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Improvement and validation of a pea crop growth model to simulate the growth of cultivars infected with Ascochyta blight (*Mycosphaerella pinodes*)

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Abstract

A model simulating the growth of a pea crop infected with Ascochyta blight was improved and validated using 6 spring pea cultivars, all equally susceptible to Ascochyta blight, but differing in architectural features (stem height, branching ability, standing ability). This model takes into account the spatial distribution of the disease, including the contribution of each layer of the canopy to the radiation interception efficiency (RIE) and the radiation use efficiency (RUE) of the crop. The decreasing contribution of each layer due to the disease was estimated by the relationship between the photosynthesis of a layer and its disease score. The effect of disease on photosynthesis was assessed in controlled conditions as a means of evaluating the effect of disease on each cultivar. All cultivars were affected equally. In field conditions, cultivars with different canopy architectures displayed differences in the profile of disease on leaves. Cultivar Aladin reached higher disease levels at the top of the plant. Epidemics affected crop growth, and the cultivars tested differed in the magnitude of the decrease in growth. Observed and simulated data were compared. The disease-coupled crop growth model gave satisfactory predictions of crop growth for the six cultivars tested.

Introduction

Ascochyta blight, caused by *Mycosphaerella pinodes* (*Mp*), is one of the most damaging diseases of pea (*Pisum sativum*) world wide. It is responsible for yield losses of up to 30% (Allard et al., 1993). Yield losses are linked to pathogen frequency, distribution and disease intensity. In France, Ascochyta blight management is currently limited to systematic chemical spraying because little is known about the relationship between disease severity and yield loss.

Previous studies were carried out at the individual plant level. They showed that disease affected yield by decreasing seed number or individual seed weight (Tivoli, 1994; Xue et al., 1997, 1998; Garry et al., 1998). Garry et al. (1998) showed that *Mp* affected photosynthetic activity. Lucas et al. (1998) and Béasse et al. (2000) showed that, when the disease established late, it affected radiation use efficiency (RUE) but had little or no effect on radiation intercepted efficiency (RIE). For other pathosystems, such as late blight/potato (van Oijen, 1991) and leaf blast/rice (Bastiaans, 1993), leaf area formation may be affected due to early growth retardation causing a feedback regulation, and disrupting leaf area formation. For *Mp*/pea, growth was generally reduced after the beginning of seed filling, and leaf area was no longer expanding at this stage (Béasse et al., 2000). Moreover, canopy structure was already fully developed when disease occurred. These

observations have been incorporated into a crop growth model, simulating crop growth in the field and integrating disease development at each canopy layer (Béasse et al., 2000). These studies concerned only the cultivar Solara. However, a large number of spring pea cultivars have been developed by breeders and are currently cultivated. Experiments concerning the behaviour of various pea genotypes with respect to Ascochyta blight have already been carried out (Wroth, 1998; Xue et al., 1998) and several studies have shown that cultivars differently response to the disease depending on plant morphology (Bretag, 1991; Wroth and Khan, 1999). In this study, we chose six spring pea cultivars currently used in commercial production, selected on the basis of architectural features (stem height, branching ability, standing ability), to improve and validate the model developed by Béasse et al. (2000) for cultivar Solara.

Materials and methods

Model description

The model established by Béasse et al. (2000) to simulate the growth of a pea crop affected by *Mp* is based on the approach of Monteith (1972), used by Ney (1994) to a simulate the growth of a healthy pea crop. Daily biomass production in a healthy pea crop (DM) depends on the radiation absorbed by the canopy, and on its transformation into biomass, either:

$$\mathrm{DM}(\mathrm{g\,m^{-2}}) = \mathrm{RIE} \times \mathrm{RUE} \times \mathrm{PAR_0} \quad (1)$$

where RIE is the radiation interception efficiency, RUE is the radiation use efficiency, and $\mathrm{PAR_0}$ ($\mathrm{MJ\ m^{-2}}$) is the incident photosynthetically active radiation. RIE is calculated from leaf area index (LAI) ($\mathrm{m^2}$ leaf $\mathrm{m^{-2}}$ ground):

$$\mathrm{RIE} = \mathrm{RIE_{max}} \times [1 - \exp^{(-\mathrm{k}\times\mathrm{LAI})}] \quad (2)$$

where $\mathrm{RIE_{max}}$ is equal to 0.99 and k the extinction coefficient is equal to 0.49 (Jeuffroy and Ney, 1997; Wroth, 1998). RUE is calculated as the ratio between daily biomass production, using DM adjusted values (see statistical analysis section), and daily PAR intercepted by the crop ($\mathrm{PARa_c}$):

$$\mathrm{PARa_c} = \mathrm{RIE} \times \mathrm{PAR_0} \quad (3)$$

Daily biomass production in a diseased pea crop ($\mathrm{DM_d}$) is calculated as follows:

$$\mathrm{DM_d}(\mathrm{g\,m^{-2}\,day^{-1}}) = \mathrm{RIE_d} \times \mathrm{RUE_d} \times \mathrm{PAR_0} \quad (4)$$

