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Tissue injury predicts colony decline in reef-building corals

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ABSTRACT: Tissue injury, in which the skeleton is stripped of living tissue, is common in reef-building corals and has potentially important demographic consequences. To examine the significance of tissue injury for natural populations, I monitored 1627 colonies in 30 taxa of Indo-Pacific branching corals at 3 to 5 mo intervals over a 2 yr period. Recent injury (inflicted within the few days prior to censusing) was a highly significant predictor of colony fate within 3 to 5 mo, for acroporid corals with small, compact branches (hispidose and corymbose growth forms). In contrast, colony size was not a significant predictor of fate for these corals after recent injury was included in the models. Both recent injury and colony size were significant predictors of fate for pocilloporids (small bushy growth form). Neither were good predictors of fate for arborescent acroporids (large, widely-spaced branches), even though recent injury was up to 3 times more common in these corals. Old injury (inflicted several weeks or more prior to censusing) covering >5% of the colony was a highly significant predictor of colony death within 3 to 5 mo for corymbose species. Colonies with both old and recent injuries were highly likely to die: 33 and 54% of colonies in separate censuses died within 3 mo. The predictive power of recent injury implies chronic or repetitive tissue loss and prolonged decline, since most recent injuries were small (<30 cm^2) and did not account for the colony decline per se. Since colony size was not as good a predictor of colony fate as recent injury for small-branched acroporids, size-based population models for these corals may be improved by incorporating tissue injury as an indicator of colony condition.

KEY WORDS: Demographic fate · Size-based population model · Growth form · Indicator · Log-linear modeling · Partial mortality · *Acropora* · Pocilloporid · Predictor · Reef-building coral · Injury

INTRODUCTION

As clonal organisms, reef-building corals grow by asexually budding new polyps and shrink when polyps die (injury). Surveys of injury in reef-building corals have shown it to be routine and ubiquitous. For example, Meesters et al. (1996) recorded injuries on 68% of colonies of 3 Caribbean species, and Hall (1998) recorded injuries on 73% of colonies of 3 Indo-Pacific species. Hughes & Jackson (1985) found injuries were responsible for more tissue loss than whole-colony mortality for 5 foliaceous Caribbean species. Potential demographic consequences of injury include reduced survivorship, growth and reproduction, colony shrinkage and increased fragmentation. Experimental studies have demonstrated reduced growth (Bak 1983, Meesters et al. 1994) and reproduction (Rinkevich & Loya 1989, Hall 1997a). Fragmentation has been linked with injury because areas devoid of live tissue become infested by boring sponges and prone to breakage (Highsmith 1982).

While experimental studies are useful for distinguishing the causes and consequences of injury, monitoring studies are useful for examining its ecological significance, because they take into account the vari-

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able susceptibility of colonies to agents such as disease and predators (Cumming 1996). Monitoring studies reveal temporal patterns of natural injury, and have shown injured colonies of foliaceous, branching and massive growth forms to be more susceptible to subsequent injury and whole-colony mortality (Hughes 1984, Hughes & Jackson 1985, Hughes & Connell 1987, Babcock 1991).

Soon after injury, colonies attempt to regenerate the missing tissue, but the rate of regeneration decreases with time and often stops before the tissue is fully regenerated (Bak & Steward-van Es 1980, Meesters et al. 1994). Thus, colonies can accumulate injuries over time, and injuries documented in surveys may represent impacts days, weeks, months or years old. Recent injury (days to weeks old) might be more closely linked to colony fate than old injury because colonies will be directing resources to regeneration, whereas colonies with older injuries may have ceased regenerating. No previous studies have examined the links between recent injury and colony fate, however. In the present study I examine the incidence of recent injury (days old) and the fates of injured colonies, for a group of 30 taxa of Indo-Pacific corals, and compare recent injury and old injury as predictors of colony fate. Recent injury is detectable in corals by the lack of algae growing on the exposed skeleton. For a few days after tissue removal the skeleton remains white, before becoming discoloured by settling algae. It grows progressively darker and after several weeks can be indistinguishable from injuries that may be years old (Cumming 1996). Recent injury is an important demographic variable because it represents the injury rate (total impact on coral colonies at a given time).

The study corals are Indo-Pacific branching corals of *Acropora* spp. and the family Pocilloporidae, which dominate many reef communities in terms of substrate cover and species diversity, and are ecologically important because their branching growth forms provide habitat for other species. Within the *Acropora*, distinct morphological groups (growth forms) are recognized (Wallace 1999, Veron 2000). In the following I compare 3: corymbose, hispidose and arborescent.

Colony size is the current standard demographic descriptor for reef-building corals; several studies have demonstrated a link between size and demography (Hughes & Jackson 1980, Hughes 1984, Done 1987, Babcock 1991). An assumption of the models is that within size classes individuals are demographically homogeneous, but in practice they may vary widely in characteristics such as age, history and incidence of injury (Hughes 1984, Hughes & Connell 1987, Babcock 1991, Cumming 1996, Meesters et al. 1997). I subdivided size classes into 2 groups based on presence/absence of recent injury, and show that injury status was a far stronger predictor of demographic fate than colony size for some growth forms (corymbose and hispidose acroporids). Both recent and old injury were highly significant predictors of colony fate for corymbose acroporids. Both recent injury and size were good predictors for pocilloporids and neither were good predictors for arborescent acroporids. For some branching growth forms, therefore, size-based population models that include a measure of colony condition may have improved accuracy and predictive power.

