BOTANICAL BRIEFS:

A New Bacterial Disease Impacts Pacific Coral Reefs

A new bacterial disease has been discovered which infects reef-building coralline algae. The infection has spread across a 3,600-mile swath of the South Pacific. It has been designated "coralline lethal orange disease," or CLOD, for the bright orange coating which attacks all species of coralline algae tested. Because of the important role played by coralline algae in reef building, this disease has the potential to greatly influence coral reef ecology and reef-building processes.

Barrier, fringing, and atoll reefs are complex ecosystems that depend on calcareous coralline algae for the maintenance of wave-resistant fronts or reef rims. Coralline algae (an order of the Rhodophyta or red algal phylum) are plants that deposit a particularly hard and geologically resistant form of calcium carbonate (limestone). These algae cement together much of the sand, dead coral, and debris of the reef to create stable substrates for other reef organisms. Many have a crust-like growth form and look like red, pink, or purple cement covering large areas of the reef, while others form upright branched heads much like some corals (and are often mistaken for corals). Crustose coralline algae, particularly species of *Porolithon* in the Pacific and in the Atlantic, are the principal cementing agents that produce the structural integrity and resilience of the outer reef rim. Coralline algae are important for the absorption of wave energy that would otherwise erode shoreward land masses, and they play a major role in building and consolidating many shallow reef habitats.

No previously known diseases cause significant mortality of coralline algae. Most ecological studies of algal diseases have concentrated on freshwater phytoplankton or benthic (bottom dwelling) diatoms; little information exists on the importance of pathogens in marine macroalgae, and none are known for coralline algae.

CLOD (coralline lethal orange disease) is similar to the coral-banding diseases in that the pathogen occurs as a one-to-two-centimeter side line or front that moves across the host and leaves completely dead skeletal-carbonate behind. Because of the critical role played by coralline algae in forming reef rims throughout the Indo-Pacific and because reef-building coralline algae extend to much greater latitudes and depths than reef-building corals, CLOD has the potential to change the ecology and building processes of Pacific reefs.

CLOD was initially discovered on southwest Aitutaki Island, Cook Islands, where it occurs from the back reef flat through the barrier-reef ridge and throughout the fore-reef spur and groove to a depth of thirty meters. CLOD attacked all twelve of the coralline species tested, including all of the species depicted in this article, but no members of the closely related non-coralline genus *Peyssonnelia*. The disease first appears as conspicuous bright orange dots that spread to become narrow circular rings (up to thirty centimeters in diameter) but can also develop unidirectionally in fan shapes or in

BY DIANE S. LITTLER & MARK M. LITTLER

ever-broadening branches. The skeletal carbonate remains of the dead host corallines are bleached white, and the upper surfaces begin to flake off following death. When the alga is totally consumed, the advancing band of CLOD forms upright filaments and globules, similar to those of terrestrial slime molds.

Transmittal of CLOD by contact: Photo taken three days after the infected fragment of *Porolithon onkodes* on the left had been placed in contact with an uninfected fragment.

The disease occurred at all study sites on Aitutaki Island, Rarontonga Island (Cook Islands), and Great Astrolabe Reef (Fiji) in 1993. Dr. Peter Craig (Chief Biologist, Department of Marine and Wildlife Resources) recently submitted samples to us documenting that CLOD is now very common on reefs in American Samoa. CLOD was not noted in Fiji by our group of four divers in 1992, although coralline algal populations were intensively and extensively investigated at the identical sites revisited during 1993 and 1994. By 1994, CLOD was present at 100 percent of the fifty sites studied in Fiji with about double the percent cover observed during 1993. In 1994, studies were also initiated in Papua New Guinea (PNG) and the Solomon Islands, where CLOD occurred at 83 percent of the PNG study sites and at 31 percent of the Solomon Islands sites. From our observations and field experiments, it seems that after the CLOD infection has destroyed much of the corallines on the reef, the community shifts from coralline algae and corals to a domination by small turfs of fleshy algae.

The CLOD disease is highly infectious. We placed experimental fragments of CLOD-infected coralline algae in contact with uninfected fragments, and within one day all of the uninfected individuals became infected. Isolated globules that formed after the host plant was totally consumed were placed on surfaces of healthy coralline plants and were also 100 percent infective and began to cause bleaching (death) of the host within about one day, confirming the globules' role as propagules or "seeds" for dispersal.

