Consequences of Long-Distance Dispersal for Epidemic Spread: Patterns, Scaling, and Mitigation

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Epidemics have impacted ecosystem functioning and human welfare substantially throughout history (Anderson et al. 2004; Crowl et al. 2008; Fisher et al. 2012; Mack et al. 2000; Pimentel 2002). Globalization (Tatem et al. 2006) and climate change (Altizer et al. 2013; Helfer 2014) are expected to encourage more frequent and severe epidemics in the near future, and a history of failed attempts to control outbreaks (Cunniffe et al. 2014; Dybiec et al. 2009; Parnell et al. 2009; Peterson et al. 2015; WHO Ebola Response Team 2014) indicates that we lack a sufficient understanding of disease epidemiology to reliably project and prevent disease spread. Plant disease epidemiology might be considered as a highly specialized field of study. However, the concepts, models, and epidemiological control philosophies are consistent with the study of biological invasions, under which disease epidemics are considered a major subset by invasion biologists (Mack et al. 2000; Simberloff 2009). There are also similarities between plant and animal (including human) diseases that have emerged from both molecular (Ausubel 2005; Sexton and Howlett 2006; Staskawicz et al. 2001) and ecological (Borer et al. 2011; Mundt et al. 2009a) perspectives. Regardless of the organism's taxonomic affiliation or that of its host(s), plant pathologists, invasion ecologists, and epidemiologists share a common goal to understand the spread, ecology, evolution, and control of invasive organisms.

A pathogen's dispersal potential is often quantified with an equation describing a dispersal gradient, i.e., the decline of propagule numbers with distance from a source (Gregory 1968), or a dispersal "kernel", i.e., the probability density function describing the post dispersal spatial distribution of propagules relative to their source

(Nathan et al. 2012). The shape of a pathogen's dispersal gradient or kernel strongly affects the expected pattern of disease spread (Clark et al. 2001; Hastings et al. 2005; Kot et al. 1996). Classic approaches to modeling organism spread (Mollison 1977; Skellam 1951) predict a "traveling wave" with an invasion front of constant velocity, defined by the organism's reproductive capacity, generation time, and dispersal potential. These traveling wave approaches were based on random walk and diffusion models, which have been successfully applied to the spatiotemporal spread of a variety of organisms (Bjornstad et al. 2002; Cummings et al. 2004; Grenfell et al. 2001; Johnson et al. 2006; Lambin et al. 1998; McIntire et al. 2013). Traveling wave models are based on a key assumption that the decline of inoculum with distance is described by a function that is bounded by an exponential (Mollison 1977). A plant disease that spreads exclusively through close proximity or direct contact between hosts is likely to have an exponentially bound distribution function and to invade as a traveling wave of constant velocity (Cummings et al. 2004; Grenfell et al. 2001), unless it is vectored by another means, such as commercial transport.

Although traveling wave models have been applied to plant disease spread (Heesterbeek and Zadoks 1987; Minogue and Fry 1983a, b; van den Bosch et al. 1988a, b, c), this approach has been questioned for aeri ally dispersed pathogens on both observational and theoretical grounds, arguing that the dispersal gradient is likely to exceed the limits of an exponential function (Ferrandino 1993; Frantzen and van den Bosch 2000; Scherm 1996; Zadoks and van den Bosch 1994). Aerially dispersed pathogens often have dispersal gradients with the leptokurtic "fat tails" typically associated with long-distance dispersal (LDD). Early in the history of plant epidemiological study, varying forms of inverse power functions were used to describe disease gradients caused by LDD pathogens (Gregory 1968). Not only do these functions generate scaling predictions through power law scale invariance properties (Gisiger 2001), but they also account for the "fat-tailed" disease gradient displayed by LDD plant pathogens (Brown and Hovmöller 2002). A consequence of this fat-tailed disease gradient is that it can generate epidemics (in both plants and animals) with invasion fronts that increase in velocity as the epidemic spreads (Cowger et al. 2005; Ferrandino 1993; Mundt et al. 2009a, b; Pybus et al. 2012), challenging conventional disease containment strategies and projections based on diffusion (traveling wave) models. The phenomena of "anomalous diffusion" and superdiffusive organism spread have also been associated with the pronounced leptokurtic shape of power law associated functions (Brockmann et al. 2006; Viswanathan et al. 2008). Theoretically, the shape of an organism's dispersal kernel (or disease gradient) conveys information on its pattern of spread that could provide insights

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valuable for disease control. However, relatively few studies of plant disease spread have been conducted from this perspective.

In this paper, we summarize a series of experimental field and modeling studies of wheat stripe rust (WSR) to better understand the consequences of LDD on epidemic spread and control of single focus outbreaks. Multiple definitions for disease focus exist in the literature (Zadoks and van den Bosch 1994), but we will use the most restricted definition of a focus – the area encompassing disease produced from the primary inoculum in the first generation of pathogen reproduction. After discussing basic patterns of WSR spread from a single focus, we will present evidence of potentially important spatial scaling relationships and the influence of outbreak conditions on subsequent epidemic spread and control that may be broadly relevant for LDD pathogens. Though many real-world epidemics begin from multiple foci, studies of single outbreak sites can provide the data needed to predict disease spread from more complex patterns of initial outbreak.

Wheat Stripe Rust

A field model for disease spread. The basidiomycete pathogen, *Puccinia striiformis* f. sp. *tritici*, produces aggregate groups of uredinia that expand as stripes on wheat leaves. Epidemics typically

begin from clearly distinguishable foci that spread in time and space through successive generations of disease by producing a large number of aerially dispersed, asexual urediniospores (Milus et al. 2009). WSR latent periods vary from 10 days to several weeks, depending on temperature, host, and abiotic conditions (Chen et al. 2002; Papastamati and van den Bosch 2007). In most instances, WSR epidemics persist for four to five latent periods, but they can last up to 10 latent periods in the warmer, more humid, wheat growing regions of the world (Chen et al. 2002; Wan et al. 2004; Wellings 2007). The sexual stage of the pathogen on the alternate host *Berberis vulgaris* occurs very rarely, if at all, in agricultural areas (Wang et al. 2015).

