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Tetracycline reduces sedimentation damage to corals

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Abstract

Sediment deposition on coral reefs occurs naturally and is also caused by man-made disturbances such as dredging; it can result in the death of scleractinian corals by an unknown mechanism. Sedimentation experiments with corals were carried out in El Nido, Northern Palawan, Philippines, in 1986, and in Honolulu, Oahu, Hawaii in 1988. Four species of Indo-Pacific reef corals (*Oxypora glabra, Montipora verrucosa, Porites lobata, Pocillopora meandrina*) were subjected to sedimentation tests with and without the antibiotic tetracycline to investigate the possible role of microorganisms in the process of sedimentation damage to corals. *O. glabra, Porites lobata* and *Pocillopora meandrina* were rapidly damaged and *O. glabra* was always killed by sedimentation. *Montipora verrucosa* was not injured and may be physiologically resistant to sedimentation damage. Tetracycline-treated seawater reduced the rate of tissue necrosis and prevented colony mortality, suggesting that tetracycline-sensitive bacteria are involved in the process of tissue necrosis and may be partially responsible for coral mortality following sediment deposition.

Introduction

In the tropics, pollution of coastal marine environments by increased sediment input from terrestrial sources can damage economically important coral reef resources (Hodgson and Dixon 1988). Darwin (1842) originally suggested that sedimentation limits coral-reef growth. Since then, research has focused on identifying coral self-cleaning mechanisms, e.g. mucus secretion, ciliary action and polyp movement (Marshall and Orr 1931, Hubbard and Pocock 1972, Bak and Elgershuizen 1976), and measuring survival time of corals buried under a thick layer of mud (Mayer 1918, Edmondson 1928, Marshall and Orr 1931, Rice 1985). Sedimentation-tolerance tests (as opposed to burial) have been conducted with 11 of the approximately 90 Atlantic coral species (Kolehmainen 1974, Rogers 1977, 1979, 1983, Rice 1985) and 12 of the approximately 500 Indo-Pacific species (Mayor 1924, Edmondson 1928, Marshall and Orr 1931, Parnrong and Chansang 1986), but the cause of coral tissue death following sediment deposition is not known.

Based on field and laboratory observations as well as some experimental work, a variety of factors acting alone or in concert has been proposed as the cause of coral tissue death following sedimentation, however, none has been confirmed. These factors include abrasion by sediments carried by strong currents (Levin 1970, Rogers 1977, Randall and Birkeland 1978, Liew and Hoare 1979), light reduction (Edmondson 1928, Yonge et al. 1932, Rogers 1977, 1979, Parnrong and Chansang 1986), smothering by physical blocking of oxygen-carrying water currents (Yonge et al. 1932, Parnrong and Chansang 1986) microbial action (Rogers 1977, 1979, Maragos 1972, Lasker 1980, Parnrong and Chansang 1986) and energy drain due to self-cleaning efforts (Hubbard and Pocock 1972, Maragos 1972, Loya 1976, Rogers 1977, Aliño 1983, Burns et al. 1984).

I carried out experiments in the Philippines and Hawaii to test the hypothesis that one of these factors, microbial action, is involved in the process of tissue necrosis following sediment deposition on reef corals.

Materials and methods

Oxypora glabra is a plate-forming coral that was rapidly damaged by sedimentation in tolerance experiments carried out in the field and in aquaria in El Nido, Palawan Island, Philippines (Hodgson 1989). In 1986, colonies of *O. glabra* were collected from 3 m at a pristine reef and four each were placed in 40-liter aquaria. *O. glabra* colonies held in aquaria were exposed to sedimentation for 7 d in an experiment to determine whether tetracycline-treated seawater (1, 10 and 100 mg l^{-1}) affects the outcome. Tetracycline is a broad-

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spectrum, water-soluble antibiotic active against gram-negative and gram-positive bacteria. Fifty grams of dried, fine, marine sediment was added to treatment aquaria and vigorously stirred twice a day at 8:00 and 18:00 hrs, resulting in sediment deposition of 40 mg cm^{-2}. Every second day, one-half of the water was siphoned from the top of each aquarium and replaced with fresh tetracycline-treated seawater; airstones were used to provide oxygen.

