Epidemiology / Épidémiologie

Temporal and spatial dynamics of mycosphaerella blight [*Mycosphaerella pinodes*] in field pea

J.X. Zhang, W.G.D. Fernando, and A.G. Xue

Abstract: The temporal and spatial patterns of epidemics of mycosphaerella blight, [*Mycosphaerella pinodes*] in field pea in western Canada were characterized during 2000 and 2001, using mathematical models and geostatistical analysis. The logistic model well described the disease progress over distances and in various directions from the inoculum source. The temporal disease progress rates measured by the linear form of the logistic model ranged from 0.187 d^{-1} for disease progress at the inoculated area to 0.288 d^{-1} for disease progress at 18 m from the inoculum source, and from 0.136 to 0.265 d^{-1} in various directions. By the end of the epidemic in both years, the steepest disease gradient was located upwind, in the north to northwest section of the field, while gradients in the east to south section became flatter. The disease gradients ranged from –0.009 m^{-1} in the downwind direction (south) to –0.183 m^{-1} in the upwind direction (north). The final disease gradients in the field ranged from –0.043 to –0.050 m^{-1}. Disease severity declined by 50% within 1.8 to 15 m and by 90% within 7 to 62 m from the inoculum source in the upwind directions. Disease declined by 50% within 16 to 44 m and by 90% within 55 to 222 m in the downwind directions. On the basis of the final disease gradients in both years, the disease severity declined by 50% and 90% within 8 to 14 m and 40 to 51 m, respectively. Geostatistical analysis showed that the range of the spatial dependence of sampling sites in the field was 18 m within rows in 2000 and across rows in both years. The range of spatial dependence was 22 m in the 45° direction in both years and in the 135° direction in 2000. It was 32 m in the 135° direction in 2001, but no range of spatial dependence was detected within rows in 2001.

Key words: field peas, *Mycosphaerella pinodes*, geostatistics, mathematical models, spatial and temporal patterns.

Résumé : En 2000 et 2001, les profils temporels et spatiaux des épidémies d'ascochytose [*Mycosphaerella pinodes*] dans les champs de pois de l'Ouest canadien ont été étudiés à l'aide de modèles mathématiques et de l'analyse géostatistique. Le modèle logistique représentait bien la progression de la maladie tant pour la distance par rapport à la source d'inoculum que pour la direction par rapport à celle-ci. Le taux de progression temporelle de la maladie, mesuré à l'aide de la forme linéaire du modèle logistique, variait de 0,187 d^{-1} pour la progression de la maladie dans la zone inoculée à 0,288 d^{-1} pour la progression de la maladie à 18 m de la source d'inoculum, et de 0,136 à 0,265 d^{-1} pour les diverses directions. À la fin des épidémies des deux années, le gradient de maladie le plus accentué était situé du côté du vent, dans le secteur nord à nord-ouest du champ, alors que les gradients des secteurs est et sud devenaient plus doux. Les gradients de maladie variaient de –0,009 m^{-1} dans la direction sous le vent (sud) à –0,183 m^{-1} dans la direction du côté du vent (nord). Les gradients de maladie finaux dans le champ variaient de –0,043 à –0,050 m^{-1}. Dans la direction du côté du vent, l'intensité de la maladie était réduite de 50% en dedans de 1,8 à 15 m de la source d'inoculum et de 90% en dedans de 7 à 62 m. Dans la direction sous le vent, la maladie était réduite de 50% en dedans de 16 à 44 m et de 90% en dedans de 55 à 222 m. En se basant sur les gradients de maladie finaux pour les deux années, l'intensité de la maladie a chuté respectivement de 50% et 90% en dedans de 8 à 14 m et de 40 à 51 m. L'analyse géostatistique a montré que la zone de dépendance spatiale des sites d'échantillonnage dans le champ était de 18 m au sein des rangs en 2000 et à travers les rangs lors des deux années. La zone de dépendance spatiale était de 22 m pour une direction de 45° lors des deux années et pour une direction de 135° en 2000. Elle était de 32 m pour une direction de 135° en 2001, alors qu'une zone n'a pu être trouvée au sein des rangs en 2001.

Mots clés : pois de jardin, *Mycosphaerella pinodes*, géostatistique, modèles mathématiques, profils spatiaux et temporels.

Accepted 6 September 2004.

J.X. Zhang and W.G.D. Fernando.[1] Department of Plant Science, University of Manitoba, Winnipeg, MB R3T 2N2, Canada.
A.G. Xue. Eastern Cereal and Oilseed Research Centre, Agriculture and Agri-Food Canada, Ottawa, ON K1A 0C6, Canada.

[1]Corresponding author (e-mail: D_Fernando@umanitoba.ca).

Introduction

Mycosphaerella pinodes (Berk. & Bloxam) Vestergren, the causal agent of mycosphaerella blight, is the most important pathogen of field pea (*Pisum sativum* L.) in western Canada (Xue and Burnett 1994). Annual yield losses due to the disease in western Canada have been estimated at 10% on average, and more than 50% in individual trials (Wallen 1974; Xue et al. 1996). In dry conditions, lesions on leaves remain small, but under favorable temperature and humidity, they will enlarge, coalesce, and become necrotic (Lawyer 1984; Roger et al. 1999a, 1999b). Airborne ascospores and pycnidiospores splashed by rain are the primary sources of inoculum (Lawyer 1984). The ascospores cause the disease over a large area and may be uniformly distributed in a field. It is estimated that ascospores can be transmitted by wind more than 1 km from a source (Lawyer 1984). Pycnidiospores are distributed more locally. Even when large numbers of pycnidiospores are released during a rain event, most will be splashed onto nearby lower leaves, causing a localized disease pattern (Lawyer 1984). Polynomial equations have been fitted to predict the stages of infection, incubation, latency, and disease development as functions of temperature and duration of moisture (Roger et al. 1999b), but characteristics of progress of mycosphaerella blight and its spread in the field under natural conditions are not well understood.

Mathematical models have been used to quantify the temporal and spatial dynamics of diseases in various crops (Madden et al. 1987; Nelson and Campbell 1993; Camann et al. 1995; Paulitz 1996). The monomolecular and logistic models were frequently employed to describe monocyclic and polycyclic diseases, respectively (Campbell and Madden 1990). Epidemic progress can be expressed, in these models, by the absolute rate of change in disease intensity (dy) with respect to change in time (dt) (Campbell and Madden 1990; Nutter and Parker 1997). Similarly, disease spread can be expressed by the absolute rate of change in disease intensity (dy) with respect to change in distance (ds). The observed pattern of decrease in disease intensity with increasing distance from the source is called the disease gradient (Campbell and Madden 1990; Reynolds and Burpee 1997).

Geostatistical analysis has been used to detect the degree of spatial dependence between neighbouring sampling sites in disease spread (Trangmar et al. 1985; Larkin et al. 1995). Dependence is determined by a semivariogram that plots the semivariances versus separation (lag) distance. In the resulting semivariogram, the lag distance at which the semivariance approaches a constant value is called the range of spatial dependence. Samples separated by a distance less than the range are spatially related, while those separated by a distance greater than the range are no longer spatially related. Geostatistical techniques can use random or structured characteristics of spatially distributed variables to quantify spatial dependence. These techniques require less strict assumptions compared with spatial autocorrelation techniques (Larkin et al. 1995; Wu et al. 2000); they require only that variance among samples be a function of the distance of separation (Lecoustre et al. 1989).

