ABSTRACT

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Within a large-scale habitat corridor experiment, I performed both experimental and observational studies to determine the effects of habitat fragmentation, habitat edge, and patch connectivity on the movement and incidence of fungal plant diseases. Increased spread of infectious disease is often cited as a potential negative effect of habitat corridors, and increases in the amount of habitat edge that are inevitable byproducts of corridor creation could also impact the incidence and development of plant disease. However, the impacts of corridors and habitat edges on plant disease dynamics remain empirically untested.

Using sweet corn and southern corn leaf blight as a model plant-pathogen system, I experimentally tested the impacts of connectivity and habitat fragmentation on pathogen movement and disease development. I found that corridors do not facilitate the movement of wind dispersed plant pathogens, that connectivity of patches does not enhance levels of foliar fungal plant disease, and that edge effects are the key drivers of plant disease dynamics. Over time, less edgy patches had higher proportions of diseased plants, and distance of host plants to habitat edges was the greatest determinant of disease development.

To test the effects of habitat connectivity and edge on the incidence of naturally occurring plant disease, I surveyed foliar lesions on three native *Lespedeza* species. I found that connectivity of habitat patches did not affect levels of disease and that incidence of wind dispersed foliar fungal diseases was significantly higher close to habitat edges, further demonstrating that edge effects play an important role in plant disease dynamics. I also found that density of host plants was significantly higher farther from habitat edges, contradicting previous studies that relate higher host densities to increased disease load. Environmental

variables also showed strong edge effects, with significantly higher temperatures and light intensities at the interior of habitat patches, providing possible mechanisms for these disease patterns. Results from both studies show that worries over the potential harmful effects of connectivity on disease dynamics are misplaced, and that, in a conservation context, diseases can be better managed by mitigating edge effects.

The Effects of Habitat Fragmentation and Connectivity on Plant Disease

by
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BIOGRAPHY

I was born and raised in a small rural Pennsylvania town. I grew up trailing after my older siblings, reading books, and getting dirty. I began my freshman year at Ohio Wesleyan University two days before my eighteenth birthday and spent the next four years studying, playing rugby, and spending time with friends. My last two summers of college, and the three years following my graduation, were spent at various field jobs, learning new skills and making new friends. Those experiences eventually led me to a technician job with the Corridor Project at that Savannah River Site and on to graduate school at NC State.

Growing up, I was blessed to have acres on which to roam, never fully appreciating my mother's command to "go play outside". Looking back, I was fortunate to have the opportunity to explore the world around me, and feel privileged to continue these explorations in my career.

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better scientist. Special thanks to Lisa Paine and Monica Iglecia for their friendship and support.

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CHAPTER 1:

EDGE EFFECTS, NOT CONNECTIVITY, DETERMINE THE INCIDENCE AND DEVELOPMENT OF A FOLIAR FUNGAL PLANT DISEASE

ABSTRACT

Using a model plant-pathogen system in a large-scale habitat corridor experiment, I found that corridors do not facilitate the movement of wind dispersed plant pathogens, that connectivity of patches does not enhance levels of foliar fungal plant disease, and that edge effects are the key drivers of plant disease dynamics. Increased spread of infectious disease is often cited as a potential negative effect of habitat corridors, but the impacts of corridors on pathogen movement have never been tested empirically. Using sweet corn and southern corn leaf blight as a model plant-pathogen system, I tested the impacts of connectivity and habitat fragmentation on pathogen movement and disease development. Over time, less edgy patches had higher proportions of diseases plants, and distance of host plants to habitat edges was the greatest determinant of disease development. Variation in average daytime temperatures and light intensities provided a possible mechanism for these disease patterns. These results show that worries over the potential harmful effects of connectivity on disease dynamics are misplaced, and that, in a conservation context, diseases can be better managed by mitigating edge effects.

INTRODUCTION

Destruction and degradation of habitat are the primary causes of loss of biodiversity (Wilcove *et al.* 1998). Fragmentation of remaining habitat negatively impacts populations by decreasing species abundances (Gonzalez *et al.* 1998, Rantalainen *et al*. 2006), limiting species distributions (Gonzalez *et al*. 1998), and increasing rates of disease (Allan *et al*. 2003, Groppe *et al*. 2001). Habitat corridors – narrow strips of habitat that connect isolated fragments – are commonly proposed in conservation management to mitigate the negative effects of fragmentation (Hilty *et al*. 2006, Rosenberg *et al*. 1997). Corridors are theorized to promote population persistence by increasing movement between isolated fragments, and are usually targeted toward organisms with small, isolated subpopulations (Rosenberg *et al*. 1997). Empirical studies have shown that habitat corridors do benefit populations by facilitating movement of a variety of organisms (Aars and Ims 1999, Debinski and Holt 2000, Gilbert *et al*. 1998, Gonzalez *et al*. 1998, Haddad 1999, Haddad *et al*. 2003). However, corridors may also increase the movement of organisms antagonistic to species of conservation concern, including predators, competitors, and – the subject of this paper – disease (Plantagenest *et al*. 2007, Simberloff and Cox 1987, Simberloff *et al*. 1992).

Predictions that corridors may facilitate disease spread have never been tested experimentally. They have been examined theoretically, but the varying results of these studies have precluded general management recommendations. Hess (1994) found that habitat corridors "may have a dark side" by facilitating the spread of contagious diseases that increase extinctions in metapopulations. However, this increase in extinctions only occurred

under specific combinations of rates of population growth and disease induced mortality that led populations to decrease enough that stochastic population fluctuations caused extinctions (Hess 1994, Hess 1996). Gog *et al*. (2002) showed that in systems where an alternative host is present, increased spread of disease by corridors does not significantly increase extinction risks. McCallum and Dobson (2002) developed a metapopulation model that included resistant host populations and reservoir hosts, and found that the benefits of corridors outweigh the risks due to increased spread of disease. Results of such theoretical studies are often dependent on a specific set of conditions, and their relationships to real-world scenarios are not well defined.

The ambiguity of theoretical studies and complete lack of empirical research into the effects of corridors on disease movement provide considerable obstacles when assessing the usefulness of corridors in a conservation context. Knowledge of how corridors affect the spread of contagious disease is integral to the protection of species of conservation concern, and empirical tests are needed to ascertain the efficacy of corridors. Using a well-studied plant-pathogen system within a landscape-scale habitat corridor experiment, I empirically tested the effects of corridors on the movement of wind dispersed plant pathogens. I also assessed the effects of habitat patch shape and habitat edge on the incidence and severity of plant disease.

Plant diseases have an enormous impact on the regulation of plant populations (Anderson *et al*. 2004, Augspurger 1987, Hudson *et al*. 2006, Jarosz and Davelos 1995, Mitchell 2003, Mitchell and Power 2006). Diseases caused by fungi and viruses can impact

plant fitness by affecting the growth, survival, and reproduction of their hosts (Augspurger 1987, Hudson *et al*. 2006). Infection may also make plants more susceptible to herbivores or less able to compete for pollinators, further lowering plant fitness (Hudson *et al*. 2006, Jarosz and Davelos 1995). By differentially increasing mortality, diseases can alter the structure of host populations through selection for more resistant host genotypes. Evidence also exists that disease supports the evolution and maintenance of sexual reproduction in plants, which furthers the diversity of genotypic resistance (Augspurger 1987, Hudson *et al*. 2006, Jarosz and Davelos 1995). By affecting primary producers, plant diseases have the potential to influence entire communities of organisms (Mitchell and Power 2006), making knowledge of plant disease dynamics integral to understanding ecosystem functioning.

Habitat corridors can alter the interactions between plant diseases and their hosts by affecting patterns of pathogen dispersal. Although patterns of disease incidence ultimately depend on a variety of factors, including host susceptibility and environmental conditions, dispersal of disease propagules is the first step in disease spread (McCartney *et al*. 2006). For wind dispersed pathogens, any aspect of the landscape that alters wind patterns will affect the movement of plant pathogens (Plantegenest *et al*. 2007). Empirical research on how corridors alter wind movement is limited, but highlights the importance of wind direction and habitat edges when estimating how wind will be altered by corridors (Baker *et al*. 2009). Recent studies show that assemblages of wind dispersed plant species were higher in patches connected by corridors, indicating that connectivity may facilitate the movement of wind-dispersed propagules (Damschen *et al*. 2008). Since corridors have the potential to

alter wind dynamics and have been shown to affect the movement of wind dispersed seeds, it can be expected that they will also affect the movement of wind dispersed plant pathogens.

Fragmentation of habitat and addition of habitat corridors can also affect populations by increasing the amount of habitat edge (Rand *et al*. 2006, Ries *et al*. 2004). Habitat edges can affect disease dynamics by acting as barriers or filters to the movement and dispersal of disease propagules (Fagan *et al*. 1999, Plantegenest *et al*. 2007). Edges also alter environmental conditions, which affect the infection and survival of plant pathogens (Agrios 1995, Alexander *et al*. 2007, Augspurger 1987, Huber and Gillespie 1992, Jarosz and Burdon 1988, Jarosz and Davelos 1995, Laine and Hanski 2006, Mitchell and Power 2006, Tscharntke *et al*. 2002). Edges can impact plant disease dynamics by facilitating spillover of reservoir species, secondary hosts, or new vectors between two distinct habitat types, or providing unique habitat types that support novel species interactions (Davies *et al*. 2001, Fagan *et al*. 1999, Harrus and Baneth 2005, Orrock *et al*. 2003, Rand *et al*. 2006, Ries *et al*. 2004). Since habitat fragmentation and corridor addition inherently increase the amount of habitat edge, and because edges have their own impacts on plant disease, it is important to simultaneously assess the effects of connectivity and edges on plant disease dynamics.

Using a large-scale replicated habitat corridor experiment, I studied the effects of habitat corridors on the movement and incidence of wind dispersed plant disease, using sweet corn (*Zea mays*) and southern corn leaf blight (*Cochliobolus heterostrophus* anamorph *Bipolaris maydis*) as a model plant-pathogen system. I conducted three experiments over the course of two years to determine whether corridors facilitate the movement of wind-

dispersed foliar fungal plant pathogens and to assess the impacts of habitat patch shape and distance to habitat edge on the incidence and severity of disease. I tested movement of disease propagules by providing a localized source of southern corn leaf blight inocula in a central habitat patch and determining the presence of subsequent infections on plants in patches that were either connected or unconnected to the source patch. I predicted that host plants located in connected patches would show higher rates of infection than plants in unconnected patches.

I assessed the effects of habitat patch shape and distance to edge on disease incidence and severity by placing plants in habitat patches with different edge-to-area ratios and at different distances from habitat edges. I determined the presence of disease on plants and also measured the development of disease lesions over time. I predicted that edges would strongly affect the incidence and severity of disease by altering rates of infection and disease development due to differences in environmental conditions (Fagan *et al*. 1999, Huber and Gillespie 1992, Jarosz and Burdon 1988, Tscharntke *et al*. 2002). Because environmental factors are key to disease development, I used temperature and light intensity data collected within our experimental landscapes to help determine the mechanisms behind the observed patterns of disease.

