Disease management of leek rust,

a study at field, farm and regional level

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Disease management of leek rust,

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1. Geleide bestrijding van preiroest kan alleen in combinatie met preventieve maatregelen tot een reductie in het gebruik van chemische bestrijdingsmiddelen tegen preiroest leiden.

2. Certificatie van plantmateriaal voor de preiteelt is noodzakelijk om de verspreiding van preiroest tegen te gaan.

3. Een succesvolle beheersing van planteziekten in de landbouw is alleen mogelijk bij voldoende aandacht voor de regionale aspecten van het pathosysteem.

4. Een mogelijke reductie van 20 % in de kilo opbrengst per hectare is geen werkelijke belemmering om klaverondergroei als plaagonderdrukkende maatregel in de preiteelt toe te passen.

5. Natuurwaarde uitgedrukt in biodiversiteit neemt voor planteziekten en plagen in Nederland voortdurend toe.

6. Ziekten en plagen spelen een even grote rol in natuurlijke vegetaties als in landbouwgewassen.

7. Beschermende maatregelen ten aanzien van potentiële plaagsoorten zoals aalscholver en bever, dienen regelmatig kritisch overwogen te worden.

8. Natuurbouw is grootschalig tuinieren.

9. Toename van het gebruik van elektronisch geld (chipkaart, cybermoney) zal de greep van de Centrale Bank op de nationale geldvoorraad verder doen afnemen.

10. Het internationale protest tegen de hervatting van de Franse kernproeven zomer 1995 heeft deze kernproeven waardevoller gemaakt voor de Franse regering.

11. Vergelijking van het huidige Europese drugsbeleid met de drooglegging in de Verenigde Staten leert ons, dat empirie in de politiek een ondergeschikte rol speelt.

P.D. de Jong
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Wageningen, 17 november 1995

Contents

Abstract

Leek rust, caused by *Puccinia allii* Rud., is an important disease of leek (*Allium porrum* L.) in the Netherlands. The quality decrease of the product associated with a light infestation of leek rust may cause severe economic loss to the grower. The aim of this study is to develop an improved pathosystem management for leek rust at field, farm and regional level with low use of fungicides, through preventive measures and supervised control based on sampling for detection.

Basic temporal and spatial characteristics of leek rust epidemics were studied under field conditions and after natural infection. The spatial distribution of diseased plants during three naturally occurring epidemics of leek rust was analysed with the Black-White join-count statistic. The spatial distribution of rust-infected leek plants was different for each of the three epidemics, ranging from random to highly clustered. The growth of leek rust epidemics in time under favourable conditions in three leek cultivars during two years was studied. In both years, the highest disease levels were found on cultivar Albana, followed by Carina and Cortina. A simple model is presented to correct the results for exchange of inoculum between adjacent plots. The results of this model indicate, that the difference in rust infection between the cultivars may be due to a reduced growth of the epidemic in young plants of cultivars Cortina and Carina. In older plants, the ranking in susceptibility was reversed, causing a less pronounced difference in infection between the cultivars. The growth of leek rust epidemics during the early stage of the epidemic in isolated plots was satisfactorily described by an exponential model.

Monte Carlo simulation was used to compare simple sampling patterns in their efficiency of detecting diseased plants under a variety of spatial distributions. Row sampling over four rows has been identified as the most effective sampling pattern. The detection level, i.e. the lowest disease level at which the probability to detect the disease is sufficiently large, was studied in relation with temporal and spatial characteristics of the disease. The calculations indicate, that a weekly sample of 50 plants resulted in detection levels, that were low enough to ensure adequate chemical control, indicating the feasibility of sampling for detection of leek rust.

A model is presented to simulate the number of fungicide sprays against leek rust under the proposed system of supervised control, including the spread of the disease between fields in one farm and the influx of spores from outside the farm. The possible reduction in fungicide sprays against leek rust due to various preventive measures are estimated with this model. The results of this preliminary model suggest, that supervised control of leek rust can result in a considerable reduction in the number of fungicide sprays, but only if the leek fields in a farm are sufficiently isolated and care is taken that the planting material is free from the disease. If it is not possible to increase the isolation between the fields, overlap between successive crops should be avoided.

Based on a simple metapopulation model, it is argued, that the possible effect of certification of planting material on the occurrence of leek rust in a region can be derived from information on the proportion of infected fields (v) and the proportion of newly planted fields with infected planting material (i) in that region. If $v \leq \sqrt{i}$, certification of planting material will be highly effective and may even cause the complete eradication of leek rust from the region.

A differentiated regional policy of leek rust management is proposed, dependent on the intensity of leek cultivation in a region. To validate the proposed strategies on field, farm and regional level, a survey of the actual disease situation in several farms in different regions is necessary. This study indicates the importance of parameters, that are rarely measured in disease surveys, such as the distance between crops in a farm and in a region and the infection level of planting material.

1 Introduction

1.1 Leek and leek rust

The origin of cultivated leek is the Mediterranean region. In western Europe, leek cultivation can be traced back to the middle ages (Bonnet, 1976; Shakespeare, 1599). Cultivated leek belongs to a very variable species, *Allium ampeloprasum* L.. Wild taxa from this species occur in the Mediterranean region and southwest Asia. *A. ampeloprasum* L. var *porrum* L. and *A. porrum* L. are both valid synonyms for cultivated leek (van der Meer and Hanelt, 1990).

Leek cultivation in the Netherlands has increased considerably in recent years and leek is now an important vegetable crop (Table 1). The acreage under leek cultivation is concentrated in the two southern provinces Noord-Brabant and Limburg. The increase in leek production has been mainly absorbed by the export market, nearly half of the production is now exported. A strict grading system is used to maintain high cosmetic quality for the exported leek.

Table 1. Area of leek cultivation in the Netherlands, percentage of area in the southern provinces Noord-Brabant (NB) and Limburg (L), and export of leek from the Netherlands (source: [1]Central Bureau of Statistics, Nl., [2]PGF; [3]estimated values)

year	area[1] ha	% area[1] NB	% area[1] L	production[2] x10^6 kg	value[2] x10^6 Dfl	export[2] % total production (kg)
1975	1285	48	28	47.6	22.0	15
1980	1644	53	26	51.0	38.2	14
1985	2458	55	29	57.6	67.7	21
1986	2577	56	30	69.7	56.7	22
1987	2642	56	31	63.8	70.1	25
1988	2933	57	32	79.9	64.8	30
1989	2808	56	34	82.2	74.0	36
1990	2873	54	36	94.3	94.9	43
1991	3552	51	38	98.8	117.5	46
1992	4117	49	40	114.3	109.7	49
1993	3934	49	41	121.0	107.7	47
1994	4250	48	42	105.6	120.4	44
1995[3]	3958	48	43	120.0	96.0	not available

The following pests and diseases can cause a strong reduction in the cosmetic quality of leek: onion thrips (*Thrips tabaci* Lindeman), leek moth (*Acrolepiopsis assectella* Zeller), white tip disease (*Phytophthora porri* Foister) and leek rust (*Puccinia allii* Rud.). This thesis focuses on leek rust. Leek rust decreases the quality of leek by forming orange pustules on leaves and shafts. At high infection levels, leading to an unmarketable product, also reduction in photosynthesis and growth is observed (Roberts and Walters, 1988).

The rusts (Uredinales) are biotrophic, mostly specialized plant parasites with an usually complex life cycle. The following terms are used to describe the life cycle. Spermagonia, aecidia, uredosori and teleutosori are spore-bearing structures representing different, sometimes overlapping, reproductive stages in the life cycle. Many rust species require two distinct host species to complete their life cycle. This condition is named heteroecism. The host species bearing the teleuto stage is called the primary host and the other is known as the alternate host. The so-called autoecious rust species can complete their life cycle on one host species.

The seven rust species, reported on *A. porrum* L. or *A. ampeloprasum* L., can be divided in three groups (Table 2) (Gäumann, 1959). Only the first group of autoecious rusts is causing damage to leek crops. The taxonomic difference between these two autoecious rusts are based on differences in the teleuto stage, that are not always very

Table 2. Rust species occurring on *A. porrum* L. and *A. ampeloprasum* L., host record and nomenclature based on Gäumann (1959). Species described on *A. porrum* L. are indicated with *, species described on *A. ampeloprasum* L. are indicated with +.

rust species	spermagonia	aecidia	uredosori	teleutosori
group I autoecious on *Allium* spp.				
Puccinia allii Rud.*	?	?	*Allium* spp.	*Allium* spp.
Puccinia porri (Sow.) Wint.*+	?	*Allium* spp.	*Allium* spp.	*Allium* spp.
group II heteroecious, primary host *Salix* spp.				
Melampsora allii-fragilis Kleb.+	*Allium* spp.	*Allium* spp.	*Salix* spp.	*Salix* spp.
Melampsora allii-salicis albae Kleb.+	*Allium* spp.	*Allium* spp.	*Salix* spp.	*Salix* spp.
group III heteroecious, primary host Gramineae				
Puccinia Fragosi Bubák*	*Allium* spp.	*Allium* spp.	*Koeleria* spp.	*Koeleria* spp.
Puccinia laguri-Chamaemoly Maire*	*Allium* spp.	*Allium* spp.	*Lagurus* sp.	*Lagurus* sp.
Puccinia Winteriana Magnus*	*Allium* spp.	*Allium* spp.	*Phalaris* spp.	*Phalaris* spp.

distinct. Laundon and Waterston (1965) and Wilson and Henderson (1966) consider them therefore as one species, *Puccinia allii* Rud.. Furthermore, the very rare occurrence of teleutosori on *A. porrum* L. in the Netherlands makes an identification on the basis of the teleuto stage impossible. Boerema et al. (1993) propose *P. allii* Rud. as the scientific name for the autoecious rust infecting leek in the Netherlands and this name is also used in this thesis.

The geographical distribution of *P. allii* Rud. includes Africa, Asia, Australasia, Europe, North and South America (Laundon and Waterston, 1965). *P. allii* Rud. has a wide host range within the genus *Allium* and there is an indication of the existence of specialized races within the rust species (Jennings et al., 1990a). Other crops that can be infected by *P. allii* Rud are onion (*A. cepa* L.), garlic (*A. sativa* L.) and Welsh onion (*A. fistulosum* L.), which is a Japanese vegetable comparable to leek (Jennings et al., 1990a; Takeuchi, 1990). In the Netherlands, rust is a rare disease in onions. *P. allii* Rud. isolates from leek can infect *A. vineale* L. (Jennings et al., 1990a). *A. vineale* L. is a common wild plant in the Netherlands. Rust isolates from wild plants of *A. vineale* L. in the Netherlands are found to be not infective on leek (Niks, personal communication) and are described as belonging to a different rust species, *P. mixta* Fuckel (Niks and Butler, 1993).

The first report of rust on *Allium* spp. in the Netherlands dates back to 1844 (Dozy and Molkenboer, ex Calkoen, 1883), referring to uredospores and teleutospores of a rust named *P. allii* Desm. on *A. cepa* L., *A. sativa* L., *A. scorodoprasum* L. and *A. vineale* L.. No reports of rust on leek were available in 1883 (Calkoen). In the Netherlands, only the uredo stage plays a role in the epidemiology of leek rust. Sometimes, a few teleutosori can be observed in heavily infected leek crops after a period of warm weather during summer. Aecidia have not been found in the Netherlands.

In the Netherlands, leek cultivation is continuous over the year, with ample overlap between the different crops. The early summer crops are planted in april and harvested in july. The so called late winter crops are planted in july and harvested in may the next year. The uredo stage of leek rust is found around the year in leek crops. In autumn and winter, the growth in number of uredosori stops due to low temperature and the number of uredosori drops due to leaf death. The disease is perfectly able to survive low temperatures during winter in infected leek plants (cf. Gäumann, 1959). As soon as temperature increases during spring, the epidemic of leek rust will start again in late winter crops, causing damage to the crop and providing inoculum to newly planted crops. This crop hopping is of paramount importance for the survival of the pathogen, because resting spores and other host species seem to play a minor role in its epidemiology.

There is a large number of leek cultivars, often grouped according to the cropping period. Each group of cultivars has specific characteristics, such as fast growth for early summer cultivars and hardiness for late winter cultivars. At present, there are no complete rust-resistant leek cultivars, but differences in susceptibility have been found among cultivars in the United Kingdom (Uma and Taylor, 1991; Smith and Crowther, 1992), Germany (Albert and Smolka, 1994) and the Netherlands (Chapter 3).

In 1973, fungicides against leek rust became available and now there are 5 compounds admitted to control leek rust. The four more recently admitted compounds are highly effective ergosterol biosynthesis inhibitors (Table 3). The costs of a treatment is between 150 and 350 Dfl per ha, including machinery and labour and depending on the fungicides used.

The off-farm value of a leek crop of good cosmetic quality is about 30.000 Dfl per ha. The presence of ten or twenty rust pustules per plant causes this value to drop sharply, to be halved or even worse. Farmers apply chemical control on a regular basis, mostly every two weeks, totalling 6 to 8 sprays per crop.

Table 3. Fungicides registered for use against rust in leek in 1994

compound	first registered for use against leek rust
chlorothalonil	1981
penconazole	1987
fenpropimorph	1993
triadimenol	1993
propiconazole	1994

1.2 Aim and approach

The aim of this study is to develop an improved pathosystem management for leek rust at field, farm and regional level, with a strong emphasis on preventive measures and low use of fungicides. A prerequisite for such a strategy is, that it must yield a sufficient cosmetic quality of leek, i.e. no more than a few rust pustules per plant can be tolerated. To secure this quality, chemical control of the disease has to be started in an early stage of the infection. Given this condition, a reduction in the use of chemical control can still be pursued in two ways. First, by chemical control which is only applied, when the disease is observed in the crop. Secondly, through preventive measures to prevent or delay the infection of a crop.

Application of fungicides to a crop in absence of the disease is undesirable both from an environmental and an economic point of view. To prevent unnecessary use of pesticides, detailed monitoring for presence of the disease is necessary. The question is here, whether it is practically feasible to detect the disease before the infection has reached an intolerable level. The early phase of plant disease epidemics often escapes observation, leaving the suggestion of a suddenly occurring, aggressive disease, while the disease was in fact already present in the crop for several weeks, multiplying at a steady, moderate rate (cf. Zadoks and Schein, 1979). To analyse the question of early presence of leek rust and the possibilities of detailed observations in that phase, research on the spatial distribution and the growth in time of the disease at the start of an epidemic of leek rust is done (Chapters 2 and 3).

An important characteristic, determining the feasibility of sampling for detection, is the spatial distribution of diseased plants (Chapter 2). The spatial pattern of diseased plants in a field can be highly complex. A wide range of statistical techniques are available to analyse spatial patterns. Some of the techniques generate complex maps and graphs. Other techniques describe the spatial pattern in more general terms as random or clustered. An appropriate statistical technique has to be applied to the spatial pattern of naturally occurring leek rust epidemics. This technique must have a low demand for data and must generate a reliable summary of the observed spatial patterns, that is useful to study the effect of spatial pattern on the feasibility of sampling for detection.

The other basic characteristic determining the feasibility of sampling for detection, is the growth in time of leek rust epidemics (Chapter 3). The observations on the growth of an epidemic consist of series of data obtained from sampling and often showing considerable variation. To use these observations as a basis for predictions of the growth of an epidemic, a growth model is applied to the observations and attention is given to the problem of parameter estimation. The early growth of a leek rust epidemic may differ between leek cultivars. The measurement of this difference is hampered by the exchange of inoculum between adjacent experimental plots. To study such differences, different cultivars are included in the experiments and a growth model is used, that incorporate the exchange of inoculum between experimental plots.

A sampling method for detection of leek rust is practically feasible, if it combines speed and simplicity with a sufficiently large probability to detect the disease before an intolerable level is reached (Chapter 4). An optimal balance between these conflicting goals has to be found by careful selection of the sampling pattern, sampling frequency and sample size. As it is impossible to test all kinds of combinations in practice, it is better to derive the optimal combination of sampling pattern, sampling frequency and sample size from a framework of mathematical equations based on observed basic

spatial and temporal characteristics. This approach, commonly described as systems analysis (Zadoks and Schein, 1979; Rabbinge et al, 1989), has as additional advantage, that the resulting mathematical framework can be used to solve similar problems in other pathosystems, often by just adapting the parameters.

The effect of preventive measures on the need for chemical control is another field of research, that is not easily accessible by experimental research (Chapter 5). Different levels of the spread of the disease between the fields on a farm, as influenced by the distance between the fields, have to be included in the experiments. Also, the disease occurrence has to be observed during a number of years, because reduction in the use of fungicides during one year may lead to the build-up of inoculum and hence to an increased use of fungicides in following years. Thus large scale, multi-year experiments are necessary. A less costly alternative is a survey of leek fields, but observations have to cover even a longer period to rule out exceptional years. As an alternative to expensive experimental research or a lengthy survey, the feasibility of preventive measures on the farm level is studied following the systems approach in Chapter 5.

