Genet and reef position effects in out-planting of nursery-grown *Acropora cervicornis* (Scleractinia:Acroporidae) in Montego Bay, Jamaica

Andrew M. Ross

Seascape Caribbean; ross.andrew@mac.com

Abstract: The reef-building coral *Acropora cervicornis* was a dominant ecosystem element on the Caribbean reef until the 1980s, when it declined by some 97% due primarily to anthropogenic ecosystem changes and disease. This branching species expanded its colony footprint and achieved local dominance largely through fragmentation and regrowth, thus is suited to nursery culture towards restoration. In this experiment, fragments of *Acropora cervicornis* of four lineages or *genets* were followed and measured for growth and health over 12 months in 2006 and 2007 on buoyant drop-loop line nurseries at one shallow and one deep fore-reef site in Montego Bay, Jamaica. Sixty-five of these corals were then out-planted to wild reef sites of similar depth and condition to their respective nurseries and monitored photographically for 11 months through 2007 and 2008. A period of rapid death was seen in the out-planted material at both sites over the first four months, followed by a period of relative stability or recuperation. *Hermodice carunculata* predation was the primary problem in the shallow fore-reef, and was combined with a banding syndrome at the deeper site. This syndrome was noted in the samples prior to planting, during a one week storage period on the seafloor. Continued slow decline occurred in the subsequent seven months in the shallow fore-reef site; however, regrowth was noted in the deeper site in the remaining material. Including these losses, final total live coral length was more than fourfold greater than the initial wild harvest: a net increase through multi-stage propagative restoration or coral gardening. Returns were noted particularly in the faster-growing genets of the nursery and larger planted corals tended to retain more material at eleven months, suggesting that propagative restoration programmes invest in stronger genets and larger corals. Adaptive management and maintenance gardening of the planted material and reef would likely have greatly improved outcomes. Rev. Biol. Trop. 62 (Suppl. 3): 95-106. Epub 2014 September 01.

Key words: *Acropora cervicornis*, coral gardening, coral restoration, coral propagation, *Hermodice carunculata*.

The *Acropora* of the tropical western Atlantic are in jeopardy, as are the ecosystems dependent on the intricate branching habitat or defensive reefal accumulations developed by these corals historically (ABRT, 2005). *Acropora cervicornis* was the dominant coral from 5m through 20m depths, an area coined by Goreau (1959) as the *Acropora cervicornis* zone, through most of the Caribbean prior to the early 1980s. Today this species is sparse throughout its range with only a handful of such thicketed habitats remaining and some authors paint a bleak picture for its future. They suggest that a naturally relatively low level of sexual reproduction, a lack of larger spawning animals, the distance between them, chronic stress and the lack of planular settlement habitats has led to an allee effect. Under this effect replacement through sexual reproduction is no longer occurring, yet adult death is continuing in a downward spiral to local extirpation, if not extinction (Stephens & Sutherland, 1999; Knowlton, 2001; Miller & Szmant, 2006).

Several authors have explored the potential for coral gardening to begin to preserve or restore these keystone corals (Bowden-Kerby, 1997; 2001; Rinkevich, 2008). Coral gardening suggests using multi-stage processes akin to silviculture for coral ecosystem restoration, wherein small amounts of coral are harvested

from the wild reef and grown in nurseries. After a period of growth these may then be refragmented, propagated for further nursery growth, planted out in reef restoration or used in experimentation, with no further collection from the wild stocks (Bowden-Kerby, 1997; Epstein, Bak & Rinkevich, 2003; Rinkevich, 2006; Shafir & Rinkevich, 2008). As such, an initial harvest may be very small yet provide large amounts of propagated material. Bowden-Kerby (2001) and Ross (2013a) both report more than an order of magnitude increase in useable *A. cervicornis* material within a single year of nursery growth. It is interesting that corals that grow so well in the nursery are still in decline in the wild (Miller, Bourque & Bohnsack, 2002; ABRT, 2005; Alcolano, Caballero & Perera, 2009). This suggests that one may separate the corals from their problems by simply separating them from the substrate: their issues appear to be particularly benthic and not waterborne or intrinsic to the corals themselves. Planting this growth from the nursery to the reef is the next step in elucidating these issues and resurrecting the Caribbean's reefs.

This work is the final in a series of studies in nursery growth of *A. cervicornis* wherein strong differences in growth rate and susceptibility to temperature, fouling and abrasion stresses were noted between genets. In these genets the fastest growers tended to also be the least susceptible to stress (Ross, 2013c), so, here we out-plant propagated corals and follow their progress to see if the differences seen in the nursery hold in the wild. The experimental queries of this work include: i) following the progress of out-planted corals grown in a novel nursery design to assess the efficacy of nursery propagation in ecosystem restoration, ii) following each of the genets to assess whether the relative strength and weakness shown in the nursery carries to the wild reef and its various and different issues.

METHODS

In April 2006, two Buoyant Drop-Loop Line (BDL) nurseries (Ross, 2013a) were set at each of two sites in Montego Bay, Jamaica: a large sand area adjacent to a large reefal stone at 15m depth at the popular dive site Widowmaker's Cave at 18°29'51.59"N, 077°56'5.00"W, and in a sand-channel of the well developed buttress formations of the Airport Reef dive site at 18°29'50.83"N, 077°55'56.24"W. These nurseries were each populated with 5 unbranched 5cm fragments from each of four presumed distinct (Ross, 2013b) coral genets marked with colour-coded wires (N=80). These corals were partially harvested in June 2007 and set to new nurseries, with the remaining nurseries moved intact to concrete block-anchors immediately to one side where they were maintained for a further five months.

