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Components of resistance to late blight (*Phytophthora infestans*) in eight South American *Solanum* species

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Abstract

Four components of partial resistance to *Phytophthora infestans* were measured after inoculation in the greenhouse and in the field of *Solanum arnezii x hondelmannii*, *S. berthaultii*, *S. circaeifolium*, *S. leptophyes*, *S. microdontum*, *S. sparsipilum*, *S. sucrense* and *S. vernei*, and four hybrid progenies of *S. microdontum* with *S. tuberosum*. The four components were infection efficiency, lesion growth rate, generation time and sporulation capacity. The results were compared with resistance ratings derived from field experiments, and with observations made on the potato cultivars Bintje, Bildtstar, Libertas and Pimpernel. Genetic variation for all components was found, while the relative importance of the components of partial resistance appeared to vary between the species. In *S. microdontum*, generation time, infection efficiency and lesion growth rate, and in *S. tuberosum* infection efficiency, lesion growth rate and sporulation capacity appeared positively associated, but in other species no such association was found. A strong hypersensitive reaction, the expression of which appeared to depend on environmental conditions, was found in *S. microdontum*. For *S. berthaultii*, infection efficiency appeared to be the main resistance component.

Abbreviations: ADPC = area under the disease progress curve; IE = infection efficiency; LGR = lesion growth rate; GT = generation time; SC = sporulation capacity.

Introduction

Late blight, caused by the fungus *Phytophthora infestans* (Mont.) de Bary, is a potato disease of worldwide importance, and resistance against this pathogen is one of the main objectives of most potato breeding programmes. Resistance can be found in several wild *Solanum* species and, to some extent, in the cultivated potato. Resistance varies from low level partial resistance, as in some potato cultivars, to immunity in some wild species [Wastie, 1991]. Major genes for resistance to the disease from the Mexican species *S. demissum* have been transferred to potato, but were soon overcome by the pathogen and their use is no longer advocated [Ross, 1986; Wastie, 1991]. The partial resistance of *S. tuberosum*, which is supposed to be polygenic [Umaerus *et al.*, 1983], appears to be durable and may help to protect potato crops from late blight, but appears to be associated with late maturity [Umaerus *et al.*, 1983]. Therefore, other sources of resistance are of interest. These have been found in some South American *Solanum* species, some of which have high levels of resistance [Van Soest *et al.*, 1984; Colon and Budding, 1988]. The high level resistance of some of these wild species appears to be partial, in contrast to the hypersensitive response conferred by the major genes of *S. demissum* and *S. stoloniferum* [Ross and Baerecke, 1951; Colon and Budding, 1988].

For partial resistance, the breeding process may

be improved by screening for separate, complementary resistance components rather than for general levels of resistance [Parlevliet, 1979]. Resistance is effectuated when one or more steps in the life cycle of the pathogen are slowed down or blocked completely. In this paper, each host factor affecting a specific step in the life cycle is termed a component of resistance.

In modelling studies with *P. infestans* by Van Oijen [1992], infection efficiency (IE) and lesion growth rate (LGR) appear to be two components with major effects on the resistance level, whereas generation time (GT), sporulation capacity (SC) and sporulation period (SP) are thought to be less effective. The real importance of these components in partially resistant potato cultivars, reviewed by Van Oijen [1991], is not clear since contradictory results have been reported. However, analysis of LGR and GT of 15 cultivars in field experiments appeared to confirm the major importance of LGR, while GT appeared to be of little importance [Colon, 1995].

Less information is available on components of resistance in wild potato species. In partially resistant Mexican *Solanum* species, IE, LGR and SC appear to be reduced [Niederhauser, 1961]. In partially resistant genotypes of *S. andigena* and *S. phureja*, IE, LGR, GT and SC have been reported to be reduced [Guzmán-N, 1964]. Nilsson [1981] confirmed that IE and LGR appear to be reduced in resistant genotypes of *S. andigena* and *S. phureja* and reported that the two components are intercorrelated.

Nothing is known so far about components of resistance in other *Solanum* species. The objective of this paper was to assess IE, LGR, GT and SC in the tuber-bearing species *S. arnezii* × *hondelmannii*, *S. berthaultii*, *S. circaeifolium*, *S. leptophyes*, *S. microdontum*, *S. sparsipilum*, *S. sucrense* and *S. vernei*, both in the greenhouse and in the field, and to relate these components to resistance assessed in the field. Most emphasis was put on IE and LGR. The general level and genetic variation for resistance in the field of these species have been reported elsewhere [Colon and Budding, 1988].

Materials and methods

Plant material

All wild *Solanum* genotypes used in these experiments (Table 1) were clones of seedlings derived from the German-Dutch Potato Gene Bank at the Braunschweig Genetic Resource Centre (BGRC) in Germany. They were maintained in the form of tubers and the stock was annually renewed through multiplication in pots in the greenhouse under natural short day conditions. Tuber set was poor in many genotypes and only limited numbers of seed tubers were available for the experiments.

Samples of four hybrid progenies of *S. microdontum*, from crosses of two genotypes of BGRC 24.981, and two of BGRC 18.570, with the susceptible diploid *S. tuberosum* SH 77-114-2988 [Colon *et al.*, in press[a]], each consisting of 18–20 randomly chosen genotypes, were also tested. They were multiplied in the same way as the wild species.

The cultivars Bildtstar and Pimpernel were used as standards in most experiments. In addition to this, the susceptible parent SH 77-114-2988 was included in the four progeny experiments. The susceptible cv. Bintje and the partially resistant

Table 1. Solanum species tested

Species and accession code	Abbreviation	Number of genotypes
S. arnezii × *hondelmannii* BGRC 27.308	*axh*	7
S. berthaultii BGRC 10.063	*ber*	3
S. berthaultii BGRC 18.548	*ber*	4
S. circaeifolium ssp. *quimense* BGRC 27.036	*crc*	3
S. leptophyes BGRC 27.196	*lph*	4
S. microdontum BGRC 18.302	*mcd*	3
S. microdontum BGRC 24.981	*mcd*	10
S. microdontum var. *gigantophyllum* BGRC 18.568	*mcd*	2
S. microdontum var. *gigantophyllum* BGRC 18.570	*mcd*	11
S. microdontum var. *gigantophyllum* BGRC 27.352	*mcd*	1
S. microdontum var. *gigantophyllum* BGRC 27.353	*mcd*	2
S. sparsipilum BGRC 7.215	*spl*	8
S. sucrense BGRC 27.370	*scr*	3
S. vernei BGRC 24.733	*vrn*	5
S. tuberosum	*tub*	4

cv. Libertas were used in some experiments, to represent the extremes in resistance of *S. tuberosum*. The cultivars and the diploid parent were available at the DLO-Centre for Plant Breeding and Reproduction Research (CPRO-DLO), and were multiplied in the field under virus-limiting conditions in a seed potato growing area. All tubers were stored at 4 °C.

It was impossible to test all genotypes in one experiment, and no more than a few seed tubers of each wild genotype were available at a time. Therefore, not all of the in total 66 wild genotypes that have been tested, were included in each experiment. The progenies were tested in a separate set of four experiments, one for each progeny.

