Vol. 342: 139–149, 2007 | MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser | Published July 24

Effect of macroalgal competition on growth and survival of juvenile Caribbean corals

Steve J. Box[1, 2,*], Peter J. Mumby[1]

[1]Marine Spatial Ecology Laboratory, School of BioSciences, University of Exeter, Prince of Wales Road, Exeter EX4 4PS, UK

[2]Coral Cay Conservation, 40–42 Osnaburgh Street, London NW1 3ND, UK

ABSTRACT: *Lobophora variegata* and *Dictyota pulchella* are dominant algal components on coral reefs across the Caribbean, but the mechanisms and outcomes of spatial competition between these algae and scleractinian corals are poorly understood. In this study the effects on growth and mortality of juvenile corals by 2 forms of algal competition, shading and abrasion, were investigated. The growth of small *Agaricia* spp. (<20 mm diameter) was monitored over a 14 mo period on shallow forereefs in Roatán, Honduras. Experimental manipulations of algal shading and algal contact with the periphery of colonies were conducted in isolation from the effects of grazing through the use of exclusion cages. Shading by *L. variegata* caused an overall loss of coral tissue and significantly increased colony mortality rates from 0 to 50% in 6 mo. The presence of *L. variegata* around the periphery of a coral colony significantly reduced the overall growth of juvenile corals, decreasing the growth rate to 60% of that of control corals, but had no detectable effect on mortality. Shading by *D. pulchella* resulted in 99% growth inhibition (i.e. to just 1% of the growth rate of control corals). Peripheral contact with *D. pulchella* (without shading) also retarded coral growth rates but to a lesser extent: to 31% of that of controls. A synthetic alga made to mimic the action of *D. pulchella* abrasion caused a similar reduction in growth rate to actual *D. pulchella,* suggesting that the reduction in coral growth occurred because of physical mechanisms rather than allelochemical inhibition. The severe inhibition of colony growth caused by the proximity of *D. pulchella* or *L. variegata* may extend a coral's period of vulnerability to whole colony mortality. Based on the monthly mortality rate observed in uncaged control corals of 0.035 ± 0.135 (SE), peripheral contact with *D. pulchella* could decrease the survivorship of corals reaching a 3 cm diameter from 29 to <2%. Peripheral contact with *L. variegata* could likewise decrease cohort survival to 11%. The ability of these common macroalgae to reduce the survivorship of juvenile corals through interference competition could contribute to the perpetuating dominance of macroalgae on many Caribbean reefs.

KEY WORDS: Macroalgae · Coral · Competition · Growth · Phase shifts · Survival probability

INTRODUCTION

Many Caribbean coral reefs are facing unprecedented levels of disturbance with declines in hard coral cover and a shift towards macroalgal dominated communities (Done 1992, Hughes 1994). The increased incidence of coral–algae interactions (Lirman 2001, River & Edmunds 2001) and growing evidence that scleractinian corals and macroalgae compete for space (Tanner 1995, Jompa & McCook 2002, Nugues & Bak 2006) suggest that such interactions may have a key role in structuring the benthic community of Caribbean coral reefs. Further, with levels of grazing being depleted in parts of the Caribbean by limited urchin abundance (Carpenter & Edmunds 2006) and fishing of herbivorous fishes (Hawkins & Roberts 2004), there is great concern that macroalgal communities could become a stable alternate community state of reefs (Hughes et al. 2003).

*Email: s.j.box@exeter.ac.uk

Macroalgae may compete with corals through a number of proposed mechanisms. These include direct physical interactions such as basal encroachment, shading or abrasion (Lirman 2001), the indirect influences on micro-habitat through flow reduction and increased localised sedimentation (Nugues & Roberts 2003), chemical induced mortality (Littler & Littler 1997), enhanced microbial activity caused by algal exudates (Smith et al. 2006) and by acting as a reservoir for coral pathogens (Nugues et al. 2004). Macroalgae may also compete at a population level through space pre-emption (Mumby et al. 2005) reducing available space for the successful settlement of coral larvae (Birrell et al. 2005).

The outcomes of competitive interactions among taxa are likely to be influenced by colony size. Small corals have a smaller reservoir of energy to invest in competitive interactions and are more vulnerable to whole colony mortality than larger individuals (Zilberberg & Edmunds 2001, Raymundo & Maypa 2003). Thus juvenile corals that have survived initial post settlement mortality and undergone early growth and division to form small colonies may be particularly susceptible to macroalgal competition. Smaller colonies also have morphological disadvantages in spatial competition due to their lack of height and consequently may be vulnerable to overtopping, abrasion and shading.

We tested the effects of 2 morphologically distinct brown algae, *Dictyota pulchella* (Hörnig and Schnetter) and *Lobophora variegata* (Lamouroux), on the growth and survival of juvenile *Agaricia* spp. in shallow forereefs dominated by the coral *Montastraea annularis*. *Agaricia* spp. are among the predominant hermatypic corals on Caribbean reefs (Aronson et al. 2004, Bak et al. 2005) and their adult populations are considered particularly vulnerable to short term changes in their recruitment success (Hughes & Tanner 2000). The algal species, *D. pulchella* and *L. variegata,* dominate the macroalgal community on many coral reefs (McClanahan et al. 1999, Mumby et al. 2005) but this is the first study to test their influence on the growth of juvenile corals (defined as having a diameter <20 mm). 2 mechanisms of macroalgal competition, abrasion and shading were investigated. The *a priori* hypotheses were (1) that shading and abrasion of either algae would inhibit growth and elevate mortality (Lirman 2001, Jompa & McCook 2002, Nugues & Bak 2006), (2) the thicker and broader thallus of *L. variegata* would cause a greater shading effect than *D. pulchella*, (3) the flexible fronds of *D. pulchella* would cause a higher level of abrasion effect than *L. variegata* and (4) that the physical attributes of *D. pulchella* fronds rather than allelochemical inhibition cause this effect.

MATERIALS AND METHODS

Study site. The study was conducted on the island of Roatán, the largest island of the Bay Islands archipelago. Roatán lies approximately 50 km from the Caribbean coast of Honduras and is considered to be the southernmost extent of the Mesoamerican barrier reef system (Harborne et al. 2001). Studies were located at the NW end of Roatán on *Montastraea annularis* dominated fringing reefs 500 m offshore (16° 16′ N, 86° 35′ W).

