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Influential disease foci in epidemics and underlying mechanisms: a field experiment and simulations

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Abstract. Pathogen invasions pose a growing threat to ecosystem stability and public health. Guidelines for the timing and spatial extent of control measures for pathogen invasions are currently limited, however. We conducted a field experiment using wheat (*Triticum aestivum*) stripe rust, caused by the wind-dispersed fungus *Puccinia striiformis*, to study the extent to which host heterogeneity in an initial outbreak focus influences subsequent disease spread. We varied the frequency of susceptible host plants in an initial outbreak focus and in the non-focus of experimental plots, and observed the progress of epidemics produced by artificial inoculation. The frequency of susceptible hosts in the initial outbreak focus increased the spread of stripe rust in the experimental plots, while frequency of susceptible hosts outside the initial outbreak focus did not. This suggests that factors influencing pathogen reproduction in the initial outbreak focus are key to the control of epidemics of stripe rust. Two mechanisms may underlie the field results. The first is the continuing, direct infection of susceptible hosts in areas outside the initial outbreak focus by disease propagules arriving from the initial outbreak focus. The second is highly local proliferation of disease caused by direct descendants of colonizing individuals originating from the initial outbreak focus. We considered these two alternatives in simulations of a generalized pathogen exhibiting fat-tailed dispersal, similar to *P. striiformis*. Simulations showed a dominant effect of conditions in the initial outbreak focus, in agreement with the field experiment, but indicated that, over time, this dominance may erode. Analysis of the duration of focal dominance led to the conclusion that both mechanisms contribute to the phenomenon of focal dominance, and that the frequency of susceptible hosts in the initial outbreak focus had a stronger influence when the proportion of propagules that remained local during dispersal was higher. Overall, our results suggest that targeting pathogen reproduction in the initial outbreak focus will have a disproportionately large impact on subsequent epidemic spread.

Key words: epidemic; fat-tailed dispersal; fungal pathogen; initial outbreak focus; local propagule deposition; pathogen invasion; Puccinia striiformis; *simulation experiment; stripe rust;* Triticum aestivum.

Introduction

The occurrence of pathogen invasions has increased greatly in recent centuries, likely owing to increased globalization (Mack et al. 2000, Crowl et al. 2008). The long-term ramifications of an accelerating pathogen invasion rate on worldwide ecosystem stability can be difficult to predict, but demonstrated impacts include an increased risk to endangered and threatened species (Harvell et al. 2002), threats to global food production (Brown and Hovmøller 2002, Brasier 2008), and substantial financial losses on national levels (Pimentel et al. 2005). Strategies to limit the spread and impact of invasive pathogens are thus becoming increasingly important (Lodge et al. 2006, Crowl et al. 2008). Implementation of tactics to control pathogen invasions is often costly and may induce lowered sensitivity of the pathogen, such that a balance between negative consequences of implementation of a control strategy and its effectiveness is necessary (Muller-Schafer et al. 2004, Horie et al. 2013).

Optimal control strategies, which seek a balance between effective control and negative impacts, have been proposed for a number of pathogen systems, including cholera (Tuite et al. 2011), oak wilt (Horie et al. 2013), and human influenza (Ferguson et al. 2006), among others (Lipsitch et al. 2003, Riley et al. 2003, Ferguson et al. 2005, Tildesley et al. 2006). Such strategies often require detailed knowledge of the pathogen and host, however, which may not be available during an invasion (Woolhouse 2011). Additionally, control strategies are typically aspatial and atemporal. That is, the geographic extent over which control measures should be applied is rarely considered, nor whether this extent should change over the course of an epidemic (Forster and Gilligan 2007, Parnell et al. 2010).

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Optimal control strategies for pathogen invasions have recently been addressed through spatiotemporal models (Forster and Gilligan 2007, Horie et al. 2013). However, the applicability of these models may be limited to those pathogens with exponentially bounded dispersal kernels. Yet, fat-tailed dispersal is common among pathogens, particularly those with aerially dispersed propagules (Ferrandino 1993, Scherm 1996, Frantzen and van den Bosch 2000).

