IDENTIFICATION OF THE MAJOR CHARACTERISTICS OF POTATO CULTIVARS WHICH AFFECT YIELD LOSS CAUSED BY LATE BLIGHT

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hoogleraar in de produktie-ecologie
met bijzondere aandacht voor de plantaardige produktie

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Identification of the major characteristics of potato cultivars which affect yield loss caused by late blight

Proefschrift

ter verkrijging van de graad van doctor in de landbouw- en milieuwetenschappen
op gezag van de rector magnificus, dr. H.C. van der Plas, in het openbaar te verdedigen
op vrijdag 13 september 1991 des namiddags te vier uur in de aula van de
Landbouwuniversiteit te Wageningen

9. Het relateren van opbrengstderving door Coloradokevers aan het percentage ontbladering (Ferro et al., 1983) of aan parameters uit de klassieke groeianalyse (Dripps & Smilowitz, 1989) is achterhaald.

Dripps, J.E. & Smilowitz, Z., 1989. Growth analysis of potato plants damaged by Colorado potato beetle (*Coleoptera: Chrysomelidae*) at different plant growth stages. Environ. Entomol. 18: 854-867.
Ferro, D.N., Morzuch, B.J. & Margolies, D., 1983. Crop loss assessment of the Colorado potato beetle (*Coleoptera: Chrysomelidae*) on potatoes in Western Massachusetts. J. Econ. Entomol. 76: 349-356.

10. Ofschoon 'spurious correlations' al in de negentiende-eeuwse literatuur zijn beschreven (Pearson, 1897) wordt in de hedendaagse nematologie nog altijd foutief tot dichtheidsafhankelijke vermeerdering van aaltjes geconcludeerd bij een significante negatieve correlatie van aaltjesvermeerderingsfactoren met de dichtheid van de uitgangspopulatie (Ferris, 1985).

Ferris, H., 1985. Density-dependent nematode seasonal multiplication rates and overwinter survivorship: A critical point model. J. Nematol. 17: 93-100.
Pearson, K., 1897. On a form of spurious correlation which may arise when indices are used in the measurement of organs. Proc. Roy. Soc. (London) 60: 489-498.

11. De gewoonte om van een 'fysiologische leeftijd' van organismen te spreken, terwijl slechts morfologische criteria voor ontwikkelingsstadia worden gegeven, getuigt van gebrekkige kennis van de ontwikkelingsbiologie.

12. De efficiëntie van onderzoeksinstituten zal toenemen door projekten die uitsluitend bestaan uit studie van de literatuur in andere disciplines dan het reguliere instituutsonderzoek.

13. In de geschiedschrijving met betrekking tot de ontdekking van de oorzaak van de aardappelziekte is de rol van Dr. Van Oije, die al voor 1845 een schimmel verantwoordelijk voor de ziekte stelde, onderbelicht.

Hecke, L., 1898. Untersuchungen über *Phytophthora infestans* De By, als Ursache der Kartoffelkrankheit. Journal für Landw. 46: 97-142.

Stellingen behorend bij het proefschrift van Marcel van Oijen: 'Identification of the major characteristics of potato cultivars which affect yield loss caused by late blight'.

Wageningen, 13 september 1991.

ter herinnering aan Kees Spitters

VOORWOORD

Het onderzoek dat in dit proefschrift beschreven wordt, is uitgevoerd op de Stichting voor Plantenveredeling (SVP, nu onderdeel van het CPRO) te Wageningen, in de periode 1986-1990. Drie personen zijn hierbij van bijzonder belang geweest: Dirk Budding, Kees Spitters en Rudy Rabbinge. Dirk Budding gaf talloze adviezen over experimenten met *Phytophthora* en nam een belangrijk deel van de uitvoering voor zijn rekening. Kees Spitters gaf wetenschappelijke begeleiding aan het onderzoek, met name wat betreft de opzet van veldproeven en het analyseren van resultaten uit experimenten en modelwerk. Hij zou als co-promotor optreden en van het proefschrift vooral de veredelingsaspekten bewaken. De meeste hoofdstukken in het proefschrift zijn kritisch door hem bekeken, maar zijn overlijden in 1990 heeft het co-promotorschap verhinderd. Rudy Rabbinge, mijn promotor, nam daarna de volledige begeleiding op zich. Zijn hulp heeft het schrijven plezieriger gemaakt en het geschrevene aanzienlijk leesbaarder.

Naast de drie genoemden hebben nog zeer velen hulp geboden. Erik Toussaint heeft me wegwijs gemaakt op de SVP, hetgeen de inwerkperiode aangenaam en kort maakte. Janine van Heesen, Peter van Leeuwen (CABO) en vele medewerkers van de proefveldendienst en andere SVP-afdelingen assisteerden bij een of meerdere proeven. Paul Keizer en Jack Groot katalyseerden de statistische gegevensverwerking en het computerwerk. Teksten zijn van inhoudelijke kritiek voorzien door Leontine Colon, Anton Haverkort (CABO), Lidwine Dellaert, Lo Turkensteen (IPO) en Walter Rossing (LUW). Adviezen met betrekking tot taalgebruik en vormgeving van het proefschrift zijn gegeven door Robert Hall, Jane Sykes, Margaret en Roger Wastie en Gon van Laar.

Ik dank mijn ouders voor het stimuleren van interesses buiten het onderzoek, en Netty van Dijk voor het helpen vinden daarvan.

LIST OF SYMBOLS

DMFR	daily multiplication factor	(d^{-1})
ε	initial light use efficiency of leaf photosynthesis	(g MJ^{-1})
E	latent fraction of the total leaf area	(-)
I	infectious fraction of the total leaf area	(-)
IE	infection efficiency	(%)
IP	infectious period	(d)
k	infection rate	(d^{-1})
l	leaf lesion coverage	(%)
LAD	leaf area duration	(m^2 d)
LAI	leaf area index	(-)
LG	lesion growth rate	(m d^{-1})
LP	latent period	(d)
LS	lesion size	(m^2)
LUE	light use efficiency	(g MJ^{-1})
PARCUM	cumulative light interception	(MJ m^{-2})
P_m	net photosynthetic rate at light saturation	(g m^{-2} h^{-1})
R	fraction of the total leaf area no longer susceptible or infectious	(-)
r_{app}	logistic rate of disease increase	(d^{-1})
R_d	dark respiration	(g m^{-2} h^{-1})
r_l	logistic rate of leaf lesion coverage	(d^{-1})
r_s	logistic rate of leaf senescence	(d^{-1})
S	susceptible fraction of the total leaf area	(-)
s	leaf senescence	(%)
SI	sporulation intensity	(m^{-2} d^{-1})
SLA	specific leaf area	(m^2 g^{-1})
SLW	specific leaf weight	(g m^{-2})
$t50_l$	inflection point of leaf lesion coverage	(d)
$t50_s$	inflection point of leaf senescence	(d)
$t50_y$	time when 50% of the total leaf area is diseased	(d)

CONTENTS

ACCOUNT

Parts of this thesis have been included, in part or in whole, in the following publications:

Chapter 1: Oijen, M. van, 1991. Light use efficiencies of potato cultivars with late blight (*Phytophthora infestans*). Potato Research 34: 123-132.

Chapter 2: Oijen, M. van, 1990. Photosynthesis is not impaired in healthy tissue of blighted potato plants. Netherlands Journal of Plant Pathology 96: 55-63.

Chapter 3: Oijen, M. van, 1991. Leaf area dynamics of potato cultivars infected to various extent by *Phytophthora infestans*. Netherlands Journal of Plant Pathology, accepted.

Chapter 4: Oijen, M. van, 1991. Components of resistance to *Phytophthora infestans* in potato: a review of the literature. Submitted to the Netherlands Journal of Plant Pathology.

Chapter 6: Oijen, M. van, 1991. Evaluation of breeding strategies for resistance and tolerance to late blight in potato by means of simulation modelling. Netherlands Journal of Plant Pathology, accepted.

Chapter 7: Oijen, M. van, 1991. Evaluating components of resistance to *Phytophthora infestans* in potato, using mathematical models of general epidemics. Netherlands Journal of Plant Pathology, accepted.

GENERAL INTRODUCTION

Potato late blight, a disease of potato foliage and tubers caused by the fungus *Phytophthora infestans* (Mont.) de Bary, has been an important research object since the major late blight epidemics in the middle of the last century. This research has been carried out in different disciplines, focussing on different aspects of the pathosystem.

Epidemiologal studies have focussed on the pathogen. The life cycle of the fungus has been studied, as well as the temporal and spatial development of blight epidemics for various genotypes of pathogen and host, and different environmental conditions (Lapwood, 1971). Epidemiology has considered the dynamics of the pathogen population, but generally the dynamics of the host (i.e. crop growth) have received less attention.

Resistance breeding research has focussed on the host. Resistance is the ability of the host to hinder the growth and/or development of the pathogen (Parlevliet, 1979). At first the aim of breeding research has been the identification of completely resistant host genotypes, but gradually partial resistance has been more strongly emphasized. The main goals in resistance breeding research now are the identification of the plant resistance characteristics that best allow screening of large numbers of genotypes, the determination of the mode of inheritance of these characteristics and the assessment of genetic variation for the characteristics among cultivated and wild genotypes (Parlevliet, 1979). Resistance breeding has not considered the dynamics of the pathogen population or host growth. The need for easy screening has led to the preponderance of tests in which the blight severity of genotypes is scored, without accounting for differences in host growth which may obscure differences in pathogen population increase.

Yield loss studies have related yield loss to disease severity by means of statistical relationships (e.g. James et al., 1972). Three linear regression methods have been used commonly, using one measurement of disease severity to explain yield loss (single point models), several measurements (multiple point models) or measurements and interpolated severity values at each instant during the epidemic, but integrated to give the area under the disease progress curve (*AUDPC*) (Rabbinge, 1988). As in breeding research, the dynamics of the disease are ignored in the single point models and the *AUDPC* method. All three methods further ignore that yield is the cumulative result of crop growth, even in pathosystems, which causes yield and yield loss to be significantly more closely related to measures of host growth, such as leaf area duration, than to disease severity at one or more times during the growing season (Rotem, Bashi and

Kranz, 1983).

The biochemical and physiological mechanisms underlying resistance have been studied in other disciplines (Clarke, 1983; Keen & Yoshikawa, 1983), but only rarely have attempts been made to relate the results to production in the field.

Problem definition and research goal

The different disciplines mentioned above have all studied specific aspects of potato late blight. However, studies that integrate the results from the different disciplines are missing. The host plant and the pathogen mutually affect each other and should be studied as a system (Zadoks, 1977), but many aspects of this interaction have been ignored. The relationship between resistance and maturity class has been indicated but not clarified. Tolerance, defined as the ability to endure the presence of the pathogen with reduced disease symptoms and/or damage (Parleviiet, 1979), has been neglected. The present study is an attempt to show how breeding research can benefit from epidemiology, yield loss studies and physiology when genotypic differences in resistance, maturity class and tolerance are analysed.

The dynamical approach of epidemiology, the measurement of host characteristics of crop and plant physiology and the assessment of genetic variation of breeding research are integrated in the present study, in an attempt to explain the effect of blight on tuber production of potato cultivars. However, the primary motivation for the study originates from breeding research. Differences between cultivars thus are emphasized more than differences between pathogen populations and environmental conditions. The goal of the present study therefore is to answer the following question: "What plant characteristics mainly explain differences between cultivars in yield loss caused by *P. infestans*?".

The attempt to combine different research approaches into one study of crop production, makes the present study an example of production ecological research (Rabbinge, 1986).

Research methodology and thesis outline

Genotypic differences in yield loss are caused by those plant characteristics for which both significant genetic variation and a major influence on loss can be demonstrated. The genetic variation can be quantified by experiments in which cultivars are compared. The influence of a particular characteristic on loss, on the other hand, is difficult to quantify experimentally because cultivars generally differ for many characteristics

simultaneously. Therefore, for this purpose simulation modelling may be preferable to experimentation (Zadoks, 1977). In the present study, experiments and literature data were used to quantify genetic variation for resistance, tolerance and host growth characteristics. Characteristics that show genetic variation were examined in sensitivity analyses of simulation models to identify those that affected yield loss most.

First experiments were done to assess whether the pathogen caused yield loss mainly by decreasing the amount of functional leaf area of its host, or by reducing the activity of the leaf area (Chapter 1). These and other experiments revealed no genetic variation for effects of the disease on the photosynthetic activity of green leaves (Chapter 2). Therefore only the processes that determine the decrease of the amount of green leaf area were investigated more closely in further experiments. In these experiments, leaf senescence was also shown to be equally affected in all examined cultivars (Chapter 3). This left genotypic differences in host growth and in resistance as the major genetically determined characteristics affecting loss. Resistance was further analysed by quantifying genotypic differences for the various stages in the life cycle of the pathogen, the so-called 'resistance components'. Data about the resistance components were collected from the literature (Chapter 4). This literature review concludes the gathering of data about genetic variation in the present study.

The consequences for yield loss of the genetic variation of the various host characteristics, reported in the first four chapters, were examined by means of simulation models. First, different types of epidemiological models from the literature were compared to assess the requirements for a model to be used in quantifying the effects of components of resistance to late blight (Chapter 5). Then a model fulfilling these requirements was constructed, combined with a host growth model, and used to assess the effects on yield loss of the characteristics for which genetic variation had been demonstrated (Chapter 6). The properties of the new epidemiological model were compared to those of four other models, to evaluate the use of simple epidemiological models as a vehicle for resistance breeding (Chapter 7). The possibilities are demonstrated of using models to test hypotheses about the mechanisms underlying genotypic differences in rate of vertical spread of late blight through potato canopies (Chapter 8).

The study is concluded with a general discussion of the validity of the conclusions, the need for further research and the applicability of the described methodology in other pathosystems.

CHAPTER 1

Light use efficiencies of potato cultivars with late blight (*Phytophthora infestans*)

Abstract

Potato cultivars of different maturity classes and levels of resistance to *Phytophthora infestans* were grown under several disease intensities in three field trials. Seasonal courses of ground cover by green foliage and final tuber yields were determined. Light use efficiencies (*LUE*) were calculated from regression analyses of yield on cumulative light interception.

Late blight reduced tuber yields by decreasing cumulative light interception without affecting *LUE*. No differences in *LUE* between cultivars or cultivar classes were detected. Therefore, the maintenance of green leaf area is important when breeding potatoes for optimal performance in the presence of late blight.

The results support the hypothesis that the correlation between lateness and reported resistance of potato cultivars is due to the vigorous foliage growth of late cultivars.

Introduction

In early attempts to analyse the effect of *Phytophthora infestans* (Mont.) de Bary on potato yields, regression analyses were used to relate yield or yield loss to the fraction of diseased leaf area observed at one or more times in the growing season (James et al., 1972). Later, attention shifted from the diseased leaf area to the remaining green leaf area, the latter being more closely related to crop growth. Rotem, Bashi and Kranz (1983) showed that, for various levels of disease, yield was linearly related to the green leaf area averaged over the growing season.

Growth is approximately linearly related to intercepted light in many crops (Monteith, 1977). Haverkort and Bicamumpaka (1986) used this relationship to improve on the analysis of Rotem, Bashi and Kranz (1983). For tropical highland potato crops in Rwanda, they determined the cumulative light interception by green foliage, instead of leaf area alone. The yields of potato crops of different cultivars and severities of late blight could largely be explained by differences in cumulative light interception by the

green leaf area. The light use efficiency (*LUE*), defined as the slope of the linear regression of tuber yield on cumulative intercepted photosynthetically active radiation, was not significantly changed by late blight. Waggoner and Berger (1987) re-analysed the data of Rotem, Bashi and Kranz (1983) and also concluded that *LUE* of potato crops was unaffected by late blight.

Assessing *LUE* is a valuable first step in the detection of cultivars that are partially resistant or tolerant to a fungal leaf disease. Partial resistance is characterized by a relatively slow development of the pathogen, while tolerance is characterized by the maintenance of production capacity at given levels of disease. If *LUE* is not affected, differences in yield loss between cultivars of similar maturity class could be due to differences in the spread of the pathogen over the foliage, i.e. variation in partial resistance. Alternatively, they may be due to differences in acceleration of leaf senescence, indicating varying levels of tolerance. If *LUE* is affected in some of the cultivars, a variation in tolerance is also indicated. The effect of disease on the photosynthesis of green leaves may then differ between cultivars. This difference could be exploited when breeding for tolerant varieties, but measuring photosynthesis does complicate the selection process.

This chapter reports the results of three field experiments, carried out at two locations in the Netherlands in 1987 and 1988, with several potato cultivars of different maturity classes and levels of partial resistance. The experiments were carried out to test the hypothesis that *P. infestans* does not affect *LUE* in a potato crop.

Table 1.1. Details of the field trials carried out in the Netherlands.

	Experiment 1	Experiment 2	Experiment 3
Location	Wageningen	Wageningen	Renkum
Year	1987	1988	1988
Date of planting	April 29	June 1	April 20
Date of inoculation	June 23	July 27	June 28
Experimental design	Completely randomized	Randomized block	Split plot
Number of replicates	4	4	4
Number of cultivars	3	3	20
Number of treatments	3	4	2
Number of harvests	1	3	1
Distance between plots [1]	9-14 m	8-9 m	0 (4.5-8) m
Size of plots	15 m^2	35 m^2	6 (170) m^2
Isolation crop	sugar beet	hemp	rye

[1] Data for the main plots in the split plot experiment (Exp. 3) are given between brackets.

Materials and Methods

Three field experiments were carried out in two years at two locations with sandy soils (Table 1.1). Planting density was 4 tubers m^{-2} in all experiments. Plots were surrounded by unharvested border rows and separated from each other by isolation crops to minimize interplot interference.

The three cultivars used in Experiments 1 and 2 were Bintje, Surprise, and Pimpernel. Experiment 2 originally also included cv. Elkana, but the cultivar was discarded because of the poor quality of the seed tubers, which resulted in only 71% emergence. The cultivars used in Experiment 3 represented two maturity classes and two levels of partial resistance to *P. infestans*. To avoid effects of hypersensitivity, only cultivars without R-genes were used. Cultivars are classified as early maturing if their maturity index in the Dutch Variety List (Anonymous, 1988; scaling from 1 to 9) is higher than 5, otherwise they are classified as late. Cultivars are considered resistant if their level of resistance to *P. infestans* in the foliage is indexed higher than 5 in the variety list, otherwise they are classified as susceptible. The cultivars used were the early, susceptible cvs Bintje, Alcmaria, Cleopatra, Climax, and Sirtema; the early, resistant cvs Surprise, Apollonia, Désirée, Meerlander, and Spunta; the late, susceptible cvs Elkana, Darwina, Mondial, Promesse, and Senator; and the late, resistant cvs Pimpernel, Alpha, Irene, Karnico, and Kardal.

Different levels of disease were obtained by applying inoculum or contact fungicide to the plots, including the border plants, in different spraying frequencies. Inoculum consisted of a suspension of *P. infestans* (race 1,2,3,4,5,7,10,11; 2.5 ml per plant; 20 000 sporangia ml^{-1}), the fungicide was maneb/fentin acetate (Maneb-Tin TS, Luxan, Elst, 34%/11% a.i., 2.25 kg in 400 l water ha^{-1}), which is not known to affect yield (J.A.J. Kardolus, pers. comm. 1990). The experiments had two treatments in common: a treatment with artificial inoculation about 2 months after planting (inoculated), and a treatment without inoculation in which fungicide was applied weekly until the foliage died (control). There was an extra treatment in Experiments 1 and 2 in which inoculum was not applied and fungicide application was stopped at the time of artificial inoculation of the 'inoculated' plots (unsprayed-A). In Experiment 2, a fourth treatment was included in which fungicide application was stopped three weeks after inoculation (unsprayed-B). Low temperatures on the night of 21 May 1988 caused frost damage in Experiment 3. Leaf browning varied from about 15 to 50%, but no plants died.

For each individual plot, the percentage ground cover by green foliage was estimated visually at weekly intervals, resulting in minimal disturbance of the plots. Ground cover of potato crops is more closely related to light interception than leaf area (Burstall and

Harris, 1983). This has been disputed recently by Firman and Allen (1989), but the experiments on which they based their criticism were considered methodologically unsound by Haverkort et al. (1991). Data of incident solar radiation were obtained from a weather station located at 2.5 km from the trial fields in Wageningen, and 6 km from those in Renkum (Table 1.1). Incident photosynthetically active radiation was assumed to be 50% of the solar irradiation. Tuber yields were determined after death of the foliage. Experiment 2 included two earlier harvests, of three and nine plants per plot, on July 26 and August 30.

Cumulative light interception over the growing season was calculated for every plot, using linear interpolation of the weekly ground cover data, and assuming the percentage of light interception to be equal to the percentage ground cover. This assumption may lead to a small overestimation of light interception (Burstall and Harris, 1983), but differences between experimental treatments are likely to be systematic.

Tuber yields were first analysed with an analysis of variance to test cultivar and treatment effects. Then yields were related to cumulative light interception by linear regression analysis to test the significance of differences between cultivars and treatments in light use efficiency.

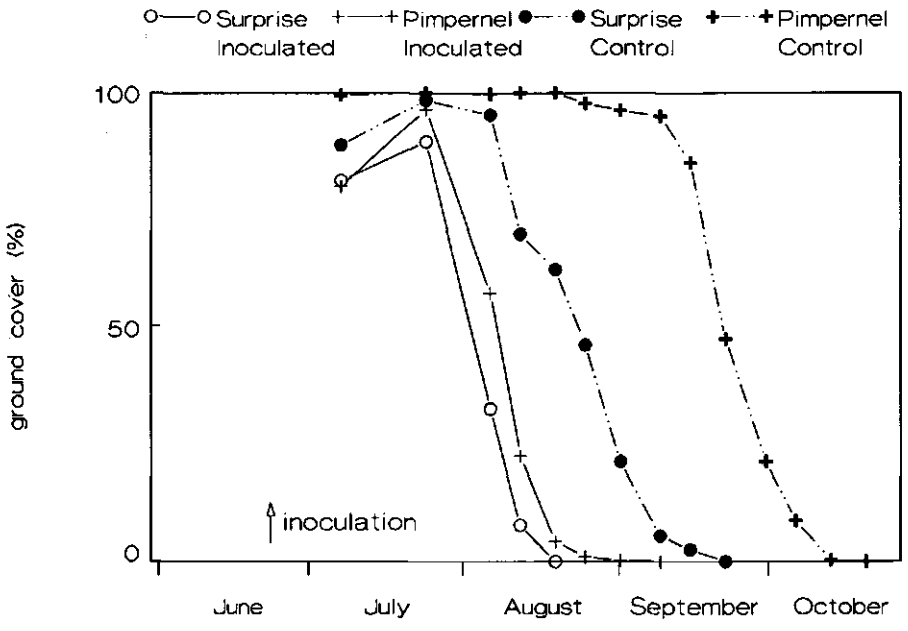


Fig. 1.1. Time course of ground cover in control and inoculated plots of cultivars Surprise and Pimpernel in Experiment 1.

Results

The growth patterns of early and late cultivars were different, as shown by the seasonal course of ground cover for inoculated plots and control plots of the early cv. Surprise and the late cv. Pimpernel in Experiment 1 (Fig. 1.1). Early cultivars had a shorter duration of ground cover. Late blight shortened this duration in both maturity classes.

Treatment effects on ground cover are shown in Figure 1.2. Disease caused the decline in ground cover to start earlier, but did not increase the rate of decline. Treatment effects were most pronounced in Experiment 1 and 2, where the reduction in ground cover started five weeks earlier in the inoculated plants than in the fungicide-protected controls (Fig. 1.2A, B). The difference between inoculated and control plants was less than three weeks in Experiment 3 (Fig. 1.2C). Full ground cover was not reached in any of the treatments in Experiment 3, probably because of the frost damage to the foliage on May 21.

Tuber dry matter yields are presented in Table 1.2. Infection by *P. infestans* led to yield losses in the inoculated and unsprayed plots. In Experiments 1 and 2, the percentage yield loss was always lowest in cv. Surprise, usually followed by cv. Pimpernel. In Experiment 3, the percentage yield loss of the cultivars was slightly

Table 1.2. Yield of tuber dry matter. Percentages of yield loss are indicated between brackets. Least significant differences (L.S.D. 0.05) for pairwise comparisons of cultivar means within treatment levels are: 0.76 t ha^{-1} (Experiment 1), 1.50 t ha^{-1} (Experiment 2) and 0.88 t ha^{-1} (Experiment 3).

Experiment	Cultivar (group)	Tuber yield (t ha^{-1})			
		Inoculated	Unsprayed-A	Unsprayed-B	Control
1	Bintje	2.87 (76%)	5.92 (50%)		11.82
	Surprise	5.32 (44%)	7.81 (18%)		9.48
	Pimpernel	4.72 (66%)	8.43 (40%)		13.97
2	Bintje	1.95 (75%)	5.68 (27%)	5.58 (28%)	7.79
	Surprise	4.19 (46%)	6.36 (18%)	7.39 (5%)	7.74
	Pimpernel	3.16 (64%)	5.97 (31%)	7.09 (18%)	8.66
3	Early/Susceptible	3.07 (23%)			4.00
	Early/Resistant	3.05 (25%)			4.06
	Late /Resistant	3.91 (43%)			6.81
	Late /Susceptible	4.24 (35%)			6.56

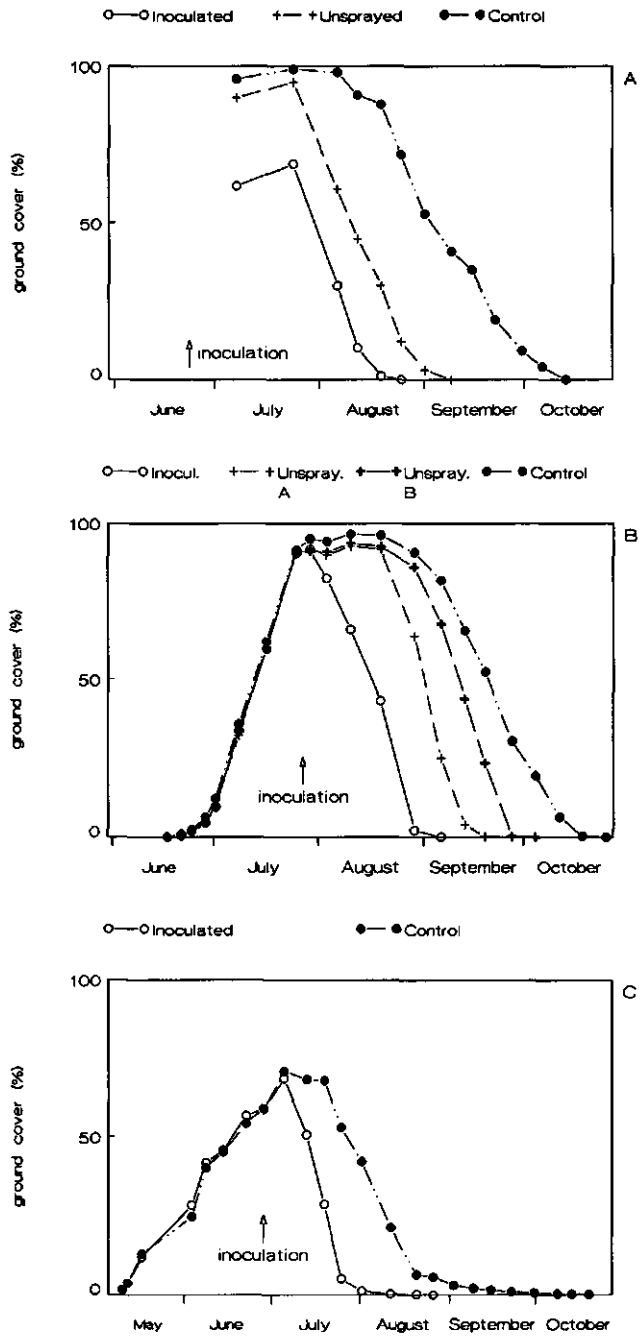


Fig. 1.2. Time course of ground cover with different treatments. Points refer to means over cultivars within treatments. A: Experiment 1; B: Experiment 2; C: Experiment 3.

correlated with the cultivar maturity index (% yield loss = 50.8 - 3.66 x index; r = -0.54, P < 0.05), indicating higher losses in the late cultivars. The percentage yield loss was not significantly correlated with the resistance index.

Figure 1.3 shows the regression lines for tuber yields on cumulative light interception for the three experiments. Haverkort and Harris (1986) showed that regression lines for yield on cumulative light interception differed systematically between cultivars of different maturity classes, with late cultivars intersecting the x-axis at higher values because of later tuber initiation. In Figure 1.3, therefore, regression lines are shown per cultivar or per group of cultivars of similar maturity class. In Experiment 2 there were three harvest dates, thus allowing comparison of regression lines between treatments per cultivar (Fig. 1.3A). These lines did not differ significantly for any of the cultivars (P > 0.05), indicating that disease did not affect the efficiency of light use. Because of different intercepts, the cultivar regression lines did differ significantly (P < 0.01), although their slopes did not (P > 0.05; Fig. 1.3B). Thus in Experiment 2 treatment or cultivar effects did not cause significant deviations from a common *LUE* of 2.06 g dry matter MJ^{-1}, but tuber initiation was later in the late cultivar Pimpernel than in the early cultivars. The same was found in Experiment 1 (*LUE* = 3.17 g MJ^{-1}; Fig. 1.3C). There was a similar lack of effect of disease in the larger group of genotypes of Experiment 3 (*LUE* = 1.81 g MJ^{-1}; Fig. 1.3D).

