The prevalence of skeletal growth anomaly and other afflictions in scleractinian corals at Waiʻōpae, Hawaiʻi

M. Takabayashi[1], T.M. Gregg[1], E. Farah[1], J. Burns[1], K. Teves[1], N.H. Cody[1]

1) University of Hawaiʻi at Hilo, 200 W. Kawili St. Hilo Hawaiʻi 96720 USA

Abstract. The health states of scleractinian corals were assessed at Waiʻōpae, southeast Hawaiʻi Island, where approximately half of the tide pools belong to a Marine Life Conservation District (MLCD) while the other half lies outside the MLCD boundary. The susceptibility of corals to afflictions is species-dependant with *Porites compressa* being the most (22.5±3.9% unhealthy) and *Pavona varians* the least (1.1±0.27% unhealthy) susceptible. The proportion of unhealthy corals was: higher inside the MLCD than outside for *Montipora* and *Pocillopora*; not different between the two areas for *Porites*; and higher outside MLCD than inside for *Pavona*. Skeletal growth anomaly (SGA) was disproportionably observed in *Montipora* spp., especially *M. capitata*, 13.6 (±0.37) % of which were afflicted by this condition. There is no clear correlation between the progression of SGA (change in size or frequency) and species, location, or season. Some coral colonies are showing a level of resilience against SGA while others have succumbed to it within the Waiʻōpae population. The prevalence of coral afflictions at Waiʻōpae is unusually high compared to other sites in the Hawaiian Archipelago, but the causes are unknown. Monitoring of coral health at this site is continuing with an incorporation of assessment of correlations to water quality characteristics.

Key words: coral disease, skeletal growth anomaly, Waiʻōpae, Hawaiʻi, *Montipora*

Introduction

Prevalence and severity of coral diseases are reported to be increasing worldwide (reviewed in Harvell et al. 2007). Types of diseases or reduced health states seem to vary according to coral species and location. In many cases, the effort to positively identify the pathogen responsible for the onset of coral diseases has been met with challenges. It is generally believed that water quality exacerbates the susceptibility of corals to pathogenically or non-pathogenically compromised health. For example, moderate increases in nutrient concentrations have been shown to substantially increase the severity of coral diseases (Bruno et al. 2003). However, specific causal relationships between particular water quality characteristics and individual coral disease are poorly understood. With the mechanisms of disease progress and mortality of corals being poorly understood, it is quite plausible that disease could even enable coral survival through reproduction of disease resistant genotypes in some cases (Lesser et al. 2007). Until the effects of coral diseases are better understood it is difficult to assess the fate and threat posed to corals in waters subjected to harmful anthropogenic influences.

Coral reefs surrounding high gradient island systems, such as Hawaiʻi, are particularly vulnerable to influences from terrigenous sources. This is especially true on the island of Hawaiʻi because the substrate is predominately composed of porous basaltic rock and, being young on a geological timescale, lacks an abundance of soil for bioremediation. Further compounding these effects is the human population growth. Hawaiʻi Island has experienced one of the fastest human population growths over the last decade or so (23.6% increase in 1990-2000) in the State of Hawaiʻi (U.S. Census Bureau 2000). Synergistically, these factors have a great potential to produce direct anthropogenic effects on fringing coral reef ecosystems.

Some of these effects can be manifested in states of reduced health of corals. Several symptoms for poor health in reef-building corals have been reported in Hawaiʻi and range from bleaching, coral tissue loss, infectious diseases for which pathogens have been identified, and other abnormalities in cellular, tissue, and skeletal morphologies of unknown causes (Gulko et al. 2000, Friedlander et al. 2005, Sutherland et al. 2004). Coral diseases have been found on most major coral species of the main Hawaiian islands (*Porities lobata, P. compressa, Montipora capitata, M. patula, and Pocillopora meandrina*: Gulko et al. 2000, Friedlander et al. 2005). The last few decades has seen an increase in the frequency of coral diseases over a range of depths and habitats in Hawaiʻi (Gulko et al. 2000, Friedlander et. al 2005). Different coral genera exhibit different types and prevalence of diseases at different locations in Hawaiʻi (Aeby 2004, this study). These conditions, with the exception of

bleaching, are poorly studied. In fact, it is not clear how well these coral conditions adhere to the definition of disease because the mechanisms and degree of functional interruption are largely unknown.