The objectives of this study were to analyze the temporal and spatial dynamics of mycosphaerella blight from a central inoculum source in a pea field, using temporal disease-progress models, spatial gradient models, and geostatistical analysis.

Materials and methods

Inoculum preparation

A single-pycnidiospore isolate of *M. pinodes* from a field-pea plant at the Agriculture and Agri-Food Canada research farm of the Morden Research Centre, Morden, Manitoba, was used in 1999. The isolate was cultured on potato dextrose agar at 20 °C, under a 14-h photoperiod provided by cool-white fluorescent light. Pycnidiospores were washed from the surface of 14-day-old cultures with sterile water containing 0.05% Tween® 20. The resulting pycnidiospore suspension was filtered through two layers of cheesecloth and adjusted to a concentration of 10^5/mL. Plants of field pea 'Profi', at the 4- to 6-node stage (approximately 2-week-old) were inoculated at a rate of 0.5 mL per plant with a DeVilbiss® model 15 atomizer (DeVilbiss Co., Somerset, Penn.). Disease was allowed to develop on inoculated plants in the field in 1999. At the end of the season, the symptomatic residues were harvested and used for inoculation in 2000. A similar method was applied to produce diseased plant residues in 2000 for inoculation in 2001.

Experimental site

Experiments were conducted at the Morden Research Centre, Agriculture and Agri-Food Canada, in 2000 and 2001, in areas isolated by at least 300 m from other pea plantings. The fields were planted on 4 May 2000 and 15 May 2001. Each year, the experimental area was 42 m × 42 m divided into 49 contiguous squares of 6 m × 6 m. Plants in the central square were inoculated at the 4- to 6-node stage on 8 June in both years by homogeneously spreading 3 kg of pieces of the infected pea residues.

Disease assessment

Disease was assessed as percentage of leaf area with symptoms (LAS), beginning with the onset of symptoms in the inoculated area and continuing until harvest. Assessments were carried out twice a week for a total of 9 times each year. There were a total of 64 sampling sites, with a 6-m space between each other. Ten tagged plants at each sampling site were assessed for LAS on each assessment date. The mean LAS of the 10 plants represented the severity at each sampling site, and the mean LAS of the 64 sites represented the mean disease severity of the field, which was used to analyze disease progress during the season in the whole field. Disease severity at each of the sampling sites, which surrounded the inoculated area in a concentric arrangement at 0, 1, 2, 6, 12, and 18 m from the edge of the inoculated area, was incorporated into an analysis of disease progress at different distances from the inoculated area. The mean disease severity of plants at the sampling sites located at 0, 1, 2, 6, 12, and 18 m from the edge of the inoculated area in the north, east, south, and west directions from the inoculated area was calculated to assess disease progress and gradients in those directions. Disease severity at sites 0, 0.7, 1.4, 4.25, 8.5, 17, and 25.5 m to the northeast, southeast, southwest, and northwest of the inoculated area was

used to analyze disease progress and disease gradients in these four directions.

Disease progress

The logistic model, $y = 1/\{1 + [(1 - y_0)/y_0] \exp(-ct)\}$ and its linear form $\ln[y/(1 - y)] = \ln[y/(1 - y_0)] + ct$, was used to analyze the temporal progress of mycosphaerella blight based on the nature of the disease progress curves and was compared with the monomolecular model, $y = 1 - (1 - y_0) \exp(-ct)$ and its linear form $\ln[1/(1 - y)] = \ln[1/(1 - y_0)] + ct$, which describes monocyclic diseases (Campbell and Madden 1990). In both models, y is a percentage of LAS, y_0 is the initial LAS, c is the rate parameter (slope) of disease progress curve, and t is number of days after inoculation. The two models were tested by linear regression analysis of logit-transformed observed values versus number of days after inoculation (Nutter and Parker 1997). The appropriateness of each model was determined on the basis of the coefficient of determination (R^2), plots of transformed predicted values and observed disease severity against time, and plots of residuals versus predicted values to examine patterns (Campbell and Madden 1990; Nutter and Parker 1997). Each year in the current study, the logistical model was fitted to disease progress in the field, disease progress over the six measured distances from the inoculated area, and disease progress in the eight directions.

Disease gradients

According to the patterns of decrease of disease at increasing distance from the inoculated area, the exponential model of disease spread, $y = a \exp(-bs)$ and its linear form $\ln(y) = \ln(a) - bs$ (Campbell and Madden 1990), was used to analyze the mycosphaerella blight final gradients and gradients in each direction of the field in both years. A rate parameter (b) describing the disease gradient in each direction was calculated to compare the gradients among directions and to develop integrated equations describing the final gradients. The rate parameter was obtained by plotting the logarithm-transformed LAS versus distance in metres with the linear form of the exponential model, where y is LAS, a is a constant, b is the rate parameter, and s is the distance in metres from the inoculum source. Distances at which LAS declined by 50% (S_{50}) and 90% (S_{90}) were calculated for the final disease gradient and for gradients in different directions, based on the linear exponential model. Data of LAS collected at each distance in each direction on each sampling date was used to determine the disease gradient in each direction. Mean LAS at each distance on the last assessment date was recorded for analysis of the final disease gradient in the field and to compare the final disease gradients of both years.

Geostatistical analysis

Geostatistical analysis was conducted on the mean disease severity at each sampling site, using GS$^+$ version 5.1 software (Gamma Design Software, Plainwell, Mich.) to determine whether disease was caused by the experimental inoculation or by endemic inoculum and to determine dependence between neighbouring sampling sites over time and direction. Spatial dependence was evaluated using the semivariance, $\gamma(h)$, that was defined as one half the average squared difference between pairs of sample-data values separated by a given lag distance (h). A semivariogram was obtained by plotting semivariance versus lag distance. Analysis of spatial dependence over time was conducted in four directions, 0°, 45°, 90°, and 135°, using data from nine assessments, where 0° represents the direction within seed rows from east to west and 90° is the direction across rows from north to south.

Results

Disease progress

At the experimental site, daily mean temperatures fluctuated from 12 to 27 °C during the epidemic seasons (June to July) in both years. Relative humidity ranged from 47% to 99%. These environmental conditions were generally conducive to the occurrence and development of mycosphaerella blight. Symptoms of mycosphaerella blight were observed on plants in the inoculated area 15 days after inoculation in 2000 and 22 days after inoculation in 2001. Disease severity in the fields (Figs. 1A and 1C) increased over time in both years.