Previous field studies (Cowger et al. 2005, Mundt et al. 2009, 2011, 2012) and preliminary simulation analyses (*unpublished data*), suggest that dispersal of pathogen propagules originating from an initial outbreak focus of disease has a dominant effect on epidemic spread, compared to inter-host movement of propagules in the areas surrounding the initial outbreak focus. Here, the term "initial outbreak focus" refers to the contiguous area encompassing all infections at the point in time when a pathogen invasion is first detected. The term "non-focus" refers to the area in the landscape that surrounds the initial outbreak focus. We propose two possible mechanisms that may drive this focal dominance, continuing infection by propagules produced in the initial outbreak focus and local proliferation of propagules produced by the initial colonizers from the initial outbreak focus. For the first mechanism, the majority of infections in the host population in the non-focus result directly from propagules produced in the initial outbreak focus. For the second mechanism, initial dispersal from the initial outbreak focus establishes a spatial pattern of colonizers in the outlying host population, and subsequent disease increase is driven by a highly local pattern of new infections around the initial colonizers. Both mechanisms are consistent with observations of organisms with fat-tailed dispersal, whereby propagule distribution patterns are primarily local but have a long-distance component crucial to colonization and spread (Kot et al. 1996, Hastings et al. 2005, Mundt et al. 2009).

In order to evaluate dominance of the initial outbreak focus in disease spread, we conducted a replicated field experiment in which plots of wheat (*Triticum aestivum*) were artificially inoculated with spores of *Puccinia striiformis*, an aerially-dispersed fungal pathogen that causes the disease stripe rust. The frequency of susceptible plants in inoculated foci and in the non-foci of the plots was manipulated in a factorial design, and the expansion of the resulting epidemics was observed.

To investigate the mechanisms behind the dominance of the initial outbreak focus, we created computer-simulated epidemics. We evaluated the relative importance of continuing direct colonization by propagules produced in the initial outbreak focus vs. local proliferation in outlying areas following initial dispersal from the focus. We considered how this relationship changes over time, and how such temporal dependencies are moderated by the degree of local pathogen deposition. Our goal in undertaking this study was to provide a better theoretical framework for understanding the differential contributions of disjoint host areas to epidemic expansion in a landscape that could then be used to develop recommendations for more effective control practices.

Methods

Field experiment

Study system.—P. striiformis is a wind-dispersed pathogen (Chen 2005) with a fat-tailed dispersal kernel (Brown and Hovmøller 2002, Sackett and Mundt 2005). During the course of a single growing season, the pathogen increases through multiple generations of asexual urediniospores that settle on leaves and infect in the presence of free moisture and favorable temperatures.

Experimental design.—We planted winter wheat in October 2010 in two fields near Culver, Oregon, USA (Field A, 44°32′17″ N, 121°13′47″ W, 16.9 ha; Field B, 44°32′56″ N, 121°11′5″ W, 11.9 ha; inter-field distance of 3.75 km). Each field was planted with 12 rectangular plots (85 × 18 m) arranged in rows and columns. All areas between plots were planted with a wheat cultivar completely resistant to the inoculated race of *P. striiformis*. Distances between plots varied, but were never less than 18 m in any direction (Appendix A: Fig. A1). A 1.5-m strip in the center of each plot was planted with either the same or a different frequency of susceptible host plants than the remainder of the plot (Fig. A1) to alter pathogen reproduction via dilution of susceptible plants (Mundt 2002).

The experiment followed a randomized, complete block design with four treatments and three replicates per field. Treatments were a factorial of host heterogeneity (either 100% or 30% frequency of susceptible plants) in the initial outbreak focus and in the non-focus of plots (Fig. A1). Susceptible plants were of the cultivar Jacmar; the 30% susceptible frequency was attained by mixing Jacmar with the completely resistant cultivar Stephens. Overall density (total number of plants per unit area) was the same in all cases. Because of a planting error, there was an extra all-susceptible plot and no mixture/susceptible plot in one replication in Field A.

We inoculated 1.52 × 1.52 m foci in the center of each plot with *P. striiformis* urediniospores of race PST-5 on 12 April 2011 (Fig A1). The areas to be inoculated were first sprayed with distilled water, and then dusted evenly with a mixture of 0.42 g of spores and 3.2 g of talc, using a PVC frame covered in clear plastic sheeting to prevent the spread of spores outside of the inoculation area. Foci were then covered for a minimum of eight hours with black plastic sheeting to maintain free moisture levels. The inoculation in Field B did not result in enough lesions for a tractable degree of disease spread. Therefore, we reinoculated foci in this field on 19 May 2011, applying inoculum to only the 1.52 × 0.76 m central area of plots (due to limited inoculum), with the

long edges of the inoculation areas parallel to the long edges of the plots.

