Vol. 126: 63–70, 2017
https://doi.org/10.3354/dao03155

DISEASES OF AQUATIC ORGANISMS
Dis Aquat Org

Published September 20

Outbreaks of *Acropora* white syndrome and *Terpios* sponge overgrowth combined with coral mortality in Palk Bay, southeast coast of India

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ABSTRACT: *Acropora* white syndrome (AWS) and *Terpios* sponge overgrowth (TSO) are serious threats to coral communities in various regions; however, information on these 2 lesions in the Indian Ocean is much more limited than in the Indo-Pacific. The present study revealed the impact of these lesions on the Palk Bay reef, India, and covered an area of 7 km^2. In total, 1930 colonies were permanently monitored to assess incidences of AWS and TSO and consequent mortality for a period of 1 yr. TSO affected 5 coral genera and caused 20.7 % mortality; overall prevalence increased from 1.3 % (n = 25) to 25.5 % (n = 492). In contrast, AWS only affected *Acropora* colonies and caused a mortality of 8 %; overall prevalence increased from 0.9 % (n = 17) to 12.9 % (n = 249). Year-round monitoring revealed an increasing trend of both AWS and TSO, followed by temperature rise. These results add to the known geographic distribution of these coral diseases and reveal the impacts of AWS and TSO on coral reefs in the Indian Ocean.

KEY WORDS: Shallow reef · Coral disease · Temperature · Mortality · Incidence

INTRODUCTION

After recognition of the first signs of disease, the numbers of coral diseases and syndromes and their geographical distributions have been found to increase (Green & Bruckner 2000, Raymundo et al. 2005). At least 102 species of corals have been affected by diseases across 54 countries, with the number of affected species increasing constantly (Green & Bruckner 2000, Thinesh et al. 2013). Outbreaks of coral diseases can cause mass mortality, thus reducing the population and resulting in the loss of community structure and significant marine biodiversity (Graham et al. 2006). For instance, in the 1980s, white band disease destroyed acroporid corals throughout the Caribbean (Gladfelter 1982, Aronson & Precht 2006). Similarly, an outbreak of white pox disease in the Florida Keys, USA, reduced *Acropora palmata* by 70 % in 1998 (Patterson et al. 2002), and white syndrome caused 36 % coral mortality around Christmas Island during 2008 (Hobbs et al. 2015). Preliminary surveys in Australia (Willis et al. 2004), the Philippines (Raymundo et al. 2005), the northwestern Hawaiian Islands, USA, (Aeby 2006), the Central Pacific (Work et al. 2008), Caribbean (Weil 2005), and India (Thinesh et al. 2013) have documented these new diseases and the amounts of damage caused. Such studies are providing important insights into diseases and their vital role in the ecology of Indo-Pacific coral assemblages.

Recently, the impact of the invasive coral-killing sponge *Terpios hoshinota* (thin encrusting sponge) on coral reefs has been increasing, and the geographical range of this species has expanded since the first reports on the reef of Guam (Bryan 1973, Plucer-Rosario 1987, Rützler & Muzik 1993). The geographical locations of such invasions include American Samoa and the Philippines (Plucer-Rosario 1987), Japan (Reimer et al. 2011a), Taiwan (Chen et al. 2009), the Great Barrier Reef in Australia (Fujii et al. 2011), Indonesia

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(De Voogd et al. 2013), and recently, India (Thinesh et al. 2015) and Mauritius (Elliott et al. 2016b). *Terpios* can cause extensive mortality to coral reefs because of its fast, overgrowing nature (linear growth rate of 11.5 to 23 mm mo^{-1}) on live corals, its larvae-producing capacity (easily spreading to nearby locations), and its long-lasting nature in the reef environment (preventing coral larval settlement) (Lin & Soong 2009, Reimer et al. 2011a). For instance, in some locations of the Western Pacific, the invasion caused mortality rates ranging from 30 to 80% (Plucer-Rosario 1987, Rützler & Muzik 1993, Liao et al. 2007, Reimer et al. 2011a, Shi et al. 2012). Moreover, this species seems to be progressively extending its global range, migrating westwards through the Indian Ocean (Elliott et al. 2016a). As a result, *T. hoshinota* is now well recognized as a potential threat to the survival of corals, and more precise information about its geographic distribution is required.

Numerous studies have reported a relationship between temperature and disease prevalence, incidence, mortality, and progression over live corals (Gil Agudelo & Garzón-Ferreira 2001, Alker et al. 2001, Kuta & Richardson 2002, Patterson et al. 2002). For example, in Florida, with the highest black band disease prevalence, a progression rate of 3 mm d^{-1} was recorded during the summer, although the species disappeared during the winter without recovery (Feingold 1988). Likewise, white plague and white pox diseases cause 20 to 30% and 50 to 80% mortality, respectively, with the highest prevalence during the summer season (Dustan 1977, Patterson et al. 2002). In contrast, dark spot disease did not cause significant mortality to the affected reef in the Bahamas and, importantly, small infections could recover (Cervino et al. 2001). These studies indicate that the disease types, the species affected, the disease mortality, and the factors influencing these diseases appear to vary with different geographical locations. Hence, it is necessary to understand the impacts of these diseases at the local scale in order to establish effective mitigation strategies.

