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

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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, R² = 0.75, p < 0.001) and WBD (R² = 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 (Gardner et al. 2003). Coral diseases have been increasing...
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 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 km², 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 Poovarasnanpatti) 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 km² of coral reef have already been degraded around the 21 islands of the GOM (Venkataramanjam 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>
<thead>
<tr>
<th>Observed lesion</th>
<th>Fig.</th>
<th>Description/morphologic diagnosis</th>
</tr>
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<tbody>
<tr>
<td>Pink spot disease</td>
<td>2a</td>
<td>Moderate (Ex); colony-wide (Lo); multifocal (Di); tissue loss (Ti); discoloration (pink color) (Le); polyp, coenosarc (St)</td>
</tr>
<tr>
<td>White plaque disease</td>
<td>2b</td>
<td>Severe (Ex); basal to peripheral (Lo); diffuse (Di); acute (Ti); tissue loss (Le); polyp, coenosarc (St)</td>
</tr>
<tr>
<td>Black disease</td>
<td>2c</td>
<td>Severe (Ex); basal (Lo); diffuse (Di); acute (Ti); tissue loss, black colored smothering mat with annular margin (Le); polyp, coenosarc, skeleton (St)</td>
</tr>
<tr>
<td>White band disease</td>
<td>2d</td>
<td>Severe (Ex); basal (Lo); diffuse (Di); acute to subacute (Ti); tissue loss (Le); polyp, coenosarc (St)</td>
</tr>
</tbody>
</table>
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 $0.8 \pm 1.0$ 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 $0.6 \pm 0.5$ 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 $29.4 \pm$
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 $1.3 \pm 0.9 \mu g \ l^{-1}$, $1.62 \pm 0.9 \mu g \ l^{-1}$, and $37 \pm 5.6 \times 10^4$ (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).
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, Haapkyllä 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.

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