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Coral disease outbreak in the Florida Keys: Plague Type II

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Abstract: A coral disease characterized by a novel pattern of rapid tissue destruction first appeared on reefs of the middle Florida Keys in June 1995. Between June and October 1995 the disease infected 17 species of scleractinian corals and the hydrocoral *Millepora alcicornis*. Localized populations of *Dichocoenia stokesi*, the species most affected, revealed up to 38% mortality. Many colonies exhibited complete tissue loss within days as the disease moved across colonies at rates of up to 2 cm per 24 hr. Typically tissue loss was initiated at the base of the colony and moved upward. At times disease progression halted and colonies retained partial tissue resembling a cap on the top of an otherwise denuded colony. Laboratory cultures of samples from the disease line revealed a dominant bacterium that, when isolated and characterized using genetic and metabolic techniques, most closely matched the genus *Sphingomonas*. Pure laboratory cultures of the bacterium produced disease in freshly collected coral colonies incubated in laboratory aquaria. The disease that we call plague type II appeared on different reefs of south Florida and the Florida Keys in 1996 and 1997. While coral mortality associated with each of the three outbreaks was regionally confined and did not recur in subsequent years on the same reefs, the high mortality rates distinguish this disease as one of the most serious yet documented.

Key words: Coral disease, coral reef health, *Dichocoenia stokesi*, invertebrate pathology, *Sphingomonas*.

In June of 1995 a new disease of scleractinian corals appeared on Alligator Reef in the central Florida Keys. Diseased colonies exhibited freshly exposed skeleton extending from the base of the corallum, an unusual pattern of tissue loss. Many colonies were completely free of tissue and displayed freshly exposed, white skeletons suggesting rapid tissue destruction. The new disease primarily affected the elliptical star coral *Dichocoenia stokesi*, a species previously unknown to be susceptible to any disease. Within four months, dead and dying coral colonies with identical signs were prevalent on reefs both north and south of the initial site. In the two years since that time, the disease spread to affect most of the reefs of the Florida Reef Tract. To date there have been three major epizootics in different reef areas of south Florida: the middle Keys in 1995, southern Keys and Dry Tortugas in 1996, and reefs north of Miami in 1997 (Richardson *et al.* 1998).

The disease is different from those previously described in a number of ways, including the coral species affected, associated microorganisms, rate and pattern of tissue destruction, virulence, and short-term effect on coral populations. Despite these novel characteristics we have termed the new disease plague type II based on a similarity in appearance to "plague"

reported on the same reefs in 1977 (Dustan 1977). This paper is a summary of investigations into the biological properties of plague type II.

MATERIALS AND METHODS

Tissue Degradation: Rates of tissue degradation were documented both *in situ* and in the laboratory. *In situ* rate measurements of tissue loss were made using calipers referenced to a nail inserted into exposed coral skeleton immediately adjacent to the disease line. For diseased colonies maintained in laboratory aquaria, the progression of tissue loss extending from the colony base was measured, again using calipers. Measurements were performed during the daytime for *in situ* colonies, and during both day and night for colonies in aquaria.

Microbiology: Microbiological samples were collected underwater using sterile 10 ml syringes equipped with sterile 21 gauge needles. While applying suction the needle tip was run along the surface of the diseased colonies, either directly on the disease line or at distances of 1 cm on each side of the line (i.e. on the surfaces of apparently healthy coral tissue or exposed coral skeleton). Mucous extrusion was not elicited from healthy tissue during sampling. Immediately upon return to shore the syringes were individually vortexed and 1 ml was withdrawn for inoculation of a 10:1 dilution series of filtered, sterilized seawater (final dilution 10^{-10}). Triplicate samples (100 µl) of each dilution were then inoculated onto marine agar plates for counting. After two to three days of incubation (aerobic, room temperature) colonies were counted and characterized by their pigmentation and morphology. Pure cultures were obtained by sterilely picking individual, isolated colonies. An isolate of the characteristic colonies that grew from the disease line was sent to the laboratories of G.W. Smith and K.B. Ritchie for genetic and metabolic-based analyses.

Electron Microscopy: Small colonies of *D. stokesi* were fixed on site in an ice-cold mixture of 2.5% glutaraldehyde and 1% paraformaldehyde in 0.1 M cacodylate in seawater at pH 7.8. No osmication was employed so that the distinction between normal and bleached tissue would remain obvious. A diamond saw was used to cut thin slices of corallum containing both uninfected and infected tissue. Slices were decalcified over a period of several days in 5% EDTA in 50% ethanol changed daily. A microwave set with a temperature maximum of 32° was used to facilitate the decalcification process. After decalcification, tissue was dehydrated in ethanol, embedded in Spurr's resin, stained in uranyl acetate and lead citrate, and examined using a transmission electron microscope.

