Tolerance to a highly variable environment does not infer resilience to future ocean warming and acidification in a branching coral

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Abstract

Coral populations from reef habitats that experience extreme daily abiotic fluctuations have been suggested as candidates to survive and proliferate under future climate change. Few studies, however, have exposed corals from dynamic environments to the synergistic effects of ocean warming and acidification to investigate whether tolerance of present-day environmental variability is maintained under future climate stress. This study assessed the impact of RCP2.6 (+0.8°C and +25 ppm) and RCP4.5 (+1.3°C and +66 ppm) ocean warming and acidification on the survivorship, primary calcification (i.e., extension), secondary calcification (i.e., densification), and protein densities of *Isopora palifera* originating from two distinct reef habitats (abiotically variable reef flat vs. stable reef slope) over 9 weeks. Temperature and pCO2 were offset from a reef slope baseline temperature of 26.0°C ± 0.6°C and pCO2 concentration of 559 ± 56 ppm, incorporating natural diurnal variability. A trade-off was observed in *I. palifera* originating from the reef flat, which significantly increased tissue protein densities but reduced densification relative to corals from the reef slope. Survivorship nor extension rates differed between corals originating from the variable or stable reef habitats. Mortality increased under RCP4.5 as extension rates increased, indicating that energetic investment in skeletal expansion becomes unsustainable under future climate stress. Increasing temperature and CO2 reduced calcification rates irrespective of the corals originating reef habitat suggesting with future climate change, exposure to greater environmental variability may not benefit coral calcification. These results demonstrate that tolerance to present-day abiotic variability does not necessarily infer resilience to moderate future ocean warming and acidification conditions.

Climate change imposes a profound threat to coral reef resilience as increases in ocean warming and acidification combine to cause frequent acute disturbances with impeded recovery trajectories. The resilience of a coral reef may be described as both resistance against disturbance induced degradation and the ability and speed at which the reef returns to its pre-disturbance state. Presently, acute disturbances are observed as impacts from cyclonic activity, which physically destroy reef structures, and marine heatwaves which cause mass coral bleaching, tissue mortality, and the rapid decay of exposed skeletons (Hughes et al. 2017; Leggat et al. 2019). Recovery from future disturbances will be dependent on the ability of the reef to return to a hard coral-dominated community and scleractinian corals to replace calcium carbonate (CaCO3) frameworks lost to acidification and erosion at a rate that exceeds the frequency of disturbance-related degradation (Eyre et al. 2018; Ortiz et al. 2018; Kline et al. 2019). By rebuilding reef frameworks, coral colonies contribute to integral economic and ecological reef services including providing physical protection to coastal communities and rugose habitats that support high biodiversity (Andersson and Gledhill 2013).

The abiotic conditions of the future are linked to anthropogenic CO2 emissions. Projections of future emission scenarios are categorized as representative concentration pathways (RCP) defined by the change in radiative forcing (Wm⁻²) between 1900 and 2100 (IPCC 2014). Under high (2.6 Wm⁻²) and moderate (4.5 Wm⁻²) levels of CO2 mitigation, expected increases in mean global sea surface temperature (SST) by mid to late century are 0.71°C ± 0.45°C and 1.28°C ± 0.56°C, which are each associated with mean changes in sea surface

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pH of \(-0.07\) (+84 ppm CO₂) and \(-0.15\) (+184 ppm CO₂) units, respectively (Bopp et al. 2013). Projections for thermal impacts are based on the intensity and persistence of an anomaly which accumulates above a regionally sensitive threshold temperature. The maximum monthly mean (MMM) SST (determined biweekly via satellite data collected between 1985 and 1993) +1°C has served as an effective threshold for determining the likelihood of a mass bleaching event especially when the intensity of the anomaly is multiplied by its longevity to form the concept of a degree heating week (DHW). Mortality under warming is less predictable, but fast-growing staghorn and tabular corals appear to be more susceptible to prolonged heat exposures (DHW > 4) than corals with thicker tissues that tend to have slower extension rates (Eakin et al. 2010). Above eight DHW, extensive mortality typically occurs across most coral taxa (Eakin et al. 2010; Hughes et al. 2018).

In the past, spring and early summer conditions have been associated with maximal periods of coral recovery following significant coral die-back (Díaz-Pulido et al. 2009). Performing experiments in this period therefore presents an opportunity to assess how coral calcification, in what should be the recovery periods of the future, is impacted under different scenarios when accompanied with ocean acidification. Calcification in corals involves two processes: expansion of the skeletal structure (primary calcification) and densification of existing structure (secondary calcification). Below threshold increases in temperature can increase coral extension rates (Anderson et al. 2017). Reduced calcification rates caused by ocean acidification often eventuate as reduced skeletal density rather than changes to skeletal expansion (Madin et al. 2012; Fantazzini et al. 2015; Tambutte et al. 2015), highlighting the potential decoupling between the two processes (primary calcification/extension vs. secondary calcification/densification) (Gladfeiter 1982). Reductions in skeletal density increase the susceptibility of deposited CaCO₃ to physical and biological erosion, such that rates of extension observable in a laboratory setting may not be realized in the field. Alternatively, at more protected reef locations, rapidly extending corals that sacrifice densification for growth would be highly sensitive to any projected increases in storm activity (Knutson et al. 2020).

