



# Unraveling the dynamics of host specificity and resistance responses to potato virus Y, and implications for crop management

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## Abstract

Potato virus Y (PVY), a virus member of the family *Potyviridae*, poses a significant threat to global agriculture, affecting crops such as potato, tomato, pepper, and tobacco. Despite its economic importance, there remains a critical gap in understanding the dynamics of PVY-host interactions and the development of effective management strategies. This study aimed to comprehensively characterize PVY isolates from sweet pepper, potato, and tomato plants, elucidating their infectivity and adaptation across diverse host species and cultivars. Initially, using antigen-trapped ELISA, we determined the optimal detection timeframe and leaf sampling strategy for detection of PVY by serological assays, showing that some hosts require a minimum incubation period and leaf selection for a reliable virus detection. By comparing PVY isolates from distinct hosts, we demonstrated that the choice of the isolate is crucial for resistance evaluations. Additionally, inoculation trials across various plant species elucidated differences in infectivity and adaptation among PVY isolates. Resistance trials in commercial cultivars of tomato and pepper plants and wild *Solanum* spp. accessions revealed susceptibility across all tested materials, challenging previous assumptions of resistant cultivars and accessions. These findings underscore the urgency of addressing PVY spread and understanding host-virus interactions to identify resistant genotypes for commercial use and for developing breeding programs directed to PVY isolates present in Brazil.

**Keywords** Host range · Plant breeding · *Potyviridae* · *Potyvirus* · Resistance screening · Viral adaptation

According to the latest update from the International Committee on Taxonomy of Viruses, potato Y virus (PVY) is classified within the species *Potyvirus yituberosi* (genus *Potyvirus*, family *Potyviridae*). It possesses a positive single-stranded RNA genome of approximately 9.7 kb in length, encoding 11 mature proteins (Inoue-Nagata et al. 2022). Ten proteins, P1, HC-Pro, P3, 6K1, CI (cylindrical inclusion), 6K2, NIa, VPg, NIb (viral polymerase), and CP (capsid protein), are derived from the cleavage of a larger

polyprotein by viral proteases. One protein, PIPO, is generated by a polymerase slippage mechanism and is expressed as the trans-frame protein P3N-PIPO. PVY stands as a serious viral threat in global agriculture, affecting crops such as potato, tomato, pepper, and tobacco (Quenouille et al. 2013). In fact, PVY has been considered a major threat to global potato production due to its high prevalence and ability to rapidly spread through fields (Karasev and Gray 2013). Its detrimental impact on crop yield is also relevant in tomato and pepper crops, underscoring the necessity for comprehensive research to identify resistant cultivars amidst its high prevalence and rapid spread in fields (Karasev and Gray 2013). Despite its importance, the current tomato portfolio of cultivars lacks a comprehensive description of resistance against PVY, thereby requiring further investigation.

Studies have revealed the substantial economic losses PVY can induce, with sweet pepper crops experiencing yield reductions ranging from 20 to 70% upon infection, particularly severe during early stages (Avilla et al. 1997). While the exact economic impact on tomato crops remains

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unquantified, its significant effects are well-documented (Quenouille et al. 2013). Thus, PVY remains a relevant concern to agriculture, threatening both yield and economic stability.

Historically, PVY posed a significant threat to Brazilian agriculture during the 1960s and 1970s. However, the development of resistant tomato cultivars, such as those in the Angela group, and hybrid peppers has substantially mitigated its impact (Nagai and Costa 1969; Nagai 1971). The rare reports of PVY occurrence in Brazilian tomato and pepper fields further diminished its economic significance in these crops (Meissner et al. 1990). Yet, recent observations suggesting a new disease named “Mexican Fire” in plants infected with PVY, highlight the resurgence of PVY in tomato fields, underscoring the potential re-emergence of this virus as a serious threat in Brazil (Lucena et al. 2025).

