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RESEARCH ARTICLE

Resistance to *Phytophthora infestans* in *Solanum tuberosum* landraces in Southern Chile

Jaime Solano[1], Ivette Acuña[2], Florence Esnault[3] & Philippe Brabant[4]

[1]Universidad Católica de Temuco, Facultad de Recursos Naturales, Escuela de Agronomía, Rudecindo Ortega 02950, Casilla 15-D, Temuco, Chile; [2]Instituto de Investigaciones Agropecuarias, CRI-Remehue, Casilla 24-0, Osorno, Chile; [3]Institut National de la Recherche Agronomique, UMR 1349 IGEPP INRA, Agrocampus Ouest, Université Rennes, 1 Keraiber F-29260, Ploudaniel, France; [4]AgroParisTech, UMR 320, Le Moulon, F-91190, Gif sur Yvette, France

Author for correspondence: Jaime Solano, e-mail: jsolano@uct.cl

ABSTRACT

The objective of this work was to evaluate the resistance of 30 Chilean potato landraces to natural infection by *Phytophthora infestans* during two growing seasons in Southern Chile. Control cultivars were 'Désirée', which has moderate susceptibility to late blight, and 'Karú', which is characterized by moderate resistance to the disease. The response of the potato landraces to late blight infection was assessed weekly by scoring the percentage of foliage destruction during the growing season. Subsequently, the relative area under the disease progress curve (AUDPCr) was calculated. A wide response variation was observed among the landraces during the occurrence of late blight. The comparative analysis of the AUDPCr showed that three landraces achieved high resistance to the disease (low AUDPCr values). These were UCT-34Cor (0.05), UCT-26Ach (0.06) and UCT-27Mu (0.10). Most of the potato landraces were classified into the range moderately resistant to moderately susceptible. The estimated values of heritabilities of the means were moderate to high for the joint analysis of the two years tested (0.65). The wide range of AUDPCr observed within potato landraces suggests the presence of partial resistance in response to the complex virulence pattern of the *P. infestans* populations present in Southern Chile.
Key words: genetic variability, late blight resistance, potato germplasm, relative area under disease progress curve (AUDPCr).

INTRODUCTION

Chile is considered to be a sub-centre of origin of cultivated potatoes (*Solanum tuberosum* ssp. *tuberosum* L.), with native and introduced genotypes co-existing in the country (Spooner et al., 2005; Solano et al., 2007). Potatoes landraces present a rich variety of colours, both of the pulp and the skin, opening up great possibilities for the development of novel gourmet products. Potato late blight, caused by *Phytophthora infestans* Mont. De Bary, is one of the most important diseases of the crop (Hijmans et al., 2000), and can completely destroy the crop in a short time. It is therefore considered to be the most serious problem affecting potato production worldwide. It affects plants at any stage of development and up to 100% of the production may be lost. Late blight infections are very difficult to control due to the genetic diversity of *P. infestans* populations (Fry, 2008).

The search for genetic resistance to late blight has intensified, since it is a practical and economic way of controlling the disease. Initially the search concentrated on specific resistance (Flier et al., 2003; Forbes et al., 2005). This type of resistance is characterized by a hypersensitive response in the form of small necrotic lesions, and is called race-specific, monogenic, qualitative, or complete resistance. It is based on the recognition of avirulence genes (*Avr*) of the pathogen by major plant resistance genes (*R-genes*) (Flor, 1971). However, this type of resistance is not very long-lasting, since *P. infestans* populations can quickly overcome it (Flier et al., 1998; Flier et al., 2003). More recently, the emphasis has been switched to non-specific resistance which is considered to be more stable over time and space (Goodwin et al., 1995; Micheletto et al., 1999; Micheletto et al., 2000; Parlevliet, 2002; Barquero et al., 2005b; Forbes et al., 2005; Naerstad et al., 2007). This type of resistance, also known as horizontal, quantitative or field resistance, is governed by a large number of minor genes (*r*), with small and additive effects; it apparently does not involve gene-by-gene interaction and therefore is assumed to be race non-specific. Its stability is attributed to its capacity to maintain a balance between all the races of *P. infestans* present in a given location. Horizontal resistance acts in different ways, either through the production of toxic exudates on the leaf surface, confinement of the fungal structures to the cell walls, low colonization of the mesophyll, slow collapse of the petioles or reduction of the pathogen reproductive range. In other words, it raises physiological or chemical barriers in the tissues of the host (Colon et al., 1995).

