Quantification of the spatial distribution and natural rate of Psa-v spread in New Zealand

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1.0 Summary

1.1 Objectives

The objectives of the study were to describe the temporal, spatial and spatio-temporal spread of the bacteria *Pseudomonas syringae* pv. *actinidiae* (Psa-v), in kiwifruit (*Actinidia deliciosa* and *A. chinensis*) during the outbreak in New Zealand.

1.2 Methods

Data included the location coordinates and infection status of all kiwifruit orchards in New Zealand from the 5th of November 2010 until the 18th of February 2013. The temporal and spatial analyses described the number and density of infected and uninfected orchards. The spatio-temporal analyses included the Knox test, the space time K function and the simulation of the outbreak using Google Earth.

1.3 Results

As of the 18th of February 2013, 62% (2066/3309) of kiwifruit orchards had a confirmed Psa-v infection, across 13 of the 17 kiwifruit producing regions. There were two epidemic waves, both of which started in spring and totalled 65% (1354/2066) of new Psa-v infections on orchards. The Psa-v outbreak began on an orchard in Te Puke and the density of infected orchards radiated outwards from this orchard. There were significant space time interactions between case orchards at least at the critical distance of between 14 and 90 days and at critical distances that varied between 1 and 20 kilometres in eight of the thirteen Psa-v infected regions. The space time K function detected clustering in space and time and clustering varied by region. Based on a maximum local spread distance of 20 kilometres, 12 long distance spread events and subsequent local spread clusters were

identified. A further 13 spread events between 10 and 20 kilometres from the nearest infected orchard were identified.

1.4 Conclusions

There is a seasonal component to the Psa-v outbreak in New Zealand, with the majority of new infections being reported in the spring of each year. A significant spatio-temporal component was identified in some regions with infected orchards, of up to 20 kilometres and 90 days in regions where these interactions were detected. The variation in local spread characteristics between regions could be attributed to different control strategies during different phases of the outbreak, climatic conditions and other regional risk factors. Based on the local spread characteristics determined in the study, 25 spread events were considered to not be feasible due to local spread. These long distance spread events warrant further investigation to inform the National Pest Management Strategy.

2.0 Introduction

Pseudomonas syringae pv. *actinidiae* (Psa-v) is the causal agent of bacterial canker in kiwifruit (*Actinidia deliciosa* and *A. chinensis*) (Balestra et al., 2008). The pathogenicity of infection on the kiwifruit vine can range from mild, with leaf spotting and little damage to the vine and little impact on fruit yield, to severe, with symptoms including black or brown discolouration of buds and shoots, cane dieback, and cankers with exudates (Vanneste et al., 2011). The impact of Psa-v on kiwifruit is often complete vine collapse and plant death, or the removal of the vine from the orchard.

In November 2010, New Zealand experienced an outbreak of Psa-v when the bacterium was identified in vines at an orchard in Te Puke (Everett et al., 2011). By the end of December 2010, 22 orchards had been identified as having kiwifruit vines infected with Psa-v. In an effort to control the outbreak, Psa-v positive orchards were placed under restricted place notices, to restrict movements of infected plant material, and all kiwifruit growers were advised to implement equipment and personal hygiene measures to limit the spread of Psa-v to other kiwifruit orchards (Anon, 2013a). Eradication of the disease was not deemed feasible by the Ministry of Primary Industries (previously the Ministry of Agriculture and Forestry) and containment of the disease through movement control measures was ineffective. Consequently, the restricted place notices were removed in early 2011. By the end of the 2010-11 growing season, Psa-v was detected in most parts of the main Te Puke growing region. Additionally, Psa-v had spread into most North Island growing regions by the end of spring 2012.

In response to the current outbreak situation, the Kiwifruit industry set up the National Pest Management Strategy (NPMS) in 2013 (Kiwifruit Vine Health Inc., 2013). The

aim of the strategy is to prevent the further spread of Psa-v and to minimise the impact of Psa-v infection on commercial kiwifruit. The NPMS defines three types of control zones for different regions; Exclusion Regions, which have no infected orchards and are greater than 10 kilometres from the nearest infected orchard, Containment Regions, which have less than 35% of orchards infected with Psa-v and Recovery Regions, which have greater than 35% of orchards with Psa-v infection.

Psa-v has affected both of the main commercial varieties grown in New Zealand; Hayward (*A. deliciosa*) and Hort 16a (*A. chinensis*) and has had a marked economic impact (Greer and Saunders, 2012). The objective of the current study was to describe and quantify the spatial, temporal and spatio-temporal spread of Psa-v during the course of the outbreak in New Zealand (up until the end of February 2013). It is intended that the results will inform the National Pest Management Strategy (NPMS) by assisting the kiwifruit industry to better manage orchards, particularly in the Exclusion regions.

3.0 Materials and methods

3.1 Outbreak area and study population

The population of interest included all kiwifruit orchards in New Zealand. Details for all orchards, including unique identification numbers (called KPINs) and the spatial coordinates and infection status of each KPIN were provided to the researchers in a dataset by Kiwifruit Vine Health (KVH). The dataset contained testing and infection status information for 3309 KPIN's spanning 17 regions and was last updated on the 18th of February 2013. The location of all kiwifruit orchards in New Zealand is shown in Figure 1.

Early in the outbreak, cases were defined as orchards with Psa-v confirmed by diagnostic testing. As the outbreak progressed and the number of orchards infected increased, cases were defined as orchards with Psa-v confirmed by diagnostics testing or through the observation of the more severe clinical signs of disease (which are blackened canes or shoots with dieback, or the presence of stem cankers with red exudate) (Vanneste et al., 2011). Both methods of Psa-v identification were considered to be cases in the current study. The date of a positive diagnostic test or the date that the clinical signs of disease were reported was recorded in the database as the date of confirmed infection.

3.2 Description of the outbreak

Based on the dataset, new variables were created for the type of symptoms detected on infected vines, the timing of Psa-v infection and the growing season when Psa-v was first detected. A binary variable for symptoms was created based on information provided about the type of symptoms used to determine Psa-v infection. The primary symptom of Psa-v infection is leaf spotting, while secondary symptoms included the more severe symptoms of the disease (Vanneste et al., 2011). The season when an orchard was diagnosed as Psa-v infected was created with four categories; spring, summer, autumn and winter. Descriptive statistics were used to summarise the number of infected and uninfected orchards, the regions where the orchards were located, the time of the year when orchards were first infected with Psa-v and the symptoms used to determine diagnosis. These categorical variables were stratified and presented as numbers and percentages. Where appropriate, significance was assessed using the Chi square test at a level of $P<0.05$. All descriptive statistics were performed using R version 2.15.2 (R

Development Core Team, Vienna, Austria). All data were stored in a spreadsheet (Microsoft Excel 2010 for Windows).

