

The novel gene *Ny-1* on potato chromosome IX confers hypersensitive resistance to *Potato virus Y* and is an alternative to *Ry* genes in potato breeding for PVY resistance

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Received: 18 May 2007 / Accepted: 20 October 2007 / Published online: 6 November 2007
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Abstract Hypersensitive resistance (HR) is an efficient defense strategy in plants that restricts pathogen growth and can be activated during host as well as non-host interactions. HR involves programmed cell death and manifests itself in tissue collapse at the site of pathogen attack. A novel hypersensitivity gene, *Ny-1*, for resistance to *Potato virus Y* (PVY) was revealed in potato cultivar Rywal. This is the first gene that confers HR in potato plants both to common and necrotic strains of PVY. The locus *Ny-1* mapped on the short arm of potato chromosome IX, where various resistance genes are clustered in Solanaceous genomes. Expression of HR was temperature-dependent in cv. Rywal. Strains PVY^O and PVY^N, including subgroups PVY^{NW} and PVY^{NTN}, were effectively localized when plants were grown at 20°C. At 28°C, plants were systemically infected but no symptoms were observed. In field trials, PVY was restricted to the inoculated leaves and PVY-free tubers were produced. Therefore, the gene *Ny-1* can be useful for potato breeding as an alternative donor of

PVY resistance, because it is efficacious in practice-like resistance conferred by *Ry* genes.

Introduction

Plants have evolved complex genetic systems to defend against pathogen attack. Elucidation of the molecular mechanism of plant disease resistance is important for several reasons. First, understanding how plants respond to pathogens is critical for the application of engineering for increased crop resistance to viruses, viroids, bacteria and fungi. In addition, the determination of the pathway from initial recognition of a pathogen to the induction defence-strategy will yield insight into basic aspects of specific signal transduction pathways.

Potato virus Y (PVY) is a member of the genus *Potyvirus* (*Potyviridae* family). Two main groups of PVY strains, PVY^O and PVY^N, separated according to their pathogenic properties, are the most harmful viruses in cultivated potato (*Solanum tuberosum* ssp. *tuberosum*). PVY^O (common, ordinary strain) induces leaf mottling, extensive necrotic and drop streak symptoms, and often leads to plant death. PVY^N (tobacco veinal necrosis strain) causes, in general, mild mosaic symptoms in potato leaves (De Bokx and Huttinga 1981; Brunt 2001; Valkonen 2007).

A subgroup of PVY^N, designated as PVY^{NTN}, is responsible for potato tuber necrotic ringspot disease, which results in losses of tuber quality (Beczner et al. 1984). The second subgroup, PVY^{NW}, originally described in Polish cv. Wilga, as PVY^{NWi} (Chrzanowska 1991), induces vein necrosis in *Nicotiana tabacum* cv. Samsun and mild mosaic in potato plants, but is serologically related to PVY^O (Chrzanowska 1994), probably as the outcome of recombina-

Communicated by R. Waugh.

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nation events in the virus coat protein gene (Glais et al. 2005).

In potato, there are two main types of resistance to PVY, extreme resistance (ER) and hypersensitive resistance (HR). The *Ry* genes for ER confer extremely high level of protection against different strains of PVY (Ross 1986; Valkonen et al. 1996). Three *Ry* genes have been localized by molecular mapping on potato chromosomes. *Ry_{sto}* (designated also as *Ry_{f_{sto}}*) derived from *S. stoloniferum* and *Ry_{adg}* from *S. tuberosum* ssp. *andigena* mapped on potato chromosomes XII (Flis et al. 2005; Song et al. 2005) and XI (Hämäläinen et al. 1997), respectively. The gene *Ry_{chc}* derived from *S. chacoense* mapped to the distal end of potato chromosome IX (Sato et al. 2006). The gene *Ry_{sto}* was also localized on chromosome XI (Brigneti et al. 1997), in the same region, where *Ry_{adg}* was mapped (Gebhardt and Valkonen 2001). Recently, however, the *Ry_{adg}*-linked markers used in the *S. stoloniferum* mapping experiments did not confirm the position of *Ry_{sto}* on potato chromosome XI (Valkonen et al. 2007).