Colon et al. (1995) indicated that resistance to late blight may be found in various wild species of *Solanum*, and to some degree in different cultivated potatoes. This resistance varies from low level partial resistance, as in

some cultivated potatoes, to immunity in some wild species. The majority of the genes identified are involved in foliar resistance, while very little is known about the genes which affect resistance in the tubers (Simko et al., 2006).

The first report of *P. infestans* in Chile dates from the 1950s, supposedly introduced from Argentina (Arentsen, 1994). Since then, migrations have played an important role in the dispersion of this pathogen (Ristaino, 2009). In Southern Chile, highly aggressive blight attacks on potato crops with losses greater than 50% have been observed during several years. This may be explained by changes in the populations of the fungus towards more virulent forms due to excessive applications of metalaxyl (Pérez et al., 2001; Páez et al., 2005) or more favourable environmental conditions (McLeod et al., 2001).

Acuña et al. (2007) determined that the populations of *P. infestans* in Chile are only of the A1 (asexual) mating type; no sexual (A2) mating types have been reported in Chile yet. However, *P. infestans* has experienced a genetic change to genotypes which are highly resistant to metalaxyl and complex pathotypes with up to 9 and 10 *avr* genes (*avr11*, *avr10*, *avr8*, *avr7*, *avr5*, *avr3* and *avr1*). In the Chiloe archipelago, the tubers of native potato varieties have been a staple food of the population for years. Many local varieties and ecotypes might have had high susceptibility to the disease before the arrival of late blight on the island. Nevertheless, with the arrival of more resistant potato varieties to the island, the selection of resistant clones could be started. Identification of some of the *avr* genes present in *P. infestans* isolates recovered from native material showed that they are complex, with the presence of various *avr* genes (Acuña et al., 2008). The objective of this work was to evaluate the resistance of 30 potato landraces to natural infections by *P. infestans* during two growing season in Southern Chile. The heritability of this resistance was also estimated.

MATERIALS AND METHODS

Plant material

Thirty potato landraces from Southern Chile, the majority from Chiloé Island, were evaluated (Table 1) for their reaction to late blight. This collection is maintained in the field at the Catholic University of Temuco. Control cultivars were 'Désirée', which has moderate susceptibility to late blight and is the most widely planted variety in Chile, and 'Karú', which is characterized by moderate resistance to late blight (Kalazich et al., 2004).

Growing seasons and crop treatment

The genotypes were evaluated over two farming seasons (2008/09 and 2009/10) with field tests established in the Pillanlelbún Experimental Station (38°39' 2.21" S; 72° 27' 3.4"), Araucanía Region, Chile. The soil conditions were pH (in water) 5.6, Olsen-P 14.6 mg/kg and 26.5% organic matter. In each season the field trials were established between October 15th and 20th, with emergence occurring in the following month. The seed dose used was equivalent to 2,500 kg/ha. The sowing distances used were 0.7 m between rows and 0.3 m between plants in the row. For each of the two seasons, a randomized complete block design was used with four replications. The crop was fertilized with a 11:30:11 N:P:K mix at a dose equivalent to 1200 kg/ha. Under these conditions the vegetative cycle was completed by mid-December. The crop was banked up 60 days after planting when the plants were between 20 an 30 cm in height. In all the seasons, weeds were controlled by one application of metribuzin, at a dose equivalent to 0.56 L/ha of the active ingredient.

