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An Outbreak of a Chronic Tissue Loss Syndrome Among Massive Corals on Majuro Atoll

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Abstract. Chronic tissue loss with a range of lesion types and signs (typically spreading only 6 mm/month) is described that is affecting massive corals on Majuro atoll (particularly *Hydnophora, Platygyra, Favia Goniastrea* and *Pavona*), as well as *Turbinaria, Scapophyllia, Leptoria* and other genera, but not *Porites*. Multiple, chronic tissue loss lesions typically progress for up to six years on a single colony before destroying all tissue, and are often accompanied by small, discrete white spots. Prevalence is locally very high (over 70%) and is shown to co-vary with the presence of nearby human settlement and near-shore water turbidity. Mortality appears to sometimes result from synergistic interactions between potential disease processes and an encrusting, coral-killing algae resembling *Lobophora*. This syndrome is restricted to the populated regions of Majuro atoll, and has not yet been detected on other atolls of the Marshall Islands, where massive colonies are typically lesion-free. As a result of this and other disease outbreaks, coral biodiversity on Majuro is in steep decline, as many once dominant corals (*Isopora, Stylophora, Montipora, Porites*, etc.) are also being extirpated locally by chronic tissue loss diseases.

Key words: RMI, coral disease, *Metapeyssonnelia*, *Hydnophora,* faviids

Introduction

The Marshall Islands, an archipelago of over 24 atolls and isolated islands, features coral reefs with high (often 100%) coral cover. Reefs on most atolls enjoy pristine conditions and have so far been little-impacted by temperature stress, storm damage or destructive fishing (Beger et al 2008). There have been only a few reports of coral disease in the RMI, including acroporid White Syndrome (Pinca et al. 2005); syndromes such as black band disease are rare, although tumor-like growth anomalies are moderately common (Beger et al. 2008). The capitol atoll of the RMI, Majuro, is densely populated, with over 27,000 residents, the majority of whom are crowded into the urbanized eastern end ("downtown") where population density exceeds 20,000/km^2, among the highest in the Pacific. Recently, a variety of disease signs were discovered in the sparsely populated, less polluted western half of Majuro. In addition to coral disease, an intense *Acanthaster* outbreak (limited to Majuro and nearby Arno atoll) peaked on Majuro from 2003-2006, killing over 90% of corals at some sites (Beger et al. 2008). Many reefs have declined greatly in ten years.

Given the long history of dense settlement on eastern Majuro, particularly after WWII, the current degraded condition, featuring low coral diversity and the disappearance of giant anemones near the "downtown" district, is hardly surprising. Even a casual inspection reveals that, despite high coral cover (dominated by *Acropora* and *Pocillopora* spp.), many coral genera that abound on the pristine "outer islands" such as Arno and Rongelap are entirely absent on these eastern reefs. These genera include *Isopora*, *Stylophora*, *Turbinaria, Scapophyllia, Merulina*, and a wide variety of massive forms (*Hydnophora*, *Astreopora*, *Favia*, *Goniastrea*, *Pavona*, *Porites*, etc.). *Symphyllia* and *Lobophyllia spp.* still persist in east Majuro. Most of these missing species, with the exception of *Merulina*, still exist in Majuro's south-central reefs (west of the airport) in the district of Ajeltake, where human population density is far lower than in downtown Majuro. Recently a very high (and growing) occurrence of chronic tissue loss and algal-overgrowth mortality among massive coral was detected in Ajeltake, prompting a multi-year study of disease progression in individual colonies in an attempt to characterize disease signs. Chronic tissue loss lesions have been found on all these taxa, most prominently on *Hydnophora microconos, Platygyra sinensis*, *Favia speciosa*, and *Pavona*

duerdeni, which are the focus of this preliminary study.