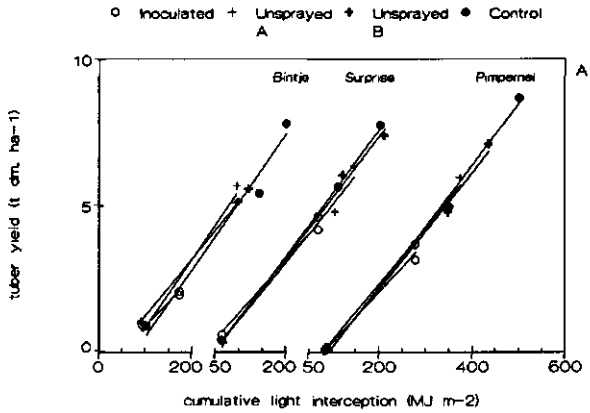
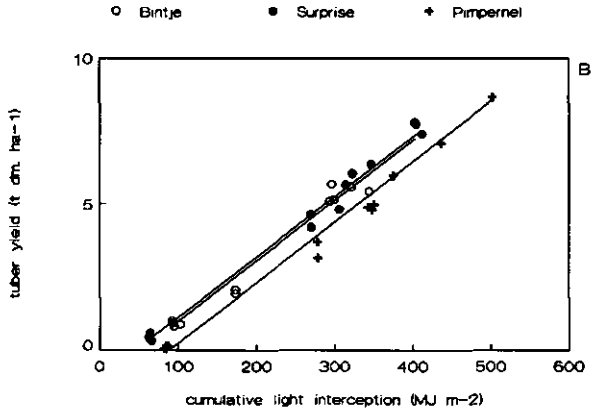
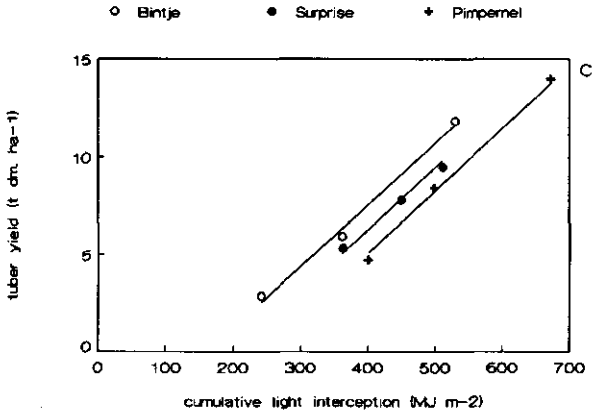
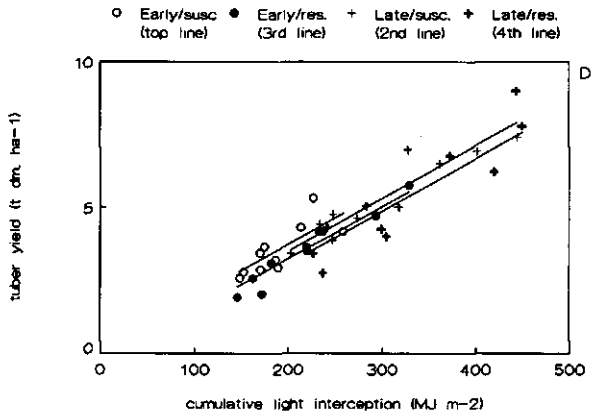


Fig. 1.3. Relationships between yield of tuber dry matter and cumulative light interception of cultivars or cultivar groups. Points refer to averages over replicates; regression lines are based on individual measurements. A: Experiment 2, four treatments per cultivar, at three harvest dates; regressions for each combination of cultivar and treatment (note shifted x-axes); B: Experiment 2, four treatments per cultivar, at three harvest dates; regressions for each cultivar; C: Experiment 1, three treatments per cultivar, at final harvest; regressions for each cultivar; D: Experiment 3, two treatments and five cultivars per cultivar group, at final harvest; regressions for each cultivar group. ↑→


Bintje
Surprise
Pimpernel
B
tuber yield (t dm. ha-1)
cumulative light interception (MJ m-2)
0
5
10
0
100
200
300
400
500
600


Bintje
Surprise
Pimpernel
C
tuber yield (t dm. ha-1)
cumulative light interception (MJ m-2)
0
5
10
15
0
100
200
300
400
500
600
700


Early/susc (top line)
Early/res. (3rd line)
Late/susc. (2nd line)
Late/res. (4th line)
D
tuber yield (t dm. ha-1)
cumulative light interception (MJ m-2)
0
5
10
0
100
200
300
400
500

Discussion

Within experiments, the *LUE* was similar for different cultivars and was not affected by different levels of disease caused by *P. infestans*. Thus, a genetic variation in tolerance, with respect to maintaining a high *LUE*, was not detected. This confirms the findings of Haverkort and Bicamumpaka (1986). Apparently differences among cultivars for loss in yield (Table 1.2) are mainly due to differences in maintenance of the green leaf area in the presence of disease. Measurements of photosynthesis in green leaves of healthy and diseased plants have confirmed the absence of disease effects (Chapter 2). Therefore, the selection of cultivars with high yields in the presence of late blight could be based on the duration of green leaf area. This is determined by the levels of partial resistance and disease-stimulated senescence.

LUE did vary between experiments: *LUE* was higher in the experiment conducted in 1987 (Experiment 1) than in the experiments of 1988. The weather conditions were, however, similar in the two years. The late planting in Experiment 2, with accompanying changes in growing conditions, and the frost damage in Experiment 3 may have contributed to lower values of *LUE* in the 1988 experiments.

It has often been reported that late cultivars show higher partial resistance to *P. infestans* than early cultivars (Umaerus et al., 1983). Such reports might arise from the erroneous equation of the percentage of foliage disease to lack of partial resistance, ignoring differences in foliage growth. A low, partial resistance should be measurable as a faster spread of the pathogen. However, irrespective of the maturity class of the host, late blight generally leads to an earlier onset of the decline in ground cover, rather than to an acceleration of this decline (Figs 1.1 and 1.2). Thus, the rate of pathogen spread appeared to be similar in early and late cultivars. Late cultivars have a longer period of foliage development, during which more leaves are formed. The fungus first mainly infects the lower leaves and then spreads to the top of the canopy (Chapter 3; Björling and Sellgren, 1955; Lapwood, 1961c, 1963). Therefore, if the levels of resistance of a late and an early cultivar are similar, the fungus will need more time to spread from the lowest leaf layers to the top in the densely foliated later cultivar. Only when a few green leaf layers remain, will further disease spread start to reduce ground cover. This explains why ground cover was reduced earlier in the early cultivars than in the late ones (Fig. 1.1). This explanation should be examined further in comparative studies of leaf formation, natural and disease-stimulated senescence, and the spread of late blight in early and late cultivars. Such studies may also clarify whether the yield differences between inoculated plants of the early cvs Bintje and Surprise (Table 1.2) are due to different levels of resistance or different levels of disease-stimulated senescence.

CHAPTER 2

Photosynthesis is not impaired in healthy tissue of blighted potato plants

Abstract

The net photosynthetic rates of green leaf tissue of potato plants of different cultivars were measured in the field and in a controlled environment after infection of the plants by *Phytophthora infestans*.

Infection had no significant effect on the net photosynthetic rate at light saturation, the efficiency of light use at low light intensities, or dark respiration. The reported effect of *P. infestans* on tuber yield seems to be caused solely by a reduction in the green leaf area. Therefore, a high rate of photosynthesis in green leaf tissue of infected plants is not a good selection criterion for potato genotypes.

Introduction

The loss in tuber yield of potatoes infected by late blight, caused by *Phytophthora infestans* (Mont.) de Bary, varies with the host cultivar and the growing environment. Haverkort and Bicamumpaka (1986), and Waggoner and Berger (1987) have recently shown that these differences in yield loss can largely be explained by differences in cumulative light interception by green leaf tissue. There does not seem to be an effect on the light use efficiency (*LUE*: the ratio of tuber yield and cumulative light interception; Chapter 1). Infection by the fungus reduces the green leaf area, by lesion growth and by stimulation of chlorosis and necrosis, but the photosynthetic activity of the remaining green leaf tissue is apparently not impaired. However, the constancy of the *LUE* does not give conclusive evidence for this hypothesis, because it is a rough measure of crop productivity as it includes seasonal variations in light interception, leaf photosynthesis, respiration and assimilate partitioning. Only direct measurements of photosynthetic rates in healthy and blighted plants can show whether the photosynthetic activity of the green leaf area is affected by late blight. Measurements of photosynthetic rates could be used to screen for host genotypic differences and thus help in the selection of blight tolerant genotypes for breeding purposes.

There are an increasing number of reports on the direct measurement of

photosynthetic rates in diseased plants. Farrar and Lewis (1987) gave examples of both positive and negative systemic effects of fungal infection on leaf photosynthetic rates. The effect of a fungus on host photosynthesis depends on the pathosystem in question. Scharen and Krupinsky (1969), and Berghaus and Reisener (1985) reported that variability between host genotypes may also exist. They found that photosynthetic rates were reduced to different extents after infection of wheat cultivars by *Septoria nodorum* and *Puccinia graminis*, respectively. So far, no reports on the effect of *P. infestans* on the rates of photosynthesis in potatoes have been published.

We measured, under controlled conditions, the rate of photosynthesis at light saturation (P_m), the light use efficiency at low light intensities (ε) and the dark respiration (R_d) of green leaves of healthy and partly blighted potato plants of two cultivars. We also measured, in the field, the P_m of three different potato cultivars infected to various degrees by blight.

Materials and Methods

Plants grown outdoors in pots were inoculated in a greenhouse five weeks after planting, and were placed in a climate chamber two weeks later to measure photosynthesis-light response curves. In a second experiment, the inoculation and measurement of light saturated rates of photosynthesis were carried out in the field.

Pot experiment. Individual seed tubers of the mid-early potato cultivar Bintje and the mid-late cultivar Surprise were planted in pots containing 10 l. of peat soil on July 20, 1988. Stems emerged during the first week after planting, and these were trimmed to one per plant within 14 days. For both cultivars 35 pots were placed in the open air and thus subjected to natural weather conditions. All pots were sprayed weekly until 30 days after planting with the mild contact fungicide chlorothalonil to prevent late blight infection while minimizing phytotoxic or other effects on the plants. The plants were transferred into the greenhouse 35 days after planting. Inoculum was prepared by making a suspension of sporangia washed off leaves of cv. Bintje plants, inoculated one month before with the complex *P. infestans* race 1,2,3,4,5,7,10,11. Test plants were inoculated 36 days after planting by spraying inoculum (146 000 sporangia ml^{-1}; about half the sporangia had germinated and released zoospores) over the lower two-third of leaf layers of the plants. Test and control plants were then capped with plastic bags to increase the humidity around the leaves. This procedure was repeated the next day with a suspension of 43 000 sporangia ml^{-1}.

Photosynthesis-light response curves were determined for eight replicates 47 to 50

days after planting. The measurement scheme followed a randomized block design with concurrent measurements of blocks. For this purpose every morning and afternoon four plants were transferred into a climate chamber (20 °C) at the Centre for Agrobiological Research (CABO) in Wageningen. The number of leaves were counted and disease severities were estimated, with the naked eye, for each separate leaf and stem internode. The plants had formed 17 to 18 leaves with distal leaflets longer than 5 cm. CO_2-exchange was measured using leaf 14 or 15 counting from the soil level, i.e. on relatively young, un-inoculated leaves (Louwerse and van Oorschot, 1969). The light intensity was reduced stepwise from about 280 W m^{-2} photosynthetically active radiation (400-700 nm) through four intermediate light levels to complete darkness. The plants were allowed to adapt for more than thirty minutes at every light intensity. Finally, the measured leaves were harvested to determine surface area, dry weight and total nitrogen content.

The photosynthetic data of each plant were fitted by non-linear regression analysis to a negative exponential function of light intensity (de Wit et al, 1978):

$$P = (P_m + R_d) \times (1 - \exp(-I \times \varepsilon/(P_m+R_d))) - R_d \qquad (2.1)$$

where P is the net CO_2 assimilation rate (g m^{-2} h^{-1}), P_m is the net CO_2 assimilation rate at light saturation (g m^{-2} h^{-1}), R_d is the dark respiration rate (g m^{-2} h^{-1}), ε is the initial light use efficiency (g J^{-1} s h^{-1}) and I is the incident photosynthetically active radiation (W m^{-2}). The results were analysed with a multifactorial analysis of variance with block, genotype and treatment as independent variables.

Field experiment. Plots of the cultivars Bintje and Surprise, and of the late cultivar Pimpernel were laid out at distances of 9 to 14 m on a sandy soil in a sugar beet crop, to minimize interplot interference (see Table 1.1: Experiment 1). Per plot of 4 by 3.75 m, 60 tubers were planted on April 29, 1987. Fifty per cent emergence was reached 20 days after planting for cv. Bintje, followed by Pimpernel and Surprise four and five days later. The experiment was arranged in a fully randomized design with three genotypes and three treatments in four replicates. One third of the plots was sprayer-inoculated 55 days after planting with a suspension of *P. infestans* (race 1,2,3,4,5,7,10,11; 150 ml per plot; 20 000 sporangia ml^{-1}). Another third of the plots, the controls, received regular sprayings with contact fungicide (maneb-tin) throughout the growing season, and remained practically free from late blight. The last third was left to natural infection, inoculum or fungicide was not sprayed.

Rates of photosynthesis were measured in different, randomly selected plots on

seven days, in July and August. This was carried out with a portable leaf chamber analyzer (LCA; Analytical Development Co. (ADC), UK). All measurements were done at light saturation. An incandescent lamp cooled by a fan was held over the enclosed leaf for at least one minute; the light intensity was 400 W m^{-2} of photosynthetically active radiation. The rate of photosynthesis was calculated following the procedure described by von Caemmerer and Farquhar (1981). The rate of photosynthesis of four distal non-infected leaflets of two or three leaf layers (top third, middle third and - if still present - bottom third) was measured in each selected plot. The four leaflets from a leaf layer were harvested as a group and total dry matter, leaf area and nitrogen content were determined.

Because the experimental design was non-orthogonal, the results were analysed using multiple linear regression on dummy variables (Snedecor and Cochran, 1980, p. 421), with day of measurement, genotype and treatment as independent variables.

Results

For each plant in the pot experiment, disease severity values were calculated separately for leaves and stems. Disease severity was expressed as a percentage of lesion

Table 2.1. Gas exchange parameters and plant characteristics of two potato cultivars, pot experiment. Parameters: photosynthetic rate at light saturation (P_m), dark respiration (R_d), initial light use efficiency (ε). Characteristics: disease severity, leaf nitrogen (N) content and specific leaf weight (*SLW*). Means and standard errors of difference.

Cultivar	Treatment	n^1	P_m	R_d	ε	Lesions		N-content	*SLW*
						leaves	stem		
			(g m^{-2} h^{-1})		(g MJ^{-1})	(%)	(%)	(g N m^{-2})	(g m^{-2})
Bintje	Inoculated	8	5.66	0.30	20.4	19.8	16.2	2.89	56.2
Bintje	Control	8	5.71	0.27	17.8	0.0	0.0	3.33	57.4
Surprise	Inoculated	8	4.70	0.29	18.7	16.5	13.2	2.48	44.3
Surprise	Control	8	4.99	0.27	16.8	0.0	0.0	2.64	47.6
	S.E.D.[2]		0.43	0.02	1.5	4.7	3.1	0.16	3.7

[1] Number of replicates. Each replicate represents one photosynthesis-light response curve with observations at six different light intensities.
[2] Standard error of difference of means. For lesion percentages only calculated for the inoculation treatment.

coverage of leaves or stem below the measured leaf. The leaves measured were green, symptomless leaves from the un-inoculated tops of the plants. When photosynthesis was measured, the average disease severity of inoculated plants was between 10 and 20%, with no significant differences between 'Bintje' and 'Surprise' (Table 2.1). All inoculated plants, except one 'Bintje' plant, had at least one stem lesion that completely

Table 2.2. Plant characteristics of three potato cultivars, field experiment. Characteristics: percentage leaf lesion coverage at three levels in the canopy, percentage stem lesion coverage, total number of leaves with distal leaflets longer than 5 cm and number of leaves still attached and at least partly green. Data on two days after planting (*DAP*). Standard errors of lesion coverage percentages and leaf numbers were lower than 13.0% and 0.72 respectively, unless otherwise indicated.

DAP	Cultivar	Treatment	Lesions				Leaf number	
			top (%)	middle (%)	bottom (%)	stem (%)	total	green
62	Bintje	Inoculated	19.4	30.6	57.8	11.4	12.4	
		Unsprayed	0.2	2.4	0.3	0.0	12.6	
		Control	0.0	0.0	0.0	0.0	12.3	
	Surprise	Inoculated	1.3	1.2	1.1	0.1	12.1	
		Unsprayed	0.0	0.0	0.0	0.6	13.3	
		Control	0.0	0.0	0.0	0.0	11.9	
	Pimpernel	Inoculated	2.0	6.5	9.8	0.0	11.1	
		Unsprayed	0.0	0.1	0.0	0.0	11.8	
		Control	0.0	0.0	0.0	0.0	12.0	
98	Bintje	Inoculated	100.0	100.0	100.0	100.0		0.0
		Unsprayed	89.1	96.3	99.2	77.1	14.0	0.4
		Control	0.0	0.0	0.0	0.0	14.2	9.6
	Surprise	Inoculated	45.3 [1]	60.5	87.4	60.4	13.7	1.0
		Unsprayed	0.0	0.6	3.2	8.1	18.0	11.0
		Control	0.0	0.0	0.0	0.0	16.1	8.9
	Pimpernel	Inoculated	48.5	79.2	78.6	39.6	15.4 [2]	1.0
		Unsprayed	7.3	13.8	40.4	0.1	16.5	7.4
		Control	0.0	0.0	0.0	0.0	19.8	11.1

[1] Standard error is 22.0%.
[2] Standard error is 1.06.

encircled the stem at some point below the measured leaf.

Infection with *P. infestans* did not significantly affect the photosynthesis parameters of either 'Bintje' or 'Surprise' (Table 2.1). The rate of photosynthesis at light saturation was higher for 'Bintje' than for 'Surprise' ($P < 0.05$). This may be explained partly by differences in the nitrogen content per unit leaf area or specific leaf weight (*SLW*) of the cultivars (Table 2.1). The nitrogen content and specific leaf weight were closely correlated ($r^2 = 0.79$, $n = 32$), and both showed a weak positive correlation with P_m ($r^2 = 0.28$ in both cases, $n = 32$).

In the field experiment, photosynthesis was measured in July (71, 72, 78 days after planting) and August (days 99, 106, 107 and 110). The average conditions of the plants on days 62 and 98, i.e. before these measurements, are shown in Table 2.2. At 62 days after planting, plants of all cultivars and treatments had formed 11 to 12 leaves per main stem, and had already dropped one or two leaves. Cv. Bintje had almost completed its leaf formation by day 62, whereas subsequent leaf formation was more pronounced in the late cultivar Pimpernel than in Surprise. Inoculation significantly reduced the number of leaves in these two cultivars.

Table 2.2 also gives data on stem lesion coverage and on leaf lesion coverage in the three canopy layers where the photosynthesis measurements were taken. As expected, disease development was strongest in inoculated plots, followed by the unsprayed plots, while the controls remained free of disease. Cultivar Bintje was more severely diseased than Surprise and Pimpernel. Leaf lesions developed fastest in the lower layers of the canopy.

The results of the photosynthesis measurements are given in Fig. 2.1. P_m varied with leaf position, being highest in the top leaf layer. Within leaf layers, P_m showed weak positive correlations with the percentage lesion coverage (from top to bottom: $r^2 = 0.07$, $r^2 = 0.06$, $r^2 = 0.31$). However, treatment effects were only significant in the lower two leaf layers, while significant genotype effects occurred at all levels. The cultivars with the lowest P_m values in the top and middle layers were Surprise ($P < 0.01$) and Pimpernel ($P < 0.05$), respectively; a genotype-treatment interaction was not present in these two layers. In the middle leaf layer, the P_m values of inoculated plants were significantly higher than those of unsprayed plants and controls ($P < 0.01$). In the lowest leaf layer, a genotype-treatment interaction was present ($P < 0.01$). The P_m values of inoculated plants of cvs Bintje and Pimpernel were higher than those of the controls ($P < 0.01$ and $P < 0.05$, respectively), whereas Surprise did not respond to treatment ($P > 0.1$). Thus 'Bintje' had the highest P_m of the inoculated cultivars ($P < 0.01$), while 'Surprise' was superior to 'Pimpernel' in the controls ($P < 0.01$).

The observed differences in P_m between leaf layers and between cultivars were

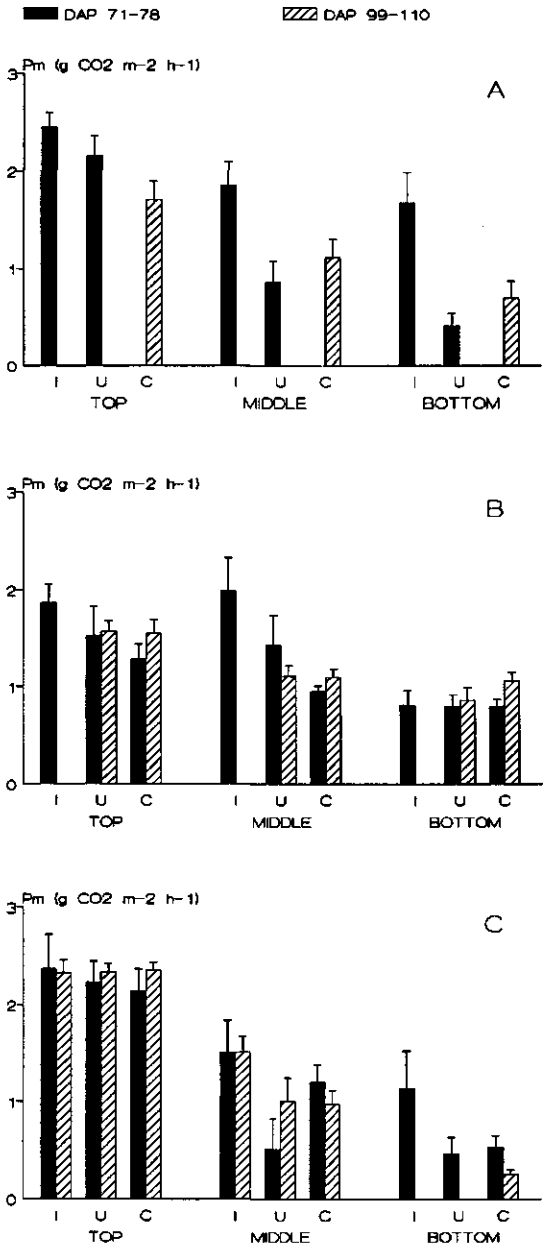


Fig. 2.1. Net photosynthetic rate at light saturation (P_m) of three potato cultivars, after three treatments (I = inoculated, U = unsprayed, C = control), measured at three levels in the canopy (top, middle and bottom leaf layer) during two periods (71-78 and 99-110 days after planting (DAP)). Means and standard errors of the mean. A: cv. Bintje; B: cv. Surprise; C: cv. Pimpernel.

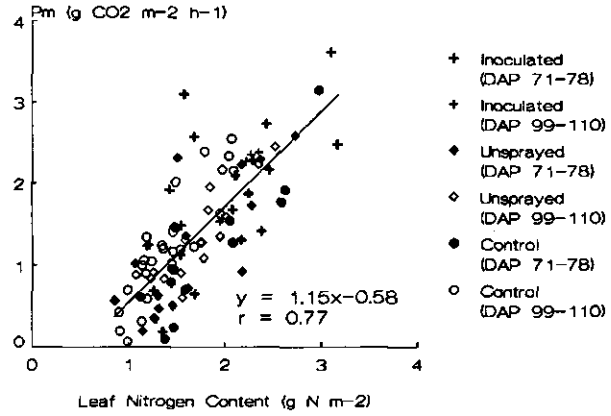


Fig. 2.2. Relationship between net photosynthetic rate at light saturation and leaf nitrogen content of potato cultivars Bintje, Surprise and Pimpernel. Different symbols indicate different treatments (inoculated, unsprayed or control) and different periods of measurement (71-78 or 99-110 days after planting). Points represent mean photosynthetic rates of four replicates of specific combinations of cultivar, treatment, day of measurement and leaf position.

associated with differences in nitrogen content (Fig. 2.2). The nitrogen content increased with the higher leaf positions, and was generally greatest in the cultivar Pimpernel, followed by Bintje. The nitrogen content also varied with time, showing a decrease in 'Surprise' and 'Pimpernel'. The positive correlation between P_m and nitrogen content in the field experiment, when all measurements are taken into consideration, was stronger than in the pot experiment ($r^2 = 0.59$; top layer only: $r^2 = 0.53$), and the correlation of P_m with specific leaf weight was again equally strong (not shown, $r^2 = 0.54$).

Discussion

The coefficient of variation of individual measurements of P_m, calculated as the square root of the error mean square divided by the overall mean, was 17% in the pot experiment and 31% for the top layer measurements in the field experiment. Thus there was a large variation in the pot experiment in spite of the homogeneity of the environmental conditions and the precision of the measurements. This indicates that much of the variation in P_m was due to the intrinsic variation between leaves. Therefore, the number of replicates needed to determine differences in photosynthetic rates between treatments or genotypes is high irrespective of the experimental conditions.

The average P_m value of young leaves was 5.3 g m^{-2} h^{-1} in the pot experiment and 2.0 g m^{-2} h^{-1} in the field. The field values are comparable to those reported by Dwelle (1985)

and Firman and Allen (1988) for field-grown potatoes. The high P_m values reported here for the plants of the pot experiment are similar to those found by J. Schans (pers. comm., 1989) for the cultivars Darwina and Irene, grown in pots in the greenhouse and examined with the same equipment used in the present study. Vos and Oyarzun (1987) also used the same equipment, but found P_m values up to 4.0 g m^{-2} h^{-1} for cv. Bintje. They reported a close relationship between age-dependent reduction in leaf nitrogen content and reduction in P_m. The present results cannot explain the differences between the P_m of field- and pot-grown plants on the basis of differences in leaf nitrogen content, since the nitrogen content was determined for whole leaves in the pot experiment and only for distal leaflets in the field. Differences between the distribution of lesions over leaf layers probably did not contribute to the discrepancy in rates of photosynthesis. The method of inoculation used in the pot experiment led to healthy plant tops and diseased lower plant parts, as was also seen in plants infected in the field (Table 2.2). Irrespective of a possible involvement of the leaf nitrogen content or disease pattern, the different growing and measurement conditions may have caused the differences in P_m found in the present study. The pot plants were optimally supplied with water and nutrients, and all leaves continuously received ample light because the pots were widely spaced. The measurement temperature of 20 °C and the longer time of adaptation to high light intensity (30 min in the pot experiment vs. 1 min in the field) could also have increased the P_m in the climate chamber.

A small positive effect on P_m was found in the lower leaf layers of cvs Bintje and Pimpernel after inoculation, possibly due to reduced shading because of foliage loss. This small effect would have negligible consequences for production. In healthy crops incident light is primarily absorbed by the higher leaf layers. This effect is enhanced in blighted potato plants, where disease occurs mainly in the lower plant parts. In the three potato cultivars studied, infection by *P. infestans* did not have a systemic effect on the P_m, R_d and ε of green leaf tissue in the plant tops. Even lesions encircling the stem did not reduce the rate of photosynthesis. Thus vascular transport was not hampered, and nor were toxic substances secreted. This is consistent with the insensitivity of the crop-*LUE* to the disease, as has been reported in the literature for a wide range of potato genotypes (Haverkort and Bicamumpaka, 1986; Waggoner and Berger, 1987).

It can be concluded that the photosynthetic rate in green leaves of infected plants is not a suitable physiological selection criterion in breeding potatoes for tolerance to late blight.

CHAPTER 3

Leaf area dynamics of potato cultivars infected by *Phytophthora infestans*

Abstract

The effect of *Phytophthora infestans* on foliage growth and senescence of three potato cultivars was studied in two field experiments. Inoculum or fungicide was applied in different frequencies to establish a range of levels of disease. At weekly intervals leaf numbers were determined as well as vertical canopy profiles of senescent and lesion covered leaf and stem area.

P. infestans reduced appearance of new leaves on the main stem only at the highest level of disease. The cultivars differed more in rate of primary infection of healthy leaves than in the subsequent increase in percentage lesion coverage of the infected leaves. Differences between cultivars in stem lesion coverage resembled the differences for leaf lesions, but in every cultivar stem lesions were most prominent in the top of the canopy, contrary to leaf lesions. *P. infestans* stimulated leaf senescence similarly in the different cultivars.

Introduction

Late blight shortens green leaf area duration (*LAD*) of potato crops, but does not reduce the efficiency of light use by the green leaves (Chapter 1; Haverkort and Bicamumpaka, 1986). The rate of photosynthesis of green leaves is not reduced by the pathogen (Chapter 2). Differences between potato cultivars in yield loss thus are only caused by differences in *LAD*. *LAD* is determined by the available leaf area at initiation of the disease, by the capacity of the host to resist extension of the pathogen through the foliage, and the capacity to tolerate the presence of disease without acceleration of senescence in non-infected leaf tissue. Every cultivar can be characterized by its level of resistance, which can be complete or partial, and its level of tolerance to blight. For breeding purposes it is important to quantify the genetic variation for these characteristics. However, studies of leaf area dynamics of blighted potato plants have tended to ignore tolerance, and have not taken genotypic differences in available leaf

area into account. Leaf appearance, growth and senescence have generally only been studied in disease-free crops, while leaf area loss caused by blight has usually been quantified as rate of increase of percentage diseased foliage, without concurrently quantifying the dynamics of undiseased leaf area (Rotem, Kranz and Bashi, 1983).

