Eur J Plant Pathol (2007) 119:87–97
DOI 10.1007/s10658-007-9163-3

FULL RESEARCH PAPER

Assessment of airborne primary inoculum availability and modelling of disease onset of ascochyta blight in field peas

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Received: 7 November 2006 / Accepted: 3 May 2007 / Published online: 5 June 2007

Abstract Ascochyta blight is a serious disease affecting field peas. In France, disease management relies mainly on scheduled chemical applications without taking into account the actual disease risk. A better understanding of the factors affecting disease onset would therefore help in the timing of the first application. Field experiments involving eight sowing dates between mid-September and mid-December were conducted for two consecutive years. The seasonal dynamics of airborne inoculum were investigated through trap plants. The weekly availability of airborne primary inoculum was extremely low during autumn and winter and was partially influenced by mesoclimatic conditions. Disease onset occurred between mid-October and early March depending on the sowing date. Generally, the later the sowing date, the longer the period between sowing and disease onset. This was due to an increase in the period between sowing and emergence. Disease onset was observed 14–35 days after emergence. A disease onset model based on the calculation of weather-dependent daily infection values (*DIVs*) was established, assuming that disease onset occurs once the temperature and moisture requirements for incubation are met. Cumulative daily infection values (*cDIVs*) were determined by sowing date and experiment through addition of consecutive *DIVs* between emergence and disease onset. A frequency analysis of *cDIVs* was performed to determine the 10th and 90th percentiles of the distribution. An analysis of the observed and predicted values showed that observed disease onset dates were almost always included in the forecast window defined by these two percentiles. This study is the first attempt to predict ascochyta blight onset in field peas and should contribute to development of a more rational fungicide application strategy.

Keywords Ascospores · Disease forecast model · Mesoclimate · Relative humidity · Temperature · Trap plants

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Introduction

Ascochyta blight is a serious disease affecting field peas in most pea-growing regions of the world, particularly in the temperate areas of Europe, North America, Australia, and New Zealand (Bretag and Ramsey 2001). The disease, mainly caused by

Mycosphaerella pinodes, infects all aerial organs of the plant (leaves, stems, flowers, pods) and can cause yield losses of up to 75% when conditions are favourable for an epidemic (Lawyer 1984). The disease affects yield either indirectly through reduction of biomass production (Béasse et al. 2000; Garry et al. 1998; Le May et al. 2005), or directly through pod infection (Béasse et al. 1999). The relative importance of these two effects depends on the location of the symptoms on the plant and therefore on the precocity and intensity of the epidemic. In a context of poor plant resistance, as is the case for the ascochyta blight/pea pathosystem, epidemic precocity depends mainly on inoculum availability and weather conditions.

As a polycyclic disease, ascochyta blight is initiated by primary inocula and develops by means of secondary inocula. Primary inoculum sources are numerous: infected seeds, soil, infected pea stubble, volunteer plants or legume weeds. Airborne inoculum is the most important source of primary inoculum in established pea-growing areas (Bretag and Ramsey 2001). It is mainly constituted by ascospores of *M. pinodes*, produced in pseudothecia on infected pea stubble left on the soil surface after harvest, and dispersed by wind for over 1.6 km (Lawyer 1984). Salam and Galloway (2005) developed a weather-based model to predict the seasonal release of ascospores of *M. pinodes* in Western Australia to help farmers decide when is the best time to sow their pea crops in order to avoid periods of ascospore release peaks. However, this model is limited to areas where infected pea stubble is left on the soil surface and where this primary inoculum source is obvious. In France, where pea stubble is usually removed before ploughing, little is known about the availability of airborne primary inoculum and it is therefore difficult to predict disease onset. As a consequence, disease management relies mainly on scheduled chemical applications without taking into account disease risks. For spring peas (sown between mid-February and mid-April), this fungicide application strategy is based on a first application at the beginning of flowering (BF), followed by a second application at BF + 15 days, and possibly by a third application at BF + 25 days. For winter peas (sown from mid-October to mid-January), extension services can provide little information to farmers and recommend an ''early'' application ''in case of early first symptoms'' without further precision (ARVALIS 2003). A better understanding of primary inoculum availability and a prediction of disease onset would therefore be particularly useful to position the first application.

The objectives of this study were (i) to characterise the pattern of airborne inoculum availability, (ii) to investigate the relationship between airborne primary inoculum availability and mesoclimate, (iii) to study the impact of sowing dates on disease onset, and (iv) to propose a predictive model of disease onset.

Materials and methods

Field experiments

Experiments were conducted during the 2003–2004 and 2004–2005 cropping seasons in Le Rheu (48°06′00″N, 1°48′00″W, 30 m above mean sea level), western France. The two adjacent fields chosen for these trials were: (i) similar in pedoclimatic environments and (ii) void of soilborne inoculum due to a rotation without pea crops during the previous five years. The experimental design consisted of eight 30 m^2 (3 m wide × 10 m long) plots sown with winter field peas (cv. Cheyenne, 80 seeds m^{-2}) at two-week intervals from mid-September to mid-December (Table 1 and Fig. 1). Cheyenne is susceptible to ascochyta blight and is currently the most cultivated winter field pea cultivar in France. Plots were sown perpendicularly to the prevailing wind direction (N-NW), the first plot being sown in the downwind. A 3 m wide buffer strip of bare soil was maintained between each plot to prevent cross-contamination of plots due to rain splash dispersal of inoculum.

