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Authors

Edmunds, Peter J.
Lenihan, Hunter S.

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ORIGINAL PAPER

Effect of sub-lethal damage to juvenile colonies of massive *Porites* spp. under contrasting regimes of temperature and water flow

Peter J. Edmunds · Hunter S. Lenihan

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Abstract In this study, juvenile colonies of massive *Porites* spp. (a combination of *P. lutea* and *P. lobata*) from the lagoon of Moorea (W 149°50′, S 17°30′) were damaged and exposed to contrasting conditions of temperature and flow to evaluate how damage and abiotic conditions interact to affect growth, physiological performance, and recovery. The experiment was conducted in April and May 2008 and consisted of two treatments in which corals were either undamaged (controls) or damaged through gouging of tissue and skeleton in a discrete spot mimicking the effects of corallivorous fishes that utilize an excavating feeding mode. The two groups of corals were incubated for 10 days in microcosms that crossed levels of temperature (26.7 and 29.6°C) and flow (6 and 21 cm s^{-1}), and the response assessed as overall colony growth (change in weight), dark-adapted quantum yield of PSII (F_v/F_m), and healing of the gouged areas. The influence of damage on growth was affected by temperature, but not by flow. When averaged across flow treatments, damage promoted growth by 25% at 26.7°C, but caused a 25% inhibition at 29.6°C. The damage also affected F_v/F_m in a pattern that differed between flow speeds, with a 10% reduction at 6 cm s^{-1}, but a 4% increase at 21 cm s^{-1}. Regardless of damage, F_v/F_m at 21 cm s^{-1} was 11% lower at 26.7°C than at 29.6°C, but was unaffected by temperature at 6 cm s^{-1}. The lesions declined in area at similar rates (4–5% day^{-1}) under all conditions, although the tissue within them regained a normal appearance most rapidly at 26.7°C and 6 cm s^{-1}. These findings show that the response of poritid corals to sub-lethal damage is dependent partly on abiotic conditions, and they are consistent with the hypothesis that following damage, calcification and photosynthesis can compete for metabolites necessary for repair, with the outcome affected by flow-mediated mass transfer. These results may shed light upon the ways in which poritid corals respond to biting by certain corallivorous fishes.

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P. J. Edmunds (✉)
Department of Biology, California State University, Northridge, CA 91330-8303, USA
e-mail: peter.edmunds@csun.edu

H. S. Lenihan
Bren School of Environmental Science and Management, University of California, Santa Barbara, CA 93106-5131, USA

Introduction

The performance of juvenile corals (≤40 mm diameter) plays an important role in coral community dynamics, particularly following large-scale disturbances (Nuedecker 1979; Vermeij and Sandin 2008). During recovery from severe disturbances, environmental stressors can influence rates of coral recruitment and the growth and survival of juvenile corals (Connell et al. 1997; Edmunds and Elahi 2007; Lenihan et al. 2008), so that coral community structure is a product of chronic and acute events acting on different but overlapping time scales. In many locations, corals are frequently disturbed by corallivores (Bouchon-Navaro 1986; Cole et al. 2008; Rotjan and Lewis 2008), and such events can result in damage that is usually chronic (Cole et al. 2008; Rotjan and Lewis 2008) and sometimes acute (Bruckner et al. 2000; Bruckner and Bruckner 1998). Adult corals have a good chance of recovery from corallivory (Rotjan and Lewis 2008), particularly when it is chronic, but for juvenile corals, the reduction in performance

arising from non-fatal injuries (Grottoli-Everett and Wellington 1997; Miller and Hay 1998; Jayewardene and Birkeland 2006; Christiansen et al. 2009) probably contributes to the high mortality of this size class (Edmunds and Gates 2004) and can affect the rate of recovery of populations following disturbances (Knowlton et al. 1988; Miller et al. 2000; Rotjan et al. 2006).

There is widespread recognition of the importance of corallivory to both corals and organisms that feed upon them (Cole et al. 2008; Rotjan and Lewis 2008), and numerous studies have addressed the effects of corallivory on coral colonies (e.g., Frydl 1979; Rotjan and Lewis 2006, 2009) and their populations (Frydl 1979; Littler et al. 1989). There has also been interest in the rates of healing of artificial lesions (e.g., Oren et al. 1997; Nagelkerken and Bak 1998; Nagelkerken et al. 1999; Oren et al. 2001), in part because such analyses provide insights into how corals recover from corallivory. Despite interest in the effects of corallivory and localized damage on reef corals, few experiments have addressed the effects of physical conditions on the ability of corals to recover from sub-lethal damage (Lester and Bak 1985; Nagelkerken et al. 1999; Kramarsky-Winter and Loya 2000). Nevertheless, it is clear that corallivory can increase coral mortality (Frydl 1979; Miller et al. 2000) and that the damage that corallivores cause can reduce growth rates (Cox 1986), in part because it is hypothesized that corals allocate limited resources to repair damage and regenerate lost tissue (Hall 2001; Henry and Hart 2005). In addition to the consequences of limited resources for wound repair, the regeneration of tissue is affected by other intrinsic factors, such as the capacity for regeneration (Meesters et al. 1997), translocation within the colony (Oren et al. 2001), disease (Aeby and Santavy 2006), and the potential induction of morphological, behavioral, and biochemical responses to the initial disturbance (Gochfeld 2004) (reviewed in Henry and Hart 2005).

