

SHORT REPORT

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Differential pathogenicity of two different recombinant PVY^{NTN} isolates in *Physalis floridana* is likely determined by the coat protein gene

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Abstract

A previous study has identified two types of recombinant variants of *Potato virus Y* strain NTN (PVY^{NTN}) in China and sequenced the complete genome of the variant PVY^{NTN}-HN2. In this study, the complete genome of isolate PVY^{NTN}-HN1 was fully sequenced and analyzed. The most striking difference between the two variants was the location of recombinant joint three (RJ3). In PVY^{NTN}-HN1, like other typical European-PVY^{NTN} isolates such as PVY^{NTN}-Hun, the RJ3 was located at nucleotide (nt) 9183, namely the 3' proximal end of the CP gene (nt. 8571-9371), thus leading to most (the first 613 nucleotides from the 5' proximal end) of the CP gene (801 bp) with a PVY^N origin and PVY^N-serotype; whereas in contrast, the RJ3 in PVY^{NTN}-HN2 was located at nt 8572, consequently leading to a CP gene of PVY^O origin and PVY^O-serotype. The varied genome composition among PVY^O, PVY^N, PVY^{N:O}, PVY^{NTN}-HN1 and PVY^{NTN}-HN2 made them useful for the investigation of possible roles of gene segment(s) in symptom formation on host plants. When *Physalis floridana* plants were infected with different PVY isolates, two types of symptoms were induced. PVY^N and PVY^{NTN}-HN1 induced mild symptoms (mainly mild mottling) whereas PVY^O, PVY^{N:O} and PVY^{NTN}-HN2 induced severe symptoms including leaf and stem necrosis, leaf-drop and stunting. These results, together with a previous study using artificial PVY chimeras, demonstrate that the CP gene, especially the 5' proximal segment (nt 8572-9183), and/or CP likely determine the pathogenicity of PVY in *P. floridana*.

Findings

Potato virus Y (PVY) is the type species of the *Potyvirus* genus in the *Potyviridae* family [1]. It infects a number of plant species in the nightshade family (*Solanaceae*) and causes a wide range of symptoms from symptomless to mosaic, mottling, lesions, stunting, necrosis and plant death, depending on the plant species, the cultivar, the virus strain and isolate [1]. PVY possesses a single-stranded positive RNA genome comprised of approximately 9700 nucleotides that encode a polyprotein of approximately 3061 amino acids [2]. The polyprotein undergoes proteolysis to form 10 mature proteins with different functions including replication, transportation and spread of the virus [1,2]. Many strains/substrains of PVY have been recognized according to the primary hosts and host reactions. For the potato-infecting PVY, the ordinary strain (PVY^O), the tobacco vein necrosis

strain (PVY^N) and the potato stipple streak strain (PVY^C) are the first ones to be recognized [1], followed by the potato tuber necrosis strain (PVY^{NTN}) and the recombinant N:O/Wilga group (PVY^{N:O} or PVY^N-Wilga) [3-5]. PVY^{NTN} is characterized by its ability to induce potato tuber necrotic ringspot disease (PTNRD) in sensitive potato cultivars [5-7], whereas PVY^{N:O} is defined by its reaction to PVY^O-specific antibody (i.e., PVY^O-serotype) but causing vein necrosis on tobacco plants (i.e., PVY^N pathotype) [5,8]. Two types of PVY^{NTN}, one recombinant and the other non-recombinant, have been identified [4,7,9]. The former is represented by PVY^{NTN}-Hun [10] and has been referred to as European (Eu)-PVY^{NTN} [7,11-13], and the latter is represented by PVY^{NTN}-Tu 660 [7] and has been referred to as North American (NA)-PVY^{NTN} [7,11,12,14]. Both Eu-PVY^{NTN} and NA-PVY^{NTN} react to PVY^N-specific antibody [11,13]. Recently, a new recombinant PVY^{NTN} variant type has been identified in Syria [15,16] and China [9]. The variant type that includes the isolates PVY^{NTN}-NW [16] and PVY^{NTN}-HN2 [9] reacts

