Patterns in the abundance of fish and snail corallivores associated with an outbreak of acute tissue loss disease on the reefs of Vaan Island in the Gulf of Mannar, India

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Abstract Outbreaks of tissue loss diseases in corals, commonly known as white syndromes on Indo-Pacific reefs, are becoming more common resulting in direct colony mortality and also affecting the animals that depend on corals for food and shelter. Following observations of widespread acute tissue loss affecting the coral *Montipora digitata* on a reef in the Gulf of Mannar, India, we compared the density of fish and snail corallivores and abundance of tissue loss lesions between the affected area and an adjacent control area. In the affected area, an average of 50% of the *Montipora digitata* cover had acute tissue loss as compared to 0.33% in the control area. This is the first report of a widespread tissue loss disease for corals on the reefs in the Gulf of Mannar. The site was resurveyed four months later and no signs of tissue loss lesions were evident. Coral-feeding butterflyfish were observed feeding directly on coral lesions in the affected area but belt transects found the density of butterflyfishes to be similar between the affected and control areas and between the two time periods (outbreak and non-outbreak). In contrast, drupellid snails were also observed feeding on lesion margins but abundance was higher in the affected area (95 snails) compared to the control area (2 snails). In the follow-up survey, the densities of drupellids in the affected area had declined and were similar to those within the control site. Drupellids are attracted to damaged coral tissue and it is possible that snails actively migrated into the affected reef area and were taking advantage of the dying coral tissue. Alternatively, a *Drupella* outbreak could have initiated the disease outbreak. Our observations add further evidence to the potential importance of corallivores on disease processes.

Keywords: coral disease outbreak, *Drupella*, butterflyfish, acute tissue loss, *Montipora*, predator response

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Introduction

Coral reefs are on the decline with hard coral cover on Indo-Pacific reefs decreasing by an average of 50% resulting from a combination of chronic anthropogenic stressors and global climate change (Bruno & Selig 2007). Outbreaks of coral disease have also emerged as a recent driver of coral reef decline yet information on disease pathogenesis remains poorly understood (Raymundo et al. 2009). Corals are the foundation species providing shelter, habitats and food for a myriad of other species (Stella et al. 2011). When corals are lost, due to diseases, it will have a detrimental effect on reef-associated animals, especially corallivores that are intimately linked to their coral resources for food and shelter (Pratchett et al. 2006). Corallivores might also inadvertently serve as vectors transmitting disease between colonies, either through predation, injury or pathogen transmission (Sussman et al. 2003, Williams & Miller 2005, Dalton & Godwin 2006). For example, butterflyfish are a known vector of trematodiasis in Hawaiian corals (Aeby 2007) and corallivorous snails transmit disease in *Acropora palmata* in Florida (Williams & Miller 2005). Conversely, coral-associated animals can also have a positive effect on their diseased coral hosts. Pollock et al. (2013) showed that predation on coral lesions by the corallivorous crab, *Cymo melanodactylus*, reduced the rate of tissue loss. There is a wide diversity of animals living in symbiosis with corals (Stella et al. 2011) and we are just beginning to understand their roles in disease processes.

The coral reefs in the Gulf of Mannar, located in southeastern India, face numerous anthropogenic threats including a past history of coral mining and destructive fishing practices and current threats from sedimentation and pollution (Edward et al. 2012). Few studies on coral disease have been conducted in this region, but baseline surveys have reported black band disease, a tissue loss disease (white band) and pink spot (Edward et al. 2012, Thinesh et al. 2013, 2014). In November 2015, during routine surveys on a reef off Vaan Island in the Gulf of Mannar, India we came across an area characterized by an abundance of acute tissue loss lesions on the common reef coral, *Montipora digitata*. Butterflyfish were observed actively feeding on the lesions and drupellids were found attached to and feeding directly on lesion margins. Our objectives were to document the severity of the outbreak and compare the densities of drupellids and coral-feeding butterflyfish in the affected areas with an adjacent control area. The site was resurveyed four months later (March 2016) to examine the longer-term effects of the disease outbreak.