Hurricane Dean passed the western tip of Jamaica on August 20, 2007. These maintenance nurseries were sequestered from August 19 for approximately one week by placing them into sediment-free hollows in the reef. Shortly after this storm these corals were planted to adjacent clean reef substrate.

At the deep nursery site the coral nurseries were tucked into a shallow depression immediately adjacent to the nurseries that was clear of sediment; however, there was macroalgae present. Out-planting occurred prior to September 3, 2007 to this same reefal stone at depths of between 14m and 12.5m. No live or dead *A. cervicornis* was present on the reefal stone, though it was present on the continental shelf some 30m away and no active disease was noted in this natural population.

For planting, diseased portions were cut away with clean side-cut pliers 1cm to 2cm ahead of the advancing disease front, as a banding syndrome (likely WBD1) had beset many samples during sequestration. Where the disease front was in the middle of the coral, it was cut into two or more independent fragments, 1cm to 2cm of live-tissue waste was cut away before the advancing disease front in this experiment. In trials at the Doctor's Cave Beach Club and elsewhere in Montego Bay waste of <5mm has proven ample (unpublished), though the author recognizes that every infection or location may be different.

Galvanized masonry nails were immersed in seawater and left outdoors in a mesh bag until they developed a uniform flat grey patina, for approximately two weeks. Fresh galvanized wire or nails will kill the contacting coral tissue; however, aging the galvanizing coating has proven effective in eliminating this contact toxicity in propagated *A. cervicornis* planting trials at the Doctor's Cave Beach Club, Montego Bay, Jamaica (unpublished).

To plant each coral, one pre-aged nail was driven into an exposed, upper portion of reefal stone or dead coral head atop the planting stone and the area around that nail picked clean of macroalgae or any accumulated sediment, though the specific planting locations were chosen to be cleaner points of bare stone. A coral was then cut from the nursery line at random, clipped of any disease and set to the nail with live tissue touching both the nail and substrate to promote attachment. The genet-indicator wire was checked by gently breaking away the thin coral venire at its free end to see its colour. A nylon cable-tie of that same colour was then used to secure the coral to the nail. As necessary, aged galvanized (steel) binding wire was used to make the fragment tight and still. Fragments tended to be horizontal; however, as they were on the upper points of the reef structure they contacted the substrate at a single point. Where a sample was cut into two or more segments to remove disease these were set bundled together, straddling the anchoring nail. Corals were set with a reasonably even distribution around the stone, though somewhat concentrated to the eastern end. The corals were mapped and numbered for repeat monitoring. Twenty-nine corals were planted and followed at this reefal stone.

The nurseries within the buttress system were anchored at 7m depth, thus the nursery corals were growing elevated to between 6.2m and 5.5m from the surface. For Hurricane Dean these shallow nurseries were bundled into a clean, shallow cave at 8m depth within the buttress system. After the storm, these bundles were moved atop a partially urchin-grazed buttress-end at 3m depth at the top, surrounded by

caverns on the shoreward sides and a large sand patch on the seaward side all at 9m depth making the planting area something of an island at 18°29'51.49"N, 077°55'57.35"W. This stone held two natural colonies of *A. cervicornis*, both of which were small and stressed, the larger (<0.25m^2) hosting a *Stegastes planifrons* nest. Between 3m and 5m depth and concentrating on the southern face of the planting stone was an area of reasonably urchin- and fish-grazed stone with occasional live coral and macroalgae. Several *Diadema antillarum* urchins were present.

Small limestone projections, knobs and nodules are common on such shallow patch reef-tops, remnants of small massive and branching corals long dead. In coral planting, such knobs were held firmly and shaken to ascertain the knob's strength given boring organisms, primarily *Cliona* sponge colonization: if stone strength was insufficient the piece would break away. For each chosen planting point a coral was cut at random from the nursery bundle and its genet indicator wire exposed. The coral was then fastened against the tested reef nodule with a cable-tie of the genet's indicator colour. The planted corals were then mapped and numbered. Thirty-six corals were planted and followed at this site.

The planted corals of each site were each photographed in turn according to their mapped location and number with a scale bar or measuring tape from the angle judged by the photographer to provide the largest size for each sample. The cable-tie's colour was visible in the photograph. Photo-sessions occurred three times over the first four months (September 3, October 23 and December 22) then again at 11 months (shallow site on July 30 and deep site on August 8). A supplemental set of photographs was taken on September 14 at the deeper site to observe the advancing disease, though this is not discussed in detail here.

The photographs were evaluated using CPCe's Accumulated Lengths function taking both total skeletal and living tissue lengths. Live and dead lengths were taken for each branch and sources of death were assessed,

where possible. No consideration was made for error associated with parallax (Ross, 2012a).

Preliminary analysis of CPCe data was carried out using Microsoft Excel software for Macintosh. Statistical tests, including ANOVA, non-parametric correlations and graphing were performed using SPSS18 statistical software. Nonparametric correlations between i) apical polyp count, ii) worm bite count, iii) active disease front count, iv) total live plus dead length, v) dead length and vi) living coral length were assessed using Spearman's rho. Correlations between Log base-10 transformation of the initial live and the final live coral lengths were provided by a Pearson's correlation. General Linear Model (GLM) ANOVA testing used the Natural Log transformed live coral length data with the factors site, visit and genet, hypothesizing:

1. There is no significant difference in living coral length between the deep and shallow sites,
2. There are no significant differences in living coral length between the different visit dates,
3. There are no significant differences in living coral length between the different coral genets,
4. There is no significant interaction between the site, visit and/or genet with regard to living coral length.