Study corals

The study sites were on a sheltered reef slope at Lizard Island, Great Barrier Reef (14° 40′ S, 145° 27′ E), at 4 to 9 m depth. The coral community was dominated by a diverse assemblage of branching *Acropora* spp. and pocilloporids (Cumming 1996, 1999), and in this respect was typical of many sheltered Indo-Pacific shallow reef slope communities (Done 1983). *Acropora* is the largest genus, comprising 114 species (Wallace 1999); 25 taxa were present in the study sites (Table 1). *Acropora* spp. are classified into growth forms based on colony shape and mode of branching (Wallace 1999). This study examines the 3 dominant growth forms in the study sites: hispidose and corymbose with small, closely packed branches, and arborescent with large, widely spaced branches (Table 1). Hispidose colonies have secondary branches (branchlets) projecting out from the main branch ('Christmas tree' morphology). Corymbose colonies have horizontal branches with short vertical branchlets, forming thick plates 6 to 8 cm in height. Arborescent colonies have large, tree-like branches. *A. divaricata* is caespito-corymbose (Wallace 1999); in the study sites it had a corymbose form with larger branches and branchlets than the other species, and is grouped as corymbose (Table 1). The fourth morphological group, the pocilloporids, have irregular, closely packed branches, forming dense bushes (Table 1).

A total of 1627 colonies in 5 hispidose, 12 corymbose, 8 arborescent and 5 pocilloporid taxa were monitored (Table 1). These 4 groups dominated the sheltered reef slopes at Lizard Island in terms of substrate cover (hispidose: 4.7%, corymbose: 5.4%, arborescent: 7.6%, pocilloporid: 2.6%, Cumming 1999). The high arborescent cover was due to numerous large *Acropora muricata* thickets more than 1 m^2 diameter (Cumming 1996). Corymbose colonies were the most abundant and speciose (Table 1).

Table 1. Number of coral colonies monitored in 30 m^2 sites at Mermaid Cove and Granite Bluff in 7 censuses from Feb 1992 to Feb 1994. Three pairs of similar species, difficult to distinguish in the field, were pooled. New recruits were identifiable to family level only. Nomenclature follows Wallace (1999)

Growth form	Species	Mermaid site 1	Mermaid site 2	Granite site 1	Granite site 2	Total
	Acropora					
Hispidose	*A. carduus*	0	0	0	3	3
	A. echinata	3	0	0	3	6
	A. elseyi	2	8	6	17	33
	A. longicyathus	7	9	2	2	20
	A. loripes	57	42	78	72	249
	Unidentified	0	0	2	0	2
Corymbose	*A. aculeus*	2	2	4	4	12
	A. caroliniana/granulosa	0	3	0	4	7
	A. cerealis	13	3	16	13	45
	A. divaricata	16	11	16	31	74
	A. latistella	1	0	2	0	3
	A. millepora	13	11	13	5	42
	A. nasuta	21	5	15	17	58
	A. sarmentosa	14	14	27	35	90
	A. secale	4	1	7	6	18
	A. selago	16	20	9	30	75
	A. tenuis	14	8	9	9	40
	A. valida	36	27	43	49	155
Arborescent	*A. austera*	0	0	2	0	2
	A. brueggmanni	3	1	0	0	4
	A. florida	0	3	4	0	7
	A. muricata	35	21	49	23	128
	A. grandis	6	6	3	4	19
	A. microphthalma	5	21	18	5	49
	A. intermedia	8	2	3	3	16
	A. yongei	1	2	1	2	6
Bushy	**Pocilloporidae**					
	Pocillopora damicornis	74	45	17	45	181
	Pocillopora edouxi/edwardsi	0	0	4	0	4
	Pocillopora verrucosa/meandrina	3	2	6	4	15
	Seriatopora histrix	4	4	7	1	16
	Stylophora pistillata	45	53	79	55	232
	Recruit	4	8	1	3	16
Total colonies monitored		407	332	443	445	1627

Agents of coral injury

The major source of tissue injury was predation by the corallivorous gastropods *Drupella cornus, D. fragum* and *D. rugosa*, which feed exclusively on live coral tissue and have strong preferences for acroporids and pocilloporids (Turner 1994, Cumming 1996, 1999, 2000a,b, Cumming & McCorry 1998). *Drupella* spp. were physically associated with 40% of the injured colonies, and field observations of frequent movements between colonies suggested they were responsible for at least 50% of injuries (Cumming 1996). *Drupella* spp. density was constant at 1 to 2 m^{-2} during the present study, and they were observed preying on all the species listed in Table 1 (Cumming 1996).

The corallivorous seastar *Acanthaster planci* and disease were also present. *Acanthaster* spp. and *Drupella* spp. predation were often distinguishable, based on several criteria (Cumming 2000a). Disease was identified by obvious black bands (in black-band disease) or by large areas with a mottled appearance, created by uneven death of tissues and subsequent algal growth. Together, these 3 agents caused most of the natural injury in this coral assemblage.

