AN ABSTRACT OF THE DISSERTATION OF

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Title: The Primary Disease Gradient of Wheat Stripe Rust (*Puccinia striiformis* f.sp. *tritici*) Across Spatial Scales

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Wheat stripe rust (WSR), also called yellow rust of wheat (*Tricitum spp.*), causal agent *Puccinia striiformis* f. sp. *tritici* (*Pst*), is a foliar disease of major economic importance on wheat, especially grown in temperate locations. WSR causes major losses of wheat yield, estimated at nearly $ 1 billion per year, and has been found in upwards of sixty countries throughout the world, particularly in temperate regions. WSR, like many diseases of widely planted agricultural crops, has been managed largely by breeding resistant cultivars of wheat and by applying fungicide, but these strategies have resulted in periodic intensifications of WSR epidemics due to *Pst* mutations overcoming resistance or becoming resistant to fungicides. However, these strategies can be augmented to control WSR and other plant diseases by understanding the epidemiology, including the dispersal patterns of their causal pathogens, factors affecting the pathogen's infection efficiency and reproduction rate, allowing managers to make

better informed decisions. In this dissertation, I have examined the spread of WSR, bridging theoretical dispersal and disease ecology with plant epidemiology to provide analyses that have direct applicability to managing the spread of diseases caused by foliar wind-dispersed plant pathogens, as well as increase our understanding of basic biological processes.

To investigate the dispersal gradient of *Pst,* I inoculated single wheat leaves with *Pst* urediniospores, and sampled all wheat leaves within two intersecting 0.3 m × 3.0 m transects in eight replicates over three years. The lesions observed on each of the top three leaves on plants within 1.5 m from the source lesion were three-dimensionally mapped. The total number of lesions within a 1.5 m radius was estimated by dividing the number of lesions observed within each 0.025 m-wide annulus by the fraction of the annulus sampled. The estimated total number of lesions produced within 1.5 m of a single source lesion ranged from 27 to 776 with a mean of 288 lesions. Eighty percent of the lesions were recorded within 0.69 m of the source infection. The proportion of total lesions observed at a given distance from the source was fit well by the Lomax and Weibull distributions, reflecting the large proportion of lesions arising close to the source, and when fit to a modified inverse-power distribution had a slope (*b*) of 2.5. There were more lesions produced on leaves higher in the canopy than on lower leaves, with more lesions being detected above than below the point of inoculation. Simultaneous measurement of lesion gradients and spore dispersal in the final year of the study suggests that this pattern is owing to greater susceptibility of upper leaves, rather than increased dispersal to upper leaves.

In addition to dispersal of pathogen propagules, disease spread requires successful infection of host tissue. In plant disease epidemiology, susceptibility of host tissue is often assumed to be constant. This assumption ignores changes in host phenology due to developmental stage. To examine relative

susceptibility of wheat leaves of different ages and leaf positions, 3-, 4-, and 5- wk old wheat plants were inoculated with equal quantities of urediniospores of *Puccinia striiformis* f.sp. *tritici,* the causal agent of wheat stripe rust (WSR). Disease severity on each leaf was assessed and fit by mixed effect linear model as function of leaf position and plant age. Younger plants had significantly greater disease severity than older plants, with mean severities of 50.4%, 30.1%, and 12.9% on plants that were 3 wk, 4 wk, and 5 wk old at time of inoculation, respectively. This effect was greater on leaves higher on the plant. Within same-aged plants, younger leaves had significantly greater disease severity than older leaves, with mean severities of 40.2%, 34.8%, and 17.7% on the uppermost, second, and third leaf, respectively. These results corroborate field data suggesting the vertical distribution of lesions was due more to differences in host susceptibility than to propagule dispersal.

The results of the primary dispersal gradient of *Pst* study were contextualized with previous studies of dispersal of cereal rusts by investigating their dispersal across spatial scales. This was accomplished by combining the local dispersal dataset with a previous *Pst* dispersal dataset across agricultural fields out to 91.4 m. These datasets were normalized by the mean number of infections observed per plant at 0.914 m from the center of the disease focus after a single generation of spread. These datasets were well-fit by a single modified inverse power function, $y = 1.17 * (x + 0.10\ m)^{-2.24}$. This function was then used to combine the local and fieldwide datasets with a primary dispersal dataset of *Puccinia graminis* f.sp. *tritici (Pgt)* out to 10.62 km, by predicting the number of lesions per plant at 2730 m from the disease source, to which all observations in the *Pgt* regional dataset were normalized, which was well fit by $y = 1.26 * (x + 0.14\ m)^{-2.39}$. I created a susceptible-latent-infectious-removed (SLIR) compartmental time-step model to assess disease spread over time across spatial scales. The modified inverse-power function fit to the combined dispersal data across all three spatial scales was

used to seed the dispersal kernel, comparing epidemics stemming from initial foci of 1.52 m with compartment sizes of 1.52 m and 0.025 m, and comparing epidemics stemming from initial foci of 152 m with compartments size of 152 m and 1.52 m. The resulting models produced very similar disease levels, as quantified by taking the area under the dispersal curve after seven generations of spread.

In this dissertation, I have examined the spread *Pst,* as a model system to better understand the spread of diseases caused by aerially-dispersed pathogens. This research will do much to fill in gaps in the literature on empirical measures of pathogen dispersal and infection, and the theoretical ramifications of modelling disease spread incorporating multiple spatial scales. It is the aim of this research to be applicable to a wide range of systems exhibiting similar attributes, particularly in regards to dispersal, in addition to aiding our understanding of Pst and other cereal rusts.

The Primary Disease Gradient of Wheat Stripe Rust (*Puccinia striiformis* f.sp. *tritici*) Across Spatial Scales

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I understand that my dissertation will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my dissertation to any reader upon request.

Daniel Farber, Author

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CONTRIBUTION OF AUTHORS

Dr. Christopher Mundt provided intellectual contributions to all chapters, and assisted in editing and data analysis. Dr. Jan Medlock assisted in analyzing dispersal data using probability density functions in chapter 2. Dr. Patrick De Leenheer assisted in building a mechanistic model of disease spread over time.

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Chapter 1 - Introduction

Economic Importance of Stripe Rust of Wheat

Wheat stripe rust (WSR), also called yellow rust of wheat (*Tricitum spp.*), causal agent *Puccinia striiformis* f.sp. *tritici* (*Pst*), is a foliar disease of major economic importance on wheat, especially when grown in temperate locations. Wheat is a widely planted crop throughout the world, with an estimated 734.8 million metric tons annual global production (Foreign Agricultural Service 2016). Domestically, 2012/2013 wheat production was 2.267 billion bushels (U.S. Department of Agriculture 2013). Stripe rust causes major losses of wheat yield. It has been found in upwards of sixty countries throughout the world (Chen 2005a). Losses of up to 100% of a field have been recorded domestically when the initial infection occurred very early in the season on particularly susceptible races of wheat (Chen 2005a). Epidemics have occurred throughout temperate wheat-growing regions. WSR is most common in cooler wheat growing regions, being most common in the Pacific Northwest domestically (Wellings 2011). Epidemic levels of WSR are more common on winter wheat, which is planted in the fall, giving more time for WSR to establish itself compared to spring wheat. Epidemics have grown significantly in extent and range over the last half-century, with over 88% of wheat now susceptible to WSR, and losses estimated at 5.47 million tons worth nearly $1 billion (Beddow et al. 2015). In addition, significant costs are incurred through application of fungicides, and opportunity costs in breeding programs occur owing to efforts required to breed for resistance to this disease.

Pst Biology

Rusts are some of the most economically important and most researched fungal pathogens of plants (Dean et al. 2012), and have created problems in cereal production since the Neolithic Revolution

in Mesopotamia over 10,000 years ago (Leonard and Szabo 2005). Stem rust, *Puccinia graminis*, the causal agent of wheat stem rust, is one of the earliest pathogens to be formally studied, and is one of the best understood pathogens in existence (Peterson 2001). Rusts in general have some of the most complex life cycles of any taxa. Their life cycles can include several stages, and both a sexual and an asexual cycle. Rusts are obligate biotrophs, meaning that they are only able to complete their life cycles on living tissues in nature, although there have been some successes culturing some stages of rusts on enriched media in laboratory experiments (Staples 2000). *Pst* is a macrocyclic, heteroecious rust, meaning it is capable of producing five life stages, which occur on two separate hosts: in its primary, sexual cycle, haploid basidia infect the alternate host, barberry (*Berberis spp.*), which then develop haploid pycnia (also referred to as spermagonia). The pycnia produce pycniospores, which disperse and cross-fertilize with receptive hyphae to form the dikaryon, with two unfused nuclei in each cell. The hyphae grow through the barberry, producing the dikaryotic aecia, from which aeciospores are dispersed. Aeciospores can only infect the primary host, wheat, which then develop into telia, the site of karyogamy, producing diploid teliospores, from which germinate basidia, the site of meiosis, to complete the cycle. In the secondary, asexual cycle, the aeciospores germinate into dikaryotic uredinia, which produce urediniospores capable of reinfecting wheat, and developing into new uredinia, completing the cycle (Hovmøller et al. 2011). The alternate host was only recently identified (Jin, Szabo, and Carson 2010).In the Pacific Northwest, only the asexual uredinial stage is of importance in contributing to the disease, and the sexual stages of the pathogen have rarely ever been observed (Wang and Chen 2015) in agricultural settings.

Despite the extreme paucity of sexual reproduction of *Pst* and therefore the near total absence of sexual recombination, the pathogen has been shown to be capable of quick genetic changes. In

comparing isolates of *Pst* from California from 1980 and post-2000, there was less than 50% genetic similarity (Milus, Kristensen, and Hovmøller 2009). The rapid nature of these genetic changes appears to be largely due to the massive proliferation of transposable elements, DNA strands that can move their relative position within a genome (known as self-transposing). Transposable elements have been shown to comprise 17.8% of contig sequences; however this number is probably considerably lower than the actual representation of transposable elements in the genome, as sequences with similar repeats are often assembled into common contigs, thereby masking many transposable elements (Cantu et al. 2011). Transposable elements were found to make up nearly 45% of the *P. graminis* f.sp. *tritici* genome (Duplessis et al. 2011), and it would be reasonable to believe the *Pst* genome contains a fairly similar percentage of transposable elements, as it has been shown that all *Pucinia* species infecting grasses form a closely related cluster (Grasso et al. 2006). These transposable elements are sections of the genome hypothesized to be derived by horizontal gene transfer, which allow for significant rearrangements of pathogen genomes without requiring sexual recombination, and which have been shown to contain high concentrations of effector genes, which encode cysteine-rich secreted proteins used by microbes in pathogenesis (Shawkat Ali et al. 2014; Stergiopoulos and de Wit 2009).

The high rate of asexual evolution of *Pst* likely plays a significant role in the pathogen's ability to overcome genetic resistance in wheat (see 'Management of WSR'), as the population structure of *Pst* has been shown to change dramatically within 2-3 years (Bayles et al. 2001), with these changes often including losing redundant effectors (Enjalbert et al. 2005), which can be allow a population of *Pst* that was once recognized by the host's plant defenses, to cause disease. Using transcriptomics, more than 400 genes encoding secreted proteins were discovered in the haustoria, over two-thirds of which exhibited upregulated expression compared with germinated spores, and 94 candidate effectors genes

were found, targeting several compounds present in wheat cells (Garnica et al. 2013). The fields of genomics, transcriptomics, and biochemistry of fungal effectors has been burgeoning in recent years, with several reviews that have examined the various classes of pathogen effectors and their mechanisms in allowing for pathogenesis (Boch, Bonas, and Lahaye 2014; Lo Presti et al. 2015; Stergiopoulos and de Wit 2009), including reviews focused on the effectors of biotrophic fungi (Koeck, Hardham, and Dodds 2011; Yin and Hulbert 2011).

Pst Races

Pst is capable of rapid mutation, creating new races with differential abilities to cause disease (Van der Plank 1969), with 55 races of *Pst* identified in a study from 2010 to 2012 (Wan, Chen, and Yuen 2015). This is in addition to a previous longer-term study which identified 115 races with diverse virulences in North America from 2000 to 2007 (Chen et al. 2010). Similar levels of diversification of *Pst* have been found in temperate wheat growing regions throughout the globe, including in India (Prashar et al. 2007), China (Liu et al. 2011), Chile (Becerra et al. 2007), France (de Vallavieille-Pope et al. 2012), and Australia (Loladze, Druml, and Wellings 2014). A worldwide analysis using microsatellite markers found a center of heterogeneity with strong evidence for recombination in *Pst* populations in central Asia, and a largely clonal population elsewhere, suggesting central Asia is the original location from which *Pst* spread (Sajid Ali et al. 2014).

The diversity of *Pst* races was correlated with the agronomic practices and environmental conditions of each region, finding increased diversity in regions with large areas of susceptible hosts, particularly in areas with low genetic diversity of those hosts. Areas in which wheat with durable resistance to WSR were planted (Hou et al. 2015) may have reduced rates of evolution to overcome resistance (Chen et al. 2009). Proximity to overwintering locations (Duan et al. 2010), and potentially to

areas in which a susceptible alternate host is present (Zhao et al. 2013) could increase rates of *Pst* evolution, although that has not been demonstrated in the Pacific Northwest (Wang and Chen 2015).

The tools used to identify these races continue to grow and evolve, despite the challenges of not being able to culture *Pst.* The tools include the use of differential cultivars, in which races of *Pst* are identified by the pattern of cultivars of wheat on which they are able to cause disease (Calonnec, Johnson, and De VALLAVIEILLE-POPE 1997; Chen 2005a); amplified fragment length polymorphisms (AFLPs) and random amplified polymorphic DNA, polymerase chain reaction (PCR)-based assays to differentiate races based on the presence of polymorphisms in the DNA (Savelkoul et al. 1999; Steele et al. 2001); and more recently using transcriptomics (Hubbard et al. 2015), and genotyping by sequencing (Ali et al. 2011). The full genome of *Pst* has been sequenced (Cantu et al. 2011), which has allowed for identification of such polymorphisms as expression of candidate effector genes (Cantu et al. 2013).

Conditions Promoting Disease

Infection by *Pst* uredinia on *T. aestivum* involves several specialized structures and processes, as described above, which require conducive environments. WSR is a temperate disease with specific environmental conditions necessary to promote infection of wheat by urediniospores. The uredinial infection process begins when an urediniospore lands on a susceptible wheat or closely related grass leaf with free moisture and air temperature ranging from 0 to 25 °C for at least 3 hr (Chen et al. 2014; Chen 2005b), although newer races of *Pst* have been found to be more aggressive in warmer conditions (Milus, Kristensen, and Hovmøller 2009). The urediniospore produces a germ tube, which develops into an haustorium, which is able to penetrate the stomata of the wheat leaves. Urediniospores of *Pst* and *P. graminis* have been shown to be very sensitive to UV radiation and air pollution (Aylor 2003; Chen et al. 2014).

The phenology, defined as the change in physiology over time (Lieth 2013), of a host or host tissues can have significant impacts on the spread of its pathogen population. This can be due to changes in host susceptibility to attacks, particularly herbivory or infection over time, which could be correlated with production of certain defensive tissues like cuticles (Khajuria et al., 2013), or inversely correlated with production of certain vulnerable tissues, such as the case for strawberries becoming more susceptible to *Botrytis cinnerea* when in bloom (Boyd-Wilson et al. 2013). The effects these phenological changes can have on susceptibility to infection can be heavily influenced by the life strategy of the pathogen, as older plants have shown greater susceptibility to necrotrophic pathogens (Dolar 1997), which consume dead material, whereas the requirement for biotrophic pathogens to complete their lifecycle on healthy host tissue would suggest a negative relationship between susceptibility and senescence (Schulze-Lefert and Panstruga 2003). As *Pst* is a biotrophic pathogen, it may be expected that wheat would have some age-related resistance (ARR) (Panter and Jones 2002) to it, such as high-temperature adult plant resistance (HTAP) (discussed further below) (Qayoum and Line 1985).

Management of Stripe Rust of Wheat

Effective management of diseases can be informed by an understanding of its epidemiology (Gilligan 2002; Merz and Falloon 2008). Due to WSR being a polycyclic disease, in which urediniospores are aerially dispersed largely very close to the source, but with significant dispersal very far from their source lesion, a small amount of disease early in a growing season can lead to a severe epidemic (Estep, Sackett, and Mundt 2014; Severns et al. 2014). In other rusts, urediniospores have been found to travel upwards of 800 kilometers (Hermansen, Torp, and Prahm 1976). This long-distance dispersal makes effective quarantines nearly impossible.

Due to the ability of WSR to accelerate quickly, to cause significant losses, and to be able to invade remote areas, early and regular scouting is recommended to allow field managers to make informed decisions to combat WSR (Gaunt and Cole 1992), along with use of trap nurseries (Duveiller, Singh, and Nicol 2007). Once disease is established in a stand of susceptible hosts, such as an agricultural field, given a conducive environment, WSR will rapidly accelerate if not managed. However, fungicides, most notably those with some degree of systemic or translaminar action – e.g., strobilurins and triazoles (Conner and Kuzyk 1988) - have shown some reduction in WSR epidemics and increase in grain yield over untreated controls, although these increase in yields were generally under 50% and did not significantly increase with two sprays relative to one (Sharma et al. 2016). Overuse of fungicides can also lead to fungicide resistance (Hahn 2014; Russell 1995), reducing their effectiveness in the long term. Therefore, fungicide use is best thought of as a tool to augment other management strategies, particularly breeding for resistance, in an integrated pest management approach (IPM) (Chen 2007; Mundt, Cowger, and Garrett 2002).

Resistance to plant pathogens has been largely grouped into two principle classes: major gene, also termed vertical or R gene resistance, in which the infected cell of a host recognizes a specific effector compound secreted by the pathogen, causing a chemical pathway leading to a hypersensitive response (HR), also termed programmed cell death, killing the plant cell along with the attacking pathogen (del Pozo and Lam 1998; Greenberg 1996; Williams 1994); and minor gene, also known as quantitative or horizontal resistance, in which a plant exhibits certain traits which reduce the ability of a pathogen to cause disease but do not eliminate it (Nelson 1978; Niks, Qi, and Marcel 2015; Poland et al. 2009). Both of these forms of resistance are present in the wheat genome (Lewellen, Sharp, and Hehn 1967), and have been actively bred to increase the level of and durability of resistance (Hou et al. 2015).

Due to single R genes controlling most major gene forms of resistance, it has been much easier for *Pst* to overcome resistance, particularly in fields with high degrees of selection pressure resulting from extensive use of genotypes containing the same single R gene (Ellis et al. 2014), although a pyramiding multiple R genes into a cultivar has been employed to reduce the probability of pathogens overcoming resistance (Castro et al. 2003; Mundt 1991). Quantitative resistance, therefore, is considered generally more durable, and has been the focus of many breeding efforts involving quantitative trait loci (QTL) identification (Hou et al. 2015; Li et al. 2010; Liu et al. 2015), which can also be pyramided (Richardson et al. 2006). A particularly widely deployed form of quantitative resistance to WSR is HTAP (high temperature adult plant resistance) (Bux, Ashraf, and Chen 2012; Uauy et al. 2005), in which the *Yr36* gene drastically reduces WSR epidemics in mature plants in warm temperatures. HTAP is less race-specific than R gene resistance, and therefore has led to greater durability (Chen et al. 2002). In addition to resistance breeding and fungicide applications, cultural methods can reduce WSR epidemics, such as planting at a later date in the fall to reduce the probability of early establishment of WSR, refraining from over-fertilization with nitrogen-based fertilizers, removing nearby refuges for *Pst*, such as susceptible grasses and volunteer wheat, avoiding overwatering, particularly when conditions are conducive to infection, and reducing connectivity of susceptible host populations including use of multiline cultivars (Mundt 2002; Neumann et al. 2004; Papaïx et al. 2014; Reddy 2013; Roelfs, Singh, and Saari 1992; Stevens 1960).

Spread of Aerially Dispersed Pathogens

The amount of disease created from a single lesion over a single day is measured as the daily multiplication factor (DMFR) (Lannou and Mundt 1996a), which when grouped together over the course an entire generation, is the reproduction rate, or R_0 (Breban, Vardavas, and Blower 2007). The R_0 is

affected by all three sides of the disease triangle – host, pathogen, and environment – as it is sensitive to changes in infection biology, growth, and dispersal. The disease gradient, the proportion of total observed progeny infections arising as a function of the location and distance relative to the inoculum source, can be thought of as a synthesis of the dispersal gradient, the proportion of total depositions of propagules as a function of the location and distance relative to the inoculum source, and infection biology (Fitt, McCartney, and West 2006; Fitt et al. 1987). The dispersal gradient is a measure of where spores land, which in turn affects the probability that a given spore will develop into a lesion, as only spores which land on healthy tissue of susceptible hosts lacking sporulating or latent lesions can develop into lesions.

Autoinfection, the reinfection of a host by a pathogen previously infecting said host, is an important aspect of the pathogen population biology and epidemiology, including in animal diseases (Zimmermann 2014). This process is in opposition to alloinfection, in which new infections occur on hosts other than the host of the previous generation's infection (Mundt 2009). Autoinfection rates of WSR are not yet known with confidence, but other wheat rust diseases, such as wheat leaf rust (*Puccinia triticana*) exhibit autoinfection rates well over 50% (Lannou et al. 2008). The rate of autoinfection affects the overall spread of a disease due to the rapid saturation of host plant tissue. This, coupled with the 'fat-tailed' distribution exhibited by many aerially dispersed pathogens, including WSR (Brown and Hovmøller 2002; Mundt and Sackett 2012; Sackett and Mundt 2005a) and its relatively short generation time, predicts higher severity levels further from the source of infection more quickly than with lower autoinfection rates over successive generations, caused by acceleration of the spread of disease (Kot, Lewis, and Driessche 1996). The high rate of autoinfection suggests that once WSR is established within

a field, a significant portion of newly arising infections on leaves higher in the canopy may come from source lesions within the same plant on lower leaves.

The autoinfection rate and the associated steepness of the dispersal gradient can have significant implications for the size of the dilution effect, in which inoculum is diluted by landing on less fit or unfit host tissue (Keesing, Holt, and Ostfeld 2006). This term was coined by Ostfeld and Keesing to describe how a relative decrease in population of white-footed mouse via an increase in biodiversity and/or species evenness would result in a decrease in the number of ticks infected with lyme disease (Ostfeld and Keesing 2000). A decrease in relative abundance of a susceptible host genotype will increase the dilution of inoculum, as a greater portion of propagules would be likely to land on less fit or unfit host tissue, resulting in fewer new infections, and fewer sources of inoculum in the following generation, when compared with the spread of WSR through a single host genotype field. This same mechanism has been cited as a tool in reducing the spread of plant pathogen populations in fields comprised of multiple host genotypes with varying degrees of susceptibility infection relative to homogenous fields (Lannou and Mundt 1996a; Papaïx et al. 2014; Zimmermann 2014).

Pst urediniospores are dispersed via two primary mechanisms: carried aerially by wind or splash-dispersed by rain (Geagea, Huber, and Sache 1999). Splash dispersal can occur as a result of raindrops or irrigation droplets hitting sporulating lesions on leaf surfaces, carrying urediniospores in the water and depositing them on other susceptible host tissue. Of these two methods, aerial dispersal has received considerably more attention and is responsible for nearly all long-distance dispersal events, whereas splash dispersal is limited to a very local scale (Sache 2000). There are additional interactions between splash dispersal and aerial dispersal, as impaction of raindrops on leaves can release additional spores relative to dry dispersal (Ahimera et al. 2004). Due to the mucilaginous covering of *Uromyces viciae-*

fabae urediniospores, they have been reported to often be aerially dispersed in clusters rather than solely (Clement et al. 1994), which likely also applies to *Pst*.

The primary disease gradient of WSR has been quantified at the fieldwide scale out to 91 m (Sackett and Mundt 2005a). The rate of success of the initial inoculum had a large impact on the amount of disease observed outside the focus, as well as on the shape of the dispersal gradient, and has been shown to have a large effect on the overall severity of the epidemic (Severns, Sackett, and Mundt 2015). WSR's disease gradients are best represented by the modified inverse power model (Mundt and Leonard 1985), a modified form of the model put forth by Gregory (Gregory 1968). This model follows the form of $Y = a * (x + c)^{-b}$, in which Y represents the amount of disease, and a, b, and c are parameters. To find the a and b parameters, the graph can be linearized by taking the log of both sides of the equation, giving $ln(Y) = ln(a) - \ln(a) - b * ln(x + c)$.The parameter *a* is the intercept and *b* is the slope. The *c* value is an offset approximating the amount of space occupied by the initial inoculum. The incorporation of the c value also has the benefit of allowing the linearized form to be defined at $x = 0$. c can be found by iteration, increasing its value by an arbitrary small value, log-transforming, and fitting the linear regression model. In this method, the log-transformed linear model with the c value in which the linear regression model had the highest R^2 value is accepted.

There are some importance differences between the inverse power model and the exponential model of dispersal (Reynolds 2011). Very close to the source, the inverse power model predicts a much steeper decrease with increasing distance than the exponential model, but further away from the source of the of disease, the inverse power model predicts a higher amount of disease than the exponential model. Because the inverse power model's rate of decrease decreases quicker than the exponential model, it both predicts a higher percentage of its progeny lesions to occur very close to the

source compared to the exponential model and a higher percentage of its new lesions to be very far from the source compared to the exponential model. For this reason, inverse-power distributions are said to have 'fat tails' (Hovestadt, Messner, and Poethke 2001; Petrovskii, Morozov, and Li 2008).

Dispersal gradients of *Pst* can act as a model system for passive aerial dispersal of small particles, such as pollen grains, spores, or mineral dusts. It has been shown through atmospheric physical simulation that particles smaller than 60 µm in diameter are capable of long-distance aerial dispersal following 'fat-tailed' distributions, with the probability of these long-distance dispersal events increasing as particle diameter decreases (Wilkinson et al. 2012). For particles of spore or pollen grain diameters, turbulence is an important factor in the shape of the dispersal plume, and they must escape the boundary layer to enter the wind current for long-distance dispersal (Chamecki and Meneveau 2011). The physics of this and the transport of such particles has been measured empirically (Chamecki, Dufault, and Isard 2012; Kelly, Troen, and Jørgensen 2014; Novak et al. 2000; Rieux et al. 2014) and has been examined theoretically, frequently with the use of lagrangian stochastic models (Boehm and Aylor 2005; Gleicher et al. 2014; Wilson and Sawford 1996).

Epidemic Modeling

Modeling the dispersal of pathogen propagules can be very useful for predicting where and when disease outbreaks are likely to occur, which can benefit managers of crops in their attempts to control epidemics and limit invasions (Aylor 1999; Jeger et al. 2007). Due to the complex physical and biological processes acting upon aerially dispersed propagules and the difficulty of measuring and quantifying these processes (Nathan 2001), it can be useful to examine the disease gradients of resulting successful infections by pathogen propagules, as they are generally sessile, and often more visible than the propagules themselves, as a proxy for propagule dispersal (Shaw 2006).

The first mechanistic model of plant disease spread was proposed by van der Plank (Van der Plank 1963), which utilized a delay-differential equation. This model is rarely used, largely due to the difficulty in analyzing such equations. Discrete-time approximations, however, have been more widely incorporated into contemporary models of population growth, which include but are not limited to plant pathogens (Allen, Lou, and Nevai 2008; Kot and Schaffer 1986; Neubert, Kot, and Lewis 1995; Zhou and Kot 2011). Many of these models are also spatially discrete, in which disease is calculated within a defined interval, area, or volume (Cunniffe et al. 2011; Gilligan and van den Bosch 2008; Yakubu and Fogarty 2006).

Compartmental models have been used to predict the fraction of a population at various stages of disease. A susceptible-infected-removed (SIR) model was developed to describe the spread of cholera in a human population (Kermack and McKendrick 1927), in which the entire population was considered to be the sum of the fraction which was susceptible to the disease but had not been infected, the fraction which was infected with disease and capable of transmitting it to others, and the removed fraction, which had been infected but was no longer capable of infecting others. This model was augmented with a fourth compartment, termed exposed for animal populations (Samanlioglu, Bilge, and Ergonul 2012), in which the fraction of the population had been infected with the disease but were not contagious. In botanical epidemiology, this compartment is often termed latently infected, giving the susceptible-latent-infected-removed (SLIR) model (Segarra, Jeger, and van den Bosch 2001).

The epidemic modeling computer simulation EPIMUL (Kampmeijer and Zadoks 1977) has been used to predict the severity of disease over time throughout a growing season by inputting parameters describing the spatial scale of the field, the spatial arrangement and biology of the hosts and pathogens, and the length of the epidemic in days. The field is an array comprised of compartments of user-defined

size containing either susceptible hosts or compartments in which no infections can occur, which could represent either resistant plants or bare ground. The parameters describing the hosts include the number of plants per compartment, the maximum number of infections per compartment, and the extent of any partial resistance (Lannou and Mundt 1996a). The pathogen is described by the amount and location or locations of initial inoculum, daily multiplication rate of the pathogen, autoinfection rate, rate of growth of individual lesions, and dispersal function, which can be isotropic or anisotropic (Soubeyrand et al. 2007), and can follow a Gaussian distribution, a modified power law distribution, or a Lambert (Lambert, Villareal, and Mackenzie 1980) model.

An aspect of disease spread models including compartmental models is spatial scale, in both the extent of the model and the size of each compartment, or its resolution (Willocquet and Savary 2004). The effects of changing spatial scale are not limited to plant pathology, but have been of great interest in many aspects of ecology, affecting biodiversity measures (Chase and Leibold 2002), population synchrony, the changes of multiple co-occurring population densities in time (Mortelliti et al. 2015), and reproduction potential (Mikaberidze et al., 2014). Spatial scale may also have significant impacts on dispersal (Cadotte and Fukami 2005; C. C. Mundt et al. 2009).