Evaluation of resistance to *P. infestans*

The inoculum of *P. infestans* were from natural infestation. The accessions were scored for the percentage of foliage destruction during the growing season (Henfling, 1987). The weekly evaluations started when the first symptoms of the disease were observed, corresponding to 75 and 70 days after planting in 2008/09 and 2009/10 respectively. They stopped 47 and 34 days later, which corresponded to 121 and 103 days after planting for the 2008/09 and 2009/10 seasons, respectively. Subsequently, the area under the disease progress curve (AUDPC) was calculated (Jeger & Viljanen-Rollinson, 2001; Yuen & Forbes, 2009) using equation (1):

$$AUDPC = \sum_{i=0}^{n-1} [(X_{i+1}+X_i)/2](T_{i+1}-T_i) \quad (1)$$

where: $AUDPC$ = area under the disease progress curve, X_i = % of foliage destruction at the ith scoring, $(T_{i+1}-T_i)$=Time elapsed between two scorings, n=Total number of scorings.

Then, the relative area under the disease progress curve (AUDPCr) was calculated. This value was obtained dividing the AUDPC by the total number of days elapsed between the first and last evaluation of the foliage destruction (equation 2):

$$AUDPCr = \frac{AUDPC / 100}{\text{(Time in days between the last evaluation and the first evaluation)}} \quad (2)$$

Evaluations with 100% of the leaf area diseased with late blight have a value of 1. All the AUDPCr values are expressed as a proportion of this value. Low AUDPCr

TABLE 1 - Identification, local name and origin of *Solanum tuberosum* landraces evaluated for resistance to *P. infestans* under natural occurrence in Southern Chile. UCT, collection of the Catholic University of Temuco.

Accession	Local name	Origin
UCT-11Mgb	Meca gato blanca	Chiloe Island
UCT-14MgRe	Redonda	Chiloe Island
UCT-17Br	Bruja	Quinchao Island
UCT-6Gc	Guadacho colorado	Chonchi Island
UCT-24Tn	Tonta	Castro, Chiloe Island
UCT-22Cm	Clavela morada	Castro, Chiloe Island
UCT-25Gñ	Guicoña	Quellón, Chiloe Island
UCT- 7Ca	Camota	Chiloe Island
UCT-18Mn	Michuñe negro	Chiloe Island
UCT-26Ach	Azul chañihue	Chiloe Island
UCT-27Mu	Murta	Quellón, Chiloe Island
UCT-28MiR	Michuñe rojo	Chiloe Island
UCT-29Mol	Molejona	Chiloe Island
UCT- 3Cl	Clavela	Los Muermos, Continent
UCT- 1Ma	Michuñe azul	Chiloe Island
UCT-16At	Azul tabla	Chiloe Island
UCT-30Ño	Ñocha	Chiloe Island
UCT-19Aq	Azul de quento	Castro, Chiloe Island
UCT-2Lv	Lengua	Castro. Chiloe Island
UTC-31Ob	Ojitos blanco	Ancud, Chiloe Island
UCT-32Ci	Cielito	Castro, Chiloe Island
UCT-20Ro	Rosada	Chiloe Island
UCT-33Cab	Cabrita	Chiloe Island
UCT-15MgRo	Meca gato rojo	Chiloe Island
UCT-21Ac	Azul cristalina	Chiloe Island
UCT-34Cor	Cordillera	Castro, Chiloe Island
UCT-35AzC	Azul caucheque	Castro, Chiloe Island
UCT-8Gb	Guadacho blanco	Ancud, Chiloe Island
UCT-9MgM	Meca gato morada	Ancud, Chiloe Island
UCT-10MgL	Meca gato morada larga	Los Muermos, continent
'Desirée'	Cultivar	Europe
'Karú'	Cultivar	Chile

values indicate low levels of infection during the evaluation period, corresponding to the more resistant genotypes (Pérez & Forbes, 2008).