Field evaluation

Three-year (1986–1988) averages of area under the disease progress curve (ADPC) for resistance to race 1.2.3.4.5.7.10.11 (1986–1987) or 1.2.3.4.5.6.7.10.11 (1988), taken from field trials as described in Colon and Budding [1988], were used as a quantitative description of the field response of the genotypes under investigation. As will be discussed later, this type of field trial emphasizes the effect of resistance due to reduced infection efficiency and lesion growth rate. In these field trials, two plots of four plants were used for each genotype in each year. The progenies and the parent SH 77-114-2988 were tested with race 1.2.3.4.5.6.7.10.11 in the same way, in two years (1990–1991). Each year the genotypes were tested in two replicates, except for the standard cultivars Bildtstar and Pimpernel, which were tested in 20 replicates each year, and SH 77-114-2988, which was tested in 11 replicates in 1991. ADPC are relative values which have no units. Average ADPC and standard errors were estimated with the residual maximum likelihood (REML) method [Patterson and Thompson, 1971], using genotypes as fixed factor and years and blocks as random factors.

Fungal material

P. infestans race 1.2.3.4.5.6.7.10.11 was provided by the DLO-Research Institute for Plant Protection (IPO-DLO), Wageningen, from a liquid nitrogen-preserved stock. The fungus was cultured at 15 °C and 100% RH under continuous low intensity fluorescent tube (Sylvania 'cool white' 40W tubes) illumination on detached leaves of the susceptible cultivars Bintje and Bildtstar. Race identity was not checked, but the isolate was replaced by a new one from the same stock after a few multiplication cycles. In some experiments, infected leaves from an isolated trial field that had been inoculated with race 1.2.3.4.5.6.7.10.11, were used as inoculum source. In one experiment natural infection with an unknown race occurred and the plants were not inoculated.

Inoculum was prepared by rinsing leaflets with the sporulating fungus in demineralized water. Sporangia were washed using a 15 µm mesh nylon sieve and resuspended in demineralized water. The sporangial suspension was placed at 10 °C for 1–2 h to induce the release of zoospores. When zoospore densities were high enough (approximately 7×10^4 ml^{-1}), sporangia were removed from the zoospore suspension by passing it through a 15 µm mesh nylon sieve. If no zoospores could be obtained, inoculations were done with sporangia (Table 2). Spore densities were determined by counting ten 3.2 mm^3 samples of inoculum using a haemocytometer. Inoculum densities were adjusted to 7×10^4 ml^{-1}, except when zoospore release was too poor to achieve this density (Table 2). In one experiment, a higher concentration, of 11×10^4 sporangia ml^{-1} (Table 2), was used to obtain enough lesions for LGR measurements, as many genotypes in this experiment were highly resistant. Similarly, the progeny of *mcd* 167 was inoculated with 5.9×10^4 sp ml^{-1}, that of *mcd* 178 with 6.9×10^4 sp ml^{-1}, that of *mcd* 231 with 10.2×10^4 sp ml^{-1}, and that of *mcd* 264 with 15.4×10^4 sp ml^{-1}.

Growing conditions of plants prior to inoculation

Nine experiments (exp. 1–9) were done in the greenhouse and three (exp. 10–12) in the field, in five successive years. Experimental conditions are given in Table 2. The progenies were tested in four other greenhouse experiments, in 1992.

If necessary, dormancy of tubers was broken with Rindite [Burton, 1989]. Whole pre-sprouted tubers or, in the case of cultivars, eye cuttings were planted in soil in a floorless greenhouse (exp. 1–3), or in sterilized peat in 2.25 l pots in the greenhouse. The temperature was set at

Table 2. Experimental conditions of experiments 1–9, drop-inoculated in the greenhouse, and 10–12, carried out in the field, spray-inoculated

Exp nr	Rindite treatment[1]	Planting date	Inoculation date	Inoculum density		k^2	Assessment days[3]	Components assessed[4]
				Zoospores/ml	Sporangia/ml			
1	no	4 May 1988	28 June	4.8×10^4	–	413	3,4,6,8,10	LGR
2	no	16 May 1989	7 Aug	5.6×10^4	0.5×10^4	590	4,5,6,9,11	LGR,SC
3	yes	24 May 1990	31 July	–	4.0×10^4	384	5,6,7	IE,LGR
4	yes	2 Apr 1991	23 May	7.0×10^4	0.1×10^4	723	4,5,6,7	LGR
5	yes	7 Aug 1991	24 Oct	2.0×10^4	3.0×10^4	475	4,5,6,7,8	IE,LGR
6	yes	10 Sep 1991	31 Oct	7.9×10^4	–	758	4,5,6,7	IE,LGR
7	no	12 June 1992	6 Aug	2.7×10^4	11.2×10^4	1995	4,5,6	IE,LGR,SC
8	no	22 June 1992	20 Aug	6.5×10^4	–	635	4,5,6	IE,LGR,SC
9	no	15 July 1992	3 Sep	7.0×10^4	–	676	4,5,6	IE,LGR,SC
10	no	16 May 1989	10 July	–	4.4×10^4	–	4,5,6,7,8	GT,LGR
11	yes	1 May 1990	26 July	–	3.8×10^4	–	5,7	LGR
12	no	9 July 1992	natural infection			–	1/9, 2/9, 4/9	LGR,SC

[1] To break dormancy
[2] Number of zoospores and sporangia in each drop of inoculum, if drop inoculation was applied
[3] Days after inoculation, or date (exp. 12)
[4] IE = infection efficiency, LGR = lesion growth rate, SC = sporulation capacity, GT = generation time

20/15 °C day/night, but in exp. 1–9 occasionally reached 30 °C. The four progeny experiments were planted at weekly intervals from the end of April. Regular applications of NPK fertilizer (12:10:18) were supplied in all greenhouse experiments, and plants were given additional Philips Son-T or Philips-HPIT illumination (80–90 W m^{-2}) at a 16 h photoperiod. In the field, in the vicinity of Wageningen, plants were grown in a sandy soil, and given the regular cultural procedures of a potato crop on this type of soil. Seed tubers were planted at a distance of 0.35 m in hills that were 0.75 m apart in exp. 10 and 11. No hills were made and planting distance was 0.75 × 0.40 m in exp. 12. No fungicides were applied.

Inoculations

In the greenhouse experiments, plants were inoculated when they had developed enough leaves that could be used, usually about 8 weeks after planting. Plants of the four progenies were inoculated 5–6 weeks after planting. Inoculations were made on the youngest fully developed leaves, positioned about one third from the top of the plant, which was considered the best way to have leaves of about the same physiological age of the different genotypes. Inoculations were made by placing separate 10 µl drops of inoculum on the lower epidermis of a leaf or leaflet. Drop sizes were determined by counting the number of drops that filled a volume of 1.5 ml. On *S. vernei*, a species with very hairy leaves, drops would not remain attached to the lower leaf surface and inoculations were made on the upper surface of the leaves. Inoculations were done at the end of the day. Plants were incubated overnight at a high humidity (90–100% RH), created by means of vaporizers in the greenhouse (exp. 1–3), by spraying water on the upper side of the leaves and covering the plants in plastic bags (exp. 4), or by placing them in a controlled-climate greenhouse at > 90% RH, or in a mist chamber. Incubation was at 15 °C, except in exp. 1–3, where the temperature gradually decreased from 20 °C at inoculation, to 15 °C at the end of the night. In the morning, the vaporizers were turned off, bags were removed, RH was reduced to 70% or plants were transferred to the greenhouse. In exp. 1–3 and in the progeny experiments, growing conditions remained as they were prior to inoculation. In exp. 4–9, plants were incubated at 70% RH in a controlled-climate greenhouse at 20/15 °C day/night temperature and under additional illumination (Philips Son-T or Philips HPIT, 80 W m^{-2}) at a 16 h photoperiod.

