



Effects of *Coralliophila violacea* on tissue loss in the scleractinian corals *Porites* spp. depend on host response

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ABSTRACT: We investigated interactions between the corallivorous gastropod *Coralliophila violacea* and its preferred hosts *Porites* spp. Our objectives were to experimentally determine whether tissue loss could progress in *Porites* during or after *Coralliophila* predation on corals with and without tissue loss and to histologically document snail predation. In 64 % of feeding scars, tissue regenerated within 3 wk, leaving no trace of predation. However, in roughly 28 % of scars, lesions progressed to subacute tissue loss resembling white syndrome. In feeding experiments, scars from snails previously fed diseased tissue developed progressive tissue loss twice as frequently as scars from snails previously fed healthy tissue. Scars from previously healthy-fed snails were 3 times as likely to heal as those from previously diseased-fed snails. Histology revealed marked differences in host responses to snails; *P. cylindrica* manifested a robust inflammatory response with fewer secondary colonizing organisms such as algae, sponges, and helminths, whereas *P. rus* showed no evident inflammation and more secondary colonization. We conclude that lesion progression associated with *Coralliophila* may be associated with secondary colonization of coral tissues damaged by predator-induced trauma and necrosis. Importantly, variation at the cellular level should be considered when explaining interspecific differences in host responses in corals impacted by phenomena such as predation.

KEY WORDS: *Coralliophila* · Coral disease · White syndrome · *Porites* · Guam

INTRODUCTION

It is thought that organisms that prey on or compete with corals can potentially transmit infectious agents resulting in lesions via physical trauma, deposition of fecal waste, or physical contact. If such organisms carry the infectious agent, they are considered vectors, and an increase in the abundance of a vector may then result in increased disease transmission. In corals, conclusive evidence for the existence of vectors is rare; the fireworm *Hermodice carunculata* is the only known case of a corallivore potentially transmitting a bacterial agent to its host coral (Sussman et

al. 2003). However, several recent studies have suggested that organisms that interact with coral may be implicated in certain infections, although the mechanisms for this effect are often unclear. For instance, physical contact between corals and the macroalga *Halimeda opuntia* facilitated white plague (Nugues et al. 2004). Crown-of-thorns starfish *Acanthaster planci* have been implicated in brown band disease (Nugues & Bak 2009), and corallivorous snails may transmit a pathogen associated with white syndrome (Antonius & Riegl 1997, Williams & Miller 2005). Raymundo et al. (2009) revealed a significant positive correlation between the abundance of obligate coral-

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livorous butterflyfish and coral disease prevalence; in reefs protected from fishing, corallivorous chaetodontids were less abundant, as were coral diseases.

Conversely, by debriding damaged tissues, predators can also reduce lesion impacts in corals. For instance, certain corallivorous fish prefer diseased or damaged coral tissue (McIlwain & Jones 1997, Aeby 2002), and animals feeding on diseased tissue can slow the progression of lesions (Cole et al. 2009). Crabs can also slow progression of white syndrome in *Acropora* (Pollock et al. 2013).

Implicit in the scenario of corallivores as vectors is a tacit assumption that these organisms are transmitting, exacerbating, or alleviating infectious diseases in corals. However, many of the diseases with which these competitors or predators are associated have no well-defined causes, particularly those involving tissue loss (Work & Meteyer 2014). Concluding that an interacting organism is a vector requires not only the presence of the suspect infectious agent in the organism, but also evidence that this agent is associated with cell death in the coral. Currently, the main evidence used to incriminate predators as vectors of coral diseases is gross evidence of lesion progression in corals in the presence of predators (Work & Meteyer 2014). However, an equally plausible explanation for this observation is that, by damaging tissues, coral predators create portals of entry for secondary colonizers that either exacerbate lesions or prevent healing. In either case, understanding the nature of interactions between host corals and corallivores can elucidate patterns of disease on coral reefs, particularly in light of the fact that at least some of these organisms, such as crown-of-thorns starfish (De'ath et al. 2012) or *Drupella* (Moyer et al. 1982), can reach outbreak population densities.