Coral colony selection. Juvenile corals of *Agaricia* spp. were haphazardly chosen by swimming along a depth stratum of approximately 8 m. *Agaricia* spp. were chosen for this study as the juveniles are abundant (Mumby 1999, Ruiz-Zarate & Arias-Gonzalez 2004) and there is no detectable interspecific difference in their diameter growth rate (Bak & Engel 1979, van Moorsel 1985, 1988). Colonies were included in the study if their estimated area was 125 mm^2 (mean 124.7 ± 6.0 mm, n = 63), they were not directly adjacent to other sessile invertebrates and were found on dead *Montastraea annularis* substrate in a location suitable for the positioning and attachment of a small cage. Each colony was tagged and its physical position and microhabitat recorded. Tagged corals were precisely mapped using a compass bearing and distance from a central, easily referenced point to facilitate relocation for repeat sampling.

Treatment protocols. Experimental manipulations were conducted in isolation from the mediating effect of grazing by the use of exclusion cages. Caging treatments standardised the environment of the juvenile corals, prevented grazing of the algal treatments and stopped the potential for random predation events from parrotfish or other external perturbations. Two types of control treatments were used: no-cage controls were used to control for the potential effects of caging on coral growth and caged controls to test the effect of algal treatments on coral growth. Including controls, the coral colonies were randomly allocated to 1 of 7 treatments.

Treatment 1 (no-cage controls, n = 13): Corals were left uncaged but all algae were cleared from their perimeter (as with other treatments).

Treatment 2 (cage controls, n = 8): A complete cage was positioned over the colony, with the same maintenance as for other treatments, but with no further manipulation.

Treatment 3 (Dictyota pulchella added, n = 8): *D. pulchella* was placed around the juvenile coral using an 'algal ring' method. Each ring was made from 3.0 g (wet weight) *D. pulchella*. This is the mean weight of *D. pulchella* clumps (mean 2.97 ± 0.44 g, n = 20) in a sampled circle the same diameter as the cage (10.5 cm). The

algae, collected from the same reef as the experiment, were placed in a nylon mesh tube shaped to form a circle; and secured using 0.5 mm nylon monofilament. The finished algal rings were maintained in sea water to allow the algae to settle out through the gaps in the mesh before placement in the cages. Algal rings were secured to the inside of the cages at 4 equidistant points using monofilament. This attachment prevented the nylon mesh from coming into contact with the coral but enabled the algae to grow out through the mesh and touch the colony. The algal ring method provides stability to the algae, allowing it to settle inside the cage without being dislodged by wave surge or stronger hydrodynamic forces. This method, however, did not perform as expected, since it was meant to simulate both shading and abrasion by *D. pulchella.* In reality, the algae did not overtop the coral but remained in contact with the periphery of the colony.

Treatment 4 (Dictyota pulchella shade, n = 8): Three grammes of *D. pulchella* were evenly distributed across the top of a cage and sandwiched between the 2 aligned layers of cage material that formed the lid. The algae could not touch the corals but shaded the area above the colony.

Treatment 5 (Dictyota pulchella mimic, n = 10): Synthetic 'mimic' algae were made to simulate the action of *D. pulchella* fronds growing around a coral colony. Mimic algae were made from ultra thin, clear polypropylene in a manner similar to that used by River & Edmunds (2001). The plastic was cut into narrow strips (3 × 50 mm, the approximate dimensions of *D. pulchella* blades: Littler & Littler 2000). Strips were tied into loose clumps with 0.5 mm monofilament to resemble clumps of *D. pulchella*. Four synthetic clumps were secured to the inside of the cage equidistant from each other and tied with monofilament. The fronds of the synthetic algae were long enough to contact the juvenile coral but not able to overtop it. Synthetic clumps could oscillate freely inside the cage in response to wave swell, pivoting around their secured point, mimicking the movement of actual *D. pulchella.*

Treatment 6 (Lobophora variegata added, n = 8): Large *L. variegata* thalli approximately 50 mm in diameter were collected from the experimental reef and laid inside the cage so as to cover the area surrounding the juvenile coral and touch its perimeter, but not to overtop the colony. One side of each thallus was secured under the edge of the cage to provide anchorage for settlement and to prevent the thalli moving freely within the cage. The close attachment of the cage to the substratum ensured that the thalli were effectively trapped in place.

Treatment 7 (Lobophora variegata shading, n = 8): *L. variegata* thalli were laid in 1 layer to completely cover the top of the cage. Thalli were laid so as to touch, but not overlap each other and were sandwiched securely between the 2 aligned layers of cage material forming the lid. The algae completely shaded the area above the colony but could not touch the coral.

No '*Lobophora variegata* mimic' treatment was included in the study as the physical movement of *L. variegata* thalli is limited by their shape and adherence to the substratum. Consequently it was thought that little insight could be gained from trying to mimic this limited movement, particularly when considering the *a priori* hypothesis that abrasion by *L. variegata* would have little effect on coral growth.

Cage design. The cages were cylindrical with a height of 4.5 cm and a diameter of 10.5 cm, constructed from 1.5 cm × 1.5 cm PVC mesh. The top of each cage had a double layer of aligned mesh secured with cable ties. The tops were hinged to allow access for photography and cage cleaning without having to remove the entire cage, and were securely closed with plastic hooks. Cages, by necessity, were small so that they could be easily fitted into the often confined locations where juvenile corals predominate. Cylindrical cages were used to reduce the possible caging artifact of flow reduction that could be exacerbated in the corners of square cages. The bottom of the cage had an extra 3 cm of mesh forming a skirt that was laid flush to the substratum. Cages were secured to the substratum using u-shaped stainless steel nails pinned around the skirt. Before use, all nails were covered by a plastic sheath of rubber tubing and sealed with silicone to prevent leaching of metal ions. The use of all plastic components in the cage mitigated potential contamination by metal ions, which can be both limiting nutrients and toxins in marine systems and could potentially affect algal and coral growth.

Treatment maintenance. All algal functional groups were removed from the periphery of the corals at the beginning of the experiment with the exception of crustose corallines whose removal might have damaged the structural integrity of the substratum. This was maintained throughout the investigation across all treatments. Cages were scrubbed 3 times weekly with wire brushes and scouring pads to remove epiphytes and other biota. The synthetic algae were wiped clean with the same frequency. All algae in the treatments were checked periodically during the cage maintenance process. *Dictyota pulchella* remained healthy for the duration of the study with no ill effects noted for either the algal ring manipulation or for algae secured in the lid mesh. Occasionally, in the *Lobophora variegata* shading treatment, thalli were torn by external forces; these were immediately replaced with new thalli.

Data collection and study duration. Corals were videoed approximately monthly (33 ± 0.69 d) using a Sony PCR120 digital video camera in an Ikelite hous-

ing. A 33 × 33 mm plastic quadrat was positioned in the same plane as the coral for scale. Footage was transferred to a computer and the area of live tissue measured using custom built software VidAna[1]. Polyp numbers per colony were also counted from video.

