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NOTE

Newly characterized distinct phases of the coral disease 'atramentous necrosis' on the Great Barrier Reef

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ABSTRACT: Previously undetected earlier phases of the coral disease 'atramentous necrosis' are documented and described. New observations relating to the occurrence of initial stages and progression of the disease are reported, and potential cause(s) are examined. In direct contrast to earlier published findings, temperature data indicated that occurrence of early bleached stages of atramentous necrosis is not correlated with warmer water temperatures; however, the relationship between temperature and disease prevalence is still unclear.

KEY WORDS: Coral disease · Lesions · Water temperature · Sediment · Great Barrier Reef

INTRODUCTION

Despite an increasing number of reports of disease affecting corals and other marine taxa worldwide (Sherman 2000, Ward & Lafferty 2004), and further increases predicted as a consequence of climate change (Harvell et al. 1999, 2002, 2004, Bruno et al. 2007), there has been comparatively little research focused on diseases of Indo-Pacific reef corals. Since the Indo-Pacific encompasses 91% of the world's coral reefs (Spalding & Grenfell 1997), knowledge of coral diseases in this region has considerable ecological importance. However, a lack of fundamental baseline data on the types and prevalence of diseases present, as well as their causes, has hampered our understanding of the role of disease in the region (Willis et al. 2004).

Seven coral diseases are observed to occur widely throughout the Great Barrier Reef (GBR) (reviewed in Willis et al. 2004), two of which were first described from the region. Brown band disease was first described from the central and northern sections of the GBR (Willis et al. 2004), and 'atramentous necrosis' from an inshore fringing reef at Magnetic Island in the central GBR, located directly off Townsville, Australia (Jones et al. 2004). The recognition of new coral diseases on the GBR suggests that disease may play a more significant role in the dynamics of Indo-Pacific coral reef communities than generally thought, and attests to the importance of more detailed, focused studies to further refine our understanding of these diseases.

Gross signs of atramentous necrosis are spreading black lesions, often covered by a white film that causes the lesions to appear greyish (Jones et al. 2004, Bourne 2005). The disease predominantly affects *Montipora aequituberculata* (Jones et al. 2004, Bourne 2005). Coral mortality attributable to the disease was first observed in December 2001 on Magnetic Island reefs and increased through March 2002, when sea surface temperatures peaked (monthly average >29.5°C). Signs apparently disappeared after March 2002 as water temperatures cooled, but reappeared in the subsequent summer when water temperatures again increased (Jones et al. 2004), leading the authors to

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conclude that outbreaks were strongly temperature-related. Similar disease signs have since been observed on reefs in both the northern and southern GBR (C. Page & B. Willis pers. obs.).

More recently, patches of bleached tissue and lesions of bare white skeleton devoid of tissue, occurring either alone or adjacent to each other, were observed by the authors when average water temperatures were still low (<24.5°C). These patches were primarily on colonies of foliaceous encrusting *Montipora* spp. that are widely distributed on fringing reefs surrounding Magnetic Island. These observations were in direct proximity to greyish white and black films described as characteristic of atramentous necrosis, which were also present on many of these corals. This raised questions about the links between the bleached patches, the white lesions devoid of tissue, and the grey/black lesions, as well as about the timing of lesion appearance in relation to water temperature.

To evaluate whether the bleached and recently dead patches represented early stages in the development of grey/black lesions, or an independent source of mortality, this study examined (1) the dynamics of bleached patches and recently dead white lesions on *Montipora* spp. and their relationship to the greyish black films previously described as atramentous necrosis, and (2) the occurrence of these observed abnormalities in relation to seasonal temperature patterns.

