Lesion healing on massive *Porites* spp. corals

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ABSTRACT: Small tissue lesions 15 to 1310 mm^2 were inflicted on massive *Porites* spp. corals living in a shallow moat (<3 m) in Okinawa, Japan. All but 1 lesion, affected by extreme mid-winter low tides, healed completely within 19 mo. Regeneration rates decreased exponentially over time and decreased in accordance with the lesion's surface area. The best predictive models were those using the derivatives of the exponential equations. A high perimeter to surface area ratio was a consequence of, and appeared to facilitate, regeneration. There was no significant difference in the rate of lesion regeneration between *Porites lutea* and *Porites lobata*.

KEY WORDS: Lesions · Massive *Porites* · Healing rates

INTRODUCTION

Massive *Porites* corals are prolific throughout the world's tropical oceans (Veron 1995); they are particularly common on reefs nearshore and are arguably the most important reef-building corals on the inner Great Barrier Reef (Australia) (Hopley 1982, Davies 1983). *Porites* colonies have long been recognised for their usefulness as temporal analogues (Buddemeier & Kinzie 1976) and have become the focus of palaeo-climatic studies. They have been used as proxies for terrestrial runoff (Isdale 1984, Boto & Isdale 1985), predictors of sea-surface temperature (McCullock et al. 1994), indicators of past spawning events (Gagan et al. 1994) and recorders of sea level (Woodroffe & McLean 1990).

In the Ryukyu Islands, the island chain in southern Japan, massive *Porites* spp. colonies are common in shallow moats <3 m deep. Disturbances to these *Porites* spp. colonies are frequent and range from (1) small lesions caused by gastropod and fish predation to (2) patchy die-off during low spring tides in winter to (3) sand-scouring around the base of the colonies during the typhoon season to (4) partial or whole colony

mortality during episodes of *Acanthaster planci* predation (author's pers. obs.).

Studies on the regeneration of coral lesions extend back several decades (Stephenson & Stephenson 1933, Kawaguti 1936, Fishelson 1973, Loya 1976). The capacity to regenerate appears to be species specific and dependent on lesion size, sea-surface temperature, water depth and whether the colony has been recently bleached (Lester & Bak 1985, Meesters & Bak 1993, Meesters et al. 1994), but appears to be independent of calcification rate (Bak & Steward-van Es 1980). Regeneration often follows negative exponential curves, with the rate of regeneration decreasing after some time, although many studies on massive scleractinian corals show that healing is often partial and incomplete (Stephenson & Stephenson 1933, Meesters et al. 1994).

Regeneration of *Porites* spp. was first reported for branching colonies by Kawaguti (1937). He indicated healing took place by the proliferation of boundary polyps. Done (1987) reported on regeneration of massive *Porites* following *Acanthaster planci* predation and suggested that living portions of injured colonies rarely encrust onto adjacent surfaces but tend to grow primarily in the direction of the original colony's radii, indicating that upward growth and subsequent fusion is most likely after infliction of wounds ≥30 000 mm^2 (which is an approximate size estimate of an extrud-

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able stomach of a sexually mature *A. planci* starfish, using Birkeland 1989).

The survival of subsurface tissue in Hawaiian *Porites compressa* colonies induces an unusual regeneration response, where colonies appear dead but regenerate through subsurface projections (Jokiel et al. 1993). In contrast, Atlantic *Porites astreoides* colonies have poor regenerative capacities, especially when lesions are >500 mm² (Bak & Steward-van Es 1980), but are quite capable of regenerating smaller lesions (Meesters & Bak 1993). Empirical measurements are not available on individual massive *Porites* spp. from the Indo-Pacific region, although rapid regeneration of massive *Porites lutea*, after suffering tissue damage due to sedimentation effects, can be inferred from community data in Brown et al.'s second table (1990). The present study focuses on the most common massive *Porites* spp. in the Indo-Pacific, *P. lutea* and *P. lobata*, and shows that 97% of tissue lesions <1310 mm² recover completely within 19 mo and that regeneration rates exponentially decrease with an increase in lesion size.

