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Velocity of Spread of Wheat Stripe Rust Epidemics

Christina Cowger, LaRae D. Wallace, and Christopher C. Mundt

First author: U.S. Department of Agriculture-Agricultural Research Service, Department of Plant Pathology, North Carolina State University, Raleigh 27695; and second and third authors: Department of Botany and Plant Pathology, Oregon State University, Cordley Hall 2082, Corvallis 97331.
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ABSTRACT

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Controversy has long existed over whether plant disease epidemics spread with constant or with increasing velocity. We conducted large-scale field experiments with wheat stripe rust at Madras and Hermiston, Oregon, where natural stripe rust epidemics were rare, to test these competing models. Data from three location-years were available for analysis. A susceptible winter wheat cultivar was planted in pure stand and also in a 1:4 or 1:1 mixture with a cultivar immune to the stripe rust race utilized in the experiments. Plots were 6.1 m wide and varied from 73 to 171 m in length. A 1.5 by 1.5-m focus was inoculated in either the center (2001) or upwind of the center (2002 and 2003) of each plot. Disease severity was evaluated weekly throughout the epidemics in each plot at the same points along a transect running upwind and downwind from the focus. Velocity of spread was calculated from the severity data and regressed separately on time and on distance from the focus. In all location-years and treatments, and at all levels of disease severity, velocity consistently increased linearly with distance, at an average rate of 0.59 m/week per m, and exponentially with time. Further, across epidemics there was a significant positive relationship between the apparent infection rate, *r*, and the rate of velocity increase in both space and time. These findings have important implications for plant diseases with a focal or partially focal character, and in particular for the effectiveness of rate-reducing disease management strategies at different spatial scales.

Additional keywords: dispersive wave, exponential model, host genotype diversity, isopaths, power law, traveling wave, turbulent diffusion.

The spatiotemporal dynamics of epidemic expansion from foci of plant diseases have been the subject of considerable study and debate. Indeed, Zadoks (24) identified this subject as one of the key controversies in plant epidemiology in the 20th century. The present paper reports field data from a study designed to test competing models of epidemic expansion in space and time.

Epidemics may begin from foci, initiated by a single infectious unit, or from inoculum that is so well-distributed that foci are unapparent (19). For most polycyclic foliar diseases, it is difficult to predict the degree to which epidemic initiation will be discretely focal (22). Many epidemics have intermediate characteristics. Disease often begins in foci because overwintering pathogen populations are reduced and concentrated into patches, or because inoculum arrives sporadically from afar (21). The process of focal spread of disease over areas ranging from a single field to 1,000 km has been described (26). How disease spreads from foci in space and time is a subject of importance in any disease system that can have focal properties.

Two classic models are often used to describe gradients of inoculum and disease spread. The exponential model is $y = ae^{-bx}$, in which y is the number of propagules deposited or the number of infections per unit area at x units of distance from the source, a is a constant depending on source strength, e is the base of the natural logarithm, and b is a parameter with spatial units of meters that describes the steepness of the disease gradient (10,12). The exponential model fits situations in which airborne diffusion is relatively unimportant and the tail of the model is relatively short. The decrease of disease with distance is caused mainly by inoculum being deposited out of the air by a constant proportion as it moves through a plant canopy. In contrast, the power law is $y = ax^{-b}$, in which y and x are as above, a is the number of propagules or infections per unit area at one unit of distance from the source, and b is a dimensionless parameter describing disease gradient steepness. The power law model describes a disease gradient that declines in slope more sharply than the exponential model near the source (6), but then flattens out, providing a more extended tail that may contain a significant proportion of the dispersed inoculum.

Berger and Luke (3) derived a parameter, the rate of isopath movement, to describe the combined temporal and spatial spread of disease, in that case of crown rust (caused by *Puccinia coronata*) on oat (*Avena sativa*). An isopath is a contour line connecting points of equal disease severity. The rate of isopath movement, or velocity (v), of the epidemic is expressed in units of distance per time.

Controversy arose over whether epidemics expand in space and time with constant or with increasing velocity. In developing a computer simulator of focal expansions, Zadoks and Kampmeijer (25) concluded that epidemics expanded radially at constant velocity after an initial phase of focus saturation. A model developed by Minogue and Fry (13) was based on the assumption of a traveling wave of disease moving through space at a constant velocity, or $v = r/b$, where r is the apparent infection rate (e.g., in propagules produced per infectious tissue unit area per unit of time) (20) and b is the dimensioned steepness parameter described previously (10).

Along similar lines, van den Bosch et al. (19) developed a model for focal disease expansions that was based on the exponential model of spore dispersal. The model predicted that an epidemic would expand as a wave of constant velocity. These authors' analyses of field data sets for stripe rust (caused by *Puccinia striiformis*) on wheat (*Triticum aestivum*) and downy

Corresponding author: C. Cowger; E-mail address: christina_cowger@ncsu.edu

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mildew (caused by *Peronospora farinosa)* on spinach (*Spinacia oleracea*) suggested that, after a period of wave stabilization, foci expanded at a constant rate for both diseases (18).

In contrast, the theory of turbulent diffusion predicts that epidemic velocity should increase with the size of the epidemic focus, because more and larger atmospheric eddies participate in diffusion at increasingly large spatial scales (17). Ferrandino (5) expressed similar ideas and challenged the constant-velocity model. He argued that a wave of constant velocity should only be expected if the tail of the dispersal distribution is bounded by an exponential function, and the latter will only occur if there is insignificant upwards escape by spores out of the canopy. However, if spores do escape the canopy, the probability that they do so increases with distance from the point of production (2,5), leading to a dispersal gradient that is steep near the source and becomes increasingly flat with distance, as in the power law. Ferrandino (5) introduced the idea of a "dispersive epidemic wave" in which velocity increases over time and with increasing distance from the focus. He analyzed data (1) on late leaf spot of peanut (*Arachis hypogaea*) caused by *Cercosporidium personatum* and concluded that the epidemic velocity increased about sevenfold over a distance of 2 to 6 m. Similarly, Scherm (17) concluded from four sets of data gathered by other researchers on potato (*Solanum tuberosum* L.) late blight (caused by *Phytophthora infestans*) that epidemic velocity increased with the spatial scale of the epidemic.

Resolution of the debate over how focal epidemics expand has been hampered by the scarcity of relevant field data. We are aware of only two published data sets from experiments designed specifically to determine whether velocity of disease spread is constant or changing. In one of those studies, Minogue and Fry (14) described a constant velocity in the spread of *Phytophthora infestans* in potato plots 36.6 m in length. However, they discarded the 3 to 4 m closest to the inoculated focus and the 9.2 m farthest from the focus, because the gradients were unexpectedly steep and shallow, respectively. Steep gradients near the focus and shallow gradients farther from the source, such as those given by the power law, are expected under Ferrandino's dispersive wave theory (5). Further, Ferrandino's reanalysis of these data (5) was suggestive of a dispersive epidemic wave.

In the second report, Frantzen and van den Bosch (7) studied the spread of *Puccinia lagenophorae* in field plots of *Senecio vulgaris* (common groundsel) that were 87 to 99 m in length. While disease gradients showed flattening over time, such as would be associated with increasing velocity, the authors were unable to obtain a sufficient number of data points to conclusively determine the trajectory of velocities over time.

