

2016

Gross and Microscopic Lesions in Corals from Micronesia

T. M. Work

US Geological Survey, thierry_work@usgs.gov

G. S. Aeby

Hawaii Institute of Marine Biology

K. A. Huguen

Woods Hole Oceanographic Institution

Follow this and additional works at: <http://digitalcommons.unl.edu/usgsstaffpub>



Part of the [Geology Commons](#), [Oceanography and Atmospheric Sciences and Meteorology Commons](#), [Other Earth Sciences Commons](#), and the [Other Environmental Sciences Commons](#)

Work, T. M.; Aeby, G. S.; and Huguen, K. A., "Gross and Microscopic Lesions in Corals from Micronesia" (2016). *USGS Staff--Published Research*. 895.

<http://digitalcommons.unl.edu/usgsstaffpub/895>

This Article is brought to you for free and open access by the US Geological Survey at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in USGS Staff -- Published Research by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

Gross and Microscopic Lesions in Corals from Micronesia

T. M. Work¹, G. S. Aeby², and K. A. Huguen³

Veterinary Pathology
2016, Vol. 53(1) 153-162
© The Author(s) 2015
Reprints and permission:
sagepub.com/journalsPermissions.nav
DOI: 10.1177/0300985815571669
vet.sagepub.com



This document is a U.S. government work and is not subject to copyright in the United States.

Abstract

The authors documented gross and microscopic morphology of lesions in corals on 7 islands spanning western, southern, and eastern Micronesia, sampling 76 colonies comprising 30 species of corals among 18 genera, with *Acropora*, *Porites*, and *Montipora* dominating. Tissue loss comprised the majority of gross lesions sampled (41%), followed by discoloration (30%) and growth anomaly (29%). Of 31 cases of tissue loss, most lesions were subacute (48%), followed by acute and chronic (26% each). Of 23 samples with discoloration, most were dark discoloration (40%), with bleaching and other discoloration each constituting 30%. Of 22 growth anomalies, umbonate growth anomalies composed half, with exophytic, nodular, and rugose growth anomalies composing the remainder. On histopathology, for 9 cases of dark discoloration, fungal infections predominated (77%); for 7 bleached corals, depletion of zooxanthellae from the gastrodermis made up a majority of microscopic diagnoses (57%); and for growth anomalies other than umbonate, hyperplasia of the basal body wall was the most common microscopic finding (63%). For the remainder of the gross lesions, no single microscopic finding constituted >50% of the total. Host response varied with the agent present on histology. Fragmentation of tissues was most often associated with algae (60%), whereas necrosis dominated (53%) for fungi. Two newly documented potentially symbiotic tissue-associated metazoans were seen in *Porites* and *Montipora*. Findings of multiple potential etiologies for a given gross lesion highlight the importance of incorporating histopathology in coral disease surveys. This study also expands the range of corals infected with cell-associated microbial aggregates.

Keywords

coral, histopathology, disease, Micronesia, Indo-Pacific, Cnidaria, bleaching, dark discoloration, symbionts

Coral reefs face a variety of threats globally,⁹ including overfishing,¹⁵ terrestrial pollution,¹⁰ global climate change, ocean acidification,²² and disease. Coral cover in the western Atlantic has declined almost 80% in the last 30 years,¹¹ leading to major shifts in reef structure and species composition in the region⁴; diseases, particularly those that cause tissue loss, are suspected to have played an important role in this decline.^{6,8,26} Diseases in corals are also being documented more frequently in the Pacific,³⁵ with recent examples including tissue loss diseases in *Acropora* sp from the Great Barrier Reef and the Marshall Islands,³¹ *Montipora* sp from Hawaii,² and *Porites* from the Philippines.²⁸

Lesions such as tissue loss, growth anomalies, or discoloration in corals are manifestations of disease.³⁹ Systematic descriptions of lesions at the gross and microscopic levels provide the foundational information for the case definition of animal diseases and provide a deductive process to assign potential causation to particular diseases.^{24,44} However, coral disease investigations have not traditionally followed a deductive approach and often lack descriptions of lesions at the microscopic level.³⁶ This limits the types of information available to interpret gross lesions and can hamper the understanding of coral disease. Now recognizing the utility of histopathology, recent studies are starting to include histology in their methods.³⁰

Field surveys are a staple of coral disease investigations and provide important demographic data on status and trends of various types of lesions in corals in the Atlantic³⁴ and Pacific.² To understand changes at the cellular level, combining field surveys with histopathology of gross lesions provides a robust method for disease investigations in corals. However, our knowledge of coral disease in the Pacific, particularly in remote atolls and regions of the South Pacific, is rudimentary to non-existent. Only 1 coral disease survey exists for Micronesia, and it is limited to the island of Guam.²⁰ As part of an interdisciplinary survey of coral reefs in the Federated States of Micronesia, our objective was to systematically characterize lesions

¹US Geological Survey, National Wildlife Health Center, Honolulu Field Station, Honolulu, HI, USA

²Hawaii Institute of Marine Biology, Kaneohe, HI, USA

³Woods Hole Oceanographic Institution, Woods Hole, MA, USA

Supplemental material for this article is available on the *Veterinary Pathology* website at <http://vet.sagepub.com/supplemental>.

Corresponding Author:

T. M. Work, US Geological Survey, National Wildlife Health Center, Honolulu Field Station, PO Box 50187, Honolulu, HI 96850, USA.