To promote reef resilience under future climate conditions, corals of the future will need to calcify rapidly in cooler periods and survive through abnormally warm periods. Survival through past warming events is linked to hard corals that demonstrate reduced extension rates and greater tissue development (Glynn 1984). Likewise, corals persisting in abiotically variable environments often have thicker tissues (Salih et al. 1998; Thornhill et al. 2011), providing greater energy reserves to cope with high environmental stress (Grottoli et al. 2014; Schoepf et al. 2015). Selective pressures on coral growth require corals to display intraspecific differences in physiology depending on the abiotic conditions of their environment. Tidal ponding in shallow lagoon reefs can expose corals to high variability in seawater temperature and carbonate chemistry, conditions which are moderated on deeper reef slopes through greater water mixing and lower irradiance (Rivest et al. 2017; Cyronak et al. 2020). Rapidly growing corals contribute significantly to maintaining reef frameworks (Brown et al. 2021), however at the cost of greater environmental sensitivity because of their thinner tissues (Loya et al. 2001; Grottoli et al. 2014). It has thus been proposed that corals residing in naturally variable habitats may be more tolerant to future climate conditions and could provide population stocks for future reef management (Camp et al. 2017, 2019). Corals from the variable environment, however, can exhibit lower rates of calcification relative to conspecifics from the stable environment (Denis et al. 2013; Camp et al. 2016). Shifting from calcification to protein is clearly beneficial when corals approach their physiological thresholds and are lacking in energy resources (Denis et al. 2013; Tambutte et al. 2015; Camp et al. 2016). However, such a compromise may not be sufficient for corals to survive prolonged environmental driven resource limitations. In this respect, present evidence suggests that corals with the thickest tissues are nonetheless failing to survive extreme marine heatwaves (Hughes et al. 2018).

Persistence of corals in abiotically variable environments suggests some coral populations have locally adapted or acclimated to conditions that exceed regional stress thresholds (Bongaerts et al. 2010; Rivest et al. 2017). Corals from high variability niches have been found to exhibit greater tolerance to elevated temperature (Oliver and Palumbi 2011; Palumbi et al. 2014; Schoepf et al. 2015) and ocean acidification stress (Dufault et al. 2012; Comeau et al. 2014). Consequently, corals from variable environments have been proposed as candidates to acclimate to future climate conditions over their lifetime. Greater thermal tolerance in corals from highly variable environments has been evidenced by reduced mortality and/or bleaching, maintained photosynthetic efficiency and greater tissue biomass (Oliver and Palumbi 2011; Thornhill et al. 2011; Schoepf et al. 2015). However, most studies that have explored the concept of enhanced abiotic tolerance in corals from naturally variable environments have investigated elevated temperature and pCO₂ in isolation or under present-day in situ conditions. This raises the question of whether any physiological tolerance observed is maintained under the interactive effects of projected ocean warming and acidification. Alternatively, corals exposed to high variability may be nearing their physiological thresholds (Schoepf et al. 2015; Rivest et al. 2017) and exhibit a trade-off between survival and growth (Okazaki et al. 2013). As such, the ability of coral colonies to tolerate elevated environmental stress is not necessarily analogous or scalable to resilience of entire reef systems. Physiological trade-offs made by corals to persist in abiotically stressful environments may impact their contribution to reef resilience by reducing their rates of positive reef accretion.

This study aimed to determine whether *Isopora palifera* originating from two distinct reef habitats of Heron Island, southwestern Great Barrier Reef (23°27'S, 151°55'E) exhibit differential
tolerance to chronic temperature and acidification stress. Specifically, we investigated whether *I. palifera* originating from variable (reef flat) and stable (reef slope) environments exhibit a physiological trade-off between calcification (surface area [SA] extension and/or densification) and tissue protein production under future climate scenarios. On the reef flat (mean depth ~ 2 m) of Heron Island, tidal fluctuations in water depth cause intense diel variation in temperature, irradiance, and water carbonate chemistry (Georgiou et al. 2015; Brown et al. 2018), with limited exposure to mechanical forces (Harris and Vila-Concejo 2013). On the reef slope (mean depth ~ 5–8 m), lower irradiance and high flow rates provide stable abiotic conditions with minimal diel variability (Brown et al. 2018; Cyronak et al. 2020). Corals originating from the variable reef flat were predicted to increase their tissue protein reserves more than reef slope conspecifics but exhibit decreased rates of calcification. Habitat dependent differences in *I. palifera* survivorship were also explored across climate treatments. Given the timing of the experiment in late spring/early summer and the experimental offsets applied, it was hypothesized that mortality would only be observed under RCP4.5 toward the end of the experiment. Exploring climate-driven survivorship and physiological changes in *I. palifera* from distinct reef habitats provides insight into how origin dependent trade-offs may influence coral survival and the contribution of corals to reef resilience under future climate change.

**Methods**

**Variability in environmental conditions between habitats**

To explore variance in environmental conditions between the reef flat and reef slope, daytime photosynthetically active radiation (PAR) and seawater temperature were recorded continuously at two sites within the reef slope and three sites within the reef flat of Heron Reef (Table 1; Figs. 1, S1). In situ pCO₂ was collected by use of Conductivity Temperature Depth units (SBE 16 plus VS SEACAT) fitted with an auxiliary CO₂ sensor (Optical CO₂ sensor, AMT Analysemesstechnik GmbH) within the reef slope (*n* = 1).

