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Effect of weather conditions on local spread and infection by pea bacterial blight (*Pseudomonas syringae* pv. *pisi*)

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Abstract

The effect of weather conditions on simultaneous local (plant to plant) spread and infection of peas (*Pisum sativum*) with bacterial blight (*Pseudomonas syringae* pv. *pisi*) was investigated by exposing susceptible bait plants for 24 h periods in infected field plots. Following exposure, bait plants were maintained in a glasshouse. Disease symptoms were recorded on 55 out of a total of 105 days on which plants were exposed. Nearly all of these infection events (53) were associated with the occurrence of rain. A series of Generalised Linear Models was fitted to the data to examine the relationships of the mean number of lesions (m) or the proportion of bait plants infected (p) to various weather variables and disease levels in the plots. Rainfall rate and wind run were the most important explanatory variables for the mean number of lesions followed by maximum temperature, rainfall duration, rainfall in the previous week and disease incidence in the surrounding crop. However, rainfall duration and disease incidence were the most important for the proportion of bait plants infected, followed by wind run. A four variable model relating the mean number of lesions to the rainfall rate, wind run, maximum temperature and either rainfall the previous week or disease incidence in the surrounding crop was considered to be the most useful for use in simulation studies.

Introduction

Bacterial blight, caused by *Pseudomonas syringae* pv. *pisi* (*Psp*) is a potentially serious seedborne disease of peas (*Pisum sativum*). Although severe losses from the disease are more frequently reported in winter-sown crops than in spring-sown crops, small, but unrecognised losses occur regularly in spring sown crops (Roberts, 1993; Roberts et al., 1995).

Transmission of disease from seed to seedling is dependent mainly on pathogen numbers on the seed and soil moisture during germination and emergence (Hollaway et al., 1996; Roberts, 1992; Roberts et al., 1996). The development of the disease in the field and subsequent effects on yield and crop quality are highly dependent on weather conditions and more specifically on the timing of weather conditions suitable for disease development in relation to crop developmental stage (Roberts, 1993b). A series of field trials was carried out at HRI-Wellesbourne and four other sites in the UK to examine the relationships among seed infection, development of disease in the field and yield of combining (dry-harvested) peas (Roberts et al., 1995). This paper reports additional work at Wellesbourne in which bait (or trap) plants were placed in selected plots to identify infection events and characterise the weather conditions resulting in local dissemination of and infection with the pathogen. The data obtained were used to develop probabilistic mathematical models to indicate the likelihood of disease for a given set of weather conditions.

Materials and methods

Field trials

Precise details of the field trials (Table 1) have been reported earlier (Roberts et al., 1995). Essentially, the trials consisted of large plots (16 × 12 m) sown with seed of pea cv Solara with different levels of infection with *Psp* Race 2 and separated by a barrier crop of cv

Consort which is resistant to Race 2. Bait plants were placed in one of the plots sown with the highest levels of seed infection. In any particular field trial, bait plants were always placed in the same plot for each exposure date.

Plant raising and placement

Seed was sown at regular intervals to ensure that the bait plants were all of a similar (developmental) age and size when exposed in the field plots. Four seeds of cv Solara were sown in a 10 cm square pot of Fisons Levington M2 compost and raised either in a glasshouse or cold frame, depending on the time of year. For each exposure in the field, six pots (i.e 24 plants) were spaced out evenly, approximately 4 m apart and at least 2 m from the edge of an infected field plot, and left for 24 h beginning at approximately 0900. Pots were sunk into the soil so that the rim of the pot was level with the surrounding soil. Following recovery from the field, bait plants were maintained in a glasshouse at ambient humidity, with a minimum temperature of 13 °C and vents opening at 18 °C. Water was applied only to the compost, to avoid wetting the leaves. Bait plants were placed in the same position in the plot on each occasion. For reasons of economy, bait plants were not placed in plots every day, but an attempt was made, based on the general weather forecast for the area to expose plants to the full range of conditions, and in particular during rainfall events. For example, if the weather was predicted to be continuously hot and sunny for an entire week, plants were exposed on only one or two days.