MATERIALS AND METHODS

Approximately weekly field surveys of coral assemblages on fringing reefs surrounding Magnetic Island commenced in September and were continued through November 2003 to monitor the occurrence of coral disease. Following initial observations of numerous bleached patches, recently dead white lesion areas, and blackened patches on *Montipora* spp.; a total of 50 haphazardly selected colonies of the coral *Montipora aequituberculata* were tagged using wire and plastic cattle tags at ~5 m depth. Tagging was done at Florence Bay (19° 07.32′ S, 146° 52.90′ E) and Geoffrey Bay (19° 09.44′ S, 146° 51.66′ E). A colony was defined as a physiologically distinct unit. Estimates to the nearest 5% were then made of colony surface areas that displayed the following characteristics: bleached, recently dead (i.e. white skeleton with no algal fouling), covered with a black film, or covered with a black film overlaid by a white film. Each whole colony and each patch displaying any of these gross signs of impaired health was photographed during each survey from September to November 2003. Photographs were then used to confirm and follow disease progression within colonies over time.

Samples were also collected from nearby untagged healthy and diseased *Montipora aequituberculata* colonies for further macroscopic and microscopic observations. Relevant temperature data for Florence and Geoffrey Bays were obtained from nearby AIMS data loggers located at the same average depth (~5 m) as the tagged colonies.

RESULTS

From repeated visual assessments and photographs of colonies both *in situ* and in the laboratory, 4 distinct phases in the progression of the disease were recognized: Phase 1 (P1), a small initial area of bleached but intact tissue (~1 to 2 cm in diameter), often appearing in depressions on the colony surface (Figs. 1 & 2a); Phase 2 (P2), a lesion of white skeleton devoid of tissue (Figs. 1 & 2a), appearing when the area of bleached tissue degenerated; Phase 3 (P3), a white film, confirmed microscopically to be made up of bacterial filaments covering the lesions (Fig. 1); and Phase 4 (P4), a black, sulphurous-smelling deposit, subsequently accumulating under the white film, giving the lesion a greyish black appearance (Figs. 1 & 2b). The greyish black shade depended on the amount of white film, which was typically loosely attached and easily swept off. Following the final phase, algae and entrapped sediment rapidly accumulated over the areas of black deposits, and obscured signs of disease occurrence (Fig. 2c). In some cases, the early disease phases (P1 and P2) developed in new patches adjacent to areas in the final black phase (Fig. 2d); while in other cases, disease progression appeared to halt, resulting in

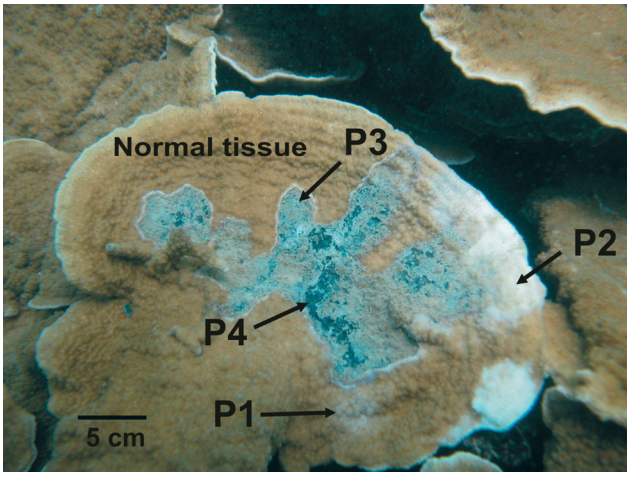

Fig. 1. *Montipora aequituberculata*. Colony displaying all 4 phases of the disease: (P1) Phase 1, initial bleaching; (P2) Phase 2, bare exposed skeleton; (P3) Phase 3, white bacterial film; and (P4) Phase 4, black sulphurous deposit

