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Australian subtropical white syndrome: a transmissible, temperature-dependent coral disease

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Abstract. Since 2000, a disease displaying white-syndrome characteristics has been observed affecting corals from the genus *Turbinaria* in the Solitary Islands Marine Park, New South Wales, Australia. Recently termed Australian subtropical white syndrome, this disease is transmissible through direct contact and by a predatory vector, but transmission through the water column has not been observed. In aquarium experiments, progressive tissue loss, extending from the region where healthy *Turbinaria mesenterina* fragments were in direct contact with samples of diseased coral, was noted in 66% of treatments. No tissue loss occurred in any of the controls or when healthy fragments were not in direct contact with diseased corals. Field experiments confirmed that the disease was infectious through direct contact. Further experiments showed that the rate of tissue loss was significantly higher when corals were exposed to summer temperatures (26°C). These results suggest that temperature increases predicted in most climate change models could lead to the loss of dominant coral species, displacing other organisms that rely on corals for food and shelter. Finally, the present study showed that removal of the disease margin provides a management tool to minimise coral tissue loss during an epizootic.

Additional keywords: elevated temperatures, infectious disease, subtropical reefs, *Turbinaria mesenterina*.

Introduction

The incidence of diseases affecting scleractinian corals and other cnidarians has increased over the past 20 years, resulting in a decline in coral cover and a shift towards algal-dominated communities at some tropical locations (Sutherland *et al*. 2004). Owing to the rapid emergence of highly virulent coral diseases/syndromes in recent times, the Caribbean region has been classified as a 'coral disease hotspot' (Green and Bruckner 2000). A total of 22 conditions, affecting 85% (45 species) of all scleractinian coral species present, have been reported from this region (Weil 2004). However, despite the greater species richness and larger geographical area, only 12 conditions have been reported from the Indo-Pacific (Willis *et al*. 2004). Willis *et al*. (2004) reported the occurrence of eight diseases/syndromes on the Great Barrier Reef (GBR) and noted a 20-fold increase in white syndrome (WS) prevalence throughout the southern GBR between 1998 and 2003.

In northern New South Wales (NSW), Australia, coral disease was first observed within the Solitary Islands Marine Park (SIMP) in 2000 (Edgar *et al*. 2003). Observations at that time indicated that many dominant coral species were affected by tissue loss that progressed as a distinct line across plate, encrusting and branching coral morphs. Dalton and Smith (2006) reported that up to six common subtropical coral genera were affected by progressive tissue loss and that the prevalence of this epizootic was higher during warmer months. In addition, the rate of tissue loss within affected colonies increased at higher seawater temperatures.

Currently the aetiology of the epizootic within the SIMP and the origin of the pathogen remain unknown. However, Dalton and Godwin (2006) identified a potential vector when they observed progressive tissue loss from feeding scars of a corallivorous nudibranch (*Phestilla* sp.); tissue loss continued following the removal of the nudibranch. The transmission of disease from an infected coral fragment that had been in contact with the nudibranch to healthy fragments led Dalton and Godwin (2006) to suggest that the disease was transmissible through direct and indirect contact (refer to Sutherland *et al*. 2004 for definitions). This potentially explained the clustered pattern of diseased colonies observed within the SIMP (Dalton and Smith 2006).

The possible role of microbial pathogens in this epizootic remains unclear. Preliminary microscopic examination revealed no evidence of fungal or protistan organisms associated with field-collected, diseased samples (Godwin 2007); however, both culture-based and culture-independent assessments have found a clear difference in the bacterial communities associated with healthy and diseased *Turbinaria mesenterina* corals (Godwin 2007). To date, many well-characterised coral diseases have been found to have bacterial aetiologies (reviewed in Rosenberg *et al*. 2007). Therefore, the bacterial community associated with the epizootic in the SIMP warrants further investigation.

Disease outbreaks are affected by abiotic factors, such as increased seawater temperature, eutrophication, sedimentation and pollution (reviewed in Sutherland *et al*. 2004). Temperature stress has been linked to at least 10 previously described

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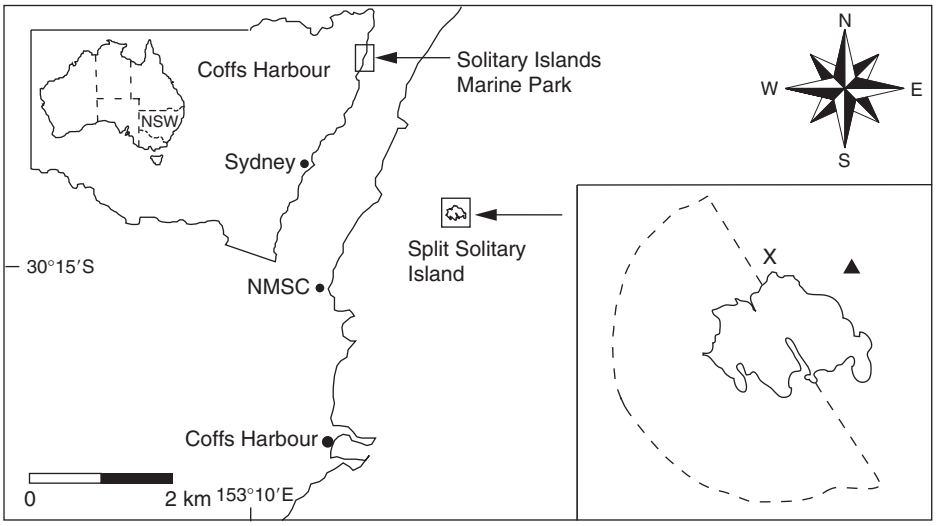

