	CO	OC	HY	ZO	SP	CCA	DE		TM	GD
										(m)	PR (%)	(mm/day)	
Bleaching	BL	1911	N	62	29	5	2	8		0–100	.2–85	?	WA
Growth anomalies	GA	1965	N	10	8	1				0–25	–	–	WC
Black band disease	BBD*	1973	Y	19(4)	6					0–25	.3–6	3–10	WA
White band disease-I	WBD-I	1977	N	2						0–10	0.1	?	VI,WC?
White plague disease-I	WPD-I	1977	N	12						10–21	3.6	3.1	FL
Shut Down reaction	SDR	1977	N	6						5–12	–	–	FL
White band disease-II	WBD*	1982	Y	3						1–25	.1–25	3–30	WC not BE
Red band disease	RBD	1984	Y	13(1)	5					2–20	–	1	WA
White patch disease[1]	WPA*	1992	Y	1						0–5	.002	15	CA,FL,BA
Caribbean yellow band [a]	YBD *	1994	Y	11						3–20	1–24	0.1–0.4	WC
White plague disease-II	WPD*	1995	Y	41(5)						3–30	.9–18	3–30	WA
Aspergillosis	ASP*	1996	Y		9(1)					1–25	1.9	.1–2.5	WA
Dark spots disease	DSD	2001	N	11(1)						1–25	1.1	–	WA
Crustose-Coralline white b.	CCWB	2004	N						3	1–20	1–6	.1–2	WC[a]
Caribbean white syndromes[2]	CWS	2004	N	15		2	1	3		2–25	–	–	WC[a]
Caribbean ciliate infection	CCI	2006	Y	21						2–25		–	WC[a]
Sea fan purple spots[3]	SFPS	2008	Y		1					2–18	–	–	ME,FL,PR
Coralline lethal orange disea.	CCLOD	2008	N						1	20	–	–	PR,CY,ME
Other coral health conditions[4]	CCH	–		15	8					1–25	–	–	WA
Other octocoral health condi.[4]	OCH	–			8					3–20	–	–	WA

* = Koch's postulates fulfilled. 1 = White patch disease is also termed white pox and patchy necrosis, 2 = White syndromes include several patterns of tissue loss exposing bands, stripes, blotches, or irregular shapes of clean skeleton (different from the other "white" diseases) with very low prevalence. 3 = PS produced by an unknown protozoan (Labyrinthulomycote). 4 = Other coral and octocorals health conditions include unhealthy-looking tissues with some degree of mortality, low prevalence, and limited geographic distribution with no pathological or etiological information. a = Including Flower Gardens, north Gulf of Mexico. Western Atlantic distribution includes the wider Caribbean and Brazil. Bleaching-affected species from Brazil have not being included in this list

Fig. 2 Photographs of the most common diseases in Caribbean corals. Active black band disease in *M. faveolata* (**a**) and *D. strigosa* (**b**), white band disease in *A. palmata* (**c**) and *A. cervicornis* (**d**), fast moving white plague disease in *D strigosa* (**e**) and *D. labyrinthiformis* (**f**), two different

ulations throughout their geographic range (Aronson and Precht 2001b). A different pattern or phase of this disease in *A. cervicornis* was described in the late 1990s and was named white band disease type II (WBD-II) (Ritchie and Smith 1998). It differed from WBD-I in having a bleaching band leading the necrotic edge of living tissue. These signs have only been observed in *A. cervicornis*, and it is not clear if these two patterns were caused by different pathogens, if the disease is expressed differently in the different species, or if the two etiologies represent different phases of the same syndrome (Weil 2004; Bythell et al. 2004).

Mortality of the surviving colonies/populations of acroporids have continued over the years due to recurrent WBD events, hurricanes and storms, bleaching, predation, and local environmental deterioration (sedimentation, turbidity, untreated sewer outflow, etc.) (Bruckner 2003; Weil et al. 2002, 2003; Wilkinson and Souter 2008; McClanahan et al. 2009), which together with the slow recovery of populations, led to these two corals being listed as threatened under the US Endangered Species Act (Hogarth 2006).

Almost concurrently with the WBD outbreak, although occurring over a shorter time span, a widespread and highly virulent infectious disease wiped out up to 99% of the populations of the black sea urchin *D. antillarum* throughout the wider Caribbean, including Bermuda (Lessios et al. 1984b; Lessios 1988). This urchin was at the time a keystone species regulating algae and coral community structure (Carpenter 1981, 1985, 1990a; Hughes et al. 1987). The causes of these events were never determined (but see Peters et al. (1983) and Rosenberg and Kushmaro, in this book). The consequence of these two epizootics was a significant change in the structure and morphology of most shallow-water coral reef communities throughout the wider Caribbean. These two outbreaks followed increasing water temperatures during an intense El Niño event, which produced limited bleaching in 1983.

2.4 White Patches and Octocoral Mortalities

Other localized invertebrate mass mortalities and disease outbreaks reported during the 1980s included: a die-off of the sea-fan *Gorgonia flabellum* in Panamá (Guzmán and Cortés 1984), and an outbreak of a thin red cyanobacteria mat in corals and octocorals termed red band disease (RBD) in Florida reefs (Rützler et al. 1983), later redescribed by Santavy and Peters (1997) and Richardson (1998) (Fig. 1k). The putative pathogens were not identified.

Several new coral and octocoral diseases were reported during the 1990s with more frequent and virulent epizootic events. White patches of clean skeletal tissue were observed in *A. palmata* (Porter and Meier 1992) which were clearly different from WBD signs, and were termed patchy necrosis (Bruckner and Bruckner 1997) and later, white pox (Rodriguez-Martinez et al. 2001; Patterson et al. 2002) (Fig. 2m). An early photograph from the USVI suggests this disease or a similar one could have been affecting *A. palmata* in the 1970s (Rogers et al. 2005). Several outbreaks of diseases with similar signs were observed in Florida, Puerto Rico, USVI, Mexico, and elsewhere in the late 1990s and early 2000s (Rodriguez-Martinez et al. 2001; Weil and Ruiz 2003; Rogers et al. 2008a) (Fig. 1h). All these terms and disease signs have now been pooled as white patch disease (Raymundo et al. 2008)

2.5 Dark Spots Disease

Dark spots disease (DSD) was first documented in the early 1990s in the Islas del Rosario archipelago, Colombia, as a type of bleaching that affected ca. 16% of *Montastraea annularis* colonies. It was called "Medallones Mostaza" ("mustard rings") (Solano et al. 1993). In 1994, similar signs were observed in other islands off Colombia mainly affecting *M. annularis*, *Siderastrea siderea*, and *Stephanocoenia intersepta*, and it was called "enfermedad de los lunares oscuros" (Diaz et al. 1995) or dark spots disease (DSD) (Figs. 2g, h). Dark spot lesions were characterized as "small, round, dark areas that apparently grow in size over time, some of which can be associated with a depression of the coral surface and others expand into a dark ring surrounding dead coral" (Garzón-Ferreira and Gil 1998). Other names characterizing different manifestations of the disease include "Dark Spots type II" in *S. intersepta*, *Colpophyllia natans*, and *Montastraea cavernosa*, and "Dark Bands" in *M. annularis*, *M. faveolata*, *S. siderea*, and *C. natans* (Weil 2004; Weil et al. 2006) (Fig. 2h). These conditions were all pooled as DSD (Raymundo et al. 2008; Weil and Hooten 2008); however, they could represent different diseases since their etiologies have not been resolved. Dark spots disease has now been found throughout the Caribbean basin (Cervino et al. 2001; Weil et al. 2002; Weil and Croquer 2009; Cróquer and Weil 2009a).

Fig. 2 (continued) etiologies of dark spots disease in *S. siderea* (**g**) and *S. intersepta* (**h**), bleached colonies of *M. faveolata* and *C. natans* (**i**), Caribbean yellow band disease in *M. franksi* (**j**) and *M. faveolata* (**k**), white patches in *A. palmata* (**l**, **m**), and Caribbean ciliate infections in *A. tenuifolia* (**n**) and *D. labyrinthiformis* (**o**) (Photos E. Weil)

Sixteen important scleractinian species have been reported to show signs corresponding to those characteristic of the disease (Table 1), and the disease appears less prevalent in the more northern portions of the Caribbean (Weil et al. 2002; Gil-Agudelo et al. 2004; Cróquer and Weil 2009a). Recently, a disease with lesions resembling DSD was described for Brazil where it was affecting *Siderastrea* sp. (Francini-Filho et al. 2008). In the Indo-Pacific, DSD has been documented in *Pavona varians* and *P. maldivensis* from Kahoolawe, Hawaii and in *P. varians*, *Psammocora nierstrazi* and *Montipora* sp. from Tutuila, American Samoa (Work et al. 2008c).

2.6 Caribbean Yellow Band Disease

Caribbean yellow band disease (YBD) (Fig. 1k) was first reported in the Florida Keys in 1997 by C. Quirolo in *Montastraea* colonies (Santavy and Peters 1997); however, Brown and Ogden (1993) published a photo of a large colony of *M. faveolata* from the Florida Keys with clear signs of YBD in a National Geographic article about bleaching, indicating that the disease could have been around in the 1980s. As with other diseases, different terms have been used for this disease (i.e., yellow blotch disease [Santavy et al. 1999], yellow band disease [Green and Bruckner 2000], and yellow blotch syndrome [Weil 2004]). Here we use Caribbean yellow band disease (YBD) following the original name and the geographic location to differentiate it from a similar syndrome described in the Red Sea with the same name (Korrubel and Riegl 1998). Signs of YBD were observed throughout the Caribbean and north to Bermuda in 1999 (Weil et al. 2002), and the disease is now widely distributed. Outbreaks of YBD were observed in Panamá in 1996 (Santavy et al. 1999), in the Netherland Antilles and Puerto Rico in 1997 (Cervino and Smith 1997; Bruckner and Bruckner 2006) and in Grenada, Mexico, Bermuda, and Puerto Rico between 2005 and 2009 (Cróquer and Weil 2009a, Weil et al., in press; Weil, unpublished data). This disease has become one of the major causes of tissue and colony mortality in three species of *Montastraea* (Fig. 2j, k), the most important reef-building genus in the region (Weil et al. 2006; Bruckner and Hill 2009; Weil et al., in press).

