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Pathosystem management of powdery mildew in winter wheat

R.A. Daamen

PATHOSYSTEM MANAGEMENT OF POWDERY MILDEW IN WINTER WHEAT

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Abstract

Winter wheat cropping has changed considerably over the years 1974-1986 in the Netherlands. Yield has been increased from 5 to 8 ton/ha, due to short-strawed cultivars and higher levels of agrochemical inputs. The changes were described. Epidemics and damage relations of powdery mildew were studied, to construct an advisory system.

In surveyed fields, mildew intensity in spring was correlated with mean temperature in October and with the average temperature during December, January, February and March and with the susceptibility of the cultivars grown. The development of mildew during May-July was correlated with the area sown with a cultivar and with its susceptibility. The high variation in mildew intensity between fields points to the need of disease monitoring in management systems.

To assess mildew intensity, pustule counts were made and the fraction of diseased leaves (I, incidence) was determined (Neth. J. Pl. Path. 92: 197-222). In fields, mean pustule number per leaf m, may be estimated from I by: $\ln(m) = 1.48 + 1.14 \ln(\ln[1/(1-I)])$. Errors of estimates were studied.

Damage by mildew was studied in 13 field experiments (Neth. J. Pl. Path. 94: 69-80 and 95: 85-105). The mildew profile in the canopy was described by a model with one parameter, for which an estimate was given. Damage was described by the function: -0.013 (SE = 0.003) kg/are per pustule-day of mildew per leaf, from second node stage to early dough at yield levels of 70 to 90 kg/are. After 1980, farmers were advised to control mildew based on data from these experiments and from the surveys.

To upgrade this advisory system a simulation approach was used to explore effects of weather on yield and damage (Neth. J. Pl. Path., in press). Yields simulated were compared with yields harvested. The difference in performance of two models could be attributed to the simulation of early growth. Mildew was introduced in a model by quantification of five parameters, taking the spatial distribution of mildew into account. Computed damage approached but underestimated measured, especially in early epidemics, by which the advisory system could not yet be upgraded.

The question arose to what extend the increased level of fertilization affected epidemics of pests and diseases (J. Phytopathology 125: 305-319). A comparison of different farming systems revealed that higher levels of fertilization stimulated epidemics of mildew, yellow rust, snow mould, leaf miners and cereal leaf beetles, while five other pathogenic species were not affected. An integrated approach towards cereal cropping and breeding is discussed.

Additional keywords: Triticum aestivum, Erysiphe graminis, Blumeria graminis, surveys, monitoring, epidemiology, resistance, profile, damage, simulation, agrochemicals, wheat ideotype.

NN08201, 1336

1. Duurzame landbouw betekent permanente zorg.

2. Ieder systeem van geïntegreerde akkerbouw vereist monitoring van ziekten en plagen. In alle teeltkundig- en veredelingsonderzoek is daarom waarneming van ziekten en plagen onontbeerlijk.

   dit proefschrift

3. Jaarlijks dient de cultivar van een akkerbouwgewas, waarin de grootste hoeveelheid bestrijdingsmiddelen is toegepast, onderscheiden te worden met een bintje aan een lintje.

4. De verordening van het landbouwschap dat in Zeeland geen wintergerst verbouwd mag worden is een klassiek voorbeeld van een preventieve maatregel om het optreden van meeldauw in zomergerst tegen te gaan.

5. Bladgroei en bladsterfte zijn in de epidemiologie van bladziekten verklarende en geen afhankelijke variabelen.

   dit proefschrift

6. In de lijst van gewasbeschermingskundige termen wordt 'gewasbescherming' gedefinieerd als 'het geheel van maatregelen gericht op het beneden aanvaardbare grenzen brengen of houden van *ziekten, plagen* en andere schadelijke factoren bij de teelt van gewassen en het beheer van (andere) vegetaties'. Dat de termen 'gewasvrije periode' en 'gewasrotatie of vruchtwisseling', als maatregelen om ziekten, plagen en het gebruik van pesticiden te beheersen ontbreken en dat in de lijst een 'gewasbeschermingsmiddel' synoniem is met een (chemisch) bestrijdingsmiddel is 'gewasbeschermingsknuddig'.

   Commissie voor de Terminologie van de Nederlandse Planteziektenkundige Vereniging, 1985. Lijst van gewasbeschermingskundige termen. Gewasbescherming 16, suppl.nr. 1.

7. Als in een teelt van tarwe het tarwestro nog groen is terwijl de korrels oogstrijp zijn, dan is de bemesting te ruim geweest en daarom in strijd met het streven naar minimalisatie van emissie van voedingsstoffen. Bovendien is de kans op schotterige tarwe groot en wordt het gebruik van loofdoodmiddelen uitgelokt.

8. De naam van de afgeleide eenheid 'vierkante meter' impliceert het bestaan van een coherent systeem van minimaal drie eenheden die van de grond-eenheid 'meter' zijn afgeleid. Daarom moet de are als eenheid van grond-oppervlak (areaal) worden gehandhaafd.

K. Schurer & J.C. Rigg, 1980. Grootheden en eenheden in de landbouw en de biologie. Pudoc, Wageningen.

9. Dat de tabel 'Voorzieningsbalansen granen, hoeveelheden' is opgedeeld in balansen voor tarwe, rogge, gerst, haver, mais, overige granen (o.a. sorghum en millet), voedergranen en besloten wordt met: totaal granen, zonder rijst, geeft meer verwarring dan dat boekweit op een pak Bambix groei-ontbijt voor het gemak een graan wordt genoemd. De verwarring kan worden voorkomen door in de tabel de categorie voedergranen af te voeren en door rijst op te nemen.

Landbouwcijfers 1988. LEI-CBS. 171-173.

10. Om ecologische rampen, zoals die veroorzaakt door DDT, in de toekomst te vermijden zal naast de toelatingscriteria van bestrijdingsmiddelen: zoals de giftigheid en de persistentie, ook de onbeheersbaarheid van het middel een criterium moeten zijn, vooral als het bestrijdingsmiddel een populatie van genetisch gemanipuleerde organismen is.

Stellingen bij proefschrift: Pathosystem management of powdery mildew in winter wheat.
R.A. Daamen, Wageningen, 14 februari 1990.

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Part I
INTRODUCTION

Powdery mildew and winter wheat

Chapter 1

General introduction

Winter wheat is, in rotation with potatoes and sugar beet, an important arable crop in the Netherlands. A widespread fungal disease of winter wheat is powdery mildew (*Erysiphe graminis* DC. f.sp. *tritici* [Em. Marchal] = *Blumeria graminis* (DC.) Speer). Epidemics of powdery mildew cause yield loss, hereafter named damage. Epidemics of powdery mildew could be limited by use of cultivar resistance, avoiding the excessive use of nitrogen fertilizers and by avoiding early sowing. After 1977, effective fungicides became available in the Netherlands to control mildew. By chemical control, epidemics of mildew are suppressed and damage due to mildew is avoided. No information was available on the financial loss due to mildew at the level set by the expenses for chemical control. In 1979, the Research Institute for Plant Protection (IPO) and the Netherlands Grain Centre (NGC), initiated a research project with the aim to identify the mildew intensity at which the damage by mildew equals the costs of chemical treatment. The author was appointed to execute this project in conjunction with the conduction of the systematic annual surveys of diseases and pests in Dutch cereal fields. Members of the Steering Committee for these activities were: Ir. M. de Boer, Ir. G.E.L. Borm, Ir. T.E.J.L.W.B. de Bruin, Dr. ir. A. Darwinkel, Ir. B.A. ten Hag, Ir. F.H. Rijsdijk, Dr. ir. A. Tempel and Ir. E. Ubels. After 1981, the research emphasis shifted from powdery mildew to glume blotch (*Leptosphaeria nodorum*), another disease of wheat. As a consequence, the aim of the project was not attained, since research on the epidemiology of powdery mildew and the effectivity of fungicides was not completed. A link was maintained between the research on powdery mildew and glume blotch and the surveys, conducted by the author, and the EPIPRE-project, initiated by the Department of Phytopathology of the Wageningen Agricultural University and the NGC, and conducted by Ir. F.H. Rijsdijk. The original aim of the EPIPRE-project was to implement a yellow rust (*Puccinia striiformis*) disease management system, in 1979 broadened into a management system of diseases and pests of wheat. After 1981, the link was transfered to the EPIPRE-project of the Research Station for Arable Farming and Field Production of Vegetables, conducted by Ir. K. Reinink.

Outline of this thesis. In Chapter 2, winter wheat cropping and the occurrence of powdery mildew over the years 1974-86 in the Netherlands is described. Part II concerns methods to estimate powdery mildew intensity. In Chapter 3 the precision of mildew pustule counts is described. This method was used in field experiments to assess mildew intensity. In Chapter 4, incidence counts of powdery mildew (percentage leaves with mildew) are studied in relation to assessments of pustule numbers. Incidence counts are used by farmers and researchers to assess mildew intensity roughly. In Part III, damage by powdery mildew is studied in field experiments. The damage in relation to wheat cultivars and nitrogen fertilization is treated in Chapter 5. The effect of the period of the mildew epidemic in the ontogeny of the crop, on damage is described in Chapter 6. In addition, the profile of mildew in the crop is described in both chapters. In Part IV, the physiological processes which underlay damage are discussed. In Chapter 7 the effect of weather on wheat yield is treated. In Chapter 8, damage by mildew is simulated on the basis of physiological data and compared to damage measured in the field at different weather conditions. In Part V, the epilogue, epidemics of powdery mildew and of other diseases and pests in winter wheat are studied in different cropping systems, and discussed in relation to an integrated design of arable farming.

Chapter 2

Wheat cropping and powdery mildew in the Netherlands

The adoption of short-strawed wheat cultivars together with the application of growth regulators and the increased use of fertilizers and pesticides, highlighted the agronomic importance of cereal diseases and pests and the variation in their occurrence (Dilz et al., 1982). Linked to the footrot survey in the Netherlands (Van der Spek et al., 1974) and stimulated by the surveys of foliar diseases in England and Wales (King, 1973), systematic annual surveys of diseases and pests in commercial fields were conducted from 1974 to 1986, to describe the occurrence of these varying biotic constraints to yield. The results of these annual surveys have been described by Van der Beek (1975, 1976, 1977 and 1978), Borm (1978), Daamen et al. (1980, 1981 and 1982), Daamen and Wietsma (1983 and 1984), Stol (1985), Versluis (1985) and Van den Hoek (1986). In this paper, a short description of wheat cropping during 1974-86 is given and a compilation of these survey reports is made to describe the occurrence of powdery mildew in relation to weather and winter wheat cropping practices.

Wheat cropping

Areas. In arable farming in the Netherlands, small grains are most often grown in rotation with potatoes and sugar beet, and sometimes with pulses (mainly peas) and other crops, mainly grass seed, oilseed rape and flax (Fig. 1). Fodder crops, predominantly maize, are usually grown on sandy soils in areas where animal production is intensive. Fodder maize is usually not grown in rotation with potatoes, sugar beet and small grains. In the period 1974-86, the area under small grains decreased by one third. The dominant small grains were winter wheat and spring barley, with 120,000 and 40,000 ha, respectively. When weather conditions in autumn were bad, as in 1974, the harvest of potatoes and sugar beet and the seedbed preparation was delayed by which winter wheat was partly replaced by spring cereals (Fig. 1). Winter wheat is mainly cropped on marine clay soils in the northern, central and southwestern parts of the Netherlands. Other minor areas of winter wheat cropping are 3,000 ha on river clay soils in the central part of the country, 4,000 ha on loess soils in the south, 5,000 ha

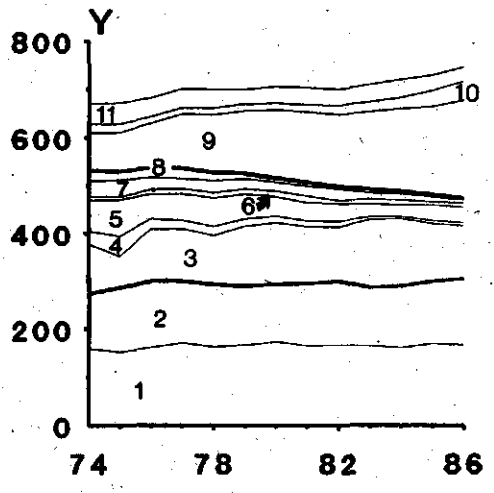


Fig. 1. Cumulative area of agricultural crops (Y, in 10^3 ha) during 1974-1986.

1. POTATOES
2. SUGAR BEET
3. WINTER WHEAT
4. SPRING WHEAT
5. SPRING BARLEY
6. WINTER BARLEY
7. OATS
8. RYE
9. FODDER CROPS
10. PULSES
11. OTHER CROPS

on improved peat soils in the northeastern and 4,000 ha on sandy soils in the eastern part of the country. Winter wheat yield per hectare increased from 5 to 8 ton. The annual national wheat production increased from 0.7 to 1 megaton in these years. The national production is negligible compared to the annual world production of 519 megaton (CIMMYT, 1989). In these years, about equal amounts of the national production were used as human food, as animal feed or for export (NGC, annual reports). Annual net import of bread wheat and wheat products amounted 0.6 megaton on average in these years. This import was mainly used to fulfil the national wheat consumption of 1 megaton food and partly to fulfil the national wheat consumption of 0.4 megaton feed (Annual reports, produktschap voor granen, zaden en peulvruchten).

Cropping practices. After ploughing and seedbed preparation, winter wheat is usually sown in the second half of October. In any year, some fields are sown very early, in September, and some fields very late, in December, but these are exceptions. The seed rate was 170 kg/ha in 1974. In response to the improvements of seed certification and seed disinfection, the seed rate decreased to 130 kg/ha in 1980 (Noordam and Van der Ham, 1979) but it increased afterwards to 155 kg/ha, indicating the gradually intensification of wheat cropping. The row distance was circa 25 cm, to enable mechanical weeding and growth of an undersown crop (Darwinkel, 1979), but it decreased to 10-15 cm during 1974-86.

The use of nitrogen fertilizer increased from 100 to 160 kg N per ha during 1974-86 (Noordam and Van der Ham, 1979). Phosphate and potassium fertilizers are usually applied in the potato crop. The estimated uptake of these fertilisers by wheat increased from 40 to 70 kg P_2O_5 and 20 to 80 kg K_2O on clay soils in this period.

Weeds were mainly controlled by herbicides. Fungicides have become commonly used since 1974, growth regulators since 1977 and aphicides since 1979. An indication of the costs of these cropping practices expressed as percentages of gross financial returns, averaged for marine clay soils, is given in Fig. 2. In this period, these direct extra costs, without the costs of labour and machinery, increased from circa 20 to 30% of the financial returns. Costs of weed control and crop protection were negligible in 1973 but amounted to 10% of the financial returns in 1986.

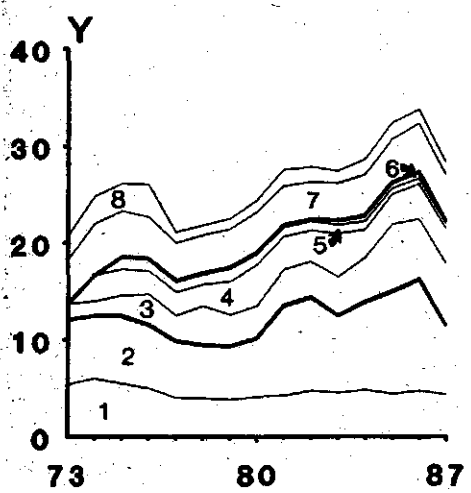


Fig. 2. Cumulative direct extra costs (Y) of winter wheat production as percentages of gross financial returns in 1973-1987, excluding costs of labour and machinery.

1. SEED
2. FERTILIZERS
3. HERBICIDES
4. FUNGICIDES
5. GROWTH REGULATORS
6. INSECTICIDES
7. ROPE, DRYING AND CLEANING
8. INSURANCE AND INTEREST

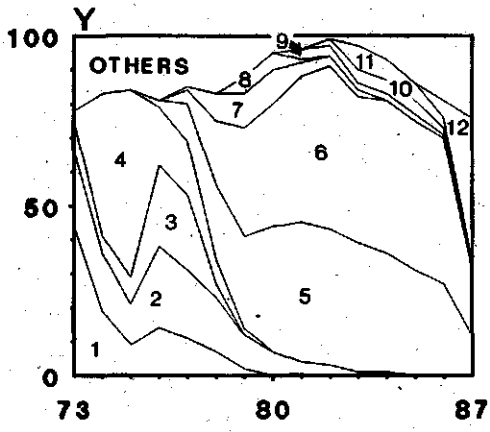


Fig. 3. Area of winter wheat cultivars (Y, as percentage of total winter wheat area) during 1973-1987.

1. MANELLA
2. CARIBO
3. LELY
4. CLEMENT
5. OKAPI
6. ARMINDA
7. NAUTICA
8. DURIN
9. MARKSMAN
10. SAIGA
11. CITADEL
12. OBELISK

Cultivars. Data on cultivars were obtained from the cultivar lists of the Government Institute for Research on Varieties of Cultivated Crops (RIVRO). In 1974, the dominant winter wheat cultivar Clement became susceptible to yellow rust (*Puccinia striiformis*). After the yellow rust epidemic of 1975 it was replaced by the older, but more yellow rust resistant cultivars Caribo, Lely and Manella (Fig. 3; R.W. Stubbs, pers. comm.). Lely became susceptible to yellow rust in 1975. In 1976, yellow rust was nearly absent due to the dry and warm summer. Lely was replaced, after the yellow rust epidemic of 1977, in 1978 by cv. Okapi, moderately yellow rust resistant and by cv. Arminda, yellow rust resistant at the adult plant stage. Okapi and Arminda were dominant during the decade whilst their resistances lasted. These cultivars were replaced by the complete yellow rust resistant cv. Obelisk in 1987.

In this period the crop stature changed. The tall cultivars Manella and Caribo and the rather tall cvs. Lely and Clement were replaced by the tall cv. Okapi and the semi-dwarf cv. Arminda, which in turn were replaced by the semi-dwarf cv. Obelisk in 1987. Total production of biomass of tall or semi-dwarf cultivars is about the same. Kernel yields of semi-dwarfs are on average higher than that of tall cultivars, as semi-dwarfs have on average a higher harvest index (Van Dobben, 1962) and they can endure higher levels of nitrogen before they lodge. The dwarf cultivars Durin, Marksman and Donata were highly suscepible to ear blight (*Fusarium spp.* and *Monographella nivalis*) and to a lesser extent also to *Septoria spp.* (Ubels et. al., 1980), which limited their agricultural value. These dwarfs never exceeded more than ten percent of the winter wheat area in this period.

On average the winter wheat cultivars vary from susceptible to moderately susceptible to mildew (Table 1). At their release, they are most often already susceptible to mildew, whilst being resistant to yellow rust. Exceptions were the cultivars Clement, Arminda, Durin and Marksman, whose partial mildew resistance degraded in two to four years after their release. This is a common phenomenon in the cereal-mildew pathosystem (Wolfe, 1984 and Barrett, 1988). The genetic variability in powdery mildew populations is maintained by spontaneous mutations and by sexual and asexual recombination (Leijerstam, 1972 and Menzies and MacNeill, 1986). The variability of mildew resistance in wheat cultivars will increase by recent interspecific recombinations of *Triticum spp.* (Moseman et al., 1984 and Van Silfhout, 1989).

Table 1. RIVRO mildew resistance ratings of winter wheat cultivars[1]

Cultivar	Year 74	75	76	77	78	79	80	81	82	83	84	85	86
Manella	5	5	5	5	5	5	5	-	-	-	-	-	-
Caribo	4	4	4	4	4	4	4	4	4	4	-	-	-
Lely	4	4	4	4	4	-	-	-	-	-	-	-	-
Clement	8	7	5	5	4	3	3	-	-	-	-	-	-
Okapi	-	-	-	4.5	4.5	4.5	4.5	4.5	4.5	4.5	5.5	5	5
Arminda	-	-	-	7	7	6	6	6	6	6	6	6	6
Nautica	-	-	-	6	6	6	6	6	6	6	6	-	-
Durin	-	-	-	-	-	7.5	7.5	7	7	-	-	-	-
Marksman	-	-	-	-	-	-	-	7	7	7	7	7	8
Saiga	-	-	-	-	-	-	-	-	6	6	6	6.5	6.5
Citadel	-	-	-	-	-	-	-	-	-	6	6	6	6.5
Granada	-	-	-	-	-	-	-	-	-	-	6.5	6	6.5
Obelisk	-	-	-	-	-	-	-	-	-	-	-	-	6.5

[1] Higher ratings indicate greater resistance.

Table 2. Estimates by farmers of average annual kernel yield (ton ha^{-1} at 16% humidity) and of pesticide use in the fields surveyed.

Year[1]	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984
Number of fields	141	78	66	102	136	389	746	831	911	896	845
Yield	6.6[2]	5.4	6.1	5.9	7.2	6.5	6.9	7.4	8.0	7.6	8.6
Percentage of fields sprayed to control:											
Eyespot	28	9	4	9	-[3]	3	14	10	8	9	22
Leaf diseases[4]	9	41	21	63	-	51	19	55	22	86	51
Leaf & ear d.[4]	60	77	79	89	140	48	80	84	113	160	145
Aphids[4]	4	10	51	36	17	81	76	84	101	100	100

[1] 1974-1978: data derived from the fields surveyed, 1989-1984 data derived from fields in the EPIPRE system from which a limited number were surveyed.
[2] Based on 82 fields of the survey.
[3] Sprays to control diseases not separated and lumped under leaf & ear diseases.
[4] Sprays to control leaf & ear diseases and aphids are most often combined in one treatment.

Surveyed fields. Estimates of average annual kernel yield and pesticide use in the fields surveyed in the period 1974-84 are given in Table 2. These data were derived from the surveyed fields in the period 1974-1977 (D. van der Beek, pers. comm.) and from the EPIPRE fields (Rijsdijk and Hoekstra, 1979, Rijsdijk et al., 1980a, 1980b, 1981, Drenth and Reinink, 1982 and 1984 and Reinink, 1985) and from the data-bases of the EPIPRE advisory system. Such data are not available for the years 1985 and 1986. An increase of gross kernel yield and an increase of chemical crop protection during 1974-84 was found. Fields were selected by the disease specialists of the extension service during 1974-77 and from the EPIPRE fields (Zadoks,

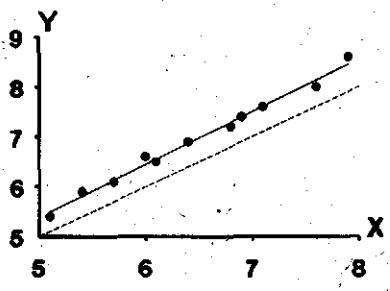


Fig. 4. Estimates of average annual kernel yield of the fields surveyed (Y, ton/ha at 16% moisture content) in relation to average annual national yield (X, ton/ha at 17% moisture content), in the period 1974-1984. The drawn line is the regression line, the broken line is the 1:1 relation.

1981 and 1984; Rijsdijk, 1983; Rijsdijk et al., 1989; Rabbinge and Rijsdijk, 1983 and 1984; Blokker, 1983; Reinink, 1984 and 1986; Rossing et al., 1985, Drenth et al., 1989; Drenth and Stol, 1990) during 1978-84. Comparison of the average annual yield recorded for the surveyed fields (Table 2) with the national average yield (Statistical Yearbooks, CBS-LEI) is given in Fig. 4. It shows that the surveyed fields yielded slightly above the national average.

In the period 1974-84, circa 12% of the fields were sprayed, mainly during the first half of May, to control eyespot (*Pseudocercosporella herpotrichoides*), in 42% of the fields leaf diseases before ear emergence were controlled, and in 94 % of the fields leaf and ear diseases after ear emergence were controlled. On average, 60% of the fields were sprayed to control aphids, mainly *Sitobion avenae*. Sprays against leaf and ear disease and aphids consisted most often of a mixture of a fungicide and an insecticide. In this period, it was also common practice in the Netherlands to add the fungicide maneb as a tank mix with other pesticides, especially with insecticides. During 1974-84, the number of tank mixes applied per field increased from 1 to more than 2 per field.

The use of fungicides to control eyespot has not significantly increased. Decisions to control eyespot are based on field inspections and an economic threshold. The use of fungicides to control leaf or leaf and ear diseases increased, but shows variation between years. To illustrate this effect, the use of fungicides to control leaf and/or leaf and ear diseases, as percentages of fields treated, is plotted against an estimate of average annual damage. This was estimated as the effect of one fungicide application at flowering in the RIVRO trials in these years (Fig. 5). The figure shows that in years when fungicides were highly profitable in the RIVRO trials, especially in 1983 and 1984, the use of fungicides in the surveyed fields was high. Thus, the gradual increase of chemical crop protection, as indicated in Table 2, was confounded with high disease intensities in the years 1983 and 1984.

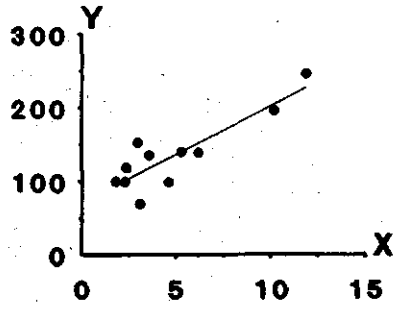


Fig. 5. Estimated annual use of fungicides to control leaf and/or leaf and ear diseases (Y, use in percentage of fields treated, see Table 2) in relation to an estimate of damage (X, kg/are), the effect of one fungicide application at flowering on clay soils in RIVRO trials (H. Bonthuis and K. Roodenburg, pers. comm.), in the period 1974-1984.

The use of insecticides to control aphids increased but also showed variation between years (Rabbinge and Carter, 1984). Aphids are relatively more damaging in high yielding crops compared to low yielding crops (Roermund et al., 1986; Rossing, submitted).

In Table 3, the percentage of fields is given in wich certain active ingedients of fungicides and insecticides were used in the surveyed fields during 1974-84. These data were derived from the surveyed fields during 1974-1977 (D. van der Beek, pers. comm.) and from the data-bases of the EPIPRE advisory system during 1980-1984. The benzimidazoles benomyl, carbendazim and thiofanaat-methyl were gradually replaced by the ergosterol biosythesis inhibitors, the morpholines tridemorph and later fenpropimorph,

Table 3. Pesticide use in winter wheat as percentage of fields in which the specified pesticide was sprayed. Mixtures are counted twice or more depending on the number of compounds, see Table 2.

Year	1974	1975	1976	1977	1980	1981	1982	1983	1984
Number of fields[1]	141	78	66	102	746	831	911	896	845
Fungicides:[2]									
Benomyl	52	24	15	13	6	3	2	5	8
Benodanil	0	0	0	18	0	0	0	0	0
Carbendazim	35	50	67	84	51	27	31	13	17
Captafol	0	0	0	3	1	16	40	78	77
Captan	0	0	0	0	0	1	2	4	0
Ethirimol	0	1	3	5	0	0	3	1	0
Fenpropimorph	0	0	0	0	0	5	4	34	55
Maneb	60	87	80	121	90	108	118	141	149
Mancozeb	7	8	11	3	+	+	+	+	0
Prochloraz	0	0	0	0	0	0	0	2	1
Propiconazole	0	0	0	0	0	+	+	26	22
Pyrazophos	0	0	0	0	2	2	3	+	1
Sulphur	11	12	15	17	3	3	4	5	4
Thiabendazole	0	0	0	0	1	1	+	0	0
Thiophanate-methyl	3	4	8	7	5	2	1	1	+
Triadimefon	0	0	0	1	59	73	40	82	66
Triadimenol	0	0	0	0	0	0	0	0	+
Tridemorph	1	39	6	56	+	0	0	0	0
Triforine	0	0	0	1	1	1	+	+	1
Zineb	1	0	0	4	6	3	+	+	0
Ziram	1	3	0	0	0	0	0	0	1
Insecticides:									
Demephion	0	0	3	2	4	2	1	0	0
Dimethoate	1	1	27	12	30	33	42	39	36
Formothion	0	5	0	0	+	+	1	1	1
Fosfamidon	0	0	0	0	1	1	1	1	1
Heptenophos	0	0	0	0	0	0	+	1	1
Oxydemeton-methyl	1	1	15	2	4	2	3	4	4
Parathion	1	3	0	1	1	1	2	1	1
Pirimicarb	1	1	11	14	36	43	47	45	51
Thiometon	0	0	0	0	1	3	4	4	4

[1] 1974-1977, surveyed fields; 1980-1984, all EPIPRE fields from which survey fields were selected and data were complete.

[2] +: used in < 0.5 % of the fields.

and by the triazoles triadimefon and propiconazole. The hydroxypyrimidine ethirimol, a selective mildew fungicide, was not commonly used in winter wheat. The carboxamide benodanil was only used in 1977 when rusts were widespread. Sulphur and the dithiocarbamates mancozeb, zineb and ziram but not maneb were replaced by the above mentioned ergosterol biosynthesis inhibitors and by the phtalimide captafol. The use of the dithiocarbamate maneb increased, despite its negligible agricultural value (Ten Heggeler, 1985). Maneb was cheap and because of its manganese content it was only partly regarded as a fungicide and partly as a fertilizer, so it persisted.

Application of ethirimol and tridemorph to control mildew was low. Tridemorph was mainly used to control rusts in 1975 and 1977. After 1978, triadimenol was mainly used to control mildew before heading and to control mildew, brown rust and speckled leaf blotch after heading. Before heading, rusts were nearly absent after 1977. Mildew strains less sensitive to triadimefon were found from 1981 onwards in the Netherlands (De Waard et al., 1986). A decrease over time in sensitivity of the mildew population to current fungicides is a common phenomenon (Wolfe, 1984). To retard this development, Dutch farmers were advised after 1981 to alternate the use of triazoles with the morpholine fenpropimorph (Hollomon, 1982).

Insecticides came into general usage to control aphids. The use of cheap organophosphorous compounds, mainly dimethoate, decreased slightly in favour of the more expensive but selective aphicide, the carbamate pirimicarb.

Weather. Mean monthly temperatures and total monthly precipitation during 1974-86 are given in Fig. 6a,b, together with averages of the period 1950-80. Data were derived from the monthly reports of the Royal Netherlands Meteorological Institute (KNMI), from five main stations for temperature, and from all meteorological stations for precipitation. On average, temperature is lowest in January-February and highest in July-August, but Fig. 6a illustrates that the mean monthly temperature varies considerably. Precipitation shows an annual trend, superseded by the annual variation (Fig. 6b).

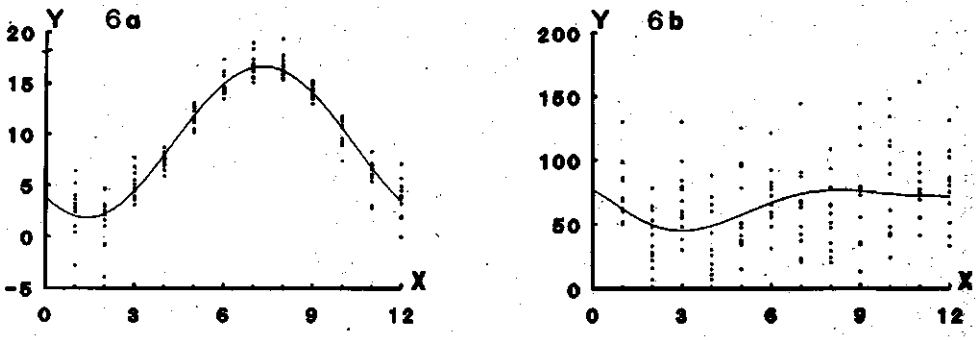


Fig. 6a,b. Mean monthly temperature (Fig. 6a, Y in ^{o}C) and total monthly precipitation (Fig. 6b, Y in mm) in the years 1974-1986, X is number of month. The lines are averages over the years 1950-1980.

