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FULL RESEARCH PAPER

Evaluation of models to predict take-all incidence in winter wheat as a function of cropping practices, soil, and climate

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Abstract The incidence and severity of take-all, caused by *Gaeumannomyces graminis* var. *tritici* (Ggt), in susceptible crops depend on climate, soil characteristics and cropping practices. Take-all can be controlled by modifying crop rotation, crop management and fungicide treatment. When available, fungicides are used as a seed treatment and are partially effective. There is currently no reliable method for helping farmers to optimise their choice of cropping system to improve take-all control. In this study, we defined 16 models, based on various mathematical functions and input variables, for predicting disease incidence in a wheat crop as a function of soil characteristics, climate, crop rotation and crop management. The parameters of these models were estimated from field experiments carried out at six sites in the north of France over a ten-year period. The root mean squared error of prediction (RMSEP) values of the models were estimated by cross validation and compared. RMSEP was in the range 16.34–65.93% and was higher for the models based on multiplicative functions. The lowest RMSEP value was obtained for a dynamic model simulating disease incidence during the crop cycle and which included among input variables the percentage of diseased plants determined at GS30.

Keywords Epidemiology · Decision support · Integrated crop protection · Model selection

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Introduction

The incidence and severity of take-all, caused by the soilborne fungus *Gaeumannomyces graminis* var. *tritici* (Ggt), in susceptible crops depend on climate, soil environment (soil texture, structural stability, depth, water availability, pH and nutritional status), crop rotation and crop management (Cook 2003). These factors may affect the survival of primary inoculum in the soil, its growth and propagation and the risk of infection (Hornby et al. 1998). The sequence of crops in the rotation determines the build-up of primary

inoculum, depending on the number of host crops in succession. Rotation and soil tillage determine the position of infectious residues in the soil profile, as shown for eyespot (Colbach and Meynard 1995). The amount of wheat at risk from take-all increases with increasing number of host crops in the rotation (Colbach et al. 1994), but wheat monoculture can lead to a biological suppression of the disease known as take-all decline (Hornby 1983), which has been attributed to antagonistic microflora (e.g. fluorescent pseudomonads). Sowing date determines the length of the autumn period during which roots may be infected from the primary inoculum, with earlier sowing favouring pre-winter infections (Colbach et al. 1997). Conversely, late sowing, associated with lower soil temperatures, results in conditions less favourable for Ggt infection. High plant densities at the start of the growing season, when the number of roots per plant is low, increase the probability of contact between soil inoculum and the roots. Nitrogen fertilisation also affects take-all development. The use of ammoniacal forms of nitrogen fertiliser decreases the pH of the rhizosphere, stimulating the antagonistic microflora and inhibiting fungal development (Sarniguet et al. 1992).

Soil environment and climate are known to affect disease, but their effects are not as well understood as for cropping practices. Regional climate may influence primary infections. High temperatures and humidity in early autumn are known to favour seed germination, root growth, and growth of the fungal mycelium, thereby increasing the probability of contact and infection. Lucas et al. (1998) carried out a statistical and graphical analysis with Criticor software (Pierre et al. 1986) based on data collected over a 12-year period at the INRA experimental station at Le Rheu, France. They identified three cumulative rainfall and temperature periods at the beginning of the growing season that favoured development of the disease.

The take-all development depends on soil texture, as Ggt appears to be most active in shallow soils with a light texture (e.g. sandy silts and silty sands) (Cook et al. 1968), which favour fungal mobility and heat up more rapidly in spring. Clayey soils are more susceptible to compaction, resulting in poor aeration, which may limit disease expression. Calcareous clays, which are light and airy, are known to favour the development of the fungus. In a comparative study of 58 soils with different physicochemical properties in identical control conditions, Lucas and Nignon (1987) found that take-all was present in greater amounts on silty soils than on sandy soils.

Since 2002, silthiofam (Latitude® from Monsanto Agriculture SAS) has been used as a fungicidal seed treatment to control this disease in most European countries. It is currently the most effective fungicide against take-all, although it does not provide full control of the disease (Schoeny and Lucas 1999). It can delay disease development by slowing primary infection, and seems to be more effective at limiting root-to-root spread than at limiting the expansion of necrosis on diseased roots (Schoeny and Lucas 1999).

Efficient control of take-all should almost certainly include both the modification of cropping practices and the use of fungicides. However, there is currently no reliable method for determining when treated seeds should be sown based on the risk of take-all associated with soil, climate and cropping practices. A model predicting the incidence of the disease at key stages of the crop cycle could be combined with models predicting yield loss (Schoeny et al. 2001). The resulting combined model would enable farmers and their advisors to identify situations in which fungicide seed treatment is required. Such a model could also be used to define integrated crop protection strategies based on modified cropping practices.

Colbach et al. (1997) developed a model based on the epidemiological function developed by Brassett and Gilligan (1989). This model presents the advantage of including two parameters of real epidemiological significance rather than simple adjustment coefficients. These parameters are related to the primary and secondary infection cycles. In this study, we consider both the Brasset and Gilligan function (1989) used by Colbach et al. (1997) and alternative models based on other mathematical functions for predicting disease incidence. We estimate parameters for these models from experimental data collected from various sites, mostly in the northern part of France. Finally, we evaluate the precision of the predictions obtained with these models.

Materials and methods

Equations for the models

We considered both dynamic and static models, the equations of which are given below.

Equations for dynamic models

All the dynamic models considered in this study result from the integration of the kinetic function defined by Brassett and Gilligan (1989) and simplified by Colbach et al. (1997). This function is used here to link disease incidence (y) to thermal time (t).

$$y = \frac{1 - \exp^{-(C_1+C_2)\cdot t}}{1 + \frac{C_2}{C_1}\cdot \exp^{-(C_1+C_2)\cdot t}} \quad (1)$$

C_1 and C_2 are kinetic parameters associated with primary infections originating from inoculum surviving on former host crop residues and secondary infections arising from existing infections, respectively, and t is thermal time, expressed in cumulative degree-days (base 0°C) since sowing. Eq. 1 can be used to predict the disease incidence at any time during the growing season and can be easily connected with yield loss functions (Schoeny et al. 2001). More sophisticated versions of Eq. 1 were defined by Brasset and Gilligan (1989) and Gilligan and Brasset (1990) but these complex versions include a higher number of parameters that can not be easily estimated from our experimental data.

For the model to be operational, we need to link C_1 and C_2 to the soil characteristics, climate and cropping practices. Two types of relationship will be considered in this work.

We will first link C_1 and C_2 to the input variables in an additive manner

$$C_1 = \mu^{(1)} + \alpha_1^{(1)}\cdot x_1 + \cdots + \alpha_n^{(1)}\cdot x_n \quad (2)$$

$$C_2 = \mu^{(2)} + \alpha_1^{(2)}\cdot x_1 + \cdots + \alpha_n^{(2)}\cdot x_n \quad (3)$$

where $\mu^{(1)}, \mu^{(2)}, \alpha_1^{(1)}, \ldots, \alpha_n^{(1)}, \alpha_1^{(2)}, \ldots, \alpha_n^{(2)}$ are the parameters of the model and x_1,..., x_n are the input variables of the model. This model may be generalised to include interactions between input variables, but we do not consider this possibility here because published studies provide no evidence of significant interactions (Colbach et al., 1994, 1997) and because such interactions are accounted for by the multiplicative functions described below.