Extrinsic factors can also affect the healing of coral tissues following damage because the availability of energetic and cellular resources for regeneration may be altered by the physical environment (Henry and Hart 2005). Temperature is a compelling example of such a physical factor through its direct effects on coral metabolism and growth (Kramarsky-Winter and Loya 2000; Jokiel 2004; Edmunds 2008). Such effects are likely to be non-linear, typically increasing to a threshold value and declining thereafter (Iglesias-Prieto et al. 1992; Edmunds 2005), and therefore the impact of temperature on the regeneration of coral tissue is likely to be complex, and even unpredictable. A similar response characterizes the effects of water flow on corals, with physiological traits increasing with flow speed when they are mass transfer limited, with the response affected by the size and shape of the organism (Patterson 1992) and the mechanical properties of tissues (Sebens and Johnson 1991). Surprisingly, interactions among factors such as temperature, current velocity, and light intensity in the performance of damaged corals and their ability to regenerate tissue are still poorly known (Bak and Steward-Van Es 1980; Lester and Bak 1985; Nagelkerken et al. 1999; Kramarsky-Winter and Loya 2000).

Corallivorous fish are a conspicuous component of coral reefs, with at least 128 species from 11 families occupying this ecological niche (Cole et al. 2008; Rotjan and Lewis 2008). One-third of these feed almost exclusively on corals, with more than 80% of their diet based on this source (Pratchett et al. 2004; Pratchett 2005). On account of their feeding preferences, corallivorous fishes are functionally dissimilar with regard to their impacts on corals, and can be partitioned into mucus feeders, browsers, scrapers, and excavators (Bellwood and Choat 1990; Rotjan and Lewis 2008). Excavators, such as certain scarids, have the greatest impact on corals, and typically remove bites of tissue and skeleton. In addition to variation in feeding modes, corallivorous fishes also show prey preferences, and usually favor species in the genera *Acropora*, *Pocillopora*, and *Porites* (Cole et al. 2008). Based on experiments completed in natural habitats, these genera have been ranked *Acropora* > *Pocillopora* > *Porites* in terms of their ability to regenerate following localized damage (Hall 1997), and therefore it is reasonable to hypothesize that *Porites* spp. are susceptible to fish predation, although it might not be the first choice of fish for food. Interestingly, *Porites* spp. have frequently been studied with regard to its susceptibility to corallivory and its ability to recovery from sub-lethal damage (Frydl 1979; Littler et al. 1989; Nagelkerken et al. 1999; Rotjan and Lewis 2005).

The objective of this study was to explore the physiological responses of juvenile corals to sub-lethal damage under combinations of physical conditions. We sought to experimentally determine the individual and interactive roles of temperature and flow on the ability of corals to recover from damage. Although the damage we inflicted was not identical to that resulting from fish corallivores, we chose a technique that left two parallel lesions that were broadly similar in area and depth to those resulting from excavation by certain scarids (Bellwood and Choat 1990). Thus, while our research is focused on the recovery of corals from sub-lethal damage, our findings are also relevant to understanding how small poritids respond to corallivory by fishes in the excavating functional group. We reasoned that the outcome of sub-lethal damage for corals is likely to be context specific in terms of the physical environment, given the flexibility of coral phenotypes in response to varying conditions including flow, temperature, and light (Lesser et al. 1994; Edmunds 2008; Lenihan et al. 2008). Further, we reasoned that the effects of sub-lethal damage would be accentuated in juvenile corals that have fewer metabolic

resources to draw upon relative to larger colonies (Kramarsky-Winter and Loya 2000), and which experience high rates of mortality (Edmunds and Gates 2004). Studies on the early life stages of corals will help to elucidate the factors regulating the size of scleractinian populations (Vermeij and Sandin 2008). To achieve our objective, we used a manipulative experiment in microcosms to test the hypothesis that the growth, photophysiological, and healing response of juvenile colonies of massive *Porites* spp. (a combination of *P. lutea* and *P. lobata* as described here) to sub-lethal damage is independent of temperature and water flow.

Materials and methods

The experiment employed two indoor tanks that were maintained at different temperatures and contained two flow treatments, and it utilized small colonies (≤40 mm diameter) of massive *Porites* spp. The juvenile corals were collected from the lagoon of Moorea (French Polynesia) and were damaged by gouging with pliers or left undamaged (controls). The corals were exposed to combinations of temperature and flow, and the responses were assessed through growth, maximum dark-adapted quantum yield of PSII (F_v/F_m, a measure of photosynthetic efficiency, Maxwell and Johnson 2000), and change in the size of the lesions. Whole-colony growth was used as a dependent variable as it measured the response of colonies to the treatments and indirectly assessed the regeneration of skeletal material following grazing. As growth was determined as the overall change in weight, it was not possible to distinguish growth of the undamaged portion of the colony from regeneration. We reasoned that colony growth was most relevant to juvenile corals subject to partial mortality (instead, for example, of growth within the lesion), because in small size classes, colony growth is critical in escaping the risks of mortality through an increase in size (Jackson 1977). F_v/F_m was measured to assess the effect of the treatments on *Symbiodinium* sp. physiology, and the size of the lesions was used as a measure of tissue growth and healing at the site of damage.