The logistic model better described disease progress at each distance from the inoculum source ($R^2 = 0.78$ to 0.95, $P < 0.02$) than did the monomolecular model ($R^2 = 0.38$ to 0.73, $P \leq 0.07$) in both years (Table 1), suggesting that mycosphaerella blight fit an S-shaped curve describing polycyclic diseases. The rate parameter of the logistic model ranged from 0.187 d^{-1} in the inoculated area to 0.281 d^{-1} at 18 m from the inoculated area in 2000, and from 0.233 d^{-1} in the inoculated area to 0.288 d^{-1} at 18 m in 2001 (Table 1). The slopes of the disease progress curves at distances further from the inoculum source became steeper, and the slopes were steeper at the corresponding distance in 2001 than in 2000 (Table 1). Epidemic onset, as determined by examining intercepts of logit-transformed LAS versus time, was delayed with increasing distance from the inoculum source in both years because the intercepts maintained a descending trend with increasing distance (Table 1).

The disease progress over time in each direction in both years was also well described by the logistic model. In 2000, the slopes clockwise from south to northwest were steeper than those in other directions (Table 2). However, in 2001, similar slopes in all directions indicated similar disease progress, although there was a trend to a slightly shallower slope in the south and a slightly steeper slope in the northwest. The slopes in all directions ranged from 0.136 to 0.215 d^{-1} in 2000 and from 0.240 to 0.265 d^{-1} in 2001, with higher slopes in 2001 than in 2000 in corresponding directions (Table 2). Disease occurred earlier to the east of the inoculated area in 2000 and to the south in 2001, as indicated by the intercepts, while disease was delayed most in the northwest of the inoculated area in both years (Table 2). The mean disease progress curves for 2000 (Figs. 1A and 1B) and 2001 (Figs. 1C and 1D) were well described by the integrated and linear expressions of the logistic model developed in this study.

Fig. 1. Progress of mycosphaerella blight [*Mycosphaerella pinodes*] in field pea over time in the whole field in 2000 (A) and 2001 (C) in western Canada. The bars on the progress curves indicate standard error. Regression of transformed percentage of leaf area with symptoms (LAS) of mycosphaerella blight versus the number of days after inoculation in 2000 (B) and 2001 (D), respectively. LAS percentage was transformed by $\ln[y/(1 - y)]$, the linear form of the logistic model.

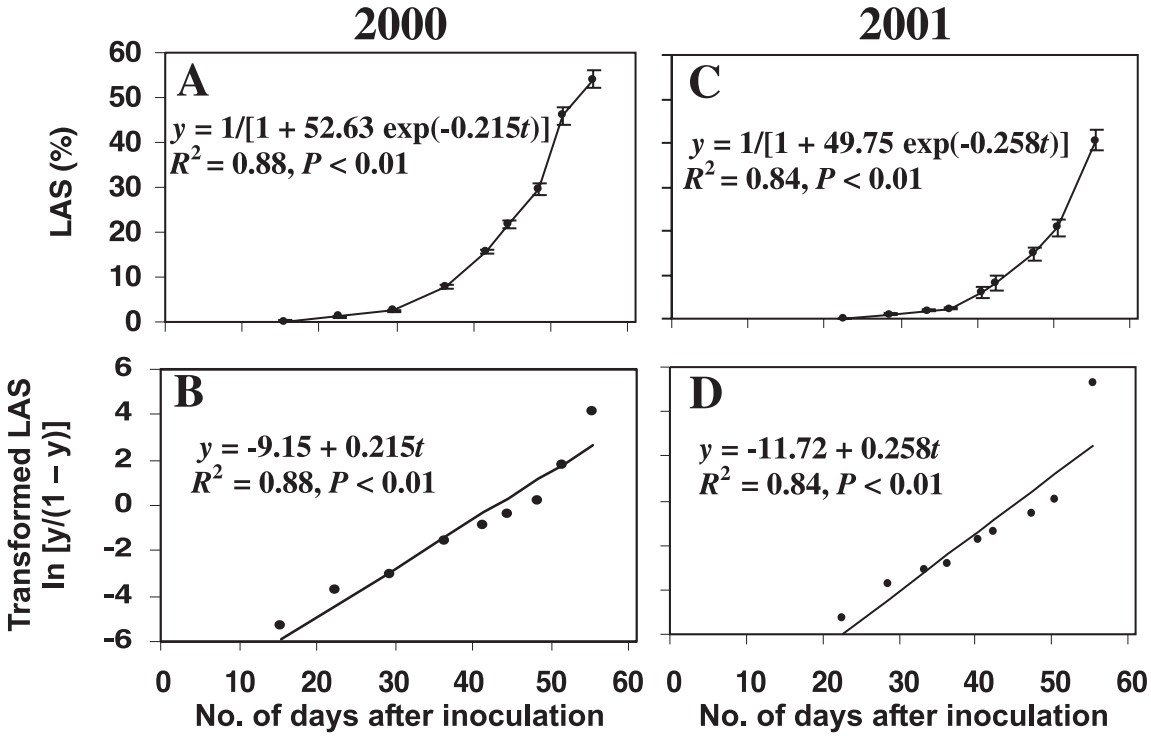


Table 1. Comparison of intercepts and of rate parameters for mycosphaerella blight [*Mycosphaerella pinodes*] progress curves in field pea, at various distances from an inoculated area in western Canada in 2000 and 2001, based on linear forms of monomolecular and logistic models.

		Year 2000			Year 2001		
Distance from inoculum source (m)	Model	Intercept, y_0*	Rate parameter, c (d^{-1})*	R^2	Intercept, y_0*	Rate parameter, c (d^{-1})*	R^2
0	Monomolecular	–2.39±0.134	0.112±0.012	0.64	–3.34±0.155	0.126±0.013	0.71
	Logistic	–6.27±0.360	0.187±0.022	0.88	–8.30±0.450	0.233±0.012	0.89
1	Monomolecular	–2.15±0.131	0.091±0.003	0.42	–3.72±0.145	0.131±0.004	0.73
	Logistic	–8.39±0.561	0.218±0.032	0.85	–9.09±0.387	0.252±0.014	0.94
2	Monomolecular	–2.10±0.126	0.092±0.007	0.41	–3.81±0.139	0.129±0.011	0.65
	Logistic	–9.31±0.624	0.232±0.022	0.86	–11.58±0.668	0.294±0.022	0.95
6	Monomolecular	–2.22±0.163	0.088±0.006	0.45	–3.23±0.126	0.096±0.007	0.42
	Logistic	–9.22±0.477	0.231±0.031	0.88	–12.15±0.724	0.271±0.018	0.84
12	Monomolecular	–2.22±0.112	0.086±0.006	0.45	–3.23±0.170	0.097±0.015	0.40
	Logistic	–9.91±0.701	0.251±0.019	0.88	–13.36±0.664	0.285±0.019	0.82
18	Monomolecular	–2.69±0.191	0.112±0.013	0.54	–3.17±0.134	0.099±0.011	0.38
	Logistic	–0.90±0.575	0.281±0.032	0.90	–13.35±0.678	0.288±0.023	0.78

Note: The slope (c) was generated by regressing the linear form $\ln[y/(1 - y)] = \ln[y/(1 - y_0)] + ct$ of a logistic model and the linear form $\ln[1/(1 - y)] = \ln[1/(1 - y_0)] + ct$ of a monomolecular model, where y is the percentage of leaf area with symptoms (LAS), y_0 is the initial LAS, and t is the number of days after inoculation. R^2, coefficients of determination.

