WHITE-BAND DISEASE IN ACROPORA PALMATA: IMPLICATIONS FOR THE STRUCTURE AND GROWTH OF SHALLOW REEFS

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In the last two decades a wide variety of organisms has been implicated in the destruction of reef-building corals (Glynn, 1973; Endean, 1976; Antonius, 1977). These fall generally into three categories: predators (Endean, 1973; Bak and van Eys, 1975; Reese, 1977), competitors for substrate (Glynn, 1973; Lang, 1973; Gladfelter et al., 1978) and disease-causing organisms (Garrett and Ducklow, 1975; Mitchell and Chet, 1975). In most instances the known impact of such organisms is restricted to portions of, or at most, single coral colonies. The impact of such organisms on whole reefs or systems has been documented only for the predatory starfish Acanthaster planci (Endean, 1973). In the present study we document the impact of another agent on a reef-wide scale.

In much of the Caribbean Sea shallow windward reefs are dominated at depths of 1 to 5 m or more by the large branched coral Acropora palmata (Adey and Burke, 1976; Adey, 1978). In some such reefs >99% of living coral surface belongs to this species (pers. obs.). In addition to quantitative dominance on such reefs this coral has one of the greatest rates of deposition of CaCO$_3$ per unit tissue surface (Gladfelter et al., 1978; Gladfelter and Gladfelter, unpublished) as well as high linear growth rates (5-10 cm/yr, Gladfelter et al., 1978) and consequently healthy A. palmata reefs exhibit some of the greatest measured reef growth rates (Adey and Burke, 1976 and unpublished calculations by Gladfelter and Gladfelter of 10.3 kg CaCO$_3$/m$^2$/yr). Furthermore, the high structural complexity of A. palmata dominated reefs results in fish communities of greater diversity than in other reefs in comparable areas (Gladfelter and Gladfelter, 1978).

Throughout much of its range, A. palmata is subject to a necrosis which can cause extensive local mortality of the coral. I have observed this necrosis (= "white band disease") in the northeastern Caribbean Sea (Virgin Islands, St. Marten, Antigua), Curaçao, Nicaragua (Miskito Cays), and it has been observed in Panama (P. Glynn, pers. comm.) and south Florida (A. Antonius, pers. comm.). The incidence (proportion of colonies affected in a small area) of this disease varies greatly within its range and cannot presently be positively correlated with any man-induced or natural factors: the area of highest observed incidence of this disease is on the seaward reef front off northeastern St. Croix, Virgin Islands, which is nearly unaffected by any form of man-induced disturbance. At the time of this study the affected area was about 5 hectares and was noticeable in low level aerial photographs (Fig. 1). The disease is characterized by a sharp line of advance where the distally located, brown zooxanthella-bearing coral tissue is cleanly and completely removed from the skeleton, leaving a sharp white zone about 1 cm wide that grades proximally into algal successional stages. Microscopic examination of a number of affected branches revealed no consistent possible causative organism. However, current work by E. Peters (pers. comm.) suggests that bacteria might be responsible. The live zooxanthella-bearing coral tissue, which is concentrated in longitudinal strips between skeletal costae, peels off the skeleton, curls into little balls, and is carried away by its own ciliary action, often in strands of mucus. In all instances the disease progressed from a basal shaded portion of the colony toward the tips of branches.
Series of in situ photographs were taken to study the course of the white band disease on a section of the bank barrier reef of northeastern St. Croix, Virgin Islands, beginning November 1976. Photographs taken at 12-h intervals near sunrise and sunset revealed no difference in the rate of advance of white band necrosis between day and night. Rates of advance determined on the basis of semi-monthly photographs made over 3 months were 5.5 mm/d (SD = 2.6 mm/d, \( n = 83 \); range = 0.8–13.9 mm/d, no statistically significant difference between months). In all but one of the original 45 branches photographed, the disease eventually progressed to the tip of all branches destroying the entire colony. In photos taken of whole colonies (Fig. 2) the disease progressed to completion in 23 of 24 whole colonies and on all but one branch of the remaining colony. In the latter case some recovery was observed as the remaining coral tissue deposited a lip of carbonate and grew proximally as a crust.

