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Edge effects, not connectivity, determine the incidence and development of a foliar fungal plant disease

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Abstract. Using a model plant–pathogen system in a large-scale habitat corridor experiment, we 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 used in conservation, but the impacts of corridors on pathogen movement have never been tested empirically. Using sweet corn (*Zea mays*) and southern corn leaf blight (*Cochliobolus heterostrophus*) as a model plant–pathogen system, we tested the impacts of connectivity and habitat fragmentation on pathogen movement and disease development at the Savannah River Site, South Carolina, USA. 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. Variation in average daytime temperatures provided a possible mechanism for these disease patterns. Our results show that worries over the potentially harmful effects of conservation corridors on disease dynamics are misplaced, and that, in a conservation context, many diseases can be better managed by mitigating edge effects.

Key words: Cochliobolus heterostrophus; *connectivity; conservation; corridors; edge effects; habitat fragmentation; pathogen dispersal; plant disease;* Zea mays.

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, limiting species distributions, and increasing incidence of disease (Gonzalez et al. 1998, 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 (Rosenberg et al. 1997). Empirical studies have shown that habitat corridors do benefit populations by facilitating movement of a variety of organisms (Haddad et al. 2003, Gilbert-Norton et al. 2010). 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 (Simberloff and Cox 1987, Plantegenest et al. 2007).

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Although there are no empirical studies of the effects of corridors on plant disease, more general studies of connectivity and disease, as well as theoretical corridor models, offer some insight. Studies that characterize connectivity by the physical distance between individuals or groups of plants have found, both at small scales and across large landscapes, that greater connectivity increases the severity and incidence of disease (e.g., Thrall et al. 2003, Laine and Hanski 2006). Although these studies lead to inferences about conservation impacts of connectivity, there have been no empirical studies in which structural habitat connectivity has been manipulated to test for corridor effects. While there have been many modeling studies of corridors (Hess 1994, Gog et al. 2002, McCallum and Dobson 2002), results of such theoretical studies are often dependent on specific sets of conditions, and their relationships to real-world scenarios are not well defined, highlighting the need for experimental research.

The ambiguity found across different theoretical studies and the 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, we empirically tested the effects of corridors on the movement of wind-dispersed plant pathogens. We also assessed the effects of habitat patch shape and habitat edge on the incidence and severity of plant disease.

Habitat corridors can alter the interactions between plant diseases and their hosts by affecting patterns of pathogen dispersal (Sullivan et al. 2011). Although patterns of disease incidence ultimately depend on a variety of factors, including host susceptibility and environmental conditions, dispersal of disease propagules determines disease spread (McCartney et al. 2006). Studies of wind-dispersed pathogens in host metapopulations have found that better connected (spatially closer) host populations have higher incidence of disease, although microclimate was also an important component of disease dynamics (Thrall et al. 2003, Laine and Hanski 2006). For wind-dispersed pathogens, landscape features like corridors have the potential to alter wind patterns and affect the movement of plant pathogens (Plantegenest et al. 2007).

Fragmentation of habitat and addition of habitat corridors can also affect populations by increasing the amount of habitat edge (Ries et al. 2004, Rand et al. 2006). 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 (Augspurger 1987, Jarosz and Burdon 1988, Agrios 2005, Mitchell and Power 2006). Because 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, landscape corridor experiment, we studied the effects of 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. We conducted three experiments 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. We predicted that host plants located in connected patches would show higher rates of infection than plants in unconnected patches. We also 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 (Jarosz and Burdon 1988, Fagan et al. 1999).

Methods

Site description

We conducted our study in a large-scale replicated habitat corridor experiment located at the Savannah River Site, a National Environmental Research Park south of New Ellenton, South Carolina, USA. This model system has been shown through a meta-analysis to yield results that are qualitatively similar to those obtained across many larger, observational studies (Gilbert-Norton et al. 2010). The corridor experiment began in 2000 and consists of eight 50-ha experimental landscapes. Each replicate landscape (block) is composed of five ~1-ha regenerating habitat patches located in mature loblolly (*Pinus taeda*) and longleaf (*P. palustris*) pine plantation matrix. Within each of the eight blocks, patches are arranged with a central 100 × 100 m source patch and four peripheral patches 150 m away. The central patch is connected to one peripheral patch by a 25 m wide corridor (Fig. 1). The design of this experiment includes controls for patch shape that allow us to test whether movement between patches is facilitated by the connectivity provided by a structural habitat corridor (Appendix). Importantly for the interpretation of this study, isolated winged patches with corridors that do not connect to other patches have 50% more edge relative to their area than equal-sized but more compact rectangular patches (Fig. 1).

