A Major Quantitative Trait Locus for Resistance to *Potato leafroll virus* Is Located in a Resistance Hotspot on Potato Chromosome XI and Is Tightly Linked to *N*-Gene-Like Markers

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Potato leafroll virus (PLRV) causes one of the most widespread and important virus diseases in potato. Resistance to PLRV is controlled by genetic factors that limit plant infection by viruliferous aphids or virus multiplication and accumulation. Quantitative trait locus (QTL) analysis of resistance to virus accumulation revealed one major and two minor QTL. The major QTL, *PLRV.1*, mapped to potato chromosome XI in a resistance hotspot containing several genes for qualitative and quantitative resistance to viruses and other potato pathogens. This QTL explained between 50 and 60% of the phenotypic variance. The two minor QTL mapped to chromosomes V and VI. Genes with sequence similarity to the tobacco *N* gene for resistance to *Tobacco mosaic virus* were tightly linked to *PLRV.1*. The cDNA sequence of an *N*-like gene was used to develop the sequence characterized amplified region (SCAR) marker NI27₁₁₆₄ that can assist in the selection of potatoes with resistance to PLRV.

Potato leafroll virus (PLRV; genus *Potyvirus*, family *Luteoviridae*) causes one of the most widespread and important virus diseases in potato (Barker and Waterhouse 1999; Syller 1996). In potato, PLRV infection causes yield reduction ranging from 10 to 95% (van der Zaag 1987; Watson and Wilson 1956). The distribution of PLRV in infected plants is restricted to the phloem. The virus is persistently transmitted by aphids and is not mechanically transmissible (Casper 1988). Spread of PLRV in the potato crop can be controlled by insecticide application. A more economic and environmentally more acceptable way to control PLRV is the cultivation of potatoes with enhanced resistance (Barker and Waterhouse 1999).

There are two main types of resistance to PLRV. One operates against infection by viruliferous aphids and the other one limits virus multiplication and accumulation (Barker and Harrison 1986). The latter type of resistance is of great importance because resistant plants are poor sources of PLRV for the aphid vectors (Barker 1987; Barker and Woodford 1992).

Resistance to PLRV infection has been described as polygenically controlled (Davidson 1973), whereas resistance to

PLRV accumulation seems to be under simple genetic control. Barker and Solomon (1990) and Brown and Thomas (1994) proposed that the second type of resistance is due to a single dominant gene. Later studies supported the hypothesis of two unlinked, dominant complementary genes conferring resistance to PLRV accumulation (Barker et al. 1994). An oligogenic segregation of resistance was also supported by the experiments of Świeżyński and associates (1990). Expression of resistance under major gene control seemed to be modified by other factors (Brown and Thomas 1994). These modifier genes affecting resistance have not been further studied. It is, however, accepted that the presence of a major gene without a suitable genetic background does not sufficiently protect a plant from PLRV (Świeżyński 1993). The description of the inheritance of resistance to PLRV is derived from classical genetic analysis. Map positions of genes for resistance to PLRV and their molecular basis are unknown.

A number of wild and cultivated *Solanum* species host genes for resistance to PLRV (Barker and Waterhouse 1999; Świeżyński 1993). Among others, *S. chacoense* seems to be the most promising source of resistance for exploitation in potato-breeding programs (Barker and Waterhouse 1999; Brown and Thomas 1994). *S. chacoense* was part of the pedigree of the diploid interspecific hybrid clone DG83-2025, which carries multiple resistance traits (Zimnoch-Guzowska et al. 2000), including resistance to PLRV accumulation. Thus, the PLRV resistance present in clone DG83-2025 might originate from *S. chacoense*.

In this paper, we report the mapping with DNA markers of resistance to PLRV accumulation in the *Erwinia* population (Zimnoch-Guzowska et al. 2000), an F1 hybrid family of clone DG83-2025, and the susceptible clone DG81-68. One major and two minor quantitative trait loci (QTL) were identified. The major QTL mapped to potato chromosome XI within a hotspot for resistance to various pathogens and was tightly linked to potato genes with sequence similarity to *N*, a tobacco gene for resistance to *Tobacco mosaic virus* (TMV) (Whitham et al. 1994).

RESULTS

PLRV resistance tests.

