Earlier this year, Mr. Terry Lilley, an Eyes-of-the-Reef volunteer, alerted us of an unusual outbreak of coral mortality at Makua and Anini on the North shore of Kauai (Fig. 1a). A field investigation of coral mortalities at Makua on August 5, 2012, revealed a severely degraded reef with sedimentation and algal turf overgrowth and large numbers of corals manifesting varying degrees of tissue loss. Corals affected were mainly Montipora patula and microscopy revealed them to be infected with cyanobacteria and fungi. Further details are available in a prior report (21815) filed with the State of Hawaii.

The present report covers findings of a field investigation of dead and dying corals at Anini (Fig 1a). As in Makua, Mr. Lilley noted that mortality of corals was unusual and above and beyond what he recognized as background levels. As in Makua, many corals were manifesting lesions some of which appeared transmissible and to progress rapidly (several linear centimeters per week). As a follow up to the August 5 Makua investigation, we carried out a field investigation at Anini to sample corals in attempts to figure out what may be causing mortality there. Between 30 September and 1 October 2012, Dr. Greta Aeby (University of Hawaii-UH), Ms. Amanda Shore (UH), and I accompanied Mr. Lilley to Anini to photodocument lesions, sample corals, semiquantify the extent of the outbreak through point intercept and belt transects, apply epoxy to affected corals to try and stem progression of lesions, and collect samples of corals to try and culture the cyanobacteria at UH. Paired normal and lesion tissues were collected from 15 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 Anini. Survey data, results of epoxy treatment, and culture results are available from Dr. Greta Aeby (UH).

Findings: The waters on the reef were laden with particulates, and throughout the reef, corals (mainly Montipora capitata) were encroached by sediments and turf algae (Fig. 1b). Live coral cover appeared unusually low as compared to what would be expected on a healthy reef (Fig. 1c). Gross lesions in affected corals occupied ca. 10-80% of the colonies and manifested as distinct semi-circular to amorphous areas of tissue loss revealing bare skeleton covered by amorphous flocculent black to grey material with a distinct black band delineating normal tissues (Fig. 1d); clumps of what appeared to be sediment covered dead skeleton and borders of the lesions (Fig. 1e). Paired lesion and no-lesion samples were taken from 15 Montipora capitata and examined on microscopy.

On microscopy, of 17 samples samples with lesions, ten had tissue death (necrosis) associated with cyanobacteria, five had necrosis with cyanobacteria and fungi, and the remainder had no recognizable microscopic lesions. Of 13 "non-lesion" tissues, one each had either a mild infestation with chimeric parasites, necrosis, necrosis associated with fungi, or reduced numbers of symbiotic algae. Paired normal tissues were not always available, and multiple lesions were collected for two colonies thereby explaining the unequal sample size between lesion and non lesion tissues.
Final diagnosis: Accession 1-Necrosis; Accession 2-Undetermined; Accession 3- Necrosis; Accession 4-Undetermined; Accession 5-Necrosis; Accession 6-Metazoan; Accession 7-Necrosis; Accession 8-Mollusc; Accession 9- Necrosis; Accession 10-Undetermined; Accession 11-Necrosis; Accession 12-Undetermined; Accession 13-Necrosis; Accession 14- Necrosis; Accession 15-Necrosis; Accession 16-Necrosis; Accession 17-Necrosis; Accession 18-Undetermined; Accession 19-Necrosis; Accession 20-Undetermined; Accession 21-Necrosis; Accession 22- Bleaching; Accession 23-Necrosis; Accession 24-Undetermined; Accession 25-Necrosis; Accession 26-Undetermined; Accession 27- Necrosis; Accession 28-Undetermined; Accession 29-Necrosis; Accession 30-Undetermined.

Comments: The overall picture at Anini 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). Cyanobacteria alone were most commonly (59%) associated with lesions with fungal infections affecting an additional 29% of corals. No other bacteria or other associated agents were seen. The microscopic findings for Anini and Makua were very similar. Based on the large percentage (59%) of corals affected by cyanobacteria only at Anini, I suspect this organism is playing a primary role in causing lesions with the likelihood that fungi may pose a complicating factor. In aggregate, 88% of corals with lesions at Anini are infected with cyanobacteria and fungi, and these organisms affect M. capitata at Anini and M. patula at Makua. The presence of cyanobacteria and fungi could explain the transmissibility and spread of the lesions observed in the field at both sites. Given the scale of the event, the large numbers of corals affected, and the consistent preponderance of a few agents (cyanobacteria and fungi) associated with gross lesions that look similar in both Makua and Anini, this outbreak would have to qualify as an epidemic. This is the first time a cyanobacterial/fungal disease on this scale has been documented in Hawaiian corals.

Management: As at Makua, corals at Anini are being heavily impacted by cyanobacteria and fungi. The reefs at both locations are heavily overgrown by turf algae, have low coral cover and large amounts of suspended solids in the water column that appear to be settling onto corals. Whilst sedimentation and presence of cyanobacterial disease appear closely associated, cause and effect remains to be proven. That said, the similarity of the findings at Makua and Anini strongly suggests a common underlying cause, and it is difficult to conclude that the degraded environmental conditions at both sites are not, in some way, driving the occurrence of these infectious diseases on corals. Sedimentation on reefs typically originate from land, and logical potential nearby sources of terrigenous input would be the nearby Hanalei, Wahina, or Kahihiwai Bay watersheds. Whilst other reefs on Hawaii suffer from sedimentation, clearly something on North Kauai is promoting epidemic cyanobacterial disease in corals, and understanding what drives this phenomenon and how to mitigate or prevent it would seem critical to recovering the reefs at Anini and Makua.

Report Date (mm/dd/yyyy): 11/21/2012 Necropy report: Available upon request
Copies of this report sent to: 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.

NOTE: Information in this report supersedes any information from previous reports regarding this case
1. a) Map of locations surveyed-green dot Makua (Report 21815); red dot Anini (this report). Inset shows map of Kauai with approximate area (yellow rectangle) covered by panel a. b) Reef at Anini; note abundant turf algae (red dot) and low cover of live coral that has a more orange color (yellow dot). c) *Montipora*-dominated nearshore reef from Molokai as comparative; note abundant coral cover and lack of turf algae. d) *Montipora capitata* with characteristic cyanobacteria-associated lesion; note bare coral covered by mats of sediment (red dot) and black cyanobacterial mat (arrow) overtaking live coral (yellow dot). e) Close up of d; note sediment on dead coral (red dot) and black cyanobacterial mat (arrow) adjacent to live coral (yellow dot).