Disease assessments were made at the end of the wheat growing season (after approximately 4.4 pathogen generations), at sampling points located downwind (east) of foci at 6.1-m intervals along plot midlines. At each sampling point, a 62.5×62.5 cm frame was placed over the plants by two observers. The exterior of this frame was made of PVC pipe, and the interior consisted of 25 2.5×2.5 cm sampling squares sectioned off by twine threaded through the PVC frame. We recorded the number of stripe rust lesions on the topmost leaf of the most centrally located susceptible tiller in each sampling square. For leaves with too many lesions to distinguish individually, we recorded the percentage of leaf area covered by lesions, then estimated the number of lesions per Sackett and Mundt's (2005) method. Differences in seed head morphology allowed easy distinction between the susceptible and resistant cultivars. When there were no tillers of the susceptible cultivar in a sampling square, we counted the lesions on the nearest susceptible tiller in an adjacent sampling square. Disease prevalence for each susceptible leaf was calculated by dividing the observed number of lesions by the maximum number of lesions/leaf observed at each field (20 for Field A, 28 for Field B).

Data analysis.—We tested the hypothesis that the initial outbreak focus is the only area where frequency of susceptible plants influenced disease spread. Disease spread was measured by constructing disease gradients (average disease prevalence vs. distance), then calculating the area under the disease gradient (AUDG) for each plot (Madden et al. 2007). We used a linear mixed modeling framework, with ln(AUDG) as the response variable. The frequencies of susceptible hosts in the initial outbreak focus and in the non-focus were fixed effects. Random effects included field and replicate nested within field. We evaluated models using the PROC MIXED procedure in the SAS statistical program (SAS Institute 2008).

Simulations

Model description.—We developed a one-dimensional simulation model based on stochastic spatial contact models (Mollison 1977, Xu and Ridout 1998) to simulate epidemics of a generalized pathogen capable of dispersal and reproduction on stationary hosts in discrete time. During each time step, individuals in each discrete spatial host unit produce descendant propagules that are then distributed among spatial units according to a specified dispersal function. Host units are arbitrary in the simulations, and can be considered as a single host individual or as a grouping of any number of hosts of interest. Although these models assume discrete generations, they closely approximate results of continuous time models with overlapping generations at the edge of epidemic expansion where prevalence is low (Madden et al. 2007).

At the beginning of each simulated epidemic, pathogen propagules infected a subset of host units. These individuals then produced offspring according to a Poisson distribution with mean and variance equal to the number of effective spores produced by each lesion each generation (the basic reproduction number, R_0). Offspring then dispersed according to the modified power dispersal kernel

$$\frac{\psi}{2}\lambda^{\psi}(\lambda + |d|)^{-(\psi+1)} \quad (1)$$

where d is the distance from the transect center and Ψ and λ are distribution parameters (Madden et al. 2007, Clobert et al. 2012). For all simulations, the value of λ was set to 0.5, corresponding to the half-width of the propagule source (Mundt 1989). The value of Ψ was 1, in accordance with observations of inverse-power exponents of approximately two for disease gradients associated with numerous pathogens capable of long-distance dispersal (Mundt and Leonard 1985, Mundt 1989, Aylor and Ferrandino 1990, Sackett and Mundt 2005, Frezal et al. 2009, Mundt et al. 2009).

In the time step following dispersal, newly arrived propagules at each host produced s offspring that then dispersed according to the dispersal kernel among all other hosts, with this cycle repeating at each subsequent time step. Pathogen propagules produced offspring only once, directly after dispersal, so that each time step represents a pathogen generation. When propagules dispersed to hosts already occupied by other propagules, the number to successfully colonize the host was the product of the number of propagules and the proportion of the host tissue that was unoccupied.

This simulation model is succinctly described by the set of recursive equations

$$I(x_i, t+1) = \sum_{j=1}^{N} k(|x_j - x_i|)\gamma I(x_j, t)H(x_i, t)$$

$$H(x_i, t+1) = H(x_i, t) - I(x_i, t+1)$$

$$R(x_i, t+1) = R(x_i, t) + I(x_i, t) \quad (2)$$

where $I(x_i, t)$, $H(x_i, t)$, and $R(x_i, t)$ are the number of infected or spore-producing (I), uninfected (H), and removed (R) units of tissue on the host at transect coordinate x_i at generation t. The term $k(|x_j - x_i|)$ is the probability distance kernel based on the distance ($|x_j - x_i|$) between hosts at transect coordinates x_j and x_i and γ is equal to R_0. We used Matlab (The Mathworks, Natick, Massachusetts, USA) to develop the simulation models and run the simulations.