Study organisms

Within these landscapes, we used the model plant–pathogen system sweet corn (*Zea mays*) and southern corn leaf blight (*Cochliobolus heterostrophus* anamorph *Bipolaris maydis*) to conduct our 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. 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 us 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 for rapid dispersal and infection during our experiments. These spores are distinct from the majority of other fungal spores and are easily identified (see Plate 1; White 1999). Importantly, neither corn nor southern corn leaf blight were found in or near (conservatively, within 8 km) our experimental landscapes.

Data collection

Experiment one.—To test the effect of corridors on the movement of southern corn leaf blight, we placed plants in all five patches in all eight blocks. We then provided a source of spores by inoculating only plants in the center

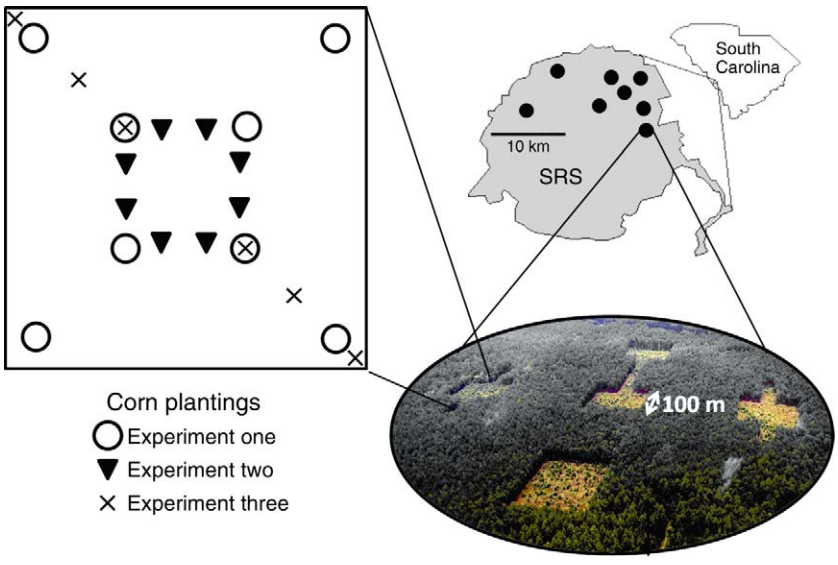

FIG. 1. Experiment at the Savannah River Site, South Carolina, USA. Each of eight blocks (black circles in the SRS map) is composed of five habitat patches with a 100 × 100 m center patch and one surrounding patch connected by a linear habitat corridor. The other three surrounding habitat patches are of two types: rectangular patches control for the increase in habitat area that is a by-product of corridor addition and have the area of a corridor added onto a square habitat patch, and winged patches control for the increase in both habitat area and edge and have two 75-m dead-end "corridors" on either side of a square patch. Habitat patches are regenerating longleaf pine (*Pinus palustris*) savannah cut into a pine plantation matrix. Habitat edges are distinct transitions between the savannah-like patches and 25 m tall pine plantation. The inset shows the placement of corn (*Zea mays*) plants for each of the three experiments (with each symbol representing an individual corn plant). Photo credit: Ellen Damschen.

patches 4, 11, and 37 days after placing them into the field. A detailed explanation of how plants and diseases were propagated can be found in the Appendix.

We collected data on presence or absence of disease lesions during four separate sampling periods: 14–19, 26–27, 33–36, and 47–49 days after placing plants into the field. We classified disease as present or absent on each of the top five leaves of each plant. We photographed lesions during the first, third, and fourth data collection periods. We 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.

We analyzed tissue samples by placing them in a moist chamber under a fluorescent light to induce sporulation. We determined positive samples by identifying the spores of southern corn leaf blight using a dissecting microscope. We also determined presence or absence of disease using the photographs of lesions taken in the fourth sampling period. Using tissue samples and photographs of lesions, we scored each plant as positive, negative, or indeterminate for disease. Based on the results of experiment one, we conducted two separate experiments the following year.

