Vol. 119: 189–198, 2016
doi: 10.3354/dao02996

DISEASES OF AQUATIC ORGANISMS
Dis Aquat Org

Published May 26

Emerging coral diseases in Kāneʻohe Bay, Oʻahu, Hawaiʻi (USA): two major disease outbreaks of acute *Montipora* white syndrome

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ABSTRACT: In March 2010 and January 2012, we documented 2 widespread and severe coral disease outbreaks on reefs throughout Kāneʻohe Bay, Hawaiʻi (USA). The disease, acute *Montipora* white syndrome (aMWS), manifested as acute and progressive tissue loss on the common reef coral *M. capitata*. Rapid visual surveys in 2010 revealed 338 aMWS-affected *M. capitata* colonies with a disease abundance of (mean ± SE) 0.02 ± 0.01 affected colonies per m of reef surveyed. In 2012, disease abundance was significantly higher (1232 aMWS-affected colonies) with 0.06 ± 0.02 affected colonies m^{-1}. Prior surveys found few acute tissue loss lesions in *M. capitata* in Kāneʻohe Bay; thus, the high number of infected colonies found during these outbreaks would classify this as an emerging disease. Disease abundance was highest in the semi-enclosed region of south Kāneʻohe Bay, which has a history of nutrient and sediment impacts from terrestrial runoff and stream discharge. In 2010, tagged colonies showed an average tissue loss of 24% after 1 mo, and 92% of the colonies continued to lose tissue in the subsequent month but at a slower rate (chronic tissue loss). The host-specific nature of this disease (affecting only *M. capitata*) and the apparent spread of lesions between *M. capitata* colonies in the field suggest a potential transmissible agent. The synchronous appearance of affected colonies on multiple reefs across Kāneʻohe Bay suggests a common underlying factor. Both outbreaks occurred during the colder, rainy winter months, and thus it is likely that some parameter(s) associated with winter environmental conditions are linked to the emergence of disease outbreaks on these reefs.

KEY WORDS: Coral disease · Kāneʻohe Bay · *Montipora capitata* · Hawaiʻi · Disease outbreak · Emerging diseases

INTRODUCTION

Emerging diseases are defined as diseases whose geographical range, host range, or prevalence has recently increased (Daszak et al. 2000). A recent example is the outbreak of seastar wasting disease on the west coast of North America that killed millions of asteroids (Hewson et al. 2014). Climate change and human disturbance have been cited as major influences contributing to disease outbreaks in wildlife populations (Harvell et al. 1999, 2007, Daszak et al. 2000), with environmental degradation from anthropogenic activity suggested as the most important factor (Dobson & Foufopoulos 2001).

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Publisher: Inter-Research · www.int-res.com

Worldwide coral reefs are in decline and facing the combined threat of chronic local anthropogenic impacts and global climate change (Hughes et al. 2003, Pandolfi et al. 2003, Bellwood et al. 2004). Marine diseases have also played an important role and were largely responsible for the catastrophic loss of coral reefs throughout the Caribbean (Aronson & Precht 2001, Gardner et al. 2003). Hawai'i (USA) is one of the most isolated archipelagos in the Indo-Pacific (Jokiel 1987), and the main Hawaiian Islands are under increasing human pressures from overfishing (Friedlander & DeMartini 2002), episodic sewage spills, and poor water quality (Friedlander et al. 2008, Knee et al. 2008, Dailer et al. 2012). Coral diseases have been documented at low prevalence on reefs throughout the Hawaiian archipelago (Vargas-Ángel 2009, Aeby et al. 2011), but recently disease outbreaks have occurred, predominantly in the common reef coral *Montipora capitata* Dana, 1846 (Aeby et al. 2010, 2015, Ross et al. 2012).

Fig. 1. *Montipora capitata* colony exhibiting acute tissue loss indicated by the white exposed coral skeleton (acute *Montipora* white syndrome). Stripes on scale bar = 1 cm

Kāne'ohe Bay, located on the northeastern side of O'ahu, is a complex estuarine system with a large barrier coral reef and numerous patch and fringing reefs (reviewed by Bahr et al. 2015). Coral cover is composed predominantly of large thickets of *Porites compressa* Dana, 1846 that are interspersed with colonies of *M. capitata* (Jokiel 1987, Maragos 1995). Kāne'ohe Bay, especially the semi-enclosed southern sector, has a long history of reduced water quality with large inputs of nutrients and suspended sediments from the numerous streams that feed into the bay (Cox et al. 2006, De Carlo et al. 2007). Stream discharge into Kāne'ohe Bay is characterized by extended periods of low flow with sporadic periods of intense runoff associated with rainstorms predominantly during the winter months (De Carlo et al. 2007, Ostrander et al. 2008, Drupp et al. 2011).

