Modeling of Take-All Epidemics to Evaluate the Efficacy of a New Seed-Treatment Fungicide on Wheat

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ABSTRACT

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Take-all, caused by *Gaeumannomyces graminis* var. *tritici*, is a damaging disease of wheat that remains difficult to control. The efficacy of an experimental fungicide, applied as a seed treatment, was evaluated in five naturally infested field experiments conducted during three cropping seasons. Plants were sampled and assessed for take-all incidence and severity at different growth stages. Nonlinear models expressing disease variables as a function of degree-days were fitted to the observed data. The incidence equation involved two parameters reflecting the importance of

primary and secondary infection cycles. The earliness of infection was identified as an important variable to interpret the effects of the fungicide. In an early epidemic, the fungicide significantly reduced take-all incidence during all or most of the cropping season, whereas in late epidemics, it provided only moderate reductions of incidence. The seed treatment reduced incidence by delaying the primary infection cycle. The fungicide significantly reduced severity during the whole epidemic. It appeared more efficient in limiting root-to-root spread than in slowing down the extension of necrosis on diseased roots.

Additional keywords: disease progress curve, point of inflection, *Triticum aestivum*.

Take-all of winter wheat (*Triticum aestivum* L.), caused by the soilborne fungus *Gaeumannomyces graminis* (Sacc.) Arx & D. Olivier var. *tritici* J. Walker, remains a damaging disease in all cereal-producing areas (27). Cultural practices such as crop rotation (16,18,29), prolonged wheat monoculture (15,40,46), delayed sowing (6,17,26), and fertilization with ammoniacal forms of nitrogen (17,32,33,39,43) have shown partial efficacy in limiting take-all risk, but none of them are entirely satisfactory for growers. Indeed, efficacy of crop rotation depends on the introduction of alternative crops that are, in some areas, limited in choice and can result in reduced economic returns. Although long-term wheat monoculture can lead to biological suppression of the disease, known as take-all decline (22,25,41,42), its short-term economic impact can be severe. Delayed sowing increases soil erosion and risks for nitrate leaching, and sometimes reduces yields (19,36). Moreover, no commercial cultivar showing resistance or tolerance is available yet.

Research has, so far, been unsuccessful in providing consistent, effective, and practical chemical control measures (7). Drenching with benomyl was efficient against shallow inoculum in pot experiments (2) but mostly ineffective against deeper inoculum (9) and gave inconsistent results under natural conditions (4,28). In naturally infested fields, soil treatment by incorporation of benomyl showed some efficacy but was unsuitable for commercial cropping areas owing to the high rate needed (35 kg/ha) and the difficulty of application (3). The sterol biosynthesis-inhibiting fungicide nuarimol was incorporated at a much lower concentration than benomyl, but it permitted only a partial control of take-all (5,8,28). Seed treatments involving other sterol biosynthesis-inhibiting fungicides were concurrently investigated. In artificially infested field experiments, triadimefon gave inconsistent results (1,20). Seed treatment with triadimenol reduced autumn infections in an inoculated field experiment, resulting in a delayed development of the disease and

a reduced "whitehead" symptoms percentage (11); an experimental seed-coating method improved the performance of this fungicide in artificially infested field experiments (31). However, triadimenol efficacy was questioned under natural conditions (6,38).

Fungicide potential has often been evaluated in bioassays or in artificially infested fields. Those conditions have often led to promising effects that were, unfortunately, not repeatable in naturally infested fields. Moreover, the few experiments conducted to investigate the fungicide efficacy under natural conditions have been unable to take into account the effect of the fungicide on the whole disease progress curve, as they were usually based on a limited number of sampling occasions during the cropping season.

Preliminary field experiments with an experimental seed-treatment fungicide from Monsanto (Monsanto Europe S.A., Brussels, Belgium) indicated promise for reduction of take-all (30). In this paper, we determine the potential of a sister compound to control take-all under natural conditions by characterizing its effects on disease incidence and severity at different sampling dates throughout the cropping season. We also propose methods for evaluating the effects of such a fungicide on a multiexperiment basis, taking into account aspects of disease epidemiology.

MATERIALS AND METHODS

Field experiments. Experiments were conducted during three cropping seasons (1994–1995, 1995–1996, and 1996–1997) on naturally infested fields located at Le Rheu and Pacé, Brittany, western France (Table 1). The two locations, approximately 5 km apart, presented a similar soil texture (15% loam, 70% silt, and 15% sand). Fields were chosen to provide a high take-all risk: depending on the experiment site, one or two host crops (winter wheat or winter barley) preceded the assessed winter wheat (Table 1). Therefore, three experiments (LR295, LR296, and P297) were second wheat crops, and two experiments (LR396 and P397) were third wheat crops.

The efficacy of an experimental fungicide, MON65500 (*N*-allyl-4,5-dimethyl-2-trimethylsilylthiophene-3-carboxamide) (10), applied as a seed treatment, was investigated in randomized complete block

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designs consisting of three to six blocks according to experiments (Table 1). Experimental treatments comprised a control (untreated seeds) and two fungicide rates (25 and 50 g a.i./100 kg of seed in all experiments except P397; 12.5 and 25 g a.i./100 kg of seed in P397). Plot size differed among experiments (Table 1).

