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ORIGINAL PAPER

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# Two allelic or tightly linked genetic factors at the *PLRV.4* locus on potato chromosome XI control resistance to potato leafroll virus accumulation

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**Abstract** A novel locus for potato resistance to potato leafroll virus (PLRV) was characterized by inheritance studies and molecular mapping. The diploid parental clone DW 91-1187 was resistant to PLRV accumulation in both inoculated plants and their tuber progeny. The resistance to PLRV accumulation present in DW 91-1187 was not transmitted to any $F_1$ offspring when crossed with a PLRV susceptible clone. Instead, one half of the $F_1$ individuals exhibited undetectable amounts of PLRV as determined by ELISA during the primary infection assay, but accumulated PLRV in their tuber progeny plants. The other half was clearly infected both in the inoculated and tuber-born plants. The inheritance of resistance to PLRV accumulation may be explained by a model of two complementary alleles of a single gene (*PLRV.4*) or by two complementary genes that are closely linked in repulsion phase. Random amplified polymorphic DNA (RAPD) and inter-simple sequence repeat (ISSR) markers linked to the *PLRV.4* locus were selected. The two complementary factors were closely linked in coupling phase to the alternative alleles $UBC864_{600}$ and $UBC864_{800}$ of DNA marker UBC864. These markers may be used for marker-assisted selection of genotypes having both factors for resistance to PLRV accumulation. The *PLRV.4* locus was mapped to a central position on linkage group XI of the potato molecular map, where no resistance locus has been mapped previously.

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## Introduction

Potato leafroll virus (PLRV) is one of the most significant and widespread viral pathogens of potato (Syller 1996; Barker and Waterhouse 1999). Besides reduction of total tuber yield, infection with PLRV can lead to losses in marketable yield due to the expression of net necrosis in tubers (Novy et al. 2002). Growing PLRV-resistant potato cultivars is the most effective and environmentally safe way to control the disease (Barker and Waterhouse 1999).

Resistance to PLRV infection, which means that fewer plants become infected by viruliferous aphids (*Myzus persicae* Sulz) (Valkonen 1994), seems to be under complex genetic control (Davidson 1973). However, for exploitation in potato breeding, resistance to PLRV accumulation, which is related to the inhibition of PLRV spreading within the potato phloem, is the more important characteristic because plants resistant to viral accumulation are poor sources of PLRV for aphids (Barker 1987; Barker and Woodford 1992; Barker and Waterhouse 1999). This type of PLRV resistance in potato has been described by a simple genetic model (Barker and Solomon 1990; Barker et al. 1994; Flis and Wasilewicz-Flis 1998).

A first quantitative trait locus (QTL) analysis for resistance to PLRV accumulation in a diploid potato cross revealed one major QTL, *PLRV.1*, on potato chromosome XI and two minor QTL, *PLRV.2* and *PLRV.3*, on chromosomes VI and V, respectively (Marczewski et al. 2001). *PLRV.1* maps to a "hot spot" for resistance to various pathogens (Gebhardt and Valkonen 2001) and is closely linked to the resistance gene-like (RGL) loci *St3.3.13*(*a*) and *Nl-27* (Marczewski et al. 2001). *PLRV.1* explained more than 50% of the phenotypic variation for PLRV resistance. There was no interaction between the major and minor QTL indicating that *PLRV.2* and *PLRV.3* contributed independently to the PLRV

resistance level and may be considered as a genetic background which modifies the resistance.

The diploid clone DW 84-1457 is a different source of PLRV resistance than the one used for mapping *PLRV.1*. DW 84-1457 was selected at the Plant Breeding and Acclimatization Institute, Młochów, Poland, and is highly resistant to PLRV accumulation. Its pedigree includes the PLRV-resistant clones MPI 44 1016/10, MPI 49 540/2 and MPI 44 335/130, originating from the collection at the Max-Planck Institute for Plant Breeding Research, Cologne, Germany (Dziewońska and Waś 1994). Here we report the molecular mapping of *PLRV.4*, a new major locus for PLRV resistance, which originated from clone DW 84-1457. Our data suggest that either two complementary alleles or tightly linked genetic factors at the *PLRV.4* locus are required for the expression of resistance to PLRV accumulation.