Statistical analysis

A randomized complete block design was used with 32 treatments and four repetitions. The AUDPCr data were subjected to analysis of variance (ANOVA). A previous analysis showed that the square root transformation homogenized the residual variances between seasons and gave more discriminating results. Because the AUDPCr was easier to interpret (proportion of what should be observed for a fully susceptible accession) we have presented the AUDPCr values in the results section and in the tables but all the statistical tests have been realized on √AUDPCr.

The following model was used for each season:

$$P_{ik}=\mu+G_i+B_k+E_{ik}\ ,$$

(3)

where: μ = general mean, G_i = genetic effect of landrace i, B_k = environmental effect of block k, and E_{ik} = residual effect.

We also performed the analysis on the whole dataset pooling the two seasons together with the following model:

$$P_{jk}=\mu+G_i+Y_j+(GxY)_{ij}+B(Y)_{jk}+E_{ijk},$$

(4)

where: μ = general mean, G_i = genetic effect of landrace i, Y_j = season effect of season j, $(GxY)_{ij}$ = interaction between genotype i and season j, $B(Y)_{jk}$ = effect of block k nested in year j, E_{ijk} = residual effect. The statistical program used was R package version 2.15.0.

In a first approach we considered each experiment at the time and submitted the data to a fixed model analysis. We then compared the different landraces to 'Karú' using a two-tailed Bonferroni *t* test ($p \leq 0.05$) which resulted in

a P_c of 0.0017 for each of the 30 comparisons. In a second approach we considered the experiment as a representative sample of experiments that could be performed. As a consequence we considered all effects as random. This allowed the calculation of the different variances, namely σ^2_G, σ^2_B and σ^2_E using equation 3, and σ^2_G, σ^2_Y, σ^2_{GxY}, σ^2_B and σ^2_E with equation 4. The variances were estimated with the restricted maximum-likelihood method implemented in the lmer function of the R package. In the case of the model defined by equation 4 and under the assumption that the genetic *vs.* season interaction (GxY) effects were normally distributed, we used the GxY mean square as residual mean square of the F statistic to test the null hypotheses (σ^2_G,=0 and σ^2_Y =0).

Heritabilities

Broad sense heritabilities (H^2) were calculated considering the 30 accessions and the two standard potato cultivars. Two different heritabilities were calculated each season, one (H_1^2) based on individual values measured at the plot level and the other (H_m^2) based on the mean of the four replications (equations 5 and 6):

$$H_1^2 = \sigma^2_G/(\sigma^2_G+\sigma^2_B+\sigma^2_E), \quad (5)$$

$$H_m^2=\sigma^2_G/(\sigma^2_G+(\sigma^2_B+\sigma^2_E)/4), \quad (6)$$

Two different heritabilities have also been calculated on the whole dataset taking into account the genotype *vs.* season interaction, one (H_{1b}^2) based on individual values measured at the plot level and the other (H_{mb}^2) based on the mean of the eight available values for each genotype (two seasons and four replications each season) (equations 5b and 6b):

$$H_{1b}^2=\sigma^2_G/(\sigma^2_G+\sigma^2_{GxY}+\sigma^2_B+\sigma^2_E), \quad (5b)$$

$$H_{mb}^2=\sigma^2_G/(\sigma^2_G+\sigma^2_{GxY}/2+(\sigma^2_B+\sigma^2_E)/8), \quad (6b)$$