In India, the coral reefs in Palk Bay are currently not in good condition (Asir Ramesh 1996) because of anthropogenic disturbances (trap fishing, seaweed collection, household wastage dumping), climatic effects, geographic position (directly exposed to sunlight during low tide for an extended period), and sporadic diseases (Sridhar et al. 2008, Sukumaran et al. 2011). However, to date, the impacts of coral diseases on the Palk Bay reef have not been assessed. In this study, we documented the temporal incidence, prevalence, and induced mortality of coral genera caused by *Acropora* white syndrome (AWS) and *Terpios* sponge overgrowth (TSO) in Palk Bay reef.

MATERIALS AND METHODS

Study site

Surveys were conducted in an east–west direction in one part of the Palk Bay fringing reef, situated between the longitudes 79° 17′ 40″ and 79° 8′ E and at a latitude of 9° 17′ N (Fig. 1). The sampling area comprised a narrow reef running parallel to the land, with a width between 200 and 600 m, a depth between 1 and 5 m, and an approximate distance from shore of 1 to 4 km. The reef is not a marine protected area. The region has been impacted by human factors, including oil and waste discharge, high fishing pressure (largely in the form of trap fishing) as well as land-based disturbances (Asir Ramesh 1996, Karuppanapandian et al. 2007). In 2008, estimated live coral cover was 37.8%; the remaining parts were covered by seagrass and sand (Sukumaran et al. 2008).

Field data collection

A preliminary assessment was carried out during December 2009. Five permanent monitoring sites with similar coral communities were randomly se-

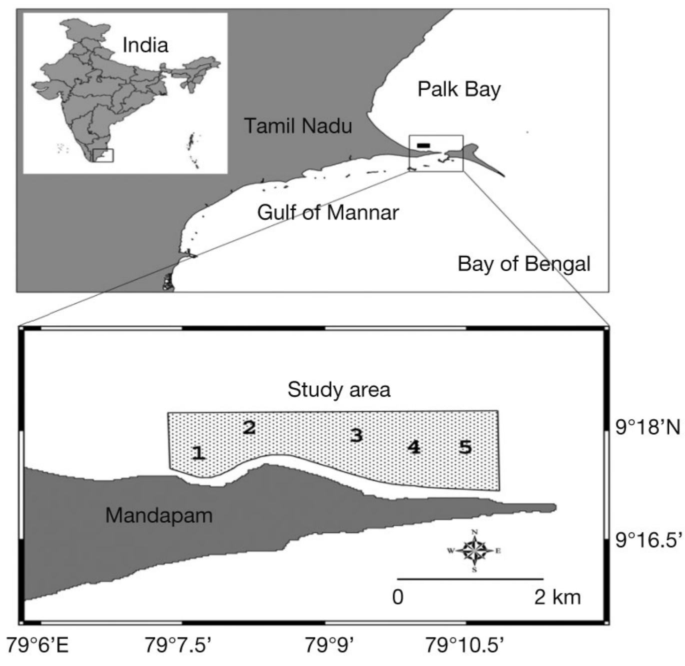

Fig. 1. Study area: Palk Bay, Indian Ocean. 1–5: permanent monitoring sites

lected (Fig. 1). A total of 40 transects (3200 m^2), with eight 20 × 4 m transects per site were established end to end parallel to the reef crest apparently along the same depth, with a gap of at least 20 m between transects. Benthic cover was determined along a transect line using the line intercept method (English et al. 1997). Permanent buoys with markers were placed at both ends of each transect to replicate the same transects each time for monthly monitoring between January 2010 and December 2010. Coral colonies within the transects were counted each month. We considered only an autonomous mass of skeleton with a live tissue size >10 cm in diameter as a colony because of the difficulties in following smaller colonies during subsequent visits. Colonies along each transect were individually marked to ensure accurate identification during subsequent surveys.

Two types of lesion morphologies, consistent with previously described AWS lesions (white discrete band separating healthy tissue from exposed skeleton) and TSO (black smothering mat with annular margin) were identified by following the coral disease handbook of Raymundo & Harvell (2008) and research articles (Rützler & Muzik 1993, Shi et al. 2012).

Total healthy (live colony without any lesions), affected (colonies with above-mentioned lesions), and dead (without any live tissue) colonies were counted within each transect once a month to calculate each lesion's prevalence (percent of corals with lesions), incidence (newly infected colonies per month), and coral mortality. The prevalence of each lesion was calculated as follows: prevalence = (number of lesion-affected colonies/total number of colonies) × 100. Mortality was calculated by dividing the number of dead colonies by the total number of coral colonies, and incidence was calculated as: incidence = (number of new infections/total number of colonies) × 100 (Raymundo & Harvell 2008). *In situ* identification of *Terpios hoshinota* was performed based on morphological characteristics by following Rützler & Muzik (1993). For *ex situ* confirmation of identification, a small portion of the mat was peeled off the active portion and placed in a sterile 100 ml centrifuge tube at 4°C, transported to the laboratory, and preserved in 70% ethanol at −20°C until further analysis. The mat was washed twice with distilled water to remove ethanol, and subsequently 1.5 ml of 30% sodium hypochlorite solution was added to digest the sample. After 1 h, the sample was centrifuged at 3000 rpm (1077 × *g*) for 5 min (Eppendorf). The suspension was microscopically examined to identify the species-specific, distinctive spicules and cyanobacteria (Shi et al. 2012).