Parents and 137 F1 plants of the *Erwinia* population were screened for the relative amount of virus in secondarily in-

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fectured plants. When tested by enzyme-linked immunosorbent assay (ELISA) in 1999, PLRV was detected in all tuber progeny plants of the parent DG81-68 and 101 F1 hybrids. For the parent DG83-2025 and the remaining 36 F1 hybrids, the mean values of two ELISAs did not exceed an A_{405} of 0.2 and were only slightly higher than the values obtained from non-inoculated control plants (mean A_{405} values ≤ 0.1). Parent DG83-2025 and the 36 F1 hybrids had been subjected to additional greenhouse inoculation with PLRV by aphids in the previous year. They expressed, therefore, high resistance to PLRV accumulation. The PLRV resistance of these plants was confirmed in the subsequent vegetative generation. The data from two ELISAs were highly correlated ($r = 0.91$, $P < 0.001$). The frequency distribution of mean A_{405} values of 15 plants per genotype for the second ELISA is shown in Figure 1. The distribution exhibited significant positive skewness and negative kurtosis. The number of modes suggests that more than one peak was present (Table 1). The mean A_{405} value of the susceptible parent DG81-68 was close to the population mean.

QTL for PLRV resistance.

Amplified fragment length polymorphism (AFLP) and restriction fragment length polymorphism (RFLP) markers, in-

cluding resistance gene-like (RGL) markers that map to the 12 linkage groups of each parent of the *Erwinia* population (Zimnoch-Guzowska et al. 2000), were tested for linkage to QTL for PLRV resistance using the t test. Significant ($P < 0.01$) and positive (low A_{405} values) effects were detected in both ELISAs with markers located on linkage groups XI and V of the PLRV-resistant parent DG83-2025 and on linkage group VI of the susceptible parent DG81-68 (Table 2). Analysis of variance showed that distal markers on the north arm of chromosome XI explained between 48 and 60% of the phenotypic variance, indicating that a major QTL for PLRV resistance is located in this map region (Table 2, Fig. 2). RFLP marker GP76 identified one of the two smaller effects (Table 2) and is located in the distal segment of the south arm of chromosome VI (Gebhardt et al. 1991; Zimnoch-Guzowska et al. 2000). AFLP marker HM4-26 explained between 11 and 16% of the phenotypic variance and is located on linkage group V of the DG83-2025 parent (data not shown). The AFLP marker allele had a highly distorted segregation ratio (Table 2). The map position of the AFLP locus *HM4-26* could not be determined with certainty relative to existing molecular maps of potato chromosome V. No evidence was found for interaction between the three QTL.

Fig. 1. Frequency distribution of A_{405} values (means of 15 plants per genotype) measured by enzyme-linked immunosorbent assay in 137 F1 hybrids of the *Erwinia* population secondarily infected with *Potato leafroll virus*. The parent values are indicated by arrows. d Kolmogoroff-Smirnoff = 0.1255202 and $P < 0.05$. Chi-square = 24.87698, df = 9, and $P = 0.0031184$.

Table 1. Statistical parameters for the frequency distribution of enzyme-linked immunosorbent assay 2 in the *Erwinia* population

Plant identification	Mean value of A_{405}	Standard deviation	No. of modes	Skewness	Kurtosis
DG83-2025	0.007	0.007	... ^a	...	...
DG81-68	0.680	0.228	...	...	...
F1 progeny	0.617	0.468	2	0.323	-1.189

^a ... = Not applicable.

N-like genes linked to PLRV resistance.

The major QTL for PLRV resistance mapped to a position on the potato molecular map, where a cluster of genes with sequence similarity to the tobacco *N* gene for resistance to

Fig. 2. Position of quantitative trait locus *PLRV.1* on linkage group XI of the *Potato leafroll virus*-resistant parent DG83-2025. Restriction fragment length polymorphism (GP125, GP185), amplified fragment length polymorphism (HM*-**, EM*-**; Zimnoch-Guzowska et al. 2000), resistance gene-like marker loci and map distances in cM are shown on the left of the linkage group. The amount of phenotypic variance explained at the marker loci (R^2) in enzyme-linked immunosorbent assay 2 is shown on the right. The sequence characterized amplified region marker NI27₁₁₆₄ explained the largest amount of the phenotypic variance and, therefore, determines the position of *PLRV.1*.