Both controls and *Dictyota pulchella* manipulations were carried out between October 2003 and November 2004. The synthetic algae manipulation was conducted between October 2003 and May 2004. *Lobophora variegata* treatments could not be manipulated during stormy periods due to the thalli being displaced from the cage. The timeframe of this treatment was reduced to the 6 mo between May and November 2004.

Photosynthetically active radiation (PAR) readings. Photosynthetic active radiation (PAR) measurements were made using a Macam Q102 digital radiometer attached to 2 waterproof 100 mm^2 silicon photodiode detectors. One detector recorded surface irradiance whilst the other was used for subsurface measurements. All measurements were made at the study site. Repeat readings (n = 16) were taken inside a standard cage attached to the substratum at 8 m with the detector placed in an equivalent position to a caged coral recruit. The reduction in PAR caused by shading of *Dictyota pulchella* and *Lobophora variegata* was measured by placing the detector under the relevant algal treatment (n = 10 for each treatment). To confirm the non-shading properties of the algal mimic treatment, measurements were also taken in this cage treatment (n = 10). To account for fluctuations in ambient PAR levels, all readings were paired, with an ambient PAR reading taken outside the cage immediately after each treatment measurement and with a reading taken at the surface. The proportional reduction in PAR due to treatment was calculated by dividing the PAR level of the treatment by the respective ambient reading.

Statistics. The relative change in area of juvenile corals was calculated as a percentage by the formula: $(\Delta A/A_i) \times 100$ where ΔA = the total change in surface area of the colony across the study duration and A_i = the initial colony size. This calculation reflected a loss of colony area as a negative value rather than a zero. Overall percentage change in polyp number was calculated in the same manner.

The data for percentage change in coral size and in polyp number were both normally distributed (Ryan-Joiner test for normalcy). Between treatments there was no statistically significant departure from homogeneity of variance and no outliers. These data therefore conformed to the assumptions of ANOVA without transformation. A 1-way ANOVA was used to test for differences between cage and no-cage control treatments for percentage change in area and for percentage change in polyp number.

To test the effect of algal treatment on overall percentage increase in colony size a 1-way ANOVA with 1-tailed Dunnett multiple comparisons with a control (Zar 1996) was used, with the expectation that treatments would have means less than control.

To investigate growth rate the area measurements for the juvenile corals were converted to geometric diameters, $\varnothing = 2 \times \sqrt{}$ (area ÷ π), where ∅ is the diameter and π is the mathematical constant pi. Growth of the colony was expressed as monthly diameter linear extension rate (mm mo^{-1}) (van Moorsel 1985, 1988, Lam 2000). Monthly growth rate was calculated by: $(\Delta\varnothing/\Delta t) \times 30$, where $\Delta\varnothing$ = the change in diameter between sampling and Δt = time in days between sampling.

The data for linear extension rate of monthly diameter were normally distributed for control treatments (Ryan-Joiner test for normalcy) and a repeated measures ANOVA was used to test the effect of treatment on the mean growth rate across the study duration between the no-cage control and the cage control.

In the absence of a cage, some corals disappeared from the no-cage controls. These events were characterised by the sudden vanishing of the entire colony and not the slow reduction in area that would be expected during spatial competition. These disappearances were probably due to whole colony predation events by parrotfishes, from which the caged treatments were protected. The loss of colonies in no-cage treatments was anticipated and a higher number of replicates were used for this treatment to allow for such losses. Corals in the no-cage control group that suffered whole colony predation events were omitted from growth comparisons between treatments since their demise was an external factor to which caged corals were not exposed. Colony mortality events in all treatments that followed a gradual decline in area were included in the growth analysis since their demise was likely to be a direct result of the treatment and not of external forces.

The number of periods during the study (defined as the time interval between samplings: mean = 33 ± 0.69 d), that the number of polyps in a colony increased, decreased or remained the same, were tallied. Differences between treatments were tested using chi-square but were converted to percentages for plotting.

The mortality rates (k) were calculated by dividing the number of whole colony mortality events by the number of corals in the treatment and then using the decay rate formula $k = \ln(1 - N) \div t$, where N = the proportion of mortalities; and t = the total elapsed time of the treatment in months. The standard error and the 95% confidence intervals for the no-cage mortality rate were calculated based on the binary probability distribution, with the assumption of independent mortality.

[1]The software is available free of charge from http://www.ex.ac.uk/msel

Table 1. Mean (±SE) photosynthetic active radiation (PAR) measurements for cage control and algal (*Dictyota pulchella* and *Lobophora variegata*) shading treatments (number of measurements in parentheses). Proportion: ambient irradiance remaining within each treatment. In algal treatments this is combined effect of the exclusion cage and the algae. Algal mimic: algal mimic treatment illustrating that this treatment did not affect PAR levels compared to cage control

Irradiance ($\mu E\ m^{-2}\ s^{-1}$)	Cage control (16)		*Dictyota* shading (10)		*Lobophora* shading (10)		Algal mimic (10)	
Surface	908.8	39.8	822.2	37.9	759.8	32.4	936.7	49.5
Ambient at 8 m (A)	213.9	20.8	208.9	8.3	175.2	2.7	235.5	7.28
Treatment (T)	115.8	9.4	54.6	7.8	24.5	1.4	132.2	1.74
Proportion (T ÷ A)	0.56	0.02	0.25	0.03	0.14	0.01	0.57	0.02

RESULTS

Control corals, caging effects and growth rate trends

The cages reduced photosynthetically active radiation (PAR) to 56% of ambient irradiance at 8 m depth (Table 1). However this had no detectable effect on the growth of juvenile corals between the 2 control groups (cage and no-cage) in this study. No significant differences were found between juvenile corals growing in the cage control treatment compared to the no-cage control for the 3 different growth parameters measured (Fig. 1, Tables 2 & 3); nor was there a statistically significant difference between the proportion of periods where polyp number increased, decreased or remained the same (chi-square $p > 0.05$) (Fig. 2).

Corals in the control treatments exhibited linear growth in diameter ($R^2 = 0.99$) with the number of polyps in a colony exhibiting an exponential growth pattern over time ($R^2 = 0.99$) (Fig. 3). The mean colony growth (measured as diameter linear extension per month), pooled between controls (n = 16) across all time intervals in the study (t = 14 mo), was 0.86 ± 0.07 mm mo^{-1} (mean ± SE).

