Multi-scale spatial patterning of corals and their symbionts

by

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(Under the Direction of Craig W. Osenberg)

Abstract

Habitat plays a critical role in community dynamics, yet is usually considered a static, rather than dynamic component of a system. Habitat dynamics (including interactions with habitat occupants) are especially relevant when the habitat is living and exhibits variation on a similar time scale as its occupants. Corals and their symbionts are one system where habitat dynamics are relevant. Symbiont settlement processes, movement, and interactions with coral all influence the development of coral colonies and the spatial patterns of colonies in the landscape. These patterns then feed back to affect the dynamics of the symbionts. This dissertation addresses the relationship between corals and their symbionts at both an individual and population level to determine unknown effects of symbionts upon host corals and explore long-term dynamics of the coral and symbiont due to feedbacks within the system through a combination of laboratory, field, and modeling studies.

I first explored the spatial distributions of two corallivorous snails, documenting aggregations at multiple spatial scales. Additionally, I explored the role of chemical cues in creating aggregations, and the effect of the aggregations on coral growth. I also tested the effects of the spatial distribution of damage on the response of the coral, demonstrating that the distance between coral lesions affects healing rate, linear extension of the coral, and coral morphology. At a larger spatial scale, I explored how landscape configuration generates

long-lasting heterogeneity in the density of symbionts across a landscape. The amount of heterogeneity depends on landscape configuration, and the persistence of this heterogeneity through time depends on factors such as post-settlement density-dependent mortality of the occupant. Finally, I examined feedbacks between occupants and corals with models that included interactions between corals and occupants as well as coral dynamics. Collectively these studies demonstrate how interactions between habitat and occupants can play an important role in the spatial distribution of corals and occupants, which can affect not only the morphology of the coral, but also the distribution of corals and occupants across the landscape.

INDEX WORDS: spatial pattern, coral reefs, dynamic habitat, symbionts, corallivory

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Introduction

1.1 Overview

1.1.1 Spatial heterogeneity and species interactions

Spatial heterogeneity plays an important role in species interactions. For example, spatial heterogeneity affects predator-prey dynamics. In some cases, heterogeneity stabilizes predator-prey systems (Huffaker, 1958; Hastings, 1978), while in others it is actually a destabilizing force (Kareiva, 1987). Spatial heterogeneity also facilitates competitive coexistence (reviewed in Amarasekare (2003)) and affects the distribution of and competition for mutualists (e.g. Addicott (1978)). It also plays an important role in maintaining biodiversity by contributing to coexistance among multiple species, such as in the case of two obligate mutualists and an exploiter (e.g. plant, pollinating seed parasite, and non-pollinating seed parasite), that only coexist in a spatially heterogeneous environment (Wilson *et al.*, 2003). Heterogeneity also stabilizes larger food webs (e.g. species involved with the Baltic cod fishery Lindegren *et al.* (2009)). Thus, spatial heterogeneity is an important factor to include when studying species interactions.

Spatial heterogeneity comes in many forms, including the distribution of habitat occupants, resources in the habitat, and the habitat itself. In many systems, organisms who use the habitat are clustered in space. Sometimes this heterogeneity is the result of predator-

avoidance (Dixson *et al.*, 2012). Many species also exhibit clustering due to conspecific attraction (Sweatman, 1988; Tupper & Boutilier, 1995; Lecchini *et al.*, 2005). Heterogeneous distributions can be due to resource use. If an underlying resource is heterogeneous, the distribution of organisms that use that resource is likely also heterogeneous. For example, ungulates are distributed in clusters across the Serengeti due to variation in nutrients (Seagle & McNaughton, 1992), and some reef fish demonstrate heterogeneous patterns due to varying qualities of corals (Tolimieri, 1995; Holbrook *et al.*, 2000; Wilson & Osenberg, 2002; Shima & Osenberg, 2003).

Many systems have heterogeneous resources. A resource could be structure, where some structures offer more protection than others, or a food source. Plants offer food and shelter to many organisms, and are often distributed heterogeneously in space due to factors such as variation in soil (John *et al.*, 2007) or seed dispersal mechanisms (e.g. Thomson *et al.* (2011)). Additionally, even if a resource itself is fairly homogeneous in space, the quality of the resource can be heterogeneous. For example, some coral patches are higher quality patches with higher rates of survival compared to lower quality patches (Shima & Osenberg, 2003). Finally, resources can be heterogeneous in nutritional value (e.g. spatial variation in nutrients available to grazers, Seagle & McNaughton (1992)).

Spatial heterogeneity is also driven by heterogeneity in habitat. One example of habitat heterogeneity is structural heterogeneity. Structure provides refuges and mediates interactions between predators and prey and includes aspects such as rocks or vegetation that create topography and often serve as refuges for prey. Heterogeneity modifies the interactions between both reef fish (Mikheev *et al.*, 2010) and spiders (Birkhofer *et al.*, 2010) and their prey. In fact, the functional response of predator-prey dynamics between a ground dwelling spider and their prey is dependent on the presence of moss (a structural refuge) (Vucic-Pestic *et al.*, 2010). Refugia can also be spatial, rather than structural, where prey occupy predator-free areas (e.g. Kauffman *et al.* (2007)). Even if an area of the landscape is not structurally complex, movement of predators and prey among various types of habitat can still provide

refuges capable of stabilizing the interaction as just a small difference in demographic rates in spatial refuges stabilizes predator-prey dynamics (Poggiale & Auger, 2004; Goldwyn & Hastings, 2009). Habitat heterogeneity plays an important role in driving heterogeneity in resources and occupants, as well as mediating species interactions.

1.1.2 Dynamic habitat

In many cases, the resource and the habitat are the same. This is particularly true for biogenic habitats (e.g. kelp, corals, trees). In these instances, the habitat is not static, but a dynamical component of the system. These dynamics provide additional complexity to the system because they provide the potential for feedbacks between a habitat and its occupants. These dynamics contribute to the spatial heterogeneity in a system. Habitat is dynamic at two spatial scales. First, habitat is dynamic within a patch. For example, the morphology of the branching coral *Acropora* influences the symbiont community: tightly branched corals provide a greater diversity of refuges and host a more diverse community than corals with more open branches (Vytopil & Willis, 2001). At the landscape level, habitat heterogeneity takes the form of a configuration of patches (e.g. refuges and foraging grounds in Yellowstone National Park, Kauffman *et al.* (2007)). At both scales, dynamic habitat plays an important role in community dynamics by affecting the distribution and quality of habitat in a system.

An integral part of the biogenic habitat dynamics are the effects that habitat occupants have on the habitat. Some occupants are beneficial, while others negatively affect the habitat. Mutualists confer benefits (e.g. nutrients and defense against stressors), while predators, parasites, and grazers have deleterious effects. Some habitats have occupants of both types. For example, the seagrass *Thalassia testudinum* receives nutrients from the suspension feeding mussel *Modiolus americanus* (Peterson & Heck, 2001), but is grazed upon by a variety of invertebrates and fishes (Moran & Bjorndal, 2005). Some of these interactions have a spatial component, and can give rise to a heterogeneous distribution of habitat: e.g. the patches of dead trees created by tussock moths (Maron & Harrison, 1997), which have limited mobility

Figure 1.1: Branching *Pocillopora* coral with resident fish (photo credit: Brockin Inaglory)

and therefore create dense aggregations that subsequently kill these trees.

A habitat is not only affected by its occupants, but it influences their behaviors and dynamics as well. As a result, these systems may have long term dynamics not easily predicted by short-term interactions. For example, as grazers deplete seagrass, they not only deplete their food source, but make themselves more vulnerable to predation by eliminating their refuge. Parasites that focus on certain areas of a host, such as a coral or plant, might alter the within patch heterogeneity via morphology or quality of the host, thereby affecting their own population dynamics.

Therefore, when considering spatial dynamics of any organism and its habitat, it is crucial to include the dynamics of the habitat along with those of the occupying species. Feedbacks among these players potentially affect emerging spatial patterns and heterogeneity, and will provide insight in the dynamics both within and among patches in the landscape.

1.2 Study System

Corals are colonial organisms and provide habitat for many symbionts, making reefs an ideal system to study habitat related feedbacks. Algae live within coral tissue, small fish seek shelter among coral branches, and snails sit atop mounding colonies. In addition to receiving shelter and benefits from their habitat, these organisms affect the host coral. In some cases, these are mutualistic relationships. Endosymbiotic *Symbiodinium* fix carbon for consumption

by coral polyps, fish excrete nitrogen (Holbrook *et al.*, 2008), and small crustaceans protect against sediments and predators (Stewart *et al.*, 2006; McKeon *et al.*, 2012; McKeon & Moore, 2014). Other occupants negatively affect the coral, such as corallivorous snails that consume coral tissue, (Turner, 1994; Zeid *et al.*, 2004), and vermetid gastropods that spread mucus nets over tissue blocking light and nutrients (Shima *et al.*, 2010). Reefs exhibit heterogeneity ranging from the morphology of a colony to the distribution of colonies in the landscape. This variation is likely to arise, in part, from the feedbacks between corals and their symbionts. Therefore, corals and symbionts offer the opportunity to explore the spatial relationships of a dynamic biogenic habitat and its occupants.

1.2.1 Corals

Corals are modular organisms formed of many individual polyps. Polyps host symbiotic algae and consume particles from surrounding water (e.g. Palardy *et al.* (2006)). Individual polyps are connected and share nutrients. Polyps secrete calcium carbonate, leading to changes in coral morphology and the accretion of coral reefs. Corals exhibit variation in morphology between species (such as between branching colonies in Figure 1.1 and mounding colonies in Figure 1.2), as well as within a species (Figure 1.2). The way in which corals grow and the effects of symbionts on their growth likely affects their survival, morphology, and spatial patterning.

Coral growth is affected by a variety of factors. Many of these factors have differential effects that depend on polyp position. For example, light benefits polyps at the top of the colony more than polyps near the base. Water flow brings nutrients to corals, and affects polyps upstream and downstream differently. These effects can lead to variation in coral morphology (Mistr & Bercovici, 2003). Some factors (e.g. herbivores) damage corals in specific spatial patterns, and other factors (e.g. the translocation of nutrients) affect how corals heal and regenerate after damage. This damage and regrowth could further alter coral morphology. Little is known about the specific relationship between damage and morphology,

Figure 1.2: Examples of variation in massive *Porites* morphology

but see Zvuloni *et al.* (2008) and Shima *et al.* (2010) for studies that link coral morphology to vermetid gastropods.

The healing and regrowth of coral tissue after damage is affected by the size and shape of the lesion (Meesters *et al.*, 1997; Van Woesik, 1998; Oren *et al.*, 1997), as well as the depth (Bonaldo *et al.*, 2011). In many corals, polyps located near the damaged area play an important role in regeneration (Oren *et al.*, 1998), yet this varies from a few centimeters to colony-wide integration (Oren *et al.*, 2001) and differs among coral species and the extent of damage, due, in part, to the degree of nutrient translocation (Oren *et al.*, 1997). Growth is reduced after damage (Meesters *et al.*, 1994), although subsequent compensatory growth may eliminate long-term differences in size, especially in branching corals (Jayewardene, 2010). While many studies have manipulated the size, shape, and origin of damage (Henry & Hart, 2005), none have explored the effects of the distance between scars on coral growth and tissue generation. If translocation is localized, then two areas of damage that are in close proximity should regrow more slowly than two areas of damage that are more isolated because a greater number of neighboring polyps will be healthy and can contribute to healing. Differences in skeletal growth resulting from different patterns of damage could play a role

in determining coral morphology.

Spatial patterns of coral are also affected by coral growth at a larger spatial scale. Differential settlement, growth, and survival patterns play an important role in determining the heterogeneity of a coral reef. For example, coral settlement is heterogeneous in Moorea, with some sites and depths receiving more settlers, as well as differences in post-settlement survival due to the presence of grazers (Adjeroud *et al.*, 2007). Additionally, coral survival depends on neighboring corals during outbreaks of predators (Kayal *et al.*, 2011) and structural complexity increases juvenile survival (Brandl *et al.*, 2014). Coral growth and survival is also affected by the symbiont community.

1.2.2 Symbionts

Many symbionts rely on coral for habitat. Corals provide important structural refuges for the reef community, mediating predator-prey dynamics (e.g. Beukers & Jones (1998)) and competition. Both live coral (Coker *et al.*, 2012) and reef complexity (Rogers *et al.*, 2014) are important in maintaining reef fish populations. In addition to reef fish, corals host a variety of symbionts. Scientists with National Geographic's "One Cubic Foot" project found more than 600 individuals within a cubic foot quadrat in Moorea, French Polynesia (Liittschwager, 2012).

Some of these symbionts are beneficial for the coral. For example, guard crabs (*Trapezia spp.*) and *Alpheus* shrimp defend against coral predators such as corallivorous snails (*Drupella*) (McKeon & Moore, 2014), pincushion stars (*Culcita*) (McKeon *et al.*, 2012), and the crown of thorns starfish (*Acanthaster planci*) (Glynn, 1980; Pratchett *et al.*, 2011). These crustaceans also ameliorate deleterious effects of vermetid gastropods (Stier *et al.*, 2010) and remove sediments from corals (Stewart *et al.*, 2006; McKeon *et al.*, 2012). Other coral-associated mutualists provide services other than defense, e.g. small fish provide nutrients to corals (Holbrook *et al.*, 2008, 2011).

While some symbionts protect corals, others are themselves stressors. Two common

corallivores on Indo-Pacific reefs are the gastropods *Coralliophila violacea* and *Drupella cornus*. These species feed on coral tissue and share similar habitats. In fact, a recent study found that these two snails were major contributers to coral mortality in Hawaii (Couch *et al.*, 2014). *Drupella cornus* (Figure 1.3a) is associated with increased disease prevalence (Antonius & Riegl, 1997, 1998; Nicolet *et al.*, 2013) and widespread coral decline (Boucher, 1986; Shafir *et al.*, 2008; Turner, 1994). They feed by scraping coral tissue with their radula, creating large white feeding scars (Figure 1.3c). They occur on a wide variety of corals, but are usually attracted to fast-growing, branching species such as *Pocillopora spp.* and *Acropora spp.* (Shafir *et al.*, 2008; Schoepf *et al.*, 2010; Al-Horani *et al.*, 2011), however, they will feed on slower growing mounding corals such as *Porites spp*, especially following a major disturbance (Hoeksema *et al.*, 2013). Recruitment appears to be increased in the presence of adult conspecifics (Turner, 1994), possibly because snails are attracted to chemicals given off by coral mucus (Kita *et al.*, 2005).

Coralliophila violacea (Figure 1.3b) also is a coral predator, although it appears to be less problematic than *D. cornus* (Brawley & Adey, 1982; Baums *et al.*, 2003). It feeds by inserting its radula into the coral polyp and slowly removing nutrients. *C. violacea* is a "prudent sessile feeder" (Oren *et al.*, 1998) that creates local carbon sinks because nearby polyps transport carbon to where the snail is feeding. *C.violacea* are therefore able to feed in one area for an extended time period. These snails are found almost exclusively on *Porites* corals (both massive and *P. rus*) (Zeid *et al.*, 2004).

1.2.3 Corals and symbionts as a dynamical system

Coral symbionts rely on corals for structure, protection, and in some cases, food. They are affected by the coral, yet themselves affect coral dynamics by influencing coral growth, morphology, and survival. It is therefore important to consider the dynamics of both habitat (corals) and occupants (symbionts) when studying the dynamics of spatial patterning on a coral reef.

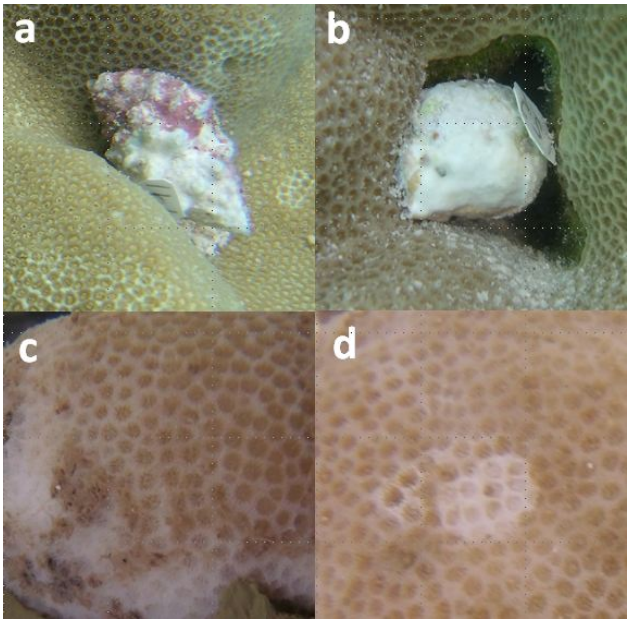

Figure 1.3: *Drupella cornus* (a) and *Coralliophila violacea* (b) with respective scars (c and d)

In addition to being driven by movement patterns of juvenile and adult symbionts, feedbacks could be important, for example, if the movement of corallivorous snails on a colony (or possibly between colonies) is influenced by the local abundance of conspecifics and patterns of damage. If individuals are attracted to conspecifics, large feeding aggregations may result, giving rise to aggregated patterns of feeding scars. The responses of the coral to this damage could further affect coral growth, morphology, and survival, and these trends could create feedbacks that affect the coral morphology as well as the symbiont community (Vytopil & Willis, 2001). While many studies have examined short term effects of various scars on tissue regeneration, the effect of damage patterning and long-term effects on morphology is unknwon. these feedbacks also could arise through the settlement of symbionts to coral.

Most coral symbionts, like other marine organisms, have a pelagic larval stage, after which they settle to corals. Larvae settle to corals based on a variety of cues such a habitat quality (Tolimieri, 1995; Holbrook *et al.*, 2000) and the amount of surrounding habitat (Stier & Osenberg, 2010; Morton & Shima, 2013). Thus, the morphology and spatial arrangement of corals can potentially affect the settlement of larval symbionts. There are two competing

hypotheses for how the spatial arrangement and amount of habitat affects settlement: 1) the field of dreams (FOD) hypothesis ("if you build it they will come"), which predicts that additional habitat will result in a proportional increase in the total number of settlers (but no change in the number of symbionts per coral) (Palmer *et al.*, 1997; Stier & Osenberg, 2010); and 2) the propagule redirection hypothesis (Osenberg *et al.*, 2002a), which predicts that new habitat directs settlers away from existing habitat with the total number of settlers the same before vs. after the habitat increase. Because these symbionts affect the growth, morphology, and survival of coral, settlement events can have long-term implications for the dynamics of coral systems. While studies have examined recruitment dynamics of symbionts and their short-term effect on corals, the long-term consequences are unknown.

Whether the effects of coral symbionts are the result of settlement patterns or post-settlement processes such as movement, the dynamic nature of their coral habitat leads to important feedbacks that affect the spatial structure and dynamics of coral reefs. This dissertation addresses the relationships between corals and symbionts at both a population and individual level to elucidate still unknown effects of symbionts on their corals as well as long term dynamics due to the feedbacks within the system. In this dissertation, I study these relationships using a combination of laboratory studies, field experiments, and mathematical models and simulations. In the second chapter, I document aggregations of two corallivorous snails, describe drivers of those aggregations, and test the effect of aggregations on coral growth. In the third chapter, I examine the effects of aggregated consumption on the response of the coral. To do this, I document the spatial patterns of coral lesions, test the effects of the distance between lesions on lesion healing, coral growth, and coral morphology, and document the effects of chronic damage on coral morphology. In the fourth chapter, I look at a larger spatial scale and examine the effects of landscape configuration on settlement distributions. I describe parameter regimes where the heterogeneity in settlement persists, and compare the heterogeneity due to landscape configuration to heterogeneity due to variation in patch quality. Finally, in the fifth chapter I incorporate habitat dynamics

and the effect of occupants on habitat to explore spatial patterns that emerge across the landscape.

Chapter 2: Aggregation patterns of two corallivorous snails and consequences for coral dynamics[1]

[1]Hamman, E.A. To be submitted to *Coral Reefs*

2.1 Abstract

Spatial heterogeneity plays an important role in consumer-resource interactions. It arises from variability in the underlying distribution of the resource and/or the consumer, as well as the habitat in which the consumer-resource interaction occurs. In some cases, the resource *is* the habitat, especially when the habitat is biogenic (e.g. kelp, corals, seagrasses). In these systems, the resulting dynamics can be particularly rich because the consumer-resource interactions are coupled with changes in the habitat (i.e., resource) that are due to the consumer-resource interaction. In Moorea, French Polynesia, two corallivorous snails, *Coralliophila violacea* and *Drupella cornus*, feed and live on massive *Porites* corals. Here, I 1) document the spatial patterns of the snails among sites, within sites, and on corals; 2) examine the drivers of smaller-scale aggregations by testing the attraction of the snails to chemical cues coming from conspecifics and corals; and 3) test the effects of aggregations of snails on coral growth by manipulating snail density. The distributions of both snails were highly heterogeneous among sites across the island, and both species were spatially aggregated both among and within corals. The source of chemical attraction that caused the small-scale clustering differed between the two snails. *D. cornus* was attracted to conspecifics and corals damaged by conspecifics, whereas *C. violacea* was attracted to damaged corals (regardless of the cause), and not conspecifics. Increasing snail density caused a linear decline in coral growth that was similar for the two snail species. The combination of the clustered spatial pattern of both snail species and their negative effects on coral growth could lead to important feedbacks in which high densities of snails reduce coral cover in localized areas and create spatial dynamics that affect the spatial distributions of both corals and snails across the reef.

2.2 Introduction

The spatial distribution of organisms over a landscape is often highly variable. In some cases, these spatial distributions arise from underlying variation in the habitat (e.g. vegetation distributions are linked to soil type and available nutrients, John *et al.*, 2007), or predators (e.g. African ungulates occupy areas rarely used by predators, Thaker *et al.*, 2011). In other cases, the heterogeneity results from intrinsic movement patterns (e.g. plants with lower seed dispersal distances exhibit more clustering than plants that disperse over greater distances, Seidler & Plotkin, 2006). Finally, the spatial distributions also can result from underlying heterogeneity in the physical characteristics of the environment that define the habitat (e.g. pools and riffles in streams). In many systems, the habitat is dynamic and will change in response to changing environmental conditions. Changes in the habitat will often influence the distribution of the occupants as well. While a static habitat might support a fairly consistent spatial distribution of organisms, organisms that use a dynamic habitat will be constantly altering their distribution as the habitat changes.

However, many habitats are comprised of biogenic structures (e.g. kelp, corals, seagrasses, and trees). In these systems, some of the organisms that occupy the habitat are also consumers of it. Thus, the habitat and resource are the same, and the dynamics of the consumer-resource interaction become intrinsically linked to the spatial and temporal dynamics of the habitat within which the consumer and resources interact. When the occupant is a consumer of the habitat, habitat loss is not solely due to stochastic events and environmental factors, but can also occur due to the activity of the consumer, particularly in sites with high densities of consumers.

One system in which these coupled dynamics of habitat and consumers may be particularly important is coral reefs. Corals are a biogenic habitat that host a variety of symbionts whose effects potentially drive coral dynamics. Some of these symbionts have positive effects on the coral. For example, *Trapezia* crabs protect corals from predators (Glynn, 1980;

Pratchett *et al.*, 2000; Pratchett, 2001; McKeon *et al.*, 2012; McKeon & Moore, 2014) and other stressors (Stewart *et al.*, 2006; Stier *et al.*, 2010, 2012). Other symbionts negatively affect corals. For example, many organisms that use coral as habitat are corallivores and consume living coral tissue (Rotjan & Lewis, 2008).

Corallivory is an important driver of overall reef health and affects coral mortality, disease, and recruitment. Outbreaks of corallivores such as the seastar, *Acanthaster planci*, and the gastropod, *Drupella cornus*, can cause widespread mortality of corals. For example, *A. planci* reduced coral cover by 90% in a 2.5 year period in Guam (Chesher, 1969), and *D. cornus* reduced coral cover by 75% on areas of backreef at Nigaloo Reef in Western Australia (Turner *et al.*, 1994). Additionally, diseases can be transmitted by corallivores (e.g. white band is transmitted by *Coralliophila abbreviata*, Williams & Miller, 2005). Finally, corallivory affects community composition through processes such as the preferential predation of newly settled (Penin *et al.*, 2010) and juvenile (Lenihan *et al.*, 2011) corals. Because corals provide food and habitat for many corallivores, corals and their symbionts provide the opportunity to investigate feedbacks between consumers and their resource/habitat and their effects on spatial patterns and dynamics.

I focus on two corallivorous gastropods, *Drupella cornus* and *Coralliophila violacea* (Fig. 2.A5 a,b). Both gastropods feed on coral tissue and can play an important role in reef dynamics, especially when they occur at high densities. In fact, a recent study noted these two snails are major contributors to coral mortality in Hawaii (Couch *et al.*, 2014). Furthermore, both species are often commonly listed among the most abundant gastropods across the Indo-Pacific, occurring in high numbers in Kenya (e.g. McClanahan, 1990) and the Red Sea (Zeid *et al.*, 2004; Al-Horani *et al.*, 2011).

Drupella cornus (Fig. 2.A5a) feeds by scraping coral tissue with its radula, leaving behind large, white feeding scars (Fig. 2.A5c). This snail is associated with widespread coral decline (Boucher, 1986; Shafir *et al.*, 2008; Turner, 1994) and while they prefer faster-growing branching corals (e.g. *Acropora* and *Pocillopora*) (Taylor, 1978; Shafir *et al.*, 2008; Schoepf

et al., 2010; Al-Horani *et al.*, 2011), they will feed on slower-growing mounding corals such as *Porites* or even temporarily mushroom corals (Hoeksema *et al.*, 2013; Moerland *et al.*, 2016) when preferred prey are scarce. These snails are often found in large aggregations, possibly due to increased settlement near adult conspecifics (Turner, 1994) and chemicals in coral mucus (Kita *et al.*, 2005).

Coralliophila violacea (Fig. 2.A5b) does not directly kill the polyp from which it feeds. Instead, it slowly removes nutrients, leaving behind small, circular scars (Fig. 2.A5d). Because of this feeding behavior, this snail is known as a "prudent sessile feeder" (Oren *et al.*, 1998). As it feeds, it creates carbon sinks as nearby polyps transport nutrients to where the coral tissue is being damaged by the snail. This allows the snail to feed in one area for long time periods. It is frequently found on *Porites* corals (Taylor, 1978; Soong & Chen, 1991). While there are relatively few studies of *C. violacea*, related species in the Caribbean, *Coralliophila abbreviata*, *Coralliophila caribaea* and *Coralliophila galea*, are fairly well studied and is a contributor to widespread decline of *Acropora palmata* (e.g. Brawley & Adey, 1982; Miller, 2001; Baums *et al.*, 2003) and are spatially clustered (Potkamp *et al.*, 2016) .

Because these two snails both consume and live on similar corals (when preferred prey of *Drupella* is rare), yet feed (and thus, likely affect the coral) through different mechanisms, they provide an interesting opportunity for a comparative investigation of the drivers and consequences of spatial patterning in consumer populations. In this paper I address three questions: 1) How are *D. cornus* and *C. violacea* distributed in space and do patterns of aggregation exist at multiple spatial scales (island-wide, among sites, among quadrats, and within corals)? 2) What chemical cues drive the aggregations of these two species at smaller spatial scales? and 3) How does this variation in local density (resulting from aggregation) affect coral growth? To address these questions, I used a combination of field surveys and experiments conducted in Moorea, French Polynesia.

2.3 Methods

2.3.1 Spatial distributions of *D. cornus* and *C. violacea*

To quantify spatial patterns among corals and sites, I surveyed 8 areas in the Moorea lagoon (see Fig. 2.A6 for a map of sites) in June and July 2011. The lagoon of Moorea is shallow (up to approximately 3m depth), and following the *A. planci* outbreak in 2008 (Kayal *et al.*, 2011), the dominant corals were massive *Porites* species and *Porites rus*. Of the sites surveyed, two (North Shore Crest, Haapiti Crest) were located directly behind the reef crest. Three sites were backreef sites (Temae, Ava Iti, and West), and three were on the fringing reef (Pharmacie, Hazard Marker, and Hilton). At each site, I surveyed ten 25 m by 0. 5m belt transects oriented perpendicular to shore. All transects were approximately 5m apart. Each transect consisted of 0.5 m by 0.5 m quadrats placed over live coral centered along the transect (for an illustration of the method, see Fig, 2.C8). Each quadrat was centered on the transect and laid contiguous to the previous quadrat or whenever live coral was encountered (if it was preceded by sand or a dead coral). Within each quadrat, I recorded the coral species, number of snails (*C. violacea* and *Drupella* spp.), and visually estimated the percent of live coral tissue in the quadrat. In total, I surveyed 80 transects, with 4-24 quadrats per transect (depending on the amount of live coral cover in the transect).

To model the distribution of snails across multiple spatial scales, I used generalized linear mixed effects models using the glmmADMB package in R (Skaug *et al.*, 2011). I modeled the abundance of each snail species as a function of coral cover (area of *Porites rus*, massive *Porites*, and branching corals). Because *C. violacea* is only found on *Porites rus* and massive *Porites*, I did not include branching corals in the analysis of *C. violacea* abundance.

To test for spatial clustering, I compared a model with (Negative Binomial Distribution, NBD) and without (Poisson) statistical overdispersion. I also included zero-inflated versions of the two distributions (although a zero-inflated Poisson could not be fit for *C. violacea*).

I selected the best model using AIC and tested the significance of overdispersion using a likelihood ratio test. Statistical overdispersion (or spatial clustering), can occur at any spatial scale, and is captured by the overdispersion term (k) in the negative binomial model or as additional variation in the Poisson model. To account for spatial variation at the multiple spatial scales represented in the hierarchical survey (quadrat, transect, and site), I included site and transect as nested random effects. I also used a likelihood ratio test to determine the significance of the random effects (clustering at multiple spatial scales) by comparing a model with the best-fit distribution with and the corresponding model without the random effects. The presence of random effects at a given level indicate that snails were aggregated across that spatial scale.

To determine spatial patterns on a single coral, I conducted a separate survey in 2014. To obtain independent values of nearest neighbor distances, I located corals with more than two snails (91 corals with *Drupella* and 107 corals with *Coralliophila*), haphazardly selected a snail, and measured the distance from shell to shell of its nearest neighboring conspecific. I then compared the distribution of those distances to the expected distribution of nearest neighbors (derived in Appendix 2.7), assuming snails were distributed randomly in space ("counts" follow a Poisson distribution), and assuming snails were clustered in space ("counts" follow NBD). I selected the best model (Poisson vs. NBD) using AIC and tested for goodness of fit using a multinomial likelihood ratio test.

