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FIRST PROTOZOAN CORAL-KILLER IDENTIFIED IN THE INDO-PACIFIC

BY

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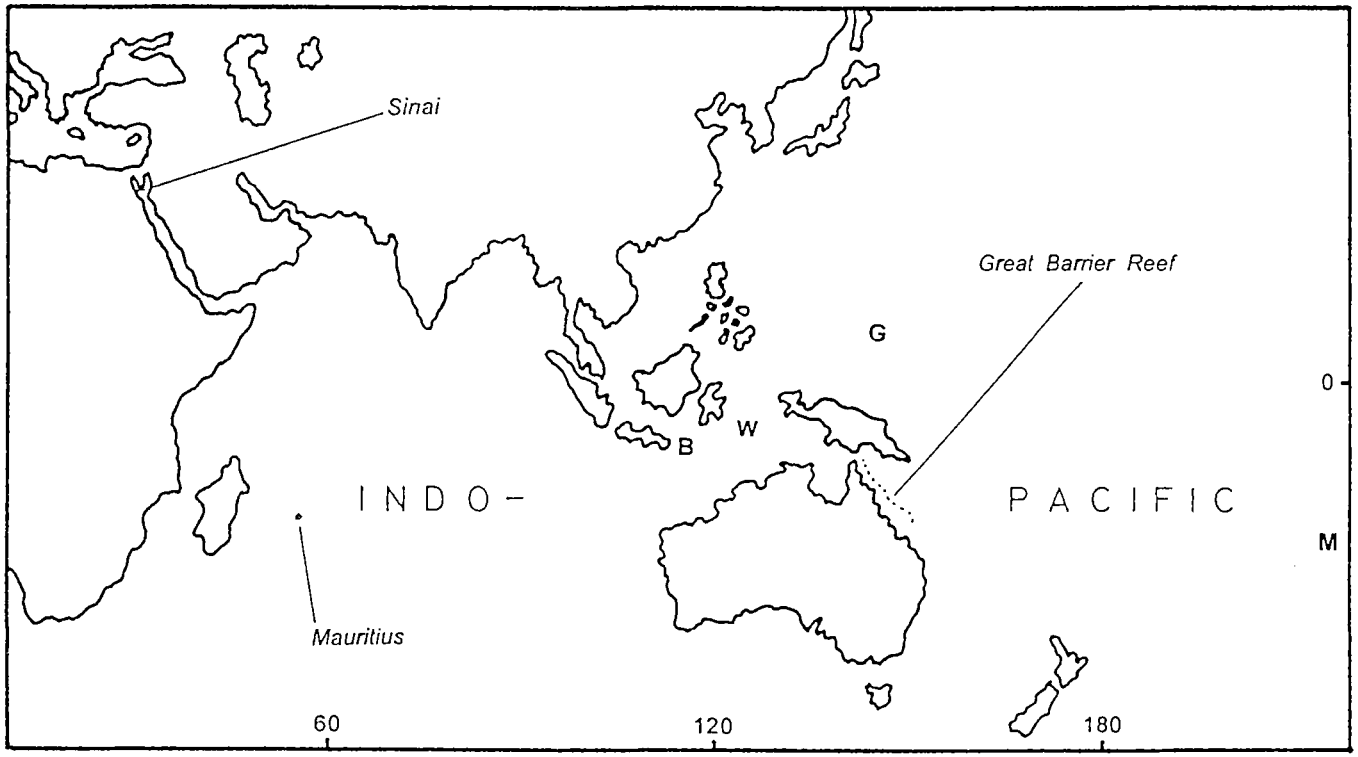

Figure 1. Chart of Indo-Pacific region showing the three SEB observation sites where corals infected with *Halofolliculina corallasia* were investigated: the coral reefs along the coast of Sinai, Red Sea; around the island of Mauritius, Indian Ocean; and in the area of Lizard Island, Great Barrier Reef, Pacific. Motupore Island on the SE coast of Papua New Guinea is not marked on the chart. Sites that were investigated with negative result (no SEB found) are: B: Bali; W: Wakatobi Islands; G: Guam; and M: Moorea.

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ARNFRIED ANTONIUS[1] and DIANA LIPSCOMB[2]

ABSTRACT

A unique coral disease has appeared on several Indo-Pacific reefs. Unlike most known coral diseases, this one is caused by an eukaryote, specifically *Halofolliculina corallasia*, a heterotrich, folliculinid ciliate. This protist is sessile inside of a secreted black test or lorica. It kills the coral and damages the skeleton when it settles on the living coral tissue and secretes the lorica. Thus, the disease was termed Skeleton Eroding Band (SEB). The ciliate population forms an advancing black line on the coral leaving behind it the denuded white coral skeleton, often sprinkled with a multitude of empty black loricae. This disease was first noted in 1988 and since has been observed infecting both branching and massive corals at several locations in the Indo-Pacific.

INTRODUCTION

More than a quarter century has passed since the first coral-killing syndrome, the Black Band Disease (BBD), was observed in the Caribbean Sea (Antonius, 1973). In the beginning regarded as a rare curiosity rather than a threat, it was soon followed by reports of other deadly syndromes, such as Shut-Down-Reaction (SDR) (Antonius, 1977), microbial infection (Ducklow & Mitchell, 1979), and White Band Disease (WBD) (Antonius, 1981a; and Gladfelter, 1982). All these observations were made in the Caribbean Sea or Western Atlantic (Garrett & Ducklow, 1975; Antonius, 1981b; and Dodge et al., 1982).