Trap plants

Inoculum availability was assessed indirectly through trap plants. Each week from mid-September to mid-March (2003–2004) or mid-May (2004–2005), trays containing 20 trap plants (5-leaf Cheyenne pea seedlings) were placed at 1 m from the four corners of the trial (Fig. 1). After seven days of exposure, trap plants were incubated in a dew chamber (12 h-photoperiod, 20°C night/day, 100% relative humidity (RH)) for four days. The amount of viable spores deposited on trap plants was estimated as the number

Table 1 Sowing and emergence dates in field trials conducted in France in 2003–2004 and 2004–2005

Year	Sowing number	Sowing date	Emergence date
2003–2004	S1	15/09/03	29/09/03
	S2	29/09/03	13/10/03
	S3	13/10/03	03/11/03
	S4	27/10/03	17/11/03
	S5	07/11/03	01/12/03
	S6	24/11/03	05/01/04
	S7	08/12/03	19/01/04
	S8	19/12/03	02/02/04
2004–2005	S1	15/09/04	27/09/04
	S2	27/09/04	11/10/04
	S3	11/10/04	25/10/04
	S4	25/10/04	15/11/04
	S5	08/11/04	06/12/04
	S6	22/11/04	03/01/05
	S7	06/12/04	10/01/05
	S8	20/12/04	17/01/05

of resulting lesions (small, purplish-black, irregular flecks) on the five lower stipules of the plants after incubation. A mean number of lesions per plant was calculated per tray and per week. Preliminary experiments based on the method proposed by Onfroy et al. (2007) confirmed that after four days of incubation the five lower stipules of a Cheyenne pea plant all exhibited the same susceptibility to ascochyta blight (data not shown).

Emergence and disease assessment

Emergence, defined as the stage when 50% of the plants had their first true leaf emerged, was dated in most of the cases through regular assessment of the plots (Table 1). If missed, the emergence date was estimated with an ad hoc quadratic relationship derived from the trial data:

$$y = -4.08x^2 + 57.36x + 97.49 \quad (R^2 = 0.91) \qquad (1)$$

where y is the emergence date in degree-days (from 0°C) since sowing and x is the sowing rank (from 1 to 8) (Fig. 2).

Disease was assessed at 1–2 week intervals on ten plants sampled at random in each plot. Disease onset was defined as when at least one lesion was observed on 50% of the assessed plants.

Weather data

An automatic weather station was set up near the trial plots. Air temperature and RH were measured with a temperature and humidity probe (HMP45AC, Vaisala, www.vaisala.com), precipitation was measured with a tipping bucket rain gauge (ARG100, Campbell Scientific Inc., www.campbellsci.com), and wind speed and direction were measured with a wind monitor (05103, RM Young, www.youngusa.com). Sensors were placed at 1.40–2.20 m above ground level. The data logger (CR10X, Campbell Scientific Inc.) scanned the sensors every 10 s and stored the 15-min averages.

Model conception

The ascochyta blight onset model is based on the calculation of weather-dependent daily infection values (*DIVs*). This method derives from initial work of Shane and Teng (1983) and further refined by Wolf

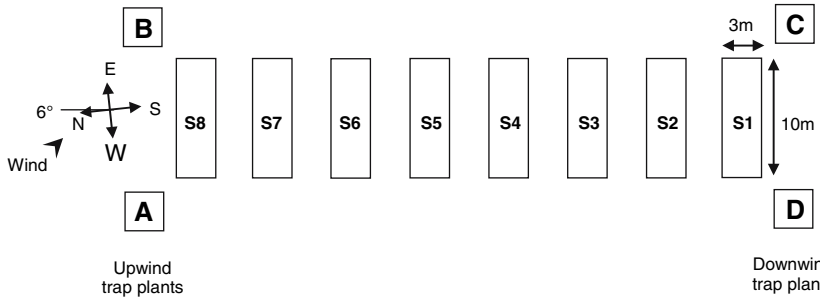

Fig. 1 Schematic representation of the experimental design used in field experiments conducted in France in 2003–2004 and 2004–2005. Eight pea plots were sown between mid-September (S1) and mid-December (S8). Trap plants were placed at 1 m from the field experiment. A and B are upwind trap plants. C and D are downwind trap plants. The arrow indicates the prevailing wind direction (N-NW)