MATERIALS AND METHODS

Monitoring corals. In February 1992, I permanently marked four 30 m^2 sites at 5 to 8 m depth on the leeward fringing reef slopes at Mermaid Cove and

Granite Bluff, Lizard Island (see Cumming 1999). Each square metre was mapped separately (30 maps for each site), and each coral colony numbered (*Acropora* spp. and pocilloporids). On 7 censuses at 3 to 5 mo intervals from February 1992 to February 1994, colony size and size of injuries were recorded for each colony. Their demographic fates: growth, shrinkage, death or stasis, were recorded on each of the 6 repeat censuses. Colonies, including loose fragments, were classified into size classes based on maximum diameter of living tissue: Size Class 1: 0–5 cm; 2: >5–10 cm; 3: >10–20 cm; 4: >20–30 cm; 5: >30–40 cm, and 6: >40 cm. 'Shrinkage' and 'growth' therefore indicate substantial changes in size because they represent a change of size class. Many colonies recruited after monitoring began, many died and a few moved during the 2 yr period, so a large proportion were not present for the entire census period. Specimens of many of the species are lodged in the Museum of Tropical Queensland, Townsville, Australia; specimen numbers 48292–48328, 49225–49245.

Preliminary surveys of the study reef revealed that *tissue* injuries, in which tissue is removed but the skeleton remains intact, comprised the majority of injuries for branching corals. Skeletal breakage was uncommon. Most tissue injuries were not surrounded by live tissue but were bordered by either dead skeleton or the edge of the colony. Subsequent sampling and analysis was concentrated on these types of injuries only.

Recent injury is defined as an area of bare white skeleton that has not yet been discoloured by the ubiquitous turf algae that settle on all exposed skeleton. Turf algal colonisation rate was measured by placing freshly injured pieces of *Acropora muricata* (grazed by *Drupella* spp.) on a patch reef at Lizard Island at 3 m depth in January/February 1992. Algae discoloured these after 3 to 5 d. I subsequently defined recent injury as injury inflicted within the 3 to 5 d prior to censusing, although it is recognised that algal growth rate might vary between sites and might decline as water temperature drops, giving recent injuries a longer life span in winter. Longer-term observations indicated at least 3 to 4 wk for a recent injury to become covered with algae to the point where it was indistinguishable from old injury (and the surrounding reef substrate).

Predicting the demographic fates of colonies. The full data matrices, with sites pooled, were 4-way contingency tables containing counts of colonies making state/fate transitions, with growth form, size and recent injury as state variables (describing initial state of the colony) and fate as the fourth factor. Separate matrices were constructed and analysed for each pair of census dates. Colony size was not recorded in July 1992 so state/fate transitions involving this census were not constructed.

To determine whether the state variables were significant predictors of fate, the appropriate statistical techniques are hierarchical log-linear models (Caswell 1989). For all tests, the HILOGLINEAR procedure in SPSS for Windows Release 6.0 was used (SPSS 1993) and comparisons were based on the likelihood ratio:

$$G^2 = -2\sum_{\text{cells}}(\text{observed})\log(\text{observed/expected})$$

Analyses were included only if no more than 20% of expected values were less than 1, following Fingleton (1984). Pooling was used to achieve sample sizes large enough to overcome this problem. Colony size classes were pooled into maximum diameters of 0 to 20 cm and >20 cm. For fates, shrinkage and whole-colony mortality were pooled, and growth and stasis were pooled. This high level of pooling was necessary because injured colonies comprised only 5 to 10% of the population at any census, so sample sizes for injured colonies were sometimes low (see 'Results').

The growth forms had significantly different responses (see 'Results'), so I decomposed the 4-way contingency tables and analyzed separate 3-way tables for each growth form. Using standard notation for hierarchical log-linear models (Caswell 1989), the null hypothesis that fate is independent of injury and size is written as *IS,F*, where *I* = recent injury: present or absent, *S* = colony size: 0 ≤ 20 cm or >20 cm, *F* = fate of the colony at the subsequent census: stasis/growth or shrinkage/death. The presence of the interaction, *IS*, implies the presence of all lower order terms involving the components of that interaction, so *IS,F* asserts that fate is independent of the 2 state variables, injury and size, and of their interaction. The statistical significance of a term in the model is assessed by comparing the value of G^2 and the degrees of freedom of a main effect or interaction with their values following addition or deletion of that term from a null model. Testing terms against this null model examines the effects of injury and size against a model containing no other factors. A better test is against a model in which the effect of the other term has already been added; the test is then whether a term provides any additional information after the other has been added (Caswell 1989). Therefore, in all tests, I used the null model *IS,SF* to test for the effect of recent injury after size has been included, and the model *IS,IF* to test for the effect of size after recent injury has been included.

Analyses for old injury were restricted to corymbose colonies over 2 time periods, where sample sizes were large enough.

The log-linear model used to test the null hypothesis that all growth forms responded in the same way was: *ISF,ISM*, where *M* = colony growth form: hispidose, corymbose, arborescent or pocilloporid. This model

tests for differences between transition matrices and differs from the above model, which is applied to a single transition matrix. The test is one of conditional independence, and asserts that fate depends on injury and size, but is independent of growth form (Caswell 1989).

RESULTS

Size and frequency of injuries

The proportion of colonies with recent injury was consistent between censuses (Fig. 1). Averaged over all censuses, 5.2% of hispidose, 6.0% of corymbose, 13.7% of arborescent and 5.7% of pocilloporid colonies had recent injury. Recent injury was significantly more common for arborescent colonies than for the other growth forms on 4 census dates (Fig. 1). Most recent injuries were small; 50% were <10 cm^2 and 82% were <30 cm^2 (Fig. 2), indicating that it was unusual for colonies to lose large areas of tissue in a short time. These small injuries usually involved tissue loss from just 1 or a few branches within a colony. All growth forms had this pattern of injury size (Fig. 3).