METHODS

Field measurements. The study was undertaken in a well-flushed, clear water, inner reef moat, no more than 3 m deep at low spring tide, on the eastern side of Ikei Island, Okinawa, Japan (lat. 26° 23' N, long. 128° 00' E). The experiment commenced on 22 September 1994 and ended 19 mo later. Treatments involved making 3 differently sized tissue lesions to each of 10 massive *Porites* spp. colonies that ranged in size from 70 to 100 cm in diameter. Small samples were extracted from each colony, well away from the experimental lesions, for species identification.

Lesions were inflicted in colonies between the depths of 10 and 50 cm below low water datum, at similar angles ranging from 15 to 40° to the horizontal plane. To create the lesions, plastic plates with circular stencil holes, 100, 200 and 400 mm² in area, were held over each colony while a jet of high pressure air was blasted over the plates. Lesions were at least 50 cm apart. Because the 30 subsequent lesions varied in size and shape they were categorized into 3 size classes (A 15–150, B 151–350, C 351–1310 mm²). For lesion identification, stainless steel nails were set either side of each lesion along its central axis. The distance between the nail markers varied consistently in accordance with the lesion size class. Nails were set 10 cm apart for A size lesions, 12 cm apart for B size lesions and 15 cm apart for C size lesions.

Lesion recovery, together with algal density and abundance of sediment on each lesion, were monitored biweekly for a month, then intermittently until complete healing 19 mo later. Monitoring involved measuring the lesion's longest axis and the length of its perpendicular axis to the nearest millimetre, sketching each lesion (to assess changes in lesion morphology) and measuring at least 4 other axes and their orientation depending on the lesion's shape. Care was taken not to disturb sediment or algae on the lesions. These measurements were later transcribed to mm² grid paper from which the lesion's surface area and perimeter length were calculated.

Given the usefulness of massive *Porites* as sclerochronological indicators, it was hypothesized that they may also record partial mortality and healing events in their skeletons. To test this hypothesis, a core, 55 mm in diameter and 50 mm long, was extracted from 3 different *Porites* colonies using a pneumatic drill. The cores were taken precisely over the areas where the C size lesions had healed. Cores were subsequently cut longitudinally and examined under magnification for scars.

Data analysis. Two data sets were derived from the field measurements; one recorded a change in the lesion's surface area over time, the other recorded the lesion's perimeter to surface area ratio. A general model without an asymptote was used to estimate the regeneration abilities of the corals. The changes in surface areas over time were analysed using the exponential equation

$$\text{Lesion surface area } y = be^{-at} \tag{1}$$

where *b* is the intercept, e is the exponential, *a* is the slope of the curve, and *t* the time in days, and the equivalent equation

$$\ln y = \ln b - at \tag{2}$$

where the dependent variable is transformed to a natural logarithm. Since there is no natural logarithm for a dependent variable of 0, that is, where a lesion is completely healed, +1 was added to the dependent variable for calculations. It follows that the time constant $1/a$ is the length of time it takes for the original quantity $\ln b$ to be reduced to $1/e$ of its original value. Least-squared regressions, fitting data to Eq. (2), and slope comparisons (via analysis of covariance after testing the slopes for assumptions of parallelism, Sokal & Rohlf 1995) were undertaken initially on data for *Porites lutea* and *P. lobata*. There were no significant differences in the slopes (Table 1); therefore, the surface area estimates were amalgamated for each size class at each observation time and used as replicates in the calculations. To calculate the rate of change in healing over time, Eq. (1) was differentiated to get

$$dy/dx = -a \times be^{-at} = -ay \tag{3}$$

To test the hypothesis that a lesion's perimeter to surface area ratio had no significant association with heal-

Table 1. *Porites lutea* and *P. lobata*. Regression analyses and slope comparisons for lesion healing on massive corals. Data were fitted to the equation $\ln y = \ln b - at$, where $\ln y$ is natural logarithm transformation of the lesion size + 1, b is the intercept, a is the slope of the line, and t the time in days. n is the number of data points, R^2_{adj} is the percentage variance accounted for, p_{reg} is the probability of regression, and p_{comp} is the probability of no difference between slopes. ••• p < 0.001, ns: not significant