| 2013 slope reference | Reef slope | Reef flat | Present day | RCP2.6 | RCP4.5 |
|----------------------|------------|----------|-------------|--------|--------|
| Temperature (°C)     | Mean 25.7±0.6 | 25.9±0.7 | 26.4±0.8 | 26.6±0.6 | 26.8±0.6 |
|                      | Q3 26.3±0.7  | 26.1±0.7 | 29.2±1.6  | 27.8±0.8 | 27.9±0.9  |
|                      | Q1 25.4±0.6  | 25.7±0.7 | 24.3±0.8  | 25.2±0.8 | 26.0±0.7  |
| Flux (μmol photons m⁻² s⁻¹) | Mean 0.9±0.4 | 0.3±0.2 | 4.8±1.6 | 1.7±1 | 1.8±1.1 |
|                      | Q3 408±12 | 480±74 | 430±54 | 559±56 | 584±52 |
|                      | Q1 429±22 | 646±115 | 657±103 | 739±66 | 766±57 |
|                      | Flux 39±20 | 327±149 | 439±136 | 306±53 | 308±51 |
| pH                   | Mean NA | 155±65 | 541±257 | 256±36 | 264±24 |
|                      | Q3 NA | 311±114 | 1060±479 | 652±93 | 627±24 |
|                      | Q1 NA | 11±19 | 35±16 | 10±2 | 11±2 |
|                      | Flux NA | NA | NA | 8.08±0.049 | 8.03±0.049 |

*Fig. 1.* Experimental timeline. Timeline of experiment, indicating the time of coral collection (yellow), acclimation, progressive treatment ramping (light blue), and 6 weeks of experimentation (dark blue) broken into two sampling periods (period A: 22nd Nov – 14th Dec; period B: 14th–26th Dec – 2nd Jan). Coral cohorts were sampled in the same order at each sampling period to allow for equal time between sampling for each cohort.

Table 1. Environmental metrics across treatments and habitats. Average daily mean, Q3, Q1, and flux (Q3–Q1) of temperature, daytime PAR (06:00–18:00) and pCO₂ on the reef flat and reef slope of Heron Island and within experimental treatments (± SD). *In situ pCO₂ data recorded during Nov–Dec 2015 from the Heron Island reef flat (*n* = 1) and reef slope (*n* = 1).
and reef flat ($n = 1$) over the same period (01 November–02 January), but in 2015–2016 (Table 1; Figs. 2, S1).

**Coral collection**

*I. palifera* coral fragments were collected in late October 2019 from the reef flat and reef slope of Heron Island. Corals on the reef flat were randomly collected near the border of the scientific zone to Shark Bay, encompassing an area of ~0.075 km$^2$ (Fig. S1). Reef slope corals were collected around Harry’s Bommie on the southeast reef slope (area ~0.005 km$^2$) (Fig. S1). Each coral fragment was collected from a different coral colony and a total of 154 fragments were collected from each habitat. Coral fragments, herein referred to as “nubbins,” were collected using a hammer and chisel. Nubbins used in the experiment were standardized to a length between 4 and 7 cm, with nubbins greater than 7 cm carefully cut to size with an electric brick saw. Ten nubbins per reef habitat were collected as an initial sample and measured for buoyant weight (BW), volume, and living tissue SA upon collection.

**DHW calculations**

Daily temperature anomalies, or “hotspots” ($HS_i$), were calculated as in Eakin et al. (2010); when daily SST ($T_i$) exceeded MMM + 1°C (MMM = 27.3°C for Heron Island); then the MMM was subtracted from $T_i$. 

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**Fig. 2.** Temperature and CO$_2$ conditions in situ and within experimental treatments over time at Heron Island. (a) Temperature (°C) recorded in situ on the reef flat and reef slope of Heron Island and (b) within experimental treatments. Dashed horizontal line represents the Heron Island coral bleaching threshold, defined as one degree above the MMM temperature (+1°C; 28.3°C). (c) CO$_2$ (ppm) recorded in situ on the reef flat and reef slope of Heron Island in 2015 and (d) within experimental treatment sumps over the course of the experiment. Points indicate individual measurements, with lines and ribbons representing 24-h means (±SE). The solid vertical lines indicate the achievement of full treatment conditions (22 Nov 2019) and the date between the two sampling periods (4 weeks exposure to full treatment; 19 Dec 2019).
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HS\textsubscript{t} = T\textsubscript{j} (≥ 28.3°C) – 27.3, defined for HS\textsubscript{t} ≥ 1

Daily HS\textsubscript{t} were then summed across previous experimental days (e < 43) and divided by seven to determine the evolution of thermal stress in degree heating weeks (DHW\textsubscript{i}) over the experimental period:

\[
\text{DHW}_i = \frac{1}{n-i} \sum_{n=i}^{j} (\text{HS}_n - \frac{27.3}{7})
\]

Future SST scenarios do not currently hypothesize a reduction in SST variability. Daily mean and daily Q3 temperatures were therefore used separately as inputs to calculate mean-DHWs and Q3-DHWs respectively. Mean-DHW then captures average daily stress with Q3-DHW capturing stress associated with the upper quartile of the daily SST variability.

