# INJURY AND REGENERATION OF COMMON REEF-CREST CORALS AT LIZARD ISLAND, GREAT BARRIER REEF, AUSTRALIA

Thesis submitted by
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in partial fulfilment of the requirements for a Doctor of Philosophy Degree in the Department of Marine Biology of James Cook University.

# THESIS DEDICATION

This thesis is dedicated to the loving memory of my father and grandfather.
To my father, thank-you for always believing in my academic abilities and for encouraging me to further my education, I hope I have made you proud.
To my grandfather, my sincere thanks for the financial support that has enabled me to continue my studies and for showing me that quiet achievers can make a difference.

# ABSTRACT:

Corals are frequently injured by natural processes and human activities. The response of scleractinian corals to damage is dependent on the nature and extent of damage, the characteristics of the injury, the life-histories of the coral, and the prevailing abiotic and biotic conditions. In this thesis I have examined several aspects of injury including (1) the nature and extent of natural injury, (2) the response of corals to injuries with different characteristics and (3) the influence of morphology and life-history in response to damage.

The spatial and temporal patterns of coral injury were recorded to determine the nature and extent of damage in common reef-crest corals at Lizard Island. The total amount of partial mortality on reef-crest corals was low (<2%) although there was a three-fold difference among sites. Sites with low partial mortality had reef-crest assemblages dominated (both numerically and in cover) by tabular and bushy corals. These corals have low levels of partial mortality, and on average, fewer small colonies with injuries. Conversely, the site where the partial mortality was three times higher had a lower abundance and cover of tabular corals, and an increase in the number and cover of massive and digitate corals. Massive and digitate corals, on average, have a higher amount of partial mortality and more small colonies with injuries.

The amount of injury present on a colony at a particular time is a balance between vulnerability (i.e. frequency of injury and resilience to damage) and recovery rate. An investigation into the patterns of injury over time showed that vulnerability to damage and recovery of injuries was species specific. In general *Goniastrea retiformis* had a high number of old injuries, a slow regeneration rate, and was injured infrequently, suggesting that injuries tended to accumulate on colonies over time. The addition of new injuries was also low for *Acropora gemmifera*, however colonies had few pre-existing injuries and faster recovery rates, reducing the accumulation of injuries on colonies. The injury dynamics for *A. hyacinthus* differed between censuses because of a change in injury regimes from routine to catastrophic, the latter regime caused by an outbreak of *Acanthaster planci*. Under routine conditions, there were few pre-existing injuries on colonies, a moderate addition of new injuries, and rapid regeneration, suggesting a fast turn-over of injuries. Under catastrophic conditions, there were many more pre-existing injuries, a high number of new injuries, and more injuries increasing in size than recovering, resulting in an accumulation of injuries.

The regeneration of injuries was influenced by the characteristics of the injury including initial size, type, and position within the colony The complete regeneration of an injury was more probable for small injuries (0 - 4 $cm^2$) than larger injuries. However, recovery rates were also dependent on the type of injury as scraping injuries had a much faster regeneration rate than tissue mortality or breakage. Additionally, recovery was influenced by the position of injuries within colonies for one species *Porites mayeri* where the rate of regeneration of central injuries was greater than edge injuries. Conversely, the recovery of central and edge injuries was similar for *A. robusta, A. hyacinthus, A. palifera, Pocillopora damicornis*, and *Porites lichen*. Variations in levels of partial mortality, zones of tissue from which regeneration can take place, degrees of settlement by other organisms, intensities of damage, and amounts of resources available for regeneration all contributed to the differences in recovery rates found between injuries with varying characteristics.

The regeneration of injuries requires resources that are in limited supply. In this study, there was a marked effect of injury on reproduction for *A. hyacinthus*, *A. gemmifera* and *G. retiformis*, inferring a trade-off between reproduction and regeneration. Presumably the resources usually available for gamete production are being reallocated towards polyp regrowth and defence against fouling organisms. In contrast, injury had no effect on the survival or growth of colonies over nine months for the three species. This result suggests that future reproduction is being preserved through the iteration of new polyps but at the expense of current reproduction. It also suggests that these species are resistant to damage since their survival was unaltered by damage in the short-term.

Species resistant to damage have evolved two alternative, but not mutually exclusive, strategies in response to injury. Corals can invest resources in defensive mechanisms to avoid damage (avoidance strategies) or regrow lost parts after injury has occurred (tolerance strategies). Both strategies were utilised by corals in this study, although the amount of investment in either strategy varied. Generally, the longer-lived species, *G.*

*retiformis* and *A. gemmifera*, seemed to invest more resources towards defence than the shorter-lived *A. hyacinthus* since the number of new injuries present on colonies was higher for the latter species. Conversely, the shorter-lived coral invested more in tolerance strategies by responding to infrequent damage events or minimal tissue losses with rapid regrowth. The cost of such a strategy is that shorter-lived species are more vulnerable to repetitive injury.

Experimental studies showed that branching species had more regrowth potential than massive and semi-massive species supporting the hypothesis by Jackson (1979) that morphology plays a role in the pattern of investment in regeneration and defence. The morphology of a coral influences its longevity, reproductive output, growth rate, and other life-history processes including regeneration. Consequently, the morphological strategy of an organism has evolved over time in response to a large number of biotic and abiotic processes including partial mortality.

In conclusion, this study on injury and regeneration of scleractinian corals has increased our knowledge of the underlying mechanisms that affect the recovery of corals from damage, and has provided a basis for understanding the consequences of different injury regimes on coral reefs. This is important because injury can adversely affect corals at the individual, population and community level and thus impact on the general ecology of coral reefs.

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# CHAPTER 1: GENERAL INTRODUCTION

## 1.1 INTRODUCTION

Coral reefs are one of the most diverse and complex ecosystems in the world and provide important resources for a large number of taxa, including humans (Hatcher *et al.* 1989). The increasing level of human exploitation of these resources in the past two decades has resulted in the degradation of many reefal ecosystems (Colloquium and forum on global aspects of coral reefs, Miami 1993). Unfortunately, while the impact of humans on reefs has been well documented, our understanding of the mechanisms of effect, and their consequences has been much less substantial (Hatcher *et al.* 1989). With the exception of a few notable studies, mainly in the Caribbean and the Red Sea, this lack of understanding of mechanisms and their specific consequences is particularly true for studies of damage in scleractinian corals. Monitoring programs assessing the status of coral reefs have focused on the loss of species diversity and coral cover without any in-depth knowledge of species-specific sensitivities to damage, or the complex processes influencing recovery. The general objective of this thesis is to further our knowledge of the processes associated with injury and regeneration for scleractinian corals.

Scleractinian corals are modular organisms that grow by the iterations of polyps and shrink through the loss of polyps (e.g. Hughes and Jackson 1985; Babcock 1991). The loss of polyps through damage from an individual colony resulting in a dead patch is known as partial mortality. Partial mortality in corals is caused by both natural processes such as predation, competition, disease, and physical disturbances, as well as anthropogenic activities including diving, fishing, boating, mining and coastal development (e.g. reviews by Connell 1973; Brown and Howard 1985; Craik *et al.* 1990; Grigg and Dollar 1990). The ability of corals to recover from damage (regeneration) is dependent on the nature and extent of damage, the resulting characteristics of the injury, the life history of the coral, and the prevailing biotic and abiotic conditions (Bak *et al.* 1977; Bak and Steward-Van Es 1980; Bak 1983; Liddle and Kay 1987; Liddle 1991; Meesters *et al.* 1992; Meesters and Bak 1993, 1995; Meesters *et al.* 1997b; Oren *et al.* 1997).

### 1.1.1. NATURE AND EXTENT OF DAMAGE

The nature and extent of damage to corals is highly variable (Bythell *et al*. 1993; Meesters *et al*. 1996, 1997a). This variability results from the unpredictable nature of the large number of agents that can cause damage in corals, as well as the varying susceptibilities of corals to injury. For example, the agents of coral damage are often patchy in nature and operate to different extents, both within and between different habitats and depths (e.g. *Drupella*, Cumming 1996; Tropical storms, Bythell *et al*. 1993). Vulnerability to damage is often associated with morphology since the shape of a coral significantly influences the type of interaction that it will have with the surrounding environment (Jackson 1979). Differences in physical defenses (e.g. skeletal density of branches and polyps, nematocyst densities and spiky protrusions) among morphologically similar species may also change their vulnerability to damage (Bythell *et al*. 1993; Meesters *et al*. 1996, 1997a).

### 1.1.2. CHARACTERISTICS OF INJURIES

In corals, the ability to recover from damage is partially determined by the characteristics of the injury (e.g. size, shape, type and position of injury within the colony). Corals have a greater ability to regenerate small injuries than large ones (Bak *et al*. 1977; Bak and Steward-Van Es 1980; Oren *et al*. 1997), most probably due to the fact that injuries with a high perimeter to injury area ratio recover faster than injuries with a low perimeter to injury area ratio, at least for injuries surrounded by a large amount of healthy tissue (Meesters *et al*. 1997b, Oren *et al*. 1997). The regeneration of different types of injury inflicted on particular coral species is variable. For example, recovery of tissue injuries was slightly greater than that of tissue and skeleton injuries for *Porites astreoides* and *Agaricia agaricites* while *Montastrea annularis* showed the opposite pattern (Bak *et al*. 1977; Bak and Steward-Van Es 1980). The position of the injury within a colony may also influence rate of regeneration in some species (e.g. *Acropora palmata*, Meesters and Bak 1995) but not others (e.g. *Porites astreiodes*, Meesters *et al*. 1992).

### 1.1.3. LIFE-HISTORIES OF CORALS

The life-history traits peculiar to a particular coral species have evolved over time in response to a large number of biotic and abiotic processes including partial mortality. The ability of corals to resist damage has occurred through the evolution of defenses such as hard skeletons, a protective mucous coating, and nematocysts. However, due to the unpredictable nature of the numerous agents that can cause damage in corals, investment in a whole suite of defenses covering all possible scenarios would be too costly. Consequently, corals have also evolved the ability to regenerate polyps lost as a result of damage. Investment in defenses to protect against damage or the regeneration of lost parts once damage has occurred requires resources. If these resources are in finite supply, then a trade-off must occur in the allocation of resources between defence, regeneration and the other life-history traits (Stearns 1991). Trade-offs between regeneration and other life history traits have been demonstrated experimentally for several coral species (e.g. growth, Loya 1976; Bak 1983; Meesters *et al*. 1994; reproduction, Rinkevich and Loya 1989; Van Veghel and Bak 1994).

### 1.1.4. PREVAILING BIOTIC AND ABIOTIC CONDITIONS.

The amount of resources available for the regeneration of injuries within colonies of the same species is not uniform. Unfavourable biotic and abiotic conditions around the coral may cause added stress to the colony and influence the amount of resources available for regeneration (e.g. Kay and Liddle 1989; Meesters *et al*. 1992; Meesters and Bak 1993). For example, regeneration of injures was slower in bleached than unbleached colonies of *Montastrea annularis*, *Porites astreoides* and *Meandrina meandrites* (Meesters and Bak 1993) while regeneration was slower for colonies of *Acropora palmata* and *Montastrea annularis* in higher sediment regimes but not for colonies of *Diploria strigosa*, *Porites astreoides*, and *Siderastrea siderea* (Meesters *et al*. 1992).

## 1.2. GENERAL AIMS AND SIGNIFICANCE

Evidently, the underlying processes associated with the regeneration of injuries in scleractinian corals is complex and related to the nature and extent of injury, the characteristics of the injury, the life-histories of corals, and the prevailing biotic and abiotic conditions. Studies examining these processes have been conducted predominantly on common species of corals from the Caribbean and the Red Sea, with very few studies of this nature having been carried out on the speciose fauna of the Great Barrier Reef, Australia. Consequently, the major aim of this study is to broaden and extend our knowledge of the processes of injury and regeneration of scleractinian corals, with a particular emphasis on common reef-crest species from the Great Barrier Reef, Australia. Several aspects of injury have been examined in this thesis including (1) the nature and extent of natural injury, (2) the response of corals to injuries with different characteristics and (3) comparisons of morphological and life-history strategies of corals that enable them to respond to potential damage in different ways.

The spatial and temporal patterns of coral injury will be investigated in Chapter 2 to determine the nature and extent of natural injury on reef-crest corals at Lizard Island. Studies of the spatial patterns of coral injuries provide a snapshot of the dynamics of partial mortality, and allow inferences to be made about the relative vulnerabilities of corals to damage as a function of colony size, morphology and location. Temporal studies of coral injury, on the other hand, examine rates of injury and recovery, and provide an insight into the effects of differing injury regimes on the population dynamics of corals.

Morphological theory predicts that sessile, marine animals that are strongly committed to their place of settlement will invest more resources towards defence and maintenance than more fugitive species (Jackson 1979). In Chapter 3, I will examine differences in the responses of eleven species of corals with varying morphologies to injury to ascertain whether morphology influences recovery. Two characteristics of injury, type and position within the colony will also be examined to determine their effect on recovery since the cost of regeneration is likely to be dependent on the amount of damage inflicted, and the degree of colony integration.

Recovery of different types of injury appears to be linked with the amount of tissue and skeleton that must be replaced, as well as the ability of the coral to out compete organisms that have settled onto the injured area. In Chapter 4, I will examine if (1) the amount of colonisation of the injured site by algae, (2) the area of tissue from which regeneration can occur, and (3) regeneration rates, vary among colonies with tissue mortality, scraping or breakage injuries for two common reef-crest corals, *Acropora hyacinthus* and *Montipora tuberculosa*.

Life-history theory predicts that organisms with indeterminate growth will balance resource allocation between somatic longevity and reproductio (Kirkwood 1981). In Chapter 5, I investigate the effects of injury on colony growth, reproduction and survival for *Acropora hyacinthus*, *A. gemmifera* and *G. retiformis* to determine if there are any trade-offs between regeneration and the other life-history traits. Two factors, injury size and frequency of damage will be varied to test the hypothesis that the impact of an injury will depend on the size and frequency of damage: the larger and more frequent the injury, the greater the impact.

In conclusion, by examining the following three aspects of injury and regeneration: (1) the nature and extent of natural injury (2) the response of corals to injuries with different characteristics and (3) the influence of morphology and life-history on response to damage, for common reef-crest corals of the Indo-Pacific, a better understanding of the underlying processes associated with the regeneration of injuries can be gained. This is important because injury can adversely affect corals at the individual colony, population and community level and thus impact on the general ecology of coral reefs.

# CHAPTER 2: THE NATURE AND EXTENT OF NATURAL INJURY ON REEF-CREST CORALS AT LIZARD ISLAND

## 2.1. ABSTRACT

The spatial and temporal patterns of injury on reef-crest corals around Lizard Island were recorded to determine the nature and extent of damage in common reef-crest corals at Lizard Island. The total amount of partial mortality (proportion of coral cover that is dead) on reef-crest corals was low (< 2%) although there was a three-fold difference among sites. Reef-crest assemblages with low levels of partial mortality were dominated (both numerically and in cover) by tabular and bushy corals. These corals had low amounts of partial mortality and on average fewer small colonies with injuries. In contrast, the reef-crest assemblage were the partial mortality was three times higher had a lower abundance and percent cover of tabular corals and an increase in the number and cover of massive and digitate corals. Massive and digitate corals, on average, had a higher amount of partial mortality and more small colonies with injuries.

The pattern of injury over time (net effect of initial injury status, fate of injuries over 12 months and the addition of new injuries) for *Goniastrea retiformis*, *Acropora gemmifera* and *A. hyacinthus* was dependent on species, colony size, initial injury size and census year. *Goniastrea retiformis* had a high number of pre-existing injuries, slow regeneration rates, and few new injuries, suggesting that injuries tended to accumulate on colonies over time. The addition of new injuries was also low for *Acropora gemmifera*, however colonies had few pre-existing injuries and faster recovery rates, reducing the accumulation of injuries on colonies. The injury regime differed among censuses for *A. hyacinthus* because of a change in injury regimes from routine to catastrophic, the latter regime caused by an outbreak of *Acanthaster planci*. Under routine conditions there were few pre-existing injuries on colonies, a moderate addition of new injuries, and a rapid regeneration of injuries suggesting a fast turn-over of injuries. Under catastrophic conditions, the number of pre-existing injuries was higher, the number of new injuries was high, and more injuries were increasing in size than recovering resulting in an accumulation of injuries.

The number of injuries initially present on colonies, and the addition of new injuries was influenced by colony size. This effect was most pronounced for *A. hyacinthus* since there was at least an order of magnitude difference in the size of small and large colonies. For all species, small colonies had fewer pre-existing injuries, and for *A. hyacinthus* colonies in both censuses, the frequency of injury was higher for large colonies. In contrast, the fate of a pre-existing injury was independent of colony size since most injuries did not change their status over 12 months in either census year, and only a relatively small proportion (< 10%) regenerated, shrank, or grew in size.

Thus, species, colony size, injury size, the composition of coral assemblages and time are all important in determining the spatial and temporal patterns of injury for reef-crest corals. Understanding routine and catastrophic mortality of corals is important because of their role in structuring coral reef populations and communities.

## 2.2 INTRODUCTION

Corals are modular organisms that grow by the iteration of polyps and shrink through the loss of polyps (e.g. Hughes and Jackson 1985; Babcock 1991). Loss of polyps from an individual colony through damage is known as partial mortality. Partial mortality in corals is caused by abiotic and biotic disturbances such as predation, competition, disease, physical disturbances and anthropogenic activities including diving, fishing, boating, mining and coastal development (e.g. reviews by Connell 1973; Brown and Howard 1985; Craik *et al*. 1990; Grigg and Dollar 1990; Brown 1997). Mortality in scleractinian corals has often been quantified in terms of loss of coral cover (review by Connell 1996). However, few studies have focused on partial mortality in individual colonies (but see Hughes and Jackson 1980, 1985; Done 1987; Babcock 1991; Bythell *et al*. 1993; Meesters *et al*. 1996, 1997a; Ruesink 1997) despite the fact that losses of living tissue through partial mortality often can have far greater demographic consequences (in terms of reproduction and survival) for coral populations than whole colony mortality. Studies on the spatial and temporal patterns of natural injury are fundamental to an understanding of the processes that structure coral reef communities (e.g. disturbance, competition and predation).

Recent studies of partial mortality have shown that injury patterns differ among colonies of different sizes and shapes, between habitats or depth zones, and among species with similar morphologies (Bythell *et al*. 1993; Meesters *et al*. 1996, 1997a). The presence of an injury on a colony at a particular time is a function of how vulnerable a coral is to damage (where vulnerability is a function of probability of being injured and resilience once damage has occurred), and how fast it recovers from an injury. Although it is very difficult to monitor all the agents that cause damage in corals, monitoring tissue loss over time provides an insight into the various injury profiles of coral species and the subsequent demographic outcomes (Hughes and Jackson 1985; Babcock 1991; Bythell *et al*. 1993; Ruesink 1997). For example, the incidence of partial mortality is higher in large colonies, while whole colony mortality is higher in small individuals (Hughes and Jackson 1985; Babcock 1991; Bythell *et al*. 1993). Additionally, there are different injury profiles among and within habitats and depths because the agents of coral damage (e.g. predators, hurricanes, environmental stresses) are often patchy in nature and operate to different extents in different habitats and depths (Cumming 1996; Bythell *et al*. 1993; Meesters *et al*. 1997a). Vulnerability to damage is often associated with morphology since the shape of a coral significantly influences the type of interactions that it will have with the surrounding environment. Corals closely associated with the bottom (e.g. encrusting species) are greatly affected by benthic interactions while more upright species (e.g. tabular and branching species) are likely to be subjected to biological and physical interactions within the water column (Jackson 1979). Given an equal chance of injury, the amount of damage recorded on different species at a particular time is also variable (Bythell *et al*. 1993; Meesters *et al*. 1996) due to differences in physical defenses (e.g. skeletal density of branches and polyps, nematocyst densities, spiky protrusions) and regeneration capabilities (Loya 1976; Bak 1979; Bak and Steward-Van Es 1980; Bak 1983; Bythell *et al*. 1993; Meesters *et al*. 1996, 1997a).

The general aim of this chapter is to quantify the nature and extent of natural injury in corals to determine their importance in structuring coral communities. Specifically, I will examine (1) the spatial pattern of injury in terms of partial mortality for reef-crest coral communities at four sites, (2) the distribution of uninjured and injured colonies and high and low levels of partial mortality on corals as a function of colony morphology and colony size, (3) the influence of coral composition and size-structure on spatial patterns of injury, and (4) the temporal patterns of injury over two years.

## 2.3. MATERIALS AND METHODS

The spatial and temporal patterns of coral injury were recorded to determine the nature and extent of damage in common reef-crest corals at Lizard Island. An injury was defined as any area on a colony that was not healthy living tissue and included all colonisers (e.g. barnacles, tube worms, and algae) that had settled on damaged sections in the past.

### 2.3.1. Description of study sites

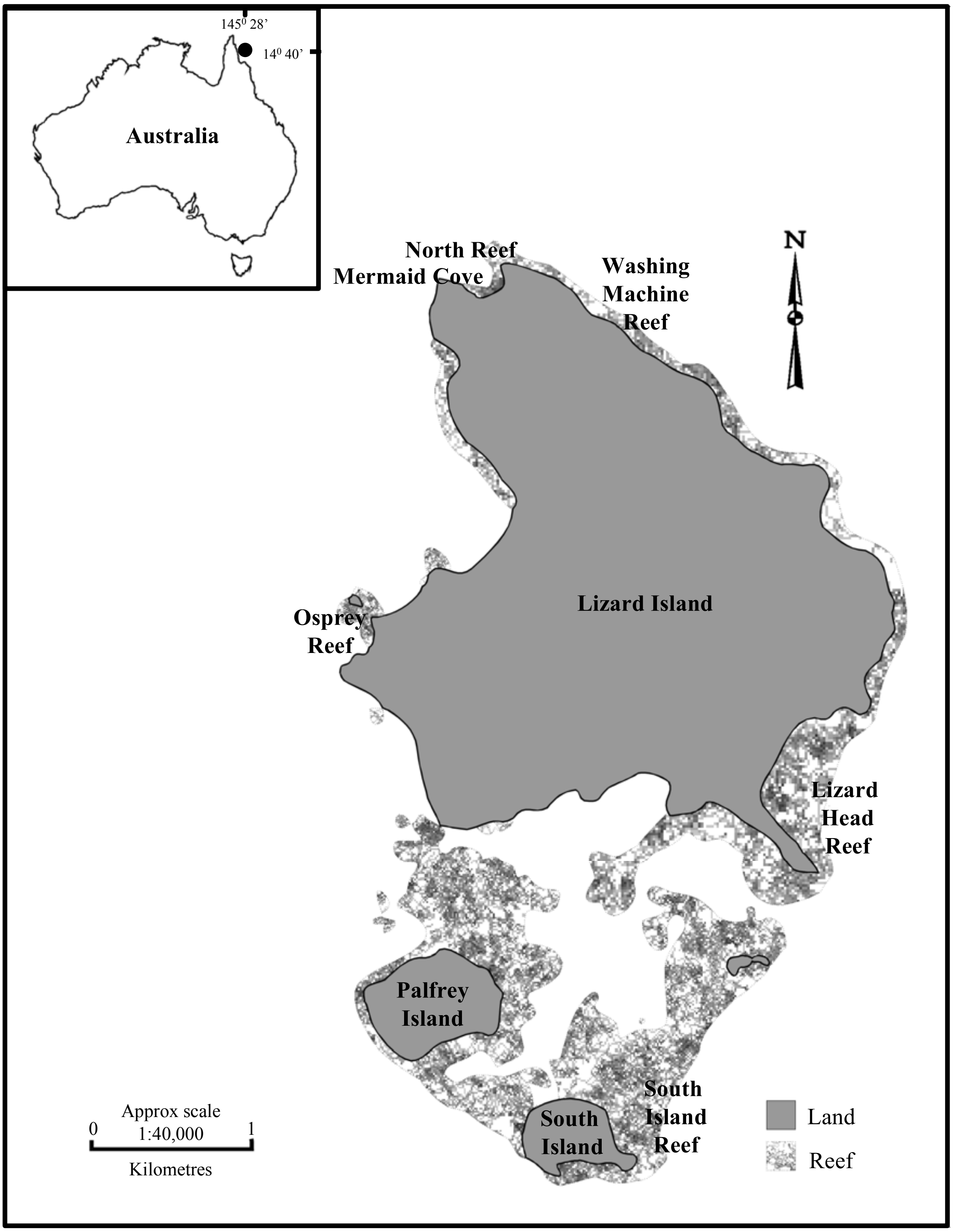
This study was conducted on the fringing reefs around Lizard Island ($14^0$40’, $145^0$28’), a continental island of the Great Barrier Reef, approximately 25 km off the eastern coast of mainland Australia. The Lizard Island complex consists of Lizard Island and three smaller islands, Palfrey, South, and Bird Islets, all of which are connected by reefs (Pichon and Morrissey 1981). These islands are protected from oceanic swells by the outer barrier reef. The study sites were South Reef, Lizard Head, Washing Machine, North Reef, and Mermaid Cove (Figure 2.1). South Reef and Washing Machine are exposed sites subjected to the dominant southeast trade winds. In contrast, Mermaid Cove, North Reef and Washing Machine are relatively protected sites, sheltered from the southeast trade winds by Lizard Island. The section of reef that was examined at all sites was the reef crest. The reef crest is defined as the narrow strip of reef between the reef flat and reef slope (Pichon and Morrissey 1981).

### 2.3.2. Spatial patterns of injury

To determine the extent of natural injury to reef-crest corals at Lizard Island I recorded the amount of injury present at four sites in February 1994: South Island, Lizard Head, Washing Machine and North Reef (Figure 2.1). The number of injuries per colony, injury size, and colony size were measured, and individual colonies were categorised into five morphological groups for scleractinian corals along ten, 5 metre line transects at each site. The position of the initial line transect at each site was haphazardly chosen and each transect thereafter was separated by 5 meters. The five morphological groups were bushy corals (Pocilloporid and Acroporid colonies), massive corals (Poritids, Faviids and Mussids), tabular Acroporids, digitate Acroporids, and others (consisting mainly of Isoporans and Montiporids).

The size of injuries and coral colonies were measured to the nearest 0.5 cm using a tape measure. To obtain an estimate of the projected area of the injuries and colonies, the maximum diameter and diameter perpendicular to the maximum diameter were recorded for colonies, and the length and breadth measured for injuries.

Figure 2.1. A map of Lizard Island showing the location of study sites.



Injuries less than 1cm$^2$ were counted but not accurately measured. The projected area of an injury was estimated by multiplying the length and breadth together since the shape of most injuries resembled that of a rectangle. In contrast, most colonies are elliptical in shape so the formula for calculating the area of an ellipse was used to estimate colony size.

### 2.3.2.1. Patterns of tissue loss among sites

The amount of injury at a particular site is a function of the amount and frequency of damage occurring as well as how fast an injury is regenerated. A measure of rates of injury and recovery is provided by partial mortality which is the proportion of total coral cover that is dead. To investigate whether the pattern of injury and recovery differed among sites, the amount of partial mortality per transect was quantified for each of the four reef-crest sites around Lizard Island. Differences between sites were tested with an analysis of variance (ANOVA). The data were arcsin square-root transformed to meet the assumptions of the ANOVA. An *a posteriori* Tukey's test was used to distinguish significant differences between sites.

### 2.3.2.2. The effect of morphology and colony size on injury patterns

Damage in scleractinian corals is size-specific and some coral morphologies are more vulnerable to damage than others (Bythell *et al.* 1993; Meesters *et al.* 1996, 1997a). To determine the effect of colony size and growth form on injury patterns, the distribution of colonies into particular injury categories were examined for small ($\leq$ 100 cm$^2$) and large (> 100 cm$^2$) colonies belonging to the four most common morphological groups at each site. The injury categories included (1) injured and uninjured colonies, and (2) injured colonies with $\leq$ 5% and >5% tissue loss. Frequency analyses were used to test the effect of site, colony size and morphology on injury patterns. A logistic regression analysis using backward eliminations was used to derive a minimal model, and an F-test was used to detect differences between effects because the data were over-dispersed (Collett 1991; De'ath and Moran 1998).

### 2.3.2.3. Coral community composition and size-structure within sites

The size-structure and composition of a given coral community influences community level injury patterns since vulnerability to damage is size-specific and related to morphology. The coral communities at the four sites are described in terms of numerical abundance and percent cover of the five morphological groups. Percent cover is estimated by summing the intercept of each group along each transect and dividing this number by the total length of the transect. An ANOVA was used to test for differences between sites in mean percent cover and number of colonies per transect. The data was log- transformed to meet the assumptions of ANOVA. The size-structure of the morphological groups was examined in relative terms, that is, the proportion of small and large colonies among morphological groups and sites. Differences in the distribution of colonies among these groups were tested with a logistic regression analysis (as described previously).

### 2.3.3. Temporal patterns of injury

To determine natural rates of injury and regeneration for large and small reef-crest corals I recorded the number of new injuries, and followed the fate of old injuries on individual colonies belonging to three species and two size-classes every 3-4 months for 2 years at Mermaid Cove, Lizard Island (Figure 2.1). The survey commenced in February 1994 and ended with a final census in February 1996. A total of 8 censuses were conducted over this period.

The three species investigated were *Acropora hyacinthus*, *A. gemmifera* and *Goniastrea retiformis*, chosen because they represented three of the main morphological groups investigated for the spatial study. These species were also considered because of their differing life-histories and their abundance on the reef-crest. *Acropora hyacinthus* is a fast-growing, relatively short-lived tabular coral, *A. gemmifera* is a digitate coral with a moderate growth rate and life-span, and *G. retiformis* is a massive species with a long life-span and slow growth rate. Colonies for each species were distinguished by size: the average diameter of small colonies was < 10 cm and the average diameter of large colonies was > 20cm. Plastic horticultural tags nailed to the substratum close to each colony were used to mark individual corals and facilitate their relocation during subsequent censuses. A total of 90 colonies were tagged, 15 colonies per species and size-class. Due to tag losses and a few colony deaths after one year of monitoring (February 1994 - 1995), a new set of colonies were tagged and monitored in the second year (February 1995 - 1996). The final number of colonies per species and size-class ranged from 8 - 11.

The addition of new injuries, and the fate of old injuries were observed by comparing photographic images of the tagged colonies from consecutive censuses. The colonies were photographed with a Nikonos V underwater camera. Colonies of *Acropora hyacinthus* and *A. gemmifera* were photographed from above at a height of approximately 3 meters. A tile of known area was placed in each photograph and used as a size calibration. The horizontal plane of large colonies of *A. hyacinthus* and *A. gemmifera* is an appropriate way to monitor the incident of injury because their growth form is relatively two-dimensional. In contrast, *G. retiformis* has a more three-dimensional shape and thus required a rather different sampling protocol. *Goniastrea retiformis* colonies were divided up into quarters by placing two small chains at right angles over the colony. Each quarter and the horizontal plane were photographed, usually from a distance of 1m. The size standard for each of these photographs was a reference tile of known area. Small colonies were photographed with a 35 mm close-up lens, with a ruler attached to the close-up frame providing a scale.

#### 2.3.3.1. The fate of injuries

Using the photographs taken at the beginning of February 1994 and February 1995, I individually labelled and then classified into two size-classes (small, $\leq$ 1cm$^2$ and large, >1 cm$^2$) each injury present on tagged colonies. This classification will be referred to as initial injury size from herein while injuries present on colonies at the beginning of each census will be referred to as pre-existing injuries. I subsequently traced the fate of these pre-existing injuries by following their transitions between size-classes from photographs taken of the same colonies one year later. Injuries that stayed the same size remained in their initial size-class, injuries that decreased in size either shrank to the smaller size-class or recovered completely (i.e. regenerated), and injuries that increased in size either migrated to the larger size-class or stayed in the larger size-class.

Differences in the distribution of injuries among injury size, years, species and colony size were determined using a log-linear analysis. Colonies were nested within yearly censuses to maintain temporal independence, an assumption of the analysis. F-tests were used to compute probabilities because the data were over dispersed (Collett 1991; De'ath and Moran 1998). A tree-based model was used to explore the relationship between the fate of injuries and census intervals, species, initial injury size and colony size (Clark and Pregibon 1997). These models split the data set into increasingly homogeneous subsets resulting in the production of a classification tree.