Numerous coral diseases occur on the reefs in Kāne'ohe Bay, including *Porites* trematodiasis (Aeby 2007), *Porites* growth anomalies (Stimson 2011), *Porites* bleaching with tissue loss (Sudek et al. 2015), and *Montipora* white syndrome (Aeby et al. 2010), among others. Comparatively low disease levels (usually <1% prevalence) have been consistently reported for most diseases (Aeby 2007, Aeby et al. 2010, Williams et al. 2010, Stimson 2011, Sudek et al. 2015). However, in March of 2010 and January of 2012, *M. capitata* colonies on the reefs in Kāne'ohe Bay experienced major outbreaks of a disease that caused rapid tissue loss (>5 cm of recently denuded white skeleton; Fig. 1). The disease was termed acute *Montipora* white syndrome (aMWS) to distinguish it from the less virulent disease (<5 cm recently denuded white skeleton) chronic *Montipora* white syndrome (cMWS), which is commonly found year-round on Kāne'ohe Bay reefs (Aeby et al. 2010). Here we report the spatial pattern and extent of the 2 outbreaks within Kāne'ohe Bay, disease abundance, duration and virulence of the disease on individual colonies, and histopathology.

MATERIALS AND METHODS

Spatial distribution within Kāne'ohe Bay

Large numbers of *Montipora capitata* colonies with acute tissue loss were initially observed on several reefs within Kāne'ohe Bay. The large geographic scale suggested by the reported coral mortalities precluded detailed coral colony counts and thus calculation of disease prevalence. Instead, we opted to document the spatial extent of this outbreak using rapid visual assessments in south, central, and north regions of Kāne'ohe Bay (Fig. 2). Three teams, each comprising 1 snorkeler and 1 boat driver, followed reef contours, with the snorkeler surveying all *M.*