Species	Intercept (SE)	Slope (SE)	n	R^2_{adj}	p_{reg}	p_{comp}
Lesions 15–150 mm^2						
P. lutea	3.07 (0.20)	−0.027 (0.003)	20	0.798	•••	ns
P. lobata	3.54 (0.41)	−0.028 (0.007)	18	0.494	•••	
Lesions 151–300 mm^2						
P. lutea	4.96 (0.38)	−0.019 (0.003)	24	0.601	•••	ns
P. lobata	4.83 (0.35)	−0.018 (0.003)	23	0.604	•••	
Lesions 301–1310 mm^2						
P. lutea	6.32 (0.32)	−0.013 (0.002)	22	0.676	•••	ns
P. lobata	5.92 (0.36)	−0.014 (0.002)	19	0.611	•••	

ing rate, the ratios and times until complete healing were analysed for concordance via Kendall's τ statistic (Sokal & Rohlf 1995).

RESULTS

All lesions were covered with approximately 30% filamentous algae after 2 wk and with at least 60% algae and conspicuous deposits of sediment after 1 mo. Regeneration commenced with an initial 2 to 3 mm upward growth of the healing edge followed by lateral encroachment of soft tissue and skeleton toward the lesion's centre within the first month. The healing edge was a conspicuous pink colour in the first month, then became pale.

Macroalgae (*Padina* sp.) were common on the lesions in winter (December 1994)—4 mo after the lesions were inflicted. No macroalgae were present after February 1995. Lesion recovery was not deterred by filamentous or macroalgae. No evidence of grazing was seen on the lesions throughout the study. A pink fleck consistently appeared on the healing edge as it closed. The fleck turned pale 1 mo prior to complete healing. The lowest tides of the year were co-incident with the coldest yearly air temperatures (in December and January 1994/1995 the early morning low spring tides extended to −22 and −23 cm respectively, while the air temperature averaged 6°C). It was during this period that 3 lesions expanded (2 of them were at the most shallow depths, i.e. at −11 cm and −13 cm below datum; mean lesion depth below datum was −29.8 cm, with a standard deviation of 10.4; Table 2).

Function equations for *Porites lutea* and *P. lobata* showed slight differences between the intercepts b, because of initial differences in lesion size, and slight, but non significant, differences in the slopes a (Table 1). The healing functions for all 3 lesion size classes are given in Fig. 1. Lesions decreased exponentially in size through time. Using Eq. (3), the derivative of the negative exponential function, the healing rate for a lesion y between 15 and 150 mm^2 is $-0.029y$ mm^2 d^{-1}; for a lesion y between 151 and 350 mm^2 the healing rate is $-0.017y$ mm^2 d^{-1}; for a lesion y between 351 and 1310 mm^2 the healing rate is $-0.012y$ mm^2 d^{-1}. These functions show that the rate of healing decreases as the size of the lesion increases (Fig. 1).

Lesions progressively healed from variable shapes to ovals and circles, consequently inducing a higher perimeter to surface area ratio. Indeed, lesions with a high perimeter to surface area ratio showed significant negative correlations with healing time (for lesions 15 to 150 mm^2 Kendall's τ = −0.52, p < 0.001; for lesions between 151 and 350 mm^2 Kendall's τ = −0.48, p < 0.001; for lesions between 351 and 1310 mm^2 Kendall's τ = −0.62, p < 0.001; Table 2). In other words, lesions with a high perimeter to surface area ratio healed faster than those with a low ratio. Rarely (6% of the time) did sequential ratios decrease over time (Table 2). The edges of some narrow, elongate lesions bridged at their narrowest point to form 2 lesions. This increased the perimeter to surface area ratio, effectively facilitating healing.

All 3 cores examined showed scars where the lesions had been. Lesions healed over accumulated debris and sediment (Fig. 2a, b). Spaces between the trabeculae and synapticulae were conspicuously large directly above the damaged surface, giving a porous appearance (Fig. 2c).