Coralliphila violacea is a muricid corallivorous snail that prefers poritid corals in the Indo-Pacific and Red Sea. The unique feeding mode of this snail, referred to as 'prudent feeding,' involves insertion of a proboscis into the coral polyp's coelenteron, allowing the snail to remain stationary for extended periods while consuming coral tissues (Ward 1965, Brawley & Adey 1982). Snails remain firmly attached for months, killing the underlying tissue and creating a distinct scar surrounded by accreted coral skeleton. Snails are often clustered on massive corals but singly on branching corals (Chen et al. 2004), and this creates variably sized zones of tissue injury that stimulate host tissue regeneration. In *Porites*, Oren et al. (1998) documented translocation of carbon into these injury zones from surrounding tissue to promote

healing, with the net result of creating a continual supply of food for *C. violacea*.

In Guam, acute to subacute tissue loss (white syndrome) in *Porites* is highly prevalent (Myers & Raymundo 2009), and our surveys of coral diseases occasionally noted progressive tissue loss beyond *Coralliphila* feeding scars on *Porites* spp. Further, some tissue-loss lesions sometimes displayed signs of a snail scar within the lesion. To shed light on the role of this snail in *Porites* tissue loss, we set out to (1) experimentally determine whether tissue loss could progress in *Porites* in the presence of *Coralliphila* or in association with a recent feeding scar; and (2) document histology of snail predation to better understand potential underlying mechanisms of tissue damage and host response.

MATERIALS AND METHODS

All studies were carried out between March and December 2011 in the University of Guam Marine Lab wet lab (tank studies) and at Luminao Reef, Guam (field observations). Corals for histology were collected from Luminao and Gabgab reefs, fixed, and sent to the National Wildlife Center Honolulu Field Station, Hawaii, USA, for processing and analysis.

To examine the fate of scars *in situ*, 25 colonies of the branching coral *Porites cylindrica* hosting a snail, but with no gross clinical signs of tissue loss, were tagged in Luminao reef, Guam, and the snail was removed. The scar was photographed and condition of the tissue margin noted. Scars were photographed weekly for 3 wk after snail removal and scored as (1) healing: tissue deposition over dead skeleton along the scar margin; (2) static: an eroded scar margin with no tissue deposition; or (3) progressing: continued tissue loss beyond the original scar border. To test for the ability of snails to transmit tissue loss between colonies, 40 plates of *P. rus*, ranging from 4 to 8 cm in diameter, were collected from Gabgab reef, Guam. Ten plates had acute to subacute tissue loss, and 30 plates were clinically healthy on collection. Twenty adult snails were collected from the same reef. The 10 plates containing lesions ('diseased plates') and 10 apparently healthy plates (corals with no gross evidence of lesions) were placed in individual 2 l aerated aquaria and randomly arranged in water baths at the University of Guam Marine Lab. Snails were starved for 24 h, then randomly distributed among the 20 fragments. Snails assigned to diseased fragments were placed on the margin of an active lesion, while snails assigned to

healthy fragments were haphazardly placed on healthy tissue. Snails were allowed to feed for 1 wk, after which they were removed and immediately placed on the remaining 20 healthy fragments, randomized in additional aquaria. We tracked which fragments were fed upon by snails that had previously fed on tissue-loss lesions vs. those previously fed apparently healthy fragments. Snails were allowed to feed for an additional week and then removed. Resultant scars were monitored for 2 wk and scored as healing, static, or progressing, as described above. Chi-squared tests were used to test whether the fate of feeding scars differed between treatments (diseased-fed vs. healthy-fed), and ANOVAs were used to examine differences in the rate of lesion progression or healing. Data were transformed, where necessary, to meet assumptions of normality.

Histology of snail predation scars on coral tissue was examined on 11 and 13 colonies each of *P. cylindrica* and *P. rus*, collected from Luminao reef (*P. cylindrica*) and Gabgab reef (*P. rus*) in August 2010 and immediately fixed in Z-fix® concentrate diluted 1:5 in seawater as per the manufacturer's recommendations. Gross lesions were classified as recent (≤ 10 d old; no skeletal accretion and partial tissue loss) or old (> 10 d; skeletal accretion apparent and complete tissue loss). Tissues were processed for histology and interpretation as described by Work et al. (2012). Briefly, tissues were decalcified, embedded in paraffin, sectioned at 5 μm , and stained with hematoxylin and eosin, stains adequate to identify cyanobacteria (Sato et al. 2010) and Gram-positive or Gram-negative microcolonies of bacteria (Work & Aeby 2014). On microscopy, changes were classified as 'host response' or 'organisms associated with host response.' Host responses included necrosis, deposition of hyaline or basophilic membranes, fragmentation, cleft formation, hypertrophy, atrophy, or infiltration of mesogleal cells (inflammation). Organisms were categorized as helminths, fungi, algae, or ciliates.