A model system for disease ecology requires pathogen and host manipulation in an environment conducive to infection, but without interference from naturally occurring inoculum. We have maintained stocks of older club wheat (*Triticum aestivum* ssp. *compactum*) cultivars of varying stripe rust susceptibility and cultures of WSR races that cause disease on the older cultivars but not those currently planted for production. Using this system of older cultivars and WSR races, we experimentally initiated disease outbreaks with specifically designed plantings that were of minimal risk to growers and were unaffected by inoculum that might be produced in commercial fields. Our studies of WSR disease spread took place in two arid agricultural regions of central Oregon (Jefferson County) and north central Oregon (Umatilla County). Irrigation generated a vigorously growing wheat population and lower overnight temperatures contributed to the overnight dew formation necessary for WSR infection.

Establishment of wheat stripe rust outbreaks. Rectangular plots of wheat cultivar monocultures and mixtures were mechanically planted with long axes oriented east-west, aligning approximately with the prevailing wind direction during the epidemic season. Between experimental plots, completely resistant cultivars were planted to reduce the incidence of between-plot disease spread (Fig. 1). We replicated and experimentally manipulated the focus host composition and size (area) of the focus as well as the remainders of the experimental plots (at-risk populations) using combinations of susceptible, partially resistant, and resistant wheat cultivars to test specific disease spread hypotheses.

To inoculate experimental WSR foci, early tillering wheat plants were misted with water, dosed with a urediniospore-talc mixture in a large plastic enclosure, and covered with black plastic overnight to maintain moisture and ensure dew formation (Sackett and Mundt 2005a; Severns et al. 2014). Inoculations occurred in the late afternoon/early evenings from mid-March to mid-April to allow for the greatest number of latent periods (3.5 to 5.0 latent periods).

Measuring disease spread. Disease prevalence (synonym = plant disease severity), as a percentage of the maximum number of lesions (~100 lesions/tiller), was visually estimated within 1.52 m × 1.52 m quadrats centered over the focus and at regular distance intervals downwind and upwind along the long central axis of each plot. Flags were placed at each sampling point, and the estimate of prevalence was recorded, usually as the average between two assessors at opposite sides of the plot. The computer program DISTRAIN (Tomerlin and Howell 1988) was used to train personnel in the estimation of WSR disease prevalence. Initial disease prevalence values in the focus (produced by the primary disease generation before disease was detected outside of the focus) ranged from 0.001 to 30% (Severns et al. 2015). A degree-day model of WSR development (Shrum 1975) was used in conjunction with data from local weather stations to estimate lengths of latent periods.

The locations (distance from the center of the focus) of specific levels of disease prevalence (1, 5, 10, 25, 50, and 75% isopaths) were recorded for each weekly assessment date and used to estimate the advance of the epidemic front. Assessments were terminated during the later stages of grain fill, when plant senescence suppressed disease progress and obscured visibility of rust lesions. Epidemic velocity was calculated as the distance traveled between sampling dates divided by the time interval. In addition, disease prevalence was plotted against distance from the focus, and the area under that disease

Fig. 1. Top: An aerial photograph of four field plots (7.6 m × 61.0 m or 61.0 m × 61.0 m) containing experimental wheat stripe rust (WSR) infections initiated in Madras, OR, and photographed on 18 June 2004 (Sackett and Mundt 2009). WSR-infected plants appear as yellow regions; disease gradients appear to have a relatively long, continuous tails. Bottom: Mean WSR disease prevalence of these four plots versus the distance downwind from the focus at weekly intervals beginning on 20 May 2004. The mean disease gradients show the leptokurtic distribution of WSR and that disease occurs over far greater distances than are apparent in the aerial photograph (compare disease gradient on 17 June 2004 versus aerial photograph on 18 June 2004).

gradient (AUDG) (midpoint method: Madden et al. 2007; Shaner and Finney 1977) was used to quantify epidemic magnitude at latent periods of interest (Fig. 1).

Disease spread simulations. We used a spatially explicit simulation model (EPIMUL) to cross-validate field experiments and to explore epidemiological scenarios over a larger range of parameter values than was possible in the field. EPIMUL (Kampmeijer and Zadoks 1977) was originally published over 40 years ago, but has since been converted into different programming languages and modified to evaluate the influence of different dispersal gradients, lesion expansion, host resistance variation, changes in pathogen reproductive rates, virulence, and effective spore dispersal stochasticity (Boudreau and Mundt 1994; Lannou and Mundt 1996, 1997; Mundt et al. 1986; Sackett and Mundt 2005b; Severns et al., unpublished data). EPIMUL disperses effective spores (those generating sporulating infections) from a user-defined origin over a grid of compartments, each of which can be assigned different values for the number of infection sites, inoculum production per infection, degree of susceptibility to infection, latent period, infectious period, and lesion growth. EPIMUL uses a “boxcar technique” to move infected sites sequentially through unoccupied, latent, infectious, and removed categories (Teng 1985). Effective spores produced from each compartment are distributed over grid space on a daily time-step via the user defined dispersal function. We used a modified inverse power function (Mundt 1989; Mundt and Leonard 1985) because this function provides a more realistic

representation of primary disease gradients away from source plants in field experiments than an exponentially bound function (see Estimation of the WSR primary disease gradient at different spatial scales below). The modified inverse power function is

$$y = a * (x + c)^{-b} \quad (1)$$

where y = the number of effective spores, a is a constant proportional to the amount of source inoculum, x = the distance from the source lesion, c is a constant that allows for a finite amount of disease at the source, and b modifies the curve steepness.

Dispersive Waves of Disease Spread

We conducted straightforward field experiments to determine whether wheat stripe rust epidemics spread as traveling or dispersive waves (Cowger et al. 2005). Traveling waves driven by exponentially bound dispersal functions are expected to attain a constant rate of expansion quickly (Madden et al. 2007) (Fig. 2) with perhaps no buildup period at all when the basic reproduction number (R_0) is large (Frantzen and van den Bosch 2000), as it is for WSR. Alternatively, dispersive waves will have epidemic fronts that always increase in velocity, an emergent property of the disease gradient not being exponentially bound (Ferrandino 1993; Kot et al. 1996) (Fig. 2). “Epidemic front” has been defined somewhat differently among epidemiological studies. In some cases, the front has been based on the farthest locations at which the disease is detected