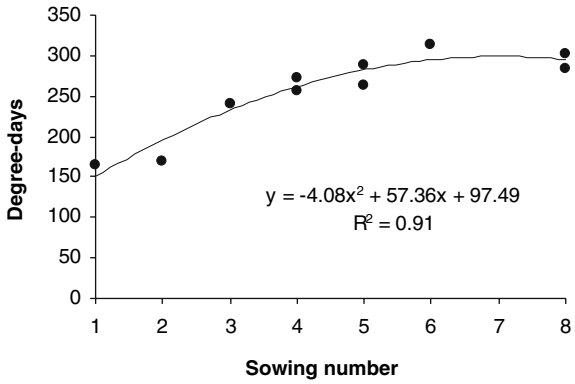

Fig. 2 A quadratic model predicting the time (expressed in degree-days from 0°C) required for emergence of the first leaf for sowing dates ranging between mid-September and mid-December in field trials conducted in France in 2003–2004 and 2004–2005

and Verreet (2005) on *Cercospora beticola* infection prediction. This modelling approach is suitable for any fungal epidemic initiated by airborne primary inocula. *DIVs* were calculated using hourly data from the automatic weather station. The effects of temperature and moisture on incubation period (time elapsed between infection and the appearance of the first symptom) were quantified through the calculation of two specific indices. *DIVs* were computed as the average of hourly values of the product of these two indices:

$$DIV_i = \frac{1}{24}\sum_{j=1}^{24} M_{ij} \times T_{ij} \in [0;1] \text{ (dimensionless)} \quad (2)$$

where DIV_i is the infection value for day i ranging between 0 (no fungal growth) and 1 (optimal growth), M_{ij} is the moisture index for day i and hour j, and T_{ij} is the temperature index for day i and hour j. M_{ij} and T_{ij} were based on published data obtained from artificial inoculation of pea plants under controlled conditions (Roger et al. 1999a, b).

M_{ij} is a binary variable that expressed the capacity of the environment to provide satisfactory moisture conditions for incubation. Roger et al. (1999a) showed that leaf wetness (free water on the leaf surface) or high RH was required for infection. From these results, M_{ij} was set to 1 when rainfall was ≥0.2 mm (resolution of the rain gauge) or when RH was greater than a threshold τ:

if rainfall ≥ 0.2 mm or

$$\text{RH} > \tau\ \%\ , M_{ij} = 1 \text{ (dimensionless)} \quad (3a)$$

$$\text{otherwise, } M_{ij} = 0 \text{ (dimensionless)} \quad (3b)$$

Rather than setting τ to an arbitrary level, we tested seven threshold values: 65, 70, 75, 80, 85, 90 and 95%. From the analysis of the performance of the corresponding models, the best threshold value was then determined (see below).

T_{ij} was expressed as the ratio between the length of the incubation period at optimal and at observed temperatures. We assumed that all infection processes (germination, appressorial formation, penetration) stopped at 0°C and thus set the T_{ij} value to zero when temperature was equal to or below 0°C. *Tij* was calculated as follows:

$$\text{if } t \leq 0°\text{C}, T_{ij} = 0 \text{ (dimensionless)} \quad (4a)$$

$$\text{if } t > 0°C,\ T_{ij} = \left(\frac{IP_{opt}}{IP_t}\right)_{ij} \in]0;1] \text{ (dimensionless)} \quad (4b)$$

where IP_{opt} is the length of the incubation period at optimum temperature and IP_t is the length of the incubation period at temperature t.

The shortest incubation period obtained at optimum temperature (15–25°C) is one day (Roger et al. 1999b). At temperatures less favourable to pathogen growth, IP_t is described by a quadratic function of temperature t and varies according to the moisture regime (leaf wet or not). In the absence of leaf wetness sensors or of leaf wetness simulation models, leaf surface was assumed to be wet when rainfall was ≥0.2 mm (resolution of the rain gauge):

$$IP_{opt} = 1 \text{ (in days)} \quad (5a)$$

if rainfall ≥ 0.2 mm,

$$IP_t = 0.0171t^2 - 0.6457t + 6.8 \text{ (in days)} \quad (5b)$$

$$\text{otherwise, } IP_t = 0.0307t^2 - 1.195t + 12.1 \text{ (in days)} \quad (5c)$$

Although the domain of validity of the equations proposed by Roger et al. (1999b) was 5–30°C, extrapolation of this model for temperatures in the

range of 0–5°C was based on results of Hare and Walker (1944). Using radial expansion as a criterion for measurement of the effect of temperature on the mycelial growth of *M. pinodes* on potato dextrose agar plates, these authors showed that 8 days after inoculation, there was no fungal growth at 0°C and 36°C; at 4°C, the diameter of the colony was 10 mm, compared to 60–70 mm at optimum temperature. They also assumed positive fungal growth between 0 and 4°C.

For each value of RH threshold τ, cumulative daily infection values (*cDIVs*) were determined for each sowing date by adding *DIVs* between emergence and disease onset:

$$cDIV = \sum_{i=1}^{n} DIV_i \tag{6}$$

where *cDIV* = cumulative daily infection value, i = ith day of the calculation period, and DIV_i = weather-dependent DIV for day i.