Email: thierry_work@usgs.gov

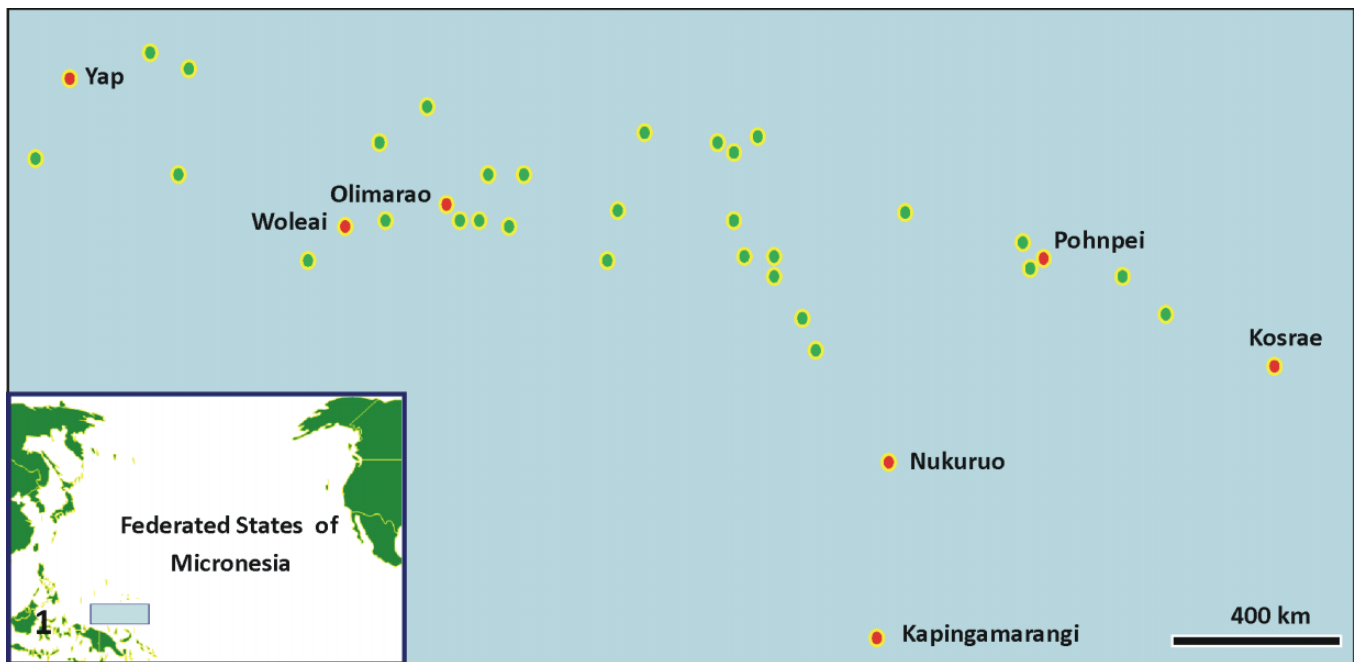


Figure 1. Map of the islands of Federated States of Micronesia (circles), located north of Papua New Guinea (see blue rectangle in inset map of Pacific). Red circles are islands where surveys were done.

encountered in corals in the region at the gross and microscopic levels. We report here on a variety of host responses and potential etiologic agents of disease in multiple species of corals, including the discovery of heretofore undescribed potential symbionts.

Methods

We surveyed corals for lesions in 7 islands in Federated States of Micronesia spanning the entire archipelago (Fig. 1). Two 25-m transects were laid end to end, separated by about 10 to 15 m and parallel to the reef crest at depths of 6 to 10 m, and corals were surveyed for lesions within a 6-m wide swath along the transect. All gross lesions on the transect were photographed and classified into 3 broad categories, including tissue loss, discoloration, and growth anomaly. Tissue loss was subdivided as follows: acute, where a distinct margin of tissues was apposed to bare white skeleton; subacute, where a distinct margin of tissues was apposed to a variably sized band of bare white skeleton that became progressively overgrown by turf algae, with increasing distance from the tissue margin; and chronic, where tissues were apposed to skeleton completely overgrown by epibiota.³⁹ Discoloration was classified as bleaching, dark, or other discoloration. Bleaching comprised tissues that were white, and it was subdivided as diffuse or localized. Dark discoloration comprised variably sized distinct irregular dark brown to black areas. Other discoloration included all other forms of abnormal tissue pigmentation. Growth anomalies were categorized as umbonate, exophytic, rugose, or nodular.⁴¹

Coral fragments (2–5 g) were collected with chisel or bone shears and placed into individually numbered Whirl-Pak bags

in seawater. Fragments with lesions were collected ensuring inclusion of the border between normal and lesional tissues. When available, paired grossly normal fragments were also collected. Fragments were fixed in Z-Fix (Anatech Ltd) diluted 1:5 with seawater and decalcified in dilute formic acid/formaldehyde solution (Cal-Ex II, Fischer Scientific). Tissues were then embedded in paraffin, sectioned at 5 μ m, and stained with hematoxylin and eosin.³⁸ To confirm presence of fungi or bacteria, Grocott's methenamine silver or Gram stains were used, respectively.²⁷

Microscopic changes were broadly categorized by agent associated with cell pathology, if present, and host response. Skeletal spaces were differentiated from gastrovascular canals based on cells lining the space; spaces lined by calicodermal cells were classified as decalcified skeleton, whereas those lined by gastrodermis or containing mesenterial filaments were classified as gastrovascular canals. Agents were identified according to their microscopic morphology and included sponges or cnidarian,¹³ helminths,¹⁴ algae,¹⁸ fungi,¹⁷ or crustacean.²⁹ Sponges consisted of metazoa with a matrix containing spicules, choanocytes, and presence or absence of zooxanthellae; cnidaria were metazoa with nematocysts; helminths were vermiform metazoa with or without a gut; algae were metazoa with cell walls; fungi were elongate branching filamentous structures with or without septa; and crustacea were metazoa with gut, muscle, reserve inclusion cells, cuticle, hepatopancreas, and segmented appendage. Host response or changes included tissue fragmentation, suspect wound repair, hyperplasia of basal body wall, necrosis, or inflammation.^{38,40,41,45} Tissue fragmentation comprised variably sized clumps of intact cells near the main intact tissue section; suspect wound repair

comprised regeneration of epidermis on exposed mesoglea of basal body wall or epidermal metaplasia of exposed calicodermis; hyperplasia of basal body wall comprised widespread proliferation of gastrodermis, mesoglea, and calicodermis of basal body wall with reduced formation or absence of mesenterial filaments and lack of polyp structures such as actinopharynx or tentacles; necrosis comprised cells manifesting cytoplasmic hypereosinophilia or shrinkage associated with pyknosis or karyorrhexis; inflammation comprised infiltrates of tissues with larger-than-normal amounts of mesogleal cells. Microscopic changes were not mutually exclusive, and in such cases, if an agent was associated with a lesion, it took priority when categories were assigned. If a host response was associated with only the lesion, the most severe change took priority during category assignment. Finally, we noted the presence/absence of symbiotic cell-associated microbial aggregates in the upper or basal body wall.³⁷ These are characterized by variably sized cell-associated clusters of symbiotic gram-negative bacteria in the gastrodermis or epidermis with no associated host response.

To aid in the interpretation of lesions, a series of supplemental figures are available highlighting diverse aspects and variations of normal microscopic anatomy of corals (see Supplemental Figs. S1–S12).