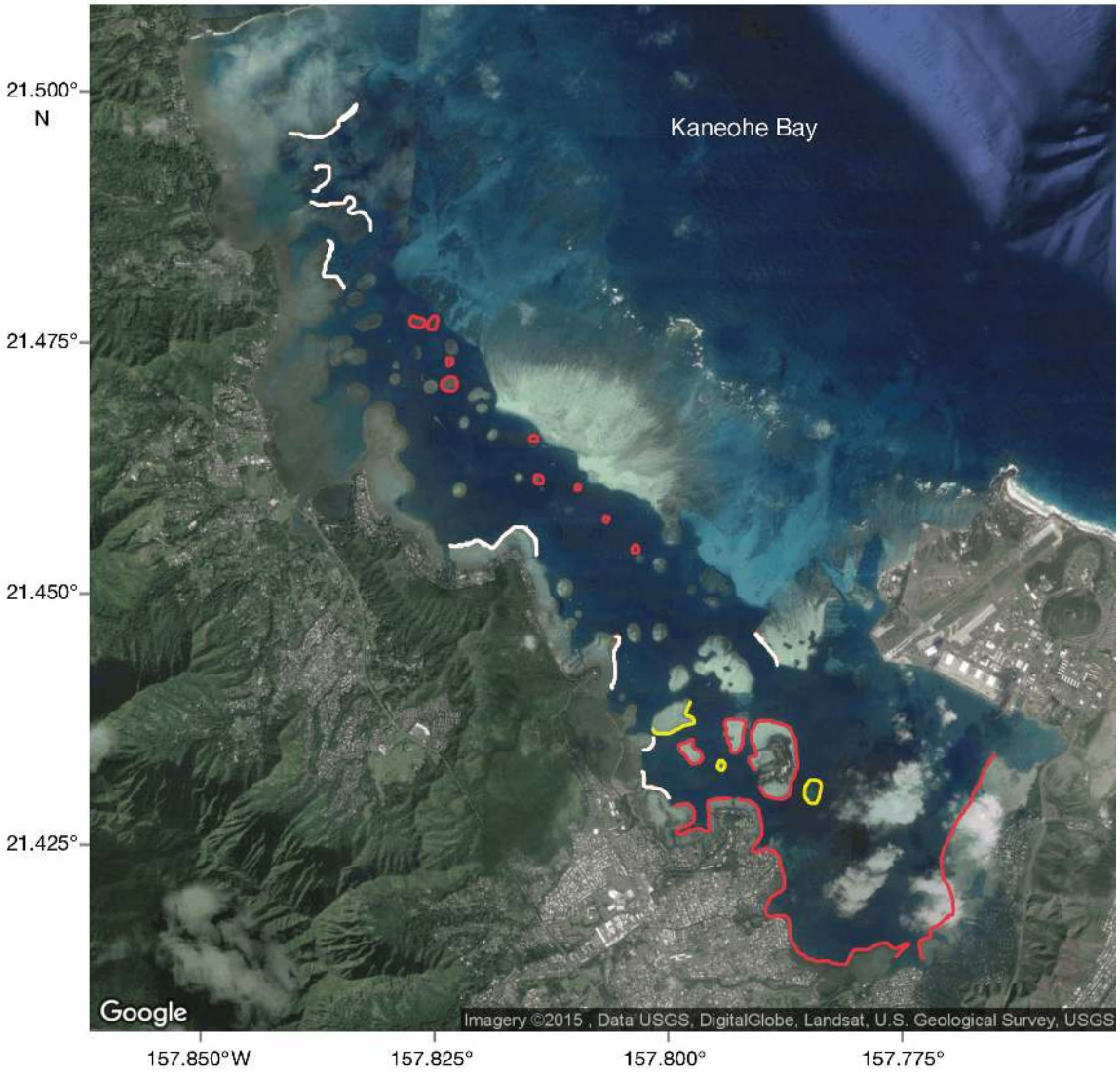

Fig. 2. Reef areas visually surveyed during the 2010 and 2012 outbreaks. Yellow lines indicate areas surveyed exclusively in 2010, white lines were areas surveyed exclusively in 2012, and red indicates reef areas surveyed during both years

capitata colonies and enumerating and photographing colonies manifesting aMWS (recent tissue loss >5 cm). Snorkelers surveyed *M. capitata* colonies from ~1 m onto the reef flat to the bottom of the reef slope (~10 m). Disease abundance was defined as the number of affected colonies per linear meter of reef surveyed. In March 2010, teams surveyed the fringing reefs within south Kāne'ohe Bay and patch reefs within the south, central, and north regions of Kāne'ohe Bay. In January 2012, additional fringing reefs in central and north Kāne'ohe Bay were added to the areas surveyed. The GPS coordinates at the start and stop of each survey were recorded, and the linear distance surveyed was estimated using Google Earth.

Duration and virulence of aMWS

In March 2010, 19 *M. capitata* colonies with aMWS on the reefs surrounding Coconut Island (southern end of Kāne'ohe Bay) were tagged and photographed, and the rate of tissue loss was monitored weekly for 4 wk. The complex 3-dimensional structure of the colonies prevented use of digital analysis to measure tissue loss. Hence, *in situ* visual estimates of the proportion of each colony that was healthy, dead, or with recent tissue loss were recorded for each of the 4 weeks. The colonies were resurveyed in May 2010 and the disease state noted (no disease, chronic tissue loss [<5 cm recent tissue loss], or acute tissue loss [>5 cm recent tissue loss]).

No individual colonies were tagged during the 2012 outbreak.

Histopathology

In March 2010, paired lesion and normal samples from 15 colonies of *M. capitata* manifesting varying degrees of acute tissue loss were collected, fixed in zinc formaldehyde, and processed for histopathology for subsequent microscopic examination as described previously (Work et al. 2012).

Environmental data

We examined available environmental data for Kāneʻohe (rainfall, sea surface temperatures, and stream flow) to determine whether any anomalous weather conditions preceded the outbreaks. Sea surface temperatures for the years 2006 to 2012 were obtained from the weather station at the Hawaiʻi Institute of Marine Biology (https://www.hawaii.edu/himb/). For rainfall, the standard precipitation index (SPI) was calculated as described by Guttman (1999). This index normalizes rainfall to its distribution (gamma) and normalizes it to a mean of 0 and SD of 1 such that large negative values (<2) indicate severe drought whilst large positive values (>2) indicate unusually wet years. Rainfall data from January 1986 through December 2015 were obtained from the National Weather Service (www.ncdc.noaa.gov/cdo-web/datatools/selectlocation) for Stn KĀNEʻOHE 838.1 HI. SPI was calculated on a month–year basis, plotted as a grid, and values were visualized. For stream flow, data from January 1986 through December 2015 were obtained from the USGS stream gauge at Kāneʻohe Bay (no. 16275000 Heʻeia Stream at Haʻik Valley) available at http://maps.waterdata.usgs.gov/mapper/index.html. The mean annual flow and monthly percent of mean annual flow were summarized for rainfall and stream flow data.