Dispersal has been measured at differing scales, but prior to this study, the same system has not been examined at differing spatial scales. Here, I investigate primary disease spread caused by aerially dispersal of pathogen propagules, using WSR caused by *Pst* as a model system. I have described the primary local disease gradient of WSR empirically, adding one of relatively few studies quantifying dispersal from a single source infection. This included three-dimensional analyses of uredinia and urediniospore dispersal. I investigated the relationship between dispersal of *Pst* urediniospores and the resulting uredinial infections as a function of wheat age-induced phenological changes, addressing

hypotheses derived from the empirical measurements of dispersal of *Pst* regarding the vertical gradient of progeny uredinia. And I investigated the spread of diseases caused by aerially dispersed pathogens by comparing the primary disease gradients of *Pst* at the local scale investigated herein with previous primary disease gradient studies of cereal rust at the fieldwide and regional scales. This comparison of disease gradients is used to examine questions of the effects of spatial scaling in modeling spread of aerially dispersed particles, relevant to a broad set of ecological systems, including other aerially dispersed plant pathogens. In all, the aims of this thesis are to provide a better understanding of the spread of WSR and other plant diseases caused by aerially dispersed pathogens, which can have direct implications on efforts to control these diseases more effectively, and to increase our understanding of theoretical relationships between dispersal at multiple spatial scales.

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Chapter 2 - Local Dispersal of *Puccinia Striiformis* f. sp. *tritici* from Isolated Source Lesions

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Abstract

Understanding how disease foci arise from single source lesions has not been well studied. Here, single wheat leaves were inoculated with *Puccinia striiformis* f. sp. *tritici* urediniospores, and all wheat leaves within two intersecting 0.3 × 3.0 m transects were sampled in eight replicates over 3 years. The lesions observed on each of the top three leaves on plants within 1.5 m from the source lesion were three-dimensionally mapped. The total number of lesions within a 1.5 m radius was estimated by dividing the number of lesions observed within each 0.025 m-wide annulus by the fraction of the annulus sampled. The estimated total number of lesions produced within 1.5 m of a single source lesion ranged from 27 to 776, with a mean of 288 lesions. Eighty percent of the lesions were recorded within 0.69 m of the source infection. The proportion of total lesions observed at a given distance from the source was fitted well by the Lomax and Weibull distributions, reflecting the large proportion of lesions arising close to the source, and when fitted by an inverse power distribution had a slope (*b*) of 2.5. There were more lesions produced on leaves higher in the canopy than on lower leaves, with more lesions being detected above than below the point of inoculation. Simultaneous measurement of lesion gradients and spore dispersal in the final year of the study suggests that this pattern is due to greater susceptibility of upper leaves, rather than increased dispersal to upper leaves.

Introduction

Aerially dispersed plant pathogens include causal agents of some of the most devastating and economically important diseases, such as rusts and mildews (Brown & Hovmøller, **2002**). The spread of lesions reflects a synthesis of the physical dispersal of propagules with the biological interaction of successful infection, growth, and reproduction by a pathogen on a susceptible host. Understanding the spread of aerially dispersed pathogens in space and time can aid in efforts to control epidemics (Fitt *et al*., **2006**). An initial disease outbreak can be referred to as a focus, and can be initially formed by an isolated lesion resulting from a single infection (Zadoks & Van den Bosch, **1994**). Pathogens aerially dispersed by relatively small propagules, such as powdery mildews or rusts, have been shown to closely follow inverse power distributions within relatively uniform landscapes such as agricultural fields of annual crops. Inverse power distributions have very steep dispersal gradients, but with 'fat tails', in which a small but consequential number of propagules are dispersed at great distances from the source (Kot *et al*., **1996**). The degree to which inoculum is dispersed short versus long distances has implications for several important epidemiological evolutionary processes due to the number of founder events, including overcoming host resistance and maintaining pathogen genetic polymorphisms in gene-for-gene systems (Lannou *et al*., **2008**; Mundt, **2009**; Wingen *et al*., **2013**), as well as selecting for reduced pathogen latent periods (van den Berg *et al*., **2012**).

Several studies examining rust dispersal in crop canopies have been undertaken. However, sources of inoculum for these studies have most often contained enough propagules to form several, in some cases several thousand, initial infections on individual plants (Willocquet *et al*., **2008**), on several plants within a focal area (Asea *et al*., **2002**; Sackett & Mundt, **2005**), or on entire fields (Gitaitis *et al*., **1998**). More recently, it was demonstrated that the highly local dissemination of wheat

leaf rust requires the study of individual lesions if the full epidemiological impacts of dispersal gradients are to be understood (Lannou *et al*., **2008**). Collecting such data presents a number of challenges. To accurately describe dispersal from a single source requires lesion counts in areas of high lesion density. It also relies on the presence of isolated initial lesions, requiring both successful germination of the source spores, and absence of background infections from outside sources of inoculum (Lannou *et al*., **2008**; Mundt, **2009**). Assessing the dispersal gradient from a single lesion can help address how aerially dispersed pathogens spread at the disease front, as well as how diseases progress throughout a growing season (Mundt *et al*., **2009b**). Lesion counts per leaf, the sampling unit used in this study, can be used to determine if propagules are randomly distributed or over-dispersed (Waggoner & Rich, **1981**). This information can be useful in predicting when and where disease is likely to occur, and can give insights into the effectiveness of methods field managers may employ to slow the spread of disease (Severns *et al*., **2014**).

Wheat stripe rust (WSR) is a plant disease capable of causing significant crop losses and reductions in grain quality (Wellings, **2011**). Many pathogens disperse via multiple mechanisms. *Puccinia striiformis* f. sp. *tritici*, the causal agent of WSR, spreads by direct contact of a lesion and healthy uninfected wheat leaf tissue, by water dispersal, and through the air (Van der Plank, **1963**; Geagea *et al*., **1997**, **1999**). Of these methods, aerial dispersal appears to play the largest role, especially in long-distance dispersal, although humidity appears to have a significant role in urediniospore dissemination, especially in regards to urediniospores being dispersed individually or in clusters (Rapilly, **1979**). Urediniospores of *P. striiformis* have been shown to travel over 500 km (Brown & Hovmøller, **2002**), and to be able to cause disease upon deposition. This long-distance dispersal of urediniospores can cause entry of isolated lesions to a field or region (Hovmøller *et al*., **2002**).

Although dispersal gradients of plant pathogens have been studied empirically using field experiments, as well as simulated using computer models (Mundt & Leonard, **1985**), there have been few attempts to describe the dispersal emanating from single isolated lesions. To accurately describe dispersal from such a single source requires lesion counts rather than an estimate of disease severity, which is an average over a unit area. This study examined the local pattern of *P. striiformis* in wheat that originated from artificially inoculated, isolated lesions over the course of single generations of dispersal. These data were used to describe the horizontal and vertical distribution of progeny lesions within the wheat canopy and to estimate the reproductive potential of individual lesions.

As *P. striiformis* urediniospores are able to disperse over several hundred kilometres, capturing the entirety of the dispersal gradient was not feasible. However, this study should help elucidate the creation of foci and dispersal within a field, particularly at the disease front.

Materials and methods

Measuring disease gradients

The study site was chosen to minimize contamination from naturally occurring inoculum of WSR and other wheat pathogens. Field experiments were conducted in commercial wheat fields within a 6 km radius of Culver, OR, USA. This is an irrigated agricultural region of approximately 25 000 ha in the high desert of central Oregon and geographically isolated from major regions of wheat production in the state. Its climate is not conducive to WSR overwintering due to low winter temperatures.

All plots were 6 m radius circles located within fields used to study effects of conditions at disease outbreak sites on subsequent disease spread (Severns *et al*., **2014**). The fields were 13.7 × 73.2 m in 2012 and 7.6 × 42.7 m in 2013 and 2014, with the long dimensions oriented east–west. Predominant winds in the region are from the west-northwest (Van de Water *et al*., **2007**). All eight

plots containing a single source lesion were composed of monoculture plots of the soft white winter wheat cv. Jacmar, a cultivar no longer commercially planted (Table 1). An additional plot contained three source lesions, and was therefore excluded from the two-dimensional dispersal study, but was used to examine the vertical distribution of lesions and urediniospores (see 'Spore trapping'). Jacmar is susceptible to the races of *P. striiformis* used in this study, but resistant to the races that predominated in Oregon during the course of the study. The plots were surrounded by at least an 18.3 m buffer of soft white winter wheat cv. Stephens, which is resistant to races of *P. striiformis* used here. The field was subject to standard agronomic practices for wheat production in the area, including overhead irrigation approximately every 7–10 days beginning in March (Mundt & Sackett, **2012**), which provided soil moisture to encourage dew formation and successful infection events within the plant canopy.

Inoculations were performed in early spring, after temperatures became conducive to *P. striiformis* germination, but as early as possible to minimize the chance of interference from other sources of inoculum. Inoculations were performed with WSR race PST 5 in 2012 and 2013, but it began to exhibit reduced viability under field conditions after being grown over many generations in the controlled environment of the growth chamber, so race PST 29 was used in 2014. The two races have highly similar virulence patterns on known wheat genotypes (Chen, **2005**). The method of Lannou *et al*. (**2008**) was used to inoculate single leaves. Inoculated plants were selected for proximity to the centre of the plot from north to south and 6.1 m apart from east to west within plots, while ensuring approximately uniform coverage of wheat plants within 5 m of the plant to be inoculated. Leaves were inoculated with a mixture of 0.25 g urediniospores: 3.75 g talc. The inoculum was transferred from a vial via a pencil-top eraser to Scotch tape labels, which were placed on the adaxial surface of the uppermost leaf at least as wide as the eraser. The tape labels trapped transpiration water from the wheat leaves,

ensuring optimal leaf moisture for germination of the urediniospores. The inoculated leaf was marked with a Sharpie felt pen above the inoculation point in years 2012 and 2013. In 2014, the inoculated tiller was marked with a plastic zip-tie at its base, as it was easier to identify. About 30 mL water was poured into a white plastic bag, which was shaken and then placed over two wooden dowels taller than the inoculated wheat plant and staked down, acting as a moist chamber. Inoculations were performed close to sunset and on cloudier days, as it was found that prolonged exposure to radiant light greatly reduced the rate of successful germination in greenhouse tests. The plastic bag and the tape were removed the following morning.

The plots were searched for lesions after approximately 1.75–1.90 generations of disease spread (Table 1), as calculated by the Shrum (**1975**) degree-day model. This time period allowed for nearly all lesions of the second generation to be visibly sporulating, but without the possibility of third generation lesion expression. All tillers within two overlapping, perpendicular 3.0 × 0.30 m transects centred on the original lesion were inspected (Fig. 1a). The transect quadrant and distance from the source of the 0.025 m-wide distance interval (Fig. 1b) containing each tiller and the leaf position of each lesion were recorded, with the uppermost leaf, often referred to as the flag leaf, being considered leaf 1 and leaf number increasing sequentially going down the wheat stem. Due to senescence of lower leaves, leaves below leaf 3 were not considered in 2012 and 2013, and leaves below leaf 4 were not considered in 2014. When individual lesions were not distinct due to age and/or multiple overlapping lesions, a severity estimate was taken for the leaf and each 5% severity was considered to be one lesion. The rationale for this conversion was that the maximum number of lesions on a leaf was approximately 20.

Spore trapping

In 2014, spore traps were placed in a single plot, at three distances from an inoculated leaf along the four cardinal directions, in order to compare the dispersal of spores to the dispersal of lesions (Fig. 2). Each spore trap comprised a 0.91 m tall wooden dowel with three identical, artificial wheat leaves made of sections of laser-printer overhead projector slides, cut to the size of a scanned representative wheat leaf, and containing an approximately 10 mm long by 2.5 mm wide tab at the base to be taped on to the dowel with duct tape. Each artificial wheat leaf had a 1 mm grid pattern printed on it to aid in counting spores under a compound microscope. The artificial wheat leaves were placed grid-side down on a piece of varnished cardboard containing a small quantity of vacuum grease. The grease was spread thin by holding a razor on the top of the artificial leaves and pulling the leaves by the tab three times, or until the grease was spread evenly across the entire grid-side, excluding the tab. The artificial leaves were taped grease-side down at 0.13, 0.38 and 0.64 m from the top of the dowels.

Three spore traps were placed in each cardinal direction, approximately 0.15, 0.6 and 1.5 m from a tiller inoculated as described above, at locations where they would come into direct contact with as few wheat leaves as possible, such as between tractor passes. Exact locations of spore traps were recorded, along with the locations of all lesions (Fig. 2). Because of the late date of inoculation for this plot, the environment was extremely conducive to infection by *P. striiformis*, resulting in three lesions within the width of the eraser used to transfer the inoculum to the tape, approximately 7 mm on leaf 1, which was first observed 15 June 2014, as well as a stray source lesion 0.47 m from the source to the southeast at azimuth 319.4° on leaf 2, and a stray source lesion 1.82 m from the source to the southwest at azimuth 192.9° on leaf 1 (Fig. 2). Eleven days after initial placement, the spore traps were removed from the dowels and taped greased-side down on a blank laser printer overhead transparency

in a three-ring binder, using one sheet per dowel, labelling each spore trap by leaf position. *Puccinia striiformis* urediniospores were identified visually by size and shape using a Leica DM750 microscope under ×100 magnification, a Leica DFC 295 camera, and the Leica application suite to store and edit images.

Data analysis

All analyses were performed using R v. 2.14.0 (R Development Core Team, **2015**). Three-dimensional maps of lesion distributions were produced using the scatterplot3d package (Ligges & Mächler, **2003**). The modified inverse power distribution (Mundt & Leonard, **1985**),

$$y = a \times (x + c)^{-b} \qquad \text{(eq. 1)}$$

in which y is the mean number of lesions present on all leaves per tiller per 0.025 m, and x is the distance from the inoculum source, was fitted to the dispersal gradient of progeny lesions from single source lesions. The modified inverse power regression was linearized by taking the $\log_{10}$ of both sides, giving

$$\log(y) = \log(a) - b \times \log(x + c) \qquad \text{(eq. 2)}$$

where a is the intercept and b is the slope. The parameter c is an offset approximating the amount of space occupied by the initial inoculum. The incorporation of the c parameter also has the benefit of allowing Eqn. (2) to be defined at $x = 0$. The value of c was estimated by iteratively fitting Eqn. (2) with ordinary least-squares linear regression in increments of 0.001 m, and choosing the value of c that returned the highest R^2 value and lowest P-value.

Because several distance intervals contained zero lesions in individual plots, data from all plots of 2012–2014 were aggregated to enable the log–log transformed inverse power distribution to be fitted to them. While two different races of inoculum were used, the physical properties of the urediniospores are uniform, therefore the dispersal gradient should not differ between them. This minimized the amount of binning necessary to ensure at least one lesion per distance interval, allowing for log-transformation. When zero lesions were recorded at a single distance interval, it was binned by taking the mean number of lesions between the zero-lesion distance interval and an adjacent lesion-containing distance interval, which occurred in nine instances, all of which were greater than 1.04 m from the source, resulting in a bin size of 0.051 m in those instances.

In addition to binned log–log transformed linear regression, the Weibull and modified Pareto (also known as the Lomax) probability density functions (PDFs) and associated cumulative density functions (CDFs) were used to examine the probability of a given proportion of the total number of progeny lesions at a given distance. This was accomplished by multiplying the observed number of lesions per 0.025 × 0.30 m distance interval by the fraction of the total 0.025 m-wide annulus sampled. As fitting these PDFs did not require log-transformation, no binning of lesions across distance intervals was necessary. The Weibull CDF,

$$y = 1 - \mathrm{e}^{-\left(\frac{x}{b}\right)^{k}} \qquad \text{(eq. 3)}$$

was fitted by maximum likelihood estimation (MLE) to the cumulative number of lesions as a proportion of total lesions observed as a function of the distance from the source lesion. The derivative was taken to obtain the PDF:

$$y = \frac{k}{v} \times \left(\frac{x}{v}\right)^{k-1} \times e^{-\left(\frac{x}{v}\right)^{k}} \quad \text{(eq. 4)}$$

The modified Pareto distribution, a modified form of the Pareto distribution with the CDF:

$$y = 1 - \left(1 + \frac{x}{v}\right)^{-k} \quad \text{(eq. 5)}$$

was fitted in the same method as above, and the derivate was taken to get the PDF:

$$y = \frac{k}{v} \times \left(1 + \frac{x}{v}\right)^{-(k+1)} \quad \text{(eq. 6)}$$

The distributions were fitted by the simulated annealing algorithm (SANN), an algorithm that is particularly robust in its ability to find the global minimum in the presence of local minima (Bélisle, **1992**), as built into the 'Optim' function in R (R Development Core Team, **2015**). The initial parameters and temperature of the SANN algorithm were fitted iteratively by the lowest maximum log likelihood. These continuous distributions were made into discrete probability mass functions, as the data set was discretely organized by plant. These analyses were used to compare plots to each other by: leaf containing the mother infection, year of study, and replicate. This was accomplished by creating a flexible model with a varying number of parameters: in analysing the PDFs and CDFs of lesions as a function of distance plus a single blocking factor, a *v* parameter and a *k* parameter for each level of the given blocking factor was fitted as above. In fitting the PDFs and CDFs with two or more blocking factors, the first level of each blocking factor after the first was treated as the reference, with each successive level of the blocking factor adding an additional *v*- and *k*-parameter. The fitted modified Pareto and Weibull distributions were compared to each other by Akaike information criteria (AIC). The R script

used to analyse and plot the PMFs and CMFs as a function of distance and as a function of distance and source have been included (Fig. S1).

Results

The three uppermost *T. aestivum* leaves were healthy enough to visually count WSR lesions in all plots (Fig. 3). Additionally, leaf 4 was readable in 2014 (Fig. 4c,d). There was an overall mean of 0.27 lesions observed per *T. aestivum* tiller. The distribution of lesions across leaves varied significantly, with 0.12 lesions on leaf 1, 0.097 lesions on leaf 2, and 0.037 lesions on leaf 3. The number of lesions observed on a given leaf layer, as well as the relative proportions of lesions on each leaf layer, varied according to the leaf layer containing the source lesion (Fig. 4).

The mean number of lesions observed within the 3 × 0.3 m transects of all plots was 144.9. Extrapolated based on the fraction of each concentric 0.025 m-wide annulus covered by the sampling transects, there was a mean of 275.77 lesions within a 1.52 m radius of the source. The sum of all lesions at a given distance across all plots two-dimensionally extrapolated is presented in Figure 5. Of two-dimensional extrapolated lesions, 50% occurred within 0.23 m and 80% occurred within a mean of 0.69 m of the source lesion.

The mean number of lesions observed within 0.025 m of the source, approximately the radius of an average *T. aestivum* 'Jacmar' plant, was 7.058. This represents an autoinfection rate per plant of 2.56% of the two-dimensional extrapolated lesions within 1.52 m of the source lesion.

By aggregating lesion observations from all uncontaminated dispersal study replicates, a non-zero lesion count was observed in 53 out of 60 0.025 m-width intervals, with no interval greater than 0.051 m-width lacking one or more lesions. Binning only zero-count distance intervals with lesion counts

from adjacent distance intervals minimized the total number of bins and minimized their effect on the regression. In aggregate, the dispersal gradient of lesions from a single source lesion within 1.5 m was described by the equation fitted by least-squares of the log–log transformed data (Fig. 6). The dispersal gradient, when the amount of disease at distance = 0 m was normalized to 1 by dividing the observed lesion count within each 0.025 m-width interval by the number of lesions observed within the distance interval of 0–0.025 m from the source lesion, resulted in the equation fitted by least-squares of the log–log transformed data (Fig. S2). In examining the aggregated data from all plots from 2012 to 2014, variance in mean observed lesions per 0.025 m-wide increased with distance from the source (Fig. 6).

Non-linear PDFs and CDFs were converted to discrete probability mass functions (PMFs) and cumulative mass functions (CMFs) using each 0.025 m as a distance interval, and fitted to the proportion of total lesions observed within each 0.025 × 0.30 m bin, as distance from the source increased (Figs 7 & 8, respectively). The PMFs and CMFs separated by individual source leaf were also plotted (Figs S3 & S4, respectively). Of the non-linear distributions, the modified Pareto distribution better fitted the data than the Weibull, and each additional blocking factor, source leaf, year, and replicate, lowered the AIC as well (Table 2). However, when the data was two-dimensionally extrapolated to estimate the number of lesions in the entirety of each annulus, the Weibull fitted better than the modified Pareto (data not shown).

Upon resampling the plot containing the spore traps after approximately 1.8 generations, there was no significant difference between the number of urediniospores observed on the basal and the apical half of the spore traps (Fig. 9). Due to the presence of additional source lesions, there was no significant correlation between horizontal distance from the inoculated lesion and number of spores observed after a generation of dispersal. However, lesion counts significantly decreased as leaf position

increased going down the stem ($P < 2e{-}16$), while mean urediniospores per mm^2 significantly increased as spore trap position increased going down the dowel ($P = 0.013$; Fig. 10).

Discussion

The dispersal gradient of *P. striiformis* lesions from a single point source aggregated across all plots was best fitted by the inverse power model, as was the case in several previous field-wide studies (Mundt, **1989**; Sackett & Mundt, **2005**). The dispersal gradient had an exponent (*b*) well within the range found in previous studies on continental-scale dispersal (Mundt *et al*., **2009a**). As in the study by Lannou *et al*. (**2008**) on *P. triticina*, autoinfection was measured by direct counts, while alloinfection could not be completely measured due to long-distance dispersal. The observed dispersal gradient within 1.5 m of the source was substantially steeper than observed in a previous study on *P. triticina*(Frezal *et al*., **2009**). However, this leaf rust study was conducted using individual source leaves that were nearly saturated with infections, and contained spurious lesions within the plots in addition to the inoculated source lesions, which could result in much shallower infection gradients. The dispersal gradient was also over-dispersed, as in Lannou *et al*. (**2008**). While the count of progeny autoinfection lesions observed was considerably lower than in wheat leaf rust, this could be expected due to the much larger lesion size of WSR, and the more suitable environment for leaf rust infection in Lannou *et al*. (**2008**). The autoinfection rate per plant observed in the present study was more closely in agreement with previous studies on *P. graminis* f. sp. *avenae* (Leonard, **1969**) and *P. coronata* (Mundt & Leonard, **1985**, **1986**). When examining individual plots or plots aggregated by year or leaf position or source lesion, the dispersal gradient, often referred to as a dispersal kernel when describing the set of random draws from a given PDF (Holland, **2010**), was well fitted by the Weibull distribution, which has

previously been used to model the distribution of wind speed in the boundary layer (Kelly *et al*., **2014**), as well as biological dispersal data (Brøseth *et al*., **2005**).

A consequence of the dispersal gradient from a single source lesion being very steep near the source while also having a fat tail in which the exponent is unbounded, is that polycyclic epidemics have a high probability of invading new hosts at very low disease severities, but in succeeding generations can very quickly increase severity locally to near-saturation, resulting in accelerating rather than travelling waves of disease (Kot *et al*., **1996**). This can help explain why observed aerial dispersal in the field has been up to 20 times as fast as predicted by simple diffusion models (Andow *et al*., **1990**). The steepness of the dispersal gradient correlates with a reduction of importance of the dilution effect (Schmidt & Ostfeld, **2001**), which has been postulated as a primary mechanism in slowing epidemics (Mundt, **2002**). This is due to a greater proportion of urediniospores being deposited on the susceptible host tissue of the plant containing the source lesion relative to a shallower dispersal gradient in which a greater proportion of the spores would be deposited on a surface other than the plant containing the source lesion. This is due to the fact that, given the fat tail of the dispersal, it is likely that the pathogen could spread from one plant to others within fields at low frequency (Hastings *et al*., **2005**) thereby creating new foci, but that a very large proportion of spores would be deposited on the same plant as the source lesion, causing reinfection, or autoinfection, rather than deposition on a surface in which the spores could not cause infection (Mundt *et al*., **2010**).

The number of progeny lesions observed at or above the leaf containing the source lesion was greater than would be expected if urediniospore dispersal via sedimentation was the primary driver of vertical lesion distribution, and was greater than found in wheat leaf spread (Frezal *et al*., **2009**). The number of urediniospores decreased as spore trap height increased, contrary to the observed

distribution pattern of lesions, but in agreement with the expectation of a greater proportion of spores being present in the middle or lower portion of the canopy early in an epidemic (Aylor, **1999**). This result indicates that the observed vertical distribution of lesions was due to forces other than urediniospore dispersal. One potential explanation for this discrepancy in location of urediniospores versus lesions is a vertical gradient in environmental conditions and host physiology necessary for urediniospores to germinate, successfully infect a host, survive latently, and form sporulating lesions. There are vertical environmental gradients within the canopy of a wheat field, as UV radiation, temperature, leaf moisture, wind speed and wind direction are all inter-related and affected by the position in the canopy. Each of these environmental factors can affect the fitness of a given urediniospore (Eversmeyer *et al*., **1988**). Another potential factor driving the discrepancy between uredinial lesion spread and urediniospore spread is the decreasing age of wheat leaves with decreasing leaf position. Younger leaves have fewer hairs, thinner cuticles, and have been shown in other systems to correlate with increased disease severity (MacHardy, **1996**). Recent experiments with controlled inoculations (D. H. Farber and C. C. Mundt, unpublished data) showed that a substantially higher proportion of urediniospores form sporulating lesions on younger leaves as compared to older leaves.

Environmental heterogeneity can be a primary determinant of spatial and temporal patterns of dispersal (Wright, **2002**; Heino, **2013**). However, non-uniform patterns of dispersal can arise even in relatively homogenous environments, including conventional single-crop agricultural fields (Neubert *et al*., **1995**). Conventionally, dispersal gradients have been fitted by exponential or inverse power distributions (Sackett & Mundt, **2005**). Alternatively, the Weibull PDF has been fitted to biological dispersal data (Brøseth *et al*., **2005**; Quinn *et al*., **2011**). Use of PDFs has advantages over the inverse

power law distribution, as they do not require taking the log, thereby enabling inclusion of zeroes, a large component of count data sets.

Cumulative density functions, the integrals of PDFs, are useful in examining the distances at which given proportions of the total number of new lesions arise from a single source lesion. It has been shown that there is a point at which the graph of the CDF rapidly flattens as it approaches its asymptote, often stated as approximately the distance at which 80% of cumulative lesions are observed, which is referred to here as the n80. This can be thought of as the division between two separate dispersal processes. Within the n80, the pathogen is locally dispersed, with urediniospores being deposited without being transported by the wind current, while the other 20% make it into the wind current, and therefore have a relatively high probability of long-distance dispersal. This 80/20 division of dispersal methods has been found to maximize overall disease spread (Zawolek & Zadoks, **1992**). These findings could also support previous ecological work on 'the 80/20 rule', which states that 20% of individuals account for 80% of the disease spread (Woolhouse *et al*., **1997**).

Another challenge of modelling dispersal lies in accurately assessing the quantity of disease. Many studies of polycyclic diseases in agricultural settings have utilized disease severity estimates, largely due to feasibility restrictions of counting the enormous number of lesions present when inoculating large focal areas with many propagules. Estimating disease severity can be imprecise, especially at low levels of disease, as is the case at the disease front. A relatively small inaccuracy in estimation of disease severity, for example, estimating 0.2% severity when the actual severity is 0.1% in a 1 m^2 area, could translate to an additional 372 lesions, assuming four healthy, susceptible leaves per tiller, three tillers per wheat plant, and 1550 wheat plants per 1 m^2.

While this study was performed in a relatively uniform, conventionally managed agricultural field, there were still a number of variables that could not be held constant. One such factor was the leaf position of the source infection. Due to the low success rate of inoculation by *P. striiformis*, especially early in the season when overnight temperatures were often at or below freezing, many plots had to be inoculated after a generation had passed and no sporulation was observed on the inoculated leaf. This temporal shift in the start of the successful inoculation caused several corresponding environmental factors to change as well. Later in the season the wheat was at a later stage of development, so in order to maintain consistent methods of inoculating the highest position leaf that was at least as wide as the eraser used, a higher position leaf had to be inoculated. Additionally, the weather was generally warmer later in the season, and the wind patterns different. The generation time of the rust was also shorter later in the season.

Because of the environmental heterogeneity present even in a conventionally managed agricultural system, more replicates of the local dispersal study would be ideal. However, given the time and space constraints present, and the relative consistency of the dispersal gradients, it is felt that this study should further the understanding of the spread of diseases caused by the aerial dispersal of small spores, by examining the local dispersal from a single source lesion over a representative set of environmental variables within a wheat field.

This study represents one of the only studies to examine dispersal from a single source lesion, enabling a better understanding of disease spread at the disease front. Destructively sampling all wheat leaves within transects and counting lesions should provide a more accurate dispersal gradient than severity estimates. The parameters obtained from this study can be used to model disease spread over time at a finer scale, allowing for modelling of infection of individual plants.

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Table 2.1 Locations and timings of inoculations and disease assessments used to measure the spatial distribution of stripe rust lesions around initial single-lesion leaves.

Plot	Source Leaf	Inoculation Date	Sampling Date	Growing Degree Days
1	4	04/05/2012	19/06/2012	683
2	4	04/05/2012	18/06/2012	670
3	4	15/05/2012	18/06/2012	670
4	2	16/05/2013	26/06/2013	648
5	1	16/05/2013	24/06/2013	613
6	1	16/05/2013	24/06/2013	613
7	5	18/04/2014	07/06/2014	658
8	4	29/04/2014	15/06/2014	688

Table 2.2 Aikake Information Criterion (AIC) and Maximum Likelihood estimate (MLE) for each model and distribution.