Hawai'i Island is the largest of all Main Hawaiian Islands and contains some of the most well-developed coral reefs. However, data on diversity, abundance, and distribution of compromised health conditions in corals around Hawai'i Island are largely absent thus far. Our monitoring program has been surveying corals in the tide pools at Wai'ōpae, Hawai'i Island, the southern part of which was designated as a Marine Life Conservation District (MLCD) where no collection of any organisms is allowed. The initial results of prevalence rates and changes in types of unhealthy states in corals from 2006 to 2008 are presented here.

Material and Methods

All field surveys were conducted in Wai'ōpae tide pools that fall inside and outside of the MLCD (19°29'55"N 154°49'06"W, Fig. 1).
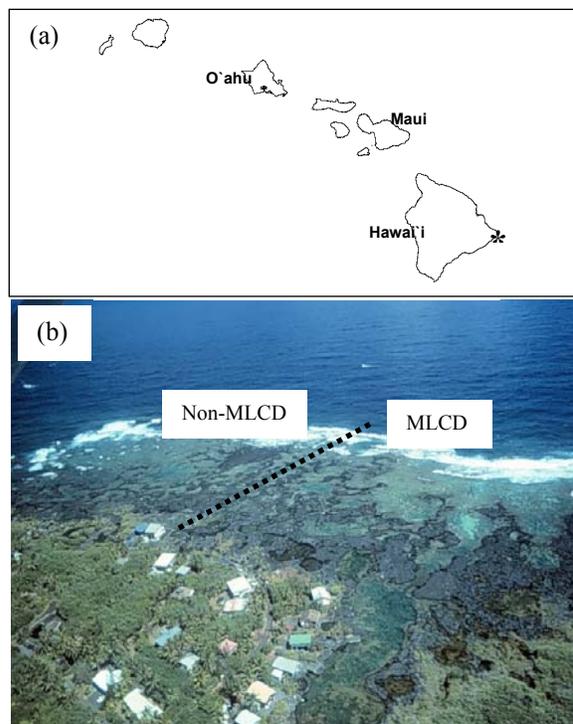

Figure 1: (a) Map of Main Hawaiian Islands showing the location of Wai'ōpae on Hawai'i Is., indicated by *. (b) Aerial photograph of Wai'ōpae tide pools showing the boundary (broken line) of the Marine Life Conservation District. Note that this photograph was taken circa 1999. Currently there are many more houses along the shoreline. Photo taken by J. Coney, UHH.

For transect surveys, coral colonies along six randomly placed 25m transect lines each inside and outside of the MLCD were surveyed on four occasions in 2006 and again in June 2008. Coral colonies along these transects were identified to species, and proportions of colony areas occupied by various health states were visually estimated. To monitor changes in the health state of individual coral colonies, locations of four colonies of each of the six most common coral species(*Montipora capitata, M. flabellata, M. patula, Porites lobata, Pocillpora meandrina, Pavona varians*) were recorded inside of the MLCD, and four additional colonies of each species outside of the MLCD. These coral colonies were originally randomly selected regardless of their health status and repeatedly observed monthly between Feb 2007-March 2008 by both visual inspection and image analysis. For the image analysis, a photo was taken from the plain parallel to the coral colony within the frame size that fit the entire colony. The relative areas of the entire colony and that of unhealthy states were calculated using an image analysis software, ERDAS Imagine 9.1 (Leica Geosystems) at the GIS Laboratory at University of Hawai'i at Hilo. All statistical analyses were performed using Minitab 15 (Minitab Inc.).

