Regeneration from injury and resource allocation in sponges and corals

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1. Introduction

Sessile bottom-dwelling animals (the “epifauna”) provide important biogenic habitat for other trophic levels including fish and invertebrates, and contribute much of the biomass, biodiversity and physical frameworks in marine ecosystems exposed to natural and man-made disturbances (BRADSTOCK AND GORDON, 1983; SAINSBURY, 1987; HUTCHINGS, 1990; JONES, 1992; SAINSBURY *et al*. 1993; AUSTER *et al*., 1996; KOSLOW *et al*., 2001). The ability of epifauna to survive and recover from disturbances is thus vital to the long-term persistence of other resident organisms, as well as the total biodiversity in these ecosystems.

The regeneration of injured or lost body parts is integral to epifauna survival, and is essential for community recovery from disturbances that injure these animals e.g., predation, storms, bottom-fishing (FISHELSON, 1973; LOYA, 1976a; PEARSON, 1981; TILMANT, 1982; BYTHELL *et al*., 1993; CONNELL *et al*., 1997; LASKER and COFFROTH, 1999). Yet regeneration following injuries is sometimes incomplete or impossible. Regeneration in these cases is often limited in predictable ways by many intrinsic and extrinsic factors. Additionally, somatic growth, sexual reproduction and other life history processes may compete with regeneration for energetic and cellular resources (RINKEVICH, 1996). Limited regeneration combined with impaired life history processes may therefore alter subsequent community and ecosystem recovery to its former state.

The goal of this review is to highlight limits on ecological recovery imposed by the physiological and histological regeneration of individuals after mechanical disturbance and damage. Owing to the preponderance of these types of regeneration studies on particular sessile epifauna groups, this review is restricted to sponges (Phylum Porifera), hard corals (Order Scleractinia), soft corals and sea fans (both Order Alcyonacea).

The first part of this review examines how intrinsic and extrinsic factors limit regeneration in sponges and corals. The second part reviews ecological consequences of regeneration for other life history processes. One important result is that wedemonstrate considerable support for Rinkevich's hypothesis that competing resource demands between regeneration and other life history processes impairs growth, sexual reproduction and the ability to interact with other individuals in recovering sponges and corals. We conclude by discussing ecological consequences of regeneration for larger-scale community and ecosystem structure and function.

2. Factors limiting regeneration in sponges and corals

Many factors affect the capacity for sponges and corals to regenerate lost tissues, skeletons and body parts. We have grouped these factors into two categories, intrinsic (individual-dependent) and extrinsic (environment-dependent) factors (*cf* KRAMARSKY-WINTER and LOYA, 2000).

2.1 Intrinsic factors

2.1.1 Size

The greater surface area of large sponges and corals relative to those with smaller body sizes makes them more vulnerable to disturbance and damage (JACKSON, 1979). Yet the capacity for an injured sponge or coral to regenerate from wounds is positively correlated with increasing body size (BAK *et al*., 1977; WOODLEY *et al*., 1981; HUGHES, 1984; HUGHES and JACKSON, 1985; BYTHELL *et al*., 1993; BAK and MEESTERS, 1998; KRAMARSKY-WINTER and LOYA, 2000). Size-dependent survivorship of wave-generated or experimentally-derived fragments in scleractinians (HIGHSMITH *et al*., 1980; TUNNICLIFFE, 1981; LIDDLE and KAY, 1987; RICHMOND, 1987; SMITH and HUGHES, 1999) and gorgonians (LASKER, 1990) also suggests increased regenerative capacity with increasing size.

Size-dependent regeneration capacity may reflect the relative proportion of injured to healthy tissue remaining after a disturbance. Injuries may occur more frequently on larger sponges and corals, but affect relatively smaller areas of a large animal in contrast to smaller individuals that experience relatively greater tissue loss by the same disturbance (HUGHES and JACKSON, 1985; BAK and MEESTERS, 1998). A wound on a small individual simply has a higher circumference/surface area ratio, which leaves relatively less healthy tissue bordering the wound to contribute to regeneration (MEESTERS *et al*., 1996b).

Several authors have noted that rapid healing in sponges and corals seems to depend on the availability of resources derived from adjacent healthy tissue (KOROTKOVA, 1970; CONNELL, 1973; LOYA, 1976b; WULFF, 1991). While it appears that responses to injuries and subsequent regeneration events are often localized in tissues adjacent to the wound (MEESTERS *et al*., 1994; MESZAROS and BIGGER, 1999; LIRMAN, 2000a), some evidence points to the potential for more extensive i.e., colony-wide, responses to wounding e.g., in the colonial scleractinians *Favia favus* and *Platygyra lamellina* (OREN *et al*., 1997a, b). The measurement of intracolonial transport of ^{14}Clabelled compounds in these species demonstrated widespread reallocation of coral resources through physiologically integrated member units of the colony (“modules”), particularly in corals with spatially extensive wounds. Thus, a large individual sponge or coral with greater total resource availability may recover quickly from a large wound if those resources can be rapidly transported around the animal to the injured site.

At first, the positive correlation between individual size and regenerative capacity described thus far seems to conflict with results from the solitary fungiid coral *Fungia granulosa*, where small corals recovered more rapidly from small injuries than larger individuals (CHADWICK and LOYA, 1990). However KRAMARSKY-WINTER and LOYA (2000) noted that this could be because of the different methods used to damage the corals; the former method inflicted relatively less damage to skeletal material in thin, smaller corals than in larger corals that must regenerate more skeletal material and potentially deeper tissues as well. The importance of wound depth is discussed later in section 2.2.1.3.

If enough uninjured area is available to regenerate damaged tissue, sponge or coral size may not be relevant to the injury response. Sufficient resource availability may explain the apparent lack of relationship between coral size and regenerative capacity in the scleractinians *Acropora palmata* (LIRMAN and FONG, 1997; LIRMAN, 2000a), *Agaricia agaricites* forma *purpurea* (BAK and STEWARD-VAN ES, 1980), *Porites astreoides* (BAK and STEWARD-VAN ES, 1980), *Montastraea annularis* (MEESTERS *et al*., 1994) and the gorgonian *Plexaura homomalla* (WAHLE, 1983a). Thus, the adaptive significance of individual- or colony-wide integration, and therefore body size during regeneration may only be apparent when injury responses are not localized, or when wounds are relatively large (BAK and STEWARD-VAN ES, 1980; WAHLE, 1983a; OREN *et al*., 1997a, b, 1998, 2001).

2.1.2 Age

Regeneration from injuries is also an age-dependent process. Juvenile sponges may not regenerate at all, possibly because archaeocytes are heavily invested in growth processes instead of maintenance processes during this life stage (SIMPSON, 1984). In the case of juvenile cnidarians comprised of a single "primary" polyp, regeneration may not even proceed if central portions of this unit are destroyed (BAK and ENGEL, 1979). However, this might also reflect effects of wound location (see section 2.1.3) if the oral aperture of a coral polyp is an important organizing centre for the direction of regeneration phenomena.

Tissue age within an individual sponge or coral may also limit regeneration as it appears to in other sessile epifauna. For example, young bryozoan zooids show fewer lesions in nature, regenerate faster and are fouled less often by epibionts than older zooids (MENON, 1972; JACKSON and PALUMBI, 1979; PALUMBI and JACKSON, 1982, 1983; JACKSON and HUGHES, 1985). These age-based restrictions in regenerative capacity could reflect evolution of resource translocation strategies towards younger regions of the organism at the periphery of the animal where the risk of injury is highest (PALUMBI and JACKSON, 1982). In the case of colonial organisms, this translocation polarity gradient would also direct resources from feeding "source" modules into young growing "sink" modules. In these cases, reduced regeneration rates in older (proximal) parts may be an unavoidable cost of a translocation strategy designed to emphasize peripheral growth at the expense of proximal repair.

2.1.3 Morphology

The morphological diversity exhibited by sponges and corals has predictable ecological consequences related to resource allocation to different life history processes, including regeneration. Increasing morphological complexity (i.e., “unitary” animals < “runner-” < “vine”- <, “plate”- <, “mound”- < “tree”-shaped animals, using the terminology of Jackson, 1979) results in a more integrated whole-animal response to resource allocation. The latter require greater investments towards somatic maintenance and repair to survive in their habitats (JACKSON, 1979; KOJIS and QUINN, 1985), and should thus regenerate more completely or faster than those less integrated. The wide interspecific variation in regeneration rates among shape variants corroborates this prediction (e.g., VAN VEGHEL and BAK, 1994; HALL, 1997; NAGELKERKEN and BAK, 1998). In general, massive mounding taxa tend to regenerate faster than plating forms (FISHELSON, 1973; BAK and ENGEL, 1979; RIEGL and VELIMIROV, 1991; NAGELKERKEN and BAK, 1998).

Polyp size in colonial corals is also related to regenerative capacity. Small polyp taxa such as *Acropora variabilis* and *Pocillopora danae* regenerate more rapidly than large polyp taxa such as *Favia favus* and *Platygyra lamellina* (FISHELSON, 1973; RIEGL and VELIMIROV, 1991). These differences may be related to the amount of uninjured tissue and skeleton available to supply the wounded area. Corals with small polyps tend to seal and repair wounds by forming a bilayer of encroaching soft tissue over the lesion and secreting an underlying layer of calcium carbonate that envelopes organisms and sediments that may have fouled the lesion (FISHELSON, 1973). Macropolypal corals tend to regenerate from remnant corallites deeper in the coral that eventually re-establish contact with one another to form a continuous layer (FISHELSON, 1973).

The degree to which individuals are constructed of repeated modular units may also have consequences for regenerative capacity. The biological significance of coloniality to marine animals is evident in both the general competitive superiority of colonies to unitary animals, and in the ability to translocate resources between modules and survive despite injuries and death of individual colony members (JACKSON, 1977; BUSS, 1979). While many authors agree that sponges may not have truly "colonial" equivalents (BERGQUIST, 1978; ROSEN, 1979), corals are represented by both solitary (single-polyped) and colonial (multi-polyped) taxa and thus allow the importance of coloniality in regeneration to be considered.