3.3 Temporal analysis

A new variable called growing season was defined as the period running from September the 1st until August the 31st of the following year. Based on the data, three growing seasons, 2010/11, 2011/12, 2012/13 were created. The categorical variables were stratified and presented as numbers and percentages. Where appropriate, significance was assessed using the Chi square test at a level of $P<0.05$. Epidemic curves of the days when orchards became infected were presented. All temporal analyses were conducted using R version 2.15.2 (R Development Core Team, Vienna, Austria).

3.4 Spatial analysis

Spatial analyses were conducted to describe the distribution of kiwifruit orchards throughout New Zealand, and to describe the progression of the outbreak. The Longitude and Latitude coordinates of the KPIN was available for all except 83 orchards in the KVH database. A map of point data for case and control orchards was created to allow the visualization of kiwifruit orchards. As the density of orchards varied across New Zealand, adaptive edge corrected Gaussian kernel density estimation was used to smooth the location data (Bowman and Azzalini, 1997). This smoothing facilitated visualisation of the geographical clustering of orchards. All maps were created using R version 2.15.2 (R Development Core Team, Vienna, Austria), with the kernel density estimations created using the library “spatstat” (Baddeley and Turner, 2005).

3.5 Spatio-temporal analysis

The Knox test was applied to examine whether cases were close to each other in both space and time using the Excel add-in "Spatial Statistics" (SSTAT v4.70). In each region, the closeness of each case pair was simulated for 99 iterations, at critical distances and times. The critical distances of 1, 2, 3, 4, 5, 10, 20 and 30 kilometres and at critical times of 14, 30, 60 and 90 days were selected *a priori*. Significant space and time clustering was determined if the Knox test returned a P-value of less than 0.10 at any critical time and distance pairing for more than the expected number of case pairs.

The space-time K function (nearest neighbour test) was used to further evaluate space-time clustering in the regions (Diggle et al., 1995). For each region, the number of days since the start of the outbreak in that region was determined. Where enough cases were present, infected orchards were stratified by whether the orchard was confirmed positive through primary or secondary symptoms. The temporal component was described as between 1 and 30 days in daily steps, while the spatial component was between 1 and 5 kilometres in 100 metre steps. The overall significance of clustering was evaluated over 99 simulations and relationship between space and time plotted using the R version 2.15.2 (R Development Core Team, Vienna, Austria) and the "Splancs" (Rowlingson and Diggle, 1993) library. Significance was determined if the data statistic was not within the normal range.

The presence of long distance spread events was determined by simulating the spread of Psa-v during the course of the outbreak. In the simulation, the coordinates of the orchards were plotted in space and the orchards became infected over time. Each orchard was modelled with a theoretical local spread zone of 10, 20 and 30 kilometres around an infected orchard. The outbreak was started at the first infected orchard in Te Puke. Any

orchard that subsequently became infected, and was outside of the 10 kilometre zone around an infected orchard, was then coded as a long distance spread event and became part of a separate local spread cluster. Long distance spread events were further categorised as between 10 and 20 kilometres from the nearest infected orchard and greater than 20 kilometres from the nearest infected orchard. The long distance spread model was created using Google Earth 6.2 (Google Inc, 2013) and R version 2.15.2 (R Development Core Team, Vienna, Austria).

4.0 Results

4.1 Description of the outbreak

The epidemic began when the first test positive infection of Psa-v was confirmed on an orchard in Te Puke on the 5th of November 2010. As of the 18th of February 2013, 62% (2066/3309) of kiwifruit orchards in New Zealand had a confirmed infection with Psa-v on at least one vine in the orchard. In Te Puke 98% (1078/1097) of orchards were positive for Psa-v infection by the 18th February 2013 (Table 1). In the regions affected by Psa-v, the average proportion of orchards positive for the infection was 50%. In February 2013, four regions had not had a reported case of Psa-v. Of the orchards positive for Psa-v infection, 64% (1118/2066) were identified through primary symptoms (Table 2).

4.2 Temporal description of the outbreak

There was one KPIN missing the date of Psa-v infection confirmation. This KPIN was excluded from temporal analysis. The temporal pattern of Psa-v infected orchards is shown in Figure 2. Two waves of infection can be identified, starting in approximately the spring (September) of 2011 and again in the spring (September) of 2012. In total, 65% (1354/2066)

of Psa-v infections on orchards were identified in spring (Table 3). In the growing season when Psa-v was first confirmed, 11% (230/2065) of orchards were infected, 53% (1097/2065) were infected in the second growing season and 36% (738/2065) in the third growing season. On average, 33% of Psa-v infections were diagnosed in spring of the second and third growing seasons. There was a significant difference between growing season and time of year when Psa-v was diagnosed ($P<0.001$). Of the orchards infected in the 2010/11 growing season, 229 orchards were in Te Puke and one orchard was in Tauranga East (Table 4). The orchard in Tauranga East was confirmed infected on the 5th of August 2011. In the second (2011/12) growing season, 65% (719/1097) of infected orchards were located in Te Puke. In the 2011/12 growing season, Psa-v had spread to nine other regions. In the 2012/13 growing season, 17% (129/738) of newly infected orchards were located in Te Puke and by the end of study period 98.3% (1078/1097) of orchards in Te Puke were positive for Psa-v. Three new regions were identified as infected in the 2012/13 growing season.

4.2 Spatial description of the outbreak

The overall point map for the Psa-v outbreak is presented in Figure 3. Infected orchards are located predominantly in the Bay of Plenty region. The highest density of infected orchards occurred in the Te Puke region in all years of the outbreak (Figure 4). In the 2010/11 and 2012/13 growing seasons, the first orchard to have a reported Psa-v infection is situated centrally in the area with highest density of infected orchards.