Experiment two.—We designed experiment two to examine the effects of corridors on the movement of southern corn leaf blight spores, while removing the confounding factors of differing environmental conditions and within-patch spore dispersal on disease development. Like experiment one, we placed corn plants in all five patches within all eight blocks, inoculating only plants in center patches. Unlike experiment one, we placed all plants at the same distance from habitat edges (Fig. 1; Appendix). We 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 dispersal could occur.

We collected peripheral patch plants six days after placing them into the field. We 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, we collected tissue samples from the plants by clipping lesions from leaf tissue and placing them into individual petri dishes. Tissue samples were incubated and analyzed as in experiment one.

Experiment three.—We designed experiment three to assess the effects of patch shape and distance to edge on disease development. Unlike experiments one and two, we did not test spore dispersal but instead inoculated all plants equally to determine differences in disease development (Fig. 1; Appendix).

Six days after inoculation, we marked 10-cm sections on each of three leaves per plant (see Plate 1). We photographed the marked sections six, 10, 14, and 18 days after inoculation. We analyzed the photographs to

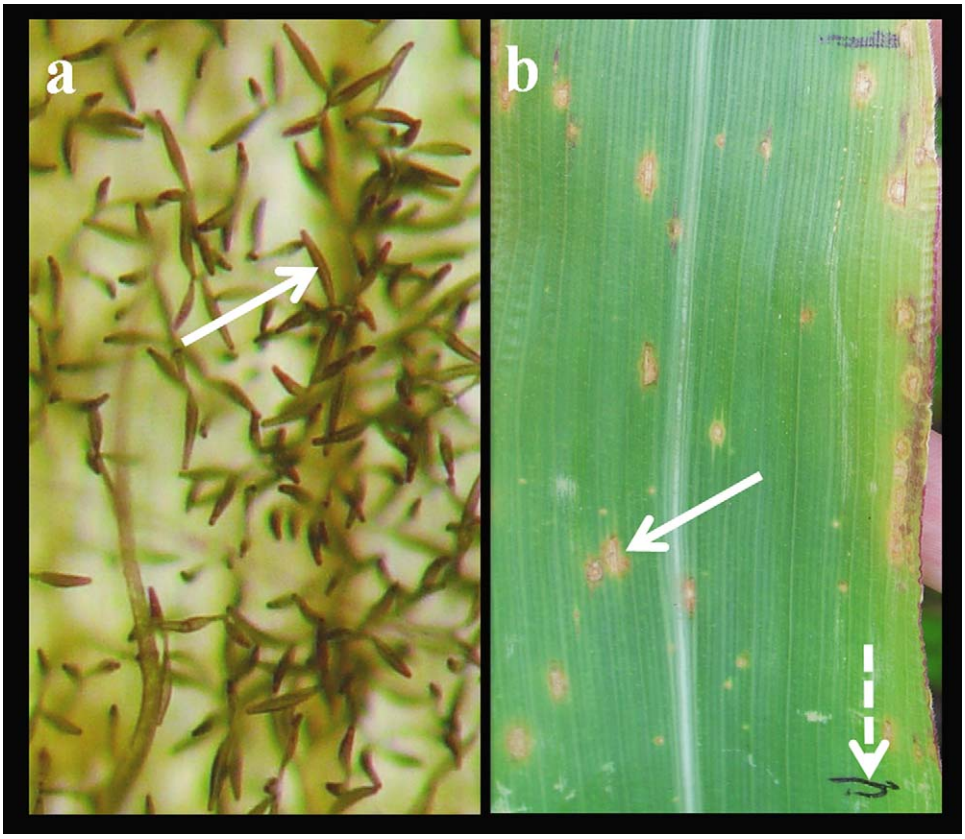

PLATE 1. Spores and lesions of southern corn leaf blight (*Bipolaris maydis*). (a) Spores of southern corn leaf blight are easily distinguished under microscopy. (b) Southern corn leaf blight creates distinct lesions on the corn leaf (solid arrow). In experiment three, four-inch sections (1 inch = 2.54 cm) were marked on the leaf surface to delineate areas in which to monitor disease development (dashed arrow). Photo credit: Brenda Johnson.

determine the percentage of marked leaf area covered with lesions and the percentage of leaf area damaged by herbivores for all leaves. We tested the effects of patch shape and distance to edge on the percentage of marked leaf area covered with lesions over time, and included levels of herbivory as a covariate.