Regeneration studies concur with the hypothesis that solitary corals have poorer regenerative capacities than their colonial counterparts. This is partly due to the generally slower growth of solitary corals (CHADWICK and LOYA, 1990). The autonomy of a single polyp as both supplier and consumer of resources may reduce its regenerative capacity relative to colonial corals that can translocate resources from one polyp to another (KRAMARSKY-WINTER and LOYA, 2000), particularly if daughter segments are still attached to the solitary parent coral e.g., during asexual reproduction (YAMASHIRO and NISHIHIRA, 1998). Furthermore, as sufficient healthy tissue is necessary for regeneration, severely injured solitary corals without this critical biomass may not regenerate the large unitary polyp (KAWAGUTI, 1937), and instead bud off smaller daughter polyps as a last effort to preserve the genet (e.g., JOKIEL AND BIGGER, 1994; KRAMARSKY-WINTER and LOYA, 1996). The importance of an "organizing centre" controlling regeneration has also been particularly well documented in solitary fungiid corals. Generally, a mouth must be present in order for these corals to undergo tissue and skeletal regeneration e.g., in *Fungia* spp. (PREOBRAZHENSKII, 1979; CHADWICK and LOYA, 1990; JOKIEL and BIGGER, 1994; KRAMARSKY-WINTER and LOYA, 1996; but see YAMASHIRO and NISHIHIRA, 1998 for an exception in the fungiid coral *Diaseris distorta*), without which corals will not recover from their wounds.

2.1.4 Genotype

Regenerative capacities differ between growth forms of *Montastraea annularis* (VAN VEGHEL and BAK, 1994; WEIL and KNOWLTON, 1994) and could have a genetic basis (KNOWLTON *et al*., 1992; VAN VEGHEL and BAK, 1993; LOPEZ *et al*., 1999). Few studies have examined differences between genets but preliminary evidence demonstrates that regeneration varies among genetically distinct corals (MEESTERS *et al*., 1996b), suggesting that the genotype modulates injury responses.

2.2 Extrinsic factors

2.2.1 Wound characteristics

The nature of injuries to sessile epifauna caused by natural and anthropogenic disturbances is considerably variable, e.g., the spatial extent of a single disturbance event varies from millimeters and centimeters (e.g., predation, bioerosion) to hundreds and thousands of kilometers (e.g., bottom fishing, storms) (JACKSON, 1991; NYSTRÖM *et al*., 2000). Unfortunately, regeneration from wounds found under natural conditions is rarely examined (3 out of 98 cases in Table 1), and *in situ* lesion properties are rarely characterized concomitantly during the same regeneration study. Furthermore, in contrast to the relatively rapid healing of small wounds, the regeneration of lost modules from increasingly larger lesions has not been very thoroughly investigated, despite the broad size range of wounds found under natural conditions (Table 4). Wound characteristics largely determine the capacity for any sponge or coral to regenerate and recover.

2.2.1.1 Wound size

Incomplete regeneration from large wounds and rapid recovery from small lesions are widely noted phenomena in sponges (HOPPE, 1988; DUCKWORTH, 2003) and scleractinians (BAK *et al*., 1977; BAK and ENGEL, 1979; BAK and STEWARD-VAN ES, 1980; CHADWICK and LOYA 1990; MEESTERS *et al*., 1997a; KRAMARSKY-WINTER and LOYA, 2000; LIRMAN, 2000a; OREN *et al*., 2001). Extrapolating data from studies that estimated regeneration rates in mm^2 from Table 1, it appears that there is an approximately linear decrease in daily regeneration rates with increasing wound size (R^2 = 0.35) (Fig. 1).

Regenerative capacity depends on lesion size because small injured surface areas require fewer resources than larger wounds. For example, significantly more carbon products had to be transported from healthy to damaged areas in the scleractinians *Favia favus*, *Platygyra lamellina* and *Porites* spp. when colonies exhibited large versus small wounds (OREN *et al*., 1997b, 1998). Regeneration capacity of *Montastraea annularis* was highly dependent on wound size, and the amount of tissue that could be regenerated was a linear function of initial lesion area (MEESTERS *et al.*, 1996a, 1997a). Estimating this function for other species could be of value as a predictive tool: if the mean wound size can be used to predict the rate of physiological recovery of colonies, then the expected ecological pattern of recovery of multiple damaged species in a disturbed community (a pattern difficult to observe directly) could be estimated indirectly from the frequency distribution of wound sizes on different species.

2.2.1.2 Wound perimeter

Wound perimeter is a function of both lesion size and shape: a highly convoluted wound has a smaller surface area/perimeter ratio than a circular wound of the same surface area. Short-term regeneration rates are largely determined by wound perimeter, after which time lesion surface area and the surface area/perimeter ratio may become more important to healing (OREN *et al.*, 1997b). The positive relationship between wound perimeter and lesion regeneration capacity was also corroborated by regenerative studies in scleractinians (MEESTERS *et al.*, 1996a; VAN WOESIK, 1998; LIRMAN, 2000a).

As the perimeter of a linear versus circular lesion of the same surface area is associated with more healthy tissue bordering the wound (OREN *et al.*, 1997a), lesion perimeter restricts regeneration by limiting the amount of resources available to wounds with small perimeters. Ultimately, perimeter-based limitations in regenerative capacity have probably evolved to reduce fitness costs associated with high resource allocation to healing large wounds (MEESTERS *et al.*, 1997a).

2.2.1.3 Wound depth

Deep wounds might be predicted to regenerate less well than more superficial ones as the former penetrate both superficial as well as deeper tissue and skeletal elements. Yet at least one study has demonstrated that deeper wounds could be regenerated more rapidly than superficial injuries in some cases, e.g., in the coral *Porites astreoides*, but not in *Agaricia agaricites* (BAK and STEWARD-VAN ES, 1980).

These discrepancies might be explained by taxonomic differences in ability to draw on resources to seal, repair and restore dead tissue and skeletal elements. For example, the solitary coral *Fungia granulosa* possesses deep tissue reserves that penetrate into the corallum and enable the pollyp to draw upon more resources to heal injuries (KRAMARSKY-WINTER and LOYA, 2000). Such "reserves" are becoming increasingly more apparent: isolated patches of healthy tissue surrounding perforated skeletal regions can initiate tissue re-growth in *Porites compressa* (JOKIEL *et al*., 1993); regeneration from remnant tissues located deep in skeletal frameworks of corals permits recovery following bleaching events (RIEGL and PILLER, 2001) or extreme environmental variations (LIRMAN *et al*., 2002). The presence of deep tissue reserves may be combined with other factors e.g., position on an individual and the nutritional status of that individual (BARNES and LOUGH, 1992) to affect regeneration from deep wounds.

2.2.1.4 Wound location

There is much evidence to suggest that regenerative capacity depends on where a wound is inflicted on a colony. For example, an experimentally damaged top of the vase-shaped sponge *Neofibularia nolitangere* healed more quickly than a wound inflicted on lateral surfaces (HOPPE, 1988). The co-ordination of various stages of the regeneration process may also be disrupted by wounds at different locations. Distal lesions in the scleractinian *Acropora palmata* were rapidly sealed by a thin transparent layer of undifferentiated cells with no apparent calcification and no zooxanthellae. In contrast, wounds near the colony base were sealed and calcified almost simultaneously (BAK, 1983; MEESTERS and BAK, 1995). Tissue regeneration and calcification in these more proximal wounds were characterized by the random emergence of new polyps and calices with a slow-growing pigmented lip migrating inwards from the wound perimeter (MEESTERS and BAK, 1993; LIRMAN, 2000a), a structure also observed in *Montastraea annularis* (massive morph), *Meandrina meandrites*, *Porites astreoides*, *P. lobata* and *P. lutea* (MEESTERS and BAK, 1993; VAN WOESIK, 1998). In contrast to species in which distal wounds are more rapidly healed, wounds on branch tips of the gorgonian *Plexaura homomalla* regenerated more slowly than injuries on primary branches (WAHLE, 1983a), and naturally occurring wounds on more centrally-located colony portions of the corals *Porites astreoides* and *Siderastrea siderea* healed more quickly than wounds on peripheral regions (RUESINK, 1997). In some cases, wound location has no effect on rate of regeneration (e.g., MEESTERS *et al*., 1992). These discrepancies might be explained by the degree to which resources can be made available to certain areas on a sponge or coral.

In corals that have variable skeletal thickness on different parts of the colony, wounds in areas where the skeleton is thin have higher ratios of tissue to skeleton surface area, thus conferring greater resource availability for regeneration (CHADWICK and LOYA, 1990). Other sources of among-site variation in regeneration rate might be unavoidable consequences of modular growth. For example, there is growing evidence that many species preferentially transport resources from proximal to distal modules. KIM and LASKER (1998) considered that depletion of colony resources by exterior modules, or “self-shading”, is an emergent property of colonial organisms that can determine growth capacities. Resource translocation and activity of enzymes involved in growth and skeletogenesis are also biased toward distal parts of scleractinians (GLADFELTER, 1983; ISA and YAMAZATO, 1984) but this direction may be important only in colonies that have functional polymorphisms such as feeding specializations that differ between proximal and distal modules (WAHLE, 1983a). Enhanced regenerative capacity in *Plexaura homomalla*, *Porites astreoides* and *Siderastrea siderea* in more central portions in these cases may therefore reflect the larger amount of healthy tissue surrounding proximal wounds and not a translocation polarity gradient (*sensu* WAHLE, 1983a). Similar regeneration rates at the tops versus sides of hermatypic coral colonies can also be explained by sufficient resources reaching the wounded areas, as naturally shaded sides of colonies are photoadapted by accumulating more chlorophyll in side polyps (MEESTERS *et al*., 1992). If such translocation gradients are common among different colonial species, then wound location could limit regenerative capacity if it imposes gradient-based or colony-based restrictions on resource availability at some parts of a wound. The existence and direction of such gradients should be corroborated across a wider range of species. Functional correlations between tissue and skeleton surface area ratios, translocation gradients and spatial variation in wound healing should also be verified experimentally in more cases.