4.3 Spatio-temporal description of the outbreak

The Knox test, identifying clustering of cases within space and time, showed significant interaction between some critical time and critical distance values in eight out of

13 Psa-v affected regions. In Te Puke, at the critical distances of 1 and 2 kilometres, all the critical time combinations (14, 30, 60 and 90 days) were significant between the case pairs ($P<0.01$) (Table 5). The Opotiki and the Waikato regions did not have any significant space time interaction between case orchards. The Hawkes Bay, Poverty Bay and Kerikeri regions did not have enough case pairs for analysis using the Knox test. In the remaining regions, there were significant interactions between case pairs at least at the critical time of 14 days and at a critical distance that varied between 1 and 20 kilometres.

Ten regions had enough Psa-v cases to conduct a space time K function analysis. All analysed regions showed evidence of clustering up to 30 days and up to 5 kilometres. Based on a Monte Carlo simulation with 99 iterations, the Coromandel, Tauranga East, Waihi and Whakatane regions all had significant ($P<0.001$) clustering in space and time when simulated using the space time K function (Figure 5). Tauranga West and Katikati showed significant ($P<0.05$) space time clustering when only primary symptoms were considered.

The Psa-v outbreak was modelled using the maximum local spread distance determined using the Knox test and space time K function of 20 kilometres (a 10 kilometre radius around an infected property). Long distance spread events (and subsequent local spread clusters) were identified in Te Puke, Waihi, Franklin, Opotiki, Waikato (Te Awamutu), Te Kaha (East of Opotiki), Franklin, Coromandel (Opoutere), South Auckland, Cambridge (Waikato), Hawkes Bay, Coromandel (Hikaui) and Poverty Bay (Figure 6). A further 13 spread events were identified that were between 10 and 20 kilometres from the nearest infected orchards. All other spread events were within 10 kilometres of the nearest infected orchards.

5.0 Discussion

This study has explored the spatial, temporal and space time clustering of the Psa-v outbreak in kiwifruit in New Zealand between November 2010 and February 2013. The Psa-v outbreak showed a seasonal pattern, particularly in the 2011/12 and 2012/13 growing seasons, with the majority of new infections being reported in the spring of each season. In each growing season, different regions were affected by Psa-v as the epidemic spread. In the first two growing seasons, the Te Puke region was the area that was most affected by the outbreak and by the third season thirteen regions were affected. Significant spatio-temporal clustering was identified using both the Knox test and the space time K function. Clustering indicates that the spread of Psa-v is not occurring randomly, but rather that Psa-v is spreading to orchards via local spread. Although clustering varied by region, across all regions 98% of Psa-v spread was within 10 kilometres of an infected orchard.

The Knox test identified significant clustering between infected orchards of critical distances up to five kilometres at a critical time of 14 days in seven regions. Across all regions, the maximum critical time and space clusters were identified at 90 days and 20 kilometres. The space-time K function identified the clustering of infected orchards within regions, up to 30 days and up to five kilometres from the nearest infected orchard. For both the Knox and space time K function the results for individual regions varied, but the variation was consistent between the two analyses. These results indicated that local spread is occurring in all regions at a minimum of two kilometres of an infected orchard and in a minimum time window of 14 days. However, local spread can reach a maximum of 20 kilometres within a three month period. These findings should be taken into consideration

when responding to new infections, particularly those found in areas defined by the NPMS as an Exclusion Zone.

The local spread of Psa-v can be through aerial transmission, mostly associated with wet weather (Judd et al, 2012), and orchard management practices (Judd et al., 2012; Miller and Horner, 2012; Tyson et al., 2012a; Tyson et al., 2012b). Long distance spread of the disease can be via infected kiwifruit plant material such as grafting material, seedlings, and possibly pollen along with fomite transmission through the movement of soil with Psa-v disease present via people and vehicles (Anon, 2011; Everett et al., 2012). The analyses presented in the current study did not attempt to determine the method or mechanism of local spread, but will account for any and all factors that would be creating the clustering of infected orchards in time or space. Additionally, the variation of the spatio-temporal analyses regionally suggests that the spread of Psa-v between orchards is not only contingent on factors specific to the pathogen, but also to management, demographic and environmental factors (Horner and Manning, 2012). Froud et al (unpublished data) identified several orchard specific factors; commercial variety grown, and the size, elevation and region of the orchard, as risk factors for an orchard being positive for Psa-v infection during the New Zealand Psa-v outbreak. These factors have not been accounted for in the spatio-temporal analyses presented in the current study.

As well as orchard specific reasons for the differences in the local spread parameters in each region, other factors could be affecting the mechanisms for Psa-v to spread between orchards. The density of orchards in the region, the climatic conditions and the control measures put in place during the outbreak could all have an effect on the spread of Psa-v in a region. While the density of orchards has been accounted for in the current study, the

other two factors have not. Climatic conditions have been recognised as a risk factor for Psa-v spread (Horner and Manning, 2012) and Beresford et al have developed a weather risk model based on key levels of rainfall and temperature (Anon, 2013b). The control strategies undertaken during different phases of the outbreak may have altered the spread characteristics of Psa-v within a region. Early in the response to Psa-v in New Zealand aggressive containment actions were undertaken where all Psa-v positive orchards had Psa-v symptomatic vines and a buffer zone around them cut back to the vine leaders (Anon, 2011). Once the numbers of positive orchards became larger, aggressive management was only applied to those orchards that showed the more severe symptoms and orchards with leaf spotting only were no longer managed in this way. Within the first 12 months of the outbreak the industry was no longer funding aggressive management in the Te Puke region and growers were encouraged to remove symptomatic material themselves. The current study identified that Te Puke had significant local spread up to 2 kilometres and up to 90 days in the Knox test, but significance could not be determined using the space time K function. The outbreak occurred for the longest period of time in Te Puke and the control practices changed over this period. It is also likely that the introduction of the pathogen to New Zealand was up to nine months prior to its detection in Te Puke and that the silent spread period (the period between Psa-v release into New Zealand and official detection) was extended (Anon, 2011) and this would have the most impact on orchards found in the Te Puke region. The Opotiki and the Waikato regions did not have any significant space time interaction between case orchards. This could be due to there being more than one local spread cluster in both of these regions, indicating that several long distance spread events occurred.