Fig. 1. Location of Split Solitary Island (SSI) within the southern region of the Solitary Islands Marine Park. Location of the site where the disease transmission experiment was conducted and where the *Turbinaria mesenterina* samples for the aquarium studies were collected (X). Location of the VEMCO VR2 acoustic listening station (▲). The sanctuary zone boundary is indicated by the broken line.

coral disease epizootics including: black band disease (Antonius 1981; Boyett *et al*. 2007); shut down reaction (Antonius 1977); white pox disease (Patterson *et al*. 2002); dark spot disease (Gil-Agudelo and Garzon-Ferreira 2001); and diseases caused by *Vibrio* species (Toren *et al*. 1998; Ben-Haim *et al*. 2003; Cervino *et al*. 2004). Gross characterisation of the stresses affecting hard corals has previously been problematic because many causes of the lesions, both from abiotic stressors (temperature, increased nutrients and variable salinity levels) and biotic stressors (competition, predation and disease), may appear outwardly similar. For example, a suite of white diseases/syndromes has been documented throughout the tropics and all have some phenotypic similarities. This led Work and Aeby (2006) to highlight the importance of a systematic approach to describing gross lesions because, without such an approach to *in situ* classification, geographical comparisons may be confounded.

Initial observations of spreading tissue mortality affecting scleractinian corals within the SIMP were characterised by a region of recently exposed coral skeleton that may or may not have sloughing tissue present between apparently healthy tissue and the exposed skeleton (Dalton and Smith 2006). This epizootic appeared to have similar phenotypic characteristics to the WS described by Willis *et al*. (2004). However, the coral species affected by WS throughout the GBR were from the families Acroporidae, Pocilloporidae, Poritidae and Faviidae. There were no reports of WS affecting *Acropora solitaryensis*, *Psammocora* and *Turbinaria* spp., all of which have been observed with progressive tissue loss at subtropical locations. More recently, on the GBR, Ainsworth *et al*. (2007) failed to detect bacterial communities at the lesion boundary of plate-forming acroporian corals affected by WS and consequently suggested that tissue loss was primarily caused by programmed cell death.

The microscopic characteristics of the disease affecting *Turbinaria* spp. in subtropical eastern Australia appear to differ from those described for WS on the GBR. Microbiological assessment of disease lesions consistently reveals a distinct bacterial community in the actively advancing disease margin, with significantly higher numbers of *Vibrio* spp. and *Roseovarius* spp. present in the diseased tissue than in the neighbouring healthy tissue (Godwin 2007). Such changes in the bacterial community suggest that bacterial processes contribute to the degradation of the coral tissue. In light of these dissimilarities, the name Australian subtropical white syndrome (ASWS) has been adopted to distinguish this epizootic from other white disease/syndrome outbreaks. This nomenclature is consistent with the protocol proposed by Work and Aeby (2006) as it: (*i*) describes the geographical distribution of the disease; (*ii*) identifies the hosts; and (*iii*) characterises the morphology of the lesions, which are similar to WS on the GBR, but may have different aetiology to other reported WS epizootics.

The present study aimed to describe progressive tissue loss in corals in the SIMP to compare with previously described WS outbreaks on the GBR and in other Indo-Pacific regions. More specifically, we conducted *in situ* observations and laboratory and field experiments to: (*i*) systematically describe the lesions caused by ASWS in the SIMP; (*ii*) determine the possible mode of disease transfer; and (*iii*) determine whether seawater temperature affects the rate of infection between colonies and disease virulence (rate of tissue loss across affected coral colonies).

Materials and methods

Study site

Reefs adjacent to Split Solitary Island (SSI) (30°14′S, 153°10′E; Fig. 1) were selected for the collection of *Turbinaria mesenterina* fragments for aquarium experiments and for an *in situ* disease transmission experiment. All aquarium experiments were carried out inside a controlled environment room at the National Marine Science Centre (NMSC), Coffs Harbour, NSW, from 2004 to 2007. *Turbinaria* corals growing on

greywacke bedrock dominate the epibenthic community on the reefs adjacent to this island (Harriott *et al*. 1994). The temperature of the seawater varies between 17 and 26°C in this region (AIMS 2007).

