Lesion Expansion as an Epidemic Component

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ABSTRACT

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A simulator for the enlargement of cohorts of circular lesions on cohorts of host tissue was used to examine five epidemiological parameters: radial rate (mm day⁻¹) of lesion expansion, k_{exp} ; maximum basic infection rate, R_m ; proportion of lesion area as infectious, f ; initial lesion size (mm²), z ; and proportion of susceptible host sites, s . Based on the proportion of disease severity at day 50 and the proportion of the total disease that originated solely from lesion expansion, k_{exp} was the most sensitive of the five parameters. A radial rate of only 0.1 mm day⁻¹ resulted in a proportion of >0.7 of the diseased area that came from lesion expansion. In an extensive survey of phytopathological literature, many plant pathogens had radial rates greater than 0.1 mm day⁻¹, which would result in a proportion of >0.95 of the total disease that comes from lesion

expansion. Susceptible host sites, s , was a sensitive parameter, as this determined the host area into which lesions could expand. Naturally, R_m was a sensitive parameter for the proportion of disease on day 50, as it controlled the overall speed of the epidemic. Initial lesion size was a relatively insensitive parameter, although z interacted significantly with s . The greatest proportion of disease that originated from lesion expansion occurred with fast k_{exp} , small z , and low values of s , R_m , and f . The model was validated with lesion numbers and severities obtained in natural epidemics of *Cercospora medicaginis* on alfalfa and *Exserohilum turcicum* on maize. We recommend that the 'epidemic quintuplet' used to describe polycyclic epidemics be expanded to the 'epidemic sextuplet' with the inclusion of k_{exp} , since lesion expansion is a major component of many polycyclic epidemics.

Additional keywords: epidemic simulation, lesion expansion rates, modeling.

In some plant pathosystems, e.g., several *Cercospora* spp. on various hosts, *Coccomyces hiemalis* on cherry, and *Puccinia* spp. on many hosts, the pathogen causes discrete lesions or pustules that remain constricted in size over the life of the host tissue. In contrast, in a great many other pathosystems, the lesions continue to grow radially or linearly after their initial appearance, until much or all of the host unit is symptomatic. It is this latter group of plant pathosystems that is the subject of analysis in this paper.

The recognition of lesion expansion as an important epidemic component seems to have begun in the early 1960s with the reports of Lapwood (44,45) and Knutson (36) on potato late blight. van der Plank (84) recognized that the growth of individual lesions contributed to the total severity of disease in an epidemic. However, he stated (p. 77 in literature citation 84) that since lesion expansion was not logarithmic, lesion growth "...does not decide the form of increase of a population of lesions, which is logarithmic..." (read exponential). The implication was that new lesions were far more important in an epidemic than expanding lesions. Zadoks and Schein (p. 168 in literature citation 98) also stated that lesion growth was "certainly not a negligible matter [in an epidemic]," but they failed to elaborate further.

A major consequence of lesion expansion in an epidemic is that disease intensifies even though susceptible host tissue is no longer available for infection, as in *Exserohilum turcicum* on fungicide-sprayed sweet corn (6). Also, lesions continue to expand even though environmental conditions are no longer favorable for infection, as in *P. striiformis* on wheat (22). Furthermore, the host area involved in the expansion of lesions gives rise to considerably more area for production of inoculum in a much shorter time (43).

That is, the expanding lesion area is infectious almost immediately, whereas new infections that originate after dispersal of inoculum become infectious only after the passage of one latent period.

The rate of lesion expansion can be considerable depending on the pathosystem. Mehta (53) reported that a single lesion of *Helminthosporium sativum* enlarged over a period of 33 days to cover 23% of the area of a wheat leaf. For *P. striiformis*, the lesions expanded at 19 mm² day⁻¹ (22). On potato, lesions of *Phytophthora infestans* (36) and a severe strain of *Alternaria solani* (61) expanded at >25 mm² day⁻¹. *E. turcicum* on corn has unusually large lesions from expansion over time; these lesions may increase at a rate of >43 mm² day⁻¹ (86). The rates of lesion expansion for more than 40 plant pathosystems are given in Table 1. The fastest rates of lesion expansion found in the literature were 600 mm² day⁻¹ for *Aspergillus niger* causing smut and 250 mm² day⁻¹ for *Fusarium moniliforme* causing endosepsis in figs (81).

The size of lesions at their initial appearance and their subsequent enlargement have long been used by plant breeders and plant pathologists to rank crop cultivars for resistance to various pathogens (Table 2). Johnson and Taylor (33) considered lesion size as a very useful component of resistance. The rate of lesion expansion has also been used to rank the aggressiveness of strains or races of bacterial and fungal pathogens, respectively (Table 2).