Given the existence of both a sewage outfall (near the southeastern atoll corner) and the scarcity of toilets (causing most fecal waste to be left on shore), along with piggeries along the shore, the influence of sewage microbes and nutrients on coral health deserves examination. The link between sewage and coral disease has already been considered in the Caribbean (Mitchell and Chet 1975, Harvell et al. 1999, Kaczmarsky et al. 2005) and parts of the Pacific (Dinsdale et al. 2008) but not in Micronesia. Fine scale mapping of disease incidence was done in an attempt to elucidate the possible role of water quality.

Material and Methods

Most time series photography was done at the "Sea-wall" site (N 7.058 E 171.225; see Figs. 1, 9) using a Sony F828 digital camera in an Ikelite housing. For ease and safety of shore entry, site visits (SCUBA and snorkel) were restricted to high tide, which usually coincided with late afternoon, optimizing lighting conditions. Depths of the central shelf, where most massive colonies were found, ranged from 2.5 to 5 meters. On each visit, approximately forty colonies were photographed "free hand" (without a camera positioning frame), either directly overhead or at an individually optimized camera angle (determined by the location of lesions and surrounding reef structure) three to four times each year. Underwater navigation was based on a mental map that developed after multiple visits. Images were contrast- and color-optimized with Adobe Photoshop (auto-levels command). Spreading rates of lesions were determined using small objects (coins or rulers) for scale and as aids for stacking images, their precise alignment being based on intricate surface features.

Disease rates were determined by photographing all massive colonies in the central reef shelf during swims up to 1 km in length, and sequentially pooling the photographs into sets of 20-30 colonies. While not overly abundant, these colonies are conveniently conspicuous. The great majority of *H. microconos* and *P. sinensis* colonies in a 5 km reef segment between Peace Park and Ajeltake Elementary School (see Fig. 9; n=260 colonies) were photographed and analyzed visually for the presence or absence of disease signs or algal overgrowth.

Scoring condition of colonies

Lesions were categorized as either active or inactive (fish bite marks were ignored). Active lesions have boundaries lacking surface relief and may be marked with a band pattern or various colonizing algae, particularly bright green filamentous forms. Inactive lesions appear "sunken", surrounded by a raised calcified rim formed by coral growth.

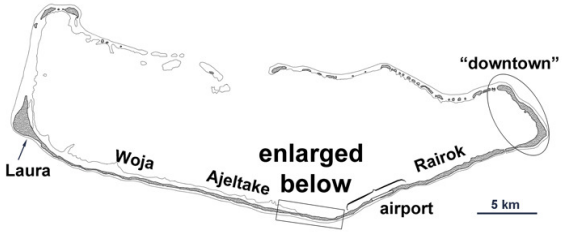

Figure 1: Study site on Majuro (in box). Note bracket near the airport, indicating a "gap" region of very few residences and pollution sources.

Results

Lesions on massive corals (Fig 2) were diverse, due in part to a variety of opportunistic algae that often carpets and masks freshly-killed surfaces.