2.3.2 Chemical cues

To test the attraction of the two snails to chemical cues from conspecifics, corals, and coral damage, I conducted laboratory choice trials with four options in each trial. I conducted these experiments in an aquarium with four chambers 15 cm by 10 cm extending from a central area (Appendix 2.C7). Each chamber was separated from the central area by a mesh barrier placed 5 cm into the side chamber. An overhead flow system delivered water to the back of each chamber, which exited the system from the floor of the central area; thus

water flowed from each chamber into the central area. I ran three different experiments, each providing four options to the snails (one in each of the four chambers). The first experiment offered "snails" (i.e. two snails), "coral," (i.e. one undamaged coral), "snails + coral" (i.e. two snails consuming a coral), and an "empty" chamber. The second experiment offered combinations of snails and corals, but in all cases the snails were placed in mesh sacks to prevent feeding during the trial. In this experiment, options were "snails + snail scar" (i.e. two snails and a coral fed upon by snails), "snail scars" (i.e. a coral fed upon by snails with no snails present), "snails + artificial scar" (i.e. two snails and a coral damaged with a waterpik), and "artificial scar" (i.e. a coral damaged by a waterpik without snails). The final experiment offered only corals with options of "coral" (i.e. undamaged coral), "artificial scar," and "snail scar."

I collected *C. violacea*, *D. cornus*, and juvenile massive *Porites* from the Moorea lagoon three days prior to their use in an experiment. From the time the snails and corals were collected to the start of the experiments, I held snails and corals together in plastic containers with mesh sides to enable water flow and placed corals not receiving snail damage directly in the water table. Half an hour before the start of the experiment, I removed snails from the corals and used a waterpik to create artificial scars similar in size to the natural scars.

I placed the appropriate corals and snails into their chambers (behind the mesh barriers), and placed the test snail in the center of the middle area. If the test snail showed no movement after 30 minutes, it was replaced. After two hours (preliminary observations indicated that few snails move after this time), I recorded the final location of the test snail, removed the corals and snails, and wiped down each aquarium. Each snail was only used in one trial.

To analyze the data, I used a multinomial exact test followed by pairwise comparisons with Bonferonni-adjusted P-values for the first two experiments. For the third experiment, I used a priori contrasts and a multinomial exact test to address three questions: 1) are snails attracted to coral ("empty" vs. the other three treatments, 2) are snails attracted to

damaged coral (undamaged vs. damaged corals), and 3) are snails attracted to a certain kind of damage (artificial vs. natural damage)?

2.3.3 Effect of snail abundance on coral growth

To test for effects of aggregation, I varied the density of snails on juvenile corals. If aggregations (or higher densities of snails) had greater consequences for corals than the additive effects of an individual snail, I hypothesized the effect of local density on coral growth would be nonlinear. I tested the effect of snail density on the growth of juvenile massive *Porites* in a laboratory experiment conducted from July-August 2011 (*C. violacea*) and June-July 2012 (*D. cornus*). For each experiment, I collected 40 juvenile colonies from pavement sites near the reef crest on the north shore of Moorea, French Polynesia, and transported them to the Richard B. Gump field station. I collected the snails from fringing reef sites, and starved them for three days prior to the experiment.

I attached corals to plastic mesh with Zspar epoxy and weighed them using the buoyant mass technique (Davies 1989). I then randomly assigned corals to a treatment of 0, 1, 2, or 3 snails and placed the snails directly on the coral in large tubs with a flow through system covered by shade cloth. Cages (*C. violacea*) and tethers (*D. cornus*) kept the snails on their assigned corals. Throughout each experiment I monitored the snails and replaced any that were missing or dead. At the end of each experiment I weighed the corals and measured the surface area using the foil technique (Marsh Jr, 1970). I standardized the growth of each coral by dividing its change in mass by its surface area. In both experiments, I excluded several corals from the analysis due to death prior to the conclusion of the experiment. These corals were distributed across all treatments. Six corals died in the *C. violacea* experiment, (three that had one snail, one that had two snails, and two with three snails), and three corals died in the *D. cornus* experiment (one each of a control coral, coral with one snail and coral with two snails). I analyzed the data using a linear regression and tested for non-linearities by comparing linear, exponential, and quadratic models and selecting the best model using

AICc.

2.4 Results

2.4.1 Spatial distributions of *D. cornus* and *C. violacea*

Both snail species exhibited non-random variation across all spatial scales as the negative binomial model was a better fit than the Poisson models (Table 2.1). Additionally, overdispersion parameter was significant for both snails ($G^2(1) = 2984.1, P < 0.0001$ and $G^2(1) = 162.8, P < 0.0001$, Fig. 2.1B) for *C. violacea* and *D. cornus*, respectively), demonstrating that both species were aggregated due to factors other than coral cover or the differences between site and transect locations accounted for by the random effects. For *C. violacea*, but not for *D. cornus* model fit was improved by including a parameter for excess zeros (Table 2.1), suggesting patterns of aggregation that were even more extreme than could be captured by the negative binomial model alone.

Although both species were aggregated spatially at all scales, their specific patterns of variation differed, as did the likely causes. *C. violacea* are found on both *Porites rus*, and massive *Porites* (Table 2.2), although only *P. rus* was a significant contributor to their distribution ($z = 2.49 P = 0.013$ and $z = 0.67, P = 0.50$ for *P. rus* and massive *Porites*, respectively). For *D. cornus*, the area of branching corals was the only significant coral cover type ($z = 3.15, p = 0.0016$). Thus, part of the pattern of aggregation was attributable to the availability of these substrates. However, even after accounting for variation in coral cover, the nested sampling random effect was significant for both snails ($G^2(3) = 146.8, P < 0.0001$, $G^2(3) = 18.6, P = 0.0003$ for *C. violacea* and *D. cornus*, respectively). *C. violacea* showed more variation among sites than transects within a site, and the opposite was true for *D. cornus* (Fig. 2.1A).

At the smallest spatial scale (on a coral), most snails were very near to a conspecific – 69% of *D. cornus* and 58% of *C. violacea* were ≤ 2 cm from the nearest conspecific (Fig. 2.2

and 2.A5). For both snails, the expectation derived from a negative binomial distribution was the best fit, but in neither case was it a good fit ($G^2 = 7.6 * 10^{-6}$, $p < 0.0001$ and $G^2 = 1.36 * 10^{-13}$, $p < 0.0001$ for *D. cornus* and *C. violacea*, respectively). The high occurrence of snails < 2 cm from their nearest neighbor resulted in poor goodness of fit. Thus, snails were even more aggregated than expected by the negative binomial as including overdispersion (variance > mean) in counts failed to adequately describe the observed clustering demonstrated by the number of snails touching the nearest neighboring conspecific.

2.4.2 Chemical cues

Both snail species preferentially moved towards chemical cues. In the first experiment (Fig. 2.3a), both species exhibited significant preference (*D. cornus*, $p < .0001$, *C. violacea*, $p = .0001$), with both species showing the lowest use of the empty chamber. *D. cornus* preferred the combination of conspecifics and coral above all other options. *C. violacea* also preferred this combination over the chamber with only snails, although the use of the snail-only chamber did not differ significantly from that with coral alone. In the second experiment (Fig. 2.3b), in which every chamber contained a coral, neither snail species showed a significant preference (*D. cornus*, $P = 0.48$, *C. violacea*, $P = 0.50$).

In the third experiment, both species showed significant preference, albeit for different treatments (Fig. 2.3c). Because this experiment was analyzed using contrasts, I present the preference of each snail species to those three contrasts (coral vs. no coral, damaged coral vs., undamaged coral, and corals damaged by conspecifics vs. artificially damaged with a waterpik). *D. cornus* was attracted to corals ($p = .0005$), preferred damaged over undamaged corals ($p = .012$), and preferred corals damaged by conspecifics over corals that were artificially damaged ($p = 0.021$). In contrast, *C. violacea* chose chambers with corals ($p = .0001$), did not show significant preference for damaged vs. undamaged corals ($p = 0.096$, although this effect is in the same direction as observed for *D. cornus*), but

(unlike *D. cornus*), preferred coral damaged artificially over corals damaged by conspecifics ($p = 0.0034$). Thus, the two snail species showed opposite responses to naturally damaged vs. artificially damaged corals. This result contrasts with the result in the second experiment (e.g. compare "snail scar" with "artificial scar").

2.4.3 Effect of snail abundance on coral growth

Coral skeletal growth declined linearly with snail density, and there was little evidence of nonlinear effects (Fig. 2.4). Neither the quadratic model ($AIC_c = 416.27, \Delta AIC_c = 364.29$) nor the exponential model ($AIC_c = 174.79, \Delta AIC_c = 954.32$) provided a better fit to the data than the linear model ($AIC_c = -779.65$). The addition of each snail resulted in an approximately 17% reduction in the growth of juvenile massive *Porites* ($F_{1,67} = 11.2, p = 0013$). Additionally, there was no detectable difference in the effect of the two snail species ($F_{1,67} = 0.0044, p = 0.953$). Despite the experiments occurring in different summers, the growth rates of the control corals in both experiments were very similar to each other.

2.5 Discussion

Both *C. violacea* and *D. cornus* are clustered in space at multiple spatial scales, and are attracted to chemical cues (damaged corals for *C. violacea* and both conspecifics and conspecific damage for *D. cornus*) that likely contribute to these aggregations. Corals grow more slowly in areas where snails are aggregated, and although the snails feed through different mechanisms, they have the same approximate negative effect on coral growth.

The aggregation of *Drupella* in space is consistent with previous work. Studies on the Great Barrier Reef (Turner *et al.*, 1994; Turner, 1994; Cumming, 1999) found aggregation of *Drupella spp.* across sites in Nigaloo Reef, although these sites encompassed an area greater than that included in this study from Moorea. Additionally, studies in Kenya show increased *Drupella* abundance in disturbed and overfished areas (McClanahan, 1997, 1990)

and studies in the Red Sea document higher densities in areas disturbed by divers (Guzner *et al.*, 2010). However, a study conducted in the Red Sea did not find significant differences in density across sites (Schoepf *et al.*, 2010), although these sites were much nearer to one another than those in this study.

Multiple mechanisms likely drive differences in snail distribution at small scales (on a single coral) and larger scales (along corals or sites). Clustering of *D. cornus* among corals and sites is likely due, in part, to the recruitment patterns of *D. cornus*. Juvenile *D. cornus* often settle near adults (Turner, 1994), and groups of *D. cornus* located near each other are closely related, suggesting groups of larvae settle together (Johnson *et al.*, 1993). Aggregations of *D. cornus* are also expected on branching corals, because *D. cornus* prefers branching corals over mounding corals such as massive *Porites*. While this preference likely played an important role in aggregations (as shown by the magnitude and significance of coral cover), the better fit of the NBD distribution demonstrates aggregation outside of this preference. Additionally, many of these preferred branching corals are either not present or in very low abundance in Moorea due to an *A. planci* outbreak in Moorea (Kayal *et al.*, 2011). At smaller spatial scales, previous studies have shown an attraction of *Drupella* to conspecifics (Schoepf *et al.*, 2010), but did not test the observed attraction to corals damaged by conspecifics (Fig. 2.3).

The heterogeneity in *C. violacea* distributions is likely driven by different factors than those that drive variation in *D. cornus*. *C. violacea* does not have much spatial genetic structure at a small scale (Lin & Liu, 2008), so the clustering of *C. violacea* is likely due to settlement of a genetically similar cohort. Additionally, plenty of the preferred species for *C. violacea*, massive *Porites* (Soong & Chen, 1991) and *Porites rus*, are present in Moorea, so aggregations are not due to patches of preferred corals.

Feeding strategy may also influence species distribution patterns. Because *C. violacea* feed by creating carbon sinks (Oren *et al.*, 1998), the snails likely choose areas of the coral where this could be accomplished easily. Previous studies show carbon sinks were not ob-

served around a single snail (Oren *et al.*, 1998), but only in areas of multiple conspecifics or coral growth areas (such as at colony margins and damaged areas). Therefore, *C. violacea* likely choose areas with large aggregations of conspecifics or damaged areas of coral. The pattern I observed in my choice experiments is consistent with this idea. The combination of snails and corals was the most popular choice in the first choice experiment (Fig. 2.3), and corals with artificial scars were the most popular choice of the third experiment. While there was no preference in the second experiment, all corals were damaged, so if damage is the most important cue, rather than the conspecifics, it is plausible that no type of damage would be preferred. A potentially confounding factor in these experiments, is that all corals were removed from the reef with a chisel, and even after sitting in the water table to recover, likely gave off some resulting cues from handling. However, these were likely much less than the signals from fresh scars.

The responses of *D. cornus* to chemical cues were much clearer, and demonstrate the importance of conspecifics in shaping distributional patterns. In all choice experiments, options involving conspecifics (either their presence or the damage they create), were chosen most often (Fig. 2.3), and snails could detect damage by conspecifics even in their absence (Fig. 2.3c). This attraction to conspecifics was previously described by Schoepf *et al.* (2010), where both adults and juveniles preferred conspecifics over food. Our study extends these results and shows that snails are more attracted to conspecifics over undamaged coral and even to corals damaged by conspecifics over coral damaged artificially.

Both snails had extremely similar negative, linear effects on coral growth (Fig. 2.4). These similar effects were surprising, especially because *D. cornus* creates a much larger scar than *C. violacea*, suggesting that they might have had larger per capita effects. Instead, the equality of their per capita effects suggests that even though *D. cornus* consumes more tissue, *C. violacea* has a comparable effect because it creates a carbon-sink that likely reduces the resources available for growth over an area much larger than the scar it creates. The rate of recovery of a coral might differ however, as *C. violacea* does not permanently remove

or damage as much tissue as *D. cornus*.

Finally, the interaction between snail aggregations (due to either conspecifics or coral damage) and the effect of the snails on coral growth could cause important feedbacks in the system at multiple spatial scales. On a coral, this could increase spatial heterogeneity through variation in growth rates across the surface of the coral (high where snails are absent and low in the vicinity of snail aggregations), which would increase topographic complexity of the colony. If snails move to areas of damage and high conspecifics, I might expect larger scale variation in coral growth rates linked to the presence of snails. Among corals and among sites, these localized effects could lead to heterogeneity on these spatial scales as well. As snails are able to move from coral to coral (Hamman, personal observation), snails could create patchiness in coral growth rate survival. Similarly, other researchers have also noted the potential for feedback loops due to coral tissue damage and increases in corallivory and disease transmission (Guzner *et al.*, 2010).

As snails deplete the resource, they also remove their habitat, making themselves more vulnerable to predation. As a result, snails will eventually move from depleted areas of the coral to new areas, forming new aggregations (Knowlton *et al.*, 1981). Additionally, during outbreaks with high amounts of coral mortality, there could also be subsequent die-offs of snails. Through the interplay of these interactions, there could be spatial patterns that emerge from these dynamic "hot spots" of localized increases in snail density. These patterns are also observed in host-parasitoid systems. For example, parasitoids of the tussock moths cause pattern formation during outbreaks. The extent of the localized reductions in the host are limited by the dispersal distance of the parasitoid (Wilson *et al.*, 1999). Host-parasitoid systems are, in some ways, analogous to systems where a biogenic habitat is consumed by an organism. For example, in the snail-coral example, snails do not parasitize their host, but they do require the coral for both food and shelter. If the coral dies, the snails are limited in their ability to redistribute themselves over the reef. It is likely then that I might expect a similar creation of dynamic patches of healthy and unhealthy corals emerge from long-term

interactions between consumers and the biogenic habitat they occupy.

2.6 Tables and Figures

	Model	log likelihood	AIC	Δ AIC
C. violacea	Poisson w/ Random Effects	-2931.2	5872.4	3013.2
	Negative Binomial w/ Random Effects	-1439.1	2890.3	31.0
	Zero Inflated Negative Binomial w/ Random Effects	-1422.6	2859.2	0
	Zero Inflated Negative Binomial without Random Effects	-1505.8	3021.5	162.3
D. cornus	Poisson w/ Random Effects	-514.3	10406.	160.8
	Zero Inflated Poisson w/ Random Effects	-442.4	898.9	19.0
	Negative Binomial w/ Random Effects	-432.9	879.8	0
	Zero Inflated Negative Binomial w/ Random Effects	-432.9	881.8	2
	Negative Binomial without Random Effects	-442.2	894.4	14.6

Table 2.1: Model Selection Information

	Porites rus	massive *Porites*	Branching Corals
C. violacea	$\mathbf{3.3 \pm 1.3}$	0.6 ± 1.0	-
D. cornus	4.0 ± 2.9	2.4 ± 1.6	$\mathbf{9.2 \pm 2.9}$

Table 2.2: Results from GLMM. Parameter estimates for *C. violacea* are from the zero-inflated negative binomial fit, and estimates for *D. cornus* are from the negative binomial fit. Significant fixed effects are bolded ($p < 0.05$), and estimates include $\pm$ one standard error.

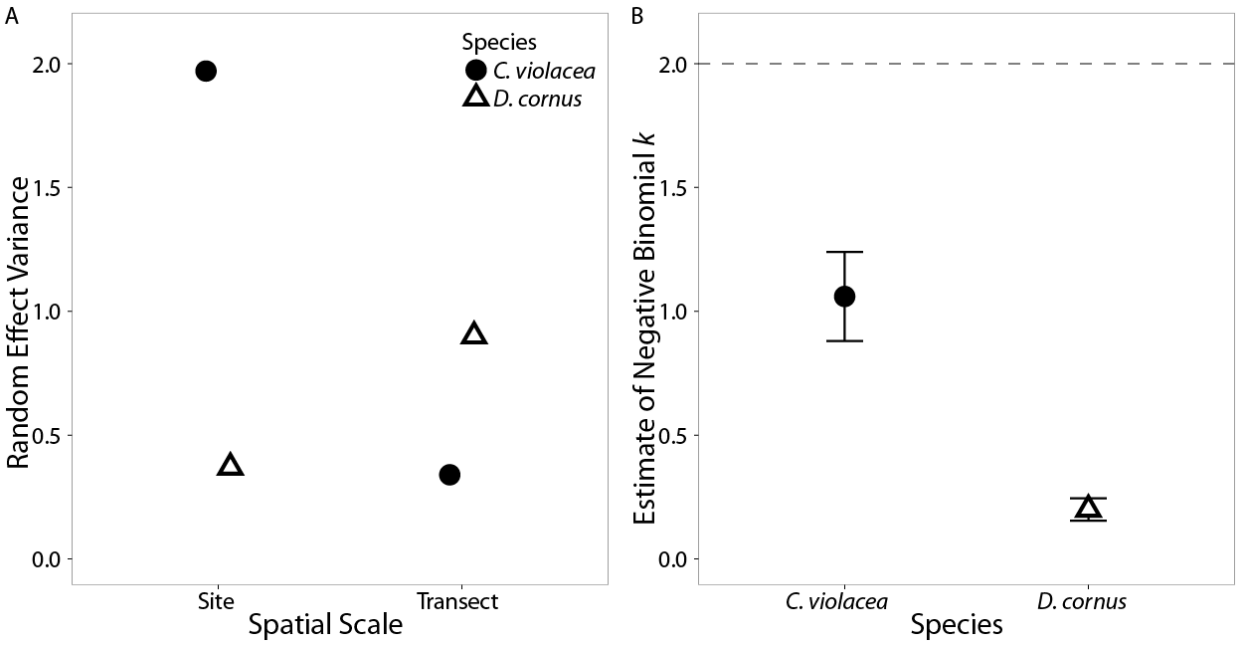

Figure 2.1: Evidence of spatial aggregation of both snails at multiple spatial scales. Both sets of estimates are from a Negative Binomial GLMM, which was the best fit for both snails. The estimated variance of the random effects (A) differs based on spatial scale (quadrats are nested in transects nested in sites). The two snails differed in the spatial scale with the largest variation. There was also evidence of clustering represented in the estimate of k, or the overdispersion parameter (B). Values less than 2 (dotted line) indicate spatial aggregation among the quadrat counts.

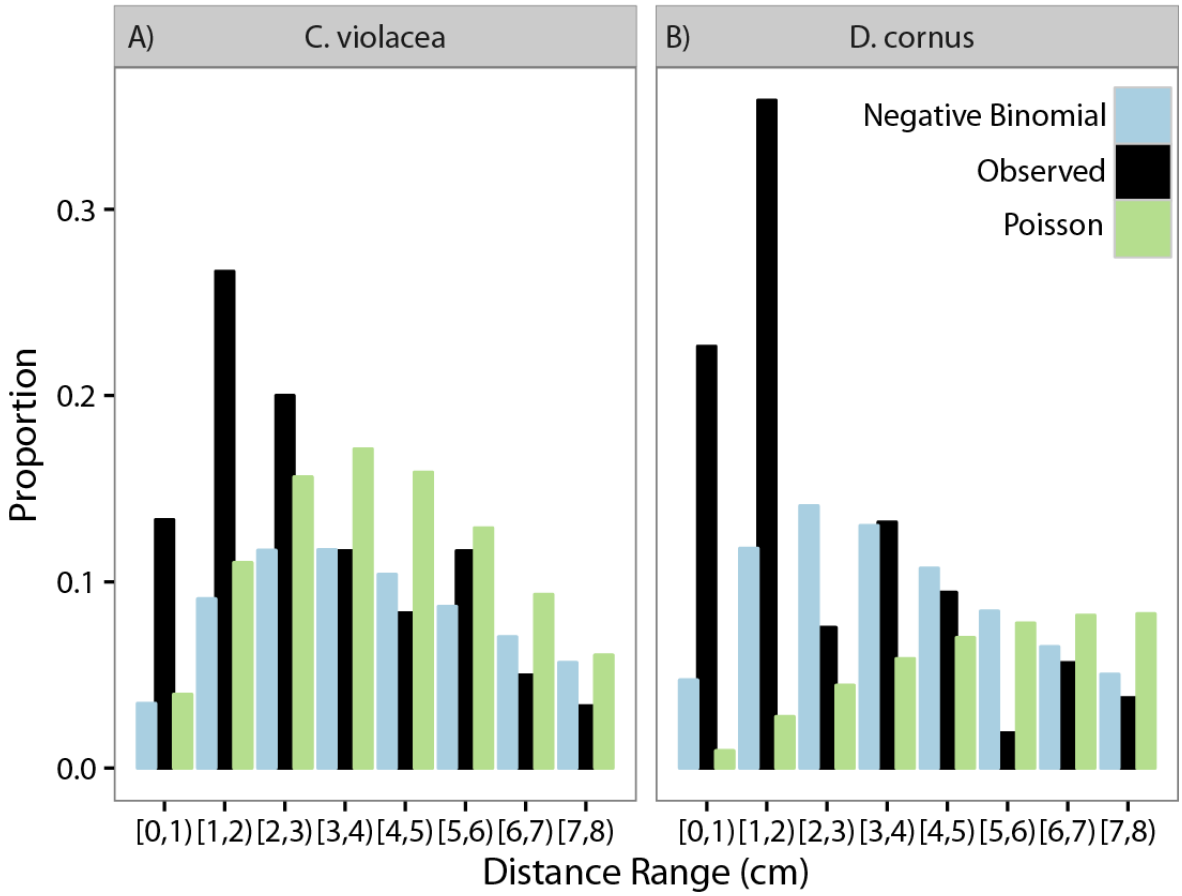

Figure 2.2: Distributions of nearest neighbor distances in (A) *Drupella* and (B), *C. violacea*. Observed values are shown by black points (all values ≥ 7 are grouped), and expected values under complete spatial randomness (Poisson - green), and spatial clustering (negative binomial blue). The Poisson and Negative Binomial distributions are not truncated, while the final class for observed contains all values greater than 7 cm. While the negative binomial distribution was the best fit for both snails (Poisson $\Delta AIC_C = 2.96$ and 11.38 for *Coralliophila* and *Drupella*, respectively) , it was not a good fit for either of them as it failed to capture the high frequencies of snails < 2 cm from their nearest neighbor.

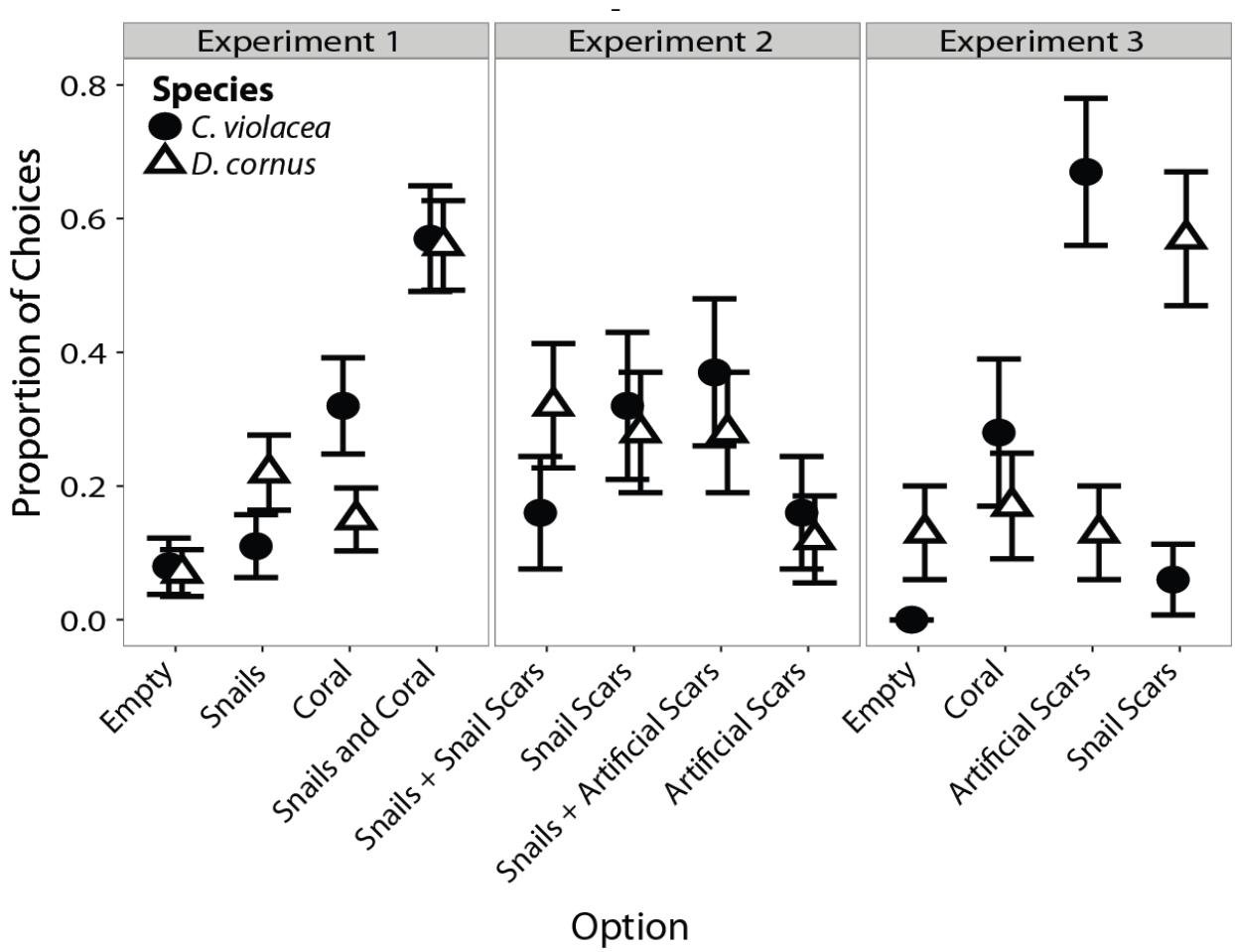

Figure 2.3: Attraction of snails to corals, conspecifics, and damage. Only snails that made a choice among the provide options were included in this figure. In Experiment 1, 40/56 Coralliophila and 55/63 *Drupella*, 19/28 *Coralliophila* and 25/32 *Drupella* in Experiment 2, and 18/20 *Coralliophila* and 23/25 *Drupella* made a choice.

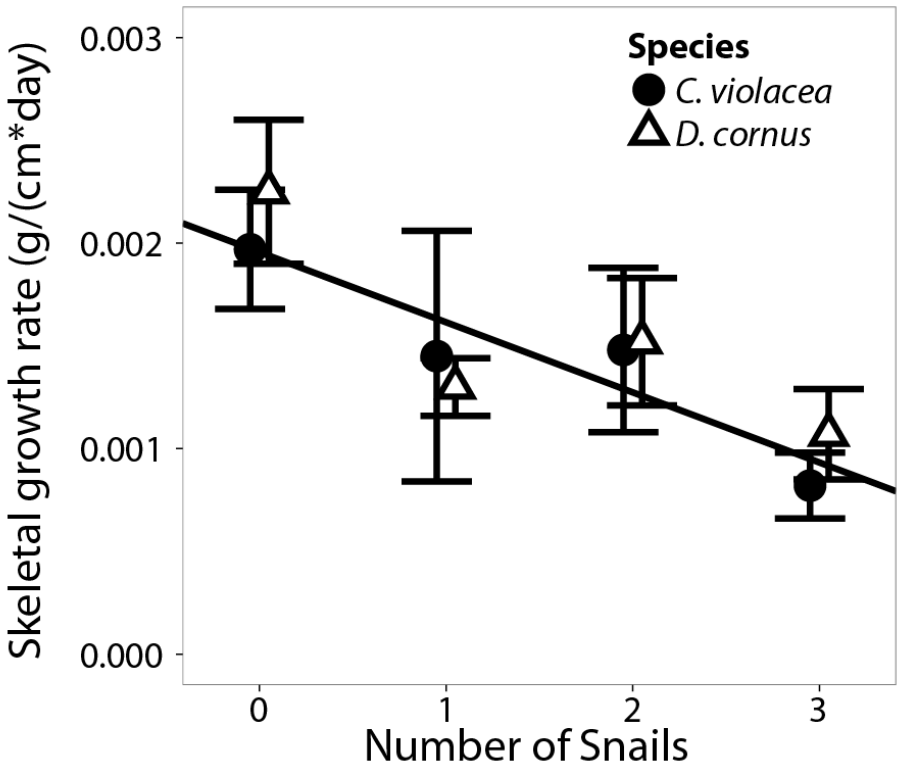

Figure 2.4: Snails decrease coral growth with increasing abundance. Results from experiments conducted in 2011 and 2012 also fail to demonstrate a difference in effect between the two snail species. Error bars represent ±1 standard error and the line the best-fit from a linear regression.

