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REPORT

High CO_2 detrimentally affects tissue regeneration of Red Sea corals

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Abstract Ocean acidification (OA) from rising atmospheric carbon dioxide (CO_2) is threatening the future of coral reef ecosystems. Mounting experimental evidence suggests that OA negatively impacts fundamental life functions of scleractinian corals, including growth and sexual reproduction. Although regeneration is regarded as a chief life function in scleractinian corals and essential to maintain the colony's integrity, the effect of OA on regeneration processes has not yet been investigated. To evaluate the effects of OA on regeneration, the common Indo-Pacific corals *Porites* sp., *Favia favus*, *Acropora eurystoma*, and *Stylophora pistillata* were inflicted with lesions (314–350 mm^2, depending on species) and incubated in different pCO_2: (1) ambient seawater (400 µatm, pH 8.1), (2) intermediate (1,800 µatm, pH 7.6), and (3) high (4,000 µatm, pH 7.3) for extended periods of time (60–120 d). While all coral species after 60 d had significantly higher tissue regeneration in ambient conditions as compared to the intermediate and high treatments, reduction in regeneration rate was more pronounced in the slow-growing massive *Porites* sp. and *F. favus* than the relatively fast-growing, branching *S. pistillata* and *A. eurystoma*. This coincided with reduced tissue biomass of *Porites* sp., *F. favus*, and *A. eurystoma* in higher pCO_2, but not in *S. pistillata*. *Porites* sp., *F. favus*, and *S. pistillata* also experienced a decrease in *Symbiodinium* density in higher pCO_2, while in *A. eurystoma* there was no change. We hypothesize that a lowered regenerative capacity under elevated pCO_2 may be related to resource trade-offs, energy cost of acid/base regulation, and/or decrease in total energy budget. This is the first study to demonstrate that elevated pCO_2 could have a compounding influence on coral regeneration following injury, potentially affecting the capacity of reef corals to recover following physical disturbance.

Keywords High CO_2 · Ocean acidification · Regeneration · Global change · Red Sea

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Introduction

Increasing carbon dioxide (CO_2) emissions drive ongoing ocean acidification (OA) and place reef-building corals in a vulnerable state (Hoegh-Guldberg 2012). Atmospheric CO_2 levels have risen at an accelerating rate during the industrial era, approximately 100 times faster than over the past 650,000 years, as a direct consequence of burning of fossil fuels and deforestation (Royal Society 2005). CO_2 absorbed by the oceans is changing seawater carbonate chemistry by making the oceans more acidic, thereby driving a shift toward lower carbonate ion concentration ($[CO_3^{2-}]$), aragonite saturation state (Ω_{arag}), and pH (Kleypas et al. 1999; Zeebe and Wolf-Gladrow 2001). Atmospheric CO_2 has recently reached 400 ppm, the highest level in recorded history (Monastersky 2013). Predictions warn of a decrease of 0.3–0.5 pH units in oceanic surface water by the end of this century (IPCC 2007).

Scleractinian corals, the framework builders of coral reefs, are extremely sensitive to small changes in their

immediate environment. Such changes may result in excursions from their optimal environmental conditions (Kleypas and Hoegh-Guldberg 2008). The coral's engineering role is particularly susceptible to global climate change (Madin et al. 2012). Mounting experimental evidence indicates that reductions in surface-ocean pH and carbonate saturation state could have major effects on scleractinian corals (Hoegh-Guldberg et al. 2007; Kroeker et al. 2010; Anthony et al. 2011; Erez et al. 2011). Key life functions in corals such as growth and reproduction can be affected by high pCO_2/low pH (Kleypas and Langdon 2006; Albright and Langdon 2011), but until now, the effects on the ability of corals to recover from damage (regeneration) have not yet been investigated.

Damage or partial mortality in scleractinian corals originates from both natural causes such as predation, competition, disease, and physical disturbances, as well as human activities including fishing, diving, boating, mining, and coastal development (reviewed by Grigg and Dollar 1990). Regeneration is a life-preserving process in scleractinian corals and essential in preserving the integrity of the colony (Henry and Hart 2005). Lesion recovery depends on accelerated growth rates (D'Angelo et al. 2012), a process requiring both energy and tissue reserves (Oren et al. 1997, 2001; Fine et al. 2002). For most coral species, the rapidity of this process is vital, since fast lesion healing restores the functional use of polyps (photosynthesis, defense, reproduction, feeding, and storage; Hughes and Jackson 1985; Sebens 1987; D'Angelo et al. 2012) and minimizes the risk of invasion by competitors, pathogens, and bioeroders (Jackson and Palumbi 1979; Hughes and Jackson 1985; Titlyanov et al. 2005; Titlyanov and Titlyanova 2008). As a general rule, regeneration is believed to have the highest priority among life functions since natural selection should favor regenerative processes above other requirements (Karlson 1988). This has been demonstrated by several studies on corals reporting resource trade-offs, with injury causing a reduction in fecundity (Rinkevich and Loya 1989; Van Veghel and Bak 1994; Rinkevich 1996) and growth (Meesters et al. 1994; but see Denis et al. 2013).

Indeed, many studies show how regenerative capacity of scleractinian corals is affected by the prevailing biotic and abiotic conditions (i.e., local environment; Lester and Bak 1985; Fisher et al. 2007; Denis et al. 2011). Seasonal changes in regeneration capacity associated with different temperature and radiation regimes have been reported in several studies (Kramarsky-Winter and Loya 2000; Fisher et al. 2007; Denis et al. 2011). Exposure to abnormally high water temperatures, however, and subsequent bleaching due to expulsion of algal endosymbionts may reduce or completely prevent regeneration (Meesters and Bak 1993; Meesters et al. 1997a; Fine et al. 2002). Similar reduction in regeneration rates has been observed in habitats with high sedimentation levels due to shading of the endosymbionts and the high cost of sediment rejection (Meesters et al. 1992). Fisher et al. (2007) observed slower regeneration in deep-water corals compared to shallow water corals, most likely explained by less radiant energy and subsequent low carbon reserves. In another study, there was surprisingly rapid regeneration of experimentally injured corals on reefs after a major oil spill (Guzman et al. 1994). Anthropogenic pollution leading to nutrient enrichment and eutrophication substantially increases growth of marine algae and seaweeds, which settle within lesions and hinder tissue regeneration and recovery (Ramati 1994; Titlyanov and Titlyanova 2008). Greater understanding of how this important process is influenced under high CO_2 would provide fresh insights into the resilience of reef-building corals in a high CO_2 world.