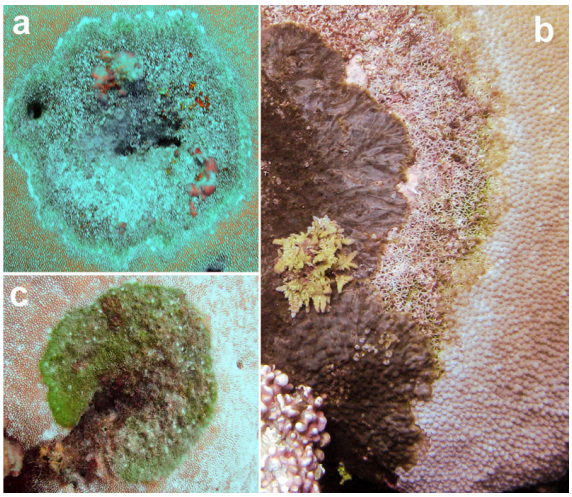

Figure 2: Typical appearance of lesions on *Hydnophora microconos.*

Several hundred colonies with chronic tissue loss have been photographed, with extended time series (between 5 and 12 frames over two or more years) captured for fifteen *H. microconos*, five *P. sinensis*, three *F. speciosa* and one *Scapophyllia* colony at the seawall site. Lesions are also common on colonies of *P. duerdeni* and *Goniastrea retiformis*. A species of *Favites* is rarely seen with lesions. The similarity of signs and spreading rates, along with their geographic overlap, supports the pooling of these lesions together within a single syndrome. This chronic tissue loss syndrome involves multi-focal lesions (from 2 to over 10 cm) on single colonies, some of which were apparent for as long as five or six years (Fig 3). Even though these lesions have not yet been examined microscopically, the chronic and progressive tissue loss is consistent with disease processes as opposed to other biological processes such as feeding scars. Rate

of lesion progression varies from 0.1-0.4 mm/day. On many *H. microconos* colonies, a narrow (1-2 mm) irregular, brownish, darkened band is joined by discontinuous, irregular inner unpigmented (white) banding. Outside the lesion, coral tissue often displays a subtle hypo-pigmented band 5-10 mm wide; this "halo" region may also appear grey (Fig. 4). Such banding is not apparent on other taxa, and even on *H. microconos* is often not visible due to the sporadic involvement of a distinctive, opportunistic encrusting algae (appearing dark olive-green) whose growth roughly matches the rate of lesion expansion (and is further considered below). Also, grass-green filamentous algae show non-persistent growth on some lesions (Figs. 2b, 6).

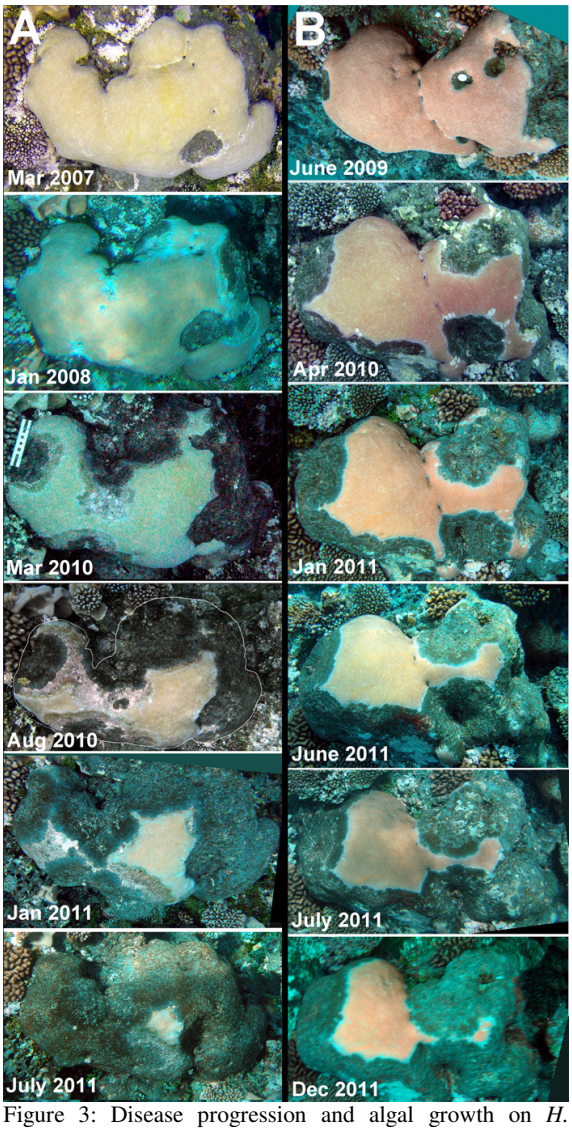

Figure 3: Disease progression and algal growth on *H. microconos* colonies at the seawall site.

Lesions frequently originate at invertebrate burrows (i.e., those of alpheid shrimp) (Fig. 4), but also appear on unblemished coral surfaces anywhere on the colony. Note the green growth on *F. speciosa* lesions (Figs. 4, 6), which is also found on many *P. sinensis* lesions.

