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ORIGINAL PAPER

Characterization of the expression and inheritance of potato leafroll virus (PLRV) and potato virus Y (PVY) resistance in three generations of germplasm derived from *Solanum etuberosum*

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Abstract Potato virus Y (PVY) and potato leafroll virus (PLRV) are two of the most important viral pathogens of potato. Infection of potato by these viruses results in losses of yield and quality in commercial production and in the rejection of seed in certification programs. Host plant resistance to these two viruses was identified in the backcross progeny of a *Solanum etuberosum* Lindl. somatic hybrid. Multiple years of field evaluations with high-virus inoculum and aphid populations have shown the PVY and PLRV resistances of *S. etuberosum* to be stably expressed in two generations of progeny. However, while PLRV resistance was transmitted and expressed in the third generation of backcrossing to cultivated potato (*Solanum tuberosum* L. subsp. *tuberosum*), PVY resistance was lost. PLRV resistance appears to be monogenic based on the inheritance of resistance in a BC_3 population. Data from a previous evaluation of the BC_2 progeny used in this study provides evidence that PLRV resistance was partly conferred by reduced PLRV accumulation in foliage. The field and grafting data presented in this study suggests that resistance to the systemic spread of PLRV from infected foliage to tubers also contributes to the observed resistance from *S. etuberosum*. The PLRV resistance contributed by *S. etuberosum* is stably transmitted and expressed through sexual generations and therefore would be useful to potato breeders for the development of PLRV resistant potato cultivars.

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Introduction

Potato leafroll virus (PLRV; Genus *Polerovirus*; Family *Luteoviridae*) and potato virus Y (PVY; Genus *Potyvirus*; Family *Potyviridae*) are the two most important virus pathogens of potato worldwide. Early season infection of potato by PVY has been documented as reducing yields by up to 33% (Nolte et al. 2003). Planting of seed infected with PVY can result in yield losses of up to 80% (Bantarri et al. 1993). PLRV can cause similar yield losses in potato and additionally can result in further economic losses to growers through rejection in the marketplace due to net necrosis. Net necrosis is a visual symptom of PLRV infection expressed as necrotic lesions in the tubers of some varieties making them aesthetically unsuitable for sale to consumers, or for processing into potato products.

Potato virus Y and PLRV are vectored most efficiently by green peach aphid [*Myzus persicae* (Sulzer)] (Ragsdale et al. 2001). As a result, green peach aphid and other aphid species were identified as the potato pest most frequently targeted for pesticide applications

in the United States (Wiese et al. 1998). However, green peach aphid has been identified as having four, insecticide resistance mechanisms that have eliminated the efficacy of several classes of insecticides that previously were effective in their control (Zamoum et al. 2005). The increasing frequency of fungicide applications for the control of newer genotypes of late blight also has increased aphid populations in some field situations by harming beneficial fungi that attack aphids (Lagnaoui and Radcliffe 1998). Poor aphid control combined with increased acreages of PVY susceptible cultivars have resulted in reports of increased potato seed lot rejections by state seed certification agencies. PLRV-infected hairy nightshade [*Solanum sarrachoides* (Sendtner)] and potato plants, when compared with non-infected plants, also have been found to be superior hosts for green peach aphid (Alvarez and Srinivasan 2005; Srinivasan et al. 2006). Increased nymph survival and adult fecundity on PLRV-infected plants can result in increased green peach aphid populations—control of PLRV therefore can also have positive benefits for reducing the vector transmission of PVY and other non-persistently transmitted potato viruses.

Host plant resistances to PVY, PLRV, and green peach aphid can be effective components of an integrated pest management (IPM) approach for the control of these insect-vectored viruses. Currently, none of the 13 most widely grown potato cultivars in North American are classified as having resistance to PVY or PLRV (Corsini and Brown 2001).

The Germplasm Resources Information Network (GRIN) of the US National Plant Germplasm System characterizes *Solanum etuberosum* Lindl., a wild potato species native to Chile, as having resistances to PVY, PLRV, potato virus X (PVX), and green peach and potato aphid. *S. etuberosum*, classified as a 1EBN species, does not cross readily with tetraploid or diploid clones of the cultivated potato and has been characterized as having an E-genome distinct from the A-genome of *S. tuberosum* (Matsubayashi 1991; Perez et al. 1999). Sexual barriers to hybridization with cultivated potato were overcome through the use of somatic hybridization (Novy and Helgeson 1994a; Thieme et al. 1999). High levels of resistance to PVY and PLRV were reported in somatic hybrids of *S. etuberosum* and their sexual progeny (Novy and Helgeson 1994b; Thieme et al. 1999; Gavrilenko et al. 2003). Resistances to PVY, PLRV, and green peach aphid, derived from *S. etuberosum*, also were identified in the BC_1 and BC_2 progeny of the somatic hybrids (Novy et al. 2002), indicating they were transmissible through generations of backcrossing to cultivated potato.

The objective of this study was to assess the stability of expression of PVY and PLRV resistances in progeny derived from *S. etuberosum* following multiple years of field evaluation in Idaho. An additional component was an evaluation of the inheritance of PVY and PLRV in the third sexual generation derived from the *S. etuberosum* progenitor, with an emphasis on elucidation of the possible mechanisms conferring PLRV resistance based on field and graft inoculation data.

Materials and methods

Plant materials

A diploid ($2n = 2x = 24$) *S. etuberosum* clone from PI 245939 was somatically hybridized with a S. *tuberosum* subsp. tuberosum haploid-wild species hybrid [US-W 730 × *S. berthaultii* (PI 265857)] (Novy and Helgeson 1994a). One BC_1 individual, P2-3 (somatic hybrid 2-9-3B × Atlantic), was again successfully crossed back to the *S. tuberosum* cultivar, Katahdin for the generation of the BC_2 progeny used in this study (Table 1). Clone P2-3 does not tuberize well under field conditions, limiting its use in field screening for virus infection. BC_1 clone, P2-4 (with adequate field tuberization), was used in its place and was shown to adequately portray the phenotypic response of P2-3 to virus infections based on genomic in situ hybridization (GISH) analysis which indicated P2-4 had a base set of 12 *S. etuberosum* chromosomes—analogous to the 11 *S. etuberosum* chromosomes identified in P2-3 (Dong et al. 1999). A high level of genetic homozygosity in self-fertile *S. etuberosum* (Spooner et al. 1992, 1996) also makes for a strong case that segregation for virus resistance alleles has not occurred. Therefore, P2-3 and P2-4 can be considered genetically identical for virus-resistance genes contributed by *S. etuberosum*. The parentages of two BC_3 families used in field and grafting screenings for PLRV resistance also are given in Table 1.