Data analysis

Data did not have a normal distribution, even with transformation, so non-parametric statistics were used. Spatial distribution of disease (no. of aMWS colonies m^{-1} reef surveyed) was examined among regions (south, central, north) using a Kruskal-Wallis 1-way analysis of variance for each year (2010, 2012) followed by a Wilcoxon pairwise comparison among regions. A Wilcoxon 2-group test examined differences in disease levels (no. of aMWS colonies m^{-1} reef surveyed) between years and reef types (fringing versus patch). All statistics were run on JMP® version 11.2.0.

RESULTS

Spatial distribution within Kāneʻohe Bay

In 2010, 15.4 km of reefs (fringing, patch, and Coconut Island fringing) were surveyed (Table 1) and 338 *Montipora capitata* with aMWS were found, representing a mean abundance of 0.02 (SE ± 0.01) diseased colonies per linear meter of reef surveyed. Affected colonies were on fringing and patch reefs throughout the bay and occurred from the reef flat down to the lower reef slope. South and north regions of Kāneʻohe Bay had a significantly higher abundance of aMWS compared to the central region (Kruskal-Wallis, df = 2, $\chi^2 = 7.97$, p = 0.019; Table 2, Fig. 3). No significant differences were found in disease abundance on fringing (n = 4) versus patch (n = 6) reefs in south Kāneʻohe Bay (Wilcoxon 2-group test, $Z = -0.21$, p = 0.83).

For the 2012 disease outbreak, we surveyed 21.4 km of reefs (Table 1) and found 1232 *M. capitata* colonies with aMWS (mean ± SE disease abundance = 0.06 ± 0.02 colonies m^{-1}) and a similar pattern of distribution on reefs as in 2010. Disease abundance was significantly higher in south Kāneʻohe Bay (1179 affected colonies) compared to the north and central bay (Kruskal-Wallis, df = 2, $\chi^2 = 9.14$, p = 0.010; Table 2, Fig 3). No significant differences were found in disease abundance in fringing (n = 7) versus patch (n = 12) reefs (Wilcoxon 2-group test, $Z = 0.55$, p = 0.58). Average disease abundance was significantly higher in 2012 compared to 2010 (Wilcoxon 2-group test, $Z = -2.03$, p = 0.043).

Table 1. Survey effort (km) in Kāneʻohe Bay (Hawaiʻi, USA) during the 2010 and 2012 disease outbreaks

Region	Reef type	2010	2012
South	Fringing	7.3	8.6
	Patch	3.5	2.4
	Coconut Island fringing	2.3	2.3
Central	Fringing	0	3
	Patch	0.7	0.7
North	Fringing	0	2.8
	Patch	1.6	1.6
Total distance (km)		15.4	21.4

Table 2. Wilcoxon pairwise comparisons of regional disease abundance (number of corals with acute *Montipora* white syndrome m^{-1} linear reef surveyed) in disease outbreaks in Kāne'ohe Bay (Hawai'i, USA) in 2010 and 2012

	Mean difference	*Z*	p
2010			
South vs. central	5.29	2.4	0.02
North vs. central	4.28	2.5	0.01
South vs. north	−0.54	−0.2	0.82
2012			
South vs. central	4.81	2.1	0.03
North vs. central	−2.01	−1	0.3
South vs. north	5.69	2.6	0.01