Seed beds were prepared by plowing and rotary harrowing. Seeds (cv. Forby in LR295 and cv. Soissons in all other experiments) were sown at 330 seeds per m². Fertilizer (0:18:24 [N-P-K] in LR295 and 0:14:21 [N-P-K] in all other experiments) was applied before plowing at 550 to 600 kg/ha. Ammonium nitrate (33.5% N) was applied according to the predictive balance sheet method (37) to provide a nonlimiting amount of nitrogen for plant growth. Rates ranged from 100 kg/ha in two spring applications (growth stage [GS] 23 and GS31 according to Zadoks et al. [48]) in P397 to 180 to 195 kg/ha in three spring applications (GS23, GS31, and GS33) in all other experiments. Other nonexperimental chemical treatments comprised herbicides (including fluroxypyr or isoxaben) applied shortly after sowing and in spring if needed (LR295); fungicides (including cyprodinil or epoxiconazole against eyespot and propiconazole + fenpropimorph or epoxiconazole against foliar diseases) applied at the beginning of stem elongation and at flowering, respectively; and an insecticide (including tau-fluvalinate) applied in autumn (LR295, P297, and P397).

Sampling and disease assessment. To study the temporal progress of the take-all epidemic, plants were sampled and assessed throughout the cropping season (Table 2). In all experiments except LR295, four samples of 25 cm × 2 rows were taken from each plot at each sampling date; in LR295, because of the small plot size, one sample of 50 cm × 2 rows was taken per plot (Table 1). Root systems were washed free of adhering soil, and take-all was visually assessed on 10 randomly chosen plants per sample. Disease incidence was assessed as the proportion of plants with characteristic symptoms (black stelar discoloration) on nodal roots. Disease severity was assessed in two ways: (i) the proportion of diseased roots (PDR) was calculated after counting, for each plant, the total number of nodal roots and the number of these with at least one take-all lesion, whatever its length (in all experiments except LR295); and

(ii) the proportion of nodal root system with symptoms was scored according to a five-class scale (0 to 4) corresponding to no symptoms, 1 to 10%, 11 to 30%, 31 to 60%, and 61 to 100% of the root system necrotic, respectively (33). A weighted take-all index (TAI) was then calculated according to the formula

$$\text{TAI (\%)} = \frac{\sum_{i=0}^4 m_i n_i}{\sum_{i=0}^4 n_i}$$

in which n_i is the number of plants in class i , and m_i is the mean of class i (%). This calculation leads to a 80% maximum TAI. In contrast to PDR, TAI is a severity variable that takes the necrosis length into account and, therefore, reflects the extension of necrosis on diseased roots.

Statistical analysis and disease modeling. Incidence and severity data were first analyzed for each experiment and sampling date. The effects of the seed treatment on the disease variables were tested by analyses of variance using the general linear model (GLM) procedure of the SAS software (statistical analysis system; SAS Institute Inc., Cary, NC). Means for significant effects were compared with least difference analyses.

Take-all progress curves were determined for each experiment and treatment. The independent variable used in model fitting was thermal time expressed as cumulative degree-days (basis 0°C) since sowing. The baseline of 0°C represents the minimum air temperature for wheat growth (34).

Incidence data were fitted to a nonlinear model involving primary and secondary infections, as proposed by Colbach et al. (17) from initial work by Brassett and Gilligan (13)

$$y = \frac{1 - e^{-(c_1 + c_2)t}}{1 + \left(\frac{c_2}{c_1}\right)e^{-(c_1 + c_2)t}} \quad (1)$$

in which c_1 is the equation parameter associated with primary infections (arising from inoculum surviving saprophytically on former

TABLE 1. Description of the field experiments conducted in France in 1994 to 1997

Experiment ^y	Location	Previous crops ^z	Sowing date	Number of blocks	Plot size	Number of sampling dates	Number of samples per date	Sample size
LR295	Le Rheu	fb, ww	21 Oct. 1994	6	2.6 m × 5.5 m	6	1	50 cm × 2 rows
LR296	Le Rheu	fb, ww	2 Nov. 1995	3	6 m × 30 m	7	4	25 cm × 2 rows
LR396	Le Rheu	ww, wb	2 Nov. 1995	3	6 m × 30 m	7	4	25 cm × 2 rows
P297	Pacé	m, ww	17 Oct. 1996	4	3.9 m × 15 m	6	4	25 cm × 2 rows
P397	Pacé	ww, ww	17 Oct. 1996	4	6 m × 25 m	6	4	25 cm × 2 rows

^y Experiments were coded with the initials of the location followed by the rank of the assessed wheat crop ('2' for the second wheat crops and '3' for the third wheat crops) and the year of the experiment.

^z fb = Faba bean, m = maize, wb = winter barley, and ww = winter wheat.