Weather records

Wherever possible rainfall and leaf wetness data were obtained from electronically-recorded, half-hourly data collected in fields adjacent to the experiments or when these data were not available from daily farm records (an official UK-meterological office weather station) (Anon. 1982) at HRI-Wellesbourne. Wind run and temperature data were obtained from the farm records. The validity of the electronically collected data was checked by comparison with records from the farm meterological station.

Disease records

Symptoms of bacterial blight were recorded on bait plants 3–4 weeks after exposure in the field. The number of plants with symptoms of pea bacterial blight was recorded for each pot and, if symptoms were observed, the number of lesions on each plant. Lesions resulting from bird, weevil or mechanical damage were recorded separately. Where a leaf was completely killed by disease the number of lesions was assigned a value of 20, as this was the maximum number which can be recorded on a single leaf. The presence of the pathogen was confirmed in representative lesions by comminuting in a drop of sterile tap water and streaking the resulting suspension on plates of King's medium B. The identity of typical *Psp* colonies was confirmed by a serological agglutination test (Lyons and Taylor, 1990) and by pathogenicity to pea seedlings, cv. Kelvedon Wonder (Taylor et al., 1989)

Disease incidence and severity in the field plots were recorded as part of the trials themselves following standard protocols (Roberts et al., 1995). These disease records were made on only four occasions during the growing season and therefore linear interpolation was done, using the INTERPOLATE directive of Genstat (Payne et al., 1993), to obtain values for days in between the recording dates.

Statistical analysis

Relationships between variables (defined in Table 2) were investigated using the general linear modelling procedures of Genstat (Payne et al., 1993). To examine the fit of the models, predicted values and adjusted data (partial residuals) were plotted against each variable in the model. Predicted values were calculated for the means of all the other variables in the model and adjusted data were calculated by adding residuals for each datum to the predicted values.

Results

Disease was not observed on control plants which had not been exposed as baits in the field. Disease was recorded on bait plants on 55 out of a total of 105 days on which plants were exposed. Nearly all of these infection events (53) were associated with the occurrence of rain. On the two occasions when infection was recorded in the absence of rain, it was directly associated with either bird or weevil damage. There were a total of seven occasions when infection was recorded only in association with damage. The disease and weather data are summarised in Table 2 (grouped according to infection in the absence of damage).

Table 1. Field trials at HRI-Wellesbourne used for exposure of bait plants

Sowing date	Harvest date	First bait plant exposure	Last bait plant exposure	Seed infection
20 Nov 1989	27 Jul 1990	09 May 1990	25 Jun 1990	40%
06 Mar 1990	23 Jul 1990	26 Jun 1990	06 Jul 1990	40%
06 Nov 1990	02 Aug 1991	28 Jan 1991	31 Jul 1991	1.25%
27 Feb 1992	24 Jul 1992	11 May 1992	06 Jul 1992	25%

Table 2. Weather and disease data for exposure of bait plants on 105 days. Summarised for (a) days on which disease was recorded on bait plants in the absence of bird or weevil damage and (b) days on which disease was not recorded in the absence of bird or weevil damage

	(a) Infection days. N=48			(b) Non-infection days. N=57		
	Mean	Min	Max	Mean	Min	Max
Plants infected	12.9	1	24	0	0	0
Lesions/plant (*m*)[1]	13.8	0.05	278	0	0	0
Rainfall[2] (*rain*)	6.8	0.1	49.4	1.21	0	12.4
Duration (*dur*)	5.8	1	18	1.9	0	15
Mean rate (*mrate*)	0.9	0.1	4.1	0.39	0	3
Max rate (*xrate*)	1.8	0.1	14.2	0.6	0	6
Wet[3] (*wet*)	13.3	0	24	6.9	0	17.5
RPD (*rpd*)	4.2	0	38.4	1.7	0	35.6
RPW (*rpw*)	16.1	0	79.6	12.2	0	79.6
DSR	1	0	19	1.8	0	12
Wind run (*wind*)	273	80	573	220	78	518
Max temp (*maxt*)	17.4	7.7	28.8	15.2	1.1	29.2
Min temp	8.7	–1.4	15.6	6	–1.1	15.5
Mean temp	13.1	4	19.7	10.6	0.8	23.5
Pinf (*pinf*)	0.9	0.1	1	0.7	0	1
Score	0.75	0.15	1.72	0.88	0	2.16
ΣScore	82	3.2	180.5	88.4	0	180.5
VGS	11.9	3.5	19	9.9	3	19
RGS	4.3	0	9.6	3.1	0	9.7

