Lessons learned from Psa-V incursions to new regions in 2012: Waikato, Hawkes Bay, Gisborne, Coromandel and Kerikeri

March 2012

It has been nearly two and a half years since the first case of Psa-V was detected in Te Puke in 2010. Over the last year, a number of new incursions have occurred in previously 'clean' regions. Observations and case studies have provided some information on the spread of the bacteria within these regions. We now understand more about the patterns of infection and expression of the disease.

Infection risk factors

The following three factors stood out as being important in the prevention of infection:

1) Orchard hygiene protocols; few orchards were found to be undertaking best practice orchard hygiene protocols prior to infection occurring within a region.

2) Comprehensive spray programmes; growers in non-infected regions generally did not have proactive protectant programmes in place before Psa-V arrived in a region. Some time periods had little or no spray coverage, particularly over summer.

3) Managing vines; where infection was promptly removed, spread of Psa-V within a region was more easily managed. Environmental factors such as frost and wind appeared to predispose vines to infection by Psa-V.

The concentration of bacteria needed to enable infection to occur in a kiwifruit vine is still unknown. We do know that once an orchard is confirmed positive with Psa-V it is highly unlikely the disease can be eradicated. Therefore prevention of entry is the only viable option.

Four 'Exclusion' regions remain in New Zealand kiwifruit growing areas. Whangarei, North Auckland, Manawatu/Whanganui and Nelson. Their ability to remain free of Psa-V will be very important in future seasons. As more 'Containment' and 'Recovery' regions switch to new varieties to replace H16A, lessons learned from previous incursions become useful in making management decisions. For example for new varieties to succeed through the juvenile phase, when they are more susceptible, levels of inoculum and risk of infection should be as low as possible to enable graft success.

Table 1 Dates Psa-V was identified for each region outside of the Bay of Plenty in 2012

Region	First +ve test result	Total # of +ve orchards	Total # of KPINs
		At 20th March 2013	
Franklin	18 November 2011	38	103
Waikato	20 August 2012	20	86
Coromandel	6 August 2012	16	45
Hawke Bay	10 October 2012	2	52
Kerikeri	23 October 2012	1	105
Gisborne	20 November 2012	5	73

It is likely that in the majority of new incursions, infection would have been present for some time before being identified. The three most likely pathways for long distance movement of Psa-V are believed to be via plant material such as budwood or rootstock, orchard equipment and heavy machinery and through contractors and orchard workers transferring infection on tools boots and clothes etc. While wind and weather played an important part in dispersal of the disease in Te Puke where high densities of the very susceptible H16A were in close proximity it is unlikely that weather related dispersal events have accounted for all the incursions seen in new regions in Spring 2012 (Table 1).

Another possible source of infection is thought to be via bees and pollen, although this pathway carries a much reduced risk compared with the three mentioned above. Research conducted in 2011 found that within 6-9 days there was a substantial reduction in Psa-V within artificially inoculated hives (Pattemore et al. 2011). A rapid decline of bacterial populations occurs in the first 24hrs, meaning few bacteria may remain for long periods of time. However Psa-V could still be detected up to 2 weeks later in plastic discs in inoculated hives. It is prudent to consider hives as potential sources of the bacteria for at least 14 days because it is unknown how many bacteria are required to start an infection. More work is currently underway to see how long naturally infected hives and bees retain the bacteria. Pollen is considered a more likely source of Psa-V infection because it is directly applied to kiwifruit flowers. Some regions and growers interviewed had not undertaken any artificial pollination at all (e.g. Waikato), and this pathway for transmission is less relevant in these examples.

Psa-V dispersal

The exact distance Psa-V can travel via weather dispersal has not been quantified. Based on international advice and domestic observations of spread in Te Puke a 10km radius is used to set controlled area limits by KVH during new incursions in regions previously free from Psa-V. The assumption is made that wind dispersal is more likely to occur over a maximum of 10km, but be much less likely to occur across distances up to 15km. Monitoring of those orchards closest to the initial infection site, can help suggest the possible trajectory of wind-blown inoculum to other nearby orchards.