Results

We sampled a total of 76 colonies from 7 islands—including 17 colonies each from Kapingamarangi and Kosrae, 16 from Woleai, 12 from Pohnpei, 9 from Yap, 3 from Nukuro, and 2 from Olimarao—comprising 30 species of corals among 18 genera, with *Acropora*, *Porites*, and *Montipora* dominating. Of 76 colonies examined, tissue loss constituted the majority of lesions that we encountered (41%), followed by discoloration (30%) and growth anomaly (29%). Of 31 cases of tissue loss, most lesion samples were subacute (48%; Fig. 2), followed by acute (Fig. 3) and chronic (Fig. 4; 26% each). Of 23 samples with discoloration, 40% were dark discoloration (Figs. 5, 6), with bleaching (Fig. 7) and other discoloration (Fig. 8, 9) each comprising 30%. Of 22 growth anomalies, umbonate (Figs. 10, 11) composed half of those seen in 10 species of 6 genera. In addition, exophytic growth anomalies (Fig. 12) were seen in 1 each of *Acropora cerealis* and *Acropora surculosa*; nodular growth anomalies, in 3 *Lobophytum crassus* and 2 *Montipora grisea*; and rugose growth anomalies, in 1 each of *M. grisea*, *Montipora sp.*, *Porites evermanni*, and *Hydnophora microconos* (Supplemental Table 1).

For 9 cases of dark discoloration, fungal infections predominated (7 of 9, 77%); for 7 bleached corals, depletion of zooxanthellae from the gastrodermis made up a majority of microscopic diagnoses (4 of 7, 57%); and for growth anomalies other than umbonate, hyperplasia of the basal body wall was the most common microscopic finding (7 of 11, 63%). For the remainder of the gross lesions, no single microscopic finding composed >50% of the total.

Host response varied when agent was considered a primary microscopic diagnosis. For 10 cases with algae as the primary

agent (Figs. 13, 14), the dominant host response was fragmentation (6 of 10 cases, 60%), whereas necrosis was the dominant host response associated with cases having fungi as the primary agent (10 of 19, 52%; Figs. 15, 16). Crustacea (Fig. 17) and bivalves (Fig. 18) did not manifest an evident host response, and the single case of helminth infestation was associated with fragmentation.

For primary diagnoses comprising host responses, there was variation in associated microscopic lesions. Of 11 cases of fragmentation, wound repair was the most common associated finding (6 of 11, 54%; Fig. 19). In cases of depletion of zooxanthellae, atrophy was the most common associated finding (4 of 6, 66%; Fig. 20). Most cases of necrosis (7 of 8, 87%; Fig. 21) had no associated lesions, and 7 of 10 (70%) cases of hyperplasia of the basal body wall (Fig. 22) had no associated microscopic findings. Hyperplasia of the epidermis was seen in only a single umbonate growth anomaly (Fig. 23). Of 12 corals manifesting microscopic evidence of inflammation, *Porites* (5 of 12, 41%) and *Montipora* (4 of 12, 33%) composed the majority of genera, with *Isopora*, *Lobophytum* and *Acropora* having 1 instance each.

Two metazoans closely associated with coral tissues elicited no host response in both normal and diseased tissues. One metazoan was elongate with numerous nematoblasts and nematocytes compatible in morphology with microcnidaria (Fig. 24). These were found in 2 *Porites sps* from Pohnpei, 1 *M. grisea* from Kapingamarangi, and 1 *Porites rus* from Kosrae. Another apparent symbiont comprised unidentified multicellular (2–4 nuclei) organisms within the mesoglea of a single *M. grisea* from Kapingamarangi (Fig. 25).

Of 67 paired normal tissues, 27, 22, and 18 originated from colonies with tissue loss, growth anomalies, and discoloration, respectively. Seventy-one percent of paired normal fragments had no microscopic lesions, whereas lesions were seen for 7 of 27 (26%) paired normal fragments for tissue loss, 8 of 22 (36%) for growth anomaly, and 4 of 18 (22%) for discoloration. For the 7 paired normal fragments from tissue loss colonies, 3 had fragmentation, and 1 each had fungi, algae, sponges, or inflammation. Of the 8 paired grossly normal fragments from growth anomaly colonies, 5 had fungi, and 3 had necrosis. Of the 3 paired normal fragments from discolored colonies, 2 had fungi, and 1 had inflammation.

Cell-associated microbial aggregates were seen in corals from Woleai, Kosrae, Kapingamarangi, Nukuro, and Pohnpei, with *A. cerealis*, *A. hyacinthus*, *A. surculosa*, *Galaxea fascicularis*, *Hydnophora exesa*, *Platygyra daedala*, *Porites cylindrica*, and *P. evermanni* infected.

Discussion

The findings in this study reinforce a concept that distinct gross lesions in corals have multiple potential etiologies and host responses, thus highlighting the utility of histopathology in coral disease surveillance. In this study, the most common agents associated with tissue loss were algae and fungi, and host response was most commonly fragmentation and necrosis.

