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REVIEW

Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals

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ABSTRACT: Since the mid-1990s, coral diseases have increased in number, species affected, and geographic extent. To date, 18 coral diseases, affecting at least 150 scleractinian, gorgonian, and hydrozoan zooxanthellate species, have been described from the Caribbean and the Indo-Pacific. These diseases are associated with pathogens including bacteria, cyanobacteria, fungi, and protists and with abiotic stressors including elevated seawater temperature, sedimentation, eutrophication, and pollution. Etiologies of only 5 of the 18 coral diseases have been determined through fulfillment of Koch's postulates. Corals and other invertebrates utilize innate immune mechanisms including physiochemical barriers and cellular and humoral defenses against pathogens. Here we review the described coral diseases, known etiologies, and efforts to determine unknown etiologies. We define disease terms, discuss the limitations of Koch's postulates, describe alternative techniques for identifying disease-causing organisms, and review coral immunology.

KEY WORDS: Coral · Disease · Immunology · Invertebrate · Review

INTRODUCTION

Coral reefs are in severe decline. The most reliable estimates suggest that worldwide 27% have already been lost, with another 16% at serious risk of loss (Wilkinson 2002). Coral disease is thought to be a major cause for this decline (Dustan 1999, Porter et al. 2001). Epizootics have been reported for several coral species (Goreau et al. 1998, Richardson 1998, Richardson et al. 1998a,b, Harvell et al. 1999, 2001, Porter et al. 2001) and evidence is mounting of substantial declines in the biodiversity and abundance of reef-building corals worldwide (Hayes & Goreau 1998, Porter & Tougas 2001, Wilkinson 2002). Within the Caribbean, populations of elkhorn and staghorn corals, *Acropora palmata* and *A. cervicornis*, are being decimated by disease (Gladfelter 1982, Bythell & Sheppard 1993, Aronson & Precht 1997, 2001, Aronson et al. 1998, 2002, Greenstein et al. 1998, Miller et al. 2002, Patterson et al. 2002), with losses of *A. palmata* in the Florida Keys National Marine Sanctuary (FKNMS) averaging 87% (Fig. 1) or greater (Miller et al. 2002, Patterson et al. 2002, Sutherland & Ritchie in press). On most Caribbean reefs, loss of acroporids is accompanied by an apparent ecological phase shift from coral-dominated substrata to algal-dominated substrata (Hughes 1994). While severe population declines of Caribbean acroporid corals have led to the identification of *A. palmata* and *A. cervicornis* as candidates for inclusion on the Endangered Species List (Diaz-Soltero 1999), the impact of most diseases on coral populations is poorly understood.

Coral disease is becoming more widespread. The first coral disease was reported in 1965 and during the subsequent 3 decades, only 4 new diseases were reported (Table 1). Beginning in the mid-1990s, reports of novel coral diseases increased worldwide, and by 2002, 13 new diseases were described (Table 1, Fig. 2). In the FKNMS, number of locations exhibiting disease increased from 26 to 131 stations (404% increase) and

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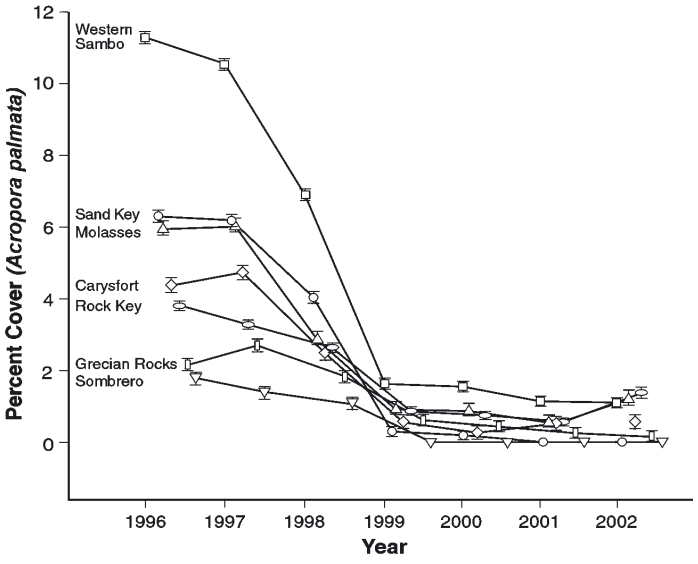

Fig. 1. *Acropora palmata.* Population decline (% cover) by 2002 on each of 7 reefs surveyed in the Florida Keys National Marine Sanctuary. (□) Western Sambo Reef: 91%; (○) Sand Key Reef: 100%; (△) Molasses Reef: 79%; (◇) Carysfort Reef: 89%; (⬭) Rock Key Reef: 58%; (▯) Grecian Rocks Reef: 95%; (▽) Sombrero Reef: 100%. Data in graph are mean ± SD

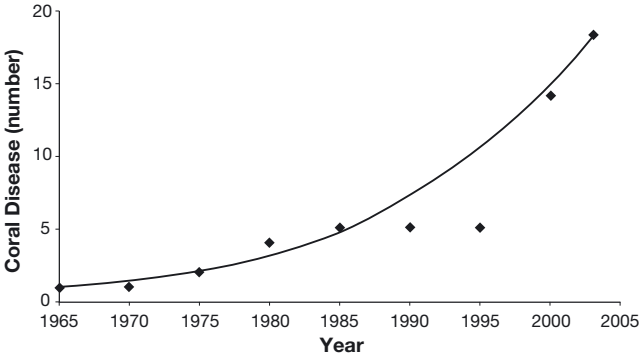

Fig. 2. Exponential increase in the number of described coral diseases since the first report of disease in 1965. Although increased awareness and increased observational time might explain some of this increase, many diseases, such as white plague, are so common and so distinctive that their first description may reasonably be assumed to mark their first appearance as a phenomenon influencing the population dynamics of corals

number of coral species exhibiting disease increased from 11 to 36 (218% increase) between 1996 and 2000 (Porter et al. 2001). These disease increases parallel a 37% decline in living coral over the same time period and at the same stations (Porter et al. 2002).

Biodiversity of corals is much greater in the Indo-Pacific than in the Caribbean. Age and geographic extent of the Indo-Pacific region contributes greatly to its richness (Veron 1995). Whereas only 53 coral species are found in Discovery Bay, Jamaica (Wells 1973), 362 coral species are found in Milne Bay, Papua New Guinea (Werner & Allen 1998) and 350 coral species are found in the Great Barrier Reef, Australia (Veron 1985). Despite the greater species richness of the Indo-Pacific, the number of species affected by disease is proportionally much lower than in the Caribbean (Tables 2 & 3).

Of the 18 coral diseases described to date, 4 are reported globally: black band (BBD), white plague-like diseases (WPL-L), shut-down reaction (SDR), and skeletal anomalies (SKA); 9 are found exclusively in the Caribbean: white band Types I (WBD I) and II (WBD II), white plague Types I (WPL I), II (WPL II), and

Table 1. First report of coral diseases in the Caribbean and the Indo-Pacific

Disease	Abbreviation	Year	Source
Skeletal anomalies	SKA	1965	Squires (1965)
Black band	BBD	1973	Antonius (1973)
White plague Type I	WPL I	1977	Dustan (1977)
Shut-down reaction	SDR	1977	Antonius (1977)
White band Type I	WBD I	1982	Gladfelter (1982)
Aspergillosis	ASP	1996	Smith et al. (1996)
White pox	WPD	1996	Holden (1996)
Vibrio shiloi-induced bleaching	VSB	1996	Kushmaro et al. (1996)
Yellow blotch/band	YBL	1997	Santavy & Peters (1997)
White plague Type II	WPL II	1998	Richardson et al. (1998a)
White band Type II	WBD II	1998	Ritchie & Smith (1998)
Yellow band	YBD	1998	Korrûbel & Riegl (1998)
Dark spots	DSD	1998	Goreau et al. (1998)
Skeleton eroding band	SEB	2000	Antonius & Lipscomb (2000)
Fungal-protozoan syndrome	FPS	2000	Cerrano et al. (2000)
White plague Type III	WPL III	2001	Richardson et al. (2001)
Pink-line syndrome	PLS	2001	Ravindran et al. (2001)
Vibrio coralliilyticus-induced bleaching and disease	VCB	2002	Ben-Haim & Rosenberg (2002)

Table 2. Caribbean scleractinian, hydrozoan, and gorgonian coral species affected by diseases (total number of reported species affected by each disease and total number of reported diseases affecting each species). For WPL and WBD: I = Type I, II = Type II, III = Type III, X = type not determined. See Table 1 for disease definitions

	BBD	WPL	SDR	SKA	ASP	WBD	WPD	YBL	DSD	No. of diseases
Scleractinians										
Agaricia agaricites		I, II						X	X	4
Agaricia fragilis		X								1
Agaricia lamarcki		II		X						2
Agaricia tenuifolia		X								1
Acropora cervicornis			X	X		I,II				4
Acropora palmata	X		X	X		I	X			5
Colpophyllia natans	X	I,II,III		X				X	X	7
Cladocora arbuscula		X								1
Dendrogyra cylindrus		II								1
Dichocoenia stokesi	X	II	X	X						4
Diploria clivosa	X	X								2
Diploria labyrinthiformis	X	I,II		X				X	X	6
Diploria strigosa	X	II		X				X		4
Eusmilia fastigiata		II								1
Favia fragum	X	X		X				X		4
Isophyllastrea rigida		I							X	2
Isophyllia sinuosa		X								1
Leptoseris cucullata		X								1
Madracis decactis	X	II								2
Madracis formosa				X						1
Madracis mirabilis	X	II								2
Manicina areolata		II		X						2
Meandrina meandrites	X	II							X	3
Montastraea annularis	X	I,II,III	X	X				X	X	8
Montastraea cavernosa	X	I,II		X					X	5
Montastraea faveolata	X	I						X	X	4
Montastraea franksi	X	X						X	X	4
Mussa angulosa		I								1
Mycetophyllia aliciae		X								1
Mycetophyllia danaana		X								1
Mycetophyllia ferox		I								1
Mycetophyllia lamarkiana		I								1
Oculina diffusa		X								1
Porites astreoides	X	I		X				X		4
Porites porites		X		X						2
Scolymia cubensis		X								1
Siderastrea radians	X	X		X						3
Siderastrea siderea	X	I,II	X	X					X	6
Solenastrea bournoni		II								1
Solenastrea hyades	X	X	X							3
Stephanocoenia michelinii	X	I,II							X	4
Total	**19**	**38**	**6**	**16**	**0**	**2**	**1**	**9**	**11**	
Hydrozoans										
Millepora alcicornis		II		X						2
Millepora complanata		X								1
Total	**0**	**2**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	
Gorgonians										
Gorgonia flabellum	X				X					2
Gorgonia ventalina	X			X	X					3
Gorgonia sp.					X					1
Plexaura flexuosa	X									1
Plexaura homomalla	X									1
Plexaura sp.					X					1
Plexaurella sp.					X					1
Plexaurella homomalla				X						1
Plexaurella flexuosa				X						1
Pseudoplexaura spp.				X	X					2
Pseudoplexaura porosa				X						1
Pseudopterogorgia acerosa	X									1
Pseudopterogorgia americana	X				X					2
Total	**6**	**0**	**0**	**5**	**7**	**0**	**0**	**0**	**0**	
Total no. of species	**25**	**40**	**6**	**22**	**7**	**2**	**1**	**9**	**11**	

Table 3. Indo-Pacific scleractinian and gorgonian coral species affected by diseases (total number of reported species affected by each disease and total number of reported diseases affecting each species). See Table 1 for disease definitions

	BBD	WPL-L	SKA	VSB	VCB	SEB	YBD	PLS	FPS	No. of diseases
Scleractinians										
Acropora aspera						X				1
Acropora capillaris		X								1
Acropora clathrata	X	X	X			X	X			5
Acropora cytherea	X									1
Acropora downingi	X	X				X	X			4
Acropora florida	X	X				X	X			4
Acropora formosa	X		X			X				3
Acropora gemmifera	X									1
Acropora hemprichi		X								1
Acropora humilis	X	X				X				3
Acropora hyacinthus	X	X				X				3
Acropora intermedia	X									1
Acropora microclados	X									1
Acropora microphthalma	X									1
Acropora millepora	X									1
Acropora monticulosa	X									1
Acropora nobilis	X	X	X			X				4
Acropora palifera	X	X								2
Acropora pharaonis	X	X					X			3
Acropora robusta	X									1
Acropora sarmentosa	X									1
Acropora squarrosa		X								1
Acropora tenuis						X	X			2
Acropora valenciennesi			X							1
Acropora valida		X	X			X	X			4
Acropora variabilis		X								1
Acropora virgata			X							1
Alveopora gigas	X									1
Astreopora myriophthalma	X									1
Cladocora caespitosa									X	1
Coscinarea monile		X								1
Cyphastrea chalcidicum						X				1
Cyphastrea microphthalma							X			1
Cyphastrea serailia						X				1
Echinophyllia aspera	X									1
Echinopora gemmacea		X								1
Enallopsammia rostrata			X							1
Favia favus	X	X								2
Favia matthaii	X									1
Favia pallida	X	X								2
Favia stelligera	X	X				X				3
Favia valenciennesii			X							1
Favites abdita						X				1
Favites pentagona	X	X								2
Goniastrea pectinata	X	X								2
Goniastrea retiformis	X	X				X				3
Goniopora columna	X									1
Goniopora somaliensis	X									1
Goniopora sp.	X									1
Hydnophora microconos	X	X				X				3
Leptastrea purpurea						X				1
Leptoria phrygia	X	X								2
Leptoseris explanata						X				1
Leptoseris glabra		X								1
Leptoseris mycetoseroides		X								1
Lobophyllia corymbosa		X								1
Madrepora kauaiensis			X							1
Madrepora oculata			X							1
Montipora aequituberculata	X	X								2
Montipora ehrenbergi		X								1
Montipora florida	X									1
Montipora foliosa			X							1
Montipora informis			X							1
Montipora monasteriata						X				1

Table 3 (continued)

	BBD	WPL-L	SKA	VSB	VCB	SEB	YBD	PLS	FPS	No. of diseases
Scleractinians (continued)										
Montipora patula			X							1
Montipora verrucosa	X		X							2
Montipora sp.	X		X							2
Mycedium elephantotus		X								1
Oculina patagonica				X						1
Pavona gigantea			X							1
Pachyseris gemmae	X									1
Pachyseris rugosa						X				1
Platygyra daedalea		X								1
Platygyra lamellina	X	X								2
Platygyra pini			X							1
Platygyra sinensis			X							1
Pocillopora damicornis	X	X			X	X				4
Pocillopora eydouxi						X				1
Pocillopora verrucosa	X	X				X				3
Pocillopora meandrina			X							1
Podabacia crustacea		X								1
Porites sp.	X									1
Porites compressa			X					X		2
Porites harrisoni							X			1
Porites lichen							X			1
Porites lobata			X							1
Porites lutea	X	X	X				X	X		5
Porites nodifera							X			1
Pratzia mirabilis			X							1
Stylophora erthyaea			X							1
Stylophora pistillata	X	X				X				3
Symphyllia radians		X								1
Turbinaria mesenterina	X									1
Turbinaria reniformis		X					X			2
Verrillofungia concinna			X							1
Total	**45**	**38**	**24**	**1**	**1**	**24**	**12**	**2**	**1**	
Gorgonians										
Corallium rubrum									X	1
Eunicella cavolini									X	1
Eunicella singularis									X	1
Eunicella verrucosa									X	1
Leptogorgia sarmentosa									X	1
Paramuricea clavata									X	1
Total	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**6**	
Total no. of species	**45**	**38**	**24**	**1**	**1**	**24**	**12**	**2**	**7**	

III (WPL III), aspergillosis (ASP), white pox (WPD), yellow blotch/band (YBL), dark spots (DSD); and 6 are apparently endemic to the Indo-Pacific: yellow band (YBD), skeleton eroding band (SEB), pink-line syndrome (PLS), fungal-protozoan syndrome (FPS), *Vibrio shiloi*-induced bleaching (VSB), *V. coralliilyticus*-induced bleaching and disease (VCB). WPL-L in the Indo-Pacific may or may not be etiologically related to the 3 Caribbean WPL diseases. Etiologies and mechanisms of tissue death of the majority of coral diseases are not understood (Richardson 1998).