Twenty-one percent of all colonies had recent injuries on at least 1 of the 7 censuses: 18.3% of hispidose, 22.0% of corymbose, 35.7% of arborescent and 14.2% of pocilloporid colonies.

The proportion of colonies with old injury on >5% of the skeleton was 14.6% in December 1992, 16.5% in December 1993 and 21.4% in February 1994. It varied between growth forms, and was lowest in corymbose colonies (means: 6.4 to 12.3%) and highest in arborescent colonies (means: 26.5 to 37.6%; Fig. 4). Old injury was 1.5 to 4 times more common in arborescent colonies than the other growth forms, and a significantly higher proportion of arborescent colonies had old injury in the 1992 and 1994 censuses (Fig. 4). In February 1994, 50 and 49% of arborescent colonies had old injury in 2 of the 4 sites.observed

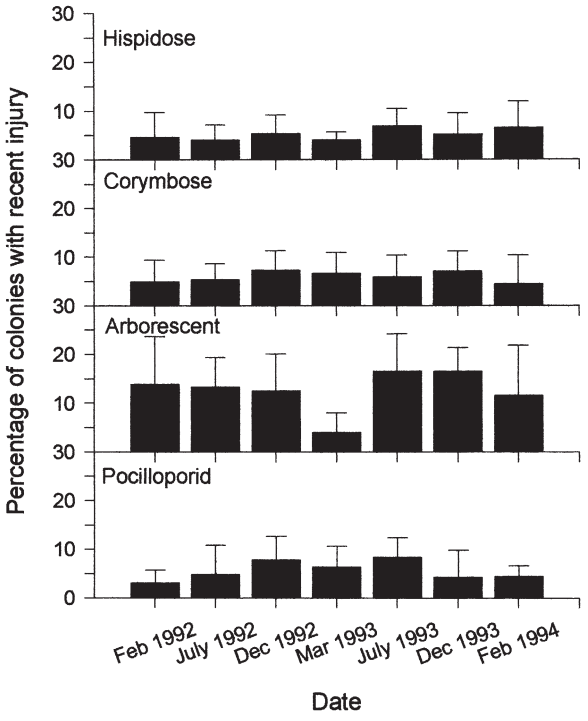

Fig. 1. Percentage of colonies with recent injury, by growth form and census date. Each 30 m^2 site is 1 replicate; n = 4 for all dates except March 1993 when 1 site was not censused due to poor weather. Total colonies censused: hispidose, Feb 1992 n = 228, Jul 1992 n = 246, Dec 1992 n = 238, Mar 1993 n = 169, Jul 1993 n = 234, Dec 1993 n = 237, Feb 1994 n = 238; corymbose, Feb 1992 n = 467, Jul 1992 n = 488, Dec 1992 n = 469, Mar 1993 n = 342, Jul 1993 n = 427, Dec 1993 n = 400, Feb 1994 n = 383; pocilloporid, Feb 1992 n = 225, Jul 1992 n = 254, Dec 1992 n = 265, Mar 1993 n = 199, Jul 1993 n = 272, Dec 1993 n = 292, Feb 1994 n = 305; arborescent, Feb 1992 n = 166, Jul 1992 n = 175, Dec 1992 n = 169, Mar 1993 n = 99, Jul 1993 n = 165, Dec 1993 n = 162, Feb 1994 n = 153. Vertical bars are 95% confidence intervals. A significantly higher proportion of arborescent colonies were injured on 4 censuses: Feb 1992, Jul 1992, Jul 1993 and Dec 1993 (1-way ANOVA, Student-Newman-Keuls tests)

Fates of injured colonies

Substantial shrinkage (a reduction of size class), and whole-colony mortality (both referred to subsequently as 'decline') are presented separately in Table 2. Death of large colonies did not occur for arborescents and was infrequent for hispidose and pocilloporids, but was substantial for corymbose colonies. From March to July 1993 death of large corymbose colonies was 3 to 8 times higher (17.3%) than the other periods. Pocilloporids were the only group that conformed to the expectation established by previous studies of reef-building corals, that smaller colonies have higher rates of death and larger colonies have higher rates of shrinkage. Small hispidose and arborescent, and large corymbose colonies, had exceptions to this pattern in 1 to 2 periods each.

The null hypothesis of conditional independence, that fate was independent of growth form, was rejected for all 4 dates (December 1992 to March 1993: G^2 = 25.043, p = 0.015; March 1993 to July 1993: G^2 = 32.647, p = 0.001; July 1993 to December 1993: G^2 = 22.277, p = 0.035; December 1993 to February 1994: G^2 = 23.557, p = 0.023; df = 12 for all tests). This indicates that fates of injured colonies varied between growth forms, and each growth form was therefore examined separately using the data matrices shown in Table 3.