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In 1985, a first account was given on coral diseases in the Indo-Pacific (Antonius, 1985a) and an inventory of known syndromes 10 years later (Antonius, 1995a). Thereafter, new deadly syndromes on reef corals were reported in rapid succession, such as *Sphingomonas*-infections (Richardson et al., 1998), Type II WBD (Ritchie & Smith, 1998), Yellow Band Disease (YBD) (Korrubel & Riegl, 1998), *Aspergillus sydowii* damaging Gorgonians (Smith et al., 1996; Nagelkerken et al., 1997; Smith et al., 1998; Geiser et al., 1998; ISRS, 1999; Shinn, 2000; and Weir et al., 2000), and, although not a disease sensu stricto, Tissue Bleaching (TBL) (Glynn, 1993; Adjerout et al., 1995; Kushmero et al., 1996; Brown, 1997; and others). Accounts on reef-deterioration (Antonius, 1998) were given for Western Atlantic (Bruckner & Bruckner, 1997; Antonius & Ballesteros, 1998; and Goreau et al., 1998) as well as Indo-Pacific locations (Riegl et al., 2001). The majority of the responsible diseases appear to be caused by bacterial pathogens (Antonius, 1995a).

We report here the presence of a disease of corals in the Indo-Pacific (Fig. 1) caused by a ciliated protozoan. The progress of the disease is similar to other "band" diseases, such as BBD or WBD (Antonius, 1985b), the infection spreading as a line of pathogenic agents moving over a coral head leaving behind denuded skeletons. Unlike all the other diseases mentioned, which attack the soft tissues of corals, the new syndrome also damages the coral's skeleton. Therefore, we have named it Skeleton Eroding Band (SEB).

The organism associated with the disease is identified as *Halofolliculina corallasia*, a new species of folliculinid, heterotrich ciliate, which makes SEB not only the first known (stony) coral disease caused by a protozoon but also the first caused by an eukaryote.

The first time *H. corallasia* was noted on corals in reefs around Motupore Island, Papua New Guinea was in 1988. Black loricae were seen in microscopic preparations and drawn and photographed (Antonius, pers.obs.), but the phenomenon was not investigated any further. The same year, the syndrome was observed in reefs of Lizard Island, Australia and was listed in transect counts as "BBD-grey" (Antonius, pers.obs.). In 1990, the syndrome was registered as a rare occurrence in reef transects around Mauritius (Antonius, 1993) but its importance was not diagnosed. This finally happened in 1994 during a coral-reef survey in the Gulf of Aqaba, Straits of Tiran, and Ras Mohamed, Sinai, Red Sea (Antonius, 1995c, 1996).

In the following years, all previous observation sites (except Motupore) were revisited in order to investigate the SEB syndrome in detail. In 1998, SEB was mentioned for the first time in an official report (Antonius, 1998), and first photographs were presented in 1999 (Antonius, 1999c). In summer 2000, in order to gather badly needed data on the occurrence of SEB and other diseases (Antonius, 2000a), Pacific locations such as Moorea (Polynesia), Guam (Micronesia), Bali, and Wakatobi Islands (Indonesia) were investigated.

METHODS

Field assessments

Frequency of occurrence of SEB was investigated using a fast-working field technique, the Belt Method (Antonius, 1995b). It is a semiquantitative time-count technique that developed out of routine checks on reef health. Essentially, it is a derivative of visual census techniques that were developed to count reef fishes (e.g. Brock, 1954; Antonius et al., 1978; and Russell et al., 1978) and to assess *Acanthaster* infestations (e.g. Antonius, 1971; and Endean, 1974). While surveying a coral reef, it is relatively easy to note down all cases of active diseases on corals that are encountered. To make this approach semiquantitative merely requires standardization with: (1) a time-frame limiting the duration of each individual survey; and (2) organizing the numbers of every disease encountered into categories.

The time-frame of one single survey is 30 minutes. During this period, the diver swims fairly close to the reef surface and registers all pathologic syndromes on corals in a path, or "belt", about 2 m wide. One such 30-minute survey is considered a "scan". The diver's speed during such a scan may vary. In scarcely populated reef areas and/or reef zones with large coral colonies, the diver will proceed faster and cover more distance than in densely populated reef areas and/or reef zones with smaller coral colonies. However, experience has shown that the total number of coral colonies investigated during one scan remains surprisingly constant (e.g. Antonius, 1988a). In order to assure this consistency, the time count has to be interrupted every time the diver traverses completely barren reef areas.

The categories for the instances of diseases encountered range from zero to six. Zero (0) naturally means no syndrome found. A disease is considered condition: (1) "rare" when 1-3 cases are found during a 30-minute scan; (2) "moderate" when 4-12 cases are found; (3) "frequent" when 13-25 cases are found; (4) "abundant" when 26-50 cases are found (Antonius, 1988a); (5) "epidemic" when 51-100 cases are found; and (6) "catastrophic" at the end of the scale when the number of syndromes exceeds 100, which means that the number of diseases actually become uncountable (Antonius, 1991). (Note: the correct term for a code 5 conditions is "epizootic." This, however, has led to so much confusion with "epizoism" (Antonius & Ballesteros, 1998) that we prefer the unequivocal "epidemic".) The particular numerical values of these six categories have been determined as the most useful based on practical experience during many years of fieldwork. Since the Belt Method is fast and simple to use -- requiring only a watch, a writing slate, and a pencil -- it is very well suited to survey large reef tracts. Under reasonably calm conditions, kilometers of linear reef extension can be surveyed during one day.