The HR to PVY is strain specific in potato. Hypersensitivity to PVY⁰ and/or PVY^N was described in wild *Solanum* species (Valkonen 1997; Ruiz de Galarreta et al. 1998; Solomon-Blackburn and Barker 2001). HR was also observed in cultivated potato, however, only after infection with the ordinary strain of PVY (Jones 1990; Valkonen et al. 1998; Sorri et al. 1999). Potato cultivars expressing HR to PVY^N infection were not reported so far (Valkonen 2007). The first HR gene, *Ny_{thr}*, causing necrotic response to PVY⁰ infection in potato mapped on potato chromosome IV (Celebi-Toprak et al. 2002). In this paper, we report the first potato HR gene, which induces necrotic response and restriction of common and necrotic variants of PVY. The gene, designated as *Ny-1*, mapped on potato chromosome IX.

Materials and methods

Plant material and PVY strains

The tetraploid potato (*Solanum tuberosum* ssp. *tuberosum*, $2n = 4x = 48$) population 'RxA', consisting of 200 F₁ individuals, was obtained from a cross between Rywal, a cultivar with HR to PVY and the susceptible cv. Accent. Rywal originated from a cross of Polish cv. Dalia and Polish clone DM 1696. This clone was developed from Dutch cv. Edzina × Polish clone DM 51/5-7 cross. Further pedigree information for cultivars Dalia and Edzina, and clone DM 51/5-7 are compiled in Świeżyński et al. (1997) and the potato pedigree database (<http://www.dpw.wau.nl/pv>). Four PVY variants: PVY⁰LW, PVY^NNy, PVY^NW and

PVY^{NTN}Bo, which derived from the corresponding potato cvs: Lipiński Wczesny, Nysa, Wilga and Bona, were multiplied and maintained in cv. Samsun tobacco plants.

Resistance assays for PVY strains

Two types of assays were performed to evaluate the resistance of cvs Rywal and Accent to PVY strains. Plants were grown for 2 weeks in the greenhouse and transferred 1 week before the infection experiments to growth chambers with controlled environmental conditions (20 or 28°C, 16 h light, 100 μm/s/m², 8 h dark). For each cultivar, six detached leaves or six plants were mechanically inoculated with a sap extracted from the tobacco plants, infected with PVY⁰LW, PVY^NNy, PVY^NW or PVY^{NTN}Bo. Inoculated detached leaves and plants were divided into two groups. One group was incubated at 20°C, the other at 28°C. Water-treated detached leaves and plants were used as controls. Inoculated detached leaves were placed on a wet filter paper in two plastic trays, which were covered with glass plates to maintain high humidity. Hypersensitivity was visualized after 4–6 days. Nine days post-inoculation, 1 g samples of inoculated and non-inoculated upper leaves from each of the plants were collected in order to detect the virus by RT-PCR.

For the mapping experiments, two tuber-derived plants per clone of each F₁ individuals of the 'RxA' population were screened for HR to PVY^NW and PVY^{NTN}Bo infection at 20°C. In addition, three corresponding detached leaves were scored by inoculation with PVY^{NTN}Bo. Plants which developed necrotic symptoms were classified as resistant.

Detection of PVY RNA by RT-PCR

RNA was isolated using TRIZOL method (Chomczyński and Sacchi 1987). Reverse-transcription reactions contained 1 μg of DNase I treated RNA from PVY or mock inoculated plants. The reactions were primed with oligo(dT) and elongated by the PVY specific sequence (5'oligo(dTx30)GTCTCCTGATTGAAGTTTACAG3'). 200 U M-MuLV Reverse Transcriptase (Fermentas, Vilnius, Lithuania) were used to synthesize first-strand cDNA. PCR amplification from PVY coat protein RNA (PVYCP) was performed with the PVYCPf and PVYCPp primers for 30 cycles at 94°C for 30 s, 53°C for 30 s, and 72°C for 45 s. The primer sequences were: PVYCPf, 5'AAATACTCGG GCAACTCAATCACAA3'; PVYCPp, 5'CACCGTCCAA CCCGAAAAG3'. They were based on coat protein sequences for various PVY strains, available in GenBank database. PrimerSelect software from Lasergene 6.1 set (DNASTAR Inc., Madison WI, USA) was used for primer design.