For each τ, a frequency analysis of *cDIV* values was performed to determine the 10th and 90th percentiles (respectively named *P10* and *P90*). These values define a climatic window that contains 80% of *cDIV* values. In probabilistic terms, this indicates that there is an 80% chance that disease onset will occur within the defined climatic window. For the seven values of τ tested and for each experimental situation (year × sowing date), we compared the observed disease onset date to the forecast window defined by an early date (corresponding to the *P10* value of *cDIV*) and a late date (corresponding to the *P90* value of *cDIV*). In cases where disease onset occurs outside the forecast window, it is more detrimental if it occurs before the forecast window than after. The deviations of observed from predicted values were computed separately for the cases where disease onset occurred before and after the forecast window:

if $DO_{obs} < DO_{P10}$,

$$\alpha = \sum_{k=1}^{n} (DO_{obs} - DO_{P10})_k \in Z^{-}(\text{in days}) \tag{7a}$$

if $DO_{obs} > DO_{P90}$,

$$\beta = \sum_{k=1}^{n} (DO_{obs} - DO_{P90})_k \in Z^{+}(\text{in days}) \tag{7b}$$

where DO_{obs} is the disease onset observed in experimental situation k (expressed in days since emergence), DO_{P10} and DO_{P90} are respectively the disease onset predicted at 10th and 90th percentiles of the *cDIV* distribution (expressed in days since emergence) and α and β quantify respectively the total prediction errors (in days) when disease onsets were either earlier or later than the predicted window. The value of τ that resulted in the lowest values of both α and β was chosen as the best RH threshold.

Statistical analyses

The effect of trap location (A, B, C or D) on weekly airborne inoculum availability was analysed with the GENMOD (generalized linear model) procedure of the SAS software package (SAS Institute Inc., Cary, NC) assuming a Poisson distribution of lesion counts and using the log link function. The CONTRAST statement was used to perform custom hypothesis tests such as the equality of the numbers of lesions per trap plant at upwind locations A and B.

The effect of mesoclimatic variables on airborne primary inoculum availability was investigated through multiple regressions by using the stepwise model-selection method of the REG procedure of SAS. The F statistic for a variable to be included and to stay in the model had to be significant at the 0.05 level. Simple residuals (predicted minus observed values) were tested for normality using the UNIVARIATE procedure (Shapiro-Wilk and Kolmogorov-Smirnov tests). The performance of the model was analysed using a regression approach (prediction versus observation). The estimated values of the slope and intercept of the fitted model ($y = ax + b$) were compared to those of the $y = x$ line (i.e., we tested whether the slope was equal to 1 and whether the intercept was equal to 0) using a t-test.

Results

Seasonal dynamics of airborne inoculum availability

Depending on field experiments, airborne inoculum availability was assessed indirectly using trap plants for 27 or 36 weeks (Fig. 3). For both experiments, the

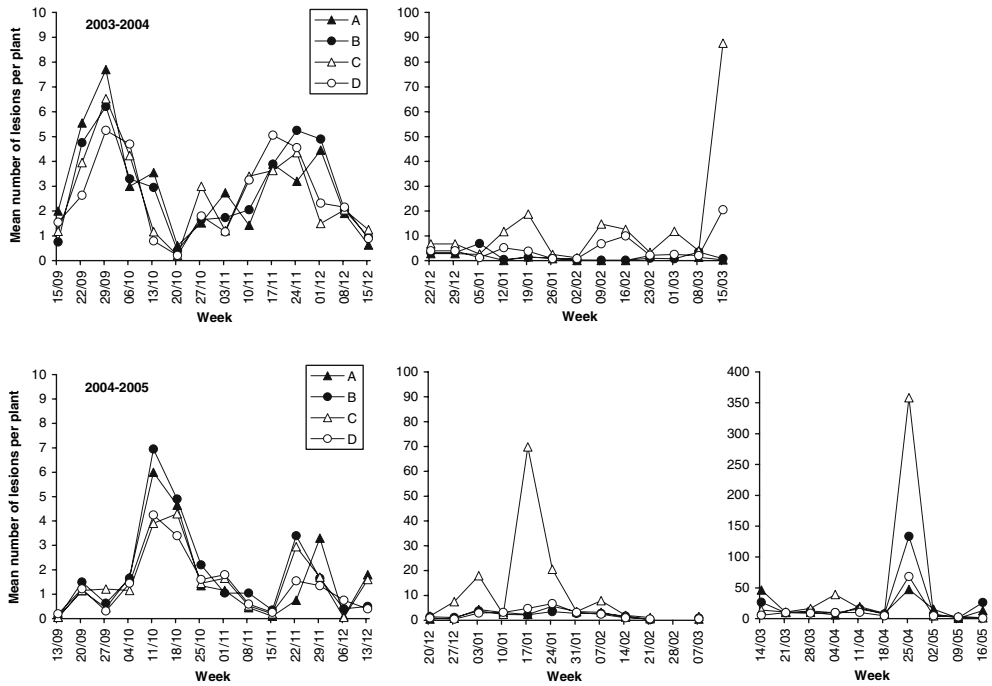

Fig. 3 Availability of aerial inoculum of ascochyta blight indirectly assessed on trap plants placed at the four corners of field experiments conducted in France in 2003–2004 and 2004–2005 cropping seasons. A and B are upwind trap plants. C and D are downwind trap plants

