Vol. 106: 69–77, 2013
doi: 10.3354/dao02634

DISEASES OF AQUATIC ORGANISMS
Dis Aquat Org

Published September 24

Coral diseases are major contributors to coral mortality in Shingle Island, Gulf of Mannar, southeastern India

T. Thinesh[1,2,*], K. Diraviya Raj[1], G. Mathews[1], J. K. Patterson Edward[1]

[1]Suganthi Devadason Marine Research Institute, 44 Beach Road, Tuticorin 628 001, Tamil Nadu, India
[2]*Present address:* Centre for Ecological Sciences, Indian Institute of Science, Bangalore 560012, India

ABSTRACT: The present study reports coral mortality, driven primarily by coral diseases, around Shingle Island, Gulf of Mannar (GOM), Indian Ocean. In total, 2910 colonies were permanently monitored to assess the incidence of coral diseases and consequent mortality for 2 yr. Four types of lesions consistent with white band disease (WBD), black disease (BD), white plaque disease (WPD), and pink spot disease (PSD) were recorded from 4 coral genera: *Montipora*, *Pocillopora*, *Acropora,* and *Porites*. *Porites* were affected by 2 disease types, while the other 3 genera were affected by only 1 disease type. Overall disease prevalence increased from 8% (n = 233 colonies) to 41.9% (n = 1219) over the 2 yr study period. BD caused an unprecedented 100% mortality in *Pocillopora*, followed by 20.4 and 13.1% mortality from WBD in *Montipora* and *Acropora,* respectively. Mean disease progression rates of 0.8 ± 1.0 and 0.6 ± 0.5 cm mo^{-1} over live coral colonies were observed for BD and WBD. Significant correlations between temperature and disease progression were observed for BD ($r = 0.86$, $R^2 = 0.75$, $p < 0.001$) and WBD ($R^2 = 0.76$, $p < 0.001$). This study revealed the increasing trend of disease prevalence and progression of disease over live coral in a relatively limited study area; further study should investigate the status of the entire coral reef in the GOM and the role of diseases in reef dynamics.

KEY WORDS: *Montipora* · *Pocillopora* · *Acropora* · *Porites* · White band · Disease · Progression · Laccadive Sea · Indian Ocean

INTRODUCTION

Because coral reefs occur near the junction of land, sea, and atmosphere, their natural habitats experience both the marine and terrestrial results of any climatic change and are vulnerable to human activities. Although reef communities have been described as 'disturbance-adapted' ecosystems (Hughes & Connell 1999),coral reefs have declined over the past 20 to 50 yr (Pandolfi et al. 2003). The causes of this decline are a complex mixture of direct human-imposed and climate-related stresses, and include factors such as outbreaks of disease that can reduce coral abundance (Aronson & Precht 1997, Porter et al. 2001) and diminish reproductive potential (Edmunds 1991, Kuta & Richardson 1997).

Disease has been described as the absence of health and as an alteration in general morphology that can be observed visually (Raymundo et al. 2008). Global mortality of corals because of diseases has increased in the recent past (Wilkinson 2000, 2002, Hughes et al. 2003). Caribbean coral reefs, in particular Acroporidae, have been particularly devastated, and have suffered an 80% regional decline in coral coverage during the last 3 decades (Gardneret al. 2003). Coral diseases have been increasing

*Email: thina.sathesh@gmail.com

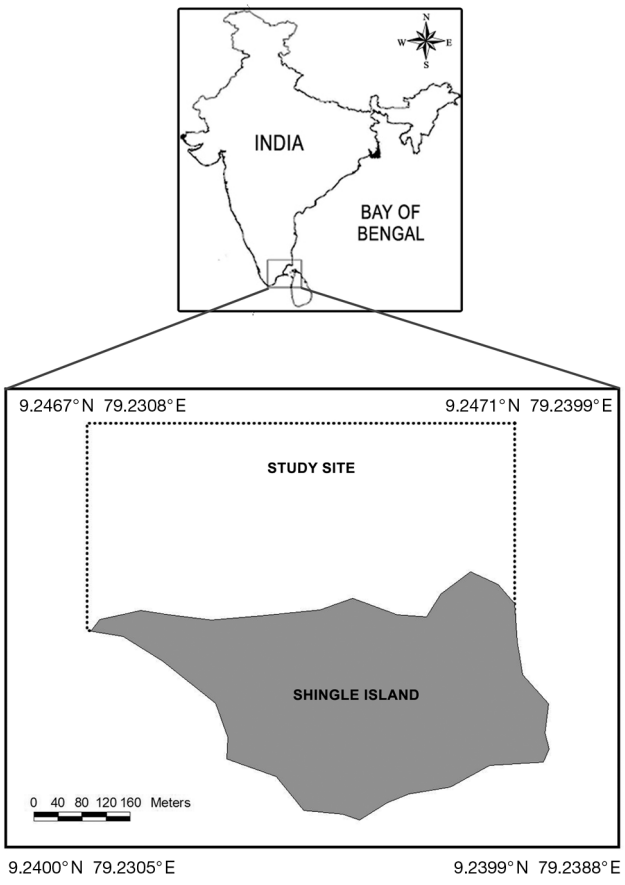

Fig. 1. Study area: Shingle Island, Gulf of Mannar, Indian Ocean

in frequency in the Atlantic and Pacific Oceans (Harvell et al. 2004). As a result, disease has become a main concern in coral health (Willis et al. 2004).

