CHAPTER 23

Indo-Pacific Colored-Band Diseases of Corals

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Introduction

Colored-band diseases are defined as those which exhibit a distinct pigmented band along the progressing disease front, separating recently exposed skeleton from clinically healthy coral tissue. This band may be comprised of a consortium of micro-organisms at least some of which are involved in coral tissue death or consumption, or it may largely consist of a single species. Unlike many other coral diseases, band diseases are often distinctive in gross appearance and are, therefore, relatively easy to assess in the field and distinguish from other sources of mortality, such as predation. In the Indo-Pacific, three diseases involving the formation of a colored band have been described. This chapter will review what is currently known about two of them: brown-band disease (BrB) and black-band disease (BBD). A third band disease, skeletal eroding band (SEB) is briefly mentioned here and will be discussed fully in Chapter 26.

Brown-Band Disease (BrB)

History and Geographical Range

A brief report by Dinsdale (1994) noted a "brown band" affecting the scleractinian *Acropora formosa* (later renamed as *Acropora muricata;* Wallace 1999) on the Great Barrier Reef (GBR), Australia, and was distinct in its appearance from black-band disease. This report was cited by Bruckner (2002), who presented the first comprehensive list of diseases reported in the literature, broken down by ocean/geographic region. The first formal description of brown band was presented in Willis *et al.* (2004) and this constitutes how the disease is currently described.

To date, BrB has been verified in the Indo-West Pacific as far south as the GBR (Willis *et al.* 2004), northward through Indonesia, Palau, and the Philippines to Guam and Pagan, Commonwealth of the Northern Mariana Islands (CNMI) (Haapkylä *et al.* 2009; Page *et al.* 2009; Raymundo *et al.* 2009; Myers and Raymundo 2009; Vargas-Angel and Wheeler 2009), and westward to Tanzania (Zanzibar) (Weil and Jordan-Dahlgren 2005). It has not been observed east of Guam nor from the Red Sea (Table 23.1). Aeby *et al.* (2009) report a ciliate disease affecting a plating *Acropora* in American Samoa, but the gross description of disease signs differs from that considered typical of BrB.

Host Range

Willis *et al.* (2004) reported BrB affecting 12 species of branching *Acropora*, as well as staghorn, tabular, corymbose and digitate growth forms, and *Isopora*. In addition, *Montipora* spp., *Pocillopora* spp., *Echinopora* sp. and other unidentified faviids and pocilloporids were reported susceptible, all from the GBR (Willis *et al.* 2004). In the Philippines, Indonesia, Guam and Zanzibar, the disease has only been observed on acroporids, particularly thicket-forming species. These include *Acropora muricata* (as described in Wallace 1999), *A. aspera, A. surculosa* and *A. acuminata* (as described in Randall and Myers 1983) in Guam (Lobban *et al.* 2011; Raymundo *et al.* unpublished observations); *A. intermedia* in the Philippines (Raymundo, unpublished data) and *A. pulchra* in Indonesia (Haapkylä *et al.* 2009). These reports suggest the disease still primarily targets the genus *Acropora*, though knowledge of host range may expand as surveys broaden.

Epizootiology

Though information on its geographic range continues to expand, BrB is generally not very prevalent. In the Philippines, it was only observed on three out of 22 reefs surveyed and mean prevalence on these reefs was 0.33% +/− 0.2% (Raymundo, unpublished data). In Guam, it is equally rare; only two sites out of 13 surveyed showed infected corals and prevalence on these reefs averaged 1% (Myers and Raymundo 2009). In Palau, it existed on four out of eight reefs, with a mean prevalence of 0.68% (Page *et al.* 2009) and on the GBR, it was observed on all three reefs surveyed, with a prevalence of 0.31% (Willis *et al.* 2004). The recent report from Japan was from a fringing reef off Zamani Island in the Marine Protected Area of the Kerama archipelago, west of Okinawa. The disease showed a low

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Table 23.1 Summary of geographic range of colored band diseases in the Pacific and Indian Oceans and the Red Sea, as reported in published literature.

Location	BrB	BBD	Survey years	Source
Philippines	+	+	1985, 2003, 2004–8	Antonius 1985a; Kaczmarsky 2006; Raymundo *et al*. 2009
Great Barrier Reef, Australia	+	+	1988–98, 2004, 2006	Willis *et al*. 2004; Page and Willis 2006; Page and Willis 2008
Indonesia	+	+	2005, 2007	Haapkyla *et al*. 2009
Main Hawaiian Islands	–	–	2006, 2007	Vargas-Angel and Wheeler 2009
Northwest Hawaiian Islands	–	–	2003, 2006, 2007	Aeby 2006, Vargas-Angel and Wheeler 2009
Guam	+	+	2005–7	Myers and Raymundo 2009, Vargas-Angel and Wheeler 2009
Northern Mariana Islands	–	+	2007	Vargas-Angel and Wheeler 2009
American Samoa	–	+	2004–6	Vargas-Angel and Wheeler 2009
Palau	+	+	2004, 2005	Page *et al*. 2009
Marshall Islands	+	+	2003–present	Jacobson, personal communication
Fiji	?	+	1995	Littler and Littler 1996
New Caledonia	–	–	2005–present	Job, personal communication
Pacific Remote Island Areas (Wake, Johnston, Baker, Howland, Jarvis, Palmyra, Kingman)	–	–	2006, 2007	Vargas-Angel and Wheeler 2009

prevalence (0.8%) and was not observed in other surveyed reefs (Weil *et al.* 2012). While these prevalence values are similar to those reported for other diseases (see discussion on black-band disease, below), BrB is recorded from fewer sites than most other diseases, which may be a function of low acroporid host abundance or lack of accurate field diagnosis, as the band is sometimes difficult to distinguish from living tissue without the aid of magnification.