Monitoring of environmental parameters and statistical analysis

Temperature data from January 2010 to December 2010 was obtained from the National Oceanic and Atmospheric Administration (NOAA). The comprehensive large array data stewardship system (https://www.class.ncdc.noaa.gov/saa/products/search?datatype_family=SST100) was used to calculate mean monthly sea surface temperatures. Regression analysis was performed for total number of each observed coral genera against total number of dead colonies in each genera as a result of *Terpios* to test if there was any positive relationship between colony abundance and mortality irrespective of genera. Repeated measures ANOVA was used to check whether the numbers of corals affected by each lesion differed over the months. Kolmogorov-Smirnov test (K-S) for normality was performed for incidence. The data were converted into proportions by dividing the number of newly infected coral colonies by the total number of colonies. The results from the K-S tests showed that the calculated proportion data for the 12 mo was normally distributed ($p > 0.05$). All statistical analyses were carried out using Excel and SPSS.

RESULTS

The dominant benthic substrate of the Palk Bay is hard substrate with a mean percentage cover of 24.9 ± 7.13% (SD), followed by 23.7 ± 4.9% live coral cover, 20.5 ± 5.41% sand, 17.1 ± 0.31% other macro and turf algae, 9.3 ± 3.21% *Halimeda*, 3.5 ± 1.12% sea grass, and 1.0 ± 0.59% sponges. Among the live corals, *Acropora* had the highest density at 11%, followed by *Porites* (7.1%), *Favia* (2.8%), *Goniastrea* (2.19%), and *Platygyra* (0.5%). The proportion of the coral community based on colony count was as follows: 46.6% *Acropora*, 30.1% *Porites*, 11.9 to 9.3% *Goniastrea*/*Favia*, and 2.1% *Platygyra*.

A gradual increase in *Terpios* prevalence was noticed at the study site over the 1 yr period. Out of 1930 colonies examined, 25.4% (n = 492) were affected by *Terpios* sponges during the study period. This percentage had increased from the initial amounts of 1.3% (n = 25). Incidence values for TSO were also measured during the monitoring period. Maximum incidence of 3.1 ± 0.56% (SD) was observed in June followed by the highest temperature, and the lowest incidence of 1.3 ± 0.56% was observed in January with an average monthly incidence of 2.2 ± 0.16% (SE) (Fig. 2). The *Terpios* sponge affected all ob-

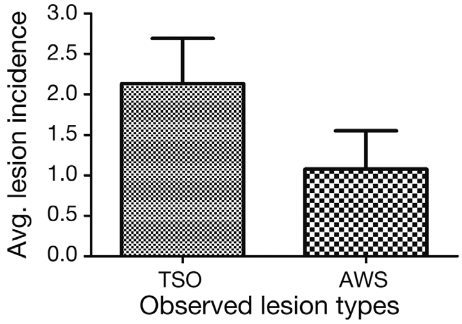

Fig. 2. Average (±SE) monthly incidence of *Acropora* white syndrome (AWS) and *Terpios* sponge overgrowth (TSO) from surveys conducted between Jan 2010 to Dec 2010

served coral genera. Results of the repeated measures 1-way ANOVA showed that there was a difference in incidence over the months (F = 111.864, df = 11, n = 492, p < 0.001; Table 1). However, there was no significant variation in TSO incidence among genera (F = 2.658, df = 4, n = 492, p = 0.063).

Similarly, a gradual increase in AWS was also noticed at the study site over the 1 yr period. Of 1930 colonies examined, 12.9% (n = 249) were affected by AWS during the study period. This percentage had increased from the initial amounts of 0.9% (n = 17). The incidence values for AWS were also measured during monitoring period. Maximum incidence was observed for both lesions during the summer months, followed by the highest temperature. Maximum disease incidence was 2 ± 0.46% (SD) during May, followed by 1.5 ± 0.46% during June; the lowest incidence was observed in December at 4 ± 0.46%, with a monthly average mean incidence of 1.07 ± 0.13% (SE) (Fig. 2). Fig. 3 shows underwater photographs of observed lesions, and Fig. 4 shows AWS and TSO prevalence trends during the monthly monitoring period. Fig. 5 shows the details of each monthly incidence pattern during the study period, along with temperature patterns. Variation in AWS incidence was observed over the months (F = 41.712, df = 11, n = 249, p = 0.003; Table 2).