Transmission experiments

In order to find out how the SEB syndrome spreads within a given coral population, infection as well as contagion experiments were set up in reefs and in aquaria, following tried methods (Antonius, 1985b). To test infectiousness, SEB-diseased coral colonies were surrounded by three types of target specimens of different coral species: some healthy, some with small injuries, and some with active WBD. At

Sinai and Lizard Island, experimental sample size in reefs consisted of 10 SEB-diseased specimens, each one surrounded by five of these target specimens of different species. Exactly the same arrangement was repeated in aquaria. In Mauritius, an experimental series using 12 SEB-diseased specimens was conducted in reefs alone. To test contagiousness, SEB-diseased specimens were brought into direct contact with healthy ones also representing a variety of species. They were usually tied to each other by very fine fishing lines. Experimental sample size at Sinai and Lizard Island consisted of 10 diseased-healthy coral pairs of varying species in reefs, as well as 10 such pairs in aquaria. In Mauritius, a total of 15 coral pairs were tested in reefs alone.

Pathology

Under the fieldwork conditions of the project, it was not possible to achieve axenic cultures of the pathogen for testing Koch's principle. However, in order to test the influence of possible bacterial synergists on disease behavior, they were removed from the hypothetical SEB consortium by antibiotics. At the Sinai research site, three specimens each of small, infected colonies of *Acropora, Stylophora,* and *Goniastrea* were exposed to the antibiotics penicillin, erythromycin, and gentamycin following a tried procedure (Antonius, 1985a). One gram of the respective antibiotic was tied into a watertight corner of a one-gallon plastic bag. The bag was then put over a small SEB-infected coral, tied at the base, and the antibiotic released. The bag thus contained about two liters of water plus antibiotic, and was removed after 24 hours. Further behavior of these SEB infections was then kept under microscopic observation for about three weeks.

Sample collection

Diseased coral specimens showing the black band of SEB infection were, whenever possible, first photographed *in situ* at the original reef location and then transferred into holding tanks or aquaria for more detailed close-up photography. Live material was also photographed under stereo- as well as research compound-microscopes. These SEB-diseased coral pieces were then fixed in 4% buffered formaldehyde in seawater, individually wrapped in soft plastic bags, and stored and transported in small glass jars filled with preservation fluid. A collection of these samples from Sinai, Mauritius, and Lizard Island is stored at the Institute for Paleontology, University of Vienna, Austria.

Microanatomy

Preserved individuals of *Halofolliculina corallasia* were separated from SEB-diseased coral splinters, embedded in paraffin, sectioned by microtome, transferred to microscopic slides, stained, and preserved under cover slides, generally following methods with a very successful record (Antonius, 1965). Samples are stored at the Department of Biological Sciences, George Washington University, Washington, DC, USA. For documenting the impact of SEB on the coral skeleton, standard scanning electron microscopy techniques were employed. Samples are stored at the Institute for Paleontology, University of Vienna, Austria.

RESULTS

HALOFOLLICULINA CORALLASIA, sp. nov.

Protozoa, Ciliata, Heterotrichida, Coliphorina, Folliculinidae, *Halofolliculina corallasia,* sp. nov.

The species name refers to its appearance on the coral when viewed through a microscope: Greek *lasios* (λασιοσ) means "densely overgrown" or "villous".

Diagnosis

As in other members of the genus *Halofolliculina* (Hadzi, 1951), *H. corallasia* is sessile in a lorica that either lies flattened against the substrate or stands almost upright, in both cases partially embedded in the coral skeleton (Hadzi, 1951). The lorica has an average length of 220 μm (maxima 370 μm, minima 135 μm) and a width of 95 μm (maxima 130 μm, minima 55 μm). The main body within this total length measures an average of 135 μm and the neck, 85 μm. The width of the neck averages 35 μm. The form of the lorica is sac-like with a rounded posterior and a cylindrical neck that angles up from the surface at about 45 degrees (Fig. 7). The neck of the lorica has a single sculpture line circumscribing it. The cell body is attached at its pointed posterior end to the base of the lorica. The cell is large and elongate with two conspicuous pericytostomial wings measuring 175-200 μm when fully extended and bearing the adoral zone of membranelles (feeding cilia). These pericytostomial wings are somewhat unequal in length. The cell is highly contractile and when disturbed retracts completely into the lorica. Two thin flaps of lorica material (one dorsal and one ventral) form an operculum that plugs the opening of the lorica when the animal is contracted (Fig. 8). The somatic cilia are uniform. The nucleus is condensed and oval, rather than beaded. This species differs from other described members of the genus in that the lorica is colored a dark smokey grey to black (clusters of which appear as black spots on the infected coral, Figs. 3 and 4), its size is comparatively small, and it is found infecting corals. A detailed anatomical description of the new species is in preparation (Lipscomb & Antonius, in prep).

Type material

Holotype and paratypes presently are located at the junior author's laboratory, the Department of Biological Sciences, George Washington University, Washington, DC, and will ultimately be deposited in the collections of the Department of Systematic Biology (Invertebrate Zoology), Smithsonian Institution, Washington, DC, USA. The material was isolated from *Acropora downingi* collected in 2 m depth in Mersa Bareika, Ras Mohamed, Sinai.

THE SEB DISEASE

Under the microscope, a live advancing front of *Halofolliculina corallasia* appears as a dense coat of bifurcated beige "tentacles" (pericytostomial wings) emerging from flask-shaped, black loricae (Fig. 5). In this foremost front, loricae often are packed so densely that they form an almost indistinguishable black mass (Fig. 4). Sometimes

their line follows the structure of the coral skeleton, such as the rim of a coral cup or corallite (Fig. 5). In about 10% of all SEB cases encountered in the field, small quantities of nonpathogenic cyanophytes were found dispersed throughout the live SEB front producing the gas bubbles visible in Figs. 3 and 4. They do not change SEB's behavior in any way. The basal part of each lorica is embedded in the spongy, trabecular structure of the coral skeleton, which is broken up into splinters by the ciliate. When observing live material, the slightest jolt makes all protist retract instantly into the loricae but, after only half a minute or so, they will slowly reappear extending their bifurcated wings, resembling a bed of microscopic garden eels to the eye of a diver (Fig. 5).