The tanks were 200 cm × 95 cm and filled to 14-cm depth with unfiltered seawater pumped directly from Cook's Bay (located adjacent to the laboratory; pumping distance ~10 m). These tanks contained 266 l of seawater that was continuously refreshed at 1,000 ml min^{-1}, with the entire volume replaced every 4.4 h. Each tank was fitted with a chiller and heater that maintained the temperature at either ≈26.7 or ≈29.6°C and was illuminated on a 12-h light/12-h dark cycle with two 1,000-W metal halide lamps (Sylvania BT37, Metalarc) suspended overhead. The higher temperature was close to ambient seawater temperature when the experiment was conducted in April/May 2008, and both temperatures spanned the range that occurs in the shallow lagoon of Moorea (Lenihan et al. 2008). The lamps were screened to ≈700 μmol photons m^{-2} s^{-1} (measured with a LiCor LI 193SA), which is lower than the highest irradiance recorded at the collection site at noon on a sunny day (≈1,200 μmol photons m^{-2} s^{-1}). The lower intensity was selected to ensure that the daily integrated light in the experiment was similar to that delivered as a sine curve by normal daylight in the field. Two 170-cm-long raceways were constructed from PVC sheets inserted into each tank, and each raceway was fitted at one end with one or two submersible pumps (2,600 l h^{-1}) that created a low or a high flow treatment, respectively. The outflow from each pump passed through a 5-cm long flow straightener to reduce turbulence, and was adjusted with a valve to achieve target flow speeds of about 5 and 20 cm s^{-1}, which are ecologically relevant for the shallow lagoon where the corals were collected (Lenihan et al. 2008; H. Lenihan, unpublished data). These pumps also maintained vigorous mixing within the tanks that ensured the water was fully aerated.

The corals were collected on April 27, 2008, from reef pavement at 3–4 m depth in the lagoon where high flow speeds (ca. 20 cm s^{-1}) are common (Lenihan et al. 2008). Eighty juvenile colonies of massive *Porites* spp. were collected haphazardly and were removed from the substratum with a chisel that caused minimal damage to the coral tissue. Microscopic inspection of a subset ($n = 20$) of colonies that were killed and bleached at the end of the experiment revealed that 85% were *P. lutea* and 15% *P. lobata*. It proved unreliable to identify the corals to species when they were alive before the experiment began, but the random allocation of colonies to treatments reduced the likelihood that taxonomic effects influenced the experimental outcomes.

Freshly collected corals were returned to the laboratory and glued (with Z Spar, A788 epoxy) to the tops of pieces of numbered PVC pipe (≈2 cm long and 1.5 cm diameter), the bottoms of which were fitted with a 6-mm diameter nylon screw. This procedure created experimental units that could be inserted into PVC racks drilled to accept the screw attached to their bases, thereby holding 20 corals upright in a linear array. The prepared corals were fitted onto the PVC racks and left to recover for ≈36 h in the tank held at ambient temperature and illuminated for 12 h each day at 700 μmol photons m^{-2} s^{-1}. Following recovery, 40 corals were injured by gouging with 10-mm wide, snub nose pliers. The gouges were made in haphazard locations on the colonies, and the pliers were applied to create a single scar (mimicking one bite on each colony) consisting of a pair of rectangular lesions ≈12 mm × 6 mm. The pliers were rinsed in seawater between damaging colonies to reduce contamination, and the sizes of the lesions (length × width)

were recorded with calipers (±1 mm). The gouging removed tissue and skeleton to 1–2 mm depth but did not remove the full thickness of tissue, which extends 2–5 mm into the skeleton of massive *Porites* spp. (Edmunds 2008). These lesions are similar in dimensions and shape both to the paired lesions seen naturally on *Porites* spp. on the reefs of Moorea (P. J. Edmunds, personal observation), and those frequently recorded on *Porites* spp. and attributed to the excavating bites of scarids (e.g., Fig. 14B in Bellwood and Choat 1990; Fig. 1a in Rotjan and Lewis 2008). In cases where scarids (and other fishes) remove the entire tissue thickness when they bite poritid corals, the outcome (i.e., the extent of recovery) is likely to differ from that reported here.

Following gouging, the corals (undamaged and damaged) were weighed by a buoyant procedure (Davies 1989) so that their growth (change in weight of the skeleton) could be determined and returned to the tank at ambient temperature. Following 3 h of darkness (beginning at 18:30 h), photosynthetic efficiency of the corals was assessed as maximum dark-adapted quantum yield of PSII (F_v/F_m) using a Diving-PAM (Walz GmbH) fitted with an 8-mm diameter probe and operated with constant settings. F_v/F_m was selected as an dependent variable because photosynthesis is affected by temperature and flow (Patterson 1992; Jones et al. 1998) and, therefore, we reasoned that such effects might influence the response to damage through modified availability of photosynthetically fixed carbon. Measurements of F_v/F_m were made with the probe held 5-mm above the tissue using a plastic spacer, and all measurements were made at haphazard positions on the corals, and for the damaged corals, ≈10 mm outside the area of gouging. The initial records of F_v/F_m were used to evaluate the overall condition of the corals at the start of the experiment, and to determine whether the incubation procedure caused a deterioration in condition of the corals, regardless of the temperature and flow speeds employed. Finally, the corals were allocated randomly to each of the four racks, with 20 undamaged and 20 damaged colonies on each rack. The following morning (April 30), the incubations began with the allocation of the racks at random to either the high flow or the low flow treatments within the ambient and the cool tanks. Thereafter, the experiment was maintained for 10 days until its conclusion at 20:00 h on May 10, 2008.