Apart from disease management on the farm level, preventive measures can also play a role in the disease management on a regional level. The feasibility of preventive measures on a regional level are discussed in Chapter 6. A simple theoretical metapopulation model is used to study the effect of strict certification of planting material on the disease occurrence on a regional level.

In Chapter 7, the methodology used across the chapters is discussed, i.e. the relation between the epidemiological data obtained from the experiments and their use in the models. The practical implications of this study for pathosystem management at field, farm and regional level are discussed and a differentiated regional policy of leek rust management is proposed. Recommendations for further research are given, with emphasis on new approaches for disease management on a regional level.

2 Analysis of the spatial distribution of rust-infected leek plants with the Black-White join-count statistic

P.D. de Jong and J. de Bree
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Abstract

Leek rust, caused by *Puccinia allii* Rudolphi, is an important disease of leek (*Allium porrum* L.) in the Netherlands. For the development of a practical sampling method for early detection of leek rust in commercial fields, information on the spatial distribution of the disease is necessary. In this study, the spatial distribution of diseased plants during three naturally occurring epidemics of leek rust was observed. The observations were analysed with the Black-White join-count statistic. The spatial distribution of rust-infected leek plants was different for each of the three epidemics, ranging from random to highly clustered. These results show, that in the development of a practical sampling method for detection of leek rust, it is necessary to take into consideration a possibly clustered distribution of diseased plants.

Keywords: leek, *Allium porrum*, leek rust, *Puccinia allii*, spatial distribution, Black-White join-count statistic

2.1 Introduction

Rust of leek, caused by *Puccinia allii* Rudolphi, is an important disease of leek (*Allium porrum* L.) in The Netherlands. Decline in leek quality due to light infestation may cause severe economic loss to the grower. Therefore, chemical control of the disease must be started in an early stage of the infection. However, applying fungicides to a crop in the absence of the disease, is undesirable both from an environmental and an economic point of view. The most direct method of assessing the presence of the disease in the crop is sampling. For the development of a practical sampling method for early detection of leek rust in commercial fields, general information on the possible spatial distribution of the disease is necessary.

A straightforward and much used method for analysis of the spatial distribution of diseased plants is to assess presence or absence of the disease in all plants in a row

or in a transect across rows and to use the ordinary runs statistic to test randomness of infected plants. (Campbell et al, 1984; Madden et al, 1982, 1987a, 1987b; Jeger et al, 1987). The ordinary runs statistic is a particular join-count statistic. Join-count statistics are widely applied measures of spatial autocorrelation for nominal data (Cliff and Ord, 1981). Another join-count statistic, the Black-White statistic (BW) has a flexibility not present in the ordinary runs statistic that permits application to a two-dimensional array of plants (e.g. a set of rows) and to detect spatial autocorrelation not only between nearest neighbours, but also between second and higher order neighbours.

Gray et al (1986) proposed a method for spatial analysis of nominal data very similar to the use of join-count statistics, that they called two-dimensional distance class analysis, without referring to the join-count statistics. The two-dimensional distance class analysis as presented by Gray et al (1986) requires computer simulation to obtain the probability distribution of the test statistic, whereas the probability distributions for the join-count statistics can be approximated with standard distribution functions (Cliff and Ord, 1981).

In this chapter, the BW join-count statistic is used to detect non-randomness in the spatial distribution of rust-infected leek plants in naturally occurring epidemics.

2.2 Materials and methods

2.2.1 Fields observations

The spatial distribution of diseased plants was assessed during three naturally occurring leek rust epidemics in fields nearby Wageningen: on 18 September 1991 in a 60 m x 45 m field, on 3 July 1992 in a 30 m x 35 m field and between 8 July and 6 August 1993 in a 30 m x 50 m field. The fields received treatment with pyrethroids to control insect damage but were not treated with fungicides. All fields were row planted with the highly susceptible cultivar Albana. Details on the fields are given in Table 4. Presence or absence of rust was assessed on consecutive plants within selected rows. Distances between selected rows were 5 rows in 1991 and 10 rows in 1992 and 1993. In 1992 and 1993 plants in transects across rows were also assessed; the distance between the transects being 5 m and 10 m respectively. In 1993, observations on the different dates were made on the same rows and approximately the same transects. The number of observed rows, transects and plants per row or transect are given in Table 4.

Table 4. Dimensions of the fields, between and within row planting distances and the number of observed rows, transects and plants per row or transect for the three observed epidemics

	1991	1992	1993
dimensions (mxm)	60x45	30x35	30x50
row length (m)	45	35	50
between row planting distance (m)	0.50	0.50	0.50
within row planting distance (m)	0.16	0.16	0.16
number of observed rows	22	3	5
number of plants per row	270	210	240
number of observed transects	-	4	5
number of plants per transect	-	56	54

2.2.2 Statistical analysis

Randomness in the one-dimensional spatial distribution of diseased plants was tested with the BW join-count statistic (Cliff and Ord, 1981). The mathematical definition of the BW statistic is:

$$BW = \tfrac{1}{2}\sum_{i=1}^{n}\sum_{j=1}^{n}\delta_{ij}(x_i - x_j)^2 \qquad (1)$$

in which $x_i = 1$ if plant i is infected and $x_i = 0$ if plant i is healthy (i=1...n), and $\{\delta_{ij}; i,j=1...n\}$ form a binary connection matrix, in the sense that $\delta_{ij} = 1$ if the plants i and j are defined to be neighbours, and $\delta_{ij} = 0$ otherwise (including $\delta_{ii} = 0$). In formula (1), the sum of squares is taken to avoid negative values and the factor ½ is added, because every possible pair of plants occurs twice in the summation. Thus, the BW statistic is the number of times, that a healthy plant is neighboured by an infected plant. It must be emphasized, that with the BW statistic, neighbourship is merely a matter of definition. For calculation of the BW statistic for spatial autocorrelation between k^{th} order neighbouring plants, plants are regarded as neighbours if there are k-1 plants in between.

The data presented in this study, consist of sets of rows (or transects) with no spatial connection between the rows (or transects). When calculating the BW statistic for first order neighbours, $\delta_{ij} = 1$ if plant i and plant j are in the same row (or transect) and there are no other plants in between and $\delta_{ij} = 0$ otherwise. For second order

neighbours, $\delta_{ij} = 1$ if plant i and plant j are in the same row (or transect) and there is only one plant in between and $\delta_{ij} = 0$ otherwise. For third order neighbours, $\delta_{ij} = 1$ if plant i and plant j are in the same row (or transect) and there are two other plants in between and $\delta_{ij} = 0$ otherwise, etc.

The BW statistic is asymptotically normally distributed under the null hypothesis of no spatial autocorrelation. An approximate test of significance is therefore provided by evaluating the BW statistic as a standard normal deviate, as suggested by Cliff and Ord (1981). The first two moments of the BW statistic are then used to specify mean (μ) and variance (σ^2) of the normal distribution. Computational formulae for the first two moments are given in Appendix 1. The appropriateness of the use of the normal distribution as an approximation of the probability distribution of the BW statistic has been confirmed by the results of Monte Carlo simulation, using 5 rows with each 50 plants and combinations of disease frequency of 5 % or higher with order of neighbouring ranging from one to 25.

For every set of rows (or transects), BW statistics were calculated for any order of neighbouring less or equal to half the length of the row (or transect). With even higher order of neighbouring, the normal approximation of the probability distribution of the BW statistic appears to be less reliable. A left-sided test was applied to detect low values of the BW statistic, that indicate positive spatial autocorrelation between diseased plants (i.e. clustering) and a significance level of 0.05 was used. Characterization of the spatial distribution of diseased plants in a set of rows (or transects) as random or clustered was based on the outcome of the BW statistic for first order neighbouring. The BW statistics for higher order neighbouring were used to obtain an indication for the extent of the clustering.

2.3 Results

For all three years, observations were made when the number of pustules per infected plant was low and infection could only be perceived by close observation. Despite the low numbers of pustules per plant, there was a considerable percentage of the plants infected and diseased plants could be found throughout the fields.

The results of the analysis of the spatial distribution of diseased plants are given in Table 5. The frequency of infected plants was 30.4 % for the observation on the '91 epidemic and 25.5 % for the '92 epidemic, as estimated from the row observations. For the '93 epidemic, the frequency of infected plants increased from 6.2 % to 81.3 %, between 8 July and 6 August, as estimated from the row observations.

In the '91 epidemic, positive spatial autocorrelation within rows was significant

for every order neighbouring tested, indicating a strongly clustered distribution of diseased plants. In the '92 epidemic, there was no significant positive autocorrelation within transects and significant positive spatial autocorrelation within rows was found for 13th, 21st, 35th, 36th, 43rd and 96th order neighbouring, but not for first order neighbouring. Therefore the spatial distribution was considered to be random both for rows and for transects. In the '93 epidemic, the number of cases of significant positive spatial autocorrelation within the sets of rows increased from 14 on 8 July to 65 on 22 July, always including first order neighbouring, and the spatial distribution of diseased plants along rows was therefore clearly clustered. The spatial distribution of diseased plants along the transects was also clustered for the whole period of observation.

Table 5. Frequency of diseased plants in the sample (f), the number of tests done (t), the number of cases of significant positive spatial autocorrelation (α = 0.05) in the set of tests (s) and the order of neighbouring (k) over which significant positive spatial autocorrelation occurred

			f(%)	t	s	k
1991	18 Sep	rows	30.4	135	135	1-135
1992	3 Jul	rows	25.5	105	6	13,21,35,36,43,96
		trans	24.1	28	-	-
1993	8 Jul	rows	6.2	120	14	1-4,6,13,14,21,22,67,73,81,115,117
		trans	7.0	27	2	1,3
	12 Jul	rows	6.8	120	21	1-5,7,9,11-14,16,18-22,24,25,27,29
		trans	12.2	27	3	1,2,16
	16 Jul	rows	7.8	120	40	1-11,16-19,21,25,27,28,38,71,74,92,94-100,102-104, 106,108,113-115,119,120
	22 Jul	rows	16.0	120	19	1-10,12,14,35,40,82,90,94,95
		trans	14.4	27	5	1-5
	31 Jul	rows	39.3	120	28	1-24,27,96,106,117
		trans	46.3	27	11	1,2,4-9,12,13,20
	6 Aug	rows	81.3	120	65	1-23,25-30,32-37,40,41,43-50,52,55,61,63-65,67,73, 76,81,83,84,86,87,90,92,93,103,119,120
		trans	86.3	27	4	1,6,7,12

2.4 Discussion

The fields analysed in this study consisted of at least 10.000 plants. Therefore the fields could not be assessed completely, but had to be sampled. The numbers of rows and transects included in the observations in this study were the largest possible, given the limitations in man power. The rows and transects were chosen to be evenly distributed over the fields. For efficient use of the BW statistic for characterization of spatial patterns, more information is needed on the minimal sample size that still give representative results.

Although the BW join-count statistic permits analysis of two-dimensional spatial data, the use of the statistic was restricted in this study to one-dimensional analysis, due to the character of the fields. Gray et al (1986) present examples of two-dimensional spatial analysis, but their examples are limited to small latices of at most 300 elements. An alternative way of sampling, i.e. assessment of all plants in a number of square patches in the field, will allow for a two-dimensional analysis, but will restrict the analysis to a low order of neighbouring. An additional problem is, that the leek plants were row-planted and not standing in an exact lattice.

Simultaneous statistical inference using BW statistics for several order of neighbouring poses intractable problems because of mutual dependence of the individual BW statistics. Therefore, no test for randomness using several BW statistics for different order neighbouring simultaneously can be constructed and characterization of the spatial distribution as random or clustered has to be based on the BW statistic for one particular order of neighbouring only, the first order neighbouring being a logical choice. The BW statistics for higher order neighbouring provide an indication for the extent of the clustering. The presence of significant positive spatial autocorrelation for first till k^{th} order neighbouring can be interpreted as an indication of cluster size. However, the significance level of such complex conclusions remains undefined, due to the already mentioned mutual dependence of the individual BW statistics.

The strong clustering found during the 1991 epidemic can be explained by the nature of the source of inoculum. In 1991, the sampled field was infected from a nearby source, consisting of a 5 m x 7 m leek plot at a distance of 5 m from the border of the field. It was in the vicinity of this point source, that there was a very large cluster of infected plants, reflected in the analysis by the presence of significant spatial autocorrelation for every order of neighbouring tested. Such a point source may occur in a commercial setting, when part of a previously planted neighbouring crop is left unharvested.

In 1992 and 1993, the sampled fields were infected from a source field measuring 15 m x 40 m at a distance of approximately 25 m and planted one month

earlier than the observed fields. As the sources of inoculum were so identical for 1992 and 1993, other factors must have caused the difference in spatial distribution found in the 1992 and the 1993 epidemic. As leek rust is wind-borne, wind-direction and wind-speed are other important factors determining the spatial distribution of diseased plants. However, the data presented in this study are not detailed enough to draw any conclusion on the effect of wind-direction and wind-speed on the spatial distribution in the observed epidemics.

From the results of this study, it is clear, that the spatial distribution of rust-infected leek plants is highly variable, depending among other factors on the nature of the inoculum source.

In Chapter 4, the effect of the spatial distribution of the diseased plants on the efficacy of different sampling patterns for detection of leek rust will be discussed, and a practical sampling pattern is proposed with a high efficacy even under strongly clustered distribution of diseased plants.

3 Growth of leek rust epidemics in time in three cultivars during the early stage of the epidemic

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Abstract

The growth of leek rust epidemics in time under favourable conditions in three leek cultivars during two years was analysed. In both years, the highest disease levels were found on cultivar Albana, followed by Carina and Cortina. A simple model is presented to correct the results for exchange of inoculum between adjacent plots. The results of this model indicate, that the difference in rust infection between the cultivars may be due to a reduced growth of the epidemic in young plants of cultivars Cortina and Carina. In older plants, the ranking in susceptibility was reversed, causing a less pronounced difference in infection between the cultivars. The growth of leek rust epidemics during the early stage of the epidemic in isolated plots was satisfactorily described by an exponential model.

Keywords: leek, *Allium porrum*, leek rust, *Puccinia allii*, relative growth rate, sampling, epidemic

3.1 Introduction

Rust of leek, caused by *Puccinia allii* Rudolphi, is an important disease of leek (*Allium porrum* L.) in the Netherlands. *P. allii* is a macrocyclic, autoecious rust (Laundon and Waterston, 1966). In the Netherlands, only the uredo stage plays a role in the epidemiology of leek rust. Sometimes, a few teleutosori can be observed in heavily infected leek crops after a period of warm weather during summer. Aecidia have not been found in the Netherlands.

In the Netherlands, leek cultivation is continuous over the year, with ample overlap between the different crops. The early summer crops are planted in April and harvested in July. The so called late winter crops are planted in July and harvested in May the next year. The uredo stage of leek rust is found around the year in leek crops.

In autumn and winter, the growth in the number of uredosori is stopped due to low temperature. As soon as temperature increases during spring, the epidemic of leek rust will start again in late winter crops, causing damage to the crop and providing inoculum to newly planted crops.

There is a large number of leek cultivars, often grouped according to the cropping period. Each group of cultivars has specific characteristics, such as fast growth for early summer cultivars and hardiness for late winter cultivars. Although there is no complete resistance against leek rust at present, differences in susceptibility have been found among cultivars in the United Kingdom (Uma and Taylor, 1991; Smith and Crowther, 1992).

The quality decrease of the product associated with a light infestation of leek rust may cause severe economic loss to the grower. Therefore, chemical control of the disease has to be started in an early stage of the infection. However, applying fungicides to a crop in the absence of the disease is undesirable both from an environmental and an economic point of view. To prevent unnecessary use of pesticides detailed monitoring for presence of the disease is necessary. To assess the presence of the disease in the crop before the disease has increased beyond an intolerable level, frequent sampling is needed and the interval between two samplings should not be taken too long. For the determination of a safe sampling interval, information on the growth of an epidemic of leek rust in time during the early stage of the epidemic is required.

The aim of this chapter is to derive a simple predictive model for the early stage of the epidemic to support the development of a sampling method. The growth of leek rust epidemics under favourable conditions in three cultivars during two years was analysed. A simple model was constructed to correct the results for exchange of inoculum between adjacent plots.

3.2 Materials and methods

3.2.1 Field observations

Naturally occurring leek rust epidemics were observed in two experiments during summer 1992 and 1993 on a sandy soil nearby Wageningen. In both experiments the same three cultivars were used: Albana, Cortina and Carina. Albana is a fast growing cultivar for harvest in September, Cortina is a cultivar for harvest in late autumn and Carina is a hardy cultivar for spring harvest. In the 1992 experiment, half of the experiment including all three cultivars was planted on 21 may, the other half of the experiment, also including the three cultivars, was planted on 2 July. In the 1993

experiment, there was one planting date: 14 May. For every planting date, the planting material was homogeneous with respect to age and size.