Climate change (Harvell et al. 2002), water pollution (Szmant 2002), and over-fishing (Jackson et al. 2001) are the major environmental stress factors responsible for the increase in the incidence of coral diseases. Physical properties of the environment could have an effect on the progression and transmission of coral diseases (Santavy & Peters 1997, Bruckner 2002). These conditions may decrease host resistance and increase the virulence of coral pathogens (Bruckner & Bruckner 1997a, Goreau et al. 1998). Many studies have reported the link between environmental parameters and disease (Haapkylä et al. 2010, Aeby et al. 2011). Despite the major ecological impacts of coral diseases, the etiology of most coral diseases still remains unclear. Mechanisms and pathways involved in transmission of a pathogen, infection, and subsequent mortality are poorly understood.

The Gulf of Mannar (GOM), Indian Ocean, covers an area of 10 500 km^2, includes 21 coral islands (8°N, 79°E), and supports 94 species of corals belonging to 37 genera. In total, 3600 species of flora and fauna support more than 100 000 inhabitants from 47 villages along the coast. However, its proximity to the mainland and coastal population pressure pose threats to the ecosystem. Coral reefs in the GOM occur mainly around all of the 21 uninhabited islands, among which 2 islands (Vilanguchalli and Poovarasanpatti) have sunk underwater because of coral mining. Coral is used for construction material and as raw material for the lime industry, as well as for ornamental purposes. Many studies have investigated the coral reefs and associated resources in the GOM, especially in the area of Mandapam (Pillai 1971, 1996). Degradation in the GOM has been heavy in the past few decades due to mining; over 32 km^2 of coral reef have already been degraded around the 21 islands of the GOM (Venkataramanujam et al. 1981, Shepard & Wells 1988, Patterson Edward et al. 2008).

Recently, reefs in the GOM area have been recovering well due to proper protection and awareness creation; however, coral diseases have been identified as a recent threat for the survival of corals in the GOM. Many diseases have been observed causing harm to these corals (Thinesh et al. 2008, 2009). Although extensive research has been carried out on coral reefs and associated biota in the GOM and Palk Bay, very little is known about coral diseases. The present study examined the types and frequency of occurrence of coral diseases on Shingle Island (Fig. 1), the northernmost of the 21 islands in the GOM. In addition, we also examined the *in situ* progression rates of black disease (BD) overgrowth and white band disease (WBD).

MATERIALS AND METHODS

The 4 types of lesions consistent with previously described coral diseases were identified by following the coral disease handbook of Raymundo & Harvell (2008). Assessment was carried out on Shingle Island (Fig. 1) from January 2009 to December 2010. Five permanent monitoring sites were haphazardly selected to quantify disease signs in coral colonies. Three 20 × 4 m transects each were placed end to end parallel to the reef crest, with a gap of at least 20 m between transects. Permanent buoys with permanent markers were placed at both ends of each transect to replicate the same transects each time for

monthly monitoring. Total healthy, diseased, and dead colonies were counted every month to calculate disease prevalence (percent corals with lesions), incidence (newly infected colonies per month), and coral mortality (Raymundo & Harvell 2008).

Progression rate over a live coral colony was measured for BD and WBD on the genera *Pocillopora* and *Montipora*, respectively. To measure the rate of disease progression along coral colonies (English et al. 1997), colored cable ties were used to mark the original position on bare skeleton of the lesion, and the distance from the initial cable tie to the new lesion front was measured in subsequent surveys using Vernier calipers. To crosscheck the progression rate, monthly measurements were also taken from selected permanent reference points. The density of the newly added coral recruits (newly added corals that can be detected in the field visually) was also recorded monthly along each transect. Physical parameters such as temperature and nutrient parameters such as phosphate and nitrate concentrations were measured at the diseased sites using standard methods (Grasshoff et al. 1999). The pour plate technique was used to estimate the total bacterial count in marine water samples (Vanderzant & Splittstoesser 1992). The correlation between water temperature and disease progression rate was analyzed by Pearson correlation analysis. Mean value was calculated by monthly prevalence.

RESULTS

A gradual increase in disease prevalence was noticed at the study site during the 2 yr study period. Observed diseases are described in Table 1 following Work & Aeby (2006). Out of 2910 examined colonies (*Pocillopora* 750, *Montipora* 1520, *Acropora* 320, *Porites* 320), 41.9% (n = 1219) were diseased by the end of the study period during December 2010. This percentage had increased from the initial amount of 8% (n = 233) during January 2009 while during December 2009 it was 26.9% (n = 782).

Coral mortality of 90.6% was observed for total affected colonies. The intensity of coral mortality varied significantly between coral genera. The highest mortality was observed in the genus *Pocillopora* (100%), caused by BD, followed by the genus *Montipora* (20.4%), caused by WBD, and the genus *Acropora* (13.1%), caused by WBD. The lowest mortality was observed in the genus *Porites* (0.9%), caused by pink spot disease (PSD) and white plaque disease (WPD). Underwater photographs of coral diseases are given in Fig. 2.

The details of the disease prevalence trend for each disease type during the monitoring period are given in Fig. 3. These 4 diseases (BD, WBD, PSD, and WPD) were observed with various percentages in the 4 coral genera (*Pocillopora, Montipora, Acropora*, and *Porites*), and among these, BD was dominant at 25.8% with a mean value of 16.5 ± 8.0% followed by WBD at 13.7% in *Montipora* with a mean value of 10.5 ± 3.0%. PSD and WPD percentages were 1.9 and 0.5%, with mean values of 2.1 ± 0.5 and 0.8 ± 0.4%, respectively. Within these observed genera, *Porites* was found to be affected by 2 diseases (WPD and PSD), while the other genera were affected by only 1 type of disease: WBD in *Acropora* and *Montipora* and BD in *Pocillopora*.