2.2.2 Water temperature

Seasonal increases in water temperatures could be predicted to enhance regenerative capacity due to higher metabolic rates and thus higher growth rates (discussed in KRAMARSKY-WINTER and LOYA, 2000). In some cases, corals have been found to regenerate more rapidly at higher water temperatures, but these effects were likely confounded by increased resource demands imposed by the onset of gametogenesis during colder months (KRAMARSKY-WINTER and LOYA, 2000). In other cases, complete acclimation of corals to ambient conditions allow injuries to regenerate more quickly in areas with above average water temperatures, possibly because corals normally experience occasional influxes of much colder oceanic water that may temporarily stress the animals (LESTER and BAK, 1985).

Short-term regeneration in hermatypic corals that are chronically exposed to abnormally high water temperatures may not be impaired due to coral acclimation to ambient conditions (LESTER and BAK, 1985). However subsequent morbidity due to expulsion of algal symbionts (“bleaching”) in acclimated hermatypic corals may limit regeneration (MEESTERS and BAK, 1993). Regeneration is reduced and sometimes completely absent following experimentally-induced lesions in naturally bleached colonies of *Montastraea annularis*, *Porites astreoides*, *Meandrina meandrites* and *Oculina patagonica* when compared to controls (MEESTERS and BAK, 1993; MEESTERS *et al.*, 1997a; FINE *et al.*, 2002). Artificially bleached clones of *M. annularis* also exhibited reduced regeneration, with some corals showing no signs of regeneration (MASCARELLI and BUNKLEY-WILLIAMS, 1999). Depletion of zooxanthellae due to bleaching may be critical to understanding reduced regeneration in bleached corals because algal symbionts provide energy for coral functions such as mucus production, ciliary action and amoebocytic activity that facilitate regeneration and also protect corals against disease (MASCARELLI and BUNKLEY-WILLIAMS, 1999; MESZAROS and BIGGER, 1999). A blockage of resource translocation between bleached and unbleached areas is also likely to restrict transport of energy and cells from healthy areas to lesions (FINE *et al*., 2002).

2.2.3 Food availability

Depth-related variation in regenerative capacities of hermatypic corals is frequently cited as evidence of food limitation on regeneration rates. Food availability varies with zooxanthellae species and density, degree of symbiont photoacclimation (i.e., concentrations of chlorophyll c_2) and energy reserves and sources (NAGELKERKEN *et al*., 1999). Any change in these factors can result in altered regenerative capacities. For example, slower initial regeneration in deep versus shallow water colonies of the scleractinians *Porites astreoides* and *Stephanocoenia michelinii* was due to reduced energy reserves as light levels decreased with depth, and was not associated with changes in zooxanthellae density or photoacclimation (NAGELKERKEN *et al*., 1999). Since rate of polyp regeneration was positively correlated with depth, energy must have been acquired independently of zooxanthellae or chlorophyll concentrations. This could be achieved heterotrophically or by reallocating energy from other life history processes (NAGELKERKEN *et al*., 1999) including the resorption of reproductive structures (SZMANT and GASSMAN, 1990; SIER and OLIVE, 1994). The positive phototropic response demonstrated by some regenerating zooxanthellate scleractininans (KAWAGUTI, 1937; FRANZISKET, 1970) could also reflect an evolutionary adaptation that satisfies increased intracolonial energetic demands during regeneration. Thus, food limitation may restrict regenerative capacity for colonies in food-poor areas such as for hermatypic corals in deep water, and may select for the evolution of life history traits such as increased dependence on heterotrophic food sources. The role of food availability in ahermatypic sponges and corals should be examined, but it is expected to impose similar restrictions during regeneration.

2.2.4 Sedimentation

Clearing and preventing the accumulation of sediments is energetically demanding (ROGERS, 1990; GUZMÁN *et al.*, 1994), but few studies have demonstrated the effects of sedimentation on regenerative capacity. High levels of sedimentation could increase energy requirements (due to costs of rejecting sediment from the colony) and decrease energy availability (due to shading of photosynthetic endosymbionts; MEESTERS *et al.*, 1992). Sponges and corals are predicted to exhibit slower regeneration rates in habitats with high sediment levels, particularly in species with inefficient mechanisms for handling and removing sediment, e.g., *Acropora palmata* (MEESTERS *et al.*, 1992).

Interestingly, GUZMÁN *et al.* (1994) found faster regeneration of lesions in several scleractinan coral species at sites polluted by the 1986 oil spill at Bahía Las Minas, Panama, than at unaffected areas. Coastal oil pollution and the subsequent sediment leaching from nearby mangroves and seagrass beds did not serve to enhance regenerative capacity directly, but instead triggered the clearing of oily sediments and polyp regeneration by reallocating resources from other life history processes including growth and sexual reproduction (GUZMÁN *et al.*, 1991, 1994). Thus, sedimentation stress may not directly impair regenerative capacity, at least in species with efficient sediment rejection mechanisms (TOMASCIK and SANDER, 1987; MEESTERS *et al*., 1992). Instead, stress-based impairment of other life history processes such as calcification in hermatypic corals (BAK, 1978) may trigger acute survival responses that re-direct resources into colony maintenance and sediment clearing mechanisms.

2.2.5 Disturbance history

The disturbance regime that a sponge or coral experiences is important in determining its capacity to regenerate from injuries imposed by a disturbance event because past injuries (and the energy and cell resources used to recover from them) limit the resources available for regeneration from more recent injuries (LANG DA SILVEIRA and VAN'T HOF, 1977; CUMMING, 2002). The frequency of disturbance relative to the rate of recovery of resources used for regeneration could be of critical importance for predicting community patterns of recovery from chronic disturbance such as repeated bottom fishing. In places where disturbance frequency is high and colonies often experience new injuries while still regenerating from old injuries, then such colonies might be in a chronic state of regeneration. For example, coral "fate" (i.e., whether a coral escapes or is injured/killed by a disturbance event) strongly depends on the occurrence of recent damage by hurricanes; injured corals are significantly more likely to be injured again than previously uninjured corals (HUGHES, 1984; HUGHES and JACKSON, 1985; BABCOCK, 1991). Uninjured corals also are more likely to escape again than be injured or killed, suggesting that other factors (perhaps resistant genotypes) may affect whether a coral is injured or not (HUGHES and JACKSON, 1985). Susceptibility to grazing by parrotfish was also higher in post-hurricane coral communities (BYTHELL *et al*., 1993, 2000). WAHLE (1983a, 1985) documented patterns in wounds on gorgonians, and concluded that cumulative large injuries could be as important as mortality in structuring coral reef communities because of repeated resource demands on regenerating individuals.

2.2.6. Conditions that may select for rapid regeneration

2.2.6.1 Frequently disturbed habitats

Differences in regenerative capacity may be caused by adaptive responses to selection for rapid re-growth in habitats where the probability for injury is high (KOTT, 1981; MEESTERS *et al*., 1996b; BELL, 2002). Such an adaptive response to frequent injury may also indirectly enhance regeneration by selecting for asexual proliferation, suggesting that the two are somewhat distinct but correlated processes (VORONTSOVA AND LIOSNER, 1960; ALVARADO, 2000). Rapid regeneration in animals should evolve in concert with the evolution of asexual strategies such as fragmentation to disperse and colonize new habitats (KARLSON, 1988). Asexually-derived sponge or coral propagules must re-attach themselves to the seafloor and escape mortality by rapidly re-establishing large body sizes, processes that necessitate rapid sequestration and translocation of resources to growing areas. Thus, regenerative capacity is expected to be high in taxa under selective pressure to propagate asexually, for example in habitats where injury frequency and mortality of larvae and small fragments are high, and in habitats where resource competition is intense (HIGHSMITH, 1982; HEYWARD and COLLINS, 1985; KARLSON, 1986; LASKER, 1990; LEWIS, 1996). Several studies report *Acropora palmata* to be one of the fastest regenerators (BAK, 1983), with a well documented life history strategy of prolific fragmentation in response to disturbance (LIRMAN and FONG, 1997).

2.2.6.2 Low latitudinal habitats

The numerous regeneration studies listed in Table 1 permit comparisons between organisms adapted to low- and high-latitude habitats. Note that because there have been no studies on regeneration in corals from high latitudes, these comparisons included only studies on typically sub-tropical and tropical low latitude sponges and corals versus more polar sponges. Among 12 studies of small wounds (Table 2), regeneration rates for nine of 10 low latitude species were higher than rates for the two high latitude species. Among 16 studies of large wounds (Table 3), five of the six highest regeneration rates were observed in low latitude species and nine of the 10 lowest rates were observed among high latitude species. These findings suggest that sponges and corals in lower latitudes generally exhibit faster regeneration than those from higher latitude habitats. The only exception to this trend was the high rate of regeneration from large wounds observed in the polar sponge *Stylopus* sp. (Table 3). This species regenerated from large wounds more quickly than many low latitude sponge and scleractinian species (Table 3). Resources for regeneration may not be restricted in *Stylopus* sp. and some other high latitude sponges. Intense predation by spongivores may also have selected for fast regeneration in these species while tough, fibrous outer sponge ectosome skeletons could have eliminated the need for fast regeneration in others (AYLING, 1981).