Table 4. Annual prevalence (% fields infected) and average annual incidence (% leaves or glumes infected) of powdery mildew on winter wheat.

Year	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986
Number of fields surveyed													
May:	143	88	89	105	124	305	219	132	123	107	176	-	-
July:	143	88	94	105	124	129	164	138	152	143	123	94	94
Prevalence													
May:													
Leaves	26	12	20	61	-	17	38	38	1	47	27	-	-
July:													
Leaves	80	75	82	74	-	47	38	72	48	71	90	88	79
Glumes	70	37	55	62	-	6	5	24	8	2	38	7	11
Incidence													
July:													
Leaves[1]	-	4	3	6	2	2	0	[1] 12	7	9	22	13	14
Glumes	-	4.9	0.6	3.3	0.4	0.0	0.0	0.7	0.0	0.1	1.0	0.7	0.1

[1] 1975-1980 expressed as average % leaf surface diseased (severity); 1981-1986 as average % leaves with mildew (incidence).

Mildew

Field surveys. During 1974-86, the prevalence of mildew averaged 29% fields at first to second node stage, mainly in May (Table 4). Annual mildew prevalence in May was positively correlated with the mean temperature in October and with the average temperature over the months December, January, February and March (Fig. 7). High temperatures encourage earlier sowing, seedling development and mildew development, so that more fields may become infected (Last, 1953; Turner, 1956; Smedegard-Petersen, 1967 and Yarham et al., 1971). As the optimum temperature for mildew development is 15-20 °C, correlations of mildew prevalence in May with average temperature in winter and early spring are plausible. Mildew epidemics in individual fields depend accordingly on temperature (Stephan, 1984). In addition to these factors, a third factor (X_3), the average annual weighted mildew resistance

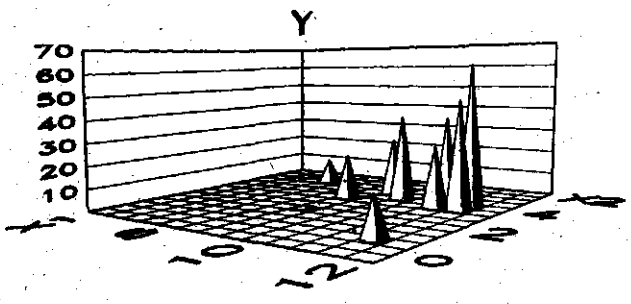


Fig. 7. Annual prevalence of mildew in May (Y, percentage of surveyed fields with mildew), in relation to average temperature in October (X_1, °C) and average temperature over the months December, January, February and March (X_2, °C). Regression:

$$Y = -132 + 12\ X_1 + 10\ X_2 \quad (R^2 = 0.83)$$

rating of the cultivars, had a significant negative correlation with the prevalence of mildew in May. An increase of one point in average resistance of the cultivars grown in the Netherlands was correlated with a decrease of 11% mildew prevalence in May, hardly changing the effect of the two other factors (see Fig. 7): $Y = -69 + 12X_1 + 11X_2 - 11X_3$ (R^2=0.94).

In July 1974-86, mildew was present on leaves in 70% of the fields, and on ears in 27% of the fields (Table 4). These annual prevalences of mildew on leaves and ears was not correlated with the annual mildew prevalence in May, presumably because different types and different amounts of fungicides were applied in these years (Table 2,3). The incidence of mildew on ears was low and averaged 1% glumes during 1975-86. Both annual prevalence and incidence of mildew on ears tended to decrease in this period (Table 4), presumably due to the increased use of fungicides. On leaves, mildew intensity averaged 3% leaf surface (severity) in July over the years 1975-80, and on average 13% of the leaves had mildew (incidence) in July averaged over 1981-86 (Table 4).

Annual mildew incidence on leaves (% leaves) and on ears (% glumes) in July was negatively correlated with average temperature during April, May and June, r=-0.94 and r=-0.54, respectively. Annual mildew prevalence (% fields) on ears was negatively correlated with total precipitation in March, April, May and June (r=-0.69). But annual mildew prevalence (% fields) and severity (% leaf surface) on leaves did not show such correlations with weather conditions in these early summer months nor with annual mildew prevalence in May. Though clear correlations of annual mildew prevalence in May with temperatures were found (Fig. 7), no consistent correlations of annual mildew intensity in July with weather nor with annual mildew prevalence in May were obtained. This is presumably due to the different use of fungicides in these years, which affected mildew intensity in July.

The relation of powdery mildew epidemics in spring and summer with weather are still under discussion. Graf-Marin (1934) reported that mature barley plants, which had already headed, became immune to mildew. In addition, he concluded that old leaves were more resistant to mildew infection than young leaves due to their thicker cuticle. Tapke (1951) reported that conditions that promoting a lush, tender growth of the crop further promote mildew epidemics and he concluded that this caused the inconsistent relationships between weather and mildew reported in the literature. Last (1953 and 1957) emphasized the dominant role that the host resistance has on mildew epidemics in spring and summer. Cereals are highly susceptible to mildew during rapid growth, whether due to nitrogen, temperature or different sowing dates. Alongside host resistance, mildew spore production and dispersal are favoured by periods with high temperatures, many hours of sunshine, high windspeed and low precipitation (Smith and Davies, 1973 and Polley and King, 1973 and Limpert et al., 1984). In addition to these factors, Channon (1981) and Dutzman (1985) added high relative humidity as a factor to identify high risk periods for spore production and dispersal. Subsequent processes of mildew infection and development are limited by high temperatures or heavy rain (Aust, 1973; Stephan, 1980 and Eckhardt et al., 1984). After detailed studies on powdery mildew of barley, Hau, Aust and Kranz (1983) concluded that our limited knowledge of the resistance of host tissue, which depends on leaf position, age, temperature and nitrogen, limits our understanding of the effect that weather has on cereal mildew epidemics in spring and summer. Thus, the effect of weather on powdery mildew epidemics in spring and summer is rather complex. It is questionable whether significant correlations between annual mildew intensity in July and mean monthly weather data would be

obtained if data unaffected by fungicides would have been available.

Observations by farmers. Regular observations on disease intensity were made by farmers participating in the EPIPRE advisory system. Average mildew intensity during the seasons 1981 to 1984 in the different cultivars are given in Fig. 8a,b,c,d. Average disease intensity was high early in May 1981 and 1983, intermediate in 1984 and low early in May 1982. As a consequence, many fields were sprayed with fungicides before flowering in 1981 and 1983 and relatively few in 1982 (Table 2). Rusts were nearly absent before flowering in these years. Mildew increased from early May till mid June. This increase was more pronounced in 1982 and 1984 than in 1981 and 1983. After mid June, the mildew intensity was rather stable in 1981, 1982, and 1984, and it decreased in 1983 (Fig. 8a,b,c,d). Mildew intensity in July was low in 1982, moderate in 1981 and 1983 and high in 1984. Average mildew intensities early in May and in July, as observed by the farmers, correspond with the mildew intensities in these periods in the more limited number of fields surveyed (Table 4). The use of fungicides to control leaf and ear diseases (Table 2) is not correlated with the mildew intensities observed by farmers in June-July. These fungicides, among which maneb, were also used to control other diseases than mildew, mainly brown rust and leaf and glume blotches. Even this abundant use of fungicides did not eliminate powdery mildew.

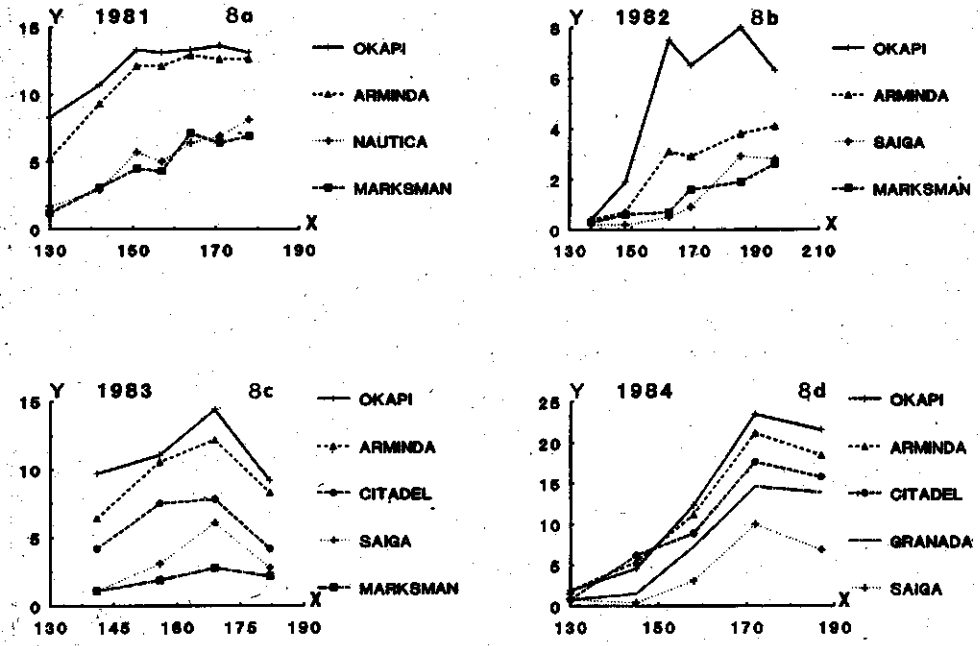
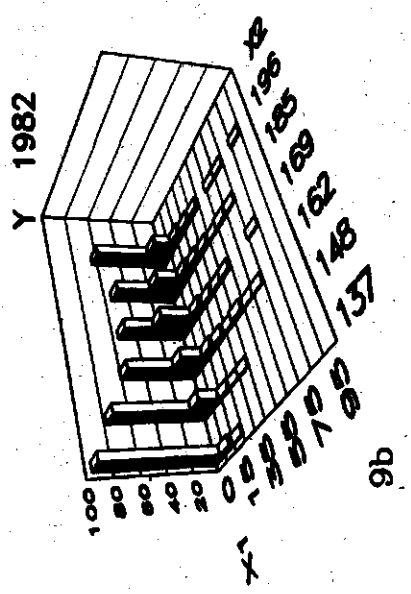
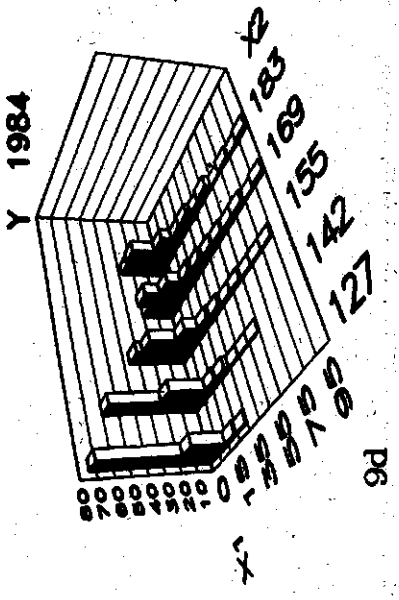
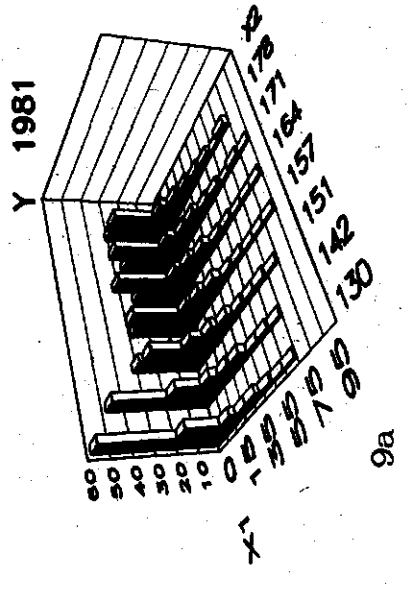


Fig. 8a,b,c,d. Average mildew intensity (Y, percentage leaves with mildew) in different winter wheat cultivars during the season (X, daynumber since 1 January) observed by EPIPRE farmers.







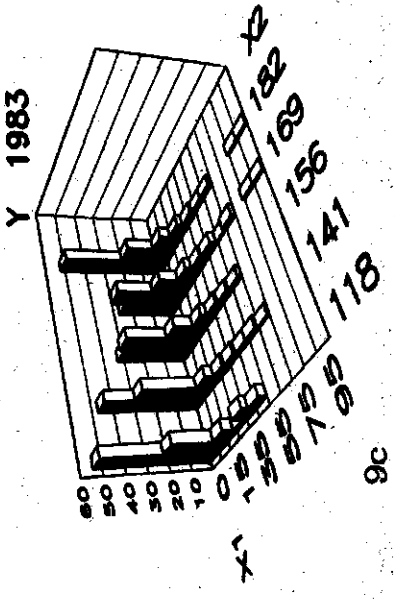


Fig. 9a,b,c,d. Percentage fields (Y) with different mildew intensities (X_1, number of leaves with mildew from 120 leaves) in time (X_2, daynumber since 1 January), observed by EPIPRE farmers.

The mildew susceptible cultivar Okapi, and to a lesser extend also Arminda (Table 1), both sown on a large area (Fig. 3), were most heavily mildewed (Fig 8a,b,c,d). Cultivar Marksman, moderately mildew resistant in these years and sown on a small area, had low mildew intensities in 1981, 1982 and 1983. Nautica, Saiga, Citadel and Granada, cultivars with a mildew resistance comparable to that of Arminda (Table 1) but sown on a small area (Fig. 3), were less mildewed than Arminda. Cultivar Nautica in 1981 and cv. Saiga in 1982 had about the same mildew intensity as the moderately resistant cv. Marksman. The scale effect (Wolfe, 1984; Limpert, 1987 and Barrett, 1988) is presumably caused by the difference in selection that the cultivars impose on the mildew population in the Netherlands. The cultivars Arminda and Okapi, sown on circa 80% of the area, mainly determined the genetic composition of the mildew population. The other cultivars differed presumably in the genetical basis of their resistance from Arminda and Okapi, and as their areas were small, they usually escaped severe epidemics.

Disease management. The occurrence of powdery mildew as described above reveals the well-known facts that powdery mildew epidemics can be limited by stubble management and delayed sowings, by which the frequence of primary infections of the winter wheat crop is lowered (Fig. 7). In addition, the use of many cultivars which differ in their origin of mildew resistance may limit mildew epidemics considerably (Fig. 8a,b,c,d). Despite these correlations with temperature and with cultivar resistance and area, the mildew intensity varied considerably between fields (Fig. 9a,b,c,d). Even during a severe mildew epidemic in the country, such as in spring 1983, circa half the number of fields were healthy. This variation stresses the need to avoid calendar sprayings and to develop disease management based on disease monitoring. In addition to monitoring, knowledge of the dynamics of disease development and of damage are necessary for economic decisions. A supervised control system, based on an economical rationale may help to lower the average pesticide use, and in the meantime more knowledge is obtained about diseases which are competing with people for food.

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Part II
METHODS

Measures of disease intensity

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Measures of disease intensity in powdery mildew (Erysiphe graminis) of winter wheat. 1. Errors in estimating pustule number

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Abstract

Assessments of pustule number and severity of powdery mildew on winter wheat in the Netherlands were made in commercial fields and in experimental plots. The sample variance (s^2) of the number of pustules per leaf (m) was fairly constant over years, varieties, growth stages and leaf postitions, but depended strongly on the average pustule number: $s^2 = 2 \cdot 2\bar{m}^{1.5}$. The effect of sample size on the precision of the estimate is discussed and it is concluded that it is difficult to estimate low disease intensities accurately. Estimates are given for the detection level of pustule counts in relation to sample size.

Mildew intensity on the lower surface of leaves can be estimated from the intensity on the upper surface. This method reduces the duration of the observation, but introduces an additional error. At low disease intensities and small sample sizes this method is more efficient than sampling mildew on both surfaces of leaves. The common practice of assessments of the upper surface of leaves only may not be the most efficient method.

Additional keywords: Triticum aestivum, epidemiology, sampling method, detection level.

Introduction

The choice of a disease assessment method is a difficult problem. No widely accepted method for foliar diseases of wheat is yet available, and the choice of a method is usually a compromise between the objectives and the resources available. A clear distinction can be made between the resources in an experiment and monitoring a disease in commercial fields. This paper considers the precision of counting pustules as a method to assess mildew *(Erysiphe graminis)* intensity. In a following paper, the precision of incidence counts as method to assess mildew intensity is discussed. To avoid confusion, intensity will be used in this paper to refer to the quantity of disease, irrespective of the assessment method; incidence is used when the intensity is expressed in proportion of leaves diseased and severity when intensity is in proportion of leaf surface diseased.

In studies of yield loss caused by powdery mildew in winter wheat in the Netherlands, assessments of disease were made by counting the number of pustules on leaves. There were three reasons for using this method. First, the aim was to estimate yield loss at low disease intensities; pustule counts are then not time-

consuming. Second, the assessment method should be defined exactly, so that it can be used independent of observer bias. Third, pustule counts can be transformed easily to the more common percentages, using disease assessment keys.

Rouse et al. (1980, 1981) studied the distribution of mildew pustules on wheat leaves. They found that the distribution on artificially inoculated seedling leaves fitted the negative binomial distribution, but on spontaneously infected leaves at the end of tillering the distribution did not fit any of the tested distributions. Koch (1978) described the precision of barley mildew assessments. He concluded that assessments of the percentage leaf surface diseased could not be used to estimate low disease intensities.

The objective of this paper is to examine the precision of mildew pustule counts on full grown wheat plants as a method of disease assessment at low disease intensities. Diseases in wheat are usually assessed only on the upper surface of leaves, although mildew can occur also on the under surface. Attention is given to the error introduced by observing the upper surface alone.

Materials and methods

Data. This paper is based on two data sets, one from commercial fields and one from field experiments, both in the Netherlands.

Commercial fields. In 1980 and 1981, 31 and 11 commercial fields, respectively, of different winter wheat varieties were sampled during May-June. A sample of 40 tillers per field was taken and the development stage determined (Zadoks et al., 1974). The top full grown leaf (flag leaf of adult plant) was designated leaf position 1. For each leaf position the number of mildew pustules was counted on the upper and lower surface of the leaves. In the same samples, leaves without disease were counted in the three upper positions. The number of disease-free leaves was counted like farmers will normally do in the field. All observations were made with the naked eye by experienced observers. Fields were sampled whether or not fungicides had been used.

Field experiments. From 11 field experiments (1980 to 1983), 31 sets of mildew pustules counts (upper leaf surface) in untreated plots were available. In each assessment at least 60 and usually 90 tillers were sampled. Average pustule number and sample variance were computed per leaf position for each sample. From the same pustule counts the number of leaves without disease was determined. The mildew assessments were were made at a speed of roughly 100 tillers per hour, at low disease intensities. The duration of the observation increased at higher disease intensities, and pustule numbers above 20 were estimated instead of counted.

Statistics. To obtain an estimate of the mildew severity, key 1.1.2 (Ubels and Van der Vliet, 1979) which is in common use in the Netherlands, was used to transform the average pustule number into percentage values. According to this key, for fully grown winter wheat, 5 pustules correspond to 1% severity.

Statistical analyses were performed by means of Genstat V, release 4.04 A and B (Alvey et al., 1983). Analyses of variance were carried out using multiple regression analyses with dummy variables for main effects only. For example the simple relation

between two variables, x and y, is described as:

$$y = a + b \cdot x + \text{error1} \quad (1)$$

and can be analysed by means of ordinary least squares regression to estimate a and b and the error with $n - 2$ degrees of freedom. If the relation between the variables depends on other factors, for example years with three levels ($i = 1, 2, 3$) and varieties with two levels ($j = 1, 2$), the relation for main effects only will have the form:

$$y(\text{year, variety}) = a(\text{year}) + a(\text{variety}) + [b(\text{year}) + b(\text{variety})] \cdot x + \text{error2} \quad (2)$$

The combined effect of years and varieties in this example can be evaluated by calculating the F-statistic over the sums of squares (SS) of models 1 and 2:

$$F = \frac{(\text{SSerror1} - \text{SSerror2}) / [2(i - 1) + 2(j - 1)]}{\text{SSerror2} / [n - 2 - 2(i - 1) - 2(j - 1)]} \quad (3)$$

by which the significance of factors (as in analyses of variance) can be evaluated in covariance designs. Since the factors may be partially confounded, this analysis is followed by a test on each individual factor, adjusted for the other factors. Chatterjee and Price (1977) gave a clear treatment of these tests.

To summarise the fit of a regression model, the ordinary R-squared statistic (R^2) will be used, computed as:

(SStotal − SSerror) / SStotal.

Results

Sample variance of pustule number per leaf. The sample variance of the population density of an organism is often variable, and dependent on the mean density. Taylor (1961) proposed a power function to describe the sample variance (s^2) in relation to population density (M):

$$s^2 = a \cdot M^b \quad (4)$$

in which a and b are parameters. This function is used to describe the relation between the sample variance [Var(m)] of the pustule counts from the upper surface of leaves and the mean number of pustules ($\overline{m}$) in the sample. Taking logs is a convenient way to stabilise variance and to estimate the parameters a and b:

$$\ln[\text{Var}(m)] = \ln(a) + b \cdot \ln(\overline{m}) \quad (5)$$

First, it was tested whether the relation was independent of years, varieties and growth stages, for each leaf position.

Analyses of variance on main effects (see Methods) were carried out, using the data set of the field experiments (Table 1). The results show no strong evidence for deviations due to years, varieties or growth stages. For the top leaves, these factors had a weak significant effect ($p < 0.05$). Further analyses showed that this deviation was due mainly to a year effect; in 1981 parameters a and b deviated (top leaf). In 1981 the attack of mildew was relatively severe and it is possible that the attention of the observers was weakened and the low disease intensities on the top leaf not properly observed. No data are available to analyse this deviation further. Since this deviation was slight and occurred on the top leaf in one year only, the parameters for each individual leaf

Table 1. Sample variance of mildew pustule counts. Analyses of variance to test for deviations from common a and b in $\ln[Var(m)] = \ln(a) + b \cdot \ln(\overline{m})$ due to years (1980 to 1983), growth stage of the crop (DC ≤ 59, DC > 59) and varieties (Arminda, Okapi, Caribo). Experimental plots (n = 32), forth leaf position n = 17.

Source of variation	df	Top leaf SS	Leaf 2 SS	Leaf 3 SS	Leaf 4	
					df	SS
1. common a and b	1	95.1	146.3	169.2	1	101.1
2. different a and b	12	2.3	2.1	2.2	12	0.8
2a. years	6	1.3	0.8	0.7	6	0.2
2b. growth stage	2	0.2	0.1	0.3	2	0.1
2c. varieties	4	0.4	1.2	0.2	4	0.4
3. residual	18	1.1	2.2	2.2	3	0.5
4. total	31	98.5	150.7	173.5	16	102.3
All factors	F 12/18	3.2*[1]	1.4ns[1]	1.5ns	F 12/3	0.4ns
Years	F 6/18	3.7*	1.1ns	1.0ns	F 6/3	0.2ns
Growth stage	F 2/18	0.2ns	0.3ns	1.5ns	F 2/3	0.4ns
Varieties	F 4/18	1.5ns	2.5ns	0.4ns	F 4/3	0.7ns

[1] ns = not significant ($p > 0.05$); * = significant at $p = 0.05$.

position are assumed constant over the years, varieties and growth stages. Estimates of the parameters and R^2 are given in Table 2. The fit of the descriptive model is good ($R^2 > 0.96$). Parameters a and b have the same magnitude for the four leaf positions and do not differ significantly between leaf positions ('a posteriori testing'). The conclusion is that average values of a and b can be used, irrespective of the leaf position. Thus, the sample variance of pustule counts [Var(m)] can be described by:

$$Var(m) = 2.2\ \overline{m}^{1.55} \qquad (6)$$

To illustrate the relation, the data for the third leaf are plotted in Fig. 1.

Sample size and precision of estimation. The variance of the mean pustule number

Table 2. Sample variance of pustule counts. Estimates of ln(a) and b in $Var(m) = \ln(a) + b \cdot \ln(\overline{m})$ and their standard deviations; and an estimate of a and its 95% confidence limits. Experimental plots n = 32, fourth leaf n = 17.

Leaf position	ln(a)	b	R^2	a
top leaf	0.87 (0.09)	1.49 (0.05)	0.97	2.4 (2.9-2.0)
2nd leaf	0.78 (0.07)	1.56 (0.05)	0.97	2.2 (2.5-1.9)
3rd leaf	0.79 (0.09)	1.55 (0.05)	0.97	2.2 (2.6-1.8)
4th leaf	0.64 (0.08)	1.61 (0.05)	0.99	1.9 (2.3-1.6)
Average	0.81 (0.03)	1.55 (0.02)	0.98	2.2 (2.4-2.1)

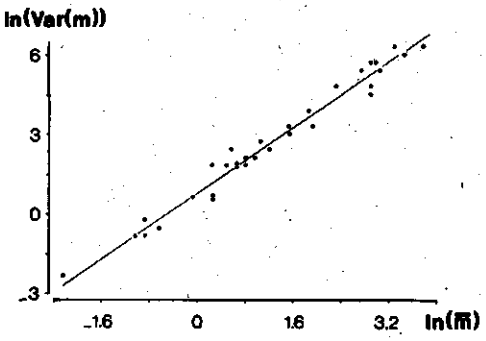

Fig. 1. Sample variance, log-transformed (ln[Var(m)]) in relation to log mean pustule number ($\ln(\overline{m})$), upper surface third leaf position. Regression: $\ln[\mathrm{Var}(m)] = 0.789 + 1.55 \ln(\overline{m})$; $R^2 = 0.97$.

$[\mathrm{Var}(\overline{m})]$ on the upper surface of a leaf from a sample of size n will be estimated as usual by:

$$\mathrm{Var}(\overline{m}) = \mathrm{Var}(m)/n$$

substitution of (6) gives:

$$\mathrm{Var}(\overline{m}) = \frac{2.2\,\overline{m}^{1.55}}{n} \qquad (7)$$

Approximate confidence limits for mean pustule number can be computed by:

$$\overline{m} \pm u_p\sqrt{\mathrm{Var}(\overline{m})} \qquad (8)$$

in which u_p is the $(100 - 0.5p)$ percentile of the normal distribution; p was arbitrarily chosen as 5%. The approximate confidence limits for sample means at low disease intensities at fixed sample sizes (15 and 30), were computed from (7) and (8) (Table 3). The width of the 95% confidence limits depends strongly on the disease intensity.

To estimate the sample mean with a certain precision, a particular minimum sample size is required. The coefficient of variation (CV) can be used as a measure of precision:

$$CV = \frac{\sqrt{\mathrm{Var}(\overline{m})}}{\overline{m}} \qquad (9)$$

As the sample mean $(\overline{m})$ is an estimate of the population mean (M), (7) and (9) can be used to estimate the required sample size n at different population densities. Substitution of (7) into (9) gives Equation 10, for estimation of the required sample size, n:

$$n = \frac{2.2M^{-0.45}}{CV^2} \qquad (10)$$

In Fig. 2 the relation between the required sample size and the population mean is given for fixed coefficients of variation. If $M < 20$, a coefficient of variation of the mean of 0.1 is not attainable with $n < 50$. Apparently, in wheat powdery mildew the sample error is large, especially at low disease intensities. The detection level of pustule counts is therefore of special interest.

The detection level is defined as the disease intensity M_d (in pustules per leaf), at

Table 3. Estimated 95% confidence limits for mean pustule number ($\overline{m}$) on the upper surface of leaves, for two sample sizes (n).

$\overline{m}$	n = 15		n = 30	
	lower	upper	lower	upper
1	0.2	1.8	0.4	1.6
3	1.1	4.9	1.7	4.3
5	2.1	7.9	3.1	6.9
7	3.3	11	4.5	9.5
9	4.5	14	6.0	12
11	5.7	16	7.5	15
13	7.0	19	9.0	17
15	8.3	22	10	20
17	9.6	24	12	22
19	11	27	13	24

which there is a 97.5% probability of presence of the disease on a sample of n leaves. So at least 1 pustule should be present in a sample of n leaves, thus at average 1/n pustules per leaf. Assuming approximate normality of the mean pustule number, the detection level M_d can be estimated by:

$$\frac{1}{n} = M_d - u_p\sqrt{Var(M_d)}$$

where u_p is the 97.5 percentile of the normal distribution ($u_p = 1.96$) and $Var(M_d)$ can be estimated by (7). Hence:

$$\frac{1}{n} = M_d - 1.96\,\frac{\sqrt{2.2\,M_d^{1.55}}}{n} \qquad (11)$$

Equation 11 is solved by iteration. A sample size of 15 leaves gives a detection level of $M_d = 0.5$ pustules per leaf, and for 30 leaves $M_d = 0.2$ pustules per leaf. So mildew intensities below 0.5 pustules per leaf cannot be properly estimated from samples of 15 leaves or fewer.

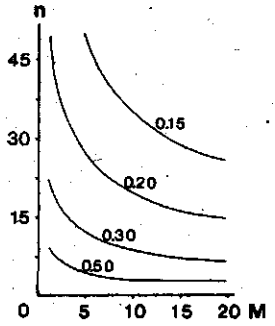

Fig. 2. Minimum sample size (n, leaves) required to estimate the pustule number (M) per leaf with a certain precision. The values plotted are the different coefficients of variation of the mean.

Sampling powdery mildew on the upper and lower surface of leaves. It is common practice in cereals to assess diseases on the upper surface of leaves only, although mildew can occur on both surfaces. If mildew on both surfaces of the leaves is of interest (e.g. causing damage), the assessment of upper surface only causes a systematic underestimation of the mildew population of interest. If there exist a relation between the mildew intensity on the upper and under surface of leaves, then it is possible to predict the mildew intensity on both surfaces from the intensity on the upper surface of leaves. Such a prediction introduces an additional error. On the other hand, let us assume that this procedure is twice as quick as the direct estimate. In other words, it allows for a doubling of the sample size and a gain in precision due to a reduction of the sampling variation. The question is now whether the error due to sampling a limited part of the population (upper surface leaves only) can be neglected when compared with the reduction of the error due to sampling variation. This question can be answered by comparison of two variance estimates. One is when pustules are counted at both surfaces of n leaves. The other is the variance of the predicted pustule number on both surfaces from the pustule number on the upper surfaces of 2n leaves.

