

Short Communication

Cellular reactions to sedimentation and temperature stress in the Caribbean coral *Montastraea cavernosa*

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Abstract

We present evidence of cellular responses to increased sedimentation and temperature in *Montastraea cavernosa* collected off Broward County, Florida. We sampled corals from six different sites approximately, 500–1000 m off shore, 10–15 m depth. Six samples were collected from four sites adjacent to areas of underwater marine dredging (project sites), while the remaining two samples were obtained far away from the influence of the marine dredging (control sites). SSTs around collection time ranged 0.6–0.9 °C over the 40-year monthly mean. All specimens collected at project sites exhibited histopathological evidence of mild to moderate sedimentation stress including changes in size and number of mucocytes in epidermis and gastrodermis, attenuation of the epidermal and gastrodermal tissues, presence of cellular debris, and changes in number of zooxanthellae. These findings corroborate results of laboratory-based, sand-application experiments. In addition to the above-noted changes, one specimen exhibited multiple lesions consisting of unusual gastrodermal detachment with infiltration of amoebocytes into the adjacent mesoglea. Tissues surrounding detachment injuries exhibited marked to severe cellular changes. Accumulations of amoebocytes at lesion sites are seldom observed in wild corals. This response may be part of an organized reaction to injury and infection, as has been documented in sea anemones and gorgonians; however, further research is needed on the nature and role(s) of the scleractinian amoebocytes.

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Assessment of tissue and cellular reactions to sublethal environmental impacts has received limited attention in scleractinian corals. Histopathology provides a visual means by which to evaluate the coral's susceptibilities to physical injuries, contaminants, and toxicants, as well as the mechanisms of damage and repair in target cells and tissues (Peters et al., 2005). For example, studies by Peters and Pilon (1985), Riegl and Bloomer (1995), and Vargas-Ángel et al. (2006) provide clear evidence of epithelial attenuation, and atrophy and damage to the coral's epidermal muco-

cytes and cilia associated with experimental sedimentation stress. These studies also indicate that prolonged and increased exposure to sedimentation stress result in diminished mucous secretion, overall attenuation of epidermal and gastrodermal cell layers, accumulation of cellular debris, and eventually, loss of tissue integrity and necrosis. Correspondingly, in a study of prolonged exposure to high temperature stress on Panamanian reef corals, Glynn and D'Croz (1990) reported decreased zooxanthellae densities and necrotic zooxanthellae, in addition to epidermal and gastrodermal erosion, retractor muscle atrophy and altered mesogleal pleat structure, with loss of tissue architecture and necrosis.

The above studies are prominent in that they have established a baseline against which to compare the normal ver-

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sus the stressed condition in scleractinian corals, and provide a framework to survey for coral cellular responses to environmental stress in field conditions. The objectives of the present study were twofold: (1) to use histopathological techniques to survey for coral cellular reactions to sedimentation and temperature stress under field conditions, and (2) to compare these field-obtained findings with results obtained from laboratory-based manipulations (e.g., Peters and Pilson, 1985; Vargas-Ángel et al., 2006; Glynn and D’Croz, 1990).

In the present study, tissue samples of the Caribbean coral *Montastraea cavernosa* were collected off Broward County, Florida, on August 8–9, 2005, as part of a larger project aimed at assessing potential gross and histopathological effects of increased turbidity and sedimentation on corals in situ during offshore dredging operations. Treatment and control tissue samples came from colony edges or entire small colonies (3–5 cm diameter) dwelling at 11–15 m depth. Using hammer and chisel a total of eight coral samples were collected; six from four reefs (hereafter project sites: BA1SM2, BA1SM3, BA2SM2, and BA2SM4, respectively) adjacent to each of two distinct areas exposed to underwater dredging activities (BA1 and BA2, respectively) and the remaining two, from distant control reef sites (FTL2 and FTL5, respectively) located approx. 2–10 km upstream from the dredging activity. Within minutes after collection all tissues were fixed in Z-Fix (zinc-formalin solution, Anatech Ltd., diluted 1 part to 4 parts seawater) for 18–24 h, and processed using standard histological techniques to produce paraffin blocks. Two representative 4–5 µm-thick sections per specimen were obtained and one each were stained with Harris hematoxylin and eosin (H&E) and modified Movat’s Pentachrome procedure (developed by P.P. Yevich and C.A. Barszcz, see Peters et al., 2005). For reference, the histopathological condition of all experimental corals (from project and control sites) was compared with other samples of *M. cavernosa* (eight specimens) collected from the same general vicinity and depth one month prior to the initiation of the dredging operations (April 4–5, 2005).