In the field experiments, inoculations were

made about ten weeks after planting, when the crop had fully developed, except for exp. 12, where natural infection was observed six weeks after planting, and no inoculation was done. Inoculum was made as described, and sprayed across the plots late in the evening with a gas-driven atomizer. Plots were thoroughly wetted prior to inoculation, and in exp. 11 they were covered with a thin plastic sheet after inoculation. Experiments 10 and 11 were situated in a late blight field trial, which was given overhead irrigation every morning and every evening.

Assessment of resistance components

Experiments were done in four (exp. 1, 2, 3 and 11, and the progeny experiments) or five (exp. 4–10 and 12) randomized blocks. Each genotype was represented by one plant in each block, though in some cases blocks were incomplete due to insufficient seed.

Infection efficiency (IE). (IE) was assessed in the greenhouse, in the experiments 3 and 5–9, and in the four progeny experiments. Infection efficiency was expressed as the chance p of a single spore to be successful, which is more meaningful epidemiologically than the percentage of inoculum drops resulting in infection. The chance p was estimated, on single plant basis, from the fraction of inoculum drops that resulted in growing lesions, using the formula $p = 1 - H^{1/k}$, given by Swallow [1987] to estimate small probabilities of virus transmission by single vectors from data derived from group inoculations. H is the fraction of unsuccessful inoculations and k is the number of spores in each inoculum drop. The factor k (Table 2) was estimated from the inoculum density and the drop size. Estimated p values of plants with at least one unsuccessful inoculation were found to be in the range 0.00–0.08. If all inoculations on a plant had been successful and consequently p equalled 1.0, which is an exceptionally large value, an alternative p for this plant was estimated on the total of plants of that genotype in the experiment. This was done for 63 of the 290 plants that were tested in exp. 3 and 5–9, and for 28 of the 332 plants of the four progenies. If p was 1.0 for all plants of a genotype in an experiment, the lower limit for p was estimated as if one inoculum drop had not given infection and the resulting value was assigned to all plants of this genotype. This was done for 20 plants in exp. 3 and 5–9, of six wild genotypes, and for SH 77-114-2988 in the experiments with the progenies of *mcd* 231 and *mcd* 264.

For the wild species, the resulting p values were analyzed for the entire data set, with the average p values of the susceptible cv. Bildtstar in each of the experiments as covariates, while those of the four progenies were analyzed for each experiment separately. The analyses were done using a general linearized model [McCullagh and Nelder, 1989], with a Poisson distribution and a logarithmic link function. Residuals were checked for uniformity. Average p values and standard errors of the genotypes were predicted from the model. To compare groups of genotypes, the mean p values and standard errors of the groups were predicted from a similar model, with groups instead of genotypes as independent variable.

Lesion growth rate (LGR). In all experiments except exp. 12, lesions were measured daily from the first appearance of symptoms, usually 4 days after inoculation, until leaflets of the most susceptible genotypes were nearly destroyed by the pathogen, 7–11 days after inoculation. The greatest length of the lesion, usually parallel with the secondary leaf veins, and the largest width along an axis perpendicular to the first measurement, were measured. The area of the measured ellipse, $^1/_4$ π.length.width, was square root transformed, and the average linear lesion growth rate in mm day^{-1} was estimated by linear regression on time. Lesion growth rates were estimated for lesions that reached a final size of more than 5 mm^2 and only LGR values larger than 0.1 mm day^{-1} were included in the analysis. Small lesions and low LGR values were usually associated with highly necrotic lesions that did not extend beyond the originally inoculated area, whereas other lesions on the same genotype grew to a much larger size. These non-growing lesions were regarded as unsuccessful infections, the pathogen being stopped by a hypersensitive reaction, and were therefore not included in the LGR analysis. The analysis was restricted to plants with at least two LGR values, and to genotypes of which at least two plants fulfilled this requirement. Average LGR values were calculated for each leaf (con-

sisting of one to several leaflets), and the averages were analyzed for the combined data sets of the greenhouse and of the field, with the average LGR values of the susceptible cv. Bintje in exp. 1, and the cv. Bildtstar in each of the other experiments as covariates. For the cultivars, data from several experiments were combined in one analysis. Average LGR and standard errors of the wild genotypes were estimated with REML [Patterson and Thompson, 1971], using genotypes as a fixed factor, leaf position (= leaf age) as covariate, experiments (for the cultivars) and plants as random factor, and the number of lesions on a leaf as weight. The random factor blocks was found to be zero or negative, and dropped from the model. In the field experiments the random factor blocks was also dropped for the same reason. In the progeny experiments, REML was also used, with replicates, leaves and plants as random factors and genotypes and leaf positions as fixed factors. Residuals were checked for uniformity.

Generation time (GT). GT was measured in the field in exp. 10. As soon as lesions became visible, which was 4 days after inoculation, a strip of cellotape was gently pressed against the lesion on the lower side of the leaf each morning. The strip was placed on a microscope slide, a drop of lactophenol-acid fuchsin was added and examined for the presence of sporangia under a microscope at 320× magnification. Lesions were followed in this way till 7 days after inoculation, or until they had begun to sporulate. Generation times were estimated through logistic regression, as time in days after inoculation at which 50% of the finally achieved number of actually sporulating lesions was reached.

Sporulation capacity (SC). SC was determined in exp. 2, 7, 8, 9 and 12, on leaves that had also been used to assess LGR. Leaves with well-developed lesions were collected immediately after the last LGR measurement and incubated at 100% RH on wet filter paper in closed containers (exp. 2), or in closed plastic tubes where a few drops of water were added if leaves were dry. Incubation was in the dark for 3 days (exp. 2), 44h (exp. 7) or 24h (exp. 8, 9 and 12). After incubation, lesions in exp. 2, with the underlying piece of paper, were transferred to plastic tubes and stored at –18 °C. Sporangia were washed off with 20 ml of 0.8 M NaCl (to prevent germination of sporangia), tubes were shaken for 30 minutes at 200–300 rpm, and the sporangia were counted. In the other experiments, sporulation was stopped by adding 10 ml 0.8 M NaCl and spores were washed off by shaking the tubes for 10 seconds on a Whirlmix, after which the leaves were discarded. Spores were allowed to settle on the bottom of the tube, the volume of the spore suspension was reduced to 2.2 ml through careful removal of the upper part of the fluid, and spores were resuspended by shaking the tubes before counting. Very few spores were found in the discarded fluids. Sporangial densities were determined by counting two 3.2 mm^3 samples in a haemocytometer. From spore counts and total volume the total numbers of spores produced were derived and, with lesion circumference estimated as $^1/_2\pi$(length + width), expressed in spores cm^{-1} lesion circumference. The circumference was used instead of the area, because *P. infestans* lesions mainly sporulate at their edges. Spore counts were then log-transformed (^{e}log) and averaged per sample, and the averages were analyzed for the combined data sets of the greenhouse and of the field, with the average SC values of the susceptible cv. Bildtstar in each of the experiments as covariates. For the cultivars, data from several experiments were combined in one analysis. Average SC and standard errors were estimated with REML [Patterson and Thompson, 1971], using genotypes as a fixed factor, leaf position as covariate and experiments (for the cultivars) and plants as random factor. The random factor blocks was found to be zero or negative and dropped from the model. Residuals were checked for uniformity. Estimates of SC and standard errors were then transformed back to a linear scale (e^{SC}) and least significant ratios (LSR) were calculated from LSD.