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to PVY^O-antibody and induces veinal necrosis on tobacco and PTNRD on sensitive potato cultivars [9,16]. Reverse transcription-PCR (RT-PCR) based genotyping has been successfully used to characterize the genome features of the Eu-PVY^{NTN}-like isolate PVY^{NTN}-HN1 and the PVY^{NTN-NW}-like isolate PVY^{NTN}-HN2 in China [9]. Here we report the differential responses of *Physalis floridana* to PVY^{NTN}-HN1 and PVY^{NTN}-HN2 infections. PVY^{NTN}-HN1 and PVY^N induced mottling on *P. floridana* whereas PVY^{NTN}-HN2, PVY^O and PVY^{N:O} induced severe symptoms including leaf and stem necrosis, leaf-drop and stunting. The results, together with the genome make-ups of various PVY isolates, suggest that the CP gene plays a significant role in symptom induction in *P. floridana*, consistent with the results reported by Bukovinszki *et al.* [17].

The greenhouse maintained PVY isolates PVY^{NTN}-HN1 (formerly PVY sample 1 [9]), PVY^{NTN}-HN2, PVY^N-Jg, PVY^O-RB and PVY^{N:O}-Mb58 in 'Russet Burbank' plants/tubers [5,7,9,11-13] were used in this study. PVY^{NTN}-HN1 and PVY^{NTN}-HN2 were obtained in China [9], while the rest were from Canada [5,7,11-13]. All isolates have been characterized molecularly by P1 gene- and recombinant joint (RJ)-based RT-PCR assays [12,13], pathologically by tobacco- and potato-based bioassays, and serologically by PVY^O- and PVY^N-antibody-based ELISA assays [5,7,9,11-13,18]. Moreover, except for PVY^{NTN}-HN1, all of the isolates have been sequenced fully (PVY^{NTN}-HN-2, PVY^N-Jg, PVY^O-RB) or partially (PVY^{N:O}-Mb58) (accession numbers are HM367076, AY166867, GQ200836, AY745493 for PVY^O-RB, PVY^N-Jg, PVY^{NTN}-HN-2, and PVY^{N:O}-Mb58 respectively). To better understand the isolate PVY^{NTN}-HN1, especially to reveal the exact nucleotide locations of the recombinant joints that had been detected by RT-PCR [9], the complete genome of PVY^{NTN}-HN1 was sequenced. The same nine sets of PCR primers (for primer sequences, see reference [7]) that had been used to clone/sequence various isolates of PVY [5,7,9,18] were used. Each primer pair resulted in a DNA fragment of 1.0 to 1.3 kb, overlapping with adjacent fragments with approximately 100 bp at each end. Each fragment was cloned into a pGM-T cloning vector (TIANGEN Biotech, Beijing, China) according to the manufacturer's instructions; and two clones of each fragment were sequenced from both forward and reverse directions using the universal T7 promoter and SP6-promoter primers at the Sangon Biological Engineering Technology & Services Co. Ltd (Shanghai, China). The complete genome sequence (GenBank accession number HQ631374) was confirmed by re-sequencing overlapping cDNA clones obtained from a separate experiment from RNA isolated from PVY^{NTN}-HN1 infected tobacco leaves. Sequence identities were analyzed using BLAST

(http://www.ncbi.nlm.nih.gov/BLAST). For detection of the recombinant events, complete nucleotide sequences of various PVY isolates were aligned using ClustalW2 (http://www.ebi.ac.uk/Tools/clustalw2/index.html) [19]. The aligned sequences served as inputs for similarity scanning using the program SimPlot [20, generously provided by the author at http://sray.med.som.jhmi.edu/]. The resulting similarities were plotted along the nucleotide sequences of the virus genome.