seasonal pattern showed two main phases. The first phase (mid-September to mid-December) corresponded to a period during which there were generally no significant differences between the four trap plant locations. During this phase, inoculum availability was extremely low and very few lesions (between 0 and 8 lesions per plant per week) were observed. The second phase (mid-December to early/mid-March) corresponded to a period during which downwind trap plants (mainly location C) trapped significantly more airborne inoculum than upwind trap plants (locations A and B). During this phase, up to 90 lesions per plant per week were observed on the downwind trap plants. Low levels of airborne inoculum continued to be observed on the upwind trap plants. The fact that the high levels of inoculum were all restricted to downwind trap plants suggested that the corresponding inoculum was probably a pycnidiospore-like secondary inoculum splashed at short distance from early-sown infected plants. In addition to these two phases, the 2004–2005 seasonal dynamic showed a third phase (mid-March to mid-May) during which high levels of airborne inoculum were detected on both the upwind and downwind trap plants. Up to 350 lesions per plant per week were observed in mid-April. The fact that high levels of inoculum were detected on downwind but also on upwind trap plants tends to support the hypothesis that this trapped inoculum was an ascospore-like secondary inoculum dispersed by wind at long distance from infected plants.

Relationship between airborne primary inoculum availability and mesoclimatic variables

Here we focused solely on the phases of the airborne inoculum availability dynamics corresponding to airborne primary inoculum. The levels of airborne inoculum availability assessed on upwind trap plants (locations A and B) from mid-September to mid-March were averaged to generate a new dependent variable, which was analysed using multiple regression methods. Variables derived from temperature (minimum, maximum, mean, sum of degree-days) and rainfall (sum, maximum, number of rainy days) were considered as potential explanatory variables. The best model selected by the stepwise method involved degree-days and maximum rainfall:

$$IP = 0.02198 \times DD + 0.11185 \times R_{max} (R^2 = 0.71) \quad (8a)$$

where *IP* is the level of primary inoculum expressed as lesions per plant and per week, *DD* is the sum of degree-days (from 0°C) calculated during the corresponding week and R_{max} is the maximum daily rainfall measured during the corresponding week.

Simple residuals (predicted minus observed values) were normally distributed according to the Kolmogorov-Smirnov test ($P > 0.15$) and near-normally distributed according to the Shapiro-Wilk test ($P = 0.048$). The histogram of residuals showed that two under-estimated values (−3.96 and −4.40) had a great impact on the distribution (Fig. 4). Removing the corresponding observed values from the analysis improved the normality of the distribution (Shapiro-Wilk test: $P = 0.372$) and only affected the regression equation slightly:

$$IP^* = 0.0192 \times DD + 0.1171 \times R_{max} (R^2 = 0.76) \quad (8b)$$

However, the prediction of the weekly airborne primary inoculum availability given by this model has to be considered with caution. Indeed, the slope of the prediction versus observation regression line was significantly <1 (estimate = 0.395, standard error = 0.072, $n = 50$) and the intercept significantly >0 (estimate = 1.176, standard error = 0.183, $n = 50$) (Fig. 5).

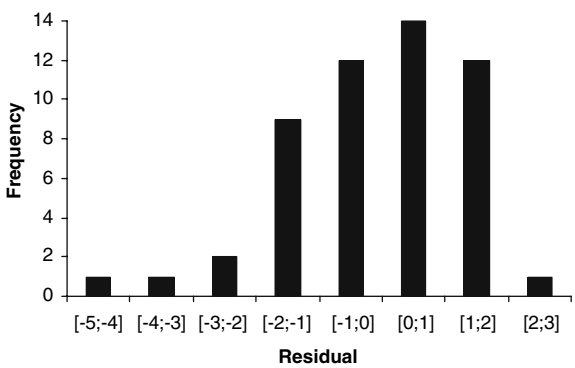

Fig. 4 Histogram of the simple residuals (predicted minus observed values) of the multiple regression established between airborne primary inoculum and mesoclimatic variables

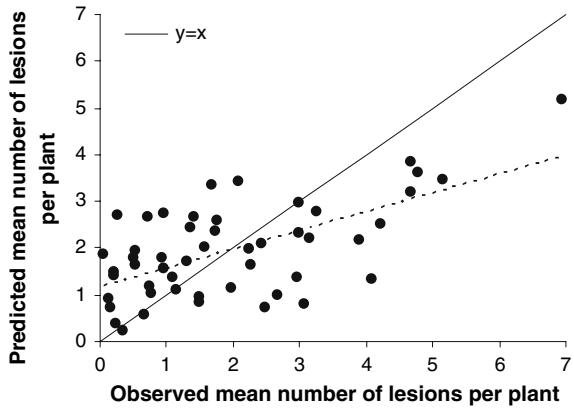

Fig. 5 Comparison between predicted and observed levels of airborne primary inoculum. Predicted values were calculated by the following equation: $IP^* = 0.0192 \times DD + 0.1171 \times R_{max}$ ($R^2 = 0.76$), where *IP** is the level of airborne primary inoculum expressed in lesions per plant and per week, *DD* is the sum of degree-days from 0°C, calculated during the corresponding week and R_{max} is the maximum rainfall measured during the corresponding week. The dotted line represents the prediction versus observation regression line