**Experimental design**

Temperature and pCO\textsubscript{2} conditions were manipulated using a flow-through ocean warming and acidification simulation system, described in detail by Dove et al. (2013). Briefly, this system was comprised of three treatment sumps that continuously drew seawater from the Heron Island reef flat via the Heron Island Research Station holding tank (Fig. S2). Very large sumps (10,000 liters) maintained in the dark combined with high flow rates were used to effectively eliminate any interaction between the sump walls and the bulk of the water that passes through them (i.e., differential confinement effects) (Schoepf et al. 2013; Cornwall and Hurst 2015). Diel variation of temperature and pCO\textsubscript{2} were controlled to simulate natural variation in bi-hourly measurements recorded at the Pacific Marine Environmental Laboratory (PMEL) MAPCO\textsubscript{2} ocean observation buoy near Harry’s Bommie (https://www.pmel.noaa.gov/co2/story/Heron+Island) in 2013. Conditions in each sump were manipulated using a computer-controlled feedback system that responded to conditions measured in experimental aquaria (SCIWARE Software Solutions) by injection of air enriched to 30% CO\textsubscript{2} or CO\textsubscript{2}-free air (Sparosorb soda lime, Intersurgical) and the use of industrial-scale heater chillers (Rheem HWPO17-1bb; Accent Air equipped with Eurotherm 3216 temperature regulators, Invensys Process Systems and CO\textsubscript{2}-Pro CO\textsubscript{2} regulators, Pro-Oceanus with an accuracy of ± 0.5% CO\textsubscript{2} concentration). Sump, as opposed to tank, pCO\textsubscript{2} was controlled and monitored to allow organisms and their biology to influence in-tank pCO\textsubscript{2}. Manipulated treatment seawater was then pumped from the sumps into experimental tanks (Fig. S2). Within the tanks, seawater pH was measured at 08:00 and 20.00 h daily using a pHep sensor (HI 98128, Hanna Instruments), calibrated every other day (pH\textsubscript{sw} buffers 4.00 and 10.00) (Table 1). Three tanks per treatment were randomly selected for pH measurements on each sampling occasion.

Corals were exposed to three distinct treatments: (1) present-day temperature and pCO\textsubscript{2} of conditions measured during 2013 on the reef slope (Table 1), (2) RCP2.6 temperature and pCO\textsubscript{2} (set as +0.5–1°C and +50–100 ppm above 2013 conditions), and (3) RCP4.5 temperature and pCO\textsubscript{2} (set as +1.5–2°C and +150–200 ppm above 2013 conditions). In the first week of acclimation, temperature, and pCO\textsubscript{2} were maintained at present-day treatment levels across all tanks (Fig. 1). Over the following 2 weeks, temperature and pCO\textsubscript{2} were increased at quarter increments to treatment levels (Fig. 1). Exposure of corals to full treatment conditions occurred for 6 weeks. During the experiment water flow rates were maintained between 1.0 and 1.6 L min\textsuperscript{-1} and wavemakers (Nano 900, Hydor) continuously circulated tank water. Seawater temperature (HOBO pendant logger) and PAR (Odyssey PAR sensor) were continuously measured in each treatment by randomly rotating two probes per treatment between tanks (Table 1; Fig. 2).

A total of 24 experimental tanks (n = 8 per treatment) were randomized across two outdoor tables (Fig. S3) and evenly spaced to minimize light variability. Tanks and lids were covered with filters (Marine Blue #131, Lee Filters) to mimic the light environment of the reef slope collection site (Dove et al. 2020) (Table 1). Six coral nubbins, ramets from distinct colonies, from the same habitat were randomly suspended using nylon fishing line from a bamboo stick, with two bamboo sticks (one from each habitat) placed in each tank. Each bamboo stick was considered a cohort, with each cohort rotated into an adjacent tank of the same treatment every third day (Fig. S3). This was done to minimize any effects of environmental differences between tanks. “Cohort,” rather than “tank,” therefore represents the random effect within this experimental design. Throughout the experiment, tank surfaces, bamboo sticks, and fishing line attached to the corals were cleaned daily and epilithic algae growing over exposed coral bases was carefully removed using forceps.

**Physiological analyses**

Mortality of corals was tracked throughout the experiment and defined as 100% tissue mortality (Fig. 3). Coral nubbins were measured for their BW and living tissue SA three times throughout the experiment: (1) at the end of acclimation, (2) after 4 weeks of exposure to full treatment conditions (beginning 22 November 2019), and (3) after 6 weeks of exposure to full treatment conditions (beginning 15 December 2019) (Fig. 1). It took ~ 2 weeks to collect the BW and SA data, but there is no overlap between sampling periods as the order in which organisms were sampled meant that there was equal time between sampling for each cohort. The BW method was used to assess net calcification (Davies 1989), as it incorporates the rate of coral calcification and bioerosion by internal bioeroders. BW was not adjusted to a dry-weight equivalence as this assumes a skeletal density (Comeau et al. 2014). Instead, the change in living SA was adopted as a proxy for coral extension (primary calcification). Changes in BW for statistically fixed SA changes was adopted as a proxy for change in
skeletal density (secondary calcification), driven either by reduced coral densification or increased skeletal erosion. Living tissue SA was estimated from photographs of the left- and right-hand side (LHS and RHS) and bottom surface of each coral, using image processing software ImageJ (National Institute of Health) (Fig. S6). The relative changes in coral BW and living tissue SA were calculated over each sampling period (Fig. 1). Relative change accounts for size-dependent differences in coral growth rates, by standardizing increases in growth as a percentage of the corals initial BW or SA. Growth changes were also standardized to the number of days between sampling for each coral cohort. Herein, the relative change in BW and SA per day will be expressed as %ΔBW d⁻¹ and %ΔSA d⁻¹.

Relative change \(\left(\%\Delta \text{d}^{-1}\right) = \frac{(M_{\text{final}} - M_{\text{initial}})}{M_{\text{initial}}} \times 100\) \(\div\) no. days

where \(M_{\text{final}}\) is the final and \(M_{\text{initial}}\) is the first measurement taken for each sampling period and no. days is the total number of days between the first and final measurements.

