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NOTE

Brown-band syndrome on feeding scars of the crown-of-thorn starfish *Acanthaster planci*

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Abstract Despite the growing impact of coral diseases on reef ecosystems, little is known about the role of coral predation in disease transmission. An experiment on the coral reefs of Derawan Island, Indonesia, revealed brown-band syndrome on *Acropora cytherea* coral colonies following predation by the crown-of-thorn starfish *Acanthaster planci*. To experimentally exclude predation, living coral tissue adjacent to feeding scars was enclosed using cages and monitored for 15 days. Compared with similarly caged but uninjured colonies, which showed no sign of disease or tissue loss, preyed upon colonies showed a higher incidence of the disease, coupled with further tissue mortality. This study provides preliminary evidence that *A. planci* might promote the transmission of some coral diseases.

Keywords Coral disease · *Acanthaster planci* · Predation · Ciliates · Emergent · Syndromes · Injury

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Introduction

Brown-band syndrome (BrB) was recorded for the first time in 2003 on the Great Barrier Reef (Willis et al. 2004). The disease is characterized by a brown zone of variable width at the interface between healthy coral tissue and recently exposed skeleton. The brown coloration is due to massive aggregations of ciliates, packed with zooxanthellae from the ingested coral tissue. At present, the etiology of this syndrome as well as other ciliate infections is unclear. It is unknown whether the ciliates are the primary cause of tissue loss and whether additional factors compromising host resistance and/or increasing ciliate virulence may be required to initiate the disease (Cróquer et al. 2006; Page and Willis 2008).

Several environmental factors, including elevated temperature, declining water quality, and transport of aeolian dust from Saharan Africa, have been proposed to influence the prevalence and severity of coral diseases (Hayes et al. 2001; Harvell et al. 2002; Sutherland et al. 2004; Bruno et al. 2003, 2007). However, the biological factors inducing and spreading diseases, in particular the role of coral predation, remain largely unknown. Corals are preyed upon by a wide variety of corallivores. Among these, the fireworm *Hermodice carunculata* (Sussman et al. 2003), the snail *Coralliophila abbreviata* (Williams and Miller 2005), the nudibranch *Phestilla* sp. (Dalton and Godwin 2006), and several species of butterflyfishes (Aeby 1998, Aeby and Santavy 2006) have been shown to play a role in disease transmission, either by acting as vectors and/or stressors.

The corallivorous starfish (i.e., seastar) *Acanthaster planci* has caused major damage to scleractinian communities on Indo-Pacific reefs since the early 1960s (Moran 1986). However, it has never been implicated in disease transmission. The aim of the present experiment

was to test whether coral disease could be facilitated by *A. planci* predation. Living tissue around feeding scars on colonies affected by the starfish was protected from further predation using enclosures and monitored over time for signs of tissue loss and disease. Enclosures were also placed on healthy colonies to serve as controls. Results suggest that corallivory by *A. planci* could promote the spread of BrB.

Materials and methods

The experiment was performed on a 3–6 m reef slope in Derawan Island, east Kalimantan, Indonesia (N02°17′20.0″, E118°15′46.4″) for 15 days in October 2007. The study area had a large crown-of-thorn starfish *A. planci* population. Eight *Acropora cytherea* table colonies, exhibiting fresh feeding scars inflicted by *A. planci* but no signs of disease, were tagged. The starfish typically leaves circular bite marks on affected corals (Fig. 1a). The marks are often grouped or overlapped, and their diameter is determined by the size of the starfish. Immediately following feeding, the scar is generally slightly brownish because of remains of decaying coral tissue. It rapidly turns white, and 2–3 days later progressively turns green as benthic algae colonize the newly exposed coral skeleton. These signs are in contrast with white syndrome, which is characterized by a narrow width of freshly exposed white skeleton and a regular tissue front (Willis et al. 2004).