TMV (Whitham et al. 1994) has been identified (Leister et al. 1996). *St3.3.13(a)*, one of four RFLP loci identified by the *N*-like marker *St3.3.13* in the *Erwinia* population (Zimnoch-Guzowska et al. 2000) was most closely linked to the QTL *PLRV.1* (Table 2, Fig. 2). Full-length cDNA sequences of two members of the *St3.3.13* gene family have been described (Hehl et al. 1999). Polymerase chain reaction (PCR) primers were designed for the cDNA NI-27, resulting in two amplification products of 2 and 1.2 kb predominantly in the resistant parent DG83-2025. Both PCR fragments segregated in the *Erwinia* population (Fig. 3). The 2-kb PCR product mapped to linkage group II (data not shown) and the 1.2-kb PCR product mapped to linkage group XI, 1 cM proximal to the *St3.3.13(a)* locus (Fig. 2). Presence of the 1.2-kb NI-27 product was linked with increased resistance to PLRV and explained 50 and 60% of the phenotypic variance in ELISAs 1 and 2, respectively (Table 2). All 36 PLRV resistant F1 plants had the 1.2-kb NI-27 PCR product.

DISCUSSION

QTL analysis of resistance to PLRV accumulation identified one major QTL and two additional QTL with smaller effects on three different potato chromosomes. The major QTL *PLRV.1* on chromosome XI explained more than 50% of the phenotypic variance. The presence of one locus with a major effect on resistance explains the bimodal distribution of resistance phenotype. Our findings corroborate the hypothesis, derived from classical inheritance studies, that resistance to PLRV accumulation is controlled largely by a single locus (Barker and Solomon 1990; Brown and Thomas 1994; Flis and Wasilewicz-Flis 1998). Interactions between the major QTL and the secondary effects on chromosomes V and VI were not detected, indicating that these QTL did not interfere with the expression of the major QTL but contributed independently to the overall resistance level. One of the secondary QTL effects at the *PLRV.2* locus was inherited from the susceptible parent. This supports earlier observations that the genetic background can influence the observed level of resistance to PLRV due to a major gene (Flis and Wasilewicz-Flis 1998; Świeżyński 1993).

PLRV.1 maps to potato chromosome XI distal to the anchor RFLP marker *GP125* and close to the RGL locus *St3.3.13(a)*. This region of the potato genome contains a number of single genes for resistance (*R* genes) to various pathogens (Gebhardt and Valkonen 2001). Among those are genes for extreme resistance to *Potato virus Y* (PVY) (Brigneti et al. 1997;

Table 2. Mapping of quantitative trait locus (QTL) for *Potato leafroll virus* (PLRV) resistance to potato linkage groups

QTL	Marker	Origin of the resistance QTL allele	Linkage group	No. of plants per genotype class ^a		ELISA ^b 1				ELISA 2			
				N ₁	N ₀	X ₁ ^c	X ₀ ^c	R ² ^d	P ^e	X ₁	X ₀	R ²	P
<i>PLRV.1</i>	NI27 ₁₁₆₄	DG83-2025	XI	60	73	0.18	0.66	0.50	0.000	0.21	0.94	0.60	0.000
<i>PLRV.1</i>	<i>St3.3.13(a)</i>	DG83-2025	XI	40	49	0.20	0.66	0.48	0.000	0.24	0.93	0.56	0.000
<i>PLRV.2</i>	GP76	DG81-68	VI	30	40	0.55	0.36	0.12	0.003	0.74	0.41	0.12	0.003
<i>PLRV.3</i>	HM4-26	DG83-2025	V	101	32	0.39	0.65	0.11	0.000	0.51	0.94	0.16	0.000

^a Two genotype classes are distinguished by presence (1) or absence (0) of a specific allele (polymerase chain reaction, restriction fragment length polymorphism, or amplified fragment length polymorphism) at the specified marker locus.

^b ELISA = enzyme-linked immunosorbent assay.

^c X₁ and X₀ are the phenotypic means (A_{405}) of the marker genotype classes.

^d R² is the amount of the phenotypic variance explained by the marker.

^e P is the probability for the absence of linkage between the QTL and the marker allele.