Coral mortality of 74.7% (n = 554) was observed for total affected colonies. The highest mortality (53.9%) of total affected colonies was caused by TSO, followed by 20.7% for AWS. The intensity of coral mortality caused by AWS and TSO varied significantly among coral genera. Of total affected colonies, the highest mortality was observed for the genus *Acropora* (35.8%; n = 323), caused by AWS (23.3%) and TSO (12.5%), followed by *Porites* (25.3%), *Favia* (22.1%), *Platygyra* (20%), and *Goniastrea* (13.8%), caused by TSO (Fig. 6). Regression analysis shows that there is a positive relationship between coral colony abundance and colony number, which indicates that there are no generic differences in susceptibility to TSO (R^2 = 0.772; Fig. 7).

Table 1. Repeated measures 1-way ANOVA result of *Terpios* sponge overgrowth (TSO) incidence and the variation in the TSO incidence between genera. F = mean square conditions/ mean square error

Source	Tests of between-subject effects				
	Type III SS	df	MS	F	p
Incidence	0.118	11	0.118	111.864	0.000
Genera	0.011	4	0.003	2.658	0.063
Error	0.021	20	0.001		

DISCUSSION

Based on our results, prevalence of AWS and TSO in the coral reefs of Palk Bay has increased, inducing coral mortality. The AWS lesion type in our study site is similar to the field signs reported in *Acropora* colonies from different locations, including the Indo-Pacific region (Bruno et al. 2007, Haapkylä et al. 2009, Aeby et al. 2011, Roff et al. 2011). Studies from different geographical locations have shown the susceptibility of *Acropora* colonies to white syndrome; the areas where such susceptibility has been reported include the northwestern Hawaiian Islands (Green & Bruckner 2000, Aeby 2005, Aeby et al. 2011), Marshall Island (Jacobson 2006), and Wakatobi Marine National Park, Indonesia (Haapkylä et al. 2007). Similarly, in our study site, *Acropora* was the only coral genus affected by white syndrome, although other coral genera were found near the affected colonies. The difference in susceptibility between different species is probably due to the pathogen specificity and the difference in immunological responses (Mullen et al. 2004, Fuess et al. 2017).

In this study, AWS increased from 1.3 to 12.9% during the study period, causing mortality in over 20% of *Acropora* coral colonies. Similarly, the mortality caused by white syndrome in *Acropora* colonies was 4.9% in the southern Great Barrier Reef (Roff et al. 2011) and 36% around the Christmas Island (Hobbs et al. 2015). In the Indo-Pacific, AWS prevalence increased by 150 times within a period of 5 yr (Willis et al. 2004). Hence, the increasing trend and mortality rate as a result of white syndrome within a short period of time in our study site will likely reduce *Acropora* coral cover in Palk Bay, similar to what has been reported from the Caribbean (Aronson & Precht 2001).

