Diseases, Harmful Algae Blooms (HABs) and Their Effects on Gulf Coral Populations and Communities

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7.1 Introduction

Corals in the Gulf exist in a harsh environment, which only allows a small subset of the typical Indo-Pacific fauna and flora to persist and/or form viable populations (Sheppard and Sheppard 1991; Sheppard et al. 1992; Samimi-Namin and van Ofwegen 2009; Chaps. 11 and 12). Environmental factors have been identified as the major killers of corals and these factors regulate population dynamics and coral reef community structure (Chaps. 2, 5, 10 and 16). Among these, extreme temperature variability, salinity variability and turbidity (as a result of coastal construction, Chap. 16) have been isolated as prime killers.

However, a host of biological agents are also capable of wreaking havoc on coral populations. In the Gulf, several of the major invertebrate nemeses of corals that exist in the Indian Ocean are absent. The crown-of-thorns starfish (COTS) *Acanthaster planci* has only ever been reported in two individuals from a single locality in Iran (Price and Rezai 1996), although this may be changing as COTS appear to be spreading from the Gulf of Oman into the Gulf, with new records from the Musandam Peninsula near the Straits of Hormuz (Mendonça et al. 2010). Furthermore, the coralivorous snail *Drupella cornus,* that can form equally devastation outbreaks, does not occur and the local *Coralliophila* spp. have not yet been reported to cause significant impacts on corals by predation as they do, for example, in the Caribbean.

Other biological agents do however take a significant toll on Gulf corals. In particular, coral diseases are one of the most destructive agents responsible for recent losses of coral. The Gulf harbors a unique disease, Arabian Yellow Band (AYB) that has a different dynamics from diseases with similar names observed elsewhere. Three other diseases have also been described (Riegl 2002; Benzoni et al. 2010; Samimi-Namin et al. 2010), and several uncharacterized syndromes are also known. While some diseases have been observed to be unusually common and have unusual dynamics (Riegl 2002), the 2010 bleaching event in the SE Gulf appears to have triggered locally significant outbreaks of a white syndrome that has taken a significant toll on UAE coral populations, similar to such phenomena in the Caribbean and Pacific (Bruno et al. 2007; Brandt and McManus 2009; Eakin et al. 2010; Bruckner and Hill 2009).

Highly destructible biological agents are Harmful Algae Blooms (HABs, or also called Red Tides) that have caused significant mortality lately both inside the Gulf (Samimi-Namin et al. 2010) and the Arabian Sea (Bauman et al. 2010; Foster et al. 2011). HABs have been occurring in all parts of the Gulf but appear to become more frequent, more lethal and more widespread, which is causing concern for reefs (Sheppard et al. 2010; Chap. 16).

This chapter describes the known coral diseases from the Gulf (Fig. 7.1) and explores their dynamics using simple mathematical models of the SIR (susceptible-infected-recovered) type (Anderson and May 1979; Mena-Lorca and Hethcote 1992). We explore the role of coral diseases in regulating populations of reef building corals and why some diseases are more frequent and persistent than others. Based on the outcomes of the models, we are able to speculate why, with increased ocean warming due to climate change (Sheppard and Loughland 2002) diseases have become more frequent and may become important agents that determine

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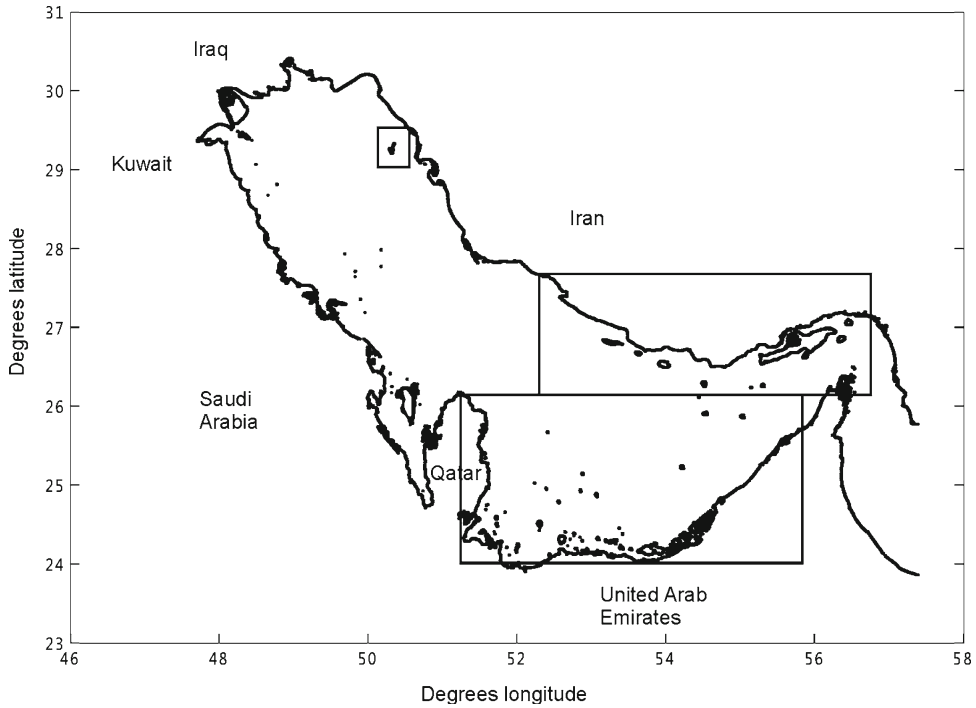

Fig. 7.1 Data for the present article were collected in the boxed area and concentrated on Iran, Qatar and the United Arab Emirates

future coral population structure and community dynamics much like the role of environmental stresses on reefs in the past. This chapter also discusses Harmful Algae Blooms and their importance in coral mortality dynamics in the Gulf.

7.2 Coral Diseases – Description and Etiology

Coral reef diseases occur globally, in most reef habitats and in most locations including reefs near human population centers and remote offshore locations. They generally affect a low proportion of the susceptible species, although localized outbreaks have produced significant mortalities to scleractinian corals, gorgonians, sea urchins, reef fish, sponges, algae and other coral reef organisms (Peters 1993; Harvell et al. 2001; Williams and Bunkley-Williams 2000). While coral reef diseases have not yet received much attention in the Gulf, diseases have been reported from other locations since the early 1970s (Antonius 1988). These first disease epizootics were reported as early as 1978 from the Caribbean and over the next two decades they caused large die-offs of two of the dominant structure forming corals, *Acropora palmata* and *A. cervicornis* (Bruckner 2003). Between 1982 and 1984, a disease of unknown cause spread throughout the western Atlantic, decimating populations of a keystone species, the herbivorous sea urchin, *Diadema antillarum*. These mortality events triggered massive increases in fleshy macroalgae and concurrent losses of coral cover, biodiversity and habitat in many locations (Lessios et al. 1984; Hughes 1994). There are now over 30 named diseases in the Caribbean basin affecting 45 zooxanthellate scleractinian corals, 3 hydrozoan corals, 10 octocorals, 2 zoanthids, 9 sponges and 2 crustose coralline algae (Green and Bruckner 2000; Weil 2004; Bruckner 2009a, b, c). In contrast, there are only four types of coral disease known from the Gulf. While an unprecedented increase in disease has been documented in the Caribbean, much less is known about the status of disease in the Indo-Pacific and, in particular, the Gulf region.

Increasing discovery of diseases throughout many reefs of the world may suggesting a rapid emergence and/or discovery of diseases, although there remain critical information gaps (Bruckner 2002). For example, current research has highlighted potentially important linkages between climate warming and disease, with thermal anomalies and bleaching events being followed immediately by outbreaks of disease (Miller et al. 2006; Miller et al. 2009; Bruckner and Hill 2009). This situation could also be confirmed for the Gulf after the 2010 bleaching event (Riegl and Purkis, personal observation). Changing environmental conditions could affect the coral holobiont, including the ability of the coral host to fight infection, and the virulence of potential pathogens (Rosenberg and Ben-Haim 2002). Pollution, including nutrient loading, sedimentation and other anthropogenic stressors could further reduce the health of the coral community, causing a disruption in the symbiosis with zooxanthellae, altering the composition and virulence of the microbial community

found in the surface mucopolysaccharide layer of the coral polyp, and reducing the resistance of the coral to pathogenic organisms. The prevalence of coral diseases has been found to correlate with ocean heat and coral disease outbreaks in the aftermath of bleaching events have been widely reported (Bruno et al. 2007; Brandt and McManus 2009).

Between 1972 and 2005 coral diseases were reported on 39 genera and 148 species worldwide, with disease observations in 63 countries (Bruckner 2009b). Although Pacific reefs exhibit a much higher diversity of reef-building corals relative to the Atlantic, and they constitute about 92% of the world's coral reefs (Spalding and Greenfell 1997), only 14% of the global observations of coral disease were from the Red Sea and Indo-Pacific (Green and Bruckner 2000; Sutherland et al. 2004; Bruckner 2009a, b, c). In the Gulf and the Arabian Sea, coral diseases are not rare and outbreaks and dynamics have been noted (Coles 1994; Korrubel and Riegl 1998; Riegl 2002), but to date, little systematic or quantitative ecological work has been undertaken.

7.2.1 Yellow Band Disease

The term "Yellow Band Disease" describes a coral disease that primarily affects faviids in the Caribbean and Indo-Pacific, and *Porites* and *Acropora* in the Gulf (Fig. 7.2). In general, the term "YBD" appears to have been first used in 1994 in Florida (although not published as such) and corals manifesting similar signs have been observed and reported using different names in the past, including ring bleaching in the 1970s, yellow pox, yellow blotch disease and yellow band/blotch (Bruckner 2009a). Nomenclature developed by the Coral Disease and Health Consortium, and published in the Coral Disease Handbook (Raymundo et al. 2008), recommended the terminology Caribbean Yellow Band Disease (CYBD) for this condition in the western Atlantic. This is differentiated from a similar condition in the Pacific, termed Pacific Yellow Band Disease (PYBD). Arabian Yellow Band Disease (AYBD) was first described by Korrubel and Riegl (1998) from Dubai. Arabian YBD shows important differences from both CYBD and PYBD in gross appearance, patterns of spread and coral species susceptibility. Arabian YBD is an aggressive affliction that is very fast-spreading on *Acropora* and slower-spreading, but persistent on *Porites*. In contrast, CYBD only affects faviid corals and is characterized by very slow rates of tissue loss (1–2 cm per month). Lesions originate at the margins of colonies or they form focal lesions that are completely surrounded by normal tissue having normal appearance. Affected tissue is pale to lemon yellow in coloration, in contrast to surrounding dark green or brown tissue associated with AYBD. Over time, tissue first affected by CYBD becomes darker and dies, and the pale yellow band of blotch slowly expands outward, advancing across the colony. Rates of tissue loss from AYBD average 1–2 cm/week, while CYBD advances by only 1 cm/month or less in the Caribbean. Unless the coral is rapidly killed (which can be the case in Gulf *Acropora*), infections from both diseases may remain active for 5–10 years or more. The greatest rate and extent of tissue loss from AYBD occurs in summer, which is also observed in CYBD (Bruckner and Bruckner 2006a; Ballantine et al. 2008).