RESULTS

The shallow site saw greater survivorship than the deeper site. Sixty-five corals were set in September 2007. Forty-six (70.8%) were still in place and 35 (53.8%) were still alive by the summer of 2008. Of the 36 corals planted at the shallower site, eight were missing and much partial mortality was observed, but all 28 corals present were alive at 11 months (77.8%). At the deeper reefal stone site, of the 29 samples set, 11 were lost (37.9%) and 11 died entirely, leaving only seven surviving (24.1%, Table 1). Damages were observed by the first measurement and continued through the first

four months with every coral in this experiment suffering partial mortality.

At both sites partial mortality was substantial, with 47% of tissue lost in the shallow site and 91% in the deep (Table 3). Losses were continuous from planting through the final measure in the deep site even after the initial pulse of predation and disease; however, the shallow site saw a short period of growth prior to its period of decline (Table 3). The deep site saw a rapid decline early, with disease killing corals entirely. However, after an initial pulse, the continued decline was primarily through remaining or continuing disease and sample loss after the December visit. Lost corals may have been attributed to waves in the shallow site, though curious or clumsy spear-fishermen and recreational snorkelers may also have

TABLE 1
Partial and full mortality and losses at 11 months per site and per genet

	Genet	Count	Some Live	All Dead	Missing
Shallow	Orange	9	8	0	1
	White	9	7	0	2
	Blue	10	8	0	2
	Green	8	5	0	3
	Total	36	28	0	8
Deep	Orange	6	3	3	0
	White	7	1	3	3
	Blue	9	3	2	4
	Green	7	0	3	4
	Total	29	7	11	11

TABLE 2
N-values changing through the experimental period with sample loss

Date	Depth	N
1. Sept. 3	Shallow	36
	Deep	29
2. Oct. 23	Shallow	36
	Deep	29
3. Dec. 22	Shallow	35
	Deep	29
4. Jul. 31/Aug. 8	Shallow	26
	Deep	18

TABLE 3
Total live lengths per location

Visit	Shallow (cm)	Shallow (% change)	Deep (cm)	Deep (% change)
1	2 505.7	/	1 684.5	/
2	2 589.2	+3.33	304.0	-81.95
3	1 741.3	-32.75	217.2	-28.55
4	1 338.9	-23.11	149.6	-31.12
Totals	-1 166.8	-46.57	-1 534.9	-91.12

* N-values are in Table 2.

played culprit. At the deeper site an Antillean Z-trap (fish trap) was removed from atop the reefal stone and samples on one occasion, and the location is a popular recreational SCUBA site. Later in the experiment the decomposition of the distal skeleton was rapid, such that dead samples may have simply broken or rotted away.

The September 14 interim observations at the deep site show disease starting either at the benthic contact point, possibly associated with damages incurred during planting, or at the branch tips suggesting vectoring or facilitation by *Hermodice carunculata* (Fire-Worm) with pathogen entry at the bite or lesion. *H. carunculata* attacks were common in this interim visit but were obscured by more general (disease) death by the time of the formal monitoring visit in October. There was no indication that disease started at the points where disease was cut away as an advancing disease front was not necessarily discernable from a *H. carunculata* bite.

The trend over time for mean live coral length is great initial partial mortality, followed by a levelling out in mortality and slight growth at the deep site but a continued slow reduction at the shallow site (Table 4). In the month after planting, the shallow buttress site saw growth, with a per-coral average increase of 3.4%, or 2.4cm per coral. However, in the following period *H. carunculata* worms found these new corals and consumed some 30.8% of the total crop over two months; an average of 22.2cm per coral. This site continued to decline slightly in average live coral length through the remainder of the experimental period through continued *H. carunculata* predation.

The deeper site saw damage almost immediately with disease present in the first photo. Between loss, disease and *H. carunculata* feeding this site's mean coral length declined by some 82.6% in the first six weeks and a further 28.6% by December. However, from this greatly depressed state it was able to regrow by some 10.7% in the remaining seven months (Table 4) during which it was monitored.

TABLE 4
Gains and losses per location per visit as mean live lengths per coral, excluding lost samples

Visit	Shallow cm	Shallow % change	Deep cm	Deep % change	Deep cm excl. dead	Deep % change, excl. all-dead samples
1	69.6	/	60.2	/	60.2	/
2	72.0	+3.45	10.5	-82.56	-27.6	-54.15
3	49.8	-30.83	7.5	-28.57	-18.1	-34.42
4	47.8	-4.02	8.3	+10.67	+21.4	+18.23
Total	-21.8	-31.32		-86.21	-38.8	-64.45

* Percent change is the length difference between visits as expressed as percent grown or lost. N-values are in Table 2.

New apical polyp formation occurred in the initial period at the shallow site, often associated with healing cut-points or undocumented damage to apical areas from storage. They also formed on the branch trunk as they would in nursery corals and in areas associated with a worm bite or halted disease front. This did not occur immediately afterwards, but after an often-prolonged period of rest or healing, and often only once the dead portions had rotted or broken away. New apical formation was the source of restarted growth between the third and final visit as worm damage left few apical polyps for growth.

Both the shallow and deep sites saw an increase in the average number of apical polyps per coral between the third and final visits as the remaining tissue started to re-grow (Fig. 1). The difference between this and Table 3 at the shallow site was that, as apical polyp and branch formation continued, predation also continued.