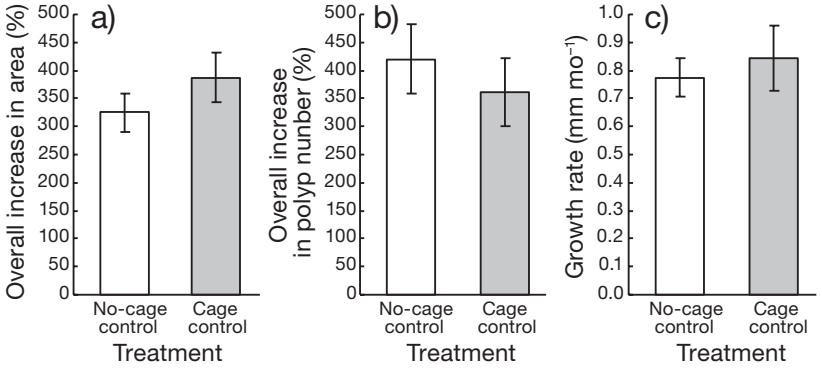

Fig. 1. Comparison of juvenile corals in cage control with no-cage control treatments using 3 different parameters for growth. (a) Overall increase in colony area as percentage increase of initial size; (b) overall increase in number of polyps per colony as percentage increase in initial number; (c) monthly growth rate as linear diameter extension. All data are means (±1 SE, n = 8)

Effects of *Dictyota pulchella* on juvenile coral growth

The *Dictyota pulchella* shade treatment reduced PAR to 25% of ambient irradiance; this was the combined effect of the exclusion cage and the algae (Table 1). This had a highly significant effect on the overall growth of *Agaricia* spp. (Fig. 4, Table 4). The mean monthly growth rate was reduced to 0.01 mm mo^{-1}, i.e. less than 1% of the growth rate of corals in the cage control (0.84 mm mo^{-1}) (Table 2). The *D. pulchella* shade treatment effectively retarded the corals' further expansion. Direct contact with *D. pulchella* also had a highly significant effect on the overall growth of juvenile corals (Fig. 4, Table 4). The growth rate was limited to 31% of that of the cage controls (Table 2). Contact with the algal mimic caused a highly significant reduction in overall growth and reduced the growth rate to 41% that of the cage controls. The overall growth of corals exposed to the algal mimic was not significantly different from the growth of the *D. pulchella* added treatment (Fig. 4, Tables 2 & 4). The algal mimic did not affect PAR levels within the cage compared to cage controls (Table 1), so the effects on growth rate are not attributable to light inhibition.

All 3 *Dictyota pulchella* treatments significantly altered the percentage of periods in which the number of polyps in a colony increased, decreased or remained the same, compared to cage control corals over the same study period (chi-square $p < 0.01$ for *D. pulchella* shade and *D. pulchella* added; $p = 0.02$ for algal mimic). Each of the *D. pulchella* treatments increased the proportion of periods in which the number of polyps in the colony did not change (Fig. 2).

Table 2. Growth and mortality of *Agaricia* spp. in each treatment compared to control values for the same study period. Mean growth rate is mean linear diameter expansion in mm mo^{-1}, relative growth rate (Rel. rate) for algal treatments is proportion of respective cage control growth rate. Mortality: total colony mortality; n: number of total colony mortalities; Time: time elapsed from beginning of study; Mortality rate: monthly mortality rate; *significant differences in mortality compared to cage control (chi-square, $p < 0.05$) (duplicate data for control treatments have been omitted for clarity)

Treatment	Growth rate (mm mo^{-1}) Mean	SE	Rel. rate	Mortality	n	Time (mo)	Mortality rate (k)
No cage	0.78	0.07		5*	13	14	0.035
Cage control	0.84	0.12		0	8	14	0
Dictyota pulchella added	0.26	0.08	0.31	0	8	14	0
Dictyota pulchella shade	0.01	0.26	0.01	2	8	14	0.021
No cage	0.85	0.10			13		
Cage control	0.78	0.22			8		
Lobophora variegata added	0.47	0.09	0.60	0	8	6	0
Lobophora variegata shade	−1.06	0.48	−1.36	4*	8	6	0.116
No cage	0.71	0.10			13		
Cage control	0.89	0.11			8		
Algal mimic	0.37	0.06	0.42	0	10	10	0

Table 3. Results of ANOVAs between no-cage and cage control corals for 3 growth parameters for *Agaricia* spp.: (a) 1-way ANOVA of percentage increase in overall colony size; (b) 1-way ANOVA of percentage increase in overall number of polyps per colony; (c) repeated-measures ANOVA of differences in monthly linear extension rate of diameter

Source	df	SS	MS	*F*	p
(a) Percentage increase in size					
Treatment	1	15 939	15 939	1.30	0.274
Error	14	172 180	12 299		
Total	15	188 119			
(b) Percentage increase in polyp number					
Treatment	1	14 340	14 340	0.47	0.503
Error	14	424 949	30 354		
Total	15	439 289			
(c) Monthly growth rate					
Factor A	1	0.242	0.242	0.211	0.647
Factor S	103	79.540	0.772		
A × S	103	118.225	1.148		
Total	207	198.008			

Effects of *Lobophora variegata* on juvenile coral growth

The *Lobophora variegata* shade treatment reduced ambient PAR to 14%, as a combined effect of the exclusion cage and algal shading (Table 1). This treatment had a highly significant effect on the overall growth of *Agaricia* spp. (Table 4, Fig. 4), causing the most pronounced effect on coral growth of all treatments and resulting in a net loss of colony tissue area over time (Table 2).

The number of periods in which polyp numbers increased was half that of cage controls, and the number of periods that exhibited a loss in polyp number was 6 times higher. This was a highly significant result (chi-square, $p < 0.01$) (Fig. 2).

Contact with *Lobophora variegata* around the periphery of a colony also had a significant effect on the overall growth of *Agaricia* spp. (Table 4). However,

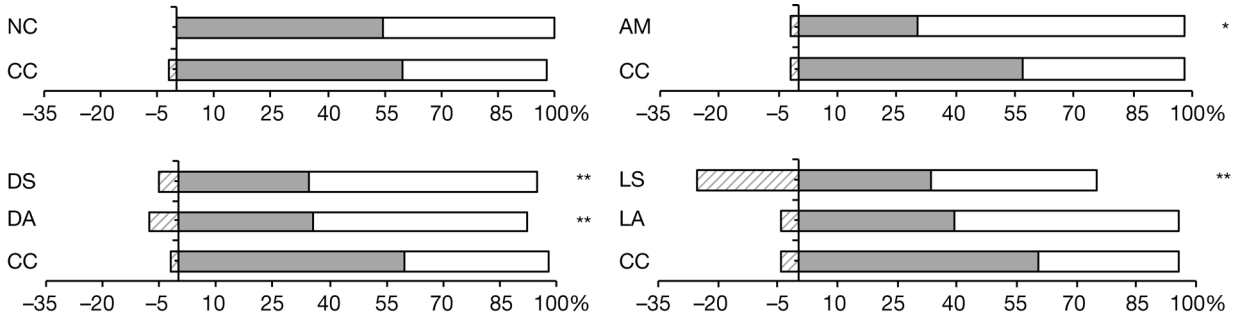

Fig. 2. Percentage of sampling periods during which number of polyps within a colony of *Agaricia* spp. increased (grey), decreased (hatched) or did not change (white) for each treatment. Treatments are compared to cage control for the same study period. **Highly statistically significant differences between treatments and cage control (chi-square < 0.01); *statistically significant differences (chi-square < 0.05). CC: cage control; NC: no-cage cage control; AM: algal mimic; DS: *Dictyota pulchella* shade; DA: *D. pulchella* added; LS: *Lobophora variegata* shade; LA: *L. variegata* added

