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Assessment of the profile of powdery mildew and its damage function at low disease intensities in field experiments with winter wheat

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Abstract

Damage by mildew to winter wheat was studied in 11 field experiments in the Netherlands. Damage is described by the simple function: -0.013 (SE = 0.003) kg are^{-1} per pustule-day of mildew per leaf, from second node stage to early dough at yield levels of 70 to 90 kg are^{-1}, in disease-free plots. No deviations from linearity at disease stresses from zero to two thousand pustule-days per leaf were observed. Years, cultivars or soil types did not affect the damage function significantly. The effects of mildew on some yield components were suggested.

Mildew profiles in untreated plots could be described by the equation: $CM = CLA^b$, in which CM and CLA are the cumulative pustule number and the cumulative leaf area, respectively, calculated both from top to bottom of the canopy, and totals standardized at unity. Estimates of the gradient parameter b averaged 3.4 (SE = 0.9). Observed differences in steepness of the profiles did not affect the damage function significantly.

Additional keywords: Triticum aestivum, Erysiphe graminis, vertical distribution, triadimefon, AUDPC-value, economic threshold, seed number.

Introduction

Chemical control of powdery mildew cost Dutch farmers 2-3% of their wheat yield of c. 8 t ha^{-1} per treatment in the early eighties. The profitability of mildew treatments was hard to determine as yield losses of 2 kg are^{-1} are difficult to assess with statistical significance in field experiments.

Percentage damage due to mildew has been estimated by $2\sqrt{MD}$, in which MD is the percentage leaf surface diseased at flowering (Large and Doling, 1963). As this function relates damage to mildew intensity at a single growth stage, it cannot be used to estimate the amount of mildew which can be tolerated during a period. Total damage depends on the onset and course of the epidemic (Calpouzos et al., 1976; Teng and Gaunt, 1980; Zadoks, 1985). In disease management systems, farmers or scouts can observe the onset of epidemics, but its course has then to be forecast. A damage function, which describes damage due to disease intensity during the season is then needed to evaluate the economic effect.

A study was undertaken to estimate damage caused by mildew epidemics at low to moderate intensities and to reveal whether certain developmental stages of the crop

are particularly sensitive to a mildew attack. To enable application of results to farmers' fields, damage caused by more or less spontaneous mildew epidemics with and without pesticide stress was assessed in field experiments. Special attention was given to the mildew profiles in the canopy, since leaves at different positions contribute differently to crop growth and yield (Spiertz et al., 1971; Heyland et al., 1979; Rabbinge et al., 1985). In a previous paper (Daamen, 1988) effects of cultivars on damage by mildew were described.

In this paper, disease intensity is used as the instantaneous quantity of disease, irrespective of the assessment method, and disease stress is the disease intensity integrated over time.

Materials and methods

Except the farm 'de Eest', experiments were located on farms in the Netherlands where mildew is common and other diseases, especially rusts, are infrequent (Fig. 1). Commercial cultivars were selected for their susceptibility to mildew and their moderate susceptibility to other diseases. The conditions of the experiments are given in Table 1. Except for weed, disease and pest control, crop husbandry was decided by the farm manager. If possible, weeds were controlled with MCPP or MCPA, to avoid an interac-

Table 1. Experimental conditions of the winter wheat trials.

Experiment:	BO80	EE80	SB80	VP80	BO81
Cultivar	Okapi	Caribo	Caribo	Caribo	Okapi
Soil	clay 40%	clay 40%	clay 35%	sand	clay 40%
Previous crop	sugar-beet	potato	sugar-beet	sugar-beet	potato
Sowing date	22 October '79	19 October '79	22 October '79	—	16 October '80
N-mineral[1]	35	130	60	manure	80
N fertilization	107 + 60	78	140 + 50	40 + 60	65 + 60
Weed control	29 October 8 l Tok Ultra 7 May 4 l MCPP	25 March 4 l MCPP	28 April 3 l MCPP + 2 kg Faneron	15 April 6 l Tok Ultra	16 October 8 l Tok Ultra 7 May 3 + 3 l MCPP + MCPA
CCC	28 April 1.5 l 16 May 1 l	none	28 April 0.8 l	11 April 0.8 l 23 April 0.8 l	23 April 1 l 8 May 1 l
Aphids[2]	3 July Pir.	1 July Dim.	11 July Pir.	2 July Pir.	23 June Pir.
Treatments	9	4	7	7	8
Replicates	6	6	6	6	6
Plot size m^2	4.6 × 15	4.5 × 18	7 × 6	18 × 5	7.5 × 6.2
harvested	2.2 × 14	1.5 × 11	6.5 × 4	16 × 3	5.6 × 4.2
Harvest date	15 August '80	18 August '80	21 August '80	8 August '80	12 August '81
Fungicide application		6 June Milgo E			23 June Bavistin M

[1] Estimated kg N available in February.
[2] Pir. = Pirimor, Dim. = dimethoate.

tion of herbicides with mildew (Ibenthal and Heitefuss, 1979). The name of the farm and the year of harvest were abbreviated to name the experiments (Fig. 1, Table 1). The first N application in BO80, EE80 and SB80 was c. 30 kg N ha^{-1} higher than common practice, to stimulate the start of mildew epidemics. In March 1980, plastic roofs were placed over mildew-infected plants in a corner of the experiments BO80 and SB80 and removed in April, to provide more mildew inoculum. In 1981, three mildew-infected plants were planted directly in each plot of BO81 and LP81 on 3 April, and in 1982, five such plants in each plot of WS82 on 10 May. Mildew epidemics were completely spontaneous in the other experiments.

Treatments, the different mildew epidemics. To produce different epidemics, the fungicide triadimefon (Bayleton, 0.5 kg ha^{-1}), commonly used by farmers, was used. Applications of triadimefon were planned to provide plots without mildew (2-4 applications), plots with early or late or intermediate epidemics, and plots with undisturbed mildew epidemics. Rationale of fungicide applications was to reduce the correlations between mildew intensities on successive dates.

In 1980, mildew did not develop in EE80, and the experiment was used to estimate the possible phytotoxic or phytotonic effects of triadimefon. To eliminate the scarce mildew, all plots were treated on 6 June with the selective mildew fungicide ethirimol (Milgo-E, 1 l ha^{-1}). In 1981, all plots of BO81 were treated on 23 June with carben-

LP81	WR81	VP81	WR82	WS82	GV82
Okapi clay 50% potato 13 October '80 46 80+30	Arminda loess 26% potato 24 November '80 — 110+35	Okapi sand potato 17 October '80 45 125+30	Arminda loess 28% potato 26 October '81 36 100+60	Okapi clay 32% sugar-beet 23 November '81 30 104+52+39	Okapi improved peat potato 15 October '81 60 78+39
21 April 2.5 l MCPA 3.5 l MCPP	4 April 6 l Tolkan	7 May 4 l MCPP	13 April 6 l DM-68	28 April MCPP+MCPA 2 June MCPA	28 April 6 l Arelon Combi
21 April 2 l	5 May 1.5 l	7 April 1 l 23 April 1 l	16 April 1 l	28 April 1.5 l	none
25 June Pir.	22 June Pir. 10 July Pir.	23 June Pir.	11 June Pir.	22 July Pir.	2 July Pir.
8 6 12.5×3 10×2.5 13 August '81	4 8 8×4 5×3 15 August '81	8 6 8.5×6.5 6×4.5 5 August '81 23 June Captafol	4 6 7×9 4×6 12 August '82 11 June Bayleton CF	4 6 7×9 4.5×7.5 10 August '82 22 June Bayleton CF	4 6 7×9 6.75×4.5 11 August '82