#### 2.3.3.2. The addition of new injuries

For each yearly period, the number of new injuries present on the photographed colonies each 3 - 4 months was recorded for small and large colonies belonging to the three species. New injuries were detected on colonies by comparing photographs from consecutive censuses. The census intervals for the first yearly period (1994 - 1995) were February to May, May to August, August to November and November to February, and the census intervals for the second yearly period (1995 -1996) were February to June, June to October and October to February. Changes in the number of new injuries present on colonies among species and colony sizes over time were tested using a repeated measures ANOVA. The two yearly periods were analysed separately. The ANOVA model consisted of species and colony size as fixed factors and colonies as a random factor nested within the fixed factors. The data were log-transformed to conform to the assumptions of the analysis.

The frequency of injuries on colonies over the census periods (i.e. number of census intervals that a colony sustained an injury) within each year was also estimated. Colonies were divided up into the following categories: no injuries, injured at a single census, injured at two censuses, injured at three censuses, and injured at four censuses.

## 2.4. RESULTS

### 2.4.1. General summary of results

The spatial and temporal patterns of injury on reef-crest corals around Lizard Island were influenced by colony size, location, species, initial injury size and time. For the spatial study, the incidence of partial mortality was low, and differed among sites due to variations in the composition and size-structure of the reef-crest assemblages, and the contrasting injury profiles of small and large colonies with differing morphologies. The pattern of injury over time (net effect of initial injury status, fate of injuries over 12 months and the addition of new injuries) for *G. retiformis*, *A. gemmifera* and *A. hyacinthus* was dependent on species, colony size, initial injury size and census year. The results of the spatial and temporal studies of injury have been discussed in detail below.

### 2.4.2. Spatial patterns of injury

The injury profile of 673 colonies was surveyed at 4 sites around Lizard Island and a total of 4374 injuries were recorded. Of the total number of colonies recorded, 287 were small ($\leq$ 100cm$^2$) and 386 colonies were large (> 100cm$^2$). The number of uninjured colonies surveyed was low, making up 27% of the total (n = 184). The majority of these uninjured colonies (72%) were small in size (n = 132 and 52 for uninjured small and large colonies respectively). For injured colonies, the majority of injuries present were $\leq$ 1cm$^2$ in size with only 18% of the total number of injuries being > 1cm$^2$. This pattern was consistent over all sites (Table 2.1).

#### 2.4.2.1. Patterns of tissue loss between sites

Partial mortality was relatively low at all sites (Figure 2.2). Nevertheless, striking differences were detected in the amount of partial mortality present between sites. The average amount of partial mortality per transect was approximately three times higher at Washing Machine than the other three sites (ANOVA, $F_{3,36} = 18.67$, p = 0.0001, Figure 2.2). Conversely, no significant differences were detected in the average amount of partial mortality per transect for South Island, North Reef and Lizard Head (Tukey's Test, Figure 2.2).

Figure 2.2. Mean percent partial mortality per transect for the four study sites around Lizard Island. (Error bars = one SE).

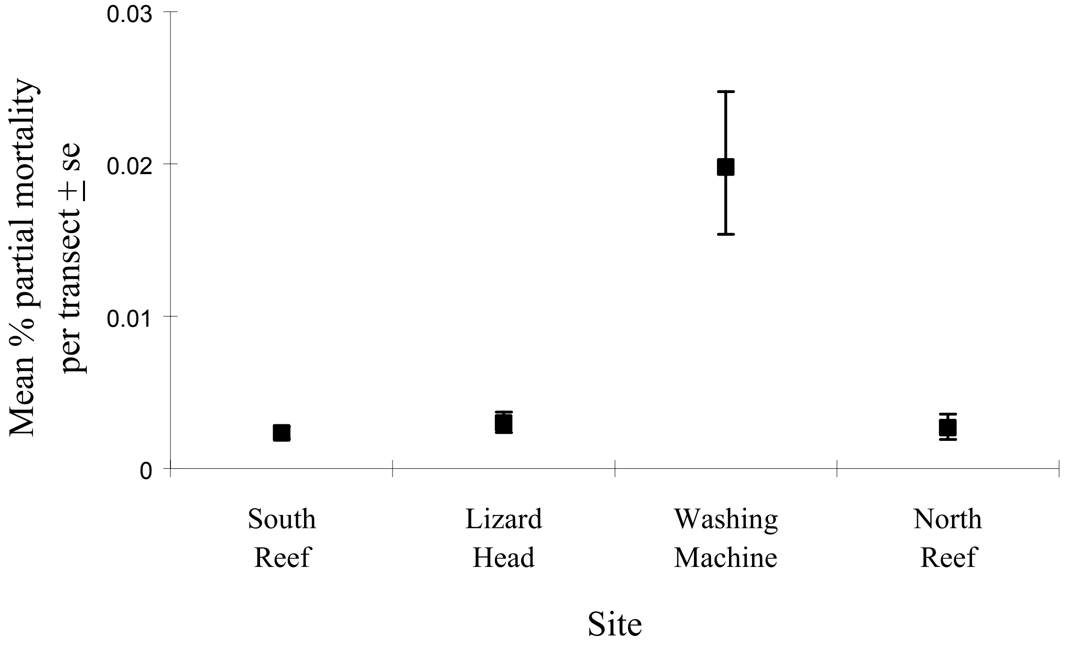


Table 2.1. The number of small (≤ 1 $cm^2$) and large (> 1 $cm^2$) injuries present at South Island (SI), Lizard Head (LH), Washing Machine (WM) and North Reef (NR), Lizard Island.

| Site | Number of Small Injuries | Number of Large Injuries | Percentage of Small Injuries | Percentage of Large Injuries |
|---|---|---|---|---|
| SI | 1146 | 160 | 88% | 12% |
| LH | 745 | 128 | 85% | 15% |
| WM | 645 | 186 | 79% | 21% |
| NR | 1050 | 305 | 77% | 23% |
| TOTAL | 3595 | 779 | 82% | 18% |

#### 2.4.2.2. The effect of morphology and colony size on injury patterns at the four sites

The number of injured and uninjured colonies and colonies with low and high levels of partial mortality was consistent among sites for the different colony sizes and morphological groups (Table 2.2). For the four morphological groups there were generally fewer uninjured colonies than injured colonies and most injured colonies had low amounts of partial mortality. Approximately 50% of small colonies and less than 25% of large colonies were uninjured and there was an even mixture of injured colonies of both size classes with ≤ 5% (low) and > 5% (high) partial mortality.

Conversely, striking differences were detected in the distribution of uninjured and injured colonies amongst small and large colonies belonging to the four morphological groups, and amongst morphological groups for injured colonies with high and low levels of partial mortality (Table 2.3 and 2.4). For small colonies, there was an even mixture of uninjured and injured colonies for bushy and massive corals, slightly more uninjured colonies for tabular corals, and approximately twice as many injured colonies for digitate corals. The percentage of uninjured large colonies was low (<20%) across all morphological groups. The distribution of injured colonies with high and low levels of partial mortality also varied amongst groups. The majority of bushy and tabular corals had low amounts of partial mortality while massive and digitate corals had a mixture of colonies with high and low levels of partial mortality. The percentage of colonies with high and low levels of partial mortality was consistent among colony sizes. For example, 46% and 63% of small and large colonies respectively had low amounts of partial mortality.

Table 2.2. The distribution of colonies within injury categories (uninjured versus injured and ≤ 5% and > 5% partial mortality (PM)) amongst sites for morphological group and colony size expressed as a percentage of the total (n).

| Effect | Site | % Uninjured Colonies | % Colonies ≤ 5% PM |
|---|---|---|---|
| | | ***Morphological Group*** | |
| Bushy corals | North Reef | 33 (40) | 63 (27) |
| | South Island | 28 (53) | 63 (38) |
| | Lizard Head | 29 (68) | 82 (28) |
| | Washing Machine | 31 (59) | 44 (41) |
| Massive corals | North Reef | 54 (24) | 27 (11) |
| | South Island | 24 (29) | 32 (22) |
| | Lizard Head | 33 (33) | 50 (22) |
| | Washing Machine | 35 (48) | 45 (31) |
| Tabular corals | North Reef | 12 (51) | 73 (45) |
| | South Island | 16 (45) | 87 (38) |
| | Lizard Head | 22 (37) | 79 (29) |
| | Washing Machine | 28 (18) | 62 (13) |
| Digitate | North Reef | 14 (21) | 50 (18) |
| | South Island | 44 (18) | 70 (10) |
| | Lizard Head | 19 (31) | 68 (25) |
| | Washing Machine | 22 (46) | 64 (36) |
| | | ***Colony Size*** | |
| Small | North Reef | 56 (52) | 43 (23) |
| | South Island | 43 (56) | 44 (32) |
| | Lizard Head | 39 (95) | 53 (58) |
| | Washing Machine | 50 (84) | 52 (42) |
| Large | North Reef | 10 (98) | 64 (88) |
| | South Island | 15 (106) | 67 (90) |
| | Lizard Head | 18 (77) | 73 (63) |
| | Washing Machine | 11 (105) | 51 (93) |

Table 2.3. Analysis of deviance for logistic regression models assessing the effects of site, morphology and colony size on (1) uninjured and injured colonies and (2) injured colonies with ≤ 5% and > 5% partial mortality.

| Effect | df | Change in Deviance | F-ratio | P |
|---|---|---|---|---|
| | | *Uninjured and Injured Colonies* | | |
| Morphological Group | 4 | 59.58 | 229.17 | 0.000 |
| Group * Colony Size | 3 | 65.48 | 335.8 | 0.000 |
| Residual | 25 | 1.63 | | |
| | | *Partial Mortality* | | |
| Morphological Group | 3 | 28.42 | 8.20 | 0.000 |
| Residual | 28 | 32.35 | | |

#### 2.4.2.3. Coral community composition and size-structure among sites

The composition and size-structure of the coral community at Lizard Island differed significantly among the four sites (Table 2.5 and 2.6, Figure 2.3, 2.4 and 2.5). The number of colonies per transect for each morphological group was relatively low, and similar at all four sites with the exception of tabular corals. For this group, there were approximately twice the number of colonies at North Reef than with Washing Machine. The number of tabular corals did not vary between South Island, Lizard Head or North Reef, nor between South Island, Lizard Head or Washing Machine (Tukey's Test).

Table 2.4. (a) The percentage of uninjured colonies among morphological groups and colony sizes and (b) the percentage of colonies with $\leq$ 5% partial mortality (PM) for the four morphological groups.

| *Morphological Group* | *Colony Size* | *Percentage of uninjured colonies (total)* |
|---|---|---|
| Bushy corals | Small ($\leq$100cm$^2$) | 49 (95) |
| | Large (>100cm$^2$) | 18 (106) |
| Massive corals | Small ($\leq$100cm$^2$) | 48 (92) |
| | Large (>100cm$^2$) | 12 (43) |
| Tabular corals | Small ($\leq$100cm$^2$) | 63 (19) |
| | Large (>100cm$^2$) | 11 (132) |
| Digitate corals | Small ($\leq$100cm$^2$) | 36 (36) |
| | Large (>100cm$^2$) | 18 (80) |

| *Morphological Group* | *Percentage colonies $\leq$ 5% PM (total)* |
|---|---|
| Bushy corals | 62 (134) |
| Massive corals | 41 (86) |
| Tabular corals | 78 (125) |
| Digitate corals | 37 (89) |

The percent coral cover per transect was also low (< 20%) and showed a similar pattern among sites to that observed for coral abundance (Figure 2.3). The average percent cover was similar for all morphological groups among sites with the exception of tabular corals which differed significantly between North Reef and Washing Machine by a factor of two (Figure 2.4). The distribution of small ($\leq$100cm$^2$) and large (>100cm$^2$) colonies was consistent among sites, and within groups among sites, but differed between groups (Table 2.6, Figure 2.5). Bushy corals were made up of an even mixture of small and large colonies, massive corals and other corals were predominantly small in size, and tabular and digitate corals had a greater number of large colonies (Figure 2.5). Overall, tabular corals were at least an order of magnitude larger in size than the other three main morphological groups (average size ± SE for tabular, bushy, digitate and massive corals were respectively: 4579 ± 73 cm$^2$, 425 ± 24 cm$^2$, 306 ± 7 cm$^2$, and 181 ± 7 cm$^2$).

Table 2.5. A summary of the ANOVA results testing for differences in (a) mean number of colonies per transect and (b) mean percent cover between sites and morphological groups.

| Source of Variation | df | Type III SS | Mean Square | F-ratio | P |
|---|---|---|---|---|---|
| ***Number of colonies per transect*** | | | | | |
| Site | 3 | 0.222 | 0.074 | 1.10 | 0.353 |
| Morphological Group | 4 | 2.722 | 0.068 | 10.06 | 0.000 |
| Site * Group | 12 | 1.809 | 0.151 | 2.23 | 0.012 |
| Error | 180 | 12.177 | 0.068 | | |
| ***Percent cover per transect*** | | | | | |
| Site | 3 | 0.012 | 0.004 | 0.61 | 0.612 |
| Morphological Group | 4 | 0.873 | 0.218 | 32.21 | 0.000 |
| Site*Group | 12 | 0.236 | 0.020 | 2.91 | 0.001 |
| Error | 180 | 1.220 | 0.007 | | |

Figure 2.3. The number of colonies per transect among morphological groups at each site around Lizard Island. (Number of transects = 10, error bars represent one SE).

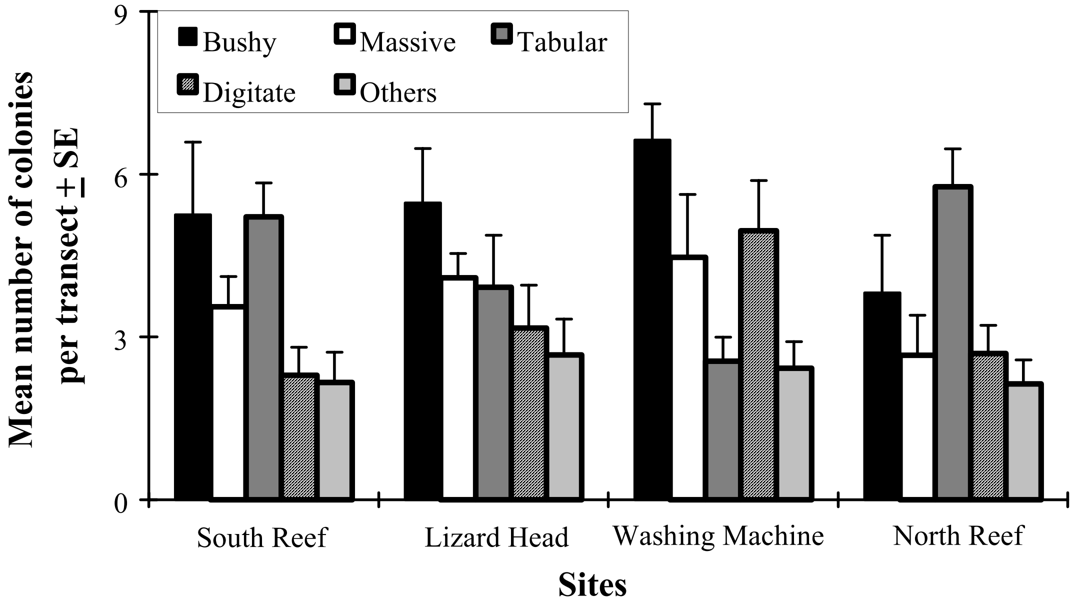


Figure 2.4. The coral cover per transect of morphological groups at each site around Lizard Island. (Number of transects = 10, error bars represent one SE).

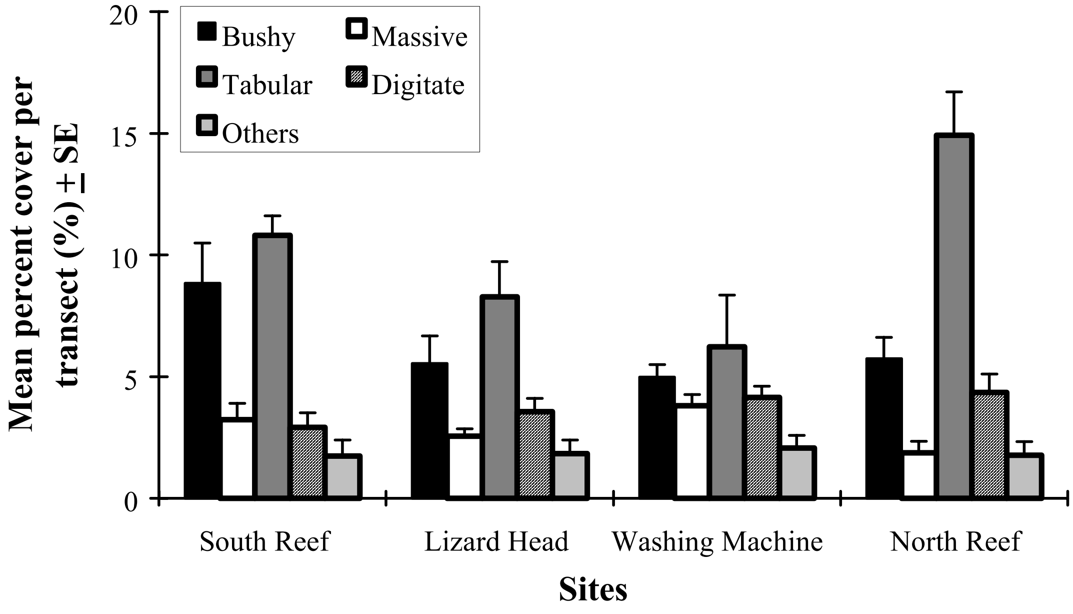


Figure 2.5. The distribution of small and large colonies among morphological groups and sites at Lizard Island. (SI = South Island; LH = Lizard Head; WM = Washing Machine; NR = North Reef).

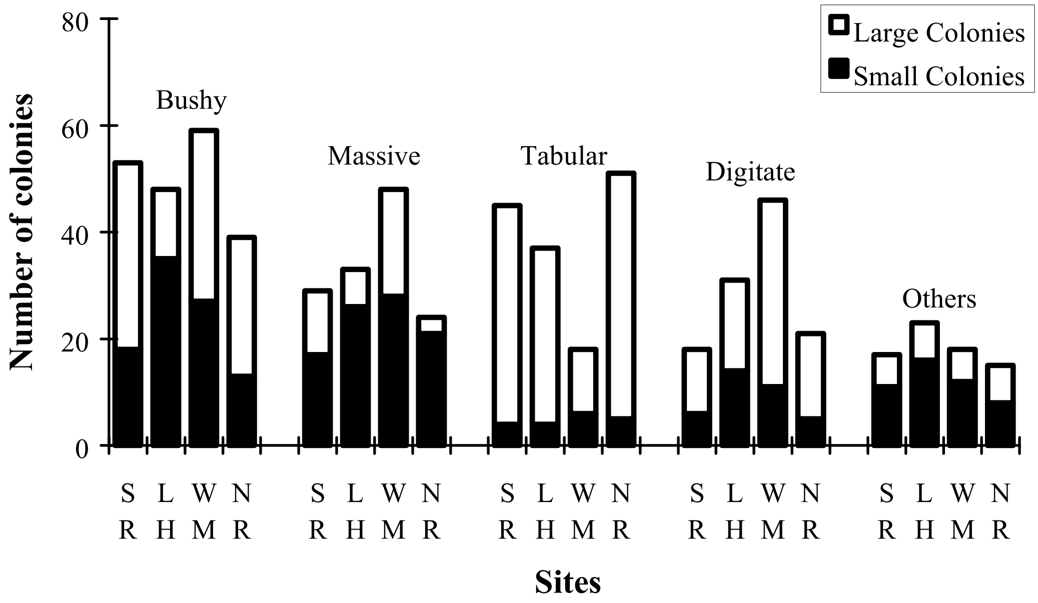


Table 2.6. Analysis of deviance for the logistic regression model assessing the effects of morphological group and sites on the distribution of small and large colonies.

| Effect | df | Change in deviance | F-ratio | P |
|---|---|---|---|---|
| Morphological Group | 4 | 117.39 | 10.434 | 0.000 |
| Residual error | 15 | 42.19 | | |

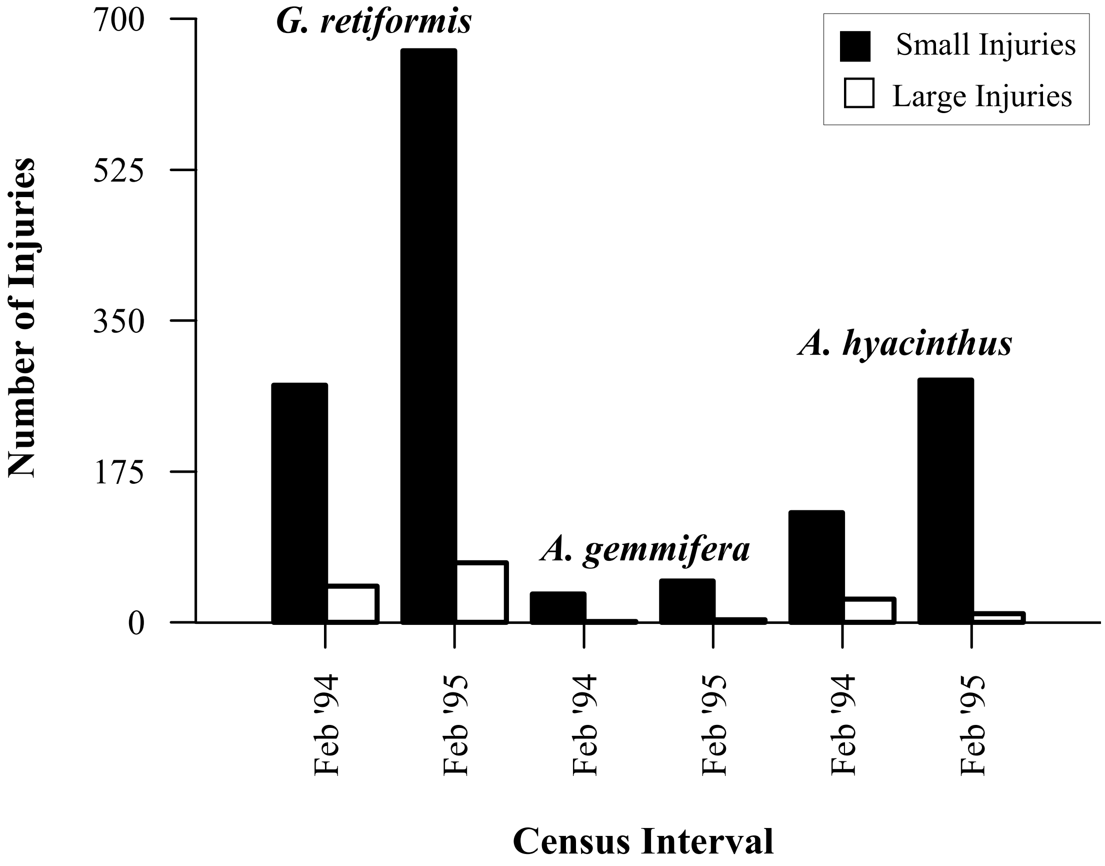
### 2.4.3. Temporal patterns of injury

#### 2.4.3.1. Initial Status of Injuries

A total of 1579 injuries were recorded initially on colonies of *G. retiformis, A. gemmifera* and *A. hyacinthus* over two censuses (February 1994 and February 1995).

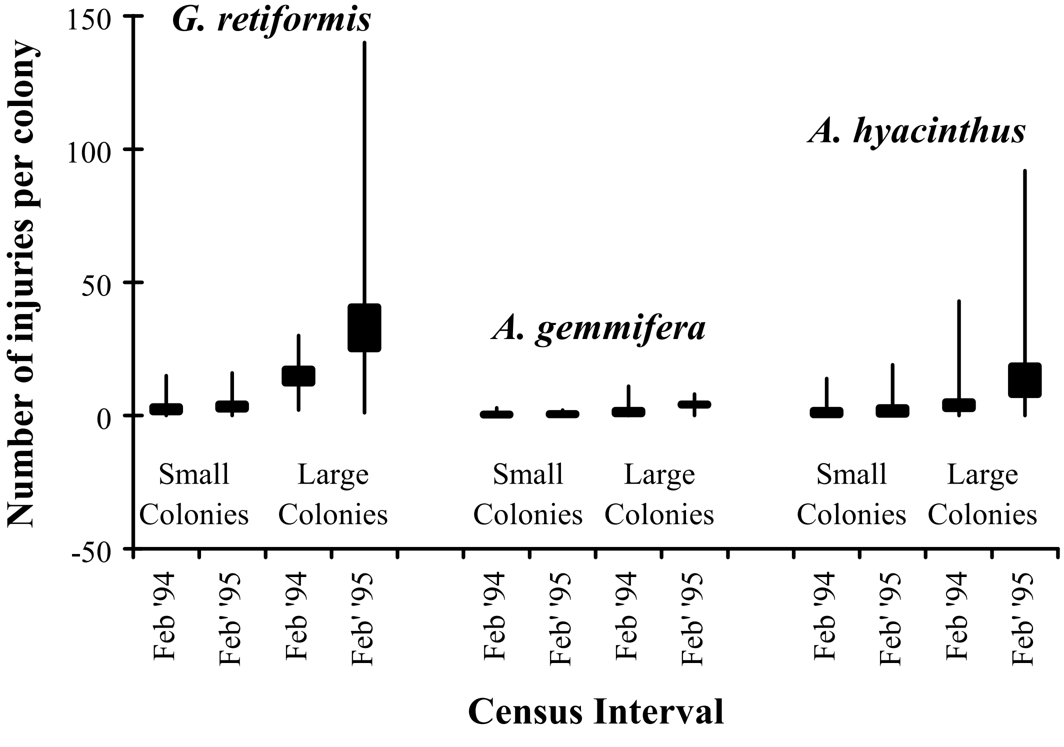
The initial distribution of these injuries was dependent on their size, species and census year (Log-linear Analysis, F-ratio = $7.747_{(2,7)}$, $P = 0.0168$). The proportion of small and large injuries present on colonies was fairly consistent for all species and both censuses, with over 80% of these injuries being $\leq 1\text{cm}^2$ in size (Figure 2.6). In contrast, the number of injuries varied among species and census years. *Goniastrea retiformis* colonies had the highest number of injuries (total number = 1031), twice that of *A. hyacinthus* colonies and 12-times that of *A. gemmifera* colonies. The number of injuries present on *G. retiformis* colonies in February 1994 was approximately half that observed in February 1995.

Figure 2.6. The number of small and large injuries on colonies of *G. retiformis*, *A. gemmifera* and *A. hyacinthus* in February 1994 and February 1995.



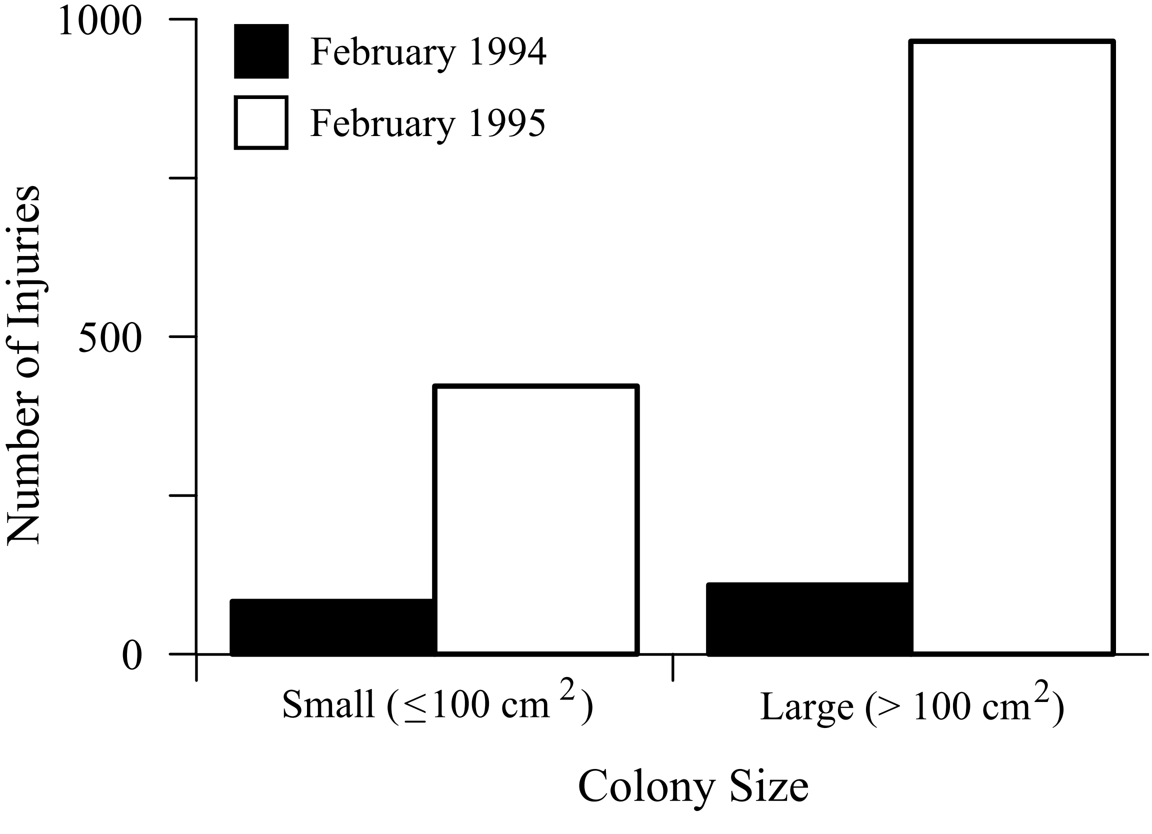
Similarly, the number of injuries present on *A. hyacinthus* colonies was greater in February 1995 than February 1994, by a factor of two for small injuries and a factor of three for large injuries. This increase in the number of injuries in February 1995 for both *G. retiformis* and *A. hyacinthus* was caused by the random selection of colonies that had, on average, more injuries per colony , and the inclusion of one large *G. retiformis* colony with an exceptionally high number of injuries (Figure 2.7). In contrast, the number of small and large injuries on *A. gemmifera* were similar for both years.

Figure 2.7. The range ( | ) and average number of injuries per colony ± SE ( ▮ ) present on large and small colonies of *G. retiformis*, *A. gemmifera* and *A. hyacinthus* in February 1994 and February 1995. (Sample sizes for small and large colonies of *G. retiformis*, *A. gemmifera* and *A. hyacinthus* are 11,11,9,10,10,9,10,9,10,8,10, and 10 respectivlely).



The initial distribution of injuries was also dependent on colony size and census year (Log-linear analysis, F-ratio = $5.534_{(1,10)}$, P = 0.017). The majority of injuries were found on large colonies, with less than 20% of injuries being present on small colonies (Figure 2.8). Furthermore, there were approximately twice as many injuries present on large colonies in February 1995 than February 1994 (Figure 2.8), mainly due to a general increase in the number of injuries on colonies for *A. hyacinthus* and *G. retiformis* (Figure 2.7).