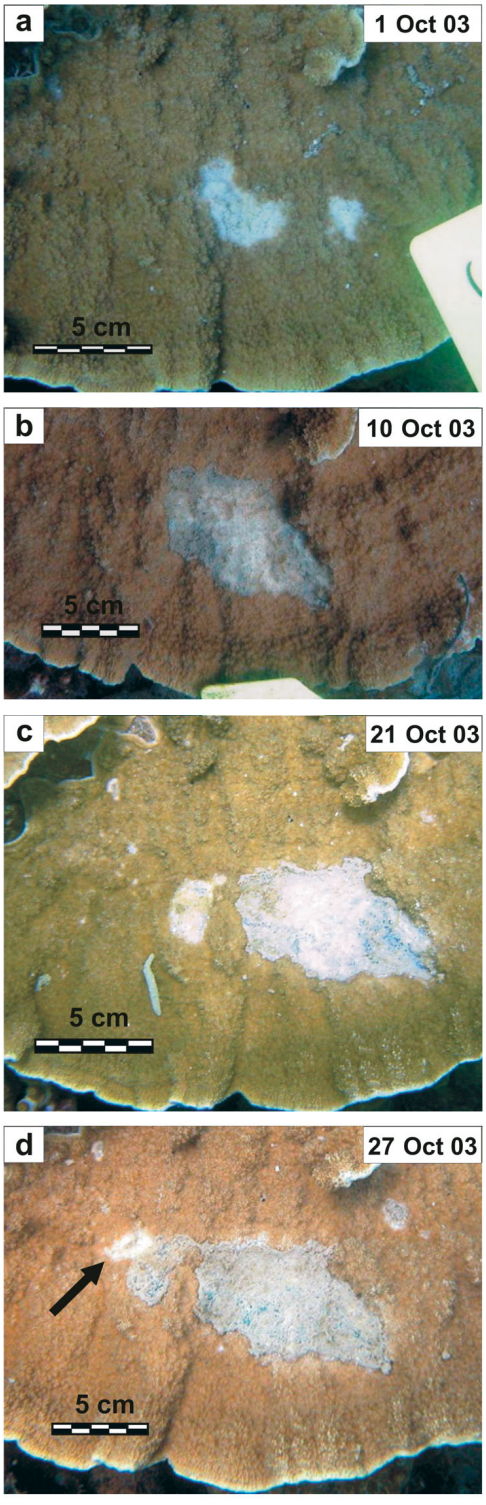

Fig. 2. *Montipora aequituberculata*. Progression of the disease *in situ* on the same colony as in Fig. 1 over a 3 wk period: (a) Phases 1 and 2, small initial infection areas; (b) Phases 2, 3, and 4, patches have joined and spread; (c) infected area has spread, older areas becoming obscured by algae and trapped sediment; and (d) a new area showing initial bleached phase of infection (black arrow)

healthy tissue directly abutting the final disease phases (Fig. 1).

At the beginning of the tagging study (October 1), only 6 of the 50 tagged colonies (12%) were categorized as healthy (i.e. showing no gross signs of bleaching or lesions). The remaining 44 colonies displayed the following signs: 21 (42%) had patchy bleaching alone (P1); 3 (6%) had areas of recently exposed white bare skeleton (P2); 10 (20%) had both bleached areas and bare skeleton (P1 to P2); and 10 (20%) exhibited all of the gross disease signs simultaneously (P1 through P4).

In subsequent surveys, 3 of the 6 initially healthy colonies developed areas of patchy bleaching and/or recently exposed skeleton. The remaining 3 did not develop additional disease signs at any point during the study (Fig. 3a). Of the 21 initial colonies with patchy bleaching alone, 10 (48%) remained bleached and showed no other disease signs throughout the observation period, while the other 11 (52%) progressed to later phases and incurred partial mortality (Fig. 3b). All 3 initial colonies with recently exposed skeleton also developed additional disease signs (Fig. 3c). In several cases, initially bleached patches were observed to undergo all 4 phases sequentially, with patch size remaining approximately the same. Alternatively, the size of lesions increased so that all 4 distinct phases of disease progression were clearly present on different parts of the same colony (Fig. 1).