In Caribbean, Pacific and Arabian YBD, centers of infection generally coalesce with other bands as the patches of exposed skeleton increase in size, eventually killing the coral. Environmental drivers of these diseases are incompletely characterized, although infections have been reported to emerge as colonies began recovering from bleaching (Bruckner 2009a). There is some evidence from the Indo-Pacific suggesting that YBD is a disease of zooxanthellae associated with four species of *Vibrio* bacteria (Cervino et al. 2001, 2008). In some cases, the band may be associated with a microbial mat, including cyanobacteria.

YBD has been a common affliction in the Gulf and the Arabian Sea and has reached epizootic proportions over the last decade in the Caribbean, where it is devastating populations of the major framework corals in the genus *Montastraea* (Bruckner and Bruckner 2006a, b; Bruckner and Hill 2009). Similar disease signs are reported for *Diploastrea*, a faviid from Indo-Pacific reefs.

AYBD is a common affliction of corals observed throughout the Gulf and also in the northern Arabian Sea. It was first reported in 1998 from reefs near Jebel Ali in Dubai, United Arab Emirates (Korrubel and Riegl 1998). So far, there have been no published reports of AYBD from outside the Gulf region, although a single case was observed on *Acropora* on the Yanbu barrier reef in the Saudi Arabian Red Sea in 2008 (Bruckner, personal observation). The following species have so far been confirmed as affected: *Acropora downingi, A. clathrata, A. pharaonis, A. valida, Porites lutea, P. lobata, P. harrisoni, Turbinaria reniformis, Cyphastrea microphthalma*. Four other taxa of corals have also been reported with this condition in the Indo-Pacific (Sutherland et al. 2004). The disease is active in all seasons, which is in contrast to black band disease which, in the Arabian region, almost completely disappears in winter. Infections progress faster in summer (~2 cm per week) than in winter (~1 cm per week). It is the most common disease on corals in the Gulf, but rapidly decreases in importance outside the Gulf.

AYBD forms a bright yellow band of one to several cm in diameter at the interface of healthy and dead coral. In *Acropora*, it is well-developed and defined, forming a band up to a few centimeters in width. In *Porites*, it can either be a band (on the finger-like protuberances of *Porites harrisoni*) or a large yellow, greenish blotch on massive *Porites*,

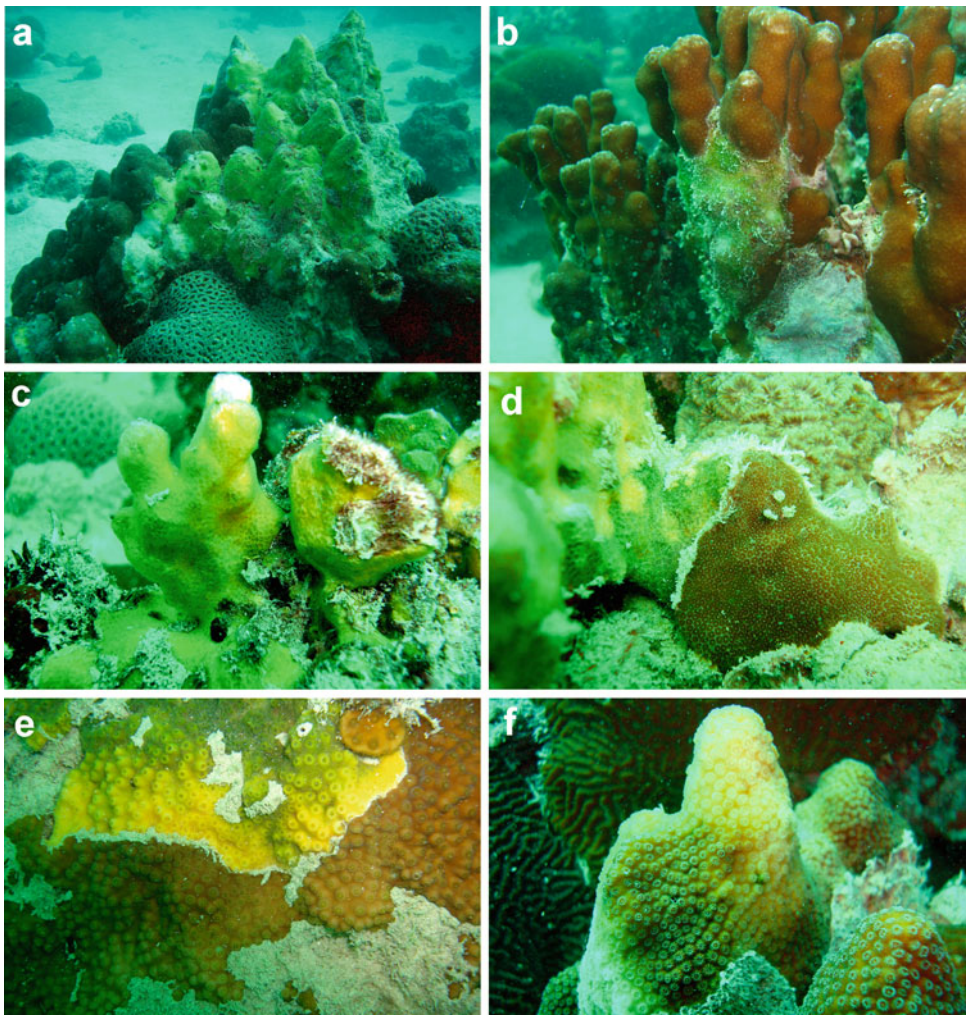

Fig. 7.2 Yellow Band Disease on massive corals: (**a**) *Porites lutea*; (**b**) *Porites harrisoni*; (**c**) *Porites harrisoni* entirely killed by YBD (Oct. 2009, Abu Dhabi); (**d**) *Porites lutea* (Abu Dhabi, May 2008); (**e**) *Turbinaria reniformis* (Abu Dhabi, May 2008); (**f**) *Cyphastrea microphthalma* (Abu Dhabi, October 2008)

depending on location. The band migrates across the coral, producing a margin of decaying tissue adjacent to healthy tissue and leaving behind dead skeleton that may retain a yellow pigmentation, remain greenly discolored (particularly in the genus *Porites*) or, in *Acropora*, is usually pure white. The yellow band is raised above the surrounding tissue and appears often to have a thin mucous envelope (it can look "puffy"); affected tissue is slimy to the touch. No filaments are visible (as in the black band), rather it appears completely homogeneous.

The AYBD seems to also have an inactive stage with different gross visible signs. Once the advance of AYBD stops and recent tissue loss is no longer apparent, the exposed skeleton may retain a green coloration; tissue immediately adjacent to the lesion has clearly stopped to die back and can even begin to lay down new skeleton to reclaim the lost area. This situation is commonly observed on *Porites harrisoni* and *Porites nodifera*. It is rarely observed in *Acropora*, which tend to be completely killed by AYBD once affected.

On *Acropora*, progress of the disease band is rapid (up to 2 cm per week in the warm season). The disease frequently initiates at the center of the colony (potentially being spread through damsel- or butterfly fish bites) and spreads outwards (Fig. 7.3). Once afflicted, the entire colony dies. Whenever adjacent colonies are in tissue-contact (as is frequently the case in dense *Acropora* thickets), the disease passes seemingly unhindered from one colony to the next. It only appears to stop where physical contact among colonies is broken.

No work to identify possible etiologic agents or experimental work to characterize linkages with environmental parameters is reported. The disease can be readily transmitted among certain corals, suggesting the presence of a microbial agent as the cause. Placement of an infected colony onto unaffected tissue of a neighboring coral is sufficient to

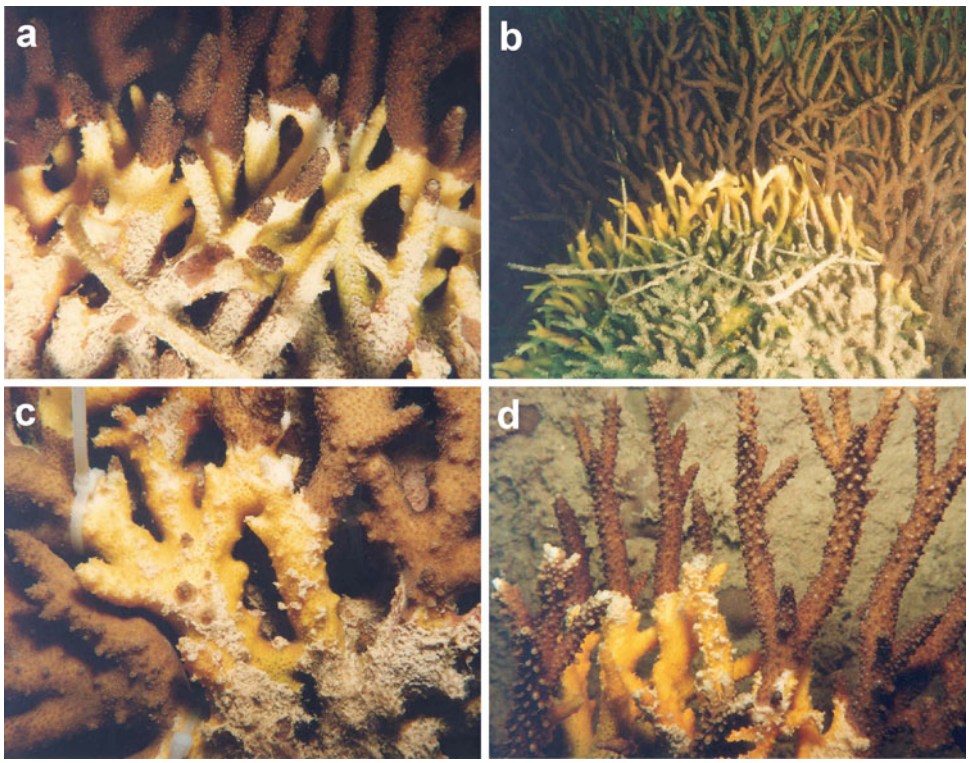

Fig. 7.3 Yellow Band Disease on *Acropora.* (**a**) *Acropora clathrata.* Cable ties were attached 1 week prior to this photograph being taken. Disease spread was ~2 cm in this week (January 1996. Jebel Ali, Dubai). (**b**) *Acropora clathrata* colony being killed from the centre outwards. This is a common pattern (January 1996, Jebel Ali, Dubai). (**c**) *Acropora downingi* marked to measure spread of YBD (January 1996, Jebel Ali, Dubai) (**d**) *Acropora pharaonis* (January 1996, Jebel Ali)

transfer and spread the disease. If infected *Acropora* branches are broken off and transplanted, so that an active, yellow band comes in contact with the healthy tissue, then infection usually takes place within a week. When inactive yellow bands (i.e. the dead but greenish stain) were placed in contact with healthy tissue, successful trans-infection was neither achieved in *Porites* nor *Acropora*. The spread of the disease can be stopped by creating a "disease break" by removing all live tissue adjacent to the yellow band. Removal of tissue either by water jet, breaking of *Acropora* branches or chiseling away sections of *Porites* tissue plus skeleton was equally successful.