The observed temporal pattern consisting of epidemic waves beginning at the start of the spring growing season and the majority of primary symptoms being reported at this time, which coincides with bud break and leaf formation, identifies infection with Psa-v being reported after the winter dormancy period. However, these orchards may have become infected earlier than the appearance of new leaves. The latency period of Psa-v is unknown, although there is evidence that leaf spotting occurs between 9 and 14 days after a favourable wet weather event (Beresford, pers. comms). The association between the primary clinical sign of leaf spotting and the more severe secondary clinical signs of Psa-v is unknown. Psa-v has been shown to survive in leaf litter and pruning debris over winter (Tyson et al., 2012a), but it is unknown whether vines can become infected during this time if exposed to infected materials. Consequently, reporting time may not be closely associated with infection in plants, as there are currently so many unknown variables regarding the latent period of Psa-v or the infection pathway. This unknown latent period would have affected the spatio-temporal analyses.

The current study identified that 98% of newly infected orchards were within 10 kilometres of an infected orchard. In total 12 spread events over 20 kilometres from the nearest infected orchard and 13 spread events between 10 and 20 kilometres from the nearest orchard were identified. Based on the spatio-temporal analyses, these spread events are unlikely to be caused by local spread. Given that that the time to clinical signs and subsequent time to reporting may be variable, care must be taken when interpreting these spread events. While 12 long distance spread events with subsequent local spread clusters have been identified, any orchard confirmed Psa-v positive within that new cluster within 90 days of the first reported infected orchard could have been the first infected

orchard in that cluster. Once a region had a report of Psa-v infection, it is then likely that growers would be more vigilant in checking vines for the clinical signs of disease.

6.0 Conclusions

In this study, spatio-temporal analyses were used to investigate the regional spread characteristics of Psa-v between New Zealand kiwifruit orchards from November 2010 until February 2013. The analyses were used to determine that the outbreak was clustered in space and time, and to determine local and long distance spread characteristics. There were clear temporal and spatial components to the Psa-v outbreak in New Zealand. Epidemic waves occurred in the spring of each year since the start of the outbreak. A significant interaction between space and time was identified, with the clustering of infected orchards (local spread) identified up to 20 kilometres and a maximum of three months. The local spread characteristics of Psa-v varied by region and the variation could be attributed to different control strategies during different phases of the outbreak, the seasonality of the clinical signs of Psa-v and other regional-level risk factors. A total of 12 infected orchard clusters were identified that were further in distance from the nearest infected orchard than could have occurred due to local spread alone. The reason for the spread of Psa-v into these clusters requires further investigation. A further 13 infected orchard clusters were identified that were between 10 and 20 kilometres from an infected orchard. As 98% of newly infected orchards were within 10 kilometres of an infected orchard, these clusters also require further investigation to determine if they are independent long distance movement events or if there are other biological or climatic events that may have contributed to extreme local spread such as severe weather events.

7.0 Further work

The 25 spread events that cannot be accounted for by local spread warrant further investigation. This further investigation would aim to identify if these events are the result of the movement of high risk infected plant materials, fomite transmission, whether Psa-v infection in these areas has occurred via a current unidentified vector, or if there are other biological or climatic events that may have contributed to extreme local spread such as severe weather events. This investigation could be achieved by matching long distance spread events with at risk movement events based on Ministry of Primary Industry and KVH new region incursion investigations and interview records.

To conduct a contact network analysis between orchards that have reported becoming infected in the 2012/13 growing season to determine the current local spread pathways.

Match spatio-temporal spread patterns with outbreak management strategies, climatic and orchard-level factors. This would be particularly pertinent in the Te Puke region and regions infected in the 2012/13 growing season.

Determine if Psa-v spread is different in Hayward and Hort 16A varieties. These analyses would need to be conducted at the block level.

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Tables and Figures

Table 1: Number and percentage of Psa-v infected and uninfected orchards, stratified by region. Data from the KVH database and were current on the 18th of February 2013.

Region	Psa-v status	
	Negative n (%)	**Positive n (%)**
AUCKLAND	27 (100)	0 (0)
COROMANDEL	29 (64.4)	16 (35.6)
FRANKLIN	67 (63.8)	38 (36.2)
HAWKES BAY	50 (96.2)	2 (3.8)
KATIKATI	229 (48.6)	242 (51.4)
KERIKERI	105 (99.1)	1 (0.9)
OPOTIKI	80 (36.5)	139 (63.5)
POVERTY BAY	66 (94.3)	4 (5.7)
SOUTH ISLAND	145 (100)	0 (0)
TAURANGA EAST	87 (29.1)	212 (70.9)
TAURANGA WEST	143 (47.4)	159 (52.6)
TE PUKE	19 (1.7)	1078 (98.3)
WAIHI	7 (15.2)	39 (84.8)
WAIKATO	67 (77.9)	19 (22.1)
WANGANUI/ HOROWHENUA	27 (100)	0 (0)
WHAKATANE	44 (27.3)	117 (72.7)
WHANGAREI	51 (100)	0 (0)

Table 2: The number and percentage of Psa-v infected orchards, stratified by region and whether diagnosis was confirmed by primary or secondary symptoms during the outbreak. Data from the KVH database and were current on the 18th of February 2013.

Region	Psa-v diagnosis confirmation	
	Primary Symptoms n (%)	**Secondary Symptoms n (%)**
COROMANDEL	11 (68.8)	5 (31.3)
FRANKLIN	23 (60.5)	15 (39.5)
HAWKES BAY	2 (100)	0 (0)
KATIKATI	123 (50.8)	119 (49.2)
KERIKERI	1 (100)	0 (0)
OPOTIKI	56 (40.3)	83 (59.7)
POVERTY BAY	4 (100)	0 (0)
TAURANGA EAST	124 (58.5)	88 (41.5)
TAURANGA WEST	66 (41.5)	93 (58.5)
TE PUKE	620 (57.5)	458 (42.5)
WAIHI	22 (56.4)	17 (43.6)
WAIKATO	10 (52.6)	9 (47.4)
WHAKATANE	56 (47.9)	61 (52.1)

Table 3: The number and percentage of Psa-v infected orchards, stratified by the growing season and time of year when diagnosis was confirmed. Data from the KVH database and were current on the 18th of February 2013.