## Materials and methods

### Plant materials

The diploid potato (*Solanum tuberosum* L.) $F_1$ population 'Ns' (Marczewski et al. 1998), was used for mapping PLRV resistance genes. The Ns population consisted of 140 clones produced by crossing the PLRV resistant female parent DW 91-1187 with the susceptible male parent DW 83-3121. DW 91-1187 originated from a cross of the PLRV resistant full sib clones DW 88-4477 and DW 88-4476, which were progeny of the PLRV resistant and susceptible parent DW 84-1457 and DG 82-199, respectively. The Ns population has been used previously to map the *Ns* gene conferring resistance to potato virus S and to identify *Ns*-linked molecular markers (Marczewski et al. 1998, 2002; Marczewski 2001).

### Test for resistance to PLRV

Thirteen single-eye plugs from the parents and each $F_1$ individual were planted in pots under greenhouse conditions. Three plants per clone were used as non-inoculated controls. Ten plants per clone were inoculated with PLRV using *Myzus persicae* (Sulz) as the virus vector, as described (Marczewski et al. 2001). The plants were tested 5 weeks after inoculation by quantitative ELISA as described by Syller (1991). Transmission of PLRV to *Physalis floridana* Rydb. plants was used as a positive control of the aphid inoculation procedure. Two tubers from each inoculated plant were planted in the greenhouse. Tuber-derived plants were examined by ELISA for secondary PLRV infection.

### PCR amplification

DNA extraction, PCR amplification of inter-simple sequence repeat (ISSR) markers and electrophoresis were performed as previously described (Marczewski 2001). PCR conditions for amplifying the random amplified polymorphic DNA (RAPD) markers were described by Marczewski et al. (1998). A pseudo-sequence characterized amplified region (pseudo-SCAR) marker was developed according to the procedure of Chagué et al. (1996). The markers GP250, Tal 1 and Nl-27 were amplified as described by Oberhagemann et al. (1999), Chen et al. (2001) and Marczewski et al. (2001), respectively, resulting in PCR products of 750 bp for GP250, 1,600 and 1,700 bp for Tal 1 and 1,164 bp for Nl-27. PCR analyses of the markers St3.3.11 and CP117 were performed in 20 µl of 20 mM Tris-HCl pH 8.4, 50 mM KCl, 1.5 mM $MgCl_2$, 0.1 mM of each deoxynucleotide, 0.25 µM of each primer, containing 1 U *Taq* DNA Polymerase (Gibco BRL) and 30 ng genomic DNA. Forward (5′-AGGTGCTTGTTTTGTTG-TAA-3′) and reverse (5′-CTGTGCCACCCGTTGAGA-3′) primers for St3.3.11 (GenBank accession U60083) were designed according to the Primer Select Program (DNA STAR, Madison, Wis., USA, version for Windows 3.10, 1998). The PCR primers for CP117 (GenBank accession AJ487326) were as follows: forward (5′-GAATTTGCG-TAACAGACCTAACT-3′) and reverse (5′-TAAATAA-TAAAAACATACTT-3′). The PCR parameters for amplifying a 415 bp fragment of St3.3.11 were: 94°C for 60 s, followed by 40 cycles of 93°C for 15 s, 52°C for 20 s, 72°C for 60 s, and a final extension time of 5 min at 72°C. The annealing temperature was 44°C for amplification of PCR products of CP117.

### Mapping of the locus *PLRV.4*

DNA samples of the parental clones P18 and P40 and eighty $F_1$ individuals of the diploid mapping population F1840 (Gebhardt et al. 1991, 2003; Leister et al. 1996) were used to map the pseudo-SCAR marker $UBC864R_{600}$. Segregation of $UBC864R_{600}$ was scored as presence or absence of the marker fragment. The map position was identified relative to the restriction fragment length polymorphism (RFLP) map existing for this population using the software package MAPRF (E. Ritter, NEIKER, 01080 Vitoria, Spain). The linkage group XI of parent DW 91-1187 was constructed based on scoring cleaved amplified polymorphic sequence (CAPS) and SCAR markers in the Ns population, using the same software.

## Results

### PLRV resistance tests

The parents and 140 $F_1$ individuals of the Ns population were screened for the relative amounts of PLRV in the inoculated and tuber progeny plants. The absorbance values for PLRV in the primary infected plants clearly showed a bimodal distribution (Fig. 1a), indicating the presence of a major gene for PLRV resistance. The PLRV