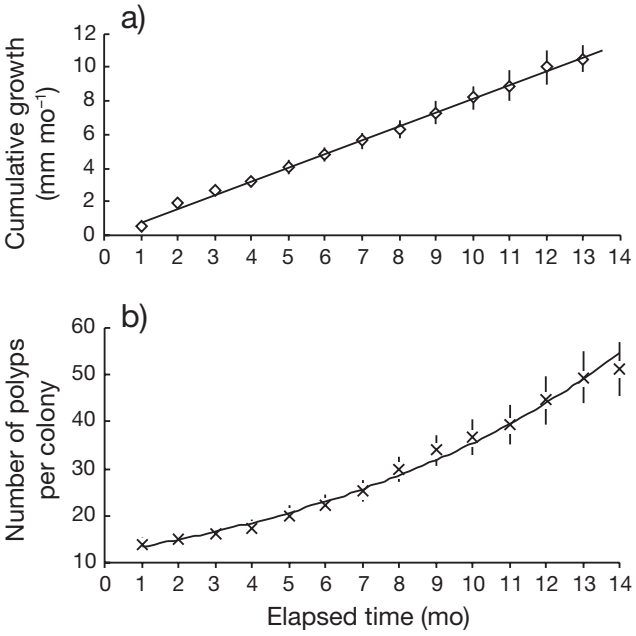

Fig. 3. Growth rates of juvenile *Agaricia* spp. in control treatments plotted against time in months from beginning of study for (a) colony diameter, (b) number of polyps per colony. Data points are monthly means (±1 SE, n = 16) pooled between controls. Trend lines illustrate linear growth pattern in colony diameter (R^2 = 0.99) and exponential growth pattern in number of polyps per colony (R^2 = 0.99)

the degree to which *L. variegata* growth around a colony reduced growth rates was less than for other treatments (Table 2). *L. variegata* did not significantly affect the percentage of periods in which the number of polyps in a colony increased, decreased or remained the same (Table 4; Figs. 2 & 4).

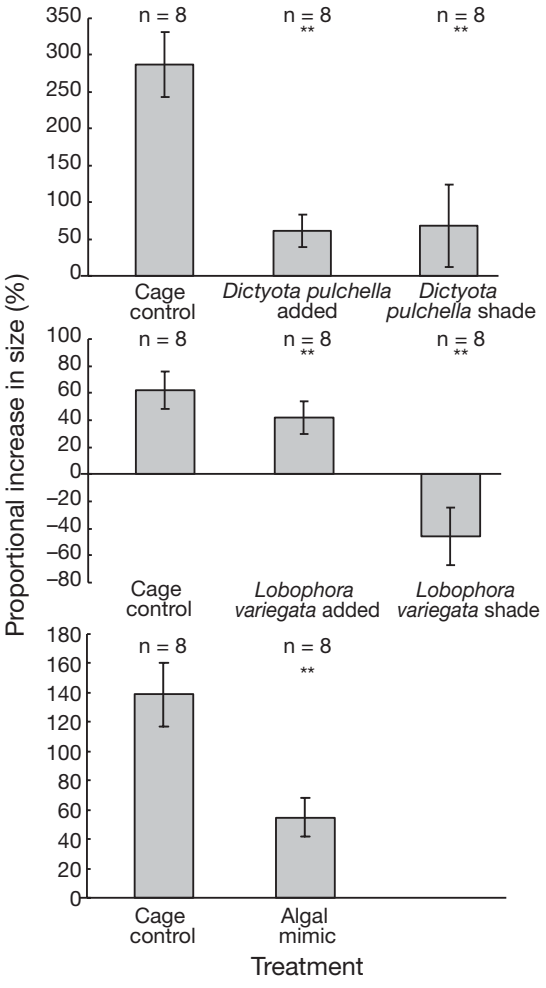

Fig. 4. Overall change (mean ± 1 SE) in size (percentage of initial size) of juvenile *Agaricia* spp. colonies for all treatments compared to cage control over the same study period. **Highly statistically significant differences between algal treatment and cage control ($p < 0.01$)

Effect of macroalgae on mortality

Only the *Lobophora variegata* shade treatment caused a significant increase in mortality compared to the cage control (chi-square, $p < 0.05$; Table 2). However, there was a statistically significant difference in whole colony mortality between caged and no-cage control groups (chi-square, $p < 0.05$). The 5 whole colony mortalities from 13 individuals for no-cage controls represent a monthly mortality rate of 0.035 ± 0.135 (SE) (Table 2). If whole colony mortality rate is assumed to be constant until juvenile corals reach some refuge in size, such as growing beyond the gape size of parrotfish, then the proportion of

Table 4. Results of 1-way ANOVA (1-tailed Dunnett multiple comparisons with control) testing overall proportional growth of juvenile corals in algal treatments compared to cage control over the relevant time scales. Seq: sequential; Adj: adjusted; Diff: difference

Source	df	Seq SS	Adj SS	Adj MS	*F*	p
Treatment	5	312 829	312 829	62 566	6.15	0.000
Error	60	610 123	610 123	10 169		
Total	65	922 952				
Level treatment		Diff. of means	SE of Diff.	*t*		Adj. p-value
D. pulchella added		−102.1	41.17	−2.480		$p < 0.0001$
D. pulchella shade		−94.5	41.17	−2.296		$p < 0.0001$
D. pulchella mimic		−108.1	37.95	−2.848		$p < 0.0001$
L. variegata added		−120.8	41.17	−2.935		$p < 0.0001$
L. variegata shade		−208.7	41.17	−5.068		$p < 0.0001$

the juvenile population that survives over time can be calculated using an exponential decay function. With a mortality rate of 0.035 the decay function for percentage survival over time would be $S = 100 \times e^{t\ (-0.035)}$, where S is the remaining percentage of the population, and t is time in months.