Hämäläinen et al. 1997), hypersensitive resistance to *Potato virus A* (PVA; Hämäläinen et al. 1998, 2000), a gene for resistance to potato wart caused by the fungus *Synchytrium endobioticum* (Hehl et al. 1999), and a gene for resistance to the root knot nematode *Meloidogyne chitwoodi* (Brown et al. 1996). Moreover, QTL for resistance to late blight caused by *Phytophthora infestans* (Leonards-Schippers et al. 1994) and the bacterial pathogen *Erwinia carotovora* subsp. *atroseptica* (Zimnoch-Guzowska et al. 2000) have been mapped to the same genome segment. Clustering of genes controlling monogenic and polygenic resistance to pathogens may occur by chance or may be a consequence of reduced recombination fractions in the proximity of the centromere. The resistance gene cluster on chromosome XI has, however, the longest possible distance from the centromere, as it is located at the distal end of the long chromosome arm (Dong et al. 2000). Alternatively, the clustering, particularly of genes for resistance to PVY, PVA, and now PLRV, suggests that some of these genes have a similar molecular basis, either being alleles of a single locus or having evolved from a common ancestor by local gene duplications with subsequent functional diversification. Tightly linked to the resistance gene cluster on the long arm of potato chromosome XI are several genes with sequence similarity to the *N* gene for resistance to TMV (Hämäläinen et al. 1998; Hehl et al. 1999; Leister et al. 1996; Whitham et al. 1994). Cloned potato genes for resistance to *Potato virus X* (PVX) (Bendahmane et al. 1999, 2000) and the root cyst nematode *Globodera pallida* (Van der Vossen et al. 2000) belong to the same superfamily of resistance genes as *N*. The colocalization of *N*-like genes suggests that genes with sequence similarity to known *R* genes are the molecular basis for some resistance factors in the cluster on chromosome XI, including *PLRV.1*.

A sequence characterized amplified region (SCAR) marker was developed for *PLRV.1* based on the *N*-like cDNA sequence NI-27 (Hehl et al. 1999). Interestingly, two amplification products were obtained, both present in the PLRV-resis-

tant parent, one of which was tightly linked to *PLRV.1* on chromosome XI, and the other one mapped to an independent locus on chromosome II. The cDNA NI-27 is one member of a multigene family. Primers for sequences conserved between different members of the family can detect, therefore, more than one gene. The marker NI27₁₁₆₄ can be used to select for PLRV resistance in offspring of clone DG83-2025. It remains to be tested to what extent this marker is indicative for presence of PLRV resistance in wide germ plasm pools of potato, where resistance to PLRV accumulation can be present separately and in combination with resistance to infection by viruliferous aphids (Dziewońska and Waś 1994; Solomon-Blackburn and Barker 1993; Świeżyński et al. 1989). Resistance to infection is controlled by factors of yet unknown map position. The possibility exists that the marker NI-27₁₁₆₄ is located within or physically very close to the *PLRV.1* resistance gene. In this case, strong linkage disequilibrium is expected between this marker and presence of the PLRV resistance allele of DG83-2025, even in a wide germ plasm pool. Markers have been found that are diagnostic for the presence of the *Ry_{adg}* gene for resistance to PVY in a wide potato germ plasm pool (Kasai et al. 2000; Sorri et al. 1999). These markers are based on a different *N*-like sequence that mapped to the same resistance hotspot on potato chromosome XI that includes *Ry* and *PLRV.1*. PCR markers derived from genes with sequence similarity to *R* genes are, therefore, highly useful as tools for marker-assisted selection for pathogen resistance.

MATERIALS AND METHODS

Plant material.

The population used for mapping PLRV resistance genes was the *Erwinia* population described by Zimnoch-Guzowska and associates (2000). The parents of the *Erwinia* population were diploid clones DG83-2025 (seed parent) and DG81-68 (pollen parent). Clone DG83-2025, descending from intercrossing the wild potato species *S. chacoense* and *S. yun-*

Fig. 3. Electrophoretic patterns of the sequence characterized amplified region marker NI27₁₁₆₄ in the *Erwinia* population. Lanes are as follows: DG81, the *Potato leafroll virus* (PLRV)-susceptible parent DG81-68; DG83, the PLRV-resistant parent DG83-2025; 1, 79, 34, and 36, examples for resistant F1 individuals; 33, 143, 145, and 97, examples for susceptible F1 individuals; M1 and M2, molecular-size markers; C, negative control [no template DNA was added to the polymerase chain reaction (PCR)]. Two PCR products of 1.2 and 2 kb (indicated by the arrows) were obtained in the PLRV-resistant parent DG83-2025, which segregated in the F1 offspring resulting in four different marker phenotypes represented by two plants each: both fragments (lanes 34 and 36), none of the fragments (lanes 145 and 97), either the 1.2-kb fragment (lanes 1 and 79) or the 2-kb fragment (lanes 33 and 143) are present. The 1.2-kb fragment is linked to *PLRV.1*, whereas the 2-kb fragment is unlinked and maps to linkage group II.