PVY is a generalist virus and exhibits a broad host range, experimentally infecting over 400 species across 30 families (Edwardson and Christie 1997; Jeffries 1998) and understanding the host range of viruses is crucial for virus diagnosis (Dijkstra 1992; McLeish et al. 2019). However, the determinants of host range in plant virus genomes and their implications for virus fitness and pathogenicity remain largely unknown. Despite this, it is known that the inability of a virus to infect a particular plant host may arise from various factors, including the failure to complete essential steps of the infection cycle, such as replication or systemic movement, or the presence of active and specific resistance mechanisms within the plant (Kang et al. 2005). Additionally, host range expansion is a common phenomenon among plant viruses, often at the cost of reduced fitness in the original host (Agudelo-Romero and Elena 2008; Bedhomme et al. 2012; García-Arenal and Fraile 2013). Furthermore, after serial passages in a specific host, the infectivity in the original host can diminish, suggesting potential constraints on a virus adapted to one host's ability to infect another one within its host range (Yarwood 1979). This implies that a virus adapted to one host may not necessarily be able to infect another host within its host range.

Nevertheless, even among generalist viruses, significant host-virus associations exist, with host specialization emerging as a successful strategy for increased prevalence (Malpica et al. 2006). Such specialization often involves genetic changes within the virus genome, potentially leading to alterations in host range. Additionally, host jumping and adaptation within plant species are not sporadic events in plant virus evolution but rather significant drivers of viral emergence (Vassilakos et al. 2016). These events carry epidemiological consequences, impacting viral survival and spread. Therefore, elucidating virus-host interactions holds immediate implications for control measures.

PVY exists as a complex of strains, delineated based on host range, serological properties and molecular

characteristics (Singh et al. 2008). These strains are generally classified as PVY<sup>C</sup>, PVY<sup>O</sup>, and PVY<sup>N</sup>. Studies investigating different PVY strains have revealed exceptional nucleotide diversification through mutation and/or recombination, enabling adaptation to new cultivars or diverse environments and resulting in varying degrees of infectivity (Karasev and Gray 2013; Nigam et al. 2019). PVY<sup>O</sup> and PVY<sup>N</sup> predominantly comprise potato isolates, which are less adept at infecting peppers, while PVY<sup>C</sup> primarily consists of pepper isolates with limited adaptation to potato (Moury 2010). However, it is noteworthy that the PVY<sup>C</sup> clade also includes potato-infecting isolates (Dullemans et al. 2011). Interestingly, in tomato fields, a PVY<sup>C</sup> isolate from commercial tomato production was grouped within the same clade as potato-infecting isolates but exhibited an inability to infect potatoes (Chikh-Ali et al. 2016). In addition to the C, O, and N strains, a large number of recombinants can be easily found, particularly in potato production fields, where they are more prevalent than non-recombinant strains (Galvino-Costa et al. 2012; Karasev et al. 2011). This prevalence poses a challenge for developing PVY-resistant potatoes, as there are currently no resistant cultivars available.

Phylogenetically, the host species appears to significantly influence the distribution of PVY, as evidenced by studies demonstrating differential infectivity among isolates across hosts (Cuevas et al. 2012). This effect becomes apparent when certain isolates successfully infect one host while failing to do so in others (Green et al. 2017).

Therefore, our study aims to address basic concepts of virus-hosts interaction at a mechanically inoculation and detection level, filling this gap in knowledge by understanding (1) the dynamics between three PVY isolates, identified in three distinct host species, and (2) the capacity to infect its original host and other hosts. We also consider the recent increase in incidence of PVY in tomato crops (Lucena et al. 2025) and search for resistant commercial sweet pepper and tomato cultivars, alongside wild solanum lines utilized in breeding programs. Our findings yield valuable insights that can contribute to breeding programs and help understanding the intricate dynamics of PVY-host interactions.

First, we used three PVY isolates collected from different hosts: PVYCa collected from a sweet pepper (*Capsicum annuum*) plant, PVYSt from a potato (*Solanum tuberosum*) plant and PVYSI from tomato (*Solanum lycopersicum*), all of them from the district of PAD-DF, close to Brasília, the Federal District in Brazil. Seeds were sown in polystyrene trays containing 128 cells and subsequently transplanted to 500 mL pots, containing organic potting mix and substrate (1:1 ratio), and kept in a greenhouse.