Field screening for virus resistances

Screening of germplasm for resistance to PVY and PLRV was conducted at Kimberly, Idaho. Plots of entries consisted of five hills replicated three times in a randomized complete block (RCB) design. Field testing consisted of the use of PVY/PLRV infected spreader rows interspersed among entry rows. PVY^{O} was the primary strain represented in the seed based on reverse transcription polymerase chain reaction (RT-PCR) analyses using primers developed for PVY strain identification. However, the presence of PVY^{N} and its rec-

Table 1 Description of breeding clones and cultivars used in field evaluations for infection response to PVY and PLRV, as well as PLRV grafting studies

Entry	Description	Parentage	Entry	
			Field	Grafting
16-1	Diploid parent of somatic hybrid	*S.etuberosum* (PI 245939)	No	Yes
463-4	Diploid parent of somatic hybrid	US-W730 × *S. berthaultii*	Yes	Yes
2-9-3B	Tetraploid somatic hybrid	463-4 + 16-1 (*S. etuberosum*)	No	Yes
Atlantic	Parent of BC_1 clone, P2-3	Wauseon × Lenape	Yes	Yes
P2-3	BC_1 of somatic hybrid	2-9-3B × Atlantic	No	Yes
P2-4	BC_1 of somatic hybrid	2-9-3B × Kathahdin	Yes	Yes
Katahdin	Parent of P2-4 and BC_2	USDA 40568 × USDA 24642	Yes	Yes
Etb 5-31-2	BC_2 of somatic hybrid	P2-3 × Katahdin	Yes	Yes
Etb 5-31-3	BC_2 of somatic hybrid	P2-3 × Katahdin	Yes	No
Etb 6-21-3	BC_2 of somatic hybrid	P2-3 × Katahdin	Yes	Yes
Etb 6-21-5	BC_2 of somatic hybrid	P2-3 × Katahdin	Yes	Yes
Etb 6-21-12	BC_2 of somatic hybrid	P2-3 × Katahdin	Yes	No
A92303-7	Parent of BC_3	A86332-7 × Ranger Russet	Yes	Yes
A86102-6	Parent of BC_3	A7532-1 × A8173-4	Yes	No
GemStar Russet	Parent of BC_3	Gem Russet × A8341-5	Yes	No
A00ETB12-1	BC_3 of somatic hybrid	A92303-7 × Etb6-21-3	Yes	No
A00ETB12-2	BC_3 of somatic hybrid	A92303-7 × Etb6-21-3	Yes	Yes
A00ETB12-3	BC_3 of somatic hybrid	A92303-7 × Etb6-21-3	Yes	Yes
A00ETB12-4	BC_3 of somatic hybrid	A92303-7 × Etb6-21-3	Yes	No
A00ETB11-1	BC_3 of somatic hybrid	A86102-6 × Etb6-21-3	Yes	No
A01687-1–37	BC_3 family of somatic hybrid	Etb 6-21-3 × GemStar Russet	Yes	No
A00ETB02-1–24	BC_3 family of somatic hybrid	Etb 5-31-3 × GemStar Russet	Yes	No
Russet Burbank	PVY and PLRV susceptible control	Sport of Burbank	Yes	Yes
Ranger Russet	PVY resistant and PLRV susceptible control	Butte × A6595-3	Yes	No
Liu	PVY and PLRV resistant control	II 70 4154 × I 65 751 132	Yes	Yes

ombinants cannot be totally discounted in the field evaluation. Spreader rows provided virus inoculum for dispersion by native aphid populations. Manual inoculations of PVY, using macerated leaf tissue from spreader rows verified as PVY-infected using DAS-ELISA, also were conducted to ensure a rigorous test of germplasm. Details of this field screening protocol are described in Corsini et al. (1994).

Ten tubers from each plot of the field trial were harvested and a progeny plant from each tuber was grown in the greenhouse. Tuber-progeny plants were then assayed for PVY and PLRV using DAS-ELISA. Monoclonal PVY antiserum used in the assay detects O, C, and N strains of PVY (Scottish Agriculture Science Agency [SASA®] Edinburgh, Scotland) ensuring a stringent assay; PLRV antiserum was obtained from BioReba, Ag® (Reinach, Switzerland). Assayed plants (and associated tubers) were considered infected if the following two absorbance criteria at 405 nm were met: (1) absorbance value was greater than the mean of the non-infected control plus four times its standard deviation, and (2) the value was greater than 0.100. On this basis, percentages of infected tuber-progeny plants for each entry in each replication were obtained. Statistical analyses were conducted using JMP® Software (SAS, Cary, NC, USA).

PLRV grafting study

Plant material, protocol, and design

Plant materials used in the PLRV grafting study are shown in Table 1. The *S. etuberosum* somatic hybrid parent, 16-1, was originally included in the evaluation but difficulties in maintaining viable plants during the course of the study precluded the collection of data. BC_2 clones, Etb 5-31-2, and Etb 6-21-3, were included as PLRV resistant clones based on 4 years of field evaluation in Idaho, and in the case of Etb 6-21-3, in field and cage evaluations in Minnesota (Novy et al. 2002). Etb 6-21-5, although identified as PLRV resistant in Minnesota (Novy et al. 2002), was not as resistant to PLRV in Idaho, but nonetheless was classified as having moderate PLRV resistance. In addition, two BC_3 clones, A00ETB12-2 and A00ETB12-3, identified as PLRV resistant in 2 years of field trials in Idaho also were included.

Seed tubers of most entries were obtained from a non-virus inoculated field trial in Aberdeen, Idaho. However, tubers of the BC_2 entries Etb 5-31-2, Etb 6-21-3, and Etb 6-21-5 were from the virus-field study described previously, that were found to be uninfected following their assay for PLRV, PVY, and PVX using

DAS-ELISA. Uninfected tubers were warmed at room temperature until initial sprouting of eyes occurred. If little tuber dormancy was observed, tubers were placed back into cold storage until tubers of clones with longer tuber dormancy had sprouted. This was done to ensure a more even emergence of plants for use in grafting following planting of seed pieces in the greenhouse. Entries P2-3 and 2-9-3B, with limited tuberization in the field, were derived from tissue cultured plantlets.

Potato leafroll virus infected tubers of 'Ranger Russet,' 'Torridon,' and three breeding clones were obtained from a field trial planted in Idaho in 2003. Before use as the PLRV-infected scion in the grafting study, fully mature leaves from two stems of each plant were tested for PLRV, PVY, and PVX using a DAS-ELISA test. Plants that were positive for PLRV and negative for PVY and PVX were used as scions to inoculate plants of the breeding clones and cultivars identified in the grafting study in Table 1. Prior to grafting with the PLRV-infected scions, plants to be used as the rootstock were screened for PLRV, PVY, and PVX using a DAS-ELISA test. No plants were identified as infected with PLRV or PVX, however, PVY infection was noted in some entries.

Grafting protocol was as described by Barker and Harrison (1985), with additional detailed instruction on the grafting technique provide by Ching-Pa Yang and Charles Brown, USDA-ARS, Prosser, WA (personal communication). Rootstock, scion, and grafted plants were all grown in the greenhouse. With few exceptions, ten plants per entry were grafted with PLRV-infected scions; excluding test plants that died due to poor greenhouse growth, etc., 91% of the completed grafts were successful 28 days after grafting.