Several developers of simulators for plant disease epidemics have been concerned with lesion expansion as an epidemic component (Table 3). Even in the first epidemic simulator, EPDEM, Waggoner (88) had a function for the enlargement of lesions of *Phytophthora infestans*. Since the growth of most pathogens in plant tissues is strongly affected by temperature, many of the modelers used temperature as a modifying factor in their functions for lesion expansion. For powdery mildews, free water on the leaves slows colony growth, and this effect has been modeled (3,68,80). Many modelers recognized the limiting effect of the decreasing area of asymptomatic tissue on the space available into which lesions could expand. Consequently, the correction factor, $1 - y$ (in

which y = proportion of diseased area), has been incorporated into the lesion expansion function of several epidemic simulators (Table 3). Also, several modelers envisioned that lesions would not expand into areas with latent infections, so lesion expansion was limited to only healthy tissue by a correction factor of $1 - y - l$, in which l = proportion of area with latent infections. However, the growth of existing lesions over latent sites in natural systems is not well defined (43). As the density of lesions per leaf increases, the initial size of lesions usually decreases and their rate of expansion slows (70). If leaf age or leaf position affects lesion size or expansion, then this effect can also be included in the model (27). Mundt (56) recommended that a lesion expansion function be added to the simulator EPIMUL to investigate host mixtures, and this recommendation was eventually fulfilled (43).

Emge et al. (22) drew attention to the epidemiological consequences of lesion expansion for *P. striiformis* on wheat. The enlarging lesions continued to sporulate profusely over the total lesion area, and removals (diseased sites past the infectiousness stage) in the basic infection rate equation (p. 40 in literature citation 84) was not a factor in stripe rust epidemics. Additionally, the increase in the basic infection rate, R , as the epidemic intensified caused the shape of the plotted disease progress curve for stripe rust to be

much steeper in the terminal phase compared with progress curves for stem and leaf rusts. Shaner (71) also noted the effect of expanding pustule size on the shape of the progress curve for *P. recondita*.

Vanderplank (85) was very critical of the shape of the simulated, transformed progress curves for *C. apii* on celery (7), but he was unaware that the model contained a lesion expansion function that greatly increased the diseased area. Shrum (74) also illustrated the effect of enlarging lesions on the shape of the progress curves for simulated epidemics of *P. striiformis*.

Of the many epidemic simulators that have been developed with functions for lesion expansion (Table 3), only a few of the researchers seemed to have conducted sensitivity analyses for this function. In his simulator, Shrum (74) used the lesion growth rate of Emge et al. (22) for *P. striiformis*. In the sensitivity analysis, the lesion expansion rate was a very sensitive parameter as determined by the relative changes in the epidemic rate and final amount of disease. For their model of wheat stripe rust, Luo and Zeng (50) also found the rate of lesion expansion to be one of the most sensitive parameters. Johnson and Teng (32) found the rate of lesion expansion to be the most sensitive parameter in their model for early blight on potato. In a general epidemic model (12), in EPIMUL modified with a lesion expansion function (43), and in a simulator