Microelectrodes: Oxygen-sensitive microelectrodes (outside diameter at tip <50 µm) were used to examine the concentrations and dynamics of oxygen microenvironments associated with a diseased colony of *Dichocoenia stokesi*. Measurements were conducted in a 10 l, continuously aerated aquarium filled with seawater at 33°C. Microelectrode tips were positioned using a micromanipulator. Electrodes were calibrated using air-saturated and N_2-flushed seawater. Electrode voltage was measured using a picoammeter. The application of this technique for the study of coral diseases has been described previously (Carlton and Richardson 1995).

Infection experiments: Disease transmission and infection exeriments were carried out in the laboratory using freshly collected healthy and diseased colonies of *Dichocoenia stokesi*. Experimental colonies ranging in size from six to seven cm in diameter at the colony base were collected on Conch Reef at depths of 10-14 m. In disease transmission experiments healthy corals were placed in 100 l aquaria along with actively diseased colonies. Colonies were not touching, and were a minimum of 10 cm apart. Healthy colonies were visually monitored for disease signs. Additional infection experiments were conducted using pure cultures of a bac-

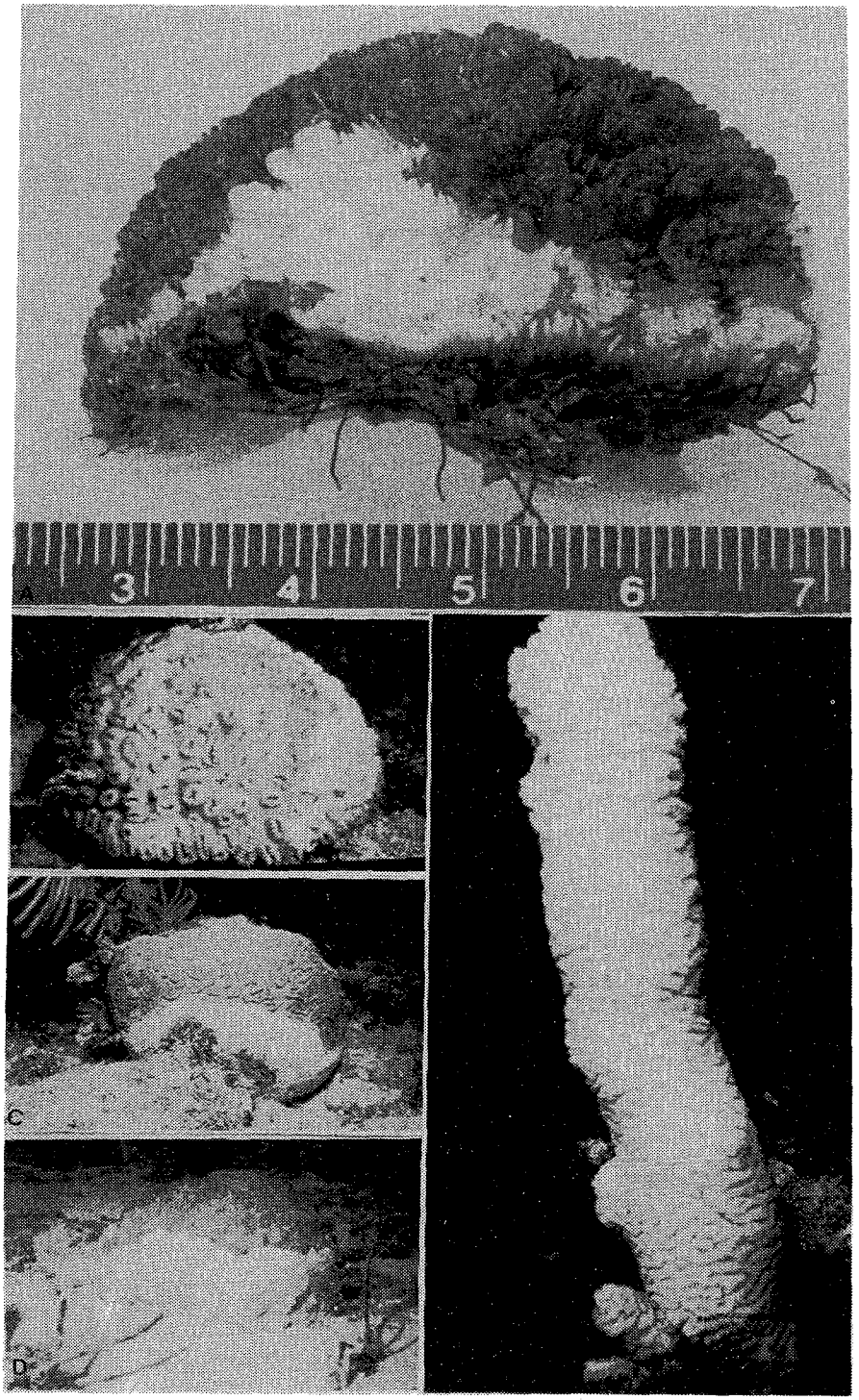

Fig. 1. Coral colonies infected with plague type II. A. Freshly collected *Dichocoenia stokesi* colony. Scale is in cm. B. *D. stokesi* with plague type II tissue loss from two infection points. C. *Diploria labyrinthiformis*. D. *Solenastrea bournoni*. E. *Dendrogyra cylindrus*. Plates b-e were photographed *in situ* at depths between eight and ten m on reefs of the northern Florida Keys.