Materials and methods

The Gulf of Mannar covers an area of approximately 10,500 sq. km and includes a chain of 21 uninhabited islands surrounded by fringing and patch reefs (Raj et al. 2015). The survey site was a shallow (~3meters) reef off Vaan Island in the Gulf of Mannar (Fig. 1). The site consisted of

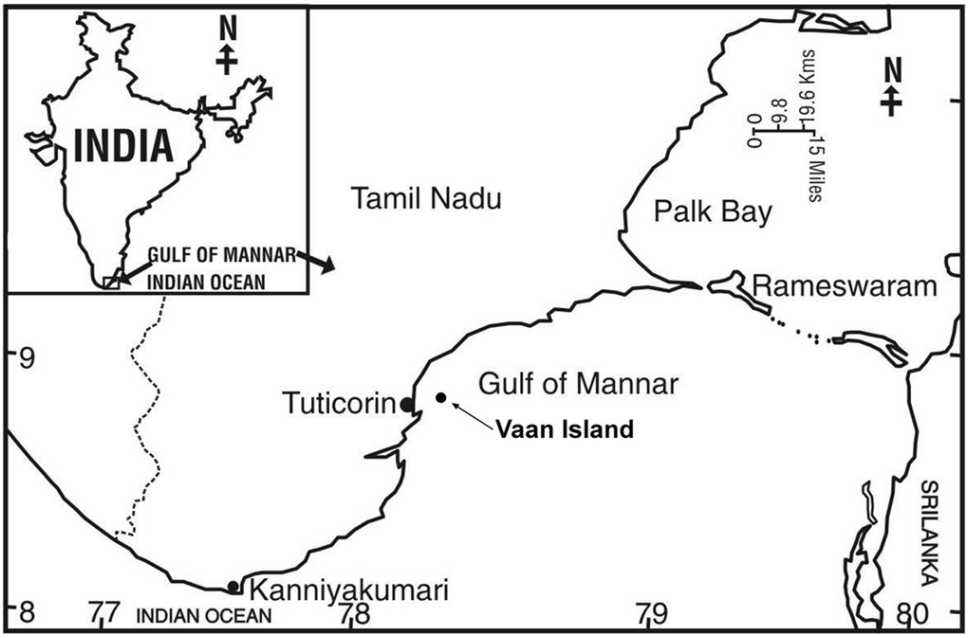

Fig. 1 Map of India showing the location of the Gulf of Mannar and Vaan Island where the disease outbreak occurred

almost monospecific stands of intertwined *M. digitata* colonies, so individual colonies could not be accurately counted within transects. Hence, disease prevalence could not be calculated, so disease severity was determined by measuring the proportion of *M. digitata* cover that had acute tissue loss lesions. Three 20-meter lines were haphazardly laid on the reef parallel to each other and separated by at least 5 meters. Coral cover and tissue loss was quantified via line intercept whereby the substrate underlying the entire 20-meter tape measure was recorded with corals identified to species level and health state (healthy vs. tissue loss lesions). All drupellids within 50 cm on each side of all transect lines (20 x 1m belt) were recorded including the number of animals occurring in any clusters. Drupellids can be cryptic and so all coral surfaces within the belt, including the base and underside of all colonies, were examined. All coral-feeding

butterflyfish within a wider 2.5 meters on each side of the line (20 x 5m belt) were also recorded. Surveys were conducted within the affected area and in a nearby control area on the same reef approximately 200 meters away that was also dominated by *M. digitata.* Survey locations were marked with a handheld GPS unit and the sites were relocated and resurveyed four months later using these GPS units.

Disease severity was defined as the proportion of *M. digitata* cover affected by acute tissue loss. Drupellid and butterflyfish densities were normalized to number of animals/m^2 reef surveyed. A coefficient of dispersion (variance/mean) was calculated for the drupellids within the outbreak area to determine whether snails were clustered (CD>1), overly dispersed (CD<1) or showed no spatial pattern (CD=1) (Sokal & Rohlf 1981).

Results

Lesion description

Large areas of acute tissue loss, as indicated by a bright, white skeleton lacking algal colonization, occurred along colony branches with many branches completed denuded. The

Fig. 2. Photograph illustrating acute *Montipora* white syndrome found on *M. digitata* on a reef in the Gulf of Mannar. White on the colony indicates areas of recent tissue loss

lesions appeared to progress from the base of the branches upward as many branch tips had tissue remaining (Fig 2). Most lesions were bordered by a wide band of grey discolored tissue

that frequently extended up to the tips of the branches (Fig 3). Microscopic examination revealed the grey area to be degraded coral tissue with skeletal elements protruding through.