2.7 Appendix

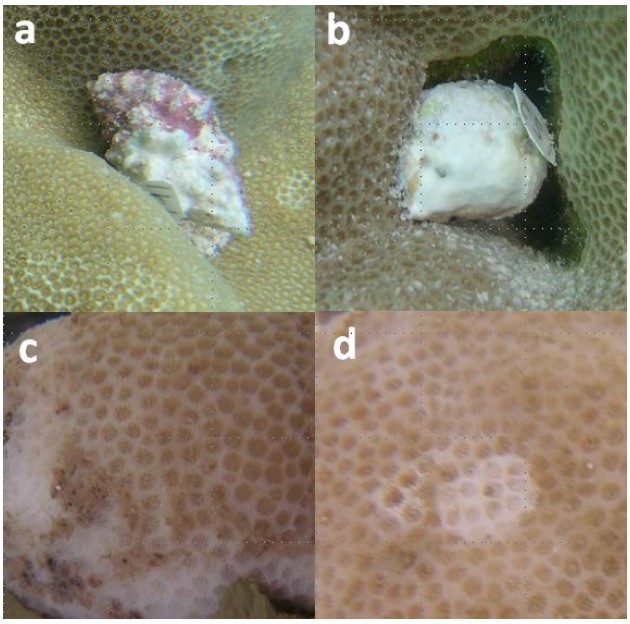

Figure 2.A5: *Drupella cornus* (a) and *Coralliophila violacea* (b) with respective scars (c) and (d)

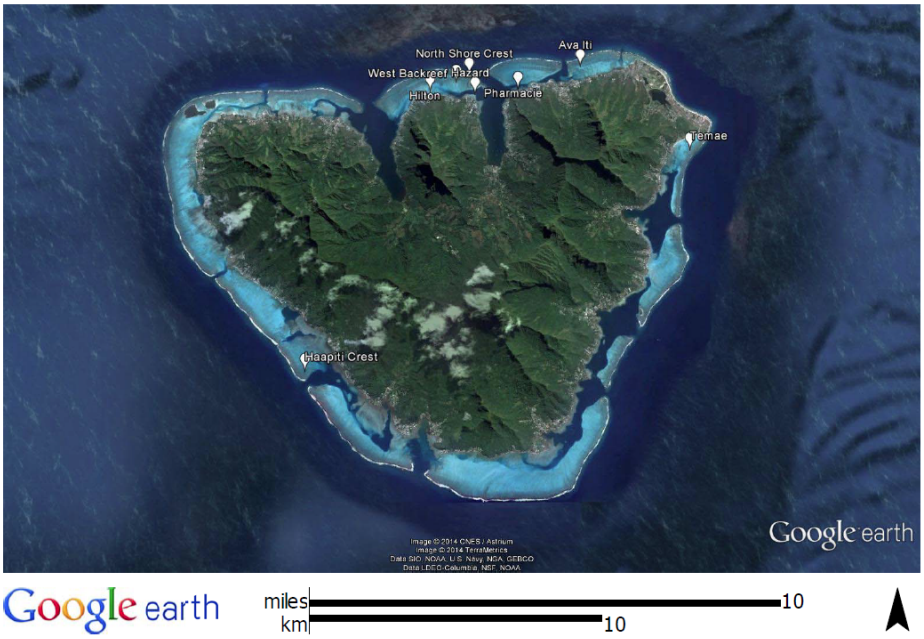

Figure 2.A6: Approximate location of sampling sites

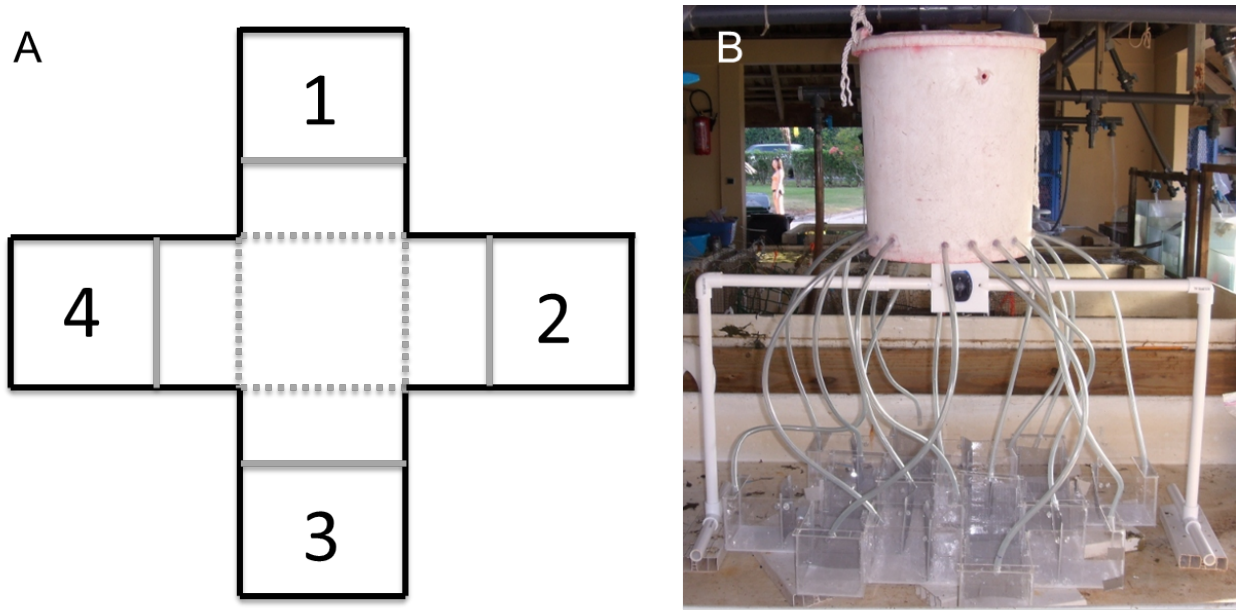

Figure 2.C7: Choice experiment aquarium. Each unit consists of 4 chambers (A) off a central area with the ends separated from the center via mesh dividers. Flow enters the chamber from an overhead bucket (B) through the backs of the chambers so that the water flows over the option (e.g. coral, snail) to the center of the chamber. The set-up in (B) consists of 4 units that run simultaneously.

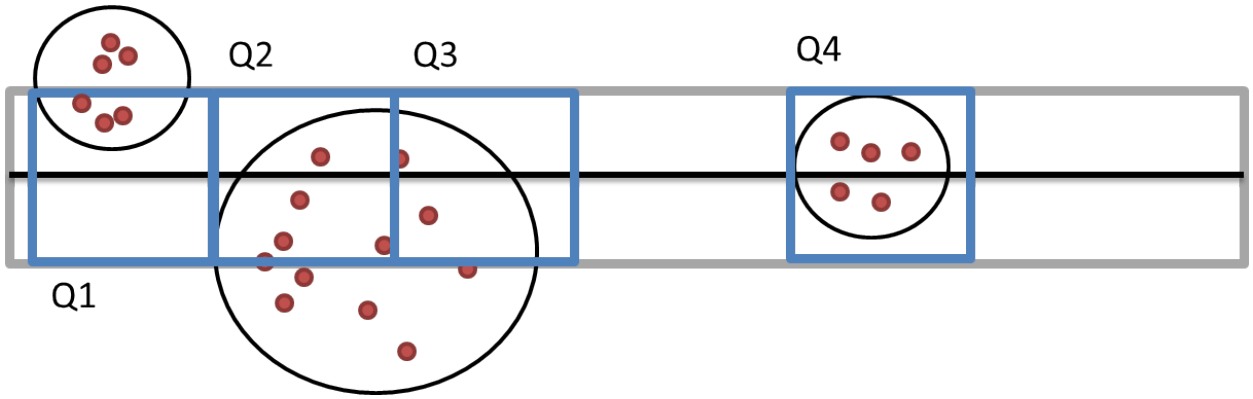

Figure 2.C8: Diagram of transect method. At each site, 10 parallel transects (black line) were placed approximately 5m from each other. Along each transect, I placed a quadrat (blue box) at each point where there was live coral. Some corals only partially fell in the quadrat (e.g Q1), while others were completely within the quadrat (e.g. Q4). Additionally, some corals were too large for a single quadrat, and multiple quadrats were used for the same coral (e.g. Q2 and Q3). For the among site analysis, each transect was used as a replicate (sum of all quadrats with live coral). For the within-site analysis, the quadrats were used.

Expected Nearest Neighbor Distributions

I measured the distance from a snail to its nearest conspecific and compared it to expected distributions (derived in this section) for the distribution of nearest neighbors under 1) Com-

plete Spatial Randomness ("counts" of snails follow a Poisson distribution), or 2) Aggregation ("counts" of snails follow a Negative Binomial distribution).

If a circle is centered at the origin, the probability that it is empty is a function of its radius x. I can find the cdf by considering the space it takes for the first event to occur:

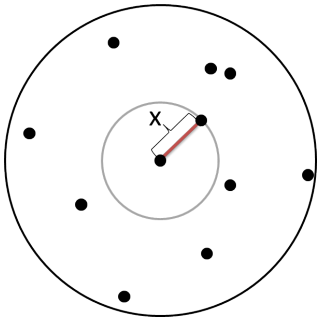

Figure 2.C9: To find the distance between one point and its nearest neighbor (x), I find the radius of the circle when the next point is encountered.

Random Distribution

To determine what the pdf is of randomly distributed nearest neighbors, I assume that snails are distributed as a Poisson Spatial Process.

$$F(D) = P(D \leq d) = 1 - P(D \geq d)$$

$$F(D) = 1 - P(\text{no neighbors are found between 0 and D})$$

$$F(D) = 1 - P(n = 0)$$

$$F(D) = 1 - \frac{(\lambda A)^0 e^{-\lambda A}}{0!}$$

$$F(D) = 1 - e^{-\lambda \pi x^2}$$

To find the pdf with respect to x (the distance, which is continuous), rather than n (which

is discrete)

$$f_D(x) = \frac{d}{dx}\left[1 - e^{-\lambda\pi x^2}\right]$$
$$f_D(x) = 0 - (-2\lambda\pi x e^{\lambda\pi x^2})$$
$$f_D(x) = 2\lambda\pi x e^{\lambda\pi x^2}$$

Clustered Distribution

Finding the pdf of nearest neighbors if snails are clustered is similar to above, only instead of assuming an underlying Poisson distribution, I assume the underlying distribution is a negative binomial.

$$F(D) = P(D \leq d) = 1 - P(D \geq d)$$
$$F(D) = 1 - P(\text{no neighbors are found between 0 and D})$$
$$F(D) = 1 - P(n = 0)$$
$$F(D) = 1 - \begin{pmatrix} k+0-1 \\ \\ 0 \end{pmatrix} \left(\frac{A}{A+\alpha}\right)^0 \left(\frac{\alpha}{A+\alpha}\right)^k$$
$$F(D) = 1 - \left(\frac{(k-1)!}{0!(k-1-0)!}\right)(1)\left(\frac{\alpha}{A+\alpha}\right)^k$$
$$F(D) = 1 - \left(\frac{\alpha}{\pi x^2 + \alpha}\right)^k$$

To find the pdf with respect to x (the distance, which is continuous), rather than n (which is discrete)

$$f_D(x) = \frac{d}{dx}\left[1 - \left(\frac{\alpha}{\pi x^2 + \alpha}\right)^k\right]$$

$$f_D(x) = \frac{2\pi k x}{\alpha}\left(\frac{\alpha}{\alpha + \pi x^2}\right)^{k+1}$$

Chapter 3: Spatial distribution of damage affects the healing, growth, and morphology of coral[1]

[1]Hamman, E.A. To be submitted to *Oecologia*

3.1 Abstract

Many predators and herbivores do not kill their prey, but rather remove or damage tissue. As a result, prey are often able to heal or regenerate this lost tissue. If the prey are modular organisms (e.g., plants and cnidarians), regeneration is frequently influenced by other modules interconnected to damaged ones. For example, many coral predators remove tissue from colonies consisting of many polyps, and these polyps often share resources with their neighbors. Thus, the distribution of tissue loss on a coral colony could affect tissue regeneration and coral growth by influencing the availability of healthy tissue in the neighborhood of damage. In particular, I hypothesized that spatially aggregated damage might be slow to heal due to competing demands on nearby polyps. To document spatial patterns of corallivory and their implications, I conducted: 1) field surveys documenting the spatial distribution of lesions on corals; 2) field experiments testing the effect of the distance between lesions on coral tissue healing, skeletal growth, and morphology; and 3) field surveys relating corallivore presence to coral growth and morphology. Lesions were aggregated at multiple spatial scales, and most lesions had other lesions within 2 cm. When lesions were near one another, coral tissue regeneration was depressed, although there was no effect on whole-colony growth. However, after a year, linear extension was lower in the neighborhood of the lesions. Additionally, corallivores (*Coralliophila violacea*) with low movement rates decreased growth and increased topographically complexity. These results suggest that corallivores that create clusters of coral damage have a greater effect on coral growth and recovery from damage than corallivores that spread damage throughout the colony.

3.2 Introduction

Many biogenic habitats (e.g. groves of aspen trees, grasslands, kelp forests, and coral reefs) consist of modular or colonial organisms. While each unit (e.g. plant ramet, or coral polyp)

is distinct and contributes to the overall growth of the organism (genet or colony), the units often are interconnected. These connections allow the units to communicate with one another and share resources (e.g. Marbà *et al.*, 2002; Alpert *et al.*, 2003; Stuefer *et al.*, 2004). The benefits of these connections often depends on the underlying habitat and resource distributions. As a result of their interconnectedness, modules in better habitats can benefit neighboring polyps in resource poor or deleterious microhabitats. For example, buried ramets of *Psammochloa villosa* that are connected to unburied ramets survive better and grow larger than ramets without these connections (Yu *et al.*, 2004).

Similarly, after part of the colony or genet is damaged, unaffected units are able to contribute resources to facilitate the regrowth and healing of the affected units. Thus, the health, proximity, and connectivity of neighboring units may influence the rate of recovery of the entire genet. For example, shoots of clonal plants with intact connections to other shoots receive nutrients from these other shoots and recover from defoliation better than those with severed connections (Schmid *et al.*, 1988). Additionally, damaged units with many healthy neighbors should have a greater healing potential than damaged units with few healthy neighbors. In fact, some researchers advocate the use of lesion healing rate as an indicator of the health of a colony (e.g. in corals Fisher *et al.*, 2007). In some cases, though, damage might be too great and/or the condition of the colony too poor; thus, reallocation or resources to the damaged region could harm the colony (Hellström *et al.*, 2006).

The implications of partial damage and ramet connectivity in a modular organism is especially well illustrated in corals, which are colonial, modular organisms composed of interconnected polyps. Corals are damaged by a diversity of factors, such as storms, pathogens and predators (corallivores), and these factors often result in the partial death of the colony and/or the loss of biomass. For example, different species of corallivores consume different parts of the coral (Rotjan & Lewis, 2008), including mucus (consumed by Alpheid shrimp), tissue (consumed by corallivorous butterflyfishes and gastropods such as *Coralliophila spp.* and *Drupella spp.*), or both the tissue and the skeleton (consumed by triggerfish and par-

rotfish). Lesions created by these different predators affect coral skeletal growth and tissue regeneration differently; excavating predators create the most damage, while browsing predators that consume only mucus have the least effect (Cameron & Edmunds, 2014).

Lesions can provide substrate for competitors to settle or entry points for disease (Katz *et al.*, 2014). Thus, rapid healing of lesions is essential for the continued growth and survival of the colony. Corals can respond to damage by rapidly regenerating tissue to cover a lesion from connecting areas of the coral (DAngelo *et al.*, 2012). Newly regenerated tissue usually extends out from the border of the damaged area (although if a coral has deep, healthy tissue that was undamaged, new tissue might appear to come from the interior of the lesion). This regrowth of tissue is facilitated by the gastro-vascular canals through which polyps share nutrients. In *Favia favus*, the entire colony participates in the regeneration of damaged tissue (Oren *et al.*, 1997). In other corals, there is a limited distance over which coral polyps can share nutrients. For example, in massive Porites, feeding by the coralivore *Coralliophila violacea* creates carbon sinks in which carbon is translocated to where the snail is feeding, but this effect is limited to polyps within 5 cm of the damage (Oren *et al.*, 1998). Translocation, as well as other factors, can affect the rate of regeneration and the total amount of tissue that is healed (see Henry & Hart, 2005). For example, smaller lesions and lesions with a greater perimeter to area ratio heal more quickly and completely than larger lesions and lesions with a smaller perimeter to area ratio (e.g. Meesters *et al.*, 1997; Oren *et al.*, 1997; Van Woesik, 1998), presumably because areas heal faster when they have a greater number of neighboring, healthy polyps.

On reefs, corals are affected by a diverse community of predators, thus this community of predators is likely to create patterns of damage on a coral with cascading effects on coral regeneration and skeletal growth. For example, the proximity of neighboring lesions will likely influence the amount of resources available to regenerate coral tissue at a focal lesion. Therefore lesions that are clustered or aggregated will be located nearer to other lesions, and thus will likely have lower regeneration rates than isolated lesions (Welsh *et al.*, 2015).

Additionally corals with lesions that are dispersed might heal lesions more quickly, but temporarily allot fewer resources to the skeletal growth of the colony, leading to a trade-off of increased tissue regeneration and decreased coral skeletal accretion. Finally, by heavily depleting a smaller pool of resources, aggregated damage might have longer-term effects on skeletal growth. If areas far from lesions grow normally, this could lead to differential growth within a colony which might alter the morphology of the coral. While many studies document the role of various factors in regeneration and link these factors to the availability of resources that can be shared throughout the colony, none explicitly consider the how the spatial patterning of damage influences the response of the coral.

Thus, the spatial patterning of lesions is an unexplored, but potentially important factor, that can influence the ability of coral to recover from damage. Therefore, I designed a study to address the following questions. 1) How is coral damage distributed on the reef (e.g., are lesions spatially aggregated or overdispersed)? 2) How does lesion proximity affect tissue regeneration and skeletal growth? 3) How does location on the coral colony and distance between lesions affect spatial patterns of coral growth? 4) How does chronic damage affect coral morphology?

3.3 Methods

3.3.1 Field surveys of coral damage

I quantified the spatial patterns of coral damage by conducting photoquadrat surveys in the backreef of the north shore of Moorea, French Polynesia, where massive *Porites* was the dominant coral. At each of two sites on either side of Cooks Bay, I haphazardly selected two large, focal corals. The backreef of the north shore of Moorea has a strong, unidirectional flow running from the reef crest to the shore. Therefore, I ran four 25m transects in the cardinal directions from the bommie to capture variation both parallel to and perpendicular to flow. Along each transect, I randomly selected 10 locations where I took a photograph of

a .25m by .25m quadrat. I excluded photos that were not dominated by live massive Porites coral, or that were not in focus. This yielded 95 photoquadrats.

In each photograph, a technician identified lesions (areas of visible skeleton within continuous coral tissue), and circled each lesion using using Image J. For each lesion, I quantified its area and perimeter, and I obtained the x-y coordinate of the lesion centroid (i.e. center). Two technicians analyzed images, but there was no detectable effect of technician identity, so I combined all data in the final analyses with a total of 3957 lesions.

With the data from these surveys, I quantified: 1) the spatial distribution of lesions among quadrats; 2) the spatial distribution of lesions within quadrats; and 3) the shape (area:perimeter) of the lesions. To determine how lesions were distributed among quadrats, I modeled the counts among all quadrats using generalized linear models relating the number of lesions to the area of coral in the quadrat and including site as a factor to control for differences in corallivore communities or other damaging factors) that might differ between the two sites. I fit both Poisson and Negative Binomial distributions and compared the models using AIC. I also tested for statistical over-dispersion using a test developed by Cameron & Trivedi (1990), and examined the distribution of lesions across sites by comparing each of the sites sampled using a Tukey post-hoc test.

To study spatial point patterns of lesions within a quadrat, I used the Clark-Evans nearest neighbor method (Clark & Evans, 1954), which compares the observed mean nearest neighbor distance to that expected under complete spatial randomness, and classified each quadrant as either "clustered", "random" or "even". Lesions with with "clustered" would have more lesions in close proximity than lesions in quadrats with "even" lesion distributions. I also examined the distribution of nearest neighbor distances between lesion centroids across all quadrats. To describe the shape of the lesion, I compared the area of the lesion to its perimeter. To do this, I fit the equation $P = \pi\sqrt{2}\sqrt{\frac{A}{c\pi} + \frac{A}{\pi}}$, where P is the perimeter, A is the area of the lesion, and c relates the width and height of the ellipse. Values of c near 1 indicate circular lesions (relatively equal heights and widths and a high area:perimeter

ratio). Larger values of c indicate a narrower lesion (unequal heights and widths and a lower area:perimeter ratio).

3.3.2 Effect of damage distribution on lesion healing and whole colony growth

I conducted a field experiment to determine how the spacing between lesions affected lesion healing (i.e. tissue regeneration) and skeletal growth. I collected 40 small massive Porites colonies without existing damage near the reef crest on the north shore of Moorea, French Polynesia, placed them in individual ziplock bags, transported them to the Richard B. Gump South Pacific Research Station, and kept them in a flow-through seawater table. Each coral was attached to plastic mesh with Z-Spar marine epoxy. After 24 hours, I determined the buoyant weight (Davies, 1989) and randomly assigned each coral to one of four experimental treatments: no damage, or two lesions separated by either 1.2 mm, 3.5 cm, or 6 cm. I used a waterpik and 1 cm diameter circular stencil to make lesions on the tops of the corals and removed all tissue from within the lesion area. I handled all corals similarly regardless of treatment. I weighed corals after damaging to ensure that their mass was the same as prior to damage.

I deployed corals in the field by mounting them to cinderblocks and arranging them in a line perpendicular to the current. Corals were blocked in space and time, with one replicate of each treatment in each block. After 20 days and 39 days, I returned corals to the lab and weighed and photographed them. I analyzed photographs using Image J to document changes in lesion size at each timepoint. To determine the effects of damage on coral growth, I tested three hypotheses with a priori orthogonal contrasts at each time point: 1) Is there a significant effect of damage on coral growth? (undamaged corals vs. corals that received damage); 2) Do adjacent lesions respond differently than lesions that are separated? (corals with centers separated by 1.2 cm vs. corals with lesions with centers separated greater than 3.5 cm) 3) Does the degree of spacing between non-adjacent lesions influence healing or

growth? (corals with lesions centers separated by 3.5 cm vs. corals with lesions separated by 6 cm). I analyzed these contrasts in R using linear mixed effects models using the lme4 (Bates *et al.*, 2015) and lmerTest (Kuznetsova *et al.*, 2015) packages. I treated coral ID and block as random effects, time as a fixed effect, and tested the three hypotheses along with interactions. I removed one coral from the growth analysis because it was dropped during transport, and three corals from the lesion healing analysis due the poor quality of the photographs.

3.3.3 Effect of acute damage distribution on coral linear extension

While the previous field experiment tests the short term effect of damage on the skeletal growth of the entire colony, it does not provide long-term data on the colony growth response at different locations within the coral colony. Therefore, I conducted a second field experiment to test the effect of the distance between lesions on the growth of specific parts of the colony. I collected 60 juvenile massive *Porites* corals near the reef crest and returned them to the lab and damaged the corals with a waterpik (as in the previous experiment) with lesion centroids 1.2 cm and 3.5 cm apart. Control corals were handled similarly, but not damaged.

To create a reference line from which to measure spatially explicit growth, I placed the corals in a bath of 15 mg/L of Alizarin Red S dye for 2 days. Alizarin Red S is a vital stain that is incorporated into the skeleton as the coral polyps deposit calcium carbonate. Because I damaged the corals before placing them in the dye, the damaged regions did not incorporate large amounts of dye. After removing the corals from the dye, I attached them to a plastic base, buoyantly weighed and photographed each coral, and deployed them in the field as in the first experiment. I placed corals attach to cinderblocks in two sand flats on the backreef of the north shore of Moorea.

After one year, I collected the corals, re-weighed them, and placed them in a bleach solution to remove the tissue. I then used an angle grinder to cut the corals in half, ensur-

ing that the cuts passed through the locations. I sanded and dampened the corals before photographing them so that growth lines were clear. To analyze the images, I used Image J and marked damaged areas (indicated by a low density of Alizarin), traced the dye line, and traced the outer surface of the skeleton. The region between the dye line and the outer surface represented skeletal accretion during the course of the experiment. To measure growth, I traced >100 growth lines (each demarcating the edge of a polyp as it accreted calcium carbonate) from the outer edge of the dye to the edge of the colony. I modeled linear extension as a function location on the coral colony (e.g., edge vs. top of the colony). I did this by measuring the angle of growth relative to the vertical growth line. I included the individual coral as a random effect, fit several models (e.g. growth as linear, quadratic, vs. cubic functions of angle) (Table 3.3), and selected the best model using AIC. I analyzed all data in R (version 3.3.2).

3.3.4 Effect of chronic damage on coral morphology

To evaluate the effects of chronic damage on coral morphology, I compared long-term patterns of coral growth in regions near the corallivorous gastropod, *C. violacea* with coral growth in regions that lacked this predator. Oren et. al referred to this snail as a "prudent sessile feeder" because it feeds in one location for extended periods of time and creates carbon sinks (Oren *et al.*, 1998). I found 15 massive *Porites* corals on the north shore of Moorea, French Polynesia approximately 1m in diameter with *C. violacea* present. I randomly selected one snail on each colony, and found a similar location on the same colony with respect to height and flow that was at least 20 cm from the nearest snail. At each location, I quantified coral morphology, using a triangular piece of plexiglass (30 cm per side) with a 20 cm by 20 cm grid in the center. The grid contained 20 pre-drilled holes at random locations. The plate was centered at the snail (or control location) and laid flat upon the coral (resting on at least three points). I then measured the perpendicular distance from the coral to the plate and used the variance of these distances to quantify surface rugosity (i.e.,"bumpiness");

corals with high variances were more topographically complex than corals with low variance. I remeasured each coral after one year (using a nail as a reference point), calculated the change in variance and analyzed this change using a mixed effects model with coral as a random variable.

I also measured linear extension of each coral near and far from the snail, by inserting a nail at each location and measuring the amount of vertical growth between the initial and final surveys. While corals created new tissue and extended up the nail, I only measured the vertical extension based upon the surface of the adjacent colony. I tested for a difference in linear extension (with vs. without snails) by using a mixed effects model with coral as a random variable.

3.4 Results

3.4.1 Surveys of coral damage

Coral lesions were variable at multiple spatial scales. Across all spatial scales, there was significant over-dispersion ($Z = 1.92, P = .027$), indicating that lesions were spatially clustered. Additionally, lesion density varied among sites. The two sites east of Cook's Bay had significantly fewer lesions than one of the sites on to the west of Cook's Bay, but were not different from the second western site.

Coral lesions were common among all quadrats. The number of lesions per quadrat ranged from 2 to 611, with a mean of 42 lesions per quadrat, or from 0.01 lesions/ cm^2 to 0.96 lesions/ cm^2 with a mean of 0.12 lesions/ cm^2 (Fig. 3.2B). Among quadrats, lesions were very clustered, with some corals having extremely high densities of lesions, while many had lower levels (Fig. 3.2A). The model with the best fit for the number of lesions was the negative binomial fit including site (Table 3.1).

Within quadrats, lesions showed a variety of spatial distributions. Lesions on 12 quadrats indicated spatial clustering, 74 were indistinguishable from spatial randomness, and nine

had a demonstrably even distribution of lesions. Even though the spatial point pattern of lesions was variable, lesions were sufficiently dense (or clustered) to be in close proximity to other lesions. The centers of 75% of the lesions were <1.76 cm from the center of the nearest neighboring lesion (Fig. 3.2D). Scars were approximately elliptical with the major axis exceeding the minor one by 15% (Fig. 3.2B: $c = 1.15 \pm 0.01$; estimate $\pm$ SE), and this shape appeared to be independent of overall lesion size.

3.4.2 Effect of damage distribution on lesion healing and colony growth

Lesions farther apart showed increased rates of tissue regeneration (Fig. 3.3A). At the midpoint of the experiment, tissue regeneration was 81% higher on corals with lesion centers separated by at least 3.5 cm compared to lesions with centers 1.2 cm apart ($t_{23.1} = -2.8, P = 0.011$), however there was no difference between lesions with lesions separated by 3.5 cm and 6 cm ($t_{22.2} = 0.26, P = 0.8$). However these effects of lesion spacing, which were present at 20 days, were not detectable by the conclusion of the experiment (day 39, $t_{23.8} = 1.9, P = 0.068$).

Skeletal growth showed a slightly different pattern than tissue regeneration (Fig. 3.3B). Lesions did not significantly decrease skeletal growth relative to undamaged corals at the midpoint ($t_{31.2} = 1.8, P = 0.089$) or conclusion of the experiment ($t_{35} = -0.63, P = 0.53$). Additionally, spacing between lesions did not significantly change colony growth at either time point

3.4.3 Effect of acute damage distribution on coral linear extension

After one year, the linear extension of corals depended on both the position on the coral colony and the damage pattern of the coral. The model that best described the data included a quadratic and cubic term (Table 3.2) as the relationship between coral growth and the angle of growth decreased nonlinearly from the top of the coral. Corals without any lesions

(control) grew 1.37 cm at the top of the coral (i.e. 0°). For control corals, linear extension decreased 19% for polyps that were 45° from the top of the colony, and by 50% at the point where the coral extended horizontally (i.e. at 90°, Fig. 3.4).

Corals with lesions far apart (centers separated by 3.5 cm), showed only a 7% reduction in growth compared to the undamaged corals, and this reduction across the colony was not significant (Table 3.3, Fig. 3.4). In contrast, corals that had two lesions in close proximity of one another (lesion centers separated by 1.2 cm) showed a 26% reduction in growth coral on the top of the coral compared to control corals, but no difference beyond 45° (Fig. 3.4). Thus, the response of a coral to damage after one year depended on the spacing between lesions – effects were only apparent in the region of the damage and only when lesions were in close proximity to one another (i.e., within 2 cm).

3.4.4 Effect of chronic damage on coral morphology

I observed differences in the change in coral morphology based on the presence of the corallivorous snail, *C. violacea*. Near the snail, there was an increase in the topographic complexity, while in areas away from the snails, topographic complexity decreased ($t(28), P = 0.047$, Fig. 3.5A). Linear extension of coral was reduced by 68% in the presence of *C. violacea* ($t(18.5), P = 0.0023$, Fig. 3.5B). These results support the hypothesis that snails increase the heterogeneity of coral morphology by decreasing linear extension rates of the coral near where they feed and that this effect is quite localized (e.g., much smaller than the scale over which I measured topographic heterogeneity, 20 cm).

3.5 Discussion

The spatial patterning of lesions (i.e. how close lesions are to neighboring lesions) plays an important role in the recovery of a coral from damage. Lesions near one another heal more slowly than those farther apart (Fig. 3.3A) and over longer time periods (a year),

the spatial patterning of the damage differentially affect skeletal growth patterns across the surface of the coral. When lesions are close together, there is a local reduction in linear extension near the locations, while corals with lesions far apart only show a slight decrease in linear extension compared to corals without damage (Fig. 3.4). Finally, chronic damage, such as that produced by the corallivorous gastropod, *C. violacea*, not only reduces linear extension of coral, but also has the potential to affect the shape of corals, creating mosaics of depressed growth (where snails feed) and elevated growth (away from the corallivore), resulting in more topographically complex corals (Fig. 3.5). Because lesions are generally very near one another (Fig. 3.2), these findings suggest that the effect of coral damage is more severe than effects obtained from studies of single lesion, resulting in slower healing rates and longer term decreases in skeletal growth.

These results likely arise because resources are shared among polyps in a local neighborhood. The presence of nearby, healthy polyps facilitate the regeneration of tissue in areas that have been damaged. The ability of a polyp to contribute to the healing of a neighbor is likely affected by its proximity to the area of damage, as well as its own health, and the health of nearby polyps. These findings build on past studies. For example, lesions with a greater perimeter to area ratio heal more quickly than similarly sized lesions (but with small perimeters) because there is more healthy tissue bordering the lesion that can extend in and heal the lesion (e.g. Meesters *et al.*, 1997). Similarly, smaller lesions heal more quickly than larger lesions (see Henry & Hart, 2005). Previous studies have noted that lesions in clusters heal more slowly than single lesions (e.g. Welsh *et al.*, 2015), and lesions in higher numbers are more likely to grow in size. Of course, these results could simply result from more damage created when lesions are clustered. Here, I demonstrate that these results could arise from spacing, not just a higher proportion of damaged tissue. The spatial patterns of damage likely interact with characteristics of the lesion due to their combined effects on the ability of polyps to share resources. For example, previous studies demonstrate the effect of size and shape on healing (Meesters *et al.*, 1997; Oren *et al.*, 1997; Van Woesik, 1998), so it

is likely that corals with elliptical lesions far apart from one another will regenerate tissue much more quickly than lesions with circular lesions near to one another.