METHODS

Site Description:

I conducted my study in a large scale replicated habitat corridor experiment located at the Savannah River Site, south of New Ellenton, SC. The corridor experiment began in 2000 and consists of eight 50 hectare experimental landscapes. Each replicate landscape (block) is comprised of five ~1-ha regenerating habitat patches located in the middle of a mature pine plantation matrix comprised of loblolly pine (*Pinus taeda*) and longleaf pine (*P. palustris*). Within each of the eight blocks, patches are arranged with a central 100x100 meter source patch and four peripheral patches 150 meters away. The central patch is connected to one peripheral patch by a 25 meter wide corridor (Figures 1.1 and 1.2).

The design of this experiment allows me to test whether corridors facilitate movement of organisms by providing peripheral patches that are connected and unconnected to a center patch. By placing a source of pathogens in the center patch and then measuring pathogen dispersal into the connected and unconnected peripheral patches, I can determine whether the connectivity provided by the corridor facilitates movement of the focal organisms (Tewksbury *et al*. 2002).

Corridor addition not only provides connectivity, but also adds habitat area and edge, making it necessary to differentiate the effects of connectivity from those of increased area and edge. For this reason, the three unconnected peripheral patches are of two types: rectangular or winged. Rectangular patches control for the increase in habitat area that is a byproduct of corridor addition and have the area of a corridor added onto a square habitat

patch. If an effect is due to an increase in habitat area, not to connectivity, we would see the same effect in rectangular patches as in connected patches. Rectangular patches also have a low edge-to-area ratio when compared to patches connected by a corridor. Winged patches control for the increase in both habitat area and edge and have two 75 meter dead end "corridors" on either side of a square patch. If an effect is due to an increase in habitat edge, we would see this effect in unconnected winged patches as well as connected patches. Winged patches have a higher edge-to-area ratio than either rectangular or connected patches (Tewksbury *et al*. 2002).

Study organisms:

Within the experimental landscapes, I used the model plant-pathogen system sweet corn (*Zea mays*) and southern corn leaf blight (*Cochliobolus heterostrophus* anamorph *Bipolaris maydis*) to conduct my experiments. Extensive knowledge of this economically vital agricultural crop and its diseases makes it an ideal model organism to study the movement of plant pathogens in my system. Southern corn leaf blight is a wind-dispersed foliar fungal pathogen endemic to the southern United States and currently distributed worldwide. Southern corn leaf blight is specific to corn, allowing me to control host density and eliminate complexities due to alternative hosts. The disease is polycyclic and can complete a life cycle in less than three days, allowing time for dispersal and infection throughout the course of the experiments. Infected tissue presents elongated tan to buff brown lesions between leaf veins (Figure 1.3a). Spores of southern corn leaf blight are elongate (15-20 x 70-160 micrometers), curved, and tapered at the end (Figure 1.3b) (White

1999). These spores are distinct from the majority of other fungal spores and easily identified. Importantly, neither corn nor southern corn leaf blight were found in or near (conservatively, within 5 miles) our experiment.

Data collection:

General

For all experiments, I propagated corn plants from seed (#5802 Temptation Hybrid from Park Seed Company) in eight inch plastic azalea pots using Sta-green® all purpose potting mix. I chose this variety of sweet corn because it is highly susceptible to southern corn leaf blight, increasing the likelihood of infection. I maintained the plants in greenhouses located on the Savannah River Site and watered them daily.

I propagated and maintained southern corn leaf blight inocula in petri dishes on standard media under fluorescent light. To inoculate plants, I prepared solutions of inocula by washing spores from the media into water. I used a hemacytometer to determine the concentration of spores in solution. I used southern corn leaf blight spores present on a media of sorghum pellets as a secondary method to inoculate plants. I also used *B. maydis* spores propagated on oat grain as an independent source of inocula by placing them on the soil surface.

Experiment one

I placed non-inoculated plants (39-42 days old) at two distances (seven meters and 40 meters) from the corner of each patch in all five patches at all eight sites (for a total of 8 plants/patch; Figure 1.1). In the center patches, I placed two pots at each location (16

plants/patch) in order to increase the amount of source inocula that would be present. Plants remained in the field for approximately 50 days (June to July, 2008) and were hand watered as needed.

To test the effect of corridors on the movement of southern corn leaf blight, I provided a source of spores by inoculating only plants in the center patches. I inoculated the plants in the center patches four days after placing them into the field by dropping two sorghum pellets covered with southern corn leaf blight spores into the whorl of each plant and pipetting 2-3 mL of water over the pellets. I also placed approximately one tablespoon of oat grain on which southern corn leaf blight spores were growing into each pot. I reinoculated plants eleven days after placing them into the field by pipetting approximately 2 mL of a 5000 spores/mL solution of inocula into the whorl of each plant. I additionally placed approximately one tablespoon of oat grain onto the soil in each pot 37 days after plants were placed into the field.

I collected data on presence or absence of disease lesions during four separate sampling periods: 14-19 days, 26-27 days, 33-36 days, and 47-49 days after placing plants into the field. I classified disease as present or absent on each of the top five leaves of each plant. I photographed lesions during the first, third and fourth data collection periods. I collected tissue samples during the fourth data collection period by cutting out pieces of leaf tissue that had lesions and placing them into sealed plastic petri dishes.

I analyzed tissue samples by placing them in a moist chamber under a fluorescent light to induce sporulation. I determined positive samples by identifying the spores of

southern corn leaf blight using a dissecting microscope. I also determined presence or absence of disease using the photographs of lesions taken in of the fourth sampling period. Using tissue samples and photographs of lesions, I scored each plant at positive, negative, or indeterminate for disease.

I analyzed data from experiment one to test for the effects of patch shape, connectivity, and distance to edge on the presence of disease (analysis described below). Based on the results of experiment one, I conducted two separate experiments the following year.

Experiment two

I designed experiment two to examine the effects of corridors on the movement of southern corn leaf blight spores while removing the confounding factor of differing environmental conditions on disease development. Unlike experiment one, I placed all plants at the same distance from habitat edges. I also placed plants in the field for a shorter time, allowing primary dispersal from source plants located in the center patches, but removing plants from the field before secondary or within-patch dispersals could occur. I created uniform environmental conditions for development of diseased lesions, which were used to assess where spores landed.

During each of four sampling periods, I placed plants (39-41 days old) into all five patches at two of the eight corridor sites. I repeated over four consecutive sampling periods until the experiment had been conducted in all eight sites (May to June, 2009). In each site, I placed eight non-inoculated plants into each peripheral patch and twelve inoculated plants

into each center patch. I distributed the plants evenly around the center 25x25 meters of each patch (Figure 1.1).

To ensure high amounts of source inocula, I inoculated center patch plants prior to placing them into the field. I inoculated plants at 31 and 35 days after germination by pipetting 2 mL of a 2000 spores/mL solution into the whorl of each plant. To avoid contamination, I grew and inoculated these plants in a separate greenhouse located 3.5 miles from the greenhouse where I grew the non-inoculated plants. I moved all non-inoculated plants into the field prior to moving inoculated plants and covered inoculated plants with individual plastic bags.

I collected peripheral patch plants six days after placing them into the field. I placed each plant into an individual plastic bag to prevent cross-contamination during transport and to provide standard environments for spores to infect and develop into lesions. After the plants had been covered for 5-6 days, I collected tissue samples from the plants by clipping lesions from leaf tissue and placing them into individual petri dishes.

I analyzed tissue samples using the same protocol as in experiment one. I did not use photographs to determine disease in this experiment and all plants were scored as either positive or negative for disease based on tissue samples.

Experiment three

I designed experiment three to assess the effects of patch shape and distance to edge on disease development. Unlike experiments one and two, I did not test spore dispersal but instead inoculated all plants equally to determine differences in disease development.

During a single sampling period (June to July, 2009), I placed non-inoculated plants (26 days old) into ten winged and ten rectangular patches among eight blocks. I placed plants at three distances (zero, 20, and 40 meters) from the north and south facing corners of each patch (Figure 1.1).

I inoculated all plants two days after placing them into the field by pipetting 2 mL of a solution of 2000-4000 spores/mL into the whorl of each plant. Plants had produced lesions three days after inoculation.

Six days after inoculation, I marked four inch sections on each of three leaves per plant (Figure 1.3). I photographed the marked sections six, ten, 14, and 18 days after inoculation. I analyzed the photographs to determine the percent of marked leaf area covered with lesions and the percent of leaf area damaged by herbivores for all leaves. I analyzed data to test for effects of patch shape and distance to edge on the percent of marked leaf area covered with lesions over time and to determine whether levels of herbivory affected levels of disease.

Environmental data

Temperature and light intensity data were collected using HOBO data loggers between 16 June and 14 July, 2008. Data loggers were placed in 16 locations in each patch: approximately 2, 14.5, 27, and 42 meters along diagonals transects coming from the corners of the patches. Data were recorded in all five patches at each site every ten minutes for four days per site. For analyses, only data recorded between 7:00 AM and 7:00 PM were used to avoid bias due to nighttime uniformity of light and temperature.

Because southern corn leaf blight pathogens are wind dispersed, I used data collected by Department of Energy meteorological stations to determine average wind directions for experiment two. Meteorological stations are located approximately 4.4 to 9.2 kilometers from our sites and collect data on wind speed and direction 61 meters above the ground every 15 seconds.

Statistical analyses:

In experiment one, I analyzed the effects of patch shape and distance to edge on the presence of southern corn leaf blight. For each distance within each patch, I summed the number of plants showing presence of disease and the total number of all plants examined. The dependent variable was the ratio of infected plants over the total number of plants measured. Independent variables included fixed effects of distance from edge and patch shape and a random effect of site. I analyzed data in SAS 9.1.3 using a generalized linear mixed model (PROC GLIMMIX) and specifying a binomial distribution and logistic link for the data.

I analyzed experiment two similarly to experiment one, omitting the variable of distance to edge in all aspects of the analyses.

I analyzed experiment three to determine the effects of distance to edge and patch shape on disease development and to determine whether levels of herbivory affected levels of disease. I determined the percentages of leaf area covered by lesions and damaged by herbivores for rounds one and four of sampling and used these data to determine any differences in the initial or final amounts of disease and herbivory. For each distance within

each patch, I averaged the percentages of leaf area covered by lesions and herbivory. Percent covered by lesions was used as the dependent variable and distance to edge, patch shape, a distance by shape interaction, and percent herbivory were used as fixed independent variables. Site was used as a random independent variable.

To assess disease development, I determined the increase in percent leaf area covered by lesions between the first and fourth sampling periods and used this as a dependent variable. For each distance within each patch, I averaged the difference in percent leaf area covered by lesions and analyzed this response using fixed independent variables of distance to edge, patch shape, and a distance by shape interaction. I used site as a random independent variable. I performed all analyses for experiment three in SAS 9.1.3 using a mixed model (PROC MIXED).