For purposes of this study, sediment accumulation rates were derived from four traps for the period between June 14 and August 10, 2005. The estimated sediment accumulation rate for the dredge areas BA1 and BA2 amounted to 58.4 and 40.9 mg/cm²/day, respectively. Sediment accumulation rates during the same period at the control sites FTL2 and FTL5 were 24.7 and 29.2 mg/cm²/day, respectively. Accumulated sediments were roughly composed as follows: >500 µm: 4.3 ± 4.2%; 250–500 µm: 8.7 ± 7.0%; 125–250 µm: 30.4 ± 21.3%; 63–125 µm: 16.6 ± 6.7%; and <63 µm: 40.1% ± 25.4% (means ± SD% dry weight) (Gilliam et al., in press). Additionally, between July and August, 2005, mean sea surface temperatures (SSTs) off southeast Florida reached 0.6–0.9 °C (29.54 °C and 30.22 °C, respectively) warmer than the 40-year long-term mean (28.98 °C and 29.25 °C; ICOADS 1960–2000, 2005; 1° grid centered at 26°N; 80°W; <http://dss.ucar.edu/datasets/ds277.0>). These prolonged, elevated temperatures resulted in focal and diffuse pallor in some corals at shallow depths throughout the region (3–10 m; B. Vargas-Ángel, pers. observ, Gilliam, 2006).

Grossly, at the time of collection, project site specimens ($n = 6$) exhibited mild to moderate signs of stress including polyp swelling, tissue thinning, discoloration, slight pallor, and tight polyp retraction, as well as distended and discolored oral disks. Sediment accumulation between polyps was also observed in some cases (Table 1). These changes are consistent with those observed in laboratory, sedimentation stress studies conducted on scleractinian corals by Peters and Pilson (1985) and Vargas-Ángel et al. (2006). Gross changes in control site specimens ($n = 2$) were minor, consisting mainly of slight polyp retraction. No injuries or breaks in the tissues covering the skeletons were observed in any of the study corals.

Histologically, all samples collected from project sites exhibited evidence of sedimentation stress, ranging from mild to moderate and including: (1) multifocal to diffuse changes in number and size of mucocytes in the surface body wall epidermis and gastrodermis of the oral disk, tentacles, and coenenchyme (“common tissue” between the polyps); also the epidermal and gastrodermal mucocytes

Table 1

Summary of gross changes observed at time of collection in coral individuals from project and control sites collected, Aug 8–9, 2005, and expressed as presence/absence of each condition

Sample ID	Collection site	Gross changes							
		TSSWL	TSDEF	BLESL	OTDIS	PORET	ORASL	MUCSH	SEDAC
MC303	BA1SM2	x	x						
MC304	BA1SM3	x			x	x	x		
MC305	BA1SM3				x			x	x
MC308	BA2SM2				x		x		
MC309	BA2MS2			x		x			
MC310	BA2SM4				x		x		
MC323	FTL2					x			
MC326	FTL5					x			

TSSWL, tissue swelling; TSDEF, tissue deflation; BLESL, slight/focal pallor/bleaching; OTDIS, other type of discoloration; PORET, polyp retraction; ORASL, oral disc morphological change (including distention, retraction, or protrusion); MUCSH, mucous sheets; SEDAC, sediment accumulation between polyps.

incorporated variable amounts of the pentachrome dyes, indicating a change in the biochemical composition of the mucus; (2) attenuation of the epidermis and gastrodermis, particularly in the coenenchyme and middle polyp region, as well as the mesenteries; (3) changes in number of zooxanthellae, mainly in the coenenchyme, oral disk, and mesenteries; (4) focal to multi-focal appearance of cellular debris, particularly in the middle and lower polyp gastrodermis, as well as the calicodermis (calicoblastic epithelium) of the basal body wall; (5) slight to moderate swelling of the calicodermis, possibly indicating compromised calcifying capability. In addition, some specimens also exhibited focal to multifocal reductions in abundance of spirocysts and an increased abundance of granular cells in the tentacles (Table 2). In contrast, some tissue and cellular attributes remained unaltered in most specimens, indicative of the moderate and reversible nature of the above-noted pathologies, including: (1) general cell architecture and tissue integrity; no widespread necroses; (2) mesogleal thickness and integrity, particularly in tentacles and oral regions, as well as mesenteries; mesogleal pleat structure also appeared unchanged; (3) retractor muscle structure, thickness, and staining properties; (4) architecture and integrity of the mesenteries and basal body wall gastrodermis; (5) in reproductive colonies (MC305, 304, and 318) spermaries and oocytes appeared normal and with no evidence of degradation or resorption. These observations are comparable to the pathologies documented following two weeks of controlled sanding in laboratory manipulations using *Astrangia danae* and *M. cavernosa* (Peters and Pilson, 1985; Vargas-Ángel et al., 2006; see also Peters, 1984).