Here, we tested how regeneration changed in high pCO_2/low pH conditions. We chose to use pCO_2 which are far-future predicted values (Barry et al. 2010) in order to amplify potential physiological and biochemical response thresholds of injured corals (Pörtner 2008). These are based on the "Logistic" emission scenarios for the year 2300 under anticipated global use of fossil fuel resources (Caldeira and Wickett 2005). Since previous studies on scleractinian corals showed that the capacity for regeneration is species-specific and depends on morphology and life history strategy (Meesters et al. 1996; Hall 1997; Henry and Hart 2005), we chose to perform this study on several species representative of contrasting life histories. This study is the first to present experimental data on the regenerative abilities of slow-growing versus fast-growing species from the Red Sea under various pCO_2/pH conditions.

Materials and methods

Experimental setup

The study was carried out in an outdoor seawater flow-through system at the Interuniversity Institute (IUI) for Marine Sciences in Eilat (Gulf of Aqaba, Red Sea; 29°30′N, 34°55′E). Seawater was pumped from 30-m depth and continuously transferred into three 1,000-L mixing tanks. pH was regulated using a pH controller (AquaMedic, Germany), connected to pH electrodes located in each mixing tank, and calibrated using NBS scale. Seawater pH was manipulated by bubbling pure CO_2 to attain the desired pH. Four common Indo-Pacific coral species were examined: the massive slow-growing corals *Porites lutea* [visual inspection of the corals suggested they were *P. lutea*, but due to the difficulties in species ID within the genus *Porites* (Forsman 2003), we refer to the samples

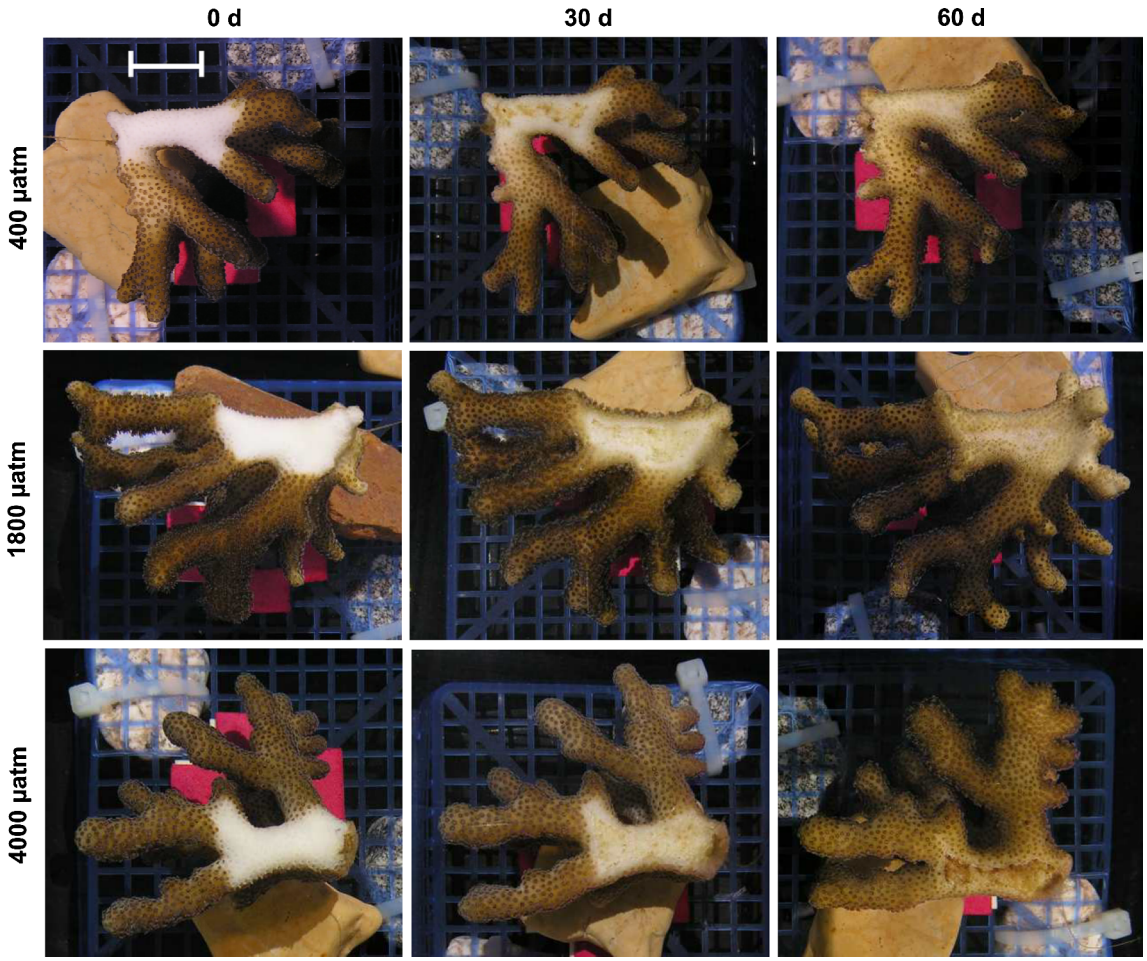

Fig. 1 Lesion recovery of representative *Stylophora pistillata* fragments after 0, 30, and 60 d under three pCO_2 treatments (400, 1,800, and 4,000 µatm). Thirty days from onset of the experiment, similar size lesions in 400 µatm (control) demonstrated a higher recovery rate when compared to 1,800 µatm (intermediate) and 4,000 µatm (high). After 60 d, there was complete tissue recovery in 400 µatm (control), whereas fragments in the intermediate and high treatments showed only partial recovery

hereon as *Porites* sp.] and *Favia favus*, as well as the branching fast-growing species, *Stylophora pistillata* and *Acropora eurystoma*. Colonies were collected in December 2010 from the reef in front of the IUI at 10 m depth. *Stylophora pistillata* and *A. eurystoma* colonies were fragmented. Surface area estimations of the colonies/fragments were performed using geometric measurements given the noninvasive and highly accurate nature of this technique (Naumann et al. 2009). Colonies (surface area, mean ± SEM = 58.2 ± 0.9 cm^2) or fragments (surface area, mean ± SEM = 65.1 ± 1.3 cm^2) of each species were transferred into aquaria (six 20-L aquariums X six replicates by species/by condition) and supplied with running seawater (0.5 l min^{-1}) at three different pCO_2 levels: (1) 400 µatm (control, pH 8.1), (2) 1,800 µatm (intermediate, pH 7.6), and (3) 4,000 µatm (high, pH 7.3).