TABLE 1. Rates of lesion expansion determined for various plant pathosystems

Parameter and pathosystem	Note	Rate (per day)	Source ^a	Parameter and pathosystem	Note	Rate (per day)	Source ^a
Area (mm ²)				<i>Botrytis cinerea</i> , tomato	Stems	1.8	58
<i>Alternaria alternata</i> , persimmon	Fruit	2.8–4.6	62	<i>Botrytis squamosa</i> , onion		3.2	82
<i>Alternaria macrospora</i> , cotton		1.2–2.1	76	<i>Colletotrichum graminicola</i> , corn		0.9	47
<i>Alternaria solani</i> , potato	Susceptible cultivar	0.6–1.4	19	<i>Cytospora chrysosperma</i> , aspen	Stress-affected	0.1–4.4	26
	Resistant cultivar	0.02–0.1	19	<i>Diaporthe ambigua</i> , fruit trees	Branch cankers	0.7–1.1	77
		1.2	32	<i>Exserohilum turcicum</i> , corn	Not treated	6.0	15
	Severe strain, susceptible cultivar	25.0	61		Fungicide-treated	2.5	15
	Severe strain, resistant cultivar	18.0	61	<i>Fusarium sambucinum</i> , potato	Tubers	0.2–1.2	69
		1.15–1.35	75	<i>Peridermium harknessii</i> , Ponderosa pine	Galls	0.1	93
<i>Aspergillus niger</i> , fig		600.0	81	<i>Phytophthora capsici</i> , pepper	Roots, susceptible cultivar	7.1	17
<i>Colletotrichum gloeosporioides</i> f. sp. <i>aeschynomene</i> , joint vetch		0.9–10.3	49		Roots, resistant cultivar	1.4	17
<i>Erysiphe graminis</i> f. sp. <i>hordei</i> , barley		1.0–2.5	3	<i>Phytophthora cinnamomi</i> , Eucalyptus sp.		4.0	72
		0.3–1.4	21	<i>Phytophthora cinnamomi</i> , Banksia sp.		6.5	72
		0.7–1.4	59	<i>Phytophthora infestans</i> , potato	Leaf midrib	3.7–7.3	45
		1.4–2.8	79	<i>Phytophthora sojae</i> , soybean	Susceptible cultivar	12.0	92
		1.0–2.7	80		Resistant cultivar	1.0	92
<i>Erysiphe graminis</i> f. sp. <i>tritici</i> , wheat		0.28–1.24	54	<i>Puccinia coronata avenae</i> , oats	Susceptible cultivar	0.048	48
		0.07	70		Resistant cultivar	0.014–0.025	48
<i>Exserohilum turcicum</i> , sweet corn		25.0	6	<i>Puccinia graminis avenae</i> , Avena sterilis		0.005	83
		43.0	86	<i>Pyricularia oryzae</i> , rice		1.7	35
<i>Fusarium moniliforme</i> , fig		225.0	81	<i>Rhynchosporium secalis</i> , barley	Susceptible cultivar	2.5	94
<i>Phaeoisariopsis griseola</i> , bean	No virus	0.36–0.41	5		Resistant cultivar	1.8	94
	Virus-infected	0.2–0.3	5	<i>Septoria nodorum</i> , wheat		1.3	63
<i>Phytophthora infestans</i> , potato	Leaves	26.4–45.2	36		Susceptible cultivars	1.01	42
	Tubers at 13°C	0.6–12.9	41		Resistant cultivars	0.49	42
<i>Phytophthora palmivora</i> , cacao		1.4–2.4	57	<i>Xanthomonas campestris</i> pv. <i>oryzae</i> , rice		10.0–25.0	37
<i>Puccinia recondita</i> f. sp. <i>tritici</i> , wheat		0.02–0.03	40		Susceptible cultivars	14.0–34.0	96
	Pathogen strains	0.002–0.007	46		Resistant cultivars	7.0–21.0	96
	Fast-rusting cultivar	0.04–0.07	71	Radius (mm)			
	Slow-rusting cultivar	0.02–0.03	71	<i>Cercospora apii</i> , celery		0.1–0.5	... ^b
<i>Puccinia sorghi</i> , corn		2.9	86	<i>Cercospora medicaginis</i> , alfalfa		0.1	13
<i>Puccinia striiformis</i> , wheat	Greenhouse	9.0–19.0	22	<i>Cercosporidium personatum</i> , peanut		0.05–0.09	2
	Field	2.0–6.5	22	<i>Colletotrichum acutatum</i> , apple	Fruit	0.4–1.24	34
	Susceptible cultivar	19.0	50	<i>Colletotrichum gloeosporioides</i> , apple	Fruit	0.37–3.1	34
	Resistant cultivar	4.0	50	<i>Colletotrichum orbiculare</i> , cucumber		0.6–3.0	52
<i>Septoria nodorum</i> , wheat	Susceptible cultivars	0.39	42	<i>Diplocarpon rosae</i> , rose	Susceptible cultivar	0.35	... ^c
	Resistant cultivars	0.17	42		Resistant cultivar	0.17	... ^c
<i>Thielaviopsis basicola</i> , tobacco	Susceptible cultivar	0.5	30	<i>Phytophthora infestans</i> , potato	Leaf lamina	1.3–1.5	44
	Resistant cultivar	0.1	30	<i>Pleiochaeta setosa</i> , lupine		0.03–0.04	95
<i>Uromyces appendiculatus</i> , bean		0.14–0.19	5	<i>Sclerotinia sclerotiorum</i> , pear	Fruit, fungicide-treated	0.0–2.8	78
					Fruit, not treated	2.8	78
Length (mm)				<i>Trichothecium roseum</i> , stone fruits		0.03–0.25	29
<i>Anisogramma anomala</i> , hazelnut	Branches	2.7	31	<i>Xanthomonas campestris</i> pv. <i>citri</i> , citrus		0.03–0.04	25
<i>Aphanomyces euteiches</i> , pea	Roots, susceptible cultivar	5.0	38	<i>Xanthomonas campestris</i> pv. <i>citrumelo</i> , citrus		0.01	25
	Roots, resistant cultivar	2.3–3.5	38	<i>Xanthomonas campestris</i> pv. <i>dieffenbachia</i> , anthurium		1.0	97

^a In Literature Cited.