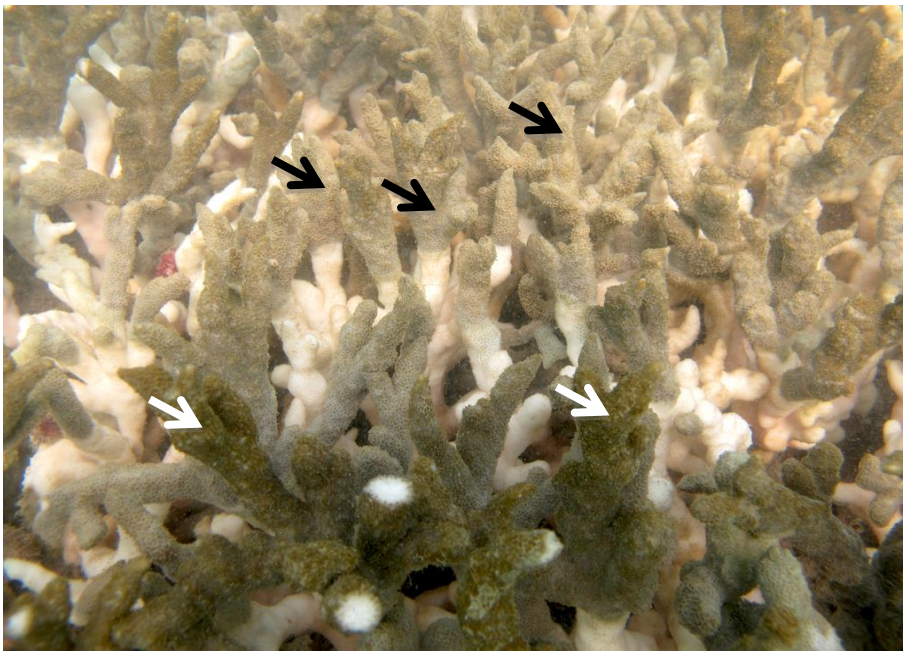

Fig. 3 Photograph showing the extent of the degraded tissue (grey discoloration) on affected colonies. White areas on the colony indicate recent tissue loss. Black arrows indicate areas of degraded tissue. White arrows indicate healthy tissue

Outbreak severity

The reef in the affected area had an average of 77.3% (SE±8.6) coral cover and was dominated by *Montipora digitata* (avg. 76.3 SE±5.8%). 43.5% (SE±8.5) of the *Montipora* cover had acute tissue loss. Within the adjacent control area, coral cover averaged 50.8% (SE±1.7) with 47.3 %(SE±0.4) composed of *M. digitata* cover. Acute tissue loss was found in 0.33% (SE±0.33) of the *Montipora* cover. However, these lesions were attributed to predation, as they were small, discrete areas of tissue loss lacking the grey border indicative of potential disease. No evidence of acute tissue loss (disease) in either the affected or control site was found in the follow-up survey four months later. In the affected area, 29% (SE±5.1) of *M. digitata* cover had the lower half of the branches of colonies dead and covered in algae (Fig 4) and this was presumed to be reflective of the amount of coral cover lost during the prior disease outbreak, including tissue lost to predation. None of the colonies in the control site had this distinct pattern of dead lower branches.

Fig. 4 Photograph illustrating (a) the initial lesions caused by acute *Montipora* white syndrome and (b) 4 months later, the lesion areas covered by algae indicating partial colony mortality

Butterflyfish surveys

Three species of coral-feeding butterflyfish (*Chaetodon octofasciatus, C. plebius, C. collare*) were found along transects within the affected and control areas. Butterflyfish densities were similar between control and affected areas and between time periods within each area (Fig. 5).

Drupellid surveys

During the November 2015 outbreak, drupellid densities were higher within the affected area as compared to the control area but due to the small sample size (n=3 transects/site) formal statistics could not be applied. However, the number of drupellids found within the 60 m^2 (3 transects) of reef surveyed in the affected area (n=95) was much higher than found in the control area (n=2). In contrast, drupellid densities were similar between the affected and control sites when resurveyed in March 2016 (Fig. 6). A total of four snails were found within the affected area and two snails found within the control area. During the outbreak period, snails within the affected area showed a clustered spatial pattern (CD=3.1) ranging from single snails up to clusters of 18.