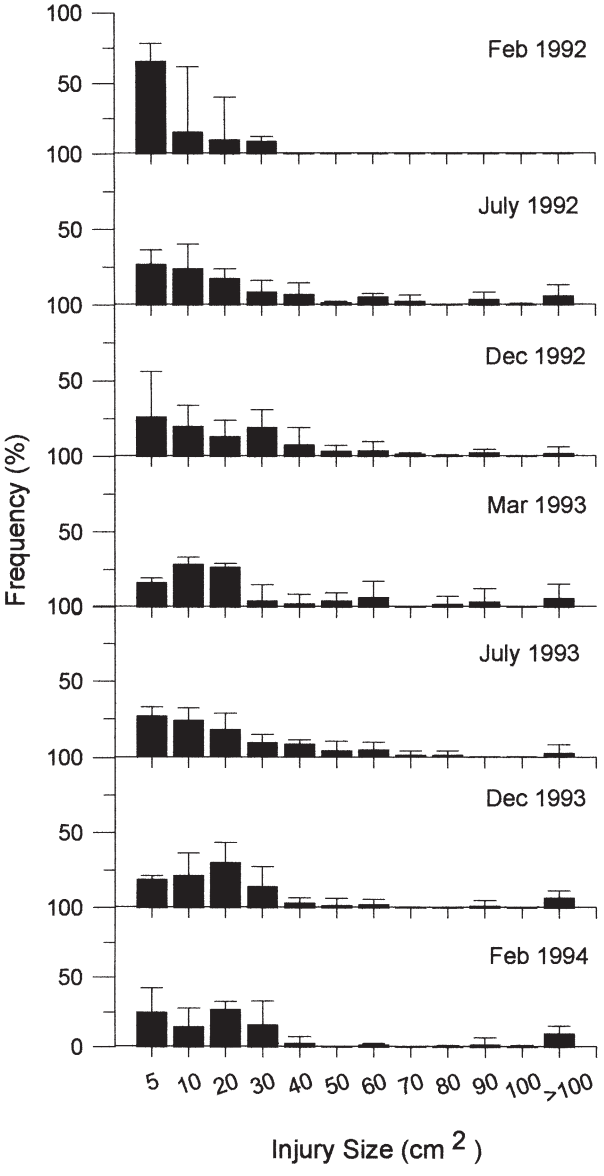

Fig. 2. Size-frequency distributions of recent injuries, defined as white areas of bared skeleton not yet discoloured by settling algae, and up to approximately 5 d old (see 'Materials and methods'). Each 30 m^2 site is 1 replicate; n = 4 except in Feb 1992 when injuries were not measured in 2 plots, and Mar 1993 when 1 plot was not censused due to poor weather. Values on the *x*-axis are upper bounds of scar size classes. Vertical bars are 95% confidence intervals

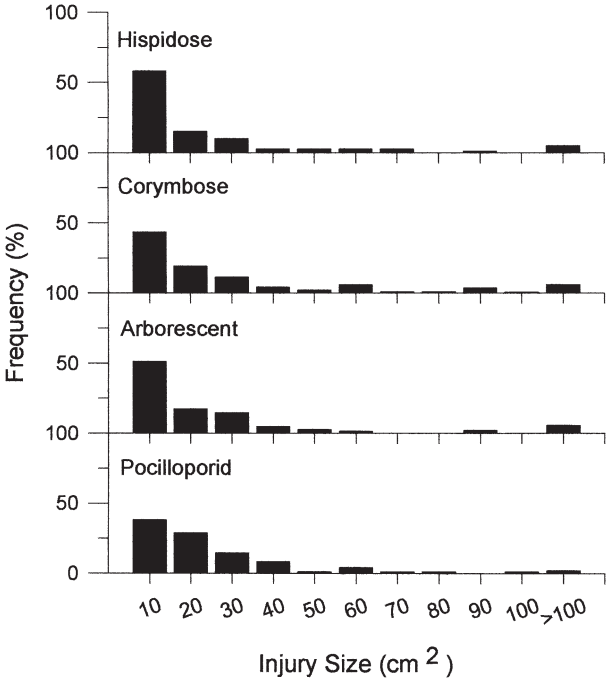

Fig. 3. Size-frequency distributions of recent injuries, by growth form. Data are pooled for all sites and all census dates. Sample sizes: hispidose, n = 79; corymbose, n = 165; pocilloporid, n = 97; arborescent, n = 143. Distributions for all growth forms are similar (Homogeneity test: χ^2 = 26.07, df = 18, p = 0.1; categories 70 > 100 pooled)

Fig. 5 shows that injured hispidose, corymbose and pocilloporid colonies were much more likely to undergo decline than uninjured colonies. In contrast, injury and decline were not related in arborescent colonies; rates of decline were similar for different colony sizes and for injured and uninjured colonies. Larger injured colonies were less likely to decline than smaller injured colonies of corymbose acroporids and pocilloporids, and were more likely to decline for hispidose colonies.

Recent injury was a highly significant predictor of colony fate for hispidose, corymbose and pocilloporid colonies (Table 4). Recent injury was a much stronger predictor of fate than was colony size for hispidose and corymbose forms. After recent injury was included in the models, size was not significant for hispidose colonies. For corymbose colonies, it was not significant in 2 analyses, and marginal in 2 analyses. The interaction between size and injury was significant for corymbose colonies from December 1992 to March 1993, when the proportion of uninjured colonies declining increased with colony size, while the proportion of injured colonies declining decreased with colony size (Fig. 5).

For arborescent colonies, injury was not a significant predictor of colony fate, and size was marginally significant for one analysis. For pocilloporids, both injury and colony size were significant predictors of fate, with smaller colonies and injured colonies having higher rates of decline (Table 4; Fig.5).