TABLE 2. Correspondence between calendar dates, cumulative days, degree-days since sowing, and growth stage of plants at the sampling occasions of field experiments conducted in France in 1994 to 1997

Sampling occasion	1995				1996				1997			
	Date	Days since sowing	Degree-days since sowing ^x	Growth stage ^y	Date	Days since sowing	Degree-days since sowing ^x	Growth stage ^y	Date	Days since sowing	Degree-days since sowing ^x	Growth stage ^y
1	6 Feb.	108	992.1	22	30 Jan.	89	582.3	22	3 Mar.	137	900.1	23
2	22 Feb.	124	1,148.6	23	26 Feb.	116	696.6	23	25 Mar.	159	1,119.3	31
3	15 Mar.	145	1,305.7	30	20 Mar.	139	828.7	30	14 Apr.	179	1,325.8	33
4	4 Apr.	165	1,476.9	31	10 Apr.	160	1,001.2	31	5 May/12 May ^z	200/207 ^z	1,565.1/1,639.5 ^z	41/45 ^z
5	27 Apr.	188	1,698.6	33	30 Apr.	180	1,241.2	34	2 Jun.	228	1,962.2	65
6	30 May	221	2,142.1	65	3 Jun.	214	1,628.9	65	25 Jun.	251	2,342.1	83
7					1 Jul.	242	2,114.9	83				

^x Basis 0°C.

^y Decimal wheat growth stage according to Zadoks et al. (48). 00 to 09 = Germination; 10 to 19 = seedling and main stem elongation; 20 to 29 = tillering; 30 to 39 = stem elongation; 40 to 49 = booting; 50 to 59 = inflorescence emergence; 60 to 69 = flowering; 70 to 87 = grain filling; and 90 to 99 = ripening.

^z The first value corresponds to P397, and the second one to P297.

host crop residues), c_2 is the equation parameter associated with secondary infections (arising from existing infections), and t is the sum of degree-days since sowing. This equation is a continuous function with an asymmetrical sigmoid shape and ranges from $y =$

0 at $t = 0$ to $y = 1$ as t tends to infinity. It was obtained by integrating and reparameterizing the equation, giving the rate of disease incidence increase as a function of time (dy/dt)

$$\frac{dy}{dt} = (k_1 p + k_2 n y)(1 - y) \quad (2)$$

in which k_1 is the rate of primary infection, k_2 is the rate of secondary infection, p is the amount of soil inoculum, and n is the total number of plants. These parameters were assumed to remain constant with time, and c_1 and c_2 were defined as $c_1 = k_1 p$ and $c_2 = k_2 n$.

Severity data were fitted to the three-parameter Weibull model (44)

$$y = k(1 - e^{-\alpha t^\beta}) \quad (3)$$

in which k is the asymptotic level of the disease variable, α and β are the two other equation parameters, and t is the sum of degree-days since sowing. This equation has already been used to describe the temporal progress of botanical epidemics (35). It ranges from $y = 0$ at $t = 0$ to $y = k$ as t tends to infinity with $k = 1$ in the PDR equation and $k = 0.8$ in the TAI equation. Although the biological interpretation of the parameters is difficult, it makes allowance for essential prerequisites for epidemic development. Indeed, initial and final conditions (a nil value at sowing date and a maximal value as time tends to infinity or to a maximum with respect to cropping duration, respectively) are not always respected by other nonlinear models (14). Since equation parameters have no biological interpretation, we chose the time required for reaching the point of inflection of the progress curves as an indicator of the seed-treatment efficacy. The point of inflection is a mathematical feature corresponding to the annulment of the second derivative and marking the stage in the epidemic when a progressive increase in the rate of change of the disease variable is halted (13). It occurs when

$$t = \left(\frac{\beta - 1}{\alpha \beta} \right)^{1/\beta}$$

Models were fitted to experimental data using the nonlinear model (NLIN) procedure of SAS; after a series of iterations, adjustments estimated the parameters for each experimental treatment.

Analyses of variance were performed to test the effects of seed treatment on incidence parameters c_1 and c_2 and on the time required for reaching the point of inflection of severity curves. This was made possible by grouping data from all experiments except P397, in which the highest rate of fungicide was not investigated, and by verifying the absence of experiment $\times$ fungicide interaction. If needed, a Box and Cox transformation (12) was used to homogenize variances: for example, if the linear regression of $\ln[\text{variance}(\text{parameter})] = a + b \ln(\text{parameter})$ was significant, parameter c_i was transformed as follows.

$$c_{i \text{ transformed}} = c_i^{1-b/2}$$

RESULTS

Take-all epidemics. Take-all developed early in experiment LR295: 26% of the plants were diseased in the untreated plots at the beginning of tillering (108 days since sowing [dss]) and 100% were diseased at flowering (221 dss) (Fig. 1A). Roots were only slightly affected during tillering (108 to 124 dss); TAI then increased regularly during stem elongation and reached 59% at flowering (Fig. 1C).