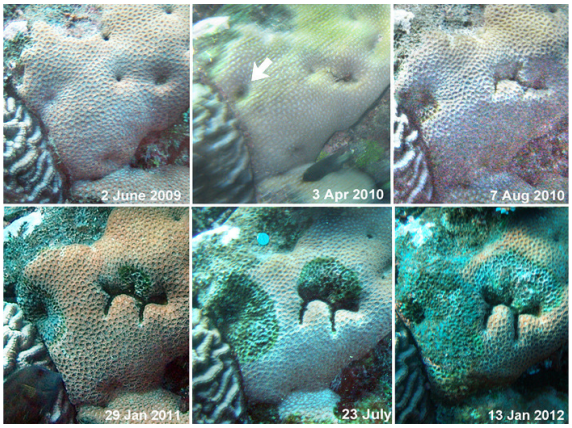

Figure 4. *Favia speciosa*: appearance of two lesions from invertebrate burrows, including that of alpheid shrimp. US dime shown for scale (fifth panel).

White, unpigmented oblong patches of live tissue roughly 5-15 mm in diameter are a conspicuous and common, but not universal, feature of *H. microconos* lesions (Fig. 5, 7; note also Fig. 3B, second panel). They typically appear after lesions are established, suggesting a secondary process. Such spots are found less frequently near *P. sinensis* lesions but are not found on any other taxa. These "white spots" can be transient, often disappearing, or may spread and migrate laterally; a small patch of central tissue may die. When present, white spot abundance varies from a few to dozens (up to 50 on a large colony) and can be found in locations, such as the low cusp connecting two convex coral surfaces, where fish bite marks do not usually occur. The spatial dynamics of white spots are further considered below (Fig. 7).

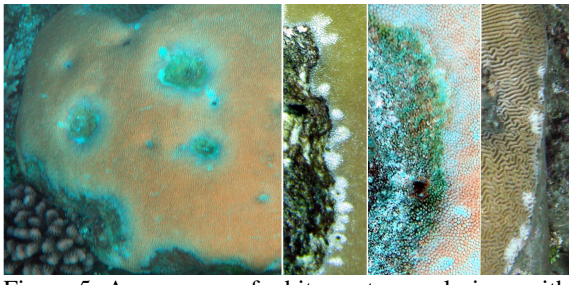

Figure 5: Appearance of white spots near lesions with and without encrusting algae on *H. microconos*, and on *P. sinensis* (far right panel).

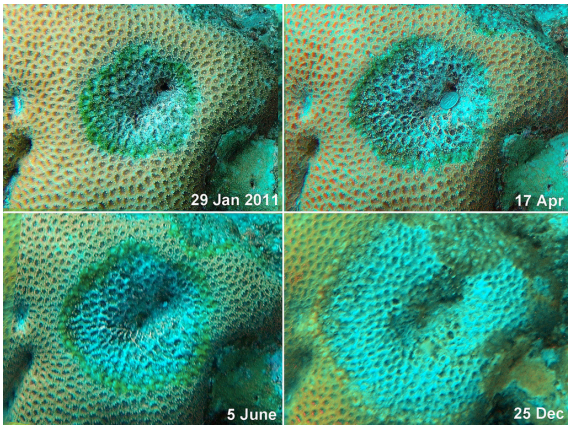

Figure 6. *Favia speciosa*: A year-long progression of one of two lesions on this colony (US dime shown for scale).

A three year time series demonstrates the shifting appearance of white spots near *H. microconos* lesions (Fig 7).