RESULTS

Of the 25 snail scars monitored in intact colonies *in situ*, the majority (64 %) healed within 3 wk (Fig. 1). Healing lesions had intact margins with healthy tissue overgrowing bare skeleton. Two scars remained

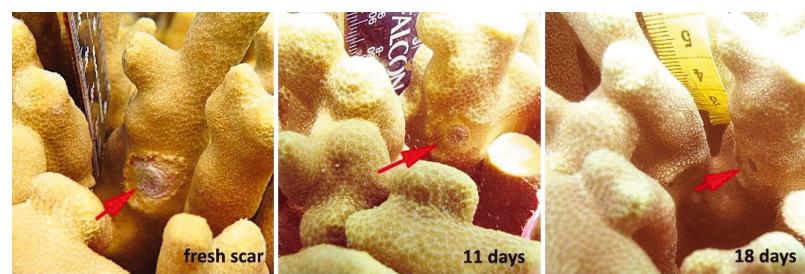


Fig. 1. Time series photographs of a colony of *Porites cylindrica* with a single healing *Coralliophila violacea* scar (red arrow). Note the skeletal accretion around the new scar and the tissue deposition that resulted in rapid healing

static, with a margin of skeletal accretion surrounding the scar with no evident regeneration of coral tissues after snail removal. Seven scars (28 %) progressed, showing gross signs of tissue loss similar to white syndrome.

In the laboratory feeding experiment, several plates developed more than 1 feeding scar due to snail movement (Fig. 2). We tracked the fate of each scar formed and later pooled the data for statistical tests, after tests for an effect of source plate on lesion fate revealed no significant differences. The majority of feeding scars on corals exposed to snails previously fed diseased tissue exhibited progressing lesions after snails were removed, while the majority of scars formed by snails previously fed healthy tissue healed (Table 1; $\chi^2 = 4.891$; $p = 0.0270$). Interestingly, the rate at which scars either healed or formed progressing lesions was not significantly different between treatments (roughly 0.25 cm d^{-1} ; ANOVA $F_{1,24} = 0.8168$; $p = 0.385$), although the apparent difference in tissue loss rate in plates with healthy-fed snails (Table 1) was due to a single lesion that progressed very rapidly. When that lesion was removed from the analysis, the rate at which lesions progressed in healthy-fed scars was reduced to 0.16 ± 0.09 (SD) cm d^{-1} , but this difference was still not significant (ANOVA $F_{1,24} = 0.013$; $p = 0.908$).

A number of host responses to snail predation were observed histologically (Table 2). The most common host response for both *P. cylindrica* and *P. rus* was deposition of basophilic membranes either on the surface (Fig. 3A) or within (Fig. 4A) tissues, necrosis with hyaline membrane formation (Fig. 3A–D), and ablation of the surface body wall (Figs. 3A & 4A). For *P. cylindrica*, surface body wall ablation, hypertrophied calicodermis, and mucus cell hypertrophy were seen exclusively in new snail lesions (Fig. 3), whereas cleft formation between surface and basal body wall was present only in