2.7 Caribbean Ciliate Infection

In 2004, ten coral species were observed with dead areas preceded by a dark band different from BBD in reefs off Venezuela. This band was formed by dense populations of a ciliate protozoan (*Halofoliculina* sp.) (Cróquer et al. 2006a) (Fig. 2n, o). Further surveys found the same ciliate infecting up to 22 coral species throughout the Caribbean, and the condition was termed Caribbean ciliate infection (Cróquer et al. 2006b; Weil et al. 2006; Weil and Hooten 2008; Weil and Croquer 2009; Cróquer and Weil 2009a; Weil et al., in press).

2.8 Aspergillosis and Purple Spots

A widespread epizootic of a fungal infection producing wide areas of tissue mortality surrounded by purple pigmentation (an immune response by the host) in the early 1990s, affected thousands of colonies of the sea fan *Gorgonia ventalina* (Fig. 1j) in many reef localities (Nagelkerken et al. 2007a, b). It was suggested that this was the same problem responsible for widespread *Gorgonia* mortalities in 1984 in Central America (Guzmán and Cortés 1984; Garzón-Ferreira and Zea 1992). The putative pathogen was later identified as the common terrestrial fungus *Aspergillus sydowii* and the disease was called aspergillosis (ASP) (Smith et al. 1996). At least eight other abundant octocoral species throughout the wider Caribbean have been reported to be affected by ASP (Weil 2001, 2002; Harvell et al. 2001; Smith and Weil 2004; Weil et al. 2006). Two independent reports have confirmed the impact of aspergillosis on the reproductive output of *G. ventalina* colonies, essentially reducing fitness and potential population recovery (Petes et al. 2003; Flynn 2008; Flynn and Weil 2008). Other disease signs have been observed in other octocoral species such as the common and abundant encrusting *Briareum asbestinum* and *Erythropodium caribaeorum*, which have been affected throughout their geographic range by "necrotic-like" lesions, which progress rapidly, sometimes killing large areas in a short time (Harvell et al. 1999; Weil 2004; Weil et al. 2006) (Fig. 1i).

In the last 4–5 years, colonies of the sea fan *G. ventalina* have been observed with small purple spots in Mexico and Florida (Harvell et al., 2008), and more recently, in Puerto Rico (Weil and Hooten 2008) (Fig. 1m). These purple spots are caused by a protozoan (Labyrinthulomycote) that infects colonies mostly during the summer (C.D. Harvell, personal communication). Prevalence has been increasing in several reefs off the southwest coast of Puerto Rico in the last few years (Weil, unpublished data 2009).

2.9 Other Diseases

Several other signs of presumed diseases such as white spots, white bands, white stripes and rings, pigmentation responses, dark bands, tissue loss and tissue "necrosis,"

that are usually found in a few colonies of a wide range of coral and octocoral species have been observed throughout the region in recent years (Fig. 1d, e, f, g and h). Furthermore, many other important members of the coral reef community such as hydrocorals, sponges, zoanthids, and other important calcifying organisms have been affected by diseases in the Caribbean for some time (Fig. 1n–s). In the late 1990s and early 2000s, the common crustose coralline alga (CCA) *Neogoniolithon accretum* and at least two other species were observed with an advancing, thin, white band separating healthy-looking tissues from dead areas (Weil 2004; Weil and Hooten 2008). The condition was termed crustose coralline white syndrome (CCWB) (Fig. 1s) and was found in high prevalence in deep reef habitats (18–23 m) of Puerto Rico and Grenada (Ballantine et al. 2005; Weil et al. in press; Weil, unpublished data 2007). Since then, signs of the condition have been observed throughout the Caribbean and in many locations in the Indo-Pacific and Indian Ocean (Weil, unpublished data 2008). More recently, signs similar to coralline orange lethal disease (CLOD) described for the Pacific (Littler and Littler 1995) have been observed on deep (20 m) CCA in Puerto Rico, the Cayman Islands, and Mexico and the condition termed Caribbean CLOD (CCLOD) (Weil et al., in press) (Fig. 1r).

3 Current Status of Coral Diseases

The Caribbean has been dubbed a "disease hot spot" due to the fast emergence and high virulence of coral reef diseases, their widespread geographic distribution, wide host ranges, and frequent epizootic events with significant coral mortalities. The Wider Caribbean includes the Gulf of Mexico, Florida, the Bahamas, and Bermuda and only about 8% of the coral reef area worldwide (Spalding and Greenfeld 1997), yet over 60% of all disease reports up to 2000 came from this region (Green and Bruckner 2000).

Local environmental and anthropogenic stresses in combination with global warming trends have been proposed as factors that could affect species susceptibility/resistance to pathogens, as well as enhance bacterial growth and virulence favoring local disease outbreaks, which can then be dispersed by the rapid currents in the basin (Peters 1997; Epstein et al. 1998; Goreau et al. 1998; Richardson 1998; Richardson and Aronson 2002; Weil 2004; Weil et al. 2006; Harvell et al. 2007).

Besides the Caribbean, the south coast of Brazil is the only other area with significant coral reef formations in the Western Atlantic, and until recently, no coral diseases had been reported for this region. Besides distance, two major dispersion barriers, the outflows of the Amazon and Orinoco Rivers, separate Caribbean and Brazilian coral reefs, which could also be effective barriers to dispersing pathogens. Nevertheless, several diseases with similar signs to those in the Caribbean were recently described for this region (Acosta 2001; Francini-Filho et al. 2008).

At least eighteen disease conditions affecting corals and other important reef organisms have been described for the Wider Caribbean (Table 1, Figs. 1 and 2). Most do not have defined pathologies nor have they been well characterized (Bythell et al. 2004; Weil et al. 2006). Of these, ten diseases affecting corals show consistent signs that allow their recurrent identification, black band disease (BBD), white plague disease (WPD), Caribbean yellow band disease (YBD), white band disease (WBD), white patches (WPA) (formerly called patchy necrosis, *Acropora* serriatosis, and white pox), dark spots disease (DSD), red band disease (RBD), Caribbean ciliate infection (CCI), growth anomalies (GA), and bleaching (BL). Other "white" diffuse/inconsistent signs (bands, spots, stripes, etc.) producing minor tissue loss affect several corals and have been grouped as Caribbean white syndromes (CWS). Unhealthy-looking conditions such as dark areas, bands, pigmentation responses, etc. have been pooled into "Other compromised health" conditions (OCH) until their etiologies and pathologies are clarified to avoid further confusion (Table 1, Fig. 1). Most "white" diseases are characterized by the recently exposed white skeleton after the tissue died, so identification is based on the appearance of the skeleton in contrast with the edge of live tissue and not on any pathology of the tissues (Bythell et al. 2004; Lesser et al. 2007; Work et al. 2008a).

Four diseases with consistent signs [aspergillosis (ASP), red band disease (RBD), growth anomalies (GA), and sea fan purple spots (PS)], and several other conditions affect common and abundant octocoral species (Table 1, Fig. 1). Little histopathological work has been done other than in corals and a few octocorals (Weil et al. 2006), a significant gap in our current approaches to the study of cnidarians diseases. Descriptions of the common Caribbean and Indo-Pacific coral-octocoral diseases can be found in Richardson (1998), Rosenberg and Loya (2004), Raymundo et al. (2008), Beeden et al. 2008; and Weil and Hooten (2008).

Compared to the Caribbean, only a few coral diseases and diseases in other organisms have been reported for the Indo-Pacific (Littler and Littler 1995; Korrubel and Riegl 1998; Willis et al. 2004; Galloway et al. 2007) and the Red Sea (Loya 2004). The first reported coral disease for the Indo-Pacific was black band disease affecting two massive faviid species in the Philippines and later, seven other species in the Red Sea (Antonius 1985). The disease showed similar signs to the Caribbean BBD, but the bacterial mat seemed to have a different species composition (see later). Besides BBD, brown band disease (BrB), skeletal eroding band (SEB), ulcerative white spots (UWS), atramentous

necrosis (AtN), growth anomalies (GA), and white syndromes (WS) are the most commonly found diseases with low and variable prevalence and limited geographic distribution (Willis et al. 2004; Raymundo et al. 2008; Galloway et al. 2007). The number, distribution, and prevalence of diseases has been increasing across the Indo-Pacific and the Red Sea as more research is being done, with several reports of epizootic events across the region (Green and Bruckner 2000; Willis et al. 2004; Rosenberg and Loya 2004; Weil and Jordán-Dahlgren 2005; Page and Willis 2006; Raymundo et al. 2003, 2008; Work and Aeby 2006; Aeby 2006a, b; Galloway et al. 2007; McClanahan et al. 2009).

3.1 Pathogenesis

Descriptions of many coral diseases are limited and often confounded by the lack of clear diagnostic criteria and the absence of pathological observations, so that similar disease signs may reflect multiple conditions in one or more coral species (Bythell et al. 2004; Weil 2004; Work and Aeby 2006; Raymundo et al. 2008; Work et al. 2008a). There is evidence that different pathogenic bacteria and fungi can produce similar signs in the same and/or in different species (Toledo-Hernandez et al. 2008; Sunagawa et al. 2009).