Limited evidence suggests injury or stress may be involved with disease pathogenesis (see below) and therefore plays a role in the spread of the disease within a population. Page *et al.* (2009) found a strong positive correlation between host coral bleaching and the prevalence of BrB infections (Page *et al.* 2009), while Raymundo first verified the disease in Guam initiating along exposed margins of laboratory-housed fragments (Lobban *et al.* 2011). Bleached tissue could provide the requisite signals triggering ciliate aggregation, though this remains to be investigated. If the stress is widespread and affects multiple species, such as a bleaching event or predator outbreak, then the "double whammy" of an initial stress resulting in tissue loss and potential entry wounds, followed by disease infecting surviving colonies, has a much greater potential for impact.

Pathogenesis

Brown bands are composed of dense aggregations (≤120 cells per square millimeter) of mobile ciliates which appear to directly consume coral tissue (Lobban *et al.* 2011; Raymundo, personal observation; Fig. 23.1a). Endosymbiotic zooxanthellae *(Symbiodinium)* are visible within the ciliates and give the band its characteristic color. Zooxanthellae may reach densities of >50 cells/ciliate (Ulstrup *et al.* 2007; Fig. 23.1b). Bourne *et al.* (2008) provided molecular evidence that zooxanthellae observed within the ciliates most likely originate from the corals from which the ciliates were sampled, suggesting that the ciliates are either directly consuming living coral tissue (carnivory) or feeding on dead or decaying tissue (histophagy).

Ciliates move freely within the coral skeleton during active feeding. This is followed by a quiescent period, when they appear to stop feeding and become immobile on the coral skeleton. These immobile ciliates comprise the band that is visible to the naked eye. After this period, they disappear from the coral which is then rapidly colonized by algae. Efforts are underway to describe the life cycle, and thereby provide a more complete picture of the role of the ciliates in BrB pathogenicity (Lobban *et al.* 2011).

Injury to tissue may provide a point of entry or an aggregation cue, or both. BrB infections were observed to develop and progress on *Acropora cytherea* branches following predation by Crown-of-Thorns starfish *(Acanthaster planci)* (Nugues and Bak 2009), which creates patches of exposed skeleton and a break in the tissue, and stimulates mucous production along the damaged tissue border. In aquarium-reared *A. surculosa,* bands were initiated at the broken, exposed base of fragmented branches (Lobban *et al.* 2011). The corallivorous gastropod *Drupella* sp. was a highly successful vector; feeding behavior on *A. muricata* initiated band formation on 40% of experimental fragments (Nicolet *et al.* 2013). Lastly, Lamb and Willis (2011) noted that BrB was one of the most prevalent diseases on reefs frequented by tourists, while the disease was absent from their control sites. These observations suggest that the disease may opportunistically follow an unrelated stress to the host and may require an entry wound in order to be initiated.

The rate at which BrB progresses within a colony can be quite rapid. In Guam, ciliate presence on *Acropora surculosa* was

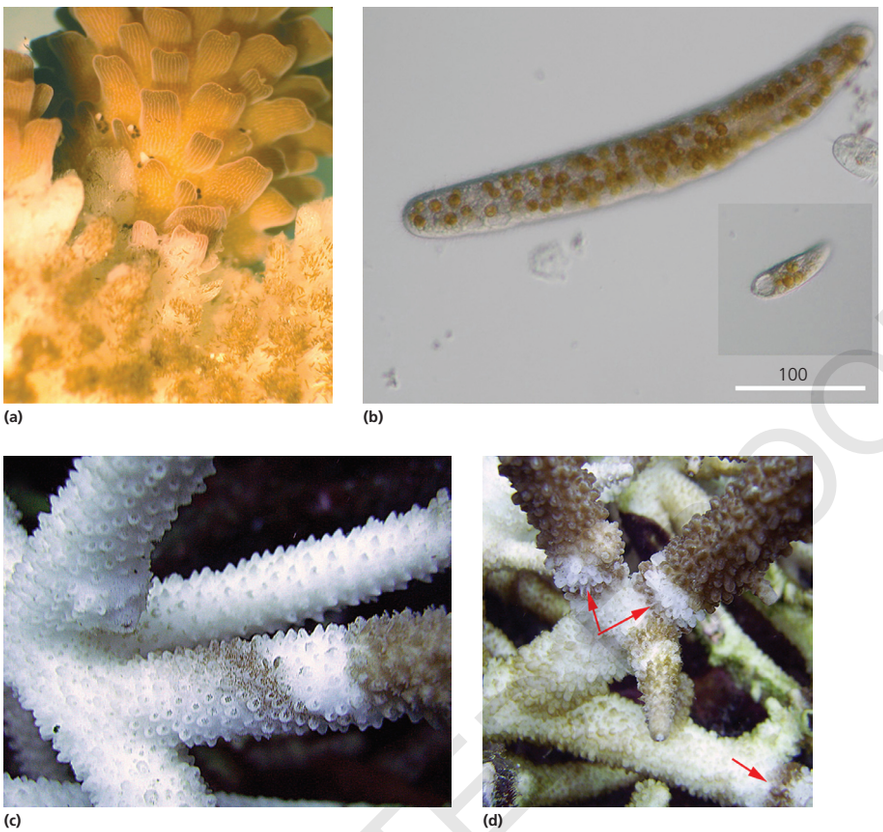

Fig. 23.1 (a) brown band infection on *Acropora surculosa,* showing dense ciliate band adjacent to healthy, intact tissue (photograph: L. Raymundo); (b) a scuticociliate from Guam taken from a brown band infection on *Acropora muricata,* showing recently consumed zooxanthellae from the coral host (photograph: C. Lobban); (c) brown band infection on *Acropora intermedia* from the central Philippines, showing a distinct white zone between healthy tissue and the ciliate band (photograph: L. Raymundo); (d) multiple brown band infections within a single colony of *A. intermedia* (photo: L. Raymundo).

confirmed microscopically and ciliates were observed to actively feed along the exposed tissue margin from the fragment base upward, consuming tissue at an average rate of 1.85 mm/h (Lobban *et al.* 2011). Within five days, 88% of the fragments (n = 30) were completely devoid of tissue. Willis reported similarly high tissue loss rates *in situ* on the GBR (>5 cm/day) starting from the branch base and progressing upward (Willis, personal communication). Haapkylä *et al.* (2009) reported *in situ* progression rates averaging 1.2 cm/day, representing the highest rates of tissue loss among four diseases monitored in Indonesia.