A second series of sediment and tetracycline tests was carried out in 1988 in Hawaii using three common coral species (*Montipora verrucosa, Porites lobata, Pocillopora meandrina*) collected from Kaneohe Bay, Oahu. Both *M. verrucosa* and *Porites lobata* have a wide range of growth forms. Plate-shaped colonies were collected and broken into several pieces of about 100 to 200 cm^2. *Pocillopora meandrina* is a branching species; branch clusters were broken from large colonies. The experiments were performed in 665-liter black-plastic tanks with concrete bottoms (area 9 500 cm^2). Ten colonies of each of the three species (30 in total) were placed in each tank. Colonies were allowed to heal and adjust to tank conditions with flow-through seawater for 5 d, then the water was turned off, leaving an airstone to supply oxygen.

Tetracycline was added to treatment tanks to a concentration of 10 mg l^{-1} based on the results of the previous work in the Philippines. Three hundred grams (dry weight) of fine, freshwater-washed, marine sediment was added to each tank and stirred, producing a concentration of 0.5 g l^{-1} and deposition of 30 mg cm^{-2}. The water was stirred to resuspend the sediments once a day at 09:00 hrs. Every second day, one-half of the water was siphoned from the top of each tank and replaced with fresh tetracycline-treated seawater. The experiment was run twice for 10 d.

Results

The process of sediment deposition and the progression of tissue damage to corals that were injured was the same in the Philippines and Hawaii; therefore, these events are described qualitatively first, followed by quantitative results for the two separate experiments.

The shape and slope of the coral surface affected sediment deposition. When sediment fell on a coral colony, it tended to lodge on relatively flat or concave areas. Areas which were markedly convex, such as pinnacles or thin branches, did not generally accumulate sediment. Since water currents were not powerful enough to remove sediment, a sedimentation rate greater than the biological cleaning rate resulted in sediment buildup.

Following sediment deposition, the entire coral surface was covered with a thin layer of gray-brown sediment. Corals repeatedly cleared off sediment between stirring periods. When a thick layer of sediment built up or became lodged in depressions, or the tissue was damaged, the colony was often not able to clear off the sediment.

The first sign of tissue damage was "bleaching" (loss of zooxanthellae) followed by extrusion of white mesentarial filaments. Coral tissue changed from a firm and even color, becoming puffy and mottled, often with 1 mm diam holes in the tissue. As necrosis progressed, tissue broke up into strands, leaving bare white skeleton exposed. Sediment overlying damaged coral tissue changed color, became dark, grayish-black and gave off an odor of hydrogen sulfide. A network of microscopic filaments formed over damaged areas. When sediment was washed away from these patches, tissue necrosis was clearly visible. Patches of tissue necrosis slowly grew in size under the sediment layer. Tissue necrosis was not contagious and did not spread to areas that, due to morphology, were not covered by sediment (e.g. small convex knobs) unless the entire colony was near death. No cases were recorded of tissue necrosis originating at an edge of a broken coral fragment. Necrosis always began on a flat or concave sediment-covered surface. Few colonies survived after a large portion of their surface area (>80%) was damaged, even if sedimentation was stopped and clean water was added.

Philippine coral

When sediment was added without tetracycline, all *Oxypora glabra* colonies developed patches of tissue necrosis by Day 2, and died within 7 d (Fig. 1). Tetracycline-treated seawater alone had no effect on *O. glabra* coral tissue at 1 and 10 mg l^{-1} (data not plotted). The final results of the sediment with 100 mg l^{-1} tetracycline treatment were not significantly different from the results of the 100 mg l^{-1} tetracycline without sediment treatment; in both cases, damage was significantly greater than in all other treatments except sediment without tetracycline ($p<0.05$; Tukey test of means). Percentage tissue necrosis was significantly greater in the sediment without tetracycline treatment than for all others ($p<0.05$). Mean percent damage in 1 and 10 mg l^{-1} tetracycline treatments with sediment was not significantly different from 0% damage ($p<0.05$). Tetracycline prevented whole-colony death for 7 days at all three concentrations.

