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Coral defences: the perilous transition of juvenile crown-of-thorns starfish to corallivory

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ABSTRACT: The transition from the post-settlement herbivorous juvenile to the coral-eating stage of crown-of-thorns starfish (COTS) is a fundamental step to seed population outbreaks that decimate tropical coral reefs. How the highly cryptic juveniles fare during this transition is poorly understood. We show that the juveniles are vulnerable to attack by coral during this ontogenetic diet shift to coral prey. We monitored the condition, growth, and survival of juvenile COTS during the first 3.5 mo on a diet of *Acropora* sp. In initial encounters, juveniles often withdrew their arms to avoid the defensive nematocysts of the corals. Within the first 67 d of being offered coral, 37.8% of the juveniles experienced various levels of sublethal and lethal damage. Damaged arms were reduced to ~65.4% of the length of an intact arm, but most injured juveniles were able to regenerate their arms with an average predicted recovery time of ~4 mo. Although sublethal damage slowed the growth of injured juveniles, their capacity to regenerate is likely to contribute to the success of this highly prolific species. Despite being the prey of COTS, coral can influence the survival of juveniles, and potentially reduce their ecological impact by prolonging their growth to reproductive maturity, delaying their transition into a coral predator, and thereby hindering recruitment into the adult population.

KEY WORDS: *Acanthaster* sp. · Sublethal injury · Lethal injury · Arm regeneration · Ontogenetic diet shift · Juvenile sea star

1. INTRODUCTION

Echinoderms are among the most ecologically influential species in coral reef ecosystems (Foster 1987, Birkeland 1989b, Wolfe et al. 2018). Species with boom-and-bust population cycles often act as an intermittent disturbance that alters the structure of benthic communities (Uthicke et al. 2009). For tropical reefs across the Indo-Pacific, the corallivorous crown-of-thorns starfish (COTS) *Acanthaster* sp. is the most influential of these. At low population densities, COTS can increase coral diversity (Done & Potts 1992), while in their high-density phase, they cause mass coral mortality (Chesher 1969, Endean & Stablum 1975, Leray et al. 2012, Roche et al. 2015). Outbreaks of COTS threaten critical coral habitat in already imperilled tropical reefs (Wakeford et al. 2008, De'ath et al. 2012, Kayal et al. 2012).

Despite years of research, the causes of COTS population outbreaks remain a complex and largely unresolved problem (Pratchett et al. 2017). A number of hypotheses attempt to explain the rapid proliferation of COTS, with a focus on the survival of early life history stages. Temporal pulses of increased larval survival caused by enhanced phytoplankton food levels due to anthropogenic nutrient runoff are considered

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to be one of the main drivers of outbreaks (nutrient runoff hypothesis) (Birkeland 1982, Brodie et al. 2005, 2017, Fabricius et al. 2010). However, COTS larvae are also resilient to the nutrient-poor conditions typical of coral reefs (larval resilience hypothesis) (Olson 1987, Wolfe et al. 2015a,b), and the juveniles can withstand food scarcity for extended periods of time during their initial herbivorous phase (juveniles-in-waiting hypothesis) (Deaker et al. 2020a,b). Juvenile COTS may remain in the herbivorous phase for a prolonged period, with the potential to build up in numbers over multiple years before giving rise to an outbreak (Deaker et al. 2020a, Wilmes et al. 2020).

The overfishing hypothesis posits that the loss of COTS predators has caused unchecked population expansion (Endean 1969). This hypothesis is supported by evidence that marine protected areas with a more intact trophic structure are less impacted by outbreaks (Sweatman 2008, Mellin et al. 2016, 2019, Vanhatalo et al. 2017). More than 80 species of fish and invertebrates prey on the gamete, larval, juvenile, and adult stages of COTS (Cowan et al. 2017), and COTS DNA has been found in the stomachs of 18 species of reef fish, suggesting that they are an integral part of the food chain (Kroon et al. 2020). A number of traits indicate that predation has been a significant evolutionary selective force on COTS, including the production of eggs and larvae with saponin toxins (Lucas et al. 1979), cryptic colouration, venomous spines, and nocturnal foraging behaviour (Zann et al. 1987). Thus, both anthropogenic eutrophic pollution and overfishing may offer explanations as to why outbreaks occur (Moran 1988, Pratchett et al. 2014, Condie et al. 2018).

As with other benthic marine invertebrates, the early post-settlement stages of COTS experience high rates of mortality and are particularly vulnerable to predation within the first days to months after settling (Keesing & Halford 1992b, Hunt & Scheibling 1997, Wilmes et al. 2018). Their small size and limited mobility heighten their risk to predator damage and ingestion compared to larger subadults and adults. A population of juvenile COTS (20–120 mm diameter, Ø) on a reef in Fiji had 99% mortality attributed to disease and predation (Zann et al. 1987). Predation was the primary reason for high mortality rates (0.45–9.6% d^{-1}) of post-settlement juveniles (0.6–13 mm Ø) in *in situ* experiments (Keesing & Halford 1992a, Keesing et al. 1996, 2018). As the post-settlement stages of COTS are highly cryptic and difficult to detect within their reef rubble habitat, predatory attacks have not been witnessed. Consequently, their natural predators are poorly understood and are considered to be predominately mobile benthic invertebrates (e.g. polychaetes and crustaceans) (Keesing et al. 2018, Balu et al. 2021) rather than fishes (Sweatman 1995, Cowan et al. 2017).