It is tempting to link differences in regeneration rates to differences in water temperature as discussed earlier in section 2.2.2. But latitudinal gradients in metabolic rates that affect activities such as regeneration are perhaps more strongly influenced by cycles of growth and reproduction. In turn, these life history processes are controlled by food availability (discussed earlier in section 2.2.3) that could vary between low and high latitude habitats (CLARKE, 1993; BROCKINGTON and CLARKE, 2001). Thus, latitudinal trends in regenerative capacity presented here could be related more to variations in production processes across latitudes than temperature differences.

3 Ecological consequences of regeneration in sponges and corals

Regeneration, growth, reproduction and species encounters require significant energetic resource investments from animals. Energy and cells are finite resources distributed within a sponge or coral and allocated to these often costly life history processes. Individuals that are irreversibly committed to such processes regenerate slowly from injuries (e.g., during gametogenesis, KRAMARSKY-WINTER and LOYA, 2000), or simply do not regenerate at all. Regeneration of very large wounds may halt in favour of growth and reproduction if the fitness of the individual is threatened by resource limitation, as would be the case in spatially extensive lesions (MEESTERS *et al*., 1997a) or in the case of solitary individuals that can revert to asexual budding of daughter polyps.

Energy and cells allocated to life history processes are often redirected into regeneration following injury, and this change in resource allocation potentially limits energy availability for other vital life history processes (MEESTERS *et al*., 1997a). Occasionally trade-offs in favour of regeneration are avoided, and other life history processes are sometimes enhanced (e.g., growth, see section 3.3). Thus, alternate explanations other than energy limitation are required to understand how regeneration may affect other vital life history processes in sponges and corals.

3.1 Potential for sharing resources between regeneration and other life history processes

A well-studied interaction between regeneration and life history processes is the effect of regeneration on sexual reproduction. RINKEVICH (1996) proposed three hypotheses, not necessarily mutually exclusive of one another, to explain dynamics of sexual reproduction following regeneration in corals: (1) energy may be allocated hierarchically to various life history processes, (2) energy may not limit either regeneration or reproduction and (3) regeneration may not be restricted by energy, but instead by potentially limited sources of totipotent stem cells. Support for the first two hypotheses is derived from evidence that energy availability and allocation to life history processes varies in space and time (BEN-DAVID-ZASLOW and BENAYAHU, 1999). The third hypothesis is supported by evidence for the roles that cellular resources play in the life histories of animals in general (reviewed by ZERA and HARSHMAN, 2001). The present review now provides further support for this latter hypothesis by examining cellular events of injury responses in sponges and corals and by demonstrating how regeneration shares cellular "currencies" with other life history processes in these animals.

Sponges and corals rapidly respond to sub-lethal injuries by sealing and defending the wound milieu to ensure additional contents and to prevent foreign particles from entering the lesion, processes that require cellular and energetic resources to fuel (NEEDHAM, 1952; STORR, 1964, 1976). Sealing is effected either by morphallaxis (usually in the case of small wounds) that contracts underlying tissues and stretches/ re-arranging existing cells (HAY, 1966; HARRISON, 1972; STORR, 1976) or by epimorphosis involving a cascade of cell migration, differentiation, and proliferation events (NEEDHAM, 1952; ALVARADO, 2000). Defense is effected through cell- and antibody-mediated immune responses that lead to the phagocytic destruction of foreign matter and dead cells. Remarkable antibody production and phagocytic abilities are demonstrated by wandering totipotent amoeboid cells (amoebocytes) and by lymphocyte-like cells (which may themselves be derived from amoebocytes).

Regeneration is initiated by progenitor blastema cells that originated from dedifferentiated somatic cells or the aggregation of totipotent stem cell reserves (THOUVENY and TASSAVA, 1998). Dedifferentiation is a return to the cell cycle through the destabilization of the differentiated state of various somatic cell types, prompted by signals from tissue surrounding the wound (BROCKES, 1998). Although many cell types of marine invertebrates are permanently committed to their differentiated states, several cell lines can dedifferentiate to follow new fates (GALLIOT and SCHMID, 2002) e.g., sponge choanocytes and pinacocytes are potentially able to dedifferentiate and contribute to blastema formation. In other taxa, totipotent stem cells contribute more to the blastema bud rather than somatic cells, typically in the form of amoebocytic reserve cells including archaeocytes (in sponges) and wandering amoebocytes and interstitial i-cells (in cnidarians). New body parts are then constituted from the subsequent redifferentiation of somatic cells or differentiation of stem cells and/or the repeated budding or fission of individual modular components, e.g., LEE and ELIAS (1991, 2000). Cellular resources used by sponges and corals during the injury responses are summarized in Table 5.

Thus, limited sexual reproduction (or other life history processes) following regeneration may be understood in terms of resource limitations including the restricted production of germ cells from stem cell precursors (RINKEVICH, 1996). This hypothesis can be extended to explain apparent trade-offs between major life history processes and regeneration in sponges and corals. Resources that fuel major life history processes (i.e., energy) or that are required for structures involved in these processes (i.e., somatic cells and stem cells) are all potentially limited by the competing demands on these resources for regeneration from injuries. Some potential trade-offs are not well studied, e.g., effects on the ability to interact with other individuals, but preliminary examples provide numerous and interesting directions for further research across a taxonomically broad group of organisms. Some important cellular resources for these life history processes are provided in Table 6.

3.2 Effects of regeneration on growth

Sponge and coral growth is achieved by the geometrical expansion of the solitary body plan or, in the case of colonial organisms, the re-iterated budding of new modules and their subsequent organization into various colonial architectures. Growth and regeneration might appear to be fundamentally the same processes (BERRILL, 1951; MARTÍNEZ, 2002). However growth rates and patterns are frequently altered while sponges and corals regenerate. The source of regenerative material in the wound milieu is also different from that surrounding new body parts during normal growth (TARDENT, 1963; GOSS, 1992), and in the case of colonial organisms, module ontogeny itself is also influenced by the state of parent modules (WATKINS, 1958). These differences suggest that growth and regeneration may be distinct (but related) processes.

Somatic growth is often reduced in regenerating sponges and corals (BAK, 1983; KOBAYASHI, 1984; LIDDLE and KAY, 1987; GUZMÁN *et al*., 1991; YOSHIOKA and YOSHIOKA, 1991; BRAZEAU and LASKER, 1992; JOHNSON, 1992; GUZMÁN *et al*., 1994; MEESTERS *et al*., 1994; LIRMAN, 2000b; DUCKWORTH, 2003). This may be related to the exhaustion of a limited source of reserve cells. For example, coenenchyme restoration following repeated branch injuries in the gorgonian *Plexaura flexuosa* depresses already depleted cellular resources (LANG DA SILVEIRA and VAN'T HOF, 1977). Greater oxygen uptake and rapid senescence of injured tissues is followed by the generation of reactive oxygen species that could change growth through altered gene expression, e.g., in colonial hydroids (BLACKSTONE, 2000). Significant energy investment may also be directed into the production of anti-oxidants instead of growth (PONCZEK and BLACKSTONE, 2001).

The consequences of regeneration for growth may depend on the severity of disturbance and the overall effects on the rest of the animal (e.g., dislodgment versus fragmentation *sensu* WARD, 1995), and the two processes may not always be traded off (e.g., LESTER and BAK, 1985). Growth rates can even be many times faster in injured than in uninjured sponges (A.M. AYLING, 1981; A.L. AYLING 1983; HOPPE, 1988; TURON *et al*., 1998; DUCKWORTH, 2003) and corals (MARAGOS, 1972; LOYA, 1976b). One possible explanation is that rapid growth after injury evolved to re-establish body plan symmetry (WOOD-JONES, 1907; MARAGOS, 1972). Alternatively, a wound at the growing edge of the sponge or coral upsets normal resource flow among modules so that undamaged tissues near the wound have excess resources to divert into their own compensatory growth, e.g., in bryozoans (HARVELL and HELLING, 1993).

Colony pattern formation (astogeny) can also be altered during regeneration. The morphological plasticity of most sessile colonial animals is of considerable adaptive significance as it allows these organisms to alter module arrangement into various architectures e.g., runners, vines, sheets, mounds, plates and trees in different environments (JACKSON, 1979). In general, injured colonies and those in physically disturbed habitats tend to approximate linear arrays of modules or encrusting mounds instead of tree-shaped forms (WOOD-JONES, 1907; JACKSON, 1979). This change in colony astogeny may be due to altered gastrovascular flow that helps regulate colony pattern formation in cnidarians (DUDGEON and BUSS, 1996; LASKER and SÀNCHEZ, 2002). Occasionally, novel morphs are observed during regeneration e.g., asymmetrical growth in normally symmetrical reef corals (BROWN, 1994). Because genes that regulate colonial development are likely expressed in interstitial cells in some cnidarians (MILLER, 2000), altered morphology could be produced by altered levels of gene expression through interstitial or other reserve cell depletion during regeneration.

3.3 Effects of regeneration on sexual reproduction

Regeneration frequently proceeds at the expense of sexual reproduction in sponges and corals. Regenerating individuals have reduced fecundity or remain sexually infertile, while others may prematurely shed inviable sexually-derived larvae (REISWIG, 1973; TUNNICLIFFE, 1981; WAHLE, 1983b; KOJIS and QUINN, 1985; RICHMOND, 1987;

RINKEVICH and LOYA, 1987; HOPPE, 1988; RINKEVICH and LOYA, 1989; GUZMÁN and HOLST, 1993; VAN VEGHEL and BAK, 1994; SMITH and HUGHES, 1999; LIRMAN, 2000b; OREN *et al*., 2001; HENRY *et al*., 2003; HENRY and KENCHINGTON, 2004). HALL and HUGHES (1996) considered that unusually large but sexually immature corals may represent evidence of altered investments in sexual reproduction owing to the energetic demands imposed by previous injuries. Other factors associated with disturbance could also be significant and co-vary with injury. For example, WARD (1995) noted reduced fecundity in overturned colonies of the scleractinian *Pocillopora damicornis*. She ascribed this reduction to energy limitations imposed by reduced light availability and increased exposure to sedimentation stress in overturned corals rather than to resource demands following regeneration.