Attempts to calculate epidemic expansion velocities from historical field data have led to conclusions of both constant (9,18) and increasing (17) velocity. However, some retrospective analyses have relied on assumptions that do not always hold true. For example, calculating epidemic velocity using the square root of a focal area with a given level of disease (9) will only be correct if the epidemic is expanding equally in all directions. In fact, disease foci often advance faster in some directions (e.g., downwind), so the developing epidemic may not be radially symmetric.

The dynamics of spatiotemporal epidemic spread have important implications for how disease is managed. For example, host genotype heterogeneity (variety mixtures or multilines) reduces disease primarily because dilution of susceptible plants with resistant plants reduces the apparent infection rate, r (8,22). If focal epidemics expand with increasing velocity, it follows that the disease-reducing benefits of host heterogeneity will increase relative to nonmanaged situations at progressively larger scales (described below) (16).

We conducted a field experiment with artificial establishment of epidemics at disease foci in order to test the models discussed above. Our hypotheses were as follows. (i) Focal epidemics expand with increasing velocity over space and time, rather than with constant velocity (Fig. 1). According to this prediction, which is consistent with the power law, disease severity gradients should flatten over time. Thus, there should be an inverse relationship between velocity and isopath level, because with gradients that become flatter with time, isopaths of lower disease severities will travel farther per unit of time and will do so sooner. (ii) A reduction in r, the apparent infection rate (20), will slow the increase of velocity over time and space. Thus, the velocities for epidemics of different r will diverge as those epidemics expand in space and time (Fig. 1B). To test this hypothesis, we compared velocities in plots of a wheat cultivar susceptible to stripe rust with those in plots of the same cultivar mixed with an immune one in order to slow epidemic progression.

MATERIALS AND METHODS

Experimental design, planting, and crop management. The experiment was conducted at two locations in central and northeastern Oregon in each of three winter wheat seasons: 2000–01, 2001–02, and 2002–03 (the experimental growing seasons are hereafter termed 2001, 2002, and 2003). The locations were the Central Oregon Agricultural Research Center in Madras, and the Hermiston Agricultural Research & Extension Center. Both locations are semiarid and subject to large diurnal temperature

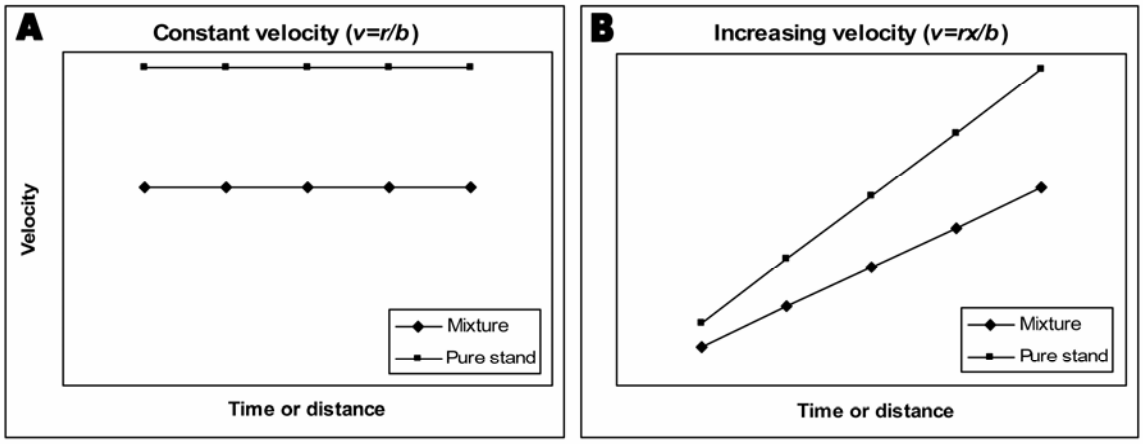

Fig. 1. Hypothetical example of epidemic velocity versus time or distance for epidemics in pure stands of susceptible plants or mixtures of susceptible and immune plants, under assumptions of **A,** constant velocity ($v = r/b$) or **B,** increasing velocity ($v = rx/b$), where x is time or distance.

fluctuations, with prevailing winds from the west. Natural stripe rust epidemics were rare at both locations.

Data are presented here from three location-years: Madras 2001, Hermiston 2002, and Madras 2003. In the other three location-years, digital photography did not provide reliable severity data (Hermiston 2001); disease intensity was too low to analyze isopath levels above 1% (Madras 2002); or a severe take-all epidemic confounded the stripe rust severity assessments (Hermiston 2003).

The soft white winter common wheat 'Stephens' (C.I. 017596) and the soft white winter club wheat 'Jacmar' (P.I. 608016) were chosen for the experiment because they are, respectively, immune (no visible symptomology or pathogen reproduction) and susceptible to *Puccinia striiformis* f. sp. *tritici* race CDL5. The two treatments in the experiment were a pure stand of 'Jacmar' and a mixture of 'Jacmar' and 'Stephens'. Each treatment was replicated three times at each location in each year.

In the first 2 years, seed of the two cultivars was mixed for the mixture treatment in a 1:4 ratio of 'Jacmar' to 'Stephens'. At the end of each growing season, the actual percentage of 'Jacmar' tillers in the mixture plots was determined by counting approximately 80 randomly chosen heads at each of at least five widely spaced sites in each plot. The two cultivars can be distinguished by their different spike morphologies. The proportion of 'Jacmar' seed in the mixtures was increased to 50% in the third year in order to allow higher disease levels to develop.

The experiments were planted in Madras on 3 November 2000, in Hermiston on 16 to 17 October 2001, and in Madras on 26 September 2002. The early planting date in Madras in 2002 was chosen in order to allow postplanting irrigation while the system was still in operation, because low moisture levels had hindered emergence and caused thin stands the previous year.

The seeding rate was 323 seeds per m^2 and the spacing between rows was 0.20 m. In both locations, the rows were planted parallel to the prevailing west-to-east wind direction.

Plot lengths in Madras were 73.2 m in 2001 and 152.4 m in 2003 (Fig. 2). Plot lengths in Hermiston in 2002 ranged from 107.0 to 170.7 m because the experiment was planted in a quarter of a pivot-irrigated circle (Fig. 2). Areas between plots and at least 6.1 m around each experiment were planted to 'Stephens' wheat. In Madras in 2003, the plots were planted in two tiers, one east of the other, with the two plots of the second replicate split between the two tiers (Fig. 2). All plots in all years were 6.1 m wide.

In 2002 and 2003, plots received overhead irrigation during the period from stem elongation to hard dough at a rate sufficient to maintain a vigorous and uniform crop. The amount and frequency of irrigation was determined by local soil and rainfall conditions, and by available irrigation equipment and scheduling. In Madras, stationary lines provided irrigation once per week. In Hermiston, plots were irrigated three to five times per week, depending on crop needs, with a center pivot. Fertilizer and pesticides were applied consistent with local commercial practices.

Inoculations. Stripe rust epidemics were initiated by artificial inoculation of a focus that was either centered (2001) or offset in the upwind direction (2002 and 2003) in order to provide a greater downwind distance for epidemic development (Fig. 2).