Tissue loss or damage from predation is often impossible to distinguish from tissue loss or damage from disease. To the human eye, there are not many ways in which coral tissue can exhibit signs of stress. Corallivores, including fishes, gastropods, and other invertebrates, produce predation scars that are easily confused with disease signs. The best method to distinguish between predation and disease is to observe the progress of the condition in the absence of predation, via predator exclusion *in situ* or predator removal under laboratory conditions. A number of coral abnormalities, some of which have been described in the literature as coral diseases, are likely associated with predation rather than disease. For example, predation by the stoplight parrotfish *Sparisoma viride*, was initially described as a coral disease termed rapid wasting syndrome (Cervino et al. 1997). Subsequent investigations identified fish predation as the primary cause of skeletal loss (Bruckner & Bruckner 2002), but the possible role of fungi in the dissolution of the skeleton (Cervino et al. 1997, Hayes & Goreau 1998) has not been fully vetted. Further, in some cases, disease signs attributed to coral disease (e.g. WPL, WBD, and WPD)

may be difficult to distinguish from predation scars produced by corallivores including the gastropod *Coralliophila abbreviata*, and the fire worm *Hermodice caruculata* (Patterson et al. 2002).

In order to facilitate an understanding of disease processes and causation in corals, it is necessary to understand general disease terminology and coral immunity. The objectives of this paper are to: (1) define disease terminology and relate these terms to coral disease, (2) discuss the process of proving disease causation, (3) review described coral diseases, known etiologies, and efforts to determine unknown etiologies, (4) illustrate each of the known Caribbean coral diseases, and (5) review coral immunology.

DISEASE TERMINOLOGY

A disease is any impairment (interruption, cessation, proliferation, or other disorder) of vital body functions, systems, or organs (Stedman 2000). The term syndrome is synonymous with disease (Stedman 2000). Etiology is analysis of causes, development, and consequences of a disease (Kinne 1980). Disease causation (i.e. etiology) may be attributed to pathogens, environmental stressors, or a combination of biotic and abiotic factors. Biotic diseases are caused by pathogenic microorganisms such as viruses, bacteria, fungi, and protists and are often species-specific (Peters 1997) and infectious (Kinne 1980). Abiotic diseases result from both natural and human-induced environmental stressors including change in ambient conditions or exposure to pollutants. Biotic and abiotic diseases are often closely related. Biotic diseases may be associated with environmental stressors that: (1) hinder the resistence of host organisms, (2) promote growth and virulence of pathogens, (3) trigger the pathogenic process, or (4) increase the rate of disease transmission (Kushmaro et al. 1996, 1998, Peters 1997, Toren et al. 1998, Ben-Haim et al. 1999, 2003b, Alker et al. 2001, Banin et al. 2001a, Israely et al. 2001, Ben-Haim & Rosenberg 2002, Kuta & Richardson 2002, Richardson & Kuta 2003). Abiotic diseases may be exacerbated by secondary opportunistic infections (Peters 1997).

Etiologies of 11 diseases affecting scleractinian and gorgonian corals may involve pathogens including bacteria, cyanobacteria, fungi, and protists (Table 4). Ten diseases are associated with abiotic stressors in-

Table 4. Caribbean and Indo-Pacific coral diseases associated with biota. Koch's postulates have been fullfilled for only 5 diseases. See Table 1 for disease definitions

Disease	Biota	Koch's postulates	Source
BBD	*Phormidium corallyticum* (cyanobacterium)	No	Rützler & Santavy (1983)
	Trichodesmium spp. (cyanobacteria)		Frias-Lopez et al. (2002, 2003)
	Cyanobacterium		Cooney et al. (2002), Frias-Lopez et al. (2003)
	Desulvovibrio spp. (bacteria)		Garrett & Ducklow (1975), Schnell et al. (1996), Cooney et al. (2002)
	Beggiatoa spp. (bacteria)		Ducklow & Mitchell (1979b)
	Heterotrophic bacteria		Garrett & Ducklow (1975), Cooney et al. (2002), Frias-Lopez et al. (2002)
	Marine fungus		Ramos-Flores (1983)
WPL II	*Aurantimonas coralicida* (bacterium)	Yes	Richardson et al. (1998a,b), Denner et al. (2002)
SKA	*Petrarca madreporae* (crustacean)	No	Grygier & Cairns (1996)
	Podocotyloides stenometra (trematode)		Cheng & Wong (1974), Aeby (1998)
	Endolithic fungi		Le Champion-Alsumard et al. (1995), Ravindran et al. (2001)
	Aspergillus sydowii (fungus)		Smith et al. (1998), Dube et al. (2002)
	Order Siphonales (algae)		Morse et al. (1977, 1981),
	Entocladia endozoica (algae)		Goldberg et al. (1984)
WBD II	*Vibrio charcharia* (bacterium)	No	Ritchie & Smith (1995a)
WPD	*Serratia marcescens* (bacterium)	Yes	Patterson et al. (2002)
ASP	*Aspergillus sydowii* (fungus)	Yes	Smith et al. (1996), Geiser et al. (1998)
VSB	*Vibrio shiloi* (bacterium)	Yes	Kushmaro et al. (1996, 1997, 1998, 2001), Rosenberg et al. (1998)
VCB	*Vibrio coralliilyticus* (bacterium)	Yes	Ben-Haim & Rosenberg (2002), Ben-Haim et al. (2003a,b)
SEB	*Halofolliculina corallasia* (protozoan)	No	Antonius & Lipscomb (2000)
PLS	*Phormidium valderianum* (cyanobacterium)	No	Ravindran & Raghukumar (2002)
FPS	*Trichoderma* spp. (fungi)	No	Cerrano et al. (2000)
	Clodosporium spp. (fungi)		Cerrano et al. (2000)
	Penicillum spp. (fungi)		Cerrano et al. (2000)
	Humicola spp. (fungi)		Cerrano et al. (2000)
	Ciliate (protozoan)		Cerrano et al. (2000)

Table 5. Caribbean and Indo-Pacific coral diseases associated with abiotic stressors. See Table 1 for disease definitions

Disease	Abiotic stressors	Source
BBD	Elevated temperature	Antonius (1981, 1985a), Rützler et al. (1983), Edmunds (1991), Carlton & Richardson (1995), Kuta & Richardson (2002), Richardson & Kuta (2003)
	Eutrophication	Antonius (1981, 1985a), Kuta & Richardson (2002)
	Sedimentation	Littler & Littler (1996), Bruckner et al. (1997), Frias-Lopez et al. (2002)
	Pollution	Antonius (1985a), Al-Moghrabi (2001)
	Fecal contamination	Frias-Lopez et al. (2002)
SDR	Elevated temperature	Antonius (1977)
	Sedimentation	Antonius (1977)
SKA	Solar UV radiation	Peters et al. (1986), Coles & Seapy (1998)
ASP	Elevated temperature	Alker et al. (2001)
	Sedimentation	Smith et al. (1996), Shinn et al. (2000), Weir et al. (in press)
	Poor water quality	Kim & Harvell (2002)
WPD	Elevated temperature	Patterson et al. (2002)
	Fecal contamination	Patterson et al. (2002)
	Precipitation	Sutherland & Ritchie (in press)
DSD	Elevated temperature	Gil-Agudelo & Garzón-Ferreira (2001)
VSB	Elevated temperature	Kushmaro et al. (1998), Toren et al. (1998), Ben-Haim et al. (1999), Banin et al. (2001a), Israely et al. (2001)
VCB	Elevated temperature	Ben-Haim & Rosenberg (2002), Ben-Haim et al. (2003b)
YBD	Elevated temperature	Riegl (2002)
FPS	Elevated temperature	Cerrano et al. (2000)

cluding temperature extremes, sedimentation, eutrophication, and pollution (Table 5). VSB and VCB are examples of conditions with a combination of abiotic and biotic factors contributing to disease causation. Infections with *Vibrio shiloi* and *V. coralliilyticus* do not occur in the absence of elevated seawater temperature (Kushmaro et al. 1998, Toren et al. 1998, Ben-Haim et al. 1999, 2003b, Banin et al. 2001a, Israely et al. 2001, Ben-Haim & Rosenberg 2002).

An infectious disease is one in which the causal agent can be transmitted from one host individual to another (Kinne 1980, Peters 1997, Stedman 2000). Disease transmission can be either horizontal or vertical. Horizontal disease transmission is the transmission of infectious agents from an infected individual to a susceptible contemporary (Stedman 2000). Vertical disease transmission is the transmission of infectious agents from an infected individual to its offspring (Stedman 2000). There is no evidence to date to associate coral diseases with vertical transmisssion.

Horizontal disease transmisssion can be either direct or indirect. Direct transmission requires contact between infected and uninfected individuals. In terrestrial environments direct transmission may occur via physical contact or respiratory aerosols. For sessile marine invertebrates, including scleractinian, gorgonian, and hydrozoan corals, direct physical contact only occurs between close neighbors. Inanimate or living agents are required for indirect transmission. Vectors are living agents that transmit pathogens (Stedman 2000) and in the coral reef environment may include predatory and/or herbivorous arthropods, annelids, mollusks, echinoderms, and fishes. The only known vector for a coral disease is the marine fireworm *Hermodice carunculata*, which transmits *Vibrio shiloi*, the causal agent of VSB, to its coral host (Sussman et al. 2003). Contaminated water is an inanimate mode of indirect disease transmission and waterborne pathogens are significant in marine ecosystems.

Infection begins with invasion and multiplication of microorganisms in host tissues and may result in cellular injury. Infection does not always impair the host and therefore is not synonymous with disease (Stedman 2000). For instance, intimate associations between 2 different genetic entities such as host and microorganism are known as symbioses (living together) and range from parasitism to commensalism. A parasite is an organism that grows in or on a host and causes harm to the host (Kinne 1980). A pathogen is a parasite that causes damage to a host, resulting in disease and possibly mortality (Peters 1997). All biotic diseases are symbiotic relationships between a host and a pathogen (Kinne 1980).

Pathogenesis is the process by which infection leads to disease (Stedman 2000). Virulence is the capacity of a pathogen to cause disease (i.e. degree of pathogenicity, Stedman 2000) and is influenced by the interaction between the host, the pathogen, and the environment (Peters 1997). Virulence factors affecting the severity of coral diseases are poorly understood. However,

elevated seawater temperature increases the virulence of *Vibrio shiloi* (Kushmaro et al. 1998, Banin et al. 2001a), *V. coralliilyticus* (Ben-Haim & Rosenberg 2002), *Aspergillus sydowii* (Alker et al. 2001) and the BBD microbial consortium (Table 5, Kuta & Richardson 2002, Richardson & Kuta 2003). Virulence factors affecting VSB include both heat-stable and heat-sensitive toxins that target the zooxanthellae and play a role in pathogenesis (Rosenberg et al. 1998, Ben-Haim et al. 1999).

Susceptibility is the capacity of a host to become infected (Kinne 1980). Host susceptibility may vary between species and within species or individuals and according to environmental stressors, nutrition, genetics, age, and developmental stage (Kinne 1980, Peters 1997). Resistance is a measure of susceptibility of a host to an invading organism, i.e. the ability of an organism to maintain its immunity to or to counteract a disease agent (Kinne 1980, Stedman 2000). Resistance and susceptibility of corals can vary with health status (Kim et al. 2000a) and size class (Dube et al. 2002, Kim & Harvell 2002, Nugues 2002) of the host. Corals are likely more susceptible to (i.e. less resistant to) disease when they are exposed to environmental stressors including sub- and supra-optimal temperature and salinity levels, or poor water quality associated with anthropogenic disturbances including eutrophication, sedimentation, and pollution (Mitchell & Chet 1975, Ducklow & Mitchell 1979a, Johnston et al. 1981, Glynn et al. 1984, Peters 1984, Hodgson 1990, Richmond 1993, Frias-Lopez et al. 2002). Physical stressors such as temperature-induced coral bleaching may also promote disease susceptibility (Kushmaro et al. 1997, Harvell et al. 1999).

Epizootiology is the study of the occurrence, distribution, and control of a disease in an animal population (Stedman 2000). The term is synonymous with epidemiology in human populations (Stedman 2000). Incidence is the number of individuals with new cases of a disease during a specified time period in a specified population (Stedman 2000). Prevalence is the number of cases of a disease in a population at a specific time (Stedman 2000). An epizootic is analogous to an epidemic in human populations and is defined as: (1) a disease occurence with a frequency in excess of the expected frequency in an animal population during a given time interval (Stedman 2000) or (2) an outbreak of an infectious animal disease within a localized region (Kinne 1980). Epizootics may result from: (1) introduction of a new pathogen into a susceptible population, (2) increase in pathogen numbers or virulence, or (3) lowered resistance of the host population (Peters 1997). Corals and other marine organisms of the Caribbean have sustained epizootics in recent years. Two separate epizootics affecting the sea fans *Gorgonia ventalina* and *G. flabellum* have occurred since the 1980s (Garzon-Ferreira & Zea 1992, Nagelkerken et al. 1997b), and both have been attributed to ASP (Smith et al. 1996, Geiser et al. 1998). An epizootic affecting the long-spined sea urchin *Diadema antillarum* occurred in 1983 and resulted in catastrophic reductions in urchin populations (Lessios et al. 1984). WBD (Type I) and WPD epizootics, which began in the mid-1970s and the mid-1990s, respectively, have decimated populations of acroporid corals on Caribbean coral reefs (Gladfelter 1982, Bythell & Sheppard 1993, Aronson & Precht 1997, 2001, Aronson et al. 1998, Patterson et al. 2002).