Traveling Wave Model

Dispersive Wave Model

Fig. 2. Conceptual representation of the traveling wave (top) and dispersive wave (bottom) models of epidemic spread (adapted from Frantzen and van den Bosch 2000). The traveling wave model is characterized by the constant velocity of spread at the invasion front and is generated by dispersal kernels that are bounded by an exponential (Ferrandino 1993). We have represented velocity (y-axis of the right-hand traveling wave figure) as a nearly straight line with slope = 0. This represents a scenario in which the basic reproductive number (R_0 , number of new infections/parent infection/generation in a completely susceptible population) is relatively high. If the R_0 values are low, the velocity of the invasion front increases with time until it plateaus, after which velocity remains constant (Frantzen and van den Bosch 2000). The dispersive wave model is characterized by an increasing velocity of spread at the invasion front, regardless of time. This form of epidemic spread arises when the disease gradients/dispersal kernels are heavy-tailed, and thus approach zero disease more gradually in the tails than is the case for an exponential function.

at a given point in time, with a front defined by joining points in different directions from the original outbreak. In other cases, the epidemic front has been based upon isopaths of a given level of disease prevalence. We generally measured the disease front based on the position of a given disease level along lines down the center of rectangular plots oriented roughly parallel with the predominant wind direction.

Field experiments consisted of two treatments replicated three times in each of three different environments. One treatment was a monoculture of a highly susceptible wheat cultivar and the other treatment was a mixture of the same susceptible cultivar mixed with a resistant one to slow the rate of epidemic spread (Mundt 2002). Differences in seed head morphology allowed us to follow epidemic spread on the susceptible genotype in both the monoculture and mixture, so we could directly compare the disease gradients in populations with relatively fast versus slow rates of spread. Long plots (7.6 m wide $\times$ 73 to 171 m long) and early growing season inoculations were used to ensure that epidemics spread over a sufficient time and distance to distinguish traveling from dispersive waves. It was necessary to track WSR spread in these long plots because shorter ones would truncate the disease gradient. Doing so could fail to capture the tail of the dispersal gradient, yielding dispersal kernels that appear to be exponentially bound and disease that spreads as a traveling wave, when in reality neither is the case. Truncating dispersal kernels is a common phenomenon in ecological studies that can misrepresent organism spread (Nathan et al. 2003; Tackenberg 2003).

Over the four to five latent periods of experimental WSR outbreaks, the epidemic fronts accelerated over space and time in upwind and downwind directions and in monocultures and mixtures (Fig. 3). We developed a simple model (Mundt et al. 2009a, b) to describe increasing epidemic velocity over space and time caused by pathogens with fat-tailed dispersal, under the assumption that the initial position of the epidemic front at the first measurement of spread (x_0) is proportional to the temporal rate of disease increase (r):

$$v_x = x/b \quad (2)$$

where v_x is velocity at distance x when velocity (distance/time) is expressed in time units of t/t_0 and t_0 = the time used to measure initial velocity (v_0), i.e., the first movement of disease outside of the outbreak focus. Equation 2 predicts that two epidemics with the same power law dispersal exponent (b) but different intrinsic growth rate (= apparent infection rate, r) will attain the same velocity at a given distance, but it will take the front of an epidemic with a lower r a longer time to arrive at that distance (Fig. 4). The position of the

Fig. 3. Mean (three replicates) of wheat stripe rust disease gradients produced from outbreaks (0 distance on the x-axis) in susceptible monocultures (**A**) and mixtures (**B**) in the upwind (negative x-axis values) and downwind (positive x-axis values) recorded at weekly intervals beginning after the first latent period (innermost gradient) (adapted from Cowger et al. 2005). Note the increasingly large gaps between disease prevalence lines as the epidemic progresses, which suggests a dispersive wave. Adapted from Cowger et al. (2005).

Fig. 4. Spread of the 30% disease prevalence isopath levels of wheat stripe rust on susceptible hosts in monocultures (black diamonds) and mixtures of susceptible and resistant hosts (white diamonds) from experimentally initiated outbreaks (adapted from Cowger et al. 2005). **A**, Advance of the 30% isopath over time away from the outbreak. **B**, Epidemic velocity – [distance (week n) – distance (week $n-1$)]/week – versus distance from the outbreak. The circled points in 4B show a similar epidemic velocity of 5 m/week at 10 m from the outbreak in monocultures and mixtures, but that it takes an extra two weeks for the 30% isopath to reach the same distance in mixtures (Fig. 4A).

epidemic front is predicted to increase exponentially with time t (Madden et al. 2007) and can be described (Mundt et al. 2009a, b) as

$$x_t = x_0(b/b-1)^{t/t_0} \quad (3)$$

where x_t = distance from the center of the focus to that of the epidemic front at time t , x_0 = the distance of the first movement of disease outside of the outbreak focus, b is the exponent (slope) of the modified inverse power law (Equation 1), and t_0 = the time at which x_0 is measured. The quantity $(b/b-1)$ approaches infinity as b approaches 1 (an infinitely shallow gradient), approaches 0 as b approaches infinity (an infinitely steep gradient), and is 2.0 for the common case of an inverse power law with $b = 2$.

Field data have been shown to conform to Equations 1 and 2 and to be repeatable over environments, in both monocultures and mixtures, downwind or upwind, and regardless of the disease prevalence level used to follow the epidemic front (Fig. 5). This increase in the velocity of disease spread over space and time strongly suggests that the WSR epidemics move across the landscape as a dispersive wave (Cowger et al. 2005; Mundt et al. 2009a).

It may seem esoteric to experimentally ascertain whether a plant disease spreads as a traveling or a dispersive wave. After all, epidemics can cause severe economic damage, regardless of the mode of spread. However, the effectiveness of disease control strategies is likely to differ depending on the pathogen's dispersal gradient (Tildesley et al. 2009, 2012).

Plant pathogens often have relatively high reproductive capacities and consequently increase rapidly, particularly at the epidemic front, where pathogens are least likely to be limited by host availability (Farber et al. 2017). If the dispersal gradients of innately explosive diseases have long tails that are not exponentially bound, disease can be dispersed over increasingly greater distances with very small increases in outbreak disease levels (Severns et al. 2015), potentially leading to continental scale spread (Brown and Hovmøller 2002).