Growing season	Time of year (season)				
	Spring	**Summer**	**Autumn**	**Winter**	**Total**
2010/11	4 (0.2)	46 (2.2)	70 (3.4)	110 (5.3)	230 (11.1)
2011/12	660 (32.0)	182 (8.8)	140 (6.8)	115 (5.6)	1097 (53.1)
2012/13	690 (33.4)	48 (2.3)	0 (0)	0 (0)	738 (35.7)
Total	1354 (65.6)	276 (13.4)	210 (10.2)	225 (10.9)	2065

*The growing season is from 1st September until the 31st of August the following year

Auckland, the South Island, Whanganui/Horowhenua and Whangarei had not reported Psa-v infection on any orchards as of the 18th of February 2013.

Note: 1 missing value

Table 4: The number of Psa-v infected orchards, stratified by the growing season when diagnosis was confirmed. Data from the KVH database and were current on the 18th of February 2013.

	Growing season*		
Region	**2010/11**	**2011/12**	**2012/13**
COROMANDEL	0	1	15
FRANKLIN	0	7	31
HAWKES BAY	0	0	2
KATIKATI	0	37	205
KERIKERI	0	0	1
OPOTIKI	0	75	64
POVERTY BAY	0	0	4
TAURANGA EAST	1	132	79
TAURANGA WEST	0	24	135
TE PUKE	229	719	129
WAIHI	0	25	14
WAIKATO	0	2	17
WHAKATANE	0	75	42

*The growing season is from 1st September until the 31st of August the following year

Auckland, the South Island, Whanganui/Horowhenua and Whangarei had not reported Psa-v infection on any orchards as of the 18th of February 2013.

Note 1 missing value

Table 5: Knox test probabilities of case orchard clustering at critical times and distances, stratified by region.

Region	Number of cases	Critical distance (km)	Critical time (days)			
			90	60	30	14
Te Puke	1064	**2**	<0.01	<0.01	<0.01	<0.01
		1	<0.01	<0.01	<0.01	<0.01
Coromandel	15	**10**	-	-	0.12	0.03
		5	-	-	-	0.14
		4	-	-	-	0.11
		3	-	-	-	0.11
		2	-	-	-	0.08
Franklin	37	**4**	-	-	0.2	0.04
		3	-	0.12	0.13	0.04
		2	-	0.19	0.14	0.08
		1	-	-	-	0.14
Katikati	238	**20**	<0.01	<0.01	0.01	<0.01
		10	<0.01	<0.01	0.01	<0.01
		5	<0.01	0.01	0.03	0.02
		4	0.03	0.03	0.03	<0.01
		3	0.12	0.19	0.12	0.04
		2	0.13	0.05	0.2	0.18
		1	0.04	0.01	0.13	0.06
Tauranga East	211	**20**	0.05	<0.01	0.13	0.15
		10	<0.01	<0.01	<0.01	<0.01
		5	<0.01	<0.01	<0.01	<0.01
		4	<0.01	<0.01	<0.01	0.01
		3	<0.01	<0.01	<0.01	<0.01
		2	<0.01	<0.01	<0.01	<0.01
		1	<0.01	<0.01	0.01	<0.01
Tauranga West	159	**10**	0.06	-	-	-
		5	0.05	0.17	0.2	-
		4	0.04	0.19	-	-
		3	0.14	0.15	0.1	-
		2	0.14	0.11	0.07	0.11
		1	0.07	-	0.11	0.18
Waihi	38	**5**	-	0.06	0.05	0.01
		4	0.09	0.03	0.01	0.01
		3	0.11	<0.01	<0.01	<0.01
		2	0.08	<0.01	0.02	0.03
		1	0.17	0.05	0.09	0.1
Whakatane	116	**10**	0.08	0.19	-	-
		5	0.02	0.12	0.01	0.04
		4	0.05	0.16	0.01	0.02
		3	0.02	0.11	0.02	0.01
		2	<0.01	<0.01	<0.01	0.02

Region	Number of cases	Critical distance (km)	Critical time (days)			
			90	60	30	14
		1	<0.01	0.09	0.01	0.01