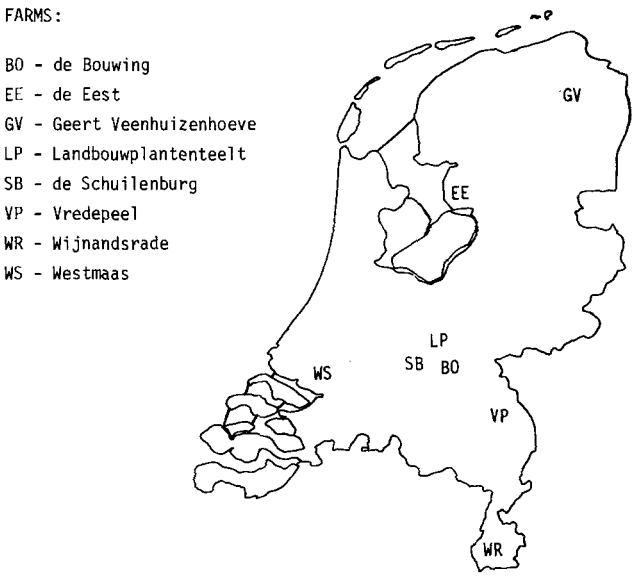


Fig. 1. Location of the experiments in the Netherlands.

dazim and maneb (Bavistin M 72, 3 kg ha^{-1}) to control speckled leaf blotch, *Mycosphaerella graminicola*, and VP81 was treated with captafol (Ortho-Difolatan 4F, 2 l ha^{-1}) to control glume blotch, *Leptosphaeria nodorum*. In 1982, damage by mildew before flowering was of interest and triadimefon and captafol (Bayleton CF, 2 kg ha^{-1}) was applied to all plots of WR82 on 11 June and of WS82 on 24 June.

Disease assessments. Disease intensity was assessed, more or less fortnightly, on samples of 10 (1980) or 15 (1982) culms per plot. In 1981, samples comprised 15 culms before and 10 after heading. Disease symptoms were assessed in the green leaf area only. The number of mildew pustules on each fully expanded leaf was assessed (Daamen, 1986). Mildew infection was severe in April 1980 only in VP80 and on the first observation date the percentage leaf area diseased was determined rather than pustule number. Intensity of other diseases was assessed as the absolute percentage leaf area diseased, disregarding chlorosis. The proportion dead leaf area was estimated for each leaf layer. The number of glumes with disease symptoms was determined as a measure of disease intensity on ears.

Computations. The average disease intensity was determined per leaf in each layer and per plot. Disease intensities were averaged over leaf layers, corrected for the proportion of dead leaf area, and the area under the disease progress curve (AUDPC-value, in disease-days) was computed. The starting date of the epidemic was derived from the first date of observation of the diseases in the experiments. It was assumed that kernel filling and epidemics stopped at the date the crop reached early dough (DC83). The AUDPC-value was computed as: total pustule number per culm, divided by the number of green leaves per culm integrated over time, or:

$$\text{AUDPC} = \sum_{t=s}^{t=e} \left[\sum_{j=1}^{j=d} \left(\sum_{i=1}^{i=n} x_{ij} \right)/n \right] \Big/ \sum_{j=1}^{j=d} (1 - f_j)] \qquad (1)$$

in which: x_{ij} is the disease score on leaf i of layer j; n is sample size; j is leaf layer 1 (top) to d; f_j is proportion dead leaf area; t is time in days from start date s, to end date e. A linear interpolation between observation dates was used to integrate the disease intensities over time.

Plots were combine-harvested and grain yield was standardized at 16% moisture content. Seed weight was used to estimate seed number per m2. In 1980, plots with treatments: 'without mildew', 'early' and 'undisturbed mildew epidemics', were twice the size of other plots. One half of each was used for three destructive measurements. Two rows 0.5 m long were harvested at random to estimate culm density, approximately one quarter of this sample was used to measure the green area index (GAI, m^2 per m^2 soil).

Analyses of covariance were done with Genstat IV and followed by a least significant range test according to Tukey. Residuals were inspected on inequality of variances and on curvilinearity and if necessary, transformed variates were analysed. One plot of BO81 with treatment 'without mildew' was excluded from analysis; for unknown causes the plot yield was recorded as 56.1 kg are^{-1}, which is far too low for this particular experiment.

Realized treatments: the mildew epidemics. In the Netherlands, mildew epidemics usually start in May, reach their highest intensity between flowering and milky ripe, and then decrease. Infections in autumn and early spring may occur, especially on sandy or improved peat soils. These may cause very early and severe epidemics with a stable or decreasing mildew intensity at shooting to heading.

Epidemics in the experiments, averaged per treatment are shown in Fig. 2. Those which did not differ significantly ($p < 0.05$) at any time were lumped. Undisturbed epidemics in WR81 and WR82, on loess soil, showed the usual disease development. In SB80, BO80 and LP81, on river clay, disease pattern was more or less similar. Epidemics varied in these experiments, according to years, and presumably also to the availability of nitrogen (Table 1). Conditions for mildew development were unfavourable in 1980 compared to 1981 and 1982. May and early June 1980 were unusually dry. In VP80, on sandy soil, leaves rolled up due to drought in mid June. In other experiments drought stress was not experienced. EE80 was located in the North East Polder where mildew is usually infrequent, especially in 1980. In VP81 and BO81, mildew infection was rather early, and heavy and moderate epidemics developed in the experiments, respectively. On the improved peat soils in the north east of the Netherlands, mildew epidemics are usually very early and severe. The absence of mildew in GV82 was exceptional, its cause being unknown. Mildew intensity on ears was low: three pustules per ear in SB80 and VP81, and one per ear in BO81 and WR81 and zero in the other experiments. Mildew on ears was therefore ignored in further analyses.

Fungicide applications caused significantly different mildew epidemics. Complete control was easily obtained in 1980, but not in 1981 and 1982, especially in WR81 and WR82, due to a decrease in sensitivity of mildew to triadimefon (De Waard et al., 1986). It was not possible to create clear-cut early or late mildew epidemics in the experiments. Fungicide application resulted in early to intermediate mildew epidemics.

