Mr. Terry Lilley, an Eyes-of-the-Reef volunteer, alerted us of an unusual outbreak of coral mortality at Tunnels and other portions of the north shore of Kauai. Although Mr. Lilley has been monitoring these reefs for many years, it is only this year that mortality has been unusually noticeable. Many corals throughout the reefs of North Kauai were manifesting lesions some of which appeared transmissible and to progress rapidly (several linear centimeters per week). Because of the extensive mortality evident from Mr. Lilley's photos, we decided to carry out a field investigation to sample corals in attempts to figure out what may be causing this mortality. On 5 August 2012, Ms. Megan Colvin (UH) and I accompanied Mr. Lilley to Tunnels, a site particularly hard hit, to photodocument lesions, sample corals, and semiquantify the extent of the outbreak through point intercept and belt transects. Paired normal and lesion tissues were collected from 16 different coral colonies, fixed in zinc formalin, and processed for microscopic examination. This report focuses on the gross and microscopic pathology encountered in corals from Tunnels. Survey data are available from Dr. Greta Aeby (HIMB).

On microscopy, of samples with lesions, tissue death (necrosis) associated with fungi or cyanobacteria comprised the majority affected colonies (11/16) followed by necrosis alone (3/16), helminth infestation (1/16) and suspect parasitic corals (1/16). Of the paired "non-lesion" tissues, only three of these were without microscopic changes. Of the remainder, increased inflammatory cells were present in basal body wall (6/16), necrosis associated with fungi or algae was present in 3/16, and reduced numbers of symbiotic algae, atrophy, or dissociating gastrodermal cells were evident in the remainder.

Final diagnosis: Accession 1-Metazoan; Accession 2-Necrosis; Accession 3-Necrosis; Accession 4-Necrosis; Accession 5-Necrosis; Accession 6-Fungal infection; Accession 7-Bleaching; Accession 8-Necrosis; Accession 9-Undetermined-Normal tissue; Accession 10-Fragmentation; Accession 11-Bleaching; Accession 12-Necrosis; Accession 13-Necrosis; Accession 14-Necrosis; Accession 15-Undetermined; Accession 16-Necrosis; Accession 17-Undetermined; Accession 18-Necrosis; Accession 19-Undetermined-Normal tissue; Accession 20-Necrosis; Accession 21-Atrophy; Accession 22-Necrosis; Accession 23-Undetermined-Normal tissue; Accession 24-Necrosis; Accession 25-Undetermined; Accession 26-Necrosis; Accession 27-Undetermined; Accession 28-Fragmentation; Accession 29-Undetermined; Accession 30-
Necrosis; Accession 31-Undetermined; Accession 32-Necrosis.

Comments: The overall picture was one of a severely degraded reef impacted by sediments and turf algae. Microscopic changes evident in tissues of "non-lesion" corals were suggestive of animals undergoing some sort of chronic stress (inflammatory cells, foci of necrosis, degenerating gastrodermis, loss of symbiotic algae, atrophy of tissues). The most common organisms associated with lesions were cyanobacteria and fungi with helminths and parasitic corals rarely seen. No other bacteria were seen. One plausible scenario is that loss of resilience in the coral host (as evidenced by changes in non-lesion tissues) allowed secondary colonization by fungi and cyanobacteria leading to tissue loss, however, confirmation of this would require controlled experimentation. That said, the presence of cyanobacteria and fungi could explain the transmissibility of the lesions observed in the field. It is tempting to conclude that degraded conditions of the reef could have precipitated infection by fungi and cyanobacteria leading to the lesions seen here, however, confirming this would require longitudinal studies and more systematic sampling over time.

Management: Corals at tunnels are being heavily impacted by fungi and cyanobacteria, and these findings raise a series of questions: 1) What is the identity of these micro-organisms? 2) What is their role in the genesis of lesions in corals? 3) What are the environmental drivers that allow such organisms to become established in corals? Knowing the identity of the organisms would allow us to know if these are single or multi-agent caused diseases. For example, is it only one species of cyanobacteria and fungus responsible or multiple species? Figuring this out would require additional laboratory work (culture, genotyping). Determining the role that these organisms play in causation of lesions is also important. Although our data are compelling, and although cyanobacteria have been documented as primary coral pathogens, the present findings are associative and not proof in themselves of cause-effect. For example, perhaps there is an underlying problem that allows secondary invasion of cyanobacteria or fungi? Answering this would require laboratory manipulations using cultured and purified pathogens under controlled settings. Finally, if we determine that these organisms can cause lesions in corals, then the final, key, and most complex question is what are the environmental drivers that permit corals to become infected with these pathogens, and are there management options to limit the impact of these drivers? Answering this would require longer term sampling of corals over time, preferably before, during, and after an epizootic concomitant with controlled manipulative studies.

Note that in 2009, we documented similar lesions in Montipora from Hanalei Bay with similar microscopic manifestations, so these organisms have been in Kauai since at least that time.

Report Date (mm/dd/yyyy): 9/4/2012
Copies of this report sent to:

If you have questions regarding this case, contact Thierry M. Work MS, DVM, MPVM at 808-792-9520. Include above Case Number. Diagnostic findings may not be used for publication without the pathologist's knowledge and consent.