Alternatively, C_1 and C_2 may be linked by multiplicative relationships, as defined below:

$$C_1 = \mu^{(1)} \times x_1^{\alpha_1^{(1)}} \times \cdots \times x_n^{\alpha_n^{(1)}} \quad (4)$$

$$C_2 = \mu^{(2)} \times x_1^{\alpha_1^{(2)}} \times \cdots \times x_n^{\alpha_n^{(2)}} \quad (5)$$

where $\mu^{(1)}, \mu^{(2)}, \alpha_1^{(1)}, \ldots, \alpha_n^{(1)}, \alpha_1^{(2)}, \ldots, \alpha_n^{(2)}$ are the parameters of the model and x_1,..., x_n are the input variables of the model.

With multiplicative relationships, the effect of one input variable depends on the values taken by the other variables. The effects of different variables may therefore be amplified, or may be cancelled out if one of the variables is null. Relationships of this type have been proposed for take-all and for eyespot (Colbach et al., 1997, 1999), but it is unclear whether they are more appropriate than linear models.

By combining Eqs. 1, 2, and 3 or Eqs. 1, 4 and 5, we can predict disease incidence as a function of cumulative temperature, soil characteristics, climate and cropping system.

Equations for static models

We can also model take-all epidemics by directly relating the disease incidence at one wheat growth stage to agronomic variables and climatic factors. We chose to focus on disease incidence on nodal roots at midstem elongation, corresponding to GS32 and GS33 in Zadoks growth scale (1974), because disease incidence at this growth stage has been reported to be strongly correlated with yield loss (Schoeny et al. 2001) in the region of this study. As for the previous kinetic function, it is also possible to use a linear model defined as follows

$$y = \mu + \alpha_1 \cdot x_1 + \cdots + \alpha_n \cdot x_n \quad (6)$$

or a multiplicative model defined as follows:

$$y = \mu \times x_1^{\alpha_1} \times \cdots \times x_n^{\alpha_n} \quad (7)$$

Where y is the disease incidence at GS32-33, μ, α_1, ..., α_n are the model parameters and x_1,..., x_n are the input variables of the model.

Input variables

The models considered in this study include variables related to climate, soil texture, incidence of the disease at an early stage, crop rotation and wheat crop management. The choice and transformations of input variables are summarised below.

Climatic variables

Four input variables for climate are defined. Three are based on results from Lucas et al. (1998) and correspond to cumulative rainfall in mm. These variables are described in detail in Table 1. The fourth variable is cumulative temperature, expressed in degree-days (base 0°C) from sowing to 31 January.

Soil texture

This is the only soil component for which data are recorded in the database. Soil textures were grouped according to the major types found in France, giving a total of four groups: silts (corresponding to silts, silty sands and sandy/clayey silts), sands (corresponding to sandy-silty soils), clays (corresponding to clayey silts, silty clay and fine clayey silts) and calcareous clay soils.

Inoculum

The risk of disease varies with the origin of the inoculum and its location. We therefore used the 'pre-previous crop × previous crop × soil tillage' combination to reflect the initial inoculum level, with nine levels, depending on the data set. We distinguished between host, non-host and amplifying crops as described by Colbach et al. (1994). Cases of wheat monoculture are considered separately. Two levels of soil tillage are distinguished: conventional tillage based on ploughing (till) and direct drilling without soil inversion (no-till). According to Colbach and Meynard (1995), this variable may affect the risk of primary infection only.

Sowing density

(number of seeds m^{-2}) is used as the input variable relating to population density as this variable was the only one recorded in all the trials analysed.

Total available ammoniacal nitrogen

As different nitrogen fertilisers may be used for different types of applications, we defined a variable 'N' corresponding to the amount of ammoniacal nitrogen fertiliser available to the plant in the first few centimetres of soil. This variable takes into account the dose, form and number of nitrogen applications over the entire crop cycle and is defined as follows:

$$N = \sum_{i=1}^{q} \mathrm{Dose}_i \times \%\mathrm{NH4}_i,$$

where Dose_i is the ith dose of fertiliser applied to the crop (kg ha^{-1}) and $\%NH_{4i}$ is the fraction of

Table 1 Description of the three climatic variables

Climatic variable	Definition	Start of period (days)	Duration of the period	Min	Max	Mean
P1	Cumulative rainfall in mm since sowing	20	19 days	1.0	76.0	40.7
P2	Cumulative rainfall in mm since sowing	81	9 days	0.0	45.0	11.8
P3	Cumulative rainfall in mm since sowing	124	25 days	5.5	90.4	40.4

Exponential(Pi) was used in the multiplicative models

this application supplied in ammoniacal form. This fraction is 50 if the fertiliser was applied in the form of ammonium nitrate, 75 if a mixture of ammonium nitrate and ammonia in solution was applied and 100 if ammonia in solution, urea or ammonium sulphate was applied.

Fungicide

This variable takes two levels in this study: seed treatment with the fungicide applied at a rate of 25 g a.i./100 kg of seed (F^+) versus use of untreated seeds (F–). A few trials in which the fungicide was applied at a rate of 12.5 g a.i./100 kg of seed were included with the rest of the treated crops as Schoeny and Lucas (1999) found that the application of fungicide at this rate had a similar effect on the disease.

Disease incidence on the seminal root system 'Fsem30'

Disease incidence measurements on seminal roots were available for GS30 in most field trials. For one of the trials, the stage closest to GS30 available for such data was GS15, and for seven trials it was GS31.

Database used to estimate model parameters

The data used in this study were obtained from three published research studies aiming (i) to establish models describing the effects of cropping systems on disease dynamics (Colbach et al. 1997); (ii) to evaluate the effect on disease dynamics of various rates of seed treatment with the fungicide silthiofam combined with various nitrogen fertilisation strategies (Schoeny and Lucas 1999) or soil tillage techniques; (iii) to assess the effect of late irrigation on fungal dynamics at the end of the crop cycle and the effect of different summer fallow management systems on disease dynamics (Ennaifar et al. 2005).

Experimental trials

The database includes 25 field trials carried out with diverse experimental designs. In each trial, two to 24 different cropping systems were tested, with two to 10 blocks. These trials were carried out over a ten-year period, between 1992 and 2003, at six locations in the northern half of France: Chartres, Grignon, Le Magneraud, Le Rheu, La Verrière, Mons-en-Chaussée. These trials generated data for 204 site/year/cropping system combinations. These combinations will be referred to hereafter as 'treatments'.