Relationship between disease onset and sowing date

Disease onset occurred between mid-October and early March (Fig. 6). Generally, the later the sowing date, the longer the period between sowing and disease onset. This was mainly explained by an increase in the duration of the period between sowing and emergence. Disease onset was observed 14–35 days after emergence. Pea plants had 2–5 leaves when disease onset occurred. The length of the period between emergence and disease onset (expressed in days after emergence, *DAE*) appeared to be independent of sowing date. For instance, short periods were observed for either early or late sowing dates. In addition, regression analyses indicated that little variability in *DAE* was accounted for by mesoclimatic (such as cumulative degree-days or rainfall during the period) and biological (such as cumulative primary inoculum during the period) variables. Only a weak simple linear regression linking cumulative degree-days to DAE was found:

$$DAE = 0.066 \times DD + 11.898 \ (R^2 = 0.36) \quad (9)$$

where *DD* is the cumulative degree-days (from 0°C) between emergence and disease onset.

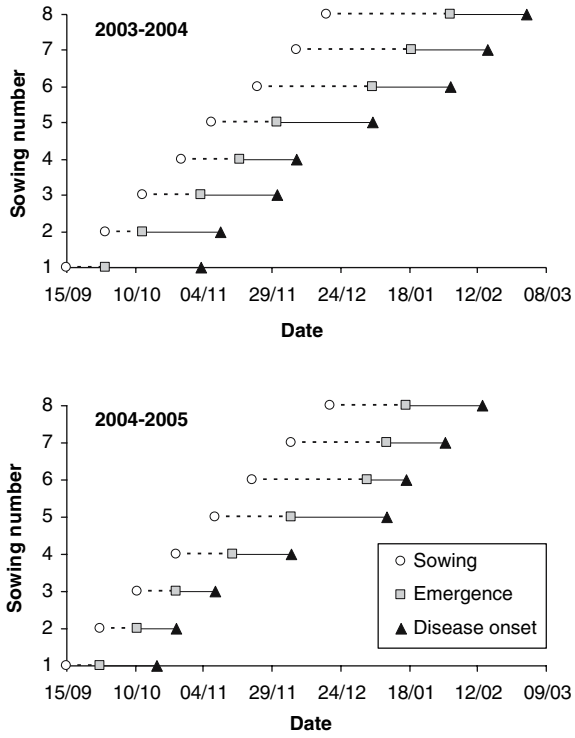

Fig. 6 Schematic representation of sowing, emergence and ascochyta blight onset dates in field trials conducted in France in 2003–2004 and 2004–2005

This model suggested that cumulative degree-days could partially explain the variability of *DAE* in the sense that the greater the accumulation of degree-days, the later the disease onset. In fact, considering the known positive effect of temperature on infection processes, this was highly improbable. This relation simply illustrated the fact that with late disease onsets (i.e., more days between emergence and disease onset), cumulative degree-days are inevitably greater than with early onsets. This showed the limitations and pitfalls of this approach and suggested that rather than trying to explain disease onset with measured values of mesoclimatic variables, we should consider effective values (effective in the fungal infectious process) to predict the occurrence of disease onset.

Weather-dependent modelling of disease onset

The hypothesis underlying this approach is that disease onset occurs once the temperature and moisture requirements for incubation are met. Cumulative daily infection values (*cDIVs*) varied between 0.5 and 11.2 according to the experiment, sowing occurrence and RH threshold (Table 2). As expected, the lower the RH threshold, the greater the *cDIVs*. For a 70% threshold, the 10th and 90th percentiles were 4.3 and 10.0, respectively. Using this threshold, observed disease onset dates were almost always included in the forecast window defined by the *P10* and *P90* predicted disease onset dates (Fig. 7). The observed date occurred before the forecast window in S8/2003–2004 (−4 days) and S6/2004–2005 (−2 days), and it occurred after in S1/2004–2005 (+1 day). Furthermore, the length of the predicted window varied greatly. Comparisons between the total prediction errors showed that the 70% RH threshold provided the best trade-off between the α and β type deviations (Fig. 8).

Discussion

Airborne primary inoculum availability

Airborne inoculum availability was assessed indirectly through trap plants. Trap plants, as opposed to Rotorod or Burkard spore samplers, are a robust way of assessing viable airborne inoculum. This method was well adapted for weekly assessment of inoculum release, and has been successfully used in previous studies concerning *M. pinodes* on pea (Roger and Tivoli 1996) or *Didymella rabiei* on chickpea (Trapero-Casas et al. 1996).