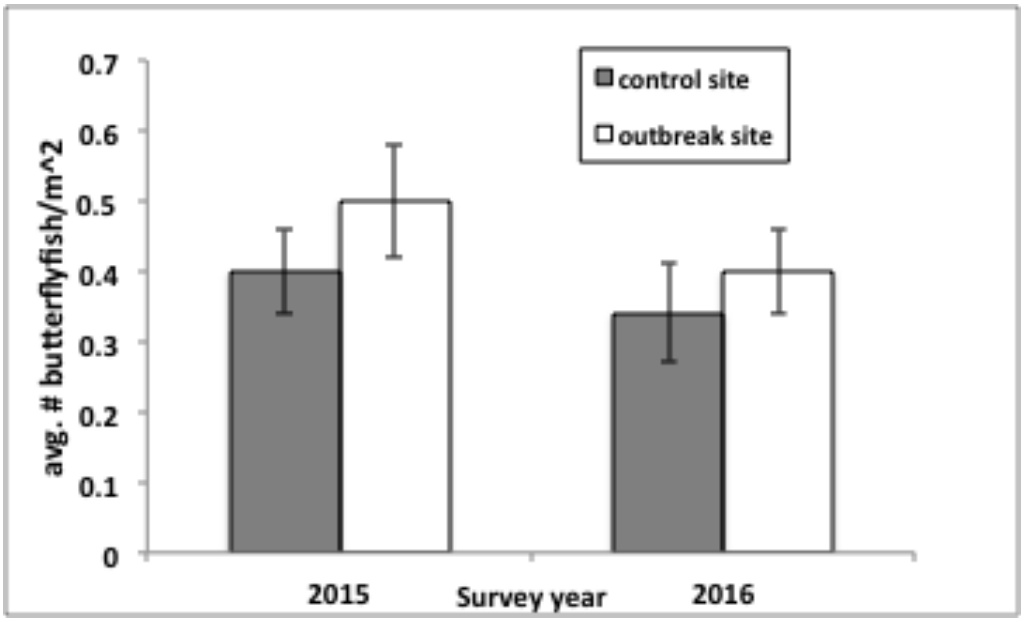

Fig. 5 A comparison of butterflyfish densities within and outside reef areas with acute *Montipora* white syndrome and between outbreak (November 2015) and non-outbreak (March 2016) time periods. Data reflect mean and standard error of number of butterflyfish within three replicate belts in each area

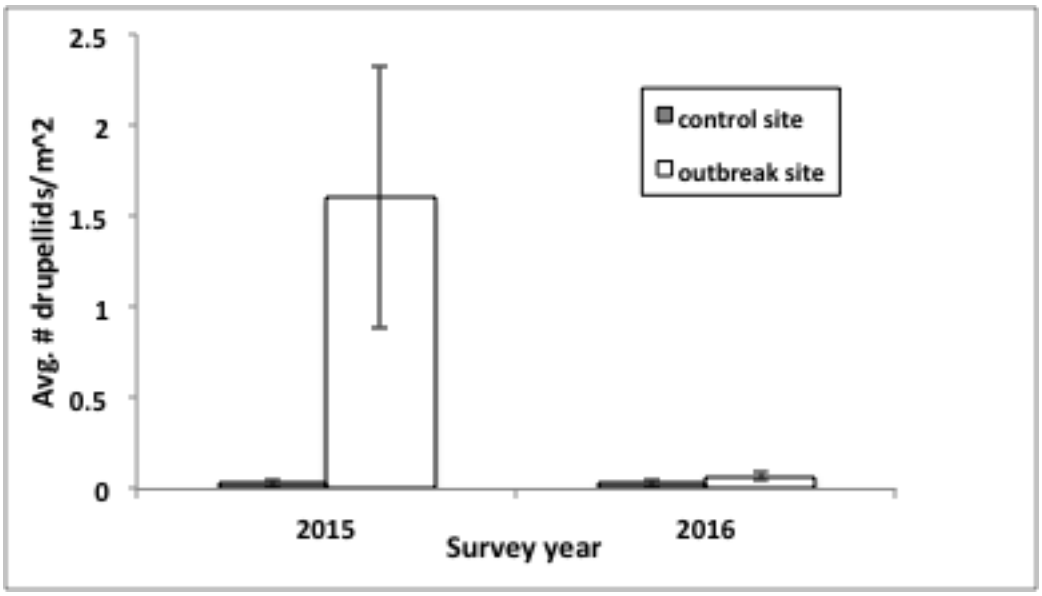

Fig. 6 A comparison of drupellid densities within and outside reef areas with acute *Montipora* white syndrome and between outbreak (November 2015) and non-outbreak (March 2016) time periods. Data reflect mean and standard error of number of drupellids within three replicate belts in each area