Experimental measurements

Three types of experimental measurement were used in this study. The first was soil analysis (for granulometric determinations), which was carried out in most trials. For trials in which this analysis was not carried out for the experimental plot, we assumed soil texture to be similar to that observed in another trial at the same site. The second type of measurement concerned weather data. Mean daily temperatures and daily rainfall were recorded throughout the crop cycle, at meteorological stations located at about 100 m from the experimental plots. The third type of measurement concerned the disease, and such measurements have been described in detail elsewhere (Colbach et al. 1997; Schoeny and Lucas 1999). For each cropping system and each block, the percentage of plants with diseased roots was assessed three to seven times during the crop cycle. Disease incidence was assessed in two ways before stem elongation (beginning at GS30): the percentage of plants with diseased seminal roots and the percentage of plants with diseased nodal roots. After GS30, only the percentage of plants with diseased nodal roots was assessed. Each disease assessment was based on three to six samples of eight to 20 plants per block (Table 2). The percentage of diseased plants was determined for each sample and means were then calculated for each block of each treatment. The percentage of plants with diseased nodal roots is referred to hereafter to as the 'incidence' and is denoted y.

The treatments studied covered the entire range of percentages of diseased plants, from early to late stages (Fig. 1).

Estimation of parameters for dynamic models

The parameters of dynamic models were estimated in two steps. The values of C_1 and C_2 in

Table 2 Description of the plant samples used for disease measurements

Trial	Year of experiment	No. of samples	Sampling stage (GS) available	No. of blocks	Sample dimensions	No. of plants
Rh92	1991–92	4	15-30-50-80	NA	NA	NA
Rh93	1992–93	4	15-30-50-80	NA	NA	NA
Lv92	1991–92	4	15-30-50-80	NA	NA	NA
Char93	1992–93	4	15-30-50-80	4	25 cm × 2 rows	8
Gri93	1992–93	4	15-30-50-80	5	50 cm × 2 rows	10
Péro93	1992–93	4	15-30-50-80	4	25 cm × 2 rows	10
LR295	1994–95	6	22-23-30-31-33-65	6	50 cm × 2 rows	10
LR296	1995–96	7	22-23-30-31-34-65-83	3	25 cm × 2 rows	10
LR396	1995–96	7	22-23-30-31-34-65-83	4	25 cm × 2 rows	10
P297	1996–97	6	23-31-33-45-65-83	4	25 cm × 2 rows	10
P397	1996–97	6	23-31-33-41-65-83	4	25 cm × 2 rows	10
LR298	1997–98	6	30-32-33/34-39/43-65/69-83	4	25 cm × 2 rows	10
LG498	1997–98	6	22-31-33/34-39/43-65/69-83	4	25 cm × 2 rows	10
LG100	1999–00	3	31-39-65	3	A handful	10
LG300	1999–00	3	31-39-65	4	A handful	10
LG400	1999–00	3	31-39-65	4	A handful	10
LG600	1999–00	3	31-39-65	nd	A handful	10
Gri201	2000–01	5	15-30-32-51-73	4	50 cm × 2 rows	20
Gri601	2000–01	5	15-30-32-51-73	4	50 cm × 2 rows	20
Gri702	2001–02	5	15-30-33-65-89	4	25 cm × 2 rows	10
Gri202	2001–02	5	15-30-33-65-89	4	25 cm × 2 rows	10
LR202	2001–02	5	15-30-33-65-89	4	25 cm × 2 rows	10
LM202	2001–02	5	15-30-33-65-89	6	25 cm × 2 rows	10
BJ202	2001–02	5	15-30-33-65-89	6	25 cm × 2 rows	10
LR203	2002–03	4	15-33-59-83	4	25 cm × 2 rows	10

GS: growth stage according to Zadoks decimal scale (Zadoks et al. 1974)

NA: not available

Eq. 1 were first estimated for each treatment. All the estimates for C_1 and C_2 were then used to estimate the values of the parameters $\mu^{(1)}, \mu^{(2)}, \alpha_1^{(1)}, ..., \alpha_n^{(1)}, \alpha_1^{(2)}, ..., \alpha_n^{(2)}$ of Eqs. 2–5.

Step 1

Estimation of C_1 and C_2 for each treatment. The values of C_1 and C_2 were estimated for each treatment by the weighted least squares method, using the non-linear model (NLIN) procedure of SAS with the WEIGHT option (SAS Institute Inc. 1989). This method involved determining the values of C_1 and C_2 minimising the following equation

$$S(C_1, C_2) = \sum_{i=1}^{n} \sum_{j=1}^{k} \left[y_{ij} - f(t_i; C_1, C_2) \right]^2 \times \omega_i$$

where f is the function defined by Eq. 1, y_{ij} is the measured disease incidence on date t_i for block j and $\omega_i = \frac{1}{\sigma_i^2}$ with $\sigma_i^2 = \frac{1}{k-1} \sum_{j=1}^{k} \left(y_{ij} - y_{i.} \right)^2$, n is the number of dates of measurement for a given treatment and k is the number of blocks. Weighting made it possible to take into account the heterogeneity of variances for measurements made on different dates.

Step 2

Estimation of the parameters of Eqs. 2–5. The method used to estimate the parameters $\mu^{(1)}, \mu^{(2)}, \alpha_1^{(1)}, ..., \alpha_n^{(1)}, \alpha_1^{(2)}, ..., \alpha_n^{(2)}$ depends on the equations for the model concerned. If linear functions (2) and (3) are used, the parameters $\mu^{(1)}, \mu^{(2)}, \alpha_1^{(1)}, ..., \alpha_n^{(1)}, \alpha_1^{(2)}, ..., \alpha_n^{(2)}$ are estimated by the least squares method, using the values of C_1

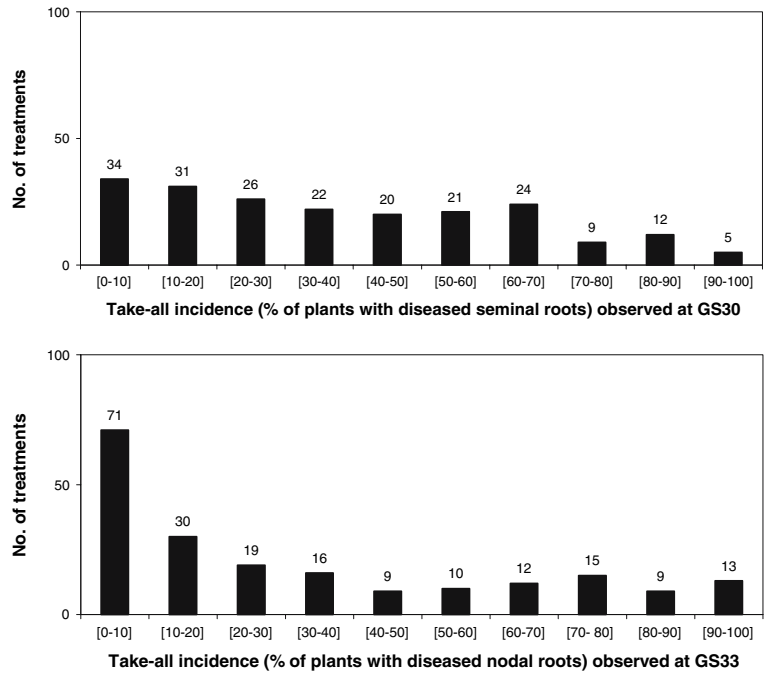

Fig. 1 Frequency distribution for take-all incidence measurements

and C_2 obtained in the first step as observations. This method is applied using the linear model (GLM) procedure of SAS Institute Inc.(1989). Box and Cox transformation (Box et al. 1978) is carried out to estimate the parameters linking C_1 to the input variables, to take into account heterogeneity in the variances of the estimators obtained in step 1. This transformation is not required for C_2. If the multiplicative functions (4) and (5) are used, the method of Colbach et al. (1999) is applied. Relationships are first linearised by logarithmic transformation and the parameters $\mu^{(1)}, \mu^{(2)}, \alpha_1^{(1)}, ..., \alpha_n^{(1)}, \alpha_1^{(2)}, ..., \alpha_n^{(2)}$ are then estimated with the GLM procedure of SAS, using the values of C_1 and C_2 obtained in step 1 as observations. Alternatively, the parameters of multiplicative functions may be estimated directly, using an iterative non-linear regression algorithm. However, the application of such an algorithm to a model including a large number of parameters often results in convergence problems.