It is unclear whether the ciliates are the primary cause of tissue mortality or whether they opportunistically feed on diseased or injured tissue, or both, depending on specific conditions. Some records of the disease (e.g., Bourne *et al.* 2008) note a zone of tissue loss (Fig. 23.1c) between healthy tissue and the ciliate band and suggest a possible microbial agent kills tissue that is subsequently fed upon by the ciliates. Other observers have noted very low densities of the ciliates without actual band formation, which may reflect a late stage in the feeding cycle or opportunistic, nonpathogenic feeding. Still other cases note a very dense band immediately adjacent to living tissue (i.e., the white zone is absent). These observations suggest that ciliate populations may be able to switch from opportunistic feeding on necrotic tissue to a pathogenic state causing tissue mortality, but the mechanism by which this happens is poorly understood at present. Aggregation that results in band formation may be triggered by chemical signals released from injured, diseased or stressed tissue, from the ciliates themselves, or even from the released zooxanthellae.

Clinical Features

Gross Description of the Lesion

In the field, the disease manifests as a golden-brown band (due to consumed zooxanthellae in the ciliates) up to 1 cm wide. The disease appears to progress by consumption of tissue by the ciliate band, leaving behind stark white skeleton which is rapidly colonized by algae (usually within one week). As stated above, there may be a narrow band of white skeleton as a result of tissue loss between the band and healthy coral tissue at the advancing disease front. If affecting a branching species, the band circumscribes the branch and usually begins from the base of the fragment, rapidly progressing upward. There is usually a single band per branch, though multiple branches per colony may be

affected (Fig. 23.1d). On tabular and massive species, the band generally begins at the colony margin, though on rare occasions it has been observed beginning medially on a colony (Page, personal communication).

Histopathology

To date, no reports of histopathological examinations of brown-band disease in corals have appeared in the literature. Work is underway, however, to sample, describe and identify BrB ciliates from corals in different locations, as reports from the Great Barrier Reef (GBR) (Bourne *et al.* 2008) and Guam (Lobban *et al.* 2011) have identified different ciliate species associated with BrB (see below).

Microbial Community

To date, two species of Scuticociliates (Class Oligohymenophora, Sublass Scuticociliatia) comprising the visible bands have been identified: *Parauronema longum,* from the GBR (Bourne *et al.* 2008) and *Porpostoma guamensis,* from Guam (Lobban *et al.* 2011). Molecular work accomplished by Sweet and Bythell (2012) led to their proposal that *Porpostoma guamensis* should be reconsidered as *Philaster guamensis* and this is the currently accepted designation and the GBR (Sweet and Bythell 2012). Additional ciliates associated with the disease have also been reported in recent work by Sweet and Bythell (2012): *Philaster* sp., *Varistrombidium* sp., and *Euplotes* sp. All three of these, and *P. guamensis*, were found in diseased tissue containing zooxanthellae from the host coral. Of these four species, *Philaster* sp. was considered to be the most etiologically important as it burrowed rapidly into and under live coral tissue along the lesion front. This observation of aggressive *Philaster* behavior, and the absence of visible necrotic tissue along the lesion margin, combined with molecular analysis indicating bacterial involvement (Sweet and Bythell 2012) may explain the frequent appearance of a band of exposed tissue between apparently healthy tissue and the visible ciliate band. These observations suggest that pathogenic bacteria may be the primary cause of tissue death in some cases, with the ciliates primarily feeding on necrotic tissue resulting from bacterial infection.

Ulstrup *et al.* (2007) demonstrated that ingested zooxanthellae continue to be viable and photosynthetically active for an unknown period of time, suggesting the ciliates may benefit from this association by absorbing photosynthate and having access to greater oxygen concentrations. However, as the life cycle of the ciliates is still currently under investigation (Lobban *et al.* 2011) it is not known how long the zooxanthellae remain viable within the ciliates or the stability of this association.

Yarden *et al.* (2009) report the first description of fungal communities associated with BrB-affected colonies of *Acropora muricata*. Though fungal communities were taxonomically similar in healthy and diseased tissue, the authors noted that the relative abundance of fungi increased significantly in diseased tissue. Because the role of these fungi in the coral host is currently unknown, it remains unclear whether fungi opportunistically increase when host tissue is compromised and play some role in the disease process, or whether the fungi have a role in host defense.

Complications and Controversy

Santavy and Peters (1997) speculated that Dinsdale's (1994) report of Indo-Pacific "brown band" could be Caribbean red-band disease which affects at least 20 Caribbean species and in which cyanobacteria are implicated. However, no formal description was made in these early reports and more recent work has failed to demonstrate any evidence that these two diseases are one and the same.

Sweet *et al.* (2012) reviews the aquarium trade grey literature regarding brown jelly syndrome (BJS) of cultured corals, reported as the second most common disease among cultured corals. It is reportedly associated with the ciliate *Helicostoma nonatum* (e.g., Borneman 2001; Borneman and Lowrie 2001), but no controlled experiments have demonstrated causation. It is characterized by a rapidly progressing brown jellylike coating on corals, accompanied by tissue necrosis and excess mucous production. This condition may have been confused with BrB when initial observations of BrB appeared in the scientific literature (e.g., Willis *et al.* 2004). Both diseases are associated with ciliates which tend to form a bandlike feeding front. However the jellylike coating is not consistent with reported disease signs for BrB, and it remains unclear whether these two conditions represent the same disease operating under different environmental conditions or whether they are separate diseases. Cooper *et al.* (2007) also attributed *H. nonatum* (apparently a synonym of *H. notatum* described by Carey (1992), to tissue loss and mortality in spat of *Porites astreoides* and adult colonies of *P. astreoides* and *Montastraea faveolata* in laboratory aquaria. Currently, it is not known how many species of ciliates may be associated with this disease, and what role they play in the disease process. An added source of confusion is the lack of molecular validation of the taxonomy of the subclass Scuticociliata (see review by Sweet *et al.* 2012).