Spearman's rank correlations between genotypic means of ADPC, IE and LGR were estimated for each of the four progenies.

Statistical analyses were carried out using Genstat [Payne *et al.*, 1987]. For all components significance levels were computed at $P = 0.05$, unless stated otherwise. LSD values of genotypic means of IE were calculated from s.e.d. as $t_{240} \times \sqrt{(s.e.d._1^2 + s.e.d._2^2)}$. LSD values of genotypic

means of LGR and SC were calculated from s.d. and n as $t_{0.05;\,df} \times$ s.d. $\times \sqrt{[(n_1 + n_2)/n_1 n_2]}$. If results from several experiments were averaged, overall error variances were derived from the error variances of the experiments as a weighted average, using the number of replicates in an experiment as weighing factor [Chapter 14, Cochran and Cox, 1957].

Results

Solanum tuberosum

Measurements of GT, IE, LGR and SC of the two standard cultivars in the 12 experiments are given in Table 3. The cv. Pimpernel (ADPC = 0.46) was moderately resistant, whereas the cv. Bildtstar (ADPC = 0.66) was susceptible. Averaged for all experiments, IE, LGR, and SC were all significantly reduced in the cv. Pimpernel compared to the cv. Bildtstar, although in some experiments the differences were too small to be significant. The GT on the cv. Pimpernel was 5.0 days, which is not much longer than the 4.6 days found on the cv. Bintje (Table 8).

Results of IE, LGR and SC of three cultivars, from a subset of experiments, are given in Table 4. ADPC values ranged from 0.46 (Libertas, Pimpernel) to 0.66 (Bildtstar). The cv. Bildtstar had significantly higher IE, LGR and SC than the two partially resistant cultivars. It had 1.9 × higher IE, 2.0 × higher LGR and 2.4 × higher SC than the cv. Libertas. The cv. Libertas had lower IE and LGR than the cv. Pimpernel, while the two cultivars were equal with respect to SC.

Solanum arnezii × *hondelmannii*

Infection efficiency and LGR of six genotypes and SC of two genotypes of this species were measured (Table 5), while GT was not tested. Partial resistance of the genotypes was moderate, ADPC values ranging from 0.27 to 0.48.

Infection efficiency was significantly lower on all genotypes compared with the cv. Bildtstar, except *axh* 71, and showed no apparent relationship with ADPC. Lesion growth rate appeared significantly reduced compared with the susceptible standard Bildtstar in all genotypes except *axh* 53, both in the greenhouse and in the field, and *axh* 57 in the greenhouse, while it did not appear

Table 3. Resistance components infection efficiency (IE; the chance $p \times 10000$; predicted from exp. 3 and 5–9), linear lesion growth rate (LGR; mm/day), and sporulation capacity (SC; × 1000 spores/cm) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on the standard cultivars Bildtstar, susceptible, and Pimpernel, partially resistant. n = the number of inoculum drops (IE) or lesions (LGR, SC). Least significant differences (LSD) between overall genotypic means at $P < 0.05$ were 4.8 for IE, derived from a generalized linear model analysis of 29 plants of each cultivar, and 0.47 for LGR. Least significant ratio (LSR) for SC, derived from analysis of variance of the experiment means, was 2.0

Exp.	IE				LGR				SC			
	Bildtstar		Pimpernel		Bildtstar		Pimpernel		Bildtstar		Pimpernel	
	Mean	n	Mean	n	Mean	n	Mean	n	Mean	n	Mean	n
1	–		(42.2)	48	–		(4.04)	42	–		–	
2	–		–		3.96	22	2.86	15	268	37	73	6
3	28.1	72	2.3	86	(4.13)	43	–		–		–	
4	–		–		3.39	40	3.05	58	–		–	
5	21.3	130	19.6	117	2.94	82	1.73	68	–		–	
6	60.0	102	56.6	109	4.41	102	3.48	107	–		–	
7	22.8	96	20.8	64	5.91	96	3.54	64	163	19	109	14
8	54.6	89	45.1	95	3.58	87	2.37	91	180	25	121	23
9	53.1	104	34.7	82	3.85	102	2.39	74	49	19	10	12
10	–		–		–		(1.29)	35	–		–	
11	–		–		2.11	12	1.32	11	–		–	
12	–		–		2.37	14	1.99	22	44	14	10	20
Mean[1]	40.0		29.9		3.61		2.53		109		40	

[1] Values between brackets not included.

Table 4. Infection efficiency (IE; the chance $p \times 10000$; predicted from exp. 3 and 5–9), linear lesion growth rate (LGR; mm/day; averages of exp. 7, 8 and 9, and of exp. 11 and 12), generation time (GT; days post inoculation; exp. 10) and sporulation capacity (SC; × 1000 spores/cm; average of exp. 7, 8 and 9, and of exp. 12) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on *S. tuberosum*, measured in the greenhouse or in the field. ADPC = area under the disease progress curve (three-year average). *n* = numbers of plants (IE, LGR, SC) or lesions (GT). The maximum LSD or LSR, for the lowest *n*, are given

Genotype	ADPC	IE (*n*)	LGR (*n*)		GT (*n*)	SC (*n*)	
			Greenhouse	Field		Greenhouse	Field
Libertas	0.46	16.8 (19)	2.22 (15)	1.18 (8)	5.1 (13)	54 (14)	20 (5)
Pimpernel	0.46	24.8 (29)	2.81 (15)	1.73 (8)	5.0 (13)	54 (12)	20 (5)
Bildtstar	0.66	32.5 (29)	4.50 (14)	2.27 (6)	–	109 (14)	54 (3)
LSD ($P < 0.05$)	0.05	4.8	0.44	0.45		1.6	3.7

Table 5. Infection efficiency (IE; the chance $p \times 10000$; exp. 3 and 5), linear lesion growth rate (LGR; mm/day; exp. 2, 3 and 5) and sporulation capacity (SC; × 1000 spores/cm; exp. 2 and 12) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on *S. arnezii* × *hondelmannii* BGRC 27.308 and two potato cultivars, measured in the greenhouse or in the field. ADPC = area under the disease progress curve (three-year average). *n* = numbers of plants (IE) or leaves (LGR, SC). Values in italics are significantly lower than those of the cv. Bildtstar at $P < 0.05$

Genotype	ADPC	IE (*n*)		LGR (*n*)		SC (*n*)	
		Mean	s.d.	Greenhouse	Field	Greenhouse	Field
axh 54	*0.27*	*0.0* (2)	0.0	–	–	–	–
axh 66	*0.31*	–	–	*1.72* (10)	–	–	–
axh 70	*0.35*	*9.8* (3)	4.6	*0.98* (6)	*0.52* (3)	–	–
axh 53	*0.35*	*10.3* (3)	4.7	3.84 (11)	1.46 (3)	89 (2)	–
axh 71	*0.35*	26.4 (4)	6.7	*2.16* (11)	*0.95* (3)	–	9 (3)
axh 58	*0.40*	*0.0* (1)	0.0	–	*0.84* (3)	–	–
axh 57	*0.48*	*18.6* (4)	5.6	3.86 (13)	*1.06* (3)	–	–
Pimpernel	*0.46*	*24.8* (29)	1.9	*2.97* (100)	*1.54* (13)	73 (14)	22 (5)
Bildtstar	0.66	32.5 (29)	2.2	4.16 (108)	2.19 (6)	163 (23)	54 (3)
s.d. (df)	0.08 (289)	–		1.34 (88)	0.52 (142)	2.0 (64)	5.5 (24)

related to ADPC. The SC was slightly lower compared to the cv. Bildtstar in *axh* 53 and *axh* 71, but these differences were not significant.