[1] Symbols used in equations are given in parentheses.
[2] Key and units for variables: Rainfall – amount of rain (mm); Duration – duration of rainfall (h); rate – rainfall rate (mm h^{-1}); Wet – duration of leaf wetness (h); RPD – rain previous day (mm); RPW – rain previous week (mm); DSR – days since rain (d); Wind run – (km); Pinf – disease incidence in surrounding crop; Score – disease score in surrounding crop (0–4 scale); ΣScore – cumulative disease score in surrounding crop; VGS – vegetative growth stage; RGS – reproductive growth stage.
[3] 41 and 43 values for infection and non-infection days respectively.

Modelling

For the purpose of modelling the effect of weather conditions on disease in the bait plants, plants exposed on the seven occasions when disease was associated only with bird or weevil damage were considered not to be infected and a value of zero assigned to the number of plants infected and the mean number of lesions. All the variables summarised in Table 2 were examined.

Fitting of models was problematical due to the high level of inter-correlation between variables (Table 3). Thus, variables measuring different aspects of the same type of weather (e.g. rainfall, duration, rate) tended to be mutually exclusive. Other variables which generally increased with time during the growing season such as growth stage of the surrounding crop, temperature, disease incidence and cumulative disease score for the surrounding plot were also highly correlated.

Table 3. Within-group (i.e. after correction for rain days, by inclusion of a factor for the occurence of rain) correlations between variables, for key see Table 2. 103 df

Plants inf.	1	1																		
Lesions	2	0.39	1																	
Rainfall	3	0.49	0.31	1																
Duration	4	0.54	0.21	0.71	1															
Mean Rate	5	0.46	0.33	0.77	0.46	1														
Max Rate	6	0.44	0.20	0.85	0.52	0.89	1													
Wetness	7	0.45	0.25	0.44	0.69	0.38	0.391													
RPD	8	0.27	0.09	0.13	0.15	0.19	0.19	0.241												
RPW	9	0.08	–0.02	–0.06	0.01	–0.12	–0.07	0.03	0.34	1										
DSR	10	–0.14	–0.05	–0.02	–0.15	–0.12	–0.08	–0.29	–0.22	–0.25	1									
Wind run	11	0.22	0.31	–0.02	0.16	0.01	–0.09	0.24	0.02	–0.10	–0.26	1								
Max temp	12	0.18	–0.14	0.04	–0.09	0.23	0.18	0.02	0.07	0.11	–0.10	–0.12	1							
Min temp	13	0.21	–0.08	0.22	0.05	0.26	0.26	0.11	0.24	0.26	–0.16	–0.12	0.69	1						
Mean temp	14	0.20	–0.12	0.14	–0.03	0.27	0.23	0.07	0.15	0.18	–0.13	–0.12	0.94	0.90	1					
Pinf	15	0.27	–0.02	0.13	0.03	0.25	0.22	–0.00	0.11	0.17	–0.20	–0.08	0.70	0.51	0.66	1				
Score	16	–0.10	0.10	–0.08	–0.03	–0.03	–0.09	–0.10	–0.05	–0.14	–0.17	0.06	–0.03	–0.22	–0.13	0.41	1			
ΣScore	17	–0.10	–0.14	–0.01	–0.06	0.17	0.10	0.02	0.07	–0.17	–0.12	–0.03	0.45	0.29	0.42	0.66	0.39	1		
VGS	18	0.21	–0.10	0.14	0.01	0.27	0.25	0.08	0.17	0.16	–0.11	–0.19	0.80	0.65	0.80	0.85	0.01	0.66	1	
RGS	19	0.18	–0.15	0.13	0.01	0.26	0.23	0.08	0.17	0.11	–0.02	–0.19	0.76	0.65	0.78	0.67	–0.27	0.57	0.93	1
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19

As it was clear that disease only occurred on rain days, a factor for the occurrence of rain was incorporated into all models. Twenty missing values in leaf wetness duration were replaced by the duration of rainfall multiplied by 5.8 (the mean ratio of leaf wetness to rainfall duration), except when this value exceeded 24 (the maximum possible number of hours of leaf wetness) when they were replaced with 24.

