

Development of natural late blight epidemics in pure and mixed plots of potato cultivars with different levels of partial resistance

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Late blight, caused by *Phytophthora infestans*, is the most severe disease of potato worldwide. Controlling late blight epidemics is difficult, and resistance of host cultivars is either not effective enough, or too easily overcome by the pathogen to be used alone. In field trials conducted for 3 years under natural epidemics, late blight severity was significantly lower in a susceptible cultivar growing in rows alternating with partially resistant cultivars (mixtures) than in unmixed plots of the susceptible cultivar alone. Partially resistant cultivars behaved similarly in unmixed and mixed plots. Mixtures of cultivars reduced disease progress rates and sometimes delayed disease onset over unmixed plots, but did so significantly only for the slowest epidemic. This suggests that reduction of area under the disease progress curve (AUDPC) in mixtures resulted from the cumulative action of minor effects. Disease distribution was focal in all plots at all dates, as shown by Morisita's index values significantly exceeding 1. Significant yield increases for the susceptible cultivar, and occasionally for the partially resistant ones, were observed in mixed-cultivar plots compared with single-cultivar plots. These results show that cultivar mixtures can significantly reduce natural, polycyclic epidemics in broadleaved plants attacked by pathogens causing rapidly expanding lesions.

Keywords: cultivar mixture, diversification strategy; Morisita's index, *Phytophthora infestans*, potato late blight, resistance gene management

Introduction

Modern agroecosystems are dominated by large patches of genetically homogeneous crop genotypes. This pattern of plant distribution favours the rapid development of plant diseases, which pose a major constraint on food production. Examples of devastating epidemics in genetically homogeneous crops abound in modern history, from the late blight epidemics that ravaged Irish potato crops in the 1840s and led to the Great Irish Famine (Bourke, 1964) to the destruction of Texas male sterile cytoplasm genotypes by *Cochliobolus heterostrophus* in maize crops in the 1970s in the USA (Marshall, 1977). Conversely, natural ecosystems are usually less prone to rapid and severe epidemics, because host genotypes are more diverse than in agroecosystems and are distributed in small patches (Jarosz & Burdon, 1991; Antonovics *et al.*, 1994).

Spatial diversification of host resistance therefore appears as a major technique to achieve successful and durable management of crop pathogens by genetic means. It can operate both between fields, e.g. cultivar rotation and mosaic cropping (Wolfe, 1984); and within fields, e.g. alternate strips or rows, mixed cropping of host cultivars or isogenic lines (Browning & Frey, 1969; Wolfe & Barrett, 1980; Wolfe, 1985; Zhu *et al.*, 2000). Three factors have been known to account for disease reduction and subsequent yield benefit in heterogeneous crops: (i) the dilution of inoculum due to the presence of resistant plants (lack or reduction of inoculum production on resistant plants, and loss of inoculum infectious to susceptible hosts by deposition of some of the spores produced on resistant plants); (ii) the physical barrier constituted by resistant plants, limiting inoculum deposition on susceptible components of the mixture; and (iii) induction of defence reactions in the host through challenge by avirulent races of the pathogen (Lannou *et al.*, 1994a; Lannou *et al.*, 1994b; Lannou *et al.*, 1995; Wolfe, 1985; Garrett & Mundt, 1999; Finckh *et al.*, 2000). The operation of these mechanisms implies that cultivar mixtures (and multilines) will work best (i) when alloinfection is high,

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i.e. against pathogens dispersed on a shallow gradient, producing nonexpanding lesions and attacking narrow-leaved plants (Lannou *et al.*, 1994a; Lannou *et al.*, 1994b); (ii) when combining susceptible with totally resistant plants, on which no inoculum is produced, rather than with partially resistant plants; and (iii) against polycyclic rather than monocyclic epidemics. These conditions are often met in airborne, wind-dispersed pathogens such as the rust and powdery mildew pathogens (e.g. Akanda & Mundt, 1996; Browning & Frey, 1969; Mundt *et al.*, 1994; Ntahimpera *et al.*, 1996; Wolfe & Barrett, 1980). However, mixtures or intercropping have also proved useful in controlling airborne or soilborne diseases (Autrique & Potts, 1987; Saur & Mille, 1997; Finckh *et al.*, 2000) that do not meet all of these criteria.

Cultivar mixtures have been primarily considered as a means of preserving resistance, either by enhancing the durability of race-specific resistance genes (Wolfe, 1984; Wolfe, 1985) or by providing a dynamic way to manage genetic resources (Allard, 1990; Reinhold *et al.*, 1990; Balfourier *et al.*, 1994; Le Boulc'h *et al.*, 1994). Enhancing the durability of disease resistance is also a problem in pathosystems which do not comply with the theoretical criteria for success of genotype mixtures, such as those involving broadleaved plants with total or partial resistance. One such example is potato late blight, caused by the oomycete *Phytophthora infestans*. In this system, both total, race-specific and partial, supposedly race-nonspecific resistances are available, although their use is currently unsatisfactory. Race-specific resistance has proved too short-lived to be of any durable value when used alone, while levels of partial resistance are currently too low to be effective enough in current agricultural systems (Wastie, 1991). Furthermore, the highest levels of partial resistance are found in genotypes with undesirable agronomic traits, such as lateness (van der Plank, 1957; Toxopeus, 1958), in combinations that have proved difficult to break in breeding programmes. Despite these shortcomings, genetic resistance has the potential to allow growers to achieve substantial reductions of the large number of fungicide applications currently used to manage late blight (Fry, 1977), and therefore constitutes the cornerstone of integrated control strategies.