Interactions between COTS and their predators are not always lethal, with evidence of sublethal predation indicated by partially removed arms (McCallum et al. 1989, Messmer et al. 2017, Balu et al. 2021). As is typical of stellate sea stars, COTS are well known for their ability to recover from trauma such as arm damage and loss due to their extensive capacity for regeneration (Lawrence 1992, Byrne et al. 2019, 2020). Sublethal damage, indicated by the presence of wounds and damaged arms, is commonly reported in sea star populations (Lawrence & Vasquez 1996, Lawrence et al. 1999). It appears COTS are particularly susceptible to arm loss compared with sympatric sea stars (e.g. *Linckia* and *Culcita*) because their body wall is less calcified and so is soft and fragile (Birkeland 1989a). Depending on the reef and COTS density, body size, and sex, observations of arm damage in populations vary between 2 and 83% (Pratchett et al. 2014, Messmer et al. 2017, Budden et al. 2019). The presence of damaged and regenerating arms provides lasting evidence of sublethal predation until the arm has fully recovered (McCallum et al. 1989, Rivera-Posada et al. 2014).

The extent, cause, and impact of lethal and sublethal predation during the juvenile stage of COTS are poorly understood. Variation in the severity of injuries and recovery and regeneration time is likely to affect the timing of the ontogenetic diet shift of the juveniles from herbivory to corallivory, a key bottleneck for recruitment into adult populations (Deaker et al. 2020a). A high percentage (91.3%) of juveniles (6–44 mm Ø) with damaged arms were observed in field surveys in the Great Barrier Reef, indicating high rates of sublethal predation (Wilmes et al. 2019). The greatest incidence of arm damage was reported for 6 to 18 mm Ø juveniles, the size range that they transition to coral (Wilmes et al. 2019). This is a vulnerable phase for the juveniles as they move from sheltered coralline algae habitat to more exposed coral branches (Zann et al. 1987). Coral itself is a risky venture for juvenile COTS. The tentacles and outer tissue of coral polyps contain nematocysts, stinging cells that are reported to attack COTS and cause damage to the arms and central disk of the juveniles (Yamaguchi 1974, Birkeland & Lucas 1990).

The shift from herbivory to corallivory is a key transition for COTS, as corallivory fuels rapid growth to

reproductive maturity and the outbreak population phase (Lucas 1984, Deaker et al. 2020a). However, little is known about how the juveniles fare during this transition. We investigated the potential that coral may be a leading cause of sublethal and lethal damage for juvenile COTS. We characterised the initial interactions between COTS and coral, and monitored the condition of the juveniles for 3.5 mo. Juveniles suffered from both sublethal and lethal damage from coral defences. We determined the impact of arm damage on this rapid growth phase and compared the growth of injured and uninjured juveniles. As several previous studies of juvenile sea stars show that they have a reduced capacity to feed, lower energy reserves, and slower growth following arm autotomy (Diaz-Guisado et al. 2006, Barrios et al. 2008), we expected that injured juvenile COTS would also have a reduced growth rate, as energy would be reallocated to arm repair (Lawrence 1992).

2. MATERIALS AND METHODS

Adult *Acanthaster* sp. were collected near Cairns, QLD (16° 550′ S, 145° 460′ E), and transported to the National Marine Science Centre (NMSC), Coffs Harbour, NSW, where they were maintained in aquaria at 26°C. Two males and 2 females were spawned, and the larvae were reared as described in Deaker et al. (2020b). When the larvae reached the brachiolaria stage (≥16 d), settlement was induced using polycarbonate sheets covered in crustose coralline algae (CCA) that had been cultured in the aquaria system at NMSC for >2 yr. When the juveniles reached 1 to 2 mm Ø, they were collected from the sheets and were maintained on a diet of coralline algae for 10 mo in flow-through 1 µm filtered seawater (FSW) at 26°C.

2.1. Transition to and growth on coral

To quantify the behaviour of COTS when coral first became available to them, juveniles (n = 37) were offered a choice of coralline algae (~4 cm^2), coral (*Acropora valida*, ~4 cm^2), or the bare substrate of their containers for 48 h. Each juvenile was placed in the centre of a container (6 × 4 × 2 cm, 1 juvenile per container) at approximately equal distances from the different substrates. Containers were supplied with flow-through FSW (see below). The juveniles' choice of substrate was recorded after 1 h, and their movement was then tracked by recording their position hourly for 48 h without disturbing the containers. The presence–absence of feeding scars on the coral and the coralline algae was recorded.