The 1992 experiment consisted of 24 plots, measuring 8 m x 12.5 m. The six combinations of cultivar and planting date were distributed over 4 blocks in a complete randomized block design. The 1993 experiment consisted of 6 isolated plots measuring 10 m x 10 m. The plots were surrounded by a barley crop. The distance between plots was 25 m. The six plots were planted with successively Albana, Cortina and Carina and a repetition of this order. Both experiments were row planted.

The increase of the epidemic was measured by estimating the number of uredosori per plant on randomly selected plants. During the 1992 experiment, the growth of the epidemic was observed weekly from 5 August till 16 September. During this period, there was a considerable difference in growth stage between the two planting dates, as was intended. The plants of the first planting had about two times as much leaves as the plants in the second planting. The epidemic increase in the 1993 experiment was monitored weekly from 28 July till 1 September. In the 1992 experiment, 24 plants per plot were observed. In the 1993 experiment, 1000 plants per plot were observed on 28 July, 4 August and 11 August, 200 plants per plot on 18 August and 100 plants per plot on 25 August and 1 September. The higher number of plants observed in the 1993 experiment was necessary due to the very low percentage of infected plants at the begin of the observations. Details on the experiments are given in Table 6.

Table 6. Details on the 1992 and the 1993 field experiments

	1992	1993
Between row distance (m)	0.50	0.50
Within row distance (m)	0.16	0.16
Plot size (m x m)	8 x 12.5	10 x 10
Total number of plots	24	6
Between plot distance (m)	0	25
Planting date	21-5	14-5
	2-7	
Cultivars	Albana	Albana
	Cortina	Cortina
	Carina	Carina
Observation period	5-8 - 6-9	28-7 - 1-9
Number of plants observed per plot	24	1000 - 100

3.2.2 Mathematical modeling

Growth of the average number of uredosori per plant in a plot during the early stage of a epidemic of leek rust was assumed to follow an exponential model:

$$X(t)_i = X(0)_i \, e^{r_i t} \qquad (2)$$

in which $X(t)_i$ is the mean number of uredosori per plant in plot i at t days after the first observation, $X(0)_i$ is the mean number of uredosori per plant in plot i at the first observation and r_i is the relative growth rate in plot i. Different values of the relative growth rate r_i were assumed for every combination of cultivar and planting date.

In the 1992 experiment, exchange of uredospores between adjacent plots was likely to occur. A simple model was constructed to correct the results for exchange of inoculum between plots. In this model, the number of infections initiated by autoinfection during one day is assumed to be proportional to the number of uredosori present in the plot, thus assuming exponential growth in absence of alloinfection. Furthermore, it was assumed, that the number of infections initiated in a plot due to alloinfection from an adjacent plot is proportional to the number of infections initiated by autoinfection in that source plot during the same day.

For infections, it takes a period of latency to become uredosori. The latent period was assumed to be constant for all infections (p days). In other words, the number of uredosori occurring during one day in plot i due to autoinfection is proportional to the number of uredosori that was present in the plot p days before that day. The corresponding proportionality factor is the relative multiplication factor R_i and different values were assumed for each combination of cultivar and planting date. The number of uredosori occurring during one day in plot i due to alloinfection from an adjacent plot j is proportional with a factor *disp* to the product of R_j times the number of uredosori that was present in plot j p days before that day.

The mathematical model based on these assumptions is:

$$X(t+1)_i = X(t)_i + R_i * X(t-p)_i + \sum_{j=1}^{n} disp * R_j * X(t-p)_j \qquad (3)$$

in which $X(t)_i$ is the mean number of uredosori per plant in plot i at day t, R_i is the relative multiplication factor for plot autoinfection in plot i, *disp* is the proportionality factor for alloinfection from neighbour plot j and n is the number of plots contributing to alloinfection.

The proportionality factor for alloinfection *disp* is assumed to be equal for all adjacent plots, i.e. plots sharing a side or a corner. Furthermore, it is assumed, that between plots that are not adjacent, exchange of uredospores is negligible. Model (3)

only holds, when size and planting density are equal for all plots.

The following relation was used to calculate the relative growth rate r from the relative multiplication factor R:

$$R = (e^{r}-1)*e^{rp} \qquad (4)$$

Formula (4) follows from the requirement, that model (2) and (3) must be identical after omission of the term for alloinfection from model (3) (see Appendix 2).

3.2.3 Parameter estimation

A general procedure in non-linear curve fitting to obtain estimates of parameters (Θ) in the model, is minimizing the sum of squares of the residuals:

$$\sum_{j=1}^{m} (y_j - f(x_j,\theta))^2 \qquad (5)$$

in which y_j is an observation, $f(x_j,\Theta)$ the corresponding value of the model and m is the total number of observations, i.e. the product of the number of plots times the number of observation days (Richter and Söndgerath, 1990). In minimizing (5), all observations are weighted equally, which is a natural procedure, when the variance of the observations is the same for all observations (homogeneity of variance). However, with population growth, often an increase of the variance of the observations is found with time. A logarithmic transformation of the observations can sometimes be useful to remove the inhomogeneity of variances (Doucet and Sloep, 1992). The corresponding estimation of Θ is obtained by minimizing the sum of squares of the residuals of the logarithmic transformed values:

$$\sum_{j=1}^{m} (\ln(y_j) - \ln(f(x_j,\theta)))^2 \qquad (6)$$

A more general procedure to obtain these so called least square estimates of parameters (Θ) for observations with a complicated error structure, is minimizing the sum of squares of the residuals weighted for the variance in the individual observations:

$$\sum_{j=1}^{m} \frac{1}{\sigma_j^2} (y_j - f(x_j,\theta))^2 \qquad (7)$$

in which σ^2_j is the variance of an observation. Usually, σ^2_j will be unknown and replaced by estimated variances. In this study, variances are estimated by taking the measured variance in the observations. Without further assumptions, this method of estimation, known as weighted least squares, has only an intuitive appeal, but under certain distributional assumptions regarding y_j a widely accepted justification is found in likelihood theory (Richter and Söndgerath, 1990).

The approximate minimum of (5), (6) and (7) for models (2) and (3) was found numerically with the down hill simplex algorithm (Press et al, 1986). The initial value for the mean number of uredosori per plant in a plot $X(0)_i$ was estimated as a parameter with a positive value, that was restricted to be within the 95 % confidence limits of the observed initial mean number of uredosori per plant in that plot. For the 1992 experiment, (Θ) consisted of 24 initial values $X(0)_i$ and r values for each of the six combinations of cultivar and planting date. For model (3), the parameter *disp* was estimated in addition. For the 1993 experiment, (Θ) consisted of six initial values $X(0)_i$ and three cultivar dependent r values. For both experiments, an alternative (Θ) having only one pooled r value was estimated. The parameter estimation was performed by using the observations from all plots in an experiment simultaneously. The length of the latent period (p) in model (3) was not estimated as a parameter, but considered as a constant. The value of this constant p was taken as the shortest period between infection and occurrence of sporulating uredosori as observed in various experiments with artificial inoculation during 1991, 1992 and 1993. Model (3) was initialized for $-p \le t < 0$ by neglecting alloinfection over this period and backward calculation of $X(t)_i$ with model (2) starting from $X(0)_i$.

As a measure for goodness of fit, a non-linear coefficient of determination (R^2) is used, defined as:

$$R^2 = 1 - \frac{\sum_{j=1}^{m} (y_j - f(x_j,\hat{\theta}))^2}{\sum_{j=1}^{m} (y_j - \bar{y})^2} \qquad (8)$$

in which ($\hat{\Theta}$) is the set of estimated values for (Θ).

3.3 Results

In the 1992 experiment, the first planting was already infected when the second planting was planted. Therefore, the disease levels in the first planting were higher than in the second planting. In both plantings, highest disease levels were found on cultivar Albana, followed by Carina and Cortina. The same ranking was found in the 1993 experiment. In the 1993 experiment, the epidemic started later and consequently, the disease level during the observation period was lower than in the 1992 experiment. The mean pustule number per plant at the first and last date of observation are given in Table 7 for the 1992 experiment and in Table 8 for the 1993 experiment.

In both years, there was a strong dependence of the variance in the observations on the level of the observed mean number of uredosori per plant (Figure 1 and Figure 2), a commonly described phenomenon (Daamen, 1986a). This inhomogeneity of variance can be explained by several factors. First, some plants only have very low pustule numbers, even at high levels of disease infection, thus causing the variance in the observed mean pustule number to increase with increasing disease levels. Secondly, at increasing numbers of uredosori per plant, the estimation of the number of uredosori

Table 7. Mean uredosori number per plant in the 1992 experiment at the begin and the end of the observation period; values are the averages per combination of cultivar and planting date over the four blocks; values with the same letter are not significantly different (ANOVA on plot means per observation date, $\alpha = 0.05$)

Date	Planted on 21-5			Planted on 2-7		
	Albana	Cortina	Carina	Albana	Cortina	Carina
5-8	65.6a	4.2b	19.7c	0.7b	0.2b	0.8b
16-9	1637a	559b	837c	337bd	88d	165d

Table 8. Mean uredosori number per plant in the 1993 experiment at the begin and the end of the observation period; values are the averages per cultivar; values with the same letter are not significantly different (ANOVA on plot means per observation date, $\alpha = 0.05$)

Date	Albana	Cortina	Carina
28-7	0.34a	0.08a	0.18a
1-9	58.0a	21.2b	41.3ab

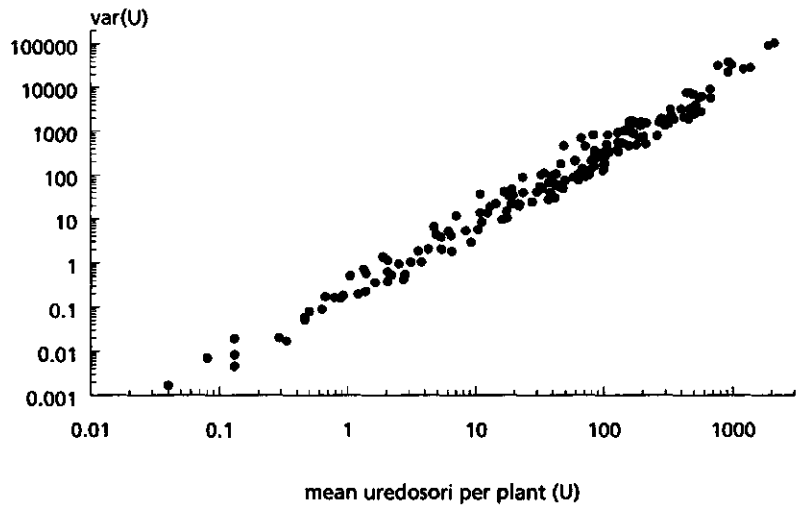

Figure 1. Relation between sample variance in the observations and the level of observed mean number of uredosori per plant for the 1992 experiment

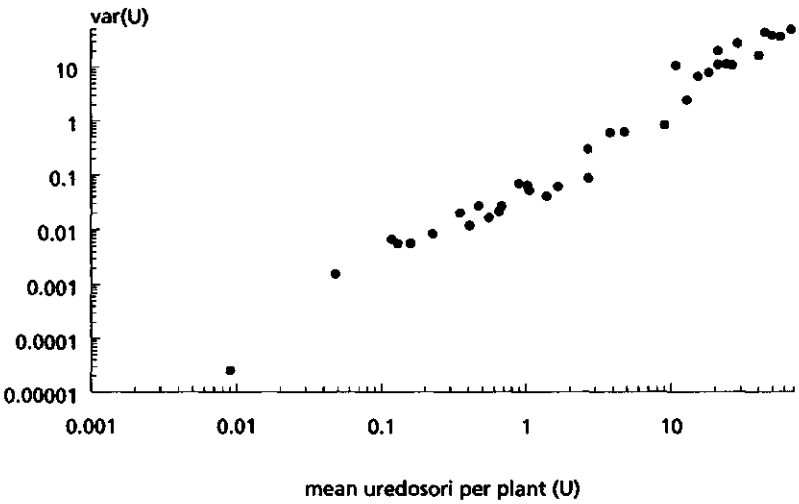

Figure 2. Relation between sample variance in the observations and the level of observed mean number of uredosori per plant for the 1993 experiment

per plant became more inaccurate. It was more easy to distinguish between 20 and 50 uredosori per leaf than between 200 and 500 uredosori per leaf. Furthermore, in the 1993 experiment, the number of plants observed per plot decreased with increasing disease level.

Given the strong dependence of the variance in the observations on the level of the observation, the weighted sum of squares (7) is more likely to produce reliable estimates for the model parameters from a theoretical point of view. However, since this method of parameter estimation is not always possible, e.g. due to lack of information on the variance in the observations, the results of the other two estimation methods are given as well for comparison.

The length of the latent period (p) in model (3) was taken as 12 days. This was the shortest period between inoculation and occurrence of sporulating uredosori as observed during August and September 1991 in inoculated leek plants (cv. Jolant), that were transferred to the experimental site. In greenhouse experiments, the latent period was equal for the cultivars Albana, Cortina and Carina, although somewhat longer than for cultivar Jolant under field conditions (Table 9).

Table 9. The latent period(p), i.e. the shortest period between artificial inoculation and the occurrence of sporulating uredosori as observed in various experiments and details on these experiments

Year	Date of inoculation	Location of inoculated plants (*)	Cultivar	Age of plants (months)	Number of plants	p (days)
1991	1-8	1	Jolant	2	40	12
	8-8	1	Jolant	2	40	10
	14-8	1	Jolant	2	40	11
	21-8	1	Jolant	2	40	11
	28-8	1	Jolant	2	40	12
	4-9	1	Jolant	2	40	12
	11-9	1	Jolant	2	40	12
	18-9	1	Jolant	2	40	12
1992	10-7	2	Jolant	5	144	12
1993	15-4	2	Albana	8	39	14
	15-4	2	Cortina	8	47	14
	15-4	2	Carina	8	47	14
	18-6	3	Albana	4	45	14
	18-6	3	Cortina	4	45	14
	18-6	3	Carina	4	45	14

(*) 1. experimental site (same as for 1992 and 1993 field experiments), 2. under plastic tunnel at institute, 3. greenhouse 20 °C

The values for the relative growth rates as estimated with the two models and the three methods for parameter estimation are given in Table 10 for the 1992 experiment and in Table 11 for the 1993 experiment. As plots were isolated in the 1993 experiment, only the simple exponential model (2) was applied.In general, good fit was obtained with both models, except when the logarithmic transformation (6) was used for parameter estimation.

Estimation of the relative growth rates with the simple exponential model (2), gave no clue on the differences in infection observed in the 1992 experiment. The estimated relative growth rate was lowest for Albana as compared to Cortina and Carina in both plantings (Table 10), whereas the observed disease levels were highest (Table 7). However, the results of the dispersion model (3) indicated, that the high values for the relative growth rate in the second planting as estimated with the simple exponential model (2) can be explained by influx of inoculum from the severely infected plots of the first planting (Figure 3). The dispersion model (3) produced estimates for the relative growth rate for the second planting of cultivars Cortina and Carina in absence of alloinfection, that were lower than the estimates obtained with the simple exponential model (2). These results suggest, that the difference in rust infection between the cultivars was due to a reduced growth of the disease in young plants of cultivars Cortina and Carina. In older plants, the ranking in susceptibility was reversed, causing the difference in infection between the cultivars to become less pronounced.

For the 1993 experiment, the simple exponential model (2) resulted in a good fit with the observed uredosori numbers (Figure 4). The same ranking in the estimates for the relative growth rate was found as for the first planting of the 1992 experiment, that had about the same age during observation (Tables 10, 11). In both cases, cultivar Cortina had the highest relative growth rate, followed by Carina and Albana. Only method (5) for parameter estimation resulted in a higher relative growth rate for Albana than the one for Carina in the 1993 experiment. The estimated values for the relative growth rate for the three varieties in the 1993 experiment were higher than those for the first planting of the 1992 experiment (Tables 10, 11).

Due to the late start of the epidemic in the 1993 experiment, no observations could be done on the epidemic growth in the young crop. However, the initially observed disease levels suggest, that the susceptibility of the three cultivars in young plants was in a reversed order compared to the susceptibility in older plants (Table 7).