*Data are followed by standard error.

The mean disease progress curves for both years had steeper slopes compared with the disease progress curves in specific directions, except for the northwest in 2000 and the northwest and southwest in 2001 (Table 2). Compared with the intercepts of the specific directions, the intercepts of the mean disease progress curves in both years indicate a later disease onset, showing that the mean disease-progress model underestimated disease onset in both years (Table 2).

Disease gradients

The exponential model explained the high variance of disease decrease from the inoculated area in different direc-

Table 2. Comparison of intercepts and of rate parameters for the mean progress curve and directional progress curves of mycosphaerella blight [*Mycosphaerella pinodes*] in field pea, in western Canada in 2000 and 2001, based on the linear form of a logistic model.

Direction from inoculum source	Year 2000		Year 2001	
	Intercept, y_0	Rate parameter, c (d^{-1})	Intercept, y_0	Rate parameter, c (d^{-1})
North	–6.89±0.230	0.139±0.022	–10.64±0.483	0.251±0.033
Northeast	–7.01±0.421	0.136±0.024	–10.94±0.427	0.252±0.028
East	–6.42±0.322	0.136±0.018	–10.84±0.355	0.250±0.036
Southeast	–6.90±0.292	0.143±0.011	–10.28±0.553	0.245±0.034
South	–7.88±0.233	0.192±0.034	–10.03±0.621	0.240±0.018
Southwest	–8.65±0.477	0.208±0.033	–10.83±0.648	0.258±0.045
West	–8.39±0.565	0.199±0.027	–10.34±0.667	0.248±0.036
Northwest	–8.97±0.577	0.215±0.035	–11.25±0.625	0.265±0.038
Mean*	–9.15±0.458	0.215±0.032	–11.72±0.579	0.258±0.057

Note: Data are followed by standard error. The slope (c) was generated by regressing the linear form $\ln[y/(1 - y)] = \ln[y/(1 - y_0)] + ct$ of a logistic model, where y is the percentage of leaf area with symptoms (LAS), y_0 is the initial LAS, and t is the number of days after inoculation.

*Mean disease progress curve generated by the mean percentages of LAS on nine assessment dates.

Table 3. Comparison of gradients of mycosphaerella blight [*Mycosphaerella pinodes*] of field pea in various directions from the

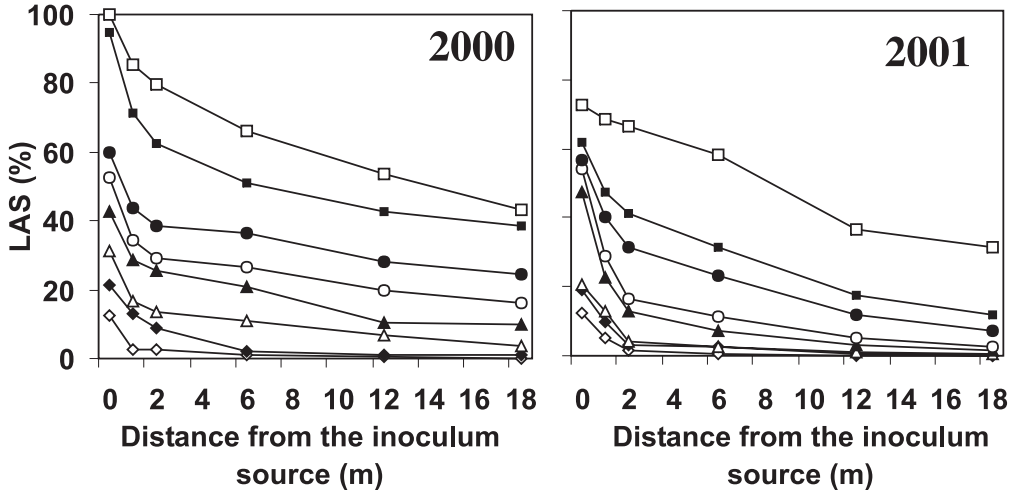
Day*	North		Northeast		East		Southeast	
	b	r	b	r	b	r	b	r
Year 2000								
22	–0.018±0.003	–0.95	–0.017±0.002	–0.95	–0.017±0.003	–0.94	–0.017±0.002	–0.95
36	–0.053±0.009	–0.94	–0.047±0.004	–0.98	–0.032±0.009	–0.87	–0.039±0.007	–0.94
44	–0.086±0.009	–0.98	–0.076±0.007	–0.97	–0.054±0.014	–0.88	–0.067±0.013	–0.93
51	–0.413±0.043	–0.99	–0.258±0.015	–0.99	–0.263±0.109	–0.80	–0.173±0.047	–0.89
55	–0.054±0.009	–0.98	–0.040±0.004	–0.98	–0.024±0.001	–0.99	–0.014±0.001	–0.88
Year 2001								
28	–0.017±0.002	–0.96	–0.017±0.001	–0.97	–0.018±0.004	–0.92	–0.018±0.003	–0.93
36	–0.033±0.013	–0.81	–0.033±0.009	–0.88	–0.067±0.001	–0.96	–0.060±0.006	–0.98
42	–0.106±0.014	–0.95	–0.106±0.011	–0.97	–0.097±0.021	–0.93	–0.094±0.012	–0.96
50	–0.166±0.025	–0.97	–0.188±0.009	–0.99	–0.264±0.013	–0.98	–0.227±0.015	–0.99
55	–0.183±0.025	–0.98	–0.064±0.003	–0.99	–0.021±0.006	–0.90	–0.020±0.004	–0.88

Note: The slope (b) was generated by regressing $\ln(y) = \ln(a) - bs$, where y is the percentage of leaf area with symptoms (LAS), s is the distance in observed values; all are significant ($P = 0.02$).

*Number of days after inoculation.

tions (Table 3). The disease severity on each sampling date decreased in a negative exponential pattern with increasing distance from the inoculated area in both years (Fig. 2). Therefore, the exponential model, with distance from the inoculum source in metres (s) as the independent variable and $\ln(y)$ as the dependent variable, was used to compare the disease gradients in eight directions in both years. In 2000, disease started to spread from the inoculated area 22 days after inoculation, with similar gradients in all directions, as described by b (Table 3). By day 36, disease gradients in all directions increased, with the steepest gradient in the north-to-northeast section and the shallowest gradient in the east-to-south section. These patterns indicate that more disease occurred in plants in the east-to-south section than in other directions, at distances further from the inoculum source, whereas fewer numbers of infected plants occurred as distance increased in the north-to-northeast direction. By day 44, steeper disease gradients occurred in all directions compared with those on the previous assessment dates, although gradient patterns were similar to those of the previous sampling date. The steepest gradient remained in the north during this period, and the shallowest gradient in the east. By day 51, disease gradients in all directions had the greatest increase with the steepest slope in the north and the shallowest slope in the southeast. By the end of the season, disease gradients became much shallower in all directions than those on the previous assessment dates because more plants at further distances were infected (Table 3). The steepest slope in 2000 was consistently in the north because much less disease occurred in plants at further distances in this direction than in other directions (Fig. 3), and the shallowest slope was consistently in the southeast because disease severity in plants at further distances was much higher in this direction than in other directions (Table 3, Fig. 3).