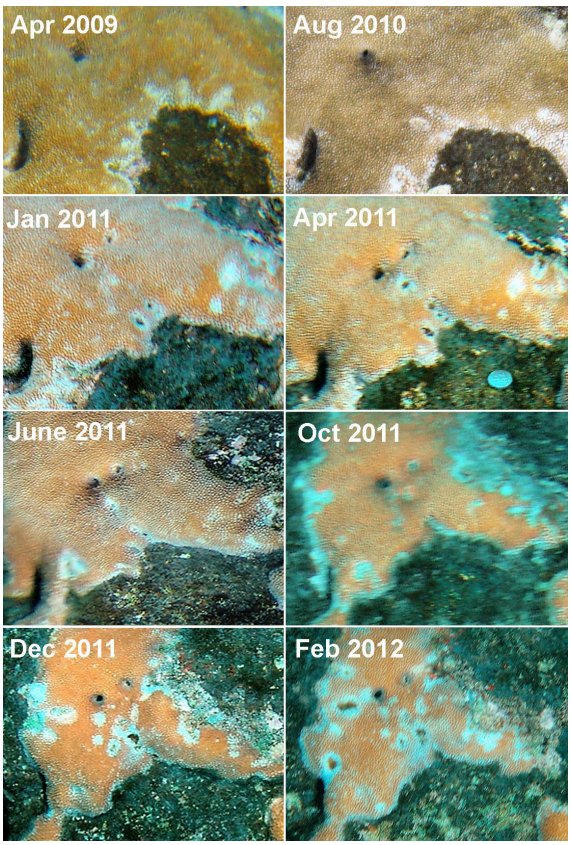

Figure 7: Progression of white spots near lesions on *H. microconos*. US dime (17.5 mm diameter) indicates scale.

Lesions and encrusting algal overgrowth are particularly common on *Pavova duerdeni* (Fig. 8), with lesions lacking banding and often showing green algal overgrowth. The tightly-adhering, encrusting algae are identical to those growing on *H. microconos* and other taxa, and most closely resemble the genus *Lobophora*, an algae that is abundant at this site in both its adhering and leafy forms. Rate of chronic tissue loss, shown below, vary across a colony (from <0.1 – 0.3 mm/day).

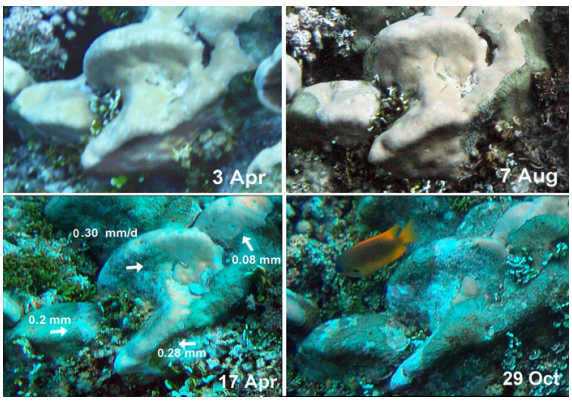

Figure 8. *Pavona duerdeni* with lesions and algal overgrowth, 2010-2011, immediately adjacent to colony shown in Fig 3A; US quarter shown for scale.

Over 260 *Hydnophora* and *Platygyra* colonies (combined) were scored for lesion type and % living tissue along a 5 km reef segment (Fig 9). Note the low occurrence and small size of lesions at the eastern end, with a disease "hot spot" at the seawall site.

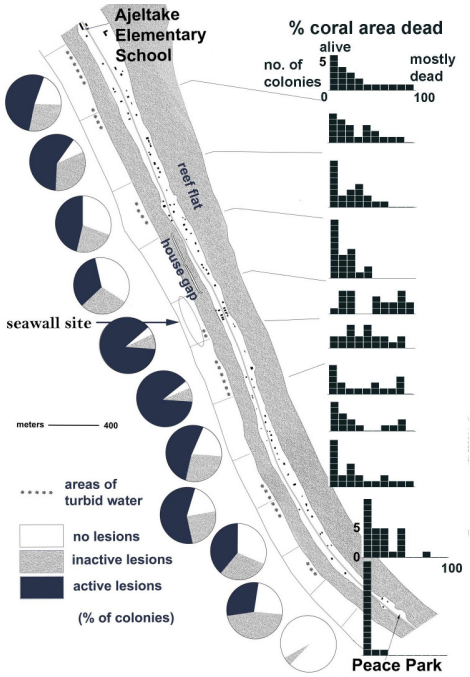

Figure 9. Lesion abundance (n=260 *Hydnophora* and *Platygyra* colonies surveyed), divided into 11 reef segments along a pollution gradient. Residences and roads shown to scale. Histograms depict % area of lesions (algae or exposed skeleton)/colony in each sample (n=20-30).