DISCUSSION

Massive *Porites* spp. generally grow laterally by extratentacular budding of corallites that are no larger than 1.5 mm, and grow vertically by periodic and
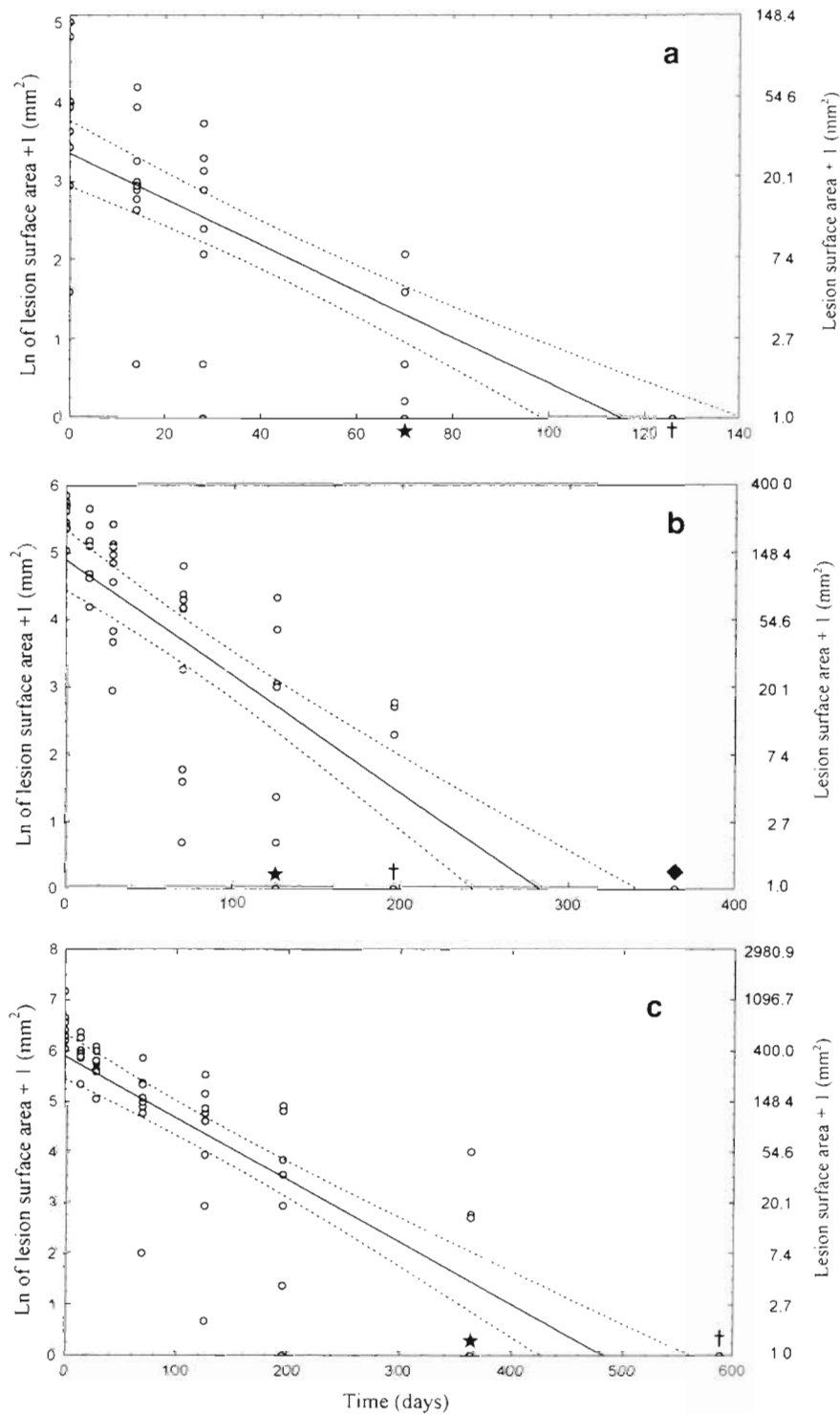
Fig. 1. Regeneration of lesions on massive *Porites* spp. using the exponential equation $y = be^{-at}$ and its equivalent $\ln y = \ln b - at$; fitted regressions and 95% confidence limits (dashed lines) are drawn on each graph. (a) (Size A) Lesion size $y = 3.36 - 0.029 \times$ days (SE for b is 0.21, SE for a is 0.003, $R^2_{adj} = 0.641$, $p < 0.001$). Note overlap of 2 (★) and 6 (†) data points at 0 on the left y-axis. (b) (Size B) Lesion size $y = 4.89 - 0.017 \times$ days (SE for b is 0.23, SE for a is 0.002, $R^2_{adj} = 0.625$, $p < 0.001$). Note instances of overlap of 3 data points each (★, †, ◆) at 0 on the left y-axis. (c) (Size C) Lesion size $y = 5.91 - 0.012 \times$ days (SE for b is 0.22, SE for a is 0.001, $R^2_{adj} = 0.690$, $p < 0.001$). Note overlap of 4 (★) and 3 (†) data points at 0 on the left y-axis. Lesion A from Colony 4 and lesion C from Colony 9 were removed from the model because of their considerable expansion in winter; these lesions were at the most shallow depths, i.e. at −13 cm and −11 cm low water datum, respectively (Table 2). The time constants $1/a$ for (a), (b), and (c) are 34, 59 and 83 d, respectively