Two cages made of 1 cm rigid plastic netting were fixed on each colony to prevent further predation by *A. planci*: one on top and one underneath (Fig. 1b and inset in b). Cages were 6 cm high by 40 × 25 cm and were anchored with steel wires. The top cage was placed over one or more feeding scars with a minimum of half of the caged area occupied by living coral tissue. The bottom cage was placed directly underneath mirroring the top cage. As controls, four *A. cytherea* colonies showing no signs of

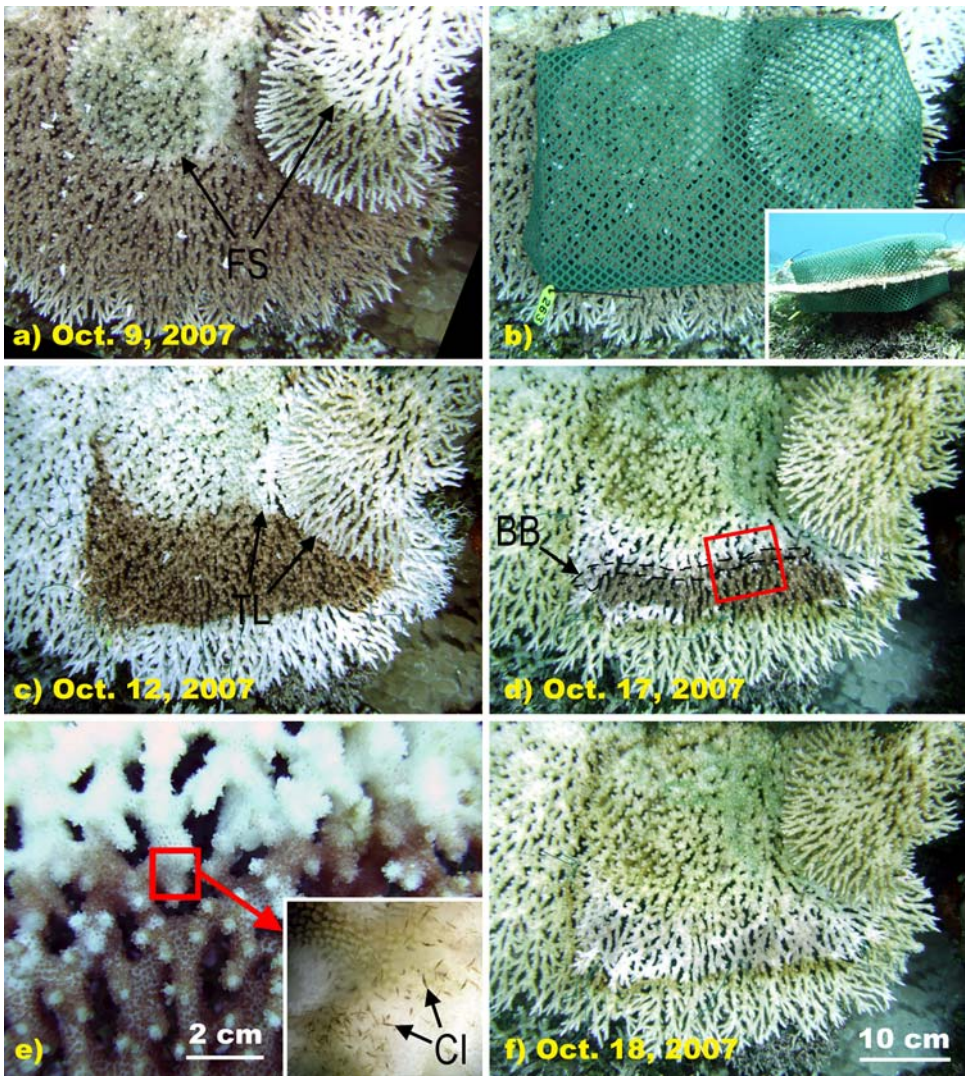

Fig. 1 In situ time series photographs of *Acropora cytherea* colony showing the spread of brown-band disease (BrB) following predation by *Acanthaster planci*. **a** Living coral tissue and feeding scars (FS) at the start of the experiment. **b** As in **a**, but following caging. Inset shows both top and bottom cages. **c** After 3 days, tissue loss (TL) occurred on the right of the caged area, but no aggregation of ciliates is visible. *A. planci* fed on all coral tissue around the top cage. **d** After 8 days, BrB associated with tissue loss is visible. The brown-band (BB) is delimited by *dashed* lines. The *red rectangle* represents the area enlarged in the next photograph. **e** Close-up view of the brown band. Inset shows a microscopic view of a sample of the band showing the ciliates (CI). Ciliates were ca. 300–400 μm in length. **f** After 9 days, only decaying coral tissue and ciliates remain

predation were caged using the same procedure. The control colonies were selected if no starfish predation scars were visible within at least 20 m from each colony, to prevent predation around the cages during the duration of the experiment. All colonies were distributed over an approximately 300 × 50 m stretch of continuous reef.