At the end of the experiment, nubbins were frozen in liquid nitrogen and stored at \(-80^\circ\text{C}\). A subset of two corals per cohort (totaling 48 corals, \(n = 8\)) were water-piked to remove coral tissue using 50 mL of 0.06 M phosphate buffer saline solution. Centrifugation for 5 min at 4500 rpm was used to separate host tissue and endosymbiotic dinoflagellates. Tissue protein concentrations were determined using protein spectrophotometry methods.

Fig. 3. Physiological characteristics of \(I.\ palifera\) between treatments over the experiment. (a) Recorded survivorship of corals by treatment over time. (b) Representative images of the progression of \(I.\ palifera\) mortality. Corals were first observed to pale in color (left), become progressively covered in algae (middle) and ultimately experience full tissue mortality and total algal coverage (right). (c) Changes in living tissue surface area (mean ± SE) of \(I.\ palifera\) between climate treatments and sampling periods. (d) Changes in protein densities (mean ± SE) of \(I.\ palifera\) between climate treatments and habitats.
outlined by Whitaker and Granum (1980). Total branch proteins (mg) were standardized to the wax-dipped coral SA to determine the protein density per unit of living tissue SA (mg cm\(^{-2}\)) (Holmes 2008). Relative changes in protein densities (\(\% \Delta \text{protein d}^{-1}\)) were calculated using the average of the initial samples as a baseline measurement.

\[
\text{Protein density (\% \Delta \text{protein d}^{-1}) = \left(\frac{P_{\text{final}} - P_{\text{initial}}}{P_{\text{initial}} \times 100}\right) \div \text{no. days}}
\]

where \(P_{\text{final}}\) is the protein density measured in corals after 6 weeks of experimentation and \(P_{\text{initial}}\) is the average protein density of the initial samples sacrificed at the beginning of the experiment.

**Statistical analysis**

All statistical analyses were conducted using R version 3.5.3 software (R Core Team 2021). The daily means, upper quartile (Q3), and flux (Q3-Q1) of seawater temperatures, pCO\(_2\), and PAR were analyzed for differences within and between experimental treatments (present-day [PD], RCP2.6, and RCP4.5) and reef habitats (reef flat and reef slope), with the logger location (tank or site) included as a random effect (Table S1). Survivorship was explored using a generalized linear mixed effect model with a binomial distribution using the function ‘glmer’ within the lmer4 package (Bates et al. 2018) and initially included the interaction of treatment, habitat and sampling period, with PAR as a covariate and cohort as a random effect (Table S1). The interactive effects of treatment, reef habitat, and sampling period on changes in I. palifera living tissue SA (\(\% \Delta \text{SA d}^{-1}\)), calcification (\(\% \Delta \text{BW d}^{-1}\)), and protein density (\(\% \Delta \text{Protein d}^{-1}\)) were analyzed using linear mixed effects (lme) models (nlme package; Table S1) (Pinheiro et al. 2017). Physiological trade-offs in secondary calcification (skeletal densification) were then explored by fixing SA as a covariate in the analysis of calcification. To explore the interactive effects of temperature and pCO\(_2\) on densification, we substituted treatment for the interaction between mean temperature and mean pCO\(_2\), while maintaining reef habitat and SA (Table S1). To establish an initial protein density, protein densities were determined for samples collected prior to experimentation and analyzed for differences between reef habitats. Physiological trade-offs between protein production and densification between habitats and treatments were explored using living tissue SA as a covariate.

For all models, the “Anova” function in car was used to determine the significance of fixed effects and their interactions, with type II error structures applied for models that were not suggestive of interactions, and type III for models that were (Fox et al. 2012). Significant interactions were explored by pairwise comparison of estimate marginal means using the emmeans package (Lenth et al. 2018), with Tukey HSD adjusted \(p\) values. Model selection was conducted using the function “stepAIC” in the package MASS with the maximum likelihood (ML) method (Ripley et al. 2013). The best fit model was then run using the restricted ML method. All variables were tested for collinearity using the “vif” function in the package car (Fox et al. 2012) and models were tested for homogeneity of variance and normality of distribution through graphical analyses of residual plots. Models \(R^2\) were determined using the function “r.squaredGLMM” from the package MuMIn (Barton and Barton 2015).

**Results**

**In situ and experimental conditions**

Diurnally variable treatment conditions were maintained throughout the duration of the experiment (Figs. 2, S4). PD temperature conditions were +0.3°C above the 2013 baseline (26.0°C ± 0.6°C), with RCP2.6 (26.8°C ± 0.6°C) and RCP4.5 (27.3°C ± 0.5°C) maintained significantly higher than PD at 0.8°C and 1.3°C, respectively (\(\chi^2 = 252, p < 0.0001\)) (Table 1, S2; Fig. 2). Mean temperatures did not exceed Heron Island’s coral bleaching threshold (MMM + 1°C) of 28.3°C in any treatment (mean DHW = 0). Q3 temperatures, however, were significantly different among treatments and with PD, RCP2.6, and RCP4.5 treatment experiencing Q3 DHWs less than 1°C, 4°C, and 8°C week\(^{-1}\), respectively (\(\chi^2 = 548, p < 0.0001\)) (Fig. 2; Table S2). Thermal variability (flux) did not differ among treatments (Table S2).