Solanum berthaultii

Infection efficiency of seven genotypes, LGR of two genotypes and SC of one genotype, representing two accessions, were measured (Table 6), while GT was not assessed. Partial resistance was high, ADPC values ranging from 0.00 to 0.22.

Infection efficiency appeared generally very low and always much lower than those of the cvs. Pimpernel and Bildtstar. Few lesions could be measured and for BGRC 18.548 LGR values of the 25 lesions that developed were not significantly different from those of the cv. Bildtstar, while for BGRC 10.063 LGR values were very low in nine out of the ten lesions that were measured, so low that it is doubtful whether these lesions should be termed growing lesions. Sporulation capacity was measured on *ber* 41, and found to be not significantly different compared with the cv. Bildtstar.

Solanum circaeifolium ssp. *quimense*

Three genotypes of this accession, all without symptoms in the field trials, were tested, in exp. 5 and 8; they were inoculated with 80, 106 and 136 drops of inoculum respectively. No lesions developed; on one plant two necrotic spots appeared which did not develop into lesions and may have been due to other causes than late blight infection.

Table 6. Infection efficiency (IE; the chance $p \times 10000$; exp. 3 and 6), linear lesion growth rate (LGR; mm/day; exp. 1 and 6) and sporulation capacity (SC; × 1000 spores/cm; exp. 9) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on *S. berthaultii* BGRC 10.063 (*ber* 9, 11 and 29) and BGRC 18.548 (*ber* 38, 39, 41 and 44) and two potato cultivars, measured in the greenhouse. ADPC = area under the disease progress curve (three-year average). *n* = numbers of plants (IE) or leaves (LGR, SC). Values in italics are significantly lower than those of the cv. Bildtstar at $P < 0.05$

Genotype	ADPC	IE (*n*)		LGR (*n*)	SC
		Mean	s.d.	Greenhouse	Greenhouse
ber 9	*0.00*	*0.0* (2)	0.0	–	–
ber 11	*0.06*	*0.4* (4)	0.8	–	–
ber 29	*0.17*	*1.0* (5)	0.8	*0.16* (5)	–
ber 39	*0.01*	*0.0* (2)	0.0	–	–
ber 44	*0.01*	*0.0* (4)	0.0	–	–
ber 41	*0.11*	*7.8* (1)	4.1	3.18 (6)	268 (8)
ber 38	*0.22*	*2.0* (1)	2.1	–	–
Pimpernel	*0.46*	*24.8* (29)	1.9	*2.97* (100)	*73* (14)
Bildtstar	0.66	32.5 (29)	2.2	4.16 (108)	163 (23)
s.d. (df)	0.08 (289)	–		1.34 (88)	2.0 (64)

Solanum leptophyes

Infection efficiency and LGR of four genotypes of this species were measured (Table 7), while GT and SC were not tested. Partial resistance was moderate to low, ADPC values of the genotypes varied between 0.48 and 0.60.

On *lph* 98, all inoculations were successful, and the *p* value given to this genotype in Table 7 is the lower limit that was estimated. Both IE and LGR appeared to vary among the host genotypes and were not consistently lower on the most resistant genotypes than on the most susceptible genotypes or the cv. Bildtstar.

Solanum microdontum

Infection efficiency of 16 genotypes, LGR of 26 genotypes, GT of nine genotypes and SC of eight genotypes of six accessions were measured (Tables 8, 9 and 10). ADPC values of the genotypes ranged from 0.00 to 0.75.

In the accessions BGRC 24.981 and BGRC 18.570, infection efficiency varied widely and showed no relationship with field-tested resistance (Tables 9 and 10). On *mcd* 175, *mcd* 176, *mcd* 179, *mcd* 262 and *mcd* 265, all inoculations were successful and the lower limits of *p* are given. *S. microdontum* exhibited the potential of a strong

Table 7. Infection efficiency (IE; the chance $p \times 10000$; exp. 3, 6 and 9) and linear lesion growth rate (LGR; mm/day; exp. 6 and 9) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on *S. leptophyes* BGRC 27.196 and two potato cultivars, measured in the greenhouse. ADPC = area under the disease progress curve (three-year average). *n* = numbers of plants (IE) or leaves (LGR). Values in italics are significantly lower than those of the cv. Bildtstar at $P < 0.05$

Genotype	ADPC	IE (*n*)		LGR (*n*)
		Mean	s.d.	Greenhouse
lph 102	*0.48*	29.0 (7)	3.5	*3.33* (29)
lph 100	*0.55*	*23.4* (7)	3.1	3.80 (25)
lph 106	*0.56*	*21.9* (6)	3.2	*2.13* (11)
lph 98	0.60	> 26.6 (5)	3.8	3.67 (16)
Pimpernel	*0.46*	*24.8* (29)	1.9	*2.97* (100)
Bildtstar	0.66	32.5 (29)	2.2	4.16 (108)
s.d. (df)	0.08 (289)	–		1.34 (88)

Table 8. Linear lesion growth rate (LGR; mm/day; exp. 1 and 2), generation time (GT; days post inoculation; exp. 10) and sporulation capacity (SC; × 1000 spores/cm; exp. 2) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on *S. microdontum* BGRC 18.302 (*mcd* 125, 127 and 130) and BGRC 18.568 (*mcd* 218 and 224) and three potato cultivars, measured in the greenhouse or in the field. ADPC = area under the disease progress curve (three-year average). *n* = numbers of leaves (LGR, SC) or lesions (GT). Values in italics are significantly lower than those of the cv. Bildtstar (ADPC) or Bintje (LGR, SC) at $P < 0.05$

Genotype	ADPC	LGR (*n*)	GT	SC
		Greenhouse	Field	Greenhouse
mcd 127	*0.08*	*0.93* (7)	–	–
mcd 130	*0.16*	–	4.6 (3)	–
mcd 125	*0.57*	3.87 (17)	–	*10.9* (2)
mcd 218	*0.14*	*2.29* (8)	–	–
mcd 224	*0.38*	*3.38* (13)	–	–
Pimpernel	*0.46*	*2.97* (100)	5.0 (13)	*11.2* (14)
Bildtstar	0.66	–	–	–
Bintje	0.80	4.16 (108)	4.6 (11)	12.0 (23)
s.d. (df)	0.08 (289)	1.34 (88)		0.7 (64)

Table 9. Infection efficiency (IE; the chance $p \times 10000$; exp. 3 and 7), linear lesion growth rate (LGR; mm/day; exp. 3, 4, 7 and 11), generation time (GT; days post inoculation; exp. 10) and sporulation capacity (SC; × 1000 spores/cm; exp. 7) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on *S. microdontum* BGRC 24.981 and three potato cultivars, measured in the greenhouse or in the field. ADPC = area under the disease progress curve (three-year average). *n* = numbers of plants (IE), leaves (LGR, SC) or lesions (GT). Values in italics are significantly lower than those of the cv. Bildtstar at $P < 0.05$