The methods of parameter estimation (5) and (6) produced values for the relative growth rate comparable to those obtained with the theoretically more attractive method (7). However, in some cases the differences were considerable. One example is the zero value for the relative growth rate in the second planting of cultivar Cortina as estimated with model (3) and parameter estimation method (6), that falsely suggests

Table 10. Relative growth rates for the different combinations of cultivar and plant age as estimated from the 1992 experiment and the corresponding value for the non-linear coefficient of determination (R^2); for model (3) also the estimate for the proportionality factor for alloinfection (*disp*) is given

Model (*)	Parameter estimation method (*)	Relative growth rate (d^{-1})							*disp*	R^2
		Pooled	Planted on 21-5			Planted on 2-7				
			Albana	Cortina	Carina	Albana	Cortina	Carina		
2	5	0.09								0.72
2	6	0.10								0.04
2	7	0.09								0.69
2	5		0.08	0.12	0.10	0.14	0.15	0.17		0.94
2	6		0.08	0.12	0.10	0.15	0.17	0.19		0.84
2	7		0.07	0.12	0.09	0.14	0.15	0.18		0.84
3	5	0.08							0.04	0.91
3	6	0.09							0.04	0.86
3	7	0.08							0.04	0.87
3	5		0.08	0.13	0.11	0.13	0.08	0.12	0.02	0.95
3	6		0.08	0.10	0.09	0.11	0.	0.09	0.04	0.88
3	7		0.07	0.10	0.09	0.12	0.05	0.11	0.03	0.85

(*) numbers refer to the numbers of the formulae in the text

Table 11. Relative growth rates for the different cultivars as estimated from the 1993 experiment and the corresponding value for the non-linear coefficient of determination (R^2)

Model (*)	Parameter estimation method (*)	Relative growth rate (d^{-1})				R^2
		Pooled	Albana	Cortina	Carina	
2	5	0.16				0.85
2	6	0.18				0.22
2	7	0.16				0.83
2	5		0.15	0.20	0.14	0.89
2	6		0.16	0.20	0.17	0.39
2	7		0.15	0.20	0.16	0.85

(*) numbers refer to the numbers of the formulae in the text

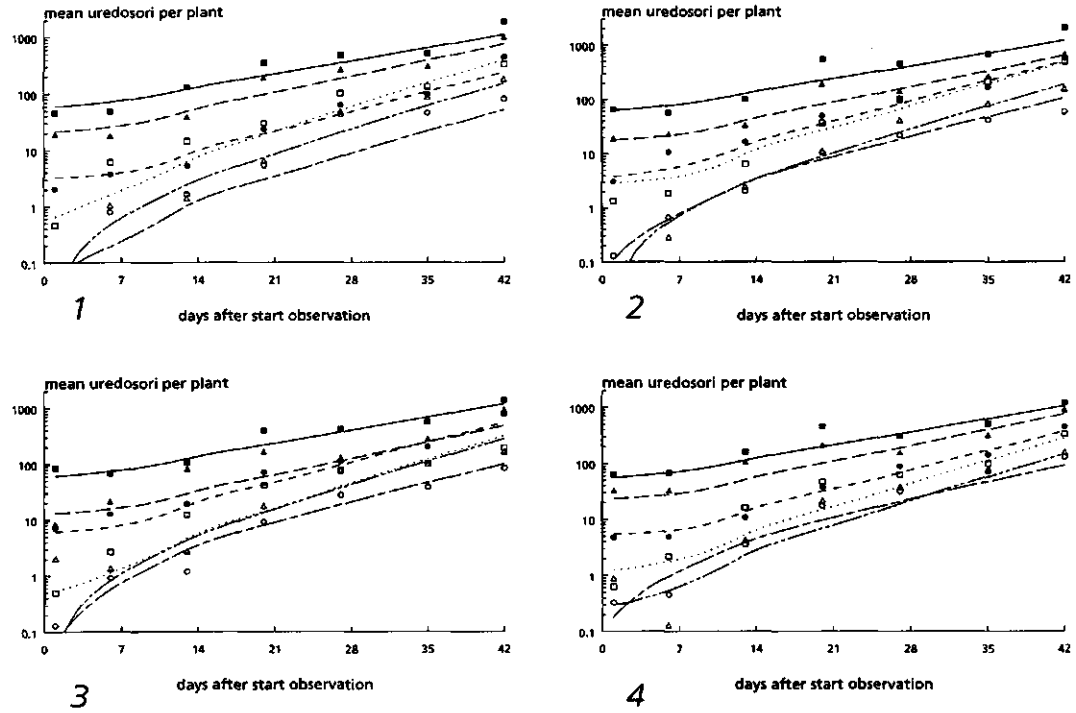

Figure 3. Observed (markers) and simulated (lines) leek rust epidemics in the individual plots for the 1992 experiment; the dispersion model (3) was used together with method (7) for parameter estimation; first planting: (■,——) Albana, (▲,— —) Carina, (●,- - -) Cortina; second planting: (□,· · · ·) Albana, (△,— - - —) Carina, (○,— · —) Cortina; the numbers under the graphs refer to the block number

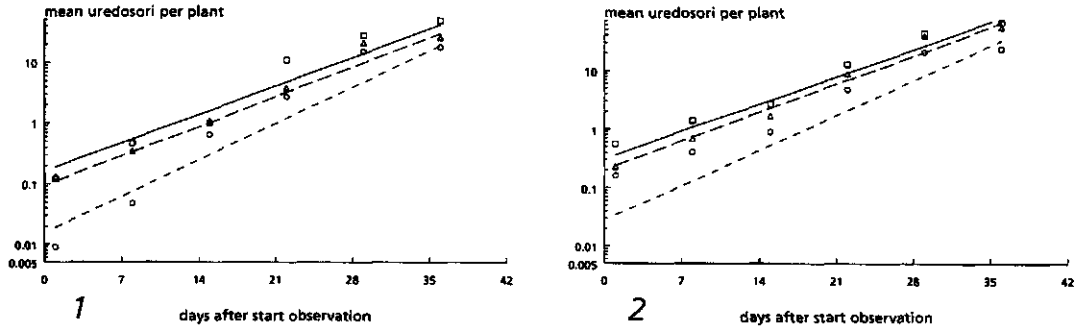

Figure 4. Observed (markers) and simulated (lines) leek rust epidemics in the individual plots for the 1993 experiment; the exponential model (2) was used together with method (7) for parameter estimation; (□,——) Albana, (△,– –) Carina, (○,- - -) Cortina; 1. the three western plots, 2. the three eastern plots

complete resistance in young plants of cultivar Cortina. The ranking in relative growth rates between the varieties and planting dates in the 1992 experiment did not depend on the method of estimation. In the 1993 experiment, method (5) produced a different order from the other two methods.

3.4 Discussion

The dispersion model (3) was kept as simple as possible to reduce the number of parameters as much as possible. In fact, only one parameter was used to characterize transport of spores between plots and this parameter was estimated from the disease observations simultaneously with the relative growth rates. The price paid for this simplification was that several assumptions had to be made. The transport of spores between plots was assumed to be symmetrical and constant over the observation period and to occur only between neighbouring plots. Another simplification was, that the length of the latent period was taken identical for all infections, irrespective of the combination of cultivar and planting date. This simplification is partly justified by the observations and the findings of Jennings et al. (1990b).

A more detailed model for exchange of inoculum between plots was given by Paysour and Fry (1983). The dispersion model (3) in this study is more suited to correct experimental results for exchange of inoculum between adjacent plots than the more detailed model of Paysour and Fry (1983), but it is less useful for theoretical studies on the effect of plot size and distance. In the dispersion model (3), the value for the parameter for alloinfection is specific for a particular experimental layout and dispersal conditions and this value can not be extrapolated to other experimental layouts or dispersal conditions.

The highest values for the non-linear coefficient of determination (8) are always obtained, when the simple method (5) for parameter estimation is used. This is not surprising, as method (5) is in fact maximization of the non-linear coefficient of determination (8). The sometimes poor fit obtained with the logarithmic transformation (6) is a general drawback of this method (Doucet and Sloep, 1992). In botanical epidemiology, the logarithmic transformation is often used to transform the exponential growth function in a linear function, in order to apply linear regression techniques. However, in applying the logarithmic transformation and subsequent linear regression, the thus estimated parameters not necessarily produce an optimal fit of the exponential model to the non-transformed data.

The results of both experiments show, that the growth of an epidemic of leek rust is dependent on the age of the crop. Jennings et al. (1990b) found a decrease in

susceptibility with increasing plant age in the leek cultivar Musselburgh. The results from the present study also indicate, that the effect of plant age is differing from cultivar to cultivar. In cultivar Albana, susceptibility was higher in younger plants than in older plants, whereas in cultivar Cortina it was the other way round. The inconsistency in the ranking of the susceptibility for leek rust among several leek cultivars at different observation dates as found by Smith and Crowther (1992), can be explained by assuming a similar interaction between cultivar and the effect of crop age on susceptibility.

Both experiments showed a consistent effect of cultivar and plant age on the growth of the leek rust epidemic. However, from the differences in the relative growth rates as estimated for the 1992 and the 1993 experiment, it is clear, that other factors than cultivar and plant age can have an effect on the growth of the leek rust epidemic. Although it is beyond the scope of this study to identify the exact causes of these differences, weather conditions and growing conditions of the leek crop can be mentioned as possible ones.

The growth of leek rust epidemics during the early stage of the epidemic in isolated plots was satisfactorily described by an exponential model. For predictive purposes, the value for the relative growth rate should be chosen with some care, as this parameter was found to be variable, depending among other factors on cultivar and plant age. The consequences of the variability of the relative growth rate for the determination of a safe sampling interval will be discussed in Chapter 4.

4 Sampling for detection, leek rust as an example

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Abstract

Computer simulation was used to compare four sampling patterns in their efficiency of detecting diseased plants under a variety of spatial distributions of infected plants. The sampling patterns compared were: diagonal sampling, double diagonal sampling, row sampling over two rows and row sampling over four rows. Row sampling over four rows was the most effective sampling pattern. The detection level, the lowest disease level at which the probability to detect the disease is sufficiently large, was studied in relation to temporal and spatial characteristics of the disease, as found in two epidemics in 1992 and 1993. For both epidemics, a weekly sample of 50 plants resulted in detection levels, that were low enough to ensure adequate chemical control, indicating the feasibility of sampling for detection of leek rust. Weekly sampling is more efficient than fortnightly sampling.

Keywords: sampling, leek, *Allium porrum*, leek rust, *Puccinia allii*

4.1 Introduction

For some diseases of agricultural crops, measures have to be taken as soon as the disease has entered the crop. This is the case for quarantine diseases and for diseases that cause cosmetic damage at very low disease levels to ornamental, vegetable or fruit crops. An example of the latter category is rust in leek (*Allium porrum* L.) caused by *Puccinia allii* Rudolphi. The presence of a low number of the orange-brown uredosori of this rust causes a decrease in leek quality, that may result in a severe economic loss to the grower.

Information on the presence of a disease in a crop can be based on regional forecasts, spore trapping and crop sampling. In this paper, a methodology for practical crop sampling for detection is presented. Leek rust is used to test the utility of the approach.

A sampling method has three important components: sampling pattern,

sampling frequency and sample size. A practical sampling method should meet the requirements of reliability, simplicity and speed. A sampling method for detection is reliable when the probability of detecting the disease is sufficiently large. In general, the probability of detection increases with increasing disease levels. Therefore, it is useful to introduce the concept of detection level. In this study, the detection level is defined as the lowest disease level, at which the probability of detecting the disease is at least 95 %. The detection level for leek rust can be expressed both in terms of the percentage of diseased plants in the field or the mean number of uredosori per plant in the field.

The detection level will be influenced by temporal and spatial characteristics of the disease, the identifiability and perceptibility of the disease, and the sampling method used. Temporal characteristics of the disease are important, because sampling for detection will always be done repeatedly, e.g. weekly, until the disease is found. The early stage of a leek rust epidemic can be described by an exponential growth function and estimates for the relative growth rates in different epidemics are available (Chapter 3).

Two spatial characteristics determine the detection level. First, the incidence-severity relationship gives information on what proportion of plants in a field is diseased given a certain level of mean number of rust pustules per plant. Incidence-severity relationships for leek rust are presented in this study. Secondly, it is important to know how the diseased plants are distributed over the field, randomly or clustered. The spatial distribution of rust-infected leek plants can be highly variable (Chapter 2)

Simulation studies on incidence sampling indicated that the precision of a sampling pattern can differ with different spatial distribution of diseased plants (Lin *et al.*, 1979; Delp *et al.*, 1986; Disthaporn *et al.*, 1993). Comparing sample patterns under a range of real field situations is experimentally laborious. In this study, Monte Carlo simulation was used to compare four simple sampling patterns under a variety of simulated spatial distributions of infected plants. Monte Carlo simulation is the technique of simulating complex stochastic processes by drawing random numbers from a relevant distribution.

The ability to identify and perceive the disease are other factors influencing the detection level. The orange-brown uredosori produced by leek rust on the leaves and sheath of leek plants are characteristic, and no confusion with other diseases is possible. However, when the number of uredosori per plant is low, these may be overlooked, depending on the accuracy of the observer.

In this study, the detection level of leek rust, as obtained with different sampling methods, is studied in relation to temporal and spatial characteristics of the disease. The results are used to compare practical sampling methods for detection of leek rust.

4.2 Material and methods

4.2.1 Sampling pattern

For random sampling, and assuming perfect observation of the disease in sampled plants, the probability of non-detection with one sample can be calculated to a good approximation as:

$$P_{nd}=(1-I)^S \quad (9)$$

in which P_{nd} is the probability of non-detection, I is the proportion of diseased plants and S the sample size (number of plants observed).

Monte Carlo simulation was used to estimate the probability of non-detection of four practical sampling patterns (see below) for a clustered distribution of the diseased plants. Simulated fields were represented by matrices, whose elements had the value zero to represent healthy plants or one to represent diseased plants. For every field, the centres of the clusters were randomly selected by drawing the X- and Y-co-ordinates from a uniform distribution. Then, rhomb shaped clusters of diseased plants were simulated around these centres. The clusters were equal in size and allowed to overlap.

The percentage of diseased plants used for the simulation were 0.1, 0.5, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12, 15 and 20 %. With each proportion, cluster size was varied from no clustering, i.e. random distribution, to maximum cluster size, i.e. diseased plants distributed over one or two clusters. Disease incidence in clusters was simulated by drawing a random number from a standard uniform distribution for every plant in a cluster and comparing this number with the specified probability. When the random number was smaller than or equal to the specified number, then the plant was considered to be diseased. Otherwise, the plant was considered to be healthy. All plants outside the clusters were considered to be healthy. The probability of plants in a cluster being infected was chosen as 0.25, 0.50, 0.75 or 1.

For every specified combination of proportion of diseased plants, cluster size and probability of plants in a cluster being infected, 100 fields with 200 rows and 600 plants per row were simulated.

From every of the 100 simulated fields, 10 samples were taken according to four sampling patterns: diagonal sampling, double diagonal sampling, row sampling over two rows and row sampling over four rows (Figure 5). The sample sizes used were 10, 20, 30, 40, 50, 60, 70, 80, 90 and 100 plants.

With diagonal and double diagonal sampling, plants were selected by increasing the X-co-ordinate (row number) of the previously sampled plant with a fixed number and increasing the Y-co-ordinate with a random number drawn from a uniform

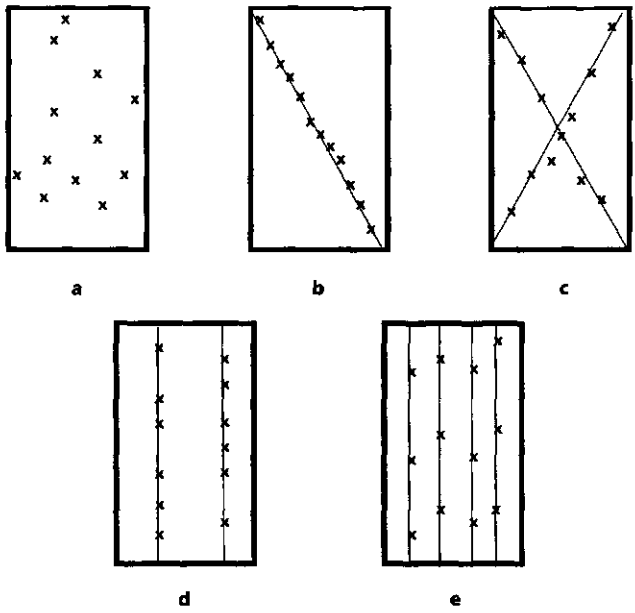

Figure 5. The sampling patterns compared: a. random sampling, b. diagonal sampling, c. double diagonal sampling, d.row sampling over two rows, e. row sampling over four rows.

distribution such that the sampled plants were lying around a diagonal through the field. With row sampling, the width of the field is divided in equal sections. In each section, a row is randomly selected. The selected rows are again divided in equal sections and one plant is selected randomly in each section.

For each combination of sampling pattern and sample size, the probability of non-detection was calculated on the basis of 1000 samples for each specified combination of proportion of diseased plants,cluster size and probability of plants in a cluster to be infected.

The pseudorandom numbers were generated using the algoritm UNIFL described in Bratley *et al.*. (1987). The computer program performing the calculations was written in FORTRAN (VAX FORTRAN version 5.0) and run on a VAX 3600 computersystem.