The disease onset occurred later in LR296 and LR396 compared with LR295: few plants were diseased at the beginning of stem elongation (160 dss) (Fig. 1A), and on that date, PDR and TAI were nearly nil (Fig. 1B and C). Incidence increased rapidly thereafter, and almost all plants were diseased at grain filling (242 dss) (Fig. 1A), but less than 50% of the roots were diseased and TAI was less than 50% (Fig. 1B and C). Take-all epidemics were similar

Fig. 1. Progress of take-all for three disease assessment methods in untreated plots of field experiments conducted in France in 1994 to 1997. **A**, Disease incidence; **B**, proportion of diseased roots; and **C**, take-all index (TAI).

in the two experiments, but there was slightly more disease in the second wheat crop (LR296) than in the third wheat crop (LR396). This was particularly true for TAI values (Fig. 1C).

During the 1996–1997 cropping season, take-all epidemics were different in the two experiments. In P297, disease developed at stem elongation (159 dss) and all plants were diseased at flowering (228 dss) (Fig. 1A). On that date, 52% of the roots had characteristic take-all symptoms and TAI was 32% (Fig. 1B and C). In P397, take-all onset occurred at tillering (137 dss). Disease developed earlier and spread more rapidly than in P297, since almost all plants were diseased before ear emergence (200 dss) (Fig. 1A). At flowering, 78% of the roots were diseased and TAI was 61% (Fig. 1B and C).

On the basis of the earliness of the disease onset and of the progress of PDR and TAI, we arbitrarily classified the different epidemics as follows: early and severe (LR295), late and severe (P397), late and moderate (P297), and late and slight (LR296 and LR396).

Effect of seed treatment on disease incidence. In LR295, the lowest rate of fungicide (25 g a.i./100 kg of seed) significantly reduced take-all incidence until after the beginning of stem elongation (GS31) (Table 3). The highest rate of fungicide (50 g a.i./100 kg of seed) maintained disease incidence at a significantly lower level than in the control through GS65 (Table 3). Establishment and progress of the pathogen, as measured by disease incidence, were delayed by the highest rate of fungicide for 3 to 5 weeks throughout

TABLE 3. Effect of an experimental seed treatment on take-all incidence (proportion of diseased plants) of wheat in field experiments in France in 1994 to 1997[†]

Experiment	Seed treatment (a.i./100 kg seed)	Growth stage [‡]							
		22	23	30	31	33–34	41–45	65	83
LR295 ^v	Untreated	26 a	53 a	66 a	78 a	91 a	... ^w	100 a	...
	Treated (25 g)	10 b	21 b	42 b	54 b	89 ab	...	100 a	...
	Treated (50 g)	4 b	16 b	33 b	48 b	78 b	...	88 b	...
LR296 ^x	Untreated	1 a	1 a	1 a	11 a ^y	47 a	...	72 a	95 a
	Treated (25 g)	0 a	0 a	3 a	7 ab	25 b	...	54 a	...
	Treated (50 g)	0 a	0 a	2 a	2 b	25 b	...	70 a	96 a
LR396 ^x	Untreated	1 a	0 a	1 a	5 a	29 a ^y	...	63 a	96 a
	Treated (25 g)	0 a	0 a	0 a	3 a	18 ab	...	46 a	...
	Treated (50 g)	0 a	0 a	0 a	3 a	7 b	...	43 a	83 b
P297 ^z	Untreated	...	1 a	...	3 a	33 a	53 a	96 a	100 a
	Treated (25 g)	...	0 a	...	3 a	17 ab	—	87 a	96 a
	Treated (50 g)	...	0 a	...	1 a	8 b	46 a	84 a	97 a
P397 ^z	Untreated	...	8 a	...	36 a	71 a ^y	98 a	100 a	100 a
	Treated (12.5 g)	...	5 a	...	30 a	63 ab	90 b	100 a	100 a
	Treated (25 g)	...	3 a	...	25 a	52 b	82 c	100 a	100 a

[†] For each experiment, means within a column followed by the same letter are not significantly different at $P = 0.05$ according to Fisher's least significant difference test.

[‡] Decimal wheat growth stage according to Zadoks et al. (48).

^v Twenty plants per plot were assessed for characteristic take-all symptoms. Incidence values are means of six replicates.

^w ... = No assessment.

^x Forty plants per plot were assessed for take-all symptoms. Incidence values are means of three replicates.

^y This effect was significant at $P = 0.1$.

^z Forty plants per plot were assessed for take-all symptoms. Incidence values are means of four replicates.

TABLE 4. Effect of an experimental seed treatment on the take-all index (proportion of necrotic nodal root system) of wheat in field experiments in France in 1994 to 1997[†]