Corymbose colonies with both recent and old injuries were highly likely to die within 3 to 5 mo (33% and 54%), and were more likely to die than colonies with old injuries only (Table 5, Fig. 6). Old injury was a highly significant predictor of colony fate after recent

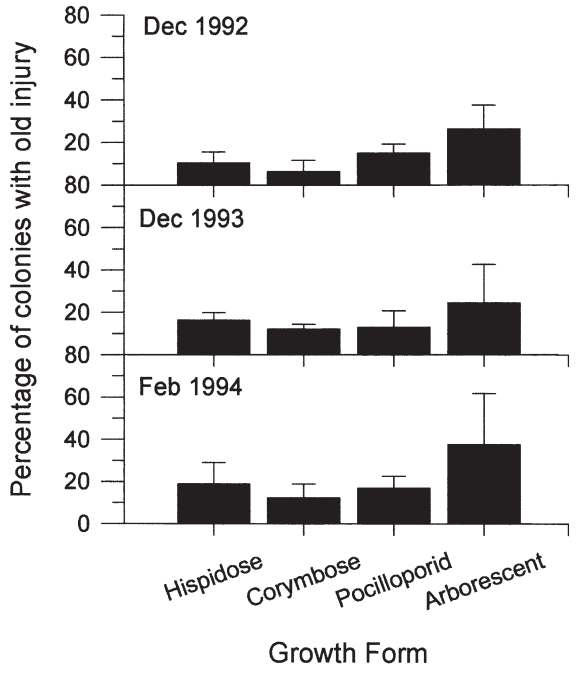

Fig. 4. Percentage of colonies with old injury, by growth form and census date. Each 30 m^2 site is 1 replicate (n = 4). Vertical bars are 95% confidence intervals. Total colonies censused: hispidose, Dec 1992 n = 212, Dec 1993 n = 197, Feb 1994 n = 192; corymbose, Dec 1992 n = 436, Dec 1993 n = 351, Feb 1994 n = 338; pocilloporid, Dec 1992 n = 225, Dec 1993 n = 255, Feb 1994 n = 253; arborescent, Dec 1992 n = 124, Dec 1993 n = 119, Feb 1994 n = 93. A significantly higher proportion of arborescent colonies had old injuries in the 1992 and 1994 censuses, and there were no differences in the 1993 census (1-way ANOVA on arcsin transformed data, Student-Newman-Keuls tests)

injury was included, whereas recent injury was only marginally significant in 1 of the 2 tests (Table 6).

DISCUSSION

This study has shown recent injury to be a highly significant predictor of colony decline (shrinkage or death) for hispidose and corymbose acroporids, and pocilloporids, over time scales of 3 to 5 mo. Thus, recent injury is an important demographic variable for these groups of compact, branching corals. It was a much stronger predictor than colony size for the acroporids, despite colony size being an important demographic variable for reef-building corals (Loya 1976, Hughes 1984, Hughes & Jackson 1985, Hughes & Connell 1987, Babcock 1991). Thus, although it is now customary to classify corals by size in monitoring studies (e.g. DeVantier et al. 1998), including a measure of colony condition with respect to injury should streamline the accuracy and predictive power of models for compact branching corals.

A powerful application of the present study will be in developing indices of reef status and 'health', an increasingly important focus in the current climate of reef degradation. Reef monitoring programs have begun to incorporate colony injury as a measure of coral reef vitality (Dustan 1994, Ginsburg et al. 1996, AGRRA 2000 [http://coral.aoml.noaa.gov/agra/index.html]). To predict future change (or lack of change), a link between missing tissue and colony decline is assumed, and this is also supported by Hughes & Jack-

Table 2. Percentage of colonies that shrank and died, classified by colony size (maximum diameter of living tissue)

Growth form	Time interval	Small (0–20 cm)			Large (>20 cm)		
		%Shrank	%Died	n	%Shrank	%Died	n
Hispidose	Dec 92–Mar 93	0	2.6	78	7.1	0	14
	Mar 93–July 93	7.6	5.1	118	5.6	0	36
	July 93–Dec 93	3.1	4.3	162	10.8	1.5	65
	Dec 93–Feb 94	4.1	3.0	169	4.8	1.6	63
Corymbose	Dec 92–Mar 93	0.5	5.8	207	12.5	6.2	80
	Mar 93–July 93	2.5	11.9	243	6.2	17.3	81
	July 93–Dec 93	3.2	11.8	314	5.3	4.2	95
	Dec 93–Feb 94	2.7	6.1	293	7.1	2.0	99
Arborescent	Dec 92–Mar 93	6.7	4.4	45	5.7	0	35
	Mar 93–July 93	3.4	6.9	29	3.8	0	26
	July 93–Dec 93	6.4	11.5	78	9.9	0	71
	Dec 93–Feb 94	7.2	7.2	83	10.6	0	66
Pocilloporid	Dec 92–Mar 93	2.4	6.4	125	3.2	0	31
	Mar 93–July 93	2.1	19.7	142	2.5	0	40
	July 93–Dec 93	5.3	17.9	190	6.2	3.1	64
	Dec 93–Feb 94	0.9	13.2	212	13.6	0	66

Table 3. Matrices of colonies making state-fate transitions, classified by the presence of recent injury, colony size (small or large) and growth form. Colony size is maximum diameter of living area. Only matrices with ≤20% of expected values <1 are included, following Fingleton (1984). Growth and shrinkage involve substantial change in size because they were measured as a change in size class (see 'Materials and methods')