Mean temperatures recorded concurrently in situ revealed that the reef flat (26.4°C ± 0.8°C) was 0.5°C warmer than the reef slope (25.9°C ± 0.7°C) \((p < 0.0001)\) (Table 1; Fig. 2). Mean daily temperature did not exceed the bleaching threshold (mean DHW = 0). Q3 temperatures measured within the reef slope were equivalent to the 2013 baseline and did not exceed the regional bleaching threshold during the experimental period (Q3 DHW = 0). Q3 temperatures measured within the reef flat, however, accumulated Q3 DHW < 2. Thermal variability (flux) was significantly greater on the reef flat (4.8°C ± 1.6°C) than within the reef slope (0.3°C ± 0.2°C) \((\chi^2 = 765, p < 0.0001)\) (Tables 1, S2; Fig. 2).

Unstable power supply and subsequent research station power cuts meant that targeted pCO\(_2\) levels where not achieved within the experimental sumps, although differences between treatments were achieved (Table 1). Daily mean pCO\(_2\) was significantly different between treatments and reef habitats (\(F = 113.64, p < 0.0001\)), with PD and RCP2.6 treatments significantly lower than RCP4.5 treatment and all treatments significantly higher than the reef flat (430 ± 54 ppm) and reef slope (480 ± 74 ppm) (Tables 1, S3; Fig. 2). The mean upstream pCO\(_2\) conditions were 559 ± 56 ppm, 584 ± 52 ppm, and 625 ± 56 ppm for PD, RCP2.6, and RCP4.5 treatments across the experimental period (Table 1). CO\(_2\) variability (flux) was constant across treatments but not locations, with the reef flat (439 ± 136 ppm) significantly greater than reef slope (327 ± 149 ppm) and the 2013 reef...
slope reference (39 ± 20 ppm) \( (F = 88.6, p < 0.0001) \). Mean daytime (06:00–18:00) PAR was significantly higher on the reef flat \((541 \mu \text{mol quanta m}^{-2} \text{s}^{-1})\) than reef slope \((155 \mu \text{mol quanta m}^{-2} \text{s}^{-1})\) \( (\chi^2 = 252, p < 0.0001) \), however, did not differ across experimental aquaria \((- 250 \mu \text{mol quanta m}^{-2} \text{s}^{-1})\) (Tables 1, S4; Fig. S5).

**Coral survivorship and growth characteristics**

Coral survivorship was influenced by the individual effects of treatment \( (\chi^2 = 14.56, p < 0.0007) \) and sampling period \( (\chi^2 = 7.35, p = 0.007) \) (Table S1). Coral nubbins exposed to RCP4.5 treatment conditions experienced a 21% and 15% increased risk of mortality relative to PD and RCP2.6 treatments, respectively (Table S5). Nubbin mortality risk was additionally 15% greater in sampling period B than period A (Table S5). Approximately 19% of the variation in nubbin survival was explained by the fixed effects of treatment and period, with reef habitat nonsignificant and eliminated as a predictor from the best fit model (Tables S1, S5).

Following the removal of dead nubbins from the data, coral extension \((\% \Delta \text{SA d}^{-1})\) was best explained by a model that included the interaction between treatment and period \( (\chi^2 = 6.96, p = 0.036) \) (Tables S1, S6). Extension rates were equivalent \((- 0.35\% \text{ d}^{-1})\) among treatments in period A but increased to \(- 0.58\% \text{ d}^{-1}\) with increasing temperatures in period B \((\text{PD} < \text{RCP4.5})\) (Figs. 3, S6). Again, reef habitat did not add any explanatory power to this model and observations of greater surface areal expansion in living corals correlated with an increase in mortality risk in Period B under RCP4.5 (Fig. 3; Tables S1, S6).

There was no habitat difference in the SA of initial samples collected from the field and trimmed to size \((t\text{-test}, p = 0.69)\), but the BW differed \((t\text{-test}, p < 0.02)\), with reef flat corals

**Fig. 4.** Graphical representation of model outputs for the variation in calcification of *I. palifera*. (a) Calcification \((\text{mean} \pm \text{SE})\) was greater in *I. palifera* originating from the stable reef slope compared to corals originating from the highly variable reef flat. (b) Positive correlation between calcification and living tissue surface area change. Points represent individual coral nubbins, and solid line and shaded ribbon represent mean \(\pm\) SE. (c) The interactive effect of mean temperature and mean pCO₂ \((\text{ppm}; \text{boxed values})\) on calcification \((\text{mean} \pm \text{SE})\), where progressive increases in pCO₂ resulted in an increasingly negative relationship with temperature.

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having a reduced BW/SA ratio relative to reef-slope native corals. The best fit model for variance in BW (%ΔBW d\(^{-1}\)) included the covariates origin (\(\chi^2 = 23.77, p < 0.0001\)), SA (%ΔSA d\(^{-1}\)) (\(\chi^2 = 43.58, p < 0.0001\)), and the interaction between treatment and sampling period (\(\chi^2 = 25.30, p < 0.0001\)) (Tables S1, S7). Post hoc analyses revealed corals originating from the reef flat densified significantly less than those native to the reef slope (for an extension rate fixed at 0.43% d\(^{-1}\)) and incurred a further reduction in skeletal density under RCP4.5 in sampling period B compared to PD and RCP2.6 treatments (\(\chi^2 = 25.30, p < 0.0001\)). This model explained 30% of the variance (Table S7). The specific effects of mean temperature and mean pCO\(_2\) between sampling time points were also examined by substituting continuous abiotic variables for the categorical predictor of treatment (Table S1). This model explained 35% of the variance in secondary calcification rates, with %ΔSA d\(^{-1}\) (\(\chi^2 = 25.66, p < 0.0001\)) and reef habitat (\(\chi^2 = 21.93, p = 0.0001\)) maintaining their statistical significance, together with the interaction between mean temperature and mean pCO\(_2\) (\(\chi^2 = 22.39, p < 0.0001\)) (Fig. 4; Tables S1, S8). The positive relationship found between changes in BW and SA supported the assumption that increases in living SA coincide with increased CaCO\(_3\) contributions to skeletal extension (Fig. 4b). The gradient of %Δ(BW) d\(^{-1}\) to %Δ(SA) d\(^{-1}\) was 0.0079 ± 0.0016 suggesting that over Period A coral extension added ~0.003%(%Δ(BW) d\(^{-1}\)) but that over Period B and under RCP4.5, extension added ~0.005%(%Δ(BW) d\(^{-1}\)). At mean pCO\(_2\) concentrations of 530 ppm, increasing temperature from 25.5°C to 27.5°C had little effect on skeletal density, but as mean pCO\(_2\) increased to 680 ppm, there was a decrease in density that was equivalent to ~0.015%(%ΔBW) d\(^{-1}\) C\(^{-1}\) when corals extended at equivalent rates (Fig. 4c). At 27.5°C and 680 ppm, reef flat and reef slope corals are therefore contributing 1.2-fold (0.006 : 0.005) or 2.6-fold (0.013 : 0.005) more CaCO\(_3\) to densification than to extension, respectively.