Estimation of the parameters of static models

The parameters $\mu, \alpha_1, ..., \alpha_n$ of static models are estimated in a single step, directly from disease incidence measured at GS32-33. If the linear relationship (6) is used, the parameters are estimated by the least squares method, after arcsine transformation of the observations to better meet the homogeneity of variance and normality assumptions, e.g. Younger (1998). If the multiplicative relationship (7) is used, the parameters are estimated by the least squares method after linearization by logarithmic transformation. In all cases, the GLM procedure of SAS was used.

Selection of the input variables

Not all the variables described above necessarily have a significant effect. We used two selection procedures. The first, the 'bibliographic' method, was based on the results obtained in published studies. The aim of this method was to identify variables for which a significant effect on disease development had been reported in the literature. The parameters associated with these variables were estimated from our data and the variables were included in the models only if the corresponding parameter values were consistent with the effects reported in the literature. The second

procedure involved two steps: the selection of variables from previous studies as described above, followed by manual backward selection based on a series of Fisher's tests carried out with a significance threshold of 5%. Only significant variables were selected.

Names and characteristics of the models

We defined 16 models on the basis of the equations, input variables and selection methods described above (Table 3). Eight of these models were dynamic and were based on Eq. 1. Four of these models used linear relationships between C_1, C_2 and input variables, with the other four using multiplicative relationships. The other eight models were static and based on Eq. 6, which is linear, or Eq. 7, which is multiplicative. Particular attention was paid to the variable 'Fsem30' because this variable requires disease incidence measurement during the crop cycle, which may defer the use of the model until after sowing. We therefore developed models with and without the inclusion of this variable. Finally, eight of the 16 models described in Table 3 are based on 'bibliographic' selection and the others are based on selection by means of statistical tests.

Methods for evaluating models

The aim of this evaluation was to analyse the quality of adjustment of the models to the data and to evaluate their predictive value. For each of the 16 models, several evaluation criteria were estimated from measurements of disease incidence at GS32-33. The coefficient of determination was calculated as follows $R^2 = 1 - \frac{\sum_{l=1}^{N}\left(y_l - y_l^{(p)}\right)^2}{\sum_{l=1}^{N}\left(y_l - y_.\right)^2}$ where y_l is the observed disease incidence at GS32-33 in the lth treatment (averaged over blocks), $y_l^{(p)}$ is the disease incidence predicted by the model, $y_.$ is the mean of all observations and N is the total number of observations. The mean squared error (MSE) was calculated as follows: $\mathrm{MSE} = \frac{1}{N}\sum_{l=1}^{N}\left(y_l - y_l^{(p)}\right)^2$. The root mean squared error (RMSE) was calculated as $\mathrm{RMSE}(\%) = \sqrt{\mathrm{MSE}}$ and the relative root mean squared error (RRMSE) was calculated as $\mathrm{RRMSE} = \frac{\sqrt{\mathrm{MSE}}}{y_.}$. MSE, RMSE and RRMSE can be used to assess the quality of adjustment of the model to the data. The closer R^2 is to one and the smaller the values obtained for MSE, RMSE and RRMSE, the better the adjustment of the

Table 3 Characteristics of the 16 models

	Name	Type	Equation	Fsem30[z]	Selection
1	DynL0$^-$	Dynamic	Linear	Absent	Bibliographic
2	DynL0$^+$	Dynamic	Linear	Absent	Statistical + Bibliographic
3	DynL1$^-$	Dynamic	Linear	Present	Bibliographic
4	DynL1$^+$	Dynamic	Linear	Present	Statistical + Bibliographic
5	DynM0$^-$	Dynamic	Multiplicative	Absent	Bibliographic
6	DynM0$^+$	Dynamic	Multiplicative	Absent	Statistical + Bibliographic
7	DynM1$^-$	Dynamic	Multiplicative	Present	Bibliographic
8	DynM1$^+$	Dynamic	Multiplicative	Present	Statistical + Bibliographic
9	StaL0$^-$	Static	Linear	Absent	Bibliographic
10	StaL0$^+$	Static	Linear	Absent	Statistical + Bibliographic
11	StaL1$^-$	Static	Linear	Present	Bibliographic
12	StaL1$^+$	Static	Linear	Present	Statistical + Bibliographic
13	StaM0$^-$	Static	Multiplicative	Absent	Bibliographic
14	StaM0$^+$	Static	Multiplicative	Absent	Statistical + Bibliographic
15	StaM1$^-$	Static	Multiplicative	Present	Bibliographic
16	StaM1$^+$	Static	Multiplicative	Present	Statistical + Bibliographic

[z] the variable Fsem30 represents the incidence of the disease assessed at an early wheat growth stage, corresponding to GS30 on Zadoks growth scale (Zadoks et al. 1974)

model to the data may be considered to be. Bias, defined as $\frac{1}{N}\sum_{l=1}^{N} y_l - \frac{1}{N}\sum_{l=1}^{N} y_l^{(p)}$, is another useful criterion, because it can detect systematic over- or underestimation by the model.

RMSE cannot be used to study the predictive quality of our models in a rigorous manner, because this criterion generally favours models with large numbers of input variables and parameters (Miller 1990). We therefore evaluated the predictive quality of our models by estimating the root mean squared error of prediction (RMSEP) by cross-validation (Wallach and Goffinet 1989). According to this method, one of the 25 trials is removed from the database and its parameters are estimated with the 24 remaining trials, with prediction of the disease incidence for the trial not included in the estimation. This procedure is repeated 24 times, such that 25 estimates are obtained, one for each trial.

Results

The values for MSE, RMSE, RRMSE, R^2, and bias for disease incidence at midstem elongation are presented in Table 4.

Performance of models not including 'Fsem30'

Dynamic models

Dynamic models with linear relationships outperformed dynamic models with multiplicative relationships for most of the criteria. The best dynamic model with linear relationships (DynL0$^+$) had an RMSEP of 34.28. The best dynamic model with multiplicative relationships (DynM0$^-$) had an RMSEP of 36.98 (Table 4). These models had RMSE values of 22.43 and 26.22, and bias values of 1.54 and 5.44, respectively.