Even if the exact dispersal range of Psa-V via weather and airborne inoculum was understood, there still remains the unknown of how much inoculum is required to successfully produce infection in a plant. As the bacteria travel further via wind its dispersal is dissipated and the likelihood of reaching a host target becomes smaller. Because of this gap in knowledge, from a practical orchard management perspective, the more important factor in reducing disease spread will be the early detection of plants with symptoms.

Psa-V test results

Psa-V testing results from August 2011 to July 2012 were analysed for four localities within the Bay of Plenty. Katikati/Waihi, which have been combined as one area, Tauranga, Opotiki and Whakatane. The analysis looked at the number of samples where retests within 2 months, 3 months and 6 months confirmed a positive result after an initial not detected result. Over the course of the Waikato incursion and within the previous year and a half in the Waikato several not detected results were also recorded. Subsequent positives for those samples were found within six months. These results are also in Table 1.

Table 2. Testing for Psa-V—assessment of testing and retesting within regions

August 2011-July 2012	**Not Detected**	**Re-test within 2 months (+ve)**	**Re-test within 3 months (+ve)**	**Re-test within 6 months (+ve)**
Katikati /Waihi	10	4	0	6
Tauranga	28	11	3	14
Opotiki	18	12	2	4
Whakatane	21	14	2	5
August 2011-Nov 2012				
Waikato	3	2	0	1

Several issues have been identified from the review and are relevant for pathway assessments undertaken. Two orchards in the Waikato incursion saw what they thought were Psa-V symptoms prior to a positive test result. One orchard took a sample in spring 2011 and got a not-detected result. The next test was not taken until spring 2012. The other orchard saw suspicious symptoms 6-8 months prior to the first positive test result, but did not get a test at the time of first symptom appearance. If more testing had been undertaken earlier, Psa-V might have been identified in the region much earlier.

Incursion patterns

There have been two patterns of incursion in the North Island over the previous two years. One pattern was seen in Gisborne, Hawkes Bay and Kerikeri. A single orchard or small cluster of orchards was found positive with Psa-V, and subsequent wider spread of the disease has not been observed in the region. In these cases it is likely the initial infection point has been identified in the early stages, and with good use of protective sprays and speedy removal of infected material the bacteria is contained within the initial focal site/sites.

The other pattern is that seen in Coromandel, Waikato and during the Franklin incursion in 2011. An initial infected orchard was found, and then subsequent monitoring and testing revealed a much wider spread of the disease than was initially thought. It is likely in these cases that the disease may have been present six months- one year before detection, and not been picked up in previous monitoring rounds.

Plant material

Assessments done for Waikato, Coromandel, Gisborne and Franklin incursions revealed linkages between infected orchards via budwood or rootstock, however because not all affected orchards in each region had inward budwood movements, or symptoms in recently grafted budwood material (last two years), it is not possible to definitively pinpoint budwood as a source of initial infection.

There has been little research looking at the symptom expression of infected budwood after it has been grafted. Infection in budwood is most commonly vascular in nature and likely asymptomatic at the time of grafting. Observations in orchards with suspected asymptomatic budwood show it can lead to cane dieback, cankers, stunting of new growth and overall less vigorous growth of new shoots and canes.

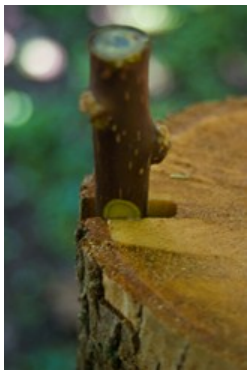

Figure 1: New graft

Figure 2: Pruning tools

Figure 3: Heavy orchard machinery

Some of these symptoms, particularly stunting of new growth, and less vigorous growth of shoots and canes, can also be associated with poor graft take. It can be difficult to distinguish between effects in the early stages after grafting. An inherent difficulty also exists in distinguishing between budwood infection, and infection occurring from inadequate hygiene during grafting, via tools. Insufficiently protected new grafts, would also be susceptible to infection, particularly when weather events wash off waxes and pruning paints. Again making it difficult to distinguish between latent infection in plant material and infection via large grafting wounds and exposure to aerosol Psa-V.