Inoculations were performed when wheat plants were approximately at the five-leaf stage. Inoculations took place on 22 March 2001 in Madras, 14 March 2002 in Hermiston, and 3 March 2003 in Madras.

Each inoculated focus was a square measuring 1.5 m on a side. During application of inoculum, a clear plastic tent was erected over each focus to ensure that inoculum remained within the focus area. Inoculation was performed with urediniospores of

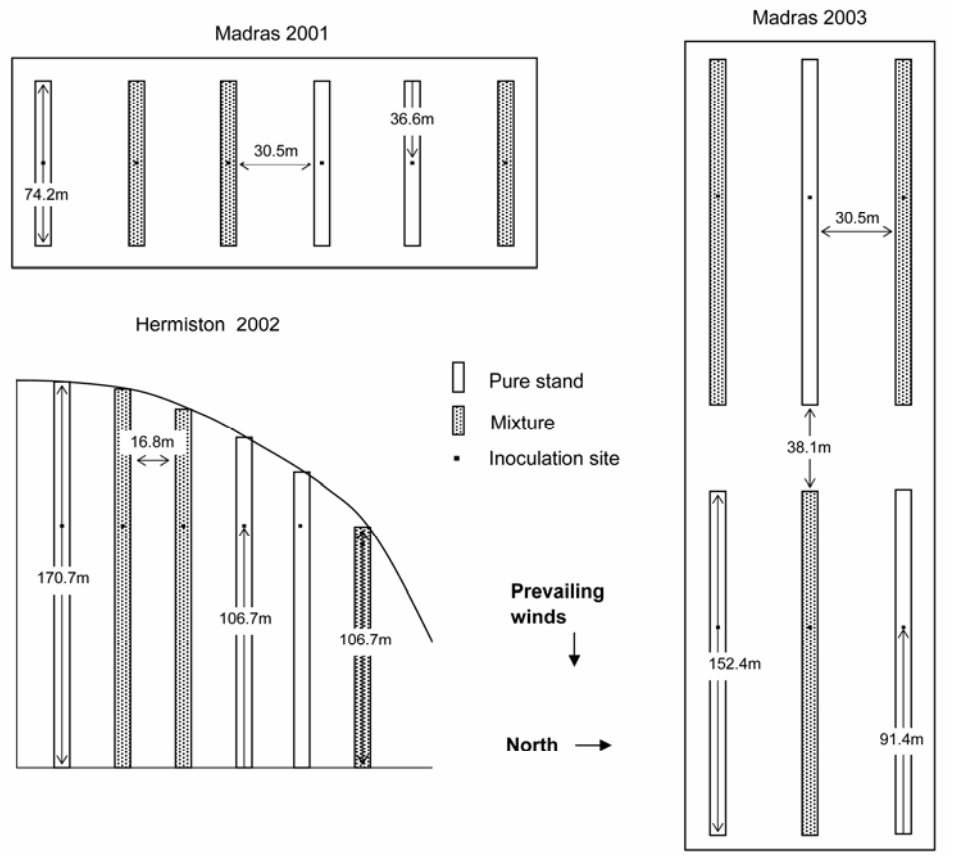

Fig. 2. Schematic representation of plots and inoculation sites for three location-years of a winter wheat/stripe rust experiment on velocity of epidemic spread. Pure stands were of susceptible plants, and mixtures were a 1:4 (2001 and 2002) or 1:1 (2003) ratio of susceptible to immune plants. All plots were 6.1 m wide.

Puccinia striiformis race CDL5 (obtained from R. Line, USDA-ARS, Pullman, WA) that had been grown on 'Jacmar' seedlings in a growth chamber. Where insufficient fresh spores were available, frozen spores were also used to ensure sufficient inoculum. Each focus received between 0.25 and 0.5 g of spores, depending on the balance of fresh to frozen inoculum, since the frozen inoculum was known to possess a lower viability. Stripe rust spores were mixed with talc at approximately 1:10 by volume, and filtered through eight layers of cheesecloth onto the wheat seedlings. In Madras in 2003, water was applied to the seedlings with a spray bottle immediately prior to inoculation in order to encourage infection.

Immediately after inoculation, which was conducted in the late afternoon, each focus was completely covered with black plastic that was securely anchored to the soil on all sides. The plastic was applied in order to facilitate dew formation and remained on the foci overnight for approximately 14 h.

Disease assessments. Disease assessments were performed weekly from the first appearance of rust lesions in the foci until host senescence precluded accurate observations. The assessent periods were 14 May to 2 July 2001, 18 April to 30 May 2002, and 9 April to 18 June 2003. Disease was assessed at 6.1-m intervals along a transect running east (downwind) and west (upwind) from the focus, with additional assessment points in the focus and at 1.5 and 3.1 m in both directions. The last 6 m of each transect was not assessed to avoid edge effects.

At each assessment point, two researchers assessed disease severity as the percentage of leaf area covered by stripe rust lesions, including all leaf levels, in a 0.75- × 1.5-m area on their respective sides of the east-west transect. The two observers' estimates were averaged for each assessment point. With few exceptions, the same two observers conducted assessments in a particular experiment throughout a growing season.