Experiment	Seed treatment (a.i./100 kg seed)	Growth stage [‡]							
		22	23	30	31	33–34	41–45	65	83
LR295 ^v	Untreated	2 a	5 a	12 a	19 a	34 a	... ^w	59 a	...
	Treated (25 g)	1 b	1 b	3 b	4 b	14 b	...	35 ab	...
	Treated (50 g)	0 b	1 b	2 b	5 b	5 b	...	14 b	...
LR296 ^x	Untreated	0 a	0 a	0 a	1 a	3 a	...	16 a ^y	46 a
	Treated (25 g)	0 a	0 a	0 a	0 a	1 b	...	5 b	...
	Treated (50 g)	0 a	0 a	0 a	0 a	1 b	...	5 b	25 a
LR396 ^x	Untreated	0 a	0 a	0 a	0 a	2 a ^y	...	6 a	31 a
	Treated (25 g)	0 a	0 a	0 a	0 a	1 ab	...	4 a	...
	Treated (50 g)	0 a	0 a	0 a	0 a	0 b	...	2 a	11 b
P297 ^z	Untreated	...	0 a	...	0 a	2 a	7 a	32 a	56 a
	Treated (25 g)	...	0 a	...	0 a	1 ab	...	15 ab	35 ab
	Treated (50 g)	...	0 a	...	0 a	0 b	2 a	6 b	14 b
P397 ^z	Untreated	...	1 a	...	2 a	11 a	33 a	61 a	72 a ^y
	Treated (12.5 g)	...	0 a	...	2 a	5 b	23 b	37 b	63 b
	Treated (25 g)	...	0 a	...	1 a	4 b	9 c	35 b	65 b

[†] For each experiment, means within a column followed by the same letter are not significantly different at $P = 0.05$ according to Fisher's least significant difference test.

[‡] Decimal wheat growth stage according to Zadoks et al. (48).

^v Nodal root systems of 20 plants per plot were examined for take-all symptoms. Percentage values are means of six replicates.

^w ... = No assessment.

^x Nodal root systems of 40 plants per plot were examined for take-all symptoms. Percentage values are means of three replicates.

^y This effect was significant at $P = 0.1$.

^z Nodal root systems of 40 plants per plot were examined for take-all symptoms. Percentage values are means of four replicates.

the cropping season. In LR296, the high rate of fungicide significantly reduced disease incidence during part of stem elongation (GS31 to GS34). The effect was less marked afterwards, as treated and untreated plots no longer differed significantly (Table 3). In LR396, the highest rate of fungicide also reduced incidence at stem elongation (GS34), and its effect was again noticeable at grain filling (GS83) (Table 3). In P297, the proportion of diseased plants in the plots treated at the highest rate of fungicide was significantly reduced at midstem elongation (GS33), but subsequent reductions were not significant (Table 3). In P397, disease incidence was significantly lower in treated plots than in untreated plots at GS41, even at the lowest rate of fungicide (12.5 g a.i./100 kg of seed) (Table 3).

Effect of seed treatment on disease severity. In LR295, the lowest rate of fungicide significantly reduced TAI through GS33, and this effect was prolonged through GS65 with the highest rate of fungicide (Table 4). In LR296 and LR396, TAI and PDR were usually lower in treated plots than in untreated plots until GS83 (Tables 4 and 5). In P297, the protection ensured by the highest rate of fungicide lasted through GS83 with regard to TAI and PDR (Tables 4 and 5). In P397, seed treatment significantly reduced take-all infection whatever the rate. TAI and PDR were reduced through GS83 and GS65, respectively (Tables 4 and 5).

Epidemic modeling and effect of seed treatment on disease progress curves. The equation chosen to describe an increase of take-all incidence over time fitted well to the observed data as illustrated for LR295 (Fig. 2A). The r^2 values showed that at least 92% of the variability was explained by the model in all experiments (Table 6). Parameters associated with primary (c_1) and secondary (c_2) development of take-all were successfully estimated for all experimental treatments. Considering the c_1 estimates in untreated plots, LR295 and LR296 were the most favorable sites for primary infection, P397 was the least favorable, and LR396 and P297 had intermediate levels of primary infection (Table 6). In contrast, secondary infection in untreated plots was slightly higher in P397 than in the other experiment sites, as shown by the c_2 values (Table 6). The highest rate of fungicide significantly decreased c_1 , whereas no effect of the seed treatment on c_2 was observed (Table 7).

The Weibull model was satisfactorily fitted to severity data as illustrated for LR295 (Fig. 2B); r^2 values were generally higher than 80% (Tables 8 and 9). Equation parameters were also successfully estimated for all experimental treatments, even those in which the last sampling date was missing. The seed treatment increased the time necessary to reach the point of inflection of PDR (I_{PDR}) and TAI (I_{TAI}) progress curves (Tables 8 and 9). I_{PDR} was significantly increased whatever the fungicide rate, and I_{TAI} was significantly increased with the highest rate (Table 10).

DISCUSSION

Take-all developed differently depending on experimental sites, offering a representative range of expected epidemics under natural

Fig. 2. Illustration of take-all progress curves for different rates of an experimental seed treatment in a field experiment at Le Rheu, France, in 1995. A, Disease incidence; and B, take-all index (TAI). Symbols represent observed means for each experimental treatment. Lines are fitted curves (from sowing to harvest).