Inside the top cage, living tissue of each colony was photographed and visually examined for signs of tissue mortality and disease on days 0, 3, 8 and 15 during which the cages were cleaned. Photographs were imported into Photoshop 7. Repeated photographs were adjusted to the initial photograph using the rotate and scale functions and juxtaposed to check for tissue loss and confirm field observations.

Results and discussion

Brown-band syndrome coupled with further tissue mortality was observed on five of the eight colonies affected by *Acanthaster* predation (Fig. 1c–f). None of the remaining three colonies preyed upon by *A. planci* and none of the four control colonies exhibited signs of tissue loss or disease. There was a significant difference in BrB incidence between the predation and control treatments (chisquare test, $\chi^2 = 4.29$, d.f. $= 1$, $P < 0.05$). Microscopic examination of small, 2 cm^2 samples of visually infected tissue was performed for the first two cases of BrB and confirmed the presence of dense populations of ciliates (Fig. 1e, inset). Tissue loss was sometimes observed without the presence of BrB (Fig. 1c), in which case the symptoms were characteristic of white syndrome. However, this may be due to a decline in ciliate densities. As densities of ciliates decrease, the brown band becomes lighter and may eventually turn white (Willis et al. 2004, pers. obs.). Microscopic examinations were not performed on recently dead white patches or on healthy colonies to check for the presence of ciliates. However, all colonies exhibiting tissue loss showed signs of BrB during the monitoring period.

These results suggest a role of predation by *A. planci* in the transmission of BrB. The starfish may be a vector of the disease that transmits primary pathogen(s) during feeding. However, to date, only one corallivore, the fireworm *H. carunculata*, has been demonstrated to be a disease vector (Sussman et al. 2003). The starfish may also facilitate the spread of the disease by acting as a stressor. The feeding scars may provide an entry point for the pathogen(s) and/or increase the virulence of the ciliates. Ciliates have been found to rapidly colonize decaying tissue on small, ca. 4 cm^2 artificial injuries, but they do not necessarily progress in band-like aggregations and cause further tissue loss (Page and Willis 2008). However, *A. planci* typically produces scars reaching 100 cm^2 in size and leaves large amount of decaying coral tissue following feeding (Fig. 1a, pers. obs.). This tissue may attract a high number of opportunistic ciliates. At high densities, the latter could become a primary pathogen and cause coral mortality. Furthermore, ingested zooxanthellae remain photosynthetically competent inside the ciliates, potentially allowing them to withstand oxygen limitations within the brown band (Ulstrup et al. 2007).

Predation by *A. planci* also rapidly reduces colony size especially during massive outbreaks (Moran 1986). Such decrease in size could deplete energetic reserves below which disease resistance is compromised. The starfish fed all around the cages leaving small patches of living coral tissue (Fig. 1c). This occurred in all eight preyed upon colonies (and none of the four control colonies), but always preceded, or occurred simultaneously, with signs of BrB or tissue mortality.

These experimental results have limitations, and the relevance of this experiment to natural conditions remains to be established. For example, the cages may have excluded ciliate predators or may have affected the removal of decaying tissue by preventing access to large scavengers, or reducing water flow. Furthermore, *A. planci* may consume coral tissue so rapidly that subsequent disease may not cause significant coral mortality. Nevertheless, predation-induced disease could impair coral recovery following *Acanthaster* outbreaks by reducing the survival and regrowth of remaining coral patches. This may lead to unpredictable synergistic effects (Nyström et al. 2000) and represent a significant threat to Indo-Pacific reef corals.

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