Throughout the experiment, the seawater temperature was recorded (every 30 min) with loggers (Hobo Aquapro, Onset Computer Company), and light intensity and water motion were measured periodically. The light intensity was measured using a 4π quantum sensor (LiCor LI 193SA) at three positions along each of the two raceways in each tank. Water motion was assessed as net water transport, which was measured by timing the repeated passage of neutrally buoyant particles along a known distance. This technique was not intended to measure the absolute flow speed experienced by each coral, but instead, characterized the net movement of water along the raceway. As the water motion varied along the raceway, and upstream corals affected the flow around downstream corals, the position of all corals within each raceway was randomized daily. Randomization was achieved by removing the corals from the racks each morning and replacing them in the same racks but at new, randomly selected positions; this was accomplished without touching the coral tissue or removing them from the seawater.

At the conclusion of the experiment, F_v/F_m was again measured after 3 h of dark adaptation, the status and sizes of the lesions was measured, and the buoyant weights recorded. The status and size of lesions were measured by ranking them based on signs of healing (evidenced by the return of color and tissue over the skeleton), or as almost/fully healed (where the color was almost the same as the rest of the coral and the lesion was indistinct), and assessing their sizes as described earlier. The buoyant weights were recorded, and the change in buoyant weight converted to dry weight assuming that the skeletal aragonite had a density of 2.93 g cm^{-3} (Jokiel et al. 1978). Following the final weighing, the corals were dried at 60°C, and their tissue areas measured using aluminum foil (Marsh 1970) to normalize growth rates to area (mg cm^{-2} day^{-1}).

To test for treatment effects, three-way fixed-effects ANOVAs were used in which growth and F_v/F_m were dependent variables in two separate analyses, and the fixed factors were damage regime (undamaged vs. damaged), temperature (two levels), and water motion (two levels); interaction terms were pooled with the error term when not significant at $P \geq 0.25$ (Quinn and Keough 2002). The percentage change in lesion areas during the experiment was compared between treatment combinations with a one-way ANOVA. F_v/F_m values were log transformed prior to analyses, and the statistical assumptions of the procedures were tested through a graphical analysis of residuals. Planned comparisons between undamaged and damaged corals were completed according to Sokal and Rohlf (1995). The effect of the treatments on the status of the lesions at the conclusion of the experiment was evaluated with a 2 × 4 contingency table in which the number of corals with lesions categorized as "showing signs of healings" versus "almost/fully healed" was tested for independence of the treatment levels using a χ^2 test. Coral colonies were treated as statistical replicates in all analyses, even though the experimental design was pseudoreplicated (Hurlbert 1984). We rationalized our statistical approach by the independence of the corals created by their small aggregated tissue volume relative to that of seawater in the tanks (a volume ratio of <1:7,300), and the rapid replacement of this water (every

4.4 h) by the constant inflow. Nevertheless, we cannot exclude the possibility that our treatments were biased by an undetected effect that was unique to one of the tanks. An additional experiment with the same apparatus demonstrated, however, that the treatment effects were reproducible, and therefore not simply a result of a temporally random process such as a dead animal falling into one tank. In the additional experiment, a manipulation similar to the one described here was completed with *Pocillopora verrucosa*, although it was replicated in two independent trials, one of which was concurrent with the present experiment (H. Lenihan and P.J. Edmunds, unpublished data). Statistical analysis of the *P. verrucosa* experiment revealed no effect of trial, thereby demonstrating that the treatment conditions (as used here for *Porites* spp.) were reproducible.

Results

The microcosms maintained the treatments with a high degree of precision. The mean temperatures in the two tanks were 26.7 ± 0.01°C (n = 533) and 29.6 ± 0.01°C (n = 528) (±SE), and the mean light level in the tanks was 770 ± 12 μmol photons m^{-2} s^{-1} (±SE, n = 72). The mean low flow speed was 5.6 ± 0.1 cm s^{-1} (±SE; n = 30 for all flow measurements) in the low temperature tank, and 5.8 ± 0.3 cm s^{-1} in the high temperature tank. For the high speed treatments, flow was 21.0 ± 0.5 cm s^{-1} (low temperature tank) and 21.8 ± 0.7 cm s^{-1} (high temperature tank) (all ±SE, n = 30). Net water transport did not vary between low speed treatments (t-test, t = 0.908, df = 58, P = 0.368) or between high speed treatments (t-test, t = 1.004, df = 58, P = 0.320).

During the 10-day incubations, none of the massive *Porites* spp. died, although one colony showed signs of partial mortality. Apart from this colony, all others appeared healthy throughout the experiment, and there were no signs of bleaching or poor health. Growth rates were obtained from the majority (96%) of the corals, but three colonies were excluded from the analysis due to computational errors. Overall, growth rates ranged from 0.04 to 7.81 mg cm^{-2} day^{-1}, and the mean rates for each treatment varied from 1.09 ± 0.17 to 1.95 ± 0.31 mg cm^{-2} day^{-1} in the low flow/high temperature and high flow/low temperature treatments, respectively (±SE, n = 8–10). The damage affected growth (Fig. 1), but the effects were dependent on temperature (Table 1). The interaction between damage and temperature reflected outcomes that were unaffected by flow, with damage increasing mean growth rates by 25% at 26.7°C (from 1.39 to 1.74 mg cm^{-2} day^{-1}), but reducing them 25% at 29.6°C (from 1.58 to 1.18 mg cm^{-2} day^{-1}); in both cases, these percentages reflect the differences in the response to damage at each temperature after averaging across flow regimes (Fig. 1). In addition to these statistically significant effects, there was also a trend (P = 0.060) for the effects of damage to interact with flow, with damage increasing mean growth (pooled between temperatures) rates by 27% at 21 cm s^{-1}, but reducing them by 23% at 6 cm s^{-1} (Fig. 1). Relative to undamaged corals, planned contrasts revealed that damage increased growth significantly under the high flow/low temperature treatment (ANOVA, F = 4.773, df = 1,70, P = 0.032), and there was a