Other factors could affect the ability of units within a colonial organism to share resources. Other stressors (e.g. sedimentation, Sheridan *et al.*, 2014) can reduce the overall health of the colony and therefore decrease the resources that neighbors have available to heal damaged units, and would therefore decrease the ability of the colony to recover from a damage event. For example, studies tracking isotopes demonstrated that bleached corals have lower connectivity (i.e., fewer isotopes were transported across the colony), and lower rates of lesion healing, compared to unbleached colonies (Fine *et al.*, 2002).

While I failed to detect an effect of spacing on short-term coral calcification (see Fig. 3.3), other studies have detected a trade-off between skeletal growth and tissue regeneration (see review in Henry & Hart (2005)), suggesting that reallocating resources from calcification to tissue regeneration reduces the coral's ability to calcify for a certain time period following regeneration. However, other studies demonstrate compensatory growth in corals following damage (e.g. Jayewardene, 2010). I observed neither, although there was a trend towards a trade-off (Fig. 3.3B).

I did detect a decrease in linear extension near lesions after one year for lesions located near one another (Fig. 3.4), but this effect was localized to the area near the s, whereas corals with lesions far from one another showed no significant difference from corals without any s. This suggests that longer-term effects of damage might be limited to certain spatial distributions of damage due to limitations of the coral to transport resources. Many lesions are created by consumers that target certain areas of the coral. For example, some fish frequently feed on tops of colonies (Rotjan & Lewis, 2008; Welsh *et al.*, 2015)), and smaller organisms, such as the gastropod *Drupella cornus* juveniles seek out crevices for protection from predators (Schoepf *et al.*, 2010). Other types of organisms do not feed on coral, but inflict other types of damage. Macro-algae abrade coral (Jompa & McCook, 2003), and vermetid gastropods deploy nets that contact coral and reduce growth (Shima *et al.*, 2010,

2015). In these cases, damage is more likely to occur on more "peaked" parts of the coral, where contact with foliose algae or vermetid nets is more likely. Depending on the location of damage, these organisms could facilitate either topographically diverse corals (e.g. as I observed for *C. violacea*: Fig. 3.5) by decreasing growth in crevices, or could create less topographically complex corals by decreasing growth on the peaks. These effects could not only alter coral growth and morphology, but the effects could feed back to influence organisms that exploit coral morphology to shelter from predators.

3.6 Tables and Figures

Model	Distribution	log-likelihood	AIC	Δ AIC
Coral Area + Site	Negative Binomial	-427.02	864.04	0
Coral Area	Negative Binomial	-435.50	875.01	10.97
Coral Area + Site	Poisson	-1783.40	3576.80	2712.77
Coral Area	Poisson	-2081.63	4167.26	3303.23

Table 3.1: Model Selection Information - Field Surveys. Models were fit for the number of coral lesions at the quadrat level. The best model included both the area of live tissue, site, and an overdispersion term indicating spatial clustering.

Model	log-likelihood	AIC	Δ AIC
Treatment+Angle^2 + Angle^3+Treatment*Angle^2 + Treatment*Angle^3	301.05	-580.10	0
Treatment+Angle + Angle^2 + Angle^3+ Treatment*Angle + Treatment*Angle^2 + Treatment*Angle^3	285.92	-543.84	36.26
Treatment+Angle+Treatment*Angle	279.70	-543.41	36.69
Treatment+Angle + Angle^2+Treatment*Angle + Treatment*Angle^2	281.53	-541.06	39.04
Treatment+Angle^2+Treatment*Angle^2	115.28	-214-57	365.53
Treatment+Angle^3+Treatment*Angle^3	-195.02	406.04	986.14

Table 3.2: Model Selection Information - Effect of Lesion Spacing on Linear Extension

Effect	Value	Std. Error	t-value	p-value
Intercept	1.37	0.47	29.50	< 0.0001
Near	-0.37	0.052	-7.16	< 0.0001
Far	-.10	0.048	-2.08	0.038
Angle^2	-0.00016	< 0.0001	-26.05	< 0.0001
Angle^3	< 0.0001	< 0.0001	15.97	< 0.0001
Near*Angle^2	0.0001	< 0.0001	9.15	< 0.0001
Far*Angle^2	-0.00001	< 0.0001	-1.34	0.18
Near*Angle^3	> -0.0001	< 0.0001	-7.72	< 0.0001
Far*Angle^3	< 0.0001	< 0.0001	2.51	0.01

Table 3.3: Parameter Estimates - Effect of Lesion Spacing on Linear Extension (Table 3.2)

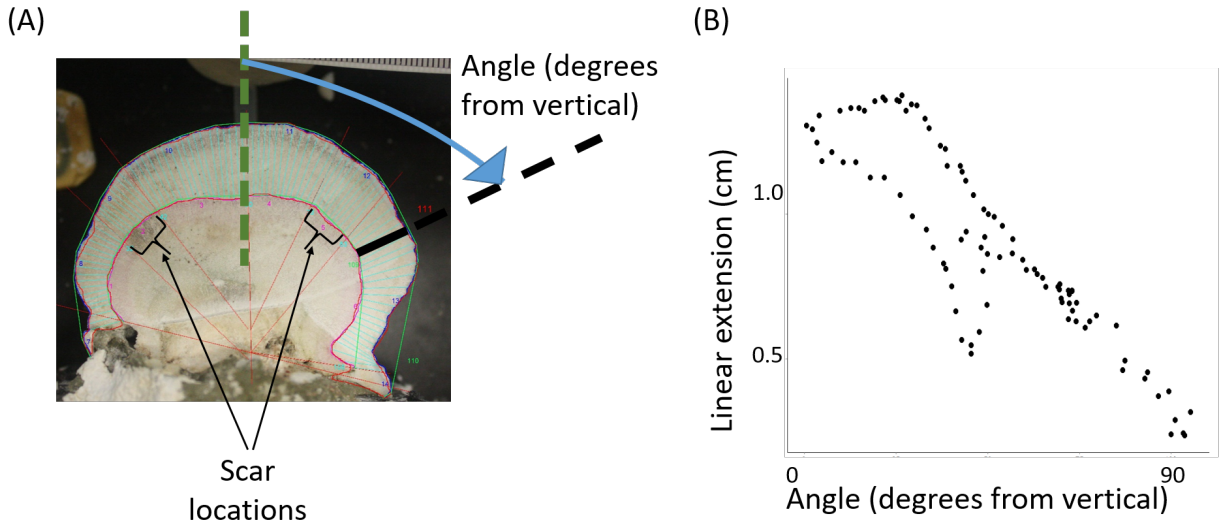

Figure 3.1: Example of an analyzed image (A) and corresponding growth data (B) for a coral included in the Alizarin study. In panel (A), green lines indicate growth lines out from the Alizarin stain (pink line) to the edge of the coral. Scar locations are indicated (marked by red lines in the original image). I analyzed growth as a function of angle (x-axis in panel B, shown in panel A)

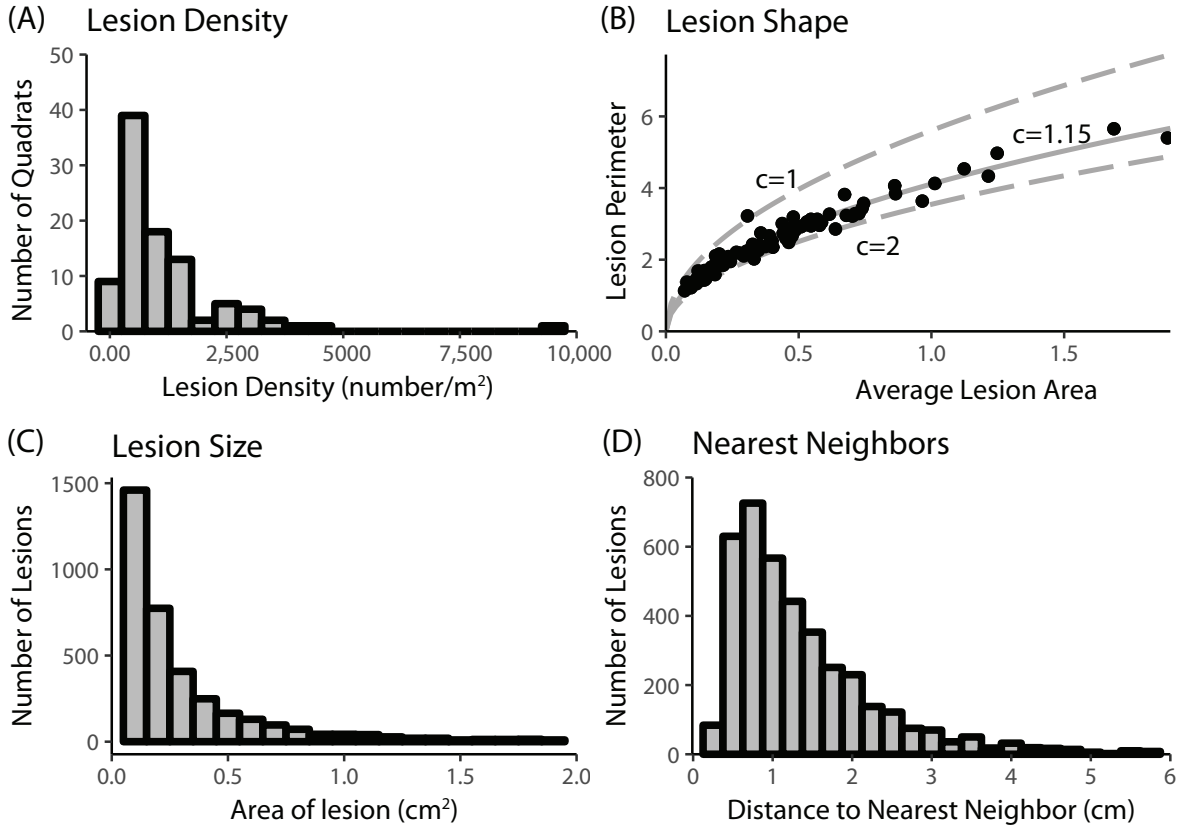

Figure 3.2: Spatial patterning of coral lesions, giving A) the density (number of lesions per live area of coral) among quadrats, B) the shape of the lesions, C) lesion size, and D) the distance to the nearest neighboring lesion. In panel B), the dashed lines represent predictions for c=1 (equal elliptical axes), and c=2 (the major elliptical axis is twice that of the minor). Results are based on 97 quadrats (panels A, B), and 3957 lesions (panels C,D).

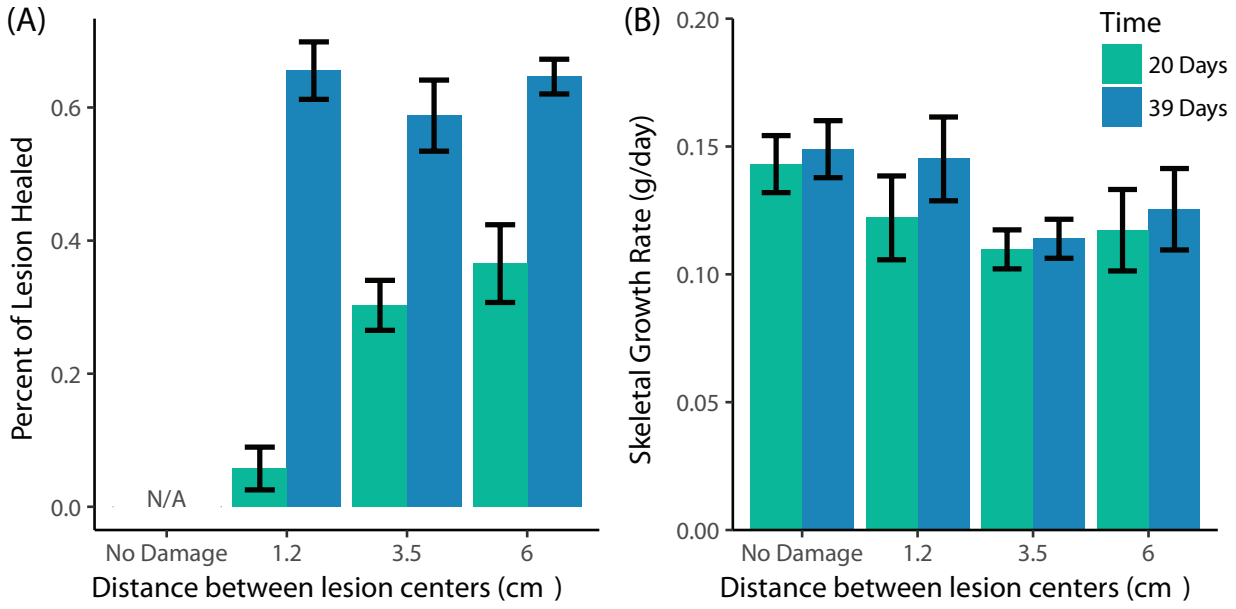

Figure 3.3: Effect of lesion spacing on A) percent of lesion healed and B) coral skeletal growth rate and after 20 (green bars) and 39 (blue bars) days: A). The percent of lesion healed was significant between corals with lesions with centers separated by 1.2 cm and by at least 3.5 cm after 20 days, but not 39. There were no significant effects between distance between lesion centers and skeletal growth. Values plotted are fitted values from the mixed effects model and error bars represent $\pm$ 1 SE

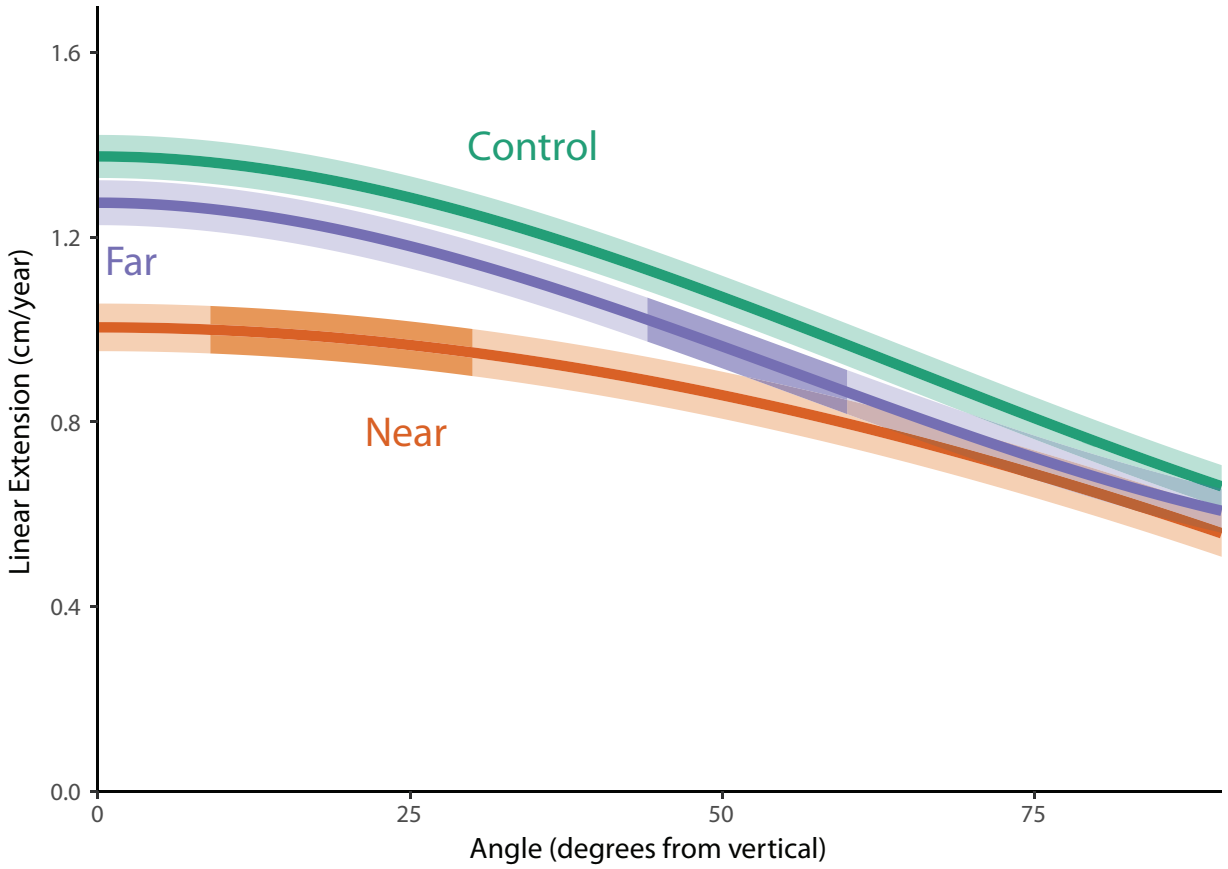

Figure 3.4: Effect of lesion spacing (Control: no lesions, Near: lesion centers separated by 1.2 cm, Far: 3.5 cm) on coral growth rates at different locations on the colony (represented by the angle of growth). Lines give best fits,lightly shaded regions indicate ± SE, and darkly shaded regions indicate the average locations in each treatment (corals had two lesions, with one lesion located to each side of 0°).

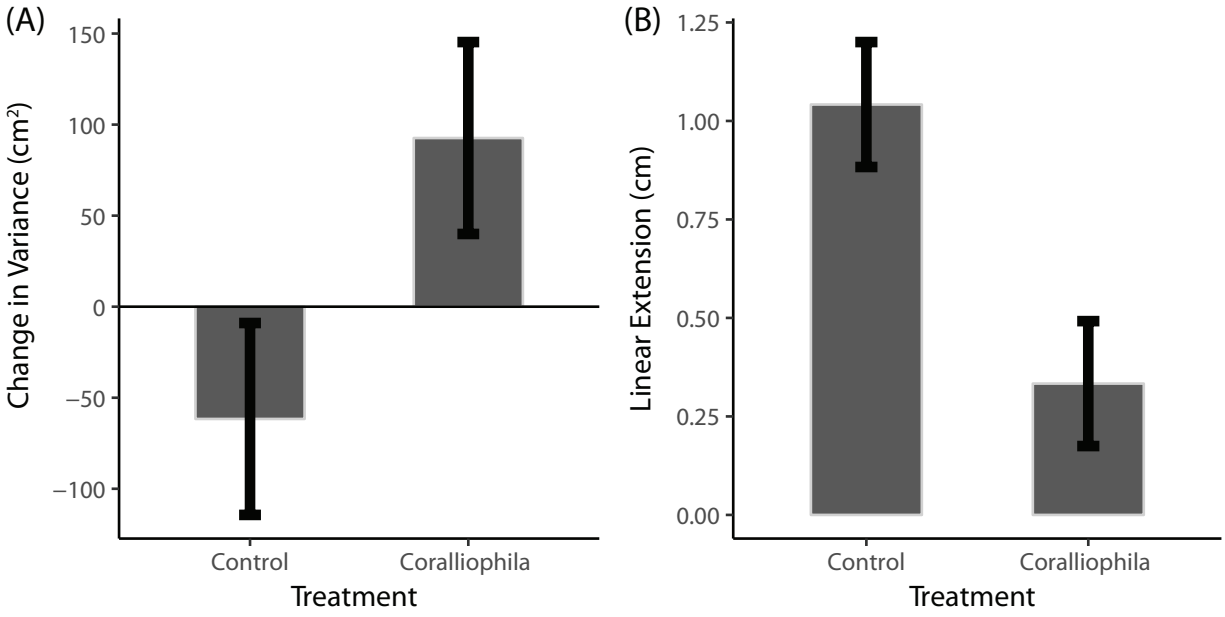

Figure 3.5: Effect of damage on coral morphology. A) *C. violacea* are associated with bumpier morphology (higher variance), and facilitate crevices by decreasing linear extension near their feeding site (B). Error bars represent ± 1 SE.

Chapter 4: Landscape configuration drives persistent spatial patterns of occupant distributions[1]

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4.1 Abstract

The density of organisms varies among discrete habitat patches, often due to inherent properties of a patch (e.g. due to factors that affect colonization or within patch survival). However, density within a patch could also be driven by the density of adjacent patches, even if they are identical in quality, because these patches draw colonists away from other habitat (a phenomenon we call propagule redirection). Here, we explore the consequences of neighboring patches on colonization (i.e. settlement) of organisms with a dispersive larval stage and the long-term implications of these heterogeneous colonization patterns. We determine the situations in which heterogeneous settlement patterns are retained and the conditions under which the settlement patterns fade to a homogeneous distribution. Heterogeneous patterns of settlement are retained at equilibrium when there is a low supply of settlers, weak density–dependent survival after settlement, and longer times between settlement events. We illustrate the application of this approach to a reef fish system. We show that the spatial variation produced by propagule redirection (among identical patches) is comparable to spatial variation expected when patches vary in quality. Thus, variation in density arising from the spatial patterning of otherwise identical habitat can play an important role in shaping long-term spatial patterns of organisms occupying patch habitats.

4.2 Introduction

Many landscapes are fragmented, either naturally or through human activity, yielding a system of patches heterogeneous in size and connectivity. Accordingly, the distribution of organisms that occupy these patches is also often heterogeneous, with high densities of organisms in some patches, but low densities in others. Differences in the density of organisms among patches are often attributed to differences in the intrinsic properties of a patch that affect the colonization of the patch or survival within the patch. For example, organisms

differentially colonize patches based on size (Sih & Baltus, 1987), quality (Tolimieri, 1995; Holbrook *et al.*, 2000), micro-environmental conditions (Lecchini *et al.*, 2003), the presence of predators (Wesner *et al.*, 2012), the presence of conspecifics (Stamps, 1988) and the presence of heterospecifics (Mönkkönen *et al.*, 1990). Properties extrinsic to the patch, such as the surrounding matrix (e.g. Gustafson & Gardner, 1996; Ricketts, 2001) or the spatial configuration of patches (e.g. MacArthur & Wilson, 1967; Stier & Osenberg, 2010) also lead to variation in density through their effects on colonization. In such cases, intrinsically identical patches may harbor very different densities of organisms (e.g. Stier & Osenberg, 2010).

The population level effects of differential patterns of colonization of patches within a network is well articulated in the metapopulation and metacommunity literature (e.g. Hanski, 1994; Gonzalez *et al.*, 1998; Koelle & Vandermeer, 2005). In these studies neighboring patches increase colonization (e.g. Hill *et al.*, 1996; Eaton *et al.*, 2014) by providing propagules. However, this literature has rarely examined how otherwise hospitable neighboring patches might reduce colonization.

In contrast to the metapopulation literature, the foraging literature has explored possible positive and negative effects of patch density and configuration (e.g. Ryall & Fahrig, 2006). For example, if a plant (i.e., "patch") has few flowers, it may not attract pollinators. But, if that plant is near another, then the combined floral display may recruit pollinators into the general area, increasing the visitation rate of pollinators to plants and individual flowers (reviewed in Mitchell *et al.*, 2009). However, plants with many neighbors may have decreased per-capita visitation rates from pollinators because flowers compete with one another for the potentially limited supply of pollinators (see review by Morales & Traveset, 2008). Similar beneficial and deleterious effects of patch density observed in these behavioral studies may also exist at the population level. Thus, models of spatial population dynamics that incorporate potential deleterious effects of neighboring patches might inform our understanding of the dynamics of organisms that occupy heterogeneous landscapes.

Metapopulations and foraging (e.g., pollinator) systems form a dichotomy with respect to the scale of migration and patch size. In metapopulations, individuals can be born, mature, and reproduce within a single patch; migration rates among metapopulations also are typically low. In contrast, foragers must visit multiple patches within a short time-frame–no single patch is large enough to support the dynamics of an entire population. Many systems are intermediate with respect to these extremes.

For example, adult aquatic beetles colonize ponds (after emerging from terrestrial pupation sites) where they reproduce and frequently remain for the rest of their life. Colonization rates in one pond (a patch) could be influenced by its own characteristics as well as the landscape of ponds in which the focal pond occurs. Experimental tests (Resetarits & Binckley, 2009, 2013) demonstrated that beetle colonization was reduced in ponds that contained predators, demonstrating an effect of patch quality. However, colonization also was reduced in predator-free ponds if they occurred near ponds that contained predators, showing that variation in quality of neighboring ponds also affected colonization. The experiments conducted by Resetarits and Binckley also manipulated the total number of ponds at a site. In contrast to the results for pond quality, they did not find any discernible effect of patch number on per pond colonization. Thus, in this system, it was the quality of the patch or the landscape (i.e. in terms of predator density) and not simply the availability of ponds (i.e. landscape configuration) that drove colonization patterns

In contrast, Stier and Osenberg (2010) varied landscape configuration of corals (keeping intrinsic properties of corals the same) and found that the presence of neighboring corals reduced the per-coral colonization rates of focal coral patches by larval reef fish by over 70%. Although there was an increase in total colonization with increased habitat, it was not proportional to the total amount of habitat. Thus, neighboring patches (i.e. corals) redirected colonists from focal patches, resulting in propagule redirection (also see: Carr & Hixon, 1997; Osenberg *et al.*, 2002a; Morton & Shima, 2013). As a result of this "competition" among neighboring patches for colonists, the addition of new patches to a region may limit

the potential increase in total colonization. At the extreme, there may be no increase at the regional level. These two examples (beetles and fishes) illustrate how the strength of propagule redirection can vary across systems. Reef fish represent one point near the end of this redirection spectrum, in which strong redirection results in spatial heterogeneity in colonization due to the presence or absence of neighboring patches (Stier & Osenberg, 2010), whereas aquatic beetles represent the other end of the spectrum, in which weak redirection leads to colonization patterns that are independent of the local landscape configuration.

Variable colonization driven by the combined effects of the spatial distribution of (otherwise identical) habitat patches and propagule redirection can have long-term consequences for population dynamics (Stier & Osenberg, 2010), especially for species in which colonists remain associated with habitat patches for most of their life history (as is the case for the beetles and reef fish). While the strength of propagule redirection (and other processes that affect colonization) establishes the initial spatial pattern of colonists, the degree to which these patterns propagate over longer time frames will depend on the post-colonization processes. These post-colonization processes will be affected by within-patch factors such as the local environment and density dependence. For example, mortality of newly settled fish is often density–dependent (Osenberg *et al.*, 2002b; Hixon & Jones, 2005; White *et al.*, 2010), with negative density dependence possibly arising through competition for refuges from predators on the interior of the coral colony (e.g. Holbrook & Schmitt, 2002). When density–dependent mortality is strong, the spatial variation in colonization resulting from propagule redirection may be eliminated as large cohorts are quickly and disproportionately reduced in density relative to patches with few colonists. In contrast, when density–dependent mortality is weak, we expect the spatial patterns established during colonization to persist through the post-colonization phase.

Here, we develop a model to investigate how spatial patterns can be created in a landscape consisting of identical habitat patches. In particular, we investigate how propagule redirection leads to different long-term spatial patterns of occupants that depend on

the proximity of neighboring patches. We also consider how density–dependent mortality, density–independent mortality, colonist supply, and colonization frequency affect the long-term spatial patterns that result from different forms of propagule redirection. Finally, we apply our framework to a reef fish system, using data to parametrize our model. Because our model assumes homogeneous quality, we also contrast predictions derived from our propagule redirection model to one including habitat quality.

4.3 Methods

Although our approach is general, we develop it with reference to corals (habitat patches) and coral occupants (such as fish). This is a demographically open system where colonists to a patch are not the progeny of the local adults. In most coral reef systems, late-stage larvae (produced by relatively distant adults, but see Jones *et al.*, 2009) enter shallow reef areas from the pelagic zone, and settle from the water column into a coral. After settlement (i.e. colonization), many coral occupants have limited ability to move to other corals. Thus, the occupants often reside within the colonized coral for the entire juvenile and adult portions of their life cycle. Extrapolation to other contexts can be done by slightly redefining life stages. For example in the beetle example, adults, rather than larvae, colonize patches.

Our model includes three components, each of which corresponds to sequential events that start with a larval fish's arrival on the reef and end with its death: 1) potential settlers (i.e. colonists) arrive at random locations uniformly distributed in the landscape; 2) corals (i.e. habitat patches) compete for settlers based on the spatial distribution of corals within the landscape; 3) settlers that successfully arrive at a coral survive (or die) over the inter-pulse interval. The first two phases collectively define the "settlement phase," while the third defines the "post–settlement phase." We assumed settlement events are discrete, pulsed events and occur at regular time intervals, mirroring the life history of many marine organisms, while survival occurs continuously in time (see Fig. 4.A2 in Appendix A). Corals

in the landscape are identical in all ways (e.g. size and quality), except for their spatial proximity to other corals. The matrix surrounding the corals also is assumed uniform.

As a result, spatial variation in expected patch occupancy is only driven by the effects of landscape configuration (i.e. the number of nearby patches) on settlement. However, some additional variation arises because each model component is intrinsically stochastic, leading to variation in the spatial arrangement of corals, larval density, and the survival of fish. Therefore, we developed a stochastic model of settlement and post-settlement dynamics on a spatially explicit landscape. We also developed a deterministic approximation to the stochastic model to gain an understanding of the role of specific processes. We start with a general description of the model.

4.3.1 Settlement processes

Settlement (colonization) events occur in a landscape of identical, discrete, circular habitat patches (corals). Settlers (larval fish), arrive in the landscape, perceive signals from coral patches, and choose a coral based upon these signals (e.g. Lecchini *et al.*, 2005; Dixson *et al.*, 2014). Potential settlers then travel to the selected coral but incur mortality in transit. Because reef fish settlement events are pulsed and often linked with the lunar cycle (e.g. Robertson, 1992), we model settlement events discretely in time.

For a settlement event, we assume that the density of arriving larvae is L, and the initial locations of the larvae are independent and uniformly distributed over the landscape: i.e. the distribution of potential settlers is a spatial Poisson point process with density L. The number of settling fish larvae in the landscape then has the distribution $S \sim \text{Pois}(L|\mathcal{O}|)$, where $\mathcal{O}$ is our notation for the landscape, and $|\mathcal{O}|$ is its area.

Let $\{(x_i, y_i)\}_{i=1}^{K}$ be the locations of K corals in the landscape. Each coral has radius R, signal magnitude m, and signal degradation rate ρ. On the coral, the signal has strength m, but as distance from the coral increases, we assume the strength of the signal decreases exponentially. Therefore, a potential settler arriving at location (x, y) perceives a signal from

coral i of magnitude

$$\Lambda_i(x, y) := me^{-\rho d_i(x,y)} \tag{4.1}$$

where $d_i(x, y)$ is the distance from the potential settler's location (x, y) to the edge of coral i (the center of the coral is located at (x_i, y_i)):

$$d_i(x, y) := \max(0, \sqrt{(x - x_i)^2 + (y - y_i)^2} - R). \tag{4.2}$$

A settler selects a coral with a probability that is equal to the relative strength of the signals: a settler arriving at position (x, y) will choose coral i with probability $\Lambda_i(x, y)/\left(\sum_j \Lambda_j(x, y)\right)$, so long as the total signal strength exceeds a detection threshold, θ. Once a settler chooses a coral, it must survive the travel to the selected coral. We assume that the mortality rate and velocity during travel is constant, so that the probability of survival starting at location (x, y) and traveling to coral i is $e^{-\mu d_i(x,y)}$.