For all trials, the detection was done by antigen-trapped ELISA in nitrocellulose membranes (dot-ELISA) using a house made polyclonal antibody at a concentration of 1 µg/mL, as described in Nagata et al. (1995). This antibody

detects both PVY<sup>O</sup> and PVY<sup>N</sup> strains (Inoue-Nagata et al. 2001; Fonseca et al. 2005). The crude sap diluted in 0.5x PBS of each sample was applied on a nitrocellulose filter and treated with 1 µg/mL anti-PVY after blocking with skimmed milk, and later with anti-rabbit IgG alkaline phosphatase-conjugated antibody produced in goat (Sigma-Aldrich), diluted 1:30,000. Samples were considered positive if a purple color developed after incubation with a solution with nitro blue tetrazolium (NBT) and 5-bromo-4-chloro-3-indolyl-phosphate (BCIP) by visual inspection.

In a pilot test, the detection of PVY in sweet pepper cv. Ikeda, our model cultivar, by serology proved to be challenging due to low level of detection in early post-inoculation stages (*data not shown*). Due to this, the optimal time for inoculum collection was determined by testing the second and third leaves of plants 3, 5, 7, 9, 11, and 13 days post-inoculation (d.p.i.) of cv. Ikeda, using 10 plants each. Our aim was to determine which leaves, and the minimal time to collect samples to avoid false negative results. The inoculation was done using leaves of infected plants ground (~1:10) in 0.05 M phosphate buffer, pH 7.0, in plants with 2–4 true leaves. At this stage, we used the PVYCa and PVYSt isolates due to their ability to infect pepper plants (*data not shown*).

The serological test demonstrated that PVY remained undetectable until 13 d.p.i. under the tested conditions, regardless of the PVY isolate. This implies that the virus remains below detection levels in the plant until at least 11 d.p.i. Notably, no infections were observed until 11 d.p.i., with positive detections emerging only two days later (Sup. Figure 1). While the dot-ELISA method is commonly employed due to its cost-effectiveness and suitability as an initial screener for a large number of plants, our results suggest that PVY detection is only reliably possible after at least 13 d.p.i., indicating a narrow window for serological detection within this timeframe considering the sweet pepper cultivar Ikeda. Consequently, screening plants for PVY during the early stages of infection may yield false negative results, as the virus may be present in the field but remain undetectable at these early stages.

In serological tests, a single leaf, preferably the youngest, is typically collected for detection. We conducted experiments to determine which of the younger leaves is most suitable for the detection test. For PVYCa, the virus was detected in the second youngest leaf in 4 out of 5 inoculated plants, and in the third leaf in 2 out of the same 5 plants. For PVYSt, 4 positives out of 5 were detected in the second leaf, while 1 out of 5 were detected using the third leaf (Sup. Figure 1). The detection test was performed at 13 d.p.i. In conclusion, our findings suggest that for the detection of PVY in sweet pepper plants using dot-ELISA, testing should be conducted at least 13 d.p.i., preferably using the second youngest leaf. Note that our experiments were exclusively conducted with Ikeda peppers, as detection in tomato and

potato cultivars posed no challenges during previous laboratory tests (*data not shown*). Therefore, all PVY detections in our experiments were performed with at least 13 d.p.i. and using the second youngest leaf.

To investigate whether the host from which the PVY isolate originated influences resistance responses, PVYCa, PVYSt and PVYSl were used for inoculation of 27–30 plants of sweet pepper cv. Ikeda, potato cv. Atlantic and tomato cv. Santa Clara.