Parameter combinations.—For the simulations, we translated our experimental design from the field experiment to the simulation environment. Simulated epidemics consisted of 10 pathogen generations; hosts were situated one unit apart along a one-dimensional transect 11 985 units long; carrying capacity was 10 000

infections per host unit. Each epidemic was initiated by dispersing one propagule to each of the five host units at the center of the transect.

Experimental treatments were modeled by designating two distinct regions of the simulated space: the initial outbreak focus, consisting of the five-unit block located at the midpoint of the transect, which we inoculated; and the non-focus, i.e., the space on either side of the focus (Appendix B: Fig. B1). Each region had either 100% or 20% frequencies of susceptible hosts, with $R_0 = 10$ for susceptible hosts and $R_0 = 0$ for the resistant hosts.

We also varied two factors in the simulations that were constant in the field experiment. The first was the local propagule deposition of the pathogen, defined as the proportion of propagules produced by an infection that never disperses farther than its host unit of origin. That is, if local propagule deposition is 0.1, then 90% of the propagules disperse to host units other than the unit where they originated. Simulations were run with local propagule deposition at nine levels, ranging from 0.1 to 0.9 in increments of 0.1.

The second additional factor in the simulations was cessation of pathogen reproduction in the initial outbreak focus. Either the epidemic progressed normally, or pathogen reproduction in the initial outbreak focus ceased after one pathogen generation. This corresponds to a disease control measure applied only to an initial outbreak focus that completely suppresses pathogen reproduction.

Data analysis.—As in the field experiments, the AUDG was used to indicate epidemic outcome. The AUDG for susceptible hosts was calculated at the end of each time step for each of four replicates of each parameter combination.

For each combination of local propagule deposition and cessation treatment, we developed multiple linear regression models at each of the last nine time steps of the simulated epidemics. (In the first time step, there is disease only in the initial outbreak focus, so AUDG does not differ among treatments.) The frequencies of susceptible hosts in the initial outbreak focus and in the non-focus were predictor variables. We used the values of the squared semipartial correlations (part-R^2) associated with each predictor variable as a measure of the influence of the areas of the landscape where the frequencies of susceptible hosts were varied on epidemic expansion. A predictor variable's part-R^2 measures its unique contribution to the coefficient of determination of a regression model that includes it and other predictors (Cohen et al. 2003).

We defined the duration of focal dominance as the length of time, starting from the time of initial inoculation, that the influence of the initial outbreak focus, quantified as the part-R^2 associated with the frequency of susceptible hosts in the initial outbreak focus, exceeded the influence of the non-focus (the part-R^2 for frequency of susceptible hosts in the non-focus).

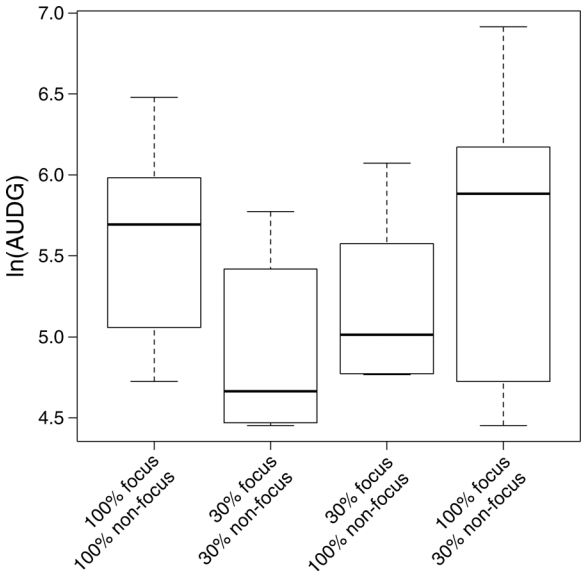

FIG. 1. Area under the disease gradients (AUDGs; measured as meters) for artificially inoculated epidemics of wheat stripe rust in a field experiment to assess the influence of initial outbreak foci on disease spread. Percentages refer to frequency of susceptible hosts in different regions of the field plots, 100% (monoculture of a susceptible cultivar) or 30% (mixture of 30% susceptible and 70% resistant cultivar). AUDGs were based on prevalence of disease on the susceptible host genotype assessed at the end of the growing season. Boxplots show medians, upper, and lower limits of interquartile ranges (IQR), and upper and lower limits of $1.5 \times$ IQR for three replicates at each of two locations. Focus refers to the initial outbreak focus in the plot and the non-focus refers to the area surrounding the initial outbreak focus.