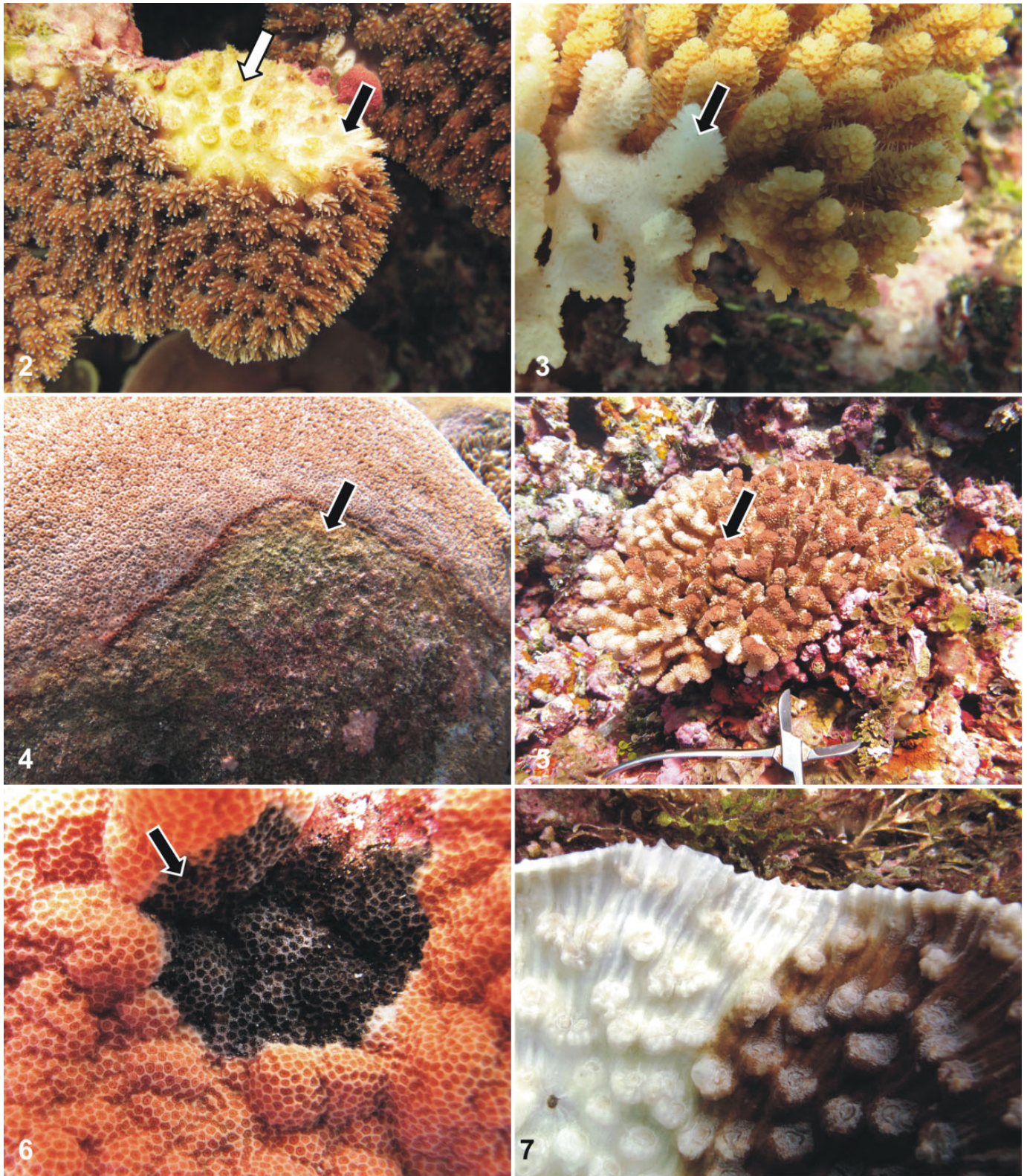


Figure 2. Subacute tissue loss, *Galaxea fascicularis* from Kosrae. Note band of bare white skeleton bereft of tissues (black arrow) progressing to green algal turf cover (white arrow). **Figure 3.** Acute tissue loss, *Acropora surculosa* from Woleai. Note bare white intact skeleton bereft of tissues (arrow). **Figure 4.** Chronic tissue loss, *Astreopora myriophthalma* from Woleai. Note distinct area of skeleton overgrown by turf and coralline algae (arrow) separated from normal tissues by a thin dark band. **Figure 5.** Dark discoloration, *Pocillopora verrucosa* from Woleai. Note dark brown branch tips (arrow) that, on microscopy, had fungal invasion. **Figure 6.** Dark discoloration (arrow), *Porites* sp from Nukuruo that, on microscopy, had fungal invasion. **Figure 7.** Diffuse bleaching, *Mycedium robokaki* from Kosrae. Note diffuse white discoloration (left) demarcated from normal brown tissue (right).

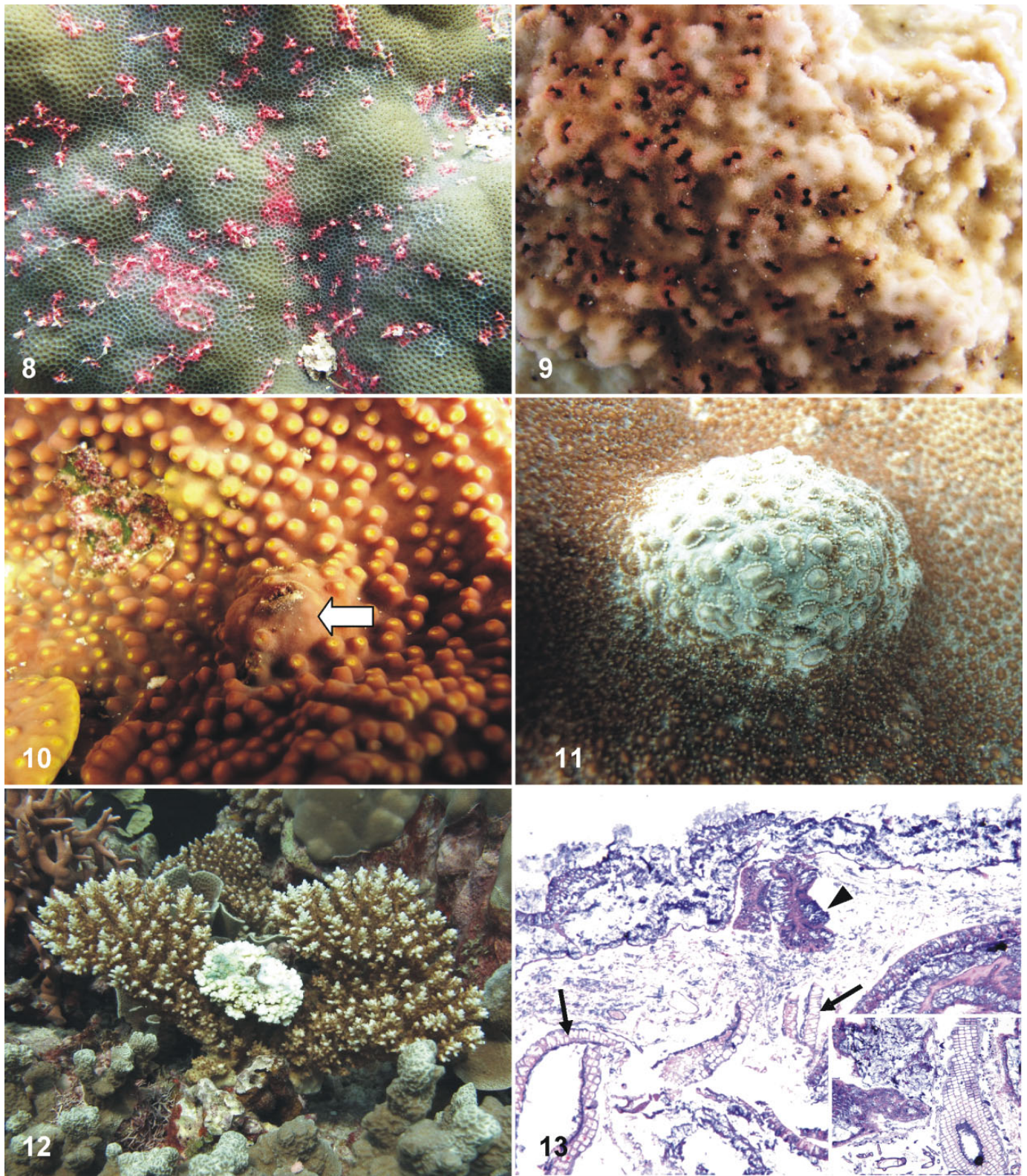


Figure 8. Multifocal pink discoloration, *Porites* sp from Kapingamarangi. Algae and inflammation were seen on microscopy. **Figure 9.** Multifocal dark discoloration, *Montipora* sp from Kapingamarangi. On cut surface and on histology, bivalve molluscs were associated with discoloration. **Figure 10.** Umbonate growth anomaly (arrow), *Turbinaria reniformis* from Pohnpei. Histologically, this was associated with a crustacean. **Figure 11.** Umbonate growth anomaly, *Hydnophora excesa* from Kosrae, cause unknown. **Figure 12.** Exophytic growth anomaly, *Acropora cerealis* from Kosrae, cause unknown. **Figure 13.** Subacute tissue loss, *Galaxea fascicularis* from Kosrae in Figure 2. Note macroalgae (arrows) associated with fragmented tissues (arrowhead). Inset: higher magnification of algae with cell walls (right) and tissue fragment (left). Hematoxylin and eosin.