Juveniles (mean ± SE at start = 16.43 ± 0.20 mm Ø, n = 37) were then provided with live coral prey (*Acropora* spp.) for 3.5 mo (111 d). Juveniles were not injured prior to being offered coral, and they were checked daily for survival and condition and if they were positioned on their food or not feeding. We also noted any behavioural change, but these observations were not analysed statistically. Coral was replaced as necessary to ensure that the juveniles could feed ad libitum. The juveniles were fed *A. valida* collected from the Solitary Islands, NSW (NSW DPI Permit P14/001-1.0), and *A. tenuis* supplied by an aquarium supply company (Cairns Marine) and collected under permit (Great Barrier Reef Marine Park Authority Permit G16/38631.1). Juveniles that struggled with the transition to coral were provided with CCA on small pebbles as a refuge. The CCA pebbles were cultured at ~26°C in aquaria.

The containers holding the juveniles were haphazardly distributed in a flow-through seawater system. Each container was supplied with 1 µm UV FSW through an individual dripper adjusted daily to maintain flow and stable temperature (mean = 25.82°C ± 0.28 SD, n = 95). Temperature was monitored using a Hach® HQ40d multi-controller with a Hach® PHC101 probe. The containers were cleaned every few days to prevent fouling.

The juveniles were photographed weekly for 3.5 mo using an Olympus Tough TG-5 digital camera mounted on a GorillaPod (Joby) stand. Diameter, the distance from the tip of one arm to the tip of the opposite arm, and the length of each arm were measured from photographs using ImageJ software (ver. 1.52a, National Institutes of Health, USA). Arm length was measured along the midline from the tip (excluding the spines) to where the arm met the central disk.

2.2. Arm regeneration and growth of injured juveniles

A damage index was used to determine the severity of arm damage for juveniles that were injured and survived. For each damaged arm, the length of the remaining arm was calculated as a percentage of the mean length of the intact arms from the same individual (damage index = $\frac{100 \times \text{damaged arm length}}{\text{mean intact arm length}}$).

To follow the regeneration of damaged arms, the

damage index was determined at the time when each arm was most damaged (T_{max}) and again at the end of the experiment (3.5 mo). The data were used to calculate the rate of regeneration (% d^{-1}) and predict the time (d) for each arm to completely recover to the length of an intact arm. The damage index could not be calculated for one juvenile, as it received damage to all arms.

As there was a delay from when juveniles were offered coral to when they started growing, initial growth rates were calculated from the time point where there was appreciable increase in their mean diameter. For injured juveniles, the growth rate in diameter and arm length (damaged and non-damaged arms) was calculated over 44 d from when their diameter started increasing at 67 d to the end of the experiment (111 d). These data were compared to the growth of arms of a subset of randomly selected juveniles (n = 10) that had no injuries over a similar time frame of 47 d from when their diameter started increasing (20–67 d).

2.3. Statistical analysis

To determine if COTS showed a preference for coral, coralline algae, or no choice (bare substrate) in the 48 h choice experiment, the number of time points each juvenile was recorded on each substrate (of 48) was ranked, and the ranks were analysed using a Friedman's rank test using the IBM SPSS Statistics program (ver. 25.0). Post hoc Wilcoxon tests were used where Friedman's test indicated significant differences among substrates ($p < 0.05$).

The growth rate of arms from injured juveniles (n = 10) and uninjured juveniles (n = 10) was compared using a non-parametric Kruskal-Wallis test, as the data were heteroscedastic (Levene's test, $p < 0.05$) and the residuals of the model were not normally distributed on quantile–quantile plots. The growth rates of damaged and non-damaged arms from injured juveniles (n = 10) were analysed using a linear mixed model (package nlme) (Pinheiro et al. 2019) with individual as a random factor. A Spearman's rank correlation coefficient (ρ) was used to analyse the correlation between the damage index (%) and the regeneration rate (% d^{-1} T_{max}) for each damaged arm (n = 50), the regeneration rate (% d^{-1} T_{max}) averaged across arms for each juvenile (n = 9), and the time to complete recovery (d) for each damaged arm (n = 50), as the data were not normally distributed (Shapiro-Wilks test, $p > 0.05$). Analyses were performed in R (ver. 4.0.2) (R Core Team 2020), and graphs were made using ggplot 2 (Wickham 2016). All data are presented as means and SEs.

3. RESULTS

3.1. Transition to coral

In the first 48 h that juveniles were offered coral in addition to coralline algae, they were most often positioned on coralline algae (77.4%) and rarely on coral (15.5%) or bare substrate (7.1%) ($\chi^2 = 28.83$, df = 2, $p < 0.0001$, coralline algae > coral = bare substrate, Fig. 1a). Of the 37 juveniles, 48.6% were recorded on coral over the 2 d. One juvenile fed on coral immediately and remained on coral for 47 h. The other juveniles explored both coralline algae and coral. Of the juveniles, 10.8% left feeding scars only on coral, and 18.9% left feeding scars on both substrates. The remaining 70.3% of juveniles only left feeding scars on coralline algae, including 5 juveniles that were recorded on coral for <3 time points and 2 other juveniles that were recorded on coral for 19 and 24 time points, respectively (of 48).

Most juveniles (87.8%) started to eat coral within the first 14 d of being offered this diet. A number of juveniles exhibited a reflex response to being stung when they encountered coral. Their arms recoiled and twisted when their tube feet came into contact with the polyps (Fig. 1b,c). While feeding, some juveniles appeared to hold their arm tips off the coral. Some juveniles retreated vertically after attempting to feed, walking on the tips of a few arms (Fig. 1d).