Design was a RCB with two replicates. Each entry was represented by a maximum of five plants per block. Plants did not grow at the same rate despite previously described efforts, therefore test plants were divided based on size and the five largest plants per entry were identified and grafted. This replicate was labeled Block A. Block B, consisting of the next five largest plants was subsequently grafted ~4 days later to ensure plant size at grafting was comparable among the two replicates. The differences in mean size of the rootstocks (all were at least 10 cm when grafted) among the blocks and the differences in the weather conditions between these two time periods were not expected to be significant in terms of virus development. Success rate of grafts after 10 days was similar among the two blocks.

Tubers were harvested from each plant in the graft experiment including the negative controls ~106–110 days after grafting. Tubers were then placed into cold storage and in the spring of 2005 scooped eyes were planted in the greenhouse in individual 3–4 in. pots. The emerged plants were arranged in the greenhouse in a completely random design. The number of tuber-progeny plants tested varied across entries because not all parent clones survived the graft screen and not all tuber-progeny plants survived for subsequent PLRV testing.

Test of graft inoculated plants and progeny tubers for PLRV

DAS-ELISA absorbance values for PLRV in foliage were determined 28 and 50 days after grafting in 2004; PVY and PVX titers also were determined concurrently with the PLRV assessment. Tuber-progeny plants of these plants were then tested in the spring of 2005 when emerged plants were 4–6 in. tall using the BioReba kit for DAS-ELISA. For the grafted plants, fully expanded leaves were taken from stems that originated just below the graft union and, if available, the fourth or fifth leaf from the shoot apex was used. For the tuber-progeny plants any fully expanded leaves just below the growing point were used. The leaves were crushed using a plant press to express the sap. Two hundred μl of sap was diluted in 750 μl of Blotto buffer and 200 μl of the dilution was used for coating the microplate (NUNC-Immuno^{TM} Plate, MaxiSorp^{TM} Surface, part no WC449824, Nalge Nunc International, Portsmouth, NH, USA). For the grafting study only, PLRV readings for two wells per sample were taken, for all other readings only one well was used per sample. Extracted sap from leaf samples were used for DAS-ELISA on the same day that they were collected. Each plate contained wells with (1) sample buffer only, (2) PLRV infected potato leaf sample, (3) PVY infected potato leaf sample, and (4) PVX infected potato leaf sample. The plates were read after developing for 30 min after the addition of the substrate, except for PLRV readings for the grafting screen, which were recorded after 40 min as this gave better separation of absorbance values. The plates were read at 405 nm using the Kinetic Reader Model EL312E by BIO-TEK Instruments Inc. (Highland Park, Box 998 Winooski, VT 05404-0998, USA) which was programmed to subtract the value of a blank well (sample buffer only) from the absorbance values of the other wells.

Data analysis of grafted plants

DAS-ELISA absorbance values for PLRV were averaged over the subsamples. DAS-ELISA values for PLRV on a plot mean basis were used for statistical analysis. Transformations were used to stabilize the variances prior to analysis of variance in all cases.

Analysis of variance was completed using SAS PROC GLM. A two-way model was fit of Entry × Block. The effect of prior PVY infection on the induced PLRV infection was tested for the entries, which had this problem. PVY status of the test plants prior to grafting was analyzed as categorical variables (Yes/No) because a number of plants had off scale readings a two-way model was fit of Entry and PVY × block. The effect of source of PLRV inoculum, i.e., the genotype of the scion, was tested. The two-way model was fit to Entry and Scion × Block using SAS PROC GLM. Descriptive statistics were calculated using untransformed values using SAS PROC MEANS.

Data analysis of progeny tubers

DAS-ELISA values of the tuber-progeny plants were analyzed on an entry mean basis. The percentages of infected tubers were calculated for an entry based on plus/minus screening in which all tubers with A_{405} over 0.100 were considered to be infected. Given that block effects were found to be not significant in the original parental grafting screen, the block from which the tubers came was not considered as part of the analysis. Spearman's rank correlation (SAS PROC CORR) was used to test the association between the parent and the progeny generation.

Results

Field screening for virus resistance

Evaluation of BC_2 for PLRV resistance

The percentages of virus-infected tuber-progeny plants of BC_2 clones exposed to PLRV and PVY during 4 years of field evaluations are presented in Tables 2 and 3. Two BC_2 clones, Etb 6-21-3 and Etb 5-31-2, as well as BC_1 clone, P2-4, had significantly lower percentages of PLRV-infected tuber-progeny plants than other entries (Table 2). The remaining BC_2 entries had levels of PLRV-infected tuber-progeny plants intermediate to those observed for the previously described resistant clones and PLRV susceptible, Russet Burbank. Somatic hybrid parent, 463-4, was classified as susceptible to PLRV, with percentages of PLRV-infected tuber-progeny plants most similar to values observed for PLRV susceptible Russet Burbank.

Evaluation of BC_2 for PVY resistance

The highest level of PVY resistance was observed in BC_2 clone, Etb 5-31-3, which had significantly lower levels of PVY than all other entries (Table 3). Similar to observations for PLRV infection, the remaining BC_2 clones, while not as highly resistant to PVY as Etb 5-31-3, were nonetheless significantly more resistant than PVY susceptible Russet Burbank. Somatic hybrid parent, 463-4, had a level of PVY resistance interme-

Table 2 Percentage of PLRV-infected tuber-progeny plants of somatic hybrid parent 463-4, BC_1 clone P2-4, five BC_2 clones (boldface), and cultivar checks

Clone	% PLRV positive[a]	
	2001–2004	2002–2004
Etb 6-21-3	3a	2a
Etb 5-31-2	8a	5a
Etb 6-21-5	41b	45bc
Etb 5-31-3	53b	55bc
Etb 6-21-12	73c	69cd
Ranger Russet	78c	83de
Russet Burbank	98d	100e
463-4	–[b]	95e
P2-4	–	10a
Atlantic	–	59bc
Katahdin	–	65bcd

Atlantic and Katahdin are in the pedigrees of the BC_2 clones. Field evaluations were conducted at Kimberly, Idaho with PLRV infection verified using DAS-ELISA

[a] Values followed by the same letter are not significantly different at the 5% level of significance using the least significant difference (LSD) test

[b] Dash indicates not represented in every year of that time period

Table 3 Percentage of PVY-infected tuber-progeny plants of somatic hybrid parent 463-4, BC_1 clone P2-4, five BC_2 clones (boldface), and cultivar checks

	% PVY positive[a]	
	2001–2004	2002–2004
Etb 5-31-3	3a	0a
Etb 6-21-12	28b	27bcd
Etb 6-21-5	49cd	43cde
Etb 6-21-3	52d	49de
Etb 5-31-2	59d	58e
Ranger Russet	32bc	26bc
Russet Burbank	94e	96f
463-4	–[b]	54e
P2-4	–	20ab
Atlantic	–	64e
Katahdin	–	17ab

Atlantic and Katahdin are in the pedigrees of the BC_2 clones. Field evaluations were conducted at Kimberly, Idaho with PVY infection verified using DAS-ELISA

[a] Values followed by the same letter are not significantly different at the 5% level of significance using the least significant difference (LSD) test

[b] Dash indicates not represented in every year of that time period

diate to that observed in Etb 5-31-3 and Russet Burbank. BC_1 clone, P2-4, and cultivar parent, Kathadin were classified as resistant to PVY with percentages of infected tuber-progeny plants not statistically different from observations for Etb 5-31-3.