All of the observed coral diseases were found to have increased from the initial observations at all study sites. However, variation in disease prevalence was observed between genera for different diseases. BD on *Pocillopora* colonies increased from 7.3 to 100% during the course of the study period. WBD also increased from 8.2 to 22.2% and 4.1 to 19.4%, respectively, for the genera *Montipora* and *Acropora* during the monitoring period.

Table 1. Observed disease descriptions. Ex: extent; Lo: location; Di: distribution; Ti: time; Le: lesion; St: structure(s) affected (see Work & Aeby 2006, their Fig. 1 and Table 2)

Observed lesion	Fig.	Description/morphologic diagnosis
Pink spot disease	2a	Moderate (Ex); colony-wide (Lo); multifocal (Di); tissue loss (Ti); discoloration (pink color) (Le); polyp, coenosarc (St)
White plaque disease	2b	Severe (Ex); basal to peripheral (Lo); diffuse (Di); acute (Ti); tissue loss (Le); polyp, coenosarc (St)
Black disease	2c	Severe (Ex); basal (Lo); diffuse (Di); acute (Ti); tissue loss, black colored smothering mat with annular margin (Le); polyp, coenosarc, skeleton (St)
White band disease	2d	Severe (Ex); basal (Lo); diffuse (Di); acute to subacute (Ti); tissue loss (Le); polyp, coenosarc (St)

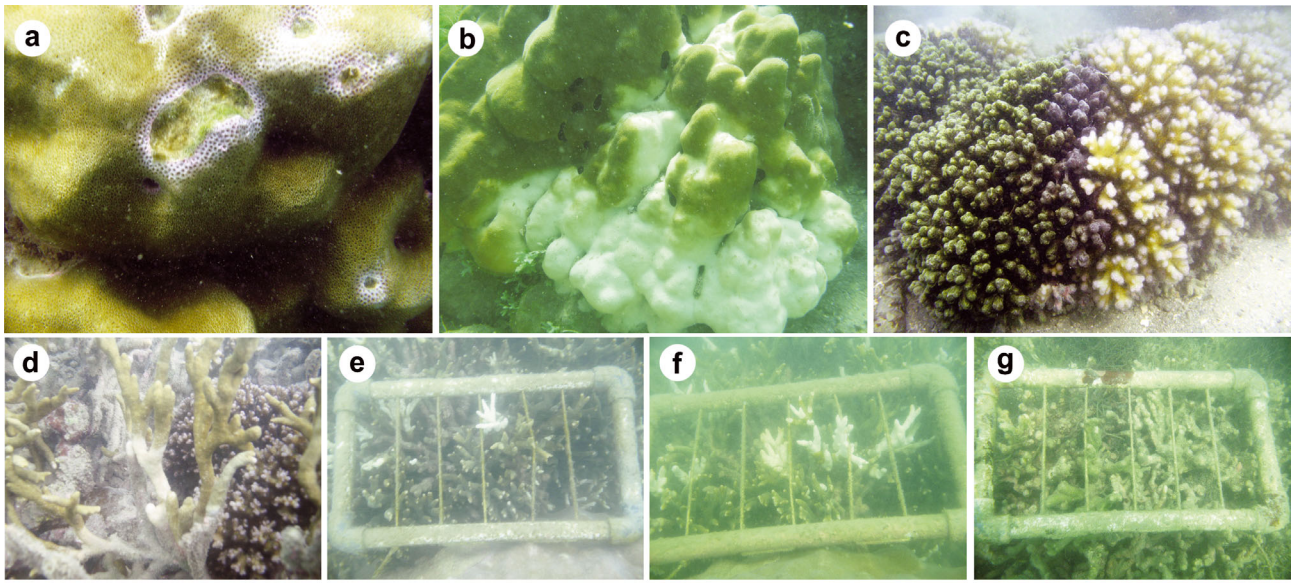

Fig. 2. Underwater photographs illustrating the appearance of observed disease signs and progression of white band disease in *Montipora* within a quadrat. (a) Pink spot disease; (b) white plaque disease; (c) black disease; (d) white band disease. (e) Initial stage of white band disease within a quadrat; (f) secondary stage (progression over colony branch); (g) final stage (dead colonies covered by secondary algae)

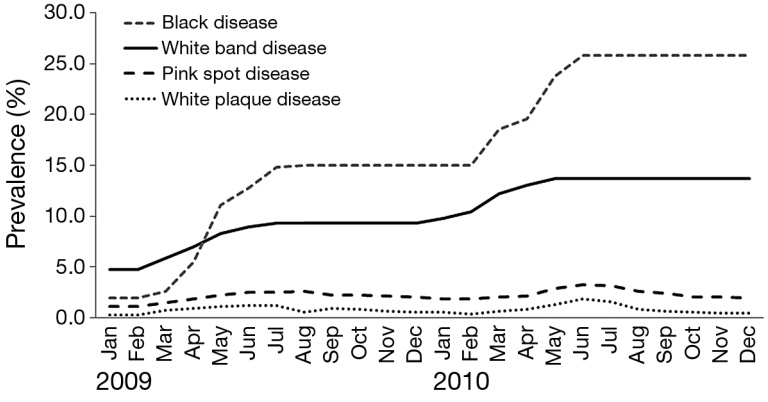

Fig. 3. Overall prevalence (% of corals affected) of observed coral diseases during the monitoring period (January 2009 to December 2010)