The radiation use efficiency in a diseased pea crop ($\mathrm{RUE_d}$) is calculated as follows:

$$\mathrm{RU}E_d = \mathrm{RUE} \times \sum_{i=1}^{n} C_i \times (P_\mathrm{m}/P_0)_i \quad (5)$$

where C_i is the contribution of each node i to the PAR absorbed by the whole canopy and $(P_m/P_0)_\mathrm{i}$ is the reduction of photosynthesis by the disease for each node i (D_i):

$$\mathrm{C_i} = \frac{\mathrm{PARa_i}}{\mathrm{PARa_c}} = \frac{\exp^{-\mathrm{k}\times\sum_{\mathrm{j=i+1}}^{\mathrm{n}}\mathrm{LAI_j}} - \exp^{-\mathrm{k}\times\sum_{\mathrm{j=1}}^{\mathrm{n}}\mathrm{LAI_j}}}{1 - \exp^{-\mathrm{k}\times\sum_{\mathrm{j=1}}^{\mathrm{n}}\mathrm{LAI_j}}} \quad (6)$$

$$(P_\mathrm{m}/P_0)_i = a \times D_\mathrm{i} + b \quad (7)$$

with $a = -0.2396$ and $b = 1.0329$ for cultivar Solara (Béasse et al., 2000).

Cultivars

In order to improve and validate the model, six commercial semi-leafless spring pea cultivars differing in stem height, branching ability and standing ability were used in field and glasshouse experiments (Table 1).

Field experiments

Experimental design and treatments

Two field experiments were conducted at Le Rheu (Brittany, western France) in 1999 and 2000. Experimental treatments combined the six cultivars previously described and two disease scenarios (inoculated and non-inoculated plots) in a split-plot design with three replicates. Plot size was 8.5 $\mathrm{m^2}$. All cultivars were sown at the same density (80 seeds $\mathrm{m^{-2}}$). Artificial inoculation was achieved at the six- to seven-leaf stage by dispersing barley grains colonised by a mixture of *Mp* isolates on the ground as described by Tivoli et al. (1996). Non-inoculated plots were

Table 1. Architectural features of the six spring pea cultivars used in field and glasshouse experiments (Variétés de protéagineux 1999. Edition UNIP-ITCF, Paris, France)

Pea cultivar	Stem height	Branching ability	Standing ability
Aladin	High	Yes	Good
Athos	Low	Yes	Good
Baccara	High	Yes	Intermediate
Bridge	Low	Yes	Bad
Obelisque	High	No	Good
Solara	Intermediate	Yes	Intermediate

sprayed fortnightly, starting at flowering, with chlorothalonil (1500 g ha^{-1}, Visclor 500L, Isagro France) to prevent natural Ascochyta blight infestation.

Climate measurements

Daily mean temperature, rainfall and global incident radiation (GR) were recorded with an automatic weather station located 1 km away from the trials. Incident photosynthetically active radiation (PAR_0) was calculated as follows:

$$PAR_0 = 0.48 \times GR \quad (8)$$

Disease assessment

Ten main stems were randomly sampled per plot once a week from flowering to seed filling (Table 2). For each node, disease severity was visually assessed on stipules (the main photosynthetic organs of semi-leafless pea cultivars) using a 0–5 scale (Tivoli, 1994): 0 = no symptoms, 1 = a few necrotic flecks, 2 = numerous necrotic flecks, 3 = coalescing necrotic flecks covering less than 50% of the stipule area, 4 = 50–75% of the stipule area necrotic, 5 = more than 75% of the stipule area necrotic.

Leaf area assessment

Leaf area was assessed twice in 1999 and three times in 2000 (Table 2) on ten main stems randomly sampled per plot. The leaf area (stipules + tendrils) of each node was measured using a leaf area meter (LI-3100, LI-COR). Leaf area index (LAI) of node *i* was calculated as the product of the leaf area of node *i* and the number of stems per m^2. We assumed that the LAI of branches did not differ from that of the main stem at each of the nodes.

Dry matter assessment

Dry matter was measured three to four times during the experiment (Table 2) by harvesting biomass over an area of 0.8 m^2. The collected biomass was oven-dried for 48 h at 80 °C and weighed.