Evaluation of BC_3 for PLRV resistance

Five BC_3 clones derived from PLRV resistant Etb 6-21-3 were evaluated in 2003 and 2004. Two clones were identified as being as resistant to PLRV as their Etb 6-21-3 parent, while the remaining three clones were as susceptible as Russet Burbank (Table 4). PLRV-resistant clones A00Etb12-2 and 12-3 were again tested for an additional year in 2005, with no breakdown of resistance.

In 2004, a population of 37 BC_3 progeny derived from the intercrossing of Etb 6-21-3 with the PLRV susceptible cultivar, GemStar Russet (family A01687), was assessed in the field for PLRV resistance. In addition, the five BC_3 clones previously evaluated in 2003 and 2004 were included in the study. A total of 42 BC_3 progeny of Etb 6-21-3 were evaluated in 2004 and found to segregate for resistance to PLRV (Fig. 1). On the basis of an LSD value of 41 calculated at the 5% level of significance, 19 BC_3 clones (45%) were found to have percentages of PLRV-infected tuber-progeny plants that did not differ statistically from Etb 6-21-3 (No infected tuber-progeny). Two resistant clones, A01687-11 and -26, were again evaluated in 2005, with a similar resistant response to PLRV infection in tuber-progeny plants (Table 4), again confirming that PLRV resistance in the BC_3 was stably expressed over years of evaluation.

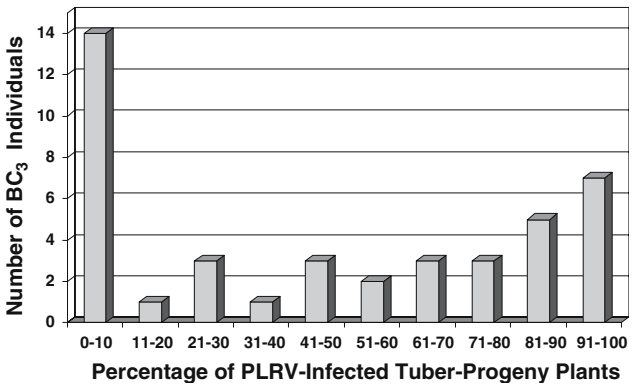

Fig. 1 Distribution of 42 BC_3 individuals for percentages of tuber-progeny plants infected with PLRV following field evaluation at Kimberly Idaho, 2004. The BC_3 individuals were derived from PLRV resistant clone Etb 6-21-3, which had no infected tuber-progeny plants in this evaluation. The PLRV-susceptible parents of the BC_3 ranged from 67 to 90% PLRV-infected, tuber-progeny plants in the field evaluation. The PLRV-susceptible, control cultivar, Russet Burbank, had 100% infected tuber-progeny, whereas PLRV-resistant, control cultivar, Liu, had no infected tuber progeny

Table 4 Percentage of PLRV-infected tuber-progeny plants in seven BC_3 clones (boldface), their parents (italicized), and cultivar standards following 2 or 3 years of field testing at Kimberly, Idaho

Clone	% PLRV positive[a]		
	2003–2004	2003–2005	2004–2005
Etb 6-21-3	0a	0a	0a
A00Etb12-3	0a	0a	0a
A00Etb12-2	5a	3a	5ab
A00Etb12-4	87bc	–[b]	–
A00Etb11-1	90bc	–	–
A00Etb12-1	100c	–	–
A01687-11	–	–	20b
A01687-26	–	–	12ab
Gemstar Russet	–	–	67 (04 only)
A86102-6	78b	–	–
A92303-7	95c	–	–
Ranger Russet	75b	–	–
Russet Burbank	100c	100b	99c

Infection was verified using DAS-ELISA

[a] Values followed by the same letter are not significantly different at the 5% level of significance using the least significant difference (LSD) test

[b] Dash indicates not represented in every year of that time period

Segregation for PLRV resistance appeared to most closely fit a genetic model in which PLRV resistance was conferred by a dominant allele. Under a genetic model in which cultivated potato has an analogous locus with recessive alleles for susceptibility to PLRV, Etb 6-21-3 would have a simplex genotype (i.e., Aaaa). Conversely, Etb 6-21-3 could be considered hemizygous for the resistance allele, if cultivated potato has no locus corresponding to that found in the E-genome of *S. etuberosum*. In either scenario, a 1:1 segregation would be expected. A chi-square test using Excel software did not reject the 1:1 segregation model with a calculated *P*-value of 0.44.

The PLRV resistance of Etb 6-21-3 was not confounded in the BC_3 progeny by its hybridization with PLRV resistant, advanced breeding clones and cultivars as parents. All such parental clones used in the synthesis of the BC_3 generation had 67–90% PLRV-infected, tuber-progeny progeny, and differed significantly from Etb 6-21-3 in percentages of PLRV infected tubers at the 5 and 1% levels of significance.

Evaluation of BC_3 for PVY resistance

Twenty-four BC_3 individuals derived from the intercrossing of PVY resistant Etb 5-31-3 with GemStar

Russet (family A00ETB02) were assessed in the field in 2005 for resistance to PVY. Unlike the observation that a high percentage of BC_3 expressed PLRV resistance, the PVY resistance of Etb 5-31-3 was not observed in any of its progeny. The percentage of infected tuber-progeny plants in the BC_3 ranged from 57 to 100%, with 75% of progeny having >90% PVY-infected tubers. These findings were in stark contrast to the 3% PVY-infected tubers observed in parent Etb 5-31-3 following 4 years of field evaluations.

PLRV grafting study

Graft-inoculated plants

Graft inoculations with PLRV infected scions were successful with all entries except the *S. etuberosum* fusion hybrid parent, 16-1, in which all individual plants of this clone died before data could be collected. Clone 16-1 has been identified as being sensitive to soil moisture variations in the greenhouse relative to other potato clones. The deaths of 16-1 plants in this grafting study were thought to relate to its soil moisture sensitivities and not to the grafting procedure.

Large differences in PLRV concentrations in leaves of graft-inoculated plants were observed among genotypes (Tables 5, 6). The recorded DAS-ELISA absorbance values for each clone at 28 and 50 days following grafting of the PLRV infected scion are given in Table 5. With a few exceptions, the mean absorbance values, which give an estimate of PLRV titers, did not vary greatly at the two timepoints. However, a trend was observed for PLRV to decrease over time in entries with the lowest viral concentrations and for PLRV to increase in more susceptible entries. Viral load at day 50 gave clearer separation of the entries and seemed to better represent the resistance responses of the genotypes.