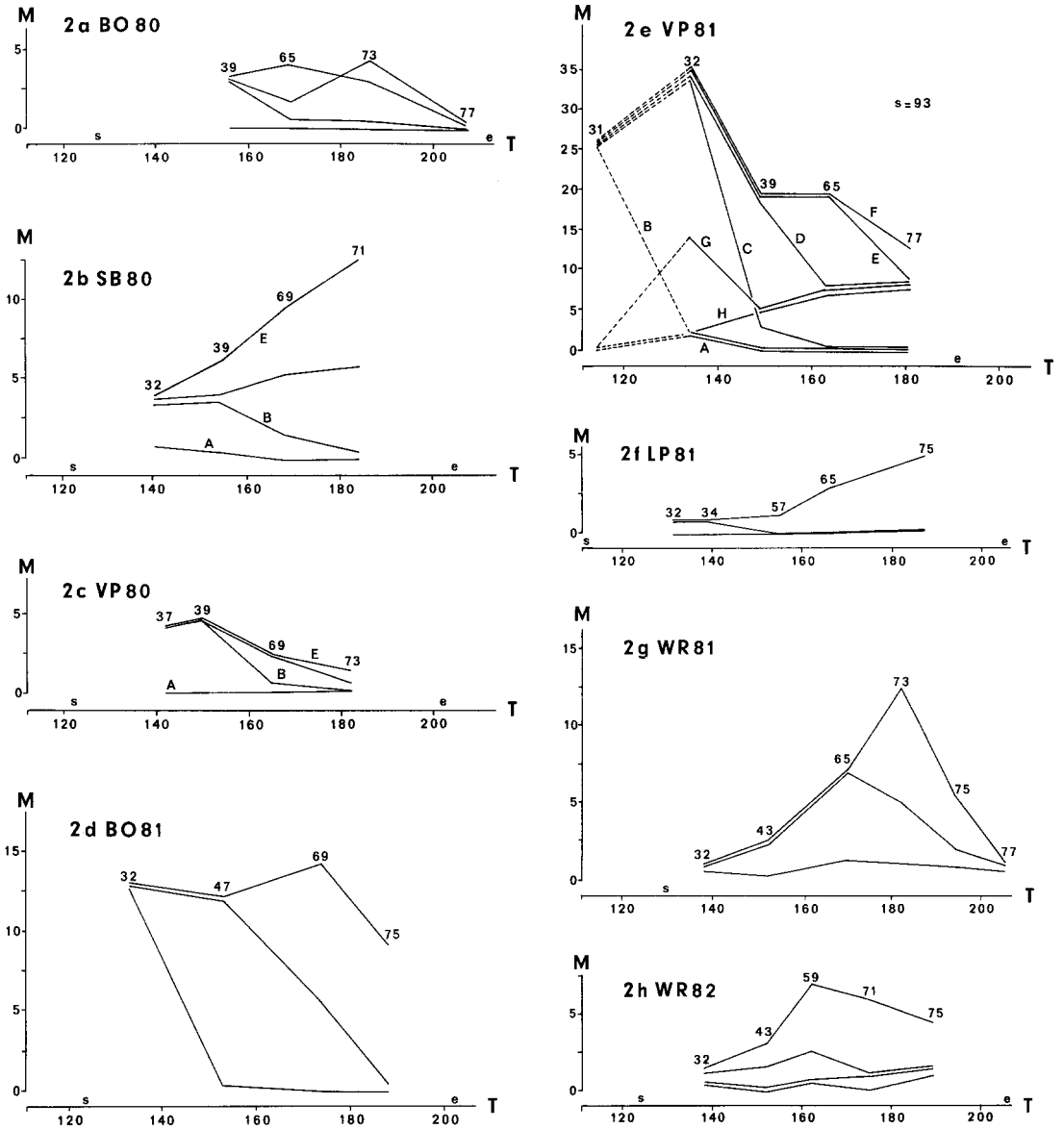


Fig. 2a, b, c, d, e, f, g, h. Mildew epidemics in the eperiments, M is pustule number per leaf and T is Julian day. Numbers in the figures represent developmental stages and capitals different treatments (Table 6). Only grouped epidemics whose patterns differed significantly from others ($P < 0.05$) due to fungicide application are shown. s is starting date of the epidemic and e is date of early dough.

Table 2. Other diseases and lodging in the experiments.

Experiment	Variate	Assessment date	Mean of variate	Significance of treatment[1]
BO80	speckled leaf blotch	—	46 %-days per leaf	ns
	sharp eyespot	727	50 % culms infected	ns
	leaf rust	727	0.1 % leaf area	ns
	glume blotch	727	4 % glumes infected	ns
	ear blight	727	1.5 % glumes infected	ns
	lodging	727	54 %	ns
SB80	speckled leaf blotch	—	55 %-days per leaf	ns
	leaf rust	725	2 % leaf area	ns
	lodging	725	30 %	ns
VP80	none			
BO81	speckled leaf blotch	—	90 %-days per leaf	* *
	leaf rust	707	0.02% leaf area	ns
	lodging	723	29 %	ns
VP81	glume blotch	—	37 %-days per leaf	ns
	lodging	717	1 %	ns
LP81	speckled leaf blotch	—	58 %-days per leaf	ns
	leaf rust	706	0.2 % leaf area	ns
	lodging	727	3 %	ns
WR81	leaf and glume blotch	—	120 %-days per leaf	*
	leaf rust	724	0.2 % leaf area	ns
	glume blotch	724	4 % glumes infected	*
	ear blight	724	1 % glumes infected	ns
WR82	speckled leaf blotch	—	61 %-day per leaf	* *
	lodging	708	10 %	ns

[1] Fungicide application, ns = not significant ($p > 0.05$); * = $p < 0.05$; * * = $p < 0.01$.

Realized treatments: other diseases and lodging. In Table 2, other diseases and lodging are listed. Leaf blotches due to glume blotch (*Leptosphaeria nodorum*) and speckled leaf blotch (*Mycosphaerella graminicola*) were common alongside mildew and with mildew were treated as covariates. There was little brown rust (*Puccinia recondita*) in the experiments. Though it was treated as a covariate when above 0.1% leaf area diseased. Glume blotch on ears and ear blight (*Fusarium* spp.) were treated as covariates if incidence was above 2%. Lodging was always treated as a covariate and if necessary, distinction was made between complete and moderate lodging. Stems were assessed for eyespot (*Pseudocercosporella herpotrichoides*) in May, its incidence was usually below 5%. In BO80, the disease (25% in May) turned out to be sharp eyespot (*Rhizoctonia cerealis*), which infected 50% of the stems on 25 July. Stem base diseases were not treated as covariates because of their insensitivity to triadimefon and of their low frequency.

Experiments without mildew. EE80, WS82 and GV82 (Fig. 1) failed, due to mildew being below 25 pustule-days per leaf. A significant ($p < 0.05$) fungicide effect of 2 kg are^{-1} (Table 3) occured only in EE80. The yield response is presumably due to speckled leaf blotch and brown rust, which severities were 2.5% and 0.2% on the 2nd

Tabel 3. Experiments without mildew, yields (kg are^{-1}) with (F) and without (−) application of triadimefon.