Table 2. Correspondence between calendar dates, degree-days since sowing, growth stages and assessment types at the sampling occasions of field experiments conducted in 1999 and 2000

Year	Date	Degree-days since sowing[a]	Growth stage[b]	Disease assessment	Leaf area assessment	Dry matter assessment
1999	24 May	798.3	BF	x		x
	31 May	923.2		x		
	7 June	1029.0		x		x
	14 June	1130.1	EF	x	x	
	21 June	1249.5		x		x
	28 June	1369.6	EBSF	x	x	
	5 July	1501.5		x		x
2000	29 May	843.8	BF	x	x	
	5 June	952.3		x		x
	12 June	1060.3		x	x	
	19 June	1206.5	EF	x		x
	25 June	1304.6		x	x	
	3 July	1488.1	EBSF	x		x
	10 July	1569.3		x		

[a]Basic 0 °C.

[b]BF = the beginning of flowering is the date on which the first reproductive node flowers; EF = the end of flowering is the date on which the last reproductive node flowers; EBSF = the end of the beginning of seed filling is the date on which the last reproductive node reaches the beginning of seed filling stage.

Glasshouse experiments

Plant production
Pea seeds were sown in 0.5 l pots (two seeds per pot) filled with a 1:1:1 (v/v) soil-sand-peat mixture. Pots were placed in a glasshouse where temperature was maintained at 15 ± 5 °C. Plants were grown under natural light supplemented with artificial light (300 μmol m^{-2} s^{-1} PAR) for 6 h per day until the five- to seven-leaf stage.

Inoculum production and plant inoculation
A single spore isolate of *M. pinodes* (*Mp* 91.31.12) was used. A mother pycnidiospore suspension was prepared by culturing the fungus on V8 juice agar for 10 days at 20 ± 1 °C with a 12 h photoperiod of white light (wavelength: 350–750 nm), flooding the culture with distilled water, gently scraping the agar surface and filtering through four layers of muslin to remove mycelium and agar fragments. The concentration of this pycnidiospore suspension was determined with a haemocytometer. In order to achieve a range of disease scores, dilutions were carried out from the mother suspension to obtain the following inoculum concentrations: 10^4, 5×10^4, 10^5, 5×10^5 and 10^6 pycnidiospores ml^{-1}. Tween (at 0.2%) was added to the pycnidiopore suspensions as a wetting agent. Plants were sprayed with the pycnidiospore suspensions (20 ml per 10 plants per cultivar and per inoculum concentration). Control plants (10 plants per cultivar) were sprayed with sterile distilled water supplemented with 0.2% Tween. Disease development was promoted by covering all sprayed plants with a transparent plastic bag for four to seven days and placing the pots in a growth chamber (14-h photoperiod, 15 ± 1 °C night and day) until photosynthetic activity measurement.

Photosynthetic activity measurement
Photosynthetic activity was measured in a duplicated experiment. Control plants and inoculated plants with disease scores of 1, 2, 3 or 4 on the fifth to seventh stipule were selected for measurement. For each cultivar, three stipules were gently harvested per disease score. Harvested stipules were placed individually in the chamber of an infrared CO_2 analyser (ADC, LC-A2) with constant temperature (20 °C), relative humidity (60%) and light intensity (800 μmol m^{-2} s^{-1}). Ambient CO_2 concentration was 380 ppm. Net photosynthetic rate was determined by measuring CO_2 exchange rate. Means were calculated for each disease score and each cultivar. For a given disease score, the relative net photosynthetic rate (P_m/P_0) was calculated as the ratio between the net photosynthetic rate of diseased stipules (P_m) and the net photosynthetic rate of healthy stipules (P_0).

Statistical analysis

The statistical analyses were carried out by using SAS software package (SAS Institute, 1997). The effect of node and treatment and their interaction on LAI, and the effect of node and cultivar and their interaction on disease score, were tested by using the least significant difference (LSD-test). Effect of the plant genotype on the reduction of photosynthesis due to the disease was estimated through simple linear regression by using the GLM procedure. For each cultivar, the estimated coefficients a and b of the linear regression ($y = a.x + b$) were compared to those obtained for Solara by means of a t-test (Scherrer, 1984). The coefficients estimated for the Solara cultivar were compared to those obtained by Béasse et al. (2000) in the same way.

In order to validate our model, the simulated and observed dry matter data were compared by using simple linear regressions. If the model gave good estimation of total dry matter (i.e., the null hypothesis) then the linear regression equation is $TDM_{sim} = a \times TDM_{obs} + b$, with $a = 1$ and $b = 0$. For each cultivar, a t-test (Scherrer, 1984) was used to compared the estimated values of the coefficients of the linear regression to the expected ones (i.e., $a = 1$ and $b = 0$).

Results

Disease severity

The Ascochyta blight epidemics were more severe in 2000 than in 1999. In both years, all cultivars presented similar disease profiles, with high disease scores towards the base of the plant and lower disease scores towards the top (Figure 1). Disease evolved equally for each cultivar. However, Aladin was attacked significantly more strongly at the top of the plant than were the other cultivars.