Growth form	Time interval	Type of transition	Small (0–20 cm)		Large (>20 cm)	
			Absent	Present	Absent	Present
Hispidose	July 93–Dec 93	Stasis/growth	144	6	52	5
		Shrinkage/death	10	2	5	3
Corymbose	Dec 92–Mar 93	Stasis/growth	192	2	59	6
		Shrinkage/death	7	6	10	5
	Mar 93–July 93	Stasis/growth	206	2	57	5
		Shrinkage/death	27	8	11	8
	July 93–Dec 93	Stasis/growth	261	6	80	6
		Shrinkage/death	39	8	6	3
	Dec 93–Feb 94	Stasis/growth	262	5	78	12
		Shrinkage/death	19	7	5	4
Arborescent	July 93–Dec 93	Stasis/growth	60	4	44	20
		Shrinkage/death	13	1	2	5
	Dec 93–Feb 94	Stasis/growth	64	7	43	16
		Shrinkage/death	11	1	6	1
Pocilloporid	July 93–Dec 93	Stasis/growth	140	6	50	8
		Shrinkage/death	37	7	4	2

son (1985), Hughes & Connell (1987) and Babcock (1991), who have shown a significantly higher likelihood of death in colonies with missing tissue, for foliaceous, massive and branching (*Acropora* spp. and *Pocillopora damicornis*) corals respectively.

Demographic significance of colony morphology

Branching morphology is an important ecological variable. Arborescent acroporids were 2 to 3 times more likely to be injured than small-branched colonies, but injured arborescent colonies were not more likely to decline, unlike the other growth forms. This reflects a fundamental difference between the life histories of arborescent and other branching growth forms. Injury may even benefit arborescents by increasing rates of asexual reproduction by fragmentation, since branches not covered by live tissue are more likely to break (Highsmith 1982).

Other life history traits in which arborescent colonies differed from the other growth forms are: (1) similar numbers of small and large colonies, whereas small colonies were 2 to 3 times more numerous than large colonies for the other growth forms, (2) no mortality of large colonies between censuses, and (3) similar likeli-

Table 4. Log-linear analyses of the effects of recent injury and colony size on fate of 4 common growth forms, over 3 to 5 mo intervals. df = 2 for main effects, df = 1 for interactions. Each factor was examined after the other factor had been included in the model (see 'Materials and methods'). Only analyses with ≤20% of expected values <1 are included, following Fingleton (1984). Significant effects (α = 0.05) are in **bold**. Sample sizes are given in Table 3

Growth form	Time interval	Recent injury		Size		Interaction	
		G^2	p	G^2	p	G^2	p
Hispidose	July 93–Dec 93	6.509	**0.011**	0.561	0.454	0.045	0.831
Corymbose	Dec 92–Mar 93	25.822	**<0.0001**	4.008	**0.045**	6.642	**0.010**
	Mar 93–July 93	32.310	**<0.0001**	0.263	0.608	1.629	0.202
	July 93–Dec 93	18.634	**<0.0001**	3.785	0.052	0.086	0.769
	Dec 93–Feb 94	22.917	**<0.0001**	1.449	0.229	1.834	0.176
Arborescent	July 93–Dec 93	2.970	0.085	4.014	**0.045**	1.292	0.256
	Dec 93–Feb 94	0.479	0.489	1.301	0.583	0.152	0.697
Pocilloporid	July 93–Dec 93	7.415	**0.006**	8.747	**0.003**	0.097	0.755

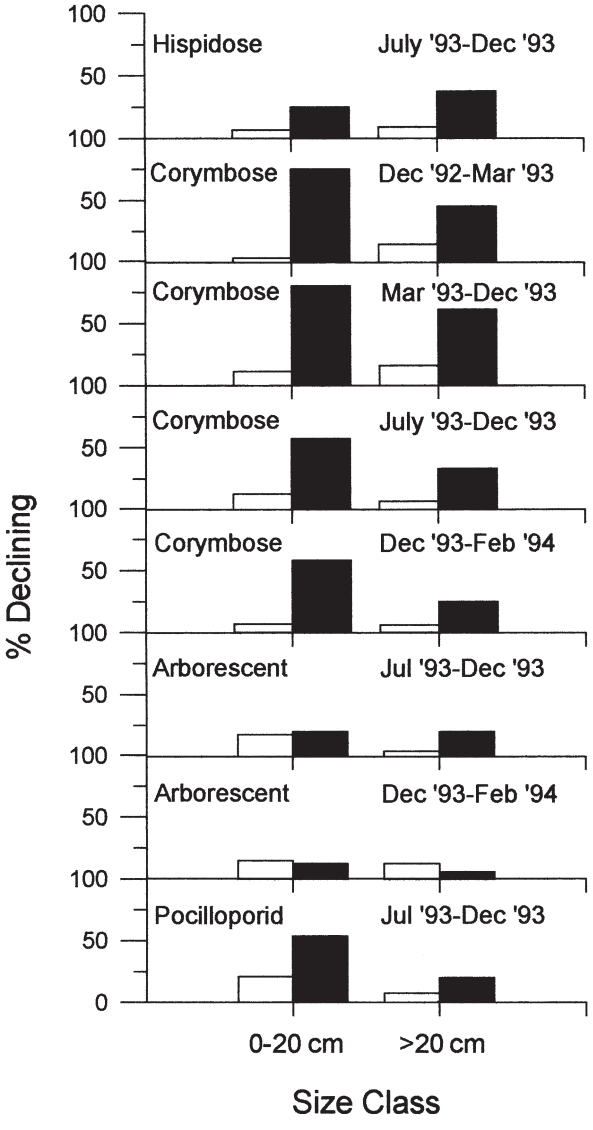

Fig. 5. Percentage of colonies that declined (shrank or died) within 3 to 5 mo, classified by presence/absence of recent injuries, colony size, growth form and census date. White bars: recent injury absent; black bars: recent injury present. Sample sizes are given in Table 3. Percentage declining is calculated within categories; e.g. for hispidose July 1993 to Dec 1993, 25% of small colonies with recent injury declined, calculated as number of injured colonies shrinking/dying (2) divided by total number of injured colonies (8)