Somatic hybrid 2-9-3B had very low-PLRV absorbance values (0.0239 and 0.0166) at both 28 and 50 days (Table 5)—not significantly different from values observed for the non-infected control (Table 6). Conversely, 463-4, its fusion parent, had absorbance values that ranged from 0.6497 to 0.6503 (Table 5). This finding supports *S. etuberosum* as the source of resistance to PLRV observed in the somatic hybrid and its sexual progeny. With the exceptions of A00ETB12-2 and Etb 6-21-5, all *S. etuberosum*-derived entries had significantly smaller absorbance values 50 days following graft inoculations than did the PLRV susceptible cultivars and advanced potato selections (Table 6).

Etb 6-21-5, classified as having moderate resistance to PLRV in field trials (Table 2), had statistically significant lower percentages of PLRV-infected, tuber progenies than did Russet Burbank. However, this level of resistance was not observed in the grafting study, where PLRV titers, as reflected in absorbance values did not differ significantly from those of Russet Burbank (Table 6). This discrepancy between mean accu-

Table 5 Mean DAS-ELISA absorbance values at 405 nm for Potato leafroll virus (PLRV) generated by sampling fully expanded leaves from below the graft union of graft inoculated entries

Entry	Entry background	Sample size	Time post-inoculation			
			Day 28		Day 50	
		Day 28/Day 50	Mean	SD	Mean	SD
Negative control	NA	28/26	0.0009	0.0036	0.0007	0.0073
2-9-3B	Somatic hybrid	4/4	0.0239	0.0182	0.0166	0.0090
P2-4	BC_1	9/9	0.0987	0.1012	0.3473	0.1825
Etb 6-21-3	BC_2	8/8	0.1104	0.0737	0.0911	0.1524
P2-3	BC_1	8/8	0.1179	0.1146	0.1345	0.1050
Liu	Resistant control	9/9	0.1539	0.1239	0.0548	0.0480
A00ETB12-3	BC_3	10/10	0.3621	0.2782	0.3347	0.3012
Etb 5-31-2	BC_2	9/8	0.3923	0.2137	0.2894	0.1653
Katahdin	Parent clone	9/9	0.4000	0.2866	0.6556	0.2749
A00ETB12-2	BC_3	9/9	0.6128	0.3603	0.6737	0.2364
463-4	Somatic hybrid parent	9/9	0.6497	0.1966	0.6503	0.2779
Atlantic	Parent clone	9/9	0.8026	0.2113	0.8515	0.1857
Etb 6-21-5	BC_2	8/8	0.8287	0.1872	0.7656	0.1534
A92303-7	Parent clone	9/9	0.8397	0.1926	1.1108	0.1260
Russet Burbank	Susceptible control	9/9	0.8436	0.3516	0.9201	0.2552

These are the actual values that were then transformed for statistical analysis (Table 6). The sample size is the total number of plants that were analyzed across blocks per entry on the given sample date. The negative control consisted of a pool of single ungrafted plants from most of the entries

Table 6 Analysis of mean accumulation of PLRV in the graft inoculation screen

Entry	Entry background	Day 28	Entry	Day 50
Negative control	NA	0.0263a	Negative control	0.0317a
2-9-3B	Somatic hybrid	0.1525ab	2-9-3B	0.1325ab
P2-4	BC_1	0.3010b	Liu	0.2373bc
Etb 6-21-3	BC_2	0.3284b	P2-3	0.3670cd
P2-3	BC_1	0.3372b	Etb 6-21-3	0.4322de
Liu	Resistant control	0.3943bc	Etb 5-31-2	0.5279e
A00ETB12-3	BC_3	0.5958cd	A00ETB12-3	0.5665e
Etb 5-31-2	BC_2	0.6118cd	P2-4	0.5837e
Katahdin	Parent clone	0.6127cd	Katahdin	0.7992f
A00ETB12-2	BC_3	0.7817de	463-4	0.8007f
463-4	Somatic hybrid parent	0.8069de	A00ETB12-2	0.8265fg
Atlantic	Parent clone	0.9011e	Etb 6-21-5	0.8888fg
A923037	Parent clone	0.9218e	Atlantic	0.9286fgh
Russet Burbank	Susceptible control	0.9234e	Russet Burbank	0.9650gh
Etb 6-21-5	BC_2	0.9238e	A923037	1.0575h

The least square means of the transformed A_{405}-values for 28 days after inoculation and 50 days after inoculation. Control is the non-inoculated negative control. The ranking of the entries changed slightly from 28 to 50 days, but generally the ranking was the same. Means with the same letter after them are not significantly different as determined by Fisher's protected LSD at $\alpha = 0.05$

mulation of PLRV following graft inoculation and percentage of infected tuber-progeny plants in field trials is especially glaring for BC_3 clone, A00ETB12-2. In 3 years of field trials, its percentage of PLRV-infected tubers was not significantly different from that of its PLRV-resistant parent, Etb 6-21-3 (Table 4).

Evaluation of tuber-progeny plants of PLRV grafted plants

The percentage of infected tuber-progeny plants and the mean absorbance values of tuber-progeny plants derived from graft-inoculated plants are presented in Table 7. For comparison purposes, the absorbance values of the parental plants at 50 days following graft inoculation are also included in the table.

An assessment of the relationship between the mean absorbance values of parent clones at day 50 and the mean absorbance value of their tuber-progeny populations was conducted using Spearman's rank correlation. A high correlation was found [$r_s = 0.78$ ($P = 0.003$)], indicating parental virus titers were closely aligned with values observed in tuber-progeny populations. Comparisons between mean absorbance values of the parent clone and the percentage of infected tuber-progeny plants [$r_s = 0.64$ ($P = 0.03$)] while still highly significant, was not as robust. Comparison between DAS-ELISA absorbance values of tuber-

Table 7 Evaluation of tuber-progeny plants of graft-inoculated entries for PLRV infection and their comparison with the PLRV absorbance values of their parental plants

Tuber-progeny plants						Parental plants—Day 50		
Entry	Entry background	Sample size	Mean	SD	Percent susceptible ($A_{405} > 0.1$)	Sample size	Mean	SD
Negative control	NA	25	–0.0119	0.00651	0	26	0.001	0.007
Etb 6-21-3	BC_2	19	0.244	0.302	47	8	0.091	0.152
Etb 5-31-2	BC_2	26	0.436	0.362	69	8	0.289	0.165
Liu	Resistant control	16	0.489	0.478	56	9	0.055	0.048
P2-4	BC_1	21	0.597	0.341	81	9	0.347	0.183
A00ETB12-3	BC_3	28	0.635	0.498	68	10	0.335	0.301
A00ETB12-2	BC_3	22	0.727	0.390	82	9	0.674	0.236
Russet Burbank	Susceptible control	21	0.756	0.453	76	9	0.920	0.255
Etb 6-21-5	BC_2	13	0.797	0.127	100	8	0.766	0.153
Atlantic	Parent clone	23	0.828	0.527	78	9	0.852	0.186
Katahdin	Parent clone	21	0.866	0.509	81	9	0.656	0.275
463-4	Somatic hybrid parent	23	0.928	0.273	100	9	0.650	0.278
A92303-7	Parent clone	20	1.220	0.312	95	9	1.111	0.126
2-9-3B	Somatic hybrid	NA	NA	NA	NA	4	0.017	0.009
P2-3	BC_1	NA	NA	NA	NA	8	0.135	0.105

progeny plants and percentage of PLRV infected tuber-progeny plants also was highly correlated [r_s = 0.77 (P = 0.004)].