Results

The pooled survey data from randomly placed transects in 2006 and 2008 showed that the ten most common scleractinian coral species found at Wai'ōpae had significantly different (Kruskal-Wallis test, $p<0.001$) prevalence of colonies showing symptoms of any unhealthy states, with *Porites compressa* and *Montipora capitata* being most unhealthy and *Pavona varians* being the most healthy (Fig. 2).
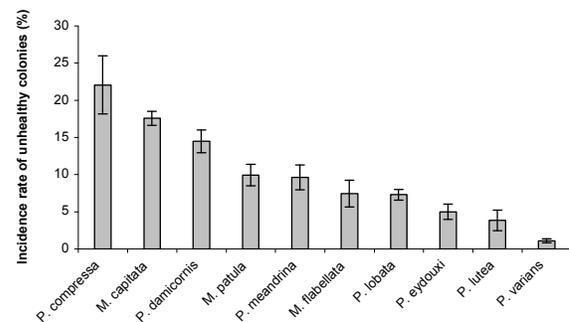

Figure 2: Mean incidence rates (% ± std. error, n=6-694) of unhealthy corals according to species at Wai`ōpae, Hawai'i pooled from the 2006 – 2008 random transects.

The susceptibilities to different types of afflictions were species- and genus-dependent. Comparison among common genera showed significant differences among genera in susceptibility to bleaching, skeletal growth anomalies, and tissue loss of unknown causes (Kruskal-Wallis, $p<0.001$) with

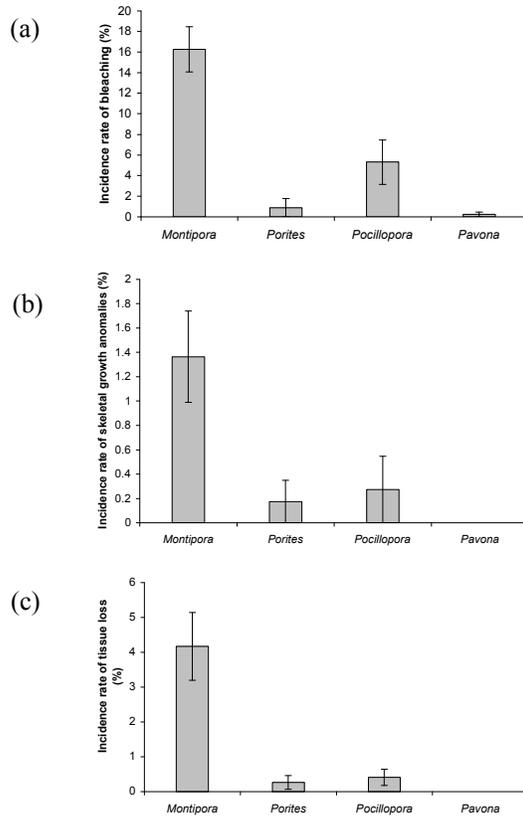*Montipora* being the most susceptible to all three conditions (Fig. 3).

(a)

(b)

(c)

Figure 3: Mean incidence rates (% ±std. error, n=22-121) of (a) bleaching; (b) skeletal growth anomalies; and (c) tissue loss observed in the four most common coral genera at Wai`ōpae, Hawai'i from the 2006 – 2008 random transects.

Comparisons of incidence rates of unhealthy colonies between inside and outside of the Wai'ōpae MLCD revealed no consistent trend across coral genera (Fig. 4). However, the individual genus data showed that the prevalence of unhealthy colonies was higher inside the MLCD than outside for *Montipora* and *Pocillopora* (both p<0.00), same between two areas for Porites (p>0.05), and lower inside the MLCD than outside for *Pavona* (p<0.05) (Fig. 4).

The monthly repeated observations of coral colonies representing the six most common species have produced a large database of image archives that will continue to expand. With these images, skeletal growth anomalies were the easiest to track since the growth anomalies have well defined boarders that can be traced by image analysis. *Montipora capitata* showed the highest incidence rate of skeletal growth anomalies of all coral species in the random transect surveys of 2006-2008 (19.32±3.91%, data not shown).
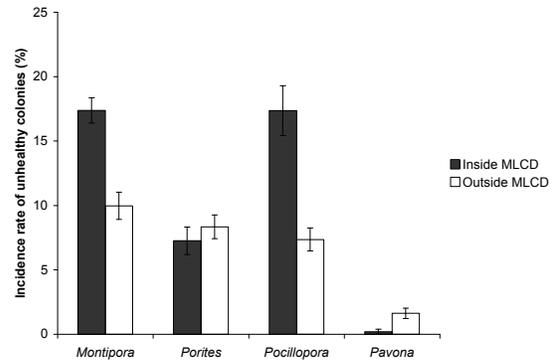
Figure 4: Comparison of the mean incidence rates (% ± std. error, n=77-612) of unhealthy corals inside and outside of the Wai'ōpae Marine Life Conservation District (MLCD) separated into the four main genera.

