VI1601: What causes symptomless Psa to become symptomatic?

COVER NOTE

Kiwifruit plants can often be 'infected' with *Pseudomonas syringae* pv. *actinidiae* (Psa) but without symptoms. The time between infection and symptom development is known as the 'latency' phase. During that phase, infected plants are symptomless carriers.

It is extremely difficult to identify plants that are in a latency phase and to know how long the phenomenon can last. Understanding why an infected plant does not express symptoms and what are the triggers to break the latency phase (i.e., the triggers that will lead to the plant expressing symptoms), are important pieces of information that are needed to put in place strategies to prevent or manage outbreaks.

In this work, we asked how does Psa survive in symptomless kiwifruit plants; what changes occur in the plant defence system when Psa is introduced; and which genes or metabolites are responsible or required for Psa to stay alive in symptomless plants. We also looked for triggers that would signal the end of the latency phase (i.e., the triggers that will lead to the plant expressing symptoms). To answer these questions, experiments were carried out on potted plants on five cultivars of kiwifruit Gold3, Hort16A, Hayward, Green11, and Actinidia arguta 'Hortgem Tahi'. Additional experiments were also carried out on Gold3 plants to allow the identification of the plant, and of the bacterial genes that might be responsible for infected plants to stay symptomless.

The major findings obtained over the last 12 months were as below:

- Psa can multiply inside the plant without showing symptoms, even in the susceptible cultivars, and symptoms may not develop due the number of bacteria used to infect the plants.
- Keeping the plants of Hort16A and Gold3 or Hayward and Green11 under flood-like conditions did not always result in the suppression of symptom expression especially when inoculation was with low concentrations of Psa.
- The concentration of inoculum introduced and potentially the rate of growth of the plant were influential in determining whether an inoculated plant would become a symptomless carrier.
- Analysis of genomic data indicates that in the first 48 hours post-inoculation, bacterial gene expression is similar in plants expressing symptoms and in symptomless plants. In contrast, a large number of kiwifruit genes express differently in plants inoculated with a low concentration of Psa (plants that would become symptomless carriers) versus plants inoculated with a high concentration of Psa (plants that would express symptoms). In particular, a number of auxin responsive proteins were highly overexpressed within 3 hours of inoculation.

The conclusions from the overall project are that:

- In all the cultivars tested, Hayward, Gold3, Hort16A, Green11 and in *A. arguta,* leaf infection and stem inoculation can lead to Psa multiplying endophytically without the plant expressing symptoms.
- Psa was able to survive and multiply in symptomless potted plants of *Actinidia arguta*, a species known to be resistant to Psa. This result suggests that plants of resistant cultivars or species may be symptomless carriers and pose a risk to spread the disease.

- The bacterial load (number of bacteria per unit of plant tissues) in symptomless tissues can be as high as that in tissues showing symptoms.

- The bacteria were able to travel at least short distances in symptomless plants.

- The physiological state of the bacteria influences its ability to infect, but once the bacteria is in the plant it seems that it is the plant that determines whether the interaction will lead to symptom development.

- The physiological state of the plant and its rate of growth seem to be factors controlling symptom expression.

- Results obtained 2 years in a row suggest that following leaf infection some cultivars, e.g., Hayward' are able to kill the pathogen while others cannot. This ability to kill the pathogen could be an important characteristic of plants that are not systemically colonised.

While metabolites and genes that were differentially expressed in symptomless plants have been identified in this work further work is needed to fully understand the mechanisms supporting pathogen multiplication in a symptomless plant. Some of the environmental triggers that could break latency have been identified, e.g., inoculum concentration and water stress; however, other factors that interfere with plant growth might also be involved.

Practically, the results of this project mean that late summer and autumn infections are most likely to allow Psa in the tissues without any visible symptoms. The usefulness of late season application of protectants might have been underestimated.