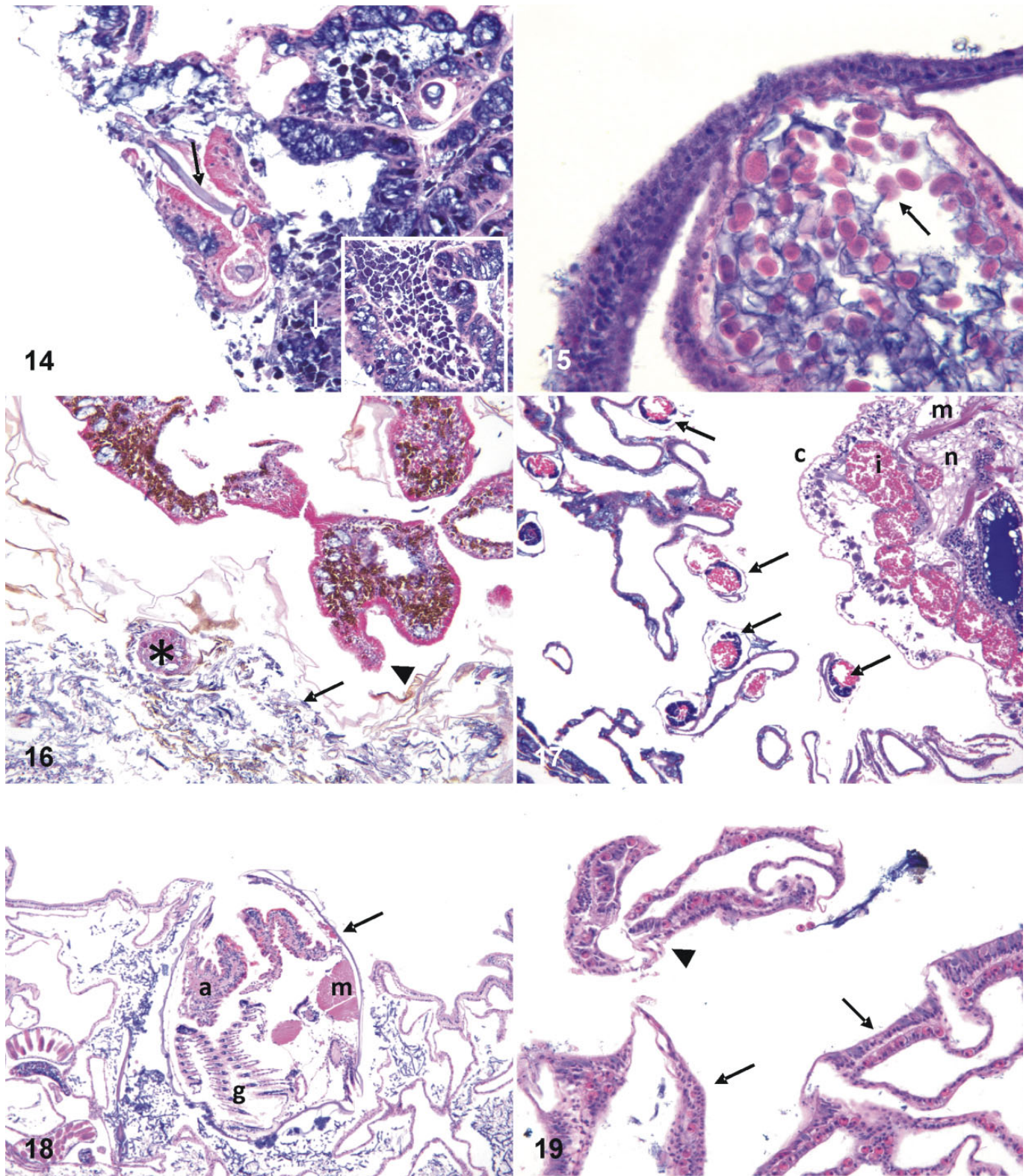


Figure 14. Multifocal pink discoloration, *Porites* sp (Fig. 8). Note filaments of algae (black arrow) surrounded by hypereosinophilic fragmented tissues and marked adjacent infiltrates of granular brown cells (white arrow). Inset: higher magnification of granular brown cells that, on other studies,²¹ have stained positive with melanin. Hematoxylin and eosin (HE). **Figure 15.** Dark discoloration, *Pocillopora verrucosa* (Fig. 5). Note ovoid to reniform eosinophilic fungal fruiting bodies within skeleton near upper body wall (arrow). HE. **Figure 16.** Dark discoloration, *Porites* sp (Fig. 6). Note mats of fungal hyphae (arrow) associated with fragmentation and necrosis of adjacent basal body wall (asterisk) manifesting hypertrophy of calicoderms (arrowhead). HE. **Figure 17.** Umbonate growth anomaly, *Turbinaria reniformis* (Fig. 10). Note large metazoan

In other studies of tissue loss in *Acropora* from the Pacific, algae, fungi, helminths, and sponges were associated with various host responses, including wound repair, fragmentation, and necrosis.³⁸ A longitudinal study of tissue loss in *Montipora capitata* in Kaneohe Bay Hawaii⁴⁵ revealed ciliates commonly associated with rapidly progressing tissue loss (acute tissue loss) and helminths or chimeric parasites⁴³ associated with slowly progressing tissue loss (subacute tissue loss). Thus, a pattern continues to be confirmed with tissue loss diseases in corals in the Indo-Pacific that are associated with a variety of hosts responses and potential etiologic agents, even when the condition is examined for a single species and a single location.