4.2.2 Sampling frequency and sample size

The following function was chosen to describe the incidence-severity relationship (Nachman, 1981; Daamen, 1986b):

$$I=1-e^{-aM^b} \quad (10)$$

Equation (10) may be linearized by the complementary log log transformation to:

$$\ln(-\ln(1-I)) = \ln a + b*\ln M \quad (11)$$

in which I is the incidence (proportion of diseased plants) and M is the severity (mean number of uredosori per plant). The parameters a and b were estimated using observations of plants in a grid in field experiments in 1992 and 1993 with cultivars Albana, Cortina and Carina. The linearized function (11) was fitted to the transformed field data using least squares regression. Standard F-tests were used to analyse whether a combination of separate regression lines for each year, or even separate regressions for year-cultivar combination, resulted in a significantly better fit.

The growth of the number of uredosori in the early stage of a leek rust epidemic can be described by an exponential function (Chapter 3):

$$M_t=M_0e^{rt} \quad (12)$$

in which M_t is the mean number of uredosori per leek plant on day t, M_0 is the initial number of uredosori per leek plant and r is the relative growth rate. Estimates for the relative growth rate for epidemics in 1992 and 1993 derived from a previous study, were 0.1 and 0.2 respectively (Chapter 3). The initial number of uredosori per plant (M_0) was chosen arbitrarily at different low levels between one uredosorus per 10,000 plants and one uredosorus per 100 plants.

With consecutive sampling, the probability that the disease has not been detected in any sample equals:

$$Ptotal_{nd}=P_{nd_1}*P_{nd_2}*\ldots*P_{nd_{n-1}}*P_{nd_n} \quad (13)$$

in which $Ptotal_{nd}$ is the total probability of non-detection with n samples and P_{ndn} is the probability of non-detection for the n^{th} sampling.

By using equations (9), (10), (12) and (13), the detection level can be calculated for every combination of sampling frequency and sample size, assuming random sampling or an equivalent sampling pattern. As the onset of the disease can be on any of the days in the interval between two samplings, the probability of detecting the

disease must be calculated as the average of the probabilities of detection over all possible starting days.

4.3 Results

4.3.1 Sampling pattern

The results of the Monte Carlo simulation showed, that with a clustered distribution of diseased plants, there was an effect of sampling pattern on the probability of non-detection. For all percentages of diseased plants and degrees of clustering simulated, diagonal sampling yielded the highest probability of non-detection, followed by double diagonal sampling and row sampling over two rows, which were about equally

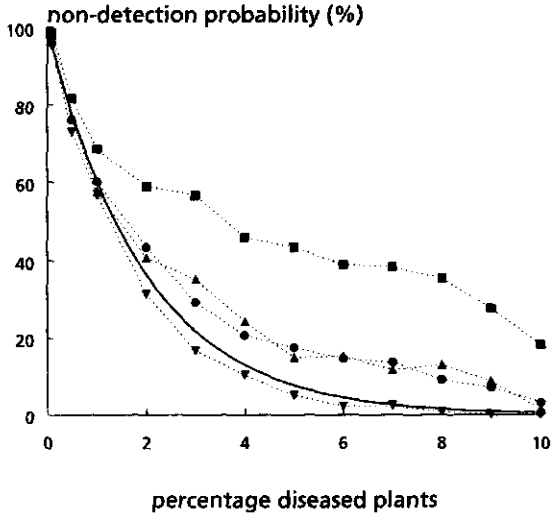

Figure 6. The non-detection probability of diseased plants distributed over two clusters with a sample size of 50 plants as function of sampling pattern and percentage infected plants (the probability of plants in a cluster to be infected equals one); ■ diagonal sampling, ▲ double diagonal sampling, ● row sampling over two rows, ▼ row sampling over four rows; the solid line represents the theoretical relationship for random sampling given in equation (9)

efficient.With row sampling over four rows, the probability of non-detection was lowest and always less or equal to that expected on the basis of the theoretical relationship for random sampling given in equation (9). The effect of sampling pattern on the probability of non-detection was most pronounced at a percentage of diseased plants between 2 and 8 % distributed over one or two clusters and the probability of plants in a cluster to be infected equal to one. These results are illustrated by Figure 6, in which the probability of non-detection is given as function of the percentage of infected plants with a fixed sample size of 50 plants, and Figure 7, in which the probability of non-detection is given as function of sample size at 4 % diseased plants.

The time required for walking through a 100 m x 100 m field is measured as 2 minutes for the diagonal pattern and 5 minutes for row sampling over four rows, not including the time needed for observing the sampled plants.

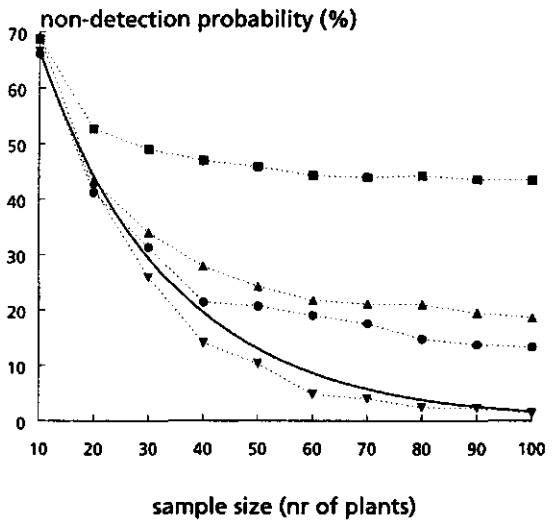

Figure 7. The non-detection probability at 4 % diseased plants distributed over two clusters as function of sampling pattern and sample size (the probability of plants in a cluster to be infected equals one); ■ diagonal sampling, ▲ double diagonal sampling, ● row sampling over two rows, ▼ row sampling over four rows; the solid line represents the theoretical relationship for random sampling given in equation (9) diseased plants.

4.3.2 Sampling frequency and sample size

Linear regression of the transformed incidence and severity data showed a significantly better fit for a model consisting of separate regression lines for 1992 and 1993, than for a single regression line (Table 12, Figure 8). The use of separate regression lines for each year-cultivar combination did not significantly improve the fit of the former model (Table 12).

The detection level, i.e. the lowest disease level at which the probability of detecting the disease is at least 95 %, is given in Table 13. It was calculated for a sampling interval of 7 days, a sample size of 50 plants and different values for M_0, and r and the incidence-severity relationships as found in 1992 and 1993. With the 1992 incidence-severity relationship, the detection level increased with increasing values for M_0, whereas with the 1993 incidence-severity relationship, this effect was absent. There was a strong effect of both the value for r and the incidence-severity relationship on the detection level. The effect of the incidence-severity relation was especially strong, when the detection level was expressed in number of uredosori per plant.

Table 12. Regression analysis for severity and incidence data using equation (10) to describe the relation between incidence and severity and equation (11) to calculate F values (n=73).

	ln a	b	R^2	d.f.	F	p
pooled	-1.772	0.781	0.85	1		
years			0.95	3	59.99	<0.001
1992	-1.305	0.657				
1993	-2.390	0.944				
years+ cultivars			0.96	11	1.77	>0.1
92,Albana	-1.175	0.604				
92,Cortina	-1.335	0.681				
92,Carina	-1.336	0.661				
93,Albana	-2.599	0.988				
93,Cortina	-2.172	0.871				
93,Carina	-2.564	1.081				

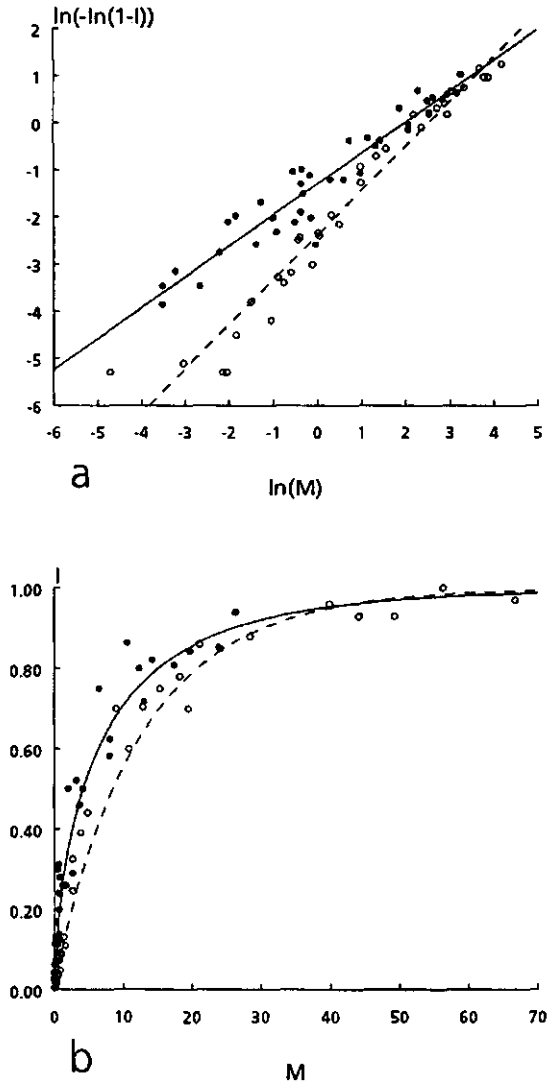

Figure 8. Incidence-severity relationship for leek rust, ● 1992 observations, O 1993 observations, the solid line gives the fit obtained by linear regression for the 1992 data and the dashed line for the 1993 data; a. complementary log log transformed data, b. original data

Table 13. Detection level as calculated with equations (9), (10), (12) and (13) for different values of M_0, r and the incidence-severity relationships (IS) as found in field experiments in 1992 and 1993; the sample size was 50 plants and the sampling interval 7 days

detection level in % infected plants in the field

	IS-1992		IS-1993	
M_0	r=0.1	r=0.2	r=0.1	r=0.2
1.10^{-4}	2.8	6.1	4.2	8.7
1.10^{-3}	3.0	6.6	4.2	9.5
1.10^{-2}	4.2	7.0	4.2	8.7

detection level in mean number of uredosori/plant in the field

	IS-1992		IS-1993	
M_0	r=0.1	r=0.2	r=0.1	r=0.2
1.10^{-4}	0.03	0.11	0.44	1.00
1.10^{-3}	0.04	0.12	0.45	1.10
1.10^{-2}	0.06	0.13	0.45	1.00

In figure 9, the detection level is given as function of the sample size for a sampling interval of 7 days and 14 days as calculated with the relative growth rate and the incidence-severity relation as found in 1992 and 1993, respectively. The stepwise pattern in the figure is caused by the discrete steps in the calculations for time and sample size. The detection levels as calculated with the 1993 parameters were much higher than those calculated with the 1992 parameters. With the 1993 parameters, increasing the sampling interval from seven days to fourteen days had a stronger effect on the detection level than with the 1992 parameters (Figure 9).

With the 1993 parameters, weekly sampling was clearly more efficient than fortnightly sampling. With weekly sampling and a sample size of 50 plants, the detection level was 10 % diseased plants or one uredosorus per plant, whereas with fortnightly sampling and a sample size of 100 plants, the detection level was 15 % diseased plants or two uredosori per plant.

The time needed for observation of one plant is about 30 seconds. Sampling 50 plants along 4 rows in a 100 m x 100 m field will take about 30 minutes.

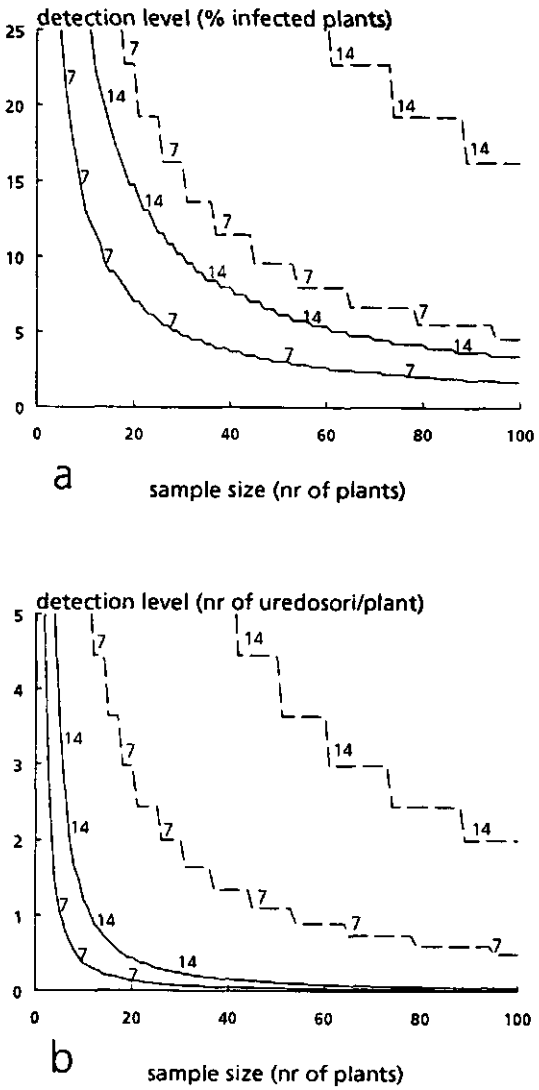

Figure 9. Detection level for consecutive sampling as function of sample size and sample interval (entries, days), solid lines give the calculations for the 1992 parameters, the dashed lines for the 1993 parameters.

4.4 Discussion

The probability of overlooking the disease under clustered distribution is much higher for diagonal sampling than for the other sampling patterns. With diagonal sampling, the field area covered is smallest, causing the very small decrease in the non-detection probability with increasing sample size. Lin *et al.* (1979), Delp *et al.* (1986) and Disthaporn *et al.* (1993) found in similar simulation studies, that those sampling patterns covering the largest field area were most efficient. In the present study it was shown, that row sampling over four rows has a non-detection probability less or equal to that of random sampling, independent of the spatial distribution of diseased plants. Delp *et al.* (1986) found the same result for stratified random sampling. However, stratified random sampling is cumbersome, whereas row sampling is a convenient sampling pattern for practical application.

The distance to be walked with row sampling over four rows is longer than with other sampling patterns, but the extra time needed for walking through the field will only be about 3 minutes longer in a 100 m x 100 m field as compared with diagonal sampling, and is largely compensated for by the much lower sample size needed. An additional advantage of sampling along rows is that less damage is done to the crop than with diagonal sampling.

Sampling 50 plants along 4 rows in a 100 m x 100 m field will take about 30 minutes. The problem in this is not so much the time needed as the dullness of sampling for detection. Sampling has to be continued as long as no disease is observed and it requires discipline of the farmer to complete 50 plants, while all plants observed so far are healthy.

It was assumed that the field is uniform in its susceptibility for the disease. When there are areas known in the field where disease is more likely to occur than elsewhere, these areas should be sampled preferentially as suggested by Seem *et al.* (1985).

Given the low average uredosori number per diseased plant at relevant disease incidence levels, the sampled plants must be thoroughly examined. Theunissen and Legutowska (1992) compared the accuracy of detailed observation of pest and disease symptoms in leek with a superficial observation,i.e. scanning the plants from above, and found a very low accuracy of the superficial observation for leek rust. But even with a detailed observation, perfect observation does not seem realistic, and it is likely, that the accuracy of observation is related to the average number of uredosori per infected plant. Information on this relation can be collected in a study like that of Seem *et al.* (1985) on the detection of grapevine downy mildew.

The detection level, required for adequate control of leek rust, depends on the efficacy of chemical control in relation to the damage threshold. At present,

information on such variables and their relationships with plant growth and environmental conditions is scarce. A mean number of one pustule per plant is a safe detection level for adequate chemical control of leek rust, although this level may in fact be too strict.

The detection level resulting from a given sampling frequency and sample size was found to be highly dependent on the growth rate and the incidence-severity relationship of the disease. However, these parameters are unknown for an epidemic that still has to be detected. The calculations based on the 1992 and 1993 epidemics indicate, that a sampling interval of 7 days is more efficient than a sampling interval of 14 days. For both epidemics, a weekly sample of 50 plants resulted in detection levels, that were low enough to ensure adequate chemical control, indicating the feasibility of sampling for detection of leek rust. As a previous study showed that rust-infected leek plants at low incidence are often clustered (Chapter 2), it is essential that row sampling over four rows be used as this simple sampling pattern has the lowest probability of overlooking the presence of the disease.

5 The reduction of chemical control of leek rust, a simulation study

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Abstract

A model is presented which simulates chemical control against leek rust under a system of supervised control. The spread of disease between fields in one farm and the influx of spores from outside is part of the model. The possible reduction in fungicide sprays against leek rust following various preventive measures is estimated with this model. Results from this preliminary model suggest that supervised control of leek rust will result only in a considerable reduction in the number of fungicide sprays if the leek fields are sufficiently isolated and the planting material is initially disease free. Where isolation between the fields is not feasible, overlap between successive crops should be avoided. Without the use of these preventive measures, there is little prospect for supervised control and the grower will have no option but the heavy use of fungicides.