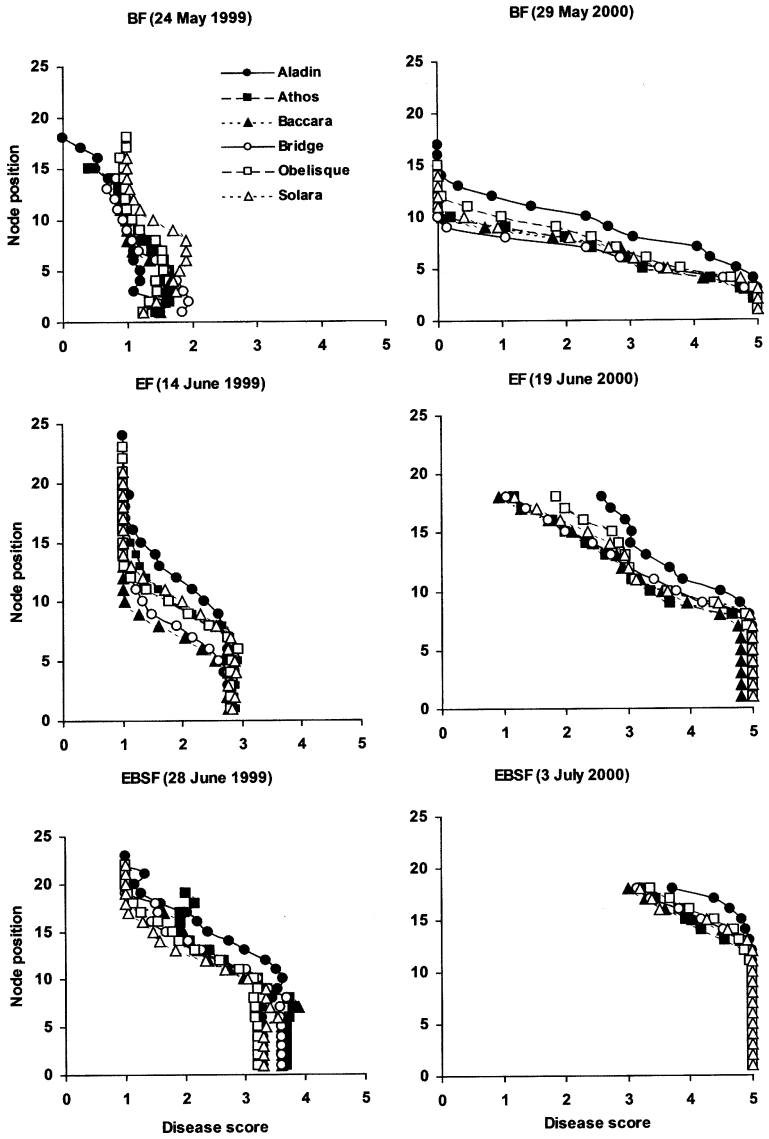

Figure 1. Disease severity profile on stipule for the six spring pea cultivars in inoculated plots from field experiments in 1999 and 2000 (BF = beginning of flowering; EF = end of flowering; EBSF = end of the beginning of seed filling).

Canopy structure

Although sown at the same density (80 seeds m^{-2}), cultivars differed in plant density in 1999 (Table 3). No significant difference was observed in 2000. Stem densities ranged from 61 to 104 (Obelisque and Bridge, respectively) in 1999 and from 76 to 118 (Obelisque and Bridge and Solara, respectively) in 2000. Disease had no impact on plant and stem densities (data not shown).

LAI profile differed considerably among cultivars in 2000 (Figure 2). Indeed, Athos, Baccara, Bridge and Solara differed in the level of LAI peak (from 0.45 to 0.80 m^2 leaf m^{-2} ground), but not in the position of this peak on the height of the plant (nodes 14–15). The peak of Aladin was located higher on the plant (node 19). No significant differences were observed between the LAI profiles in inoculated and non-inoculated plots in 2000, except for Athos and Bridge, which differed in LAI profile (Figure 3).

Table 3. Plant and stem densities of the six spring pea cultivars in field experiments conducted in 1999 and 2000

Pea cultivar	Plant number per m^2		Stem number per m^2	
	1999	2000	1999	2000
Aladin	68abc	68a	83c	104b
Athos	63bc	64a	83c	89c
Baccara	74ab	65a	97ab	112ab
Bridge	78a	73a	104a	118a
Obelisque	59c	66a	61d	76d
Solara	78a	66a	94b	118a

Means in a column followed by the same letter are not statically different ($P = 0.05$) according to LSD test.