The selection of variables based on the results of statistical tests makes it possible to include fewer variables in models, to reduce the number of parameters and to improve the precision of predictions slightly. For example, for the models DynL0$^-$ and DynL0$^+$, the RMSEP values obtained were 38.12 and 34.28, respectively and the numbers of parameters were equal to 29 and 21 respectively (Table 4). The ranges of variation for the predicted incidences were similar with all dynamic models and were found to lie within the range 0–100%.

Table 4 Statistical evaluation of the various models

	Model	Number of parameters	MSE (%)^2	RMSE (%)	RRMSE	RMSEP (%)	Bias (%)	R^2	Predicted values[x]		
									Min(%)	Max(%)	Mean (%)
									0[y]	98.75[y]	31.96[y]
1	DynL0$^-$	18 + 11 = 29[z]	460.43	21.46	0.67	38.12	1.17	0.50	4.39E–02	95.25	30.78
2	DynL0$^+$	14 + 7 = 21	503.22	22.43	0.70	34.28	1.54	0.46	4.36E–02	82.31	30.41
3	DynM0$^-$	17 + 10 = 27	687.41	26.22	0.82	36.98	5.44	0.26	6.77E–03	97.94	26.52
4	DynM0$^+$	15 + 8 = 23	722.47	26.88	0.84	39.68	5.96	0.22	6.96E–03	93.80	26.00
5	StaL0$^-$	19	385.25	19.63	0.61	41.26	0.41	0.58	0.005	82.76	31.54
6	StaL0$^+$	16	409.79	20.24	0.63	32.07	0.64	0.56	0.003	82.74	31.32
7	StaM0$^-$	19	443.70	21.06	0.66	40.10	7.08	0.52	–0.35	88.15	24.87
8	StaM0$^+$	17	453.69	21.30	0.67	33.18	7.15	0.51	–0.24	86.22	24.81
9	DynL1$^-$	17 + 11 = 28	164.15	12.81	0.40	16.95	0.54	0.82	3.54E–02	99.28	31.42
10	DynL1$^+$	15 + 8 = 23	180.17	13.42	0.42	16.34	0.48	0.81	3.30E–02	98.94	31.48
11	DynM1$^-$	17 + 12 = 29	439.13	20.96	0.66	23.23	4.53	0.52	4.38E–03	99.34	27.43
12	DynM1$^+$	16 + 8 = 24	447.97	21.17	0.66	22.83	4.87	0.52	5.71E–03	98.36	27.08
13	StaL1$^-$	20	125.96	11.22	0.35	19.38	0.24	0.86	0.01	95.09	31.72
14	StaL1$^+$	17	126.23	11.24	0.35	16.43	0.20	0.86	0.02	94.60	31.76
15	StaM1$^-$	20	315.80	17.77	0.56	65.93	2.96	0.66	–0.39	162.64	29.00
16	StaM1$^+$	17	353.11	18.79	0.59	58.22	2.82	0.62	–0.33	155.39	29.13

[x] predicted values of take-all incidence (%) measured on nodal roots at GS33

[y] Min, max and mean values of take-all incidence (%) on nodal roots at GS33 calculated from the observed values of the database

[z] number of parameters in submodel C_1 + number of parameters in submodel C_2 = Total number of parameters

Static models

As for dynamic models, the MSE, RMSE and RMSEP values obtained for static models were lower for linear than for multiplicative functions (Table 4). The best linear static model ($StaL0^+$) had an RMSEP of 32.07. The best multiplicative static model ($StaM0^+$) had an RMSEP of 33.18. These models had RMSE values of 20.24 and 21.30, and biases of 0.64 and 7.15, respectively.

The selection of variables on the basis of statistical tests again slightly decreased the RMSEP, thereby increasing the precision of predictions. For example, the RMSEP values of the linear models $StaL0^-$ and $StaL0^+$ were 41.26 and 32.07, respectively (Table 4).

Comparison of static and dynamic models

The lowest RMSEP value was obtained with the dynamic model $DynL0^+$ (RMSEP = 34.3). The $StaL0^+$ model was the static model with the lowest RMSEP value (RMSEP = 32.1) and therefore the highest predictive accuracy. The RMSEP values of the two models are not very different. These models also have similar R^2 values (0.46, 0.56) and biases (1.54, 0.64), resulting in realistic predictions in both cases (Table 4). We can therefore conclude that these two models have comparable performances.

Performance of the models including 'Fsem30'

Taking into account 'Fsem30' greatly improved predictive capacity, reducing RMSEP and increasing R^2 in all models except multiplicative static models: the RMSEP of the $DynL0^+$ model was 34.28, whereas that of the $DynL1^+$ model was 16.34. These models had RMSE values of 22.43 and 13.42, and biases of 1.54 and 0.48, respectively. The RMSEP values for the $StaL0^+$ and $StaL1^+$ models were 32.07 and 16.43, respectively. These models had RMSE of 20.24 and 11.24, and biases of 0.64 and 0.20, respectively. The use of Fsem30 also improved the RMSEP of the dynamic models with multiplicative relationships; $DynM0^+$ had an RMSEP of 39.68, whereas the RMSEP of the $DynM1^+$ model was 22.83. These two models had RMSE values of 26.88 and 21.17, and bias values of 5.96 and 4.87, respectively.

In models not including 'Fsem30', disease levels were often underestimated in cases of major attack and overestimated in cases of low-level attack (Figs. 2b, d; 3b and d).

The ranges of variation for the predicted incidences were similar with all dynamic models and were found to lie within the range 0–100%. For the static models including 'Fsem30', the predicted incidences were not in the range 0–100% (Table 4): values exceeding 100% were predicted in some cases.

The poor performances of dynamic models not including 'Fsem30' are due to the poor prediction of C_1 (Fig. 4). The R^2 values obtained for C_1 are higher for $DynL1^+$ and $DynM1^+$ than for $DynL0^+$ and $DynM0^+$. By contrast, the R^2 values obtained for C_2 were similar for models including and not including 'Fsem30' (not shown).

The lowest RMSEP values were obtained with the dynamic model $DynL1^+$ (RMSEP = 16.34) and the static model $StaL1^+$ (RMSEP = 16.43). The RMSEP values of the two models are not very different. These models also have very similar R^2 values (0.81, 0.86) and biases (0.48, 0.20), resulting in realistic predictions in both cases (Table 4). We can therefore conclude that these two models have comparable performances.