Most of the juveniles (59.4%, n = 22) were able to transition to coral with no damage within the first 6 wk. Despite the availability of coral, 1 uninjured juvenile continued to eat coralline algae for 63 d. The other 37.8% were damaged by coral, with injuries primarily to their arms and occasionally to the central disk (Fig. 2). Four juveniles with the most severe injuries, including damage to all arms and a perforated central disk, did not recover and perished (10.8%, Fig. 2e–h). The injured juveniles that survived arm damage (27.0%) were able to regenerate partially lost arms (n = 9, Fig. 2a–d) except for 1 juvenile that did not show signs of arm recovery.

Injuries dissuaded a number of juveniles from eating coral. Two of the severely damaged COTS that died had not eaten coral for 12 to 15 d. A number of juveniles reverted to coralline algae before reinitiating a coral diet. Of the injured juveniles, 1 recommenced on a coral diet after 3 d, while 3 were observed on coralline algae for 14 to 27 d before

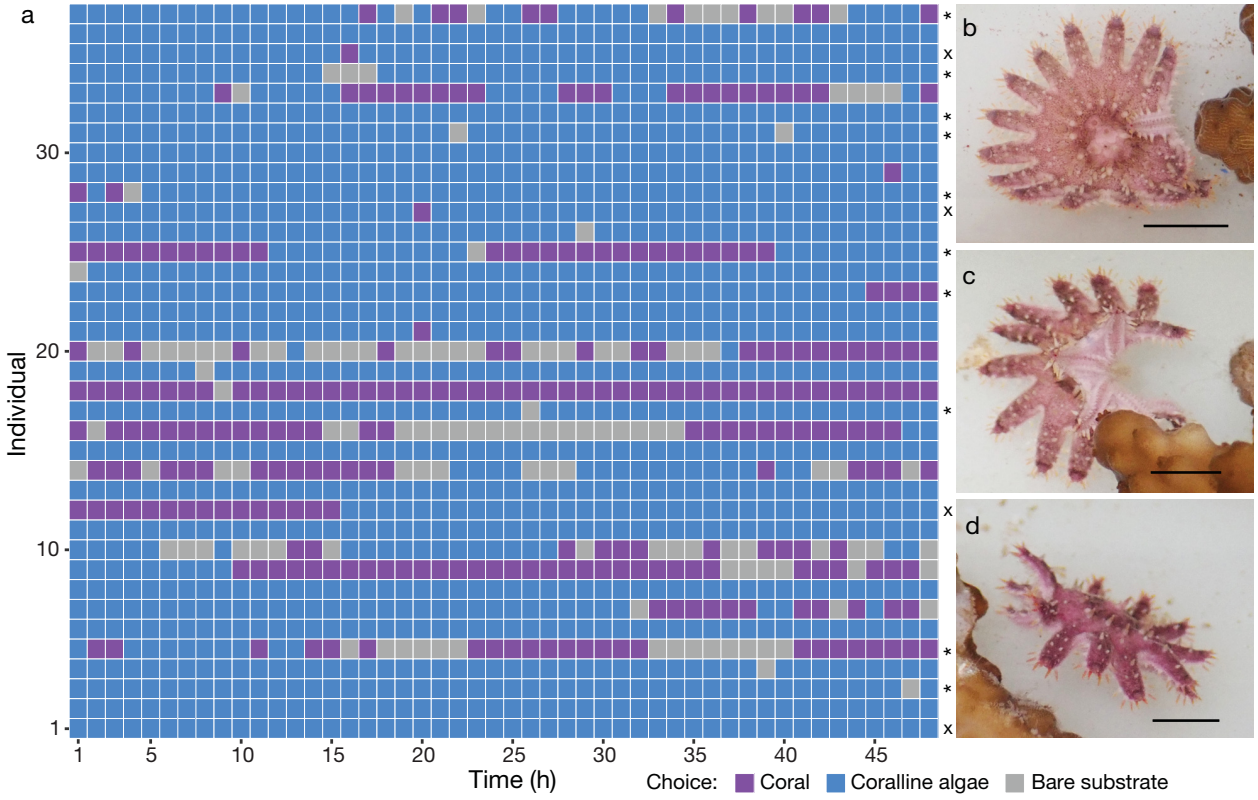

Fig. 1. Behaviour of juvenile crown-of-thorns starfish (COTS) at the start of a coral diet. (a) Position of juvenile COTS (n = 37) when first offered coral. Juveniles were offered coralline algae and coral simultaneously, and their position on the algae, coral, or bare substrate was recorded each hour for 48 h. Each row represents the position of 1 individual juvenile over time, and each box represents the location of that juvenile recorded every hour. The symbol at the end of each row indicates if the juvenile was subsequently injured and survived (*) or perished (×) during the experiment. (b–d) Juvenile COTS being stung during the transition to a coral diet. (b,c) Arms of 2 juveniles recoiling on contact with coral. (d) Juvenile vertically retreating after attempting to eat coral. Scale bar = 5 mm