To obtain the settlement to a focal coral, we determine λ_i, the number of settlers that select and survive the travel to coral i. If there are S settlers in the entire landscape-wide cohort, then λ_i is a Binomial random variable where S is the number of "trials," and p_i is the "success probability" that, given a settler detects enough signal to choose a coral, a given settler will choose, and survive travel to, coral i:

$$p_i = \frac{1}{|\mathcal{O}|} \iint_{\mathcal{O}} \frac{\Lambda_i(x, y)}{\sum_j \Lambda_j(x, y)} e^{-\mu d_i(x,y)}\, dx\, dy. \tag{4.3}$$

Because we assume that the settler choices and survival are independent of each other, the number of arrivals at the focal coral is a thinned Poisson random variable, $\lambda_i \sim \text{Pois}(L|\mathcal{O}|p_i)$. In subsequent sections, when referring to the parameters of a focal coral, we will suppress the dependence on the index i in the notation. Thus, a coral with n residents transitions to having $n + \lambda$ residents when $t = \gamma k$ where γ is the time between settlement pulses and k is an index that keeps track of subsequent cohorts.

4.3.2 Post-settlement processes

In contrast to the periodic structure of the settlement pulse events, post-settlement deaths occur on each coral continuously in time due to density–independent and density–dependent mortality. We used a stochastic version of a framework previously used successfully to model reef fish recruitment dynamics (Osenberg *et al.*, 2002b; Shima & Osenberg, 2003). The number of residents or fish in a coral patch at a time t, denoted $N(t)$, decreases as fish die due to density–independent mortality α, and density–dependent mortality β. We assume that mortality rates and density–dependent effects are independent of fish age, and thus aggregate all cohorts together into a single measure of density, N.

Because many corals have very low numbers of residents and therefore demographic stochasticity has a potentially large effect, we used a continuous time Markov chain. To simulate mortality, we used Gillespie's method (Gillespie, 1977). If a coral has n occupants, the time until the next one dies (i.e. the coral has $n-1$ occupants) is an exponential random variable with rate parameter $\alpha n + \beta n^2$. When populations are large, this discrete process is well approximated by a continuous dynamic (see Fig. 4.A2 in Appendix 4.8.1).

4.4 Analysis and Results

We took two complementary approaches to analyze and interpret the stochastic, spatially explicit model and evaluate the effects of propagule redirection under different landscape configurations. First, we simulated both settlement and post-settlement dynamics using the parameters described above. Second, we constructed a deterministic approximation of the stochastic model and explored a non-dimensionalized parameter space. Below we explain each approach and provide the results of our analyses. Finally, we explore the ramifications of propagule redirection in a realistic system and compare the effects of propagule redirection to variation in habitat quality.

4.4.1 Simulations of the full stochastic model

Settlement processes

To determine how spatial patterns might be produced (or diminished) from spatial variation in settlement arising from propagule redirection within a heterogeneous landscape, we implemented a fully stochastic version of the model and manipulated the landscape configuration. We used three types of landscapes: landscapes with evenly distributed patches, randomly distributed patches, and clustered patches. We created evenly distributed landscapes (Fig. 4.1A) using Spatstat's simple sequential inhibition model (Baddeley & Turner, 2005), random landscapes (Fig. 4.1B) using a spatial Poisson process, and clustered landscapes (Fig. 4.1C) using a preferential attachment model (Hein & McKinley, 2013).

For each landscape type, we created 100 replicate landscapes each with 40 corals for each landscape configuration. For each replicate landscape, we simulated settlement and post settlement dynamics using the parameters in Table 4.1. For each of the 3×100 runs, we simulated dynamics for at least 50 settlement events (the actual number was randomly selected to fall between 50 and 150). We recorded the mean number of settlers on each coral, λ, and number of occupants on each coral after the last settlement event and subsequent post-settlement dynamics. To reduce edge effects, we only analyzed the interior by creating a buffer of 10% of the landscape width on all sides.

The three landscapes (Fig. 4.1A,B,C) resulted in different mean settlement rates. The clustered landscape (Fig. 4.1C) had a lower mean settlement rate (Fig. 4.1F) compared to the more evenly distributed landscapes (Fig. 4.1A,B) because fewer fish were able to survive the transit to a coral when the corals were clustered: i.e. the average distance to a coral was greater in the clustered landscape than the even landscape. The three landscapes (Fig. 4.1A,B,C) differed in the resulting spatial variation in settlement. Landscapes with evenly distributed patches (Fig. 4.1A,D) had very little variation in settlement because all corals had similar neighborhoods and thus similar degrees of propagule redirection. In

contrast, corals in clustered landscapes had the greatest amount of variation in settlement (Fig. 4.1F) because some corals were surrounded by other corals on all sides, whereas some corals had relatively few neighbors (i.e. compare the red vs. blue points in Fig. 4.1C,F). Landscapes with complete spatial randomness were intermediate in variance. Across all of the simulated landscapes, the maximum and minimum settlement rates varied 2.9 ± 0.04 SE -fold, 3.4 ± 0.06 SE -fold, and 4.7 ± 0.11 SE -fold for the landscapes with evenly distributed, randomly distributed, and clustered patches, respectively.

Post-settlement processes

This heterogeneity in settlement resulted in spatial variation in the long-term number of occupants (Fig. 4.1G,H,I): i.e., the density of occupants was more heterogeneous in the clustered landscapes (compare Fig. 4.1G with Fig. 4.1I). The results, depicted in Fig. 4.1, arose for one particular set of landscape configurations and parameter values. We therefore conducted the simulations for two levels of density dependence, moderate and strong ($\beta =$ 0.056 and 0.00056: see below, "Application to a realistic system"), and summarized the results using an index of dispersion (variance/mean) for each life stage (i.e. larvae, settlers, and occupants).

The index of dispersion for larvae was always one, by definition (indicating a random distribution, Fig. 4.2A). Although larvae were randomly distributed, the spatial dispersion of settlers was more heterogeneous than a Poisson spatial process in all landscapes due to propagule redirection. The greatest heterogeneity occurred in the clustered landscapes (Fig. 4.2B, also seen in Fig. 4.1). Because settlement was unaffected by post-settlement processes, dispersion of settlers did not vary across the two levels of density dependence. The translation of this spatial heterogeneity in settlement to long-term spatial variation in occupant density depended upon the strength of density–dependent mortality (Fig. 4.2C). When density dependence was moderate ($\beta = 0.00056$), there was demonstrable variation in density in both the random and clustered landscape configurations, although heterogeneity

was greatest in the landscapes with clustered corals and when corals were evenly distributed, the little heterogeneity established during settlement faded due to the small amount of density-dependent mortality in post-settlement dynamics (Fig. 4.2C). This pattern mirrored that seen in the settlement stage (compare Fig. 4.2B and C). In contrast, when density dependence was strong ($\beta = 0.056$, spatial variation was eliminated, even when settlement was highly heterogeneous (Fig. 4.2C). At the most intense levels of density dependence, the index of dispersion was < 1, indicating that densities were more homogeneous than expected given uniformly random larval rain.

In summary, these simulations show that spatial heterogeneity in occupants can be created in a landscape of otherwise identical habitat patches, if: 1) there is propagule redirection (i.e. habitat competes for colonists: Stier and Osenberg, 2010); 2) the distribution of patches in the landscape creates spatial variation in patch configurations (Fig. 4.1, 4.2); and 3) post-settlement density dependence is sufficiently weak that the variation in settlement persists over the long-term (Fig. 4.1, 4.2).

4.4.2 Analytic Approximation

Because there is no analytic solution for certain long-term properties of the above stochastic model, we analyzed a deterministic approximation. Here, we introduce the analytic approximation and a simple case of a clustered coral and an isolated coral to explore the relationship between the model parameters and long–term patterns. For a focal coral, we used the ODE

$$\frac{d\bar{N}}{dt} = -\alpha\bar{N} - \beta\bar{N}^2 + \sum_{i=0}^{\infty} \bar{\lambda}\delta_{\gamma i}(t), \tag{4.4}$$

where the parameters α (density–independent mortality rate), β (density–dependent mortality rate) and $\bar{\lambda}$ (settlement intensity) are the same as above. In the last term, we use a sequence of Dirac-delta functions to produce instantaneous pulses at the times γi of size $\bar{\lambda}$.

For reasonably large population sizes, this ODE closely approximates the stochastic system (Appendix 4.8.1 in the Supporting Information). Studying the long-term behavior of $\bar{N}(t)$ provided valuable insights that facilitated our interpretations of the simulation results and the behavior of the system in various regions of the parameter space described by the density–independent mortality rate (α), density–dependent mortality rate (β), time between arrivals (γ), and settlement intensity (λ).

To simplify the analysis, we calculated the number of settlers on a focal coral in two extreme habitat configurations: a) when a coral is completely isolated (i.e. when settlement is maximized): $\bar{\lambda}_{\text{isolated}}$, and b) when a coral is completely surrounded by other corals (i.e. when settlement is minimized): $\bar{\lambda}_{\text{clustered}}$. The expected number of settlers to an isolated coral and to a clustered are (Appendix 4.8.3):

$$\bar{\lambda}_{\text{isolated}} = \lambda_{\text{isolated}} = L\pi R^2 + \frac{2\pi L}{\mu^2}\left[\mu R + 1 - \left(\frac{m}{\theta}\right)^{-\mu/\rho}\left(\frac{\mu}{\rho}\ln\left(\frac{m}{\theta}\right) + \mu R + 1\right)\right] \tag{4.5a}$$

$$\bar{\lambda}_{\text{clustered}} = L\pi R^2. \tag{4.5b}$$

The number of settlers to a clustered coral is simply the larvae that arrive directly over the patch, while an isolated coral receives those that land directly over the coral as well as those that arrive within the detection region and survive the travel to the coral. The difference in settlement is driven by the second term in Eq. 4.5a, which provides the number of settlers that arise from larvae that must swim to the coral from other locations (and survive the journey). When mortality (μ) is low (or corals are small), the relative variation in the number of settlers (to an isolated vs. a clustered coral) is very high.

To study the approximation (Eq. 4.4), we reduced the complexity of the analysis via nondimensionalization by substituting $\tau = \alpha t$ and defining two dimensionless parameter

groups,

$$\nu := \frac{\beta\bar{\lambda}}{\alpha^2} \text{ and } \Delta := \alpha\gamma. \tag{4.6}$$

The parameter ν is a measure of the relative strength of the density–dependent processes and is proportional to the settlement intensity to a coral and the ratio of density–dependent processes to the (square of) density–independent mortality. The parameter Δ weighs the timing of the settlement events to the strength of density–independent mortality. We further simplified the equation by substituting $\tilde{N} = \frac{\bar{N}}{\alpha/\beta}$ to obtain

$$\frac{d\tilde{N}}{d\tau} = -\tilde{N}\left(1+\tilde{N}\right) + \nu\sum_{k=0}^{\infty}\delta_{k\Delta}(\tau) \tag{4.7}$$

with $\tilde{N}(0) = \tilde{N}_0$.

To analyze the solution to Eq. 4.7, we took a two-step approach. First, we solved the system on intervals between settlement pulses (i.e. during the post-settlement processes). Specifically, suppose that the population is of size n when the settlement pulse occurs ($t = k\Delta$ or one of the settlement peaks in Fig. 4.A2). Then, during post-settlement phases, the solution takes the form

$$\tilde{N}(\tau) = \frac{(n+\nu)e^{-(\tau-k\Delta)}}{1+(n+\nu)(1-e^{-(\tau-k\Delta)})}, \text{ for } \tau \in (k\Delta, (k+1)\Delta]. \tag{4.8}$$

Second, we found the equilibrium by setting the magnitude of settlement pulses equal to the deaths that occurred between each pulse. The equilibrium value of the population size immediately prior to a settlement event is (see Appendix 4.8.2):

$$\tilde{N}^* := \lim_{k\to\infty}\tilde{N}(k\Delta) = \frac{1+\nu}{2}\left(-1+\sqrt{1+\frac{4\nu e^{-\Delta}}{(1-e^{-\Delta})(1+\nu)^2}}\right). \tag{4.9}$$

Equation 4.9 provides the equilibrium number of fish on a coral for a given level of

settlement (reflected in ν). That settlement is determined by the environment (which can affect larval supply and mortality during settlement) as well as the spatial configuration of the coral landscape (which affects the degree of propagule redirection). Thus, in a single landscape, there may be isolated corals for which settlement is high and other corals (i.e. those near other corals) for which settlement is low.

We used the cases of an isolated vs. clustered coral (Eq. 4.5a and 4.5b) to examine how the most extreme heterogeneity in settlement patterns is modified by post-settlement processes (as defined by ν and Δ) to drive spatial patterns in the equilibrium number of occupants on the coral ($\tilde{N}^*$). In particular, we determined to what extent and under what conditions the spatial variation in settlement is maintained as spatial variation in occupants. Long-term patterns of occupant density (as reflected in Eq. 4.9) depend upon: 1) settlement intensity (i.e., $\bar{\lambda}$, as reflected in ν); 2) density–dependent mortality (i.e., β as reflected in ν), 3) density–independent mortality (i.e., α as reflected in ν and Δ), and 4) the time between settlement events (i.e., γ as reflected in Δ).

For $R = 1$ and $\mu = 0.4$, an isolated and a clustered coral would experience an almost 18-fold difference in settlement: i.e., $\bar{\lambda}_{\text{isolated}}/\bar{\lambda}_{\text{clustered}} = 17.9$. We then examined the conditions under which this disparity in settlement (i.e. as measured by $\bar{\lambda}_{\text{isolated}}/\bar{\lambda}_{\text{clustered}} = 17.9$), led to disparity in the equilibrium number of occupants on corals (i.e. as measured by $\bar{N}^*_{\text{isolated}}/\bar{N}^*_{\text{clustered}}$). In particular, we examined this relationship under different values of ν obtained by changing either density dependence (β, Fig. 4.3A), or larval supply (L, Fig. 4.3B).

As ν increases (i.e. the strength of density–dependent mortality and settlement intensity grows relative to the strength of density–independent mortality), the variation in the long-term number of occupants among corals (green curves in Fig. 4.3A,B) decreases, despite the 17.9-fold variation in settlement: e.g., when $\nu = 700$, the 17.9-fold variation in settlement was reduced to an a 1.1-fold variation in equilibrium density (Fig. 4.3A,B). By contrast, as ν decreases, the variation in settlement rate persisted, creating long-term variation in occupancy patterns among patches (purple curves in Fig. 4a,b): e.g., when $\nu = 7$, the

17.9-fold variation settlement was reduced to only 3.5-fold (Fig. 4.3A,B).

We also compared the relative equilibria on an isolated vs. clustered coral across the full parameter space of the non-dimensionalized system (i.e., using Eq. 4.8). As $\nu \to 0$, the relative spatial variation in occupant density approached the relative variation in settlement (in this case, 17.9). When ν was small, (i.e. weak density dependence and low larval input), the heterogeneity in settlement persisted and led to long-term heterogeneity in the numbers of coral occupants. Thus, in under-saturated systems, long-term heterogeneity in the number of occupants can be driven solely by varying degrees of clustering among otherwise identical habitats. In contrast, increasing ν caused the system to become saturated, so differences in settlement due to propagule redirection mattered little to the equilibrium number of occupants on the coral: i.e., the equilbrium numbers on isolated and clustered corals was approximately equal (Fig. 4.4).

The effect of the time between settlement pulses (i.e. via Δ) was more complicated. When ν was very small, increasing the time between settlement pulses (i.e. Δ) increased spatial heterogeneity; however when ν was large, increasing the time between pulses decreased heterogeneity (Fig. 4.4). For the calculations of these critical values and limits see Appendix 4.8.4.

4.5 Application to a realistic system

We sought to define plausible parameter regimes to guide our theoretical analyses above. We also sought to evaluate the potential importance of settlement redirection in the context of a real system. To do this, we turned to reef fish as a model system (Table 4.1). We used data collected from field studies in Moorea (Shima & Osenberg, 2003; Stier & Osenberg, 2010)), as well as from a meta-analysis of density–dependent mortality in reef fish (Osenberg *et al.*, 2002b).

To estimate parameters that governed redirection (Eq. 5.3: larval density, L, larval sur-

vival rate μ, coral signal decay rate, ρ, and the detection threshold, θ), we used settlement data of four fish species to isolated pairs of corals, pairs of central corals surrounded by a circle of ten neighboring corals, and the ten neighboring corals (Stier & Osenberg, 2010). We expressed larval density, L, as a function of the number of settlers on the isolated pair, and then used the data from other corals to estimate coral signal parameters (ρ and θ) and the larval survival rate (μ). We searched parameter space using a random grid search and minimized the difference between the observed and simulated values. However, there are more parameters (i.e., 4) than reference points (i.e., 3 types of corals: isolated, central, and circle), so we present the results from one possible parameter set, although there was little difference in final results when we evaluated other parameter options.

The estimation of post-settlement processes are described by parameters: α, the density–independent mortality rate, and β, the density–dependent mortality rate. We obtained an estimate of density–independent mortality, $\alpha = 0.0001$, from a meta-analysis for reef fish (Osenberg *et al.*, 2002b). That meta-analysis also provided an estimate of density–dependent mortality: $\beta = 0.0005$. In addition, we also used an estimate of density–dependent mortality from a detailed study conducted in Moorea on one species of reef fish, *Thalassoma hardwicke* (Shima & Osenberg, 2003), $\beta = 0.056$. We consider this latter estimate to represent very strong density dependence (and thus a worst-case scenario for settlement redirection persisting through time); wherease the estimate from the meta-analysis is a more typical (moderate) estimate.

The application also required that we specify a spatial landscape. Because we were using studies from Moorea to motivate our analyses, we used Google Earth to identify coral habitat on an 1000m^2 area of backreef in Moorea. We then overlaid this habitat area with circles of 1m^2 radii to define units of habitat within which fish potentially settled and interacted.

In addition to propagule redirection, prior work in the Moorea system suggests that spatial variation in habitat quality can produce spatial variation in density–dependent mortality and settlement: high quality sites receive more settlers and incur lower levels of

density–dependent mortality (Shima & Osenberg, 2003). This covariance between density-dependence and settlement generates a pattern called "cryptic density-dependence" (CDD: see Wilson & Osenberg (2002), Shima & Osenberg (2003)). For our purposes, we wanted to include possible effects of settlement redirection as well as other mechanisms that could produce spatial variation or that might mask or alleviate the effects of propagule redirection. Therefore we simulated dynamics under four scenarios: i) a control (i.e., no propagule redirection or variation in density–dependent mortality), which served as a baseline for comparison; ii) the presence of spatial variation in habitat quality, but not propagule redirection (i.e., covariance between settlement cues and density-dependence as articulated in CDD); iii) the presence of propagule redirection only (i.e., variable settlement due to redirection, but spatially uniform habitat quality); and iv) the presence of both propagule redirection and variation in habitat quality (i.e., spatially variable settlement due to redirection and settlement cues, and variable density-dependence due to CDD).

To simulate spatial variation in density-dependence, we sampled from the distribution of β from Shima & Osenberg (2003) which had a mean of 0.056 (for strong density-dependence) or from a distribution with those values divided by 1000 (thus achieving a mean of 0.00056: i.e., moderate density dependence that was comparable to the mean from the meta-analysis of Osenberg et. al 2002). Thus with only propagule redirection, density-dependence was spatially uniform (with $\beta = 0.056$ or $\beta = 0.00056$), whereas with habitat quality, the mean level of density-dependence was the same, albeit spatially variable. To represent spatial variation in settlement due to habitat quality, we assumed that expected settlement to a coral was proportional to $1/\beta$ (as is the case in CDD: Shima & Osenberg (2003)), and then adjusted the signal to yield the same average signal as in the propagule redirection scenario.

We therefore evaluated eight scenarios ([presence vs. absence of spatial variation in habitat quality] X [presence vs. absence of propagule redirection] X [strong ($\beta = 0.056$) vs. moderate ($\beta = 0.00056$) mean density dependence]). For each scenario, we simulated settlement and post-settlement dynamics, as in the previously described stochastic simulations,

recorded the index of dispersion for settlement and occupants, and repeated this 100 times.

Both propagule redirection and CDD generated spatial variation in settlement compared to the null scenario when applied to the Moorea landscape (Fig. 4.5A). Heterogeneity due to propagule redirection was 1.6 times greater than generated by CDD (i.e., habitat quality) alone. Additionally, when we included effects of both habitat quality (CDD) and settlement redirection, the resulting settlement pattern had an index of dispersion 2.1 times greater than that produced by CDD alone, and 1.3 times greater than that produced by settlement redirection alone. Thus, settlement redirection played a comparable or larger role in determining settlement patterns than spatial variation in habitat quality.

Density-dependence is generally assumed to reduce spatial variation caused by settlement, and this was the case when density dependence was strong (Fig. 4.5B). In all scenarios, the index of dispersion was close to 1, indicating that occupant density was spatially variable, but not distinguishable from that expected under a Poisson spatial processes. This was true even when density-dependence was spatially variable (as in CDD). In contrast, under moderate density-dependent mortality, there was greater variation in occupant density than expected by a Poisson process, except in the control scenario. For example, in the presence of propagule redirection (and spatially uniform density-dependence), the spatial variation in settlement was maintained for the occupants. In the presence of spatial variation in density-dependence, the index of dispersion actually increased, intensifying spatial variation in the density of occupants, and the combination of redirection and CDD was 1.9 times that of CDD alone or 3.6 times that of redirection alone.

These simulations demonstrated that landscape configuration (which creates variation in the proximity of neighboring patches) and spatial variation in settlement cues (due to differences in habitat quality) create approximately equal spatial variation in settlement. Under moderate (and weak) density dependence, these settlement patterns create long-term patterns of occupant density that are equally variable (when density dependence is spatially uniform) or magnified in intensity (when density dependence is spatially variable).

4.6 Discussion

Heterogeneity in the number of occupants among habitat patches is often attributed to intrinsic characteristics of the patches. However, here we demonstrate that the configuration of patches in a landscape also can create spatial heterogeneity in colonization and resident density among otherwise identical patches. Therefore, the spatial distribution of patches alone can cause long term heterogeneity in a system independent of other factors.

This heterogeneity requires that habitat patches vary in their proximity to other patches, and that this proximity reduces the input of colonists to these patches. In such a system, landscapes with a few isolated patches and many clustered patches will exhibit far greater heterogeneity than a landscape where all patches are similarly spaced. The phenomenon in which patch configuration creates settlement heterogeneity, propagule redirection, has not been investigated widely. Stier & Osenberg (2010) found that neighboring corals reduced the settlement of reef fishes to focal corals. Morton & Shima (2013) found a similar pattern in a temperate fish when comparing discrete habitat patches (i.e. but not when comparing single patches of increasing size). Other marine organisms display related spatial patterns of settlement. For example, larvae of the intertidal barnacle, *Balanus glandula* show spatial heterogeneity in settlement due to depletion of larvae from the water column. Downstream sites had lower settlement, either because larval densities were depleted as larvae settled upstream (Gaines *et al.*, 1985), or because predators reduced larval density (Gaines & Roughgarden, 1987). Upstream sites have also been shown to "steal" larvae from downstream sites in a coral reef system on the Great Barrier Reef: fish recruitment was diminished on corals immediately downstream of other suitable corals (Jones, 1997a). These settlement shadows (sensu Jones, 1997a) are analogous to patterns created via propagule redirection. We note, however, that these examples of settlement shadows largely presume directional supply of larval leading to a depletion of larval stocks from upstream to downstream areas. This phenomenon would act in consort with propagule redirection to generate spatial variation

in settlement: in the extreme, settlement shadows deplete larval density (L in our model) creating an upstream-downstream gradient in settlement, while propagule redirection would promote variation in settlement perpendicular to flow (i.e., creating variation in settlement for a given larval density).

The above evidence comes from marine organisms in which larvae are the dispersive stage. Propagule redirection is likely in other systems as well. For example, many freshwater organisms have a dispersive adult stage but a relatively sedentary larval stage: e.g., aquatic insects such as mosquitoes or odonates have dispersing adults that oviposit in or colonize ponds and lakes. In these systems, the availability and distribution of ponds could generate heterogeneity: ponds with many neighboring ponds may attract fewer ovipositing females or colonizing adults. However, experiments with aquatic beetles showed that colonization rates were independent of local pond (patch) density among patches without predators (Resetarits & Binckley, 2009, 2013). It is not clear what produced these disparate results, but it is likely that the degree of propagule redirection will depend on the scale of movement of the dispersive stage, the scale of their sensory abilities (how far they can discern among habitat patches), the spacing of habitats in the landscape (relative to movement and sensory abilities), and the mortality incurred moving across the landscape. It is possible that beetles and reef fish experiments were conducted at different relative levels of these factors. Further research should investigate how the relative scales of these processes produce differences in the spatial patterns of colonization.

Spatial scale can also alter expected patterns because organisms make choices at multiple spatial scales. In our analyses, we have focused on a relatively small (or local) scale, in which the density of potential colonists was spatially uniform (and reflected in larval density, L). However, at a larger scale (a "region"), colonists may be attracted to regions with more patches. Thus regions with many patches could draw in more potential colonists, but within a region, propagule redirection could generate variation in colonization: i.e., at one scale (the region) neighbors draw in more colonists, but at a smaller scale (patch) neighbors reduce

colonization. This is analogous to what happens with pollinators: more apparent floral displays (or higher plant densities) recruit more pollinators, but within a flowering patch, a lower proportion of flowers are visited as flowers compete for pollinators (e.g. Ohashi & Yahara, 1999; Grindeland *et al.*, 2005). Resolving the relative importance of these processes (and the sensory cues used at these different scales) will be an important next step.

Heterogeneous patterns established by redirection and the arrangement of patches within a region are most likely to persist when the system is undersaturated, due either to low density–dependent mortality, high density–independent mortality, a limited supply of colonists, or a short life-span (which reduces the number of co-occurring cohorts within a patch). As a system nears saturation, the distribution of occupants in the system becomes more homogeneous, obliterating the spatial patterns first established during the colonization phase. The linkage between patterns established at settlement and those that persist at later life stages has a long history in "supply-side ecology" (e.g. Gaines *et al.*, 1985). While these studies are typically at a much larger spatial scale, another difference between that tradition and what we studied here is that the settlement patterns in supply-side ecology are often attributed to stochastic processes (e.g. due to unusually large settlement events) or intrinsic properties of sites (e.g. that lead to greater settlement). Here, the neighborhood itself generates spatial variation in settlement.

The effects that neighboring patches have on settlement, and resulting in long-term spatial patterns, have important implications for applications such as habitat restoration. Many restoration techniques focus, not on the target organisms, but instead on the restoration of their habitat. For example, flower patches are added for pollinators (e.g. Wratten *et al.*, 2012), and reef structures are created for marine invertebrates and fish (e.g. Burt *et al.*, 2009). If habitat is added to an area, the expectation is that the added habitat will attract colonists and help re-establish the target population. However, if redirection occurs, this additional habitat might not lead to a higher density of organisms—it may simply attract colonists away from the existing habitat (Pickering & Whitmarsh, 1997), and if density de-

pendence is weak, there may be little net benefit of the habitat restoration on the focal species (Carr & Hixon, 1997; Osenberg *et al.*, 2002a).

Although we incorporated realism into our application by applying our approach to a realistic landscape, our overall approach ignored several complexities of real systems that allowed us to focus on the effect of neighbors. Not only did we focus on local among-patch competition for colonists (see above discussion about spatial scale), but we also made simplifying assumptions with regard to patch characteristics. For example, we assumed that all patches were uniform in size, although patch size is an important variable that can affect colonization. Patch size and isolation of a patch affects colonization of ponds by amphibians (Laan & Verboom, 1990) and aquatic insects (Wilcox, 2001). As illustrated in our application to Moorea, habitat quality (and associated signal) also likely varies and leads to differences in colonization (e.g. Holbrook *et al.*, 2000; Shima & Osenberg, 2003; Resetarits & Binckley, 2009, 2013; Resetarits & Silberbush, 2016). Furthermore, signals can vary within and between types of habitats (Dixson *et al.*, 2014). Empirically, we might expect a correlation between the strength of these signals and habitat quality. This may be especially important for biogenic habitat (such as corals or trees); in which the habitat patches either mask their signals (to hide from colonists that harm that habitat) or enhance their signals (to attract beneficial colonists). Our realistic landscape simulations indicated that variation in signal strength will likely intensify spatial heterogeneity in settlement (Fig.4.5).

Finally, we assumed that the intrinsic qualities of the patches (e.g. size) were static throughout time. While this may be true for some habitats, many biogenic habitat patches (e.g. corals, trees), are dynamic, and as a result, their characteristics change through time. Their dynamics can be affected by many processes (including competition, predation, and disease dynamics) that drive spatial pattern in the growth and survival of the organisms that occupy the biogenic habitat. For example, coral symbionts (e.g. fish or crabs) increase coral growth and survival by providing nutrients to the coral (Holbrook *et al.*, 2008, 2011) and by defending the coral from predators or other harmful factors (Glynn, 1980; Pratchett

et al., 2000; Stier *et al.*, 2010). As a result, we might expect clusters of patches to do poorly, not because they compete for food and not because of increased disease transmission, but instead because they compete for beneficial symbionts. This form of competition, driven by propagule redirection, may be an important process in systems in which biogenic habitat harbors beneficial symbionts that have open demographics.

4.7 Tables and Figures

Parameters		
Description	Label	Values Used for Stochastic Simulations
coral radius	R	1m
coral signal magnitude	m	1
coral signal decay rate	ρ	0.75 m^{-1}
larvae survival decay rate	μ	0.4 m^{-1}
density–independent mortality rate	α	0.0001 day^{-1}
time between settlement events	γ	28 days
density–dependent mortality rate	β	0.00056 and 0.056 m^2 $fish^{-1}day^{-1}$
larval density	L	0.33 larvae m^{-2}
Dimensionless Groups		
Description	Label	Definition
relative strength of density–dependent processes to density–independent processes	ν	$\frac{\beta\lambda}{\alpha^2}$
time between settlement events relative to occupant longevity	Δ	$\alpha\gamma$

Table 4.1: List of parameters and dimensionless groups.