Influence of Landscape Variables on Disease Spread

Once we established that WSR epidemics spread with increasing velocity, we investigated how different landscape features influence disease spread. In one set of experiments, we simultaneously evaluated the effects of focus area, host population size (plot size), and susceptible host spatial heterogeneity, by comparing factorial combinations of host population sizes with 1:3 width/length ratios (6.1 m × 18.3 m, 12.2 m × 36.6 m, and 24.4 m × 73.2 m), × three mixtures to generate spatial heterogeneity (100, 50, and 25% susceptible plants in mixture with resistant plants) inoculated with a small area focus (0.38 × 0.38 m), plus the largest plot sizes inoculated with a large area focus (1.52 × 1.52 m) (Mundt et al. 2011). Results were consistent among three field experiments conducted in different years; an example from one year is shown in Figure 6. Epidemic spread was faster when the foci were large and when mixtures contained a greater proportion of susceptible plants. By contrast, plot size had no detectable impact on epidemic spread. From a landscape perspective, a larger host area is expected to increase the epidemic magnitude owing to a greater incidence of disease build-up and pathogen dispersal from the increase in host abundance (Fleming et al. 1982; Waggoner 1962), though this may occur only when plot sizes are much smaller than those in our study (see literature summary in Sackett and Mundt 2009).

Most of our WSR field experiments used rectangular plots, sometimes narrow, to maximize space use and enable a greater number of replicates and treatments in each field. It was important, however, to determine whether the aspect ratio of experimental plots influenced the patterns of WSR spread. When we compared WSR spread from the same-sized foci in square plots (61 m × 61 m) and rectangular plots (6.1 m × 61 m), the square and rectangular plots produced epidemics that spread very similarly (Sackett and Mundt 2009). Not only did these results suggest that aspect ratio may not be as influential for epidemic spread as previously hypothesized (Fleming et al. 1982; Waggoner 1962), but also that plot shape is unlikely to confound our experimental results.

Different disease prevalence levels within foci of the same size should influence the subsequent spread of disease (van der Plank 1963; Waggoner 1962). Analytic relationships, under the assumptions of the scale invariance property of the power law and homogeneous host and environment, suggest that there may be a straightforward relationship between the initial prevalence of disease in the focus (P_0) and distance (x) of an epidemic front from that focus at a given point in time t (x_t):

$$x_t = aP_0^{1/b} \quad (4)$$

Fig. 5. Mean (three replicates) epidemic velocity of wheat stripe rust spread over upwind and downwind distance estimated from the 10, 20, 30, and 40% isopaths in susceptible monocultures and mixtures of susceptible and resistant hosts. **A**, Madras, Oregon 2001 experiment. **B**, Hermiston, Oregon 2002 experiment. **C**, Madras, Oregon 2003 experiment (adapted from Mundt et al. 2009a). In each case, the rate of disease spread always increases in velocity, never appearing to slow or level off, indicating that WSR is likely to spread as a dispersive wave.

where a is a constant that is proportional to the overall disease level at time t and b is the exponent of the inverse power function describing the disease gradient. If b is approximately 2, as is often the case for long distance dispersed pathogens (Ferrandino 1996; Gregory 1968; Harper 1977; Mundt 2009; Shaw et al. 2006; Waggoner 1983), then a plot of the front position against the square root of P_0 should be linear. Though we have not yet experimentally manipulated outbreak disease prevalence to evaluate this potential relationship, we analyzed the relationship between P_0 values (0.01 to 30%) from several different experiments (years 2000–2014) and the magnitude of

Fig. 6. Wheat stripe rust disease gradients at final disease assessment for experimentally initiated outbreaks in plots that varied in focus and plot area, in susceptible monocultures and mixtures of susceptible and resistant hosts. **A**, Main effect of ratio of susceptible and resistant plants (averaged over plot sizes). **B**, Main effect of focus size in large plots (averaged over all susceptible/resistant host frequency treatments). **C**, Lack of plot size effect when the focus size was held constant (averaged over all susceptible/resistant host frequency treatments). Adapted from Mundt et al. (2011).

disease at the final disease assessment (area under the disease gradient, AUDG) from all similarly planted plots with $1.52 \text{ m} \times 1.52 \text{ m}$ foci (Severns et al. 2015). Using EPIMUL, we projected the epidemic magnitude along a continuum spanning the range of outbreak disease prevalence values from our field experiments for 4.2 latent periods, the mean epidemic length at the last disease reading; experimental disease readings were terminated when plant senescence prevented collection of accurate prevalence estimates. This modeling suggested a simple power function (exponent of the power function = 0.59) that, when overlaid onto the scatterplot of WSR field experiment results, explained ~53% of the variation (Fig. 7A). Experimental plots were too short to contain a single disease prevalence level throughout all generations of the epidemics. We thus relied on simulations and the location of a single prevalence level (1%) recorded on a daily basis to evaluate the potential relationship between outbreak disease prevalence (P_0) and the extent of epidemic spread. Simulations similarly implied that a simple power function may describe the relationship between P_0 and the expanse of spread for the 1% isopath (Fig. 7B). The exponents (estimates of $1/b$) derived from the end epidemic magnitude (0.59) and spread distance (0.44) power functions suggest that a square root law may be useful for projecting the later patterns of WSR epidemic spread from outbreak disease levels.

Invasion ecologists have hypothesized that the successful colonization of an environment and the spread of invaders can be

Fig. 7. **A**, Predicted relationship between outbreak disease levels and epidemic magnitude (area under the disease gradient) of wheat stripe rust based on a modified version of the simulation model EPIMUL (Kampmeijer and Zadoks 1977). Wheat stripe rust disease spread simulations ran to 4.2 latent periods (solid line) superimposed on the scatterplot of experimental outbreak disease levels and epidemic magnitude at final disease assessment from 4.2 to 4.5 latent periods (mean 4.24 latent periods) from field studies in 2000–2014 (adapted from Severns et al. 2015). **B**, Predicted extent of the 1% isopath distance at 4.2 latent periods from WSR outbreak disease levels spanning the range of experimental outbreaks in Fig. 5A.

explained by outbreak propagule pressure (the number and size of propagules) and survival probability (reviewed by Simberloff 2009). Perhaps the apparent square root law association relating WSR outbreak disease prevalence to end epidemic magnitude is more broadly applicable to biological invasions by organisms with similarly long-tailed dispersal gradients.