A direct estimate of the sample variance of the total pustule number (m_t) on both surfaces of the leaf was obtained from 46 samples of the data set of commercial fields (using Equation 5). Estimates were: ln(a) = 1.17 (sd. 0.084; a = 3.2); b = 1.51 (sd. 0.048); R^2 = 0.96. The b-value in (4) can thus be assumed to describe the sample variance of total pustule number (m_t) and pustule number on upper surface (m_u, see Table 2), but the a-values cannot. So the variance of the mean total pustule number $\overline{m}_t$ is described by:

$$\mathrm{Var}(\overline{m}_t) = \frac{a_t \cdot \overline{m}_t^{\,b}}{n_t}\ ;\ a_t = 3.2;\ b = 1.53 \qquad (12)$$

The next step is to describe the variance of the predicted total pustule number ($\overline{m}_t$) from the pustule number at the upper surface only. Suppose that the ratio between mean pustule number on lower surface ($\overline{m}_l$) and upper surface ($\overline{m}_u$) of leaves is constant:

$$\frac{\overline{m}_l}{\overline{m}_u} = c \qquad (13)$$

For each sample of the data set of commercial fields, the ratio (c) of pustule number at lower and upper surface was calculated. Analyses of variance were carried out for each leaf postition, to check for constancy of the parameter c for the different years, varieties and growth stages. The results of these analyses showed no significant deviations from a common parameter c. The estimates of c for each leaf postition have about the same magnitude (Table 4); that for the third leaf is lower than those for the other leave positions, but this difference is not significant ('a posteriori testing'). The ratio, c, of the pustule numbers is thus independent of the leaf position. The estimate of a common c for the top four leaves is: c = 0.52 (sd. 0.04); the residual variance equals 0.23. For illustration, parameter c is plotted in Fig. 3 in relation to the pustule number on the upper surface for the third leaf position.

Table 4. Relation of mean pustule number on lower ($\overline{m}_l$) and upper surface ($\overline{m}_u$) of leaves. Fitted values for c (= $\overline{m}_l/\overline{m}_u$) and its standard deviation. Commercial fields n = 42, fourth leaf n = 41. Top leaf and second leaf 15 and 3 observations, respectively, with c = 0/0 were excluded.

Leaf position	c	
top leaf	0.58	(0.16)
2nd leaf	0.56	(0.05)
3rd leaf	0.47	(0.05)
4th leaf	0.52	(0.06)
Average	0.52	(0.04)

The total pustule number ($\overline{m}_t$) can be predicted from the pustule number on the upper surface of leaves ($\overline{m}_u$) using (13):

$$\hat{\overline{m}}_t = (1 + c)\cdot\overline{m}_u \tag{14}$$

The variance of this new estimate may be calculated by adding the average conditional variance and the variance of conditional average (Rao, 1965, p. 97):

$$\begin{aligned}\mathrm{Var}(\hat{\overline{m}}_t) &= \mathrm{E}\cdot\mathrm{Var}(\hat{\overline{m}}_t|\overline{m}_u) + \mathrm{Var}[\mathrm{E}(\hat{\overline{m}}_t|\overline{m}_u)] \\ &\approx \mathrm{Var}(1 + c)\cdot[\mathrm{Var}(\overline{m}_u) + \overline{m}_u^2] + (1 + c)^2\cdot\mathrm{Var}(\overline{m}_u)\end{aligned} \tag{15}$$

The variance of $\overline{m}_u$ is described by (7) and the variance of c was 0.23. Assume that sampling the upper surface alone is twice as quick as sampling the whole leaf. Sampling the upper surface alone on 2n leaves is a better strategy than sampling n whole leaves if the variance from equation 15 is less than or equal to the variance of equation 12:

$$\frac{a_u\cdot\overline{m}_u^b}{2n}\cdot[(1 + c)^2 + \mathrm{Var}(c)] + \overline{m}_u^2\cdot\mathrm{Var}(c) \leqslant \frac{a_t\cdot(1 + c)^b\cdot\overline{m}_u^b}{n}$$

or when

$$n \leqslant 14.25\,\overline{m}_u^{-0.47} \tag{16}$$

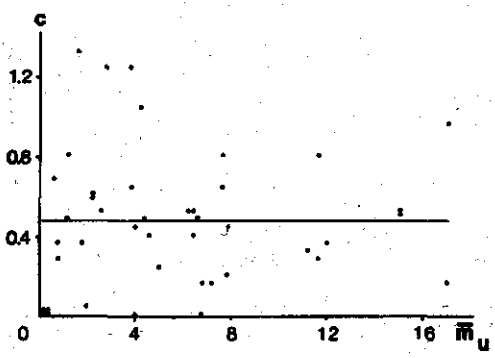

Fig. 3. Ratio (c) of mean pustule number on lower ($\overline{m}_l$) and upper surface ($\overline{m}_u$) of the third leaf, plotted against mean pustule number on the upper surface. Ratio: $\overline{m}_l/\overline{m}_u$ = 0.47 (sd. = 0.05).

As the sample mean $\overline{m}_u$ is an estimate of the population mean M_u, (16) may be used to estimate the breakpoint sample size at different mildew intensities. The breakpoint (n) indicates the sample size at which both methods are equal in precision, at lower sample sizes it is more efficient to double the sample size and to inspect the upper surface of the leaves only. For a range of M_u from 1 to 20 pustules per leaf, n was calculated using (16):

M_u	1	5	10	15	20
$n \leqslant$	14	6	4	4	3

Thus only for low disease intensities and small sample sizes is it more efficient to double the sample size and to inspect the upper surface only. For example; inspection of the upper surface of 12 leaves instead of both surfaces of 6 leaves is efficient at disease intensities of up to 5 pustules per (upper surface) leaf.

Discussion

No statistical frequency distribution has yet been found to describe the dispersion of powdery mildew pustules on leaves. Rouse et al. (1981) found that the negative binomial gave the closest fit, though it still deviated significantly, and it has been shown that only the dispersion of pustules among artificially inoculated seedling leaves fits the negative binomial (Rouse et al. 1980). Nevertheless, the sampling error of pustule counts was adequately described using Taylor's (1961) power function. This description does not yield more insight into the underlying dispersal process, but that was not the aim of this paper.

Koch (1978) found that in barley powdery mildew, the sampling error of assessments of the percentage leaf surface diseased became unacceptably large at disease severities below 2%. The sampling error of wheat powdery mildew pustule counts depends also on the disease intensity. The error is large at intensities below 5 pustules per leaf (1% severity). Even using pustule counts it is difficult to determine low disease intensities, but at least the error can be estimated.

The error introduced by sampling only the upper surface of leaves and prediction of mildew intensity on the lower surface instead of direct assessments on both surfaces has been estimated, and suggests that at low disease intensities (< 5 pustules per leaf) and small sample sizes (< 12 leaves) it is more efficient to sample the upper surface only instead of both surfaces. In other situations it is better to sample both surfaces and to halve the size of the sample.

In this paper the occurrence of mildew on the leaf sheaths was not accounted for. Estimates based on examination of only the upper surface of leaves will become very inefficient if the whole mildew population on a plant or crop is of interest. Assessment of the total pustule number per tiller may be far more accurate than the sampling of the upper surface of leaves which is currently the common practice.

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Samenvatting

Meting van meeldauwaantastingen (Erysiphe graminis) in wintertarwe. 1. Schattingsfouten van het aantal puistjes

Aantallen puistjes meeldauw per blad werden geteld in praktijkpercelen en veldproeven met wintertarwe. De steekproefvariantie van het aantal puistjes was tamelijk constant in de jaren, rassen, gewasstadia en bladposities, maar was sterk afhankelijk van het gemiddeld aantal puistjes ($\overline{m}$): $s^2 = 2{,}2\,\overline{m}^{1{,}5}$. Het effect van de steekproefgrootte op de nauwkeurigheid van de schatting wordt besproken en het blijkt dat het moeilijk is om lichte aantastingen nauwkeurig te schatten. Er worden schattingen gegeven van de detectiegrens in afhankelijkheid van de steekproefgrootte.

Meeldauwaantastingen aan de onderkant van het blad, kunnen worden geschat uit de aantasting op de bovenkant van het blad. Deze methode levert een tijdsbesparing op, maar ook een extra onnauwkeurigheid. Alleen bij lichte aantastingen en kleine steekproeven is deze methode efficiënter dan een directe tweezijdige bemonstering. Het schatten van meeldauw op de bovenkant van bladeren is, hoewel algemeen gebruikelijk, waarschijnlijk niet de meest efficiënte methode.

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Measures of disease intensity in powdery mildew (Erysiphe graminis) of winter wheat. 2. Relationships and errors of estimation of pustule number, incidence and severity

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Abstract

Assessments of pustule number, incidence and severity of powdery mildew on winter wheat in the Netherlands were made in commercial fields and in experimental plots. Assuming a constant leaf and pustule size, a leaf can carry at most M_m pustules. If the number of clusters, each consisting of n pustules, follows a binomial distribution, then the relation between incidence (I, proportion diseased leaves) and severity (S, proportion diseased leaf surface) is: $(1 - S) = (1 - I)^{n/M_m}$. The model explains the main effects of leaf, pustule and cluster size on incidence-severity relations and gave a good description of the measured relation. The negative binomial distribution poorly described the measured relation. The model of Nachman (1981) gave a good description of the relation between mean pustule number ($\overline{m}$) and incidence (I). The relation found in commercial fields, irrespective of fungicide treatments, was: $\ln(\overline{m}) = 1.48 + 1.14 \ln(\ln[1/(1 - I)])$. The effects of years, varieties, growth stages and leaf positions on this relation were not significant. Incidence assessments can be used to predict the pustule number, but this method is less efficient than the use of direct pustule counts. Estimates are given of the variance of the predicted pustule number.

Additional keywords: Triticum aestivum, epidemiology, sampling method, detection level, multiple infection.

Introduction

In a previous paper the precision of counting pustules as a method to assess mildew *(Erysiphe graminis)* intensities in field experiments was evaluated. Pustule counts can be used easily by a farmer to monitor crops with low mildew intensities, but this method cannot be used for all the diseases he may need to monitor. Recently, incidence assessments (proportion of leaves with disease) have been used in disease and pest monitoring by farmers (Rabbinge and Mantel, 1981; Rijsdijk, 1982; Zadoks, 1984). Incidence assessment is simple, and, since the same method can be used for different diseases and pests, it might facilitate the adoption of supervised control systems. Seem (1984), reviewing disease incidence-severity relations, pointed out that no consensus existed on the various assessment methods. He proposed that severity should be defined as 'the quantity of disease affecting entities within a sampling unit'. This is a very broad definition including both absolute and relative measures. However such a distinction

is needed in this paper. Following Seem (1984), with modifications, the following terms will be used:
intensity is used to refer to the quantity of disease, irrespective of the assessment method;
pustule number is the disease intensity expressed as the number of pustules on a leaf, so it can be considered as a relative density;
severity is the disease intensity expressed as the proportion of leaf surface which is visibly diseased;
incidence is the disease intensity expressed as the proportion of leaves which are visibly diseased.

James and Shih (1973) studied the disease incidence-severity relation for powdery mildew in commercial wheat fields. They concluded that incidence can be used to estimate severity at low disease intensities. Rouse et al. (1981) studied the incidence-severity relation for the same disease in experimental plots. They used the model of James and Shih (1973), but found different parameter values. Ward et al. (1985a, b) studied the precision of two sampling methods for cereal aphids and concluded that incidence sampling was not significantly less accurate than direct counting.

The objective of this paper is to analyse and describe the incidence-severity relation for mildew in the Netherlands. The precision of this relation when used as a calibration line in supervised control systems is evaluated and compared to the precision of direct pustule counts.

Materials and methods

Data. This paper is based on two data sets, one from commercial fields and one from field experiments, both in the Netherlands. A description of the assessment methods and statistics used is given by Daamen (1986).

Sample variance of incidence. Let D be the number of diseased leaves in a random sample of size n, from a field in which a proportion p of the leaves is diseased. Then D will follow a binomial distribution with parameters p and n. The sample variance of the proportion of leaves with disease (I) is given by:

$$\text{Var (I)} = \frac{p \cdot (1 - p)}{n} \qquad (1)$$

In contrast to the direct or pustule counts, the sample variance of incidence is independent of the disease species. Statistical tables are available for confidence limits of proportions (Rohlf and Sokal, 1969).

The detection level of the sampling method depends only on the sample size. The detection level is defined as that disease incidence (p_d) at which there is a 97.5% chance of finding the disease in a sample of n leaves:

$$(1 - p_d)^n = 0.025 \qquad (2)$$

Solving (2) gives for $n = 100$ a detection level $p_d = 0.036$; and with $n = 500$, $p_d = 0.0074$. Thus sampling 100 leaves from a field in which 3.6% of the leaves are diseased

results in a probability of 2.5% that the sample is clean.

Incidence-severity models. It is reasonable to assume that spores from a cloud landing on a crop are randomly distributed over the leaves. If each landed spore causes disease, then the relation between the mean incidence (I) and the mean pustule number M can be described using the zero term of the Poisson distribution (multiple infection transformation, see Gregory, 1948):

$$(1 - I) = e^{-M} \tag{3}$$

If pustule and leaf size are constant, and a leaf can carry a maximum of M_m pustules, the relation between pustule number and severity (S, proportion leaf surface diseased) will be:

$$S = \frac{M}{M_m} \tag{4}$$

Substitution of (4) into (3) yields the incidence-severity relation:

$$(1 - I) = e^{-M_m \cdot S} \tag{5}$$

Spore dispersion is random only at the start of an epidemic in a field. Normally clustering will occur, owing e.g. to the failure of spores generated by a pustule to escape from a leaf. Assume that clustering results in clusters with a constant number, n, of pustules. The mean number of clusters on a leaf, N, equals M/n. Then a random distribution of clusters on the leaves following a Poisson distribution will give the incidence-severity relation:

$$(1 - I) = e^{-N}; \; N = \frac{M}{n} \tag{6}$$

Elimination of N gives:

$$(1 - I) = e^{-\frac{M}{n}} \text{, or } (1 - I) = e^{-\frac{M_m}{n} \cdot S} \tag{7}$$

Model 7 is in fact equivalent to (5), it merely considers clusters of n pustules rather than single pustules with a Poisson distribution.

The assumptions of a Poisson distribution are not violated statistically when disease intensity is low. At higher disease intensities the probability that two or more cluster-initiating spores land in the same place and form only one visible cluster cannot be neglected. Moreover, a leaf cannot carry more than N_m clusters ($N_m = M_m/n$). If A cluster-initiating spores land at random on a leaf, each potential cluster site receives, on average, A/N_m cluster-initiating spores. The resulting mean number of visible clusters (A_v) on a leaf will be (Poisson distribution, see also Justesen and Tammes, 1960):

$$A_v = N_m(1 - e^{-\frac{A}{N_m}}), \text{ or } e^{-A} = (1 - \frac{A_v}{N_m})^{N_m} \quad (8)$$

in which $A_v = M/n$; M is the number of visible pustules per leaf; and $N_m = M_m/n$. Substitution of (8) into (6) yields:

$$(1 - \frac{A_v}{N_m})^{N_m} = (1 - I); \quad A_v = \frac{M}{n}, \quad N_m = \frac{M_m}{n}$$

Elimination of A_v and N_m and substitution of (4) yields the incidence-severity relation:

$$(1 - S) = (1 - I)^{\frac{n}{M_m}} \quad (9)$$

where S and I are the severity and incidence in proportions, n is the cluster size (in pustules) and M_m is the maximal number of pustules on a leaf.

Model 9 can also be derived in one step, assuming that a leaf can carry a = 0, 1, 2....N_m clusters of n pustules ($N_m = M_m/n$). If the number of clusters per leaf follows a binomial distribution, the probability of sampling a leaf without clusters will be:

$$p(a = 0) = (1 - \frac{A_v}{N_m})^{N_m}; \quad N_m = \frac{M_m}{n}, \quad A_v = \frac{M}{n} \quad (10)$$

and (10) can be rewritten as incidence-severity relation 9, because $p(a = 0) = (1 - I)$ and $M/M_m = S$ (4). A Poisson distribution will approach the binomial distribu-

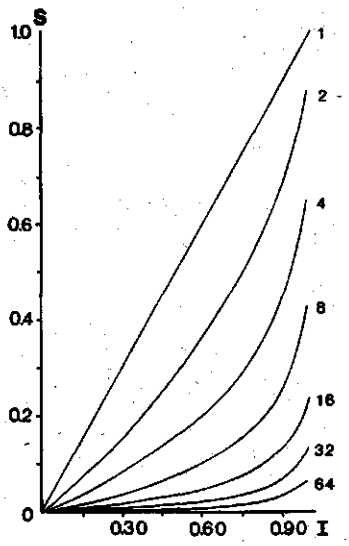

Fig. 1. Incidence (I) and severity (S) relations generated by the binomial model (see text). The data in the figure represent the maximal number of clusters per leaf.

tion when N_m is large and A_v is small, then (7) may be considered as a special case of (9).

Incidence-severity relations, generated by the binomial model (9) are shown in Fig. 1. All the generated incidence-severity relations pass through the origin. The relation simplifies to the straight line S = I, if one cluster destroys the whole leaf ($n = M_m$). When all leaves in an infinitely large population are diseased; I = 1 with solution S = 1. Since n/M_m is usually much smaller than unity, the slope of S is very steep near I = 1 (Fig. 1). For example when a leaf can carry 10 clusters of size n ($n/M_m = 0.1$) and 98% of the leaves are diseased (I = 0.98), then S is estimated as 0.32, or roughly one third of the leaf surface is diseased.

James and Shih (1973) proposed the exponential incidence-severity relation:

$$I = (1 - r^{100 \cdot S}); \quad 0 < r < 1 \tag{11}$$

in which parameter r defines the slope of the exponential relation and S represents the severity as a proportion, which equals M/M_m (4). Thus (11) can be rewritten:

$$(1 - I) = e^{100 \frac{M}{M_m} \cdot \ln (r)}; \quad 0 < r < 1 \tag{12}$$

and becomes (7) when:

$$\frac{M_m}{n} = -100 \cdot \ln (r), \text{ or } r = e^{-\frac{M_m}{100 \cdot n}} \tag{13}$$

Models 7 and 11 are thus equivalent; they differ only in their underlying assumptions and the meaning of the parameters. James and Shih (1973) do not explicitely state the underlaying assumptions of the model. The parameter, r, of their model is difficult to interpret, but it can be calculated (13) from the parameters cluster size (n) and the maximal number of pustules on a leaf (M_m), assuming mildew clusters follow a Poisson distribution. Since model 7 is a special case of (9), so is model (11).

Nachman (1981) proposed the following relation between incidence and mite density:

$$(1 - I) = e^{-t \cdot M^u} \tag{14}$$

in which t and u are parameters and M is the mean density, or, in the context of mildew infestations, the mean pustule number. Nachman (1981) derived the model without explicit assumptions about any statistical distribution. Comparison of (14) and (7), shows that (7) is a special case of (14). If parameter u of equation 14 equals unity, the models are equal and the parameter n (cluster size) is then the inverse of parameter t of the Nachman model.

Waggoner and Rich (1981) concluded that the distributions of diseases they studied usually followed the negative binomial distribution. Their incidence-severity relations were generally also described by the first term of the negative binomial:

$$(1 - I) = (\frac{M}{k} + 1)^{-k} \tag{15}$$

where k is a positive parameter indicating the degree of clustering. Comparison of models 7, 9 and 14 with model 15, do not reveal any condition under which the models are equal. If parameter k of (15) approaches infinity, (15) becomes (3), but that is not interesting.

Results

Estimation of parameters for the incidence-severity relations. The binomial incidence-severity relation (9), the Nachman model (14) and the negative binomial (15) were fitted to the data. Since the data are taken from samples of the unknown disease incidence and severity in the field, both variables are measured with error. Here, we consider the data now as exact measurements (at least the incidence on the samples); the error of estimation will be treated in the next section. Taking logs, model 9 is rewritten:

$$\ln\left(\frac{1}{1-S}\right) = \frac{n}{M_m} \cdot \ln\left(\frac{1}{1-I}\right) \qquad (16)$$

The ratio n/M_m is treated as one parameter: q. The model has a test condition; the described relation must pass through the origin when fitted to the data. Inspection of residuals revealed heteroscedasticity; by trial and error a fourth root transformation was found to stabilize the variances. Analyses of variance were carried out on the data sets of commercial fields and experimental plots to test for deviation of the intercept from zero (test condition) and for variation in q among years, varieties and growth stages (Table 1). For both data sets it can be assumed that q is constant: neither test revealed a significant difference. For the data set of experimental plots, however, the

Table 1. Analyses of variance to test for dependence of the incidence severity relation: $\ln(1-S) = q * \ln(1-I)$, in years, growth stage (DC ⩽ 59; DC > 59) and varieties (Arminda, Okapi, Caribo). Commercial fields n = 42, years 1980, '81; experimental plots n = 32, years 1980 to 1983. Analysis of the transformed model, see text.

Source of variation	Comercial fields			Experimental plots		
	df	SS	F	df	SS	F
1. common q	1	2.900		1	2.286	
2. intercept	1	0.002	1.3ns[2]	1	0.022	15.6***[2]
3. residual	40	0.050		30	0.045	
4. different q	4	0.006	1.2ns			
4a. years	1	0.004	3.1ns			
4b. growth stage	1	0.000	0.0ns			
4c. varieties	2	0.002	0.6ns			
5. residual	36	0.044				
6. total[1]	42	2.952		32	2.352	

[1] Not corrected for the mean.
[2] ns = not significant ($p > 0.05$); *** = significant at $p = 0.001$.

Table 2. Fitted values for the parameters of the incidence severity relation in the transformed model: $\ln[1/(1 - S)] = q \cdot \ln[1/(1 - I)]$, see text; their standard deviations; and estimates of q and its 95% confidence limits.

Location	df	q * *0.25	R^2	q
commercial fields	41	0.305 (0.006)	0.98	0.008 (1.010-0.007)
experimental plots	31	0.312 (0.009)	0.97	0.009 (0.012-0.007)

relation does not pass through the origin. The intercept is estimated as +0.11 (s.e.: 0.03). After retransformation the incidence-severity relation does not pass through the origin, as the severity is 0.00017 (= 0.017 percent) at zero incidence. Though the deviation is statistically significant, it is negligibly small from a biological point of view. The disease intensities in the experimental plots covered a greater range than those in the commercial fields, where fungicides were used to control severe outbreaks of mildew. Because of this greater range, a significant deviation of the intercept from zero can be detected more easily.

When the equation is forced through the origin, the estimates of parameter q (n/M_m) do not differ significantly for the two data sets (see Table 2). The R^2 values are high, so the transformed model gives a good description of the data. As M_m was 500 (see Methods), estimates of the average cluster size (n) are: 4 for the commercial fields and 4.5 for the experimental plots. It is interesting to compare the estimates of q with the estimates of parameter r in Canada (James and Shih, 1973). The estimates of parameter r ranged from 0.30 to 0.67 for individual leaf positions. These values correspond with values of q ranging from 0.008 to 0.025 (13). The value estimated here of 0.009 for the three top leaves is within this range. Another comparison can be made with the data of Rouse et al. (1981), who used the model of James and Shih (11) to describe the relation for a field experiment in the USA. They estimated a parameter value which corresponds to a very large value of q: 0.076 (see Discussion). Zaharieva et al. (1984) described incidence-severity relations for mildew on winter wheat in Bulgaria. The parameters they estimated can not be compared with the estimates of q, as they used other models to describe the incidence-severity relation.

The more complex Nachman model (14) was also fitted. Taking logs and redefining the parameters, the model was rewritten:

$$\ln(\overline{m}) = a + b \cdot \ln\left[\ln\left(\frac{1}{1 - I}\right)\right] \qquad (17)$$

This model is linear with the parameters $a = (1/u) \cdot \ln(1/t)$ and $b = 1/u$. Analyses of variance were carried out to test for deviations due to years, varieties or growth stages from common parameters a and b, see Table 3. Residuals showed no heteroscedasticity. The results of these tests showed no significant deviations due to years, varieties or growth stages. The estimates of the parameters are given in Table 4. An estimate of 1/t of the original model (14) is given, because this is an estimate of the average cluster size if u equals 1, as discussed above. For the experimental plots the parameters were also estimated for the individual leaf positions. No significant effects of leaf posi-

Table 3. Analyses of variance to test for dependence of the relation between incidence and mean pustule number: $\ln(\overline{m}) = a + b \cdot \ln(\ln[1/(1 - I)])$, on years, growth stage (DC ⩽ 59; DC > 59) and varieties (Arminda, Okapi, Caribo). Commercial fields n = 42, years 1980, '81. Experimental plots n = 32, years 1980 to 1983.

Source of variation	Commercial fields			Experimental plots		
	df	SS	F	df	SS	F
1. common a and b	1	74.5	240.7*** [1]	1	74.4	271.9***
2. residual	40	12.4		30	8.2	
3. different a and b	8	3.3	1.5ns [1]	12	4.6	1.9ns
3a. years	2	1.4	2.5ns	6	2.4	2.0ns
3b. growth stage	2	0.4	0.7ns	2	0.2	0.4ns
3c. varieties	4	0.9	0.8ns	4	1.0	1.2ns
4. residual	32	9.1		18	3.6	
5. total	41	86.8		31	82.6	

[1] ns = not significant; *** = significant at p = 0.001.

Table 4. Estimated parameters of the relation between incidence (I) and mean pustule number ($\overline{m}$) in the Nachman model: $\ln(\overline{m}) = a + b \cdot \ln(\ln[1/(1 - I)])$, their standard deviations; and an estimate of 1/t, see text.

Leaf position	df	a	b	R^2	1/t
three top leaves					
commercial fields	40	1.48 (0.11)	1.14 (0.07)	0.86	3.6
experimental plots	30	1.63 (0.12)	1.42 (0.09)	0.90	3.2
single leaf layers experimental plots					
top leaf	30	0.89 (0.10)	1.19 (0.03)	0.98	2.1
2nd leaf	30	0.90 (0.08)	1.23 (0.05)	0.95	2.1
3rd leaf	26	1.07 (0.07)	1.37 (0.06)	0.95	2.2
average	88	0.99 (0.05)	1.24 (0.02)	0.97	2.2

tion on estimated parameters were found, so the estimates of common parameters a and b are also given. As an illustration a plot of data from the commercial fields is given (Fig. 2). The R^2 values are high, so the transformed model gives a good description of the data.

For the three top leaves together, estimates of parameter b from the experimental plots and from the commercial fields differ significantly. A small value of b indicates that pustule number increases less with increasing incidence than is the case with a larger value of b. The difference in estimates of b is in agreement with the difference in the measurement of incidence: on both surfaces of the leaves in the commercial fields and on the upper surface only in the experimental plots. For the commercial fields, the estimate of b is close to unity, and then models 9 and 14 are nearly the same. For this reason the conditions required to fit model 9 were satisfied in the commercial fields but not in the experimantal plots.

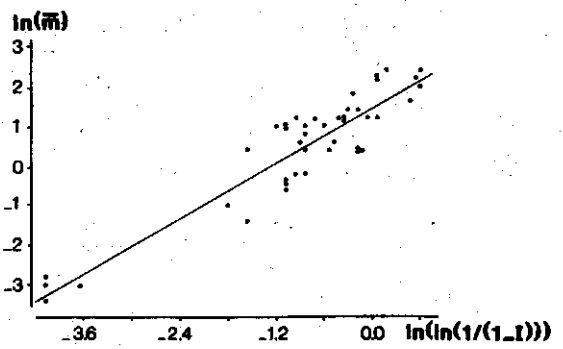

Fig. 2. Relation between transformed incidence (ln(ln[1/(1 − I)]) and log mean pustule number (ln($\overline{m}$)); data from commercial fields, top three leaves together. Each point represents a sample of 40 tillers.
Regression: ln ($\overline{m}$) = 1.48 + 1.14 ln(ln[1/(1 − I)]); R^2 = 0.86.

Comparison of the three top leaves together with the single leaf positions (experimental plots) shows that the R^2 values for the single leaf positions are slightly higher (Table 4). Both parameters, a and b, are larger for the three top leaves together than for the single leaf positions, resulting in a larger pustule number at equal incidences. This is a common result when averages are taken from non-linear relations (here a convex relation). As disease intensities on the different leaf positions are always different, the effect is fairly strong.

The estimates of the average cluster size (1/t) are 3.6 for the commercial fields and 3.2 for the experimental plots. These estimates are slightly lower than those obtained using the binomial model (4.5 and 4 respectively), because parameter b does not equal unity.

For the samples from the commercial fields, the fitted relation between incidence (I, proportion diseased leaves, n = 120, top three leaves both sides) and pustule number $\overline{m}$, average pustule number per leaf (upper surface) is:

$$\ln(\overline{m}) = 1.48 + 1.14 \ln\left[\ln\left(\frac{1}{1 - I}\right)\right] \qquad (18)$$

The relation between incidence and mean pustule number based on the negative binomial (15) could not be made linear in k and I. The model was fitted to the data of the experimental plots by an optimization procedure using least squares. The estimates of parameter k were 0.69 for the three top leaves together and 1.9, 0.68 and 0.57 for the top, second and third leaf, respectively. The fits were not acceptable. Moreover the Nachman model gives the same relation for each leaf position, while the negative binomial does not. An optimization assuming Poisson-distributed errors was also tried, but performed no better.

Precision of predicted pustule number from incidence and sample size. Both the binomial model and the Nachman model gave a good description of the measured incidence-severity relation. As the Nachman model is more flexible than the binomial

model, the precision of the estimated pustule number from a new incidence count will be analysed using the Nachman model as calibration line. The relation between pustule number and incidence has been described with the assumption that incidence was measured without error (18). The variance of the predicted pustule number in a sample can be estimated as usual by:

$$\text{Var}\,[\ln(\overline{m})] = MS_e \cdot [1 + \frac{1}{n_c} + \frac{(x - x_c)^2}{SS_x}] \qquad (19)$$

in which MS_e is the residual variance (= 0.31); n_c is the number of observations of the estimating set (= 42); x is the new observation (= ln [ln (1/(1 − I))]) made to estimate the pustule number; x_c is the mean value of x in the estimating set (= −0.855); and SS_x is the sum of squares of the x-values (= 57.2). Thus, for a new sample an estimate of the pustule number and its variance can be given, if incidence is measured as was done for the determination of the estimating set. However, we require an estimate of the pustule number in the field and of the dependence of its variance on the incidence count, when the latter is considered a random variable.

Equation 18 describes the conditional expectation of log (pustule number), given the incidence; and (19) describes its conditional variance. If we consider the incidence I as a random variable rather than a known fixed variable, the expectation of ln(M) can be estimated using (18). The variance of this estimate is more complex but it can be decomposed into two parts: the mean conditional variance and the variance of conditional mean (Rao, 1965):

$$\text{Var}\,(\ln(\hat{M})) = E \cdot \text{Var}\,(\ln(\hat{M})|x) + \text{Var}\,(E\,(\ln(\hat{M})|x))$$

The mean conditional variance can be calculated from (19); the expectation of $(x - x_c)^2$ is equal to $(Ex - x_c)^2 + \text{Var}(x)$. The variance of the conditional mean may be estimated by a Taylor approximation with respect to x (18). The 'unconditional' variance can thus be estimated as:

$$\text{Var}[\ln(\hat{M})] \approx MS_e \cdot (1 + \frac{1}{n_c} + [\frac{(Ex - x_c)^2 + \text{Var}(x)}{SS_x}]) + 1.14^2 \cdot \text{Var}(x) \qquad (20)$$

in which Ex can be estimated as ln(ln[1/(1 − I)]). The variance of x can be approximated by a Taylor expansion with respect to I:

$$\text{Var}(x) = \frac{\text{Var}(I)}{[\ln(1 - I)]^2 \,.\, (1 - I)^2} \qquad (21)$$

The variance [Var(I)] of the new incidence count can be estimated from (1), so, from a new incidence count the pustule number in a field can be estimated by (18) and its variance estimated using equations 20, 21 and 1. If the incidence in a field is known accurately, (20) equals (19), so the variance of the estimated pustule number cannot be smaller than the conditional variance, under the assumption that incidence is not a random variable.