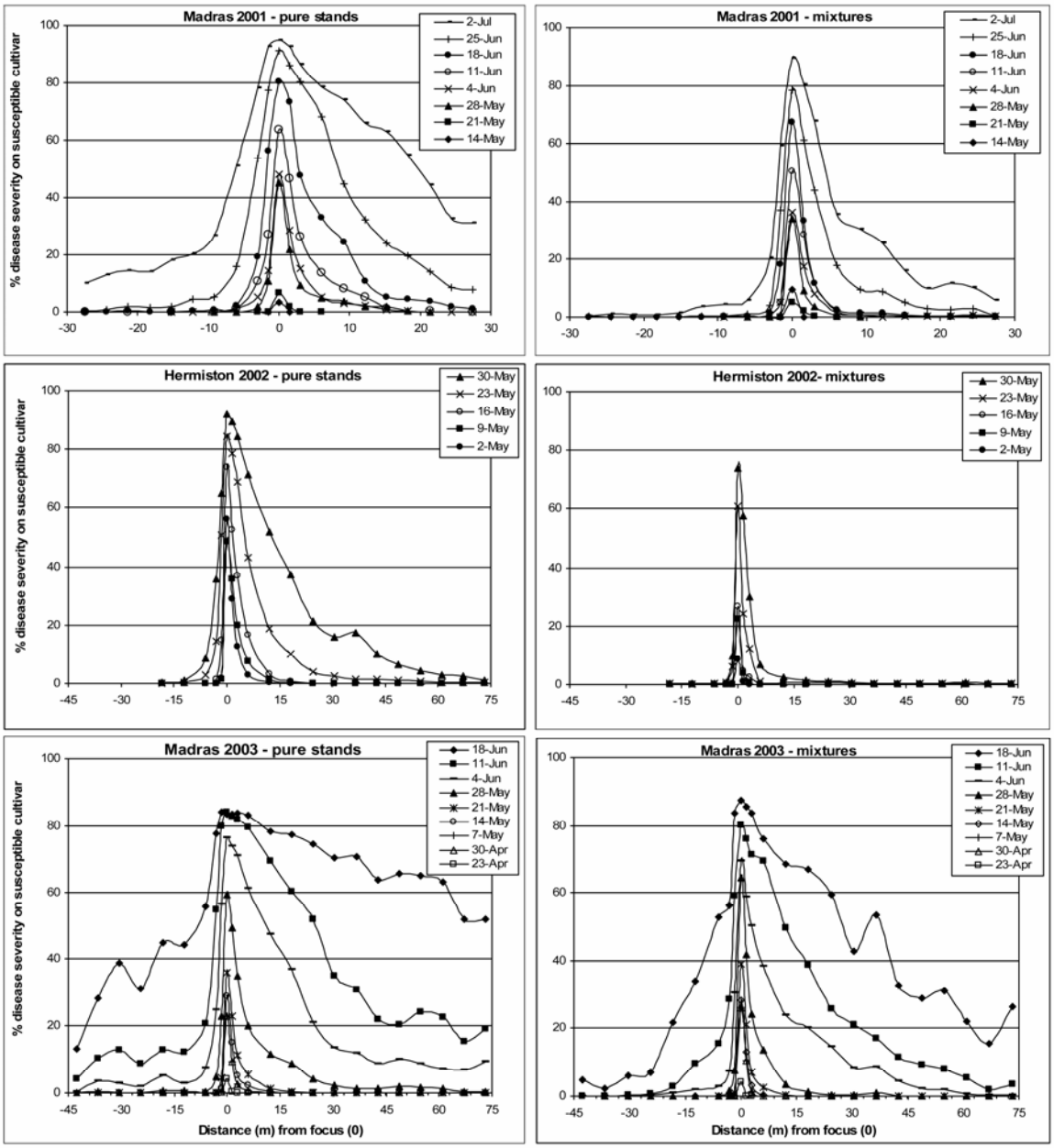

Fig. 3. Percent leaf area covered by lesions of wheat stripe rust along transects upwind (negative numbers) and downwind (positive numbers) from an inoculated focus in field experiments conducted in Hermiston and Madras, OR. Readings were taken on a susceptible cultivar grown in pure stands and on the same susceptible cultivar when mixed with an immune cultivar. Percentages of susceptible plants in the mixtures were 11, 12, and 30% in 2001, 2002, and 2003, respectively. Each data point is the mean of three replicates.

In mixture plots, disease was evaluated on susceptible cv. Jacmar only. Prior to heading, assessments in those plots were conducted on the entire canopy, and those assessments were later corrected for the percentage of 'Jacmar' in the mixture, as determined by tiller counts. After heading, assessments in mixture plots were performed on 'Jacmar' tillers only.

Statistical analysis. For each location-year, two to four severity levels for isopaths were chosen for analysis according to whether severity data were available from at least two treatment replicates on at least five dates for both pure and mixed plots in both the upwind and downwind directions. In this way, at least four velocities were calculated for each treatment-by-direction-by-isopath combination in each experiment.

Linear interpolation was used to estimate the distance reached by each selected disease severity level on each assessment date in each plot. Linear interpolation was used because there was no *a priori* conceptual basis for use of a curvilinear interpolation method. Velocity was calculated in units of meters per week as [distance(week$_n$) – distance(week$_{n-1}$)]/week.

Regression was performed to determine whether velocities increased with respect to time and distance. The SAS procedure PROC AUTOREG (SAS Institute, Cary, NC) was used to test for serial correlation among regression residuals, since the dependent variable velocity was derived from data collected in the same locations over time. With positive serial correlation, the error variance will be too small and confidence intervals too narrow, while the reverse is true for negative serial correlation. Where serial correlation was detected, PROC AUTOREG provided parameter estimates using maximum likelihood, rather than ordinary least-squares regression, with appropriate corrections of slope and standard error estimates.

Velocity was regressed on time separately for each combination of treatment, direction from the focus (upwind or downwind), and isopath level. Residual plots indicated that natural logarithm transformation considerably reduced heteroscedasticity and generally gave lower coefficients of variation and higher adjusted-R^2 values. Velocity was also regressed on distance in the same manner, and in that case, residual plots gave no reason to transform velocity data.

In addition, for each location-year, regressions were performed across treatments and directions to estimate slopes by isopath, across isopaths and treatments to estimate slopes by direction, and across isopaths and directions to estimate slopes by treatment. To separate slope estimates, 95 and 90% confidence intervals were calculated for each slope using the degrees of freedom and standard error pertaining to that slope.

The apparent infection rate r (20) was calculated for each combination of location-year, treatment, and direction by using PROC AUTOREG to regress logit-transformed severity data on time in days for the final five assessment dates of each epidemic and across all available distances starting at 3.1 m from the focus. Slopes of velocity versus time and velocity versus distance were regressed on the resultant r values, also using PROC AUTOREG.

RESULTS

Of the three epidemics discussed here, those in Madras in 2001 and Hermiston in 2002 were less severe than the Madras epidemic of 2003 (Fig. 3). There was more upwind disease development in Madras than in Hermiston, probably because medium- and high-speed winds (>2.2 m/s) blew more consistently from due west in Hermiston, while varying more in direction in Madras (Fig. 4).

The percentage of susceptible cv. Jacmar in the mixture plots as determined by tiller counts was as follows: Madras 2001, 11%; Hermiston 2002, 12%; and Madras 2003, 30%.

Velocity versus time. Velocities increased with time at every isopath level analyzed, although the slopes varied considerably (Tables 1 to 3). In all years, the relationship was exponential, with velocity slopes becoming steeper over time (example in Fig. 5). The slopes of different isopath levels were never significantly different within a year (Tables 1 to 3).

In Madras, velocity slopes were steeper in the downwind than the upwind direction ($P < 0.05$ in both 2001 and 2003, Tables 1 and 3). In Hermiston, the directional difference in slope was not significant (Table 2), although the downwind slope estimate was more than double that of the upwind slope.

Velocity slopes were steeper in pure stands than in mixtures ($P < 0.05$) in Madras in 2001 (Table 1), when disease levels were relatively low and the mean percentage of susceptible cv. Jacmar was only 11%. Mixtures and pure stands did not have significantly different effects on velocity slopes in 2003 in Madras, when the epidemic was more severe and the mean percentage of 'Jacmar' in the mixture was 30% (Table 3). There was also no significant difference in Hermiston in 2002, where the mean slope of velocity in mixtures differed from zero at only $P \leq 0.1019$ (Table 2).

Velocity versus distance. Velocities increased with distance at every isopath. Velocity increased at a mean rate of 0.607 m/week per m in 2001 in Madras, 0.572 m/week per m in 2002 in Hermiston, and 0.598 m/week per m in 2003 in Madras (Tables 4 to 6). The relationship between velocity and distance was approximately linear in all years (example in Fig. 6). In other words, for a given isopath level, the rate of velocity increase remained approximately the same at all sampled distances from the epidemic focus.