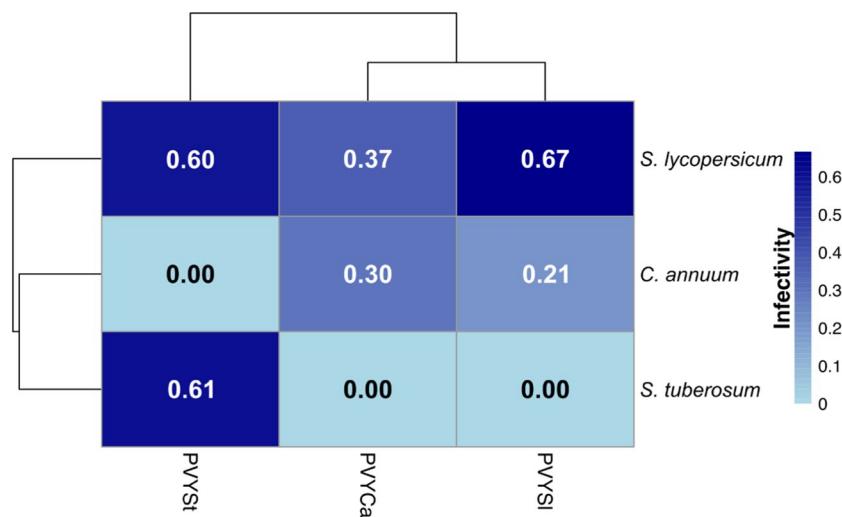
Sweet pepper plants were infected with PVYCa (8 positives out of 27, Infection Rate (IR) of 30%) and PVYSl (6/29, IR=21%), but not with PVYSt. Tomato plants were infected by all isolates: PVYCa (11/30, IR=37%), PVYSt (18/30, IR=60%) and PVYSl (20/30, IR=67%). Potato plants were infected by PVYSt (17/28, IR=61%), but neither PVYCa nor PVYSl infected them. This suggested a strong specificity of the isolates to the hosts (Gebre Selassie et al. 1985; Fereres et al. 1993; Romero et al. 2001; Moury 2010). None of the combinations yielded a 100% IR. Interestingly, PVYCa was unable to establish infection in potato plants, while PVYSt failed to infect sweet pepper plants, indicating a clear distinct interaction between these two viruses and hosts. Actually, the responses of pepper and tomato plants against the inoculation of PVYCa and PVYSl were similar, and clearly differed from the ones of PVYSt.

A Generalized Linear Model (GLM) with a binomial distribution was fitted to assess the interaction effects of species, virus isolate, and host on the infection proportion. The significance of the model coefficients was evaluated to determine the effect of each factor and their interactions on the infectivity. The model showed that the original hosts generally had higher infection proportions compared to non-original hosts, with some exceptions, such as tomato infected with PVYSt (Fig. 1). It was also possible to detect three different patterns in non-original hosts, in which pepper plants could be infected by PVYCa and PVYSl, tomatoes by all isolates and potatoes only by PVYSt. This is consistent with the expectation that viruses are better adapted to their original hosts.

Further studies could explore the mechanisms behind the observed infection patterns, such as differences in plant immune responses or viral replication efficiency in original versus non-original hosts. Understanding these underlying factors could improve the prediction of viral spread and the development of resistant plant cultivars. In conclusion, our findings confirm the importance of considering isolate specificity in screening and management strategies for disease control (reviewed in Karasev and Gray 2013).

Our systematic evaluation of diverse host-pathogen interactions aimed to uncover potential cross-species transmission patterns of PVY and their implications for disease spread and management strategies. We observed that hosts (genotype, physiology and phenology) may influence and

**Fig. 1** Infectivity proportions of the three plant species for each PVY isolate. Darker blue shades represent a higher number of infected plants, while lighter shades represent fewer infected plants



shape the PVY population, with certain isolates showing limited impact on specific hosts upon initial infection. This phenomenon suggests the presence of antagonistic pleiotropy, wherein mutations beneficial in one host may be detrimental in another (Whitlock 1996). Furthermore, phylogenetic analysis revealed a strong correlation between PVY phylogeny and host species origin, with pepper isolates clustering together and no specificity observed for PVY isolates in tomatoes (Quenouille et al. 2013). Based on the evidence that the choice of the isolate is crucial for screening purposes, we selected PVYCa to test sweet pepper cultivars and PVYSI to test tomato cultivars. We did not screen potato cultivars for resistance to PVY as all commercial cultivars are known to be susceptible (Karasev and Gray 2013).

Seeds of commercial cultivars of sweet pepper ( $n=5$ ) and tomato ( $n=18$ ) were searched in the market and subjected to inoculation trials, conducted twice, in Autumn and Summer, to ensure consistent results. Inoculations were performed and symptoms recorded, both in a greenhouse environment. Based on paired t-test ( $-1.2371$ ,  $p$ -value 0.2297), Wilcoxon signed-rank test (29.5,  $p$ -value 0.2781), and Cohen's d ( $-0.264$ ), there was no statistically significant difference rates between 1st and 2nd repetitions. The results from both trials were similar, prompting the calculation of the IR based on combined data.