The present chapter quantifies the effect of partial resistance, tolerance and varietal patterns of foliage growth and senescence on the dynamics of green leaf area in blighted crops of three potato cultivars. The spatial distribution of lesions over different leaf positions and stem internodes is included in the study because the rate of leaf destruction may depend on the position of lesions (Lapwood, 1961c; Wenzl, 1967).

Materials and Methods

Data were gathered in two field experiments using the cultivars Bintje, Surprise and Pimpernel. Foliage resistance to blight of these cultivars is rated as 3, 7 and 8 respectively (Anonymous, 1988; scaling from 1, very susceptible, to 9, very resistant). Planting dates were April 29, 1987 (Experiment 1) and June 1, 1988 (Experiment 2). Experiment 1 comprised three levels of disease, while Experiment 2 had four. The highest level of disease was established by spraying a suspension of sporangia over the plots, on June 23, 1987 and July 27, 1988 ('inoculated'). Disease was absent or low in a treatment where fungicide was applied weekly until the foliage died ('control'). Intermediate levels of disease were established without the use of inoculum by stopping fungicide application eight or ten weeks after planting ('unsprayed-A' and 'unsprayed-B', the latter only in Experiment 2). Experiment 1 followed a completely randomized design while Experiment 2 had a randomized block design; both experiments had four replicate plots per combination of cultivar and treatment. All plots in Experiment 2 were sprinkler-irrigated on rainless days. Further details of the cultivars, the treatments and the lay-out of the experimental plots, have been reported elsewhere (Chapter 1; Table 1.1).

In each plot, four observation plants were chosen, i.e. sixteen plants per cultivar-treatment combination. At various positions along a representative main stem of each observation plant, stem internodes and leaves were tagged. At weekly intervals, after inoculation, the percentages lesion coverage and senescence of these tagged internodes and leaves were visually estimated. In Experiment 1, these weekly measurements were repeated four times, at three positions along the stem, in the bottom, middle and top of the canopy. In Experiment 2, every third internode and leaf was studied weekly until the leaf had no green area left. With the growth of the plants, newly appeared leaves were tagged and included in the measurements. In both experiments, the number of leaves on the chosen main stems, with distal leaflets longer

than 5 cm, was also determined weekly.

For each separate leaf studied in Experiment 2, the data of increasing lesion coverage and senescence were fitted by non-linear regression analysis to logistic functions of time (the logistic giving better fit than the linear or exponential regression):

$$\begin{aligned} l &= 100 / (1 + \exp(-r_l(t - t50_l))) \\ s &= 100 / (1 + \exp(-r_s(t - t50_s))) \end{aligned} \qquad (3.1)$$

where l is the percentage lesion covered area of the leaf (%), s is the senesced percentage of the non-lesion covered area of the leaf (%), t is time after inoculation (d), r_l and r_s are the logistic rates of increase of lesion coverage and senescence (d^{-1}), $t50_l$ and $t50_s$ are the inflection points of the curves (d), i.e. the number of days after inoculation when l or s is 50%. The estimates of the parameters r and $t50$ were subjected to a multiple linear regression analysis (Snedecor and Cochran, 1980), with genotype, level of disease and leaf position as independent variables. Analysis of variance was used to examine cultivar effects on stem lesion coverage, and to examine cultivar and treatment effects on leaf number.

Results

Lesion occurrence. In Experiment 1, all leaves of inoculated 'Bintje' plants showed

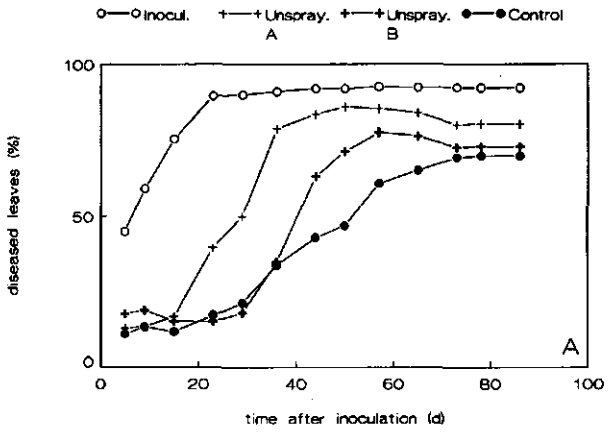
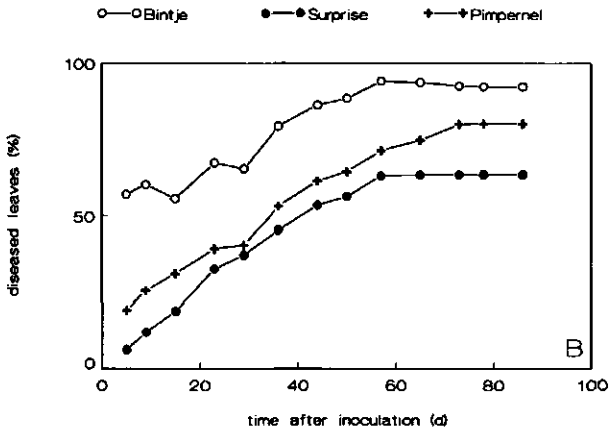
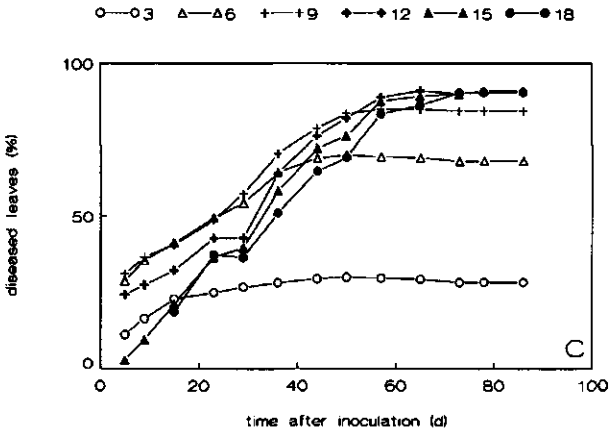
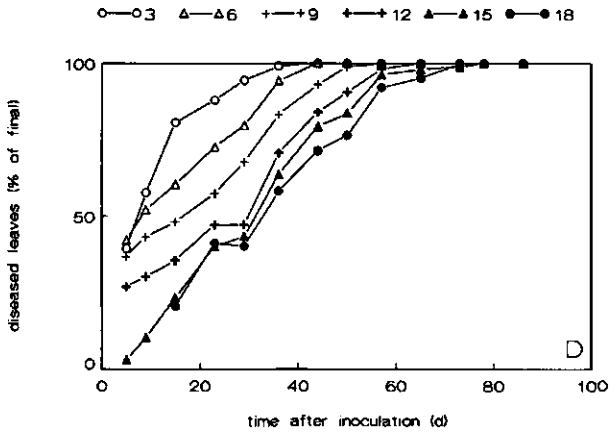


Fig. 3.1. Time course of percentage of leaves with lesions of *P. infestans* in Experiment 2. Points refer to estimates taken from multiple regression analyses on treatment, cultivar and leaf position. A: the effect of treatments; B: the effect of cultivars; C: the effect of leaf position, counted from the bottom; D: as C, but corrected for natural leaf senescence. ↑→


Bintje
Surprise
Pimpernel
diseased leaves (%)
100
50
0
0
20
40
60
80
100
B
time after inoculation (d)


3
6
9
12
15
18
diseased leaves (%)
100
50
0
0
20
40
60
80
100
C
time after inoculation (d)


3
6
9
12
15
18
diseased leaves (% of final)
100
50
0
0
20
40
60
80
100
D
time after inoculation (d)

lesions by the first day of measurement, i.e. seven days after inoculation, whereas fewer leaves with lesions were found on 'Pimpernel' (90%) and 'Surprise' (70%). Leaves of control plants of all cultivars were free of symptoms, whereas on unsprayed plants disease was found on leaves of 'Bintje' (11%) and 'Pimpernel' (2%). By the time of the last assessment the percentages for the unsprayed treatment had only slightly increased, while control plants still had no diseased leaves.

The first day at which the diseased area of a leaf was one per cent or more, was considered to be its time of primary infection. Primary infection was retarded in the control plants in Experiment 2. However, in spite of the continuous fungicide application

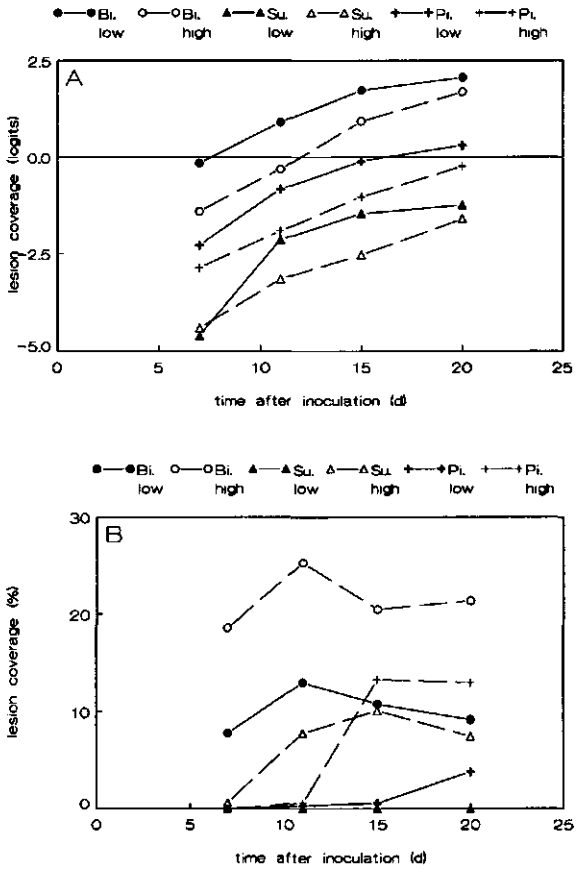


Fig. 3.2. Percentage lesion coverage (*I*) in leaves and stems of inoculated plants of cultivars Bintje (Bi), Surprise (Su) and Pimpernel (Pi) in Experiment 1, at two levels (high and low) in the canopy. A: *I* in leaves (*I* transformed to logits: $\ln(I/(100-I))$; B: *I* in stem internodes (*I* in %).

most leaves did not escape infection (Fig. 3.1A). Control plants thus became infected in Experiment 2 alone, probably because of the greater availability of natural inoculum, due to the late planting date, and better infection conditions, as a result of the occasional sprinkler-irrigations. In the unsprayed treatments of Experiment 2, the percentage diseased leaves increased significantly above that in the control plants within two weeks after fungicide application was stopped (Fig. 3.1A). The differences between cultivars were large (Fig. 3.1B). More than half the leaves of 'Bintje' already showed disease five days after inoculation, while similar levels of disease were reached by 'Pimpernel' and 'Surprise' only six and seven weeks later. These cultivar differences partly arose from premature natural infection in this experiment, causing the percentage diseased foliage area in 'Bintje', 'Surprise' and 'Pimpernel' to be 2.6%, 0.2% and 0.2%, respectively, two days before the artificial inoculation. Many old leaves, at the third and sixth stem node, counted from the bottom, escaped disease because of early, natural senescence (Fig. 3.1C). However, if we correct for natural leaf senescence by considering the number of diseased leaves relative to the final number becoming diseased at the same leaf position

Table 3.1. Experiment 2. Inflection time ($t50$) and logistic rate (r) of leaf lesion extension (l) and leaf senescence (s) of three cultivars exposed to four treatments; data are averages over leaf positions. Standard errors of inflection time and logistic rate were less than 2.0 d and 0.05 d^{-1}, respectively, unless otherwise indicated.

Cultivar	Treatment	$t50_l$ (d)	r_l (d^{-1})	$t50_s$ (d)	r_s (d^{-1})
Bintje	inoculated	12.5	0.70	17.2 [1]	1.08
	unsprayed-A	27.7	0.73	29.2	0.94
	unsprayed-B	31.9	0.63	36.9 [2]	0.90
	control	39.9	0.84	39.0	0.95
Surprise	inoculated	26.5	0.86	24.5	1.02
	unsprayed-A	40.0	0.91	36.4	0.88
	unsprayed-B	46.2	0.87	42.4	0.84
	control	47.5	0.87	44.3	0.89
Pimpernel	inoculated	24.3	0.75	23.4 [3]	0.97
	unsprayed-A	36.4	0.89	35.1	0.82
	unsprayed-B	46.3	0.80	40.1	0.79
	control	56.2	0.81	51.7	0.84

[1] Standard error is 4.3 d.
[2] Standard error is 2.1 d.
[3] Standard error is 2.3 d.

(Fig. 3.1D), we see that old leaves showed disease symptoms earlier than young leaves.

Fig. 3.1 shows the main effects of treatment, cultivar and leaf position. However, 13% of the variation in final number of diseased leaves was due to different responses of cultivars to treatments and leaf positions ($P < 0.01$). Primary infection was less impaired by continuous fungicide application in 'Pimpernel' than in 'Surprise', but 'Bintje' responded the least to treatment and leaf position.

Lesion coverage. In Experiment 1, the average percentage of the area of leaves covered with lesions (l) in inoculated plants was highest in cv. Bintje (Fig. 3.2A), followed by 'Pimpernel'. Leaf lesion coverage was most prominent in the old leaves low in the canopy whereas stem lesions were predominantly found in the higher stem internodes (compare Figs 3.2A and B). Logistic regression analysis was applied to the time courses of l in each of the 820 leaves studied in Experiment 2. Of these leaves, 143 died without disease symptoms and two could not be fitted satisfactorily with the logistic curve (r^2, the proportion variation accounted for, being less than 0.5). For the remaining 675 leaves r^2 averaged 0.99.

r_l did not vary strongly between cultivars, treatments and leaf positions (Tables 3.1

Table 3.2. Experiment 2. Percentage of variation in parameters of increasing lesion coverage and senescence, accounted for by adding terms to multiple regression models, and level of significance (P)[1] of each addition. Dependent variables: inflection time ($t50_l$) and logistic rate (r_l) of leaf lesion extension (l) and leaf senescence (s). Independent variables: block effects and main and interaction effects of cultivar, treatment and leaf position.

Added term	$t50_l$[2]		r_l[2]		$t50_s$[3]		r_s[3]	
	%	P	%	P	%	P	%	P
Block	1.0	**	0.1	n.s.	0.6	*	0.7	n.s.
Cultivar (C)	13.2	**	5.4	**	2.4	**	2.2	**
Treatment (T)	40.7	**	2.3	**	25.9	**	5.0	**
Leaf position (P)	15.4	**	1.5	*	37.6	**	6.2	**
C x T	2.0	**	2.3	**	2.2	**	0.7	n.s.
C x P	0.4	n.s.	3.3	**	1.3	*	1.1	n.s.
T x P	4.2	**	2.8	n.s.	3.9	**	1.2	n.s.
C x T x P	1.4	n.s.	3.3	n.s.	1.4	n.s.	3.4	n.s.
Total	78.3		20.9		75.2		20.5	

[1] ** = $P < 0.01$, * = $P < 0.05$, n.s. = not significant.
[2] $n = 675$
[3] $n = 466$

and 3.2; Fig. 3.3A). Only 21% of the variation in r_l was explained by differences between these variables (Table 3.2). $t50_l$, on the other hand, showed more variation, of which 78% was accounted for by differences between cultivars, treatments and leaf positions (Table 3.2). $t50_l$ was lowest in 'Bintje', while 'Surprise' and 'Pimpernel' did not differ significantly from each other (Table 3.1). As expected, $t50_l$ increased with longer periods of fungicide application (Table 3.1). Leaf position affected $t50_l$ in that young leaves reached 50% lesion coverage later than old leaves (Fig. 3.3A). Late cultivars, like 'Pimpernel', thus have an advantage over early cultivars, in that they longer continue to form new leaves

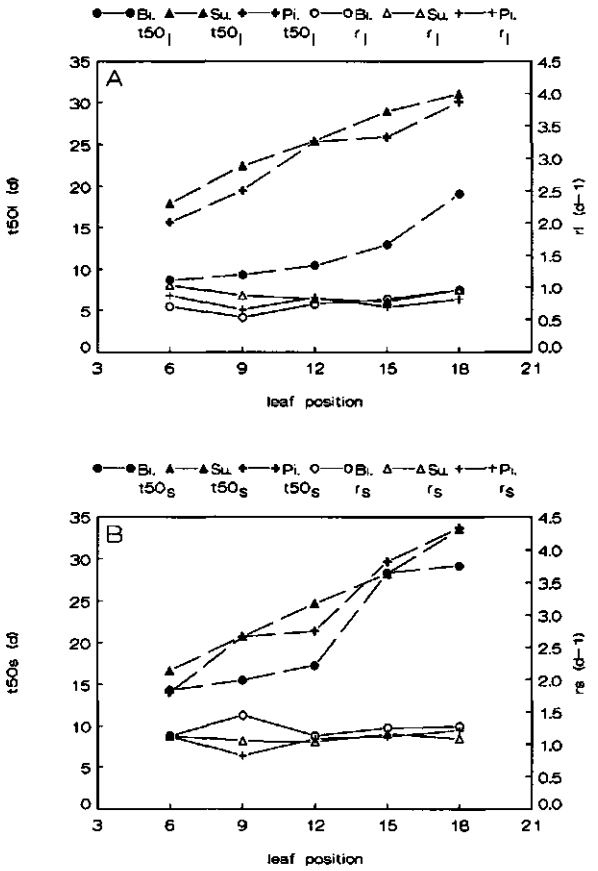


Fig. 3.3. Parameters of increasing leaf lesion coverage (*l*) and leaf senescence (*s*) as functions of leaf position counted from the bottom, of cultivars Bintje, Surprise and Pimpernel in the inoculated treatment of Experiment 2. Results of logistic curve fitting, characterized by the logistic rate parameter *r* and the inflection point *t50*. A: parameters for *l*; B: parameters for *s*.

with large $t50_l$ (Fig. 3.4). Only at very high levels of disease, as in the inoculated treatment, appearance of new leaves may be significantly reduced (Fig. 3.4; Experiment 1: Table 2.2).

Leaf senescence. Logistic curves of increasing percentage senescence (s) were fitted satisfactorily for 466 leaves (r^2 averaged 0.96). r_s, like r_l, was relatively constant over cultivars, treatments and leaf positions (Table 3.2). Effects on $t50_s$ strongly parallelled those on $t50_l$ (Table 3.1, Fig. 3.3), and the two parameters were closely correlated: $t50_s = 0.93 \times t50_l + 1.75$ ($r^2 = 0.95$, $n = 338$). The disease thus accelerated leaf senescence to the same extent in all cultivars.

Discussion

Effects of resistance, tolerance and foliage size on yield loss. *P. infestans* causes yield reduction in potato by reducing *LAD* (Chapter 1; Haverkort and Bicamumpaka, 1986). In the present experiments, *LAD* was reduced mainly by the coverage of leaves by lesions. Leaf lesion coverage started at the bottom leaf layers and gradually spread to the top of the canopy (Fig. 3.3A). The process was described very well, for individual leaves, by logistic curves, characterized by r_l and $t50_l$. In spite of the widely different resistance ratings of the cultivars, r_l hardly depended on cultivar and level of disease (Table 3.2). $t50_l$ was the main parameter explaining genotypic differences, and corresponded well with cultivar resistance ratings. Therefore $t50_l$ might be useful as selection criterion in

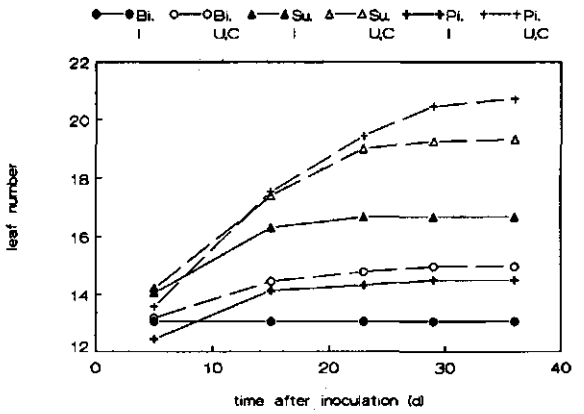


Fig. 3.4. Time course of average total number of leaves per main stem in inoculated (I) unsprayed or control (U,C) plants of cultivars Bintje, Surprise and Pimpernel in Experiment 2. Points for inoculated and non-inoculated plants refer to averages over 16 and 48 plants, respectively.

resistance breeding programmes. *LAD* was also reduced because *P. infestans* accelerated leaf senescence, but no genotypic differences in tolerance were detected: $t50_s$ approximately equalled $t50_l$ for all cultivars irrespective of treatment and leaf position. Fortunately such tolerance may not be needed, since only a small proportion of the yield loss was accounted for by the observed acceleration of senescence (9% and 12% in Experiments 1 and 2, respectively: Chapter 6). A third process reducing *LAD* was the reduction of new leaf area formation during the epidemic. Appearance of new leaves on the main stem was hampered at high levels of disease (Fig. 3.4). This reduction of leaf area due to reduced leaf appearance and growth is probably unimportant compared to the effect of lesion coverage, as late blight generally appears at a late stage of crop development when further leaf area expansion is restricted anyway. However, in potato-growing regions with high natural levels of initial inoculum, such as central Africa and Mexico, the disease may appear earlier (L.J. Turkensteen, pers. comm., 1990). Apart from differences between cultivars in the rate of reduction of green leaf area, the amount of host leaf area at disease initiation should be considered. Late cultivars, as Pimpernel, may be more tolerant to leaf loss than early cultivars because they form more leaves (Fig. 3.4; Taylor, 1953). Therefore late cultivars can longer maintain green leaf area in the top of the canopy, while the disease spreads from the lower leaf layers upward.

The role of stem lesions. Primary infection of fully developed leaves did not often originate from lesions on sustaining stem internodes, since the disease was most prominent in old leaves, low in the canopy, where stem lesions were almost absent (Fig. 3.2). The prominence of stem lesions in the plant tops may have been caused by the artificial inoculation, sprayed drops of inoculum being intercepted mainly in the axils of leaves high in the canopy. Leaf infection, on the other hand, may be more dependent on the higher humidity around leaves at lower positions. This is consistent with the finding that old leaves showed disease earlier than young leaves, while the subsequent extension of lesion coverage occurred at similar rates, lesion growth being less affected by the microclimate than infection efficiency.

The role of epidemiological components of resistance. Already seven days after inoculation the cultivars in Experiment 1 differed widely in lesion coverage. Since at that time all visible disease originated from the first infection cycle after inoculation, the differences between the cultivars can only be explained by differences in infection efficiency and early lesion growth. Differences in latent period or sporulation may have contributed only very little. In Experiment 2, differences between cultivars also became visible early after inoculation while increasing only little afterwards (Fig. 3.3A). The method of inoculation used in the present study (commonly used in resistance breeding trials) provides a high and uniform level of initial inoculum, and may therefore be

inadequate for cultivars of which the level of partial resistance depends mostly on a long latent period or a low rate of sporulation. Because of the low percentage of variation in r_i accounted for by differences between cultivars, treatments and leaf positions (Table 3.2), differences between cultivars in growth of individual lesions may be more important, in selection procedures, than the total increase in lesion covered area within leaves.

CHAPTER 4

Components of resistance to *Phytophthora infestans* in potato: a review of the literature

Abstract

Six components of resistance to potato late blight are distinguished: infection efficiency, latent period, lesion growth rate, lesion size, infectious period and sporulation intensity. The use of quantifying these components for breeding purposes is discussed. An overview is given of literature data on the influence of various host characteristics, the pathogen isolate and environmental factors on resistance components. The literature is found to be inconsistent about genetic linkage of components and about the correlation between individual components and overall resistance levels in the field. It is concluded that both research and breeding would benefit from greater standardization of component definitions and experimental methods.

Introduction

Components of resistance represent the different stages and events in the life cycle of a pathogen with which the host can interfere. Parlevliet (1979) mentions infection efficiency (*IE*), latent period (*LP*), colony or lesion size (*LS*), infectious period (*IP*) and sporulation intensity (*SI*). If any of these stages or events is entirely obstructed, the resistance is complete and epidemics are prevented. If the obstruction is incomplete, the resistance is partial and epidemics are only delayed or slowed down. This review focusses on partial resistance in potato foliage to *Phytophthora infestans* (Mont.) de Bary. Partial resistance to this pathogen is believed to be more durable than complete resistance (Thurston, 1971), but no successful partially resistant cultivars have been introduced as yet (Umaerus et al., 1983). Breeding for partial resistance will benefit from more knowledge about the relative importance of the different components in reducing the rate of epidemic development, the genetic variation available for the components, and the strength with which they are associated (Parlevliet, 1979).

Jones, Giddings and Lutman already reported about components of resistance to potato late blight in 1912, and many studies have followed. Therefore much information

is available but, as stated by Thurston (1971), "the main problem in evaluating (...) general resistance to *P. infestans* is integrating and correlating the disease reactions given by different investigators". Resistance studies differ in definitions of the components, measurement methods and experimental conditions. Often the definitions or methods are not clearly stated, thus making comparison to other studies altogether impossible.

Any partitioning of the life cycle of the pathogen, and concurrent division of host partial resistance into components, is to some extent arbitrary. The life cycle could be analysed in less detail, leading to lumping of components, while greater detail would increase the number of components. *IE* may e.g. be split into efficiency of penetration and efficiency of establishing a food relationship (Berggren et al., 1988), and *SI* may be split up in rate of production of sporangia and rate of sporangium detachment from the sporulating lesion surface (Bashi et al., 1982). Alternatively, *IE* and *SI* can be combined with dispersal efficiency into a 'daily multiplication factor' (*DMFR*, Vanderplank, 1963; Oort, 1968). Lesion size (*LS*) is considered a component of resistance, although it is a complex characteristic depending on both lesion growth rate and the elapsed period of lesion growth before the time of observation. The more simple trait of lesion growth rate (*LG*) may therefore be a better measure of lesion expansion than *LS*. Efficiency of dispersal of sporangia and sporangium catch by leaves, processes that depend on plant habit, are not considered as resistance components but as factors affecting escape (Parlevliet, 1979). In this review the component list of Parlevliet (1979) will be used, extended with *LG*.

The degree of association between components can only be assessed if the components are adequately quantified. If *LS* is expressed as lesion area, and *SI* is expressed as sporangium production per lesion, these components will seem positively correlated, even when lesion growth and sporulation are independent processes.

The apparent level of partial resistance of potato plants to *P. infestans* depends on the cultivar, the host predisposition as determined by plant and leaf age and previous growing conditions, the pathogen isolate and the environment. The influence of variation in these factors on the overall level of partial resistance has been studied more intensively than their influence on individual components, and rarely have the components been quantified in the field. The complicated causes of disease make it difficult in field experiments to distinguish differences in tissue susceptibility from heterogeneous spore deposition and differences in microclimate. Therefore most component studies have been carried out in the greenhouse or the laboratory. This facilitates the comparison of results, but does not show the importance of the components in the field.

In this review many publications, in which components of resistance to potato late blight are quantified, are brought together. The extent to which the various resistance components are affected by different characteristics of the host, the pathogen and the environment, is assessed. Field studies are especially emphasized. From this overview of the literature conclusions are drawn about how the methodology of experimental components analyses in research and resistance breeding may be improved.

The effect of the host genotype

One of the first studies of differences between potato cultivars in levels of components of resistance to *P. infestans* was carried out by Jones et al. (1912). They demonstrated that both *LG* and *IE* of three potato cultivars varied according their level of resistance in the field, the better correlation being with *LG*. They concluded that the level of partial resistance was mainly determined in the mesophyll of the leaves. A large selection of the literature about such components analyses comparing different cultivars is listed in Table 4.1. Most studies include the easily measurable components *IE* and *LS* or *LG*. *LP* and *SI* are less often measured, while information about *IP* is very rare. The studies were mostly done under controlled conditions, using leaf tissue. Field estimates of *IE* and *LG* were given by Colon and Budding (1990), of *LP* and *IP* by Lapwood (1961b), and of *SI* by James and Fry (1983). Stem lesions were included in the studies of Lapwood (1961d) and Pietkiewicz (1976). Differences between cultivars were similar for leaf and stem lesions.

The genetic variation for components is generally larger in wild *Solanum* species than in genotypes of *S. tuberosum* (Guzman-N, 1964; Nilsson, 1981; Colon and Budding, 1990). However, attempts to transfer the resistance from wild species into cultivated species have not yet been very successful (Umaerus et al., 1983).

The relative importance of the different components for overall resistance may be deduced from the strength of the correlation of the components with disease progress in the field. Because statistical analyses were usually not given, the reported correlations (Table 4.1) should be taken with caution. The correlations do not show a consistent pattern. For every component, studies can be found that support or contradict a great relevance for field behaviour. For example, of the 15 studies listed in Table 4.1 for which the strength of the correlation of both *IE* and *LS* or *LG* with field behaviour is given, *IE* reduces disease best in three cases, in six cases *LS*/*LG* seems more important, while six studies found similar correlations of both components with field behaviour.