Model	Distribution	Parameters	MLE	AIC
lesions ~ distance	modified pareto	2	4975.73	9955.46
lesions ~ distance	weibull	2	4990.48	9984.96
lesions ~ distance + source	modified pareto	8	4979.31	9974.62
lesions ~ distance + source	weibull	8	4960.00	9936.00
lesions ~ distance + source + year	modified pareto	13	4932.17	9890.34
lesions ~ distance + source + year	weibull	13	4948.21	9922.42
lesions ~ distance + source + year + replicate	modified pareto	26	4912.46	9876.92
lesions ~ distance + source + year + replicate	weibull	26	4931.77	9915.54

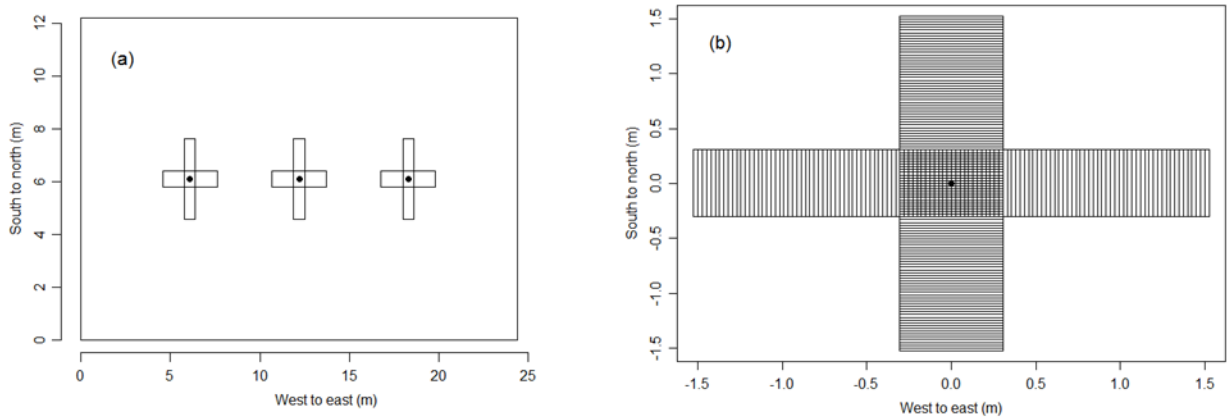

Fig. 2.1 Plot design: **a)** example of crossing 3.0 m by 0.30 m sampling transects used to measure the distribution of wheat stripe rust lesions around initially inoculated leaves (open circles); **b)** all tillers within the crossing rectangles were destructively sampled, with the number of lesions on each leaf recorded. In the crossing region within 0.15 m of the source lesion across or along rows, the coordinates of the tillers were recorded to the nearest 0.025 m. Outside of the crossing region, the quadrant and distance from the source to the midpoint of the bin containing the tiller was recorded.

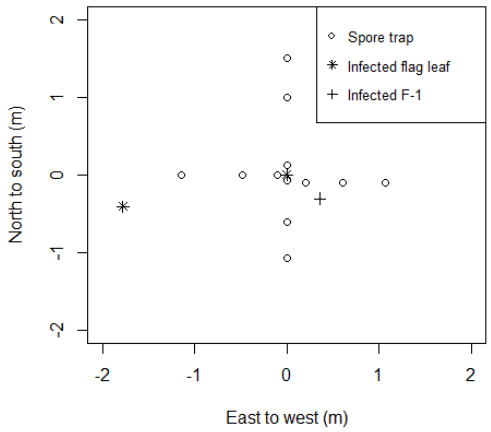

Fig. 2.2 Location of spores traps and *P. striiformis*-infected leaves.

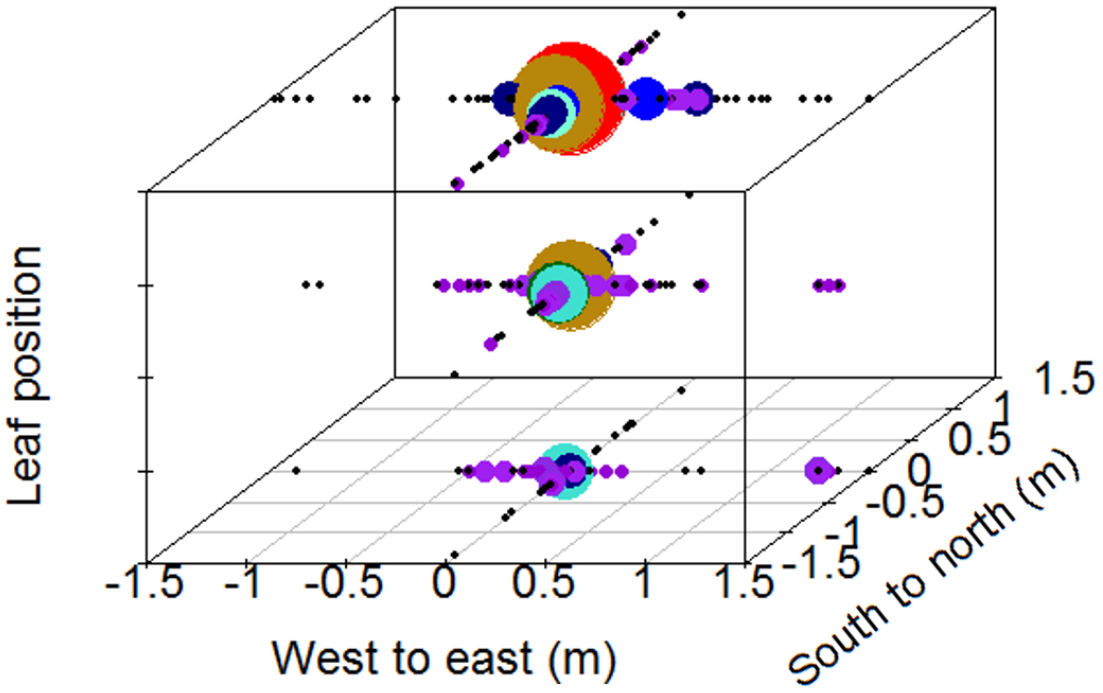

Fig. 2.3 All observed lesions on the uppermost three leaves of all tillers sampled in all plots 2012 through 2014.

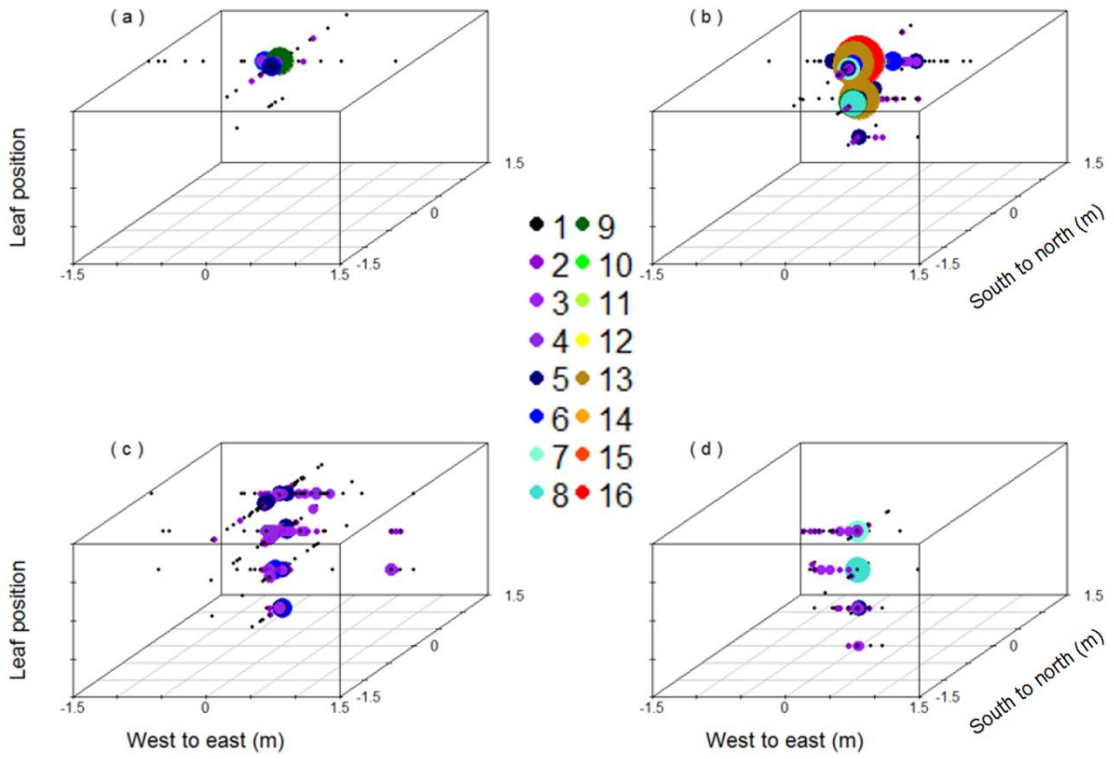

Fig. 2.4 Three-dimensional maps of lesions, aggregated by leaf position of source lesion. the source lesion was on **a)** leaf 1 in plots 4 and 6; **b)** leaf 2 in plot 3; **c)** leaf 4 in plots 1, 2, 5, 8; **d)** leaf 5 in plot 7.

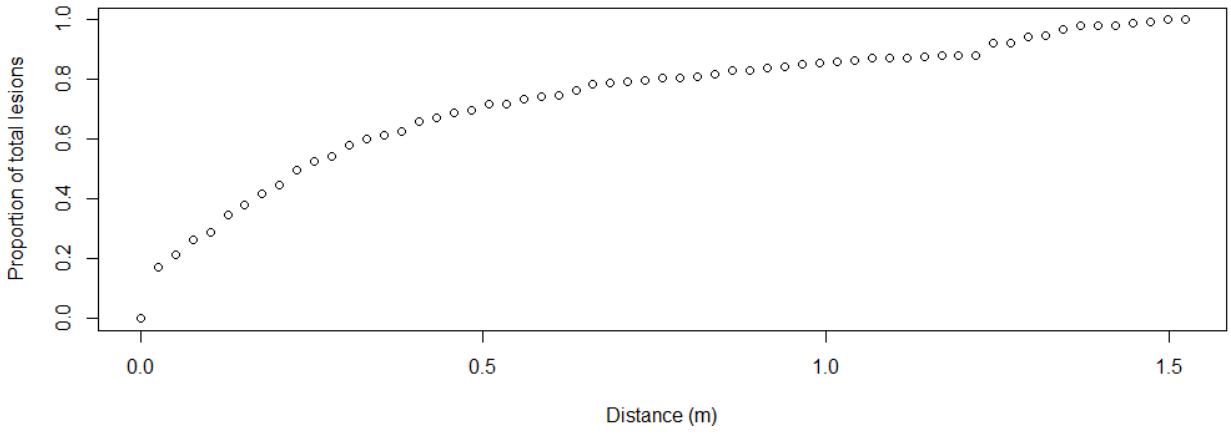

Fig. 2.5 Cumulative proportion of lesions across all plots by distance from source lesions, extrapolated two-dimensionally.

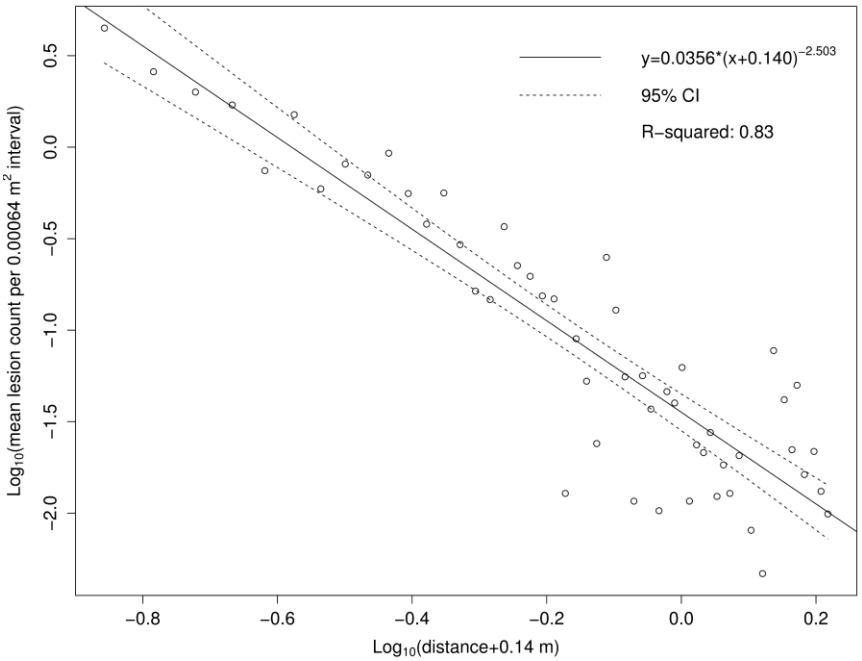

Fig. 2.6 Linear regression of log-transformed lesions as a function of log-transformed distance with best-fit c offset, aggregated from all plots 2012–2014 to maximize non-zero lesion count coverage of distance intervals, with 95% confidence interval (CI).

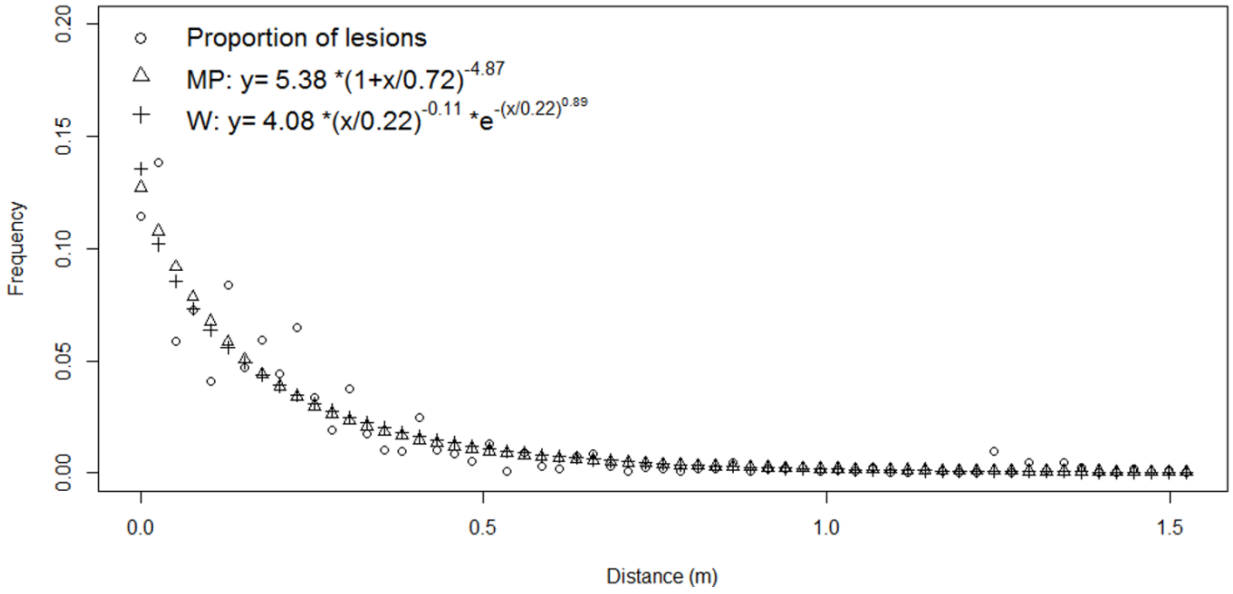

Fig. 2.7 Proportion of total progeny lesions observed as a function of distance from source lesion with best-fit modified pareto (MP) and weibull (W) probability mass functions.

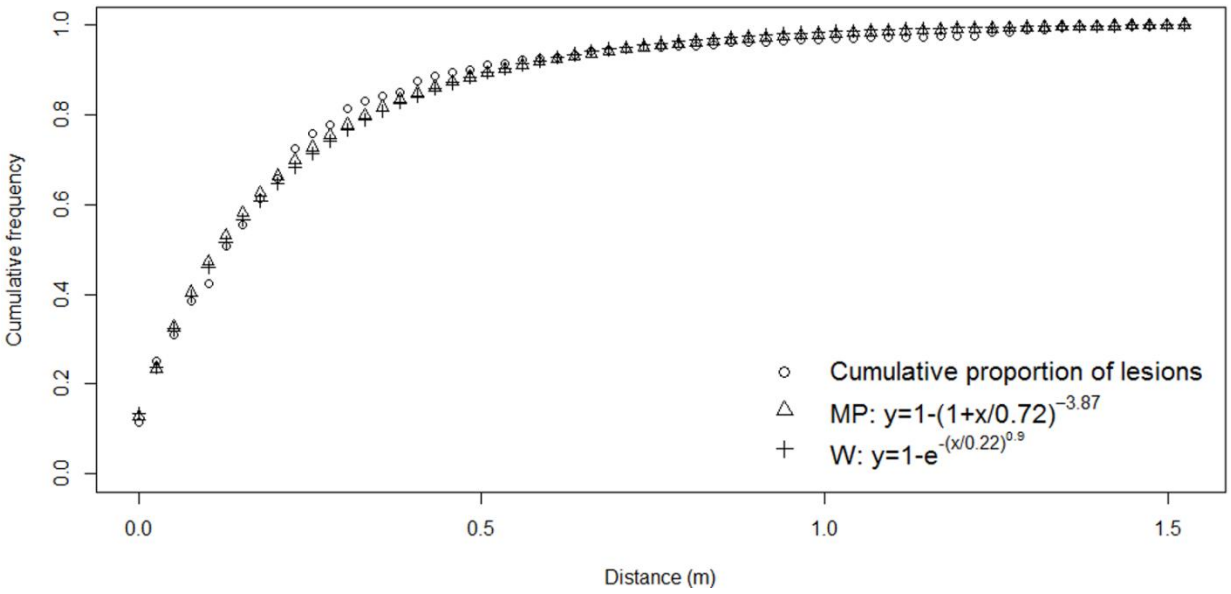

Fig. 2.8 Cumulative proportion of total progeny lesions observed as a function of distance from source lesions with best-fit modified pareto (MP) and weibull (W) cumulative mass functions.

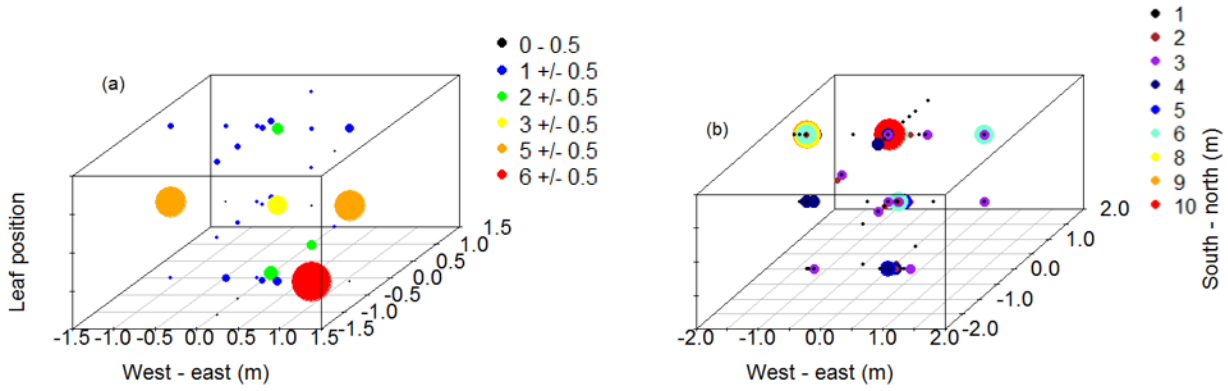

Fig. 2.9 Three-dimensional maps of a) the mean number of urediniospores per mm^2 per leaf trap and b) the total number of lesions in the plot containing the spore traps.

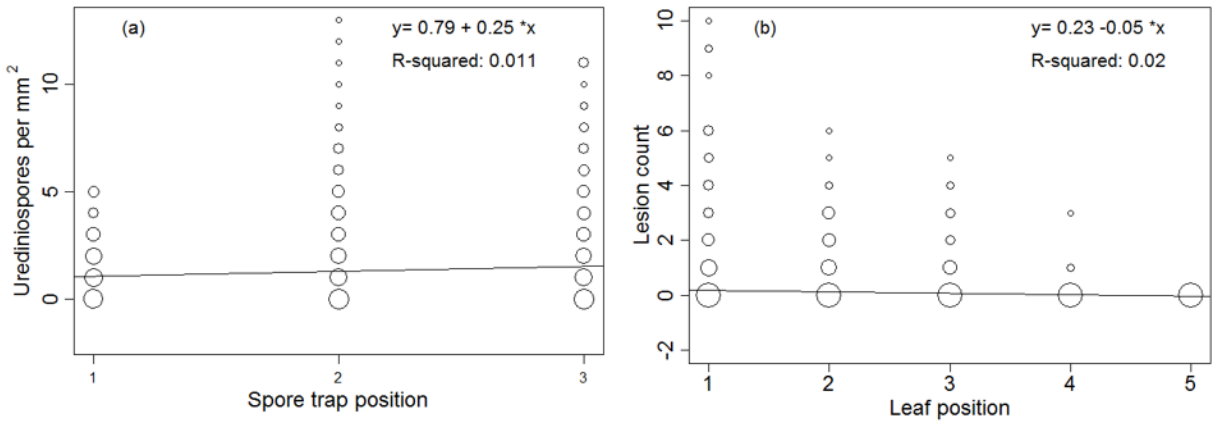

Fig. 2.10 Vertical distribution of *P. striiformis* in the plot containing spore traps. **a)** The number of urediniospores as a function of spore trap position (1 = 0.75 m, 2 = 0.5 m, 3 = 0.25 m aboveground). **b)** The number of lesions as a function of leaf position, which decreases in height sequentially. Point size is equal to 1 + $\log_{10}$(frequency of observation).

Chapter 3 - Effect of Plant Age and Leaf Position on Susceptibility to Wheat Stripe Rust

Abstract

In addition to dispersal of pathogen propagules, disease spread requires successful infection of host tissue. In plant disease epidemiology, susceptibility of host tissue is often assumed to be constant. This assumption ignores changes in host phenology due to developmental stage. To examine relative susceptibility of wheat leaves of different ages and leaf positions, 3-, 4-, and 5- wk old wheat plants were inoculated with equal quantities of urediniospores of *Puccinia striiformis* f.sp. *tritici,* the causal agent of wheat stripe rust (WSR). Disease severity on each leaf was assessed and fit by mixed effect linear model as function of leaf position and plant age. Younger plants had significantly greater disease severity than older plants, with mean severities of 50.4%, 30.1%, and 12.9% on plants that were 3 wk, 4 wk, and 5 wk old at time of inoculation, respectively. This effect was greater on leaves higher on the plant. Within same-aged plants, younger leaves had significantly greater disease severity than older leaves, with mean severities of 40.2%, 34.8%, and 17.7% on the uppermost, second, and third leaf, respectively. These results corroborate field data suggesting the vertical distribution of lesions was due more to differences in host susceptibility than to propagule dispersal.

Introduction

Epidemics, defined as spatiotemporal changes in disease severity (Madden, Hughes, and van den Bosch 2007), require virulent pathogens, susceptible hosts, and conducive environments to coincide in time and space. Therefore, to better understand epidemic spread, it is important to understand not

just propagule movement, but also differences in availability of susceptible host tissue over both time and space, as these differences can result in changes in pathogen growth, reproduction, and infection efficiency. Several models have been developed to account for the epidemiological effects of changing host architecture and phytochemistry as a plant grows (Kushalappa and Ludwig 1982; Ferrandino 2008; Tivoli et al. 2012; Costes et al. 2013). A very common assumption in plant disease epidemiology is that the degree of susceptibility of host tissue is constant both temporally and spatially. Violation of this assumption could significantly alter expected patterns of disease spread. However, relatively little attention has been paid to the interaction of disease progress and developmental stage of the host. Plant defenses have largely been viewed singly, but it has been argued that host-pathogen relationships occur along ecological continua, including factors affecting the fitness of the host (Agrawal and Fishbein 2006; Barrett et al. 2009), but quantification of the magnitudes of these effects is sparse.

There have been many reviews written on the genetic and biochemical basis for host susceptibility (e.g.: (Schie and Takken 2014)) and host resistance (e.g.:(Michelmore, Christopoulou, and Caldwell 2013; Chisholm et al. 2006), as well as the differences in types of resistance between race-specific major gene resistance and race non-specific minor gene resistance (Flor 1971; Johnson 1984; St.Clair 2010). Changes in host phenology throughout a growing season can have immense impacts on host susceptibility to infection by pathogens. Host phenology, defined as the timing of changes of developmental phase of a species over time in response to biotic and abiotic factors (Lieth 2013), has been shown to affect the ecological interactions of plants, including changes in susceptibility of plants to herbivory (Karban 1987; Boege and Marquis 2005; Asch and Visser 2007) and to disease (see below) with developmental stage of host plants.

Changes in host phenology occur both over time, as a plant ages, and spatially, from older plant tissues to younger ones. In domesticated crops such as wheat, these phenological changes can vary depending on a complex interaction of climatic and agronomic factors (Tao, Zhang, and Zhang 2012).The rates of these changes are dependent on many interacting ecological factors. The resource availability theory predicts that there is a negative relationship between rate of plant growth and amount of defense (Kikuzawa and Lechowicz 2011). Therefore, annual plants grown in nutrient-rich environments could be expected to exhibit greater intra-plant variation in leaf tissue phenology than longer-lived plants in nutrient-poor environments.

In addition to environmental effects, the life strategies of the microbiota of the phyllosphere can also affect host susceptibility. Differences in population sizes of epiphytic microbes as a function of leaf position within a plant, and as a function of plant age have been observed, with population sizes typically increasing with plant age, but with an inverse relationship between populations of introduced microbes and plant age (Jacques 1996). Disease severity has been found to be correlated with host phenology in varying ways, which might be accounted for, in part, by the life strategy of the pathogen. Necrotrophic pathogens, which preferentially derive their nutrition from plant tissue which they have killed, have often exhibit greater infection efficiency as plant tissues age. In the case of the facultative saprophyte *Ascochyta rabiei*, younger chickpea leaflets were less susceptible to infection than older leaflets (Dolar 1997). The relationship is not always as simple as this, as some necrotrophic pathogens have displayed the greatest infection efficiency during specific developmental stages based on the presence of certain plant structures. For example, strawberry becomes much more susceptible to infection by *Botrytis cinerea* once it flowers (Mertely, MacKenzie, and Legard 2002). For obligate pathogens such as rusts and mildews, the requirement of healthy host tissue for completion of their life

cycles (Schulze-Lefert and Panstruga 2003) suggests that host susceptibility may decrease in later phenological stages, as plants redirect resources away from aging tissues to younger tissues. Previously, older soybean plants were found to have reduced susceptibility to infection by *Phakopsora pachyrhizi*, but susceptibility did not significantly vary by leaf position (Bonde, Nester, and Berner 2012). Likewise, the rust *Puccinia jaceae*, a biocontrol agent on yellow star thistle, had reduced infection efficiency on plants older than 4-6 weeks, but did not have a clear relationship with leaf position (Bennett, Bruckart, and Shishkoff 1991).

As biotrophic pathogens, such as *Puccinia striiformis* f.sp. *tritici* (*Pst*), require healthy host tissue to complete their life cycles, it follows that senescence could have a significant effect on their ability to cause disease. Age-related resistance (ARR), in which, as plant tissues age they become less susceptible to pathogen infection, has been found in many systems, and can take many forms, including reducing susceptibility of plants to infection by viruses, bacteria, and fungi, by race-specific or non-specific mechanisms (Panter and Jones 2002). It has been previously shown that wheat plants with high-temperature adult plant resistance (HTAP), a form of genetic race non-specific resistance, were found to be more resistant to stripe rust at later growth stages and at higher temperatures (Quayom and Line 1985). More general physiologic changes in susceptibility may certainly occur as well. In this study, I tested the effect of plant age and leaf position on the susceptibility of wheat to *Pst* when grown in a common environment.

Materials and Methods

Study site and materials

Three runs of the experiment were performed in the Oregon State University West Greenhouse facility, which was controlled to maintain nominal temperatures of 18/16° C daytime/nighttime

temperatures. Actual daily high and low temperatures for the greenhouse were recorded, with a mean high of 23.1° C and a mean low of 17.3° C. For each run of the experiment, seeds were planted on three dates at one-week intervals. For each planting date of each run, ten seeds of soft white winter wheat (*Triticum aestivum*), cultivar 'Jacmar', which has not been found to exhibit HTAP or hypersensitivity reation (HR) to the race of *Pst* used in this study, were planted in each of 10 cylindrical 3.785 L plastic plant pots containing potting Sungro Sunshine LA4 P soil. All plants were grown directly below grow lamps in the greenhouse in a randomized complete block design, where each of the 10 blocks contained one pot of each of the three plant ages. This blocking structure was maintained during the inoculation procedure and during post-inoculation growth in the greenhouse (see below). The plants were watered every other day to keep soil at or near field capacity. Plants were fertilized every other week with 0.39 ml dry Miracle Gro fertilizer (ScottsMiracleGro, Marysville, OH) per pot, dissolved into 100 ml water. Lights were set to a 16 hour on/8 hour off schedule, turning on at 6:00 and turning off at 22:00. During hours when the lights were scheduled to be on, if ambient light levels exceeded 90 µmol photons m^{-2} s^{-1}, the lights shut off and turned back on when light levels dropped below 75 µmol photons m^{-2} s^{-1}.

Inoculation

Inoculations were performed with *Pst* CDL 29 (Chen 2005c), which was stored in liquid nitrogen. Fresh inoculum was produced by inoculating plants grown at 15° C with frozen inoculum, and harvesting the progeny urediniospores. Each block of three pots received a mixture of 15.2 mg of urediniospores and 228 mg of talc. This mass of inoculum contained approximately 858,800 urediniospores, as calculated by the density of similar-sized urediniospores of *P. hordei* (Teng and Close 1978).