Sweet pepper cultivars were inoculated with PVYCa, resulting in infection across all five cultivars. The IR ranged from 45 to 82%, averaging 74% (Fig. 2, green bars). Notably, severe symptoms such as blistering and interveinal chlorosis, along with leaf abscission and severe damage, were observed, particularly in cv. Ikeda (Sup. Figure 2). Despite displaying strong symptoms, cv. Ikeda exhibited the lowest infection rate among all cultivars (45% IR).

The absence of resistant sweet pepper cultivars contradicts the description of these cultivars as resistant to PVY infection, according to the seed company. This discrepancy

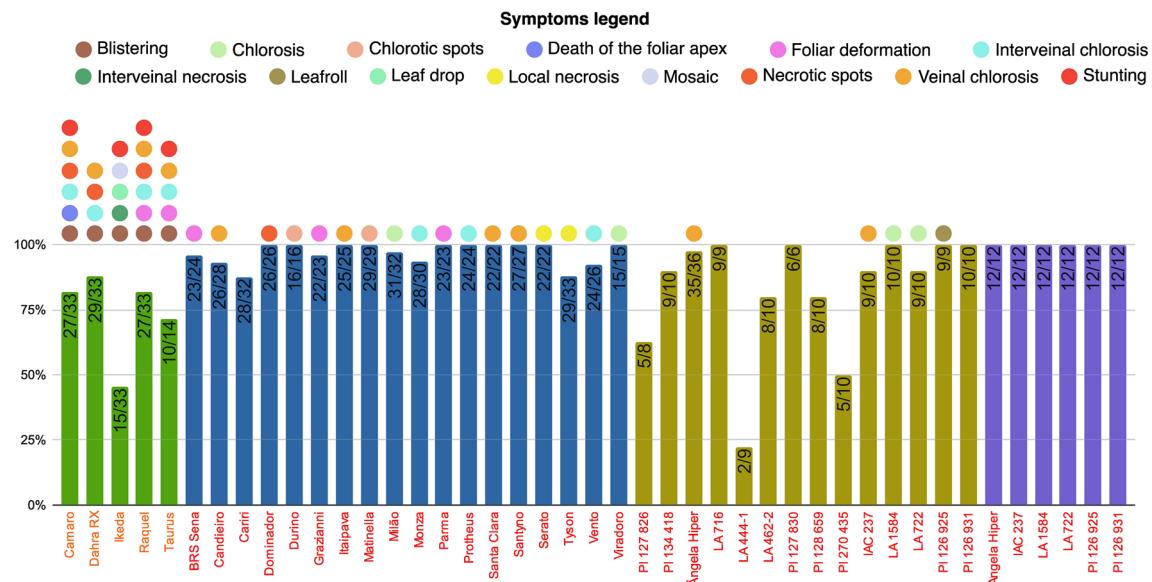
underscores the importance of using multiple isolates during cultivar screening, considering potential infection barriers. Indeed, previous studies have demonstrated such barriers, such as the findings that isolates from potatoes poorly infect pepper plants, consistent with our results (Blanco-Urgoiti et al. 1998; Romero et al. 2001; Moury 2010).

Tomato cultivars (18 in total) were mechanically inoculated with PVYSI in the greenhouse, with all cultivars displaying susceptibility to the virus. The infection rates were even higher compared to sweet peppers, with ten cultivars exhibiting 100% IR, and the lowest rate at 88%, averaging 96% for all cultivars (Fig. 2, blue bars).

Despite the high infection rates, tomato cultivars exhibited mild symptoms (Sup. Figure 3). This raised concerns about the detection of PVY in tomato fields, as visual inspections may miss strains inducing mild or no symptoms, potentially serving as undetected inoculum sources.

There are no studies that elucidate these questions in commercial cultivars, primarily because PVY is well studied in potatoes but not in other crops. In these cases, ELISA detection methodology can be used, ruling out false negatives based on symptomatology. Although the observation of mild symptoms in tomato plants has already been reported (Costa et al. 1960) and is in agreement with the results found here, the appearance of strong symptoms of necrosis caused by PVY, present in the middle third of the plant in tomato production fields, cannot be ruled out (Lucena et al. 2025). This means that the symptoms development may be related to the viral isolate, the cultivar, environment aspects, simultaneous mixed infections (for example the combination of PVY and potato virus X (Vance 1991) or PVY and potato spindle tuber viroid (PSTVd) (Qiu et al. 2014) or a combination of them or unknown factors).