Because the BBD microbial mat consistently contained dominant populations of the same microorganisms, Carlton and Richardson (1995) proposed that it was caused by a microbial consortium instead of a single pathogen. The three mayor players were a cyanobacterium (*Phormidium corallyticum*), a sulfide-oxidizing bacterium (*Beggiatoa* sp.), and a sulfate-reducing bacterium (*Desulfovibrio* sp.). The dark coloration during the day is provided by the red cyanobacterial pigment phycoerythrin. Richardson (1997) demonstrated that BBD sulfate-reducing bacteria are functionally specific to BBD pathogenicity, and suggested that they may be species-specific. Recent molecular studies, however, suggest that the primary pathogen may be a nonphotosynthetic, eubacterial heterotroph (Cooney et al. 2002; Frias-Lopez et al. 2002). Furthermore, these results also showed that *P. corallyticum*, originally identified as the cyanobacterial component of BBD (Rützler and Santavy 1983; Taylor 1983) may not be the cyanobacterium associated with BBD. Recent studies indicated that the BBD mat is dominated by an unidentified cyanobacterium most closely related to the genus *Oscillatoria* (Cooney et al. 2002; Frias-Lopez et al. 2003). At least three different taxa of cyanobacteria associated with BBD were identified by Frias-Lopez et al. (2003), who showed that they vary between the Caribbean and Indo-Pacific.

Furthermore, differences in composition of the bacterial community have been reported for BBD affecting corals in Florida, the Bahamas, and the US Virgin Islands (Voss et al. 2007). The mat composition seems to be variable spatially and/or temporally, or the components may have been misidentified originally (Voss et al. 2007; Sekar et al. 2008). Most recently, a single cyanobacteria ribotype was found to be associated with both red band disease (RBD) and BBD in corals from Palau, having a 99% sequence identity with a Caribbean strain (Sussman et al. 2006). Further research is needed to clarify whether RBD and BBD are the same.

Denner et al. (2003) identified the bacterium *A. coralicida* as the putative pathogen of WPD-II in the coral *D. stokesi* in the Florida Keys. Another 40 coral species have been reported to be susceptible to WPD (Weil 2004; Sutherland et al. 2004) because they showed signs similar to those described for *D. stokesi* colonies infected with *A. coralicida* (Richardson et al. 1998a). However, *A. coralicida* has not been consistently found in corals with signs of WPD, and probes developed and used to identify the pathogen are insufficiently specific to consistently incriminate this bacterium (Bythell et al. 2004; Polson et al. 2009). Even though *A. coralicida* has been found in a few other coral species, Koch's postulates have only been verified for *D. stokesi* (Richardson et al. 1998b; Denner et al. 2003; Pantos et al. 2003). Recent analyses of several diseased tissue samples from *Montastraea faveolata* colonies with typical WPD-II signs failed to find *A. coralicida* (Sunagawa et al. 2009), and no other experimental data show that any of the other species reported with WPD signs have been actually infected by *A. coralicida.* Therefore, WPD signs in these colonies and other species of corals might be caused by a different agent.

More than 20 years after the WBD epizootic, most populations of acroporids have not recovered and the disease agent associated with this epizootic has not been identified. A potential pathogen named *Vibrio charchariae* was identified but Koch's postulates were never fulfilled until recently. Results from controlled isolation/inoculation experiments in Puerto Rico showed that the potential cause of WBD type II is possibly a *Vibrio* species very close to *Vibrio harveyi*, a synonym of *V. charchariae* (Gil-Agudelo et al. 2006). This study also reported that other *Vibrio* species tested produced similar WBD signs, but their virulence was lower than *V. harveyi*.

The putative pathogen associated with white pox (WPX) signs on *A. palmata* in the Florida Keys was identified as the bacterium *Serratia marcescens*, a common gut bacterium in sheep, other mammals, and fish (Patterson et al. 2002). The disease was then called *Acropora* serratiosis; however, all conditions with similar signs (white patches, patchy "necrosis") in *A. palmata* have been recently pooled as "white patches" (WPA) (Raymundo et al. 2008; Weil and Hooten 2008). The disease agent in WPA-infected colonies outside the Florida Keys has never been identified, and preliminary

results from a few samples from St. John did not show any correlation between signs of "white pox" and presence of *S. marcescens* (Polson et al. 2009).

The cause of dark spots disease is still unknown. Gil-Agudelo et al. (2004) examined bacterial flora of mucus in corals affected with DSD and found that the corals were infected with *Vibrio charchariae* whereas this bacterium was absent in normal corals. Experimental infections of corals in the field using this bacterium failed to replicate DSD signs. Corals maintained in aquaria incidentally developed DSD, and treatment with antibiotics led to further tissue loss, thereby arguing against a bacterial etiology for DSD (Gil-Agudelo et al. 2004). In Florida, fungi have been associated with *S. siderea* affected with DSD (Galloway et al. 2007). Chemical analyses of colonies of *S. siderea* resistant and susceptible to DSD in Puerto Rico suggested that phenotypic plasticity in antimicrobial activity may affect microbial infection and survival in the host colonies (Gotchfeld et al. 2006).

Recent experimental evidence suggests that a particular combination of four *Vibrio* species infects and kills zooxanthellae in the coral endoderm producing the characteristic signs of yellow band disease in both Caribbean and Indo-Pacific corals (Cervino et al. 2004a, b, 2008). However, the mechanisms by which the zooxanthellae and the coral tissue are killed are unclear. The onset of infection seemed to be temperature-dependent (Weil et al. 2009b), and prevalence increased under high-nutrient conditions (Bruno et al. 2003) and high water temperatures (Harvell et al. 2009; Weil et al., in press).

Further evidence indicates that the dynamics of bacterial communities in corals are more complicated and more responsive to changes in environmental conditions than previously thought (Ritchie 2006; Gil-Agudelo et al. 2006, 2007; Voss et al. 2007; Toledo-Hernandez et al. 2008). Similar to other coral diseases, recent findings have revealed that *A. sydowii*, the pathogen causing aspergillosis in sea fans and other octocorals (Smith et al. 1996; Smith and Weil 2004) has been found in sea fans without disease signs. Furthermore, ASP signs could be produced by other *Aspergillus* species and fungi in other groups (Toledo-Hernandez et al. 2008).

Changes in the composition and dynamics of the bacterial community after environmental or biological changes in the coral host could be related to different bacteria producing similar disease signs over time, sometimes indicating (although not conclusively proving) a potential development of resistance to the initial pathogenic agent (Reshef et al. 2006). In a recent study of *Montastraea* corals showing typical white plague signs in Puerto Rico, the primary WPD pathogen *A. coralicida* was not found after screening mucus and tissue samples (Sunagawa et al. 2009). Similar results were reported for the fungal disease ASP (Toledo-Hernandez et al. 2008) and for bacterial bleaching in *O. patagonica* in the Mediterranean (Ainsworth et al. 2008). A suite of other fungal species can produce similar aspergillosis signs in sea fans (Smith and Weil 2004; Toledo-Hernandez et al. 2008). These results emphasize the need to examine tissue microscopically in attempts to identify potential causative agents, to follow up with appropriate laboratory confirmation, and to be cautious about naming and describing diseases without a clear pathogenesis.

Many cnidarian diseases have yet to be characterized. Their etiologies have not been properly described and their putative pathogens have not been identified (Ritchie et al. 2001; Weil et al. 2006; Work et al. 2008a). As researchers become more familiar with disease signs and more pathological studies are carried out, the number of described diseases affecting corals and other reef organisms could grow. Koch's postulates have only been verified for five diseases and in most cases, for only one species in each condition (Table 1); so there is ample room for new pathologies and changes in our understanding of these diseases.

3.2 Geographic Distribution

Most coral diseases in the Caribbean have spread throughout the region (Table 1; Fig. 3). White plague, YBD, DSD, ASP, BBD, CCI, GA, and bleaching show the widest geographic distribution, from Bermuda to Trinidad and Tobago, the northern coast of Venezuela and Colombia, Central America, and the southern region of the Gulf of Mexico (Weil 2004; Weil and Croquer 2009). With the exception of the white band disease outbreak and the mass mortality of *D. antillarum,* no correlations between dispersion patterns of most of the recent epizootic events (ASP, WPD, YBD, etc.) with current patterns in the region have been reported. There is some evidence, however, that WBD spread against the predominant current in St. Croix in the early 1980s (Gladfelter 1982). A dispersion pattern following local and/or regional water currents is expected for new putative, infectious agents introduced or "activated" in one particular location, as was the case of the agent killing the sea urchins in the early 1980s (Lessios et al. 1984a,b Lessios 1988; Carpenter 1990a, b).