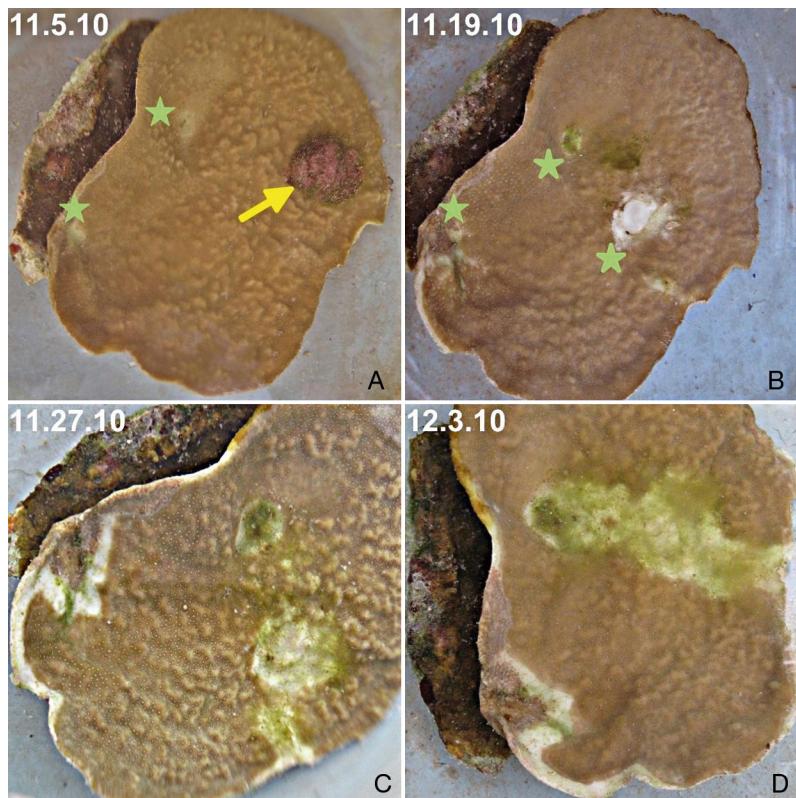


Fig. 2. Time series photographs of progressive tissue loss resembling white syndrome in *Porites rus*, originating from 3 feeding scars (green stars in panel A,B, with the snail indicated by the yellow arrow in A creating a third scar). Tissue continued to slough after the snail was removed on November 5, 2010 (panels B–D). Areas of tissue loss coalesced, forming a large lesion, with additional tissue loss initiating along the left border of the fragment around another feeding scar (panels C and D). Dates are given as mm.dd.yy

Table 1. Fate of feeding scars formed on healthy *Porites rus* plates ($n = 10$ per treatment; several plates contained >1 scar) partitioned by feeding treatment. Diseased-fed: *Coralliophila violacea* snails previously fed diseased coral tissue; healthy-fed: *C. violacea* snails previously fed healthy coral tissue. Census taken 7 d after removal of snails

Metric	Diseased-fed	Healthy-fed
Total feeding scars	13	14
Number of lesions progressing (%)	9 (69)	4 (28)
Number of feeding scars healing (%)	3 (23)	9 (65)
No lesions formed (%)	1 (8)	1 (7)
Lesion progression rate ($\text{cm d}^{-1} \pm \text{SD}$)	0.24 ± 0.3	0.47 ± 0.62
Feeding scar healing rate ($\text{cm d}^{-1} \pm \text{SD}$)	0.27 ± 0.3	0.21 ± 0.16

old lesions (Fig. 4). For *P. rus*, wound repair was seen only in new lesions, whereas necrosis and cleft formation were seen only in old lesions. Mucus and calicodermal cell hypertrophy and inflammation, characterized by prominent infiltrates of melanin-like granular cells, were seen only in *P. cylindrica*

(Figs. 3A–D). Algae (Figs. 3E,F & Fig. 4B) were the dominant organisms seen on microscopy and were present only in old lesions for *P. cylindrica*, whereas algae were seen in both new and old lesions for *P. rus* (Table 2). Endolithic sponges, fungal hyphae, and helminth worms constituted other organisms associated with lesions.

