European Journal of Plant Pathology **102:** 365–375, 1996.

Rain-driven epidemics of *Phytophthora porri* on leek

W.D. Smilde[1,2], M. van Nes[1,2] and H.D. Frinking[1]
[1]*Department of Phytopathology, Wageningen Agricultural University, P.O. Box 8025, 6700 EE Wageningen, The Netherlands;* [2]*DLO-Centre for Plant Breeding and Reproduction Research (CPRO), Department of Vegetable and Fruit Crops, P.O. Box 16, 6700 AA Wageningen, The Netherlands (Fax +31-317-416513)*

Accepted 21 November 1995

Key words: Allium porrum, host growth, degree-days, incubation period, splash dispersal, oospores

Abstract

White tip disease of leek (*Allium porrum*), caused by *Phytophthora porri*, was studied in field experiments. On fields infested by soil-borne inoculum (oospores), relatively short periods of explosive disease increase alternated with periods in which apparently no new infections occurred. The analysis of rain data and disease data, using a degree-day model for incubation periods at constant temperatures, confirmed the hypothesis that disease increase of *P. porri* is significantly correlated with rain; R^2_{adj} was 0.91, 0.41 and 0.51 in 1992, 1993 and 1994, respectively. Correlations were highest early in the season. Lack of correlation later in the season may be ascribed to the effect of lesion death, which may be caused by total or partial leaf death, by desiccation or by other fungi overgrowing *P. porri*, and to the effect of secondary infection by zoosporangia, which appears to be not so strongly rain-driven as primary infection. Zoosporangia were observed in fields on water-logged light-green lesions. High lesion densities of leaf tips and leaf units at 10–20 cm above the leaf axils indicated that most infections depend on free water, either in puddles or in a water basin near the leaf axils. Although disease correlates well with rain data, disease forecasts will be unreliable as long as rain forecasts are unreliable.

Introduction

White tip disease of leek (*Allium porrum* L.) is a soil-borne leafspot disease, caused by *Phytophthora porri* Foister. The disease, first recorded in Scotland in 1928 by Foister [1931] as a common and disastrous disease, steadily decreased in importance in Scotland during 1931–1948, until a resurgence took place in 1949 [Foister, 1961]. In the Netherlands it was first described by Van Hoof [1959], mentioned by Van Bakel [1964] as a minor problem, but later by Alofs [1986] as one of the most prominent diseases of leeks during winter. The Dutch leek area has increased from 1285 ha in 1975 to 4250 ha in 1994 [De Kraker and Bosch, 1993; CBS, 1994] and epidemics of *P. porri* have become common, especially in the southern provinces of the Netherlands, where 80% of the Dutch leek production is concentrated.

Foister [1931] observed that the onset of epidemics of *P. porri* in Scotland may be early in August or as late as December. He could not explain this variation, partly because of his theory that sporangia, growing saprophytically on soil and discharged by wind, were the most important infection agents. He did not find sporangia on leaves under field conditions. Legge [1951] demonstrated that a *Campanula*-isolate of *P. porri* may produce sporangia in non-sterile soil. Saprophytic growth in non-sterile soil was, however, limited. Van Hoof [1959] did not find sporangia on leaves at all, and suggested that oospores are the main source of infection. Taylor [1965], however, reported the occurrence of sporangia on wet foliage after prolonged dews for a few days. Ogilvie and Walton [1941] observed that from a few initial attacks the disease spread outwards in a circle, most infections taking place during wet weather. Yokoyama [1976] studied epidemics of *P. porri* on winter-grown onion (*Allium cepa* L.) in the south of Japan and concluded that disease was most severe in March when rainfall and temperature were relatively high in previous December and February.

Grill [1985] reported that the onset of *P. porri* on leek in France occurred between September 5 and October 15 in 1980–1984, and observed that humid conditions and relatively high temperatures in winter favour the disease.

The initial (oospore-) inoculum of *P. porri* is soil-borne, but infections occur only on the plant parts above the ground. Secondary inoculum (sporangia) is only found on wet leaves. The wet sporangia are not discharged easily by wind only, so the main discharge mechanism could be rain splash. Spore discharge by rain splash may therefore be a limiting factor for disease progress. This crucial role of splash dispersal has been described in other pathosystems, e.g. black pod (*P. palmivora* (Butler) Butler) of cocoa [Thorold, 1975], leather rot (*P. cactorum* (Lebert & Cohn) Schröter) of strawberry [Madden et al., 1992], and other plant diseases [Fitt et al., 1989]. In analogy with these pathosystems, we hypothesize that for *P. porri* rain is the main factor explaining disease progress, because of the direct effect on the discharge and dissemination of spores, and the indirect effects on sporulation and infection. The direct and indirect effects cannot be separated from each other in field experiments.

In the present study, epidemic progress curves of *P. porri* were obtained in three successive years. These curves probably describe rain-driven epidemics, i.e. epidemics for which rain is the main factor for disease progress. Several problems related to the interpretation of these curves were studied. First, the degree of correlation between rain and disease was quantified, and periods with low correlation were identified. Sporulation was observed qualitatively. Second, the turn-over of leaves was studied, as the continuous appearance and death of leaves might disturb the correlation between rainfall and disease increase. Third, lesion dynamics were investigated by direct observation of the fate of individual lesions, and by a study of disease profiles.

This study aims at a better understanding of the relative importance of the various processes affecting epidemics of *P. porri*, and may contribute to an effective research strategy for disease control.

Materials and methods

General. A series of field experiments was performed in 1992–1994 (Table 1). The experiments were located at CPRO-DLO ('De Goor'), Wageningen. Plants were sown in April in seedbeds and transplanted to fields in the first week of July, in planting holes of 12 cm depth and at planting distances of 12 × 50 cm. Fertilizer was applied according to professional advice based on soil samples [De Kraker and Bosch, 1993]. Weather data were recorded in a weather station placed at the border of the field in 1992 and 1993. In 1994 these data were obtained from the meteorological station 'De Haarweg' at ca. 3 km distance. In all experiments the crop was initially infected by soil-borne inoculum, produced by infested plants at the same field in previous years.