The term "Yellow Band Disease" has been used to describe coral diseases from different regions and likely include conditions that are not necessarily caused by the same etiologic agent. Cervino et al. (2001) indicate that yellow band has been around for a long time, with the first reports by Dustan in the 1970s using a different name (ring bleaching). Yellow blotch disease and yellow band/blotch disease have been used interchangeably with YBD in publications about Caribbean YBD. While Cervino et al. (2008) compare Indo-Pacific YBD with Caribbean YBD, linkages between these two conditions are not fully understood. Gross signs of YBD on faviid corals from the Caribbean are similar to YBD on *Diploastrea* spp. However, gross signs on *Fungia* spp. are different, but similar etiologic agents have been observed among all tested species (Cervino et al. 2008). Four *Vibrio* species (*V. rotiferianus, V. harveyi, V. alginolyticus* and *V. proteolyticus*) have been isolated from YBD tissue in the Caribbean genus *Montastraea* and the Indo-Pacific genera *Diploastrea* and *Fungia*. Cervino et al. (2008) noted that the consortium attacks zooxanthellae within gastrodermal tissues, causing degenerated and deformed organelles and reduced photosynthetic pigments. Zooxanthellae from infected corals also had decreased cell division compared with zooxanthellae from the healthy corals. At present, it is not known whether the AYBD indeed belongs into the same group of diseases.

7.2.2 Black Band Disease

This is the oldest known coral disease and was originally described from the Caribbean (Antonius 1988), and subsequently found in all oceans (Antonius 1985). The name stems from the microbial consortium formed at the interface of healthy and diseased tissue, which is dominated by a filamentous cyanobacterium that retains a black to reddish black

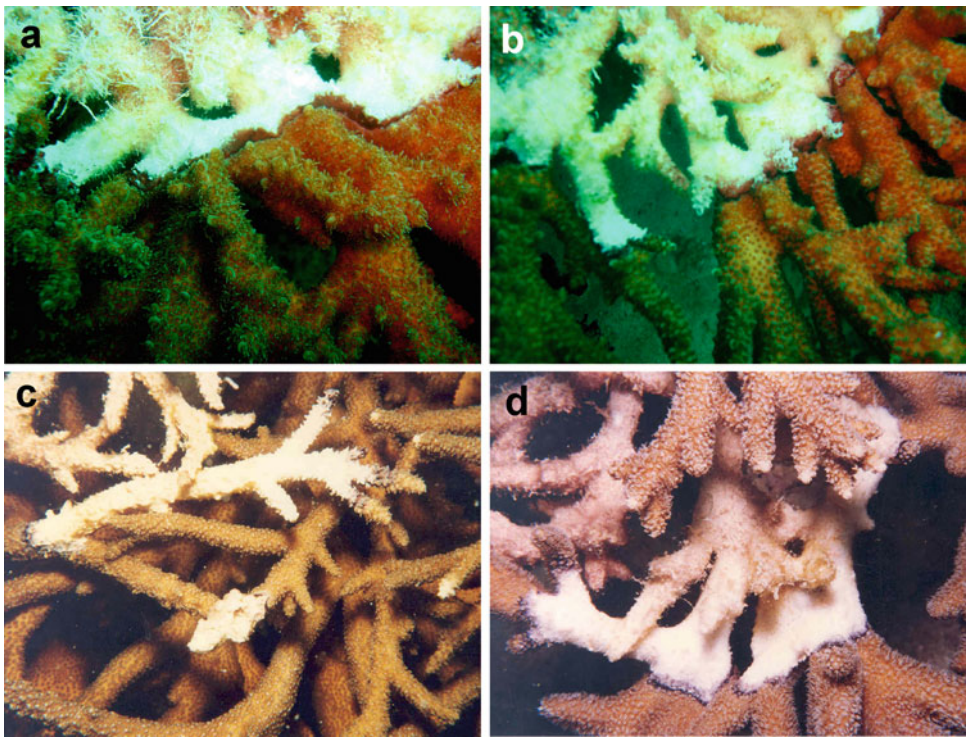

Fig. 7.4 Black Band Disease is relatively rare in the Gulf and has so far only been described from *Acropora* (**a**), (**b**) BBD on *Acropora downingi* in September 2009 in Abu Dhabi (Ras Ghanada), (**c**) on *Acropora clathrata* in September 1995, (**d**) on *Acropora downingi* in October 1995 in Dubai (Jebel Ali)

pigmentation. BBD is global in distribution. Throughout its range in the Indian and Pacific Oceans, and so also in the Gulf, this disease primarily affects *Acropora*. In the western Atlantic it has been identified on 25 scleractinian corals, 6 branching gorgonians and sea fans but not on *Acropora* spp. (Green and Bruckner 2000). BBD generally affects a low percentage of corals (<1%) at the community level (Edmunds 1991; Kuta and Richardson 1996; Bruckner et al. 1997), but it occurs in most reef environments, and localized epizootics have been observed in the USVI, Jamaica, Florida and Puerto Rico (Peters 1984; Bruckner and Bruckner 1997; Bruckner 1999; Bruckner 2002) and in 2011 in Abu Dhabi. The disease may exhibit a clumped distribution (Kuta and Richardson 1996; Bruckner et al. 1997), affecting up to ten corals within a 2 m radius area (Peters 1984). A greater percentage of the corals may be affected by BBD in areas with high coral cover, and in habitats with a high density of colonies or dominance by susceptible species, and also in areas with unusually clear water (Bruckner 1999). BBD shows similar dynamics in Arabia, although the target species differ (Fig. 7.4). It is a moderately aggressive affliction that is fast-spreading on *Acropora* and also occurs on other coral genera. Affected tissue is covered by a thick, well-defined black band of cyanobacteria and other microorganisms; the band frequently starts in the center of an *Acropora* colony or the tops of faviid colonies and slowly expands outward, as the tissue affected dies. Rates of tissue loss have not been ascertained, but are slower than in YBD. In the Gulf, BBD advances with greatest rate and extent of tissue loss occurring in summer. It becomes rare, or disappears completely, in winter (Riegl 2002). There also seems to be some density dependence in its occurrence, since the disease became rarer after the 1996 *Acropora* mass mortality. This decrease in frequency could have been a result of the disease's preferred host (*Acropora* spp.) having virtually disappeared from the area, or a reaction to the generally decreased coral frequency with resultant lessened opportunity for infection. More than one center of infection can occur on a single colony and they can coalesce with other bands as exposed skeleton increases in size, eventually killing the coral. Environmental drivers of the disease are incompletely characterized, but Antonius (1985) found increasing prevalence near a phosphate terminal.

In the Gulf, the following species have so far been confirmed as affected: *Acropora downingi, A. clathrata, A. pharaonis, A. valida*, *Favia pallida, F. speciosa, Platygyra daedalea, P. lamellina, Cyphastraea microphthalma*. It is not unlikely that the disease is as species-unspecific as it is in other regions and that more thorough search in future will reveal more dynamics than is presently known. BBD has not been reported from the Arabian Sea but likely occurs there.

In the Caribbean, BBD typically advances at rates of about 3 mm/day (Rützler et al. 1983), and occasionally increases to

a maximum of 1 cm/day (Antonius 1981). Considerable variation in spreading rates is observed over the duration of individual infections (Rützler et al. 1983) and also between species, depths, seasons and locations (Bruckner 1999, 2002). BBD occurs year round on tropical Caribbean reefs, while infections often disappear in winter months in Florida and other northern reefs, such as the Gulf, when temperatures decline below 20°C. BBD can kill small (<50 cm^2) corals in several days while larger corals experience partial mortality before signs of BBD disappear (Bruckner 2002). However, BBD may reappear later that season or the following year, and individual colonies can be affected by BBD for multiple years (Feingold 1988; Kuta and Richardson 1996; Bruckner and Bruckner 1997). While BBD does not appear to have caused large die-offs of important reef-building corals, individual colonies lose substantial amounts of tissue that may affect their reproductive potential or their ability to resist other stresses (Edmunds 1991). Kuta and Richardson (1996) noted that corals continue to lose tissue after signs of BBD disappear. In the Gulf, the first major BBD epidemic was observed in 2011, as a consequence of that year's bleaching event.

7.2.3 White Syndromes

Since the first report of White Band Disease (WBD) by Antonius (1985) in the Red Sea and Indo-Pacific, corals with similar disease signs have been reported from throughout the Pacific and Indian Oceans using a highly varied terminology including WBD, white plague, plague-like and white syndrome (Sutherland et al. 2004; Willis et al. 2004; Bruckner 2009b). Because gross signs of these conditions are similar, and they can be readily confused by mortality from other biotic agents including predation by COTS and *Drupella*, differentiation of the various types can be difficult. The term "White Syndrome" was first used in the Red Sea in 1996 and Australia in 2001. In the Gulf, syndromes that resemble these conditions are becoming increasingly common.