Most reefs in the northern Gulf of Mexico are far from the US mainland and deeper than 22 m where water temperatures are cooler and light conditions are reduced, conditions that might limit the development of diseases. A short-lived outbreak of a white syndrome was observed in 2005 in the deep coral reef areas of the Flower Garden Banks (Hickerson and Schmahl 2006). Other conditions such as growth anomalies, "mottling syndrome" and "pale ring syndrome" were reported by Borneman and Wellington (2005), and more

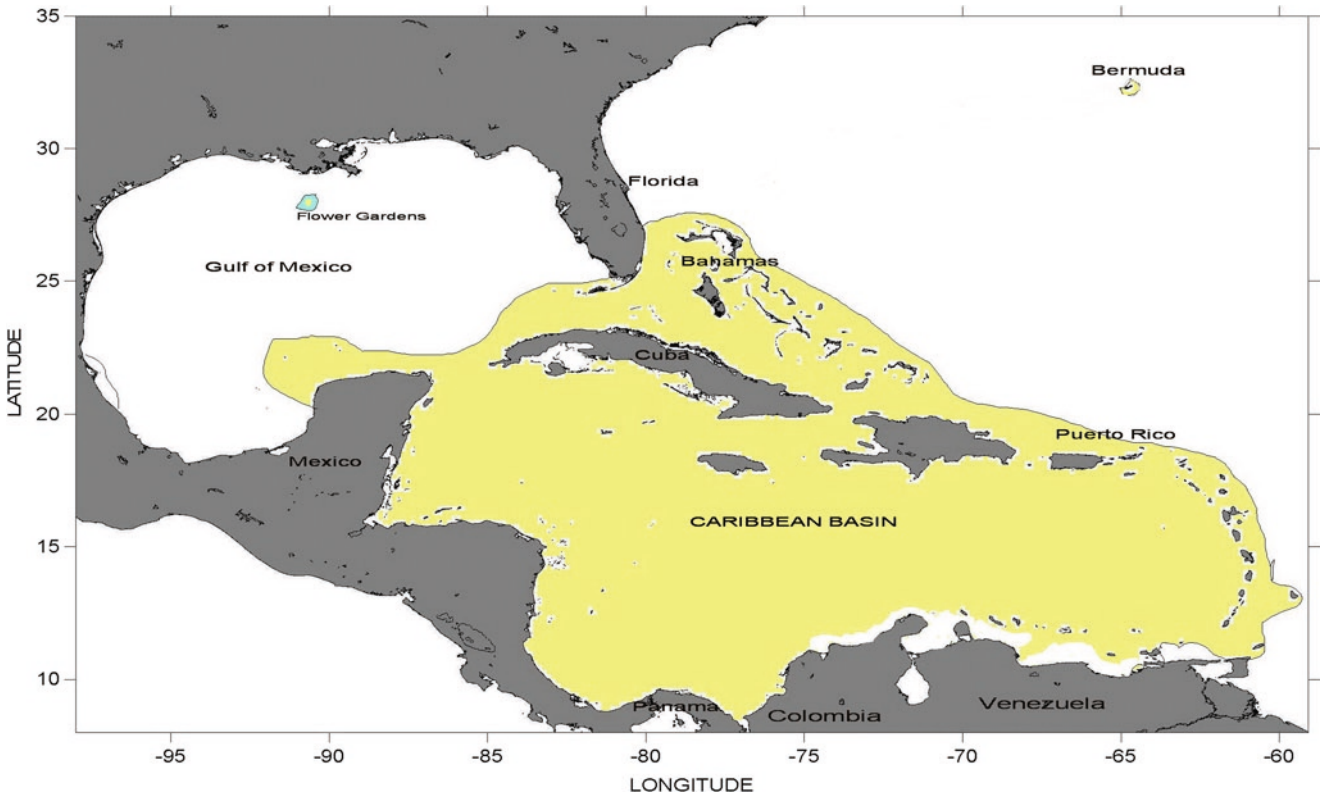

Fig. 3 Geographic distribution of the most common coral and octocoral diseases in the wider Caribbean: BBD, WPD, YBD, DSD, GAN, CCI, ASP, white syndromes and other compromise health conditions. Crustose coralline white band is also distributed throughout the wider Caribbean. A virulent white syndrome was observed in 2005 in the Flower Gardens in the north area of the Gulf of Mexico and bleaching has affected all reefs across the region

recently, Caribbean ciliate infections and GA were observed during disease surveys in the two major banks (Zimmer et al. 2009). Caribbean ciliate infection (CCI) was first observed in Venezuela in 2005 (Cróquer et al. 2006a, b), but recent surveys have reported this condition in corals in at least six other countries in the region (Bermuda, Puerto Rico, Grenada, Caymans, Mexico, and Panamá) (Cróquer and Weil 2009a; Weil and Croquer, unpublished data 2009), but this is the deepest report so far.

Until recently, only one disease, affecting zoanthids, was reported from Brazil (Acosta 2001). The first signs of scleractinian coral and octocoral diseases were observed in 2005 in the Abrolhos Bank, the largest reef system in Brazil. Conditions with signs similar to WPD, BBD, RBD, ASP, GA, and octocoral compromised health conditions ("tissue necrosis") were recently reported from this area (Francini-Filho et al. 2008). From 2005 to 2007, the distribution of these diseases has widened and their prevalence and virulence have increased producing significant coral and octocoral mortalities in the Abrolhos Bank. Based on estimates of disease prevalence and progression rates, as well as on the growth rates of a major reef-building coral species (the Brazilian-endemic *Mussismilia braziliensis*), it is predicted that eastern Brazilian reefs will suffer a massive coral cover decline in the next 50 years, and that *M. braziliensis* will be nearly extinct in less than a century if the current rate of disease mortality continues (Francini-Filho et al. 2008).

Limited connectivity between the Caribbean and the Brazilian reefs suggests that either these diseases are produced by different agents, possibly triggered by similar environmental changes (increase in water temperatures), or they have similar etiologies to their Caribbean counterparts. If pathogens are different, this shows the limited responses (signs) cnidarians can develop when affected by infectious diseases and other agents, and the importance of identifying putative pathogens.

3.3 Depth Distribution

Caribbean wide surveys indicate that WPD, YBD, DSD, and ASP have the widest depth distribution, from 1 to 25 m (Weil 2004; Cróquer and Weil 2009a, b; Weil et al., in press). If the pathogens are species-specific, disease infections would be limited to the depth distribution of the host species. Most of

the species affected by the major diseases (*M. faveolata, M. franksi, M. cavernosa, M. ferox, Agaricia lamarcki*, etc.) have a wide depth distribution, some down to 90 m. However, some diseases affecting these species have limited depth distribution. White plague disease has been more prevalent in deeper habitats (10–25 m) in Puerto Rico (Weil et al., in press); however, recently, a colony of *M. ferox* was observed with signs of WPD at 50 m off the southwest coast of Puerto Rico, the deepest record so far (H. Ruiz, personal communication). YBD has only recently been observed below 20 m in the Caymans and Puerto Rico (Weil, unpublished), which could be related to different zooxanthellae composition in deeper corals. Bleaching has the deepest distribution of all diseases with pale or white corals (of a few different species) observed down to 100 m in some reefs in the Caymans in 2009 (McCoy C, personal communication 2009). Aspergillosis has the widest depth distribution among octocoral diseases, with sea fan colonies showing signs of this condition from 1 to 25 m (Jolles et al. 2002; Kim and Harvell 2002; Flynn and Weil 2008; Flynn and Weil, in press).

3.4 Prevalence, Incidence, and Virulence

Prevalence is the proportion of infected colonies in a population or a community. It is expressed by absence/presence per individual and usually does not give any indication of the severity (virulence) of the disease, which could include the number (and size) of the lesions that are present, the rate of tissue mortality, the proportion of the colony that is affected, or the rate of spread of the disease in the population (Work et al. 2008a, b; Weil et al. 2008). Most surveys are done yearly or only during "outbreaks" and, prevalence can vary greatly even over short periods of time. Prevalence of white pox disease (= white patches) on *A. palmata* colonies off St. John ranged from 0% to 52% (Rogers et al. 2008b). Disease incidence is a rate expressing the number of newly infected colonies over time. It requires temporal monitoring of the same reef area with mapping, tagging, and photographing of colonies along the sampled area (Weil et al. 2008; Work et al. 2008b).

Average disease prevalence at the coral community level for major coral diseases in the Caribbean remains low (<6%) and has not changed significantly in the last 10 years (Weil et al. 2002; Weil and Croquer 2009). However, in some localities, prevalence of some chronic diseases within populations could be much higher and, even low levels of disease over long periods of time can produce significant mortalities in reef communities. Frequent monitoring is needed to address the spatial and temporal variability in prevalence and virulence (number of lesions and rate of disease advance) and to assess disease incidence (new cases of infected colonies over time) at population and/or community levels and their cumulative effects.

One-time surveys are limited in that they only reflect the disease status at a particular time. Data for different reefs generated with different methods could be difficult to compare, especially if the areal extent of the surveys are not the same and the data are collected by different people without standardization of the disease identification. Similarly, data generated with the same methods but taken in different seasons and/or different years could also generate problems of interpretation. Prevalence will go down when the disease has run its course, and most susceptible colonies have died, so their proportion relative to the resistant survivors drops. A recent disease manual with two sets of underwater disease identification cards, one for the Caribbean and one for the Indo-Pacific, were published with the goal of standardizing the disease identifications, nomenclature used to describe and characterize them, and methodology to estimate prevalence, incidence, virulence, and their variability (Raymundo et al. 2008; Beeden et al. 2008; Weil and Hooten 2008).

Prevalence of WPD, YBD, WBD, BBD, and DSD showed high seasonal variability in Caribbean localities with usually higher prevalence and frequent outbreaks during summer's high water temperatures (Borger 2003; Gil-Agudelo et al. 2004; Borger and Steiner 2005; Bruckner and Bruckner 2006; Weil et al., in press; Weil, unpublished). In the late 1990s and early 2000s, YBD was a seasonal disease in La Parguera, Puerto Rico, active and highly visible during Summer--Fall, nearly disappearing from some colonies and completely from others during the Winter--Spring. In the last 6 years however, this seasonality disappeared. Prevalence of YBD increased every year with corresponding increase in severity (number of lesions and rate of disease advance) to epizootic levels in many reefs in Puerto Rico and other Caribbean localities (Bruckner and Bruckner 2006; Weil and Croquer 2009, Cróquer and Weil 2009a, b; Weil et al., in press). Moreover, increase in prevalence and severity over time covaried significantly with increasing average winter and yearly water temperatures (Fig. 4a, see below) (Weil 2008; Harvell et al. 2009; Weil et al., in press), thus warmer winters seem to have affected disease dynamics. However, co-variation does not mean causality, so the increase in prevalence could also result from increased incidence (number of new infected colonies per unit time) until most susceptible colonies were infected in the population. Prevalence could stay high due to the warmer temperatures until all susceptible colonies die from the disease and the relative proportion of diseased colonies is reduced over time (Bruckner and Hill 2009).