Although there is no information regarding the resistance to PVY infection in any of these 18 tomato cultivars, they were chosen due to the agronomic characteristics they



**Fig. 2** Comparative infection rates of tomato and pepper cultivars, and wild *Solanum* spp. accessions. Sweet pepper cultivars are represented by green bars, tomato cultivars by blue bars, wild *Solanum* spp. accessions by golden bars, and their second-generation plants by purple bars. The number inside each bar indicates the number of plants positive for PVY infection followed by a slash and the total number of tested plants. Sweet pepper and tomato cultivars were

possess, but more importantly to the resistance to other pathogens. Altogether, they are resistant to bacteria, fungi, nematodes or even virus infection. This includes the resistance of BRS Sena to begomoviruses, Itaipava and Viradoro to tospoviruses, Grazianni and Vento to tobamoviruses, Serato to tospoviruses and tobamoviruses and Cariri, Candieiro, Durino, Milão, Monza, Parma, Protheus, Santyno, and Tyson to begomoviruses, tobamoviruses and tospoviruses.

However, our tests revealed that none of the commercial sweet pepper or tomato cultivars exhibited resistance to the tested PVY isolates, highlighting the necessity of seeking new materials through breeding programs. This emphasizes the urgency of addressing PVY susceptibility in commercial cultivars to mitigate potential production losses and ensure crop health. Note that these cultivars, when infected, may serve as a reservoir of the PVY isolates.

As no commercial cultivar was resistant to PVY infection, wild lines of *Solanum* spp. accessions were screened in an attempt to obtain potential resistance sources. Fourteen wild tomato materials from the Instituto Agronômico de Campinas Germplasm Collection of *Solanum* species were screened: *Solanum pimpinellifolium* (PI 126 931, LA 722, LA 1584 and PI 126 925), *S. habrochaites* (PI 134 418 and PI 127 826), *S. lycopersicum* (Angela Hiper), *S. pennellii* (LA 716) and *S. peruvianum* (LA 462-2, PI 127 830, PI 270 435, IAC 237, LA 444-1 and PI 128 659). The wild tomato species were tested once, due to limited

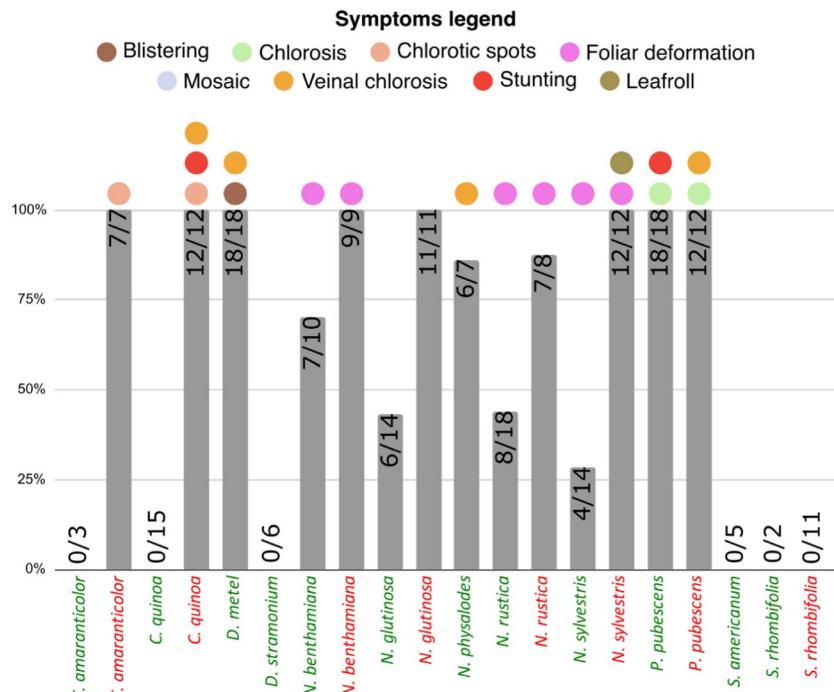
evaluated in two different seasons in the greenhouse, with the number of plants representing the sum of positive and tested plants. The isolate used for inoculation is indicated by distinct colors on the x-axis: PVYCa in orange and PVYSI in red. Colored circles above the graph denote the presence of symptoms, with an absence of a circle indicating no symptoms

seed availability, with Angela Hiper being the exception and tested three times.