I analyzed temperature data to determine the effects to patch shape and distance to edge on temperature and light intensity. For each distance within each patch shape, I averaged temperature and light intensity and used these as dependent variables. Independent variables included fixed effects of distance to edge, patch shape, and a distance by shape interaction and a random effect of site. I then analyzed the data by patch shape, using average temperature and light as dependent variables and distance to edge as the fixed independent variable. I also analyzed data by distance to edge, using average temperature and light as dependent variables and patch shape as the fixed independent variable. Both of these analyses also used site as a random independent variable. Because data were recorded

at each of four corners within each patch, I used corner as a repeated measure in all of these analyses. I performed these analyses in SAS 9.1.3 using a mixed model (PROC MIXED).

Wind data were analyzed to determine average wind directions during experiment two. Wind direction angles were grouped into 20 degree increments and averages were determined using basic circular statistical functions in Oriana 3.0 (Kovach Computing Services, Anglesey, Wales).

RESULTS

In experiment one, patch shape had a slightly significant effect on the proportion of plants diseased ($F_{2,51}$=2.70, p=0.08). Rectangular patches had significantly higher proportion of diseased plants than winged patches (t=2.28, df=51, p=0.03). There were no significant differences in proportions of diseased plants between rectangular and connected patches (t=1.27, df=51, p>0.10) or winged and connected patches (t=0.82, df=51, p>0.10) (Figure 1.4a). Distance to edge had no significant effect on the presence of disease.

In experiment two, once environmental conditions besides connectivity were controlled, patch shape had no significant effect on where spores landed ($F_{2,22}$=1.36, p>0.10) (Figure 1.4b).

In experiment three, initial levels of disease differed with distance to edge in rectangular patches, but not winged patches. In rectangular patches, plants at 40 meters had significantly less percent of leaf area covered by lesions than plants at zero meters or plants at 20 meters (t=-2.08,-2.00, df=38, p=0.04, 0.05, respectively) (Figure 1.5a). There were no

significant differences in the initial percent of leaf area covered by lesions due to distance in winged patches ($F_{2,38}$=1.57, p>0.10) (Figure 1.5b). There were no significant differences in the final amount of disease between distances for either rectangular or winged patches ($F_{2,38}$=0.19, 1.57, p>0.10, respectively) (Figure 1.5). There were no significant distance by shape interactions and no significant effects of level of herbivory for either sampling period.

Disease development (measured as the changed in percent of leaf area covered by lesions between of the first and fourth sampling period) varied with host distance to edge and the magnitude of this effect was dependent on patch shape. For rectangular patches, the difference in the percent of leaf area with lesions increased with increased distance of the hosts from the patch edge. Plants 40 meters from the edge had significantly higher disease development than plants zero meters from the edge (t=2.02, df=38, p=0.05). However, there were no significant differences between plants at 20 meters and plants at either zero or 40 meters (t= -1.24, 0.80 df=38, p>0.10, respectively) (Figure 1.5a). For winged patches, disease development was greatest at the intermediate distance of 20 meters. Plants at this distance had significantly higher disease development than plants zero meters from the edge (t=-2.36, df=38, p=0.02). Disease development on plants 40 meters from the edge did not differ significantly from that of plants zero or 20 meters from the edge (t=0.87, -1.58, df=38, p>0.10, respectively) (Figure 1.5b). There was no significant distance by shape interaction in this analysis.

For each patch shape, temperature increased significantly with increased distance from patch edges ($F_{3,116}$>56.0, p<0.001 for all patch shapes) (Figure 1.6a). Temperatures

recorded farthest from the patch edge (42 meters) were significantly higher in winged patches than rectangular patches ($t=-2.45$, $df=86$, $p=0.02$) (Figure 1.6). There were no significant differences between temperatures in different patch shapes at any other distance measured. Light intensity followed the same pattern as temperature, with significant increases in intensity with increased distance from the patch edge ($F_{3,116}>24.1$, $p<0.001$ for all distance increases in all patch shapes) (Figure 1.6b). However, unlike temperature, there were no significant differences between any patch shapes at any individual distance from the patch edge. There was no significant distance by shape interaction in this analysis.

During experiment two, for six of the eight sites average wind directions were from the southwest (between 204° and 222°) and for the other two sites there was no predominent wind direction (wind directions did not differ significantly from random).

DISCUSSION

My results indicate that corridors did not facilitate the movement of wind dispersed plant pathogens; patches connected by corridors did not have higher proportions of diseased plants than unconnected patches. Patch shape or "edginess" and distance of host plants to patch edges did have significant effects on presence of disease and levels of disease development. These findings contradict the hypothesis that habitat corridors facilitate pathogen spread and increase levels of disease, and emphasize the importance of edge effects in regulating plant-pathogen interactions.

Experiment one showed that connectivity of patches does not increase levels of disease. The duration of this experiment allowed not only for primary dispersal from the source patch, but also subsequent dispersals and infections, making results representative of population level processes that occur throughout a growing season. At the end of the experiment, a higher proportion of plants in unconnected rectangular patches were diseased (Figure 1.4a), indicating that patch shape, not connectivity, regulates plant disease dynamics.

Experiments two and three were designed to independently test two key elements of plant disease dynamics: spore dispersal and disease development. Experiment two explicitly tested primary dispersal of fungal spores from a source patch, while prohibiting secondary dispersals and controlling factors that could affect disease development. Results from experiment two indicate no significant effects of patch shape or connectivity on spore dispersal (Figure 1.4b). Primary dispersal from the center patch takes spores to all patch types; however, there is high variation in the numbers of plants infected per patch. A similar experiment of longer duration would allow for subsequent dispersal both within and between patches, and may have shown an effect similar to that seen in experiment one.

Experiment three tested the effects of patch shape and distance to edge on disease development, while eliminating the variability of spore dispersal. Initial levels of disease differed only in rectangular patches, where plants farthest from the edge (40 meters) had significantly less disease than plants closer to the edge (zero and 20 meters). Distance to patch edge had a significant effect on levels of disease development in both rectangular and winged patches; however, the effect differed between the two patch shapes. In rectangular

patches, disease development increased with increased distance from the edge and was greatest on plants located 40 meters from the edge (Figure 1.5a). In winged patches, highest disease development occurred on plants 20 meters from the edge, with lower levels of development at zero and 40 meters from the edge (Figure 1.5b). Both these patch types have the same total habitat area, but differ in their edge-to-area ratio with rectangles having more "core" habitat area, further indicating that edge effects are driving disease development.

Results from experiment three provide an explanation for the effect of patch shape seen in experiment one. Experiment one plants were located at distances of 7 and 40 meters from the edge, and experiment three plants were located zero, 20, and 40 meters from the edge (Figure 1.1). In experiment three, plants at the farthest distance (40 meters) had a significant increase in disease development in rectangular patches, but lower levels of development in winged patches (Figure 1.5). Because plants in experiment one were located at the distance of elevated disease development in rectangular patches (40 meters), but not for winged patches (20 meters), experiment one favored higher disease development in rectangular patches. Although plants at 40 meters had lower initial levels of disease in rectangles, the significance of the increase in disease development between the first and fourth sampling period show that, though initially lower, disease development at this distance was favored over time.

Increased disease development due to the location of plants in experiment one may have allowed for higher levels of subsequent within-patch dispersal in rectangular patches. Plants from experiment one remained in the field for approximately 50 days, allowing for

multiple subsequent dispersal of southern corn leaf blight. While wind can carry fungal spores over large distances, shorter dispersal of a few meters are more common (Agrios 2005, Takan *et al*. 1994), making within-patch dispersal more frequent than longer-distance between-patch dispersal. In experiment one, once primary dispersal from the source patch resulted in infection of plants in rectangular patches, the location of these plants encouraged higher levels of disease development than in other patch types. Greater disease development provided more spores for subsequent dispersal, resulting in a higher proportion of infected plants in rectangular patches.

Temperature and light are key factors affecting disease development (Harvell *et al*. 2002, Huber and Gillespie 1992, Jarosz and Burdon 1988, Jarosz and Davelos 1995) and can be used to explain patterns of disease seen in my experiments. Understandably, temperature and light intensity follow similar patterns throughout our landscapes, where increased distance from the patch edge leads to increases in light intensity and average temperatures (Figure 1.6). While the highest temperatures for all patch types were recorded farthest from the edge, winged patches had significantly higher temperatures than rectangular patches at this farthest distance (Figure 1.6a).

These differences in light and temperatures have important consequences for disease development. In winged patches, disease development was lower at the farthest distance from the edge. Temperatures here may be above the optimal range for southern corn leaf blight development, while areas adjacent to the edge may be too cool or receive too little light for disease development, resulting in higher rates of disease development in plants at an

intermediate distance (20 meters) from the edge in winged patches. In rectangular patches, temperatures at the farthest distance (~40 meters) were lower than those in winged patches, and did not limit southern corn leaf blight development.

My findings contradict previous studies that show significantly higher levels of disease in shaded areas (Chapter 2; Jarosz and Burdon 1988). Jarosz and Burdon (1988) found that incidence and severity of a leaf scald disease in a naturally occurring plant population was higher in plants shaded by the tree canopy. In my study, disease levels in plants near habitat edges were lower than or equal to levels in plants farther from the edge (Figure 1.5). In natural plant communities host plants vary in density and susceptibility to disease (Burdon 1993, Burdon and Chilvers 1982, Jarosz and Burdon 1988), and plant diseases vary in virulence and host specificity (Agrios 1995), making the real world far more complex than my experiments. It is possible that higher levels of disease in shaded areas are artifacts of longer term community-level interactions and would be seen in established naturally occurring plant populations in our system. However, the contradiction between my findings and those of previous studies highlights the complexity of plant disease dynamics. My research provides experimental evidence that the key mechanisms driving plant-pathogen interactions in fragmented landscapes are due to habitat edges.

To the extent that the pattern I found holds in other plant-disease system, it suggests that forest preservation at the landscape scale may offer services to agriculture. Our openings were surrounded by a forested matrix, just as some agricultural fields are bordered by forest patches. My results suggest that forest preservation can actually create an

additional service by reducing disease incidence and severity. Thus, the reduction of plant disease can be added to other services maintained by forests in agricultural landscapes, such as increased biocontrol and pollinator services (Isaacs *et al*. 2009).

Our corridor system did not influence dispersal of southern corn leaf blight; however, while the spatial scale and level of replication in the corridor experiment is extensive, it may not be appropriate for studying the effects of connectivity on pathogens capable of long distance dispersal. Movement of southern corn leaf blight in a 1970 epidemic spread rapidly over two fronts separated by the Appalachian Mountains (Mundt *et al*. 2009), showing that the scale at which epidemics of fungal plant pathogens can occur is far larger than our corridor experiment.

Corridors may, however, impact dispersal of other plant pathogens. Many plant viruses depend on insect vectors for dispersal to new hosts, and the movements of these insects may be affected by corridors. Studies have shown the corridors affect the movement of insects pollinators such as butterflies (Haddad and Baum 1999, Haddad *et al*. 2003, Tewksbury *et al*. 2002) and disease levels of insect caused galls are higher in patches connected by corridors (Sullivan *et al*., in review). Yet, no experimental studies have examined the role of corridors in the spread of vectored plant diseases.