Keywords: epidemiology, supervised control, leek rust, prevention, simulation

5.1 Introduction

The pathogen causing leek rust, *Puccinia allii* Rudolphi, needs to infect new crops of leek (*Allium porrum* L.) before the harvest of infected crops to ensure its survival. Resting spores and alternative hosts are of minor significance in the pathogen's epidemiology. Only the uredosori are commonly present in the Netherlands, teleutosori are rarely observed. Rust of leek can infect *A. vineale* L. (Jennings et al., 1990a) which is a common wild plant in The Netherlands but isolates are not infective on leek (R. Niks, pers. comm.) and are described as a different rust species, *P. mixta* Fuckel (Niks and Butler, 1993). Leek cultivation is continuous over the year in the Netherlands, with ample overlap between the crops. The early summer crops are planted in April and harvested in July. The late winter crops are planted in July and harvested during winter and spring until May the following year. The disease is able to survive low temperatures during winter in infected plants, permitting regrowth once temperatures rise. At present, there are no completely resistant leek cultivars, but differences in susceptibility

have been found among cultivars in the United Kingdom (Uma and Taylor, 1991; Smith and Crowther, 1992), Germany (Albert and Smolka, 1994) and the Netherlands (Chapter 3).

Research on the control of leek rust has concentrated on breeding for resistance (Jennings et al., 1990a; Uma and Taylor, 1991; Smith and Crowther, 1992) and the optimal use of fungicides (von Meyer and Kessler, 1990; Chapter 4), but little attention has been given to preventive measures. This study focuses on the opportunities for and benefits of preventive measures for individual growers, to reduce the need for chemical control against leek rust; these can be divided into strategic measures and tactical measures (Conway, 1984). Strategic measures are: 1.reduction of influx of spores from outside the farm by moving to a less dense production region, 2. increase of the isolation between fields within the farm to reduce spread of spores between fields, 3. change to a cropping pattern with less overlap between successive crops. Tactical measures are: 1. use of less susceptible cultivars (hypothetical) or cropping practices that reduce the disease increase, 2. inspection of planting material and refusal of infected planting material. To obtain information on the effect of the three strategic measures, research at the cropping system level is necessary, but is expensive due to the large experiments required. A survey of leek farmers is a less costly alternative for experimental research, but observations must cover several years. Before starting expensive experimental research or a lengthy survey, the possible effect of preventive measures can be explored with simulation modelling. In this paper, a simulation model is used to estimate the possible reduction in fungicide sprays against leek rust due to preventive measures.

5.2 Materials and methods

5.2.1 Model

Consider a leek grower having four leek fields, one is planted with a winter crop, to be harvested in April or May, another planted with an early summer crop in April and harvested in July or August, a third field planted with an autumn crop in June and harvested in November and the last field planted in July or August with next year's late winter crop, and harvested in April or May next year. This cycle is repeated every year. The grower has adopted the following system of supervised control of leek rust, and monitors crops weekly and applies fungicide to a crop every fortnight, as soon as disease is detected in the crop, i.e. when the mean number of uredosori per plant is one. With an appropriate sampling method, this is practically feasible (Chapter 4). The fungicide does not affect the uredosori already present, but it protects the crop against

new infections. No fungicide is applied in a period of 14 days before harvest.

The mean number of uredosori per plant in a field increases from autoinfection, due to alloinfection from other infected fields on the same farm and due to influx of spores from outside the farm. The number of infections initiated by autoinfection during one day is assumed to be proportional to the number of uredosori present in the field, thus assuming exponential growth in the absence of alloinfection (Chapter 3). The number of infections initiated in a field due to alloinfection from another field on the same farm is assumed to be proportional to the number of infections initiated by autoinfection in the source field during the same day. It is assumed that there is a constant influx of spores from outside the farm.

The latent period (from infection to uredosorus) was assumed to be constant for all infections (p days). The number of uredosori occurring during day t in field i due to autoinfection is assumed to be proportional to the number of uredosori present in that field p days before. The corresponding proportionality factor is the relative multiplication factor R. The number of uredosori occurring during day t in field i due to alloinfection from field j is proportional with a factor $c_{ij}R$ to the number of uredosori present in field j p days before. The mean number of uredosori per plant in a field, $X(t)_i$, is initialized by equating to X_0, i.e. the disease level in the planting material, for the period starting from planting till p days after planting. As a result of the above assumptions, the increase in mean number of uredosori per plant in a field, p or more days after planting, is described by the following relation:

$$X(t+1)_i = X(t)_i + \left(R*X(t-p)_i + \sum_{j=1, j\neq i}^{n} c_{ij}*R*X(t-p)_j + f\right) * s(t-p)_i \qquad (14)$$

with the following variable (dimensions are given in []):

$X(t)_i$, mean number of uredosori per plant in field i at day t [pustule.plant^{-1}],

and the following parameters:

R, daily relative multiplication factor for autoinfection [day^{-1}],

p, duration of the latent period [day],

n, the number of fields contributing to alloinfection [-],

c_{ij}, proportionality factor for alloinfection of field i from a neighbour field j [-],

f, constant daily number of uredosori due to influx of spores from outside the farm [pustules.plant^{-1}.day^{-1}],

$s(t)_i$, the factor accounting for the effect of a protecting fungicide in field i on day t [-].

5.2.2 Parameters

The period of the year favourable for increase of leek rust is assumed to be between 1 April and 1 November when the latent period is assumed to be constant and 14 days long (Jennings et al., 1990a, b; Uma and Taylor, 1991, Chapter 3). The relative growth rate of the disease r was found to be variable with the growth stage of the crop (Chapter 3). As a simplification, r is assumed to be constant at a value of 0.12 day^{-1} for a highly susceptible cultivar (Chapter 3). To simulate partial resistance or cropping practices that restrain the disease increase, a relative growth rate of 0.06 day^{-1} was assumed, without changing the length of the latent period. The daily relative multiplication factor R used in (14) is calculated from the relative growth rate r in the absence of alloinfection, and the latent period p with the following equation:

$$R = (e^{r}-1)*e^{rp} \qquad (4)$$

(Chapter 3).

From 1 November to 1 April, weather conditions were assumed to be unfavourable for new infections and r = 0. During this period, the mean number of uredosori per plant is assumed to be halved due to weathering of the leek plants. This is a conservative estimate based on observed reductions (Table 14). The parameters c_{ij} are measures for spore transport between fields and their values will be lower with better isolation between fields. It is convenient to restrict the analysis where c_{ij} is independent of the two particular fields, i.e. assume $c_{ij}=c$ for all i,j. For an estimate of how c may be related to the distance between the fields and the size of the fields, all individual plants in the source field can be considered as point sources. We use an exponential model (Kiyosawa and Shiyomi, 1972) to describe the infection gradient

Table 14. Observed reduction in mean number of uredosori per plant in fields planted with hardy cultivar Carina (planted begin of July) during two winters (sample size was 90 plants, values with the same letter within one season are not significantly different at α=0.05 according to LSD-test)

date	mean number of uredosori per plant	reduction (%)
17 Dec 1991	58.1a	41.5
26 Feb 1992	34.0a	
30 Nov 1992	122.0a	97.5
11 Mar 1993	3.0b	

from these point sources and assume a homogeneous spatial distribution of the disease. The average number of infections c received per plant in the recipient field as a result of spores originating from all plants in the source field can be calculated as:

$$c = \frac{1}{N}\sum_{k=1}^{N}\sum_{l=1}^{N} a e^{-bd_{kl}} \qquad (15)$$

in which d_{kl} is the distance in meters between plant k in the recipient field and plant l in the source field, N is the number of plants in each equal sized field, a is a scaling parameter and b is a parameter determining the steepness of the infection gradient. Estimates for b for uredospores of leek rust are not available. For uredospores of *P. graminis* Pers. and *P. recondita* Rob.&Desm. estimates for b range from 0.02 to 1.4 m^{-1} (Aylor, 1987; Fitt et al., 1987), the first value representing the shallowest gradient and dispersal over the longest distance. By fitting a model very similar to model (14) to the results of a previous experiment with leek rust, c was estimated to be between 0.02 and 0.04 for adjacent plots measuring 12.5mx8m and with a planting distance of 0.5mx0.2m between and in the row (Chapter 3). With these geometrical characteristics and c = 0.04 and b = 0.02 (representing maximal spore dispersal), a value of $4.9*10^{-5}$ was obtained for a by heuristically applying (15). With (15), the decline of c with increasing distance between borders of fields was calculated. As c is inversely proportional to the isolation between fields, -log(c) can be considered as a measure of isolation between fields and is plotted in Figure 10 as a function of distance. The values for c used in the model were 10^{-2} to simulate poor isolation between fields and 10^{-7} for good isolation. Intermediate values were also used. According to (15) and the estimated parameters, assuming the most favourable conditions for spore dispersal, the two extreme values for c correspond with distances of 200 m and 800 m between two 100mx100m fields, respectively (Figure 10).

The value for f is a measure for the level of background infection as determined by the intensity of leek growing and presence of leek rust in the region. These factors cannot be manipulated by individual farmers. The values for f used in the simulations are 10^{-7} for low and 10^{-5} for a high background infection levels, representing 0.01 and 1 infection per day, respectively, for a field with 100.000 plants (approx. one hectare). The factor $s(t)_i$ equals 0.05 when field i is protected and 1 otherwise, thus simulating an efficacy of the fungicide of 95 %. X_0 represents the disease level in the planting material and was assumed to be zero or 0.1 pustule per plant, to simulate healthy and infected planting material, respectively.

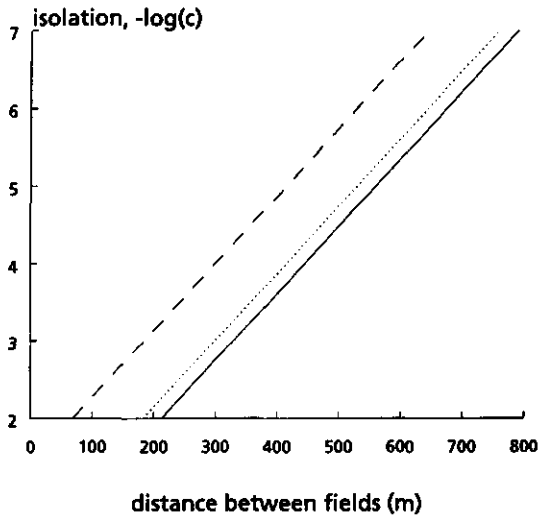

Figure 10. The relation between isolation (-log(c)) and the distance between the border of equally sized fields as calculated with (15), $a = 4.9*10^{-5}$, $b = 0.02$ and planting distance 0.5mx0.2m for fields measuring 10mx10m (dashed line), 50mx50m (dotted line) and fields measuring 100mx100m (solid line)

5.2.3 Model scenarios

The model (14) is used to calculate the yearly total number of sprays during ten years for ten scenarios. Every scenario is defined by a combination of the three cropping conditions that are influenced by strategic preventive measures: level of background infection (f), isolation between fields (c) and cropping pattern. The three cropping patterns used are given in Figure 11. Pattern A has the maximum possible overlap between successive crops. Pattern B has overlap only between the autumn and winter crops. Pattern C differs from pattern B in that the harvests of the winter and summer crops are delayed by one week. Thus two crops overlap one week with subsequent crops. Cropping patterns A and C are combined with two levels of background infection level (f) and isolation between fields (c). Cropping pattern B is combined with two levels of f and only one value of c, because the lack of overlap between crops makes the model results insensitive to the latter parameter. The resulting ten scenarios are given in Table 15. For every scenario, calculations are done for healthy and diseased

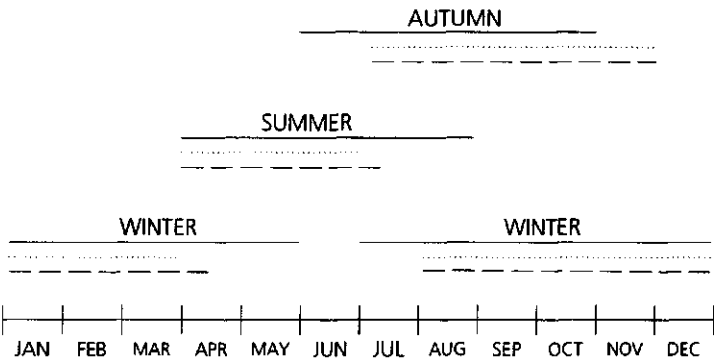

Figure 11. The cropping patterns used for the simulations; solid line: pattern A, the maximal possible overlap between the successive crops; dotted line: pattern B, only overlap between the autumn crop and the winter crop; dashed line: pattern C, differs with pattern B, in that the harvests of the winter crop and the summer crop are delayed by one week

planting material (X_0) and normal and slow rust increase (r), factors that are influenced by the tactical preventive measures. The calculations were started on 1 April in the first year. The number of pustules per plant in the standing winter crop is given a range of arbitrary values to test the sensitivity of the model to this initial disease level.

5.3 Results

For all scenarios, a stable equilibrium for the annual number of sprays occurs before the fifth year and was independent of the number of pustules per plant in the standing winter crop at the beginning of the simulations. The simulated total number of sprays in the fifth year is given in Table 15 for the ten scenarios. With scenarios 1 and 2, i.e. when the cropping pattern had ample overlap between successive crops (cropping pattern A) and poor isolation between the fields in the farm ($c = 10^{-2}$), a large number of sprays was simulated independently of the influx of spores from outside the farm (f).

5.4 Discussion

The loss of quality associated with a light infestation of leek rust may cause severe economic loss to the grower, due to the high value of the crop and the strict grading system. Therefore, regular chemical control of the disease has to start as soon as the disease has entered the crop and there is therefore little practical use for a quantitative control threshold, as was also found by Nyrop et al. (1989) for chemical control against leek moth (*Acrolepiopsis assectella* Z.). Applying fungicides to a leek crop in the absence of the disease is undesirable, both from environmental and economic points of view. A sampling procedure for detection of leek rust has already been developed (Chapter 4). Although the procedure of delaying chemical control until detection of the disease does not use a quantitative control threshold, it can still be regarded as a form of supervised control (sensu Theunissen (1984)). In the intensive leek growing areas in the south of the Netherlands, it is common practice to grow poorly isolated and overlapping crops on the same farm. The simulation results obtained suggest that under these cropping conditions, supervised control is not effective in reducing the number of sprays against leek rust. An alternative strategy of supervised control is to protect all crops during the growth of one or more visibly infected crops, instead of spraying the visibly infected crops only; this was evaluated in preliminary calculations but did not reduce the simulated number of sprayings under these conditions, because the chemical protection of visibly uninfected crops itself required extra fungicide applications.

The use of healthy planting material was a prerequisite for reduction in fungicide sprays by any other preventive measure. Most leek growers in the Netherlands order their planting material from specialized nurseries about four months before planting but this may be infected in the nursery and should be inspected before acceptance. Refusal of infected planting material will lead to the additional costs of obtaining alternative planting material without a timely order, and this may compel the farmer to accept inferior material with latent infections. The simulation results point to the importance of good nursery hygiene. With healthy planting material, the simulated number of sprays was greatly reduced by increased isolation between the fields in one farm. The simulation results are supported by the findings of Weisz et al.(1994), who found a reduction in infestation by colorado potato beetle (*Leptinotarsa decemlineata* Say) and early blight (*Alternaria solani* Sor.) and a subsequent decrease in the need for chemical control due to increased isolation of potato fields. Isolation between fields can be obtained by increasing the distance between fields and by growing border rows with a buffer crop, e.g. maize. For small farms, increasing the distance between fields will require purchase of land or exchange of land with a non-leek farmer. Little information is available in the literature on the quantitative effect of a buffer crop on spore

dispersal. Boudreau and Mundt (1992) found that the addition of a single maize row between source plants and trap plants did not alter the dispersal gradient of bean rust (*Uromyces appendiculatus* (Pers.) Unger). Change to a cropping pattern with no overlap in time between successive crops also reduced the simulated number of sprays, provided that healthy planting material was used. A serious drawback, however, is the shorter growing period that will inevitably result in a lower yield.

The results from this study suggest further research along two lines. Firstly, a more comprehensive model needs to be developed, containing variable growth rates and latent periods depending on weather conditions and growth stages of the crop, and a more realistic description of the dispersal of spores between fields and the influx of spores from outside the farm. Substantial experimental work will be needed to quantify these relations. Secondly, the results of the model should be tested against field observations. The scale of the problem requires large experiments, as performed by Weisz et al.(1994). To avoid such large experiments, the field observations can be collected in a survey of leek growers applying supervised control which should cover a sufficient number of growers representing a range in cropping patterns, farm size, etc. over several years to rule out exceptional weather conditions. The efforts in further research will be rewarded by an increased insight into the possibilities for reducing fungicide use by means of preventive measures; a similar study may be useful for other pathosystems in which supervised control has not resulted in the expected reduction in pesticide use.

6. A model to study the effect of certification of planting material on the occurrence of leek rust

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Abstract

Based on a simple model, the possible effects of certification of planting material on the occurrence of leek rust in a region can be derived from information on the proportion of infected fields (v) and the proportion of newly planted fields with infected planting material (i) in that region. If $v \leq \sqrt{i}$, certification of planting material will be highly effective.