Plant DNA isolation, PCR, electrophoresis, DNA cloning and sequencing

Extraction of plant genomic DNA and electrophoresis were performed as previously described (Flis et al. 2005). Inter-simple sequence repeat (ISSR) markers were amplified in 20 μ l of 20 mM Tris–HCl pH 8.4, 50 mM KCl, 2% DMSO, 2.5 mM MgCl₂, 0.2 mM of each deoxynucleotide, 0.3 μ M of primer, containing 1 U *Taq* DNA Polymerase (Invitrogen, Carlsbad, CA, USA) and 30 ng genomic DNA as template. The PCR parameters were: 40 cycles of 94°C for 20 s, 42°C for 30 s, 72°C for 2 min and a final extension time of 10 min at 72°C. The ISSR marker UBC895₁₂₀₀ was cloned and sequenced as previously described (Marczewski et al. 2006) and compared to the NCBI sequence database with the BLASTN program.

Three markers: sequence characterized amplified region (SCAR) SC895₁₁₃₉ (NCBI GenBank accession EF555209), GP41₄₄₃ (AJ487356) and conserved ortholog set II (COSII) C2_At3g16840₁₁₀₀ (SGN-U284031) were amplified in 20 μ l of 20 mM Tris–HCl pH 8.4, 50 mM KCl, 1.5 mM MgCl₂, 0.1 mM of each deoxynucleotide, 0.2 μ M of each primer, containing 1 U *Taq* DNA Polymerase (Invitrogen) and 30 ng genomic DNA as template. Forward and reverse primers, designed as described in Marczewski et al. (2006), were: f: 5'GGTAGCTCTTGATCTCGTCTT3', r: 5'GTA GCTCTTGATCACCCATTT3' for SC895; f: 5'GTTGGT ACCAGGCTTGTT3', r: 5'CATTTCGGTGCTTTAGGAT3' for GP41 and f: 5'TCCAGTGTCCAAAGAAAGAAA3', r: 5'ATGCTCATGTCCCCGAAACC3' for C2_At3g16840. The PCR parameters for amplifying SC895₁₁₃₉ and C2_At3g16840₁₁₀₀ were: 94°C for 60 s followed by 40 cycles of 93°C for 25 s, 56°C for 35 s, 72°C for 90 s and a final extension time of 5 min at 72°C. GP41₄₄₃ was amplified at the annealing temperature of 54°C.

Detection of PVY in field-grown plants

Four groups of cv. Rywal plants, 60 plants each, were subjected to PVY-exposure field trials at Młochów, during five vegetation seasons, 1999–2003. One group consisted of plants grown from the virus-free tubers, whereas in the other groups three successive tuber progenies were planted. Each third row of the field trials was planted with tubers of PVY-infected potato plants. In addition, during each year 120 plants grown from field tubers harvested in the north-eastern region of Poland were planted in a greenhouse at Młochów. The presence of PVY was tested in leaves of 5- to 6-week-old plants by enzyme-linked immunosorbent assay (ELISA) using the monoclonal cocktail Bioreba AG kit (Reinach, Switzerland).