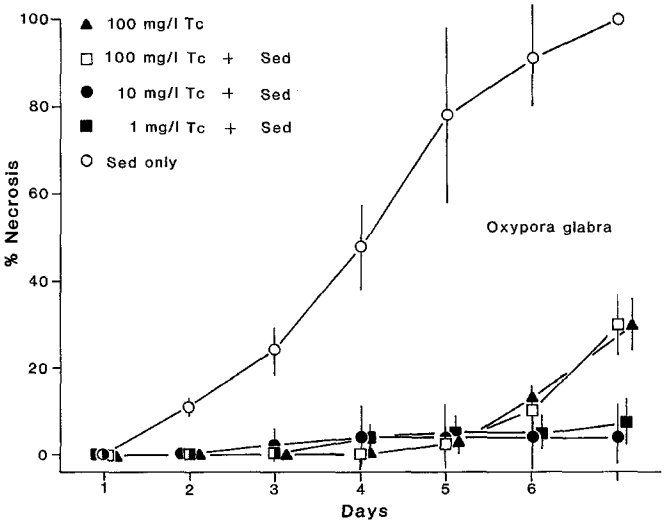

Fig. 1. *Oxypora glabra.* Mean percent tissue necrosis ($n=8$) following exposure to specified concentrations of sediment (Sed) and tetracycline (Tc) for 7 d. Error bars are ±1 standard deviation

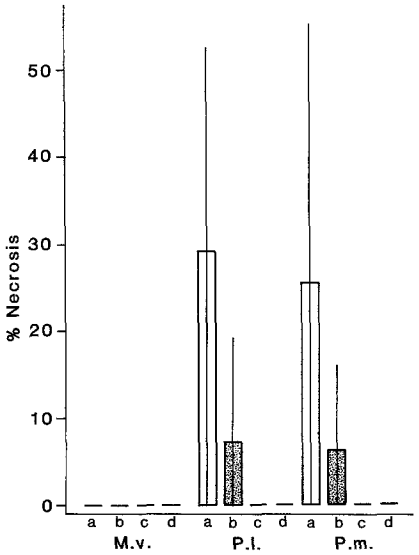

Fig. 2. *Montipora verrucosa* (M. v.), *Porites lobata* (P. l.), and *Pocillopora meandrina* (P. m.). Mean percent necrosis in colonies ($n=30$ per treatment) following exposure to combinations of tetracycline and sediment. a: sediment; b: sediment and tetracycline; c: seawater; d: seawater and tetracycline. Error bars = 1 standard deviation

Hawaiian corals

No Hawaiian coral colonies were damaged following exposure to 10 mg l^{-1} tetracycline treatment without sediment for 10 d (Fig. 2). For both *Porites lobata* and *Pocillopora meandrina,* mean percentage damaged area in the sediment-only treatment was significantly greater than the area in the sediment with tetracycline treatment, plain seawater and tetracycline-treated seawater (Student's *t*-test; $p<0.05$). For *Porites lobata* and *Pocillopora meandrina*, the results of the tetracycline plus sediment treatment were not significantly different from the control (Student's *t*-test; $p>0.05$). No test corals were killed. *Montipora verrucosa* was not damaged in any treatments.

Discussion and conclusions

Observations of corals subjected to sedimentation in aquaria and the field, as well as previous studies (e.g., Marshall and Orr 1931, Hubbard and Pocock 1972, Mitchell and Chet 1975, Dyer 1986) indicate that sediment deposition is affected by physical and biological factors. Physical factors include sediment characteristics, concentration and fall-velocity of sediment particles, and geometry of the coral-colony surface. In addition, sediment deposits may be physically removed by water currents and biological processes described in the "Results" section. Sediment build-up occurs when the deposition rate exceeds the rate of removal.

A sediment layer is required before tissue necrosis begins. Although no colonies that continuously cleared their surfaces were visibly damaged, other potential deleterious effects such as reduced energy levels and reproductive output were not assessed.