Temperature data

To interpret the influence of microclimate among patches and across edge distances for disease patterns, we collected temperature data using HOBO dataloggers (Onset Computer, Pocasset, Massachusetts, USA) between 16 June and 14 July 2008. Dataloggers were placed in 16 locations in each patch: ~2, 14.5, 27, and 51 m along diagonal transects coming from the corners of the patches. Data were recorded in all five patches at each site every 10 min for four days per site. For analyses, only data recorded between 07:00 and 19:00 hours were used, as nighttime temperature was expected to be nearly uniform across edge distances.

Statistical analyses

In experiment one, we analyzed the effects of patch shape and distance to edge on the presence of southern corn leaf blight. For each distance within each patch, we 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 to edge and patch shape and a random effect of site. We analyzed data in SAS 9.1.3 using a generalized linear mixed model (PROC GLIMMIX; SAS Institute, Cary, North Carolina, USA), specifying a binomial distribution and logistic link for the data. We analyzed experiment two similarly to experiment one, omitting the variable of distance to edge in all analyses.

We 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. We 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, we averaged the percentages of leaf area covered by lesions and herbivory. Our dependent variable was percentage covered by lesions,

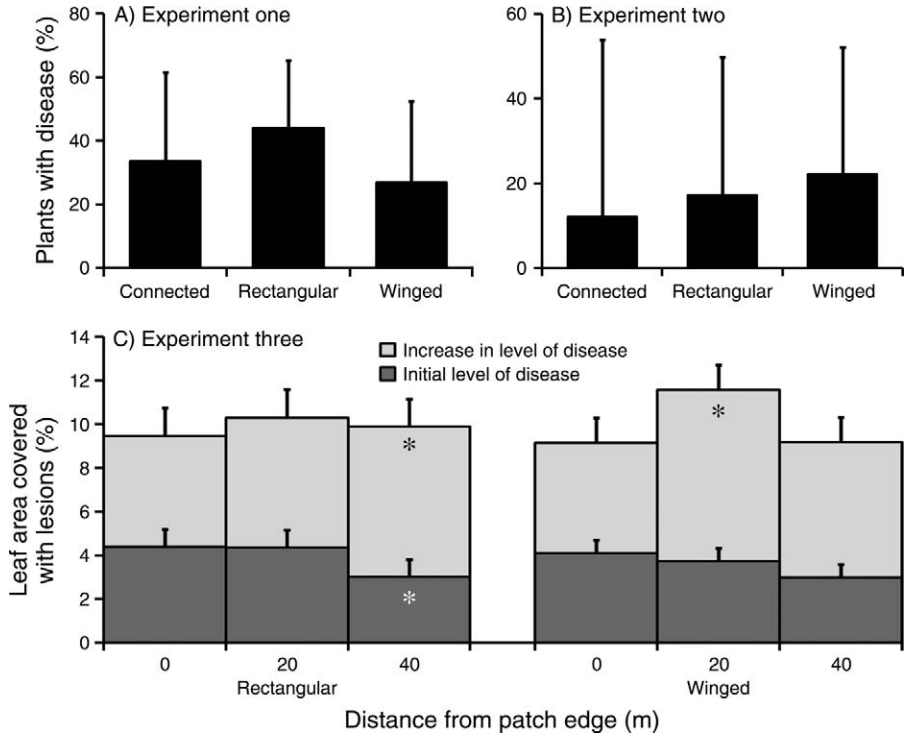

FIG. 2. (a) In experiment one, disease dispersal and development throughout an entire growing season resulted in a significantly higher percentage (mean + SE) of diseased plants in rectangular than in winged patches, but no significant differences between connected patches and either winged or rectangular patches. (b) In experiment two, we found no significant effect of connectivity or patch shape on where spores dispersed (mean + SE). (c) In experiment three, levels of disease, measured as the percentage of leaf area covered by disease lesions (mean + SE), differed at initial levels (dark fill) only in rectangular patches (left-hand columns), where plants 40 m from the edge had significantly lower initial levels of disease than plants 0 or 20 m from the edge; for winged patches (right-hand columns), there were no significant differences in initial levels of disease. There was significantly higher disease development (light fill) in rectangular plots for plants 40 m from the edge, and in winged plots for plants 20 m from the edge vs. 0 m from the edge.
* $P < 0.05$.

and our independent variables included fixed effects of distance to edge and patch shape. We included percentage herbivory as a covariate and site as a random independent variable.

