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-
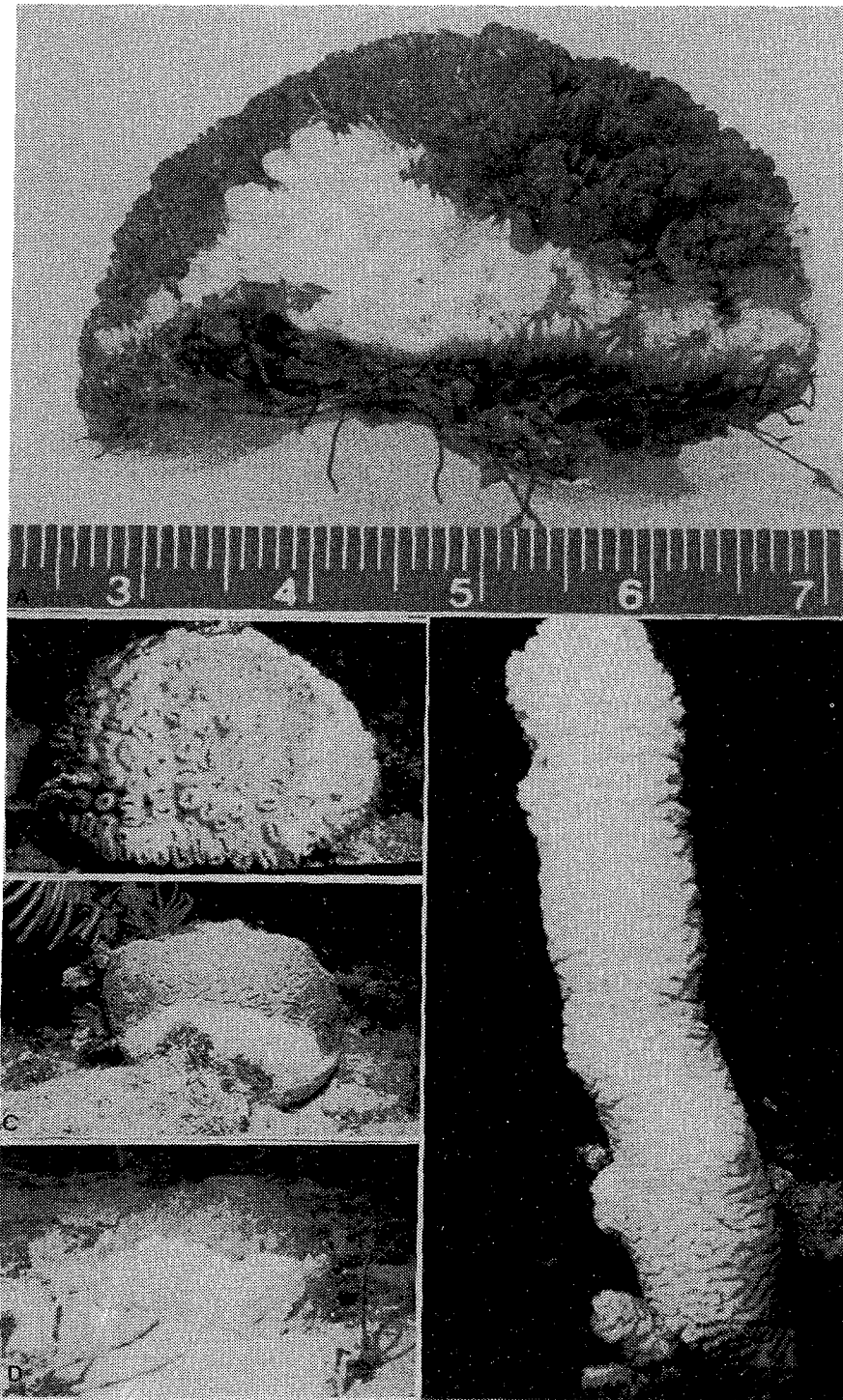
Fig. 1. Coral colonies infected with plague type II. A. Freshly collected *Dichocoenia stokesi* colony. Scale is in cm. B. *D. stokesi* with plague type II tissue loss from two infection points. C. *Diploria labyrinthiformis*. D. *Solenastrea bournoni*. E. *Dendrogyra cylindrus*. Plates b-e were photographed *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 bacteri-
al isolate grown on marine agar plates, or onto
uninoculated marine agar plates used as con-
trols. Colonies were visually monitored for dis-
ease. 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 exhibit-
ed 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 ini-
tiation 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 irreg-
ular 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 pat-
terns. 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 tis-
sue along the way. The rate of tissue degrada-
tion 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 pro-
gression and cessation of disease line move-
ment. 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 dur-
ing 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
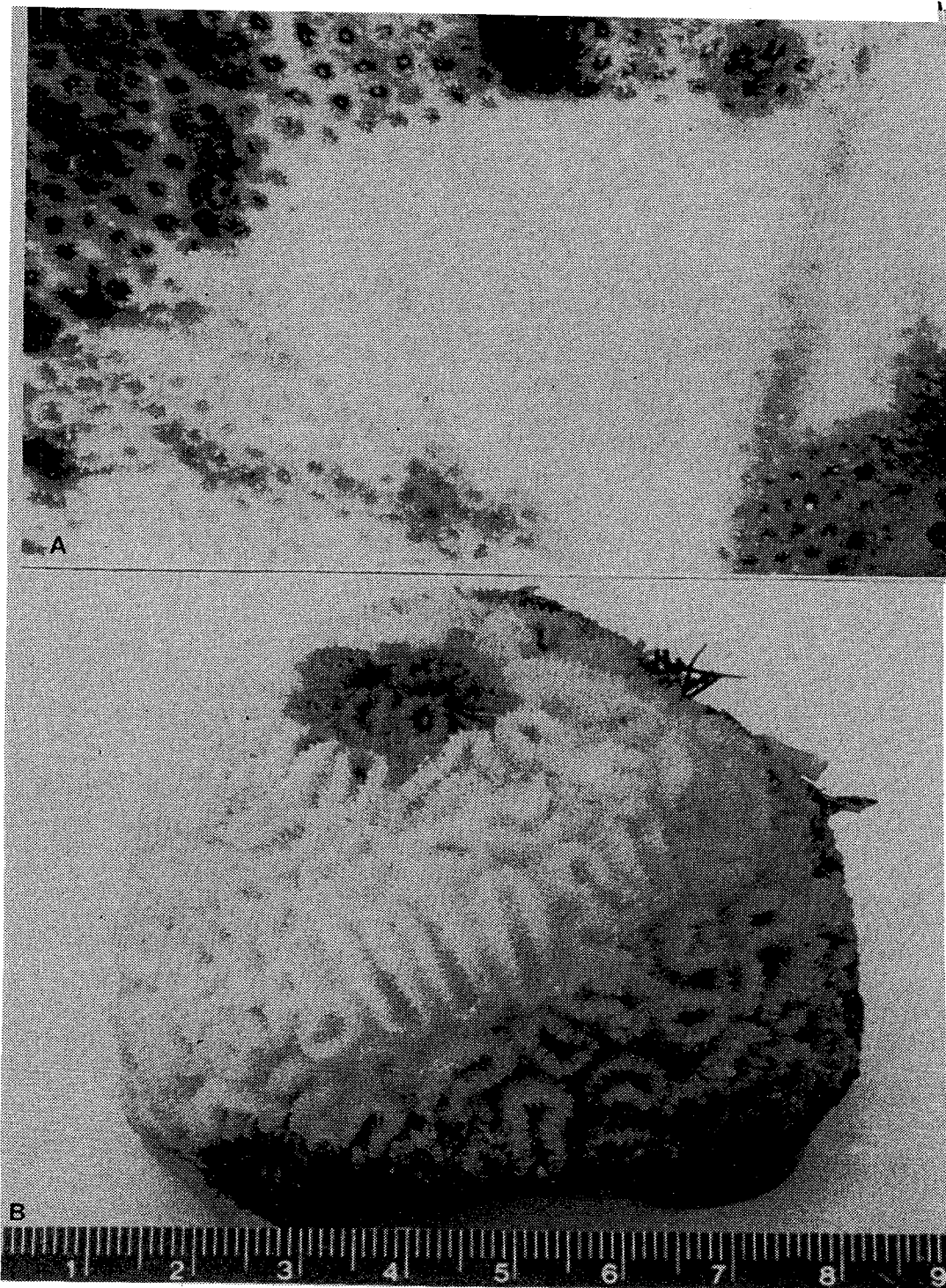long-term (days to months) cessation of disease