Disease transmission aquarium experiment

Six samples (~30 cm × 10 cm) from healthy *T. mesenterina* colonies were collected on 2 October 2004, at a depth of 10–12 m, using a hammer and chisel to fragment the colonies. The fragments were placed into individual plastic resealable bags, filled with seawater and transported to the NMSC in a 60-L plastic container filled with seawater. On return to the NMSC, all samples were further broken into ~25-cm^2 portions and placed into individual 25-L tanks located in an outside raceway. Each tank was supplied with flow-through seawater, aerated and shaded from direct sunlight with 70% shade cloth, which resulted in a maximum light intensity similar to *in situ* conditions (150 μmol photons m^{-2} s^{-1}). These replicate portions were monitored for 1 week after which those that showed signs of tissue recovery at the broken margins, no decrease in pigmentation, and no signs of tissue sloughing were used in the disease transmission experiment. Four replicate portions from each of the six colony fragments were used in this experiment.

Twelve 7-L aquaria in a controlled environment room were filled with 6 L of sand-filtered seawater (FSW) and aerated with an air stone. The water temperature was maintained at 21°C (ambient seawater temperature). Lighting in the room was supplied by four 40-W, 10 000-Kelvin and four 40-W actinic fluorescent tubes and the fragments were exposed to 100 μmol photons m^{-2} s^{-1} of light for 10 h day^{-1}. Two healthy coral fragments, ~5 cm apart, were randomly placed in each aquarium and monitored for 5 days, during which time all appeared healthy.

At SSI, six colonies of *T. mesenterina* displaying progressive tissue loss (Fig. 2) were fragmented adjacent to the disease margin using a hammer and chisel, leaving at least 20 mm of live coral tissue adjacent to the exposed margin. These fragments were placed into separate, plastic, resealable bags that were filled with seawater and transported to the NMSC in a 60-L plastic container filled with seawater. On return to the NMSC, these fragments were randomly placed onto one of the healthy fragments in each of the six treatment aquaria. This resulted in six healthy coral fragments in direct contact with a diseased fragment and six healthy fragments ~5 cm away from the diseased fragments. As a procedural control, healthy coral fragments were placed in direct contact with another healthy fragment in six control aquaria and monitored for any signs of tissue loss (which may occur as a result of colony interactions or smothering). The fragments were monitored daily for signs of spreading tissue loss. The FSW was replaced every 4 days and the wastewater was appropriately sterilised before disposal. Each control and treatment replicate was photographed and, using Image-Pro software, the rate of tissue loss was determined for each fragment that displayed tissue loss. In addition, tissue that sloughed off the coral skeleton was viewed daily under a stereomicroscope (up to 1000× magnification) and the microbes were photographed to observe any changes in the composition of the microbial community associated with the sloughing tissue.

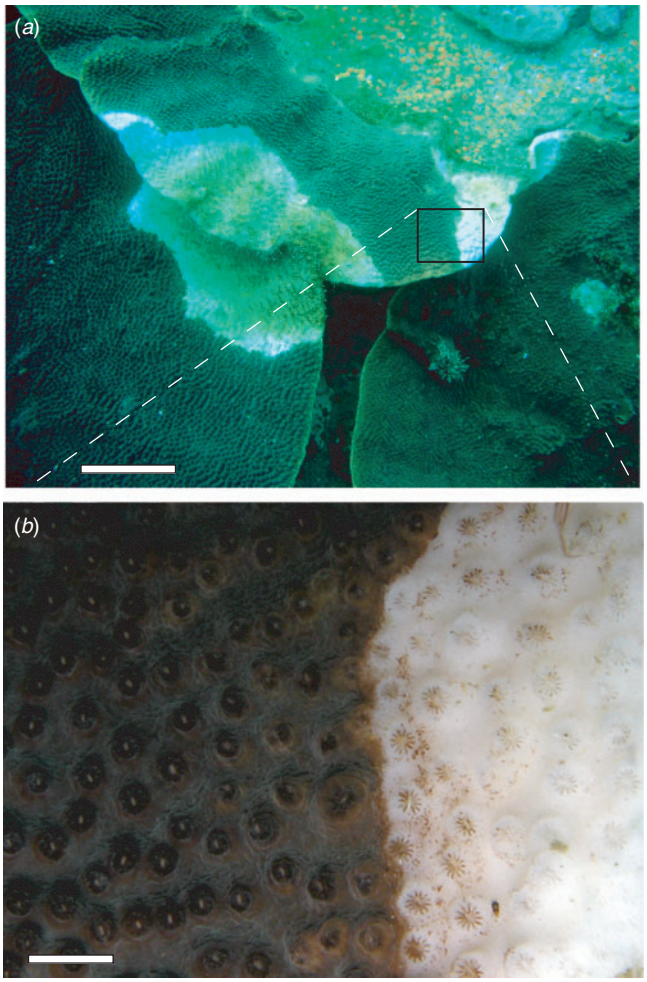

Fig. 2. (*a*) Three colonies of *Turbinaria mesenterina* in direct contact displaying progressive tissue loss at the touching margins. Scale bar = 40 mm. (*b*) Close up of the affected region showing the demarcation between apparently healthy coral tissue and the recently exposed coral skeleton. Scale bar = 5 mm.