PROVING DISEASE CAUSATION

Since the 19th century, it has been generally accepted that in order to prove disease causation by a biotic agent, Koch's postulates must be fulfilled (Koch 1882). Koch's postulates require that: (1) the putative pathogen be found in every diseased individual, (2) the putative pathogen be isolated from a diseased individual and grown in pure culture, and (3) the disease be induced in experimental organisms by transferring the pathogen from the culture (Koch 1882). A fourth postulate, that: (4) the same pathogen be isolated from the experimental organism after the disease develops, was later added to Koch's list but was not required by Koch himself. Limitations of the postulates were immediately apparent, and Koch was never able to fulfill his own formulation for disease causation for cholera or leprosy. The accepted etiologic agent of leprosy, *Mycobacterium leprae*, remains unproven by fulfillment of Koch's postulates (Fredericks & Relman 1996).

Although Koch's postulates have elucidated the etiologies of countless diseases, this method of proving disease causation has numerous limitations. Koch's third postulate cannot ethically be fulfilled for fatal diseases that exclusively affect humans (e.g. HIV). Koch's postulates cannot be fulfilled according to the strict definition of the procedure for diseases that: (1) are caused by unculturable bacteria, fungi, or viruses (2) are caused by a consortium of microorganisms, (3) are caused by abiotic stressors, (4) require a vector or a carrier state, (5) cause subclinical or latent infection, or (6) cause injury through systemic attack via virulence factors such as toxins (Fredericks & Relman 1996, US EPA 2000). In addition, Koch's postulates ignore the classic paradigm of disease causation through pathogen interaction with host and environment. Fulfillment of Koch's postulates unequivocally demonstrates disease causation by a specific pathogen; however, failure to fulfill these postulates does not eliminate the possibility that: (1) the putative pathogen

does in fact cause the disease, nor that (2) the suspected pathogen is a commensal organism (Fredericks & Relman 1996).

Several scientists have formulated alternative postulates for disease causation that take into account the limitations of those established by Koch (Rivers 1937, Huebner 1957, Hill 1965, Johnson & Gibbs 1974, Evans 1976, Falkow 1988, Fredericks & Relman 1996). These revisions of Koch's postulates address viral etiology (Rivers 1937, Huebner 1957, Johnson & Gibbs 1974, Evans 1976), carcinogenesis (Evans 1976), antibody response (Huebner 1957, Evans 1976), epidemiology (Huebner 1957, Evans 1976), genetic diseases (Falkow 1988), abiotic diseases (US EPA 2000), and molecular techniques for determining disease causation (Fredricks & Relman 1996).

When Koch's 19th century postulates cannot be fulfilled due to characteristics of a disease that may be attributed to the nature of its etiology or the mechanisms of disease causation, then 21st century techniques can be employed to identify disease-causing microbes (Fredricks & Relman 1996, Ritchie et al. 2001). The genotype-based technique of 16S and 18S rRNA gene sequence amplification can be used to identify putative pathogens and to generate specific probes and primers for use with *in situ* nucleic acid hybridization. *In situ* hybridization identifies a putative pathogen within diseased tissues. Nucleic-acid amplification techniques are highly sensitive and may also target microbes that are insignificant to disease causation (e.g. commensals or contaminants). However, these techniques provide a means of associating putative pathogens with diseased tissues (Fredricks & Relman 1996).

Limitations of Koch's postulates have quickly become evident in the emerging field of coral disease etiology. Koch's postulates have been successfully fulfilled for only 5 (WPL II, WPD, ASP, VSB, VCB) of the 18 coral diseases identified to date in the global oceans (Table 4; Kushmaro et al. 1996, 1997, 1998, 2001, Smith et al. 1996, Geiser et al. 1998, Richardson et al. 1998a,b, Rosenberg et al. 1998, Ben-Haim & Rosenberg 2002, Patterson et al. 2002, Ben-Haim et al. 2003a,b, Denner et al. 2003). Attempts to fulfill Koch's postulates have been unsuccessful for other coral diseases. Although a microbial consortium, dominated by cyanobacteria, is widely accepted as the causal agent of BBD, Koch's postulates have not been, and technically cannot be, fulfilled for this disease. Koch's postulates assume that a single microorganism, which can be grown in pure culture, causes a disease. In the case of BBD, a microbial consortium is thought to be required to induce disease (Carlton & Richardson 1995, Richardson et al. 1997). WBD II is always associated with *Vibrio charcharia*, but attempts to fulfill Koch's postulates with this bacterium have been unsuccessful to date (Ritchie & Smith 1995a).

Molecular techniques have the potential to increase our knowledge of coral disease processes that cannot be understood through the use of 19th century criteria for disease causation (Ritchie et al. 2001, Bythell et al. 2002, Cooney et al. 2002, Frias-Lopez et al. 2002, 2003). Community DNA can be isolated from coral tissue or its associated surface mucopolysaccharide layer (SML). Both diseased and apparently healthy corals have a bacterial community associated with SML, and this bacterial community is known to shift under conditions of stress (e.g. disease and bleaching, Ritchie & Smith 1995a,b). Total community DNA from SML and coral tissue can be isolated, amplified, cloned, and compared to known sequences in a database e.g. GenBank). A phylogenetic tree can then be constructed and used to identify the evolutionary relationships of putative pathogens. Although these molecular techniques do not prove disease causation, their rapid and broad screening can establish an association between potential pathogens and disease, aiding in understanding of the infectious disease processes (Ritchie et al. 2001). To make significant advances in understanding coral disease etiology, alternative criteria for disease causation, in addition to those postulated by Koch, need to be developed, utilized, and accepted by coral disease researchers.

GLOBAL CORAL DISEASES

Black band

Black band disease (BBD; Fig. 3A) affects corals worldwide (Rützler & Santavy 1983, Antonius 1985a, 1988, Edmunds 1991, Carlton & Richardson 1995, Littler & Littler 1996, Miller 1996, Bruckner & Bruckner 1997ab, Bruckner et al. 1997, Green & Bruckner 2000, Al-Moghrabi 2001, Dinsdale 2002). BBD is characterized by a darkly pigmented microbial mat, which forms a band (1 to 30 mm wide and ca. 1 mm thick) that separates living tissue from recently denuded skeleton (Fig. 3A, Rützler & Santavy 1983, Carlton & Richardson 1995).

The microbial consortium that composes the band is dominated by a filamentous cyanobacterium, the identity of which has long been believed to be *Phormidium corallyticum* (Rützler & Santavy 1983). However, recent studies utilized molecular techniques to characterize the BBD consortium, and, while a single cyanobacterium species was associated with the disease, this cyanobacterium was not a member of the genus *Phormidium* (Table 4; Cooney et al. 2002, Frias-Lopez et al. 2002, 2003). The 16S rRNA gene sequencing

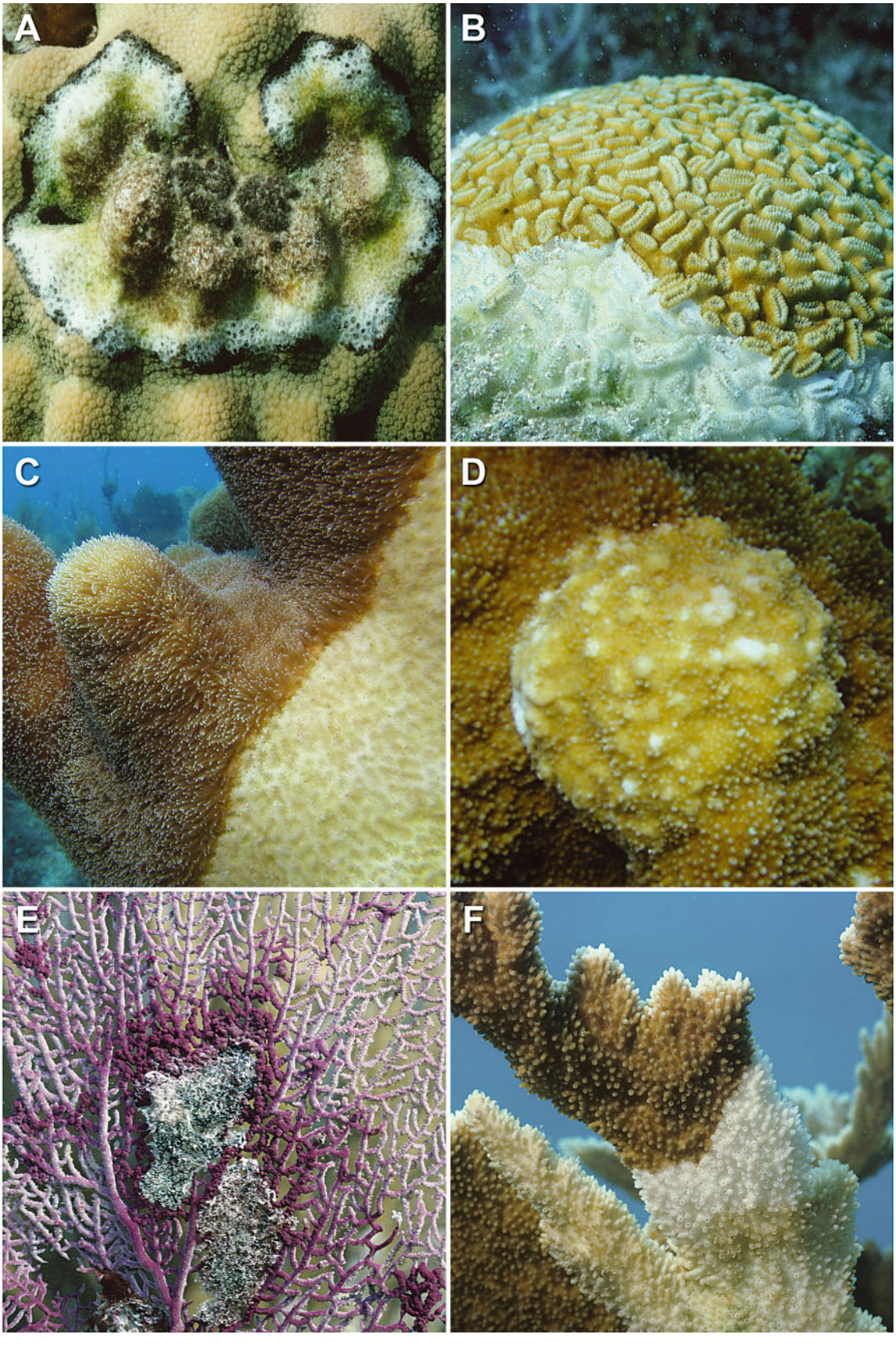
A
B
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Fig. 3. Caribbean coral diseases: (A) black band (BBD) on *Montastraea annularis* complex; (B) white plague Type II (WPL II) on *Dichocoenia stokesi*; (C) WPL II on *Dendrogyra cylindrus*; (D) skeletal anomaly (SKA) on *Acropora palmata*; (E) aspergillosis (ASP) on *Gorgonia* sp.; (F) white band Type I (WBD I) on *A. palmata*. Photographs by J.W.P. and C.T.

identified at least 3 different taxa of cyanobacteria associated with BBD and determined that these taxa vary between the Caribbean and Indo-Pacific (Frias-Lopez et al. 2003). In the Caribbean, 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). In the Indo-Pacific, the BBD cyanobacterium is most closely related to the genus *Trichodesmium* (Frias-Lopez et al. 2003), and this genus (specifically *T. tenue*) has also been isolated from BBD mats in the Caribbean (Frias-Lopez et al. 2002).

Since 16S rRNA gene sequencing did not conclusively identify the cyanobacteria associated with BBD, but rather provided a most homologous match (Frias-Lopez et al. 2003), there is currently a discussion about the identitiy of the BBD cyanobacteria. It is important to note that the only 2 studies targeting the physiology of BBD cyanobacteria in the laboratory (Taylor 1983, Richardson & Kuta 2003) both used cultures isolated from BBD that contained cyanobacteria identified as *Phormidium corallyticum* based on the morphology of this species (Rützler & Santavy 1983). Future research will determine whether or not *P. corallyticum* is an essential component of the BBD microbial consortium.

Other microbes identified in the BBD consortium include sulfate-reducing bacteria *Desulfovibrio* spp. (Garrett & Ducklow 1975, Schnell et al. 1996, Cooney et al. 2002), sulfide oxidizing bacteria *Beggiatoa* spp. (Ducklow & Mitchell 1979b), a multitude of heterotrophic bacteria (Garrett & Ducklow 1975, Cooney et al. 2002, Frias-Lopez et al. 2002), and a marine fungus (Ramos-Flores 1983). Two molecular studies carried out to investigate BBD (Cooney et al. 2002, Frias-Lopez et al. 2002) each found over 50 bacterial species associated with BBD. These included a wide assortment of proteobacteria, *Cytophega* sp., and an α-proteobacterium closely related to the pathogen that causes juvenile oyster disease. The disease consortium, and not a single microorganism alone, is thought to be required for disease causation (Carlton & Richardson 1995, Richardson et al. 1997). Koch's postulates have not been fulfilled with any component of the consortium (Carlton & Richardson 1995).