Spatial Scaling Relationships

Scaling laws that govern biological systems are among the most fundamental and elusive aspects of study that lead to general biological predictions (Brockmann et al. 2006; Brown et al. 2004; Chave 2013; Enquist et al. 1998; Haskell et al. 2002; Levin 1992; Marquet et al. 2005; Milne 1998; Urban 2005). For epidemiologists, the scale invariance property of the power law unites the patterns of LDD disease spread in plants and animals (Brockmann et al. 2006; Eubank et al. 2004; Gisiger 2001; Jeger et al. 2007; Vespignani 2011). This property suggests that factors influencing disease spread at small scales may be the same factors influencing spread over greater spatial scales (Gisiger 2001). Below, we present evidence of scale-independent disease spread through three different approaches.

Epidemic patterns in historical epidemics. We found that models describing the plot level change in WSR velocity with distance from the focus and the change in position of the epidemic front over time (Equations 2 and 3) were also capable of describing the continental scale spread of other plant (potato late blight, southern corn leaf blight) and animal (West Nile virus, avian influenza H5N1) diseases characterized by long-distance dispersal (Table 1). Estimates of the inverse power law exponent (b) of WSR experimental epidemics and the continental scale spread of other LDD diseases ranged from 1.74 to 2.36 (Mundt et al. 2009a, b), despite more than five orders of magnitude difference in the geographic extent of disease spread. The mean b -value over all diseases was 1.98, conforming quite closely to epidemic spread predicted by the inverse square law (Mundt et al. 2009b). Similar inverse square spread was independently calculated for the North American West Nile Virus epidemic using phylogenies of virus sequence mutations to infer the rate of spread of the epidemic front (Pybus et al. 2012).

The inverse square law has been invoked broadly to describe the movement of wind-dispersed pathogens, pollen, and seeds (Ferrandino 1996; Gregory 1968; Harper 1977; Mundt et al. 2009a,b; Shaw et al. 2006; Waggoner 1983), and is what would be expected if most of the decline in transmission with distance from a source is driven primarily by simple dilution (geometric dispersion over increasing area of an agent originating from a point-source). Assuming that LDD diseases have similar epidemic spread properties that arise innately from the shape (kurtosis) of their disease gradient (Gisiger 2001; Lindström et al. 2011), the inverse square law (the amount of disease

is inversely proportional to the square of the distance from its source) could be a useful rule of thumb for approximating, reconstructing, and projecting epidemic spread for suspected but unverified LDD diseases.

Our experimental protocols and test environments for WSR were designed to provide favorable conditions for disease spread. Similarly, the continental-scale epidemics that we evaluated are from historical epidemics noted for their severity and fast movement. It is thus relevant to ask how the relationships described above would pertain to less optimal epidemics. Ojiambo et al. (2017) have recently conducted similar analyses for continental scale movement of cucurbit downy mildew in North America, capturing natural variation in spread patterns that occurred over seven sequential years. They found that epidemic spread comported with our model generally, but with greater variation in b -values of the power law that were estimated from the epidemics.

Estimation of the WSR primary disease gradient at different spatial scales. Characterizing the primary disease gradient is challenging, as relatively large sources of inoculum are required to track pathogen dispersal over large distances, but large inoculum sources also saturate infection sites locally, obliterating gradients close to the source (Lannou et al. 2008). To determine whether dispersal is similar over a broad range of spatial scales, we compared studies of primary disease gradients—the first generation of disease occurring outside of the focus—from single- and multilesion foci (in 1.52×1.52 m foci generated through uniform inoculations). The single lesion study focused on the distribution of first generation daughter lesions from 0 (source) to 1.5 m away from the source, while the primary disease gradient produced by 1.52×1.52 m foci was characterized from 0 to 91.4 m from the source. The primary disease gradients from single- and multilesion foci generated similarly shaped fat-tailed disease gradients (Farber et al. 2017; Sackett and Mundt 2005a) that are typical for LDD organisms (Clobert et al. 2012). Both disease gradients were well-described by the inverse power law function (Equation 1) (Farber et al. 2017; Sackett and Mundt 2005a) and had similar exponents. When we combined data from the single- and multilesion foci (normalized to a common sampling point at 0.91 m), the merged data were well-fit by a common gradient (Fig. 8).

Currently, our WSR disease spread data are limited to the areas within experimental fields, leaving unverified how the disease gradient scales across a broader landscape. However, a study of wheat stem rust (*P. graminis* f. sp. *tritici*) spread on the island of St. Croix may provide a suitable surrogate disease to assess this potential upscaling relationship. Kingsolver et al. (1984) tracked the spread of wheat stem rust from experimental foci on the pathogen-isolated island of St. Croix in the Caribbean Sea. Although WSR and wheat stem rust are different species, they share similar characteristics that

Table 1. Estimates of the exponent, b , of the power law function $y = a/x^b$ required to obtain observed patterns of disease spread

Epidemic	Maximum distance (km) ^a	Temporal regressions ^b			Spatial regressions ^d		
		b	LCL ^c	UCL	b	LCL	UCL
WSR, monoculture (Cowger et al. 2005)	0.032	1.96	1.78	2.21	1.75	1.56	1.98
WSR, mixture (Cowger et al. 2005)	0.009	2.00	1.80	2.29	1.93	1.61	2.41
Potato late blight 1845 (Bourke 1964)	700	2.01	1.64	2.81	2.36	2.11	2.67
Stem rust 1923 (Stakman and Harrar 1957)	2,180	1.95	1.93	1.96	1.86	1.75	1.99
Southern corn leaf blight 1970 (Moore 1970)	1,886	2.14	1.90	2.51	1.96	1.38	3.38
West Nile virus 1999-2002 (in Mundt et al. 2009a)	2,563	1.83	1.59	2.24	1.90	1.76	2.06
Avian influenza H5N1 2004–2006 (HEWSweb 2006)	9,329	1.74	1.56	2.01	2.05	1.82	2.36
Mean of all diseases		1.95			1.97		

^a Maximum distance of disease spread observed in each data set.

^b For temporal analyses, b was estimated as $e^s/e^s - 1$, where s is the slope of the regression of $\ln(x_t)$, position of the epidemic front) on time.

^c LCL and UCL are 95% lower and upper confidence limits of b back-calculated from standard errors of the regression slope.