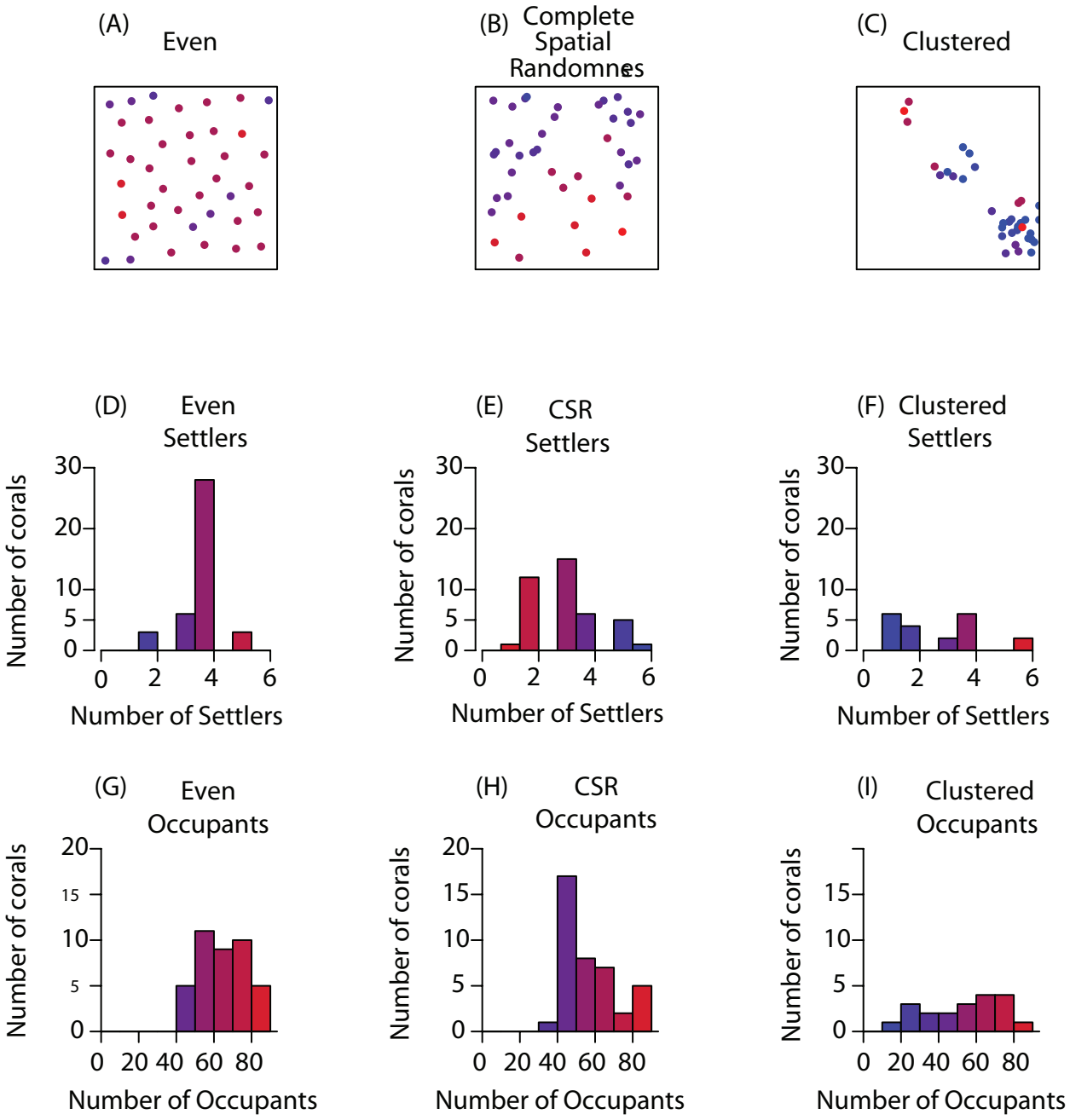

Figure 4.1: Landscape variation leads to heterogeneity in occupancy patterns. The top row of images show the three landscape configurations with corresponding histograms of settlement (middle row) and occupancy (bottom row). The gradient of isolation from blue for the most clustered coral and red for the most isolated coral are indicated in all images. $\mu = 0.4$, $\rho = 0.75$, $\alpha = 0.0001$ $\beta = 0.00056$ $\gamma = 28$, $R = 1$ and $L = 0.33$

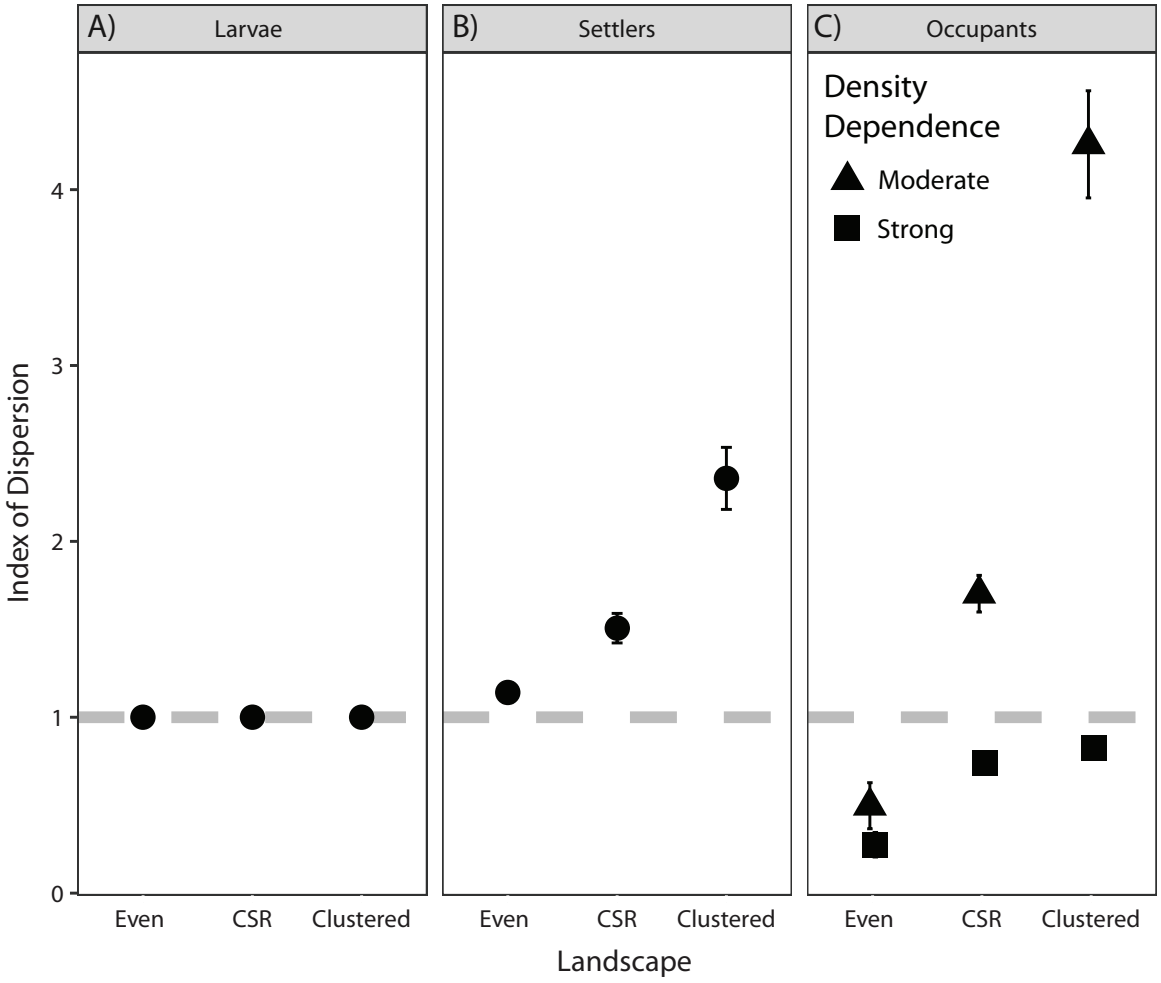

Figure 4.2: Spatial heterogeneity in A) larvae, B) settlers, and C) occupants for three landscape configurations (even, random, and clustered). An index of dispersion (variance/mean) > 1 indicates a heterogeneous distribution for the focal life stage, $= 1$ indicates a random (dashed horizontal line), and < 1 indicates a more even distribution. For each landscape configuration, we simulated 100 replicate landscapes, each with 40 corals. Results are given for the end of the simulation (i.e., after at least 50 settlement pulses). In all simulations, we set $\mu = 0.4$, $\rho = 0.75$, $\alpha = 0.0001$, $\gamma = 28$, $R = 1$, $L = 0.33$, and $\beta = 0.00056$ (moderate) and $\beta = 0.056$ (strong). Error bars represent 95% confidence intervals (based on the 100 landscapes)

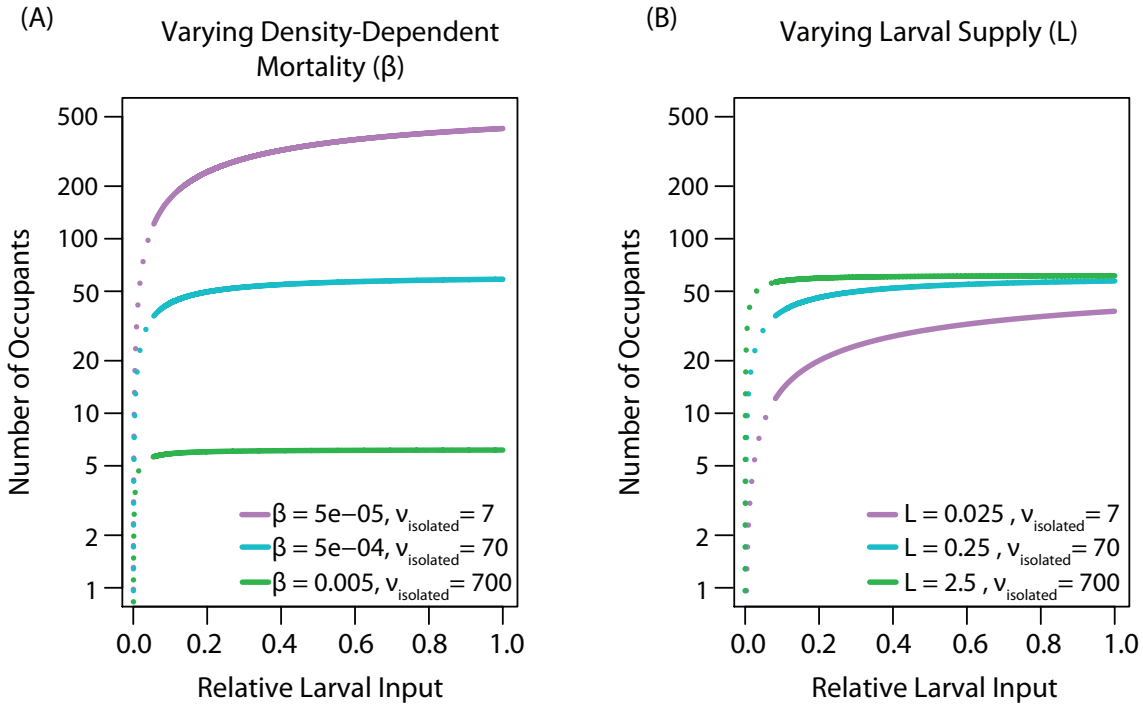

Figure 4.3: The relationship between the equilibrium number of occupants and settlement rate for corals that vary in their degree of isolation (and thus their relative settlement) for three different levels of the non-dimensionalized parameter, ν, achieved by changing either a) the strength of density dependence, β, or b) larval density (L). Relative settlement gives the settlement to a coral relative to that received by an isolated coral. Thus, an isolated coral has a relative settlement of 1.0. Solid lines indicate the equilbrium (Eq. 4.9) for corals with inputs between $\lambda_{\text{clustered}}$ and $\lambda_{\text{isolated}}$. Dashed lines extend those relationships to settlement rates that are $< \lambda_{\text{clustered}}$ and thus reveal the full relationship. Colors indicate values of ν_{isolated} (the value for ν that corresponds to ν_{isolated}, note that $\nu_{\text{isolated}} = 17.9 \times \nu_{\text{clustered}}$): $\nu_{\text{isolated}} = 7$ (purple), $\nu_{\text{isolated}} = 70$ (teal), and $\nu_{\text{isolated}} = 700$ (green). For these calculations, $R = 1$ and $\mu = .4$.

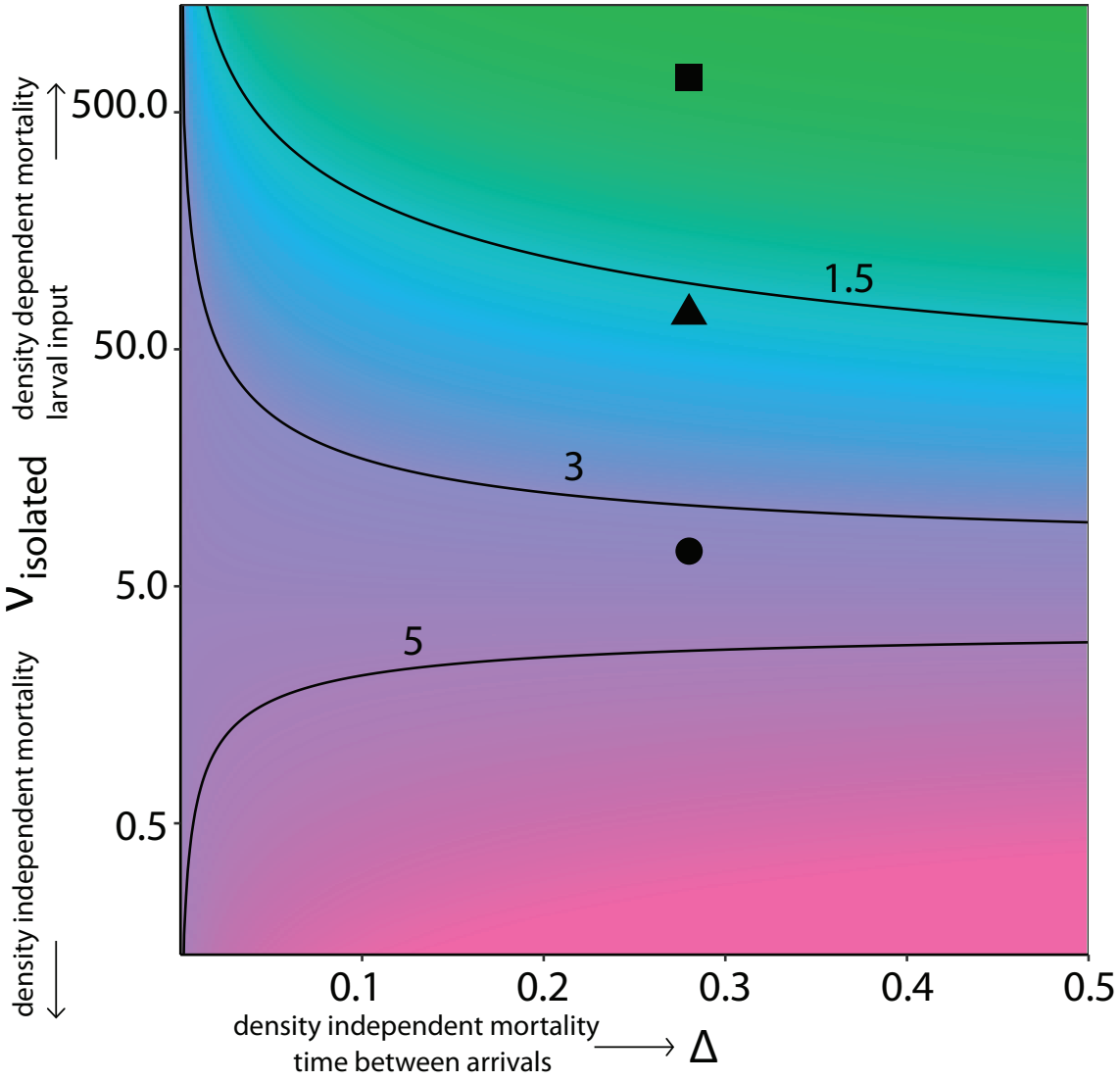

Figure 4.4: Relative density of occupants on an isolated coral versus one surrounded by other corals (clustered), $N^*_{\text{isolated}}/N^*_{\text{clustered}}$, as indicated by the color of each pixel and expressed as a function of the two non-dimensionalized parameters, ν and Δ. Initial settlement densities were approximately 17.9:1 (isolated:clustered): The color of each pixel represents the magnitude of the ratio between the equilibrium of a completely isolated and a completely clustered coral. Green pixels (values closer to 1) indicate that there is little spatial variation in occupant density (despite the 17.9-fold variation in settlement). This homogeneous pattern is primarily facilitated by increased density dependence (increasing ν) and increased larval supply (increasing L). Pink pixels (indicating $N^*_{\text{isolated}} \gg N^*_{\text{clustered}}$) indicates that the spatial variation in settlement persists over the long term. The three points give the results from Figure 4.3 (circle: $\nu_{\text{isolated}} = 7$, triangle: $\nu_{\text{isolated}} = 70$, square: $\nu_{\text{isolated}} = 700$). Contour lines indicate $N^*_{\text{isolated}}/N^*_{\text{clustered}} = 1.5, 3,$ and 5.

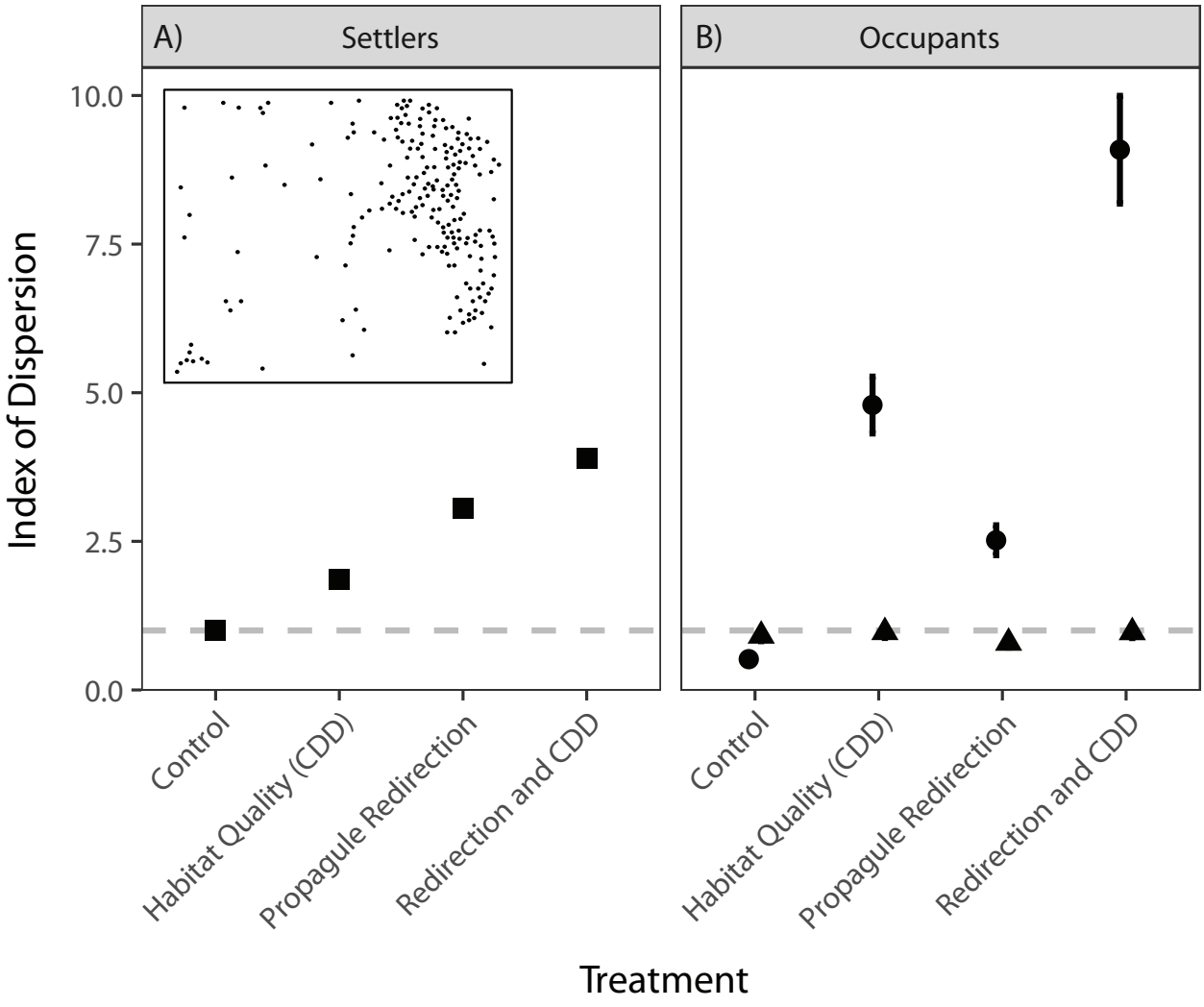

Figure 4.5: Spatial heterogeneity in A) settlers, and B) occupants for a portion of the back-reef of the north shore of Moorea, French Polynesia (inset of panel A). An index of dispersion (variance/mean) > 1 indicates a distribution more heterogeneous than random (i.e. Poisson) for the focal life stage, $= 1$ indicates a random spatial distribution (dashed horizontal line), and < 1 indicates a more even distribution. Propagule redirection creates slightly more heterogeneity in settlement than variation in habitat quality alone (as captured by the phenomenon of cryptic density dependence; Shima & Osenberg (2003)). Under moderate ($\beta = 0.00056$) density–dependent mortality (circles), propagule redirection increased the heterogeneity in occupants by 1.6-fold compared to differences due quality alone. Under strong ($\beta = 0.056$) density–dependent mortality (triangles), variation among patches was not distinguishable from a Poisson spatial process. In all simulations, we set $\mu = 0.4$, $\rho = 0.75$, $\alpha = 0.0001$ $\gamma = 28$, $R = 1$ and $L = 0.33$. Error bars represent 95% CIs.

4.8 Appendix

4.8.1 Comparison of the ODE approximation to the mean of the stochastic model

As described in the Methods section, we use a stochastic and a deterministic model in tandem to study settlement shadow dynamics. Because these mathematical systems are non-linear (by virtue of the density–dependent mortality), the deterministic model is *not* simply the mean of the stochastic version. In fact, the deterministic model systematically underestimates the mean of the stochastic process due to an application of Jensen's Inequality. We use this subsection to justify this claim.

The stochastic model is a continuous-time Markov chain denoted $N(t)$. For a given initial condition $n \in \{0, 1, 2 \ldots\}$, we define a collection of functions $\{p_k(t)\}_{k\geq 0}$ that respectively describe the probability that there are k settlers on a focal coral at time t, Recalling that γ is the time between settlement pulses, we can write the master equations for this Markov chain for all $t \in [0, \gamma)$ as follows: The first term on the right-hand side results from the rate of the transition $k \to k-1$ while the second term corresponds to the transition $k+1 \to k$. Noting that the size of the initial pulse λ is a Poisson distributed random variable, the last term captures the effect of the initial pulse ($\delta_0(t)$ is a Dirac-delta function concentrated at the initial time $t = 0$).

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term captures the effect of the initial pulse ($\delta_0(t)$ is a Dirac-delta function concentrated at the initial time $t = 0$).

$$\begin{aligned}
\frac{d}{dt}\mathbb{E}_n[N(t)] &= \frac{d}{dt}\sum_{k=1}^{\infty} kp_k(t) \\
&= \sum_{k=1}^{\infty} k\big[-(\alpha k + \beta k^2)p_k(t) + (\alpha(k+1) + \beta(k+1)^2)p_{k+1}(t)\big] \\
&= -\sum_{k=1}^{\infty}(-\alpha k - \beta k^2)p_k(t) \\
&= -\alpha\mathbb{E}_n[N(t)] - \beta\mathbb{E}_n\big[N^2(t)\big]\,.
\end{aligned}$$

By Jensen's Inequality, we observe that

$$-\alpha\mathbb{E}_n[N(t)] - \beta\mathbb{E}_n\big[N^2(t)\big] \leq -\alpha\mathbb{E}_n[N(t)] - \beta(\mathbb{E}_n[N(t)])^2.$$

Now, note that the right-hand side of this inequality is precisely the form seen in the ODE we use in our deterministic approximation $\bar{N}(t)$ defined by Eq. 4.4:

$$\bar{N}(t)' = -\alpha\bar{N}(t) - \beta(\bar{N}(t))^2.$$

This implies that the mean of the stochastic process $N(t)$ is decreasing faster than the approximating deterministic process $\bar{N}(t)$. For larger populations, this effect is negligible, but this may not be the case in the dynamics of smaller populations.

4.8.2 Convergence of the ODE to equilibrium

As seen in right panel of Figure 1, when the population is relatively large there is a stable recurring pattern in the time periods marked initially by a settlement pulse and followed by a mortality phase. To assign a single number for this pattern, we focus on the sequence of values $\{N(i\Delta)\}_{i=0}^{\infty}$ (Eq. 9) corresponding to the population sizes just before settlement pulses

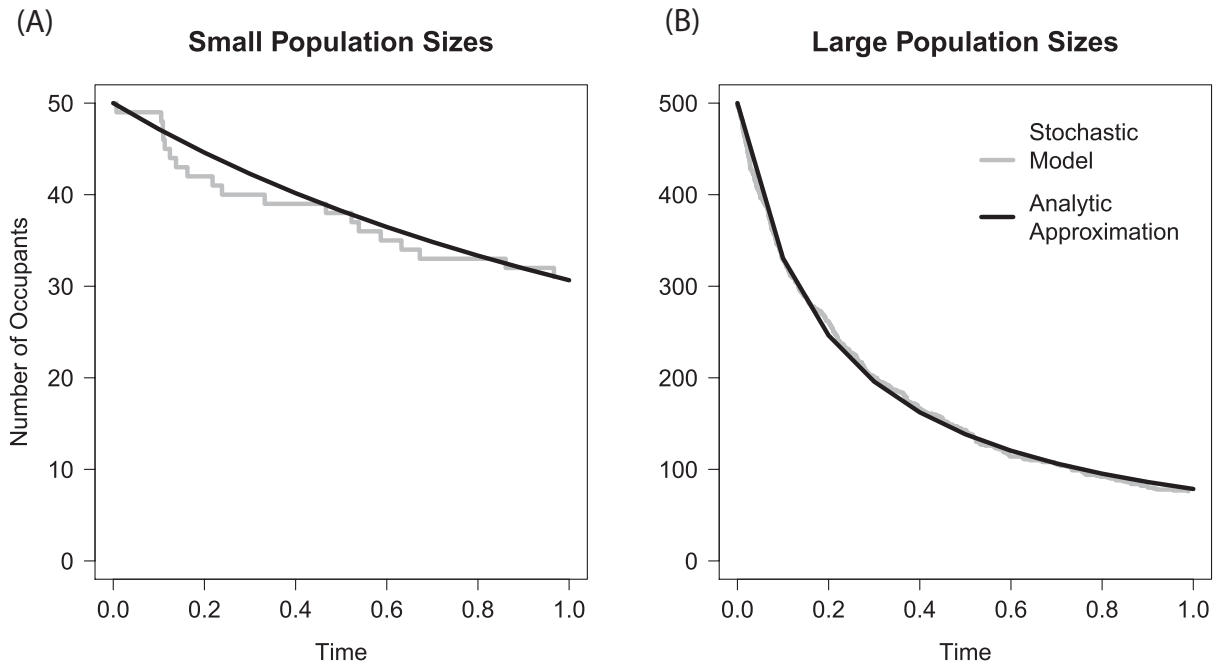

Figure 4.A1: A comparison of the stochastic model (described in "Post-Settlement Processes") and the analytic approximation for post-settlement mortality (4.4). Small population sizes started with $\lambda = 50$ and large populations with $\lambda = 500$. At large population sizes, the analytic approximation nicely approximates the stochastic process. However, at small populations sizes, the approximation underestimates density–dependent mortality.

occur. Over time, in the deterministic model, these values converge to a long-term steady value, $\tilde{N}^*$. To compute this quantity, we found the solution to the ODE (Eq. 7) between settlement pulses and then note that periodic equilibrium occurs with the number of deaths that occur in the time between settlement pulses is equal to the size of the pulses. That is to say, we look for the point when the sequence of values, $\{N(i\Delta)\}_{i=0}^{\infty}$ (Eq. 9) satisfies the condition $N((i+1)\Delta) = \varphi(N(i\Delta))$ where

$$\varphi(x) = \frac{(x+\nu)e^{-\Delta}}{1+(x+\nu)e^{-\Delta}}.$$

The function has one fixed point and the formula is given by Eq. 9. Furthermore, because φ is increasing and concave down, for all $x < \tilde{N}^*$ we have $\varphi(x) > x$. On the other hand,

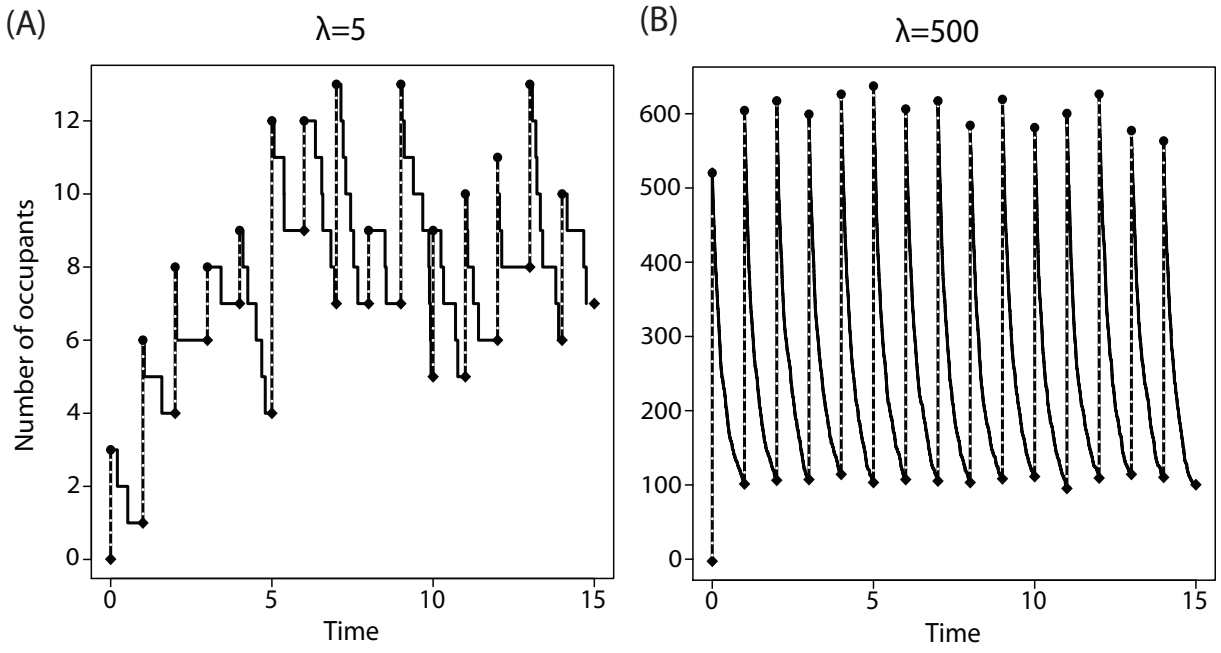

Figure 4.A2: The stochastic dynamics of fish within a single coral under A) low and B) high settlement intensities. New fish arrive during discrete settlement events (circles) that are followed by continuous (but stochastic) declines in abundance (solid lines) until just before the arrival of another settlement event (diamonds). Empty corals accumulate larvae and eventually converge on an equilibrium at which the number of settlers equals the number of deaths (of recent settlers and older fish) during the inter-pulse period. At high larval densities, the stochastic dynamics resemble a continuous approximation. In both panels, $\alpha = 0.5$, $\beta = 0.005$, and $\gamma = 1$

if $x > \tilde{N}^*$, then $\varphi(x) < x$. It follows that $\{N(i\Delta)\}_{i=0}^{\infty}$ is a bounded, monotone sequence of points and therefore the sole fixed point is asymptotically stable.

