Spatial scaling relationships for spread of disease caused by a wind-dispersed plant pathogen

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Abstract. Spatial scale is of great importance to understanding the spread of organisms exhibiting long-distance dispersal (LDD). We tested whether epidemics spread in direct proportion to the size of the host population and size of the initial disease focus. This was done through analysis of a previous study of the effects of landscape heterogeneity variables on the spread of accelerating epidemics of wheat (Triticum aestivum) stripe rust, caused by the fungus Puccinia striiformis f. sp. tritici. End-of-season disease gradients were constructed by estimating disease prevalence at regular distances from artificially inoculated foci of different sizes, in field plots of different dimensions. In one set of comparisons, all linear dimensions (plot width and length, focus width and length, and distance between observation points) differed by a factor of four. Disease spread was substantially greater in large plot/large focus treatments than in small plot/small focus treatments. However, when disease gradients were plotted using focus width as the unit distance, they were found to be highly similar, suggesting a proportional relationship between focus or plot size and disease spread. A similar relationship held when comparing same-size plots inoculated with different-sized foci, an indication that focus size is the driver of this proportionality. Our results suggest that power law dispersal of LDD organisms results in scale-invariant relationships, which are useful for better understanding spatial spread of biological invasions, extrapolating results from small-scale experiments to invasions spreading over larger scales, and predicting speed and pattern of spread as an invasion expands.

Key words: epidemiology; invasions; long-distance dispersal (LDD); power law; Puccinia striiformis f. sp. tritici; scaling; Triticum aestivum.

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INTRODUCTION

Ecological processes occur at widely different temporal and spatial scales, and observed ecological patterns can be influenced by both the scale of study and the scale of measurement (Wiens 1989, Levin 1992, Schneider 2001, Leibold et al. 2004). Of particular significance is that ecological studies are often conducted at small spatial and temporal scales, while ecological processes and patterns often extend to much larger scales (Wiens 1989, Levin 1992). This dilemma has resulted in a search for relationships that are predictive across a wide range of scale. For example, power laws and fractals have been used to develop scaling relationships for a variety of ecological processes and patterns (West et al. 1997, Ritchie and Olff 1999, Haskell et al. 2002). Percolation theory (With 2002), metapopulation approaches (Leibold et al. 2004), and network models (Proulx et al. 2005) have also been used to infer ecological processes and
patterns across scales. Issues of scale are also expected to be crucial to ecological studies of disease (Smith et al. 2003, Matthews and Haydon 2007). Individual pathogen infections occur at the microscopic spatial scale and often at time scales of only hours. However, rapid pathogen reproduction can facilitate disease spread at the continental or even inter-continental level over relatively short timeframes (Brown and Hovmöller 2002, Aylor 2003, Mundt et al. 2009b). Inoculum deposition can be concentrated at a surprisingly small spatial scale (Lannou et al. 2008, Mundt 2009), yet a minority of propagules can be dispersed hundreds or even thousands of kilometers (Brown and Hovmöller 2002, Griffin et al. 2002, Aylor 2003), making a disproportionate contribution to the rate of disease spread (Hastings et al. 2005). Therefore, spatial scale is critical to the study of epidemics caused by pathogens with propagules that have the potential for long-distance distance dispersal (LDD), either through air—such as foot-and-mouth disease (Keeling et al. 2001), West Nile Virus (Peterson et al. 2003), avian influenza (Kilpatrick et al. 2006), and many diseases of plants (Brown and Hovmöller, 2002)—or through water, such as Aspergillosis of coral (Weir-Brush et al. 2004).

Epidemic invasions have substantial impacts on both ecosystem function and human welfare (Mack et al. 2000, Pimentel 2002, Anderson et al. 2004, Crowl et al. 2008). The frequency of epidemic invasions is likely to increase with globalization (Tatem et al. 2006) and, through a variety of mechanisms, also may be exacerbated by pollution, habitat destruction, and climate change (Weiss and McMichael 2004). Elucidating the processes underlying establishment and spread of epidemic invasions, including an understanding of how epidemic patterns translate across spatial scales, can contribute significantly to mitigation of epidemic invasions (Meyers et al. 2003, Gewin 2004, Meyers et al. 2005, Smith 2006, Riley 2007, Brooks et al. 2008).