Discussion

High levels of resistance to both PVY and PLRV were observed in *S. etuberosum*-derived germplasm over multiple years of field evaluations in Idaho, demonstrating the stability of expression of virus resistances derived from *S. etuberosum*. Transmission of PLRV to tuber-progeny plants of graft inoculated plants was found to be higher than the transmission observed in the field evaluations (Table 7). Flis et al. (2005) made similar observations that graft-inoculation of PLRV generally resulted in a greater percentage of PLRV infected tubers than did PLRV vectored by green peach aphid (a primary vector of PLRV in the field evaluations). However, while the relative magnitude of PLRV transmission increased in the grafting evaluations, the overall ranking of clones did not. Clones identified as resistant in field studies (with the exceptions of A00ETB12-2 and Etb 6-21-5) also showing a statistically lower accumulation of PLRV in foliage at day 50 relative to susceptible entries (Table 6).

The data presented corroborates previous findings of PVY and PLRV resistances in BC_2 progeny evaluated in a field screening and field cage trial conducted in Minnesota in 1999 (Novy et al. 2002). The protocols used in the screening of *S. etuberosum*-derived germplasm in the field in Minnesota and in Idaho are very similar, allowing comparisons between the two studies (Tables 8, 9). The major protocol differences between the two studies were: (1) the Minnesota trial used transplanted plants that were derived from tubers planted in the greenhouse, whereas the Idaho trials used planted seed pieces, and (2) the Idaho field evaluations incorporated the mechanical inoculation of emerged plants with PVY in addition to aphid-mediated infection; mechanical inoculation with PVY was not conducted in Minnesota.

Table 8 Percentage of PLRV-infected tuber-progeny plants of five BC_2 clones, and the susceptible cultivar check Russet Burbank in field trials conducted in Minnesota in 1999 (Novy et al. 2002) and in Idaho (2001–2004)

Clone	% PLRV positive[a]	
	Idaho	Minnesota
Etb 6-21-3	3a	13a
Etb 5-31-2	8a	70bc
Etb 6-21-5	41b	34ab
Etb 5-31-3	53b	–[b]
Etb 6-21-12	73c	93c
Russet Burbank	98d	87c

PLRV infection was verified using DAS-ELISA

[a] Values followed by the same letter are not significantly different at the 5% level of significance using the least significant difference (LSD) test (ID) or Duncan's Multiple Range Test (MN)

[b] Dash indicates not represented in that evaluation

Table 9 Percentage of PVY-infected tuber-progeny plants of five BC_2 clones, and the susceptible cultivar check Russet Burbank in field trials conducted in Minnesota in 1999 (Novy et al. 2002) and in Idaho (2001–2004)

Clone	% PVY positive[a]	
	Idaho	Minnesota
Etb 5-31-3	3a	–[b]
Etb 6-21-12	28b	28a
Etb 6-21-5	49cd	4a
Etb 6-21-3	52d	8a
Etb 5-31-2	59d	13a
Russet Burbank	94e	82b

PVY infection was verified using DAS-ELISA

[a] Values followed by the same letter are not significantly different at the 5% level of significance using the least significant difference (LSD) test (ID) or Duncan's Multiple Range Test (MN)

[b] Dash indicates not represented in that evaluation

PLRV resistance

The percentages of PLRV infected tuber-progeny plants among BC_2 clones were similar at Idaho and Minnesota, with the exception of clone Etb 5-31-2 (Table 8). Noted as susceptible to PLRV infection in the field evaluation at Minnesota, it displayed a low percentage of infected tuber-progeny plants in Idaho and would be classified as resistant. Contributing factors to the observed disparity in the response of Etb 5-31-2 to PLRV infection at the two sites are not fully evident, but could relate to differences in virulence between Idaho and Minnesota PLRV isolates. Flis et al. (2005) reported differences in resistance response among PLRV resistant clones when challenged with two different PLRV isolates and theorized they might be different strains of PLRV.

If differences in virulence exist among Idaho and Minnesota PLRV isolates, then Etb 6-21-3 which had consistent resistant responses to PLRV at both sites may have additional resistance mechanism(s) not present in Etb 5-31-2. Mechanisms of PLRV resistance previously identified in the literature that are possibly contributing to the observed PLRV resistance of Etb 6-21-3 include: (1) resistance to infection, (2) resistance

to accumulation, and (3) resistance to PLRV movement (Solomon-Blackburn and Barker 2001;Thieme and Thieme 2005).

Solanum etuberosum and closely related *S. palustre* (formerly classified as *S. brevidens*) have been identified as having resistance to PLRV accumulation (Jones 1979; Chavez et al. 1988; Gibson et al. 1988). It is likely that this mechanism of resistance also is contributing to the PLRV resistance observed in the *etuberosum*-derived germplasm used in this study. This statement is supported by observations of a low percentage (0–20%) of infected *etuberosum*-derived BC_2 plants following aphid vectored transmission of PLRV in field and cage evaluations relative to the susceptible cultivar, Russet Burbank (67–100%) (Novy et al. 2002); only one exception was seen in BC_2 clone Etb 5-31-4 which had 63% infection of plants in the field evaluation, and likely had not inherited resistance to PLRV accumulation in the foliage. However, subsequent DAS-ELISA testing of progeny plants grown from harvested tubers of the BC_2 showed no significant differences in secondary infection relative to Russet Burbank with infection percentages of 64–92% (Novy et al. 2002)—indicating resistance to accumulation of PLRV in the foliage did not result in a significant decrease in infected tuber-progeny plants under high inoculum and aphid densities. The exceptions to this finding being BC_2 clones Etb 6-21-3 and Etb 6-21-5 which had significantly lower percentages of infected tuber-progeny plants than susceptible Russet Burbank. The lower percentages of infected progeny plants observed in these two clones suggest they may have an additional resistance mechanism in combination with resistance to PLRV accumulation in the foliage.