Experiment EE 80		6 June	Yield	Significance
Treatment 1		−	67.4	F1/17 = 7.3 *[1]
2		F[2]	69.4	
Experiment WS 82	**18 May**	**2 June**	**Yield**	**Significance**
Treatment 1	−	−	88.7	F3/15 = 0.6 ns
2	F	−	90.0	
3	−	F	88.7	
4	F	F	88.2	
Experiment GV82	**19 April**	**24 May**	**Yield**	**Significance**
Treatment 1	−	−	77.2	F3/15 = 0.1 ns
2	F	−	77.3	
3	−	F	77.6	
4	F	F	77.5	

[1] ns = not significant, $p > 0.05$; * = $p < 0.05$.
[2] F = triadimefon as 0.5 kg ha^{-1} Bayleton.

leaf at that time, respectively. Ear blight intensity, 2% of the glumes were infected on 23 July, showed a significant ($p < 0.05$) fungicide effect, though no significant correlation between yield and diseases was obtained. In WS82, diseases were nearly absent. On 6 July, speckled leaf blotch severity was 2% on the 4th leaf. In GV82, an epidemic of glume blotch developed, 10% of the total leaf area being diseased on 20 July (Table 3).

Results

Damage. To describe damage, plot yield was analysed statistically in dependence of mildew stress and covariates, taking the experimental design into account (Daamen, 1988). Plot yield varied from 40 to 90 kg are^{-1}, mildew stress from zero to two thousand pustule-days per leaf. The results of linear damage functions are shown in Fig. 3 and Table 4. All experiments showed significant damage, except drought-stricken VP80. Estimates of the damage per pustule-day of mildew per leaf varied from 0.010 (SE = 0.002) to 0.017 (SE = 0.004) kg are^{-1} and do not differ significantly from the value 0.0125 (Daamen, 1988). The R^2-values are rather low and depend on variation in disease stress in the experiments. The standard errors of residuals (SE) are below 3 kg are^{-1}, excluding VP80 and VP81. To assess whether the damage in the experiments can be described by a linear function, a quadratic component of the mildew stress was added. This addition did not give a significant improvement of the description of the data

Table 4. Estimates of the partial damage function $Y = Y_0 + b \cdot MD$; Y is yield in kg are^{-1}, MD is mildew stress in pustule-days per leaf and the significance of different b-values for each observation date.

Experiment	$\hat{Y}_O$	SE_{YO}	$\hat{b}$	SE_b	SE	R^2	df	Different b-values[1]
BO80	80.6	1.6	−0.017	0.004	2.9	0.50	44	F3/41 = 0.8 ns
SB80	75.3	2.1	−0.014	0.002	2.9	0.81	32	F3/29 = 0.5 ns
VP80	54.4	2.3	−0.009[3]	0.008	5.5	0.34	35	F3/32 = 0.3 ns
BO81	77.0	1.5	−0.010	0.002	2.2	0.86	35	F3/32 = 2.9 ns
VP81	57.3	2.5	−0.009	0.002	5.2	0.58	38	F3/35 = 4.2 **
VP81C[2]	73.4	6.1	−0.010	0.002	4.1	0.80	20	F4/17 = 2.8 ns
LP81	85.4	1.4	−0.011	0.005	2.4	0.41	37	F4/33 = 1.0 ns
WR81	73.0	1.7	−0.011	0.003	1.9	0.74	20	F5/15 = 1.2 ns
WR82	80.5	2.1	−0.016	0.006	2.2	0.78	15	F4/11 = 1.4 ns

[1] ns = not significant, $p > 0.05$; * $p < 0.05$; ** $p < 0.01$.
[2] As VP81 but plots with fungicide application on 7 April eliminated.
[3] not significant, $p > 0.05$.

in any of the experiments, so linearity was accepted.

Partial effects of covariates were often non-significant, presumably due to colinearity. In BO81, speckled leaf blotch showed a significant effect on yield: −0.036 kg are^{-1} (SE = 0.012) per %-day per leaf. In WR81, glume blotch correlated significantly with yield: −1.1 kg are^{-1} (SE = 0.4) per % glumes infected. Lodging was significant in WR82: −0.13 kg are^{-1} (SE = 0.02) per % lodging. Effects of covariates on yield are assumed to be additive in the analysis; deviation from additivity of mildew and the blotches on yield was tested for and found not to be significant in any experiment.

Experiment VP81 was analysed differently, as mildew intensity was assessed as percentage on the first observation and as pustule number on the following observations. This resulted in two estimates of damage, one from the very early and one from the later disease stress. The later attack showed a negative correlation with yield, its estimate (−0.009) being comparable to those of the other experiments (Fig. 3, Table 4). The very early mildew attack, however, had a positive relation with yield; its partial effect was 0.005 (SE = 0.001) kg are^{-1} yield increase per %-day of mildew. This is a surprising result. Plots with a fungicide application on 7 April had relatively low yields. If these plots were excluded from analysis (VP81C, Table 4), the very early attack was negatively correlated with yield, though not significant (−0.017 kg are^{-1}, SE = 0.009).

The developmental stage of the crop might affect damage. A multiple point model (Madden et al., 1981; Teng and Shane, 1983) should then give a better fit than the AUDPC approach used here. On the other hand, weather conditions during the season might affect damage. Shaw and Royle (1987) analysed damage in relation to weather by integration of disease intensity over thermal time instead of chronological time. If the developmental stage of the crop or weather or both affect damage, the damage functions may be different at each observation date. The significance of different damage functions (different b-values) compared to an average damage function (one b-value) was tested (last column of Table 4). None of the experiments did indicate a dependence

