Vol. 106: 181–185, 2013
doi: 10.3354/dao02652

DISEASES OF AQUATIC ORGANISMS
Dis Aquat Org

Published October 11

NOTE

Contrasting recovery following removal of growth anomalies in the corals *Acropora* and *Montipora*

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ABSTRACT: Growth anomalies (GAs) in scleractinian corals drain energy from the host and can result in partial or entire colony mortality. Here I show that growth anomaly removal is an effective treatment for the branching coral *Acropora acuminata*, with 90% of subjects remaining GA-free 9 mo following the procedure. In contrast, the encrusting coral *Montipora efflorescens* did not respond positively to treatment, with GAs re-developing in 100% of treated subjects. There was no clear evidence that injuries sustained during GA removal increased susceptibility to GA development in either coral species. Based on these results, I hypothesize that the factors inducing GAs in *Acropora acuminata* are localized, whereas those in *Montipora efflorescens* appear more systemic throughout the colony — perhaps the result of a genetically-based factor, or a persistent causative agent such as a virus. GA removal may therefore be effective for targeted rescues of particular coral species and morphologies in reef systems with low overall disease prevalence and is likely to be most effective for scleractinian corals if complimented by management actions that address the ultimate drivers of GAs on coral reefs.

KEY WORDS: Growth anomaly · Recovery · Clonal regrowth · Injury · Morphology · Coral disease management

INTRODUCTION

In vertebrates, it is common to use medical intervention in an attempt to cure disease. Many invertebrates, including corals, also suffer from disease. A common disease affecting scleractinian (hard) corals are growth anomalies (GAs) (Sutherland et al. 2004, Vargas-Angel 2009). Coral GAs frequently manifest as hyperplasia of the basal body wall and not a particular cell type per se, meaning they are not synonymous with true neoplasia (Work et al. 2008a, Williams et al. 2011b). The condition can be progressive, reducing coral colony fitness and reproductive potential (Work et al. 2008a, Burns & Takabayashi 2011, Stimson 2011) and lead to partial or entire colony mortality over time (Williams et al. 2011a). The prevalence of GAs on reefs appears to be positively correlated with local human population numbers (Aeby et al. 2011a,b), colony density (Aeby et al. 2011b), and coral bleaching stress (McClanahan et al. 2009), and although unconfirmed, a viral causative agent has been postulated (Aeby et al. 2011b) due to the abundance of herpes-like viruses seen in stressed corals (Vega Thurber et al. 2008, 2009). The progressive and detrimental nature of coral GAs to the coral colony and their often high prevalence within coral populations means they can potentially threaten coral reef ecosystems. Unlike tissue loss diseases that have extirpated corals (Aronson & Precht 2001), however, the role of GAs in reef ecosystem function and resilience remains unclear.

Mitigation actions that directly reduce the ultimate drivers of coral GAs on reefs are clearly desirable, but in some instances local-scale intervention may also be

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necessary and appropriate. Little is known about how hard corals respond to direct attempts to prevent or halt disease establishment and progression at the individual colony scale. In particular, the effectiveness of surgical GA removal in preventing the re-development of coral GAs throughout an individual colony has not been previously explored. Corals are capable of wound repair following injury (Henry & Hart 2005, Work & Aeby 2010), but injuries themselves can increase the susceptibility of corals to disease (Page & Willis 2008). In the case of coral GAs, it is unclear whether removing the growths, while potentially alleviating the burden of disease, would permanently cure the colony. During a previous study, Williams et al. (2011b) noted that GAs could be removed more easily from branching corals than from encrusting or massive corals. In the present study, GAs were removed from individual colonies of 2 hard coral species (branching and encrusting) to address the following null hypotheses: H_{01}: GA removal has no effect on GA re-development. H_{02}: physical injury does not increase susceptibility of corals to GA development.

MATERIALS AND METHODS

Study site

Experimental manipulations were conducted at Palmyra Atoll National Wildlife Refuge (05° 52′ N, 162° 06′ W), in the Northern Line Islands, central Pacific (Fig. 1). Palmyra is located 1930 km south of the main Hawaiian Islands and has been protected by the US Fish and Wildlife Service since 2001. In 2009, Palmyra became part of the Pacific Remote Islands Marine National Monument, and represents a functionally intact ecosystem, with low (<0.4%) overall coral disease prevalence (Williams et al. 2008, 2011a). Palmyra contains a variety of reef habitats that vary in coral cover and diversity; however, this study focused on coral communities on the shallow (<5 m) western reef terrace (Fig. 1). Two dominant coral genera in this habitat are encrusting *Montipora* and tabular and branching *Acropora* corals (Williams et al. 2013).