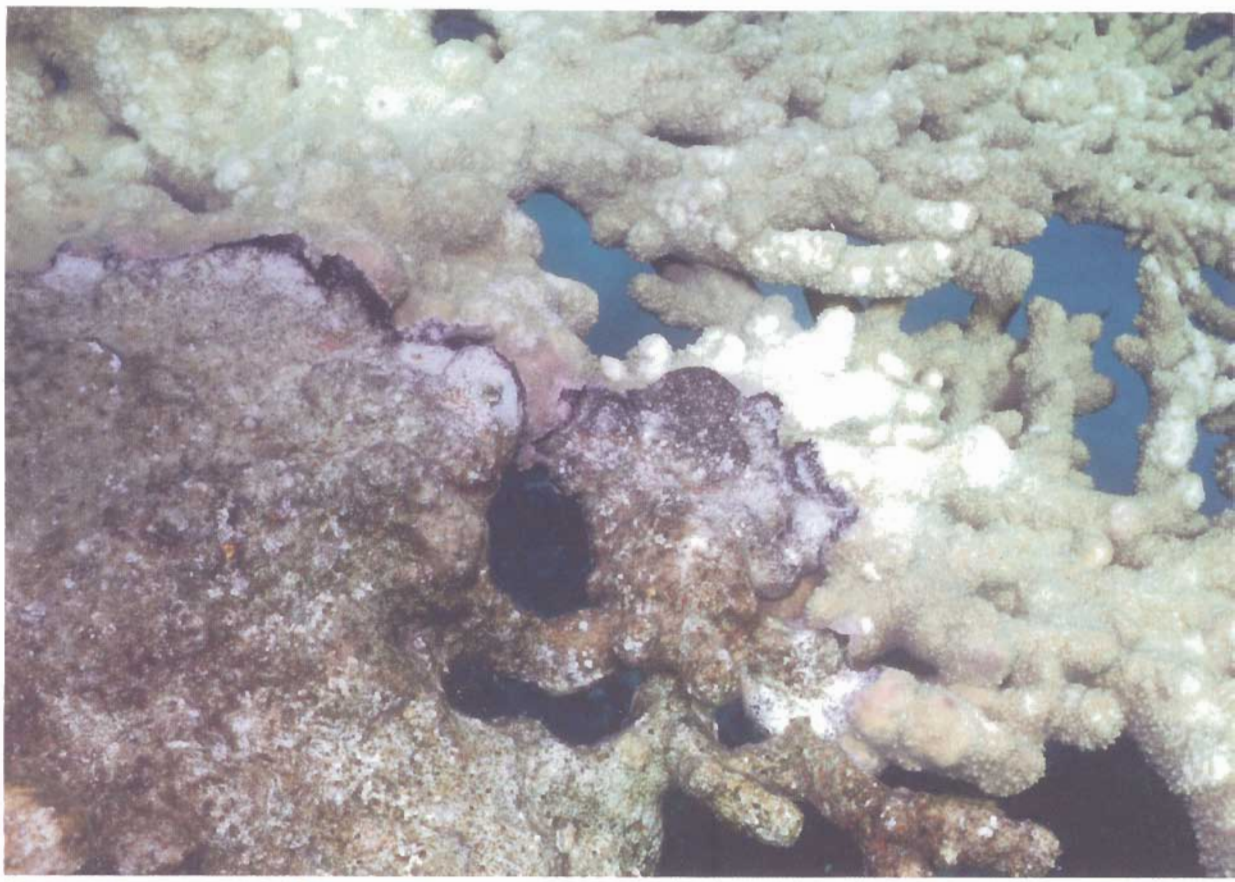
Upon cell division (asexual reproduction) vermiform, migratory larval stages, are produced. These ciliated larvae scout the terrain ahead of the SEB band and locate a suitable spot not too far from this band (Fig. 4). There they penetrate the living coral tissue (which appears unharmed up to this moment), settle down in clusters (Fig. 4), and secrete pseudochitinous loricae which are shaped by the rapid spinning of the larvae. It is the chemicals associated with the unhardened lorica, combined with the mechanical disruption caused by the spinning larvae, that appears to damage the coral skeleton (Antonius, 2000 b) and initiates lysis of the coral tissue. The mechanics of this process can be observed clearly on live material under the microscope. As a consequence of these processes, not only is the coral tissue gone once infection has passed over an area, but the bare coral skeleton has lost all fine structure, its surface looking like a microscopic rubble field (Fig. 6). When viewed with scanning electron microscopy, the rounded imprints of the tips of loricae are clearly visible (Fig. 6). Interestingly, coral polyps appear unharmed ahead of the advancing front of destruction (Fig. 4). SEB does not fall into the recently described category of epizoism syndromes (Antonius & Ballesteros, 1998; and Antonius, 1999a, b) but represents a genuine disease forming an advancing front which leaves behind a naked skeleton (Antonius, 1981a, 1988b; and Richardson et al., 1998).

Figure 2. (Opposite page above.) Underwater photograph of a 3 m-diameter colony of *Acropora downingi* with a very large SEB infection in Mersa Bareika, Ras Mohamed, Sinai. The black band of pathogenic *Halofolliculina corallasia* separates live coral tissue (above right) from denuded skeleton (below left). The older, tissue-stripped parts of the skeleton at the left bottom of the picture are overgrown by algal turf.