Disease transmission in situ

On 17 January 2007, colonies of *T. mesenterina* on the northern side of SSI at a depth of 10–14 m were evaluated for signs of spreading tissue mortality. One week later, using a pneumatic grinder with a diamond-tip blade, 21 coral fragments were removed from colonies displaying progressive tissue loss. At least 20 mm of apparently healthy coral tissue remained adjacent to the region where sloughing tissue was noted in each fragment. Latex gloves were used when handling diseased fragments to ensure that no cross contamination occurred between diseased and healthy controls. These fragments were attached to colonies that showed no signs of previous stress using 30-mm plastic builders' clamps (diseased–healthy treatments). To account for stress resulting from interactions between *T. mesenterina* corals, nine coral fragments (~25 cm^2) were removed from apparently healthy colonies using the grinder and clamped onto different healthy corals (healthy–healthy control). Finally, nine builders'

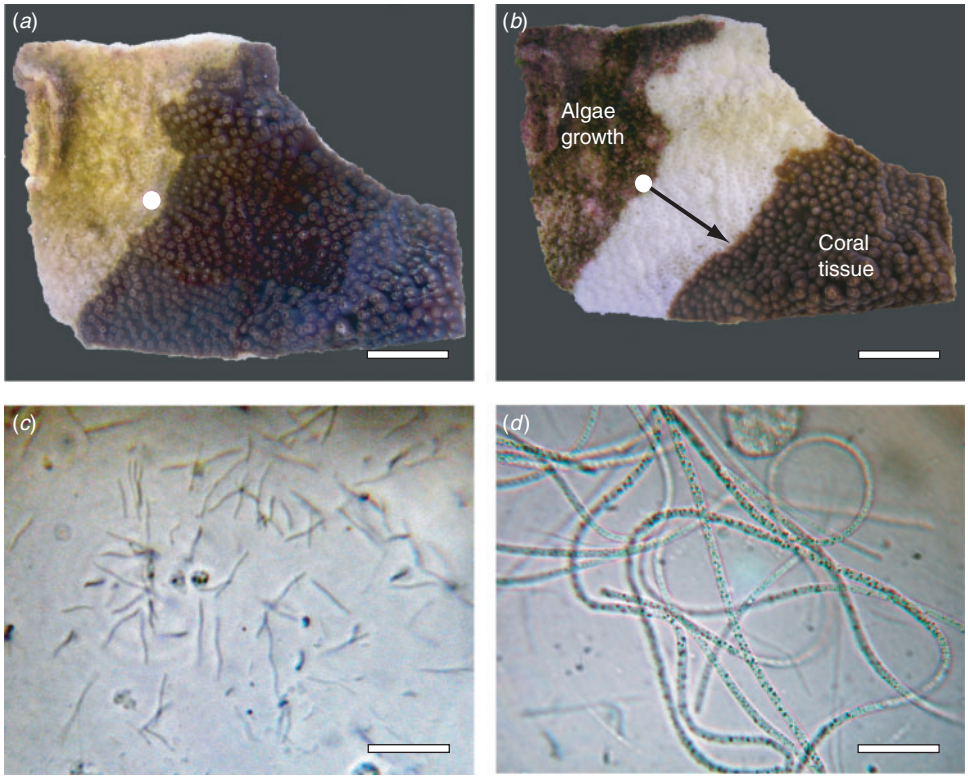

Fig. 3. (*a*) Diseased fragment used in the temperature progression experiment; the white circle indicates the reference mark for linear measurements; the purple hue on the coral tissue was caused by the actinic lighting. Scale bar = 10 mm. (*b*) Progressive tissue loss (arrow) observed during the temperature progression experiment. Scale bar = 10 mm. (*c*) Motile spirochaete-like bacteria observed colonising sloughing coral tissue. Scale bar = 20 μm. (*d*) Opportunistic gammaproteobacteria (possibly *Beggiatoa* sp.) covering dead coral tissue in the laboratory experiments. Scale bar = 40 μm.

clamps were attached to healthy colonies to determine if there was a clamp effect (clamp control).

All replicates were photographed during the initial and subsequent dives and monitored weekly for 4 weeks for signs of any tissue loss extending from the point of contact with the treatment fragments. When tissue sloughing was observed extending from the treatment-colony interface, the length of tissue loss was measured. All diseased colonies used in the experiment as the source of disease fragments were also monitored weekly over the duration of the experiment to determine if removing the diseased margin stopped disease progression. Hourly seawater temperature data from the SSI VEMCO VR2 acoustic listening station at a depth of 14 m, 100 m from the field experiment site (Fig. 1), were obtained from the NSW Department of Primary Industries and used to calculate average daily seawater temperature data during the *in situ* experiment.