In most studies, the components are correlated (Table 4.1). However, this association may largely be caused by the nonrandom choice of genotypes for the studies, where

Table 4.1. Studies of components of resistance of potato genotypes to *Phytophthora infestans*. Components studied (*IE* = infection efficiency, *LP* = latent period, *LG* = lesion growth rate, *LS* = lesion size, *SI* = sporulation intensity, *IP* = infectious period), number of potato genotypes screened, correlation of components with field resistance (in order of decreasing strength of the correlation, equally correlated components separated by a slash, uncorrelated components preceded by a minus-sign), and presence (+) or absence (-) of a positive correlation between the components studied. Controlled conditions unless otherwise indicated.

Source	Components studied	Number of genotypes screened	Correlation with field	Correl. betw. comp.
Jones et al. (1912)	*IE, LG*	3	*LG, IE*	+
Vowinckel (1926) [1]	*LP, LS*		*LP/LS*	
Crosier (1934)	*IE, LP, LS*	4		
Kammerman (1951) [2]	*IE, LP, LS, SI*		*LS, SI, -IE/LP*	
Schaper (1951)	*LP, SI*	33	*LP, SI*	+
Müller & H. (1953)	*IE*	12	*IE*	
Desmukh & H. (1956)	*IE, LP, SI*	2	*IE/LP/SI*	+
van der Zaag (1956, 1959)	*IE, LG, SI*	4	*IE, SI, LG*	+
Gallegly & N. (1959)	*LG*		*LG*	
Umaerus (1960) [3]	*IE, LG*	9	*LG*	
Hodgson (1961)	*IE*	4	*IE*	
Lapwood (1961b)	*IE, LP*[4], *LG, SI, IP*[4]	7	*SI, -IE*	
Lapwood (1961c)	*LG*	7	*LG*	
Lapwood (1961d)	*IE, LS, SI*	44		+
Niederhauser (1961)	*IE, LG*		*IE/LG*	
Hodgson (1962)	*IE, LS, SI*	8	*LS, IE/SI*	-
Jeffrey et al. (1962)	*LP*	8		
	LP	4	*LP*	
	IE, LP, LG	2	*LG*	-
Knutson (1962)	*IE, LS, SI*	5	*SI, -IE/LS*	-
Weihing & O'K. (1962)	*LP, LG, SI, IP*	11	*SI/LG*	+
Lapwood (1963)	*SI*	14	*SI*	
Umaerus (1963)	*IE*	2	*IE*	
	LG, SI	4	*LG, SI*	+
Guzman-N. (1964)	*IE, LP, LS, SI*	10	*LP/LS/SI, IE*	+
Main & G. (1964)	*IE, LP, LG, SI*	21	*-IE/LP/LG/SI*	
Takase (1968)	*IE, LS*		*IE*	
Malcolmson (1969)	*IE, LP, LS, SI*		*-IE/LP/LS/SI*	-
Umaerus (1969a)	*IE*	44	*IE*	
Umaerus (1969b)	*IE*	103		
Umaerus (1969c)	*IE, LG, SI*	3		
Lapwood (1971)	*IE, LP, LG, SI*	9	*IE, LG, -SI/IP*	
	SI	12	*SI*	
Pietkiewicz (1976)	*IE, LS, SI*	30	*LS, IE/SI*	+
Umaerus & L. (1976)	*IE, LS, SI*	11	*IE/LS, SI*	+
Malcolmson & K. (1980)	*IE, LS*			+

Table 4.1, continued.

Source	Components studied	Number of genotypes screened	Correlation with field	Correl. betw. comp.
Nilsson (1981)	*IE, LS*	7	*IE, LS*	+
Carnegie & C. (1982)	*IE, LG*	12		
James & F. (1983)	*IE*[4]	4		
Victoria & T. (1984)	*LS*	6		
Berggren et al. (1988)	*LG*	3	*LG*	
Gees & H. (1988)	*IE, LP, LG, SI*	9	*-IE/LG/SI*	
Colon & B. (1990)	*IE*[4], *LG*[4]	8	*LG, IE*	

[1] Reported by Thurston (1971); [2] Reported by Knutson (1962); [3] Reported by Umaerus (1970); [4] Field measurement.

often very susceptible and very resistant genotypes were compared. Since furthermore most studies were done with only a few genotypes, the evidence of association of components remains small. There was also no convincing evidence for the statement of Parlevliet (1979) that correlations involving *IE* tend to be less strong than correlations between other components. Pietkiewicz (1976) has perhaps studied the matter of association between components most comprehensively. He found slightly positive correlations between most pairs of components in a set of thirty, mainly Polish, cultivars, but only the correlation between *IE* and *LG* was statistically significant.

Little research has been done to explain the differences between cultivars. Berggren et al. (1988), in a histological study of the infection process, concluded that differences between cultivars in level of partial resistance primarily reside in hyphal growth, the "preinfectional processes (not being) major discriminating factors determining general levels of resistance". The opposite conclusion of Wilson and Coffey (1980) they attributed to the use, in the latter study, of the cv. Pimpernel, which has an uncommon resistance against penetration of the epidermis. Differences between cultivars regarding disease escape caused by differences in plant habit are mentioned by Lapwood (1961c). Stephan (1965) pointed out that sporangium production may limit disease progress in the field only during the initial stages of epidemic development, the supply of sporangia no longer being limiting in later stages. He mentioned the work of Lapwood as confirmation of this view, since Lapwood (1961a) found differences between cultivars in the field to be explained by differences in sporangium production, while these cultivar differences diminished at later stages of the epidemics.

Differences between cultivars in level of partial resistance have often been related to cultivar maturity class; early cultivars are reported to be more susceptible than late ones (Toxopeus, 1960). In general the correlation between earliness and susceptibility is more evident than the correlation between lateness and resistance: there are fewer resistant early cultivars than susceptible late cultivars. Schaper (1951) found more variation in *LP* and *SI* in maincrop and late cultivars than in early cultivars. The observed correlation may originate from the lower selection pressures to which early ripening genotypes have generally been subjected in variety testing trials, escaping the main disease periods by early natural death (Gallegly and Niederhauser, 1959; Vanderplank, 1963; Umaerus et al., 1983). Lateness and partial resistance may also have been introduced together from wild species used in plant breeding. Ross (1986) estimates that 85% of the German potato cultivars contain genes from *Solanum demissum*. As a third explanation, the level of partial resistance of early cultivars may have been underrated because of their generally low foliage area, causing disease percentages to be scored as high while the absolute levels of pathogen extension are similar to those found in late cultivars. The latter explanation is perhaps the most attractive because measurements of components instead of overall level of partial resistance tend to show no correlation with lateness (Lapwood, 1963; Main and Gallegly, 1964). Umaerus (1969b) indeed found no increase in average lateness after selecting for a low *IE*.

The effect of plant and leaf age

Malcolmson (1969) found that plants inoculated early in the season showed higher *IE*, longer *LP*, lower *LS* and lower *SI*, compared to plants inoculated later in the season. Inoculation at a relatively young plant age thus changed only *IE* in the direction of higher susceptibility. Warren et al. (1971, 1973) found *IE* of *P. infestans* to be influenced by leaf position on the plant, even to the extent that at some leaf positions many 'hypersensitive lesions' (lesions that stop growing after an initial period of normal growth) occurred. Carnegie and Colhoun (1980, 1982, 1983) also studied leaf and plant age effects on components of resistance but disagreed with the findings of Warren et al. (1971, 1973), and attributed the variability of *IE* in their experiments to stress caused by artificial growing conditions of the plants. They found that *IE* did not respond to leaf age and only little to plant age, while *LG* generally increased with leaf age but decreased with plant age. Takase (1968) found that only in relatively young crops *IE* was highest in the older leaves. Later in the growing season the region of maximum susceptibility changed to higher leaf positions.

The effect of lesion position

The lower surface of each leaf is more susceptible to infection than the upper surface (e.g. Umaerus, 1969a), but in field epidemics this is more than compensated for by more frequent deposition of sporangia on the upper surface of leaves (Björling and Sellgren, 1955).

The epidemiology of potato late blight can be complicated by potato leaves responding differently to the presence of lesions, when the lesions are positioned differently on the leaf. Several authors have studied this by measuring the relation between the position of lesions on a leaf and the time until death of the leaf (Lapwood, 1961c; Weihing and O'Keefe, 1962; Stephan, 1965; van Oijen, unpublished results 1988). Differences between cultivars may also be important here. Lapwood (1961a) found that the average time till leaflet death after primary infection in the field, varied between cultivars from 3.5 to 6.5 days. Lapwood (1971) compared European and Mexican potato cultivars in the field and found that some of the Mexican cultivars already shed leaves when the lesions on them were still very small. He further confirmed earlier findings (Lapwood, 1961c) that usually the cause of leaf death was the advance of the fungus through the tissues, while less frequently leaves started to yellow and die because of damage to the vascular system. There were cultivar differences in the proportion of leaves that died due to such indirect effects of the disease.

Another complication of late blight epidemiology is the occurrence of stem lesions. Primary foci of the disease often start from stem lesions (van der Zaag, 1956). The stem lesions may originate from diseased tubers or from primary infection of stems by external inoculum in periods unfavourable to leaf infection. During short periods of high humidity *IE* is higher in leaf axils than on leaf blades (van der Zaag, 1956). In the further development of the epidemics stem lesions do not normally play an important role, except in long periods of hot and dry weather. Under such weather conditions, which are adverse to infection and sporulation, and may also be too hot for optimal lesion growth, the fungus can survive for long periods in stem lesions (van der Zaag, 1956; Clayson and Robertson, 1956; Rotem and Cohen, 1974). Much of the damage to the foliage under such conditions may arise from stems breaking at the site of stem lesions, followed by loss of all leaf area above the breaking point (Rotem et al., 1983).

The effect of host predisposition

Umaerus (1970), Thurston (1971), Pietkiewicz (1976), Ullrich (1976) and Darsow et al. (1988) review studies about factors that change the susceptibility of potato plants to *P.*

infestans. They mention nutrition, daylength, light intensity, plant and leaf age, and the presence of other pathogens in the plant. Some reports exist on the relationship between these factors and individual resistance components. A better N-nutrition of the plant may lead to lower *LG* (Lowings and Acha, 1959), but Carnegie and Colhoun (1983) found *LG* to increase linearly with rates of NPK fertilization at planting. Umaerus (1969c) also reported *LG* to be dependent on mineral nutrition, with minor effects of nutrition on *IE* and *SI* as well. *SI* seems to be reducable by both a too poor and a too rich nutrient availability (Cohen and Rotem, 1987). Such nutritional effects have recently been reviewed by Schmitthenner and Canaday (1983). Umaerus (1963) found that a short daylength during plant growth increases *IE*, *LG* and *SI*, such that differences between partially resistant and susceptible cultivars tend to decrease. Victoria and Thurston (1974) found *LS* to be increased by growing plants at low light intensity.

Since susceptibility of plants to infection depends on so many factors, it is probable that the presence of disease itself will also affect some components of resistance. *LP* can be reduced at high levels of disease in some cereal pathosystems (Mehta and Zadoks, 1970; Shearer and Zadoks, 1972; Johnson and Taylor, 1976, as quoted by Leonard and Mundt, 1984). *SI* can also be reduced in diseased plants (Parlevliet, 1979; Leonard, 1969 and Imhoff et al., 1982, as quoted by Leonard and Mundt, 1984). Induced foliage resistance after pretreatment with *P. infestans* has been demonstrated (Nandris et al., 1979). Doke et al. (1987) found that rubbing hyphal wall components of *P. infestans* into leaves of potato reduced *IE* in other leaves between one and twenty days after the treatment. Infection of lower leaves by *P. infestans* induced resistance in higher leaves of tomato (Fischer et al., 1988).

The effect of the pathogen isolate

Differences in aggressiveness between isolates have been shown by, amongst others, Knutson and Eide (1961), Jeffrey et al. (1962), Caten (1970), Latin et al. (1981) and Tooley and Fry (1985). Knutson and Eide showed variation between isolates in *IE*, *LP*, *LG*, *SI* and stem infection. Jeffrey et al. confirmed the dependence of *LP* on the isolate used. Caten found variation in *LP* and *SI* between single zoospore isolates derived from one parental isolate.

The effect of the environment

The study of Crosier (1934), relating resistance components to environmental variables as temperature and humidity, is still a source of much information. Crosier showed both

LP and lesion growth in stems to depend strongly on temperature while *IE* and *SI* depend more strongly on air humidity. Duration of leaf wetness also affects *IE* (Umaerus, 1969a). Cohen and Rotem (1987) and Schrödter (1987) have more recently reviewed studies about the dependence of sporulation of potato late blight on temperature, leaf wetness and humidity. Schrödter also briefly reviews the literature about environmental effects on *IE*. Harrison and Lowe (1989) added wind speed as an environmental factor affecting *SI*. A strong dependence of *LG* on temperature was reported by van Oijen and Budding (1988). Van Bruggen et al. (1987) and Martin et al. (1987) found that acid rain had only marginal effects on *IE*, *LG* and *SI*. It is still unclear to what extent differences in components with different positions in the canopy (e.g. differences in *SI*, *IE* and *LG* with leaf position: Lapwood, 1961b) are due to differences in microclimate.

Mechanisms of resistance

Biochemical, cytological and histological studies about the process of infection of *P. infestans* in compatible host cultivars, have been reviewed by Clarke (1983) and Keen and Yoshikawa (1983). The mechanisms underlying the components of resistance have not yet been clearly identified. Behnke (1979, 1980) found *LG* to be reduced in potato genotypes regenerated from calli selected after screening with a culture filtrate of *P. infestans*. The actual toxin was isolated by Stolle and Schöber (1984) but not yet used for studies of mechanisms of resistance. Morphological differences between cultivars may also affect components of partial resistance. Ullrich (1976) suggested that wettability of leaves may be a resistance component for which cultivars differ. In this respect Schöber (1987) refers to previous work of Henniger and Bartel (1963), who found that a high density of leaf hairs decreases *IE*.

Correlations, but no causal relationships have been demonstrated between resistance components and nutritional compounds such as sugars (Warren et al., 1973) or amino acids (Child and Fothergill, 1967), although *LG*, *IP* and *SI* all depend on the food relationship between host and pathogen.

Phytoalexins are no longer thought to be the sole factors that condition resistance (Érsek and Király, 1986).

Discussion and conclusions

In spite of much research about components of resistance to *P. infestans*, it is still largely unclear to what extent the values of the various components are determined by inheritable characteristics of the host, by phenological characteristics, or by pathogenic

or environmental factors. Research in this respect has been slow because the resistance mechanisms have not yet been sufficiently elucidated. This indicates a need for more mechanistic research. Progress in resistance breeding research could become faster by greater standardization in definitions, measurement methods and set-up for the experiments. The standardization might in part be realized by the common inclusion of a small number of standard genotypes in resistance component tests. This may preclude differences in opinion about the role of resistance components which often arise because of the different selections of genotypes studied (Parlevliet and van Ommeren, 1975). Identifying those resistance components that are most effective in reducing the rate of epidemic development, may be easier when additional methods, such as mathematical modelling, are used together with the experimental research.

Some conclusions about breeding methodology can also be drawn. None of the resistance components correlates significantly better with field behaviour than the others, although the components tend to be only weakly associated. Therefore analyses of components of resistance of potato genotypes to *P. infestans*, for breeding purposes, should be aimed at improving most or all components simultaneously. This should not be done under controlled conditions only. Field measurements, much negelected so far, should be done more often since genotype-environment interaction is probably strong for most components (Lapwood, 1961b; Cohen and Rotem, 1987).

CHAPTER 5

Models of fungal leaf diseases with components of resistance: a review of the literature

Abstract

Many epidemiological models that calculate the epidemic rate from knowledge of some of the components of resistance (infection efficiency, latent period, lesion growth rate, infectious period, sporulation intensity) have been published. This literature is reviewed to determine how the impact of these models on resistance breeding research may be increased, with emphasis on breeding for resistance to potato late blight.

Component models differ for 1) the number of components incorporated, 2) the way the components are quantified, 3) the inclusion of horizontally or vertically heterogeneous disease distribution, 4) the treatment of host growth, 5) the model type: deterministic or stochastic. These differences affect the usefulness of the models for assessing the relative importance of the resistance components. The models may be put to better use if more attention is paid to correct initialization and parameterization, and if comprehensive sensitivity analyses are carried out.

Introduction

Epidemiological models of fungal leaf diseases, in which components of resistance (see Chapter 4) are incorporated, were reviewed by Berger (1977). The following is an extended and updated review. Jeger and Groth (1985) indicate how epidemiological models could be used to calculate disease progress curves from knowledge of the components, in order to evaluate the overall level of partial resistance of cultivars. They prefer this method of cultivar evaluation to multiple regression and correlation techniques. Epidemiological models with resistance components may also be used for quantifying interplot interference in cultivar evaluation trials (Paysour and Fry, 1983; Elston and Simmonds, 1988). This review is intended to evaluate the use of models for analyzing the sensitivity of late blight to changes in resistance components of potato cultivars. The interaction of these genotypic differences with environmental conditions is not emphasized.

SEIR-models in human disease epidemiology

Components of resistance were introduced in epidemiological models by Kermack and McKendrick (1927). Their general model, consisting of two linked differential equations, was applied to infectious diseases in human populations of fixed size. In the model susceptible, infectious and removed individuals were distinguished. Susceptible individuals became infectious at a rate directly proportional to the product of the densities of susceptible and infectious individuals. The proportionality constant, later termed 'daily multiplication factor' (*DMFR*) in the botanical literature, thus was a measure of rate of production, dispersal and infectivity of infectious propagules. Infectious individuals were removed at a constant relative rate accounting for loss of infectiousness, isolation or death. The reciprocal of the removal rate was the average infectious period (*IP*) of diseased individuals, *IP* thus being exponentially distributed. The model has been termed the first 'SIR-model', after the initial letters of the three characteristic groups of individuals distinguished within the population (see Hethcote, 1976). A fourth group was later distinguished: individuals that had already been exposed to the disease, but were not yet infectious (SEIR-models, Anderson and May, 1982). The period before the start of infectiousness was called the latent period (*LP*).

SEIR-models in plant disease epidemiology

Vanderplank (1963) introduced SIR- and SEIR-models in plant disease epidemiology. The latent and infectious periods in his SEIR-model, however, were not exponentially distributed, but assumed constant for all disease units, i.e. lesions. His model is therefore formulated as a time-delayed differential-difference equation, later termed the 'paralogistic' equation (Zadoks and Schein, 1979). Preliminary analysis of this equation was carried out by Vanderplank (1963). However, the paralogistic equation is analytically less tractable than the original SEIR-model from human epidemiology, although its dynamics are not dissimilar (Jeger, 1986, 1987). Some analytical results can be derived if the removal term is left out, which reduces the paralogistic to a 'para-exponential' SEI-model, describing only the initial phase of epidemics. Corsten (1964) derived a formula for the steady state rate of exponential disease increase in the discrete-time analog of this SEI-model, expressed in terms of the resistance components *LP*, *IP* and *DMFR*. Oort (1968) presented a sensitivity analysis of the exponential rate in the discrete-time Corsten-model by varying the components, and found the strongest sensitivity for changes in *LP*. This in fact constitutes the first use in plant disease epidemiology of mathematical models for testing the sensitivity of disease progress to the levels of

individual resistance components. The initial settings of *LP*, *IP* and *DMFR* did not correspond to parameters of real epidemics, but more realistic and extensive numerical analyses were given by Zadoks (1971; Rabbinge et al., 1989), pointing to a major importance of *LP* in the analyses of 1971, while showing the prime importance of *DMFR* in 1989.

Alternative distributions for *LP* and *IP*

As indicated above, *LP* and *IP* were assumed to be exponentially distributed in the human disease SEIR-models, while in the first plant disease SEIR-models *LP* and *IP* were supposed to have zero variance among lesions. Alternative distributions for *LP* and *IP* were later introduced. Berger and Jones (1985) used distributed delays for the *LP* in their model, keeping *IP* fixed. The method of distributed delays offers a range of distributions, from a step function to almost normal distributions. Berger and Jones (1985) used four delay intervals and thus approached a normal distribution for *LP* quite closely. This seems to be a realistic distribution for the *LP* (Shaner, 1980). Knudsen et al. (1987) used distributed delays for both *LP* and *IP*.

It is still unclear how important the distribution of *LP* and *IP* is in epidemic models. Vanderplank (1963) thought that the distribution had little effect on calculated disease progress, while Berger and Jones (1985) had the opposite view.

Models of the exponential phase

Straightforward multiplication of the components can be considered as the simplest model for assessing the contribution of components to the initial exponential phase of epidemics (Zadoks, 1977). Van der Zaag (1959) found that the ranking of potato cultivars with respect to partial resistance to *P. infestans* closely followed the ranking of their multiplicated components. However, the analyses of Vanderplank (1963) and Oort (1968) already showed that this method unjustifiably considers all components to have the same effect on overall resistance, and the method has not been applied frequently. Slightly more sophisticated models of the exponential phase of epidemics were given by Leonard and Mundt (1984) and Gumpert et al. (1987; Gumpert, 1989). The model of Leonard and Mundt is a continuous model in which sporulation intensity (*SI*) varies during the *IP*. *SI* first increases linearly to a maximum and then decreases to zero during the remainder of the *IP*. *LP* is again a constant: it is the period before *SI* starts to rise. The model of Gumpert is very similar, although it is formulated as a discrete time model that derives the r_{app} (the logistic rate of disease increase) as the main eigenvalue of the matrix

describing 'lesion production' by lesions of different ages. The models of Leonard and Mundt (1984) and Gumpert et al. (1987, 1989) have been validated by comparison with field data. According to Gumpert (1989) the model of Leonard and Mundt was slightly better because of its more realistic treatment of the spore production pattern. Only Leonard and Mundt analyse the sensitivity of their model to changes in individual components. They conclude that *LP* is most important in diseases in which r_{app} and *LP* are both relatively high. They suggest that for diseases with short *LP* (as potato late blight), infection efficiency (*IE*) and *SI* are more important.

Modelling lesion growth

Zadoks (1977) mentions five components that should be included in a components analysis aiming at the evaluation of breeding material (*IE*, *LP*, lesion growth rate (*LG*), *IP*, *SI*). There is no published evidence that leaving out one or more of these components of the infection cycle leaves the model dynamics unaltered (Teng, 1985). However, a common feature of the models discussed so far is the absence of *LG* as a separate resistance component. Lesions are assumed to occupy a fixed lesion area from the beginning of their latent period. A fixed lesion area of 0.3 cm^2 was used by Michaelides (1985) in his simulation model of *P. infestans*. Michaelides treated sporangium dispersal in great detail, thus complicating the experimental determination of parameter values for his model, while losing realism by oversimplifying lesion growth. A fixed lesion area was also used by Shaner and Hess (1978) in their discrete-time model of *Puccinia recondita* in wheat. They were able to explain differences between cultivars in r_{app} by integrating measured component values in their model, but they did not directly analyse the sensitivity of their model to changes in the components. A sensitivity analysis of the model was presented by Kulkarni et al. (1982), who found that time to 100% leaf destruction was most affected by changes in *LP*, *SI* and *IE*, while changing *IP* had only a marginal effect. They also tested some pairwise changes, and found that the response was generally additive. Shaner (1983) later found differences between cultivars in the rate of lesion growth following latency. He therefore modified his model by including a linear growth rate of uredinia area, independent of their density, and found that a low uredinial growth rate may well contribute to a lower rate of disease progress in the field.

Incorporating *LG* in epidemiological models may be expected to be even more important in diseases with indeterminate lesion growth, i.e. without fixed final lesion sizes, as potato late blight. Berger and Jones (1985) incorporated a constant relative growth rate of the total diseased leaf area in their model. This neglects both the increasing limitation of susceptible host area when the disease progresses and the fact

that even individual lesions generally have decreasing relative area growth rates. This also applies to potato late blight: blight lesions have a constant radial growth rate (Gees and Hohl, 1988; van Oijen, 1989) so that lesion areas increase as a quadratic function of time instead of exponentially. Therefore, Berger and Jones (1985) indicate that it would be "more satisfactory to develop a separate submodel for lesion expansion, with the submodel based on the density and ages of lesions". Rouse (1985) incorporated space limitation of lesion growth in a model in which the area growth rate of individual lesions reduces linearly with the approach of a constant maximum lesion area. A submodel of lesion expansion, with a dynamically changing lesion size distribution, was incorporated in a model of potato late blight by van Oijen (1989), in which circular lesions were Poisson-distributed over leaflets, while their diameters changed by a radial growth rate that was proportional to the fraction of free leaf area on infected leaflets. Lesions in most pathosystems are not distributed randomly, so that the Poisson-distribution might better be replaced by a negative binomial distribution (Waggoner and Rich, 1981). Lapwood (1961c) reported that even within potato leaves and leaflets lesions of late blight are not randomly distributed. He found relatively many lesions on the distal leaflet and on tips and edges of leaflets. Waggoner and Rich (1981) further suggested abandoning the direct proportionality between lesion formation rate and the product of susceptible and infectious sites. Such nonlinear incidence rates are investigated intensively in human epidemiology (Liu et al., 1987).

A sensitivity analysis with the model of van Oijen (1989) showed that r_{app} was most sensitive to changes in *LG*, followed by *IE* and *IP*, and finally *LP*. Taking into account the genetic variation reported for the different components (see also Chapter 4), *LG* and *IE* seem to offer the best possibilities for improving the level of partial resistance in breeding programmes (van Oijen, 1989). Shrum (1975, cited by Loomis and Adams, 1983), included *LG* in his model of wheat stripe rust. Sensitivity analysis of *LG* and *SI* showed that *LG* always influenced the epidemic rate strongly, while *SI* only affected disease progress in weather unfavourable to the pathogen. Rapilly and Jolivet (1976) also incorporated *LP* and *LG* in EPISEPT, their model of *Septoria nodorum* in wheat. Recently Rapilly and Delhotal (1986; Rapilly, 1987) published a sensitivity analysis of EPISEPT, showing that r_{app} correlated more closely with *LG* than *LP*. Aust et al. (1983) published a model of barley powdery mildew, in which both rate and duration of lesion growth rate depended on temperature, but no sensitivity analysis was given.

Stochasticity

The early models of human infectious disease, as e.g. the model of Kermack and

McKendrick (1927), were deterministic (Bailey, 1975). However, gradually stochasticity has been incorporated and is now featured in most human epidemiological models (Becker, 1979). Models of plant disease epidemics on the other hand are still usually deterministic (Gilligan, 1985). The results of these models may differ from those of stochastic models, in spite of the large number of disease units involved, because of the non-linearity of pathosystems (Rouse, 1985). Preliminary stochastic models have been presented by Teng et al. (1977) and Sall (1980), for barley leaf rust and grape powdery mildew, respectively. In these models, which do not include *LG*, parameter values for a subset of the resistance components are drawn from a uniform or normal probability distribution. The stochastic models provide estimates of the variation in disease progress rates. Such estimates of variation are useful if the model is used in disease forecasting, because they set boundaries to the reliability of the forecasts. However, if the model is used for explaining system behaviour in terms of its components or to assess the importance of resistance components for breeding purposes, this variation is unwanted because it obscures the relation between the individual components and disease progress rate, and thus complicates the identification of the major components.

Horizontal heterogeneity

Epidemiological models usually model 'general epidemics', defined by Zadoks and Schein (1979) as epidemics developing spatially homogeneously from homogeneously distributed initial disease. However, gradually more models now incorporate the spatial aspect of development of epidemics. Paysour and Fry (1983) used a model to calculate the level of interplot interference in experiments with potato late blight. They showed the effect of plot shapes, sizes and distances on interference, but did not consider the role of resistance components. The relation between disease progress and two resistance components, comparable to *IE* and *LG*, was studied by Elston and Simmonds (1988) in their model of sugarcane smut. They used their model to quantify interplot interference in variety trials but did not examine the question whether the relative importance of resistance components depends on the strength of the interplot interference.

Vertical heterogeneity and host growth

The vertical distribution of disease in the crop may be of epidemiological importance. Ullrich (1958), Hodgson (1961) and Lapwood (1961c, 1963, 1971) reported that potato late blight started at the lower leaf layers and gradually spread to the top of the canopy. It is not yet fully clear whether this is caused by higher susceptibility of older leaves, better

microclimatic conditions for infection in the lower canopy or more deposition of sporangia low in the canopy. Björling and Sellgren (1955) found 2-4 times as many sporangia deposited on middle and bottom leaves as on top leaves, both in incipient and well-developed epidemics. Few models simulate both host growth and the vertical distribution of the pathogen over leaf layers (Waggoner, 1990). The vertical distribution of barley powdery mildew is included in the model EPIGRAM (Aust et al., 1983), in which upper leaves are taken to be the most resistant to the pathogen. However, the information is not used to calculate crop photosynthesis and growth: EPIGRAM requires seasonal courses of leaf area as input. The model can therefore only be used to describe experimental results, extrapolation to unmeasured circumstances or host genotypes is impossible. Host growth has been incorporated, without considering the vertical disease distribution, in epidemiological models that assume the leaf area to grow according to a logistic function of the undiseased leaf area (Berger and Jones, 1985; van Oijen, 1989). Waggoner (1990) gives equations for crop photosynthesis when the disease is horizontally or vertically heterogeneously distributed. These equations may conveniently be used as submodels for host growth in models of pathosystems.