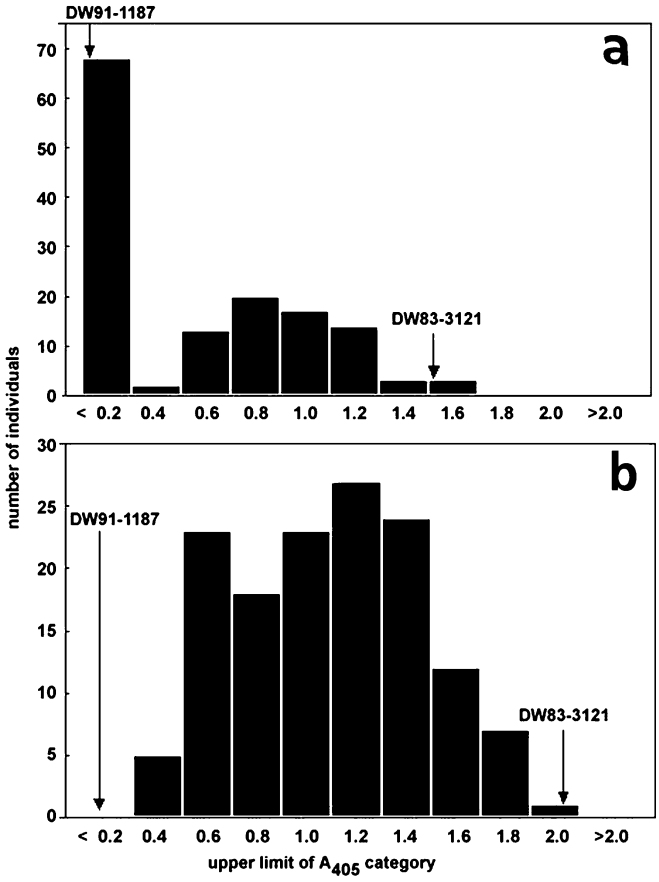


**Fig. 1a, b** Frequency distribution of $A_{405}$ values (means of ten individual plants per clone) measured by ELISA in 140 $F_1$ individuals of the Ns population inoculated with PLRV. The parent values are indicated by *arrows*

resistant parent DW 91-1187 had very low ($A_{405}$<0.05) and the susceptible parent DW 83-3121 had very high ($A_{405}$>1.4) absorbance values. For non-inoculated control plants $A_{405}$ values ranging from 0.02 to 0.1 were observed. Inoculated plants of the Ns population were divided in two groups; group A, having $A_{405}$ values higher than 0.4, with quantitative variation, and group B, having $A_{405}$ values lower than 0.2. Seventy-two $F_1$ individuals were in group A and the remaining 68 in group B. This fitted a 1:1 segregation ratio ($\chi^2$=0.114, $P$>0.05) expected for a single, dominant gene present in the heterozygous state in the resistant parent DW 91-1187. In the secondary infection assay, PLRV was detected in all tuber progeny plants of DW 83-3121 and all $F_1$ offspring, with mean absorbance values ranging from 0.3 to 2.0 (Fig. 1b). Only plants of the parent DW 91-1187 were still resistant to secondary infection by tuber-borne PLRV. No foliar symptoms of infection with PLRV were found in recovery bioassay tests on *Physalis floridana* plants, which had been inoculated by *Myzus persicae* (Sulz) aphids fed on the tuber progeny plants of DW 91-1187.

## RAPD and ISSR markers linked to PLRV resistance

Polymorphism between DNA fragments amplified from the parents using RAPD and ISSR primers has been reported previously (Marczewski et al.1998; Marczewski 2001). Two bulked DNA samples were constructed from eight $F_1$ plants of group A and eight $F_1$ plants of group B. Seventy-six primers were used for RAPD analysis of the DNA bulks. Two DNA fragments of 380 and 650 bp were amplified only in bulk B with the primers OPI03 and OPA18, respectively. When these RAPD primers were tested on the whole Ns population, the RAPD fragments $OPI03_{380}$ and $OPA18_{650}$ were present in 95% of the plants, indicating higher levels of resistance to PLRV accumulation, and in only 5% of the plants with significant PLRV titers after inoculation (Table 1). This showed that both markers were closely linked to the PLRV resistance locus. Of the ISSR primers tested (Marczewski 2001), 30 primers generated specific DNA fragments in the resistant parent DW 91-1187. Two of these ISSR fragments generated with primer UBC864 in the resistant parent DW 91-1187 (Fig. 2, lane 2), segregated in the Ns population as alternative alleles and were linked to the PLRV resistance locus. The 600 bp fragment $UBC864_{600}$ was linked to the "resistance allele" (Table 1), whereas the 800 bp fragment $UBC8648_{800}$ was linked to the susceptibility allele.