Epidemic curves. To obtain epidemic progress curves of *P. porri*, the number of diseased leaves per plant (D) was determined in naturally infested leek crops in 1992/3, 1993/4 and 1994, mostly at weekly intervals, from planting in July till the end of the experiment in February, March and December, respectively. Three cultivars were used in 1992 (in plots of 45 plants, with three rows of 15 plants) and in 1993 (in four single-row plots of 10 plants). In 1994, three epidemic curves were obtained from three plots of 180 plants with different cropping histories (no, one or two previous years with a diseased crop), supposedly leading to different levels of natural infestation from soil. In 1992 plots were planted adjacently; in 1993, they were separated by a single row of plants (cvs. Carina or Derrick), and in 1994 they were separated by fallow strips of 2 m width.

Correlation of disease and rain. From the epidemic progress curves, the degree of correlation was determined for the average daily increase of the number of diseased leaves (DR) and the average daily rainfall (RR) in an infection period (the period in which the fungus probably penetrated the leaf) one incubation period before the observation interval of DR. The infection period was calculated from measured daily mean temperatures during the incubation periods. First, at each time of observation the shortest and longest incubation time were calculated as 92 and 154 degree-days above −3 °C, respectively, multiplied with the daily mean temperatures during incubation. The degree-day model was derived from infection experiments in growth cabinets at constant temperatures (Smilde et al., submitted). Second, for each observation interval the corresponding infection period was calculated by subtracting the longest incubation time from the observation interval's first day, and the shortest incubation time from the observation interval's last day. Third, the average rainfall ($mm.d^{-1}$) in the

Table 1. Summary of experiments

Purpose	Planting date	Observed variables	Interval (days)	Plants total	Plants plot^{-1}	Plots	Blocks[1]	Cvs[2]
Epidemic curves	9-7-92	D,T,R	7	135	45	3	–	1–3
Turn-over of leaves	"	N,N_{new}	30	360	15	8	–	1–8
Epidemic curves	7-7-93	D,T,R	7	120	10	12	1–4	1–3
Turn-over of leaves	"	N,N_{new}	30	200	10	20	1–4	1–5
Turn-over of lesions	"	U_{new},X_{new}	7	45	5	9	5–7	1–3
Disease profiles	"	U(i),X(i)	"	"	"	"	"	"
Causes of lesion death	"	D,D_{new}	5–14	600	300	2	–	1
Epidemic curves	14-7-94	D,T,R	7	540	180	3	–	1–3

[1] Block codes 1–7 indicate seven different blocks
[2] Cultivar codes 1: Carina, 2: Wintina, 3: 91021, 4: Derrick, 5: Platina, 6: Gavia, 7: Portant, 8: Porino

calculated infection period was determined. Regression analysis of DR on RR yielded the R^2_{adj} values and their levels of significance.

Turn-over of leaves. To measure the turn-over of leaves, the total number of leaves (N) and the numbers of new leaves (N_{new}) were determined at monthly intervals from October to February, in 1992/3 in the middle rows (15 plants) of plots of 45 plants of 5 cultivars, and in 1993/4 in 4 plots of 10 plants of 8 cultivars. The same plants were also used for monitoring epidemic progress, as described above. N_{new} was determined with the help of 1-cm plastic pegs that were placed on the second leaf (top leaf = first leaf) at each time of observation.

The average lifetime of leaves (LN) was calculated as the inverse of the relative growth rate of leaves ($N.\delta/N_{new}$, with δ = the observation interval for N_{new}; cf. Table 2). In principle, relative death rates of N could also be used to calculate LN, but considering that growth rates were less influenced by short-term weather and disease fluctuations than death rates, and that full-grown plants have a rather constant number of 8–10 leaves, growth rates were preferred.

Turn-over of lesions. The distribution of lesions over leaves was determined from 6-9-93 till 15-11-93, using diagrams (Figure 1) for recording the length of each leaf and the position of each lesion on 10-cm sections of leaves graphically at weekly intervals. A total of 45 plants was used, representing 3 cultivars and 3 blocks. From plots with 10 plants each, five plants were chosen randomly for observation. The plants were not used in another experiment. The turn-over of lesions was studied through comparison of diagrams of successive