Mapping of the locus *Ny-1*

The diploid mapping population 'Erwinia' (Zimnoch-Guzowska et al. 2000) was used to map the SCAR marker SC895₁₁₃₉ linked to the gene *Ny-1*. SC895₁₁₃₉, amplified from parental clone DG 81-68, was scored as presence or absence of the marker fragment in 78 F₁ progeny. The map position was identified relative to the amplified fragment length polymorphism (AFLP) and restriction fragment length polymorphism (RFLP) map existing for this population using the software package MAPRF (E. Ritter, NEIKER, 01080 Victoria, Spain). The position of *Ny-1* in cv. Rywal was confirmed in the population 'RxA' by detecting linkage to the anchor markers GP41 and C2_At3g16840, specific for potato chromosome IX (PoMaMo database, <http://gabi.rzpd.de/database/maps.shtml>) and tomato chromosome 9 (Tomato-EXPEN 2000 map, <http://www.sgn.cornell.edu/search/markers/markersearch.pl>), respectively.

Results

Reaction of cvs Rywal and Accent to infection with PVY strains

When detached leaves or whole plants of PVY susceptible cv. Accent were mechanically inoculated with strains PVY-^OLW, PVY^NNy, PVY^NW and PVY^{NTN}Bo, no symptoms were observed at 20 or 28°C (not shown). The systemic, symptomless infection was also associated with plants of cv. Rywal inoculated and grown at 28°C. In contrast, at 20°C, necrotic lesions were observed in leaves of cv. Rywal, 4–6 days post-inoculation for each strain (Fig. 1). The viral RNA was not detected in the upper, non-inoculated leaves of cv. Rywal collected 9 days after inoculation (Table 1). Increasing the temperature from 20 to 28°C allowed the virus to occasionally move from inoculated leaves to leaves on upper parts of the plant, which remained symptom-free but contained detectable amounts of PVY. The systemic virus spread did not occur when the inoculated leaves were detached from the plant prior to their transfer to the higher temperature (not shown). However, during 5 years of experiments for the field-grown plants, no more than occasional systemic infections with PVY, resulting in plant death, were observed.

Mapping of the gene *Ny-1*

Hypersensitive resistance was observed in 93 F₁ hybrids of the mapping population 'RxA' using both the detached leaf