Experimental growth anomaly removal

To address potential between-species variation in response to GA removal, I focused on 2 coral species with different morphologies: *Montipora efflorescens* (an encrusting coral) and *Acropora acuminata* (a branching, or staghorn, coral). Both species suffer from GAs at Palmyra, with cases almost exclusively manifesting as hyperplasia of the basal body wall (Williams et al. 2011b).

I set up 4 treatment levels along a 50 × 10 m transect on Palmyra's western terrace in October 2008, and all coral colony subjects were re-visited approximately 9 mo later in July 2009. To address hypothesis 1, 10 colonies of each species were subjected to complete GA removal using a hammer and chisel so that no visible GA tissue remained (level: treated). Coral colonies were chosen haphazardly, but the number of overall GAs and their size was standardized as best as possible among individual corals. I deliberately chose colonies in an early stage of infection (displaying one to five ~4 cm^2 GAs) in an attempt to maximize the positive response of the treatment. To determine if this treatment had a significant effect on the change in the number of new GAs developing on individual colonies, 5 additional colonies of each species with GAs were marked in October 2008 for re-assessment in July 2009 (level:

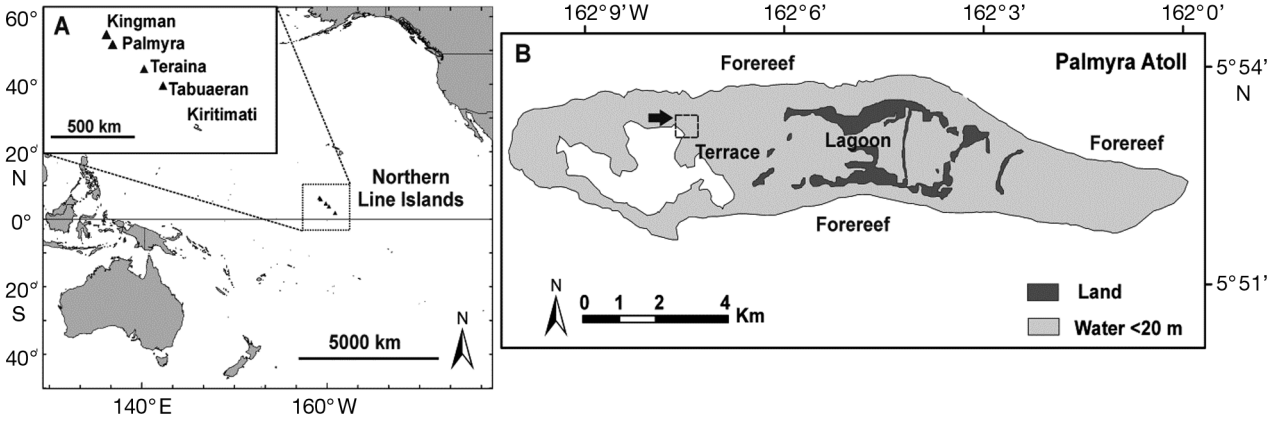

Fig. 1. Location of (A) Palmyra Atoll in the Northern Line Islands and (B) the shallow western terrace study site, indicated by the square and black arrow

GA control). To address hypothesis 2, I removed pieces of living tissue from 5 colonies of each species that appeared healthy at the beginning of the experiment (level: procedural control). Care was taken to mimic the wounds left on the colonies subjected to GA removal (level: treated), with each procedural control colony having three ~4 cm^2 areas of tissue removed, spread evenly across the colony surface. To determine if these injuries increased susceptibility to GA development, 5 colonies of each species that appeared healthy at the beginning of the experiment in October 2008 were re-assessed in July 2009 for signs of GA development (level: healthy). A fully balanced design across treatment levels could not be achieved due to permitting constraints of this initial study within the refuge.

Within-species colony sizes (maximal widths) were standardized as best as possible (mean ±1 SE: *Acropora acuminata* = 250 ± 40.5 cm, *Montipora efflorescens* = 44 ± 5.2 cm), but it was not possible to standardize for colony size between species, as *A. acuminata* grows far larger and more spatially expansive than *M. efflorescens*. Each colony subject used was marked with a uniquely numbered tag attached to the adjacent reef substrate, and their positions mapped with respect to the permanent transect.

Data analyses

All analyses were completed using R 2.15.2 (R Foundation of Statistical Computing). To test for an effect of treatment on the change in number of GAs per colony between the 2 time points (hypothesis 1), I used a Kruskal-Wallis test owing to the non-normal nature of the data and the relatively small sample sizes (4 levels: treated, GA control, procedural control, healthy). Planned contrasts (treated versus GA control) were completed using Dunn's pairwise comparisons to account for the unequal sample sizes across treatment levels. To determine whether injuries increased susceptibility to GA development (hypothesis 2), I used a Fisher's exact test based on a contingency table to account for the small sample sizes, specifically comparing the procedural control colonies to the healthy individuals. The response variable in this case was binomial: presence of GAs in July 2009 or not.