After a 1-month acclimation period in the above-mentioned pCO_2 treatments, tissue lesions were inflicted using a regulator air-pick. To standardize the injury procedure for all colonies/fragments, the air-pick was held at a distance of 3 cm away from the fragment/colony and air pressure was kept constant. A template made of PVC was used to obtain a lesion of a specific area and protect the surrounding healthy tissue from damage. All the coral species were inflicted with a lesion area of 5–6 % of the total colony/fragment surface area. *Porites* sp. and *F. favus* colonies were inflicted with circular lesions with a surface area of 314 mm^2 (SE = 9; diameter = 20 mm) on top of the colony. *Stylophora pistillata* and *A. eurystoma* fragments were inflicted with wedge-shaped lesions with a surface area of 350 mm^2 (SE = 14) at the base (center) of the branch (Fig. 1). All tissues were removed from the injured areas leaving the underlying skeleton of the colony intact and without damage. In this way, we ensured that lesion repair in our study did not involve calcification mechanisms.

Corals were maintained in their respective pCO_2 treatment under ambient seawater temperature (ranging 21.6–24.3 °C as

Table 1 Carbonate chemistry of seawater in the three pCO_2 treatments (400, 1,800, and 4,000 µatm) calculated from pH_{NBS}, total alkalinity (TA), ambient seawater temperature, and salinity (41 ppm) using the program CO_2SYS (Pierrot et al. 2006)

pH_{NBS}	TA (µeq kg^{-1})	pCO_2 (µatm)	DIC (µmol kg^{-1})	HCO_3^- (µmol kg^{-1})	CO_3^{2-} (µmol kg^{-1})	$CO_{2(aq)}$ (µmol kg^{-1})	Ω_{arag}
8.18 (0.04)	2,501.6 (20.12)	404.5 (3.42)	2,122.6 (18.14)	1,846.4 (15.96)	265.5 (2.07)	10.6 (0.09)	4.02 (0.03)
7.62 (0.03)	2,499.3 (23.36)	1,788.3 (16.97)	2,430.7 (23.07)	2,296.8 (21.8)	82.3 (0.78)	51.6 (0.48)	1.23 (0.01)
7.31 (0.04)	2,501.3 (32.34)	3,811.9 (49.67)	2,550 (33.23)	2,397.9 (31.25)	42.1 (0.54)	110 (1.43)	0.63

All data shown are the mean (±SD). Dissolved inorganic carbon (DIC); aragonite saturation state (Ω_{arag})

measured in the aquaria during the experimental period; comparable to long-term SST for that time of the year in the Gulf of Aqaba). Ambient light intensity was reduced by 50 % with the use of plastic netting (1-mm mesh). The light conditions were the same for all treatments ranging 350–500 µmol quanta m^{-2} s^{-1} at midday during the experimental period. Corals were fed once a week with *Artemia salina* nauplii (400,000 per aquarium) for the entire duration of the experiment.

Monitoring software (AquaMedic, Germany) in the pH system showed that daily pH variability was low (±0.05 pH units) throughout the experiment. Temperature and pH_{NBS} in the aquaria were measured daily (CyberScan pH 11; Eutech Instruments Pte Ltd., Singapore). Total alkalinity (TA) in the aquaria and mixing tanks was measured regularly using a Metrohm 862 compact titrosampler (Cohen 2011). pCO_2, dissolved inorganic carbon (DIC), HCO_3^-, CO_3^{2-}, $CO_{2(aq)}$, and Ω_{arag} were calculated from the pH_{NBS} and TA measurements using the program CO_2SYS (Pierrot et al. 2006), selecting the constants of Mehrbach et al. (1973). Experimental seawater parameters are shown in Table 1.

Tissue regeneration

For tissue regeneration measurements, photographs were taken at various time intervals during the experiment by digital camera at a fixed distance using a scale (CoolPix 8400, Nikon, Japan) and analyzed with CPCe 4.0 (NCRI, USA) image analysis software (Kohler and Gill 2006). Recovery of injuries was then quantified and expressed as percentage of injury regenerated (as per Meesters et al. 1992). Mean values are presented ± SE of the mean (SEM) for percentage tissue regeneration from day 1 to day 60 for all the species ($n = 6$; Fig. 2) and the end of the experimental period ($n = 6$). The length of the experiment differed between species because of differences in recovery length (*Porites* sp. and *F. favus*, 120 d; *A. eurystoma*, 100 d; *S. pistillata*, 60 d).