Duration of focal dominance was regressed on local propagule deposition for both continuous and cessation simulations in order to examine the mechanisms underlying focal dominance in epidemics. Regressions were performed using the stats and lmSupport packages in R (R Development Core Team 2013).

RESULTS

Field experiment

Disease gradients indicated spread of stripe rust into the non-focus by the end of the experiment in all plots (Fig. 1, Appendix A: Fig. A3). AUDG in plots with 30% susceptible plants in the initial outbreak focus was lower than that in plots with 100% susceptible plants in the initial outbreak focus ($P = 0.010$). There was less evidence to suggest that the frequency of susceptible hosts in the non-focus was also influential ($P = 0.157$). Moreover, there was little indication of an interaction between the frequency of susceptible hosts in the initial outbreak focus and the frequency of susceptible hosts in the non-focus ($P = 0.151$; Table 1, Table A1).

Simulations

Simulated epidemics steadily expanded in time and space (Fig. 2). The frequency of susceptible hosts in the

TABLE 1. ANOVA table for a general linear mixed model of stripe rust epidemic spread in a field experiment based on the frequencies of susceptible hosts in the initial outbreak focus and in the non-focus, as well as the interaction of those frequencies

Source of variation	Mean square	df	*F*	*P*
Fixed effects				
Initial outbreak focus susceptible host frequency	1.75	1, 15	8.59	0.010
Non-focus susceptible host frequency	0.45	1, 15	2.22	0.157
Interaction	0.47	1, 15	2.29	0.151
Random effects				
Field	6.20	1, 4	79.28	0.001
Block (field)	0.08	4, 15	0.38	0.816
Residual	0.20			

initial outbreak focus significantly influenced AUDG for at least six generations in all simulation sets ($P < 0.05$). In addition, the frequency of susceptible hosts in the initial outbreak focus was a significant predictor of AUDG for all nine generations when local propagule deposition was less than 0.7 with focal pathogen reproduction cessation, and for all values of local propagule deposition when focal pathogen reproduction continued throughout the simulation. The frequency of susceptible hosts in the non-focus significantly impacted AUDG for at least four generations in all simulation sets (Appendix B: Table B1).

In general, the influence of the initial outbreak focus decreased over time, while the influence of the non-focus increased (Fig. 3, Fig. B2). The specific pattern of temporal change of the initial outbreak focus and non-focus influences depended on cessation treatment, as well as local propagule deposition (Fig. 3, Fig. B2).

Temporal trend lines of the influences of the initial outbreak focus and the non-focus intersected for all but the two highest levels of local propagule deposition (0.8 and 0.9) for continuous focal reproduction and at all local propagule deposition rates for arrested focal reproduction (Fig. 3; Fig B2). This intersection point indicates the point in time at which the initial outbreak focus and non-focus influences become equal. Duration of focal dominance was shortened by cessation of focal pathogen reproduction ($\bar{x} = 3.68$, SD = 1.60, $n = 9$ simulations), relative to simulations where pathogen reproduction was allowed to continue throughout the epidemic ($\bar{x} = 6.98$, SD = 1.30, $n = 9$ simulations; Figs. 3 and 4; Table B2).

Duration of focal dominance increased as local propagule deposition increased (Fig. 4), regardless of whether focal pathogen reproduction was continuous (slope = 5.240, SE = 1.315, $P < 0.010$) or arrested (slope = 7.053, SE = 0.997, $P < 0.001$; Fig. 4); there was no evidence to suggest a difference in slope between the continuous and arrested epidemics ($t = -1.010$, df = 12, $P = 0.293$). The duration of focal dominance was significantly shorter when the cessation treatment was applied, however, evident from a difference in intercepts between continuous and arrested epidemics ($t = 5.461$, df = 12, $P < 0.001$).