WBD, which was first documented on corals from the Red Sea off Saudi Arabia and Egypt in 1981, and the Philippines also in 1981 (Antonius 1985), shares many similarities with regards to species affected and patterns of tissue loss in the Gulf. Antonius (1981, 1985) reported WBD in the Red Sea on 17 genera and 31 species of corals, including 11 acroporids (Egypt, Saudi Arabia) and 22 species (13 genera) in the Philippines, including two hitherto unrecorded genera (*Montipora* and *Podabacia*). Cases were also reported over the last 10 years in Australia, Egypt, Guam, Oman, UAE, India, Malaysia, Mauritius, Palau, Papua New Guinea and the Philippines (Coles 1994; Riegl 2002). Willis et al. (2004) observed a 20 fold increase in the number of corals affected by white syndrome between 1998 and 2003, infections spread from 75% of the regions and 45% of the reefs in 1998 to all regions and 89% of the reefs by 2003 (Willis et al. 2004). Also in the SE Gulf, a locally dramatic increase in the frequency of white syndrome has occurred, in particular in the aftermath of the 2010 bleaching event (Figs. 7.5 and 7.6). A disease that is similar to white syndrome and white plague was also reported in a subtropical location (Solitary Islands) off Australia. Six coral genera were affected, with new observations for *Turbinaria* (2 species). Disease incidence in the Gulf and in the Solitary Islands varied throughout the year and was lowest in winter. This, and disease frequency was comparable to AYBD dynamics in the Gulf (Gulf AYBD: 7%, Solitary Islands WS: 6.2%) and highest in summer (Gulf AYBD: 14%; Solitary Islands WS: 13.6%; Riegl 2002; Dalton and Smith 2006).

WBD was, like BBD, originally described from the Caribbean. It played a dominant role in the precipitous (90–98%) decline of *A. cervicornis* and *A. palmata* populations during the 1980s and 1990s (Bruckner 2002; Aronson and Precht 2001; Gardner et al. 2003). It is the only disease to date that has caused major changes in composition and structure of reefs over large areas of the Caribbean (Green and Bruckner 2000). WBD was first documented on reefs around St. Croix, USVI in 1978, and later throughout the Caribbean. During the 1980s, the prevalence of WBD varied from 1–2% to 26% in the British Virgin Islands (Davis et al. 1986), up to 33% in Parguera, Puerto Rico (Goenaga and Boulon 1992), 40% in Florida and Belize (Antonius 1981), 64% in the USVI (Gladfelter et al. 1977) and as high as 80% in Jamaica and the Netherlands Antilles (Rogers 1985). It has been described from the Gulf (Riegl 2002) where, like in the Caribbean it occurs on most coral species but seems to affect some more severely. These unfortunately appear in both cases to be the dominant frame-builders (*Acropora* spp. and *Porites harrisoni* in the Gulf, and *Acropora* in the Caribbean).

7.2.4 Pink Spots and Pink Line Disease

Originally described from the Indo-Pacific, discolored tissue on acroporids that manifest as pink bands, spots or blotches have received some attention in the Gulf (Benzoni et al. 2010). Abnormal pigmentation in *Porites* characterized by circular spots, irregular blotches, lines, rings with a pink coloration can occur in response to physical and/or pathogenic stress, including fish bites, parasite infections, burrowing or boring invertebrates, and microorganisms (Ravindran et al. 2001; Ravindran and Raghukumar 2002; Willis et al. 2004; Raymundo et al. 2008). For example, pink, swollen nodules can occur on the coral colony as a reaction to trematode infections (Aeby 2003), to physical and chemical changes due to cyanobacteria (Ravindran and Raghukumar 2006), and to mechanical/chemical stress caused by barnacle larvae

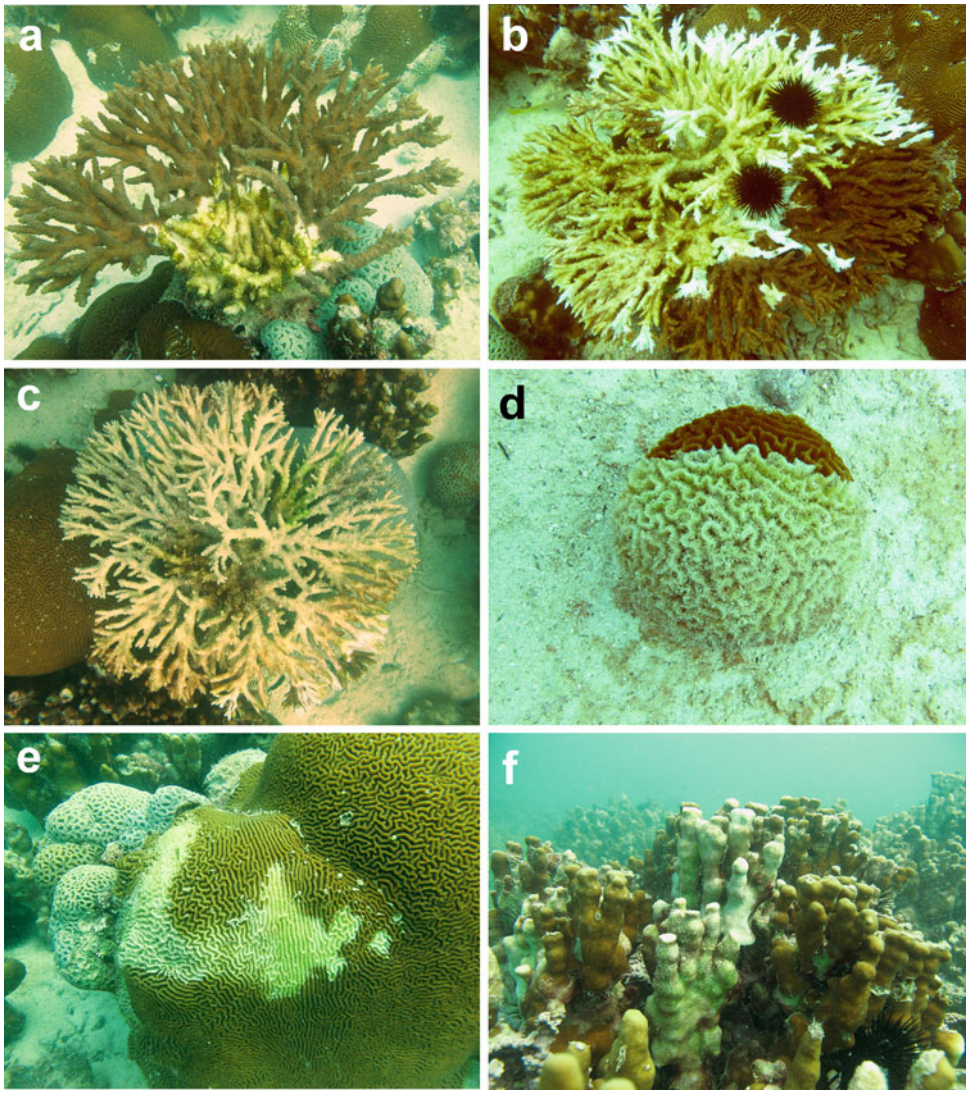

Fig. 7.5 White syndromes in the immediate aftermath of the 2010 bleaching event. (**a**, **b**, **c**) *Acropora clathrata* in various stages of infection. Images were taken about 1 month after the end of the thermal anomaly. Disease spread in colony (**c**), which has only remnant live tissues at its tips, was ~5c per week. (**d**) *Platygyra daedalea* mostly killed by a *white* syndrome. (**e**) In this *Platygyra daedalea* it is unclear whether the *white area* is remnant bleaching, or the onset of a *white* syndrome. (**f**) *Porites harrisoni* convalescing from bleaching (tips in foreground still pale) and ravaged by a *white* syndrome (*white tips* are dead)

(Benzoni et al. 2010). Pink coloration can also be the result of serpulid larvae growing on the surface of *Porites* colonies and causing mechanical and/or chemical irritation. During a catastrophic coral mortality associated with a red tide in the Gulf in 2008–2009 (Samimi-Namin et al. 2010) the pink pigmentation was found on almost all surviving massive *Porites* colonies (Fig. 7.7a). Pink coloration was mainly due to serpulid worms overgrowing the colonies surfaces (Fig. 7.7b–c) but was also observed around barnacles (Fig. 7.7d) and adjacent to recently smothered coral tissues. It is not known whether trematode larvae or cyanobacteria were also associated with these lesions.

The occurrence of pink spots may also be related to other cases of altered pigmentation such as the various dark spot/band diseases that collectively affect 14 species of coral in the Caribbean (Gil-Agudelo et al. 2004; Weil 2004) and several taxa in the Indo-Pacific and Red Sea (Bruckner, personal observation). DSD was first noticed in Colombian reefs in 1992 during a bleaching event (Solano et al. 1993; Garzón-Ferreira and Gil 1998) which is an interesting parallel to the emergence of the pink spots in the Gulf. DSD affected >16% of six species (over 1,545 colonies); the two most abundant species (*Montastraea annularis* and *Siderastrea siderea)* had the highest number