A coral growing at the normal (algal-free) growth rate would take approximately 36 mo to reach an arbitrary size refuge of 3 cm diameter. With a monthly mortality rate of 0.035, 29% of a cohort would survive this 36 mo period (Fig. 5). In contrast, coral surrounded by *Lobophora variegata* and growing at an inhibited rate would take 63 mo to reach the same diameter of 3 cm, whilst corals surrounded by *Dictyota pulchella* would take 115 mo. Growth rate inhibition increases the exposure time of recruits to size dependent mortality events affecting overall cohort survival. Rather than 29% of the population surviving to a size of 3 cm, growth inhibition could reduce the cohort survival of corals surrounded by *L. variegata* to 11% and those surrounded by *D. pulchella* to less than 2% (Fig. 5).

The upper and lower 95% confidence intervals for the monthly mortality rate of 0.035 were calculated as 0.009 and 0.075. At the lower 95% confidence interval, 73% of a population would survive to 3 cm diameter. This survival proportion was reduced to 56 and 35% for corals growing at inhibited growth rates due to the proximity of *Lobophora variegata* and *Dictyota pulchella*, respectively. At the upper 95% confidence interval only 7% of the population would survive to the 36 mo required to reach 3 cm. This is reduced to 0.8 and 0.02% for corals growing at the inhibited rates caused by *L. variegata* and *D. pulchella* (Fig. 5).

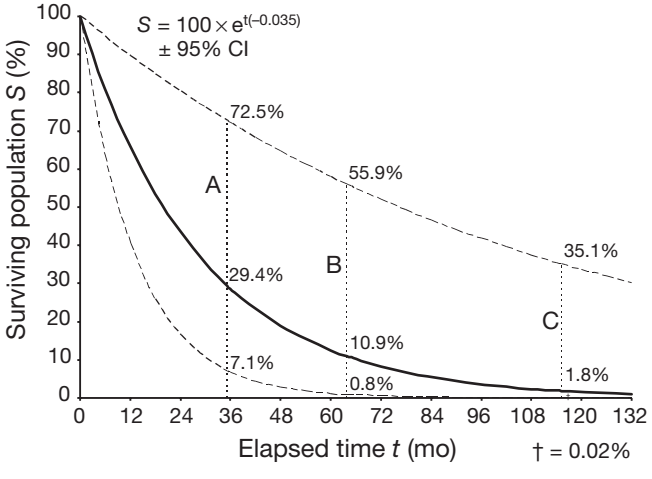

Fig. 5. Exponential decay function for population of juvenile corals plotted against time showing measured colony mortality rate from no-cage controls of 0.035 mo^{-1} (continuous line) with upper and lower confidence intervals (0.075 and 0.009, respectively; dashed lines). Vertical dotted lines: percentage of population surviving to size-refuge of 3 cm diameter following 36 mo growth at (Line A) normal rate, (Line B) inhibited growth rate due to proximity of *Lobophora variegata*, and (Line C) inhibited growth rate due to proximity of *Dictyota pulchella*. Percentages inside graphs: proportions of population surviving up to indicated time points at the respective mortality rates

DISCUSSION

Rapid growth during early life history stages is essential, because corals suffer size-dependent mortality rates (Edmunds & Gates 2004) with juvenile corals experiencing high levels of mortality (Bak & Engel 1979, Edmunds 2000). Small colonies are particularly vulnerable to physical breakage and tend to fare worse in spatial competition with neighbours (Zilberberg & Edmunds 2001). The recorded mean linear growth for juvenile *Agaricia* spp. in this study was similar to rates reported previously for Agaricidae (Bak & Engel 1979, van Moorsel 1988, Vermeij 2006) and our observations of linear growth rates in diameter extension are consistent with those of other studies on juvenile corals (van Moorsel 1985, Lam 2000). Here, we found that shading and abrasive actions of algae on juvenile *Agaricia* spp. can severely limit coral growth rates and cause elevated mortality. The nature and intensity of the impacts varied between algal species.

Our manipulations of shading and abrasion differed in their realism. Placing algae around the periphery of experimental coral colonies appeared to be a fair representation of reality as such interactions occur frequently on many Caribbean reefs. Our manipulation of shading, however, is likely to underestimate the full impact of algal overgrowth since full shading occurs when algae overtop the coral and therefore shading stress also involves extensive abrasion. Our experiments removed this contributory abrasive stress, by placing the algal shade several centimetres above the coral colony. Conversely, our shading manipulation may also overestimate the intensity of the shading affect as the algae were maintained in stasis 4 cm above the coral, whereas in reality algal fronds would move from side to side and cause fluctuations in light penetration.

Despite these limitations, shading effects by both algal species were still found to exert significant deleterious impacts on coral growth and survival. Whilst we cannot rule out that shade in combination with reduced water movement was responsible for the observed mortality, or reductions in overall growth, the random orientation of the caged corals (often in cryptic, sheltered habitats) is likely to have reduced any systematic bias that might occur in a strongly directional current.

In algal shading treatments the combined effect of shading by the algae and the exclusion cage reduced the penetration of PAR reaching coral tissues to a greater extent that the exclusion cage alone. Reductions in PAR below a threshold level result in a reduction in photosynthetic production within a coral colony (Chalker et al. 1983). The results of our study suggest that the *Dictyota pulchella* shade treatment reduced the growth rate to just 1% of caged corals, with PAR being reduced to 25% of ambient. The *Lobophora variegata* shade treatment reduced ambient PAR to 14% and had a more severe effect on coral colonies, causing not just a reduction in growth rate but also overall shrinkage of live tissue area as polyps died. The thicker thallus of this alga and the overlapping nature of its thalli are the likely causes of the greater PAR limitation and growth inhibition caused by this alga compared to *D. pulchella*. While we cannot discount the possibility that mounting an algal canopy several centimetres above the coral had little influence on local water flow across the coral, our results clearly suggest that reductions in PAR can severely impair the growth rate of juvenile corals.

Algal proximity may influence coral growth rates through a variety of mechanisms including (1) increased localised sedimentation which can decrease coral growth rates (Rogers 1990), (2) reduced encounter rates with food particles (Sebens 1997), (3) decreased uptake of dissolved nutrients (Atkinson & Bilger 1992), (4) reduced efficiency of oxygen exchange (Finelli et al. 2006), (5) enhanced microbial activity that is detrimental to coral health (Smith et al. 2006), (6) reduced coral growth through allelochemical inhibition (Littler & Littler 1997), and (7) reduced coral growth through abrasion-mediated polyp retraction (River & Edmunds 2001). Our results suggest that a close proximity of *Dictyota pulchella* has a marked deleterious impact on coral growth rate (although not as severe as that of shading). The reduction in growth rate was similar using both real and mimic *D. pulchella,* suggesting that it is likely to be caused by abrasion or other direct physical mechanisms rather than an additional factor innate to living *D. pulchella*, such as allelochemical or algal–microbial inhibition (Smith et al. 2006). However, further studies of allelochemical interactions involving these algae and scleractinian corals are needed. The presence of *Lobophora variegata* around colonies did have a detectable effect on the growth rate of corals, but to a lesser extent than *D. pulchella*. This result is compatible with the proposed impact of abrasion on corals, as a branching alga such as *D. pulchella* has more fronds, which oscillate to a greater extent, than the broad, fan shaped thalli of *L. variegata*. The proximity of *L. variegata* to the perimeter of a colony would also possibly enable it to subsequently overtop and shade corals or creep across their surface, causing their mortality (Tanner 1995, Jompa & McCook 2002, Nugues & Bak 2006).