BBD affects 19 (of 66) Caribbean shallow-water scleractinian species (Table 2; Rützler et al. 1983, Kuta & Richardson 1996, Garzón-Ferreira et al. 2001, Porter et al. 2001, Wheaton et al. 2001) and 45 (of approximately 400) Indo-Pacific scleractinian species (Table 3; Antonius 1985a,b, Miller 1996, Green & Bruckner 2000, Al-Moghrabi 2001, Riegl 2002, Frias-Lopez et al. 2003). Six Caribbean gorgonian species are also affected by BBD (Table 2; Antonius 1981, 1985b, Feingold 1988). Caribbean corals most susceptible to BBD include *Diploria strigosa*, *D. labyrinthiformis*, *Montastraea annularis*, *M. cavernosa*, *M. faveolata*, *M. franksi* and *Colpophyllia natans* (Antonius 1981, Ramos-Flores 1983, Rützler et al. 1983, Kuta & Richardson 1996). These corals are massive species and the dominant reef-builders. *Acropora cervicornis*, *A. prolifera*, and *Porites porites* appear resistant to BBD infections (Antonius 1981). BBD was recently reported affecting *A. palmata* in the Colombian Caribbean (Garzón-Ferreira et al. 2001). Prior to this report *A. palmata* was thought to be resistant to the disease. While BBD is rarely reported on acroporids in the Caribbean, this genus is among the most susceptible to BBD on the Great Barrier Reef, Australia (Miller 1996, Dinsdale 2002). A BBD epizootic affecting faviid corals occurred at Looe Key Reef, Florida in 1986 (Peters 1993). BBD most often affects fewer than 1% of coral colonies on any reef area at any one time (Edmunds 1991, Kuta & Richardson 1996), but the susceptibility of major frame-building species greatly enhances the threat that BBD poses to the reef community.

Infection with BBD usually begins on upper surfaces of a coral colony (Antonius 1981) as a small darkly-pigmented patch (1 to 2 cm diameter). The patch quickly forms a ring, the circumference of which rapidly increases as the band migrates horizontally across the coral. As the microbial mat migrates, it kills all tissue and leaves behind bare skeleton (Antonius 1981, Carlton & Richardson 1995). Horizontal movement of the band is greatest at the front of the band (adjacent to living tissue) during the day, and at the back of the band (adjacent to dead skeleton) at night. This migration pattern results in a widening of the microbial mat during the day and a contraction at night (Richardson 1996).

BBD progresses at an average rate of 3 mm d^{-1}, but is capable of advancing up to 1 cm d^{-1} (Antonius 1981, 1985a, Edmunds 1991, Carlton & Richardson 1995). This rapid rate of tissue loss, coupled with the slow growth rate of scleractinian corals, denudes living coral tissue quickly and allows for complete colony mortality. However, BBD may disappear before complete colony mortality occurs (Carlton & Richardson 1995). This cessation of BBD most often occurs with the onset of lower seawater temperatures (Carlton & Richardson 1995).

BBD has been widely reported as a seasonal phenomenon, with most active infections occurring during late summer and fall, and cessation occurring during winter (Antonius 1981, 1985a, Edmunds 1991, Carlton & Richardson 1995). Seasonality of BBD is related to summer seawater temperatures in excess of 25°C (Rützler et al. 1983, Edmunds 1991, Kuta & Richardson 2002, Richardson & Kuta 2003). When BBD disappears in the winter, remaining living coral tissue survives (Antonius 1981, Carlton & Richardson 1995). However, seasonal reappearance of warm seawater coincides with observations of reinfection, and reinfection makes complete colony mortality possible (Carlton & Richardson 1995, Kuta & Richardson 1996). It is important to note that BBD has been reported year-round, even at seawater temperatures as low as 20°C (Kuta & Richardson 1996), and that the cyanobacteria associated with BBD are capable of photosynthesis at temperatures as low as 18 and 20°C (Taylor 1983, Richardson & Kuta 2003).

BBD has been proposed to be correlated with other environmental and physiological stressors, including terrestrial runoff (Littler & Littler 1996, Bruckner et al. 1997, Frias-Lopez et al. 2002), coral overgrowth by algae (Bruckner et al. 1997), eutrophication (Antonius 1981, 1985a, Kuta & Richardson 2002), and pollution (Antonius 1985a, Al-Moghrabi 2001), including human fecal contamination (Frias-Lopez et al. 2002, Table 5). However, very little quantitative data, and no definitive results, have supported a positive correlation. Pollution has been implicated in extending depth range, frequency, and severity of BBD (Antonius 1985a). BBD has been frequently reported from shallow depths (Rützler et al. 1983, Antonius 1985a, Kuta & Richardson 2002) and reefs with low coral species diversity (Bruckner & Bruckner 1997b, Bruckner et al. 1997, Kuta & Richardson 2002).

The mechanism by which BBD kills coral tissue is directly linked to dynamics of the microbial mat community, which produces a vertical zonation of oxygen and sulfide microenvironments that migrates on a diel basis. The dominant constituent of BBD, the cyanobacteria, forms the scaffolding of the band and is always present throughout the band. The cyanobacteria undergo oxygenic photosynthesis during the day, producing an oxygen supersaturated oxic zone in the top $\frac{1}{2}$ to $\frac{2}{3}$ of the band. The cyanobacteria adapt to the high sulfide environment of the disease band by performing oxygenic photosynthesis in the presence of sulfide (Richardson & Kuta 2003). The anoxic base of the band is dominated by sulfate-reducing bacteria *Desulfovibrio* spp. The oxic/anoxic interface contains sulfide-oxidizing bacteria *Beggiatoa* spp., and this zone migrates vertically on a diel basis in response to changes in light intensity (Viehman & Richardson 2002) and photosynthetic activity occurring within the band (Carlton & Richardson 1995, Richardson et al. 1997). Under low light conditions (e.g. shade, darkness) the surface community of BBD is dominated by cyanobacteria, but when light intensity increases, *Beggiatoa* spp. migrate to the band surface (Viehman & Richardson 2002). At night, in absence of oxygen production, *Desulfovibrio* spp. undergo sulfate reduction, increasing sulfide concentrations in the band. As a result, sulfide is present throughout the band at night and the oxic/anoxic interface migrates to the band surface (Carlton & Richardson 1995, Richardson et al. 1997).

Presence of sulfide and anoxia at the base of the band (adjacent to coral tissue) is thought to be the cause of tissue lysis and death (Carlton & Richardson 1995). BBD-induced coral mortality presumably releases inorganic nutrients (NH_4^+ and PO_4^{3-}) that support cyanobacterial photosynthesis. Furthermore, because the disease band directly overlies coral tissue that is being degraded, nutrients supplied by tissue mortality diffuse directly into the band, providing concentrated nutrients to the microbial consortium, and fueling their growth and reproduction (Carlton & Richardson 1995). This process of tissue death may serve as the source for elevated nitrite levels associated with seawater immediately surrounding BBD-affected corals (Kuta & Richardson 2002).

The mechanism of BBD transmission remains unknown. However, BBD may be infectious (Edmunds 1991, Kuta & Richardson 1996) and transmitted in the water column (Kuta & Richardson 1996, Bruckner et al. 1997). Sediment patches on the surfaces of apparently healthy corals may serve as reservoirs of the cyanobacteria associated with BBD (Richardson 1997).

Disease signs similar to BBD were reported in 1983 (Rützler et al. 1983) and documented as a new disease termed red band (Richardson 1993). Red band reportedly affects *Gorgonia ventalina* in Belize, Puerto Rico and the Florida Keys (Santavy & Peters 1997), and *Montastraea annularis* and *M. faveolata* in the Colombian Caribbean (Garzón-Ferreira et al. 2001). Red band is characterized by a red-brown to brown-black microbial mat that forms a migrating band that separates recently denuded skeleton from living tissue (Santavy & Peters 1997). The condition was considered not to be BBD because *Phormidium corallyticum*, accepted at the time to be the dominant component of the BBD consortium, was not present in red band (Rützler et al. 1983). Instead, red band was dominanted by other species of cyanobacteria, identified as *Schizothrix calcicola*, *S. mexicana* (Rützler et al. 1983) and *Oscillatoria* spp. (Richardson 1993). The RBD microbial consortium may include other cyanobacteria, heterotrophic bacteria, the sulfur-oxidizing bacterium *Beggiatoa* sp., and the nematode *Araeolaimus* sp. (Santavy &

Peters 1997). The recent discovery that BBD is associated with at least 3 different taxa of cyanobacteria (Frias-Lopez et al. 2003) and the lack of new reports of red band disease since the early 1990s, suggest that red band is not a distinct disease, but rather BBD.

White plague

White plague (WPL; Fig. 3B,C) has affected Caribbean and Indo-Pacific corals since the late 1970s (Dustan 1977, Richardson et al. 1998a) and early 1980s (Antonius 1985a), respectively. In the Caribbean, the disease reported in the 1970s has been renamed WPL I to distinguish this condition from 2 new diseases with similar disease signs: WPL II (Richardson et al. 1998a) and WPL III (Richardson et al. 2001). WPL II is an infectious biotic disease (Richardson et al. 1998a) caused by a new genus and species of bacterium, *Aurantimonas coralicida* (Table 4; Richardson et al. 1998b, Denner et al. 2003). Pathogenesis and transmission of WPL II are not understood. Histopathology of WPL I affected tissues shows necrosis at lesion boundaries (Peters 1984, Bythell et al. 2002) accompanied by dense clusters of coccoid bacteria that do not resemble the rod-shaped *A. coralicida* from WPL II infections (Bythell et al. 2002). Similarly, 16S rDNA sequencing indicates that *A. coralicida* is not associated with a WPL-like disease affecting Caribbean corals (Pantos et al. 2003). These studies suggest that there is indeed more than one etiologic agent associated with the various WPL diseases in the Caribbean. The causal agent(s) of WPL I and III are unknown.

In the Indo-Pacific, WPL-like (WPL-L) disease signs have been reported. Authors that documented WPL-L disease in the Indo-Pacific referred to the disease as WBD (Antonius 1985a, Coles 1994, Riegl 2002). Species affected and disease signs reported for the Indo-Pacific (Antonius 1985a, Coles 1994) are indicative of a WPL-L disease. In the future, Indo-Pacific researchers must distinguish between WPL-L and WBD-like diseases, which are characterized, respectively, by a sharp line of tissue loss that progresses across a coral colony and by a ring of tissue loss that progresses up or down acroporid coral branches. WPL-L disease affects 38 Indo-Pacific scleractinian species (Table 3; Antonius 1985a, Coles 1994, Riegl 2002).

WPL I progresses slowly (3.1 mm d^{-1} maximum) and is characterized by a sharp line of tissue loss where healthy tissue is immediately adjacent to recently denuded skeleton (Fig. 3B,C; Dustan 1977). WPL I affects at least 13 Caribbean scleractinian species (Table 2; Dustan 1977, Richardson et al. 1998a).

WPL II progresses rapidly (2 cm d^{-1} maximum) and is characterized by a sharp line of disease progression. At times a narrow band (2 to 3 mm) of bleached tissue separates healthy tissue and bare skeleton, but more commonly the disease line appears the same as for WPL I, i.e. healthy tissue is immediately adjacent to recently denuded tissue (Richardson et al. 1998a,b). Another distinguishing characteristic of WPL II is that infection most often begins at the base of the coral colony and progresses upward in a concentric ring around the entire colony (Richardson et al. 1998a,b). Three major epizootics of WPL II have been reported in South Florida since the mid-1990s (Richardson et al. 1998a). The number of species affected by WPL II is increasing. In 1995, Richardson et al. (1998a,b) reported 17 species of scleractinian corals affected on reefs in the Florida Keys, and by 2000 this number had increased to 32 species (Weil et al. 2002). Highest disease prevalence of WPL II has been recorded for *Dichocoenia stokesi*, with mortality as high as 38% (Richardson et al. 1998a).

WPL III was first documented in 1999 in the northern Florida Keys. WPL III appears to exclusively affect large colonies (3 to 4 m diameter) of *Colpophyllia natans* and *Montastraea annularis* (Table 2). Tissue loss attributed to WPL III is extremely rapid and greatly exceeds loss rates attributed to WPL I and WPL II (Richardson et al. 2001).

Surveys of WPL prevalence conducted in Puerto Rico (Bruckner & Bruckner 1997a) and St. Lucia, West Indies (Nugues 2002) did not distinguish type. However, based on slow rate of disease progression (1.3 mm d^{-1} maximum) measured in St. Lucia (Nugues 2002) and rapid disease progression (1.4 cm d^{-1} maximum) measured in Puerto Rico (Bruckner & Bruckner 1997a) it is likely that the diseases surveyed were WPL I and II, respectively. Highest disease prevalence was recorded for *Diploria labyrinthiformis* (47% of colonies affected) in Puerto Rico (Bruckner & Bruckner 1997a) and for *Montastraea faveolata* (19% of colonies affected) and *Colpophyllia natans* (13% of colonies affected) in St. Lucia (Nugues 2002).

Nugues (2002) observed diseased individuals of 4 scleractinian species, *Isophylastrea rigida*, *Montastraea faveolata*, *Mussa angulosa*, and *Mycetophyllia* sp., not yet documented as susceptible to WPL I (Table 2). Surveys conducted within the FKNMS documented WPL (type not determined) on 14 additional species (Table 2; Porter et al. 2001).

Shut-down reaction

Shut-down reaction (SDR) is a condition that most often affects corals contained in aquaria and is rarely observed in natural coral reef environments. SDR occurs only on wounded corals (e.g. predation, diver

contact) and is always associated with abiotic environmental stressors (e.g. temperature extremes, sedimentation, Table 5; Antonius 1977). SDR begins at and radiates from the interface between wound and healthy tissue. Tissue is sloughed off the affected colony at the rapid rate of 10 cm h^{-1}. Once SDR is triggered, complete colony mortality is inevitable. SDR is contagious, indicating presence of a pathogen, and can be both directly and indirectly transmitted from an infected colony to a stressed, but otherwise apparently healthy colony, via physical contact between neighboring colonies and water transport, respectively (Antonius 1977). SDR cannot be transmitted to unstressed coral colonies (Antonius 1977).

SDR has been experimentally induced in aquaria for 6 Caribbean scleractinian species (Table 2; Antonius 1977). In the Caribbean, SDR has been reported affecting only 3 individual coral colonies in the field; the 2 species affected were *Montastraea annularis* and *Acropora cervicornis* (Table 2; Antonius 1977). Prior to recent reports of SDR in the Red Sea (species affected not reported, Antonius & Riegl 1997, 1998), new cases of the condition had not been documented since the first report in the late 1970s (Antonius 1977).

Skeletal anomalies

Skeletal anomalies include tumors, galls, nodules, and other abnormalities of coral tissue and skeleton. Skeletal anomalies of scleractinian corals have been observed on reefs throughout the world including the Florida Keys (Peters et al. 1986), Netherlands Antilles (Bak 1983), Hawaii (Squires 1965, Cheng & Wong 1974, Hunter & Peters 1993 Grygier & Cairns 1996, Aeby 1998), Guam and Enewetak (Cheney 1975), Oman (Coles & Seapy 1998), Japan (Yamashiro et al. 2000), and the Great Barrier Reef, Australia (Loya et al. 1984).