Total protein, *Symbiodinium* density, and chlorophyll concentration

At the end of the experimental period, a set of colonies/fragments was sampled ($n = 4$ per species from each pCO_2 treatment), processed, and analyzed: *Porites* sp. and *F. favus* after 120 d; *A. eurystoma* after 100 d; *S. pistillata* after 60 d. Coral tissue was removed carefully using an airbrush containing 0.2 µm filtered sea water (FSW). An electric homogenizer (DIAX 100 homogenizer Heidolph Instruments GmbH & Co., KG, Schwabach, Germany) was used to homogenize the tissue extract for 30 s, followed by 100 µl of the homogenate removed for total protein analysis. Separation of coral tissue and *Symbiodinium* for cell density and chlorophyll concentration analysis were done by the following. The remaining homogenate was centrifuged for 5 min at 5,000 rpm (rcf 2,500 m s^{-2}; centrifuge 4K15 Sigma Laborzentrifugen GmbH, Osterode, Germany) followed by removing the supernatant. The pellet was resuspended in 1 ml FSW, homogenized, and centrifuged for 5 min at 5,000 rpm (rcf 2,500 m s^{-2}). The procedure was repeated twice more in order to remove remaining tissue. *Symbiodinium* were then resuspended in 1 ml FSW for cell counts on a haemocytometer. The cells were counted visually using a microscope (YS100, Nikon, Japan) and multiplied by 10,000 in order to get total cells present in each sample. Total number of *Symbiodinium* cells was normalized per surface area. Chlorophyll *a* was extracted from *Symbiodinium* in 1 ml acetone (90 %) at 4 °C for 15 h. Concentrations of chlorophyll *a* were calculated using spectrophotometry (Ultrospec 2100 pro, GE Bioscience, USA) and the standard equations (Jeffrey and Humphrey 1975). Chlorophyll concentration was calculated per *Symbiodinium* cell. Total protein was quantified using the Quick Start Bradford Protein Assay Kit and Quick Start Bovine Serum Albumin Standard Set (Bio-Rad Laboratories, Hercules, CA, USA). Optical density was read at 595 nm using an ELISA reader (PowerWave XS, BioTek, USA). Total protein to surface area ratio was determined to examine changes in coral biomass.

Statistical analyses

The effect of pCO_2 on tissue regeneration ($n = 6$) was analyzed separately for each species, using a one-way analysis of variance (ANOVA). A multiple comparisons test (Tukey's) was used to distinguish groups that differed significantly. The effect of pCO_2 on total protein, *Symbiodinium*

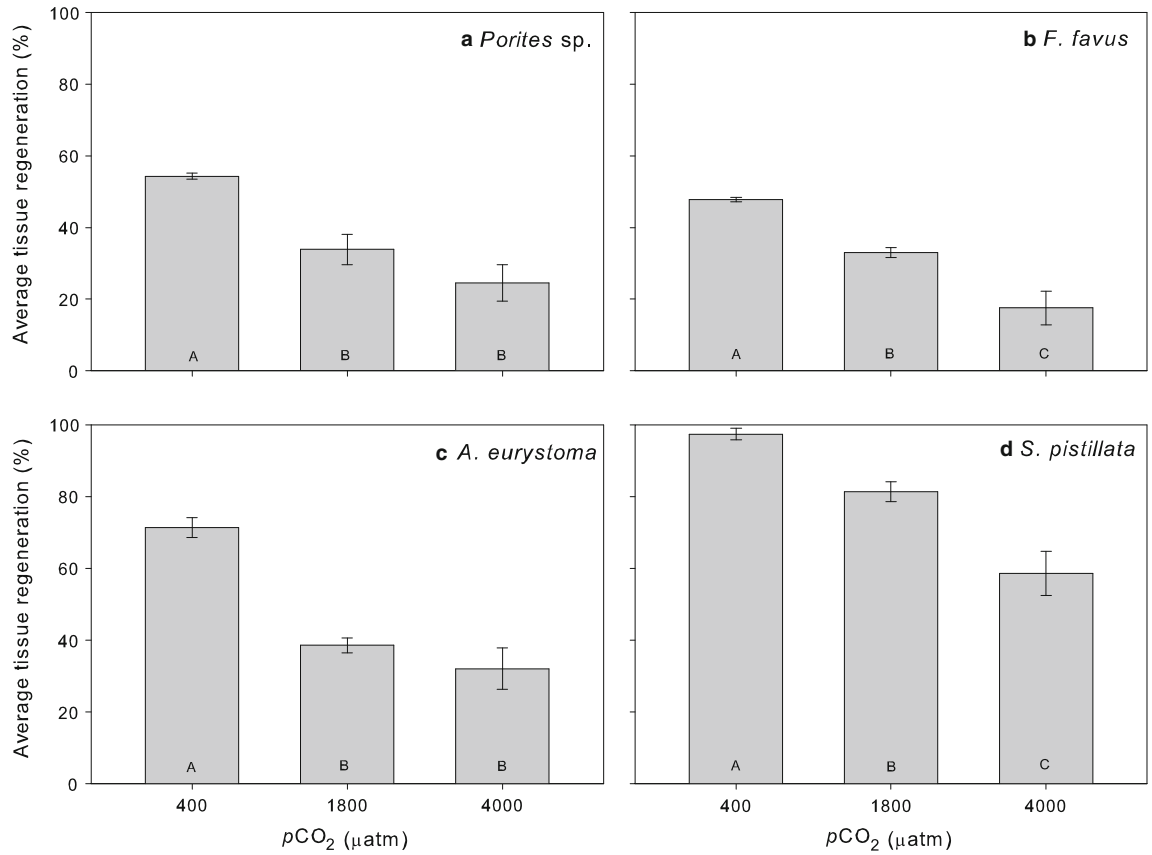

Fig. 2 Percentage tissue regeneration of lesions after 60 d under three pCO_2 treatments (400, 1,800, and 4,000 µatm) in: **a** *Porites* sp. (mean ± SEM; $n = 6$), **b** *Favia favus* (mean ± SEM; $n = 6$), **c** *Acropora eurystoma* (mean ± SEM; $n = 6$), and **d** *Stylophora pistillata* (mean ± SEM; $n = 6$). *Letters* indicate significant differences in percentage tissue regeneration between different pCO_2 treatments (Tukey, $p < 0.05$)

density, and chlorophyll concentration ($n = 4$ for each parameter) was analyzed separately for each species, using the Kruskal–Wallis test. Post hoc Mann–Whitney *U* tests were run for separation of significant factors. Differences between factors were considered significant for a *p* value < 0.05. Unless otherwise specified, mean values are presented ± SEM. SPSS version 20 (SPSS IBM, New York, USA) was used to perform the statistical analysis.

Results

During the experimental period, there was no mortality of colonies/fragments. Growth of turf algae within lesion areas was recorded. Similar to other studies, we observed the regenerating coral tissue quickly overgrowing the algal settlement (Meesters and Bak 1993; Titlyanov et al. 2005).