DISCUSSION

Evidence that snails were transmitting a pathogen leading to tissue loss in *Porites* was equivocal, although our results clearly showed that snail feeding was associated with progressive tissue loss in snails previously exposed to acute tissue loss lesions. Histology failed to detect bacterial or viral inclusions associated with either old or new snail-induced lesions, although the use of light microscopy cannot preclude the presence of viruses that were not numerous enough to form inclusions. The lack of concordance between the assumption that tissue loss (white syndrome, WS) is caused by bacteria and absence of histological evidence of the presence of bacteria associated with cell death in WS-affected coral has been pointed out elsewhere (Ainsworth et al. 2007, Work et al. 2012), although it has also been suggested that there may be more than 1 causal agent associated with this suite of disease signs (Work et al. 2012, Bourne et al. 2015). We propose that secondary colonizers, primarily algae, may be responsible for lesion progression after snail removal, but the mechanism by which they might cause progressive tissue loss remains unknown. Recent discussions of the role

of endoliths on calcium carbonate-secreting organisms cite both positive and negative effects on the host (Tribollet 2008). Fungi penetrate and dissolve coral skeleton and attack both endolithic algae and coral polyps (Bentis et al. 2000). *Ostreobium* spp., endolithic algae in many normal corals, can colonize

Table 2. Percent of *Porites cylindrica* and *P. rus* manifesting particular histological changes partitioned by whether *Coralliphila violacea* snail-induced lesions were new (≤ 1 wk) or old (> 1 wk), and host response or organisms associated with the lesions

Parameter	<i>Porites cylindrica</i> (n = 11)				<i>Porites rus</i> (n = 13)				All colonies	
	New	Old	Total	%	New	Old	Total	%	n	%
Host response										
Basophilic membrane	4	2	6	55	2	4	6	46	12	50
Necrosis	2	3	5	45	0	6	6	46	11	46
Hyaline membrane	5	2	7	64	1	3	4	31	11	46
Fragmentation	4	0	4	36	3	2	5	38	9	38
Cleft formation	0	2	2	18	0	4	4	31	6	25
Hypertrophy calicodermis	2	0	2	18	0	0	0	0	2	8
Mucus cell hypertrophy	3	0	3	27	0	0	0	0	3	13
Inflammation	2	1	3	27	0	0	0	0	3	13
Wound repair	0	0	0	0	1	0	1	8	1	4
Atrophy	0	0	0	0	0	1	1	8	1	4
Organisms										
Algae	0	1	1	9	2	0	2	15	3	13
Fungi	0	1	1	9	1	1	2	15	3	13
Sponges	0	1	1	9	0	1	1	8	2	8
Helminths	0	2	2	18	0	0	0	0	2	8

host skeleton following tissue loss, penetrating live coral tissue, which could influence regeneration (Fine et al. 2006). The absence of bacteria associated with cell death in early lesions in *P. cylindrica* and *P. rus* argues against snails acting as vectors of bacterial pathogens, as these would presumably be visible in early lesions if they were transmitted by snails. However, the more common presence of fungi and algae in both early and late *P. rus* lesions is intriguing and suggests more rapid colonization of this species by secondary organisms.

Work et al. (2012) and Bourne et al. (2015) discussed the evidence for multiple causative agents for WS, which may constitute a suite of diseases. Our results suggest that different processes may be at work in the 2 coral host species we examined, with similar gross signs of disease associated with a single corallivore. Predation scars can serve as entry wounds that become invaded after the snail moves. At least 2 other coral diseases, viz. skeletal eroding band (Page & Willis 2008) and brown band disease (Nicolet et al. 2013), can be initiated by damage to the coral surface. Additionally, our evidence indicates that snail feeding facilitates colonization of coral by secondary organisms. Clearly, snails can cause tissue necrosis and, depending on the host's ability to respond, subsequent progressive tissue loss associated with secondary colonizers.

Our feeding experiments revealed that rates of tissue loss and healing were not significantly different, either between or within feeding treatments. Tissue

loss rates were highly variable, and our small replicate size may account for a failure to reveal a possible treatment effect. Healing rate is a process dictated by how rapidly new tissue can be produced to resheet bare skeleton. Thus, this process would not be expected to differ between treatments if disease signs were absent within the lesion. Lozada-Misa et al. (2015) reported *in situ* tissue loss lesion healing rates of 0.15 ± 0.3 (SD) cm d^{-1} in *P. cylindrica* and 0.015 ± 0.002 cm d^{-1} for massive *Porites*. Thus, the rates we observed for *P. rus*, a close relative, are comparable. Furthermore, the artificial environment of the aquaria may influence both tissue loss and healing processes.