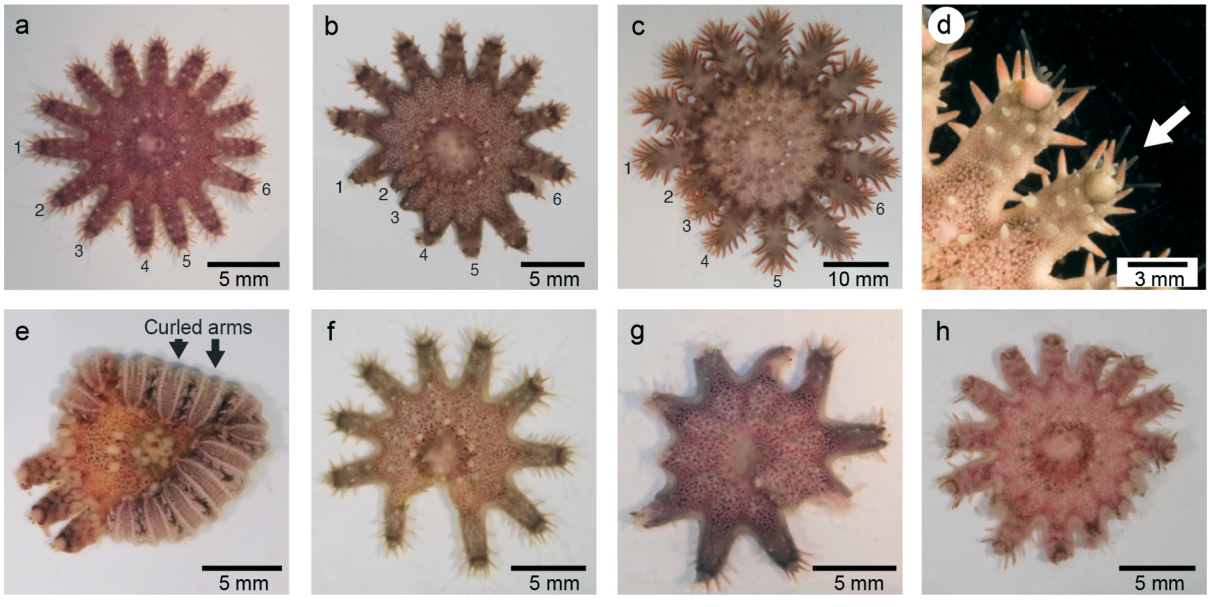

Fig. 2. Juvenile crown-of-thorns starfish (COTS) injured during the transition to a coral diet. A juvenile COTS (a) with undamaged arms (arms 1–6) at the start of the experiment, (b) after injury by coral (42 d), and (c) after subsequent growth and arm regeneration (101 d). (d) Regenerated arm tips, indicated with white arrow, were pale yellow compared to the pink tips of the original arms. (e–h) Four juveniles with ultimately fatal damage to the arms and central disk

attempting to eat coral again. The most injured surviving juvenile with damage to all 15 arms did not transition to coral for the duration of the experiment.

3.2. Juvenile growth on coral

Juveniles provided with coral did not show appreciable growth for some time. There was a delay of ~47 d for intact juveniles and ~67 d for the injured–regenerating juveniles between the time from which coral prey was offered to when they started to grow. At the end of the experiment (111 d), the mean diameter of the intact and injured juveniles was 40.51 ± 1.96 and 24.97 ± 3.74 mm (mean ± SE), respectively (Fig. 3a). The mean arm length at 111 d was 11.64 ± 0.83 and 5.96 ± 0.86 mm for the uninjured and injured juveniles, respectively (Fig. 3b). The mean diameter of the juveniles that died (20–42 d) was 14.66 ± 0.71 mm (n = 4, Fig. 3a).

3.3. Arm damage and regeneration

All sublethal and lethal injuries occurred within the first 45 d with the exception of 1 incidence of arm damage at 67 d. For the 10 juveniles with sublethal injuries, the mean time of initial arm damage was 31.5 ± 1.44 d (mean ± SE, range = 12–67 d) after being presented with coral. The average time when damaged arms were at their minimum length was 58.6 ± 2.28 d (range = 33–94 d, mean length = 2.34 ± 0.09 mm).

The incidence of damaged arms on individual juveniles ranged from 5.56 to 100% (43.12 ± 9.85%, n = 10 juveniles). A total of 65 arms were damaged across all juveniles (42.2%, n = 154 arms). Arms either were damaged along the length of the arm (n = 10) or had partial arm loss, where the tip was completely removed (n = 55). Regenerating arm tips were often pale yellow–white compared to the pink of the original arms (Fig. 1d). Some injured arms lost and regrew spines that did not differ in appearance from the non-regenerated spines.