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.
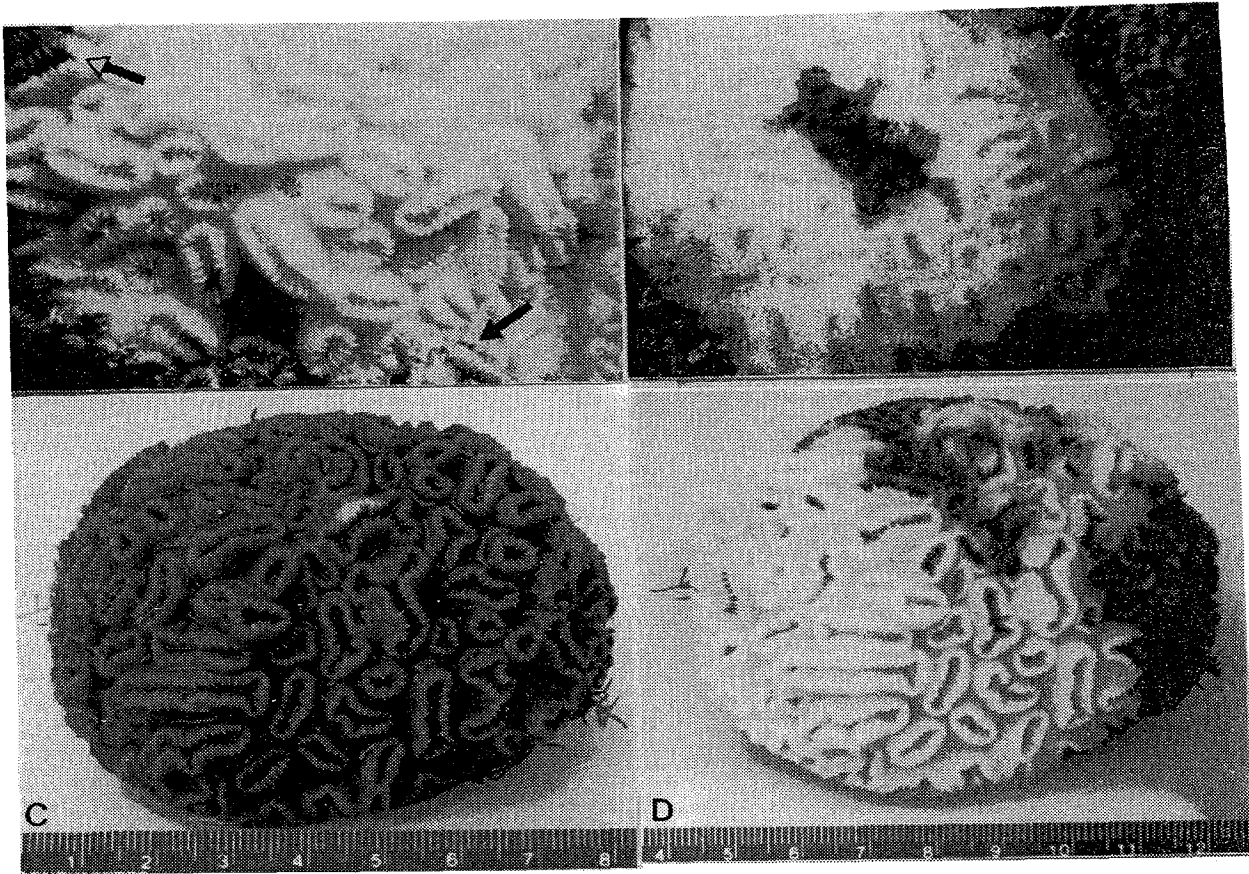
Fig. 3. Tissue loss patterns and infection of *D. stokesi*. a. *In situ* experiment documenting tissue loss progression. The nail (closed arrow) was placed at the edge of the disease line, in exposed coral skeleton, and the colony photographed three days later. The open arrow indicates the new disease 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 tis-