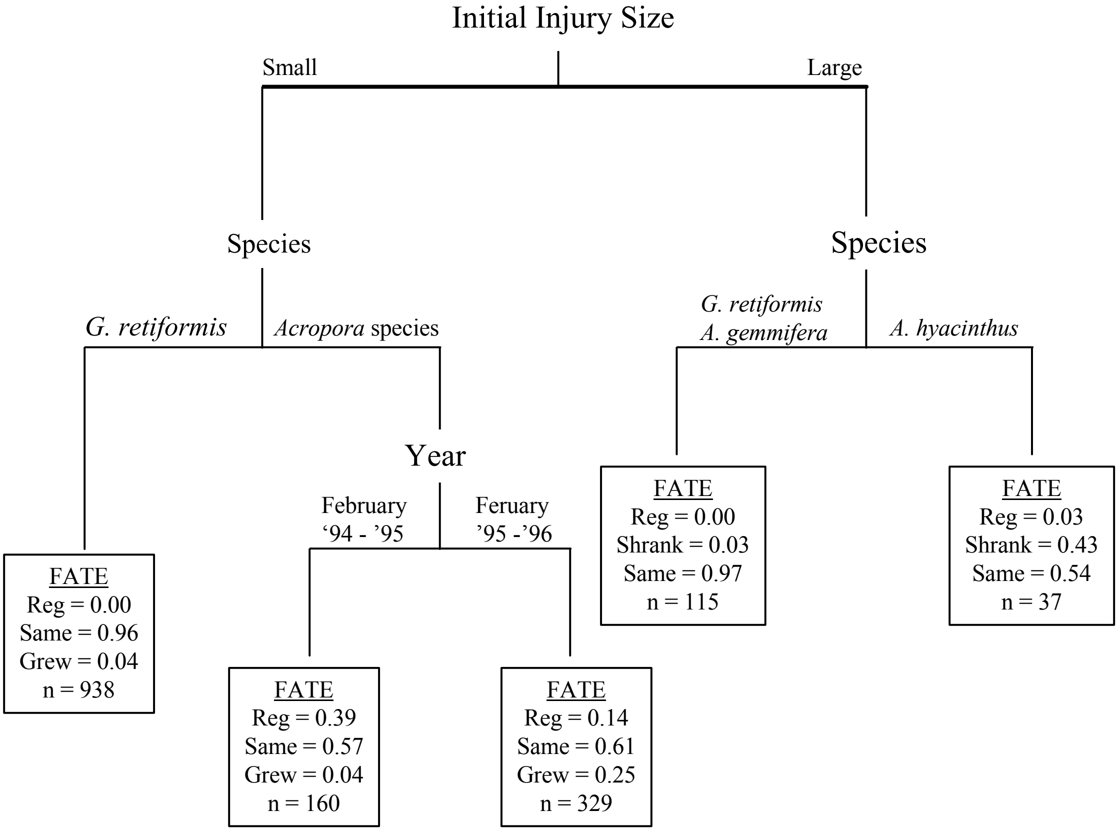
Figure 2.8. The distribution of injuries between small and large colonies in February 1994 and February 1995.



### 2.4.3.2. Fate of Injuries

There were striking differences in the fate of injuries observed on colonies of *G. retiformis*, *A. gemmifera* and *A. hyacinthus* over 12 months from Feb'94 - Feb'95 (census 1) and Feb'95 - Feb'96 (census 2). Whether an injury recovered, shrank, increased in size, or remained in the same size-class was highly dependent on the initial size of the injury (Table 2.7; Classification Tree, primary split, Figure 2.9). The fate of small injuries was further dependent on species and census year (Classification Tree, secondary and tertiary splits, Figure 2.9). For colonies of *G. retiformis*, the status of small injuries did not change over 12 months for either census period (i.e. 96% of injuries stayed the same size, Table 2.7). In contrast, the status of small injuries was more dynamic for the two *Acropora* species, with a small proportion of injuries getting larger or recovering, and the majority of injuries staying the same size (Table 2.7).

Figure 2.9. A classification tree showing the relationship between fate and initial injury size, species and census. The proportion of injuries belonging to each fate have been provided beneath the terminal split, along with the number of injures (n) for that subset of data. (Reg = regeneration; Shrank = injuries that decreased in size from large to small; Same = injuries stayed the same size; Grew = injuries that increased in size from small to large).



There was also a change in the fate of small injuries between yearly intervals, particularly for *A. hyacinthus* (Table 2.7). In the first census, the proportion of small injuries regenerating was higher (48%) than the proportion of small injuries getting larger (1%) while the opposite pattern was true for the second census (14% of small injuries regenerating and 28% of small injuries getting larger). The fate of large injuries was dependent on species (Classification Tree, secondary split, Figure 2.9). The majority of large injuries (97%) belonging to colonies of *G. retiformis* and *A. gemmifera* did not change status over a 12 month period (Table 2.7, Figure 2.9). In contrast, the fate of large injuries for *A. hyacinthus* were more dynamic, with a small proportion of large injuries shrinking into the smaller size class or regenerating (Table 2.7, Figure 2.9). The fate of injuries was not influenced by colony size since the majority of injuries stayed the same size, irrespective of colony size (n = 156 and 1168 for small and large colonies respectively), and less than 10% of injuries shrank/regenerated or grew in size (shrank/regenerated: n = 22 and 112; grew: n = 14 and 107 for small and large colonies respectively).

#### 2.4.3.3. New Injuries

A total of 989 new injuries were recorded on 117 colonies over two 12 month periods, 456 new injuries from Feb’94 - Feb’95 and 533 new injuries from Feb’95 - Feb’96. The majority of these injuries (n = 890) were less than 1 $cm^2$ in size. The new injuries were observed predominantly on *A. hyacinthus* (n = 868) with only 80 and 41 new injuries being found on *G. retiformis* and *A. gemmifera* respectively. The average number of new injuries differed among species and colony size for each yearly census (between subject effects, Table 2.8, Figure 2.10). Additionally, the accumulation of injuries on *A. hyacinthus*, *A. gemmifera* and *G. retiformis* over 3 -4 monthly intervals was highly variable for the 1994 - 1995 census interval but consistent over time for the 1995 - 1996 census interval (within subject effects, Table 2.8, Figure 2.10). Generally, the average number of new injuries was consistenly low for *G. retiformis* and *A. gemmifera*, and small colonies of *A. hyacinthus*. This pattern did not change over time with the exception of the May’94 - Aug’94 interval for small *A. gemmifera* colonies where the average number of new injuries doubled (Figure 2.10). In contrast, the number of new injuries recorded on large *A. hyacinthus* colonies over time was highly variable (Figure 2.10). Average numbers of new injuries for large *A. hyacinthus* colonies were approximately 2 - 5 times higher in the second census, with the exception of August 1994, where the number of new injuries recorded was 5 times higher than any other census interval between Feb'94 - Feb'95.

Table 2.7. The number of injuries that regenerated, shrank, stayed the same, or grew over 12 months, cross-classified by initial injury size, species and census interval.

| SMALL INITIAL SIZE | | | | |
|---|---|---|---|---|
| | Census | Fate | | |
| Species | Interval | Regenerated | Same Status | Grew |
| *G. retiformis* | Feb'94 - Feb'95 | 4 | 261 | 10 |
| | Feb'95 - Feb'96 | 0 | 639 | 24 |
| *A. gemmifera* | Feb'94 - Feb'95 | 2 | 27 | 4 |
| | Feb'95 - Feb'96 | 7 | 38 | 3 |
| *A. hyacinthus* | Feb'94 - Feb'95 | 61 | 65 | 1 |
| | Feb'95 - Feb'96 | 39 | 163 | 79 |

| LARGE INITIAL SIZE | | | | |
|---|---|---|---|---|
| | Census | Fate | | |
| Species | Interval | Regenerated | Shrank | Same Status |
| *G. retiformis* | Feb'94 - Feb'95 | 0 | 1 | 40 |
| | Feb'95 - Feb'96 | 0 | 3 | 66 |
| *A. gemmifera* | Feb'94 - Feb'95 | 0 | 0 | 1 |
| | Feb'95 - Feb'96 | 0 | 0 | 4 |
| *A. hyacinthus* | Feb'94 - Feb'95 | 0 | 16 | 11 |
| | Feb'95 - Feb'96 | 1 | 0 | 9 |

Table 2.8. Repeated measures ANOVA testing the effect of species and colony size on the number of new injuries per colony over time for the two consecutive years. (a = Feb’94 -‘95 and b = Feb’95 - 96).

a. Feb’94 -May’94, May’94 - Aug’94, Aug’94 - Nov’94 and Nov’94 - Feb’95.

Between Subject Effects

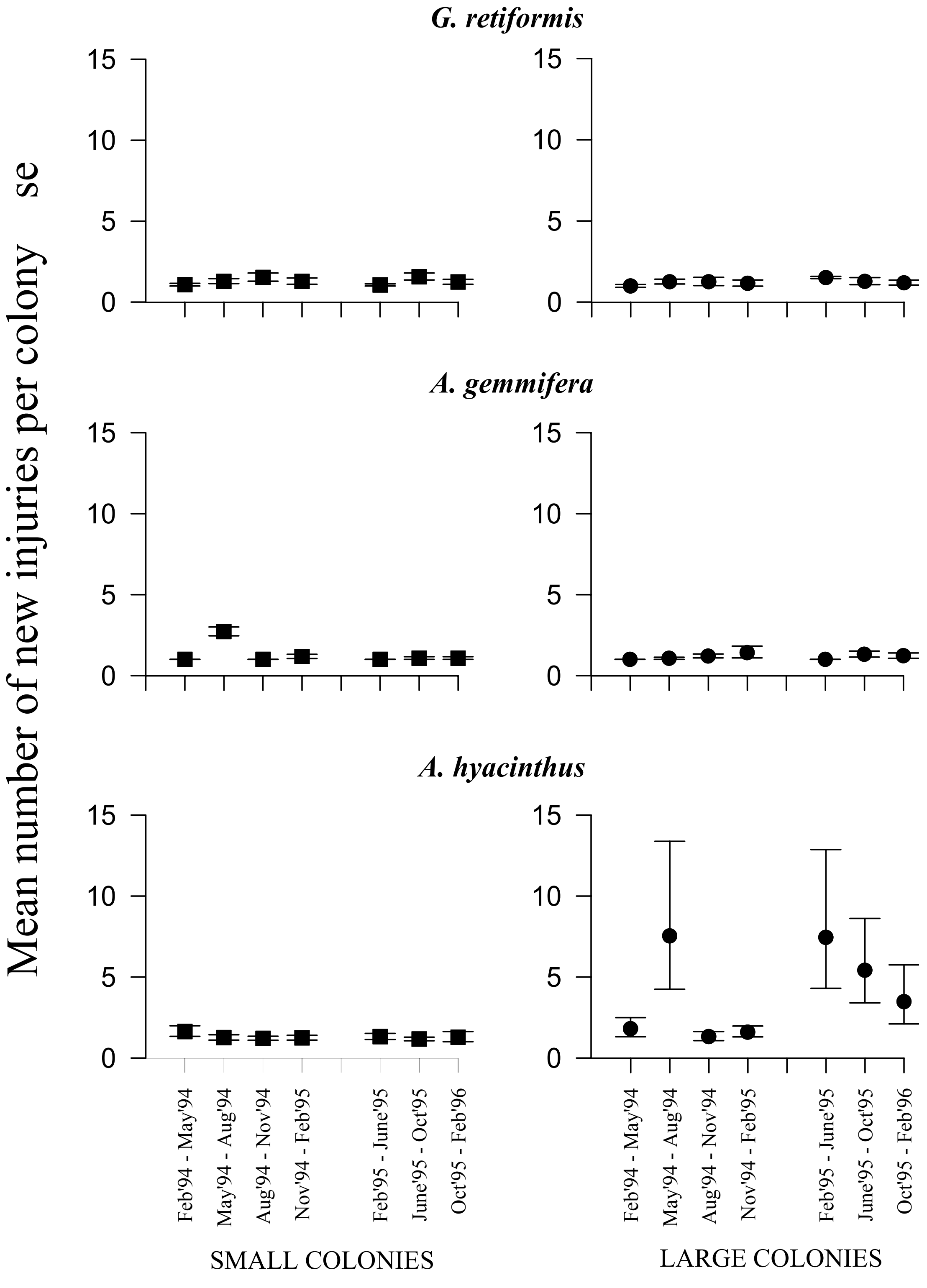
| Source of Variation | df | Type III SS | Mean Square | F-ratio | P |
|---|---|---|---|---|---|
| Species | 2 | 2.001 | 1.001 | 15.26 | 0.000 |
| Colony Size | 1 | 0.414 | 0.414 | 6.31 | 0.015 |
| Species*Colony Size | 2 | 0.998 | 0.499 | 7.61 | 0.001 |
| Error | 51 | 4.197 | 0.066 | | |
| Within Subject Effects | | | | | |
| Time | 3 | 0.668 | 0.223 | 3.20 | 0.024 |
| Time*Species | 6 | 1.721 | 0.287 | 4.12 | 0.001 |
| Time*Colony Size | 3 | 0.686 | 0.229 | 3.29 | 0.022 |
| Time*Colony Size*Species | 6 | 1.595 | 0.266 | 3.82 | 0.001 |
| Error | 192 | 13.353 | 0.070 | | |

b. Feb’95 - June’95, June’95 - Oct’95 and Oct’95 - Feb’96.

Between Subject Effects

| Source of Variation | df | Type III SS | Mean Square | F-ratio | P |
|---|---|---|---|---|---|
| Species | 2 | 3.764 | 1.882 | 9.87 | 0.000 |
| Colony Size | 1 | 2.608 | 2.608 | 13.68 | 0.000 |
| Species*Colony Size | 2 | 2.871 | 1.436 | 7.53 | 0.001 |
| Error | 51 | 9.726 | 0.191 | | |
| Within Subject Effects | | | | | |
| Time | 2 | 0.095 | 0.048 | 1.02 | 0.366 |
| Time*Species | 4 | 0.376 | 0.094 | 2.01 | 0.099 |
| Time*Colony Size | 2 | 0.213 | 0.106 | 2.28 | 0.108 |
| Time*Colony Size*Species | 4 | 0.223 | 0.056 | 1.19 | 0.319 |
| Error | 102 | 4.765 | 0.047 | | |

Figure 2.10 . The mean number of new injuries per colony over time for large and small colonies of *G. retiformis*, *A. gemmifera* and *A. hyacinthus*. (Error bars represent one SE).



### 2.4.3.4. Frequency of Injury

The number of census intervals during which a colony sustained an injury was low for both colony sizes of *G. retiformis* and *A. gemmifera* and for small colonies of *A. hyacinthus* (Table 2.9). In fact, most of these colonies were either injured only in one census period or not at all. In contrast, large colonies of *A. hyacinthus* were injured more often, with 2 or 3 injury events being the most common observed. The frequency of injury among species and colony sizes was consistent over years.

Table 2.9. The number of censuses in which colonies sustained injures recorded over 3 - 4 monthly intervals from 1994 - 1995 and 1995 - 1996 for small and large colonies of *G. retiformis*, *A. gemmifera* and *A. hyacinthus*.

| | Colony | Frequency of Injury | | | | |
|---|---|---|---|---|---|---|
| Species | Size | None | Once | Twice | 3-times | 4-times |
| | | *Februrary 1994 - February 1995* | | | | |
| *G. retiformis* | Small | 5 | 6 | 0 | 0 | 0 |
| | Large | 4 | 5 | 0 | 0 | 0 |
| *A. gemmifera* | Small | 7 | 3 | 0 | 0 | 0 |
| | Large | 4 | 6 | 0 | 0 | 0 |
| *A. hyacinthus* | Small | 6 | 4 | 0 | 0 | 0 |
| | Large | 0 | 3 | 3 | 3 | 1 |
| | | *February 1995 - February 1996* | | | | |
| *G. retiformis* | Small | 6 | 5 | 0 | 0 | - |
| | Large | 6 | 4 | 0 | 0 | - |
| *A. gemmifera* | Small | 6 | 3 | 0 | 0 | - |
| | Large | 5 | 4 | 0 | 0 | - |
| *A. hyacinthus* | Small | 4 | 4 | 0 | 0 | - |
| | Large | 1 | 2 | 3 | 4 | - |

## 2.5. DISCUSSION

Colony size, location, species and time are important in determining the spatial and temporal pattern of injury in scleractinian corals. Vulnerability to damage was species-specific and dependent on the injury regime (i.e. routine versus catastrophic). Understanding routine and catastrophic mortality of corals is important because of their role in structuring coral reef populations and communities through such processes as differential mortality and competitive reversals (Connell 1978; Bak and Luckhurst 1980; Porter *et al.* 1982; Hughes and Jackson 1985; Hughes 1989; Bythell *et al.* 1993; Karlson and Hurd 1993).

### 2.5.1. Spatial patterns of injury

The overall amount of damage (partial mortality) was low ($\leq$2%) among sites around Lizard Island. However, distinct differences were detected between sites with Washing Machine having approximately three times the amount of partial mortality of the other sites (Figure 2.2). This pattern was influenced by the species composition and size-structure of these particular reef-crest assemblages (Figure 2.3, 2.4 and 2.5) because the injury profiles of reef-crest corals was dependent on colony size and morphological group (Table 2.4). Thus the low amount of partial mortality observed at North Reef, Lizard Head and South Reef was due to the domination of bushy and tabular corals (both in terms of numerical abundance and percent cover), corals with low amounts of partial mortality and fewer injured small colonies. In comparison, the higher levels of partial mortality at Washing Machine were due to a smaller number and percent cover of tabular corals and a greater presence of massive and digitate corals (which have more colonies with > 5% partial mortality and fewer uninjured small colonies ) became more important. Partial mortality was higher in massive and digitate corals possibly because they are longer-lived and slower growing than bushy and tabular corals and the recovery of their injuries is slower (Hall 1997). Therefore colonies tend to accumulate injuries over time. Colony size will also affect the degree of damage on a colony. For example, massive and digitate corals are smaller than tabular corals, so any damage inflicted on these corals will affect a disproportionately greater area of the colony.

Alternatively, massive and digitate corals may be more prone to certain types of damage than bushy and tabular corals.

The presence of an injury on a colony at a particular time is a function of how vulnerable the coral is to damage and how fast the injury recovers. In this study, the patterns of injury for small and large colonies of the four main morphological groups were consistent among sites, and therefore the turn - over of injuries on corals was independent of location. A complex interaction among the many biotic and abiotic agents which cause damage to corals and variations in recovery rates of injuries are most likely responsible for this pattern rather than a uniform set of circumstances at each site. Conversely, the distinct differences detected in the proportions of injured and uninjured colonies between morphological groups and colony size, and the proportion of colonies with high and low levels of partial mortality among morphological groups infers that vulnerability to damage and recovery is size-specific and differs among morphological groups.

Vulnerability to damage is complex and dependent on many interrelated factors (e.g. genetics, attractiveness to predators, vulnerability to disease, resistance to physical damage, colony size and location). Morphology also plays a major role (Jackson 1979; Meesters *et al*. 1996, 1997a; Hall 1997) in the degree of vulnerability to damage because (1) the shape of a colony affects the types of interactions that may occur (Jackson 1979; Meesters *et al*. 1996, 1997a), and (2) the mechanical properties of the coral determine resistance to physical damage (e.g. Chamberlain 1978; Liddle and Kay 1987). Meesters *et al*. (1996) classified injuries into two types, type I injuries that were found on the edge of living tissue, and type II injuries that were completely surrounded by living tissue. A survey of these different types of injury demonstrated that *Porites astreoides* (a semi-massive species) had a greater number of peripheral injuries (type II) compared with the branching coral, *Acropora palmata*. This result infers that corals with a large circumference in contact with the substratum are more at risk from benthic interactions than species with a small attachment area. However, corals with a smaller basal attachment (bushy and tabular corals) are more vulnerable to major physical disturbances (e.g. Woodley *et al* 1981), and their resistance to physical damage is dependent on the mechanical strength of coral skeleton, orientation and thickness of branches, and the degree of bioerosion (e.g. Chamberlain 1978; Liddle and Kay 1987).

The rate of recovery of injuries is also complex and related to the characteristics of the injury including the size, shape, position, and type of injury (Bak *et al.* 1977; Bak and Steward-Van Es 1980; Meesters *et al.* 1992; Rinkevich 1996; Meesters and Bak 1995; Meesters *et al.* 1997b; Hall 1997), the degree of colonisation by other organisms of the injured area (Bak *et al.* 1977)**,** and the amount of resources available for regenerative processes (Loya 1976; Bak 1983; Rinkevich and Loya 1989; Van Veghel and Bak 1993; Meesters *et al.* 1994; Rinkevich *et al.* 1996). The injury patterns observed among colony size and morphological group are dependent on both vulnerability to and recovery of injuries. Distinguishing between these two effects can only be achieved by recording the frequency of injury and following the fate of injuries over time as discussed below.

### 2.5.2. Temporal patterns of injury

The temporal patterns of injury (the number of injuries present initially on colonies, the addition of new injuries, and the fate of pre-existing injuries over a 12 month period) was influenced by species, colony size, initial injury size and census year (Table 2.7 and 2.8, Figure 2.6, 2.8, 2.9 and 2.10). The number of pre-existing injuries observed on the massive coral, *G. retiformis* was high while the recovery of these injuries and the addition of new injuries over time was low. Thus, the turn-over of injuries for this long-lived species is sluggish due to slow regeneration rates coupled with a gradual accumulation of new injuries. Although the number of new injuries appearing over time was also low for the digitate coral *A. gemmifera*, its colonies had very few injuries initially and the rate of regeneration of these injuries was faster. Thus, for this moderately long-lived species, the turn-over rate of injuries is much greater, reducing the accumulation of injuries over time. The accumulation of new injuries and the fate of pre-existing injuries for these two species did not change between census years and is probably characteristic of their natural injury regimes in relatively benign conditions. In contrast, the accumulation of new injuries and the fate of pre-existing injuries changed dramatically over census years for the tabular coral, *A. hyacinthus* (Table 2.7

and 2.8, Figure 2.10). In the first census (Feb'94 - Feb'95), the injury regime was quite dynamic with a high turn-over of both new and pre-existing injuries due to relatively high rates of regeneration (Table 2.7). During the second census (Feb'95 - Feb'96), this species was targeted by the corallivore *Acanthaster planci,* resulting in a marked increase in the number of new injuries over time (Figure 2.10), a lower number of injuries recovering, and a greater number of injuries increasing in size (Table 2.7). Thus the turn-over of injuries was greatly reduced in the second census, most probably due to the fact that larger injuries have poorer regeneration rates (e.g. Bak and Steward-Van Es 1980). The injury dynamics of *A. hyacinthus* as observed in the two yearly censuses are characteristic of a species under two different disturbance regimes. Routine levels of damage as seen in the first census appears to have Liddle effect on *A. hyacinthus* colonies. This apparent resistant to damage, if considered in association with their high recruitment and growth rates, may account for their dominance in reef-crest communities at Lizard Island during times of Liddle disturbance. In contrast, the injury dynamics of the second census reveals a species under stress, since the rate of recovery of injuries was down, the number of new injuries was increasing, and pre-existing injuries were getting larger. The continuation of this pattern would most definitely have resulted in the death of the majority of *A. hyacinthus* colonies at this site. The short-time decline in abundance of this dominant species may not be detrimental to the community however, as their removal creates space for other species, thus promoting diversity (e.g. Connell 1978; Bythell *et al*. 1993).

Several studies in the Caribbean have also highlighted interspecific differences in the injury regimes of corals with contrasting morphologies and life-histories (Bythell *et al* 1993; Meesters and Bak 1995; Meesters *et al*. 1996, 1997a; Ruesink 1997). For example, the sub-massive (flattened) coral, *Porites astreoides* had fewer injuries to the internal regions of its colony and recovered more rapidly than the massive species *Siderastrea siderea* (Ruesink 1997). *Porites astreoides* recruits more readily, grows more rapidly and has a higher turn-over of colonies than *S. siderea* (Bak and Engel 1979; Ruesink 1997). High rates of recovery of injures on corals have been associated with a fast growth rate for some species (Hall, 1997; Ruesink 1997) but not others (e.g *Montastrea annularis*, Bak *et al*. 1977; Hughes and Jackson 1985). Regeneration requires resources and is facilitated by the development of new polyps (Bak *et al*. 1977;

Bak 1983). Consequently, variations in regeneration among species result from the different patterns of resource allocation that have evolved towards regeneration and other demographic traits. Further differences may arise due to variations in certain physiological processes (such as skeletogensis and photosynthetically produced energy compounds, Barnes and Chalker 1990) that are an integral part of the recovery of injuries.

Interspecific differences in injury regimes are not restricted to corals with contrasting morphologies. Subtle variations have also been detected between species with similar morphologies but contrasting life-histories (e.g. Hughes and Jackson 1985; Bythell *et al*. 1993). For example, routine (chronic) injury rates for two massive species, the meandroid *Diploria strigosa* and the cerioid *Montastrea annularis* differed significantly, being low for the former and high for the latter species (Bythell *et al* 1993). Furthermore, the incidence of partial mortality was much higher for semi-massive colonies of *Porites astreoides* than semi-massive colonies of *Meandrina meandrites* (Meesters *et al*. 1997a). In addition to contrasting life-histories, differences in the structural and chemical defences of these species may account for some of the variations detected among injury regimes of corals with similar morphologies (Meesters *et al*. 1997a). High levels of toxins may deter predators as shown for soft corals (e.g. Coll *et al*. 1982), long tentacles armed with nematocysts may discourage competitors (Lang and Chornesky 1990), and polyps or branches with dense skeletons may resist mechanical damage (e.g. Liddle and Kay 1987)

The number of pre-existing injuries and the addition of new injuries were greatly affected by colony size. This pattern was particularly evident for *A. hyacinthus* due to at least an order of magnitude difference in the average size of small and large colonies. The majority of pre-existing injuries were found on large colonies for all species (Figure 2.7), and the addition of new injuries was lower for small colonies as compared with large colonies for *A. hyacinthus* (Figure 2.10). Additionally, the frequency of injury was also greatest for large colonies of *A. hyacinthus* (Table 2.9). Given that most colonies were injured only once in 12 months, (with the obvious exception of large colonies of *A. hyacinthus*), larger colonies have a greater probability of being injured by chance alone because they have been exposed longer than most small colonies to partial mortality agents (Hughes and Jackson 1985; Babcock 1991, Bythell *et al*. 1993). Frequency of injury may be higher in large *A. hyacinthus* colonies because they have a greater surface area and thus a greater probability of being injured. Vulnerability to injury is also highly dependent on the causative agent, and the morphological characteristics of the coral (Jackson 1979). For example, small colonies are likely to be vulnerable to peripheral injuries due to their high circumference to surface area ratios (Jackson 1979; Meesters *et al*. 1997a; Meesters *et al*. 1997b). Generally, injury has a disproportionately greater effect on small corals than large corals, often resulting in the death of small colonies (Hughes and Jackson 1985; Babcock 1991; Bythell *et al*. 1993; Meesters *et al*. 1997a). High mortality of small colonies is likely to be related to the high ratio of damaged to undamaged tissue which occurs after injury so that there are fewer polyps to carry out biological functions (e.g. feeding, photosynthesis, and maintenance) as compared with larger colonies, and the colony dies (Connell 1973; Hughes and Jackson 1985; Babcock 1991).

The characteristics of an injury such as shape, size, type and position within the colony can all influence rates of recovery (Bak *et al*. 1977; Bak and Steward-Van Es 1980; Meesters *et al*. 1992; Meesters and Bak 1985; Hall 1997; Meesters *et al*. 1997b; Oren *et al*. 1997). In this study, the fate of an injury was highly dependent on initial size (Table 2.7; Figure 2.9). Although most injuries remained the same regardless of initial size, a greater proportion of smaller than larger injuries recovered. It has been suggested that the slower recovery rates observed for larger injuries are due to finite resources available for regeneration, trade-offs in available resources between regeneration and competition due to the settlement of other organisms onto the injury site, and/or less healthy tissue per area of damage from which regeneration can occur (Loya 1976; Bak *et al*. 1977; Bak and Steward-Van Es 1980; Rinkevich and Loya 1989; Meesters *et al*. 1994; Rinkevich 1996; Meesters *et al*. 1997b; Oren *et al*. 1997).

In conclusion, colony size, species, initial injury size and the composition of coral communities are all important in determining the spatial and temporal patterns of injury for reef-crest corals at Lizard Island. Differing vulnerabilities to damage, both among and within species under different injury regimes may dramatically alter the dynamics of coral populations (e.g. Hughes 1984; Bythell *et al*. 1993). Routine injuries cause the death or partial mortality of coral colonies, a loss often offset by larval recruitment, asexual reproduction and growth (Bak and Luckhurst 1980; Hughes and Jackson 1985), while catastrophic events (e.g. cyclones, bleaching events and predator outbreaks) may have a deleterious, short-term effect on targeted species (Bythell *et al.* 1993). This, in turn, may impact on coral communities since the differential mortality of dominant species leads to the promotion of species diversity through the freeing up of space for settlement and the growth of less common species (e.g. Connell 1978). Consequently, understanding routine and catastrophic mortality on coral reefs is important because of their role in structuring coral reef populations and communities (Connell 1978; Bak and Luckhurst 1980; Porter *et al.* 1982; Hughes and Jackson 1985; Hughes 1989; Bythell *et al.* 1993; Karlson and Hurd 1993).

# CHAPTER 3: INTERSPECIFIC DIFFERENCES IN THE REGENERATION OF ARTIFICIAL INJURIES ON SCLERACTINIAN CORALS.

## 3.1 ABSTRACT

Routine injury of corals occurs frequently and is caused by natural processes and human activities. Morphological theory predicts that sessile, marine animals that are strongly committed to their place of settlement will invest more resources into maintenance and defence than species that are more fugitive in nature (Jackson 1979). In this study I conducted several field experiments to determine if the regenerative ability of injured colonies differed among species with different morphologies, and if regeneration was influenced by the position of the injury within the colony or the type of injury. Eleven species exhibited a wide range of responses to damage, from little to no change to complete recovery within 71 to 286 days. Regenerative ability was able to be ranked according to morphological attributes for these species (arborescent > bushy > tabular > massive > submassive), suggesting that morphology does influence the recovery of the coral from injury. Regeneration from different types of injury and injuries located at different positions within a colony also varied among species. Repair of scraping injuries was greater than tissue injuries, while regrowth of a new branch was slowest of all. With the exception of *Porites mayeri* which repaired more of its central injury than its edge injury, recovery rate of central and edge injuries was not significantly different for *Acropora robusta*, *A. hyacinthus*, *A. palifera*, *Pocillopora damcornis*, and *Porites lichen*. These results imply that the amount of damage caused by injuries of equitable size differ both for the species it is inflicted upon, and the type of injury generated. The ability to recover from injuries in important in corals since poor regenerative ability can lead to a reduction in colony fitness.

## 3.2 INTRODUCTION

Damage or injury to coral colonies originates from natural causes (e.g. predation, competition, storm and cyclone damage) or human activities such as anchoring, diving, mining, and pollution (e.g. reviews by Connell 1973; Brown and Howard 1985; Craik *et al*. 1990; Grigg and Dollar 1990; Brown 1997). Corals have varying susceptibilities to damage by these processes due to morphological differences (Woodley *et al*. 1981; Hughes 1989; Glynn 1990; Chadwick-Furman 1995). For example, branching species are more prone to storm and diver damage than massive species (Woodley *et al*. 1981; Chadwick-Furman 1995), and massive and encrusting species are more vulnerable to overgrowth by algae (Hughes 1989). Their ability to recover from damage is also variable since regeneration is energetically costly, and species differ in the allocation of resources between regeneration and other demographic processes (e.g. Bak *et al*. 1977; Bak and Steward-Van Es 1980; Meesters *et al*. 1992; Meesters and Bak 1993). Morphological theory of sessile, marine animals predicts that species with a greater commitment to their place of settlement will invest more resources towards defence and maintenance (Jackson 1979). This hypothesis was based on eight shape parameters which characterised the different morphological strategies of sessile animals. These parameters included tissue area and volume (potential for feeding, respiration and reproduction, etc.), skeletal volume (measure of support material and strength), substratum utilisation, directional growth (vertical or horizontal), substratum interactions (pre-emption of space, peripheral encounters), and holes (complexity of form in determining the availability of habitats for other organisms). The impact that an injury has on a colony will be directly related to these parameters, particularly tissue and skeleton volume and tissue area. The first major objective of this study is to determine if there is a correlation between morphology and rates of regeneration in corals by monitoring the recovery of artificial lesions for a range of species with contrasting shapes.

The recovery of injuries in corals is a complex process that is intrinsically related to the characteristics of the injury (e.g. type and position within the colony: Bak *et al*. 1977; Bak and Steward-Van Es 1980; Meesters *et al*. 1992; Meesters and Bak 1995). Routine damage in corals results in the removal of tissue, or tissue and skeleton from a colony.