The overall impact of the disease on a heavily affected reef was followed over an 18 mo period (Table 1) by taking a series of overlapping color photos (ca. 2 × 3 m) along three 50-m transects (yielding three 3 × 50-m quadrats) in October 1977 and again in April 1979. The resulting photos were analyzed under the dissecting microscope with the aid of a 10 × 10 ocular grid. At the end of this period a mean of 9 new colonies were infected per 150-m² transect. However, the mechanism of spread is unknown and although it seems to be contagious within small areas of reef, newly infected colonies are not necessarily adjacent to previously diseased ones, and healthy colonies immediately adjacent to diseased ones do not necessarily develop the infection (Fig. 2). The overall decrease in
Figure 2. Underwater photographs of two *A. palmata* colonies, one infected with white band disease, the other healthy, taken over 8-mo period: 1A taken 9/25/78, 1B taken 11/7/78, 1C taken 5/21/79. a, line of necrosis of coral tissue (progressing distally); b, healthy colony, adjacent to infected colony but unaffected by disease; and c, large carnivorous gastropod on algal-covered dead skeleton.
Table 1. Impact of white band disease on three reef (150-m²) quadrats over 18-mo period

<table>
<thead>
<tr>
<th></th>
<th>MeanProjected Surface* Area (m²)</th>
<th>No. Colonies</th>
<th>Δ18 Mos</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10/77</td>
<td>4/79</td>
<td></td>
</tr>
<tr>
<td>Total A. palmata colonies</td>
<td>131 (118-138)</td>
<td>124† (116-136)</td>
<td>-7 (39-51)</td>
</tr>
<tr>
<td></td>
<td>87%</td>
<td>83%</td>
<td></td>
</tr>
<tr>
<td>Healthy colonies</td>
<td>70‡</td>
<td>46</td>
<td>-24</td>
</tr>
<tr>
<td></td>
<td>47%</td>
<td>31%</td>
<td>-16%</td>
</tr>
<tr>
<td>Diseased colonies</td>
<td>61§</td>
<td>78</td>
<td>+17</td>
</tr>
<tr>
<td>Dead colonies</td>
<td>41%</td>
<td>52%</td>
<td>+11%</td>
</tr>
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* Multiple coral strata not considered in projected area values.
† Reduction due to collapse of a few colonies.
‡ Values for total coral tissue on healthy and diseased colonies.
§ Values for total dead (i.e., coral tissue removed) surface on diseased and dead colonies.

Live coral tissue during this time was 16% while the increase in dead standing colony surface was 11%. The difference was due to the collapse of a few dead colonies during this period. This represents a substantial decrease in CaCO₃-depositing tissue. The impact of such an agent of mortality on the health of this coral community as well as on reef growth is thus potentially catastrophic. Other than encrusting algae other reef builders (i.e., scleractinians and Millepora) did not increase noticeably during the course of the study. Since this "epidemic" was probably occurring several years prior to this study, the large number of dead colonies at the start was probably a result of the disease.

As the disease progressed outward on a coral colony a successional sequence of colonizing organisms ensued. Coral colonies in an advanced state of disease thus exhibited successional sequences of up to 1 yr or more. Except for a few cm immediately proximal to the line of tissue necrosis which typically teemed with small invertebrates (ciliates, copepods, annelids, nematodes) the remainder of the skeleton was covered predominantly with red algae, normally filamentous forms up to about 1 yr followed by crustose corallines in increasing abundance thereafter. Within a year, increased numbers of herbivorous gastropods were evident. Carbonate borers (especially clionid sponges), which are rarely if ever present in skeletons of A. palmata beneath live coral tissue, became increasingly prevalent in older dead branches, thus weakening their structure. During periods of storm swell, it was these branches (and colonies) that generally toppled first (Table 1).

In summary the overall changes brought about on this reef due to the white band disease-induced decrease of Acropora palmata were: (1) a decrease in structural complexity of the reef surface; (2) a decrease in live coral tissue and hence (3) a reduction in carbonate deposition rate for the reef; (4) an increase of both filamentous and crustose algae, possibly leading to increased productivity (Rogers and Salesky, 1981); (5) an increase in abundance and diversity of small invertebrates including scavengers on necrotic coral tissue, borers and herbivorous gastropods. As a result of these changes I also predict a change in fish community structure associated with the reef correlated with changes in reef structure and available forage (Gladfelter and Gladfelter, 1978).
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Literature Cited


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ON THE GROWTH OF A LABORATORY-READED SEA URCHIN, DIADEMA ANTILLARUM (ECHINODERMATA: ECHINOIDEA)

John C. Bauer

The sturdy and ubiquitous sea urchin Diadema antillarum Philippi has demonstrated a consistent pattern of growth in previous laboratory and field studies (Randall et al., 1964; Lewis, 1966; Bauer, 1976). A rare opportunity to determine growth under controlled conditions arose when two juvenile Diadema with test