Both circumstantial evidence, such as the observation of neighbouring effects between resistant and susceptible genotypes in experimental breeder's plots (Connolly *et al.*, 1995) or of interplot interference in field trials (James *et al.*, 1973; Paysour & Fry, 1983), and direct experimentation (Ntahimpera *et al.*, 1996; Garrett & Mundt, 2000; Garrett *et al.*, 2001) suggest that cultivar mixtures might limit the development of pathogens inducing large, expanding lesions on broadleaved plants, as in the case of *P. infestans* on potato. Experiments in the USA (Garrett & Mundt, 2000) and Ecuador (Garrett *et al.*, 2001) showed that mixtures of potato cultivars with diverse levels of resistance decreased blight severity in inoculated plots away from outside sources of inoculum. Both studies dealt with a global assessment of the effect of mixtures by comparing the mean observed and expected disease

progress in the mixed plots. Here, it is shown that mixtures (produced by planting different patterns of adjacent rows of three different cultivars with different partial resistance to *P. infestans*) leads to a reduction of the epidemic on the susceptible component of the mixture, and that host diversity effects are larger on cumulative measures of epidemics (such as area under the disease progress curve, AUDPC) than on individual epidemic parameters (date of appearance of first symptoms, daily progress rate).

Materials and methods

Experimental design

Three potato cultivars were used: Bintje (B), susceptible to foliage blight; Claustar (Cl), moderately susceptible; and Charlotte (Ch), moderately resistant according to the French National List of Potato Cultivars (Anonymous, 1998). None of these cultivars is known to possess race-specific resistance genes to *P. infestans*, although examination of the pedigrees indicates that cv. Charlotte might carry the resistance genes R1 and/or R3 and Claustar might carry R1 and/or R4. All three genes, however, are overcome by at least 90% of *P. infestans* isolates recently present in France (Andrivon, 1994; Lebreton *et al.*, 1998). As late maturity is known to be correlated with blight resistance (van der Plank, 1957; Toxopeus, 1958), the three cultivars were chosen within the same maturity group (early maincrop) in order to minimize lateness as a factor.

Field plots were established on a silt loam soil at the INRA experimental facilities in Le Rheu (48°01' N, 1°43' W) from 1993 to 1998, using Elite seed tubers. In all plots, rows were 70 cm apart and plants within a row were 35 cm apart. Plots were separated from one another by 2 m wide, bare paths, and the trials were at least 1 km away from the nearest potato field. As natural late blight infections only developed in 1993, 1997 and 1998, the experiments carried out from 1994 to 1996 are not considered.

Plots were monitored for blight outbreaks at weekly intervals following plant emergence, and disease assessments started when the first symptoms were observed. Subsequent assessments were made at weekly (1993) or biweekly (1997, 1998) intervals, depending on the conduciveness of the weather to blight progression, and continued for 2–5 weeks until the first plants died. In all plots and years, disease severity was assessed visually as the proportion of foliage diseased on each plant, according to the 1–9 illustrated scale of Cruickshank *et al.* (1982).

1993 trial

Five plots (7.7 × 7.0 m), each consisting of 12 rows of 20 tubers each, were planted side by side on 13 May. Three of these plots consisted of 10 adjacent rows of either B, Ch or Cl, bordered on each side by a row of B. One of the remaining two plots was a three-way mixture, with one

row of each of the three cultivars alternated in a pattern repeated four times (B-Ch-Cl-B-Ch-Cl-B-Ch-Cl-B-Ch-Cl); the other plot was a two-way mixture, with one row of B and two rows of Ch alternated in a pattern replicated four times (B-Ch-Ch-B-Ch-Ch-B-Ch-Ch-B-Ch-Ch). The order of the plots in the field was pure B/three-way mixture/pure Ch/two-way mixture/pure Cl. Weeds were controlled with Linural 50 Flowable (Tradi-Agri, France; 450 g L⁻¹ linuron, 2.2 L ha⁻¹) applied on 24 May. Ridges were finally established on 18 June, and the first symptoms of late blight were observed on that date. Vines were killed on 5 August with 1 kg ha⁻¹ diquat (Reglone 2, Sopra, France). The plots were harvested on 23 September, each row being harvested separately. Tubers were counted, graded and weighed.

1997 and 1998 trials

Four plots (7.70 × 4.90 m), each consisting of 12 rows of 15 tubers, were planted on 23 April 1997 and 11 May 1998. Two of these plots were made of 10 rows of either B or Ch, bordered on each side by a row of B. The remaining two plots consisted of four repetitions of a basic pattern made of either one row of B and two rows of Ch (B-Ch-Ch-B-Ch-Ch-B-Ch-Ch-B-Ch-Ch), or two rows of B and one row of Ch (B-B-Ch-B-B-Ch-B-B-Ch-B-B-Ch). In 1997, weeds were controlled with Sencoral 35 WP (Bayer, France SA; 35% metribuzine, 2 kg ha⁻¹) applied on 14 May. Persistent rain between planting and crop emergence prevented herbicide being sprayed in 1998, so plots were weeded manually in June. Ridging was completed on 3 June 1997 and 8 June 1998. Colorado potato beetles were controlled with Mavrik Flo (Parthena, France; 280 g L⁻¹ tau-fluvalinate; 0.2 L ha⁻¹) applied on 20 June 1997, and 22 June and 27 July 1998. The first disease scorings were made on 1 July 1997 and on 26 June 1998. Haulms were killed on 8 August 1997 and on 25 August 1998 with 1 kg ha⁻¹ diquat (Reglone 2). The plots were harvested on 1 September 1997 and on 17 September 1998, each row being harvested separately. In both years, tubers were graded and weighed.