Disease incidence (percent corals with new lesions per month) values for each observed disease were measured during the monitoring period. For BD, maximum incidence value 21.5% was observed when the temperature reached 32.5°C. For WPD, the maximum value was 5% when the temperature reached 30.5°C. For WBD and PSD, maximum incidence values 2.8 and 4.1%, respectively, were observed when the temperature exceeded 30°C. Disease incidence decreased to 0 (no incidence) for all of the observed diseases when the temperature was around 28°C. A significant positive correlation was detected between temperature and disease incidence for BD (R^2 = 0.71, $p < 0.0001$), but no significant correlation was detected between temperature and incidence of other lesions (p = 0.2, 0.12, and 0.11 for WBD, PSD, and WPD, respectively).

Details of the correlation between temperature and diseases are given in Fig. 4. The monthly progression of BD and WBD over live colonies was measured during the 2 yr study period. For BD, a mean progression value of 0.8 ± 1.0 cm mo^{-1} with a maximum value of 3.3 cm mo^{-1} was noticed in the genus *Pocillopora* during May 2010 when the temperature exceeded 31°C. The lowest progression rate was observed during September 2009 with 0.1 cm mo^{-1}. No progression of BD or WBD was observed during the months of October, November, and December in both study years, when the temperature was between 27 and 28°C. For WBD, a mean progression value of 0.6 ± 0.5 cm mo^{-1} with a maximum value of 1.5 cm mo^{-1} was observed during May 2009, followed by 1.4 cm mo^{-1} during May 2010 in the genus *Montipora*. The lowest progression rate was observed during January 2009 with 0.1 cm mo^{-1}. The progression rates of both BD and WBD were significantly and positively associated with seawater temperature (Fig. 5). Mean temperatures were 29.4 ±

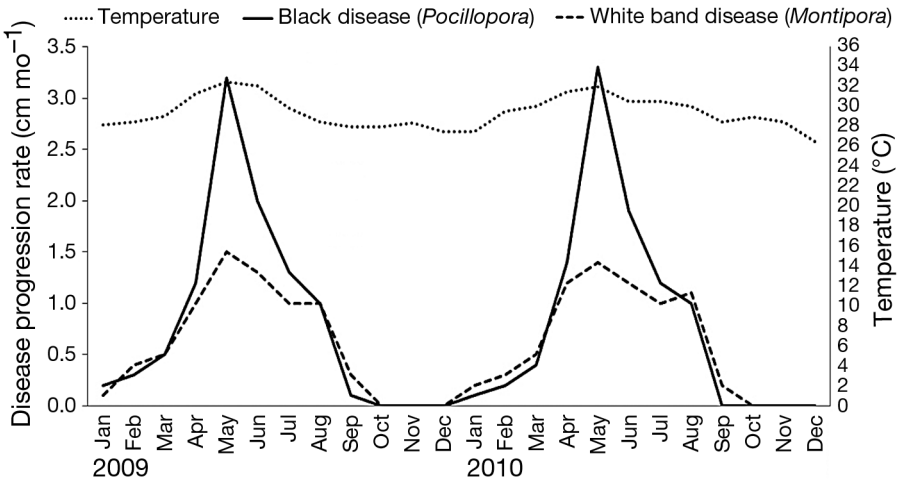

Fig. 4. Changes in disease progression rate of black disease in *Pocillopora* and white band disease in *Montipora* during the monitoring period (January 2009 to December 2010

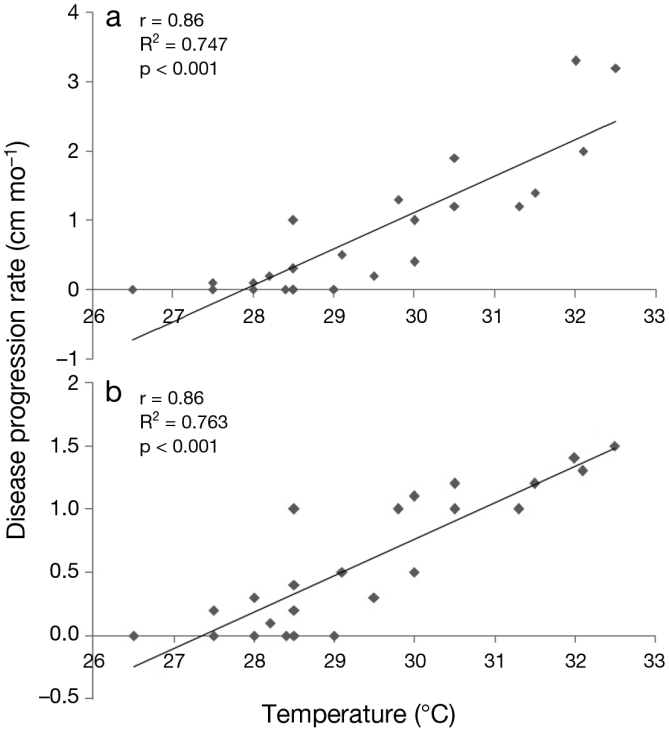

Fig. 5. Relationship between increasing temperature and (a) black disease progression in *Pocillopora*, (b) white band disease progression in *Montipora*

1.6°C, with highest temperatures observed during May 2009 (32.5°C) and the lowest during January 2010 (27.5°C).