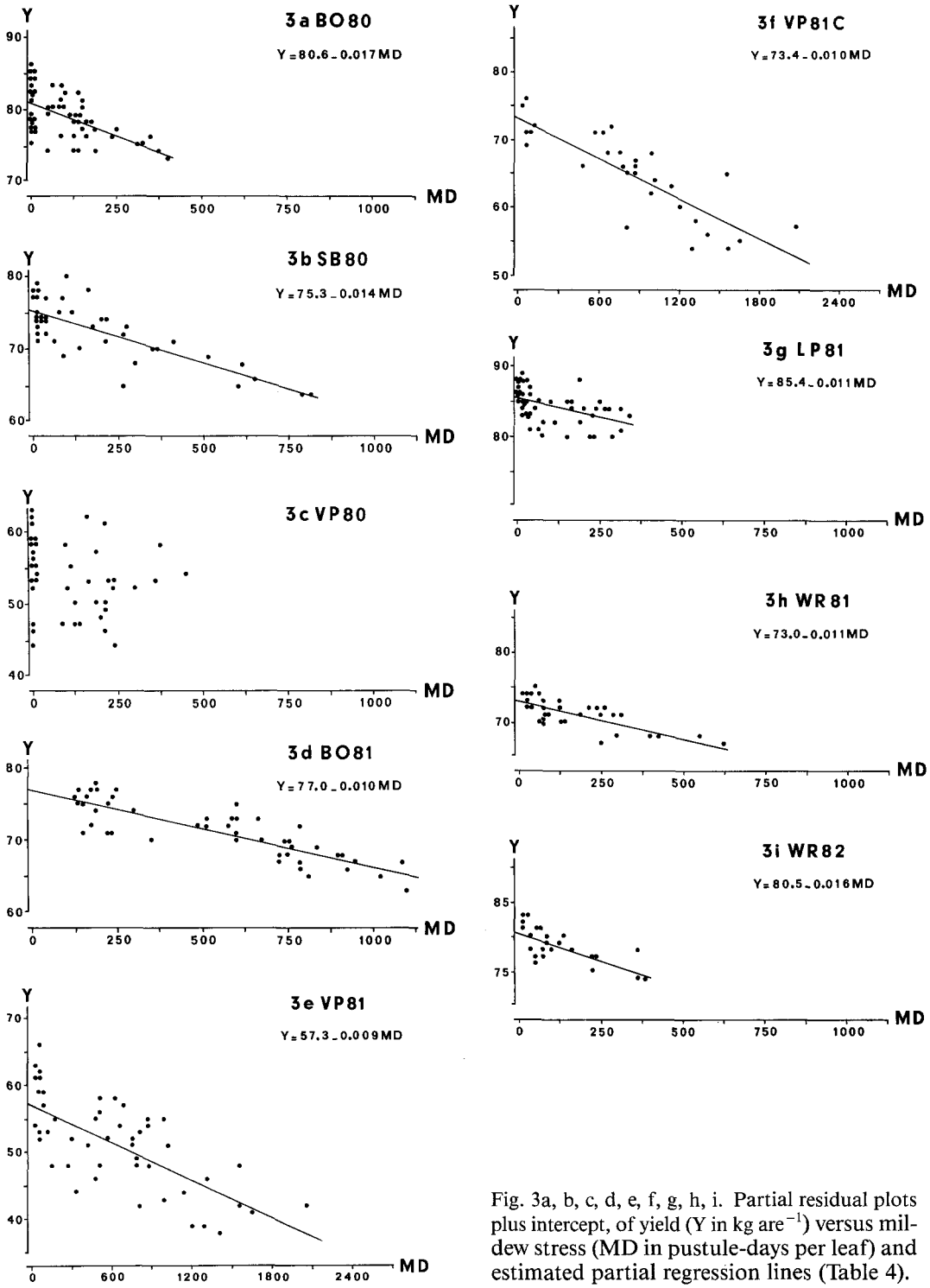


Fig. 3a, b, c, d, e, f, g, h, i. Partial residual plots plus intercept, of yield (Y in kg are^{-1}) versus mildew stress (MD in pustule-days per leaf) and estimated partial regression lines (Table 4).

Table 5. Estimates of the partial effect of mildew stress (MD, in pustule-days per leaf) on seed number: $SN = SN_O + b \cdot MD$, and the significance of different b-values for each observation date.

Experiment	$\hat{SN}_O^1$	SE_{NO}	$\hat{b}^2$	SE_b^3	SE[1]	R^2	df	Different b-values[3]
BO80	14.7	0.5	—	ns	0.9	0.25	44	F3/41 = 0.3 ns
SB80	16.8	0.5	−1.7	0.6	0.8	0.57	32	F3/29 = 0.0 ns
VP80	12.1	0.9	—	ns	2.1	0.15	35	F3/32 = 2.5 ns
BO81	16.1	0.3	−1.1	0.4	0.5	0.62	35	F3/32 = 1.2 ns
VP81	14.9	0.6	−1.4	0.5	1.2	0.41	38	F3/35 = 0.6 ns
VP81C[4]	16.7	1.4	−1.7	0.4	0.9	0.68	20	F3/17 = 0.3 ns
LP81	16.9	0.4	—	ns	0.6	0.45	37	F4/33 = 1.1 ns
WR81[5]	18.6	1.0	—	ns	0.7	0.56	8	F5/3 = 0.3 ns
WR82	21.6	1.1	—	ns	1.2	0.44	15	F4/11 = 1.2 ns

[1] In thousands per m^2.
[2] In numbers per m^2 per pustule-day of mildew per leaf.
[3] ns = not significant, $p > 0.05$.
[4] As VP81 but plots with fungicide application on 7 April eliminated.
[5] Seed numbers were estimated in only half of the replicates.

of the damage function on observation date, except VP81 due to the plots with fungicide treatment on 7 April. Thus, mildew stress expressed as area under the disease progress curve (AUDPC-value) gave an adequate description of damage, which confirms results of Frank and Ayers (1986), and of Carver and Griffiths (1981, 1982). The method Shaw and Royle (1987) proposed can be viewed as a sophisticated and formal description of the method used here. According to their theory, results of this study imply that the loss rate l in kg are^{-1} day^{-1}, and M in pustules $leaf^{-1}$, equals:

$$l = dy/dt = -0.013\ M \qquad (2)$$

Seed numbers. Seed numbers were analysed in a similar way as the yields (Table 5). Estimates of seed numbers in the absence of disease were between 12,000 per m^2 in drought stressed VP80 and 22,000 per m^2 in WR82. In SB80, BO81 and VP81, experiments which had highest mildew stress, the effect of mildew on seed number was significant. Estimated decrease of seed number varied from 1.1 (SE = 0.4) to 1.7 (SE = 0.6) per m^2 per pustule-day of mildew per leaf. Interactions of the relation with observation date were not significant in any of the experiments (last column Table 5). R^2-values are lower than those of the yield analyses, as seed number is only one of the yield components. Percentage decrease of seed number was 0.009 per pustule-day, whereas percentage damage averages 0.016 per pustule-day of mildew (Table 4 and 5). Thus in these experiments, decrease of seed number explains slightly more than 50% of damage.

Green area index and culm density. In BO80, no significant effect of treatment on GAI, yield and yield components was present. GAI was 2.2, 5.6 and 1.6 on 29 April,

Table 6. Fungicide applications, crop characteristics and final yield of three different treatments in experiments of 1980.

	Experiment SB80				Experiment VP80			
	date	treatment			date	treatment		
		A	B	E		A	B	E
Fungicide[1]	409	1/2 F	—	—	404	1/2 F	—	—
application	507	1/2 F	—	—	502	1/2 F	—	—
	523	F	—	—	520	1/2 F	—	—
	604	—	F	—	602	—	F	—
	619	1/2 F	1/2 F	—	617	1/2 F	1/2 F	—
GAI[2]	422	2.1a	2.2a	1.9a	416	1.5a	1.6a	1.5a
	602	5.3a	4.8ab	4.4b	529	4.9a	3.9a	3.2a
	702	3.9a	4.1a	3.3a	630	2.5a	2.2a	1.4b
Ear number m^{-2}	—	611 a	619 a	569 a	—	440 a	384 a	335 b
Seed number[3]	—	16.6a	15.9ab	15.2b	—	12.9a	12.2a	10.6a
Yield[4]	—	72.6a	70.0a	63.6b	—	56.4a	54.8a	49.5a

[1] 1/2 F and F are one half and a full dosage of triadimefon as 0.5 kg ha^{-1} Bayleton.
[2] Different letters indicate significant ($p < 0.05$) differences between treatments.
[3] in 10^3 per m^2.
[4] in kg are^{-1}.