Keywords: epidemiology, disease eradication

The biology of leek rust, caused by *Puccinia allii* Rud., and its management at the farm level in the Netherlands is discussed in Chapter 5. Also on a regional level, preventive measures can be taken which reduce the amount of inoculum and increase the distance between sources of inoculum and endangered fields. In this context, a region is defined as an area where the cultivation of a crop is concentrated. Examples of preventive measures on a regional level in other pathosystems in the Netherlands are the provincial regulations concerning the timely clearing of silage of fodder beets preventing spread of beet yellow viruses, clearing or covering of heaps with harvest debris of potatoes that may house *Phytophthora infestans*, and the designation of protected regions preventing the spread of fire blight into pear orchards (Anonymous, 1993; Schouten, 1992). Reviews on the effectiveness of sanitation measures can be found in Zadoks and Schein (1979) and Lester (1986).

Possible preventive measures against the spread of leek rust on a regional level include a strict certification of planting material ensuring that only healthy planting material is used; a minimal distance between leek crops and a crop free period in a whole (sub-)region. The last two measures have a strong impact on farm management and are unlikely to be accepted as regulations by leek growers.

To explore the effects of certification of planting material on disease occurrence on a regional level, a simple extension of Levins' (1969) metapopulation model was constructed. As a simplification it is assumed, that every harvested leek field is replaced

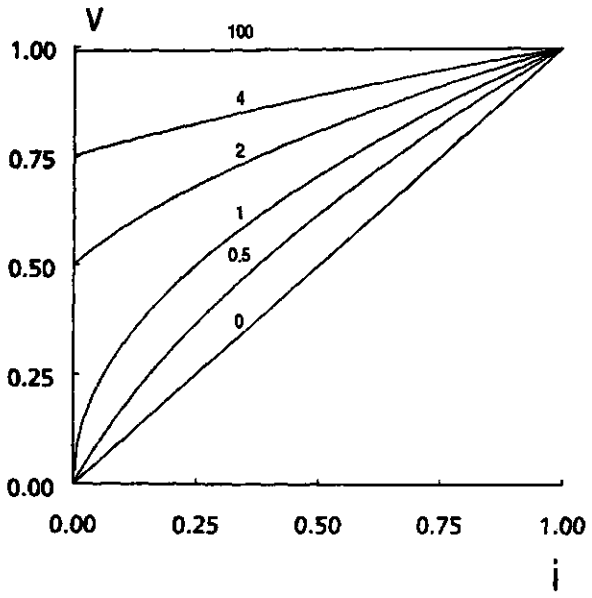

Figure 13. The fraction of fields infected (v) at equilibrium as function of the fraction newly planted fields with infected planting material (i) according to the model; entries: c/h, i.e. the ratio between a parameter reflecting colonization ability (c) and the instantaneous harvest and planting rate (h);note that for $c/h \leq 1$, $v = 0$ if $i = 0$, and that for $c/h > 4$, v is relatively independent of i

immediately by a newly planted field. Thus, the number of leek fields is assumed constant. The temporal change in the fraction of infected fields (v) can then be described as a function of a parameter reflecting the colonization ability of leek rust (c), the instantaneous harvest and planting rate (h) and the fraction of newly planted fields with infected planting material (i):

$$dv/dt = cv\,(1-v\,) - hv + ih \qquad (16)$$

in which $(1-v)$ is the fraction of fields, that are not infected. It is assumed, that the colonization ability c is independent of the level of infection in individual fields, because these levels are kept low with chemical control. For the same reason, it is assumed, that the harvest rate h is the same for infected and non infected fields. The harvest rate h will be rather constant. A reduction in the growing period of a leek crop of one month will cause a substantial reduction in yield, but will increase h by only 10 to 20 %. The colonization ability c can vary much more, depending on the distance between the leek fields in a region.

The equilibrium for v, i.e. the value for v when $dv/dt = 0$, is dependent on the ratio c/h and i (see Appendix 3). In Figure 13, the equilibrium for v is given as function of i at different values of c/h. At a ratio c/h larger than 4, reduction in i has little effect on the fraction of infected fields. If the ratio c/h equals 1, then $v = \sqrt{i}$. At values for c/h equal or lower than 1, v is larger than zero if i is larger than zero, thus the persistence of leek rust in a region is caused by the use of infected planting material and the disease may be eradicated from the region by strict use of healthy planting material (see Figure 13).

When information on the actual fraction of infected leek fields in a region (v) and on the proportion of newly planted fields with infected plant material (i) is available, Figure 13 can be used to estimate the ratio c/h. A representative survey of planting material and leek fields, until now not performed, is necessary to provide this information. If $v \leq \sqrt{i}$, then the ratio c/h is estimated between 0 and 1 and the fraction of infected fields will be highly dependent on the proportion of fields planted with infected planting material. In this situation, an obligatory certification of planting material will be highly effective and may even cause the complete eradication of leek rust from the region. For estimated values of c/h greater than 1, the reduction in the disease intensity on a regional level will be less dramatic and there will be less justification for an obligatory certification of planting material. Implementation on a voluntary basis of the certification of planting material can still be very useful, delaying the start of rust epidemics in individual leek fields and hence reducing the number of fungicide sprayings required.

7 General discussion

7.1 Scientific approach

The aim of this study is to develop an improved pathosystem management for leek rust at field, farm and regional level, with a strong emphasis on preventive measures and low use of fungicides. Because information of basic epidemiological characteristics of leek rust epidemics was lacking, these basic temporal and spatial characteristics of leek rust epidemics were studied in experiments under field conditions and after natural infection. The resulting information was integrated in mathematical models that were used to gain better insight in the functioning of the pathosystem at various spatial scales. That insight was used in the development of supervised control measures. The methodology of the individual experiments and models is discussed in the separate chapters. Here, the methodology used across the chapters, i.e. the relation between the epidemiological data obtained from the experiments and their use in the models will be further discussed.

In Chapter 2, the Black-White join-count statistic showed to be an useful technique for the analysis of the spatial distribution of diseased plants. The Black-White join-count statistic, although widely used in other disciplines, is rarely used in plant disease epidemiology. The Black-White join-count statistic provides a descriptive and static analysis. In this study, a static analysis was sufficient, because a sampling pattern was identified in Chapter 4, that was insensitive to the spatial distribution of diseased plants. By assuming the use of this sampling pattern, the calculations of the detection probabilities in Chapter 4 could be done without information on the changes in time of the spatial distribution. The results of the static spatial analysis demonstrated the practical need for such a sampling pattern. More detailed research on the spatial distribution of diseased plants was done for other pathosystems and detailed, explanatory and dynamic models are available, as recently reviewed by Zadoks and van den Bosch (1994). In pathosystems with a strong differential interaction between host cultivars and pathogen races, a practical application of these detailed models is the use of cultivar mixtures suppressive to the disease (cf. van den Bosch et al, 1990). Presently, there is no differential interaction known for the leek rust pathosystem and early British experiments with cultivar mixtures to suppress leek rust remained without success (Anonymous, 1982, 1983).

Information on the growth of leek rust epidemics in time was a prerequisite for the studies on sampling and supervised control (Chapter 4) and management of leek rust (Chapter 5). The investigation of sampling procedures was used for the development of a reliable, simple, accurate and biological sound sampling method for

leek rust. The management systems concern the use of preventive measures to reduce the need for chemical control of the disease. An exponential growth model did satisfactorily describe the early epidemic growth in time in two years from end of July till begin of September (Chapter 3). During this period, the weather is in general favourable for the disease and infected crops show a maximal growth of the leek rust epidemic. The estimates of the relative growth rate during this period were used as constant growth parameters in the simulation models in Chapter 4 and Chapter 5. These models were meant to represent the pathosystem during a much longer period, incorporating periods with possibly less favourable weather conditions. As a result, the models in Chapter 4 and 5 will test the effect of the proposed control strategies under "worst case" situations with regard to disease increase (see below).

At the start of the study, information on basic spatial and temporal characteristics of leek rust epidemics was lacking and experimental research had to be concentrated on the most essential processes (Chapter 2 and 3). Therefore, the models in Chapter 4 and 5 were kept as simple as possible, with the minimal level of complexity necessary for the goals of the models. A higher level of detail of the models would have decreased their representativity, because of the addition of assumptions, that could not be checked against experimental observations (cf. Shannon, 1976).

For a formal uncertainty analysis of the models in Chapter 4 and 5, i.e. an analysis of the effect of errors in assumptions and parameter estimates on the model output, more information on the leek rust pathosystem is necessary than is actually available. In absence of a formal uncertainty analysis, robustness of the models was aimed at by selecting the model assumptions and parameter values on basis of a "worst case" approach. By this the outcome of the models is biased to chemical control.

In Chapter 6, a metapopulation model was used as the basis for a feasibility study to develop guidelines for leek rust management on the regional level. The metapopulation model was used without external data input. For this model to become practically applicable, it still need to be coupled with survey data on the actual disease situation in a region. The model results give an indication, which parameters are to be measured in such a survey. The distance between crops in a farm and in a region and the level of infection of planting material are parameters rarely measured in most disease surveys. The model results indicate their importance in explaining the actual disease situation in a region.

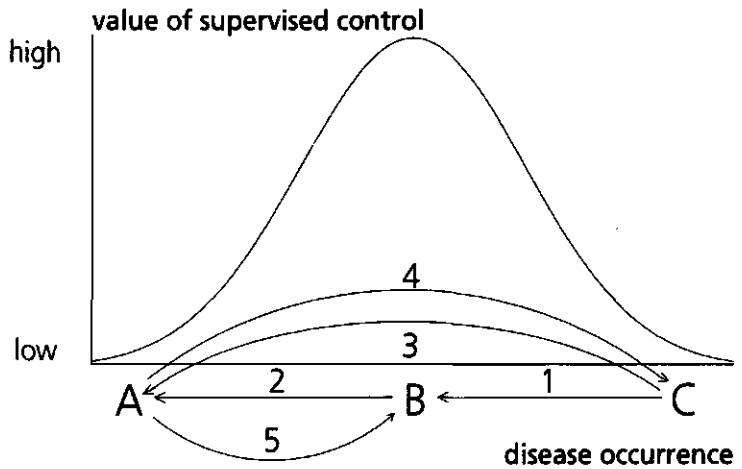

Figure 14. Theoretical relation between value of supervised control and disease intensity, for explanation see text

7.2 Practical implications for disease management at farm level

In Chapter 5, it was shown, that supervised control of leek rust will lead to a reduced use of fungicides, provided that preventive measures on the farm level are adopted. This is illustrated in Figure 14, that may also represent other pathosystems. The abscissa of Figure 14 gives a compound measure of disease occurrence, combining the frequency and intensity of occurrence and damage caused by outbreaks. In point C,there is a high probability of damaging outbreaks every year from the start of the growing season onwards. Leek rust epidemics are common and chemical control has to be applied on a regular basis, starting early in the growing season. Hence, in point C, the value of supervised control in terms of a reduction in sprayings is very low. As long as preventive measures on the farm level are not implemented, a majority of the leek growers is in point C.

With preventive measures on the farm level, the leek rust intensity can be reduced to point B (arrow 1). These preventive measures are the use of healthy planting material and sufficient isolation between leek fields in space or in time (Chapter 5). In point B, outbreaks of leek rust are at a lower frequency and occur usually later, so that

fields can escape from an epidemic, but heavy outbreaks still occur in some fields. In point B, supervised control based on sampling for detection and application of fungicide after detection of leek rust (Chapter 4), is very valuable to ensure reliable disease management with an average low input of fungicides by selective application, as was demonstrated in Chapter 5.

The intensity of leek rust can be further reduced to point A by even better prevention or the still hypothetical use of a resistant cultivar (arrow 2). In point A, outbreaks are very rare, occurring less than once every ten years. Here, supervised control is no longer useful, as no farmer will be spraying routinely against such a rare disease, nor will he be constantly monitoring. An example of such a disease, that is no longer a serious problem in the Netherlands is rust of Phaseolus beans caused by *Uromyces appendiculatus*.

Figure 14 also illustrates, what may happen if a leek cultivar will become available with a much higher level of resistance against leek rust than the presently available cultivars. The disease intensity can be reduced directly from point C to point A (arrow 3), and there will be no interest in other ways of disease prevention or supervised control. As long as the resistance is durable, leek rust will be no problem and a reduction in fungicide use is obtained. But if the resistance is broken, the disease intensity will again increase to point C (arrow 4). Again, fungicides will be used heavily, until a new resistant cultivar is introduced. This has been the case in the barley mildew pathosystem (Wolfe and McDermott, 1994). Because in general, the durability of resistance can not be predicted, preventive measures as indicated in Chapter 5 remain important, even if a leek cultivar is available with complete resistance against leek rust, to reduce the possible amplitude in disease intensity (arrow 5) and to avoid a return to full dependence on chemical control once disease resistance is broken.

7.3 Practical implications for disease management at regional level

For so far, the practical implications of this study are discussed on the farm level, i.e. from the point of view of individual farmers. In Chapter 6, it was argued on basis of a simple model, that the possibilities for disease prevention on a regional level are dependent on the extent to which the disease situation in an individual farm is influenced by the disease situation at other farms in the region. In Chapter 6, this mutual influence between the leek fields in a region was summarized by a ratio between two parameters: *c/h*. If the distance between the fields is shorter and the crops remain longer in the field, the spread of the disease between fields in the region will be larger as is reflected by a higher *c/h* value. If *c/h* is less than one, the spread of

Table 16. The priority for a regional policy for leek rust management as function of the intensity of the leek cultivation for three categories of regions based on values for *c/h*

cropping intensity	*c/h*	priority for regional policy
low	< 1	certification of planting material
medium	≥ 1 ≤ 4	supervised control together with preventive measures on farm level
high	> 4	decrease of intensity of leek cultivation

the disease from one leek field in the region to another is so low, that the disease can only persist in the region due to the use of infected planting material. If *c/h* is higher than 4, the spread of the disease between fields in the region is so strong, that on the average more than 75 % of the leek fields will be infected, even if only healthy planting material is used.

By using the value of *c/h* as an indication of the intensity of leek cultivation and the resulting disease spread between fields in a region, three categories of regions can be distinguished (Table 16). Each of these three categories has it own priority with regard to the regional policy for disease management. In the regions with a low intensity of leek cultivation (*c/h* < 1), the disease can only persist due to the use of infected planting material and the certification of planting material is the first priority for regional disease management. After successful implementation of certification of planting material in these regions, most individual farms experience a disease occurrence as indicated by point A in Figure 14, or the disease occurrence can be reduced to such level by an increased isolation of the leek fields within the individual farm. Promotion of supervised control has therefore low priority as a regional policy for disease management. In the regions with a medium intensity of leek cultivation($1 \leq c/h \leq 4$), the individual farms experience a disease occurrence as indicated by point B in Figure 14, or the disease occurrence can be reduced to such level by preventive measures on farm level, i.e. the use of healthy planting material and an increase in isolation between the leek fields within a farm. In these regions, the promotion of supervised control together with preventive measures on the farm level is the first priority for regional disease management. The regions with a high intensity of leek cultivation (*c/h*) are the problem regions. In these regions, most individual farms will experience a disease occurrence as indicated by point C in Figure 14 and it is

mostly impossible to reduce the disease occurrence to point B due to the high spread of the disease from other farms in the region. In these regions, the hard to achieve priority for disease management on a regional level is the reduction in the intensity of the leek cultivation. Together with reduction in intensity of the leek cultivation, supervised control and preventive measures on farm level has to be promoted. Another possible, but still hypothetical, solution for these regions is the introduction of a complete resistant variety. It should be kept in mind, however, that the spread of a virulent strain of the pathogen will be fast in these regions.

7.4 Further research

The systems approach used in this study has been a strong tool to reach the aim of the development of an improved pathosystem management for leek rust at field, farm and regional level. To validate the proposed strategies for management of the leek rust pathosystem, based on the systems approach used, research on farm and regional level is necessary. Surveys are indispensable to assess the actual situation in a region with regard to disease occurrence, infection of planting material and the spread between fields in one farm or in a region. The models of Chapter 5 and Chapter 6 indicate the importance of parameters, that are rarely measured in disease surveys. Those parameters are the distance between crops in a farm and in a region, their overlap in time and the use of infected planting material. Based on a survey of the disease situation in several farms in different regions, including these parameters, the most promising control strategies, adapted to the specific farms and their regional situations, can be identified and tested on pilot farms. Observations on the changes in leek rust occurrence and resulting fungicide use after the introduction of the control strategies in the pilot farm will yield valuable information on the efficacy of the proposed disease management strategies and the possibilities for further improvement.

Most epidemiological models in phytopathology concentrate on the field level and some models are developed for the spread of diseases on the continental level (Heesterbeek and Zadoks, 1987; Zadoks and van den Bosch, 1994). However, very few models are developed to study pathosystems at a regional level. The results of the preliminary metapopulation model of Chapter 6 indicate its usefulness for the development of disease management policies on a regional level and suggest, that more attention need to be given to the development of metapopulation models in phytopathology.