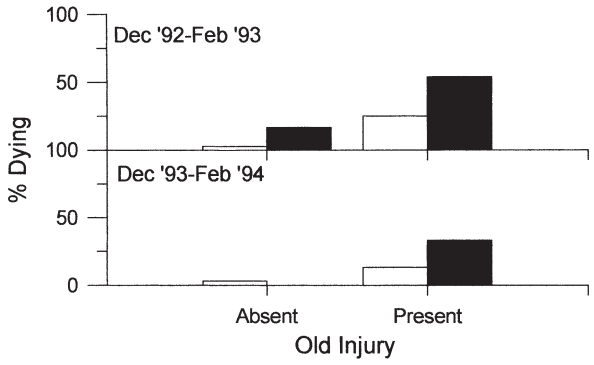

Fig. 6. Fates of corymbose colonies with recent and old injuries, within 3 to 5 mo. Sample sizes are given in Table 5. White bars: recent injuries absent; black bars: recent injuries present

Table 5. Frequencies of corymbose colonies making state-fate transitions, classified by the presence/absence of recent injury, and old injury comprising >5% of the colony

	Recent injury:	Absent		Present	
Time interval	Old injury:	0–5%	>5%	0–5%	>5%
Dec 92–Mar 93	Alive	252	6	5	6
	Dead	7	2	1	7
Dec 93–Feb 94	Alive	324	26	10	12
	Dead	11	4	0	6

hood of shrinkage and death for small colonies, whereas death was much more likely than shrinkage for small corymbose and pocilloporid colonies.

The trend established for massive and foliaceous corals of higher death rates in smaller colonies and higher shrinkage rates in larger colonies (Hughes & Jackson 1980, Hughes 1984, Done 1987, Babcock 1991), held for arborescent and hispidose acroporids, and pocilloporids. But for corymbose acroporids, larger colonies had higher death rates than smaller colonies in 2 of the 4 census periods. These results indicate considerable variation in the population dynamics of Indo-Pacific branching corals.

Demographic significance of recent tissue injury

The data presented here represent the first data on injury rate for these reef-building corals. The fact that the sizes and frequencies of injuries were similar in all 7 censuses indicates a degree of predictability. Rates

Table 6. Results of log-linear analyses of the effects of recent and old injury on corymbose colony fate. df = 2 for main effects, df = 1 for interactions. Significant effects ($\alpha = 0.05$) are given in **bold**. Sample sizes are in Table 5

Time interval	Recent injury		Old injury		Interaction	
	G^2	p	G^2	p	G^2	p
Dec 92–Mar 93	3.517	0.061	7.651	**0.006**	0.215	0.643
Dec 93–Feb 94	1.628	0.202	9.278	**0.002**	1.682	0.195

ranged from 5% (hispidose) to 14% (arborescents) of colonies with recent injuries in a census, and 14% (pocilloporids) to 36% (arborescents) injured at least once in 2 yr.

Most of the injuries were too small to be responsible for the substantial colony decline that they foreshadowed in the small-branched groups (82% were <30 cm^2). This suggests that recent injuries are often not isolated events but signals of prolonged decline, involving chronic or repetitive injuries. Alternatively, colonies have severe reactions to small injuries, leading to physiological decline over the following few months. Such adverse reactions have not been noted in experimental studies (Meesters et al. 1994, Hall 1997a,b).

Two behavioural traits of the predators *Drupella* spp. lead to their prey colonies being repeatedly or chronically injured. First, groups of *Drupella* spp. reside on colonies for periods ranging from days to months, grazing small portions each night. Second, *Drupella* spp. require a colony to have some dead branches in order to occupy it because they do not crawl over live coral tissue (Cumming 1999, 2000a). An injured colony is thus susceptible to predation by *Drupella* spp.

Old injuries, a measure of previous colony shrinkage, were good predictors of death for corymbose colonies, indicating expected future decline. Colonies with both recent and old injuries had a much greater probability of death than colonies with either one or the other. The tendency of previously injured colonies to shrink further (and die outright) has also been shown for the massive corals *Goniaspera aspera*, *G. favulus* and *Platygyra sinensis* by Babcock (1991) and for *Pocillopora damicornis* and pooled species of *Porites* (3 species) and *Acropora* (11 species) by Hughes & Connell (1987). Hughes (1984) showed that injured colonies of the foliaceous Caribbean species *Agaricia agaricites* were almost twice as likely to be injured again the following year.

These results all support the concept of natural colony decline, or 'senescence', despite the traditional belief that colonial organisms do not undergo aging or senescence (Harper 1980). Babcock (1991) suggested a form of environmental senescence related to coral colony growth and morphology. Rinkevich & Loya (1986) reported endogenous senescence; they detected decreases in reproduction and growth in the months preceding whole-colony death of *Stylophora pistillata*, with tissue injury beginning about 3 mo before the death of the colony.

Recent injuries may point to colonies that are physiologically weak, with reduced defenses and therefore susceptible to attack by predators, competitors or disease, or with less optimal microhabitat characteristics, leading to more abiotic impacts. Monitoring colonies *in situ* describes the ecological significance of injury for colonies exposed to all of these potential impacts.

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