The size of skeletal growth anomalies was expressed as relative to the entire coral colonies that housed them to account for growth of corals. Generally, the relative size of skeletal growth anomalies increased slightly or remained the same both inside and outside of the MLCD between February 2007 and February 2008 (Fig. 5). In one of the 8 colonies, the skeletal growth anomaly occupied >90% of the entire colony surface for the duration of our observations, eventually resulting in death of the entire colony by February 2008 (Colony "In 1", Fig. 6a-b). Other *M. capitata* colonies maintained the same relative sizes of skeletal growth anomalies (e.g. Colony "In4", Fig. 6c-d) or remained completely devoid of growth anomalies for the same observation period. Overall, there were no significant correlations between the skeletal growth anomalies (change in size or frequency) and species, location, or season (all p>0.05).
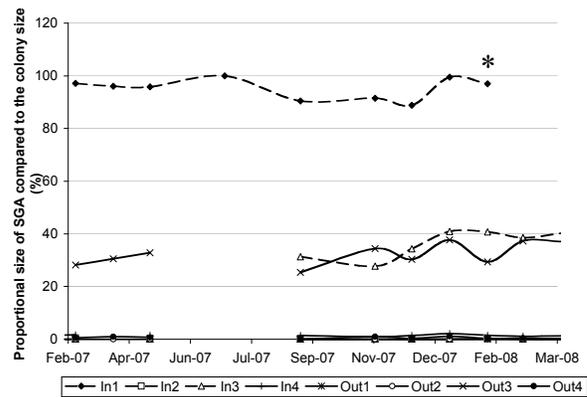
Figure 5: Changes in the sizes of skeletal growth anomaly (SGA) proportional (%) to the whole colony size of four repeatedly analyzed *M. capitata* colonies inside the MLCD ("In1"-"In4") and four colonies outside ("Out1"-"Out4"). Data for July 2007 for colonies except Colony "In1" are unavailable. *The first colony inside the MLCD ("In1") completely died; see Fig. 6.
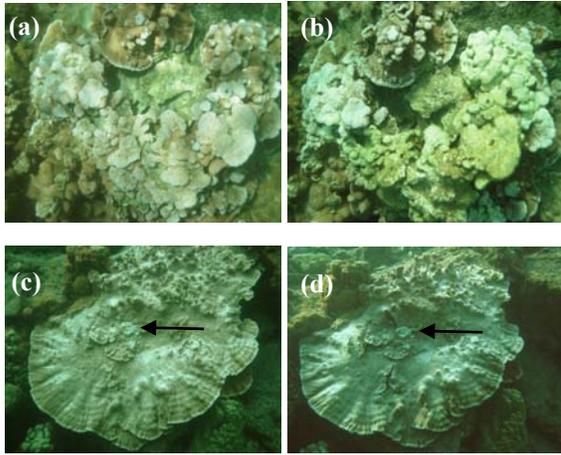
Figure 6: Examples of changes in skeletal growth anomalies in repeatedly observed *Montipora capitata* colonies at Wai`ōpae, Hawai'i. Colony "In1" in February 2007 (a) and in February 2008 (b), showing the expansion of skeletal growth anomalies (shown as white patches with anomalous textures in the photos) resulting in the colony's death by February 2008. Colony "In4" in February 2007 (c) and in February 2008 (d), showing no change in the relative size of the skeletal growth anomaly (indicated by arrows) over the same period of time.

Discussion

Hawaiian Archipelago showcases geologic history in a linear geographic scale from the southeast end to the northwest extreme. Hawai'i is the southern-most, and thus youngest (<500,000 years old, Juvik and Juvik, 1998), island of the archipelago. The incipient fringing reefs around Hawai'i Island, therefore, signifies the beginning of coral reef ecological succession along the Hawaiian island chain. Despite such a young geologic age, the reefs at Wai'ōpae on the east side of Hawai'i Island boast a relatively high level of live coral cover (mean 38.9% cover outside of the MLCD and 44% inside, 2004 surveys Hallacher et al. unpublished). Our monitoring program that began in June 2006 which is still continuing has depicted the health status of coral species there.