Statistical analyses

Disease progress in time

Disease severity scores (1–9) on each plant were converted into percentages of foliage tissue blighted using the median severity of each class as the transformation factor. Severity data for each row in each plot were calculated from the resulting percentages, averaged over rows of the same cultivar in each plot, and plotted over time. Incidence curves (plots of the proportion of plants with symptoms over time) were not used, as the fast progression of the epidemics did not permit more than 1–2 points with incidence frequencies different from either 0 or 100% to be obtained in most of the plots, making fitting of any distribution meaningless.

Direct comparisons of disease progress curves were performed. The goodness of fit of the data to the exponential,

logistic and Gompertz models were assessed through linear regression against time of disease severity (s) transformed as $\ln(s)$, $\ln[s/(100 - s)]$ and $-\ln(-\ln s)$, respectively. These three models were chosen because they fit numerous plant disease progress curves (Campbell & Madden, 1990). The best fitting model was selected based on the ANOVA parameters of the regressions, and epidemic parameters were compared between pairs of curves by calculating confidence intervals of the differences (Campbell & Madden, 1990). Furthermore, AUDPCs were calculated for each cultivar, year and plot using the trapezoidal integration method (Campbell & Madden, 1990), and analysed by ANOVA with years as replicates.

Spatial aggregation of disease

Mixtures are known to work best when disease is focal. To check for differences in the spatial structure of the epidemics in pure and mixed plots, Morisita's dispersion index (Morisita, 1959) was calculated for each plot (each plant being considered a quadrat) and at each date as:

$$I_{\delta} = n \{[(\sum_i x_i^2) - \sum_i(x_i)] / [(\sum_i x_i)^2 - \sum_i(x_i)]\}$$

where n is the number of plants and x_i is the disease severity on plant i ($i = 1 \dots n$) (Thal & Campbell, 1986). Values of I_{δ} reflect three different types of distribution: uniform (regular) distributions are characterized by $I_{\delta} < 1$; random distributions by $I_{\delta} = 1$; and aggregated (contagious) distributions by $I_{\delta} > 1$ (Morisita, 1959; Shuh *et al.*, 1986). Calculated I_{δ} values were compared with 1 as described by Campbell & Madden (1990).

Yield

Yield data (weight of tubers per row) were averaged for each cultivar, year and plot, and analysed by ANOVA separately for each cultivar, with years as replicates. No comparison of total yield per plot was attempted, because of the large differences in yield potential between cultivars. All ANOVA calculations were performed with the GLM module of the SAS statistical software (ver. 6.12; SAS Institute, Cary, NC, USA).

Results

Progress of epidemics in time

The Gompertz model generally fitted the disease progress curves best ($R^2 = 0.88-0.99$; $P < 0.05$ in all but one plot); the logistic and exponential models consistently gave lower R^2 and higher P values over the set of curves than the Gompertz model, except for epidemics on Charlotte in 1998 (data not shown) which all models fitted poorly. Therefore comparisons were subsequently made using the parameters of the Gompertz model.

Disease progressed significantly faster in 1997 and in 1998 than in 1993 (Table 1). There were no significant differences in the time of appearance of the first outbreaks between cultivars or plots in a given year (Fig. 1; Tables 2 and 3), confirming that the cultivars used possessed no

Table 1 Differences between epidemic parameters for late blight development in pure plots of potato cultivars Bintje (B, susceptible) and Charlotte (Ch, partially resistant) under natural infection

Treatments compared	Year	Daily progress rate ^a	df	Date of first outbreak ^a	df
Epidemic pressure in different years					
B pure	1993–97	-0.088**	5	nt ^b	
	1993–98	-0.115**	7	nt ^b	
	1997–98	-0.026	6	nt ^b	
Ch pure	1993–97	-0.095**	5	nt ^b	
	1993–98	-0.153**	7	nt ^b	
	1997–98	-0.058	6	nt ^b	
Effect of partial resistance on epidemic progress					
B pure–Ch pure	1993	0.028*	6	0.723	6
	1997	0.021	4	0.704	4
	1998	0.010	8	1.195	8

^aDaily progress rate and date of first outbreak determined as parameters of the linear regression over time of severity data transformed with the Gompertz model.

^bStatistical comparisons of dates of first outbreaks between years are not informative to compare these epidemics as they reflect differences in climatic conditions more than cultivar effects.

**, * indicate differences significant at $P = 0.05$ and $P = 0.10$, respectively.

efficient race-specific resistance towards local populations of *P. infestans*. As expected, AUDPC showed that disease was significantly more severe in pure Bintje (B) than in pure Charlotte (Ch) or Claustar (Cl) stands, but all three cultivars behaved similarly in alternate plantings (Fig. 1). With a few exceptions – mainly involving the 1993 epidemics – the comparison of epidemic parameters showed no significant differences between treatments (cultivars and planting patterns; Tables 2 and 3). However, a one-sided Fisher sign test on the differences between pure and alternated plots showed a consistent reduction in daily disease progress rate by mixtures ($P = 0.0002$). The performance of Charlotte was not significantly different between pure and alternated plots (Table 2). The proportion of susceptible plants in the mixed plots did not significantly affect the performance of Bintje or Charlotte (Fig. 2; Table 3).

Spatial aggregation of disease

Although declining over time, values of Morisita's index were significantly >1 ($P < 0.01$) in all plots and at all dates (Table 4), indicating an aggregated structure of epidemics in both pure and mixed plots.