New coral recruits were noticed from August 2009, and the recruit density varied according to the genus. Recruits of the genera *Montipora*, *Acropora*, and *Porites* were observed predominantly. The numbers of recruits of *Montipora* were high, with 25 recruits during June 2009 followed by 20 recruits for *Acropora*. Only 3 recruits were recorded for the genus *Porites* during June 2009. The density of recruits increased significantly since the initial observation. Over the entire study period, the highest number of recruits was observed for the genus *Montipora* (160 recruits) followed by the genera *Acropora* and *Porites* (62 and 15 recruits, respectively). Notably, no recruits were found for the genus *Pocillopora*. Numbers of newly added recruits during the study period are given in Fig. 6.

Mean values of 1.3 ± 0.9 µg l^{-1}, 1.62 ± 0.9 µg l^{-1}, and 37 ± 5.6 (× 10^4) CFU ml^{-1} were observed for phosphate and nitrate concentrations, and total heterotrophic bacterial count, respectively. No significant correlations were observed between disease incidence and these parameters (data not shown).

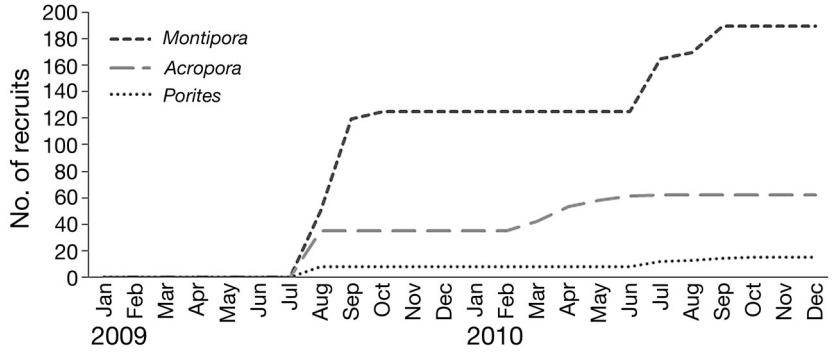

Fig. 6. Cumulative number of recruits (newly observed colonies) at the study site during the monitoring period (January 2009 to December 2010). No recruits were found for the genus *Pocillopora*

DISCUSSION

Increases in the prevalence of coral diseases have been documented in many reef regions. For example, the prevalence of coral diseases on reefs in Dominica (West Indies) increased from 8.75 to 15.32% in 2 yr at 1 study site (Coral Gardens); at a second site (Salisbury), these values increased from 2.62 to 5.62% (Borger & Steiner 2005). In the Florida Keys, USA, disease prevalence ranged from 1 to 28.2% (Santavy et al. 2001). Our research results are consistent with other studies; we observed an increase in disease prevalence from the initial observation of 8.1 to 41.9% during the course of our study.

WBD has been reported to affect many genera, predominantly affecting *Acropora* in many reefs (Bruckner 2005), whereas it has been reported less often in *Montipora* (Antonius 1985). Prevalence of WBD of 20 to 33% was reported in Puerto Rico and up to 40% in Florida (Davis et al. 1986). In our study, the genera *Montipora* and *Acropora* were the only ones affected by WBD, and prevalence was found to increase from 4.7 to 13.7%. *Porites* has been reported to be a susceptible host for multiple diseases including WPD and PSD (Raymundo et al. 2005, Haapkylä et al. 2007), and indeed, *Porites* was the only genus affected by 2 types of disease (WPD and PSD) in our study. Disease prevalence in *Porites* increased from 1.4 to 2.4%, but these values are lower than the disease prevalence in *Porites* colonies of 20% reported at a Philippines reef (Raymundo et al. 2003).

Coral community losses of 30 to 80% caused by BD have been observed on reefs in Guam (Plucer-Rosario 1987) and more recently in Taiwan (Chen et al. 2009). Overall prevalence of BD increased from 1.9 to 25.8%, and BD prevalence in *Pocillopora* increased from 7.3 to 100%. In our study, BD only affected *Pocillopora*; the other coral genera were not affected by this syndrome, and other observed diseases did not affect *Pocillopora*, although all of the observed diseases and coral genera occurred close together. This could be due to the variation in physiological and immunological responses of corals against diseases and other stresses (Palmer et al. 2011). New recruits were observed for *Acropora*, *Montipora*, and *Porites* at our monitoring site. However, no recruits were noticed for *Pocillopora*. This will likely lead to a change in the structure of the coral community in our study area, as has been observed in the Florida Keys, where the dominant coral taxon (Acroporidae) was replaced by coral taxa with significantly lower growth rates as a result of WBD (Aronson & Precht 2001).

Sea surface temperature was significantly associated with disease incidence (BD) and disease progression (BD and WBD), such that the highest disease incidence and progression was observed when temperature reached a maximum. Our research results are consistent with many other works, where mass mortalities due to diseases were recorded during high water temperatures (Cerrano et al. 2000, Riegl 2002, Bruno et al. 2007). Similarly, Caribbean yellow band disease prevalence around Puerto Rico was significantly correlated with temperature over the past decade (Harvell et al. 2009). Other studies from the Caribbean (Bruckner & Bruckner 1997b) and the Great Barrier Reef (Raymundo & Harvell 2008) provide further evidence for a correlation between temperature and disease prevalence. In our study, the maximum progression rate for both BD and WBD occurred during the summer season when temperatures were above 31°C. Moreover, progression of the diseases was observed only when the temperature went above 29°C. As supported by previous literature, temperature above this level likely influences the coral diseases by reducing the corals' immune power to fight against the pathogens (Bruno et al. 2007, Harvell et al. 2009). A finding of Raymundo & Harvell (2008) provides further evidence of coral susceptibility during temperature stress.