For these two cultivars, the LAI in inoculated plots was higher at the bottom of the canopy and lower at the top than in non-inoculated plots. Disease had no effect on leaf development in 1999 (data not shown).

Genotype effect on radiation interception

LAI profile showed large differences among cultivars (Figure 2). As radiation interception is dependant on this distribution, differences were also observed in the contribution of nodes to interception (Figure 4). Cultivars presented a same general profile, low PAR interception towards the bases, high interception towards the top of the plant, but they also expressed important differences on nodes.

Genotype effect on the reduction of photosynthesis due to the disease

For each cultivar, P_m/P_0 linearly decreased with disease scores (Figure 5). Estimated slopes and intercepts were not significantly different between cultivars. In addition, they were not significantly different from those obtained by Béasse et al. (2000). Consequently, a linear regression common to all cultivars was used:

$$(P_{\mathrm{m}}/P_0)_i = -0,24510D_i + 1,0973 \qquad (9)$$

Model validation

Results showed that the model gave a good estimation of the total dry matter for three of the six cultivars (i.e., Athos, Obelisque and Solara)

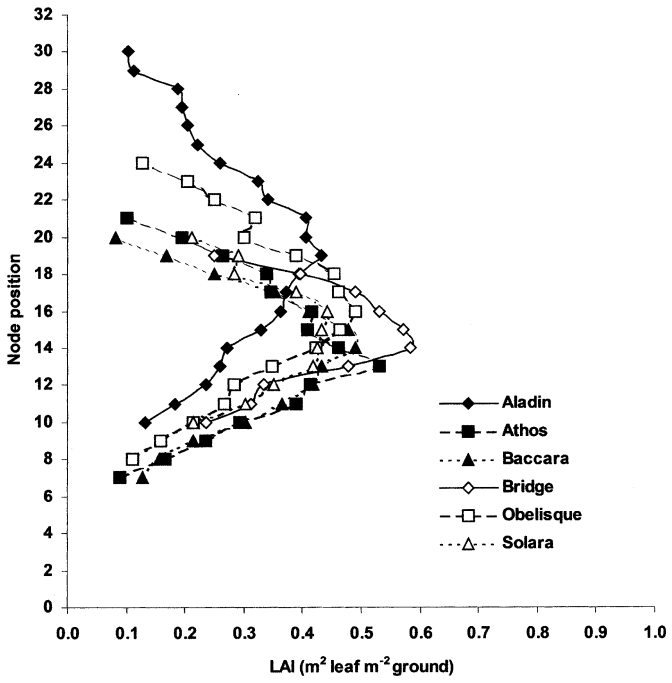

Figure 2. Leaf area index (LAI) as a function of node position for the six spring pea cultivars in non inoculated plots in 2000 field experiment (25 June 2000, EF + 1 week).