Using a model plant pathogen system, I demonstrated that corridors do not increase disease levels by facilitating pathogen dispersal, but that habitat fragmentation alters levels of fungal plant disease through edge effects. My findings confirm results of other research into the potentially harmful effects of corridors that have shown that negative effects of corridors

are almost uniformly caused by edge effects, and not by connectivity (Haddad *et al*. 2010). My results can be generalized to natural plant-pathogen systems, where fungal diseases represent 30% of emerging infectious diseases of plants (Anderson *et al*. 2004). The majority of these fungi rely on agents such as wind and water to be disseminated to new hosts (Agrios 2005), and would be impacted by fragmentation and corridors similarly to my study organisms. Because we know relatively much about edge effects, we can potentially mitigate their negative effects by reducing the amount of edge or by softening edges.

Human land use practices will cause continued fragmentation of natural habitat, and further research into how changes to the landscape impact plant disease dynamics is fundamental. Corridors do affect attributes of host plants, such as density and dispersal (Damschen *et al*. 2006, Fagan *et al*. 1999, Mitchell *et al*. 2002, Orrock *et al*. 2003, Rand *et al*. 2006, Ries *et al*. 2004) that will, over time, shape populations of pathogens. However, my study provides evidence that corridors do not facilitate the movement of wind dispersed fungal plant pathogens, and still show more promise than risk for conservation.

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APPENDIX

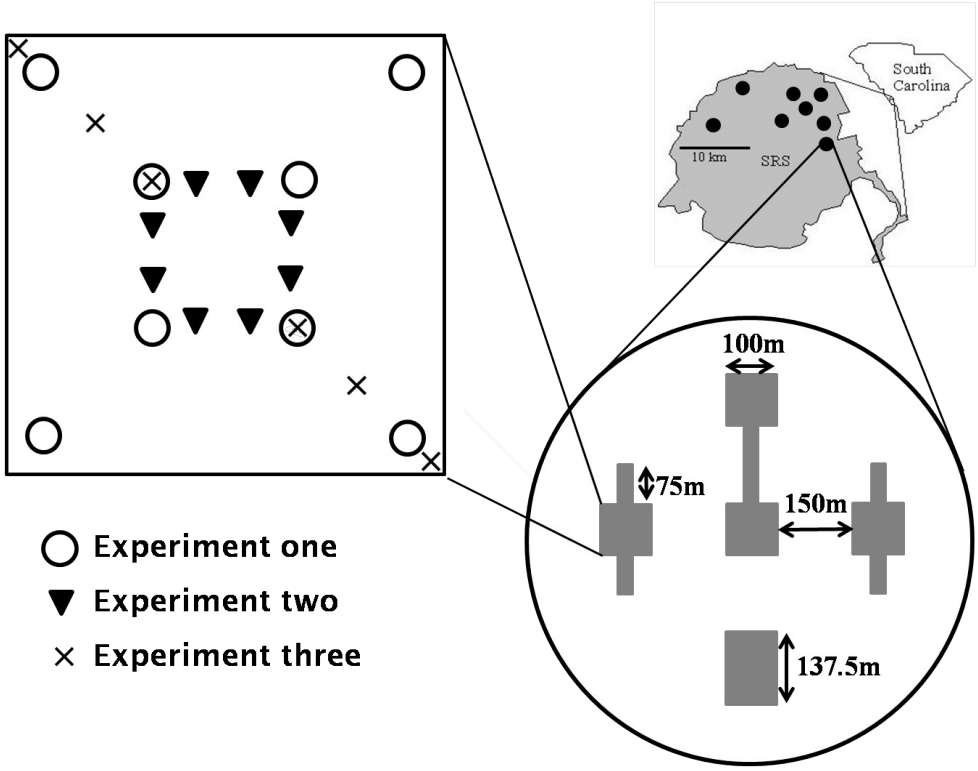

FIGURE 1.1: Location, layout, and dimensions of habitat corridor blocks, and locations of corn plants for each of three experiments. Each of eight blocks is comprised of five habitat patches with the center patch and one surrounding patch connected by a linear habitat corridor. The inset shows the placement of corn plants for each of the three experiments.

FIGURE 1.2: Aerial photograph of one of eight experimental blocks. Habitat patches are regenerating longleaf pine savannah cut into a pine plantation matrix. Habitat edges are distinct transitions between the savannah like patches and 25 meter tall pine plantation. Photo credit: Ellen Damschen.

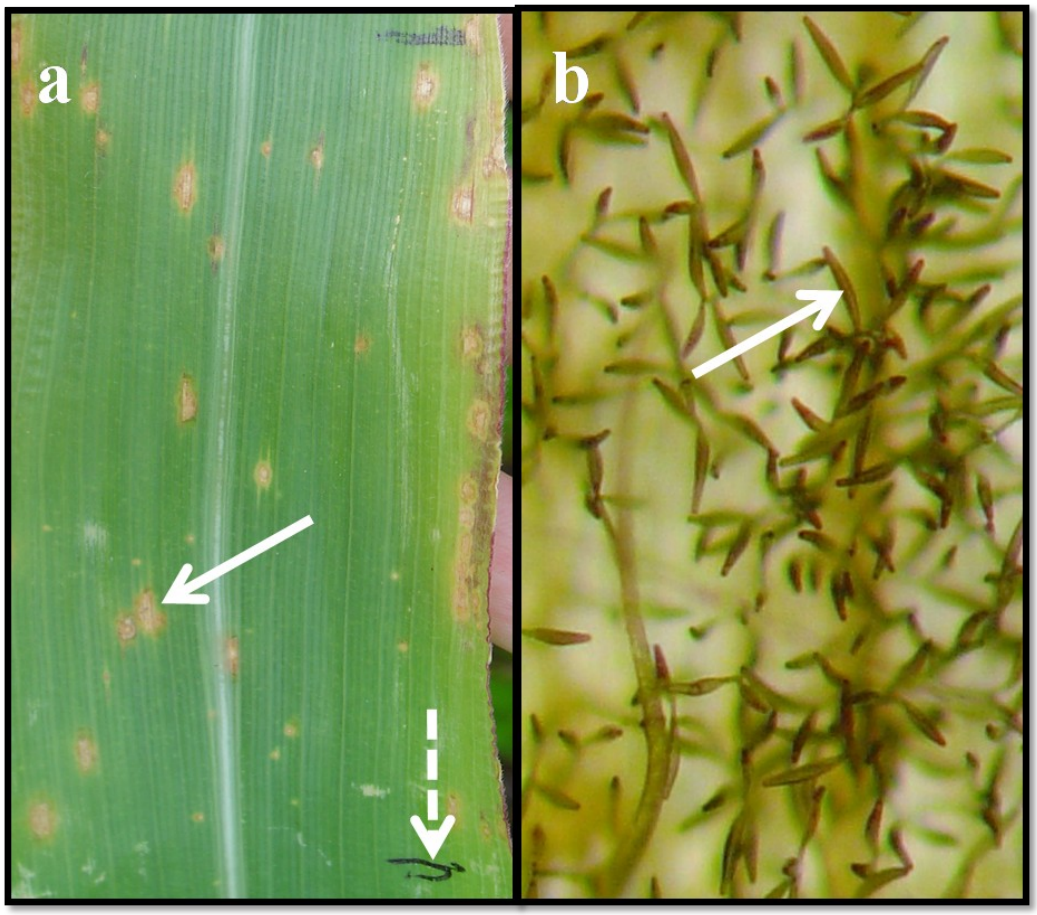

FIGURE 1.3: Lesions and spores of southern corn leaf blight (*Bipolaris maydis*). Southern corn leaf blight creates distinct lesions on the corn leaf (a, solid arrow). In experiment three, four inch sections were marked on the leaf surface to delineate areas in which to monitor disease development (a, dashed arrow). Spores of southern corn leaf blight are easily distinguished under microscopy (b).

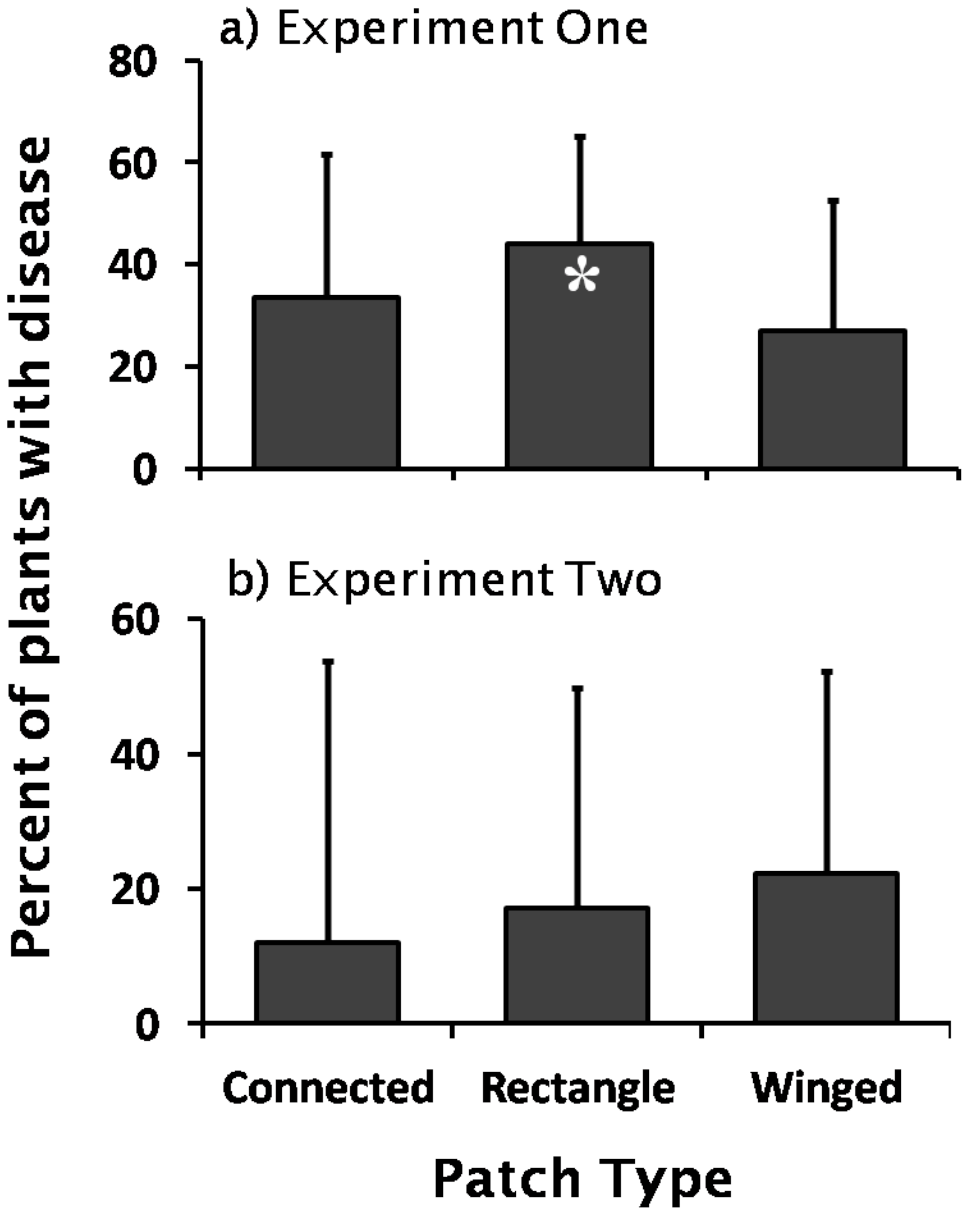

FIGURE 1.4: Effects of patch shape on the proportion of plants infected in experiments one and two. In experiment one, disease dispersal and development throughout an entire growing season resulted in a significantly higher proportion of diseased plants in rectangular patches than winged patches, but no significant differences between connected patches and either winged or rectangular patches (a). Tests of spore dispersal in experiment two resulted in no significant effects of connectivity or patch shape on where spores dispersed (b). Asterisk represents $p<0.05$ and error bars represent one standard error.