^d For spatial analyses, b was estimated as the inverse of the slope of velocity regressed on distance.

would suggest similar patterns of spore dispersal. Urediniospores of both species are aerially dispersed, similar in size (15–32 µm) (Chen et al. 2014; Leonard and Szabo 2005), and lesions are produced in comparable positions within the wheat canopy (Farber 2016; Leonard and Szabo 2005). We were able to analyze primary disease gradient data to a 10.6 km distance in the 1957 data set of Kingsolver et al. (1984). The inverse power law provided a reasonable fit to the data when we included the combined WSR data sets with the four available, downwind, stem rust data points (Fig. 8). These data also showed that dispersal from a single initial WSR lesion could result in lesions 10 km distant (Table 2), though infection density would be so low as to be practically undetectable in the first generation of spread, an observation with important implications for eradication attempts (see Effects of control treatment timing and area below). Complementarity between the different WSR and stem rust disease gradients suggests that scaling over an even larger landscape may be possible, provided that the disease gradient is sufficiently characterized, and the primary mechanism driving dispersal is the same across spatial scales (Kristensen et al. 2013).

Scaling relationships with focus dimensions. Power law properties of the disease gradient suggest that the physical dimensions of the focus could scale with the patterns of epidemic spread. To address this possibility, we altered plot length, plot width, and focus

Fig. 8. Combined primary disease gradients from experimentally initiated single lesions (circles, from Farber et al. 2017), aggregate wheat stripe rust lesion outbreaks (diamonds, 1.52 × 1.52 m foci, from Sackett and Mundt 2005a) into susceptible monocultures, and wheat stem rust (triangles, 2.4 ha inoculated fields, from Kingsolver et al. 1984) fit by the modified inverse power function $y = a(x+c)^{-b}$. Adapted from Farber (2016).

Table 2. Mean wheat area estimated to contain a single stripe rust lesion after one generation of spread from a single lesion at distance zero (Farber 2016)

Distance from source lesion (m)	Mean area containing a single lesion in first generation of spread
0	0.4 cm ²
1	64 cm ²
2	0.03 m ²
8	0.7 m ²
32	19 m ²
500	1.4 ha
1,000	7.1 ha
2,000	37.3 ha
5,000	335 ha
10,000	1,762 ha

width four-fold and evaluated the resultant epidemics (Fig. 9). When the end-of-season disease levels were plotted versus distance from the focus in meters, it was clear that outbreaks from larger foci produced epidemics of greater magnitude than those originating from smaller foci (Fig. 10A, C, E). However, when we expressed the distance as multiples of the focus width, final assessment disease gradients resulting from small and large foci were nearly identical (Fig. 10B, D, F). Similar evidence of a scaling relationship occurred when plot size was held constant and focus dimensions were altered (Mundt and Sackett 2012), suggesting that focus size was the dominant variable influencing this scaling relationship. These results also support the square-root relationship between outbreak disease prevalence and distance of spread (Fig. 7), as the width of a square focus is also the square-root of the number of infections contained within the outbreak when disease density per unit area is constant, as was the case in our experiments. These results also are consistent with the work of Shaw et al. (2006), who concluded from a model of area sources that the median distance of aerial dispersal is proportional to the radius of a source field.

The focus area scaling relationship described above should be useful for comparing epidemics arising from different landscape dimensions. Such a scaling relationship may only apply to more local disease spread, as our experiments took place within relatively small agricultural fields (~18–20 ha). However, the scale invariance of the power law suggests the potential for the relationship to be conserved over greater spatial scales (Gisiger 2001; Marquet et al. 2005). We evaluated data from naturally occurring soybean rust epidemics during annual south-to-north spread in North America over a 7-year period (Mundt et al. 2013). The yearly maximum extent of soybean rust spread was highly correlated with area of the initial epidemic outbreak, supporting the scaling prediction of the power law. Ojiambo et al. (2017) recently reported similar results for cucurbit downy mildew in the U.S. They found significant correlations between current and ending epidemic extent as early as late April, despite the fact that cucurbit fields in the northern U.S. had not yet been planted at that time of year. Although this area-based scaling relationship is not commonly evaluated by epidemiologists and those studying biological invasions, it may have broad relevance.

Both focus dimensions and the amount of disease in the outbreak (P_0) appear to scale with end epidemic magnitude and expanse of disease spread. However, we do not yet understand precisely how focus dimensions and disease density interact, and if there are trade-offs between the two factors over the course of an epidemic. Fundamentally, the role that disease aggregation plays in influencing an epidemic must be key, but it still remains a poorly understood aspect of disease epidemiology (Brown and Bolker 2004; Leclerc et al. 2013; Mundt 2009) that deserves closer scrutiny.

Fig. 9. Schematic representation of the plot and focus size variable combinations to experimentally determine the scaling effects of plot variables on the patterns of epidemic spread (adapted from Mundt and Sackett 2012). Plots lengths, plots widths, and focus widths were varied four-fold.

Importance of Initial Epidemic Conditions

Results described thus far suggested a conceptual model in which the epidemiological pattern is determined primarily by the initial generation of disease spread, with subsequent pathogen reproduction being highly localized and largely independent of other sources of inoculum within the host population (see Conclusions). This

conceptual model posits that pathogen reproduction in the focus is the primary determinant of subsequent disease spread. Treating foci for disease control would be effective only very early in the first generation of pathogen reproduction, and local culling of asymptomatic plants around a focus (including the focus) would have an insignificant effect on disease spread past a treatment

Fig. 10. Mean wheat stripe rust gradients at final disease assessment for large foci/large plots and small foci/small plots in 2006 (A-B), 2007 (C-D), and 2008 (E-F). Distance from the focus in the left column is expressed in meters and for the same disease gradients the distance in the right column is expressed by the number of focus widths. Scaling by the focus width generated highly similar mean disease gradients even though the focus and plot dimensions differed four-fold (adapted from Mundt and Sackett 2012).

timing threshold. Below, we discuss field and simulation studies that test these predictions.

Pathogen reproduction in the focus. We conducted several field experiments to determine whether pathogen reproduction inside the focus could be the primary determinant of subsequent epidemic spread. One set of field experiments consisted of a full factorial, replicated combination of treatments where the focus (<1% of total plot area) was either a monoculture of a susceptible genotype or a 30:70 mixture (susceptible/resistant plants), and the remaining plot area (the at-risk population) was either a monoculture or a 30:70 mixture (Estep et al. 2014). Only the frequency of susceptible hosts in the focus had a significant effect on subsequent disease spread (Estep et al. 2014). A second field experiment had the same factorial arrangement and approach, but instead featured a susceptible monoculture and a partially resistant monoculture to alter pathogen reproduction (Severns et al. 2014). Again, the degree of host susceptibility in the focus explained the epidemic magnitude at the final disease assessment (Fig. 11). Results with mixtures in the second study (Severns et al. 2014) were not as clear as in the earlier one (Estep et al. 2014), which we believe was due to differential effects of take-all (*Gaeumannomyces graminis* var. *tritici*) on the susceptible club wheat cultivar (*Triticum aestivum* ssp. *compactum*) and the resistant common wheat cultivar (*T. aestivum*) (Severns et al. 2014).