TABLE 1

Coral species observed to be affected by plague type II on reefs of the Florida Keys from June to October 1995. Susceptibility of these species to black band disease and plague type I is also noted[1]

Plague type II	Black band	Plague type I
Agaricia agaricites		
A. lamarcki		
Colpophyllia natans	+	+
Dendrogyra cylindrus		
Dichocoenia stokesi		
Diploria labyrinthiformis	+	
D. strigosa	+	
Eusmilia fastigiata		
Madracis decactis		
M. mirabilis		
Manicina areolata		
Meandrina meandrites		
Montastrea annularis[2]	+	+
M. cavernosa	+	
Siderastrea siderea	+	
Solenastrea bournoni		
Stephanocoenia michelinii		+

1 Additional species affected by these diseases are not listed here.
2 Species complex.

terium isolated from a coral infected with plague type II. In these experiments, healthy colonies were placed in aerated two l beakers placed within the 100 l aquarium. Coral colonies were placed onto lawns of the bacterial isolate grown on marine agar plates, or onto uninoculated marine agar plates used as controls. Colonies were visually monitored for disease. All transmission/infection experiments were performed using filtered (Whatman #1) surface seawater collected offshore of the northern Florida Keys.

RESULTS

Corals infected with plague type II exhibited an abrupt line separating apparently healthy tissue from exposed coral skeleton (Fig. 1A). Tissue loss typically began at the base of the colony and progressed upward. Usually the initiation of tissue loss occurred at an isolated point on the basal edge of the colony that quickly spread to ring the entire colony base. This area then progressed upward exposing coral skeleton. Less frequently, tissue loss began at more than one point and a more irregular disease progression occurred (Fig. 1B).

During each plague type II disease event the coral species *Dichocoenia stokesi* exhibited the highest disease incidence, with infection of up to 33% of all *D. stokesi* colonies observed on separate reefs at any given time (Richardson *et al.*, 1998). In 1995 coral mortality rates were also documented, with values of up to 38% mortality of *D. stokesi* occurring within survey areas (Richardson *et al.* 1998). A number of other scleractinian coral species were observed on affected reefs with identical tissue loss patterns. While quantitative disease incidence data were not obtained for these additional coral colonies, a total of 17 species of scleractinian corals, plus one hydrocoral species, were observed with identical disease signs during 1995 (Table 1). Three representative examples are shown in Fig. 1 (C, D, E).

Close visual inspection of diseased colonies revealed a very fine but distinct line consisting of a narrow band of bleached coral tissue between normally pigmented tissue and exposed coral skeleton Fig. 2A). The bleached area, approximately two to three mm wide, migrated across coral colonies destroying tissue along the way. The rate of tissue degradation of infected *D. stokesi* was documented both *in situ* and in laboratory aquaria. Rates were highly variable, ranging from a steady progression of tissue loss (e.g. Fig. 2B) to an irregular pattern consisting of intermittent progression and cessation of disease line movement. The maximum rate of tissue loss (two cm/24^{hr}) was the same for infected corals on the reef (Fig. 3A) and in laboratory aquaria. Rates of one cm/12^{hr} were observed both during day and night in aquaria placed outdoors. The intermittent movement pattern was observed both in laboratory and *in situ* colonies. The only pattern observed in the field but not in the laboratory was a permanent, or long-term (days to months) cessation of disease