^b R. D. Berger, unpublished data.

^c C. Forcelini and R. D. Berger, unpublished data.

for powdery mildew of barley (28), the various rates of lesion expansion had substantial effects on the final amounts of disease and on the shapes of the disease progress curves. Many of these curve shapes were like the curve shapes observed by Emge et al. (22) for the natural progress of wheat stripe rust, a disease with considerable lesion expansion.

In this paper, we examined lesion expansion as a component of epidemics, with comparisons of rates and amounts of lesion expansion found in the literature. Several epidemiological parameters were examined with a simulator to determine their influence on the final amount of disease and on the proportion of disease that arose solely from lesion expansion.

TABLE 2. Examples in the use of rate or amount of lesion expansion to rank cultivars for resistance or to rank pathogenic strains or races for aggressiveness

Parameter and pathosystem	Source ^a
Ranking of cultivars	
<i>Alternaria solani</i> , potato	19,60,61
<i>Cercospora arachidicola</i> , peanut	55
<i>Cercosporidium personatum</i> , peanut	2,55
<i>Colletotrichum orbiculare</i> , cucumber	52
<i>Erysiphe graminis</i> f. sp. <i>tritici</i> , wheat	54
<i>Microcyclus ulei</i> , rubber tree	18
<i>Phytophthora infestans</i> , potato	44,45
<i>Pleiochaeta setosa</i> , lupine	95
<i>Puccinia coronata avenae</i> , oats	48
<i>Puccinia recondita</i> , wheat	71
<i>Puccinia striiformis</i> , wheat	22,50
<i>Rhynchosporium secalis</i> , barley	94
<i>Sclerotinia sclerotiorum</i> , pear fruit	78
<i>Septoria nodorum</i> , wheat	42,43,63,64
<i>Thielaviopsis basicola</i> , tobacco	30
<i>Xanthomonas campestris</i> pv. <i>citri</i> , citrus	25
Ranking of pathogenic strains or races	
<i>Colletotrichum gloeosporioides</i> f. sp. <i>aeschnomene</i>	49
<i>Diaporthe ambigua</i> , fruit trees	77
<i>Erysiphe graminis</i> f. sp. <i>hordei</i> , barley	79
<i>Erysiphe graminis</i> f. sp. <i>tritici</i> , wheat	54
<i>Fusarium moniliforme</i> , fig	81
<i>Phytophthora infestans</i> , potato tubers	41
<i>Phytophthora sojae</i> , soybean	92
<i>Puccinia graminis avenae</i> , <i>Avena sterilis</i>	83
<i>Puccinia recondita</i> f. sp. <i>tritici</i> , wheat	46
<i>Rhynchosporium secalis</i> , barley	94
<i>Xanthomonas campestris</i> pv. <i>citri</i> , citrus	25
<i>Xanthomonas campestris</i> pv. <i>oryzae</i>	37,96

^a In Literature Cited.

TABLE 3. Some simulation models of plant disease epidemics with functions for lesion expansion

Pathosystem	Function ^a	Source ^b
<i>Alternaria solani</i> , potato	Rate of expansion based on temperature	90
<i>Alternaria solani</i> , potato	Relative rates of lesion expansion	32
<i>Botrytis squamosa</i> , onion	Rate of Sutton et al. (82) adjusted by temperature and relative humidity	1
<i>Cercospora apii</i> , celery	Proportion of disease based on lesion age and limited by $1 - y - l$	7
<i>Erysiphe graminis</i> , barley	Rate of expansion affected by temperature, leaf age, and position, and limited by $1 - y$	4,27
<i>Fusarium oxysporum</i> , Douglas fir	Rate of lesion elongation based on temperature	14
General	Proportional increase of lesion size based on existing lesion area and limited by $1 - y$	12
<i>Helminthosporium maydis</i> , corn	Lesion expansion based on lesion age and temperature	91
<i>Mycosphaerella</i> , <i>Chrysanthemum</i>	Lesion growth based on temperature, relative humidity, cultivar, and limited by $1 - y$	51
<i>Phytophthora infestans</i> , potato	Lesion expansion determined by temperature and leaf wetness	88,89
<i>Phytophthora infestans</i> , potato	Rate of lesion area increase based on lesion age, cultivar, temperature, and limited by $1 - y$	16
<i>Puccinia recondita</i> , wheat	Rate of enlargement of uredinal area based on uredinal age and cultivar	71
<i>Puccinia striiformis</i> , wheat	Rate of expansion based on cultivar and crop growth stage	50
<i>Puccinia striiformis</i> , wheat	Rate of expansion adjusted for age of lesion and plant, temperature, and limited by $1 - y - l$	74
<i>Puccinia striiformis</i> , wheat	Rate of lesion expansion added to EPIMUL, limited by $1 - y - l$	43
<i>Septoria nodorum</i> , wheat	Rate of expansion dependent on temperature and relative humidity	65
<i>Uncinula necator</i> , grape	Rate of colony growth dependent on temperature and rain, and limited by $1 - y$	68
<i>Venturia inaequalis</i> , apple	Rate of growth based on temperature	39