Discussion

Clearly, the chronic progressive tissue loss and algal overgrowth described here are causing a high level of mortality among the massive coral populations of Majuro, with a similar occurrence of chronic tissue loss well on its way to eliminating the remaining *Isopora, Stylophora and Turbinaria* populations (among others). Rates of chronic tissue loss are exceptionally slow, only 3-12 mm/month compared to 5-12 cm/month for yellow band disease, and 60 cm/month for white syndromes (Gil-Argudelo et al. 2000). The signs, including common white spots, do not resemble any known disease.

The geographic distribution of processes described above is striking, being restricted to certain populated areas of Majuro and entirely absent on the other atolls so far examined. The fact that most massive corals are absent from eastern Majuro suggests that this region has a long history of impact. Indeed, two remaining *Platygyra* colonies that were present at a Rairok study site in 2003 were both killed by a similar process of chronic tissue loss. No colonies of *Hydnophora*, *Pavona*, *Isopora*, *Scapophyllia*, or other typically common massive coral can be found in Rairok; *Hydnophora* and *Pavona* first appear at the airport site. However, a fossil fragment of *Scapophyllia* was found on land in Rairok, demonstrating that it once grew here (pers. obs.).

There is an apparent correlation of chronic tissue loss with a pollution gradient, with the most lesions found near residences and plumes of brown water, suggesting that this local outbreak may be caused by local sources of pollution. These pollution gradients have not yet characterized by microbial or chemical assays. This pattern is similar to that reported from the Line Islands (Dinsdale et al. 2008) but on a smaller spatial scale. Perhaps, given the karstic structure of the atoll foundation, i.e. the likely presence of water-eroded cavities within the subtidal rock, there may be areas where wastewater from septic tanks flows efficiently onto the reef; this in turn might explain the persistent presence of turbid brown water at certain locales where lesion occurrence is very high.

In fact, the impact of this syndrome is (evidently) so severe immediately to the west of the Ajeltake school site that no surviving *Hydnophora*, *Platygyra,* or *Pavona* colonies can be found, only dead skeletons.

Patterns of lesion abundance (Fig. 9) are at odds with observations of two large, rare colonies of *Leptoria phrygia*, the only two known on Majuro. One colony just west of the seawall "hotspot" is unaffected, but one located 2 km to the east is, unfortunately, seriously affected. Yet this may reflect small-scale geographic variations in water quality; note that lesions are much less abundant and severe immediately to the west of the "hotspot", where the unaffected colony is found.

The syndrome on massive corals and other corals such as plating *Turbinaria* species could be referred to as chronic tissue loss syndrome to distinguish it from other syndromes like "dark band" that are less persistent or white syndrome which produces a faster rate of tissue loss.

It appears that associated filamentous and encrusting algae are acting opportunistically. Less than 5% of colonies suffering tissue loss at the seawall site have active algal growth over live coral tissue. Yet overgrowth by the *Lobophora*-like algae is often found on unaffected coral, particularly *Pavona duerdeni,* and also on many other taxa; like the chronic tissue loss syndrome, this algal overgrowth is limited to the populated portions of Majuro atoll in the RMI.

The existence of small lesions that fail to expand significantly over several years complicates the task of assessing disease on Majuro. It appears that these chronic processes are more common in the eastern, less-polluted end of the 5 km study site.

Further study, i.e. using histological and molecular techniques, is needed to better characterize these chronic lesions on Majuro, particularly to tease out the origin of the enigmatic white spots. A metagenomic analysis of the microbial metabiomes of *Hydnophora* colonies found on Arno vs. various locations on Majuro would be of interest. An investigation of the other polluted, densely populated region, Ebeye on Kwajelein atoll, is also warranted.

Acknowledgement

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