DISCUSSION

Our results provide predictions about the ways in which limiting pathogen reproduction within spatial subregions of the host landscape may ultimately impact epidemic expansion for pathogens with fat-tailed dispersal. Specifically, our results suggest that, the higher the local propagule deposition of such a pathogen, the longer the period of time during which an initial outbreak focus of disease is the main contributor to epidemic expansion in the host population that surrounds the initial outbreak focus. As such, limiting pathogen reproduction in the initial outbreak focus alone may reduce the majority of subsequent expansion of an epidemic when the epidemic is caused by a pathogen with fat-tailed dispersal and high local propagule deposition. Description of this relationship between propagule dispersal properties and disease control are of the type that could be useful to the development of general guidelines for managing pathogen invasions.

Two primary inferences from the simulation studies form the basis for our predictions. First, the influence of the initial outbreak focus always changed over the course of the simulated epidemics but, regardless of its trajectories, was always greater than the influence of the non-focus during early epidemic expansion. The time point at which the influence of the initial outbreak focus host dropped below that of the influence of non-focus depended on local propagule deposition. The initial outbreak focus was more influential than the non-focus for a longer period of time the higher the local propagule deposition of the pathogen (Fig. 3). Thus, pathogen reproduction in the initial outbreak focus had a longer-lasting effect on epidemic expansion compared to that occurring in other regions of the host landscape the higher the local propagule deposition of the pathogen.

The mechanism for a persistent influence of the initial outbreak focus was addressed through cessation of focal pathogen reproduction in the simulations. Any difference in the effect of focal host composition on duration of focal influence between epidemics with continuous focal pathogen reproduction and those with arrested focal pathogen reproduction must be attributed to continuing spread from initial outbreak focus to the