A further object of study for component models is the relationship between host size and disease escape. Most models show that whenever the susceptible leaf area decreases below a certain threshold, the area of infectious tissue can no longer increase, and that some susceptible tissue will remain uninfected when the epidemic has ended (Kermack and McKendrick, 1927; Vanderplank, 1963). The magnitude of this disease escaping fraction of the host area depends on a nonlinear function of all components that influence the epidemic rate, thus pointing to a close link between host characteristics determining escape and resistance (van Oijen, 1989).

Initialization and parameterization

When the performance of epidemiological models is tested, the status of the initial inoculum is rarely paid attention to. Jeger (1986) indicates that model epidemics starting from a number of equally aged latent lesions may differ strongly from epidemics started from infectious lesions, if the model used has a distributed *LP*. The initialization of models with time delays, as the paralogistic equation of Vanderplank (1963), is further complicated by the necessity to define the level of disease for some period of time (equal to the maximum delay in the model) preceding the simulated period.

Parameterizing epidemiological models can be difficult due to the bad correspondence of model parameters and variables with actually measured quantities. Methods of measuring resistance components usually aim at maximum discrimination

between cultivars or treatments, or ease of measurement. For modelling purposes, however, components should be measured in a way that corresponds best to their function in the life cycle of the pathogen. Measurements of lesion growth rate (*LG*) are more useful than measurements of lesion size at an arbitrary time after inoculation. Determining the total sporangium production per unit of area of diseased leaf tissue is better than the more common practice of quantifying sporulation as number of sporangia washed off per leaflet or lesion, again at arbitrary times after inoculation. Only measurement of *SI* on an area basis allows a clear separation of *SI* from lesion size (or integrated *LG*) and *IP* in fungal diseases with expanding lesions.

Disease progress rate is generally quantified differently in measurements and component models, thus complicating model validation. Total diseased tissue is measured, while latent, infectious and removed tissue are modelled. Models with a fixed size for all lesions, including latent ones, give special problems here, since in actual epidemics latent lesions occupy much less leaf area than visible lesions. The paralogistic equation of Vanderplank (1963), for example, calculates the total infected (latent + infectious + removed) leaf area, whereas only infectious and removed leaf area can be observed. This is a further argument in favour of models that include *LG*, where latent lesions can realistically have zero or negligible area.

Because of the bad correspondence of measurements with model parameters, most data from the literature (Table 4.1) are unsuited for a component model of potato late blight. Exceptions are the values of *IE* from James and Fry (1983), *LP* from Lapwood (1961b) and Jeffrey et al. (1962), *LG* reported by Gees and Hohl (1988) and Colon and Budding (1990), and *IP* and *SI* from Lapwood (1961b).

Models of *P. infestans*

The model of potato late blight by Michaelides (1985) has been discussed above. Waggoner (1968), Bruhn and Fry (1981) and Stephan and Gutsche (1980) also published models of *P. infestans* with some resistance components, though never *LG*, but did not use their models to study the role of the components. The special epidemiological role of stem lesions as survival mechanisms under adverse weather conditions of *P. infestans* in potato, has been incorporated in a forecasting model of Sparks (unpublished). This feature seems of little importance for models that focus more on differences between cultivars than on the effect of weather conditions.

Discussion and conclusions

Neither experimental nor modelling studies have conclusively shown which components most effectively reduce disease progress rate in fungal leaf diseases. Whereas the results of experimental components analyses yield contradictory results due to differences in genotypes tested and trial conditions applied (Chapter 4), the modelling studies have suffered from inadequate parameterization and unsufficient validation. The models, however, might be put to better use. Too often a sensitivity analysis with regard to resistance components is absent (Jeger and Groth, 1985), although the usefulness of such an analysis for the guidance of breeding efforts has been emphasized repeatedly (Zadoks, 1971, 1977). Whenever such a sensitivity analysis is indeed present, the analysis is usually restricted to one-parameter changes. Simultaneous changes of more than one resistance component should be evaluated too. These multi-parameter changes should take into account that some parameters may not vary independently in the real pathosystem, if they are genetically or physiologically linked. *LG* should be included in models of fungal leaf diseases with indefinitely expanding lesions. The importance of this component has been shown by the few models that do include it: disease progress rate calculated by these models shows great sensitivity to *LG*. If a disease spans a long period of the host growing season, which applies to most fungal leaf diseases, especially when partially resistant genotypes are evaluated, the disease is incorrectly simulated by models that consider the host leaf area as constant. For such diseases the effect of the pathogen on host growth should be explicitly modelled if the model is to be used for analysing effects of host characteristics on yield loss caused by the disease.

Because of the reasons mentioned above the following model analyses of potato late blight have been undertaken. 1) A model of the pathosystem that incorporates host growth and all major resistance components, including *LG*, was used to assess the effects of host growth, resistance and tolerance characteristics on yield loss (Chapter 6). 2) The effect of simultaneous changes of multiple resistance components was determined (Chapter 7). 3) A more detailed model, with foliage stratification to allow simulation of a vertically heterogeneous disease distribution was used to test hypotheses about the mechanisms underlying genotypic differences in the dynamics of profiles of late blight within potato crops (Chapter 8).

CHAPTER 6

Evaluation of breeding strategies for resistance and tolerance to late blight in potato by means of simulation modelling

Abstract

A field experiment with three potato cultivars, where plants were inoculated with *Phytophthora infestans*, was used to parameterize a model of potato growth and blight population dynamics. The model was validated by accurately simulating another field experiment with the original parameter settings. Sensitivity analysis with the model showed that late cultivars are longer able to maintain a green canopy in the presence of disease, but still suffer more yield loss than early cultivars. The level of partial resistance of a cultivar was more important than its level of tolerance, and other plant characteristics. The model calculations showed that only between 4 and 15% of the yield loss in the experiments was due to accelerated leaf senescence caused by the disease; the major part of the loss was caused by lesion coverage of leaves.

Introduction

Yield loss caused by *Phytophthora infestans* (Mont.) de Bary varies between potato cultivars. The variation is caused by different rates of spread of the pathogen through the crops, and by differences in crop response to the presence of the disease. In two previously reported field experiments, carried out in 1987 and 1988 (Chapters 1 and 3) with three cultivars, differing in maturity class and level of partial resistance, measurements were made of seasonal courses of foliage and tuber growth, leaf senescence and coverage of leaves by blight lesions. These measurements were used in the present chapter to explain the differences in yield loss between the cultivars, and to determine the plant characteristics that have the greatest influence on loss. To achieve these goals a simple simulation model was constructed, that includes the growth of both the host crop and the pathogen population. The model was parameterized on the basis of the 1987 experiment and validated using the experiment of the following year. The model was subjected to a sensitivity analysis, focussing on the effects of plant characteristics on yield loss.

Model structure and parameterization

The model was constructed by combining the potato crop growth model LINTUL (Spitters and Schapendonk, 1990) and the late blight population dynamics model BLIGHT (van Oijen, 1989). The most essential features of these models are given below.

Crop growth in LINTUL is linearly related to light interception, which has an asymptotic relationship with the Leaf Area Index (*LAI*). The partitioning of growth between leaves, stems, roots and tubers is governed by the stage of crop development which is calculated from the temperature sum (°C d) and the maturity class of the cultivar. Early cultivars initiate tuber growth at a lower temperature sum than late cultivars, at the expense of foliage and root growth. Leaf area is calculated by multiplying leaf weight with a constant Specific Leaf Area (*SLA*).

The growth of the pathogen population is modelled spatially homogeneously in BLIGHT. Only lesions on leaves are considered; stem lesions are not included because of their negligible effect on leaf loss (Chapter 3). The density of leaf lesions and their size distribution change dynamically. These processes are controlled by the amount of available host leaf area and by five parameters: infection efficiency (*IE*), latent period (*LP*), lesion growth rate (*LG*), sporulation intensity (*SI*) and sporulating period (*IP*). These parameters, which determine the level of partial resistance of a cultivar, are called 'resistance components'. Acceleration of leaf senescence caused by the disease is included in the model. At any time during the epidemic, disease severity, expressed as the percentage of lesion covered leaf area, is assumed to cause an equal percentage of leaf senescence of the non-lesion covered leaf area (Chapter 3).

Leaf lesion coverage and accelerated leaf senescence caused by late blight start in the bottom leaf layers and gradually move upwards in the canopy (Chapter 3; Lapwood, 1961c). Therefore blight reduces total light interception mainly by reducing the area of green leaves, not by causing overshadowing of green leaves by lesion-covered or senesced leaves. The Light Use Efficiency (*LUE*), which relates crop biomass to the amount of light intercepted by green leaf area, is not reduced by the disease (Chapter 1; Haverkort and Bicamumpaka, 1986). Therefore the interaction of host and parasite is modelled simply by calculating the dynamics of loss of green leaf area caused by pathogen spread and accelerated leaf senescence.

In the experiments used for model parameterization and validation (Table 1.1: Experiment 1 and 2, respectively), epidemics were initiated artificially by spraying inoculum over plots about one month after emergence. The model thus assumes homogeneous input of inoculum on the date of inoculation (Table 6.1).

The experiments were done with three cultivars: the early susceptible cv. Bintje, the

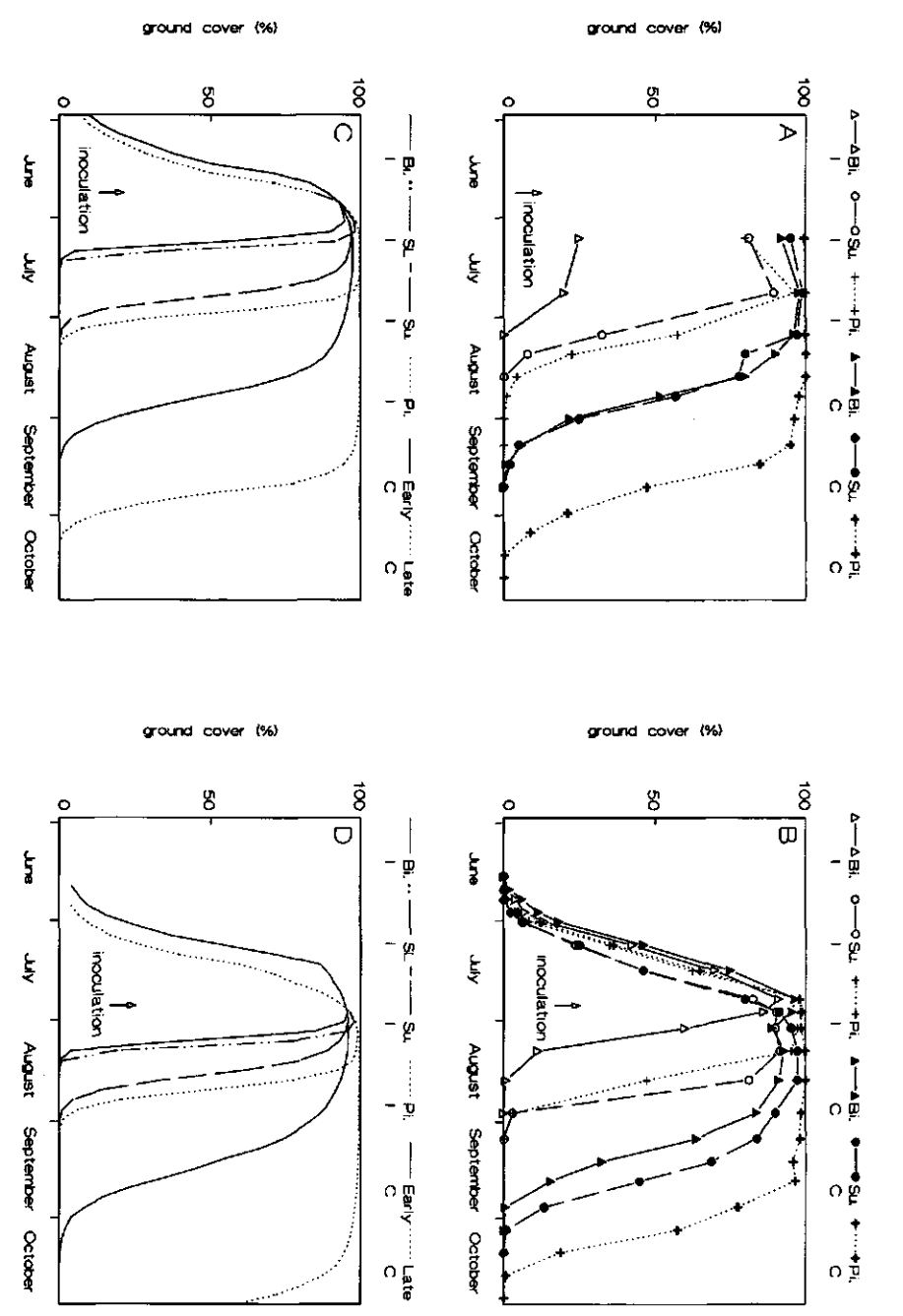
Table 6.1. Complete listing of model parameter settings and inputs that were different for simulations of different cultivars (early/late, susceptible/resistant) or years (1987/1988).

Maturity class:		
Onset of tuber filling (°C d)	Early: 150.7	Late: 234.5
Leaf senescence rate	[1]	[1]
Level of partial resistance:		
Infection efficiency (*IE*, %)	Susc.: 2.4	Res.: 1.2
Lesion growth rate (*LG*, m d^{-1})	Susc.: 0.003	Res.: 0.0015
Year:		
Seasonal course of temperature	1987: measured daily	1988: measured daily
Seasonal course of light	1987: measured daily	1988: measured daily
Date of inoculation	1987: June 23	1988: July 27
Inoculum density (sporangia m^{-3})	1987: 4. x 10^{6}	1988: 4. x 10^{7}
Light use efficiency (*LUE*, g MJ^{-1})	1987: 2.95	1988: 2.35
Maturity class x Year:		
Date of emergence (early cvs)	1987: May 19	1988: June 18
Date of emergence (late cvs)	1987: May 23	1988: June 24

[1] The relation between leaf senescence rate and the actual temperature, the temperature sum and the maturity class was given by Spitters and Schapendonk (1990).

early resistant cv. Surprise and the late resistant cv. Pimpernel. In the simulations these three cultivar types were examined and a hypothetical late susceptible one. Cultivar earliness or lateness was parameterized by a maturity class of 6.5 or 3.5, respectively (Spitters and Schapendonk, 1990). This causes the late cultivars to show a marked delay in tuber filling in favour of foliage growth (Jones and Allen, 1983).

Cultivar resistance level was parameterized by means of the resistance components. These were not measured in the experiments. The component parameters of susceptible cultivars were assumed to have the most 'susceptible' value reported in the literature (as in van Oijen, 1989). For the resistant cultivars the values of *IE* and *LG* were set 50% lower, which is still in the range of observed genetic variation for these components (Table 6.1). The model parameters that represent the *LAI* at emergence and the efficiency of inoculum dispersal were also not assessed in the experiments. These parameters were set at values that caused the best agreement between simulations and measurements of host leaf area dynamics and pathogen population growth in cvs Bintje and Pimpernel in the 1987 experiment (Chapter 1: Experiment 1). Although in 1988 planting was much delayed (1 June 1988 vs. 29 April 1987), leading to


A
B
C
D
ground cover (%)
0
50
100
June
July
August
September
October
inoculation
Bi.
Su.
Pi.
C
Early
Late

← Fig. 6.1. Measured and simulated time courses of ground cover in inoculated (I) and control (C) plots of the susceptible early cv. Bintje (Bi), the resistant early cv. Surprise (Su), the resistant late cv. Pimpernel (Pi) and a hypothetical susceptible late cultivar (SL). Note: only the maturity class affects the simulated time courses in control plots, lines for susceptible and resistant cultivars fall together. A: Measurements 1987; B: Measurements 1988; C: Simulations 1987; D: Simulations 1988.

a shorter growing season and lower yields, the 1988 experiment was simulated with the parameter settings derived for the 1987 experiment. Only inoculum density was set ten times higher, to account for better infection conditions in 1988 because of previous sprinkler irrigation, and the *LUE* was lower, in accordance with the experimental results (Chapter 1) (Table 6.1).

Table 6.2. Comparison of cumulative light interception (*PARCUM*) and yield of tuber dry matter in field measurements and simulations.

Year	Cultivar	Treatment	*PARCUM* (MJ m^{-2})		Yield (t ha^{-1})	
			Measured	Sim.	Measured	Sim.
1987	Susc./Early [1]	Control	530	515	11.82	11.51
		Inoculated	242	211	2.87	2.88
	Susc./Late	Control		666		13.93
		Inoculated		216		1.76
	Res./Early [2]	Control	512	515	9.48	11.51
		Inoculated	363	342	5.32	6.41
	Res./Late [3]	Control	672	666	13.97	13.93
		Inoculated	401	360	4.72	4.92
1988	Susc./Early [1]	Control	403	448	7.79	7.92
		Inoculated	174	212	1.95	2.44
	Susc./Late	Control		554		8.98
		Inoculated		192		1.22
	Res./Early [2]	Control	404	448	7.74	7.92
		Inoculated	270	298	4.19	4.40
	Res./Late [3]	Control	502	554	8.66	8.98
		Inoculated	279	299	3.16	3.03

[1] Measurement data for cv. Bintje.
[2] Measurement data for cv. Surprise.
[3] Measurement data for cv. Pimpernel.

Results

Experimental data for the seasonal courses of percentage ground cover by green leaves, averaged over the cultivars, have been presented earlier (Chapter 1). For ease of comparison with simulation results the data for the inoculated and control treatments of the separate cultivars are shown here (1987: Fig. 6.1A; 1988: Fig. 6.1B). In 1987 ground cover in the controls decreased earliest in the early cvs Bintje and Surprise, while in the inoculated plots the blight susceptibility of cv. Bintje caused an early foliage death (Fig. 6.1A). Results were similar in 1988, except that cv. Surprise slightly outlasted cvs Pimpernel and Bintje in the control and inoculated treatments, respectively (Fig. 6.1B).

Seasonal courses of ground cover, cumulative light interception during the growing season and final tuber yields were simulated very well for the 1987 experiment (Fig. 6.1C; Table 6.2). The only exception was the overestimation of tuber yield of the control plots of cv. Surprise, in spite of an accurate value for cumulative light interception (Table 6.2).

When the same parameter settings were applied to the 1988 experiment, the agreement between simulations and measurements was again good for most cultivar types and treatments (Fig. 6.1D; Table 6.2). However, the green leaf area duration of late cultivars was overestimated in the absence of disease (Fig. 6.1D), compared to the ground cover measurements of the control plots of cv. Pimpernel (Fig. 6.1B), but yield was estimated well (Table 6.2).

After simulating the two experiments, the model was used to assess the influence of different plant characteristics and experimental conditions on yield loss. The characteristics and conditions included in this sensitivity analysis are listed in Table 6.3.

Maturity class and partial resistance. Lateness leads to a slightly longer green leaf area duration in blighted crops, both for resistant and susceptible cultivars, although resistance is the more important trait for prolonging the growing season (Figs 6.1C, D). Yield, on the other hand, is lowest in blighted late cultivars, because of a later onset of tuber filling (Table 6.2).

Growth characteristics. Changing plant growth characteristics generally has little effect on yield of blighted crops (Table 6.3), except for increasing the relative rate of leaf growth during the early exponential phase (up to LAI=0.75), which may increase yields considerably. Increasing SLA, which increases the light intercepting leaf area without reducing dry matter allocation to the tubers, has a less positive effect. Changing assimilate distribution such that tuber filling starts earlier, but at a slower rate, while leaf growth continues simultaneously for a longer period (as found in cv. Désirée; Spitters and Schapendonk, 1990), also increases yields, but only in blighted cultivars of the

Table 6.3. Simulated tuber yields of blighted crops of four potato cultivar types, and their response to variation in plant attributes and experimental conditions. Yields in percentages of the reference simulated yields for the inoculated treatment, averaged for 1987 and 1988, given in Table 6.2. The multiplication factor was used to alter the specified parameters with respect to the reference.

	Multiplication factor	Susc. Early	Susc. Late	Res. Early	Res. Late
Reference yield (=100%) (t ha^{-1})		2.66	1.49	5.41	3.98
Growth characteristics:					
1. Early leaf growth	1.2	115	111	112	108
2. Assimilate distribution	[1]	99	121	96	105
3. Specific Leaf Area	1.2	107	108	107	106
4. Leaflet area	1.2	99	100	100	100
Tolerance component:					
1. Accelerated senescence	0.0	117	126	112	120
Resistance components:					
1. Lesion growth rate (*LG*)	0.8	115	124	115	121
2. Infection efficiency (*IE*)	0.8	108	112	108	111
3. Sporulation intensity (*SI*)	0.8	104	107	104	107
4. Latent period (*LP*)	1.2	104	106	103	104
5. Infectious period (*IP*)	0.8	103	104	104	106
Experimental conditions:					
1. Day of inoculation	+ 30 [2]	295	408	174	219
2. Inoculum density	0.001	206	240	174	209

[1] Onset of tuber filling times 0.6, slope of allocation to tubers times 0.8.
[2] Inoculation postponed by 30 days.

susceptible late type. Increasing the area of individual leaflets, which does not affect the total leaf area while allowing blight lesions to continue growth longer before reaching the edge of the leaf, hardly increases the epidemic rate and yield loss.

Tolerance to acceleration of leaf senescence. When acceleration of leaf senescence was neglected, yields of inoculated plots increased by 12 to 26% (Table 6.3). This corresponds to leaf senescence accounting for 4 to 15% of yield loss caused by blight in the experiments of 1987 and 1988. The remaining, major fraction of the yield loss thus was caused by direct leaf loss because of lesion extension.

Resistance components. As reported earlier (van Oijen 1989), the radial growth rate of lesions (*LG*) is the component that affects yield loss the most (Table 6.3).

Experimental conditions. When inoculation was postponed thirty days, or inoculum density reduced by a factor of one thousand, the yield of late cultivars benefited the most

(Table 6.3).

Discussion

Yields in the 1988 experiment, where planting was delayed, were very low (Table 6.2). However, they were accurately modelled for all cultivars in both treatments, using the parameter settings derived for 1987 except for the *LUE*. The succesful simulations of host growth and epidemic development in the two years indicate that differences in yield loss caused by *P. infestans* may fully be explained by the incorporated differences between cultivars in partial resistance (*IE* and *LG*) and maturity class-dependent rates of leaf growth, leaf senescence and timing of tuber growth initiation.

The green leaf area duration of control plants of late cultivars, such as Pimpernel, was overestimated by nearly a month for 1988 (Fig. 6.1D compared to 6.1B). This extra month of crop growth caused only a small overestimation of light interception and yield (Table 6.2) because of the low input of light at the end of the season. In 1988, the crops were planted at June 1, while the relation between crop development stage and temperature sum that was used in the model was determined for crops planted in April (Spitters and Schapendonk, 1990). The late planting may have caused foliage death at lower temperature sums than usual, especially for the control plants of late cultivars which reached into periods with much shortened daylengths and colder nights. Crop phenology of these cultivars may thus have been simulated poorly, causing the overestimation of leaf area duration.

Growth and tuber yield of cv. Surprise were simulated well for both years and treatments, except for the unexplained overestimation of yield in the control treatment in 1987 (Table 6.2).

The simulations show earliness to be an advantage in reducing yield loss (Table 6.2). Early cultivars escape part of the epidemics by completing a greater fraction of their tuber filling period before the disease causes premature foliage death. Therefore an altered assimilate distribution pattern, in which tubers are initiated earlier but leaf growth continues longer, simultaneously with tuber filling, increases the yield of blighted late cultivars (Table 6.3). However, in the case of mild epidemics, either initiated by lower levels of inoculum or by a later inoculation date, the yields of late cultivars are increased more than those of early cultivars (Table 6.3). Inoculum density thus affects the differences in yield between late and early cultivars. Therefore breeders should take into account the natural inoculum density of *P. infestans*, under actual potato growing conditions, when defining the required resistance levels of cultivars differing in maturity class.

The simulations show that late cultivars suffer more yield loss than early cultivars, when their levels of blight resistance are equal (Table 6.2). Experiments, on the other hand, often show a positive correlation between cultivar lateness and resistance (Umaerus et al., 1983). A possible explanation for this apparent contradiction may be that late cultivars have been subjected to a stronger selection pressure, in previous resistance breeding work, precisely because of their low yields in the presence of blight. Another explanation may be that resistance has erroneously been equated to ground cover, which indeed is maintained longer by late cultivars (Figs 6.1C, D). Ground cover or disease severity may only be useful as selection criteria for groups of genotypes of similar maturity class, but even then genotypes with uncommon patterns of assimilation distribution, such as cv. Désirée, may be wrongly assessed. Therefore measurement of resistance components is preferable (van Oijen, 1989).

Acceleration of leaf senescence by the disease was shown to have caused 4 to 15% of the yield loss. However, no genetic variation for this aspect of tolerance has been found among the cultivars used (Chapter 3). Photosynthesis and *LUE* are not affected by the disease in any of the cultivars (Chapters 1 and 2). Since, furthermore, other plant growth characteristics affect yield loss only little (Table 6.3), screening for increased levels of components of partial resistance, particularly *IE* and *LG* (van Oijen, 1989), is the best breeding strategy aiming at reduced yield loss caused by late blight.

CHAPTER 7

Evaluating components of resistance to *Phytophthora infestans* in potato, using mathematical models of general epidemics

Abstract

Five models of general epidemics, spatially homogeneous, were all shown to fit well to disease progress data for *Phytophthora infestans* on a susceptible potato cultivar. The models were: the logistic equation, the paralogistic or Vanderplank equation, two models from medical epidemiology with similar complexity, and a slightly more complex model with explicit treatment of lesion expansion. The use of the models for analysing the sensitivity of disease progress to changes in resistance components is discussed. Sensitivity analysis of the most complex model, within the range of available genetic variation for resistance components, indicates lesion expansion and infection efficiency as the components offering the best perspectives for resistance breeding. Improving two components simultaneously is shown to act slightly stronger than additively on the restriction of disease progress, although not enough to add other components to the list of breeding objectives. Pitfalls in using models for component sensitivity analysis, in the form of erroneous model initializations, are discussed, including implications for the role of components in the development of natural epidemics and in resistance breeding trials.

Introduction

Damage to crops by disease may be reduced by using completely or partially resistant cultivars that reduce pathogen build-up. Because of the swiftness with which most pathogens adapt to newly introduced completely resistant cultivars, partial resistance is nowadays favoured above complete resistance in most resistance breeding programmes, including those for potato late blight (Parlevliet, 1979; Umaerus et al., 1983).

Partial resistance consists of several components, each affecting a different stage in the life cycle of the pathogen (Parlevliet, 1979). Zadoks (1977) distinguishes five components that determine the development of epidemics and may be affected by the host plant, and thus can be used in breeding: infection efficiency (*IE*), latent period (*LP*),

lesion growth rate (*LG*), infectious period (*IP*) and sporulation intensity (*SI*). To determine the relative contributions of these resistance components to the reduction of disease progress, two approaches are generally used (Jeger and Groth, 1985). The first approach comprises the experimental determination of correlations between individual resistance components and disease progress rate. This requires extensive field experimentation using many genotypes, since no isogenic lines exist that differ for one resistance component alone. For the potato - *P. infestans* pathosystem only preliminary analyses of this nature have been performed (e.g. Pietkiewicz, 1976). Therefore, even though many studies about components of resistance to potato late blight have by now been published, it is still unclear which component has the greatest effect on disease progress. The second approach for evaluating components comprises the construction of a mathematical model of the pathosystem, and a sensitivity analysis with this model. By this, the response of yield or disease progress rate to changes in resistance component parameters is assessed.

In the present chapter some of the models most frequently used for analysing epidemics are compared. The comparison is restricted to simple models, without host growth or environmental effects on parameters. The model of which the structure most closely corresponds to the potato late blight pathosystem is used for evaluating the role of the components of resistance. The extent to which the component evaluation may be influenced by differences in model initialization, is studied in a final section.

The study presented in this chapter is of a theoretical nature: models are compared and analysed. Comparisons of simulations of potato late blight with experimental data are presented in Chapter 6.

Comparison of the structures of the different models

In resistance breeding trials, artificial inoculation is applied on relatively small plots. The resulting epidemics, developing without a strong spatial heterogeneity, are called 'general epidemics' (Zadoks and Schein, 1979). In this paragraph, five simple models of general epidemics are compared with regard to their suitability for analysing the role of resistance components in breeding trials.

Many plant disease progress curves can be described by the simple logistic equation. In this equation the transition of tissue from susceptible (S) to infectious fractions of the leaf area (I = 1-S) is directly proportional to both S and I (Table 7.1). In the terminology of Hethcote (1976) such epidemiological models with S- and I- categories are called SI-models.

Since infectious leaf area in reality only remains infectious for a limited time (the

Table 7.1. Models of general epidemics.