Black-Band Disease (BBD)

History and Geographical Range

Black-band disease (BBD) was first noted in reefs off Belize on the western Caribbean in the early 1970s. It was described as a microbial assemblage that formed mobile dark filamentous bands which progressed across healthy coral colonies, actively destroying tissues and leaving behind a clean, white, bare skeleton (Antonius 1973, 1981).

Black-band disease has now been reported from many other locations outside the Caribbean. The first Indo-Pacific records came from the Philippines and the Red Sea in the mid-1980s (Antonius 1985a, 1988a). It has since been reported from the Gulf of Aqaba (Al-Moghrabi 2001; Zvuloni *et al.* 2009), Great Barrier Reef (Dinsdale 1994), Fiji (Littler and Littler 1996),

Papua New Guinea (Frias-Lopez *et al*. 2003), Palau (Sussman *et al*. 2006), Guam (Burdick *et al*. 2008; Myers and Raymundo 2009), Indonesia (Haapkylä *et al.* 2009), Japan (Yamashiro, 2004; Weil *et al.* 2012) and other localities (Green and Bruckner 2000; Sutherland *et al*. 2004). It has not been sighted in Hawaii or the Pacific Remote Islands (Table 23.1), though extensive surveys in these areas have been undertaken (Aeby 2006; Vargas-Angel and Wheeler 2009). Until 2000, the majority of observations of BBD in the Indo-Pacific were from Saudi Arabia (106), Philippines (18), Australia (13) and Egypt (9) (Green and Bruckner 2000) though the geographic range has expanded in recent times with additional survey efforts (Sutherland *et al*. 2004). As research continues, the known geographic distribution of BBD will certainly expand.

Host Range

Few species were reported as susceptible to BBD in the early reports from the Indo-Pacific. Two faviid species in the Philippines, and eight faviids and one merulinid in the Red Sea constituted the first report of infected species (Antonius 1985a). Since then, 21 species in five families were found to host BBD in the first disease surveys on the GBR (Dinsdale 2002) and more recent surveys have increased that number to at least 40 species in 12 genera and seven families, with branching species in the genus *Acropora* the most commonly affected (Page and Willis 2006). Sutherland *et al*. (2004) compiled a comprehensive list of scleractinian species affected by BBD worldwide, listing 45 species of host corals for the Indo-Pacific, though localities were not mentioned. We present a current list of reported host species and their localities in Table 23.2. Though there are reports of octocorals and hydrocorals affected by BBD in the Caribbean (Green and Bruckner 2000; Weil 2004; Sutherland *et al*. 2004; Weil and Rogers 2011), the only nonscleractinians reported to be affected in the Indo-Pacific are two soft corals and the hydrocoral *Millepora,* all from the GBR (Page and Willis 2006).

Interestingly, although host range appears to be rather broad, in certain locations (Philippines, Rosell and Raymundo 2008; Great Barrier Reef, Sato *et al*. 2009; Guam, Myers and Raymundo 2009; Palau, Sussman *et al*. 2006), BBD is rarely observed to infect more than one species on a given reef, even when other documented host species are present in the vicinity. However, a different pattern was observed in the Red Sea (Al-Moghrabi 2001; Zvuloni *et al*. 2009), with species from multiple families simultaneously infected within a given area.

Epizootiology

Only recently have quantitative data on BBD distribution, prevalence and rates of within-colony disease progression in Indo-Pacific localities been available. In the GBR for example, BBD was found in 74% of the reefs surveyed between 1999 and 2004 (n = 19) which encompassed 1000 km of the Queensland coast (Page and Willis 2006). These results indicate that BBD is widespread along the GBR, but they contrast with those of previous surveys in which BBD was found in 19% of 110 reefs surveyed between 1993 and 1996 (Miller 1996) and between 2–17% of 48 reefs surveyed in 1998 and 2004 by the long-term monitoring program of the Australian Institute of Marine Science (Page and Willis 2006). Page and Willis (2006) argue that these differences may be largely explained by variability in the survey methods, with minor variability attributed to differential spatial distribution of the disease and/or seasonal variability in prevalence.

Despite its widespread distribution, wide host range and apparent increase in the number of reefs affected, BBD prevalence was still very low (<0.9%) and stable from 1999 to 2004 in the GBR (Page and Willis 2006). Results of extensive surveys in other island groups showed similarly low prevalence of BBD: the Philippines: 1.5% (Raymundo, unpublished data); Guam: 0.24% (Myers and Raymundo 2009); and Indonesia: 0.02% (Haapkylä *et al*. 2009). These prevalence values suggest that the disease is not normally highly transmissible. However, monitoring in Australia by Sato *et al.* (2009), the Red Sea by Zvuloni *et al.* (2009), and Guam (Raymundo *et al*. unpublished observations) show that the disease may reach seasonally high outbreak levels, which may be driven by a unique combination of environmental variables (see below). Transmissibility may be affected by the abundance of requisite vectors or a combination of factors necessary to promote the formation of the consortium, but this remains to be investigated.

The spatial distribution of the disease within reefs may be clumped on small spatial scales (<40 m^2) (Page and Willis 2006; Zvuloni *et al.* 2009). Though this is predicted with infectious disease and waterborne pathogens, Edmunds (1991) did not find a clumped distribution of infected colonies in the Caribbean.

Temperature, light and sedimentation all appear to play a role in the epizootiology of Indo-Pacific BBD which is a pattern also seen in the Caribbean. A number of studies have noted increased prevalence during warm seasons (Boyett *et al*. 2007; Sato *et al*. 2009, Australia; Antonius 1988a; Zvuloni *et al.* 2009, Red Sea; Raymundo *et al*. unpublished observations, Guam). Seasonally warmer temperatures and increased light intensity enhance both transmission between colonies and rate of progress within colonies (Boyett *et al.* 2007; Sato *et al.* 2009), suggesting that warmer temperatures may enhance the virulence of the disease and/or increase host susceptibility. Further, annual infection rates of colonies from the GBR (Sato *et al.* 2009) and the Red Sea (Zvuloni *et al*. 2009) suggest that previous infections may make colonies more susceptible to reinfection during the next warm season.