The growth rate of the arms of injured (0.058 ± 0.013 mm d^{-1}, n = 10) and uninjured (0.049 ± 0.007 mm d^{-1}, n = 10) juveniles did not differ (χ^2 = 1.15, p = 0.284) in the first ~40 d after their arms started to increase in length (injured: 67–111 d, uninjured: 20–62 d). There was no significant difference between the growth rate of the damaged (0.057 ± 0.012 mm d^{-1}, n = 65) and intact (0.061 ± 0.013 mm d^{-1}, n = 89) arms of injured juveniles (67–111 d, $F_{1,143}$ = 1.92, p = 0.1681). However, more severely damaged arms grew at a faster rate than less damaged arms, with a significant negative correlation between the rate of recovery (% d^{-1}) compared to an intact arm and the damage index (T_{max}) (ρ = –0.630, p < 0.005, n = 50, Fig. 4b). When damaged arms were averaged on an individual juvenile basis, the regeneration rate and damage index (T_{max}) were not correlated (ρ = –0.667, p = 0.058, n = 9). At their most damaged, most arms were <70% of the length of intact arms (65.4 ± 2.3%, n = 29 arms, Fig. 4a). By 3.5 mo, most of the damaged arms (72%, n = 36 arms) had recovered to ≥80% of the length of an intact arm of the same individual (85.1 ± 1.6%, Fig. 4c). The damage index (T_{max}) was weakly yet significantly correlated with the time for an arm to recover to the size of an intact arm (p = 0.01711, ρ = –0.337, Fig. 4d). The recovery time of 48 of the 50 damaged arms was 101.98 ± 5.14 d (range = 33–189 d). Two arms had

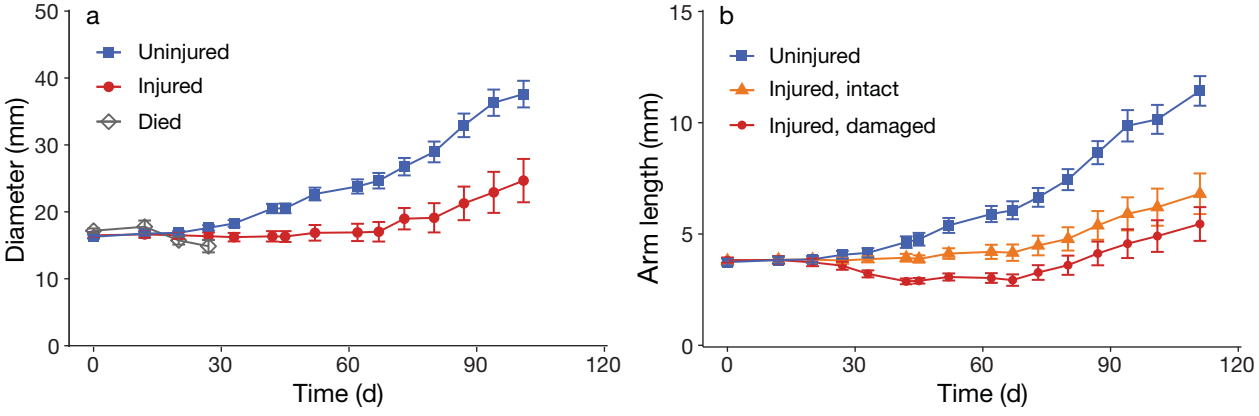

Fig. 3. Impact of damage from corals on the growth of juvenile crown-of-thorns starfish fed coral for 3.5 mo (111 d). (a) Mean diameter (±SE) of uninjured juveniles (n = 23) and juveniles that were injured and able to recover (n = 10) or died (n = 4). (b) Mean arm length (±SE) of uninjured juveniles (n = 10) and the intact and damaged arms of injured juveniles (n = 10)

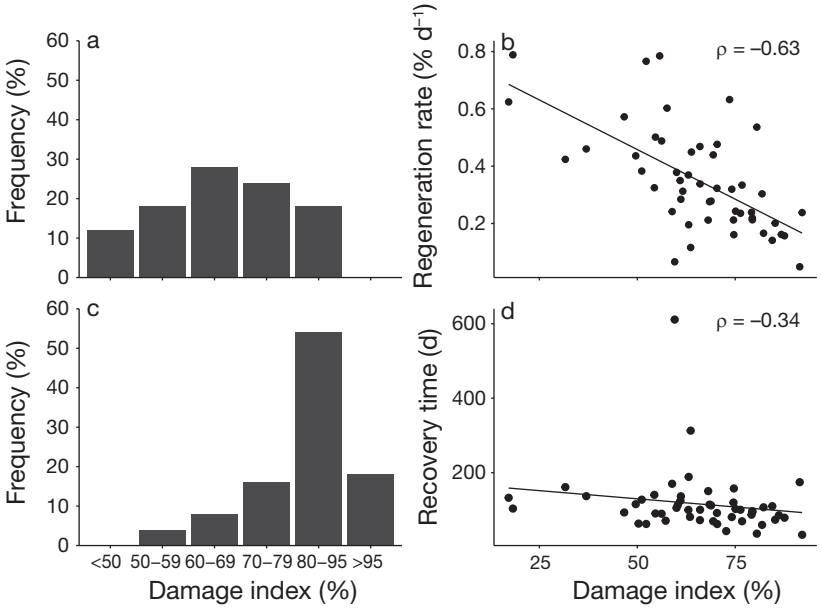

Fig. 4. Severity of arm damage (n = 50 arms) of 9 juvenile crown-of-thorns starfish, where the size of the remaining damaged arm could be compared to the mean size of an intact arm of the same juvenile (damage index, %). Left: Damage index (a) at the maximum amount of arm damage (27–94 d, T_{max}) and (c) at the end of the experiment (3.5 m, 111 d). Right: Correlation (Spearman's ρ) between the damage index at the time of maximum arm damage and (b) the rate of regeneration of each arm, showing that the arms with the greatest amount of damage regenerated at the fastest rate, and (d) the predicted recovery time to the length of an intact arm

very slow regeneration, with a projected recovery time of 313 and 611 d.