The incidence of unhealthy corals at Wai'ōpae is much higher than other Hawaiian locations. What is especially anomalous, visibly and statistically, is the number of *Montipora* that have succumbed to afflictions, especially to skeletal growth anomalies. For example, Aeby (2004) found the mean incidence rate of unhealthy *Montipora* to be less than <1% in uninhabited Northwestern Hawaiian islands, compared to 22.1% at Wai`ōpae. Our preliminary surveys have found that the proportions of *Montipora* colonies with skeletal growth anomaly were <1% at Leleiwi and Ke`ei (data not shown), both sites near relatively high human populations on Hawai'i Island.

There is an on-going debate on whether pathogens are the primary cause of coral diseases or if pathogens incite secondary opportunistic infection or reaction when corals are already under physiological stress (Harvell et al. 2007, Lesser et al. 2007). The physiological function of coral colonies may change due to environmental stress affecting the coral, resulting in altered environmental conditions for coral-associated bacteria (Pantos et al. 2003). Regardless of directness of the link, reports are suggesting that increases in the number of both new diseases and species affected are linked to human-induced alterations in coral reef environments both in terms of land-based sources of pollution, sedimentation, and eutrophication as well as global climate changes such as seawater temperature rises and ocean acidification (Bruckner & Bruckner 1997, Harvell et al. 1999, Porter et al. 2001, Rosenberg & Ben-Haim 2002, Sutherland et al. 2004, Bruno et al. 2003, Pantos et al. 2003, Aeby 2004). Most studies have been unable to identify exogenous organisms or intracellular infection by parasitic agents for skeletal growth anomalies. It has been suggested that anomalous skeleton formation and growth may be influenced by environmental factors; however, no clear correlation has been established. Several investigators have postulated that disease incidence might be associated with elevated nutrients with or without sediment loads delivering potentially pathogenic organisms to the marine environment (Richardson 1998, Sutherland et al. 2004, Breitbart et al. 2005, Bruno et al. 2003).

Causes of the observed high rates of unhealthy states in corals at Wai'ōpae are also not known. The porous basaltic substrate and proximity of the reef to the coastline due to the absence of continental shelf or well-developed reef crest found at this site are typical of other windward locations on the same island. What might be negatively affecting environmental conditions for benthic organisms at Wai'ōpae is the reduced water circulation due to the substrate forming tide pools. All of the corals that were surveyed in this study were found in tide pools with varying levels of connection to open water. The water circulation pattern in Wai'ōpae tide pools has not been investigated but is expected to be very complex because of the high level of complexity in substrate topography as well as freshwater inputs through groundwater and land runoffs. At low tides, many of the tide pools are ponded, whereas the seawater level at high spring tides covers the tops of tide pool walls and reaches the residential houses along the shoreline.

Water quality inside the tide pools would likely affect benthic organisms such as corals more than mobile organisms. If coral afflictions at Wai'ōpae are direct or indirect results of terrigenous stressors, than accumulation of runoff, groundwater, and reduced water circulation at low tides must be exacerbating their effects. This might partially explain why the

coral genera that are more susceptible than others, such as *Montipora* and *Pocillopora*, are showing higher unhealthy rates inside the MLCD than outside the MLCD (Fig. 4). The tide pools inside the MLCD are more isolated and ponded at low tide than those outside the MLCD (Fig. 1b). Another compounding effect that has the potential of creating differences in stressor level in tide pools inside and outside the MLCD is the human use. Of the estimated 87,190 people who visited Waiʻōpae during 2004-2005, 90.0% of them stayed within the 50-acre MLCD (Shea Flanders and Settlemier, 2007). A larger proportion (36%) of people participated in non-consumptive underwater activities, such as snorkeling, inside the MLCD than outside (21%, Shea Flanders and Settlemier, 2007). The MLCD at Waiʻōpae was designated as a no take zone in 2003. The effect of this MLCD designation needs to be carefully assessed in terms of the benefits of various species protection at the cost of increased visitor attraction and usage. These results from coral monitoring illustrate the complexity of the many factors influencing coral health. More detailed analysis comparing multiple environmental differences inside and outside the MLCD may shed light on what elements must be incorporated into conservation protection in order to adequately address coral health concerns.