Table 2. *Porites lobata*, *P. lutea* and *P. solida*. Ratio of lesion perimeter to surface area on massive corals, where H indicates completely healed, and R indicates the lesion was re-classified into a smaller size class at the first observation. Depth of lesion below low water datum (LWD, cm) is given. nc: data no longer collected

Date	Colony 1 *P. lobata*	Colony 2 *P. lobata*	Colony 3 *P. solida*	Colony 4 *P. lutea*	Colony 5 *P. lobata*	Colony 6 *P. lutea*	Colony 7 *P. lutea*	Colony 8 *P. lutea*	Colony 9 *P. lutea*	Colony 10 *P. lobata*
A size lesions (15 to 150 mm^2)										
Depth below LWD	−20	−34	−29	−13	−25	−37	−47	−28	−10	−46
Sep 1994	0.36	2.25	0.54	0.48	0.34	0.73	0.65	0.65	1.33	0.59
Oct 1994	0.53	3.14	0.84	0.65	0.58	1.08	0.95	1.24	1.26	1.08
Nov 1994	0.81	H	0.95	0.85	0.71	1.5	1.4	3.14	1.50	1.12
Dec 1994	1.5		H	1.27	2.07	3.14	2.5	4.00	H	2.63
Feb 1995	H			0.76[a]	H	H	H	H		H
Apr 1995				1.23						
Sep 1995				H						
B size lesions (151 to 300 mm^2)										
Depth below LWD	−25	−45	−32	−18	−30	−35	−40	−25		−42
Sep 1994	0.33	0.31	0.28	0.25	0.27	0.23	0.21	0.47	R	0.28
Oct 1994	0.37	0.45[b]	0.35	0.33	0.29	0.31	0.22	0.72		0.45
Nov 1994	0.33	1.32	0.38	0.32	0.30	0.40	0.24	1.28		0.75
Dec 1994	0.44	2.23[b]	0.51	0.36	0.36	0.76	0.52	3.14		3.38
Feb 1995	1.25	H	0.92	0.48	0.46	3.00	3.14	H		H
Apr 1995	2.11		1.07	1.00	H	H	H			
Sep 1995	H		H	H						
C size lesions (301 to 1310 mm^2)										
Depth below LWD	−34	−27	−26	−30	−32	−38	−32		−11	−48
Sep 1994	0.22	0.18	0.19	0.17	0.14	0.16	0.20	R	0.25	0.17
Oct 1994	0.33	0.22	0.29	0.19	0.23	0.17	0.23		0.32	0.23
Nov 1994	0.39	0.25	0.25	0.19	0.27	0.23	0.24		0.27[a]	0.24
Dec 1994	1.69[b]	0.28	0.37	0.21	0.90	0.39	0.31		0.72	0.34
Feb 1995	3.14	0.32	1.17	0.25	0.50[a]	0.38	0.42		nc	0.65
Apr 1995	H	0.43	2.00	0.38	0.61	0.39	0.82			1.47
Sep 1995		0.43	H	1.33	H	1.5	H			H
Apr 1996		H		H		H				