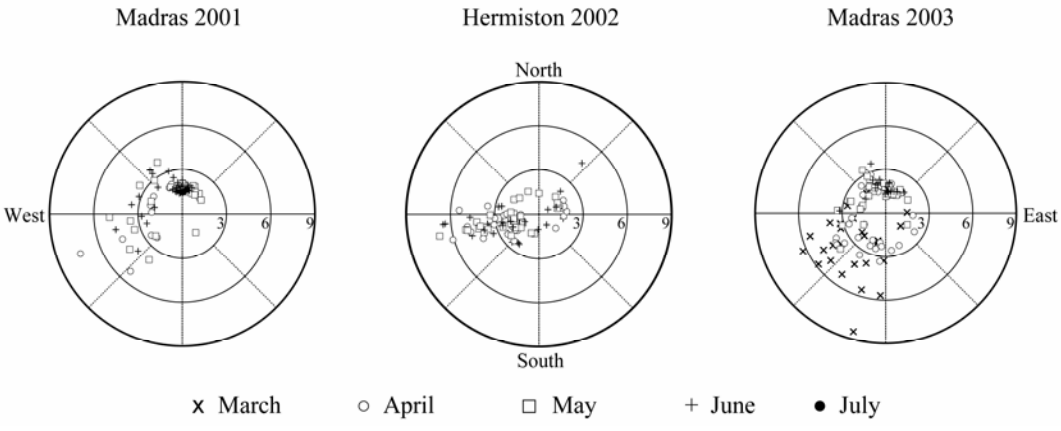

Fig. 4. Wind speeds (m/s) and wind directions measured during three field experiments with wheat stripe rust in Hermiston and Madras, OR. Each data point is the mean wind speed for a day, with data presented from the day of focal inoculation until the day of the last disease assessment.

In Madras, velocity slopes were significantly steeper upwind than downwind in both years ($P \leq 0.05$) (Tables 4 and 6). In Hermiston in 2002, there was no significant directional influence on velocity (Table 5).

Mixtures and pure stands did not have significantly different effects on velocity slopes ($P \leq 0.05$) in Madras in 2001 (Table 4) nor in Hermiston in 2002 (Table 5). In Madras in 2003, however, velocity slopes were significantly steeper in pure stands than in mixtures ($P \leq 0.05$) (Table 6).

Velocity and *r*. Values of the apparent infection rate *r* ranged from 0.162 to 0.404 per day (Fig. 7). Within a location-year, *r* values were lower in mixtures than in pure stands in 2001 and

TABLE 1. Regression of the natural logarithm of epidemic velocity (*v*) on time in plots of winter wheat inoculated with stripe rust in Madras, OR, in 2001

				Slope		
Treatment[x]	Direction	Severity isopath	Regression R^2	Estimate (ln(*v*)/wk)[y]	*P* value[z]	Intercept
Pure stand	Downwind	10%	0.333	0.986	0.0496	–6.413
Pure stand	Downwind	20%	0.644	0.269	0.0017	–0.284
Pure stand	Downwind	30%	0.779	0.364	<0.0001	–1.062
Pure stand	Downwind	40%	0.714	0.356	0.0005	–1.099
Mean			0.326	0.462 a	<0.0001	–1.988
Pure stand	Upwind	10%	0.645	0.340	0.0005	–1.018
Pure stand	Upwind	20%	0.747	0.222	<0.0001	–0.181
Pure stand	Upwind	30%	0.825	0.169	<0.0001	0.188
Pure stand	Upwind	40%	0.822	0.171	<0.0001	0.117
Mean			0.260	0.258 a	<0.0001	–0.471
Mixture	Downwind	10%	0.686	0.300	0.0003	–0.726
Mixture	Downwind	20%	0.896	0.243	<0.0001	–0.388
Mixture	Downwind	30%	0.483	0.186	0.0084	0.005
Mixture	Downwind	40%	0.464	0.174	0.0147	0.045
Mean			0.633	0.228 a	<0.0001	–0.282
Mixture	Upwind	10%	0.221	0.099	0.0769	0.658
Mixture	Upwind	20%	0.479	0.071	0.0042	0.846
Mixture	Upwind	30%	0.416	0.056	0.0173	0.954
Mixture	Upwind	40%	0.460	0.047	0.0153	1.008
Mean			0.248	0.068 b	0.0001	0.868
Pure stand mean			0.267	0.325 a	<0.0001	–0.953
Mixture mean			0.334	0.152 b	<0.0001	0.266
Downwind mean			0.286	0.322 a	<0.0001	–0.941
Upwind mean			0.362	0.149 b	<0.0001	0.307
		10%	0.230	0.354 a	0.0002	–1.281
		20%	0.446	0.192 a	<0.0001	0.097
		30%	0.418	0.201 a	<0.0001	–0.019
		40%	0.381	0.199 a	<0.0001	–0.089

[x] Mixture of 11% of the susceptible cultivar with 89% of the immune one. In the mixture, velocities were calculated based on disease levels on the susceptible cultivar only.

[y] Means followed by the same letter and not separated by a double line are not significantly different at $P \leq 0.05$. Differences among isopath means are also not significant at $P \leq 0.10$.

[z] H_o: Slope = 0.

TABLE 2. Regression of the natural logarithm of epidemic velocity (*v*) on time in plots of winter wheat inoculated with stripe rust in Hermiston, OR, in 2002

				Slope		
Treatment[x]	Direction	Severity isopath	Regression R^2	Estimate (ln(*v*)/wk)[y]	*P* value[z]	Intercept
Pure stand	Downwind	10%	0.497	0.519	0.0049	–3.024
Pure stand	Downwind	20%	0.510	0.609	0.0041	–4.256
Mean			0.482	0.564 a	<0.0001	–3.640
Pure stand	Upwind	10%	0.758	0.562	<0.0001	–5.022
Pure stand	Upwind	20%	0.670	0.462	0.0003	–4.242
Mean			0.459	0.485 a	0.0001	–4.354
Mixture	Downwind	10%	0.360	0.432	0.0666	–3.884
Mixture	Downwind	20%	0.237	0.736	0.2683	–7.233
Mean			0.251	0.457 a	0.0407	–4.237
Mixture	Upwind	10%	0.000	0.005	0.9662	–0.578
Mixture	Upwind	20%	0.007	0.086	0.8546	–1.395
Mean			0.002	0.022	0.8753	–0.740
Pure stands			0.263	0.429 a	<0.0001	–3.161
Mixtures			0.081	0.239 a	0.1019	–2.489
Downwind			0.257	0.403 a	0.0005	–2.952
Upwind			0.110	0.147 a	0.0316	–1.557
		10%	0.184	0.309 a	0.0026	–2.470
		20%	0.178	0.338 a	0.0059	–2.931

[x] Mixture of 12% of the susceptible cultivar with 88% of the immune one. In the mixture, velocities were calculated based on disease levels on the susceptible cultivar only.

[y] Means followed by the same letter and not separated by a double line are not significantly different at $P \leq 0.10$.

[z] H_o: Slope = 0.

2002, but not in 2003. In Madras but not in Hermiston, upwind *r* values were higher than downwind *r* values. Considering Madras upwind epidemic development separately, the rate of increase of velocity in time was positively associated with *r* (Fig. 7A). Across all 12 combinations of location-year, treatment, and direction, velocity increase in space was strongly related to *r (*Fig. 7B) (*P* = 0.004).