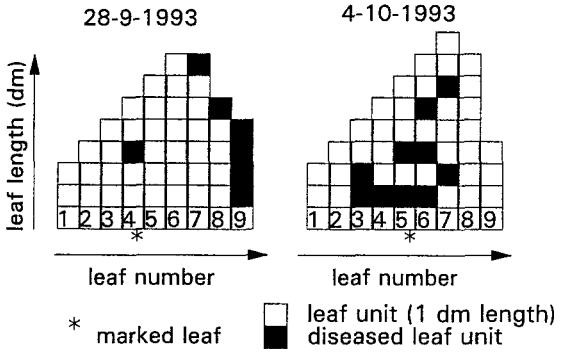


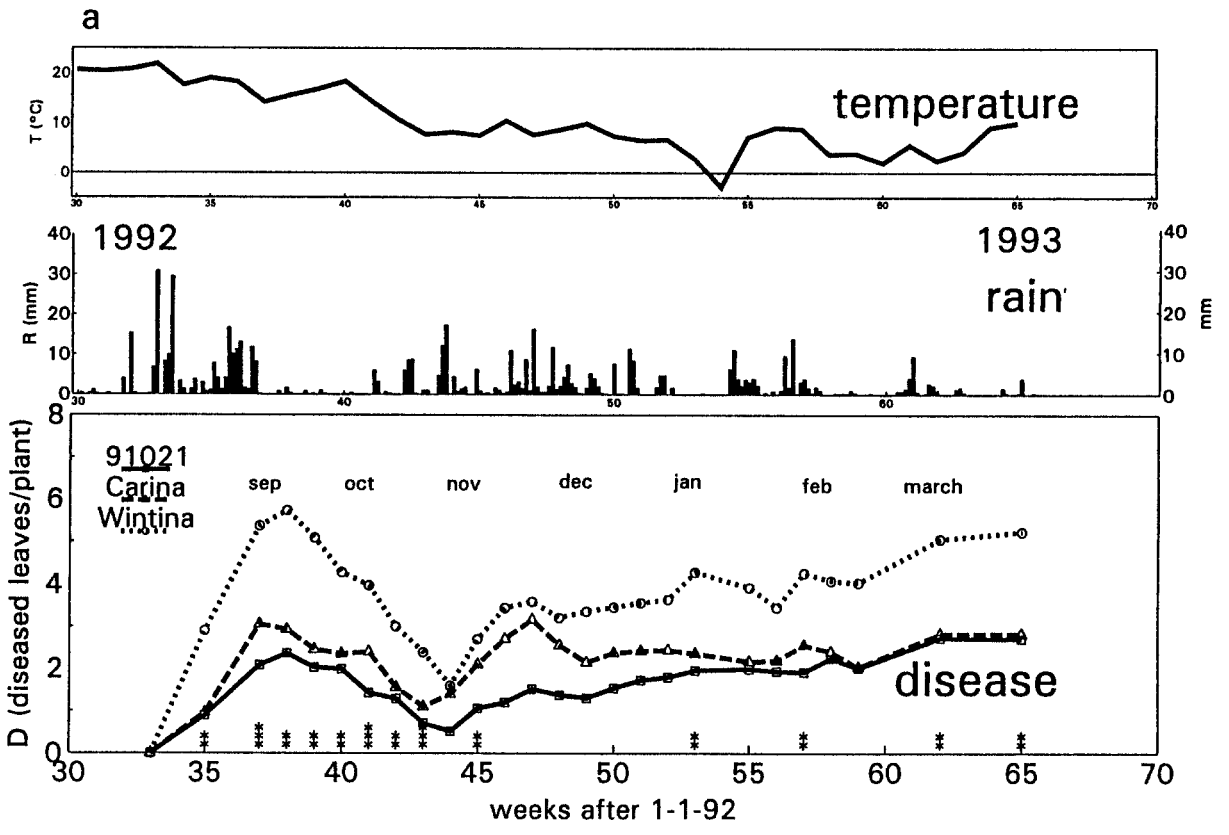
Figure 1. Two plant diagrams of the same plant before (left) and after (right) week 40 of 1993, illustrating the method of observation of disease profiles. During week 40 one new leaf appeared and one old leaf died. Therefore the leaf numbers of individual leaves were raised by one. Leaf 4(→5) was marked with a peg, leaf 7 (→8) lost a lesion, and leaf 8 (→9) lost a diseased leaf tip.

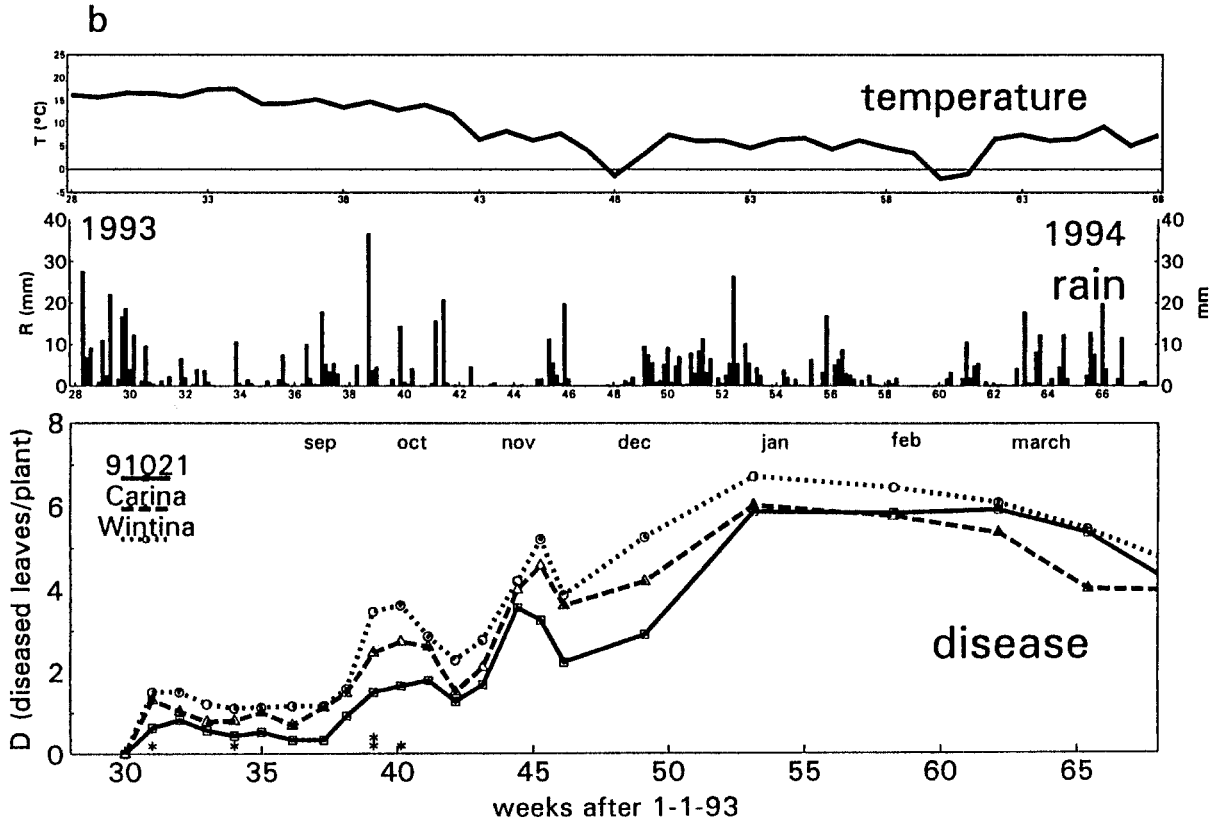
observation times. One leaf of each plant was marked with a peg to make this comparison possible.

The average lifetime of diseased leaf units LX was calculated as the inverse of the relative decay rate of diseased leaf units ($X.\delta/X_{removed}$, with δ = the observation interval for $X_{removed}$). For calculating the average lifetime of leaf units LU the inverse growth rate ($U.\delta/U_{new}$) was used, because growth rates of leaves were less sensitive to environmental factors than decay rates.

Disease profiles. Two types of disease profile were studied from the diagrams mentioned above: disease as a function of leaf number and disease as a function of distance along the leaf, measured from the soil surface.