sue. 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
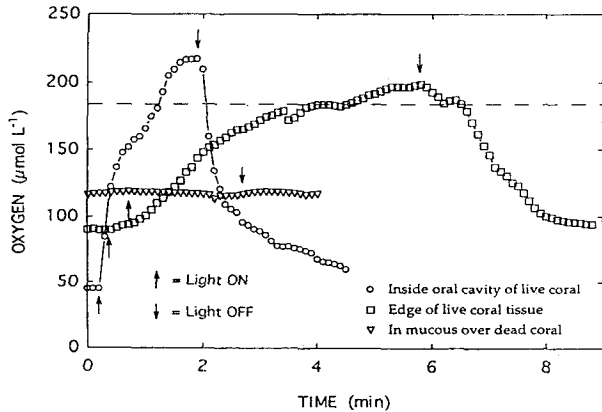
Fig. 5. O_2 concentrations associated with live, diseased, and dead areas of a freshly collected *D. stokesi* colony infected with plague type II. For healthy tissue the electrode tip was positioned inside a coral polyp. Measurements of the disease line were performed at the edge of the pigmented/bleached 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 result-

ed 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 report-
ed susceptible to plague (type I) on these same
reefs (Dustan, 1977). Three other species, how-
ever, (*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 dis-
ease exhibits tissue loss initiated almost exclu-
sively 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 associ-
ated with the disease line of plague type II is
different from that of other coral diseases. The
bacterium isolated in this study was character-
ized using genetic and metabolic approaches
and found to match most closely with the aero-
bic 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 character-
ized microbiologically. The presence and iden-
tity of microorganisms found in black band dis-
ease has been known for some time, and
includes the cyanobacterium *Phormidium
corallyticum*, sulfidogenic bacteria, sulfide-
oxidizing *Beggiatoa* spp., fungi, and many het-
erotrophs. 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 consor-
tium 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 gen-
erated 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 pre-
dominant 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 het-
erotrophic eubacteria). They were also able to
fully describe the disease state responsible for
the recent sea fan epizootic observed through-
out 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 observa-
tions 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 extreme-
ly variable types of bacteria (Peters *et al.* 1983,
Peters 1993). The very recently emerging coral
diseases (rapid wasting disease, white pox, yel-
low band, etc.; see Goreau et al. - this volume)
are either in the process of being microbiologi-
cally characterized or will be in the near future.

The direct measurement of oxygen concen-
tration 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 sur-
rounding 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.

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 especie 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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