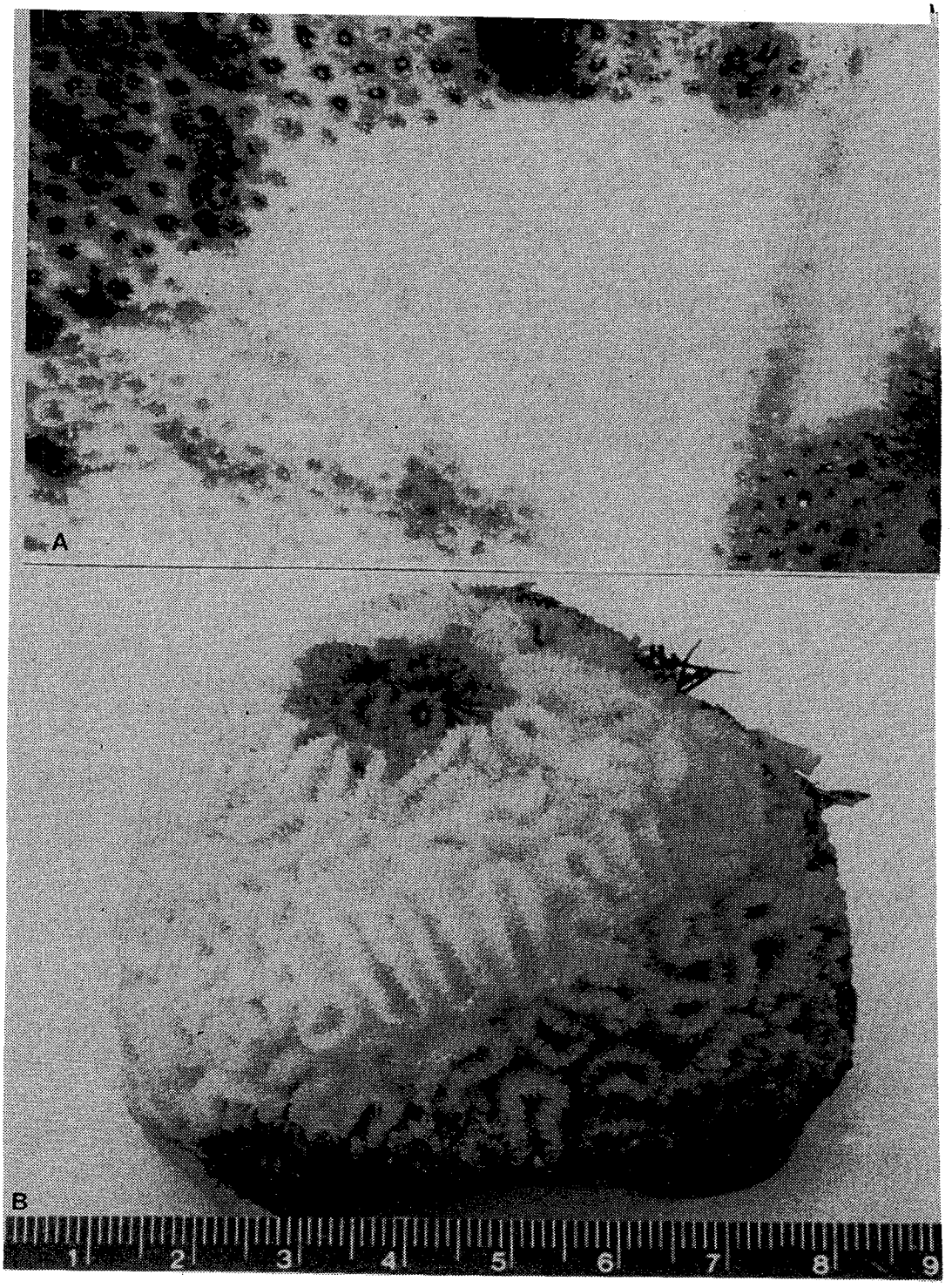

Fig. 2. Plague type II characteristics. a. *In situ* macrophotograph revealing the distinctive line between healthy (pigmented) and bleached coral tissue grading into exposed coral skeleton. This coral colony is the same as shown in Fig. 1d. b. Freshly collected *D. stokesi* revealing typical "cap" of remaining healthy tissue. Such colonies usually exhibit continued tissue degradation resulting in 100% tissue loss. Scale is in cm.