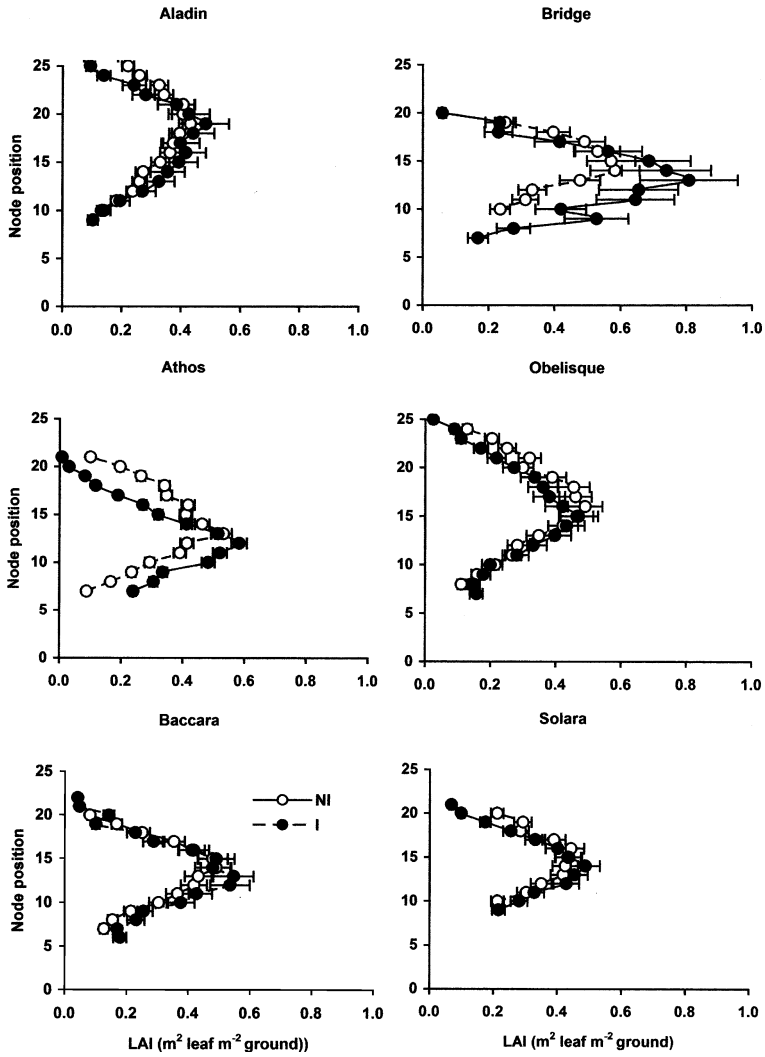

Figure 3. Leaf area index (LAI) as a function of node position for the six spring pea cultivars in non inoculated (NI) and inoculated (I) plots in the 2000 field experiment (25 June 2000, EF + 1 week).

(Table 4). For the three other cultivars, the last point could explain the model deviation (Figure 6). For these cultivars, the senescence is sooner than for the others. This last point is then likely due to the effect of senescence on the dry matter. Consequently, a second analysis of model was done without this point. Under these conditions, the model was validated. However, it should be noted that all *a*-values are below 1 (not significantly) and all *b*-values are positive (not significantly), so that TDM_{sim} always shows the same trend: overestimation for low values of TDM_{obs} and underestimation for high values of TDM_{obs} (see Figure 6).

Discussion

We used the disease-coupled crop growth model developed by Béasse et al. (2000) to assess the dynamic effects of disease on crop growth. This model is based on the combination of disease progression in the canopy (number of nodes affected by the disease) and the structure of the canopy (LAI profile). The steps of the calculation were: (1) estimation of the contribution of each node to radiation absorption (C_i) as suggested by Waggoner and Berger (1987), (2) calculation of the reduction in the contribution of each node due to

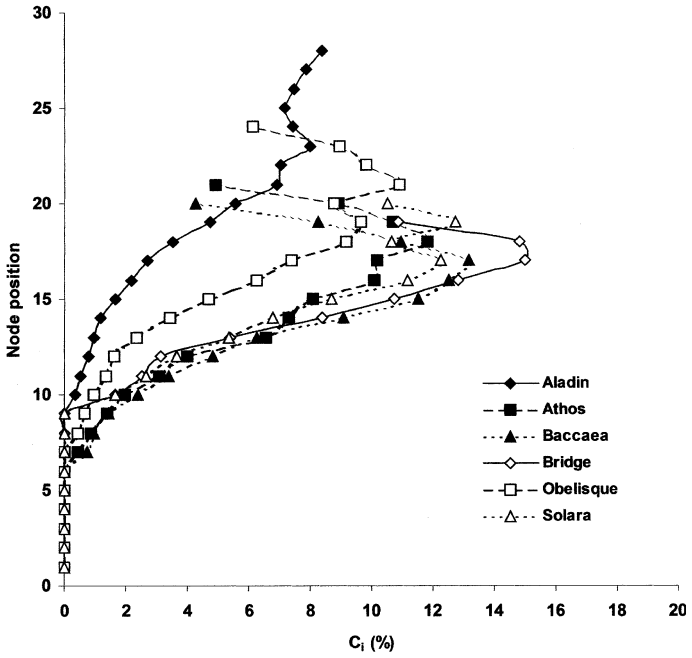

Figure 4. Contribution to radiation interception (C_i) as a function of node position in field experiment (25 June 2000, EF + 1 week).