Rates of tissue loss are also highly variable and depend on the virulence of the pathogen, the susceptibility/resistance of the host, synergistic environmental conditions, and the duration of the infection (Bruckner 2002; Bruckner and Bruckner

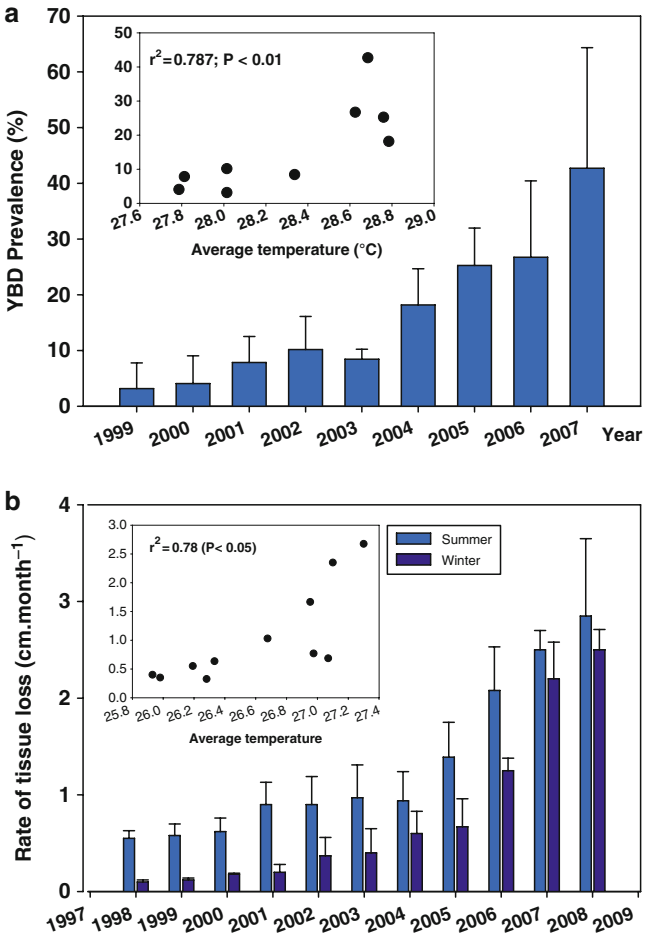

Fig. 4 Temporal changes in the dynamics of Caribbean yellow band disease. (**a**) Increase in YBD prevalence in the coral genus *Montastraea* in reefs off the south-west coast of Puerto Rico from 1999 to 2007 and the positive and significant ($r^2 = 0.787$, $P \leq 0.01$) correlation with average yearly surface water temperature (*inset*). (**b**) Seasonal variability in YBD lesion growth rates (virulence) measured in over 100 tagged colonies of *M. faveolata* in La Parguera, Puerto Rico from 1999 to 2008, and the significant positive co-variation ($r^2 = 0.54$, $P \leq 0.05$) between linear YBD lesion growth rates and the average seasonal surface seawater temperature for the same period (*inset*). (Modified from Weil et al., in press)

2006; Harvell et al. 2007). For example, rates of tissue loss were significantly higher during the fourth WPD outbreak even though the same pathogen seemed to have been the cause (Richardson and Aronson 2002). In Puerto Rico, the number of YBD disease lesions in *M. faveolata* colonies increased significantly over time with some very large colonies showing over 32 lesions at once. This led to a significant increase in rate of tissue loss, killing the colonies faster (Weil et al., in press).

The average rate of tissue loss (advance of the disease edge) estimated from *M. faveolata* colonies over the years in Mona and Desecheo islands was generally low (0.5–1.0 cm/month) and variable across colonies, months, and localities (Bruckner and Bruckner 2006). Similar results from over 200 tagged colonies of the same species that were checked bi-annually were obtained in La Parguera. However, the rates of tissue loss increased with time, from 0.2 to 3.6 cm $month^{-1}$ (Fig. 4b) and the seasonality observed in prevalence and virulence tended to decline over the years. Both prevalence and rates of tissue loss significantly increased from 2003 to 2008 and were correlated with increasing water temperatures (Weil et al., in press; Weil, unpublished) (Fig. 4). In Colombia, *M. annularis* and *S. siderea* had the highest prevalence of dark spot disease (10% and 5%, respectively) whereas the disease was much less common in *M. faveolata*, *M. franksi*, *S. intersepta*, and *M. cavernosa* (Gil-Agudelo 1998). Recent wide geographic surveys in the Caribbean showed lower prevalence values of DSD than those reported for Colombia and in general, lower prevalence in northern compared to southern localities (Weil and Croquer 2009).

3.5 Host Ranges

Overall, host ranges for most Caribbean coral reef diseases have remained stable or have increased over the years with many other reef organisms observed with similar disease signs in the region (Table 1) (Weil 2004; Bruckner 2009). Corals and other reef invertebrates are relatively simple organisms with a limited range of signs or visible "responses" to infections. Unless we do histology, most of what we can see (or characterize) are the external manifestation of responses of the diseased tissues or, just the pattern of tissue mortality. These include, but are not limited to, different patterns of tissue loss, pigmentation changes, general tissue conditions, mucus release, and growth anomalies. There has been no consistent histopathological research that could provide reliable descriptors for the different pathologies (Work and Aeby 2006; Work et al. 2008a).

Bleaching (presumably due to elevated temperature) has the widest host range affecting at least 62 corals, 29 octocorals, eight sponges, five hydrocorals, and two zoanthids in the Caribbean (McClanahan et al. 2009; Prada et al. 2009) (Table 1, Fig. 5). Bleached corals are still alive, and if conditions return to normal quickly enough, most colonies can fully recover. Four coral diseases, WPD, CCI, BBD, and RBD had the widest host ranges in the Caribbean with 41, 21, 19, and 13 susceptible scleractinian coral species, respectively (Table 1). Caribbean yellow band has been reported in 11 species of important reef-building corals. The total number of coral species affected by DSD has increased over time possibly due to the expansion of surveys. Sixteen important scleractinian species have been reported to show signs corresponding to those characteristic of the disease (Table 1)

Fig. 5 Bleached colonies of important reef-building species during the 2005 event. In many reefs of Puerto Rico and the US Virgin Islands, up to 90% of all colonies of important reef-building species were fully or significantly bleached [*Montastraea faveolata* (**a**, **b**), *A. palmata* (**c**), *D. cylindrus* (**d**), *C. natans* and *S. intersepta* (**e**), and *D. strigosa* and *S. siderea* (**j**)]. Bleached colonies of species that never been observed bleached in these reefs included *Scolymia cubensis* (**f**) and *Mycetophyllia ferox* (**g**) among others. The event produced significant mortalities in the agaricids and acroporids (**h**, **i**). Adjacent colonies of the same species showed significant differences in bleaching intensity in some localities (i.e., *M. faveolata* and *C. natans* (**k**, **l**), and some species did not bleach in certain areas (i.e., *Meandrina meandrites*) (**m**). Several species of octocorals bleached in many localities (**n**) (Photos E. Weil)