TABLE 5. Effect of an experimental seed treatment on the proportion of diseased nodal roots per plant of wheat in field experiments in France in 1994 to 1997^v

Experiment	Seed treatment (a.i./100 kg seed)	Growth stage ^w							
		22	23	30	31	33–34	41–45	65	83
LR296 ^x	Untreated	0 a	0 a	0 a	1 a	5 a	... ^y	20 a	46 a
	Treated (25 g)	0 a	0 a	0 a	0 a	1 b	...	6 b	...
	Treated (50 g)	0 a	0 a	0 a	0 a	1 b	...	7 b	28 a
LR396 ^x	Untreated	0 a	0 a	0 a	0 a	2 a	...	7 a	42 a
	Treated (25 g)	0 a	0 a	0 a	0 a	2 a	...	4 a	...
	Treated (50 g)	0 a	0 a	0 a	0 a	0 a	...	3 a	18 a
P297 ^z	Untreated	...	0 a	...	0 a	3 a	12 a	52 a	75 a
	Treated (25 g)	...	0 a	...	0 a	2 ab	...	31 ab	58 ab
	Treated (50 g)	...	0 a	...	0 a	0 b	7 a	14 b	30 b
P397 ^z	Untreated	...	1 a	...	4 a	19 a	46 a	78 a	94 a
	Treated (12.5 g)	...	1 a	...	4 a	8 b	30 b	57 b	90 a
	Treated (25 g)	...	0 a	...	3 a	7 b	15 c	58 b	88 a

^v For each experiment, means within a column followed by the same letter are not significantly different at $P = 0.05$ according to Fisher's least significant difference test.

^w Decimal wheat growth stage according to Zadoks et al. (48).

^x Nodal roots of 40 plants per plot were assessed for take-all necrosis. Percentage values are means of three replicates.

^y ... = No assessment.

^z Nodal roots of 40 plants per plot were assessed for take-all necrosis. Percentage values are means of four replicates.

conditions. Epidemics were classified on the basis of the evaluation of disease incidence and severity over time. They were partly linked to early climatic events known to be responsible for early infections. Thus, moderate and severe development of the disease (LR295, P297, and P397) occurred in the sites with the highest accumulation of degree days (1,148.6 in 1994–1995 and 900.1 in 1996–1997 versus 696.6 in 1995–1996) and precipitation (370 mm in 1994–1995 and 319 mm in 1996–1997 versus 235 mm in 1995–1996) from sowing to tillering (GS23).

The effect of seed treatment differed among the epidemics. In an early epidemic (LR295), the fungicide significantly reduced disease incidence during all or part of the cropping season, depending on the fungicide rate. There was a 3- to 5-week delay in the incidence of plants treated at the highest rate of fungicide compared with untreated plants throughout the cropping season. In late epidemics (LR296, LR396, P297, and P397), the seed treatment provided moderate reductions in disease incidence at the infection onset.

In contrast to disease incidence, severity variables were significantly reduced by the seed treatment during the whole epidemic. This effect was still apparent as late as flowering, at which stage the reduction in the proportion of necrotic root system (TAI) achieved through the high rate of seed treatment (50 g a.i./100 kg of seed) ranged from 67 to 81% among experiments. Root-to-root spread was also greatly limited, since the reduction in PDR ranged from 57 to 73%. Assessment of the absolute numbers of diseased

and total roots, for calculation of PDR per plant, is a time-consuming but valuable way to distinguish between effects of the seed treatment on disease development and host growth. The reductions in PDR achieved with the seed treatment were exclusively due to

TABLE 6. Parameter estimates of take-all incidence progress curves^y

Experiment	Seed treatment (a.i./100 kg seed)	Parameter		<i>r</i> ^{2z}
		<i>c</i> ₁	<i>c</i> ₂	
LR295	Untreated	2.60 × 10 ⁻⁵	4.37 × 10 ⁻³	0.97
	Treated (25 g)	2.75 × 10 ⁻⁶	5.41 × 10 ⁻³	0.94
	Treated (50 g)	6.33 × 10 ⁻⁶	4.37 × 10 ⁻³	0.92
LR296	Untreated	1.22 × 10 ⁻⁵	4.20 × 10 ⁻³	0.97
	Treated (25 g)	8.64 × 10 ⁻⁶	3.81 × 10 ⁻³	0.98
	Treated (50 g)	1.77 × 10 ⁻⁶	5.44 × 10 ⁻³	0.99
LR396	Untreated	5.56 × 10 ⁻⁶	4.44 × 10 ⁻³	0.93
	Treated (25 g)	6.17 × 10 ⁻⁶	3.73 × 10 ⁻³	0.93
	Treated (50 g)	3.38 × 10 ⁻⁶	4.07 × 10 ⁻³	0.99
P297	Untreated	1.84 × 10 ⁻⁶	5.06 × 10 ⁻³	0.97
	Treated (25 g)	8.74 × 10 ⁻⁷	5.31 × 10 ⁻³	0.98
	Treated (50 g)	3.55 × 10 ⁻⁷	5.75 × 10 ⁻³	0.97
P397	Untreated	5.92 × 10 ⁻⁷	7.90 × 10 ⁻³	0.99
	Treated (12.5 g)	1.13 × 10 ⁻⁶	6.97 × 10 ⁻³	0.97
	Treated (25 g)	1.86 × 10 ⁻⁶	6.16 × 10 ⁻³	0.97

^y Parameters *c*₁ and *c*₂ were estimated by fitting the equation

$$y = \frac{1 - e^{-(c_1 + c_2)t}}{1 + \left(\frac{c_2}{c_1}\right)e^{-(c_1 + c_2)t}}$$

to incidence data for each experimental treatment, in which *y* is the proportion of diseased plants and *t* is the sum of degree-days since sowing.