To assess disease development, we determined the increase in percentage leaf area covered by lesions between the first and fourth sampling periods and used this as a dependent variable. We then averaged this increase for each distance within each patch, and we analyzed this response in the analysis described previously. We performed all analyses for experiment three in SAS 9.1.3 (SAS Institute, Cary, North Carolina, USA) using a mixed model (PROC MIXED).

We analyzed the effects of patch shape and distance to edge on temperature. For each distance within each patch shape, we averaged temperature and used this as the dependent variable. Independent variables included fixed effects of distance to edge, patch shape, and a distance by shape interaction, and a random effect of site. Because data were recorded at each of four corners within each patch, we used corner as a repeated measure in all of these analyses. We performed these analyses in SAS 9.1.3 using a mixed model (PROC MIXED).

RESULTS

In experiment one, patch shape had a marginally significant effect on the proportion of plants diseased ($F_{2,30} = 3.04$, $P = 0.06$). Rectangular patches had a significantly higher proportion of diseased plants than winged patches (Fig. 2a; Tukey's comparison of means, $t = 2.38$, df = 30, $P = 0.02$). There were no significant differences in proportions of diseased plants between rectangular and connected patches ($t = 1.43$, df = 30, $P = 0.16$) or winged and connected patches ($t = 0.82$, df = 30, $P = 0.42$). 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 (Fig. 2b; $F_{2,22} = 1.36$, $P = 0.28$) .

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 m

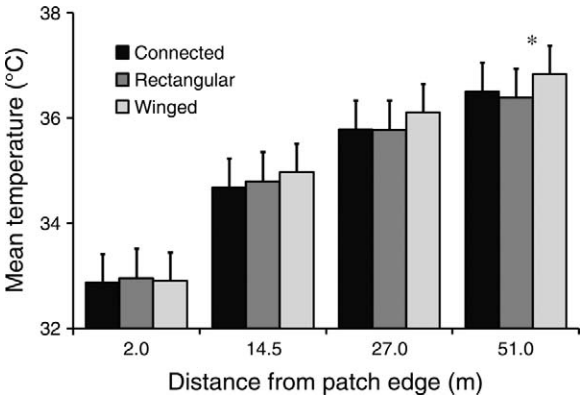

FIG. 3. Temperature (mean + SE, from readings taken between 07:00 and 19:00 hours) increased with increased distance from patch edge. At the farthest distance (51 m), temperature was significantly higher in winged patches than in rectangular patches.
* $P < 0.05$.

had significantly less percentage leaf area covered by lesions than plants at 0 m or plants at 20 m, representing a 45% proportional decrease in leaf area covered between plants nearer to and far from the edge (Fig. 2c; $t = -2.08, -2.00$, df = 38, $P = 0.04, 0.05$, respectively). There were no significant differences in the initial percentage of leaf area covered by lesions due to distance in winged patches (Fig. 2c; $F_{2,38} = 1.57$, $P = 0.22$). There were no significant differences in the final amount of disease between distances for either rectangular or winged patches (Fig. 2c; $F_{2,38} = 0.19, 1.57$, $P = 0.83, 0.22$, respectively). 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 change in percentage of leaf area covered by lesions between the first and fourth sampling period) varied with host distance to edge and this effect was different within different patch shapes. For rectangular patches, the difference in the percentage of leaf area with lesions increased with increased distance of the hosts from the patch edge. Plants 40 m from the edge had significantly higher disease development than plants 0 m from the edge, representing a 35% increase between plants near to and far from the edge ($t = 2.02$, df = 38, $P = 0.05$). However, there were no significant differences between plants at 20 m and plants at either 0 or 40 m (Fig. 2c; $t = -1.24, 0.80$ df = 38, $P = 0.22, 0.43$, respectively). For winged patches, disease development was greatest at the intermediate distance of 20 m. Plants at this distance had significantly higher disease development than plants 0 m from the edge, representing a 55% increase between plants near to and at intermediate distance from the edge ($t = -2.36$, df = 38, $P = 0.02$). Disease development on plants 40 m from the edge did not differ significantly from that of plants 0 or 20 m from the edge (Fig. 2c; $t = 0.87, -1.58$, df = 38, $P = 0.39, 0.12$, respectively). There was no significant distance by shape interaction.