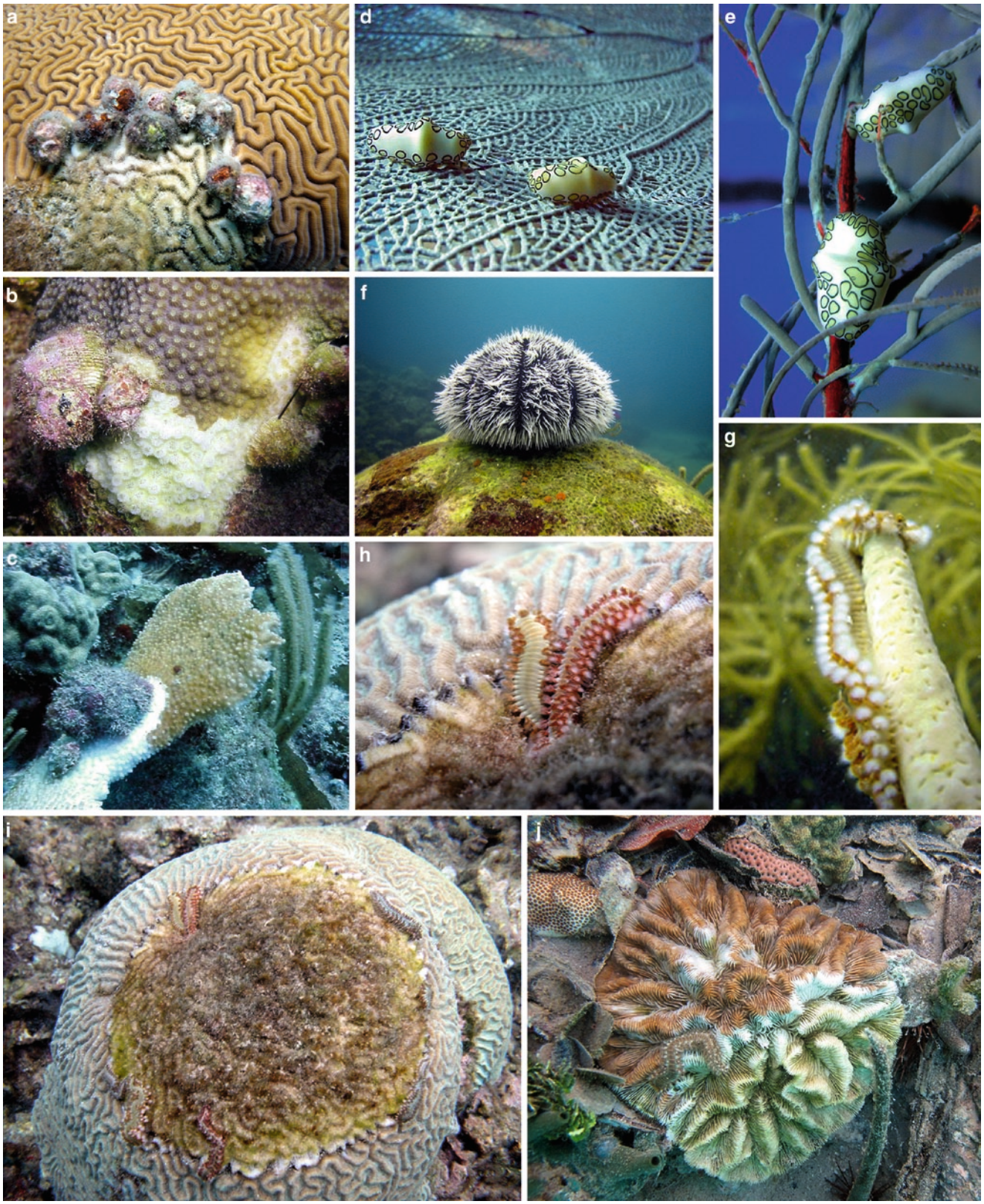

Fig. 6 Potential invertebrate vectors (reservoirs?) of coral diseases in the western Atlantic include the snails *Coralliophila abbreviata* and *C. caribaea*, here seen preying on *D. labyrinthiformis* (**a**), *M. faveolata* (**b**) and *A. palmata* (**c**); the flamingo tongue *Cyphoma gibossum*, a common predator of sea fans (**d**) and other octocorals (**e**); sea urchins which are "omnivorous" and can pick up pathogens from sediments or turf algae and move them to corals (**f**); and the fireworm *Hermodice carunculata* which preys on both octocorals (**g**) and corals. This fireworm has been frequently observed eating at the edges of diseased and healthy areas of corals with BBD (**h**, **i**), WPD (**j**) (Photo by C. Rogers), and white band disease (Photos E. Weil)

(Gil-Agudelo et al. 2004; Weil et al. 2002; Cróquer and Weil 2009a).

White plague disease, BBD, RBD, and DSD have been reported to affect five, four, one, and one coral species, respectively, in Brazil. Nine of the most common and abundant octocorals in the Caribbean seem to be susceptible to ASP, six to BBD, at least five to RDB, and several to other compromised health conditions and growth anomalies. Overall, at least six different diseases affect five of the most important and most common reef-building genera (including *Montastraea*, *Diploria, Colpophyllia, Acropora,* and *Agaricia*), and four different diseases affect 11 of the main reef-building genera (Fig. 7).

Most of the information used to compile lists of susceptible species came from single observations in space and time. A new host was added to the list if a single, or just a few colonies of a species was (were) observed with the disease signs without any verification of the pathology and/or etiology. As mentioned above, Koch's postulates have only been verified for a few pathogens in a few species, usually one for each condition [i.e. *Diploria strigosa* (BBD) (Rützler and Santavy 1983, *A. palmata* (white patches) (Patterson et al. 2002), *D. stokesi* (WPD) (Denner et al. 2003); *A. cervicornis* (WBD) (Gil-Agudelo et al. 2006); *G. ventalina,* and *G. flabellum* (ASP) (Smith et al. 1996; Geiser et al. 1998)]. Confirmation of the pathology of all the potentially susceptible species for each disease has never been done. The temporal dynamics of these infections (species could become resistant after the first infection, or susceptible colonies could be quickly eliminated from the population) has never been properly investigated.

Overall, only a fraction of the listed host species for each disease is usually observed with the disease signs when conducting typically annual field surveys, and the actual number of susceptible species (host range) for each disease will only be determined when the same pathogen is not only found in each species, but is shown to be the cause of the disease signs.

3.6 Vectors and Reservoirs

There are only two reports with experimental evidence of invertebrates acting as reservoirs (organism, substrate, or other media where the pathogen spends some time and completes part of its life cycle) and vectors (organisms or other media that act as a carrier and delivery medium for a pathogen) of a coral disease. The fireworm *Hermodice carunculata* in the Mediterranean is a vector for *Vibrio shiloi*, the pathogen that causes bacterial bleaching in the coral *Oculina patagonica* (Sussman et al. 2003), and the predatory Caribbean snail *Coralliophila abbreviata*, harbors *S. marcescens*,

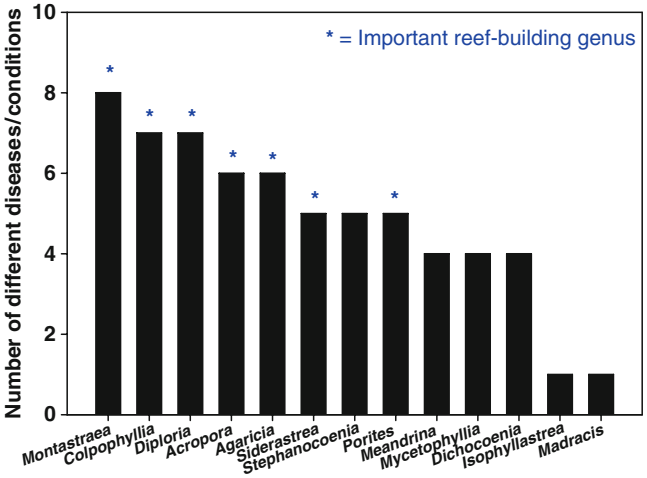

Fig. 7 The number of different diseases affecting the most important Caribbean reef-building scleractinian coral genera (*). Eleven genera of the 26 reported for the Caribbean are affected by at least four or more different diseases/conditions

the pathogen responsible for WPA in *A. palmata* in the Florida Keys (Williams and Miller 2005) (Fig. 6c).

Hermodice carunculata is a coral predator, ingesting *Vibrio shiloi* and keeping it alive in its gut (Sussman et al. 2003). This worm (or a similar species) is very common in coral reef communities across the Caribbean and is one of the main predators of acroporid and massive corals, hydrocorals, and octocorals. It is frequently observed feeding on the edges of WPD, YBD, and BBD active lesions in colonies of *M. faveolata, D. strigosa,* and *C. natans,* and ASP-infected sea fans (Fig. 6g – j). It is then possible that this fireworm could act as a vector and a reservoir for one or several of these Caribbean diseases as well. To date, disease reservoirs have only been identified for BBD (biofilms in reef sediments, which contain nonpathogenic versions of the BBD consortium) (Richardson 1997), and WPD (*Halimeda opuntia* mats, which seem to harbor the WPD pathogen) (Nugues et al. 2004).

Other potential disease vectors and possible reservoirs include parrotfishes (i.e., *Sparisoma viride*), damsel fishes (*Stegastes planifrons* and *Microspathodon chrysurus*), and the butterfly fish *Chaetodon capistratus*, a common coral predator capable of moving the BBD pathogen from diseased to healthy corals in experimental settings (Aeby and Santavy 2006), and the snail *Cyphoma gibossum*, a predator of sea fans and other octocorals (Fig. 6d, e). Fishes tend to directly bite diseased and healthy colonies of important reef-building species in the Caribbean potentially moving pathogens around. Vectors may be involved in disease transmission and spread at both local and regional scales. This is an important aspect of the dynamics of coral reef diseases, particularly as populations of many of these potential vectors have been significantly increasing (mostly as a consequence of overfishing

of their main predators or recent successful reproduction) for which there is little information.

4 Environmental Drivers

The multiple and complex biological associations within the coral holobiont and the currently changing environmental conditions complicate attempts to isolate individual drivers/causes and to make predictions and/or extrapolations based on short-term and single-locality studies. Of many potential factors, increasing sea water temperature seems to be one that may have favored the emergence of coral diseases (Harvell et al. 1999, 2002, 2007, 2009). Evidence of this includes the following:

1. Bleaching events and most of the early disease outbreaks affecting coral reef organisms and other marine animals occurred during the warm Summer and early Fall seasons (Harvell et al. 1999; Weil et al. 2006; Van Oppen and Lough 2009).
2. The first Caribbean-wide surveys of coral diseases conducted during the Summer--Fall season in 1999 showed an increase in disease prevalence at the community level from Bermuda in the north-west Atlantic to the more tropical southern Caribbean (Venezuela and Colombia), suggesting a potential relationship with warmer temperatures (Weil et al. 2002).
3. The infection of *Pocillopora damicornis* by *V. coralliilyticus* (bacteria producing bleaching in this species) showed no signs of infection below 22°C, tissue bleaching from infection between 24°C and 26°C, and rapid tissue lyses from 27°C to 29°C (Ben-Haim et al. 2003a, b).
4. Recent evidence indicates that high water temperatures compromise host susceptibility and increase virulence (Harvell et al. 2002; Ritchie 2006; Bruno et al. 2007; Weil et al. in press, which would presumably increase prevalence and incidence over time.
5. Functional gene analysis of samples from experimental *Porites compressa* colonies subjected to environmental stressors (increased temperature, elevated nutrients and CO_2, and lower pH) showed increased abundance of microbial genes involved in virulence, stress resistance, sulfur and nitrogen metabolism, and coral-associated microbiota (Archaea, Bacteria, protists) shifted from a healthy community (e.g., Cyanobacteria, Proteobacteria, and zooxanthellae) to a community of microbes often found on diseased corals (Thurber et al. 2009)
6. Water temperature and disease prevalence in *A. palmata* colonies in St. John, USVI, were positively correlated, but bleached colonies exhibited a stronger relationship than unbleached colonies. In addition, a positive relationship between the severity of disease, as estimated by the area of the disease lesions, was apparent only for bleached corals (Muller et al. 2008).
7. Bacteria populations in corals change in composition, abundance, and possibly virulence when temperature increases (Ritchie 2006; Bourne et al. 2007; Weil et al. 2009b; Harvell et al. 2009; Sunagawa et al. 2009). Similar correlations of increased prevalence with increasing water temperatures have been found in a long-term study in the Great Barrier Reef (Selig et al. 2006; Bruno et al. 2007).