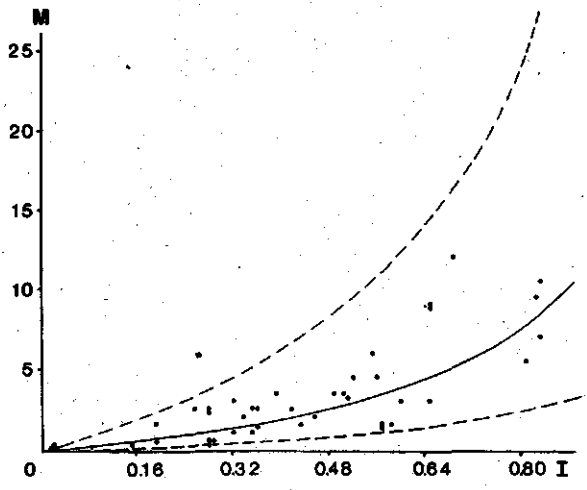

Fig. 3. Relation between incidence (I) and the mean (M) (solid line) and 95% confidence limits (broken lines) of the number of powdery mildew pustules; calculated using the Nachman model. Data from commercial fields.

Using the equations, 95% confidence limits were constructed and transformed to the original scales I and M. In Fig. 3 the relation between incidence and pustule number is plotted, with the data from the estimating set. Table 5 shows the confidence limits for the estimates of pustule number in samples and in fields, for different sample sizes. Clearly, the confidence limits are determined mainly by the conditional variance (19).

Table 5. Mean pustule number on upper surface of a leaf (M) estimated from the incidence (I), and estimated 95% lower (LL) and upper (UL) confidence limits for samples and three different sample sizes (n) for fields.

I	M	Samples (n = 120)		Fields (n = 60)		Fields (n = 120)		Fields (n = 240)	
		LL	UL	LL	UL	LL	UL	LL	UL
0.05	0.15	0.05	0.47	0.03	0.86	0.03	0.65	0.04	0.56
0.15	0.55	0.18	1.7	0.14	2.2	0.16	1.9	0.17	1.8
0.25	1.06	0.35	3.2	0.30	3.8	0.32	3.5	0.34	3.4
0.35	1.68	0.55	5.1	0.49	5.8	0.52	5.4	0.54	5.3
0.45	2.45	0.80	7.5	0.73	8.2	0.77	7.8	0.79	7.6
0.55	3.40	1.1	10	1.0	11	1.0	11	1.1	11
0.65	4.65	1.5	14	1.4	15	1.5	15	1.5	15
0.75	6.39	2.1	20	1.9	21	2.0	20	2.0	20
0.85	9.14	2.9	28	2.7	30	2.9	29	2.9	29
0.95	15.39	4.9	48	4.8	53	4.7	50	4.8	49

Efficiency of direct pustule counts and incidence counts as methods to estimate pustule number. To evaluate the efficiencies of direct pustule counts and incidence counts to predict the pustule number in a field, it is assumed that the duration to select a leaf from a field and the duration to count a pustule or to score a diseased leaf are equal and require t time units. Both methods are assumed to be equal in precision, if the 95% lower and upper confidence limits of the estimated pustule number are equal. The ratio of time units, both methods require at equal precision is a measure of efficiency of one method as compared to the other. It is not possible to calculate these ratios by means of a simple equation. The number of time units (t_i) needed to assess mildew incidence in samples of n_i (60, 120 and 240) leaves, at different mildew incidences (I) was calculated by:

$$t_i = n_i + n_i * I \tag{22}$$

The upper (UL) and lower (LL) 95% confidence limits of Table 5 were used to calculate the mean pustule number ($\overline{m}$) and the number of leaves (n_d), at which the direct pustule counts would give the same confidence limits. The 95% confidence limits for the direct pustule counts can be estimated by (Daamen, 1986):

$$UL = \overline{m} + 1.96\sqrt{\frac{2.2\,\overline{m}^{1.55}}{n_d}}, \text{ and } LL = \overline{m} - 1.96\sqrt{\frac{2.2\,\overline{m}^{1.55}}{n_d}} \text{ and} \tag{23}$$

$$\overline{m} = (UL + LL)/2, \text{ so} \tag{24}$$

$$n_d = \frac{2.2[(UL + LL)/2]^{1.55}}{[(UL - LL)/(3.92)]^2} \tag{25}$$

and the number of time units (t_d) needed for the direct pustule counts was computed by:

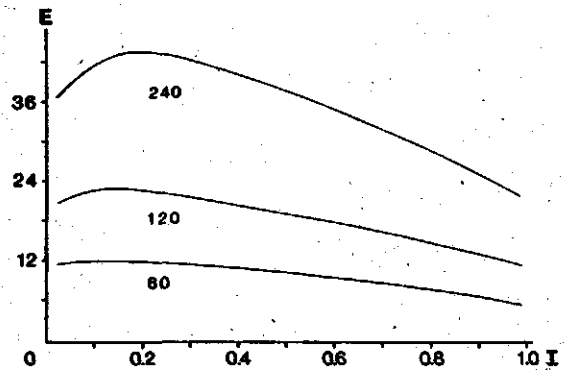

Fig. 4. Efficiency (E) of direct pustule counts compared to incidence counts, in relation to mildew incidence (I). The data in the figure represent the sample size (n = number of leaves) of the incidence counts.

$$t_d = n_d + n_d \cdot \bar{m} \quad (26)$$

The efficiency (E) of the direct pustule counts as compared to the incidence counts was computed by:

$$E = t_i / t_d \quad (27)$$

The estimated efficiencies, assuming that equal time units are required to sample a leaf or to score a diseased leaf or to score a pustule, are shown in Fig. 4. The sample size of the direct pustule counts is not fixed and can be computed from (25). It is clear that direct pustule counts are 10 to 20 times more efficient than incidence counts, when the sample size of the latter are 60 or 120 leaves.

Discussion

In this paper an incidence-severity relation was derived, assuming: constant leaf, pustule and cluster size; and a binomial distribution of the number of clusters on leaves. Although it is a crude simplification of the problem, it gives an explanation of effects of leaf, pustule and cluster size on incidence-severity relations. The model of James and Shih (1973) is a special case of the binomial model presented here. The underlying assumptions of the model of James and Shih have now been made explicit, and an equation has been given to calculate the parameter r of their model from the maximal number of clusters that a leaf can carry. The present model was developed for powdery mildew on fully grown winter wheat, where pustule and leaf size are fairly constant and cluster formation takes place by new infections. The model might also be applicable to analyse incidence-severity relations for diseases which exhibit a strong systemic growth in the leaf, when a minimum lesion size is defined. The cluster size then represents the average lesion size relative to the minimum lesion size.

James and Shih (1973) estimated the incidence-severity relation in Canada for mildew in commercial winter wheat fields, after the second node stage. The relation estimated in the Netherlands agrees with theirs; the distributions of mildew in the crops are roughly similar in the two continents. James and Shih (1973) concluded that the relation varied between years and depended on leaf position. The effects of these factors were not significant in the present study. For mildew in an experimental wheat field in the USA, before the second node stage, Rouse et al. (1981) found a different relation. They concluded that the early onset of the epidemic had caused this difference. In the context of the model developed in this paper, it is more plausible to explain the difference by the smaller leaf size of the young plants they studied as compared to the fully grown plants in this study. The effect of leaf size (as the maximal number of pustules a leaf can carry) on the relation is described by the model developed in this paper. Incidence-severity relations can be improved by taking leaf size into account. A problem to be solved is the relation between cluster size (or lesion size) and the state of the epidemic. In the present study, no attempt was made to expand the model to incorporate the age distribution of clusters or lesions.

The model of Nachman (1981) was used as a description of the relation between incidence and pustule number, and as a calibration line. No significant effects were found for different years, varieties, growth stages or leaf position. Since incidence and

pustule number were not measured independently, Mandel's (1984) sophisticated method to remove bias and to estimate the error structure of the relation could not be used. Instead, conditional statistics were used to approximate the error in the pustule number in a field predicted from an incidence count of a sample. The precision with which the incidence is determined has little effect on the error of the estimated pustule number, which is relatively large and depends mainly on the conditional variance (19). The conditional variance depends mainly on the residual error of the fitted regression line. Although the residual error was relatively small (high R^2 values), it becomes relatively large after retransformation to the original scales, the error stands in the exponent. If it takes as long to count one diseased leaf (incidence) as to count one pustule, than the direct pustule count is far more efficient. Ward et al. (1985a, b) concluded that both methods, incidence and direct counting, were equal in precision over a range of aphid intensities, but they considered only the variance of the conditional mean. In the present study with powdery mildew, the mean conditional variance could not be neglected, since it was the main determinant of the error in the predicted pustule number. For powdery mildew in winter wheat, incidence counts have advantages above pustule counts for application in supervised control systems, but the disadvantage is that incidence counts are less efficient than direct pustule counts. From a research point of view, pustule counting is prefereable above incidence counting. From a farmers point of view, incidence counting is preferable to pustule counting or severity assessment as the method is applicable to all the diseases and pests he has to monitor in his wheat crop. Moreover, not only is the application easy, but also the traning of farmers. For these reasons in the Netherlands equation 17 plus one standard error is used in the EPIPRE system to transform farmers' incidence counts to pustule numbers since 1985.

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Samenvatting

Meting van meeldauwaantastingen (Erysiphe graminis) in wintertarwe. 2. Samenhang en schattingsfouten van het aantal puistjes, de fractie zieke bladeren en de fractie ziek bladoppervlak

Het aantal puistjes meeldauw per blad en de fractie zieke bladeren werden bepaald in praktijkpercelen en veldproeven met wintertarwe. De fractie ziek bladoppervlak kon worden afgeleid uit het aantal puistjes. Het verband tussen de fractie zieke bladeren en de fractie ziek bladoppervlak werd bestudeerd. Bij aanname van een constante blad- en puistjesgrootte kan een blad maximaal M_m puistjes herbergen. Als het aantal meeldauwkolonies (elk bestaande uit n puistjes) een binomiale kansverdeling volgt,

dan kan het verband tussen de fractie zieke bladeren (I) en de fractie ziek bladoppervlak (S) worden beschreven door: $(1 - S) = (1 - I)^{n/M_m}$. De overeenkomsten van dit model met andere modellen wordt besproken en schattingen van parameters worden vergeleken. Het model verklaart hoe blad-, puistjes- en koloniegrootte het verband tussen de fractie zieke bladeren en de fractie ziek bladoppervlak beïnvloedt. Het model beschreef het verband tussen de metingen goed maar niet volledig.

De negatieve binomiaal beschreef het verband tussen de metingen slecht. Het model van Nachman gaf een goede beschrijving van het verband tussen het gemiddeld aantal puistjes kper blad ($\overline{m}$) en de fractie zieke bladeren (I). Voor de praktijkpercelen werd dit verband beschreven door: $\ln(\overline{m}) = 1{,}48 + 1{,}14 \ln(\ln[1/(1 - I)])$. De invloed van jaren, rassen, gewasstadia en bladpositie op het verband was niet significant.

De fractie zieke bladeren kan gebruikt worden om het gemiddeld aantal puistjes per blad te voorspellen, maar deze methode is minder efficiënt dan een directe telling van het aantal puistjes. Schattingsfouten van het voorspelde aantal puistjes per blad worden gegeven.

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Part III
RESULTS

Damage assessment

Neth. J. Pl. Path. 94 (1988) 69-80

Chapter 5

Effects of nitrogen fertilization and cultivar on the damage relation of powdery mildew (Erysiphe graminis) in winter wheat

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Abstract

The damage relation of powdery mildew in winter wheat was studied in field experiments in 1981 and 1983, in the Netherlands. No firm conclusion was obtained on the effect of nitrogen supply (175 and 235 kg ha^{-1} N totally) on the damage relation. The relation was not affected by cultivars (four) and did not differ significantly between both years. The measured relation averaged 0.0125 kg are^{-1} damage per pustule-day mildew per leaf, at yields of 70-90 kg are^{-1}. The effect of the fungicide triadimefon on yield could be ascribed to its effect on diseases. The vertical distribution of mildew in the crop was described.

Additional keywords: Triticum aestivum, epidemiology, vertical distribution, AUDPC-value, tolerance, integrated control, *Mycosphaerella graminicola*, triadimefon.

Introduction

Powdery mildew epidemics in winter wheat reduce yield. The three yield components, tiller number, grain number and kernel weight may be affected by mildew (Royse et al., 1980). The damage (kg or percentage yield reduction) depends on the intensity of the epidemic (Large and Doling, 1963) and on the location of mildew in the crop (Rabbinge et al., 1985). Cultural practices such as the use of resistant cultivars (Wolfe, 1983), application of fungicides, split or lowered nitrogen applications (Last, 1954 and 1962) reduce the intensity of mildew epidemics and thus reduce damage caused by mildew. The question arose whether this reduction of damage can be ascribed totally to the reduction in intensity of the epidemic or whether the damage relation depends also directly on nitrogen application or cultivar. If the damage relation of powdery mildew is independent of these cultural practices, models which describe their effects on yield and on intensity of mildew may be easily connected by means of a simple damage function, for application in supervised or integrated control systems.

Two field experiments were done to determine whether the damage relation of powdery mildew in winter wheat depends on nitrogen supply or cultivar. Attention is given to the location of mildew in the crop.

Materials and methods

The experiments were carried out at the farm 'De Bouwing' near Wageningen, situated

Table 1. Experimental conditions of the nitrogen and cultivar field experiment with winter wheat.

	Nitrogen experiment	Cultivar experiment
Cultivar	Okapi	four different
Sowing date	16 Oct. 1980	25 Oct. 1982
Seed rate	300 grains m^{-2}	330 grains m^{-2}
Row distance	12.5 cm	15 cm
Nitrogen in January (0-1 m)	80 kg ha^{-1} N	45 kg ha^{-1} N
Nitrogen applications	24 Febr. 65 kg ha^{-1} N 21 May, two levels	17 Febr. 60 kg ha^{-1} N 6 May, 60 kg ha^{-1} N
Chlormequat	23 April and 8 May, CCC 1 l ha^{-1}	6 May, CCC 1 l ha^{-1}
Control of:		
mildew	two levels	two levels
foliar diseases	23 June, 3 kg ha^{-1} maneb and carbendazim (Bavistin-M 72)	10 June, 2 kg ha^{-1} captafol (Ortho-Difolatan-80) 23 June, 2 kg and 1 l ha^{-1} captafol and fenpropimorph (Corbel)
aphids	23 June, 0.25 kg ha^{-1} pirimicarb (Pirimor)	24 June, 0.25 kg ha^{-1} pirimicarb (Pirimor)
Design	completely randomized block	split-plot; cultivars on main plots and mildew treatment on plots
Replications	6	6
Plot size	7.5 × 6.2 m^2	8 × 7 m^2
Net plot size	5.7 × 4.2 m^2	6 × 4.5 m^2
Harvest date	12 August 1981	4 and 8 August 1983

on heavy clay soil (50-60% clay). Usually, moderate mildew epidemics occur on this farm. The experimental conditions of the nitrogen experiment in 1981 and the cultivar experiment in 1983 are given in Table 1. Nitrogen was applied as calcium ammonium nitrate. In both experiments, fungicides were sprayed in June (Table 1) to control foliar diseases and to preserve the effects of the mildew treatments before that moment.

Nitrogen experiment, 1981. In the nitrogen experiment the spontaneous infection of mildew was supplemented by planting three mildew infected plants per plot on 3 April, 1981. To create different mildew epidemics, treatments with the fungicide triadimefon (0.5 kg ha^{-1} Bayleton) and application of nitrogen in plots with a full (F) and without (O) mildew control and with a high (H) and low (L) nitrogen top-dressing were:

	13 May	21 May	3 June
F + L	triadimefon	30 kg ha^{-1} N	triadimefon
O + L	—	30 kg ha^{-1} N	—
F + H	triadimefon	90 kg ha^{-1} N	triadimefon
O + H	—	90 kg ha^{-1} N	—

Cultivar experiment, 1983. On 15 April 1983 eight mildew infected plants per subplot of cultivar Okapi were planted to supplement the spontaneous mildew infection. Three commercial cultivars were used of different origin and susceptibilities to mildew and one line (SVP7348/5/4) was used because of its low susceptibility. The latter was kindly provided by the Foundation for Agricultural Plant Breeding (SVP). The origin of the cultivars and the line is:

Nautica	Mildress × Manella, crossed in 1962
Arminda	Carsten 854 × Ibis, crossed in 1963
SVP7348/5/4	(Record × Vogel 219) × (Record × Vogel 219), crossed in 1973
Durin	<(Vilmorin 29 × Vg 8058) × Cappelle Desprez> × [<(Cl 12633 × Cappelle Desprez[4]) × (Heine 110 × Cappelle Desprez)> × Nord Desprez], crossed in 1966

To create different mildew epidemics, treatments with triadimefon in plots with (F) and without (O) mildew control were carried out on 6 May and 10 June.

Disease assessment. Diseases were assessed on samples of 15 tillers per plot, taken from the area to be combine-harvested. The nitrogen experiment was sampled on 13 May (DC 32, Zadoks et al., 1974), 2 June (DC 47), 23 June (DC 69) and 7 July (DC 75). During the last three dates, 10 tillers per plot were sampled. On 23 July (DC 83) lodging was assessed. The cultivar experiment was sampled on 10 May (DC 31), 26 May (DC 33), 8 June (DC 47), 22 June (DC 69) and 4 July (DC 73).

For each green and fully unfolded leaf, the number of mildew pustules at the upper surface was counted and averaged for eacht leaf position (Daamen, 1986). The percentage leaf area, without clorosis, with speckled leaf blotch (*Mycosphaerella graminicola* (*Septoria tritici*)) was estimated. These were main diseases in both experiments. Mildew intensity on ears was below one pustule per ear and other ear diseases were virtually absent in both experiments. In the nitrogen experiment, leaf rust (*Puccinia recondita*) severity was low, 0.02% on the second leaf on 4 July. This late attack did not show any significant effect of the triadimefon applications. In the cultivar experiment, 8% of the tillers were infected by eyespot (*Pseudocercosporella herpotrichoides*) and 3% by *Fusarium* brown footrot on 1 May. The damage caused by these diseases is neglected as disease intensities were low. Moreover, these foot diseases are insensitive to triadimefon thus their intensities will be independent of the treatments against mildew. Thus it is assumed that yield reduction in the experiments is only due to mildew and speckled leaf blotch; the damage caused by other diseases is neglected.

Computations. To summarize the disease intensities during the growth period, disease intensity was averaged over leaf positions and the area under the disease progress curve (AUDPC-value in disease-days per leaf) was calculated. In the nitrogen experiment, there was hardly any mildew in the plots on 5 May. That date was taken as starting date of the epidemic. It is assumed that kernel filling stops and all leaves are dead at early dough (DC 83), so 21 July was taken as the end of the epidemic. In the cultivar experiment, 1 May was taken as the start and 26 July as the end of the epidemic, for the same reasons. Yields were standardized at 16% moisture content.

Analyses of variance and covariance designs were done with Genstat V. Analysis of variances were followed by a least significant range test; Tukey's w-procedure. Residuals

were inspected on heteroscedasticity and when necessary, variables were transformed to stabilize variances. One plot of the nitrogen experiment (F +H) was ecluded from the analyses. The yield of this plot (65.6 kg are^{-1}) was very low because of heavy lodging (60%).

Results

Mildew epidemics. The course of the mildew epidemics in the nitrogen experiment is shown in Fig. 1. An effect of the nitrogen treatment was absent ($p > 0.05$). Mildew control by triadimefon on 13 May and 3 June was nearly complete. Mildew intensity in the untreated plots was rather stable from 13 may to 23 June. The decrease in intensity at the end of the season is mainly caused by the dying of the diseased third leaf.

The course of the epidemics in the cultivar experiment is shown in Fig. 2. The mildew intensities in cv. Arminda and the SVP-line did not differ ($p > 0.05$). Mildew control by triadimefon on 6 May and 10 June, though not complete, decreased mildew intensities during the whole period ($p < 0.01$), except at the first observation on 10 May. Average disease intensity increased in May and was rather stable from 26 May to 8 June, when the healthy flag leaf appeared and the diseased fifth leaf died. Mid June, mildew intensity increased despite the dying of the diseased fourth leaf. From 23 June to 4 July, mildew intensity decreased due to the partial dying of the diseased third leaf and the overall treatment with fenpropimorph on 23 June.

Differences in partial resistance to the local mildew population caused three different types of mildew epidemics. Nautica had less mildew on 10 May than the other cultivars

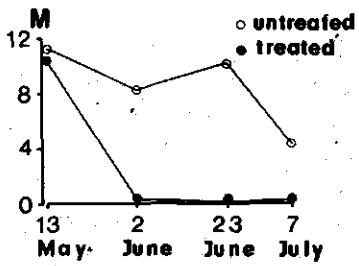


Fig. 1. Course of mildew epidemics (M is average pustule number per leaf) in treated and untreated plots of winter wheat cultivar Okapi in 1981.

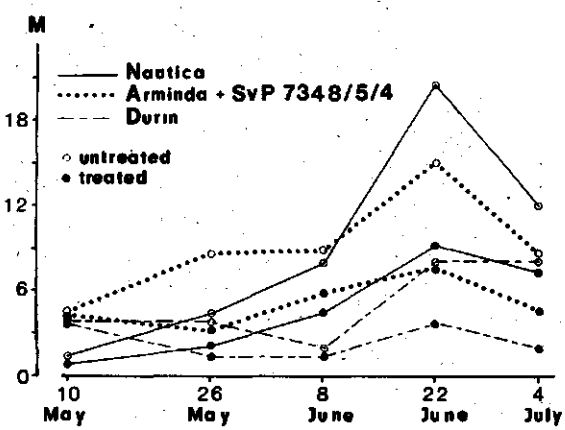


Fig. 2. Course of mildew epidemics (M is average pustule number per leaf) in treated and untreated plots of different winter wheat cultivars in 1983.

($p < 0.01$), but intensity grew steadily and in July Nautica had the highest disease intensity. Durin showed the opposite trend, starting with a mildew intensity comparable to those in Arminda and the SVP-line, but decreasing in May. On 8 June, Durin had less mildew than the other cultivars ($p < 0.01$). The course of the epidemics in Arminda and the SVP-line was intermediate.

Location of mildew in the crop. Fig. 3 and 4 show the cumulative vertical distribu-

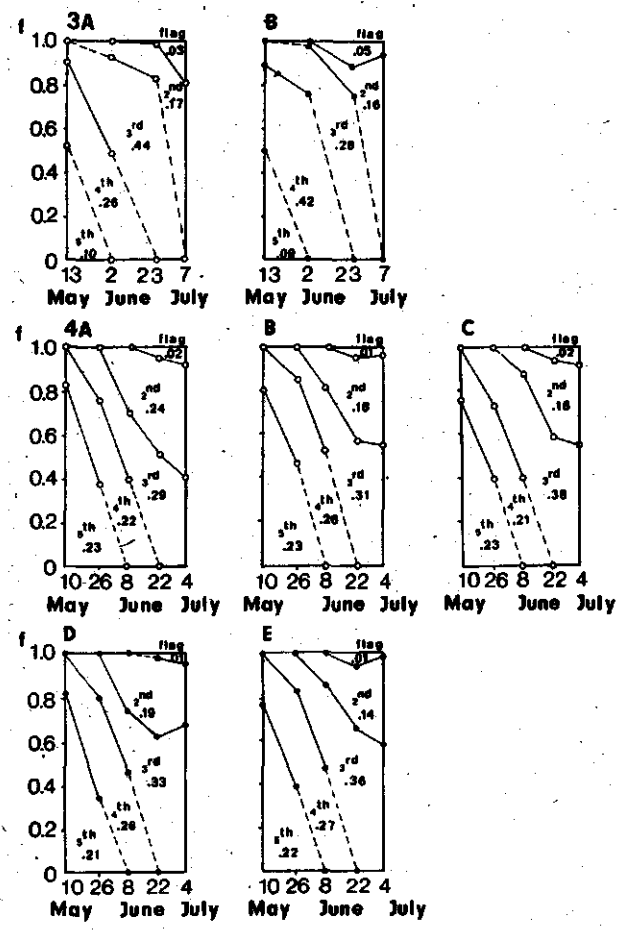


Fig. 3. Cumulative vertical distribution of mildew in an experiment with winter wheat cv. Okapi, 1981. Pustule number per leaf, expressed as a fraction (f) of the total pustule number per tiller in untreated (3a) and treated (3b) plots. Broken lines are extrapolations. The entries represent the average fraction per leaf during the observation period.

Fig. 4. Cumulative vertical distribution of mildew in an experiment with different winter wheat cultivars, 1983. Pustule number per leaf as a fraction (f) of the total pustule number per tiller in untreated plots of cv. Nautica (4a), Arminda and SVP7348/5/4 (4b) and Durin (4c) and in treated plots of cultivars Nautica and Arminda (4d) and Durin and SVP7348/5/4 (4e). Broken lines are extrapolations. The entries represent the average fraction per leaf during the observation period.

tion of mildew in the nitrogen and cultivar experiment, respectively. Only treatments with significant effects ($p < 0.05$) are shown. As example to read the figures, see Fig. 3a; on 2 June the fifth leaf is dead, the 4th leaf carried 0.50 proportion of the mildew of the whole culm, the 3rd carried 0.43 proportion, the 2nd 0.07 and the flag leaf was clean. In the untreated plots, both in 1981 and 1983, mildew on the third leaf was predominant within the period of measurements. In 1981 this domination was more pronounced as it was in 1983, presumably because the epidemic in 1981 was early and heavy relative to 1983. In both years, mildew treatments changed the location of mildew in the crop slightly ($p < 0.05$). In treated plots, mildew on upper leaves, the third in 1981 and flag and second in 1983, was relatively less and on the fourth leaf more than in the untreated plots. This effect was more pronounced in 1981 than in 1983. The cultivars showed small differences in the location of mildew in the crop ($p < 0.05$), corresponding with differences in growth curves of their epidemics. An early attack will infest lower leaves, a late attack upper leaves. The second leaf of Nautica and the third leaf of Durin had relatively more mildew than on Arminda and the SVP-line (Fig. 4a, b, c). The mildew epidemic in Nautica was relatively late and in Durin relatively early (Fig. 2). This effect is strengthened by differences in growth rhythm of the cultivars; Nautica is early heading and Durin late, Arminda intermediate. Accordingly the mildew epidemic in Nautica was late compared to that in Durin. In the treated plots, the same trend was observed.

Yield and disease intensities. Yields, summarised disease intensities (mean number of disease-days per leaf) and lodging are given in Tables 2 and 3 for the nitrogen and cultivar experiment, respectively. In the experiment of 1981, late nitrogen application (30 or 90 kg ha^{-1} N on 21 May) had no significant effect on yield, mildew or speckled leaf blotch intensities, while fungicide treatment was significant and prevented a damage of 6.1 kg are^{-1} (SE = 0.8). Mildew and speckled leaf blotch intensities were positively correlated ($r = 0.73$) and showed negative correlations with yield; $r = -0.83$ and $r = -0.65$ for mildew and speckled leaf blotch respectively. Nitrogen reduced the yield component kernel weight in untreated plots, presumably by an increase of the number of grains per unit area.

Table 2. Yield, grain weight, disease intensities of powdery mildew and speckled leaf blotch and percentage lodging of sprayed (F) and unsprayed (O) winter wheat at two different nitrogen levels (H = high, L = low). Entries are mean values of six replications.

Treatment	Yield[1]	Grain weight[2]	Mildew[3]	Leaf blotch[4]	Lodging
F + L	76.7a[5]	49.7a	151a	46a	23a
O + L	71.0b	48.7a	580b	117b	14a
F + H	78.8a	49.5a	156a	41a	30a
O + H	71.9b	46.3b	600b	122b	26a

[1] In kg are^{-1}.
[2] In mg.
[3] In pustule-days per leaf.
[4] In percent-days per leaf.
[5] Different letters indicate significant ($p < 0.05$) differences between treatments within colums.

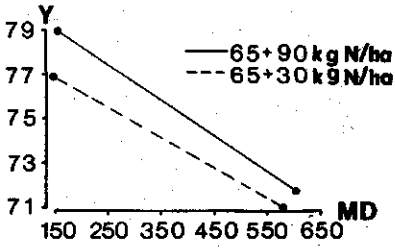
Table 3. Yield, grain weight, disease intensity of powdery mildew and of speckled leaf blotch in sprayed (F) and unsprayed (O) plots of four different cultivars of winter wheat. Entries are mean values of six replications, split plot design.

Cultivar	Treatment	Yield[1]	Grain weight[2]	Mildew[3]		Leaf blotch[4]	
Nautica	O	80.2	40.6	649	(6.5)[6]	142	(5.0)[6]
Nautica	F	86.4	40.9	332	(5.8)	108	(4.6)
Arminda	O	79.0	33.0	588	(6.4)	131	(4.9)
Arminda	F	84.7	33.9	378	(5.9)	85	(4.4)
SVP7348/5/4	O	76.6	34.8	711	(6.6)	176	(5.2)
SVP7348/5/4	F	82.4	36.5	358	(5.8)	138	(4.9)
Durin	O	85.8	36.5	395	(6.0)	159	(5.0)
Durin	F	89.1	37.4	180	(5.1)	123	(4.8)
SED between varieties		1.3	1.9		(0.14)		(0.10)
SED within a variety		1.0	1.5		(0.14)		(0.09)

Notes [1] to [4], see Table 2.
[6] log transformed disease intensities.

In the cultivar experiment of 1983, fungicide and cultivar treatment was significant, their interaction was not significant ($p > 0.05$). Durin yielded more than the other cultivars, whereas the SVP-line yielded less. Two times control of mildew prevented a damage of 5.2 kg are^{-1} (SE = 0.5). Arminda had relatively less speckled leaf blotch. Mildew and speckled leaf blotch were positively correlated ($r = 0.48$), mildew was negatively correlated with yield ($r = -0.68$), the correlation between yield and speckled leaf blotch was not significant ($p > 0.05$). Kernel weight of Nautica was higher than of the other cultivars. Fungicide treatment did not significantly affect kernel weight, indicating that damage by diseases occurred before kernel filling.