Dispersal of LDD pathogens is often characterized by “fat-tailed” dispersal kernels, such as the power law, which are not exponentially bound and result in accelerating epidemic fronts (Ferrandino 1993, Kot et al. 1996, Clark et al. 2001, Hastings et al. 2005, Mundt et al. 2009a, b). Similarly, “anomalous diffusion” has been suggested to result in fat-tailed dispersal kernels and superdiffusive spread of a range of organisms (Brockmann et al. 2006, Viswanathan et al. 2008). Because of the scale-invariant nature of the power law (Gisiger 2001), patterns of disease spread caused by LDD pathogens might be expected to repeat at different spatial scales. Indeed, we have previously found that a simple descriptive model of accelerating epidemic spread based on power law dispersal holds over approximately six orders of spatial magnitude, from experimental field plots to intercontinental spread, and for a wide variety of animal and plant diseases caused by LDD pathogens (Mundt et al. 2009a, b).

Herein we test spatial scaling relationships through further analysis of an experimental study of the effects of landscape heterogeneity variables on the spread of accelerating epidemics of wheat (*Triticum aestivum*) stripe rust, caused by the fungus *Puccinia striiformis f. sp. tritici* (Mundt et al. 2011). Those experiments incorporated treatments that allowed us to test the hypothesis that final epidemic extent is directly proportional to the size of field plots and the size of initial disease foci that initiated the epidemics.

**METHODS**

**Field methods**

Field methods have been described in detail (Mundt et al. 2011) and are summarized briefly below. A subset of the treatments in the field experiments is used in the current analysis.

Experiments were conducted in 2006, 2007, and 2008, in commercial wheat fields in an area of irrigated agricultural production in Jefferson County, OR, USA. Agronomic practices were standard for commercial wheat production in this region, including overhead irrigation at approximately 7–10 day intervals beginning in March. Two winter wheat genotypes were planted, one highly susceptible and the other completely resistant to race PST 5 of *P. striiformis*, the race used for artificially inoculating disease foci. The susceptible genotype was grown either in monoculture or in mixture with the resistant one in different proportions. (The primary purpose of these mixtures in the original study was to test the effect of susceptible host frequency on disease spread, which is not addressed...
here.) Experimental plots were arranged each year in a large field of about 16 hectares, separated from one another by 18 to 50-m buffer zones of the resistant wheat variety, in order to minimize spore dispersal between plots.

In 2006 and 2007, the field experiments consisted of 12 treatments replicated three times in a split-plot design. Main plots were arranged in a randomized complete block design in 2006 and a Latin square in 2007. The three main-plot treatments were a monoculture of the susceptible host genotype; a mixture of the susceptible genotype and the resistant one, with a target of 50% susceptible; and a mixture with a target of 25% susceptible plants. The four subplot treatments were small, medium, and large plot sizes (6.1 × 18.3, 12.2 × 36.6, and 24.4 × 73.2 m, respectively) inoculated with a small initial disease focus (0.38 × 0.38 m), plus the large plot size inoculated with a large disease focus (1.52 × 1.52 m). The medium-sized plots are not relevant to the current study. In 2008, the experimental design was a modified Latin square with seven treatments and three replications, but no split-plot arrangement (Mundt et al. 2011). Two host frequencies (100% and 25% susceptible) and three spatial arrangements (small plots with small foci, large plots with small foci, and large plots with large foci, with dimensions as described above) were combined factorially to create six treatments. The seventh treatment was a monoculture of the susceptible genotype, in a large plot with an extra-large focus (3.05 × 3.05 m). Field maps showing the complete layout for all three years can be found in Fig. 1 of Mundt et al. (2011).