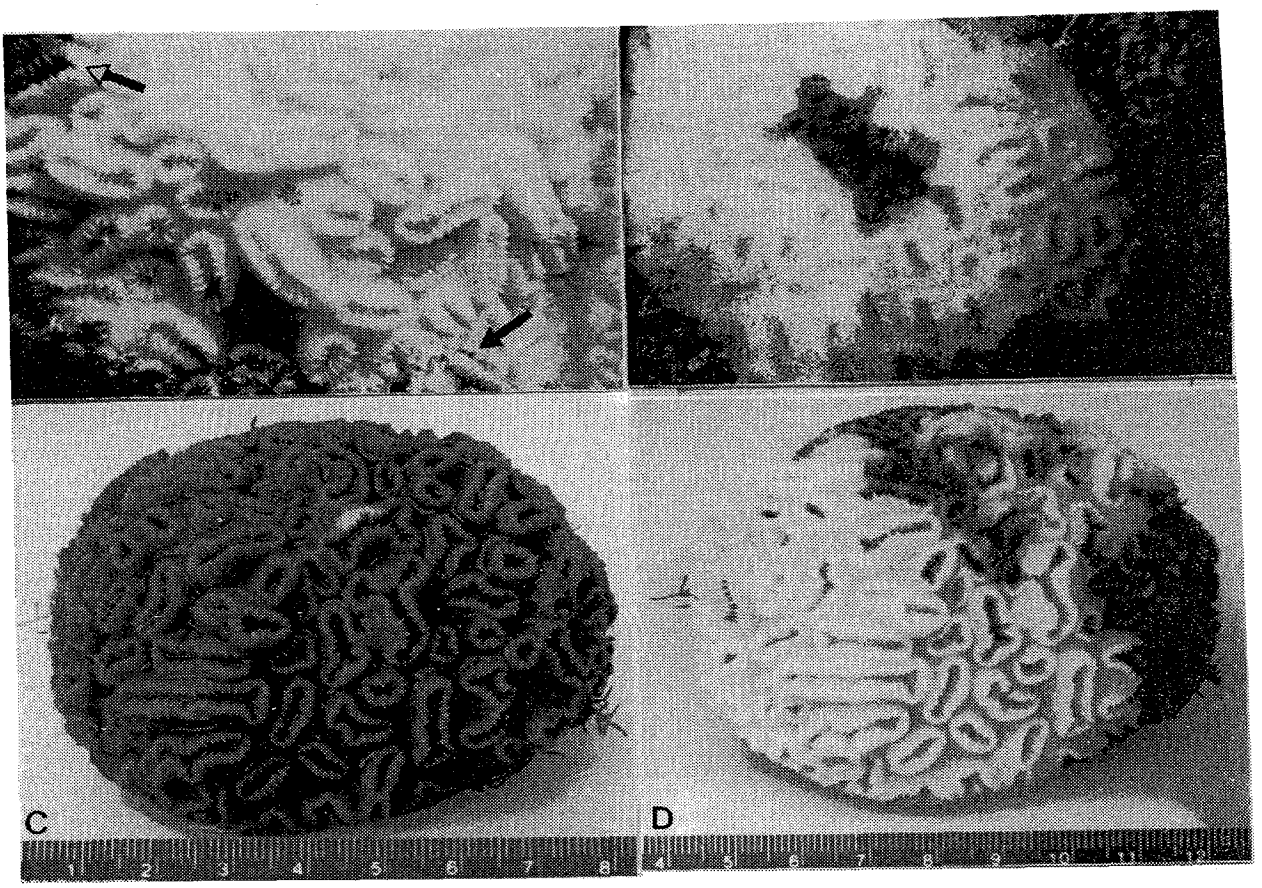

Fig. 3. Tissue loss patterns and infection of *D. stokesi*. a. *In situ* experiment documenting tissue loss progression. The nail (closed arrow) was placed at the edge of the disease line, in exposed coral skeleton, and the colony photographed three days later. The open arrow indicates the new disease line. b. *In situ* photograph of the top of a *D. stokesi* colony with old and new plague type II tissue loss. The small area in the center is the only tissue remaining on this colony. The area outside of freshly exposed coral skeleton is skeleton colonized by microalgae after a previous plague infection. d. Laboratory infection experiment. This freshly collected *D. stokesi* colony was placed on a lawn of the *Sphingomonas* bacterial culture isolated from plague type II. e. The same colony after two days of incubation in a laboratory aquarium.

line progression. In the laboratory all colonies that became infected lost 100% of their tissue, whereas in the field some colonies were observed with healthy tissue adjacent to surface areas with heavy secondary colonization by microalgae. Such areas were present in the pattern typical of skeletal exposure due to plague type II. Fig. 3B shows one such colony. This particular colony also exhibited renewed active disease.

Samples collected from the line of pigmentation dividing healthy and bleached tissue contained a few intact zooxanthellae and numerous small, motile bacteria, while the bleached tissue area contained bacteria and amorphous tissue. Transmission electron microscopy (Fig. 4) revealed that the bleaching was associated with degeneration of zooxanthellae, not simple pigment loss. Sterile samples for microbiological analysis were collected *in situ* (using SCUBA) from three species of corals with plague type II (*Dichocoenia stokesi*, *Dendrogyra cylindrus*, and *Diploria labyrinthiformis*). Plating revealed that these contained varied bacterial populations (i.e. differences in pigmentation and colony morphology) depending on whether the disease line, apparently healthy coral tissue, or exposed coral skeleton was sampled. Samples collected from the disease line of each of the three coral species were uniformly

