

# Potato Progress

Research & Extension for the Potato Industry of Idaho, Oregon, & Washington

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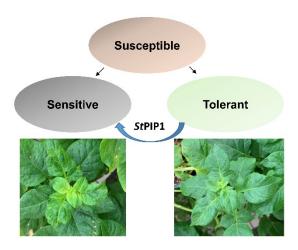
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## From tolerant to sensitive: how a small peptide produced by potato plants determines the fate of potato-PVY interaction

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Different potato cultivars respond differently to potato virus Y (PVY) and to different strains of PVY. We can categorize cultivars into two main groups, those that are susceptible, and those that are resistant to PVY (**Figure 1**). Susceptibility means that the virus is able to infect the plant systemically. Systemic infection by the virus can have two outcomes: sensitivity or tolerance. Cultivars that are sensitive to PVY develop clearly visible symptoms such as mosaic and molting in leaves, and in some cultivars that are infected with necrotic strains, ringspots on tubers, a disease known as the potato tuber necrotic ringspot disease. The main issue with sensitive cultivars infected with PVY is that they produce lower yields, and the yield penalty can be very severe (up to 70% yield decrease) [1-3]. Contrary to sensitive cultivars, cultivars that are tolerant to PVY produce no or mild symptoms, but still can incur a yield penalty. However, another issue with tolerant cultivars is that infection with PVY is difficult to identify without molecular tools, and PVY-infected tolerant plants can be a significant, undetected source of PVY.

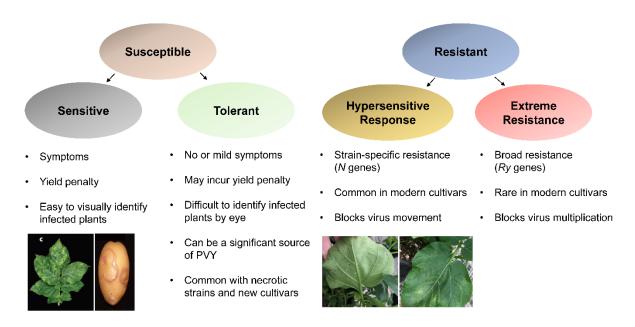


Figure 1. Potato cultivars differ in their response to potato virus Y (PVY).

In my lab, we are interested in better understanding the interactions between potato and PVY, whether it be in the susceptibility or resistance context, so that we can use that knowledge to develop PVY management strategies. To do so, we have been studying the cultivar Premier Russet because it gives us the possibility to look at resistance (i.e. Premier Russet shows lower infection rate to PVY strains O, N:O, and N-Wilga, likely via hypersensitive response-type resistance) as well as susceptibility (i.e. Premier Russet is susceptible/tolerant to PVY strains N and NTN) (**Table 1**). By analyzing changes in the transcriptome of potato in response to PVY inoculation [4], we were able to identify genes whose expression was strongly affected by PVY. Several of those most affected genes had their expression repressed by PVY. These results prompted us to investigate the exact role that these genes may play in the response to PVY. Amongst these genes, we focused our efforts on a gene that codes for a small peptide that we called *St*PIP1.

**Table 1. Infection rate in Premier Russet plants inoculated with different PVY strains.** Plants were mechanically inoculated with PVY in a greenhouse. PVY infection was determined by RT-PCR on non-inoculated leaves. Numbers represent number of infected plants over total number of plants.

Control	O	N:O	N-Wilga	N	NTN
0/12	3/12	2/12	5/12	9/12	10/10

We used several experimental approaches to understand the function of *St*PIP1. Amongst those, we generated Premier Russet plants that produce *St*PIP1 constitutively (i.e. overexpress), which means that they produce the peptide constantly, and inoculated them with different strains of PVY, i.e. O and N-Wilga to study the role of *St*PIP1 in the context of resistance, and NTN to study the role of *St*PIP1 in the context of susceptibility. Then, we monitored systemic infection

and symptoms development. While we did not observe any major changes in the response of the plants to PVY-O or PVY-N-Wilga, we found that Premier Russet plants that overexpress *St*PIP1 developed clearly visible symptoms (i.e. rugose mosaic) starting 30 days post inoculation with PVY-NTN, while normal Premier Russet plants showed no or very mild symptoms when infected with PVY-NTN (**Figure 2**). In other words, **by overexpressing** *St***PIP1**, **we broke the tolerance of Premier Russet and converted it to a cultivar that is sensitive to PVY-NTN.** 



**Figure 2. Symptoms of** *St***PIP1-overexpressing lines compared to Premier Russet (PR) upon inoculation with PVY-NTN.** Symptoms including rugose mosaic, stunting, and chlorosis were clearly visible in *St***PIP1** overexpressors infected with PVY-NTN. A) Mock-and PVY-NTN-inoculated PR and *St***PIP1-overexpressing line** #1 (PIP-OE1) 44 days after inoculation. B) Close up images of canopy leaves of mock-and PVY-NTN-inoculated PR and PIP-OE1 47 days after inoculation. C) Three independent *St***PIP1-overexpressing lines** (PIP-OE14, PIP-OE8, and PIP-OE1) infected with PVY-NTN showing clearly visible symptoms compared to a relatively asymptomatic PR control 69 days post inoculation.

We were also able to show that StPIP1-overexpressing Premier Russet plants that were symptomatic when infected with PVY-NTN had a large number of well-known defense genes that were switched on, while normal Premier Russet plants had very few genes that were switched on in response to PVY-NTN infection (for details, please see [5]). From these results, we hypothesize that the symptoms observed are due to an excess of energy devoted to plant defense responses to the detriment of the overall plant fitness.

Tolerant cultivars represent a challenge for identifying PVY-infected potato plants without the use of molecular tools, which may lead to an underestimation of PVY infection in seed lots. Given that both sensitive and tolerant cultivars may incur yield penalty, planting sensitive cultivars may at least offer the advantage of easier visual identification of infected plants. Here, we have identified *St*PIP1 as one genetic determinant of tolerance versus sensitivity to PVY. Selecting and planting cultivars that constitutively express *St*PIP1 could make roguing of PVY-infected plants easier. Alternatively, we hypothesize that external foliar application of synthetic *St*PIP1 will trigger the development of clearly visible symptoms, similar to those observed in plants that express *St*PIP1 constitutively, that would not otherwise be apparent (**Figure 3**). Such an externally applied treatment might be useful in seed production, making infected plants clearly visible and easily rogued. We are currently investigating this possibility.



Figure 3. Hypothesis: External application of *St*PIP1 (e.g. foliar spray, root solution) will trigger the development of symptoms in PVY-infected plants that would not otherwise show symptoms.

#### For questions, please email <u>Aymeric.goyer@oregonstate.edu</u>

This research was published in the Journal of Experimental Botany [5] <a href="https://academic.oup.com/jxb/advance-article-abstract/doi/10.1093/jxb/erab078/6157931">https://academic.oup.com/jxb/advance-article-abstract/doi/10.1093/jxb/erab078/6157931</a> and was funded by grants from the Northwest Potato Research Consortium, the U.S. Department of Agriculture National Institute of Food and Agriculture, and the Western SARE.

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