Different damage relations. In Fig. 5 and 6, mean yield per treatment is plotted against mildew intensity in pustule-days. Analysis of covariance were applied to study these relations, taking into account the correlation between mildew and speckled leaf blotch. Tests were performed on the significance of average relations between yield and the diseases and on the significance of different damage relations, due to nitrogen (Table 4, 1981) and cultivar (Table 5, 1983). Corrections were applied for the experimental design. In these analyses, damage relations were assumed to be linear. No tendency of heteroscedasticity or curvilinearity was detected in residuals. The combined effect of diseases on yield was significant (row 4, Tables 4 and 5) in both experiments. The partial effect of mildew on yield was significant, in both years ($p < 0.001$, row 4b). Speckled leaf blotch showed only a significant partial effect in 1983 ($p < 0.05$, Table 5, row 4a). Damage caused by the diseases is mainly attributed to mildew and not to speckled leaf blotch. Interactions of the damage relations with nitrogen or cultivar were not significant ($p > 0.05$, Table 4 and 5 row 6). In other words, the damage relations of mildew and speckled leaf blotch were independent of the nitrogen and cultivar treatment. Tests on interactions between mildew and speckled leaf blotch were not significant (row 8), so at these disease intensities the damage relations of the two diseases



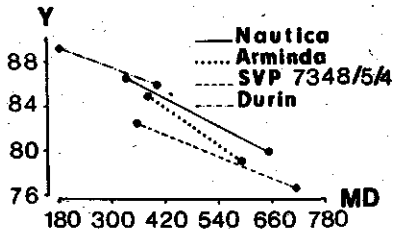


Fig. 5. (Left) Yield (Y, in kg are^{-1}) in relation to mildew intensity (MD, in pustule-days per leaf), assuming linearity, at two different nitrogen levels, 1981. Dots represent mean yields, not corrected for covariates.

Fig. 6. (Right) Yield (Y, in kg are^{-1}) in relation to mildew intensity (MD, in pustule-days per leaf) assuming linearity, for four different winter wheat cultivars, 1983. Dots represent mean yields, not corrected for covariates.

Table 4. Analysis of variance on yield to test for interactions of damage by diseases and nitrogen.

Source of variation[1]	df		SS	MS	F[2]
1. H	5		23.1	4.6	
2. N	1		6.2	6.2	
3. L	1		25.9	25.9	
4. MD + ST	2		217.0	108.7	43.3 ***
4a. ST adj. MD		1	0.1	0.1	0.0 ns
4b. MD adj. ST		1	67.9	67.9	27.0 ***
5. Residual	13		32.6	2.5	
6. Interactions ST.N + MD.N	2		11.6	5.8	3.0 ns
6a. ST.N adj. MD.N		1	3.3	3.3	1.7 ns
6b. MD.N adj. ST.N		1	0.5	0.5	0.3 ns
7. Residual	11		21.0	1.9	
8. Interaction MD.ST	1		0.1	0.1	0.0 ns
9. Residual	10		20.9	2.1	
10. Total	22		305.1	13.9	

[1] H = replications; N = nitrogen treatments; L = % lodging; MD = pustule-days mildew; ST = %-days leaf blotch; R = cultivars.
[2] ns = not significant ($p > 0.05$); * = $p < 0.05$; *** = $p < 0.001$.

may be considered independent.

Yield estimates of the three commercial cultivars, without disease were nearly equal (ca. 94 kg are^{-1}). The high yield of Durin, compared to Nautica and Arminda, can be ascribed to its relatively high mildew resistance. Durin without protection had nearly the same mildew intensity and yield as Nautica and Arminda with two fungicide applications (Fig. 6).

Slope of the damage relations. The estimates of the partial effects of mildew and speckled leaf blotch which deviated signifcantly from zero ($p < 0.05$) are given in Table

Table 5. Analysis of variance on yield to test for interactions between damage by diseases and varieties.

Source of variation[1]	df		SS	MS	F[2]
1. H	5		27	5.4	
2. R	3		402	134.1	
3. H.R	15		113	7.5	
4. ST + MD	2		307	153.5	33.4 ***
4a. ST adj. MD		1	20	19.8	4.3 *
4b. MD adj. ST		1	71	70.6	15.4 ***
5. Residual	22		101	4.6	
6. Interactions ST.R + MD.R	6		33	5.5	1.2 ns
6a. ST.R adj. MD.R		3	22	7.5	1.8 ns
6b. MD.R adj. ST.R		3	12	3.9	0.9 ns
7. Residual	16		68	4.2	
8. Interaction MD.ST	1		0	0.1	0.0 ns
9. Residual	15		68	4.5	
10. Total	47		950	20.2	

Notes [1] and [2], see Table 4.

Table 6. Estimated damage (kg are^{-1}) per pustule-day mildew per leaf and percent-day speckled leaf blotch per leaf, assuming linearity and their standard errors.

	Experiment 1981	Experiment 1983
All leaves:		
mildew	0.013 (0.002)	0.012 (0.003)
leaf blotch	ns	0.040 (0.019)
Top three leaves:		
mildew	0.014 (0.003)	0.013 (0.003)
leaf blotch	ns	ns

ns = not significant ($p > 0.05$).

6, for all and for the top three leaf positions. Comparison of the estimates of the damage per pustule-day mildew, assuming a linear damage relation, show that they did not differ significantly when all leaves or top three leaves were compared. The estimates of 1981 in cv. Okapi did not differ significantly from those of 1983, in cultivars Nautica, Arminda, Durin and the SVP-line.

Neglecting other effects of fungicides. The analyses above assume that fungicide treatment affect yield only by reducing intensities of mildew and speckled leaf blotch. Other diseases, phytotoxic or stimulating effects of fungicides are neglected. An impression of the validity of these assumptions can be obtained when the effect of fungicides on yield in anova design is compared with the effect of diseases on yield in analysis of covariance. If triadimefon determines yield not only through mildew and speckled leaf

Table 7. R^2 values of the yields explained by the fungicide treatments (anova) or by the main diseases (linear regression of mildew and speckled leaf blotch).

	Experiment 1981	Experiment 1983
Fungicides	0.84	0.93
Diseases	0.89	0.89

blotch, then a greather percentage of the yield variation should be explained by mildew treatment compared to disease intensity (Table 7). The R^2 values were high, ranging from 0.84 to 0.93, and did not differ much between both types of analyses. Thus it can be concluded that the diseases mildew and speckled leaf blotch and the fungicide treatment explained in statistical terms an equal part of the yield differences.

Discussion

Two applications of triadimefon gave nearly complete mildew control in 1981, despite the high mildew intensity at onset of the epidemic. In 1983 the spraying was not that successfull, even though mildew intensity was lower. This effect was also observed in commercial fields and attributed to a decrease in mildew sensitivity to triadimefon in 1983 (De Waard et al., 1986). Moreover the effectivity of triadimefon was lower due to the cold and wet weather in spring 1983 compared to 1981 (Kuck, 1987).

Darwinkel (1980) found effects of different nitrogen applications on winter wheat yield and on mildew intensity, in field experiments in the Netherlands. No significant effects of different late nitrogen applications on yield and mildew intensity were found in the experiment described above. It is likely that the fertilizer was available for the crop, as precipitation in May after the 21th was 38 mm. The nitrogen treatment showed a significant effect on grain weight, indicating that the treatment resulted in a different uptake of nitrogen by the crop. Total nitrogen supply was high, 175 kg ha^{-1} N in the low and 235 kg ha^{-1} N in the high nitrogen treatment. In commercial fields of the Netherlands, total nitrogen supply (including available nitrogen in the soil) was about 220 kg ha^{-1} N in 1983. Presumably, the effect of nitrogen on mildew is negligible at these high application levels, but no firm conclusion can be drawn from the nitrogen experiment, as nitrogen concentration and distribution in the crop were not measured.
In 1983, the cultivars sustained three different mildew epidemics, reflecting the differences in partial resistance to the local mildew population. The cultivars did not show significant different damage relations to mildew.

The question is whether this conclusion can be extrapolated to all commercial cultivars in the country, in the scope of integrated control. In literature on disease management, no connection with this question was found. In resistance breeding this question concerns the concept of tolerance and the conclusion above state that cultivars were equal in tolerance (Schafer, 1971) to mildew. Gaunt (1981) discussed the concept and questioned whether true tolerance (Schafer, 1971) in fact exists. He suggests to measure disease by several different parameters, including chlorosis, senescence, leaf area and other plant fysiological functions; the whole syndrome. I agree with Gaunt, that the term tolerance to disease (the whole syndrome) is then redundant and the term tolerance to pathogens

may be restricted to the study of the relation between the pathogen and the syndrome. As only pustule number was determined in this study, no conclusion on tolerance to mildew (interpretation Gaunt, 1981) of the tested cultivars can be made. Whichever terminology is used, the cultivars did not show significant different damage relations to powdery mildew intensity and the above raised question remains to be answered.

Kramer et al. (1980) give support for this extrapolation. One may reason that plant breeders select high yielding cultivars which are exposed to mildew. As a consequence, they select indirectly for less negative slopes of damage relations and differences between slopes are eliminated. Therefore, it seems reasonable to assume that all partially resistant commercial cultivars in the country have the same damage relation to their mildew strains. One might speculate, that completely resistant cultivars escaped such an indirect selection and might show more negative slopes of their damage relation when attacked by a new mildew strain.

The slopes of the two measured damage relations in both years, assuming linearity, did not differ significantly and averaged 0.0125 kg are^{-1} per pustule-day mildew per leaf, from first node stage onwards, at yields of 70-90 kg are^{-1} in the Netherlands. The measured damage cannot be explained by a reduction of weight per kernel, as in the cultivar experiment no significant effect of diseases on grain weight was found. Further analysis showed that the partial effects of the main diseases on yield adjusted for grain weight were significant ($p < 0.001$) in both experiments. So it is indicated that the damage by mildew and speckled leaf blotch is caused by a reduction of the grain number per unit area, in these experiments.

The vertical distribution of mildew depended on cultivar, but this was not exclusively a cultivar effect. It was indicated that the mildew location in the crop depended mainly on the moment of mildew infection in the ontogeny of the crop. In integrated control systems, the choice of resistant or partially resistant cultivars is a prerequisite. Inputs such as fertilisers and fungicides are minimized, maximizing net yield. All input factors can affect yield directly and also indirectly through effects on disease intensity. The study of the impact of all these factors and their interactions on yield in field experiments is complicated (Walters et al., 1984 and Widdowson et al., 1982). The present study indicates that the direct effects of cultivars, and presumably also of late nitrogen application at high fertilization levels, on yield do not interact with their indirect effects through powdery mildew. The fungicide triadimefon affected yield only through its effect on diseases in this study. Crop models, which describe or explain the quantitative effects of inputs on yield, and epidemiological models, which describe the effects of these cultural practives on mildew, can then be connected by a simple damage function, to optimize the integrated control system.

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Samenvatting

Effecten van stikstofbemesting en ras op de schaderelatie van meeldauw (Erysiphe graminis) in wintertarwe

In 1981 en 1983 werd in veldproeven met wintertarwe de relatie tussen schade (in kg are^{-1}) en meeldauw onderzocht. Of de relatie onafhankelijk is van de stikstofbemesting (175 en 235 kg ha^{-1} N totaal), kon niet met zekerheid worden vastgesteld. De schaderelatie werd niet significant beïnvloed door de vier getoetste rassen. In de twee jaren werd een vergelijkbare schade van gemiddeld 0.0125 kg are^{-1} per puistdag meeldauw per blad gemeten, bij een opbrengstniveau van 70-90 kg are^{-1}. Tevens werd de locatie van meeldauw in het gewas beschreven.

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Chapter 6

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 '8
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 Ult 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 m²	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-

P81	WR81	VP81	WR82	WS82	GV82
kapi	Arminda	Okapi	Arminda	Okapi	Okapi
ay 50%	loess 26%	sand	loess 28%	clay 32%	improved peat
otato	potato	potato	potato	sugar-beet	potato
3 October '80	24 November '80	17 October '80	26 October '81	23 November '81	15 October '81
5	—	45	36	30	60
0+30	110+35	125+30	100+60	104+52+39	78+39
April	4 April	7 May	13 April	28 April	28 April
2.5 l MCPA	6 l Tolkan	4 l MCPP	6 l DM-68	MCPP+MCPA	6 l Arelon
3.5 l MCPP				2 June	Combi
				MCPA	
April 2 l	5 May 1.5 l	7 April 1 l	16 April 1 l	28 April 1.5 l	none
		23 April 1 l			
June Pir.	22 June Pir.	23 June Pir.	11 June Pir.	22 July Pir.	2 July Pir.
	10 July Pir.				
	4	8	4	4	4
	8	6	6	6	6
2.5×3	8×4	8.5×6.5	7×9	7×9	7×9
0×2.5	5×3	6×4.5	4×6	4.5×7.5	6.75×4.5
August '81	15 August '81	5 August '81	12 August '82	10 August '82	11 August '82
		23 June	11 June	22 June	
		Captafol	Bayleton CF	Bayleton CF	

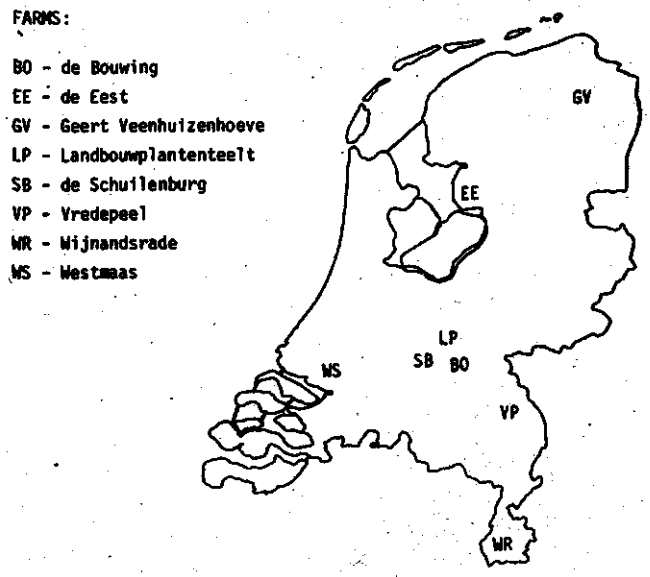


Fig. 1. Location of the experiments in the Netherlands.

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:

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

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.

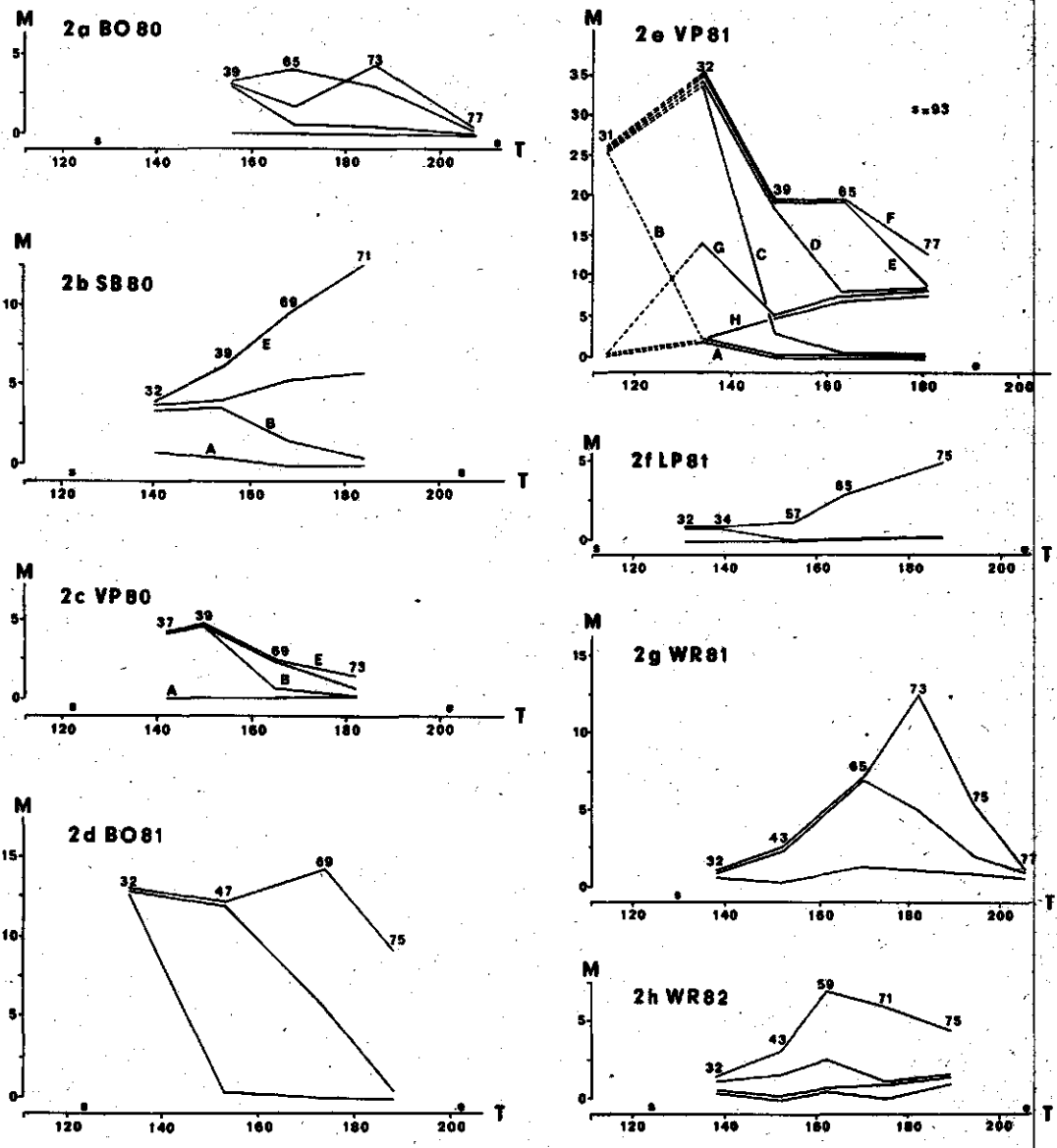


Fig. 2a, b, c, d, e, f, g, h. Mildew epidemics in the eperiments, M is pustule number per leaf and T is Julian day. Numbers in the figures represent developmental stages and capitals different treatments (Table 6). Only grouped epidemics whose patterns differed significantly from others ($P < 0.05$) 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 $Y = Y_o + b \cdot MD$; 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}	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

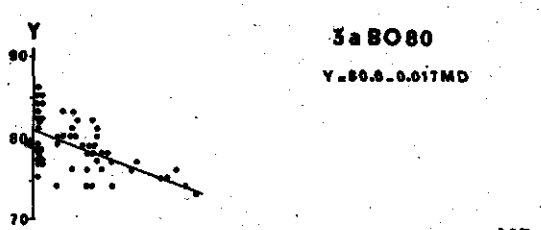
[1] ns = not significant, $p > 0.05$; * $p < 0.05$; ** $p < 0.01$.
[2] As VP81 but plots with fungicide application on 7 April eliminated.
[3] not significant, $p > 0.05$.

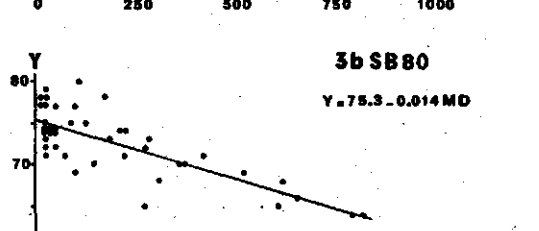
in any of the experiments, so linearity was accepted.

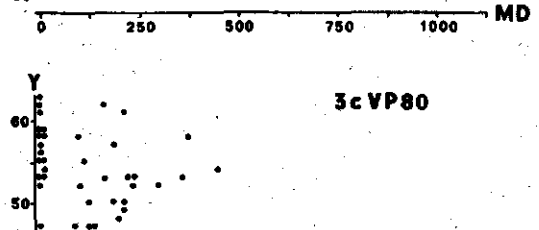
Partial effects of covariates were often non-significant, presumably due to colinearity. In BO81, speckled leaf blotch showed a significant effect on yield: −0.036 kg are^{-1} (SE = 0.012) per %-day per leaf. In WR81, glume blotch correlated significantly with yield: −1.1 kg are^{-1} (SE = 0.4) per % 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.

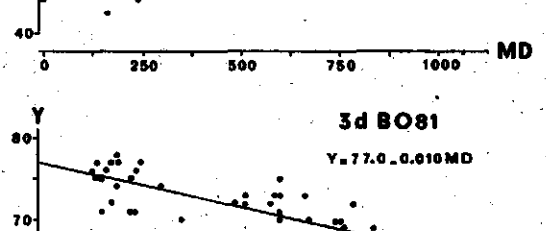
Experiment VP81 was analysed differently, as mildew intensity was assessed as percentage on the first observation and as pustule number on the following observations. This resulted in two estimates of damage, one from the very early and one from the later disease stress. The later attack showed a negative 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).

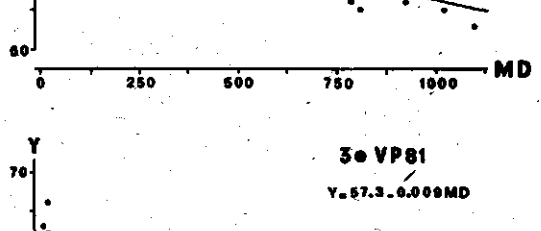
The developmental stage of the crop might affect damage. A multiple point model (Madden et al., 1981; Teng and Shane, 1983) should then give a better fit than the AUDPC approach used here. On the other hand, weather conditions during the season might affect damage. Shaw and Royle (1987) analysed damage 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

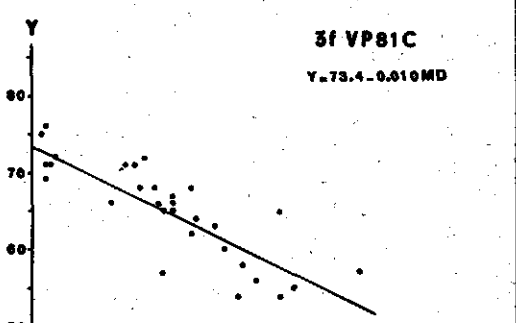


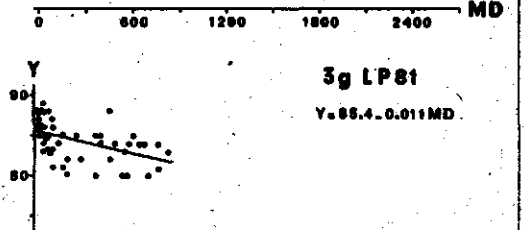


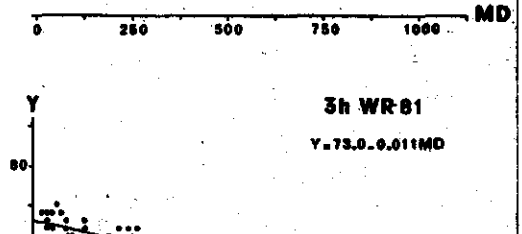












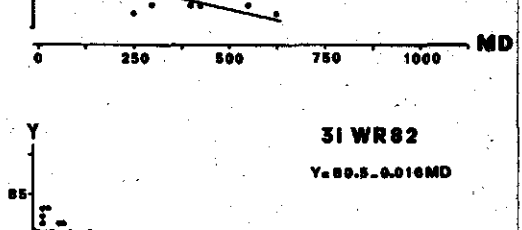


Fig. 3a, b, c, d, e, f, g, h, i. Partial residual plots plus intercept, of yield (Y in kg are^{-1}) versus mildew stress (MD in pustule-days per leaf) and estimated partial regression lines (Table 4).

Table 5. Estimates of the partial effect of mildew stress (MD, in pustule-days per leaf) on seed number: $SN = SN_0 + b \cdot MD$, and the significance of different b-values for each observation date.

Experiment	$\hat{SN}_0$[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-

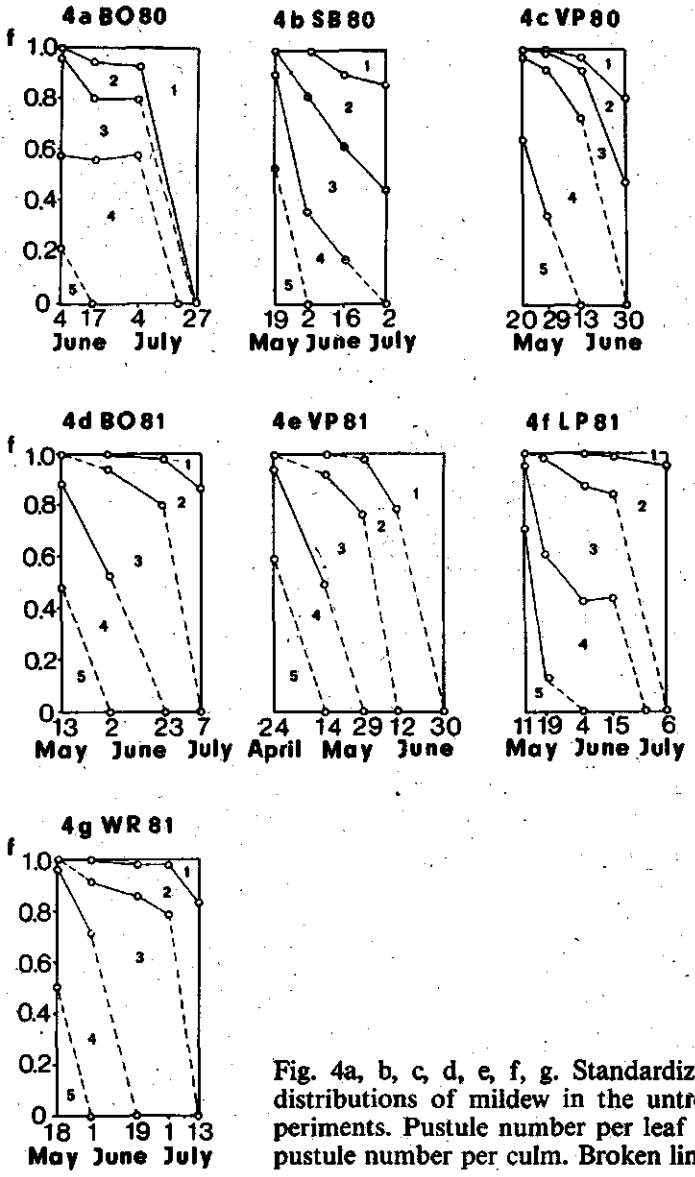


Fig. 4a, b, c, d, e, f, g. Standardized cumulative vertical distributions of mildew in the untreated plots of the experiments. Pustule number per leaf as fraction (f) of total pustule number per culm. Broken lines are extrapolations.

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.

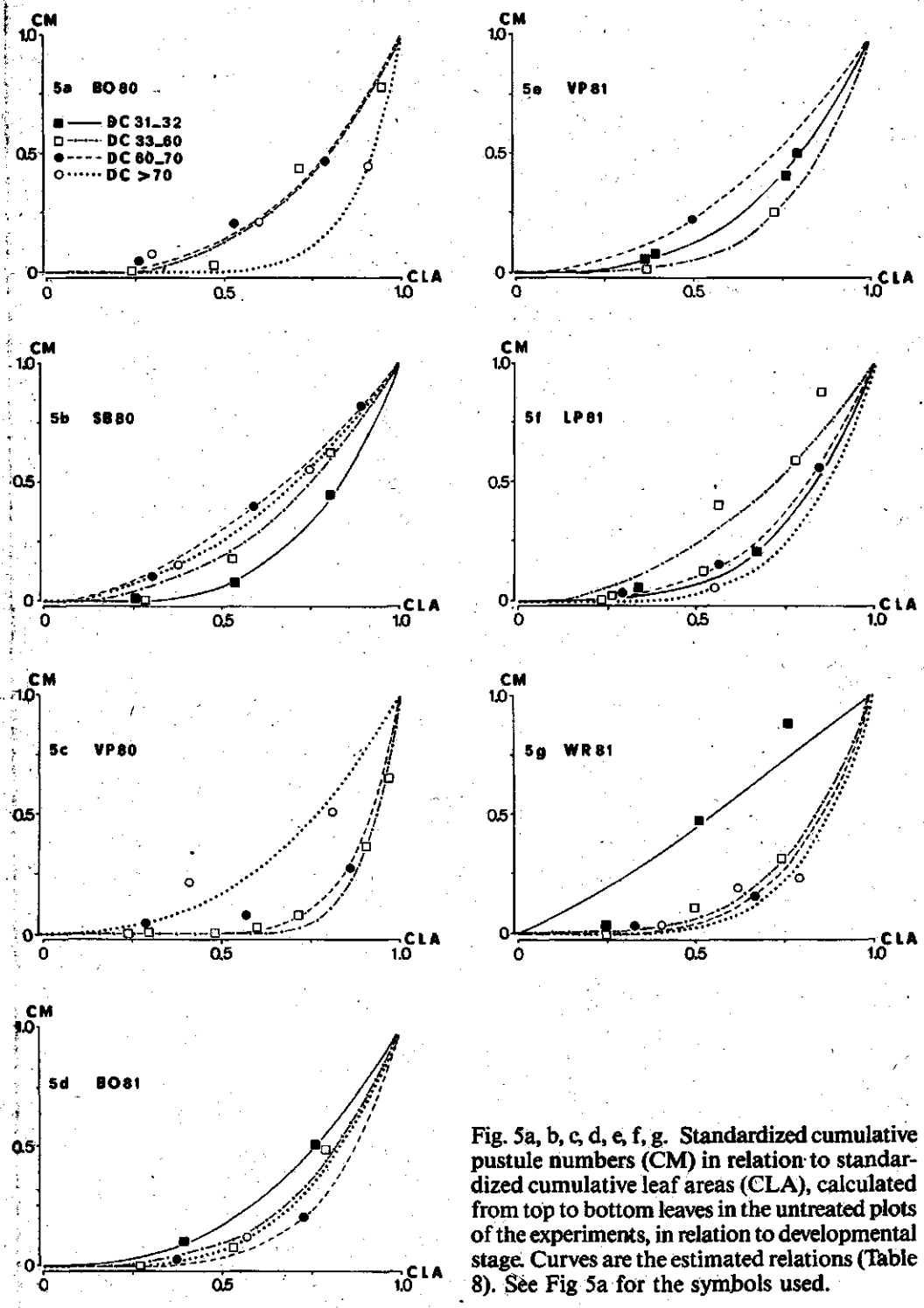


Fig. 5a, b, c, d, e, f, g. Standardized cumulative pustule numbers (CM) in relation to standardized cumulative leaf areas (CLA), calculated from top to bottom leaves in the untreated plots of the experiments, in relation to developmental stage. Curves are the estimated relations (Table 8). See Fig 5a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

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

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 $b \cdot \bar{y}$, 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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Part IV
DISCUSSION

Processes which underlay damage

Chapter 7

Effects of powdery mildew and weather on winter wheat yield. 1. Variation of weather between years

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Abstract

A vital question to upgrade disease management systems is whether damage functions, established in years with specific weather conditions, can be applied also in other years. A simulation approach was used to explore effects of weather on yield and damage. Two existing models of wheat and a third, a combination of both, were used to compute yield, first in absence of mildew. In a second paper, effects of mildew will be reported. Yields simulated for different years were not significantly correlated with yields harvested, adjusted for their increase over years, in the Netherlands. Differences in performance between the models could be attributed to the method of simulating development and growth early in the cropping season.

Additional keywords: Triticum aestivum, simulation, SUCROS87, NWHEAT.

Introduction

The averaged measured damage function of powdery mildew in winter wheat (Daamen, 1988 and 1989) was implemented in a management system to assess the profitability of fungicide application in farmers' fields in the Netherlands. Though damage was measured under conditions approaching those of farmers' fields, it is uncertain to what extent this damage function may be extrapolated to other weather conditions (Jenkyn, 1984), in different years or locations, and to crop husbandry practices other than those of the experiments. Repeated measurements in other years and locations may answer this question, but will not develop general methods to enable these extrapolations to be made. The study of physiological processes which underlay damage, provide another option to answer the question.