As anticipated, PVY^{NTN}-HN1 shared highest sequence identities with PVY^{NTN}-Hun, a representative of typical Eu-PVY^{NTN}, at both complete nucleotide and polyprotein levels at 99.2% and 99.1%, respectively. It was followed by PVY^{NTN}-HN2/PVY^{NTN-NW}, PVY^{N:O}, PVY^N and PVY^O, represented by isolates PVY^{NTN}-HN2, PVY^{N:O}-Mb112, PVY^N-N605 and PVY^O-RB, respectively (Table 1). The sequence identities between PVY^{NTN}-HN1 and PVY^N-Jg, a NA- PVY^N [7,11], were 90.9% and 95.9% at the complete nucleotide and polyprotein levels, respectively (Table 1). As expected, the sequence identities between PVY^{NTN}-HN1 and PVY^{N:O}-Mb58 (accession number AY745493, partial length) were similar to that between PVY^{NTN}-HN1 and PVY^{N:O}-Mb112 (data not shown). Further comparison of PVY^{NTN}-HN1 with PVY^{NTN}-HN2 at mature protein level revealed that the two shared high sequence identities for all proteins (97.8 - 100%) but the CP (Table 1), which was similar to that exhibited in PVY^{NTN}-Hun vs PVY^{NTN}-HN2 [9]. Sequence screening of PVY^{NTN}-HN1 against PVY^O (e.g., PVY^O-RB) and PVY^N (e.g., PVY^N-605 or PVY^N-Jg) using SimPlot [20] revealed three recombinant joints at nt 2419, 5844 and 9183 in PVY^{NTN}-HN1 genome (Figure 1D), resulting from the genome recombination between PVY^N and PVY^O. In contrast, the RJs in PVY^{NTN}-HN2 were located at nt 2521, 5867 and 8572 (Figure 1D) [9]. PVY^{NTN}-HN1 shares identical RJs with PVY^{NTN}-Hun (data not shown). The location of RJ3 in PVY^{NTN}-HN1 and PVY^{NTN}-Hun at nt 9183, namely the 3' proximal end of CP gene (nt 8571-9371), led to most (the first 613 nt from the 5' proximal end) of the 801-bp-long CP gene of a PVY^N origin, which eventually resulted in a PVY^N-serotype of these isolates [5,7,9]. In contrast, the RJ3 in PVY^{NTN}-HN2/PVY^{NTN}-SYR-NB-16N (accession number AB270705) at nt 8572, namely the 5' end of the CP gene, led to the complete CP gene of a PVY^O origin, which further resulted in a PVY^O-serotype [9,15]. One RJ, namely RJ1, was present in PVY^{N:O} isolates including PVY^{N:O}-Mb112 and PVY^{N:O}-Mb58 at nt 2397 [5] (Figure 1D), resulting in a recombinant genome in which the segment prior to the RJ was from PVY^N and the remainder from PVY^O [5].

Previous study has revealed the pathotypes of PVY^{NTN}-HN1 and PVY^{NTN}-HN2 on tobacco and potato [9]. To further characterize the biological properties of

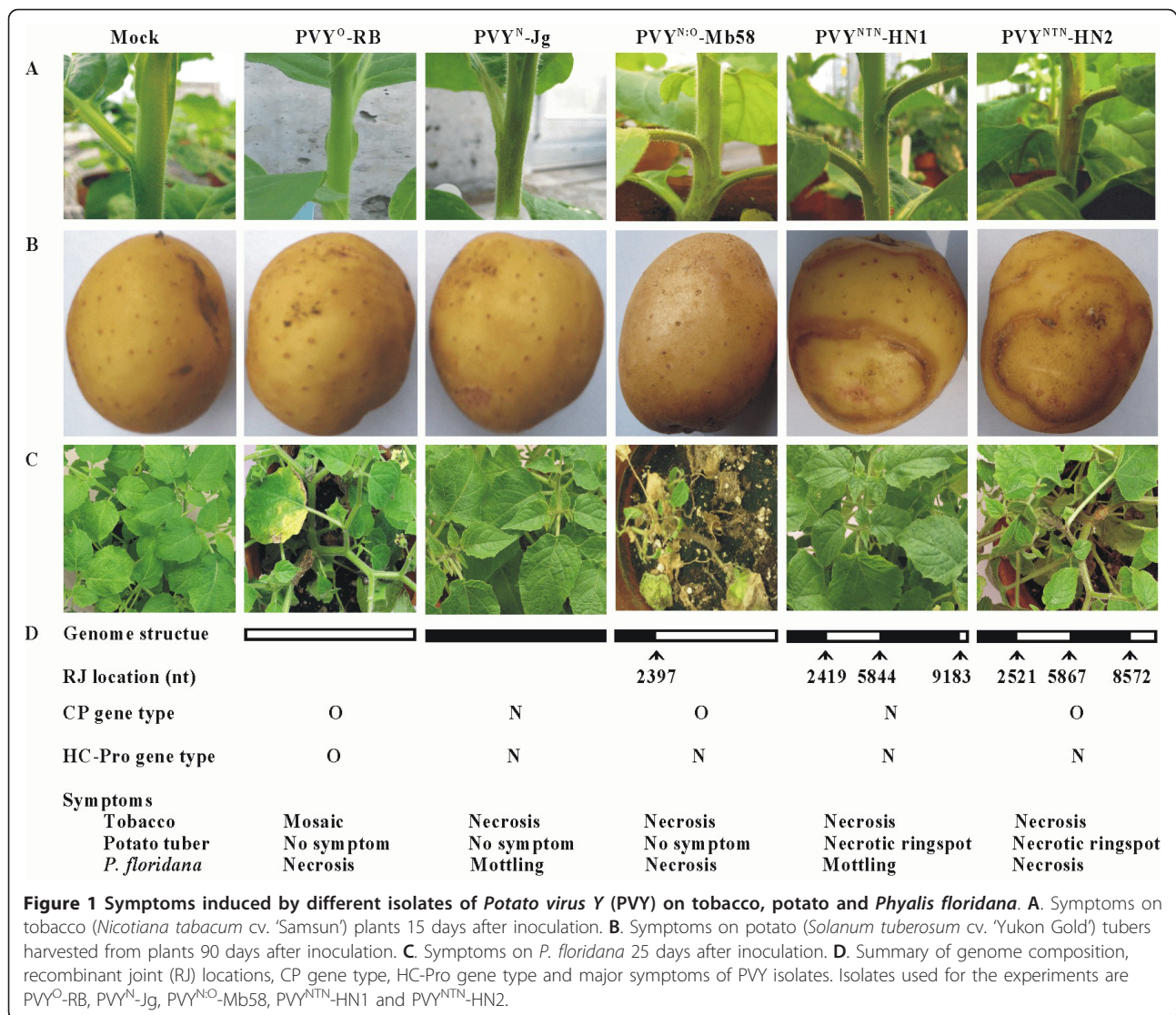
Table 1 Identities between isolate PVY^{NTN}-HN1 and other isolates of *Potato virus Y* (PVY) at both nucleic acid and protein levels