Discoloration included bleaching, dark discoloration, pink multifocal discoloration in *Porites*, and discoloration secondary to burrowing molluscs. As per other instances of bleaching in corals,⁷ the typical histologic finding for this lesion was depletion of zooxanthellae often associated with atrophy of tissues. Prolonged bleaching leads to loss of tissue biomass in other coral species.³² Dark discoloration was associated with fungal infections in 80% of the corals. Fungi were associated with foci of necrosis and typically invaded the lower and upper skeleton, often with formation of structures compatible in morphology with fruiting bodies near the surface body wall. These findings are consistent to those found in *Montipora* and *Pavona* from Hawaii and American Samoa, where corals with dark discoloration manifested endolithic hypermycosis.⁴² Hence, this represents a range extension of this condition to Micronesia. Future studies for this disease should focus on identifying the fungi associated with these lesions and the drivers that promote overgrowth of skeleton. The presence of fruiting bodies only near the epidermis was an interesting phenomenon. Given that light does not influence distribution of endolithic fungi,¹² other drivers must be influencing fruiting body production near the upper body wall in corals, and these merit further exploration. Other discolorations included multifocal pink discoloration in *Porites* associated with algal infiltrations and multifocal dark pinpoint cavities in *Montipora* sp. that appeared as little dark spots associated with endolithic bivalves. Other endolithic organisms, such as barnacles, associated with multifocal punctate cavities have been documented in a variety of corals in the Pacific and Atlantic.³ Our findings emphasize that pink spots in corals are not invariably associated with trematode larvae,¹ as pointed out elsewhere.⁵ *Porites* trematodiasis has a very distinct "pink spot" that is greatly swollen and never has pinpoint cavities, sediment, or algae observed on the swelling. Careful observation of pink spots in the field, followed by histologic examination, is required to discriminate trematodiasis from other causes.

Growth anomalies were common lesions in corals throughout Micronesia. Like growth anomalies in *Acropora* elsewhere in the Pacific,⁴¹ growth anomalies in *Acropora* from Micronesia

manifested as hyperplasia of the basal body wall occasionally associated with necrosis. A similar finding predominated in nodular and rugose growth anomalies in other species of corals, and others have found hyperplasia of basal body wall in the Caribbean.²⁵ However, umbonate growth anomalies seen in a variety of species here had a greater range of microscopic manifestations above and beyond hyperplasia of the basal body wall, including epidermal hypertrophy, bleaching, fragmentation, fungal infections, and endolithic crustacea. The crustacea were seen exclusively in *Turbinaria*, and their morphology with a series of rootlets distributed locally within the skeleton is consistent with rhizocephalans, parasitic barnacles often found in crabs that also grow rootlets throughout their host.³³ The coral skeleton surrounding the rootlets was thicker than surrounding normal tissues, a finding also observed with crabs that cause growth anomalies in corals.¹⁶ Determining the identity of this crustacean and whether it caused the skeletal growth anomaly would require additional investigations.

We also identified 2 new organisms in coral tissue, regardless of disease state, that were not associated with any evident host cell pathology. As such, we suggest that these organisms are new putative symbionts in corals. The first of these were found in *Porites* and *Montipora* and contained distinct nematocysts (stenoteles) characteristic of hydrozoa.¹³ Symbiotic hydroids have been found attached to the surface body wall of corals,^{19,23} but no cnidaria have been reported deep in the skeleton as seen here. The other putative symbiont was a distinct, small, multicellular organism (metazoan) in the mesoglea of *M. grisea*. This metazoan was smaller and within the mesoglea, distinguishing it from another metazoan in this genus, a chimeric parasite from *M. capitata* that is located within gastrovascular canals and is associated with tissue loss.⁴³ Elucidating the identity of both organisms may be difficult, as they were found infrequently and in low numbers.

The pattern of prevalence of cell-associated microbial aggregates in corals in Micronesia mirrored that seen elsewhere in the Pacific,³⁷ with *Platygyra*, *Acropora*, and *Porites* being commonly infected with total absence of these organisms in *Montipora*. This study expands the range of species infected with these aggregates to include *A. cerealis*, *A. surculosa*, *G. fascicularis*, and *H. exesa*. Cell-associated microbial aggregates are tissue-associated bacteria that are found in high prevalence in dominant genera of corals in the Indo-Pacific, such as *Pocillopora*, *Acropora*, and *Porites*. They are thought to be facultative symbionts that are important to coral health and immunity; they could also have an important evolutionary role, possibly contributing to the dominance of specific coral genera on coral reefs.³⁷ The documentation of their presence in Micronesia expands their range in the Pacific, and confirming their role in coral biology merits further study.

Figure 17. Continued. (crustacean) with striated muscle (m), reserve inclusion cells (i), nervous tissue (n), and cuticle (c) surrounded by rootlets (arrows). HE. **Figure 18.** Multifocal dark discoloration, *Montipora* sp (Fig. 9). Note mollusc (arrow) with gills (g), mantle (a), and muscles (m) embedded in coral with little host response (epidermis on top) protruding from surface body wall and corresponding to black dots. HE. **Figure 19.** Acute tissue loss, *Acropora surculosa* (Fig. 3). Note tissue fragmentation (arrowhead) and regeneration of epidermis (arrows). HE.