Temperature effects on disease progression

To determine if there was a relationship between seawater temperatures and the rate-of-spread of disease across affected *T. mesenterina* corals, a controlled temperature experiment was carried out. During early summer 2005, eight diseased fragments were removed from *T. mesenterina* colonies at SSI using a hammer and chisel. These fragments were transported in plastic resealable bags filled with seawater and individually randomised into eight, 14-L aquaria filled with FSW and positioned underneath fluorescent lights, as previously described.

Before starting the disease progression experiment, a pin was positioned at the interface between the living coral tissue and exposed skeleton (Fig. 3*a*) as a reference point for subsequent measurements of tissue loss. The water temperature in four of the treatment aquaria was increased over a 4-h period from 21 to 26°C. During warmer months, corals are regularly exposed to daily temperature fluctuations of up to 5°C within the SIMP (AIMS 2007). Using sterilised vernier callipers, the distance from the reference pin to the coral tissue/exposed skeleton interface was recorded for all fragments every second day throughout the experiment (Fig. 3*b*). The experiment continued for 8 days and the rate-of-spread of disease across the fragments in the 21°C control and 26°C treatment aquaria was averaged within each measuring period.

Statistical analysis

The aquarium and *in situ* transmission experiments were analysed using Fisher's exact 1-tailed tests (FET); the categorical response variables progressive tissue loss and no tissue loss were compared between healthy corals exposed to diseased fragments (treatments) and healthy fragments (controls). Disease

progression data from the temperature experiment were tested for homogeneity of variance and normality via Levene's and the Anderson–Darling test, respectively; no transformation was necessary. Using Minitab 13 software, the rate of tissue loss per day, calculated from measurements recorded during the temperature experiment, was analysed via repeated-measures ANOVA and the data were compared between temperatures. The analyses tested the hypothesis that seawater temperature affects the rate of tissue loss in corals affected by ASWS. Finally, *post-hoc* (Tukey pair-wise) comparisons were used to test for differences within treatments over time.

Results

Characterisation of ASWS

In *Turbinaria* corals, progressive tissue loss can be described generally by the initial appearance of exposed coral skeleton at the outer edge (peripheral), adjacent to regions of abnormal skeletal growth, or where an accumulation of sediment occurs on corals with laminar, plate or encrusting morphology. Observations from the present study indicated that, typically, the lesion progressed through the colony as a distinct smooth or serpiginous (wavy or indented) margin, with tissue loss on the upper and lower sides of laminar corals and uniform progression along branches. In all affected coral species, lesion progression generally displayed a distinct margin of exposed skeleton adjacent to apparently healthy coral tissue; however, during calm sea conditions, sloughing tissue remained attached. Exposed coral skeleton was rapidly colonised by encrusting and filamentous algae (i.e. within days following tissue loss).

Disease transmission aquarium experiment

Disease transmission and coral tissue loss were only observed in healthy fragments that were in direct contact with diseased fragments. Coral tissue loss, extending from the point of contact between one healthy fragment and a diseased fragment, was observed within 2 days of the start of the experiment. Within 6 days, 66% of healthy coral fragments in direct contact with diseased fragments displayed tissue loss. Tissue loss adjacent to the region where the healthy fragment touched the diseased samples advanced at a rate of 1.28 ± 0.11 mm day^{-1} (mean $\pm$ s.e.) over the 2-week experimental period and tissue loss was greatest during the first 6 days of observation. On day 9, two of these affected fragments developed a sheet of obvious white filamentous growth on their surface, which may indicate a secondary infection. Consequently, tissue-loss measurements recorded from these replicates during day 6 and on subsequent measurement days were removed from the dataset. The network of fine filaments quickly extended over the sloughing tissue and then further covered the remaining coral tissue. Microscopic assessment revealed motile filaments that contained reflective sulfur granules, characteristic of the bacterium genus *Beggiatoa* and other gammaproteobacteria. By the end of the experiment, coral tissue remained on the skeleton of only one of the fragments that displayed progressive tissue loss.

Disease did not occur in healthy fragments that were in the same treatment aquarium, but not in direct contact with diseased fragments. In addition, no tissue loss or obvious stress (i.e. increased mucus production) was observed in any of the control replicates (both direct and indirect). However, partial tissue bleaching under the region where healthy fragments were covered by other healthy fragments was noted in all controls; subsequent recovery of pigmentation was observed following the removal of the upper fragments.

Microscopic observations of all affected treatment fragments indicated a succession of microbial colonisation. Following initial tissue loss, large numbers of motile, spirochaete-like bacteria (10–15 μm long and ~2 μm wide) were observed at the coral tissue/exposed skeleton interface, 4 days after the onset of the experiment (Fig. 3*c*). During days 6 and 8, many different ciliates were observed feeding on sloughing coral tissue, bacteria and zooxanthellae. On day 9, *Beggiatoa* spp. or similar bacteria were observed covering two of the treatment fragments (Fig. 3*d*).