^a $1 - y$ = proportion of asymptomatic tissue, in which y = proportion diseased; $1 - y - l$ = proportion of healthy tissue, in which l = proportion of latent infections.

^b In Literature Cited.

MATERIALS AND METHODS

A subroutine for the enlargement of cohorts of lesions (10) was added to a model for cohorts of host tissue (9) to test the sensitivity of the parameters of lesion expansion considered to be important in an epidemic. These parameters were the radial rate (millimeters per day) of expansion of circular lesions, k_{exp} ; the maximum basic infection rate, R_m ; the proportion of expanded lesion area that was infectious, f ; initial lesion size (square millimeters), i.e., the size of a 1-day-old lesion, z ; and the proportion of total host sites that were susceptible to be infected, s . The size of a host site was considered to be the size of an initial lesion. The concept of susceptible and resistant sites being randomly dispersed in host tissue has been supported by evidence, e.g., for *P. coronata* on oats, in which susceptible and resistant sites occurred side by side (Fig. 1E in literature citation 48). The maximum basic infection rate was interpreted as the rate of infection, in the presence of ample inoculum, on host cohorts that have no infection sites already occupied by a pathogen. In the model, the daily basic infection rate, R , for each cohort was adjusted in a linear function (9) depending on the proportion of sites already diseased or occupied by latent infections. The parameters k_{exp} , R_m , f , z , and s were varied over their ranges of typical values in several combinations. The effect of the length of the latent period, p , on an epidemic has been widely studied (9,12) (Fig. 6.19 in literature citation 98), and this parameter was not examined here. In simulations to perform the sensitivity analyses, the latent period was 6 days, and the host growth increased from 10 cm² on day 0 to 5,000 cm² on day 50 at a logistic rate of 0.25 day⁻¹. The epidemics were initiated with 12 infections distributed in differential latencies over the first 6 days, i.e., two infections on each day. The results were expressed as the proportion of disease on simulated day 50 and the proportion of the total disease on day 50 that resulted solely from lesion expansion.

To validate the simulation model for lesion expansion, information from two experiments with *C. medicaginis* on alfalfa (*Medicago sativa*) in June and July 1991 was used. In a 20 × 20-m plot of recently harvested 'Florida 77' alfalfa, 10 plants were marked for assessment. The fourth and fifth leaves from the base of a shoot on each marked plant were tagged. The area of the tagged leaves was estimated by comparison to standard area diagrams. The time of appearance of individual lesions of *C. medicaginis* on the tagged leaves was noted in assessments made three times per week for 5 weeks. Lesion sizes were estimated on each assessment day by comparison to standard circular diagrams. To perform the validation, the leaf area was set in the model at the mean area (115.6 cm² for June and 91.9 cm² for July) measured in the plots. The

other parameters were set as $k_{\text{exp}} = 0.07 \text{ mm day}^{-1}$, $z = 1 \text{ mm}^2$, $f = 0.7$, and $p = 8$ days. The simulated epidemics were initiated by seven latent infections for June and 12 latent infections for July to achieve the number of lesions on the first assessment date. Two epidemics of *E. turcicum* on maize (87) were also used for validation of the model. For these latter epidemics, the model parameters were $p = 7$ days, $k_{\text{exp}} = 0.5 \text{ mm day}^{-1}$, $z = 2 \text{ mm}^2$, and $f = 0.7$. The best fit to the number of lesions observed over time was obtained by adjusting only the R_m and s parameters. For validation, the logit values of disease severities obtained with the final values of R_m and s used to generate the number of lesions then

were compared with those logit values of the disease estimated in the experimental fields.