Microscopy provided a potential explanation for the behavior of gross lesions induced by *Coralliphila*. Deposition of basophilic membrane within coral tissues has not been seen previously in *Porites* examined histologically in the Pacific (Williams et al. 2011, Sudek et al. 2012, Work et al. 2014, 2016), and this change might serve as a useful histological marker of snail predation in corals. The necrosis seen would fit with the snail's strategy of secreting proteolytic enzymes to aid in consumption and digestion of coral tissues (Ward 1965). However, we saw differences in host response to predation. Like *P. compressa* (Sudek et al. 2012), *P. cylindrica* manifested a prominent inflammatory response comprising hypertrophied calicodermis and infiltrates of melanin-like granule cells. Other congeners, such as massive *Porites*, can mount robust inflammatory responses,

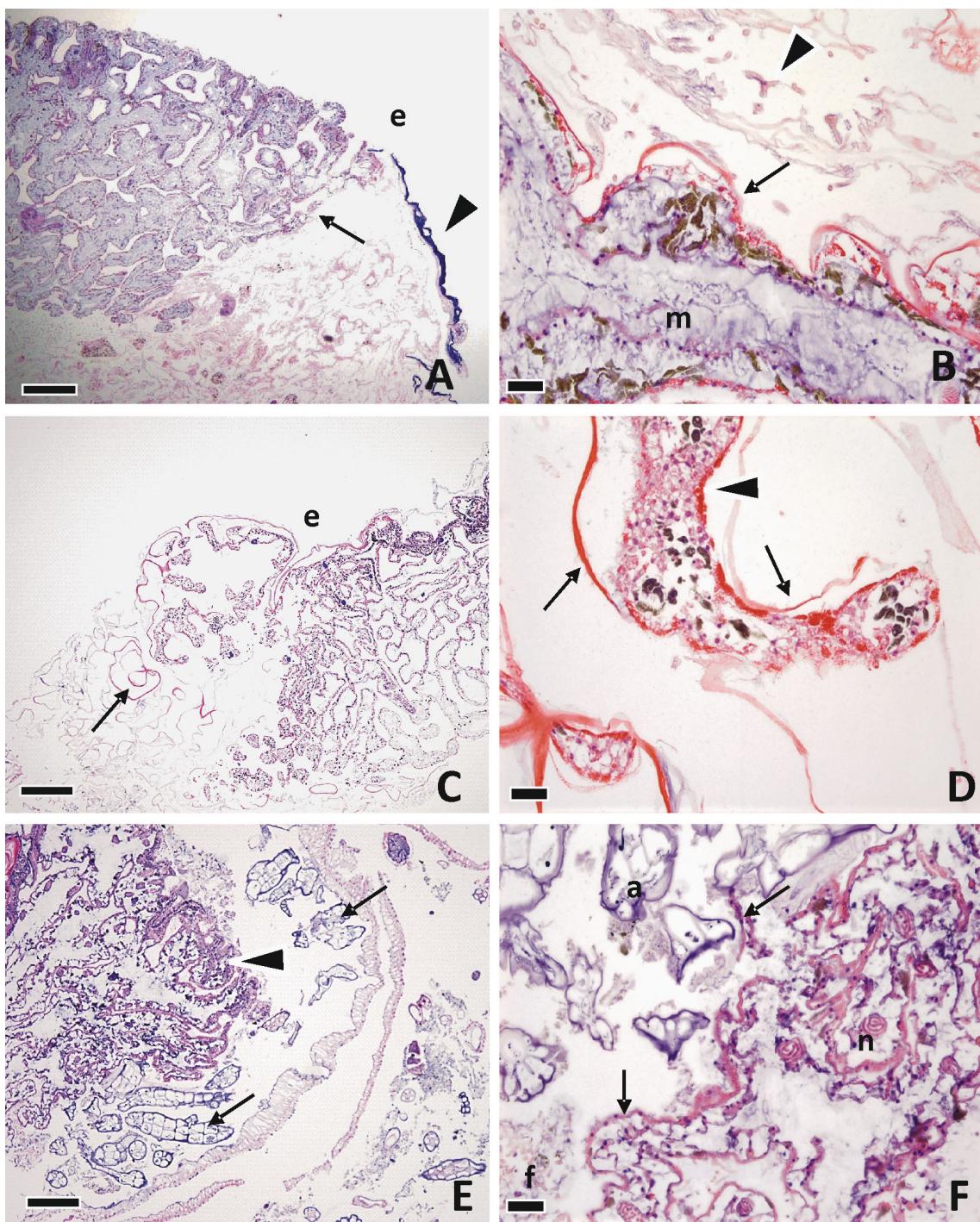


Fig. 3. Hematoxylin and eosin stained tissue sections of new (≤ 10 d) *Coralliophila violacea* snail-induced lesions in (A–D) *Porites cylindrica* and (E,F) *P. rus*. with paired low (A,C,E) and high (B,D,F) magnification photomicrographs; e: epidermis, m: mucus, a: algae, f: fungi, n: nematocyst. (A) Diffuse full thickness necrosis of surface and basal body wall; arrow points to border of intact (upper left) and necrotic (lower right) tissues with deposition of basophilic membrane (arrowhead); scale bar = 500 μ m. (B) Close up of Panel A at arrow. Note copious mucus within basal body wall and deposition of hyaline membranes (arrow) with occasional fungal hyphae within necrotic debris (arrowhead); scale bar = 20 μ m. (C) Necrosis of surface and basal body wall with deposition of hyaline membranes (arrow); scale bar = 500 μ m. (D) Close up of Panel C at border of necrotic area; note basal body wall becoming progressively attenuated with hypertrophied calicodermis (arrowhead) and deposition of hyaline membranes (arrows); scale bar = 20 μ m. (E) Invasion of coral tissues by algae (arrows) associated with necrosis (arrowhead); scale bar = 200 μ m. (F) Close up of Panel E at arrowhead; note algae and fungi apposed to clumps of gastrodermal cells manifesting pyknosis and hypereosinophilia (arrows); scale bar = 20 μ m