4.8.3 Expected number of settlers on an isolated and a clustered coral

In our system, colonists arrive as a spatial Poisson process, which is to say that the total number drawn has a Poisson distribution and the location of each is drawn uniformly at random from the domain. The landscape contains circular patches with radius R, the centers of which are generated by one of the three spatial point processes (even, random, clustered, described in the main text. Within these landscape configurations, the patches have varying degrees of isolation. The most extreme cases occur when a patch is completely isolated or completely surrounded by other patches. We can analytically compute the exact formula for the distribution of the larval input in these two extreme cases.

In the case of the completely clustered patch, the expected larval input, $\lambda_{\text{clustered}}$ includes only settlers that arrive directly on the coral. Since colonists arrive uniformly at random with density L, the mean number of arrivals is simply the density times the area of the patch:

$$\lambda_{\text{clustered}} = L\pi R^2.$$

Calculating the expected colonist input to a completely isolated coral is more complicated. Suppose there is a circular coral patch of radius R centered at the origin of the x-y plane. We compute the expected number of settlers that arrive at the coral assuming that their initial location in the water column is uniformly distributed over a circular landscape, $\mathcal{O}$, centered at the origin and having radius $R_{\mathcal{O}}$. However, due to the detection threshold, θ, we only consider the area to R_*, or the radius where the signal is detectable, or where $\theta = me^{-\rho(R_*-R)}$. Thus, we integrate to $R_* = \frac{ln(m/\theta)}{\rho} + R$.

In this setting, Eq. 3, which describes the probability that any given settler arrives at

the focal coral, takes the form

$$p(R_*) := \frac{1}{|\mathcal{O}|}\iint_{\mathcal{O}} \frac{\Lambda_1(x,y)}{\sum_j \Lambda_j(x,y)} e^{-\mu d_1(x,y)}\, dx\, dy$$
$$= \frac{R^2}{R_*^2} + \frac{1}{\pi R_{\mathcal{O}}^2}\int_{\theta=0}^{2\pi}\int_{r=R}^{R_*} e^{-\mu(r-R)}\, dr\, d\theta$$

where the first term is the probability that a settler descends into the water column directly onto the coral and the second term includes the possibility of mortality in the travel from the initial location to the coral. Integrating, we find

$$p(R_*) = \frac{R^2}{R_*^2} + \frac{\mu R + 1 - (\mu R_* + 1)e^{\mu(R-R_*)}}{\mu^2}$$

Substituting $R_* = \frac{ln(m/\theta)}{\rho} + R$,

$$p(R_*) = \frac{\rho^2 R^2}{\ln(m/\theta)^2} + \frac{2\pi}{\mu^2}\left[\mu R + 1 - \left(\frac{m}{\theta}\right)^{-\mu/\rho}\left(\frac{\mu}{\rho}\ln\left(\frac{m}{\theta}\right) + \mu R + 1\right)\right]$$

The number of settlers that arrive at the focal coral is then a thinned Poisson process with mean $L|\pi R_*^2|p(R_*)$

$$\lambda_{\text{isolated}} = L\pi R^2 + \frac{2\pi L}{\mu^2}\left[\mu R + 1 - \left(\frac{m}{\theta}\right)^{-\mu/\rho}\left(\frac{\mu}{\rho}\ln\left(\frac{m}{\theta}\right) + \mu R + 1\right)\right].$$

4.8.4 Effect of Δ on spatial heterogeneity

The effect of time between settlement pulses relative to the settlement rate depended on ν. There was a critical value, $\nu_{\text{isolated}} = \sqrt{\frac{\lambda_{\text{isolated}}}{\lambda_{\text{clustered}}}}$, at which changing Δ had no effect on the resulting heterogeneity. Instead, at this critical value of ν, the variation in occupant numbers (between an isolated and clustered coral) is constant and equal to $\sqrt{\frac{\lambda_{\text{isolated}}}{\lambda_{\text{clustered}}}}$ (i.e., heterogeneity was present, but reduced from that imposed at settlement). In addition,

as $\Delta \to 0$ the relative density of occupants approached this same value (i.e., $\sqrt{\frac{\lambda_{\text{isolated}}}{\lambda_{\text{clustered}}}}$); and as $\Delta \to \infty$, the relative density approached a constant, $\frac{\nu_{\text{isolated}}(1+\nu_{\text{clustered}})}{\nu_{\text{clustered}}(1+\nu_{\text{isolated}})}$. Thus, the effects of increased density–independent mortality and time between arrivals (which affect Δ) cannot be viewed through the same lens as we viewed effects of ν. Although increasing Δ suggests a reduction in saturation, the homogenizing effects of density dependence still operate immediately following settlement pulses. Thus, increasing Δ cannot strictly maintain the spatial variation created by propagule redirection; some homogenization will be achieved (except in the limiting case as $\nu \to 0$).

Chapter 5: Biogenic habitat, propagule redirection and interactions with patch occupants create spatial pattern [1]

[1] Hamman, E.A., S.A. McKinley, A.C. Stier, and C.W. Osenberg To be submitted to *American Naturalist*

5.1 Abstract

Many habitats are biogenic and host a variety of occupants. The distribution of these occupants is often heterogeneous among patches, and this heterogeneity can be generated by the spatial configuration of the landscape through a process called propagule redirection. In many systems, these patches are comprised of biogenic habitat. The dynamics of the habitat are directly affected (positively or negatively) by resident organisms. Here, we develop models to explore the dynamics of biogenic habitat patches and symbionts using coral reefs as a study system. Both corals (the habitat patch) and their symbionts (e.g. snails and fish) have pelagic larvae that settle to a reef during pulsed settlement events. Coral growth and survival are affected by their symbionts, and the symbionts undergo density-independent and dependent mortality. We explore how species interactions (i.e. their direction and magnitude) between habitats and symbionts, in combination with larval propagule redirection, alters both the coral and symbiont populations. When symbionts are harmful, distributions of corals and symbionts are more variable than when symbionts have no effect on corals, and heterogeneity is reduced when symbionts have a beneficial effect. We contrast this with a settlement regime where corals don't compete for settling symbionts (a pattern referred to as "field of dreams"), and in this case spatial patterns remain random. Thus, the combination of propagule redirection and symbiont-habitat interactions generate spatial pattern in landscapes.

5.2 Introduction

Many habitats are biogenic, and provide important food and refuges to their occupants. For example, corals, kelps, and seagrasses all comprise important marine habitats. In the case of these biogenic habitats, they not only provide important protection from predators and food for their occupants, but also undergo their own population dynamics. These dynamics

are not only driven by internal demographics and environmental components (e.g. seagrass growth and reproduction is linked to variation in environmental factors such as salinity and temperature, McDonald *et al.*, 2016), but also by interactions with their occupants.

Biogenic habitats are often influenced by their occupants. Many of these occupants are symbionts that have a tight association with the host habitat. Often these interactions are beneficial to the host. For example, ants living in *Acacia* trees defend their host from herbivores, and in return receive nectar and nesting sites from the host (Palmer *et al.*, 2008); seagrasses reduce predation on mussels, which in turn increase nutrient availability resulting in increased growth of the seagrass (Peterson & Heck, 2001). In contrast, some symbionts of biogenic habitat harm the habitat. While marsh cordgrass reduces predation on *Littoraria irrorata* (Lewis & Eby, 2002), *Littoraria irrorata* damages the plant by either consuming tissue (Teal, 1962) or facilitating growth of fungus (Silliman & Newell, 2003). Thus, positive and negative interactions between habitats and occupants could play important roles in the dynamics of both species.

Corals are an important biogenic habitat that host both harmful and beneficial symbionts. While coral occupants benefit from inhabiting corals, which provide refugia (Holbrook & Schmitt, 2002; Almany, 2004) or access to food (Turner *et al.*, 1994; Oren *et al.*, 1998), coral occupants also affect coral growth and survival. Beneficial occupants increase coral growth by supplying the coral with nutrients (Meyer *et al.*, 1983; Holbrook *et al.*, 2008, 2011), reduce hypoxic stress by increasing flow within branches (Goldshmid *et al.*, 2004), or increase growth and survival by removing sediments (Stewart *et al.*, 2006; Stier *et al.*, 2012) or nets of vermetid gastropods (Stier *et al.*, 2010). Symbionts also increase survival by defending against predators such as corallivorous snails (*Drupella*), pincushion stars (*Culcita*) (McKeon & Moore, 2014), and the crown of thorns starfish (*Acanthaster planci*) (Glynn, 1980; Pratchett, 2001; McKeon & Moore, 2014). Corals also host harmful symbionts that decrease growth by consuming coral tissue (Cox, 1986; Turner *et al.*, 1994; Shantz *et al.*, 2011) or by altering abiotic conditions (Brown & Osenberg, in review).

Because most marine organisms have a dispersive life stage, settlement events play a critical role in establishing spatial patterns of coral occupants, especially for organisms with limited post-settlement movement, including many species that are resident within coral colonies. Settlement patterns can be created via attraction to conspecifics (Lecchini *et al.*, 2007), habitat quality (Shima & Osenberg, 2003), or the availability or configuration of habitat (Jones, 1997b; Stier & Osenberg, 2010; Hamman *et al.*, in prep). For example, recent work indicates that habitat patches compete for colonists and incoming propagules can be redirected away from focal habitats (we refer to this as "propagule redirection" sensu Stier & Osenberg, 2010). This competition among patches for colonists can generate long-term spatial heterogeneity among static habitat patches (Hamman *et al.*, in prep). However, because corals are themselves responsive to the density of occupants, the spatial patterns in occupants should feed back to alter spatial patterns in the coral as well as their occupants.

Under propagule redirection, corals with many neighbors receive relatively few settlers, whereas isolated corals receive many settlers. Because these settlers can have effects on coral growth and survival, the spatial patterns created by propagule redirection can alter coral dynamics. For example, if propagule redirection occurs in beneficial symbionts, then corals that are clustered in space would exhibit lower growth and survival rates, but isolated corals would have greater growth and survival. Such a pattern could resemble the expectations arising from competition (e.g., due to food limitation or interference competition: (Tarnita *et al.*, 2017)), but in this case it is mediated through effects on the density of beneficial coral occupants. Conversely, if propagule redirection occurs in harmful symbionts, then clustered patches of corals would have greater performance due to the dilution of symbionts by neighboring corals. Thus, feedbacks between corals and their occupants could create spatial patterns in coral-symbiont systems that have not been previously appreciated.

Here, we build on existing models of patch colonization and post-colonization dynamics to examine how feedbacks between habitats and occupants create spatial patterns, and how those patterns depend on: 1) the strength and direction of the effect of the symbiont on

the coral and 2) the amount of density-dependent mortality in the symbiont population. We contrast the results under propagule redirection to those obtained when settlement is unaffected by the proximity of neighboring corals (a scenario that we term "field of dreams", sensu Palmer et al. 1997).

5.3 Model

We built our model with coral reef systems in mind, and followed the framework first developed by Hamman et al. (in review). Hamman et al, however, examined occupants patterns that arose in static landscapes of patches. In contrast, we now let the habitat patches (corals) respond dynamically to the density (and type) or occupants that inhabit the coral. Thus, we consider coral symbionts with dispersive life stages that both positively and negatively affect their habitat. During each timestep:

- Juvenile corals recruit to the coral population based on a Poisson Spatial Process (i.e. their arrival locations are completely random in space)
- Symbionts colonize corals via propagule redirection. Colonization depends on larval density and the relative strength of settlement cues emitted from corals, which are assumed to increase with coral size
- Symbionts undergo density-indepdendent and density-dependent mortality
- Corals grow as a function of size, symbiont density, and symbiont effect
- Corals undergo mortality as a function of size, symbiont density, and symbiont effect

Thus, new coral recruits and the growth and mortality of existing corals alter the landscapes into which symbionts settle. In turn, those symbionts then affect coral growth and survival. The functional forms that govern these interactions are described and justified below. Because the dynamics occur on a monthly basis (one settlement pulse of both corals

and symbionts followed by mortality and coral growth), the forms presented below represent average monthly rates. We assume no post-settlement movement (if a coral dies, the symbionts on that coral also die).

5.3.1 Symbiont settlement and survival

Settlers settle and survive as in Hamman *et al.* (in prep). If a settler arrives in the landscape on a coral, it remains on that coral. If it arrives between corals, then it responds to signals emitted from corals, chooses one, and then moves to that coral. If it survives that travel, then it has successfully settled to the coral. We assume that corals emit a signal of magnitude $f(R)$, where R is the radius of the coral, which decays with distance from the coral:

$$\Lambda_i(x, y) := f(R)e^{-\rho d_i(x,y)} \tag{5.1}$$

where $d_i(x, y)$ is the distance from the potential settler's location (x, y) to the edge of coral i (the center of the coral is located at (x_i, y_i)):

$$d_i(x, y) := \max(0, \sqrt{(x - x_i)^2 + (y - y_i)^2} - R). \tag{5.2}$$

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The probability that a settler will choose coral i is assumed proportional to the relative signals it receives from all corals in the landscape, $\Lambda_i(x, y)/\left(\sum_j \Lambda_j(x, y)\right)$, as long as the total signal strength is greater than the minimum detection threshold, θ. After choosing a coral, the settler must survive travel to that coral from its arrival location. We assume that the transit mortality occurs at a constant rate and thus has the form $e^{-\mu d_i(x,y)}$, where μ is the transit motality rate (although expressed per distance rather than per time) rate. Therefore, the probability that a settler will settle to (i.e. arrive at) coral i is

$$p_i = \frac{1}{|\mathcal{O}|} \iint_{\mathcal{O}} \frac{\Lambda_i(x,y)}{\sum_j \Lambda_j(x,y)} e^{-\mu d_i(x,y)}\, dx\, dy. \tag{5.3}$$

5.3.2 Coral growth

We model coral growth as the linear extension of the two-dimensional coral, and assume it to be a function of coral size, symbiont density, and symbiont effect. Thus, the change in the radius of a coral (over a time step) is simply the linear extension, G_i:

$$R_{i,t+1} = R_{i,t} + G_i \tag{5.4}$$

We then must determine the best way to model linear exention (i.e., G). In particular, we must specify how growth varies as a function of symbiont density and of coral size. We begin by specifying the effect of symbiont density. We assume that a symbiont has an effect E (positive for beneficial symbionts and negative for harmful symbionts). Thus, the total symbiont poulation of a species can be expressed as ED_i, where D_i is the density of the symbiont inhabiting coral i. We assume that coral growth rate asymptotes as $D \to \infty$. In other words, we assume that the addition of another symbiont will have no effect on the corals growth rate when symbiont density is already very high. We assume that the effect of one symbiont is greatest at low densities. Therefore, the growth rate of a coral during one timestep is,

$$G_i = \frac{g_i(R)}{1 + e^{-E*D_i}} \tag{5.5}$$

where, $g_i(R)/2$ is the size specific linear extension rate for a coral without symbionts (i.e., $G(D = 0)$). As the density of a beneficial symbiont increases, the growth rate approaches $g_i(R)$; whereas as the density of a harmful symbiont increases, the growth rate approaches 0.

There is no consensus concerning the general relationship between size and growth. While

some studies show coral growth is independent of size (Kinzie & Sarmiento, 1986, e.g. in *Pocillopora damicornis*), others indicate that growth decreases with size (Hughes & Connell, 1987). We chose to model growth as size-dependent, with growth rate decreasing as size increased, and eventually approaching 0 as the coral size approached a maximum size:

$$g_i = g_{max}\left(1 - \frac{1}{e^{R_{max}-R_i)}}\right), \tag{5.6}$$

where g_{max} is the maximum amount of linear extension per timestep, R_i is the radius of coral i, and R_{max} is the maximum size of the coral. For our simulations, we set R_{max} at 2.5m based on Soong (1993)). Thus, combining Eq 3-5, we obtain:

$$R_{i,t+1} = R_{i,t} + \frac{g_{max}\left(1 - \frac{1}{e^{R_{max}-R_i)}}\right)}{1 + e^{-E*D_i}} \tag{5.7}$$

5.3.3 Coral survival

For coral survival, we consider two components: size-dependent survival (Eq. 5.8), and symbiont-dependent survival (Eq. 5.9). We assume that survival increases with coral size:

$$S_{i,R} = S_{min} + \frac{R_i\left(S_{max} - S_{min}\right)}{k + R_i} \tag{5.8}$$

where k is a constant, S_{max} is maximum coral survival (i.e. for the largest corals), S_{min} is the minimum probability of survival (i.e. for the smallest corals), and R_i is the radius of coral i. Thus, even the largest coral has a non-zero probability of dying (e.g. due to a cyclone or anchor).

Certain species of coral symbionts improve coral survival rates by defending against coral predators, clearing sediments, etc. Other species increase mortality by consuming coral tissue. As in Eq. 5.6 for growth, we assume that survival saturates as symbiont density increases. Therefore, the survival function based on symbiont density is

$$S_{i,D} = S_{min} + \frac{S_{max}}{1 + e^{-E*D_i}}. \tag{5.9}$$

5.3.4 Coral settlement

Corals settle at rate C with their arrival locations (x, y) drawn from a uniform distribution across the landscape. We assume all coral settles with starting size R_{min}.

5.3.5 Notes on coral size, signal, and the number and density of symbionts

Because we model coral signal production relative to coral size, and the density of symbionts affects coral growth and survival, we explored the effects of various initial coral signals and the relationship between coral size, signal and symbiont density. We start by considering a completely isolated coral, where the number of settlers (λ_{iso})it receives is the combination of the settlers that land on the coral $L\pi R^2$, and the settlers that arrive elsewhere and survive travel to the coral,

$$\lambda_{iso} = L\pi R^2 + \frac{2\pi L}{\mu^2}\left(e^{-\mu R}(\mu R + 1) - \left(\frac{m}{\theta}\right)^{-\mu/\rho}\left(\frac{\mu}{\rho}\ln(m/\theta) + 1\right)\right) \tag{5.10}$$

where R is the radius of the coral, μ is the survival decay constant, θ is the signal detection threshold, L is larval density, m is initial signal magnitude (a function of R). We start by assuming that signal magnitude is proportional to the radius of the coral, and model $m = R$.

If we plot the number of settlers as a function of coral radius (Fig. 5.3), separating the settlers that arrive on the coral from those that travel to the coral, we see that for our parameters (Table 5.1), the attraction region contributes the majority of settlers to the coral (Fig. 5.3C). However, as coral size increases, the area of the attraction region increases linearly (Fig. 5.3B,E), while those that land on the coral increases by R^2 (Fig. 5.3A,D). This leads to more settlers, although the relative importance of the attraction basin decreases

(Fig. 5.3F).

However, this analysis focuses on the number of settlers, rather than density. Yet, we assume it is density that influences coral growth and survival. While the number of symbionts increases with coral size, the density of settlers decreases for the isolated coral (Fig. 5.3). This result has potentially interesting ramifications on the effect of symbionts to coral growth because depending on the effect of the symbiont, it can diminish the advantages of being a large colony, as the symbiont effect will be diluted (and there will be weaker effects of redirection on the coral colony). For harmful symbionts, this phenomena will benefit corals as they grow out of the size range where symbiont effects are greater due to higher settler densities. Of course, the implications of this result will depend on whether the effects of symbionts are density or abundance-dependent. In some instances, simply having more symbionts might matter more than density.

To compare the effect of initial signal magnitude, we compared abundance and density for corals with three functions for initial signal magnitude $m = R$, $m = R^2$, and $m = e^R$. The initial signal form did not change the overall dynamics as the area of the coral continued to grow much larger than the attraction region (Fig. 5.4).

We now move to looking at two corals, C_1 and C_2 that compete for settlers. The respective probabilities that a given larval symbiont settles on one or the other are:

$$P(C_1) = \int_y \int_x \frac{m_1 e^{-\rho\sqrt{x^2+y^2}-R_1} e^{-\mu\sqrt{x^2+y^2}-R_1}}{m_1 e^{-\rho\sqrt{x^2+y^2}-R_1} + m_2 e^{-\rho\sqrt{(x-d)^2+y^2}-R_2}} \tag{5.11}$$

$$P(C_2) = \int_y \int_x \frac{m_2 e^{-\rho\sqrt{(x-d)^2+y^2}-R_2} e^{-\mu\sqrt{(x-d)^2+y^2}-R_2}}{m_1 e^{-\rho\sqrt{x^2+y^2}-R_1} + m_2 e^{-\rho\sqrt{(x-d)^2+y^2}-R_2}} \tag{5.12}$$

We integrate over the area defined by the detection threshold, θ. Because this threshold refers to the where the total signal (i.e. sum of the sources) exceeds the threshold, the actual integration limits for equations 5.11 and 5.12 is fairly complicated. $\theta = m_1 e^{-\rho\sqrt{x^2+y^2}-R_1} + m_2 e^{-\rho\sqrt{(x-d)^2+y^2}-R_2}$, so to simplify, let the bounds on x will be determined by the coral

on that side (Coral 1 for the minimum, Coral 2 for the maximum). The y bounds will be determined by the larger coral. These bounds are based on the single coral exploration are

$$x_{min} = -\left(\frac{\ln(m_1/\theta)}{\rho} + R_1\right) \tag{5.13}$$

$$x_{max} = \left(\frac{\ln(m_2/\theta)}{\rho} + R_2\right) + d \tag{5.14}$$

$$y = \pm\left(\frac{\ln(\max(m_1, m_2)/\theta)}{\rho} + \max(R_1, R_2)\right) \tag{5.15}$$

We start by considering two corals of equal size (Fig. 5.5). When both corals are of equal size, they have identical settlement rates that asymptote when the distance between the corals is large: i.e. each coral is essentially isolated and thus the respective number of settlers converges to λ_{iso} and the density converges to $\frac{\lambda_{iso}}{\pi R^2}$. However, larger corals of equal size (i.e. both corals have $R = 2$ rather than $R = 1$, density is lower for both corals (compare solid lines ($R = 1$) to dotted lines ($R = 2$ in Fig. 5.5). However, when corals are of unequal size, the larger coral has an advantage in attracting symbionts. This competitive advantage reduces settlement to the small coral (Fig. 5.6). This effect is magnified when the coral initial signal scales to the area of the coral R^2 rather than the perimeter R (Fig. 5.6).

5.4 Simulations

We simulated dynamics following the algorithm and functional forms above on small landscapes measuring 50m by 50m and for at least 100 timesteps using the values in Table 5.1. We contrasted two settlement scenarios 1) propagule redirection (Stier & Osenberg, 2010; Hamman *et al.*, in prep), in which coral compete for settlers, and 2) the field of dreams hypothesis (Palmer *et al.*, 1997). For all simulations, initial coral signal magnitude was R, or coral radius. For each simulation, we recorded information on both the symbiont and coral populations. To quantify the distribution of symbiont densities among corals, we used

an index of dispersion (var/mean, =1 under complete spatial randomness). To describe the coral population, we quantified the variance in the size distribution, and the number of corals in the landscape.

Without propagule redirection (i.e., under the field of dreams scenario), very little heterogeneity was observed in settlement or occupant patterns, with only small amounts of heterogeneity occurring when symbionts were harmful, and slight overdispersion occurring when symbionts were beneficial to corals (Fig. 5.7A). Similarly, a small amount of heterogeneity (i.e., overdispersion) persisted in occupant distributions, but only under moderate density-dependent symbiont mortality (Fig. 5.7B). Corals with harmful symbionts had lower rates of growth and survival, and generated very small amounts of heterogeneity. However, that small amount of heterogeneity decreased under strong density-dependent symbiont mortality (triangles in Fig. 5.7A,B).

In the presence of propagule redirection, the effect of symbionts on coral was more pronounced. When symbionts harmed coral, the index of dispersion for settlers was at least five times greater than that observed in the presence of symbionts that did not have any effects on coral (Fig. 5.7C). The effect on occupants was even greater: i.e., the index of dispersion was ten times greater than observed with "neutral" symbionts (Fig. 5.7D). These effects were greatly reduced when density-dependence was stronger. In contrast, beneficial symbionts had levels of heterogeneity that were slightly reduced compared to the situation when symbionts did not affect corals.

Without progagule redirection, coral populations showed no strong responses to symbionts: neither the number corals nor the variation in coral size varied appreciably due to the symbiont effect or the strength of density-dependence (Fig. 5.8 A,B). In contrast, in the presence of propagule redirection and moderate density-dependence, there were strong effects when symbionts harmed coral. As symbiont effects became more deleterious, corals died at younger sizes, leading to reduced number of corals that were less variable in size (Fig. 5.8 C, D).

5.5 Discussion

Interactions between biogenic habitat and their symbionts can create feedbacks that generate spatial pattern in occupant distributions, and alter size distributions and abundance for the habitat patch. The amount of variation, both among symbiont and coral populations, is mediated through variation in symbiont distributions. We observed increased heterogeneity only when there was moderate (not strong) density-dependent mortality in the symbiont population (Figs. 5.7,5.8), creating different symbiont effects across the landscape. When symbiont density-dependent mortality increased, reducing the variation in symbionts across the landscape, the distribution of both corals and symbionts became more homogeneous. Additionally, the heterogeneity was generated by propagule redirection, as little heterogeneity existed when settlement to a coral was independent of the landscape configuration (i.e. under a "field of dreams" scenario): compare panels A and B with C and D in Figs. 5.7,5.8. The heterogeneity in settler and occupant distributions was created by interactions between propagule redirection and the symbiont effect (the greatest amount of heterogeneity occurred when harmful symbionts exhibited propagule redirection).

This model ignores many complexities or unknown ecological factors. We assumed all symbionts in a given simulation were identical, whereas in many natural systems, beneficial and harmful symbionts occupy the same patches (e.g. some corals host both vermetid gastropods and guard crabs (Stier *et al.*, 2010)). We also assumed habitat quality was consistent among all habitat patches. Including variation in quality would introduce additional heterogeneity, although previous studies indicate relative magnitude heterogeneity due to propgaule redirection and variation in quality are similar (Hamman *et al.*, in prep). Yet, coral patches vary greatly in quality, due to variation within and among coral taxa in terms of quality that influences fish settlement (Shima & Osenberg, 2003; Dixson *et al.*, 2014). Including variation in quality which would introduce additional heterogeneity; however, our previous simulations of propagule redirection (with static habitat) suggested that

the effects of propgaule redirection and habitat quality were approximately comparable in magnitude (Hamman *et al.*, in prep). We also assumed habitat quality was constant through time, where habitat qualtity likely varies in natural systems due to environmental factors (Hoegh-Guldberg *et al.*, 2007, e.g. climate change and ocean acidification), or due to biotic interactions. For example, corallivores could be attracted to damaged coral resulting from predation (Hamman, in prep). Furthermore, (e.g. predation increases the vulnerability of corals vulnerable to disease, resulting in lower quality corals (Katz *et al.*, 2014). In addition, some coral symbionts could exhibit conspecific attraction (e.g. Turner *et al.*, 1994; Lecchini *et al.*, 2005, 2007), which could enhance heterogeneity in the density of symbionts and provide a form of positive feedback to patterns initially established through propagule redirection.

We also assumed once a symbiont settled, they remained on the coral, and if the coral died, the symbionts also died. However, if symbionts are able to move post-settlement, interesting patterns might emerge from harmful symbionts that consume and kill a coral, then move to neighboring corals. This movement, along with dispersal limitations results in pattern formation in other systems with harmful symbionts (e.g. tussock moths Maron & Harrison, 1997). Future studies that incorporate these additional complexities will lead to greater understanding of how interactions between habitats and occupants generate spatial pattern.

5.6 Tables and Figures

Parameters		
Description	Label	Values Used for Stochastic Simulations
initial coral radius	R_{min}	0.1m
maximum coral radius	R_{max}	2.5m
coral signal decay rate	ρ	0.75 m^{-1}
larvae survival decay rate	μ	0.4 m^{-1}
density–independent mortality rate	α	0.0001 day^{-1}
time between settlement events	γ	28 days
density–dependent mortality rate	β	0.0005 and 0.05 m^2 $fish^{-1}day^{-1}$
larval density	L	0.33 larvae m^{-2}
coral recruitment rate	C	5 corals $month^{-1}$
coral survival constant	k	0.1
coral minimum survival	S_{min}	0.5 $month^{-1}$
coral maximum survival	S_{max}	0.98 $month^{-1}$

Table 5.1: List of parameters

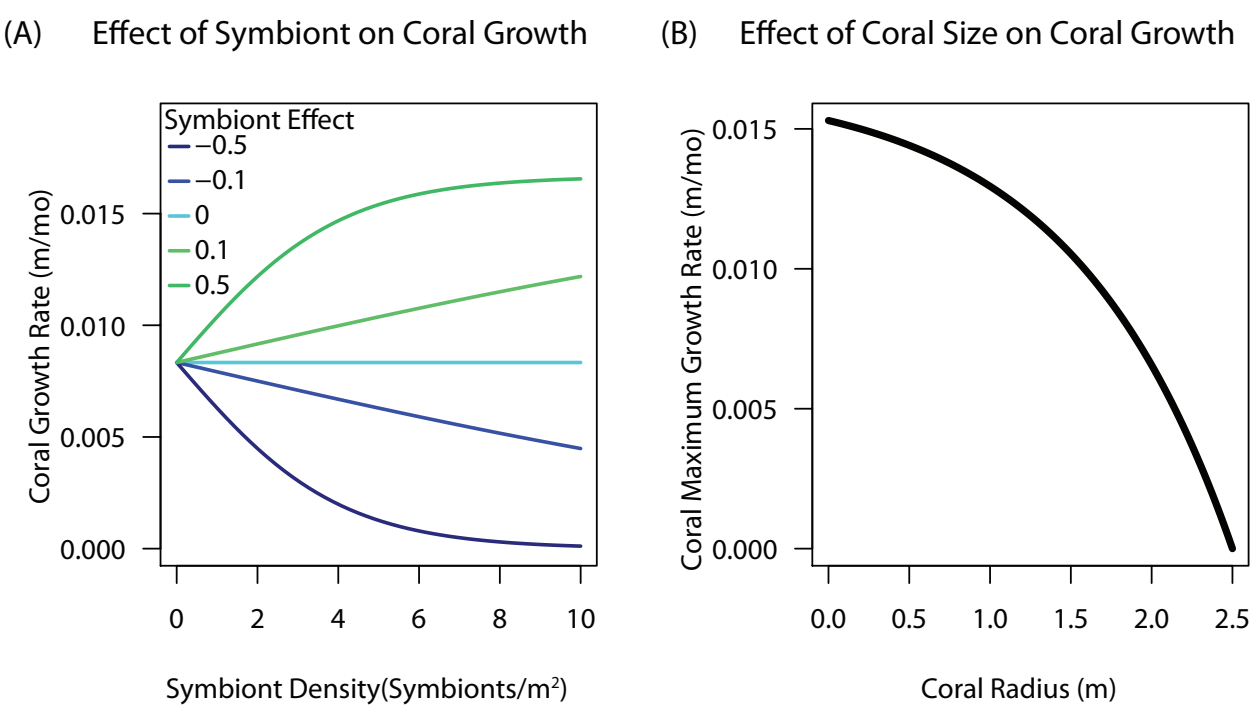

Figure 5.1: The effect of symbiont density (A) and coral size (B) on coral monthly growth rates. The maximum coral growth rate is determined by size (B), and modified by the symbiont community.