In addition to the above-noted changes, one specimen (MC304) exhibited unusual epithelial detachment injuries, characterized by accumulations of amoebocytes in the mesoglea adjacent to the lesion sites (Fig. 1A and B). A lacuna or space surrounding the cells was a common feature. Also, acidophilic granules were observable in the cytoplasm of some amoebocytes; these are proposed to have lysogenic or peroxidase functions (see Olano and Bigger, 2000). In specimen MC304, detachment injuries were multi-

focal and situated in the coenenchyme, often in close proximity to the body wall and its attachment to the carbonate skeleton. Tissues immediately adjacent to these injuries revealed histological evidence of localized, marked to severe stress, including: (1) hypertrophy and distortion of mucocytes; (2) swelling of the calicodermis; (3) clumpy, dark-staining (or sparse and pale-staining) mucous secretions; (4) shrunken zooxanthellae; (5) hydropic degeneration, characterized by vacuolation and cloudy swelling in cells of the gastrodermis and calicodermis; and (6) cellular debris in the mesoglea. Mucocytes in the coenenchymal gastrodermis incorporated little of the pentachrome dyes, indicating reduced or lost mucous secreting function (Fig. 1A and B). The mesoglea stained unevenly, with paler patches, indicating degenerative changes. Also, myonemes stained gray rather than the usual red with the pentachrome dyes, and the occurrence of pale-staining nuclei, particularly in the calicodermis and gastrodermis, may be indicative of karyolysis, a necrotic change (see Glynn and D'Croz, 1990). Concordant with these changes, specimen MC304 also revealed focal sloughing and erosion of the gastrodermis of the coenenchyme. Sloughed tissue was necrotic, hypereosinophilic, and contained degenerate zooxanthellae. Gastrodermal erosion was distal to any of the detachment injuries detected. Grossly, however, specimen MS304 did not differ substantially from any of the other project site specimens.

Histologically, control corals also exhibited changes compared to normal corals, and consisting mainly of an augmentation in the number and size of mucocytes in the epidermis and gastrodermis, as well as an increase in mucous secretions indicated by abundant, green-staining material in the mucocytes (pentachrome procedure; Fig. 1C). One of the control specimens exhibited some debris accumulation particularly in cells of the gastrodermis (Table 2). Overall, these changes were mild and we interpret them as evidence of exposure to moderately high and prolonged sea surface temperatures. Glynn and D'Croz (1990) indicated that exposure to increased and prolonged temperature affected the coral's mucous secreting appara-

Table 2
Summary of histopathological changes in coral individuals from project and control sites, collected Aug 8–9, 2005, and expressed as presence/absence of each condition

Sample ID	Collection site	Histological changes											
		MCHYP	MCHPO	MCRED	EPATT	GDDEB	ZORED	ZODEG	GDDET	GDSLO	MSAAM	TESPG	
MC303	BA1SM2	x		x	x	x							x
MC304	BA1SM3	x	x	x	x	x	x	x	x	x	x	x	x
MC305	BA1SM3		x	x	x			x					x
MC308	BA2SM2		x	x	x			x					x
MC309	BA2MS2	x						x					
MC310	BA2SM4	x	x					x					
MC323	FTL2	x					x						
MC326	FTL5	x											

MCHYP, hypertrophy of mucocytes in gastrodermis; MCHPO, hypotrophy of mucocytes in epidermis; MCRED, reduction in mucocyte number in epidermis; EPATT, attenuation of the epidermis; GDDEB, accumulation of debris in the gastrodermis; ZORED, zooxanthellae reduction; ZODEG, degradation/degeneration of zooxanthellae; GDDET, tissue detachment injuries; GDSLO, focal sloughing and necrosis of gastrodermis; MSAAM, multifocal accumulations of amoebocytes in mesoglea; TESPG, reduction of spirocysts and increased of granular cells in tentacles.

tus. In addition, with reference to the characteristics noted above, the “normal” condition is characterized by: (1) absence of debris and/or swelling in the gastrodermis or calicoderms; (2) absence of swelling of the mucocytes; (3) uniform, smooth-staining mesoglea and mucous secretions. Also, the myonemes of the epitheliomuscular cells lining the mesoglea are well-defined and stain bright red (Fig. 1D).