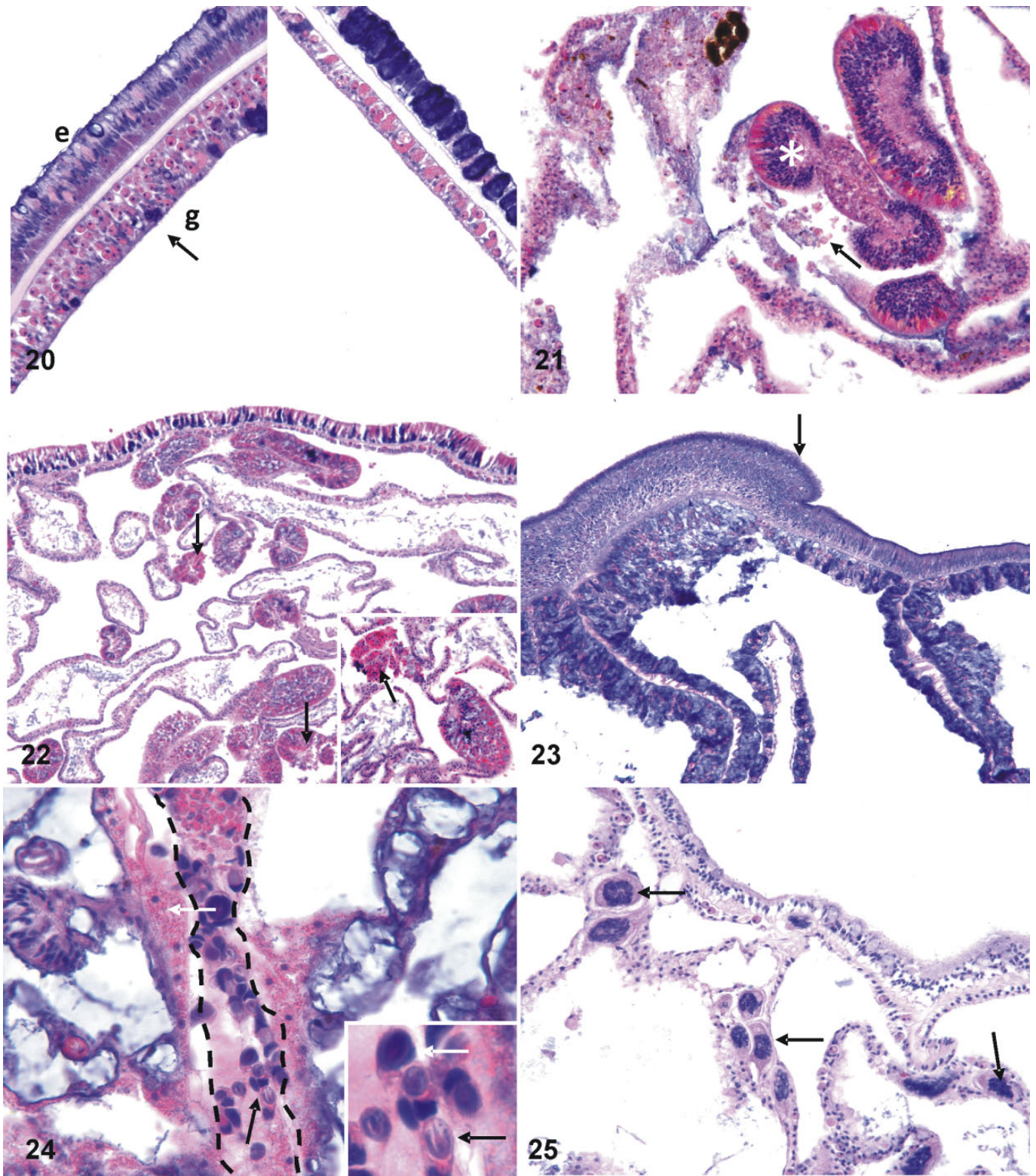


Figure 20. Bleaching, *Mycedium robokaki* (Fig. 7). Left panel is normal upper body wall with tall columnar epithelium (e) with plump gastrodermis (g) replete with zooxanthellae (arrow); contrast with right panel, showing depleted zooxanthellae in thin gastrodermis and thin epidermis where cells have mostly atrophied and are effaced by basophilic mucocytes. **Figure 21.** Histology of chronic tissue loss in *Astreopora myriophthalma* (Fig. 4). Note necrosis and dissociation (arrow) of mesenterial filaments (asterisk). Hematoxylin and eosin (HE). **Figure 22.** Histology of *Acropora cerealis* with exophytic growth anomaly (Fig. 12). Note hyperplasia of body wall with lack of evident polyp formation on upper body wall and necrotic mesenterial filaments (arrows) characterized by cytoplasmic hypereosinophilia and pyknosis. Inset: higher magnification of necrotic

Twenty-eight percent of paired grossly normal fragments had a microscopic lesion. This phenomenon has been documented elsewhere and indicates that some coral lesions at the microscopic level will extend to tissues that appear grossly normal.⁴⁵ For example, in this study, 11% of paired normal fragments from lesions with fungi also had fungal infections associated with cell pathology, suggesting that fungal infections may be more widely disseminated than suggested by gross lesions alone. In other studies, certain microscopic lesions—such as chimeric parasites in *M. capitata* with tissue loss (in Hawaii)—were found to be more systemically distributed and likely to be seen in both normal and lesional tissues than ciliates or helminths, which were more restricted to grossly abnormal tissues.⁴⁵ Therefore, gross observations do not always provide clear-cut results between healthy and diseased tissues. However, the findings here provide important baseline information on coral disease and should serve as a foundation for future investigations to understand the role of particular agents in causing lesions.

Acknowledgements

Dr Douglas Fenner graciously assisted with the identification of corals. We thank Ray Dalio and the Dalio Family Foundation for their support of the Woods Hole Oceanographic Institution Access to the Sea program, through which this work was partially funded, and the crew of the *R/V Alucia* for logistical support. Susan Knowles and anonymous reviewers provided constructive comments. Mention of products or trade names does not imply endorsement by the US government.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

