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Outbreak of the coral disease, *Montipora* White Syndrome in Maui, Hawai‘i

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Abstract. In July 2008, an outbreak of the tissue loss disease *Montipora* White Syndrome (MWS) was discovered in the ‘Āhihi Kīna‘u Natural Area Reserve on the southern shore of Maui, Hawai‘i. The outbreak occurred in a small semi-enclosed near shore pond that is dominated by the affected coral, *Montipora capitata.* Disease prevalence and coral cover were quantified periodically from September 2008 to March 2011. MWS prevalence ranged from 9.5% in 2008 to 1.8% ±0.2% in 2011. Diseased colonies observed over time showed a mean tissue loss of 40.8% ± 10.6% per colony per year. The mean benthic cover of the species of coral affected *M. capitata* declined from 48.5% ± 20.9% to 27.5% ± 1.5% within two years representing a total reduction in coral cover of 43.3% ± 14.8%. This is the highest reported MWS prevalence and the first report of a significant reduction in coral cover associated with a disease outbreak in Hawai‘i.

Key words: Maui, Coral disease, *Montipora capitata*, *Montipora* white syndrome.

Introduction

Reports of coral disease have increased globally over the past 3 decades (Richardson 1998; Ward and Lafferty 2004). While levels of disease in Hawai‘i have been low relative to the Caribbean, there have been several recent reports of outbreaks of coral disease throughout the Hawaiian Archipelago. An outbreak of *Acropora* white syndrome occurred in 2003 on reefs within the Papahānaumokuākea Marine National Monument (Aeby 2006) and an outbreak of *Montipora* White Syndrome was investigated in 2006 in Kāne‘ohe Bay, O‘ahu (Aeby et al. 2010).

Recently, another disease outbreak was observed in the ‘Āhihi Kīna‘u Natural Area Reserve (NAR) located on the southwestern tip of Maui, Hawai‘i. Extraction of marine resources has been prohibited within the reserve boundary for more than 30 years (DOFAW 2008), and the reserve is located on Maui’s most recent lava flow with little development in the upslope and adjacent areas resulting in relatively low input of land based pollutants. While anthropogenic stress due to extractive use and development is low, the area was frequented by hundreds of visitors daily prior to a recent closure. Over the past decade, the NAR became a popular ecotourism destination with increasing numbers of visitors and associated activities such as kayaking, wading, and snorkeling within the reserve (HTA 2003; DOFAW 2008; Rodgers and Jokiel 2008). Potential negative impacts from visitor use such as trampling of marine and terrestrial organisms, damage to archaeological sites, and litter and human waste accumulation due to the lack of restroom facilities led to the need for further regulatory measures. In this case, a two-year closure of much of the coastal area of the reserve was enacted in August 2008 in addition to the longer-term closure to extractive uses. The closure was later extended an additional two years. We report here on an outbreak of the coral disease *Montipora* White Syndrome (MWS), first observed during surveys as part of a program monitoring the NAR before and after the most recent closure (Stender et al. 2010).

Material and Methods

Site Description

The outbreak occurred in a small pool, approximately 38 m in maximum diameter. The area is semi to fully enclosed, depending on tides, by a naturally formed basalt wall sheltering it from wave-action. The sheltered nature of the enclosure allowed the growth of fragile plating and branching forms of *Montipora capitata* at high benthic coverage. High coral coverage and rugose growth forms resulted in high

spatial complexity, providing nursery habitat for parrotfishes (Stender et al. 2010).

Benthic Cover

Benthic cover data were collected in September 2008 and January 2010 using 0.5 m^2 adjacent, non-overlapping photo-quadrats along two 25 m transects. Transects were located along the east wall of the pond and at the entrance along the western edge of the pond (Fig. 1). Both transects were approximately 1.5 m in depth. Point-counts were conducted using PhotoGrid software (Bird 2001). Fifty randomly selected points were superimposed on each photograph, and the substratum directly beneath the center of the point was recorded. Data were analyzed to determine the percentage of surveyed benthos covered by coral, algae, and bare substratum. In July 2010 and March 2011, benthic cover data were collected using the point intercept methodology, in which the benthos directly under the transect line was recorded every 0.25 m.

Figure 1: *Montipora* pond in the ʻĀhihi Kīnaʻu Natural Area Reserve System (NARS) Maui, Hawaiʻi. The pond is ~38m in maximum diameter with two 25m transects located on the east and west edges of the pond.

Disease Prevalence

Disease data were collected along the western transect in September 2008 and January 2010 and along both transects in July 2010 and March 2011. All coral colonies within each 25 x 2 m belt transect were counted. Species and maximum diameter (cm) were recorded for all colonies. Lesion type and description were recorded for each colony exhibiting disease. Disease prevalence and the proportion of diseased vs. healthy colonies were calculated by dividing the density of diseased colonies by the density of all colonies surveyed.

Rate of Mortality

Eight colonies of *M. capitata* exhibiting signs of MWS were marked and revisited every two months from March 2010 to March 2011. Colonies were evenly distributed around the perimeter of the pond. They were selected based on presence of disease signs, and ease of assessment via photo documentation. Corals were assessed *in situ* for percentage of colony surface area that appeared to be healthy, dead or diseased. Digital images were taken for comparison with previous months to confirm *in situ* estimates. Rates of tissue loss were calculated as total change in percentage dead surface area over the course of the year of observations.