The simplest possible explanation for the strong effect of outbreak host susceptibility on epidemic spread might be that spore production from the outbreak simply continued to provide the majority of the inoculum for new infections over generations (Aylor and Ferrandino 1989). This does not seem to be the case, however, as we found that halting spore production in the focus by applying a potent combination of fungicides and herbicide at 2.3 latent periods after inoculation had no statistically detectable effect on subsequent WSR spread compared with unsprayed plots (Severns et al. 2014). Our results instead suggest that a pattern of infection is established early in the first generation of spread from the focus, with subsequent disease increase by those founders being dominated strongly by local deposition and disease intensification (Aylor and Ferrandino 1989). WSR simulations suggested that a strong influence of the focus occurs only when local inoculum deposition is very high as compared with longer, more diffuse inoculum spread (Estep et al. 2014).

Effects of control treatment timing and area. Controlling an epidemic outbreak is an important and difficult challenge to overcome, especially for diseases characterized by long distance dispersal (Cunniffe et al. 2014; Dybiec et al. 2009; Filipe et al. 2012; Leclerc et al. 2013; Zeng and Luo 2008). Frequently, disease

escapes an insufficiently sized quarantine, ring cull, vaccination, and/or spray treatment, and spreads from the focus to the untreated, at-risk population (e.g., Keeling et al. 2001; Peterson et al. 2015; WHO Ebola Response Team 2014). Assuming the treated outbreak(s) are the only ones in existence, pathogen dispersal must exceed the expanse of the treatment area to escape containment. Usually, little is known about the relative effectiveness of area-based treatments and how large these containment/eradication areas must be to provide effective control (but see Cunniffe et al. 2014; Tildesley et al. 2010 for exceptions).

After exploratory WSR disease spread simulations suggested that earlier treatments might be more effective and efficient at controlling a WSR outbreak than a later treatment of an increased area, we conducted a full factorial experiment with three spray timings, 1.125, 1.25, and 1.5 latent periods postinoculation (estimated from a degree-day model), and three area treatments: focus only, 3 × 3 focus widths (including the centrally located focus), and 5 × 5 focus widths (including the focus). Each treatment combination was replicated four times, including four untreated foci (Severns et al., unpublished data). We also wrote a new spray subroutine in EPIMUL to evaluate whether the results from the cessation of spore production in the field experiment could be validated in silico.

Both the field experiments (Fig. 12) and EPIMUL simulations (Fig. 13) indicated that early treatments had a large effect in suppressing epidemics, but only if implemented early in the first generation of disease spread. Delaying treatment but compensating with a larger area appeared to have only a minor suppressive effect on the subsequent epidemic (Fig. 12). Our experimental plot dimensions limited the area we could spray, so we used simulations to expand the diameter of the spray up to 100 focal widths as a means to assess potential area and timing trade-offs that might occur at high (5%) and low (0.05%) outbreak prevalence levels. Simulations for both outbreak disease levels were largely consistent with the field experiments; they suggested that earlier treatments would be more effective than a delayed treatment over a larger area (Fig. 13). While an increase in the treatment area did reduce the total amount of disease to match that of early treatments (>90% total disease reduction at 100 focal widths), this level of disease reduction occurred because a large number of host plants were removed. Earlier treatments yielded similar levels of suppression with a much smaller culled area (Fig. 13), which would be more cost effective. Both our field experiments and simulations strongly suggest that earlier treatments are far more efficient at reducing the severity of an epidemic. They also support the conceptual model that disease spread becomes largely independent of the focus early in the epidemic.

Our studies also suggest that a WSR outbreak would essentially be impossible to eradicate. In simulations with a low R_0 of 4.0 (the WSR outbreaks in our field experiments had mean R_0 values of ~70), we found that, to have at least an 80% chance of eradicating WSR at 1.125 latent periods and at an initial prevalence level (P_0) of 1.2%, ring culls would need to exceed a radius of 120 focal widths (Severns et al., unpublished data). Wheat is an extensively grown crop with high host densities and a large number of potential infection sites per host. In contrast, other crops, natural plant populations, and systemically infecting pathogens of animals might have many fewer potential infection sites and, thus, a greater opportunity for successful eradication. For example, we calculated that there are as many potential infection sites for WSR in a 20-ha wheat field as there are humans on the entire planet. Effectiveness of control practices may also depend on initial outbreak levels (Fig. 13) and epidemic velocity. Interrelationships among pathogen reproduction rates, initial outbreak prevalence, host density, and disease control are currently under investigation in our research program.

Conclusions

In our studies of WSR epidemic spread, we focused on characterizing the long tails of the disease gradient and applying the scale invariance properties of the power law to understand the patterns of spread over local, regional, and continental scales, and identifying more efficacious approaches for epidemic mitigation. Initial studies

Fig. 11. Mean epidemic magnitude at final disease assessment (area under the disease gradient, AUDG) for all factorial combinations of partially resistant and susceptible hosts in the focus and at-risk populations (remainder of population outside of focus). Wheat stripe rust AUDG values are grouped by the focus cultivar, not the hosts in the at-risk population (data from Severns et al. 2014).

confirmed that the WSR epidemic front accelerates as a dispersive wave (Cowger et al. 2005), which is directly attributable to the long tails of the disease gradient (Clark et al. 2001; Ferrandino 1993; Hastings et al. 2005; Kot et al. 1996; Madden et al. 2007; Mundt et al. 2009a). The power law function used to describe the long-tailed WSR disease gradient appears to manifest via an approximation to an inverse square relationship. This relationship led to the development of a conceptual model of disease spread in which the epidemiological pattern is determined primarily by the initial generation of pathogen dispersal. Subsequent pathogen production occurs as highly localized infections with disease intensification and, especially at lower disease prevalence and long distances, the establishment of subfoci that arise through longer distance dispersal and stochastic events. Shortly following the initial outbreak, disease spreads independently from the more distant inoculum sources, as well as the focus. As the long tail of the disease gradient extends and becomes shallower with distance, the position of a given disease isopath accelerates from its previous position over time.