RESULTS

First season

Great differences were observed in leaf damage due to late blight in all the evaluations (4.3 to 62.5%). The accessions with the lowest percentage of leaf damage were UCT-34Cor, UCT-6Gc and UCT-26Ach, which at the sixth evaluation (121 days after planting) presented 4.37%, 10.0% and 11.8% of foliage destruction respectively. It should be added that the late blight symptoms occurred later, especially in accessions UCT-34Cor, UCT-26Ach and UCT-30Ño. The accessions presenting the greatest leaf damage towards crop maturity were UCT-29Mol, UCT-16At, UCT-32Ci and UCT33-Cab, all with 62.5% leaf damage. The cultivars Desirée and Karú presented moderate levels of leaf damage with 53.75% and 31.25% of foliage destruction respectively. Based on the AUDPCr, the observed differences between accessions have proved highly significant ($F_{Geno\ (31,93)} = 6.32$, $P<0.0001$). The highest AUDPCr value was found for accession UCT-33cab, with a value of 0.25 (Table 2). This was followed by accessions UCT-28MiR and UCT-24Tn with AUDPCr values of 0.23 and 0.22, respectively. The lowest AUDPCr value corresponded to accession UCT-34Cor, with 0.01. This was followed by UCT-26Ach and UCT-30Ño with AUDPCr values of 0.03 and 0.04 respectively. The control cultivars presented AUDPCr values of 0.12 (‘Désirée’) and 0.07 (‘Karú’).

Second season

This season presented a high incidence of late blight, associated with a rainy spring and summer and a high level of precipitation in January. This led to rapid progress of the disease, with accession UCT-33Cab for example presenting 100% leaf damage at 103 days after planting. On the other hand, the results show that significant differences exist in leaf damage due to late blight in all the evaluations (28 to 100%). In this season, the accessions with the lowest percentage of leaf damage were UCT-34Cor, UCT-26Ach and UCT-27Mu, which at 103 days after planting presented 28.0%, 28.75% and 30.0% damage respectively. During the first 90 days after planting accessions UCT-34Cor, UCT-26Ach and UCT-27Mu presented very low levels of leaf damage, not exceeding 10%. By contrast, several accessions reached high levels of leaf damage due to late blight early in the season, for example UCT-33Cab, UCT-25Gñ and UCT-14MgRe with 100.0%, 96.75% and 94.25% of foliage destruction respectively.

Significant differences were observed for AUDPCr ($F_{Geno\ (31,93)} = 8.72$, $P<0.0001$), with the highest value presented by accession UCT-25Gñ at 0.49 (Table 2). High values were also recorded for accessions UCT-32Ci and UCT-33Cab, with 0.46 for both of them. As in the previous season, the lowest value for the AUDPCr corresponded to accession UCT-34Cor with 0.09. This was followed by accessions UCT-27Mu and UCT-26Ach with 0.09 for both of them. The control cultivars presented AUDPCr values of 0.20 (‘Désirée’) and 0.17 (‘Karú’).

Two seasons

The results observed for each season independently showed noticeable differences between seasons for the mean level of attack and for the relative response of the varieties. The overall mean AUDPCr was 0.14 for 2008/09, while for 2009/10 it was 0.25. A lower level of disease and

TABLE 2 - Mean area under the disease progress curve of the 32 *Solanum tuberosum* landraces evaluated for resistance to *Phytophthora infestans* under natural occurrence in Southern Chile in 2008/09 and 2009/10.

Accessions	2008/09		2009/10	
	AUDPCr	Comparison with 'Karú' [1]	AUDPCr	Comparison with 'Karú'
UCT-11Mgb	0.21	> [2]	0.19	=
UCT-14MgRe	0.14	=	0.36	>
UCT-17Br	0.18	=	0.20	=
UCT-6Gc	0.08	=	0.18	=
UCT-24Tn	0.22	>	0.17	=
UCT-22Cm	0.19	>	0.22	=
UCT-25Gñ	0.20	>	0.49	>
UCT-7Ca	0.12	=	0.31	=
UCT-18Mn	0.16	=	0.32	=
UCT-26Ach	0.03	=	0.09	=
UCT-27Mu	0.12	=	0.09	=
UCT-28MiR	0.23	>	0.26	=
UCT-29Mol	0.18	=	0.27	=
UCT-3Cl	0.19	=	0.25	=
UCT-1Ma	0.12	=	0.24	=
UCT-16At	0.19	=	0.21	=
UCT-30Ño	0.04	=	0,29	=
UCT-19Aq	0.12	=	0.37	>
UCT- 2Lv	0.20	>	0.22	=
UTC-31Ob	0.10	=	0.29	=
UCT-32Ci	0.21	>	0.46	>
UCT-20Ro	0.15	=	0.23	=
UCT-33Cab	0.25	>	0.46	>
UCT-15MgRo	0.10	=	0.25	=
UCT-21Ac	0.15	=	0.38	>
UCT-34Cor	0.01	=	0.09	=
UCT-35AzC	0.20	=	0.30	=
UCT-8Gb	0.14	=	0.20	=
UCT-9MgM	0.09	=	0.25	=
UCT-10MgL	0.12	=	0.26	=
'Desirée'	0.12	=	0.20	=
'Karú'	0.07	Control	0.17	Control