Juvenile corals experience high levels of mortality (Bak & Engel 1979, Edmunds 2000) which may be an important factor structuring coral reef communities (Miller et al. 2000). In the present study, cages protected corals from grazing (predation), with predation events in uncaged corals assumed to be caused by the scarid *Sparisoma viride* (Bruggemann et al. 1994). Our inference of reduced grazing is consistent with the observation of Sato (1985) that recruits survive better when not exposed to direct grazing pressure. Whether such predation events comprise an active prey selection by *S. viride* or an inadvertent consequence of grazing requires further clarification.

In the absence of grazing, shading by *Lobophora variegata* was lethal to juvenile *Agaricia* spp. However, impacts of this alga on corals are varied. Firstly, van Steveninck et al. (1988) found that contact with corals reduced the growth rate of *L. variegata* by 35%, and therefore competitive interactions occur in both directions. Several studies (van Steveninck et al. 1988, Jompa & McCook 2002) concluded that *L. variegata* is a superior competitor to both Caribbean corals (*Agaricia agaricites*, *A. lamarcki*, *Meandrina meandrites*, *Mycetophyllia aliciae* and *Stephanocoenia michelinii*) and Pacific corals (*Porites cylindrica*) in the absence of grazing. Whilst these results are consistent with our own, recent studies by Nugues & Bak (2006) revealed that adults of several Caribbean coral species are competitively superior to *Lobophora* spp. A notable exception was *A. agaricites,* which was found to be relatively rapidly overgrown by *Lobophora* spp. Thus it is feasible that our results present a 'worst case' scenario for coral-algal overgrowth, given that the genus studied (*Agaricia*) may be particularly susceptible to overgrowth. Whilst Nugues & Bak (2006) called for a species-specific examination of coral-algae competition, we extend their position to include an analysis by coral size.

Unlike shading by *Lobophora variegata*, *Dictyota pulchella* was not found to cause direct mortality of juvenile coral. We concede, however, that the power of this test was low (2 dead corals from a total of 8). Future studies with a larger sample size may in fact detect a weak, although statistically significant direct impact on mortality by this alga. However, the reduction in coral growth rate brought about by the proximity of either *D. pulchella* or *L. variegata* could have profound effects on the predator-caused rates of mortality in juvenile corals. Whilst this study used a relatively small sample size to calculate monthly mortality rate, the estimated rate of 0.035 mo^{-1} is similar to the rates

reported for Agaricidae by other studies (Edmunds 2000) of 50% yr^{-1} which is equivalent to 0.058 mo^{-1} and is within our calculated 95% confidence intervals. Irrespective of the precise predator-caused mortality rate, the influence of growth inhibition on the relative probability of juvenile coral survival is clearly important. Even at the lowest mortality rate (lower 95% confidence interval) calculated in this study the survival probability of a coral reaching 3 cm diameter was reduced from 73% in the absence of algal competition, to 56 and 35% in the presence of *L. variegata* and *D. pulchella*, respectively. Consequently, this mechanism of interference competition may be an important contributory factor to the apparent persistence of macroalgal blooms on some reefs, without requiring algae to cause direct mortality in corals.

Our results contribute to a wider understanding of the impact of macroalgae on coral population dynamics. Macroalgae affect coral recruitment by pre-empting settlement space (Birrell et al. 2005) and increasing the mortality rate of settled corals (by both the direct and indirect mechanisms described above). Our results help elucidate the outcome of coral–algal interactions but these results need to be interpreted in the light of the patch dynamics of macroalgae which determine the frequency and duration of coral–algal interactions. The simple survivorship calculations we present here in implicitly assume that coral growth rate is retarded permanently for a fixed period of time. In reality, growth retardation may not be continuous because of fluctuations in algal cover (Mumby et al. 2005). Indeed it is the outcome of algal dynamics that partly determine the net growth rate of juvenile corals, with more persistent algal contact having a larger impact on coral growth than short ephemeral contact. Thus the implications of our plot of mortality are simply that algal dynamics will strongly dictate the position on the *x*-axis at which corals become large enough to escape the risk of predation.

Unfortunately, data on algal patch dynamics are limited. Working in Belize, Mumby et al. (2005) concluded that the annual turnover of *Lobophora variegata* and *Dictyota pulchella* patches was 12 to 60 and 7 to 20% yr^{-1}, respectively. Whilst the natural persistence of *L. variegata* patches is shorter than that of *D. pulchella*, the results of the current study show that this alga is faster at exerting a detrimental effect on juvenile corals (within 6 mo). Additionally, despite having a slower rate of effect on juvenile corals, the increased persistence of *D. pulchella* patches means that it is liable to be in contact with, or shade, juvenile corals for sufficient time to exert an inhibitory effect. Therefore, both species of algae present a viable competitive threat to juvenile coral survival within relevant temporal and spatial scales.

Acknowledgements. We thank the Darwin Initiative and Peter Raines (Coral Cay Conservation) for supporting this research through a grant and PhD studentship to S.J.B. Thanks to Pura Vida, Roatán, for logistical support and the staff and volunteers of the Roatán CCC base for their generous assistance in the field.