It is not known when the health states of corals at Waiʻōpae began to deteriorate to the present levels because there had been no regular monitoring effort focusing on the health of corals there before this program. Regarding skeletal growth anomalies specifically, the disease state does not seem to advance rapidly (Fig. 5 and Fig. 6). One exception to this was found in the colony, "In1", that was heavily (>90%) covered with skeletal growth anomaly for 10 months of our observation without showing further deterioration before succumbing rapidly to turf algal invasion in the last two months (Fig. 5). In order to monitor changes in health conditions of corals at Waiʻōpae effectively, we will continue our periodic random transect surveys and photo documentation of repeatedly observed colonies. Current efforts are being focused on evaluating the water quality in Waiʻōpae tide pools and assessing potential correlations between water quality characteristics to incidence, types, and progression of coral afflictions.

References

Aeby GS (2004) Baseline levels of coral disease in the Northwestern Hawaiian Islands. In: Northwestern Hawaiian Islands 3rd Scientific Symposium, 2–4 November 2004. NOAA National Marine Fisheries Service Pacific Islands Fisheries Science Center, Honolulu.

Breitbart M, Bhagooli R, Griffin S, Johnston I, Rohwer F (2005) Microbial communities associated with skeletal tumors on *Porites compressa*. FEMS Microbiol Lett 243:431-436

Bruckner AW, Bruckner RJ (1997) Outbreak of coral disease in Puerto Rico. Coral Reefs 16: 260

Bruno JF, Petes LE, Harvell CD, Hettinger, A. (2003). Nutrient enrichment can increase the severity of coral diseases. Ecol Lett 6: 1056-1061

Friedlander A, Aeby G, Brainard R, Clark A, DeMartini E, Godwin S, Kenyon J, Kosaki R, Maragos J, Vroom P (2005) The State of Coral Reef Ecosystems of the Norhwestern Hawaiian Islands. The State of Coral Reef Ecosystems of the United States and Pacific Freely Associated States: 2005. Northwest Hawaiian Islands Chapter.

Gulko D, Maragos J, Friedlander A, Hunter C, Brainard R (2000) Status of coral reefs in the Hawaiian Archipelago. Status of Coral Reefs of the World 2000:219-238

Harvell CD, Kim K, Burkholder JM, Colwell RR, Epstein PR, Grimes DJ, Hofmann EE, Lipp EK, Osterhaus AD, Overstreet, RM, Porter JW, Smith GW, Vasta GR (1999) Emerging Marine Diseases, Climate Links and Anthropogenic Factors. Science 285:1505-1510

Harvell CD, Jordan-Dahlgren E, Merkel S, Rosenberg E, Raymundo L, Smith G, Weil E, Willis B. (2007). Coral disease, environmental drivers and the balance between coral and microbial associates. Oceanography 20: 58-81.

Juvik SP, Juvik JO (1998) Atlas of Hawaiʻi. Third Edition. University of Hawaiʻi Press, p 38.

Lesser MP, Bythell JC, Gates RD, Johnstone RW, Hoegh-Guldberg, O (2007) Are infectious diseases really killing corals? Alternative interpretations of the experimental and ecological data. J Exp Biol Ecol 346: 36-44

Pantos O, Cooney RP, Tissier MDA, Barer MR, O'Donnell AG, Bythell JC (2003) The bacterial ecology of a plague-like disease affecting the Caribbean coral *Montastrea annularis*. Environmental Microbiology 5(5): 370-382

Porter JW, Dustan P, Jaap WC, Patterson KL, Kosmynin V, Meier OW, Patterson ME, Parsons M (2001) Patterns of spread of coral disease in the Florida Keys. Hydrobiologia 460: 1-24

Richardson LL (1998) Coral diseases: what is really known? TREE 13:438-443

Rosenberg E, Ben-Haim Y (2002) Microbial diseases of corals and global warming. Environ Microbiol 4(6): 318-326

Shea Flanders L, Settlemier C (2007) Effectiveness of the Waiʻōpae Tide Pools Marine Life Conservation District after three years. Cape Kumukahi Foundation Report.

Sutherland KP, Porter JW, Torres C (2004) Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar Ecol Prog Ser 266:273-302