Genotype	ADPC	IE (*n*)		LGR (*n*)		GT	SC (*n*)
		Mean	s.d.	Greenhouse	Field	Field	Greenhouse
mcd 167	*0.02*	*0.0* (4)	0.0	*0.32* (5)	–	–	121 (4)
mcd 199	*0.02*	*4.4* (3)	3.1	*0.26* (6)	–	–	–
mcd 176	*0.08*	> 29.0 (2)	9.8	*2.06* (6)	–	–	147 (4)
mcd 178	*0.10*	–		*0.80* (4)	*0.38* (3)	–	–
mcd 182	*0.26*	–		–	*1.04* (3)	5.7 (11)	–
mcd 179	*0.27*	> 33.0 (3)	8.5	*3.23* (21)	–	–	220 (4)
mcd 181	*0.35*	–		–	1.84 (4)	4.4 (11)	–
mcd 180	*0.46*	–		–	1.58 (2)	–	–
mcd 175	*0.48*	> 27.8 (2)	9.5	*2.32* (5)	–	–	180 (3)
mcd 164	*0.53*	*7.4* (4)	3.5	*3.13* (13)	–	–	–
Pimpernel	*0.46*	*24.8* (29)	1.9	*2.97* (100)	*1.54* (13)	5.0 (13)	*73* (14)
Bildtstar	0.66	32.5 (29)	2.2	4.16 (108)	2.19 (6)	–	163 (23)
Bintje	0.80	–		–	–	4.6 (11)	–
s.d. (df)	0.08 (289)	–		1.34 (88)	0.52 (142)		2.0 (64)

necrotic reaction in all but the most susceptible genotypes. The colour of the necrotic tissue could be very dark brown or even black. The expression of this reaction varied between and within plants, leaves and even lesions of the same genotype. Some lesions were necrotic and non-growing at one end, while they expanded rapidly, with little necrosis, at the other end. Younger leaves gave this necrotic reaction more often than older leaves. In the greenhouse, very low LGR were found on all highly resistant genotypes except *mcd* 176 (Table 9), which also gave a susceptible reaction with respect to IE, and *mcd* 249 (Table 10). The susceptible *mcd* 263 (Table 10) had a low IE, and a low LGR in the greenhouse, while LGR was not measured in the field. In the field, differences in LGR were smaller, but still significantly lower on three partially resis-

Table 10. Infection efficiency (IE; the chance $p \times 10000$; exp. 3 and 7), linear lesion growth rate (LGR; mm/day; exp. 3, 4, 7 and 11), generation time (GT; days post inoculation; exp. 10) and sporulation capacity (SC; × 1000 spores/cm; exp. 7) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on *S. microdontum* BGRC 18.570 (*mcd* 231–265), BGRC 27.352 (*mcd* 276) and BGRC 27.353 (*mcd* 298 and 310), and three potato cultivars, measured in the greenhouse or in the field. ADPC = area under the disease progress curve (three-year average). *n* = numbers of plants (IE), leaves (LGR, SC), or lesions (GT). Values in italics are significantly lower than those of the cv. Bildtstar at $P < 0.05$.

Genotype	ADPC	IE (*n*)		LGR (*n*)		GT	SC (*n*)
		Mean	s.d.	Greenhouse	Field	Field	Greenhouse
mcd 231	*0.00*	*0.2* (6)	0.5	*0.29* (4)	–	7.1 (2)	–
mcd 234	*0.02*	*0.7* (4)	1.1	*0.19* (5)	–	–	–
mcd 244	*0.06*	–		–	*1.05* (2)	5.3 (5)	–
mcd 264	*0.06*	*1.3* (4)	1.5	–	–	–	–
mcd 249	*0.20*	*8.6* (5)	3.3	3.86 (12)	*0.78* (2)	–	–
mcd 245	*0.35*	*1.1* (3)	1.6	–	*0.72* (2)	–	–
mcd 258	*0.34*	*13.6* (3)	5.4	*1.47* (20)	–	–	44 (4)
mcd 262	*0.57*	> 35.5 (4)	7.6	*3.66* (26)	–	–	109 (11)
mcd 238	0.61	*3.6* (1)	4.8	–	2.14 (5)	4.1 (6)	–
mcd 263	0.67	*3.7* (3)	2.8	*0.99* (4)	–	–	–
mcd 265	0.75	> 35.1 (4)	7.6	*3.70* (22)	–	–	121 (46)
mcd 276	*0.21*	–		–	1.56 (2)	4.2 (7)	–
mcd 310	*0.04*	–		–	–	7.1 (3)	–
mcd 298	*0.22*	–		–	–	5.6 (2)	–
Pimpernel	*0.46*	*24.8* (29)	1.9	*2.97* (100)	*1.54* (13)	5.0 (13)	*73* (14)
Bildtstar	0.66	32.5 (29)	2.2	4.16 (108)	2.19 (6)	–	163 (23)
Bintje	0.80	–		–	–	4.6 (11)	–
s.d. (df)	0.08 (289)	–		1.34 (88)	0.52 (142)	–	2.0 (64)

tant genotypes than on one susceptible genotype, and than on the cv. Bildtstar. Generation times of some genotypes are given in Tables 8, 9 and 10. They appeared to be longer on resistant than on susceptible genotypes, but only a few lesions were followed. In BGRC 24.981 and BGRC 18.570 (Table 10), SC appeared generally high, sometimes higher than on the cultivars (Table 9), while in BGRC 18.302 (Table 8) it was significantly less on one resistant genotype than on the cv. Bildtstar. The genotype *mcd* 167, with very low LGR, produced high numbers of spores (Table 9).

In the progenies of *mcd* 167, *mcd* 178, *mcd* 231 and *mcd* 264, LGR was highly variable, whereas IE was highly variable in *mcd* 167 × SH 77-114-2988, but less so in the other progenies. IE and LGR were significantly correlated with ADPC, except for LGR in the progeny of *mcd* 231 (Table 11). Multiple regression of ADPC on both IE and LGR did not give higher correlations (not shown), probably because the two components appeared closely correlated (Table 11).

Solanum sparsipilum

Infection efficiency of five genotypes, LGR of seven genotypes and SC of two genotypes of this species were measured (Table 12). No data were collected on GT. ADPC values of the genotypes ranged from 0.26 to 0.76.

Infection efficiency and LGR were sometimes, but not consistently found low on partially resistant and high on susceptible genotypes. One susceptible genotype had a very low SC compared to the cultivars, a difference which was highly significant, while another, more susceptible genotype produced high spore numbers. Sporulation capacity was not assessed on the partially resistant genotypes.

Solanum sucrense

Infection efficiency of three genotypes and LGR of one genotype of this species were measured (Table 12), while SC and GT were not assessed. Two genotypes were highly resistant (ADPC below 0.02), the other genotype was moderately susceptible, its ADPC value being 0.37.