Samenvatting

Roest in prei wordt veroorzaakt door een parasitaire schimmel, *Puccinia allii*. Het is een lastige ziekte in de commerciële teelt van prei. De oranjebruine sporehoopjes ontsieren het produkt, waardoor de verkoopprijs sterk kan dalen en de teler grote verliezen kan lijden. Uitbreiding van de schimmelziekte in een aangetast gewas kan goed met chemische middelen gestopt worden, maar eenmaal aanwezige sporehoopjes verdwijnen hierdoor niet. Zowel vanuit een economisch als een milieu-technisch gezichtspunt bezien dient het gebruik van chemische middelen tot een minimum beperkt te blijven. De chemische middelen moeten daarom alleen toegepast worden in een preigewas, als zeker is dat de ziekte in het gewas voorkomt en dus niet als een routinematige handeling onder het motto "baat het niet dan schaadt het niet". Het vaststellen van de aanwezigheid van de roest in een groot commercieel preiveld is geen eenvoudige klus, want de sporehoopjes zijn maar klein en zitten verscholen in het gewas. Als men wacht met het uitvoeren van een chemische bestrijding totdat de ziekte duidelijk zichtbaar is, dan zijn de planten al zodanig beschadigd, dat hun verkoopprijs veel lager zal zijn.

In dit proefschrift wordt een eenvoudig recept voor het waarnemen van de ziekte voorgesteld. Hiermee kunnen preitelers met een betrekkelijk kleine inspanning de ziekte in een preiveld kunnen aantonen, lang voordat de ziekte schade heeft veroorzaakt. Deze methode bestaat uit het wekelijks nauwkeurig bekijken van 50 planten, die op een bepaalde manier over het preiveld verdeeld zijn. Als de ziekte waargenomen wordt, kunnen de wekelijkse waarnemingen gestopt worden en kan gestart worden met chemische bestrijding. De manier, waarop de waargenomen planten over het veld verdeeld zijn is van groot belang voor het succes van deze waarnemingsmethode. Met behulp van computerberekeningen is aangetoond, dat de planten het best gelijkmatig verdeeld over vier rijen gekozen kunnen worden, waarbij deze rijen zelf ook gelijkmatig over het veld verdeeld moeten zijn. Wanneer de planten langs een diagonaal of langs beide diagonalen door het veld worden gekozen, hetgeen een gebruikelijke methode is voor gewasbemonstering, dan zullen veel meer planten bekeken moeten worden om met dezelfde nauwkeurigheid de ziekte op te kunnen sporen.

Voordat dit eenvoudige recept opgesteld kon worden, is eerst onderzocht hoe snel de preiroest zich vermenigvuldigt in een preiveld en hoe de aantasting verdeeld kan zijn over het veld. Hiervoor zijn veldproeven uitgevoerd en is uitgebreid aandacht besteed aan de wiskundige methoden om de veldgegevens te analyseren. Voor de analyse van de verdeling van zieke planten over het veld is gebruik gemaakt van een veel gebruikte statistische methode (Black-White join-count statistic), die binnen de

planteziektenkunde echter nauwelijks toegepast wordt. De mogelijkheden en beperkingen van deze methode zijn behandeld. In de experimenten, beschreven in dit proefschrift, werd het ras Albana duidelijk meer aangetast dan de rassen Cortina en Carina. Bij de wiskundige modellen, die gebruikt zijn voor de analyse van de groei van de ziekte in deze rassen, is zowel rekening gehouden met de kruisinfectie tussen de verschillende experimentele veldjes onderling, als met de verschillen in nauwkeurigheid van waarnemen bij verschillende aantastingsniveaus.

In dit proefschrift is ruime aandacht gegeven aan de mogelijkheden om te voorkomen, dat preivelden geïnfecteerd raken met preiroest. Preiroest kan buiten de preiplant niet overleven. De roest overwintert in de zogenaamde winterprei, die in juli of augustus wordt geplant en het volgende jaar wordt geoogst, soms pas in mei. Vanuit deze winterprei infecteert de roest in het voorjaar de nieuw geplante preivelden. Aangezien het hele jaar door prei op het veld staat kan preiroest steeds van gewas naar gewas overspringen. Roestsporen worden door de wind van het ene gewas naar het andere verspreid en dit gebeurt makkelijker wanneer de preivelden dicht bij elkaar liggen. Behalve dat een nieuw geplant preiveld geïnfecteerd kan raken door preiroest vanuit een naburig besmet veld, kan een nieuw geplant preiveld ook besmet raken doordat een gedeelte van het plantmateriaal besmet was. In dit proefschrift is een wiskundig model gebruikt om het aantal bespuitingen tegen preiroest te berekenen, dat nodig is onder verschillende omstandigheden wat betreft de verspreiding van de ziekten tussen velden binnen een bedrijf. Hierbij is ervan uitgegaan, dat een bestrijding tegen preiroest pas wordt uitgevoerd wanneer de ziekte in het veld is waargenomen. De resultaten tonen het belang van preventieve maatregelen aan. Door de preivelden voldoende ver uit elkaar te leggen en ervoor te zorgen, dat het plantmateriaal voor honderd procent gezond is, wordt de kans dat een preiveld met preiroest besmet wordt sterk verkleind en hoeft er veel minder vaak een chemische bestrijding toegepast te worden. Wanneer het niet mogelijk is de velden voldoende ver uit elkaar te leggen, dan moet het elkaar overlappen in de tijd van opeenvolgende preiteelten vermeden worden. De resultaten van het model illustreren ook wat er gebeurt als er geen voldoende aandacht aan preventieve maatregelen wordt gegeven. De ziekte zal dan zo snel de gewassen infecteren, dat het aantal op waarneming van de ziekte gebaseerde bespuitingen weinig verschilt van het aantal bespuitingen bij een routinematige bestrijding. Het waarnemen van de ziekte heeft dus alleen zin bij voldoende aandacht voor preventieve maatregelen.

Een eenvoudig wiskundig model is gebruikt om het effect van het gebruik van ziek plantmateriaal op het optreden van de ziekte in een regio te bereken. De resultaten geven aan, dat ook op regioniveau het gebruik van gezond plantmateriaal een belangrijke preventieve maatregel kan zijn. Wanneer in een bepaalde streek de

preivelden ver uit elkaar liggen, maar preiroest toch regelmatig voorkomt, dan is het verstandig te inventariseren hoe vaak het plantmateriaal besmet is. Wanneer dan blijkt, dat het merendeel van de besmettingen veroorzaakt worden door besmet plantmateriaal, kan door een strenge controle op de gezondheid van het plantmateriaal de preiroest in die streek een zeldzame ziekte worden.

Op basis van de resultaten van de wiskundige modellen in dit proefschrift wordt een streekafhankelijke aanpak van de preiroest voorgesteld. In gebieden met een lage teeltdichtheid, waar toch veel preiroest voorkomt, is een strenge controle op de gezondheid van het plantmateriaal van belang. In gebieden met een gemiddelde teeltdichtheid, is het toepassen van preventieve maatregelen op bedrijfsniveau samen met bestrijding op basis van waarneming de aangewezen methode om de ziekte met een minimum aan bespuitingen te beheersen. In gebieden met een zo hoge teeltintensiteit, dat een voldoende afstand tussen preivelden niet mogelijk is, kan het aantal bespuitingen tegen preiroest alleen teruggebracht worden door het verlagen van de teeltintensiteit.

De voorgestelde maatregelen op veld-, bedrijfs- en streekniveau zijn nog niet uitgebreid onder praktijkomstandigheden getoetst. Het best kan dit gebeuren door het inventariseren van de ziekte op een groot aantal bedrijven, in verschillende streken en gedurende een aantal jaren. De resultaten van dit proefschrift geven aan, dat het meten van de afstand tot naburige preivelden en het ziekteniveau van plantmateriaal van groot belang is bij deze inventarisatie.

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Appendix 1 Mean and variance of the BW statistic

Under assumption of nonfree sampling, i.e. situation that the probability of an individual plant to be infected is not known from an independent sample:

$$\mu = \frac{S_0 n_d n_h}{n^{(2)}} \tag{1}$$

$$\sigma^2 = \frac{1}{4}\left[\frac{2S_1 n_d n_h}{n^{(2)}} + \frac{(S_2 - 2S_1) n_d n_h (n_d + n_h - 2)}{n^{(3)}} + \frac{4(S_0^2 + S_1 - S_2) n_d^{(2)} n_h^{(2)}}{n^{(4)}}\right] - \mu^2 \tag{2}$$

where n_d is the number of diseased plants, n_h is the number of healthy plants, n is the total number of plants and

$$n^{(b)} = n(n-1)\ldots(n-b+1) \tag{3}$$

$$S_0 = \sum_{i=1}^{n}\sum_{j=1}^{n} \delta_{ij} \tag{4}$$

$$S_1 = \frac{1}{2}\sum_{i=1}^{n}\sum_{j=1}^{n} (\delta_{ij} + \delta_{ji})^2 \tag{5}$$

$$S_2 = \sum_{i=1}^{n} (\delta_{i.} + \delta_{.i})^2 \tag{6}$$

where

$$\delta_{i.} = \sum_{j=1}^{n} \delta_{ij} \tag{7}$$

$$\delta_{.i} = \sum_{j=1}^{n} \delta_{ji} \tag{8}$$

For a set of rows with no spatial connection between the rows, it follows from

formulae (1) from Chapter 1 and (1) to (8), that S_0, S_1 and S_2 can be calculated as:

$$S_0=2N(M-K) \tag{9}$$

$$S_1=4N(M-K) \tag{10}$$

$$S_2=N(16(M-2K)+8K) \tag{11}$$

where N is the number of rows, M is the number of plants per row and K is the order of neighbouring ($K \leq \frac{1}{2}M$).

Source of formulae (1) to (8) is Cliff and Ord (1981).

Appendix 2 Derivation of equation (4) from chapter 2

A discrete version of model (2) from Chapter 3 is:

$$X_t = X_0(1+r_d)^t \quad (1)$$

with difference equation:

$$X_{t+1} = (1+r_d)X_t \quad (2)$$

in which r_d is the growth rate for discrete time steps (days).
Formula (2) can be rewritten as:

$$X_{t+1} - X_t = r_d X_{t-p}(1+r_d)^p \quad (3)$$

in which p is the latent period in days.
Omitting the term for alloinfection from model (3) from Chapter 3 yields:

$$X_{t+1} - X_t = RX_{t-p} \quad (4)$$

Combination of equation (3) and (4) gives:

$$R = r_d(1+r_d)^p \quad (5)$$

From equation (1) from Chapter 3 and (1) it follows that:

$$r_d = e^r - 1 \quad (6)$$

Substituting r_d by r in (5) gives:

$$R = (e^r - 1) * e^{rp} \quad (7)$$

Appendix 3 Equilibrium for v in model (16) from Chapter 6

$$cv(1-v)-hv+ih = 0 \tag{1}$$

$$\frac{c}{h}v(1-v)-v+i = 0 \tag{2}$$

$$-\frac{c}{h}v^2+(\frac{c}{h}-1)v+i = 0 \tag{3}$$

For $c = 0$ the solution is:

$$v=i \tag{4}$$

For $c > 0$ and $i > 0$, there is a positive solution:

$$v = \frac{(\frac{c}{h}-1)+\sqrt{(\frac{c}{h}-1)^2+4\frac{c}{h}i}}{2\frac{c}{h}} = \tag{5}$$

$$= \frac{1}{2}(1-\frac{h}{c})+\frac{1}{2}\sqrt{(1-\frac{h}{c})^2+4i\frac{h}{c}} \tag{5b}$$

For $i = 0$ and $c/h > 1$, there is a positive solution:

$$v=1-\frac{h}{c} \tag{6}$$

Nawoord

Kort voor het drukklaar maken van de tekst van dit proefschrift kreeg ik een overdruk van een artikel uit 1983: "Discovering an epidemic before it has reached a certain level of prevalence" (Metz, Wedel and Angulo, Biometrics 39: 765-770), dat een wiskundig stevig onderbouwde uitwerking geeft van een van de problemen uit Hoofdstuk 4 van dit proefschrift. Alleen de toepassing is verschillend: vroegtijdige detectie van ziekten in een proefdierencentrum. Hoofdstuk 4 was al als artikel gepubliceerd en ik heb het daarom niet meer veranderd, ondanks de nieuwe ideeën die ik uit bovengenoemde referentie kon putten. Hiermee worden naar mijn idee twee voor de hand liggende zaken geïllustreerd. Ten eerste, een artikel - en dus ook een proefschrift - kan nooit meer zijn dan de weergave van de stand van zaken op een bepaald moment. Nieuw beschikbaar gekomen referenties (en dat kunnen best artikelen van twaalf jaar of langer geleden zijn) en nieuwe discussies kunnen een reeds gepubliceerd verhaal in een heel ander perspectief plaatsen. Ten tweede, oplossingen liggen soms al kant en klaar voor het grijpen in naburige vakgebieden en het is daarom goed, ook voor fytopathologen, om kennis te nemen van de geschiedenis en de ontwikkelingen in de naburige vakgebieden. De binnen de fytopathologie invloedrijke en veel geciteerde modellen van Vanderplank, gepubliceerd in 1963, waren in meer generieke vorm al gepubliceerd in 1927 door Kermack en McKendrick. Een vergelijkbare voorkeur bij fytopathologen om het wiel opnieuw uit te vinden wordt in Hoofdstuk 2 gesignaleerd. En door niet op de hoogte te zijn van het bovengenoemde artikel uit 1983, en wie weet hoeveel meer artikelen uit de humane en veterinaire epidemiologie, heb ook ik mij aan dezelfde fout schuldig gemaakt, waarvan ik nu mijn vakbroeders beticht.

Binnen de afdeling van IPO-DLO, die gedurende de afgelopen vier jaar getooid is met de namen "Kwantitatieve Ecologie" en "Systeemanalyse en geïntegreerde bestrijding", heb ik een stimulerende werkomgeving gevonden, juist omdat binnen deze afdeling verschillende disciplines vertegenwoordigd zijn. Hoewel het modelmatige werk binnen deze afdeling de komende jaren minder aandacht zal krijgen, hoop ik dat de multidisciplinaire atmosfeer nog lang mag heersen en zijn vruchten afwerpen. Het nadenken en discussiëren over andere problemen dan die van het directe eigen onderzoeksgebied wordt tegenwoordig steeds meer als een luxe gezien. Hierbij wordt over het hoofd gezien, dat deze uitwisseling van gedachten de efficiëntie van onderzoek kan verhogen.

Dit pleidooi voor samenwerking kan natuurlijk niet zonder een woord van dank aan allen, met wie ik de afgelopen vier jaar heb samengewerkt. Gerard Hasper en Gijs Schelling voor hun hulp bij de veldexperimenten. Jan Theunissen voor zijn kennis, collegialiteit en enthousiasme als leider van de vollegrondsgroenten groep. Jan Willem

Henfling en Kees Booij, de afdelingshoofden, die steeds het belang van promoveren benadrukten en mij de ruimte hebben gegeven om aan het proefschrift te werken. Joop de Bree en Hans Heesterbeek voor hun commentaar, dat zich niet alleen tot de wiskunde in de artikelen beperkte, en alle deelnemers aan de promovendigroepen en de anonieme referenten voor hun waardevolle kritiek en suggesties.

Veel dank ben ik verschuldigd aan Rudy Rabbinge en Richard Daamen, voor het op zich nemen van de taak van respectievelijk promotor en co-promotor. Ik kon altijd rekenen op gedegen en verfrissend commentaar op manuscripten, vaak al na verbluffend korte tijd. Zo bleef de vaart erin. Ook wat betreft de organisatorische aspecten van het proefschrift.

Tenslotte, omdat zij er als eerste waren, wil ik mijn ouders bedanken voor hun voortdurende belangstelling voor mijn studie en werk.

Curriculum vitae

Pieter Dirk de Jong werd op 15 juli 1964 in Den Haag geboren. Hij behaalde in 1982 het VWO-diploma aan het Gymnasium Haganum te Den Haag. In 1987 voltooide hij de opleiding tot planteziektenkundige aan de Landbouw Universiteit te Wageningen, met als afstudeervakken nematologie, fytopathologie en theoretische productie ecologie. Van juni 1988 tot maart 1991 was hij in dienst van het toenmalige Centrum voor Agrobiologisch Onderzoek (CABO), nu AB-DLO, als medewerker van het project Simulation and Systems Analysis in Rice Production. Van maart 1991 tot mei 1995 was hij in dienst bij het Instituut voor Planteziektenkundig Onderzoek (IPO-DLO) binnen het project geleide bestrijding van bovengronds infecterende schimmelziekten in vollegrondsgroenten. Sinds 1 oktober 1995 werkt hij bij het Rijksinstituut voor Visserij Onderzoek (RIVO-DLO) aan projecten op het gebied van kustwaterecosystemen.