Fig. 2. Gradients of mycosphaerella blight [*Mycosphaerella pinodes*] of field pea over distance from the inoculated area at each assessment time in 2000 and 2001 in western Canada. Gradient curves were made on days 22 and 28 (◇), days 29 and 33 (◆), days 36 and 36 (Δ), days 41 and 40 (▲), days 44 and 42 (○), days 48 and 47 (●), days 51 and 50 (■), and days 55 and 55 (□) in 2000 and 2001, respectively. The integrated expressions of the exponential model describing the final disease gradient curves (□) were $y = 0.89 \exp(-0.043s)$ for 2000 and $y = 0.731 \exp(-0.05s)$ for 2001. LAS, leaf area with symptoms.



inoculum source over time, in western Canada in 2000 and 2001.

South		Southwest		West		Northwest	
b	*r*	*b*	*r*	*b*	*r*	*b*	*r*
–0.018±0.004	–0.91	–0.018±0.002	–0.95	–0.017±0.003	–0.95	–0.016±0.003	–0.91
–0.037±0.007	–0.94	–0.042±0.004	–0.98	–0.042±0.006	–0.96	–0.044±0.005	–0.96
–0.068±0.018	–0.91	–0.069±0.005	–0.98	–0.071±0.007	–0.97	–0.072±0.007	–0.97
–0.382±0.095	–0.94	–0.301±0.048	–0.94	–0.383±0.074	–0.95	–0.266±0.023	–0.99
–0.042±0.002	–0.99	–0.025±0.006	–0.90	–0.034±0.008	–0.93	–0.044±0.005	–0.97
–0.017±0.005	–0.88	–0.018±0.002	–0.94	–0.018±0.003	–0.93	–0.016±0.003	–0.91
–0.063±0.007	–0.98	–0.048±0.008	–0.95	–0.049±0.016	–0.87	–0.026±0.009	–0.81
–0.084±0.018	–0.93	–0.098±0.001	–0.98	–0.105±0.014	–0.96	–0.101±0.010	–0.96
–0.193±0.012	–0.98	–0.153±0.023	–0.95	–0.139±0.022	–0.96	–0.107±0.014	–0.97
–0.009±0.002	–0.92	–0.040±0.003	–0.98	–0.049±0.005	–0.97	–0.075±0.005	–0.97

metres from the edge of the inoculated area, and *a* is a constant of integration. *r*, correlation coefficients of back-transformed predicted values versus the

In 2001, by day 28 after inoculation, spread of disease from the inoculated area occurred in all directions, showing similar gradients (Table 3). Steeper gradients occurred in the east-to-south direction, and shallower gradients occurred in the northwest-to-northeast direction by day 36 (Table 3). These spread patterns showed that more disease occurred in the east-to-south direction adjacent to the inoculated area during this period, than in the northwest-to-northeast direction. The gradient increased greatly in all directions between day 36 and day 50, during which disease gradients remained highest in the east-to-southeast direction and lowest in the northwest. By the end of the season (day 55), disease gradients in all directions became flatter, as indicated by decreases in slope (Table 3). In 2001, the plants in the east-to-south section of the inoculated area had shallower gradients with higher disease severity, while plants in the northwest-to-north direction had lower disease severities and the steepest gradients (Table 3, Fig. 3). The final dis-

Fig. 3. Disease spread from the inoculated area in the field, assessed on day 55 in both 2000 and 2001. The central square with S (source) was the inoculated area with infected residue. The darker color indicates higher disease severity (leaf area with symptoms).

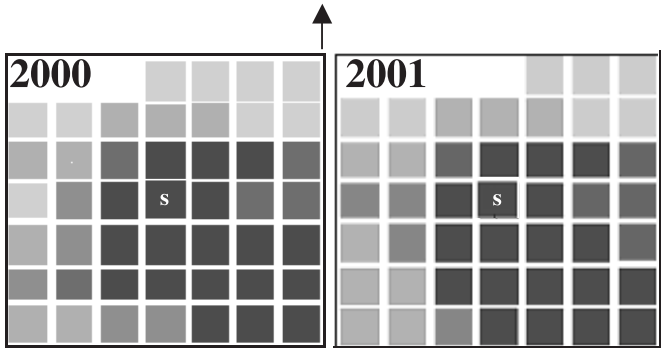


Table 4. Comparison of the final disease gradients, directional disease gradients, and disease decline with distance from the inoculum source for mycosphaerella blight [*Mycosphaerella pinodes*] in field pea in western Canada in 2000 and 2001.

Direction from inoculum source	Intercept, *i**	Slope, *b**	R^2	*r*	S_{50} (m)	S_{90} (m)
Year 2000						
North	–0.127±0.081	–0.054±0.009	0.90	–0.98	10.5	40.3
Northeast	–0.067±0.044	–0.040±0.004	0.95	–0.98	15.6	55.8
East	–0.079±0.008	–0.024±0.001	0.99	–0.99	26.0	94.2
Southeast	–0.080±0.040	–0.014±0.034	0.77	–0.88	42.8	158.8
South	0.016±0.020	–0.042±0.002	0.99	–0.99	16.0	55.2
Southwest	–0.142±0.040	–0.025±0.006	0.81	–0.90	22.0	86.4
West	–0.180±0.075	–0.034±0.008	0.82	–0.93	15.1	62.4
Northwest	–0.160±0.056	–0.044±0.005	0.95	–0.97	11.9	48.5
Final gradient†	–0.101±0.034	–0.043±0.004	0.97	–0.99	13.8	51.3
Year 2001						
North	–1.017±0.237	–0.183±0.025	0.94	–0.98	1.8	7.0
Northeast	–0.309±0.036	–0.064±0.003	0.99	–0.99	6.0	31.1
East	–0.315±0.056	–0.021±0.006	0.75	–0.90	18.0	94.6
Southeast	–0.291±0.050	–0.020±0.004	0.82	–0.88	21.2	105.9
South	–0.302±0.018	–0.009±0.002	0.84	–0.92	43.5	222.3
Southwest	–0.256±0.033	–0.040±0.003	0.98	–0.98	10.9	60.2
West	–0.245±0.047	–0.049±0.005	0.96	–0.97	9.1	42.0
Northwest	–0.245±0.062	–0.075±0.005	0.98	–0.97	6.0	27.4
Final gradient†	–0.313±0.038	–0.050±0.004	0.97	–0.98	7.7	40.2

Note: The slope (*b*) was generated by regressing $\ln(y) = \ln(a) - bs$, where *y* is the percentage of leaf area with symptoms (LAS), *s* is the distance in metres from the edge of the inoculated area, and *a* is a constant of integration. R^2, coefficients of determination obtained by regressing the transformed observed values versus *s* with $\ln(y) = \ln(a) - bs$; *r*, correlation coefficients of back-transformed predicted values versus the observed values with $y = a \exp(-bs)$; S_{50} and S_{90}, distances in metres at which the disease declined by 50% and 90%, respectively.

*Data are followed by standard error.