Nine randomized complete blocks (RCBs) of plants were inoculated, and the tenth block served as a non-inoculated control. Plants were 3, 4, or 5 wk-old at the time of inoculation. All plants were

moved from the greenhouse to the outdoors for 12 hr prior to inoculation, as exposing wheat plants to cooler temperatures has been shown to increase infection efficiency (unpublished data). Prior to inoculation, each block of plants was placed in an open cardboard box 0.56 m in length x 0.42 m in width x 0.51 m in height, and plants were misted with water from a squirt bottle. The inoculum-talc mixture was hand-pumped out of a DeVilbiss Powder Blower model 119 (DeVilbiss Healthcare, Somerset, PA) rotated around the top of the inoculation box. The uninoculated control plants were placed in the inoculation chamber, misted with water, and placed in the dew chamber. The plants were placed in the dew chamber at 13° C overnight, and were then returned to the greenhouse where the growing conditions remained as they were prior to inoculation.

Disease assessment

All plants were sampled approximately 1.75 generations after inoculation, and the exact generation stage as predicted by Shrum's degree-day model (Shrum 1975) was recorded. This allowed for the maximum number of lesions to sporulate without encroaching on the subsequent generation. The disease severity as estimated by visual observation of percent of leaf area covered by WSR lesions of all leaves on the main stem of all plants was recorded. Any leaf that had senesced to the extent that it was no longer possible to accurately visually assess disease severity was excluded from the study.

Data analysis and model fitting

The effect of leaf position and plant age on disease severity over time was analyzed using R statistical computing software (R Development Core Team 2015). Leaf position was considered a quantitative variable, as it has been shown to be closely tied to growing degree days (GDD) and to attach to the culm at evenly spaced intervals (Karow et al. 1993) Leaves 1-3 were considered suitable for analysis as they were present at the time of inoculations, and had not senesced at the time of disease

assessment. Leaf 1 was the uppermost, fully expanded leaf at the time of inoculation, and leaves 2 and 3 were sequentially lower. Disease severity was regressed against the number of degree days elapsed between inoculation and disease assessment date to examine the disease severity at equal plant and pathogen developmental stages across runs. Mean disease severity was aggregated across all plants in each pot (the experimental unit). Differences in disease severity between runs were examined using two-way ANOVA followed by Tukey's Honest Significant Difference post-hoc test to determine which runs differed from each other.

I tested the linear effects of leaf position, plant age, mean daily temperature, and all interactions thereof as fixed effects using the step function in the "lmerTest" package for the R Statistical Programming Language (Bates 2015). Block nested within run was considered as a random effect with potential to influence the intercept. The likelihood ratio test using maximum likelihood estimation was used to compare mixed effects models with differing measures of plant age, the cumulative degree days from planting date to inoculation date, and the cumulative degree days from planting date to sampling date (Baayen, Davidson, and Bates 2008; Barr et al. 2013). The fixed effects most correlated with disease severity were plant age and leaf position, each of which are examined in more detail below, as well as their interaction. Inclusion of mean daily temperature, mean daily high temperature, and mean daily low temperature, as well as each of their interactions with all other explanatory variables, resulted in linear mixed models with greater AIC values, and were therefore eliminated from the final model. Figs. were generated using the base R package and with the ggplot2 package (Wickham, Chang, and Wickham 2013).

Results

Mean disease severity decreased linearly with plant age as measured by cumulative degree days from planting date to inoculation date, as well as from planting date to sampling date. While direct comparisons of degree days from planting date to inoculation date and degree days from planting date to sampling date was not possible given the differing fixed effects, they were very similar in fitting the data, with AIC values of 2309.2 and 2315.3, respectively. Therefore, the final model included degree days from planting date to inoculation date, as that resulted in a lower AIC, and more closely addressed the questions this study aimed to examine regarding the effect of plant age on susceptibility to infection. The random effect of block nested within run was highly significant (P value: $2.2*10^{-16}$) as tested by the chi-squared test. The best fit model of severity *s* was

$$s = 177.49 - 4.39d - 40.00l + 1.00d * l$$

eq. 1

in which *d* is defined as degree days from planting date to inoculation date, and *l* is defined as the leaf position from the top of the tiller. This model was used to test significance of the effects described below.

Magnitude of Fixed Effects on Disease Severity

The overall mean disease severity across all plants in all treatments was 31.0 percent, which is within a range in which susceptible leaf area is not a significantly limiting factor, but disease severity differences are likely to be large enough to be significant (James 1974). Disease severity was highly correlated with plant age, calculated as cumulative degree days from planting date to inoculation (P value: $<2*10^{-16}$). Mean disease severity and variance across the uppermost three leaves in all pots is reported for each leaf and each age plant (Table 1). Younger plants had greater disease severity than older plants on each individual leaf (Fig. 1). Disease severity decreased linearly as leaf position increased (Fig. 2; P value: $2.01*10^{-10}$), with mean severities of 40.2%, 34.8%, and 17.7% on the uppermost, second, and third leaf, respectively.

Mean disease severity across the uppermost three leaves decreased with cumulative degree days. Additionally, disease severity was greater on younger plants than on older plants, with mean severities of 50.4%, 30.1%, and 12.9% on plants that were 3 wk, 4 wk, and 5 wk old at time of inoculation, respectively. There was a significant interaction of leaf position and plant age on disease severity, with disease severity decreasing more rapidly as a function of cumulative degree days on upper leaves than on lower leaves (Table 2). This was due to a much greater extent to the wide variation in disease severities on the uppermost leaf in the different age treatments, relative to the range of disease severities on the third leaf in the different age treatments.

Discussion

Our study demonstrated a greater disease severity on younger leaf tissue, which suggests a greater proportion of urediniospores were able to successfully infect and produce sporulating progeny uredinia on younger wheat plants and younger wheat leaves within a given plant. Additionally, it has been observed that lesion expansion, which is particularly important in WSR (Shrum 1975; Berger, Filho, and Amorim 1997), is reduced at later growth stages of wheat (Luo and Zeng 1995). These results could help inform breeders of the trade-offs conferred by selecting for delayed senescence (Wang et al. 2015).

This study quantifies the relative receptivity of a highly susceptible cultivar of wheat to infection by *Pst* as influenced age of plant and leaf tissue within an individual plant. These results corroborate previous observations of greater host resistance at later growth stages in agricultural fields (Panter and Jones 2002; Runyon, Mescher, and Moraes 2010; Bux, Ashraf, and Chen 2012), as well as help elucidate how senescence-driven resistance reduces susceptibility to stripe rust to a greater extent on younger leaves relative to older leaves within a single plant. Examining cumulative degree days from planting date to sampling date could act as a better gauge of phenological changes in host susceptibility than relying solely on temporal metrics (McMaster and Wilhelm 1997). These results suggest a general physiologic response within a susceptible cultivar, in addition to differences among cultivars caused by HTAP race non-specific resistance in wheat (Qayuom and Line 1985; Milus and Line 1986; Chen 2013) and race non-specific ARR in *A. thaliana* (Quirino, Normanly, and Amasino 1999; Diener and Ausubel 2005)

The vertical gradient of disease severity, in which severity per leaf declined from upper, younger leaves to older, lower leaves of wheat plants, corroborates our previously observed vertical distribution of lesions in field plots (Farber, Medlock, and Mundt 2016), where a majority of progeny lesions were observed at or above the source leaf. This study also provides evidence to support a plausible alternate

explanation of the mechanism resulting in the observed vertical gradient of stripe rust lesions in the field: that differences in host susceptibility to infection by *Pst* urediniospores by age and leaf position was the primary driver of the resulting observed vertical distributions of lesions, as opposed to the hypothesis that a vertical gradient of urediniospore deposition resulted in a greater proportion of urediniospores landing on upper leaves than lower leaves.

Our results also may be of relevance to the temporally changing factors affecting the rate of spread of plant epidemics. The greater disease severity on younger leaves within a plant, which are higher in the canopy, could help explain the relatively frequent long-distance dispersal events observed in WSR, as urediniospores emanating from uredia higher in the canopy could potentially have a greater probability of getting transported by higher speed wind currents compared with urediniospores originating from lower in the canopy (Aylor 1999; Pfender et al. 2006). These long-distance dispersal events contribute to accelerating rates of disease spread (Mundt et al. 2009; Cowger, Wallace, and Mundt 2005). Therefore, incorporating a temporal and vertical gradient of susceptibility into disease spread models could increase their ability to accurately predict the spread of epidemics.

Our study could help elucidate the complex ecological relationships between plant development, environmental conditions, herbivory, and disease pressure from pathogens with greatly differing disease pressure. Many of these relationships have been theorized (Develey-Rivière and Galiana 2007; Tylianakis et al. 2008; Kikuzawa and Lechowicz 2011; Alcázar et al. 2011). Due to the great number of interacting variables (Grulke 2011), however, more studies are needed to understand how age-dependent disease risk affects the portion of a plant's carbon budget going to disease. This research could inform future studies of the biochemical basis for non-specific ARR to obligately biotrophic pathogens. Plant growth and development is regulated by several metabolites, or phytohormones, including 3-indole-aceteic acid (auxin), gibberellins, cytokinins, and many other compounds (Goodman,

Király, and Wood 1986). Likewise, senescence is regulated by its own set of phytohormones, principally ethylene, jasmonic acid, salicylic acid, and abscisic acid (Häffner, Konietzki, and Diederichsen 2015), but can also be counteracted by the aforementioned plant growth phytohormones. In absence of pathogens, senescence has been described as a form of programmed tissue death, which redirects resources to other parts of the plant. In annual grasses such as wheat, this redirection is largely due to redirecting resources towards the uppermost leaves, which receive the most photosynthetically active radiation (PAR). While HTAP has been well-documented in many landraces of wheat (Bux, Ashraf, and Chen 2012), little attention has been paid to how susceptibility to infection by *Pst* varies by leaf position and plant age for wheat genotypes lacking HTAP. However, it has been shown that several defense genes are upregulated during senescence of *A. thaliana* and, for many of these genes, this increase in mRNA transcripts occurs independently of pathogens or salicylic acid accumulation (Quirino, Normanly, and Amasino 1999). Similar work in wheat could aid in our understanding of the mechanisms underlying the ARR conferred by changes in phenology addressed here, which could be a benefit to controlling epidemics by breeding for resistance.

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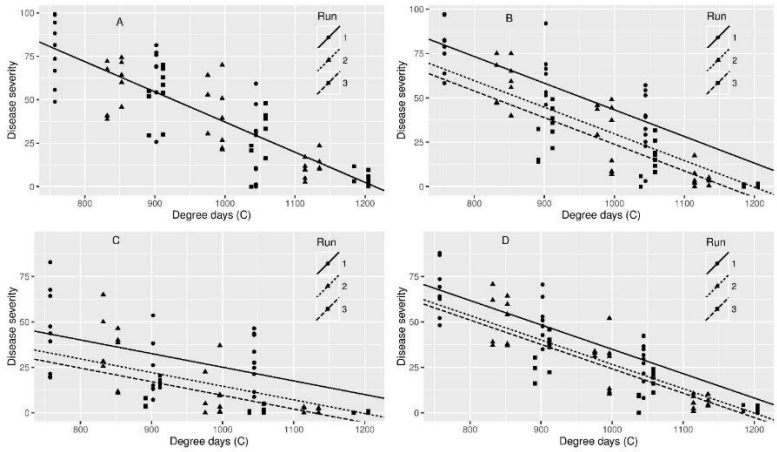
Table 3.1 Mean wheat stripe rust severity and variance (Var) on each of the uppermost three leaves of wheat plants by age in weeks at time of inoculation. Variation produced by run was excluded.

	Stripe rust severity (%)					
Plant age (wk)	**Leaf 1[a]**		**Leaf 2**		**Leaf 3**	
	Mean	**Variance**	**Mean**	**Variance**	**Mean**	**Variance**
3	63.8	252.8	56.5	161.4	31	294
4	44.5	289.2	34.4	193.9	11.4	141.2
5	15.5	151.4	14.4	100.8	10.8	57.3

[a] Leaf 1 was the youngest, fully expanded leaf at time of inoculation; leaves 2 and 3 were sequentially older.

Table 3.2. Parameters of $Y = a + bx$ and Aikake Information Criteria (AIC) for mixed linear regression of wheat stripe rust severity as function of age by individual leaf and experimental run, in which a is the severity at 0 degree days, and b is the change in severity for an increase in degree days of one. Random effect of run was not significant on leaf 1.

Leaf	Run	*a*	*b*	AIC
1	Fixed effects	191.29	-0.16	793.14
1	1	191.29	-0.16	
1	2	191.29	-0.16	
1	3	191.29	-0.16	
2	Fixed effects	168.37	-0.14	782.03
2	1	176.64	-0.14	
2	2	166.33	-0.14	
2	3	162.13	-0.14	
3	Fixed effects	83.39	-0.07	750.68
3	1	90.71	-0.07	
3	2	81.79	-0.07	
3	3	77.67	-0.07	
Mean	Fixed effects	151.73	-0.13	746.85
Mean	1	155.41	-0.13	
Mean	2	150.57	-0.13	
Mean	3	149.22	-0.13	



	Severity ~ age	R squared
A	y = 211 −0.2 *x	0.71
B	y = 182.5 −0.2 *x	0.8
C	y = 91.6 −0.1 *x	0.54
D	y = 162.8 −0.1 *x	0.57

Fig. 3.1 Linear mixed-effect model of the mean wheat stripe rust severity (% of maximum) per pot as a function of cumulative degree days from planting date to inoculation date, with experimental run as a random intercept. A) Leaf 1 (no significant effect of run); B) Leaf 2 C) Leaf 3; D) Mean of uppermost three leaves. Leaves are numbered in descending order, with 1 being the uppermost fully expanded leaf at time of inoculation.

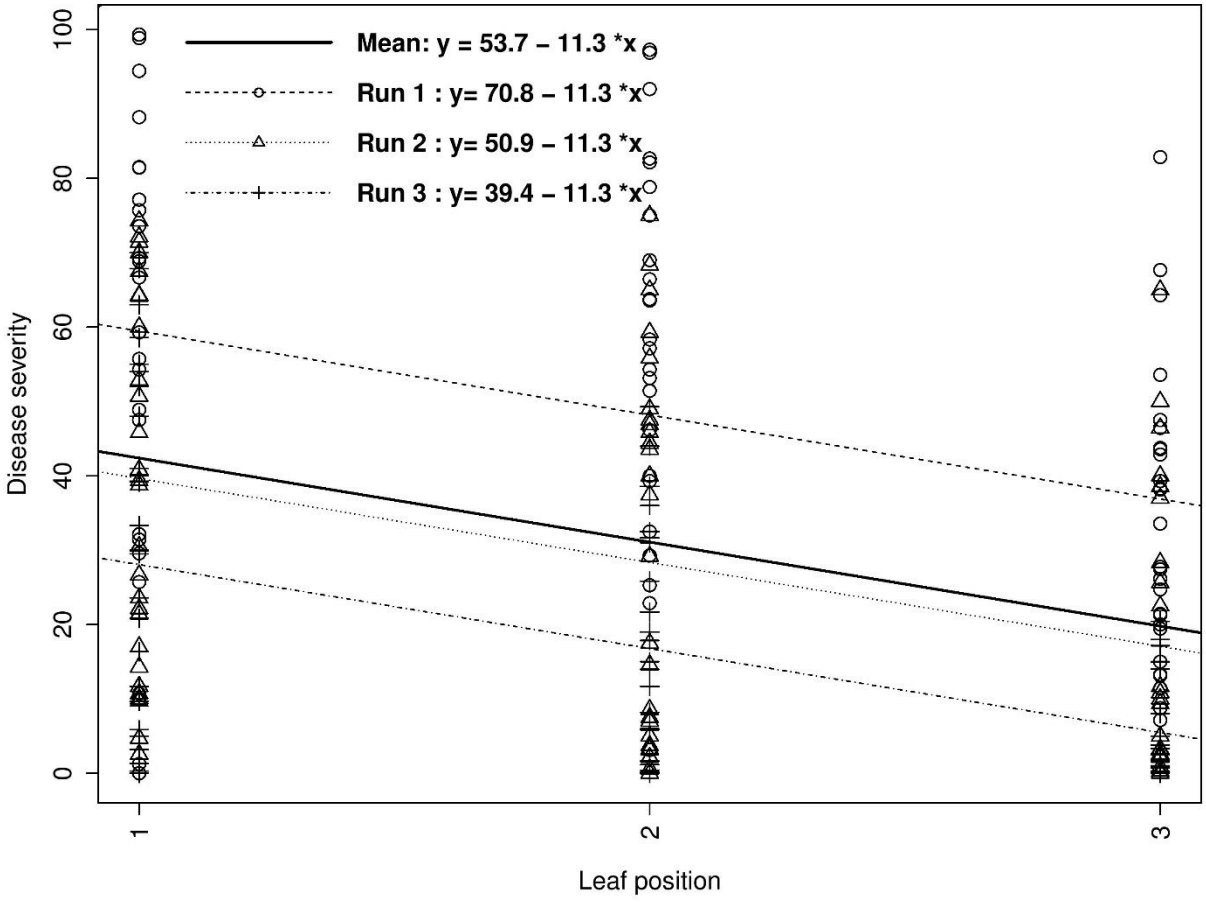


Fig. 3.2 Random intercept linear regression of the effect of leaf position on wheat stripe rust severity across three plant age treatments (3, 4, and 5 wk-old at inoculations) in three runs of a greenhouse experiment. Leaf positions are in descending order, with 1 being the topmost leaf at time of inoculation. Disease severity was visually measured as the percent of maximum leaf area covered by lesions. Each datum is the mean all leaves at a given leaf position on all plants within a single pot (the sampling unit).

Chapter 4 - Modeling the Spread of Cereal Rusts Across Spatial Scales

Introduction

Scaling, in ecology, is the study of how components or processes of an ecosystem change with another variable (Storch, Marquet, and Brown 2007). One of the most salient ecosystem variables is that of spatial scale. Spatial scaling, the changes in ecosystems as a function of the grain or resolution, and the extent of distances being considered (Turner et al. 1989), is of great interest, and has been shown to cause significant changes in many ecological dynamics, including the relationship of species diversity to productivity (Chase and Leibold 2002), population synchrony (Lande et al. 1999; Liebhold, Koenig, and Bjørnstad 2004; Mortelliti et al. 2015), and reproduction potential (Mikaberidze, Mundt, and Bonhoeffer 2014). There has been growing interest in how dispersal varies across multiple spatial scales (Cadotte and Fukami 2005; Mundt et al. 2009; Bullock and Nathan 2008).

Dispersal of organisms or their propagules is an important component of spatial ecology and epidemiology, and can have great impact on population dynamics (Hassell 2000), in that dispersal can allow species to use spatially disparate resources (Gilbert 2012), to invade new locations (Clark, Lewis, and Horvath 2001), and to genetically diversify (Ibrahim, Nichols, and Hewitt 1996). Mode of dispersal can interact with environmental heterogeneity to influence species co-occurrence (Heino 2013). The dispersal gradients of organisms with differing modes of dispersal can also be best fit by differing functions, e.g. with rain-splash dispersed propagules more often being better fit by exponential distributions, and aerially dispersed propagules more often being better fit by inverse-power distributions (Fitt et al. 1987). Aerial dispersal of small particles has been described by fat-tailed distributions, often in the form of an inverse power or modified inverse power distribution. Given advancements in computational power which drastically reduced the time required to fit models by maximum likelihood estimation (MLE), non-linear probability density functions (PDFs) have been used to

describe dispersal kernels - the set of random draws following a given probability density function describing the proportion of total dispersal at a given distance from a source (Holland 2010). Unlike fitting the inverse power distribution, these PDFs do not require log-transformation so they are useful in handling datasets with a large number of zeros, such as is expected with count data in the tails of dispersal gradients. Changes in the shape of the dispersal kernel can have significant changes in the speed and rate of acceleration of the population changes of invading organisms such as plant pathogens (Kot, Lewis, and Driessche 1996).

Many organisms are aerially-dispersed passively by wind (Bullock and Clarke 2000; Rieux et al. 2014; Okubo and Levin 1989). The spatiotemporal dynamics of their populations are greatly affected by the physical processes of air movement, most notably wind speed, direction, and turbulence (Aylor 1990; Novak et al. 2000; Shaw et al. 2006), as well as physical attributes of the particles being dispersed, such as their size, mass, and surface roughness (Petroff et al. 2008). These attributes of wind are in turn affected by environmental heterogeneity and corridors (Damschen et al. 2014; Bohrer et al. 2008). Understanding how wind-dispersed organisms spread across a variety of scales can improve our understanding of their prevalence in the ecosystems that they inhabit, and the likelihood of introductions into new regions, which could be of particular importance for invasive plants or infectious diseases.

While aerial dispersal is widely recognized as an important component of ecological population dynamics, measuring it has proven challenging due to the small physical size of many propagules, the large number of propagules produced by a dispersing population, the complexity of physical processes involved, and the long distances over which propagules can disperse (Nathan 2001). Long-distance dispersal (LDD) in particular plays an extremely important role in ecological processes, particularly in disease ecology (Bialozyt, Ziegenhagen, and Petit 2006; Filipe et al. 2012; Wingen, Shaw, and Brown

2013; Christopher C. Mundt et al. 2009), and changes in the tail of the models describing LDD lead to large changes in the populations they predict (Bullock and Clarke 2000). In plant epidemiology, to overcome the challenge of measuring the immense quantities of often microscopic propagules, the pattern of spread of disease can be used as a proxy for dispersal, assuming host susceptibility is homogenous. Most empirical dispersal studies are not able to assess the entire range of distances of dispersal, as this can be upwards of 500 km for aerially dispersed small propagules (Aylor 1982). On the other end of the spectrum, local aggregation, which can be indicative of accelerating spread, as in many foliar diseases, requires a small spatial scale to accurately measure (Lannou et al. 2008). Therefore, it would be greatly beneficial to our understanding of dispersal if it were possible to evaluate a set of empirical dispersal data across spatial scales. A useful feature of inverse power distributions is that they are theoretically scale-independent, in that the shape parameter remains unchanged across scales, with only the constant of proportionality changing (Gisiger 2001; Marquet et al. 2005). Therefore, it may be possible to compare dispersal gradients across multiple scales using a single inverse power distribution.

I used stripe rust of wheat (*Triticum aestivum*) as a model to study dispersal at multiple spatial scales. The wind-dispersed basidiomycete pathogen, *Puccinia striiformis* f. sp. *tritici*, causes local lesions on wheat leaves. Rust species are well-known for their ability to be dispersed long distance by wind (Aylor 2003) and are currently of substantial significance on cereal crops worldwide (Hovmøller et al. 2008). Similar rust fungus species are common in many other agroecosystems and also are frequent in natural systems such as prairies (Mitchell et al., 2002) and forests (Sinclair et al., 1987). RepeaTable scaling relationships, consistent with inverse power law dispersal, were found for stripe rust and other plant and animal disease caused by LDD pathogens of divergent taxonomy and at scales ranging over five orders of spatial magnitude, from small field plots to intercontinental spread (Christopher C. Mundt et al. 2009; Mundt et al. 2013; Mundt and Sackett 2012). In the current study, I compared the primary disease gradients of rust across multiple spatial scales, comparing empirical datasets with greatly

differing grain size and extent. I then determined whether the predicted spatiotemporal spread of disease depended upon the scale at which data were collected to estimate dispersal kernels in simulations. To do so, I constructed a spatiotemporal susceptible-latently infected-infected-removed (SLIR) model (Ogut and Bishop 2007), equivalent to the susceptible-exposed-infected-removed (SEIR) model (Goleniewski 1996; Gilligan and van den Bosch 2008; Cunniffe et al. 2011) to examine disease spread over time across local, landscape, and regional scales, given a biologically relevant range of inputs for reproduction rate, length of latent period, and length of infectious period.

Methods

Field methods

Dispersal kernels of wheat rusts at the local (Farber, Medlock, and Mundt 2016), fieldwide (Sackett and Mundt 2005a), and regional (Kingsolver, Peet, and Underwood 1984) scales were estimated from gradients of disease spread from artificially inoculated sources. These studies have been described previously in detail, but are summarized below. Primary disease gradients (gradients determined after one generation of disease spread from the inoculated source) of WSR were conducted in 2012, 2013, and 2014 in commercial wheat fields in Jefferson County, OR. Standard agronomic practices for irrigated commercial wheat were used. The study was conducted on a wheat cultivar that is highly susceptible to WSR, and was bordered by a resistant cultivar. The plots were planted in tractor passes 1.52 m wide, each containing 7 rows 0.19 m apart. Plant spacing within rows was not controlled, but averaged approximately 0.254 m per plant.

The local dispersal study was conducted in 6 m radius plots, which were thoroughly inspected prior to inoculation to ensure no sporulating infections. A single WSR lesion was established by placing a mixture of *P. striiformis* urediniospores and talc on a piece of adhesive tape, which was adhered to the adaxial surface of the uppermost wheat leaf. This plant was then covered with a white plastic bag

containing approximately 30 ml water. The bag and tape were removed approximately 16 hr after inoculation. After 1.2 – 1.5 latent periods (Shrum 1975), the plots in which the inoculated leaf contained a sporulating lesion were checked again to ensure no background infection. After 2.75 – 2.9 latent periods from initial inoculation (1.75 – 1.9 latent periods of disease spread), the location and number of lesions on each of the non-senesced leaves of all plants within two crossing 3.0 m by 0.3 m transects centered on the inoculated plant were recorded.

Plots used for the fieldwide primary disease gradient study were 6.1 m wide and varied in length from 128 to 171 m and were 6.1 m wide. Studies were conducted in two semi-arid, irrigated locations in Oregon (Hermiston and Madras) and utilized the same susceptible and resistant varieties as the local dispersal study. An area 1.52 m by 1.52 m in each plot was sprayed with water, inoculated with a 1:10 mixture of PST urediniospores and talc, and covered overnight with plastic. As in the local dispersal study, the success of inoculation and lack of background infections was confirmed visually approximately 1.2 latent periods after inoculation. After approximately 2.5 latent periods, disease severity in two 0.3 m sections of plants was assessed at 0.9, 1.5, 2.1, 3.0, 4.6, 6.1, 9.1, and 12.2 m from the center of the focus, and every 6.1 m further from the focus, assessing a single designated leaf position in a given field and year. While disease severity was assessed in the upwind and downwind directions, only the downwind sites were included in this study.

Long-distance dispersal data unaffected by outside sources of inoculum are highly difficult to acquire. However, I were able to take advantage of data from an regional study conducted from 1954-1957 on the island of St. Croix in the United States Virgin Islands, although only the most complete 1956-57 field study is included here. Though stripe rust and stem rust are caused by different *Puccinia* species, urediniospores are similar in size, and the epidemiology of the two disease are very similar (Roelfs 1989). Despite their respective names, each species can infect both leaves and stems of wheat.

Near the northeast edge of the island, a 2.5 ha field of wheat cv. Baart, which is susceptible to stem rust of wheat (SR) was inoculated with *Puccinia graminis* f.sp. *tritici*, causal agent of SR. The prevailing winds are from northeast to southwest. Ten downwind sites were planted with equal parts wheat cv. China and Baart. Their locations were 0.48, 2.09, 2.73, 4.34, 6.27, 6.92, 8.52, 10.62, 13.35, and 15.12 km from the focus. Fields were routinely checked visually, and once the first rust lesions appeared, twenty to thirty culms in all downwind locations were surveyed every two to three days. Twenty-six days after the initial 2.5 ha focus was inoculated, at the tail end of the second latent period, rust was observed in downwind sites up to 10.62 km from the source. Up to ten pustules per culm were recorded, above which severities were estimated, in each field. These disease severities were converted to estimated lesion counts as in the fieldwide dispersal study.

Regression and Dispersal Kernel

The modified inverse power regression (Mundt 1989),

$$y = a * (x + c)^{-b} \qquad \text{Eq. 1,}$$

in which a is the amount of disease at the source, b is the shape of the dispersal gradient, and c is an off-set, allowing the amount of disease at the source to be quantified, hypothesized to be related to focus width (Aylor 1987; Mundt and Leonard 1985; Mundt 1989), was fit to all dispersal data. Eq. 1 has been shown to fit aerial dispersal in relatively homogenous environments, particularly relative to the exponential distribution (Sackett 2004; Aylor 1987). As in previous studies (Brophy and Mundt 1991; Sackett and Mundt 2005a) I used the half-width of the inoculated focus for *c*. An exception was for data sets incorporating individual plant data. For these data sets, the modified inverse-power regression (eq. 1) was fit by iteratively increasing the c value from eq. 1, and taking the $\log_{10}$ of the explanatory variable and of the response variable, and fitting them by simple linear regression. The model returning the

highest R^2 value was considered the best fit when that model was within an order of magnitude of the half-width of the source focus.

The local and fieldwide dispersal dataset were combined by normalizing lesion counts to the one distance that was common to both data sets (0.914 m) to account for the differing levels of initial inoculum in the two trials Kingsolver et al. (1984) reported pustules per culm at low severities, and severity as a percentage at higher severities describing the regional dispersal. The severities were converted to estimates of pustule counts, using their recommendation of 10 pustules = 1% severity. The regional dataset was normalized to the combined local and fieldwide dataset by dividing all observations by the result of inputting their closest sampling point outside the focus, 2730 m, into eq. 1.