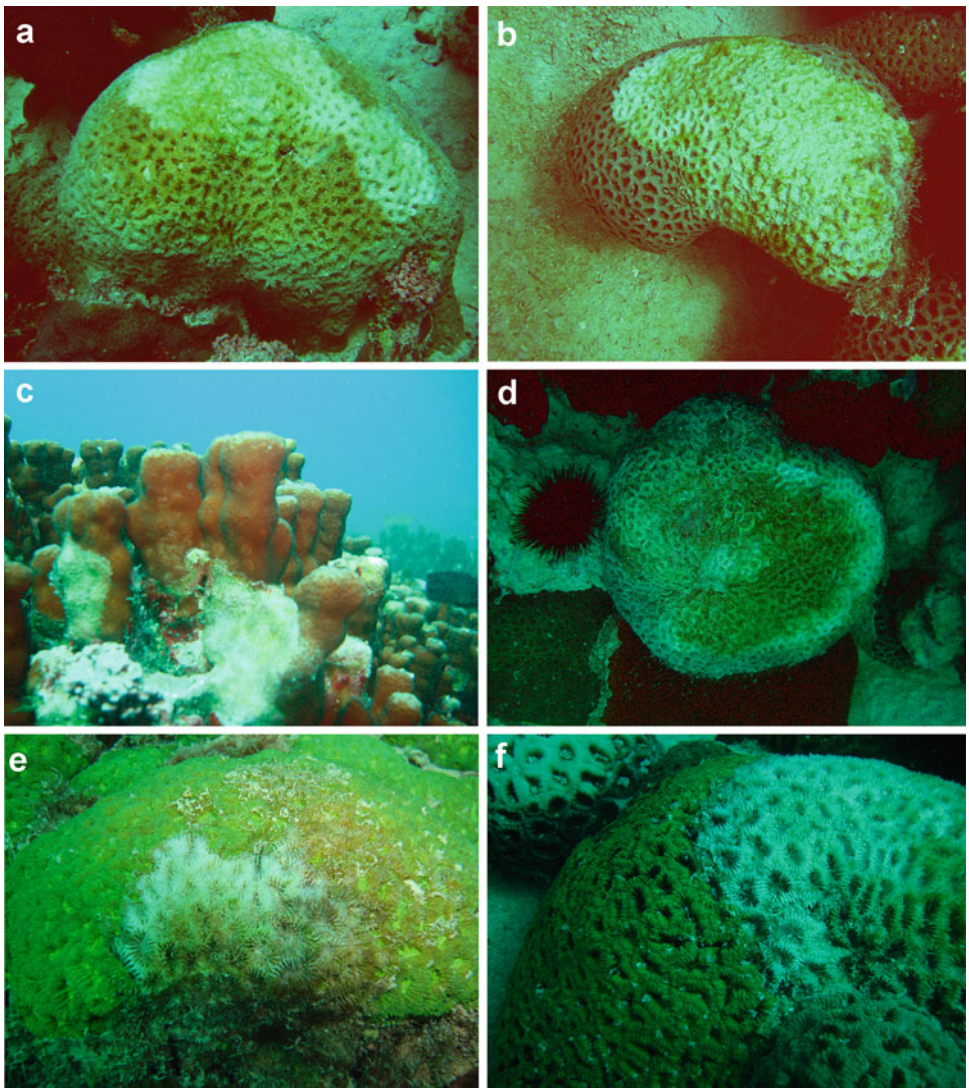

Fig. 7.6 *White* syndrome prior to the 2010 bleaching event (**a, b**) *Favia pallida* with tissue necroses from a rapidly-spreading *white* syndrome (Abu Dhabi, May 2009), (**c**) either a *white* syndrome or an unusually pale YBD on *Porites harrisoni* (Abu Dhabi, October 2008), (**d**, **e**) *Favia favus* with slowly-spreading *white* syndrome, (**f**) *Favia speciosa* with rapidly-spreading *white* syndrome. The speed of spreading can be deduced from the width of the bright *white* band. *Greenish areas* are overgrown by algal turf. The wider the *white* band, the wider the recently denuded area of skeleton and the faster the progression of the disease

of infections (Gil-Agudelo and Garzón-Ferreira 2001). Cervino et al. (2001) reported prevalence rates of 42–56% for *Stephanocoenia intersepta* and *S. siderea* in Bonaire, Turks and Caicos, and Grenada. Gochfeld et al. (2006) reported a mean prevalence of 31.5% on St. Thomas, USVI, 50.3% on Culebra, Puerto Rico, and up to 80% in the Bahamas for *S. siderea*, with the highest incidence during August and sudden declines each year in October. Galloway et al. (2007) and Work and Aeby (2008) suggest DSD to be the result of fungal invasion, while Borger (2005) noted it to be associated with stress, including physical stress like pink spots. Thus, the frequency of pink spots and DSD may be related, but evidence from the Gulf is yet to be forthcoming.

7.2.5 Ciliate Infections

Two other conditions are widely reported from the Indo-Pacific, skeletal eroding band (SEB) and brown band disease (BrBD). Brown band disease is characterized by a brown band of variable width flanked by healthy tissue at the advancing front of the disease, adjacent to live tissue, with exposed white skeleton at the trailing edge (Willis et al. 2004). The band moves in both directions along the branch, destroying coral tissue. Dense populations of ciliates, packed with zooxanthellae from coral cause brown coloration. Skeletal Eroding Band is characterized by masses of black loricae of *Halofolliculina corallasia*, a colonial

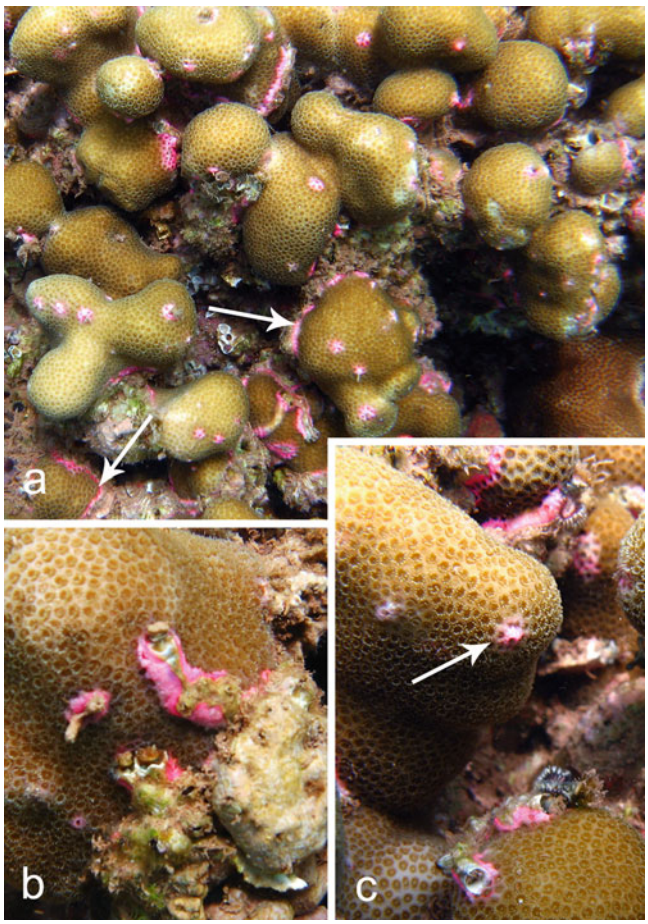

Fig. 7.7 Pink pigmentation on *Porites* colonies at Qeshm and Larak Islands, Iran, at 4 m depth. (**a**) Pink coloration is always adjacent to tissues recently smothered by serpulid worm overgrowth. (**b**) Serpulid worms overgrowing *Porites* tissues leads to pink discoloration. (**c**) Swollen coral tissues around a barnacle aperture

heterotrich ciliate embedded within the skeleton of corals. The ciliates form a front separating live tissue from a white zone of recently denuded skeleton (Antonius 1999; Riegl and Antonius 2003; Winkler et al. 2004). The front advances like BBD, causing progressive tissue loss at rates of up to 1 cm per day; it differs, though, in that it is also associated with skeletal damage from the embedded loricae.

A condition similar to SEB has been reported from the Caribbean, and both SEB and BrBD have been observed in the Red Sea (Antonius 1999; Bruckner, unpublished observation). These conditions both manifest with signs that are similar to BBD in gross appearance, which may explain why they have not been reported from the Gulf (Fig. 7.8). Also, the species susceptible to these conditions in the Indo-Pacific are among the dominant corals in the Gulf. While not yet reported, it is likely that these diseases can also be found in the Gulf.

7.3 Dynamic Models of Coral Diseases

The above review shows clearly that coral diseases are a typical feature of Gulf communities, as indeed of any coral communities in other oceans as well. In the Gulf they vary in their visible impact from almost negligible (BBD), to a regular and/or sometimes common feature (AYB, PS), to worrying epidemics taking a significant toll on local coral populations (WS, BBD, especially in the aftermath of bleaching events, such as in 2010 in the SE Gulf). Since diseases have long been identified as one of the most important regulators of animal populations (Anderson and May 1979), it is worthwhile to examine their dynamics in the Gulf from a theoretical viewpoint.

Simple disease models of the SI or SIR-type divide populations into healthy but susceptible (S) individuals, such that are infected and infectous (I) and such that are recovered and (in some cases) immune (R). SIR models have been used with great success to explain the dynamics of microparasites and hosts (Anderson and May 1979; Edelstein-Keshet 2005). Variations (Pybus et al. 2001; Mena-Lorca and Hethcote 1992; Mangel 2006) shall be used in the following to explore the dynamics of Gulf coral diseases, their effects on population and thus community dynamics and how this may change under climate change.

The distribution and dynamics of Gulf corals is largely defined by the availability of suitable substratum, such as rocky areas of capstone or, in the rare instances where these are available, true reef substrates (Riegl 1999; Purkis and Riegl 2005; Chaps. 2, 3 and 5). Thus, while an upper carrying capacity is set for corals by the environment (Riegl and Purkis 2009; Chap. 5), we can accept their birth rate, i.e. the production of gametes and larvae and presumably also the settlement rate, to be exponential. Population size would then be, if we exclude at the moment competition and space limitations:

$$dN / dt = (b - d)N \tag{7.1}$$

Where b denotes a birth-rate constant, and d is a death rate constant. Net growth rate is then $r = b - d$, leading to exponential growth when $r > 0$, stagnation when $r = 0$, and decline otherwise (which can realistically be achieved by adding competition and space limitations into Eq. 7.1). Corals will settle on all suitable substrata, and grow unfettered until competitive interaction limits their expansion when they come into physical contact with their neighbors (for dynamic expression see Riegl and Purkis 2009, and Chap. 5). Direct physical contact without aggressive reaction is sometimes possible with conspecific or congeneric individuals but rarely with less related individuals.

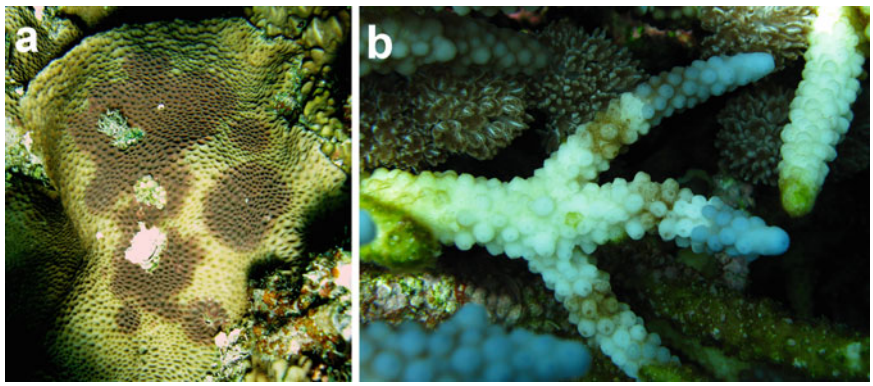

Fig. 7.8 Diseases that might exist in the Gulf but have so far been overlooked. (**a**) Brown Spots on *Gardineroseris planulata* in the Saudi Red Sea. (**b**) Brown Band Disease in *Acropora hemprichi* in the Saudi Red Sea

Table 7.1 Observed population parameters for some Gulf diseases and their relevance to persistence or outbreak in a coral population of the size given in column 1 according to the SI model. Densities were standardized to 10.000 corals. *obtained by further decomposition of eq.7.4

	N corals in diseased patch	i proportion of infected corals	β, simple mass action incidence	v, rate of loss due to death or healing	$\beta - \beta i > v$?*	Disease will spread in coral population
BBD	10.000	0.00001	0.1	0.5	<0.5	No
AYBD	10.000	0.01	0.25	0.3	0.2475 < 0.3	Mostly not
WS	10.000	0.5	0.75	0.3	0.56 > 0.3	Yes

Thus, especially *Acropora* are capable of forming dense thickets within which branches of adjacent colonies may touch or even intergrow (Fig. 7.8). Disease transmission among individual colonies will occur unhindered across such tissue bridges (personal observation). It will also occur easier among individuals that are closely-spaced than among individuals with greater distances. These observed dynamics suggests that the greater the density of corals susceptible to a diseases, the more frequent will disease transmission occur. If λ is the average number of adequate contacts for disease transmission per unit time, the dependence on density suggests a transmission coefficient β of:

$$\beta = \lambda / N \tag{7.2}$$

The total population N, consists of healthy corals that become infected (S for susceptible colonies), infected ones that can pass the disease on (I for infectious) and such that have not been killed by the disease ($1 - v$ corals are killed) and recover (R for recovered), $N = S + I + R$. The rate at which healthy individuals then become infected due to contact with infectious individuals is:

$$I' = \beta SI \tag{7.3}$$

This is known as the simple mass action principle and it assumes that a doubling of N (S, I and R) would also double the per-time unit contact rate λ. This is plausible for the situation in the Gulf: in thickets, more than one coral will be situated close to a diseased coral, so the radially spreading disease (see Sect. 7.2) could then infect several, instead of just one neighbor in the same unit of time.