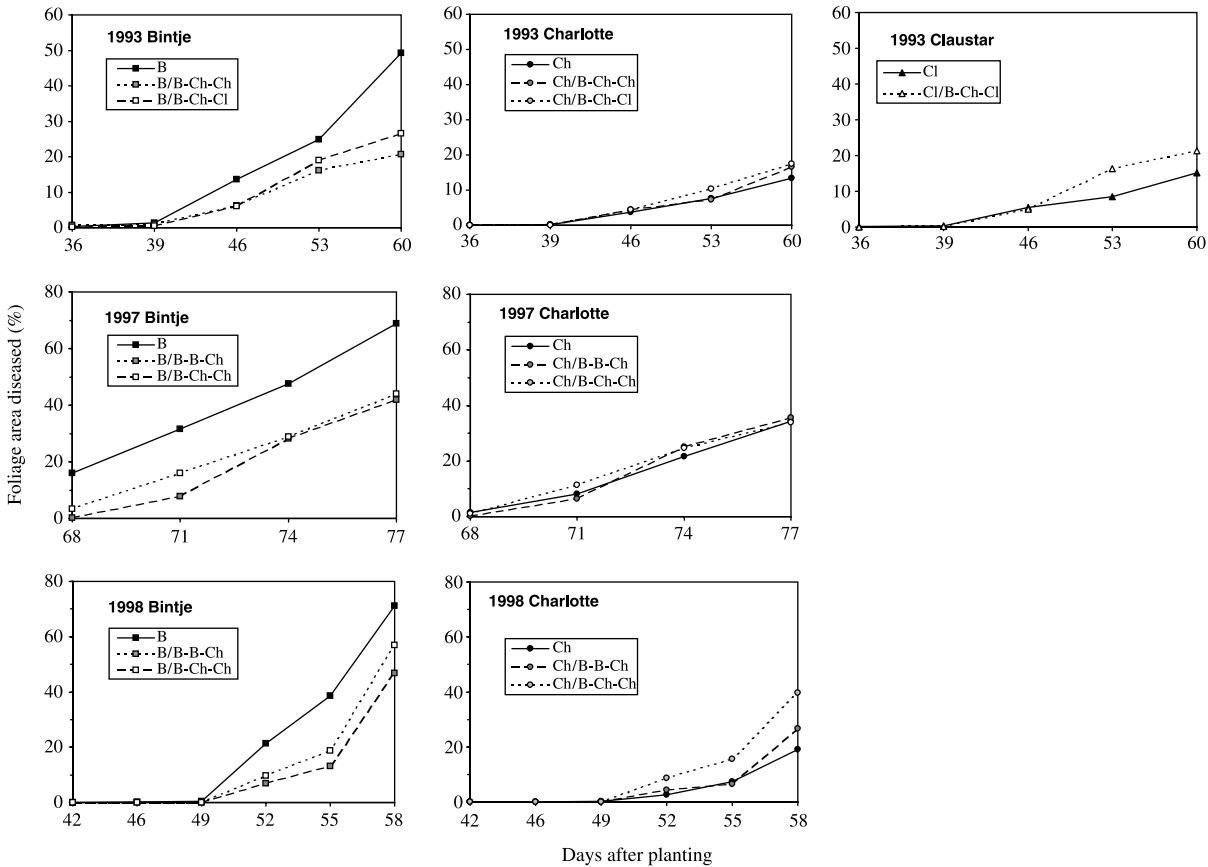


Figure 1 Progress of natural late blight epidemics in pure or mixed plots of potato cultivars Bintje (B, susceptible); Charlotte (Ch); and Claustar (Cl, both partially resistant). Disease is expressed as the mean percentage of foliage area blighted, calculated over all plants from each cultivar in each plot.

Treatments compared ^a	Year	Daily progress rate ^b	df	Date of first outbreak ^b	df
Bintje					
B pure//B in B-Ch-CI	1993	0.013	6	0.290	6
B pure//B in B-Ch-Ch	1993	0.033**	6	1.263*	6
	1997	0.018	4	0.710	4
	1998	0.065	8	-3.965	8
B pure//B in B-B-Ch	1997	0.047	4	-4.258	4
	1998	0.044	8	-3.024	8
Charlotte					
Ch pure//Ch in B-Ch-CI	1993	0.007	6	-0.280	6
Ch pure//Ch in B-Ch-Ch	1993	0.041	6	-0.162	6
	1997	0.008	4	-0.527	4
	1998	0.023	8	1.264	8
Ch pure//Ch in B-B-Ch	1997	0.051	4	-3.753	4
	1998	0.065	8	3.254	8

^aPlots: B, 12 rows of Bintje; Ch, 12 rows of Charlotte; B-Ch-Ch = one row of Bintje and two rows of Charlotte, repeated four times; B-B-Ch = two rows of Bintje and one row of Charlotte, repeated four times. Rows were 20 tubers each in 1993, 15 tubers each in 1997 and 1998.

^bDaily progress rate and date of first outbreak determined as the parameters of the linear regression over time of severity data transformed with the Gompertz model.

**, * indicate differences significant at $P = 0.05$ and $P = 0.10$, respectively.

Table 2 Comparison of epidemic parameters for late blight development on potato cultivars Bintje (B, susceptible), Charlotte (Ch, partially resistant) and Claustar (Cl, partially resistant) in pure and mixed plots

Treatments compared ^a	Daily progress rate ^b	df	Date of first outbreak ^b	df
1993				
B in B-Ch-Ch//B in B-Ch-CI	0.020**	6	0.973**	6
Ch in B-Ch-Ch//Ch in B-Ch-CI	0.003	6	0.118	6
1997				
B in B-B-Ch//B in B-Ch-Ch	0.065*	4	4.978*	4
Ch in B-B-Ch//Ch in B-Ch-Ch	-0.043	4	3.226	4
1998				
B in B-B-Ch//B in B-Ch-Ch	0.021	8	0.941	8
Ch in B-B-Ch//Ch in B-Ch-Ch	-0.041	8	1.988	8

^aPlots: B, 12 rows of Bintje; Ch, 12 rows of Charlotte; B-Ch-Ch = one row of Bintje and two rows of Charlotte, repeated four times; B-B-Ch = 2 rows of Bintje and one row of Charlotte, repeated four times. Rows were 20 tubers each in 1993, 15 tubers each in 1997 and 1998.