Modeling

Two models were used to examine the spread over time of epidemics following the combined normalized disease gradients described above. EPIMUL (Kampmeijer and Zadoks 1977) was used to simulate disease spread in two-dimensions, EPIMUL has since been translated into more efficient programming languages and modified over the years (Mundt and Leonard 1986; Lannou and Mundt 1996b; Sackett and Mundt 2005b; Boudreau and Mundt 1994) to allow for different dispersal kernels, incorporate lesion expansion, partial resistance, flexibility of host and initial inoculation arrangements, alter pathogen reproduction over time, and to provide both deterministic and stochastic options for inoculum dispersal. This SIR (susceptible-infectious-removed) model establishes spatial host compartments, each of which can be assigned different values for 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 infection sites sequentially through the unoccupied, latent, infectious, and removed categories. Propagules produced from each compartment are dispersed to all others on a daily time-step via the chosen dispersal kernel.

Due to limitations in the maximum array size, EPIMUL was unable to simulate distances beyond 1000 host units, so a secondary model was developed to simulate disease one-dimensionally over the distances covered in the fieldwide and regional studies. I developed an SLIR (susceptible-latent-infectious-removed) time-step model in MATLAB (MATLAB Release 2016a 2016), with four interacting arrays representing a field of locations x from 1 to n at all times t from 1 to T: p, the susceptible, uninfected fraction of each compartment; u, the latently infected fraction, with a latent period of l days; v, the infectious, actively sporulating fraction, with an infectious period of d days; and w, the removed fraction (Fig. S4.1). Each compartment was assumed to contain m sites available for infection. An initial infection or infections were placed at location x at time 0 to initialize the epidemic. At each subsequent time t, the fraction of each compartment latently infected was given by the following set of equations:

$$u(t+1,x_i,1) = \left(\sum_{j=1}^{n} k(x_{i,}x_j) * r_0 * \left(v(t,x_j)\right)\right) * \left(m - \sum_{d=1}^{l} u(t,x_i,l_d) - \sum_{s}^{d} v(t,x_i,s_d) - w(t,x_i)\right) \quad \text{Eq.2}$$

for newly arising latent infections, and

$$u(t+1,x_i,j+1) = u(t,x_i,j) \quad \text{Eq. 3}$$

for all days j from 1 to $l-1$. The dispersal k from a given source x_j to destination $x_i$followed the modified inverse power function eq. 1, as a function of the distance from the source $|x_j - x_i|$. r_0 is the daily multiplication factor (DMFR), defined as the number of effective spores produced from a single infection during a single day.

The actively infectious fraction was given by the equations

$$v\left(t+1,x_{,i},1\right) = u(t,x_i,j_l) \quad \text{Eq. 4}$$

and

$$v(t+1, x_i, j+1) = v(t, x_i, j) \quad \text{Eq. 5}$$

for all days j from 1 to $d-1$. The removed fraction was defined by

$$w(t+1, x_i) = v(t, x_i, j_d) \quad \text{Eq. 6}$$

The healthy fraction of each compartment was all tissue not in one of the previous three arrays:

$$p(t+1, x_i) = m - \sum_{d=1}^{l} u(t, x_i, l_d) - \sum_{s}^{d} v(t, x_i, s_d) - w(t, x_i). \quad \text{Eq. 7}$$

Validation of the model

Input parameters from the fieldwide and local primary disease gradients were used in the SLIR model to validate the model's ability to reconstruct the observed levels of disease from the field experiments. To simulate the fieldwide primary disease gradient, the dispersal gradient, in which the c was set to half the width of the focus,

$$y = 40.5 * (x + 0.762)^{-2.39} \quad \text{Eq. 8,}$$

and the initial disease severity in the focus of 6.2% were derived from the Madras 2002 data. Input parameters used to subsequently model WSR spread in EPIMUL (Sackett and Mundt 2005b), an infectious period of 14 days, a latent period of 17 days, and a DMFR of 5, were input to the MATLAB model, considering each compartment to represent a 1.524 m block of wheat plant with an m of 18000.

To examine the appropriateness of examining dispersal as a function of distance in one-dimension, a comparison between epidemic velocity given a single row of 100 1.524 m compartments with 50 empty rows on either side was compared with the epidemic velocity of a single row of 100 1.524 m compartments in which all other rows in the field contained identical susceptible compartments. The

furthest compartment in this row at which at least 50% severity was observed was log-transformed and plotted against the day for days 44, 56, 70, 84, and 98.

Simulating Full-Season Epidemics

The disease gradients were modeled with compartment sizes of 0.0254 m, representing the width of an average wheat plant, 1.524 m, the size of the focus used in the fieldwide studies, and 152.4 m, approximately the width of the focus in the regional study. Fitting eq. 1 was accomplished by setting the c value, which is likely related to the size of the source, and can affect the steepness of the b parameter to half the width of the focus (Aylor 1987; Chamecki, Dufault, and Isard 2012).

Two sets of epidemics were simulated across fields from a single focus comparing the local and fieldwide spatial scales, and the fieldwide and regional spatial scales. The local and regional spatial scales could not be directly compared due to computational limitations. The local and fieldwide spatial scale comparisons had a single initial disease focus of 1.524 m, as used in the fieldwide dispersal study, and the fieldwide and regional comparisons had a disease focus of 152.4 m, approximately the width of the focus used in the regional study (Table 1). The length of the epidemic was 98 days, with a latent period and an infectious period of 14 days each. To compare the effects of scale in the local dispersal dataset with the field wide dispersal dataset, the compartment size treatments, the approximate focus size of the local dataset was compared with the fieldwide, and the fieldwide focus width was compared with regional focus width. The maximum number of infections, m, was scaled based on 300 infections per 0.0254 m, approximately the size of an individual wheat plant. The resulting dispersal gradients at the final day of the simulation were fit by the modified inverse power distribution (eq. 1), which returned the proportion of the total effective spores deposited within a given distance from the source. Figs. were produced with MATLAB and with the base and ggplot2 (Wickham, Chang, and Wickham 2013) package in R (R Development Core Team 2015). In addition to the simulations governed by the best-fit

normalized modified inserse power distrubiton, combined across all spatial scales, full season epidemics were simulated using the non-normalized dispersal kernels from a single spatial scale. Simulations comparing dispersal over 91.44 m fields, as above, were run with dispersal kernels from the local dataset, and from the fieldwide dataset. Simulations comparing dispersal over 10.62 km fields, as above, were run with dispersal kernels form the fieldwide dataset, and from the regional dataset.

Results

Disease gradients

Individual spatial scale datasets were well-fit by the iterative best-fit *c* modified inverse power distribution at the local (Fig. 1a) and fieldwide (Fig. 1b,) spatial scales. However, the regional dataset, containing only four observations in the tail of the epidemic outside the source, was poorly fit by the best-fit *c* modified inverse power distribution, and may have been greatly influenced by an "outlier" at 3.9 km. Therefore, the regional dataset was fit by the modified inverse power distribution with a half-width *c* (Fig. 1c). The normalized local, fieldwide, and regional data combined in all two-way comparisons (Fig. 2a - 2c) were fitted well by the modified inverse-power model containing the *c* value that returned the highest R^2, as was the data set combining observations at all three spatial scales (Fig. 2d). The kernel slopes (*b*-values) ranged from 1.91 to 2.69, and values of *c* were proportional to the size of the smallest host unit included in the regression (Figs. 2 and 3).

The one-dimensional SLIR model was able to closely recreate empirical disease spread data. The fieldwide primary disease gradient from Madras 2002 was recreated using both 0.0254 m (Fig. 3a) compartments and 1.524 m compartments (Fig. 3b). The dispersal kernel was governed by the modified inverse-power distribution fit to the combined normalized data across all three spatial scales, which when modeling by 0.0254 m compartments, took the form

$$y = 0.23 * (x + 0.14\ m)^{-2.39}$$ Eq. 9.

A more biologically appropriate c value, half the width of the compartment was used to model the epidemic when using a compartment size of 1.524 m:

$$y = 0.23 * (x + 0.76\ m)^{-2.39}$$ Eq. 10

and when using a compartment size of 152.4 m:

$$y = 0.23 * (x + 76.2\ m)^{-2.39}$$ Eq. 11

Disease spread across a single row in a two-dimensional array simulated in EPIMUL in which all compartments in all rows were susceptible exhibited a greater velocity than did a simulation using the same input parameters to simulated disease spread across a single row in which all compartments other than in a single central row did not contain susceptible plants (Fig. 4). Disease spread over time simulated one-dimensionally was very similar over the course of seven latent periods when comparing compartment sizes of 1.524 m and 0.0254 m (Fig. 5 - 13) across initial prevalences (P_0s) of 1, 5, and 10 %, and reproductive rates per latent period (R_0s) of 14, 35, and 70 in both comparisons between local and fieldwide spatial scales, as evidenced by similar AUCs at the end of full-season epidemics (Fig. 14) out to 91.44 m. This closeness of AUCs from the full-season epidemic simulations across spatial scales also was observed in all combinations of R_0s and P_0s in comparisons of fieldwide and regional (Fig. 7, Fig. 15) to 10.6 km. The closesness of simulations using single spatial scale non-normalized dispersal gradients over the course of a full season of disease spread was shown by AUC, although this relationship was not as close as observed using the same normalized combined dispersal kernel as above (Figs. 16 and 17).

Discussion

Here I have demonstrated the similarity of gradients of diseases caused by aerially dispersed small propagules across spatial scale using cereal rusts as a model system. I have demonstrated that the gradients of cereal rust lesion counts are well fitted by the same modified inverse power function at the local, fieldwide, and regional scales (Fig.2). As noted by Lannou et al. (Lannou et al. 2008) large inoculum sources are required to track disease gradients at long distances, but large inoculum sources make it impossible to obtain useful data as short distances. These results have the potential to aid in our ability to understand the dispersal gradients of small, passively wind-dispersed particles by combining data sets to provide a common function a cross a large spatial extent, thereby enabling estimation of both local dispersal and the tails of dispersal gradients, which are very difficult to quantify in single-scale empirical studies. I were able to normalize our local and field-level data to data at a common distance in both data sets. Because there was no common distance between field-level and regional-level data, the field-level data were thus extrapolated to the near distance from the source in the regional data set for normalization. Though not ideal, this procedure resulted in a linear relationship across the three data sets on the transformed scale (Fig. 2).

This study corroborates the finding that propagule pressure at the outbreak along with a modified power distribution function can predict epidemics to a great extent (Severns, Sackett, and Mundt 2015). The power-law has been shown previously to be scale-independent and has been used to model fractal phenomena in which similar processes are repeated across scales (Gisiger 2001). The spatial scale at which systems are examined can have significant impacts on the observations in ecological studies, including in dispersal studies. Kristensen, Barro, and Schellhorn (Kristensen, Barro, and Schellhorn 2013) found that the insect *Eretmocerus hayati* dispersed differently at the local, field, and landscape scale, and that measuring or modeling its dispersal from solely a local scale would generate observations or predictions of significantly slower dispersal than if the larger spatial scales

were included. The rationale given for this finding is the differing dominant dispersal mechanisms of *E. hayati* at differing spatial scales, in which diffusion plays a larger role at the local and field scales, but wind-advection is the primary driver of dispersal at the landscape scale. This may be the case for a significant fraction of flying arthropods, and may be a significant driver of insect invasions into new regions (Compton 2002; Wiktelius 1981).

It is possible that multiple mechanisms of dispersal act on *Pst*. It has been shown that *Pst* urediniospores can be dry-dispersed aerially by wind, can be splash-dispersed by rain drops or irrigation, or can be dispersed aerially, released from the uredinia from water drops impacting wheat leaves, which transfer that force to the uredinia (Fitt, McCartney, and Walklate 1989; Sache 2000). The mechanism of dispersal has been shown to have a significant role in the shape of a particle's dispersal gradient (Meredith 1973; Fitt, McCartney, and West 2006). However, where flying insects are capable of travelling many meters in the absence of wind, it may be reasonable to believe the vast majority of *Pst* urediniospores are deposited well within a meter in the absence of wind (Farber, Medlock, and Mundt 2016). Therefore, it may be possible to extrapolate beyond the extent of a dispersal study (or a study of the primary disease gradient acting as a proxy for dispersal) given that it samples a distance such that a sufficient portion of the dispersal gradient can be captured. I have demonstrated this with the closeness of fit of these primary disease gradients of cereal rusts across the local, field, and regional scale, and our ability to predict similar epidemics using differing compartmental sizes.

This research could help to ascertain the number of progeny uredinial lesions one could expect at a given distance from a source uredinium or uredinia. Relatedly, this could predict the number of plants or the area over which a field manager would have to search to find a single progeny uredinial lesion at a given distance from a single source lesion (Table 4). This could give some insight into the likelihood of invasions of passively aerially dispersed pathogens into new fields or region (Clark, Lewis,

and Horvath 2001), and could aid field managers in understanding the likelihood of significant levels of propagules reaching their fields given knowledge of their neighbors' inoculum loads. Use of our combined data (Fig. 2) shows that, though a single uredinum could result in disease at a distance 10 km away, it would be nearly impossible to find even with extensive sampling procedures. Thus, a single infection might be expected to spread to very long distances in an extensively grown agricultural crop. In less extensively grown crops or in sporadic populations of native plants, it is possible that infection would not spread owing to stochastic events.

A challenge of implementing this research is the scale-dependent nature of the *c* parameter. While this improves the fit compared to the inverse-power distribution of the primary disease gradient of WSR (Sackett and Mundt 2005b) and allows for log-transformation at the source location, its biological interpretation is not entirely clear. It has been considered a function of the width of the focus (Aylor 1987; Mundt and Leonard 1985; Mundt 1989) and has been set to half the width of the focus (Sackett and Mundt 2005a), but the relationship does not appear to be consistent, as the best-fit *c* fit to the local disease gradient was 0.14 m (Farber, Medlock, and Mundt 2016). This may be due, in part, to the fact that local scale data were derived from an agricultural planting in which there was much less distance between plants within rows than between rows (Farber et al., 2016).

A limitation of this model is the assumption of homogeneity of the hosts in their contribution to the epidemic over time and locations within a given compartment. Plant architecture can influence dispersal of their pathogens (Costes et al. 2013) and have been included in models (Gigot et al. 2014; Baccar et al. 2011; Garin et al. 2014), and plant phenology can influence its susceptibility to infection, effectively altering the R_0 (Johnston et al. 2016; Grulke 2011; Panter and Jones 2002). While the dispersal gradient of aerially dispersed pathogens may be independent of scale as a function of distance,

further exploration is needed to examine three-dimensional dispersal across spatial scales, particularly in cases such as wheat in which there is a significant vertical gradient in susceptibility to infection by *Pst.*

I used a one-dimensional simulation of disease spread, which functioned well to recreate empirical observations of primary disease gradients. However, a spatially explicit two-dimensional model could offer significant improvements, particularly when compartments lack homogeneity, such as could be the case with row structure or plant architecture heterogeneity (Suzuki and Sasaki 2011; Bohrer et al. 2008) or anisotropic dispersal (Savage et al. 2011). However, two-dimensional models are much more computationally intensive and were not feasible to examine dispersal across such large arrays at this time. The position of the epidemic front is expected to increase exponentially over time for an epidemic driven by power law dispersal (Madden, Hughes, and van den Bosch 2007; Christopher C. Mundt et al. 2009). This was the case for simulation of both single rows and 100 by 100 blocks, though the slope was greater for the latter. This suggests that same qualitative patterns were found, but epidemics progressed faster in the two-dimensional epidemics because there were more compartments to contribute inoculum.

The results of this study can have significant impacts on future ecological studies. Prior studies have found significant changes in biodiversity and population dynamics at different spatial scales (Martiny et al. 2011). However the relationships between spatial scale and diversity are not always clear; plant species diversity was found to be correlated positively with environmental heterogeneity, but spatial scale did not have a predicTable effect on that relationship (Lundholm 2009). This research suggests that spatial scale does not appear to have a significant effect on the dispersal gradients of cereal rusts. This may be due to the dominant mechanism driving dispersal, wind advection, to be the same at each spatial scale considered. These relationships might not hold true for intra-plant dispersal, for example. However, this study provides evidence that primary disease gradients were similar across

spatial scales that range from the intensification of an initial outbreak area (local) to invasions into new regions.

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Table 4.1 Summary of all epidemic simulations. All simulations were 98 days long, with latent period and infectious period 14 days each. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

Run	Length of field (m)	Compartment size (m)	*m*	N	R0	P0
1	91.44	0.0254	300	3600	70	0.01
2	91.44	0.0254	300	3600	35	0.01
3	91.44	0.0254	300	3600	14	0.01
4	91.44	0.0254	300	3600	70	0.05
5	91.44	0.0254	300	3600	35	0.05
6	91.44	0.0254	300	3600	14	0.05
7	91.44	0.0254	300	3600	70	0.1
8	91.44	0.0254	300	3600	35	0.1
9	91.44	0.0254	300	3600	14	0.1
10	91.44	1.524	18000	60	70	0.01
11	91.44	1.524	18000	60	35	0.01
12	91.44	1.524	18000	60	14	0.01
13	91.44	1.524	18000	60	70	0.05
14	91.44	1.524	18000	60	35	0.05
15	91.44	1.524	18000	60	14	0.05
16	91.44	1.524	18000	60	70	0.1
17	91.44	1.524	18000	60	35	0.1
18	91.44	1.524	18000	60	14	0.1
19	10620	1.524	18000	6969	70	0.01
20	10620	1.524	18000	6969	35	0.01
21	10620	1.524	18000	6969	14	0.1
22	10620	1.524	18000	6969	70	0.05
23	10620	1.524	18000	6969	35	0.05
24	10620	1.524	18000	6969	14	0.05
25	10620	1.524	18000	6969	70	0.05
26	10620	1.524	18000	6969	35	0.01
27	10620	1.524	18000	6969	14	0.1
28	10620	152.4	180000	70	70	0.01
29	10620	152.4	180000	70	35	0.01

30	10620	152.4	180000	70	14	0.01
31	10620	152.4	180000	70	70	0.01
32	10620	152.4	180000	70	35	0.01
33	10620	152.4	180000	70	14	0.01
34	10620	152.4	180000	70	70	0.01
35	10620	152.4	180000	70	35	0.01
36	10620	152.4	180000	70	14	0.01

Table 4.2 Predicted wheat stripe rust lesions per plant after a single generation of disease spread emanating from a single source lesion, and the area over which one would be expected to search to encounter a single lesion.

Distance	Predicted lesions per plant	Expected number of plants needed to find a single infection	Expected m^2 needed to find a single infection
0	15.40768	0.064902698	4.18727E-05
0.0254	10.33557	0.096753251	6.24215E-05
0.0508	7.340963	0.13622191	8.78851E-05
0.1	4.237895	0.235966205	0.000152236
0.5	0.4046055	2.471543269	0.001594544
1	0.1015292	9.849383232	0.006354441
2	0.02246913	44.505506	0.02871323
4	0.004626611	216.1409291	0.139445761
8	0.000916402	1,091.2238	0.704015355
16	0.000177892	5,621.391418	3.626704141
32	3.41794E-05	29,257.40607	18.87574585
64	6.5331E-06	153,066.738	98.7527342
128	1.2455E-06	802,892.984	517.9954735
500	4.77561E-08	20,939,715.81	13,509.49407
1000	9.08369E-09	110,087,468.9	71,024.17348
2000	1.72752E-09	578,865,504.7	373,461.6159
5000	1.92518E-10	5,194,319,492	3,351,173.866
10000	3.66078E-11	27,316,612,653	17,623,621.07

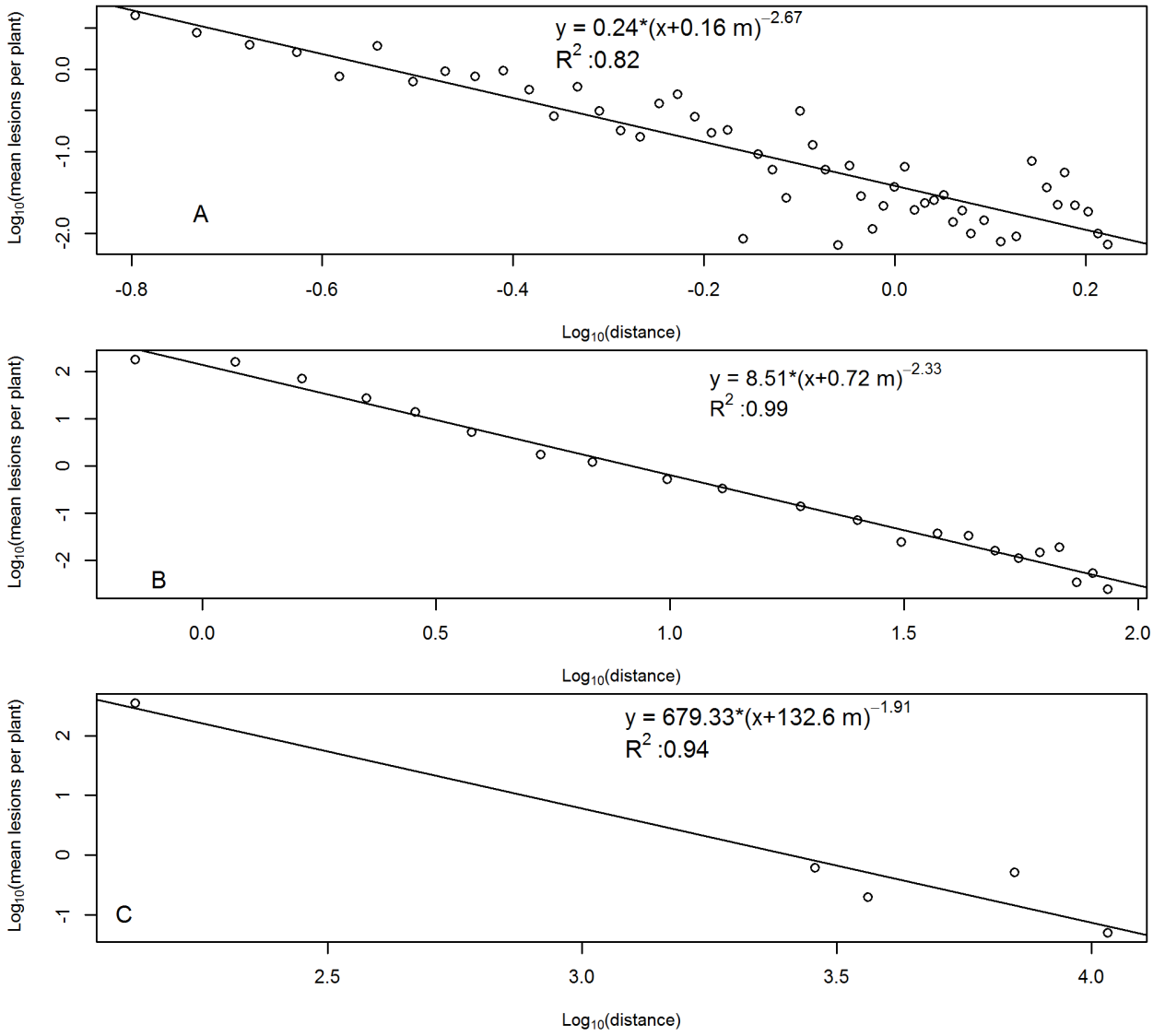

Fig. 4.1 Mean lesion counts of *Puccinia striiformis* f.sp. *tritici* as a function of distance from the source (non-normalized), fit by the modified inverse-power distribution by individual spatial scale data. a) Local, to 1.52 m; b) fieldwide, to 91.44 m; c) regional, to 10620 m.

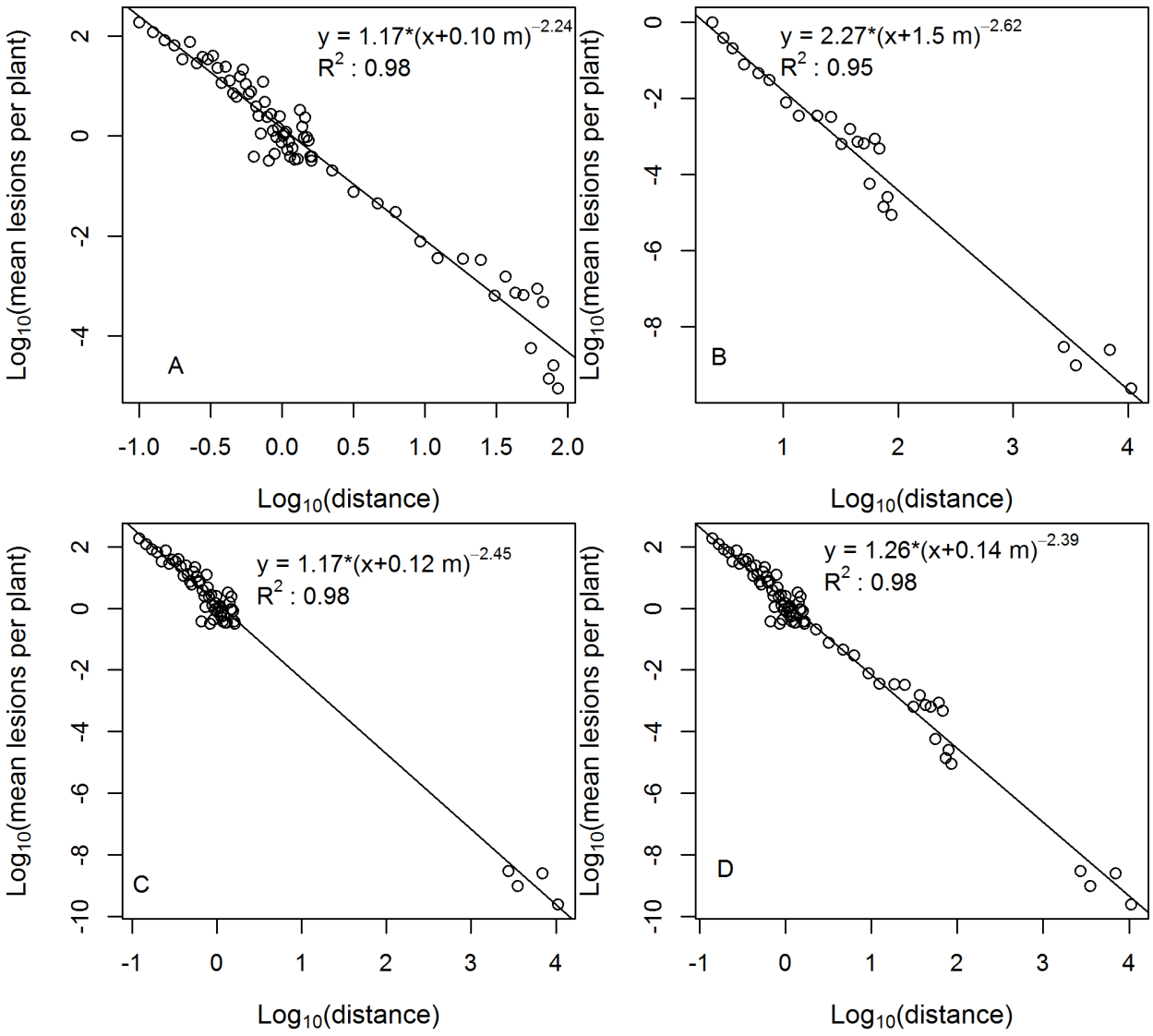

Fig. 4.2 Cereal rust (*Puccinia striiformis* f.sp *tritici* at the local and field scales, and *Puccinia graminis* f.sp. *tritici*) best-fit *c* modified inverse power distributions of combined field data normalized to the number of lesions observed or estimated at 0.9144 m. a) local and field data; b) field and regional data; c) local and regional data; d) local, field, and regional data.

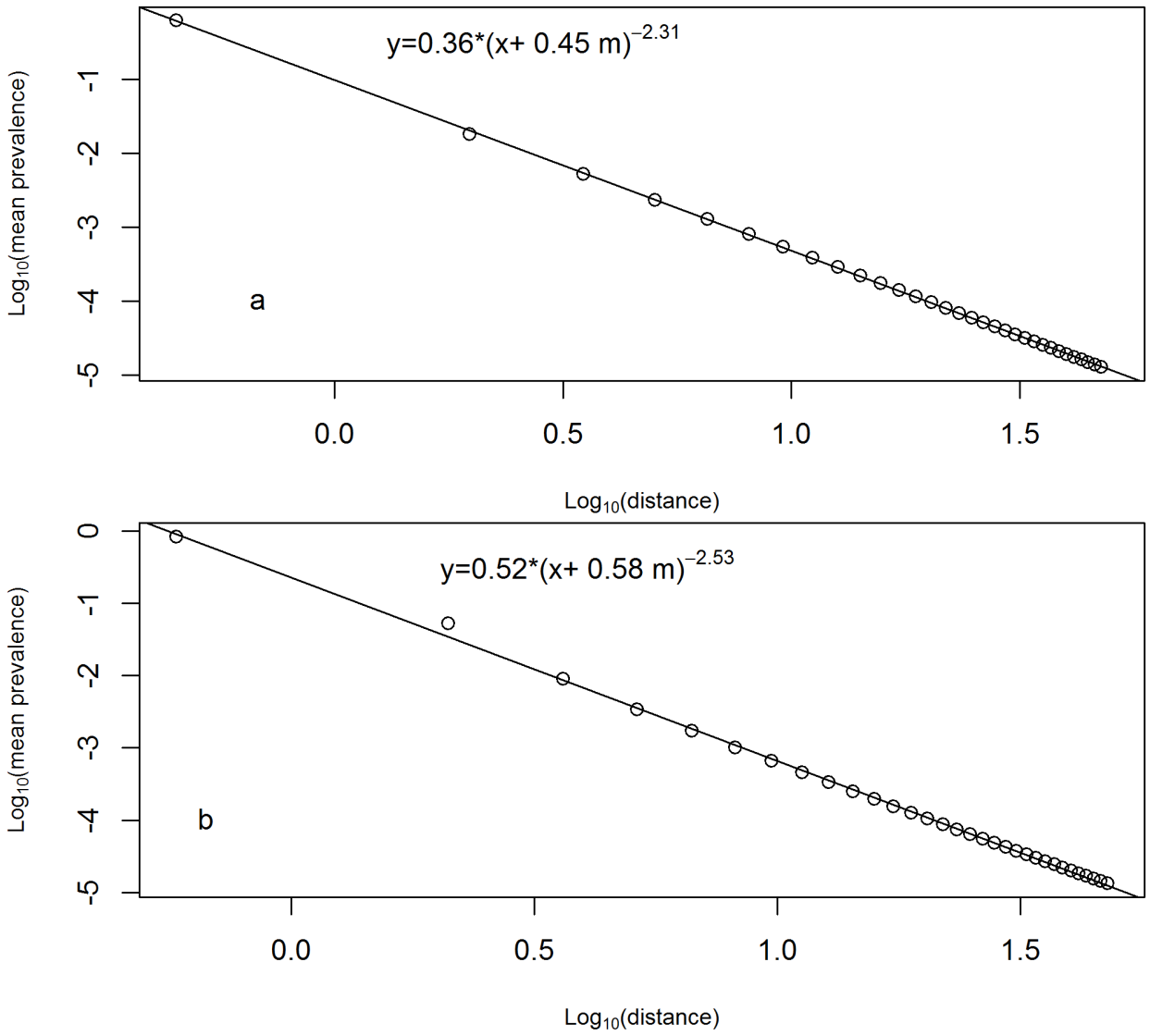

Fig. 4.3 The primary dispersal gradient fit by the modified inverse power distribution of the Madras 2002 field study (Sackett and Mundt 2005a), $y = 40.50 * (x + 0.762\ m)^{-2.39}$, simulated in MATLAB, with compartment size of a) 0.0254 m, and b) 1.524 m.