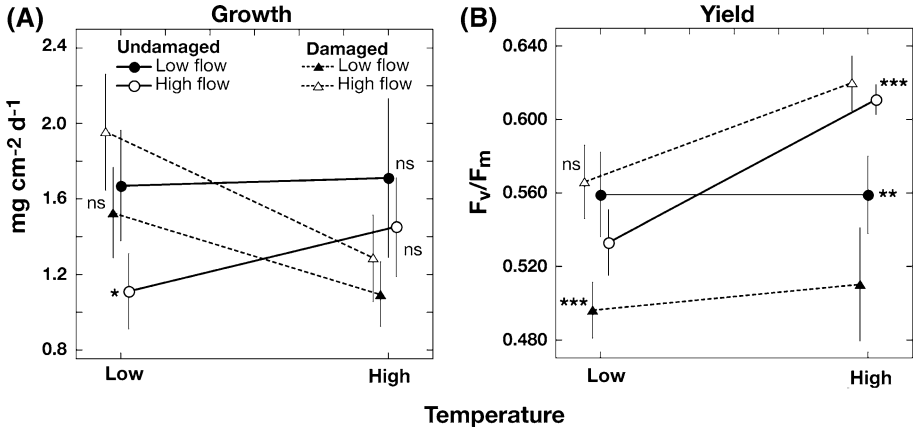

Fig. 1 Interaction plots for juvenile massive *Porites* spp. displaying **a** whole-colony growth (i.e., growth of undamaged portion of the colony plus regeneration of skeleton within the lesion) and **b** maximum dark-adapted quantum yield of PSII (F_v/F_m) for colonies either undamaged (*circles*) or damaged (*triangles*) and exposed for 10 days to orthogonal combinations of temperature [low (26.7°C) or high (29.6°C)] and flow [low (6 cm s^{-1}, *filled symbols*) or high (21 cm s^{-1}, *open symbols*)]. Results of planned contrasts of undamaged versus damaged groups within each temperature and flow combination were completed according to Sokal and Rohlf (1995) using the MS_{error} from the ANOVAs for significance testing; *ns* = not significant at $P > 0.050$, * = significant at $P \leq 0.050$, ** = $P < 0.010$), and *** = $P < 0.001$. Contrasts tested for differences between *circle* and *triangle symbols* of the same color located within each temperature treatment in each panel of the figure. Means ± SE displayed (n = 10), with symbols offset for clarity

Table 1 Results of a three-way model I ANOVA comparing growth rate (mg cm^{-2} day^{-1}) of juvenile massive *Porites* spp. among flow regimes, temperatures, and damage treatments (all fixed factors)

Source	SS	*df*	MS	*F*	*P*
Damage	0.020	1	0.020	0.030	0.864
Temperature	0.584	1	0.584	0.885	0.350
Flow	0.059	1	0.059	0.089	0.766
Damage × temperature	2.642	1	2.642	4.006	0.049
Damage × flow	2.420	1	2.420	3.669	0.060
Error	46.172	70	0.660		

Statistical interactions of flow × temperature and flow × temperature × damage were not significant ($P \geq 0.472$) and were pooled with the error term (and are not displayed in the table) for significance testing (Quinn and Keough 2002)

Table 2 Results of a three-way model I ANOVA comparing maximum dark-adapted quantum yield of PSII (F_v/F_m, log transformed) of juvenile massive *Porites* spp. among flow regimes, temperatures, and damage treatments (all fixed factors)

Source	SS	*df*	MS	*F*	*P*
Damage	0.019	1	0.019	2.069	0.155
Temperature	0.090	1	0.090	10.061	0.002
Flow	0.096	1	0.096	10.750	0.002
Damage × temperature	0.006	1	0.006	0.724	0.398
Temperature × flow	0.048	1	0.048	5.330	0.024
Damage × flow	0.088	1	0.088	9.832	0.003
Damage × temperature × flow	0.034	1	0.034	3.837	0.054
Error	0.628	70	0.009		

trend for damage to reduce growth under the low flow/high temperature treatment ($F = 2.905$, $df = 1,70$, $P = 0.093$); the other two contrasts were also not significant but showed no trend (ANOVA, $F < 0.754$, $df = 1,70$, $P > 0.388$).