Tetracycline-sensitive microorganisms increased the rate of coral-tissue damage and mortality due to sediment deposition. The fact that tissue necrosis only occurred in areas directly underlying sediment deposits and did not occur at the edges of recently broken fragments indicated that initiation of necrosis was dependent on the sediment matrix. However, the fact that the necrotic area of *Oxypora glabra* colonies after 7 d of sedimentation in 1 and 10 mg l^{-1} tetracycline-treated seawater was not significantly different from 0% suggests that sediment deposition by itself, that is without the involvement of tetracycline-sensitive microorganisms, may not be harmful to some coral species over this time interval.

The sequence of deterioration of coral tissue and changes in the sediment layer were indistinguishable in the field (Hodgson 1989) and in aquaria, but damage occurred more quickly in aquaria. At a field sedimentation rate of 20 mg cm^{-2} d^{-1}, damage to *Oxypora glabra* began at 14 wk and there was no mortality (Hodgson 1989). One explanation for this disparity could be that the higher sedimentation rate used in aquaria (40 mg cm^{-2} d^{-1}) was above the tolerance threshold for this species whereas the lower field rate was closer to it. Alternatively, it is possible that some factor associated with aquaria, e.g. water-fouling, enhanced damage rates.

The reduced rate of tissue necrosis following sedimentation in antibiotic-treated seawater generates questions concerning the mechanism of coral necrosis. For example, what is the origin of microorganisms involved? Do bacteria breed rapidly in the sediment matrix using coral mucus as a food source? Does low dissolved oxygen, hydrogen sulfide or some other bacterial product kill the tissue? Is there a sequential succession of microorganisms that take part in the tissue-necrosis process?

Bacteria are ubiquitous in the marine environment and bacterial production rates on coral reefs have been measured in the water column (Moriarty et al. 1985a), in sediments (Sorokin 1973, 1978, Burns et al. 1984, Moriarty et al. 1985b) and in coral mucus (Mitchell and Chet 1975, Paul et al. 1986). Bacteria involved in coral-tissue necrosis following sediment deposition could come from any or all of these sources.

Purified coral mucus is low in caloric value (3.95 to 5.2 cal mg^{-1} dry weight), and in one species (*Fungia scutaria*) has a lipid:protein:carbohydrate ratio of approximately 3:35:62, and contains at least 17 amino acids (Krupp 1982). *Vibrio alginolyticus* comprised 20 to 30% of the bacterial community residing in *Porites astreoides* mucus (Ducklow and Mitchell 1979). Two other potentially pathogenic bacteria associated with coral mucus are *Beggiatoa* sp. and *Desulfovibrio* sp. (Mitchell and Chet 1975).

Some evidence suggests that sediment, especially after forming a depositional layer, may stimulate population growth of pathogenic bacteria that are benign at low densities. When bacteria and sediment are mixed in seawater, chemical and physical forces enhance microbial adhesion to sediment particles (Egan 1987). A number of studies have suggested that respiration and reproduction increase once

microorganisms become attached to sediment particles (Egan 1987). When sediment particles have formed a macroscopic deposition layer, rapid microbial population growth leads to development of irreversibly bound biofilms (Egan 1987). In this way, sediment may provide a substrate for increased activity and population growth that can lead to successful invasion of host tissues (Mitchell and Chet 1975).

The physiological response of corals to infection has not been studied in detail. Extracts of several species of gorgonians show high antimicrobial activity, while none is found in three Caribbean scleractinians (*Acropora palmata, Porites porites*, and *Montastrea* sp.) (Burkholder and Burkholder 1958). During field surveys in Hawaii, many *Montipora verrucosa* colonies were observed with substantial sediment accumulations that appeared to have remained in place for several weeks without causing tissue damage. *M. verrucosa* may possess biological resistance to infection by microbes or some other biological or physical factor which prevents tissue damage.

In contrast to the conclusions of previous workers regarding the importance of biological cleaning mechanisms (Hubbard and Pocock 1972), some corals may depend on cleansing water currents and physiological resistance to infection to survive sedimentation damage. Interspecific differences in physiological resistance to infection by tetracycline-sensitive bacteria may be the most important determinant of sedimentation tolerance of reef corals.

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