Model	Type [1]	Equations [2]		Initialization	
logistic	SI	S	= 1 - I		
		dI/dt	= k x S x I	I(0)	= I_0
General Epidemic M.	SIR	S	= 1 - I - R		
		dI/dt	= k x S x I - I/IP	I(0)	= I_0
		dR/dt	= I/IP	R(0)	= 0
Extended General Epidemic Model	SEIR	S	= 1 - E - I - R		
		dE/dt	= k x S x I - E/LP	E(0)	= 0
		dI/dt	= E/LP - I/IP	I(0)	= I_0
		dR/dt	= I/IP	R(0)	= 0
paralogistic	SEIR	S	= 1 - y		
		E	= y(t) - y(t-LP)		
		I	= y(t-LP) - y(t-LP-IP)		
		R	= y - E - I		
		dy/dt	= k x S x I	y(t)	= f(t), -LP-IP ≤ t ≤ 0
BLIGHT	SIR	S	= 1 - I - R		
		dI/dt	= $L_i$xda/dt + axdL_i/dt - I/IP	I(0)	= 0
		R	= L_i x a - I		
		dL_E/dt	= k x S x I - L_E/LP	L_E(0)	= L_{E0}
		dL_i/dt	= L_E/LP	L_i(0)	= 0
		da/dt	= f(LG) [3]	a(0)	= 0

[1] Model typification according to Hethcote (1976). SI models include susceptible (S) and infectious (I) leaf area. SIR and SEIR models add removed (R) and latent (E) leaf area.
[2] Abbreviations of leaf area fractions: S: susceptible; E: latent; I: infectious; R: removed. Abbreviations of resistance components: IP: infectious period; LP: latent period; LG: radial lesion growth rate; k: infection rate. Abbreviations of lesion variables: L_E: latent lesion density; L_i: sporulating lesion density; a: average lesion area.
[3] For the derivation of the function relating lesion area increase to lesion radius increase, see van Oijen (1989).

infectious period, IP), an extra category of removed leaf area (R = 1-S-I) can be defined, that can no longer become infected or cause infection. Such models are SIR-models. If the transition rate from I to R is taken to be directly proportional to I only, we get the General Epidemic Model, first published by Kermack and McKendrick in 1927 (Table 7.1).

Again more realistic are SEIR-models which include latently infected leaf area (E = 1-

S-I-R) that has been exposed to infection, but will only become infectious after a latent period (*LP*). SEIR-models used in medical epidemiology assume that the rates of transition for E-I and I-R are directly proportional to E and I, respectively, while in plant disease models the rates of these transitions generally equal the rate of S-E at times *LP* and *LP+IP* earlier. SEIR-models are thus formulated as a set of continuous differential equations, the Extended General Epidemic Model, for human diseases (Anderson and May, 1982), and as a time delayed differential-difference equation, the paralogistic equation, for plant diseases (Vanderplank, 1963) (Table 7.1).

Berger and Jones (1985) have indicated that a further resistance component, lesion expansion, is needed in models of diseases such as late blight, where lesions grow indefinitely without reaching a predetermined final size. Radial lesion growth rate (*LG*) has been added to the SEIR-model by van Oijen (1989) using a dynamically changing frequency distribution of lesion sizes. In this model, BLIGHT, lesion growth and sporulation start at the same time after infection (the latent period, *LP*), so that latent lesions occupy no leaf area and the model is simplified to an SIR-model (Table 7.1).

The five models discussed represent the life cycle of the pathogen with increasing comprehensiveness. Therefore more resistance components can be studied with the later models, at the cost of increased data demand for parameter estimation. None of the models includes all components distinguished by Zadoks (1977). The infection rate parameter *k*, however, which appears in every model (Table 7.1), is the product of sporulation intensity (*SI*), spore dispersal efficiency and infection efficiency (*IE*). This parameter thus combines three processes of which especially dispersal is difficult to quantify. Therefore in general only the remaining components are measured, while *k* is quantified by fitting the models on measured disease progress curves.

Another criterion for model usefulness, apart from the number of components, is whether the components are quantified in a way that corresponds to how they are most easily and reliably measured. Therefore in BLIGHT lesion expansion was quantified as radial growth rate of individual lesions, this being for potato late blight a more constant measure than relative or absolute growth rate of lesion area (Gees and Hohl, 1988; van Oijen, 1989). *LP* was defined, in all models that included it, as it is generally measured: time between infection and sporulation. *IP*, on the other hand, is generally measured as the duration of sporulation of lesions, while for late blight, where only the shifting outer edges of lesions sporulate, the measure used in the models is the much shorter duration of sporulation of infected tissue. *IP* 'per lesion' would be an inconvenient model parameter, being by definition negatively correlated with *LG*, since lesions stop sporulating shortly after they have outgrown the leaf area available to them. For *IP* 'per tissue' the genetic variation is small (Lapwood, 1961b; Vanderplank, 1963).

Table 7.2. Data of measured genetic variation for resistance components, and settings of the corresponding parameters in epidemiological models. Note: not all components are represented in every model.

		k [1] (d^{-1})	IP [1] (d)	LP [1] (d)	LG [1] ($m\ d^{-1}$)
Data	field	6.4-8.5x10^{6} [3]	0.75-1 [4]	4-5 [4]	0.001-0.003 [5]
	laboratory	0.26-2.40 [3]			
Models	logistic	0.19	-	-	-
	General Epidemic	0.51	1	-	-
	Extended Gen. Epidemic	2.50	1	4	-
	paralogistic	2.75	1	4	-
	BLIGHT	765 [2]	1	4	0.003

[1] Abbreviations of resistance components as in Table 7.1.
[2] In BLIGHT, the infection rate parameter k measures the increase in lesion density instead of the increase in infected leaf area, and is therefore expressed as lesions $m^{-2}\ d^{-1}$.
[3] k is not a directly measurable component: the field data refer to variation in sporulation intensity (SI; sporangia $m^{-2}\ d^{-1}$) of 4 genotypes (Lapwood, 1961b), the laboratory data refer to infection efficiency (IE; %), also of 4 genotypes (James and Fry, 1983).
[4] 4 genotypes (Lapwood, 1961b).
[5] 3 genotypes (L.T. Colon, pers. comm., 1988).

Components analysis using the different models

Data are available of variation between potato genotypes in components of resistance to *P. infestans* (Table 7.2; van Oijen, 1989). These data were used to fit the different models to a disease progress curve measured in a field trial with the susceptible potato cv. Bintje, where the percentage foliage disease had been recorded weekly (Fig. 7.1A). IP, LP, and LG, if included in the model, were kept at the most 'susceptible' value reported, while k was varied to achieve optimal fit. For each model, the resulting parameter settings (Table 7.2) caused good correspondence with the field data ($r^2 >$ 0.99; Fig. 7.1A). The model curves are not identical. Disease severity approaches 100% in the logistic equation and with BLIGHT, but with the other models a lower asymptotic value is reached. In these latter models the infectious leaf area (I) can be shown to decrease whenever the remaining susceptible leaf area (S) drops below the threshold value of $k^1 \times IP^1$ (Anderson and May, 1982). A corollary of this threshold theorem states that partly covering the crop with fungicide (i.e. reducing S), or increasing the level of

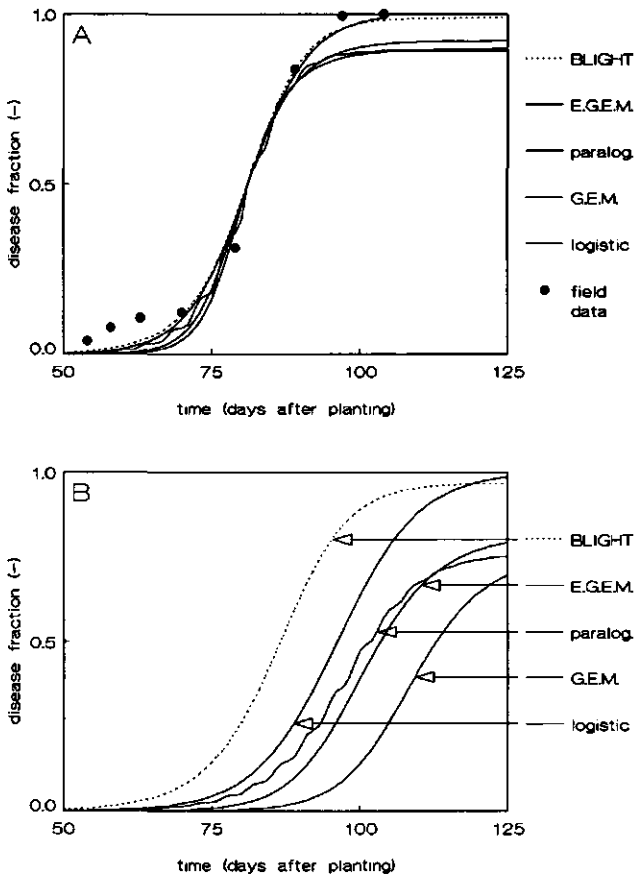


Fig. 7.1. Disease progress curves generated by five models. A: Best fit of the models on disease progress data of *Phytophthora infestans* on potato cultivar Bintje, field measurements 1988; B: Effect of reducing the infection rate parameter *k* by 25%.

partial resistance by decreasing *k* or *IP*, may suffice to prevent epidemics (van Oijen, 1989).

To assess the differences between the models in their response to changes in components, *k* was subsequently reduced by 25% in every model. This caused the time needed to reach a disease severity of 50% ($t50_y$) to be increased differently in the different models (Fig. 7.1B). The most complex model, BLIGHT, showed an increase in $t50_y$ of 6 days, while the other models showed increases of 15-32 days. Apparently the simpler epidemiological models, that possess fewer resistance components, may fit disease data equally well as the more complex models, but show greater sensitivity to

changes in the components they do have. Therefore, simplifying models should be justified by extensive experimentation.

Components analysis with various numbers of components changed

The influence of the resistance components on disease progress was studied using BLIGHT. The sensitivity analysis of BLIGHT started with the parameter settings given in Table 7.2, belonging to the disease progress curve included in Fig. 7.1A, the 'standard curve'. Deviations from the standard curve were evoked in three ways: 1 changing

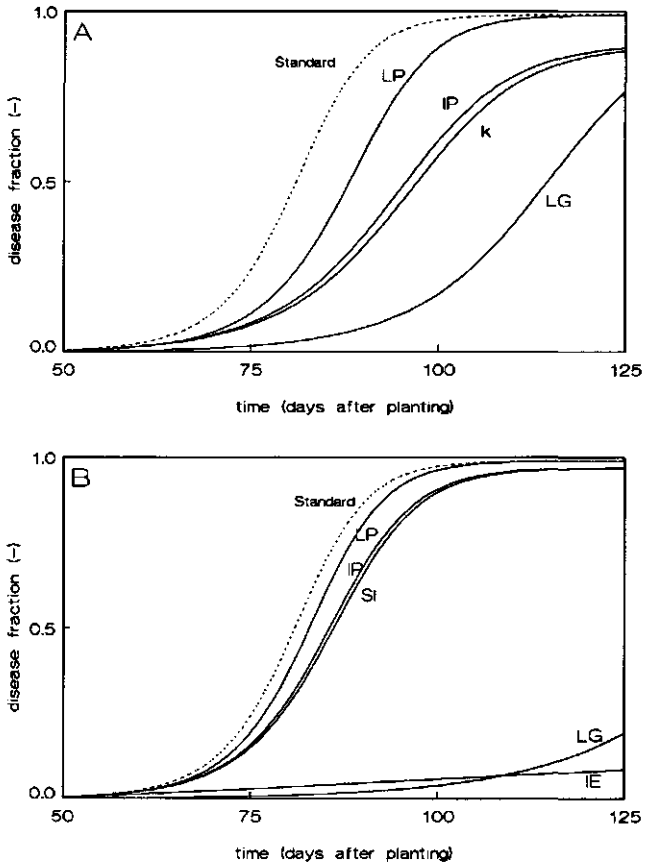


Fig. 7.2. Disease progress curves, simulated using BLIGHT. A: The 'Standard susceptible genotype' and hypothetical genotypes in which *k*, *IP* or *LG* were halved, or *LP* doubled; B: The 'Standard susceptible genotype' and genotypes in which *IE*, *SI* (both through *k*), *IP*, *LP* or *LG* were set to the most resistant value within the genetic ranges listed in Table 7.2.

individual components by 50%, 2 changing individual components according to the available genetic variation, 3 changing pairs of components.

Changing individual components by 50%. When resistance components were halved (doubled for *LP*), disease progress slowed down the most in the case of *LG*, followed by *k* and *IP*, and finally by *LP* (Fig. 7.2A). *LG* represents the radial lesion growth rate. Changing this component thus affects areal growth rate quadraticly, which explains the strong effect on disease progress. Changing *LP* has the least effect, which may be explained as follows. Lesions only sporulate on their outer edges. Whenever a leaflet has been infected and a lesion has started to grow, after latency, some time is needed before the whole leaflet is covered by the lesion and has showed sporulation. The time between leaflet infection and sporulation on a particular leaf spot thus depends on *LP*, *LG* and on the distance of the spot from the spore infection site. Thus *LP* only partly determines the time course of sporulation of a lesion and so has a minor effect on disease progress.

Changing individual components according to the available genetic variation. To account for the genetic variation in components, the parameter settings were changed from the most 'susceptible' value in the genetic range to the most 'resistant' value (Table 7.2). *IE* and *SI*, components that are lumped into *k* in the model, were evaluated by reducing *k* with equal percentages as the respective components. The sensitivity analysis showed that the reported genetic variation for *LG* and *IE* suffices for strong reductions in disease progress rate, while the genetic variation for *LP*, *IP* and *SI* affects disease progress much less (Fig. 7.2B; van Oijen, 1989).

Changing pairs of components. The effect of changing components simultaneously was studied next. Two components from *k*, *LG*, *LP* and *IP* were varied, while the remaining two were kept at maximum susceptibility (Table 7.2). For every change the increase in $t50_y$ relative to the standard curve, was calculated. For four component pairs the iso-$t50_y$ lines, combining parameter settings causing equal increases in $t50_y$, were collected in one 'life cycle sensitivity graph' (Fig. 7.3; van Oijen, 1990). As an example the simultaneous change of *IP* and *k*, such that the individual changes would increase $t50_y$ by 10 days, is emphasized in Fig. 7.3A. The combination increases $t50_y$ by 27 days. This indicates a slightly stronger than additive effect on the slowing down of disease progress. The genetic variation of the components (Table 7.2) was visualized in a similar graph by emphasizing the collection of component values that are possible if components can be varied independently (Fig. 7.3B). If, on the other hand, genetic linkage between components exists, then not all combinations of values are possible and the hatched area in the graph should have been smaller. The figure shows that the genetic variation for *LP* and *IP* is insufficient to markedly increase $t50_y$, irrespective of the

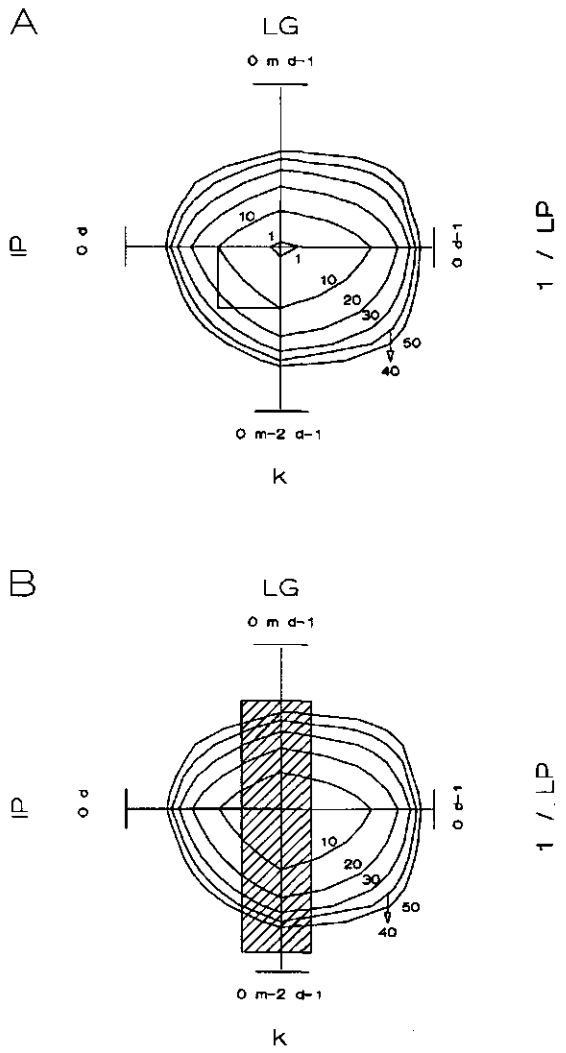


Fig. 7.3. 'Life cycle sensitivity graphs', calculated using BLIGHT. The graphs show the effect of varying different pairs of resistance components on the time of 50% foliage disease ($t50_y$). Axes indicate components, curved lines indicate the increase in $t50_y$ in days relative to $t50_y$ for the 'Standard' disease progress curve (Fig. 7.2). At the centres of the graphs, where the axes meet, all components are set at the most susceptible value within the genetic ranges listed in Table 7.2, i.e. the component parameter settings causing the 'Standard' disease progress curve. At the outer ends of the axes resistance is maximal, i.e. *LG*, *IP*, *k* or the reciprocal of *LP* is zero.
A: The example of decreasing *IP* or *k* or both by 40%. The individual changes increase $t50_y$ by 10 days whereas the combined changes increase it by 27 days. B: The genetic variation for resistance components (indicated by the hatched area).

values of *LG* and *k*. This shows that resistance is mainly determined by the level of *LG* and *k*.

Components analysis using different model initializations

The standard disease progress curve for BLIGHT (Figs 7.1A, 7.2) resulted from a low initial density of latent lesions (L_{E0} = 5 lesions m^{-2}). However, in resistance breeding trials disease is often initialized by spraying large quantities of inoculum over plots of healthy plants. Many of the lesions that are formed during the epidemic then are directly caused by this large temporary influx of external inoculum. This obscures the polycyclic nature of

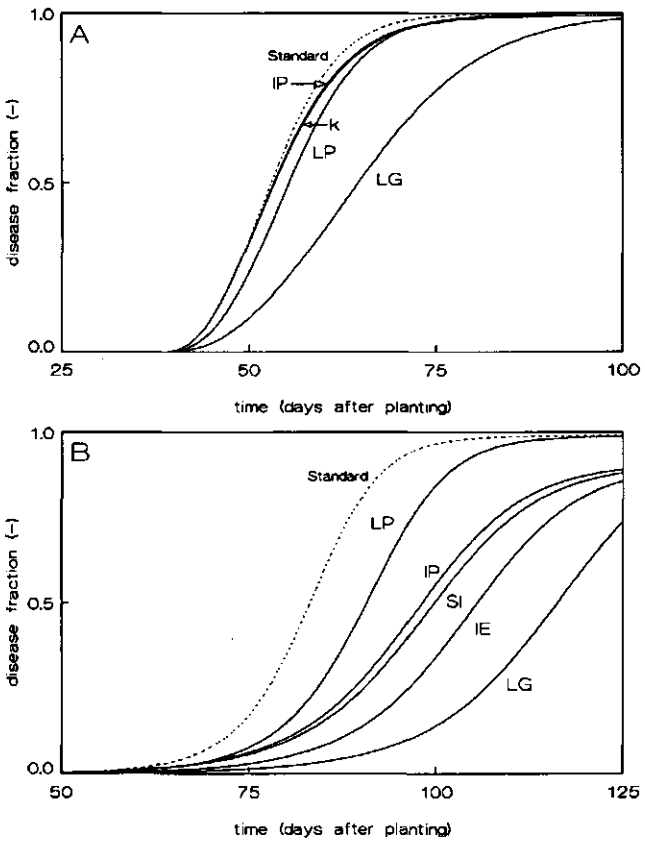


Fig. 7.4. Disease progress curves, simulated using BLIGHT. A: As Fig. 7.2A, but initial latent lesion density (L_{E0}) increased a hundredfold; B: As Fig. 7.2A, but L_{E0} set at zero, a temporary influx of external inoculum assumed, and *IE* and *SI* separately quantified.

the disease and may reduce the importance of those resistance components that affect the build-up of inoculum during later infection cycles in the epidemic. Simulations confirm this. If the initial latent lesion density is raised to 500 lesions m^{-2}, the time before these first lesions start to grow (*LP*) and the subsequent rate of growth of these lesions (*LG*) are the dominant components (Fig. 7.4A), while the components that only affect later pathogen generations (*k* and *IP*) are less important. However, the high initial lesion density reduces $t50_y$ at all component values, to the extent that differences between component effects are minimized (Fig. 7.4A).

The previous analyses started from fixed numbers of initial latent lesions. Such model initializations do not fully apply to resistance breeding trials with artificial inoculation. In such trials genotypical differences in *IE* affect the effectiveness of the inoculation itself and thus cause differences between genotypes in the density of the first generation of lesions. To determine the magnitude of this error, and to establish the importance of *IE* in resistance breeding trials with artificial inoculation, a simulation was carried out where *k* was split into *IE*, dispersal efficiency and *SI*. The model was initialized by assuming a temporary influx of external inoculum into a healthy crop. The result of the simulation (Fig. 7.4B) confirms that *IE* is more important in breeding trials than was apparent from changing *k* in the simulations with a fixed initial lesion density (Fig. 7.2A).

Discussion

The logistic and paralogistic equations are the most prominent models in plant disease epidemiology. The logistic equation is too simple to be of much use in components analysis. The more comprehensive paralogistic equation has been criticized by Jeger (1986) for its mathematical untractability and the fact that its structure, a time delayed differential-difference equation, does not correspond to the vast theory of linked differential equations in medical epidemiology. Jeger therefore recommends using the human disease SEIR-model discussed above. However, lesion growth rate was not introduced in any of these models. In BLIGHT this resistance component was introduced and it was demonstrated that it strongly affects disease progress.

The sensitivity analyses show that, while disease progress is most sensitive to *LG* (Fig. 7.2A), genetic variation for *IE* is large enough to offer equally good perspectives for resistance breeding (Fig. 7.2B). Although improving two components simultaneously may lead to slightly stronger than additive effects (Fig. 7.3A), genetic variation for *IP* and *LP* is not sufficient to warrant breeding for improvement of these components (Fig. 7.3B). If, however, genetic linkage between these components and *LG* or *IE* exists, they may still be useful for indirect selection.

In BLIGHT, sensitivity of disease progress to changes in *LP* is less than in the paralogistic equation, which was analysed extensively by Zadoks (1971). The difference is partly caused by differences in model structure, the greater sensitivity in the paralogistic being related to its smaller number of parameters, as explained above. The method of model initialization, however, also affects the outcome of sensitivity analyses. The analyses show that a high level of initial inoculum may not only alter the ranking of the components, but may also obscure differences between genotypes that would have become apparent in natural epidemics, initiated from lower levels of inoculum whereafter more disease cycles would take place (Fig. 7.2A compared to 7.4A). Differences between genotypes with respect to *IE* may be obscured if the models are initialized with fixed numbers of first generation lesions, thereby ignoring that varietal differences in *IE* also affect the effectiveness of the artificial inoculation (Fig. 7.4B).

This analysis shows that the proper use of multi-component models may help in avoiding some of the pitfalls, when evaluating the role of resistance components in breeding research. The necessity of considering lesion growth rate, the importance of studying effects of simultaneous changes of more than one component, and the need for correct model initialization, have been demonstrated. If, furthermore, the genetic variation for the different resistance components is taken into account, the main components can be identified, as were *LG* and *IE* in the case of potato late blight.

CHAPTER 8

Modelling the dynamics of late blight profiles

Abstract

A model of potato late blight was extended to simulate the dynamics of the vertical distribution of *Phytophthora infestans* over leaf layers in a potato canopy. This model was used to explain why resistant cultivars are characterized more by retarded upward spread of the pathogen than by reduced rates of spread within leaf layers. The simulation results showed that resistant cultivars probably differ from susceptible cultivars in more respects than components of resistance alone. Three resistance mechanisms were formulated, quantification of which in the model mainly affected vertical spread.

Introduction

The spatial distribution of crop disease is increasingly often taken into account in epidemiological models. Recent simulation models of *Phytophthora infestans* (Mont.) de Bary include horizontal dispersal of sporangia in order to predict interplot interference (Paysour and Fry, 1983) or to study yield loss in heterogeneously affected crops (Ferrandino, 1989). The vertical distribution within the crop has received less attention. Yield loss estimates related to the total amount of disease are of limited value as the vertical disease profile strongly determines to what extent the production capacity of the crop is affected. *P. infestans* first attacks the lower leaves that contribute little to crop photosynthesis (Chapters 2 and 3). The present chapter reports attempts to simulate the seasonal development of profiles of foliage coverage by blight lesions, as observed in 1988 on three potato cultivars (Chapter 3, Experiment 2). These simulations are intended to increase the understanding of the mechanisms that underly genotypic differences in disease dynamics.

Model description

In the model of potato growth and blight epidemiology, presented in Chapters 6 and 7, only two foliage layers were distinguished: healthy upper leaves and diseased lower

leaves. For the purpose of profile simulation this model has been modified. Multiple leaf layers are distinguished, each corresponding to a leaf area index of 0.2. With the growth of the crop new layers are added at the top. Epidemics are initiated by assuming sporangia to be deposited uniformly over the leaf layers, one month after simulated emergence. This corresponds to the artificial inoculation applied in the field experiment. Subsequent epidemic development within the crop depends on the parameter settings for the resistance components: infection efficiency (*IE*), latent period (*LP*), lesion growth rate (*LG*), infectious period (*IP*), and sporulation intensity (*SI*). Sporangia, produced by lesions at an arbitrary leaf layer, are dispersed equally to higher and lower leaf layers. The dispersal pattern is negatively exponential, causing leaves further from the source leaf to intercept less sporangia. The values of the resistance components are equal for all leaf layers, except for the value of *IE*, which is set higher for the five lowest leaf layers. This accounts for the observation that the lowest leaves are attacked first (Chapter 3), due to increased susceptibility in old leaves, or because of the higher humidity low in the canopy, which may favour infection.

Simulation results and discussion

The simulations were aimed at explaining the experimental data of lesion coverage of individual leaves that have been presented in Chapter 3 (Fig. 3.3A). For ease of comparison with simulations the datapoints are reproduced here (Fig. 8.1). The values of $t50_l$ (the number of days after inoculation before a leaf reaches 50% lesion coverage) increase with leaf position, counted from the bottom, and are higher in the resistant cvs Surprise and Pimpernel than in the susceptible cv. Bintje. The values of r_l (the logistic rate of increasing leaf lesion coverage), on the other hand, are independent of both leaf number and cultivar. Resistance thus is only expressed by a higher $t50_l$.

The resistance component parameters were initially set at the values used for cv. Bintje in the two-layer model of the previous chapters. These settings correspond to the most 'susceptible' values reported in the literature (Table 7.2). The extinction coefficient of sporangium dispersal describes the steepness of the exponential decrease of sporangium interception with distance from a source leaf. This extinction coefficient was iteratively adapted to fit the data for cv. Bintje. The dynamics of disease profiles in this susceptible cultivar were simulated reasonably well (Fig. 8.1): $t50_l$ increased with leaf number, while r_l was constant.

In subsequent simulation runs the settings of the resistance component parameters were changed to attempt reproducing the dynamics of disease profiles of the two resistant cultivars. Five runs were done, in each of which the value of a resistance

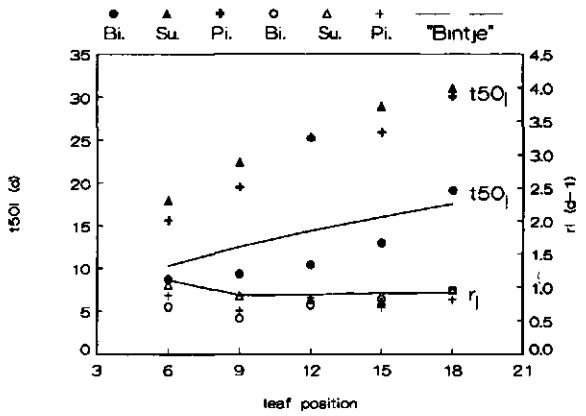


Fig. 8.1. Parameters of increasing lesion coverage, at different leaf positions counted from the bottom, of cultivars Bintje (Bi), Surprise (Su) and Pimpernel (Pi). Data for individual leaves were fitted to logistic curves, characterized by the inflection point $t50_l$ (left y-axis, closed symbols) and the infection rate parameter r_l (right y-axis, open symbols). Points: experimental data (taken from Fig. 3.3A); lines: 'Bintje'-simulations.

component was changed according to the genetic variation available for that component. This was done by setting the component parameter at the most 'resistant' value found in the literature (Table 7.2). When the value of *IE*, *LP* or *LG* was changed, $t50_l$ indeed increased, but r_l decreased to the same extent (Fig. 8.2A), contrary to the observed constancy of r_l. This unrealistic negative correlation between $t50_l$ and r_l also occurred when changing *SI* or *IP* (not shown). These model results indicate that the resistant cultivars differ from the susceptible cultivar in more respects than just one resistance component. Genotypic differences in growth characteristics as leaflet area, specific leaf area, and the rate of early leaf growth, have earlier been shown to have little effect on blight dynamics and yield loss (Chapter 6). The effects of these growth characteristics on profiles of $t50_l$ and r_l were now shown to be negligible too: the sensitivity coefficient, i.e. the percentual change of $t50_l$ or r_l divided by the percentual change of the growth characteristic examined, was less than 0.11 at all leaf positions. Therefore the cultivars probably did differ for resistance characteristics, but according to a more complex mechanism than the simple alteration of one resistance component.

The values of $t50_l$ increase with leaf position in all cultivars because the disease starts in the bottom leaf layers and gradually moves upwards in the canopy. The $t50_l$-values are higher in resistant cultivars than in susceptible cultivars, at all leaf positions (Fig. 8.1), but the r_l-values are equal. The disease thus reaches the various leaf positions at later times in resistant cultivars, but once disease is visible in a particular leaf layer, it spreads within

that layer at the same speed as in susceptible cultivars. Resistance is thus characterized by a slow vertical spread of the disease to higher leaf layers, without an accompanying slower spread within leaf layers. Various mechanisms may be proposed to account for this characteristic of resistant cultivars. Four such hypothetical mechanisms were evaluated by incorporating them in the model and testing whether their incorporation affects calculated $t50_l$-values more strongly than r_l-values.