DISCUSSION

We have provided strong evidence that focal epidemics of wheat stripe rust expand in time and space with increasing velocity, not with constant velocity. Increasing velocity was observed in three experiments that incorporated 3 years, two locations, both pure stands and mixtures, and epidemics of differing intensity. Our data

TABLE 3. Regression of the natural logarithm of epidemic velocity (*v*) on time in plots of winter wheat inoculated with stripe rust in Madras, OR, in 2003

				Slope		
Treatment[x]	Direction	Isopath	Regression R^2	Estimate (ln(*v*)/wk)[y]	*P* value[z]	Intercept
Pure stand	Downwind	10%	0.916	0.953	<0.0001	–9.467
Pure stand	Downwind	20%	0.976	0.873	<0.0001	–9.050
Pure stand	Downwind	30%	0.870	0.771	<0.0001	–7.948
Mean			0.781	0.832 a	<0.0001	–8.422
Pure stand	Upwind	10%	0.797	0.634	<0.0001	–6.861
Pure stand	Upwind	20%	0.746	0.546	<0.0001	–5.963
Pure stand	Upwind	30%	0.675	0.459	<0.0001	–5.026
Mean			0.639	0.543 b	<0.0001	–5.915
Mixture	Downwind	10%	0.762	0.725	<0.0001	–7.462
Mixture	Downwind	20%	0.976	0.772	<0.0001	–8.058
Mixture	Downwind	30%	0.910	0.726	<0.0001	–7.729
Mean			0.855	0.738 a	<0.0001	–7.707
Mixture	Upwind	10%	0.680	0.562	<0.0001	–6.283
Mixture	Upwind	20%	0.728	0.459	<0.0001	–5.229
Mixture	Upwind	30%	0.500	0.429	0.0005	–4.884
Mean			0.578	0.486 b	<0.0001	–5.489
Pure stand			0.590	0.652 a	<0.0001	–6.747
Mixture			0.633	0.629 a	<0.0001	–6.812
Downwind			0.753	0.771 a	<0.0001	–7.918
Upwind			0.475	0.513 b	<0.0001	–5.646
		10%	0.590	0.673 a	<0.0001	–7.036
		20%	0.611	0.632 a	<0.0001	–6.664
		30%	0.494	0.602 a	<0.0001	–6.493

[x] Mixture of 30% of the susceptible cultivar with 70% of the immune one. In the mixture, velocities were calculated based on disease levels on the susceptible cultivar only.

[y] Means followed by the same letter and not separated by a double line are not significantly different at $P \leq 0.05$. Differences between pure stand and mixture means and among isopath means are also not significant at $P \leq 0.10$.

[z] H_o: Slope = 0.

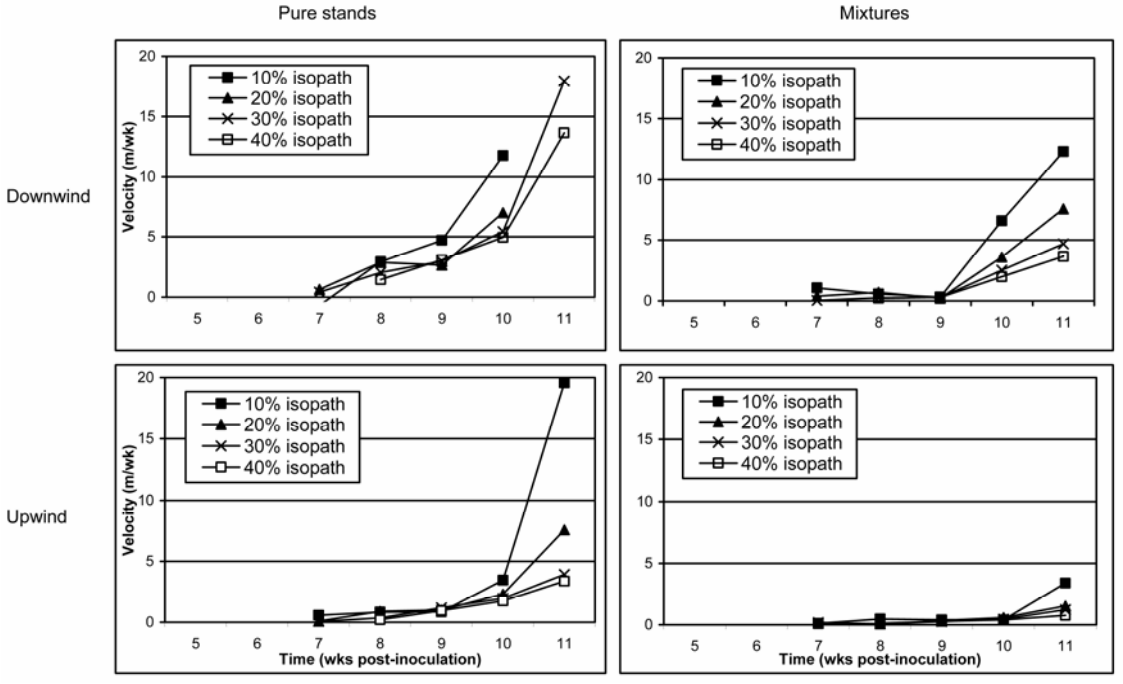

Fig. 5. Epidemic velocities plotted against time, following focal inoculation with *Puccinia striiformis* in winter wheat plots in Madras, OR, in 2001. Readings were taken on a susceptible cultivar grown in pure stands and on the same susceptible cultivar when mixed with an immune cultivar. Each data point is the mean of three replicates.

indicate that the increase of velocity was approximately exponential in time and linear in space. Mechanistic explanations for these relationships will be evaluated in a subsequent publication.

We used elongated, rectangular plots for reasons of economy, because square plots of the same length would have occupied very large areas. However, in 2004 we conducted experiments comparing a limited number of square and elongated, rectangular plots of pure stands at two locations (K. E. Sackett, L. D. Wallace, and C. C. Mundt, *unpublished data*). The results were virtually identical to those presented here: velocity increased approxi-

TABLE 4. Regression of epidemic velocity (v) on distance from artificially established stripe rust foci in winter wheat, Madras, OR, in 2001

Treatment[x]	Direction	Severity isopath	Regression R^2	Slope		Intercept
				Estimate (ln(v)/wk)[y]	P value[z]	
Pure stand	Downwind	10%	0.714	0.578	0.0005	–2.103
Pure stand	Downwind	20%	0.796	0.424	<0.0001	–0.032
Pure stand	Downwind	30%	0.884	0.625	<0.0001	–0.973
Pure stand	Downwind	40%	0.835	0.597	<0.0001	–0.638
Mean			0.813	0.586 a	<0.0001	–1.111
Pure stand	Upwind	10%	0.993	0.817	<0.0001	–2.020
Pure stand	Upwind	20%	0.960	0.607	<0.0001	–0.803
Pure stand	Upwind	30%	0.929	0.505	<0.0001	–0.373
Pure stand	Upwind	40%	0.929	0.506	<0.0001	–0.309
Mean			0.953	0.719 b	<0.0001	–1.243
Mixture	Downwind	10%	0.905	0.663	<0.0001	–1.593
Mixture	Downwind	20%	0.972	0.634	<0.0001	–0.785
Mixture	Downwind	30%	0.873	0.748	<0.0001	–1.100
Mixture	Downwind	40%	0.847	0.752	<0.0001	–0.884
Mean			0.903	0.645 ab	<0.0001	–0.908
Mixture	Upwind	10%	0.951	0.840	<0.0001	–1.432
Mixture	Upwind	20%	0.958	0.625	<0.0001	–0.684
Mixture	Upwind	30%	0.929	0.575	<0.0001	–0.515
Mixture	Upwind	40%	0.855	0.499	<0.0001	–0.378
Mean			0.912	0.732 b	<0.0001	–0.899
Pure stands			0.872	0.601 a	<0.0001	–0.953
Mixtures			0.924	0.638 a	<0.0001	–0.787
Downwind			0.873	0.596 a	<0.0001	–0.905
Upwind			0.951	0.730 b	<0.0001	–1.103
		10%	0.859	0.648 a	<0.0001	–1.470
		20%	0.897	0.527 b	<0.0001	–0.484
		30%	0.940	0.616 ab	<0.0001	–0.721
		40%	0.922	0.585 ab	<0.0001	–0.488

[x] Mixture of 11% of the susceptible cultivar with 89% of the immune one. In the mixture, velocities were calculated based on disease levels on the susceptible cultivar only.