Tissue regeneration

Tissue regeneration of *Porites* sp. colonies after 60 d in the intermediate and high treatments was 20 and 29 % lower compared to the control, respectively ($F_{2,15} = 15.7$, $p < 0.001$; Fig. 2a). After 120 d, tissue regeneration in the same colonies was 42 and 54 % lower in the intermediate and high treatments compared to the control, respectively ($F_{2,15} = 28.81$, $p < 0.001$). No significant difference was found between the intermediate and high treatments at both time points (after 60 and 120 d). For *F. favus* colonies, there was a significant difference in tissue regeneration between all pCO_2 treatments after 60 d as determined by one-way ANOVA and post hoc comparison test ($F_{2,15} = 28.38$, $p < 0.001$), with a decrease of 15 and 30 % in the intermediate and high treatments, respectively (Fig. 2b). Similar results in the same colonies were found after 120 d with a significant decrease in tissue regeneration in the intermediate and high treatments of 30 and 52 %, respectively ($F_{2,15} = 32.88$, $p < 0.001$). *Acropora eurystoma* fragments in the intermediate and high treatments experienced a 22 and 39 % decline in tissue regeneration after 60 d, respectively, compared to the control ($F_{2,15} = 29.42$, $p < 0.001$; Fig. 2c). After 100 d, tissue regeneration in the same fragments was 42 and 47 % lower in the intermediate and high treatments, respectively

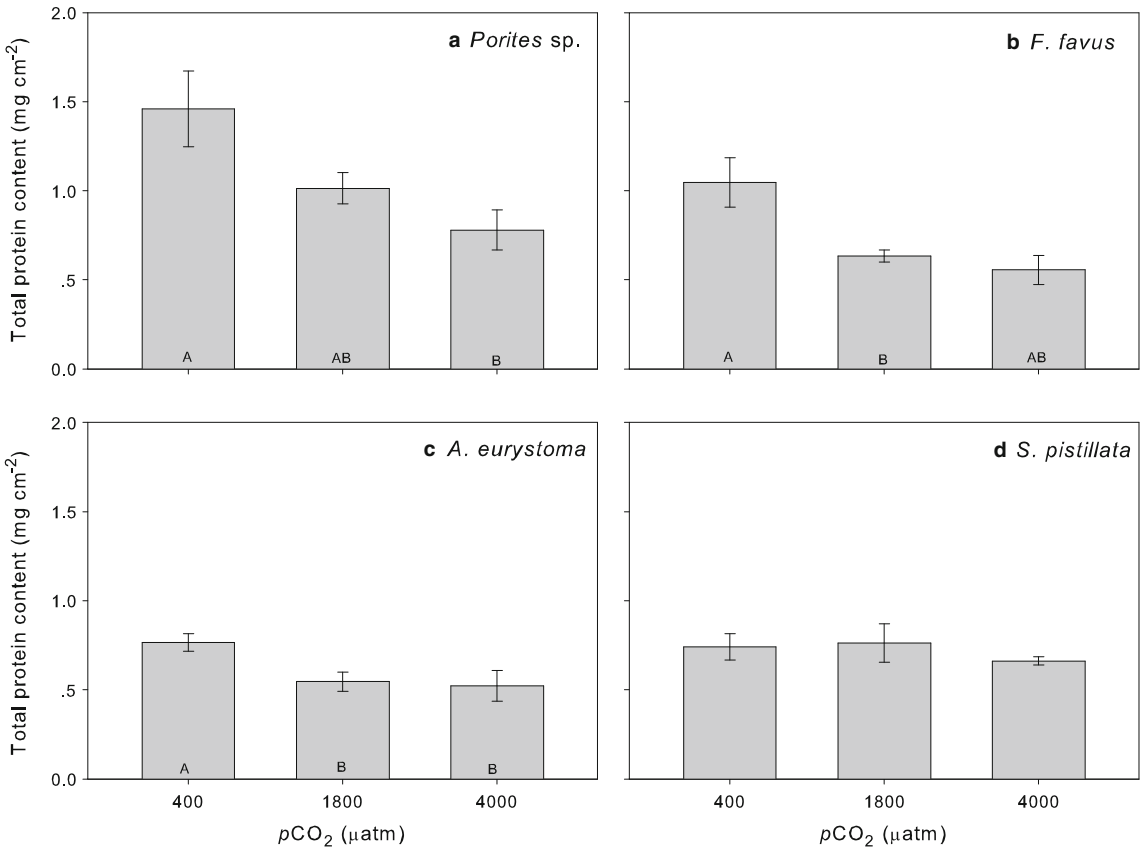

Fig. 3 Total protein content (mg cm^{-2}) under three pCO_2 treatments (400, 1,800, and 4,000 µatm) in: **a** *Porites* sp. (mean ± SEM; $n = 4$), **b** *Favia favus* (mean ± SEM; $n = 4$), **c** *Acropora eurystoma* (mean ± SEM; $n = 4$), and **d** *Stylophora pistillata* (mean ± SEM; $n = 4$). *Letters* indicate significant differences in total protein content between different pCO_2 treatments (Mann–Whitney, $p < 0.05$)

($F_{2,15} = 41.4$, $p < 0.001$). There was no significant difference between the intermediate and high treatments at both time points (after 60 and 100 d). One-way ANOVA revealed a significant difference in tissue regeneration between all pCO_2 treatments for *S. pistillata* fragments after 60 d ($F_{2,15} = 23.54$, $p < 0.001$), with a 16 and 39 % decrease in the intermediate and high treatments, respectively, compared to the control (Fig. 2d).

Total protein, *Symbiodinium* density, and chlorophyll concentration

Total protein concentration of *Porites* sp. was significantly higher in the control treatment (1.45 ± 0.21 mg cm^{-2}) than in the high treatment (0.77 ± 0.11 mg cm^{-2}) (Kruskal–Wallis ANOVA, Mann–Whitney, $df = 2, p < 0.05$; Fig. 3a); however, no significant change was found in the intermediate treatment (1.01 ± 0.08 mg cm^{-2}). For *F. favus*, there was a significant difference in total protein concentration only between the control (1.04 ± 0.13 mg cm^{-2}) and intermediate treatment (0.63 ± 0.03 mg cm^{-2}) (Kruskal–Wallis ANOVA, Mann–Whitney, $df = 2$, $p < 0.05$; Fig. 3b). In both the intermediate and high treatments, total protein concentration of *A. eurystoma* declined significantly (0.54 ± 0.05 and 0.52 ± 0.08 mg cm^{-2}, respectively) as compared to the control (0.76 ± 0.04 mg cm^{-2}) (Kruskal–Wallis ANOVA, Mann–Whitney, $df = 2, p < 0.05$) (Fig. 3c). There were no significant differences in total protein concentration of *S. pistillata* between all pCO_2 treatments (Fig. 3d).