In Coromandel some growers believe that infected G9 budwood grafted in 2010 may have been a contributing factor in infection reaching the region. In Gisborne Psa-V potentially entered the region through budwood. Vascular infection was identified as one of the more plausible pathways of entry. Infection then clearly spread from the initial focal infection site to several other orchards via movement of rootstock. Male budwood shared privately between orchards in different regions, poses a significant risk of disease transmission. Especially if adequate testing and regular monitoring for symptoms does not accompany these arrangements.

Hygiene and understanding of Psa-V-V infection timing

Good hygiene protocols are important in maintaining Psa-V free regions in New Zealand. In the Waikato implementation of standard and consistent hygiene protocols among orchard workers and contractors ranged from December 2010, to October 2011. There was no vine to vine hygiene practiced by any contractor during winter or summer pruning, thinning or harvest in 2011 or 2012. Tools were either 1) put in a bucket of steriliser every two hours, 2) at every break, or 3) every morning before work began. In the case of mulchers, tractors and spray equipment shared between orchards, equipment was hosed down and water blasted but not sanitised in some cases until as late as spring 2012.

A lack of understanding of the exact time period when Psa-V first entered the country, combined with a belief that orchards were safer in regions further away from focal infection points have probably contributed to a lack of more rigorous hygiene protocols. This situation was common in most regions where Psa-V has been found except Kerikeri. Kerikeri region had a Psa-V scare in October 2011. Hygiene protocols were strictly maintained after this time, and when Psa-V was found and confirmed in October 2012, removal of infected plants was swift and monitoring appeared to confirm the bacteria had not moved from the initial focal point.

Spray programmes

An analysis of the 2011-2012 industry spray diary indicated considerable room for improvement in what and how the industry is using agrichemicals to control Psa-V (Max and Max Kiwifruit Journal Jan/Feb 2013). It also indicated non-infected regions were not proactive at having protectant sprays in place before the disease arrived (Table 3). Organic growers were typically less prepared than their conventional counterparts. There was also little evidence to suggest growers were applying sprays before wet weather events.

The results of the industry wide analysis are mirrored in an analysis of spray diaries for individual regions hit by new incursions in Spring 2012. Assessments revealed that spray coverage during the 2011-2012 period would have been insufficient to protect orchards from either wind dispersed infection or repeat localised infection once orchards had become infected.

Although fine hot weather reduces the incidence of Psa-V expression and spread via weather events, research has shown that temperatures in New Zealand over summer are not warm enough to inhibit reproduction of the bacteria. Recent case studies have backed this up with observations made by growers in Hort16A and Gold3. Table 3 below shows that in Hawke Bay, Gisborne, Nelson, Northland, Southern North Island and Waikato, spray coverage is unlikely to have been sufficient from late spring to late summer in 2012 (Fruit set to end of Monitoring, coloured in yellow).

Table 3. Hort16A hectares sprayed for Psa-V with at least one spray in 2012 season by seasonal interval (includes non-KVH recommended sprays).

Spray Diary Periods	Auckland	Bay of Plenty	Hawke Bay	Gisborne	Nelson	Northland	Southern North Island	Waikato
Dormant	35%	59%	44%	4%	46%	23%	2%	31%
Pre-Flowering	35%	66%	38%	28%	21%	23%	2%	30%
Flowering	22%	19%	0.0%	28%	3%	1%	0%	6%
Fruit Set	9%	5%	0.0%	0.1%	0%	7%	0%	0.3%
Fruit set to Monitoring	28%	98%	0.0%	18%	0%	13%	0%	6%
Monitoring	20%	77%	0.0%	0.2%	0%	0%	1%	6%

Growers are reminded about the importance of maintaining hygiene practices, and keeping up protective spray programmes. Especially in 'Containment' and 'Exclusion' regions, growers should act now before Psa-V is identified, as there is likely to be a time lag in identification after infection has set in.
For further details from the Waikato pathway assessment please see report available on the KVH website. www.kvh.org.nz/case_studies

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