[1] Comparison with 'Karú' based on the Bonferroni *t* test (P<0.05) and performed on √AUDPCr.
[2] >, significantly different from 'Karú' with higher mean value; =, not significantly different from 'Karú'.

a smaller range of variation were observed for 2008/09 compared to 2009/10.

The residual variances for AUDPCr were found to be statistically different between seasons (P=0.014), therefore the square root transformation was used to homogenize the residual variances (P=0.32) and provide better resolution.

The correlation between seasons for the mean √AUDPCr of the 32 landraces was significant (r=0.49, P=0.0042), and in accordance with this result the effect of varieties was also significant over the two seasons ($F_{Geno\ (31,186)} = 11.04$, $P< 0.0001$). The comparisons of means are presented in Table 3. Fourteen accessions presented an AUDPCr significantly different from that of Karú: thirteen presented higher values and only one (UCT-34Cor) presented an AUDPCr (0.05) significantly lower than Karú's (0.12). UCT-26Ach also presented a low AUDPCr value (0.06) that was not significant because of the multiple comparison correction. Nevertheless, the observed probability associated with the individual comparison between UCT-26Ach and Karú was low (P=0.0042).

Variances and heritabilities

The analysis under the random effects hypothesis proved that the genetic variances (σ^2_G) were significantly different from zero in 2008/09 and 2009/10 and that the estimated values of σ^2_G were very close between seasons (0.00727 and 0.00852 in 2008/09 and 2009/10, respectively). When the analysis was performed on the two seasons together we observed a decrease of σ^2_G (0.00447) which was the consequence of a significant genetic *vs.* season interaction ($\sigma^2_{GxY} = 0.00315$) of the same order of magnitude as σ^2_G. Nevertheless the σ^2_G remained significantly different from zero. As a consequence the heritabilities (Table 4) at the single plot level have been estimated higher in 2008/09

TABLE 3 - Mean area under the disease progress curve of the 32 *Solanum tuberosum* landraces evaluated for resistance to *Phytophthora infestans* under natural occurrence in Southern Chile for joint analysis of the two seasons (2008/09 and 2009/10).

Ranking	Accessions	Comparison with 'Karú'[1]	AUDPCr
1	UCT-34Cor	<[2]	0.05
2	UCT-26Ach	=	0.06
3	UCT-27Mu	=	0.10
4	'Karú'	Control	0.12
5	UCT-6Gc	=	0.13
6	'Desirée'	=	0.16
7	UCT-30Ño	=	0.16
8	UCT-9MgM	=	0.17
9	UCT-8Gb	=	0.17
10	UCT-15MgRo	=	0.17
11	UCT-1Ma	=	0.18
12	UCT-17Br	=	0.19
13	UCT-10MgL	=	0.19
14	UCT-20Ro	=	0.19
15	UTC-31Ob	=	0.19
16	UCT-24Tn	=	0.20
17	UCT-16At	=	0.20
18	UCT-11Mgb	=	0.20
19	UCT-22Cm	=	0.21
20	UCT-2Lv	>	0.21
21	UCT-7Ca	>	0.22
22	UCT-3Cl	>	0.22
23	UCT-29Mol	>	0.23
24	UCT-19Aq	>	0.24
25	UCT-28MiR	>	0.24
26	UCT-18Mn	>	0.24
27	UCT-35AzC	>	0.25
28	UCT-14MgRe	>	0.25
29	UCT-21Ac	>	0.26
30	UCT-32Ci	>	0.33
31	UCT-25Gñ	>	0.34
32	UCT-33Cab	>	0.35