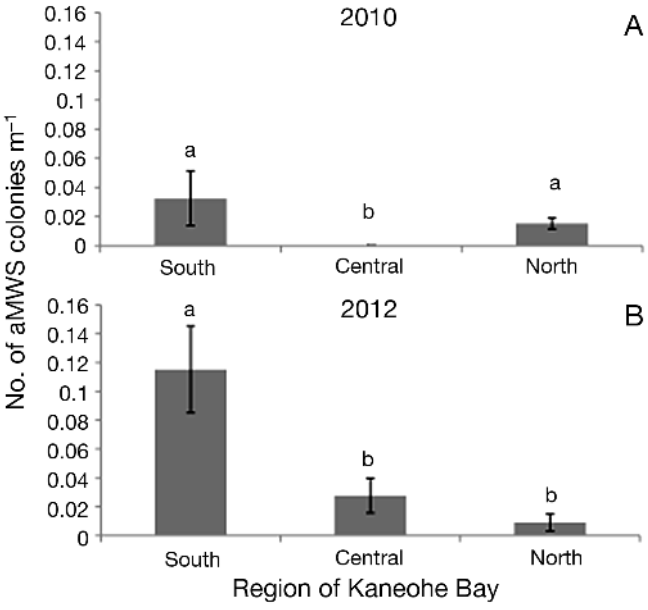

Fig. 3. Differences in disease abundance among the regions of Kāne'ohe Bay (Hawai'i, USA) during the 2010 and 2012 acute *Montipora* white syndrome (aMWS) disease outbreaks. (A) Data collected from the rapid surveys during the 2010 outbreak and (B) data collected during the 2012 outbreak. Data represent the mean and standard error. Different letters indicate significant differences among regions within each year (Wilcoxon pairwise comparisons, $p < 0.05$)

Duration and virulence of aMWS

The initial severity of tissue loss on the 19 tagged colonies ranged from 2% (disease just starting) to 95% (disease well established) (mean = 19% of the colony with new lesions). After 1 mo, additional tissue loss on individual colonies ranged from 3 to 96% (mean = 23.5%). At the end of the 4 wk observation period, 17 of the 19 colonies (90%) continued to have active tissue loss, with 12 colonies (71%) having acute tissue loss (>5 cm recent tissue loss) and the remainder (29%) converting to cMWS (<5 cm recent tissue loss). Of 13 colonies relocated in May 2010, 12 colonies had cMWS (92%) and 1 had no active lesions.

Gross lesion description and histopathology

Gross lesions manifested as different-sized areas of acute tissue loss, revealing bare white intact skeleton, with variable borders. Frequently, tissue adjacent to the bare skeleton had a lighter color ranging from approximately 1 to 10 cm (Fig. 4), which under dissection microscopy appeared as degraded coral tissue. In other cases, tissues bordering the areas of tissue loss were also lighter in color, but the margin was narrow (<1 cm), and the tissue was intact but thinning (Fig. 5A). On microscopy, the predominant lesion, for all but 2 corals, was segmental ablation of surface body wall with epidermal regeneration and occasional necrosis of mesenterial filaments and gastrodermis (Fig. 5B–D). Microbes (ciliates) were associated with lesions in 2 cases; otherwise, no morphological evidence of infectious agents was seen.

Fig. 4. Acute *Montipora* white syndrome showing tissue loss revealing intact bare white skeleton (black arrow), with variably sized border of pale tissue (white arrow)

Environmental data

The outbreaks occurred during the winter months with peaks of rainfall and stream outflow (see Fig. S1 in the Supplement, available at www.int-res.com/articles/suppl/d119p189_supp.pdf) and colder sea surface temperatures (Fig. S2). The standard precipitation index suggested that 2010 was a drier year; however, this was not the case for 2012 (Fig. S3).

DISCUSSION

Spatial extent of the disease outbreak

This study reports on 2 of the most severe and spatially widespread coral disease outbreaks reported from the Hawaiian archipelago. Other coral disease outbreaks have occurred in Hawai'i but not in the spatial extent or the number of cases found in the current study (Aeby 2005, Aeby et al. 2010, 2015, Ross et al. 2012). The large numbers of colonies affected (338 in 2010 and 1232 in 2012) and the spatial extent (spanning reefs across ~7.5 km from south to north Kāne'ohe Bay) illustrate the scale and severity of these outbreaks. Prior surveys in Kāne'ohe Bay found few cases of aMWS in *Montipora capitata* (Aeby et al. 2010). The high number of infected colonies found during these 2 outbreaks would classify this as an emerging coral disease.

Coral disease surveys and monitoring have been ongoing in Hawai'i since 2003 (Aeby 2006, Aeby et al. 2011, Vargas-Ángel & Wheeler 2008), and we are beginning to see a pattern of increased frequency of disease outbreaks. The first documented coral disease outbreak in Hawai'i, acute *Acropora*