Table 11. Ranges of, and Spearman's rank correlation coefficients between genotypic means of infection in the field with race 1.2.3.4.5.6.7.10.11 of *P. infestans* (area under the disease progress curve; ADPC), and infection efficiency (IE) and lesion growth rate (LGR; mm/day) measured in the greenhouse, of four hybrid progenies of *S. microdontum* crossed with the susceptible *S. tuberosum* pollinator SH77-114-2988. The average ADPC of the susceptible parent was 0.50. The IE values of the susceptible parent were 34.6, 20.2, > 29.5 and > 22.4 and the LGR values 4.29, 3.97, 3.69 and 2.47 in the four experiments respectively. The numbers of genotypes are given between brackets. All correlations are significant at $P < 0.01$, except when marked ns.

Wild parent	Range			Correlation coefficient (r)		
	ADPC	LGR	IE	IE vs. ADPC	LGR vs. ADPC	IE vs. LGR
mcd 167	0.02–0.62	0.44–4.00	0.0–51.8	0.70 (20)	0.75 (17)	0.74 (17)
mcd 178	0.16–0.61	0.74–4.78	0.4–21.7	0.61 (18)	0.66 (16)	0.91 (18)
mcd 231	0.04–0.29	0.96–4.24	0.0–22.9	0.59 (18)	0.29 (12) ns	0.78 (12)
mcd 264	0.03–0.21	0.25–2.84	0.6–25.9	0.75 (18)	0.74 (17)	0.84 (17)

Table 12. Infection efficiency (IE; the chance $p \times 10000$; exp. 5 and 9), linear lesion growth rate (LGR; mm/day; exp. 1 and 9) and sporulation capacity (SC; × 1000 spores/cm; exp. 2 and 9) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on *S. sparsipilum* BGRC 7.215, *S. sucrense* BGRC 27.370 and two potato cultivars, measured in the greenhouse. ADPC = area under the disease progress curve (three-year average). n = numbers of plants (IE) or leaves (LGR, SC). Values in italics are significantly lower than those of the cv. Bildtstar at $P < 0.05$

Genotype	ADPC	IE (n)		LGR (n)	SC
		Mean	s.d.	Greenhouse	Greenhouse
spl 408	*0.26*	*3.6* (7)	1.5	3.87 (4)	–
spl 390	*0.31*	–	–	*0.74* (4)	–
spl 402	*0.47*	*0.7* (3)	1.2	–	–
spl 410	*0.45*	33.1 (4)	5.2	*3.09* (9)	–
spl 400	0.64	21.8 (2)	5.4	*2.87* (5)	–
spl 381	0.64	*11.1* (5)	2.6	*2.04* (11)	*9* (6)
spl 399	0.73	–	–	*3.06* (8)	–
spl 382	0.76	–	–	3.66 (7)	147 (3)
scr 447	*0.02*	*0.1* (5)	0.2	–	–
scr 454	*0.02*	*0.0* (3)	0.0	–	–
scr 469	*0.37*	*22.8* (8)	3.3	*1.47* (14)	–
Pimpernel	*0.46*	*24.8* (29)	1.9	*2.97* (100)	*73* (14)
Bildtstar	0.66	32.5 (29)	2.2	4.16 (108)	163 (23)
s.d. (df)	0.08 (289)	–		1.34 (88)	2.0 (64)

Very low IE were found on the resistant genotypes, whereas on the susceptible genotype IE was slightly, but significantly lower than on the cultivars. The LGR of the moderately susceptible genotype was significantly lower than that of the cvs. Pimpernel and Bildtstar.

Solanum vernei

Infection efficiency and SC of four genotypes and LGR of five genotypes of this species, were measured (Table 13). ADPC values of the genotypes ranged from 0.10 to 0.41.

Infection efficiencies were found very low compared to the cultivars on all genotypes. Lesion growth rates on all genotypes were significantly lower than on the cv. Bildtstar, both in the greenhouse and in the field. Sporulation capacity in the greenhouse was significantly lower than on the cv. Bildtstar on all genotypes, while these differences were comparable, but mostly not significant in the field.

Table 13. Infection efficiency (IE; the chance $p \times 10000$; exp. 8), linear lesion growth rate (LGR; mm/day; exp. 1, 8 and 12) and sporulation capacity (SC; × 1000 spores/cm; exp. 8 and 12) of *Phytophthora infestans* race 1.2.3.4.5.6.7.10.11 on *S. vernei* BGRC 27.733 and two potato cultivars, measured in the greenhouse or in the field. ADPC = area under the disease progress curve (three-year average). n = numbers of plants (IE) or leaves (LGR, SC). Values in italics are significantly lower than those of the cv. Bildtstar at $P < 0.05$

Genotype	ADPC	IE (n)		LGR (n		SC (n)	
		Mean	s.d.	Greenhouse	Field	Greenhouse	Field
vrn 531	*0.10*	–		*2.88* (7)	–	–	–
vrn 530	*0.20*	*2.4* (4)	1.2	*1.65* (7)	*0.78* (4)	*18* (6)	11 (4)
vrn 514	*0.24*	*4.9* (6)	1.4	*2.01* (10)	–	*20* (8)	–
vrn 515	*0.29*	*0.2* (3)	0.4	–	*1.02* (3)	–	*4* (3)
vrn 526	*0.41*	*13.4* (5)	2.7	*3.02* (21)	*1.57* (5)	*24* (7)	12 (5)
Pimpernel	*0.46*	*24.8* (29)	1.9	*2.97* (100)	*1.54* (13)	*73* (14)	22 (5)
Bildtstar	0.66	32.5 (29)	2.2	4.16 (108)	2.19 (6)	163 (23)	54 (3)
s.d. (df)	0.08 (289)	–		1.34 (88)	0.52 (142)	2.0 (64)	5.5 (24)

Discussion

Here genetic variation for components of resistance to *P. infestans* in wild *Solanum* species was considered, related to resistance in the field.

The data should be interpreted with care: the measurements of components, especially of IE, strongly depend on the quality of the inoculum and the environmental conditions during inoculation and incubation. These factors obviously differed between experiments, as indicated by the variation in the results of the standard cultivars. Also, experiments were done in different seasons of the year. The numbers of lesions per plant on which GT, LGR and SC have been assessed, were often low. Nevertheless, the fact that of the two standard cultivars, Pimpernel always had the lowest LGR and SC, and nearly always also the lowest IE, indicates that the conditions of the tests allowed for a fairly reliable ranking of genotypes with respect to these components.

Lesion growth rates assessed in the field appeared much lower than those assessed in the greenhouse. Sporulation capacity of leaves collected in the field were also lower than SC of leaves from the greenhouse. This would suggest that either field-grown leaves are better able to withstand infection or, in the case of LGR, that climatic conditions in the field are less favourable for the development of the disease. Nevertheless, since no apparent inversions in the ranking order of the standard genotypes were seen when comparing field results with data from the greenhouse, both methods appeared suitable to assess LGR. Similarly, leaves both from the field and from the greenhouse appeared suitable to assess SC.

There appeared to be much genetic variation for IE, LGR and SC among the 66 wild genotypes and the four potato cultivars under consideration. Low IE and LGR values were found in some genotypes of all the wild species except *S. leptophyes*. Generation times were long in some genotypes of *S. microdontum*, while they were not assessed for the other wild species. Low SC were found in *S. sparsipilum* and *S. vernei*, while in *S. microdontum* SC could be very high. Sporulation capacity was not assessed for *S. leptophyes*. In *S. microdontum*, IE and LGR appeared positively associated, and negatively associated with GT. The positive association of IE and LGR was supported by the results of progenies of genotypes of this species crossed with susceptible diploid *S. tuberosum*.