Novy et al. (2002) reported that resistance to infection, due to aphid resistance, did not confer resistance to PLRV in the BC_2 clones used in this study. Instead, resistance to PLRV accumulation in foliage does appear to contribute (Novy et al. 2002). The significantly lower percentage of infected plants grown from progeny tubers in this study and that of Novy et al. (2002) suggests Etb 6-21-3 also may have an additional resistance mechanism limiting PLRV movement to the tuber—a mechanism which was effective against both Minnesota and Idaho isolates of PLRV. This resistance mechanism also appears evident in the PLRV grafting study, whereby Etb 6-21-3 had the lowest percentage of infected progeny plants among entries (Table 7). Resistance to PLRV movement to developing tubers may also explain the anomaly of why BC_3 clone, A00ETB12-2, with high-PLRV titer following grafting inoculations (Table 6), had consistently low percentages of infected tuber progeny plants in 3 years of field evaluations (Table 4).

Resistance to the movement of PLRV has previously been documented in potato (Barker 1987; Wilson and Jones 1992; Derrick and Barker 1997). A model suggested by Derrick and Barker (1997) for resistance to PLRV movement in potato proposes that PLRV movement is impeded in the short distance movement from phloem sieve elements into companion cells with the subsequent hampering of virus multiplication in the plant. The authors found that long distance movement of the initial inoculum through phloem sieve elements occurred at the same rate in both resistant and susceptible potato genotypes.

The progeny derived from Etb 6-21-3 segregated for resistance to PLRV with distribution being skewed toward a resistant response (Fig. 1). The stringency of this field evaluation for PLRV infection response was evident in the high-infection rate (67–100%) of the susceptible potato clones and varieties included in the same study as the BC_3. The high percentage (45%) of BC_3 progeny with resistance statistically similar to their PLRV resistant parent, Etb 6-21-3, fits a genetic model in which PLRV resistance is dominant with Etb 6-21-3 having a simplex genotype or being hemizygous. This statement also is backed by molecular analyses conducted in our program, which have localized PLRV resistance to one genomic region (Gillen and Novy, in press). The findings of our research suggest this resistance is likely associated with reduced systemic spread of PLRV with a subsequent reduction in infected tuber-progeny plants. Evidence of a major gene for PLRV resistance confirms an earlier observation by Chavez et al. (1988) that PLRV resistance in *S. etuberosum* appeared to be under simple genetic control. However, the mechanism of resistance was identified by the authors as resistance to PLRV titer buildup in foliage. Confirmation that resistance may also be conferred by reduced systemic spread was not possible in that no testing of progeny tubers for PLRV infection was conducted (Chavez et al. 1988).

PVY resistance

Comparisons of percentages of PVY infected tuber-progeny plants from Idaho and Minnesota show similar statistical inferences with all BC_2 having significantly less PVY infected tubers than PVY susceptible Russet Burbank at both sites (Table 9). However, the Idaho site had a much higher percentage of PVY infected tubers than did Minnesota, likely relating to the incorporation of mechanical inoculation of PVY at Idaho and not at Minnesota. Etb 5-31-3, which was not

an entry in the Minnesota evaluations, was statistically the most resistant entry among the BC_2 clones evaluated in Idaho (Table 3).

The low percentage of PVY-infected tuber progeny plants of the cultivar Katahdin (Table 3) was not expected. Kathadin had been noted in its release manuscript as having resistance to mild mosaic (Clark 1931), which is thought to indicate it had resistance to current season infection by PVA and possibly PVX (Dr. Joe Pavek, personal communication); screening for PVX resistance by our program has shown Katahdin to be susceptible to PVX infection, so it is likely the authors were referring to PVA resistance. No mention is made of resistance to PVY by Clark (1931). In subsequent evaluations, a moderate level of foliar resistance was observed following mechanical inoculation of foliage with PVY^o, but Katahdin was still found to be statistically more susceptible to PVY infection of the foliage than the *S. etuberosum* fusion parent, 16-1, its tetraploid somatic hybrids, and three of five BC1 progeny—indicative that *S. etuberosum* was contributing a higher level of PVY resistance than observed in Katahdin (Novy and Helgeson 1994b). It was initially thought that Katahdin was not highly resistant to PVY and therefore, as a parent in the pedigree of the *S. etuberosum*-derived germplasm, would not confound subsequent evaluations of the germplasm for resistance to PVY. Results shown in Table 3, indicate that Katahdin, with its unexpectedly low percentages of PVY-infected progeny tuber plants, may have resistance to the transmission of PVY from foliage to tuber, and therefore cannot be excluded as contributing to the PVY resistance observed in Etb 5-31-3. However, the consistently lower percentages of PVY-infected tuber progeny plants in Etb 5-31-3 relative to Katahdin over 3 years of field evaluations suggest *S. etuberosum* is likely the primary contributor to the PVY resistance of Etb 5-31-3.

Surprisingly, the high level of PVY resistance observed in two generations of progeny derived from the *S. etuberosum* somatic hybrid were lost by the third backcross generation. No BC_3 individual derived from PVY resistant BC_2 clone, Etb 5-31-3, had less than 50% PVY infected tuber-progeny plants, with 75% of the progeny having ≥90% infected tubers. These results suggest the preferential elimination of genomic regions of *S. etuberosum* associated with PVY resistance by the third sexual generation. This finding is in stark contrast to observations for PLRV resistance, where a large percentage of BC_3 progeny exhibited high levels of PLRV resistance (Fig. 1). Differences in transmission likely relate to structural differences between the E and A genomes of *S. etuberosum* and cultivated potato (Perez et al. 1999).

In summary, high levels of resistance to both PVY and PLRV were observed in progeny derived from *S. etuberosum*. Following the third generation of backcrossing to cultivated potato, PLRV resistance was effectively transmitted whereas PVY resistance was lost, confirming previous reports that the two virus resistances are apparently conferred by different genes in species of section *Etuberosum* (Valkonen et al. 1994; Novy et al. 2002). Observed disparities in transmission of the two virus resistances are thought to relate to structural differences between the E and A-genomes of *S. etuberosum* and cultivated potato, respectively. PLRV resistance appears to be monogenic based on the inheritance of resistance in a BC_3 population. Molecular analyses conducted in our program, also supports a monogenic form of resistance with one chromosome region of *S. etuberosum* identified as being associated with resistance to PLRV. On the basis of field and grafting studies, it is postulated that resistance to PLRV accumulation, which has been previously confirmed in section *Etuberosum*, also is contributing to the PLRV resistance observed in our germplasm. However, it also appears that a major component of the observed PLRV resistance relates to the inhibition of the systemic spread of PLRV from infected foliage to tubers. Our program is currently working to identify markers closely associated with this mechanism of resistance that can be used for marker-assisted selection in potato breeding programs.