The CLOD pathogen has not been previously reported, although it is neither easy to overlook nor rare. The answer may be the following possibilities: (1) the disease has been around for decades or centuries but was uncommon until it recently evolved a highly infectious nature and rapid distribution, (2) it has just been introduced into Pacific reef systems from some restricted obscure location, (3) it was formerly so rare or short-lived that no one has noticed it, or (4) it was always widespread but has gone unnoticed.

Given the extensive research on Pacific reefs and the fact that coralline hosts are so abundant and widespread, the fourth hypothesis is unlikely. The first and second hypotheses combined warrant consideration in light of contemporary theory and evidence, which states that natural selection acts on an infectious organism to maximize its "fitness" (in the scientific sense; for example, the ability to survive into succeeding generations)as measured by the number of hosts infected. Virulence, that is,

CLOD attacking the jointed upright coralline *Amphiroa* sp.

the harming of a host, is of little consequence to the disease, if the disease (for example, CLOD) can infect increased numbers of hosts (for example, corallines) in the process. High virulence is therefore extraordinarily likely to evolve whenever a disease gains access to an abundance of new hosts. Crustose coralline algae are among the

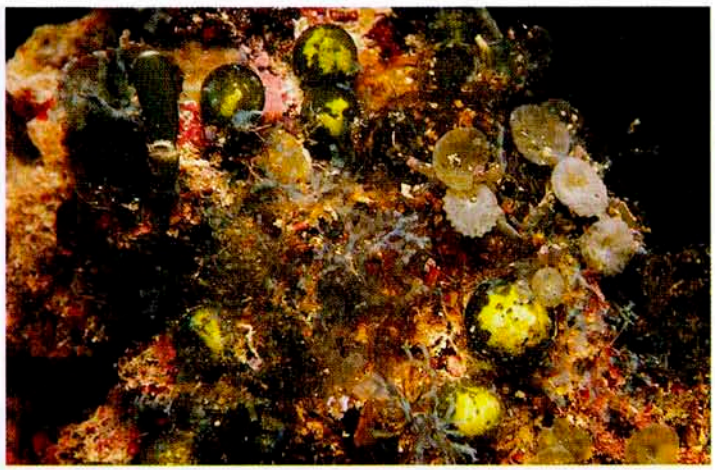
most abundant and widespread space occupiers of marine shallow-water hard substrata worldwide, and this pattern has persisted for millions of years. On the Great Astrolabe Reef, CLOD has gone from non-existent in 1992 to being present in all sites studied in 1993 and 1994. However, at Nacalevu Reef, CLOD declined during 1993-1994 because the study site no longer had abundant reef-building coralline hosts. Pathogen-induced changes in population and community structure may be more common on reefs than suspected, but overlooked or attributed to other causes if the growth rate and successional events are extremely rapid or restricted to small dense patches of host taxa. In other words, events may happen so rapidly on such a scale that no one notices.

Although the overall abundance of CLOD is low, its infective properties, growth rate, and dispersal potential are substantial. CLOD may still be in an early state of increasing dispersal and virulence. The lateral spreading rate of the infection within a continuous patch of coralline crusts is rapid (up to 2.9 millimeters per day) compared with the growth rates of other sessile occupiers of space on reefs (for example, coralline algae, corals, sponges, or fleshy algae which grow several millimeters per year).

The growth habit of active coralline lethal orange disease, showing two contiguous patches of living *Porolithon onkodes* at top and to the right of the band and dead skeletal material below.

Recovery from CLOD seems limited to thin regrowths of the coralline from refuge holes in the host. This escape was observed in numerous cases. Another coralline algal escape mechanism is very high wave-energy that appears to inhibit attachment of CLOD propagules. This latter factor may be a life-saving advantage for algal ridges or reef rims which protect many Pacific islands and atolls. □

Thin regrowths of *Porolithon onkodes* recovering from isolated depressions (refuge pits) following infection by CLOD. The only other refuges from CLOD appear to be areas of high wave energy and possibly deep or shaded habitats with extremely low levels of light.



Algal turf community consisting of a diversity of very small fleshy algae that overgrow dead coralline algae (and, subsequently, live corals) following attacks of CLOD.

This article was abstracted from a detailed three-year study by the authors published in Science, *Vol 267, pp. 1356-1360 (1995). Funding was provided by MARPAT Foundation, the Christensen Research Institute, and the Smithsonian Marine Station at Link Port, Florida (Contribution No. 379).*

Littler, D.S. and M.M. Littler. 1995. A New Bacterial Diesease Impacts Pacific Coral Reefs. *Ocean Realm*, 1995 (September): 37-39, 5 photographs.