A decline was noted generally through the experimental period in each genet. At the shallow site, Orange and Green lost 28% and 41% tissue respectively until day 110 (visit 3). Orange then held at 28% while Green declined by 61% by the final visit. Blue lost 21% of its average length by day 110 and declined a further 25% by the final visit. White also declined by 21% by day 110 but had regained an average of some 3.1cm per coral by the final visit (Table 5).

At the deep site, Orange again remained relatively steady between days 110 and 340, with 77% and 78% loss respectively. White and
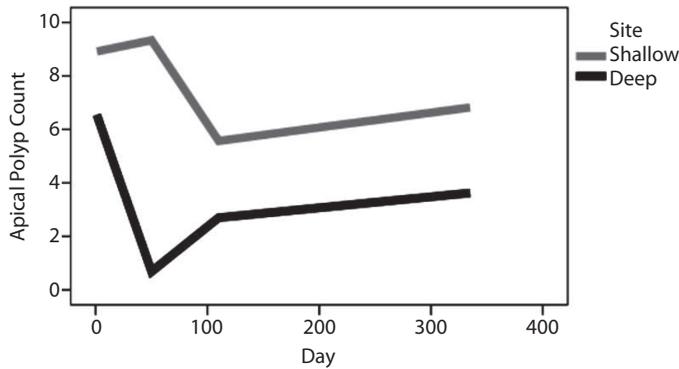

Fig. 1. Apical polyp counts over the 11 months of the experimental period. N= 29 at the deep site, N= 36 at the shallow.

TABLE 5
Advances and declines as mean lengths per genet per visit (V) in cm, excluding lost samples. N-values are in Table 2

	Genet	Living V 1	Living, V 3	% loss	Living, V 4	% loss	Dead, V 3	Dead, V 4
Shallow	1) Orange	91.58	65.05	28	65.56	28	34.61	28.76
	2) White	49.26	38.93	21	42.04	15	19.41	14.54
	3) Blue	60.35	47.86	21	45.53	25	29.38	10.45
	4) Green	79.33	46.69	41	31.18	61	32.45	25.62
	Total	70.13	49.63		46.08		28.96	19.84
Deep	1) Orange	63.07	14.29	77	13.97	78	34.46	18.77
	2) White	52.15	3.16	94	3.35	94	40.49	23.07
	3) Blue	59.94	6.61	89	10.47	83	39.95	24.84
	4) Green	65.92	7.13	89	0	100	42.45	21.83
	Total	60.27	7.80		6.95		39.34	22.13

* N-values are in Table 2.

Blue saw greater losses, losing 94% and 89% respectively at day 110, with White remaining steady through day 340 while Blue improved its average coral size by some 3.8cm per coral. Green expired entirely within the experimental period at this site. A marked decline in dead skeleton length was noted between days 110 and 331/340, by as much as or more than 50% in some cases, due to breakage and erosion.

General Linear Model (GLM) ANOVA testing (Adjusted R Squared 0.235) showed the live length to be significantly different between sites (p <0.001), between different measurement visits (p <0.001) and between genets (p= 0.008). Post hoc testing showed that Blue, White and Green genets made up the subset with the least coral length and White, Green and Orange made up the subset with the most coral. As may be seen in Figures 4 and 6, these post hoc differences were largely associated with uneven initial amounts of coral and the particular failure of the Green genet at both sites. A significant interaction between site and day (p= 0.022) speaks to rapid disease onset in the early measurements at the deeper site and *H. carunculata* attack in between visits at the shallower site.

The differences between the genets seen in Table 4 are illustrated in Figure 2, whereby a rapid decline in live coral length is seen in the deep site in all genets with a continued fall in the Green genet to zero, but a mild increase in the Blue genet to the final visit. In the shallow site a brief period of growth may be seen in the White and Orange genets and a mild decline in the Green and Blue, followed by a general decline in all genets. After this early decline, the Blue genet saw a further mild decline while the Green genet suffered a much more pronounced decline and the White genet began to regrow. In both cases the Orange genet remained relatively stable after the initial decline, with no particular re-growth or further decline after day 110. Green declined generally throughout and could be described as the weaker performer based on this figure, whereas the Blue and White saw some improvements in mean length and could be described as the stronger performers.

There was no correlation between recent *H. carunculata* attack and length of living tissue occurring through the 11 months (Spearman's rho r= -.064, p= 0.356). This was in part due to the worm's apparent lack of preference in coral size, but also that once the coral was attacked the amount of living coral for assessment was reduced. Similarly, a strongly negative correlation occurred between *H. carunculata* bites and the apical polyp count (Spearman's rho r= -0.333, p <0.001) as the worms actively ate the branch tips.