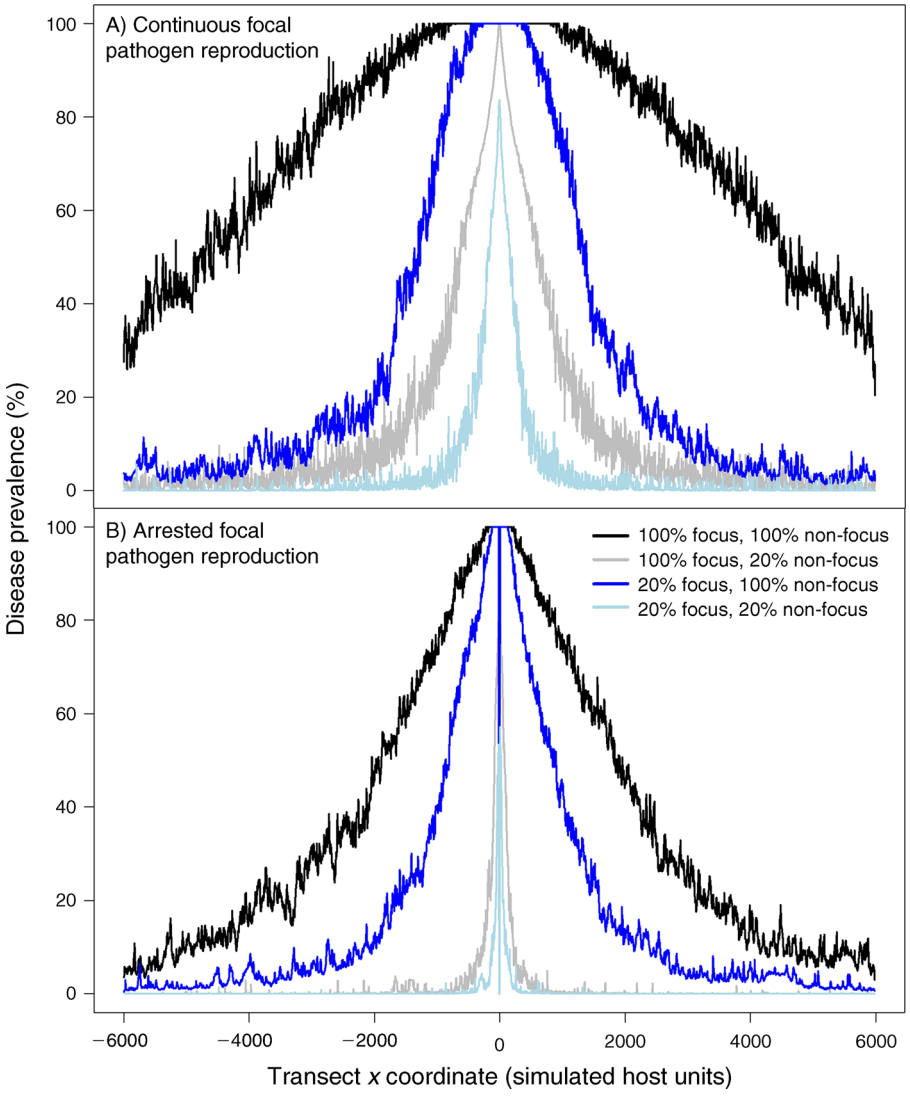

FIG. 2. Disease gradients in simulated epidemics after 10 pathogen generations, following inoculation at transect midpoints. Disease prevalence is the average over four replicates and nine local propagule deposition values associated with simulated pathogen dispersal. (A) Results of simulations in which pathogen reproduction in the initial outbreak focus (i.e., the focus) was continuous throughout the epidemic. (B) Results of simulations in which pathogen reproduction in the initial outbreak focus was arrested after a single pathogen generation.

non-focus. By contrast, identical results between continuous and arrested foci would indicate that proliferation of colonizers outside the initial outbreak focus was solely responsible for influential foci. Our results were intermediate between these two extremes, suggesting that continuing infections from the initial outbreak focus and proliferation of colonizers both played a role in the simulated epidemics. This is further supported by the fact that duration of focal dominance increased with local propagule deposition even with arrested focal reproduction. Results of the field experiment do not allow us to determine whether dominance of the initial outbreak focus in the stripe rust epidemics was driven primarily by proliferation. However, a subsequent field experiment (Severns et al., *unpublished manuscript*) showed that culling the initially inoculated focus with a fungicide/herbicide mixture after 1.3 generations of infectious disease spread had essentially no impact on the subsequent spread of disease, suggesting that the influence of the initial outbreak focus must be due primarily to local proliferation of colonizers.

Clearly, targeting interventions (e.g., culling or use of a chemotherapeutic agent) to disease outbreak sites would require successful identification of an initial outbreak focus at an early stage of epidemic spread. Foci of plant disease have often been observed in both agricultural (Zadoks and van den Bosch 1994) and in natural ecosystems (Gilbert 2002). Further, some plant

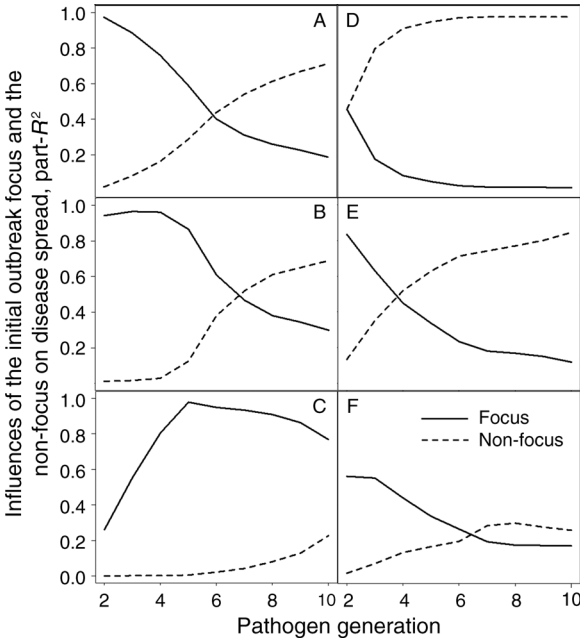

FIG. 3. Temporal change in the influence of the initial outbreak focus (i.e., the focus) and of the non-focus on spread of a plant pathogen during simulated epidemics. Influences of the initial outbreak focus (solid line) and the non-focus (dashed line) were quantified as the squared semipartial correlations (part-R^2) associated with the frequency of susceptible hosts in these two regions of the host landscape, respectively, when such frequencies served as factors in a regression model of pathogen spread. Results are shown for different levels of local propagule deposition (LPD) associated with dispersal of the pathogen: (A and D) LPD = 0.1, (B and E) LPD = 0.5, and (C and F) LPD = 0.9. Panels (A–C) show results for simulations with continuous pathogen reproduction in the initial outbreak focus throughout the epidemic; panels (D–F) show results when focal pathogen reproduction was arrested after the first pathogen generation.