For the disease profile over leaf numbers, the standardized number of lesions at the i^{th} and younger


a
temperature
T (°C)
1992
1993
rain
R (mm)
mm
91021
Carina
Wintina
sep
oct
nov
dec
jan
feb
march
disease
D (diseased leaves/plant)
weeks after 1-1-92


b
temperature
T (°C)
1993
1994
rain
R (mm)
mm
91021
Carina
Wintina
sep
oct
nov
dec
jan
feb
march
disease
D (diseased leaves/plant)
weeks after 1-1-93

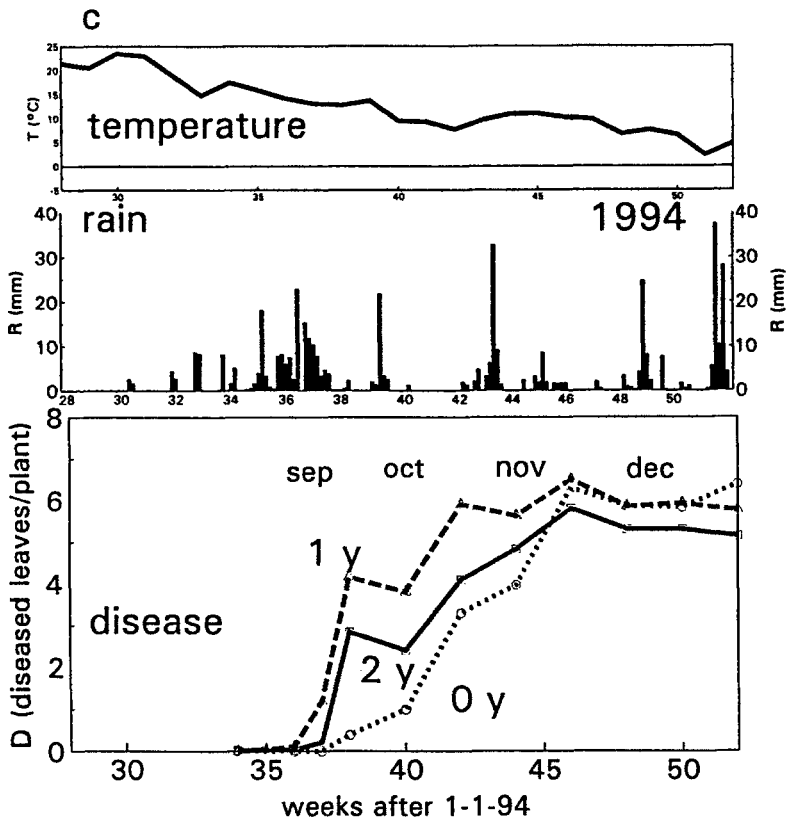


Figure 2. Epidemic curves in 1992/3 (a) and 1993/4 (b) and 1994 (c), with daily rainfall and weekly mean temperature data. In (a) and (b) three cultivars were used. Significance of cultivar effect is indicated below disease graphs with * ($P<0.05$), ** ($P<0.01$) and *** ($P<0.001$) in F-tests (replicates in (a) 3 rows and in (b) 4 blocks). In (c) three plots were used with natural infestation from 0, 1, or 2 previous years with diseased leek crops grown at the same place.

leaves x′(i), and the standardized number of leaves u′(i) for all leaf numbers i were calculated for each plant, following Daamen [1989]. Averages of x′(i) and u′(i) were taken over groups of ten values in a column of x′(i) or u′(i) values sorted on size for each cultivar. Plots of x′(i) against u′(i) were constructed with these averages.

For the disease profile over distances along the leaf, the lesion density was plotted as a function of distance along the leaf. The lesion density was calculated as the number of lesions divided by the total number of leaf sections at each distance along the leaf, and was expressed as a percentage.

Causes of lesion death. To study the causes of lesion death, diseased leaves of 600 plants were marked with a plastic peg from 17-9-93 till 5-11-93 at intervals of 5–14 days. At each observation time new diseased leaves (D_{new}) were marked with a peg and counted; pegs were removed from dead leaves (without green leaf area) and leaves that had lost signs of *P. porri* without dying off totally. In this way the effect of leaf death, which could be an important reason for disappearance of lesions, was distinguished from other causes of lesion death.

Data from this experiment were also used for the calculation of the lifetime of diseased leaves LD (= $D.\delta/D_{removed}$).

Results

Epidemic curves. In Figure 2, epidemic curves, rain- and temperature data are presented for three seasons. The curves fluctuated strongly from August till November, indicating (1) that infection occurs during short periods, alternated with longer periods without new infections, and (2) that the lifetime of