In cases where energy does not seem to be limiting, sexual reproduction may be suppressed following regeneration due to the exhaustion of stem cell resources used in gamete production. Gametogenesis proceeds epigenetically from stem cell precursors in most sponges and corals (NIEUWKOOP and SUTASURYA, 1981; TARDENT, 1985), and these are the same cell resources that are also heavily invested in regeneration.

In some cases, no trade-off between regeneration and sexual reproduction is apparent. For example, WAHLE (1983a) suggested that a preset hierarchy of energetic demands, in which sexual reproduction is a high priority, may require the correlated evolution of an independent reserve energy supply for reproduction in gorgonians that cannot be tapped for module regeneration. Alternatively, energy for regeneration may not be limiting unless the wound is sufficiently large or colonies are chronically damaged

(WAHLE, 1983a). Furthermore, once an individual has irreversibly committed to a reproductive phase, sexual reproduction can occur at the expense of all other processes, including regeneration (CAMPBELL, 1974), although studies of an analogous trade-off (involving vertebrate immune responses) suggest that the trade-offs between somatic defense and reproduction may only be evident when the individual must simultaneously supply resources to both processes (BONNEAUD *et al*., 2003), i.e., during the sexual reproduction season.

Locally enhanced sexual reproduction within an individual sponge or coral may occur following injury. For example, SOONG and LANG (1992) demonstrated that more polyps were fertile closest to experimentally damaged colony margins in the corals *Porites astreoides*, *Siderastrea radians* and *S. siderea.* SOONG and LANG explain this small-scale increase in fecundity by the localized enhancement of resource movement into adjacent healthy polyps (resources that would have otherwise have gone into the previously uninjured polyps). Such localized enhancement probably does not compensate for overall reduced allocation to reproduction for the colony as a whole, and may occur in some modules as a by-product of resource movement among modules.

3.4 Effects of regeneration on outcome of encounters with other organisms

Sponges and corals often inhabit space-limited ecosystems such as shallow-water coral reefs. These organisms have evolved elaborate mechanisms to cope with encounters between individuals, e.g., anti-predator defenses, competitive strategies, co-operative fusion with conspecifics and antagonistic tissue responses that could require significant resource investment (RINKEVICH and LOYA, 1985; ROMANO, 1990; FRANK and

RINKEVICH, 1994; TANNER, 1995, 1997). Therefore, to the extent that these interactions require resources also used for regeneration, it is reasonable to expect impaired species interactions during and after regeneration. This general scenario has not been rigorously tested, but both energy and stem cells are shared between these processes.

3.4.1 Anti-predator defenses

The effects of chronic regeneration on defensive capabilities are not well studied. However, defenses are frequently impaired after damage. Long sclerites are naturally produced in colony tips of the gorgonian *Briareum asbestinum*, but shorter and possibly less effective sclerites were noted in amputated tips (WEST, 1997), suggesting reduced anti-predator capabilities in regenerating fragments. BYTHELL *et al*. (1993) noted moderately more intense grazing by parrotfish on damaged colonies of *Montastraea annularis*, *Porites astreoides* and *Diploria strigosa*, suggesting that defensive capabilities in these scleractinians were probably reduced. It is not known whether depletion of energy stores or cells that produce anti-predator structures such as spicules or nematocyts were responsible for impaired anti-predator defense mechanisms in regenerating colonies, but it is likely that a lack of either resource impairs defense mechanisms in regenerating colonies.

3.4.2 Competitive abilities

Sponges and corals exhibit a diverse and energetically costly (POTTS, 1977; EDMUNDS and SPENCER DAVIES, 1986; ROMANO, 1990; TANNER, 1995, 1997) set of structures and mechanisms to defend growing space against encroaching neighbours. These defenses include overgrowth, sweeper tentacles, nematocysts, allelochemicals, mucus and xenogeneic histocompatibility reactions. These competitive encounters result in the overgrowth and subsequent phagocytosis of the inferior individual, bare zones around individuals, repeated reversals in outcomes or stand-offs associated with inhibited growth but enhanced protection at the periphery of the individual (e.g., JACKSON and BUSS, 1975; KARLSON, 1980; MÜLLER *et al*., 1983; CHORNESKY, 1989; HARVELL and PADILLA, 1990; XING and QIAN, 1999). Few studies have examined whether regeneration affects competitive outcomes in sponges and corals. But if regeneration limits the allocation of resources to competitive processes, then one could expect to observe impaired structural defenses and reduced cell- (e.g., histocompatability, cytotoxicity) and humoral- (e.g., secretion of cytokine-like molecules, antibodies) mediated immune responses.

Several lines of evidence suggest that competitive interactions are impaired during regeneration. First, lesions are rapidly fouled by hydroids, serpulid worms and algae (e.g., BAVESTRELLO AND BOERO, 1986; VAN DER KNAPP 1993; BAVESTRELLO *et al*., 1997) that can overgrow the host (VELIMIROV AND WEINBAUER, 1992), suggesting strong localized inhibition of competitive abilities at the lesion site. Corals with higher degrees of existing injuries are also more susceptible to the activities of boring organisms such as sponges, polychaetes, sipunculids, echinoids and barnacles (HUTCHINGS, 1986; PEYROT-CLAUSADE and BRUNEL, 1990; SCOFFIN *et al*., 1997) and to indirect predation by fish attracted to pre-existing boring organisms exposed by wounding (STORR, 1976).

Second, tumours and abnormal skeletal growth sometimes follow lesion fouling in scleractinians (RANDALL and ELDREDGE, 1976; KAUFMAN, 1981; BAK, 1983; LOYA *et* *al*., 1984; PETERS *et al*., 1986; WIELGUS *et al*., 2002) and octocorals (GOLDBERG and MAKEMSON, 1981; BENAYAHU, 1998). These malformations are often initiated by mechanical damage to corals caused by cyclones, corallivores and boring organisms, followed by algal invasion and subsequent encapsulation of the damaged tissue and organisms by skeletogenic calcium deposition (WOOD-JONES, 1907; THEODOR, 1964, 1967; KAUFMAN, 1981; PETERS, 1984) and amoebocyte proliferation (GOLDBERG and MAKEMSON, 1981). Tumours potentially affect coral survival by reducing reproductive potential and by increasing susceptibility to further damage and disease through the death of coral polyps and increasing coral structural fragility (PETERS *et al*., 1986).

Third, regeneration appears to limit the ability of corals to defend themselves against pathogenic endolithic fungi by restricting materials available to skeletogenic processes that would otherwise result in the encapsulation of the invading fungi (BENTIS *et al*., 2000). Restricted resource availability may also explain the particular virulence of disease in regenerating coral fragments versus regenerating (attached) coral colonies (BAK and CRIENS, 1981).

With respect to whole colony or individual competitive abilities, damaged colonies of the scleractinian coral *Montastraea cavernosa* were more rapidly overgrown by the encrusting sponge *Rhaphidophlus venosus* (AERTS, 2000). Grazing by herbivorous reef fish appeared to prevent the overgrowth of *Porites cylindrica* colonies by foliose brown algae, even permitting the regeneration of coral tissue over dead skeleton (JOMPA and MCCOOK, 2002). Mechanical abrasion and smothering are both potential mechanisms that reduce coral growth and regeneration processes by causing coral polyp retraction and therefore limiting coral access to energy and metabolites (TANNER, 1995; RIVER and EDMUNDS, 2001).

Many competitive interactions are hierarchical: some individuals are competitively superior to others (JACKSON, 1979). However, some reversals in competitive outcomes that have been explained by intrinsic or environmental conditions (CHORNESKY, 1989) may actually reflect changes in resource availability following regeneration from wounds caused by competitive interactions. The competitively superior octocoral *Clavularia inflata* was unable to overgrow the competitively inferior scleractinian *Acropora longicyathus*: regeneration in *C. inflata* following high predation damage by reef fish on mid-shelf reefs seemed to impair its ability to compete (ALINO *et al*., 1992). Damaged corals may experience long-term overgrowth and competitive exclusion by sponges (AERTS, 2000) or by larger, more energy-rich conspecific colonies (ZILBERBERG and EDMUNDS, 2001). Patterns of resource allocation could be measured in regenerating colonies under competition as they have been for bleached *Oculina patagonica* corals, which exhibited significantly reduced competitive ability (FINE and LOYA 2003).

Conversely, the evolution of rapid regeneration combined with continuous asexual fragmentation to escape competitive encounters can result in frequently damaged individuals actually overgrowing otherwise competitively superior species. For example, explants of the bioeroding sponge *Cliona orientalis* initially showed some signs of deterioration when grafted to various coral species, but eventually proliferated by lateral growth and fragmentation to overgrow the host coral substrate (SCHÖNBERG and WILKINSON, 2001).

3.4.3 Self- and non-self recognition abilities

The ability of many marine invertebrates to distinguish among different classes of conspecifics (allorecognition) is well documented, particularly in hydractiniid hydroids and botryllid tunicates. In laboratory mating experiments, allorecognition behaviour segregates as one or more loci that are so polymorphic that only close kin (siblings, or parents and offspring) are likely to share alleles in common by descent. Allele sharing typically regulates competitive interactions between growing colonies such that closely related colonies fuse (or at least moderate their competitive responses) while distantly related colonies avoid fusion or engage in aggressive cytotoxic rejection behaviour (GROSBERG and QUINN, 1988; GROSBERG *et al*., 1996).

Besides the potential for deficiencies in energy reserves that fuel allorecognition processes, resource limitation following regeneration may impair allorecognition capabilities by restricting the production of cell-surface markers and cell adhesion molecules (e.g. proteoglycans, polysaccharides) that regulate histocompatibility in colonial marine invertebrates (e.g., COOMBE and PARISH, 1988; FERNÀNDEZ-BUSQUETS and BURGER, 1999; MÜLLER *et al*., 1999; SCHMID *et al*., 1999).