†Final gradients in the field were derived from the mean percentages of LAS at each distance level.

ease gradients for both years were characterized by the linear and integrated expressions of the exponential model (Table 4, Fig. 2). The final disease gradient in 2000 was shallower than in 2001 as described by slope (Table 4), showing that disease spread further from the inoculum source in 2000 than in 2001.

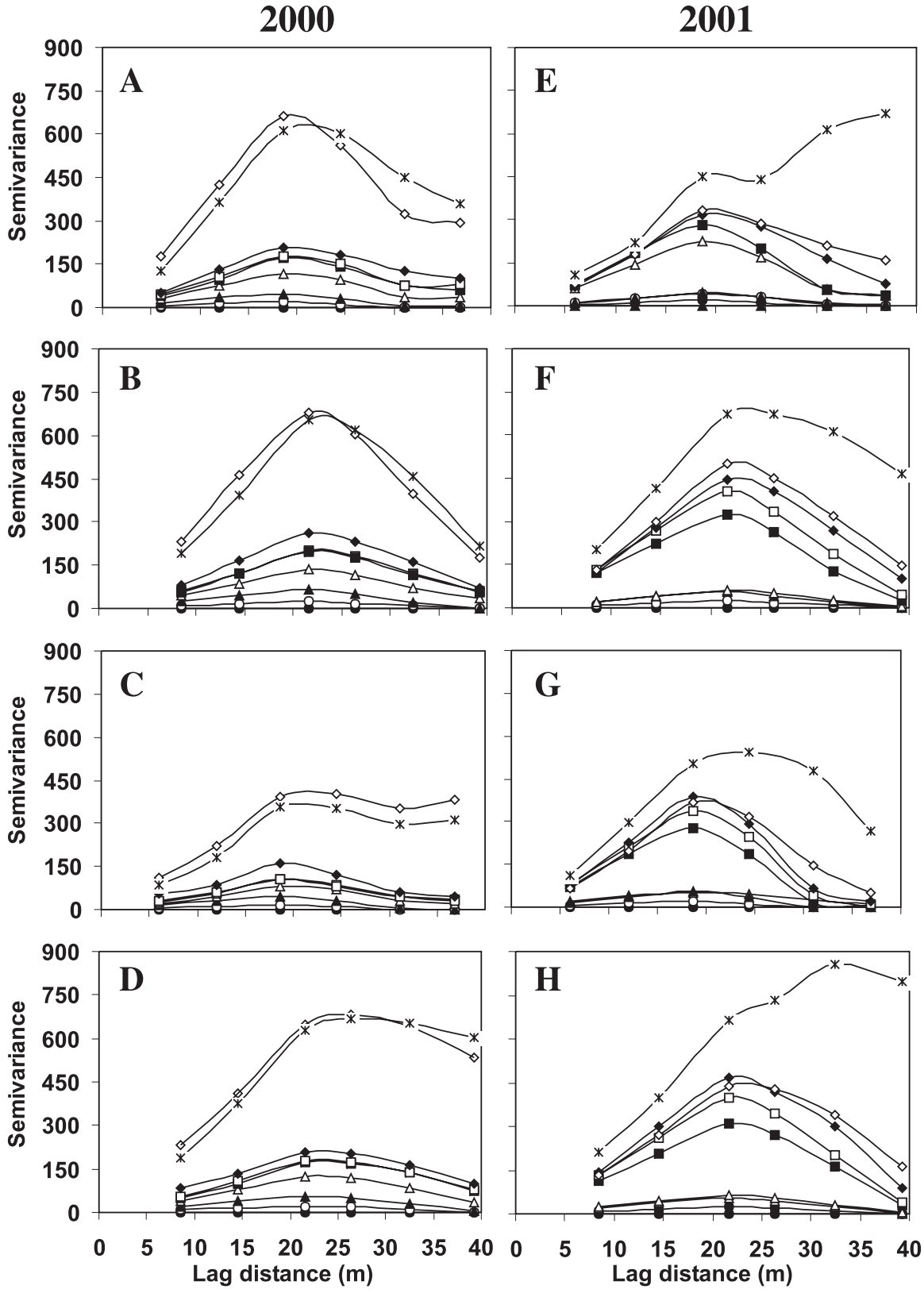
The distances at which disease declined by 50% (S_{50}) and 90% (S_{90}) were calculated in all directions, using the exponential model, based on the data collected at the end of the season (Table 4). In 2000, disease severity declined by 50% within 10 to 16 m and by 90% within 40 to 63 m from the inoculum source in upwind directions, north, west, and northwest (wind-direction data were obtained from the Morden Weather Station of Environment Canada located approximately at 1 km from the experimental field). Disease declined by 50% within 16 to 43 m and by 90% within 55 to 159 m from the inoculum source in downwind directions, east, southeast, and south. In 2001, disease severity declined by 50% within 1.8 to 9.1 m and by 90% within 7 to 42 m from the inoculum source in the upwind directions. Disease declined by 50% within 18 to 44 m and by 90% within 94 to 223 m from the inoculum source in the downwind directions. In the upwind directions, disease spread furthest to the west of the inoculum source and least to the north in both years. In the downwind directions, disease spread furthest to the southeast of the inoculum source and least to the south in 2000, but furthest to the south and least to the east in 2001 on the basis of the predicted values for S_{50} and S_{90} (Table 4). For the final disease gradients, disease declined by 50% and 90% within 13 and 52 m in 2000, and within 7 and 41 m in 2001, respectively (Table 4).

Geostatistical analysis

Changes in the patterns of semivariograms were observed over time in both years (Fig. 4). The semivariogram patterns were similar in all directions over time before day 48 in 2000 and day 50 in 2001. Semivariogram slopes increased over time within the 18-m distance from the edge of the inoculated area, within and across rows, and then decreased after 18 m before day 48 in 2000 and day 50 in 2001 (Figs. 4A, 4C, 4E, and 4G). However, semivariogram slopes increased over time within 22 m and then decreased in the 45° and 135° directions (Figs. 4B, 4D, 4F, and 4H) before day 48 in 2000 and day 50 in 2001.

The degree of spatial dependence differed with the direction (strong anisotropy) in both years. In general, similar increases in semivariance were detected in directions over time, before day 48 at 0°, 45°, and 135° in 2000 (Figs. 4A, 4B, and 4D) and before day 50 at 45°, 90°, and 135° in 2001 (Figs. 4F–4H). The minimum increase in semivariance over time was observed before day 48 across rows in 2000 (Fig. 4C) and before day 50 within rows in 2001 (Fig. 4E). In directions 0° and 45° in 2000 (Figs. 4A and 4B) and 45° and 90° in 2001 (Figs. 4F and 4G), semivariance increased with increasing lag distance within 18 and 22 m, then decreased. However, semivariance in the 90° and 135° direc-

Fig. 4. Oriented semivariograms for percentage of leaf area with symptoms (LAS) caused by *Mycosphaerella pinodes* in field pea on nine assessment dates in 2000 and 2001, in western Canada. (A) and (E), within rows (0°), representing the direction from east to west. (B) and (F), 45°. (C) and (G), across rows (90°), representing the direction from north to south. (D) and (H), 135°. Semivariograms were made on days 15 and 22 (●), days 22 and 28 (○), days 29 and 33 (▲), days 36 and 36 (Δ), days 41 and 40 (■), days 44 and 42 (□), days 48 and 47 (◆), days 51 and 50 (◇), and days 55 and 55 (*) in 2000 and 2001, respectively.