Exposure to the intermediate treatment had a significant effect on *Symbiodinium* density of *Porites* sp. (0.25 ± 0.03 cells × 10^6 cm^{-2}), resulting in a 51 % decrease compared to the control (0.51 ± 0.09 cells × 10^6 cm^{-2}) (Kruskal–Wallis ANOVA, Mann–Whitney, $df = 2$, $p < 0.05$; Fig. 4a). Although the density was lower in the high treatment (0.32 ± 0.05 cells × 10^6 cm^{-2}) for this species, this decrease was not significant. There was a significant decrease in *Symbiodinium* density in *F. favus* of 44 and 70 % in the intermediate (0.43 ± 0.1 cells × 10^6 cm^{-2}) and high (0.23 cells × 10^6 cm^{-2}) treatments as compared to the control (0.77 ± 0.04 cells × 10^6 cm^{-2}), respectively (Kruskal–Wallis ANOVA, Mann–Whitney, $df = 2$, $p < 0.05$; Fig. 4b). In contrast, *Symbiodinium* density in *A. eurystoma* from all pCO_2 treatments did not differ

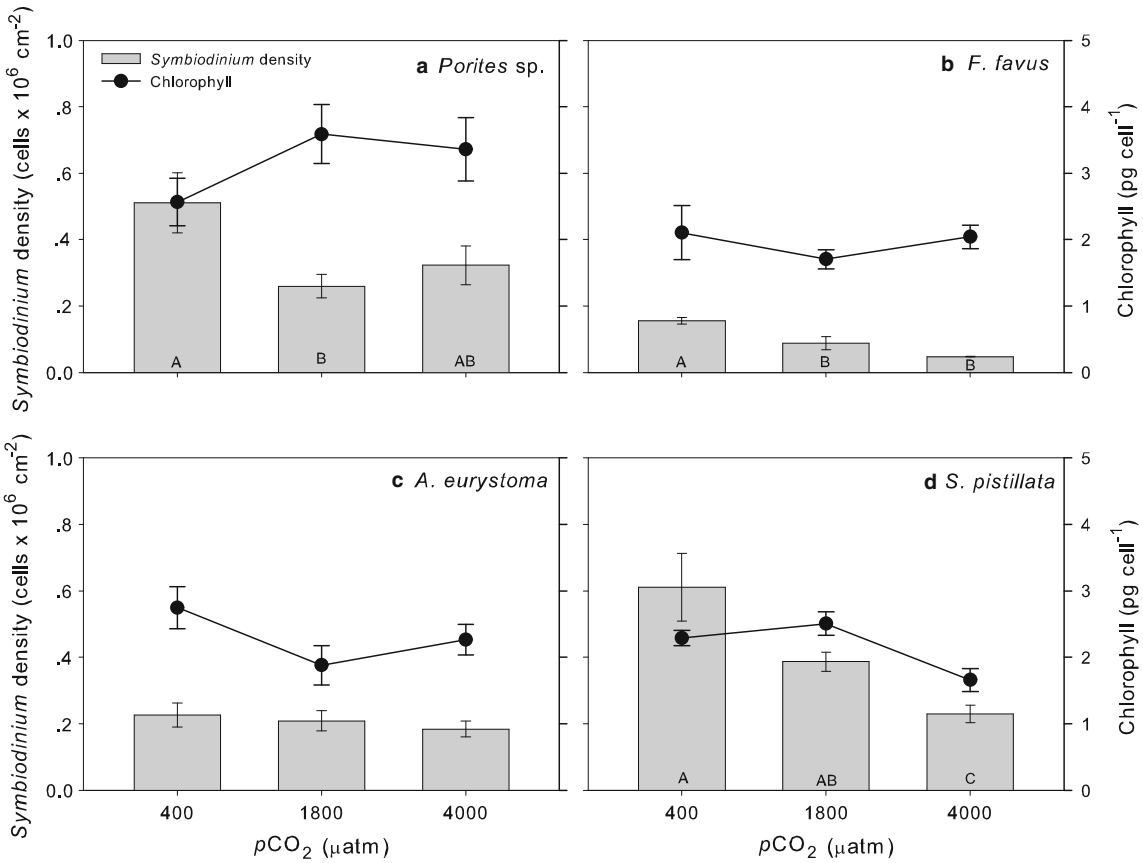

Fig. 4 *Symbiodinium* density (cells × 10^6 cm^{-2}) and chlorophyll concentration (pg $cell^{-1}$) under three pCO_2 treatments (400, 1,800, and 4,000 µatm) in: **a** *Porites* sp. (mean ± SEM; $n = 4$), **b** *Favia favus* (mean ± SEM; $n = 4$), **c** *Acropora eurystoma* (mean ± SEM; $n = 4$), and **d** *Stylophora pistillata* (mean ± SEM; $n = 4$). *Letters* indicate significant differences in *Symbiodinium* density between different pCO_2 treatments (Mann–Whitney, $p < 0.05$)

significantly (Fig. 4c). ANOVA followed by post hoc comparison test revealed a significant difference in *Symbiodinium* densities of *S. pistillata* between all pCO_2 treatments (Kruskal–Wallis ANOVA, Mann–Whitney, $df = 2$, $p < 0.05$; Fig. 4d), with a decrease of 38 and 62 % from control (0.61 ± 0.1 cells × 10^6 cm^{-2}) to intermediate (0.38 ± 0.02 cells × 10^6 cm^{-2}) and high (0.23 ± 0.02 cells × 10^6 cm^{-2}) treatments, respectively.