A tumor is an abnormal tissue proliferation (Sinderman 1990) and, in corals, is often associated with an abnormal skeletal growth (Yamashiro et al. 2000). Tumors result from neoplasia, hyperplasia, or hypertrophy. Neoplasia (neoplasm) is uncontrolled cell proliferation. Hyperplasia and hypertrophy are nonneoplastic controlled cell proliferation and nonneoplastic increase in cell size, respectively (Sinderman 1990). The terms tumor and neoplasia are considered by many to be synonymous (Stedman 2000).

The first suspected neoplasm of a scleractinian coral was documented in 1965 on *Madrepora kauaiensis* in the Hawaiian Islands (Table 3, Squires 1965). This condition, also observed on *M. oculata* (Table 3), has recently been reinterpreted as a polyp hypertrophy characterized by gall formation (i.e. a parasite-induced proliferation of tissues, Grygier & Cairns 1996). These lesions develop on *Madrepora* spp. when the crustacean *Petrarca madreporae*, an obligate endoparasite of corals, invades a normal coral polyp as a larva and matures within the polyp, causing development of an enlarged (hypertrophied) corallite with abnormal septae (Table 4; Grygier & Cairns 1996).

Other skeletal anomalies have been attributed to interactions with foreign organisms in the skeleton (Table 4). *Porites compressa* and *P. lobata* in Kaneohe Bay, Oahu, Hawaii develop grossly visible pink nodules in response to the encystment of the digenetic trematode *Podocotyloides stenometra* within the tentacles of the coral polyps (Table 3; Cheng & Wong 1974, Aeby 1998). The scleractinian corals *P. lobata*, *P. lutea*, *Manicina areolata*, and *Montastraea cavernosa* can detect invasion by endolithic fungi and respond by surrounding the site of fungal penetration within a layer of thickened calcium carbonate produced by hypertrophied calicoblasts (Tables 3 & 4; Le Champion-Alsumard et al. 1995, Ravindran et al. 2001, E. C. Peters pers. comm.). However, this defense mechanism fails to hinder fungal advancement, and, as hyphae penetrate the layer of calcium carbonate repair, the coral repeats the process, resulting in a calcareous skeletal protuberance composed of a number of carbonate layers (Le Champion-Alsumard et al. 1995). Nodules on the gorgonian coral *Gorgonia ventalina* are attributed to both infection with *Aspergillus sydowii*, the causal agent of aspergillosis (Dube et al. 2002), and to infestation with filamentous green algae of the Order Siphonales (Tables 2 & 4; Morse et al. 1977, 1981). The algal nodules are hyperplasias of the axis epithelial cells that produce the endoskeletal gorgonin. Amoebocytes infiltrate the associated mesoglea and encapsulate the algal filaments (Morse et al. 1977, 1981). Aspergillosis-associated nodule formation may be a defense mechanism that sequesters fungal hyphae and limits spread of infection (Smith et al. 1998). Nodules on the gorgonian corals *Pseudoplexaura* spp. are the result of skeletal encapsulation of the marine microalgae *Entocladia endozoica* (Tables 2 & 4; Goldberg et al. 1984).

Peters et al. (1986) and Coles & Seapy (1998) described the only known true neoplasms (tumors) of corals. Peters et al. (1986) observed neoplasms, termed calicoblastic epitheliomas, on *Acropora palmata* in the Florida Keys (Table 2). These calicoblastic epitheliomas result from proliferation of calicoblasts and associated tissues and are characterized by raised (up to 1 cm high), irregularly shaped, smooth, white lumps that develop on all parts of the coral colony. Mean growth rate of tumors is 0.12 mm d^{-1} or 25 to 44 mm yr^{-1} (Peters et al. 1986). Similar calicoblastic epitheliomas affect *A. valenciennesi* and *A. valida* in the Gulf of Oman, Indian Ocean (Table 3; Coles & Seapy 1998).

Coral skeletal anomalies are characterized by: (1) thinning of coral tissue covering anomalies (Peters et al. 1986, Coles & Seapy 1998), (2) increased porosity of coral skeleton (Peters et al. 1986, Coles & Seapy 1998, Yamashiro et al. 2000), (3) loss of mucous secretory cells and nematocysts (Peters et al. 1986, Coles & Seapy 1998), (4) loss of zooxanthellae (Bak 1983, Peters et al. 1986, Coles & Seapy 1998, Yamashiro et al. 2000), (5) loss, reduction, or degeneration of normal polyp structures (Bak 1983, Peters et al. 1986, Coles & Seapy 1998, Yamashiro et al. 2000), and (6) reduced fecundity (Yamashiro et al. 2000).

Skeletal anomalies pose a serious threat to affected corals. Loss of the primary defense mechanism, mucous secretory cells, inhibits removal of foreign material from the coral surface, contributing to cell death and increasing susceptibility to invasion by filamentous algae. Porous skeletons may be more susceptible to storm-related damage (Peters et al. 1986). Loss of zooxanthellae reduces fecundity, skeletal growth, calcification rates, and nutrition (Bak 1983, Porter et al. 1989, Brown 1997a). Polyp destruction limits reproductive capacity (Peters et al. 1986, Yamashiro et al. 2000).

Skeletal anomalies (SKA; Fig. 3D) affect 16 Caribbean (Table 2) and 24 Indo-Pacific (Table 3) scleractinian species, 1 Caribbean hydrozoan, and at least 5 species of Caribbean gorgonians (Table 2; Squires 1965, Cheney 1975, Morse et al. 1977, 1981, Bak 1983, Goldberg et al. 1984, Loya et al. 1984, Peters et al. 1986, Hunter & Peters 1993, Le Champion-Alsumard et al. 1995, Grygier & Cairns 1996, Coles & Seapy 1998, Green & Bruckner 2000, Yamashiro et al. 2000, Ravindran et al. 2001, Dube et al. 2002). Acroporids appear to be the most susceptible to neoplasia (Peters et al. 1986, Coles & Seapy 1998), and this may be due to the rapid growth rates of this genus (Peters et al. 1986). *Acropora palmata* is capable of linear extension rates as high as 47 to 99 mm yr^{-1} (Gladfelter et al. 1978).

With the exception of microorganism-induced nodule or gall formation (Cheng & Wong 1974, Morse et al. 1977, 1981, Goldberg et al. 1984, Le Champion-Alsumard et al. 1995, Grygier & Cairns 1996, Aeby 1998, Ravindran et al. 2001, Dube et al. 2002), the etiology of coral skeletal anomalies is unknown (Peters et al. 1986, Coles & Seapy 1998, Yamashiro et al. 2000). Parasitic and commensal organisms have been ruled out as potential causal agents of the skeletal anomalies described by Peters et al. (1986) in the Florida Keys and by Yamashiro et al. (2000) in Japan. Solar UV radiation has been hypothesized as a possible initiator of neoplasia formation (Table 5; Peters et al. 1986, Coles & Seapy 1998).

CARIBBEAN CORAL DISEASES

Aspergillosis

Mass mortalities of the sea fans *Gorgonia ventalina* and *G. flabellum* were reported throughout the Caribbean during the 1980s (Garzón-Ferreira & Zea 1992). A second epizootic affecting *Gorgonia* spp. began in 1995 (Nagelkerken 1997ab, Slattery 1999), which was less virulent but more widespread than the 1980s epizootic (Nagelkerken 1997a,b).

Both epizootics have been attributed to the fungus *Aspergillus sydowii* (Table 4; Smith et al. 1996, Geiser et al. 1998). *Aspergillus* disease, termed aspergillosis (ASP; Fig. 3E), destroys living tissue and degrades skeletal framework (Nagelkerken et al. 1997b). In addition to *Gorgonia ventalina* and *G. flabellum*, ASP signs have been observed on 6 additional gorgonian species from 5 genera (Kim et al. 2000b, Weil et al. 2002; Table 2). However, Koch's postulates have only been fulfilled, establishing *A. sydowii* as the causative agent, for disease cases affecting *G. ventalina* and *G. flabellum* (Smith et al. 1996).

ASP lesions are characterized by recession of rind tissue (coenenchyme) exposing the internal axial skeleton. *Aspergillus sydowii* hyphae are embedded in living tissue at the receding edge of the lesion (Smith et al. 1996). Lesions are often circumscribed by a purple halo indicative of an abundance of purple sclerites (Kim et al. 1997, Smith et al. 1998, Slattery 1999). Purple sclerite-dense nodules often erupt on affected sea fans (Kim et al. 1997, Smith et al. 1998, Dube et al. 2002). Sclerite recruitment and nodule formation may be methods of defense that sequester fungal hyphae and limit spread of infection (Smith et al. 1998). Mechanisms by which *A. sydowii* produces tissue degradation and nodule formation remain unknown, but virulence of the pathogen is known to increase with elevated seawater temperatures (30°C, Table 5; Alker et al. 2001).

The genus *Aspergillus* is not commonly found in marine environments and most often inhabits terrestrial soils. However, *Aspergillus* spp. can easily cope with the salinity of seawater (Kendrick et al. 1982) and have been isolated from marine environments (Muntanola-Cvetkovic & Ristanovic 1980, Kendrick et al. 1982, Ravindran et al. 2001). *A. sydowii* has been shown to bioerode living stony corals (Kendrick et al. 1982).

Aspergillus sydowii is a terrestrial fungus. Delivery of *A. sydowii* to the marine environment may be associated with either local sediment runoff (Smith et al. 1996) or long distance transport (Table 5, Shinn et al. 2000). If local runoff is the source, then ASP may be linked to anthropogenic disturbance. Shinn et al.

(2000) hypothesize that *A. sydowii*, and perhaps other coral disease-causing pathogens, are transported to the western Atlantic in African dust air masses. *A. sydowii* has been cultured from spores collected in the US Virgin Islands during African dust storm events. These *A. sydowii* isolates, when inoculated onto healthy sea fans, produced ASP signs (Weir et al. in press).

Terrestrial sources are a possible mode of primary transmission of *Aspergillus sydowii* hyphae and/or spores to unaffected sea fans in the marine environment. *A. sydowii* germinates but does not sporulate on sea fans. Hyphae must break free from an infected gorgonian and reach the surface of the water to produce spores (G. W. Smith pers. comm.). Secondary transmission of ASP from infected to uninfected sea fans may occur through: (1) direct physical contact with an infected individual, i.e. a diseased sea fan may brush against a close neighbor (Smith et al. 1996, Jolles et al. 2002), (2) transport of fungal hyphae in the water column (Jolles et al. 2002), or (3) transport of fungal spores (produced at the sea surface from hyphae released from diseased sea fans) in the water column (G. W. Smith pers. comm.).

Incidence and prevalence of ASP are greater at protected than at exposed sites and increase with depth in areas with low to moderate wave action, indicating that the more frequent mechanical swaying of colonies that occurs in shallow and more exposed areas may decrease sea fan susceptibility to disease (Nagelkerken 1997a). ASP is prevalent in the Florida Keys, affecting 43% of *Gorgonia ventalina* colonies Keys-wide (Kim & Harvell 2002). Poor water quality (i.e. increased turbidity and increased chl *a*) may play a role in the impact of ASP on sea fan populations (Table 5). Kim & Harvell (2002) found that severity of ASP in the Florida Keys was greatest near the city of Key West. With approximately 25500 residents and a high seasonal influx of tourists, Key West is by far the most populous town in the Florida Keys (US BOC 2000). Runoff of nutrients and pollutants into the marine environment is likely higher near Key West than in other less populated areas in the Florida Keys.

White band

White band disease (WBD; Fig. 3F) has affected Caribbean scleractinian corals since the late 1970s (Antonius 1985a, Bythell & Sheppard 1993). The disease, as first described by Gladfelter (1982), has been renamed WBD I to distinguish the condition from WBD II (Ritchie & Smith 1998). WBD I and II exclusively affect branching acroporid corals. While WBD I affects both *Acropora palmata* and *A. cervicornis* Caribbean-wide (Gladfelter 1982, Peters 1984), WBD II has been reported only from the Bahamas, exclusively affecting *A. cervicornis* (Ritchie & Smith 1998; Table 2).

WBD Type I has been implicated as the principal cause of mass mortalities of *Acropora cervicornis* and *A. palmata* that occurred in the 1980s and 1990s (Gladfelter 1982, Bythell & Sheppard 1993, Aronson & Precht 1997, 2001, Aronson et al. 1998, 2002). On most reefs, loss of acroporids was accompanied by an ecological phase shift from a coral-dominated to an algal-dominated reef (Hughes 1994). However, on the Belizean Barrier Reef, the grazing urchin *Echinometra viridis* consumed fleshy and filamentous macroalgae and allowed for a wide scale (at least 500 km^2) coral community shift (Aronson et al. 2002). Following the WBD epizootic of the late-1980s, the previously dominant coral *A. cervicornis* was replaced by the thin-leaf lettuce coral *Agaricia tenuifolia* (Aronson & Precht 1997, Aronson et al. 2002). Examination of the fossil record indicates that the wide-scale *Acropora*-to-*Agaricia* shift is unprecedented in the last 3800 yr (Aronson & Precht 1997, 2001, Aronson et al. 2002). This evidence suggests that WBD is an emergent disease and not a natural cyclic phenomenon that has occurred on Caribbean reefs in the past.

WBD I progresses rapidly (2 cm d^{-1} maximum) and has the potential to cause extensive mortality (Antonius 1981, Gladfelter 1982, Peters et al. 1983). The disease is characterized by a white band of recently denuded skeleton adjacent to a necrotic front of normally pigmented living tissue (Fig. 3F; Gladfelter 1982, Peters et al. 1983). WBD I develops at the base of a coral colony or branch and progresses upward toward branch tips in a concentric ring (Gladfelter 1982).

WBD II was first documented in 1993 in the Bahamas (Ritchie & Smith 1995a, Ritchie & Smith 1998). WBD II is distinguished from WBD I by a band (2 to 20 cm wide) of living bleached tissue separating denuded skeleton from normally pigmented tissue. The bleaching of the tissue progresses more rapidly than does the margin of necrotic tissue and can arrest, allowing the necrotic margin to catch up to normal tissue. When this occurs, WBD II resembles WBD I (Ritchie & Smith 1998) and the 2 diseases cannot be distinguished from a single observation in the field. Like WBD I, WBD II can develop at the base of a coral colony and progress upward, but WBD II is also capable of developing at tips of branches and progressing downward. When the disease begins at branch tips, tissue loss can be accompanied by skeletal degradation, i.e. dissolution and loss of branch tips (Ritchie & Smith 1998).