In general, the histopathological abnormalities observed in this study, overall hypertrophy or attenuation of coral tissues, changes in the amount and biochemical composition of mucous secretions, and changes in zooxanthellae densities, were similar to those reported both in controlled, sand-application and heat-exposure laboratory experiments, as well as in observations of sediment and temperature-stressed corals under field conditions (Lasker et al., 1984; Peters, 1984; Glynn et al., 1985; Peters and Pilson, 1985; Glynn and D’Croze, 1990; Vargas-Ángel et al., 2006). In contrast, the detachment injuries and amoebocyte accumulations found in MS304 have not been noted in histopathological examinations of scleractinians under either experimental or natural conditions, or affected by different

types of lesions (trauma or bleaching, black band disease, white band disease, and dark spots disease), except in a study involving wound infliction and repair (see Mullen et al., 2004; Kramarsky-Winter, 2004; authors, pers. observ.), perhaps because the amoebocytes are so small in these animals (Fig. 1D).

The tissue detachments documented in this study probably occurred because of breakdown of the basement membrane and myonemes at the points of contact between the gastrodermis and mesoglea. The absence of gastrodermal necrosis at the detachment injury sites suggests that the detachment may have been the primary event. Atrophy and necrosis of muscle fibers, in combination with the loss of mesogleal integrity have been previously observed in temperature-stressed corals (Glynn and D’Croze, 1990). It is not clear, however, if the localized injuries described in this study resulted due to the direct effects of increased sedimentation and temperature or indirectly, due to a nutritional depletion following excess mucus production for sediment removal, and zooxanthellae loss (bleaching). There could even be a host/pathogen component, whereby the deterioration of environmental conditions could decrease the