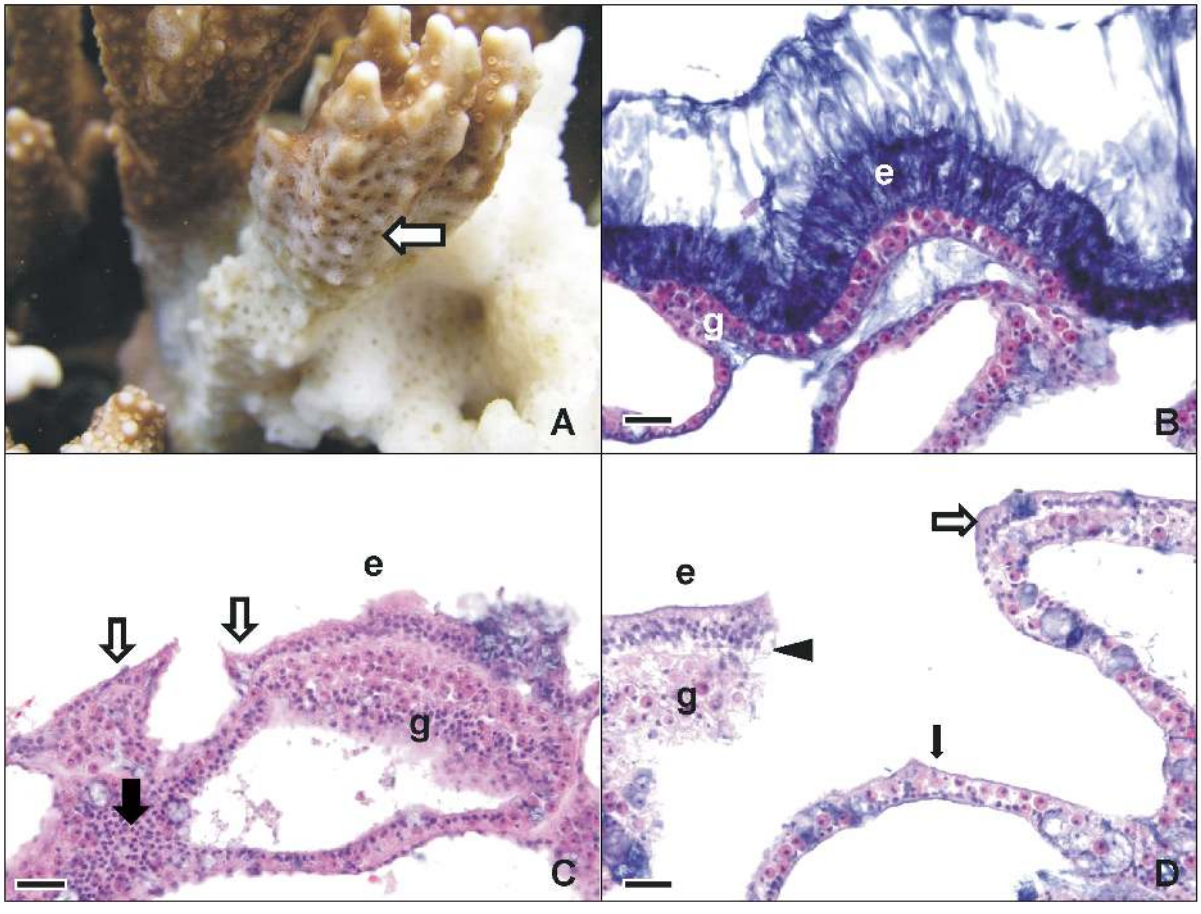

Fig. 5. (A) Close up photo of acute *Montipora* white syndrome revealing tissue thinning (arrow) progressing to bare intact white skeleton at the base. (B) Histology of normal surface body wall of *M. capitata* showing tall columnar epithelium (e) and gastodermis (g) with zooxanthellae progressing to tissue thinning. (C) Coral with tissue loss from 2010; note regenerating epidermis (white arrows) and proliferation of underlying gastrodermal cells (black arrow). (D) Coral with tissue loss from 2010; note ablation of surface body wall (arrowhead) revealing basal body wall (black arrow) and regenerating epidermis (white arrow). Scale bars for all photomicrographs = 20 µm

white syndrome, was reported in 2003 from French Frigate Shoals within the northwestern Hawaiian Islands (Aeby 2005). Six years passed before the next outbreak occurred, when in 2009 an outbreak of chronic tissue loss disease (*Montipora* white syndrome) was reported from Maui (Ross et al. 2012). An outbreak of black band disease was found on Kauai in 2011 (Aeby et al. 2015), and now 2 disease outbreaks have occurred in Kāneʻohe Bay, Oʻahu (present study). The Caribbean offers an instructive cautionary contrast. There the emergence of coral disease followed a similar pattern, with the first report of a disease outbreak that produced significant coral mortality occurring in 1975 in Florida (Dustan 1977). Since that time, numerous disease outbreaks have occurred contributing to the significant loss of live coral, including the once dominant acroporids, which have suffered an estimated 95% decline in abundance throughout their range (Rogers 1985, Bythell & Sheppard 1993, Patterson et al. 2002, Sutherland et al. 2004, Weil & Rogers 2011). Overall, reefs in the Caribbean have experienced a dramatic decline in living coral cover with as much as 80% lost (Gardner et al. 2003, Wilkinson 2008, Miller & Richardson 2014). Disease continues to plague Caribbean reefs, with an outbreak of acute tissue loss disease recently reported from the Dry Tortugas National Park in Florida (Brandt et al. 2012).