Chlorophyll concentration in *Symbiodinium* of *S. pistillata* was significantly higher in the control (2.28 ± 0.11 pg Chl $cell^{-1}$) and intermediate treatment (2.5 ± 0.17 pg Chl $cell^{-1}$) as compared to the high treatment (1.65 ± 0.17 pg Chl $cell^{-1}$) (Kruskal–Wallis ANOVA, Mann–Whitney, $df = 2$, $p < 0.05$; Fig. 4d), whereas there were no significant differences detected in *Symbiodinium* chlorophyll concentrations of *Porites* sp., *F. favus*, and *A. eurystoma*.

Discussion

The present study investigated the effect of increased pCO_2 on the regeneration of four reef coral species. Regeneration is regarded as a highly prioritized and life-preserving process which often derives energy and cells following injury at the expense of other vital biological traits (Meesters et al. 1997a). In this study, we have shown that: (1) regeneration capacity of corals is reduced in high pCO_2/low pH conditions, (2) rate of repair is species-specific and may reflect species-specific energetic management efficiency or resilience to high pCO_2, and (3) corals are able to maintain regeneration even at very high pCO_2/low pH conditions, although this may be at the expense of other biological processes.

All coral species exhibited significantly higher tissue regeneration capacity in ambient pCO_2 seawater when compared to high pCO_2/low pH treatments (Figs. 1, 2). We proclaim that high pCO_2/low pH conditions exposed the studied coral species to unfavorable conditions thereby impairing regeneration performance. Generally, the regeneration of injuries within colonies of the same species is not uniform and depends on many extrinsic factors (Henry and Hart 2005). Corals may regenerate more rapidly during seasonally high water temperatures (Kramarsky-Winter and Loya 2000) but have reduced regeneration beyond their

thermal threshold (Meesters and Bak 1993) and subsequent bleaching. Regeneration of experimentally wounded corals may sometimes cease completely following bleaching (Meesters and Bak 1993; Meesters et al. 1997a; Fine et al. 2002). Food availability may restrict regenerative capacity for corals in deep water due to reduced energy reserves as light levels decrease (Nagelkerken et al. 1999; Fisher et al. 2007). Habitats with high sedimentation levels may also slow down regeneration rates by increasing coral energy requirements from the costs of removing sediment and decrease energy availability as a result of shading the photosynthetic endosymbionts (Meesters et al. 1992). Interestingly, Guzman et al. (1994) observed faster regeneration of several injured coral species at sites polluted by a major oil spill than at unaffected locations, possibly at the expense of other life history processes including growth and reproduction. Thus, an extensive number of studies describe an intricate relationship between environmental conditions and coral regeneration performance with both positive and negative effects (Henry and Hart 2005). Our results show that exposure to high pCO_2 is negatively correlated with regeneration capacity in the examined coral species. Unfavorable conditions surrounding the colony expose the coral to stress and may influence the amount of resources available for regeneration (Meesters et al. 1997b; Cróquer et al. 2002; Fine et al. 2002).

Evidently, the underlying processes associated with the slower regeneration of injuries under high pCO_2/low pH are complex and may be related to resource trade-offs, energy cost of acid/base regulation, and/or decrease in total energy budget. Although it was not within the scope of our study to elucidate the mechanisms behind the reduced regeneration, we discuss potential aspects which may explain our results. Apparent trade-offs between major life history processes and regeneration is a well-studied area in corals (Henry and Hart 2005). Injuries may invoke a decline in reproduction (Rinkevich and Loya 1989; Hall 1998) and growth (Meesters et al. 1994) on a colony-wide scale in some species, although this is not always the case. Occasionally, trade-offs in favor of regeneration are avoided, and other biological processes such as growth (Denis et al. 2013) are enhanced. Regeneration of very large wounds may halt in favor of growth and reproduction if the fitness of the individual is threatened by resource limitation (Meesters et al. 1997a). In this study, there was a decline in regeneration capacity under intermediate and high pCO_2 for all four species. OA is generally considered to reduce the growth rates of corals (Anthony et al. 2011), their reproductive output, and early life history processes (Albright et al. 2010; Albright and Langdon 2011). While we did not measure reproductive output or growth rates of the species included, our results infer that resources available for regeneration of corals in ambient seawater are presumably being utilized for other processes under elevated pCO_2. Additionally, the colonization of lesions by algae and other fouling organisms may divert some of the resources available for regeneration toward competition, resulting in slower regeneration rates (Hall 1998).

The observed slower regeneration in increased pCO_2 may also reflect more investment in maintenance (i.e., acid/base regulation). Animals in high pCO_2/low pH conditions are required to compensate for acid–base imbalance in intra- and extracellular spaces (Fabry et al. 2008). Stress-based impairment at the cellular level in high pCO_2/low pH conditions may trigger acute survival responses in the corals that re-direct resources into colony maintenance and cell pH homeostasis. There is a range of cellular events that occur under acidosis, including metabolic depression and acid–base regulation via costly changes in cell membrane transporters (Pörtner et al. 2000; Kaniewska et al. 2012). A recent study by Venn et al. (2013) investigated changes in pH at the tissue-skeleton interface and calcification of *S. pistillata* after exposure to increasing pCO_2 levels. Except for the lowest pH treatment (pH 7.2) in the previously mentioned study, their results showed that the intracellular pH of calicoblastic cells and extracellular pH in the fluid at the tissue–skeleton interface (subcalicoblastic medium) was regulated and maintained at a steady state under increased pCO_2 conditions, suggesting increased energy investment in homeostasis. Likewise, injured corals in our study may avert energy and resources away from regeneration toward cell pH homeostasis and elevated maintenance costs.