For each patch type, temperature increased significantly with increased distance from patch edges (Fig. 3; $F_{3,116} > 56.0$, $P < 0.001$ for all patch shapes). Temperatures recorded farthest from the patch edge (51 m) were significantly higher in winged patches than rectangular patches (Fig. 3; $t = -2.45$, df = 86, $P = 0.02$). There were no significant differences between temperatures in different patch shapes at any other distance measured. There was no significant distance by shape interaction.

DISCUSSION

Our 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 of wind-dispersed fungal plant pathogens.

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 (Fig. 2a), indicating that patch shape, not connectivity, regulates plant disease dynamics.

Experiments two and three were designed to independently test two key aspects 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 (Fig. 2b). 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.

Experiment three tested the effects of patch shape and distance to edge on disease development, while eliminating the variability of spore dispersal. 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 m from the edge (Fig. 2c). In winged patches, highest disease development oc-

curred on plants 20 m from the edge, with lower levels of development at 0 and 40 m from the edge (Fig. 2c). 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 m from the edge, and experiment three plants were located 0, 20, and 40 m from the edge (Fig. 1). In experiment three, plants at the farthest distance (40 m) had significantly higher disease development in rectangular patches, but lower levels of development in winged patches (Fig. 2c). Because plants in experiment one were located at the distance of elevated disease development in rectangular patches (40 m), but not for winged patches (20 m), 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 in experiment one.

Temperature is a key factor affecting disease development (Jarosz and Burdon 1988) and can be used to explain patterns of disease seen in our experiments. Increased distance from the patch edge leads to increases in average temperatures (Fig. 3). 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 (Fig. 3).

These differences in temperature have important consequences for disease development. In winged patches, disease development was lower at the farthest distance from the edge. Temperatures there 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 m) from the edge in winged patches. In rectangular patches, temperatures at the farthest distance (51 m) were lower than those in winged patches, and did not limit southern corn leaf blight development.

Our findings contradict a number of previous studies that show significantly higher levels of disease in shaded areas (e.g., 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 our study, disease levels in plants near habitat edges were less than or equal to levels in plants farther from the edge (Fig. 2c). Also, while we selected the size and spatial distributions of our host populations, previous studies have found that disease incidence and prevalence can be dependent on interactions between host susceptibility and spatial distribution (Thrall and Antonovics 1995, Carlsson-Graner and Thrall 2002). In natural plant communities host plants vary in density, spatial distribution, and susceptibility to disease (Jarosz and Burdon 1988, Carlsson-Graner and Thrall 2002), and plant diseases vary in virulence and host specificity (Agrios 2005), making the real world far more complex than our experiments. The contradiction between our findings and those of previous studies highlights the complexity of plant disease dynamics (Sullivan et al. 2011). Our research provides experimental evidence that the key mechanisms driving plant–pathogen interactions in fragmented landscapes are due to habitat edges.

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 are dispersed by insect vectors, whose movements may be affected by corridors. Previous studies have shown that host spatial distribution does affect the occurrence of vectored diseases (Thrall and Antonovics 1995). Studies from our experiment have shown that corridors affect the movement of insect pollinators such as butterflies (Haddad et al. 2003), and disease levels of insect-caused galls are higher in patches connected by corridors (Sullivan et al. 2011). Yet, no experimental studies have examined the role of corridors in the spread of vectored plant diseases.

Using a model plant–pathogen system, we 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. Our 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 (Weldon and Haddad 2005; Haddad et al., *in press*). Our 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 our study organisms. Because we know relatively much about edge effects, we can potentially mitigate their negative effects by altering the amount of edge in fragmented systems. Our study provides evidence that corridors do not facilitate the movement of wind-dispersed fungal plant pathogens,

and that corridors still show more promise than risk for conservation.

Acknowledgments

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APPENDIX

Detailed descriptions of experimental site design, plant propagation and inoculation methods, and collection and analyses of wind data (*Ecological Archives* E092-129-A1).