Discussion

Numerous coral diseases have been reported from reefs in the Gulf of Mannar, India (Edward et al. 2012, Thinesh et al. 2013, 2014) but this is the first report of an acute tissue loss disease outbreak. We found a localized outbreak of acute *Montipora* white syndrome with tissue loss found on almost 50% of the *M. digitata* cover on a reef off Vaan Island. For most host-pathogen systems, including corals, high host abundance facilitates disease transmission between adjacent individuals (Bruno et al. 2007, Myers & Raymundo 2009). The outbreak site we surveyed was dominated by the affected species, *M. digitata*, so the severity of the outbreak was not necessarily surprising. Similarly, Aeby et al (2011) found high mortality from *Acropora* white syndrome (AWS), at a site in the northwestern Hawaiian Islands that was dominated by a monospecific stand of the susceptible species *Acropora cytherea,* whereas significantly less mortality occurred due to AWS on a more diverse, species rich area in American Samoa.

During follow-up surveys, four months later, we found no signs of recent tissue loss at either the affected or control sites. A number of studies have found disease outbreaks to be ephemeral and tissue loss lesions in corals to wax and wane with time (Aeby et al. 2010, Work et al. 2012). Disease outbreaks occur when conditions change allowing new pathogens to invade or existing pathogens to flourish (Colwell 1996, Dobson & Foufopoulos 2001), and environmental degradation from anthropogenic activity has been suggested as the most important factor contributing to disease outbreaks in wildlife populations (Dobson & Foufopoulos 2001). This appears to hold true for coral populations as coral disease outbreaks are often linked to altered environmental conditions including elevated seawater temperatures (Bruno et al. 2007, Heron et al. 2010), exposure to nutrients (Vega Thurber et al. 2014), sedimentation (Pollock et al. 2014), scuba diver activity (Lamb et al. 2014) or rainy conditions resulting in terrestrial run-off (Haapkyla et al. 2011, Aeby et al. 2016). Although the Gulf of Mannar is under protective status, which reduces fishing pressure, it is still subject to problems associated with poor water quality (Edward et al. 2012). The coastline surrounding the Gulf of Mannar is densely populated, and Gulf waters suffer from untreated domestic sewage and industrial discharge (chemical and thermal) (Easterson 1998, Easterson et al. 2000, Asha et al. 2010, Edward et al. 2012). Water clarity at the study site was always poor ranging from less than a meter to 3 meters (on a good day), indicative of problems with eutrophication. Reduced water quality in the Gulf of Mannar may be contributing to the emergence of disease outbreaks on these reefs.

The control site, which was also dominated by *M. digitata* (the affected coral species), was nearby and thus exposed to similar environmental conditions. Yet no signs of disease were found during the outbreak period, nor was there evidence of partial mortality of coral colonies in the control area during follow-up surveys. This suggests that the disease never spread from the affected site to the control site. How far a disease can spread within host populations would depend in large part on the mode of transmission. The mode of transmission is unknown for the acute *Montipora* white syndrome that we found, but if transmission required direct contact between susceptible hosts then the spatial spread of the disease could be limited by host distribution. Many coral diseases are transmitted via direct contact (Raymundo et al. 2003, Williams & Miller 2005, Aeby et al. 2010) and we saw numerous instances of a new lesion starting at the point of contact with an infected branch. The affected reef area was dominated by an abundance of intertwined and touching *M. digitata* facilitating transmission within the area, but the control site was approximately 200 meters away separated by areas of sand and sea grass beds. The widespread spatial distribution of corals on this reef might help explain why the outbreak appeared to be limited to one area of the reef. A brief broader search of other areas of the reef during the outbreak found no further areas of acute tissue loss lesions.

Disease pathogens can be host-specific, affecting one species, or genera of coral or host-generalist, affecting numerous coral genera. For example, black band disease is known to infect 16 scleractinian genera in the Indo-Pacific (Sutherland et al. 2004) whereas trematodiasis is limited to corals in the genus *Porites* (Aeby 1998). Although other coral genera were present in transects surveyed at the outbreak site, no other coral specie exhibited tissue loss lesions, suggesting this disease could be specific towards *M. digitata*. However, other coral species were much less abundant in the area, so disease susceptibility in these species cannot be dismissed.