disease invasions recur annually as the pathogen's overseasoning range is highly restricted by environment, and they spread anew each year when favorable conditions return (Mundt and Browning 1985, Aylor 2003, Christiano and Scherm 2007). There are ongoing disease surveillance systems in place for the purpose of early detection and monitoring of subsequent spread for some of these recurring plant disease epidemics (e.g., Isard et al. 2011). Surveillance systems are of course much more extensive for human diseases. In some cases, surveillance may be difficult, or there may be overlapping foci of infection. Thus, the ability to identify and treat outbreak foci is case specific. A second application of our results is in understanding how anthropogenic impacts on ecosystems might influence disease spread. For example, our work would enable a better understanding of how reductions in host diversity or effects on pathogen reproduction rate caused by habitat degradation or climate change would influence disease spread from initial outbreak foci.

The generality of our results and their relevance to the management of epidemics caused by epidemic invasions are important considerations. Scale independence of the power law (Gisiger 2001) can result in patterns of disease spread with considerable generality for pathogens with long-distance dispersal (LDD) and inverse-power dispersal kernels (Jeger 1983, Ferrandino 1993, Madden et al. 2007, Mundt et al. 2009), the type of fat-tailed dispersal exhibited by wheat stripe rust (Brown and Hovmøller 2002, Sackett and Mundt 2005) and modeled in the simulation studies. Though agricultural landscapes are simpler than natural ones (Hietala-Koivu 1999), previous work has indicated consistent patterns of disease spread for data sets including a wide range of spatial scales and a diversity of LDD pathogens of plants and animals in both agricultural and non-agricultural landscapes (Mundt et al. 2009). There is increasing evidence that propagule deposition is substantially more local than previously expected for multiple LDD plant pathogens (Mundt 2009), though local deposition remains very difficult to measure directly (Lannou et al. 2008, Mundt 2009). Our previous results with disease-spread and spatial scaling, which incorporated a wide range of spatial scale and disease systems, can only be reasonably explained if one assumes a high proportion of local deposition (Sackett and Mundt 2005, Mundt et al. 2009, 2011, 2012, 2013). Finally, from an evolutionary perspective, modeling

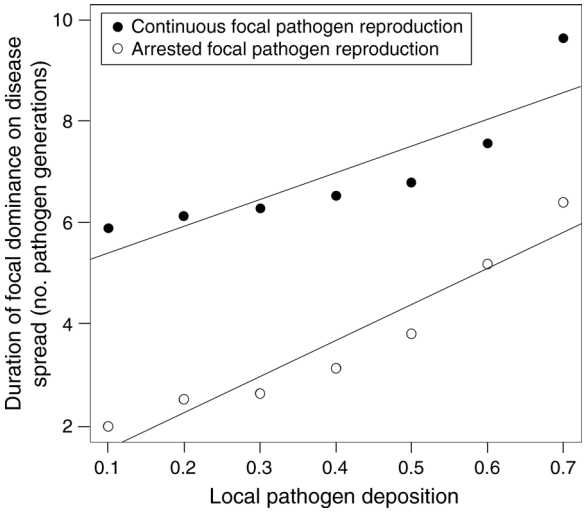

FIG. 4. Effect of local propagule deposition and cessation of pathogen reproduction on the duration of influence of the initial outbreak focus (focal dominance) on disease spread. The *y*-axis is the length of time, starting from the initiation of simulated epidemics, that the influence of the initial outbreak focus (part-R^2 associated with the frequency of susceptible hosts in the initial outbreak focus) was greater than the influence of the non-focus (part-R^2 associated with the frequency of susceptible hosts in the non-focus). In simulations, pathogen reproduction either continued throughout the epidemic (black circles), or ceased after one generation (white circles).

results suggest that pathogen reproduction and velocity of disease spread are optimized when propagule deposition is approximately 80% local (Zawolek and Zadoks 1992), a degree of local deposition that our modeling results predict to result in a strong influence of the initial outbreak focus on epidemic spread, with important implications for mitigation of epidemic invasions.

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Supplemental Material

Appendix A

Figures and tables pertaining to design and results of the stripe rust field experiment in Madras, Oregon, USA conducted during 2011 (*Ecological Archives* A024-210-A1).

Appendix B

Figures and tables pertaining to design and results of a simulation experiment of influences on expansion of an epidemic caused by a generalized pathogen (*Ecological Archives* A024-210-A2).