10 June and 22 July, respectively; ear number averaged 445 per m^2 (Van de Heijden et al., 1981). Crop characteristics of the two other experiments of 1980 are given in Table 6, the treatments are also shown in Fig. 2b,c. In SB80, GAI on 2 June, yield and seed number were significantly lower in plots without control than in plots with complete control. Crop characteristics of treatment B, a late fungicide application, were intermediate, and final yield differed significantly from untreated plots. The significant decrease in seed number in SB80 can be explained almost entirely from the decrease in ear number. In VP80, GAI and seed number were lower than in BO80 and SB80, due to drought stress. Ear number and GAI on 30 June were significantly lower in untreated plots than in plots with complete control. Final yield did not differ significantly. The reduction of GAI in SB80 and VP80 was caused by a reduction of the number of culms per m^2 (Table 6), but leaf size might have been affected also. Individual leaf, ear and neck areas of SB80 and VP80 are given in Table 7. No significant effect of treatment on size of newly formed leaves is present, which agrees with observations in barley (Carver and Griffiths, 1981). Therefore, observed differences in GAI should be attributed mainly to differences in culm density. SED between leaf positions within a treatment was c. 1.3 cm^2, so the size of flag leaves was significantly lower (c. 20-25%) than that of 2nd and 3rd leaves in these experiments of 1980.

Table 7. Green leaf, ear and neck areas in cm^2 (plane projection) in two experiments with different treatments (see Table 6).

	Experiment SB80				Experiment VP80			
	date	treatment			date	treatment		
		A	B	E		A	B	E
Flag leaf	602	16a	16a	15a	529	10a	9a	8a
Second leaf	602	23a	24a	22a	529	19a	18a	16a
Third leaf	602	24a	23a	23a	529	19a	18a	16a
Fourth leaf	602	15a	10b	11b	529	15a	12a	11a
Fifth leaf	602	2a	0b	0b	529	4a	2a	2a
Ear and neck	702	12a	12a	12a	630	8a	9a	8a
Flag leaf	702	19a	20a	19a	630	15a	15a	13a
Second leaf	702	26a	27a	25a	630	21a	21a	17a
Third leaf	702	13a	16a	16a	630	10a	12a	7b
Fourth leaf	702	2a	1a	1a	630	1a	2b	1a

Mildew profiles. The profiles of mildew in the canopies of the untreated plots of the experiments are shown in Fig. 5. The graphs illustrate the continuous infection of new top leaves and the disappearance of mildew due to lower leaves dying. Mildew predominates on lower leaves because epidemics are mostly endogenous and lower leaves are longer exposed to infection. To describe the mildew profiles, a simple power function was used:

$$CM = CLA^b \qquad (3)$$

in which CM is the cumulative pustule number divided by total pustule number per culm ($0 \leqq CM \leqq 1$), CLA is the cumulative leaf area divided by the total leaf area ($0 \leqq CLA \leqq 1$). Both variates are calculated from top to bottom of the canopy. Parameter b has no dimension and determines the steepness of the gradient, if b equals unity, the vertical distribution is uniform. As discussed above, flag leaf size was c. 20-25% smaller than that of 2nd or 3rd leaves in cv. Okapi and Caribo in 1980, which might be correlated with the dry period and high solar radiation in spring 1980. Because leaf size was not measured in all experiments, it is assumed that the different leaves have equal leaf areas, for the sake of simplicity.

The gradient parameter b was estimated by direct curve fitting of model (3). The estimated relations and b-values are given in Fig. 5 and Table 8. Estimates of b from an experiment with different cultivars on the farm 'de Bouwing' in 1983 (BO83, Daamen, 1988) and of WR82, before fungicides were applied, are also given. The simple model described the data adequately in 33 out of 36 cases.

In three cases the model did not describe the data adequately, which was correlated with high or low b-values (Table 8). In BO80, mildew density was relatively high on the dying 4th leaf early in July, for unknown reasons, by which the profile was discon-

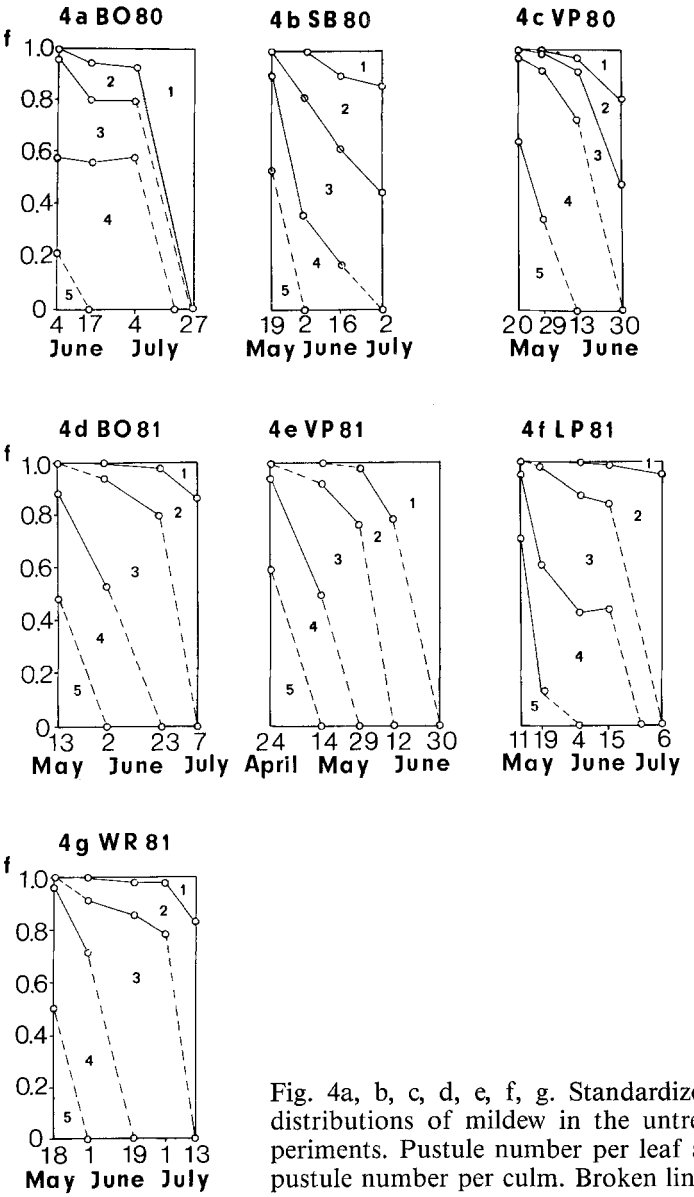
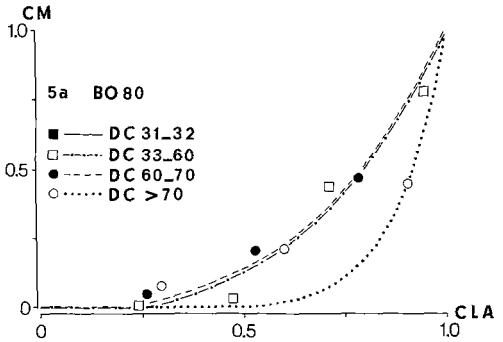
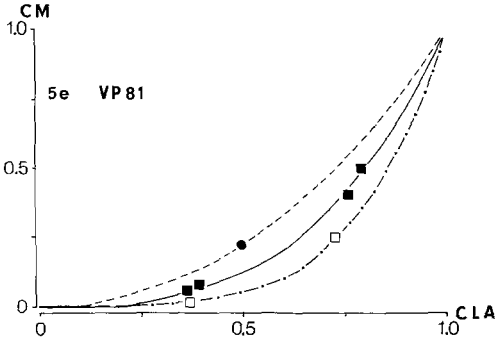
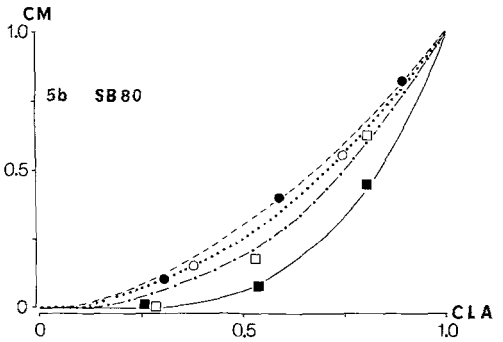