Water temperatures at the survey sites at the time of first detection (September 10, 2003) were 23.7°C (Florence Bay) and 23.6°C (Geoffrey Bay). AIMS logger records show that the average temperature for the month of September was 24.36°C, with a range of 22.61 to 26.29°C. Temperatures then increased gradually as the austral summer approached (October: average 26.50°C, range 25.19 to 27.24°C; November: average 27.26°C, range 25.47 to 28.78°C).

DISCUSSION

These results provide direct evidence of 2 previously undescribed early phases of the coral disease atramentous necrosis (Jones et al. 2004). As the first description of this disease occurred during a widespread bleaching event when lesions were recorded on colonies suffering whole colony bleaching (Jones et al. 2004), identification of the initial, bleached areas that are precursors to tissue loss associated with disease progression was potentially hindered. Thus, no connection between focal bleached areas and subsequent disease progression was made. Results now show direct links between the various disease phases, and indicate that

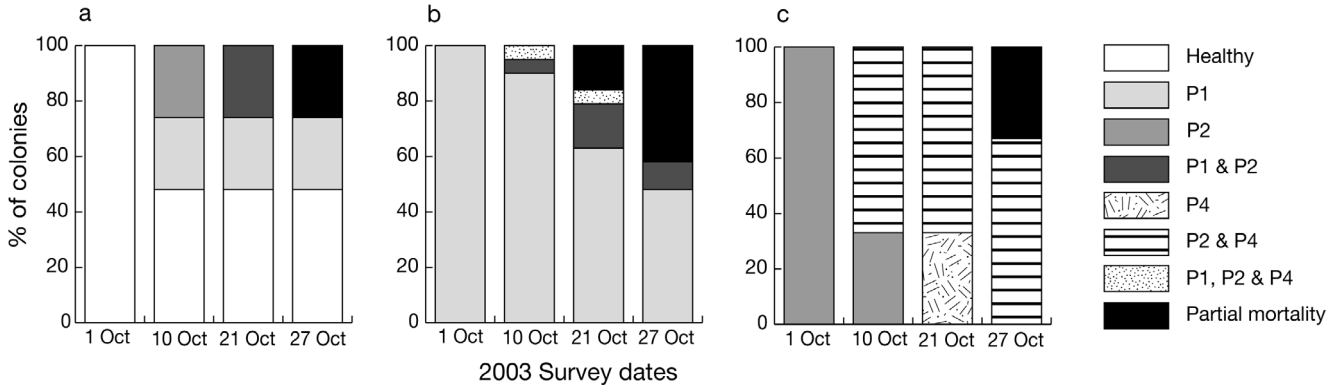

Fig. 3. *Montipora aequituberculata*. Summaries of the fate of (a) healthy colonies, (b) colonies with patchy bleaching, and (c) colonies with recently exposed skeleton. See Fig. 1 for description of phases

the black film originally described as atramentous necrosis represents only the final phases (P3 and P4).

Jones et al. (2004) suggested that the microbial community associated with these blackened lesions was specific only to degrading coral tissues associated with atramentous necrosis. Bourne (2005), however, states that the microbial communities analysed in his study, as well as those in the study by Jones et al. (2004), were more likely to be opportunistic secondary microbial communities, which may not contain the causative agent of this disease. Therefore, it is likely that the final P3 and P4 phases described herein represent secondary infections by opportunistic microbial communities, potentially associated with dying coral tissue irrespective of the initial cause of death. Our observations demonstrated that the white and black films described by Jones et al. (2004) and Bourne (2005) developed on exposed skeleton only after bleaching and tissue mortality had occurred, confirming the conclusion that these films are secondary in nature. The uniqueness of these films to this disease is therefore questionable, as is their usefulness in disease characterization (Work & Aeby 2006).