^bDaily progress rate and date of first outbreak determined as the parameters of the linear regression over time of severity data transformed with the Gompertz model.

**, * indicate differences significant at $P = 0.05$ and $P = 0.10$, respectively.

Table 3 Comparison of epidemic parameters for late blight development on potato cultivars Bintje (B, susceptible) and Charlotte (Ch, partially resistant) in different mixtures under natural infection

Yield

In each of the 3 years, the yield per plant of Bintje was higher in mixed than in pure plots, although the difference was not statistically different in 1998 (Fig. 3). No statistically significant yield differences between pure and alternated plots were observed in any of the 3 years for the partially resistant cultivar Charlotte, whereas a slight but significant yield reduction of Claustar in alternated plots was observed in 1993. The 1993 data show that both tuber number per plant and tuber weight per plant of Bintje were significantly higher in mixtures than in pure plots, whereas neither tuber number nor tuber weight of cv. Charlotte was significantly affected by mixing cultivars

(Table 5). The lower total yield of cv. Claustar in mixed plots was due to a reduction in mean tuber weight as the number of tubers per plant was similar in pure and mixed plots; this high number of small tubers explains the significant decrease in number of marketable tubers of cv. Claustar (Table 5).

Discussion

Epidemic reduction in mixtures

The data confirm and complement previous results (Garrett & Mundt, 2000; Garrett *et al.*, 2001), showing that alternate plantings of susceptible and partially resistant

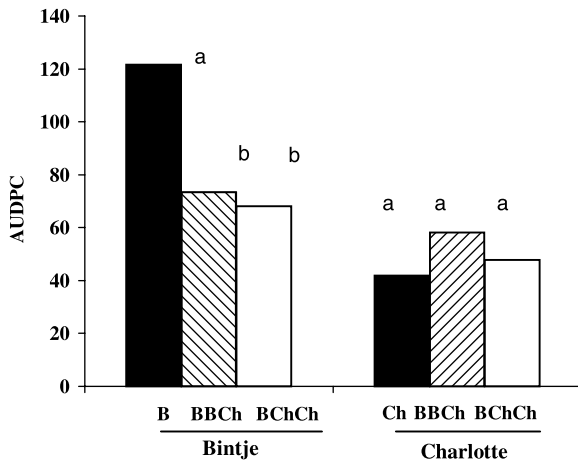


Figure 2 Mean areas under the disease progress curve (AUDPC) of natural late blight epidemics in potato cultivars Bintje (B, susceptible) and Charlotte (Ch, partially resistant) grown in pure or mixed-cultivar plots over three different years. Mixtures included either one row of Bintje and two of Charlotte, repeated four times (plot B-Ch-Ch); or two rows of Bintje and one of Charlotte, repeated four times (plot B-B-Ch). For each cultivar, bars with the same letter are not significantly different (LSD test, $P = 0.05$).

Table 4 Values of Morisita's index calculated from late blight severity data collected at different dates in pure or mixed plots of potato cultivars Bintje (B, susceptible) and Charlotte (Ch, partially resistant), grown at Le Rheu (France) in three different years. All values are significantly >1 (t -test, $P = 0.01$)

Year/date ^a	Plots ^b			
	pure B	pure Ch	B-Ch-Ch	B-B-Ch
1993				
36	10.21	na	39.59	nt
39	8.34	40.84	10.16	nt
46	2.05	1.16	1.12	nt
53	1.43	1.34	1.37	nt
60	1.26	1.31	1.16	nt
1997				
68	47.32	94.27	5.12	21.18
71	38.62	85.88	1.45	1.55
74	37.01	78.94	1.09	1.07
77	37.52	71.91	1.06	1.07
1998				
42	79.56	na	na	na
46	18.46	175.00	177.00	na
49	10.38	36.42	78.22	177.00
52	1.29	4.02	1.48	2.50
55	1.18	1.99	1.34	1.82
58	1.02	1.40	1.05	1.13

^aDates are expressed as days after planting.

^bPlots: pure B, 12 rows of Bintje; pure Ch, 12 rows of Charlotte; B-Ch-Ch = one row of Bintje and two rows of Charlotte, repeated four times; B-B-Ch = two rows of Bintje and one row of Charlotte, repeated four times. Rows were 20 tubers each in 1993, 15 tubers each in 1997 and 1998.

nt, not tested in trial; na, not applicable (all plants disease-free).