Disease transmission in situ

The temperature of the seawater ranged from 19 to 24.5°C during the *in situ* transmission experiment. During the first week of the experiment, three of the diseased fragments were lost from the treatment colonies, thus reducing the number of diseased–healthy treatments from 21 to 18. On the first observation dive (30 January), one healthy *T. mesenterina* colony in direct contact with a diseased fragment displayed tissue loss extending 48 mm from the point of contact. During the second week (6 February), coinciding with the highest seawater temperature (24.5°C), progressive tissue loss was observed in 11 diseased–healthy replicates (Fig. 4). During the third week (13 February), only three diseased–healthy replicates displayed further tissue loss; none was recorded during the final week of the experiment.

Direct contact of diseased fragments with healthy coral tissue caused significant, progressive tissue loss when compared with healthy–healthy controls (FET, $P = 0.01$). No tissue loss was observed in any of the control replicates; minor bleaching, however, was noted below some of the control fragments. The rate of disease progression was highest when the seawater temperature was above 21°C, and ceased when the temperature dropped below 21°C (Fig. 4). The maximum rate-of-spread of the disease was 6.85 mm day^{-1} during the first week, with a general trend for tissue loss to slow and even cease in some treatments following initial infection (Fig. 4). During the final observation week, tissue growth over previously exposed coral skeleton was noted in seven treatment corals.

Monitoring disease spread following the removal of the diseased margin

Eighty per cent of diseased colonies displayed no subsequent tissue loss (adjacent to the cut region) following the removal of the diseased margins. The tissue loss observed in one diseased colony resulted from water movement caused by the rotation of the grinder blade. During the second week, tissue growth was noted along the cut margin in all diseased colonies. Active disease, and consequent tissue loss, was present at regions away from the cut boundary in one of the monitored corals. No obvious stress was noted in any of the healthy control colonies that were cut using the grinder, with tissue growing over the cut region within a week. There was no tissue loss associated with clamp contact; indeed coral tissue growth over the clamp was noted in two of the clamp-control colonies.

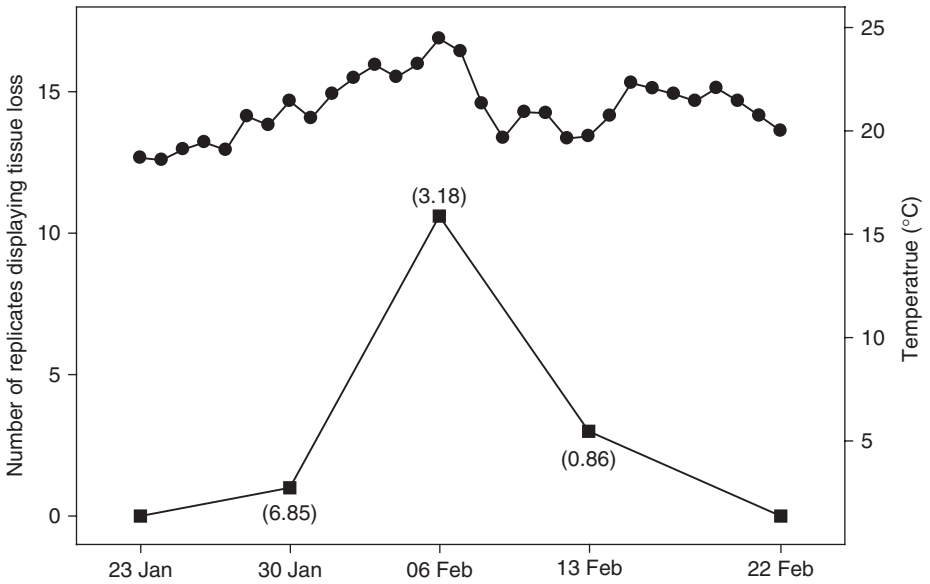

Fig. 4. Number of diseased–healthy treatment colonies observed with progressive tissue loss (squares) and the average daily seawater temperature (circles) recorded during the study period. The average rate of tissue loss (mm day^{-1}) within replicate colonies directly exposed to diseased fragments is indicated in parentheses.

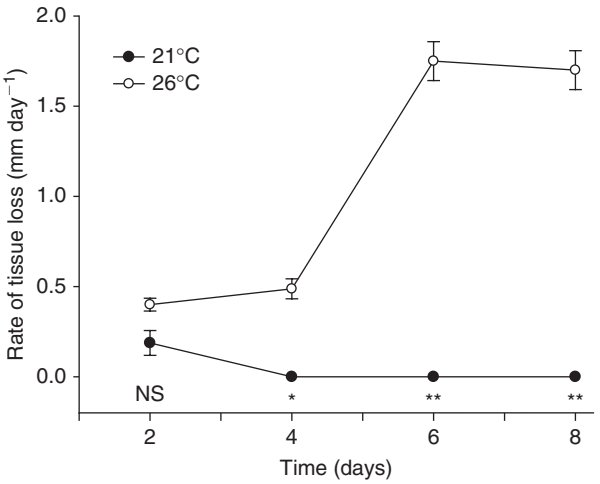

Fig. 5. Mean rate (±s.e.) of coral tissue loss extending across diseased *Turbinaria mesenterina* fragments exposed to 21 and 26°C. Measurements were taken every second day for 8 days. Within-time Tukey *post-hoc* comparisons are shown. NS, not significant; *$P < 0.05$; **$P < 0.001$.