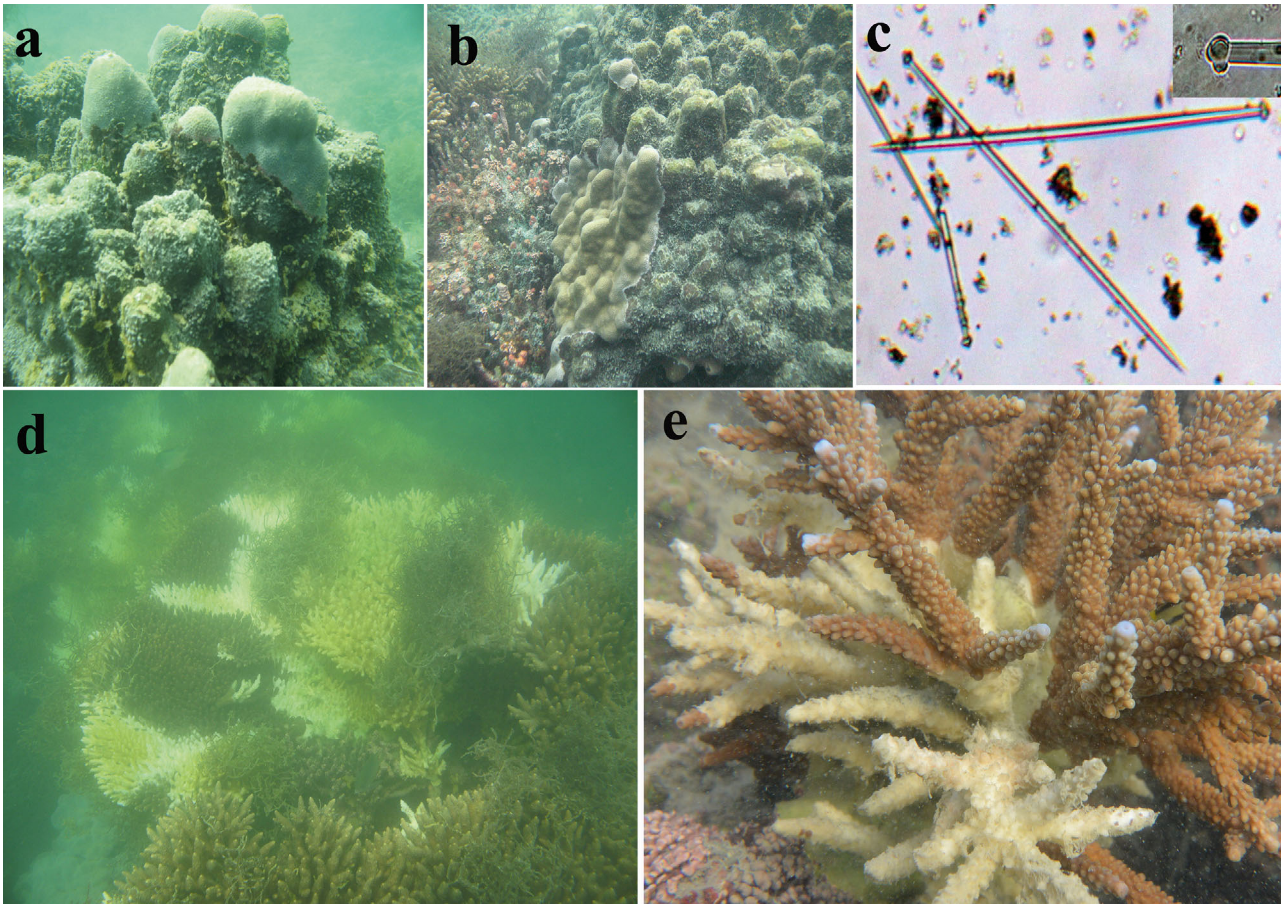

Fig. 3. Photographs of observed lesions: (a) *Terpios* sponge overgrowth (TSO) in *Porites*, (b) partially dead *Porites* colony due to TSO, (c) spicules of *T. hoshinota* showing lobed head of tylostyle spicules; (d,e) *Acropora* white syndrome in *Acropora*

Several studies have reported an influence of temperature on various coral diseases (Feingold 1988, Kuta & Richardson 2002, Weil 2004). For instance, in the northeastern Caribbean, outbreaks of white plague, yellow band, and white patch disease were recorded during summer (Miller et al. 2006). A white plague disease outbreak was recorded immediately following summer throughout the Caribbean (Cróquer & Weil 2009, Rogers et al. 2009). Recently, Lewis et al. (2017) reported a black band disease outbreak in *Dendrogyra*, immediately following warmer months in the Florida Keys. Likewise, we found an increasing trend of AWS prevalence followed by rising temperatures. Our results and the findings of the above-mentioned studies suggest that the prevalence and impacts of coral diseases could increase with future climate change.

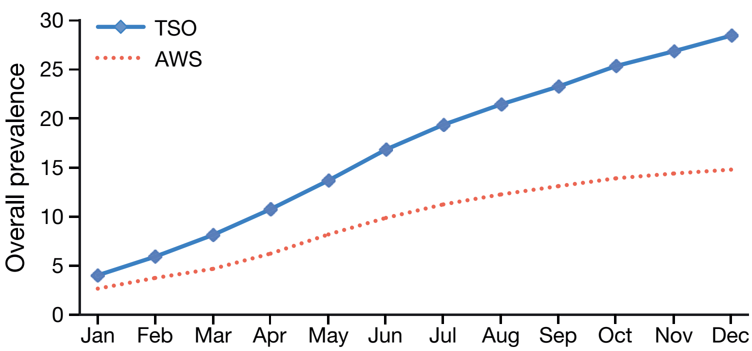

Fig. 4. Overall prevalence (% of corals affected) of each lesion type (AWS: *Acropora* white syndrome; TSO: *Terpios* sponge overgrowth) during the monthly monitoring period from Jan 2010 to Dec 2010

The overgrowth of live corals by *T. hoshinota* increased from an overall prevalence of 2.6 to 28.2% within a 1 yr period. Sponge overgrowth caused significant mortality of over 50% of live corals within our transects. Similarly, Shi et al. (2012) found increased overgrowth by *T. hoshinota* from 1.9 to 25.8%, with mortality over 75% in the South China Sea reef. Liao et al. (2007)

observed a 12% mortality rate on Green Island reef, Australia, within 1 yr. Outbreaks of *T. hoshinota* induce mortality and rapid degradation, threatening the persistence of coral communities in many geographic locations because of its tendency to quickly overgrow live corals, its high larvae producing capacity, and its long-lasting nature in the reef environment (preventing coral larval settlement) (Bryan 1973, Lin & Soong 2009, Reimer et al. 2011a,b). *Terpios* abundance is hypothesized to be higher with increased nutrient amounts. Thus, an outbreak of *Terpios* is most often associated with near-shore pollution in shallow reef areas, where nutrient levels are high due to human activities (Plucer-Rosario 1987, Rützler & Muzik 1993, Chen et al. 2009). However, factors responsible for such outbreaks are not well understood. Although we did not test the factors responsible for *Terpios* outbreaks, the high *Terpios* prevalence associated with high coral mortality in our study site may be related to anthropogenic disturbance, overfishing, and pollution (Asir Ramesh 1996, Karuppanapandian et al. 2007).