The amount of loss which occurs for these two injury types can be further categorised as partial or total for tissue injuries and superficial or extensive for tissue and skeleton injuries. A wide range of causative agents generate these different types of injuries. Generally, extensive tissue loss results in the death of polyps within a given area and is due to predation (e.g. *Acanthaster planci*), sedimentation, and solar irradiance (amongst other factors) while the partial removal of polyps (partial tissue loss) is caused by the action of polyp grazers such as some Chaetodontid fish (Glynn 1990; Stafford-Smith 1993; Brown 1988,1994). The superficial loss of tissue and skeleton is produced by scraping injuries which result from physical processes (e.g. abrasion), human activities (fin and anchor scrapes), and predation (e.g. parrot fish). The loss of extensive amounts of tissue and skeleton is the removal of bumps or branches from colonies and is due to predation by excavating fish (e.g. *Bolbometopon* sp.), physical disturbances (e.g. storms and cyclones) and human activities such as diving and anchoring (e.g. Woodley *et al*. 1981 Glynn 1990; Craik *et al*. 1990). For a given injury type, the morphological characteristics of a species (e.g. shape and tissue depth) can affect the appearance of an injury (Glynn 1990). For example, although the outcome of feeding by some parrot fish is the superficial loss of tissue and skeleton from a colony, variations in colony shape result in different wound manifestations: gouging wounds are produced in massive species, branchlets are lost in tabular corals, and tips are removed in arborescent corals (Glynn 1990). The second major objective of this study is two-fold, firstly to examine how species respond to different types of injuries, and secondly, to investigate the impact of a given injury on species with different morphologies by monitoring their response (regenerative ability) to that injury.

The position of an injury within a colony is again dependent on the causative agent, and affects rates of regeneration in bryozoans (Palumbi and Jackson 1982), the gorgonian *Plexaura homomalla* (Wahle 1983), and some scleractinian corals such as *Acropora palmata* (Meesters and Bak 1995), but not others (e.g. *Porites astreoides*, Meesters *et al*. 1992). Reduced light levels, polyp density, senescence, and substratum interactions have all been proposed to explain variations in the regeneration of lesions located at different positions within a colony (Jackson 1979; Palumbi and Jackson 1982; Darke 1991; Meesters *et al*. 1992; Meesters and Bak 1995). Preferential regeneration of one injury versus another within colonies of some species may have direct consequences for their population dynamics. For example, Meesters and Bak (1995) showed that the regeneration of proximal injuries was much slower than distal injuries on branches of *Acropora palmata,* and suggested that this pattern of recovery may promote asexual propagation by fragmentation. The third major objective of this study is to determine if there was any variation in the rate of regeneration of injuries located at different positions within a colony.

The response of corals to injury is important ecologically because injury reduces colony fitness in three ways. Firstly, regeneration requires energy so that resources may be diverted from growth and reproduction (e.g. Kobayashi 1984; Rinkevich and Loya 1989; Meesters *et al*. 1994; Van Veghel and Bak 1994). Secondly, colony survival may be jeopardised since injuries provide sites for the entry of pathogens and bioeroders and space for the settlement of other organisms such as algae, sponges, and other corals (Bak *et al*. 1977). These organisms may later compete with the coral for food and space, or cause structural damage to the coral skeleton (Wahle 1983; Hughes and Jackson 1985; Babcock 1991). Thirdly, injuries reduce the surface area available for feeding, photosynthesis and reproduction (e.g. Jackson and Palumbi 1979; Wahle 1983; Hughes and Jackson 1985). A reduction in surface area may also alter colony survivorship and reproduction since both are size-specific in scleractinian corals (e.g. Hughes and Jackson 1985; Babcock 1991; Hall and Hughes 1996). Understanding the processes associated with injury and regeneration in corals has also become very important from a management perspective because of the escalating degradation of coral reefs by human activities (e.g. Brown 1988; Craik *et al*. 1990; Hughes 1994; amongst many others). With the exception of studies which have being carried out predominantly in the Caribbean (see Table 3.1), there is a general lack of information on the regenerative abilities of scleractinian corals through field experimentation. This study is one of the first to present experimental data on the regenerative abilities of eleven species of scleractinian corals from the Great Barrier Reef.

**Table 3.1:** A list of studies showing the places and scleractinian corals for which experimental studies on injury and regeneration have been conducted.

| SPECIES | REFERENCE |
|---|---|
| Caribbean | |
| *Acropora palmata* | Bak 1983; Meesters *et al*. 1992; 1997b<br>Meesters and Bak 1995 |
| *Montastrea annularis* | Bak *et al*. 1977; Meesters *et al*. 1983, 1994; Van Veghel and Bak 1994; |
| *Meandrina meandrites* | Meesters and Bak 1993 |
| *Porties astreoides* | Bak and Steward-Van Es 1980;<br>Meesters *et al*. 1992 |
| *Agaricia agaricites* | Bak *et al*. 1977;<br>Bak and Steward-Van Es 1980 |
| *Siderastrea siderea, Diploria Strigosa* | Meesters *et al*. 1992 |
| Red Sea | |
| *Stylophora pistillata* | Loya 1976; Rinkevich and Loya 1989 |
| *Favia favus* | Oren *et al*. 1997 |
| Japan | |
| *Acropora formosa, A. nasuta* | Kobayashi 1984 |
| Western Australia | |
| *Pocillopora damicornis* | Ward 1995 |
| Great Barrier Reef, Australia | |
| *Acropora millepora,*<br>*Acropora palifera*<br>*Pocillopora damicornis*<br>*Porites lutea* | Liddle and Kay 1987 |
| *Pocillopora damicornis, Goniastrea retiformis, Porites mayeri, P. lichen, P. australiensis, Acropora robusta, A. millepora, A. cytherea, A. palifera A. hyacinthus, A. gemmifera* | This Study |

## 3.3 MATERIALS AND METHODS

This study was conducted at Lizard Island (14°40',145°28'), a continental island on the Great Barrier Reef, approximately 25 km off the eastern coast of mainland Australia (Figure 2.1). Experiments were set up at two locations, on the reef-crest at North Reef (experiment 1), and in the lagoonal back-reef of Osprey Island (experiment 2). These sites were chosen because they provided a wide selection of species and an abundance of large colonies (see Table 3.2 for average colony sizes). One hundred and ten colonies were selected haphazardly for the experiments and individually tagged. Colonies with injuries comprising greater than 25% of the total surface area were excluded. Three of the four injury types were experimentally inflicted on colonies to simulate natural injuries. These include extensive tissue loss (tissue injury), superficial tissue and skeleton loss (scraping injuries) and extensive tissue and skeleton loss (branch removal). Tissue was removed from colonies with compressed air that was delivered through the small nozzle (5mm diameter) of an air gun. To standardise the injury procedure for all colonies, the air pressure of the gun was maintained at 60 psi, and the nozzle was held at a distance of 3cm away from the colony. All visible tissue was removed from the injured areas (see Bak *et al*. 1977). A template made of water-proof paper was used to obtain an injury of a specific area by protecting the surrounding healthy tissue from damage. This procedure left the underlying skeleton of the colony relatively intact (i.e. there was no visible damage to the architecture of the skeleton). A hammer and chisel was used to remove tissue and skeleton (scraping injury) and break off branches (branch removal). Recovery of injuries was quantified by either recording the dimensions of the lesion with a tape measure or digitising the lesion area from photographs of consecutive censuses. These data were then expressed as percentage of injury regenerated (as per Meesters *et al*. 1992).

**Table 3.2:** Mean colony diameter ± se (cm) for the study species. Values are based on measurements taken of the maximum diameter and the diameter perpendicular to the maximum diameter for each colony. (n = 20).

| Species | Mean Diameter ± se |
|---|---|
| *Goniastrea retiformis* | 21 ± 2 |
| *Acropora hyacinthus* | 68 ± 7 |
| *A. robusta* | 51 ± 6 |
| *A. palifera* | 21 ± 2 |
| *A. gemmifera* | 23 ± 2 |
| *A. millepora* | 35 ± 3 |
| *A. cytherea* | 47 ± 4 |
| *Porites lichen* | 26 ± 4 |
| *P. mayeri* | 21 ± 2 |
| *P. australiensis* | 50 ± 4 |
| *Pocillopora damicornis* | 18 ± 1 |

### 3.3.1 Experiment 1:

#### Interspecific differences in recovery and injury position within colonies.

To determine if recovery from a tissue injury differed between species, or position within a colony, I inflicted a 4 $cm^2$ tissue wound at two positions (edge and centre) within 10 replicate colonies of each of seven species. Common reef-crest species with contrasting morphologies were chosen and included *Acropora robusta, A. hyacinthus*, *A. palifera*, *Pocillopora damicornis*, *Porites mayeri*, *P. lichen*, and *Goniastrea retiformis.* The growth forms of the first four species are arborescent, tabular, submassive, and bushy respectively, while the last three species are massive. For the massive, tabular and bushy corals, the injuries were located at the center and edge of the colony while for the arborescent and submassive species, injuries were located at the base (centre) and tips (edge) of branches and columns. Seventy-four days after injuring the colonies, the dimensions (length and width ± 1mm) of each lesion (amount of injured area not regenerated) was measured using a tape measure. The area of the remaining lesion was then calculated and subtracted from the original injury size to estimate the amount of recovery.

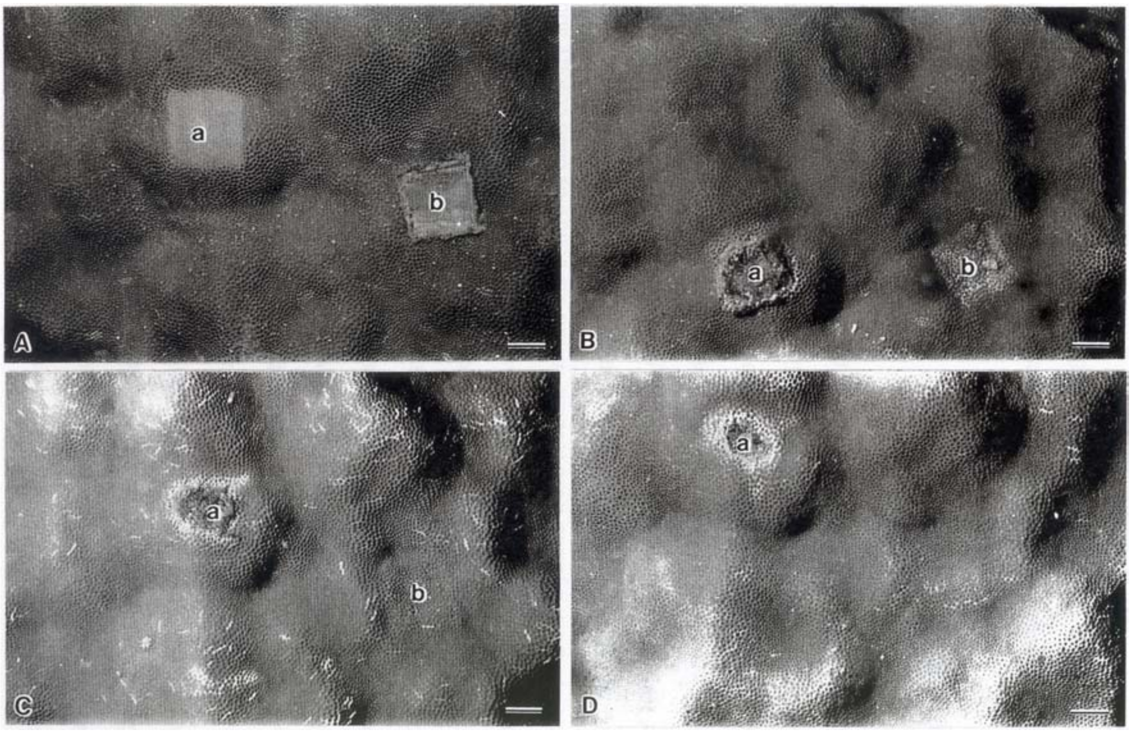
### 3.3.2. Experiment 2:

### Interspecific differences in recovery and injury types.

#### **3.3.2.1** Tissue Removal versus Scraping Injuries in Massive and Tabular Corals.

To examine the response of corals to a scraping versus a tissue injury, and to investigate the impact of a scraping injury on species with different morphologies, each of 10 replicate colonies were inflicted with both injury types, the size of the injury being 4 $cm^2$ (Figure 3.1). Two abundant coral species, the table coral *Acropora cytherea* and the massive coral *Porites australiensis* were examined to detect any interspecific differences in regeneration. Tissue was removed with compressed air as described previously, while the scraping injury was created using a hammer and chisel. For *P. australiensis*, the scraping injury was inflicted by chiselling away tissue and skeleton to a depth of 2 mm resulting in the partial injury of polyps within the injury site (average tissue depth in *Porites* is 5 mm, Barnes and Lough 1992). For *A. cytherea*, 10 -15 branchlets, 2 cm in height, were removed at their base with the chisel to create a 4 $cm^2$ projected area injury. The scraping injuries differed between *A. cytherea* and *P. australiensis* because they were simulating the type of injuries generated by some parrotfish. Injuries were located in the central section of the colony to avoid edge effects, and one of each type of injury was inflicted per colony. Each injury type was separated from the other by a distance of at least 5 cm. Recovery was monitored photographically over time and recorded as the amount of tissue produced around the injury.

**Figure 3.1:** A photographic series showing the recovery of the tissue and scraping injuries inflicted on *Porites australiensis* over time, (where a = tissue injury and b = scraping injury). A. Infliction of the injury in August. B. Recovery of injuries after 71 days. Note that the scraping injury has nearly completely regenerated while the unrepaired section of both injuries is overgrown with algae. C. Complete recovery of the scraping injury and partial recovery of the tissue injury. D. The tissue injury is still only partially recovered after 215 days. Scale bar = 1cm.



### **3.3.2.2** Tissue Loss versus Branch Removal in Branching Corals.

Branching acroporids on the Great Barrier Reef are prone to injuries (e.g. predation, physical disturbances) which result in either the removal of branches or tissue loss. To determine how branching corals respond to branch removal and tissue loss, and to detect interspecific differences in their response to these injury types, I removed the tissue from one branch and broke off another branch in ten colonies of two species of corals. These species, *Acropora millepora* and *A. gemmifera* have different branching morphologies, corymbose and digitate respectively. Branch removal resulted in a small injury at the base of the branch, the recovery of which was monitored in addition to the regeneration of the two injury types. The dimensions of the removed branch, exposed basal area, and tissue-denuded branch were measured and used to calculate branch volume lost and injury area from appropriate geometric formulas (cone shape for *A. gemmifera* branches and cylinder plus a cone top for *A. millepora* branches, see Tuma 1979 for formulas). Both injuries were located within the centre of each colony and separated by a distance of 5 cm. Recovery was measured at 74 days. Due to the loss of 2 tags, 8 out of 10 colonies were censured for *A. gemmifera*. Regeneration was defined as the growth of a new layer of polyps over the injury site for tissue removal and the exposed area at the base of the branch, while recovery of the branch was defined as the volume of new growth. The amount of new vertical growth for both tissue injury and branch removal was also recorded to allow comparisons between injury types.

### **3.3.3** Statistical Analysis

An analysis of variance (ANOVA) was used to test for differences between species and injury positions or types. A Least Significant Difference Test (LSD) was used to determine differences between significant factors within the anova model. Data not conforming to the assumptions of anova were tested using a Kruskal-Wallis One-way non-parametric ANOVA.

## 3.4 RESULTS:

### 3.4.1. Experiment 1:

**Interspecific differences in recovery and injury position within colonies.**

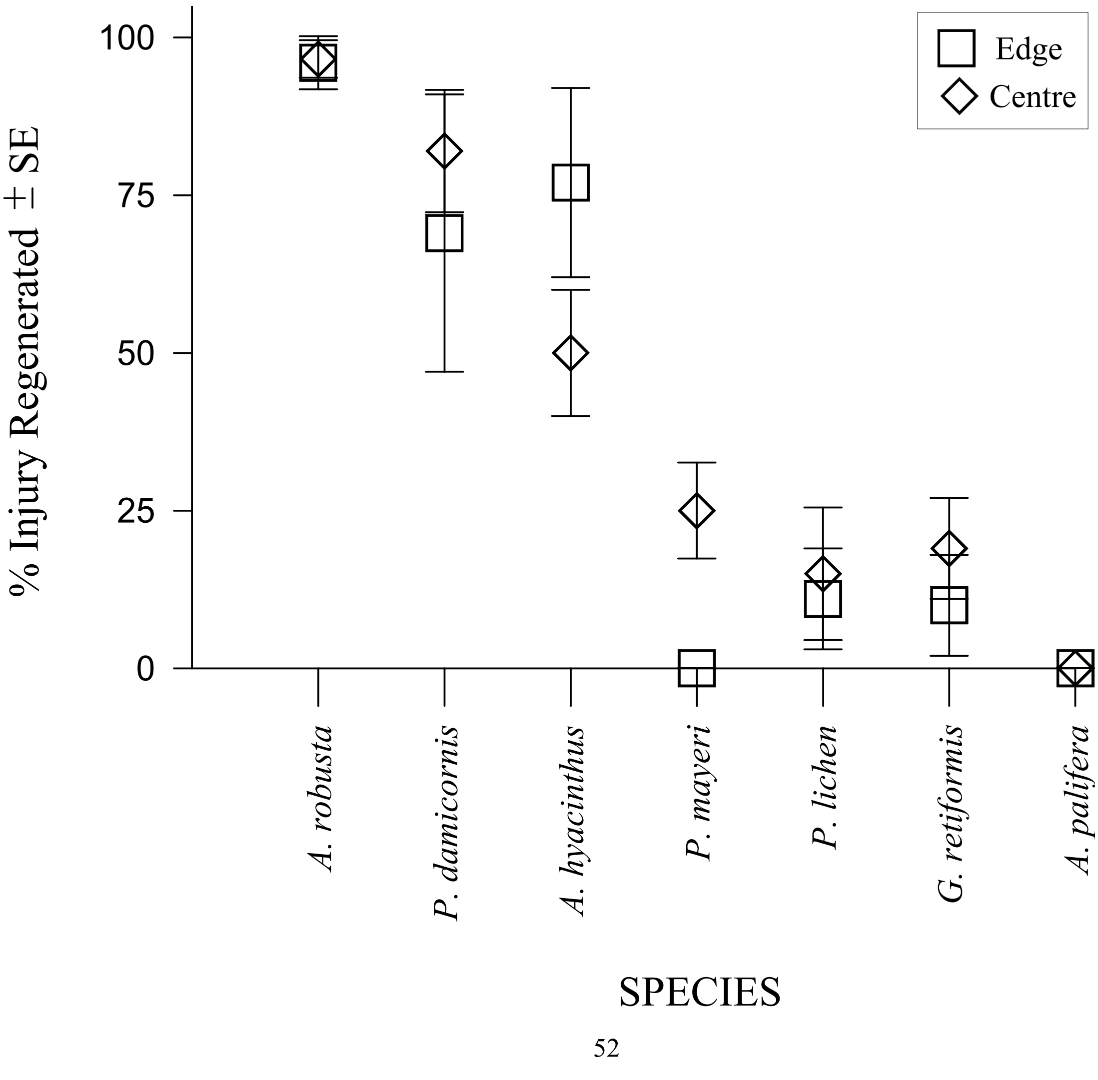
Striking differences were detected in the regenerative ability of corals (Figure 3.2, Table 3.3). In general, the branching species regenerated a greater proportion of their injuries than the massive and submassive species within 74 days (Figure 3.2). Specifically, *A. robusta* regenerated approximately 1.5 times more of the injured area within 74 days than *P. damicornis* and *A. hyacinthus*, which in turn, regenerated 4-5 times more of their injuries than did *P. lichen*, *G. retiformis*, *P. mayeri* and *A. palifera* (LSD test). The position of the injury did not affect the rate of regeneration in *A. robusta*, *A. hyacinthus*, *P. damicornis*, *P.lichen*, *G. retiformis* and *A. palifera* (LSD test). In contrast, *P. mayeri* regenerated 25% of its central injuries but none of the edge injuries within 74 days
(Figure 3.2).

The removal of tissue with compressed air resulted in the death of tissue within the template area for all species, as evidenced by the subsequent colonisation of the injury site by algae. Consequently, recovery of the injury involved the overgrowth of the algae with new polyps. Two different modes of regeneration were observed for the tissue injury. Regeneration in *Acropora* species was characterised by the production of a smooth band of tissue and skeleton which enveloped the injury and grew over the colonising algae. Polyps were then produced along the margin of the band that was closest to the uninjured tissue. In contrast, rather than producing a smooth band, the massive species (*Porites lichen, P. mayeri and G. retiformis*) and *Pocillopora damicornis* produced new polyps directly around the margin of the injury.

**Table 3.3:** Summary of ANOVA results for the comparison of the amount of regeneration between species and injury position for experiment 1 at North Reef, Lizard Island.

| Source | df | F ratio | P |
|---|---|---|---|
| Species | 6 | 54.48 | 0.000 |
| Injury Position | 1 | 0.16 | 0.686 |
| Species * Injury Position | 6 | 2.19 | 0.048 |
| Error | 122 | | |

**Figure 3.2:** The amount of regeneration of a central and edge injury for several common reef-crest species over 71 days. (Error bars represent one SE).



### 3.4.2 Experiment 2*:*

### Interspecific differences in recovery and injury types.

Marked differences were detected in the recovery of injuries inflicted on *Acropora cytherea* and *Porites australiensis*. Repair was greater for tabular *A. cytherea* than for the massive, *P. australiensis* (Figure 3.3). *Acropora cytherea* showed almost complete recovery from both injury types (i.e. scraping injury and tissue loss) by the first census at 71 days (Figure 3.3) while *P. australiensis* repaired the scraping injury much faster than the tissue injury, with recovery being complete within 153 and 286 days respectively (Figure 3.1 and 3.3). For both injury types, *P. australiensis* showed a decrease in regeneration rate with time (Figure 3.3). The mode of regeneration of these species was similar to that described previously (experiment 1), with *A. cytherea* responding the same as the other *Acropora* species and *P. australiensis* responding similarly to the other massive species.

Striking differences were also detected in recovery of tissue loss versus branch removal for *A. millepora* and *A. gemmifera* (Table 3.4). The amount of vertical extension of tissue along the branch was approximately 10 times greater than the vertical extension of a new branch (Table 3.4a). This result appears to be related to differences in the patterns of regeneration observed between injury types. Regeneration of the tissue injury in the *Acropora* species involved the sealing of the area with a fine, porous layer of tissue and skeleton, followed by the development of polyps within this area. Branch replacement also involved a number of intergrading stages. Firstly, the base of the branch where the breakage had occurred was sealed by a thin layer of skeleton and tissue. Secondly, a central axial polyp and several radial polyps then developed within this thin layer and thirdly, vertical extension of the branch occurred with the growth of the axial polyp. The initial extension of the axial polyp produced a narrow branch (0.3 cm diameter) which gradually thickened at the base.

The pattern of regeneration of a given injury type was similar for *A. millepora* and *A. gemmifera* (Table 3.4b), although some inter-colony variations were observed. All

Figure 3.3: Percentage of injury regeneration over time for *P. australiensis* and *A. cytherea*. (Error bars represent one SE).

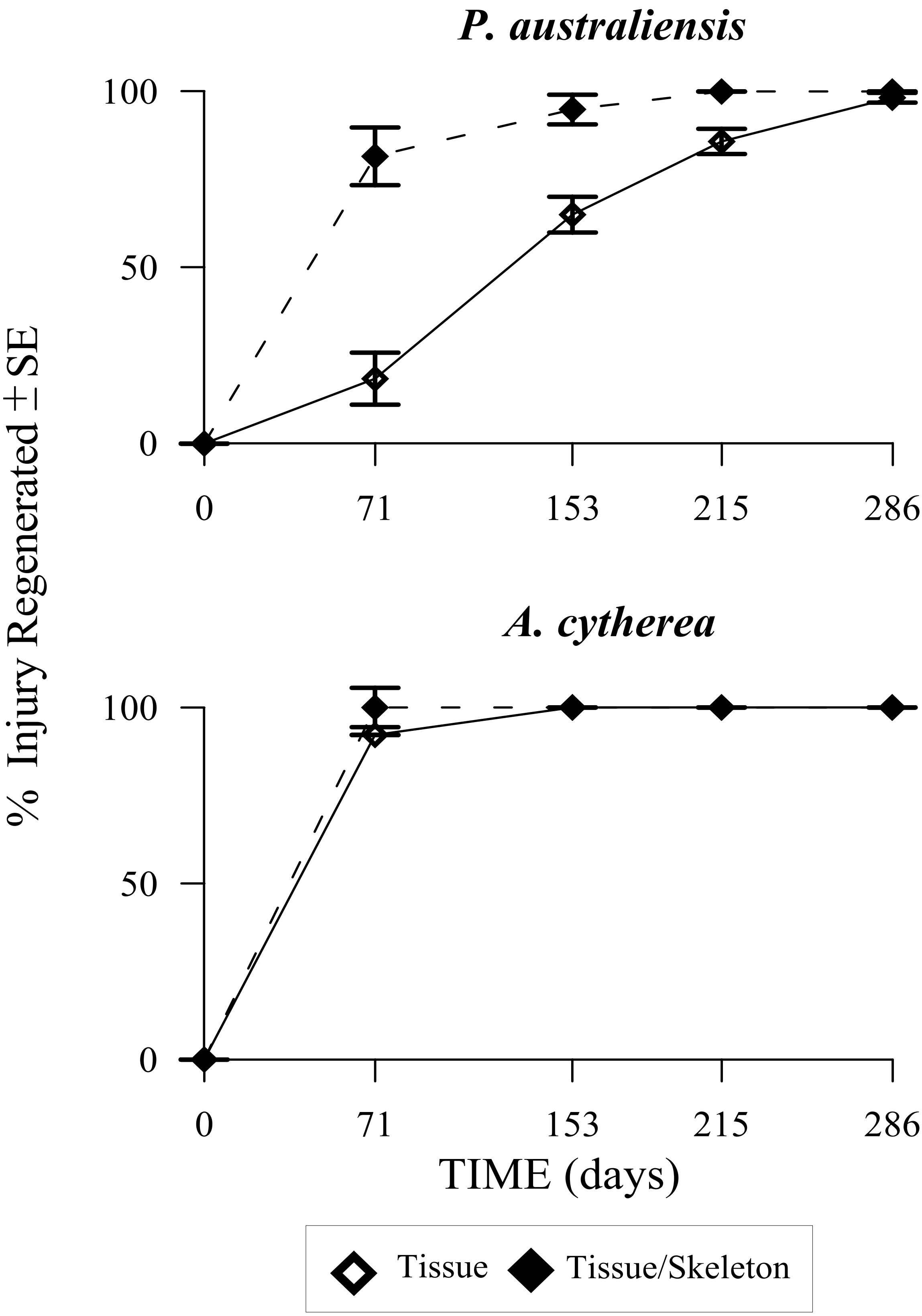


**Table 3.4:** (a) Linear extension of new growth for the two injury types (tissue removal and branch removal) inflicted on *A. millepora* and *A. gemmifera* in experiment 2b (mean ± 1 SE). KW represents the Kruskal- Wallis statistic obtained by comparing the differences in linear extension between the two injury types, and P equals the probability. (b) Injury size and regeneration (percent of injury recovered) for the two injury types inflicted on *A. millepora* and *A. gemmifera* in experiment 2b (mean ± 1 SE). (Number of colonies =8 and 10 for *A. gemmifera* and *A. millepora* respectively).

(a)

| Injury Type | *A. gemmifera* | | *A. millepora* | |
|---|---|---|---|---|
| | Regeneration | Statistic | Regeneration | Statistic |
| Linear Extension of Tissue (mm) | 3.19 ± 0.86 | KW=14.4<br>P=0.0001 | 4.3 ± 0.31 | KW=11.3<br>P=0.0008 |
| Linear Extension of Branch (mm) | 0.25 ± 0.13 | | 0.55 ± 0.12 | |

(b)

| Injury Type | *A. gemmifera* | | *A. millepora* | |
|---|---|---|---|---|
| | Regeneration | Injury Size | Regeneration | Injury Size |
| Tissue Removal ($cm^2$) | 49 ± 23.0 | 25 ± 2 | 68 ± 19.0 | 21 ± 2.0 |
| Branch Removal ($cm^3$) | 3.5 ± 1.2 | 7 ±0.7 | 7.0 ± 2.2 | 6 ± 0.9 |
| Exposed area at branch base ($cm^2$) | 100.0 | 2.92 ± 0.2 | 100.0 | 0.98 ± 0.1 |

colonies for both species showed some degree of regeneration of the tissue injury (percent of injury recovered was 68 ± 19% and 49 ± 23% for *A. millepora* and *A. gemmifera* respectively) after 74 days (Table 3.4b). Additionally, they both rapidly repaired the injured area at the base of the branch where breakage occurred, completely sealing the area in less than 74 days (Table 3.4b). However, within this sealed area, there was some variation in polyp development between colonies for *A gemmifera* with

25% of colonies displaying no signs of polyp development by the end of the experiment.

Conversely, all *A. millepora* colonies showed some polyp development within the 74 day period. Some variations in the number of colonies of each species exhibiting vertical branch extension was also detected. In *A. gemmifera*, 3 out of 8 colonies showed some signs of vertical extension, and replaced an average of 7 ± 0.7% of the original branch volume. On the other hand, the majority of *A. millepora* colonies (8 out of 10) extended vertically, showing an average of 10.4 ± 6.7% replacement of the original branch volume. Although the overall pattern of regeneration of the different injury types was similar for *A. gemmifera* and *A. millepora*, regeneration was faster in *A. millepora* than *A. gemmifera* (Table 3.4b). This was probably due to the fact that the injuries inflicted on *A. gemmifera* were larger (Table 3.4b).

## 3.5 DISCUSSION

Regenerative ability in this study differed both between species for a specific injury and between different injury types for a particular species. Generally, colonies of branching species regenerated more of their injury within a given time period than did the massive species (Figure 3.2 and 3.3, Table 3.4). Furthermore, rankings of the recovery of the different types of injuries are as follows: scraping injury >tissue injury>branch removal (Figure 3.2, 3.3 and Table 3.4). The impact that an injury has on a particular species seems to be reflected in the ability of the colony to regenerate.

### 3.5.1 Interspecific differences in regeneration

Jackson (1979) proposed that colonial organisms with morphologies that showed an increasing commitment to their place of settlement would invest more resources into defence and maintenance as opposed to those morphologies which were more fugitive in nature. A ranking of the regenerative ability of the species examined in this study show a similar result to that proposed by Jackson (1979): Arborescent (Tree) ≥ Bushy (Tree) ≥ Tabular (Plate) > Massives (Mounds) > Submassive (Sheet). The impact that a particular injury has on a coral is related to tissue and skeleton volume and tissue area.

The amount of tissue covering a colony infers something about its potential for feeding, respiration, and reproduction as well as offering a means of defence against biotic and abiotic processes through allelochemistry, nemocyst discharge, mucus production, and so on. In corals, some species are fleshier than others (Veron 1986), so that the impact of a tissue injury will depend on the depth of tissue covering the colony and implies that the deeper the tissue the greater the loss. In this study, the regeneration of these tissue injuries was slower for massive species than for branching species and may be related, in part, to tissue depth which may be greater in massive corals. However, since the repair of tissue injuries usually involves the production of both skeletal and tissue matter, the amount of investment in skeletal material will also be important. Although there is a great deal of interspecific variation in the density of coral skeletons, skeletal densities can usually be associated with different growth forms (Hughes 1987). For example, many branching *Acroporas* have a pronounced axial gradient in skeletal density, with the base being much denser than the tips (Hughes 1987). In these species, the mode of regeneration of small injuries is distinctive. The injured area is rapidly sealed with a fine, porous layer of tissue and skeleton, followed by the development of polyps within this area (Bak 1983, this study). This mode of recovery suggests that the skeleton produced in regenerating areas has a similar density to that of the faster growing tips. In contrast to the branching *Acroporas*, the submassive *A. palifera* does not have a pronounced axial gradient in skeletal density but maintains a fairly dense column from tip to base (average density = 1.9 ± se 0.12, n = 6). Additionally, it did not regenerate any of its injured area within 74 days. This infers that the production of such a dense skeleton during regeneration may incur a large drain on resources and slow recovery in *A. palifera* colonies. Similarly, differences in the skeletal investments of *A. millepora* and *A. gemmifera* influence their regeneration rates. The volume of tissue and skeleton lost in branch removal was 16% less for *A. millepora* than for *A. gemmifera*, while the amount of regeneration after 74 days was approximately double (Table 3.4).