Under our experimental conditions, the amount of spores trapped in autumn and winter was extremely low and contrasted sharply with the amount of spores trapped in spring and attributed to ascospore-like secondary inoculum. How can the low amount of spores in autumn and winter be explained? Rainfall leaching of deposited spores is unlikely to be a significant factor, since according to the literature, ascospores of *M. pinodes* have a surface coating which causes them to adhere very firmly to objects with which they come into contact (Carter and Moller 1960). Thus, the low amount of spores trapped in autumn and winter may largely reflect a low level of airborne primary inoculum. This differs greatly from previous results concerning the seasonal release of ascospores of *M. pinodes* (Roger and Tivoli 1996; Zhang et al. 2005) or other ascomycota (Inman et al.

Table 2 Cumulative daily infection values (*cDIV*) calculated for various RH thresholds in field experiments conducted in France in 2003–2004 and 2004–2005

Year	Sowing number	RH threshold (%)						
		65	70	75	80	85	90	95
2003–2004	S1	nc	nc	nc	nc	nc	nc	nc
	S2	10.4	9.9	9.0	7.8	6.7	4.8	1.9
	S3	10.2	10.1	10.0	9.3	8.3	6.8	3.6
	S4	6.7	6.7	6.7	6.6	5.8	4.9	2.5
	S5	7.5	7.4	7.1	6.5	5.4	3.9	2.8
	S6	7.3	7.2	6.6	5.9	4.5	3.3	2.6
	S7	6.3	5.7	5.1	4.1	2.7	1.7	1.2
	S8	4.7	4.0	3.4	2.5	1.6	0.9	0.5
2004–2005	S1	11.2	10.6	9.7	8.6	7.5	5.6	3.7
	S2	8.2	7.9	7.3	6.1	5.1	3.9	2.7
	S3	6.0	5.8	5.0	4.5	3.8	3.0	2.1
	S4	5.7	5.6	5.4	5.2	4.5	3.4	2.4
	S5	7.8	7.7	7.4	7.1	6.3	5.0	3.0
	S6	3.7	3.6	3.5	3.4	3.3	2.9	1.8
	S7	4.8	4.7	4.6	4.4	3.9	3.2	2.1
	S8	6.3	6.2	5.8	5.4	4.6	3.1	1.7
	10th percentile	4.8	4.3	4.0	3.7	2.9	2.2	1.4
	90th percentile	10.3	10.0	9.4	8.3	7.2	5.3	3.4

In 2003–2004, the first disease assessment achieved on S1 revealed that disease onset had already occurred. Since disease onset was not accurately dated, the corresponding *cDIVs* were not calculated

1999; Salam et al. 2003; Trapero-Casas et al. 1996) probably because these authors placed their trapping systems (trap plants or spore samplers) directly in the centre or in the vicinity of infected debris. Our experiments were conducted in fields with a rotation that excluded pea during the previous five years. In addition, the nearest pea stubble was located, 1,600 m from the trial plots and was removed in early September before ploughing. It is therefore likely that the removal of the remaining debris would have drastically reduced the primary inoculum from this source.

In this study, airborne primary inoculum availability was partially explained by cumulative degree-days and maximum daily rainfall. Reports in the literature suggest that primary inoculum availability is highly dependent upon weather conditions. Under their experimental conditions, Trapero-Casas et al. (1996) observed that the number of lesions of *D. rabiei* on chickpea trap plants was significantly correlated with the number of days with rain during weekly periods. Zhang et al. (2005) showed that ascospores of *M. pinodes* were released 1–2 days after a rain event.

Modelling of disease onset

In this study, late sowing dates delayed disease onset. In Australia, this cultural practice is recommended to reduce the exposure of young plants to high levels of primary inoculum (Bretag et al. 2000). In French conditions, delaying sowing will mainly induce a delay in emergence due to decreasing temperatures during autumn and winter and consequently delay the deposition of the primary inoculum on young susceptible pea plants. Nevertheless, this practice also has limitations: firstly, delaying sowing can in some cases reduce yield (Bretag et al. 2000), and secondly, there is a risk that sowing may not be possible due to autumn rainfall.

Although assessed in very low quantities, primary inoculum appeared to be sufficient to initiate an epidemic. Carter and Moller (1960) reported that spores of *M. pinodes* were able to survive between conducive incubation periods (such as might be expected with overnight dews followed by dry days) and retain their ability to infect when favourable moisture conditions resume. Roger et al. (1999a) showed that symptoms were able to develop provided