Reduced resources may also limit the production of structures associated with allorecognition such as fibrous contact barriers and nemtocyst-laden hyperplastic stolons. For example, experimental removal of interstitial cells prevented the formation of hyperplastic stolons (but did not improve histocompatibility) in rejection interactions between colonies of the hydroid *Hydractinia echinata* (BUSS *et al*., 1984). Allocation of interstitial cells to regeneration thus directly impairs the aggressive attack response in hydractiniid hydroids (BUSS and GROSBERG, 1990).

Regeneration may also restrict the availability of various cell types used in cell- and humoral-mediated alloimmunity responses such as archaeocytes, phagocytes, amoebocytes and lymphocyte-like cells (KOYAMA and WATANABE, 1982; VAN DE VYVER and BUSCEMA, 1985; YOSHINO, 1986; discussed in AMANO, 1990 and references therein; OLANO, 1993; COOPER and PARRINELLO, 2001; PARRINELLO *et al*., 2001). Thus, reduced cytotoxic and immunoglobulin antibody production functions may follow injury and regeneration.

These cell types are also precursors for somatic structures associated with fusion and subsequent cooperative behaviour between compatible colonies (e.g, formation of a shared choanoderm, gastrovascular system, skeleton). Colony responses to autogeneic tissue contact in the gorgonian *Swiftia exserta* were similar to those involved in tissue regeneration: both involved surface recognition, fusion between colonies and rearrangment of cells (OLANO, 1993). Thus, fusion between two otherwise histocompatible colonies may be impaired if shared resources are depleted following regeneration.

Alloimmune memory or “anamnesis” in marine invertebrates may also be affected if resources are limited by regeneration. If anamnesis is limited by somatic or stem cell availability because former allogeneic interactions have exhausted these cell resources (FRANK and RINKEVICH, 2001), then a newly regenerated sponge or coral could exhibit reduced alloimmune memory.

These diverse and scattered studies suggest that regeneration processes and allorecognition processes may compete with each other for resources used for structural or chemical defense and for cooperative fusion. Such competition could interfere with the ability to remember previous allorecognition interactions and their outcomes.

Extensive studies of the genetic basis of intraspecific allorecognition behaviour (e.g., GROSBERG and QUINN, 1988; GROSBERG *et al*., 1996) have taken advantage of the utility of clonal explants created by surgical techniques. The effects of regeneration from surgery on subsequent behavioural interactions with conspecifics have not been fully explored. Partial mortality may not alter allorecognition behaviour in species with excellent regeneration capacities such as the alcyonacean *Parerythropodium fulvum fulvum* (FRANK *et al*., 1996) in which fast regeneration is likely related to rapid, widespread intracolonial active transport of resources to injured areas (GATEÑO *et al*., 1998).

Future studies specifically designed to test the strength of associations between regeneration and allorecognition should consider both the adaptive context and the ecological conditions of interacting colonies. Species that are adapted to frequent disturbance and chronic regeneration may also have mechanisms for mitigating trade-offs between frequent regeneration demands and allorecognition interactions. Such trade-offs could be studied in species that have or lack adaptations for dealing with chronic disturbance and frequent demands on resources for regeneration. Similarly, the outcome of allorecognition interactions may depend on environmental circumstances (CHADWICK-FURMAN and WEISSMAN, 1995a, b). Reversals in these outcomes could affect the apparent strength of trade-offs between regeneration and allorecognition processes. A combination of laboratory and field studies might be required to measure the potential ecological and evolutionary significance of such trade-offs.

4 Community and ecosystem-level consequences of regeneration

The strong evidence for limited regeneration (section 2) combined with life history impairment during repairs (section 3) allow us to extrapolate ecological consequences of regeneration at the level of an individual sponge or coral to larger-scale community and ecosystem settings.

Impaired somatic growth of sponges and corals during regeneration should reduce the biomass of these animals. Delayed or reduced growth of these animals may indirectly increase biomass of others e.g., fouling and boring organisms that take advantage of space or other resources vacated by chronically regenerating corals and sponges. These changes may in turn alter biomass, densities and species composition of predators, commensals, parasites or pathogens that have specialized interactions with particular components of a community. Abnormally high growth rates in some regenerating individuals, e.g., some sponges, could have longer-term consequences for the persistence of these taxa and the communities they are associated with, as resources are exhausted after regeneration and unavailable for reproduction or interactions with other organisms. Reduced accretion of skeletal frameworks of habitat-forming sponges and corals could alter rates of e.g., reef growth and erosion. Thus, ecological consequences of impaired somatic growth during regeneration could alter the overall biodiversity and persistence of some marine ecosystems.

The strong tendency for sponges and corals to suppress sexual reproduction in favour of regeneration reduces the fecundity of populations of these animals, a character which may be directly linked to reduced recruitment of propagules (HUGHES *et al*., 2000). Recruitment of sexually-derived propagules is critical to community persistence in sponge and coral populations (e.g., CONNELL *et al*., 1997; CROPPER and DIRESTA, 1999; HUGHES *et al*., 2000) as well as to maintaining genetic diversity that could buffer these taxa from localized extinction events. Without sexual recruitment, regenerating sponges and corals may be locally extirpated over time, shifting communities to new demographic and taxonomic configurations, altering any biotic associations with these taxa, and ultimately altering some marine ecosystems.

Altered biotic associations and subsequent ecosystem changes are also direct consequences of impaired abilities to interact with other organisms following regeneration in individual sponges and corals. Densities of predators are at least temporarily increased during regeneration, competitive outcomes are sometimes reversed and fouling organisms quickly invade and can smother the damaged animal. Impaired histocompatibility and fusion processes during regeneration could also lead to altered demographics if such competitive or cooperative interactions between conspecifics have important effects on individual reproductive success.

Regeneration helps to ensure that an individual sponge or coral will survive. This life history phenomenon must therefore be considered as an important influence in the long-term persistence of sponge and coral populations, with important consequences for the structure and function of higher levels of biological assembly including the total biodiversity in marine ecosystems.

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Table 1: Quantitative regeneration studies of sponges and corals from published sources.

Species	**Wound size**	**Daily regeneration rate**	**Reference**
Sponges			
Agelas clathrodes	100-300mm^2	3.6-12.1mm^2	Hoppe, 1988
Anchinoe sp. (yellow morph)	layer cleared	0.4mm^2/mm border	Ayling, 1981
Anchinoe sp.	1000mm^2	3.7mm^2	Ayling, 1983
Aplysilla rosea	1000mm^2	6.2mm^2	Ayling, 1983
Chelonaplysilla sp.	1000mm^2	4.1mm^2	Ayling, 1983
Chondropsis sp.	1000mm^2	5.7mm^2	Ayling, 1983
Cliona celata	250mm^2	1.1-1.8mm^2	Bell, 2002
Crambe crambe	450mm^2	8.6-10.0mm^2	Turon *et al.*, 1998
Eurypon sp.	1000mm^2	0.9mm^2	Ayling, 1983
Hymedesmia sp. (orange morph)	1000mm^2	0.5mm^2	Ayling, 1983
Hymedesmia sp. (red morph)	layer cleared	0.3mm^2/mm border	Ayling, 1981
Ircinia strobilina	100-300mm^2	12.0-41.1mm^2	Hoppe, 1988
Microciona sp.	layer cleared	0.2 mm^2/mm border	Ayling, 1981
Microciona sp.	10-600mm^2	0-7.5mm^2	Ayling 1983
Neofibularia nolitangere	100-300mm^2	4.5-13.6mm^2	Hoppe, 1988
Stylopus sp. (pink morph)	1000mm^2	7.0mm^2	Ayling, 1983
Stylopus sp.	layer cleared	0.1-0.5mm^2/mm border	Ayling, 1981
Stylopus sp.	10-4200mm^2	0-270.0mm^2	Ayling, 1983
Tedania sp. (crimson morph)	12.6mm^2	no regeneration	Jackson and Palumbi, 1979
Tedania sp. (orange morph)	layer cleared	0.1mm^2/mm border	Ayling, 1981
Tedania sp. (orange morph)	1000mm^2	4.2mm^2	Ayling, 1983
Tedania sp. (orange morph sp. 1)	12.6mm^2	4.2-12.6mm^2	Jackson and Palumbi, 1979
Tedania sp. (orange morph sp. 2)	12.6mm^2	0.5mm^2	Jackson and Palumbi, 1979
Tedania sp. (pink-brown morph)	12.6mm^2	1.8mm^2	Jackson and Palumbi, 1979
Tedania sp. (pink-red morph)	12.6mm^2	1.3-1.6mm^2	Jackson and Palumbi, 1979
Tedania sp. (red-orange morph)	12.6mm^2	6.3mm^2	Jackson and Palumbi, 1979
Tedania sp. (white morph)	12.6mm^2	0.4-0.6mm^2	Jackson and Palumbi, 1979