### 3.5.2 Injury characteristics

The characteristics of an injury play a major role in establishing rates of regeneration because they determine the amount of damage which is inflicted on a colony. Marked differences were detected in the regeneration rates of the three different injury types, tissue, scraping, and branch removal. For example, the recovery of a tissue injury versus branch removal in colonies of *A. millepora* and *A. gemmifera* varied within a period of 74 days (Table 3.4), with vertical extension of new tissue being approximately 10 times faster than branch extension. This infers that the loss of tissue from a branch has less impact on a colony than the removal of a branch. This may be due to the fact that the amount of material required to replace the lost tissue is much less than that needed to replace a branch since there is less investment required for skeletal material (i.e. the framework of the branch is left intact and only a thin layer of porous skeleton is needed for recovery). Colony survival, on the other hand, may be greater following the removal of a branch as compared with a tissue injury since the exposed branch base was rapidly sealed ( > 74 days, Table 3.4). Consequently, the potential for invasion by bioeroders or settlement by superior competitors would be greater for tissue injuries since the injured area is exposed and vulnerable for a longer period of time.

*Porites australiensis* regenerated its scraping injuries more quickly than its tissue injury (Figure 3.3). Similar results were obtained in the Caribbean by Bak and Steward-Van Es (1980) for *Porites astreoides*. Bak and Steward-Van Es (1980) suggested that the repair of the tissue and skeleton injury was accelerated by damage to the skeleton, and that the rapid recovery observed was assisted by regeneration of the partially injured polyps within the injury site. I propose that the difference in regeneration rates between scraping and tissue injuries may also be related to mucus production. In scraping injuries, mucus production by the partially injured polyps may inhibit algal settlement onto the injury site, allowing regeneration to proceed without interference from competition. In contrast, it is not possible for mucus to be produced from sites where the tissue has been removed since the polyps have been killed. Consequently, the site of a tissue injury is colonised by algae and some of the resources available for regeneration will be diverted towards competition, resulting in slower regeneration rates.

Contrary to several previous studies, (e.g. Palumbi and Jackson 1982; Wahle 1983; Meesters and Bak 1995), the location of an injury within a colony did not affect regeneration for six out of the seven species investigated (LSD, Figure 3.2). Only *Porites mayeri* showed a significant difference, with the central injury regenerating 25% of its area within 74 days while no recovery was recorded for the edge injury over the same time (Figure 3.2). Variations in the regeneration of lesions located at different positions within a colony have been related to reduced light levels, polyp density, senescence, and substratum interactions (Jackson 1979; Palumbi and Jackson 1982; Darke 1991; Meesters *et al*. 1992; Meesters and Bak 1995). Meesters and colleagues (1992) demonstrated that the regeneration of artificial lesions on *Acropora palmata* and *Montastrea annularis* colonies was slower in high sediment areas and suggested that this pattern was partially due to a decline in irradiance. *Diploria strigosa*, *Siderastrea siderea* and *P. astreoides*, on the other hand, did not show a difference in the recovery of lesions in high and low sediment sites, nor was a there a difference in the rate of repair of lesions located on the top and side of *P. astreoides* colonies, even though light was reduced by 70% at the sides. Many corals survive in low light levels by photoadapting (Falkowski *et al*. 1990). This phenomena may explain why six out of the seven species in this study did not show a difference in the regeneration of lesions at the edge and center of their colonies. In contrast, *P. mayeri* may have a low capacity to photoadapt. Alternately, the difference in regeneration between these two positions in *P. mayeri* may be related to differences in polyp density. Darke (1991) demonstrated that there was a 10% decline in the density of polyps from the summit to the base of *Porites* colonies, and suggested that higher polyp density reflected relatively higher rates of tissue growth to skeletal growth. Consequently, in *P. mayeri*, lower polyp densities at the base of the colony may hinder regeneration in that area. In contrast to *P. mayeri*, regeneration of edge and central injuries was not significantly different for *Porites lichen* (Figure 3.2, Table 3.3). These two species may differ in this respect because *P. mayeri* buds extra-tentacularly while *P. lichen* buds intra-tentacularly (Veron and Pichon 1982). Studies on patterns of polyp density in massive corals that bud intra-tentactucarly have yet to be done. Senescence and substratum interactions have also been proposed to explain variations in the regeneration of injuries at different locations within the colony (Jackson 1979; Meesters and Bak 1995). However, these processes are unlikely explanations here because substratum interactions would not only affect *P. mayeri* but the other massive species as well, and senescence is most likely to occur in colonial species such as *Steginoporella* sp. and *Acropora palmata* that show a proximal to distal gradient in regeneration and growth (Palumbi and Jackson, 1982; Meesters and Bak, 1995).

In conclusion, experimental studies showed that branching species had more regrowth potential than massive and semi-massive species supporting the hypothesis by Jackson (1979) that morphology plays a role in the pattern of investment in regeneration and defence. Longevity, reproductive output, growth rate, and other life-history processes including regeneration are influenced by colony morphology. Consequently, the morphological strategy of an organism has evolved over time in response to a large number of biotic and abiotic processes including partial mortality.

Jackson's hypothesis (1979) predicted that species with a high commitment to their place of settlement would invest more in maintenance and defence. The results of this study support this hypothesis since the ranking of regenerative ability of the different morphologies was arborescent > bushy > tabular > massive > submassive. Poor regenerative ability in corals may lead to reduced colony fitness since regeneration potentially diverts resources away from growth and reproduction, reduces survival, reproductive output, feeding and photosynthetic capacity, and destroys stored reserves (e.g. Bak *et al*. 1977; Wahle 1983; Rinkevich and Loya 1989).

# CHAPTER 4: THE RESPONSE OF *ACROPORA HYACINTHUS* AND *MONTIPORA TUBERCULOSA* TO SCRAPING INJURIES, TISSUE MORTALITYAND BREAKAGE.

## 4.1. ABSTRACT

Three types of injuries commonly generated on corals by biotic and abiotic agents are tissue mortality, scraping injuries and breakage of peripheral sections of the colony. To determine if recovery was affected by injury type, colonies of *Acropora hyacinthus* and *Montipora tuberculosa* were inflicted with tissue, scraping and breakage injuries and their recovery monitored over 24 days. The influence of the tissue available for regeneration (i.e. the zone of tissue from which regeneration can occur) and the amount of settlement of algae on recovery was also measured for the different injury types and species. Overall, the amount of regeneration was up to four times greater for scraping injuries than tissue mortality and breakage. Furthermore, the amount of regeneration of the breakage injuries was approximately two - times greater for *M. tuberculosa* than for *A. hyacinthus* colonies. The regeneration rate of all injury types declined over time for both species. Recovery of injuries was influenced by the amount of algae that colonised the lesion and the zone of tissue available for regeneration. In general, the amount of regeneration of injuries was lower for injuries with a small zone of basal tissue available for regeneration, and the amount of regeneration was negatively correlated with the amount of algal settlement. The results of this study are important for understanding the role of partial mortality in the demography of scleractinian corals. Injuries impact on the population dynamics of colonies because regenerative processes require resources usually available for other processes (e.g. growth and reproduction), and slow recovery rates potentially increase the chance of whole colony mortality.

## 4.2. INTRODUCTION

Corals provide the basic framework of coral reefs, as well as food and habitats for many associated fauna and flora (e.g. Birkeland 1997). Coral reefs may flourish when disturbances are intermediate in intensity and frequency, and decline if disturbances occur more often and/or with greater magnitude, because coral communities are not given adequate time to recover between impacts (e.g. Connell 1978, 1997; Hatcher *et al* 1989; Chou *et al*. 1994; Hughes 1994). The latter scenario has become increasingly common for many reefs under human exploitation (Colloquium and forum on global aspects of coral reefs, Miami 1993). Unfortunately, while the impact of humans on reefs has been well documented, the underlying mechanisms of effect and their consequences are less understood (Hatcher *et al*. 1989). The general aim of this chapter is to investigate some of the underlying mechanisms affecting injury and regeneration in scleractinian corals.

Injury in scleractinian corals originates from natural causes (e.g. predation, competition, storm and cyclone damage), and human activities (e.g. anchoring, diving, mining and pollution) and results in the loss of tissue and/or tissue and skeleton from the colony (e.g. reviews by Connell 1973; Brown and Howard 1985; Connell 1996; Brown 1997). The response of corals to damage is complex and related to both the characteristics of the injury and the life-histories of different species (e.g. Bak *et al*. 1977; Bak and Steward-Van Es 1980; Meesters *et al*. 1992; Meesters and Bak 1993; Meesters and Bak 1995; Hall 1997; Meesters *et al*. 1997b; Oren 1997).

The regeneration of injuries is influenced by their position on the colony, and their shape, size and type. The position of an injury on a colony can influence recovery as demonstrated in bryozoans (Palumbi and Jackson 1982), the gorgonian *Plexaura homomalla* (Wahle 1983), and some scleractinian corals such as *Acropora palmata* (Meesters and Bak 1995) and *Porites mayeri* (Hall 1997), but not others (e.g. *Porites astreoides*, Meesters *et al*. 1992; *Acropora robusta*, *A. hyacinthus*, *A. palifera*, *Pocillopora damicornis* and *Porites lichen*, Hall 1997). Variations in the recovery of injuries located at different positions within a colony have been associated with the senescence of polyps (and zooids) along a proximal to distal gradient (e.g. Palumbi and

Jackson 1982; Meesters and Bak 1995), reduced light levels as a result of shading (Meesters *et al*. 1992), different polyp densities at the base and top of massive colonies (Darke 1991, Hall 1997) and substratum interactions (Jackson 1979).

Recovery of injuries is size-specific with regeneration being higher for small (1 $cm^2$) injuries as compared with larger (5 $cm^2$) injuries (Bak *et al*. 1977; Bak and Steward-Van Es 1980). However, the shape of an injury is as important as its size since recovery is influenced by both its area and the amount of healthy tissue bordering its perimeter (Meesters *et al*. 1997b; Oren *et al*. 1997). Meesters *et al*. (1997b) have suggested that resources for recovery come from those polyps bordering the lesion, while Oren *et al*. (1997) have proposed that these resources may be translocated from both neighbouring and more distant polyps, depending on the length of the injury. These conclusions have been drawn from studies that have examined only one specific injury type, where regeneration occurs at the edge of the injury (Meesters *et al*. (1997b) removed the tissue and skeleton from a colony while Oren *et al*. (1997) removed only the tissue, leaving the skeleton intact). The amount of damage incurred, and the area of tissue from which regeneration can occur, however, are dependent on the type of injury.

Coral injuries can be divided into four major categories: partial tissue loss, tissue mortality, superficial tissue and skeleton loss (e.g. scraping injuries), and substantial tissue and skeleton loss (e.g. breakage), as discussed in detail in the introduction of chapter 3. The recovery of these different types of injuries vary enormously. Bak *et al*. (1997) showed that the recovery of tissue injuries was faster than scraping injuries in *Agaricia agaricites* while the opposite pattern was true for *Montastrea annularis*. Similarly, recovery of scraping injuries was faster than the recovery of tissue injuries for *Porites australiensis* and *A. cytherea* (Hall 1997). In a comparison of breakage versus tissue injuries, the regrowth of a branch was 10 times slower than the recovery of a tissue denuded branch (Hall 1997). Recovery of different injury types appears linked to the amount of tissue and skeleton that must be replaced, as well as the ability to out compete organisms that have settled onto the injured area.

The morphology of a colony often dictates its susceptibility to different types of injury, and influences the subsequent size of any injury. For example, branching species are more prone to storm and diver damage than massive species (Woodley *et al*. 1981; Hughes 1989; Glynn 1990; Chadwick-Furman 1995), and massive and encrusting species are more vulnerable to overgrowth by algae (Hughes 1989). For branching species, differences in branch thickness can result in different sized lesion if they are broken off. This has been demonstrated for *Acropora gemmifera* and *A. millepora* where the resulting injury to the colony through breakage was 3 times larger for *A. gemmifera* (Hall 1997).

If the injured area does not regenerate within a couple of days, algae and other organisms will settle onto the injured area (e.g. Bak *et al*. 1977). These organisms may then compete with the coral for food and space. As a consequence recovery may be slowed since resources available for regeneration may be reallocated to competition. Additionally, the presence of the algae may act as an obstruction to the recovery process since the formation of chimneys around algal filaments are a common phenomena on corals. In the long-term, colony survivorship may be affected if (1) the colonising organism is a superior competitor, (2) the algal patch is used as an entry site for boring organisms that disrupt the integrity of the colony, or (3) the algae provides an infection site for diseases (Bak *et al*. 1977; Palumbi and Jackson 1982; Wahle 1983; Hughes and Jackson 1985; Babcock 1991).

The ability of many corals to recover from injuries has been well documented (e.g. Stephenson and Stephenson 1933; Bak *et al* 1977; Bak and Steward Van-Es 1980; Bak 1983; Rinkevich and Loya 1989; Meesters *et al*. 1992; Meesters *et al*. 1994; Hall 1997). Recovery of corals from injury is important because injury diminishes colony fitness by (1) reducing survival, reproductive output, feeding and photosynthetic capacity, (2) destroying stored reserves and (3) requiring the use of resources for regeneration, thus potentially diverting resources away from growth and reproduction (e.g. Bak *et al*. 1977; Wahle 1983; Hughes and Jackson 1985; Rinkevich and Loya 1989; Meesters *et al*. 1994). While the roles of the position, size and shape of injuries in recovery processes have been investigated in previous studies, the influence of injury type on recovery has largely been ignored (but see Bak *et al*. 1977; Bak and Steward-Van Es 1980). The aim of this chapter is to investigate how recovery of damage is influenced by injury type for *Acropora hyacinthus* and *Montipora tuberculosa*. Specifically I will examine if (1) the amount of colonisation of the injured site by algae, (2) the dimensions of the tissue from which regeneration can occur, and (3) regeneration rates, vary among colonies with tissue mortality, scraping or breakage injuries for the two species.

## 4.3. MATERIALS AND METHODS

To investigate the response of corals to scraping injuries, tissue mortality and breakage, I injured colonies of two species and monitored the subsequent recovery process. The species were *Acropora hyacinthus* and *Montipora tuberculosa*, chosen because of their susceptibility to the injury types under investigation, their contrasting morphologies, and abundance on the reef-crest at Mermaid Cove, Lizard Island, Australia, where the study was conducted (Figure 2.1). *Acropora hyacinthus* is a fast-growing, relatively short-lived, tabular coral consisting of a wide flat table of fused branches from which fine upwardly projecting branchlets protrude. *Montipora tuberculosa* is also a relatively short-lived, fast-growing coral but has a plate-like, foliaceous morphology.

Different techniques were used to generate the three different types of injuries on colonies. For tissue injuries, all tissue was removed from a section of the colony by blowing it away from the skeleton with compressed air. This air was discharged from the nozzle of an air-gun at a constant pressure of 60 psi. A template cut from water-proof paper was used to protect surrounding tissue from damage and to standardise the injury size. Scraping injuries were inflicted by lightly dragging a fine chisel over the surface of colonies to remove a superficial layer of tissue and skeleton (1 - 2 mm), including structures protruding above the coenosteum. Breakage was simulated by carefully chiselling away a small section of the colony. The size, shape and position of the injury on the colony (Figure 4.1a), and colony size, were standardised across all three injury regimes since these characteristics can affect recovery (Loya 1976, Bak *et al* 1977, Meesters and Bak 1995; Meesters *et al*. 1997b; Oren *et al*. 1997). All injuries were relatively small, having a mean projected area of 3.3 ± SE 0.13 $cm^2$ . Injuries were inflicted *in-situ* on the edge of colonies and only large colonies (diameter >75cm) were used. A total of 60 colonies were injured in this experiment (10 per injury type and species). The positions of all colonies were marked with horticultural tags attached to the substratum close to the colony to ensure relocation at subsequent censuses.

### 4.3.1. Amount of regeneration

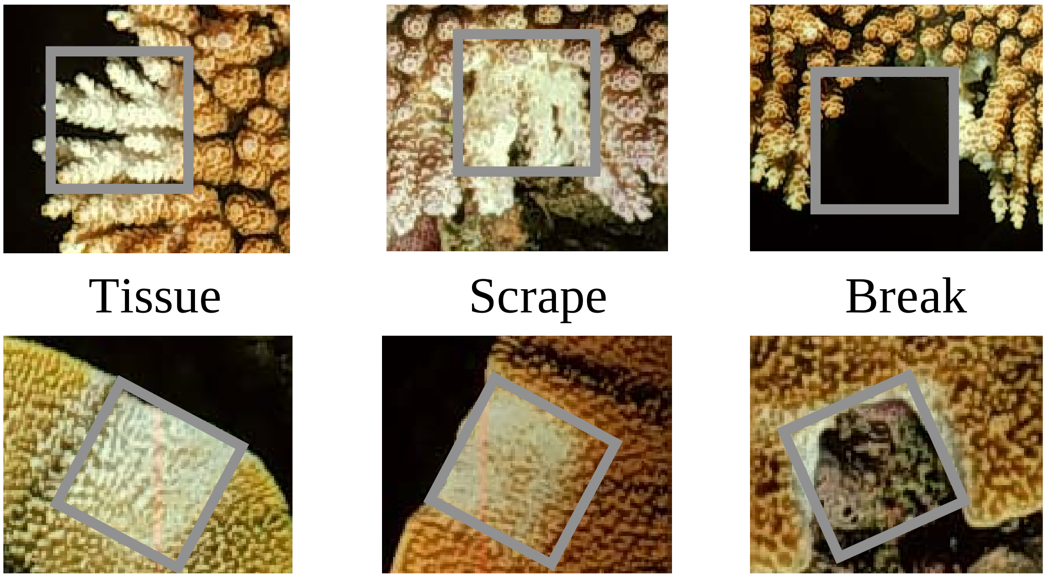
To determine the amount of regeneration of the injuries over time, they were monitored photographically with a Nikonos V underwater camera. Close-up photographs, using a 35mm close-up attachment, were taken at the time of the injury, and 12 (census 1) and 24 (census 2) days after the injuries were inflicted. A ruler attached to the close-up frame was used as a size reference. The amount of regeneration that had taken place over time was quantified from the photographs by projecting the image at its actual size onto a paper screen and tracing the damaged and recovered areas. Both areas were then digitised on a Hi-Pad-Plus Digitiser used in association with the computer package Sigma-Scan. The amount of regeneration that occurred over time has been presented graphically as a percentage of the original injury size for each injury type.

### 4.3.2. Amount of algal settlement

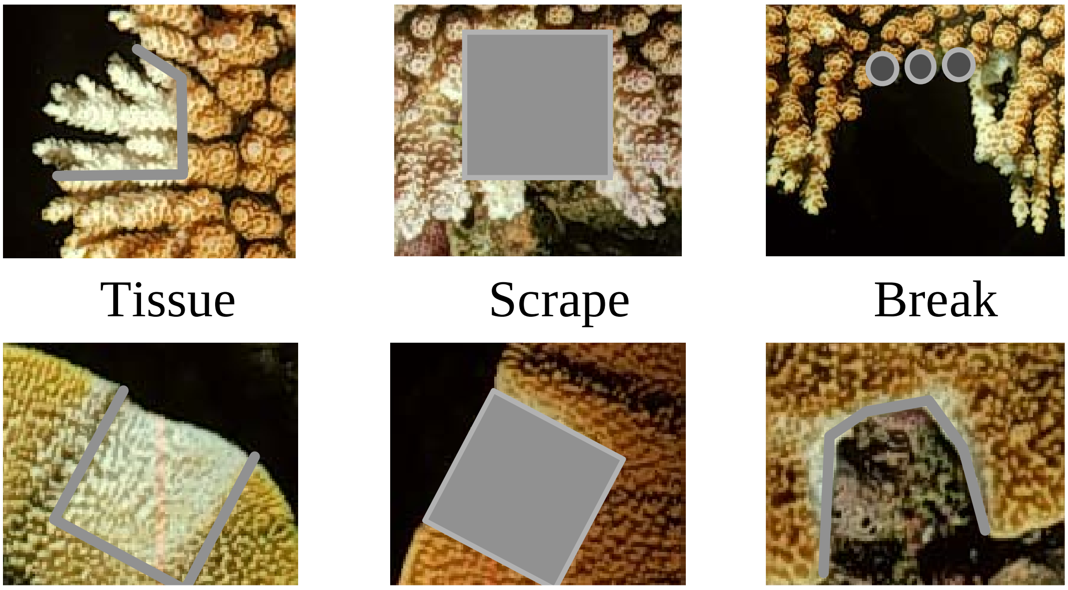
To examine the effect of colonisers on the recovery of the three different injury types, close-up photographs of the amount of algae (the initial coloniser of damaged areas) settling onto the injured areas were taken 12 and 24 days after injury. The amount of algae present on injuries over time was then determined by tracing and then digitising the areas colonised by algae (as described previously for regeneration). The amount of algae present has been presented graphically as a percentage of the original injury area for tissue and scraping injury, and as a percentage of the viable area of regeneration for breakage injuries since viable area of regeneration represents the area of damage on the colony and thus the potential site for colonisation.

Figure 4.1. Photographs of the different injury types for *A. hyacinthus* (top row) and *M. tuberculosa* (bottom row). (a) The size of the injury is outlined in grey, and (b) zone of tissue available for regeneration is represented by the area shaded in grey. For scale, the side of the box = 1.8 cm.

(a) Injury Size



(b) Zone of tissue available for regeneration.



### 4.3.3. Zone of tissue available for regeneration

The modes of regeneration for the three different injury types were examined by observing the patterns of regeneration from consecutive photographs of injuries. The influence of the bordering or contact areas of tissue from which regeneration can take place (zone of tissue available for regeneration) on the recovery of different types of injuries was then quantified by measuring the dimensions of the tissue involved in regeneration with a set of vernier callipers. For tissue injuries, regeneration occurs from the margins of the injury so that the perimeter of the injury (minus the outer edge) was measured (Figure 4.1). For breakage injuries, regeneration was activated from the area of tissue which remains after the break. To obtain the dimensions of the tissue available for regeneration for breakage injuries, the perimeter and depth of the bordering tissue was measured for *M. tuberculosa,* and the diameter of the base of branchlets was measured for *A. hyacinthus*, because the shape of this area varies with species (Figure 4.1b). For scraping injuries the dimensions of the injury were measured since regeneration occurs from the underlying partially damaged polyps. Geometric formulas for perimeters, rectangles and circles were used to calculate the dimensions of the tissue from which regeneration can take place.

### 4.3.4. Analysis of data

Differences between species and injury types in the amount of regeneration and algae present over 12 and 24 days were tested using a repeated measures ANOVA. This analysis tests for between subject effects (i.e. differences between species and injury types for the two variables) and within subject effects (differences in the variables between census one and two for species and injury types). Variables were log transformed to meet the assumptions of the analysis. For each injury type, differences in the amount of tissue available for regeneration between species were tested with a t-test. (A separate analysis was conducted for each injury types since the mode of regeneration differed among injury types). This data set was log transformed to meet the assumptions of the t-test. If the differences in amount of tissue available for regeneration between species were significant, a repeated analysis of covariance

(ANCOVA) was subsequently carried out on the amount of regeneration over 12 and 24 days, with amount of tissue for regeneration as the co-variate. This analysis factors out the effect of this variable on regeneration. A Pearson's Correlation Analysis was used to determine if there was any association between the amount of regeneration of injuries and the amount of algal settlement on injuries. The data were log transformed to meet the assumptions of the analysis.

## 4.4. RESULTS

Striking differences were detected between species and injury type in the regeneration of injuries (Table 4.1, Figure 4.2). This pattern was influenced by the amount of tissue contributing to recovery and the level of colonisation of algae onto lesions. (Table 4.2, 4.3 and 4.4; Figures 4.3 and 4.4). Furthermore, the pattern of recovery and colonisation changed over the two census times, especially among injury types (Table 4.1 and 4.2; Figure 4.2 and 4.3). These results have been discussed in detail below.

### 4.4.1. Amount of regeneration

Rates of recovery varied markedly between injury types, and also between species for breakage injuries (Between Subject Effects, Table 4.1, Figure 4.2) over 24 days. In contrast, there was no difference in recovery for scraping and tissue injures *for A. hyacinthus* and *M. tuberculosa* (Figure 4.2). The amount of regeneration of scraping injuries was high during the course of this study (> 75% of the original injury, Figure 4.2). This was in marked contrast to the recovery of tissue and breakage injuries where less than 40% of the injured area was regenerated ( Figure 4.2). However, although the regrowth of lost polyps was slow for breakage injuries, most of the section of the colony that was damaged as a result of breakage was rapidly sealed over within 12 days (also see chapter 3, section 3.4.2). For breakage injuries, the amount of recovery was approximately twice as high for *M. tuberculosa* as *A. hyacinthus* (Figure 4.2). The overall amount of recovery of all injuries types was much greater in the first 12 days as compared to the second 12 days (Within Subject Effects for census, Table 4.1, Figure 4.2). This pattern of regeneration among censuses was consistent for species but not injury types.  For injury types, the amount of recovery between censuses was approximately 1.2 - 1.5 times greater for tissue injuries and breakage as compared to scraping injuries (Figure 4.2).

### 4.4.2. Amount of algal settlement

Large differences were detected in the amount of algae that colonised the injured area in 24 days (Between subject effects, Table 4.2, Figure 4.3).  Algal cover on tissue injuries (bare skeleton) for both species was greater than 70% after 12 and 24 days, and less than 1% for breakage injuries after 12 and 24 days (Table 4.2, Figure 4.3).

Figure 4.2.  Mean regeneration of injuries (expressed as a percentage of the original injury size) for injury type and species after 12 and 24 days.  (Error bars represent one SE).

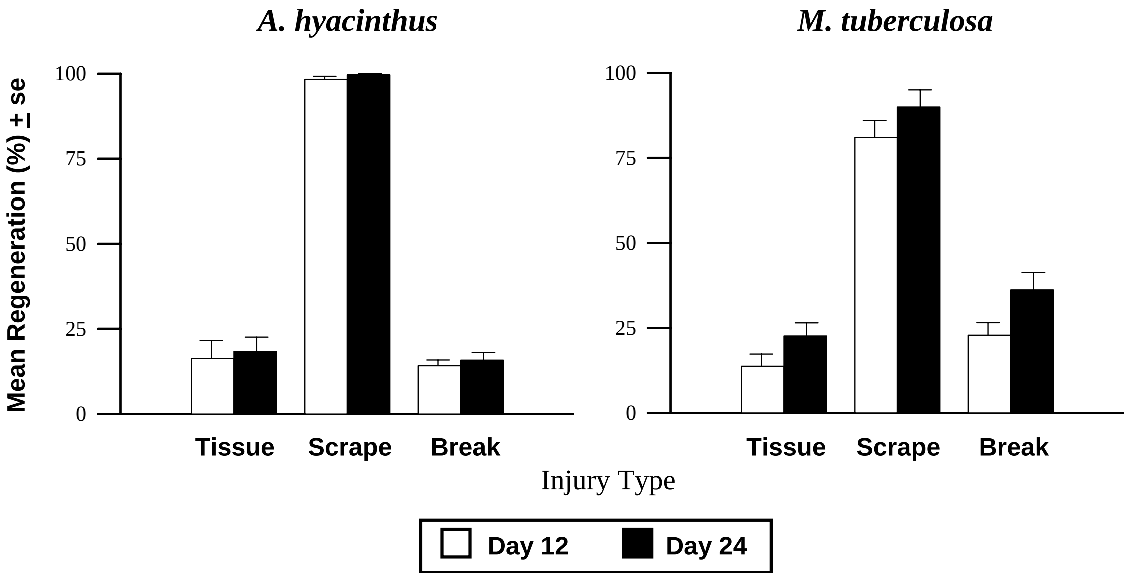


Table 4.1. Summary of the repeated measures ANOVA testing for differences in regeneration between species and injury types (Between Subject Effects) and how these patterns change between censuses (Within Subject Effects).

| Source of Variation | df | Type III SS | Mean Square | F-ratio | Probability |
|---|---|---|---|---|---|
| BETWEEN SUBJECT EFFECTS | | | | | |
| Species | 1 | 0.041 | 0.041 | 2.58 | 0.115 |
| Injury Type | 2 | 2.177 | 1.088 | 68.86 | 0.000 |
| Species * Injury Type | 2 | 0.132 | 0.066 | 4.19 | 0.021 |
| Error | 47 | 0.743 | 0.016 | | |
| WITHIN SUBJECT EFFECTS | | | | | |
| Census | 1 | 0.091 | 0.091 | 41.17 | 0.000 |
| Census*Species | 1 | 0.005 | 0.005 | 2.31 | 0.104 |
| Census*Injury Type | 2 | 0.032 | 0.016 | 7.31 | 0.002 |
| Census*Sp. * Injury | 2 | 0.012 | 0.006 | 2.69 | 0.079 |
| Error | 47 | 0.104 | 0.002 | | |

For colonies with scraping injuries, algal cover was low for *A. hyacinthus* and intermediate for *M. tuberculosa* (< 2% and <20% respectively) after 12 and 24 days. The amount of algae present at census 2 was much less than the amount present after census 1 (Census, Within subject effects, Table 4.2). There was a consistent pattern in change of algal cover over time between species but not injury type (Within subject effects, Table 4.2). Algal cover declined by 8 - 10% for tissue injuries, 50% for scraping injuries and more than 50% for breakage between census 1 and census 2 (Figure 4.3). There was a negative relationship between the amount of regeneration and amount of algal cover (Pearson’s Correlation Coefficient = -0.441 (P = 0.001) and -0.348 (P = 0.011) for day 12 and 24 respectively, Figure 4.4).

Figure 4.3. Mean algal settlement expressed as a percentage of the original injury size for injury types and species after 12 and 24 days. (Error bars represent one SE).

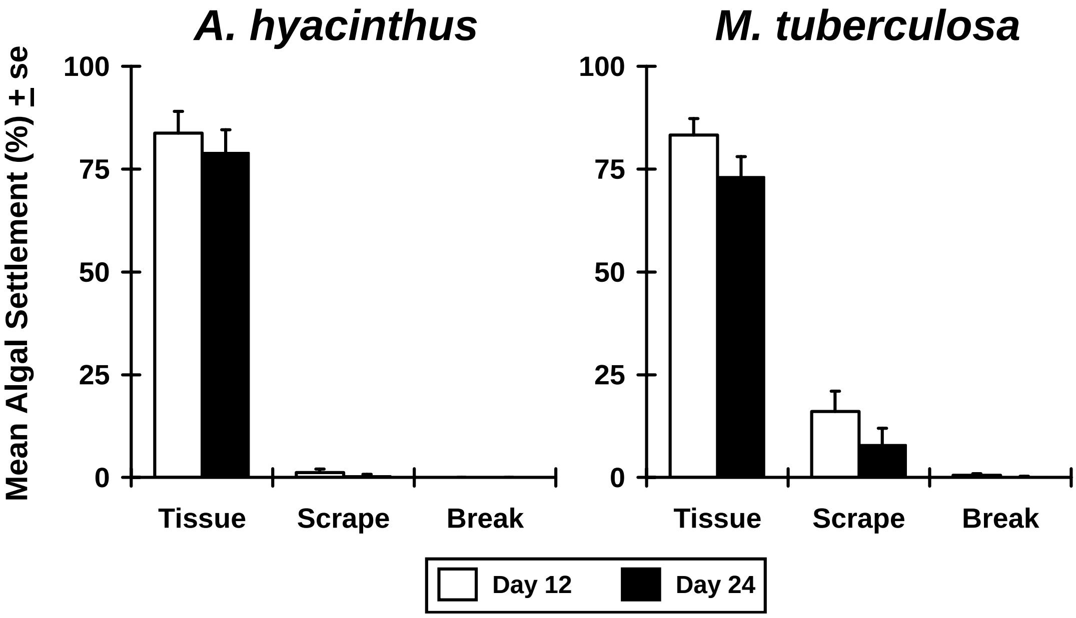


Figure 4.4 A scatter plot of the relationship between the amount of regeneration ($cm^2$) and the amount of algal settlement ($cm^2$) over 12 and 24 days.