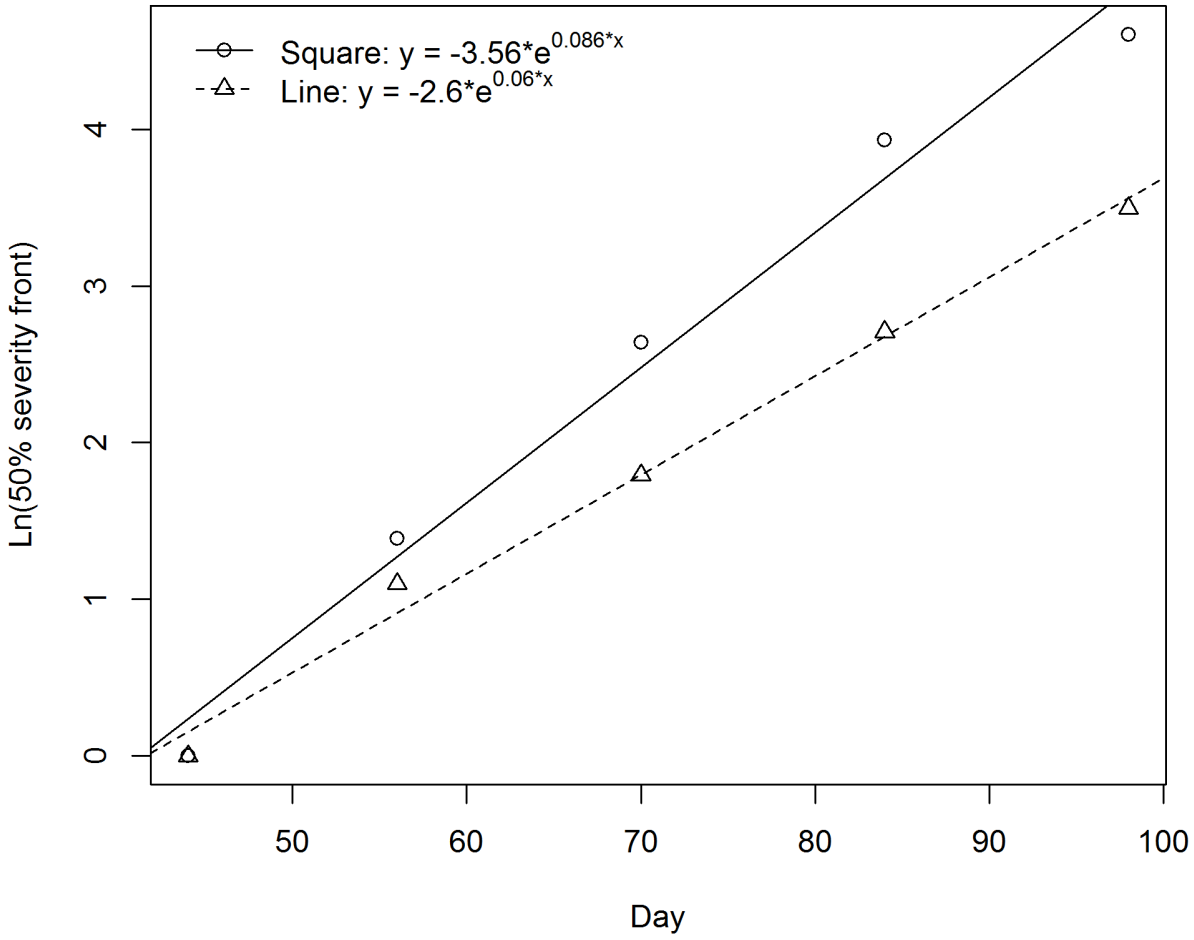

Fig. 4.4 Velocity of two identical input epidemics simulated in EPIMUL over a single row containing 100 compartments within a 100 by 100 compartment field, with a compartment size of 1.524 m by 1.524 m emanating from a single compartment at row 50 - column 1, with P_0 of 10 and R_0 of 70 over 7 generations. In 'Line,' all rows on either side of row 50 were empty, not contributing to disease; in 'Square, all rows contained susceptible plants.

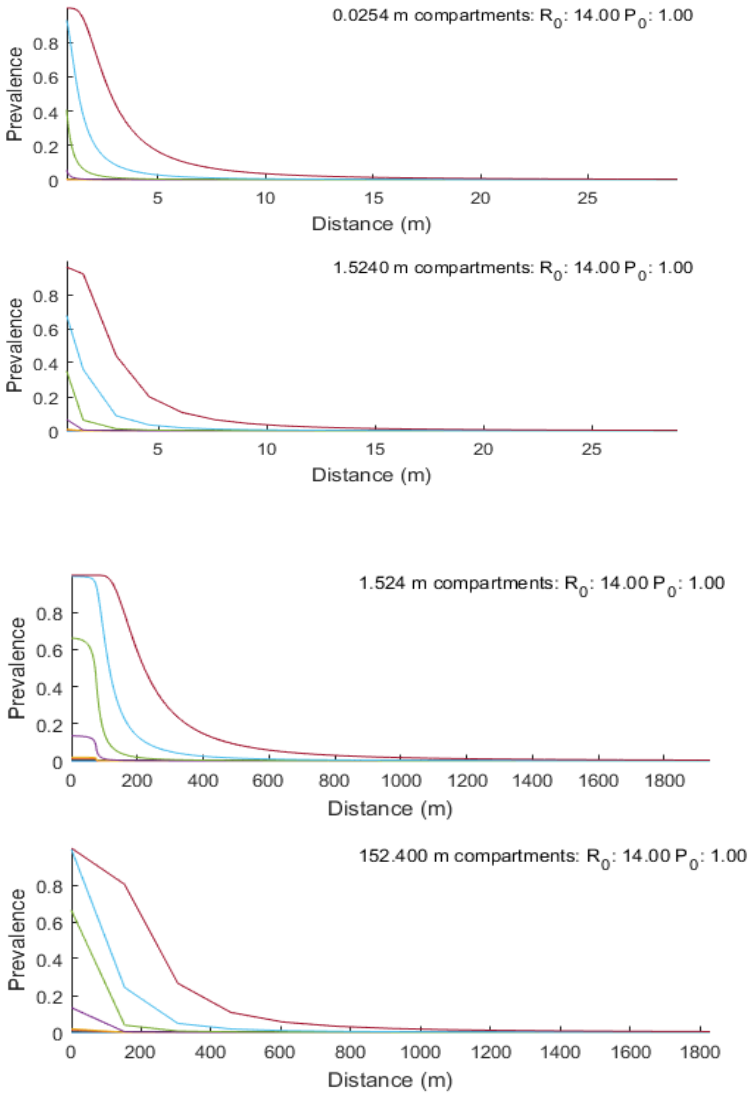

Fig. 4.5 Seven generations of disease spread with an R_0 of 14 and a P_0 from a 1.524 m focus across a 91.4 m field, with a compartment size of 0.0254 m and 1.524 m; and from a 152.4 m focus across a 10,515.6 m field with a compartment size of 1.524 m and 152.4 m. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

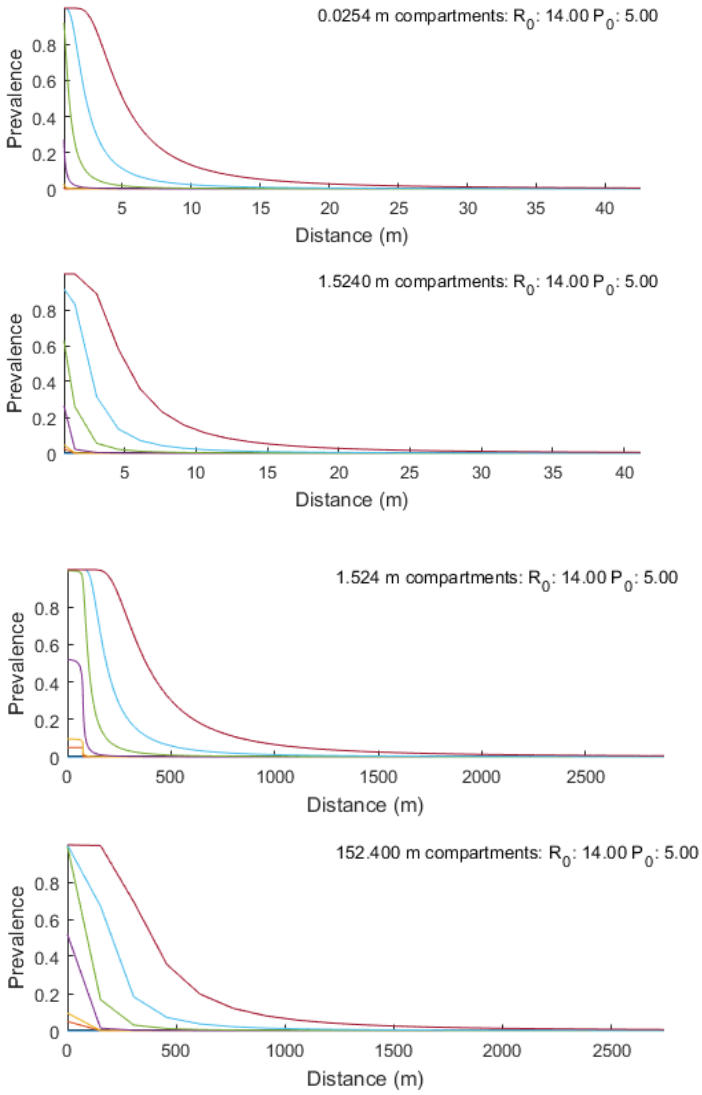

Fig. 4.6 Seven generations of disease spread with an R_0 of 14 and a P_0 of 5 from a 1.524 m focus across a 91.4 m field, with a compartment size of 0.0254 m and 1.524 m; and from a 152.4 m focus across a 10,515.6 m field with a compartment size of 1.524 m and 152.4 m. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

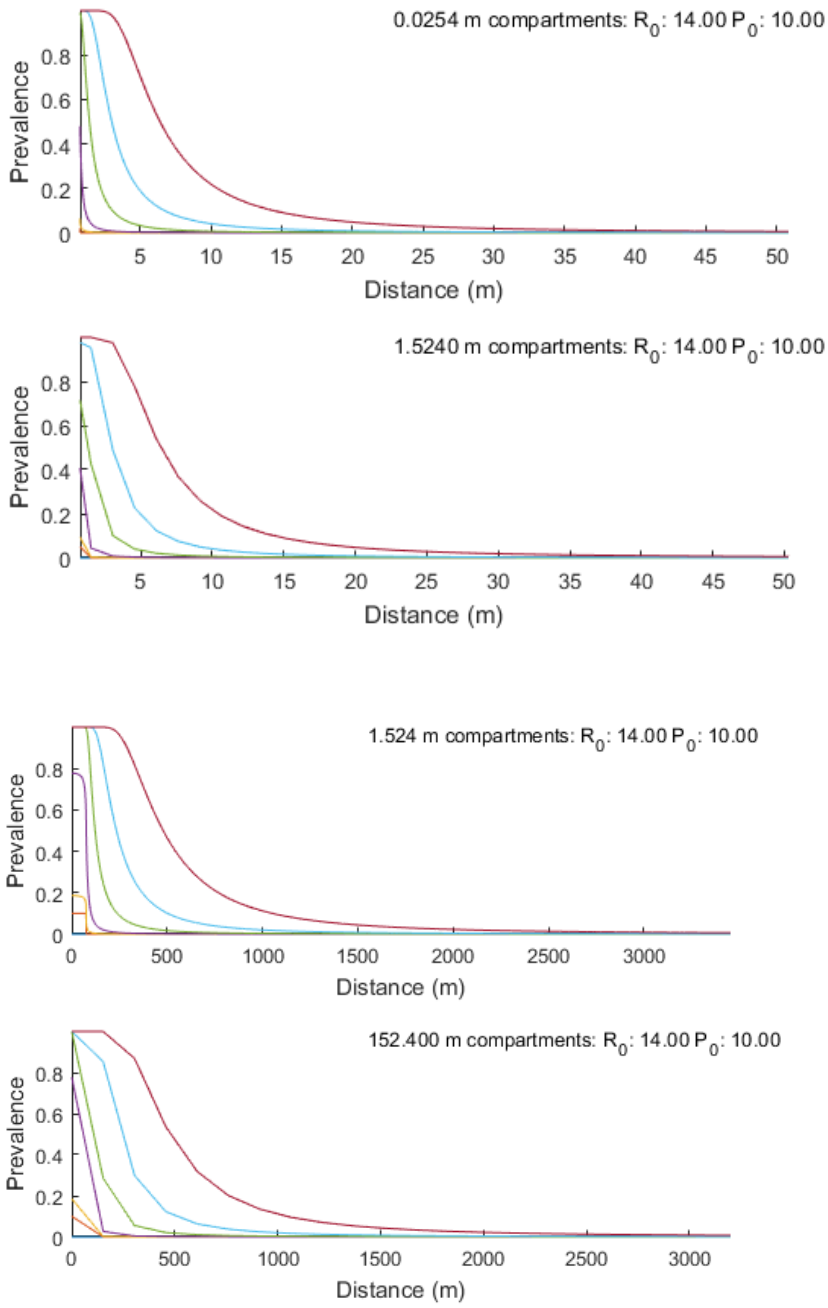

Fig. 4.7 Seven generations of disease spread with an R_0 of 14 and a P_0 of 10 from a 1.524 m focus across a 91.4 m field, with a compartment size of 0.0254 m and 1.524 m; and from a 152.4 m focus across a 10,515.6 m field with a compartment size of 1.524 m and 152.4 m. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

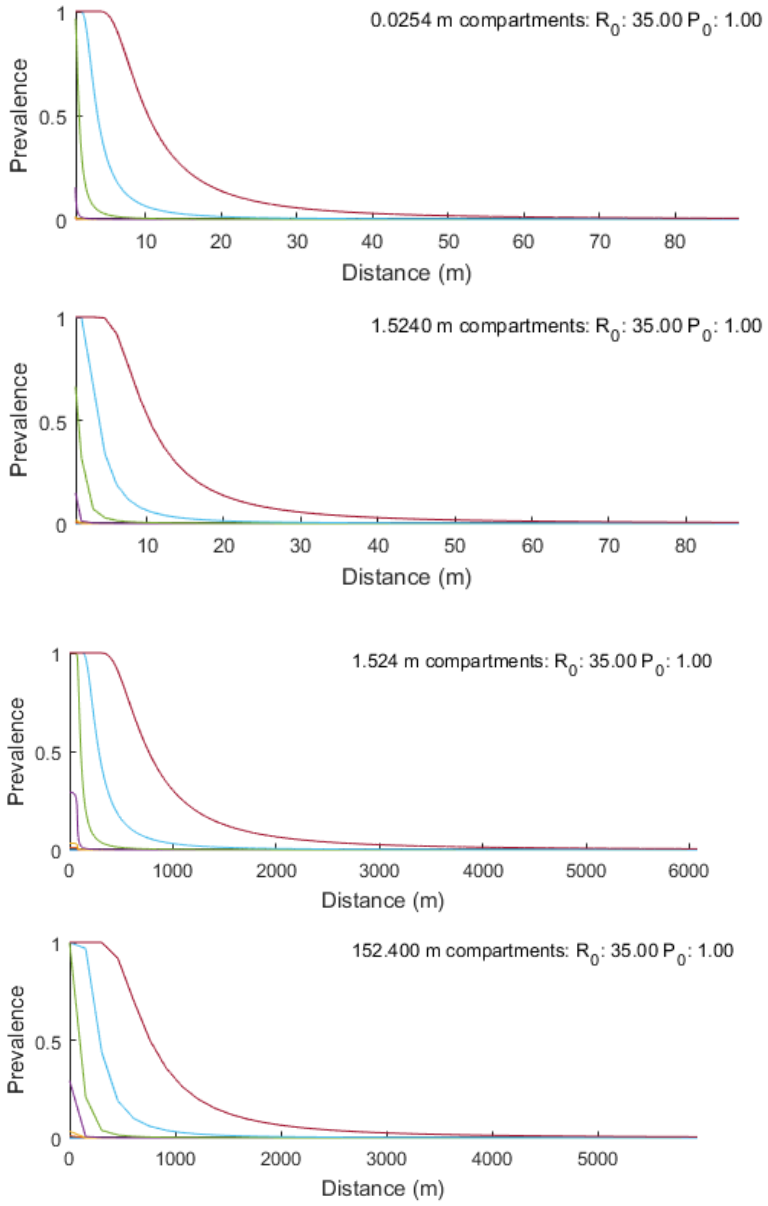

Fig. 4.8 Seven generations of disease spread with an R_0 of 35 and a P_0 of 1 from a 1.524 m focus across a 91.4 m field, with a compartment size of 0.0254 m and 1.524 m; and from a 152.4 m focus across a 10,515.6 m field with a compartment size of 1.524 m and 152.4 m. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

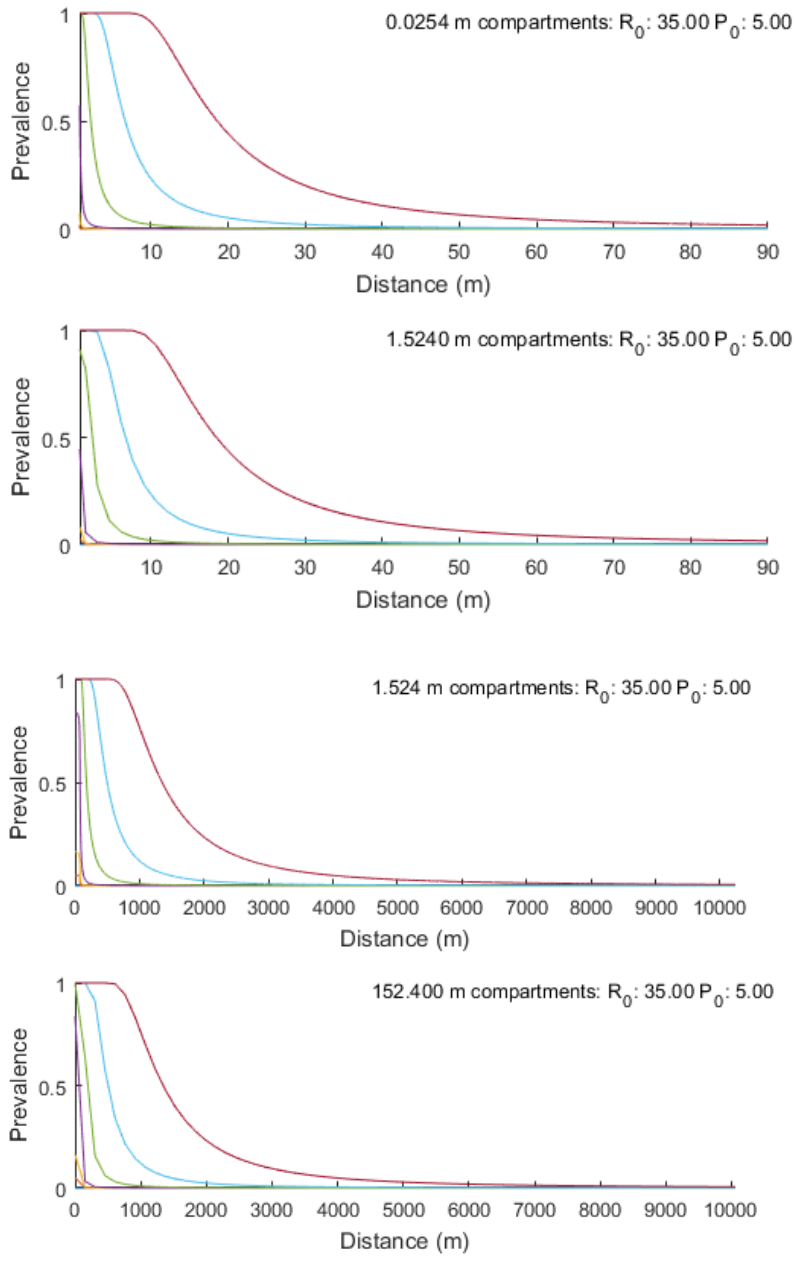

Fig. 4.9 Seven generations of disease spread with an R_0 of 35 and a P_0 of 5 from a 1.524 m focus across a 91.4 m field, with a compartment size of 0.0254 m and 1.524 m; and from a 152.4 m focus across a 10,515.6 m field with a compartment size of 1.524 m and 152.4 m. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

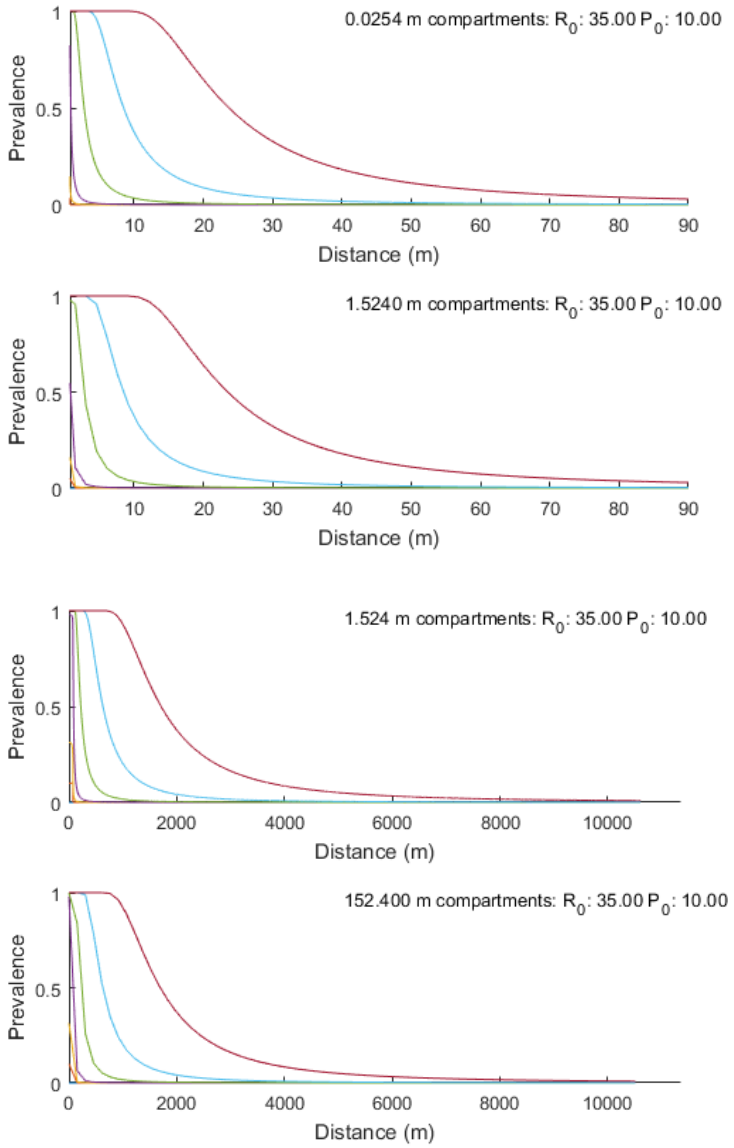

Fig. 4.10 Seven generations of disease spread with an R_0 of 35 and a P_0 of 10 from a 1.524 m focus across a 91.4 m field, with a compartment size of 0.0254 m and 1.524 m; and from a 152.4 m focus across a 10,515.6 m field with a compartment size of 1.524 m and 152.4 m. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

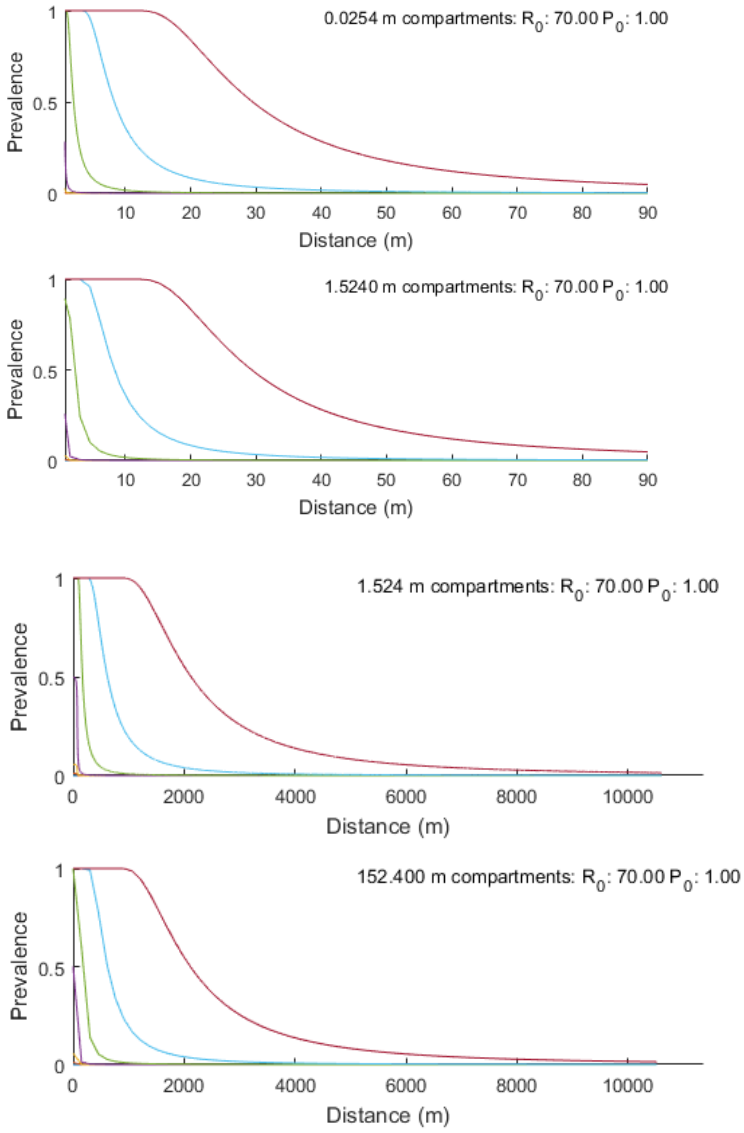

Fig. 4.11 Seven generations of disease spread with an R_0 of 70 and a P_0 of 1 from a 1.524 m focus across a 91.4 m field, with a compartment size of 0.0254 m and 1.524 m; and from a 152.4 m focus across a 10,515.6 m field with a compartment size of 1.524 m and 152.4 m. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