At the start of the experiment, mean F_v/F_m for the freshly collected corals was 0.631 ± 0.005 (±SE, $n = 80$) and was not different between the damaged and undamaged corals (*t*-test, $t = 0.466$, $df = 78$, $P = 0.643$). After the incubation, F_v/F_m for individual corals ranged from 0.519 to 0.753. At the end of the experiment, mean F_v/F_m for each treatment ranged from 0.496 ± 0.015 under the low flow/low temperature conditions to 0.620 ± 0.015 under the high flow/high temperature (±SE, $n = 10$ for all groups). Damage and temperature affected F_v/F_m, in both cases in patterns that differed between flow treatments (Table 2). The statistically significant interaction between temperature and flow was a result of effects that were independent of damage, with F_v/F_m largely unaffected by temperature at 6 cm s^{-1}, but stimulated 12% by the high temperature at 21 cm s^{-1} (i.e., when damage treatments are pooled, Fig. 1). The marginally significant three-way interaction among the main effects (ANOVA, $P = 0.054$, Table 2), however, suggested that F_v/F_m was affected by an interaction between damage and flow that varied between temperatures. To unravel the complexity of three-way interaction, planned contrasts were conducted, which revealed that damage increased F_v/F_m significantly under the high flow/high temperature treatment (ANOVA, $F = 19.579$, $df = 1,70$, $P < 0.001$), but caused declines under both the low flow/high temperature and low flow/low temperature treatments (ANOVA, $F > 9.526$, $df = 1,70$, $P < 0.003$); there was no effect under the high flow/low temperature treatment (ANOVA, $F = 2.761$, $df = 1,70$, $P = 0.101$). Finally, the interaction between damage and flow reflected effects that that were not influenced by temperature, with damage depressing the mean F_v/F_m values (pooled between temperatures) by 10% at 6 cm s^{-1} (from 0.559 to 0.503), but causing a 4% increase at 21 cm s^{-1} (from 0.572 to 0.593) (Fig. 1).

Virtually all of the corals appeared to recover quickly from damage, with only one developing a small lesion that exposed the carbonate skeleton. In the first few days following the gouging, most corals in all treatments produced excess mucus near the site of damage, but mucus production declined rapidly (<3 h) so that undamaged and damaged corals appeared identical with regard to mucus release. After the first few days, the gouged areas quickly developed the normal yellow/green color of healthy (undamaged) coral tissue, and the margins became less distinct. By the end of the experiment, six of the damaged corals (7.5%) appeared to have recovered fully in terms of tissue color and tissue continuity within the lesion, although the margins of the lesion were still visible. As most of the lesions, regardless of treatment, showed signs of recovery, it often proved challenging to identify the margins with confidence when the final measurements of size were taken. Nevertheless, the mean areas of the lesions, which initially ranged from 58 ± 3 to 75 ± 7 mm^2 (±SE, $n = 10$) in each of the treatments, decreased, ending the experiment with mean areas ranging from 32 ± 8 to 42 ± 4 mm^2 (±SE; $n = 10$). The percentage decline in size of the lesions did not vary between temperatures (ANOVA, $F = 1.340$, $df = 1,36$, $P = 0.255$) or flow speeds (ANOVA, $F = 0.351$, $df = 1,36$, $P = 0.557$), and there was no significant interaction between temperature and flow (ANOVA, $F = 0.032$, $df = 1,36$, $P = 0.582$). The mean rates of decline in lesion areas over the 10-day experiment ranged from 42 ± 5 to 54 ± 9% (±SE, $n = 10$) depending on treatment (Fig. 2), and were equivalent to healing rates of 4–5% day^{-1}. Although the sizes of all of the lesions decreased at similar rates, the visual assessment of the lesion condition was affected by the treatments (Table 3). Under the low flow/low temperature treatment, more corals than expected showed signs of complete healing, whereas under the high

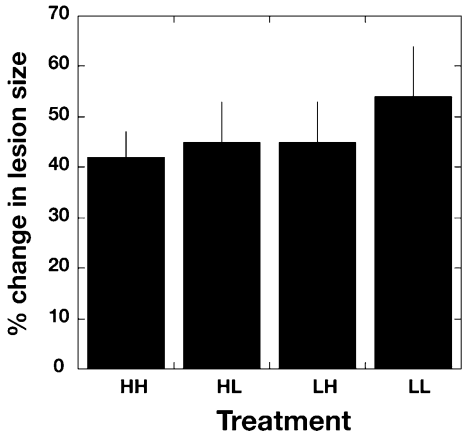

Fig. 2 Percentage change in lesion size (area) on juvenile colonies of massive *Porites* spp. after 10 days under treatments of high flow and high temperature (*HH*), high flow and low temperature (*HL*), low flow and high temperature (*LH*), and low flow and low temperature (*LL*); mean ± SE shown ($n = 10$ for all). Change in lesion area was unaffected by treatment (ANOVA, $F = 0.667$, $df = 3,36$, $P = 0.587$), and size of lesions (mm^2, data not shown) was unaffected by temperature, flow, or the interaction between the two ($P \geq 0.255$). At the start of the experiment, mean size of all lesions was 68 ± 3 mm^2 (±SE, $n = 40$)

Table 3 Contingency table showing number of *Porites* spp. colonies at the end of the experiment with scars scored as either showing signs of some healing or mostly/fully healed under the four treatment combinations; $n = 10$ corals for each treatment combination in the damaged group

	High flow		Low flow	
	High temperature	Low temperature	High temperature	Low temperature
Some healing	10	6	6	3
Mostly/fully healed	0	4	4	6

Only one coral showed no healing, as determined from a lesion that revealed fully exposed skeleton. Observations in rows were not independent of observations in columns ($\chi^2 = 9.5$, $df = 3$, $P = 0.024$)

flow/high temperature treatment, more corals than expected show some signs of healing, but none were scored as fully healed.