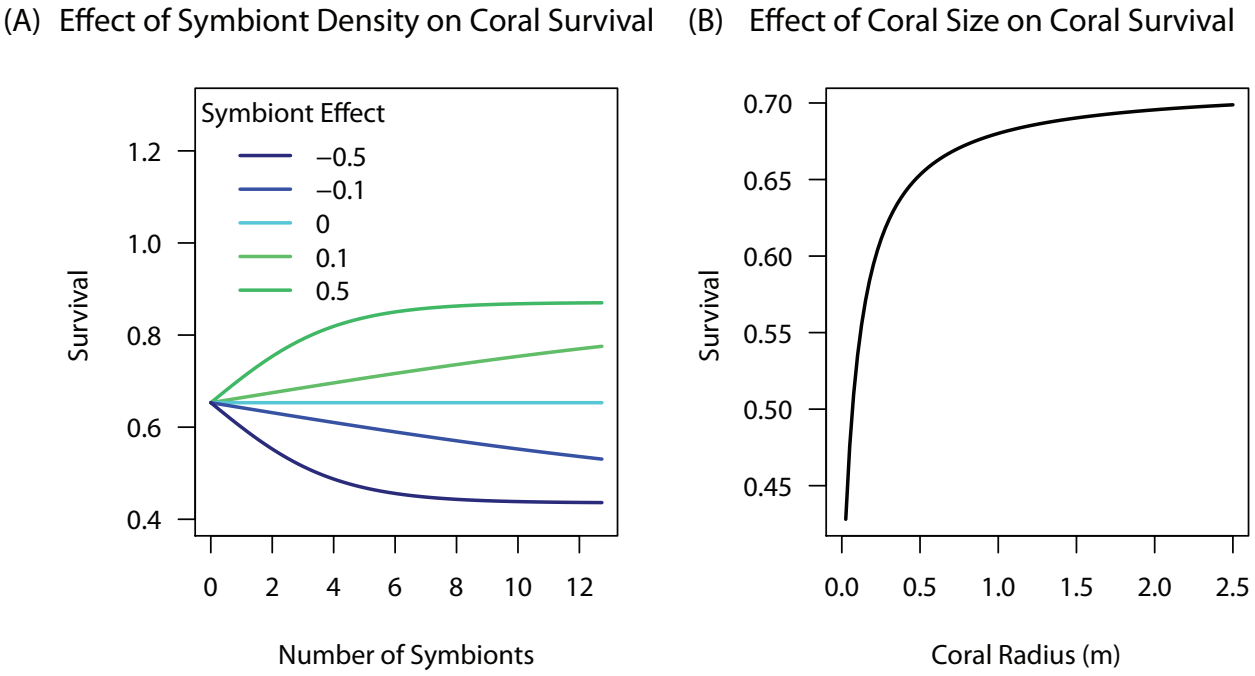

Figure 5.2: The effect of symbiont density (A) and coral size (B) on coral monthly survival rates.

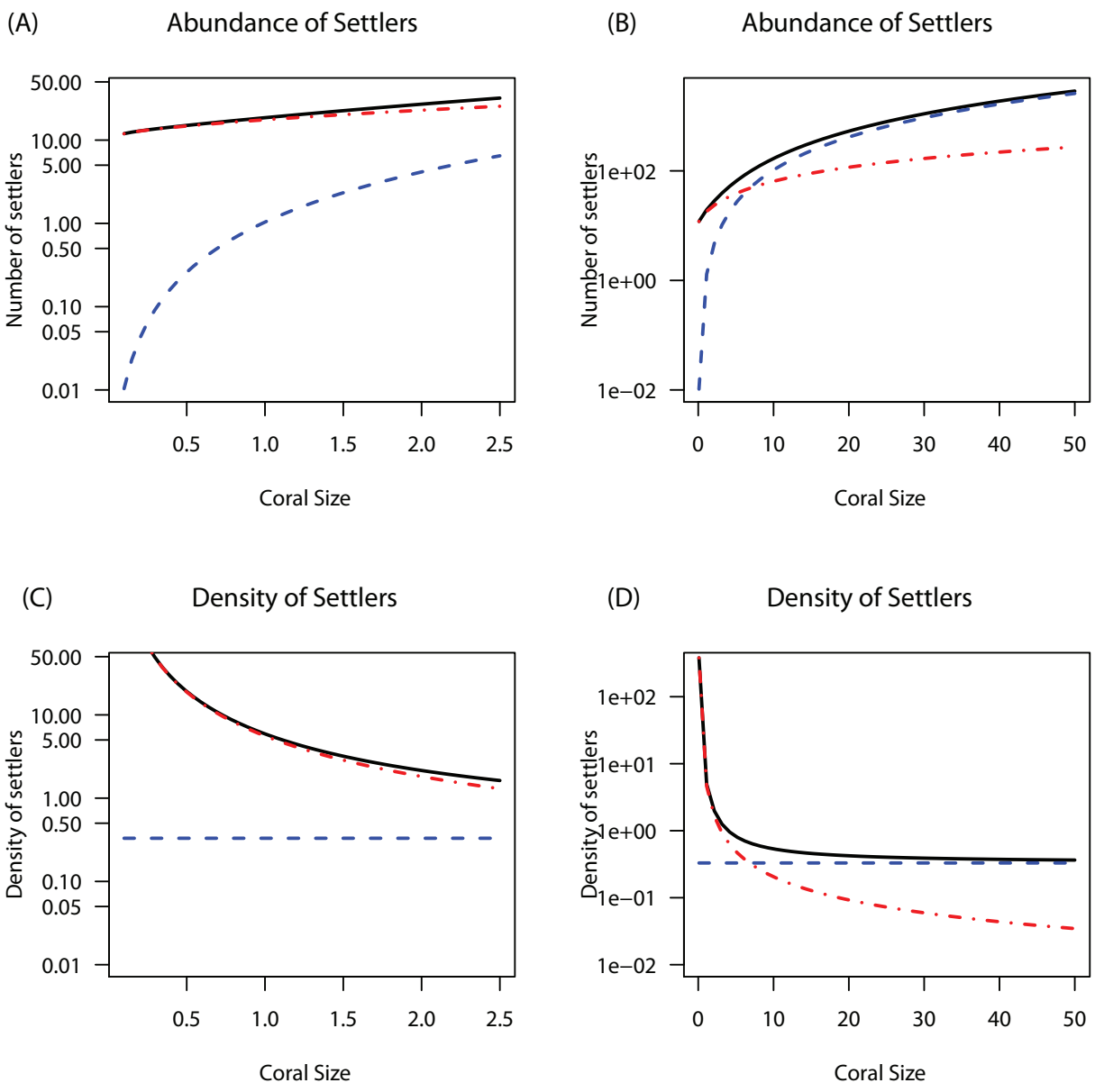

Figure 5.3: The effect of coral size on settlement numbers (Panels A and B) and settlement density (Panels C and D). Red dashed lines show the contribution of the attraction region, blue dashed lines the contribution of the coral (settlers that arrive directly on the coral), and black solid lines total settlement

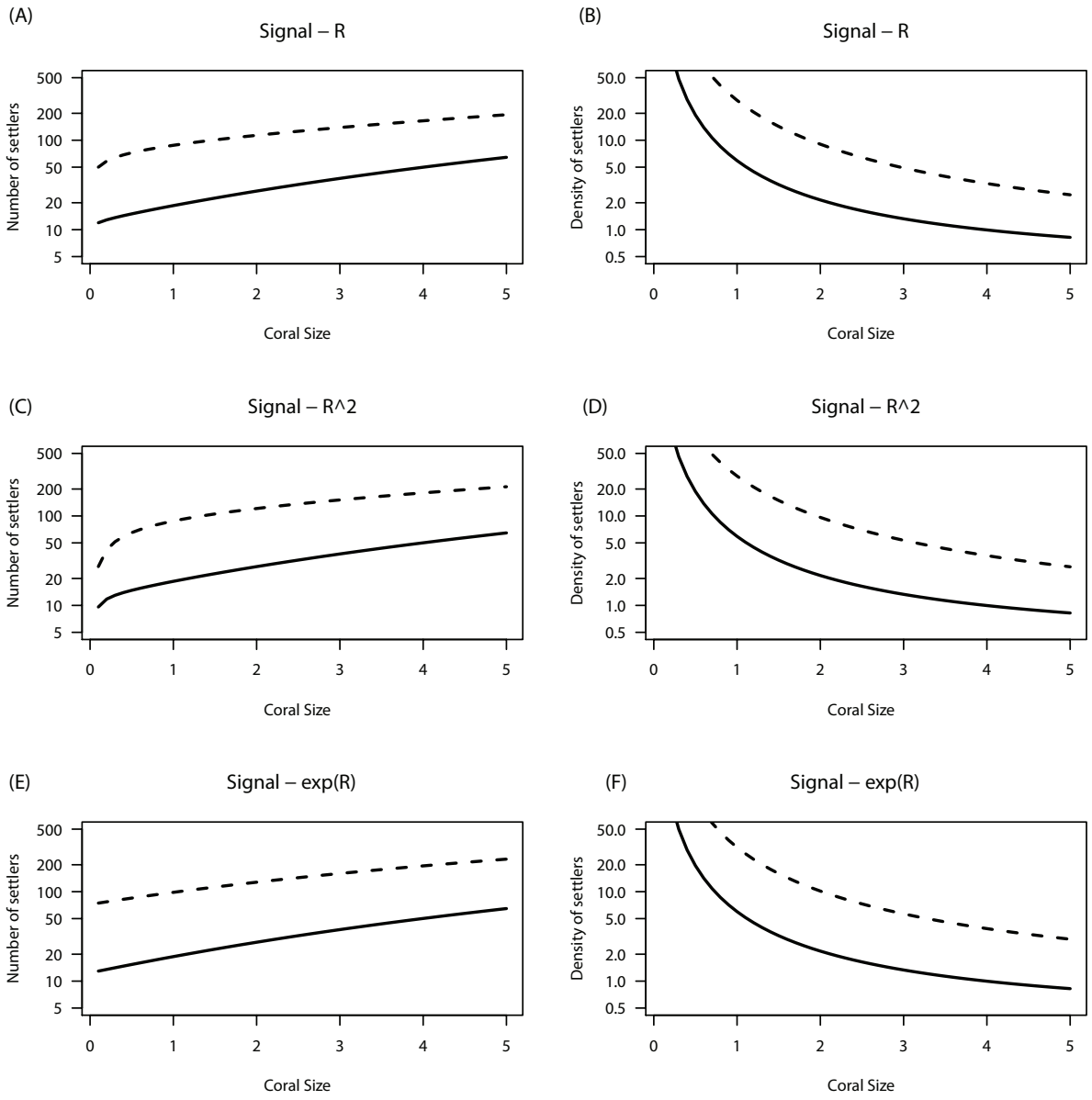

Figure 5.4: Abundance (A,C,E) and density (B,D,F) of settlers to corals with different signal magnitudes: R (A,B), R^2 (C,D), and e^R (E,F). Solid lines indicate $\mu = 0.4$, and dashed lines $\mu = 0.1$

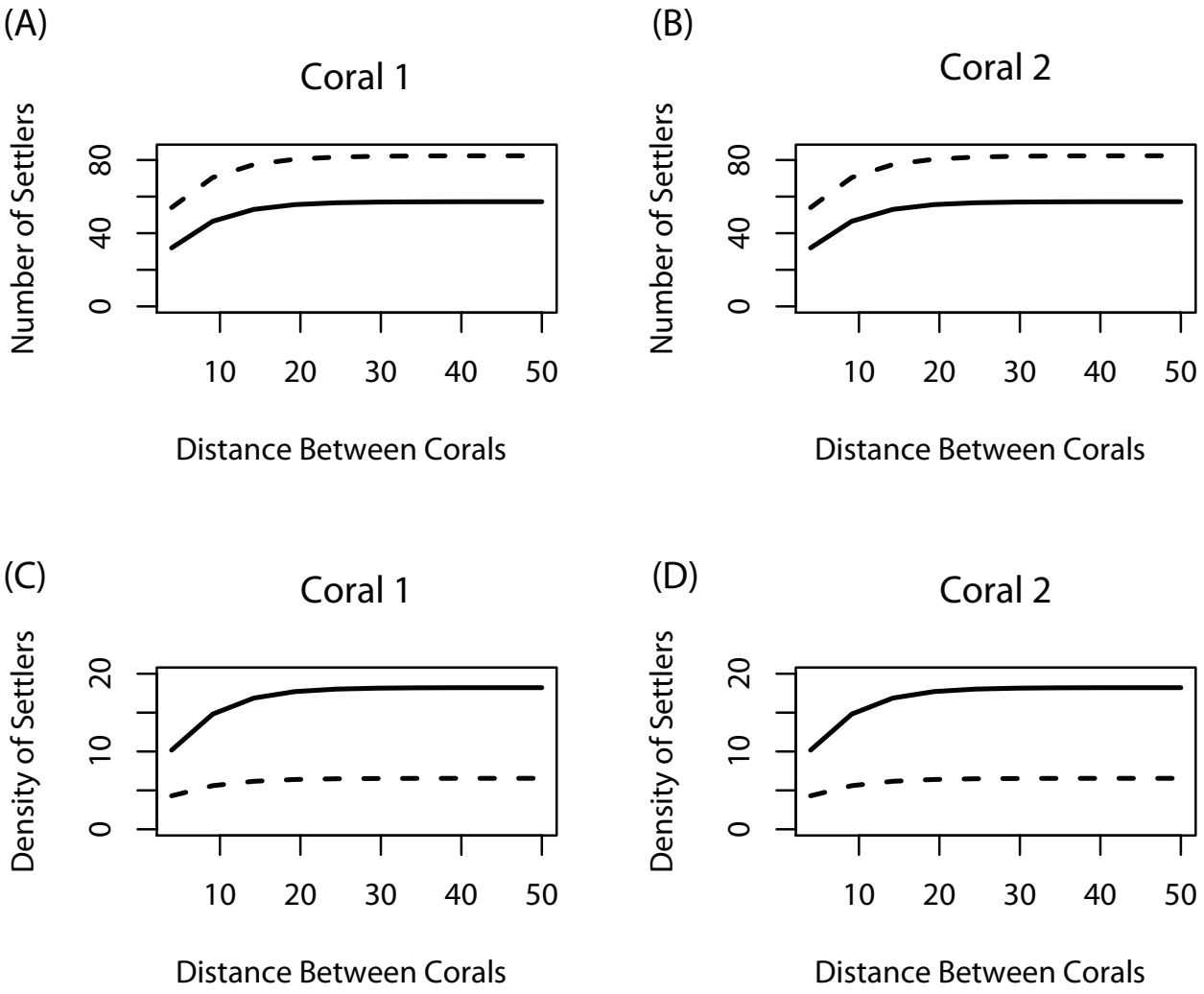

Figure 5.5: Two corals competing for settlers and the effects of distance between coral centroids on the number of settlers (A, B), and the density of settlers (C,D). Solid lines indicate $m = R = 1$, dotted lines lines indicate $m = R = 2$. For both corals, abundance of settlers increases with size and distance between corals, density increases with distance between corals, but symbiont density decreases with coral size.

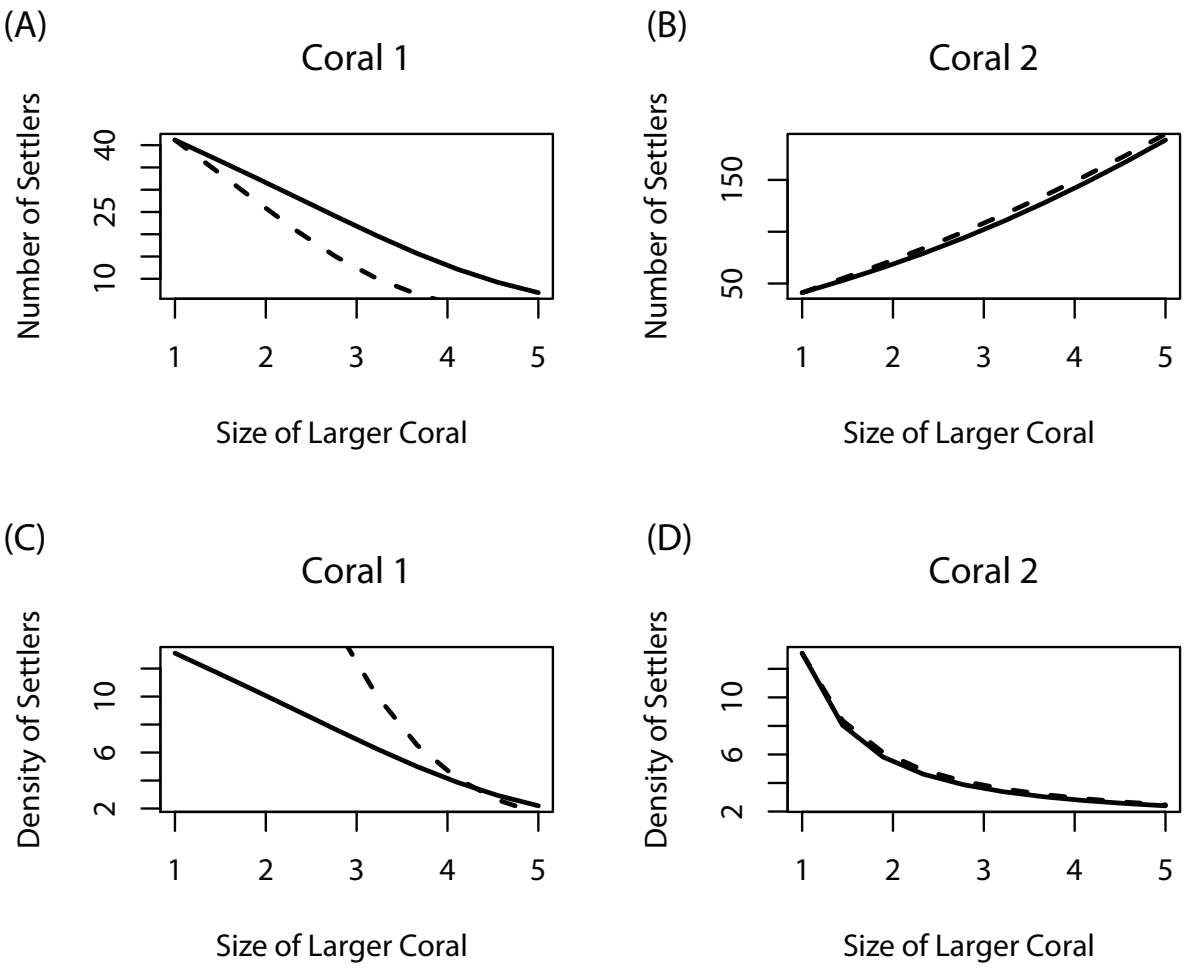

Figure 5.6: Two corals competing for settlers and the effects of distance between coral centroids on the number of settlers (A, B), and the density of settlers (C,D). Solid lines indicate $m = R = 1$, dotted lines lines indicate $m = R^2$. Coral edges are separated by $2m$

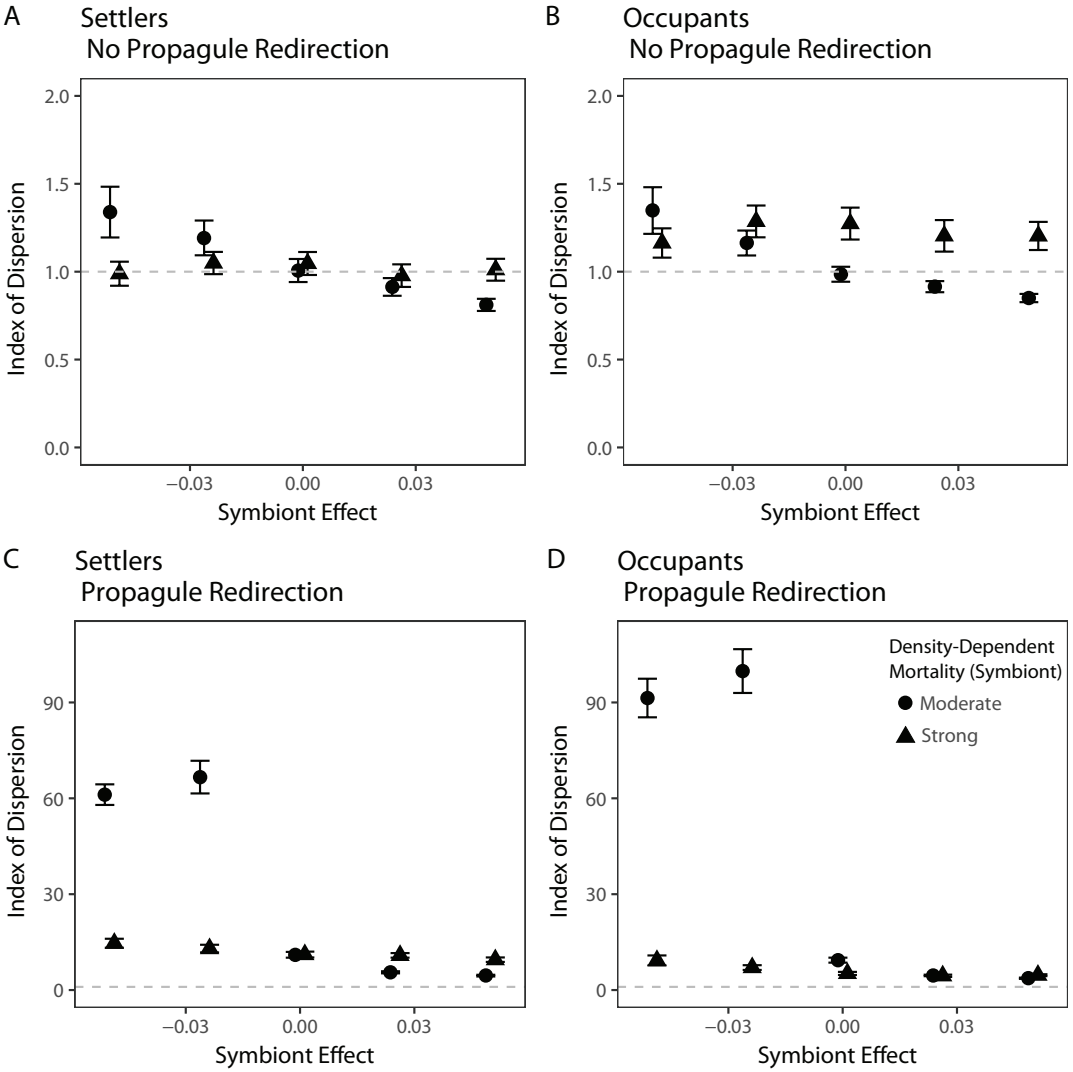

Figure 5.7: Index of dispersion (variance/mean) for settlers (A,C) to and occupants on (B,D) corals that were settled under field of dreams (A, B) and propagule redirection (C,D). The dotted line at 1 indicates complete spatial randomness. Circles indicate moderate density-dependent mortality in the symbiont population ($\beta = 0.005$), and triangles indicate strong density-dependent mortality for symbionts ($\beta = 0.05$). Propagule redirection generated more heterogeneity in both settlers and occupants than field of dreams, especially when symbionts negatively affected coral growth and survival. Error bars represent ± 95% confidence intervals

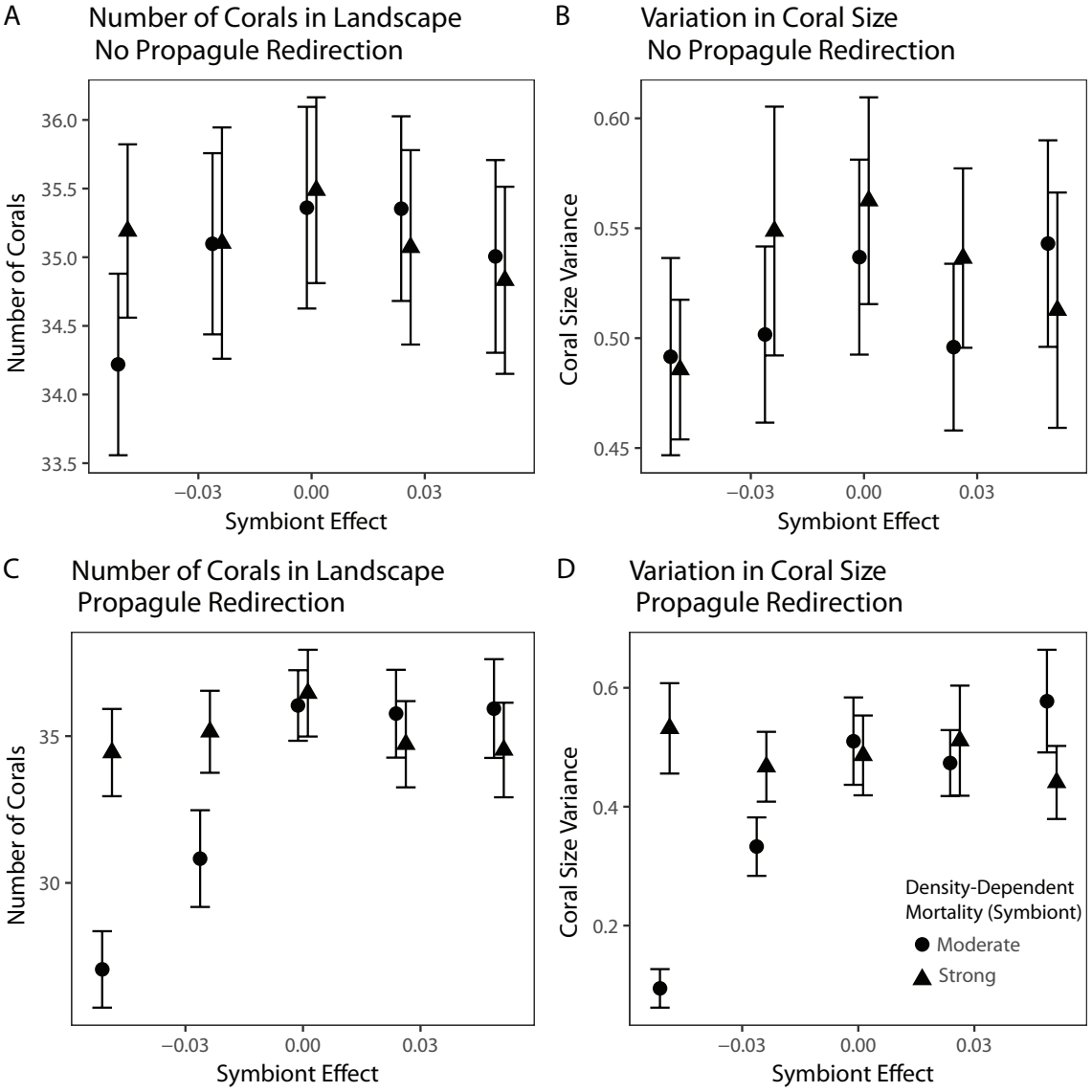

Figure 5.8: The number of corals in a landscape (A,C), and the variance of coral sizes (B,D) for simulations done without (A,B) and with propagule redirection (C,D). Circles indicate moderate density-dependent mortality in the symbiont population ($\beta = 0.005$), and triangles indicate strong density-dependent mortality for symbionts ($\beta = 0.05$). Error bars $\pm$ 95% confidence interval

Summary

In this dissertation, I explored how feedbacks between habitats and occupants create spatial pattern in both the occupant and the habitat. When occupants affect the habitat, either positively or negatively, spatial patterns can emerge due to feedbacks within the system. For example, the spatial arrangement of patches in a landscape can affect the abundance of occupants in each patch, and cues arising from conspecifics or feeding behaviors can lead to aggregations of habitat consumers. I studied these interactions in a coral reef system, and examined both localized effects of occupants on coral morphology and large-scale spatial population dynamics.

In Chapter 2, I showed how corallivorous gastropods, *Drupella cornus* and *Coralliophila violacea*, are aggregated across multiple spatial scales, (Figs. 2.1 and 2.2). At small spatial scales, chemical clues likely play an important role in these aggregations, and I tested the attractiveness of multiple cues using choice trials. While both snails respond to chemical cues, the two snails are primarily attracted to different signals. *D. cornus* is attracted to cues from corals where conspecifics have previously fed, while *C. violacea* is attracted to damaged corals (Fig. 2.3). Aggregations of snails decreased coral growth in proportion to snail abundance (Fig. 2.4). However, even though the snails feed via two different mechanisms, there was no difference between snails and their effects on growth. These patterns demonstrate the potential for occupant heterogeneity to affect their habitat by inducing variation in habitat growth rates driven by heterogeneity in corallivore abundance.

To build on the localized effects of damage due to aggregations of consumers, in the third chapter I examined the response of the coral to the spatial arrangement of coral lesions. First, I documented heterogeneity in size, shape, density, and spatial distribution of coral lesions at two sites on the north shore of Moorea (Fig. 3.2). I then used a field experiment to test the effect of distance between lesions, and found that increased distance between lesions increased the healing rate of the lesion, but did not affect the growth of the colony over a five week experiment (Fig. 3.3). However, after a year, lesions near one another decreased linear extension (i.e. skeletal growth) in the area of the lesions, but that was not the case for lesions further apart (Fig. 3.4). As a result of the effects on growth, interactions between corals and occupants consuming corals can alter coral morphology (Fig. 3.5). When habitat consumers seek out crevices of a biogenic habitat for protection from predators and reduce growth in that area, they can facilitate the development of morphology of the habitat by reducing growth in the crevices relative to growth away from the crevices.

Habitats also influence the spatial pattern of their occupants. In Chapter 4, I demonstrated how the spatial configuration of habitat creates long-lasting heterogeneity in the spatial distribution of the occupants. Previous empirical studies indicate that neighboring corals can "redirect" settling occupants away from focal corals (known as propagule redirection). We show that for certain parameter regimes, (low to moderate density-dependent mortality, larval supply, or high density-independent mortality), redirection can create long-lasting heterogeneity among coral patches (Figs. 4.2 4.3, and 4.4). By simulating dynamics of a realistic landscape with variation in habitat quality, we demonstrated that redirection contributes a similar amount of variation as variation in habitat quality. Thus, propagule redirection likely plays an important role in natural systems (Fig. 4.5). Expanding on Chapter 4, we incorporated habitat dynamics into the models of Chapter 5, and demonstrate how harmful symbionts increase heterogeneity in both symbiont and coral distributions (Figs. 5.7 and 5.8). In sum, these studies demonstrate the importance of including not only habitat dynamics, but also interactions with occupants in studies of spatial dynamics.

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