4. DISCUSSION

This is the first study of injury and regeneration in juvenile sea stars following damage caused by a natural enemy. We show that the damage caused by the tentacles and defensive cells of the 2 *Acropora* species used here can have negative impacts on the growth and survival of juvenile COTS. Coral stings caused fatalities, severely reduced arm length by up to 83%, and delayed the growth of juveniles, prolonging their herbivorous phase. Following sublethal damage, the juveniles were able to recover and regenerate lost arms. Although COTS prey on coral, coral is a hostile food source for the juveniles. Success, failure, and delay in the complete transition from herbivory to corallivory are likely to influence the timing of recruitment into adult populations.

The juveniles exhibited a suite of behavioural responses when they encountered coral, indicating that they were reacting to coral defensive mechanisms. In the first 48 h, a number of juveniles repeatedly touched coral and withdrew to bare substrate or the coralline algae-covered rocks. The arms were held aloft when feeding on coral, and the juveniles retreated in a standing-up, contorted position as opposed to moving away flat to the substrate. We also observed arm recoil after tube feet directly contacted the coral epithelium. Chemicals extracted from coral tissue have been shown to cause the tube feet of adult COTS to retract, a suggested mechanism to avoid stings (Moore & Huxley 1976). Despite reacting to stings, most juveniles persevered in their endeavour to eat coral. For some juveniles, this perseverance resulted in damage to the central disk and arms, as also noted by Yamaguchi (1974).

The shift from herbivory to corallivory varied substantially among the juveniles, from hours to months after first being offered coral. In a previous study, juvenile COTS (n = 7) switched to coral within 1 mo (Yamaguchi 1974). Here, the juveniles that were not injured completed their transition within 2 wk of being offered coral prey, with the exception of 1 of these juveniles that continued to eat coralline algae for 9 wk. In contrast, for the 10 juveniles that were injured by coral, only half completed the transition to coral in 3.5 mo. Six of these juveniles sought refuge in coralline algae while recovering from damage inflicted by corals. They re-attempted to eat coral when their arms had started to regenerate. In field surveys, the higher proportion of injured juveniles retrieved from coralline algae habitat compared to live coral may have included juveniles that had retreated to this habitat, where they were recovering from coral-inflicted damage (Wilmes et al. 2019). Coral polyps may be a leading cause of sublethal injuries in juvenile COTS. This may explain why the incidence of arm damage in juveniles was similar between areas open and closed to fishing in the Great Barrier Reef (Wilmes et al. 2019). In their vulnerable and damaged state, juvenile COTS may not be able to withstand continued exposure to coral defences. Sheltering on coralline algae may also reduce the risk of further predation due to arm damage and loss of defensive spines, decreased mobility (Lawrence &

Vasquez 1996), and potential impacts to their vision, as their ocelli are located at their arm tips (Sigl et al. 2016, Garm 2017).

The incidence of individuals with arm damage in the present study (27%) was lower than that observed for juvenile COTS found in coral habitat in nature (85%, 12–45 mm Ø) (Wilmes et al. 2019). Smaller juveniles retrieved in field surveys may be more vulnerable to coral stings compared to the juveniles used in this study that were at the largest size attainable on their algal diet (18 mm Ø). On coral reefs, scleractinian coral assemblages are more diverse, and the extent to which different species inflict damage to COTS is likely to vary depending on the potency of their defences (e.g. nematocysts, mesenterial filaments, and sweeper tentacles) (Collins 1975, Wellington 1980, Gunthorpe & Cameron 1990). While corals are not predators of juvenile COTS, our observations indicate that corals may be an important source of sublethal damage and juvenile mortality.

Sublethal damage to the juveniles may also be caused by invertebrate and fish predators (Cowan et al. 2017). The coral rubble habitat is home to a plethora of cryptic species including polychaetes and crustaceans that can prey on juveniles (Keesing et al. 2018). In a laboratory study, peppermint shrimp ate juvenile COTS aged <4 mo and damaged older, larger juveniles (7–21 mm Ø) (Balu et al. 2021). However, predatory attacks have not been witnessed on juvenile COTS in nature. Sweatman (1995) reported the partial consumption of juveniles (<70 mm Ø) when offered to 3 emperor fish species by divers. In a caging experiment open to invertebrate and fish predators, 50 to 75% and 50 to 92.5% of small (3 mm Ø) and large (13 mm Ø) juveniles, respectively, disappeared likely due to mortality, as there was no evidence of sublethal predation (Keesing et al. 2018).