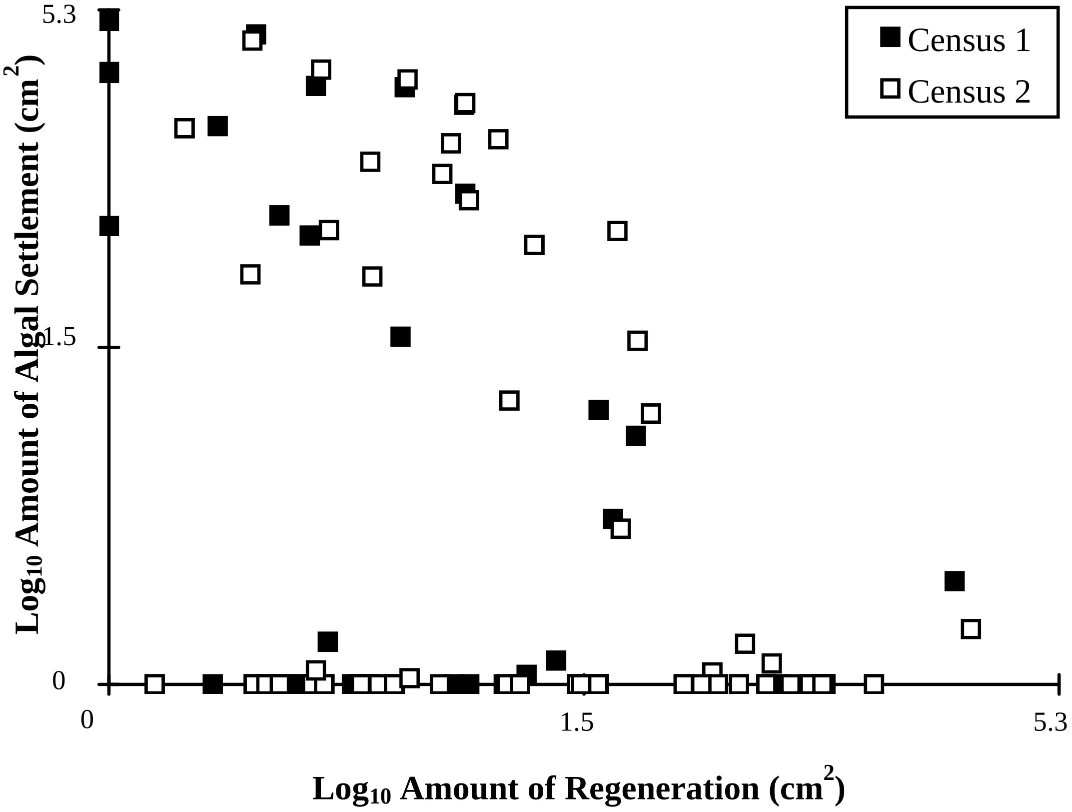


Table 4.2 A summary of the repeated measures ANOVA testing for differences in the amount of algal colonisation between species and injury types (Between Subject Effects) and how these patterns change after 12 and 24 days (Within Subject Effects).

| Source of Variation | df | Type III SS | Mean Square | F-ratio | Probability |
|---|---|---|---|---|---|
| BETWEEN SUBJECT EFFECTS | | | | | |
| Species | 1 | 0.026 | 0.026 | 2.60 | 0.114 |
| Injury Type | 2 | 7.724 | 3.862 | 392.59 | 0.000 |
| Species * Injury Type | 2 | 0.057 | 0.028 | 2.87 | 0.066 |
| Error | 47 | 0.462 | 0.010 | | |
| WITHIN SUBJECT EFFECTS | | | | | |
| Census | 1 | 0.027 | 0.027 | 13.75 | 0.001 |
| Census*Species | 1 | 0.003 | 0.003 | 1.56 | 0.218 |
| Census*Injury Type | 2 | 0.013 | 0.006 | 3.29 | 0.046 |
| Census*Sp. * Injury | 2 | 0.005 | 0.003 | 1.32 | 0.277 |
| Error | 47 | 0.094 | 0.002 | | |

### 4.4.3. Zone of tissue available for regeneration

The amount of tissue available for regeneration reflects differences in the modes of regeneration (Table 4.3). For scraping injuries, the zone of tissue area available for regeneration was equal to the original size of the injury since the partially damaged polyps within the injury contributed to the regeneration process (Table 4.3, Figure 4.1). In contrast to scraping injuries, regeneration of tissue and breakage injuries only occurred at the margins of the lesions since polyps within the lesions were either dead or had been removed (Figure 4.1). For tissue injuries, the tissue at the perimeter of the injury formed a smooth layer of tissue and skeleton which gradually grows inward, thus enveloping the injured site. Polyps were then produced along the margin of the band that was closest to the uninjured tissue. For breakage injuries, recovery was facilitated by the extension of the skeleton via the growth of new polyps.

Table 4.3. The zone of tissue available for regeneration ± one SE for each injury type and species, and the results of the t-test analysing differences between species in the amount of tissue available for regeneration for each injury type.

| Injury Type | Zone of Regeneration | Species |  | T-test |
|---|---|---|---|---|
|  |  | *A. hyacinthus* | *M. tuberculosa* | Statistic (df), P |
| Tissue | Perimeter of injury (cm) | 6.00 ± 0.21 | 6.19 ± 0.54 | 0.72 (18), 0.481 |
| Scrape | Area of injury ($cm^2$) | 3.23 ± 0.10 | 1.00 ± 0.00 | 0.47 (17), 0.642 |
| Break | Area at base of break ($cm^2$) | 0.45 ± 0.08 | 1.66 ± 0.26 | 16.13 (17), 0.000 |

The amount of tissue available for regeneration was consistent among species for tissue mortality and scraping injuries but varied markedly between species for breakage injuries (Table 4.3). For breakage injuries, the tissue area contributing to regeneration was approximately three times higher for *M. tuberculosa* than *A. hyacinthus* (Table 4.3). This difference occurred because *M. tuberculosa* has a plate morphology and the margin for regeneration is a continuous U-shape while *A. hyacinthus* is made up of discrete branchlets, and regeneration only occurs from the base of these individual branchlets (Figure 4.1). This interspecific difference in amount of tissue available for regeneration clearly had an effect on the amount of regeneration over 24 days since the significant difference in regeneration is negligible once the effect of amount of tissue available for regeneration has been adjusted for by ANCOVA (Between subject effects, species, Table 4.4). The adjusted amount of regeneration for breakage injuries was 1.95 ± 0.3 $cm^2$ and 1.24 ± 0.2 $cm^2$ for *A. hyacinthus* and *M. tuberculosa*, respectively. Thus, this result suggests that the recovery of breakage injuries is similar among species providing the area available for regeneration is the same.

Table 4.4. A summary of the repeated measures analysis of covariance testing for differences in regeneration between *A. hyacinthus* and *M. tuberculosa* for breakage injuries (Between Subject Effects) and how these patterns change between censuses (Within Subject Effects) after the effect of the amount of tissue available for regeneration (ATR) has been adjusted for.

| Source of Variation | df | Type III SS | Mean Square | F-ratio | Probability |
|---|---|---|---|---|---|
| BETWEEN SUBJECT EFFECTS | | | | | |
| Species | 1 | 0.016 | 0.016 | 1.67 | 0.214 |
| ATR | 1 | 0.049 | 0.049 | 5.18 | 0.037 |
| Error | 16 | 0.152 | 0.010 | | |
| WITHIN SUBJECT EFFECTS | | | | | |
| Census | 1 | 0.016 | 0.016 | 18.34 | 0.001 |
| Census*Species | 1 | 0.001 | 0.001 | 0.53 | 0.479 |
| Census*ATR | 1 | 0.000 | 0.000 | 0.11 | 0.743 |
| Error | 16 | 0.014 | 0.0019 | | |

## 4.5. DISCUSSION

The capacity to recover quickly from damage differed among species and injury types and was influenced by the amount of tissue available for regeneration and the proportion of the injury that was settled by algae. Interspecific differences in the amount of regeneration of different injury types have consequences for the dynamics of coral populations under varying injury regimes since injury can affect fitness.

The amount of tissue available for regeneration is dependent on the type and level of damage inflicted and interspecific variations in colony morphology. The amount of regeneration of lesions differed among injury types for both species and was 3 - 4 times higher for scraping injuries as compared with tissue mortality and breakage (Figure 4.2). The amount of tissue available for regeneration was influenced by injury type since different injury types cause varying amounts of damage to colonies, and their subsequent modes of regeneration vary. The damage caused to colonies by scraping was superficial so that the underlying polyps were only partially damaged and contributed to the regeneration process. In contrast, the recovery of tissue and breakage injuries only occurred at the margins of the injury via the growth of new polyps. This growth was initiated by the surrounding healthy polyps although it is possible that some of the resources necessary for regenerative processes may have come from polyps further away through translocation (see Oren *et al*. 1997, Chapter 5). The differences detected in recovery between injury types suggests that it is energetically more costly to produce new polyps than to repair partially damaged ones.

The amount of regeneration of breakage injuries also differed among species and was three - times larger for *M. tuberculosa* than for *A. hyacinthus* (Figure 4.2). This pattern was solely due to differences in the amount of tissue available for regeneration since (1) differences in the amount of regeneration disappear when the zone of tissue available for regeneration is accounted for by ANCOVA (Table 4.4), and (2) the amount of algae settling on this type of injury was negligible (Figure 4.3). This result highlights how the amount of damage incurred, and the subsequent amount of tissue available for regeneration is dependent on morphology. *Montipora tuberculosa* has a plate morphology while *A. hyacinthus* has a tabular morphology so that the removal of a square section from the outer edge of colonies of these two species resulted in two completely different areas from which regeneration could occur. For *M. tuberculosa*, regeneration of new tissue and skeleton occurred from the U-shaped edge created by breakage while for *A. hyacinthus* colonies, regeneration could only take place from the base of branchlets that had been removed (Figure 4.1, Chapter 3). As a consequence, for breakage events, the symmetry of the plate coral is restored a lot faster than that of the tabular coral. Maintenance of colony integrity (symmetry) in non-encrusting corals is important for such processes as defence, feeding, and mechanical stability (Jackson 1979; Hughes and Jackson 1985).

The settlement of algae onto the injured area appeared to influence recovery because there was a negative correlation between the amount of regeneration and algal cover over 12 and 24 days (Figure 4.4). The overall pattern of a reduction in regeneration with an increase in algal settlement suggests a trade-off in available resources between regeneration and competition. Differences in the amount of algae that settled onto injury sites may be linked to the production of mucus since mucus appears to act as a deterrent to the colonisation of damaged areas on corals by other organisms (Benson *et al*. 1978; Rutzler *et al*. 1983). Settlement by algae was relatively low for scraping and breakage injuries since tissue which had been partially damaged retained its ability to produce mucus. Conversely, the removal of mucus producing tissue resulted in a high settlement of algae as observed for the tissue injuries. Oren *et al*. (1997) proposed that the tentacles from surrounding healthy polyps may offer protection from the settlement of algae and sediment for small injuries with high perimeter to surface area ratios. Consequently, tentacles from healthy polyps may have protected breakage injuries from algal settlement due to a high perimeter to surface area ratio of the injury. However, this ratio would have been too low for this process to be effective for scraping injuries.

Recovery of injuries declines over time (Bak 1983; Meesters *et al*. 1992; Meesters *et al*. 1994, Figure 4.2). This decline in regeneration may result from the exhaustion of a finite supply of resources available for regeneration (Meesters *et al*. 1994, Rinkevich 1996), and/or an increasing loss over time of these available resources to competitive interactions with algae. Algae may further impede the process of regeneration over time because it steadily increases in biomass and acts as a trap for sediment. Consequently, the presence of a solid mat of algae and sediment may present an obstruction difficult for the coral to overgrow.

In general, the results of this study suggest that tissue injuries are more detrimental to the long-term survival of colonies than scraping and breakage injuries because their recovery was slower. Recovery of tissue injuries was hindered by both their greater susceptibility to colonisation by algae and a smaller zone of tissue available for regeneration. As a result, the injured area of the colony is exposed to colonisers for longer periods of time, thus increasing the probability of settlement by a superior competitor, infection by pathogens, or invasion by bioeroders (e.g. Bak *et al*. 1977). All these factors increase the risk of total mortality in colonies (Connell 1973). Breakage injuries may also impact on the population dynamics of some coral species since slow recovery rates will impede on the speed at which the symmetry of a colony is restored. Colony symmetry is important for defensive and feeding processes, and mechanical stability (Jackson 1979; Hughes and Jackson 1985). However, while breakage may be costly to individual colonies within a population, its effects on the genet population may be favourable since breakage promotes asexual reproduction through fragmentation. In contrast to tissue and breakage injuries, scraping injuries seem the have the least impact on the population dynamics of corals. The advantage of faster regeneration rates is that the functional use of polyps (photosynthesis, defence, reproduction, feeding and storage) is restored more quickly (Jackson 1979; Sebens 1987; Hughes and Jackson 1985), and the risk of invasion by competitors, pathogens and bioeroders is minimised (Jackson and Palumbi 1979; Wahle 1983; Hughes and Jackson 1985; Sebens 1987).

In conclusion, the capacity to recover quickly from damage differed among species and injury types and was influenced by the amount of tissue available for regeneration and the proportion of the injury that was settled by algae. Generally, the amount of regeneration of injuries was lower for injuries with a small amount of tissue available for regeneration and/or high levels of algal settlement. The findings of this study are important for understanding the population dynamics of corals since slow recovery rates increase the chance of whole colony mortality and regeneration processes deplete resources normally available for growth and reproduction.

# CHAPTER 5: TO SURVIVE OR REPRODUCE: THE RESPONSE OF SCLERACTINIAN CORALS TO DAMAGE

## 5.1. ABSTRACT

The response of modular organisms such as scleractinian corals to damage is dependent on the nature and extent of the wide variety of agents that cause injuries. In this study, I investigated the response of colonies of *Acropora hyacinthus*, *A. gemmifera* and *Goniastrea retiformis* to injury by monitoring their recovery over nine months. Two injury regimes, injury size and repetitive injury, were inflicted on colonies to test the hypothesis that the impact of an injury will depend on the size and frequency of damage: the larger or more frequent the injury, the greater the impact. The injury sizes comprised 8%, 16% and 25% of the colony while the repetitive treatments included colonies that were injured twice and three times over nine months. The response variables measured were survival, colony growth, regeneration and reproduction.

There was no effect of injury size or frequency of damage on the survival or growth of colonies over nine months for the three species. Additionally, all species responded to injury with the growth of new polyps to replace those lost as a result of damage. In contrast to colony survival and growth, there was a marked effect of injury on reproduction inferring a trade-off between reproduction and regeneration. Presumably the resources usually available for gamete production were reallocated towards polyp regeneration and defence against fouling organisms.

The effect of injury on reproduction was evident both close to and away from the injury, suggesting both a localised and colony-wide response. At a colony-wide scale, the effect of damage on reproduction varied between the various components of reproduction as well as among species and injury regimes. Colonies of *A. hyacinthus* were more susceptible to recurrent damage while the two other species were less tolerant of larger injuries. These effects were detected as a decrease in polyp fecundity for colonies of *G. retiformis* and in egg volume per polyp (mm$^3$) for colonies of the two *Acropora* species. At a local scale, there was a significant decline in both polyp

fecundity and egg volume per polyp ($mm^3$) for all species in response to injury. This localised decline in reproduction in response to damage was not exacerbated by injury size or frequency with one exception. Polyp fecundity was reduced by 28% for colonies of *A. gemmifera* that had been injured three times. The loss of reproductive tissue resulting from an injury comprising 75 $cm^2$ projected area of a colony was substantial and had the potential to impact on reproductive success since there were fewer gametes available for fertilisation processes. The loss of reproductive output varied among species due to morphological differences. The loss of egg volume ($mm^3$) was an order of magnitude greater for the *Acropora* species because they have branching morphologies and thus more polyps per unit projected area.

The overall response of large colonies of *A. hyacinthus*, *A. gemmifera* and *G. retiformis* to injury suggests that these species are resistant to damage (i.e. colonies are protected from damage by defensive structures, compounds or behaviours, and/or able to regrow lost parts). In this study, the survival of colonies was not affected by injury, at least in the short-term, and the colonies responded to mechanical injury by growing new polyps. As a consequence, future reproduction was preserved at the expense of current reproduction. These findings are in agreement with that predicted by life-history theory: organisms with indeterminate growth will balance resource allocation between somatic longevity and reproduction.

## 5.2. INTRODUCTION

Modular organisms are made up of repeated building blocks such as modules, ramets, zooids and polyps (Harper 1977; Hughes *et al*. 1992). These organisms grow through the iteration of modules and shrink when modules are lost (Harper 1977; Hughes *et al*. 1992). The loss of modules or body parts from plants and animals (injury) as a result of biotic and abiotic activities occurs frequently in nature (e.g. Connell 1973; Wahle 1983; Begon *et al*. 1986; Room *et al*. 1994; Meesters *et al* 1996,1997a). The survival of damaged organisms depends upon their ability to regenerate lost parts, which, in turn, is dependent on the characteristics of injury (e.g. nature, extent, timing), the prevailing biotic and abiotic conditions (i.e. local environment), and the species involved (e.g. Liddle and Kay 1987; Liddle 1991; Meesters *et al*. 1992; Meesters and Bak 1993, 1995; Meesters *et al*. 1997b; Oren *et al*. 1997).

The ability to regenerate lost modules or body parts has evolved independently in many taxa (e.g. arthropods, molluscs, chordates, echinoderms, bryozoans, corals, sponges and plants, see reviews Goss 1969; Harper and White 1974), suggesting that regeneration must offer a fitness advantage in some organisms. Life-history theory predicts that longer-lived species maximise fitness by allocating resources towards growth and maintenance rather than reproduction to enhance survival and future reproductive output (Williams 1966; Bell 1980; Stearns 1992). Conversely, short-lived species will allocate resources towards reproduction at the expense of survival (Williams 1966; Bell 1980; Stearns 1992).

Patterns of resource use within a modular organism may be determined by the degree of physiological integration (e.g. Bazzaz *et al*. 1987; Haukioja 1991; Herms and Mattson 1992). Consequently, the response of modular organisms to damage may be localised or involve the whole organism (e.g. Bazzaz *et al*. 1987; Rinkevich and Loya 1989; Haukioja 1991; Herms and Mattson 1992; Van Veghel and Bak 1994; Oren *et al*. 1997). In corals, small injuries may invoke a localised decline in reproduction (e.g Van Veghel and Bak 1994) and growth (e.g. Bak 1983), while larger injuries (> 10% of the colony) may reduce reproduction and growth on a colony-wide scale in some species (e.g. reproduction, Rinkevich and Loya 1989; Hall 1996; growth, Meesters *et al*. 1994).

The capacity to regenerate injuries in corals is strongly dependent on the characteristics of the injury. Generally, modular organisms have a greater capacity to regenerate smaller injuries than larger ones (e.g. Bak *et al*. 1977; Bak and Steward-Van Es 1980; Palumbi and Jackson 1982), recovery is usually higher for single rather than multiple episodes of injury (e.g. Tardent 1963; Strauss 1991), and recovery is constrained by the timing of injury, for example ontogenetic stage, and seasonal and reproductive cycles preclude some responses at certain times (Maschinski and Whitham 1987; Trumble *et al*. 1993; Room *et al*. 1994).

The recovery of an injury also depends on its position on a colony (e.g. Wahle 1983; Meesters *et al* 1992; Meesters and Bak 1995) and the ratio of healthy to damaged modules since both may influence the amount of resources available for regeneration (e.g. Tardent 1963; Meesters *et al*. 1992; Meesters and Bak 1985; Oren *et al*. 1997; Rinkevich 1996). The length of recovery time is also important since the longer the recovery time the greater the risk of settlement by potential competitors, invasion by boring organisms, and exposure to pathogens (Bak *et al*. 1977; Palumbi and Jackson 1982; Trumble *et al*. 1993; Hall 1997).

To date, the general objective of most experimental research into damage on scleractinian corals has been to focus on the response of colonies to relatively small injuries with differing characteristics (e.g. Bak *et al*. 1977; Bak and Steward-Van Es 1980; Meesters *et al*. 1997b; Oren *et al*. 1997, chapter 3 and 4), or the same type of lesion under different environmental conditions (Meesters *et al*. 1992; Meesters and Bak 1993) for one or several species. Trade-offs between regeneration and other demographic traits within a single species have also been examined (e.g. growth, Loya 1978; Meesters *et al*. 1994; reproduction, Rinkevich and Loya 1989; Van Veghel and Bak 1994; growth, reproduction and survival, Ward 1995). The major aim of this study is to broaden and extend our knowledge of the response of corals to damage in three ways: Firstly, to investigate the effect of damage on regeneration, colony growth, reproduction and survival for three species under two different injury regimes: repetitive injury and injury size. Secondly, to examine colony-wide and localised responses to reproduction following injury to determine if there is an integrated colony response to damage, and to see if this response varies with injury regime. Thirdly, to demonstrate the direct impact of damage on a colony by estimating the loss of reproductive output caused by an injury of a given size (75 $cm^2$ projected area). This study will focus on larger injuries (> 8% of the colony) rather than smaller injuries common to many previous experimental studies (Bak *et al*. 1977; Bak and Steward-Van Es 1980; Bak 1983; Meesters *et al*. 1992; Meesters and Bak 1993; Meesters and Bak 1995; Meesters *et al*. 1997b; Oren *et al*. 1997; Chapters 3 and 4).

## 5.3. MATERIALS AND METHODS

To quantify the effects of size of injury and frequency of damage on growth, survival and reproduction, colonies of three species of scleractinian corals were experimentally injured and their recovery was monitored. This experiment was conducted at Mermaid Cove, Lizard Island, Australia (Figure 2.1). The three species, *Acropora hyacinthus*, *A. gemmifera* and *Goniastrea retiformis* were selected because they have contrasting life-histories and morphologies. *Acropora hyacinthus* is a large, fast-growing tabular coral, susceptible to physical disturbances, with a relatively short life-span, *A. gemmifera* is a medium sized, robust, digitate coral with a moderate growth rate and life-span, and *G. retiformis* is a medium sized, slow growing massive coral with a relatively long life span (see plate for morphology, Hall and Hughes 1996). All species are hermaphroditic broadcast spawners which, on the Great Barrier Reef, release eggs and sperm simultaneously over a few consecutive nights each spring (Harrison *et al*. 1984; Willis *et al*. 1985). The experiment was initiated in February 1994, just prior to the onset of oogenesis in these species, and terminated in early November of the same year, just before mass spawning. This timing allowed for the collection of mature gonads from corals. At this stage of development, eggs and testes are clearly identifiable, and meaningful comparisons can be made between species and injury regimes.

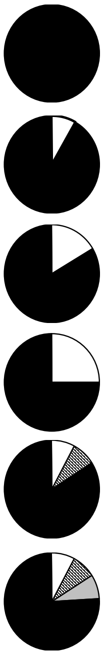
### 5.3.1. Response to injury: experimental design

Two aspects of damage were investigated for the three species (Figure 5.1). The first was injury size where 8% (Tr 1), 16% (Tr 2) and 25% (Tr 3) of colonies were injured.

The second was repetitive injury where colonies were injured twice (initially and three months later, Tr 4) or three times (initially, three months and six months later, Tr 5). For repetitive injury, the level of damage inflicted at each injury event was 8% of the colony. Undamaged colonies were used as controls for injury size while the controls for colonies repeatedly damaged (Tr 4 and Tr 5, Figure 5.1) were the treatments with one injury of an equivalent size (Tr 2 and Tr3 respectively, Figure 5.1).

Figure 5.1. An outline of the injury regimes inflicted on the three species.

**Injury Regime**



| Treatment | Injury Size | Timing |
|---|---|---|
| Control | 0% | |
| Tr 1 | 8% | Februrary ☐ |
| Tr 2 | 16% | February ☐ |
| Tr 3 | 25% | February ☐ |
| Tr 4 | 8%<br>8% | February ☐<br>May ▨ |
| Tr 5 | 8%<br>8%<br>8% | February ☐<br>May ▨<br>August ▩ |

A strict criteria was used to eliminate colonies originally included in the experiment (i.e. 20 colonies per species and treatment) to prevent the confounding of results with natural injury events. Only colonies where natural injury levels did not exceed 5% of the colony area during the experiment were used in the final analysis, resulting in an unbalanced design. Additionally, only large colonies from each species were selected to avoid any effect of colony size (average projected area ± 1 se = 2067 ± 134 $cm^2$, 329 ± 18 $cm^2$ and 250 ± 11 $cm^2$ for *A. hyacinthus*, *A. gemmifera*, and *G. retiformis,* respectively). The type of injury inflicted on colonies was tissue mortality. Tissue was removed by blowing away the tissue with compressed air (see chapters 3 and 4). Colonies were monitored photographically in February, May, August and November. To relocate the experimental colonies at each census, horticultural tags were nailed to the substratum close to the colony. At each photographic census, a 33 $cm^2$ tile was placed on the colony as a measure of scale. The response variables measured in this experiment were growth (colony growth and regeneration), survivorship, and reproduction.

### 5.3.2. Effects of injury on colony growth, regeneration and survival

To determine the effect of injury size and frequency of damage on colony growth in injured and control colonies, the increase in projected area of a colony over nine months, minus localised regrowth, was calculated from photographs of colonies taken at the beginning and end of the experiment. Photographs were projected onto paper, colonies were traced and the resulting area was digitized. Any loss of area due to natural injury that occurred within the nine month period was subtracted from the total colony area. The effect of injury size and frequency of damage on the regeneration of injuries for each species and injury treatment was calculated by digitising the amount of localised regrowth that had occurred on the injury site over nine months. This area was again determined by comparing photographs taken at the beginning and end of the experiment. Survival of colonies was monitored in-situ every three months to establish total colony mortality over nine months.

An ANCOVA was used to test the effect of size and frequency of damage on colony growth (change in size over nine months), the dependent variable being final colony size ($cm^2$) and the covariate being initial colony size ($cm^2$). ANCOVA was also used to test the effect of size and frequency of damage on the amount of lesion regenerated over nine months. The dependant variable for this analysis was regenerated area ($cm^2$) and the covariate was injury size ($cm^2$). Planned comparisons were used for both analyses to detect differences between the three injury sizes (and uninjured controls), and between colonies with different frequencies of damage and their respective size controls. Where necessary, data were log $_{(10)}$ transformed to meet the assumptions of the analyses.

### 4.3.3. Effects of injury on reproduction

Reproductive samples were collected from colonies at the end of the experiment (i.e. just prior to the mass spawning event on the Great Barrier Reef). These samples were preserved and decalcified using standard procedures (e.g. Hall and Hughes 1996). To test whether the effects of injury size and frequency were localized or colony-wide, replicate reproductive subsamples were collected midway between the center and edge of colonies, close to (<5 cm) and away ( > 15cm) from the damaged site in injured colonies and from a similar position for uninjured colonies. The response variables to injury were polyp fecundity and egg volume per polyp (mm$^3$).

The response variables were estimated from measurements and counts of eggs sampled from randomly chosen polyps per sample, 10 polyps per sample for the two *Acropora* species and 7 polyps per sample for *G. retiformis*. Different polyp numbers were used for the *Acropora*s and *G. retiformis* because they vary in the arrangement of their eggs and testes within the polyp, and the number of gonad bearing mesenteries per polyp. In the *Acropora* species, there are 8 complete mesenteries, in which two pairs bear eggs and 2 pairs bear testes (Wallace 1985). In contrast, *G. retiformis* has up to 20 mesenteries per polyp (Babcock 1991) and the central line of eggs are encapsulated with sperm. The randomly selected polyps were dissected under a dissecting microscope, and the number of eggs were counted for each polyp. A subset of the total eggs counted per sample was then randomly selected and measured using an image analysis program , (10 eggs per sample for the *Acropora*s, and all eggs per polyp for *G. retiformis*). Eggs were ellipsoid in shape, so their volume was estimated using the formula $(4/3 * \pi * d_1/2 * d_2/2 * d_3/2)$ where $d_1$, $d_2$, and $d_3$ are orthogonal diameters. To determine polyp fecundity, the presence or absence of mature gonads was recorded for each randomly selected polyp. For each sample, the volume of eggs per polyp (mm$^3$) was estimated using the following equations: egg volume per polyp (mm$^3$) = average number of eggs per polyp * average egg volume.

To test for colony-wide effects of injury size and of the frequency of damage for each species, a one-way ANOVA was carried out on the two response variables, polyp fecundity and egg volume per polyp ($mm^3$). Samples collected away from the damaged area were compared to those of the undamaged controls. Planned comparisons were used to test for differences between the three size treatments and the uninjured control, and between colonies with different frequencies of damage and their respective controls.

To test for localised effects of injury size and frequency of damage for each species, a split-plot ANOVA was conducted on the two reproductive response variables. Samples taken away from the damaged site were compared with those taken close to the damaged site. The fixed effects of the model were treatment (damage), position within colony (position), and their interaction. The random effects were colony nested within treatment, and colony nested within treatment by position. Planned comparisons were used to detect differences between the three sizes of injury, and between colonies with different frequencies of damage and their respective size controls. To meet the assumptions of the ANOVA for both analyses, the data for egg volume per polyp ($mm^3$) were $\log_{(10)}$ transformed and the data for polyp fecundity were arcsine square-root transformed.

### 5.3.4. The loss of reproductive output as a result of injury

The amount of reproductive output lost from an injury measuring 75 $cm^2$ projected area for each species was determined for the female component of reproduction using the following formula: Loss of reproductive output = egg volume per polyp ($mm^3$) * polyp density per unit projected area ($cm^2$) * projected area of injury (75 $cm^2$). Values for polyp density (number of polyps per unit projected area) in these species were obtained from Hall and Hughes (1996). A projected area of 75 $cm^2$ represents an approximate injury size of 33%, 25% and 5% for large colonies of *G. retiformis*, *A. gemmifera*, and *A. hyacinthus*, respectively.

## 5.4. RESULTS

### 5.4.1. Response to injury by scleractinian corals

Injuries of different sizes and frequencies had a marked effect on reproduction but did not influence colony growth, regeneration and survivorship over nine months. The effect of injury on reproduction was variable, differing among species and injury regimes for polyps fecundity and egg volume per polyp ($mm^3$). This result reflects differences in the response of the three species to damage. All species showed the capacity to regrow polyps lost as a result of injury. These results are presented in detail in the following paragraphs.

### 5.4.2. The effect of injury on colony growth, regeneration and survival

All colonies grew during the course of the experiment and growth rates were unaffected by either the size of the injury or frequency of damage (Table 5.1, Figure 5.2a). There was an order of magnitude of difference in the growth of colonies (change in absolute projected area) of *A. hyacinthus* as compared with *G. retiformis* and *A. gemmifera* because of differences in growth rates and colony sizes. The change in absolute size was higher for *Acropora hyacinthus* because it grows faster and has larger colonies. For all species, the area lost to injury was regenerated through the growth of new polyps. These polyps were able to overgrow algae that had settled on the injured site. There was no effect of injury size or frequency of damage on the amount of regrowth over nine months for any species (Table 5.1, Figure 5.2b). However, the amount of regeneration varied among species by an order of magnitude (Figure 5.2b) because *A. hyacinthus* produced a greater area of new tissue than the other two species (Figure 5.2b). All colonies were alive at the end of the experiment. Consequently, the short-term survival of colonies was not influenced by either injury size or frequency of damage.