[a] Ratio is less than previous ratio
[b] Lesion bridged at centre and formed 2 lesions

abrupt uplift of the lower margin of the tissue layer, with calcification concentrated at the surface (Barnes & Lough 1993). Similarly, this study showed that a lesion regenerates firstly by upward extension of the surrounding polyps, and secondly by extratentacular budding and lateral encroachment over the dead area.

The presence of filamentous algae on the lesions did not deter regeneration as long as there was an active regenerating edge. These findings are in agreement with Bak & Stewart-van Es (1980) and Meesters & Bak (1993), but I extend the generalisation that healing of lesions is not hindered by some filamentous and macroalgae (e.g. *Padina* sp.) and a light sediment loading (Barnard et al. 1974, Brown et al. 1991, Meesters et al. 1992). Lesion scars were evident in the core samples and clearly the trabeculae structures over the healed lesions were more porous than in other parts of the colony. Indeed, their existence may help clarify the extent of historical damage to Indo-Pacific *Porites* spp.

through sclerochronological investigations (Cameron & Endean 1985, Done 1987, Endean et al. 1988, Keesing et al. 1992) and refine predictive models on the occurrence of small lesions on *Porites astreoides*, for which laudable sclerochronological studies have been carried out (Ruesink 1997).

Some previous studies reported no linear relationship between colony size and regeneration capacity (Bak & Stewart-van Es 1980). Meesters et al. (1994) suggested that healing rates of *Montastrea annularis* depended on the length of the lesion perimeter, but not on the size of the lesion or size of the colony. Oren et al. (1997) showed that the perimeter length of lesions on *Favia favus* influenced recovery rates in the first month of regeneration, but in months following recovery was influenced more by the lesion's surface area, and the surface area to perimeter ratio. Regeneration does however appear to be dependent on the amount of tissue bordering the lesion, since Meesters et al. (1996)
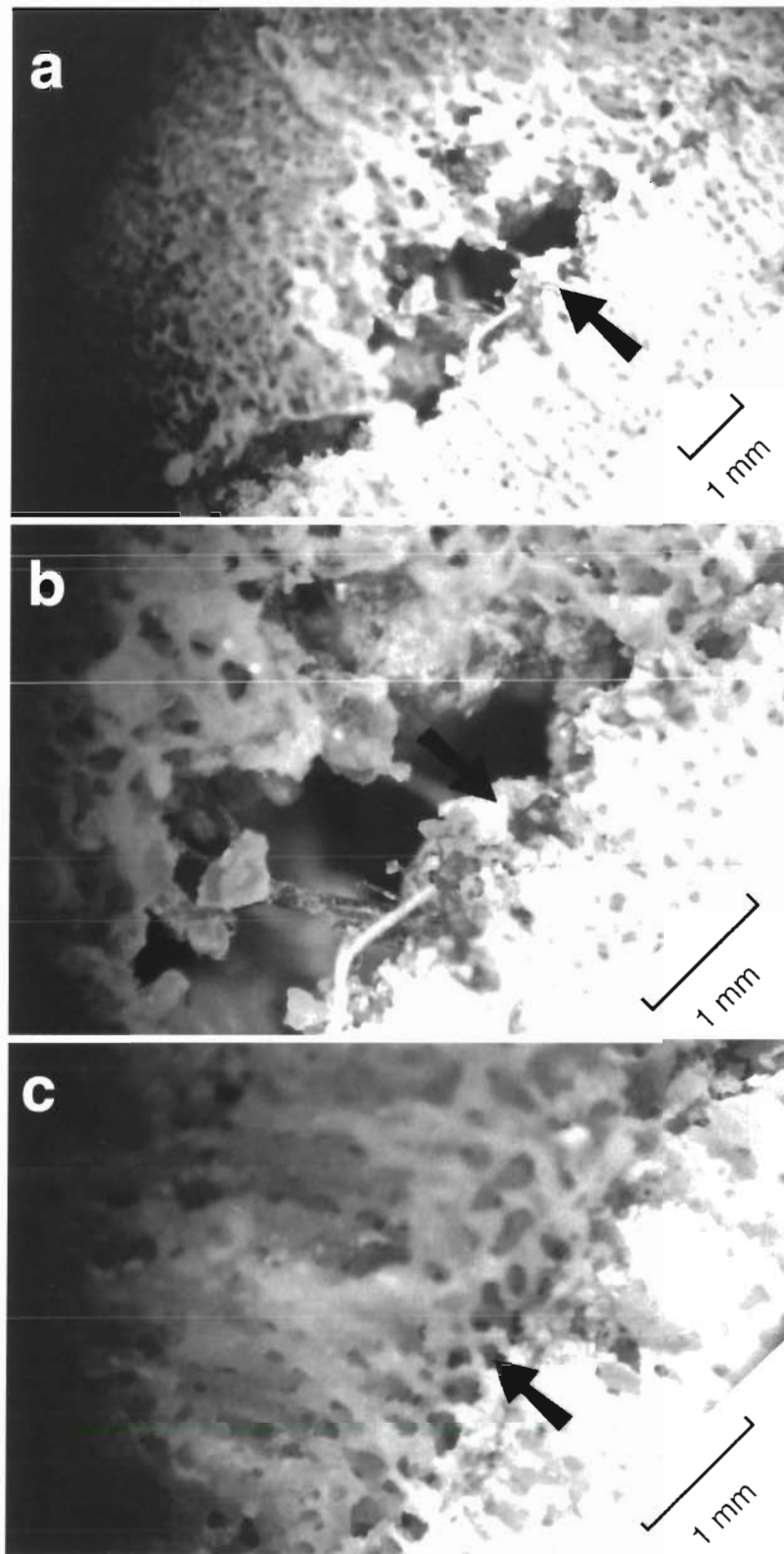
Fig. 2. *Porites lutea* and *Porites lobata*. Cross-sections of coral colonies showing lesion scars from C size lesions. The top left of each photo is near the outer edge of the colony. (a) Lesion scar from Colony 7, *P. lutea*. Arrow points to the underside of the scar. (b) Magnification of scar in (a). Arrow points to sediment particles retained on the lesion. (c) Lesion scar from Colony 10, *P. lobata*. Arrow points to the porous structure between trabeculae (vertical) and synapticulae (horizontal) directly above the healed lesion