The physiology of the host is changed by a mildew infection. Photosynthesis at light saturion, especially, drops rapidly after inoculation. This decrease is not correlated with the chlorophyll content of infected leaves (Allen, 1942), but is correlated with a decrease in activity and amount of the ribulose biphosphate carboxylase protein and changes in the effectiveness of other enzymes (Walters and Ayres, 1984). Rabbinge et al. (1985) quantified the effect of powdery mildew on photosynthesis of wheat leaves in relation to light intensity. They found that, even at low levels of infection, the assimilation rate at light saturion was considerably reduced, while the light use efficiency and dark respiration were not significantly affected. These quantified physiological processes can provide a general method to assess the effect of weather on damage by powdery mildew.

In this paper the effect of weather on yield is studied. The performance of two crop models and a combination of both, to varying weather conditions

is compared with wheat yields harvested in the Netherlands in different years. In a following paper, the simulation of yield in relation to varying mildew intensities and weather conditions will be discussed.

Materials and methods

Crop models. Two models, with estimates of parameter values under conditions in the Netherlands, were available: NWHEAT (version date: December 1986, Groot, 1987) and SUCROS87 (version date: November 1987, Spitters et al., 1989).

NWHEAT was developed to simulate the growth and development of winter wheat from temperature and radiation after sowing date, taking into account the nitrogen and water balance in crop and soil. As this model was too complex for our purpose, J.R.R. Groot kindly adapted it so that water and nitrogen were not limiting. Nitrogen still played an important role as it governs photosynthesis and death of leaves.

SUCROS87, a summary model, was developed to simulate growth and development when water and nitrogen are not limiting and growth is governed by temperature and radiation. The model, written in the language CSMP, was translated into FORTRAN77. The SUCROS87 model for winter wheat starts on 1 January.

SUCROS87E, an extended version of SUCROS87 simulates growth from sowing date onwards. Development (D) is simulated, depending on photoperiod and temperature (Groot, 1987; Reinink et al. 1986). Before a developmental stage (D) of 0.5 and a leaf area index (L) of 1.5, the daily growth of the leaf area index (ΔL) is computed from the daily growth of the shoot weight (ΔW_{sh}) corrected for the daily loss of weight by leaf death (ΔW_{ld}), multiplied by the average specific leaf area (l_{sp}):

$$\Delta L = l_{sp}.(\Delta W_{sh} - \Delta W_{ld}); \quad D<0.5 \text{ and } L<1.5 \qquad (1)$$

Although this conforms with the approach of SUCROS (Van Keulen et al., 1982) and of NWHEAT, NWHEAT and SUCROS87 differ in many aspects (Groot, 1987; Spitters et al., 1989).

To compare the response of SUCROS87E, SUCROS87 and NWHEAT, to wheather conditions, yields were simulated with the models without disease, using actual weather from Wageningen in 1975 to 1986, and a sowing date of 20 October. National average yields (statistical yearbook), yields obtained in the intensive cropping treatment of cultivar trials on marine clay soils (RIVRO Institute), and yields on best fields on the farm 'De Bouwing' near Wageningen (pers. comm. W. de Jager) were used as estimates of harvested yield in these years. Correlations among the yields were calculated and used to compare the yields over the eleven years. As harvested yields improved gradually, they were regressed on simulated yields and year, to calculate the partial regression coefficients between harvested and computed yields.

Results

Performance of the models without disease. Average yields computed by SUCROS87 and by NWHEAT were about the same (Table 1). Those of SUCROS87E were about one ton/ha higher. The models computed equal variation in yield over the years. Yields computed by NWHEAT and SUCROS87E were significantly correlated, but they were not correlated with those of SUCROS87 (Table 2). SUCROS87 and SUCROS87E differ only in the computation

Table 1. Comparison of harvested and computed yields (kg dry weight are^{-1}) in different years, at a simulated sowing date of 20 October.

Year	Harvested yield: Country average	Harvested yield: Cultivar trials[1]	Harvested yield: De Bouwing[2]	Computed yield: SUCROS87	Computed yield: NWHEAT	Computed yield: SUCROS87E
75-76	48.	62.7	52.9	63.8	54.5	63.8
76-77	45.	59.1	60.5	89.5	72.0	84.7
77-78	57.	66.5	68.0	72.6	83.4	104.3
78-79	51.	62.3	61.3	69.2	81.4	91.7
79-80	54.	63.3	71.4	79.6	72.4	88.9
80-81	58.	71.9	73.9	67.3	76.0	87.0
81-82	64.	79.2	68.0	71.7	74.8	87.3
82-83	60.	73.6	78.1	84.6	72.4	80.4
83-84	66.	79.7	98.3	60.9	78.3	90.9
84-85	56.	62.2	74.8	82.9	80.6	92.2
85-86	68.	79.6	91.6	81.2	73.5	85.1
Average	57.	69.1	72.6	74.8	74.5	86.9
CV %[3]	12.7	11.5	18.3	12.3	10.4	11.3

[1] On marine clay soils, treatment: intensive cropping.
[2] Best yielding fields on the farm.
[3] Coefficient of variation.

Table 2. Correlation coefficients of harvested and computed yields, df=9. Correlations with yields of 'De Bouwing' were with actual sowing dates. Underlined coefficients are significant at $p<0.05$.

	1.	2.	3.	4.	5.	6.	7.
1. Year	-						
2. Country average	0.83	-					
3. Cultivar trials	0.66	0.93	-				
4. De Bouwing	0.84	0.85	0.74	-			
5. SUCROS87	0.16	-0.18	-0.28	-0.04	-		
7. NWHEAT	0.38	0.35	0.13	0.35	0.10	-	
6. SUCROS87E	0.22	0.29	0.05	0.30	0.08	0.94	-

of development and growth early in the season. NWHEAT and SUCROS87E differ in many aspects, but not in the computation of development early in the season. Therefore, the difference in performance of NWHEAT and SUCROS87 is attributed to the method of simulating early growth.

The estimates of harvested yield were significantly correlated and increased significantly over the years, due to the gradually intensifying crop husbandry (Table 1,2).

The correlation coefficients between the yields harvested and computed were not significant (Table 2). Also the partial regression coefficients between yields harvested and computed, adjusted for a constant annual increase, were not significant (Table 3). Therefore, the models do not explain the variation in harvested yield between years.

Table 3. Partial regression coefficients (kg harvested per kg computed), of three different data sets of harvested yield, adjusted for a constant annual increase, with yields computed by three different models, see text. Standard errors are given between brackets.

Harvested yields	Computed yields[1] SUCROS87	NWHEAT	SUCROS87E
Country average	-0.24 (0.13)	0.04 (0.20)	0.08 (0.15)
Cultivar trials	-0.33 (0.19)	-0.15 (0.29)	-0.08 (0.22)
'De Bouwing'	-0.24 (0.25)	0.14 (0.34)	0.20 (0.24)

[1] Simulated sowing date of 20 October or actual sowing date of 'De Bouwing'

Discussion

An indication was obtained that the effect of weather on yield was not computed accurately by the models. Computed yields varied considerably between years, due to the varying weather conditions. Also, harvested yields varied considerably and the question is whether this variation should be attributed to weather or to other yield constraints. Harvested yields were at least partly limited by these other constraints, such as diseases, pests, lodging and fertilizer shortages, as harvested yields increased over years due to the gradually intensifying crop husbandry. The question then is whether the variation in harvested yields between years, corrected for an annual trend was caused by weather or by other varying constraints.

Harvested yields, in the cultivar trials and on 'De Bouwing', were in later years often higher than computed yields, in which cases it is unlikely that other yield constraints than weather dominate. Moreover, the three data sets of harvested yields were positively correlated, also after correction for trend, which indicates that the constraints of yields were similar. This suggests that the variation in harvested yields was dominated by weather.

Computed yields depended on the method of simulating development and growth early in the cropping season. Therefore, it might be necessary to give more attention to the computation of the partitioning of assimilates between root and shoot, and of the specific leaf area, in dependence of temperature (Van Dobben, 1962).

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Samenvatting

Effecten van meeldauw en weersomstandigheden op opbrengsten van wintertarwe. 1. Verschillende weersomstandigheden per jaar

Om systemen voor geleide bestrijding van ziekten te verbeteren, werd de vraag gesteld of schaderelaties, vastgesteld in jaren met specifieke weersomstandigheden, ook gebruikt kunnen worden in andere jaren. Twee bestaande modellen van tarwe en een combinatie van beide, werden gebruikt om het effect van weer op opbrengst te berekenen, eerst bij afwezigheid van meeldauw. De berekende opbrengsten in verschillende jaren toonden geen samenhang met de behaalde opbrengsten, gecorrigeerd voor een jaarlijkse toename, in Nederland. Verschillen tussen de modellen konden worden toegeschreven aan de wijze waarop de groei en ontwikkeling van tarwe vroeg in het seizoen wordt berekend.

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Chapter 8

Effects of powdery mildew and weather on winter wheat yield. 2. Effects of mildew epidemics

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Abstract

Wheat yield losses caused by powdery mildew were computed based on effects of the disease on leaf photosynthesis. Powdery mildew was introduced in a crop model of wheat by quantification of five parameters, taking the vertical and horizontal distribution of mildew in the crop into account. The most important parameters were those of the mildew intensity, the distribution of mildew in the crop, and the effect of mildew on assimilation at light saturation. Measured mildew epidemics in field experiments in three different years, were used to compute yield losses. Computed losses were compared to measured losses. On average, computed yield loss approached measured, but measured yield loss was underestimated, especially in early mildew epidemics due to the computation of partitioning and reallocation of assimilates. Other processes which may cause an underestimation are described. The use of crop models as a method to upgrade disease management systems is discussed.

Additional keywords: *Triticum aestivum*, *Erysiphe graminis* DC. ex Mérat, *Blumeria graminis* (DC.) Speer, simulation, sensitivity analysis.

Introduction

A vital question to upgrade disease management systems is whether damage functions, established in years with specific weather conditions, can be applied also in years with other weather conditions and crop husbandry practices. Though repeated measurements in many years and locations may answer this question, they will not lead to general methods to enable these extrapolations to be made. Models of crop growth, which compute yield from temperature and radiation, provide another option to answer the question if they can be combined with models of the pathogen. Rabbinge et al. (1985) quantified the effect of powdery mildew on photosynthesis of wheat leaves (Allen, 1942 and Walters and Ayres, 1984). How these effects on photosynthesis measured in the laboratory may be used to estimate loss of kernel yield and to what extend they explain yield loss caused by mildew in the field remained to be answered.

In a previous paper, the performance of three wheat models was compared to yields harvested in the Netherlands. It was indicated that the effect of weather in different years on yield could not yet be calculated accurately. Therefore, the main objective, the effect of weather on the damage function by mildew, could not be assessed in this study. Our objective for this paper was to introduce mildew in a wheat model, and to assess how this affects computed damage compared to measured damage in the field. By this comparison, crucial processes of the model determining damage are identified. In this paper, loss of kernel yield by mildew is called damage.

Materials and methods

Crop model. The general structure of the SUCROS87 model (Spitters et al., 1989) was chosen as a framework to quantify the effect of mildew. Computations were made with SUCROS87E (Daamen and Jorritsma, previous paper), an adapted version of the SUCROS87 winter wheat model (Spitters et al., 1989). Simulation begins at sowing time.

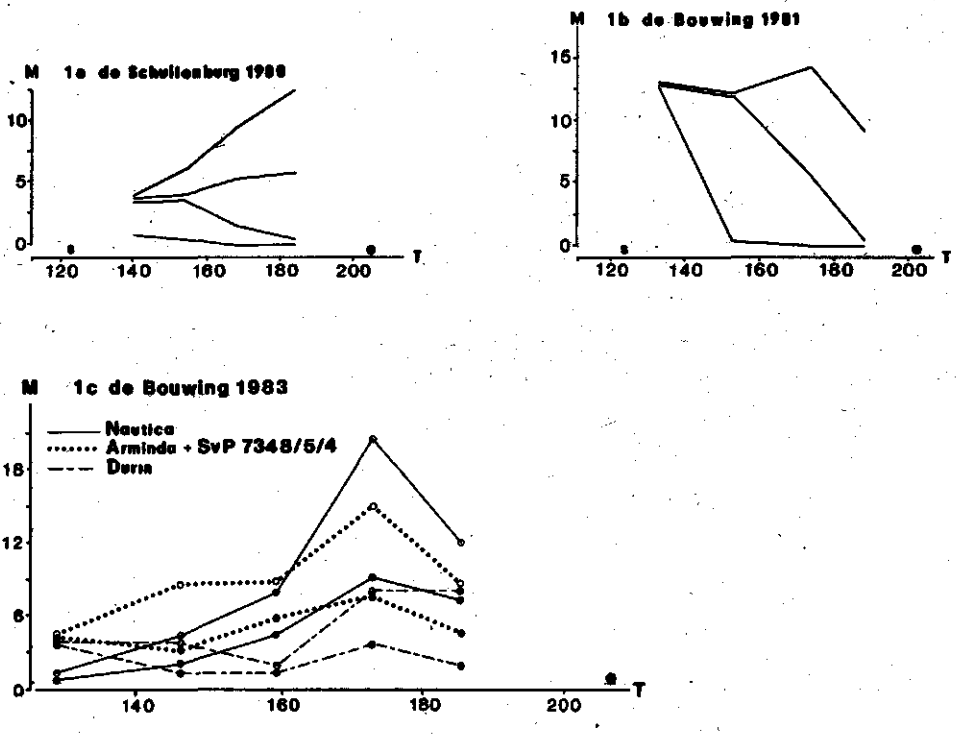

Fig. 1a,b,c. Courses of the mildew epidemics in the experiments (M is average number of mildew pustules per leaf, Day is days since 1 January), used as input in the simulation models. s is start of the epidemic, e is end of the epidemic and of kernel filling.

Field experiments. Simulated damage was compared with damage measured in three field experiments. They were near Wageningen, on the farms 'De Schuilenburg' in 1979-1980 and 'De Bouwing' in 1980-1981 and 1982-1983, and suffered light to moderate mildew epidemics (Fig. 1a,b,c). A detailed description of these experiments was given by Daamen (1988, 1989).

During the three cropping seasons, weather differed considerably (Fig. 2a,b, meteorological station Wageningen). In 1980, radiation was high during May and early June, but low in late June and early July, when temperature was below average. In 1982-1983, autumn and winter were relatively warm. Radiation was low during May and June, but high in July. The season 1980-1981 did not deviate much from average, though early spring was relatively warm with low radiation and summer had lower radiation than average.

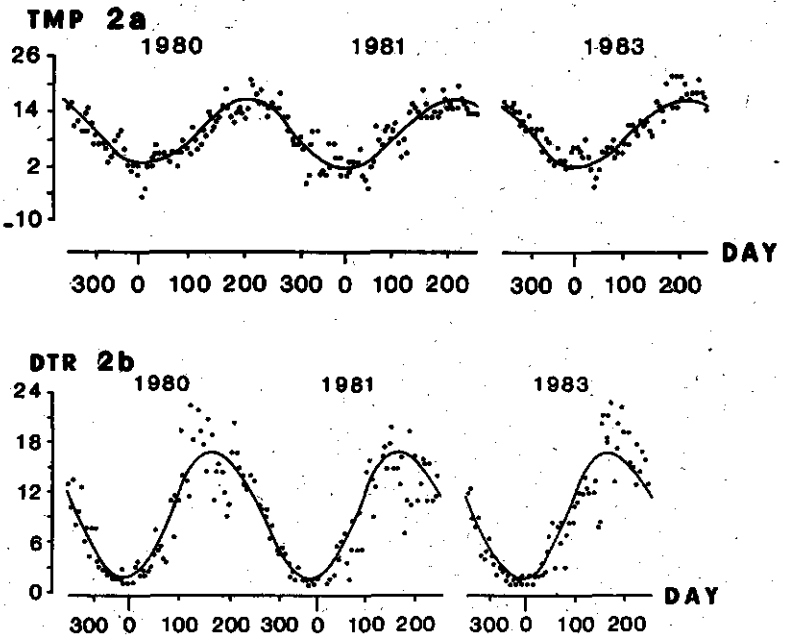

Fig. 2a,b. Average daily temperature (TMP, °C, average of five-days) and average daily total global radiation (DTR, MJ m^{-2} day^{-1}, average of five days) in Wageningen in the different cropping seasons. The curves are averages for Wageningen in 1950-1980.

Simulation of the experiments. The dates of sowing and end of kernel filling (DC83, Zadoks et al., 1974) were used to start and stop the simulations. Daily radiation and temperature (Fig 2ab) determined crop growth, and the measured mildew epidemics (Fig. 1) determined the damage. Mildew intensity was measured by Rabbinge et al. (1985) as the % leaf area diseased on single leaves both-sided (P_l), whereas in the field, average pustule number (M) at the upperside per leaf layer was determined. In the severity assessments made by Rabbinge et al. (1985), pustule size was about 0.04 cm^2 (Schans, 1983) which, at an average leaf size of 20 cm^2 (Daamen, 1989), corresponds with a rating of 0.2M % severity, uppersided, in the field (Ubels and Van der Vliet, 1979). Average mildew intensity, both-sided per leaf layer may be estimated at 1.5 times the upperside intensity (Daamen, 1986). Mean pustule number per leaf layer (M) in the field, is transformed to mean mildew severity both-sided per leaf layer (P) by (0.2 x 1.5):

$$P = a.M; \quad a = 0.3 \qquad (1)$$

Mildew profile. The number of leaves per culm, and the average mildew severity in each layer at each observation date describe the vertical distribution of mildew in the field and were used to simulate damage. Between observation dates, a linear interpolation of the disease intensity in each layer was made. Mildew on ears was negligible in the experiments.

To identify main parameters, artificial instead of measured mildew epidemics were used. The mildew profile was then estimated from the average mildew severity over all leaf layers ($\bar{P}$) or (Daamen, 1989):

$$P_d = \bar{P}.b.(L_d/L_t)^{b-1}; \quad b=3.4 \qquad (2)$$

in which P_d is the mildew severity at depth d in the canopy of leaves and L_d/L_t is the leaf area above depth d relative to total leaf area, which is computed by the model.

Effect of mildew on assimilation at light saturation. The effect of mildew severity P_i on the assimilation rate at light saturation (A_m, kg CO_2 ha^{-1} leaf h^{-1}) of single full-grown top leaves in relation to the development of the culm was studied by Schans (1983), Van Vrede (1983) and Jorritsma (1986). The relation is described by a hyperbolic equation with an additive effect of the developmental stage (equation 3 in: Rabbinge et al., 1985). In the crop model, the maximum photosynthetic capacity of healthy leaves in the canopy (A_{pm}) depends on the developmental stage and the temperature (Spitters et al., 1989). The hyperbolic equation with an additive effect of developmental stage (Rabbinge et al.,1985) was rewritten as a multiplicative one, by standardization at the average A_m of the measurements without disease stress (43.4; Jorritsma, 1986) at 20° C:

$$A_m = A_{pm}.(1 - (28 - \frac{87}{3.1 + P_i})/43.4) \qquad (3)$$

Effect of the horizontal distribution of mildew. As the effect of mildew on A_m is curvilinear (3), computations with the average of P_i per leaf layer will overestimate the effect on A_m. Though the exact distribution of pustules on full-grown leaves is not known, the negative binomial (Waggoner and Rich, 1981) was used to remove this bias. The dependence of the cluster parameter of this distribution on the mean pustule number (M) was estimated (Taylor et al., 1979) from a descriptive relation (equation 6 in: Daamen, 1986). Probability density functions of mildew at mean densities of M=1 to M=29 were generated by the negative binomial distribution, using the estimates of the cluster parameter, and (1) and (3) were used to estimate the effect of the average mildew severity P per leaf layer on A_m. The result was described by a negative exponential relation, see Fig. 3:

$$A_m = A_{pm}.(1 - c.(1 - e^{d.P})); \; P<10\ \% \qquad (4)$$

The dimensionless parameters: c= 0.46 (SE=0.01) and d= -0.29 (SE=0.01) described the computed data adequately at mildew severities below 10%; the SE of residuals was 0.007. Equation 4 was used in the model.

Other effects of mildew. The light use efficiency (E) tended to decrease with mildew intensity but was not significantly lower than that of healthy leaves (E_p) (Rabbinge et al., 1985). The model assumes no photosynthesis in the diseased leaf area, conform Waggoner and Berger (1987):

$$E = E_p.(1 - f.P/100); \quad f=1 \qquad (5)$$

in which parameter f is made explicit to facilitate the evaluation of this assumption. For other effects of mildew, such as on reflection and transmission of radiation and on maintenance and dark respirations,

diseased leaf area was assumed to behave like healthy leaf area.

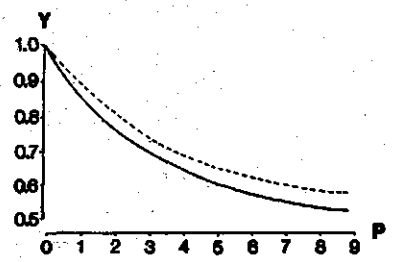

Fig. 3. Maximal photosynthetic capacity of wheat leaves at light saturation (Y, in relative units) in relation to the percentage mildew coverage (P). The solid line is the relation for individual leaves (P_1, equation 4), the broken line is the relation for the average mildew intensity in a leaf layer, taking the distribution of mildew into account (P, equation 5).

Results

Computed yields at the different mildew epidemics approached harvested (Table 1). Without mildew, computed yields were higher than harvested in 1980 and 1981 and nearly equal to harvested in 1983 (Table 1, disease free). Crop husbandry in 1980 was less intensive than in the other years. In 1981 lodging occurred in the experiment. Accordingly, end of kernel filling in the experiments in 1980 and 1981 took about two weeks earlier place than simulated, whereas such a difference was absent in 1983.

Damage by mildew was underestimated in the simulations for 1980 and 1981 ($p<0.05$), and almost exactly estimated for 1983. Underestimation of damage remained if simulations for 1980 and 1981 were not stopped by the observed dates of end of kernel filling.

Interpretation of simulated damage. From the experiments it is unclear whether differences in computed damage are due to different types of epidemics or to weather in the different years. Therefore, damage by artificial epidemics with a mildew stress of 240 pustule-days per leaf was computed at different periods in the three harvest years. In addition to damage, computed losses of total shoot weight are also given in Table 2. Mildew stress during vegetative growth affected kernel yield hardly, while damage due to mildew stress during kernel filling was high. Such a systematic effect was not observed in the loss of total shoot weight (Table 2). Simulated loss of total shoot weight was high during stress periods with high radiation, as in Spring 1980 and Summer 1983. Thus, simulated shoot weight depends mainly on the total amount of assimilates available, whereas simulated damage (loss of kernel weight) depends mainly on the assimilation during kernel filling. This difference is due to the computation of partitioning and reallocation of assimilates. These simulations show that the later the mildew stress, and the higher the radiation during kernel filling, the higher the computed damage.

In 1983, radiation was higher than average during kernel filling, and the epidemics were late, especially those in cvs. Nautica and Durin (Fig. 1,2). Radiation was slightly below average during kernel filling in 1980 and 1981. The mildew epidemic of 1980 was late and the 1981 one early. Thus, differences between years in simulated damage (Table 1) were mainly due to the different weather conditions and partly due to differences in courses of the epidemics.

The timing of the mildew stress within and between years has a clear effect on computed damage. In the field experiments, measured damage function did not depend significantly on year or on the developmental stage (Table 1 and Daamen, 1989).

Table 1. Measured and computed yields in percentages (in kg dry weight are $^{-1}$ between brackets) in relation to the mildew stress, expressed in the number of pustule-days per leaf.

Experiment	Mildew stress	Measured yield	Computed yield
De Schuilenburg 1980[1]			
cv. Caribo			
disease free	0	100 (63.3)	100 (70.4)
nearly disease free	21	99	100
early epidemic	132	98	100
suppressed epidemic	304	95	96
full epidemic	551	89	93
De Bouwing 1981[2]			
cv Okapi			
disease free	0	100 (66.0)	100 (70.9)
very early epidemic	194	97	100
early epidemic	569	94	98
full epidemic	850	89	95
De Bouwing 1983[3]			
cv. Nautica			
disease free	0	100 (79.1)	100 (78.7)
suppressed epidemic	332	96	94
full epidemic	649	92	88
cv. Arminda			
disease free	0	100 (77.1)	100 (78.7)
suppressed epidemic	378	95	95
full epidemic	588	92	93
line SVP 7348-5-4			
disease free	0	100 (77.3)	100 (78.7)
suppressed epidemic	358	95	95
full epidemic	711	91	90
cv. Durin			
disease free	0	100 (81.0)	100 (78.7)
suppressed epidemic	180	98	98
full epidemic	395	95	93

[1,2,3] year of harvest, measured relations are 0.012, 0.009 and 0.010 kg dry weight are $^{-1}$ damage per pustule-day of mildew per leaf, respectively. SED of measured yield is c. 2.2, 1.4 and 1.0 %, respectively.

Sensitivity analyses. To evaluate the importance of the parameters that quantify the effect of mildew in the model, kernel yields were computed at different parameter values for the growing seasons 1975-76 to 1985-86. Yields were computed at an artificial mildew epidemic, starting at day 120, increasing linearly up to day 180 and decreasing linearly to day 220, with a stress of 1000 pustule-days per leaf. This is a severe disease stress for the Netherlands. The response of the model to a change in value of parameter 'x', being a,b,c,d,f and the maintenance and dark respiration, respectively, was described. No estimates were available of their variances

Table 2. Computed losses due to early, middle and late artificial epidemics, each with a mildew stress of 240 pustule-days per leaf. Loss of kernel yield (damage) is given as percentage of those of clean crops. Loss of total shoot weight is given between brackets.

Epidemic	1980	1981	1983
Early, day 120-155	0.2 (2.1)	0.2 (1.5)	0.5 (1.4)
Middle, day 145-180	0.8 (1.0)	2.0 (1.3)	2.0 (1.8)
Late, day 170-205	3.4 (1.3)	4.3 (1.7)	6.1 (2.7)

in the field. Therefore, each parameter was set arbitrarily to a lower and an higher value, respectively, keeping the other parameter values at their estimated value. The responses of the model to changes in the values of the parameters was described by:

$$Yield_i = u_i + v.x + w_i.x + error;\ i\text{=harvest year} \qquad (6)$$

To identify main parameters, a standardized regression coefficient (v_m) was computed by multiplication of v from (6) with average(x)/average(Yield). This v_m is the percentage response in computed yield due to a percentage change in the value of parameter 'x'. It can be used to compare the effect that the different parameters have on the model if the average yield of the runs for each parameter are equal and if the average value of the parameters have a meaning. Here, the average yield of 83 kg dry weight are^{-1} did not deviate much and the average value of the parameters were best estimates, see methods. Results of the analyses are given in Table 3. Years explained most of the variance of computed yields (R^2-values about 0.9). Therefore, the R^2-values given in Table 3 were computed after elimination of the effect of u_i.

Response in yield by the model to a change in the disease stress itself gave a v_m-value of -0.06, with this artificial epidemic. This estimate equals the v_m-value of parameter a from equation 1 (Table 3). This was to

Table 3. Response of computed yields, with artificial epidemics of 1000 pustule-days of mildew per leaf, to changes in parameter values (x_h and x_l are higher and lower values, respectively) which quantify the effect of mildew, see text. Given are average values over the cropping seasons 1975/76 to 1985/86, by analysis of covariance (Equation 6). R^2-values were calculated after elimination of the effect of year. R^2error indicates deviations from linearity.

Equation	Parameter	$\bar{x}$	x_h	x_l	v_m*	$R^2v.x$	$R^2w_i.x$	R^2error
1	a	0.3	0.4	0.2	-0.06	0.989	0.008	0.003
2	b	3.4	4.4	2.4	0.07	0.958	0.015	0.026
4	c	0.46	0.56	0.36	-0.07	0.971	0.028	0.001
4	d	0.29	0.35	0.23	-0.04	0.986	0.012	0.002
5	f_{**}	1.	2.	0.	-0.02	0.992	0.008	0.000
-	R**	0.03	0.18	0.09	-0.01	0.990	0.010	0.000

* Standardized partial regression coefficient, average kernel yield was 83 kg dry weight are^{-1}, see text.

** Maintenance and dark respirations of diseased leaf area. The estimate without disease is 0.03 proportion leaf weight respired per day (25° C).

be expected as parameter a also determines the disease stress. The gradient parameter b of equation 2, characterizing the mildew profile and parameter c of equation 4, determining the maximum reduction of A_m by mildew, had similar v_m-values. These three appear to be the most important parameters. Parameter d of equation 4, determining at which disease intensity the maximum reduction of A_m is reached, had a lower v_m-value. The effect of parameter f of equation 5, determining the effect of mildew on the light use efficiency, was low. An increase of the maintenance and dark respirations (R) by mildew gave a v_m-value close to zero, thus it affected computed yield hardly.

On average, yield response by the model to changes in parameter values was low. This implies that the disease has relatively little effect on computed yield. Response to changes in parameter values could be described satisfactorily by a linear relation (Table 3, R^2v_2x). Interactions with year (R^2w_1x) and deviations from linearity (in R^2 error) were all relatively unimportant.

Discussion

Yield response of the model to changes in the value of parameter f was low. As this parameter determines the amount of radiation intercepted by the mildewed leaf area (f-1) this low v_m-value compared to the others indicates that mildew affects the crop mainly by changes in the momentaneous radiation use efficiency and not by shading (Waggoner and Berger, 1987 and Johnson, 1987). However, by a lower momentaneous radiation use efficiency, the rate of crop growth and leaf area growth is lowered, by which the consequent amount of radiation intercepted is lowered accordingly. Thus, damage by mildew cannot be attributed to the radiation use efficiency only, as this effect is confounded with the interception of radiation during the season. A consequence of this effect is that if crop growth is analysed in relation to the cumulative amount of radiation intercepted (Waggoner and Berger, 1987 and Lim and Gaunt, 1981), effects of diseases like mildew appear absent as small differences between the slopes of the relations are then hard to detect. To complete these analysis, the relation between the disease intensity and the total amount of radiation intercepted should be included.

Frame SUCROS87. Simulated damage due to powdery mildew approached measured, but on the average, the simulations underestimated measured damage. It was indicated that the underestimation in early epidemics was caused by the computation of the processes which determine the harvest index. However, if the computations would be made with a fixed harvest index, a systematic underestimation would be the result (Table 2). Therefore, also other processes leading to an underestimation are discussed. In the models, the assimilates produced on a day by mildewed leaves are added to those of healthy leaves. This bulk of assimilates is then partitioned and allocated to the different plant organs. However, it is questionable to what extent assimilates of mildewed leaves are transported to other organs in the plant (Heyland et al., 1979). For barley, Kern (1985) observed that the export of assimilates by mildewed leaves decreased, whereas the partitioning of assimilates was hardly affected. It is not clear from literature to what extent this reduced export is accounted for by the reduced assimilation assumed in this study. A mildew pustule may be thought of as a drain of carbohydrates (Edwards and Allen, 1966) and nitrogen, due to pustule growth and sporulation. Incorporation of this process (Van Roermund and Spitters, in prep) might improve the model. A related question is whether

compensation by healthy top leaves when lower leaves are diseased (Walters and Ayres, 1983) should be taken into account. Hwang et al. (1986) did not found such a compensation.