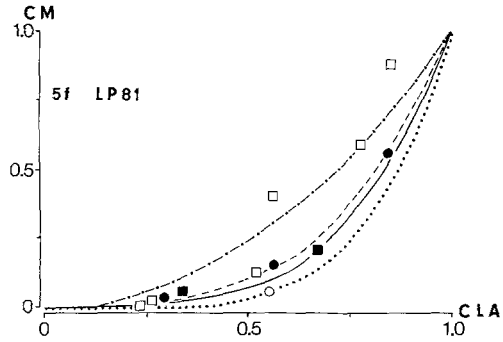
Fig. 4a, b, c, d, e, f, g. Standardized cumulative vertical distributions of mildew in the untreated plots of the experiments. Pustule number per leaf as fraction (f) of total pustule number per culm. Broken lines are extrapolations.

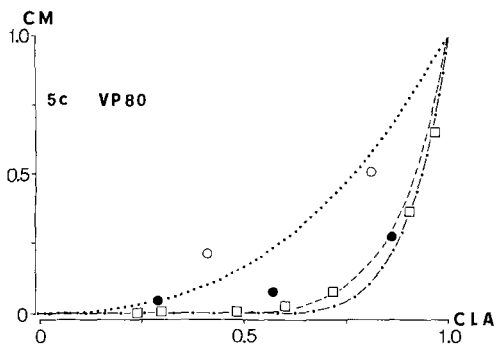
tinuous (Fig. 2a, 3a). Estimates of b were extremely high in VP80 before flowering, so mildew was more or less restricted to lower leaves. This could be expected as VP80 suffered water shortage and mildew development was slow on the rolled-up top leaves. The b-value was close to unity in WR81, at the start of the epidemic in second node stage, indicating a more or less uniform mildew profile. The 2nd leaf, which had just appeared, was nearly clean (Fig. 4g and 5g). Thus, the mildew population at that time originated presumably from an exogenous source.

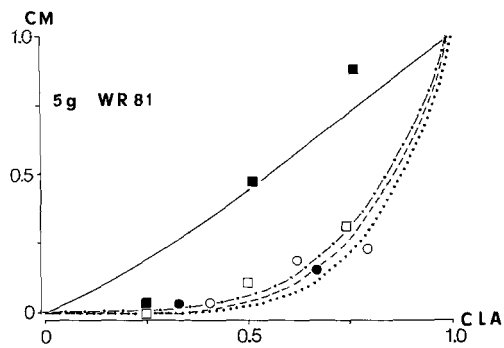












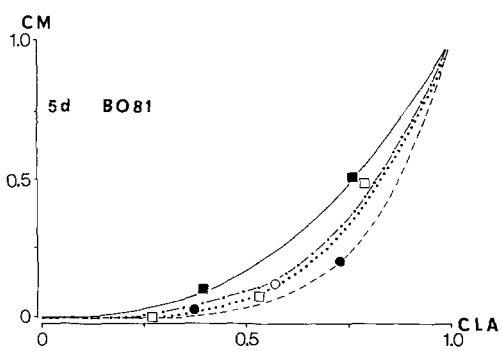


Fig. 5a, b, c, d, e, f, g. Standardized cumulative pustule numbers (CM) in relation to standardized cumulative leaf areas (CLA), calculated from top to bottom leaves in the untreated plots of the experiments, in relation to developmental stage. Curves are the estimated relations (Table 8). See Fig 5a for the symbols used.

Table 8. Estimates of the gradient parameter b, in the standardized cumulative distribution function: $CM = CLA^b$, at different developmental stages (DC) of the crop and their standard deviations.

Experiment		Developmental stages									
	cultivar	31, 32		33-60		60-70		>70		average[3]	
BO80	Okapi	—		3.0	(0.5)	3.0	(0.6)	7.9	(2.1)[2]	3.0	(0.0)
SB80	Caribo	3.9	(0.2)	2.4	(0.1)	1.8	(0.1)	2.0	(0.1)	2.5	(0.8)
VP80	Caribo	—		10.4	(1.3)[1]	8.3	(1.3)[1,2]	2.6	(0.4)	—	
BO81	Okapi	2.6	(0.1)	3.5	(0.2)	5.1	(0.4)	3.8	(—)	3.7	(1.0)
VP81	Okapi	3.1	(0.1)	4.5	(0.3)	2.1	(0.1)	—[4]		3.2	(1.2)
LP81	Okapi	3.9	(0.8)	2.1	(0.2)	3.4	(0.5)	5.0	(—)	3.6	(1.2)
WR81	Arminda	1.2	(0.2)[1,2]	4.1	(0.8)	4.7	(1.2)	5.4	(1.1)	4.7	(0.7)
WR82	Arminda	4.1	(0.3)	2.9	(0.1)	—		—		3.5	(0.8)
BO83	Nautica	3.5	(0.4)	2.5	(0.3)	2.8	(0.4)	—		2.9	(0.5)
BO83	Arminda	3.1	(0.3)	3.0	(0.3)	2.8	(0.4)	—		3.0	(0.5)
BO83	Durin	3.5	(0.2)	4.3	(0.4)	3.3	(0.3)	—		3.7	(0.5)
Average[3]		3.5	(0.6)	3.2	(0.8)	3.2	(1.1)	3.8	(1.5)	3.4	(0.9)

[1] Extreme value, see text.
[2] Model does not fit.
[3] Estimates with [1] and [2] excluded.
[4] One leaf layer only.

Estimated b-values did not show a trend with developmental stage of the crop. Such a trend might be expected when leaf formation stops, but it is presumably counterbalanced by the increase of resistance with higher leaf position (Hyde, 1976; Sander, 1983). The b-values were slightly different in the experiments, but no significant correlation was found between the estimated damage functions and the estimates of the gradient parameter b.

Discussion

Mildew profiles. Mildew profiles varied, but did not show systematic effects of year or developmental stage. The simple power function (3) described the standardized cumulative distribution well. Bad fits were obtained in situations where disease development was strongly disrupted or dominated by an exogene source.