LITERATURE CITED

Aronson RB, MacIntyre IG, Wapnick CM, O'Neill MW (2004) Phase shifts, alternative states, and the unprecedented convergence of 2 reef systems. Ecology 85:1876–1891

Atkinson MJ, Bilger RW (1992) Effects of water velocity on phosphate uptake in coral reef-flat communities. Limnol Oceanogr 37:273–279

Bak RPM, Engel MS (1979) Distribution, abundance and survival of juvenile hermatypic corals (Scleractinia) and the importance of life history strategies in the parent coral community. Mar Biol 54:341–352

Bak RP(M), Nieuwland G, Meesters EH (2005) Coral reef crisis in deep and shallow reefs: 30 years of constancy and change in reefs of Curacao and Bonaire. Coral Reefs 24: 475–479

Birrell CL, McCook LJ, Willis BL (2005) Effects of algal turfs and sediment on coral settlement. Mar Pollut Bull 51: 408–414

Bruggemann JH, van Oppen MJH, Breeman AM (1994) Foraging by the stoplight parrotfish *Sparisoma viride*. I. Food selection in different socially determined habitats. Mar Ecol Prog Ser 106:41–55

Carpenter RC, Edmunds PJ (2006) Local and regional scale recovery of *Diadema* promotes recruitment of scleractinian corals. Ecol Lett 9:268–277

Chalker BE, Dunlap WC, Oliver JK (1983) Bathymetric adaptations of reef-building corals at Davies Reef, Great Barrier-Reef, Australia. 2. Light Saturation curves for photosynthesis and respiration. J Exp Mar Biol Ecol 73:37–56

Done TJ (1992) Phase shifts in coral reef communities and their ecological significance. Hydrobiologia 247:121–132

Edmunds PJ (2000) Patterns in the distribution of juvenile corals and coral reef community structure in St. John, US Virgin Islands. Mar Ecol Prog Ser 202:113–124

Edmunds PJ, Gates RD (2004) Size-dependent differences in the photophysiology of the reef coral *Porites astreoides*. Biol Bull (Woods Hole) 206:61–64

Finelli CM, Helmuth BST, Pentcheff ND, Wethey DS (2006) Water flow influences oxygen transport and photosynthetic efficiency in corals. Coral Reefs 25:47–57

Harborne AR, Afzal DC, Andrews MJ (2001) Honduras: Caribbean coast. Mar Pollut Bull 42:1221–1235

Hawkins JP, Roberts CM (2004) Effects of fishing on sex-changing Caribbean parrotfishes. Biol Conserv 115:213–226

Hughes TP (1994) Catastrophes, phase shifts, and large-scale degradation of a Caribbean coral reef. Science 265: 1547–1551

Hughes TP, Tanner JE (2000) Recruitment failure, life histories, and long-term decline of Caribbean corals. Ecology 81: 2250–2263

Hughes TP, Baird AH, Bellwood DR, Card M and 13 others (2003) Climate change, human impacts, and the resilience of coral reefs. Science 301:929–933

Jompa J, McCook LJ (2002) Effects of competition and herbivory on interactions between a hard coral and a brown alga. J Exp Mar Biol Ecol 271:25–39

Lam KKY (2000) Early growth of a pioneer recruited coral *Oulastrea crispata* (Scleractinia, Faviidae) on PFA-con-

crete blocks in a marine park in Hong Kong, China. Mar Ecol Prog Ser 205:113–121
Lirman D (2001) Competition between macroalgae and corals: effects of herbivore exclusion and increased algal biomass on coral survivorship and growth. Coral Reefs 19:392–399
Littler DS, Littler MM (1997) Epizoic red alga allelopathic to a Caribbean coral. Coral Reefs 16:168
Littler DS, Littler MM (2000) Caribbean reef plants. Offshore Graphics, Washington, DC
McClanahan TR, Hendrick V, Rodrigues MJ, Polunin NVC (1999) Varying responses of herbivorous and invertebrate-feeding fish to macroalgal reduction on a coral reef. Coral Reefs 18:195–203
Miller MW, Weil E, Szmant AM (2000) Coral recruitment and juvenile mortality as structuring factors for reef benthic communities in Biscayne National Park, USA. Coral Reefs 19:115–123
Mumby PJ (1999) Bleaching and hurricane disturbances to populations of coral recruits in Belize. Mar Ecol Prog Ser 190:27–35
Mumby PJ, Foster N, Fahy E (2005) Patch dynamics of coral reef macroalgae under chronic and acute disturbance. Coral Reefs 24:681–692
Nugues M, Bak RP (2006) Differential competitive abilities between Caribbean coral species and a brown alga: a year of experiments and a long-term perspective. Mar Ecol Prog Ser 315:75–86
Nugues MM, Roberts CM (2003) Coral mortality and interaction with algae in relation to sedimentation. Coral Reefs 22: 507–516
Nugues MM, Smith GW, Hooidonk RJ, Seabra MI, Bak RPM (2004) Algal contact as a trigger for coral disease. Ecol Lett 7:919–923
Raymundo LJ, Maypa AP (2003) Getting bigger faster: mediation of size-specific mortality via fusion in juvenile coral transplants. Ecol Appl 14:281–295
River GF, Edmunds PJ (2001) Mechanisms of interaction between macroalgae and scleractinians on a coral reef in Jamaica. J Exp Mar Bilo Ecol 261:159–172
Rogers CS (1990) Responses of coral reefs and reef organisms to sedimentation. Mar Ecol Prog Ser 62:185–205
Ruiz-Zarate MA, Arias-Gonzalez JE (2004) Spatial study of juvenile corals in the Northern region of the Mesoamerican Barrier Reef System (MBRS). Coral Reefs 23: 584–594
Sato M (1985) Mortality and growth of juvenile coral *Pocillopora damicornis* (Linnaeus). Coral Reefs 4:27–33
Sebens KP (1997) Adaptive responses to water flow: morphology, energetics and distribution of reef corals. Proc 8th Int Coral Reef Symp 2:1053–1058
Smith JE, Shaw M, Edwards DO, Pantos O and 5 others (2006) Indirect effects of algae on coral: algae-mediated, microbe-induced coral mortality. Ecol Lett 9:835–845
Tanner JE (1995) Competition between scleractinian corals and macroalgae: an experimental investigation of coral growth, survival and reproduction. J Exp Mar Biol Ecol 190:151–168
van Moorsel GWNM (1985) Disturbance and growth of juvenile corals (*Agaricia humilis* and *Agaricia agaricites* Scleractinia) in natural habitats on the reefs of Curacao. Mar Ecol Prog Ser 24:99–112
van Moorsel GWNM (1988) Early maximum growth of stony corals (Scleractinia) after settlement on artificial substrata on Caribbean reef. Mar Ecol Prog Ser 50:127–135
van Steveninck EDD, van Mulekom LL, Breeman AM (1988) Growth inhibition of *Lobophora variegata* by scleractinian corals. J Exp Mar Biol Ecol 115:169–178
Vermeij MJA (2006) Early life-history dynamics of Caribbean coral species on artificial substratum: the importance of competition, growth and variation in life-history strategy. Coral Reefs 25:59–71
Zar JH (1996) Biostatistical analysis, 3rd edn. Prentice Hall, Upper Saddle River, NJ
Zilberberg C, Edmunds PJ (2001) Competition among small colonies of *Agaricia*: the importance of size assymetry in determining competitive outcome. Mar Ecol Prog Ser 221: 125–133

Editorial responsibility: Otto Kinne (Editor-in-Chief), Oldendorf/Luhe, Germany

Submitted: July 6, 2006; Accepted: December 18, 2006
Proofs received from author(s): June 20, 2007