Within the initial sample, absolute coral protein densities differed significantly between reef habitats, with higher protein densities found in reef slope (~1.59 mg cm\(^{-2}\)) relative to reef flat corals (~1.05 mg cm\(^{-2}\)) (\(F_{1,18} = 9.89, p < 0.006\)) (Tables S1, S9). Relative to these initial values and irrespective of coral extension rates, protein densities only increased in corals from the reef flat with the greatest increase occurring in the RCP4.5 treatment (Treatment × Habitat: \(\chi^2 = 7.37, p < 0.03\); reef slope\(_{PD}\) < reef flat\(_{PD}\) < reef flat\(_{RCP4.5}\)) (Tables S1, S10). Observed percent increases in protein density (~1.5% d\(^{-1}\)) were, however, only equivalent to an absolute increase in protein density of 0.68 mg cm\(^{-2}\) over the 43 experimental days. Climate treatments were not found to have any significant effects on relative change in protein densities (Table S1).

**Discussion**

This study investigated whether pre-exposure to high abiotic variability in *I. palifera* infers tolerance to future, albeit mitigated, ocean warming, and acidification conditions. Evidence of an origin dependent physiological trade-off between secondary calcification and tissue protein production was found in *I. palifera* originating from the highly variable reef flat environment. Reef flat corals densified their skeletons at a slower rate, but increased their tissue protein densities significantly more than reef slope conspecifics, irrespective of climate treatment. Previous literature suggests increased tissue mass facilitates coral survival under environmental stress (Thornhill et al. 2011; Okazaki et al. 2013; Schoepf et al. 2015). In this study, despite increasing protein densities, reef flat *I. palifera* did not experience greater survivorship to future temperature and pCO\(_2\) conditions. This result does not support the theory that exposure to present-day variability preconditions corals to tolerate future climate change. Coral mortality correlated with increased rates of extension, as opposed to increases in protein densities, supporting the observation that future reefs may transition to slow-growing coral communities. Our results also indicate that increasing mean temperatures (up to 27.5°C) acted synergistically with increasing CO\(_2\) (up to 680 ppm), significantly impacting secondary calcification rates of *I. palifera*. As climate conditions elevate toward RCP4.5, our findings suggest corals will experience greater mortality, irrespective of native habitat, and surviving corals originating from variable habitats will have weaker skeletons despite the outward appearance that calcification is unaffected.

In this study, we did not find support for the concept of climate pre-conditioning in corals by exposure to present-day variability. Increased mortality was observed in conjunction with RCP4.5 conditions at the maximum exposure of 6 weeks, regardless of whether corals originated from the highly variable reef flat or comparatively stable reef slope. As mortality increased, late experiment RCP4.5 conditions also led to 1.6-fold increase in SA extension rates for surviving coral nubbins irrespective of habitat origin—an expansion that has previously been observed in corals responding to thermal increases within their physiological range (Anderson et al. 2017). Rapid skeletal expansion requires energetic investment in new skeleton and tissue which can become a liability in environments where energy acquisition is impeded (Anthony et al. 2008). The coincidence of high mortality with increased coral extension rates is in line with observations of community shifts following intense underwater heatwaves to slow growing species (Glynn 1984; Eakin et al. 2010; Hughes et al. 2018). Typically, survival is attributed to greater tissue biomass and resource availability (Grottoli et al. 2014), but energetically, corals with reduced expansion rates can maintain tissue biomass.

The experimental conditions released nubbins from space limitations that potentially inhibit reef flat coral expansion in the field (Chen et al. 2018). Despite no difference in nubbin survival or extension rates, conspecifics did not converge on the same physiological responses to their new experimental
regimes. Rather, corals native to the reef flat responded in a manner that suggests that they were at, or past, their physiological limits with tissue biomass favored over skeletal biomass (Okazaki et al. 2013). By contrast, corals native to the reef slope maintained an unchanging protein density and a greater densification rate for the same growth rate, suggesting that for much of the experiment they were not subject to energy limitations. The physiological status of corals in October when they were collected from the field was different to expectations. Previous work suggests that corals native to the variable habitats should have greater tissue biomass (Oliver and Palumbi 2011; Denis et al. 2013; Schoepf et al. 2015). However, initial protein densities (and skeletal densities) were significant less in I. palifera reef flat populations, despite their experimental tendency to add protein biomass with increased levels of warming. Schoepf et al. (2015) interpret an observation of reduced tissue biomass in Acropora in heat stressed intertidal (variable) vs. subtidal (stable) natives as a greater ability to access energy reserves. In this context, I. palifera from the reef flat may always be operating under an energy deficit. Indeed, mass coral bleaching (> 60% of corals) was observed on the Heron Island reef flat 1 month following the completion of this study in February 2020 (Ainsworth et al. 2021). Given the timing of the experiment in late spring/early summer and the experimental offsets applied, coral bleaching was minimal; however, measurements of bleaching metrics (e.g., symbiont densities) to determine if the experimental treatments led to any significant changes in symbiotic community and physiology could be incorporated into future studies.