The causative agents of WBD I and II are unknown, however, efforts have been made to determine etiologies of these conditions. Tissues of WBD I-diseased and apparently healthy *Acropora palmata* and *A. cer-*

vicornis at St. Croix, US Virgin Islands and at Bonaire were found to contain Gram-negative bacterial aggregates. Aggregates were more abundant in diseased corals than in apparently healthy corals. However, other apparently healthy and diseased acroporids do not contain aggregates (Peters et al. 1983). Examination of *A. cervicornis* from the Bahamas and the Florida Keys revealed WBD I-affected colonies both with and without bacterial aggregates (Peters 1984). Thus, the role of bacterial aggregates in WBD I is uncertain. Histopathology of WBD I tissues shows no signs of necrosis or clustering of microorganisms (Peters et al. 1983, Bythell et al. 2002).

WBD II is always associated with the bacterium *Vibrio charcharia* (Table 4; Ritchie & Smith 1995a). Attempts to fulfill Koch's postulates with *V. charcharia* were unsuccessful, and therefore the significance of the bacterium to the etiology of WBD II remains unknown.

White pox

White pox disease (WPD; Fig. 4A,B), also termed acroporid serratiosis (Patterson et al. 2002) and patchy necrosis (Bruckner & Bruckner 1997a), was first documented in 1996 on reefs off Key West, Florida (Holden 1996). WPD has since been observed throughout the Caribbean (Porter et al. 2001, Rodríquez-Martínez et al. 2001, Santavy et al. 2001, Patterson et al. 2002). The disease exclusively affects *Acropora palmata* (Table 2), and is caused by the common fecal enterobacterium *Serratia marcescens* (Table 4; Patterson et al. 2002).

Serratia marcescens is a Gram-negative bacterium classified as a coliform and a member of the Enterobacteriaceae family. It is found in feces of humans and other animals and in water and soil (Grimont & Grimont 1994). The prevalence of *S. marcescens* in the marine environment is unknown. However, this bacterium has been found in the marine environment in sewage-polluted estuaries. For example, *S. marcescens* has been linked to disease of white perch *Morone americanus* in the sewage-polluted Back River, Maryland (Baya et al. 1992).

Identification of *Serratia marcescens* as a coral pathogen marked the first time that a common member of the human gut microbiota was shown to be a marine invertebrate pathogen. While *S. marcescens* is ubiquitous, its noted association with human hosts prompts speculation that improperly treated sewage may be associated with white pox disease in corals. Human enteric bacteria and viruses are prevalent in coral SML and other marine environments of the Florida Keys (Griffin et al. 1999, Lipp et al. 2002). The origin and pathogenic mechanisms of the WPD pathogen are unknown (Patterson et al. 2002).

Coral colonies affected by WPD are characterized by irregularly shaped distinct white patches of recently exposed skeleton surrounded by a necrotic front of normally pigmented living tissue (Fig. 4B). Lesions range in area from a few square centimeters to >80 cm^2 and develop simultaneously on all surfaces of the coral colony (Fig. 4A; Patterson et al. 2002). Lesions exhibit tissue loss along the perimeter and increase in area as tissue is lost from the leading edge of infection. Rate of tissue loss is rapid, averaging 2.5 cm^2 d^{-1}, and is greatest during periods of seasonally elevated temperature and rainfall (Table 5). WPD is highly contagious, with nearest neighbors most susceptible to infection (Patterson et al. 2002). The disease spread rapidly within and between reefs in the Florida Keys during the mid-1990s (Porter et al. 2001, Patterson et al. 2002).

WPD has been implicated as the principal cause of mass mortality of *Acropora palmata* within the FKNMS (Patterson et al. 2002). Between 1996 and 2002, average loss of *A. palmata* Keys-wide was 87% (Fig. 1; Patterson et al. 2002, Sutherland & Ritchie in press). Losses of *A. palmata* at Eastern Dry Rocks Reef, FL (24° 27.715′ N, 81° 50.801′ W) between 1994 and 2002, and at Looe Key Reef, FL (24° 33′ N, 81° 24′ W) between 1983 and 2000, were 97 and 93%, respectively (Miller et al. 2002, Patterson et al. 2002, Sutherland & Ritchie in press). These severe population declines of the coral community's most important primary producer and shallow water framework builder have led to the identification of *A. palmata* as a candidate for inclusion on the Endangered Species List (Diaz-Soltero 1999). Diseases of acroporid corals (WPD and WBD) are changing the composition, structure, and function of Caribbean coral reef ecosystems (Hughes 1994, Aronson & Precht 1997, 2001, Aronson et al. 2002, Patterson et al. 2002).

Yellow blotch/band

Yellow blotch/band (YBL; Fig. 4C,D) disease has been reported throughout the Caribbean since 1994 (Santavy & Peters 1997, Santavy et al. 1999, Cervino et al. 2001, Garzón-Ferreira et al. 2001, Toller et al. 2001, Weil et al. 2002). YBL affects 9 scleractinian coral species (Table 2; Garzón-Ferreira et al. 2001), but most often affects *Montastraea annularis* (Cervino et al. 2001). The causal agent of YBL is unknown.

YBL lesions are characterized by circular to irregularly shaped patches (Fig. 4C) or bands (Fig. 4D) of discolored coral tissue. Lesions can develop on all areas of the coral colony, but are most common on upper surfaces (Santavy et al. 1999). A patch of exposed skeleton is often present at the center of each circular to irregularly shaped lesion and at the edge of each band-

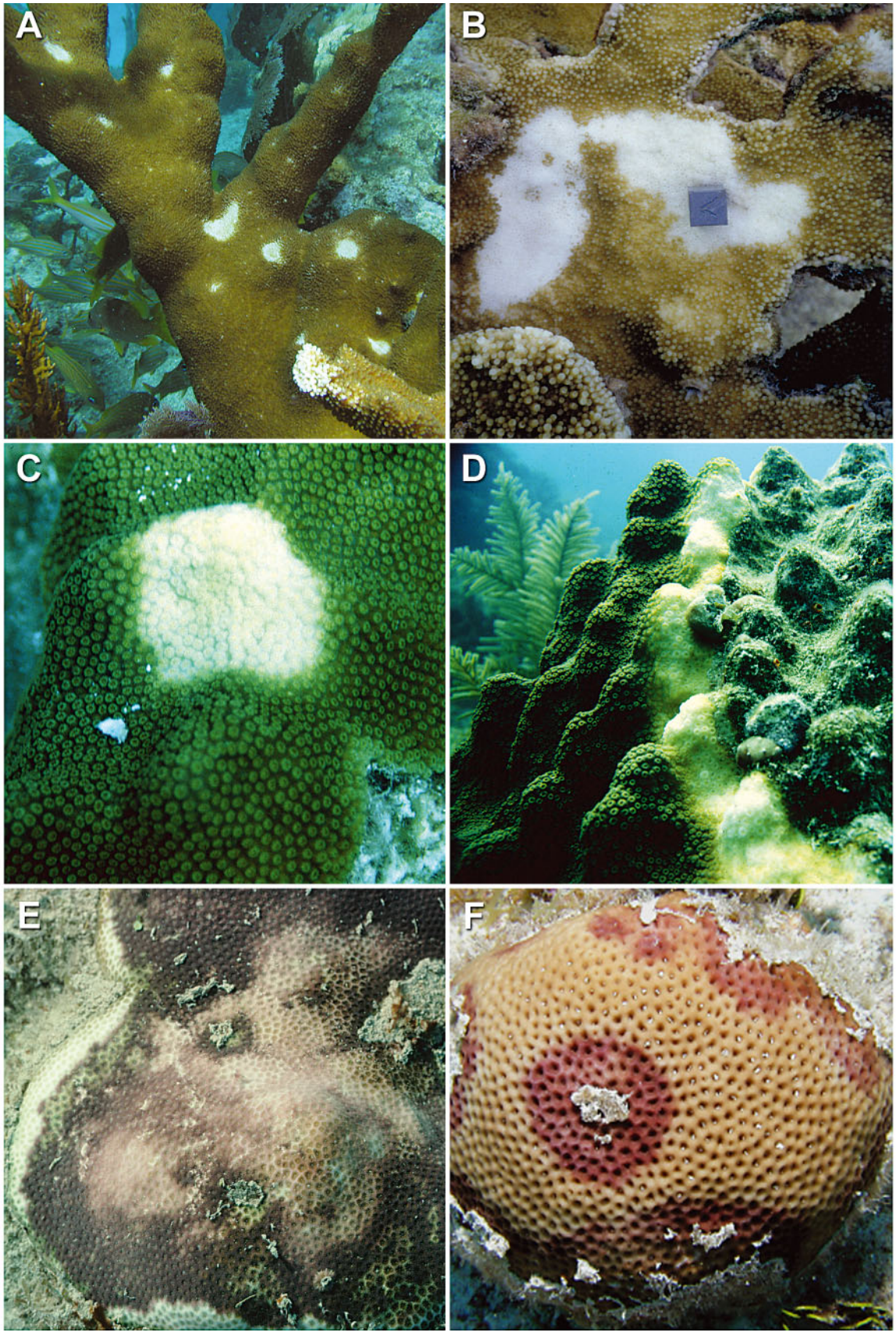
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Fig. 4. Caribbean coral diseases: (A) white pox (WPD) on *Acropora palmata*; (B) WPD lesions on *A. palmata*; (C) yellow blotch/band (YBL) circular-shaped lesion and (D) YBL band-shaped lesion on *Montastraea annularis* complex; (E) dark spots (DSD) on *Stephanocoenia michelinii*; (F) DSD on *Siderastrea siderea.* Photographs: (A) and (E) by J.W.P., (B) by K.P.S., (C) and (D) by C. Quirolo, (F) by J.W.P. and C.T.

shaped lesion. This dead zone is surrounded by rings or bands of yellow translucent tissue, which may in turn be surrounded by rings or bands of pale brown to bright-white bleached tissue. Apparently healthy tissue surrounds the outer regions of YBL lesions (Santavy et al. 1999, Toller et al. 2001). Tissue loss rate associated with YBL is approximately 0.6 cm mo^{-1} (Cervino et al. 2001).

YBL-affected coral tissues have approximately 40% fewer algal symbionts than do apparently healthy tissues, and these zooxanthellae appear vacuolated and lack organelles (Cervino et al. 2001). In San Blas (Panama) zooxanthellae within yellow and normal tissues from YBL-affected *Montastraea annularis*, *M. faveolata*, and *M. franksi* were found to be of different taxa of the genus *Symbiodinium* (Toller et al. 2001). Normal (unaffected) tissues were dominated by *Symbiodinium* sp., clade C, the taxon common in healthy corals at depth of collection (1 to 10 m). Yellow (affected) tissues predominately contained *Symbiodinium* sp., clade A, the source of the characteristic yellow color of YBL lesions (Toller et al. 2001).

Dark spots

Dark spots disease (DSD; Fig. 4E,F) was first documented in the early 1990s in Colombia and today is found throughout the Caribbean (Goreau et al. 1998, Cervino et al. 2001, Garzón-Ferreira et al. 2001, Gil-Agudelo & Garzón-Ferreira 2001, Weil et al. 2002). DSD reportedly affects 11 scleractinian species (Table 2; Goreau et al. 1998, Cervino et al. 2001, Garzón-Ferreira et al. 2001, Gil-Agudelo & Garzón-Ferreira 2001).

DSD is characterized by irregularly shaped dark spots of purple, maroon, or brown coloration on normal tissue (Goreau et al. 1998). DSD may be associated with tissue necrosis and/or depression of the colony surface (Cervino et al. 2001). When DSD is associated with tissue loss, a spot may expand into a characteristic dark ring separating dead skeleton from living tissue (Goreau et al. 1998).

Dark pigmentation of DSD-affected *Stephanocoenia michelinii* extends into, and stains, the skeleton, and may be attributed to zooxanthellae, which appear darker in pigment in diseased corals of this species. In *Siderastrea siderea* affected with DSD, skeletal staining and zooxanthellae of darker pigmentation are not evident, but zooxanthellae do appear swollen and necrotic. These observations suggest that similar disease signs observed on *S. michelinii* (Fig. 4E) and *S. siderea* (Fig. 4F) may represent 2 different diseases (Cervino et al. 2001).

In the Colombian Caribbean, DSD is correlated with depth and temperature. DSD is more prevalent at depths less than 6 m and during summer when seawater temperature is highest (Table 5). Depth distribution of DSD may be due to high disease prevalence (94%) on 2 coral species that occupy shallow depths, *Siderastrea siderea* and *Montastraea annularis*. Distribution of DSD on Colombian reefs is clumped, indicating that the disease may be contagious and therefore of biotic origin (Gil-Agudelo & Garzón-Ferreira 2001). The causal agent of DSD is unknown.

INDO-PACIFIC CORAL DISEASES

Vibrio-induced bleaching

Coral bleaching is a global phenomenon that occurs when symbiotic algae (zooxanthellae) and/or their pigments are lost from host coral tissues, resulting in pale brown to translucent living tissue. This condition reduces reproductive output, skeletal growth, calcification rates, and nutrition (Porter et al. 1989, Brown 1997a), and therefore is a sign of disease. Bleaching is most often correlated with elevated seawater temperature, but may be associated with salinity extremes (Coles & Jokiel 1992), light irradiance extremes (Brown et al. 1994, 2000, Brown 1997b, Shick et al. 1996), and pollutants (Peters et al. 1981, Harland & Brown 1989). Bleaching may result in coral mortality, but most often the coral regains its algal symbionts, resulting in full recovery (Fitt et al. 1993).

Coral bleaching, once regarded exclusively as an abiotic condition, has recently been associated with 2 bacterial pathogens of the genus *Vibrio*. In the Mediterranean Sea, bleaching of the coral *Oculina patagonica* can be induced by an infection with the bacterium *V. shiloi* (Tables 3 & 4; Kushmaro et al. 1996, 1997, 1998, 2001, Rosenberg et al. 1998). Bleaching and lysis of tissue of the coral *Pocillopora damicornis* in Zanzibar, Indian Ocean and Eilat, Red Sea are induced by infection with *V. coralliilyticus* (Tables 3 & 4; Ben-Haim & Rosenberg 2002, Ben-Haim et al. 2003a,b). *Vibrio*-induced bleaching and lysis are always associated with elevated seawater temperatures in excess of 24.5°C

(Kushmaro et al. 1998, Toren et al. 1998, Ben-Haim et al. 1999, 2003b, Banin et al. 2001a, Israely et al. 2001), and therefore are examples of diseases with etiologies associated with both abiotic and biotic factors.

Vibrio shiloi-induced bleaching

Vibrio shiloi-induced bleaching (VSB) of *Oculina patagonica* is arguably the most characterized coral disease in terms of etiology and mechanism of pathogenesis. Koch's postulates were fullfilled and showed that bleaching can be induced by infection with *V. shiloi* in apparently healthy corals maintained at elevated seawater temperatures (25 to 29°C, Kushmaro et al. 1998). Elevated seawater temperatures increases the virulence of *V. shiloi* (Table 5; Kushmaro et al. 1998, Toren et al. 1998, Ben-Haim et al. 1999, Banin et al. 2001a, Israely et al. 2001).