Mean number of lesions. A series of generalized linear models with Poisson error and logarithmic link functions (Payne et al., 1993), of the form:

$$\ln(m) = rainday + bx_1 + cx_2 \ldots \quad (1)$$

where *m* is the mean number of lesions per plant, *rainday* is a constant for the occurrence of rain, *b, c* ... are constants and x_1, x_2 ... are candidate explanatory variables, was fitted to the data. A summary of the best one, two, three and four variable models is shown in Table 4. Wind run and various measures of rainfall were the most important single variables, but a considerable reduction in residual deviance was obtained by fitting a two variable model. Wind run and mean rainfall rate were the most important explanatory variables, the inclusion of other variables was more problematical and dependant on the other variables already in the model. Including more than four explanatory variables in the model did not give significant improvements (P=0.001, based on an approximate F test of the deviance ratios). Models 10, 13, 14, 15 all appear to provide adequate descriptions of the relationship between the mean number of lesions per plant and the various variables, and it is difficult to propose a single best model, however the fit of model 13 is demonstrated in Figure 1, as a series of graphs showing the fitted values and adjusted residuals for each variable at the mean of the other variables.

Proportion of plants infected. A series of generalized linear models with binomial errors and a complementary log-log link function (Payne et al., 1993), of the form:

$$\ln[-\ln(1-p)] = rainday + bx_1 + cx_2 + \ldots \quad (2)$$

where *p* is the proportion of plants infected, *rainday* is a constant for the occurrence of rain, *b, c* ... are constants and x_1, x_2 ... are candidate explanatory variables, was fitted to the data. A summary of the best one, two and three, variable models is shown in Table 5. Measures of rainfall were the most important single variables, but, as they were highly correlated, tended to be mutually exclusive in the models. Subsequent variables were dependant on the choice of the first variable. Including more than two explanatory variables in the model did not give significant improvements (P=0.001, based on F test of the deviance ratios). Model 6 would appear to provide the best description of the relationship between the proportion of plants infected and the various explanatory variables. The fit of this model is

Table 4. Summary of the best models for the relationship between the mean number of lesions, *m*, per bait plant and weather variables. A factor (*rainday*) for the occurrence rain was fitted to all models first (Model 0)

Model no.	Model: ln(*m*) =	Residual mean deviance	df
0	+2.1	29.0	103
1	–1.0 + 0.0092*wind* [1]	19.9	102
2	+0.98 + 0.90*mrate*	22.2	102
3	–0.69 + 0.17*wet*	22.4	102
4	+4.7 – 0.18*maxt*	24.5	102
5	+1.6 + 0.058*rain*	24.6	102
6	–2.3 + 0.0086*wind* + 1.2*mrate*	10.9	101
7	–2.0 + 0.010*wind* + 0.078*rain*	13.3	101
8	–3.0 + 0.010*wind* + 0.43*xrate*	13.4	101
9	–2.0 + 0.0068*wind* + 0.11*wet*	16.9	101
10	+0.76 + 0.0062*wind* + 1.2*mrate* – 0.15*maxt*	9.1	100
11	–3.0 + 0.0093*wind* + 1.1*mrate* + 0.089*dur*	9.7	100
12	–2.8 + 0.0072*wind* + 1.0*mrate* + 0.078*wet*	9.9	100
13	+0.13 + 0.0069*wind* + 1.3*mrate* – 0.16*maxt* + 0.032*rpw*	8.3	99
14	–0.25 + 0.0070*wind* + 1.1*mrate* – 0.20*maxt* + 2.0*pinf*	8.4	99
15	+0.37 + 0.0067*wind* + 1.2*mrate* – 0.15*maxt* + 0.039*rpd*	8.5	99

[1]Key to variables: *wind* – wind run (km), *mrate* – mean rainfall rate (mm h^{-1}), *wet* – duration of leaf wetness (h), *maxt* – maximum temperature (°C), *rain* – rainfall (mm), *xrate* – maximum rainfall rate (mm h^{-1}), *dur* – duration of rainfall (h), *rpw* – rain the previous week (mm), *pinf* – disease incidence in surrounding crop, *rpd* – rainfall the previous day (mm).

demonstrated in Figure 2, as a series of graphs showing the fitted values and adjusted residuals for each variable at the mean of the other variables.