Scale: entire width of photograph at bottom = 80 cm.

Figure 3. (Opposite page below.) Close-up photograph of the same SEB infection as depicted in Fig. 2. The black front of coral-killing *Halofolliculina corallasia* proceeds upward towards the living coral tissue. The surface of the coral skeleton in the rear of the black band, freshly stripped of coral tissue (below), is sprinkled with tiny black dots consisting of clusters of empty loricae of the ciliate. This "dotted" zone distinguishes the SEB syndrome from the well-known BBD.

Scale: entire width of photograph = 8 cm.



Ecology

SEB occurs in sheltered, lagoon-type environments at a depth of 0 m to at least 35 m (Winkler, in prep.), showing the greatest abundance at depths between 0.5 m and 3 m. In this shallow range, up to 5% of any given coral species can be infected with the ciliate. SEB was found throughout different seasons of the year: April, May, September, and October around Sinai and Aqaba (Red Sea, Fig. 1); April, September, and October around Mauritius (Indian Ocean, Fig. 1); January, February, and August around Lizard Island (Pacific, Great Barrier Reef, Fig. 1); and June, July around Motupore Island, SE Papua New Guinea (Pacific, Fig. 1). Thus, SEB, occurring through warmer and cooler periods in roughly equal concentrations, does not seem to be seasonal.

SEB infects and damages a wide variety of branching and massive reef corals, including the species *Stylophora pistillata, Pocillopora damicornis, P. verrucosa, P. eydouxi, Montipora monasteriata, Acropora aspera, A. humilis, A. formosa, A. noblis, A. tenuis, A. valida, A. florida, A. hyacinthus, A. clathrata, A. downingi, Leptoseris explanata, Pachyseris rugosa, Hydnophora microconos, Favia stelligera, Favites abdita, Goniastrea retiformis, Leptastrea purpurea, Cyphastrea chalcidicum,* and *C. serailia.*

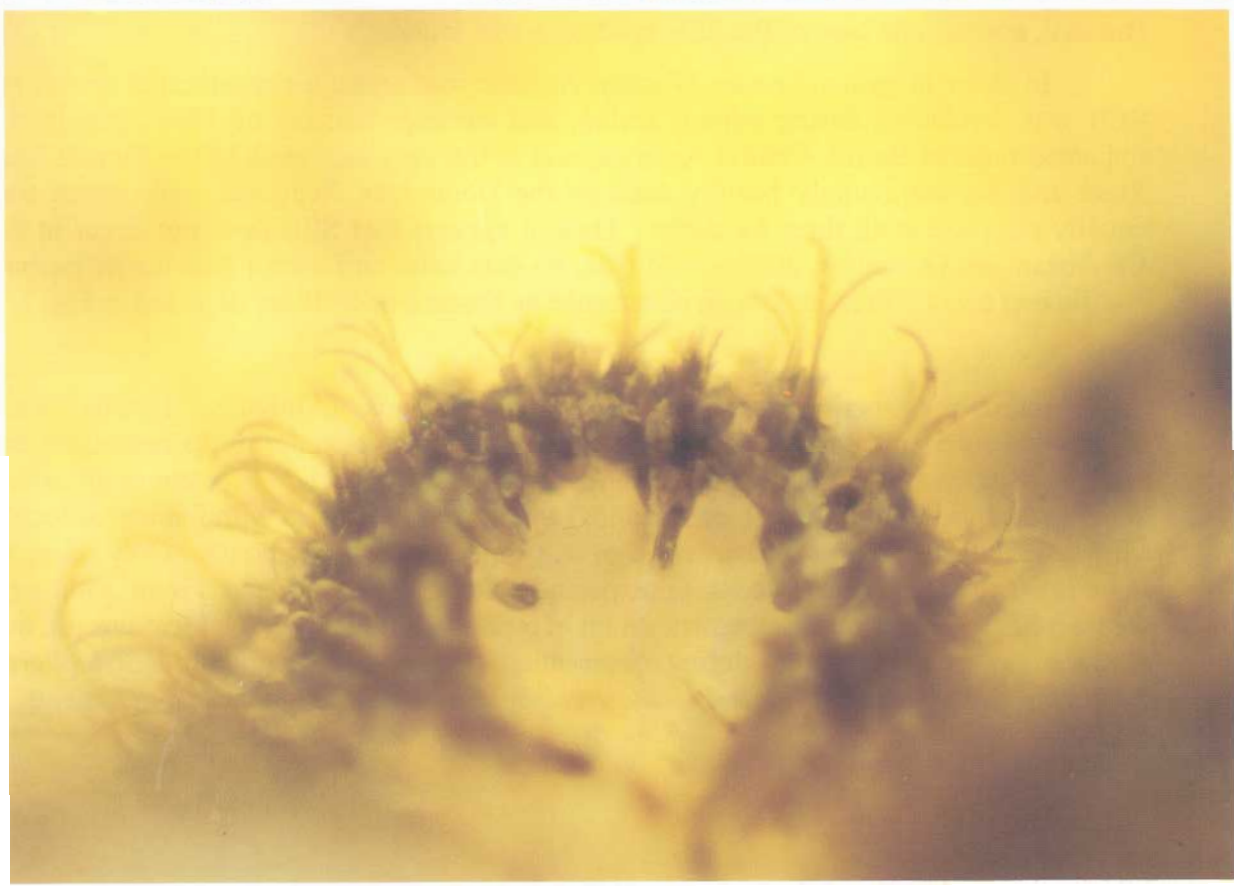
Symptoms of the disease vary. It may appear as thin lines of not more than 1 mm in width (e.g on *Stylophora pistillata*) or as thick, black bands up to 10 cm wide, encircling dead coral surface areas of 80 cm in diameter (e.g. on *Acropora downingi*) (Fig. 2; = *A. cytherea* in Antonius et al., 1990). In contrast to ordinary BBD, which leaves behind a zone of unblemished, brilliant white coral skeleton (Antonius, 1981a), the white coral skeleton immediately behind the advancing front of SEB shows a multitude of tiny black dots that are clusters of flask-like, black housings (loricae) of the ciliate (Figs. 3 and 4) left behind. SEB infections can be almost stationary, moving perhaps 1 mm per week, or comparatively fast, progressing more than 1 mm per day, resembling the behavior of BBD (Antonius, 1988b). The syndrome occurs rather evenly distributed in reef-crest areas, with no apparent tendency to form clusters.