Alcyoniids			
Gersemia rubiformis	5 polyps	0.2 polyps	Henry *et al.*, 2003
Gorgonians			
Eunicea mammosa	20mm	6.0-7.8mm	Wahle, 1983a
Paramuricea clavata	500mm	0.2mm	Bavestrello *et al.*, 1997
Plexaura homomalla	20mm	6.8-8.6mm	Wahle, 1983a
Plexaurella dichotoma	20mm	6.7-10.6mm	Wahle, 1983a
Plexaurella flexuosa	40mm	1.4mm	Lang da Silveira and Van't Hof, 1977
Plexaurella fusifera	4.5mm	0.3-0.4mm	Meszaros and Bigger, 1999
Scleractinians			
Acropora cytherea	400mm^2	5.4-5.6mm^2	Hall, 1997
Acropora formosa	not given	0-0.2mm	Stephenson and Stephenson, 1933
Acropora gemmifera	not given	0-0.1mm	Stephenson and Stephenson, 1933
Acropora hebes	10mm	0.4mm	Isa, 1987
Acropora hyacinthus	400mm^2	2.8-4.2mm^2	Hall, 1997
Acropora palifera	400mm^2	no regeneration	Hall, 1997
Acropora palmata	fragment	0.1-0.5mm	Rogers *et al.*, 1982
Acropora palmata	100mm^2	1.7mm^2	Bak, 1983
Acropora palmata	100mm^2	3.6-3.7mm^2	Meesters *et al.*, 1992
Acropora palmata	79mm^2	16.0-25.0mm^2	Meesters and Bak, 1995
Acropora palmata	>=3000mm^2	3.6-10.7mm^2	Lirman, 2000a
Acropora polymorpha	not given	0.2-0.3mm	Stephenson and Stephenson, 1933
Acropora quelchi	not given	0-0.1mm	Stephenson and Stephenson, 1933
Acropora robusta	400mm^2	5.4mm^2	Hall, 1997
Acropora sp. 1	not given	0-0.1mm	Stephenson and Stephenson, 1933
Acropora sp. 2	not given	0-0.1mm	Stephenson and Stephenson, 1933
Agaricia agaricites (forma *purpurea*)	100-500mm^2	0.7-2.2mm^2	Bak and Steward-van Es, 1980
Agaricia agaricites	100-500mm^2	0.3-0.5mm^2	Bak *et al.*, 1977
Astrangia lajollaensis	layer cleared	0-0.1 corallite	Fadlallah, 1982
Diploria clivosa	570mm^2	0.6-0.8mm^2	Guzmán *et al.*, 1994
Diploria strigosa	100mm^2	1.2-1.5mm^2	Meesters *et al.*, 1992
Diploria strigosa	570mm^2	0.5-0.8mm^2	Guzmán *et al.*, 1994
Favia favus	110-550mm^2	1.6-7.8mm^2	Oren *et al.*, 1997a
Favia favus	87-274mm^2	1.3-3.5mm^2	Oren *et al.*, 2001
Goniastrea retiformis	400mm^2	0.6-1.1mm^2	Hall, 1997
Meandrina meandrites	79mm^2	1.3-3.4mm^2	Meesters and Bak, 1993
Montastraea annularis (bumpy morph)	160mm^2	15.4-18.7mm^2	Van Veghel and Bak, 1994
Montastraea annularis (columnar morph)	160mm^2	0.7mm^2	Van Veghel and Bak, 1994
Montastraea annularis (columnar morph)	83-406mm^2	3.7-11.2mm^2	Meesters *et al.*, 1997a
Montastraea annularis (massive morph)	79mm^2	1.6mm^2	Meesters and Bak, 1993
Montastraea annularis (massive morph)	160mm^2	10.5-13.9mm^2	Van Veghel and Bak, 1994
Montastraea annularis	100-500mm^2	0.75-0.94mm^2	Bak *et al.*, 1977
Montastraea annularis	100mm^2	1.9mm^2	Meesters *et al.*, 1992
Montastraea annularis	170mm^2	4.1mm^2	Meesters *et al.*, 1994
Montastraea annularis	227mm^2	2.9-3.6mm^2	Mascarelli and Bunkley-Williams, 1999
Oculina patagonica	200mm^2	2.8-4.4mm^2	Fine *et al.*, 2002
Pocillopora damicornis	400mm^2	3.7-4.0mm^2	Hall, 1997
Porites astreoides	13.7-220mm^2	1.5-5.7mm^2	Nagelkerken and Bak, 1998

(brown morph)			
Porites astreoides (green morph)	15.1mm^2	1.7mm^2	Nagelkerken *et al*., 1999
Porites astreoides (hemisphaerical morph)	79mm^2	5.4-9.0mm^2	Meesters and Bak, 1993
Porites astreoides	100-500mm^2	0.9-2.1mm^2	Bak and Steward-van Es, 1980
Porites astreoides	100mm^2	2.7-3.1mm^2	Meesters *et al*., 1992
Porites astreoides	570mm^2	0.3-0.8mm^2	Guzmán *et al*., 1994
Porites astreoides	<=80mm	0.02-0.03mm	Ruesink, 1997
Porites australiensis	400mm^2	1.7-2.5mm^2	Hall, 1997
Porites lichen	400mm^2	0.6-0.9mm^2	Hall, 1997
Porites lobata	15-1310mm^2	0.4-4.1mm^2	van Woesik, 1998
Porites lutea	15-1310mm^2	0.4-18.3mm^2	van Woesik, 1998
Porites mayeri	400mm^2	0-1.4mm^2	Hall, 1997
Siderastrea siderea	100mm^2	0.6-1.2mm^2	Meesters *et al*., 1992
Siderastrea siderea	570mm^2	0.1-0.7mm^2	Guzmán *et al*., 1994
Siderastrea siderea	<=80mm	0.01 mm	Ruesink, 1997
Stephanocoenia michelinii (encrusting morph)	15.1mm^2	0.7mm^2	Nagelkerken and Bak, 1998
Stephanocoenia michelinii (massive morph)	16.1mm^2	0.8mm^2	Nagelkerken and Bak, 1998
Stephanocoenia michelinii (massive morph)	200mm^2	6.0mm^2	Nagelkerken *et al*., 1999

Table 2: Ranked daily regeneration rates of sponges and corals with wounds 1 - 200mm^2.

Species	**Maximum percentage of wound regenerated daily (%)**	**Group**
Tedania sp. 1 (orange morph)	100.0	warm-water sponge
Tedania sp. 3 (red-orange morph)	50.0	warm-water sponge
Acropora palmata	31.7	warm-water scleractinian
Meandrina meandrites	23.7	warm-water scleractinian
Ircinia strobilina	19.2	warm-water sponge
Montastraea annularis (massive morph)	17.6	warm-water scleractinian
Tedania sp. 4 (pink-brown morph)	14.3	warm-water sponge
Tedania sp. 5 (pink-red morph)	12.7	warm-water sponge
Porites astreoides (hemisphaerical morph)	11.4	warm-water scleractinian
Porites astreoides (green morph)	11.3	warm-water scleractinian
Porites astreoides (brown morph)	11.0	warm-water scleractinian
Montastraea annularis (columnar morph)	8.1	warm-water scleractinian
Neofibularia nolitangere	7.4	warm-water sponge
Tedania sp. 6 (white morph)	4.8	warm-water sponge
Stephanocoenia michelinii (encrusting morph)	4.6	warm-water scleractinian
Tedania sp. 2 (orange morph 2)	4.0	warm-water sponge
Agelas clathrodes	3.9	warm-water sponge
Microciona sp.	3.5	cold-water sponge
Porites astreoides	3.1	warm-water scleractinian
Stephanocoenia michelinii (massive morph)	3.0	warm-water scleractinian
Stylopus sp.	2.9	cold-water sponge
Montastraea annularis	2.4	warm-water scleractinian
Oculina patagonica	2.2	warm-water scleractinian
Favia favus	1.7	warm-water scleractinian
Diploria strigosa	1.5	warm-water scleractinian
Siderastrea siderea	1.2	warm-water scleractinian
Montastraea annularis (bumpy morph)	1.0	warm-water scleractinian
Agaricia agaricites (forma *purpurea*)	0.9	warm-water scleractinian
Porites lobata	0.9	warm-water scleractinian
Porites lutea	0.9	warm-water scleractinian
Agaricia agaricites	0.5	warm-water scleractinian
Tedania sp. 7 (crimson morph)	0.0	warm-water sponge

Table 3: Ranked daily regeneration rates of sponges and corals with wounds 201 - 4200mm^2.

Species	**Maximum percentage of wound regenerated daily (%)**	**Group**
Ircinia strobilina	13.70	warm-water sponge
Stylopus sp.	6.07	cold-water sponge
Montastraea annularis (columnar morph)	4.74	warm-water scleractinian
Neofibularia nolitangere	4.53	warm-water sponge
Agelas clathrodes	4.03	warm-water sponge
Porites astreoides (brown morph)	2.59	warm-water scleractinian
Crambe crambe	2.20	warm-water sponge
Favia favus	1.62	warm-water scleractinian
Montastraea annularis	1.59	warm-water scleractinian
Microciona sp.	1.43	warm-water sponge
Acropora robusta	1.34	warm-water scleractinian
Acropora hyacinthus	1.06	warm-water scleractinian
Pocillopora damicornis	1.02	warm-water scleractinian
Stylopus sp. (pink morph)	0.70	cold-water sponge
Cliona celata	0.65	cold-water sponge
Aplysilla rosea	0.62	cold-water sponge
Chondropsis sp.	0.57	cold-water sponge
Agaricia agaricites (forma *purpurea*)	0.44	warm-water scleractinian
Porites astreoides	0.43	warm-water sponge
Tedania sp. (orange morph)	0.42	cold-water sponge
Chelonaplysilla sp.	0.41	cold-water sponge
Anchinoe sp.	0.37	cold-water sponge
Acropora palmata	0.36	warm-water scleractinian
Porites mayeri	0.35	warm-water scleractinian
Porites lobata	0.31	warm-water scleractinian
Porites lutea	0.31	warm-water scleractinian
Goniastrea retiformis	0.28	warm-water scleractinian
Porites lichen	0.21	warm-water scleractinian
Diploria clivosa	0.14	warm-water scleractinian
Diploria strigosa	0.14	warm-water scleractinian
Siderastrea siderea	0.12	warm-water scleractinian
Eurypon sp.	0.09	cold-water sponge
Hymedesmia sp. (orange morph)	0.05	cold-water sponge
Agaricia agaricites	0.01	warm-water scleractinian
Acropora palifera	0.00	warm-water scleractinian

Table 4: Lesion properties found on sponges and corals under natural conditions.