References

1. Aeby GS. A digenean metacercaria from the reef coral, *Porites compressa*, experimentally identified as *Podocotyloides stenometra*. *J Parasitol*. 1998;**84**: 1259–1261.
2. Aeby GS, Ross M, Williams GJ, et al. Disease dynamics of *Montipora* white syndrome within Kaneohe Bay, Oahu, Hawaii: distribution, seasonality, virulence, and transmissibility. *Dis Aquat Organ*. 2010;**91**:1–8.
3. Anderson DT. Structure, function and phylogeny of coral-inhabiting barnacles (Cirripedia, Balanoidea). *Zool J Linnean Soc*. 1992;**106**:277–339.
4. Aronson RB, Precht WF. White-band disease and the changing face of Caribbean coral reefs. *Hydrobiologia*. 2001;**460**:25–38.
5. Benzoni F, Galli P, Pinchon M. Pink spots on *Porites*: not always a coral disease. *Coral Reefs*. 2009;**29**:153.
6. Borger JL, Steiner SCC. The spatial and temporal dynamics of coral diseases in Dominica, West Indies. *Bull Mar Sci*. 2005;**77**:137–154.
7. Brown BE, Le Tissier MDA, Bythell JC. Mechanisms of bleaching deduced from histological studies of reef corals sampled during a natural bleaching event. *Mar Biol*. 1995;**122**:655–663.
8. Bruckner AW, Bruckner RJ, Williams EH Jr. Spread of a black-band disease epizootic through the coral reef system in St Ann's Bay, Jamaica. *Bull Mar Sci*. 1997;**61**:919–928.
9. Carpenter KE, Abrar M, Aeby G, et al. One-third of reef-building corals face elevated extinction risk from climate change and local impacts. *Science*. 2008;**321**:560–563.
10. Fabricius KE. Effects of terrestrial runoff on the ecology of corals and coral reefs: review and synthesis. *Mar Pollut Bull*. 2005;**50**:125–146.
11. Gardner TA, Cote IM, Gill JA, et al. Long-term region-wide declines in Caribbean corals. *Science*. 2003;**301**:958–960.
12. Golubic S, Radtke G, Le Campion-Alsumard T. Endolithic fungi in marine ecosystems. *Trends Microbiol*. 2005;**13**:229–235.
13. Hyman LH. *The Invertebrates: Protozoa Through Ctenophora*. New York, NY: McGraw-Hill; 1940.
14. Hyman LH. *Platyhelminthes and Rhynchocoela, the Acoelomate Bilateria*. New York, NY: McGraw-Hill; 1940.
15. Jackson JBC, Kirby MX, Berger WH, et al. Historical overfishing and the recent collapse of coastal ecosystems. *Science*. 2001;**293**:629–637.
16. Johnsson R, Neves E, Franco GMO, et al. The association of two gall crabs (Brachyura: Cryptochiridae) with the reef-building coral *Siderastrea stellata* Verrill, 1868. *Hydrobiologia*. 2006;**559**:379–384.
17. Larone DH. *Medically Important Fungi: A Guide to Identification*. Washington, DC: American Society for Microbiology; 1976.
18. McCook LJ, Jompa J, Diaz-Pulido G. Competition between corals and algae on coral reefs: a review of evidence and mechanisms. *Coral Reefs*. 2001;**19**: 400–417.
19. Montano S, Maggioni D, Galli P, et al. *Zanclus*–coral association: new records from Maldives. *Coral Reefs*. 2013;**32**:701.
20. Myers RL, Raymundo LJ. Coral disease in Micronesian reefs: a link between disease prevalence and host abundance. *Dis Aquat Organ*. 2009;**87**:97–104.
21. Palmer CV, Mydlarz LD, Willis BL. Evidence of an inflammatory-like response in non-normally pigmented tissues of two scleractinian corals. *Proc Royal Soc B*. 2008;**275**:2687–2693.
22. Pandolfi JM, Connolly SR, Marshall DJ, et al. Projecting coral reef futures under global warming and ocean acidification. *Science*. 2011;**333**:418–422.
23. Pantos O, Bythell JC. A novel reef coral symbiosis. *Coral Reefs*. 2010;**29**: 761–770.
24. Peters EC. A survey of cellular reactions to environmental stress and disease in Caribbean scleractinian corals. *Helgolander Meeresun*. 1984;**37**:113–137.
25. Peters EC, Halas JC, McCarty HB. Calicoblastic neoplasms in *Acropora palmata*, with a review of reports on anomalies of growth and form in corals. *J Natl Cancer Inst*. 1986;**76**:895–912.
26. Porter JW, Dustan P, Jaap WC, et al. Patterns of spread of coral disease in the Florida keys. *Hydrobiologia*. 2001;**460**:1–24.
27. Prophet EB, Mills B, Arrington JB, et al. *Laboratory Methods in Histotechnology*. Washington, DC: Armed Forces Institute of Pathology; 1992.
28. Raymundo LJ, Rosell KB, Reboton CT, et al. Coral diseases on Philippine reefs: genus *Porites* is a dominant host. *Dis Aquat Organ*. 2005;**64**:181–191.
29. Ruppert AE, Fox RS, Barnes RD. *Invertebrate Zoology*. Pacific Grove, CA: Brooks/Cole; 2004.

Figure 22. Continued. mesenterial filaments (arrow; upper left) contrasted with normal filament (lower right). Note hypereosinophilia and pyknosis. HE. **Figure 23.** Histology of *Hydnophora excesa* with umbonate growth anomaly (Fig. 11). Contrast hyperplastic epidermis on growth anomaly (arrow) from adjacent normal tissue on right. HE. **Figure 24.** *Porites rus*. Note vermiform metazoan (outlined by dotted line) with stenoteles (nematocysts: black arrow). Calicodermis adjacent to metazoan is hypertrophied with granular eosinophilic cytoplasm (white arrow). Inset: stenotele consisting of capsule, central barb, and coils visible as dark spots around barb (black arrow). Dark cells near stenotele (white arrow) are nematoblasts (immature nematocysts). HE. **Figure 25.** *Montipora* sp. Note small metazoa within mesoglea of upper and basal body wall (arrows); epidermis is on top. HE.

30. Sudek M, Work TM, Aeby GS, et al. Histological observations in the Hawaiian reef coral, *Porites compressa*, affected by Porites bleaching with tissue loss. *J Invertebr Pathol*. 2012;**111**:121–125.
31. Sussman M, Willis BL, Victor S, et al. Coral pathogens identified for white syndrome (WS) epizootics in the Indo-Pacific. *PLOS One*. 2008;**3**:e2393.
32. Szmant A, Grassman N. The effects of prolonged “bleaching” on the tissue biomass and reproduction of the reef coral *Montastrea annularis*. *Coral Reefs*. 1990;**8**:217–224.
33. Walker G. Introduction to the rhizocephala (Crustacea: Cirripedia). *J Morphol*. 2001;**249**:1–8.
34. Weil E, Croquer A. Spatial variability in distribution and prevalence of Caribbean scleractinian coral and octocoral diseases: I. Community-level analysis. *Dis Aquat Organ*. 2009;**83**:195–208.
35. Willis BL, Page CA, Dinsdale EA. Coral disease on the Great Barrier Reef. In: Loya Y, Rosenberg E, eds. *Coral Health and Disease*. Heidelberg, Germany: Springer; 2004: 69–104.
36. Work T, Meteyer C. To understand coral disease, look at coral cells [published online April 11, 2014]. *Ecohealth*.
37. Work TM, Aeby GS. Microbial aggregates within tissues infect a diversity of corals throughout the Indo-Pacific. *Marine Ecology Progress Series*. 2014;**500**:1–9.
38. Work TM, Aeby GS. Pathology of tissue loss (white syndrome) in *Acropora* sp. corals from the Central Pacific. *J Invertebr Pathol*. 2011;**107**:127–131.
39. Work TM, Aeby GS. Systematically describing gross lesions in corals. *Dis Aquat Organ*. 2006;**70**:155–160.
40. Work TM, Aeby GS. Wound repair in *Montipora capitata*. *J Invertebr Pathol*. 2010;**105**:116–119.
41. Work TM, Aeby GS, Coles SL. Distribution and morphology of growth anomalies in *Acropora* from the Indo-Pacific. *Dis Aquat Organ*. 2008;**78**:255–264.
42. Work TM, Aeby GS, Stanton FG, et al. Overgrowth of fungi (endolithic hypermycosis) associated with multifocal to diffuse distinct dark discoloration of corals in the Indo-Pacific. *Coral Reefs*. 2008;**27**:663.
43. Work TM, Forsman ZH, Szabó Z, et al. Inter-specific coral chimerism: genetically distinct multicellular structures associated with tissue loss in *Montipora capitata*. *PLOS One*. 2011;**6**:e2869.
44. Work TM, Richardson LL, Reynolds TR, et al. Biomedical and veterinary science can increase our understanding of coral disease. *J Exp Mar Biol Ecol*. 2008;**362**:63–70.
45. Work TM, Russell R, Aeby GS. Tissue loss (white syndrome) in the coral *Montipora capitata* is a dynamic disease with multiple host responses and potential causes. *Proc Royal Soc B*. 2012;**279**:4334–4341.