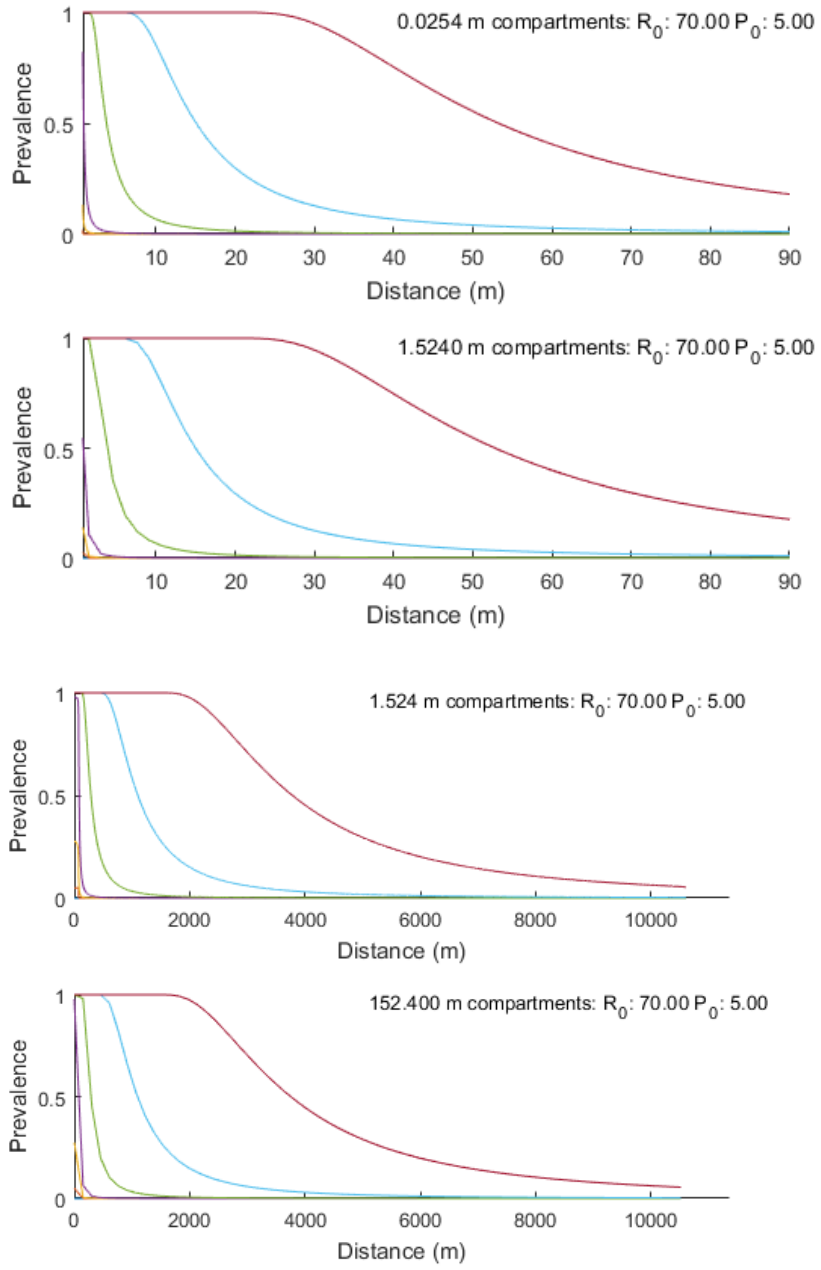

Fig. 4.12 Seven generations of disease spread with an R_0 of 70 and a P_0 of 5 from a 1.524 m focus across a 91.4 m field, with a compartment size of 0.0254 m and 1.524 m; and from a 152.4 m focus across a 10,515.6 m field with a compartment size of 1.524 m and 152.4 m. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

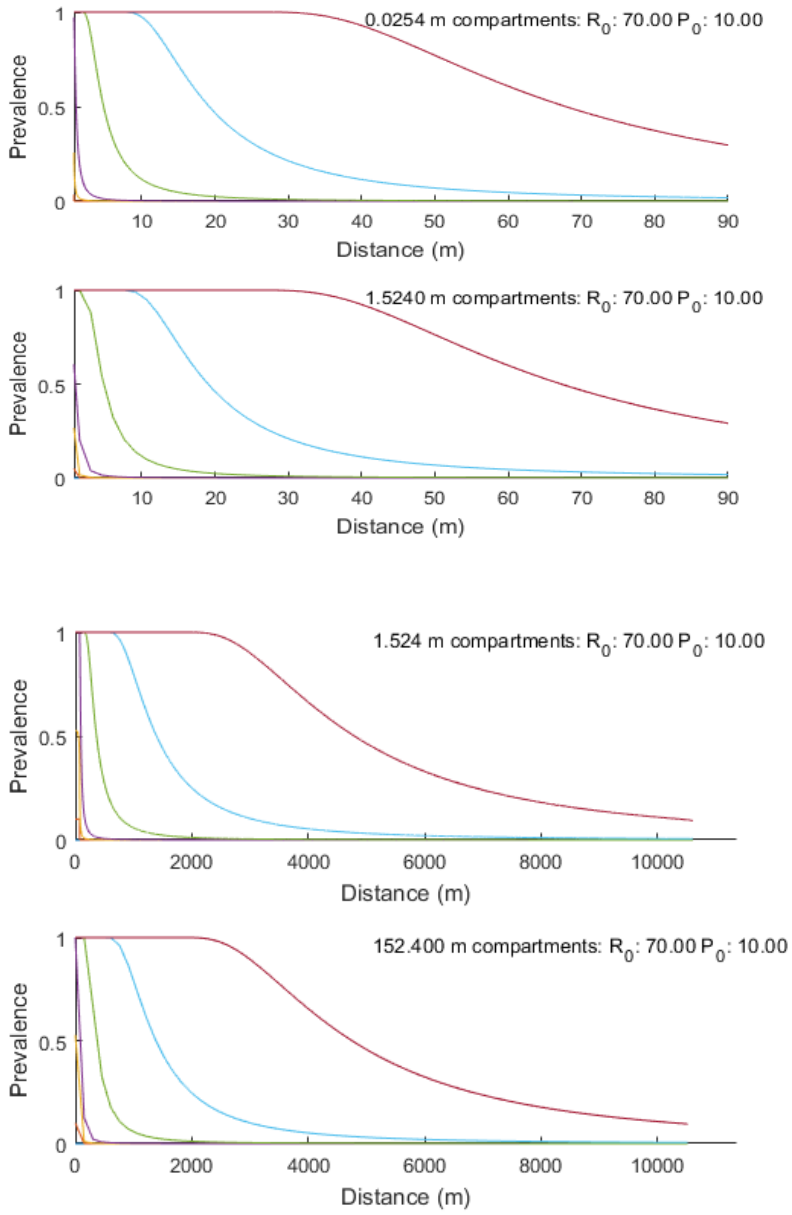

Fig. 4.13 Seven generations of disease spread with an R_0 of 70 and a P_0 of 10 from a 1.524 m focus across a 91.4 m field, with a compartment size of 0.0254 m and 1.524 m; and from a 152.4 m focus across a 10,515.6 m field with a compartment size of 1.524 m and 152.4 m. Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

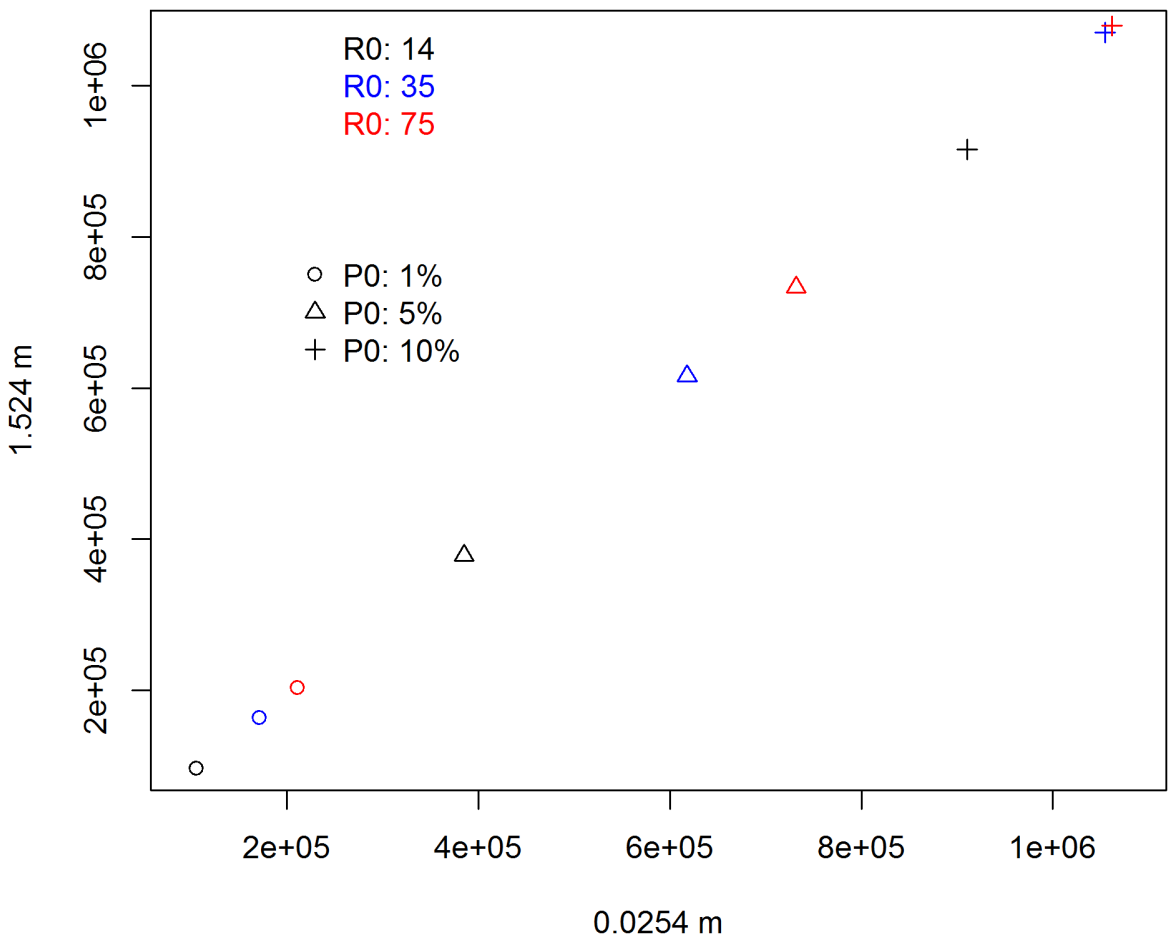

Fig. 4.14 Area under the curve (AUC) calculated for all compartments in a 91.44 m field, for all combinations of reproductive rate per generation (R_0) and initial prevalence as a percentage (P_0). Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

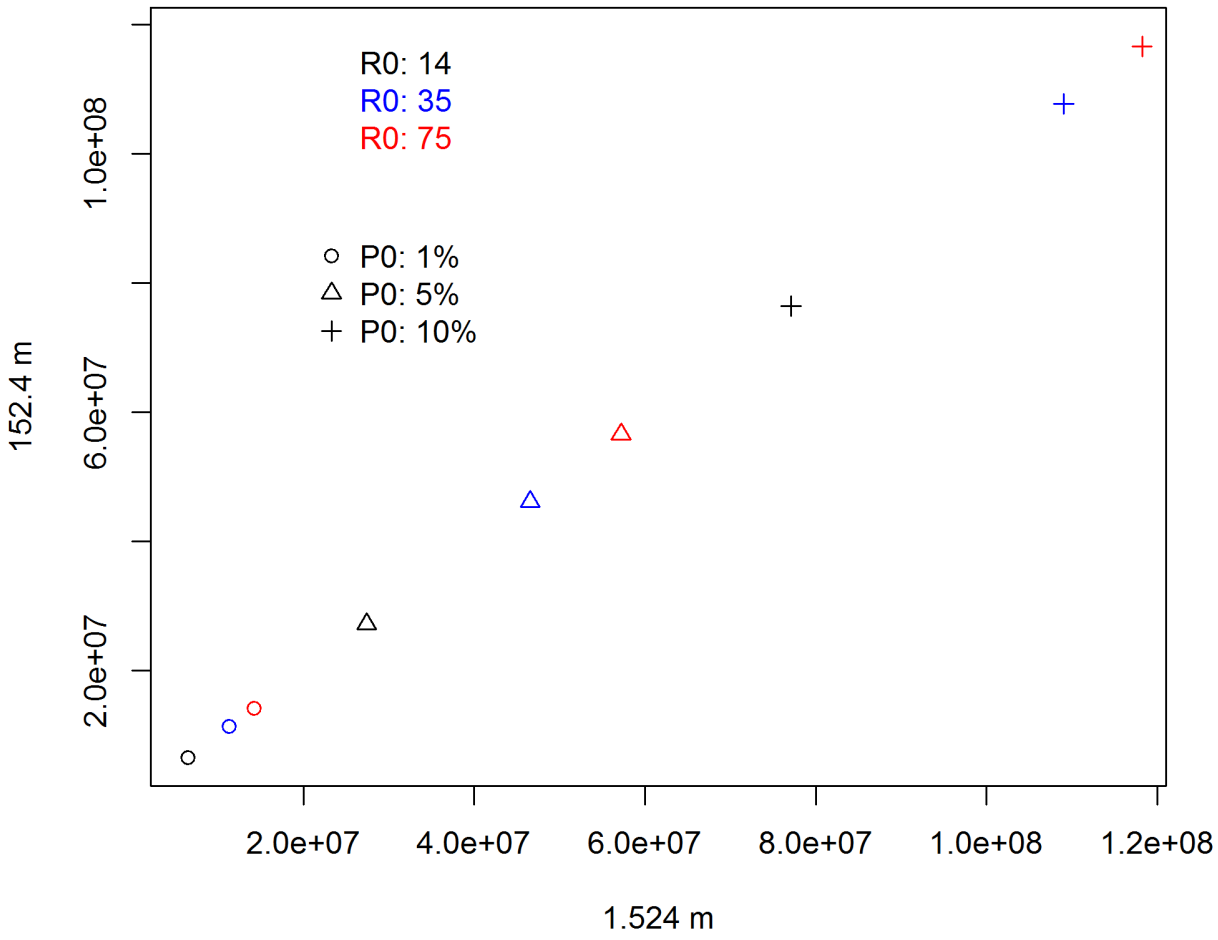

Fig. 4.15 Area under the curve (AUC), calculated for all compartments in a 10.6 km field simulation, for all combinations of reproductive rate per generation (R_0) and initial prevalence as a percentage (P_0) after 7 latent periods (day 98). Dispersal was governed by the normalized best-fit modified inverse-power distribution across local, fieldwide, and regional spatial scales, $y = 1.26 * (x + 0.14\ m)^{-2.39}$.

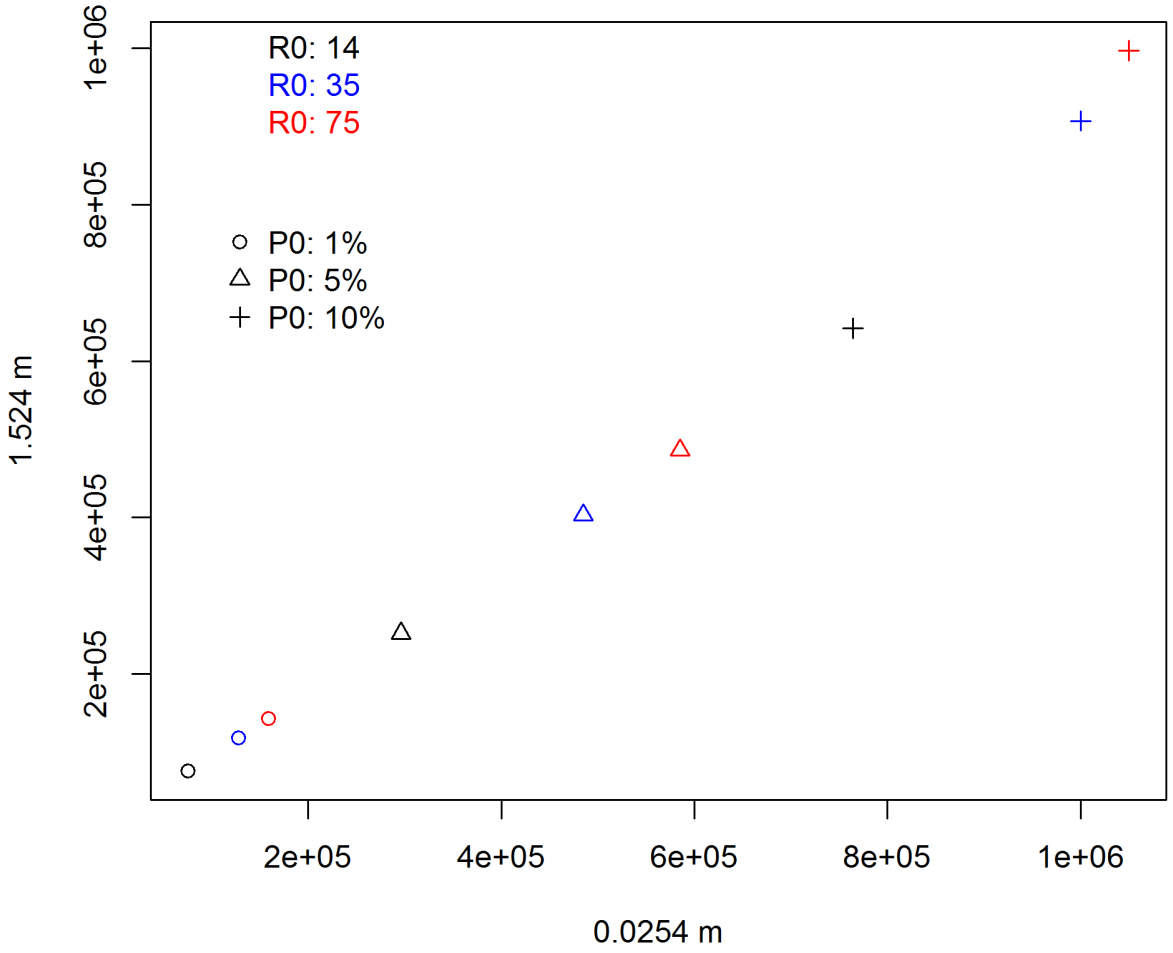

Fig. 4.16 Area under the curve (AUC) calculated for all compartments in a 91.44 m field, for all combinations of reproductive rate per generation (R_0) and initial prevalence as a percentage (P_0) after seven latent periods. Each point represents an identical simulation, with the x-coordinate being simulated with local compartment size, 0.0254 m, and non-normalized dispersal kernel, $y = 024 * (x + 0.16\ m)^{-2.67}$; and the y-coordinate being simulated with fieldwide compartment size, 1.524 m, and non-normalized dispersal kernel, $y = 8.51 * (x + 0.76\ m)^{-2.33}$.

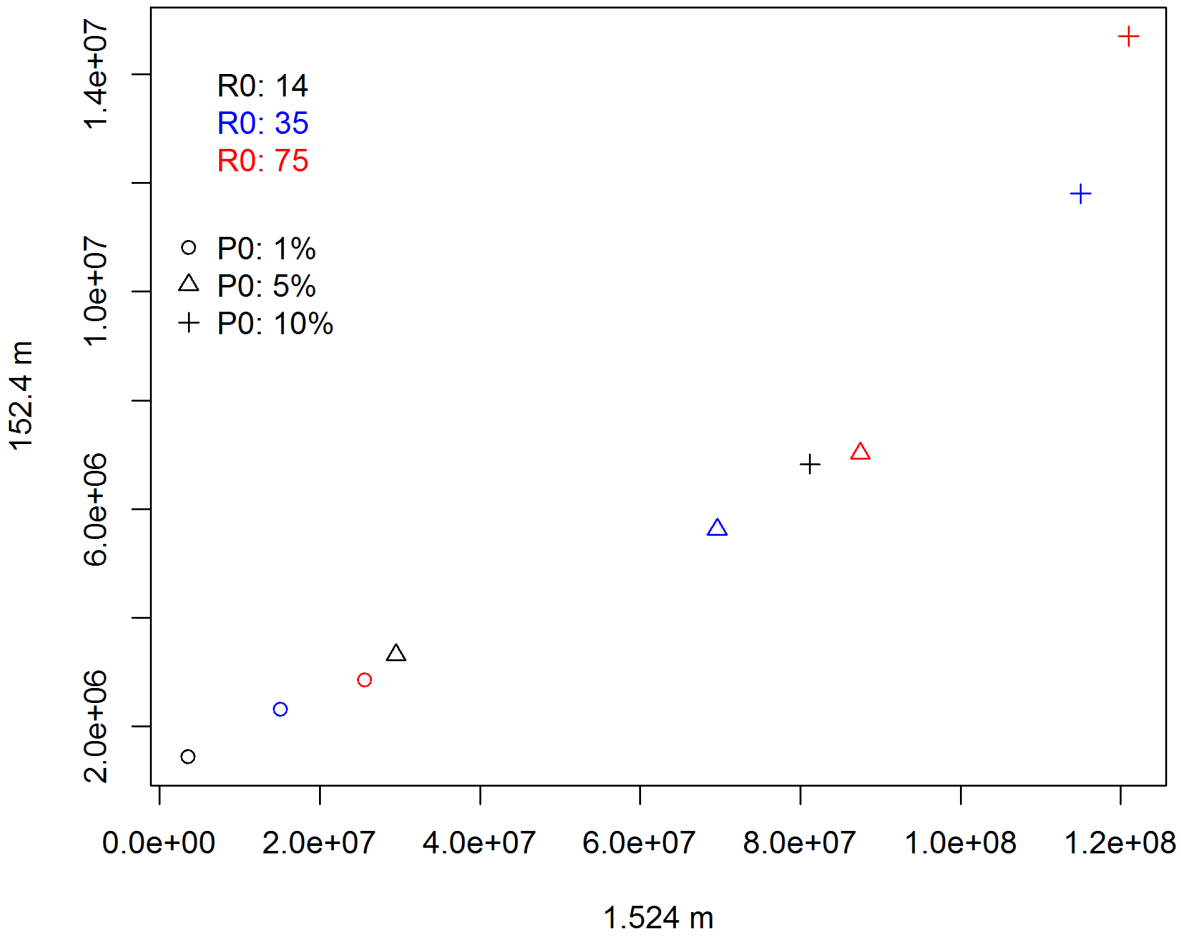

Fig. 4.17 Area under the curve (AUC) calculated for all compartments in a 91.44 m field, for all combinations of reproductive rate per generation (R_0) and initial prevalence as a percentage (P_0) after five latent periods. Each point represents an identical simulation, with the x-coordinate being simulated with fieldwide compartment size, 1.524 m, and non-normalized dispersal kernel, $y = 8.51 * (x + 0.76\ m)^{-2.33}$; and the y-coordinate being simulated with regional compartment size, 152.4 m, and non-normalized dispersal kernel, $y = 679.3\ m * (x + 132.6\ m)^{-1.97}$.

Chapter 5 - Conclusions

In this dissertation, I have examined the spread of the economically important plant pathogen *Puccinia striiformis* f.sp. *tritici (Pst),* the causal agent of wheat stripe rust (WSR), bridging theoretical dispersal and disease ecology with plant epidemiology to provide analyses that also have direct applicability to managing the spread of diseases caused by foliar wind-dispersed plant pathogens. This research will do much to fill in gaps in the literature on empirical measures of pathogen dispersal and infection, and the theoretical ramifications of modelling disease spread incorporating multiple spatial scales. It is the aim of this research to be applicable to a wide range of systems exhibiting similar attributes, particularly in regards to dispersal, in addition to aiding our understanding of *Pst* and other cereal rusts.

In chapter 2, my coauthors and I have quantified the primary dispersal gradient from isolated *Pst* uredinia. In this study, I conducted WSR lesion counts over all tillers within transects out to 1.5 m from the source to examine the progeny from a single uredinium over a single generation. I have shown that the local primary dispersal gradients of *Pst* were well-fitted by the modified inverse-power distribution, and by the non-linear Lomax Probability Density Distribution. I found that the slope of the local dispersal gradient was within the range reported in other dispersal studies at fieldwide, regional, and continental scales. I examined the vertical disease gradient of WSR lesions, and compared them with the vertical dispersal gradient of urediniospores in the same plot. I found significantly more progeny lesions at or above the leaf containing the source lesion, but did not observe this vertical gradient of urediniospores.

Arising from the observations in the field study of the vertical gradient of WSR lesions and the lack of corresponding vertical gradient of urediniospores, I hypothesized a vertical gradient of *Pst* infection efficiency could be the primary driver of the resulting pattern of WSR lesions. To test this, in

chapter 3, I designed an experiment inoculating random blocks of 3-, 4-, and 5-week-old susceptible wheat plants with uniform quantities of *Pst* urediniospores. I observed significantly greater WSR severity on younger plants than older and on younger leaves than older leaves within single treatments of plants. There was a significant interactive effect between plant age and leaf position, as well, with the differences in severity between leaf positions greater in younger plants. This experiment could point to differences in susceptibility of wheat as a function of tissue age as a significant driver of the vertical disease gradients observed in cereal rusts.

As the primary local dispersal gradient of *Pst* from isolated source lesions measured in chapter 2 filled in a gap in our knowledge of aerial dispersal of small propagules, in chapter 4 I compared these data with previous primary dispersal gradients of cereal rusts across the field and regional scales. I found a single modified inverse-power function which fit all three spatial scale dispersal datasets well. Using this modified inverse-power function to seed a dispersal kernel, I developed a susceptible-latent-infected-removed (SLIR) compartmental model to assess the spread of epidemics as a function of distance from a source. I found that similar levels of disease were obtained when modeling diseases with the same input parameters with differing resolutions, or compartment sizes. These results suggest it may be possible to model epidemics caused by pathogens with passively aerially dispersed particles using dispersal data of highly divergent spatial grain. Taken together, these studies offer significant insight into disease and dispersal ecology. As a large number of systems involve passive aerial dispersal of small propagules, the results of these studies could be applied widely, and could be built upon by future studies. As climate change and human interventions lead to new invasions of pathogens affecting animals, as well as plants, understanding their dispersal can inform decision-makers in how and where to invest limited resources to best combat these epidemics.