The worms in this reef patch were large, the largest single bite being 10.6cm long and bite lengths averaging 3.2cm overall. Worm
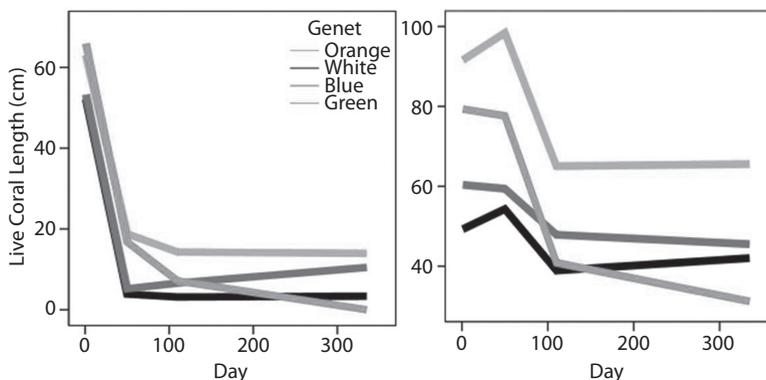

Fig. 2. Mean length of living coral per genet excluding lost samples over the 11 months of the experiment, (A) at the deep and (B) shallow sites. The Y-axis and key apply to both A and B. N= 36 at the deep site, N=29 at the shallow.

bite count was positively correlated with the amount of dead coral (Spearman's rho r= 0.544, p <0.001) but not with the total coral size including live and dead lengths (Spearman's rho r= 0.199, p= 0.086) again suggesting that worm feeding preferences are not associated with the size of the coral. Worm attacks were prevalent in the deep site but were particularly problematic in the shallow buttress site (Fig. 3A) and were the primary factor limiting coral growth at the shallow site.

Disease was negatively correlated to living tissue length (Spearman's rho r= -0.396, p <0.001). This did not mean that disease tended to occur in smaller corals, but rather disease shortened the living portion of the coral for assessment. Similarly, the count of disease fronts was negatively correlated with the number of apical polyps (Spearman's rho r= -0.387, p <0.001) as apical polyps were either killed by the disease or associated with worm attacks. Worm attacks may have played a role in disease distribution, though worm bite count was not significantly correlated with the number of disease fronts in this experiment (Spearman's rho r= -0.025, p= 0.723). Disease front count was positively correlated with the length of dead skeleton (Spearman's rho r=0.520, p <0.001) as the disease was actively killing coral and producing dead skeleton. Disease front count was not correlated with the total amount of coral (Spearman's rho r= 0.051, p= 0.461) suggesting that disease susceptibility was not related to the size of the coral. Banding disease occurred occasionally in the shallow buttress site but was particularly prevalent on the deeper reefal stone where it was the primary killer of tissue and corals (Fig. 3B).

Figure 4A suggests that there is little difference between the genets with respect to worm attack, which may be expected in a coral reliant on rapid growth and overgrowth as its strategy for reef dominance: it would not invest much in chemical defences. Figure 4B suggests a weak trend towards Green being more susceptible to disease than the other genets. This would corroborate its general weakness, though Orange trends towards being the least susceptible to disease.

There was a significant positive correlation between the initial live and the final live coral lengths (Pearson r= 0.387, p= 0.023).

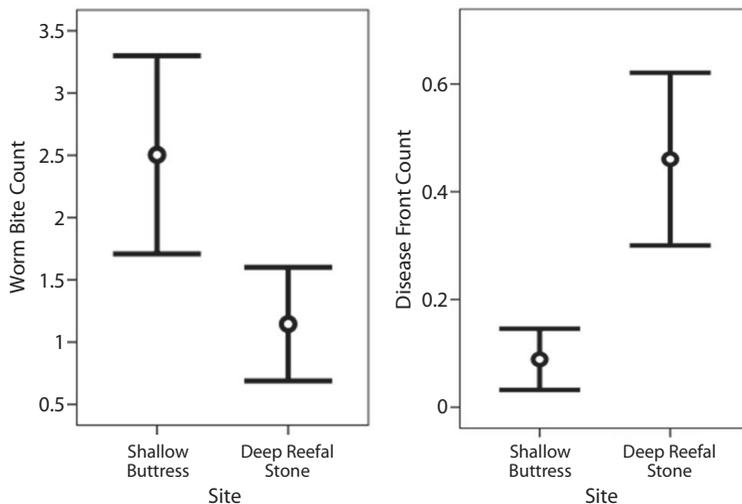Occasional partial mortality with banding disease was seen where portions of the total

Fig. 3. Mean *Hermodice carunculata* bite count (A) and disease front count (B) per living coral per site pooled through the experimental period, showing the particular prevalence of disease at the deep site and of worm attack in the shallow (±the 95% Confidence Interval). N= 36 at the deep site, N=29 at the shallow.
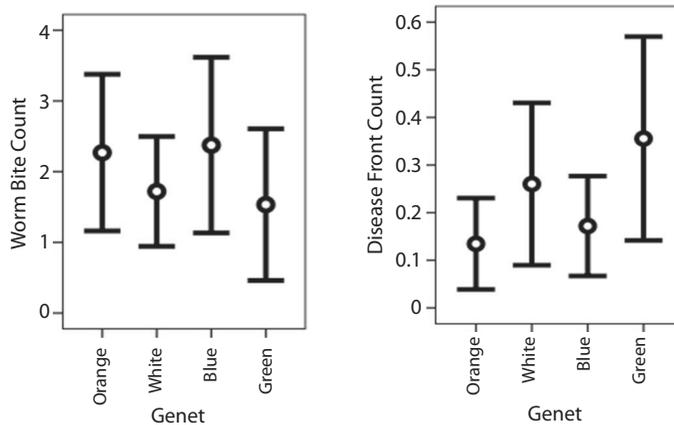
Fig. 4. Mean *Hermodice carunculata* bite count (A) and disease front count (B) per living coral per genet pooled through the experimental period, showing no significant difference between the genets in these factors (±the 95% Confidence Interval). N= 36 at the deep site, N=29 at the shallow.

coral were not killed. These were often sections that were somehow physically separated from the primary coral, e.g. sections broken away by fish-pot or diver damage or broken portions of the drop-loop overgrowth held attached by the underlying nylon line without tissue contact.