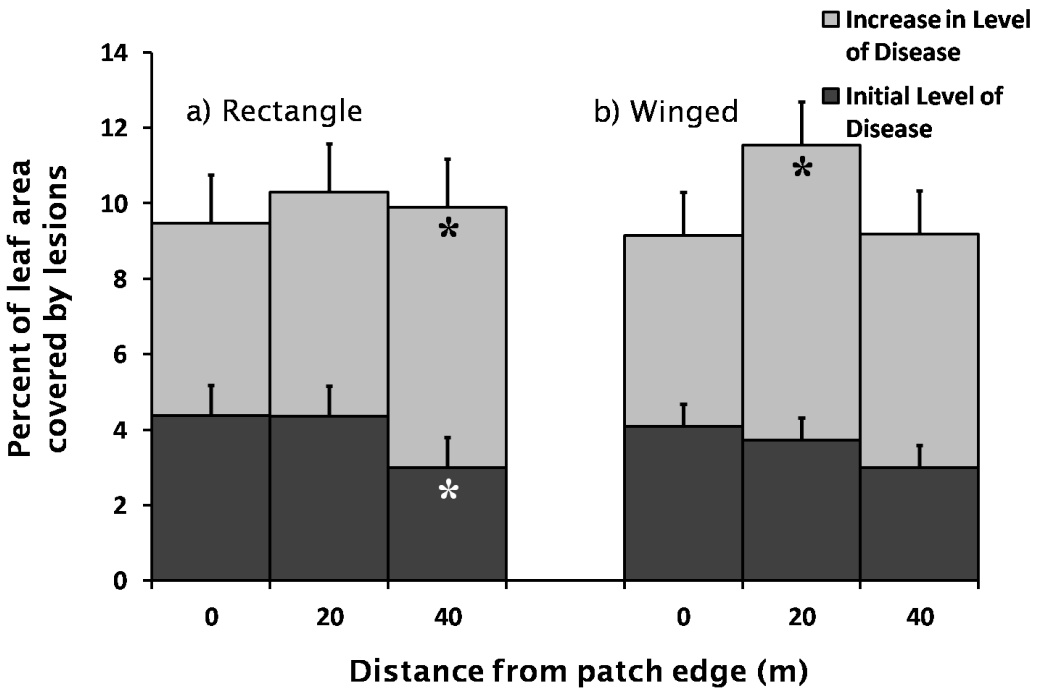

FIGURE 1.5: Effects of patch shape and distance to patch edge on disease development in experiment three. Initial levels of disease, measured as the percent of leaf area covered by disease lesions (dark bars), differed only in rectangular patches, where plants 40 meters from the edge had significantly lower initial levels of disease than plants zero or 20 meters from the edge. Lighter color bars indicated disease development over time. In rectangular patches, there was significantly higher disease development in plants 40 meters from the edge (a). For winged patches, there were no significant differences in initial levels of disease (dark bars), but plants 20 meters from the edge had significantly higher disease development than plants zero meters from the edge (light bars) (b). There were no significant differences in final disease levels between distances for either patch shape. Asterisks represents $p<0.05$ and error bars represent one standard error.

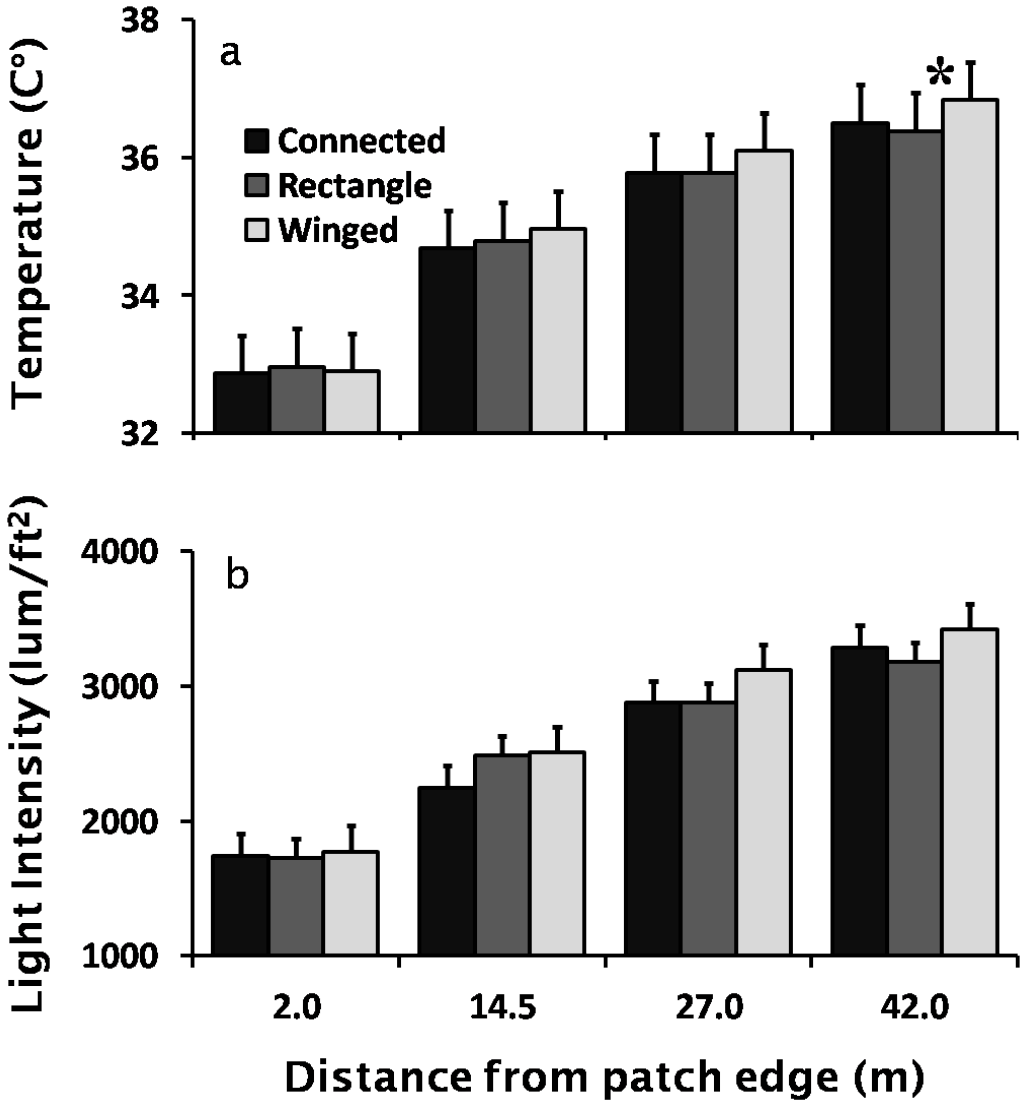

FIGURE 1.6: Effects of patch shape and distance to patch edge on average temperature and light intensity. Both temperature (a) and light intensity (b) increased with increased distance from patch edge. At the farthest distance (42 meters), temperature was significantly higher in winged patches than in rectangular patches. Asterisk represents $p<0.05$ and error bars represent one standard error.

CHAPTER 2:

HABITAT EDGES DETERMINE THE INCIDENCE OF FOLIAR FUNGI IN A NATURALLY OCCURRING PLANT POPULATION

ABSTRACT

In a study testing the effects of habitat connectivity and edge on the incidence of naturally occurring plant disease, I found that connectivity of habitat patches did not affect levels of disease and that disease incidence was significantly higher near patch edges. Habitat patches connected by corridors are theorized to have higher levels of disease due to increased movement of pathogens through corridors. Increases in the amount of habitat edge are inevitable byproducts of corridor creation, and may also impact the incidence and development of plant disease. However, the impacts of corridors and habitat edges on plant disease dynamics remain empirically untested. Working in a series of experimental landscapes designed to differentiate the effects of habitat connectivity, area, and edge, I surveyed foliar lesions on three native *Lespedeza* species. I found that incidence of wind dispersed foliar fungal diseases was significantly higher close to habitat edges, demonstrating that edge effects play an important role in plant disease dynamics. I also found that density of host plants was significantly higher farther from habitat edges, contradicting previous studies that relate higher host densities to increased disease load. Environmental variables also showed strong edge effects, with significantly higher temperatures and light intensities at the interior of habitat patches. My results indicate that managing disease levels in natural plant populations would best be accomplished by regulating amounts of habitat edge in fragmented landscapes.

INTRODUCTION

Loss of habitat is the leading threat to biodiversity (Wilcove *et al.* 1998) and the resulting fragmentation of remaining habitat has negative effects on populations of a variety of organisms (Allan *et al*. 2003, Gonzalez *et al*. 1998, Groppe *et al*. 2001, Rantalainen *et al*. 2006). Habitat fragmentation can lead to declines in species abundances (Gonzalez *et al.* 1998, Rantalainen *et al*. 2006), limit species distributions (Gonzalez *et al*. 1998), and increase rates of disease (Allan *et al*. 2003, Groppe *et al*. 2001). A commonly proposed method to counteract the negative effects of fragmentation is to connect fragments with habitat corridors – strips of habitat that connect otherwise isolated patches (Rosenberg *et al*. 1997).

Empirical and theoretical studies on both small and large scales indicate that connecting habitat fragments with corridors increases both population persistence (Gonzalez *et al*. 1998, Rantalainen *et al*. 2006) and species richness (Debinski and Holt 2000), slows rates of species loss in metapopulations (Gilbert *et al*. 1998), facilitates dispersal (McCallum and Dobson 2002), increases movement of a variety of taxa, including plants, small mammals, and butterflies (Aars and Ims 1999, Debinski and Holt 2000, Haddad *et al*. 2003) and promotes plant-animal interactions including plant pollination and seed dispersal (Tewksbury *et al*. 2002). However, the same processes that allow corridors to increase movement of species of conservation interest may also facilitate movement of their antagonists, including predators, competitors, and pathogens (Plantagenest *et al*. 2007, Simberloff and Cox 1987, Simberloff *et al*. 1992). In this study I examined the effects of

connectivity, habitat patch shape, and distance to habitat edge on the incidence of naturally occurring foliar disease on native plants.