Figure 4. (Opposite page above.) Underwater macrophotograph of the same SEB infection as shown in Fig. 2 and Fig. 3. The front moves from left to right. The rear of the black band (left) shows the usual "sprinkled" or "dotted" appearance, while in front of it (right) partly contracted, but totally unharmed, polyps are visible. The finger-like protrusion of the black band at upper right is a cluster of "scout" larvae of *Halofolliculina corallasia* penetrating the coral tissue, producing loricae and becoming sessile.

Scale: entire width of photograph = 18 mm.

Figure 5. (Opposite page below.) Microphotograph of *Halofolliculina corallasia* on a corallite of *Acropora aspera* at Lizard Island. The bifurcated, beige, pericytostomial wings of the animals are fully extended, emerging from vase- or flacon-shaped black loricae. The basal part of every lorica is embedded in the coral skeleton, producing the specific erosion of the surface typical for SEB.

Scale: entire width of photograph = 1.8 mm.



In some of the reefs under observation, frequency of occurrence of SEB has definitely been increasing over the years. It went from a code 1 (rare) to a code 3 (frequent) level (Antonius, 1995b) in the Rivière Noire area of SW Mauritius (Antonius, 1993) between 1990 and 1998. Exactly the same, a rise from code 1 to code 3, happened in Sinai in only three years (Antonius, 1998) from 1994 to 1997. Only in the reefs surrounding Lizard Island was the change very slight. It took SEB 10 years, from 1988 to 1998, to achieve a rise from a code 1 (rare) to a code 2 (moderate) frequency of occurrence.

Biogeography

As mentioned above, SEB has been found, observed, and investigated in the Red Sea and at Mauritius in the Indian Ocean as well as on the SE coast of Papua New Guinea and the Great Barrier Reef in the Pacific. Since the disease definitely occurs from the western end of the Indian Ocean to the western rim of the Pacific, an attempt was made to document SEB in other locations of the Pacific Ocean as well. Thus, reefs around the islands of Moorea (Polynesia), and around Guam (Micronesia) were investigated as well as many reef sites throughout the Wakatobi archipelago southeast of Sulawesi (Indonesia). Except for frequent epizoism by *Pneophyllum conicum* (the PNE syndrome, Antonius, 1999b), these reefs were found to be relatively healthy and no SEB whatsoever was observed. Because human-related impact on all these reefs was considered minor, an effort was made to survey reefs in Nusa Dua Watersport Bay, one of the most heavily impacted marine environments of Bali. Reef outcrops in this bay are exposed to turbidity, sewage, divers, and boat anchors, to mention just the major sources of stress. But still, not a single case of the SEB syndrome was found.

In order to gather data on Western Atlantic reef areas, a concentrated search for SEB was conducted during winter, spring, and summer seasons of 1997-2000 in the impacted reefs of Belize, Central America, and in the very sick reefs of the Florida Reef Tract and the surprisingly healthy reefs of the Dominican Republic. The result was equally negative at all three locations. Thus, it appears that SEB does not occur in the Caribbean Sea (Antonius, 2000a). To date, no data exist on Eastern Atlantic or Eastern Pacific reef areas. Present data on occurrence or absence of SEB are depicted in Fig. 1.

Pathology

Infection experiments, following tested methods (Antonius, 1985b), were inconclusive perhaps due to insufficient observation time or inability to reproduce the exact conditions that cause new infections. Freshly collected corals, some with active WBD and/or artificial injuries (see Methods) and placed in aquaria with freshly collected SEB-diseased ones did not get infected over a time span of one month. Only once, at Lizard Island, after three weeks of exposure, did a WBD-afflicted *Favia stelligera* contract SEB. The same arrangement set up in reefs at all three research locations did not produce any positive results during observation periods of one month. *Stylophora, Pocillopora, Acropora, Favia,* and *Goniastrea* species were used in these experiments.