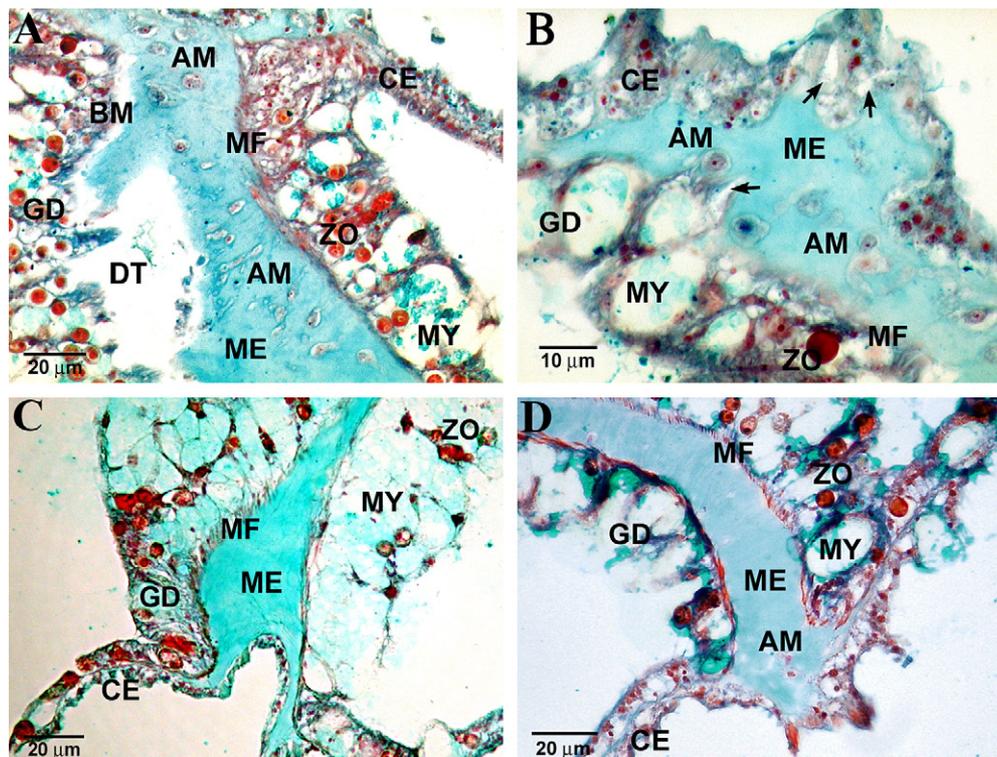


Fig. 1. Photomicrographs of histological preparations of tissues of *Montastraea cavernosa*, illustrating the pathologies described in the text. (A) Accumulation of amoebocytes at a lesion site involving the detachment of the gastrodermis from the mesoglea. A lacuna or space surrounding the amoebocytes was a common feature. Also, the acidophilic granules observable in the cytoplasm of some amoebocytes may have lysogenic or peroxidase functions (specimen MC304; Aug 8, 2005). (B) Accumulation of amoebocytes at an earlier stage of tissue detachment injury. Arrows indicate sites of early detachment of the gastrodermis and calicoderms from the mesoglea. Also note the clumpy, dark-staining (or sparse and pale-staining) mucous secretions, swelling of mucocytes and calicoderms, and the accumulation of debris in the mesoglea and calicoderms (specimen MC304; Aug 8, 2005). (C) Control coral, exhibiting swollen mucocytes and increased, pale-staining mucous secretions, indicative of mild stress (specimen MC326; Aug 9, 2005). (D) Normal tissue collected in April 5, 2005 at project site, prior to the onset of dredging and during normal thermal conditions. Note the uniform, smooth-staining mesoglea and mucous secretions, the absence of debris or swelling in the gastrodermis and calicoderms, and myonemes lining the mesoglea are well-defined, staining bright red. AM, amoebocyte; BM, basement membrane; CE, calicoderms; DT, tissue detachment; GD, gastrodermis; ME, mesoglea; MF, muscle fibers or myonemes; MY, mucocytes; ZO, zooxanthellae.

coral's ability to fight infection (see Harvell et al., 2002, 2004; Ben-Haim et al., 2003). Bacteria and fungi were observed in histological preparations of temperature-stressed corals (Lasker et al., 1984; Glynn et al., 1985). In addition, proliferation of endobiotic bacteria has been also noted in electron micrographs of sediment-stressed *M. cavernosa* (Blackwelder, Vargas-Ángel, and Renegar; pers. observ.).

While the tissue and cellular reactions to increased sedimentation and temperature stress have been relatively well documented (Lasker et al., 1984; Glynn et al., 1985; Glynn and D'Croz, 1990; Peters, 1984; Peters and Pilson, 1985; Riegl and Bloomer, 1995), the differing responses among individual colonies observed in this study may reflect differing inter-colony tolerance limits. Corals in Broward County are exposed to naturally occurring elevated sediment accumulation rates. Thus, colonies with lower stress tolerance thresholds, previously injured, or diseased, may be less able to resist the effects of increased sedimentation and exhibit increased tissue alterations. The amoebocyte accumulations at injury areas observed in this study, are seemingly not part of the typical coral stress response signature, but resemble the induced, wound-healing reactions documented for the gorgonians *Plexaurella fusifera* and *Swiftia exserta*, the sea anemone *Anthopleura elegantissima*, and the scleractinian coral *Fungia granulosa* (Patterson and Landolt, 1979; Meszaros and Bigger, 1999; Olano and Bigger, 2000; Kramarsky-Winter, 2004). Mullen et al. (2004) proposed that the epidermal mucociliary system of sedentary scleractinians provided better defense against constant exposure to sediment particles and bacteria, whereas vertically erect gorgonians and highly contractile sea anemones that more easily shed sediment depended on their larger populations of amoebocytes to participate in wound repairs and microbial infections, phagocytosing debris and killing bacteria. Although it is plausible that this response in a scleractinian is part of an organized reaction to injury and infection, further research is needed in this matter. Thus, we encourage investigators to look for this amoebocyte accumulation response during histopathological examinations of scleractinian corals, particularly in those species having a thicker or fleshy mesoglea. These type of studies assist in our understanding of the immune system of scleractinian corals, as well as the conditions under which such responses may be mounted. Furthermore, this study confirms the value of histopathology in assessing the condition of corals that grossly appear only mildly stressed.

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