	Length		Sequence Identity (%) (Nucleic acid, Protein)					
	Gene nucleotide, size (bp)	Protein size (aa)	PVY ^O -RB (HM367076)	PVY ^N -605 (X97895)	PVY ^N -Jg (AY166867)	PVY ^{NTN} -Hun (M95491)	PVY ^{N:O} -Mb112 (AY745491)	PVY ^{NTN} -HN2 (GQ200836)
5' UTR	1-188, 188	-	66.0, -	100, -	85.6, -	100, -	100, -	99.5, -
P1	189-1013, 825	275	73.0, 71.3	99.3, 99.6	92.2, 91.3	98.7, 98.2	99.6, 100	98.9, 100
HC-Pro	1014-2408, 1395	465	82.2, 90.5	98.8, 99.4	92.6, 96.8	99.1, 99.1	98.4, 99.4	98.4, 98.9
P3	2409-3503, 1095	365	97.3, 98.6	84.7, 92.6	84.6, 92.3	99.4, 99.7	99.1, 99.7	96.3, 97.8
6K1	3504-3659, 156	52	97.4, 100	81.4, 84.6	82.1, 86.5	99.4, 100	100, 100	98.1, 98.1
CI	3660-5561, 1902	634	97.3, 99.1	84.1, 95.7	84.1, 95.7	99.6, 99.5	99.3, 99.5	98.7, 99.7
6K2	5562-5717, 156	52	95.5, 100	82.1, 90.4	79.5, 90.4	100, 100	100, 100	99.4, 98.1
VPG	5718-6281, 564	188	86.9, 92.0	97.0, 99.5	95.6, 97.3	98.9, 99.5	87.8, 94.7	97.9, 99.5
NIa	6282-7013, 732	244	80.9, 93.6	99.3, 98.2	97.0, 98.8	99.2, 98.8	80.6, 92.2	99.0, 99.2
NIb	7014-8570, 1557	519	83.5, 93.6	98.8, 99.0	97.6, 98.7	98.7, 98.7	84.9, 94.4	98.2, 99.0
CP	8571-9371, 801	267	90.8, 92.9	97.5, 98.9	96.5, 98.9	99.4, 98.9	91.4, 94.4	91.6, 94.0
3' UTR	9372-9702, 331	-	98.2, -	84.0, -	85.8, -	99.1, -	98.8, -	99.1, -
Full length	1-9702, 9702	3061	87.9, 92.8	93.2, 97.3	90.9, 95.9	99.2, 99.1	94.1, 97.4	97.7, 98.7