Discussion

These results clearly demonstrate the importance of wind and rain for the local dispersal and infection process of pea bacterial blight. In the absence of rainfall, spread and infection did not occur except when associated with mechanical transmission by birds or insects. In gentle rain and light winds local spread and infection was rare, but in heavy rain and high wind it was frequent. Other variables were less important and their role not so clear due to inevitable correlations between many of them, nevertheless, it does appear that maximum temperature, rainfall in the previous week, rainfall duration and disease incidence in the surrounding crop also have some influence. It should be noted that as the occurrence of rain was the most important factor determining the occurrence of disease in the bait plants, this was fitted in all models, and hence the effects of the variables in the models are effectively estimated for days on which it rained.

A priori, it was expected that both the mean number of lesions and the proportion of infected plants would have a similar relationship with the explanatory variables: if the lesions were randomly scattered over the plants exposed, the mean number of lesions per plant, *m*, should be equivalent to –ln(1–*p*) or ln[1/(1–*p*)] (Roberts et al., 1996), which follows from the probability of a plant being infected being the same as the probability of it having one or more lesions. However, in these data, the relationship differed. This might be partly explained by aggregation of lesions: a plot of the ln(*variance*) v ln(*mean*) relationship for the mean number of lesions on plants each day had a slope of 1.45 and an intercept of 1.40, indicating aggregation (Taylor, 1961). Effectively this means that once a plant has received a hit, further hits are more likely. This could result from directional movement of the