The process of leaf dying was not considered. In the model, the relative death rate of leaves depends on temperature only. But, when assimilation decreases by mildew, the net growth rate of biomass and of leaf area decreases, accordingly. The death rate is confounded with growth, because the formation of new leaves is not explicit in the model. At flowering, assimilates are no longer allocated to leaves. A reduction in assimilation then does not lead to an increased death rate in the model, but in the field it does (Finney, 1979; Paulech and Haspelová-Horvatovicová, 1986). This is not a plea to model leaf death depending on mildew intensity, as no direct mechanism seems to operate (Walters and Wylie, 1986).

Light interception and photosynthesis were computed taking shading into account. Computed damage depended strongly on the steepness of the mildew profile. Lower leaves, being heavily diseased, had thus low photosynthetic capacities. This effect of mildew runs parallel with the effect of ageing (Rawson et al., 1983). Therefore, leaf death may be simulated in relation to ageing and shortage of carbohydrates in a leaf layer, caused by shading and by mildew.

Nitrogen. To simulate damage at different input levels of crop husbandry, the mildew intensity should be translated first into processes which govern the nitrogen dynamics. Murray and Ayres (1986) report that in mildewed barley seedlings the nitrogen uptake rate per mg root was considerably reduced and that the loss of N from the leaves as a result of sporulation was comparatively small. The reduced uptake partly explains the reduction in A_m by mildew (Walters, 1985). When the epidemic proceeds, reduced growth of the plant reduces the N demand by the plant. Considering that a high level of fertilization enhances epidemics of mildew, the nitrogen dynamics in soil, plant and pathogen, appear to have many feedback mechanisms which should be considered if damage by mildew at different levels of crop husbandry is to be modelled.

Crop models and disease management. To bypass some of these problems, measurements in the field of biomass of the different organs of the crop can be made in spring to initialize the model. The processes leading to damage can then be studied accurately. The question remains whether the models will lead to general methods and have practical advantages in terms of labour requirement above traditional yield loss studies. If they do not have these advantages, the spin-off and other informal activities in relation to the objective of the work become important. The development of general methods to construct and apply damage functions under various weather conditions and crop husbandry practices is urgently needed. Though crop models provide a physiological frame for yield loss studies, they require more quantitative physiological knowledge. If the target is to actualize a disease management system, the effort should concentrate on field experiments, which give in addition to a damage function, more information on disease management.

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Samenvatting

Effecten van meeldauw en weersomstandigheden op opbrengsten van wintertarwe. 2. Effecten van meeldauwepidemieën

Opbrengstderving van wintertarwe werd berekend aan de hand van het effect dat meeldauw heeft op de blad-fotosynthese. Een rekenmodel voor de gewasgroei van tarwe werd uitgebreid met meeldauw. Met inachtneming van de vertikale en horizontale verdeling van meeldauw in het gewas, werd meeldauw in het model gekwantificeerd door vijf parameters. De belangrijkste parameters waren die van de meeldauwintensiteit, de verdeling van meeldauw in het gewas en het effect van meeldauw op de assimilatie bij een overvloed aan licht. Epidemieën van meeldauw, gemeten in veldproeven in drie verschillende jaren, werden gebruikt om opbrengstdervingen te berekenen. Gemiddeld kwam deze redelijk overeen met de in de veldproeven gemeten opbrengstderving. De gemeten opbrengstderving werd echter onderschat, vooral bij vroege epidemieën van meeldauw door de wijze waarop de (her)verdeling van assimilaten wordt berekend. Andere mechanismen, die een onderschatting van opbrengstderving kunnen veroorzaken worden besproken. Of deze modellen als methode gebruikt kunnen worden om systemen voor de geleide bestrijding van ziekten te verbeteren wordt bediscussieerd.

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Part V
EPILOGUE

Towards an integrated production system

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Chapter 9

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Epidemics of Diseases and Pests of Winter Wheat at Different Levels of Agrochemical Input

A study on the possibilities for designing an integrated cropping system*

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With one figure

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Abstract

The cropping system with winter wheat has changed considerably during the past fifteen years in the Netherlands. Crop biomass has been increased by use of new cultivars and higher levels of fertilizers and pesticides. The question which arises is to what extend the level of fertilization affects epidemics of pests and diseases, compared to the well-known effects of pesticides and cultivars. Epidemics observed in the winter wheat fields of the Development Farming System project (DFS) at Nagele, in 1984 and 1985 provided data for this analysis. Higher levels of fertilization stimulated epidemics of yellow rust, mildew, snow mould, leaf miners and cereal leaf beetles in the fields. The magnitude of the effect was comparable to those of the pesticides used and the cultivars sown. The level of fertilization showed no effect on five other species. How these interrelations between cropping practices and the associated development of pests and diseases can be used to design an integrated approach towards cereal cropping and breeding is discussed.

Zusammenfassung

Epidemien von Krankheiten und Schädlingen in Winterweizen bei Einsatz von verschiedenen Intensitäten von Agrochemikalien Eine Vorstudie zum Entwurf eines integrierten Anbausystems

Das Anbausystem mit Winterweizen hat sich in den Niederlanden in den letzten fünfzehn Jahren beträchtlich verändert. Die Biomasse des Weizens stieg an durch die Verwendung von neuen

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Sorten und höheren Einsatz von N-Düngung und Pflanzenschutzmitteln. Es fragt sich, inwiefern die Düngungsintensität die Entwicklung von Krankheiten und Schädlingen beeinflußt, im Vergleich zu den bekannten Effekten von Pflanzenschutzmitteln und Weizensorten. Diese Frage wurde geprüft anhand von Beobachtungen über den Verlauf der Epidemien in Winterweizenschlägen in Nagele während der Jahre 1984 und 1985 ('Development Farming System' Projekt, DFS). Höhere N-Düngungsstufen stimulierten das Auftreten von Gelbrost, Mehltau, Schneeschimmel, Minierfliegen und des Getreidehähnchens. Das Ausmaß der Beeinflussung war vergleichbar mit dem durch die eingesetzten Pflanzenschutzmittel und Weizensorten. Fünf andere Schadorganismen wurden durch die Düngungsintensität nicht beeinflußt. Diskutiert wird, wie diese Interaktionen zwischen Anbaukomponenten und Krankheiten und Schädlingen zur Entwicklung eines integrierten Anbausystems und zur Züchtung von neuen Winterweizensorten genutzt werden können.

Many factors may affect crop growth and epidemics of diseases and pests in a cropping system (PALTI 1981). The cropping of cereals has changed considerably in the past fifteen years in the Netherlands. Annual increase in that period of gross winter wheat yield averaged 0.19 ton ha^{-1} or 3.2 %. This was made possible by the introduction of new cultivars which have, at the one hand a high harvest index and, on the other, that are early maturing and short strawed, so that they can withstand high levels of fertilizer application. As a consequence, the risk of yield loss due to disease and pest development and of lodging increased, which made necessary a higher level input of fungicides (since 1975), growth regulators (since 1976) and aphicides (since 1980)[a] (DE VOS and SINKE 1981, DILZ *et al.* 1982). Nitrogen application has increased from 45 kg ha^{-1} in the early fifties (DE VOS 1968) to 100 kg ha^{-1} in the mid seventies[a] and may now be 160 kg ha^{-1} on average. In intensive wheat production, nitrogen application may now be as much as 230 kg N ha^{-1}.

Crop biomass and its nitrogen content has increased (AUSTIN *et al.* 1980, SPIERTZ and DE VOS 1983). Crop height has been lowered by the use of short strawed cultivars and growth regulators, although in general crop height increases at high levels of fertilization. Through higher crop biomass and lower crop height, crop density has increased considerably. Cropping period and leaf persistance has been lengthened by the higher nitrogen levels, mainly by a longer ripening period (ANGUS and MONCUR 1985). Heading date has not changed, since the effect of nitrogen on crop development was counterbalanced by the earlier heading of new cultivars. Leaf duration before heading has presumably also been relatively increased. As in barley (RIGGS *et al.* 1980) modern wheat cultivars have a fewer number of final leaves than old cultivars, which is correlated with early maturity (HOOGENDOORN 1984).

How can these changes in crop growth, structure and fenology be related to the observed increase in pest and disease development? Firstly, the growth rate of pathogens and pests may depend strongly on the N-content of host tissue, for example for mildew, yellow rust and aphids. The cause of this interaction is presumably complex, since fertilizers may also affect cell size, thickness of cuticule and leaves, the amount of water-soluble carbohydrates etc. Secondly,

[a] Source: Kwantitatieve informatie voor de akkerbouw en groententeelt in de vollegrond. Publikaties PAGV, Lelystad, the Netherlands.

short crops are less capable of escaping from ear infections by blights and blotches (BRÖNNIMANN 1969), though the nature of the resistance correlated with tall and late maturing cultivars is still under discussion (DANON *et al.* 1982, SCOTT *et al.* 1982 and ROSIELLE and BOYD 1985). Thirdly, through a higher biomass and density of the crop, the microclimate in the canopy changes. Amplitude of the profiles of temperature and humidity in the canopy will diminish and the duration of leaf wetness will increase, which is favourable for the development of the so-called 'wet diseases' (SCOTT *et al.* 1985). Fourthly, a high crop density, with a lower death rate of leaves, and a longer cropping period with a shorter host-free period also affects disease development directly. The number of life-cycles, the death rate during cropping and during the host-free period and the probability of pathogen or pest landing on a compatible host are all affected in favour of increased disease and pest development.

The question which now arises is how much these factors affect epidemics, compared to the well-known effects of differences in cv. resistance and the use of pesticides. As diseases may show species specific reactions on changes of the host, more detailed knowledge of their reaction on low or high biomass crops cq poor or rich crops is needed, in order to have a better insight into the possibilities for managing the crop ecosystem.

In the Development Farming System project (DFS) at Nagele, three farming systems, ca 20 ha each, are being developed and studied, namely: Biodynamic (B), Integrated (I) and Conventional (C) (VEREIJKEN 1986a, 1989). The three systems are annually adapted and continuously improved. The integrated system is being developed from a less intensive conventional into a real integrated system. Epidemics in the winter wheat fields (ca 2 ha each) of these farming systems were followed to evaluate the effect of fertilization compared to the effects of pesticides and cultivars. It is assumed that field size is large enough to sustain endogenous epidemics typical of the farming system, though it should be realised that changes of some factors, such as the host-free period, may show effects only if taken on a regional scale. In a previous paper, the observations in winter barley fields were reported (DAAMEN *et al.* 1988).

Methods

In 1984 and 1985, winter wheat fields on the DFS were sampled fortnightly from May onwards. The sample size was 100 tillers for stem base diseases and aphids and 40 tillers for leaf and ear diseases. Disease intensity was determined as incidence, defined as the percentage of tillers with stem base diseases and aphids and the percentage of leaves or glumes with disease. Only disease symptoms in the green leaf area were considered. Sample variance of disease incidence can be described by that of the binomial distribution.

In the DFS project, cereals are cropped in a four year rotation on Conventional (C) and Integrated (I) farms and in a five year rotation on the Biodynamic (B) farm. The concepts of the integrated cropping programmes for winter wheat in comparison to the other cropping programmes have been treated by WIJNANDS and VEREIJKEN (1986) and by VEREIJKEN (1989). Characteristics of the different wheat cropping systems are given in Table 1 (VEREIJKEN 1986b, 1988). The seedbed was prepared by using a rotary harrow on ploughed land. Row distance was 12.5 cm in all fields. Cultivars were selected mainly on their disease resistance and weed competitive value for the I and B fields and for their yield potential in C fields. Cultivar choice was partly determined by the wish for the same

cultivar on each system, to enable a comparison with the developing integrated system. Estimated amounts of mineral N in the soil (0—90 cm) in February were 67 and 52 kg N ha^{-1}, on the C farm in 1984 and 1985 respectively, and 48 and 36 kg N ha^{-1} on the I farm in these years. Nitrogen was applied as calcium ammonium nitrate on C and I fields, in three split-applications. In 1984, 80, 40 and 40 kg N ha^{-1} was given respectively on 20 March, 3 May and early June. In 1985, 104, 60 and 40 kg N ha^{-1} was given respectively on 22 February, 7 May and 7 June. C and I fields also received 30 ton ha^{-1} sugarbeet leaves and tops, left over by the preceding crop (ca 30 kg N ha^{-1}) in both years. In B fields, organic manure was used in other crops in the rotation.

In 1984, C and I fields were sprayed aerially with the fungicides Bayleton and Captafol (0.5 kg and 1.5 l ha^{-1}) and a selective aphicide (Pirimor, 250 g ha^{-1}) on 4 July. In 1985, C Okapi was treated with Corbel, 1 l ha^{-1} on 12 June; C Saiga with Bayfidan and Captafol (0.5 + 1 l ha^{-1}) on 17 June; I Okapi with Bayfidan and maneb (0.5 l + 2 kg ha^{-1}) on 4 June and C and I fields with Pirimor on 11 July, all by a field sprayer.

Ear number was not determined but averaged 580 in C fields, 490 in I and 320 in B fields in 1982 and 1983 (UBELS and VAN DER VLIET 1985), which shows the significant effect of the cropping system on stand density. B Manella and C Saiga had a slightly faster development than Okapi in 1984. Manella was sown earlier than Okapi. In 1985, B Okapi was faster in development than the other crops, due to a lower level of fertilization (GLYNNE 1951, ANGUS and MONCUR 1985). Number of green leaves per tiller developed similarly in the different crops. The crops of farming system B had fewer leaves at ripening in both years and during heading in 1984, and during shooting in 1985, indicating a slightly higher death rate of leaves. N content of leaves was not determined.

Resistances of the cultivars to different diseases are given in Table 3, according to the Dutch descriptive list of cultivars (RIVRO); if not available, they were derived from disease survey reports (DAAMEN and WIETSMA 1984, STOL 1985). Okapi is, according to the list, a rather tall to tall and rather early to early heading cv., Manella is rather tall and early to very early heading and Saiga is a rather short and rather early heading cultivar.

Table 1
Characteristics of the different systems of wheat production

Farm type	C, Conventional		I, Integrated		B, Biodynamic	
Year	1984	1985	1984	1985	1984	1985
Previous crop	s.beet	s.beet	s.beet	s.beet	potat.	peas
Cultivar	Okapi and Saiga		Okapi	Okapi	Manella	Okapi
Sowing date, m. d	1102	1101	1102	1101	1015	1030
Seed rate, kg ha^{-1}	175	170	175	170	180	195
N^1 available, kg ha^{-1}	227	256	208	240	60	120
Seed dressing[2]	yes	yes	no	no	no	no
Growth regulator[3]	yes	yes	no	no	no	no
Herbicides	yes[4]	yes[4]	no	½ field[5]	no	no
Harrowing	yes[6]	yes[7]	yes[8]	yes[9]	no	no
Gross yield ton ha^{-1}	8.6	6.7	8.2	6.0	5.2	5.5

[1] Estimated amount mineral nitrogen in the soil in February and dressings.
[2] Panoctine 35, 3 ml kg^{-1}, a.i. guazatine 35 %.
[3] CCC, 1.5 l ha^{-1} on C Okapi on 3 May 1984 and on C Okapi on 6 May 1985.
[4] MCPP, 4 l ha^{-1} and/or MCPA, 5 l ha^{-1}.
[5] Asepta anitop, 3.5 l ha^{-1}.
[6] On 21 March, at undersowing of perennial ryegrass, 30 kg ha^{-1}.
[7] On 11 April, at undersowing of perennial ryegrass, 18 kg ha^{-1}.
[8] On 23 March, at undersowing of white clover cv. Tamar, 17 kg ha^{-1}.
[9] On 11 April, at undersowing of red clover cv. Violetta and perennial ryegrass, 10 + 10 kg ha^{-1}.

Table 2
Sampling dates, development stage (DC) and number of leaves per tiller

	1984							1985							
Date m. d	508	522	605	621	704	719	731	502	514	528	611	625	709	723	730
DC	27	32	41	61[1]	69	77	83[1]	25	30	33	49[2]	61[2]	73[2]	79[2]	85[2]
Number of leaves:															
C Saiga	4.0	4.7	5.2	4.3	3.5	2.8	2.2	3.3	3.1	4.0	3.9	3.2	3.0	2.5	1.3
C Okapi	3.8	5.1	5.5	4.4	3.9	3.3	2.8	3.0	2.6	3.8	4.1	3.7	3.3	2.5	1.7
I Okapi	3.5	4.5	5.5	4.5	4.0	4.1	2.5	3.2	3.0	4.2	4.0	3.7	3.4	2.8	1.4[3]
B Man/Okap	3.6	5.6	4.8	3.5	3.5	2.8	1.2	2.8	2.4	3.4	4.1	3.8	3.4	1.5	0.0

[1] C Saiga and B Manella further in development.
[2] As [1] for B Okapi.
[3] Lodging.

Results

Observed epidemics are given in Fig. 1a to 1m and in Table 4. They will be discussed by species, first, the stem base diseases, followed by leaf and ear diseases and pests.

Pseudocercosporella herpotrichoides

Eyespot intensity was moderate in 1985, and below 10 % in 1984. Of the C fields in 1985, Saiga as a more susceptible cultivar than Okapi was more infested (Fig. 1a). I Okapi was more infested than C and B Okapi, for unknown reasons. In 1984, no significant trends due to cv. or farming system were observed (Table 4).

Rhizoctonia cerealis

Sharp eyespot incidence was low in 1985 and low to moderate in 1984 (Table 4). In agreement with survey data, C Saiga tended to be less infected than C Okapi, in 1984. B fields were slightly less infected than C and I Okapi.

Table 3
RIVRO disease resistance ratings[1]

Cultivar		Saiga	Okapi	Manella
Eyespot,	*P. herpotrichoides*	6	7.5	5
Yellow rust,	*P. striiformis*	7—6	6—5	6
Brown rust,	*P. recondita*	6.5	5.5	5.5
Mildew,	*E. graminis*	6	5	5
Leaf spot,	*Septoria* spp.	6	7	4[2]
Ear blight,	*Fusarium* spp.	7	6.5	5[2]
Ripening diseases		6.5	7.5	5

[1] Higher ratings indicate greater resistance.
[2] UBELS *et al.* 1980.

Table 4

Percentage culms, leaves or glumes infested by diseases which occurred at a low intensity in the different fields. Entries are average incidence at DC > 60

Fields	C Saiga	C Okapi	I Okapi	B Manella[1]
Culms:				
1984 eyespot	3	4	7	4
1984 sharp eyespot	3	21	11	4
1985 sharp eyespot	4	3	5	2
1984 *Fusarium* brown footrot	6	7	10	19
Leaves:				
1984 yellow rust	0	2	4	0
1985 glume blotch	4	4	2	0
1984 snow mould	2	1	1	0
1984 cereal leaf beetle	8	5	8	3
Glumes:				
1985 glume blotch	2	1	3	0
1985 ear blight	10	8	10	11

[1] In 1985: cv. Okapi

Fusarium spp.

Fusarium brown footrot intensity was moderate in 1985 and light in 1984. C Okapi was more infested than I and B Okapi in 1985 (Fig. 1b), which might be caused by the use of seed dressing in C fields, which suppressed antagonists or allowed more diseased plants to survive (UBELS and VAN DER VLIET 1984). C Saiga also showed this trend. In 1984, B Manella was more infested than C or I fields (Table 4); the relative resistance of Manella is unknown, but the effect may have been due to the early sowing of Manella.

Puccinia striiformis

Yellow rust was nearly absent in 1985. In C and I Okapi, up to 5 % leaves were diseased in 1984, while the more resistant cv. C Saiga and B Manella were healthy (Table 4). In 1983, a moderate epidemic developed in C and I Okapi (Fig. 1c, UBELS and VAN DER VLIET 1985) while in B Okapi, without treatment, only a trace of rust was found, which is attributed to the well-known effect of fertilizers.

Puccinia recondita

In 1984 and 1985, moderate brown rust epidemics developed in the untreated B Manella and B Okapi, which are equal in resistance (Fig. 1d, e). In 1984, fungicide treatment in C and I fields controlled epidemics, except in I Okapi due to a bad aerial application in this field. The epidemic stopped in B Okapi in 1984 and 1985 due to leaf death. Brown rust epidemics were not accelerated by the higher level of fertilizers, unlike yellow rust.

Erysiphe graminis

Mildew epidemics were moderate in both years (Fig. 1f, g). Mildew intensity was higher in I Okapi than in C Okapi in 1985 for unknown reasons. Fungicides did not gave complete mildew control but arrested the increase for 3—4 weeks. In I Okapi in 1984, no control was achieved due to bad application. Saiga, being more resistant than Okapi was only slightly attacked. Mildew intensity in B fields, without treatment at a low level of fertilization, was negligible.

Mycosphaerella graminicola

Speckled leaf blotch caused moderate to severe epidemics in 1985 and 1984 (Fig. 1h, i). In both years, no significant difference in disease development due to farming system was found in May. The interpretation of the course of the epidemics of 1985 is rather complicated, as different fungicides were applied at different timings. In 1985, application of Bayfidan and maneb on 4 June in I Okapi gave moderate control of the disease in June—July, compared to the treatment with the less effective fungicide Corbel on 12 June in C Okapi, which gave hardly any control. Disease intensity rose at the end of July in I Okapi, presumably due to lodging. The more susceptible cv. C Saiga had the same disease intensity as C Okapi in June. In July, C Saiga was less infected than the Corbel treated C Okapi, due to treatment with the more effective fungicides Bayfidan and Captafol on 17 June. The untreated B Okapi had the same disease intensity as Corbel-treated C Okapi, which indicates that fertilization level did not affect disease development early in the season. In July, intensity in B Okapi decreased due to the increased leaf death.

Epidemics were less complicated to interpret in 1984. C Saiga and B Manella, being more susceptible, had more leaf blotch than cv. Okapi in July. Manella was also sown earlier. Fungicide treatment on 4 July did not gave a significant control in C and I fields. Application date was presumably too late for this disease.

Leptosphaeria nodorum

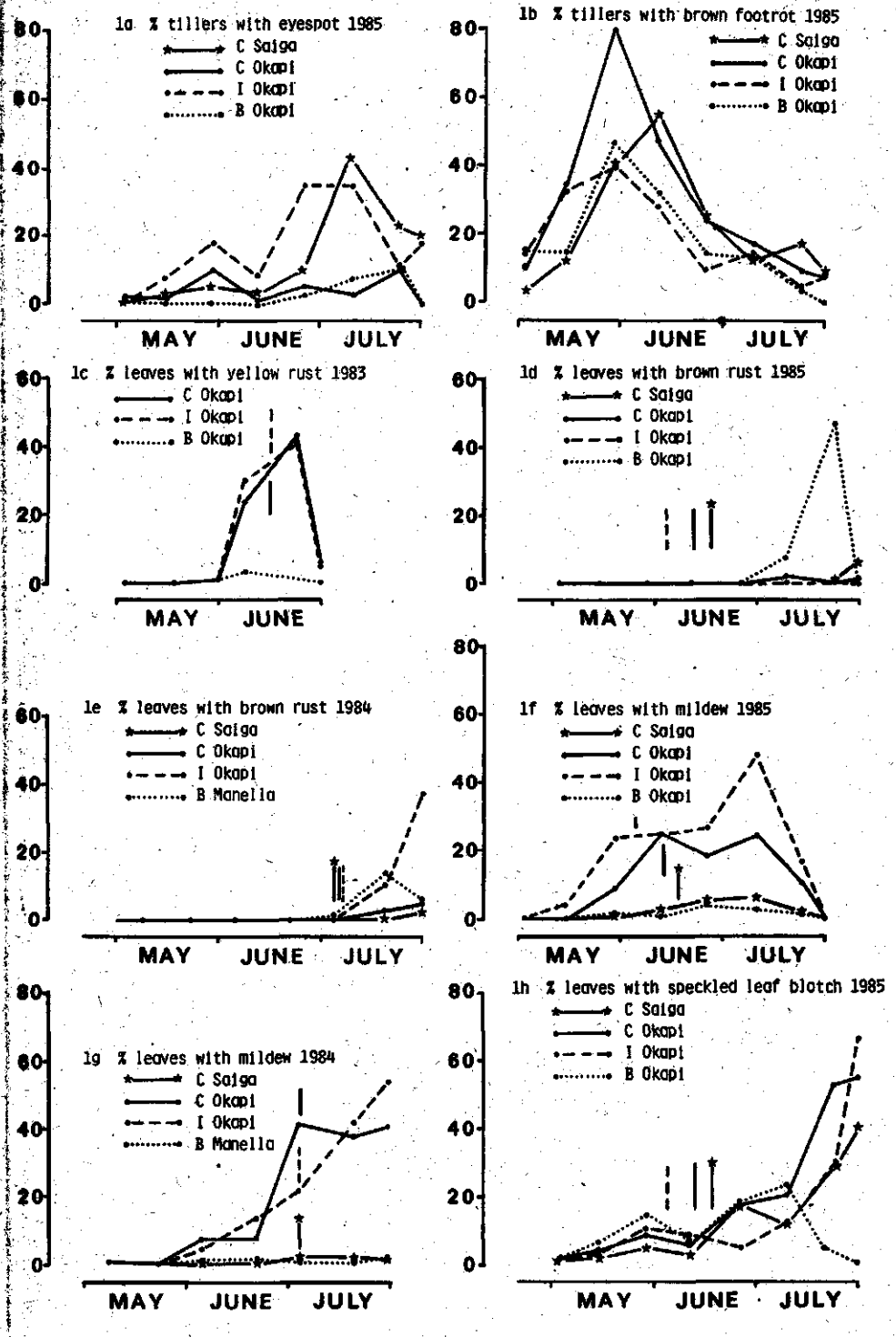
Glume blotch attack was negligible in 1984. Disease incidence was below 5 % leaves or glumes in 1985 (Table 4), which is the usual pattern on these clay soils.

Gerlachia nivalis

Snow mould attack was low in 1984 (Table 4), even absent in B Manella, and moderate in 1985. Despite fungicide treatment on C and I fields, they were more heavily infested than B fields, due to the different levels of fertilization (Fig. 1j). Ear blight (*Fusarium* spp.) was negligible in the fields in 1984 and attacked ca. 10 % of the glumes in 1985; no systematic system or cultivar effect was observed (Table 4).

Leaf miners

In 1984 leaf damage was negligible. In 1985, leaves of C Saiga were slightly more damaged than those of C Okapi. Resistance of the cvs. is not known and


1a % tillers with eyespot 1985
C Saiga
C Okapi
I Okapi
B Okapi
1b % tillers with brown footrot 1985
C Saiga
C Okapi
I Okapi
B Okapi
1c % leaves with yellow rust 1983
C Okapi
I Okapi
B Okapi
1d % leaves with brown rust 1985
C Saiga
C Okapi
I Okapi
B Okapi
1e % leaves with brown rust 1984
C Saiga
C Okapi
I Okapi
B Manella
1f % leaves with mildew 1985
C Saiga
C Okapi
I Okapi
B Okapi
1g % leaves with mildew 1984
C Saiga
C Okapi
I Okapi
B Manella
1h % leaves with speckled leaf blotch 1985
C Saiga
C Okapi
I Okapi
B Okapi
MAY
JUNE
JULY
0
20
40
60
80

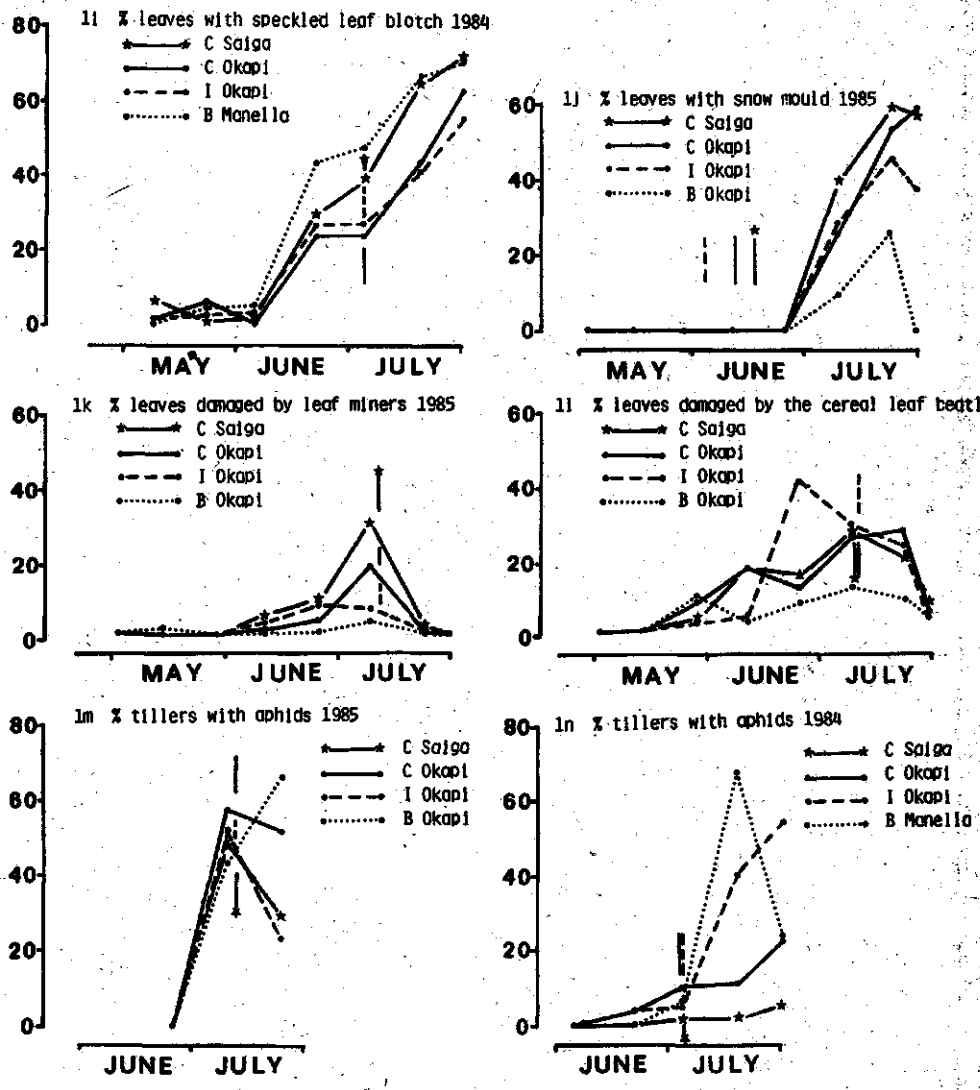


Fig. 1a to n. Diseases and pests in the different winter wheat production systems. Vertical bars indicate treatments with pesticides

survey data do not indicate differences in resistance. More leaves of C Okapi and C Saiga were mined than those of B and I Okapi in 1985 (Fig. 1k).

Lema cyanella

In 1984, leaf damage by the cereal leaf beetle was low, though B Manella had lowest damage (Table 4). In 1985, I Okapi was less damaged on 11 June and more on 25 June than C Okapi, for unknown reasons. As in 1984, leaves of B Okapi were also less skeletonized than those of C and I fields in 1985 (Fig. 1l).