[1] Comparison with 'Karú' based on the Bonferroni *t* test ($P<0.05$) and performed on √AUDPCr.
[2] >, Significantly different from 'Karú' with higher mean value; =, not significantly different from 'Karú'; <, significantly different from 'Karú' with lower mean value.

TABLE 4 - Heritabilities of √AUDPCr estimated on a sample of 32 *Solanum tuberosum* landraces evaluated for resistance to *P. infestans* under natural occurrence in Southern Chile in two different seasons (2008/09 and 2009/10) and for joint analysis of the two seasons.

Years	H_l^2	H_m^2
2008/09	0.58[a]	0.85[c]
2009/10	0.53	0.82
	H_{lb}^2	H_{mb}^2
Two seasons	0.31[b]	0.65[d]

[a] Heritability estimated at the plot level each season
[b] Heritability estimated at the plot level for the two seasons together
[c] Heritability based on the mean of the four replications each season
[d] Heritability based on the mean over seasons and replications

and in 2009/10 (0.58 and 0.53 respectively) than over the two seasons together (0.31). The estimated values of heritabilities of the means were high in each season (0.85 and 0.82 in 2008/09 and 2009/10 respectively) and moderate to high for the two seasons together (0.65).

DISCUSSION

The prevailing climatic conditions during the two farming seasons led to development of the disease in varying degrees of incidence and severity. The results showed that native potatoes contain material with widely varying response and behaviour with respect to late blight infection. In a comparative analysis of the relative AUDPC of the different accessions (Table 2), at least three native varieties present a high level of resistance to late blight disease, namely UCT-34Cor, UCT-26Ach and UCT-27Mu. The results obtained show that accession UCT-25Gñ was always among the accessions with the highest AUDPC values, translating into high relative AUDPCr indices. A similar trend was observed for accessions UCT-32Ci and UCT-33cab.

Wide variation in response to this disease in potato have been reported by various authors (Micheletto et al., 2000; Flier et al., 2003; Jenkins & Jones, 2003; Barquero et al., 2005a; Lucca et al., 2008; Andreu et al., 2009; Mendoza, 2010). Barquero et al. (2005b) reported the existence of important differences between genotypes resulting from crosses with different wild species. They also indicated that the level of resistance present within groups of genotypes varies according to the locality, suggesting that the presence of resistance is influenced by the effect of major genes, the expression of which is dependent on the variation in the avirulence genes in the *P. infestans* population present in the locations where the crop is cultivated. Likewise, genotypes resulting from somatic hybridizations or crosses with the wild species *Solanum bulbocastanum* Dunal, *S. circaeifolium* Bitter and *Solanum okadae* Hawkes & Hjert. were those which presented the lowest values for the area under the disease progress curve, with values of 60, 80 and 79 respectively compared to the varieties Alpha, Waych'a, Pimpernell and Granola, used as controls, which presented the highest values of 477, 474, 466 and 427 respectively. They showed that the resistance level may be defined in two categories: one as a high level of partial resistance and the other as complete resistance. In situations where there is a wide range of levels of infection, these suggest an incomplete, high level, non-specific resistance. In this context, Colon et al. (1995) reported that a wide expression of resistance in different accessions of a single genotype suggests the presence of numerous minor genes with additive effects scattered in the different genotypes.