[y] Means followed by the same letter and not separated by a double line are not significantly different at $P \leq 0.05$. Pure stand and mixture means are also not significantly different at $P \leq 0.10$.

[z] H_o: Slope = 0.

TABLE 5. Regression of epidemic velocity (v) on distance from artificially established stripe rust foci in winter wheat, Hermiston, OR, in 2002

Treatment[x]	Direction	Severity isopath	Regression R^2	Slope		Intercept
				Estimate (ln(v)/wk)[y]	P value[z]	
Pure stand	Downwind	10%	0.888	0.609	<0.0001	–1.604
Pure stand	Downwind	20%	0.951	0.572	<0.0001	–0.600
Mean			0.908	0.591 a	<0.0001	–1.066
Pure stand	Upwind	10%	0.874	0.543	<0.0001	–0.558
Pure stand	Upwind	20%	0.886	0.542	<0.0001	–0.506
Mean			0.931	0.576 a	<0.0001	–0.622
Mixture	Downwind	10%	0.702	0.480	0.0025	–0.198
Mixture	Downwind	20%	0.776	0.570	0.0088	–0.089
Mean			0.700	0.493 a	<0.0001	–0.094
Mixture	Upwind	10%	0.658	0.466	0.0044	–0.418
Mixture	Upwind	20%	0.052	0.337	0.6227	–0.082
Mean			0.176	0.376	0.0940	–0.224
Pure stand			0.928	0.580 a	<0.0001	–0.774
Mixture			0.744	0.534 a	<0.0001	–0.329
Downwind			0.918	0.575 a	<0.0001	–0.662
Upwind			0.905	0.547 a	<0.0001	–0.513
		10%	0.918	0.578 a	<0.0001	–0.748
		20%	0.968	0.569 a	<0.0001	–0.455

[x] Mixture of 12% of the susceptible cultivar with 88% of the immune one. In the mixture, velocities were calculated based on disease levels on the susceptible cultivar only.

[y] Means followed by the same letter and not separated by a double line are not significantly different at $P \leq 0.10$.

[z] H_o: Slope = 0.

mately exponentially in time and linearly with distance in all directions. Further, the slopes for plots of ln(velocity) versus time and of velocity versus distance were highly similar between the two plot shapes.

We expected to find that the slopes of velocity versus time would be shallower with increasing isopath levels, due to the flattening of disease gradients over time. Disease gradients did indeed flatten, as shown in Figure 3. Although in some years we observed an inverse relationship between isopath level and velocity slope, differences were not significant, and the relationship was not consistent over all 3 years. The statistical analysis of the gradients, which is complex due to changes in shape and steepness with successive pathogen generations, will be addressed in a separate paper.

TABLE 6. Regression of epidemic velocity (v) on distance from artificially established stripe rust foci in winter wheat, Madras, OR, in 2003

				Slope		
Treatment[x]	Direction	Severity isopath	Regression R^2	Estimate (ln(v)/wk)[y]	P value[z]	Intercept
Pure stand	Downwind	10%	0.968	0.677	<0.0001	–0.512
Pure stand	Downwind	20%	0.967	0.609	<0.0001	0.305
Pure stand	Downwind	30%	0.914	0.650	<0.0001	–0.222
Mean			0.953	0.646 a	<0.0001	–0.126
Pure stand	Upwind	10%	0.989	0.853	<0.0001	–1.411
Pure stand	Upwind	20%	0.910	0.703	<0.0001	–0.601
Pure stand	Upwind	30%	0.993	0.872	<0.0001	–1.129
Mean			0.960	0.808 b	<0.0001	–1.049
Mixture	Downwind	10%	0.751	0.467	<0.0001	0.217
Mixture	Downwind	20%	0.977	0.552	<0.0001	0.288
Mixture	Downwind	30%	0.935	0.614	<0.0001	–0.031
Mean			0.873	0.533 c	<0.0001	0.251
Mixture	Upwind	10%	0.902	0.541	<0.0001	–0.165
Mixture	Upwind	20%	0.994	0.760	<0.0001	–0.715
Mixture	Upwind	30%	0.984	0.803	<0.0001	–0.683
Mean			0.917	0.640 ac	<0.0001	–0.351
Pure stand			0.948	0.679 a	<0.0001	–0.355
Mixture			0.896	0.539 b	<0.0001	0.158
Downwind			0.898	0.575 a	<0.0001	0.174
Upwind			0.946	0.770 b	<0.0001	–0.902
		10%	0.850	0.571 a	<0.0001	–0.163
		20%	0.961	0.588 a	<0.0001	0.118
		30%	0.933	0.656 a	<0.0001	–0.180

[x] Mixture of 30% of the susceptible cultivar with 70% of the immune one. In the mixture, velocities were calculated based on disease levels on the susceptible cultivar only.

[y] Means followed by the same letter and not separated by a double line are not significantly different at $P \leq 0.05$. The 30% isopath slope mean is significantly different from the 20% isopath slope mean, but not from that of the 10% isopath at $P \leq 0.05$.

[z] H_o: Slope = 0.