1. *Multiple component changes.* Changing one resistance component was shown to affect vertical spread and spread within leaf layers to similar extent. However, if the component effects are not additive, some combinations of component changes might primarily affect vertical spread. Conceivably, the resistant cultivars show greater resistance for one component while being slightly more susceptible for another, such that $t50_l$-values are increased while the effects on r_l cancel out. In many pathosystems host genotypes with a low value of *IE* are characterized by high *SI*-values (Parlevliet,

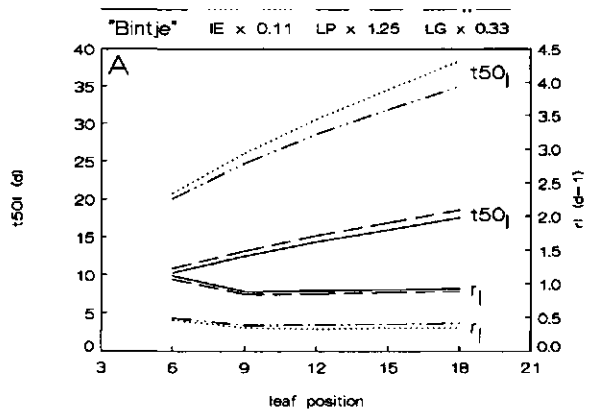
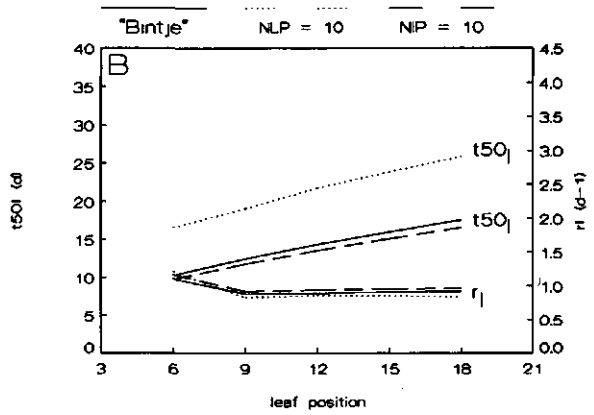
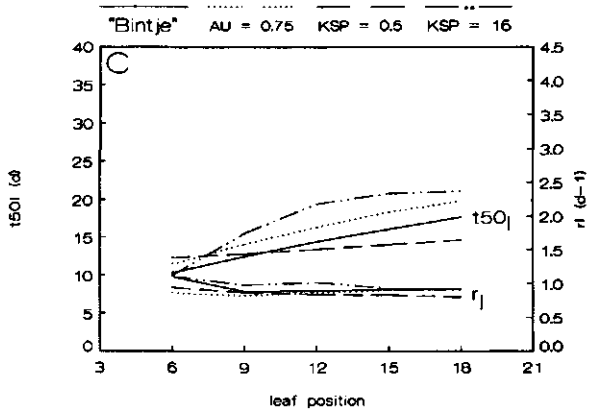
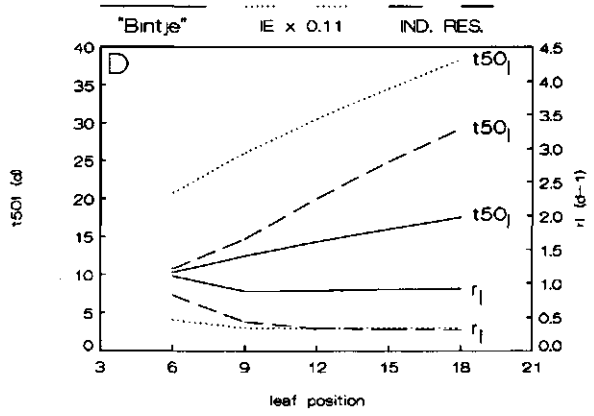


Fig. 8.2. Parameters of increasing lesion coverage at different leaf positions, derived from simulations (compare with Fig. 8.1).
A: Simulations of cv. Bintje and three cultivars in which the value of *IE*, *LP* or *LG* was set at the most resistant value reported in the literature (11%, 125% and 33% of the 'Bintje'-values, respectively; percentages calculated from data in Table 7.2). B: Simulations of cv. Bintje and two cultivars in which the exponential distribution of *LP* or *IP* was replaced by an almost normal distribution, by using ten boxcars for latency (*NLP*=10) or infectiousness (*NIP*=10). C: Simulations of cv. Bintje and three cultivars in which the fraction of sporangia remaining at the source leaf (*AU*) was increased from 0 to 0.75, or in which the exponential exctinction coefficient for sporangium dispersal between leaf layers (*KSP*) was changed from 4.0 to 0.5 or 16.0. D: Simulations of cv. Bintje and two cultivars in which the value of *IE* was set at the most resistant value reported in the literature (11% of the 'Bintje'-value), either from the beginning or after resistance induction when crop disease severity had reached 20% ('IND. RES.'). ↑→


"Bintje" NLP = 10 NIP = 10
B
t50l
t50l
rl
t50l (d)
rl (d−1)
leaf position


"Bintje" AU = 0.75 KSP = 0.5 KSP = 16
C
t50l
rl
t50l (d)
rl (d−1)
leaf position


"Bintje" IE x 0.11 IND. RES.
D
t50l
t50l
t50l
rl
rl
t50l (d)
rl (d−1)
leaf position

1979). For potato late blight information about association of resistance components is not conclusive (Chapter 4). Therefore all pairwise changes of components were examined by simulation. However, no deviations from additivity were found, neither for the $t50_l$-values of the various leaf layers nor for r_l. This confirms the results of the earlier analysed, simpler model, without foliage stratification, where pairwise component changes had only slightly superadditive effects on the time between inoculation and 50% disease of the total leaf area ($t50_y$: Chapter 7). The model thus indicated that multiple component changes cannot explain why only vertical spread is retarded in the resistant cultivars.

2. *Temporal distribution of pathogen life cycle stages.* The earliest sporangia produced after latency determine the epidemic rate more than the last (Zadoks and Schein, 1979). The slow vertical disease spread in the resistant cultivars might therefore be caused by late appearance of the first sporangia, even when average latency and total sporangium production are similar to those of the susceptible cultivar. Genotypic differences in r_l may then be small if r_l depends mainly on average latency and sporangium production. This hypothesis was quantified by changing the variation about the mean of the distributions of *LP* and *IP* without changing the means themselves. Various distributions of *LP* and *IP* were modelled using the fixed boxcar train-technique (Goudriaan and van Roermund, 1989). Variation about the mean was reduced by increasing the number of boxcars from one to ten, which changes the distribution of *LP* and *IP* from exponential to nearly normal. Reducing variation about the mean value of *LP* indeed increased $t50_l$ much more than r_l (Fig. 8.2B), while a similar change of *IP* had less effect, probably because *IP* was much smaller than *LP*. The results thus confirm that differences in temporal distribution of *LP* may explain the observed differences in $t50_l/r_l$-relations of susceptible and resistant cultivars.

3. *Spatial distribution of the pathogen.* Vertical disease spread might also be retarded if the efficiency of upward dispersal of sporangia is reduced. This hypothesis was tested. In the model the vertical spread is determined by the percentage of the sporangia that disperse out of the leaf layer where they are formed, and by the extinction coefficient for sporangium interception. Both parameters were independently changed. First, the percentage of sporangia that stay in the leaf layer where they are formed was increased from 0% to 75%. Secondly, the extinction coefficient for sporangium interception was increased fourfold. These parameter changes reduce the number of sporangia dispersed and the average dispersal distance, respectively. Both parameter changes supported the hypothesis: the restriction of sporangium dispersal affected $t50_l$ more than r_l (Fig. 8.2C).

4. *Dynamically changing levels of resistance.* Pronounced genotypic differences in

$t50_l$ were already visible within three weeks after inoculation, but these differences did not increase much thereafter (Fig. 8.1). The low $t50_l$-values of the susceptible cultivar may thus have been caused by a short period of high susceptibility early in the development of the epidemic, followed by a period of greater resistance, in which r_l-values are similar to those of the resistant cultivars. *IE* of young leaves of a susceptible potato cultivar can be reduced if the plant has had previous contact with *P. infestans* in older leaves (Doke et al., 1987). The effect of such induced resistance was tested by simulating a hypothetical cultivar of which *IE* changed from minimum to maximum resistance when the average lesion coverage of the crop reached 20%. The two consecutive levels of *IE* of this cultivar thus correspond to those of the 'Bintje'-simulation and to the run with low *IE* already presented (Fig. 8.2A). In comparison to these two runs, the cultivar with induced resistance shows intermediate values of $t50_l$, whereas r_l is mostly similar to the run with low *IE* (Fig. 8.2D). Therefore, if the susceptible cv. Bintje becomes equally resistant as the other cultivars, but at a later time, only the $t50_l$-values would be reduced, as has been observed.

Concluding remarks

A model was used here for a first rough screening of hypothetical explanations of the differences between susceptible and resistant cultivars in dynamics of disease profiles. Changes of one or more resistance components did not account for the observed differences. Only more complex explanations were found to be acceptable: 1) the length of the latent period shows less variation among lesions of resistant cultivars, 2) sporangia are dispersed over less leaf layers in resistant cultivars, 3) susceptible cultivars acquire induced resistance some time after initial infection. These explanations have in common that one of the stages in the life cycle of the pathogen shows less variation in resistant cultivars than in susceptible cultivars. This variation should be emphasized more in comparative experimental studies of different genotypes, where so far mainly average values of resistance components are compared and genotypic differences in dispersal characteristics are generally not included at all. Only such experimental research can further narrow down the list of acceptable hypotheses given above. The hypotheses are stated in quantitative terms, to allow incorporation in the mathematical model, and can therefore easily be tested experimentally.

GENERAL DISCUSSION

Why a study of disease?

All research presented in the preceding chapters presupposes some build-up of *Phytophthora infestans* in potato crops. Such research would be unnecessary if epidemics could be prevented by sanitary reduction of initial inoculum, the use of completely resistant cultivars or application of fungicides. However, sanitation without additional control measures cannot prevent epidemics because of the high multiplication rate of the fungus (MacKenzie et al., 1983). Complete resistance has also proved insufficient. The repeated introduction of completely resistant cultivars in the first half of this century was always followed shortly by the appearance of new virulent blight races. Chemical control is quite effective, but the costs of fungicides, their negative side-effects on the environment, and selection of fungicide-resistant pathogen genotypes may increasingly limit their applicability. Therefore potato growing conditions with some disease build-up should be considered. This has been acknowledged by most resistance breeding programmes, which now aim at partial resistance instead of complete resistance. Even if fungicides remain in use, partially resistant cultivars are useful in that they allow less frequent spraying, and reduce farmer risks if a spraying has to be postponed.

Problems in resistance breeding

Partial resistance is believed to be more durable than complete resistance (Thurston, 1971; Vanderplank, 1971; Umaerus et al., 1983), but no successful partially resistant cultivars have been introduced as yet (Umaerus et al., 1983; Ross, 1986). Progress in breeding for partial resistance to potato late blight has been slow mainly for three reasons. Firstly, partial resistance generally is a multigenic characteristic. This may improve its durability but also complicates the accumulation of resistance genes in new genotypes when other agronomic characteristics have to be optimized too. Secondly, the level of partial resistance of potato genotypes has generally been quantified in terms of percentage foliage diseased or amount of green leaf area. These measures of resistance are subject to confounding by genotypic differences in foliage size. Thirdly, little is known of the mechanisms underlying disease resistance. The inadequate resistance assessment methods and the lack of knowledge about resistance

mechanisms have made screening for resistance inefficient. Therefore in resistance breeding research the emphasis has shifted from the overall level of partial resistance of genotypes to components of partial resistance. The components represent different stages and events in the life cycle of the pathogen with which the host can interfere: infection efficiency, latent period, lesion growth rate, infectious period and sporulation intensity (Parlevliet, 1979). However, the many studies about components of resistance to potato late blight that have been published have not yet led to identification of the components that are of primary importance in reducing the epidemic rate (Chapter 4).

An alternative approach: production ecology

Breeding for partial resistance may benefit from an interdisciplinary, production ecological approach. Production ecology aims at unravelling the crop processes that determine yield. It analyses the effect on these processes of the interaction between the plants and their biotic and abiotic environment. The strength of the approach lies in the diversity of system features studied. This diversity not only guards against overlooking important aspects of the system, but is also needed to quantify the interaction between factors that were traditionally studied separately, in different research disciplines. The processes that mainly determine the production of a crop are identified. Subsequent research may then be concentrated on these processes and the possibilities to manipulate them. Breeding research may thus be guided to processes which can be manipulated by means of the host genotype. This 'guidance' was attempted in the present study for processes that determine yield loss caused by potato late blight.

Experiments or models?

Experiments and models were used. Experiments were used to quantify genetic variation for various plant characteristics (Chapters 1-3), while models were used to assess the effect of these characteristics on yield loss (Chapters 6 and 7). Models are needed to assess these effects because yield is the outcome of many interacting processes, and genotypes which differ in only one of these processes are rare. However, although such interactions may best be studied by simulation, the models used do require additional experimental validation. Analyses of the submodel for blight epidemiology have demonstrated that especially sporangium dispersal and the variability in the latent period may need further study (Chapter 8). In further studies it should also be verified that the non-representative nature of the induced epidemics (i.e. general instead of focal epidemics), both in the experiments and the models, has not

interfered with the identification of characteristics affecting loss.

The relation between experimentation and modelling will be discussed further in later sections of this chapter.

The *LUE/PARCUM*-analysis as a starting point of a production ecological study

The efficiency of dry mass production per unit intercepted light (*LUE*) was shown to be unaffected by blight, in all cultivars examined (Chapter 1). Differences in yield loss were therefore caused by the amount of light intercepted (*PARCUM*) alone, which points to the dynamics of leaf area as the process through which the effects of the disease were exerted. Measuring yield and light interception thus sufficed to identify the leaf area dynamics as the process to be further analysed. If the experiments would have revealed changes in *LUE* instead of *PARCUM*, effects on photosynthesis and assimilate partitioning rather than leaf area dynamics would have been probable. The *LUE/PARCUM*-analysis thus helps identifying crop physiological processes that are affected by a pathogen or by abiotic stress. The analysis therefore is a suitable starting-point for production ecological studies. However, the *LUE/PARCUM*-analysis does not always yield such unequivocal results. If the disease had reduced *LUE* as well as *PARCUM* it would have been difficult to demonstrate experimentally to what extent the reduction of *PARCUM* was directly caused by the disease, through leaf lesion coverage, or only indirectly, through the decreased *LUE* reducing leaf area growth. In that case the effect of *LUE* on *PARCUM* could have been quantified by simulating crop growth with reduced *LUE* alone, to test whether this would reduce *PARCUM* to the extent observed or less.

Resistance or tolerance?

Resistance is the ability of the host to hinder the growth and/or development of the pathogen, while tolerance is the ability to endure the presence of the pathogen with reduced disease symptoms and/or damage (Parlevliet, 1979). For foliage blight of potato these definitions can be made more precise. Resistance is the ability to hinder the increasing coverage of leaf area with lesions, while tolerance is the ability to maintain production capacity in leaf area outside the lesions. Tolerance may thus conveniently be split up in maintenance of functional leaf area outside lesions and maintenance of activity level in that functional leaf area. The first component of tolerance is measured by the acceleration of the rate of senescence caused by the pathogen, while the second component is measured by the rate of photosynthesis in green leaf area. The present

study has shown that there was no genetic variation for either component of tolerance: the activity of green leaves was not affected by the disease (*LUE* and photosynthetic rate were not affected; Chapters 1 and 2), while senescence was accelerated by the disease, but to similar extent in all cultivars examined (Chapter 3).

Genetic variation for resistance was demonstrated by differences between cultivars in rate of disease spread (Chapters 1 and 3), with corresponding differences in yield loss, while in the literature many other reports about genetic variation for partial resistance can be found (Chapter 4). Since no variation in tolerance was found, partial resistance was obviously of more importance in explaining genotypic differences in yield loss caused by late blight.

Which component of resistance?

Components of resistance were not quantified experimentally, but taken from the literature. Component values are changed when plants are grown under controlled conditions, but cannot easily be assessed accurately in the field. Therefore only a minority of the published data about genetic variation of resistance components has been determined in the field (Chapter 4). These field data were used to parameterize a simulation model of blight epidemiology. Sensitivity analysis pointed to lesion growth rate and infection efficiency as the components for which the available genetic variation offered most scope for improving the overall level of partial resistance of potato cultivars (Chapter 7). The two components were identified for different reasons: lesion growth rate because of its strong influence on disease progress, infection efficiency because of its large variation between genotypes. This illustrates the complementarity of experiments and simulation models when trying to identify plant characteristics for breeding purposes: experiments show the scope for changing plant characteristics by breeding and models help estimating the consequences of those changes for overall cultivar performance.

Resistance ... or maturity class?

The *LUE/PARCUM*-analysis has shown that the effect of blight on yield was exerted through effects on the leaf area dynamics. Genotypic differences in resistance level explained why the disease destroyed the leaf area faster in some cultivars than in others. However, apart from the resistance level the leaf area dynamics of a cultivar are also determined by growth characteristics that cause genotypic differences in foliage size and structure even in the absence of blight. Several such growth characteristics, as leaf

growth rate and leaflet size, had little effect on yield loss (Chapter 6). However, the maturity class of cultivars did strongly affect yield loss. Late cultivars yielded the most in the absence of the pathogen, but with disease late cultivars had lower yields than early cultivars at equal values of their resistance components. This interaction between maturity class and yield loss may be difficult to demonstrate experimentally, because of the problems with field measurements of resistance components and because of the lack of cultivars differing in maturity class but equal in resistance. The model analysis pointed to the following explanation for the high yield losses of late cultivars. Late cultivars continue foliage growth longer than early cultivars, at the expense of tuber filling. Therefore it takes the fungus longer to destroy all leaf area in late cultivars, but this advantage is more than offset by the shorter period of tuber growth. The fact that late cultivars appear more resistant, i.e. have more green leaf area, causes them to suffer more yield loss.

Yield loss thus is affected by both resistance level and maturity class of a cultivar. For the genotypes studied the resistance level has the greatest effect. However, the effect of maturity class is sufficiently strong to merit screening for resistance within groups of similar maturity class only, unless the screening methods aim at resistance components, instead of yield or leaf area duration.

Production ecology of pathosystems: other topics

The present study deals with foliage blight, and its effect on the quantity of tubers produced. The effect of late blight on the quality of tuber production would be a useful next research topic. This could focus on the analysis of genotypic differences in tuber infection, again using a combination of experimentation and modelling.

The production ecological framework used here for analysing genotypic differences in potato yield loss caused by late blight could be applied for other pathosystems as well. A *LUE/PARCUM*-analysis may generally be a suitable starting-point, to make a first rough selection of the production processes that are of main importance in the pathosystem studied. A subsequent analysis of the effect on production of genotypic differences in components of resistance, tolerance and host growth may also be generally useful. If the pathogen is a fungal leaf disease, the resistance components analysis may be done with the model presented here although this model may be simplified for diseases without indeterminate lesion growth (Chapter 7). However, the main host characteristics affecting yield loss may not be as easily identified in other pathosystems. If, for example, *LUE* and tolerance do show considerable genetic variation, more processes and their interactions must be studied in depth. In such cases the production ecological analysis

may involve more steps than for potato late blight, but is still useful due to its balanced treatment of the many interactions in the production system.

SUMMARY

The pathosystem *Phytophthora infestans* - potato has been studied in various research disciplines. In the General introduction it is argued that approaches typical for the different disciplines could be fruitfully combined in one study of the pathosystem. An outline of such a research programme is given and the results are presented in the following chapters.

Chapter 1 shows that the Light Use Efficiency (*LUE*: the efficiency with which light, intercepted by green leaves, is utilized for biomass production) of twenty potato cultivars, differing in levels of partial resistance and maturity class, is not affected by *P. infestans*.

Since *LUE* is not affected by the disease, it may be expected that late blight does not affect the rate of photosynthesis in green leaves of potato genotypes. This is confirmed in Chapter 2 where photosynthesis measurements on healthy and diseased plants are reported.

The disease thus does not affect the activity of green leaves. Therefore the effect of the disease on loss of green leaf area is studied in Chapter 3. Two possible modes of leaf destruction are separately quantified: acceleration of leaf senescence outside the directly infected parts of the foliage, and lesion expansion. It is shown that blight indeed accelerates senescence, but that more leaf loss is suffered due to coverage of leaves by lesions, and only for the latter process genetic variation is demonstrated. The implications are discussed of these results for the effects on yield loss of genotypic differences in partial resistance, tolerance and lateness.

Chapters 4 and 5 present literature surveys about components of resistance to *P. infestans* and the use of epidemiological models to evaluate the relative importance of these components for the reduction of the rate of disease progress.

Some of the results of the literature surveys are used, in Chapter 6, to construct a simulation model of the growth of potato cultivars and populations of the fungus. The model accurately simulates the results of the field experiments presented in Chapters 1 and 3. The simulations show that the contribution of accelerated senescence to yield loss in these experiments was less than 15%. Sensitivity analysis with the model shows an advantage of cultivar earliness in reducing yield loss, whereas other plant growth characteristics seem to have little effect on yield.

Chapter 7 compares the model of Chapter 6 to other more commonly used and simpler epidemiological models. The simpler models are shown to be more sensitive to changes in resistance components. The necessity of incorporating lesion growth rate

and considering simultaneous changes of more than one component is demonstrated. When both model sensitivity for different components and available genetic variation for the components are considered, lesion growth rate and infection efficiency seem to offer the best perspectives for improvement of potato genotypes by breeding.

Chapter 8 deals with one particular aspect of the blight epidemics reported about in Chapter 3, namely the gradual vertical spread of the pathogen from the bottom leaf layers to the canopy top. Different approaches to modelling this phenomenon are discussed. It is shown that only quite complex hypotheses explain the observations.

The work is concluded with a General discussion about the methodology followed in the present research work, the conclusions that were reached, and the further scope for analysis, by means of simulation modelling, of potato late blight and other pathosystems.

SAMENVATTING

Het pathosysteem *Phytophthora infestans* - aardappel wordt al vele jaren bestudeerd in verschillende onderzoeksdisciplines. In de inleiding van dit proefschrift wordt gesteld dat benaderingen die kenmerkend zijn voor deze disciplines kunnen worden gecombineerd in één studie van het pathosysteem.

Hoofdstuk 1 laat zien dat de efficiëntie van lichtbenutting (*LUE*: de efficiëntie waarmee licht, onderschept door groene bladeren, wordt benut voor de produktie van biomassa) van twintig aardappelrassen, die verschillen in resistentieniveau en rijpheidsklasse, niet wordt beïnvloed door *P. infestans*.

Aangezien de *LUE* niet wordt beïnvloed door de ziekte, lijkt de aardappelziekte geen effekt te hebben op de snelheid van fotosynthese in groene bladeren van aardappelgenotypen. Metingen van fotosynthese aan gezonde en zieke planten bevestigen dit (Hoofdstuk 2).

De ziekte heeft dus geen invloed op de aktiviteit van groene bladeren. Daarom wordt het effekt van de ziekte op verlies van groen bladoppervlak bestudeerd in Hoofdstuk 3. De schimmel veroorzaakt bladverlies voornamelijk door de uitbreiding van lesies over de bladeren maar ook door versnelling van bladveroudering buiten de geïnfekteerde delen van het loof. Alleen voor de lesie-uitbreiding wordt genetische variatie aangetoond. Naar aanleiding van deze resultaten wordt de relatie tussen opbrengstderving en planteigenschappen als partiële resistentie, tolerantie en laatheid bediscussieerd.

Hoofdstukken 4 is een overzicht van de literatuur over componenten van resistentie tegen *P. infestans*. Hoofdstuk 5 bespreekt literatuur waarin epidemiologische modellen worden gebruikt om te bepalen welke resistentie-componenten ziektetoename het sterkst remmen.

Enkele van de resultaten van de literatuuroverzichten worden gebruikt, in Hoofdstuk 6, om een simulatiemodel te maken van de groei van aardappelgewassen en de populatie-opbouw van de schimmel. Met het model worden de resultaten van de veldexperimenten van hoofdstukken 1 en 3 nauwkeurig gesimuleerd. Daarbij blijkt dat de bijdrage van versnelling van bladveroudering aan opbrengstderving in deze experimenten onder de 15% ligt. Gevoeligheidsanalyse van het model laat zien dat vroegheid van een ras opbrengstderving kan beperken, terwijl andere planteigenschappen slechts een gering effekt hebben.

In Hoofdstuk 7 wordt het model van het voorafgaande hoofdstuk vergeleken met andere, meer gangbare en eenvoudige epidemiologische modellen. Deze eenvoudigere

modellen missen de voor aardappelziekte noodzakelijke component lesiegroeisnelheid en reageren daardoor gevoeliger op veranderingen in de wel opgenomen resistentiecomponenten. Het effekt van gelijktijdige veranderingen van meerdere componenten op de ziekte is onderzocht. Als zowel de modelgevoeligheid voor de verschillende componenten als de beschikbare genetische variatie in overweging worden genomen, bieden de componenten lesiegroeisnelheid en infektie-efficiëntie de beste mogelijkheden voor de veredeling op resistentie tegen *P. infestans*.

Hoofstuk 8 behandelt een aspekt van de aardappelziekte-epidemieën dat al was genoemd in Hoofdstuk 3, namelijk de geleidelijke verspreiding van het pathogeen van de onderste bladlagen naar de bovenkant van het gewas. Verschillende benaderingen voor de modellering van dit verschijnsel worden besproken. Slechts vrij gecompliceerde hypothesen leiden tot een realistische modellering.

Het proefschrift besluit met een discussie over de toegepaste onderzoeksmethoden, de bereikte conclusies en de mogelijkheden voor verdere analyses, met behulp van simulatiemodellering, van de aardappelziekte en andere pathosystemen.

REFERENCES

Anderson, R.M. & May, R.M., 1982. Directly transmitted infectious diseases: control by vaccination. Science 215: 1053-1060.

Anonymous, 1988. Descriptive list of varieties of field crops. Leiter-Nypels, Maastricht, 337 pp.

Aust, H.J., Hau, B. & Kranz, J., 1983. EPIGRAM - a simulator of barley powdery mildew. Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz 90: 244-250.

Bailey, N.T.J., 1975. The mathematical theory of infectious diseases and its applications. Charles Griffin & Company Ltd., London.

Bashi, E., Ben-Yoseph, Y. & Rotem, J., 1982. Inoculum potential of *Phytophthora infestans* and the development of potato late blight epidemics. Phytopathology 72: 1043-1047.

Becker, N., 1979. The uses of epidemic models. Biometrics 35: 295-305.

Behnke, M., 1979. Selection of potato callus for resistance to culture filtrates of *Phytophthora infestans* and regeneration of resistant plants. Theoretical and Applied Genetics 55: 69-71.

Behnke, M., 1980. General resistance to late blight of *Solanum tuberosum* plants regenerated from callus resistant to culture filtrates of *Phytophthora infestans*. Theoretical and Applied Genetics 56: 151-152.

Berger, R.D., 1977. Application of epidemiological principles to achieve plant disease control. Annual Review of Phytopathology 15: 165-183.

Berger, R.D. & Jones, J.W., 1985. A general model for disease progress with functions for variable latency and lesion expansion on growing host plants. Phytopathology 75: 792-797.

Berggren, B., Widmark, A. & Umaerus, V., 1988. The expression of general resistance to late blight (*Phytophthora infestans*) in potato leaves. Potato Research 31: 611-616.

Berghaus, R. & Reisener, H.J., 1985. Changes in photosynthesis of wheat plants infected with wheat stem rust (*Puccinia graminis* f.sp. *tritici*). Phytopathologische Zeitschrift 112: 165-172.

Björling, K. & Sellgren, K.A., 1955. Deposits of sporangia and incidence of infection by *Phytophthora infestans* on upper and lower surfaces of potato leaves. Acta Agriculturae Scandinavica V: 375-386.

Bruggen, A.H.C. van, Osmeloski, J.F. & Jacobson, J.S., 1987. Effects of simulated acidic mist on germination of *Alternaria solani* and *Phytophthora infestans* in vitro and their infection efficiency and sporulation on potato. Phytopathology 77: 564-570.

Bruhn, J.A. & Fry, W.E., 1981. Analysis of potato late blight epidemiology by simulation modeling. Phytopathology 71: 612-616.

Burstall, L. & Harris, P.M., 1983. The estimation of percentage light interception from leaf area index and percentage ground cover in potatoes. The Journal of Agricultural Science 100: 241-244.

Caemmerer, S. von & Farquhar, G.D., 1981. Some relationships between the biochemistry of photosynthesis and the gas exchange of leaves. Planta 153: 376-387.

Carnegie, S.F. & Colhoun, J., 1980. Differential leaf susceptibility to *Phytophthora infestans* on potato plants of cv. King Edward. Phytopathologische Zeitschrift 98: 108-117.

Carnegie, S.F. & Colhoun, J., 1982. Susceptibility of potato leaves to *Phytophthora infestans* in relation to plant age and leaf position. Phytopathologische Zeitschrift 104: 157-167.

Carnegie, S.F. & Colhoun, J., 1983. Effects of plant nutrition on susceptibility of potato leaves to *Phytophthora infestans*. Phytopathologische Zeitschrift 108: 242-250.