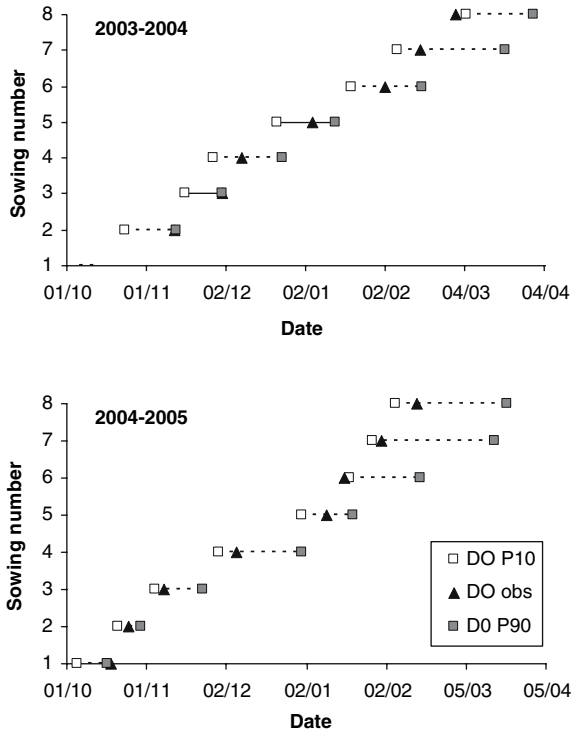

Fig. 7 Schematic representation of observed and predicted dates of ascochyta blight onset for field trials conducted in France in 2003–2004 and 2004–2005. DO_{obs} is the observed disease onset, DO_{P10} and DO_{P90} are respectively the predicted dates calculated for the 10th and 90th percentiles of the distribution of the cumulative disease infection values at 70% RH threshold

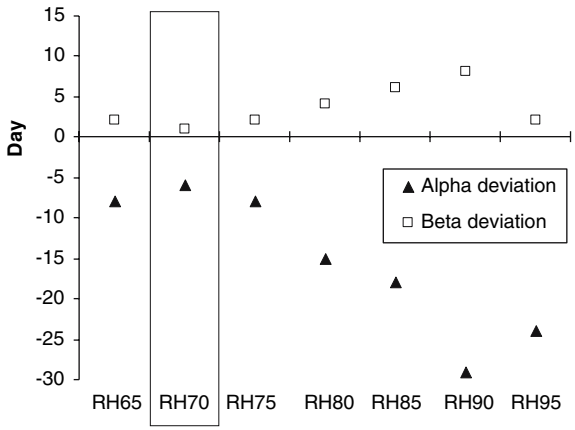

Fig. 8 Total deviations between observed and predicted ascochyta blight onset dates calculated for seven RH thresholds (65, 70, 75, 80, 85, 90 and 95%) from data obtained in field trials conducted in France in 2003–2004 and 2004–2005. Alpha and beta deviations are calculated respectively when observed disease onset occurred before and after the forecast window given by the model

that the dry period occurred after germination. Thus, the actual amount of ascospores available for disease onset in a given week is probably higher than the weekly availability assessed on trap plants and probably results not only from the weekly spore deposition but also from some viable spores from previous weeks.

Not much variability in the length of the time from emergence to disease onset was accounted for by mesoclimatic variables. A different modelling approach was used to predict the occurrence of disease onset. This was achieved through the calculation of cumulative temperature and moisture indices which were used to define a disease risk forecast window. A similar approach was used by Shane and Teng (1983) and Wolf and Verreet (2005) for *Cercospora beticola* and by Bugiani et al. (1993) for *Phytophthora infestans*. For ascochyta blight, we used data available in the literature to establish the mathematical functions. This study shows that the 70% RH threshold provided the best results for predicting disease onset. This rather low threshold (RH recorded at 1.40–2.20 m above ground level) is probably associated with optimal moisture periods within the crop canopy due to dew formation.

Wolf and Verreet (2005) proposed a negative prognosis (determining a disease-free period) based on the minimum value of the *cDIV* distribution. In our study, we chose to define the bounds of a forecast window in which disease onset is likely to happen. Except for three cases, the predicted forecast windows included the observed disease onset dates. For these three cases, the deviation varied between −4 and +1 (days), which is acceptable. The length of the forecast window was variable. The more conducive the weather conditions, the shorter the forecast window. Actual monitoring of the plants is therefore needed once the forecast window is reached and is all the more urgent if subsequent weather conditions are conducive.

To our knowledge, this study is the first attempt to predict ascochyta blight onset in field peas. Once validated with additional data, the basis of this model could be used to design a user-friendly tool to warn farmers about disease risk and possibly to advise them on an appropriate time for the first fungicide application. Therefore, this model could contribute to development of a more rational fungicide application schedule. Of course, such a tactical tool would not be of interest if fungicide applications are not econom-

ically beneficial. Finally, this model needs to be coupled to a disease progress model to predict the subsequent development of ascochyta blight and associated pea yield losses.

Acknowledgements This work was partially funded by the European Union through the Grain Legumes Integrated Project (FOOD-CT-2004-506223). We thank Gabriel Nedelec and Emile Lemarchand (INRA Rennes) for organizing the seeding, Aurélie Leclerc (INRA Rennes) for occasional technical help, and Joel Chadoeuf (INRA Avignon) for statistical advice. We also thank Cindy Morris (INRA Avignon) and Randy Kutcher (Agriculture and Agri-Food Canada) for kindly reviewing this manuscript for English language.

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