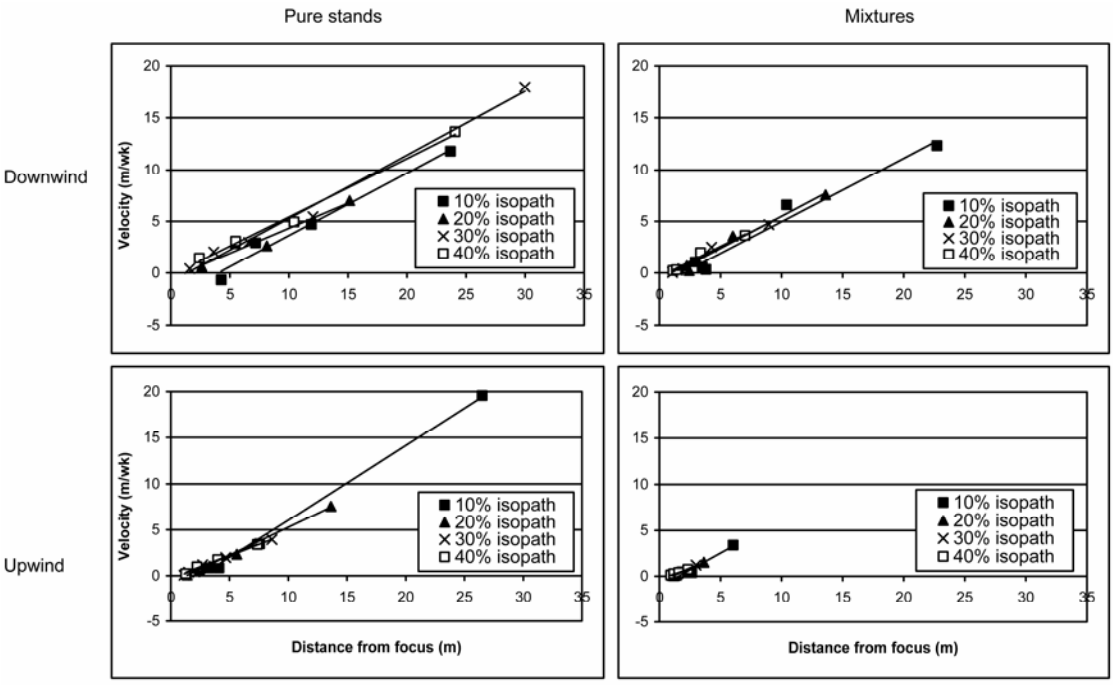

Fig. 6. Epidemic velocities plotted against distance from initial focus, following inoculation with *Puccinia striiformis* in winter wheat plots in Madras, OR, in 2001. Readings were taken on a susceptible cultivar grown in pure stands and on the same susceptible cultivar when mixed with an immune cultivar. Each data point is the mean of three replicates.

Our results also supported our second hypothesis that a reduction in *r* causes velocity to increase more slowly over time. The rates of velocity increase in both time and space were positively associated with *r*. Upwind epidemics in Madras attained a higher *r* than downwind epidemics for reasons that are unclear but may be associated with the greater multidirectionality of wind at that site than at Hermiston. We considered whether the higher upwind *r* in Madras might be an artifact: as our foci were offset from center in the upwind direction, upwind *r* values might have been based on a more proximal part of the disease curve than were downwind *r* values. However, disease was assessed upwind and downwind over the same distances in 2001 at Madras (Fig. 3).

Our data suggest that for a given epidemic, the reduction in *r* must be above some threshold in magnitude to significantly reduce the rate of velocity increase. Values of *r* were lower in mixtures than in pure stand for each combination of location-year and direction except in Madras in 2003, when the susceptible cultivar was 30% of the mixture. In that case, mixtures did not reduce velocity slopes relative to pure stands. The susceptible cultivar constituted only 12% of the mixtures in Hermiston in 2002, but velocity slopes in pure stands and in mixtures were not significantly different. In the Hermiston experiment, the slope estimate in pure stands was substantially higher than that in mixtures, but differences in disease levels and disease development among mixture plots led to a large standard error of the slope. Inspection of Figure 3 reveals a consistent pattern of differences between pure stand and mixture gradients.

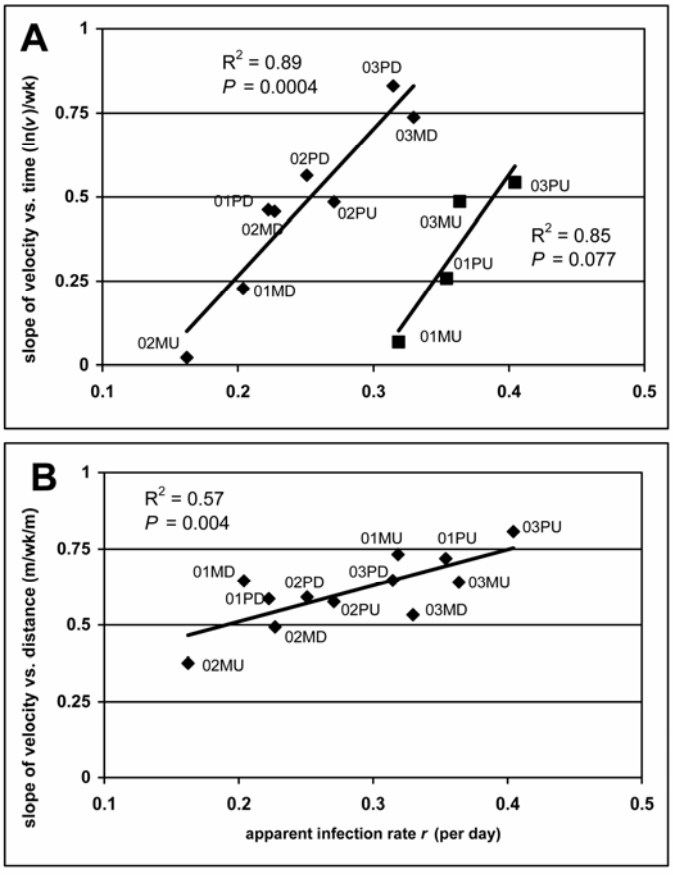

Fig. 7. Slopes of regression relationships between **A,** epidemic velocity versus time and **B,** epidemic velocity versus distance, plotted against apparent infection rate *r*, following inoculation with *Puccinia striiformis* in winter wheat plots in Madras and Hermiston, OR. Data were means of three replicate plots. Data labels 01–03 = years 2001–2003, M = mixture, P = pure stand, D = downwind, and U = upwind. Pure stands were of a susceptible cultivar; mixtures were 1:4 (2001 and 2002) or 1:1 (2003) ratio of susceptible cultivar to immune cultivar.

If a lowering of *r* does slow the increase of velocity, rate-reducing strategies for managing focally initiated diseases will have larger effects when they are executed over larger spatial scales. An example is the use of host genotype diversity, either through coordinated deployment of diverse resistance sources in pure stands over large areas or through large-scale planting of variety mixtures or multilines. Evidence from barley (23), rice (28), and wheat (15; C. Cowger, *unpublished data*) pathosystems suggests that variety mixtures are more effective in reducing disease when applied over larger spatial scales, in part because smaller plots are subject to larger amounts of exogenous inoculum, which is likely to have a higher infection efficiency than endogenously produced inoculum (8,15,22). In the case of focal epidemics, the apparent greater mixture effect over larger spatial scales may also be related to the fact that velocity of epidemic spread increases with time, but differentially according to *r*. A lower-*r* epidemic will travel a shorter distance in a single growing season than a higher-*r* epidemic. For confirmation, one can arbitrarily choose the velocity represented by the second gridline from the bottom in Figure 1B, and following that gridline horizontally, observe that about 1.2 more time units are needed for the epidemics to travel a given distance in mixtures than in pure stands.

In conclusion, evidence is in hand that the velocity of stripe rust spread from disease foci increases over both space and time. The next step is to determine the "domain of validity" (24) for this result. For example, does it hold true under more stable atmospheric conditions, where gradient theory predicts that spore dispersal from the focus may be slower and long-distance dispersal may be relatively less significant (11)? Another issue is the effect of focus size, or source strength. An examination of the literature (4,27; this study) suggests that velocities of wheat stripe rust epidemics are positively correlated with focus size. Thus, focus size may be an important determinant of the spatiotemporal properties of disease spread. A third question is whether our results will be found for other polycyclic foliar pathogens that are wind-dispersed.

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