All accessions were susceptible to PVY infection with IR between 22 to 100%: *S. pimpinellifolium* ( $n=4$ ) presented 97% of IR, *S. lycopersicum* ( $n=1$ ) presented 97%, *S. habrochaites* ( $n=2$ ) presented 77%, *S. pennellii* ( $n=1$ ) presented 100% and *S. peruvianum* ( $n=6$ ) presented 69% of IR (Fig. 3, golden bars). The only material that showed low IR was LA444-1 (*S. peruvianum*) with 22%. Although some accessions exhibited chlorosis, veinal chlorosis and leafroll, most of them exhibited no symptoms at all, suggesting tolerance of these accessions (Sup. Figure 4). Despite their susceptibility, these accessions may still be important in the search for resistance against PVY due to the lower IR compared to other tomato cultivars (Tukey's HSD = 0.1, p-value = 0.041).

Previous studies had identified the wild tomato LA444-1 as resistant against PVY based on the absence of symptoms using visual evaluation (Lourenço et al. 2005). Our findings demonstrate that LA444-1 may serve as a potential source of resistance to PVY due to its lower IR, though it remains susceptible to PVY. This underscores the challenge of selecting the isolates for resistance tests, and also of relying solely on visual cues to determine resistance, especially when infected plants exhibit only mild or no symptoms, as described previously in others wild tomato accessions (Palazzo et al. 2008).

**Fig. 3** Experiments for determination of infection rates in indicator plants using PVYSt and PVYSl isolates from potato and tomato, respectively. Each PVY isolate is depicted by a distinct color on the x-axis: PVYSt in green and PVYSl in red. The number inside each bar indicates the number of plants positive for PVY infection followed by a slash and the total number of tested plants. Colored circles positioned above the graph denote the presence of symptoms, with an absence of a circle indicating no symptoms



Interestingly, the cultivar Ângela Hiper, historically valued for its resistance to PVY, displayed an unexpectedly high IR of 98%. Since the 1960s, significant efforts had been made to introgress PVY resistance into the tomato cultivar Santa Cruz, which was highly susceptible to this important disease. In the 1970s, through backcrossing between Santa Cruz and PI 126410 (*S. peruvianum*), a new cultivar called Ângela (Nagai and Costa 1969) was released. It was quickly adopted by tomato growers due to its resistance to PVY, *Fusarium oxysporum* f. sp. *lycopersici* race 1, and *Stemphylium solani*, as well as its high yield. Between 1975 and 1988, it was used on 75–80% of the total stalked (fresh market) tomato acreage. This initial success spurred the development of new cultivars, such as Ângela Hiper (Nagai et al. 1992), derived from the original. However, despite its past success, our extensive testing consistently revealed high levels of susceptibility (averaging 98%).

This result aligns with previous studies on screening wild tomato species for resistance, in which 19 *Solanum* spp. accessions were found to be susceptible to PVY, sometimes showing symptoms and other times remaining asymptomatic (Palazzo et al. 2008). However, the results obtained here indicate a higher level of susceptibility among the accessions, with a greater number of positive plants, suggesting that this virus isolate PVYSl has a potential to infect other tomato cultivars believed to be resistant to PVY infection.

To validate our findings and rule out the possibility of genetic segregation, we conducted an additional experiment with wild tomato accessions. We generated seeds from six autopolinated non-infected wild tomato plants, including

one *S. lycopersicum*, one *S. peruvianum*, and four *S. pimpinellifolium* accessions. These seeds were then sown and subjected to PVY inoculation. All six cultivars exhibited a minimum IR of 90%, mirroring the parental generation's susceptibility. After mechanical inoculation with PVYSl, all first-generation plants displayed 100% IR, consistent with the parental generation, indicating no genetic segregation (Fig. 2, purple bars). As observed in the previous trial, no symptoms were observed in any of these plants. Thus, the susceptibility was confirmed for all commercial and wild tomato accessions to PVY infection. These findings collectively suggest that, although some may present escapes of infection, there are currently no known sources of resistance to the isolate PVYSl in tomatoes.