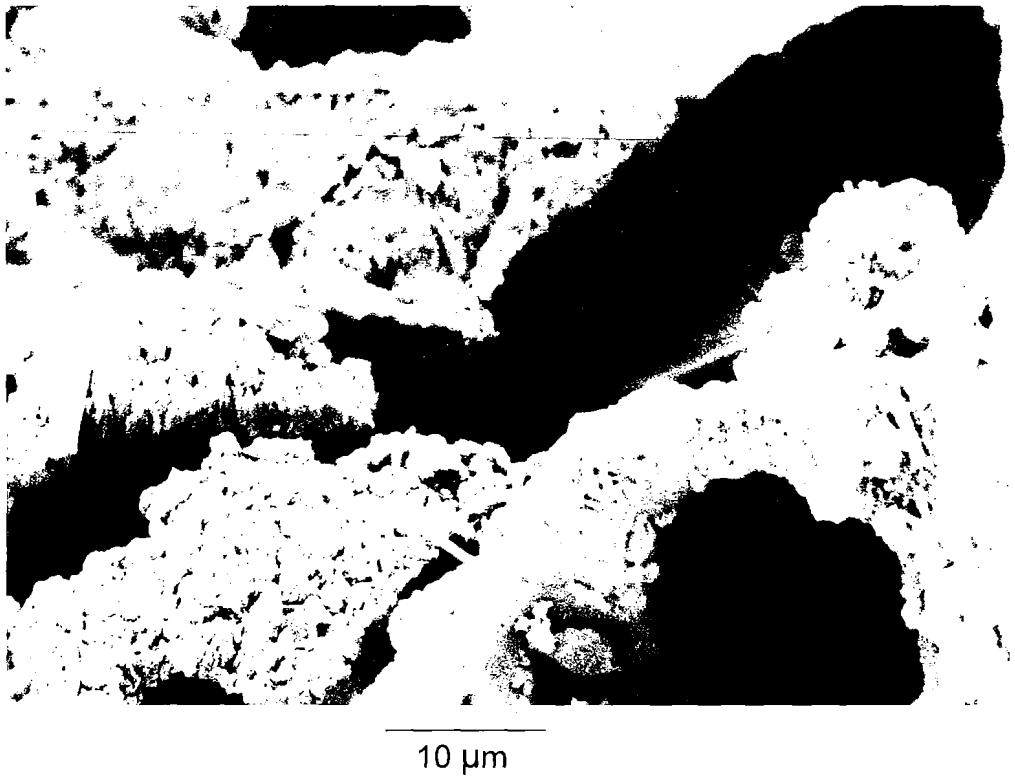


Figure 6. SEM photograph of a septum of *Cyphastrea chalcidicum* from Mauritius showing the typical etched-out holes where the posterior ends of loricae of *Halofolliculina corallasia* were partially embedded in the coral skeleton. Clearly visible is the very smooth surface inside the holes as well as the tiny splinters surrounding them broken loose in the process of excavation. These skeletal fragments make corallites and coenosteum appear like microscopic rubble fields. The term "Skeletal Eroding Band" (SEB) of the disease derives from this characteristic.

Contagion experiments were more successful. When diseased parts of one coral were brought into contact with the healthy surface of another one, the target showed first signs of SEB within one week. Only branching growth forms, such as *Stylophora, Pocillopora,* and *Acropora* species, were used in these experiments which were 100% successful in aquaria at Sinai and Lizard Island. In reefs of all three research sites, 90% of experiments were positive in which the two corals, donor and target, remained in contact. Despite being tied to each other, wave action or other disturbances sometimes separated these pairs. Successful transmissions, regardless of the combination of coral species used, resembled experimental results with BBD (Antonius, 1985b).

Several SEB infections that were made bacteria-free with antibiotics (Antonius, 1985a) were subsequently examined under stereo- and research-microscopes over periods of about three weeks. It turned out that behavior and virulence of *Halofolliculina corallasia* remained identical before and after treatment. In all "disinfected" samples, SEB fronts advanced at the same speed as before, producing scouting larvae, clusters of new loricae, erosion of coral skeleton, and lysis of coral tissue. It seems that SEB's pathogenicity and virulence are caused by *H. corallasia* without the aid of bacterial synergists.

DISCUSSION

None of the other 31 genera in the family Folliculinidae, nor the other species of the genus *Halofolliculina*, are known to cause diseases in corals or any other animal. The members of the family, however, are all sessile in loricae which may be attached to aquatic plants or invertebrates, primarily in the marine environment (Corliss, 1961). Among these, *Halofolliculina corallasia* is the only species known to date acting as a true pathogen on reef corals, an activity directly observable on live material under the microscope. In the reef areas under observation, the SEB disease caused by *H. corallasia* has certainly become more frequent and conspicuous over the past several years. The reason for this may be found in generally worsening conditions in coral-reef environments (Ginsburg, 1993; and Wilkinson, 2000). Under these conditions, increasingly virulent coral diseases not only open up the gateways for infection (Antonius, 1985b), but decaying coral tissue produces bacterial blooms (Mitchell & Chet, 1975) which are food for Folliculinids (Andrews, 1946; and Laakmann, 1903) such as *H. corallasia.* The result may well be population explosions of *H. corallasia* which subsequently lead to SEB infections on reef corals. Lysis of the coral tissue by the SEB disease most probably attracts further swarms of bacteria on which *H. corallasia* feeds, thus in an indirect way converting coral tissue into nourishment. A veritable *circulus vitiosus* thus could be initiated, leading to more infections and spreading of SEB disease in the coral population. These hypothetical conditions, however, could not be produced in the limited field working time of the project. Pathogenesis of primary infections, therefore, really could not be clarified by experiments fitting the time-frame available. Clearly, much more research is needed. In comparison, research on BBD has gone on for almost 30 years (Antonius, 1973) and is still in progress.