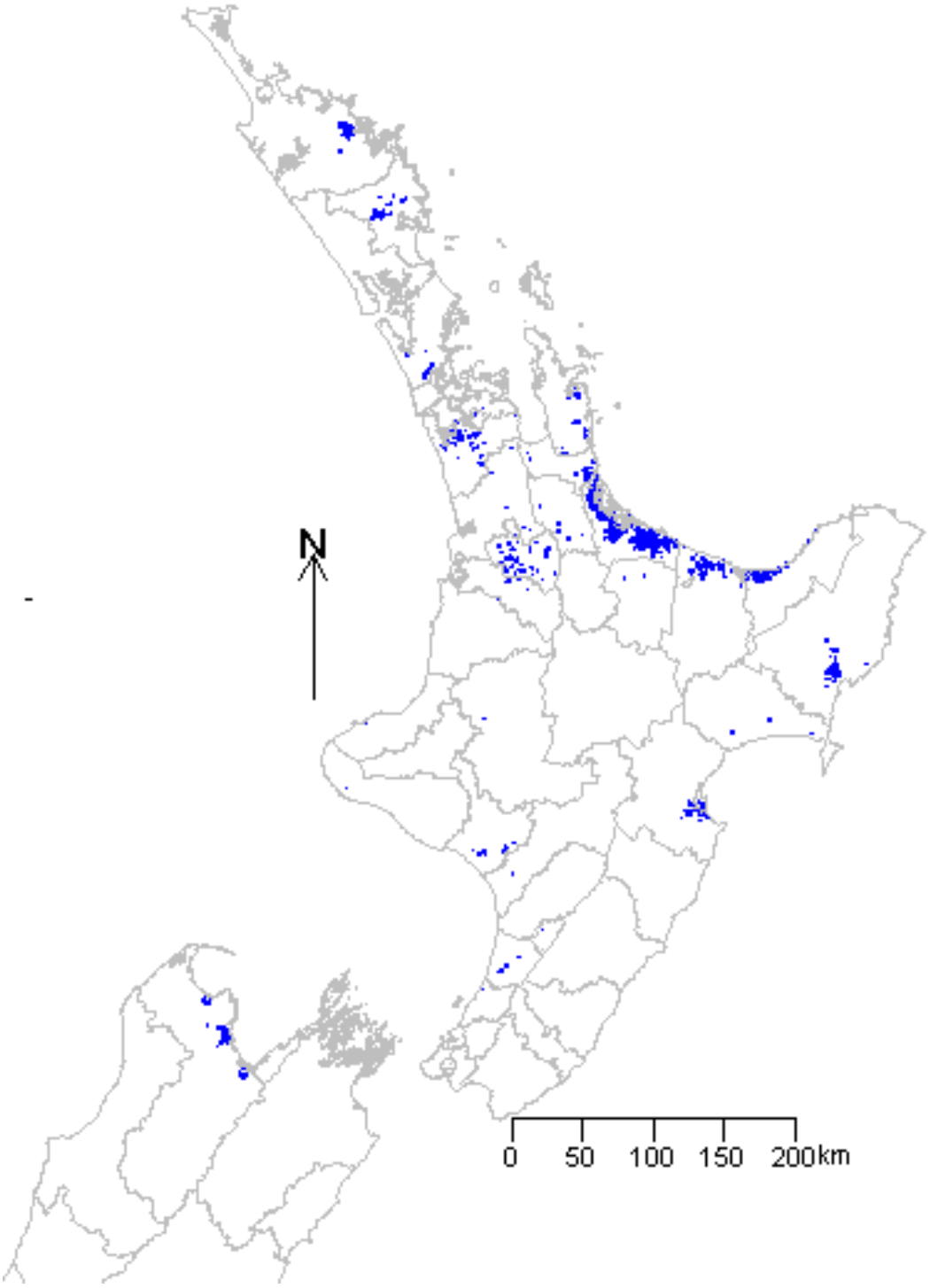

Figure 1: Location of 3309 kiwifruit orchards in New Zealand*
*Missing co-ordinate data for 32 infected orchards and 51 uninfected orchards

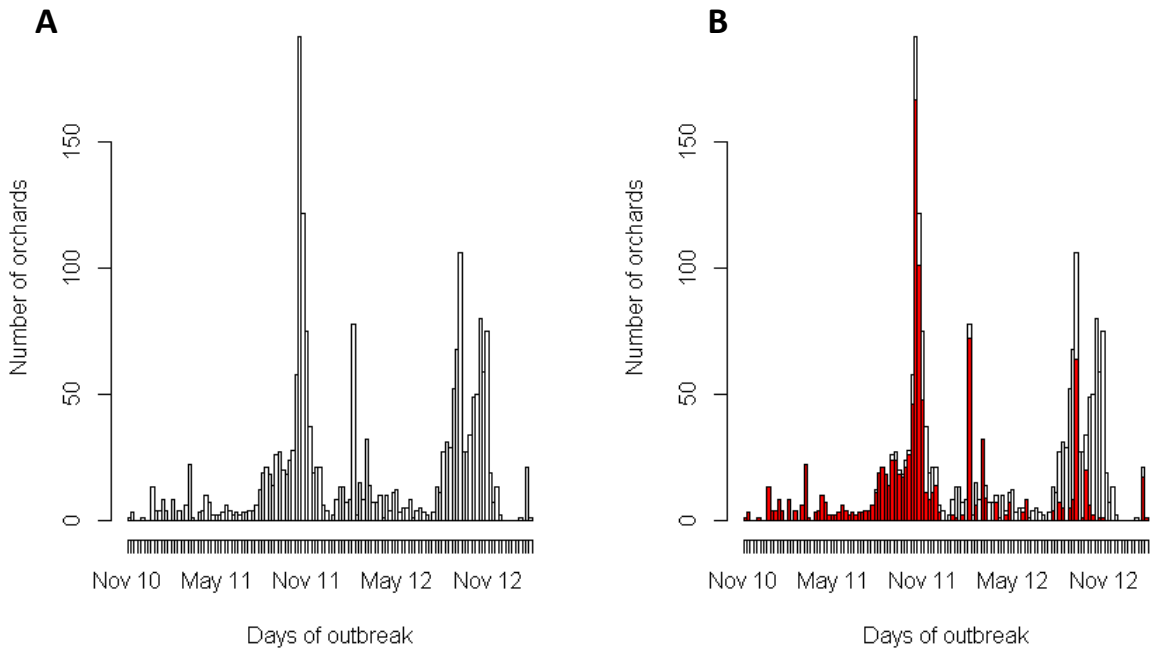

Figure 2: The epidemic curve of the Psa-v outbreak on New Zealand kiwifruit orchards (A). The epidemic curve of the number of infected orchards with orchards from the Te Puke region in red (B). Data from the KVH database and were current on the 18th of February 2013. One missing value.