The WPD and YBD outbreaks in the eastern Caribbean have been associated with higher than normal water temperatures over the years, which have also produced widespread bleaching in the area (Bruckner and Bruckner 2006; Miller et al. 2006, 2009; Rogers et al. 2008a, b; McClanahan et al. 2009). Furthermore, in contrast to previous WPD outbreaks in La Parguera, Puerto Rico, the WPD outbreak after the 2005 bleaching event peaked during the unusually warmer winter season (February–March) (Cróquer and Weil 2009b, Weil et al., in press).

A positive correlation between increasing water temperatures and increasing prevalence of YBD in *Montastraea* colonies was found over a 9-year study in Puerto Rico (Fig. 4a, b) (Weil 2008; Weil et al., in press.). Furthermore, the seasonal rates of YBD-induced tissue loss in *M. faveolata* were significantly different between 1999 and 2004, with higher rates of tissue loss during the Summer--Fall compared to the Winter--Spring. These differences, however, disappeared as winter water temperatures became warmer after 2004 (Fig. 4b), and YBD has remained active all year long with similar rates of tissue loss throughout the year (Weil 2008; Bruckner and Hill 2009; Harvell et al. 2009;

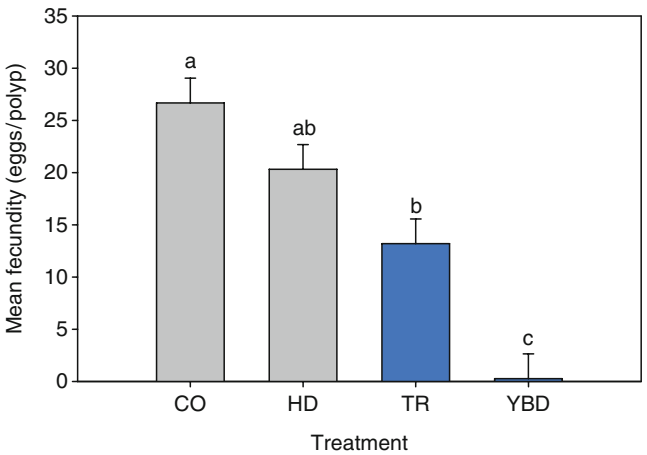

Fig. 8 Significant decline in reproductive output within diseased and healthy-looking areas of YBD-infected and control colonies of the important reef-building coral *Montastraea faveolata* in La Parguera, Puerto Rico. CO = control colonies with no signs of disease, HD healthy looking areas of diseased colonies, TR = transition areas (area between the YBD pale tissue and the healthy looking tissue), and YBD = disease area in colony (Modified from Weil et al. 2009a)

Weil et al., in press). Warmer winters favor the higher annual advance rates of lesions, significantly increasing the overall tissue and colony mortality, reducing fecundity (Fig. 8) and potentially affecting the short- and long-term recovery of populations (Weil et al. 2009a, b, in press).

High prevalence of dark spots disease seemed to be related to high water temperatures and specific depths in some localities but not in others (Gil-Agudelo and Garzón-Ferreira 2001; Borger 2003; Gotchfeld et al. 2006). Cróquer and Weil (2009a) found that populations of *S. siderea* exhibited a higher prevalence of DSD at intermediate (10 m) depths (25–40%), whereas *Stephanocoenia* populations were significantly more affected by DSD in deeper (>15 m) habitats (21–26%). In South Florida, prevalence of DSD increased during April--July but decreased during winter months (Borger and Steiner 2005). After 2 years of monitoring, Borger and Steiner (2005) suggested that DSD may be a general stress response of *S. siderea* that is exacerbated by an increase in water temperature, thereby illustrating geographic differences in environmental conditions conducive to development of DSD.

Other factors such as nutrient concentration might also affect the dynamics of some coral diseases. High nutrient exposure doubled rates of tissue loss in YBD diseased colonies of *M. faveolata* in Mexico (Bruno et al. 2003), showing response of the disease to changing nutrient conditions in surrounding waters (dissolved nutrients were artificially added). However, in similar nutrient experiments to those in Mexico, results from Puerto Rico showed no significant increase in number of lesions or rates of advance in YBD-infected colonies of *M. faveolata* when compared with controls (Bruno and Weil, unpublished). Another study showed higher prevalence of BBD in reefs closer to sewage effluents (Kaczmarsky et al. 2005).

Recent studies have shown that *A. palmata* colonies that were physically damaged by heavy swells had higher disease prevalence than undamaged colonies, with statistically greater prevalence when average monthly water temperature exceeded 28°C (Bright, personal communication 2010). Fragmentation of *A. palmata* and *A. cervicornis*, as well as other branching or columnar species during storms, leads to increases in number of colonies in the populations. However, during this process, open wounds and damaged tissues are presumably more susceptible to infection by opportunistic bacteria; thus, more intense storms predicted to occur with climate change could lead to more disease and higher mortality of these fragments.

5 Consequences and Management Implications

The potential of disease outbreaks to significantly change coral reefs was first shown by the massive mortalities of the acroporids and the black sea urchin *D. antillarum* in the Caribbean in the early 1980s. In a relatively short time and over a wide geographic region, populations of these species suffered up to 95% mortality (Gladfelter 1982; Lessios et al. 1984a, b; Carpenter 1990a, b) producing a cascade of significant ecological changes in the dynamics, function, and structure of coral reefs at local and geographic scales (Hughes 1994; Harvell et al. 1999; Aronson and Precht 2001a, b; Weil et al. 2003).

Disease etiology and dynamics seem to be highly variable across different spatial (populations, depth gradients, and reefs) and temporal scales (Weil et al. 2002; Bruckner and Bruckner 2006; Weil and Croquer 2009; Cróquer and Weil 2009a, b). Several infectious diseases have been persistent over the years throughout the Caribbean. Six of these have wide geographic distributions (BBD, WPD, YBD, DSD, ASP, and CCI) and could be considered chronic in many localities (Table 1) (Weil and Croquer 2009; Cróquer and Weil 2009a). Several more epizootics have occurred since the widespread WBD outbreak of the early 1980s, three of which have had wide geographic distributions and significant impact on the host species, WPD, ASP, and YBD (Bruckner and Bruckner 2006; Bruckner and Hill 2009; Weil and Croquer 2009; Cróquer and Weil 2009a; Weil et al. 2009a; Flynn and Weil, in press; Weil et al., in press).

Some of the most important reef-building coral genera (i.e., *Montastraea*, *Diploria*, *Siderastrea*, *Colpophyllia*) in the Caribbean are susceptible to at least five of the most prevalent and virulent diseases and several other compromised health problems (Fig. 7). Local populations of the three species of *Montastraea* and other important reef-building species in reefs off the Virgin Islands and Puerto Rico have been devastated by WPD epizootics, pervasive YBD, two intensive bleaching events, and their synergistic impact. For example, WPD from 2005 to 2007 caused more coral loss (over 60% loss in live tissue cover) than any other factor up until 2005/2006 in the USVI (Miller et al. 2009), some reefs off the east coast of Puerto Rico (García-Sais et al. 2008), and Curacao and Grenada (Weil and Cróquer 2009).

Similarly, reefs off La Parguera, on the southwest coast of Puerto Rico, showed an average loss of coral cover of 53.7% over 4 years (2004–2007), but as a consequence of two WPD epizootics, a persistent YBD outbreak and the bleaching event of 2005 (Weil et al., in press) (Fig. 9). Similar coral tissue losses have been reported for Mona and Desecheo islands west of Puerto Rico where YBD and bleaching have been the major problems (Bruckner and Bruckner 2006; Bruckner and Hill 2009).

As average water temperatures increase, bleaching events have become more frequent and intense over wider geographic scales, affecting most zooxanthellae-bearing reef organisms to increasing depths. Before 2005, bleaching in the Caribbean caused variable, but generally low coral

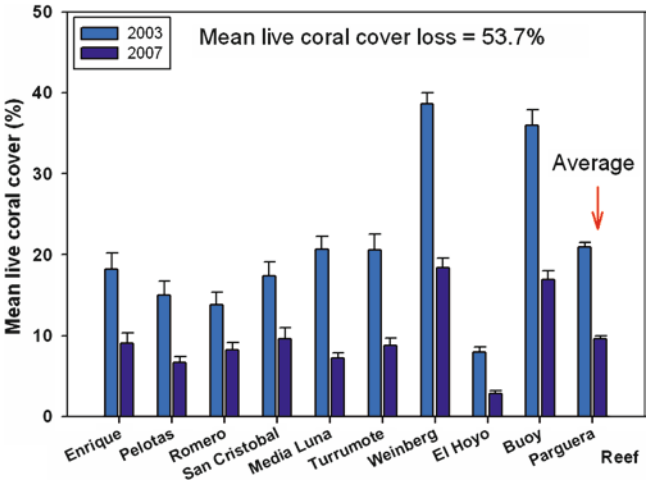

Fig. 9 Average loss in live coral cover in nine reefs off La Parguera between 2003 and 2004, and 2007. The average loss in live coral tissue for the area was 53.7% (±7.2%) (Modified from Weil et al., in press)

mortality, nothing like the mass mortalities of the scale observed in the Indo-Pacific. Only the recent extensive bleaching of 2005, the worst ever recorded for the region, produced widespread mortality in several reefs of the eastern Caribbean, which was compounded with disease outbreaks in several localities (Miller et al. 2009; Rogers et al. 2008a, b; McClanahan et al. 2009; Cróquer and Weil 2009a, b).