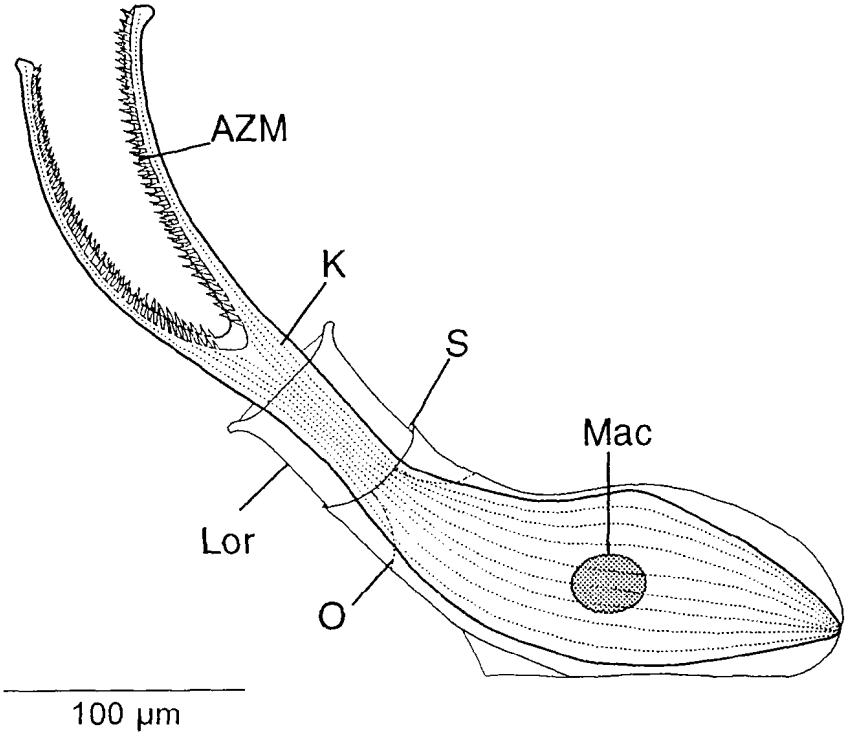


Figure 7. Drawing of *Halofolliculina corallasia* showing the major distinguishing features. The adoral zones of membranelles (AZM) line the two pericytostomial wings and create water currents that direct food into the cell mouth. The cell body sits inside a lorica (Lor), which has a sculpture line (S) around the neck, and an operculum (O) that closes off the lorica when the cell has contracted. The macronucleus (Mac) is spherical rather than beaded. Body cilia lie in rows called kineties (K).

The tendency of Folliculinids to form massive aggregations on a wide variety of substrates has been known for a long time and has been depicted variously by Andrews in excellent drawings (Andrews, 1914, 1915, 1923, 1949). All these species are epibenthic to epizoic, completely harmless and nonpathogenic. The unusual pathogenic behavior of *H. corallasia* may have developed in a way termed "Xenökie" by Hadzi (1935), who observed several different species of Folliculinids sitting with the posterior end of their loricae in the empty chambers of individual bryozoans. They did not appear to harm any live bryozoans, but this kind of behavior may well constitute the first step toward pathogenicity. As so many other pathogens do (Antonius, 1985a, 1988b; Antonius and Ballesteros, 1998; and Verlaque et al., 2000), *H. corallasia* may have learned to invade living coral tissue from the secure foothold of adjacent bare-skeleton surfaces. Interestingly, coral polyps immediately ahead of an advancing front of SEB appear undisturbed (Fig. 4), an observation also made on BBD infections more than a decade ago (Antonius, 1988b; figs. 3, 4, 5). The special surface-eroding anchoring of the lorica then possibly could have developed in response to the push-and-pull of the surrounding living (but doomed) coral tissue. How this process erodes the coral surface, produces the microscopic "rubble fields", and starts lysis of the coral tissue can be directly observed on live material. No secondary microborers are involved here. This specific type of damage and boring that *H. corallasia* inflicts to the surface of the coral skeleton (Antonius, 2000b) might also be preserved and recognizable in the fossil record (Golubic et al., 1975; and Falces, 1997).

CONCLUSIONS

SEB, up to now, has been documented on exactly 24 Indo-Pacific coral species, a number that is bound to increase in the future. So far, it has not been found in Atlantic and Caribbean waters, i.e., outside of the Indo-Pacific zoogeographic region (Antonius, 2000a). In the Indo-Pacific, the pattern of occurrence is somewhat complicated and not easy to understand. In the western Indian Ocean the disease was found at the northern (Red Sea) and southern (Mauritius) limits of reef development, but towards the eastern extreme, where Indic and Pacific waters merge (Bali, Wakatobi Islands), SEB does not seem to occur. Another 30° to the east, roughly along the 145° E meridian, SEB was found south of the equator (Great Barrier Reef, Papua New Guinea) but not in the north (Guam). Toward the eastern margin of the West Pacific (Moorea, 150°W, 17°S) SEB also seems to be absent. This confusing picture clearly needs input by more than one fieldworker. Thus, in order to clarify questions of occurrence and distribution and also to quantify the impact of SEB on coral reefs on a wider scale, marine biologists must learn to distinguish SEB from BBD (Antonius, 1999c). In our discussions with on-site scientists, we have learned that SEB usually was mistaken for BBD. SEB may have been scarce and hard to find in the past but, at least in the observation areas discussed here (Fig. 1), it passed the "threshold of noticeability" a decade ago. Our present field data on occurrence of SEB, collected on a relatively small scale but over a time span of about 10 years, suggest that its frequency of occurrence is increasing, adding substantial impact to the rising tide of coral diseases (Antonius, 1995d; Goreau et al., 1998; Richardson, 1998; Epstein, 1998; Hayes and Goreau, 1998; Harvell et al., 1999; and Porter et al., 1999).

Figure 8. Microphotograph of an empty lorica of *Halofolliculina corallasia* showing the suture line (S) and the operculum (O) that closes the opening when the cell is contracted.

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Note: In March 2001, an interesting observation was made at the Jordanian fertilizer port, close to the Saudi Arabian border (Gulf of Aqaba, Red Sea), which proved to be the most heavily polluted of all Jordanian research sites (Winkler, in prep.).

Several colonies *Echinopora gemmacea* and *Echinophyllia aspera* of about 1 m in diameter were attacked by extremely massisve and virulent SEB infections and killed entirely within a time span of just one week (Caitriona McInerney, pers. comm.).

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