Disease virulence, spread, and etiology

We found aMWS to be a virulent and progressive disease, with tagged corals in 2010 losing an average of 24% of their tissue after 4 wk. In contrast, *M. capitata* colonies affected with cMWS in Kāneʻohe Bay lose an average of 3% of their tissue per month (Aeby et al. 2010). In general, corals are slow-growing animals, with *M. capitata* growing less than 2.5 cm yr^{-1} in Hawaiʻi (Jokiel 1978), and so tissue loss from disease may require substantial recovery time.

Numerous cases of apparent disease transmission between *M. capitata* colonies were observed in the field. New infections on colonies were occurring at the direct point of contact with an affected colony, suggesting that aMWS may be infectious. The specific etiology of this disease is unknown, but under experimental conditions, pathogenic bacteria can cause gross lesions resembling cMWS (Ushijima et al. 2012) and aMWS (Ushijima et al. 2014) in *M. capitata* in Hawaiʻi. However, similar disease signs in corals can have multiple underlying etiologies, and inferring causation based on gross lesions alone is problematic as pointed out elsewhere (Bourne et al. 2009, Work et al. 2012). Indeed, we found that lesions on corals varied in size and shape, with the lesion borders varying in width, color, and tissue condition. Based on qualitative field observations, lesion morphology appeared to vary depending on the coral colony size and morphology, stage of the disease, and when during the outbreaks the lesion first appeared. Initially in the outbreaks, we found new lesions commonly bordered by a wide grey area of degraded tissue, whereas near the end of the events the lesion borders tended to be narrower areas of bleached intact tissue.

Microscopically, the most common finding in affected tissue was loss of the surface body wall and wound repair (Work & Aeby 2010) with occasional necrosis of the remaining tissues (gastroderm, mesenterial filaments). No morphologic evidence of infectious agents was observed except for 2 cases, which had ciliates. These findings are consistent with numerous other histological studies of tissue loss in corals where extracellular bacteria (commensal or pathogenic) are not observed and no compelling morphologic evidence is found incriminating bacteria as a cause of tissue loss using tools like histopathology (Work et al. 2012, 2014, 2016). Our histological findings rule out infections from cyanobacteria, metazoan or unicellular parasites, algae, fungi, or parasitic corals (Work et al. 2012, 2014, 2016). However, we cannot confirm or refute the involvement of other pathogenic microbes not visible on light microscopy. Histology also provides a lesion description at the cellular level that is critical in developing a case history of the disease, which is lacking in many other studies of coral disease (Work & Meteyer 2014).

In 2010, several months after the start of the first outbreak, 12 of 13 tagged colonies continued to lose tissue, but at a slower rate, indicating that they had progressed to cMWS. Prior studies by Work et al. (2012) found that lesions on *M. capitata* can shift between chronic and acute rates of tissue loss, and differ in their respective underlying host responses and associated agents. *M. capitata* with cMWS occurs year-round throughout Kāneʻohe Bay, with no evidence of seasonality (Aeby et al. 2010). In contrast, aMWS appeared suddenly on the reefs during winter months and then subsided. The cause(s) of these outbreaks remain unknown, but the differences in temporal pattern and rate of tissue loss between cMWS and aMWS suggest that disease processes differ.

Interspecific disease susceptibility

Porites compressa is numerically co-dominant with *M. capitata* in Kāneʻohe Bay (Jokiel 1987); however, no tissue loss lesions were observed on *P. compressa* during either outbreak, even when there was direct contact with infected *M. capitata* colonies. Differential disease susceptibility among coral genera has been found in a number of studies (Willis et al. 2004, Gochfeld et al. 2006, Vargas-Ángel 2009). In addition, Ushijima et al. (2014) showed that under experimental conditions, *P. compressa* was resistant to a bacterial pathogen that readily infected and caused acute tissue loss in *M. capitata*. *Montipora* spp. appear unusually susceptible to disease in Hawaiʻi. Indeed, all documented outbreaks of tissue loss disease in the main Hawaiian Islands have exclusively involved montiporids (Aeby et al. 2010, 2015, Ross et al. 2012). In contrast, *Porites* spp. in Hawaiʻi are more commonly affected by chronic diseases such as trematodiasis and growth anomalies, which produce coral morbidity but minimal mortality (Aeby 1991, Aeby et al. 2011, Stimson 2011). There is a pressing need to understand the physiological basis for these differences in host susceptibility to diseases.