RESULTS

Model simulations. Of the five parameters (k_{exp} , z , R_m , f , and s) examined in the model, k_{exp} was the most sensitive, i.e., small changes in this parameter caused great changes in y_{50} . A radial rate of lesion expansion of as little as 0.1 mm day^{-1} caused a great increase in disease severity by day 50 (Fig. 1A, C, E, and G) compared with simulated epidemics with no lesion expansion. For epi-

(continued on the next page)

Fig. 1. Sensitivity analysis of a simulation model for lesion expansion. All simulations were made with a 6-day latent period and begun with 12 latent infections. **A, C, E, G, I, and K,** Simulated disease severity on day 50. **B, D, F, H, J, and L,** Proportion of total disease solely from lesion expansion; note the reversal of the y-axis scales. **A and B,** Rate of lesion expansion, k_{exp} , versus initial lesion size, z ; maximum basic infection rate, R_m , 1.0; proportion of susceptible sites, s , 0.5; and proportion of area as infectious, f , 0.2. **C and D,** k_{exp} versus s ; $R_m = 1.0$, $f = 0$, and $z = 1.0$. **E and F,** k_{exp} versus f ; $R_m = 1.0$, $s = 0.5$, and $z = 1.0$. **G and H,** k_{exp} versus R_m ; $s = 0.5$, $f = 0.25$, and $z = 1.0$. **I and J,** s versus z ; $k_{\text{exp}} = 0.05$, $R_m = 1.0$, and $f = 0.25$. **K and L,** f versus R_m ; $k_{\text{exp}} = 0.2$, $s = 0.25$, and $z = 1.0$.

demics with lesions expanding at 0.1 mm day^{-1} in their radii, a proportion of >0.7 of the total disease was the result of lesion expansion (Fig. 1B, D, F, and H). For those simulated epidemics with high severities of disease on day 50, these epidemics also had high numbers of lesions and fast epidemic rates (data not shown). As found previously in a natural epidemic (22), the immediate infectiousness of the expanded lesion area resulted in a rapid intensification of disease.

s was also a somewhat sensitive parameter over all rates of lesion expansion (Fig. 1C). The disease intensified more rapidly as s increased. k_{exp} strongly interacted with s , since s determined the area into which lesions could expand. That is, a low proportion of susceptible sites for initial infections allowed considerable area for lesion expansion. The inverse of s also determined the maximum proportion of diseased area that resulted solely from lesion expansion (Fig. 1B, D, F, and H).

f was not a particularly sensitive parameter. The effect of this parameter could be detected only at slow k_{exp} ($\leq 0.2 \text{ mm day}^{-1}$) and slow R_m (≤ 1) (Fig. 1E). Even under these conditions of k_{exp} and R_m , there were higher proportions of diseased area that originated from expanding lesions compared with the diseased area from new infections (Fig. 1F).

z was a relatively insensitive parameter. An increase from 1 to 4 mm^2 (for example) caused only a slight increase in final disease (Fig. 1A and I). Naturally, z strongly interacted with s (Fig. 1I), since the total number of possible sites were reduced proportionally as larger initial lesion sizes were used.

R_m was a sensitive parameter (Fig. 1G and K), as has been noted in previous models (Fig. 6.20 in literature citation 98). The effect of higher rates of R_m were more evident at slow k_{exp} ($< 0.2 \text{ mm day}^{-1}$). R_m interacted strongly with f , as the highest amount of final disease occurred at high rates of R_m and high f (Fig. 1K). The

Fig. 1. (continued from the preceding page)

greatest proportions of diseased area that originated from lesion expansion occurred with fast k_{exp} , slow R_m , few s , small z , and low f (reduced sporulation).

Validation. The best fit of simulated to observed numbers of lesions for the epidemics of *C. medicaginis* on alfalfa in June was obtained with $R_m = 2$ and a low s (0.04). For the July epidemic of *C. medicaginis*, the values were $R_m = 10$ and $s = 0.0155$ (Fig. 2A). With these values of R_m and s , the logits of simulated disease severities were also reasonably good fits to the logits of observed disease severities (Fig. 2B).

For the two epidemics of *E. turcicum* on maize (87), the estimated values for R_m were 6 for 'Piracicaba 1' and 10 for 'Piracicaba 2'. s was estimated at 0.005 for both epidemics. With these parameter values, excellent fits of estimated to observed numbers of lesions were obtained (Fig. 3A). The resulting simulated disease severities were also excellent fits to the observed disease severities on the logit scale (Fig. 3B).