Outbreaks of tissue loss diseases in corals are becoming more common (Harvell et al. 2007, Maynard et al. 2015), resulting in direct colony mortality, which in turn affects the animals that depend on corals for food and shelter (Pratchett et al. 2006). Many reef-associated animals are attracted to and directly feed on coral lesions, and thus can affect disease processes (Aeby 1993, Morton et al. 2002, Chong-Seng et al. 2011, Pollock et al. 2013). Fireworms (Sussman et al. 2003), snails (Williams & Miller 2005, Nicolet et al. 2013, Gignoux-Wolfsohn et al. 2012) and butterflyfish (Aeby & Santavy 2006, Aeby 2007, Raymundo et al. 2009, Chong-Seng et al. 2011) have all been suggested as vectors of coral diseases, and predation by crown of thorn seastars

(Katz et al. 2014), snails (Nicolet et al. 2013) and reef fish (Aeby & Santavy 2006, Raymundo et al. 2009) can leave corals more vulnerable to infection. Conversely, crabs (Pollock et al. 2013) and butterflyfish (Aeby 1993, Cole et al. 2009) have been found to remove infected coral tissue allowing corals to recover from diseases. Our understanding of the role corallivores play in disease processes is limited thus we examined the potential *in situ* response of butterflyfish and snail coral predators to acute tissue loss lesions. We compared the density of butterflyfish and drupellids in the affected area to an adjacent control site and examined the density of animals in the two areas following the end of the outbreak. Butterflyfish were observed feeding directly on the coral lesions in the affected area, and this has been found for numerous fish species (Chong-Seng et al. 2011). However, no differences in butterflyfish densities were found between affected and control areas or between time periods during and after the outbreak. This suggests that while butterflyfish may be opportunistically feeding on coral lesions within their territories or home ranges, they are not actively moving into the area in response to the presence of widespread tissue loss. Many fish species, including butterflyfish, are territorial, aggressively keeping other species out of their territories (Tricas 1989, Roberts & Ormond 1992). Hence, the movement patterns of the butterflyfish may have been constrained by the behaviors of other reef fish. For example, Gochfeld (2010) examined the effect of territorial damselfish on the feeding behavior of butterflyfish and found that coral colonies within damselfish territories were protected from predation. However, once colonies were transplanted outside of damselfish territories, they were rapidly fed upon by several species of butterflyfishes.

In contrast, the abundance of drupellids was higher in the affected area (95 snails) compared to the control area (2 snails). Snails in the affected area also had a clustered distribution with aggregations of snails found feeding on coral lesions. It is not clear whether coral mortality resulting from disease attracts *Drupella*, or whether *Drupella* outbreaks promote coral disease (Antonius & Riegl 1997, 1998). The tissue loss lesions could have solely been the result of predation (drupellid outbreak), but we rejected that hypothesis based on a number of observations. First, the overall amount of acute tissue loss found on the reef was extremely high and much greater than would be expected from the number of snails that we found. Drupellid densities within the outbreak area, although higher than the control area, were not outside the normal range reported elsewhere. We found 1.6 snails/m^2 during the outbreak period compared to normal drupellid densities ranging from <1 snail/m^2 (Zuschin et al. 2001, Zuschin &

Stachowitsch 2007) to 2.9 snails/m^2 (Schoepf et al. 2010). In contrast, up to a hundred snails per coral colony have been reported during *Drupella* outbreaks (Boucher 1986) far greater than the total number of snails (95 snails within 60m^2) or the maximum number of 18 snails per colony that we found. Second, acroporids are a preferred food source for drupellids (Morton et al. 2002, Al-Horani et al. 2011) and we found no tissue loss on the acroporids within transects. Finally, the obvious degraded coral tissue (grey lesions) found on affected colonies was similar to disease signs found on montiporids in other regions (Aeby et al. 2016) and were not consistent with snail predation.

Drupellids are attracted to conspecifics as well as to damaged coral tissue (Morton et al. 2002, Schoept et al. 2010, Nicolet et al. 2013). It is likely that during the disease outbreak, the acute tissue loss lesions on corals attracted nearby snails with further snail aggregations occurring in response to feeding conspecifics. Four months later within the prior affected area, corals no longer had tissue loss lesions and accordingly, few snails were found. The question still remains as to whether snails were involved in spreading the disease among colonies or conversely, helped to slow-down the disease by feeding on diseased tissue and potentially removing any pathogens there within. Disease outbreaks are occurring with increasing frequency worldwide and are predicted to continue to increase through time (Burge et al. 2014, Maynard et al. 2015). Management of disease outbreaks depends on an understanding of the underlying drivers, including how the myriad of animals living in symbiosis with corals, are affecting disease processes.

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