Inter-individual variation in the transition to corallivory and the incidence of arm damage is likely to be driven by differences in the sensitivity of the juveniles to coral defences and the time taken for them to become resistant or produce counterdefences. Juveniles with a greater number of spines may have increased protection from coral tentacles (Kamya et al. 2018). Stressed COTS (Buck et al. 2016) and newly settled juvenile COTS (Johnson et al. 1991) can also secrete mucus which may serve to protect them from coral defences, similar to coral-eating wrasse (Huertas & Bellwood 2017), anemone fish (Lubbock 1980, Elliott & Mariscal 1997), and nudibranchs (Greenwood 2009) that produce a mucus to protect them from cnidarian nematocysts. Fully competent coral-eating juvenile COTS are not deterred by coral defences. Further research is needed to identify the mechanism(s) by which COTS protect themselves from coral defences.

In addition to the tolerance of coral stings, adjusting to a new diet requires physiological changes in COTS that may take a variable amount of time to develop. This may explain the 3 wk hiatus before appreciable growth was evident following the introduction to a coral diet. To avail of coral, the COTS digestive system has to produce specialised enzymes that digest coral wax esters (Benson et al. 1975, Brahimi-Horn et al. 1989). It is unknown when these enzymes start to be produced by COTS, and if there is a variation between individuals in their ability to produce them. Enzyme production may be initiated around the size that the juveniles can transition to coral, or be stimulated by contact with coral. In the latter case, juveniles may not be completely competent to eat coral when they first come into contact with it.

An increase in diameter was delayed by approximately 4 wk in the injured juveniles compared to the intact ones, likely due to energy and resources being allocated to regeneration rather than growth. The injured juveniles that struggled with the transition to coral and were offered coralline algae may have had limited capacity to grow. Coralline algae appear to have insufficient nutritional value to support the rapid growth of larger juveniles (Deaker et al. 2020a). Loss of structures due to coral-induced damage, such as the pyloric caecae that extend into the arms and are important for digestion and energy storage (Lawrence & Larrain 1994, Lawrence & Vasquez 1996), likely also inhibits the capacity of juveniles to grow. In other studies of arm regeneration following autotomy in juvenile sea stars, those with missing arms had a reduced feeding and growth rate compared to intact individuals (Diaz-Guisado et al. 2006, Barrios et al. 2008). For COTS, more severely damaged arms recovered at a faster rate compared to less damaged arms. Prioritising regeneration at the expense of somatic growth is common among sea stars as a strategy to advance recovery and minimise the risks associated with arm loss such as increased vulnerability to predators and reduced foraging capacity (Lawrence & Vasquez 1996, Diaz-Guisado et al. 2006, Maginnis 2006). Open wounds also increase susceptibility to infection from pathogenic bacteria (Lafferty & Harvell 2014, Ben Khadra et al. 2017). This may have contributed to the 99.3% mortality rate in a population of juvenile COTS in

Fiji, where 10% of monthly samples of juveniles were diseased 3 mo either side of the documented transition to coral (Zann et al. 1987).

The regeneration rate of damaged arms in juvenile COTS was similar to that documented following arm autotomy in the juveniles of other sea stars. Arms of COTS, *Helianthus heliaster*, and *Stichaster striatus* recovered by 10.1% in ~2 mo, 30% in 5 mo (Barrios et al. 2008), and 25% in 5 mo (Diaz-Guisado et al. 2006), respectively. However, the most severely injured juvenile COTS with damage to all 15 arms did not regenerate. The arm tips did not heal, a necessary step for regeneration, as regrowth occurs behind the terminal plate (Hotchkiss 2009) and requires the scaffold provided by the radial nerve cord (Byrne et al. 2019, Byrne 2020). It is unknown if arm damage and slower growth during the juvenile stage have any carryover effects to the reproductive fitness of the adult stage, where arm damage can result in reduced fecundity (Budden et al. 2019).

Sublethal predation is a commonly used proxy for predation pressure in sea star populations (McCallum et al. 1989, although see Lawrence 1992). This is particularly useful for the management of COTS, where reduced predation pressure is a hypothesised trigger of population outbreaks. As damaged juveniles are able to recover, we suggest caution using this as a tool to monitor predation rates of juvenile COTS. However, it is unlikely that corals inflict damage to larger juvenile, subadult, and adult COTS. After 3.5 mo, damaged arms showed extensive recovery. Using the model of juvenile growth proposed by Wilmes et al. (2017), arms damaged by coral are likely to be fully recovered to the length of an intact arm when the juvenile is ~35 mm diameter, based on the average predicted recovery time of 4 mo. Damage in older COTS is more likely to be caused by interactions with non-coral predators.

The early life stages of COTS are vulnerable to coral defence mechanisms. Corals prey on the larvae (Yamaguchi 1973, Cowan et al. 2017) and, as shown here, can damage or kill the juveniles. The remarkable ability of COTS to regenerate body parts across the larval, juvenile, and adult life stages attests to the importance of regenerative abilities and growth plasticity in the success of this species (Allen et al. 2019, Budden et al. 2019, Deaker et al. 2020b). As the complete transition to a coral diet may be delayed by sublethal injury, damage to the juveniles caused by coral stings or other predators is likely to influence the population dynamics of COTS. Injuries slow the growth of the juveniles to maturity, reduce their capacity to prey on coral, and prolong the waiting phase.

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