tions after day 48 in 2000 (Figs. 4C and 4D) and in the 0° and 135° directions after day 50 in 2001 (Figs. 4E and 4H) remained relatively high over time beyond 18- and 22-m lag distances. These semivariogram patterns were caused by the high disease severity that occurred in plants further to the south and southeast of the inoculated area in 2000 and to the east and southeast in 2001 (Fig. 3), showing that the disease gradients became flatter and that the disease spread further in those directions over time during the late season. Of particular interest, the semivariance that continued to increase in the late season in the 0° and 135° directions in 2001 suggested that secondary infection had occurred in both directions. The range of spatial dependence was 18 m within and across rows and 22 m in the 45° and 135° directions in 2000 (Figs. 4A–4D). However, the range of spatial dependence in the 135° direction (Fig. 4H) was approximately 32 m on the last assessment date, and no range of spatial dependence was detected in the 0° direction in 2001 (Fig. 4E).

Discussion

These experiments were conducted using infested pea residue as the source of primary inoculum, and under natural conditions. To our knowledge, this is the first comprehensive study to describe quantitatively the temporal and spatial dynamics of mycosphaerella blight in field pea with mathematical models in combination with geostatistical analysis. The logistic model effectively described disease progress over time at various distances and in various directions from the inoculum source. An exponential model quantified the disease gradients over time and in different directions by comparison of slopes of the model. The distance to which the disease spread from the inoculum source was estimated in each direction, using the exponential model. This type of information may help to detect disease development in the field and be useful for disease management, such as for the isolation of a new pea field from previous years' infested pea fields. Geostatistical analysis helped us to understand the spatial dependence between neighbouring sampling sites.

We chose the logistic model to describe disease progress in the field, based on the biological features of mycosphaerella blight progress and the coefficient of determination (R^2). Although the logistic model was the best for describing disease progress at different distances and in different directions in most cases, the best mathematical equation sometimes varied over some distances and directions. For example, the linear model, rather than the logistic model, was the best for describing the disease progress curves in the inoculated area and at 1 m from the inoculum source ($R^2 = 0.93$, $P < 0.01$) in 2001 (data not shown). The exponential model was the best model for the progress curves in the north and northwest in 2001. These results suggested that the most appropriate model was determined by the features of local disease progress in the specific distance and direction from the source.

The logistic model is usually used for describing progress curves of polycyclic diseases (Campbell and Madden 1990; Reynolds and Burpee 1997). Mycosphaerella blight is a polycyclic disease, since pycnidiospores of *M. pinodes* can cause secondary local infections under favorable environmental conditions. Although secondary infections in plants were observed only in the south-to-east section during the epidemics in both years, the logistic model adequately described the disease progress in all directions. However, this does not mean that secondary infections occurred equally in all directions.

Unlike the S-shaped disease progress curve in 2000, the disease progress curve in 2001 continued to increase until the end of the season and appeared to fit an exponential curve. This may be related to the dry weather during the early stages of the epidemic and, thus, to a delay of disease onset in 2001. Therefore, disease progress continued to follow an exponential phase during the late epidemic in 2001.

In the disease spatial spread, we found that the disease gradient was closely related to wind directions. Prevailing winds were observed from the north, northwest, and frequently from the west in both years. Disease gradients in the upwind directions were steeper than those in the corresponding downwind directions. Gregory (1968), who examined data from Cammack (1958) on the disease gradients of rust caused by *Puccinia polysora* Underw. on maize, concluded that when all directions were averaged, the mean gradient in the field was steeper than that of the downwind gradient. Although we used a different model, our results agree with this conclusion.

Variation in disease progress and disease gradients in different directions suggests that a final disease-progress model or a final disease-gradient model in a large area does not effectively describe the local characteristics of disease progress or gradient. Therefore, it is necessary that a specific model be developed to characterize disease progress or disease gradient in a specific space and time if disease is distributed unevenly over the field. Furthermore, a model combining direction, distance, and time may be best to describe the disease in space and time.

The exponential model has been used to measure the mean disease gradient caused by airborne conidia or ascospores in other pathosystems. Fernando et al. (1997) measured the gradients of fusarium head blight caused by ascospores of *Gibberella zeae* (Schwein.) Petch, which ranged from –0.11 to –0.79 m^{-1}. Gradients of wheat leaf rust caused by *Puccinia recondita* Roberge ex Desmaz. in the study by Mundt (1989) ranged from –0.07 to –0.28 m^{-1}. Fitt et al. (1987) calculated a gradient value of –0.015 for *Venturia inaequalis* (Cooke) Wint. in apple orchards. In our work, the final disease gradients (–0.043 to –0.050 m^{-1}) were shallower than those in the studies of Fernando et al. (1997) and Mundt (1989) and steeper than in the study by Fitt et al. (1987). This indicates that mycosphaerella blight on pea may spread further within a field than *G. zeae* and *P. recondita*. A point source of inoculum usually produces a steeper disease gradient than an area source (Gregory 1968). Fernando et al. (1997) used a 1-m^2 inoculum source in a field of 50 m × 50 m to measure gradients of fusarium head blight while Mundt (1989) used a 13.4-m^2 inoculum source in a field that was 173 m in length to test the spread of wheat rust. Our inoculum source was 36 m^2 in area in a field of 42 m × 42 m, much larger than the source areas in the two other studies. This is probably one of the reasons for the shallower gradients of mycosphaerella blight. In ad-

dition, the steepness of gradient is also probably related to the wind speed and characteristics of varietal resistance.

In this study, it was found that the distance at which disease declined by 90% varied with wind direction. This may have been due to variable wind speed and frequency during spore release. In the upwind directions, disease severity declined by 90% within 7 m to the north of the inoculated area in 2001, and within 62 m to the west in 2000. In contrast, in the downwind directions, disease severity declined by 90% within 55 m to the south in 2000, to 222 m to the south in 2001. We observed that most ascospores were trapped within the first 25 days during both years (data not shown). In the 25 days, northerly and northwesterly winds were the most frequent in both years. Most likely, more spores were carried to the south and southeast from the inoculum source, so that disease spread further in these directions from the inoculum source than in other directions. Ascospores can move 1.6 km or more with wind from a source (Lawyer 1984). Therefore, although the longest predicted distance at which disease declined by 90% in the downwind directions was 222 m, this does not preclude the possibility of spore dispersal further away. With limited field dimensions, we could not assess disease spread beyond our field size. However, these predicted distances are still important in designing strategies for management of mycosphaerella blight in the field. For example, a field seeded to field pea for the first time that is isolated at least 300 m from a severely infected field may have much lower disease risk than fields within this distance. A field check for disease occurrence and fungicide application might be advocated for growers whose fields are located within a 300-m area from a diseased pea field, if environmental conditions favor *M. pinodes*. If a local disease focus exists in a large-area field, a fungicide application to this focus area and plants around it at an early stage should be considered.