The link between nutrient enrichment and disease severity has been reported by many authors (Kim & Harvell 2002, Sutherland et al. 2004). In contrast, in our study no significant relationship between nutrient concentration and disease incidence was observed. This agrees with previous studies which found that nutrient enrichment did not affect disease prevalence (Gochfeld et al. 2006). Lower prevalence was also observed in remote areas, where less nutrient pollution was recorded (Bruckner & Bruckner 2006).

In our study, progression of WBD over live coral colonies occurred at a maximum of 1.5 cm mo^{-1} with a mean value of 0.6 ± 0.5 cm mo^{-1}. This rate is lower than that reported from other countries. For example, in Florida, a rapid progression of WBD of 4 cm branch length per day was reported over a live coral colony (Williams & Miller 2005); in the Philippines it was 2 cm d^{-1} (Antonius 1981), and in the Caribbean 5 mm d^{-1} (Gladfelter 1991). Similar to other observed cases of BD, we recorded a maximum progression rate of 3.3 cm mo^{-1} with a mean value of 0.8 ± 1.0 cm mo^{-1}. This progression value is lower than that observed for BBD in a Caribbean reef, where an average progression rate of 3 mm d^{-1} was recorded with a maximum of 1 cm d^{-1} (Antonius 1981, Edmunds 1991,

Carlton & Richardson 1995); in the same region, a high progression rate of 6.2 mm d^{-1} with a maximum of 2 cm d^{-1} has also been reported (Antonius 1973, Kuta & Richardson 1996).

After decades of destruction, corals in the GOM are recovering through successful reproduction and recruitment (Edward et al. 2012). However, activities such as trap fishing in the reef areas, coral algal phase shift (Diraviya Raj & Patterson Edward 2010), and bio-invasion of the exotic seaweed *Kappaphycus alvarezii* (Chandrasekaran et al. 2008) along with natural factors still pose a serious threat to the recovery of corals in the GOM. Since temperatures are expected to rise over the next few decades because of global climate change, it is possible that coral diseases will become more prevalent and lead to coral mortality at many reef sites.

Although the management of coral diseases is extremely difficult, coral disease management strategies have been successfully implemented. For instance, in the Florida Keys, Hudson (2000) treated BBD (70% effective) by removing the pathogen by suction and covering the affected area with modeling clay (lesion occlusion). In Australia, Dalton et al. (2010) found that mechanical removal of the advancing disease margin for *Turbinaria* colonies affected by a tissue loss disease ('white syndrome') was successful at halting the disease in 80% of the colonies. Since the GOM provides a livelihood to thousands of fishermen, the conservation of corals is vital. In order to save the remaining healthy reef, focused research and regular monitoring should be initiated to gain knowledge on the exact causative agents, coral host immunity, species-specific defenses, and the environmental factors that influence coral diseases, so that proper remedial measures and management strategies can be implemented.

Acknowledgements. We thank the Ministry of Environment and Forests, Government of India, for financial support. We also thank the Principle Chief Conservator of Forests and Chief Wildlife Warden, Tamil Nadu Forest Department, and Wildlife Warden, Gulf of Mannar Marine National Park, for research permission and encouragement and the Suganthi Devadason Marine Research Institute for facilities.

LITERATURE CITED

Aeby GS, Williams GJ, Franklin E, Haapkylä J and others (2011) Growth anomalies on the coral genera *Acropora* and *Porites* are strongly associated with host density and human population size across the Indo-Pacific. PLoS ONE 6:e16887

Antonius A (1973) New observations on coral destruction in reefs. Abs Assoc Isl Mar Lab Caribb 10:3

Antonius A (1981) The 'band' diseases in coral reefs. Proc 4th Int Coral Reef Symp, Manila 2:7–14

Antonius A (1985) Coral disease in the Indo-Pacific: first recording. PSZNI: Mar Ecol 6:197–218

Aronson RB, Precht WF (2001) White-band disease and the changing face of Caribbean coral reefs. Hydrobiologia 460:25–38

Borger J, Steiner SC (2005) The spatial and temporal dynamics of coral diseases in Dominica, West Indies. Bull Mar Sci 77:137–138

Bruckner AW (2002) Potential application of the U.S. Endangered Species Act as a conservation strategy. NOAA Tech Memo NMFS-OPR-24. NOAA, Silver Spring, MD

Bruckner A (2005) The global perspective of incidence and prevalence of coral diseases. NOAA, Silver Spring, MD

Bruckner AW, Bruckner RJ (1997a) Outbreak of coral disease in Puerto Rico. Coral Reefs 16:260

Bruckner AW, Bruckner RJ (1997b) The persistence of black-band disease in Jamaica: impact on community structure. Proc 8th Int Coral Reef Symp, Panama City 1: 601–606

Bruckner AW, Bruckner RJ (2006) Consequences of yellow band disease (YBD) on *Montastraea annularis* (species complex) populations on remote reefs off Mona Island, Puerto Rico. Dis Aquat Org 69:67–73

Bruno JF, Selig ER, Casey KS, Page CA and others (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biol 5:e124