The results of field experiments conducted by Flier et al. (2003) showed differences between cultivars in terms of the stability of partial resistance. The level of resistance to late blight varies widely from very susceptible to moderately resistant in terms of AUDPC and percentage of blight in tubers. They reported significant values of AUDPC for the effects of cultivar, isolation, season, cultivar by isolation and cultivar by season. The presence of a differential interaction which is independent of R-genes indicates some degree of adaptation to partial resistance of *P. infestans*, with the consequent effects on the stability and durability of the resistance. They added that the cultivar Bintje was the most susceptible (AUDPCr of 0.52). The cultivars Santé and Pimpernel presented intermediate levels of resistance with AUDPCr values of 0.21 and 0.24 respectively. Finally, the most resistant cultivar was Karnico, with AUDPCr of 1.9. The significant effects of variety found by joint variance analysis agree with the results obtained by Flier et al. (2003). The genotypes studied are stable and can be differentiated in three dissimilar groups, the resistant (AUDPCr 0.05-0.15), moderately resistant (0.15-0.30) and moderately susceptible (0.30-0.45).

Other evaluations of quantitative resistance under field and greenhouse conditions, done by Micheletto et al. (2000) showed both highly resistant and susceptible genotypes. The AUDPC results indicated a high degree of variation at the level of wild species, and within each species. These results agree with those reported by Lucca et al. (2008), who stated that the variability observed on field trials with *S. tuberosum* ssp. *andigena* (adg) and *S. tarijense* Hawkes revealed significant differences between the genotypes in the partial and final area under the disease progress curve. Likewise, Andreu et al. (2009), evaluating the susceptibility of 10 potato cultivars in Argentina, reported that the cultivar Shepody was the most susceptible with a value of 8 (on a scale of 1-9, where 9 is highly susceptible) and had the highest AUDPC values. The cultivar Ranger Russet presented values close to 2.5, with resistance which may not be race-specific. Finally, the AUDPCr values observed in the present study are lower than those reported by Mendoza (2010) in Cameroon, who reported AUDPCr values of 0.39 and 0.49 for resistant and moderately resistant material, respectively.

Andrivon et al. (2003) reported that the severity of late blight disease was significantly lower in susceptible cultivars when they were planted in rows alternating with cultivars which had partial resistance. Mixing cultivars produced a large reduction in the disease progress rate. On the other hand, Huarte & Capezio (2003) reported that genotype *vs.* environment (GxE) interactions in resistance to late blight have not yet been clearly determined. The absence of genotype *vs.* environment interactions has also been noted by Andrivon et al. (2007) for different varieties in different locations in Morocco. In general terms, the significance of the interaction does not appear to be greater than the variation due to genotype and location. This is in accordance with our results that show an interesting level of repeatability over seasons. The wide range of AUDPC observed within native potato varieties suggests the presence of partial resistance in response to the complex virulence pattern (10 *avr* genes) of most *P. infestans* populations present in Southern Chile. On the other hand, the values of broad sense heritability estimates for AUDPC are lower than those reported by Andrade et al. (2010). These authors reported broad sense heritability values of up to 0.62 for 15 families obtained by crossing six parents (INRA 92t. 114. 76, 'Robusta', 'Pollerita', 'Chotañawi', 'Jaspe' and 'Libertas') inoculated with a complex of *P. infestans* isolates (*avr1, avr3, avr4, avr7, avr8, avr10* and *avr11*) in Argentina. These values are high enough to allow for an effective selection of resistance. In our experiment, the broad sense heritability of field resistance to late blight in potato landraces reached levels of 0.65 for heritability based on the mean over seasons and replications, and the majority of the potato landraces fall into the range moderately resistant to moderately susceptible. The heritability for resistance in these native potatoes is large enough to allow progress from selection.

In conclusion, the studied native potatoes present a wide variation of responses to late blight. The resistant material identified in this experiment offers great possibilities for exploiting and making use of it, either directly by integrated crop management or indirectly

by incorporation into breeding programs. At least three accessions were distinguished with high level of resistance to the disease and low AUDPCr values: UCT-34Cor, UCT-26Ach and UCT-27Mu. These genotypes may be grown in mixtures with more susceptible materials, which could greatly reduce the disease progress rate.

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