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References

Alvarez JM, Srinivasan R (2005) Evaluation of hairy nightshade as an inoculum source for the aphid-mediated transmission of potato leafroll virus. J Econ Entomol 98:1101–1108

Bantarri EE, Ellis PJ, Khurana SMP (1993) Management of diseases caused by viruses and virus-like pathogens. In: Rowe RC (ed) Potato health management. American Phytopathological Society (APS) Press, St. Paul, Minnesota, pp 127–133

Barker H (1987) Multiple components of the resistance of potatoes to potato leafroll virus. Ann Appl Biol 111:641–648

Barker H, Harrison BD (1985) Restricted multiplication of potato leafroll virus in resistant potato genotypes. Ann Appl Biol 107:205–212

Chavez R, Brown CR, Iwanaga M (1988) Transfer of resistance to PLRV titer buildup from *Solanum etuberosum* to a tuber-bearing *Solanum* gene pool. Theor Appl Genet 76:129–135

Clark CF (1931) The Katahdin potato; A new variety. Am Potato J 8:121–125

Corsini DL, Brown CR (2001) Important potato cultivars. In: Loebenstein G, Berger PH, Brunt AA, Lawson RH (eds) Virus and virus-like diseases of potatoes and production of seed-potatoes. Kluwer Academic Publishers, Dordrecht, The Netherlands, pp 39–52

Corsini DL, Pavek JJ, Martin MW, Brown CR (1994) Potato germplasm with combined resistance to leafroll virus and viruses X and Y. Am Potato J 71:377–385

Derrick PM, Barker H (1997) Short and long distance spread of potato leafroll luteovirus: effects of host genes and transgenes conferring resistance to virus accumulation in potato. J Gen Virol 78:243–251

Dong F, Novy RG, Helgeson JP, Jiang J (1999) Cytological characterization of potato-*Solanum etuberosum* somatic hybrids and their backcross progenies by genomic in situ hybridization. Genome 42:987–992

Flis B, Syller J, Wasilewicz-Flis I, Zimnoch-Guzowska E (2005) Interaction between potato leafroll virus and potato plants and their implications for assessment of infection resistance. Am J Potato Res 82:411–417

Gavrilenko T, Thieme R, Heimback U, Thieme T (2003) Fertile somatic hybrids of *Solanum etuberosum* (+) dihaploid *Solanum tuberosum* and their backcrossing progenies: relationships of genome dosage with tuber development and resistance to potato virus Y. Euphytica 131:323–332

Gibson RW, Jones MGK, Fish N (1988) Resistance to potato leaf roll virus and potato virus Y in somatic hybrids between dihaploid *Solanum tuberosum* and *S. brevidens*. Theor Appl Genet 76:113–117

Gillen AM, Novy RG (2007) Molecular characterization of the progeny of *Solanum etuberosum* identifies a genomic region associated with resistance to potato leafroll virus. Euphytica (in press). doi:10.1007/s106810069342

Jones RAC (1979) Resistance to potato leafroll virus in *Solanum brevidens*. Potato Res 22:149–152

Lagnaoui A, Radcliffe EB (1998) Potato fungicides interfere with entomopathogenic fungi impacting population dynamics of green peach aphid. Am Potato J 75:19–25

Matsubayashi M (1991) Phylogenetic relationships in the potato and its related species. In: Tsuchiya T, Gupta P (eds) Chromosome engineering in plants: genetics, breeding, evolution. Elsevier, The Netherlands, pp 93–118

Nolte P, Miller JS, Geary BD, Corsini DL (2003) Disease management. In: Stark JC, Love SL (eds) Potato production systems. University of Idaho Extension, USA, pp 153–183

Novy RG, Helgeson JP (1994a) Somatic hybrids between *Solanum etuberosum* and diploid, tuber bearing *Solanum* clones. Theor Appl Genet 89:775–782

Novy RG, Helgeson JP (1994b) Resistance to potato virus Y in somatic hybrids between *Solanum etuberosum* and *S. tuberosum* x *S. berthaultii* hybrid. Theor Appl Genet 89:783–786

Novy RG, Nasruddin A, Ragsdale DW, Radcliffe EB (2002) Genetic resistances to potato leafroll virus, potato virus Y, and green peach aphid in progeny of *Solanum etuberosum*. Am J Potato Res 79:9–18

Perez F, Menendez A, Dehal P, Quiros CF (1999) Genomic structrual differentiation in *Solanum*: comaparative mapping of the A- and E-genomes. Theor Appl Genet 98:1183–1193

Ragsdale DW, Radcliffe EB, DiFonzo CD (2001) Epidemiology and field control of PVY and PLRV. In: Loebenstein G, Berger PH, Brunt AA, Lawson RH (eds) Virus and virus-like diseases of potatoes and production of seed-potatoes. Kluwer Academic Publishers, The Netherlands, pp 237–270

Solomon-Blackburn RM, Barker H (2001) Breeding virus resistant potatoes (*Solanum tuberosum*): a review of traditional and molecular approaches. Heredity 86:17–35

Spooner DM, Douches DS, Contreras MA (1992) Allozyme variation within *Solanum* sect. *Petota*, series *Etuberosa* (Solanaceae). Am J Bot 79:467–471

Spooner DM, Tivang J, Nienhuis J, Miller JT, Douches DS, Contreras MA (1996) Comparison of four molecular markers in measuring relationships among the wild potato relatives *Solanum* section *Etuberosum* (Subgenus Potatoe). Theor Appl Genet 92:532–540

Srinivasan R, Alvarez JM, Eigenbrode SD, Bosque-Pérez NA (2006) Influence of hairy nightshade *Solanum sarrachoides* (Sendtner) and *Potato leafroll virus* (Luteoviridae: *Polerovirus*) on the host preference of *Myzus persicae* (Sulzer) (Homoptera: Aphididae). Environ Entomol 35:546–553

Thieme R, Thieme T (2005) Resistance to viruses. In: Razdan MK, Mattoo AK (eds) Genetic improvement of solanaceous crops, vol 1: potato. Science Publishers, Enfield, NH, USA, pp 293–337

Thieme RT, Gavrilenko T, Thieme T, Hemibach U (1999) Production of potato genotypes with resistance to potato virus Y (PVY) by biotechnological methods. In: Altman A, Ziv M, Izhar S (eds) Proceedings of the 9th International congress IAPTC and biotech on ''plant biotechnology and in vitro biology in the 21st centrury'', Jerusalem, Israel. Kluwer Academic Publishers, The Netherlands, pp 557–560

Valkonen JPT, Xu YS, Rokka VM, Pulli S, Pehu E (1994) Transfer of resistance to potato leafroll virus, potato virus Y and potato virus X from *Solanum brevidens* to *S. tuberosum* through symetric and designed asymmetric somatic hybridisation. Ann Appl Biol 124:351–362

Wiese MJ, Guenther J, Pavlista A, Wyman J, Sieczka J (1998) Use, target pests, and economic impact of pesticides applied to potatoes in the United States. Report 2-CA-98, USDA NAPIAP

Wilson CR, Jones RAC (1992) Resistance to pholem transport of potato leafroll virus in potato plants. J Gen Virol 73:3219–3224

Zamoum T, Simon JC, Crochard D, Ballanger Y, Lapchin L, Vanlerberghe-Masutti F, Guillemaud T (2005) Does insecticide resistance alone account for the low genetic variability of asexually reproducing populations of the peach-potato aphid *Myzus persicae*? Heredity 94:630–639