Estimated values of the parameters for three of the best models

The parameters of three models—$DynL1^+$, $StaL1^+$ and $StaL0^+$–are presented in Table 5, essentially with the aim of providing the reader with the means to use these models. The parameter linked to variable 'Fsem30' takes a positive value in all the models. Sowing density is positively correlated with disease levels at mid-stem elongation (models $StaL1^+$ and $StaL0^+$). It has no effect on the parameters C_1 and C_2 of the $DynL1^+$ model. Crop rotations that included amplifier crops were always classified among those with the highest risk of take-all, although amplifier crops were found only as the pre-previous crop in the database. If the wheat crop assessed was the third wheat crop in the rotation,

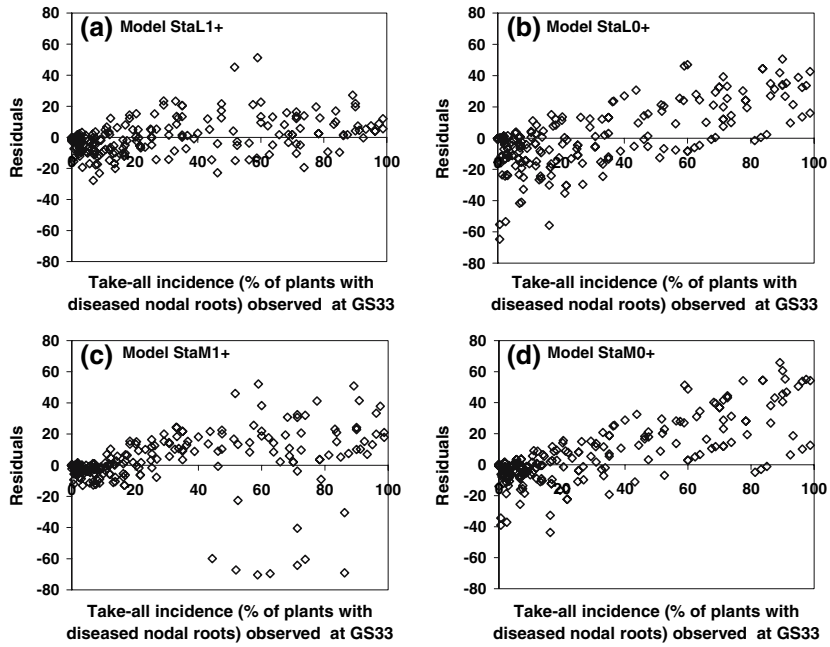

Fig. 2 Analysis of residuals (observed-predicted) as a function of the observed values for disease incidence at midstem elongation (corresponding to GS33 on Zadoks decimal scale, Zadoks et al. 1974). Four models are considered: StaL1$^+$ (**a**), StaL0$^+$ (**b**), StaM1$^+$ (**c**) and StaM0$^+$ (**d**)

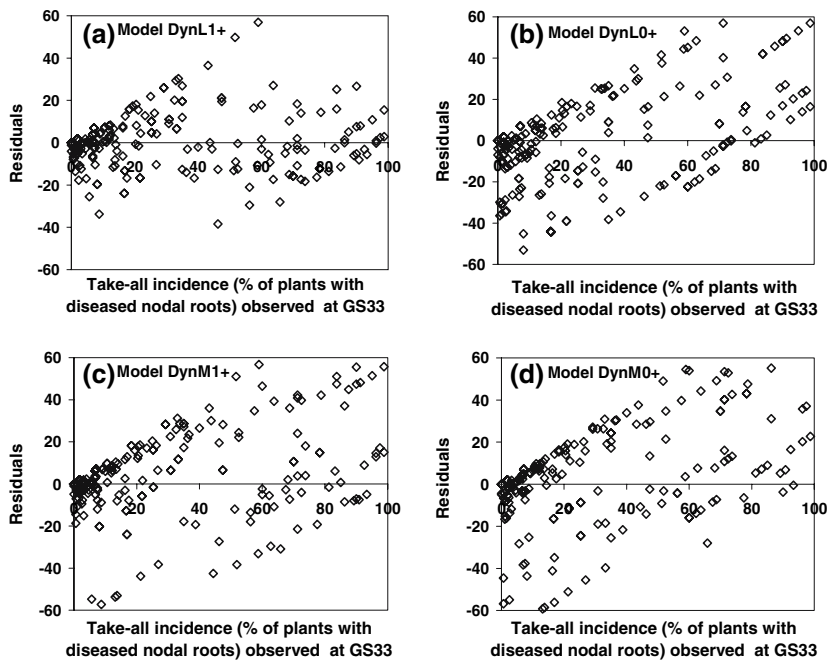

Fig. 3 Analysis of residuals (observed-predicted) as a function of the observed values for the disease incidence at midstem elongation (corresponding to GS33 on Zadoks decimal scale, Zadoks et al. 1974). Four models are considered: DynL1$^+$ (**a**), DynL0$^+$ (**b**), DynM1$^+$ (**c**) and DynM0$^+$ (**d**)

the risk of disease was similar to that for a monoculture. The risk associated with a second wheat crop did not depend on whether the pre-previous crop was a non-host or an amplifier crop. Soil texture had a significant effect in all the models. The sorting of groups of textures depended on the model, and was similar for the two static models. The lowest parameter values were obtained for sands and clays. The classification of textures was reversed for the submodels of parameters C_1 and C_2. These two parameters were strongly negatively correlated (Colbach et al. 1999). Based on C_1 and C_2, the textures of the silt and clay groups tended to favour primary

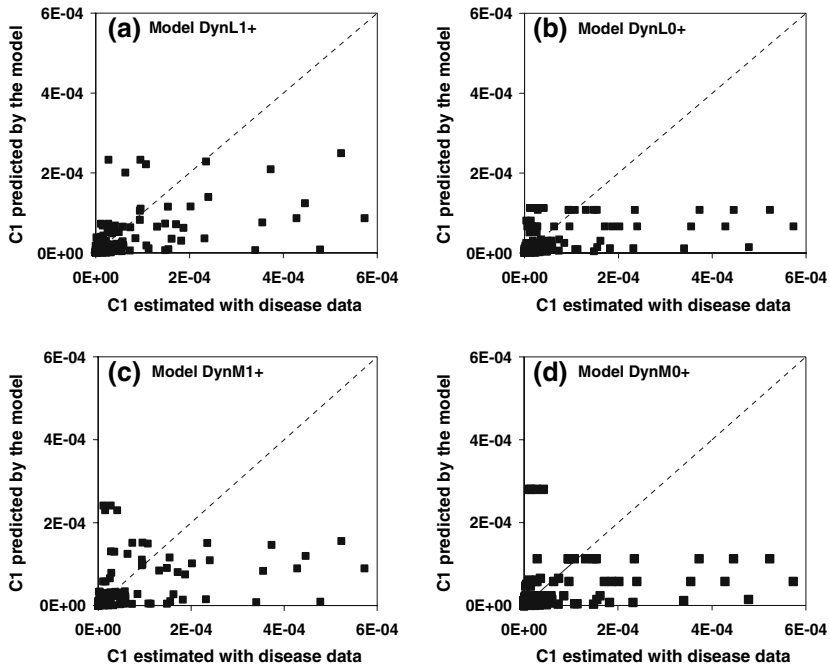

Fig. 4 Comparison of the C_1 values predicted by the models and the C_1 values estimated from the data. Four models are considered: DynL1$^+$ (**a**), DynL0$^+$ (**b**), DynM1$^+$ (**c**) and DynM0$^+$ (**d**)

infections, whereas secondary infections seemed to be favoured by sandy soils and calcareous clays. Depending on the model considered, certain climatic variables had a significant effect on disease levels. The P1 rainfall period (beginning on the 20th day after sowing and lasting 19 days) had a significant effect in all models. It decreased C_1 and increased C_2 and disease incidence during midstem elongation (StaL1$^+$ model). With StaL0$^+$, disease incidence increased with cumulative temperature. The variable linked to nitrogen was not retained in the DynL1$^+$ model, but had a significant effect in the StaL1$^+$ and StaL0$^+$ models. The parameters of the two static models indicated that an increase in ammonium application was associated with a decrease in disease levels. Fungicide application had a significant effect on disease levels in StaL0$^+$ models, but not in the StaL1$^+$ and DynL1$^+$ models.