Temporal and spatial patterns of affected coral

Disease outbreaks occur when conditions change, allowing new pathogens to invade or existing pathogens to flourish (Colwell 1996, Dobson & Foufopoulos 2001). The nearly synchronous appearance of affected colonies (based upon degree of algal colonization of denuded skeletons) on multiple reefs suggested a common underlying factor. Both disease outbreaks occurred during the winter months when ocean temperatures are lower and rain events and stream discharge into Kāneʻohe Bay are more common. An examination of the available ocean temperature, rainfall, and stream data for Kāneʻohe showed no anomalous environmental perturbations, but did confirm that typical winter conditions occurred during both outbreak periods. Winter months are suboptimal conditions for corals in Hawaiʻi, resulting in reduced growth (Jokiel & Coles 1977) and lower lipid reserves (Stimson 1987), perhaps leaving corals more vulnerable to disease. Interestingly, our findings of higher disease during colder periods contrast with other studies where anomalously warm temperatures have been linked to coral disease outbreaks (Heron et al. 2010, 2012). This highlights the complex nature of host–pathogen–environment dynamics.

The disease outbreaks also coincided with the months where there is maximum discharge of streams into Kāneʻohe Bay. During local winter rain events, stream discharge into Kāneʻohe Bay can be 2 to 3 orders of magnitude higher than usual (Oki 2004), leading to increased sedimentation, reduced salinity, and enrichment in inorganic nutrients (Cox et al. 2006, Hoover et al. 2006, De Carlo et al. 2007). Similarly, Haapkylä et al. (2011) found a 10-fold greater mean abundance of a tissue loss disease in montiporids on Australian reefs during the rainy summer months and concluded that rainfall and associated runoff were the main factors facilitating seasonal disease outbreaks. They also found that warm summer temperatures were positively correlated with disease abundance but only explained a small amount of the variance.

The effects of storm events would be magnified in south Kāneʻohe Bay, where the nearshore waters have a relatively long residence time, compared to the other regions of the bay (Bathen 1968, Cox et al. 2006, DeCarlo et al. 2007, Drupp et al. 2011), which could be contributing to the higher disease abundance we found in the south. However, these observations are confounded by host abundance, which is known to affect disease prevalence in most host–pathogen systems (Lafferty & Holt 2003, Poteet 2006) including corals (Bruno et al. 2007, Myers & Raymundo 2009). *Montipora* spp. abundance is highest in south Kāneʻohe Bay (Aeby et al. 2010), so this could, in part, explain higher disease in that region.

We saw no significant difference between disease abundance on the fringing reefs compared to the patch reefs in the bay, although our methods quantifying disease were somewhat limited given that our primary objective was to assess disease bay-wide. If the underlying trigger was exclusively direct stress from terrestrial runoff or stream discharge (reduced salinity, sedimentation, eutrophication), we would expect the nearshore fringing reefs to be more affected than patch reefs located farther from shore. This lack of a land to sea gradient in disease abundance suggests that a more systemic problem may also be occurring. Storm runoff not only results in direct eutrophication of nearshore waters but also creates a cascade of events leading to phytoplankton and zooplankton blooms (Cox et al. 2006). Both phytoplankton and zooplankton blooms can facilitate bacterial blooms through increased availability of organic matter or substrate for bacterial attachment (Huq et al. 1983, Tamplin et al. 1990). As an example, the association of *Vibrio cholera* with planktonic copepods is a known factor facilitating cholera out-

breaks in humans (Huq et al. 1983, Colwell 1996). More information is needed to understand the potential links between terrestrial runoff, plankton blooms, and coral disease outbreaks.

Acknowledgements. Thanks to Dr. S. Coles, R. Eismueller, F. Farrell, and K. Aeby for assistance in the field during the outbreaks. This study was supported, in part, by NSF grant OCE-0961814 awarded to G.S.A.

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Editorial responsibility: Garriet Smith, Aiken, South Carolina, USA

Submitted: October 8, 2015; Accepted: March 23, 2016
Proofs received from author(s): May 2, 2016