Temperature effects on disease progression

The linear rate of tissue loss increased with time for the diseased fragments maintained at 26°C. The mean rate of tissue loss increased from 0.4 mm day^{-1} during day 2 to a maximum of 1.75 mm day^{-1} after 6 days of exposure to higher temperatures (Fig. 5). However, this pattern was not apparent for fragments held at 21°C; these fragments displayed advancing tissue loss only on day 2, with no further loss observed during subsequent measurements (Fig. 5).

The rate of tissue loss was significantly higher at 26°C than 21°C ($F_{1,6} = 246.40$, $P < 0.001$). Significant effects were also evident for time ($F_{3,31} = 94.92$, $P < 0.001$) and the temperature × time interaction ($F_{3,31} = 129.32$, $P < 0.001$), the latter resulted from the increased rate of tissue loss over time within the 26°C treatment and the decreased rate of loss at 21°C over the same period. The mean rate of tissue loss at 26°C increased slightly between days 2 and 4 and there was a significant increase in disease progression between days 4 and 6 ($F_{1,7} = 108.14$, $P < 0.001$). At all times, the rate of tissue loss was significantly higher at 26°C than at 21°C (Fig. 5). In contrast, the mean rate of tissue loss within the control fragments was significantly higher during the first measurement period compared with subsequent measurements ($F_{1,15} = 25.96$, $P < 0.001$).

Discussion

Comparison of ASWS with other 'white diseases'

The ASWS affecting corals in the SIMP differs from other 'white diseases' described elsewhere. Progressive tissue loss exposing the coral skeleton (white syndrome) has not been reported to affect *Turbinaria* species on the GBR (Willis *et al*. 2004). Indeed, only black band disease has been reported to affect this genus on tropical Australian reefs. Currently, WS affecting corals of the GBR and the Indo-Pacific has not been shown to be transmissible between colonies in the field or laboratory. Willis *et al*. (2004) defined WS as 'a collective term to describe conditions resulting in white bands of tissue and/or skeleton on GBR corals', which included conditions such as white band, white plague, white pox, patchy necrosis and shut-down reaction. This holistic classification appears to incorporate coral stress conditions that have different pathologies and possibly different aetiologies. For example, Patterson *et al*. (2002) found that the common faecal enterobacterium *Serratia marcescens* caused white pox affecting *Acropora palmata* throughout the Caribbean.

In contrast, Ainsworth *et al*. (2007) found that WS affecting *Acropora* spp. lacked microbial communities at the diseased margin and suggested that apoptosis was the main mechanism of tissue loss. Roff *et al*. (2008) named the 'disease-like syndrome' affecting *Acropora hyacinthus*, which reportedly had the same pathology as the epizootic reported by Ainsworth *et al*. (2007), 'Acroporid white syndrome'. In contrast, Sussman *et al*. (2008) concluded that a member of the bacteria family Vibronacae (98% similarity to the coral bleaching pathogen *Vibrio coralliityticus*) was the pathogen causing WS in *Montipora aequituberculata* colonies adjacent to Nelly Bay, GBR. Similarly, Godwin (2007) isolated bacteria from corals affected by ASWS that were closely related to *Vibrio harveyi*. Strains of *V. harveyi* have been reported to be associated with previous disease outbreaks, including rapid tissue necrosis affecting *Pocillopora damicornis* (Luna *et al*. 2007) and white band disease type II (Gil-Agudelo *et al*. 2006). Future research will investigate the relationship between this potential pathogen and disease in the coral communities in eastern Australia.

Disease transmission

The results of these experiments indicate that the ASWS affecting *Turbinaria mesenterina* in the SIMP is transmissible through direct contact, both in laboratory and natural conditions. However, transmission through the water column was not observed. This result is consistent with field observations that apparently healthy *T. mesenterina* colonies in direct contact with diseased corals will, through time, display progressive tissue loss from the point of contact. Wider observations in the SIMP indicate that transmission across touching colonies occurs not only within a species, but also between species within the same genus, and between species from different genera (Dalton *et al*. 2010). This suggests a single aetiology for the disease affecting these species. These patterns are consistent with those of the 'rapid tissue loss' epizootic observed affecting *Acropora cericornis* colonies at White Dry Rocks, Key Largo, Florida (Williams and Miller 2005). In that study, experiments demonstrated that the condition was transmissible both through direct contact and via the corallivorous mollusc *Coralliophila abbreviata*. Interspecies transmission was also observed (Williams and Miller 2005). Raymundo *et al*. (2003) showed that *Porites* ulcerate white spot disease (PUWS) was infectious through direct contact; these researchers also observed transmission through the water column. In contrast, indirect (through the water column) transmission was not observed in the present *T. mesenterina* aquarium experiments.