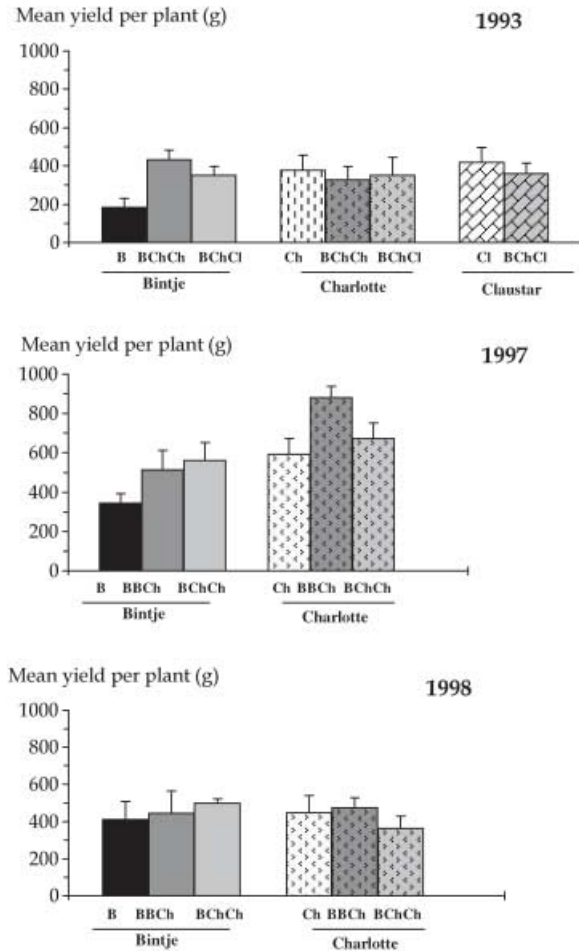


Figure 3 Yield response of potato cultivars Bintje (B, susceptible), Charlotte (Ch, partially resistant) and Claustar (Cl, partially resistant) to *Phytophthora infestans* when grown in pure stands or mixtures.

potato cultivars can limit natural late blight epidemics. Although the field trials did not include internal replicates, the consistency of the results over 3 years of trials strongly suggests that the observed reduction in disease severity over time is not due to yearly random variations. The choice was to work with natural inoculum, rather than with artificially inoculated plots as in previous reports, because the initial distribution of inoculum can markedly affect subsequent epidemic development in heterogeneous plots and hence the performance of mixtures of cultivars relative to disease control. By doing so, it was shown that disease reduction in the mixed plots was almost entirely due to the limitation of disease on the susceptible component of the mixtures, and was the result of cumulative effects over the course of the epidemic leading to a slight reduction of epidemic daily progress rate.

The reduction in disease severity in alternate plantings was translated into large yield benefits for the susceptible cultivar Bintje. The magnitude of the effect of mixtures on AUDPC and yield was unexpected, as the characteristics of the plant (partial resistance, large leaves favouring

Table 5 Yield components of individual plants in three potato cultivars grown in pure or mixed plots at Le Rheu, western France, in 1993

Cultivar/plot ^a	Tuber weight/plant (g)		Tuber number/plant	
	Total	Marketable	Total	Marketable
Bintje (B)				
Pure	133.7 a ^b	44.0 a	6.6 a	0.8 a
B-Ch-Ch	391.8 b	341.6 c	10.9 b	7.4 c
B-Ch-CI	304.7 b	131.5 b	10.1 b	2.5 b
Charlotte (Ch)				
Pure	359.0 a	283.2 a	8.0 a	5.6 a
B-Ch-Ch	297.5 a	243.9 a	6.7 a	4.9 a
B-Ch-CI	299.3 a	226.8 a	7.2 a	4.5 a
Claustar (CI)				
Pure	464.9 a	347.9 a	11.0 a	6.7 a
B-Ch-CI	385.0 b	241.0 b	10.7 a	4.5 b

^aCultivars: B = Bintje; Ch = Charlotte; CI = Claustar. Plots: B-Ch-Ch = one row of Bintje and two rows of Charlotte, repeated four times; B-Ch-CI = one row each of Bintje, Charlotte and Claustar, repeated four times. Rows were 20 tubers each. Mean values are shown over all rows of each cultivar in each plot.

^bMeans followed by the same lower case letter (not superscript) within a column and for a given cultivar are not significantly different (*t*-test, *P* = 0.05; rows of the same cultivar in each plot as replicates).

autoinfection over alloinfection); of the disease (rapidly expanding lesions); and of the experimental design (alternate rows rather than random mixtures) should have limited the efficacy of mechanisms decreasing disease severity in heterogeneous crops (Aylor, 1988; Lannou *et al.*, 1994a; Lannou *et al.*, 1994b; Mundt *et al.*, 1996). It is possible that part of the yield benefit in mixed plots was not a direct consequence of disease reduction, but of positive competition effects between cultivars when mixed (Finckh & Mundt, 1992). It was not possible to test this with the experimental data, because the trials did not include disease-free pure and mixed plots.

Despite their good performance, cultivar mixtures did not provide control compatible with the imperatives of commercial cropping; they might nevertheless prove helpful as a component of blight management in agricultural systems where pesticide inputs are low or absent (such as organic crops), or as a means contributing to reduce pesticide input in conventional production systems. Recent observations and experiments in large plots under natural infection in the UK (M.S. Wolfe, Elm Farm Research Centre, UK, personal communication) and in Denmark (L. Bødker Danish Institute for Agricultural Sciences, Denmark, personal communication) similarly showed some reduction in late blight in random mixtures of two to three potato cultivars compared with pure plots of these genotypes, although limited or no effects of mixtures were observed in Peru- and Ecuador-based experiments (Garrett *et al.*, 2001).

Epidemiological mechanisms

The general effect of cultivar mixtures in the experiments

was to decrease the epidemic spread on the susceptible cultivar within the mixed planting without altering the behaviour of the partially resistant one(s). This result suggests that, at least in the case of potato late blight, the agricultural value of mixtures might lie as much in the added protection of susceptible cultivars – which often appeal to growers because of their higher agronomic characteristics – as in the preservation of vulnerable resistance.