High sediment load may have multiple impacts on coral health as it can both reduce light availability and cause physical stress via smothering and abrasion, but links with disease prevalence are equivocal; tissue loss from silt smothering may mask disease signs, making it difficult to determine the exact cause of

Table 23.2 Host taxa reported to be affected by BBD in the Indo-Pacific.

Taxon	Locality	Source(s)
Acropora hyacinthus; A. intermedia; A. gemmifera; A. millepora; A. microclados; A. monticulosa; A. florida; A. microphthalma; A. robusta; A. humilis; A. palifera; A. sarmentosa; A. pharaonis; A. microcyathus; A. haimei; A. corymbosa; A. hyacinthus; A. humilis; A. pallida; A. eurystoma; A. hemprichii; A. nasuta; A. latistella, A. valida; A. gemmifera; A. elseyi; A. loripes; A. yongei; A muricata; A. cuneata; A. intermedia; A. brugemanni; A. clathrata; Isopora sp.	GBR, RS, In	Antonius 1985b, 1988; Dinsdale 2002; Willis *et al.* 2004; Page and Willis 2006; Haapkyla *et al.* 2009
Astreopora myriophthalma, spp.	GBR, RS	Al-Moghrabi 2001; Willis *et al.* 2004; Page and Willis 2006
Montipora verrucosa, Montipora spp.	GBR, RS, Pa, Ph, Gu	Antonius 1988; Al-Moghrabi 2001; Willis *et al.* 2004; Sussman *et al.* 2006; Myers and Raymundo 2009; Raymundo unpublished
Pocillopora damicornis; P. verrucosa, P. eydouxi	GBR, Pa, RS	Antonius 1988; Willis *et al.* 2004; Page and Willis 2006
Seriatopora hystrix, spp.	GBR	Willis *et al.* 2004; Page and Willis 2006
Stylophora pistillata	GBR, RS	Willis *et al.* 2004; Antonius 1988
Porites spp. (massive)	GBR, Gu, Ph, Pa	Willis *et al.* 2004; Dinsdale 2002; Myers and Raymundo 2009; Raymundo unpublished data; Sussman *et al.* 2006
Goniopora fruticosa, G. columna, Goniopora sp.	GBR, RS, Gu	Al-Moghrabi 2001; Page and Willis 2006; Myers and Raymundo 2009
Alveopora gigas	RS	Al-Moghrabi 2001
Goniastrea retiformis; G. pectinata	Ph, GBR, RS	Antonius 1985b; Antonius 1988; Dinsdale 2002; Zvuloni *et al.* 2009
Favia matthaii, F. favus, F. pallida, F. stelligera	GBR, RS, Ph	Antonius 1985b; Willis *et al.* 2004; Zvuloni *et al.* 2009
Favites pentagona, Favites spp.	RS	Antonius 1985b, Zvuloni *et al.* 2009
Diploastrea heliopora	In	Haapkyla *et al.* 2009
Echinopora lamellosa, E. gemmacea, horrida, E. mammiformis	GBR, Ph, Pa, RS	Willis *et al.* 2004; Rosell and Raymundo 2008; Sussman *et al.* 2006; Antonius 1988
Cyphastrea serailia	GBR, RS	Antonius 1988
Leptoria phrygia	RS	Antonius 1985b
Platygyra lamellina, Platygyra spp.	GBR, Ph, RS	Antonius 1985b; Antonius 1988; Willis *et al.* 2004; Zvuloni *et al.* 2009
Turbinaria	GBR	Willis *et al.* 2004; Page and Willis 2006
Echinophyllia aspera	RS	Al-Moghrabi 2001
Pectinidae	GBR	Willis *et al.* 2004
Pachyseris speciosa	Pa, In	Sussman *et al.* 2006
Psammacora digitata	GBR, Gu	Willis *et al.* 2004; Myers and Raymundo 2009
Coeloseris mayeri	Ph	Rosell and Raymundo 2008
Pavona decussata	Gu	Myers and Raymundo 2009
Acanthastrea	RS	Zvuloni *et al.* 2009
other Mussidae	GBR	Dinsdale 2002; Willis *et al.* 2004
Fungia sp.	Ph	Raymundo, unpublished
Hydnophora microconos, H. rigida	GBR, RS	Antonius 1985b; Willis *et al.* 2004
Millepora (O. Hydrocoralina)	GBR	Willis *et al.* 2004
Lobophytum (O. Alcyonacea)	GBR	Willis *et al.* 2004
Sinularia (O. Alcyonacea)	GBR	Willis *et al.* 2004

Notes: GBR = Great Barrier Reef; RS = Red Sea; Pa = Palau; Ph = Philippines; F = Fiji; GU = Guam; In = Indonesia.
Sources: Antonius (1985b, 1988); Al-Moghrabi (2001); Dinsdale (2002); Sutherland *et al*. (2004); Willis *et al*. (2004); Page and Willis (2006) and references therein; Sussman *et al.* (2006); Rosell and Raymundo (2008); Haapkyla *et al*. (2009); Myers and Raymundo (2009); Zvuloni *et al*. (2009); Raymundo unpublished data. All taxa are order Scleractinia unless otherwise indicated.

death. The first records of BBD from Fiji were observed in a reef impacted by terrigenous sediment (Littler and Littler 1996). Kaczmarsky (2006) noted that BBD was observed most frequently near river mouths in the Philippines, which are generally areas of high turbidity and sediment input. In Indonesia, sedimentation was positively associated with an overall loss of coral cover (Haapkylä *et al*. 2009); however, none of these studies specifically examined sedimentation and its effect on BBD prevalence. It is likely that other water quality issues (nutrient load, turbidity, pollutants) may have influenced coral loss and disease prevalence at these sites. In contrast, Page and Willis (2006) found a negative link between BBD prevalence and proximity to terrestrial influences on the GBR; BBD was more prevalent on reefs adjacent to more pristine terrestrial

catchments. These results suggest that teasing out the effects of sedimentation on BBD will require controlled manipulative studies. Turbidity lowers light availability, which would be predicted to slow the progress of a disease involving migrating photosynthetic cyanobacteria, as demonstrated in Caribbean BBD (Carlton and Richardson 1995; Viehman and Richardson 2002). However, terrestrial sediment also carries with it elevated nutrient and bacterial loads, organic particulates, and other pollutants which potential may increase host susceptibility to disease or provide nutrients to photosynthesizing pathogenic cyanobacteria. As sedimentation is a major cause of coral loss on a global scale, understanding how it influences progression and spread of individual diseases is of importance and bears investigating.