these isolates, and, moreover, to investigate whether the different RJ3 sites, namely different CP gene types, play a role in symptom induction in different plant species, tobacco (cv. 'Samsun'), potato (cv. 'Yukon Gold') and *Physalis floridana* plants were mechanically inoculated with PVY^{NTN}-HN1, PVY^{NTN}-HN2, PVY^N-Jg, PVY^O-RB, and PVY^{N:O}-Mb58 as described previously [9]. Mock (buffer)-inoculated plants were used as a healthy control. As shown in Figure 1A, petiole and stem necrosis occurred on tobacco plants 15 days after inoculation with PVY^N-Jg, PVY^{N:O}-Mb58, PVY^{NTN}-HN1 or PVY^{NTN}-HN-2. Veinal necrosis also developed on these plants. On the other hand, the PVY^O-infected plants only developed mosaic symptoms on the leaves and were free of veinal, petiole and stem necrosis (Figure 1A). No symptoms were observed on the mock-inoculated plants. Various studies have indicated that HC-Pro plays an important role in necrosis development on tobacco plants [5,21,22]. All isolates but PVY^O-RB possessed a PVY^N-type of HC-Pro gene [5,7,9,18] (Figure 1D), and therefore induced PVY^N-like symptoms including veinal/petiole/stem necrosis on tobacco plants. When inoculated to 'Yukon Gold' plantlets (5-leaf-stage), the isolates induced varied foliar symptoms including mild mottling (PVY^N-Jg), mosaic (PVY^{N:O}-Mb58) and severe mosaic/stunting/leaf deformation (PVY^O-RB, PVY^{NTN}-HN1 and PVY^{NTN}-HN2) (data not shown), consistent with the previous report [9]. No visible symptoms were observed on potato tubers produced from plants infected with PVY^N-Jg, PVY^O-RB or PVY^{N:O}-Mb58; and in contrast, distinct necrotic ringspots were observed on potato tubers harvested from plants infected with PVY^{NTN}-HN1 or PVY^{NTN}-HN2 (Figure

1B), thus confirming that both types of recombinant PVY^{NTN} isolates are capable of inducing PTNRD in sensitive potato cultivars.

It has been known that PVY^O induces necrosis in *Physalis floridana*, whereas PVY^N incites mottling in this species [1]. Using N/O hybrids comprised of the chimeric genome of PVY^N-N605 [23] and PVY^O, the symptom formation on *P. floridana* due to PVY infection was mapped to the CP gene region [17]. Because of the varied genome compositions among the isolates (Figure 1D), they could be used to investigate the putative role of genome segment(s) of PVY in symptom development on *P. floridana*, as done on tobacco [5,24]. Severe symptoms including leaf and stem necrosis, leaf-drop and stunting were observed on *P. floridana* plants infected with PVY^O-RB, PVY^{N:O}-Mb58 and PVY^{NTN}-HN2 three weeks after inoculation (Figure 1C), and as time progressed, the symptoms became more distinct. The isolate PVY^{N:O}-Mb58 led to plant death five weeks after the inoculation. On the other hand, mild symptoms, mainly mottling, were observed on PVY^N-Jg and PVY^{NTN}-HN1 infected *P. floridana* plants (Figure 1C). Taken together, it can be concluded that the CP gene originated from PVY^O is likely responsible for the severe symptoms in PVY^O-, PVY^{N:O}- or PVY^{NTN}-HN2-infected *P. floridana* plants. These results, together with the results obtained using artificial PVY chimeras [17], demonstrate that the CP gene, especially the 5' proximal segment (nt 8572-9183) of the gene, plays a critical role in symptom formation in *P. floridana* upon PVY infection, and determines the pathogenicity of PVY isolates. The 3' proximal segment of NIb gene (nt 8136-8570) does not appear to be involved in the symptom



formation in *P. floridana* as suggested by Bukovinszki *et al.* [17]. It is also noteworthy that the different symptoms incited by different PVY types/isolates in tobacco, potato and *P. floridana* can be used to uncover the genome compositions of the virus.

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Authors' contributions

XH carried out the experiments. XN designed, analyzed and wrote the paper. HC and XX collected isolates PVY^{NTN}-HN1 and PVY^{NTN}-HN2, participated in experiment planning and execution. All authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

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