## Conversion of ISSR marker $UBC864_{600}$ into a pseudo-SCAR marker

In order to obtain more reliable and specific PCR amplification, the ISSR fragment $UBC864_{600}$ was converted into the pseudo-SCAR marker $UBC864R_{600}$. Two specific primers, UBC864AC and UBC864AG, were identified experimentally by extending the sequence $(ATG)_6$ of primer UBC864 with AC or AG. The conditions for DNA amplification were similar to that used for detection of the ISSR fragment (Marczewski 2001) except for the primer concentrations (0.1 μM each) and the annealing temperature (48°C). Using these primers, a strong amplification product of 600 bp was obtained in the resistant parent DW 91-1187, which was

**Table 1** Number of $F_1$ individuals classified as susceptible (*group A*) or expressing higher resistance to PLRV accumulation after inoculation (*group B*), possessing the marker fragments $OPI03_{380}$, $OPA18_{650}$ and $UBC864_{600}$

| Phenotypic groups | $OPI03_{380}$ | | $OPA18_{650}$ | | $UBC864_{600}$ | |
|---|---|---|---|---|---|---|
| | Present | Absent | Present | Absent | Present | Absent |
| Group A | 4 | 68 | 4 | 67 | 4 | 68 |
| Group B | 61 | 3 | 63 | 5 | 63 | 5 |

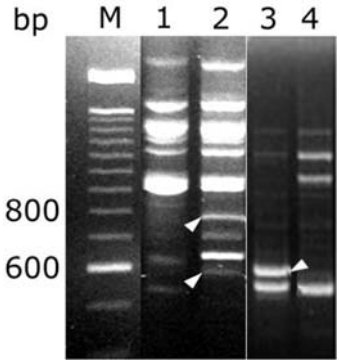


**Fig. 2** Patterns of amplified DNA of the parents DW 91-1187 (*lanes 2* and *3*) and DW 83-3121 (*lanes 1* and *4*), using ISSR primer UBC864 (*lanes 1* and *2*) and the pseudo-SCAR primer pairs UBC864AC and UBC864AG (*lanes 3* and *4*). The ISSR markers $UBC864_{600}$ and $UBC864_{800}$ (*lane 2*), and the pseudo-SCAR marker $UBC864R_{600}$ (*lane 3*) are indicated by *arrows*. Lane M contains the 100-bp DNA ladder as molecular size marker

absent in the susceptible parent DW 83-3121 (Fig. 2, lane 3).

## Mapping of the locus *PLRV.4*

The pseudo-SCAR marker $UBC864R_{600}$ segregated in the F1840 mapping population (Gebhardt et al. 2003) and mapped to a central position on linkage group XI, closely linked to the RGL locus *St3.3.11* (Leister et al. 1996). To confirm this position, the three markers St3.3.11, CP117 and Tal 1 mapping to the same region (Leister et al. 1996; Chen et al. 2001), and markers Nl-27 and GP250 mapping to both distal ends of linkage group XI (Hehl et al. 1999; Gebhardt et al. 2001) were tested by CAPS or SCAR assay for segregation in the Ns population. Polymorphisms informative for the resistant parent DW 91-1187 were obtained by digesting the PCR products of St3.3.11, Nl-27 and GP250 with restriction enzymes *Hin* fI, *Rsa* I and *Alu* I, respectively. The 1,700 bp fragment of Tal 1 was amplified only in DW 91-1187. The CP117 assay resulted in two fragments of 300 and 290 bp in DW 91-1187. The band of 290 bp was not observed in the susceptible parent DW 83-3121. Marker $UBC864R_{600}$ co-segregated with St3.3.11, Tal 1 and $OPA18_{650}$, thereby confirming the position of *PLRV.4* on chromosome XI. Based on the segregation data of *PLRV.4* and all markers tested in the Ns population, linkage group XI of the resistant parent DW 91-1187 was constructed, placing the *PLRV.4* locus to a central position, with $OPI03_{380}$ being the most closely linked marker (Fig. 3).

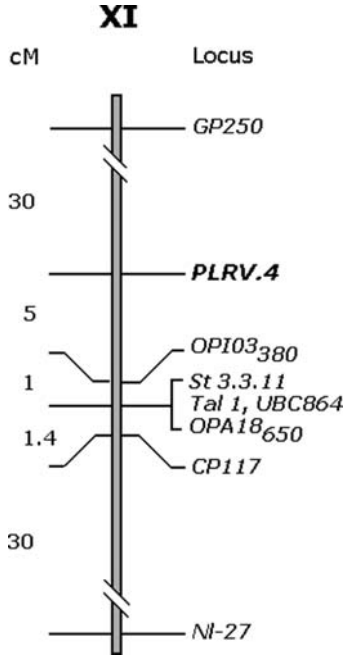


**Fig. 3** Potato linkage group XI of the PLRV resistant parent DW 91-1187, including the position of the *PLRV.4* locus for resistance to PLRV. The orientation of the linkage group is according to the alignment between cytogenetic and molecular maps (Dong et al. 2000), where the short chromosome arm is the "North" arm