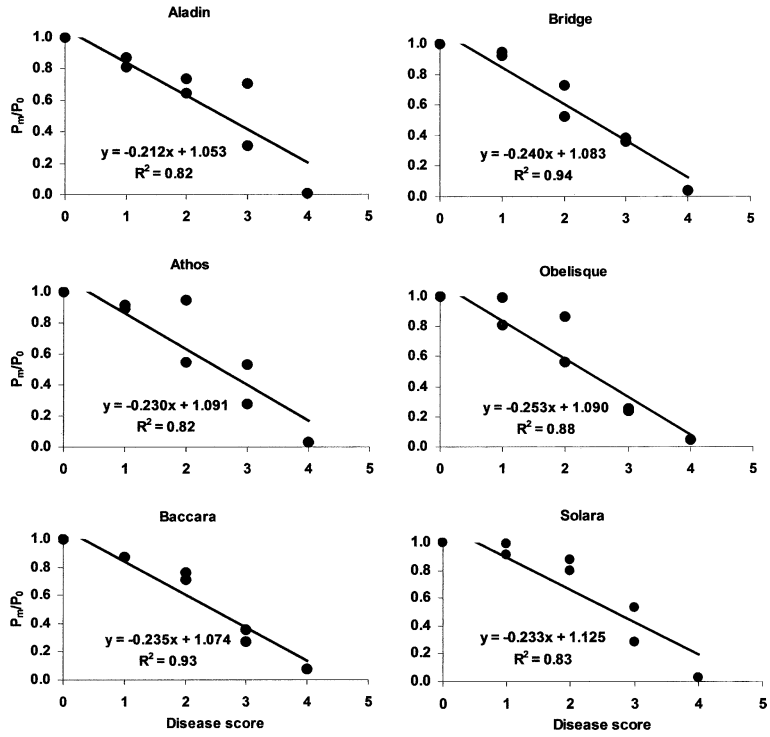

Figure 5. Effect of disease on photosynthesis: relationship between the ratio P_m/P_0 (P_m: photosynthesis for inoculated leaves, P_0: photosynthesis for healthy leaves) and disease scores for the six cultivars in controlled conditions.

Table 4. Statistical analysis (Student—Newman–Keuls test) for the validation of the crop growth model (see Scherrer, 1984 for explanation)

Pea cultivar	*a*-test*			*b*-test*		
	a-value	*t*-value (df)	*P*-value	*b*-value	*t*-value (df)	*P*-value
Aladin	0.68	2.75(4)	0.05	185.52	2.25(4)	0.08
Athos	0.79	2.04(5)	0.09	84.97	1.04(5)	0.34
Baccara	0.73	1.99(4)	0.10	137.94	1.68(4)	0.17
Bridge	0.77	2.40(4)	0.06	118.67	1.98(4)	0.11
Obelisque	0.78	2.12(5)	0.08	118.99	1.53(5)	0.18
Solara	0.82	2.45(5)	0.05	103.47	1.83(5)	0.12

*a-test; null hypothesis: $a = 1$.
*b-test; null hypothesis: $b = 0$.

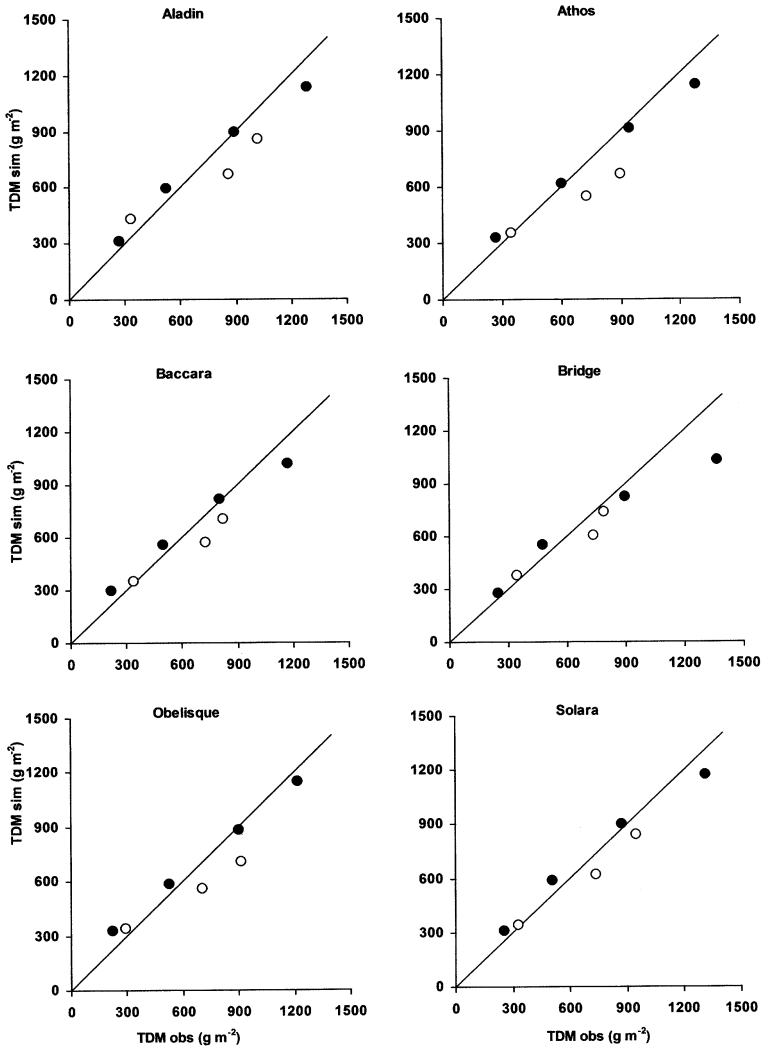

Figure 6. Comparison between total dry matter observed in diseased plots (TDM obs) and total dry matter simulated by the model (TDM sim) of spring pea cultivars in 1999 and 2000 field experiment. • TDM in 1999, o TDM in 2000.

disease, using the relationship between the relative decrease in photosynthetic activity of a diseased leaf and its disease score and, (3) summing these individual contributions to provide an estimation of the total crop growth.

