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The effects of ocean acidification on wound repair in the coral *Porites* spp.

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ABSTRACT

Scleractinian corals on tropical reefs are exposed to many natural and anthropogenic disturbances, and while much is known about their responses to such conditions, it is unclear whether the responses will remain the same in a future affected by climate change and ocean acidification. To evaluate how one aspect of these effects — wound repair — might be influenced by ocean acidification, small colonies of massive *Porites* spp. from the back reef of Moorea, French Polynesia, were damaged to simulate the effects of single bites by corallivorous fishes, and healing was measured under contrasting P_{CO2} regimes. Using experiments lasting 19–20 d and employing superficial (2013) or deep (2014) lesions, the effects of damage were evaluated at ~400 μatm (ambient) and ~1000 μatm P_{CO2} (both at ~28.5 °C) using calcification and healing as dependent variables. Damage reduced calcification of massive *Porites* spp. in 2013, but not in 2014, and P_{CO2} had no effect on area-normalized calcification or healing in both years, although biomass-normalized calcification was reduced by high P_{CO2} in 2014. Overall these results reveal the physiological resilience of this functional group of corals to the extent of ocean acidification expected by the end of this century, and suggest that over this period small colonies will remain capable of recovery from minor damage arising from fish corallivory.

1. Introduction

In the marine environment, the ability of colonial modular organisms to recover from partial mortality mitigates the negative effects of damage (Jackson and Coates, 1986). This is important for sessile taxa that cannot flee from sources of damage, and for those that are iteroparous, successful repair can increase the likelihood of future reproduction (Henry and Hart, 2005). The ability to survive damage is made possible by the capacity to regenerate damaged tissue, or to survive the loss of tissue if repair is impossible (Henry and Hart, 2005). There is a long history of interest in the regenerative capacity of colonial modular organisms (Jackson, 1979; Palumbi and Jackson, 1982; Hughes, 1983), and tropical scleractinians have served as a model system in advancing this interest (e.g., Nagelkerken and Bak, 1998; Oren et al., 2001).

Scleractinians frequently experience partial mortality through corallivory and breakage (Reese, 1977; Frydl, 1979; Rotjan and Lewis, 2008; Bruckner and Bruckner, 2016), and these events typically create lesions in which tissue and skeleton are removed (Henry and Hart, 2005). The combined effects of these lesions can sum among members of a population to represent greater tissue loss than occurs through the death of whole colonies (Hughes and Jackson, 1985). Lesions attributed to corallivory are a chronic disturbance for those scleractinians targeted by fish corallivores (Rotjan and Lewis, 2005), but the extent of damage inflicted varies among species of fish (Bellwood and Choat, 1990; Rotjan and Lewis, 2008). Some fish remove individual polyps without damaging the skeleton, while others remove large amounts of skeleton and tissue (Cole et al., 2008). For many years there was scant evidence that fish corallivory had strong effects on scleractinian communities (Hixon, 1997; Cole et al., 2008; Rotjan and Lewis, 2008), but this disturbance is now viewed as an important factor affecting the abundance, distribution, and fitness of scleractinians (Cole et al., 2008; Rotjan and Lewis, 2008).

When a scleractinian is damaged, repair requires the allocation of resources to replace damaged tissue, and subsequently biological functions, including growth and reproduction, may be impaired as resources are prioritized for this purpose (Rinkevich, 1996; Henry and Hart, 2005). The rate at which tissue is regenerated is important, because the likelihood that fouling organisms will colonize lesions increases with the duration of exposure of skeleton within the lesion (Meesters et al., 1997; Henry and Hart, 2005). Once fouling organisms colonize a lesion, it is unlikely that coral tissue will grow over the site of damage (Meesters et al., 1997). Whether coral tissues regenerate is determined by intrinsic factors, such as corallum morphology, the supply of interstitial cells, and the availability of metabolic energy (Meesters et al., 1996; Henry and Hart, 2005), as well as extrinsic factors including seawater depth and the magnitude of damage (Meesters et al., 1997; Nagelkerken et al., 1999). As global climate change leads to increases in seawater temperature (Bindoff et al., 2007), and reduced

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seawater pH (i.e., ocean acidification [Doney et al., 2009]), determining how extrinsic factors influence tissue repair in scleractinians will help to understand how they will respond to damage in the future.