This conceptual model of epidemic spread resulted from the observation that the position of epidemic fronts over time and epidemic velocity over distance followed the same model for slow and fast epidemics, and regardless of spatial scale (i.e., all epidemics demonstrated similar slopes for these relationships, merely with different starting points). Furthermore, neither the size (area) nor aspect ratio of experimental field plots appeared to influence epidemic spread. We also found that host mixtures reduced the number of infections at a given distance from the focus, but that the local disease increase around an infection appeared to be similar once disease was established (see Figure 3 of Mundt 2014). Subsequent field and simulation studies (halting pathogen reproduction in the focus) confirmed that WSR epidemic spread was driven primarily by initial outbreak conditions.

Consistent with the “80/20” approximations (Pareto principle) that arise from well-established power law properties (Newman 2005), ~80% of sampled WSR lesions were present within the first 0.7 m surrounding a single WSR source lesion (Farber 2016), similar to that described for wheat leaf rust (Frezal et al. 2009). Earlier disease spread modeling suggested that the optimum ratio of short distance/long distance dispersal for plant pathogens should be

Fig. 13. Effect of treatment timing (1.125, 1.25, 1.5, 2.0 latent periods) and area (1× to 100× focus widths) on wheat stripe rust control at 0.05% (A) and 5.0% (B) outbreak disease levels. Disease control is expressed as a percentage of an untreated population, based on area under the disease gradient beyond the distance the largest control area (100 focus widths) for all treatments.

Fig. 12. A, Mean disease gradients at 5.0 latent periods (LP) for experimental combinations of spray timing 1.125 LP, 1.25 LP, and 1.5 LP by treatment areas 1× (focus only), 3× (three focus widths), and 5× (five focus widths). Mixed models indicated that treatment timing was the only statistically significant variable explaining the end of season area under the disease gradient differences. B, Wheat stripe rust disease spread simulation results (deterministic) for the same combination of treatments under approximate field conditions; basic reproduction number (R_0 , number of new infections/parent infection/generation in a completely susceptible population) = 70, LP = 5.0, infectious period = 16 days, and 1.2% outbreak disease levels. In these simulations, treatment timing had a much greater effect on disease suppression than an increase in treatment area, as was also the case in field plots (A).

approximately 80/20 (Zawolek and Zadoks 1992). Similarly, O'Hara and Brown (1996) determined that immigration of barley powdery mildew into experimental plots decreased from 100% to near zero soon after the first appearance of an infection within a plot. Despite this local build-up of disease, the kurtotic tails of power law functions account for the small proportion of inoculum that is dispersed long distances, which enables an initial epidemic pattern to be established over a wide area in the first generation of spread. For example, combining three data sets of cereal rust dispersal at different spatial scales (Fig. 8) resulted in the prediction that a single WSR lesion at distance 0 would result in one infection/1,800 ha at 10 km in the first generation of spread (Farber 2016).

The scaling relationships and patterns of epidemic WSR spread likely apply to similar LDD wind-dispersed plant pathogens and perhaps to LDD organisms in general. Sudden oak death (Peterson et al. 2015) and foot and mouth disease of livestock (Chis Ster and Ferguson 2007; Keeling et al. 2001) have similarly shaped disease gradients and are being evaluated in current collaborations on spread of diseases caused by LDD pathogens. An intriguing aspect is the apparently common dispersal gradient for taxonomically diverse hosts and pathogens with very different dispersal mechanisms. The plant pathogens we have investigated are dispersed primarily via wind. The LDD aspect of WNV H5N1 and avian influenza are both driven by long-distance migration of a diversity of infected bird species. For foot and mouth disease, LDD can be via aerial movement of fomites, as well as through the transport of infected livestock. A commonality among these disease systems is the movement of inoculum over very long distances. Such LDD results in substantial dilution of inoculum over space, which may dominate other aspects of dispersal, resulting in a classic inverse square relationship of distance to distance. It is also important to note that our analyses are based on averages over many potential dispersal events and are mostly useful for identifying general patterns of disease spread and general principles of disease control. In contrast, this approach may have much less value for making accurate, quantitative predictions of single dispersal events, for which detailed meteorological analyses (Aylor 2017) will often be required.

The epidemiological patterns discussed here should help predict how host susceptibility, host diversity, and the spatial heterogeneity of host diversity could influence epidemic spread in natural ecosystems. There is significant potential for designing managed ecosystems that may be more innately resilient to epidemic spread (Margosian et al. 2009; Mundt et al. 2011; Plantegenest et al. 2007). To date, our research suggests that eradication attempts are almost certain to fail unless conducted very early after epidemic initiation, and that treatment of asymptomatic hosts around an outbreak appears to have little or no effect on the spread of LDD pathogens, regardless of how extensive that effort might be (under realistic scenarios). The value of early intervention is hardly a new concept, but we were surprised at how soon after epidemic initiation control must be implemented, and how relatively unimportant the size of treatment zones is compared with timing. Further study of these trade-offs for WSR and other diseases should be of high value for designing more effective and efficient disease surveillance and control methods.

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Daniel H. Farber

Daniel Farber's research aims to combine investigations of theoretical questions relating to the ecological role pathogen dispersal plays in the spread of epidemics, and directly applied questions that can help decision makers forecast epidemic severity and take appropriate control measures. He is a computational epidemiologist focusing on describing aerial dispersal gradients using ecological statistics, and constructing mathematical models to predict the spread of plant diseases over time. In this vein, he has described the disease gradients of wheat stripe rust (*Puccinia striiformis* f. sp. *tritici*) from single leaf inoculations in a wheat field, and described the disease gradients of late blight of potato (*Phytophthora infestans*) from aerial photographs of infested fields. Additionally, he has investigated pathogenicity via greenhouse and growth chamber experiments; he has used cultural, molecular, and microscopy approaches to characterize a cryptic pathogen; and he has examined the efficacy of bio-control of a soilborne pathogen. Through these complementary projects, he aims to address questions that may involve all three sides of the disease triangle, and that may apply to a wide variety of ecological interactions.

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