Under conditions of elevated seawater temperatures, infection begins with adhesion of *Vibrio shiloi* to a β-galactoside-containing receptor in mucus on the surface of the host coral (Toren et al. 1998, Banin et al. 2001b). Photosynthetically active zooxanthellae inside the host coral produce the β-galactoside-containing receptor. Only *V. shiloi* that are grown at elevated seawater temperatures are capable of adhesion (Toren et al. 1998). Therefore, adhesion only occurs under conditions of elevated temperature and if photosynthetically active zooxanthellae are present (Banin et al. 2001b).

The second step in the infection process is penetration of *Vibrio shiloi* into the epidermis of the host coral. Once *V. shiloi* are inside the tissues they multiply and transform into a viable but non-culturable state (VBNC). In other words, intracellular *V. shiloi* are not capable of forming colonies on media that support growth of extracellular *V. shiloi*. Intracellular bacteria are protected against effects of treatment with the antibiotic gentamicin. Gentamicin kills extracellular *V. shiloi* associated with coral mucus, but has no effect on *V. shiloi* that have successfully penetrated host tissue (Banin et al. 2000). At elevated temperatures, VBNC *V. shiloi* infect healthy coral and induce bleaching (Israely et al. 2001).

Following penetration into the host coral, *Vibrio shiloi* produces both heat-stable and heat-sensitive toxins that target zooxanthellae and play a role in pathogenesis. The heat-sensitive toxin bleaches and lyses symbiotic algal cells isolated from the host coral (Rosenberg et al. 1998, Ben-Haim et al. 1999). The extracellular heat-stable toxin, termed Toxin P, is a proline-rich dodecapeptide that binds to zooxanthellae and inhibits photosynthesis in the presence of ammonia (Rosenberg et al. 1998, Ben-Haim et al. 1999, Banin et al. 2001a). Inhibition of photosynthesis damages zooxanthellae and contributes to coral bleaching (Banin et al. 2001a). Toxin P is produced only under conditions of elevated seawater temperature (25 to 30°C, Banin et al. 2001a). *V. shiloi* require another virulence factor, superoxide dismutase, in order to survive inside the host coral. High levels of superoxide dismutase are produced by *V. shiloi* under conditions of elevated seawater temperature (30°C, Israely et al. 2001).

During summer in the Mediterranean Sea, *Vibrio shiloi* are present in all bleached colonies of *Oculina patagonica*. However, during winter, when water temperature drops to 16°C, intracellular *Vibrio shiloi* lyse and die, and corals affected by VSB recover. *V. shiloi* outside the coral survive the temperature shift, indicating that the coral host may play a role in seasonal demise of the pathogen (Israely et al. 2001). Reinfection of *O. patagonica* colonies the following summer is facilitated, at least in part, by the marine fireworm *Hermodice carunculata*, which serves as a reservoir and a vector of *V. shiloi*. During winter, *V. shiloi* is present in the VBNC state inside *H. carunculata*, and during summer, when the worm feeds on *O. patagonica*, the VSB pathogen is transmitted to a few coral colonies. This indirect transmission serves to restart the infection process, and the infectious VSB then spreads from colony to colony (Sussman et al. 2003).

Vibrio coralliilyticus-induced bleaching and disease

Vibrio coralliilyticus-induced bleaching and disease (VCB) affects the scleractinian coral *Pocillopora damicornis*. VCB is characterized by bleaching (Ben-Haim et al. 2003b) and lysis of coral tissue (Ben-Haim & Rosenberg 2002, Ben-Haim et al. 2003b). Koch's postulates were fulfilled and showed that VCB is caused by a new species of bacterium, *V. coralliilyticus* (Table 4; Ben-Haim & Rosenberg 2002, Ben-Haim et al. 2003a,b). Bleaching and tissue loss associated with *V. coralliilyticus* is temperature dependent. Infection with *V. coralliilyticus* induces bleaching at seawater temperatures of 24.5 and 25.0°C, and tissue lysis and colony death at seawater temperatures of 27 and 29°C (Ben-Haim et al. 2003b). Bleaching does not preceed lysis at temperatures greater than 27°C (Ben-Haim et al. 2003b). VCB is infectious and can be transmitted through direct contact between an infected coral colony and an un-infected neighbor (Ben-Haim & Rosenberg 2002). *V. coralliilyticus* produces an extracellular protease that may play a role in pathogenesis, and production of this enzyme increases at temperatures greater than 24°C (Ben-Haim et al. 2003b). Elevated seawater temperature increases either pathogen virulence or host susceptibility, or both (Ben-Haim & Rosenberg 2002).

Skeleton eroding band

Skeleton eroding band (SEB) disease was first documented in 1988 in Papua New Guinea (Antonius & Lipscomb 2000). SEB has since been observed in the Red Sea, Indian Ocean, and the Great Barrier Reef, Australia. Attempts to locate the disease in the Caribbean have been unsuccessful. Although Koch's postulates have not been fulfilled, SEB is believed to be caused by the protozoan *Halofolliculina corallasia* (Table 4). SEB affects 24 Indo-Pacific scleractinian species (Table 3; Antonius & Lipscomb 2000).

Halofolliculina corallasia is a folliculinid heterotrich ciliate that is sessile in a secreted black sac-like test called a lorica. Dense clusters of protozoans, with basal portions of their loricae embedded in host coral skeleton, form the characteristic black band of SEB. SEB advances across a coral by asexually producing migratory larval stages that move ahead of the band in masses and locate a site to settle and secrete loricae. A combination of chemicals associated with production of pseudochitinous loricae, and drilling of loricae into coral skeleton, result in skeletal erosion and tissue death (Antonius & Lipscomb 2000).

SEB resembles BBD in gross disease signs, except that SEB is not associated with a microbial mat that can be lifted from the coral surface. SEB affected corals are characterized by a black band ranging in width from less than 1 mm to 80 cm, depending on species affected. The line migrates horizontally across affected coral at a rate that may be as slow as 1 mm wk^{-1} or as fast as 1 mm d^{-1}, leaving dead skeleton in its wake. Denuded skeleton is flecked with tiny black spots composed of clusters of empty loricae, a characteristic that clearly distinguishes SEB from BBD, which leaves behind bright white skeleton (Antonius & Lipscomb 2000).

SEB is found at depths ranging from 0 to 35 m and is most common at 0.5 to 3 m. The disease does not appear to be correlated with seasonal seawater temperatures. SEB can be directly transmitted from a diseased colony to a healthy colony only via direct physical contact. Attempts to transmit the disease via seawater in enclosed aquaria were unsuccessful (Antonius & Lipscomb 2000).

Yellow band

Yellow band disease (YBD) was first observed in the Gulf of Oman, Arabian Sea in the late 1990s. The disease affects 12 Indo-Pacific scleractinian coral species (Table 3; Korrûbel & Riegl 1998, Riegl 2002). YBD is not the same disease as Caribbean yellow blotch/band disease (YBL).

YBD is characterized by a broad band of yellow-pigmented tissue (Korrûbel & Riegl 1998) and a yellow-pigmented microbial mat (B. Riegl pers. comm.) that migrate horizontally across the coral, producing a margin of decaying tissue adjacent to healthy tissue and leaving behind dead skeleton that often retains a yellow pigmentation (Korrûbel & Riegl 1998). The presence of a microbial mat suggests that YBD may be a variant of BBD (B. Riegl pers. comm.). Rate of tissue loss is correlated with seasonally elevated temperature and is greater in summer (19.7 mm wk^{-1}) than in winter (9.4 mm wk^{-1}, Table 5; Riegl 2002). YBD is transmissible from colony to colony (Riegl 2002).

Pink-line syndrome

Pink-line syndrome (PLS) was first reported in 2001 affecting the scleractinian corals *Porites compressa* and *P. lutea* of Kavaratti Island, Indian Ocean (Table 3). PLS is characterized by a band of pink-pigmented tissue separating dead skeleton from apparently healthy tissue. This band may begin as a small ring and progress outward horizontally across a coral colony. As with other coral diseases, the zone of dead skeleton is bright white in appearance, indicating a relatively rapid rate of disease progression (Ravindran et al. 2001). PLS is associated with the cyanobacterium *Phormidium valderianum*, which induces pink coloration of coral tissue, hypothesized by increasing levels of pCO_2 (Ravindran & Raghukumar 2002).

Fungal-protozoan syndrome

Epizootics affecting 6 gorgonian species, 1 scleractinian species (Table 3), 1 zoanthid (*Parazoanthus axinellae*), sponges, and encrusting coralline algae occurred in the Liguarian Sea, north-western Mediterranean, in late summer 1999. Losses of gorgonian corals were estimated to be in the millions (Cerrano et al. 2000).

Gorgonian corals affected by the syndrome, here termed fungal-protozoan syndrome (FPS), are characterized by an increase in mucus production, loss of pigmentation, and loss of coenenchyme tissue. Spicules in the coenenchyme lose the thin outer layer of epidermal cells and become disorganized. Coenenchyme tissues are colonized by fungi and coral polyps are colonized and consumed by protozoan ciliates. Fungi associated with FPS were most commonly of the genus *Trichoderma*, but *Clodosporium*, *Penicillum*, and *Humicola* were also common (Table 4; Cerrano et al. 2000).

Fungi and protozoa associated with FPS may be opportunistic rather than primary pathogens. FPS epizootics of 1999 were correlated with elevated seawater

temperature (Table 5). The etiology of FPS may stem from a combination of an abiotic stressor (i.e. elevated seawater temperature) and a biotic attack by opportunistic microorganisms (Cerrano et al. 2000).

CORAL IMMUNITY

Immunity is the protection against infectious disease and can be innate or acquired (Stedman 2000). Knowledge of the immune systems of zooxanthellate corals and other invertebrates is limited; however, these organisms are known to possess highly efficient defense mechanisms against infection. In order to fully understand coral immunity, it is helpful to first briefly review the immune mechanisms of invertebrates in general.

Invertebrates are limited to innate immunity (Roch 1999), defined as a non-specific general ability of certain cells to resist most pathogens (Stedman 2000). Vertebrates, on the other hand, utilize both innate immunity and acquired immunity (Roch 1999, Stedman 2000), a specific and highly sophisticated mechanism for developing resistance to individual pathogens (Stedman 2000). Components of the acquired immune system, including antibodies, antigen presenting cells, interactive lymphocytes, and lymphoid organs are absent in invertebrates (Sinderman 1990).

Invertebrate and vertebrate immune responses are similar in that both types of organisms employ physiochemical barriers, cellular defenses, and humoral defenses against pathogens (Roch 1999). Physiochemical barriers serve as the first line of defense against invaders and include epidermis, mucus, cuticles, tests, shells, and gut barriers (Roitt et al. 1996, Peters 1997). Cellular (whole cell) defenses depend on the ability of the organism to distinguish non-self from self, and in invertebrates include: (1) coagulation and wound healing, (2) hemocytosis, (3) phagocytosis, (4) encapsulation, and (5) immunological memory (Kinne 1980, Sinderman 1990, Roitt et al. 1996). Invertebrates employ both natural and inducible humoral (cell product) defenses (Sinderman 1990, Roitt et al. 1996) that provide the lytic properties of phagocytic cells and hemolymph (blood, Sinderman 1990).

There is evidence for vertebrate defense mechanisms in invertebrates including cytokines, complement, and immunological memory (Roitt et al. 1996). Invertebrate cytokine-like molecules regulate invertebrate host defenses by activating phagocytosis and encapsulation. Invertebrates known to produce cytokine-like molecules include protozoa, annelids, echinoderms, tunicates, and arthropods (Roitt et al. 1996). Complement participates in control of inflammation, bacterial lysis, microbial killing, and phagocytosis (Roitt et al. 1996). Phagocytosis is enhanced when complement molecules, termed opsonins, bind to invading microorganisms or other foreign material (Bayne 1990, Roitt et al. 1996). Opsonin-dependent phagocytosis is found in a number of invertebrates including echinoderms, crustaceans, mollusks, annelids, and insects (Bayne 1990, Roitt et al. 1996). Immunological memory is a mechanism by which a host can distinguish non-self from self and resist infection by previously encountered pathogenic microorganisms (Sinderman 1990). Memory has been observed in scleractinian and gorgonian corals (Theodor 1970, Hildemann et al. 1975, 1977a, 1980a, Raison et al. 1976, Johnston et al. 1981, Bak & Criens 1982, Neigel & Avise 1983), sponges (Hildemann et al. 1979, 1980b, Hildemann & Linthicum 1981, Bigger et al. 1982, 1983, Johnston & Hildemann 1983), echinoderms (Karp & Hildemann 1976, Hildemann et al. 1979), mollusks, crustaceans (Anderson 1986, Sinderman 1990), tunicates (Lakshma Reddy et al. 1975, Raftos et al. 1987), nemerteans (Langlet & Bierne 1982), and annelids (Hildemann et al. 1979).

Coral physiochemical barriers

Scleractinian and gorgonian corals utilize mucus production and a protective epidermis as physiochemical barriers (Peters 1997, Santavy & Peters 1997, Hayes & Goreau 1998). Sloughing of mucus is an important defense mechanism of corals against attachment of potentially pathogenic bacteria to surface tissues (Ducklow & Mitchell 1979a, Rublee et al. 1980). However, there are examples of mucus aiding in attachment of pathogenic microorganisms to the surface of corals. Adhesion of *Vibrio shiloi*, the pathogen that causes VSB, to the coral surface requires a β-galactoside-containing receptor in the mucus (Toren et al. 1998). Further, Lipp et al. (2002) demonstrated that potentially pathogenic enteric bacteria and viruses are concentrated on the surface mucus layers of scleractinian corals under natural near-shore conditions in the Florida Keys.

Coral cellular defenses

Cellular defenses depend on the ability of the coral to distinguish non-self (e.g. sediment, pathogens) from self. Cellular defenses documented for scleractinian and gorgonian corals include: (1) wound healing (Bigger & Hildemann 1982, Meszaros & Bigger 1999), (2) phagocytosis (Bayne 1990, Sinderman 1990, Peters 1997), and (3) immunological memory (Theodor 1970, Hildemann et al. 1975, 1977a, 1980a, Raison et al. 1976, Johnston et al. 1981, Bak & Criens 1982, Neigel & Avise 1983).