showed small colonies with lesions suffered high mortality. The findings of the present study corroborate the idea that the length of the lesion perimeter influences the healing process, as a high perimeter to surface area ratio greatly facilitates healing, and add that regeneration rates are largely dependent on the size of the lesions.

Studies on the massive Atlantic scleractinian corals *Agaricia agaricites* and *Montastrea annularis* indicate that lesions stop regenerating simultaneously and independently of lesion size (Bak et al. 1977, Bak & Steward-van Es 1980). Such a response was interpreted as a termination of the energy source that fueled the healing process (Bak & Steward-van Es 1980). The present study on massive Indo-Pacific *Porites* spp. showed that once healing begins on lesions <1310 mm^2 it progresses until closure. However, this study also showed that regeneration rates decreased with increasing lesion size. Since the response appears to be gradual, it is presumably not a chemical on/off mechanism (Thompson 1917).

A metabolic diffusion gradient and/or active pumping (Taylor 1977, Gladfelter 1983) allows photosynthetic products to be translocated from areas where they are produced to areas where they are in demand, for example to growing axial polyps (Fang et al. 1989) or regenerating edges, where zooxanthellae numbers may be low. The present results suggest that translocation may become more difficult with increasing lesion size as energy reserves are depleted (Rinkevich 1996). If lesions are too large they will not heal through lateral encroachment over the damaged area (Done 1987). The peripheral edge may gradually cease to respond as a unit because of physical constraints preventing the establishment of a coordinated translocation gradient. Establishing a multi-directional gradient may be more limiting than the establishment of a uni-directional response.

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