Bleaching occurred at the shallow site likely associated with the stress of sequestration, a reduction in depth from the shallow nursery at some 5m and 6m depth to the planting site at 5m to 3m depth and from the seasonal temperatures regardless of relocation activities, though these factors were not assessed in this study. Bleaching did not kill any samples and was only prevalent in the Green genet.

DISCUSSION

Publishing or inclusion of negative outcomes and experimental failures in reporting and published literature is necessary to build a larger picture of the lessons learned by the restoration and manipulation community (Edwards & Gomez, 2007), but also to shed larger light on the factors affecting corals generally including the reasons for presence or absence of a given species in a location. Failure may be as informative as success in restoration, as failure poses its own, arguably more interesting and fundamental questions.

At first glance this study may suggest that propagative coral enhancement of *A. cervicornis* is folly, as the successes of the nursery are not immediately translated into successes on the planted reef. In fact the opposite is true. The amount of coral initially harvested from the wild reef for 65 fragments at five cm each, some 325 linear centimetres, with nursery growth yielded some 4 277.5 centimetres for planting after a preliminary harvest for nursery re-sets and disease pruning. After 11 months, 1 521.15cm remained, giving a net increase of 1196cm or 4.7-fold from the initial wild harvest. Had this been a direct parent-to-planting relocation without an interim nursery phase, the 64.48% decline seen here would amount to some 115cm of total coral length remaining. Considering that this study and others (Knowlton, Lang & Keller, 1990; Bowden-Kerby, 2001) have found greater survivorship in larger disturbed or relocated corals, this final figure would likely have been less.

In this study the corals of the shallow, buttress-end reef grew and attached to the substrate for the first measurements, but by day 100 were heavily set upon by at least one large *Hermodice carunculata* (fire-worm) that consumed much of the branch and apical material, essentially halting growth. After the initial attack period these corals remained at

a similar size, with no particular re-growth or further decline noted in the 11 months in mean coral length, though some apical growth and further worm bites were noted. This suggests that a critical mass of coral may be planted on an appropriate area that will maintain a reasonably steady population through predation in the absence of disease, storm or similar catastrophic change.

At the deeper reefal stone site fewer worm attacks were combined with a banding syndrome that was apparently inherent in the sediments, as per Patterson et al. (2002), and possibly exacerbated by macroalgae as this site was largely ungrazed (Nugues et al., 2004) as some corals were showing disease during pre-planting storage, before they were planted to the reef. This combination possibly vectored or facilitated by the worms themselves (Sussman, Loya, Fine & Rosenberg, 2003) was able to kill some 91% of the total live coral length by day 340. This illustrates the importance of adaptive management and site choice (Rogers & Montalvo, 2004; Edwards & Gomez, 2007). Had adaptive management been employed, the shallow site would have seen particular investment in worm control, including active hunting and trapping. The deep reefal stone would have been abandoned and the remaining samples either returned to the nursery or moved to another location once disease was seen to be prevalent, likely in limited trials in a cooler season (Patterson et al., 2002; Jones, Bowyer, Hoegh & Blackall, 2004; Bruno et al., 2007). Such adaptive management is also an aspect of coral gardening (Rinkevich, 2005; 2006).

Control of corallivores is a vital aspect of restoration of *Acropora,* and the first, simplest and only viably scalable way to do so is to focus restoration efforts into protected or managed ecosystems with an appropriate compliment of grazers and invertivores. Although it was not assessed here, the shallow site in this study was at a depth that urchin grazing kept the substrate relatively bare of macroalgae and disease was rare relative to the deeper site beyond the habitual urchin-grazing depths. Upright and frondos macroalgae may harbour

cryptic corallivores (Bruckner, Bruckner & William, 1997) and develop boundary layers to water flow that collect and harbour sediments (Smith, Smith & Hunter, 2001) including those that may hold disease (Patterson et al., 2002). Thus site choice should look for locations with appropriate grazing and water flow on both macro and micro scales.

Larger corals had significantly more living tissue at the end of the experiment. This suggests that a restoration programme use the largest fragments possible to maximize the amount of material remaining after predation or disease, as well as to maximize productivity through the improved extension rates of larger corals (Ross, 2013d). Although correlations were not found with these assessments, *H. carunculata* only bites as far as it may engulf the branch, thus larger corals should be less susceptible per unit length to this type of attack. Relatedly, once a branch has been bitten it will not be attacked again: the coral's own exposed, dead skeleton protects it from further attack so long at the next worm is not larger. As these worms also only bite down to the first branch fork, a project manager may also maximize branching in the planting stock by targeted damage (Soong & Chen, 2003), nursery site choice (Bottjer, 1980; Ross, 2013b) and even parent coral choice (Bowden-Kerby, 2001; Ross, 2013c) to limit fire-worm feeding efficiency, though it also stands to reason that a coral with more branches for attack would be attacked more often. Had the worms been smaller in this experiment, the corals may have seen more protection due to their relative size, and had the corals been larger or more branched they might have also seen protection from these large worms. Disease or smaller predators, such as snails, may not be so restricted.