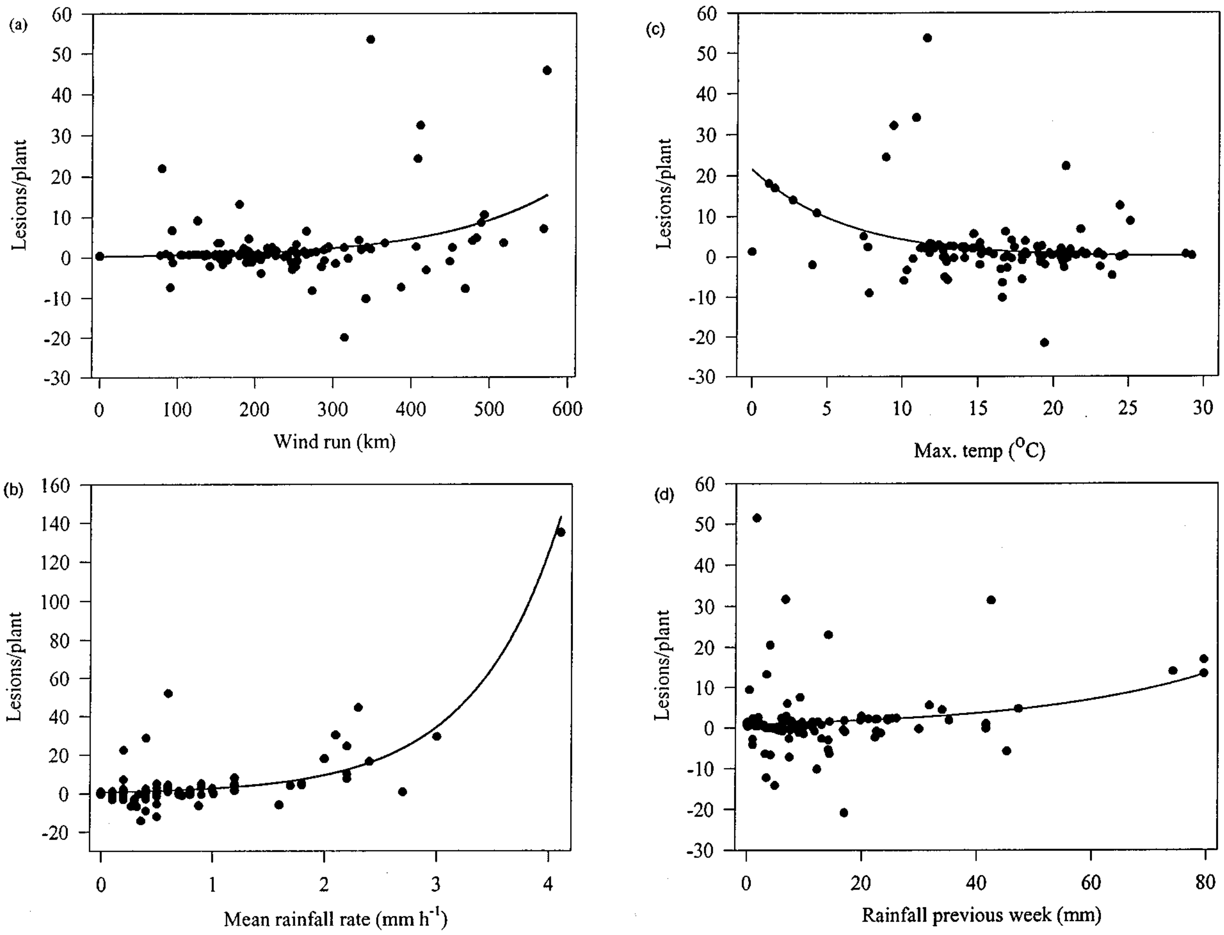

Figure 1. Relationship between the mean number of lesions per plant, *m*, and (a) wind run, (b) rainfall rate, (c) maximum temperature and (d) rainfall in the previous week for bait plants exposed for 24 h periods in a pea crop infected with pea bacterial blight. The data points are partial residuals and the solid line represents predicted values at the mean of the other variables in the model on rain days. Note that the left hand scales differ as a result of the values of the means of the other variables in the model. Model 13 (Table 4): ln(m) = 0.13 + 0.0069 *wind* + 1.3 *mrate* – 0.16 *maxt* + 0.032 *rpw*. Mean values: *wind* = 245, *mrate* = 0.063, *maxt* = 16.3, *rpw* = 14.0.

pathogen on any particular 'infection day' and would seem highly likely given the importance of wind in the lesion model. It could also result from positional effects due to non-random distribution of inoculum in the plot surrounding the bait plants. Examination of the direct relationship between m and $-\ln(1-p)$ indicated that although on average the mean number of lesions was 2.3 times that suggested by $-\ln(1-p)$, the ratio of m to $-\ln(1-p)$ tended to increase as p increased (Figure 3). This is probably an artifact of the limited number of plants exposed on each occasion, i.e. the maximum number of plants which could be infected was 24 and hence the value of p can only take discrete values at intervals of 1/24th. Thus the relationship between p and the weather variables (Table 5) may be more an artifact of the data set than real, and the lesion model(s) (Table 4) may be more generally useful in terms of simulating/predicting epidemics.

It might be expected that the amount of disease on the bait plants would depend on: (i) the amount of inoculum in the surrounding crop; (ii) the availability of inoculum or proportion available (e.g. presence on the surface of the plants rather than inside); (iii) the spread/transfer of inoculum onto bait plants; (iv) the infection process itself, i.e. ingress of bacteria into the tissues of the bait plants (at sites suitable for multiplication). Rainfall rate and wind exert most influence on (iii) and (iv), i.e. in generating splash droplets and aerosols, assisting in their movement from plant to plant, promoting mechanical damage to plants