Wound healing involves the infiltration of granular amoebocytes to immobilize invading microorganisms

(Bigger & Hildemann 1982, Sinderman 1990). Coelenterate invertebrates, including scleractinian and gorgonian corals, have the ability to repair wounds, i.e. regenerate lost tissue (Patterson & Landolt 1979, Meszaros & Bigger 1999). Wound healing in coelenterates is carried out by mobile phagocytic cells, termed amoebocytes, that migrate from uninjured tissue, accumulate at the site of injury, and arrange into interconnected cell cords that form the healing front (Patterson & Landolt 1979, Meszaros & Bigger 1999). Amoebocytes also function to recognize, engulf, and destroy microbial invaders through phagocytosis (Bigger & Hildemann 1982, Bayne 1990, Sinderman 1990, Meszaros & Bigger 1999). For corals and other tropical marine invertebrates, phagocytosis is the principal cellular defense (Sinderman 1990, Peters 1997).

Immunological memory in corals is a component of allogenic recognition, the ability to distinguish self from non-self in tissue grafts. Corals accept isogenic (intracolony) grafts through complete fusion of tissue and skeleton (Hildemann et al. 1975, 1977a, Raison et al. 1976, Bak & Criens 1982, Neigel & Avise 1983, Jokiel & Bigger 1994), but reject allogenic (intraspecies) and xenogenic (interspecies) grafts through cytotoxic interaction and necrosis (Theodor 1970, Hildemann et al. 1975, 1977a,b, 1980a,b, Raison et al. 1976, Johnston et al. 1981, Jokiel & Bigger 1994). This aggression is accelerated for repeat grafts, indicating the presence of specific induced alloimmune memory (Theodor 1970, Raison et al. 1976, Hildemann et al. 1977a, 1980a,b, Johnston et al. 1981), an immune response generally reserved for vertebrates. However, in contrast to long-term vertebrate memory, coral alloimmune memory is short-term, lasting for a period of 2 to 4 wk (Raison et al. 1976, Hildemann et al. 1977a, 1980a,b). Cytotoxic reactions to both primary and secondary allografts are accelerated under conditions of seasonally elevated seawater temperatures (27°C, Bigger & Hildemann 1982, Johnston et al. 1981), suggesting that decreased temperatures (21°C) may result in immunosuppression (Johnston et al. 1981), reducing a coral's ability to defend itself against pathogenic microorganisms.

Corals probably evolved mechanisms for cytotoxic attack due to the constant threat of overgrowth by adjacent benthic organisms. Scleractinian corals compete for space on the reef by utilizing a variety of defense mechanisms including: (1) rapid growth rate to overgrow neighbors (Lang 1973, Porter 1974, Maguire & Porter 1977), (2) extrusion of mesenterial filaments to attack neighboring species, killing coral tissue via extracoelenteric digestion (Lang 1973, Porter 1974), and (3) allelochemical attack, resulting in tissue necrosis (Theodor 1970, Hildemann et al. 1975, Raison et al. 1976, Hildemann et al. 1977a,b, 1980a,b, Johnston et al. 1981).

Corals that grow rapidly (e.g. acroporids) and therefore can overgrow and outcompete neighbors for space, possess less potent mesenterial filaments and allelochemicals than do slower growing corals (Lang 1973, Porter 1974, Bak & Criens 1982). Intracolony isografts of *Acropora cervicornis* (Neigel & Avise 1983) and *A. palmata* (Bak & Criens 1982) are characterized by complete fusion of tissue and skeleton. Intercolony allografts of these 2 species result in overgrowth of tissues of either graft over recipient colony or recipient colony over graft (Bak & Criens 1982, Neigel & Avise 1983), and fusion of the skeleton is evident in *A. cervicornis* allografts (Neigel & Avise 1983). Xenografts of *A. palmata* and *A. cervicornis* are characterized by overgrowth where *A. palmata* consistently grows over *A. cervicornis* (Bak & Criens 1982). Fusion of skeleton and overgrowth of tissues of allografts and xenografts indicate that acroporid corals utilize minimal antagonistic intraspecific and interspecific defenses (Bak & Criens 1982, Neigel & Avise 1983).

A number of cellular immune responses have been observed in scleractinian and gorgonian corals affected by coral disease. ASP-affected Caribbean gorgonian corals *Gorgonia* spp. utilize sclerite recruitment and nodule formation to sequester *Aspergillus sydowii* fungal hyphae and limit spread of infection (Smith et al. 1998). Similarly, Caribbean gorgonian corals *Pseudoplexaura* spp., with skeletal nodules caused by encapsulation of the algae *Entocladia endozoica*, produce many more sclerites than do healthy corals, and these sclerites surround and encapsulate the infected skeleton (Goldberg et al. 1984). *Pseudoplexaura* spp. with algal nodules produce large numbers of granular amoebocytes when algal filaments extend beyond the skeletal lesion and invade host mesoglea. These amoebocytes coat invading algae with a layer of mesoglea-like material, and coated algae are then encapsulated by host skeleton (Goldberg et al. 1984). Indo-Pacific scleractinian corals *Porites* spp. detect invasion by endolithic fungi and respond by surrounding the site of fungal penetration within layers of repair calcium carbonate (Le Champion-Alsumard et al. 1995, Ravindran et al. 2001).

Coral humoral defenses

Corals employ both natural and inducible humoral defenses. Lysozyme and lysosomal enzymes are natural humoral defenses used by corals (Sinderman 1990). Lysozyme is an antimicrobial lysin found in phagocytic cells that destroys susceptible bacteria by elevating levels of lysosomal enzymes (Sinderman 1990). Lysosomal enzymes are contained within membrane-bound vesicles (lysosomes) and are granules

containing bactericidal and hydrolytic substances that play a critical role in killing phagocytized microorganisms (Bayne 1990, Sinderman 1990). Antimicrobial activity is both natural and inducible in scleractinian and gorgonian corals. Corals utilize antibiotic compounds or noxious chemicals to repel potentially pathogenic or parasitic organisms (Kim 1994, Slattery et al. 1995, 1997, Jensen et al. 1996, Koh 1997, Peters 1997, Kelman et al. 1998, Kim et al. 2000a,b).

Jensen et al. (1996) and Kelman et al. (1998) tested antibiotic activity (i.e. inhibition of bacterial growth) of secondary metabolites from gorgonian corals against marine bacteria and concluded that gorgonians lack potent broad-spectrum chemical defenses. However, gorgonians do possess species-specific chemical defenses against potentially pathogenic microorganisms (Kim 1994, Kelman et al. 1998).

Kelman et al. (1998) examined extracts from various reproductive and developmental stages of the Red Sea gorgonian *Pareythropodium fulvum fulvum* against bacteria isolated from: (1) SML of *P. fulvum fulvum*, (2) apparently healthy and necrotic tissue of *P. fulvum fulvum*, (3) reef substrates adjacent to test corals, and (4) seawater in the vicinity of test corals. *P. fulvum fulvum* possessed species-specific chemical defenses against the potentially pathogenic bacterium *Vibrio* sp. isolated from necrotic tissue as well as against bacteria isolated from reef substrates and seawater, but lacked antimicrobial activity against commensal bacteria associated with SML and healthy coral tissue (Kelman et al. 1998).

Koh (1997) measured antimicrobial activity of extracts from 100 species (44 genera, 13 families) of scleractinian corals from the Great Barrier Reef against 6 marine bacteria isolated from Australian waters and one terrestrial bacterium. All 100 species exhibited antimicrobial activity against 1 marine cyanobacterium: *Synechococcus* sp. Significant activity against the other 5 marine bacteria and the terrestrial bacterium was detected in only 6 and 11 coral species, respectively, indicating that the majority of the corals did not employ chemical defenses against these potentially pathogenic bacterial species (Koh 1997).

Kim et al. (2000a) provide the first evidence of chemical defense mechanism against a known coral pathogen. Gorgonian corals possess inducible chemical defenses to resist infection with *Aspergillus sydowii*, the causal agent of ASP. Antifungal agents in crude extracts from *Gorgonia ventalina* and *G. flabellum* inhibit germination of *A. sydowii* spores (Kim et al. 2000a, Dube et al. 2002). Elevated summer seawater temperature (30°C) reduces potency of *Gorgonia* crude extracts and promotes growth of *A. sydowii* (Alker et al. 2001).

Antifungal activity associated with ASP varies with health status and size class of host gorgonians and with lesion location on diseased individuals (Kim et al. 2000a, Dube et al. 2002, Kim & Harvell 2002). Increased antifungal activity is inducible in ASP-affected gorgonians with lesions at the colony center (Kim et al. 2000a) and in diseased individuals that are larger and more mature (Dube et al. 2002). Prevalence and severity of ASP is greatest on the largest size class of affected sea fans (Dube et al. 2002, Kim & Harvell 2002). Larger and/or older colonies may be more susceptible to *Aspergillus sydowii* as indicated by reduced potency of anti-fungal agents in their crude extracts (Kim & Harvell 2002). Small colonies exhibit higher antifungal activity than do large colonies and are more resistant to ASP (Dube et al. 2002). Antifungal activity is greater in healthy than in diseased gorgonians, and this activity is concentrated at colony edges. Greater concentrations of antifungal agents in crude extracts from healthy gorgonians indicate that antifungal activity influences resistance to infection (Kim et al. 2000a).

CONCLUSION

Eighteen coral diseases, affecting at least 150 scleractinian, gorgonian, and hydrozoan zooxanthellate species, have been described from the Caribbean and the Indo-Pacific (Tables 1, 2 & 3). Despite the greater species richness of the Indo-Pacific, the number of species affected by disease is proportionally much lower than in the Caribbean. Of the approximately 400 coral species in the Indo-Pacific, only 98 (25%) have been documented with 1 or more diseases, while at least 52 of the 66 (82%) Caribbean coral species are known to be susceptible to disease. Worldwide, 22 coral species are affected by 4 or more different diseases (Tables 2 & 3), and 14 of these species belong to 4 genera (*Acropora*, *Diploria*, *Colpophyllia*, and *Montastrea*) that represent the most common, frame-building coral species. The susceptibility of these species to a wide array of diseases has the potential to change the composition, structure, and function of coral reef ecosystems.

In the Caribbean, WPL (40), BBD (25), SKA (22), DSD (11), and YBL (9) affect the greatest number of coral species (Table 2). Diseases affecting the greatest number of Indo-Pacific coral species include BBD (45), WPL-L (37), SKA (24), SEB (24), and YBD (12; Table 3). Caribbean and Indo-Pacific scleractinian corals are highly susceptible to plague-like diseases, including WPL I, WPL II, WPL III, WPL-L, WBD I, and WBD II (Tables 2 & 3). It is important to note that the gross disease signs used to identify plague-like diseases in the field (i.e. coral tissue loss and exposed white skeleton; Pantos et al. 2003) may simply be indicative of coral death. Further, in the Caribbean, gross signs of WBD differ from those of WPL only in species affected (i.e. branching species). Future research will indicate

whether plague-like signs on Caribbean and Indo-Pacific coral species represent a single disease condition, caused by a single pathogen (e.g. *Aurantimonas coralicida*, Richardson et al. 1998a,b, Denner et al. 2003), or if similar disease signs documented for the plague-like diseases represent different diseases caused by a variety of pathogens.

Accumulating evidence suggests that human activity in the watershed may be causally related to coral decline. Increases in the number of both new diseases (Fig. 2) and species affected may be directly linked to human-induced alterations in coral reef environments both in terms of land-based sources of pollution as well as global climate change issues such as global warming (Table 5). Elevated seawater temperature may be associated with the etiologies of at least 9 coral conditions and is likely the most common abiotic stressor influencing disease pathogenicity (Table 5). Further, 5 coral disease-causing pathogens, including the BBD cyanobacteria, *Aurantimonas coralicida* (WPL II), *Aspergillus sydowii* (ASP), *Vibrio shiloi* (VSB), and *V. coralliilyticus* (VCB), are most virulent at seawater temperature at or above 29°C (Kushmaro et al. 1998, Alker et al. 2001, Banin et al. 2001a, Israely et al. 2001, Ben-Haim & Rosenberg 2002, Kuta & Richardson 2002, Ben-Haim et al. 2003b, Richardson & Kuta 2003). Seawater temperature normally increases during late summer, but all current models of global climate change suggest that, on average, ocean temperatures will rise over the next century (Kleypas et al. 1999). Elevated temperature is a stressor in corals, causing thermally induced breakdown in the coral-zooxanthellae host-symbiont relationship (Porter et al. 1989, Fitt et al. 1993, Brown 1997b). Elevated temperature also promotes growth and virulence of pathogens (Kushmaro et al. 1996, 1998, Toren et al. 1998, Alker et al. 2001, Banin et al. 2001a, Israely et al. 2001, Ben-Haim & Rosenberg 2002, Kuta & Richardson 2002, Ben-Haim et al. 2003b, Richardson & Kuta 2003) and reduces immune response in host corals (Toren et al. 1998, Alker et al. 2001).

Nutrient and sediment loading may deliver potentially pathogenic organisms to the marine environment. Transport of the terrestrial fungus *Aspergillus sydowii*, the causal agent of ASP, to the marine environment may be associated with local sediment run-off from land or long-distance atmospheric transport (Smith et al. 1996, Shinn et al. 2000). Increasing evidence suggests that the health of reef organisms is affected by sewage pollution. BBD and WPD may be associated with fecal contamination of possible human origin (Frias-Lopez et al. 2002, Patterson et al. 2002). Human enteric bacteria and viruses are prevalent on coral surfaces and in nearshore, offshore, and canal waters in the Florida Keys (Lapointe et al. 1990, Paul et al. 1995a,b, 1997, Griffin et al. 1999, Lipp et al. 2002). The 1983 epizootic affecting the Caribbean long-spined sea urchin *Diadema antillarum* caused catastrophic reductions in urchin populations (Lessios et al. 1984). It has been suggested that the fecal bacterium *Clostridium perfringens* may have been involved in the urchin die-off (Bauer & Agerter 1987, 1994). Since 1996, populations of the sewage consuming reef sponge *Cliona delitrix* have increased by a factor of 10 on reefs in the Florida Keys (Ward-Paige 2003) while, concurrently, corals have declined by 37 % (Porter et al. 2002).

Etiologies of only 5 coral diseases have been determined through fulfillment of Koch's postulates, but several other disease conditions have been linked to specific biotic organisms (Table 4). The application of Koch's postulates for the identification of coral disease has severe drawbacks, and in order for the study of coral disease etiology to advance, alternative techniques for identifying disease-causing pathogens and abiotic stressors must be accepted and implemented. Knowledge of coral disease reservoirs, transmission, pathogenesis, and epizootiology is limited, and significant advances remain to be made in the field of coral immunology.

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