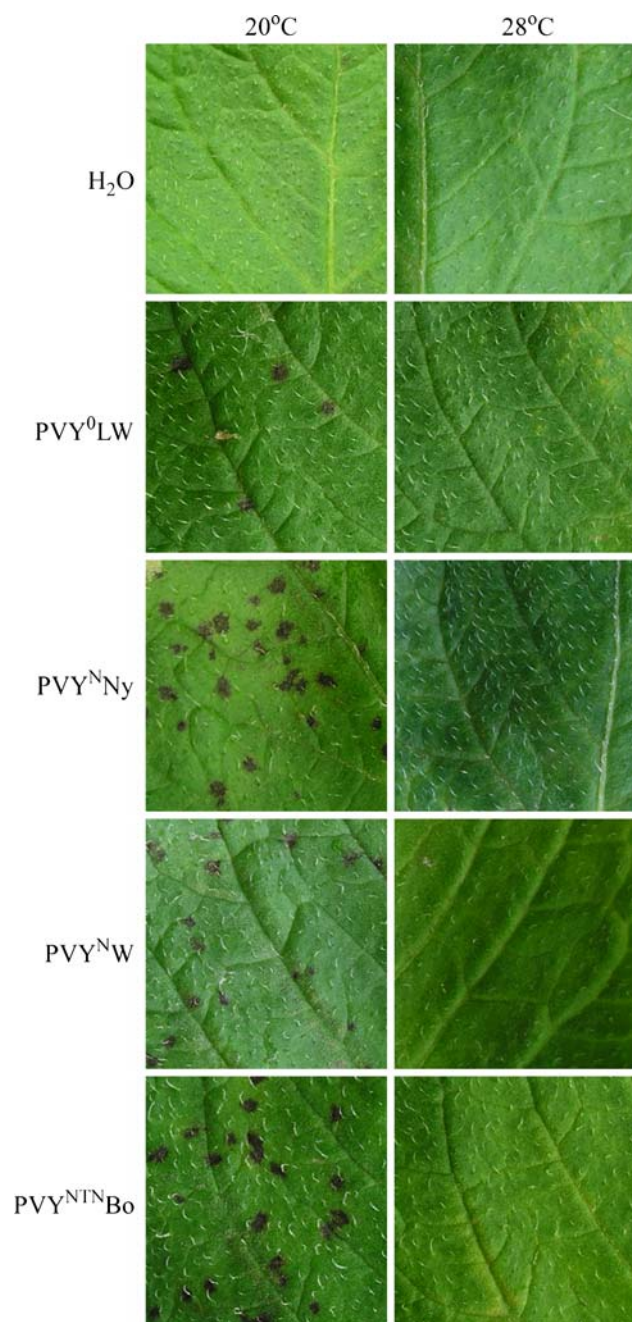


Fig. 1 Response of cv. Rywal plants to infection with PVY^{0LW}, PVY^NNy, PVY^{NW} or PVY^{NTN}Bo. The pictures of detached leaves incubated at 20 or 28°C were taken 6 days post-inoculation. Experiments were repeated twice with similar results

and the whole plant-inoculation tests. The PVY susceptible group consisted of 107 F₁ hybrids. The segregation ratio of 1:1 ($\chi^2 = 0.98$, $P = 0.32$) fits the genetic model of a single, dominant gene for HR to PVY in simplex state in cv. Rywal.

Seventy-eight ISSR primers, that were informative in previous studies (Flis et al. 2005), were tested by PCR in the parental DNA samples. Of 16 PCR products generated

Table 1 Presence of PVY RNA and symptoms in leaves of plants from cv. Rywal inoculated with PVY^{0LW}, PVY^NNy, PVY^{NW} or PVY^{NTN}Bo

Leaves	20°C		28°C	
	RT-PCR	Symptoms	RT-PCR	Symptoms
Water-treated	–	NS	–	NS
PVY-inoculated ^a	+	HR	+	NS
Non-inoculated ^b	–	NS	+	NS

Experiments were repeated twice with similar results

NS no symptoms; HR hypersensitive response; –, absence of amplicon; +, presence of PVY-specific product

^a Leaves collected 6 days post-inoculation

^b Upper leaves collected 9 days post-inoculation

specifically in the resistant parent cv. Rywal, only one, a 1,200 bp long DNA fragment amplified using primer UBC895 (5'AGAGTTGGTAGCTCTTGATC3') was seen as a faint band (Fig. 2, lane 1) in a bulked DNA sample constructed from eight HR progeny. The marker was not observed in the susceptible DNA bulk. UBC895₁₂₀₀ was cloned and sequenced in order to obtain more reliable and specific PCR amplification. Based on the cloned 1,152 bp marker, which revealed no apparent homology to any sequences available in GenBank, the SCAR marker SC895₁₁₃₉ (EF555209) was developed as the clear, strong PCR product, amplified in cv. Rywal (Fig. 2, lane 3). SC895₁₁₃₉ was used to screen the 200 F₁ individuals of the 'RxA' family. The marker fragment was observed in all 93 resistant and in 1 susceptible F₁ plant, indicating a close linkage (0.5 cM) between the SCAR marker and the locus *Ny-1*. Segregation of SC895₁₁₃₉ in the 'Erwinia' mapping population (Zimnoch-Guzowska et al. 2000) revealed the map position of *Ny-1* on the short arm of potato chromosome IX. The chromosome location was confirmed when a 443 bp sequence tagged site (STS) amplicon (Fig. 2, lane 5) of the chromosome IX specific marker GP41 (<http://gabi.rzpd.de/database/maps.shtml>) was positioned 1 cM from *Ny-1* in cv. Rywal. The locus *Ny-1* was also found to be separated by 6 cM from the COSII marker C2_At3g16840 described in the reference Tomato Genetic Map at position 88 cM on tomato chromosome 9 (Wu et al. 2006). C2_At3g16840 was scored based on two restriction fragments of 350 and 500 bp, generated after *TaqI* digestion of the approximately 1,100 bp amplicon (Fig. 2, lane 7). The marker GP41 was found in the Tomato-EXPEN 1992 genetic map at position 104.8 cM on chromosome 9 (<http://www.sgn.cornell.edu/cview>). The syntenic relationships between tomato and potato genomes implicate, therefore, the proximal orientation of C2_At3g16840 and the distal locations of SC895₁₁₃₉ and GP41 in the linkage group IX (Fig. 3).