As mentioned previously, atramentous necrosis was first reported on Magnetic Island reefs in the austral summer, specifically in late December 2001 (Jones et al. 2004) and early January 2002 (Bourne 2005), when sea water temperature averages were above 29.5°C (Jones et al. 2004). Temperature anomalies (maximum daily seawater temperatures >31.5°C) and a thermal mass bleaching event occurred in early January 2002, leading Jones et al. (2004) to suggest that temperature was a key variable, with the disease only occurring during periods of elevated summer temperatures.

In contrast, our results show that the occurrence of the disease was not associated with either mass bleaching or elevated summer water temperatures, as initial disease detection (early September) and phase progression (September to November) occurred when water temperatures were still relatively low. In addition, later follow-up surveys at the same sites throughout the austral winter (May average temperature ~23.85°C; June average temperature ~22.73°C; July average temperature ~21.74°C) found that all 4 disease phases were still present, indicating that thermal stress is not a prerequisite for disease development. However, it is possible that elevated summer temperatures may increase the prevalence and/or severity of disease cases, resulting in epizootics such as the one described by Jones et al. (2004).

High loads of fine sediment are frequently suspended in the shallow inshore waters of Cleveland Bay during windy days, storm events, or when the bay undergoes dredging for shipping access. This fine sediment often settles on Magnetic Island reefs during periods of calm weather, and can result in smothering and tissue mortality of corals if the sediment is not resuspended during rough weather or removed by the coral itself (Roy & Smith 1971, Rogers 1983, Stafford-Smith 1993). Foliaceous *Montipora* spp. are poor at removing sediment from their surfaces and may experience extensive bleaching after 6 d (Stafford-Smith 1993).

Small bleached patches on tagged corals were often observed under and around sediment mats and in concave surfaces of the colony where sediment naturally accumulates. However, bleached patches that progressed to later phases of the disease were observed on vertical as well as on concave colony surfaces, and were not noted to be associated with sediment accumulation. In addition, the coral pathogen *Vibrio coralliilyticus* has recently been implicated as a causative agent in the initial bleaching and subsequent tissue degeneration process of atramentous necrosis (Sussman et al. 2008). The pathogen may be associated with the sediment and act as a reservoir, or enhance pene-

tration of the coral tissue when deposited on the coral, though further studies are required.

Studies by Hodgson (1990) revealed that microbes may play a role in damage caused by sediment, and Jokiel (2004, his Fig. 23.2) identified black deposits adjacent to sediment accumulations on *Montipora* spp. as microbial in origin. However, the role that sediment plays in triggering the initial phase of this disease is currently unknown, and black deposits are frequently observed on dead areas of coral smothered by sediment, indicating that care should be taken not to record such instances as disease occurrence. Similar black deposits reported from diseased corals off the east coast of Africa (McClanahan et al. 2004) also suggest that black deposits used to characterize atramentous necrosis (Jones et al. 2004) are not unique to this disease.

The evidence that the final 'black' phase of the disease known as atramentous necrosis is not specifically diagnostic suggests that a new name should be developed based on the earlier phases of the disease. In fact, the initial disease stages of bleached areas followed by acute signs of tissue loss leaving bare white skeleton (P1 and P2), align more closely with descriptions of White Syndrome coral afflictions (Willis et al. 2004, Sussman et al. 2008). However, given that standard histological characteristics of tissue necrosis associated with atramentous necrosis have not yet been properly identified, and to avoid confusing the issue further, we propose that the name atramentous necrosis continue to be applied to the full range of phases of this disease until its etiology is thoroughly understood and a more accurate name can be applied (as per recommendations in Work & Aeby 2006). In addition, because there may be a variety of causes of tissue mortality that lead to the development of blackened lesions, it is important not to identify atramentous necrosis occurrence on a coral unless there is clear evidence that a bleached patch leads to tissue degeneration and subsequent mortality. Our study also highlights the need for frequent and repeated observations of diseased corals to enable detection of disease phase succession and seasonality of diseases on reefs.

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