Discussion

The results of our experiment reveal interactions between temperature and flow on the response of corals to damage, with the outcomes differing from those expected from the linear combination of the component effects. For example, if coral metabolism is accelerated at 26.7°C compared with 29.6°C (Edmunds 2005)—assuming thermal optima for physiological processes of ≈26–29°C (Buddemeier and Kinzie 1976; Iglesias-Prieto et al. 1992; Edmunds 2005)—and at 21 cm s^{-1} compared with 6 cm s^{-1} (Patterson 1992), it is reasonable to predict that our corals should have grown most rapidly at 26.7°C and 21 cm s^{-1}. Moreover, because host–symbiont communication is poorly understood (Baird et al. 2009), when we began the experiment, we had no evidence that localized damage would affect the performance of *Symbiodinium* sp. in nearby tissue. In contrast to these expectations, our analyses revealed only equivocal separate effects of damage, temperature, or flow on growth, and instead, demonstrated that damage elicited growth responses that depended on temperature, and statistically were unaffected by flow. Surprisingly, photophysiological performance (F_v/F_m) was also affected by damage, and the relationship varied with flow speed in a pattern that differed between temperatures.

Our results show that juvenile *Porites* spp. subject to sub-lethal damage: (1) grew fastest at 26.7°C compared to 29.6°C, and faster than undamaged corals, and while not statistically significant, growth also tended to be greater at 21 cm s^{-1} compared to 6 cm s^{-1} (as predicted), (2) underwent changes that affected F_v/F_m of *Symbiodinium* sp. outside the lesion, causing increases at high flow, and reductions at low flow, and (3) displayed regenerative capacities that caused damaged tissue to regain its normal appearance most rapidly at low flow and low temperature, although the healing of the lesions (i.e., their size) was unaffected by flow or temperature. These results demonstrate for juvenile massive *Porites* spp. in shallow water that localized, sub-lethal damage can be highly survivable. Further, they suggest that the ability of corals to recover from such damage is driven by rapid changes in growth and photophysiology modulated by temperature and flow, including mass transfer effects (Patterson 1992), as well as integration between the cnidarian host and the *Symbiodinium* sp. symbionts. Juvenile *Porites* spp. colonies exposed to high flow at low temperatures appear to experience a growth advantage as a result of damage, thereby promoting a positive feedback between certain kinds of damage and colony growth. Previous studies of the effects of injuries on coral growth have reported conflicting results, with the growth of *Stylophora pistillata* accelerated by damage (branch breakage) in the Red Sea (Loya 1976), but the growth of *Montastraea annularis* impeded for a protracted period (56 days) by small lesions on a reef in Curaçoa (Meesters et al. 1994). There are several reasons for these contrasting outcomes with *S. pistillata* and *M. annularis*, including differences in seawater temperature when the studies were completed, and dissimilar reproductive strategies, but the differences in depth are particularly interesting. Notably, *S. pistillata* was studied in shallow water (2–4 m) compared to *M. annularis* (7 m), and this contrast in depth is consistent with the stimulatory effect of light on coral repair (Bak and Steward-Van Es 1980; Nagelkerken et al.

1999) and the finding in the present study (which was also conducted at high light) that flow tended to enhance growth.

Damage to a coral colony requires energy to heal, and this energy fuels replacement of the tissue and the aragonite skeleton. These two processes are not independent, because tissue cannot be formed without underlying skeleton, and skeleton cannot be formed without overlying tissue (Barnes 1973). However, the relationship between the two is looser than was previously recognized (Anthony et al. 2002), with some corals maintaining skeletal extension at the expense of biomass (Anthony et al. 2002), and many increasing biomass independent of skeletal growth as suggested by seasonal variation in biomass (Fitt et al. 2000). In the case of lesions on the surface of a coral, the extent to which such damage is repaired depends on the size of the lesion and the size of the coral (Oren et al. 2001), whether fleshy algae or other biota colonize the exposed skeleton before a repair can be effected (Meesters et al. 1997), and a variety of abiotic factors (Henry and Hart 2005), two of which (temperature and flow) are the subject of this study. If the tissue is removed completely, then healing can only be initiated marginally (Meesters et al. 1997), whereas incomplete tissue removal (for example, leaving tissue within injured corallites) creates the possibility for healing from both the margins of the lesion and the residual tissue (Bak and Steward-Van Es 1980; Meesters et al. 1997). It is also possible that "prioritization rules" dictate whether resources (e.g., stem cells and energy) should be allocated to healing, reproduction, or other needs (Rinkevich 1996), with the trade-off made more complex by the possibilities of uncoupling skeletal and tissue repair (Anthony et al. 2002).

In the case of juvenile massive *Porites* spp., coral tissue extends 2–4 mm into the perforate skeleton (Edmunds 2008), and it was not completely removed by the damage we inflicted. Careful inspection of the freshly gouged corals revealed tissue in the sheared corallites within the lesions, and we suggest that this tissue played an important role in the regrowth of the damaged area. The principal evidence for the role of this residual tissue in healing is the speed with which the normal tissue coloration returned to the lesions (i.e., <10 days for lesions up to 75 mm^2 in area, cf. Henry and Hart 2005), and the absence of signs that the tissue first encroached from the margins of the wound. As the wounds healed, the skeleton appeared to be replaced at a dissimilar rate to that of the tissue, because the lesions at the end of the study still appeared slightly flattened and characterized by sheared corallites. Interestingly, the return of relatively normal tissue color within the lesions occurred most frequently in the low temperature and low flow treatment, which suggests that these conditions promote the growth of coral tissue and perhaps their *Symbiodinium* sp. (but not necessarily the skeleton).