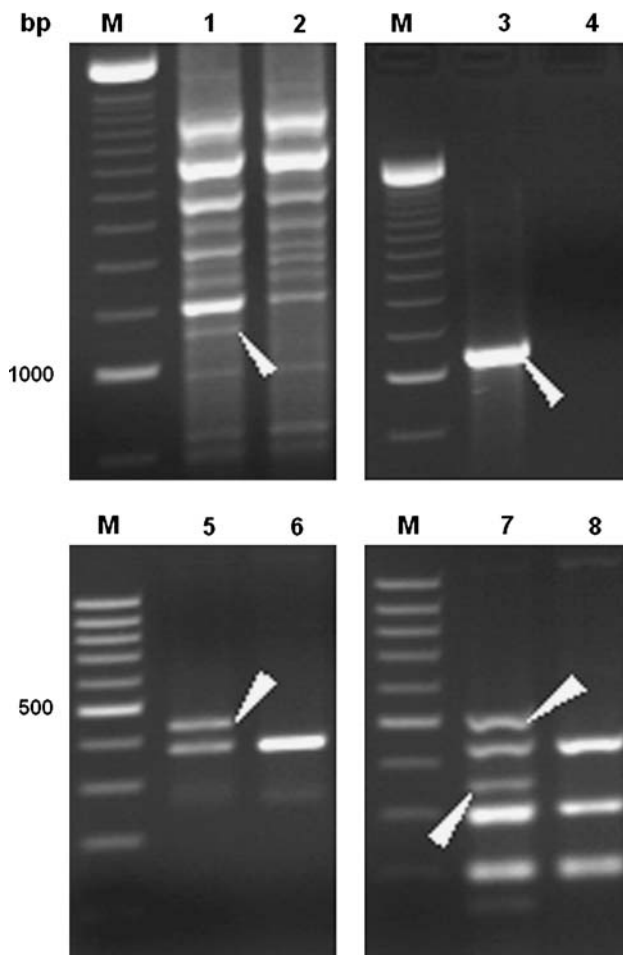


Fig. 2 Patterns of amplified DNA of resistant parent cv. Rywal (lanes 1, 3, 5 and 7) and susceptible parent Accent (lanes 2, 4, 6 and 8), indicating the ISSR marker UBC895₁₂₀₀ (lane 1), the STS marker SC895₁₁₃₉ (lane 3), the SCAR marker GP41₄₄₃ (lane 5) and the COSII marker C2_At3g16840 after digestion with *TaqI* (lanes 7 and 8). Arrows point to the marker products that were scored. Lanes M contain the 250-bp (upper) and 100-bp (lower) DNA ladders as molecular size markers

Discussion

Plant resistance is often accompanied by rapid tissue necrotization at pathogen entry sites, referred to as HR. High temperature may induce a switch from necrotic reactions to absence or mild systemic symptoms in virus infected plants (Malamy et al. 1992; Zheng et al. 2005). For example, HR to PVY^N in *S. sparsipilum* and *S. sucrense* and to PVY⁰ in potato cv. Pito was efficiently expressed at temperatures 16–18°C, indicated by the local and/or systemic necrosis, while at higher temperatures (19/24°C), only leaf drop and mosaic symptoms were developed (Valkonen 1997). The temperature-sensitive response was also observed in potato plants with the genes *Nx* and *Nb*, which induced HR to