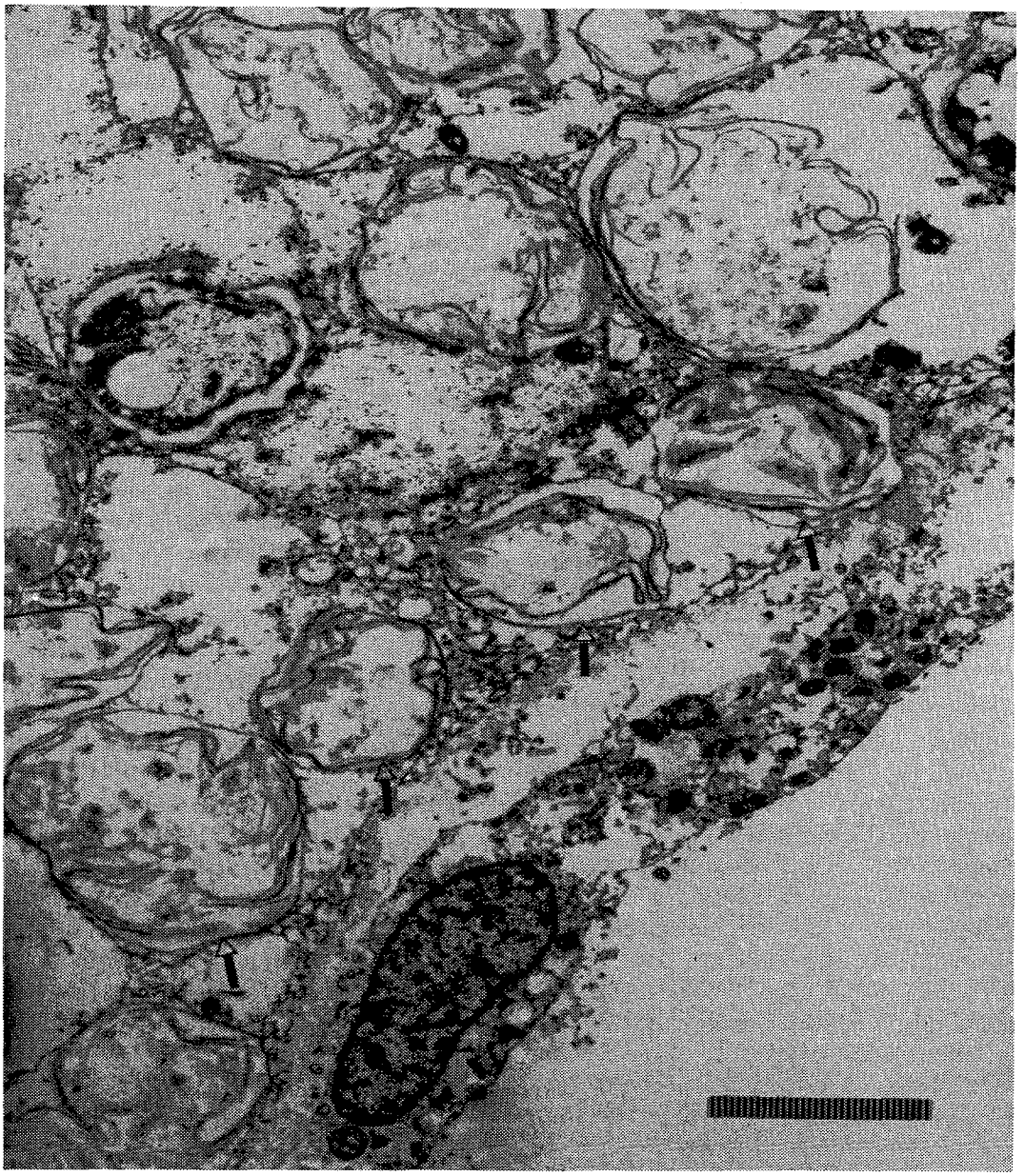

Fig. 4. Transmission electron micrograph of material from the "bleached" tissue line between healthy tissue and exposed skeleton, revealing multiple degenerating zooxanthellae (arrows) in host gastroderm. Scale bar = 5 μm.

translucent, 1.5 to 2 mm in diameter, and became pigmented (yellow) after several days of incubation. Inspection and counting of colonies from inoculated (triplicate) plates revealed differences in concentration of bacteria, with the highest concentrations (10^5cells/ml) growing from disease line samples. Samples from healthy tissue and exposed skeleton to each side of the disease lines yielded 10^3 and 10^4cells/ml respectively. Colonies that grew from the disease line were motile, gram negative rods measuring 1 x 1.5 μm. One typical bacterial colony was selected from a plate inoculated with a sample from a diseased

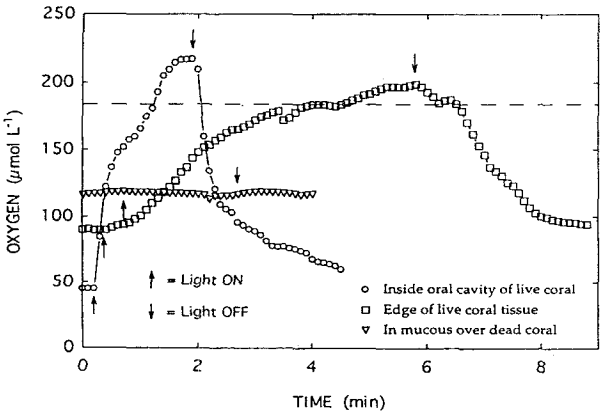

Fig. 5. O_2 concentrations associated with live, diseased, and dead areas of a freshly collected *D. stokesi* colony infected with plague type II. For healthy tissue the electrode tip was positioned inside a coral polyp. Measurements of the disease line were performed at the edge of the pigmented/bleached tissue, at a depth of 100 μm into the tissue. O_2 concentrations of the surface of exposed coral tissue were made ca. 5 mm from the disease line where the bleached tissue area appeared to grade into a thin layer of surface mucous. See text for details.

Dichocoenia stokesi tissue and further analyzed using genetic and carbon source utilization approaches. This isolate was also used for laboratory infection experiments.

Laboratory experiments revealed that the disease is contagious and could be induced by exposure to the plague type II bacterial isolate. In every case (n = 6) healthy colonies placed in aquaria with a diseased colony became diseased, with the typical plague type II pattern of tissue loss, and died within 2 days. In infection experiments, healthy colonies (n = 2) placed on a lawn of the isolate also exhibited plague type II signs and died within two days (Fig. 3 C, D). Control colonies (n = 2) remained healthy.