## Discussion

Resistance to PLRV accumulation was clearly present in the parental clone DW 91-1187, but was not transmitted to any of the $F_1$ offspring when crossed with the susceptible clone DW 83-3121. Instead, the primary infection assay showed that the offspring segregated for two phenotypic classes, one having very low and the other one having significant $A_{405}$ values for PLRV in ELISA tests. However, PLRV was clearly detected in tuber-born plants of all $F_1$ individuals. The best explanation for this observation is a model where two alleles of a single factor, or two factors closely linked in repulsion phase at the *PLRV.4* locus are required to control resistance to PLRV accumulation in the parental clone DW 91-1187. In the $F_1$ offspring, upon separation and new combinations with the alleles of DW 83-3121, one factor confers a higher level of resistance to PLRV accumulation, observed only in the primary infection test. The second factor alone results in susceptibility. These genetic factors were linked in coupling phase to alternative alleles of the UBC864 marker, both present in the resistant parent DW 91-1187. Allele $UBC864_{600}$ was diagnostic for $F_1$ plants expressing higher resistance to PLRV accumulation, while allele $UBC864_{800}$ was present in most fully-susceptible $F_1$ plants.

In an earlier study of the inheritance of PLRV resistance expression, two unlinked, dominant, complementary genes were postulated to be involved in resistance to PLRV accumulation (Barker et al. 1994). Other examples have been reported in the literature, where full resistance to a single virus was controlled by more than one gene. Epistasis between genes, both recessive and dominant, have been reported by Dogimont et al. (1997). In potato, resistance to potato virus Y (Flis 1995; Valkonen at al. 1998) and potato virus A (Hämäläinen et al. 2000) was found to be the result of epistatic gene interactions.

Conversion of the marker $UBC864_{600}$ into the more reliable pseudo-SCAR marker $UBC864R_{600}$ facilitated the molecular mapping of the *PLRV.4* locus to a central position on potato chromosome XI. This position is clearly different from the *PLRV.1* locus, which maps to a distal resistance hot spot on the long arm of chromosome XI tagged by marker GP250 (Marczewski et al. 2001). *PLRV.4* is equally distant from the *R* gene cluster on the distal end of the short arm of chromosome XI. The map segment containing *PLRV.4* also contains the RGL marker loci St3.3.11 and St3.3.13(b) (Leister et al. 1996). No other gene for resistance has been detected so far in this region of potato chromosome XI (Gebhardt and Valkonen 2001). In tomato however, the *Sm* locus for resistance to grey leaf spot disease (*Stemphylium* species) maps to a similar central segment of tomato chromosome 11. This can be inferred from the anchor marker CP117 linked to *PLRV.4* in the Ns population, which has been mapped in both species and is positioned close to the *Sm* locus on tomato chromosome 11 (Tanksley et al. 1992; Behare et al. 1991). *PLRV.4* and *Sm* may both belong to a cluster of resistance genes.

The finding of molecular markers diagnostic for agriculturally important characters is the prerequisite for marker-assisted selection in crop plants. Most useful are easy testable DNA markers that are closely linked to desirable traits, for which phenotypic selection is expensive, difficult or even impossible (Staub et al. 1996). This is the case for the resistance to PLRV accumulation described in this study. Phenotypic selection is complicated by aphid transmission of the virus and requires the analysis of two plant generations, primary infected plants and tuber-born plants derived from them. The PCR testable marker $UBC864_{800}$ may be used to identify susceptible genotypes that generate PLRV-resistant offspring when crossed with carriers of the $UBC864_{600}$ allele. Offspring having resistance to PLRV accumulation can be selected with high confidence in the first plant generation based on both markers. The model proposed above for the control of potato resistance to PLRV accumulation can now be experimentally tested by crossing plants that have one or the other marker allele and applying MAS to find plants combining both marker alleles, which should also express resistance to PLRV accumulation. Recovery of resistance to vascular transport of potato virus A has been observed in progeny with specific allele combinations of susceptible potato parents (Hämäläinen et al. 2000). Breeding diploid potatoes for recovering resistance to PLRV accumulation will be the subject of our further studies.

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