It is illustrative to first consider the infective behavior of the diseases in a closed population that only consists of susceptibles (S) and infectious (I), so that $N = S(t) + I(t)$. Infections spread, as outlined above, according to the simple mass action principle (βSI which is, due to the closed population $\beta(N - I)I$) and infected individuals can be lost from the infected pool due to death or healing according to vI. Combined, this gives the equation of infection dynamics:

$$I' = \beta I\left(N - I\right) - vI \tag{7.4}$$

The similarity to the logistic equation with an additional loss term (analogy to the Schaefer-model of fisheries) is readily visible. Combining linear terms allows us to draw more conclusions. From

$$I' = I(\beta N - v) - bI^2 \tag{7.5}$$

it can be seen that only if $\beta N > v$, can the number of infected increase from their initial value.

We can use this relationship immediately to evaluate how Gulf coral diseases will behave within the community. Table 7.1 shows estimates of β based on field work and published and unpublished data from Korrubel and Riegl (1998) and Riegl (2002), approximate N values based on mapping work by Purkis and Riegl (2005) and population extrapolations by Riegl and Purkis (2009). The values in the table show clearly that BBD at its usual frequency is a rare

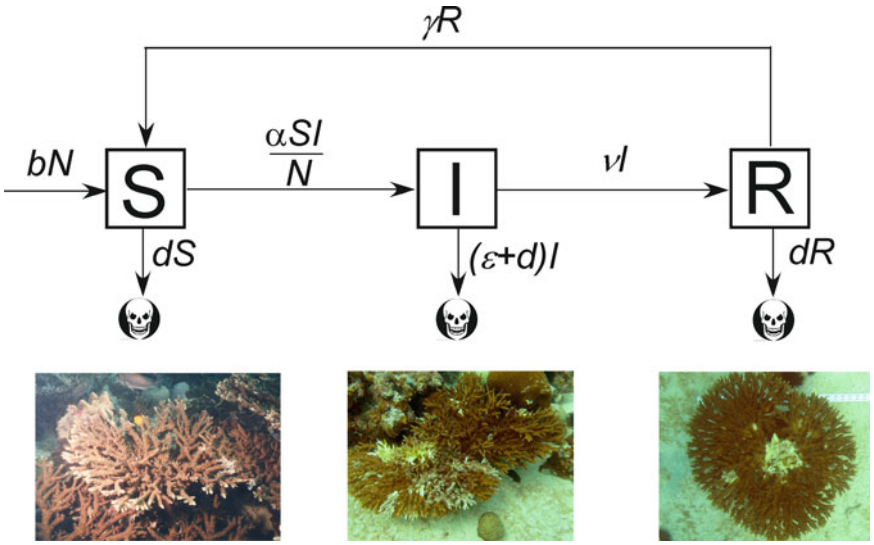

Fig. 7.9 SIRS model of coral diseases in the Gulf. *S* susceptible population, *I* infectives, *R* recovered, *d* natural mortality, *b* births, α rate of contacts, *n* rate of recovery, γ 1 – d, ε mortality due to disease, ν rate of infectives becoming resistant ('healing') N=S+I+R. *Acropora downingi* is used as model coral. Susceptibles (*S*) are in particular those corals that grow in dense thickets where often direct tissue contact occurs between adjacent colonies (image from Jebel Ali, 1995). Infected colonies (*I*) are such that have contracted the disease and show a front of active tissue loss (image from Abu Dhabi, 2010). Recovered (*R*) are colonies in which tissue loss has been arrested (image from Abu Dhabi 2010)

disease unlikely to spread and to become more common. Until 2011, it has never been observed at outbreak proportions although it can be common at high coral density (see Eq. 7.3). AYBD, although highly visible, also has not been observed to readily pass to an outbreak (Table 7.1), but in some locally constrained areas, high concentration of infected individuals as well as contagiousness has been observed. Spread to new colonies often only occurs once the originally infected colony has almost completely died, since tissue contact usually only occurs at the periphery of two touching corals. Since AYBD often spreads from the center of coral colonies towards the periphery, contagion often does not increase the number of infected individuals since the originally infected colony dies shortly after passing the disease. Locally, however, AYBD has been observed at densities high enough that would suggest them passing above threshold. WS is so far the only disease that has been observed in sufficient density and numerical frequency to make an epidemic (an outbreak exceeding a usual maximum prevalence of ~5–10% infected individuals) likely. Such a situation has occurred locally in the SE Gulf after the 2010 bleaching event. Presumably the generally weakened condition of all corals allowed a pathogen easy access to a large proportion of the population (locally >30% of all corals were found to be infected, varying in patches from 0 to >90%) and coupled with apparent contagiousness on contact, fulfilled all requirements for developing into an epidemic.

The SI model allowed evaluation of the overall behavior of the diseases, but much dynamics remains unexplored. Gulf coral populations are, for example, not closed thus we may wish to add some demographics to the above model. Not all corals always die after contracting a disease. Many cases have been observed where AYBD or BBD stopped being virulent or were successfully combated by the coral. In these cases, the coral has recovered but not obtained immunity since later re-infections have been observed. Thus we can assume that a constant proportion (γ = 1 – d, d = death rate) of recovered corals (*R*) move back into the susceptible (*S*) bin.

Above assumptions allow us to model the dynamics of a coral disease using the following formalism as a SIRS model (susceptible corals can be infected, some recover but become susceptible again; Fig. 7.9). The differential equations for such a model are

$$\begin{aligned} S'(t) &= bN - dS - \alpha SI / N + \gamma R \\ I'(t) &= \alpha SI / N - (\nu + \varepsilon + d)I \\ R'(t) &= \nu I - (\gamma + d)R \\ N'(t) &= (b - d)N - \varepsilon I \end{aligned} \tag{7.6}$$

While we have already shown theoretical differences in the persistence of the different diseases, Figs. 7.9 and 7.10 show the dynamics these diseases can impart on coral populations. One of the most sensitive parameters determining whether a coral population can sustain a disease or not, is the reproductive rate of corals in relation to the infection and mortality rates in Fig. 7.10. A threshold existed between 1.1 and 1.15 (i.e. whether the population was augmented with recruits to 10% or 15% of its overall population level) below which coral populations tended towards a strongly depressed

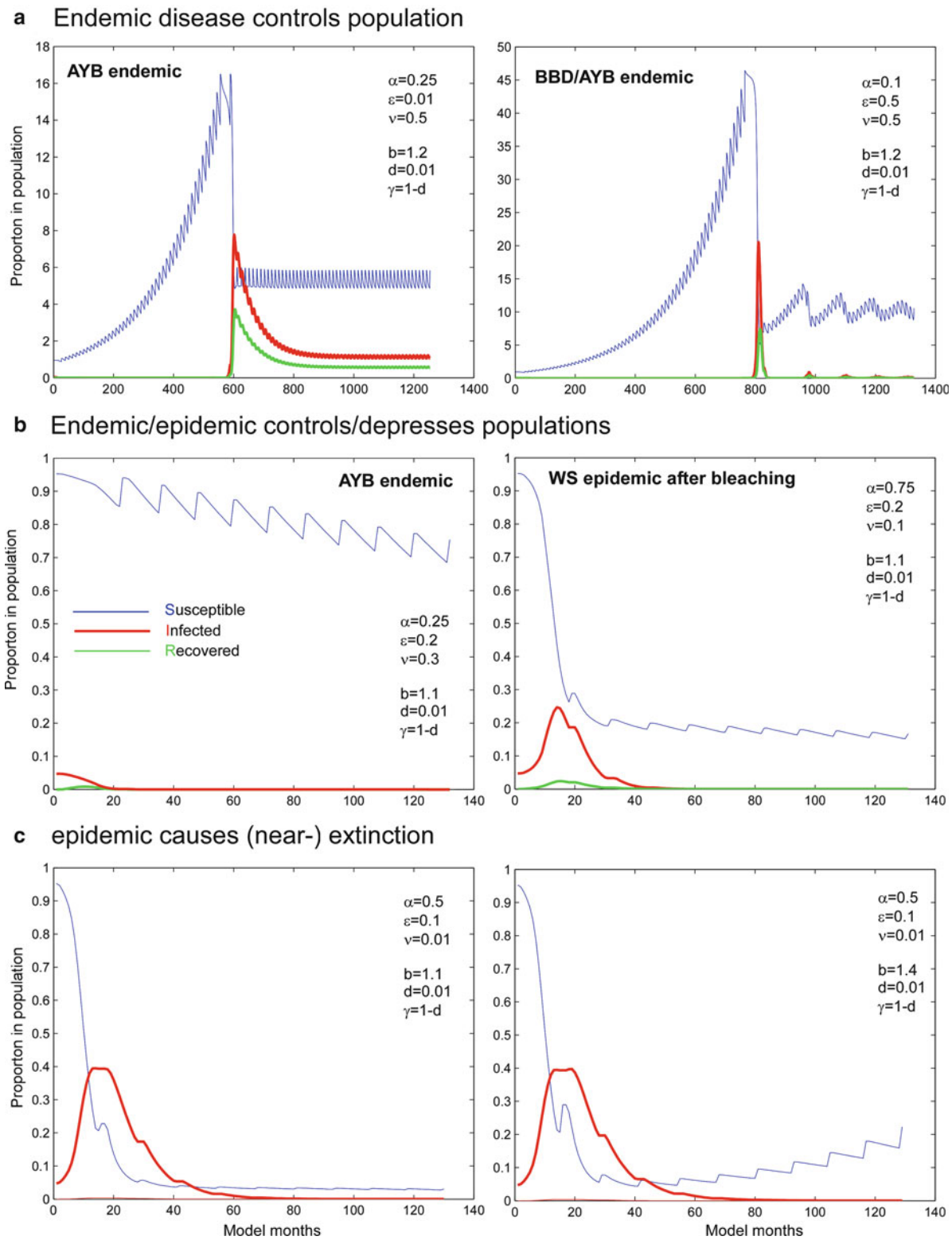

Fig. 7.10 SIRS Model results with the demographic parameters exponential reproduction (b), natural (d) and disease-induced mortality (η) added to healing (ν) and re-acquired susceptibility (γ). Movement from the recovered class into the susceptible class is automatic and no immunity is ever acquired (hence g = 1 – d). Models in (**a**) run for 100 years (1,200 months) models in (**b**) and (**c**) for 10 years (120 months). In (**a**) the proportion rises above 1, which suggests population densities at a multiple of the original starting level. Recruitment occurs as discrete event once every 12 mo (unlike Eq. 7.6) by multiplication of N by b.