Carlton RG, Richardson LL (1995) Oxygen and sulfide dynamics in a horizontally migrating cyanobacterial mat: black band disease of corals. FEMS Microbiol Ecol 18: 155–162

Cerrano C, Bavestrello G, Bianchi CN, Cattaneo VR and others (2000) A catastrophic mass-mortality episode of gorgonians and other organisms in the Ligurian Sea (Northwestern Mediterranean), summer 1999. Ecol Lett 3:284–293

Chandrasekaran S, Nagendran NN, Pandiaraja D, Krishnankutty N, Kamalakannan B (2008) Bioinvasions of *Kappaphycus alvarezii* on corals in the Gulf of Mannar, India. Curr Sci 94:1167–1172

Chen TR, Yu KF, Shi Q, Li S and others (2009) Twenty-five years of change in scleractinian coral communities of Daya Bay (northern South China Sea) and its response to the 2008 AD extreme cold climate event. Chin Sci Bull 54:2107–2117

Dalton SJ, Godwin S, Smith SDA, Pereg L (2010) Australian subtropical white syndrome: a transmissible, temperature-dependent coral disease. Mar Freshw Res 61: 342–350

Davis M, Gladfelter E, Lund H, Anderson M (1986) Geographic range and research plan for monitoring white band disease. Virgin Islands Resource Management Cooperative Biosphere Reserve Research Report 6. USVI National Park, St. Thomas, USVI

Diraviya Raj KD, Patterson Edward JK (2010) Observation on the reproduction of *Acropora* corals along the Gulf of Mannar, southeastern India. Indian J Mar Sci 39(2): 219–226

Edmunds PJ (1991) Extent and effect of black band disease on a Caribbean reef. Coral Reefs 10:161–165

Edward JKP, Mathews G, Raj KD, Thinesh T, Patterson J, Tamelander J, Wilhelmsson D (2012) Coral reefs of Gulf of Mannar, India — signs of resilience. Proc 12th Int Coral Reef Symp, Cairns 1:9–13

English S, Wilkinson C, Baker V (1997) Survey manual for marine resources, 2nd edn. Australian Institute of Marine Science, Townsville

Gardner TA, Cote IM, Gill JA, Grant A, Watkinson AR (2003) Long-term region-wide declines in Caribbean corals. Science 301:958–960

Gladfelter WB (1991) Population structure of *Acropora palmata* on the windward forereef, Buck Island National Monument: seasonal and catastrophic changes 1988-1989. In: Gladfelter EH, Bythell JC, Gladfelter WB (eds) Ecological studies of Buck Island Reef National Monument, St. Croix, US Virgin Islands. A quantitative assessment of selected components of the coral reef ecosystem and establishment of long term monitoring sites. US Department of the Interior, National Park Service, USVI, p 1–21

Gochfeld DJ, Olson JB, Slattery M (2006) Colony versus population variation in susceptibility and resistance to dark spot syndrome in the Caribbean coral *Siderastrea siderea*. Dis Aquat Org 69:53–65

Goreau TJ, Cervino J, Goreau M, Hayes R and others (1998) Rapid spread of diseases in Caribbean coral reefs. Rev Biol Trop 46(Suppl 5):157–171

Grasshoff K, Kremling K, Ehrhardt M (1999) Methods of seawater analysis, 3rd edn. Verlag Chemie, Weinheim

Haapkylä J, Seymour AS, Trebilco J, Smith D (2007) Coral disease prevalence and coral health in the Wakatobi Marine Park, South-East Sulawesi, Indonesia. J Mar Biol Assoc UK 87:403–414

Haapkylä J, Thomas JM, Flavell M, Willis BL (2010) Spatiotemporal patterns of coral disease prevalence on Heron Island, Great Barrier Reef, Australia. Coral Reefs 29:1035–1045

Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, Ostfeld RS, Samuel MD (2002) Climate warming and disease risks for terrestrial and marine biota. Science 296: 2158–2162

Harvell D, Aronson R, Baron N, Connell J and others (2004) The rising tide of ocean diseases: unsolved problems and research priorities. Front Ecol Environ 2:375–382

Harvell D, Altizer S, Cattadori IM, Harrington L, Weil E (2009) Climate change and wildlife diseases: When does the host matter the most? Ecology 90:912–920

Hudson H (2000) First aid for massive corals infected with black band disease, *Phormidium corallyticum*: an underwater aspirator and post-treatment sealant to curtail reinfection. In: Hallock P, French L (eds) Diving for science in the 21st century: Proc 20th Annu Sci Diving Symp. American Academy of Underwater Sciences, St Pete Beach, FL, p 10–11

Hughes TP, Connell JH (1999) Multiple stressors on coral reefs: a long-term perspective. Limnol Oceanogr 44: 932–940

Hughes TP, Baird AH, Bellwood DR, Card M and others (2003) Climate change, human impacts and the resilience of coral reefs. Science 301:929–933

Jackson JB, Kirby MX, Berger WH, Bjorndal KA and others (2001) Historical over-fishing and the recent collapse of coastal ecosystems. Science 293:629–637

Kim K, Harvell CD (2002). Aspergillosis of sea fan corals: disease dynamics in the Florida Keys. In: Porter JW, Porter KG (eds) The Everglades, Florida Bay, and Coral Reefs of the Florida Keys: an ecosystem sourcebook. CRC Press, Boca Raton, FL, p 813–824

Kuta KG, Richardson LL (1996) Abundance and distribution of black band disease on coral reefs in the northern Florida Keys. Coral Reefs 15:219–223