We found no generic difference in susceptibility to *Terpios*, which has also been reported from Japan and Taiwan (Reimer et al. 2011b, Chen et al. 2009, Shi et al. 2012). The multi-host nature of this sponge is mainly due to its progressive overgrowth over live coral polyps with the help of tendrils in the active front (Elliott et al. 2016a). The link between temperature and *Terpios* outbreaks remains unclear, since most of the above-mentioned studies were only performed at the documentation level. Hirose & Murakami (2011) suggested that reproduction in *Terpios* in subtropical regions is seasonal. This would be in agreement with our study, as we found the highest TSO incidence during the summer months and the lowest during winter. Similarly, Elliott et al. (2016b) found highest sponge recruitment rates during summer. A better understanding of the factors influencing *Terpios* outbreaks, gained through *in situ* experimental studies, will no doubt be helpful in the development of effective management strategies to protect reefs from this coral-killing sponge.

Overall, based on the findings of this study, we suggest that a combination of

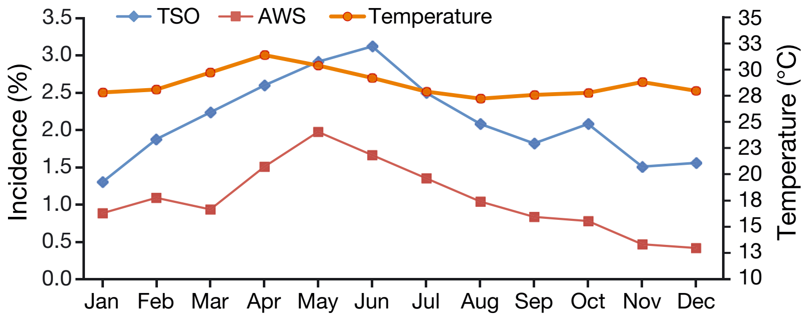

Fig. 5. Relationship between sea temperatures and changes in incidence through time for *Acropora* white syndrome (AWS) and *Terpios* sponge overgrowth (TSO)

Table 2. Repeated measures 1-way ANOVA of *Acropora* white syndrome (AWS) incidence through time

Source	Tests of between-subject effects				
	Type III SS	df	MS	*F*	p
Incidence	0.036	11	0.036	41.712	0.003
Error	0.003	4	0.001		

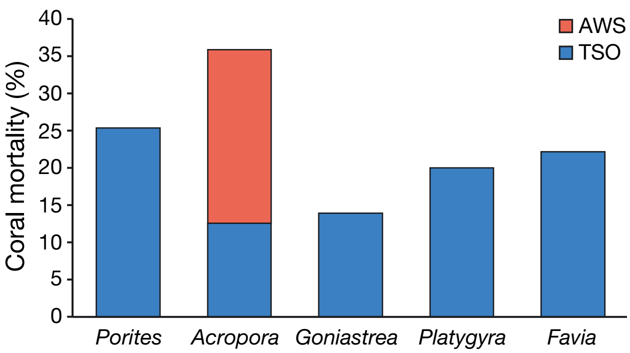

Fig. 6. Differences in overall mortality from *Acropora* white syndrome (AWS) and *Terpios* sponge overgrowth (TSO) among coral genera after 1 yr

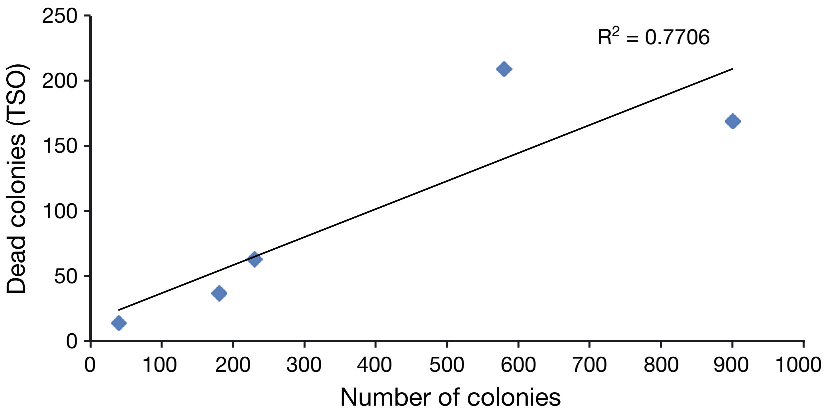

Fig. 7. Relationship between colony abundance and the *Terpios*-caused mortality (*Terpios* sponge overgrowth, TSO) irrespective of coral genera (each dot represents each genera)

TSO and AWS caused the highest mortality within the relatively short period of this study. Numerous studies have confirmed the link between anthropogenic disturbance and disease mortality (Willis et al. 2004). Hence, these diseases can potentially cause significant mortality in the Palk Bay reef, which faces various anthropogenic disturbances because of its proximity to the shore and its unprotected status. We therefore stress the importance of taking immediate steps to understand the factors influencing individual diseases as well as their ecological dynamics at a local scale.