The absence of any significant effect of cultivar mixtures on the disease progress parameters, except for the slowest epidemic (1993), suggests that the operation of these mechanisms was probably limited, and that the disease reduction achieved was the cumulative result over the course of the epidemic of minor reductions in epidemic daily progress rate. The three mechanisms accounting for disease reduction in heterogeneous crops (inoculum dilution; physical barrier effects; induction of defence reactions by avirulent races, Wolfe, 1985) are likely to occur simultaneously. Therefore isolating their effects in the field is difficult. However, the data indicate that there might have been barrier effects in the alternate plots. A visual examination of the maps of disease severity in each plot over time suggests that disease generally progressed mainly along the rows in mixed-cultivar plots (lines of least resistance), as opposed to along the diagonal (corresponding to the main wind direction during the epidemics) in the pure plots (data not shown). Part of the mixture effect might thus be due to differences in the spatiotemporal pattern of disease spread. This is consistent with the hypothesis that lower amounts of outside inoculum generally improve the efficacy of mixtures against late blight (Garrett *et al.*, 2001). A more detailed statistical analysis of spatial disease progress in the different plots is under way to test the effects of crop geometry on the incidence and severity of disease.

Despite the fact that the resistant cultivars were postulated to be race-nonspecific, adaptation of the isolates to one or other component of the mixtures cannot be excluded. Evidence for differential adaptation to partial resistance of *P. infestans* isolates identical for virulence has been reported for both foliar (Caten, 1974) and tuber infections (Bjor & Mulelid, 1991), and has been shown to occur also in other pathosystems (Chin & Wolfe, 1984; Villareal & Lannou, 2000). Furthermore, evidence from quantitative trait loci detection suggests that partial resistance to foliage blight in potato is mediated in part by genetic factors showing specific interactions with pathogen isolates or races (Simko, 2002). Such an adaptation, if occurring in the field over the course of a single epidemic, might restrict isolate movement between cultivars grown in mixtures, and hence increase effects of mixtures of cultivars on disease. This hypothesis is now being tested through typing of isolates collected in pure and mixed plots at various points of the epidemic development for aggressiveness and molecular traits, to determine whether cultivars are able to select differentially for pathogenicity within a single clonal lineage of the pathogen.