The patterns of strong (Blue and White) and weak (Orange and Green) genets seen in the nursery (in Ross, 2013c) were not overtly apparent in these wild out-plants. Predation and disease were the primary issues and apparently not subject to genet influences, as opposed to the bleaching and fouling abrasion seen in the nurseries. The Green genet was the only

genet to bleach with the stresses of relocation and summer temperatures, suggesting that the heritable susceptibilities to the nursery stresses persist. The Orange genet did not show particular regrowth, recovery or improvement at either site, remaining at a similar total length from the penultimate to the final measurement, whereas the Green genet continued to decline throughout and eventually disappeared entirely from the deeper site. The White and Blue genets similarly fared poorly early in the experimental period in both locations; however, in the final visit, they showed a modicum of recovery not seen in the weaker genets. Slight regrowth was seen in the shallow site for the White genet and in the deep site for the Blue. Based on these observations, the relative strengths and weaknesses seen in the nursery for improved growth, branching and resistance to bleaching stress continued into the out-planted coral. A trend of increased disease susceptibility in the Green genet also suggests that disease, abrasion and other stresses facilitating disease, will also be more problematic in weaker genets.

ACKNOWLEDGMENTS

RESUMEN

Efectos de la posición en el arrecife y del linaje en el crecimiento de *Acropora cervicornis* **(Scleractinia:Acroporidae) en una plantación de vivero en Bahía Montego, Jamaica.** La especie constructora de arrecifes de coral *Acropora cervicornis* era un elemento dominante del ecosistema en el arrecife caribeño hasta la década de 1980, cuando disminuyó en un 97% a nivel regional principalmente debido a cambios antropogénicos y por enfermedad. Esta especie de ramificación amplió su huella de colonia para lograr un dominio local a través de la fragmentación y el rebrote, así se adapta al cultivo de vivero para la restauración. En este experimento, fragmentos de *Acropora cervicornis* de cuatro linajes fueron seguidos y medidos para el crecimiento y la salud durante 12 meses en 2006 y 2007 en viveros en línea tipo "buoyant drop-loop" en un sitio somero y otro profundo en el arrecife frontal de Bahía Montego, Jamaica. Sesenta y cinco de estos corales fueron plantados en sitios de arrecife silvestre con condiciones y profundidad similar a sus respectivos viveros y monitoreados mediante fotográfias por 11 meses durante el 2007 y 2008. Se observó un período de muerte rápida en el material plantado en ambos sitios durante los primeros cuatro meses, seguidos por un período de relativa estabilidad o recuperación. La depredación de *Hermodice carunculata* fue el principal problema en el arrecife frontal poco profundo y se combinó con un síndrome de bandas en el sitio más profundo. Este síndrome se observó en las muestras antes de la siembra, durante un período de almacenamiento de una semana en el suelo marino. A continuación ocurrió un lento descenso en los posteriores siete meses en el sitio de arrecife frontal poco profundo; sin embargo, se observó un rebrote en el sitio más profundo con el material restante. Aún incluyendo estas pérdidas, al final la longitud de coral vivo total fue más de cuatro veces que la inicial: un aumento neto a través de varias etapas de restauración propagativa o de jardinería de coral. Los rechazos fueron observados especialmente en el linaje de crecimiento más rápido del vivero y corales plantados más grandes que tienden a retener más material en once meses, lo que sugiere que los programas de restauración propagativo deben invertir en linajes de coral más fuertes y más grandes. Probablemente se obtengan mayores resultados con un manejo adaptativo y mantenimiento de jardinería del material plantado y de arrecife.

Palabras clave: *Acropora cervicornis*, coral cuerno de ciervo, propagación de cultivo de coral, jardinería de coral, restauración, *Hermodice carunculata*.

REFERENCES

ABRT (Acropora Biological Review Team). (2005). *Atlantic Acropora Status Review Document.* Southeast Regional Office, Report to National Marine Fisheries Service, Southeast Regional Office.

Alcolano, M., Caballero, H., & Perera, S. (2009). Trend of Change of Live Stony Coral Cover in Cuban Coral Reefs. *Serie Oceanológica, 5,* 15.

Bottjer, D. J. (1980). Branching Morphology of the Reef Coral *Acropora cervicornis* in Different Hydraulic Regimes. *Journal of Paleontology,* *54*(5), 1102-1107.

Bowden-Kerby, A. (1997). Coral Transplantation in Sheltered Habitats Using Unattached Fragments and Cultured Colonies. *8th International Coral Reef Symposium, Panama, 2,* 2063-2068.

Bowden-Kerby, A. (2001). Low-Tech Coral Reef Restoration Methods Modeled after Natural Transplantation Processes. *Bulletin of Marine Science, 69,* 915-931.

Rev. Biol. Trop. (Int. J. Trop. Biol. ISSN-0034-7744) Vol. 62 (Suppl. 3): 95-106, September 2014

105

Bruckner, R. J., Bruckner, A. W., & William, E. H. (1997). Life History Strategies of *Coralliophila abbreviata* Lamarck (Gastropoda: Coralliophilidae) on the Southwest Coast of Puerto Rico. *8th International Coral Reef Symposium, Panama, 1*, 627-632.

Bruno, J. F., Selig, E. R., Casey, K. S., Page, C. A., Willis, B. L., Harvell, C. D., Sweatman, H., & Melendy, A. M. (2007). Thermal Stress and Coral Cover as Drivers of Coral Disease Outbreaks. *PLoS Biology, 5*(6), e124.

Donahue, W. F., & Schindler, D. W. (1998). Diel Emigration and Colonization Responses Of Blackfly Larvae (Diptera: Simuliidae) To Ultraviolet Radiation. *Freshwater Biology, 40*, 357-365.

Edwards, A. J., & Gomez, E. D. (2007). Reef Restoration Concepts and Guidelines: Making Sensible Management Choices in the Face of Uncertainty. *The Coral Reef Targeted Research & Capacity Building for Management Program, 38*.