^z The *r*² values correspond to the ratio of regression sum of squares to total sum of squares.

TABLE 7. Effect of an experimental seed treatment on the parameter estimates of take-all incidence progress curves^y

Seed treatment (a.i./100 kg seed)	Parameter	
	<i>c</i> ₁ ^z	<i>c</i> ₂
Untreated	3.88 × 10 ⁻¹ a	4.52 × 10 ⁻³ a
Treated (25 g)	3.63 × 10 ⁻¹ ab	4.57 × 10 ⁻³ a
Treated (50 g)	3.47 × 10 ⁻¹ b	4.91 × 10 ⁻³ a
Probability	0.066	>0.20

^y The seed-treatment effect was tested by considering all experiments except P397, in which the highest rate of fungicide was not investigated.

^z A Box and Cox transformation (12) was needed to homogenize variances.

The transformation was *c*_{1 transformed} = *c*₁^{1-1.84/2}

TABLE 8. Parameter estimates and time (in degree-days) to reach the point of inflection of the progress curves of the proportion of diseased roots (*I*_{PDR})^y

Experiment	Seed treatment (a.i./100 kg seed)	Parameter		<i>I</i> _{PDR}	<i>r</i> ^{2z}
		<i>α</i>	<i>β</i>		
LR296	Untreated	5.11 × 10 ⁻¹⁵	4.24	2,203	0.92
	Treated (25 g)	1.71 × 10 ⁻¹⁹	5.46	2,637	0.97
	Treated (50 g)	6.87 × 10 ⁻²⁰	5.61	2,516	0.96
LR396	Untreated	1.71 × 10 ⁻²⁶	7.67	2,247	0.76
	Treated (25 g)	7.03 × 10 ⁻¹⁶	4.30	3,142	0.85
	Treated (50 g)	8.35 × 10 ⁻²⁶	7.33	2,586	0.90
P297	Untreated	3.09 × 10 ⁻²⁰	5.85	2,095	0.89
	Treated (25 g)	7.30 × 10 ⁻¹⁹	5.37	2,295	0.80
	Treated (50 g)	3.23 × 10 ⁻¹⁸	5.06	2,740	0.84
P397	Untreated	1.16 × 10 ⁻¹⁶	4.90	1,706	0.97
	Treated (12.5 g)	2.43 × 10 ⁻¹⁷	5.03	1,923	0.97
	Treated (25 g)	3.84 × 10 ⁻²¹	6.17	1,979	0.97

^y Parameters *α* and *β* were estimated by fitting the equation *y* = 1 - *e*^{*αt*^{*β*}} to data for each experimental treatment, in which *y* is the proportion of diseased roots per plant and *t* is the sum of degree-days since sowing.

^z The *r*² values correspond to the ratio of regression sum of squares to total sum of squares.

TABLE 9. Parameter estimates and time (in degree-days) to reach the point of inflection of the progress curves of the take-all index (*I*_{TAI})^y

Experiment	Seed treatment (a.i./100 kg seed)	Parameter		<i>I</i> _{TAI}	<i>r</i> ^{2z}
		<i>α</i>	<i>β</i>		
LR295	Untreated	3.20 × 10 ⁻¹⁵	4.40	1,857	0.87
	Treated (25 g)	4.26 × 10 ⁻¹⁹	5.45	2,263	0.73
	Treated (50 g)	1.19 × 10 ⁻¹⁴	3.97	2,990	0.62
LR296	Untreated	1.56 × 10 ⁻¹⁸	5.34	2,079	0.92
	Treated (25 g)	4.52 × 10 ⁻¹⁸	5.02	2,729	0.99
	Treated (50 g)	4.82 × 10 ⁻²²	6.28	2,412	0.92
LR396	Untreated	8.25 × 10 ⁻²⁴	6.85	2,290	0.72
	Treated (25 g)	3.11 × 10 ⁻²⁰	5.67	2,664	0.94
	Treated (50 g)	1.51 × 10 ⁻²²	6.31	2,794	0.94
P297	Untreated	1.69 × 10 ⁻²¹	6.20	2,178	0.87
	Treated (25 g)	5.95 × 10 ⁻²¹	5.93	2,496	0.64
	Treated (50 g)	7.69 × 10 ⁻²⁰	5.46	3,052	0.75
P397	Untreated	2.20 × 10 ⁻¹⁷	5.10	1,769	0.95
	Treated (12.5 g)	6.63 × 10 ⁻¹⁶	4.56	2,019	0.94
	Treated (25 g)	1.05 × 10 ⁻²¹	6.29	2,105	0.96

^y Parameters *α* and *β* were estimated by fitting the equation *y* = 0.8(1 - *e*^{*αt*^{*β*}}) to TAI data for each experimental treatment, in which *y* is the proportion of the root system necrotic and *t* is the sum of degree-days since sowing.

^z The *r*² values correspond to the ratio of regression sum of squares to total sum of squares.