Chapter 6 – References

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Chapter 7 – Appendices

```
# Table of Contents: 1) Prep data frame; 2) Fraction of annuli sampled;

# 3) CDFs and PDFs for Modified Pareto and Weibull; 4) Discretization; 5) Model frame;

# 6) Flexible MLE model; 7) Optimization; 8) Plotting PMFs and CMFs of lesions per bin (not 2D-adjusted)

#~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~

# 1) Prep data frame

prep.df<-function(metric) #if metric is "on" distances and widths are multiplied by 0.0254

 {all4yrs <- read.csv("all11to14.csv", header=T) #import data

 nocontam <- subset(all4yrs, (rep != 1325) & (year != 2011)) #remove reps likely to contain outside inoculum

 nocontam$rep <- as.factor(nocontam$rep) #treat rep as a factor

 dists <- seq(0, 60, 1) #create a variable of distances in inches

 width<-1

 if(metric == "on")

  {nocontam$distance<-nocontam$distance*.0254

  width<-0.0254

  dists<-dists*.0254} # convert to meters

 return(list(nocontam,dists,width))}

require(htmlTable)

nocontam<-prep.df(metric = "on")[1][[1]]

dists<-prep.df(metric = "on")[2][[1]]

width<-prep.df(metric = "on")[3][[1]]


# 2) Fraction of annuli (0.0254m-width concentric rings centered on source lesion) sampled

annulus.fraction<-function(r,bin = width) # set r to dists.english and width to 1 for english
```

```
  {area.sampled<-8*(width*3*sqrt((r+width)^2-
(6*width)^2)+(((r+width)^2)/2)*asin((6*width)/(r+width)) -

                width*3*sqrt((r)^2-(6*width)^2)-(((r)^2)/2)*asin((6*width)/(r)))

  annulus<-pi*((r+width)^2-(r)^2) ## area of each whole annulus

  return(area.sampled/annulus)}


# 2.1) creates vector containing fraction of area sampled for all distances in a corresponding vector

get.area.fraction<-function(x,bin = width)

  {area.fraction<-c()

  for(i in 1:length(x))

    {if (x[i] <= 7*width)

      {area.fraction<-append(area.fraction,1)}

    else if(x[i]==8*width)

      {area.fraction<-append(area.fraction,((1+annulus.fraction(x[i],width))/2))}

    else # (x[i] >= 8*width)

      {area.fraction<-append(area.fraction,annulus.fraction(x[i],width))}}

  return(area.fraction)}


# 3) Cumulative density functions (CDFs) and Probability density functions (PDFs)

cdf.modpareto <- function(x, params)

  {b <- params[1]; c <- params[2]

  q <- 1 - ((1 + (x / b)) ^ - c)

  return(q)}

cdf.weibull <- function(x, params)

  {b <- params[1]; c <- params[2]

  q <- 1 - exp(- ((x / b) ^ c))
```

```
 return(q)}

# 4) Discretization of PDFs and CDFs to probability mass functions (PMFs) and
 #cumumlative mass functions (CMFs)
# 4.1) normalizing data by fraction of annuli sampled to examine 2-D dispersal
# set width to 1 and dist.max to 60 for english
pmf.norm <- function( cdf, params,bin = width, dist.max = tail(dists,1))
 {x <- seq(0, dist.max, width)
 q <- cdf(x + width, params) - cdf(x , params)
 q <- q / cdf(dist.max + width , params)
 f <- get.area.fraction(x,width)
 q2d <- q * f
 normalization <- 1.0 / sum(q2d)
 return(normalization)}

# 4.2) discretization; 2-D normalization is off by default
##width and dist.max set to 0.0254 and 1.524 for metric; 1 and 60 for english
pmf.binned <- function(x, cdf, params,bin = width, dist.max = tail(dists,1),adjust.2d = F)
 {if(length(x)==0)
  {return(list())}
 else{
  q <- cdf(x + width, params) - cdf(x, params) ##width-wide bins for annuli from x[n] to x[n+1]
  q <- q / cdf(dist.max + width, params)
  if (adjust.2d==TRUE)     ## 2D-corrected bins
   {q2d<-q*get.area.fraction(x,width)
```

```
    q2d<-q2d*pmf.norm(cdf, params, width, dist.max)
    return(q2d)}
   else
    {return(q)}}}

# 5) Model frame: response var ~ explanatory var (numeric) optionally + vars (factor)
get.mle.full.model<-function(formula,data)
 {nozerolesions<-data
  themodel<-model.frame(formula,nozerolesions)
  ret.list<-list()
  factor.list<-list()
  if(ncol(themodel)<3)
   {ret.no.blocking<-list(); ret.no.blocking[[1]]<-"1"; ret.no.blocking[[2]]<-1
   return(ret.no.blocking)}
  for(i in 3:ncol(themodel))
   {factor.list[[i-2]]<-factor(themodel[,i])
   ret.list[[i-2]]<-levels(factor.list[[i-2]])}
  vars.exp<-expand.grid(ret.list)
  names(vars.exp)<-names(themodel)[3:max(length(themodel))]
  return(list(ret.list,vars.exp))}

# 6) Flexible maximum likelihood estimation (MLE) model
mle.full<-function(formula, data, cdf,par.start,bin = width) #set width to 1 for english
  {nozerolesions <- subset(data, lesions > 0)
       params<-exp(par.start)
```

```
 ret <- get.mle.full.model(formula,nozerolesions)
 par.frame<-ret[[2]]  ##matrix of each unique combo of levels of explanatory vars
 lev.list<-ret[[1]]  ##list of each level
 if(length(lev.list[[1]])==1)     ##separates out the simple model
  {loglikelihood<-sum(log(pmf.binned(nozerolesions$distance, cdf,exp(par.start))) *
nozerolesions$lesions)
  return(-loglikelihood)}
 else
  {p.return<-matrix(0,ncol=2,nrow=length(par.frame[,1]),dimnames=list(NULL,c("b","c")))
  frame <- model.frame(formula, nozerolesions)
  loglikelihood <- 0
  for(i in 1:length(par.frame[,1]))   ##i is each unique combination of explanatory variables
   {level.position<-c()
   vars<-par.frame[i,]  ##vars is each combo of levels of explanatory vars
   b <- 1; c <- 1; k <- 0
   for(j in 1:length(par.frame))
    {level.position<-as.numeric(vars[j])
     if (j == 1)
        {b <- b * params[level.position + k]
        k <- k + length(lev.list[[j]])
        c <- c * params[level.position + k]
        k <- k + length(lev.list[[j]])}
     else
      {if (level.position > 1)
       {b <- b * params[level.position - 1 + k]}
      k <- k + length(lev.list[[j]]) - 1
```

```
        if (level.position > 1)
          {c <- c * params[level.position - 1 + k]}
        k <- k + length(lev.list[[j]]) - 1 }}
    p<-c(b,c)
    p.return[i,]<-p
    data.sub <- frame  ##data.sub will become subset of plants matching all explanatory variables
    for(j in 1:length(vars))
     {if (nrow(data.sub) > 0)
       {data.sub <- subset(data.sub, data.sub[j + 2] == as.character(vars[[j]]))}}
      if (nrow(data.sub) > 0)
       {logpmf <- sum(log(pmf.binned(data.sub$distance, cdf, p)) * data.sub$lesions)
       loglikelihood <- loglikelihood + logpmf}}
return(-loglikelihood)}}

# 7) optimization
optim.mle.params<-function(formula,data,cdf,params,temp.set)
 {result <- optim(par = log(params),cdf = cdf,data=data,hessian=T,
   fn=mle.full,formula=formula,method="SANN",control=list(temp=temp.set, trace=TRUE))
 params <- exp(result$par)
 return(list(params, result$value,result$hessian))}

# 7.1) polishing opitimized models
cdf.polish<-function(formula,data,cdf,params)
 {start.pars<-params
 for( i in 1:10)
```

```
  {polished<-optim.mle.params(formula,data,cdf,start.pars,100)
  print(polished[[1]])
  start.pars<-polished[[1]]}
 return(polished)}

# 7.11) Polished models (takes ~ 5 minutes for simple models; ~ 5 hours for full models)
# modp.simple.polished<-cdf.polish(lesions~distance,nocontam,cdf.modpareto,c(.6,3))
# wei.simple.polished<-cdf.polish(lesions~distance,nocontam,cdf.weibull,c(.38,1.8))
# modp.by.source.polished<-
cdf.polish(lesions~distance+source,nocontam,cdf.modpareto,modp.params.by.source)
# wei.by.source.polished<-
cdf.polish(lesions~distance+source,nocontam,cdf.weibull,wei.params.by.source)
# modp.by.source.and.year<-
cdf.polish(lesions~distance+source+year,nocontam,cdf.modpareto,rep(1,13))
# wei.by.source.and.year<-cdf.polish(lesions~distance+source+year,nocontam,cdf.weibull,rep(.5,13))
# modp.full.polished<-cdf.polish(lesions~distance+source+year+rep,nocontam,cdf.modpareto,rep(1,26))
# wei.full.polished<-cdf.polish(lesions~distance+source+year+rep,nocontam,cdf.weibull,rep(.5,26))

# 7.2) Best parameters by source
wei.params.simple<-c(0.2193340,0.8954835) #MLE: 4990.48
modp.params.simple<-c(0.7199826, 3.8729013) #MLE: 4975.73
modp.params.by.source<-c(3.408099e-01,3.277931e+02,4.665225e-01,7.695367e-02,1.508842e+00,
 1.527509e+03,2.411295e+00,8.407137e-01) #MLE: 4960.00
wei.params.by.source<-c(0.2839478,0.1961881,0.2344966,0.1586086,0.9265609,0.9586761,
 0.8934381,0.7396595) #MLE: 4979.31
modp.params.by.source.and.year<-c(8.121338e-04,3.616724e+00,4.275216e-01,1.240618e-
02,1.471364e+01,
```

```
  6.678615e+04,1.871600e+00,6.374985e-02,4.427149e+02,1.284072e+01,1.121018e-01,

  2.380552e+01,4.645725e+03) #MLE: 4932.17

wei.params.by.source.and.year<-c(1.695174e+02,1.257718e+02,2.426656e-01,2.649195e-
01,1.442253e-05,

  1.641147e-05,8.700737e-01,5.692562e-01,1.609073e-03,6.369999e-01,5.594865e+04,

  1.426493e+00,8.066950e+01) #MLE: 4948.21

modp.params.full<-c(2.273046e+02,6.336552e-01,5.793760e-01,8.110270e-
02,1.221309e+00,4.859744e+02,

  3.435491e+00,2.442690e-03,4.920535e-01,2.831168e-02,2.650981e+00,1.185005e+01,4.155104e-01,

  4.928104e+01,2.098449e-06,4.717317e+01,1.184530e-01,8.525987e+01,6.203500e+03,5.754327e-01,

  5.803005e-02,2.704096e-04,2.256827e+01,1.225187e+01,7.384364e+01,1.549730e+01)  ##MLE:
4912.46

wei.params.full<-c(2.706799e+02,1.857725e+03,2.109164e-01,7.049891e+00,2.583314e+01,1.216256e-
01,

  1.129527e+00,1.073197e-02,1.988070e-02,3.919373e+00,9.244412e-02,1.159013e+00,6.639846e-
01,6.449151e-03,

  8.949584e-03,1.665958e+00,5.419037e-02,6.957105e-03,2.072131e-01,7.078430e-
01,9.156256e+01,5.548503e-02,

  8.435567e-01,3.877758e-01,6.018804e+01,8.429349e-01)  ##MLE: 4931.77


# 7.3) AIC Testing

formula.list<-c("Lesions ~ distance","Lesions ~ distance","Lesions ~ distance + source",

  "Lesions ~ distance + source","Lesions ~ distance + source + year","Lesions ~ distance + source + year",

  "Lesions ~ distance + source + year + replicate","Lesions ~ distance + source + year + replicate")

param.list<-c(2,2,8,8,13,13,26,26)

MLE.list<-c(4975.73,4990.48,4979.31,4960.00,4932.17,4948.21,4912.46,4931.77)

distro.list<-c("Modified pareto","Weibull","Modified pareto","Weibull","Modified
pareto","Weibull","Modified pareto","Weibull")
```

```
AIC.test<-function(distribution, par.nmbr,mle) #distribution, number of parameters, mle after polishing

 {df.return<-
data.frame(matrix(0,ncol=5,nrow=length(MLE.list),dimnames=list(NULL,c("Model","Distribution","Para
meters","MLE","AIC"))))

 df.return[,1]<-formula.list

 df.return[,2]<-distribution

 df.return[,3]<-par.nmbr

 df.return[,4]<-mle

 df.return[,5]<-mapply(function(x,y) (2*x)+(2*y), param.list,MLE.list)

  return(df.return)}

htmlTable(x = AIC.test(distro.list,param.list,MLE.list),

 caption = paste("Table 2.  Aikake information criterion (AIC) and makimum likelihood estimate (MLE)
for each model and distribution."),

 rnames=F)


# 8) Plotting PMFs and CMFs of lesions per bin (not 2D-adjusted)

# 8.01) Prep plotting dataframes

get.bysource.df<-function(source.leaf)

{bysource.df<-subset(nocontam,source==source.leaf)

  return(bysource.df)}

counts<-function(data,bin = width)

 {counts <- c()

  for(i in dists)

    {counts <- append(counts, sum(subset(data, distance == i)$lesions))}

  freqs <- counts / sum(counts)

 return(list(counts,freqs))}

for(i in c(0,1,3,4))
```

```
 {print(counts(get.bysource.df(i)))}


#8.02) Equations for legends

modpareto.siple.eq.cmf<-expression("Modified pareto: y=1-(1+x/0.72)"^{-3.87})

weibull.simple.eq.cmf<-expression("Weibull: y=1-e"^{"-(x/0.22)"^{0.90}})

#Modified pareto PDF: y = (c / b )* ((1 + x / b) ^ -(c + 1))

modpareto.siple.eq.pmf<-expression("Modified pareto: y="~5.38~"*(1+x/0.72)"^{"-4.87"})

#Weibull PDF: y= (c / b )* (x / b) ^ (c - 1) * exp(- (x / b) ^ c)

weibull.simple.eq.pmf<-expression("Weibull: y="~4.08~"*(x/0.22)"^{"-0.11"}~"e"^{"-(x/0.22)"^{0.89}})

modp.by.source.eq.cmf<-c(expression("y=1-(1+x/0.34)"^{-1.51}),expression("y=1-(1+x/32.78)"^{-
1527.51}),

 expression("y=1-(1+x/0.47)"^{-2.41}),expression("y=1-(1+x/0.077)"^{-0.84}))

modp.by.source.eq.pmf<-c(expression("y="~4.43~"*(1+x/0.34)"^{-2.51}),

 expression("y="~4.66~"*(1+x/32.78)"^{-1528.51}),

 expression("y="~5.17~"*(1+x/0.47)"^{-3.41}),

 expression("y="~10.92~"*(1+x/0.077)"^{-1.84}))

wei.by.source.eq.cmf<-c(expression("y=1-e"^{"-(x/0.28)"^{0.93}}),expression("y=1-e"^{"-
(x/0.20)"^{0.96}}),

 expression("y=1-e"^{"-(x/0.23)"^{0.89}}),expression("y=1-e"^{"-(x/0.16)"^{0.74}}))

wei.by.source.eq.pmf<-c(expression("y="~3.26~"*(x/0.28)"^{-0.07}~"e"^{"-(x/0.25)"^{0.93}}),

 expression("y="~4.89~"*(x/0.20)"^{-0.04}~"e"^{"-(x/0.20)"^{0.96}}),

 expression("y="~3.81~"*(x/0.23)"^{-0.11}~"e"^{"-(x/0.23)"^{0.89}}),

 expression("y="~4.66~"*(x/0.16)"^{-0.26}~"e"^{"-(x/0.16)"^{0.74}}))


# 8.1) Plottting lesions ~ distance: cumulative = "on" plots CMF; "off" plots PMF

plot.simple<-function(data,cumulative,bin = width,adj.2d=F)
```

```
 {plot.new()

 par(mfrow=c(1,1))

 par(mar=c(4,4,1,1))

 freqs<-counts(data)[[2]]

 if (cumulative=="on")

  {plot(dists, cumsum(freqs),col="black",pch=1,xlab="Distance (m)",ylab="Cumulative
frequency",ylim=c(0,1))

  points(dists, cumsum(pmf.binned(dists, cdf.weibull, wei.params.simple,adjust.2d=adj.2d)),pch = 3)

  points(dists, cumsum(pmf.binned(dists, cdf.modpareto, modp.params.simple,adjust.2d=adj.2d)),pch =
2)

  legend("bottomright",c("Cumulative proportion of lesions",modpareto.siple.eq.cmf,

       weibull.simple.eq.cmf),pch=c(1,2,3),cex=.95)}

 else

  {plot(dists, freqs,col="black",pch=1,xlab="Distance (m)",ylab="Frequency",ylim=c(0,0.2))

  points(dists, pmf.binned(dists, cdf.weibull, wei.params.simple,adjust.2d=adj.2d),pch = 3)

  points(dists, pmf.binned(dists, cdf.modpareto, modp.params.simple,adjust.2d=adj.2d),pch = 2)

  legend("topright",c("Proportion of lesions",modpareto.siple.eq.pmf,

                weibull.simple.eq.pmf),pch=c(1,2,3),cex=.9)}

 return(list("raw data" = freqs,"weibull" = pmf.binned(dists, cdf.weibull, wei.params.simple),"modified
pareto" = pmf.binned(dists, cdf.modpareto, modp.params.simple)))}

plot.simple(nocontam,cumulative = "on")

plot.simple(nocontam,cumulative = "off")


# 8.2) Plot lesions ~ distance + source

plot.by.source<-function(data,type,xlab,ylab,x.ticks,y.ticks,ymax)  #type is cmf or pmf;tickmarks is "y" or
"n"

 {freqs<-counts(data)[[2]]
```

```
    if(type=="cmf")
      {freqs<-cumsum(freqs)}
    plot(dists, freqs,cex.axis=.5,xaxt=x.ticks,yaxt=y.ticks,
    xlab = xlab, ylab = ylab, ylim = c(0, ymax))}

# 8.21) Plot by source for all leaves with legends
plot.all.by.source<-function(cumulative)
  {source.leaf<-c(0,1,3,4)
    fig.lab<-c("a","b","c","d")
    plot.new()
    par(mfrow=c(2,2))
    mar.string<-c(4,4,4,4, 4,2,4,2, 0,2,0,2)
    tickmarks.string<-c("n","n","true","true","true","n","true","n")
    m<-rbind(c(1,2),c(3,4))
    x.label.string<-c("","","Distance (m)","Distance (m)")
    y.label.string.cmf<-c("Cumulative freq","","Cumulative freq","")
    y.label.string.pmf<-c("Frequency","","Frequency","")
    if (cumulative == "on")
      {for(i in 1:4)
        {par(mar=c(mar.string[i],mar.string[i+4],0,mar.string[i+8]))
        plot.by.source(get.bysource.df(source.leaf[i]),"cmf",x.label.string[i],y.label.string.cmf[i],
          x.ticks=tickmarks.string[i],y.ticks=tickmarks.string[i+4],ymax=1)
        points(dists, cumsum(pmf.binned(dists, cdf.modpareto,
          c(modp.params.by.source[i],modp.params.by.source[i+4]))), pch = 2,col="red")
        points(dists, cumsum(pmf.binned(dists, cdf.weibull,
```

```
        c(wei.params.by.source[i],wei.params.by.source[i+4]))), pch = 3,col="green")

legend("bottomright",c(modp.by.source.eq.cmf[i],wei.by.source.eq.cmf[i]),pch=c(2,3),col=c("red","gree
n"),
        cex=.75)
       legend("topleft",fig.lab[i],cex=.7)}}
   else
    {for(i in 1:4)
      {par(mar=c(mar.string[i],mar.string[i+4],0,mar.string[i+8]))
      plot.by.source(get.bysource.df(source.leaf[i]),"pmf",x.label.string[i],y.label.string.pmf[i],
        x.ticks=tickmarks.string[i],y.ticks=tickmarks.string[i+4],ymax=.5)
      points(dists, pmf.binned(dists, cdf.modpareto,
        c(modp.params.by.source[i],modp.params.by.source[i+4])), pch = 2,col="red")
      points(dists, pmf.binned(dists, cdf.weibull,
        c(wei.params.by.source[i],wei.params.by.source[i+4])), pch = 3,col="green")

legend("topright",c(modp.by.source.eq.pmf[i],wei.by.source.eq.pmf[i]),pch=c(2,3),col=c("red","green"),
        cex=.55)
      legend("topleft",fig.lab[i],cex=.5)}}}
plot.all.by.source(cumulative = "off")
plot.all.by.source(cumulative = "on")

# 8.5) Plot legend for PMF or CMF by source
add.legend<-function()
 {par(mfrow=c(1,1))
 par(mar=c(0,0,0,0))
 plot.new()
```

```
 legend("center",c("Proportion"," of lesions","Modified","  pareto","Weibull"),
   pch=c(1,0,2,0,3),col=c("black",NA,"red",NA,"green","green"),cex=1)}
add.legend()
```

Fig. S 2.1 R Code

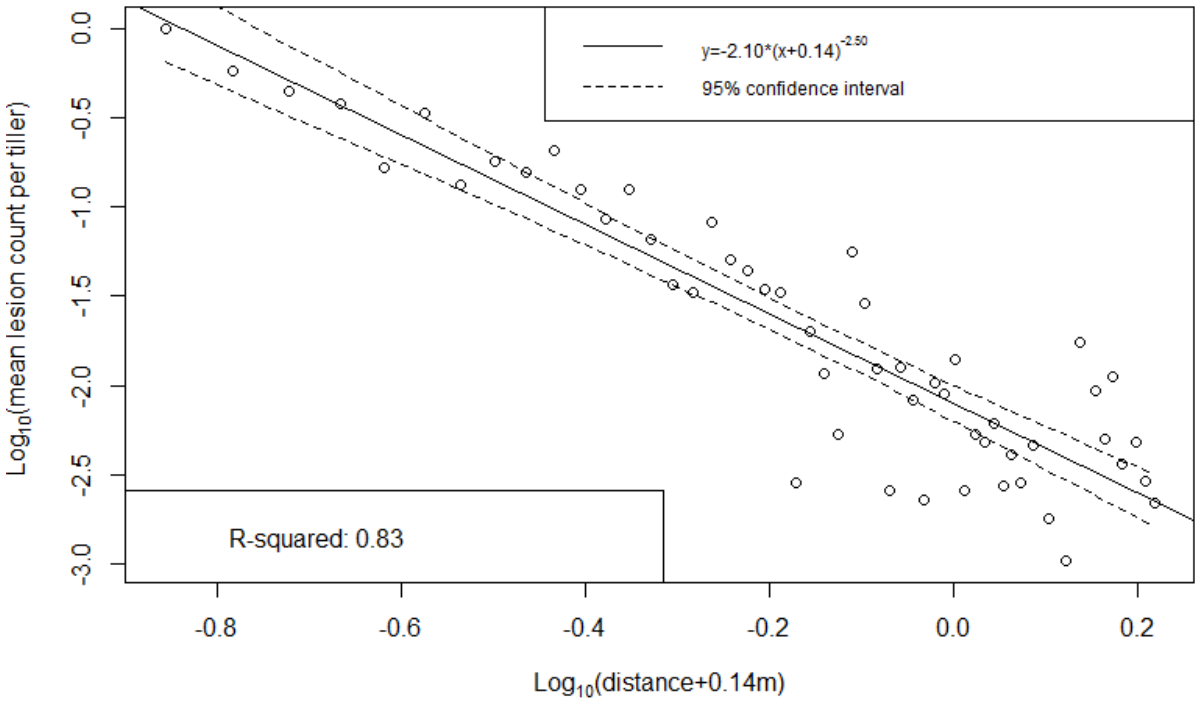


Fig. S 2.2 Linear regression of log-transformed lesions, normalized by setting the amount of disease at the source to 1, as a function of log-transformed distance with best-fit *c* offset, aggregated from all plots 2012-2014 to maximize non-zero lesion count coverage of distance intervals, with 95% confidence intervals.

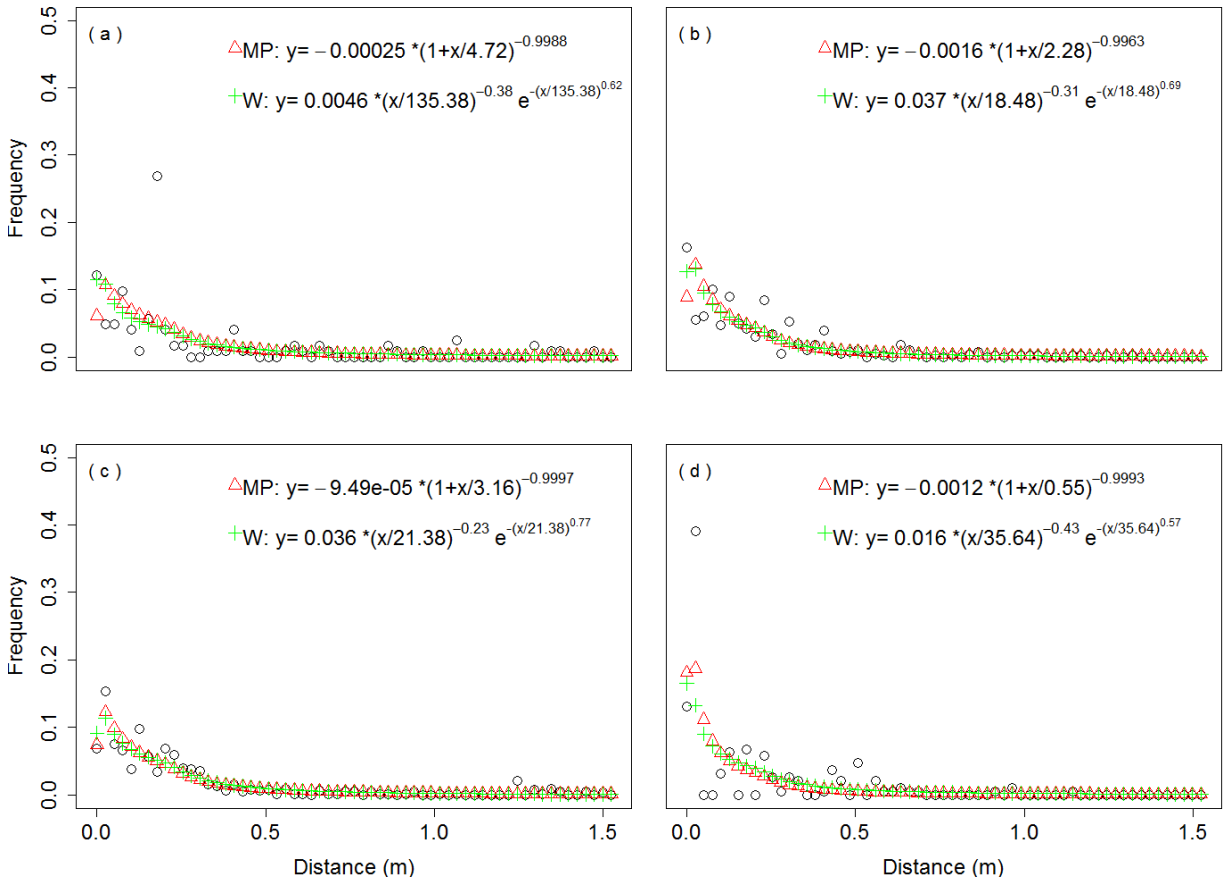
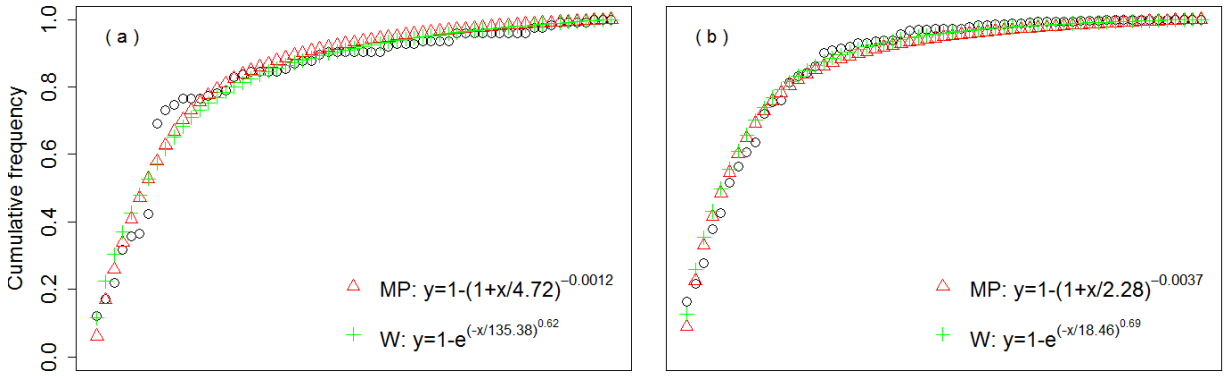


Fig. S 2.3 Proportion of total observed progeny lesions as a function of distance from single source lesions with best-fit Weibull (W) and Modified Pareto (MP) probability mass functions, grouped by leaf position of source lesion. The source lesion was on **a)** leaf 1, **b)** leaf 2, **c)** leaf 4, and **d)** leaf 5.



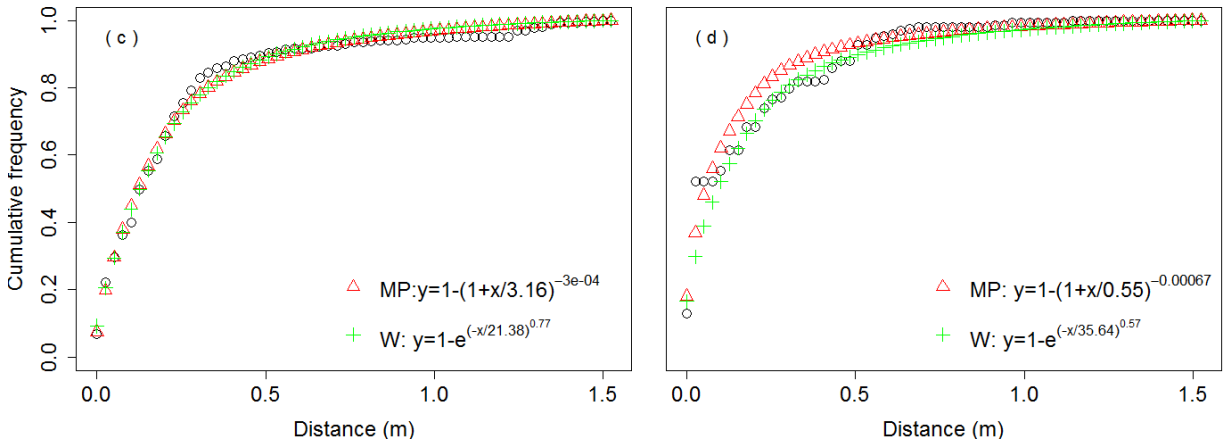


Fig. S 2.4 Cumulative proportion of total observed progeny lesions as a function of distance from single source lesions with best-fit Weibull (W) and Modified Pareto (MP) probability mass functions, grouped by leaf position of source lesion. The source lesion was on **a)** leaf 1, **b)** leaf 2, **c)** leaf 4, and **d)** leaf 5.

```
function [p,u,v,rem,dis] = epidemic_SLIR(n,T,l,s,r,m,f,u_0,e,d,lambda,size)
%EPIDEMIC_SIR
%epidemic spread with removal of sporulating infections after d days of
%infectiousness
%size - size in meters of each compartment n
u = zeros(T,n,l); %latent array
v = zeros(T,n,d); %sporulating array
p = zeros(T,n); %uninfected plant array
rem = zeros(T,n); %removed (no longer sporulating) array
kernel = square_trial(n,size);
%m sites per compartment
%lambda - weight from (0,1) of dispersal;
%(1-lambda) - weight of somatic growth
for i = 1:s:n
   p(1:l,i) = m;
```

```
end
%u_0 initial severity in compartment(s) f
for i=f
   p(1,i) = m - m*u_0;
   u(1,i,1) = u_0*m;
end
%growth for all time 1:T for all compartments 1:n
for t=1:T-1
   fprintf('Day %d\n',t)
   for i=1:n
      a = kernel(i,:); %row array produced by kernel_square
      k = min(1,(sum(a(1:n).*sum(v(t,:,:),3)*r))/m);
      p(t,i) = max(0, m - sum(u(t,i,:))-sum(v(t,i,:))-rem(t,i));
      g = growth_bev(sum(u(t,i,:))+sum(v(t,i,:)),e,m);

      u(t+1,i,1) = p(t,i)*(lambda*k+(1-lambda)*g);
      %growth of immature latent infections
      for j = 1:l-1
         u(t+1,i,j+1) = u(t,i,j);
      end
      if u(t,i,l)+sum(v(t,i,:))<m
         v(t+1,i,1) = u(t,i,l);
      else
         v(t+1,i,1) = m - sum(v(t,i,2:d));
      end
```

```
      for j = 1:d-1
         v(t+1,i,j+1) = v(t,i,j);
      end
      rem(t+1,i) = rem(t,i) + v(t,i,d);
   end
end
dis = sum(v,3)+rem;
%plotting:
Fig.()
hold on
for i = 2:l:t
   plot(.0254*size:.0254*60:n*size*.0254,dis(i,:));
end
str=sprintf('%.2f m compartments: R_0: %.2f P_0: %.2f',size*.0254,round(r,2),round(u_0,2));
title(str)

function y = square_trial(field,size)
%dispersal kernel from all source locations to all sink locations
y = zeros(field,field);
k = kernel_spatially_explicit(5000,10000,size);
   for i = 1:field
      y(i,:) = k(abs(5000-(i-1):5000+field-i));
   end

function k = kernel_spatially_explicit(source, field, size)
```

```
%returns proportion of total new infections arising in each cell of a
%field, from a single source. calls inv_power
%size is length of each compartment of field in inches
%calculates kernel by single inch, then puts 'em in bins of 'size' inches
z = inv_power(900000,1);
y = zeros(1,900000);
for i = 1:900000
   if mod(size,2) == 0 %test if bin size in inches is even
      a = abs(i-(source*size-size/2)); %subtract size/2 to center source in compartment
   else
      a = abs(i-(source*size));
   end
   y(i) = z(a+1);
end
y = y./sum(y(1,:));
k = zeros(1,field);
for i = 1:length(k)
   a = size*(i-1)+1;
   b = size*i;
   k(i) = sum(y(a:b));
end

function y = inv_power(n,size)
k = zeros(1,n);
if size==60
```

```
    for i = 1:n
        k(i) = 431.8*(i+0.762*0.656)^-2.39; %converted to 5' blocks
    end
else %size==1
    for i = 1:n
        k(i) = 8.46*(i+0.12*39.37)^-2.39; %converted to inches
    end
end
y = k./sum(k(1,:));
```

Fig. S 4.1 Epidemic SLIR model and all subroutines