Based on the previously inoculation trials, PVYCa and PVYSl have similar infectivity properties, so PVYSt and PVYSl were tested in inoculation trials of various plant species. Our tests encompassed plant species from the Solanaceae family (*Datura metel*, *D. stramonium*, *Nicotiana benthamiana*, *N. glutinosa*, *N. rustica*, *N. sylvestris*, *Nicandra physalodes*, *Physalis pubescens* and *Solanum americanum*), Amaranthaceae (*Chenopodium amaranticolor* and *C. quinoa*) and Malvaceae (*Sida rhombifolia*). Due to low seed availability, not all hosts were tested with both isolates.

Our data show evidence that PVY infected hosts within the Solanaceae and Amaranthaceae families (Fig. 3), consistent with previous reports cataloging these plants as hosts of PVY (Edwardson and Christie 1997). However, *S. rhombifolia* (Malvaceae family) plants were not infected with PVY, corroborating existing reports that malvaceous

plants are not hosts of PVY (Coutts and Jones 2014). PVYSl and PVYSt differed in the rate of IR in the tested hosts, in which PVYSl demonstrated to be more adapted to different hosts, compared to PVYSt (Fig. 3).

While both PVY isolates successfully infected most tested plants, the two exceptions were *C. amaranticolor* and *C. quinoa* plants. These two indicator plants are commonly used as test plants due to the production of easily countable local lesions after mechanical inoculation (Hollings 1956). They displayed unique symptoms upon inoculation with PVYSl. Initially, chlorotic spots with a red halo appeared on older leaves, which gradually evolved into systemic symptoms spreading throughout the plant (Sup. Figure 6). This result contradicts previous knowledge of the local infection caused by PVY (Palazzo et al. 2008), demonstrating a concern with the use of model plants and their applications. Importantly, this result was only observed when using PVYSl, while PVYSt was not able to infect this host, once again proving the importance of isolate choice. According to our results, it is crucial to exercise caution when performing detection tests, preferably conducting pilot tests to minimize the risk of false negative results and ensure accuracy.

While extensive research was conducted to elucidate the interactions between potato and PVY, such as transgenic approaches overexpressing PVY-derived coat protein, PVY-specific dsRNA (for RNAi), modified plant eIF4E, clustered regularly interspaced short palindromic repeats (CRISPR/Cas) and spray-induced gene silencing (SIGS) (Romano et al. 2001; Zimnoch-Guzowska et al. 2013; Valkonen et al. 2017; Torrance and Taliánksy 2020), other crops such as tomatoes and peppers have received comparatively less attention. This highlights the need for increased research focus on tomato and pepper to develop effective PVY management strategies.

The absence of resistant materials from commercial or breeding programs underscores the urgency of addressing the spread of PVY in tomato and pepper production fields, as it allows the virus to persist. Furthermore, our findings highlight the variability in host range adaptation among different isolates of the same species, emphasizing the need for thorough testing using diverse isolates.

Organisms continually evolve and adapt to new environments, resulting in the emergence of new characteristics, including changes in their ability to infect hosts. Therefore, a more dynamic approach to understanding the interaction between the virus and its host is essential.

A comprehensive understanding of PVY and its adaptation across various host systems is vital for developing effective control strategies against this pathogen. Integration of advanced molecular techniques with a deep understanding of viral dynamics across diverse hosts is key to mitigating the impact of PVY and safeguarding global agricultural systems from its detrimental effects.

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**Authors' contributions** Conceptualization: AKIN and IJM; Methodology: all authors; Data and sample collection: BMC, DYMS and IJM; Formal analysis: AKIN and IJM; Writing—original draft preparation: all authors; Writing—review and editing: AKIN and IJM; Funding acquisition and supervision: AKIN.

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**Data availability** The data that support the findings of this study are included within the manuscript. No additional data repositories were utilized or required.

## Declarations

**Conflicts of interest** We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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