Significant coral mortality due to bleaching was observed for the first time in Puerto Rico during this event; however, this mortality was not widespread across all species, suggesting differential susceptibility and specific resistance to bleaching. Most agaricids, *M. ferox*, and *A. palmata* showed the highest mortality due to bleaching. A high proportion of other reef organisms (octocorals, hydrocorals, and zoanthids, and a few sponges) bleached in many localities (Fig. 5), with some of these showing significant population mortalities (i.e. *Millepora* spp. *Erythropodium caribbaeorum*, and *Palythoa caribbaeorum*) (McClanahan et al. 2009; Prada et al. 2009; Weil et al., in press).

At the peak of the bleaching, outbreaks of WPD started to be observed in Puerto Rico, the Virgin Islands, and Grenada (Hernández-Delgado et al. 2006; Cróquer and Weil 2009b; Miller et al. 2009; Rogers et al. 2008a, b; Weil et al., in press). Increasing water temperature trends in the region, possibly associated with global climate change, and the frequency and intensity of bleaching events could have affected and will probably affect the emergence, dispersion, and virulence of coral reef diseases and their consequences in the recent past and the near future. An increasing number of colonies and species are being infected by more than one disease at the same time. Some colonies of *M. faveolata*, for example, have been observed with four different pathologies simultaneously (Cróquer and Weil 2009a, b; Weil and Croquer, unpublished data), which significantly increases rates of tissue and colony mortality over time.

Furthermore, recent studies showed that in addition to the general tissue loss, some diseases could significantly reduce the fitness (reproductive output) in important reef species, similar to what was reported for bleaching almost 20 years ago (Szmant and Gassman 1990). Sexual reproduction is critical to coral population dynamics and the long-term regeneration of coral reefs. Recurrent recruitment failure and low reproductive output in corals have been highlighted as explanations as to why reefs are not recovering from major coral losses (Hughes and Connell 1999; Hughes and Tanner 2000). Recent data showed that ASP significantly reduced fecundity (fitness) in *G. ventalina* (Flynn and Weil 2008) and YBD significantly affected the reproductive output of *M. faveolata* (Weil et al. 2009). Furthermore, Cervino et al. (2001, 2004a, 2008) indicated that all the pale, yellowish areas in the yellow band lesions are depleted of zooxanthellae, which would reduce local energy supply and potential energy available for the rest of the colony.

Thirty two percent of reef-building scleractinian corals around the world face an elevated risk of extinction due mainly to bleaching and disease driven presumably by elevated sea water temperature and further exacerbated by local anthropogenic stressors (Carpenter et al. 2008). The proportion of threatened (not including Near Threatened coral species) recognized by IUCN exceeds that of most terrestrial animal groups apart from amphibians, particularly because of corals' apparent susceptibility to climate change (particularly high sea water temperatures) and local anthropogenic factors (Carpenter et al. 2008). This plus all the recent reports of high mortality rates due to local and/or extensive disease outbreaks affecting coral reefs worldwide is a major cause of concern for the future of these important tropical marine communities.

Progress in coral disease research requires collaboration among experts across many different disciplines, including genetics, physiology, cell biology, ecology, pathology, microbiology, and epidemiology. We need innovative, preferably nondestructive techniques to diagnose diseases. One promising new approach involves the use of custom-designed microarrays to characterize microbial patterns (Kellogg and Zawada 2009). Sequential, frequent sampling of coral colonies with and without disease will reveal changes in microbial communities over time. Additional histological analysis of healthy and diseased corals should provide further clues. Further understanding will come from laboratory experiments that test the effects of temperature, irradiance, sediments, nutrients, and other pollutants on the development and progression of disease in corals. Currently, the tools for genotyping coral colonies

are available for only a few species, and developing these tools for other major reef-building species would help us to evaluate whether or not certain genotypes are more resistant to diseases.

Even after more than 35 years since the first report of a coral disease in the Caribbean, researchers keep finding new diseases affecting corals and other important invertebrates and CCA groups responsible for building and maintaining these important tropical communities. Diseases of coral reef organisms have become one of the most, if not the most, important factors accelerating the decline of coral reefs and the potential loss of biodiversity (when the food and energy sources and the three-dimensional limestone framework in these communities disappear), compromising the future integrity of many coral reefs in the western Atlantic. However, to date there is no report of any coral species that has been extirpated from disease, even locally.

Reefs in the Caribbean have gone through significant changes in community structure with decreases in coral cover and increases in macroalgal cover (Edmunds 1991; Carpenter 1990a, b; Hughes 1994; Bellwood et al. 2004; Rogers and Miller 2006; Weil et al., in press). The impact of diseases on the reproductive output of corals and octocorals further hinder their potential future recovery. Ongoing monitoring programs and yearly surveys in many localities have failed to show any significant recovery following the recent outbreaks (Weil, unpublished data; C.S. Rogers, personal communication). Even after 25 years, the acroporids and sea urchin populations have not been able to recover from the epizootic events of the early 1980s. Collapsed coral populations would produce significantly fewer larvae, so recruitment and juvenile survivorship would be potentially very low making it difficult, or slow, for populations to recover, even if environmental conditions and other factors are favorable. In Puerto Rico, surviving populations and new recruits and juveniles of *A. palmata* were frequently affected by storms, hurricanes, sedimentation, and anthropogenic impacts after the mass mortalities of the 1980s (Weil et al. 2003).

Even though some positive results indicating lower numbers and prevalence of coral diseases inside marine protected areas (MPAs) and/or marine reserves have been reported (Raymundo et al. 2008), more information is needed to generalize the potential "protection" these areas provide to coral populations. Coral reefs within and outside of MPAs in the US Virgin Islands had losses of over 60% of the coral cover following the disease outbreak associated with the 2005 bleaching episode (Miller et al. 2009; Rogers et al. 2008a, b). In other localities, disease prevalence inside MPAs was not lower than outside MPAs (Coelho and Manfrino 2007: Page et al. 2009). It is challenging to test the hypothesis that coral reefs within MPAs will be less susceptible to diseases than those outside protected areas. A variety of complex factors has to be controlled for, for example:

- The actual level of protection that the area receives (reflecting compliance with regulations and effectiveness of enforcement)
- The length of time that protective measures have been in place
- The type of protection (prohibition of fishing, anchoring, etc.)
- The history of fishing and any other extractive uses before the area was effectively protected [some areas might have had very little fishing to begin with, for example]
- Other stressors like runoff, pollution that could affect the condition of the marine resources
- Prevalence of diseases before the MPA was established
- The validity of the control ("reference") areas used for comparison with protected areas

Our current limited knowledge of the pathogenesis and other important aspects of most coral reef diseases (Lesser et al. 2007) undermines our ability to develop strategies to solve the problem in the near future. MPA boundaries will not prevent disease outbreaks or bleaching events, but, protecting large, genetically variable fecund populations of the most important reef-building groups could increase the species survivorship by protecting potentially resistant genotypes that could then reseed other degraded populations outside the reserves (Vollmer and Kline 2008).

6 Summary

Coral reefs are declining around the world due to natural and/or human-produced stressors, including global climate change. Recently, diseases of coral reef organisms have become increasingly important in the deterioration of these important marine communities. The Caribbean has been dubbed a "disease hot spot" due to the fast emergence of diseases and frequent epizootic events with significant coral mortalities in the last 30 years. Fifteen disease conditions affecting corals and other important reef organisms have been described, but most do not have defined pathologies nor have they been well characterized. Of these, ten conditions with consistent signs are affecting most of the important reef-building coral species, five are affecting at least 12 species of octocorals, two at least three species of crustose coralline algae (CCA), and two a single zoanthid species, while several uncharacterized conditions affect sponges and other reef organisms. Variability in disease manifestation and distribution complicates their characterization and identification. Most of these diseases have a Wider Caribbean distribution with a few, like black band disease, showing worldwide distributions.

Three widespread and several local outbreaks produced significant mortalities over a wide geographic range, with a cascade of significant changes in community structure, decreases in coral cover, and increases in macroalgal cover. The impact of diseases on the reproductive output of corals and octocorals further hinders their potential for recovery. Our limited understanding of the pathogenesis of most coral reef diseases undermines our ability to develop strategies to reduce their effects in the near future. Further advances in coral disease research require collaboration among experts across many different disciplines, development and use of new techniques, and controlled laboratory experiments. Caribbean coral reefs will benefit from protection of resistant coral genotypes along with greater efforts to reduce the manageable stresses caused by humans. Protecting large, genetically variable fecund populations of the most important reef-building species could increase the survivorship of resistant genotypes that could then reseed degraded populations. However, this has to come together with greater efforts to manage human activities that stress coral reefs, and the reestablishment of former environmental quality for the survival of coral reefs in the region.

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