By end of experiment, under RCP4.5 mean experimental conditions, I. palifera had replenished tissue biomass to levels present in corals native to the reef slope across all treatment. This replenishment did not lead to greater thermal tolerance to upper quartile stress (7.25 Q3 DHW) under RCP4.5 by end of experiment. In contrast, Oliver and Palumbi (2011) found corals from abiotically variable lagoon pools experienced lower mortality (17%) compared to corals from abiotically moderate pools (50%) when exposed to temperatures 2.2°C above Ofu, American Samoa MMM for 4–5 d (≤ 1 mean DHW, or estimated Q3 DHW < 3 based on Q3 anomaly of +3.2°C in heat treatment). Similarly, corals from variable intertidal habitats experienced 50–58% mortality, relative to 75% mortality in stable subtidal corals, when exposed to temperatures 2–3°C above long-term mean summer SST (~ 1.5°C above MMM + 1; estimated < 2.4 mean DHW) (Schoepf et al. 2015). In these previous experiments, corals were exposed to high temperatures for 4–5 (Oliver and Palumbi 2011) and 11 d (Schoepf et al. 2015). Potentially, such treatments are more akin to a relatively sudden (acute) temperature shock that more closely mirrors sudden exposure to ponding temperatures over very low tidal cycles rather than gradual stress that are allowed to accumulate slowly as spring transitions to summer under future scenarios. For shorter periods of stress, tissue biomass provides a finite reservoir of energy that is unlikely to last over longer periods. Another confounding factor is the interactive effects of elevated temperature and CO2 on coral survivorship. While Oliver and Palumbi (2011) and Schoepf et al. (2015) did not investigate elevated temperature and CO2 in combination, ocean acidification has been found to act synergistically with warming to lower corals stress thresholds (Anthony et al. 2008; Dove et al. 2013; Dove et al. 2020), potentially by increasing respiratory demands on the host (Kaniewska et al. 2015). The synergistic effects of high temperature and CO2 conditions in this study may account for the lack of difference in survival between corals pre-exposed to environmental stress and corals lacking a stressful environmental history. However, the most likely explanation is that preconditioning to SST flux (hours to days) does not equip corals to survive chronic thermal stress (weeks to months) associated with climate change.

Reduced secondary calcification rates under RCP4.5 conditions during the second sampling period was attributed to the significant interaction of temperature and CO2 on calcification. This is significant as some laboratory studies have suggested that CO2 variability may mitigate the impacts of ocean acidification (Dufault et al. 2012; Comeau et al. 2014; Enochs et al. 2018). However, in most of these previous studies, the mitigating effect of variability on ocean acidification was relative to constant exposure to high CO2 (Dufault et al. 2012; Comeau et al. 2014; Enochs et al. 2018). In our study, the interaction of naturally variable temperature and pCO2 negatively impacted coral calcification rates (Fig. 4). Similar observations were made in Reynaud et al. (2003), where calcification rates of Stylophora pistillata were maintained at elevated temperatures (28.2°C) but declined by 50% with exposure to high pCO2 (800 μatm). Anthony et al. (2008) also found Acropora intermedia exposed to warm (28–29°C), intermediate CO2 (520–700 ppm) conditions had a 30% increase in productivity relative to the control (25–26°C and 380 ppm), whereas warm, high CO2 (1000–1300 ppm) conditions resulted in declined productivity. Overall, these findings suggest that in isolation, increases in temperature below a region’s thermal threshold conditions may even benefit coral calcification and expansion; however, synergistically even moderate increases in temperature and CO2 conditions may significantly reduce calcification rates.

The results of this study have provided evidence of physiological trade-offs in I. palifera dependent on the abiotic conditions of their native environment and have illustrated the potential for moderate increases over environmental thresholds to negatively impact calcification in I. palifera. Corals pre-exposed to high abiotic variability exhibited equivalent rates of mortality under future climate conditions. Our findings did not support the theory that pre-exposure to natural variability preconditions corals to tolerate future climate change. Relatively high rates of survival in I. palifera observed here may be attributed to the corals slow growth rate or moderate climate treatments investigated. Even though our study
spanned 9 weeks, future studies could be conducted over longer time frames, during peak heat stress (mid to late summer), and across reef habitats to better understand the interactive effects of chronic climate stress on top of short-term variability on coral physiology and future reef acclimation. Reduced densification in *I. palifera* from variable reef flat habitats relative to reef slope may be a result of reef flat corals prioritizing tissue protein production in order to survive a highly stressful environment. This suggests that corals originating from variable environments may be better survivors within reef habitats with limited variability (e.g., reef slopes), however, at a cost to skeletal density. Irrespective of reef habitat, however, the capacity for corals to survive and maintain high rates of calcification will be reduced with moderate ocean warming and acidification.

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Conflict of Interest
None declared.

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