Oxygen dynamics associated with a freshly collected, diseased *D. stokesi* colony maintained in an aquarium are depicted in Figure 5. Inside the oral cavity of an apparently healthy coral polyp initial oxygen concentration ($[O_2]$) under darkness was 45.2 μM, or approximately 0.25 of the ambient oxgyen concentration of 184 μM. Illumination (285 μmol quanta $m^{-2}s^{-1}$ of photosynthetically active radiation) resulted in an increase in $[O_2]$ to 217 μM in less than two min. Extinguishing the light source resulted in an initially rapid decrease in $[O_2]$ that leveled off after ca. 2.5 min of darkness to approach the initial $[O_2]$ value. At a depth of 100 μm into the disease line tissue (i.e. the interface between pigmented and non-pigmented tissue), initial $[O_2]$ under darkness was 90.5 μM. Illumination led to an increase in $[O_2]$ at a much slower rate than measured inside the apparently healthy coral polyp, and it took ca. five min. for the $[O_2]$ to increase to just above ambient (197 μM). Cessation of illumination resulted in a return to initial $[O_2]$ after three min., again slower than exhibited by the healthy polyp. At a point 5 mm to the side of the disease line (exposed coral skeleton), $[O_2]$ was 117 μM during darkness. At this site changes in illumination did not affect $[O_2]$.

DISCUSSION

Plague type II is different from previously described coral diseases in several respects. A primary difference is the identity of susceptible coral species. The number of species of scleractinian corals (17) we found infected with plague type II is higher than has ever been reported for any other disease outbreak in the Caribbean region (cf., Peters 1993). Black band disease, which infects the second highest number of coral species, has been reported in the literature (and observed by the authors) to infect six species of scleractinians (Rützler *et al.* 1983, Edmunds 1991, Kuta and Richardson 1996, Bruckner and Bruckner 1997). All coral species affected by black band on the reefs of the Florida Keys were also susceptible to plague type II. White band disease, known to have severely affected reefs throughout the Caribbean (Gladfelter 1982, Aronson and Precht 1997) is only known to infect acroporid corals, although Antonius has reported colonies of black band-diseased corals that initially appeared to have white band disease (Antonius 1981a). No acroporids were observed with plague type II signs in 1995, 1996 or 1997 on reefs of the Florida Keys. Three of the 17 species affected by plague type II (*Colpophyllia natans*, *Montastrea annularis*,

and *Stephanocoenia michelini*) were also reported susceptible to plague (type I) on these same reefs (Dustan, 1977). Three other species, however, (*Mycetophyllia ferox*, *M. lamarckiana*, and *Porites astreoides*) susceptible to plague type I were not affected by plague type II.

The pattern of tissue loss that characterizes plague type II is novel. No other known coral disease exhibits tissue loss initiated almost exclusively at the colony base. Rather, the other "line", or band, diseases (black band, white band, red band, etc.) begin on the tops or sides of colonies, commonly producing an expanding or circular pattern of tissue loss on the body of the coral as the disease progresses (Richardson, 1998). The rate of tissue degradation is also novel, with a maximum value (2 cm/d) that is faster than all previously published coral tissue loss with the exceptions of shutdown reaction (Antonius 1981b) and the newly emerging rapid wasting disease (Goreau *et al.*, this volume).

The single dominant microorganism associated with the disease line of plague type II is different from that of other coral diseases. The bacterium isolated in this study was characterized using genetic and metabolic approaches and found to match most closely with the aerobic genus *Sphingomonas* (Richardson *et al.* 1998, Ritchie *et al.* in preparation). Only three other coral diseases (black band, white band type II, and aspergillosis) have been characterized microbiologically. The presence and identity of microorganisms found in black band disease has been known for some time, and includes the cyanobacterium *Phormidium corallyticum*, sulfidogenic bacteria, sulfide-oxidizing *Beggiatoa* spp., fungi, and many heterotrophs. Each of these microorganisms has been proposed as the black band pathogen (e.g. Ducklow and Mitchell 1979, Antonius 1981a, Ramos-Flores 1983, Rützler and Santavy 1983). It is only relatively recently that the highly structured organization of this consortium has been demonstrated (Richardson and Carlton 1993, Carlton and Richardson 1995). It is now known that the black band community is a laminated microbial mat community such as those found in illuminated, sulfide-rich benthic environments, and contains a microbially generated oxygen/sulfide interface environment maintained by the metabolism of the consortial members (Richardson 1996, Richardson *et al.* 1997). Rather than a single pathogen, it is the consortium itself that is pathogenic (Richardson *et al.* 1997).

The microbial community of white band type II has also been characterized and the predominant microorganisms identified using a combination of genetic and metabolism-based techniques (Ritchie and Smith 1995, Ritchie and Smith - this volume). These investigators were able to demonstrate the clear dominance of white band type II communities by two species of *Vibrio* (along with assorted heterotrophic eubacteria). They were also able to fully describe the disease state responsible for the recent sea fan epizootic observed throughout the Caribbean (Nagelkerken *et al.* 1997). The pathogen was identified as a species of the primarily terrestrial fungal genus *Aspergillus*, hence the disease has been characterized as a type of aspergillosis (Smith *et al.* 1996, Smith and Ritchie - this volume).