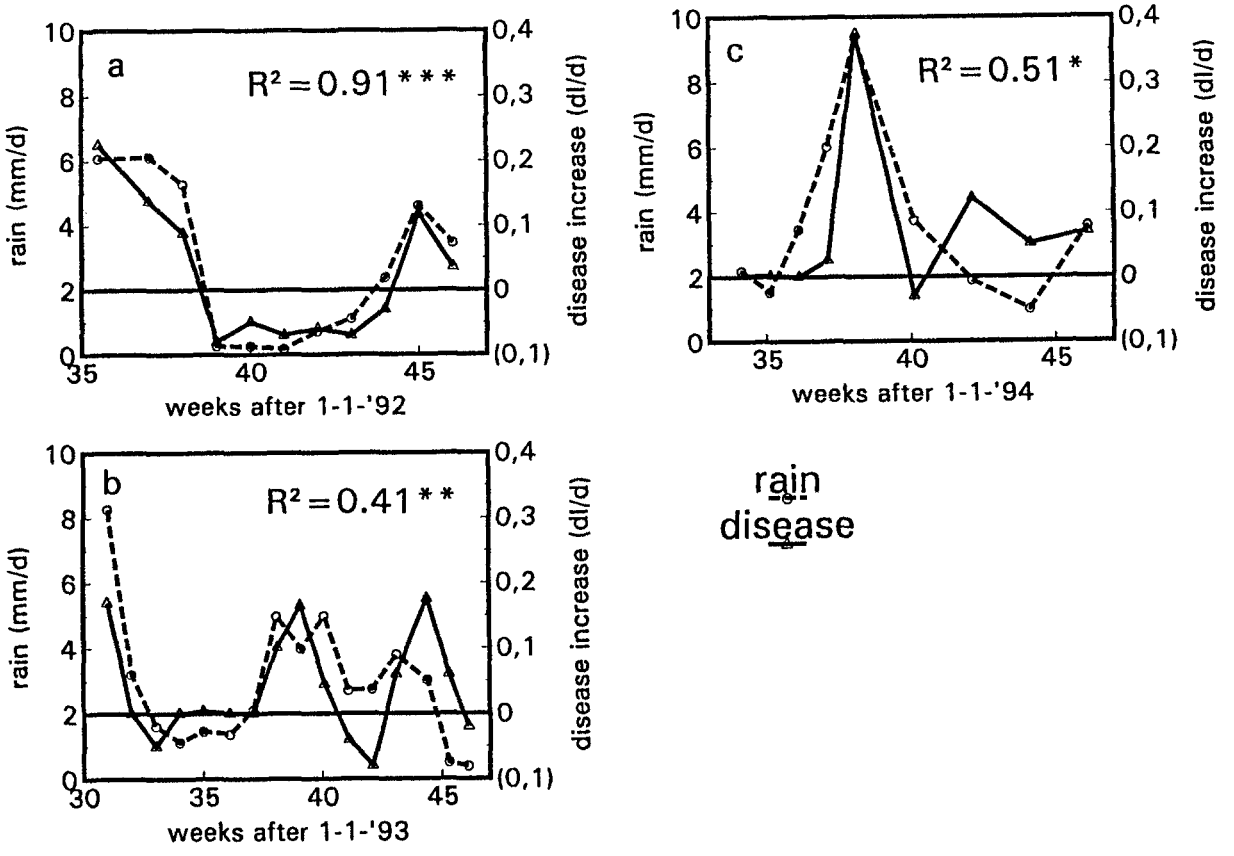


Figure 3. Transformed disease and rainfall curves in 1992 (a), 1993 (b) and 1994 (c), illustrating the relation between disease increase (DR) in observation intervals and average daily rainfall (RR) in calculated infection periods, one incubation perod before the observations. A degree-day model was used to calculate infection periods from observation intervals (see text). dl/d = diseased leaves per day.

individual lesions and diseased leaves is short relative to the total period of epidemic build-up.

Statistical significance of cultivar effects on D for single time points is indicated in Figure 2. In 1992 the cultivar effect is possibly overestimated because of a location effect which is confounded with the cultivar effect. It appears that cultivar differentiation is most pronounced before November, during or shortly after an infection wave. In 1993, differences between cultivars were generally smaller than in 1992, possibly because of the smaller plots and the proper separation of cultivar and location effects. Cultivar × time interactions are further analyzed in Smilde et al. [1995].

The onset of the epidemic in 1994 was latest in the plot without a previous leek crop. The inoculum for this late onset probably originated in the other plots. The plot with one previous diseased leek crop had more disease initially than the plot with two such crops, indicating that the amount of initial inoculum was not limiting for initial infection in either plot. Plot scores converged in November, and in December the differences between plots became negligible. This indicates the increasing importance of secondary inoculum.

Correlation of disease and rain (Figure 3). To investigate the possibility that infection periods are associated with rainfall, DR and the corresponding RR were plotted against time. In '91, '92 and '93, R^2_{adj} was 0.91, 0.41 and 0.51, respectively, for August–November. These values were statistically significant at $P<0.001$, $P<0.01$ and $P<0.05$, respectively.

The onset of the epidemics in week 35 (1992), week 31 (1993) and week 37 (1994), was preceded by >15 mm rain on a single day 8–12 d earlier. Each infection wave (2 in 1992, 3 in 1993, 2 in 1994) was preceded by >20 mm rain divided over 2–3 days, 8–21 d earlier.

Sporulation. Sporangia and zoospores were observed on and in fresh leaf material through a binocular microscope at 50× magnification in autumn and winter

Table 2. Parameters used for epidemiological studies. t = observation time, δ = length of previous observation interval, i = leaf number, max =maximum leaf number

Weather variables		
T	Daily mean temperature	[°C]
RR	Average daily rainfall	[mm.d^{-1}]
Disease variables		
D	Number of diseased leaves	[#.plant^{-1}]
DR	Average daily increase of D = [D(t)−D(t−δ)]/δ	[#.(plant.d)$^{-1}$]
D_{new}	Number of leaves, healthy at t−δ, diseased at t	[#.plant^{-1}]
$D_{removed}$	Number of leaves, diseased at t−δ, removed at t	[#.plant^{-1}]
LD	Average lifetime of diseased leaves	[d]
X	Number of diseased leaves = x(max)	[#.plant^{-1}]
X_{new}	Leaf units, healthy at t −δ, diseased at t	[#.plant^{-1}]
$X_{removed}$	Leaf units, diseased at t − δ, removed at t	[#.plant^{-1}]
LX	Average lifetime of diseased leaf units	[d]
x(i)	Number of diseased leaf units on leaf $\leq$i[1]	[#.plant^{-1}]
x′(i)	Standardized x(i) = x(i) / x(max)	[–]
Crop variables		
N	Number of leaves	[#.plant^{-1}]
N_{new}	Number of leaves that appeared since t − δ	[#.plant^{-1}]
$N_{removed}$	Number of leaves that died since t − δ	[#.plant^{-1}]
LN	Average lifetime of leaves	[d]
U	Number of leaves = u(max)	[#.plant^{-1}]
U_{new}	Leaf units that appeared since t − δ	[#.plant^{-1}]
$U_{removed}$	Leaf units that died since t − δ	[#.plant^{-1}]
LU	Average lifetime of leaf units	[d]
u(i)	Number of leaf units on leaf $\leq$i[1]	[#.plant^{-1}]
u′(i)	Standardized u(i) = u(i)/u(max)	[-]

[1] Leaves counted from top to bottom

(Figure 4). Sporulating lesions were often found on old, dying leaves or leaf tips of long leaves when these were lying on wet soil or in standing water. The sporulating lesions were light-green and often short-lived. White lesions (Figure 5) did not sporulate, unless a water-logged zone developed around the white lesion, as occurs in a humid environment.