TABLE 10. Effect of an experimental seed treatment on the time (in degree-days) to reach the point of inflection of the progress curves of the proportion of diseased roots (*I*_{PDR}) and take-all index (*I*_{TAI})^y

Seed treatment (a.i./100 kg seed)	<i>I</i> _{PDR}	<i>I</i> _{TAI}
Untreated	2,182 b ^z	2,101 b
Treated (25 g)	2,691 a	2,538 ab
Treated (50 g)	2,614 a	2,812 a
Probability	0.104	0.006

^y Estimation of the seed-treatment effect was made by considering all sites except P397, in which the highest rate of fungicide was not investigated.

^z Means within a column followed by the same letter are not significantly different at the indicated *P* value according to Fisher's least significant difference test.

reductions in the number of diseased roots per plant, since there were no significant differences in the total number of roots per plant between treated and untreated plots whatever the site (data not shown). These observations are consistent with a previous report (30). The effect of the seed treatment could, therefore, be ascribed to a direct fungicidal activity rather than to a disease escape due to a reduced root emission as underscored for triadimenol seed treatment (6,45).

Results on PDR and TAI provide a different insight than those on incidence. Indeed, at late growth stages, there were generally as many diseased plants in treated as in untreated plots, whereas the corresponding severity levels still differed among experimental treatments. These data demonstrate that assessments of disease incidence alone are clearly insufficient to evaluate the benefits of a fungicide and need to be complemented by assessment of disease severity. Just as a single disease variable is not sufficient, it is useful to complement the static date-by-date study with a dynamic analysis in order to judge the potential of a fungicide.

The shape of incidence curves and the analysis of their equation parameters allowed ranking of sites according to infection onset and disease progress. Parameter c_1 , associated with primary infections, quantified the earliness of root invasion by mycelium surviving on former host crop residues. LR295 was the experiment with the earliest infections and had milder and more humid climatic conditions until early spring than did the other experiments. Parameter c_2 , associated with secondary infections, quantified the spread of infection from one plant to another.

Seed treatment applied at the highest rate significantly reduced c_1 . Parameter c_1 depends on the rate of primary infection and the amount of soil inoculum. As the latter is assumed to be constant within plots of a single experiment, the seed treatment consequently acts on parameter c_1 by reducing the rate of primary infection. However, owing to the assessment method, it was impossible to distinguish between the mechanisms known to be involved in the infectious process. Therefore, the seed treatment could have reduced the primary infections by increasing the threshold of inoculum particle size necessary for infection (47) by reducing the mean distance between host root and inoculum particle required for successful infection (24), by limiting the ectotrophic growth of runner hyphae along the root (21), or by increasing the time necessary for initiation of the dark stelar discoloration that is characteristic of take-all (23). A similar reduction in the primary infection parameter can be achieved by delayed sowing (17), but this practice leads to possible reduction in yield potential (19). Thus, the seed treatment might be a good tool for maintenance of optimum sowing date. Nevertheless, in fields at heavy risk for take-all, a combination of seed treatment and delayed sowing might be a way to improve efficacy of take-all control. While the seed treatment significantly reduced primary infections, no effect on secondary infections (on the late contamination of healthy plants by mycelium arising from diseased plants) could be demonstrated in our experiments. The significant control of the disease achieved throughout the cropping season, as in LR295, shows that earliness of infection and primary infections are of great importance in explaining further epidemic progress. As a result, it explains how the efficacy of a seed treatment can be maintained long after its application.

The effect of the seed treatment on the severity progress curves was investigated through the analysis of the point of inflection; the faster the infection spreads, the shorter the time necessary to reach the point of inflection. This time is, therefore, inversely proportional to the infection speed. The high rate of fungicide application significantly increased the time needed to reach the point of inflection of PDR and TAI progress curves, demonstrating a delay in root-to-root spread and in the extension of root necrosis. Seed treatment appeared more efficient in limiting contamination of healthy roots from diseased roots (significant effect for both rates) than in slowing down the extension of necrosis on diseased roots (significant effect only for the highest rate of fungicide).

The experiment sites chosen provided a variety of epidemics in terms of incidence and severity. This constituted an efficient experimental frame to test fungicide potential. Results showed the efficacy of the seed treatment for the control of take-all under natural conditions. Considering the incidence, the earlier the epidemic developed, the earlier the fungicide control could occur and the longer its effects could be maintained. Collection of successive samples over time permitted the appraisal of effects of the seed treatment on disease variables throughout the cropping season. This approach was complemented by the use of nonlinear models to describe the temporal progress of disease. Epidemic modeling followed by the analysis of equation parameters and point of inflection proved to be a new and informative way to investigate the efficacy of a fungicide on a whole epidemic.

The relationship between disease progress and yield loss still remains to be established. Seed treatment with MON65500 was shown to be a useful experimental tool for obtaining different disease incidence and disease severity progress curves at one location with identical cultural practices and climatic conditions. These curves provided a large range of disease scenarios for studying crop responses to take-all attacks. Thus, epidemics were linked to consequences on wheat growth and development and to the elaboration of yield components.

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