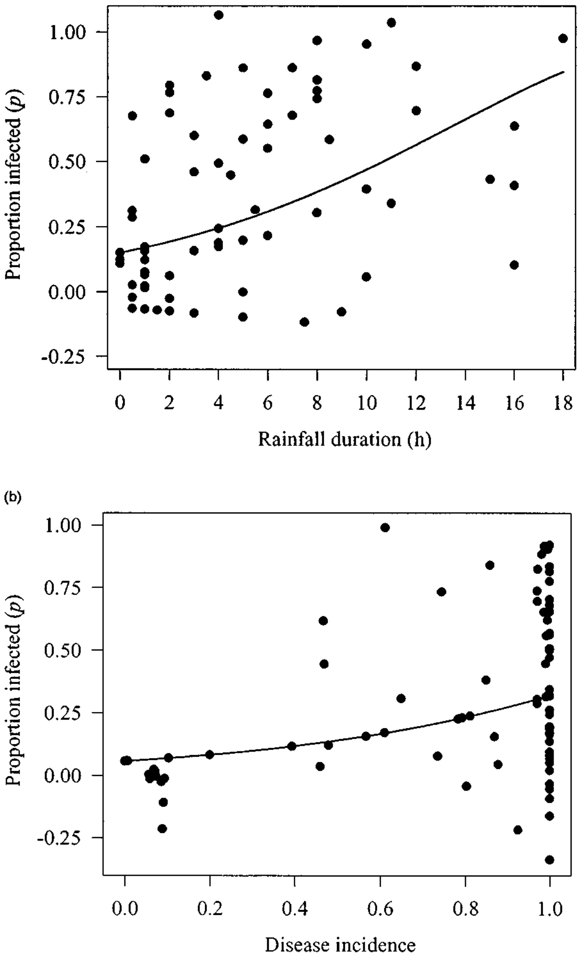

Figure 2. Relationship between the proportion of plants infected, *p*, and (a) rainfall duration, (b) disease incidence in the crop for bait plants exposed for 24 h periods in a pea crop infected with pea bacterial blight. The data points are partial residuals and the solid line represents predicted values at the mean of the other variable in the model on rain days. Note that the left hand scales differ as a result of the values of the means of the other variable in the model. Model 6 (Table 5): $\ln[-\ln(1-p)] = -3.3 + 0.14\,dur + 1.9\,pinf$. Mean values: $dur = 3.6$, $pinf = 0.80$.

Table 5. Summary of the best models for the relationship between the proportion of bait plants infected, *p*, and various explanatory variables. A factor (*rainday*) for the occurrence rain was fitted to all models first (Model 0)

Model no.	Model: $\ln[-\ln(1-p)]$ =	Residual mean deviance	df
0	–0.96	14.7	103
1	$-1.6 + 0.12dur$[1]	12.4	102
2	$-1.3 + 0.059rain$	12.5	102
3	$-1.3 + 0.24xrate$	12.9	102
4	$-1.4 + 0.59mrate$	13.0	102
5	$-1.8 + 0.066wet$	13.1	102
6	$-3.3 + 0.14dur + 1.9pinf$	11.1	101
7	$-3.2 + 0.14dur + 0.087maxt$	11.6	101
8	$-1.9 + 0.10dur + 0.44mrate$	11.7	101
9	$-1.7 + 0.12dur + 0.038rpd$	12.0	101
10	$-4.6 + 0.14dur + 2.4pinf + 0.0033wind$	10.5	100

[1] Key to variables: *dur* – duration of rainfall (h), *rain* – rainfall (mm), *xrate* – maximum rainfall rate (mm h^{-1}), *mrate* – mean rainfall rate (mm h^{-1}), *wet* – duration of leaf wetness (h), *pinf* – disease incidence in surrounding crop, *maxt* – maximum temperature (°C), *rpd* – rainfall the previous day (mm), *wind* – wind run (km).

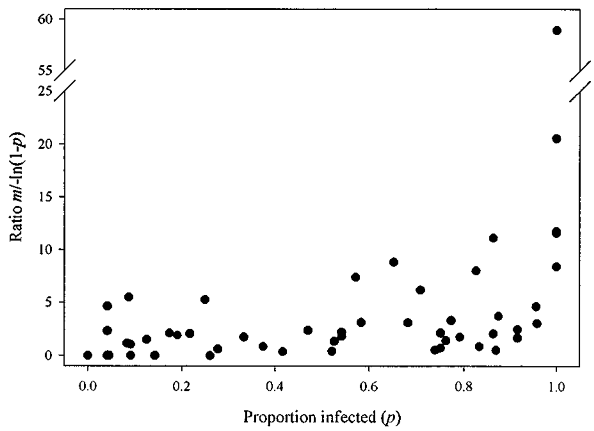

Figure 3. Relationship between the ratio of the observed mean number of lesions per plant, *m*, to the value predicted by the proportion of infected plants, $-\ln(1-p)$, and the proportion infected, *p*.