Epstein, N., Bak, R. P. M., & Rinkevich, B. (2003). Applying Forest Restoration Principles to Coral Reef Rehabilitation. *Aquatic Conservation: Marine and Fresh Water Ecosystems, 13*, 387–395.

Goreau, T. F. (1959). The Ecology of Jamaican Coral Reefs I. Species Composition and Zonation. *Ecology, 40*(1), 69-90.

Jones, R. J., Bowyer, J., Hoegh-Guldberg, O., & Blackall, L. L. (2004). Dymanmics of a Temperature-Related Coral Disease Outbreak. *Marine Ecology Progress Series, 281*, 63-77.

Knowlton, N. (2001). The Future of Coral Reefs. *Proceedings of the National Academy of Science of the United States of America, 98*, 5419-5425.

Knowlton, N., Lang, J. C., & Keller. B. D. (1990). Case Study of Natural Population Collapse: Post-Hurricane Predation on Jamaican Staghorn Corals. *Smithsonian Contributions to the Marine Sciences, 31*, 25.

Miller, M. W., Bourque, A. S., & Bohnsack, J. A. (2002). An Analysis of the Loss of Acroporid Corals at Looe Key. Florida, USA: 1983-2000. *Coral Reefs, 21*, 179-182.

Miller, M. W., & Szmant, A. M. (2006). Lessons Learned from Experimental Key-Species Restoration. pp. 219-234. *In* Precht, W. F. (ed.). *Coral Reef Restoration Handbook*. Boca Raton, FL.: CRS/Taylor & Francis.

Nugues, M. M., Smith, G. W., Van Hooidonk, R. J., Seabra, M. I., & Bak, R. P. M. (2004). Algal Contact as a Trigger for Coral Disease. *Ecology Letters*, 7, 919-923.

Rinkevich, B. (2005). Conservation of Coral Reefs Though Active Restoration Measures: Recent Approaches and Last Decade. *Processes in Environmental Science Technology, 39*, 4333-4342.

Rinkevich, B. (2006). The Coral Gardening Concept and the Use of Underwater Nurseries: Lessons Learned From Silvics and Silviculture. pp. 219-234. *In* Precht, W. F. (ed.). *Coral Reef Restoration Handbook*. Boca Raton, FL.: CRS/Taylor & Francis.

Rinkevich, B. (2008). Management of Coral Reefs: We Have Gone Wrong When Neglecting Active Reef Restoration. *Marine Pollution Bulletin, 56,* 1821-1824.

Rogers, D., & Montalvo, L. A. M. (2004). *Genetically Appropriate Choices for Plant Materials to Maintain Biological Diversity. University of California.* Report to the USDA Forest Service. Rocky Mountain Region, Lakewood, CO. Retrieved from http://www.fs.fed.us/r2/publications/botany/plantgenetics.pdf.

Ross, A. M. (2013a). *The Buoyant Drop-Loop Line Nursery: A Novel Coral Nursery for In Situ Propagation of Acropora cervicornis, Growth Performance Outcomes and a Rapid Measurement Option. Chapter 2.* Ph.D. Thesis, UWI Mona, Dept of Life Sciences.

Ross, A. M. (2013b). *The Effect of Reef Position on Genets of Acropora cervicornis in Buoyant Line Nursery Culture at Montego Bay, Jamaica. Chapter 3.* Ph.D. Thesis, UWI Mona, Dept of Life Sciences.

Ross, A. M. (2013c). *Genet Considerations in Line Nursery Propagation of Acropora cervicornis Towards Restoration, with Observations of Growth and Bleaching. Chapter 4.* Ph.D. Thesis, UWI Mona, Dept of Life Sciences.

Ross, A. M. (2013d). *The Efficacy of Nursery Head-Starting in Restoration of Acropora cervicornis. Chapter 5.* Ph.D. Thesis, UWI Mona, Dept of Life Sciences.

Patterson, K. L., J. W. Porter, K. B. Ritchie, S. W. Polson, E. Mueller, E. C. Peters, D. L. Santavy, and G. W. Smith. 2002. "The Etiology of the White Pox, A Lethal Disease of the Caribbean Elkhorn Coral, *Acropora palmata*." In *Proceedings of the National Academy of Sciences of the United States of America* 99(13): 8725-8730.

Shafir, S., & Rinkevich, B. (2008). The Underwater Silviculture Approach For Coral Reef Restoration: An Emergent Aquaculture Theme. pp. 279-295. *In* Schwartz, S. H. (ed.). *Aquaculture Research Trends*. Nova Science Publishers Inc.

Smith, J. E., Smith, C. M., & Hunter, C. L. (2001). An Experimental Analysis of the Effects of Herbivory and Nutrient Enrichment on Benthic Community Dynamics on a Hawaiian Reef. *Coral Reefs, 19*, 332-342.

Soong, K., & Chen, T. (2003). Coral Transplantation: Regeneration and Growth of *Acropora* Fragments in a Nursery. *Restoration Ecology, 11*(1), 62-71.

Stephens, P. A., & Sutherland, W. J. (1999). Consequences of the Allee Efect For Behaviour, Ecolory and Conservation. T*rends in Ecology and Evolution, 14,* 401-405.

Sussman, M. Loya, Y., Fine, M., & Rosenberg, E. (2003). The Marine Fireworm *Hermodice carunculata* is a Winter Reservoir and Spring-Summer Vector for the Coral Bleaching Pathogen *Vibrio shiloi*. *Environmental Microbiology, 5*(4), 250-255.