The microbiology of coral disease has only recently been a focus of research. Most earlier efforts were limited to microscopic observations of diseased tissue, as was the case with plague type I (Dustan 1977). Although coral tissue infected with white band type I exhibits 40 μm "packets" of gram negative bacteria, attempts to culture them have yielded extremely variable types of bacteria (Peters *et al.* 1983, Peters 1993). The very recently emerging coral diseases (rapid wasting disease, white pox, yellow band, etc.; see Goreau et al. - this volume) are either in the process of being microbiologically characterized or will be in the near future.

The direct measurement of oxygen concentration associated with diseased coral tissue has only been conducted for black band and plague type II. This approach yields information about the physiological status of biota associated with apparently healthy, actively diseased, and newly killed coral. During darkness the surrounding water is the only source of oxgyen to the coral tissue, zooxanthellae, and associated

microorganisms living on the coral. In the microelectrode studies reported here the [O_2] values recorded during darkness indicate the relative respiratory activities of diseased vs. non-diseased tissue. Apparently healthy coral had the lowest [O_2] during darkness, thus the highest relative O_2 demand. Not surprisingly, the newly dead area (exposed coral skeleton) had the highest O_2 values, thus the lowest oxgyen demand. This area also failed to respond to changes in illumination as would be expected during photosynthetic or aerobic respiratory activity. Actively diseased tissue consumed and produced O_2 during dark and light respectively, however the rates were much lower than for healthy tissue, reflecting decreased metabolic activity. The fact that the diseased tissue contains oxygen corresponds well with the results of the microbiological characterization of the presumed pathogen as most closely related to the aerobic genus *Sphingomonas* (Richardson *et al.* 1998, Smith *et al.* in preparation). The relatively low respiration rates (O_2 uptake) during darkness may be due to the small amount of microbial biomass associated with diseased tissue.

The oxygen microelectrode study also demonstrates that the mechanism of coral death by plague type II is not the same as recently demonstrated for black band-induced coral mortality. Carlton and Richardson (1995) have shown that black band is both anaerobic throughout most of the band and highly reducing due to high concentrations of sulfide. It is, in fact, the microbially mediated microenvironment of sulfide and anoxia that exists at the base of the band that kills coral tissue (Richardson *et al.* 1997). The plague type II pathogen must, therefore, be extremely virulent as it appears not to be present in large enough numbers to deplete O_2 during darkness, yet kills coral tissue at rates twice as fast as black band. Extreme virulence of the plague type II bacterial pathogen is also supported by results of the infection experiments.

Plague type II may very well be a reemergence of plague type I. It is impossible, however, to determine if they are identical diseases with the information available. Microscopic observations of samples collected during both disease outbreaks revealed populations of gram negative rods, accompanied in plague type I by filamentous flexibacteria. Different coral species were affected and tissue destruction rates were very different during the 1977 and 1995-1997 plague outbreaks. These differences, however, may be associated with long-term environmental and coral community changes that have occurred on these same reefs over this same time period (Dustan 1977, Porter and Meier 1992). As the etiology of coral diseases continues to be studied, it will become increasingly important to fully characterize diseases with associated microbial pathogens. This will require establishment and maintainance of gene sequence data for the microorganisms associated with each coral disease, which will then allow intercomparison between disease events. G. Smith and K. Ritchie have initiated and are implementing this data base.

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RESUMEN

Una enfermedad de corales caracterizada por un novedoso patrón de destrucción rápida de tejido, apareció por primera vez en los arrecifes de los Cayos Medios de Florida en junio de 1995. Entre junio y octubre de 1995 la enfermedad infectó 17 especies de escleractinios y el hidrocoral, *Millepora alcicornis*. Poblaciones localizadas de *Dichocoenia stokesi*, la especies más afectada, presentó

mortandades de hasta 38%. Muchas colonias presentan pérdida total de tejido en unos pocos días al moverse la enfermedad a través de la colonia con tasas de hasta 2 cm en 24 hrs. Típicamente la pérdida de tejido se iniciaba en la base de las colonias y se movía hacia arriba. Por momentos el progreso de la enfermedad se detenía y las colonias retenían una cobertura parcial de tejido, que parecía una gorra encima de una colonia blanqueada. Cultivos de laboratorio de muestras de la línea de enfermedad revelaron una bacteria dominante que, al aislarla y caracterizarla utilizando técnicas genéticas y metabólicas, se parece mucho al género *Sphingonomas*. Cultivos puros de laboratorio de la bacteria produjo la enfermedad en colonias, recién recolectadas, incubadas en acuarios de laboratorio. La enfermedad que llamamos plaga tipo II apareció en diferentes arrecifes del sur de Florida y los Cayos de Florida en 1996 y 1997. Mientras que la mortandad de coral asociada a cada una de las epidemias estuvo confinada regionalmente y no fue recurrente en años subsiguientes en el mismo arrecife, las altas tasas de mortalidad de esta enfermedad la distinguen como una de las más serias registradas hasta ahora.

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