Fragmentation of habitat and addition of habitat corridors not only alter habitat area, but also increase the amount of habitat edge (Rand *et al*. 2006, Ries *et al*. 2004). Edges can affect movement of organisms, differentially alter resource distribution, influence access to those resources, alter species interactions, and serve as unique types of habitat (Davies *et al*. 2001, Fagan *et al*. 1999, Ries *et al*. 2004). Edges also affect microclimate conditions by altering amounts of light and moisture (Jarosz and Burdon 1988, Tscharntke *et al*. 2002). These effects of edges can have both positive and negative consequences for populations, and understanding the effects of increased edge is integral to determining the overall impacts of habitat fragmentation and corridors (Orrock *et al*. 2003, Rand *et al*. 2006, Ries *et al*. 2004, Tscharntke *et al*. 2002, Weldon and Haddad 2005).

Habitat fragmentation, connectivity, and increases in habitat edge have the potential to alter the interactions between plant diseases and their hosts. Plant diseases have an enormous impact in regulating populations of both agricultural and natural plants (Anderson *et al*. 2004, Augspurger 1987, Hudson *et al*. 2006, Jarosz and Davelos 1995, Mitchell 2003, Mitchell and Power 2006). Habitat fragmentation is known to impact disease dynamics of plant populations (Groppe *et al*. 2001), and corridors can affect populations of host plants by increasing host plant species richness and facilitating both pollination and seed dispersal (Tewksbury *et al.* 2002, Damschen *et al.* 2006).

Infection and survival of most plant pathogens depend heavily on local environmental conditions (Agrios 1995, Alexander *et al*. 2007, Augspurger 1987, Jarosz and Burdon 1988, Jarosz and Davelos 1995, Laine and Hanski 2006, Mitchell and Power 2006) which may be altered at the edges of habitat fragments (Fagan *et al*. 1999, Jarosz and Burdon 1988, Tscharntke *et al*. 2002). Edges can impact plant disease dynamics by producing spillover effects between two habitat types, such as spillover of pathogens from natural to agricultural landscapes (Brudvig *et al*. 2009, Fagan *et al*. 1999, Rand *et al*. 2006). Edges allow access to reservoir species in other habitat types or may provide increased vectors due to spillover. Edges can also alter host populations by altering interactions like pollination or seed dispersal (Fagan *et al*. 1999, Tewksbury *et al*. 2002). Edges are known to alter abiotic conditions such as temperature, humidity, leaf wetness, and soil chemistry, which could affect growth of both host and pathogen (Alexander *et al*. 2007, Bradley *et al*. 2003, Fagan *et al*. 1999, Huber and Gillespie 1992, Jarosz and Burdon 1988).

Edges can also affect dispersal of disease propagules (Fagan *et al*. 1999, Plantegenest *et al*. 2007), which are transported either abiotically by wind or water, or biotically by insect vectors (Fagan *et al*. 1999, Jarosz and Burdon 1998). Recent studies of the effects of connectivity have shown that plant species with different modes of dispersal (abiotic vs. biotic) assemble differentially in connected and unconnected patches over time (Damschen *et al*. 2008). Since plant pathogens also have differing modes of dispersal, it is likely that connectivity would differentially influence the movement of disease propagules with different dispersal modes. Since habitat connectivity and edges influence both microclimate

and movement of propagules with differing dispersal mechanisms (Damschen *et al*. 2008, Fagan *et al*. 1999, Jarosz and Burdon 1988, Tscharntke *et al*. 2002), it is important to assess the impact of both edges and corridors on plant disease dynamics.

I examined the effects of connectivity and habitat edge on foliar diseases of naturally occurring plant populations. Foliar fungi are common plant pathogens, have widespread distributions, and are known to decrease plant fitness (Agrios 1995, Jarosz and Davelos 1995). I surveyed incidence of foliar lesions on three species of *Lespedeza*. I predicted that incidence and severity of foliar fungi would show strong edge effects because of differences in environmental conditions such as temperature, humidity, and mean leaf wetness time (Fagan *et al*. 1999, Huber and Gillespie 1992, Jarosz and Burdon 1988, Tscharntke *et al*. 2002).

I also assessed the relationships of host density to distance from edge and disease severity. Based on previous studies, I predicted that increases in host density will increase disease incidence (Burdon and Chilvers 1982, Knops *et al*. 1999, Mitchell *et al*. 2002, Plantegenest *et al*. 2007), and that distance to edge may impact host density through alterations in microclimate. Because environmental variables are important factors for disease development, I assessed temperature and light intensity as possible mechanisms behind observed disease patterns.

METHODS

Site Description

I conducted my study at the Savannah River Site, south of New Ellenton, SC, in a large scale replicated habitat corridor experiment comprised of eight ~ 50 ha experimental blocks, each consisting of five ~1 ha regenerating habitat patches located in a mixed pine matrix comprised mainly of loblolly pine (*Pinus taeda*) and longleaf pine (*P. palustris*). Patches are managed as early succession longleaf pine savannah with controlled burns applied on a two to three year rotation.

Patch arrangement consists of a central 100 x100m patch and four surrounding patches 150 meter away. The central patch and one peripheral patch are connected by a 25 meter wide corridor. The other three peripheral patches are unconnected and are of two types: rectangular or winged. Rectangular patches control for the increase in habitat area that is a byproduct of corridor addition and the same area as a corridor added onto a square habitat patch. Winged patches control for the increase in both habitat area and edge and have two 75 meter dead end "corridors" on either side of a square patch (Figure 2.1). This variety in connectivity and patch shapes allows me to differentiate between effect of connectivity, increased area, and increased edge (Tewksbury *et al*. 2002).

Study organisms:

Lespedeza hirta, L. stuevei, and *L. virginica* are perennial native legumes from the family Fabaceae. All three species co-occur throughout the southeastern United States (Miller and Miller 2005). I chose these three species because they are native to the longleaf

pine ecosystem, have widespread distributions within our study area, and are easy to identify in the field. In my study area, these species co-occur with other native and non-native *Lespedeza* congeners, including *L. repens* and *L. cuneata*. I excluded *L. repens* because it differs in morphological characteristics from my three focal species. I excluded *L. cuneata* because its distribution is not widespread in our patches (Miller and Miller 2005). *Lespedeza* species are commonly infected with a variety of foliar pathogens that cause easily distinguishable lesions on the leaf surface.

Data collection:

I conducted surveys on incidence and severity of foliar lesions on *Lespedeza* between June and August of 2007 and in August of 2008. For all three *Lespedeza* species, I collected data in one patch of each shape (connected, winged, and rectangular) in each of the eight replicate corridor sites. When duplicate patch shapes occurred, I randomly selected one patch of each shape. To determine the effects of edge on foliar lesion incidence, I selected plants along random transects in the outside 12.5 meter edge of each patch and the center 25x25 meter of each patch (Figure 2.1). Because my focal *Lespedeza* species are multistemmed, I considered stalks arising from a single point to be ramets of the same individual. Once an individual plant was chosen, I selected the tallest ramet on which to measure disease. On the selected ramet, I scored all leaves for presence or absence of foliar lesions. I sampled eight plants per distance of each *Lespedeza* species. I surveyed density of *Lespedeza* by counting the number of ramets from each individual surveyed as well as all

congener ramets (of the three *Lespedeza* species studied) in a two meter radius circle surrounding the focal ramet.

I identified three distinct types of lesions common to all three *Lespedeza* species and subsampled *Lespedeza* in September of 2009 to estimate the proportion of each lesion type. I surveyed four plants of each species at each distance (edge and center) in nine patches (three of each shape) at five of the eight replicate sites. I surveyed the proportions of each of the three types of lesions on randomly selected ramets of *Lespedeza*. I identified causes of foliar lesions in the North Carolina State University Department of Plant Pathology in September of 2009 by classifying fungal spores to genus and confirming known presence of disease on each species using Farr *et al.* (1989) as a reference.

Temperature and light intensity data were collected between 16 June and 14 July, 2008 using HOBO data loggers. Data loggers were placed at two different distances from the edges of all patches: approximately 2 meters and 42 meters along diagonal transects coming out from all four corners of the each patch (Figure 2.1). Readings of temperature and light intensity were collected every ten minutes for four days at each site.

Statistical Analyses:

I pooled data on *Lespedeza stuevei* and *L. virginica* (henceforth called *L. stuevei*) for all analyses. These two species are known to hybridize in the wild and could not be clearly differentiated in the field (Hanson and Cope 1955). I determined the effects of distance (edge or interior), patch shape, and density of congeners (number of congener ramets in a two meter radius circle surrounding the focal ramet) on the level of disease (number of leaves

showing lesions / total number of leaves per stalk). For each distance within each patch, I averaged the percent of leaves with lesions for each species and the density of congeners. For each species, dependent variables included the average percent of leaves with lesions. Independent variables included fixed effects of distance, patch shape, and congener density, and random effects of site. I analyzed data in SAS 9.1.3 using a mixed model (PROC MIXED).

I analyzed the environmental data to determine the effects of patch shape and distance to edge on average temperature and light intensity. I only used data collected between 7:00 AM and 7:00 PM when light and temperatures should be most affected by fragmentation. For each distance within each patch shape, I calculated average temperature and light intensity and used these as dependent variables. Independent variables included fixed effects of distance to edge and patch shape and the random effect of site. I performed separate analyses for each distance, using patch shape as the only fixed independent variable. Similarly, I performed separate analyses for each patch shape, using distance to edge as the only fixed independent variable. Because data were collected simultaneously in all four corners of each patch, I treated corner as a repeated measure in all analyses. I analyzed these data in SAS 9.1.3 using a mixed model (PROC MIXED).

RESULTS

For both *Lespedeza hirta* and *L. stuevei*, incidence of foliar fungal lesions was significantly higher at patch edges than patch interiors (*L. hirta*: $F_{1,64}=20.07$, $p<0.001$; *L.*

stuevei: $F_{1,76}$=6.99, p<0.01) (Figure 2.3). Connectivity of patches, patch shape, and density of host congeners did not significantly affect disease incidence. There were no significant shape by distance interactions for these analyses.

I quantified three main types of lesions on *Lespedeza*. I identified two wind dispersed foliar fungal pathogens: a rust disease of the genus *Uromyces* and a leaf spot disease of the genus *Cercospora* (Figure 2.2). Both genera are generalist pathogens on multiple *Lespedeza* species and disperse primarily by windblown spores (Farr *et al*. 1989, Sinclair 1982). Although these pathogens have not previously been identified in South Carolina, they have been confirmed as present on plants in surrounding states. I identified the third type of lesion as insect caused damage to the leaf tissue. On *L. hirta*, I found *Cercospora* leaf spots to be the most common type of lesion (60.7% of all leaves with lesions showed this type lesion), followed by *Uromyces* lesions (28.9%) and insect damage (24.5%). On *L. stuevei*, I found *Uromyces* to be the most common type of lesion (64.5%), followed by *Cercospora* leaf spots (30.3%), and insect damage (10.6%).