Each plot was inoculated with urediniospores of P. striiformis race PST 5 in the spring of each year. Disease prevalence (as a percentage of the maximum number of lesions, which is approximately 100 lesions/tiller) on the susceptible host genotype was visually estimated weekly in 0.38 × 0.38-m observation units from the end of the first latent period after inoculation until late June/early July. In large plots, disease was assessed at 0, 3.0, and 6.1 m from the center of the focus and every 6.1 m thereafter; the distance between assessment points was reduced by a factor of four in the small plots.

**Treatment comparison**

Three types of treatment comparisons were used to study potential scaling relationships for spatial spread (Fig. 1). In the first, which we will call Type A, we compared small plots with small foci to large plots with large foci. This represents a scaling of all of the linear dimensions (plot width and length, focus width and length, and distance between observation points) by a factor of four. This comparison allows us to examine the question of whether an epidemic can be scaled in direct proportion to spatial dimensions of the host population and outbreak focus.

Type B and Type C comparisons address the influence of focus size on disease spread in host populations (i.e., plots) of the same dimensions. In the original analysis (Mundt et al. 2011), host frequency and initial focus size had strong effects on epidemic spread, but plot size unexpectedly had no detectable influence. This suggested that it might be possible to simply scale epidemics by focus size, independent of the dimensions of the plot. In the Type B comparisons, large plots with small foci were compared to large plots with large foci (averaged over all host frequencies). This isolates the effect of focus size, while holding plot size constant.

In 2008, a further investigation of the effect of focus size was possible. This is the Type C comparison, where a larger range of focus sizes (small, large, and extra-large, constituting an eight-fold difference) is compared, again in large plots, but limited to monocultures of the susceptible host genotype, since the extra-large focus only occurred in monoculture plots.

**Data analyses**

In each year, the final extents of the epidemics were used to make treatment comparisons. This was done by calculating the area under the disease gradient (AUDG) for each plot, at the final assessment date, using the mid-point method (Madden et al. 2007). Two different AUDGs were calculated for each field plot, one based on distance expressed in meters (non-scaled) and a second calculated from distance expressed as focus widths (scaled). In all cases, AUDG was calculated using only observation distances common to all plot sizes in the comparison. For Type A non-scaled comparisons, AUDG was calculated to 15 m (the maximum
observation distance common to both large and small plots). In Type A scaled comparisons, the maximum observation distance was 40 focus widths for both large and small plots, and thus all observations were used. For Type B and C comparisons, plot lengths were the same for all treatments, so all observations were used to calculate the non-scaled AUDG. The scaled AUDG was calculated to a distance of 40 focus widths (maximum number of focus widths in large plots with large foci) for Type B comparisons and to 20 focus widths (maximum number of focus widths in large plots with extra-large foci) for Type C comparisons.

Log-transformed AUDG values were subjected to analysis of variance (ANOVA) using Proc Mixed of SAS (SAS Institute 2008). These analyses were done separately for each experiment as the experimental designs differed among years, and separately for scaled and non-scaled AUDG. Experimental blocks (replicates) were considered random and all other effects were considered fixed.

For 2006 and 2007, treatment effects were host frequency (main plots), plot configuration (plot size and focus size combinations; subplots), and their interaction. Type A and Type B comparisons were made using subplot linear contrasts. As there were no significant main plot $\times$ subplot interactions ($P > 0.05$) in any of the ANOVAs, these contrasts were averaged over host frequency treatments.

A split-plot design was not used in 2008, and the ANOVA included only block, treatment, and error effects. Linear contrasts for Type A and Type B comparisons were conducted within the overall treatment effect. Data from plots with extra-large foci were excluded from the ANOVA for Type B comparisons for 2008, as these plots had half as many “focus widths” available for calculating the AUDG and were not involved in the Type B comparison. Type A and Type B comparisons were conducted averaged over host frequency, as host frequency did not interact significantly ($P > 0.05$) with either plot size or focus size. A contrast for the linear effect of focus size was used to test the Type C comparison in an analysis including all 2008 treatments.