population level (~20% of starting population) and above which they tended towards exponential increase. However, as Fig. 7.10a shows, once populations had multiplied several-fold, enough susceptible corals existed to trigger an outbreak of even normally rather benign diseases ($\alpha = 0.1$). After the outbreak, depending on the behavior of the disease, it coexisted with the corals either at one stable level or in dampened oscillations. Either way, the models show that diseases clearly are capable of controlling coral population levels and identify them as important demographic factors for coral population maintenance in the Gulf (Fig. 7.10b). Aggressive diseases, such as WB, form short-lived (2-year, Fig. 7.10b) epidemics that reduce coral populations to low levels at which the corals can presumably stabilize until the next epidemic. Such dynamics has been repeatedly observed in the Caribbean. Depending on the fertility level of the affected corals, diseases can indeed drive a population, at least theoretically, almost or totally to extinction (Fig. 7.10c).

7.4 Red Tides (Harmful Algae Blooms)

An increasingly potent killer of reef corals are blooms of harmful planktonic algae, also known as Red Tides or Harmful Algae Blooms. They usually occur in summer, at high surface water temperatures, low wind speeds, good light and nutrient levels (Gilbert et al. 2002). Well-known, and cyclic in their occurrence in the Arabian Sea due to large-scale oceanographic drivers, they have become increasingly frequent inside the Gulf. The blooming algae can either be toxic themselves, or significantly increase biological oxygen demand of the water upon decay after the bloom, which can result in kills of fish and coral (Al-Ansi et al. 2002). Toxins produced by the algae may also lead to fish kills, and can accumulate in shellfish (e.g. paralytic shellfish toxins; Gilbert et al. 2002) with negative commercial implications. Since surface water temperatures in the Gulf seem to be rising (Sheppard and Loughland 2002), the increasingly suitable conditions for red tides may lead to a more frequent recurrence of such events with obvious implications for reef health (Bauman et al. 2010; Samimi-Namin et al. 2010; Foster et al. 2011).

There are clear indications that the frequency of HABs is indeed increasing, with examples being reported from almost all areas (Sheppard et al. 2010; see also Chap. 16). In Kuwait Bay a HAB incident by *Karenia selliformis* and *Prorocentrum rathymum* caused a massive fish kill in 1999 (Al-Yamani et al. 2000). HAB incidences accompanied by massive fish kills have been reported from Abu Dhabi, Dubai, Ajman, Fujaira, the waters of Iran and Oman during August 2008–May 2009 (Fig. 7.11). The main HAB species causing mortality was *Cochlodinium polykrikoides* (Matsuoka et al. 2010; Richlen et al. 2010; Sheppard et al. 2010; see also Chap. 18). The recent red tide in the Gulf and Gulf of Oman started at Dibba, north-western coast of the Gulf of Oman, in August-September of 2008 and extended towards the north, reached the Strait of Hormuz at the Iranian coast, and expanded west- and southward from there (Fig. 7.11). The bloom also extended along the Omani coastline following the current patterns. The bloom was unique in terms of its duration and geographical range (Matsuoka et al. 2010). Before this bloom, the longest previous duration of bloom caused by the same species was one and one-half months in Korea (Kim et al. 2004), and almost two months in Japan (Kim and Honjo 2005).

In the affected reefs, corals (predominantly *Porites* species) suffered mortality and subsequent mass-settlement and overgrowth by serpulids. This infestation was not limited to dead surfaces, but also occurred on live corals. Three months after the red tide, infestation levels had reached 47 ± 9 serpulids 25 cm^{-2} coral, which led to the death of 90% of all local *Porites* tissues. Several other coral species were also affected with *Goniopora* sp. the only species immune to the overgrowth (Fig. 7.12). In 281 coral colonies, less than 10% percent appeared in normal condition (8.90 ± 0.26) and the rest of colonies showed partial or complete mortality (Fig. 7.13), where before red tide, the community had 40–70% live corals (Samimi-Namin, unpublished data).

The main cause for the mortality of *Porites* species was the high infestation by the serpulid worms, but for other species it was likely a combination of increase in surrounding nutrient levels, higher sedimentation, higher mucus secretion, depletion of oxygen, and higher light attenuation, which apparently favoured the settling conditions of the fouling organisms. Samimi-Namin et al. (2010) speculate that the increased food availability to the filter-feeding serpulids may have allowed their populations to explode. This suggests that their populations might be controlled by food limitation, rather than the competitive ability of the corals. Thus corals would be defenseless if release from food limitation would allow increased serpulid survival.

7.5 Discussion

The microbial afflictions of corals in the Gulf, such as diseases and harmful algae blooms, have only recently received attention and much remains to be learned. In analogy to diseases of vertebrates, corals also seem to be faced primarily with microparasites causing diseases (the microorganisms causing AYB, BBD and WS) but when weakened, or under changed environmental conditions macroparasites can also become a problem (trematode and serpulid infections causing pink spots).

Some regionally unique features are observed in the Gulf but the diseases seem to be similar to those in the other ocean basins. More importantly, only a subset of all known diseases seems to occur in the Gulf. Some diseases, like BBD and, to a lesser extent, WS seem to be seasonal, or at least

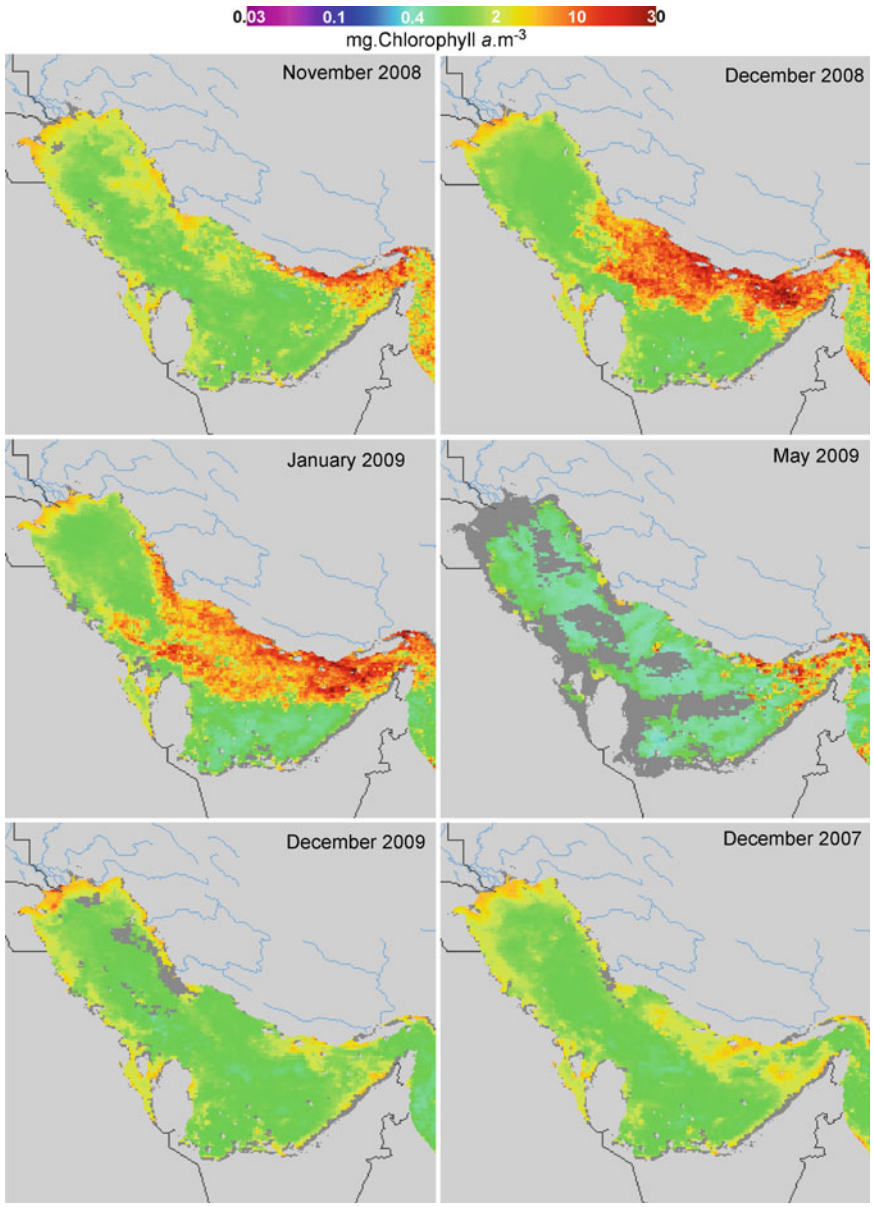

Fig. 7.11 Monthly averaged MODIS Chlorophyll *a* levels show the strong anomaly associated with the long-lasting *red tide* that caused significant coral mortality in Iran, Fujairah and Oman, and fish kills in Abu Dhabi, Dubai and Ajman over winter 2008/9. Chl a levels in December of the preceding and following year are shown for comparison (Data courtesy NOAA (http://coastwatch.pfeg.noaa.gov/erddap))

influenced by temperature. BBD has never been observed in winter, like in other high latitude areas (Florida, Kuta and Richardson 1996). WS and BBD have shown a clear spike in frequency following the 2010 and 2011 bleaching events and thus follow a pattern also observed in other places (Bruno et al. 2007; Brandt and McManus 2009) that may be linked to altered bacterial dynamics (Rosenberg and Ben-Haim 2002).