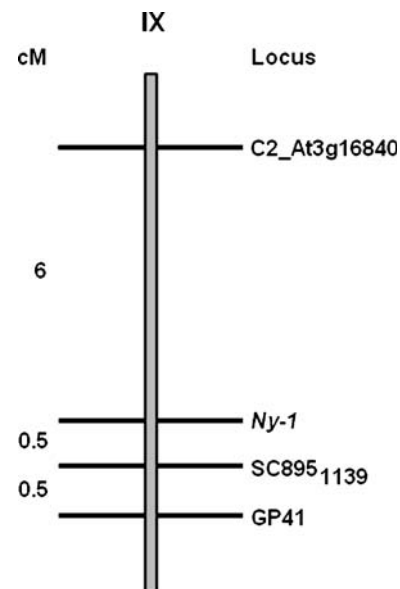


Fig. 3 Potato linkage group IX of PVY resistant parent cv. Rywal, including the position of the locus *Ny-1*. The tetraploid population obtained from a cross between potato cultivars Rywal and Accent was used for the mapping

Potato virus X (PVX) infection (Adams et al. 1986). HR induced by *Ny-1* in cv. Rywal was efficiently developed at 20°C, whereas local necrotic lesions were not revealed in plants grown constitutively at elevated (28°C) temperature. Moreover, this temperature allowed the plants to be systemically infected with PVY⁰LW, PVY^NNy, PVY^NW or PVY^{NTN}Bo with the absence of viral symptom expression. In Poland, potato cultivars are grown between April and August, when daily minimum and maximum temperatures may reach the values below 10°C and above 35°C. However, throughout 5 years of the field experiments at Młochów plants of cv. Rywal produced PVY-free tubers. Młochów is located in the central region of Poland, where PVY infection pressure is intense (Gabriel 1995). PVY was also not revealed in tubers collected in the northeastern region of Poland. Moreover, during several years of cv. Rywal reproduction at the Plant Breeding Station Sztydak, northern Poland, harvested tubers were PVY-free (R. Bruski, personal communication). It is possible that the natural PVY infection pressure caused by aphids, which transmit PVY non-persistently, was too slow to break the virus restriction at the site of infection and activate its systemic movement in *Ny-1* possessing plants, even at the higher temperatures. To our knowledge cv. Rywal is the first commercial potato cultivar, in which HR to both common and necrotic PVY strains, including PVY^NW and PVY^{NTN}, was effectively expressed.

The extreme resistance gene *Ry_{che}* mapped recently on potato chromosome IX in Japanese cv. Konafubuki was tightly linked to the marker TG421 (Sato et al. 2006). The

Ny-I-linked marker C2_At3g16840 was found to be linked to TG421, within 5 cM, in the reference Tomato-EXPEN 2000 map (<http://www.sgn.cornell.edu/search/markers/markersearch.pl>). Therefore, *Ry_{chc}* and *Ny-I* may be new members of the resistance hot spot on potato chromosome IX, in which resistance genes and quantitative trait loci to various pathogens have been previously described (Marczewski et al. 2006; Rouppe van der Voort et al. 2000; Simko et al. 2004; Smilde et al. 2005; Śliwka et al. 2006; Tommiska et al. 1998; Zimnoch-Guzowska et al. 2000).

Ry genes for extreme resistance to PVY *Ry_{sto}* have been introgressed mainly into European potato cultivars (Flis et al. 2005; Song et al. 2005). However, ER cultivars and breeding lines often exhibit male sterility (Lössl et al. 2000), which limits their usefulness in potato breeding programs. Cultivars and breeding clones derived from Polish, German, Dutch and North American gene pools are in the pedigree of cv. Rywal. Hypothetically, some of them can possess the gene *Ny-I*, and the *Ny-I*-linked markers should be helpful for their selection. Therefore, we postulate that *Ny-I*, which provides a level of the field resistance to PVY as satisfactory as the *Ry* genes, may be available and useful in several potato breeding programs.

Acknowledgments We thank Bogdan Flis and Ewa Zimnoch-Guzowska for helpful comments on the manuscript and Andrzej Pałucha for the oligo(dT)-PVY primer sequence. The research was supported by the State Committee for Scientific Research in Poland, project PBZ/KBN/110/PO4/2004.

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