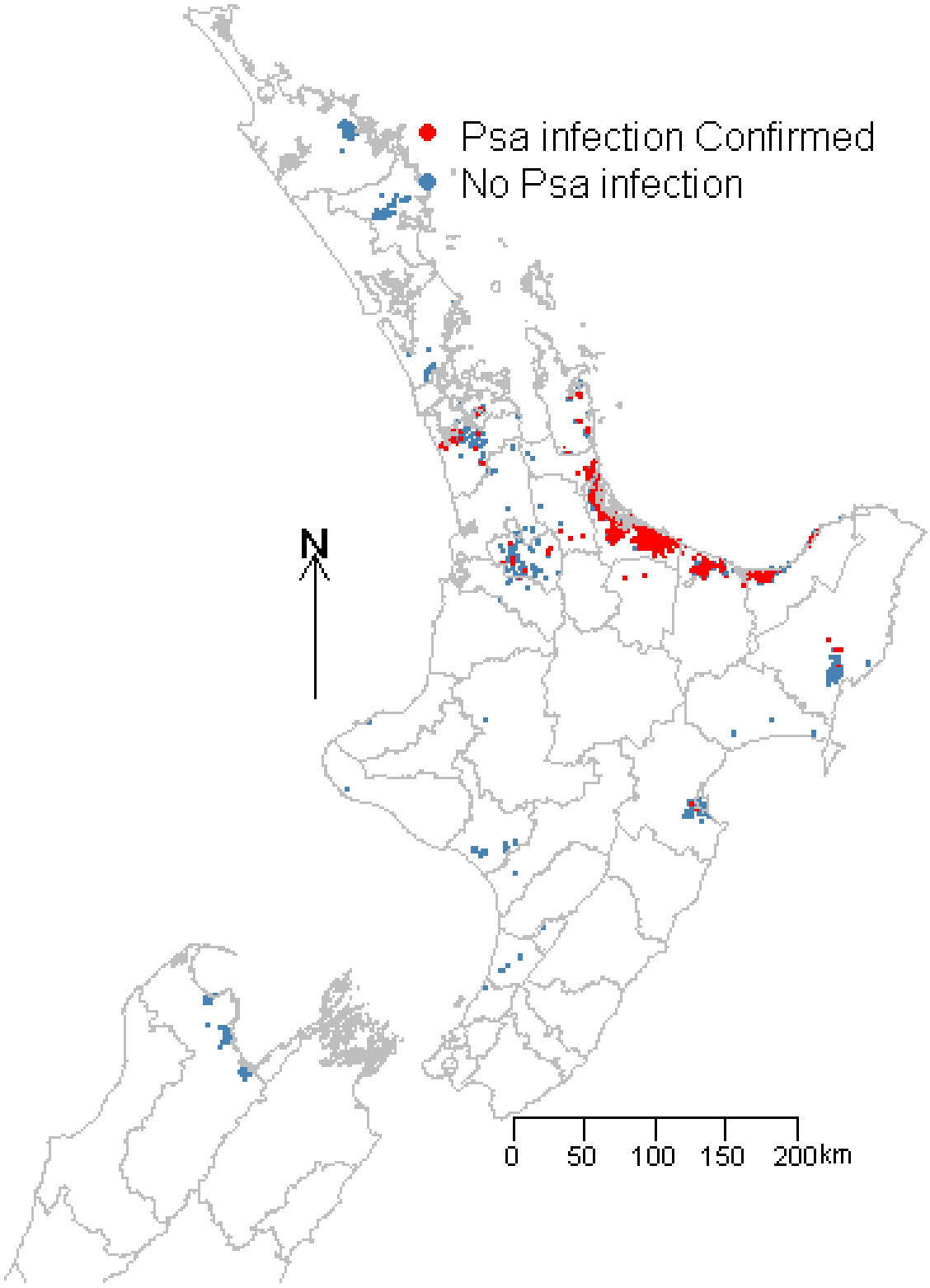

Figure 3: Point map of the infected and uninfected kiwifruit orchards in New Zealand. Data from the KVH database and were current on the 18th of February 2013*
*Missing co-ordinate data for 32 infected orchards and 51 uninfected orchards.

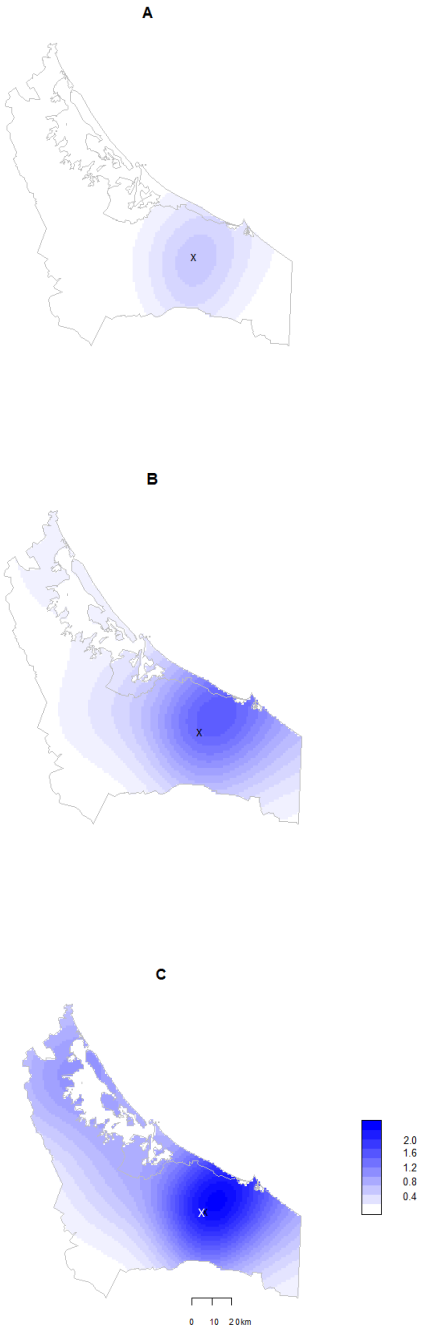

Figure 4: Kernel density maps of the Psa-v outbreak for the Western Bay of Plenty region and the 2010/11 growing season (A), the 2011/12 growing season (B) and the 2012/2013 growing season (C), shown as number of orchards per kilometre^2. X represents the first infected orchard identified on the 5th of November 2010. Data from the KVH database and were current to the 18th of February 2013*

*Missing co-ordinate data for 32 infected orchards and 51 uninfected orchards.