Performance of an organism under both "normal" and "stressful" conditions is primarily determined by the energetic status of the individual (Lesser 2013). Slower regeneration when faced with "stressful" conditions, therefore, suggests a lowered energetic state of the coral. The energetic budget of the coral holobiont relies in a large part on autotrophic carbon acquisition of symbiont photosynthesis (Muscatine 1990). Tremblay et al. (2013) observed a decrease in total autotrophic carbon acquisition of *S. pistillata* nubbins under high pCO_2/low pH conditions resulting from a severe decline of 48 % in the symbiont population. Gross photosynthesis supplied 22 % less carbon to nubbins maintained under pH 7.2 (79 ± 1 μg C cm^{-2} d^{-1}) compared to those maintained under pH 8.1 (101 ± 6 μg C cm^{-2} d^{-1}). As in the study by Tremblay et al. (2013) and other studies subjecting corals to long-term acidification (Anthony et al. 2008; Krief et al. 2010; Kaniewska et al. 2012), our results showed *Porites* sp., *F. favus*, and *S. pistillata* experienced a decrease of 37, 70, and 62 %, respectively, in *Symbiodinium* density in the high treatment (Fig. 4a, b, d) at the end of the experimental period for each species (*Porites* sp., 120 d; *F. favus*, 120 d; *S. pistillata*, 60 d). For *A. eurystoma*, however, the symbiont concentration

in the intermediate and high treatments remained essentially unaltered (after 100 d; Fig. 4c). If the overall energetic budget of the coral holobiont under high pCO_2 is gradually diminished with declining total autotrophic carbon acquisition, the energy and resources available for regeneration from injury may be limited. The performance of the holobiont under stress conditions may be attributed to the physiology of different *Symbiodinium* genotypes (DeSalvo et al. 2010), which may respond differently in various coral hosts (Abrego et al. 2008). Coral species collected from shallow water depth (up to 6 m) at our study site harbored various *Symbiodinium* clades (Karako-Lampert et al. 2004), with *S. pistillata* hosting clade A, as opposed to *Acropora* sp. and *F. favus* harboring clade C. While these differences in *Symbiodinium* genotype may imply different life strategies (Karako-Lampert et al. 2004), it does not necessarily explain patterns of regeneration under stress-related high pCO_2. In the study by Tremblay et al. (2013), in addition to significant loss of *Symbiodinium*, the host was unable to maintain its biomass with total protein content decreasing in colonies maintained at high pCO_2/low pH. Our study shows similar results at the end of the experiment with a decrease in total protein content in *Porites* sp., *F. favus*, and *A. eurystoma* under the intermediate and high treatments (Fig. 3a–c). *S. pistillata*, which was the fastest to regenerate in the study, experienced a reduction in *Symbiodinium* density but maintained a constant protein content (tissue biomass) under all pCO_2 treatments (Fig. 3d). This implies this species' robustness under acidification conditions, as shown in a study by Krief et al. (2010) and may be explained by increased rate of photoassimilates translocation from symbiont to host under reduced pH (Tremblay et al. 2013).

Differential susceptibility of scleractinian corals to increased pCO_2/decreased pH is increasingly well documented (e.g., Anthony et al. 2008), although species-specific thresholds must be investigated further. Regenerative ability in this study differed between species when comparing lesion repair after 60 d. When making this comparison, we take into account several limitations. Our study chose species representative of contrasting life histories and morphologies. Infliction of identical lesions across species is particularly difficult. Lesion shape was restricted by morphological differences between branching (wedge-shaped lesions) versus massive (circular lesions) species. Inevitably, the lesion perimeter to surface area ratios and number of polyps injured, which may influence rates of repair (Oren et al. 1997; Lirman 2000), were not the same. The fast-growing branching corals (*S. pistillata* and *A. eurystoma*) in our study regenerated faster than the slow-growing massive species (*Porites* sp. and *F. favus*) after 60 d (Fig. 2). Indeed, several studies examining regenerative ability of injured colonies with different morphologies and life history strategies have found branching corals to recover faster than massive species (Jackson 1979; Hall 1997). This pattern was conserved in the corals in the intermediate and high treatments (Fig. 2), presumably representing contrasting life strategies in response to damage under stressful conditions. The recovery rate of *S. pistillata* from injury was the fastest among all the species included in the study. This species was especially robust to the intermediate and high treatments, with up to 58 % recovery after 60 d in the high treatment (Fig. 2d). The impact of tissue injury will depend partly on the depth of the tissue as some coral species are fleshier than others (Veron 2000). A study by Hall (1998), which observed slower regeneration of tissue injuries for massive species than for branching species, was explained in part by greater tissue depth in the massive species. *S. pistillata* and *A. eurystoma* are characterized as "skinny" (i.e., thin tissue) species with high surface to volume ratio (*S*/*V*) ratio, while *F. favus* is a "fleshy" (i.e., thick tissue) species that forms massive colonies thus having a low *S*/*V* ratio (Veron 2000; Loya et al. 2001). *Porites* spp. are also known to have thick tissue with an average tissue depth of 5 mm (Barnes and Lough 1992; Veron 2000). Hence, greater tissue depth may be related to the slow rate of repair we found in the massive *Porites* sp. and *F. favus* in our study (Fig. 2a, b). *F. favus* was the slowest to regenerate, with only 18 % average recovery of lesion areas after 60 d in the high treatment (Fig. 2b). Several studies have shown polyp size in colonial corals is related to regenerative capacity. Small polyp taxa such as *Acropora* and *Pocillopora* regenerate more rapidly than large polyp taxa such as *Favia* and *Platygyra* (Fishelson 1973; Riegl and Velimirov 1991). These characteristics of *F. favus* were further magnified under the stress-inducing treatment of high pCO_2. Our results imply this species was more susceptible to high-CO_2 dosing and the *Symbiodinium* density was reduced by 70 % in the high treatment relative to the control after 120 d, suggesting *F. favus* falls into the CO_2-sensitive group.

Nonetheless, even at sub-optimal and extreme conditions, corals continued to function and recover from injury, possibly at the expense of growth, calcification, reproduction, and/or other biological functions. In the case of *Porites* sp. and *A. eurystoma*, there was no further significant decrease in tissue recovery between the intermediate and high treatments. We have demonstrated that the coral species included in the study can tolerate extremely high pCO_2 levels which convey far-future predicted values and maintain colonial integrity and regenerative ability for extended periods of time at pCO_2 reaching ten times normal conditions. It is important to note that OA studies are carried out at a much faster rate than the actual process occurring in the oceans, thereby advocating the potential of scleractinian corals to adapt/acclimatize to these conditions. Future studies on coral regeneration should incorporate multiple stressors, particularly acidification-

warming interactions, to assess the interactive effects of environmental change, improving our understanding of coral performance under future climatic scenarios.

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