Interest is growing in understanding the disease transmission process and identifying pathogen vectors and reservoirs, as knowledge of these aspects is necessary for disease management. Several fish families and benthic invertebrates feed on coral, either by removing overlying tissue or gouging the skeleton. Both processes produce a physical injury, triggering excess mucous production by the host and creating a potential entry wound for microbial pathogens. Pathogenic organisms can be transported between coral colonies in the mouth of the corallivore (Cole *et al.* 2009*)* or could be spread via feces or contact with a body surface that harbors a pathogen, as in the alga *Halimeda* (Nugues *et al.* 2004). Aeby and Santavy (2006) demonstrated a link between corallivorous chaetodontidae (butterflyfishes) and the spread of BBD in Caribbean *Montastraea*. Chaetodontids showed a preference for feeding on injured and/or diseased tissue and laboratory experiments noted more rapid disease spread in aquaria with chaetodontids present than in those without. In the Philippines, Raymundo *et al.* (2009) found a significant positive correlation between corallivorous chaetodontid abundance and coral disease prevalence, suggesting a role of this group of fishes in disease transmission. However, the mechanisms by which disease may be spread via corallivory are still under investigation. An *in situ* manipulative experiment by Cole *et al.* (2009) on the GBR demonstrated that the coral-feeding butterflyfish, *Chaetodon baronessa,* actually reduced the progression of BBD on monitored branches of *Acropora muricata*, by intense preferential feeding of band tissue. While this activity reduced the severity of BBD in infected colonies that were fed upon, the potential of the fish to pass on BBD to other colonies during feeding was not tested. In contrast, work by Zvuloni *et al.* (2009) in the Red Sea suggested that waterborne transmission appeared to be the most probable means of infection, as was initially reported by Bruckner *et al.* (1997) in Caribbean infections. These studies suggest that more than one avenue of transmission may be possible with BBD; physical injury may play a role in susceptibility via creation of an entry wound (see below), but pathogenic elements of the consortium may be either transmitted in the water or through a vector.

Pathogenesis

Most information on the pathogenesis, etiology, and histopathology for BBD comes from the Caribbean and is presented in Chapter 24. To date, no studies have compared the pathology of the disease in the Caribbean vs. Indo-Pacific. Antonius (1988b) pioneered transmission experiments with this disease, exposing 60 species of Red Sea corals to BBD–infected tissue to determine their susceptibility to the disease. Interestingly, the ease with which the tested species became infected did not reflect the prevalence of the disease in these species on the reefs surveyed. This study was among the first to explore and test methods of transmitting disease in controlled aquarium experiments, though it did not address transmission *in situ*. Two studies have since identified that the band may be initiated by physical injury to the colony surface (Peters 1997; Aeby and Santavy 2006). Lamb and Willis (2011) also noted an order of magnitude higher prevalence of the disease on reefs frequented by tourists than on reefs without tourism-related activities.

One of the most intriguing questions regarding BBD is the mechanism by which the consortium comprising the band forms. Sato *et al.* (2010) describe a successional process from samples obtained from GBR corals, which begins with a cyanobacterial patch (CP) composed of ribotypes (*Blennothrix* sp.) macroscopically distinct from those associated with the characteristic BBD band. The CP causes lesions that progress slower than those recorded for BBD, subsequently causing less tissue loss. However, this community undergoes a shift in composition to that of the typical BBD consortium, dominated by *Oscillatoria,* but also containing ribotypes associated with sulfur cycling. Lesions subsequently progress significantly faster once this community develops. This is consistent with what has been described for the consortium in Caribbean BBD (Richardson *et al.* 1997), where sulfur-cycling bacteria and the resulting high sulfide levels caused by their activity create a toxic environment leading to tissue death.

Clinical Features

Gross Description of the Lesion

In the field, the disease manifests as a dark annular band composed of filamentous cyanobacteria, immediately adjacent to clinically healthy coral tissue and preceding a zone of recently exposed white skeleton (Fig. 23.2a). The band is of variable width (from 0.1 mm to several centimeters wide; Frias-Lopez *et al.* 2003) and overlays recently killed skeletal tissue. With severe infections, there may be more than one lesion per colony. Coral tissue responds along the progressing front by extruding mesenterial filaments, though this response is not visible to the naked eye (Fig. 23.2b). Distinct coloration differences may be noted in different host species or geographic regions; coloration varying from black to brick red has been reported (Fig. 23.2c–d). Sussman *et al.* (2006) noted that band coloration appeared to be temporally stable within a colony, suggesting that the coloration is not due to photoadaptation or a response by the cyanobacterium to a change in growth conditions. Further, band