RESULTS

Treatment had a significant effect on the change in number of growth anomalies (GAs) per colony over time in *Acropora acuminata* (H = 22.53, df = 3, $p < 0.0001$), with the control colonies developing significantly more GAs over time than the treated colonies ($p < 0.0001$) (Table 1). In contrast, treatment had no effect in *Montipora efflorescens* (H = 5.71, df = 3, $p = 0.126$), with 100% of the treated colonies redeveloping GAs over time. The mean number of new GAs developing in the treated *M. efflorescens* colonies did not differ from the natural rate of GAs developing ($p = 0.0738$) (Table 1). New GAs in treated *M. efflorescens* colonies developed in both previously healthy-looking and previously treated areas (Fig. 2). In some cases the wounds left on *M. efflorescens* colonies following GA removal healed at the edges, but in no cases did clonal regrowth of healthy tissue occur over the newly available substrate. If these wounds did not re-develop as GAs, they became colonized by turf algae (mixed community of filamentous algae and cyanobacteria generally less than 2 cm tall) and crustose coralline algae. In contrast, all wounds left following GA removal in *A. acuminata* fully healed via clonal regrowth (Fig. 2). There was no clear evidence that physical injury made coral subjects more susceptible to GA development, with natural rates of GA development in apparently healthy individuals equaling those of the injured colonies for both coral species ($p > 0.999$). In summary, 1 procedural control and 1 healthy *M. efflorescens* colony each developed GAs between the 2 time points, and none of the procedural control or healthy *A. acuminata* colonies showed signs of GA development (Table 1).

Table 1. Presence of growth anomalies (GAs) on 2 species of hard coral 9 mo following 4 experimental treatment levels in October 2008: (1) treated (all GA tissue removed), (2) GA control (colonies with GAs followed over time), (3) procedural control (GA removal simulated by inflicting similar injuries), and (4) healthy (colonies without GAs followed over time)

Treatment level	No. of colony subjects	No. of colonies with GAs in July 2009
Acropora acuminata		
Treated	10	1
GA control	5	5
Procedural control	5	0
Healthy	5	0
Montipora efflorescens		
Treated	10	10
GA control	5	5
Procedural control	5	1
Healthy	5	1

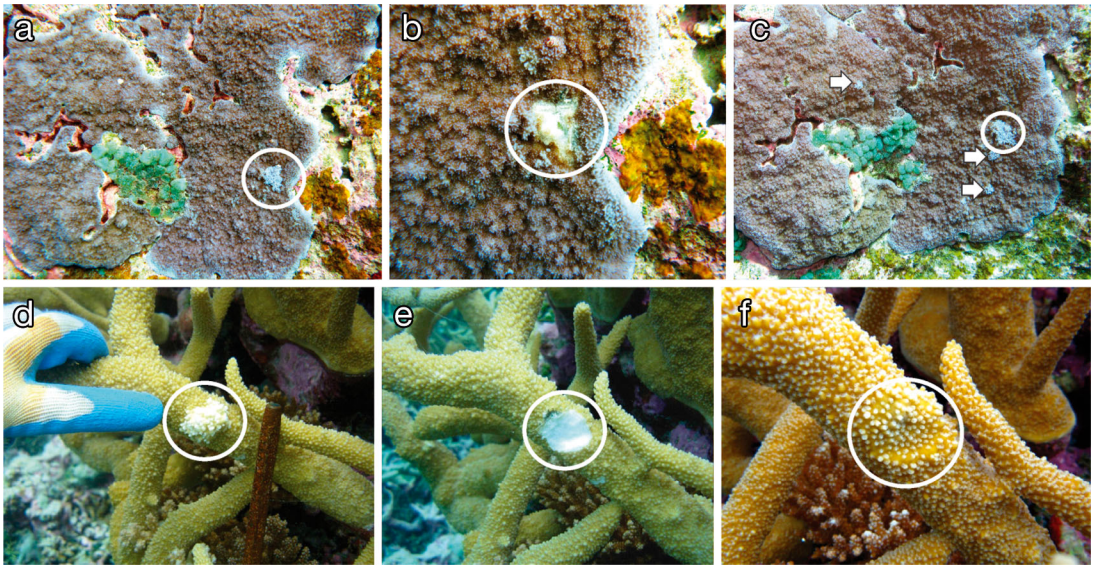

Fig. 2. *Montipora efflorescens* and *Acropora acuminata*. (a–c) *M. efflorescens* showing a negative response to growth anomaly (GA) removal. (a) October 2008, GA is present (circle). (b) Wound left after GA removal in October 2008 (circle). (c) Re-development of GA (circle) and development of novel GAs in previously healthy portions of the colony (arrows). (d–f) *A. acuminata* showing a positive response to GA removal. (d) October 2008, GA is present (circle). (e) Wound left after GA removal in October 2008 (circle). (f) Complete clonal regrowth and recovery of the afflicted area by July 2009, with just a small depression (circle) noticeable in the skeleton