Acknowledgements. We thank the Ministry of Environment and Forests, Government of India, and Suganthi Devadason Marine Research Institute for financial support and facilities to carry out the work. We also thank the University Grant Commission for current fellowship for T.T. through a D. S. Kothari Post-Doctoral fellowship.

LITERATURE CITED

Aeby GS (2005) Outbreak of coral disease in the Northwestern Hawaiian Islands. Coral Reefs 24:481

Aeby GS (2006) Baseline levels of coral disease in the Northwestern Hawaiian Islands. Atoll Res Bull 543:471–488

Aeby GS, Williams GJ, Franklin EC, Kenyon J, Cox EF, Coles S, Work TM (2011) Patterns of coral disease across the Hawaiian archipelago: relating disease to environment. PLOS ONE 6:e20370

Alker AP, Smith GW, Kim K (2001) Characterization of *Aspergillus sydowii* (Thomet Church), a fungal pathogen of Caribbean sea fan corals. Hydrobiologia 460:105–111

Aronson RB, Precht WF (2001) White-band disease and the changing face of Caribbean coral reefs. In: Porter JW (ed) The ecology and etiology of newly emerging marine diseases. Springer, Dordrecht, p 25–38

Aronson RB, Precht WF (2006) Conservation, precaution, and Caribbean reefs. Coral Reefs 25:441–450

Asir Ramesh D (1996) Studies on the ecology of coral reefs in Gulf of Mannar and Palk Bay, southeastern coast of India. PhD thesis, Annamalai University

Bruno JF, Selig ER, Casey KS, Page CA and others (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. PLOS Biol 5:6–124

Bryan PG (1973) Growth rate, toxicity, and distribution of the encrusting sponge *Terpios* sp. (Hadromerida: Suberitidae) in Guam, Mariana Islands. Micronesica 9:237–242

Cervino J, Goreau TJ, Nagelkerken I, Smith GW, Hayes R (2001) Yellow band and dark spot syndromes in Caribbean corals: distribution, rate of spread, cytology, and effects on abundance and division rate of zooxanthellae. Hydrobiologia 460:53–63

Chen SL, Kuo CY, Chen CA (2009) Spatial distribution and temporal variation of an outbreak of the cyanobacteriosponge, *Terpios hoshinota*, and benthos composition on the coral reefs of Green Island. Natl Park Bull, Taiwan 19:32–45

Cróquer A, Weil E (2009) Changes in Caribbean coral disease prevalence after the 2005 bleaching event. Dis Aquat Org 87:33–43

De Voogd NJ, Cleary R, Dekker F (2013) The coral-killing sponge *Terpios hoshinota* invades Indonesia. Coral Reefs 32:755

Dustan P (1977) Vitality of reef coral populations off Key Largo, Florida: recruitment and mortality. Environ Geol 2:51–58

Elliott J, Patterson M, Vitry E, Summers N, Miternique C (2016a) Morphological plasticity allows coral to actively overgrow the aggressive sponge *Terpios hoshinota* (Mauritius, southwestern Indian Ocean). Mar Biodivers 46:489–493

Elliott J, Patterson M, Summers N, Miternique C, Montocchio E, Vitry E (2016b) How does the proliferation of the coral-killing sponge *Terpios hoshinota* affect benthic community structure on coral reefs? Coral Reefs 35: 1083–1095

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

Feingold JS (1988) Ecological studies of a cyanobacterial infection on the Caribbean sea plume *Psudopterogorgia acerosa* (Coelenterata: Octocorallia). Proc 6th Int Coral Reef Symp, Townsville 3:157–162

Fuess LE, Pinzon CJH, Weil E, Grinshpon RD, Mydlarz LD (2017) Life or death: disease-tolerant coral species activate autophagy following immune challenge. Proc Biol Sci 284:20170771

Fujii T, Keshavmurthy S, Zhou W, Hirose E, Chen CA, Reimer JD (2011) Coral-killing cyanobacteriosponge (*Terpios hoshinota*) on the Great Barrier Reef. Coral Reefs 30:483

Gil-Agudelo DL, Garzón-Ferreira J (2001) Spatial and seasonal variation of dark spots disease in coral communities of the Santa Marta area (Colombian Caribbean). Bull Mar Sci 69:619–629

Gladfelter WB (1982) White-band disease in *Acropora palmata*: implications for structure and growth of shallow reefs. Bull Mar Sci 32:639–643

Graham NAJ, Wilson S, Jennings S, Polunin N, Bijoux J, Robinson J (2006) Dynamic fragility of oceanic coral reef ecosystems. Proc Natl Acad Sci USA 103:8425–8429

Green E, Bruckner AW (2000) The significance of coral disease epizootiology for coral reef conservation. Biol Conserv 96:347–361

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, Unsworth RK, Seymour AS, Melbourne-Thomas J and others (2009) Spatio-temporal coral disease dynamics in the Wakatobi Marine National Park, south-east Sulawesi, Indonesia. Dis Aquat Org 87:105–115