and assisting ingress through natural openings. It is clear from the relative importance of different variables in the models that, in the experiments reported here, transport and infection processes were more important than the amount or availability of inoculum. This is perhaps not surprising, as disease incidence in the plots surrounding the bait plants was generally close to 100%. In the field crop situation, however, where primary infections in a crop result from seed transmission, the amount and availability of inoculum may be much more important than in these experiments.

Splash dispersal is dependant on droplet size, which is related to rainfall rate, but not always in a consistent way (Walklate, 1989; Walklate et al., 1989). Thus, some improvements to the models may have been possible if splash intensity had been measured directly (rather than indirectly as rainfall rate). However, instruments for measuring the splash intensity of rain are not readily available.

The negative relationship with temperature (Table 4: Models 4, 10, 13, 14) may be an artefact, due to its correlation with a number of other variables, e.g. crop growth stage, dry weather. However, it may be a direct effect, as there is evidence for other diseases that infection is inhibited at high temperatures (Smitley and McCarter, 1982; Roberts, 1985).

In contrast to the results reported here, Hirano & Upper (1990) suggest that the importance of rain in the epidemiology of bacterial diseases is not in splash dispersal, but in triggering the multiplication of epiphytic pathogen populations; it is the subsequent high epiphytic populations which result in the appearance of disease (Hirano and Upper, 1983; Rouse et al., 1985; Hirano and Upper, 1990). In much of their work, however, there appears to have been no differentiation between pathogen populations which are washed from the leaf surface and those which may have been released from invisible lesions within the tissues. Moreover, several studies have shown that significant numbers ($>10^6$) of pathogenic bacteria can be released from leaf lesions by as little as 5 min gentle washing long before the appearance of visible symptoms (Leben et al., 1968; Haas and Rotem, 1976; Miles et al., 1977; Roberts, 1985).

During rain, there is a net removal of bacteria from leaf surfaces (sometimes $>90\%$,) and most of these bacteria are washed downwards, with only a small proportion (1.5%), becoming airborne in splash droplets (Butterworth and McCartney, 1991). This proportion may seem relatively small, but it is a small proportion of a very large number in the case of a diseased leaf which may have more than 10^9 pathogen cells in a single lesion (and perhaps several lesions), compared to the maxima of 10^4 to 10^6 cells/leaf or plant generally observed for epiphytic populations in the absence of symptoms (Weller and Saettler, 1980; Hirano and Upper, 1983; Grondeau et al., 1996; Hirano and Upper, 1993). As the bacteria within lesions are released very readily from wet leaves (Leben et al., 1968; Haas and Rotem, 1976; Miles et al., 1977; Roberts, 1985) the potential number of cells available for dispersal from a single spot on a single infected leaf may be greater than the entire epiphytic population on the surfaces of several thousand leaves or plants. Hence, despite the suggestion of Hirano and Upper (1990) that splash dispersal is not important in the development of epidemics of bacterial diseases, the numbers of airborne pathogenic bacteria near an infected crop are much higher during or immediately after rain than during dry weather (Graham and Harrison, 1975; Graham et al., 1977; Kuan et al., 1986). Models have also been proposed suggesting that pathogenic bacteria could be deposited over 1000 m downwind of an infected crop as a result of aerosols produced by rain impaction (Graham and Harrison, 1975; Graham et al., 1977). In the case of pea bacterial blight splash dispersal and simultaneous infection play a major role in the epidemiology of the disease.

The results presented here confirm the results of observations and experiments on several other bacterial plant diseases (Faulwetter, 1917; Faulwetter, 1919; Walker and Patel, 1964; Daft and Leben, 1972; Gottwald et al., 1988) that wet and windy conditions are the most favourable for local plant to plant spread and infection. However, this work goes further by defining more precisely the relationship between the amount or probability of infection and easily-measured weather variables, which could form the basis of simulation models to examine disease development under different climatic scenarios.

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