Discussion

The performance of the models we propose for simulating the incidence of take-all depends heavily on the mathematical function used and the input variables included in the model. We have shown that two pairs of models (DynL0+, StaL0+) and (DynL1$^+$, StaL1$^+$) have comparable performance accuracies. The first model of each pair is a dynamic linear model simulating disease incidence during the crop cycle. The second is a static linear model simulating incidence for one particular growth stage (GS33), from which it is possible to predict yield losses (Schoeny et al. 2001). The multiplicative relationships proposed to link the explicative variables to C_1 and C_2 and to disease incidence were not retained because their use led to larger errors of prediction with high levels of bias. The poor performances of models based on multiplicative relationships probably resulted from the use of logarithmic transformation, which tends to increase bias. The theoretical advantage of multiplicative equations is that they can be used to take into account interactions between a large number of variables. One advantage of the static models StaL0$^+$ and StaL1$^+$ is that they include only 16 and 17 parameters, respectively, whereas the DynL0$^+$ and DynL1$^+$ models include 21 and 23 parameters, respectively. This suggests that fewer data are required for estimation of the parameters of the StaL0$^+$ and StaL1$^+$ models than for estimation of those of the DynL0$^+$ and DynL1$^+$ models. This would be an advantage in situations in which few data are available. As explained above, the estimation of parameters for the DynL0$^+$ and

Table 5 Values of the parameters of the models DynL1$^+$, StaL1$^+$ and StaL0$^+$

Variable	DynL1$^+$		StaL1$^+$	StaL0$^+$
	C_1	C_2		
Intercept	3.08E–01 (2.78E–02)	2.43E–03 (7.11E–04)	–4.47E–02 (1.01E–01)	–2.64E–01 (2.16E–01)
FSEM30	1.00E–03 (1.68E–04)	2.09E–05 (5.31E–06)	1.02E–02 (5.03E–04)	–
P1	–2.09E–03 (2.19E–04)	8.47E–05 (8.20E–06)	3.23E–03 (7.44E–04)	–
P2	–	–6.25E–05 (1.35E–05)	–3.87E–03 (1.23E–03)	–
P3	–9.80E–04 (2.35E–04)	2.00E–05 (7.33E–06)	–	–
SdjT	–	–	–	6.98E–04 (1.55E–04)
N	–	–	–1.85E–03 (3.13E–04)	–2.43E–03 (5.19E–04)
DS	–	–	9.34E–04 (2.40E–04)	1.01E–03 (4.00E–04)
Fungicide				
F-	–	–	–	1.69E–01 (4.67E–02)
F^{+y}	–	–	–	0
Inoculum				
nh-h-Nt	3.98E–02 (3.09E–02)	–^{z}	–4.41E–02 (9.00E–02)	5.04E–02 (1.49E–01)
nh-h-T	7.18E–02 (1.71E–02)	-	2.11E–01 (5.09E–02)	2.36E–01 (8.18E–02)
am-h-Nt	7.93E–02 (1.69E–02)	–	2.79E–01 (5.05E–02)	4.56E–01 (8.25E–02)
am-h-T	7.27E–02 (1.55E–02)	–	1.96E–01 (4.78E–02)	2.39E–01 (7.57E–02)
h-nh-T	5.98E–02 (2.53E–02)	–	1.80E–01 (7.65E–02)	–9.39E–02 (1.22E–01)
h-h-Nt	9.34E–03 (4.01E–02)	–	–1.63E–02 (1.19E–01)	–2.23E–03 (1.97E–01)
h-h-T	3.08E–02 (1.44E–02)	–	1.18E–01 (4.59E–02)	1.38E–01 (7.45E–02)
Mono-Nt	1.43E–02 (2.03E–02)	–	8.02E–02 (6.07E–02)	2.32E–01 (9.95E–02)
Mono-T^y	0	–	0	0
TEXTURE				
Silt	7.30E–02 (2.11E–02)	–2.13E–03 (5.94E–04)	–4.13E–02 (6.53E–02)	9.26E–02 (1.02E–01)
Sandy-silty	–1.03E–01 (2.28E–02)	1.55E–03 (7.20E–04)	–3.45E–01 (6.97E–02)	–3.52E–01 (1.25E–01)
Clay	1.19E–01 (2.26E–02)	–4.02E–03 (6.30E–04)	–1.95E–01 (7.16E–02)	–1.33E–01 (1.12E–01)
Calcareous clay^y	0	0	0	0

– Variable not included in the model because not significant at 5% level

^{y} reference level used for parameter estimation

^{z} 'inoculum' is not an input variable of the C_2 model

P1, P2, P3: The three input variables linked to climate (rainfall, see Table 3)

SdjT: Cumulative degree-days (temperature base 0°C) from sowing to 31st January

Fsem30: The input variable linked to disease incidence scored at GS30 (Zadoks et al., 1974)

N: fraction of the nitrogen fertiliser applied in ammoniacal form

DS: sowing density (number of seeds m^{-2})

F–, F+: No fungicide treatment, treatment of the seeds with silthiofam

Nh, h, am, mono: correspond to the crops present in rotations: non-host crops, host crops, amplifier crops and monocultures

Nt, T: No-till, till

Only significant parameters at the $P = 0.05$ level were selected in the models

DynL1$^+$ models requires a preliminary step involving the estimation of C_1 and C_2 for each treatment. This step requires expensive experimentation, during which the incidence of the disease must be measured on several dates (disease assessments) during the crop cycle.

However, static linear models have the disadvantage of predicting the disease incidence only for a single growth stage. Conversely, dynamic models simulate disease progression over the whole crop cycle. They can be evaluated with incidence data at crop stages other than GS33, predict disease incidence at any crop stage and provide information about which chemical, cultural or biological forms of control are efficient during the infection cycle. Dynamic models can also be coupled with yield loss models, as proposed by Schoeny et al. (2001), with the development of models based on disease incidence on nodal roots at midstem elongation or

using the area under the disease progress curve (AUDPC). Finally, the possibility of predicting disease incidence at any crop stage may make it possible to modify late nitrogen fertilizer application or chemical treatments against aerial diseases during the growth of the individual crop. Statistically, all the variables of the four models DynL0$^+$, DynL1$^+$, StaL0$^+$ and StaL1$^+$ have a significant effect.