Hirose E, Murakami A (2011) Microscopic anatomy and pigment characterization of coral-encrusting black sponge with cyanobacterial symbiont, *Terpios hoshinota.* Zool Sci 28:199–205

Hobbs JPA, Frisch AJ, Newman SJ, Wakefield CB (2015) Selective impact of disease on coral communities: outbreak of white syndrome causes significant total mortality of *Acropora* plate corals. PLOS ONE 10:e0132528

Karuppanapandian T, Karuppudurai A, Kumaraguru K (2007) A preliminary study on the environmental condition of the coral reef habitat. Int J Environ Sci Technol 4: 371–378

Kuta K, Richardson LL (2002) Ecological aspects of black band disease of corals: relationships between disease incidence and environmental factors. Coral Reefs 21: 393–398

Lewis CL, Neely KL, Richardson LL, Mauricio RL (2017) Temporal dynamics of black band disease affecting pillar coral (*Dendrogyra cylindrus*) following two consecutive hyperthermal events on the Florida Reef Tract. Coral Reefs 36:427–431

Liao MH, Tang SL, Hsu CM, Wen KC and others (2007) The 'black disease' of reef-building corals at Green Island, Taiwan: outbreak of a cyanobacteriosponge *Terpios hoshinota* (Suberitidae: Hadromerida). Zool Stud 46:520

Lin WJ, Soong KY (2009) Growth and prevention experiments of *Terpios hoshinota* surrounding Green Island. J Natl Park 19:46–57

Miller J, Waara R, Muller E, Rogers C (2006) Coral bleaching and disease combine to cause extensive mortality on reefs of the US Virgin Islands. Coral Reefs 25:418

Mullen KM, Peters EC, Harvell CD (2004) Coral resistance to disease. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin, p 377–399

Patterson KL, Porter JW, Ritchie KB, Polson SW and others (2002) the etiology of white pox a lethal disease of the Caribbean elkhorn coral, *Acropora palmata*. Proc Natl Acad Sci USA 99:8725–8730

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

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 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

Reimer JD, Mizuyama M, Nakano M, Fujii T, Hirose E (2011a) Current status of the distribution of the coral-encrusting cyanobacteriosponge *Terpios hoshinota* in southern Japan. Galaxea 13:35–44

Reimer JD, Nozawa Y, Hirose E (2011b) Domination and disappearance of the black sponge: a quarter century after the initial *Terpios* outbreak in southern Japan. Zool Stud 50:394

Roff G, Kvennefors ECE, Fine M, Ortiz J, Davy JE, Hoegh-Guldberg O (2011) The ecology of 'acroporid white syndrome', a coral disease from the southern Great Barrier Reef. PLOS ONE 6:e26829

Rogers CS, Muller E, Spitzack T, Miller J (2009) Extensive coral mortality in the US Virgin Islands in 2005/2006: a review of the evidence for synergy among thermal stress, coral bleaching and disease. Caribb J Sci 45:204–214

Rützler K, Muzik K (1993) *Terpios hoshinota*, a new cyanobacteriosponge threatening Pacific reefs. Sci Mar 57: 395–403

Shi Q, Liu GH, Yan HQ, Zhang HL (2012) Black disease (*Terpios hoshinota*): a probable cause for the rapid coral mortality at the northern reef of Yongxing Island in the South China Sea. Ambio 41:446–455

Sridhar R, Thangaradjou T, Kannan L (2008) Comparative investigation on physico-chemical properties of the coral reef and seagrass ecosystems of the Palk Bay. Indian J Mar Sci 37:214–219

Sukumaran S, George RM, Kasinathan C (2008) Biodiversity assessment of a fringing reef in Palk Bay, India. Fish Technol 45:163–170

Sukumaran S, George RM, Vinod K, Sobhana KS, Naomi TS, Manisseri MK (2011) Temporal patterns in biodiversity and health status of reef corals of Palk Bay. Indian J Fish 58:73–77

Thinesh T, Mathews G, Diraviaraj K, Patterson JK (2013) Coral diseases are major contributors to coral mortality in Shingle Island, Gulf of Mannar, southeastern India. Dis Aquat Org 106:69–77

Thinesh T, Jose PA, Hassan S, Selvan KM, Selvin J (2015) Intrusion of coral-killing sponge (*Terpios hoshinota*) on the reef of Palk Bay. Curr Sci 109:1030–1032

Weil E (2004) Coral reef diseases in the wider Caribbean. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin, p 35–68

Weil E (2005) Current status of the marine biodiversity of Puerto Rico. In: Miloslavich P, Klein E (eds) Caribbean marine biodiversity: the known and unknown. DEStech Publications, Lancaster, PA, p 85–108

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, Maragos JE (2008) Phase shift from a coral to a corallimorph-dominated reef associated with a shipwreck on Palmyra Atoll. PLOS ONE 3:e2989

Editorial responsibility: Garriet Smith, Aiken, South Carolina, USA

Submitted: September 14, 2016; Accepted: July 18, 2017
Proofs received from author(s): September 15, 2017