Distance to edge had a significant effect on the density of host congeners, with higher average density at the interior of patches than at patch edges ($F_{1,157}$ =42.72, p<0.001) (Figure 2.4). There was no significant effect of patch shape and no significant shape by distance interaction for this analysis. When I used both distance and density as predictors of disease severity, density of congeners had no significant effect on disease incidence for either species of *Lespedeza*.

Temperature was significantly higher at patch interiors for all patch shapes ($t=-26.34$, $df=116$, $p<0.001$). When I analyzed distances separately, I found that temperatures at patch interiors (42 meters from the edge) were significantly higher in winged patches than rectangular patches ($t=-2.45$, $df=86$, $p=0.02$) (Figure 2.5). Light intensity followed the same trend as temperature, with significantly higher amount of light in patch interiors ($t=-15.89$, $df=116$, $p<0.001$). However, I found no significant differences in light intensity between shapes at either of the two distances from the edge.

DISCUSSION

By surveying naturally occurring foliar fungal disease on native plant populations, I found that habitat fragmentation significantly impacts the incidence of plant disease through edge effects, and that connectivity of patches has no impact on disease levels. As predicted, incidence of foliar fungal pathogens was dependent on distance to habitat edge, with significantly higher levels of wind dispersed foliar diseases at patch edges than at patch centers. Host density did not affect disease levels, and relationships between host density and disease were inconsistent with those of previous studies. Distance to habitat edge also significantly affected environmental variables, such as temperature and light intensity, which are known to impact disease development.

Levels of fungal disease on *Lespedeza* species were significantly higher close to the edges of habitat patches. These edges receive less sunlight and are significantly cooler than patch centers. Increased shading and lower average temperature may provide optimal

conditions for disease development, leading to higher disease levels. Previous studies have shown that shading enhances disease development in natural plant populations (Jarosz and Burdon 1988). While environment is a key factor in disease development, host susceptibility and pathogen virulence also play important roles in determining disease levels. Empirical tests of proposed mechanisms behind enhanced disease levels are needed to ascertain the true cause of differences in disease levels.

I found no effect of connectivity on disease levels. This finding contradicts previous hypotheses that corridors facilitate the movement of disease propagules, leading to higher levels of disease in patches connected by corridors (Plantagenest *et al*. 2007, Simberloff and Cox 1987, Simberloff *et al*. 1992). Theoretical models that predict increased disease transmission by corridors are also not supported by my findings, although these models were based on diseases of large vertebrates (Hess 1994, Hess 1996). Generalist wind-dispersed pathogens may not need corridors to facilitate movement. Because populations of hosts are widespread throughout the landscapes, and source inocula are plentiful, disease populations are not limited by hosts or dispersal. Disease was present on all plants, and incidence only differed in the number of leaves infected, showing that development is probably dependent on environmental variables.

Although distance to edge did have a significant effect on host density, I found no effect of host density on disease severity. In my study, I found significantly higher host densities at patch interiors, where disease severity was lower. This effect contradicts the findings of previous research that relates higher host densities to increases in disease load

(Burdon and Chilvers 1982, Knops *et al*. 1999, Laine and Hanski 2006, Mitchell *et al*. 2002). Host density can have multiple impacts on disease severity. Higher host densities can increase amounts of disease by providing more host tissue for spores to land on or by decreasing the distances spores have to travel to reach a new host. High densities may also increase disease severity by making hosts more susceptible to infection through competition for nutrients (Burdon and Chilvers 1982). The inconsistencies between my findings and previous studies may indicate that environmental conditions are stronger determinants of plant disease severity.

While the majority of the lesions sampled were caused by fungi, a small percent were caused by insects. Because dispersal modes differed between insect caused lesions and wind-dispersed fungal lesions, it could be expected that the incidence of this lesion type would differ from those of fungi. However, the same edge effects were seen in all lesion types, with a higher incidence of lesions at habitat edges (results not shown). Effects of distance to edge on insect-plant interactions have been shown in other systems (Murcia 1995), and environmental variables may have impacted insect behavior. However, plant disease lowers plant fitness, and may make host plants more susceptible to herbivores (Hudson *et al*. 2006, Jarosz and Davelos 1995). It is possible that higher incidence of foliar fungi on plant near patch edges led to the higher levels of insect caused lesions because heavily diseased host plants were less able to defend themselves against insect antagonists.

The patterns observed in this study represent long-term community level processes that occurred over several generations. Habitat edges can affect plant-pathogen dynamics by

altering the host, the pathogen, the environment, or the interactions between them. In my study, I only assessed a single growing season of the plant community, and I can only speculate on the mechanisms behind observed patterns. Higher disease at patch edges may simply be a result of better environment provided by shading from matrix trees. Higher disease levels may also have resulted from hosts at patch edges more susceptible to disease. Correspondingly, lower host densities at patch edges may be a result of higher disease levels decreasing host populations. Such top-down effects of plant diseases on their hosts are not well studied in natural plant populations, and further research is needed to better advise management decisions (Mitchell and Power 2006).

Regardless of the causes of disease patterns, my study has shown that habitat edges increase levels of disease. This is but one of many negative effects of habitat edges. Previous studies in our system have shown that edges decrease fledging success in birds (Weldon and Haddad 2005), alter levels of seed predation (Orrock *et al*. 2003), and affect butterfly densities (Haddad and Baum 1999, Haddad and Tewksbury 2005). Edges most likely alter disease levels by altering some other key factor of disease development, such as environmental variables or host susceptibility, but empirical research is needed to identify the mechanisms behind the observed disease patterns.

To the best of my knowledge, this is the first study to specifically examine the effects of connectivity and habitat edges on naturally occurring plant disease. I have shown that the impact of fragmentation and corridors on plant disease is complex and is most likely dependent on environmental conditions mediated by edge effects. My findings demonstrate

that while corridors have been predicted to increase disease levels by facilitating the movement of pathogens, habitat edges are of greater importance when determining the impact of connectivity and fragmentation on plant diseases. More specifically, I've found that host plants near habitat edges have higher disease loads, which may be negatively impacting host densities. Managers concerned with plant conservation should be mindful that habitat edges may enhance disease levels through altering environmental conditions, and choose to mitigate edge effects in fragmented landscapes.

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APPENDIX

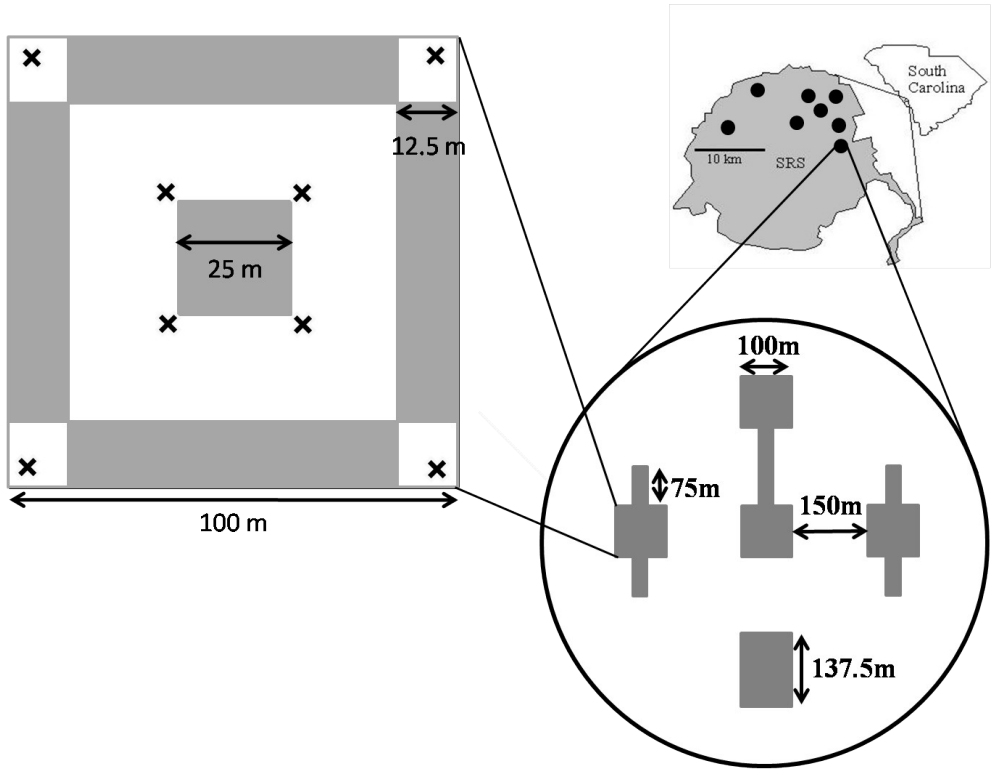

FIGURE 2.1: Location, layout, and dimensions of habitat corridor blocks and locations sampled within each patch. Each of eight replicate blocks is comprised of five habitat patches with the center patch and one surrounding patch connected by a linear habitat corridor. Unconnected patches are shaped to control for the increased habitat area and edge caused by corridor addition. Inset shows the locations sampled within each patch. I collected data on *Lespedeza* species located at within 12.5 meters of the edge and within the 25x25 meter center of each habitat patch, denoted by shaded areas. I did not sample in the 12.5x12.5 meter corners to reduce sampling biases caused by the effects of proximity to multiple edges. Temperature and light intensity data were collected in eight locations within each patch (denoted by **x**).