The model has been validated, as it application is the adapted one for the referenced cultivar of our study, the Solara one. It gave also a good fit for all the other cultivars. This good estimation of the crop growth, confirmed the simulations carried out by Béasse et al. (2000) for one cultivar (Solara) in various environments. Results of the analyses indicate a good estimation of the model for the six cultivars, if the last point is not considered. Indeed, for three of the six cultivars (Aladin, Baccara and Bridge) this last point, corresponding to the beginning of the plant senescence, is responsible for a model deviation. In fact, the cycle end of the pea crop shows an important senescence. At this time, we cannot make the difference between senescence due to the disease development, and natural senescence.

Like Garry et al. (1998) and Béasse et al. (2000), we found that *Mp* decreased photosynthesis as a function of disease score. Surprisingly, there were no differences among cultivars in the decrease in photosynthesis. The main observed effect was due to a combination of canopy structure (LAI profile) and the progression of the disease profile within the canopy. As expected, there was considerable variability in canopy structure. Height, number of nodes, number of stems per m^2 and LAI profiles differed between cultivars. These differences implied variation in three parameters, RIE_i, C_i and RUE_i. Indeed, as radiation interception was dependent on the distribution of LAI according to the different nodes of the plant, differences were observed among the cultivars in the C_i profiles.

These results confirmed the hypothesis that growth reduction of a pea crop can be explained through a reduction in RIE as well as RUE. However, in our experiments, like for Béasse et al. (2000), reduction of RIE was only related to a decrease of intercepted radiation, but not to a decrease of LAI development. The contribution of both components to the overall reduction varies and is related to the relative extent of the disease on the nodes and confirming that growth decrease was mostly the result of a decrease in RUE.

Differences in damages due to disease were observed between years and among cultivars. More damage was observed in 2000 than in 1999, probably because the disease occurred earlier than in 1999. Disease profile progressed rapidly in both years only on cultivar Aladin. As this cultivar was also the tallest, we hypothesised that the height of the plants might be related to disease progression. The "functional" nodes for photosynthesis are located high in the canopy but the disease progressed rapidly to reach them. Thus, both the rate of progression of the disease along the stem and canopy structure (LAI profile) must be taken into account to quantify susceptibility. The conjunction between the canopy structure (LAI profile) and the progression of the disease profile inside the canopy implied differences on RUE_i profile. The worse effect occurs when the canopy is low and the disease progression rapid. In that case, the disease affects rapidly the "functional" nodes, which highly contribute to the global photosynthesis. That was the case for Bridge which is susceptible to the disease in both years and for Baccara in 2000.

Despite its large effect in the quantification of sensitivity, the causes of the variability in disease progression as a function of canopy structure remain unclear. The structure of the canopy may modify microclimate and microclimate is known to influence disease development (Ali et al., 1978; Hedley and Ambrose, 1981; Rewal and Grewal, 1989). Differences in the density of stems may affect canopy architecture leading to the development of a microclimate favourable for disease development (Bretag, 1991). Canopy architecture may also affect disease spread by wind or rain splash. Bretag (1991) showed that more open canopies allow greater air movement, affecting spores dispersal and microclimatic conditions. However, cultivars Athos, Baccara, Bridge and Solara differed in the level of peak LAI in 2000, but not in the position of this peak or the height of the plant. Differences in leaf density did not lead to differences in the rates of progression of the disease profile, which were very similar.

Alternatively, the distance among stems in the canopy may have an effect, by reducing spore dissemination caused by splashing. Cultivar Obelisque had the smallest number of stems, and the largest distance between nodes. Despite this, the rate of disease progression for this cultivar was higher than that of all other cultivars except of Aladin.

The susceptibility of a plant to infection may also have an effect. Experiments concerning the

behaviour of various pea genotypes with respect to Ascochyta blight have shown these differences (Wroth, 1998; Xue et al., 1998). Thus, the influence of canopy structure on disease progression must be known and modelled. The morphology of plants is the result of the interaction between its genome and the environment during evolution (Porta-Puglia et al., 2000). The model described will be useful for breeders to design new plant types: for separating intermediate variables, classifying them in order of importance, and finally, to account for differences among cultivars. The development of this model is also very important to the development of new disease control strategies. Ascochyta blight management is currently limited to systematic chemical spraying. This model can be used in various ways, like exploring expectations for disease effects, predicting the consequences of mixtures and cultural practices on disease development, and developing decision aids for the use of chemical treatments. Finally, this study highlights the importance of testing the behaviour of the genotypes in conditions of competition in crops.

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