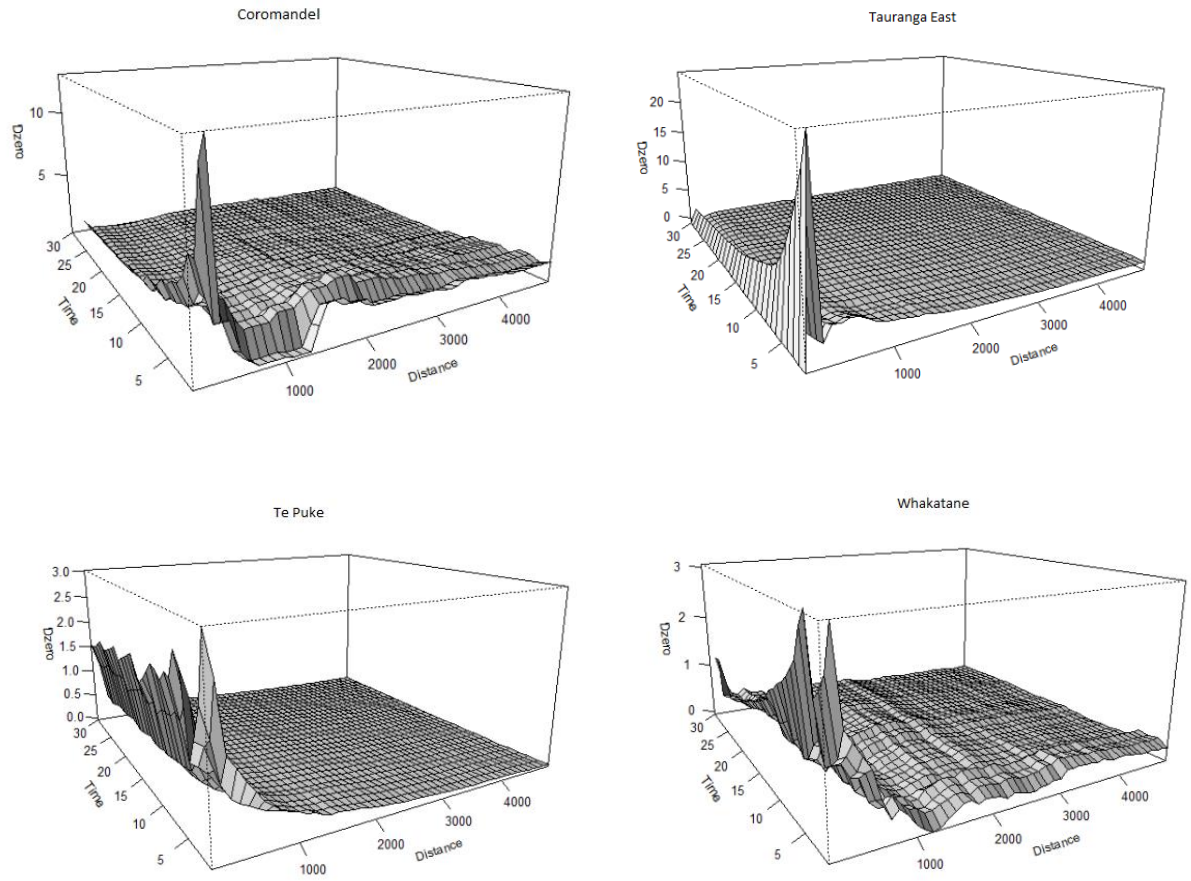

Figure 5: 3-dimensional plots of the space time K function for all the infected orchards in the Coromandel, Tauranga East, Te Puke and Whakatane regions. Data from the KVH database and were from the 5th of November 2010 to the 18th of February 2013*
*Missing co-ordinate data for 32 infected orchards and 51 uninfected orchards

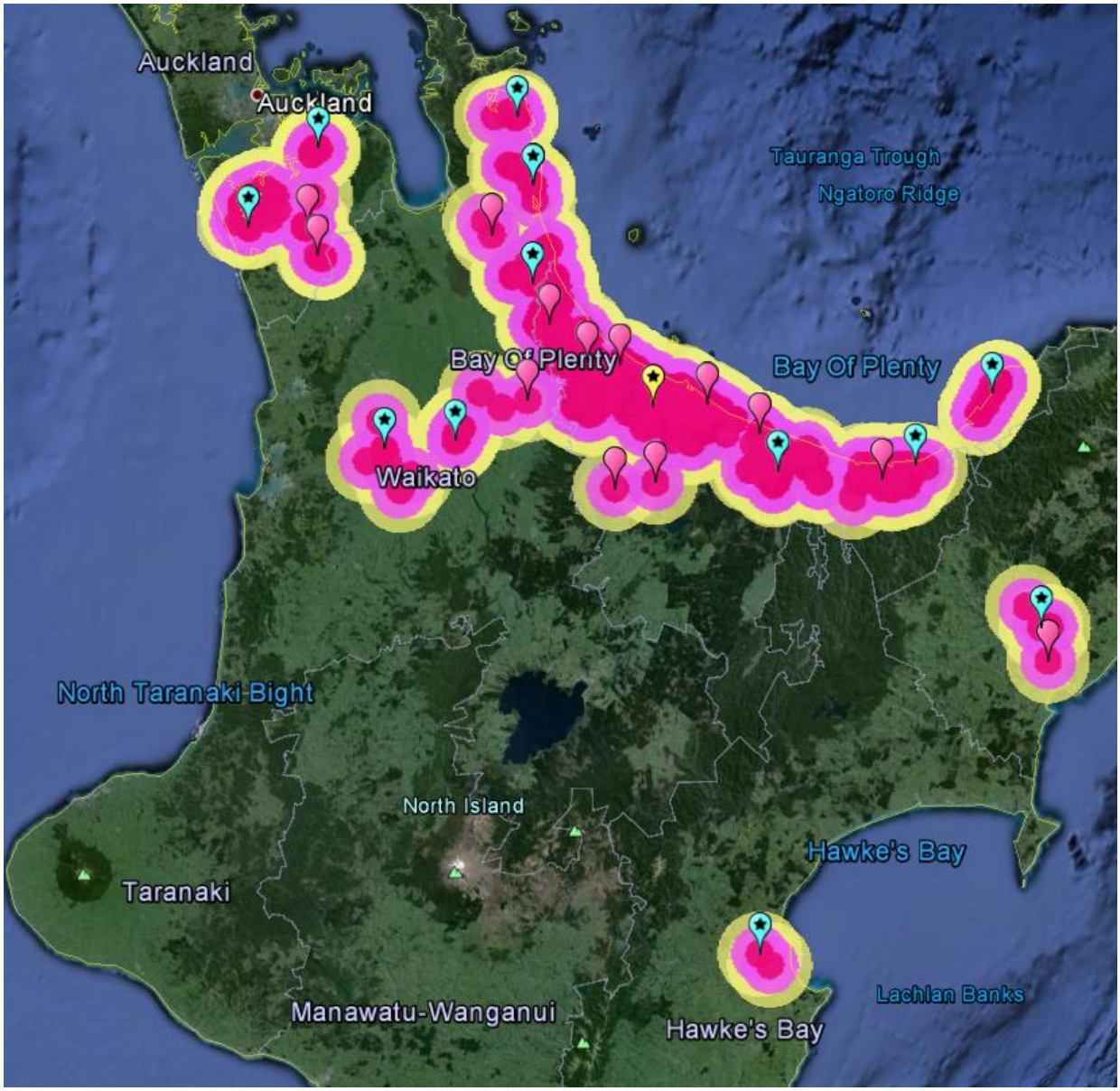

Figure 6: Google Earth image of the results of the local spread simulation. Dark pink circles show the potential Psa-v local spread of 10 kilometers from an infected orchard, light pink circles show the potential spread of 20 kilometers around an infected orchard, and yellow circles show the potential spread of 30 kilometers around an infected orchard, with a Yellow marker for the first infected orchard, Blue markers to indicate long distance spread events (>20 kilometers) and Pink markers to indicates spread events with infected orchards between 10 and 20 kilometers. Data from the KVH database and were from the 5th of November 2010 to the 18th of February 2013*

*Missing co-ordinate data for 32 infected orchards and 51 uninfected orchards