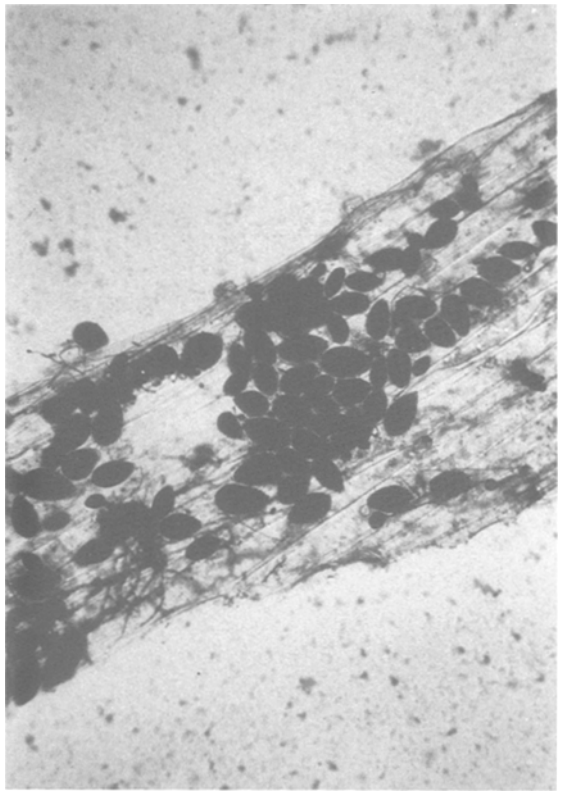


Figure 4. Sporangia of *P. porri* on the surface of a wet leek leaf, stained with cotton blue.

Table 3. Total number of leaves (N) per plant at the end of different time intervals, new leaves (N_{new}) and removed leaves ($N_{removed}$) for each interval, and the average lifetimes of leaves (LN) for full-grown plants

Interval		N	N_{new}	$N_{removed}$	LN
weeks	days				
1992 (n=120, 5 cvs)					
41	–	9.9			
41–46	35	9.9	1.3	1.3	
46–50	30	9.5	0.8	1.2	
50–59	63	9.1	1.9	2.3	
41–59	128	9.4	4.0	4.8	303
1993 (n=200, 8 cvs)					
31	–	5.1			
31–36	34	8.0	4.5	1.6	
36–42	39	8.0	3.7	3.7	
42–47	35	8.5	1.2	0.7	
36–47	74	8.2	4.9	4.4	124

Figure 5. Naturally infected leek plant, with a white lesion of *P. porri* at a characteristic position, ca. 20 cm above the leaf axils.

Table 4. Total, new and removed leaf units (U) and diseased leaf units (X) in week 37–47, 1993 (n=45), and the average lifetime of leaf units (LU) and diseased leaf units (LX) before and after week 40

Interval		Total	New	Removed	Lifetime
weeks	days	(#)	(#)	(#)	(days)
Leaf units		U	U_{new}	$U_{removed}$	LU
37	–	45			
37–40	22	47.5	12.8	10.3	82
40–47	48	46.2	13.1	14.4	169
Diseased leaf units		X	X_{new}	$U_{removed}$	LX
37	–	1.1			
37–40	22	3.3	4.1	1.9	38
40–47	48	6.0	15.8	13.1	21

Turn-over of leaves and lesions. In 1992 and 1993, plants grew from 5 leaves in July to 8–10 leaves in September (Table 3). In 1992, the full-grown plants had more leaves than in the same month of 1993, probably because plant growth was retarded stronger by the earlier onset of the epidemic in 1993. From September onwards, the number of leaves decreased slowly, in spite of some fresh growth.

The average lifetime of leaves LN was 303 d in October 1992–February 1993, and 124 d in September–November 1993. The average lifetime of diseased leaves LD was 32 d in September–November 1993 on 600 other plants (data not shown). The average lifetime of leaf units LU was 169 d in September–November 1993, whereas the average lifetime of diseased leaf units LX was 21 d, on 45 plants (Table 4).

Disease profiles over leaves. (Figure 6) In week 40 of 1992, the curves were very close to the line y = x, indicating that lesions were randomly distributed over leaves [Daamen, 1989]. The deviation from this line in week 44 of 1992 indicates that the youngest leaves had relatively few lesions.

Disease profiles over distances along the leaf. (Figure 7) At 10–20 cm above the soil surface, measured along the leaf, the lesion density was relatively high, especially during an infection wave. This indicates that many infections start in the water basin that is usually present near the leaf axils. Lesions will appear at some distance above this water basin (Figure 4) because of leaf growth during the incubation period.

Relatively high disease frequencies were also found at leaf tips of leaves longer than ca. 75 cm, indicating that infections may result from direct contact between infested soil and leaf tips.

Causes of lesion death. The causes of lesion death were observed for 1379 newly diseased leaves on 600 plants in September–November 1993. At the end of the five week period 569 of these newly diseased leaves either disappeared totally (284 leaves) or just lost the *P. porri* lesion (285 leaves), either through partial leaf death (mainly through loss of necrotic leaf tips), or through invasion by other fungi, (mainly *Pleospora herbarum* (Fries) Rabenhorst), or through desiccation of the lesions.

Discussion

Inoculum sources and effectiveness. Oospores are supposedly the main source of initial infection in autumn, as they are probably the only fungal structures that can survive in leek-free soil for some months.