Most research on the effects of ocean acidification on scleractinians has focused on calcification (Erez et al., 2011), but this phenomenon has the potential to affect other biological functions (Doney et al., 2009). As studies of the response of scleractinians to ocean acidification have broadened their consideration of treatment conditions, there has been growing attention to interactive effects, such as might occur in synergy with elevated temperature, feeding, light intensities, and flow speeds (Erez et al., 2011; Chan and Connolly, 2013; Edmunds et al., 2012; Comeau et al., 2014a). To date, the possibility that ocean acidification could modulate recovery from partial mortality has rarely been considered (but see Renegar et al., 2008; Horwitz and Fine, 2014; Hall et al., 2015). Since changes in environmental conditions associated with ocean acidification could influence the availability of cellular and energetic resources for tissue regeneration, it is reasonable to hypothesize that tissue regeneration will be affected (Henry and Hart, 2005).

This study investigated the effects of ocean acidification on the recovery of a scleractinian from partial mortality. Two experiments were performed to address the effects of modest (in 2013) and severe (in 2014) damage on the common Indo-Pacific coral, massive *Porites* spp. Experiment 1 tested the effects of ocean acidification on calcification and tissue regeneration in corals recovering from damage created with a scar extending 2–3 mm into the skeleton and tissue. Experiment 2 was similar, except that damage was more severe and consisted of a scar removing tissue and skeleton extending 5–6 mm into the skeleton and tissue. Both experiments were conducted with small colonies of massive *Porites* spp. from the back reef in Moorea, French Polynesia, and colonies were incubated in mesocosms supplied with flowing seawater, and controlled for P_{CO2}, temperature, and irradiance. Corals were damaged to simulate corallivory resulting from fishes feeding in scraping (Experiment 1) and excavation (Experiment 2) feeding guilds (Bellwood and Choat, 1990).

2. Materials and methods

2.1. Collection and acclimation

Experiments took place at the Richard B. Gump South Pacific Research Station on Moorea, and used small colonies (~4 cm diameter) of massive *Porites* spp. from 2 to 3 m depth in the back reef. Massive *Porites* spp. was used because small colonies of this taxon are common throughout the back reef, where they are subject to corallivory (Lenihan and Edmunds, 2010). Moreover, throughout the Indo-Pacific, members of this group play important roles in coral reef community structure. In Moorea, the back reef is populated by at least three species of massive *Porites* spp., with *P. lutea* and *P. lobata* particularly common (Bosserelle et al., 2014). These species are distinguished by corallite structure (Veron and Pichon, 1982), but the differences are difficult to detect underwater, and may not conform to species boundaries discerned from genetic tools (Forsman et al., 2009). Therefore, small colonies of similar shape, color, and size were combined as massive *Porites* spp., which previously we have distinguished using morphological features to 85% *P. lutea* and 15% *P. lobata* (Edmunds, 2009).

An experiment was designed in which corals were damaged in a standardized way (i.e., scars of similar area and depth), and grown in mesocosms under two P_{CO2} levels. Each P_{CO2} treatment was created in replicate tanks containing multiple corals to alleviate the limitations of pseudoreplication (sensu Hurlbert, 1984) (described below). In Experiment 1, six corals were allocated to each treatment combination (damage versus undamaged × P_{CO2}) in each of three tanks at both P_{CO2} treatments (72 corals), and in Experiment 2, 10 corals were allocated to each treatment combination in each of two tanks at both P_{CO2} treatments (80 corals).