DISCUSSION

With the model for cohorts of lesions on cohorts of leaves, the diseased area could be partitioned into areas of initial lesions and areas of lesion expansion. In simulations, an expansion rate of 0.1 mm day^{-1} in the radii of the lesions resulted in >70% of the

total diseased area coming from lesion expansion. Since many pathogens cause lesions that expand much faster than 0.1 mm day^{-1} (Table 1), the percentage of disease from lesion expansion in these pathosystems would be >95% of the total diseased area. Even for rust diseases of cereals, there is some enlargement of pustules over time (71,83) and, for *P. striiformis*, the expansion is extensive (22,50). Therefore, the generalization of Fleming (24) regarding the lack of inclusion of a lesion expansion function in his model for epidemics of cereal rust, i.e., "...[lesion expansion] contributes little to cereal rust spread relative to [new lesions] and so will not be considered further" seems inaccurate.

All simulations were conducted with a constant rate of lesion expansion. In natural epidemics, the rate of lesion expansion may be variable dependent on temperature (3,21,27,35,59,80,81,86) or leaf position (21), slowing with age of the lesion or host (15,37,71, 72,76,92) or with increased density of lesions (71), or increasing with age (30,81,92) or with longer dew periods (23,73,86).

Johnson and Taylor (33) emphasized spore numbers as a very useful variable to monitor epidemics, i.e., "spore production.... measures the sum of the effect of all components of resistance." For some pathosystems, e.g., *Uromyces appendiculatus* on bean, the area of the halo is often five to 15 times the area of the sporulating pustule (11). In those fungal pathosystems in which sporulation does not take place over the entire lesion area, the curve for spore numbers over time would be a rather poor estimate of the

Fig. 2. Validation of the simulation model for lesion expansion with two epidemics of *Cercospora medicaginis* on alfalfa. Parameters were set as maximum basic infection rate (R_m) = 2, proportion of susceptible sites (s) = 0.04, and host area of 115.6 cm^2 for the June epidemic and $R_m = 10$, $s = 0.0155$, and host area of 91.9 cm^2 for the July epidemic. The other parameters were latent period (p) = 8 days, rate of lesion expansion (k_{exp}) = 0.07 mm day^{-1} , initial lesion size (z) = 1 mm^2 , and proportion of area as infectious (f) = 0.7. **A**, Increase in $\log_{10}$ (numbers of lesions). **B**, Increase in disease severity on logit scale.

Fig. 3. Validation of the simulation model for lesion expansion with two epidemics of *Exserohilum turcicum* on maize (data of Vitti et al. [86,87]). Parameters were set as maximum basic infection rate (R_m) = 6 for 'Pira. 1' ('Piracicaba 1') and $R_m = 10$ for 'Pira. 2' ('Piracicaba 2'); latent period (p) = 7 days, proportion of susceptible sites (s) = 0.005, rate of lesion expansion (k_{exp}) = 0.5 mm day^{-1} , proportion of area as infectious (f) = 0.7, initial lesion size (z) = 2 mm^2 , and determinate host area at 100 cm^2 . **A**, Increase in $\log_{10}$ (number of lesions). **B**, Increase in disease severity on logit scale.

total severity of the disease reaction. Although the proportion of expanded area as infectious (f) was not a particularly sensitive parameter in the model, this character had sufficient effect that it should continue to be used by researchers to select cultivars for disease resistance. The value of f would likely range from 0.5 to 0.8 for species of *Cercospora*, *Cercosporidium*, *Helminthosporium*, and *Alternaria*.

The expansion of lesions during the epidemic affects the shape of the disease progress curve (7,12,22,28,71,74). Rouse (66) proposed that lesion expansion could be responsible for the change in shape of the disease progress curve from a logistic type to a Gompertz type. This conclusion may have merit, since lesion expansion was a characteristic of most of the pathosystems in which the disease progress curves were fit better by the Gompertz model than by the logistic model (8). Since lesion expansion gives rise to new inoculum in a shorter time than the cycling through latent periods (43), lesion growth contributes more to the shape of the disease progress curve than van der Plank (84) considered.

Zadoks and Schein (99) proposed that the five values of their 'epidemic quintuplet', i.e., y_0 (initial disease), p (latent period), i (infectious period), N (daily spore production per lesion), and E (spore effectiveness) "...together completely describe the polycyclic epidemic." Because lesion expansion contributes more diseased area than do newly appearing lesions in many polycyclic pathosystems, this descriptive concept should be extended to the 'epidemic sextuplet' with the inclusion of k_{exp} for lesion expansion.