Lesions property **Size**	**Source**	**Habitat, geographic region**	**Reference**
10-240mm^2	predation	coral reef, Barbados	OTT and LEWIS, 1972
2410-7510mm^2	various	coral reef, Brazil (São Paulo)	ACOSTA *et al.*, 2001
100-10700mm^2	fish, divers, bottom-associated processes	coral reef, Curaçao	MEESTERS *et al.*, 1996b
50-300mm^2 (type I lesion), 200-6000 mm^2 (type II lesion)	fish, divers, bottom-associated processes	coral reef, Curaçao	MEESTERS *et al.*, 1997b
0-50% damage	various	coral reef, Curaçao	BAK and MEESTERS, 1998
0->2000mm^2	storms, boats	coral reef, Florida USA	LIRMAN, 2000b
8-10% with >66% damage, 12-20% with 33-66% damage, 13-50% with <33% damage	predation	coral reef, GBR	DONE, 1987
40% with >66% damage, 15% with 33-66% damage, 20% with <33% damage	predation	coral reef, GBR	DONE, 1988
0-45% with <33% damage, 0-47% with 33-66% damage, 0-43% with >66% damage	predation	coral reef, GBR	CAMERON *et al.*, 1991
53% fragments <50mm long	divers	coral reef, GBR	ROUPHAEL and INGLIS, 1995
3-27% corals with >20% damage, 31% sponges with >33% damage	hurricane	coral reef, Jamaica	WOODLEY *et al.*, 1981
11-75% with 0-25% damage, 7-85% with 25-50% damage, 0-18% with 50-75% damage, 0-22% with 75-100% damage	various	coral reef, Jamaica	HUGHES and JACKSON, 1985
100mm^2	predation	coral reef, Jamaica	KAUFMAN, 1981
0-344mm^3	predation	coral reef, Panama (Atlantic)	LASKER *et al.*, 1988
0-5590mm^3	predation	coral reef, Panama (Atlantic)	VREELAND and LASKER, 1989
2-3% with <50% damage, 2-3% with >50% damage	various	coral reef, Red Sea	RIEGL and VELIMIROV, 1991

at least 1% of coral surface	predation	coral reef, Red Sea	Schuhmacher, 1992
11-24% with lesions <100mm^2, 23-30% with lesions 100-200mm^2, 17-21% with lesions 200-300mm^2, 31-42% with lesions >300 mm^2	various	coral reef, Panama (Atlantic)	Ruesink, 1997
<25mm^2	predation	coral reef, Red Sea	Oren *et al.*, 1998
<100-10000mm^2	predation, storms	coral reef, Red Sea	Oren *et al.*, 2001
500-4500mm^2	solar damage	coral reef, Thailand	Brown, 1994
1200-4000mm^2	storms	coral reef, USVI	Rogers *et al.*, 1982
600-910mm^2	predation	coral reef, USVI	Witman, 1988
16-31% dead area	various	coral reef, USVI	Bythell *et al.*, 1993

Shape	**Source**	**Habitat, geographic region**	**Reference**
lesions on side branches rectangular of uniform depth	predation	coral reef, Puerto Rico	Vreeland and Lasker, 1989
small lesions circular, larger lesions circular to narrow	predation, storms	coral reef, Red Sea	Oren *et al.*, 2001
hemisphaerical colonies have circular to elliptical wounds, annular colonies have elongate wounds	solar damage	coral reef, Thaliand	Brown, 1994

Orientation	**Source**	**Habitat, geographic region**	**Reference**
14% sponges turned over, 55% gorgonians broken at bases or pulled out of seafloor	bottom-trawling	continental shelf, Gulf of Alaska	Freese *et al.*, 1999
87% broken at base	boring sponges	coral reef, Jamaica	Tunnicliffe, 1979
0-98% broken at base, 0-1% broken at branches, 0-2% broken at tips	waves	coral reef, Jamaica	Tunnicliffe, 1981
0-2% detached, 60-95% at distal tips	abrasion, predation	coral reef, Jamaica	Wahle, 1985
40-71% detached, 22-46% broken at base	detachment, abrasion, overgrowth	coral reef, Puerto Rico	Yoshioka and Yoshioka, 1991
86% at colony margins	predation	coral reef, Red Sea	Oren *et al.*, 1998

GBR = Great Barrier Reef; USVI = United States Virgin Island

Table 5: Summary of resources required for sealing, repair and regeneration in sponges, scleractinian and alcyonacean corals.

Group	Molecules	Cell types	Energy sources
Sponges[1]	spongin (in the Demospongiae)	archaeocytes; choanocytes*; collencytes; gray cells; pinacocytes; sclerocytes; spherulous cells	fueled by gray cells, cyanobacterial symbionts or other sources
Scleractinians[2]	mineralized crystals (in species with calcified skeletons); peptide growth factors*	amoebocytes; cells from coenochyme, epithelium, gastroderm, mesenterial filaments, mesoglea; globular granular cells; i-cells; lymphocyte-like cells; "transitional" cells; sperm cells	lipids, proteins, glucose from zooxanthellae (in hermatypic corals); prey; dissolved free amino acids
Alcyonaceans[3]	mineralized crystals (in species with calcified skeletons); peptide growth factors*	amoebocytes; cells from coenochyme, epithelium, gastroderm, mesenterial filaments, mesoglea; globular granular cells; i-cells; lymphocyte-like cells; "transitional" cells; sperm cells	lipids, proteins, glucose from zooxanthellae (in hermatypic corals); prey; dissolved free amino acids

* proposed

[1] (WILSON, 1910; STOLTE, 1935; KOROTKOVA, 1970; HARRISON, 1972; BOURY-ESNAULT, 1976; THOMPSON *et al.*, 1983; SIMPSON, 1984; HOPPE, 1988; LEYS and MACKIE, 1994; HILL and HILL, 2002)

[2] (STOLTE, 1935; MUSCATINE and CERNICHIARIA, 1969; PEARSE and MUSCATINE, 1971; FISHELSON, 1973; RINKEVICH and LOYA, 1983; ISA, 1987; STIMSON, 1987; HAYES and BUSH, 1990; FERRIER, 1991; MEESTERS *et al.*, 1997a; OREN *et al.*, 1997b)

[3] (LANG DA SILVEIRA and VAN'T HOF, 1977; OLANO, 1993; MESZAROS and BIGGER, 1999; BEN-DAVID-ZASLOW and BENAYAHU, 2000)

Table 6: Summary of potentially limiting cell types shared between regeneration (Table 4) and other life history processes in sponges and corals.

Process	Sponges	Scleractinians	Alcyonaceans
Sexual reproduction	archaeocytes; choanocytes*; pinacocytes	amoebocytes	amoebocytes
Growth	archaeocytes; choanocytes; pinacocytes	amoebocytes	amoebocytes
Anti-predator defenses	sclerocytes; archaeocytes	amoebocytes; cnidoblasts	amoebocytes; cnidoblasts
Competition	sclerocytes; archaeocytes	amoebocytes; cnidoblasts	amoebocytes; cnidoblasts
Self- and non-self recognition abilities	sclerocytes; archaeocytes; collencytes	amoebocytes; cnidoblasts	amoebocytes; cnidoblasts

* proposed

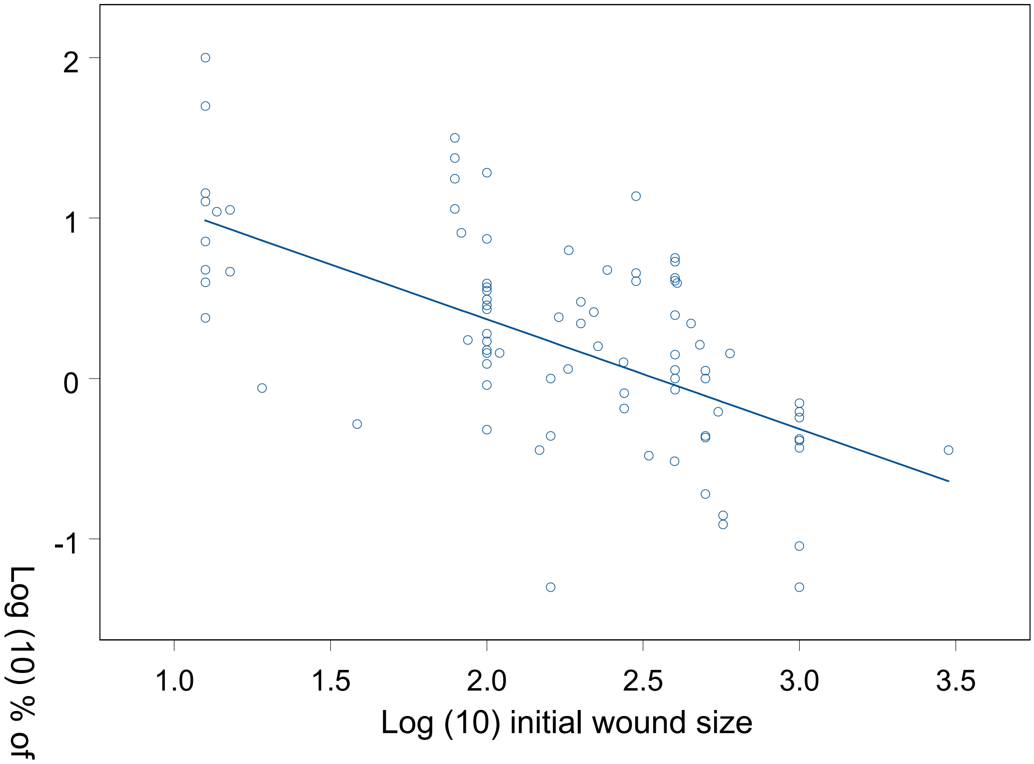

Figure 1: Linear relationship between amount of tissue regenerated per day and initial wound size based on 91 standardized published regeneration rates of colonial epifauna ($R^2 = 0.35$).