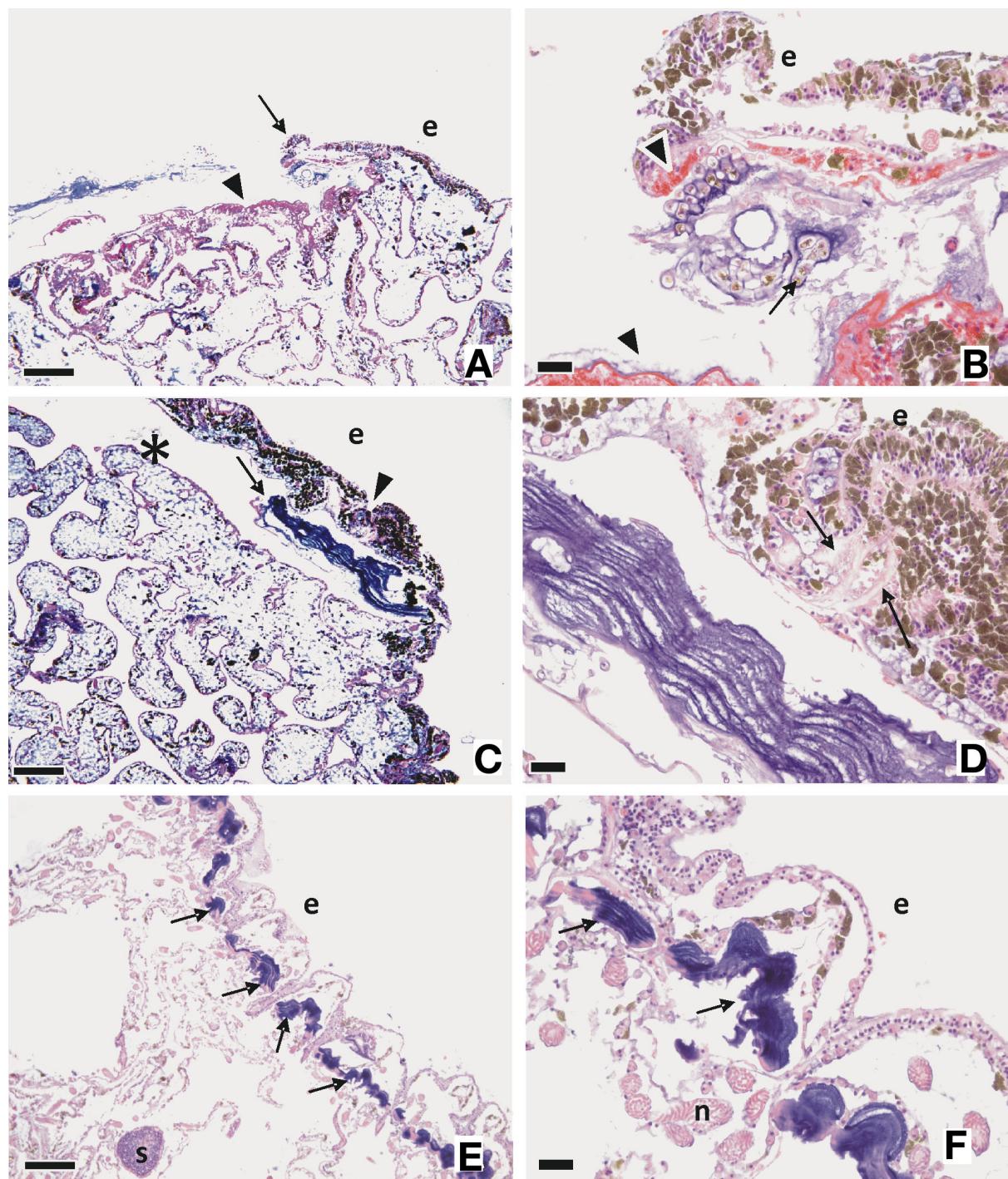


Fig. 4. Hematoxylin and eosin-stained tissue sections of old (>10 d) *Coralliophila violacea* snail-induced lesions in (A–D) *Porites cylindrica* and (E,F) *P. rus* with paired low (A,C,E) and high (B,D,F) magnifications; e: epidermis, n: nematocyst, s: spermatheca. (A) Ablation of surface body wall (arrow) with hypertrophy of calicodermis (arrowhead); scale bar = 500 μ m. (B) Close up of Panel A; note clumps of plant material with cell walls (arrow) surrounded by basophilic wispy material, hypertrophied and granular calicodermis (arrowhead), and epidermis infiltrated with melanin-like granule cells; scale bar = 20 μ m. (C) Cleft formation (asterisk) with deposition of basophilic membrane (arrow) and lifting of surface body wall that is infiltrated with melanin-like granule cells (arrowhead); scale bar = 200 μ m. (D) Close up of Panel C; note basophilic laminar membrane overlaid by surface body wall with portions of gastrodermis separated by mesoglea (arrows) and infiltrates of melanin-like granule cells; scale bar = 20 μ m. (E) Deposition of basophilic-laminated membrane below the surface body wall (arrows); scale bar = 500 μ m. (F) Higher magnification view of Panel E; note that basophilic membranes (arrows) appear to be deposited into gastrodermis; scale bar = 20 μ m

particularly of melanin-like granule-containing cells, similar to those seen in this study (Palmer et al. 2008). Calicodermal hypertrophy would explain skeletal accretion at the edges of snail lesions; evidently, deposition of aragonite by calicodermal cells forms part of the host response repertoire in *P. cylindrica* (Allemand et al. 2011). Given their close association with hypertrophied calicodermis, we suspect that hyaline membranes were acid-rich proteins of the skeletal organic matrix (Mass et al. 2014). In contrast, *P. rus* displayed a minimal inflammatory response. Colonizing organisms such as algae and fungi were present mainly in older lesions for *P. cylindrica*, whereas they were more common in early and late lesions in *P. rus*. This may be linked with the superior inflammatory response of *P. cylindrica* that would presumably dissuade colonization by opportunistic organisms for longer than *P. rus*.

In summary, our study illustrates the power of combining gross observations with microscopy to explain biological phenomena in corals and yield insights into disease processes. In this case, snails caused significant damage to corals by opening portals of entry for secondary colonizers. The degree of damage to the colony may depend, in part, on the robustness of the host inflammatory response. Whilst the snails cannot be considered vectors of infectious agents, it is possible that they could move algae and fungi between corals, and histology of snails associated with corals may shed light on this question.

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