We used both the exponential model and geostatistical analysis to describe spatial patterns of mycosphaerella blight in the field. Although the exponential model provided a great deal of information on disease gradients in various directions, the geostatistical analysis provided information that the exponential model did not. Firstly, geostatistical analysis was applied in the present study to visualize features of spatial heterogenicity of mycosphaerella blight that cannot be detected by direct observation, such as gradients described by the exponential model. In geostatistical analysis, semivariograms showed large variation in various directions in both years, suggesting a strong anisotropy of spatial patterns. This anisotropy may be caused by uneven distribution of spores from the source due to a range of wind speeds and directions. Secondly, geostatistical analysis was also used to determine the range of spatial dependence between sampling sites. Sampling sites were distributed around the central inoculated area and were located within 18 m from the inoculum source in the 0° and 90° directions, and within 22 m in the 45° and 135° directions. A 18- to 22-m range of spatial dependence in most directions was determined by geostatistical analysis, suggesting that the sampling sites located on the same side of the inoculum source were spatially related, but those located on opposite sides were not. This suggested that disease gradients were caused by inoculum arising from the inoculated area in most directions. Thirdly, in geostatistical analysis, semivariograms were assessed at increasing times after inoculation to examine how spatial patterns evolved with time in the fields in 2000 and 2001. Comparison between semivariograms can provide information on disease spread. For example, the obvious spatial dependence between sampling sites in all directions were not found until the fourth assessment date in 2000 (36 days after inoculation) and the fifth assessment date in 2001 (40 days after inoculation). The later establishment of spatial dependence between sampling sites in 2001 may be caused by the late seeding date and drier conditions at the early growth stage of pea in the year.

Patterns of disease spread vary not only with weather conditions, but also with the degree of varietal susceptibility (Gregory 1968). We examined epidemics of mycosphaerella blight in susceptible pea cultivar, 'Profi', during both years. Varieties with different levels of susceptibility could be used to determine how varietal susceptibility affects disease progress and gradients under natural conditions.

Acknowledgements

This research was funded by grants from Manitoba Pulse Growers Association to AGX and from Agri-Food, Research and Development Initiative and the Natural Sciences and Engineering Research Council of Canada to WGDF. We thank B. Conner, L. Yager, and B. Dyck for their kind support in field experiments, M. Kiehn for help in disease assessment, G. Babbish for help in geostatistics, H. Ahmed for valuable comments, and K. Prince for proofreading the manuscript.

References

Camann, M.A., Culbreath, A.K., Pickering, J., Todd, J.W., and Demski, J.W. 1995. Spatial and temporal patterns of spotted wilt epidemics in peanut. Phytopathology, 85: 879–885.

Cammack, R.H. 1958. Factors affecting infection gradients from a point source of *Puccinia polysora* in a plot of *Zea mays*. Ann. Appl. Biol. 46: 186–197.

Campbell, C.L., and Madden, L.V. 1990. Introduction to plant disease epidemiology. John Wiley & Sons, New York. pp. 75–287.

Fernando, W.G.D., Paulitz, T.C., Seaman, W.L., Dutilleul, P., and Miller, J.D. 1997. Head blight gradients caused by *Gibberella zeae* from area sources of inoculum in wheat field plots. Phytopathology, 87: 414–421.

Fitt, B.D.L., Gregory, P.H., Todd, A.D., McCartney, H.A, and Macdonald, O.C. 1987. Spore dispersal and plant disease gradients; a comparison between two empirical models. J. Phytopathology, 118: 227–242.

Gregory, P.H. 1968. Interpreting plant disease dispersal gradients. Annu. Rev. Phytopathol. 6: 189–212.

Larkin, R.P., Gumpertz, M.L., and Ristaino, J.B. 1995. Geostatistical analysis of *Phytophthora* epidemic development in commercial bell pepper fields. Phytopathology, 85: 191–203.

Lawyer, A.S. 1984. Diseases caused by *Ascochyta* spp. *In* Compendium of pea diseases. *Edited by* D.J. Hagedorn. American Phytopathological Society (APS) Press, St. Paul, Minn. pp. 11–15.

Lecoustre, R., Fargette, D., Fauquet, C., and de Reffye, P. 1989. Analysis and mapping of the spatial spread of African cassava

mosaic virus using geostatistics and kriging technique. Phytopathology, 79: 913–920.

Madden, L.V., Pirone, T.P., and Raccah, B. 1987. Analysis of spatial patterns of virus diseased tobacco plants. Phytopathology, 77: 1409–1417.

Mundt, C.C. 1989. Use of the modified Gregory model to describe primary disease gradients of wheat leaf rust produced from area sources of inoculum. Phytopathology, 79: 241–246.

Nelson, S.C., and Campbell, C.L. 1993. Comparative spatial analysis of foliar epidemics on white clover caused by viruses, fungi, and a bacterium. Phytopathology, 83: 288–301.

Nutter, F.W., and Parker, S.K. 1997. Fitting disease progress curves using EPIMODEL. *In* Exercises in plant disease epidemiology. *Edited by* L.J. Francl and D.A. Neher. American Phytopathology Society, St. Paul, Minn. pp. 24–28.

Paulitz, C.T. 1996. Diurnal release of ascospores by *Gibberella zeae* in inoculated wheat plots. Plant Dis. 80: 674–678.

Reynolds, K.L., and Burpee, L.L. 1997. Application of disease spread model. *In* Exercises in plant disease epidemiology. *Edited by* L.J. Francl and D.A. Neher. American Phytopathology Society, St. Paul, Minn. pp. 64–67.

Roger, C., Tivoli, B., and Huber, L. 1999a. Effects of interrupted wet periods and different temperatures on the development of ascochyta blight caused by *Mycosphaerella pinodes* on pea (*Pisum sativum*) seedlings. Plant Pathol. (London), 48: 10–18.

Roger, C., Tivoli, B., and Huber, L. 1999b. Effects of temperature and moisture on disease and fruit body development of *Mycosphaerella pinodes* on pea (*Pisum sativum*). Plant Pathol. (London), 48: 1–9.

Trangmar, B.B., Yost, R.S., and Uhara. 1985. Application of geostatistics of spatial studies of soil properties. Adv. Agron. 38: 45–74.

Wallen, V.R. 1974. Influence of three ascochyta diseases of peas on plant development and yield. Can. Plant Dis. Surv. 54: 86–90.

Wu, B.M., van Bruggen, A.H.C., Subbarao, K.V., and Pennings, G.G.H. 2000. Spatial analysis of lettuce downy mildew using geostatistics and geographic information systems. Phytopathology, 91: 134–142.

Xue, A.G., and Burnett, P.A. 1994. Diseases of field peas in central Alberta. Can. Plant Dis. Surv. 74: 102–103.

Xue, A.G., Warkentin, T.D., and Kenaschuk, E.O. 1996. Mycosphaerella blight of field peas — potential damage and fungicide control. *In* Proceedings of Manitoba Agri-Forum. 5–6 January 1996, Winnipeg, Man. Available from A.G. Xue at Agriculture and Agri-Food Canada, 960 Carling Avenue, Ottawa, ON K1A 0C5, Canada. pp. 48–53.