For Type A comparisons, plots of logit(prevalence) versus log(distance) were used to linearize the disease gradients (Berger and Luke 1979). Such plots would be expected to show similar slopes if there is a proportional relationship between gradients for the small plot/small focus and large plot/large focus treatments. Multiple linear regression was used to fit lines to the transformed data, with treatment as an indicator variable. An $F$-test for the interaction between log(distance) and treatment was used to test whether the slopes were equal. Variation associ-
ated with low prevalence values in gradient tails for the large plot/small focus treatments prevented useful analysis of linearized gradients for the Type B and C comparisons.

RESULTS

The Type A comparisons showed substantially larger AUDGs for non-scaled gradients in the large plot/large focus treatments than for the small plot/small focus treatments ($P = 0.02$ in 2006; $P < 0.0001$ in both 2007 and 2008). When scaled by focus width, these disease gradients were highly similar (Fig. 2) ($P = 0.96$, 0.03, and 0.83 for 2006–08, respectively). Logit-log plots (Fig. 3) show similar slopes for the linearized disease gradients of each year, further supporting a proportional relationship between the two treatments.

The Type B comparisons indicated disease spread that was proportional to size of initial focus when plot size was constant (Fig. 4). In all
cases, the AUDG was substantially greater for large foci than for small foci when distance was expressed in meters (P-values between <0.0001 and 0.02). The AUDGs for large and small foci were more similar when distance was scaled to focus width (though not as similar as for Type A comparisons); P-values for this linear contrast ranged from 0.19 to 0.95.

The Type C comparison, where the linear effect of focus size was compared in same-size monoculture plots, indicated increasing AUDG with increasing focus size (P = 0.01). The gradients were more similar (Fig. 5) and the linear effect of focus non-significant (P = 0.68) when AUDG was calculated with distance expressed as focus widths.

**Discussion**

Plots inoculated with large foci always had substantially greater extent of final disease spread than did plots inoculated with smaller foci (Figs. 2–5). Local temperature readings and a known relationship between temperature and latent period (Shaner and Powelson 1971) indicated that the pathogen passed through 4-5 latent periods from inoculation until the final disease reading in each of the three years (unpublished data), providing an opportunity for treatments to have an influence on disease patterns over multiple generations. These epidemics were previously shown to demonstrate the accelerating expansion expected of epidemics driven by power law dispersal (Mundt et al. 2011: Fig. 1 and Appendix B). Such accelerating epidemics are expected to spread at a velocity that is proportional to initial focus size (Madden et al. 2007, Mundt et al. 2009a, b). This expectation is supported by experimental results reported here, and may be of significant importance to disease control. For example, fat-tailed dispersal kernels have presented challenges to eradication of disease introductions such as citrus canker in Florida (Gottwald et al. 2001, Parnell et al. 2009), sudden oak death in Oregon (Hansen et al. 2008), and foot-and-mouth disease in the UK (Dybiec et al. 2009, Tildesley et al. 2009). Our results suggest that effective eradication measures need to be applied in direct proportion to size of the outbreak at time of implementation, rather than with a constant culling distance from the epidemic front that might be appropriate for a traveling wave epidemic.

Our results are consistent with a simple scaling relationship for the final extent of spread of wheat stripe rust. Type A comparisons provided the most thorough test, as plot dimensions, focus dimensions, and observation distances all varied proportionally. Disease gradients scaled to the size of initial disease foci were nearly identical for large plot/large focus versus small plot/small focus comparisons in 2006 and 2008 for Type A
comparisons, and P-values for the linear contrast approached 1.0. Though the P-value for the large plot/large focus versus small plot/small focus contrast was significant (P = 0.03) in 2007, the magnitude of the treatment difference was relatively small, the curves differed only over a restricted range of distance, and the F-value for the contrast was 15 times smaller than for the equivalent non-scaled comparison of that same year (Fig. 2).