A general relationship between IE and LGR, in the sense that lesions originating from inoculum drops of a higher spore density tend to have higher LGR values, has been reported for late blight on tomato [Turkensteen, 1973]. This might be due to the higher density of mycelium in lesions originating from multiple infections. It is likely that the same relation would play a role in drop inoculations of potato. Therefore, the positive associations between IE and LGR, and the genetic variation of LGR that we have found for *S. microdontum* and *S. tuberosum* may be artefacts.

However, it is unlikely that all the genetic variation observed for LGR would be due to variation in IE, since the variation for LGR was also found in field experiments which were spray-inoculated or naturally infected, and consequently most lesions stem from single infections.

Results of *S. vernei*, suggesting that both IE and LGR are low, need to be interpreted with care, as most of these plants were inoculated on the upper surface of the leaves, which is reported to be less easily infected than the lower surface [Lapwood, 1960]. Therefore, the low IE found on this species may be an underestimation. There is no obvious reason why LGR or SC would be influenced by the side of the leaf which is chosen for inoculation, once the pathogen has established itself inside the leaf. Therefore, our observations of these components of resistance made on *S. vernei* are thought to be more reliable.

To relate the genetic variation for components of resistance to *P. infestans* to the variation in ADPC, used to quantify partial resistance to *P. infestans*, was complicated, due to the unbalanced nature of many of the experiments. Results from simulations of late blight epidemics of potato indicate that IE and LGR will probably have the greatest effect on the resistance of a potato genotype, while GT and SC appear less important [Van Oijen, 1992]. Consequently, resistant genotypes are expected to have either a reduced IE, or a reduced LGR or both, compared to susceptible genotypes, while variation for SC and GT is expected to be less clearly related to resistance. It needs to be realized that our experiments were not really suitable to study associations of GT and SC with resistance. The ADPC values we used as indicators of partial resistance, were derived from field trials with small plots of 2–4 plants, interspersed with rows of susceptible plants, all of which were inoculated. Such a field trial is in fact a continuous monocyclic test [Zadoks and Schein, 1979], in which the inoculum produced by a partially resistant genotype will contribute relatively little to the disease developing on this genotype, as most of the inoculum will come from the spreader rows and from heavily infected plots nearby. This effect is called interplot interference and is known to be important in field experiments with late blight [Paysour and Fry, 1983]. As a consequence, ADPC will be influenced less by resistance components related to the production of inoculum, i.e. GT and SC, than by IE and LGR. Even so, some associations between GT or SC and partial resistance were found. Our experiments also indicated that there is genetic variation for GT and SC. The relation we have demonstrated between SC and ADPC, most clearly in *S. tuberosum*, indicates that genetic variation for this trait is comparatively large.

In our experiments with *S. tuberosum*, SC was the component that varied most between susceptible and partially resistant genotypes, with LGR and IE immediately following, and all seemed to be strongly related to ADPC. A large genetic variation for LGR and a close correlation with ADPC were also found in a larger set of potato cultivars, in which there was little genetic variation for GT [Colon *et al.*, 1995].

While in *S. tuberosum* the ranking orders of genotypes according to ADPC, IE and LGR were similar, the picture for the wild genotypes was less consistent, as many genotypes had IE or LGR values that were not in accordance with their ADPC value. In general, IE appeared to be an important component in one accession of *S. microdontum* and in *S. sucrense*, and also in *S. berthaultii*, *S. circaeifolium* and *S. vernei*, although we had no susceptible genotypes of the three latter species. Lesion growth rate appeared related to ADPC in *S. microdontum* and possibly also in *S. sucrense*, though of this species only one moderately susceptible genotype was tested, and in *S. vernei*, of which no susceptible genotypes were examined. The importance of IE and LGR in *S. microdontum* was supported by the results of the four hybrid progenies of this species. Generation time seemed related to ADPC in *S. microdontum*, while it was not measured on other species. Sporulation capacity appeared related to ADPC in one accession of *S. microdontum*.

In *S. microdontum*, interactions with the environment might account for the many exceptions to these general trends. This species exhibited the potential of a strong necrotic reaction in all but the most susceptible genotypes. Infection efficiencies of *S. microdontum* genotypes also appeared to have higher standard errors than those of the other species. These findings suggest a delicate balance between triggering and non-triggering of the necrotic response, which may strongly depend on

the environment. If the response is triggered, some lesions will have reduced growth rates, while the potential growth rate on that genotype may be much larger. Inoculation of plants with spores collected from relatively fast growing lesions did not result in increased susceptibility (data not shown), so virulent segregants of the pathogen within these lesions probably do not account for the observed variation. On very young leaves of susceptible *S. tuberosum*, necrotic or half-necrotic lesions were also found, though rarely, so this reaction, although stronger on the wild species, may not be typical for *S. microdontum*.

In *S. microdontum*, another component, which we have not studied in detail, may also interfere with the relation between IE, LGR and ADPC, namely stem infection. In this species, we repeatedly observed in field trials sudden death of a plant, which did not appear heavily infected, because its main stem was infected by *P. infestans* and broke off. This may explain why genotypes with low IE and low LGR sometimes were observed to have a high ADPC. Stem infection was also often found in other species, but usually only on genotypes with susceptible leaves.

In *S. berthaultii* BGRC 18.548, genotypes with a low IE developed lesions with a high LGR, and therefore IE probably is the main component responsible for the low ADPC values found for this accession. This suggests a different resistance mechanism in *S. berthaultii* than in the other species described in this paper. Resistance to late blight in *S. berthaultii* has been attributed to type B trichomes, typical for the species, which produce a fungistatic exudate; the resistance appears to be reduced when the exudate is washed off the leaves [Holley *et al.*, 1987]. Infection efficiency would most probably be affected by this 'external' resistance mechanism, whereas LGR needs not be affected. The other *S. berthaultii* accession, BGRC 10.063, is likely to have another mechanism in addition to this, possibly of a hypersensitive nature, which reduces LGR. It is interesting to note that of all the *Solanum* species we have tested in the field, only *S. berthaultii* was relatively susceptible in 1987 [Colon and Budding, 1988]. In that year, there had been much more precipitation during crop growth than in the two other test years; week averages for rainfall during the 50 days prior to inoculation were 16.7 mm week^{-1} in 1987 and 8.4 and 5.2 mm week^{-1} in the two other years. Heavy rain is expected to reduce the amount of exudate on the leaves, resulting in higher IE. Therefore, the major role IE appears to play in the resistance of *S. berthaultii* to *P. infestans*, in contrast to the situation in other *Solanum* species, would be in accordance with the putative involvement of type B trichomes.

We have found genetic variation for GT, IE, LGR and SC in most of the wild species discussed in this paper, though more markedly for IE, LGR and SC than for GT. Especially for IE and LGR, but also for SC, higher levels of resistance were found amongst the wild species than within *S. tuberosum*. The importance of individual components of resistance varied between the species, and our results underline the complexity and the variability of this particular plant-pathogen interaction. Although reliable estimates of components of resistance to *P. infestans* are not easily obtained, the transfer and even combination of components from different wild species may be feasible. The most obvious way to achieve this would be to analyse components of resistance of parents, in order to select the crossing combinations that are potentially the best. Field selection based on ADPC values then seems the most appropriate method to select progeny genotypes with high levels of resistance to *P. infestans*.

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