gasense with *S. tuberosum* (Zimnoch-Guzowska et al. 2000), was the donor of resistance to PLRV. One hundred thirty-seven F1 individuals of the *Erwinia* population were available for mapping QTL for PLRV resistance.

Test for resistance to PLRV.

Since 1995, the *Erwinia* population (parents and 137 F1 hybrids) were propagated in the field under natural PLRV infection pressure. When tested by quantitative ELISA (Syller 1991) using polyclonal antibodies against PLRV (Goszczyński 1987), the majority of plants of the parent DG81-68 and of 101 F1 hybrids contained high titers of PLRV. Tubers from these infected genotypes were greenhouse propagated in 1997 and 1998. Parent DG83-2025 and 36 F1 hybrids appeared highly resistant to PLRV based on very low virus titers. Plants of these genotypes were inoculated with the virus in 1998. Five single-eye plugs from each genotype were cut and planted in pots under greenhouse conditions in April 1998. The plants were inoculated with PLRV at the initial stage of leaf expansion by using *Myzus persicae* (Sulzer) aphids that had been kept for at least 1 week on the PLRV-infected cultivar Osa. The aphids were transferred onto test plants with pieces of infected source leaves. In addition, shaking PLRV-infected shoots that were colonized by aphids over the test plants ensured strong infection pressure (Syller 1991). Transmission of PLRV to *Physalis floridana* Rydb. plants was used as a positive control of the aphid inoculation procedure. The aphids were allowed to acquire and inoculate PLRV in feedings over a period of 3 days. After inoculation, plants were grown in a greenhouse under 12-h light conditions at 19 to 25°C and tubers were harvested. In spring 1999, tuber progeny plants of parents and all 137 hybrid clones (15 plants per genotype) were planted in the greenhouse and tested twice by ELISA (between 4 and 6 weeks after planting), as described by Syller (1991). Both ELISAs were done for all plants on the same date. ELISA readings were taken after 1 h of incubation with substrate at room temperature and averaged over 15 plants of each genotype. Tubers of the most resistant clones were again examined by ELISA in the 2000 season.

DNA marker analysis.

Genotyping with AFLP (Vos et al. 1995), RFLP and RGL markers, and linkage map construction were described earlier (Zimnoch-Guzowska et al. 2000). The SCAR marker NI27₁₁₆₄ was amplified using approximately 50 ng of template DNA in 50 µl of 20 mM Tris-HCl, pH 8.4; 50 mM KCl; 0.1 mM of each dNTP; 1.5 mM MgCl₂; 0.23 µM each of the primers NI27-f (5'-TAGAGAGCATTAAAGAAGCTGC-3') and NI27-r (5'-TTTTCCTACTCCCGCATG-3'); and 1 U of *Taq* DNA polymerase (Gibco BRL, Karlsruhe, Germany). The primers were complementary to positions 57 to 77 and 853 to 872, respectively, of the cDNA sequence NI-27 (EMBL accession no. AJ009720) described by Hehl and associates (1999). PCR conditions were 1 cycle of 93°C for 60 s, followed by 35 cycles of 93°C for 30 s, 62°C for 30 s, 72°C for 90 s, and a final extension time of 10 min at 72°C. PCR products were size-separated by electrophoresis in 1.5% agarose and visualized by ethidium bromide staining under standard conditions (Sambrook et al. 1989).

QTL analysis and statistics.

All marker fragments scored in the *Erwinia* population were tested for effect on PLRV resistance using the *t* test, as described by Zimnoch-Guzowska and associates (2000). Analysis of variance for single markers and testing for interaction between markers was done using SPSS software (SPSS Base 10.0; SPSS GmbH Software, München, Germany). The distribution of the phenotypic data was tested for normality based on a goodness of fit (χ^2) test.

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