Results

Benthic Cover

The affected coral, *M. capitata,* showed a 37.7% decline in cover from 48.5% (SE ±20.9%) in September 2008 to 30.2% (SE ± 6.2%) in January 2010. Subsequent surveys using the point-intercept methodology showed a further decline with mean *M. capitata* cover 30% (SE±6.0%) in July 2010 and 27.5% (SE±1.5%) in March 2011. Overall, coral cover declined by 43.3% (SE ± 14.8%) in two and a half years at this site.

Disease Prevalence

Prevalence of MWS within ʻĀhihi Kīnaʻu was initially found to be 9.5% (July 2008) and remained high for two years with subsequent re-surveys showing 8.7% in January 2010 and 8.4% (SE±3.5%) in July 2010. By March of 2011, MWS prevalence had decreased to 1.8% (SE±0.2%).

Rate of Mortality

MWS was found to be a chronic progressive disease (Fig. 2), with individually marked colonies exhibiting an average rate of tissue loss of 40.8% (SE ± 10.6%) after one year.

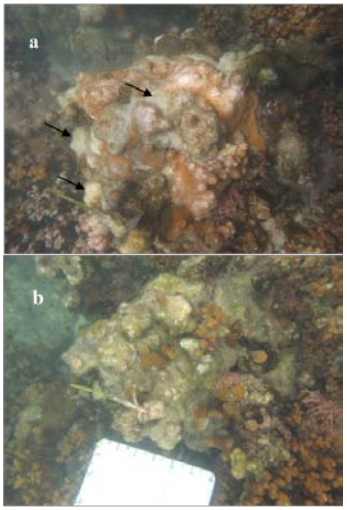

Figure 2: **a)** *Montipora capitata* colony, located in the ʻĀhihi Kīnaʻu NAR, affected by MWS in March 2010. Arrows indicate recent tissue loss from disease. **b)** The same colony shown with significant mortality from disease in Sept. 2010. Slate measures approximately 30 cm in length with tick marks every 5 cm.

Period of infection on colonies was variable, ranging from colonies that were chronically infected throughout the year to colonies that appeared disease free (no evidence of recent tissue loss), with 12.5% of colonies showing signs of recovery.

Discussion

All recorded values, including the March 2011 value, represent the highest prevalence of MWS within the Hawaiian archipelago. Prior to this observation, Kāneʻohe Bay, Oʻahu had the highest reported prevalence of MWS with a mean of 0.23% (SE ± 0.09%) (Aeby et al. 2010). Observed rates of mortality were consistent with Aeby et al. (2010) who found that MWS-affected colonies lost an average of 36.7% of their tissues, and after two years 32% of the colonies showed signs of recovery.

The dramatic loss of coral cover associated with the progression of this disease is the first report of a significant loss of coral cover associated with coral disease in Hawaiʻi. In March 2011, observations of the area showed a loss of structural integrity in addition to loss of coral cover. Previously intact coral skeletons were reduced to rubble and the fine calcareous silt usually found in the center of the pond was observed covering live corals on the reef flat of the pond. An 11 March 2011 tsunami, which affected that area, may have contributed to the breakage of already weakened coral skeletons, re-suspension of sediment, and presence of terrestrial vegetation in the pond. There were no obvious incidents of breakage of live coral in the pond. Such dramatic changes, which reduced the rugosity and structure of the reef could potentially cause changes in reef fish communities and the loss of a nursery area for parrotfishes, which are important herbivores in Hawaii.

Prevalence of MWS in the pond may have been higher than that observed in Kaneohe Bay due to the smaller spatial scale of the coral population and the dominance of the affected species. Many of the same attributes contributing to the higher than normal levels of disease in Kaneohe Bay may be magnified in the pond. For example, a basic premise of epidemiology is that increased host abundance enhances introduction and maintenance of infectious disease within a population (Anderson and May 1979; Antonovics et al. 1995; Mitchell et al. 2002). Coral disease is no exception with numerous studies finding a relationship between disease prevalence and host abundance (Bruno et al. 2007; Myers and Raymundo 2009; Haapkyla et al. 2009; Aeby et al. 2010; Williams et al. 2010). MWS is transmitted via direct contact (Aeby et al. 2010) and since *M. capitata* was the dominant coral within the site, this high host abundance would have enhanced disease transmission and thus contributed to the unusually high prevalence and persistence of the disease. In contrast, in Kaneohe Bay *M. capitata* is co-dominant with *Porites compressa,* which is not affected by this disease and so disease transmission is less successful (Aeby et al. 2010). Stressful environmental conditions have also been implicated as contributing to increased coral disease (Harvell et al. 1999, 2002, 2007). Although the site had limited anthropogenic pollution, the small and enclosed nature of the pond results in reduced water motion, sedimentation, and temperature fluctuations, which may have contributed to the disease outbreak. Low levels of water motion also allow fine calcareous sediment to settle at the center of the pond, and during times of increased water motion this sediment is easily re-suspended, greatly increasing turbidity. That such a dramatic decline in coral abundance associated with coral disease occurred in a remote and highly protected area suggests causative or synergistic factors outside the land-based pollutants traditionally associated with coral disease. The rapid disease spread and subsequent reef decline observed points toward the need for a more rapid response, including investigations into potential pathogens and methods to mitigate damage.

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