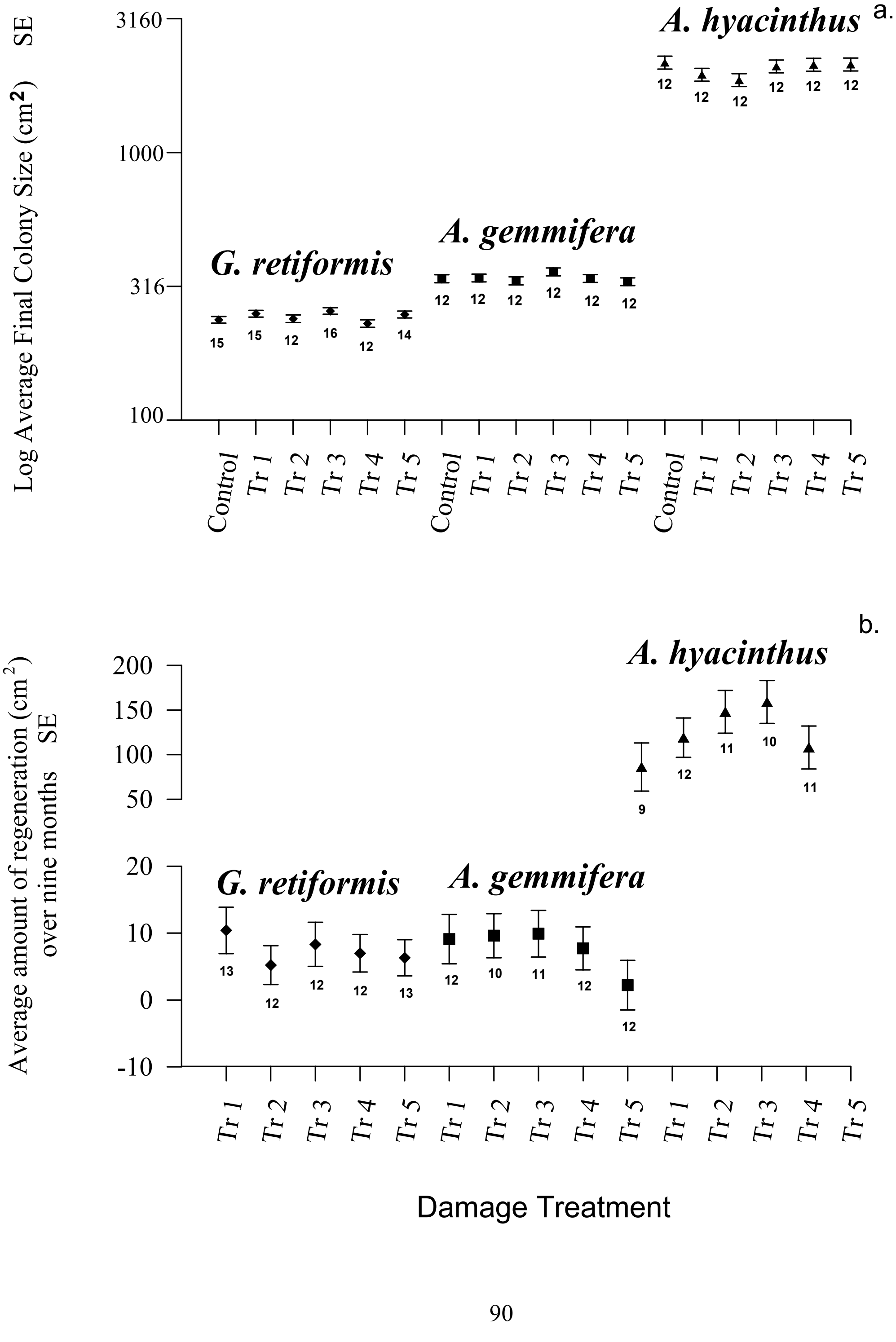
### 5.4.3. The effect of injury on reproduction

There was a marked effect of injury on reproduction for the three species at both a colony-wide and localised scale. On a colony-wide scale, this impact was variable among injury regimes and the two reproductive variables, polyp fecundity and egg volume per polyp. The localised response was more uniform, with all measured components of reproduction showing a localised decline in response to injury. No further effect was detected for the two different injury regimes at a local scale with the exception of polyp fecundity for colonies of *A. gemmifera* that had been injured three times. The loss of reproductive output associated with injury was substantial. This loss differed among species. These results are discussed in detail below.

#### 5.4.3.1. Colony-wide effects of injury on reproduction

There was a colony-wide effect of injury on reproduction for the three species, but this impact varied among the various components of reproduction and injury regimes. There was a pronounced effect of repetitive injury on reproduction for *A. hyacinthus* while *A. gemmifera* and *G. retiformis* were most affected by injury size. These effects of injury were detected by a decrease in polyp fecundity for colonies of *G. retiformis* (Table 5.2, Figure 5.3) and a decline in egg volume per polyp ($mm^3$) for colonies of the two *Acropora* species (Table 5.2, Figure 5.4).

Figure 5.2. (a) Final colony size ($cm^2$) adjusted for initial colony size by ANCOVA showing the effect of injury size and frequency of damage on absolute colony growth for each species. (b) The amount of regeneration ($cm^2$) over nine months, adjusted for initial injury size by ANCOVA, for each injury treatment by species. (Error bars represent one standard error, and the numbers below each data point correspond to the number of colonies used in the analysis).



*Acropora hyacinthus* showed a significant colony-wide decline of 24% in egg volume per polyp ($mm^3$) for colonies that had been damaged three times (Table 5.2, Figure 5.4a). This effect was produced by a decline in egg size (size control (Tr 3) = 6.3 x $10^{-2}$ + 0.002 $mm^3$ and repetitive injury x 3 (Tr 5) = 4.8 x $10^{-2}$ + 0.001 $mm^3$) but not egg number per polyp (Tr 3 = 6 $\pm$ 0.1 and Tr 5 = 6 $\pm$ 0.2). No effect on egg volume per polyp ($mm^3$) was detected for *A. hyacinthus* colonies that had been injured twice (Table 5.2). Additionally, there was no colony-wide effect of the size or frequency of damage on polyp fecundity *for A. hyacinthus* (Table 5.2, Figure 5.3a).

*Acropora gemmifera* showed a general decline in egg volume per polyp ($mm^3$) with increasing injury size (Table 5.2, Figure 5.4b). This effect was due to a reduction in both egg size and number (egg size = 5 x $10^{-2}$ $\pm$ 0.002 $mm^3$, 4 x $10^{-2}$ $\pm$ 0.002 $mm^3$, 3.5 x $10^{-2}$ $\pm$ 0.001 $mm^3$ and average egg number per polyp = 6.3 $\pm$ 0.2, 6.2 $\pm$ 0.2, 5.4 $\pm$ 0.2 for injuries comprising 8% (Tr 1), 16% (Tr 2) and 25% (Tr 3) of the colony respectively). No colony-wide effect was detected for egg volume per polyp ($mm^3$) in colonies that had been damaged repeatedly (Table 5.2, Figure 5.4b), nor for polyp fecundity under the different injury size and frequency regimes (Table 5.2, Figure 5.3b).

*Goniastrea retiformis* exhibited a significant colony-wide decline of 28 - 67% in polyp fecundity for colonies that sustained damage greater than 8% of colony area (Table 5.2, Figure 5.3c). No colony-wide effect on egg volume per polyp ($mm^3$) was detected for injury size and frequency of damage (Table 5.2, Figure 5.4c), nor for polyp fecundity from colonies that were repeatedly damaged (Table 5.2, Figure 5.3c).

### 5.4.3.2. Localised effects of injury on reproduction

There was a significant localised decline of all measured reproductive parameters for all species in response to injury (Table 5.3). This response was not exacerbated by injury size or frequency of damage with the exception of polyp fecundity for colonies of *A. gemmifera* that had been injured three times (Table 5.3, Figure 5.3b).

Table 5.1. Summary of ANCOVA results testing the effects of injury size and frequency on colony growth (cm$^2$) and regeneration (cm$^2$) for each species. The covariate for colony growth is initial colony size and the covariate for regeneration is injury size.

| Response | Injury | | Covariate | | Tr 2 vs Tr 4 | | Tr 3 vs Tr 5 | | Size | |
|---|---|---|---|---|---|---|---|---|---|---|
| Variable | $F_{df}$ | P | $F_{df}$ | P | $F_{df}$ | P | $F_{df}$ | P | $F_{df}$ | P |
| *A. hyacinthus* | | | | | | | | | | |
| Colony Growth | $1.13_{5,65}$ | 0.352 | $431_{1,65}$ | 0.000 | $2.89_{1,65}$ | 0.094 | $0.04_{1,65}$ | 0.840 | $0.004_{3,65}$ | 0.998 |
| Regeneration | $1.47_{4,47}$ | 0.227 | $12.01_{1,47}$ | 0.001 | $1.56_{1,47}$ | 0.218 | $1.57_{1,47}$ | 0.216 | $1.70_{2,47}$ | 0.194 |
| *A. gemmifera* | | | | | | | | | | |
| Colony Growth | $0.74_{5,65}$ | 0.595 | $898_{1,65}$ | 0.000 | $0.18_{1,65}$ | 0.672 | $3.07_{1,65}$ | 0.084 | $0.13_{3,65}$ | 0.940 |
| Regeneration | $0.78_{4,51}$ | 0.543 | $5.94_{1,51}$ | 0.018 | $0.18_{1,51}$ | 0.678 | $2.70_{1,51}$ | 0.107 | $0.02_{2,51}$ | 0.984 |
| *G. retiformis* | | | | | | | | | | |
| Colony Growth | $1.83_{5,77}$ | 0.117 | $739_{1,77}$ | 0.000 | $0.80_{1,77}$ | 0.373 | $0.56_{1,77}$ | 0.458 | $1.30_{3,77}$ | 0.279 |
| Regeneration | $0.44_{4,56}$ | 0.780 | $8.35_{1,56}$ | 0.006 | $0.20_{1,56}$ | 0.656 | $0.27_{1,56}$ | 0.609 | $0.90_{2,56}$ | 0.412 |

Table 5.2. Summary of ANOVA results testing colony-wide effects of injury size and frequency on egg volume per polyp (mm$^3$), and the proportion of polyps with mature gonads (polyp fecundity) for the three species.

| Response | Injury | | Tr 2 vs Tr 4 | | Tr 3 vs Tr 5 | | Size | |
|---|---|---|---|---|---|---|---|---|
| Variable | $F_{df}$ | P | $F_{df}$ | P | $F_{df}$ | P | $F_{df}$ | P |
| *A. hyacinthus* | | | | | | | | |
| EVPP (mm$^3$) | $3.14_{5,54}$ | 0.015 | $0.92_{1,54}$ | 0.343 | $9.65_{1,54}$ | 0.003 | $1.29_{3,54}$ | 0.287 |
| PF (%) | $0.98_{5,112}$ | 0.434 | $0.000_{1,112}$ | 1.000 | $2.87_{1,112}$ | 0.093 | $1.50_{3,112}$ | 0.217 |
| *A. gemmifera* | | | | | | | | |
| EVPP (mm$^3$) | $3.16_{5,54}$ | 0.014 | $2.38_{1,54}$ | 0.129 | $2.44_{1,54}$ | 0.124 | $3.96_{3,54}$ | 0.013 |
| PF (%) | $0.62_{5,106}$ | 0.686 | $0.32_{1,106}$ | 0.571 | $0.02_{1,106}$ | 0.880 | $0.749_{3,106}$ | 0.528 |
| *G. retiformis* | | | | | | | | |
| EVPP (mm$^3$) | $0.42_{5,51}$ | 0.832 | $0.01_{1,51}$ | 0.94 | $0.02_{1,51}$ | 0.892 | $0.56_{3,51}$ | 0.457 |
| PF (%) | $13.03_{5,89}$ | 0.000 | $0.03_{1,89}$ | 0.874 | $2.87_{1,89}$ | 0.094 | $17.31_{3,89}$ | 0.000 |

Table 5.3. Summary of ANOVA results testing the localised effects of injury size and frequency on egg volume per polyp ($mm^3$) and proportion of polyps with mature gonads (polyp fecundity) for the three species.

| Response | Injury | | Position | | Injury*Position | | Tr 2 vs Tr 4 | | Tr 3 vs Tr 5 | | Size | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Variable | $F_{df}$ | P | $F_{df}$ | P | $F_{df}$ | P | $F_{df}$ | P | $F_{df}$ | P | $F_{df}$ | P |
| *A. hyacinthus* | | | | | | | | | | | | |
| EVPP ($mm^3$) | $2.99_{4,45}$ | $\underline{0.029}$ | $6.01_{1,44}$ | $\underline{0.018}$ | $0.67_{4,44}$ | 0.618 | $0.78_{1,45}$ | 0.381 | $8.96_{1,45}$ | $\underline{0.004}$ | $0.53_{2,45}$ | 0.591 |
| PF (%) | $0.61_{4,93}$ | 0.662 | $5.51_{1,87}$ | $\underline{0.021}$ | $1.69_{4,87}$ | 0.158 | $0.31_{1,93}$ | 0.581 | $0.29_{1,93}$ | 0.593 | $1.26_{2,93}$ | 0.288 |
| *A. gemmifera* | | | | | | | | | | | | |
| EVPP ($mm^3$) | $1.98_{4,45}$ | 0.114 | $4.75_{1,45}$ | $\underline{0.035}$ | $2.03_{4,45}$ | 0.107 | $1.38_{1,45}$ | 0.246 | $0.52_{1,45}$ | 0.475 | $0.06_{2,45}$ | 0.068 |
| PF (%) | $1.54_{4,90}$ | 0.198 | $28.08_{1,86}$ | $\underline{0.000}$ | $2.35_{4,86}$ | 0.059 | $0.11_{1,90}$ | 1.000 | $4.19_{1,90}$ | $\underline{0.044}$ | $0.22_{2,90}$ | 1.000 |
| *G. retiformis* | | | | | | | | | | | | |
| EVPP ($mm^3$) | $0.10_{4,42}$ | 0.981 | $7.19_{1,41}$ | $\underline{0.011}$ | $1.09_{4,41}$ | 0.375 | $0.07_{1,42}$ | 0.796 | $0.25_{1,42}$ | 0.617 | $0.07_{2,42}$ | 0.932 |
| PF (%) | $7.61_{4,75}$ | $\underline{0.000}$ | $9.50_{1,72}$ | $\underline{0.003}$ | $2.33_{4,72}$ | 0.063 | $0.12_{1,75}$ | 1.000 | $0.40_{1,75}$ | 1.000 | $12.54_{2,75}$ | $\underline{0.000}$ |

There was an overall decline of up to 50% in the reproductive variables sampled close to the injury site as compared to those sampled away from the injury site, and uninjured control colonies (see position bars, Figures 5.3 and 5.4). The localised decline in egg volume per polyp ($mm^3$) was due mainly to a decrease in the number of eggs per polyp present in samples close to the damaged area as compared to those further away (Table 5.4).

There was no additive effect of size of injury or frequency of damage on any of the reproductive variables for the three species on a local scale, with one exception (Table 5.3). For *A. gemmifera*, there was a 28% decline in polyp fecundity for samples collected close to the damaged site as opposed to those samples taken away further away for colonies that had been damaged three times (Figure 5.3b).

### 5.4.4. Loss of reproductive output associated with injury

Striking interspecific differences were detected in the volume of eggs lost as a result of an injury comprising 75 $cm^2$ projected area of the colony. The loss of egg volume per 75 $cm^2$ projected area was approximately an order of magnitude greater for the two *Acropora* species (41.7 $mm^3$ and 30.2 $mm^3$ for *A. hyacinthus* and *A. gemmifera*, respectively) than *G. retiformis* (3.2 $mm^3$). The loss of eggs as a result of an injury comprising 75 $cm^2$ projected was substantial for all species since it represented a loss of 33%, 25% and 5% of egg production by large colonies of *G. retiformis*, *A. gemmifera* and *A. hyacinthus*, respectively

Figure 5.3. Proportion of polyps with mature gonads averaged over colonies for injury treatments: uninjured controls, injury size (Tr 1,2 and 3) and repetitive damage (Tr 4 and 5) and position effects within colonies (colony-wide and localised) for *A. hyacinthus* (a), *A. gemmifera* (b) and *G. retiformis* (c ). Clear bars represent samples taken away from (>15cm) the injury and grey bars represent samples close to (<5cm) the injury. (Error bars represent one SE, and the numbers within bars correspond to the number of samples).

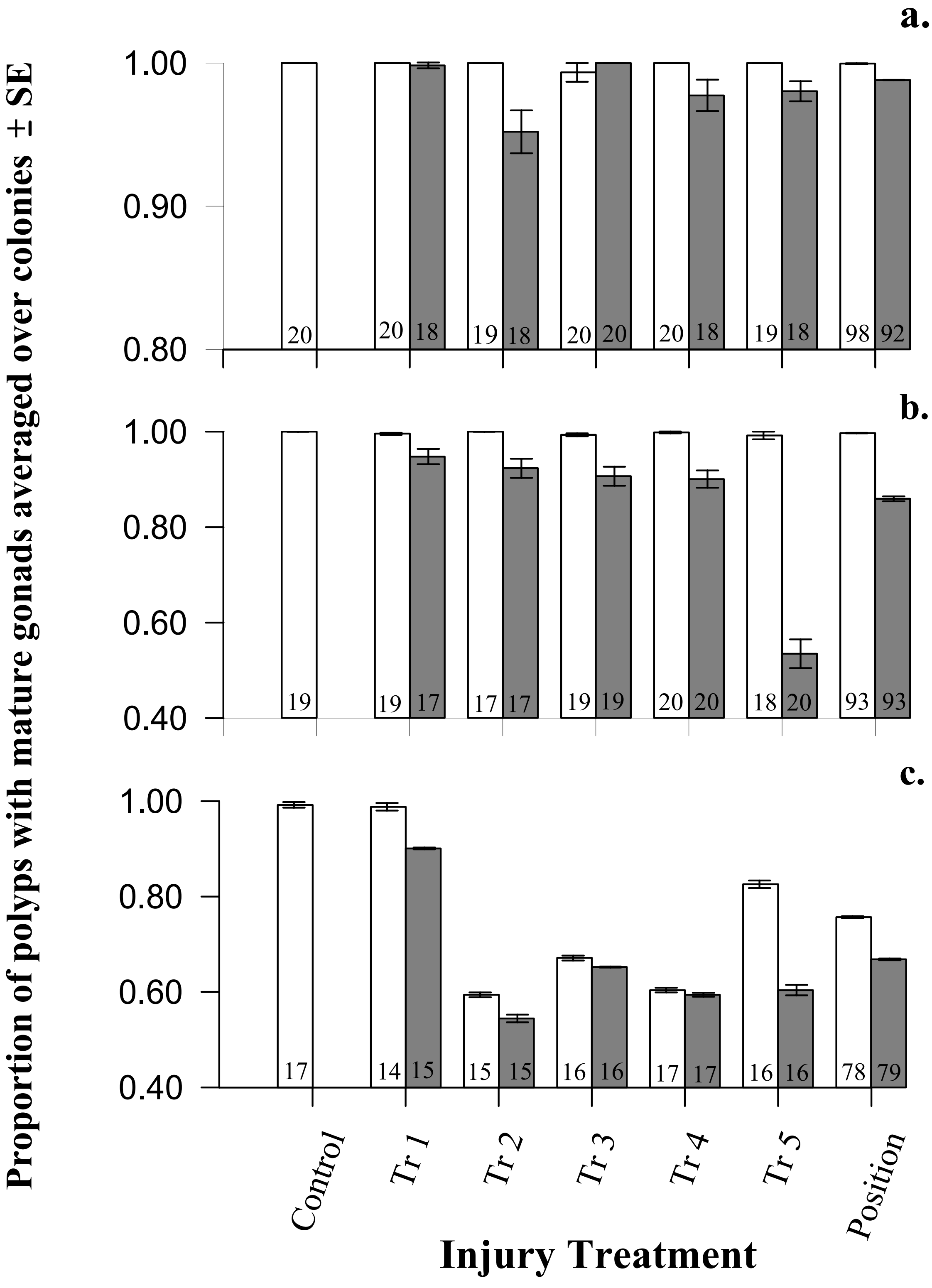


Figure 5.4. Average egg volume per polyp ($mm^3$) for injury treatments (uninjured controls, injury size (Tr 1, Tr 2, Tr 3) and repetitive damage (Tr 4 and Tr 5)) and positions within colonies (colony-side and localised) for *A. hyacinthus* (a), *A. gemmifera* (b), and *G. retiformis* (c ). Clear bars represent samples taken away from (>15cm) the injury and grey bars represent samples taken close to (<5cm) the injury. (Error bars represent one SE, and the numbers within bars correspond to the number of samples).

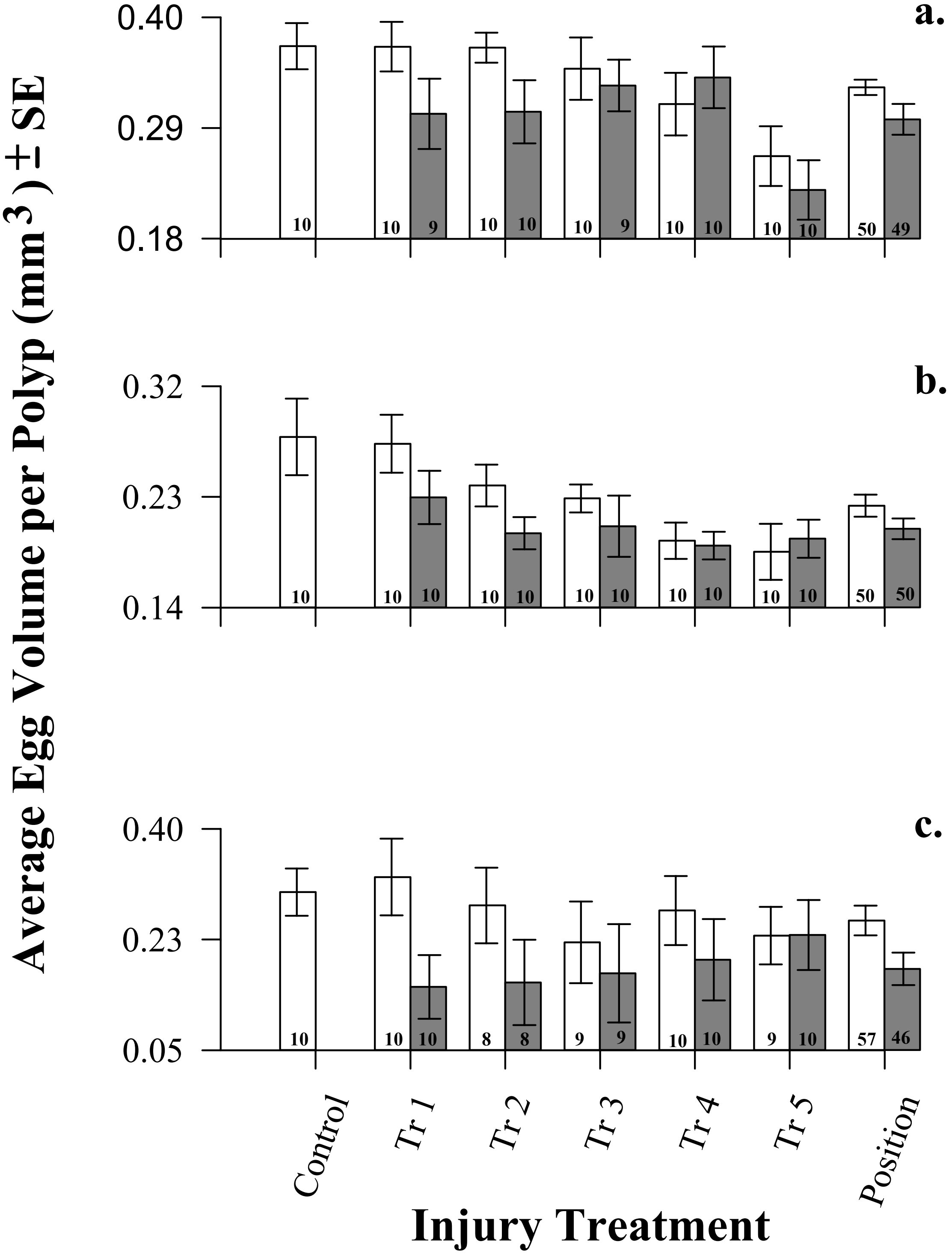


Table 5.4. Mean egg number per polyp and mean egg size (± one standard error) for samples collected close to (near) and away (far) from the injury site for colonies of *A. hyacinthus*, *A. gemmifera* and *G. retiformis*.

| | Egg number per polyp | | Egg size (mm$^3$) | |
|---|---|---|---|---|
| Species | Near | Far | Near | Far |
| *A. hyacinthus* | 5.1 ± 0.07 | 6.2 ± 0.08 | 0.06 ± 0.001 | 0.06 ± 0.0008 |
| *A. gemmifera* | 5.0 ± 0.06 | 6.0 ± 0.08 | 0.04 ± 0.0006 | 0.04 ± 0.0006 |
| *G. retiformis* | 51 ± 1.7 | 63 ± 1.7 | 0.006 ± 0.0004 | 0.006 ± 0.0003 |

## 5.5. DISCUSSION

### 5.5.1. Response to injury

The survival of modular organisms to injury is variable, and lies along a continuum from sensitive to resistant (e.g. Liddle 1991; Belsky *et al*. 1993). Organisms that are resistant to damage are those that have evolved two alternative (but not mutually exclusive) strategies to minimise damage to their tissues (e.g. Begon *et al*. 1986; Belsky *et al*. 1993; Rosenthal and Kotanen 1994). Organisms can avoid damage (avoidance strategies) through defensive behaviour (e.g. flight, camouflage, autotomy) and/or the production of structural (e.g. spines, thorns, hairs, protective coatings) and chemical (secondary chemicals) defenses. The effect of damage may also be negated through the presence of organs and physiological traits that promote the regeneration (regrowth) of lost tissues (tolerance strategies) (e.g. Gross 1969; Arnold 1988; Belsky *et al*. 1993; Room *et al*. 1994; Rosenthal and Kotanen 1994). In contrast, organisms that are sensitive to injury lack the defenses that offers protection from damage, and are unable to regenerate lost tissue (Liddle 1991; Belsky *et al*. 1993; Rosenthal and Kotanen 1994). In this study, all colonies of the three coral species investigated here survived the two injury regimes inflicted upon them over nine months, and thus appear fairly resistant to damage. Scleractinian corals have evolved a number of traits that promote resistance to damage including defensive structures (e.g. hard skeletons, protective coverings of mucus, and nematocysts), and physiological features that allow the regeneration of lost polyps. This ability to resist damage through a combination of avoidance and tolerance strategies has evolved not only in scleractinian corals, but in a large number of other modular organisms, most probably in response to the relatively unpredictable nature of the wide variety of agents that can cause them injury (e.g. White 1979; Karlson 1988; Belsky *et al.* 1993; Connell *et al.* 1996; Hall 1997).

Although the short-term survival of colonies in this experiment was not affected by damage, the longer-term survival of individuals may be compromised for several reasons. First, damage reduces the size of an individual, and mortality in modular organisms is size-specific (Harper 1977; Hughes 1984; Babcock 1991) and second, the damaged area provides a site for the entry of pathogens and boring organisms, and space for the settlement of other organisms (e.g. Bak *et al.* 1977; Begon *et al.* 1986). These settlers and invaders may later compete with the modular organism for food and space, infect healthy tissue with diseases, and cause structural damage to the supporting tissue ( e.g. Wahle 1983; Hughes and Jackson 1985; Begon *et al.* 1986; Babcock 1991).

### 5.5.2. Strategies of resistance

The results of this experiment suggest that tolerance strategies in response to mechanical damage are favoured by *A. hyacinthus*, *A. gemmifera* and *G. retiformis* because the defensive traits of corals (e.g. hard skeleton, protective mucus coating, and nematocysts) characterised in avoidance strategies are ineffective in preventing tissue mortality under experimental conditions. Belsky *et al.* (1993) proposed that tolerance strategies may be favoured over avoidance strategies in plants when (1) increased investment in defensive structures or chemicals is limited by ecological, genetic and physiological constraints, (2) increased defence does not reduce the amount of damage inflicted, (3) replacing damaged tissue is less costly than increasing investment in defensive tools, and (4) annual losses of biomass are minimal. This rationale can also be applied to the corals species investigated here because (1) they are relatively long-lived so the probability of being injured repetitively in their life-span is high (Hughes and Jackson 1985; Babcock 1991), (2) investing in specialised defenses would be mal-adaptive since the type of damage inflicted is caused by a wide range of agents, and the occurrence of such injuries is unpredictable in nature (Brown and Howard 1985; Grigg and Dollar 1990; Brown 1997), and (3) most routine injuries are small (e.g. Bythell *et al*. 1993; Meesters *et al*. 1997; Chapter 2).

Organisms regenerate lost body parts or biomass through a combination of normal growth processes, the function of specialised structures (e.g. storage organs and dormant meristems and buds) and physiological traits (e.g. compensatory photosynthesis, regenerated tissue photosynthates utilised for recovery, and rapid resource allocation) (see reviews: Goss 1969; Belsky *et al* 1993; Rosenthal and Kotanen 1994). In this study, all species had the capacity to regenerate lost polyps, most probably through many of the processes described above (e.g. budding of new polyps, reallocation of resources, utilisation of lipid reserves).

The amount of regeneration detected over nine months for *A. hyacinthus*, *A. gemmifera* and *G. retiformis* was similar regardless of the size and frequency of damage (Figure 5.2b). This consistency in amounts of regeneration among injury treatments is most probably due to two factors. First, all injuries were wedge-shaped and thus bordered by a similar amount of living tissue, a factor considered important in defining regenerative ability (e.g. Meesters and Bak 1995; Meesters *et al*. 1997b; but see Oren *et al*. 1997). Second, rates of regeneration of the repetitive injuries (i.e. those inflicted later on in the experiment) was similar to those inflicted initially because regeneration rates are exponential (Bak 1983) and most of the recovery process occurs within 10 days (Meesters *et al*. 1992; Meesters *et al*. 1997).

### 5.5.3. Trade-offs between growth, reproduction and survival

Life-history theory predicts that organisms with indeterminate growth will balance resource allocation between somatic longevity and reproductive output, resulting in a trade-off between current and future fecundity (e.g. Bell 1980; Kirkwood 1981; Bazzaz *et al*. 1987). The results of this study suggest that future reproductive output is being enhanced through the iteration of new polyps (growth) but at a cost to current reproduction since there was a negative effect of damage on reproduction but not growth and survival in damaged colonies of *A. hyacinthus*, *A. gemmifera* and *G. retiformis*.

Trade-offs can occur between regeneration and any of the various components of reproduction (e.g. number and size of offspring, Begon *et al*. 1986; Harrison and Wallace 1990). In this study, a decline in the number of eggs produced was detected for damaged colonies of *G. retiformis*, while the number and size of eggs were reduced in damaged colonies under different injury regimes for the two *Acropora* species. These results infer that resources usually available for reproduction are presumably being utilised for the formation of new polyps and to fuel competitive interactions with algae and other fouling organisms that settle on the damaged area (e.g. Bak *et al*. 1977; Van Veghel and Bak 1994; chapter 4).

The reallocation of resources in modular organisms after injury can occur not only at an organismal level (i.e. whole colony or plant, e.g. Bazzaz *et al*. 1987; Rinkevich and Loya 1989; Haukioja 1991) but also at a more localised scale (i.e. parts of the organism, e.g. Bazzaz *et al*. 1987; Haukioja 1991; Van Veghel and Bak 1994; Hall 1996). In this study, there was a localised decline in reproduction for all three species. Whether this decline has a significant impact on the reproductive output of the colony can only be assessed if the extent of the decline is known. However, I predict that the impact will be minimal in large corals if the localised response is small, given the huge reproductive potential of the rest of the colony. At a colony-wide level, the response to injury was variable and dependent on the species and injury regime. This differential pattern of response may reflect interspecific differences in life-histories. For example *A. gemmifera* and *G. retiformis* may be more tolerant of repetitive episodes of damage because these species have a longer life-span than *A. hyacinthus*. Conversely, larger amounts of damage may have a disproportionately greater effect on *A. gemmifera* and *G. retiformis* colonies because these species are significantly smaller than *A. hyacinthus*. As a consequence, fewer polyps are available to carry out biological functions (e.g. feeding and photosynthesis), reducing the resources available for growth, reproduction and maintenance.