Aphids

The grain aphid (*Sitobion avenae*) predominated over *Metopolophium dirhodum* and *Rhopalosiphum padi* in both years. Aphid intensity increased at the end of June— early July, without showing a significant effect of farming system (Fig. 1m, n). C Saiga was less infected than C Okapi in 1984, but not in 1985. In commercial fields Saiga is usually less infested with aphids than Okapi. Aphid control on C and I fields was not complete in both years and nearly absent in I Okapi in 1984, due to the bad aerial application of insecticide in this field. In B fields, aphids increased compared to the well treated C and I fields, in both years. In 1984 aphids decreased at the end of ripening in B fields, due to earlier ripening while they still continued to increase in C and I fields. This could not be found in 1985, as aphids were not sampled at that time.

Symptoms of *Pyrenophora sorokiniana*, *Pyrenophora tritici-repentis*, take-all, ergot, smuts, stem rust, BYDV, Hessian fly and Frit fly were not found. Intensity of blossom midges was not assessed.

Discussion

An evaluation of the effect of crop husbandry practices on the development of pests and pathogens in mostly done is factorial designed experiments, which results may indicate the direction of the effects. A comparison of epidemics on field and farming system level, without a balanced factorial design of prefixed levels of many factors, is a risky exercise. Another complication of such experiments is that large fields are needed by which replicates in the same year are very costly. However, experiments at the field and farming system level are necessary to evaluate the impact of the crop husbandry practices on epidemiology in its proper scale, and in relation to the complete system of cultural practices on the farm which, in this paper was for two years on the DFS-farms at Nagele.

Firstly, the cropping methods for winter wheat differed in fertilizer availability which varied from ca 80 kg N ha^{-1} in the biodynamic system to ca 225 kg N in the integrated and ca 240 kg N ha^{-1} in the conventional system. The stem base diseases, brown rust and aphids did not show a significant reaction. Stimulation of yellow rust, mildew, snow mould, leaf miners and the cereal leaf beetle was observed at the higher fertilizer levels, though fertilization level might be confounded with the level of pesticide use and thus with the level of antagonists, predators and parasitoids. Moreover, most leaf diseases showed a decrease in intensity due to accelerated leaf death at ripening at lower levels of fertilization, and aphids decreased due to earlier ripening.

Secondly, the cropping methods varied in cultivars sown and pesticides applied. Differences in cultivar resistance affect epidemics in any farming system. Also the use of pesticides affected epidemics, though control achieved was far from complete. There was an indication that survival of *Fusarium* brown footrot increased by the use of seed dressing.

Thirdly, the cropping methods differed in application of growth regulators and dates of sowing. An effect of the use of growth regulators on disease

epidemics was not observed. Earlier sowings affected presumably the intensity of *Fusarium* brown footrot and speckled leaf blotch.

Though it is premature to draw strong conclusions from these data, fertilizer use seems to have an important effect on disease and pest development. The effect was often of the same order as that of cultivar resistance or pesticide use. The obtained results are used to rank the diseases and pests whether they show a positive reaction to an increased level of fertilization.

Tentative grouping of diseases and pests for their positive response to an increased level of fertilization and its associated effects on the crop.

Strong	Moderate	Neutral
mildew	snow mould	eyespot
yellow rust	cereal leaf beetle	*Fusarium* brown footrot
	leaf miners	brown rust
		speckled leaf blotch
		aphids

The classical examples mildew and yellow rust (LAST 1954, DARWINKEL 1980 and RIJSDIJK 1980) showed a strong reaction to the level of fertilization. Snow mould and the leaf damaging insects were grouped as moderate. ELLEN and LANGERAK (1987) report a dependency of snow mould intensity on nitrogen level. No reference in the literature to such a dependency for the leaf damaging insects was found, but in general, insect growth and reproduction increases with higher N-contents of food (ENGLEMANN 1970).

Half the disease and pest species were grouped as neutral. Considering the stem base diseases, HEITEFUSS *et al.* (1977) and EL TITI and RICHTER (1987) report a slight decrease of stem base diseases at increased fertilizer levels. PREW *et al.* (1986) also report a slight decrease of eyespot intensity at high fertilization level, but HERRMAN and WIESE (1984) report no significant effect of fertilizers on eyespot, whereas GLYNNE (1951) reports an increase. Thus, the relation of eyespot intensity to the level of nitrogen fertilizers is not consistent in the literature; if there is any effect it is likely, therefore, to be small. BROSCIOUS and FRANK (1986), PREW *et al.* (1986) and PUMPHREY *et al.* (1987) report no significant effects of the level of fertilization on the other stem base diseases *Fusarium* brown footrot and sharp eyespot, though PAPENDICK and COOK (1974) showed an increase of *Fusarium* brown footrot at high nitrogen fertilizer rates, caused indirectly by plant water stress.

Of the leaf diseases, brown rust and speckled leaf blotch were neutral. There is little reference in the literature to a dependency of brown rust on fertilizers, indicating that level of fertilizers is not important for epidemics (ROELFS and BUSHNELL 1985). BOQUET and JOHNSON (1987) found a positive linear association of brown rust infection with increasing N rate, but only in a year when the epidemics were severe and yields accordingly low, but not in two years with moderate epidemics and higher yields. EL TITI and RICHTER (1986) did not found a systematic effect of farming system on the moderate brown rust epidemic of

1983. Speckled leaf blotch did not show a reaction conforming to literature references. FELLOWS (1965) reports a stimulation of infection by fertilizer application. Also, an increased crop density due to high fertilizer levels is supposed to stimulate the 'wet diseases' (SCOTT *et al.* 1985).

Aphids (mainly *S. avenae*) were also uneffected by the fertilizer level. Based on physiological knowledge (VEREIJKEN 1979, DIXON 1987), a stimulation of aphid epidemics by N fertilizers was expected and EL TITI and RICHTER (1987) confirm this expectation. On the other hand, ERLINGSON (1983) and PREW *et al.* (1986) did not observe such a dependency for *S. avenae* in field experiments. ERLINGSON (1983) found such a dependency for *R. padi* in spring barley, so aphids may show a species specific reaction.

Thus, we may conclude that most observations are in agreement with literature references. More observations need to be made on the behaviour of speckled leaf blotch and the aphid species in their dependency on fertilizers. Moreover, it should be realized that the effect of fertilizers on disease development is complicated by the timing of the application, the N : P : K balance (Anonymous 1976) and the form of the nitrogeneous fertilizer (HUBER and WATSON 1974).

On the developing integrated farm of the DFS, the emphasis is shifted from greater production to cost reduction and improvement of quality of both products and production ways, through substitution of expensive and potentially polluting inputs, especially fertilizers and pesticides by both agricultural and ecological knowledge, labour and non-chemical husbandry techniques. At the meantime, encouragement and conservation of flora and fauna in and around the fields is strived at, to stabilise the agro-ecosystem as a major preventive measure against the outbreak of pests, weeds and pathogens (work definition of the IOBC working group 'integrated farming systems'). A sound combination of cultural practices ranging from site choice and crop rotation over cultivar choice, seed rate, sowing-date and -method to fertilization can form the basis of integrated cropping methods. This combination should be directed towards prevention to lower the risk for outbreaks of pathogens and pests.

Disease advisory systems which evaluate pest and pathogen intensity economically must be used to determine justified control measures. When control measures are needed, a proper use of selective and curative instead of preventive broad spectrum pesticides is necessary. This requires a huge amount of biological knowledge of the epidemiology and population dynamics of pests and pathogens in relation to the level of inputs and cultural practices.

The level of fertilization is a main determinant of the cropping system. A sophisticated advice system by which the timing and the amount of fertilizers are adapted to the forecasted demand of the crop and a forecasted mineralization in the soil is urgently needed. Moreover, the use of slow release fertilizers, preferably organic, is advocated.

The resistance properties of cultivars determine disease intensity in any farming system. The choice of cultivars can strongly reduce growth rates of mildew and rusts, and those of stem base diseases, blotches, moulds, and ear blight, moderately. These features might not be available in one cultivar, but

monocultures of one cultivar are in contradiction with an integrated approach. Research on and use of resistance of different origin in time and in space and in cv. mixtures or multilines is therefore advocated (WOLFE 1985). Breeding for resistance is a prerequisite of an integrated system, in which relatively tall, lodging-resistant cultivars with strong weed competitive properties are preferable. In addition to conventional complete and partial resistance, hypothetically, a strategy of breeding for field resistance may be designed to widen the scope of disease prevention. This may be achieved by breeding for a higher final number of leaves per culm, without altering the heading date. In this way, the rate of leaf appearance and their rate of death both increase so that individual leaves are exposed to disease for a shorter time before heading, which could affect especially epidemics caused by pathogens that have long latent periods. Moreover, this would result in a crop with many small leaves per square metre, and since diseased leaves die earlier, it would allow accelerated leaf death to eliminate disease without losing too much of the green area index. This strategy, a partial escape resistance, is partly in opposition to the wheat ideotype designed by DONALD (1968), but in agreement with the comments made by SYME (1971, 1974).

In addition to this breeding strategy, research on and development of factors which can be used as preventive measurements is necessary, but up to now hampered by a lack of sound methods, as showed at the beginning of this discussion. Nevertheless, this knowledge and these methods to obtain them are necessary in order to construct modern systems of wheat production, that are durable, economically and environmentally sound, and adapted to the demands of the future.

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Samenvatting

Regulatie van meeldauw in wintertarwe

Tarwe wordt wereldwijd op een zeer groot areaal verbouwd. Qua produktie is tarwe het belangrijkste voedingsgewas voor de mens. In Nederland wordt voornamelijk wintertarwe, in vruchtwisseling met aardappelen en suikerbiet verbouwd. Het gewas kan worden aangetast door een algemeen voorkomende schimmelziekte, tarwemeeldauw (*Erysiphe graminis* f. sp. *tritici*, ook wel *Blumeria graminis* genoemd). Bij aantasting ontstaat schade, een verlies aan opbrengst. In 1978 kwamen chemische bestrijdingsmiddelen beschikbaar waarmee akkerbouwers tarwemeeldauw en de daardoor veroorzaakte schade effectief konden bestrijden. Door vermindering van schade werd een hogere opbrengst in kilogrammen verkregen en daarmee een hoger bedrag aan geld. Het gebruik van bestrijdingsmiddelen kost de akkerbouwer echter ook geld. Onduidelijk was, hoe groot de schade door tarwemeeldauw is ten opzichte van de kosten van chemische bestrijding. Anders gezegd: hoeveel meeldauw kan in een bepaald ontwikkelingsstadium van het gewas getolereerd worden voordat economische schade optreedt. Om na te gaan bij welke hoeveelheid meeldauw de schade gelijk is aan de kosten van chemische bestrijding, startten het Instituut voor Plantenziektenkundig Onderzoek en de Stichting Nederlands Graan-Centrum in 1979 onderzoek naar tarwemeeldauw. Deze gegevens waren nodig om een systeem van geleide bestrijding van tarwemeeldauw te kunnen invoeren. De samensteller van dit proefschrift kreeg als taak om dit onderzoek op te stellen en uit te voeren, naast de taak om het optreden van graanziekten in het land te inventariseren. Na 1981 kon de epidemiologie van tarwemeeldauw slechts oppervlakkig worden onderzocht, omdat het accent in het onderzoek werd verlegd van meeldauw naar kafjesbruin (*Leptosphaeria nodorum*).

Het onderzoek werd ontwikkeld in samenwerking met het EPIPRE-project, dat eerst door de Landbouwhogeschool en na 1981 door het Proefstation voor de Akkerbouw en Groenteteelt in de Vollegrond werd uitgevoerd. Het EPIPRE-project had als doel om een systeem voor de geleide bestrijding van gele roest (*Puccinia striiformis*) in wintertarwe in praktijk te brengen, en werd later uitgebouwd tot een systeem voor de geleide bestrijding van ziekten en bladluis in wintertarwe.

Tarweteelt en meeldauw. In hoofdstuk 2 wordt de tarweteelt en het optreden van meeldauw in de jaren 1974-86 beschreven. Wintertarwe werd in Nederland verbouwd op ruim 100.000 hectare, voornamelijk op zeekleigronden. Daarnaast werd tarwe verbouwd op rivierklei, löss, zand- en dalgronden. In 1974-86 steeg de nationale jaarlijkse produktie van 0.7 tot 1 miljoen ton, door een stijging van de opbrengst van 5 naar 8 ton tarwe per hectare. In 1974-86 werd circa een derdedeel van de nationale produktie gebruikt als voedsel, een derde als veevoer en een derde werd geëxporteerd. De nationale tarwe consumptie was 1 miljoen ton voedsel en 0.4 miljoen ton veevoer per jaar, zodat jaarlijks een netto import van 0,6 miljoen ton tarwe nodig was.

Wintertarwe wordt over het algemeen in de tweede helft van oktober gezaaid en in augustus geoogst. Rijenzaai is gebruikelijk. In 1974-86 nam de afstand tussen de rijen op de akker af van 25 cm tot 10-15 cm. De stikstofgift steeg in deze periode van gemiddeld 100 naar 160 kg N per hectare per jaar. Vanaf 1973 werden steeds meer chemische middelen gebruikt om onkruid, ziekten en bladluis te bestrijden. Tevens werd de toepassing van groei-regulatoren algemeen. De directe kosten van deze middelen, zonder kosten voor arbeid en machines, waren in 1973 te verwaarlozen, maar bedroegen in 1986 10% van de financiële bruto opbrengst van een tarwegewas.

Ziekten in tarwe kunnen worden voorkomen door resistente rassen te zaaien. In 1974-86 werd de grootte van het areaal van elk tarweras vooral bepaald

door de ziekte gele roest. Gele-roestresistente tarwerassen hadden over het algemeen een groot areaal. Tarwerassen met een goede meeldauwresistentie waren niet beschikbaar. De tarwerassen die redelijk meeldauwresistent waren, werden in de loop van 2-4 jaar steeds vatbaarder voor meeldauw. Dit is een bekend verschijnsel bij meeldauw; de meeldauwpopulatie verandert geleidelijk van genetische samenstelling. Een dergelijk effect trad ook op bij chemische bestrijdingsmiddelen. Door deze middelen een aantal jaren in de praktijk te gebruiken verandert de meeldauwpopulatie geleidelijk van genetische samenstelling, waardoor de werking tegen meeldauw afneemt.

Inventarisaties. Door verschillende onderzoekers werd in 1974-86 jaarlijks een 100-tal praktijkpercelen wintertarwe bemonsterd op ziekten en plagen.

In het eerste tot tweede knoopstadium van het gewas, meestal in mei, was gemiddeld 29% van de percelen aangetast door meeldauw. Van jaar tot jaar kwamen grote verschillen voor; slechts 1% van de percelen was aangetast in 1980, maar in 1977 was dit 61%. Het percentage aangetaste percelen was hoog in jaren met een hoge temperatuur in de maand oktober en in jaren met een hoge temperatuur in de maanden december, januari, februari en maart. In een warme oktobermaand wordt wintertarwe al vroeg geïnfecteerd door meeldauw. Daarnaast bleek het percentage aangetaste percelen hoog te zijn in jaren dat de verbouwde tarwerassen gemiddeld vatbaar voor meeldauw waren.

Tijdens de afrijping van het gewas, in juli 1974-86 was gemiddeld 70% van de percelen aangetast door meeldauw. Omdat elk jaar verschillende soorten en hoeveelheden chemische middelen werden gebruikt om ziekten te bestrijden, hield de aantasting van meeldauw in juli geen verband meer met de aantasting in mei, noch met het weer in de verschillende jaren. Akkerbouwers namen in 1981-84 waar, dat de gemiddelde hoeveelheid meeldauw vanaf begin mei tot half juni toenam, ondanks het gebruik van chemische bestrijdingsmiddelen. Vanaf half juni tot half juli was de gemiddelde hoeveelheid meeldauw tamelijk constant. Kleine verschillen in meeldauwresistentie tussen de tarwerassen bleken in het veld een groot effect te hebben op de ontwikkeling van meeldauw in mei, juni en juli. Tarwerassen, die op een klein areaal werden verbouwd, waren ook minder aangetast. Dit verschijnsel illustreert dat de genetische samenstelling van de meeldauwpopulatie een afspiegeling is van partiële resistenties van de verbouwde tarwerassen.

Epidemieën van meeldauw kunnen worden vertraagd door meer tarwerassen te zaaien die verschillen in herkomst van resistentie. Daarnaast moet tijdig de tarwestoppel worden bewerkt en wintertarwe niet vroeg maar na half oktober worden gezaaid. Dit zijn preventieve maatregelen, die niet altijd afdoende zijn. De hoeveelheid meeldauw varieerde enorm van perceel tot perceel. Zelfs tijdens een zware epidemie, zoals in het voorjaar van 1983, was meeldauw in de helft van de percelen afwezig. Dit benadrukt de noodzaak om af te zien van routinematige chemische bestrijdingen. Door de hoeveelheid meeldauw in het veld waar te nemen kan worden vastgesteld of preventieve maatregelen afdoende waren en of een chemische bestrijding moet worden overwogen.

Intensiteit van meeldauw. Het meest opvallende symptoom van meeldauw is de witte plukjes schimmelweefsel, zogenaamde puistjes, op het blad, de bladschede en later op de aar van de tarweplant. In de Planteziektenkunde is het gebruikelijk om de intensiteit van een ziekte uit te drukken in het percentage bladoppervlak dat ziek is. Dit geeft vaak verwarring, want welk bladoppervlak ziek is laat zich moeilijk schatten. Soms wordt alleen het oppervlak met schimmelweefsel geschat en soms het bladoppervlak dat niet meer groen is. In onderzoek naar en toepassing van een systeem om meeldauw niet routinematig te bestrijden moet de intensiteit van de ziekte eenduidig kunnen worden bepaald. In dit onderzoek werd het aantal puistjes meeldauw op tarwebladeren geteld. In hoofdstuk 3 wordt beschreven hoe nauwkeurig de

intensiteit van meeldauw dan wordt bepaald. Deze bleek afhankelijk te zijn van de gemiddelde intensiteit van tarwemeeldauw in een bladlaag van het gewas, en natuurlijk ook van het aantal bladeren waarop het aantal puistjes meeldauw werd geteld. Lage intensiteiten, minder dan gemiddeld één puistje meeldauw per blad, konden in veldproeven moeilijk exact worden bepaald.

Om de intensiteit van tarwemeeldauw in praktijkpercelen te bepalen is een snelle en eenvoudige methode wenselijk. Een eenvoudige methode is om van een plantmonster, een aantal planten uit een perceel, het aantal bladeren te tellen met meeldauw. Hoe dit percentage zieke bladeren samenhangt met het aantal puistjes meeldauw per blad, werd onderzocht in verschillende jaren en tarwerassen, in veldproeven en in praktijkvelden (hoofdstuk 4). Bij niet al te grote hoeveelheden meeldauw kan het aantal puistjes meeldauw per blad geschat worden uit het percentage bladeren met meeldauw. Deze schatting is echter minder nauwkeurig dan een directe telling van het aantal puistjes per blad. Een voordeel van telling van het aantal zieke bladeren is dat op dezelfde manier de intensiteit van andere ziekten, die vaak geen puistjes vormen, ook bepaald kan worden.

Schade door meeldauw. Dat meeldauw opbrengstderving, schade, kan veroorzaken was algemeen bekend. Onduidelijk was echter hoeveel schade wordt veroorzaakt door meeldauw, gedurende een bepaald ontwikkelingsstadium van het gewas. Dit is een gecompliceerd probleem omdat naast het weer ook teeltmaatregelen, zoals de rassenkeuze en de bemesting, de hoeveelheid meeldauw en de daardoor veroorzaakte schade bepalen. Het grootste deel van de onderzoekstijd werd besteed om een zogenaamde schadefunctie van meeldauw in wintertarwe vast te stellen. Indien de hoeveelheid meeldauw gedurende het seizoen bekend is, kan de daardoor veroorzaakte schade met zo'n schadefunctie worden geschat.

In hoofstuk 4 wordt een proef beschreven met vier tarwerassen, namelijk: Durin, Nautica, Arminda en de lijn SVP 7348-5-4. De rassen verschilden in genetische herkomst. Nagegaan werd of de schade veroorzaakt door meeldauw gerelateerd kon worden aan de hoeveelheid meeldauw in die rassen. De vier tarwerassen hadden een verschillend ziekte-verloop. Durin was al vroeg en zwaar aangetast, maar later in het seizoen licht. Nautica werd pas laat maar wel zwaar aangetast. Arminda en SVP 7348-5-4 waren de hele tijd tamelijk zwaar aangetast. Hierdoor was de schade door meeldauw per ras verschillend. De schade bleek echter recht evenredig te zijn met de hoeveelheid meeldauw èn met de tijdsduur van de aantasting. Een maat hiervoor is het totaal aantal puistdagen meeldauw per blad gedurende het seizoen. De parameter van deze schadefunctie was voor de tarwerassen gelijk, namelijk: een verlies van 12 gram tarwe per are per puistdag meeldauw per blad. De schade bleek dus direct afhankelijk te zijn van de meeldauwresistentie van tarwe. Het is aannemelijk dat dit ook voor andere praktijkrassen geldt.

Bovengenoemde problematiek werd ook onderzocht in een veldproef bij twee verschillende stikstofgiften (hoofdstuk 5). De ontwikkeling van meeldauw werd niet bevorderd bij de hoogste stikstofgift, wat gewoonlijk wel het geval is bij tarwemeeldauw. Er traden dan ook geen verschillen in schade op. Het is nog onduidelijk of de hoogte van de stikstofgift een effect heeft op de schadefunctie van tarwemeeldauw. De schadefunctie bedroeg in deze proef 13 gram opbrengstderving per are per puistdag meeldauw per blad.

Naast bovengenoemde veldproeven werden nog elf veldproeven uitgevoerd om de schade door meeldauw in de verschillende stadia van de gewasontwikkeling te meten (hoofdstuk 6). In drie proeven trad geen tarwemeeldauw op. Het bestrijdingsmiddel dat in deze proeven werd gebruikt, bij afwezigheid van meeldauw, toonde dan ook geen schade aan. Dit betekent dat het effect van dit middel op opbrengst veroorzaakt wordt door een bestrijding van meeldauw of eventuele andere ziekten. Uit een andere proef konden geen gegevens worden verkregen, hoewel de tarwe er wel werd aangetast door meeldauw.

Watergebrek in het droge voorjaar van 1980 bepaalde de opbrengsten in deze proef op zandgrond geheel, waardoor de schade door meeldauw daar niet kon worden gemeten. In de resterende zeven veldproeven werd de schade wel gemeten. In elke proef was de schade recht evenredig met het totaal aantal puistdagen meeldauw per blad, overeenkomstig de proef met de tarwerassen. De schadefunctie was een gemiddeld verlies van 13 gram tarwe per are per puistdag meeldauw per blad, gemeten vanaf het tweede knoopstadium van het gewas tot deegrijp, bij een opbrengstniveau van 70 tot 90 kg tarwe per are. De standaard-afwijking van de schadefuncties was 3 gram. De schadefuncties bleken onafhankelijk te zijn van de verschillende jaren waarin de proeven werden uitgevoerd, en het ontwikkelingsstadium van het gewas of het tijdstip in het seizoen waarin meeldauw tot ontwikkeling kwam. Alleen van zeer vroege aantastingen, voor het tweede knoopstadium, kon geen schade worden aangetoond. De grondsoorten waarop en de tarwerassen waarin de proeven werden uitgevoerd hadden ook geen systematisch effect op de schadefuncties. Ruim de helft van de gemeten schade werd veroorzaakt doordat meeldauw het aantal tarwekorrels per are verlaagde en iets minder dan de helft doordat de tarwekorrel kleiner was.

De plaats waar meeldauw zich in het bladerdek bevindt werd ook bepaald. De onderste, dus oudste bladeren van het gewas, hadden circa drie keer zoveel meeldauw dan gemiddeld. De bovenste bladeren waren het minst aangetast. Dit profiel van meeldauw in het gewas bleek in de verschillende veldproeven wel wat te variëren, maar dit beïnvloedde de schade niet aantoonbaar.

Advies-systeem. Op basis van de verkregen gegevens werd een advies-model voor geleide bestrijding van tarwemeeldauw opgesteld en na 1980 in de praktijk toegepast. In het model wordt het door de akkerbouwer waargenomen aantal bladeren met meeldauw omgerekend naar het gemiddelde aantal puistjes meeldauw per blad. Gebaseerd op een aantal vuistregels, die niet in dit proefschrift staan, wordt het verloop van meeldauw gedurende het seizoen geschat. Deze schatting is afhankelijk van de actuele hoeveelheid meeldauw, het tarweras, het ontwikkelingsstadium van het gewas, de grondsoort en de hoogte van de stikstofgift. De verwachte epidemie, in puistdagen meeldauw per blad, bepaalt met de schadefunctie de opbrengstderving die verwacht wordt. In navolging van het model voor gele roest, wordt de verwachte schade door meeldauw vergeleken met de verwachte kosten van het bestrijdingsmiddel, de rijspoorschade, en de vaste kosten waaronder arbeid, om te bepalen of een bestrijding bedrijfseconomisch gezien rendabel is. Doordat akkerbouwers hun percelen regelmatig op ziekte inspecteren, wordt tevens een overzicht verkregen van de actuele situatie van ziekten in wintertarwe in Nederland.

Fysiologie van schade. Onduidelijk is of de schadefunctie, vastgesteld in verschillende jaren met elk een specifieke weersgesteldheid, ook geldt voor andere jaren. Het is ook niet aan te geven of de schadefunctie gebruikt kan worden in landen met een ander klimaat. De schadefunctie zou in veldproeven geijkt kunnen worden in andere jaren of in landen met een ander klimaat. Dit is echter bewerkelijk en het leidt bovendien niet tot de ontwikkeling van een algemene methode om deze vraag op te lossen. Met simulatie-modellen, waarmee de opbrengst van tarwe wordt berekend uit de weersgegevens, temperatuur en lichtintensiteit, zou de schade door meeldauw in relatie tot het weer berekend kunnen worden. Het effect dat meeldauw op de fysiologie van tarwe heeft moest kwantitatief worden beschreven. De Landbouwhogeschool onderzocht het effect van meeldauw op de assimilatie van tarwebladeren. In hoofdstuk 7 en 8 wordt vervolgens onderzocht of de schadefunctie berekend kan worden met simulatie-modellen. Hiertoe werd meeldauw in een tarwe-model door vijf parameters gekarakteriseerd. De belangrijkste parameters waren die van de hoeveelheid meeldauw, de verdeling van meeldauw in het gewas en het

effect dat meeldauw heeft op de assimilatie bij een overvloed aan licht. De berekende schade kwam qua orde van grootte redelijk overeen met de in het veld gemeten schade. Vooral bij vroege epidemieën, voor de bloei, werd de gemeten schade echter onderschat. De fysiologie van tarwe is nog onvoldoende onderzocht om de schade die meeldauw onder verschillende weersomstandigheden veroorzaakt te kunnen simuleren. Voor de ontwikkeling van systemen voor geleide bestrijding van ziekten zijn veldproeven nog onmisbaar.

Beheersing van tarwemeeldauw. Met een systeem voor geleide bestrijding van graanziekten wordt het gebruik van bestrijdingsmiddelen gerationaliseerd. Chemische bestrijdingsmiddelen worden alleen dan gebruikt als het qua bedrijfseconomie verantwoord is. Resistente tarwerassen verliezen na een paar jaar hun effectiviteit om meeldauw te beheersen. Evenzo verliezen chemische bestrijdingsmiddelen hun effectiviteit. Er moeten dus steeds nieuwe tarwerassen worden veredeld om in de toekomst meeldauw te kunnen beheersen en om het gebruik van chemische middelen te verminderen. Tevens moet de kans op optreden van meeldauw en andere ziekten worden verkleind, door preventieve cultuurmaatregelen te nemen, zoals een stoppelbewerking en een late zaai. Door het Proefstation voor de Akkerbouw en de Groenteteelt in de Vollegrond wordt in het project 'Ontwikkeling Bedrijfs-Systemen' te Nagele, een dergelijk 'geïntegreerd' bedrijf ontwikkeld en bestudeerd in relatie tot een gangbaar en een biologisch-dynamisch akkerbouwbedrijf. Gedurende twee seizoenen werden epidemieën van ziekten en plagen in de tarwegewassen van deze drie bedrijven gemeten en geïnterpreteerd (hoofdstuk 9). De bemesting van deze drie teelt-systemen van tarwe bleek een belangrijke factor te zijn die de samenstelling, de structuur en de dichtheid van het gewas bepaalt. Meeldauw, gele roest en in mindere mate ook sneeuwschimmel (*Monographella nivalis*), bladmineerders en graanhaantjes (*Lema cyanella*) namen sterk toe bij hogere stikstofgiften. De bemesting had geen effect op de aantasting van oogvlekkenziekte (*Pseudocercosporella herpotrichoides*), *Fusarium*-voetziekte, bruine roest (*Puccinia recondita*), bladvlekkenziekte (*Mycosphaerella graminicola*) en bladluis (voornamelijk *Sitobion avenae*). De grootte van het effect dat de bemesting had op de ontwikkeling van meeldauw en gele roest was vergelijkbaar met het effect dat verschillende tarwerassen of chemische bestrijdingsmiddelen hebben op de ontwikkeling van deze ziekten. Door, naast bovengenoemde cultuurmaatregelen, tarwe te verbouwen bij een lager niveau van bemesting en door de stikstofgift beter af te stemmen op de behoefte van het gewas, kan het optreden van meeldauw en het gebruik van chemische bestrijdingsmiddelen drastisch worden beperkt.

Curriculum vitae

Richard Anton Daamen werd geboren op 1 januari 1953 te Vlissingen. In 1971 behaalde hij het diploma HBS-B aan de Rijksscholengemeenschap 'Scheldemond' te Vlissingen. In hetzelfde jaar begon hij met de studie biologie aan de Landbouwhogeschool te Wageningen, met speciale belangstelling voor de biologische bestrijding. In 1979 behaalde hij het ingenieursexamen Biologie met als vakken de Entomolgie, de Vegetatiekunde en Plantenoecologie en de Theoretische Teeltkunde. Ingenieursonderzoek is verricht bij de Deltadienst van Rijkswaterstaat te Middelburg en de 'Universidad Nacional Technical de Piura' te Peru.

Na een assistentschap voor de 'International Post-Graduate Course in Dynamic Simulation in Crop Protection' en een baan als aankomend programmeur, bij de Landbouwhogeschool, was hij van april 1979 tot 1984 in dienst bij de Stichting Nederlands Graan-Centrum, gedetacheerd op het Instituut voor Plantenziektenkundig Onderzoek, te Wageningen. In deze periode werd in samenwerking met anderen het optreden van ziekten en plagen in granen geïnventariseerd en een advies-model voor de geleide bestrijding van tarwemeeldauw en een voor kafjesbruin ontwikkeld en in praktijk gebracht. Vanaf januari 1984 is hij werkzaam op het Instituut voor Planteziektenkundig Onderzoek, waar hij naast het onderwerp van dit proefschrift vergelijkbaar onderzoek aan andere graanziekten verricht. In 1986 en 1987 is hij een aantal keren in opdracht van de FAO te Rome uitgezonden om de haalbaarheid van het door de Poolse regering opgestelde 'Pest and Disease Control Project' te evalueren.