The demonstrated importance of direct contact in disease transmission, however, poses a conundrum: how does the disease maintain such a large geographical distribution across subtropical corals if the pathogen is exotic to these locations? Progressive tissue loss has been observed affecting *Turbinaria* spp. and many other coral species along the east coast of Australia between 26°58′S and 30°53′S, and also at Lord Howe Island, ~600 km east of the Australian mainland (Dalton and Smith 2006; Dalton *et al*. 2010). One potential explanation is that the pathogen is already present within subtropical habitats and that higher seawater temperature affects the coral–pathogen dynamics, resulting in the expression of disease characteristics. Transmission, either via a vector or through direct contact, may then explain the general pattern of disease prevalence. Such a scenario is supported by observations of different vectors spreading different types of coral disease in a range of localities. Sussman *et al*. (2003) found the marine fireworm *Hermodice caranculata* to be the vector for the coral-bleaching pathogen *Vibrio shiloi* (causative agent of bleaching of *Oculina patagonica* during summer), Dalton and Godwin (2006) noted the role of a corallivorous nudibranch (*Phestilla* sp.) in the transmission of disease to healthy *Turbinaria* corals, and Williams and Miller (2005) demonstrated disease transmission via coralliophilid molluscs. In this model, the pathogen(s) causing progressive tissue loss in subtropical corals may find refuge on or within the tissues of other organisms and only become infectious when corals are stressed or when the temperature increases (Lesser *et al*. 2007).

Effects of elevated temperature

The laboratory experiments demonstrated that the rate of tissue loss was greater at higher temperatures; this was also evident in the field experiments. Thus, the results strongly support the hypothesis that seawater temperature affects the rate, and potential outcome, of tissue loss over time. Patterson *et al*. (2002) made similar observations and found that the rate of tissue loss caused by white pox was correlated with seasonal conditions and elevated seawater temperatures. These researchers suggested that higher temperatures facilitate accelerated growth in pathogens and reduce the effectiveness of the host's immune response. Temperature stress may have multiple effects on the host–pathogen system. For example, Ward *et al*. (2007) observed an increase in aspergillosis infection in sea fan corals with increasing temperature. However, there was also an increase in the production of antifungal compounds at higher temperatures. Nevertheless, the virulence of the pathogen *Aspergillus sydowii* increased more rapidly than the host resistance at temperatures ≥30°C. These results demonstrate the dynamic nature of host–pathogen interactions when considering temperature stress, and may explain some of the patterns observed in the present study. Although disease transmission between infected and healthy coral colonies and disease virulence increased with temperature, there appeared to be a general trend for both to increase initially (within the first few days), then stabilise and even decrease over time. One possible explanation for this is that, following infection, the immune response of corals might slow disease progression through infected colonies and even halt disease advancement with time.

Seawater temperature within the SIMP is highly variable between years, between seasons, within seasons and throughout the marine park (AIMS 2007). Extreme temperature change has also been noted at SSI when, during a 60-min dive, the seawater temperature varied between 18 and 24°C (S. Dalton, pers. obs.). This extreme variation may potentially benefit infected corals. As demonstrated in the laboratory and the *in situ* experiments, disease transmission and the rate-of-spread of disease within affected colonies declined and even ceased at ≤21°C. With the large variation in temperature within the SIMP, tissue loss may be minimised when seawater temperatures fall below 21°C. This observation is supported by the seasonal pattern of

prevalence of this condition within the SIMP, where significantly more corals are affected during warmer periods compared with cooler months (Dalton and Smith 2006; Dalton *et al*. 2010).

Managing coral disease at a local scale

Mechanical removal of the advancing disease margin may provide a method to prevent the complete loss of *T. mesenterina* colonies, and other tabular coral species, affected by disease. By removing 20 mm of coral tissue adjacent to the advancing disease margin, tissue loss ceased in 80% of coral colonies. Removing the margin with a grinder enabled a clean cut in the coral colony parallel to the disease margin. Relative to other methods (e.g. a hammer and chisel), this approach decreased the amount of living coral tissue and associated skeleton being removed. Mechanical removal of the disease margin may provide managing authorities with a method that minimises the effect of disease on dominant coral; however, owing to logistics, this may only be viable at a local scale.

In conclusion, the present study has demonstrated that ASWS is an infectious disease affecting *T. mesenterina* within the SIMP, and that disease progression through infected colonies increases at higher temperatures. We hypothesise that progressive lysis of the coral tissue is caused by a bacterial pathogen or pathogens (possibly a *V. harveyi* strain) that becomes more virulent when temperatures increase. With the predicted increase in seawater temperature in the near future, coral tissue loss caused by ASWS may exceed coral growth in the SIMP, resulting in a decline in turbinarian corals, and possibly other coral species. If this occurs, it may precipitate a change in the structure of epibenthic communities on subtropical reefs along the east coast of Australia. Turbinarian corals dominate many subtropical sites (Harriott *et al*. 1994; Dalton and Smith 2006) and thus the potential effect is considerable, as are the possible flow-on effects, such as the displacement of organisms that are dependent on these coral communities for food and shelter.

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