Not all the parameters associated with each variable were precisely estimated. However, we did not aim to use these values of the parameters to gain insight into development of the disease. That would require factorial experiments at appropriate sites and many studies of this type have already been carried out for take-all (Werker and Gilligan 1990). The database used here is large but unbalanced. In any case, the hierarchy of factors obtained here is similar to that obtained by Colbach et al. (1997), with a more precise estimation of the risk associated with the site. At the scale of multi-site, pluriannual databases, the variables associated with site (texture, climate, etc.) have a much greater effect on the development of take-all than do variables associated with cropping practices. These variables were highly significant and were included in all the models.

The strong effect of soil texture is consistent with the soil-borne nature of the take-all fungus and its sensitivity to oxygenation, pH and soil humidity, all of which are closely linked to soil texture. What is more surprising is that soil texture may affect C_1 and C_2 differently. As mechanisms for primary and secondary infections are different, these results would justify further experimental work to seek confirmation and explanation on the relationships between these mechanisms and soil texture.

The importance of the effect of climatic variables is hardly surprising, as epidemiologists consider climate to have a major effect on the geographic distribution of the disease (in temperate, tropical or subtropical zones), its occurrence and progression, e.g. winter cold or a lack of water in the summer may inhibit fungal activity (Hornby et al. 1998). The use of climatic variables makes it possible to take into account sowing date, rainfall, autumn temperature and regional effects of climate on take-all development, but most of the field trials were concentrated in the northern half of France. The significant effect of the P1 period on C_1 seems logical and is consistent with the work of Werker and Gilligan (1990), who indicated that very humid autumn conditions favour establishment of the disease. The significant effect of P1 on C_2 suggests that the cycle of secondary infections begins early. Rapilly (1991), Chevalier-Gérard et al. (1994) and Colbach et al. (1999) used cumulative temperature expressed in degree-days (base 0°C) from sowing to 31 January to evaluate pre-winter infection with eyespot and other wheat disease complexes. Cumulative degree-days was a significant factor only in the StaL0$^+$ model. This is logical because the StaL1$^+$ and DynL1$^+$ models include the input variable 'Fsem30', which already accounts for the effect of cumulative degree-days on infection at this timepoint.

The values of the parameters obtained for the variable 'inoculum' with the three models presented above are largely consistent with published results (Colbach et al. 1994). The effect of the variable 'inoculum' on C_2 was not investigated because we assumed that the amount of initial inoculum would not affect the parameter linked to secondary infections (Colbach et al. 1999). The observed inconsistencies, such as the higher risk associated with the 'non-host/host/tillage' combination than with the 'host/non-host/tillage' combination (StaL0$^+$ model), or the lower risk of the 'host/host/tillage' combination than of the 'host/host/no tillage' combination (all models), may be attributed to differences in the numbers of treatments for the different levels of the variable, leading to poor estimation of the impact of a particular crop rotation × soil tillage combination. This is the case, in the StaL0$^+$ model, for the levels 'non-host/host/no tillage', 'host/non-host/tillage' or 'host/host/no tillage'. The standard deviations of the corresponding parameter estimates are higher than the estimated values. Similar disease incidence obtained for a third consecutive wheat crop and for a wheat monoculture may be due to the fact that only three consecutive years are taken into account in the model and to the low representation of monocultures in the data base, both aspects giving no or poor account of the build-up of the antagonistic

microflora through wheat monoculture which is responsible for take-all decline.

Sowing density had an effect only in the static models, in which take-all is assessed at a relatively early stage (GS33), consistent with the results reported by Colbach et al. (1997). Conversely, the analysis of C_1 and C_2 parameters from the disease progress model, at sites favouring disease development, revealed a positive correlation between plant density and the parameter C_1 (relating to primary infection) in Colbach's study, but the effect of plant density on C_2 was more variable, and depended on the earliness of the secondary infection.

Many studies have demonstrated a very strong effect of mineral fertilisation on disease levels (Reis et al. 1982) with nitrogen fertilisation studied in particular detail (Lucas et al. 1997). Unlike the dynamic model for take-all developed by Colbach et al. (1997), which showed that an increase in available nitrogen led to a decrease in C_2, with no effect on C_1, the absence of an effect of nitrogen in the DynL1$^+$ model probably reflects the lesser importance of mechanisms responsible for the effects of nitrogen on the disease at the crop cycle level than of effects linked to the site characteristics (soil, climate).

The fungicide effect was significant only if 'Fsem30' was not introduced into the model. Indeed, Schoeny and Lucas (1999) reported that fungicide treatment significantly delayed epidemics (C_1 parameter significantly decreased). These results were confirmed by Bailey et al. (2005), who showed that this fungicide can control primary infections.

The results in Table 4 demonstrate the importance of the variable linked to early disease assessment. Early disease assessment on seminal roots is positively correlated with disease levels on nodal roots during later stages. This would seem to be consistent for a disease caused by a soil-borne fungus. The variable 'Fsem30' being itself an early disease measure, is subject to the effects of soil, climatic conditions during the autumn, crop rotation and the treatment of seeds with fungicide. As for the fungicide effect, other variables, such as 'cumulative degree-days from sowing to 31st January' (sowing date effect), known to affect disease epidemics were significant only if 'Fsem30' was not introduced into the model.

Taking the variable 'Fsem30' into account increased the accuracy of the models, but reduced the predictive value. The DynL1$^+$ and StaL1$^+$ models cannot be used to influence pre-sowing choices, such as fungicide application. Although acquired after sowing, the 'Fsem30' variable can be used to predict disease during the course of the crop cycle and to guide tactical decisions. Rapid visual scoring at an early stage of the crop cycle would not be expensive and would enable the adviser or farmer to identify early take-all attacks in the plot concerned. The input variable 'Fsem30' is relevant to field inoculum potential and to the initiation of an epidemic (approximation of the rate of primary infection and of former climatic and cropping conditions). In the long term, early quantification of the initial level of Ggt in the soil, which is not adequately taken into account by considering crop rotation alone, based on new molecular biological techniques (DNA analyses) (Herdina and Roget, 2000), should make it possible for models to predict disease kinetics at any later stage. This would be particularly useful for estimating the corresponding yield losses and determining the level of subsequent investment and intervention (late nitrogen fertilizer, chemical treatments against aerial diseases, etc.) during the current crop cycle, to limit losses.

The StaL0$^+$ model has a high RMSEP value but a low bias and significant variables. It may be possible to use this model, which does not include 'Fsem30', to determine the value of changes to the cropping system (rotation, soil tillage, sowing date and fungicidal seed treatment) before sowing, based on climatic series, as no post-sowing input data are required for this model.

More severe take-all disease develops between GS30 and GS33 than between sowing and GS30. Nevertheless, although for the sake of convenience, we used disease incidence measured on seminal roots at growth stage GS30-31 (information most frequently available in the database), it is possible and probably more useful for the farmer to assess disease at an earlier growth stage (e.g. GS15). A significant correlation was observed between disease incidence at GS30 and GS15; $R^2 = 0.63$ (Ennaifar 2006).

In conclusion, the models of take-all development presented here are simple and easy to use, because all the input variables are common to most surveys. It should be possible to incorporate them as disease modules, into more complex models such as crop simulation models, most of which also use these variables, or to link them to yield loss models (Schoeny et al. 2001).

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