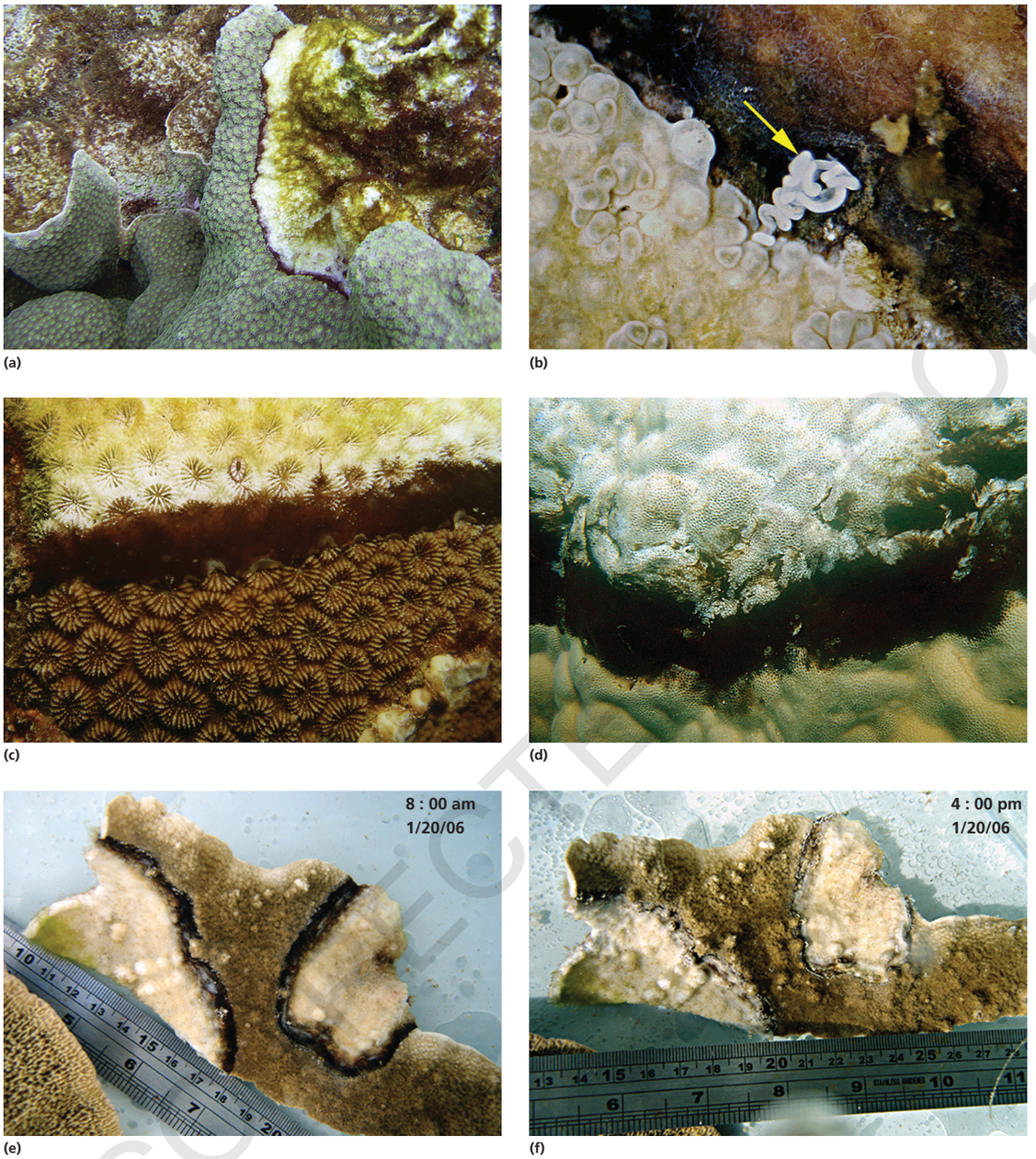

Fig. 23.2 (a) BBD on *Echinopora lamellosa,* showing the distinct white area of rapid tissue loss following the advancing band (photo: K. Rosell); (b) close up of BBD band (45X magnification), showing cyanobacterial filaments and a mesenterial filament extruded from an injured coral polyp (yellow arrow; photograph: L. Raymundo); (c) brick-red coloration of filamentous band on *Montastraea* sp. (photograph: D. Burdick); (d) black-brown coloration of the more typical BBD appearance on a massive *Porites* (photograph: D. Burdick); (e) appearance of the band in early morning with lower light conditions (photograph: L. Raymundo); (f) appearance of the band in late afternoon, after exposure to high light conditions (photograph: L. Raymundo).

thickness may vary within a single colony depending on time of day and light intensity (Fig. 23.2e–f). This suggests similarities with the Caribbean disease; sulfide-oxidizing bacteria have been demonstrated to migrate diurnally within the band (*Beggiatoa;* Richardson 1996).

Interestingly, Ainsworth *et al.* (2007) describe an "atypical" BBD infection wherein gross disease signs do not include a visible black band. The disease manifests as a white disease (See Chapter 21; White Syndrome) which may later develop into typical BBD. Using histopathological techniques and fluorescence *in situ* hybridization (FISH), the authors demonstrated that the consortium was present deep in the tissues and not visible on the surface. This may explain findings of associations between white plague and BBD outbreaks reported by Barash

et al. (2005) and Rosenberg and Ben-Haim (2002). Ainsworth *et al.* (2007) proposed the term "atypical black-band disease" for this form of BBD, and emphasized the need for further histological and molecular characterization of diseases for more accurate diagnoses.

On massive growth forms, infections usually start on an upper or side surface of a coral colony (Antonius 1981) as a small dark pigmented patch (1–2 cm diameter). The patch quickly forms a ring, the circumference of which rapidly increases as the band migrates horizontally across the coral, killing tissue in its path and leaving bare skeleton behind (Antonius 1981). On branching morphologies, such as *Acropora* and *Pocillopora*, lesions may begin at the colony base, circumscribing affected branches and progressing upward. BBD can advance at rates that vary from 0.39 cm/day (Haapkylä *et al.* 2009) to 1 cm/day (Antonius 1981, 1985b; Boyett *et al.* 2007). These rates of tissue loss are lower than those reported for BrB, but can result in significant colony mortality as they may persist for months, particularly during outbreak conditions.

Histopathology and Cytology

Ainsworth *et al.* (2007) presented histopathology of BBD in corals from the Red Sea. Diseased tissue displayed necrosis, disruption, and infiltration by cyanobacteria-dominated organisms comprising the band. Fluorescent *in situ* hybridization (FISH) analysis confirmed dense aggregations of bacteria within polyp gastrodermis indicative of BBD.

Microbial Community

Black-band disease is caused by a consortium of microbial agents. As causal agents, consortia are particularly challenging to study. Some of the questions currently under investigation include how these communities form, geographic variation in their taxonomic composition, and how they kill host tissue.