References

- Akanda SI, Mundt CC, 1996. Effects of two-component wheat cultivar mixtures on stripe rust severity. *Phytopathology* **86**, 347–53.
- Allard RW, 1990. The genetics of host–pathogen coevolution: implications for genetic resource conservation. *Journal of Heredity* **81**, 1–6.
- Andrion D, 1994. Races of *Phytophthora infestans* in France, 1991–93. *Potato Research* **37**, 279–86.
- Anonymous, 1998. *Catalogue Français des Variétés de Pomme de Terre*. Paris, France: FNPPPT/Editions du Billon.
- Antonovics J, Thrall PH, Jarosz AM, Stratton D, 1994. Ecological genetics of metapopulations: the *Silene–Ustilago* plant pathogen system. In: Real LA, ed. *Ecological Genetics*. Princeton, NJ, USA: Princeton University Press, 146–70.
- Autrique A, Potts MJ, 1987. The influence of mixed cropping on the control of potato bacterial wilt (*Pseudomonas solanacearum*). *Annals of Applied Biology* **111**, 125–33.
- Aylor DE, 1988. Development of bean rust epidemics in a field planted with alternate rows of a resistant and a susceptible snap bean cultivar. *Phytopathology* **78**, 1210–5.
- Balfourier F, Charmet G, Grand-Ravel C, 1994. Conservation of allelic multiplicity and genotypic frequency by pooling wild populations of perennial ryegrass. *Heredity* **73**, 386–96.
- Bjor T, Mulelid K, 1991. Differential resistance to tuber late blight in potato cultivars without R genes. *Potato Research* **34**, 3–8.
- Bourke PMA, 1964. Emergence of potato blight, 1843–1846. *Nature* **203**, 805–8.
- Browning JA, Frey KJ, 1969. Multiline cultivars as a means of disease control. *Annual Review of Phytopathology* **7**, 355–82.
- Campbell CL, Madden LV, 1990. *Introduction to Plant Disease Epidemiology*. New York, USA: John Wiley & Sons.
- Caten CE, 1974. Inter-racial variation in *Phytophthora infestans* and adaptation to field resistance for potato blight. *Annals of Applied Biology* **77**, 259–70.
- Chin KM, Wolfe MS, 1984. Selection on *Erysiphe graminis* in pure and mixed stands of barley. *Plant Pathology* **33**, 535–46.
- Connolly T, McNicol JW, Wastie RL, Stewart HE, 1995. Evaluating between-plant interference in field trials for assessing potato genotypes for resistance to late blight. *Annals of Applied Biology* **127**, 273–82.
- Cruickshank G, Stewart HE, Wastie RL, 1982. An illustrated assessment key for foliage blight of potatoes. *Potato Research* **25**, 213–4.
- Finckh MR, Mundt CC, 1992. Plant competition and disease in genetically diverse wheat populations. *Oecologia* **91**, 82–92.
- Finckh MR, Gacek ES, Goyeau H, Lannou C, Merz U, Mundt CC, Munk L, Nadziak J, Newton AC, de Vallavieille Pope C, Wolfe MS, 2000. Cereal variety and species mixtures in practice, with emphasis on disease resistance. *Agronomie* **20**, 813–37.
- Fry WE, 1977. Integrated control of potato late blight – effects of polygenic resistance and techniques of timing fungicide applications. *Phytopathology* **67**, 415–20.
- Garrett KA, Mundt CC, 1999. Epidemiology in mixed host populations. *Phytopathology* **89**, 984–90.
- Garrett KA, Mundt CC, 2000. Host diversity can reduce potato late blight severity for focal and general patterns of primary inoculum. *Phytopathology* **90**, 1307–12.
- Garrett KA, Nelson RJ, Mundt CC, Chacón G, Jaramillo RE, Forbes GA, 2001. The effects of host diversity and other management components on epidemics of potato late blight in the humid highland tropics. *Phytopathology* **91**, 993–1000.
- James WC, Shih CS, Callbeck LC, Hodgson WA, 1973. Interplot interference in field experiments with late blight of potato (*Phytophthora infestans*). *Phytopathology* **63**, 1269–75.
- Jarosz AM, Burdon JJ, 1991. Host–pathogen interactions in natural populations of *Linum marginale* and *Melampsora lini*. II. Local and regional variation in patterns of resistance and racial structure. *Evolution* **45**, 1618–27.
- Lannou C, de Vallavieille-Pope C, Biass C, Goyeau H, 1994a. The efficacy of mixtures of susceptible and resistant hosts to two wheat rusts of different lesion size: controlled conditions experiments and computerized simulations. *Journal of Phytopathology* **140**, 227–37.
- Lannou C, de Vallavieille-Pope C, Goyeau H, 1994b. Host mixture efficacy in disease control: effect of lesion growth analysed through computer-simulated epidemics. *Plant Pathology* **43**, 651–62.
- Lannou C, de Vallavieille-Pope C, Goyeau H, 1995. Induced resistance in host mixtures and its effect on disease control in computer-simulated epidemics. *Plant Pathology* **44**, 478–89.
- Le Boulc’h V, David JL, Brabant P, de Vallavieille Pope C, 1994. Dynamic conservation of variability: responses of wheat populations to different selective forces including powdery mildew. *Génétique Selection Evolution* **26** (Suppl. 1), 221–40.
- Lebreton L, Laurent C, Andrion D, 1998. Evolution of *Phytophthora infestans* populations in the two most important potato production areas of France during 1992–96. *Plant Pathology* **47**, 427–39.
- Marshall DR, 1977. The advantages and hazards of genetic homogeneity. *Annals of the New York Academy of Sciences* **287**, 1–20.
- Morisita M, 1959. Measuring of the dispersion of individuals and analysis of the distributional patterns. *Memoirs of the Faculty of Sciences, Kyushu University, Series E (Biology)* **2**, 215–34.
- Mundt CC, Hayes PM, Schön CC, 1994. Influence of barley variety mixtures on severity of scald and net blotch and on yield. *Plant Pathology* **43**, 356–61.
- Mundt CC, Brophy LS, Kolar SC, 1996. Effect of genotype unit number and spatial arrangement on severity of yellow rust in wheat cultivar mixtures. *Plant Pathology* **45**, 215–22.
- Ntahimpera N, Dillard HR, Cobb AC, Seem RC, 1996. Anthracnose development in mixtures of resistant and susceptible dry bean cultivars. *Phytopathology* **86**, 668–73.
- Paysour RE, Fry WE, 1983. Interplot interference: a model for planning field experiments with aerially disseminated pathogens. *Phytopathology* **73**, 1014–20.
- van der Plank JE, 1957. A note on three sorts of resistance to late blight. *American Potato Journal* **34**, 72–5.
- Reinhold M, Bjarko ME, Sands DC, Bockelman HE, 1990. Changes in resistance to powdery mildew in a barley composite cross. *Canadian Journal of Botany* **68**, 916–9.
- Saur L, Mille B, 1997. Développement du piétin-verse dû à *Pseudocecospora herpotrichoides* sur des mixtures variétales de blé tendre d’hiver. *Agronomie* **17**, 113–8.

- Shuh W, Frederiksen RA, Jeger MJ, 1986. Analysis of spatial patterns in sorghum downy mildew with Morisita's index of dispersion. *Phytopathology* **76**, 446–50.
- Simko I, 2002. Comparative analysis of quantitative trait loci for foliage resistance to *Phytophthora infestans* in tuber-bearing *Solanum* species. *American Journal of Potato Research* **79**, 125–32.
- Thal WM, Campbell CL, 1986. Spatial pattern analysis of disease severity data for alfalfa leaf spot caused primarily by *Leptosphaerulina briosiana*. *Phytopathology* **76**, 190–4.
- Toxopeus HJ, 1958. Some notes on the relations between field resistance to *Phytophthora infestans* in leaves and tubers and ripening time in *Solanum tuberosum* subsp. *tuberosum*. *Euphytica* **7**, 123–30.
- Villareal LMMA, Lannou C, 2000. Selection for increased spore efficacy by host genetic background in a wheat powdery mildew population. *Phytopathology* **90**, 1300–6.
- Wastie RL, 1991. Breeding for resistance. In: Ingram DS, Williams PH, eds. *Phytophthora infestans, the Cause of Late Blight of Potato*. Advances in Plant Pathology, 7. London, UK: Academic Press, 193–224.
- Wolfe MS, 1984. Trying to understand and control powdery mildew. *Plant Pathology* **33**, 451–66.
- Wolfe MS, 1985. The current status and prospects of multiline cultivars and variety mixtures for disease resistance. *Annual Review of Phytopathology* **23**, 251–73.
- Wolfe MS, Barrett JA, 1980. Can we lead the pathogen astray? *Plant Disease* **64**, 148–55.
- Zhu Y, Chen H, Fan J, Wang Y, Li Y, Chen J, Fan JX, Yang S, Hu L, Leung H, Mew TW, Teng PS, Wang Z, Mundt CC, 2000. Genetic diversity and disease control in rice. *Nature* **406**, 718–22.