Kuta KG, Richardson LL (1997) Black band disease and the fate of diseased coral colonies in the Florida Keys. Proc 8th Int Coral Reef Symp, Panama City 1:575–578

Palmer CV, McGinty ES, Cummings DJ, Smith SM, Bartels E, Mydlarz LD (2011) Patterns of coral ecological immunology: variation in the responses of Caribbean corals to elevated temperature and a pathogen elicitor. J Exp Biol 214:4240–4249

Pandolfi JM, Bradbury RH, Sala E, Hughes TP and others (2003) Global trajectories of the long-term decline of coral reef ecosystems. Science 301:955–958

Patterson Edward JK, Mathews G, Patterson J, Ramkumar R, Wilhelmsson D, Tamelander J, Linden O (2008) Status of coral reefs of the Gulf of Mannar, southeastern India. In: Obura DO, Tamelander J, Linden O (eds) Ten years after bleaching — facing the consequences of climate change in the Indian Ocean. CORDIO Status Report 2008. Coastal Oceans Research and Development in the Indian Ocean/Sida-SAREC, Mombasa, p 45–55

Pillai CSG (1971) Composition of the coral fauna of the southeastern coast of India. In: Stoddart DR, Young CM (eds) Regional variation in Indian Ocean coral reefs. Symp Zool Soc Lond 28:301–325

Pillai CSG (1996) Coral reefs of India: their conservation and management. In: Menon NG, Pillai CSG (eds) Marine biodiversity: conservation and management. Central Marine Fisheries Research Institute, Cochin, p 16–31

Plucer-Rosario G (1987) The effect of substratum on the growth of *Terpios*, an encrusting sponge which kills corals. Coral Reefs 5:197–200

Porter J, Dustan P, Jaap W, Patterson K and others (2001) Patterns of spread of coral disease in the Florida Keys. Hydrobiologia 460:1–24

Raymundo L, Harvell CD (2008) The objectives and scope of this manual. In: Raymundo LJ, Couch CS, Harvell CD (eds) Coral disease handbook. Currie Communications, Melbourne, p 7–16

Raymundo LJH, Harvell CD, Reynolds T (2003) *Porites* ulcerative white spot disease: description, prevalence and host range of a new coral disease affecting Indo-Pacific reefs. Dis Aquat Org 56:95–104

Raymundo LJ, Rosell KB, Reboton CT, Kaczmarsky L (2005) Coral diseases on Philippine reefs: genus *Porites* is a dominant host. Dis Aquat Org 64:181–191

Raymundo L, Work T, Bruckner A, Willis B (2008) A decision tree for describing coral lesions in the field. In: Raymundo LJ, Couch CS, Harvell CD (eds) Coral disease handbook. Currie Communications, Melbourne, p 17–32

Riegl B (2002) Effects of the 1996 and 1998 sea surface temperature anomalies on corals, fish and coral diseases in the Arabian Gulf (Dubai, UAE). Mar Biol 140:29–40

Santavy DL, Peters EC (1997) Microbial pests: coral disease in the Western Atlantic. Proc 8th Int Coral Reef Symp, Panama City 1:607–612

Santavy DL, Mueller E, Peters EC, MacLaughlin L, Porter JW, Patterson KL, Campbell J (2001) Quantitative assessment of coral diseases in the Florida Keys: strategy and methodology. Hydrobiologia 460:39–52

Shepard C, Wells S (1988) Coral reefs of the world, Vol 2: Indian Ocean, Red Sea and Gulf. IUCN and UNEP, Cambridge

Sutherland KP, Porter JW, Torres C (2004) Disease and

immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar Ecol Prog Ser 266:273–302

Szmant AM (2002) Nutrient enrichment on coral reefs: Is it a major cause of coral reef decline? Estuaries 25:743–766

Thinesh T, Mathews G, Patterson Edward JK (2008) Occurrence of cyanobacterial infection (black band disease) in Mandapam group of islands (Gulf of Mannar) southeast Tamil Nadu. Seaweed Res Util 30(Spec Issue):67–75

Thinesh T, Mathews G, Patterson Edward JK (2009) Coral disease prevalence in Mandapam group of islands, Gulf of Mannar, Southeastern India. Indian J Mar Sci 38: 444–450

Vanderzant C, Splittstoesser DF (1992) Compendium of methods for the microbiological examination of foods. American Public Health Association, Washington, DC

Venkataramanujam R, Santhanam R, Sukumaran N (1981) Coral resources of Tuticorin (S. India) and methods of their conservation. Proc 4th Int Coral Reef Symp, Manila 1:259–262

Wilkinson CR (2000) Status of coral reefs of the world. Australian Institute of Marine Science, Townsville

Wilkinson C (2002) Status of coral reefs of the world: Global Coral Reef Monitoring Network and Australian Institute of Marine Science, Townsville

Williams DE, Miller MW (2005) Coral disease outbreak: pattern, prevalence and transmission in *Acropora cervicornis*. Mar Ecol Prog Ser 301:119–128

Willis BL, Page CA, Dinsdale EA (2004) Coral disease on the Great Barrier Reef. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin, p 69–104

Work TM, Aeby GS (2006) Systematically describing gross lesions in corals. Dis Aquat Org 70:155–160

Editorial responsibility: Bernd Sures, Essen, Germany

Submitted: February 4, 2013; Accepted: June 13, 2013
Proofs received from author(s): September 12, 2013