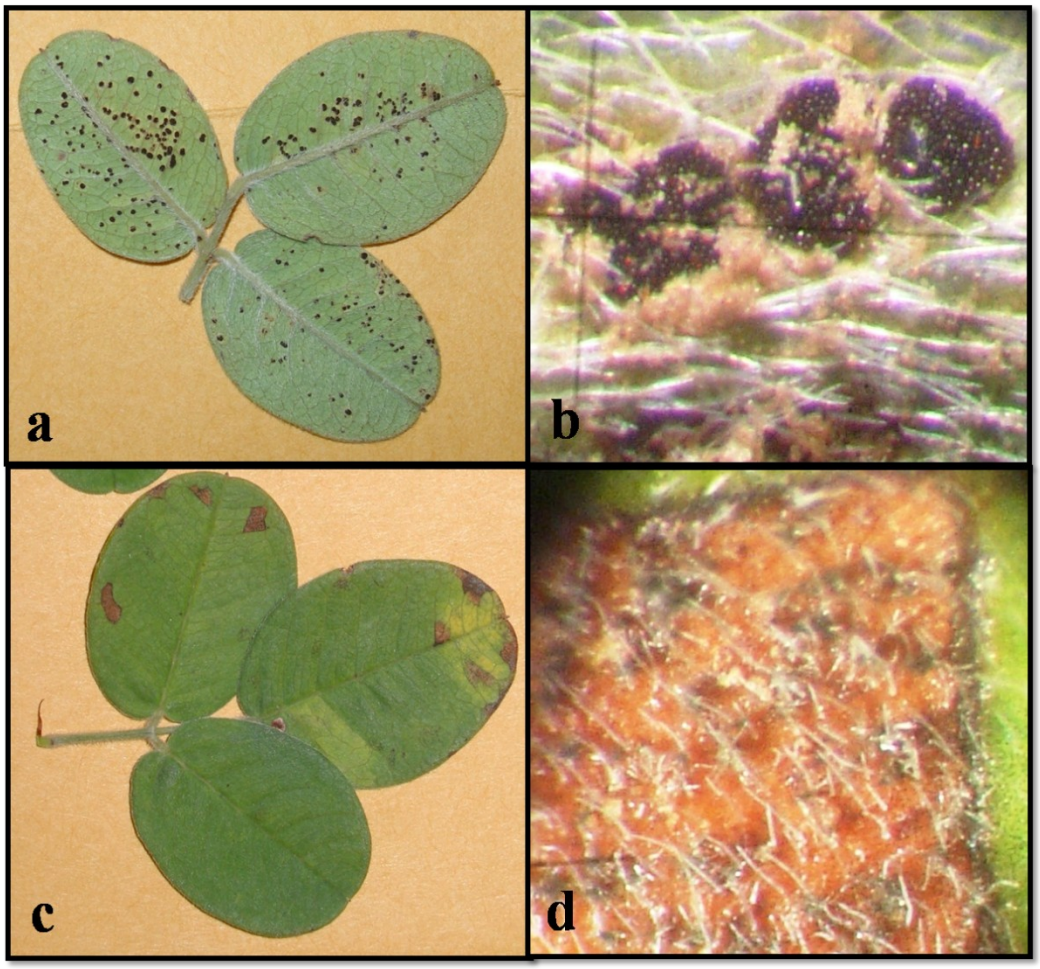

FIGURE 2.2: Lesions and spores of *Uromyces* species and *Cercospora* species found on *Lespedeza.* The two most abundant lesions on *Lespedeza* are caused by wind-dispersed foliar fungal pathogens. A) Dark spots on the underside of *L. hirta* show clumps of *Uromyces* spores. B) Uredospores (black) and teliospores (white) of *Uromyces* under microscopy. Uredospores are dispersed and infect new hosts while teliospores are produced at the end of the growing season and serve as the overwintering stage of the pathogen. C) *Cercospora* causes necrotic lesions between leaf veins on *L. hirta*. D) Dark spots within the *Cercospora* lesions are conidiophores from which spore-bearing conidia will emerge.

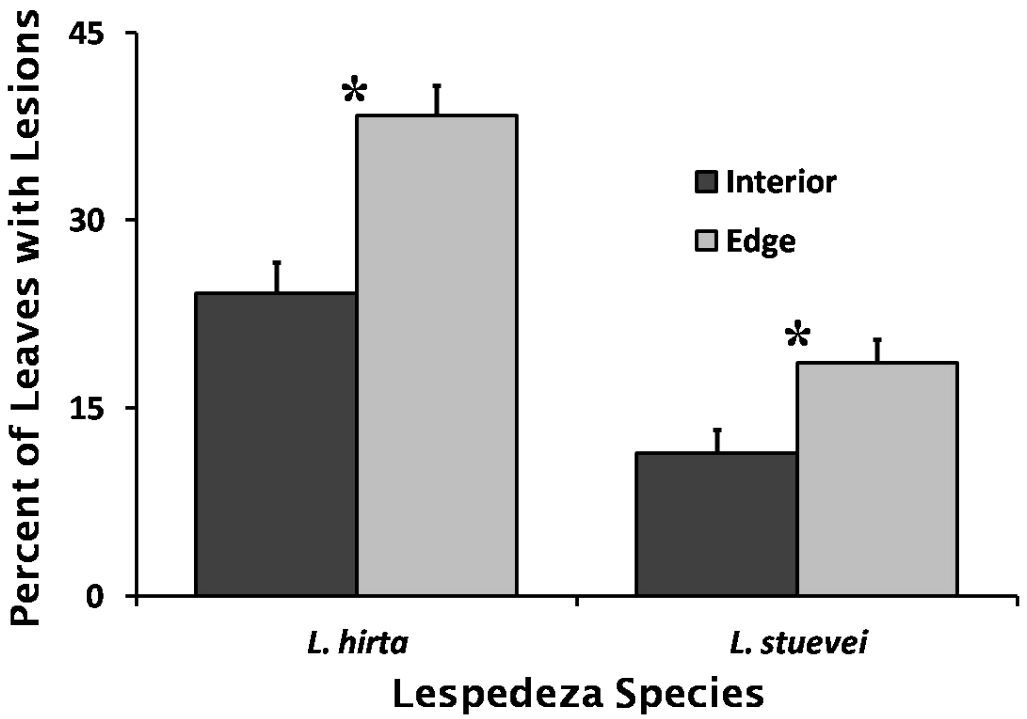

FIGURE 2.3: Effects of distance of host plants to patch edge on the incidence of foliar lesions for two species of *Lespedeza.* Distance from patch edge had a significant effect on disease incidence for both *L. hirta* and *L. stuevei*, with higher levels of disease found at patch edges than patch interiors. Asterisks represent $p<0.01$ and error bars represent one standard error.

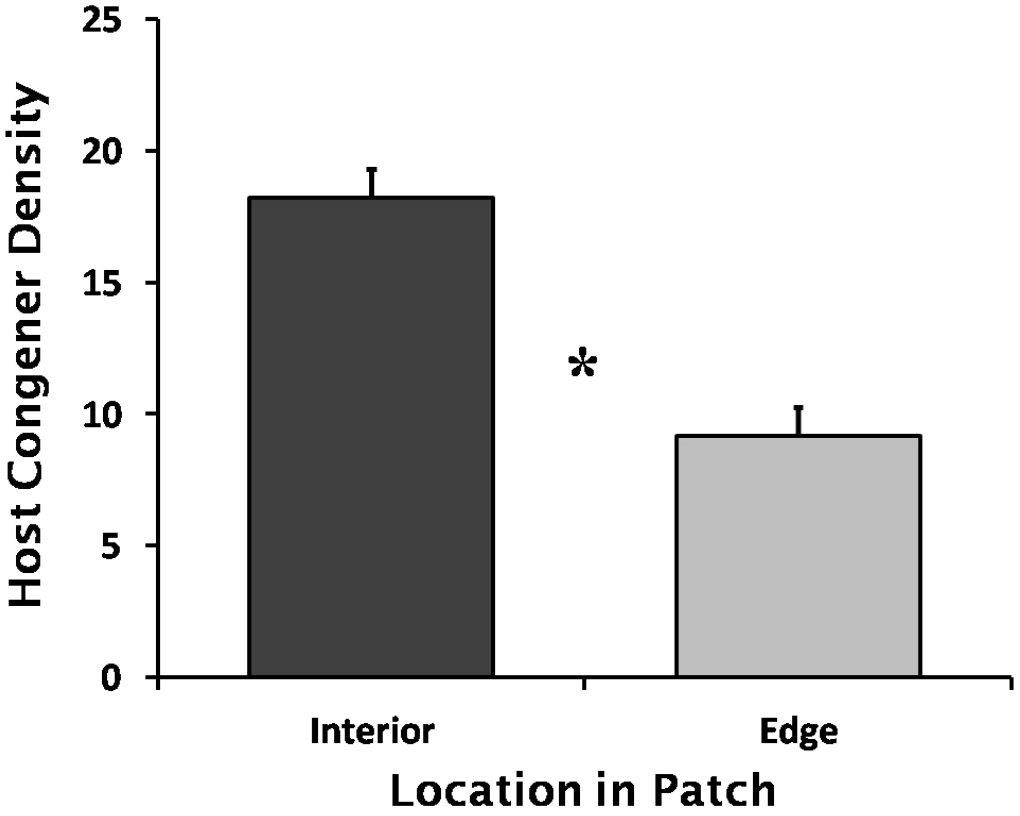

FIGURE 2.4: Effects of distance to patch edge on the density of *Lespedeza* congeners. There is a significantly higher average density of *Lespedeza* congeners in the interior of patches than at patch edges. Asterisk represents $p<0.001$ and error bars represent one standard error.

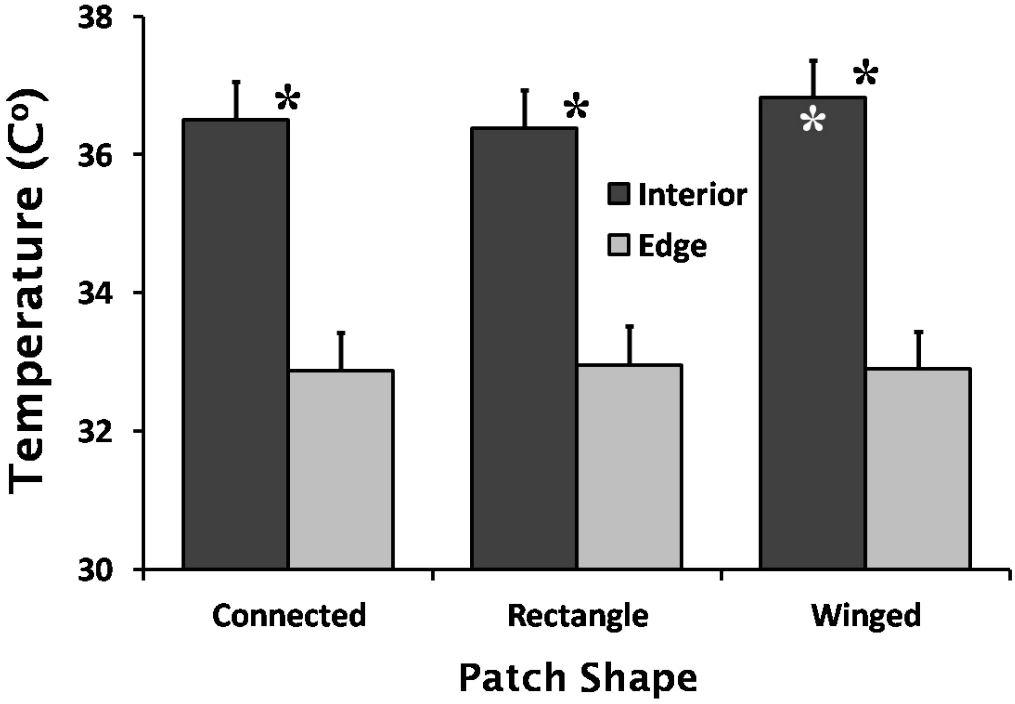

FIGURE 2.5: Effects of patch shape and distance to patch edge on average temperature. Average daytime temperatures were significantly higher at patch interiors in all patch shapes (black asterisks, $p<0.001$). At patch interiors, winged patches have significantly higher temperatures than rectangular patches (white asterisk, $p<0.05$). Error bars represent one standard error.