Caten, C.E., 1970. Spontaneous variability of single isolates of *Phytophthora infestans*. II. Pathogenic variation. Canadian Journal of Botany 48: 897-905.

Child, J.J. & Fothergill, P.G., 1967. The amino acid content of potato plant varieties and their resistance to attack by *Phytophthora* species. Journal of the Science of Food and Agriculture

18: 3-7.

Clarke, D.D., 1983. Potato late blight: a case study. In: Callow, J.A. (Ed.), Biochemical plant pathology. John Wiley & Sons Ltd., Chichester: 3-17.

Clayson, A.M. & Robertson, N.F., 1956. Survival of *Phytophthora infestans* in potato stem lesions. Plant Pathology 5: 30-31.

Cohen, Y. & Rotem, J., 1987. Sporulation of foliar pathogens. In: Pegg, G.F. and Ayres, P.G. (Eds), Fungal infection of plants. Cambridge University Press, Cambridge: 314-333.

Colon, L.T. & Budding, D.J., 1990. Components of field resistance to *Phytophthora infestans* in *Solanum* spp. In: MacKerron, D.K.L. et al. (Eds), EAPR Abstracts. Edinburgh, United Kingdom: 84-85.

Corsten, L.C.A., 1964. Een kwantitatieve beschrijving van de ontwikkeling van een schimmelpopulatie. Mededelingen van de Landbouwhogeschool 64-15: 1-7.

Crosier, W., 1934. Studies in the biology of *Phytophthora infestans* (Mont.) de Bary. Cornell University Agricultural Experiment Station Memoir 155.

Darsow, U., Junges, W. & Oertel, H., 1988. Die Bedeutung der Prädisposition für die Laborprüfung von Kartoffelblättern auf relative Resistenz gegenüber *Phytophthora infestans* (Mont.) de Bary. Archiv für Phytopathologie und Pflanzenschutz 24: 109-119.

Desmukh, M.J. & Howard, H.W., 1956. Field resistance to potato blight (*Phytophthora infestans*). Nature 177: 794-795.

Doke, N., Ramirez, A.V. & Tomiyama, K., 1987. Systemic induction of resistance in potato plants against *Phytophthora infestans* by local treatment with hyphal cell wall components of the fungus. Journal of Phytopathology 119: 232-239.

Dwelle, R.B., 1985. Photosynthesis and photoassimilate partitioning. In: Li, P.H. (Ed.), Potato physiology. Academic Press, Orlando: 35-58.

Elston, D.A. & Simmonds, N.W., 1988. Models of sugarcane smut disease and their implications for testing variety resistance. Journal of Applied Ecology 25: 319-329.

Érsek, T. & Király, Z., 1986. Phytoalexins: Warding-off compounds in plants? Physiologia Plantarum 68: 343-346.

Farrar, J.F. & Lewis, D.H., 1987. Nutrient relations in biotrophic infections. In: Pegg, G.F. and Ayres, P.G. (Eds), Fungal infection of plants. Cambridge University Press, Cambridge: 92-132.

Ferrandino, F.J., 1989. Spatial and temporal variation of a defoliating plant disease and reduction in yield. Agricultural and Forest Meteorology 47: 273-289.

Firman, D.M. & Allen, E.J., 1988. Field measurements of the photosynthetic rate of potatoes grown with different amounts of nitrogen fertilizer. The Journal of Agricultural Science 111: 85-90.

Firman, D.M. & Allen, E.J., 1989. Relationship between light interception, ground cover and leaf area index in potatoes. The Journal of Agricultural Science 113: 355-359.

Fischer, W., Schweizer, P., Christ, U., Mösinger, E., Kovats, K., Baer, G. & Binder, A., 1988. Mechanisms in systemic induced disease resistance. Phytoparasitica 16: 211.

Gallegly, M.E. & Niederhauser, J.S., 1959. Genetic controls of host parasite interactions in the *Phytophthora* late blight disease. Plant Pathology, Problems and Progress, 1908-1959.

Gees, R. & Hohl, H.R., 1988. Cytological comparison of specific (R3) and general resistance to late blight in potato leaf tissue. Phytopathology 78: 350-357.

Gilligan, C.A., 1985. Introduction. In: Gilligan, C.A. (Ed.), Mathematical modelling of crop disease. Advances in Plant Pathology 3: 1-10.

Goudriaan, J. & Roermund, H.J.W. van, 1989. Modelling of ageing, development, delays and dispersion. In: Rabbinge, R., Ward, S.A. and Laar, H.H. van (Eds), Simulation and systems management in crop protection. Simulation Monographs 32, Pudoc, Wageningen: 47-79.

Gumpert, F.-M., 1989. Measuring disease progress in pure and mixed stands of plant cultivars. Phytopathology 79: 968-973.

Gumpert, F.-M., Geiger, H.H. & Stähle, U., 1987. A mathematical model of the epidemics in homogeneous and heterogeneous host stands. Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz 94: 206-215.

Guzman-N., J., 1964. Nature of partial resistance of certain clones of three *Solanum* species to *Phytophthora infestans*. Phytopathology 11: 1398-1404.

Harrison, J.G. & Lowe, R., 1989. Effects of humidity and air speed on sporulation of *Phytophthora infestans* on potato leaves. Plant Pathology 38: 585-591.

Haverkort, A.J. & Bicamumpaka, M., 1986. Correlation between intercepted radiation and yield of potato crops infested by *Phytophthora infestans* in central Africa. Netherlands Journal of Plant Pathology 92: 239-247.

Haverkort, A.J. & Harris, P.M., 1986. Conversion coefficients between intercepted solar radiation and tuber yields of potato crops under tropical highland conditions. Potato Research 29: 529-533.

Haverkort, A.J., Uenk, D., Veroude, H. & Waart, M. van de, 1991. Radiation interception by potato canopy: Relationships between ground cover, intercepted solar radiation, leaf area index and infrared reflectance of potato crops. Potato Research 34, accepted.

Henninger, H. & Bartel, W., 1963. Die Eignung des Peroxydaseaktivitäts-Testes zur Bestimmung der 'relativen *Phytophthora*-Resistenz' (Feldresistenz) bei Kartoffeln. Der Züchter 33: 86-91.

Hethcote, H.W., 1976. Qualitative analyses of communicable disease models. Mathematical Biosciences 28: 335-356.

Hodgson, W.A., 1961. Laboratory testing of the potato for partial resistance to *Phytophthora infestans*. American Potato Journal 38: 259-264.

Hodgson, W.A., 1962. Studies on the nature of partial resistance in the potato to *Phytophthora infestans*. American Potato Journal 39: 8-13.

Imhoff, M.W., Leonard, K.J. & Main, C.E., 1982. Patterns of bean rust lesion size increase and spore production. Phytopathology 72: 441-446.

James, R.V. & Fry, W.E., 1983. Potential for *Phytophthora infestans* populations to adapt to potato cultivars with rate-reducing resistance. Phytopathology 73: 984-988.

James, W.C., Shih, C.S., Hodgson, W.A. & L.C. Callbeck, 1972. The quantitative relationship between late blight of potato and loss in tuber yield. Phytopathology 62: 92-96.

Jeffrey, S.I.B., Jinks, J.L. & Grindle, M., 1962. Intraracial variation in *Phytophthora infestans* and field resistance to potato blight. Genetica 32: 323-338.

Jeger, M.J., 1986. Asymptotic behaviour and threshold criteria in model plant disease epidemics. Plant Pathology 35: 355-361.

Jeger, M.J., 1987. Modelling the dynamics of pathogen populations. In: Wolfe, M.S. & Caten, C.E., Populations of plant pathogens: Their dynamics and genetics. Blackwell Scientific Publications, Oxford: 91-107.

Jeger, M.J. & Groth, J., 1985. Resistance and pathogenicity: Epidemiological and ecological mechanisms. In: Fraser, R.S.S. (Ed.), Mechanisms of resistances to plant diseases. Martinus Nijhoff, The Hague: 310-372.

Johnson, R. & Taylor, A.J., 1976. Spore yield of pathogens in investigations of the race-specificity of host resistance. Annual Review of Phytopathology 14: 97-119.

Jones, J.L. & Allen, E.J., 1983. Effects of date of planting on plant emergence, leaf growth, and yield in contrasting potato varieties. The Journal of Agricultural Science 101: 81-95.

Jones, L.R., Giddings, N.J. & Lutman, B.F., 1912. Investigations of the potato fungus *Phytophthora infestans*. Vermont Agricultural Experiment Station Bulletin 168, 100 pp.

Kammerman, N., 1951. Undersökninger rörande Potatisbladmöglet, *Phytophthora infestans* (Mont.) de Bary II. Sanbaneet mellan Potatisbladsaftens peroxidasaktivitet och Phytophthora resisten. Medd. Vaxskyddsanst, Stockholm 58, 32 pp. Review of Applied Mycology 31: 78.

Keen, N.T. & Yoshikawa, M., 1983. Physiology of disease and the nature of resistance to

Phytophthora. In: Erwin, D.C., Bartnicki-Garcia, S. and Tsao, P.H. (Eds), *Phytophthora*: Its biology, taxonomy, ecology and pathology. The American Phytopathological Society, St. Paul: 279-287.

Kermack, W.O. & McKendrick, A.G., 1927. A contribution to the mathematical theory of epidemics. Proceedings of the Royal Society of London, Series A 115: 700-721.

Knudsen, G.R., Spurr, H.W. & Johnson, C.S., 1987. A computer simulation model for Cercospora leaf spot of peanut. Phytopathology 77: 1118-1121.

Knutson, K.W., 1962. Studies on the nature of field resistance of the potato to late blight. American Potato Journal 39: 152-161.

Knutson, K.W. & Eide, C.J., 1961. Parasitic aggressiveness in *Phytophthora infestans*. Phytopathology 51: 286-290.

Kulkarni, R.N., Chopra, V.L. & Singh, D., 1982. Relative importance of components affecting the leaf rust progress curve in wheat. Theoretical and Applied Genetics 62: 205-207.

Lapwood, D.H., 1961a. Potato haulm resistance to *Phytophthora infestans*. I. Field assessment of resistance. Annals of Applied Biology 49: 140-151.

Lapwood, D.H., 1961b. Potato haulm resistance to *Phytophthora infestans*. II. Lesion production and sporulation. Annals of Applied Biology 49: 316-330.

Lapwood, D.H., 1961c. Potato haulm resistance to *Phytophthora infestans*. III. Lesion distribution and leaf destruction. Annals of Applied Biology 49: 704-716.

Lapwood, D.H., 1961d. Laboratory assessments of the susceptibility of potato haulm to blight (*Phytophthora infestans*). European Potato Journal 4: 117-128.

Lapwood, D.H., 1963. Potato haulm resistance to *Phytophthora infestans*. IV. Laboratory and field estimates compared, and further field analyses. Annals of Applied Biology 51: 17-28.

Lapwood, D.H., 1971. Observations on blight (*Phytophthora infestans*) and resistant potatoes at Toluca, Mexico. Annals of Applied Biology 68: 41-53.

Latin, R.X., MacKenzie, D.R. & Cole, H., 1981. The influence of host and pathogen genotypes on the apparent infection rates of potato late blight epidemics. Phytopathology 71: 82-85.

Leonard, K.J., 1969. Factors affecting rates of stem rust increase in mixed plantings of susceptible and resistant oat varieties. Phytopathology 59: 1845-1850.

Leonard, K.J. & Mundt, C.C., 1984. Methods for estimating epidemiological effects of quantitative resistance to plant diseases. Theoretical and Applied Genetics 67: 219-230.

Liu, W.M., Hethcote, H.W. & Levin, S.A., 1987. Dynamical behavior of epidemiological models with nonlinear incidence rates. Journal of Mathematical Biology 25: 359-380.

Loomis, R.S. & Adams, S.S., 1983. Integrative analyses of host-pathogen relations. Annual Review of Phytopathology 21: 341-362.

Louwerse, W. & Oorschot, J.L.P. van, 1969. An assembly for routine measurements of photosynthesis, respiration and transpiration of intact plants under controlled conditioning. Photosynthetica 3: 305-315.

Lowings, P.H. & Acha, I.G., 1959. Some factors affecting growth of *Phytophthora infestans* (Mont.) de Bary: I. *P. infestans* on living potato leaves. Transactions of the British Mycological Society 42: 491-501.

MacKenzie, D.R., Elliott, V.J., Kidney, B.A., King, E.D., Royer, M.H. & Theberge, R.L., 1983. Application of modern approaches to the study of the epidemiology of diseases caused by *Phytophthora*. In: Erwin, D.C., Bartnicki-Garcia, S. and Tsao, P.H. (Eds), *Phytophthora*: Its biology, taxonomy, ecology and pathology. The American Phytopathological Society, St. Paul: 303-313.

Main, C.E. & Gallegly, M.E., 1964. The disease cycle in relation to multigenic resistance of potato to late blight. American Potato Journal 41: 387-400.

Malcolmson, J.F., 1969. Factors involved in resistance to blight (*Phytophthora infestans* (Mont.) de Bary) in potatoes and assessment of resistance using detached leaves. Annals of Applied

Biology 64: 461-468.
Malcolmson, J.F. & Killick, R.J., 1980. The breeding values of potato parents for field resistance to late blight measured by whole seedlings. Euphytica 29: 489-495.
Martin, S.B., Campbell, C.L. & Bruck, R.I., 1987. Influence of acidity level in simulated rain on disease progress and sporangial germination, infection efficiency, lesion expansion, and sporulation in the potato late blight system. Phytopathology 77: 969-974.
Mehta, Y.R. & Zadoks, J.C., 1970. Uredospore production and sporulation period of *Puccinia recondita* f.sp. *triticina* on primary leaves of wheat. Netherlands Journal of Plant Pathology 76: 267-276.
Michaelides, S.C., 1985. A simulation model of the fungus *Phytophthora infestans* (Mont.) De Bary. Ecological Modelling 28: 121-137.
Monteith, J.L., 1977. Climate and the efficiency of crop production in Britain. Philosophical Transactions of the Royal Society of London, Series B 281: 277-294.
Müller, K.O. & Haigh, J.C., 1953. Nature of 'field resistance' of the potato to *Phytophthora infestans* de Bary. Nature 171: 781-783.
Nandris, D., Vallavielle, C. de & Bouvier, J., 1979. Studies on some interactions between potatoes and *Phytophthora infestans*. Physiological Plant Pathology 15: 1-12.
Niederhauser, J.S., 1961. Genetic studies of *Phytophthora infestans* and *Solanum* species in relation to late blight resistance in the potato. In: Recent advances in botany, Proceedings of IX International Botanical Congress, Montreal 1959. University of Toronto Press: 491-497.
Nilsson, B.-A., 1981. Component analysis of general resistance to *Phytophthora infestans* in clones from the Colombian potato collection. Potato Research 24: 239-244.
Oijen, M. van, 1989. On the use of mathematical models from human epidemiology in breeding for resistance to polycyclic fungal leaf diseases of crops. In: Louwes, K.M., Toussaint, H.A.J.M. and Dellaert, L.M.W. (Eds), Parental line breeding and selection in potato breeding. Pudoc, Wageningen: 26-37.
Oijen, M. van, 1990. Modelling the influences of components of field resistance to *Phytophthora infestans* on disease progress in potato. Phytophthora Newsletter 16: 27-28.
Oijen, M. van & Budding, D.J., 1988. Perspectieven van veredeling op partiële resistentie of tolerantie tegen *Phytophthora infestans*. Prophyta, bijlage januari: 11-16.
Oort, A.J.P., 1968. A model of the early stage of epidemics. Netherlands Journal of Plant Pathology 74: 177-180.
Parlevliet, J.E., 1979. Components of resistance that reduce the rate of epidemic development. Annual Review of Phytopathology 17: 203-222.
Parlevliet, J.E. & Ommeren, A. van, 1975. Partial resistance of barley to leaf rust, *Puccinia hordei*. II. Relationship between field trials, micro plot tests and latent period. Euphytica 24: 293-303.
Paysour, R.E. & Fry, W.E., 1983. Interplot interference: A model for planning field experiments with aerially disseminated pathogens. Phytopathology 73: 1014-1020.
Pietkiewicz, J.B., 1976. Characteristic of horizontal resistance to blight (*Phytophthora infestans*) (Mont.) de Bary in the potato. Ziemniak: 87-125.
Rabbinge, R., 1986. The bridge function of crop ecology. Netherlands Journal of Agricultural Science 3: 239-251.
Rabbinge, R., 1988. Crop loss assessment and economic injury levels. IOBC Bulletin XI/2: 42-47.
Rabbinge, R., Zadoks, J.C. & Bastiaans, L., 1989. Population models. In: Rabbinge, R., Ward, S.A. and Laar, H.H. van (Eds), Simulation and systems management in crop protection. Simulation Monographs 32, Pudoc, Wageningen: 83-97.
Rapilly, F., 1987. Apports de la modélisation et de la simulation à l'amélioration des plantes pour la résistance aux parasites: cas du couple blé *Septoria nodorum* Berk. Comptes rendus de l'Académie Agricole de France 73: 107-119.
Rapilly, F. & Delhotal, P., 1986. Sur la durabilité de résistances partielles à *Septoria nodorum*

Berk. chez le blé (*Triticum aestivum* L.): études prospectives réalisées par la simulation. Agronomie 6: 325-336.

Rapilly, F. & Jolivet, E., 1976. Construction d'un modèle (EPISEPT) permettant la simulation d'une épidémie de *Septoria nodorum* Berk. sur blé. Revue de Statistique Appliquée 24: 31-60.

Ross, H., 1986. Potato breeding - Problems and perspectives. Advances in Plant Breeding 13. Parey, Berlin.

Rotem, J., Bashi, E. & Kranz, J., 1983. Studies of crop loss in potato blight caused by *Phytophthora infestans*. Plant Pathology 32: 117-122.

Rotem, J. & Cohen, Y., 1974. Epidemiological patterns of *Phytophthora infestans* under semi-arid conditions. Phytopathology 64: 711-714.

Rotem, J., Kranz, J. & Bashi, E., 1983. Measurement of healthy and diseased haulm area for assessing late blight epidemics in potatoes. Plant Pathology 32: 109-115.

Rouse, D.I., 1985. Construction of temporal models: I. Disease progress of air-borne pathogens. In: Gilligan, C.A. (Ed.), Mathematical modelling of crop disease. Advances in Plant Pathology 3: 11-29.

Sall, M., 1980. Uses of stochastic simulation: grape powdery mildew example. Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz 87: 397-403.

Schaper, P., 1951. Die Bedeutung der Inkubationszeit für die Züchtung krautfäuleresistenter Kartoffelsorten. Zeitschrift für Pflanzenzüchtung 30: 292-299.

Scharen, A.L. & Krupinsky, J.M., 1969. Effects of *Septoria nodorum* infection on CO_2 absorption and yield of wheat. Phytopathology 59: 1298-1301.

Schmitthenner, A.F. & Canaday, C.H., 1983. Role of chemical factors in development of *Phytophthora* diseases. In: Erwin, D.C., Bartnicki-Garcia, S. and Tsao, P.H. (Eds), *Phytophthora*: Its biology, taxonomy, ecology and pathology. The American Phytopathological Society, St. Paul: 189-196.

Schöber, B., 1987. *Phytophthora infestans* (Mont.) de Bary - eine ständige Herausforderung seit 140 Jahren. Berichte der Deutscher Botanischer Gesellschaft 100: 291-303.

Schrödter, H., 1987. Wetter und Pflanzenkrankheiten: Biometeorologische Grundlagen der Epidemiologie. Springer-Verlag, Berlin, 191 pp.

Shaner, G., 1980. Probits for analyzing latent period data in studies of slow rusting resistance. Phytopathology 70: 1179-1182.

Shaner, G., 1983. Growth of uredinia of *Puccinia recondita* in leaves of slow- and fast-rusting wheat cultivars. Phytopathology 73: 931-935.

Shaner, G. & Hess, F.D., 1978. Equations for integrating components of slow leaf-rusting resistance in wheat. Phytopathology 68: 1464-1469.

Shearer, B.L. & Zadoks, J.C., 1972. The latent period of *Septoria nodorum* in wheat. 1. The effect of temperature and moisture treatments under controlled conditions. Netherlands Journal of Plant Pathology 78: 231-241.

Shrum, R., 1975. Simulation of wheat stripe rust (*Puccinia striiformis* West) using EPIDEMIC, a flexible plant disease simulator. Pennsylvania State University Agricultural Experiment Station Progress Report 347, 81 pp.

Snedecor, G.W. & Cochran, W.G., 1980. Statistical methods, 7th ed. Iowa State University Press, Ames, 507 pp.

Spitters, C.J.T. & Schapendonk, A.H.C.M., 1990. Evaluation of breeding strategies for drought tolerance in potato by means of crop growth simulation. Plant and Soil 123: 193-203.

Stephan, S., 1965. Untersuchungen zur *Phytophthora*-Prognose. Archiv für Pflanzenschutz 1: 99-129.

Stephan, S. & Gutsche, V., 1980. Ein algorithmisches Modell zur Simulation der *Phytophthora*-Epidemie (SIMPHYT). Archiv für Phytopathologie und Pflanzenschutz 16: 183-191.

Stolle, K. & Schöber, B., 1984. Wirkung eines Toxins von *Phytophthora infestans* (Mont.) de Bary

auf Kartoffelknollengewebe. Potato Research 27: 173-184.
Takase, N., 1968. Studies in breeding potatoes for resistance to *Phytophthora infestans* with special reference to laboratory assessment of resistance. Hokkaido National Agricultural Experiment Station Report 71, 118 pp.
Taylor, C.E., 1953. The vegetative development of the potato plant. Annals of Applied Biology 40: 778-788.
Teng, P.S., 1985. A comparison of simulation approaches to epidemic modeling. Annual Review of Phytopathology 23: 351-379.
Teng, P.S., Blackie, M.J. & Close, R.C., 1977. A simulation analysis of crop yield loss due to rust disease. Agricultural Systems 2: 189-198.
Thurston, H.D., 1971. Relationship of general resistance: Late blight of potato. Phytopathology 61: 620-626.
Tooley, P.W. & Fry, W.E., 1985. Field assessment of fitness of isolates of *Phytophthora infestans*. Phytopathology 75: 982-988.
Toxopeus, H.J., 1960. Studies on the resistance of tuber-bearing *Solanaceae* from Mexico to *Phytophthora infestans*. Euphytica 9: 39-56.
Ullrich, J., 1958. Die Tau- und Regenbenetzung von Kartoffelbeständen. Ein Beitrag zur Epidemiologie der Krautfaüle (*Phytophthora infestans*). Angewandte Botanik 32: 125-146.
Ullrich, J., 1976. Epidemiologische Aspekte bei der Krankheitsresistenz von Kulturpflanzen. Advances in Plant Breeding 6, Supplement to Journal of Plant Breeding. Parey, Berlin, 88 pp.
Umaerus, V., 1960. Some observations on field resistance to *Phytophthora infestans* [Mont.] de By.) in potatoes. Sveriges Utsädesförenings Tidskrift: 59-89.
Umaerus, V., 1963. Field resistance to late blight in potatoes. In: Akerberg and Hagberg (Eds), Recent plant breeding research Svalöf 1946-1961. Stockholm: 233-245.
Umaerus, V., 1969a. Studies on field resistance to *Phytophthora infestans*. 1. The infection efficiency of zoospores of *P. infestans* as influenced by the host genotype. Zeitschrift für Pflanzenzüchtung 61: 29-45.
Umaerus, V., 1969b. Studies on field resistance to *Phytophthora infestans*. 2. A method of screening young potato seedlings for field resistance to *P. infestans*. Zeitschrift für Pflanzenzüchtung 61: 167-194.
Umaerus, V., 1969c. Studies on field resistance to *Phytophthora infestans*. 4. Influence of mineral nutrition. Zeitschrift für Pflanzenzüchtung 62: 357-369.
Umaerus, V., 1970. Studies on field resistance to *Phytophthora infestans*. 5. Mechanisms of resistance and applications to potato breeding. Zeitschrift für Pflanzenzüchtung 63: 1-23.
Umaerus, V. & Lihnell, D., 1976. A laboratory method for measuring the degree of attack by *Phytophthora infestans*. Potato Research 19: 91-107.
Umaerus, V., Umaerus, M., Erjefält, L. & Nilsson, B.A., 1983. Control of *Phytophthora* by host resistance: Problems and progress. In: Erwin, D.C., Bartnicki-Garcia, S. and Tsao, P.H. (Eds), *Phytophthora*: Its biology, taxonomy, ecology and pathology. The American Phytopathological Society, St. Paul: 315-326.
Vanderplank, J.E., 1963. Plant diseases: Epidemics and control. Academic Press, New York, 349 pp.
Vanderplank, J.E., 1971. Stability of resistance to *Phytophthora infestans* in cultivars without R genes. Potato Research 14: 263-270.
Victoria, J.I. & Thurston, H.D., 1974. Light intensity effects on lesion size caused by *Phytophthora infestans* on potato leaves. Phytopathology 64: 753-754.
Vos, J. & Oyarzun, P.J., 1987. Photosynthesis and stomatal conductance of potato leaves - effects of leaf age, irradiance, and leaf water potential. Photosynthesis Research 11: 253-264.
Vowinckel, O., 1926. Die Anfälligkeit deutscher Kartoffelsorten gegenüber *Phytophthora infestans* (Mont.) de By. unter besonderer Berücksichtigung der Untersuchungsmethoden.

Arbeite der Biologische Reichsanstalt 14: 588-641. Review of Applied Mycology 6: 47-48.
Waggoner, P.E., 1968. Weather and the rise and fall of fungi. In: Lowry, W.P. (Ed.), Biometeorology. Oregon State University Press, Corvallis: 45-66.
Waggoner, P.E., 1990. Defoliation, disease and growth. In: Rabbinge, R., Goudriaan, J., Keulen, H. van, Penning de Vries, F.W.T. and Laar, H.H. van (Eds), Theoretical production ecology: Reflections and prospects. Simulation Monographs 34, Pudoc, Wageningen: 149-180.
Waggoner, P.E. & Berger, R.D., 1987. Defoliation, disease and growth. Phytopathology 77: 393-398.
Waggoner, P.E. & Rich, S., 1981. Lesion distribution, multiple infection, and the logistic increase of plant disease. Proceedings of the National Academy of Sciences USA 78: 3292-3295.
Warren, R.C., King, J.E. & Colhoun, J., 1971. Reaction of potato leaves to infection by *Phytophthora infestans* in relation to position on the plant. Transactions of the British Mycological Society 57: 501-514.
Warren, R.C., King, J.E. & Colhoun, J., 1973. Reaction of potato leaves to *Phytophthora infestans* in relation to their carbohydrate content. Transactions of the British Mycological Society 61: 95-105.
Weihing, J.L. & O'Keefe, R.B., 1962. Epidemiological potentials of potato varieties in relation to late blight. Phytopathology 52: 1268-1273.
Wenzl, H., 1967. Histologische Studien über Befall durch *Phytophthora infestans* an Kartoffelstengeln. Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz 74: 533-539.
Wilson, U.E. & Coffey, M.D., 1980. Cytological evaluation of general resistance to *Phytophthora infestans* in potato foliage. Annals of Botany 45: 81-90.
Wit, C.T. de et al., 1978. Simulation of assimilation, respiration and transpiration of crops. Pudoc, Wageningen, 148 pp.
Zaag, D.E. van der, 1956. Overwintering en epidemiologie van *Phytophthora infestans*, tevens enige nieuwe bestrijdingsmogelijkheden. Tijdschrift voor Plantenziekten 62: 89-156.
Zaag, D.E. van der, 1959. Some observations on breeding for resistance to *Phytophthora infestans*. European Potato Journal 2: 278-286.
Zadoks, J.C., 1971. Systems analysis and the dynamics of epidemics. Phytopathology 61: 600-610.
Zadoks, J.C., 1977. Simulation models of epidemics and their possible use in the study of disease resistance. In: International Atomic Energy Agency (Ed.), Induced mutations against plant diseases: Proceedings of a symposium, Vienna: 109-118.
Zadoks, J.C. & Schein, R.D., 1979. Epidemiology and plant disease management. Oxford University Press, New York, 427 pp.

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CURRICULUM VITAE

Marcel van Oijen werd op 17 augustus 1959 te Nijmegen geboren. Na het Gymnasium-ß begon hij in 1977 met de studie biologie aan de Rijksuniversiteit Utrecht. In 1981 behaalde hij het kandidaatsdiploma in de mathematische biologie. In 1985 slaagde hij *cum laude* voor het doctoraalexamen met als hoofdvak oecofysiologie en als bijvakken wijsbegeerte van de biologie, theoretische teeltkunde en informatica. Het hoodfvak oecofysiologie, uitgevoerd onder begeleiding van R. Brouwer, betekende voor hem een eerste kennismaking met de bijzondere toegankelijkheid van landbouwkundige systemen voor mathematische analyse en modelontwikkeling. In 1985 werd hij aangesteld op de Stichting voor Plantenveredeling (SVP, tegenwoordig onderdeel van het CPRO) en assisteerde bij de ontwikkeling van simulatiemodellen in het onderzoek van C.J.T. Spitters. In 1986 volgde de aanstelling tot onderzoeker op de SVP die tot dit proefschrift heeft geleid. Sinds juli 1990 werkt hij op het Centrum voor Agrobiologisch Onderzoek (CABO) aan de ontwikkeling van simulatiemodellen voor de effekten van aardappelcysteaaltjes op fysiologie en opbrengstvorming bij aardappel.