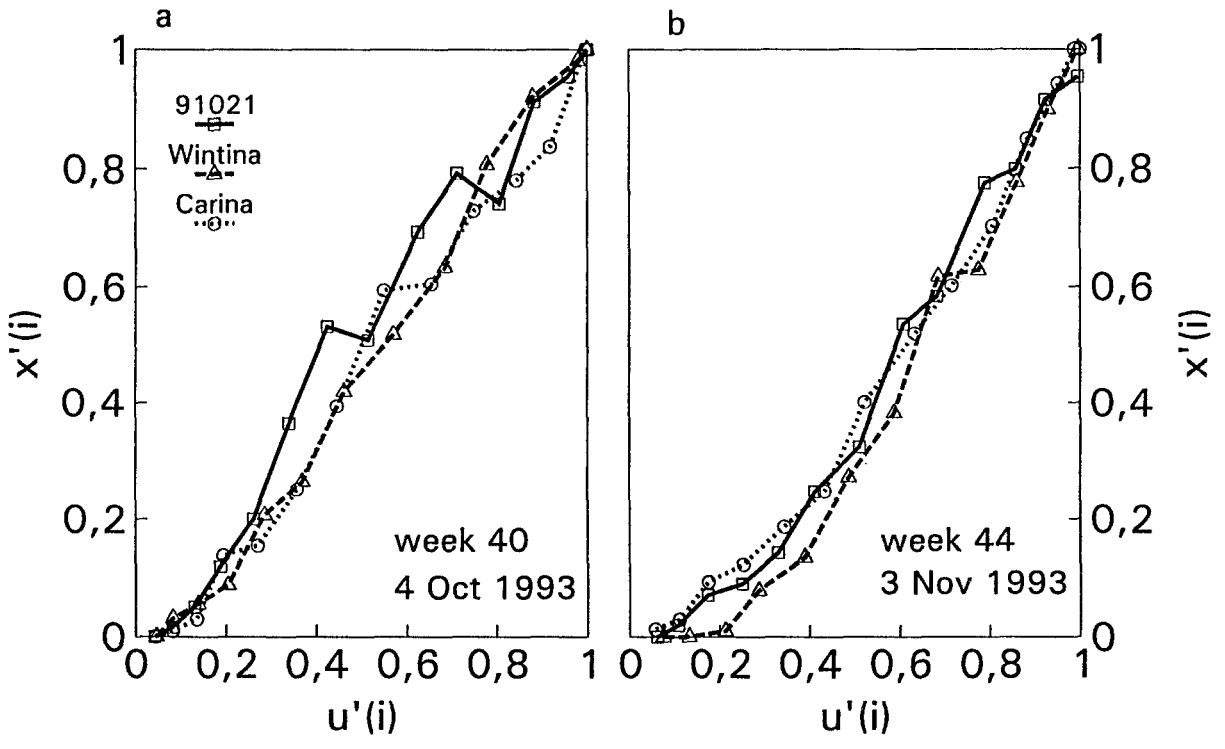


Figure 6. Disease profiles over leaves in week 40 (a) and 44 (b), 1993. The standardized cumulative number of diseased leaves x'(i) is plotted against the standardized cumulative number of leaves u(i) from i = 1 (top leaf) to i = max (bottom leaf).

Oospores are formed abundantly in some, but not in all infected leaves, and are always found in pure cultures on nutrient-rich media. The requirements for oospore production under field conditions are still unknown. As long as oospores are embedded in host tissue they cannot cause new infections, so it is unlikely that oospores play a major role for the build-up of secondary inoculum within one cropping period.

Sporangia on the surface of infected leaves may outnumber the perennated oospores and become the most important infection agents as soon as the first light-green symptoms appear in wet weather. The dominant role of foliar inoculum for intensification of disease was demonstrated in 1994, when the delayed infection in the plot without soil-infestation probably stemmed from foliar inoculum generated in the other plots at >2 m distance.

The observed absence of sporangia on dry leaves does not preclude a dominant role for these spores, as prolonged dry conditions were scarce. Nevertheless, we suppose that in exceptional cases lack of sporulation may limit disease progress and that then oospores may be needed to restart an epidemic.

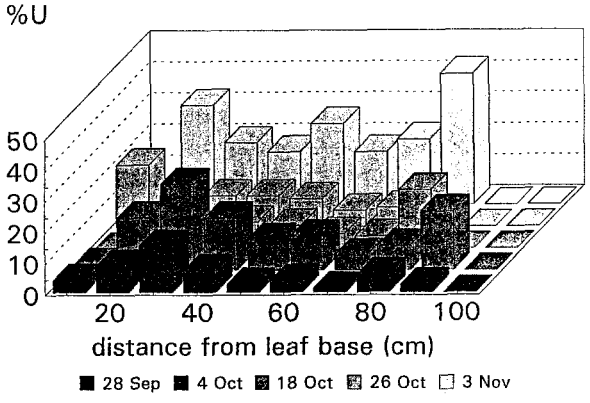


Figure 7. Disease profiles over leaf distances in weeks 39–44, 1993. The lesion density, expressed as percentage leaf units with disease, is given as a function of distance along the leaf, measured from the soil surface.

Sporulation may be poorly related to the number of diseased leaves, since many diseased leaves may never produce spores, whereas the leaves with the most abundant sporulation may easily pass unnoticed. The water status of leaf debris and soil may prove to be better estimators of sporulation than leaf wetness or air humidity

[Weste, 1983]. Unfortunately, direct measurement of soil-borne inoculum of *P. porri* is difficult, as standard selective media, containing the *Pythium* inhibitor hymexazol, inhibited growth of *P. porri* strongly [Ho, 1987].

High lesion densities of leaf units at 10–20 cm above the soil surface measured along the leaf and of leaf tips indicate that most infections depend on free water, which is almost continuously present in the water basin at the leaf axils or in puddles on the soil surface. Thus, after heavy rainfall the conditions for sporulation and infection will be good. This indirect effect of rainfall may improve a correlation between disease and rain caused by primarily by rain splash.

Shape of the epidemic curves. The epidemic curves obtained in our experiments typically showed a sudden, strong and spatially homogeneous ('inundative') onset of disease and a further epidemic development characterised by short periods of explosive disease increase, alternated by periods of marked disease decrease 3–5 weeks after an infection wave. Because of the inundative onset, the epidemic skipped the exponential phase which is normally expected in a polycyclic epidemic [Zadoks and Schein, 1979] almost totally. Only in 1992 there appears to be some evidence of inoculum build-up, as the rain rates causing an infection wave in weeks 31, 39 and 45 were ca. 8, 5 and 4 $mm.d^{-1}$, respectively, indicating that later in the season less mm rain is needed per new infection than earlier in the season.

The infection waves are supposedly associated with earlier rain events. Our analysis of the rain data, using a degree-day model to determine a putative infection period corresponding to an observed disease increase, confirms this hypothesis, but cannot give information about the relative importance of possible causes of this correlation, which include spore discharge and dissemination by rain splash, and better conditions for sporulation and infection.

Our analysis allows the identification of factors that disturb the relation between rainfall and infection. We conclude from Figure 3 that the correlation between rainfall and disease is relatively high at the onset of an epidemic and relatively low later in the season. Apparently, rainfall is necessary for initial infection, but less important for subsequent auto-infection.

Correlation between rain and disease was also poor 3–6 weeks after infection waves e.g. in week 40, 1993, when a high rainfall rate produced a rather small disease increase. Considering that shortly after infection waves most lesions are of the same age, and that the average lifetime of lesions was 21–38 d, this decrease can be understood. In other words, lack of correlation between rain and disease may be due to a wave of lesion death 3–6 weeks after a rain event. Thus, leaf and lesion dynamics should be studied to get a better insight in the epidemic.