We originally assumed that scaling all spatial variables simultaneously would be required to attain comparable disease gradients. However, field results unexpectedly showed no detectable effect of plot size on epidemic spread and no interaction of plot size with host frequency or focus size (Mundt et al. 2011). Although disease prevalence can increase with increasing plot size when plots are small, likely owing to increased retention of spores within plots as plot size increases (Paysour and Fry 1983), the response may become so small as to be undetectable at

Fig. 4. End-of-season prevalence (% of maximum number of infections) of wheat stripe rust at different distances from artificially inoculated foci for Type B comparisons of large plot/large focus (open circles) versus large plot/small focus (closed circles) treatments. Each data point is a mean over three (2006 and 2007) or two (2008) frequencies of susceptible plants, with three replications per treatment. In the left-hand panels (from Mundt et al. 2011), disease prevalence is plotted against distance in meters. In the right-hand panels, the unit distance is the focus width corresponding to each treatment. Values of F and P are for linear contrasts of large plot/large focus treatments versus large plot/small focus treatments within a mixed analysis of variance for area under the disease gradient.
larger plot sizes (summarized in Sackett and Mundt 2009). The plots in the current study were large enough to fall into this latter category. Because plot size had little or no influence on disease spread, simply scaling by focus size in large plots (Type B and C comparisons) also provided a reasonable scaling relationship, though not as close as for the Type A comparisons. In addition, practical limitations prevented us from obtaining disease readings at short intervals of distance, thus reducing the number of common distances for scaled epidemics in Type B and C comparisons. The difference in scaled AUDGs between small versus large foci was particularly large for 2006. The \( P \)-value for this difference was only 0.50, however, and likely reflects the larger experimental error reported previously for that year (Mundt et al. 2011).

Our results suggest that power law dispersal of LDD organisms (e.g., Gregory 1973, Harper 1977, Shaw et al. 2006, Fric and Konvicka 2007) produces scale-invariant relationships useful to understanding and predicting spatial spread of biological invasions. This may allow for extrapolation of results from small-scale experiments to invasions spreading over much larger distances, and for prediction of speed and pattern of spread as an invasion expands. Though dispersal mechanisms certainly differ greatly as compared to plant pathogens, it is interesting that power law relationships resulted in a direct relationship between the linear dimension of home range and dispersal distance of mammals (Bowman et al. 2002).

Though relatively homogeneous, agricultural systems often contribute ecological information of relevance to non-managed systems (Keesing et al. 2006, Borer et al. 2011). Scaling relationships described herein will be applicable to natural systems with host distributions that are relatively continuous and initial pathogen distributions that are spatially restricted. In cases of multiple initial disease foci and/or extreme heterogeneity of host spatial pattern, however, spatially explicit approaches may be required (e.g., Urban 2005, Brooks et al. 2008).

Though our field experiments required approximately 16 ha of land each year, we were still limited to studying a very narrow range of spatial scale. However, recent evaluation of soybean (Glycine max) rust (Phakopsora pachyrhizi) spread over millions of square kilometers of diverse terrain in North America (Mundt and Wallace, unpublished manuscript) showed a very strong correlation between beginning and ending epidemic extent, as would be predicted from the experimental results reported here. Further, a simple model of epidemic spread based on power law dispersal provided a good fit to a diversity of plant and animal epidemics caused by LDD pathogens that incorporated nearly a six-fold range of spatial scale (Mundt et al. 2009a, b).

Fig. 5. End-of-season prevalence (% of maximum number of infections) of wheat stripe rust at different distances from artificially inoculated foci in 2008 for Type C comparisons of large monoculture plots with small (closed circles), large (open circles) and extra large (triangles) foci. In the left-hand panel (from Mundt et al. 2011), disease prevalence is plotted against distance in meters. In the right-hand panel, the unit distance is the focus width corresponding to each treatment. Each data point is the mean of three replications. Values of \( F \) and \( P \) are for a test of the linear effect of focus size in a mixed analysis of variance for area under the disease gradient.
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