Damage to modular organisms results in the death or removal of tissue, thus reducing the feeding, photosynthetic, and reproductive capacity of the individual, and destroying part of its stored reserves (Jackson and Palumbi 1979; White 1979; Wahle 1983; Hughes and Jackson 1985; Trumble *et al*. 1993). The loss of reproductive tissue in terms of egg volume ($mm^3$) associated with an injury of 75 $cm^2$ projected area was quite substantial for the three study species as it represented the loss of 33%, 25% and 5% of the colony's production of eggs for *G. retiformis*, *A. gemmifera* and *A. hyacinthus*, respectively. Although unmeasured, a similar loss would have also occurred for sperm since the study species are hermaphroditic, and eggs and testes are both located within individual polyps (Harrison and Wallace 1990). The impact of this loss on the reproductive success of an individual colony may potentially be quite severe because there are fewer eggs (and sperm) available for fertilisation processes. A lower rate of fertilisation success may lead to fewer planula being produced which has consequences for reproductive success in the next generation. It has been proposed that broadcasting corals such as the species investigated here will maximise fitness through the production of a large number of planula with a lower probability of survival rather than producing a few well-developed propagules (Begon *et al*. 1996; Hines 1986; Harrison and Wallace 1990).

The morphological characteristics of an organism can play a major role in determining the amount of tissue lost from an organism as a result of damage. In corals for example, the density of polyps in a given projected area is determined by the size of the polyp, the spacing between polyps, and the nature and extent of branching (Hall and Hughes 1996). The 10-fold difference in loss of egg volume ($mm^3$) per 75 $cm^2$ projected area between the two *Acropora* species and *G. retiformis* is a consequence of contrasting morphologies; both the *Acropora*s are branching species and thus have approximately 10 times the number of polyps per unit projected area than the massive species *G. retiformis* (also see Hall and Hughes 1996).

In conclusion, colonies of *A. hyacinthus*, *A. gemmifera* and *G. retiformis* were resistant to damage, at least in the short-term, since the survival of colonies was not affected over nine months. The colonies responded to injury with the growth of new polyps (tolerance strategy), thus enhancing future reproductive success. However, the cost of such a strategy was a decline in current fecundity. This type of response is not unique to corals but has evolved in response to damage in many modular organisms (White 1979, Belsky *et al*. 1993). Tolerance strategies have probably evolved in modular organisms because they are injured by a wide range of agents and providing defensive mechanisms against all causes of injury would be too costly (Belsky *et al*. 1993).

# CHAPTER 6: GENERAL DISCUSSION

Vulnerability to damage varies among species and lies along a continuum from sensitive to resistant. Scleractinian corals are frequently injured by natural processes (Chapter 2) so it is most probable that they are fairly resistant to damage. A short-term investigation into the effect of damage on the survival of three species of corals supports this statement since no whole-colony mortality was observed over nine months (Chapter 5).

Species resistant to damage have evolved two alternative, but not mutually exclusive, strategies in response to injury. Corals can invest in defensive mechanisms to avoid damage (avoidance strategies) or regrow lost parts after injury has occurred (tolerance strategies) (e.g. review by Belsky *et al.* 1993). The results of this study have shown that both strategies are being utilised by most corals as evidenced by the fact that (1) scleractinian corals have invested in defensive structures (e.g. hard skeletons, nematocysts, and a protective mucous covering) to protect themselves against certain types of damage and (2) all injuries of the twelve study species (with the exception of *Acropora palifera*) that resulted in the death or removal of tissue from a colony were regenerated via the production of new polyps.

Investment in avoidance and tolerance strategies is energetically costly and has evolved in association with a wide range of other life-history traits. Given the highly unpredictable nature of the many agents that cause damage in corals, it is extremely likely that the level of investment in defences will vary, since it would be far too costly to protect against all forms of damage. In this study, some corals were found to be more vulnerable to damage than others, suggesting that investment in defensive traits is indeed variable. The addition of new injuries to colonies of the longer-lived species *Goniastrea retiformis* and *Acropora gemmifera* was less (<80%) than for the shorter-lived species, *Acropora hyacinthus* under routine levels of natural injury. This infers that long-lived species invest more resources towards defence than shorter-lived species, most probably because long-lived species have a greater probability of being injured during their life-time.

Investment in tolerance strategies (e.g. regrowth of lost parts) also appears to be variable since the ability of corals to regenerate injuries differed among species. In general, arborescent, bushy and tabular species were better at regenerating small lesions than massive and submassive species (Chapters 2, 3, 4). The proximate reason for this pattern appears to be linked to the physiological constraints associated with growth, particularly skeletogensis. The ultimate reason may again be associated with trade-offs between resistance strategies and their effect on other life-history traits. For example, short-lived species regenerate injuries more rapidly than longer-lived species while the opposite is true for investment in some mechanisms of defence. Thus, it may be advantageous for short-lived corals to respond to infrequent damage events or minimal tissue losses with rapid regrowth rather than investing in expensive defensive tools that may never be utilised during their life-span. As a consequence of this strategy, more resources can be allocated to reproduction, increasing the chance of representation in future generations. Differences in the allocation of available resources between defence and regeneration represent contrasting strategies of coral species in response to damage.

An idea of the effectiveness of resistance strategies can be obtained by examining the amount of damage present on a particular colony at any given time as a function of both injury rate and recovery. The numbers of pre-existing injuries on colonies of *G. retiformis* was much higher for *G. retiformis* than on *Acropora gemmifera* and *A. hyacinthus* (chapter 2). This was due to the fact that *G. retiformis* colonies accumulated injuries over time since the turn-over of injuries was slow ( low rates of regeneration and few new injuries) while *A. hyacinthus* colonies had a high turn-over of injuries (larger numbers of new injuries and faster regeneration rates) under routine conditions (1994 - 1995 census). The turn-over of injuries for *A. gemmifera* was intermediate between *G. retiformis* and *A. hyacinthus*. This pattern was also consistent among morphological groups since partial mortality was lower in branching and tabular corals than massive and digitate corals (chapter 2), and the regenerative ability of branching and tabular species was greater than massive and sub-massive species (chapter 3). Thus, morphology appears to play an important role in the allocation of resources towards defence and regeneration.

Jackson (1979) proposed that modular organisms with a high commitment to their place of settlement would invest more in maintenance and defence of the integrity of the colony surface as opposed to those morphologies that were more fugitive in nature. The results of this study indicate that corals with a massive or semi-encrusting form have more resources invested in defence while branching and tabular species have more regrowth potential. The amount of investment in defence and/or regeneration in response to damage is, in turn, associated with particular growth form characteristics that impact on longevity, reproductive output, growth rate, and other life-history processes. For example, a massive morphology has the advantage of a smooth, robust growth form with a large area of attachment to the substratum, making it less vulnerable to mechanical damage (e.g. storm damage), and too homogeneous in outline to offer protection or bite-sized food for many potential predators. The disadvantage of a massive morphology is that the large area of attachment increases the chance of substrate interactions, the impact of an injury cannot be minimised through breakage as seen for branching corals, and there are fewer polyps per unit area for physiological processes (feeding, photosynthesis, and reproduction). In contrast, branching species have a more fragile morphology and they are more vulnerable to mechanical damage, foraging by some coralivores, and their three-dimensional pattern of branching results in the creation of crevices suitable for hiding potential predators (e.g. *Drupella* and *Acanthaster planci*). The advantage of this growth form is a fast growth rate, more polyps per unit area for physiological processes, and fewer substrate interactions since the colony is elevated into the water column. Thus, the favouring of one strategy of resistance over another is clearly associated with morphology since morphology influences the fate of corals under different injury regimes.

Trade-offs in resource allocation not only occur between avoidance and tolerance strategies, but also among regeneration and other life-history traits when an injury is inflicted, because regeneration requires resources. Experimental injuries caused a decline in reproduction but had no affect on colony growth or short-term survival of *G. retiformis*, *A. gemmifera*, and *A. hyacinthus*, suggesting that resources are being reallocated from reproduction to regeneration. The advantage of such a strategy is that future reproduction is being enhanced through the iteration of new polyps (growth) but at the cost of current reproduction. Given that the three study species have moderate (up to 10 - 15 years) to long (> 20 years) life-spans, losses to current reproduction as a result of damage can be compensated for over several reproductive seasons.

The ability of corals to recover from injury is dependent on the magnitude, intensity, and type of damage. In general, the superficial loss of tissue and skeleton (scraping injuries) was less severe than tissue mortality or breakage (Chapter 3 and 4), and smaller injuries had a greater potential for recovery than larger ones (Chapter 2). Recovery of injuries appears to be related to the amount of damage inflicted, the zone of tissue available for regeneration, and the abundance of organisms that settle onto the injured area (Chapter 3 and 4). The pattern of response to particular types of damage and the factors affecting recovery were consistent across species. In contrast, the effect of the magnitude and intensity of damage differed among species. The impact of large injuries on reproduction was pronounced for the two smaller species, *G. retiformis* and *A. gemmifera* but not for *A. hyacinthus* (Chapter 5) The opposite trend was apparent for frequency of damage where the reproduction of *A. hyacinthus* colonies that had been experimentally damaged three times was lower than would have been accounted for by size alone (Chapter 5). Additionally, at catastrophic levels of natural damage for *A. hyacinthus*, the rate of recovery declined resulting in an accumulation of injuries on colonies (Chapter 2). These results suggest that the longer-lived species, *G. retiformis* and *A. gemmifera* are more resilient to recurrent injury since they have a lower investment in tolerance strategies than the shorter-lived species, *A. hyacinthus*. However, longer-lived species are more vulnerable to large injuries than *A. hyacinthus*. The loss of a large portion of tissue has a disproportionately greater effect on these small corals because it substantially reduces the number polyps left for physiological processes such as photosynthesis and feeding.

In summary, the results of this study demonstrate that coral species differ in their response to injury due to differences in the allocation of available resources between defence, regeneration and reproduction. Short-lived species generally invest more in regeneration while longer-lived species invest more in defence. As a consequence, resilience to different injury regimes is species-specific. Once a colony is injured, available resources are reallocated from reproduction to regeneration, thus impacting on reproductive success. However, due to the limitations of a PhD, this study has only examined some of the processes associated with injury for a very small proportion of corals from the Indo-Pacific. Given the increasing concern about human impacts on coral reefs, future research needs to directed towards a greater understanding of the life-history strategies of many more corals, especially with respect to their response to damage. In particular, I would like future research to include species both sensitive and resistant to damage, and with a wide range of morphologies, to broaden our existing knowledge in this area. In comparison to terrestrial plants, studies of this nature are rare for colonial marine organisms, providing the opportunity for some exciting and innovative science.

This study on injury and regeneration of scleractinian corals has increased our knowledge of some of the underlying mechanisms that affect the recovery of corals from damage, and provides a basis for understanding the consequences of different injury regimes on coral reefs. This is important because injury can adversely affect corals at the individual, population and community level. Injury can influence the fitness of an individual colony, either directly by reducing survivorship, reproductive output, feeding and photosynthetic capacity and destroying stored resources, or indirectly because the recovery of an injury requires resources that are normally available for reproduction and growth (Bak *et al*. 1977; Wahle 1983; Rinkevich and Loya 1989). Normally, losses due to routine injury regimes in coral populations are offset by asexual reproduction, larval recruitment and growth (Bak and Luckhurst 1980; Hughes and Jackson 1985; Babcock 1991). Conversely, under catastrophic injury regimes (e.g. cyclones, hurricanes, predator outbreaks, disease epidemics), the short-term effects on populations of the more susceptible species may be deleterious (Bythell *et al*. 1993). Rather than being detrimental to coral communities, however, this process may actually enhance species diversity by removing the dominant species that pre-empt settlement of the less common corals, and prevent their growth through competitive exclusion (e.g. Connell 1978). On the other hand, coral cover and diversity may decline dramatically in communities if the time interval between catastrophic events is insufficient to allow recovery of the more dominant species (e.g. Jamaican reefs, Hughes 1989).

In conclusion, this study has significantly increased our understanding of injury and regeneration of scleractinian corals, particularly for common reef-crest species on the Great Barrier Reef.  It has shown that the vulnerability of corals to damage is species specific and dependent on the nature and extent of injury regimes while recovery from damage is related to life-history and morphological strategies and the characteristics of the injury.  The major finding from this study is that corals, like many modular organisms, have evolved contrasting strategies in response to damage.

## REFERENCES

Arnold, A. 1988. Caudal autotomy as a defence. In "Biology of the Reptilia, Volume 2, Ecology B", Gans, C. and Huey, R.B., editors. John Wiley & Sons, New York. pp. 235-273.

Babcock. R.C. 1991. Comparative demography of three species of scleractinian corals using age- and size-dependent classifications. Ecol. Monogr. 61:225-244.

Bak, R.P.M. 1983. Neoplasia, regeneration and growth in the reef-building coral *Acropora palmata*. Mar. Biol. 77:221-227.

Bak, R.P.M. and Engel, M.S. 1979. Distributions, abundance and survival of juvenile hermatypic corals (Scleractinia) and the importance of life-history strategies in the parent coral community. Mar. Biol. 54: 341-352.

Bak, R.P.M. and Luckhurst, E. 1980. Constancy and change in coral reef habitats along depth gradients at Curacao. Oecologia 47: 145-155.

Bak, R.P.M. and Steward-Van Es, Y. 1980. Regeneration of superficial damage in the scleractinian corals *Agaricia agaricites* F. *Purpurea* and *Porites astreoides*. Bull. Mar. Sci. 30:883-887.

Bak, R.P.M., Brouns, J.J.W.M. and Heys, F.M.L. 1977. Regeneration and aspects of spatial competition in the scleractinina corals *Agaricia agaricites* and *Montastrea anularis*. Proc. 3rd Intern. Coral Reef Symp. Miami. 2:143-148.

Barnes, D.J. and Chalker, B.E. 1990. Calcification and photosynthesis in reef-building corals and algae. In "Ecosystems of the world, coral reefs", Dubinsky, Z., editor Elsevier Science Publishers, New York. pp. 109-131.

Barmes. D. and Lough, J. 1992. Systematic variations in the depth of skeleton occupied by coral tissue in massive colonies of *Porites* from the Great Barrier Reef. J. Exp. Mar. Biol. 159: 113-128.

Bazzaz, F.A., Chiariello, N.R., Coley, P.D., and Petelka, L.F. 1987. Allocating resources to reproduction and defense. BioScience 37: 58-67.

Begon, M., Harper, J.L. and Townsend, C.R. 1986. Ecology: Individuals, populations and communities. Blackwell Scientific Publications, Oxford, UK.

Bell, G. 1980. The costs of reproduction and their consequences. Am. Nat. 116: 45-76.

Belsky, A.J., Carson, W.P. and Jensen, C.L. 1993. Overcompensation by plants: Herbivore optimisation or red herring? Evol. Ecol. 7; 109-121.

Benson, A.A., Patton, J.S. and Abraham, S. 1978. Energy exchange in coral reef ecosystems. Atoll Res. Bull. 220: 33-54.

Birkeland, C. 1997. Life and Death of Coral Reefs. Chapman & Hall, New York. pp. 1-536.

Brown, B.E. 1988. Assessing environmental impacts on coral reefs. Proc. 6th Int. Coral Reef Symp. Townsville. 1: 71-80.

Brown, B.E. 1994. Solar damage in intertidal corals. Mar. Ecol. Prog. Ser. 105: 219-230.

Brown, B.E. 1997. Disturbances to reefs in recent times. In "Life and Death of Coral Reefs", Birkland, C., editor. Chapman & Hall, New York. pp. 354-379.

Brown, B.E. and Howard, L.S. 1985. Assessing the effects of "stress" on reef corals. Adv. Mar. Biol. 22: 1-63.

Bythell, J.C., Gladfelter, E.H. and Bythell, M. 1993. Chronic and catastrophic natural mortality of three common Caribbean reef corals. Coral Reefs. 12: 143-152.

Chadwick-Furman, N.E. 1995. Effects of scuba diving on coral reef invertebrates: relative vulnerabilities and critical levels of diving activity. Proc. 6th Int. Conf. Coelenterate Biology, Amsterdam. 1: 91-100.

Chamberlain, J.A.J. 1978. Mechanical properties of coral skeleton: compressive strength and its adaptive significance. Paleobiology. 4: 419-435.

Chou, L.M., Wilkinson, C.R., Liculanan, W.R.Y., Alino, P., Cheshire, A.C., Loo, M.G.K., Tangjaitrong, S., Sudara, S., Ridzwan, A.R. and Soekarno. 1994. Status of coral reefs in the Asian region. Proc. 3rd ASEAN~Australia Symp. On Living Coastal Resources, Thailand. 1: 1-10.

Clark, L.A. and Pregibon, D. 1997. Tree-based Models. In "Statistical Models in S", editors Chambers, J.M. and Hastie, T.J. Chapman & Hall, London. pp. 377-419.

Coll, J.C., La Barre, S., Sammarco, P.Wl, Williams, W.T., and Bakus, G.J. 1982. Chemical Defences in Soft Corals (Coelenterata : Octocorallia) of the Great Barrier Reef : A study of comparative toxicities. Mar. Ecol. Progr. Ser. 8: 271-278.

Collett, D. 1991. Modelling binary data. Chapman & Hall, New York.

Connell, J.H. 1973. Population ecology of reef-building corals. In "Biology and geology of coral reefs", Jones, O.A. and Endena, R., editors. Volume 2. Academic Press, New York, USA. pp. 205-245.

Connell, J.H. 1978. Diversity in tropical rain forests and coral reefs. Science. 199: 1302-1310.

Connell, J.H. 1996. Disturbance and recovery of coral assemblages. Proc. 8th Intern. Coral Reef Symp., Panama. 1: 9-22.

Connell, J.H., Hughes, T.P. and Wallace, C.C. 1996. A 30-year study of coral community dynamics: Influence of disturbance and recruitment on abundance, at several scales in space and time. Ecol. Monogr. 67: 461-488.

Craik, W., Kenchington, R. and Kelleher, G. 1990. Coral-Reef Management. In "Ecosystems of the world, coral reefs", Dubinsky, Z., editor. Elsevier Science Publishers, New York. pp. 453-467.

Cumming, R. 1996. The corallivorous gastropods *Drupella cornus*, *D. fragum* and *D. rugosa*: Ecology and impact on coral communities at Lizard Island, Great Barrier Reef. PhD Thesis, James Cook University, Townsville.

Darke, W.M. 1991. Growth and growth form of the massive coral *Porites*. PhD Thesis, James Cook University of North Queensland, Australia.

De'ath, G. and Moran, P.J. 1998. Factors affecting the behaviour of crown-of-thorn starfish (*Acanthaster planci* L.) on the Great Barrier Reef: 1: Patterns of activity. J. Exp. Mar. Biol. Ecol. 220: 83-106.

Done, T.J. 1987. Simulation of the effects of *Acanthaster planci* on the population structure of massive corals of the genus *Porites*: evidence of population resilience? Coral Reefs. 6: 75-90.

Falkowski, P.G., Jokiel, P.L. and Kinzie, R.A. 1990. Irradiance and corals. In "Ecosystems of the world, coral reefs", Dubinsky, Z., editor. Elsevier Science Publishers, New York. pp. 89-107.

Glynn, P.W. 1990. Feeding ecology of selected coral-reef macroconsumers: Patterns and effects on coral community structure. In "Ecosystems of the world, coral reefs", Dubinsky, Z., editor. Elsevier Science Publishers, New York. pp. 439-452.

Goss, R.J. 1969. Principles of Regeneration. Academic Press. New York.

Grigg, R.W. and Dollar, S.J. 1990. Natural and anthropogenic disturbances on coral reefs. In "Ecosystems of the world, coral reefs", Dubinsky, Z., editor. Elsevier Science Publishers, New York. pp. 365-400.

Hall, V.R. 1992. Life-history strategies of common reef-crest corals at Lizard Island. Honours Thesis, James Cook University, Townsville.

Hall, V.R. 1996. Effects of injury on growth, reproduction and survivorship for common reef-crest corals. Proc. 8th Intern. Coral Reef Symp., Panama. 1: 571 –574.

Hall, V.R. 1997. Interspecific differences in the regeneration of artificial injuries on scleractinian corals. J. Exp. Mar. Biol. Ecol. 212: 9-23.

Hall, V.R. and Hughes, T.P. 1996. Reproductive strategies of modular organisms: Comparative studies of reef-building corals. Ecology. 77: 950-963.

Harper, J.L. 1977. Population biology of plants. Academic Press, London, England.

Harper, J.L. and White, J. 1974. The demography of plants. Annu. Rev. Ecol. Syst. 5: 419-463.

Harrison, P.L. and Wallace, C.C. 1990. Reproduction, dispersal and recruitment of scleractinian corals. In “Ecosystems of the world, coral reefs”, Dubinsky, Z., editor. Elsevier Science Publishers, New York. pp. 133-207.

Harrison, P.L., Babcock, R.C., Bull, G.D., Oliver, J.K., Wallace, C.C. and Willis, B.L. 1984. Mass spawning in tropical reef corals. Science. 223: 1186-1189.

Hatcher, B.G., Johannes, R.E., and Robertson, A.I. 1989. Review of research relevant to the conservation of shallow tropical marine ecosystems. Oceanogr. Mar. Biol. Annu. Review. 27: 337-414.

Haukioja, E. 1991. The influence of grazing on the evolution, morphology and physiology of plants as modular organisms. Phil. Trans. Royal Soc. London B. 333: 241-247.

Herms, D.A. and Mattson, W.J. 1992. The dilemma of plants: to grow or defend. The Quart. Rev. Biol. B7: 283-335.

Highsmith, R.C. 1982. Reproduction by fragmentation in corals. Mar. Ecol. Prog. Ser. 7: 207–226.

Hines, A.H. 1986. Larval problems and perspectives in life histories of marine invertebrates. Bull. Mar. Sci. 39: 506-525.

Hughes, T.P. 1984. Population dynamics based on individual size rather than age: A general model with a reef coral example. Am. Nat. 123: 778-795.

Hughes, T.P. 1987. Skeletal density and growth form of corals. Mar. Ecol. Progr. Ser. 35: 259-266.

Hughes, T.P. 1989. Community structure and diversity of coral reefs: The role of history. Ecology. 70: 275-279.

Hughes, T.P. 1994. Catastrophes, Phase shifts, and large-scale degradation of a Caribbean reef. Science. 265: 1547-1551.

Hughes, T.P. and Jackson, J.B.C. 1980. Do corals lie about their age: Some demographic consequences of partial mortality, fission and fusion. Science. 209: 713-715.

Hughes, T.P. and Jackson, J.B.C. 1985. Population dynamics and life-histories of foliaceous corals. Ecol. Monogr. 55: 141-166.

Hughes, T.P., Ayre, D. and Connell, J.H. 1992. Evolutionary ecology of corals. Trends Ecol. Evol. 7: 292-295.

Jackson, J.B.C. 1979. Morphological Strategies of Sessile Animals. In "Biology and Systematics of Colonial Organisms", Eds. Larwood, G. & B.R. Roser. Academic Press, London. pp. 499-555.

Jackson, J.B.C. and Palumbi, S.R. 1979. Regeneration and partial predation in cryptic coral reef environments: preliminary experiments on sponges and ectoprocts. In "Biology of sponges", Levi, C. and Boury-Esnault, N., editors. CNRS, Paris. pp. 303-308.

Kay, A.M. and Liddle, M.J. 1989. Impact of human trampling in different zones of a coral reef flat. Environ. Manag. 13: 509-520.

Karlson, R.H. 1988. Size-dependent growth in two zoanthid species: a contrast in clonal strategies. Ecology. 69: 1219-1232.

Karlson, R.H. and Hurd, L. 1993. Disturbance, coral reef communities, and changing ecological paradigyms. Coral Reefs. 12: 117-166.

Kirkwood, T.B.L. 1981. Repair and its evolution: Survival versus reproduction. In "Physiological Ecology: An evolutionary approach to resource use", Townsend, A.R.R. and Calow, R., editors. Blackwell, Oxford, England. pp. 165-189.

Kobayaski, A. 1984. Regeneration and regrowth of fragmented colonies of the hermatypic corals *Acropora formosa* and *Acropora nasuta*. Galaxea. 3: 13-23.

Lang, J.C. and Chornesky, E.A. 1990. Competition between scleractinian reef corals – a review of mechanisms and effects. In "Ecosystems of the world, volume 24, Coral reefs", Dubinzky, Z., editor. Elsevier, Amsterdam. pp. 209-252.

Liddle, M.J. 1991. Recreation Ecology: Effects of trampling on plants and corals. Trends Ecol. Evol. 6: 13-16.
Liddle, M.J. and Kay, A.M. 1987. Resistance, survival and recovery of trampled corals on the Great Barrier Reef. Biol. Conserv. 42: 1-18.

Loya, Y. 1976. The Red Sea coral *Stylophora pistillata* is an r-strategist. Nature. 259: 478-480.

Maschinski, J. and Whitham, T.G. 1989. The continuum of plant responses to herbivory: The influence of plant association, nutrient availability, and timing. Am. Nat. 134: 1-19.

Meesters, E.H. and Bak, R.P.M. 1993. Effects of coral bleaching on tissue regeneration potential and colony survival. Mar. Ecol. Prog. Ser. 96: 189-198.

Meesters, E.H. and Bak, R.P.M. 1995. Age-related deterioration of a physiological function in the branching coral *Acropora palmata*. Mar. Ecol. Progr. Ser. 121: 203-209.

Meesters, E.H., Bos, A. and Gast, G.J. 1992. Effects of sedimentation and lesion position on coral tissue regeneration. Proc. 7th Intern. Coral Reef Symp., Guam. 2: 681-688.

Meesters, E.H., Noordeloos, M. and Bak, R.P.M. 1994. Damage and regeneration: Links to growth in the reef-building coral *Mostastrea anularis*. Mar. Ecol. Prog. Ser. 112: 119-128.

Meesters, E.H., Wesseling, I and Bak, R.P.M. 1996. Partial mortality in three species of reef-building corals (Scleractinia) and the relation with colony morphology. Bull. Mar. Sci. 58: 230-242.

Meesters, E.H., Wesseling, I. And Bak, R.P.M. 1997a. Partial mortality in reef-building corals (Scleractinia): Ecological effects of colony size in modular organisms. J. Sea Research. 37: 131-144.

Meesters, E.H., Pauchliet, W. and Bak, R.P.M. 1997b. Predicting regeneration of physical damage on a reef-building coral by regeneration capacity and lesion shape. Mar. Ecol. Prog. Ser. 146: 91-99.

Oren, U., Benayahu, Y. and Loya, Y. 1997. Effects of lesion size and shape on regeneration of the Red Sea coral *Favia favus*. Mar. Ecol. Progr. Ser. 6: 101-107.

Palumbi, S.R. and Jackson, J.B.C. 1982. Ecology of cryptic coral reef communities. II. Recovery from small disturbance events by encrusting bryozoan: The influence of 'host' species and lesion size. J. Exp. Mar. Biol. Ecol. 64: 103-115.

Pichon, M. and Morrissey, J. 1981. Benthic zonation and community structure of South Island Reef, Lizard Island (Great Barrier Reef). Bull. Mar. Sci. 31: 581-591.

Porter, J.W., Battey, J.F. and Smith, G.J. 1982. Pertubation and change in coral reef communities. Proc. Natl. Acad. Sci. USA. 79: 1678-1681.

Rinkevich, B. 1996. Do reproduction and regeneration in damaged corals compete for energy allocation? Mar. Ecol. Progr. Ser. 43: 297-302.

Rinkevich, B. and Loya, Y. 1989. Reproduction in regeneration colonies of the coral *Stylophora pistillata*. In "Environmental Quality and Ecosystem Stability: Vol IV-B, Environmental Quality". Spanier, E., Steinberger, Y., and Luria, N., editors. ISEEQS, Israel.

Room, P.M., Maillette, L. and Hanan, J.S. 1994. Module and metamer: Dynamics and virtual plants. In "Advances in Ecological Research", Volume 25, Begon, M. and Fitter, A.H., editors. Academic Press Limited, London. pp. 105-157.

Rosenthal, J.P. and Kotanen, P.M. 1994. Terrestrial plant tolerance to herbivory. Trends Ecol. Evol. 9: 145-148.

Ruesink, J.L. 1997. Coral injury and recovery: matrix models link process to pattern. J. Exp. Mar. Biol. Ecol. 210: 187-208.

Rutzler, Kl, Snatavy, D.L., and Antonius, A. 1983. The black band disease of Atlantic reef corals III: Distribution, ecology and development. PSZNI Mar. Ecol. 4: 329-358.

Sebens, K.P. 1987. The ecology of indeterminate growth in animals. Annu. Rev. Ecol. Syst. 18: 371-407.

Stafford-Smith, M.G. 1993. Sediment-rejection efficiency of 22 species of Australian scleractinian corals. Mar. Biol. 115: 229-243.

Stearns, S.C. 1992. The evolution of life histories. Oxford University Press, Oxford, England.

Stephenson, T.A. and Stephenson, A. 1933. Growth and asexual reproduction in corals. Scientific Report Great Barrier Reef Expedition. 3: 167-217.

Strauss, S.Y. 1991. Direct, indirect, and cumulative effects of three native herbivores on a shared host plant. Ecology. 72: 543-558.

Tardent, P. 1963. Regeneration in the Hydrozoa. Biol. Rev. Cambridge Phils. 38: 293-333.

Trumble, J.T., Kolodny-Hirch, D.M. and Ting, I.P. 1993. Plant compensation for arthropod herbivory. Annu. Rev. Entom. 38: 93-119.

Tuma, J.J. 1979. Engineering Mathematics Handbook. Definitions, theorems, formulas and tables. McGraw-Hill Book Company, New York.

Veghel, Van, M.L.J. and Bak, R.P.M. 1994. Reproductive characteristics of the polymorphic Caribbean reef building coral *Montastrea annularis*. III. Reproduction in damaged and regenerating colonies. Mar. Ecol. Progr. Ser. 109: 229-233.

Veron, J.E.N. 1986. Corals of Australia and the Indo-Pacific. University of Hawaii Press, USA.

Veron, J.E.N. and Pichon, M. 1982. Scleractinia of Eastern Australia. Part IV: Family Poritidae. Australian Institute of Marine Science Monographs Series, Volume 5. Australian Institute of Marine Science, Australia.

Wahle, C.M. 1983. Regeneration of injuries among Jamaican gorgonians: The roles of colony physiology and environment. Biol. Bull. 164: 778-790.

Wallace, C.C. 1985. Reproduction, recruitment and fragmentation in nine sympatric species of the coral genus *Acropora*. Mar. Biol. 88: 217-233.

Ward, S. 1995. The effect of damage on the growth, reproduction and storage of lipids in the scleractinian coral *Pocillopora damicornis* (Linnaeus). J. Exp. Mar. Biol. Ecol. 187: 193-206.

White, P.S. 1979. Pattern, process, and natural disturbance in vegetation. Bot. Rev. 45: 230-299.

Williams, G.C. 1966. Adaptation and natural selection: a critique of some current evolutionary thought. Princeton University Press, Princeton, New Jersey, USA.

Willis, B.L., Babcock, R.C., Harrison, P.L., Oliver, J.K. and Wallace, C.C. 1985. Patterns in the mass spawning of corals on the Great Barrier Reef from 1981 to 1984. Proc. 5th Intern. Coral Reef Congr. Tahiti. 4: 343-348.

Woodley, J.D., Chornesky, E.A., Clifford, P.A., Jackson, J.B.C., Kaufman, L.S., Knowlton, N., Lang, J.C., Pearson, M.P., Porter, J.W., Roonery, M.C., Rylaarsdam, K.W., Tunnicliffe, V.J., Wahle, C.M., Wulff, J.L., Curtis, A.S.G., Dallmeyer, M.D., Jupp, B.P., Kodhl, M.A.R., Neigel, J., and Sides, E.M. 1981. Hurrican Allen's Impact on Jamaican Coral Reefs. Science. 214: 749-755.