The question arose as to whether a relation exists with models commonly used to describe horizontal disease gradients, i.e. the power function (4) and the exponential function (5) (Minogue, 1986; Fitt and McCartney, 1986):

$$y_s = a \cdot s^{-b} \quad (4)$$

$$y_s = a \cdot e^{-b.s} \quad (5)$$

In which y_s is the disease intensity in relation to distance s from the source and a and

b are parameters determining the disease intensity and the steepness of the gradient, respectively. Equation 4 predicts an infinite disease intensity at the source (s = 0), but the model can be expanded (Mundt and Leonard, 1985). To compare the models, equation 3 is transformed. If it is assumed that leaf area is uniformly distributed with distance s from the bottom of the canopy and that the disease intensity y can be measured in different units, (3) can be written as:

$$y_s = b \,.\, \bar{y}\,(1 - s/s_t)^{b-1}; \quad 0 \leqq s \leqq s_t \tag{6}$$

in which $\bar{y}$ is the average disease intensity in the gradient, s_t is the maximal distance (top canopy) from the source and b is the gradient parameter of (3). Disease intensity in the gradient described by (6) is b times the average density at the bottom, and zero at the top of the canopy. Thus $b \,.\, \bar{y}$, the disease intensity at the source, is comparable to parameter a of model 5. Comparison of equations 4, 5 with 6 does not reveal a direct equivalence. As b (3, 6) averaged 3.4, the lowest leaves dying carried about three times more mildew than average.

Damage assessments. Concerning the methodology used to study yield loss, the factors Madden (1983) reviewed were followed. Three additional comments can be made. First, the range of damage in the experiments should be of practical interest. In this study, the purpose was to estimate damage of about 3%. Experiment VP81 is therefore not of interest, since mildew stress was too severe and farmers would spray anyhow. Second, use of inoculations at different times to obtain different disease intensities may have a strong impact on the disease profile in the canopy, which may interfere with extrapolations to farmers' fields. Third, mildew intensity was assessed with error (Daamen, 1986). Nevertheless regression techniques were used, since no simple methods to analyse functional relationships seem to be available, in particular to analyse the present set of complex data. Estimates are biased by which damage is underestimated, but the seven experiments with different mildew intensities showed the same trend (Fig. 2).

The three experiments without mildew indicated that the fungicide triadimefon did not have a direct effect, whether fytotoxic or fytotonic. In the seven experiments with mildew, no clear patterns in residuals in relation to fungicide application were detected. Except in VP81 where plots with fungicide application on 7 April had low yields, which may indicate fytotoxicity. As fungicide application is confounded with disease stress, both factors and variates were analysed together to evaluate the stability of the damage functions. Experiments containing plots with a mildew stress above 500 pustule-days (SB80, BO81, WR81, VP81) were analysed. Estimated damage functions were stable, VP81 excepted. Both VP81 and BO81 indicated that an average damage function does not describe plot yield perfectly, because fungicide application adjusted for diseases showed a significant ($p < 0.05$) effect. They had rather early and severe attacks and tended to indicate some dependence of the damage function on observation date (Table 4, last column). Therefore it is concluded that very early attacks, before the second node stage, may cause deviations from the common damage function. As the damage function was not significantly improved by an interaction with observation date, thermal time does not seem to be important in this study. However, the resolution power of the field experiments might be too small to discriminate between damage caused during different periods. A more basal physiological approach (Rabbinge et al., 1985) might

be more suited to solve this problem, if adequate models are available to estimate damage at field level from measurements on physiology at leaf level.

The decrease in seed number per m^2 with increasing mildew stress accounted for more than 50% of the damage in the experiments with highest disease stress (Table 4). The effect was attributed to a decrease in ear number (SB80) but it may also be caused by an increase of seed abortion, as in spring barley (Lim and Gaunt, 1986b). Effects of mildew on seed number per ear in wheat are reported by Royse et al. (1980), Fried et al. (1981), and Zaharieva et al. (1984), but data by Kolbe (1982) indicate that seed number per ear is less affected by mildew in wheat as it is in barley. Mildew decreased GAI sinificantly, which was mainly attributed to a decrease of culm density (SB80, VP80). Yield is often attributed to the production by the top two leaves and the ear, by which intensity of diseases is most often assessed late, during kernel filling. However, the lower leaves produce metabolites to form the top leaf layers and are a reserve pool during kernel filling (Kern, 1985), by which diseases before kernel filling may cause damage. Effects of early epidemics or of mildew on lower leaves are reported by Klischowski and Beyer (1980), Royse et al. (1980), Kolbe (1982) and in barley by Bar (1977), Carver and Griffiths (1982), Sander (1983), and by Lim and Gaunt (1986a).

Disease management. No significant effects of year, cultivar or soil type on the damage function of mildew was found in this study, but deviations may occur presumably with rather early and heavy epidemics. Damage averaged 0.013 (SE = 0.003) kg are^{-1} per pustule-day of mildew per leaf from second node stage to early dough at yields of 7 to 9 t ha^{-1}. No maximal damage level was observed at disease stresses from zero to two thousand pustule-days of mildew per leaf (Tammes, 1961). However, the disease stress itself becomes gradually limited, because a leaf can carry only a limited amount of pustules at an instant and its life-time will be shortened at high disease intensities. No indication was found that the damage function depends on yield level. But, the low yielding experiments reached early dough more early than the high yielding (Fig. 2; Glynne, 1951; Angus and Moncur, 1985). As a consequence, an equal disease intensity at each instant will result in a lower amount of pustule-days in a low-yielding crop than in a high-yielding crop. The simple damage function: -0.013 MD in kg are^{-1} and MD in pustule-days of mildew per leaf (DC32-83) may therefore be used in disease management systems at yields from 7 to 9 t ha^{-1} and MD < 2000, if duration to reach early dough is predicted from e.g. nitrogen supply. If duration to reach early dough is fixed in the management system, like in EPIPRE, then the function: -0.016 MD, in % yield and MD in pustule-days of mildew per leaf may be used. Damage caused by very early epidemics, that is before the second node stage, could not be detected in this study.

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Samenvatting

Beschrijving van meeldauwprofielen en schaderelaties bij lichte aantastingen van meeldauw in veldproeven met wintertarwe

Opbrengstderving van wintertarwe door meeldauw werd bestudeerd in 11 veldproeven in Nederland. De schade bedroeg gemiddeld 0.013 (SE = 0.003) kg are^{-1} per puistdag meeldauw per blad, vanaf het tweede-knoop stadium tot begin deegrijp bij opbrengstniveaus van 70 tot 90 kg are^{-1}, in de blanco. Bij een ziektestress van 0 tot 2000 puistdagen meeldauw per blad werd geen afwijking van een rechtlijnig verband gevonden. De schaderelatie werd niet significant beïnvloed door de verschillende jaren, rassen of grondsoorten. Het effect van meeldauw op enkele opbrengstcomponenten werd aangetoond.

Meeldauwprofielen in de onbehandelde veldjes konden worden beschreven met de vergelijking: $CM = CLA^b$, waarin CM het cumulatieve aantal puistjes is en CLA het cumulatieve bladoppervlak, beide berekend van bovenin het bladerdek naar beneden, de totalen gestandaardiseerd op één. De gradiënt parameter b bedroeg gemiddeld 3.4 (SE = 0.9). Waargenomen verschillen in steilte van de meeldauwprofielen beïnvloedde de schaderelatie niet aantoonbaar.

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