Corals were brought to the laboratory and attached to plastic tiles using epoxy (*Z*-Spar A-788, Splash Zone Compound Los Angeles, CA, USA) without damaging marginal tissues to induce a stress response. To allow recovery following collection, corals were retained in a 1000 L tank for 7 d in Experiment 1 (at 28.8 °C and 481 μmol quanta m^{-2} s^{-1} irradiance) and 6 d in Experiment 2 (at 28.9 °C and 489 μmol quanta m^{-2} s^{-1} irradiance). Temperature was measured with a certified digital thermometer (±0.05 °C model 15-077, Thermo Fisher Scientific, Waltham, MA, USA), and light intensity with a 4-π quantum light sensor (LI-193SA sensor and LI-1400 m, LI-COR, Inc., Lincoln, NE, USA), and the levels of these factors were similar to the conditions at the site of collection in April when the experiments were conducted. Light was provided using four LED lamps (LED System Model: Sol Blue, AquaIllumination®, Ames, IA, USA).

2.2. Experimental treatments

In both experiments, half of the corals were damaged using snub-nose pliers to scrape a rectangular lesion on their upper surface, and care was taken to ensure the lesions were similar in size. Pliers were rinsed in clean seawater between damaging colonies to avoid cross-contaminating replicate corals with mucus and microbes. In Experiment 1, which involved superficial damage, lesions had a mean area of 0.99 cm^2 and penetrated ~1–2 mm into the colony surface. In massive *Porites* spp., the coral tissue forms a layer 5–6 mm thick, and therefore this damage did not extend across the full width of the tissue. In Experiment 2, which involved deeper scars and more severe damage, lesions again were created with snub-nose pliers, but their area was increased to 2.5 cm^2 and they were scraped to a depth of ~5–6 mm into the skeleton. The dimensions of lesions were similar to those created by parrotfish feeding on massive corals (Bellwood and Choat, 1990; Rotjan and Lewis, 2005), with those created in Experiment 1 resembling bites of fishes in the scraping guild, and those in Experiment 2 resembling bites of fishes in the excavating guild (Bellwood and Choat, 1990). The other half of the corals was left undamaged and served as control for the damaged treatment.

Following damage, corals were retained in the acclimation tank for 1 d until mucus release attributed to damage ceased, and then control and damaged corals were randomly assigned to 150 L tanks (AquaLogic, San Diego, CA, USA) that were pre-set to treatment conditions (ambient and elevated P_{CO2}). Six tanks were used in Experiment 1 and four in Experiment 2, and all were supplied with fresh seawater at 100–200 mL min^{-1}. Half of the tanks were bubbled with ambient air, and the other half with CO_2-enriched air to maintain P_{CO2} at target values of ~400 μatm (ambient) and ~1000 μatm (elevated). The elevated P_{CO2} treatment corresponds to the pessimistic representative concentration pathway (RCP 8.5) that projects atmospheric P_{CO2} to increase to ~1370 ppm by 2100 (van Vuuren et al., 2011). Tanks were maintained at 28.0 °C in Experiment 1, and 28.8 °C in Experiment 2, with the slight difference reflecting logistical constraints. Corals were incubated for 19 d in Experiment 1 and 20 d in Experiment 2, and were randomly repositioned within the tanks daily to account for position effects.

Tanks were monitored for temperature, irradiance, salinity, pH, and carbonate chemistry. Temperature was measured twice daily using a digital thermometer and irradiance was measured using a 4-π quantum PAR sensor and meter (both described above). In Experiment 1, irradiance in the tanks was measured weekly, and in Experiment 2 it was measured daily.

In Experiment 1, salinity was recorded every 2 d using a conductivity meter (YSI 3100), and pH was measured 3 times d^{-1} (using a DG115-SC probe [Mettler-Toledo, Columbus OH, USA]), fitted to an Orion 3 star meter [Thermo Fisher Scientific, Waltham, MA, USA], calibrated on the total scale using TRIS/HCl buffers (Dickson et al., 2007). Total alkalinity (TA) was measured by potentiometric titration (T50, Mettler-Toledo) following SOP 3b (Dickson et al., 2007), and initially was calculated daily and, once stable, every 2 d. Carbonate chemistry parameters and