DISCUSSION

The 2 species of hard coral examined in this study, *Acropora acuminata* and *Montipora efflorescens*, showed contrasting responses to the growth anomaly (GA) removal treatment. *A. acuminata* appeared to respond positively, with 90% of treated colonies remaining GA-free 9 mo following the procedure. This response suggests that the factors inducing GA development in this species are localized to the GA tissue. The wounds left on the *A. acuminata* subjects following treatment rapidly healed via clonal regrowth, and similar injuries in healthy individuals did not appear to increase susceptibility to GA development. *Acropora* corals are generally fast-growing (Lang & Chornesky 1990) and can rapidly repair injuries via clonal regrowth (Fong & Lirman 1995). In addition, the GAs on the branching coral *A. acuminata* at Palmyra generally have prominent nodular or exophytic morphologies (Work & Aeby 2006, Williams et al. 2011b), facilitating GA removal with minimal damage to the host coral.

In contrast, GAs re-occurred in 100% of treated *Montipora efflorescens* colonies either by novel development on previously healthy areas of the host tissue, or more rarely by re-developing in the same location from which they had been experimentally cleared (2 of the 10 treated subjects showed this, with a maximum of 1 GA per colony). This response suggests that the factors inducing GA development in this species are distributed systemically throughout the colony, are a result of a genetically-based factor, or are perhaps a result of a persistent causative agent, such as a virus. Viruses are commonly found in the coral holobiont, particularly when corals are subjected to environmental stress, such as increases in temperature or elevated nutrients (Vega Thurber et al. 2008, 2009). As such, a viral agent has been postulated as the causative agent of GAs in some species of coral due to the increase in GA prevalence with increased proximity to human population centers (Aeby et al. 2011a,b), where viral concentrations in the water also increase (Dinsdale et al. 2008). Finally, GAs on the encrusting coral *M. efflorescens* at Palmyra are generally low-lying umbonate and bosselated structures (Williams et al. 2011b), and full GA removal requires deep vertical and horizontal excavation into the colony, leaving substantial wounds. While there was no clear evidence that these wounds led to increased susceptibility of GA development in injured coral subjects, previous work has highlighted the difficulty of removing all tissues from a lesion area in corals with similar morphologies (Work & Aeby 2010). This, in conjunction with the more perforate skeleton of *Montipora,* may somehow allow continued GA development throughout the colony even following experimental removal of obvious GA tissue, perhaps via cryptic GA tissue remaining.

In summary, despite the low overall sample sizes and the possible influence of microhabitat variations in environmental conditions across our colony subjects, the results from this initial study strongly suggest that removal of GAs can halt re-development in some hard corals, such as the branching coral *Acropora acuminata*, but not in others, such as the encrusting coral *Montipora efflorescens*. The mitigation of the ultimate drivers of GAs on coral reefs should be a management priority, and solutions are likely to be complex and disease-specific due to the intricate pathogen-host-environment triad of disease causation (Work et al. 2008b, Williams et al. 2010). However, the direct removal of GAs on individual colonies may be appropriate under certain circumstances, such as in aquaria or in systems with low overall GA prevalence where managers wish to attempt targeted rescues of charismatic corals, such as large reef-building *Acropora* corals. Targeted GA removal could also be used to complement larger-scale management actions, such as establishing local sewage treatment, that may help to mitigate against the ultimate drivers of GAs on coral reefs. Such interventions may provide an additional buffer to aid coral reef ecosystem recovery and promote resilience.

Acknowledgements. I thank the US Fish and Wildlife Service (USFWS) and The Nature Conservancy for granting access to the refuge and logistical support. The majority of funding was provided by the National Geographic Society. This work was conducted under the USFWS special use permit 12533-09015. I additionally thank I. Knapp for field support, and R. Morrison, T. Work, and 2 anonymous reviewers for comments that greatly improved the manuscript. Scripps Institution of Oceanography is a member of the Palmyra Atoll Research Consortium (PARC). This is PARC publication number PARC-0097.

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Editorial responsibility: Garriet Smith, Aiken, South Carolina, USA

Submitted: May 15, 2013; Accepted: July 19, 2013
Proofs received from author(s): September 20, 2013