AYBD differs from all other diseases in not being affected by seasonal or extreme temperature excursions. No increase was noted after the 1996, 1998, 2002, or 2010 bleaching event. Rather, the disease decreased markedly in frequency after 1996/8 probably largely due to the reduced availability of victim corals, that had died during the extreme bleaching and mortality events of that year, suggesting simple mass action dynamics of spread.

The obvious differences in the dynamics of AYBD, BBD and WS allowed several interesting questions to be theoretically pursued. AYBD is a disease that persists at relatively high levels in coral populations, BBD is rare when corals (esp. *Acropora*) are at low density, but WS can only generate transient, albeit devastating, outbreaks. The density dependence of disease frequency observed by Riegl (2002) strongly supports the mass action principle to govern infection and the use of SI and SIRS models (McCallum et al. 2001; Edelstein-Keshet 2005). Exploration of such a model showed coral diseases as potentially important drivers of community dynamics in Gulf reefs. The force of infection (parameter β in Eq. 7.5 or α in Eq. 7.6 and Fig. 7.10.) relates to the speed of disease spread (strictly speaking it is "the instantaneous per capita rate at which susceptible individuals

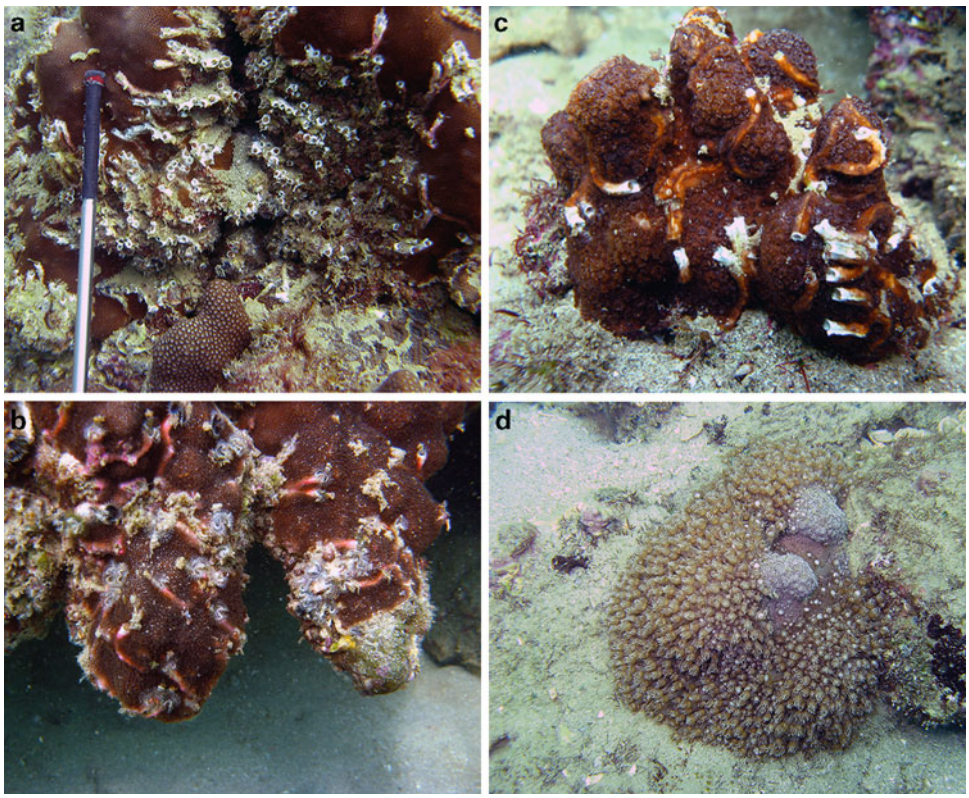

Fig. 7.12 (**a**, **b**) High density of serpulid worms on *Porites* colony, (**c**) overgrowth by serpulid worms on a *Cyphastrea* colony, (**d**) unaffected *Goniopora* colony. Scale in (**a**) = 3 cm

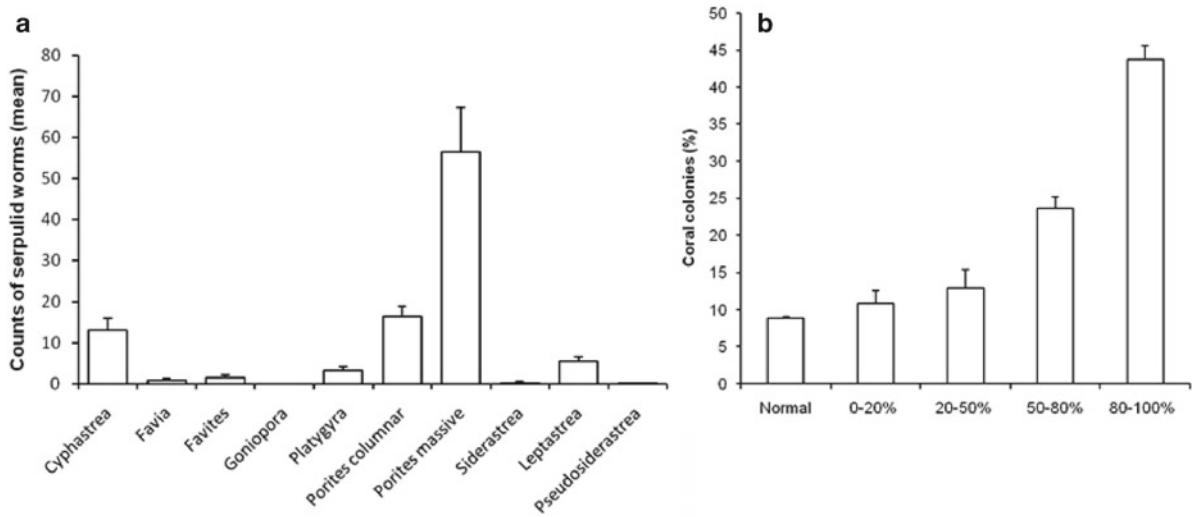

Fig. 7.13 (**a**) The infestation levels of serpulid worms on coral colonies on 100 cm^2 (n = 10) of coral. (**b**) The percentage of coral mortality about 3 months after starting of the red tide

acquire the disease"; Grenfell and Anderson 1985) and in the case of the Gulf is WS>AYBD>BBD. The measured rate of spread in WS after the 2010 bleaching events was at least twice that of YBD and many times that of BBD in a near-equilibrium *Acropora*-dominated community before the 1996 event (Riegl 1999). Also, mortality due to WS in *Acropora* was locally near-total in 2010, while in 1995 at least some colonies showed the capability to survive AYB and BBD was not observed to kill the majority of infected corals, probably due to a temperature refuge in winter when the disease is inactive.

The high force of infection in WS and its efficiency in killing the host, coupled with a low outbreak density may suggests that WS may not maintain itself as an endemic disease at low level in a population but that a true infection into the coral population has to take place from the outside every time. Recent findings clearly linked incidence of coral disease with bleaching (Brandt and McManus 2009) and found that otherwise harmless strains of bacteria can become virulent at raised temperatures (Rosenberg and Ben-Haim 2002). Also the specific pathogens causing WS and other "white diseases" have remained elusive. This suggests several possible mechanisms triggering WS outbreaks. Firstly, WS may be linked to otherwise non-pathogenic micro-organisms that become virulent at the raised temperatures causing the bleaching event. Or, weakened from the bleaching event, corals lose resistance to an otherwise only mildly pathogenic organism that now can overwhelm the corals' compromised immune system. Or, by coincidence, pathogenic bacteria may arrive at the same time in the system as the bleaching disturbance and, besides being virulent anyway, can now exploit the corals' weakened state (in a form analogous to a superinfection; Nowak and May 1994).

The dynamics of AYBD is markedly different. This is the most common disease in the Gulf and has a different outbreak threshold. N_T (the threshold number of corals per defined area required for an outbreak) is within the realm that can realistically be reached by Gulf coral populations and although the frequency of AYBD is density-dependent it also persists in sparse coral populations. This suggests that alternations of endemic and epidemic phases (as shown in Fig. 7.9a) are indeed plausible. While AYBD was observed on a variety of genera, it clearly favors *Acropora* spp. and its outbreak cycles appear to be primarily driven by these species' dynamics. At high *Acropora* density, as can be achieved after only a decade or two without major disturbances, outbreaks of AYBD may have an important function as compensatory mortality. Riegl and Purkis (2009) have shown *Acropora*, faviids and poritids to exist in a competitive environment that tends to favor dominance by the aggressive and fast-growing *Acropora*, unless faviids and poritids are allowed to reach a size- or density refuge in which they can no longer be displaced by the superior competitor. Cyclic outbreaks of AYBD at high *Acropora* density would thus exert an important "culling" effect that serves not only to control the disease, but also allows inferior competitors to reach a size-refuge, thus maintaining community diversity. Such dynamics would strengthen and potentially even overprint the purely environmentally-driven dynamics envisaged by Riegl (1999), Purkis and Riegl (2005) and Riegl and Purkis (2009).

BBD in the Gulf is a disease with a very low force of infection and its theoretical N_T is so high that is unlikely to be achieved. However, it can be achieved (Fig. 7.10) and also BBD could have a long-term effect as coral population regulator. In 2011, it was for the first time observed at outbreak levels following a bleaching event.

Climate change is likely to have strong influence on the herein discussed interplay of corals with their diseases. Positive thermal anomalies have markedly increased in the region (Nasrallah et al. 2004; Al-Rashidi et al. 2009) and with them bleaching, or at least thermal-stress events. While in the late 1990s and early 2000s it was bleaching that led to marked coral mortality, the 2010 event was primarily marked by mortality of corals regenerating from the bleaching and infected by WS. No such observations exist for the 1996 or 1998 event, and it is possible that these epidemics are a novelty. Figure 7.10 shows how devastating these disease events can be on coral populations. If they were to recur at every thermal anomaly, coral populations could potentially be depressed to a level at which at least functional, if not total, extinction was easily within the realm of the possible. Since corals are apparently at their most vulnerable to diseases when subjected to other stressors, it should be attempted at all costs to reduce stress levels on the Gulf's corals and reefs. This can be achieved by rigorous control of overfishing, pollution, and reduction of construction in the coastal zone.

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