The effects of damage on skeletal growth were influenced by temperature, with damaged corals maintaining growth rates that were virtually indistinguishable from undamaged corals at 26.7°C and 6 cm s^{-1}, but were nearly double those of undamaged corals at 26.7°C and 21 cm s^{-1}; damage impeded growth at 29.6°C, regardless of flow regime. Although we cannot rule out the possibility that damage affected growth throughout the coral, for example through reoriented growth outside the damaged areas (Tanner 1997), in the absence of evidence to the contrary, we infer that the effects of damage on growth were largely a result of skeletogenesis within the lesions. Enhancement of growth at 27°C compared with 30°C suggests that the thermal optimum for growth in massive *Porites* spp. is <30°C, which is consistent with the effects of temperature on a variety of corals (Buddemeier and Kinzie 1976; Edmunds 2005). However, temperature clearly is not the only factor determining the growth rates in the present study, because there was also a trend for it to be affected by a flow × damage interaction, with damage tending to enhance growth at 21 cm s^{-1}, but depress it at 6 cm s^{-1}. Since a primary effect of flow in aquatic environments is to modulate the transfer of nutrients to benthic taxa (Patterson 1992), a parsimonious explanation for the flow × damage interaction on growth is that the growth response to damage is limited by the transport of nutrients from seawater. Given the importance of exogenous DIC (i.e., from the seawater) to coral calcification (Allemand et al. 2004), the present results are consistent with the hypothesis that higher flow speeds increase DIC transport into coral tissues, thereby sustaining the enhanced demands for DIC as growth within the lesion accelerates at a lower temperature.

Symbiodinium sp. are not autonomous from their coral host, being dependent on them for nitrogen and in return are regulated by them (Yellowlees et al. 2008). Nevertheless, we did not expect damage to affect F_v/F_m in areas adjacent to the lesions. In contrast, damage affected F_v/F_m in these areas, although the effect was small ($\leq$16%) and influenced by flow. This finding suggests that localized injury, or recovery from it, perturbs the internal milieu within the *Porites* spp. colonies, thereby affecting the photophysiology of *Symbiodinium* sp. in areas outside of the area of damage. This study did not identify how localized injury might have more distant effects within the colony, which might be a consequence of competition between the host tissue and *Symbiodinium* sp. for metabolites used in repair and photosynthesis, respectively. Again, the strong influence of flow in modulating the effect of damage on F_v/F_m suggests that mass transfer effects can modulate this competition under certain circumstances. One situation through which such a circumstance might arise, is the demand for DIC for skeletogenesis (which is accentuated at 26.7°C as described earlier) and carbon fixation through

photosynthesis (Langdon and Atkinson 2005). Conceivably, the demands for DIC for damage repair at low flow conditions could create limited supplies of this metabolite, thereby causing incomplete photosynthetic quenching of high energy electrons originating in the light reactions of photosynthesis, and hence promoting the formation of superoxide radicals (O_2^-) (Jones et al. 1998). Once formed, O_2^- function as oxidants and can depress F_v/F_m through a variety of mechanisms known as photoinhibition (Franklin et al. 2006). If this model is applied to the present results, F_v/F_m is depressed by damage because recovery creates a shortage of DIC throughout the corals, which leads to the formation of harmful O_2^-; high flow enhances the supply of DIC, hastens the removal of O_2^-, and prevents photoinhibition of F_v/F_m.

While we contend that our study provides valuable insights into the response of *Porites* spp. to fish predation, it is important to recognize that our analysis has three limitations. First, logistics restricted our experiment to a pseudoreplicated design that makes it impossible to reject the alternative hypothesis that the treatment effects were confounded. As described earlier in the methods, there is strong evidence to argue against this interpretation. Second, while the damage we inflicted was with pliers (i.e., not fish corallivory), the damage was similar to that caused by the excavating functional group of corallivorous fishes (Bellwood and Choat 1990; Rotjan and Lewis 2008), at least when they feed in a spot biting mode. Thus, it is reasonable to infer that the response of juvenile *Porites* spp. to damage in the present study provides a good indication of how they might respond to fish predation in shallow habitats where the conditions are similar to those employed in the microcosms. Third, because small colonies of *Porites* spp. were used, and each was exposed to only one simulated fish bite, our conclusions are only truly valid for functionally similar corals subject to similar damage. Work with the Caribbean congener *P. astreoides* supports this assertion, because *P. astreoides* colonies that are heavily impacted by fish corallivory rarely recover (Rotjan and Lewis 2005). Nevertheless, our results demonstrate why the interactions between excavating corallivorous fishes and corals might be expected to be complex and dependent on environmental conditions. Just as the death of corals can affect the diversity and abundance of coral reef fishes through changes in habitat quantity and quality (Bell and Galzin 1984; Jones et al. 2004; Wilson et al. 2006), so too can changes in the abundance and diversity of corallivores affect the distribution of corals (McClanahan and Graham 2005; Berumen and Pratchett 2006; Cole et al. 2008), in part through effects on coral performance. To date, few studies have examined ecological mechanisms by which corallivory influences coral distribution (but see Nuedecker 1979; Miller and Hay 1998; Miller et al. 2000; Cumming 2002; Rotjan and Lewis 2009), and experimental analyses of coral performance (i.e., colony growth), as completed here, may help to explain how corallivory can have effects that cascade to the community level (but see Cox 1986; Littler et al. 1989; Grottoli-Everett and Wellington 1997; Rotjan and Lewis 2005; Chasqui-Velasco et al. 2007).

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