The individual pathogens have various combinations of epidemiological features to go along with their lesion expansion rates. *Phytophthora infestans* is one of the ultimate pathogens with a fast rate of lesion expansion, a short latent period, ample numbers of effective spores, large initial lesion sizes, and infection of hosts with numerous susceptible sites. A pathogen with a combination of features on the other end of the spectrum from *Phytophthora infestans* would cause only mild, tardive epidemics.

Vitti et al. (86) made a comparative, monocyclic, controlled-environment study of *P. sorghi* and *E. turcicum* on maize. *E. turcicum* had a 15-fold faster lesion expansion rate, but only 3% of the disease efficiency (lesions cm^{-2}) and 7% of spores mm^{-2} as maize rust. At the end of the 10-day monocycle, the maximum severity of rust was 12% compared with 5% for *E. turcicum*. However, in their comparative study with four natural, polycyclic epidemics of maize rust and leaf blight that lasted 34 to 70 days (87), the number of lesions per square centimeter were seven- to 100-fold greater for rust, but the final severity was three- to 18-fold greater for *E. turcicum*.

Another interesting epidemiological comparison was between leaf rust (*P. recondita*), with minimal lesion expansion, and yellow rust (*P. striiformis*), with substantial lesion expansion, of wheat (20,67). Yellow rust had nine to 40 times more sporulation per lesion than leaf rust, but leaf rust was eight to 12 times more successful in infection than yellow rust under optimum conditions. In addition, infection by leaf rust occurred over a wider range of temperatures than did infection by yellow rust. Nevertheless, the progeny/parent ratio was three times greater for yellow rust. However, yellow rust has a >200-fold greater rate of lesion expansion than leaf rust (22,43,71). The end result (in simulation) was that leaf rust was slightly more severe than yellow rust, primarily because of a 2-day-shorter latent period (67).

At first examination of a pathosystem, if only a few lesions commonly occur on a host despite the availability of inoculum, this might be considered as a resistant reaction of the host. However, if these few lesions have large expansion, then this would be considered a susceptible reaction, which seems paradoxical. This situation is fairly common, e.g., black pod of cacao, brown rot of stone fruits, Sclerotinia blight of sunflower heads, and Sclerotinia nesting of harvested bean pods in hampers. The few lesions (or fungal colonies) that occur could be from (i) few sites (host resistance or host anatomical features); (ii) the pathogen characteristics

(low numbers of infectious units or low infection efficiency); or (iii) micro-environmental limitations.

In the epidemics of the two plant pathosystems (*C. medicaginis*-alfalfa and *E. turcicum*-maize) used for validation of the lesion expansion model, the curves for the cumulative numbers of lesions tended toward a low asymptotic value. To mimic this effect, the negative power function (9) was used for the relationship of the basic infection rate to the proportion of infected area, with a small proportion of susceptible sites. The patterns for the increase in the number of observed lesions were reasonably mimicked by the model, even though the influence of environmental effects on the basic infection rate was not included in the simulations.

The two epidemics of *C. medicaginis* on alfalfa were quite different. In the June epidemic, the total leaf area was 20% greater than for the July epidemic (115.6 versus 91.9 cm^2), and the apparent infection rate for observed disease was 50% slower ($r = 0.16$ for June versus 0.32 for July). Consequently, there was a great difference in the maximum basic infection rate ($R_m = 2$ for June versus 10 for July) needed in the model to generate curves similar to observed values. The two abbreviated epidemics of *E. turcicum* on maize were quite similar and could have been fit by similar values for R_m .

The lesion expansion rate for *E. turcicum* is known to be much greater than that for *C. medicaginis* (Table 1). In the validations, the radial rates for lesion expansion needed to mimic the increase in disease severity were also greatly different (0.5 mm day^{-1} for *E. turcicum* versus 0.07 mm day^{-1} for *C. medicaginis*). The radial rate of 0.07 mm day^{-1} estimated for *C. medicaginis* in the two epidemics used for validation was quite similar to the rate (0.1 mm day^{-1}) previously reported for this fungus (13). A slower rate of lesion expansion would occur when lesions are near the leaf margins or when lesions are crowded on individual leaflets. For both pathosystems used in validation, the faster increase in epidemic rates and the lesser tendency toward asymptotes in the curves for log₁₀ (numbers of lesions) were because of lesion expansion.

Because lesion expansion is such an important portion of the total disease syndrome of many polycyclic epidemics, the reduction in lesion expansion should be emphasized to manage these pathosystems. A reduction in the diseased area from lesion expansion can be obtained by selection of cultivars with smaller initial lesion sizes and with slow rates of expansion. The application of certain pesticides also can slow the rate of lesion expansion in some plant pathosystems (15,78).

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