Although our study of leaf and lesion dynamics was limited, it highlighted the fact that the disappearance of diseased leaves was not only caused by leaf death but also by several processes causing lesion death or disappearance (leaf tip necrosis, secondary infection, desiccation). As these processes are all related in their own way to weather and plant factors, prediction of lesion death seems to be difficult.

In their terminal phase, epidemics of *P. porri* never approached the theoretical maximum of 8–9 diseased leaves, in spite of the inundative onset. It is not clear what factor determines the apparent maximum number of diseased leaves. Possibly, the epidemic just slows down at lower temperatures. This retardation may explain the slow but steady increase from 1–4 diseased leaves in weeks 43–65 after 1-1-1992, and is also illustrated by the fact that resistance appears to have a constant effect during this period. The maximum disease level of ca. 6 diseased leaves in the exceptionally wet winters of 1993 and 1994 cannot be explained in this way. The difference with the theoretical maximum may be explained by the relatively small size and protected position of the youngest leaves. In spring the disease level declines, probably because plants start to grow faster under the influence of higher temperatures.

Disease control. Control of *P. porri* by preventive spraying with protectant chemicals is not economically justified, because of the unpredictable onset of epidemics, according to Van Bakel [1964]. He advises to postpone the first spray treatment until the appearance of the first symptoms. In this way the costs of spraying may be minimized, but control of *P. porri* will be insufficient when the onset of disease is explosive, as in 1992. Unnecessary treatments may be applied when white leaf tips are caused by drought stress or by genetical disorders which are mistaken for symptoms of *P. porri*. Therefore, extension workers often recommend to spray only when rain is expected in the next week [W. Alofs, pers. comm.], except when rain is expected within 24 h and rain would wash the fungicide from the leaf surface. We can add tentatively that a forecasted 20 mm rain in one day may justify a preventive spray as long as symptoms of disease are

absent in an infested field. After observation of the first few symptoms 20 mm rain in 2–3 days may be enough to initiate an infection wave. However, local showers are hard to predict, even on the short term, so a spraying-scheme based on weather forecasts will never be reliable. Moreover, the effect of chemical control is, in general, limited when the onset of an epidemic is explosive. Other control methods, based on prevention of infection through mulching [Alofs and Pijnenburg, 1988] or host plant resistance [Smilde et al., 1995] are therefore needed.

Acknowledgements

Thanks are due to Prof. dr. J.C. Zadoks, dr. W.A.J. de Milliano, dr. H.J Schouten, ir P.D. de Jong and an anonymous referee for their useful comments. The technical assistance of ir A. Arts is acknowledged. This research was supported by the Technology Foundation (STW), The Netherlands.

References

Alofs W (1986) Het bestrijden van bladvlekken en roest in prei. Weekblad Groenten en Fruit, 19 september: 63

Alofs W and Pijnenburg H (1988) Bodembedekking voorkomt papiervlekken. Weekblad Groenten en Fruit, 16 december: 58

Centraal Bureau voor de Statistiek (1975–1994) Landbouwtellingen. Staatsuitgeverij,'s Gravenhage

Daamen RA (1989) Assessment of the profile of powdery mildew and its damage function at low disease intensities in field experiments with winter wheat. Netherlands Journal of Plant Pathology 95: 85–105

De Kraker J and Bosch H (eds) (1993) Teelt van prei. PAGV and IKC, Lelystad, 112 pp

Fitt BDL, McCartney HA and Walklate PJ (1989) The role of rain in dispersal of pathogen inoculum. Annual Review of Phytopathology 27: 241–270

Foister CE (1931) The white tip disease of leeks and its causal fungus, *Phytophthora porri* n.sp., Transactions of the Botanical Society of Edinburgh 30: 257–281

Foister CE (1961) The economic plant diseases of Scotland. A survey and check list covering the years 1924–1957. Technical Bulletin of the Department of Agriculture and Fisheries for Scotland 1: 55

Grill D (1985) Les maladies et ravageurs du poireau. Phytoma 371: 39–41

Ho HH (1987). Effect of hymexazol on growth and reproduction of low temperature *Phytophthora* species. Mycopathologia 98: 17–20

Legge BJ (1951) A *Phytophthora* crown rot of *Campanula*. Transactions of the British Mycological Society 34: 293–303

Madden LV, Wilson LL, Yang X and Ellis MA (1992) Splash dispersal of *Colletotrichum acutatum* and *Phytophthora cactorum* by short-duration simulated rains. Plant Pathology 41: 427–436

Ogilvie L and Walton CL (1941) Diseases and pests of onions and leek. Worcester City Council Agricultural Quarterly Chronicle 9: 57, 59, 61, 63, 65

Smilde WD, Van Nes M and Reinink K (1995) Resistance to *Phytophthora porri* in leek and some of its wild relatives. Euphytica 83: 131–138

Smilde WD, Van Nes M and Frinking HD ( ) Effects of temperature on *Phytophthora porri in vitro* and *in planta*. European Journal of Phytopathology, submitted

Taylor JC (1965) White tip of leeks (*Phytophthora porri* Foister. Plant Pathology 14: 189

Thorold CA (1975) Black pod disease. In: Gregory PH (ed) Diseases of Cocoa (pp. 32–40) Clarendon Press, Oxford, 423 pp

Van Bakel JMM (1964) Papiervlekkenziekte in prei (*Phytophthora porri* F.). Mededelingen Directoraat Tuinbouw 27: 198–200

Van Hoof HA (1959) Oorzaak en bestrijding van papiervlekkenziekte bij prei. Tijdschrift voor Plantenziekten 65: 37–43

Weste G (1983) Population dynamics and survival of *Phytophthora*. In: Erwin DC, Bartnicki-Garcia S and Tsao PH (eds) *Phytophthora*. Its Biology, Taxonomy, Ecology, and Pathology. APS Press, St. Paul, Minnesota, pp. 237–257

Yokoyama S (1976) Studies on the leaf blight of onion caused by *Phytophthora porri* Foister. Bulletin of the Fukuoka Agricultural Experiment Station 22: 55 pp

Zadoks JC and Schein RD (1979) Epidemiology and Plant Disease Management. Oxford University Press, 427 pp