The characteristic filamentous cyanobacterium which is the dominant component of a BBD band, as measured in biomass, has been the primary target of study for Indo-Pacific corals. However, evidence suggests that there may be several species involved. Sussman *et al.* (2006) found a single cyanobacterial ribotype to be associated with both red and black bands in corals from Palau. This ribotype had a 99% sequence identity with an unidentified Caribbean strain, *Cyanobacterium* sp.

Frias-Lopez *et al.* (2003) identified two distinct taxa of cyanobacteria associated with BBD in the Caribbean and a different genus, *Trichodesmium* spp., from Indo-Pacific (Papua New Guinea) corals. However, *Phormidium corallyticum,* originally thought to be the primary causative agent and component of the band (Rutzler and Santavy 1983; Richardson 1996), was not present. Sato *et al.* (2010), in describing the successional process resulting in active BBD lesions, identified the filamentous cyanobacterium *Oscillatoria* as the dominant band component in GBR samples, along with gamma-proteobacteria and sulfur cycling bacteria. In Red Sea faviid corals (*Favia* sp.) a new species of filamentous cyanobacteria, *Psuedoscillatoria coralii*, has been described (Rasoulouniriana *et al.* 2009). Its 16S rRNA sequences showed a 99% similarity with other samples from BBD mats from Palau, Caribbean and the Red Sea affecting faviid corals, suggesting host-specificity by this species.

As molecular tools improve, reassessment of previous identifications is expected to produce new results that could explain some of these differences. However, while gross disease signs may be fairly consistent between host taxa and geographic regions, it is not unreasonable to predict that taxonomic composition of the BBD mat may vary. These data may reflect diversity within the consortium that is only now beginning to be recognized. In spite of this apparent taxonomic diversity, however, all samples examined appear to always contain similar functional elements: filamentous cyanobacteria and bacteria involved with the sulfur cycle.

Complications and Controversy

Due to the complexity of the BBD consortium, issues such as pathogenesis and initiation of band formation have been challenging to address. Antonius (1981) originally identified the primary causative agent as *Oscillatoria submembranacea.* This was later re-examined by Rutzler and Santavy (1983), who attributed the disease to a closely related species, *Phormidium corallyliticum,* using primarily phenotypic traits (morphology, pigmentation and motility) (Rutzler and Santavy 1983). The use of morphological characters has been reported to be unreliable for the cyanobacteria (Schönhuber *et al.* 1999). The fact that more recent investigations using molecular techniques failed to find either of these species in sampled bands suggests that new tools for investigating microbial agents will resolve such issues (see discussion above; Cooney *et al.* 2002; Frias-Lopez *et al.* 2002; Sussman *et al.* 2006).

Variation in band coloration may cause some confusion in field assessments. Richardson (1992) refers to a red-band disease affecting Caribbean corals, and this term has been informally applied to cases of disease in the Indo-Pacific. However, the work by Sussman *et al.* (2006) has provided some evidence that the variation in this disease sign probably does not indicate a separate causation or epizootiology.

Skeletal Eroding Band (SEB)

The gross appearance of skeletal eroding band (SEB) is briefly discussed here as it can be difficult to distinguish from other diseases, particularly BBD, in field assessments. A full discussion of this disease can be found in Chapter 26.

Gross Description of the Lesion

Skeletal eroding band infections are characterized by black to dark-green bands or patches of *Halofolliculina* ciliates on bare coral skeleton recently denuded of tissue. The density of ciliates comprising bands or patches is highly variable. Densities can be high enough that the population appears as a contiguous dark

green to black band adjacent to living coral tissue. In contrast, populations may appear as diffuse patches, giving bare skeleton a "salt and pepper" speckled appearance.

When *Halofolliculina* ciliates are present at high densities in Caribbean corals, infections can be difficult to differentiate from BBD *in situ* without the aid of a magnifying lens (Cróquer *et al*. 2006a, 2006b). If ciliate densities are very high, it may be necessary to sample a portion of the diseased tissue and examine it under a dissecting microscope to differentiate ciliates from a filamentous cyanobacterial mat. Diffuse patches are easier to distinguish, by the speckled appearance of the skeleton immediately behind the ciliate band.

Ciliates have been observed to secondarily colonize injuries created by other sources of damage and may not, therefore, be a primary cause of tissue loss (Page and Willis 2008; Rodriguez *et al*. 2009). They appear to represent an early successional stage before algal communities develop and may be replaced by later algal successional species. However, at high densities, the bands do appear to be the primary cause of mortality and the mechanism resulting in a switch to a pathogenic state is currently not understood.

Summary

- Brown-band disease (BrBD) is distinguished by a progressing golden-brown band posterior to clinically healthy coral tissue and anterior to bare skeleton; a zone of exposed skeleton may or may not be present between healthy tissue and the band.
- BrBD is composed of mobile ciliates that consume coral tissue yet harbor viable zooxanthellae, giving the distinctive brown color to the band.
- Tissue loss with BrBD may be very rapid, but prevalence on reefs is usually low.
- BrBD mainly targets branching acroporids, and pocilloporids and faviids, rarely.
- Black-band disease (BBD) is characterized by an advancing black to brick red band, up to a few centimeters wide, and composed of a consortium of bacterial and cyanobacterial species. The band is thought to kill living coral tissue via a toxic accumulation of sulfides, leaving bare skeleton behind.
- Though rates of tissue loss may be rapid, BBD is generally found in low prevalence on most reefs. However, it has been known to reach outbreak conditions and can cause significant mortality during warm seasons.
- Prevalence of BBD may vary seasonally, as transmission and progression appear to be favored by warm water temperature and high light intensity.
- The taxonomic composition of the microbial consortium associated with BBD appears to vary geographically, temporally, or by host taxa and further investigation is necessary to resolve discrepancies.
- At present, BBD is the most comprehensively studied scleractinian coral disease, though most research progress has been made on Caribbean corals. Limited work characterizing the Indo-Pacific disease suggests some fundamental similarities in both the ecology and microbial composition of the consortium.
- Skeletal eroding-band infections are characterized by black to dark-green bands or patches of *Halofolliculina* ciliates on bare coral skeleton.

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