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Phosphite trials for control of *Phytophthora agathidicida* in kauri – re-evaluation after 10 years

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Executive summary

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Phytophthora agathidicida is a serious problem, killing kauri trees of all ages throughout northern New Zealand forests, and threatening the unique kauri ecosystem. Treatment with phosphite (phosphorous acid, phosphonate) is one of the few potential options for treating infected or threatened trees.

Following success with phosphite in *in vitro* and glasshouse trials with infected kauri, a series of trials in *P. agathidicida*-infested forests was conducted, testing the efficacy of phosphite for controlling kauri dieback. Trials aimed to determine if there was any reduction in the rate of symptom development in infected trees and/or improved health of trees in various stages of infection, or potential protection of kauri trees from infection in the forest environment. Trials also aimed to determine what concentrations and doses of phosphite would be effective against the pathogen, yet safe for kauri. The first forest trials with phosphite on kauri were small pilot studies established in 2011 to test phosphite rates, followed by replicated injection trials established in four infected ricker (young kauri tree) forests in 2012. Further trials were established in 2016 to evaluate lower phosphite injection rates and trunk sprays on rickers in three forests, with a further three forest trials established the same year to determine phosphite efficacy on larger trees. A final assessment of all trees within each trial was made in 2022.

The 2012 trial involved 162 ricker kauri in four infested forests (Huia, Whatipū, Omahuta and Raetea). It established that phosphite at 7.5% and 20% active ingredient, with 20 mL injected every 20 cm around the trunk circumference, provided excellent drying and healing of basal trunk lesions on all treated trees. Even 10 years after the initial application, with no further treatment, there were still no active lesions present on almost all treated trees, and in many cases, canopies of treated trees had started to regrow. This contrasted with untreated control trees, where most lesions remained active and spreading, in many cases ringbarking and killing the tree. However, in some treated trees there was severe phytotoxicity, with canopy yellowing and thinning soon after treatment application, and in extreme cases, accelerated tree mortality. This was most noticeable on smaller trees, especially those which had advanced kauri dieback symptoms pre-treatment. The phytotoxicity symptoms were predominantly on trees treated with the higher (20%) phosphite concentration, and much less common or severe with the 7.5% treatment. The earlier pilot trials had indicated that healthy trees could tolerate very high phosphite doses and eventually recover from any initial phytotoxicity symptoms, but this was not the case with diseased trees.

In an attempt to reduce the risk of phytotoxicity while still providing adequate disease control, the ‘Low rate and trunk spray’ trial was established in 2016, with 72 trees over three sites (Huia and two in Arapohue). Trees were in the ricker to advanced ricker/early mature size range. Phosphite concentrations of 7.5% and 4%, with 20 mL injected at 20-cm spacings, plus a 4% treatment at 40-cm spacings, were investigated. No phytotoxicity was noted with any of the treatments. All injection treatments resulted in healing of almost all basal trunk lesions, in contrast to untreated control trees where most lesions remained active and continued advancing. This trend remained throughout the 6 years of the trial, although there was a small number of injected trees that had active or semi-active lesions at the final assessment. Thus, there can be confidence that phosphite injections at concentrations between 4% and 7.5% are both safe and effective for treating diseased kauri, at least those in the ricker to advanced ricker size classes. Trunk spray application of phosphite was partially effective at healing lesions, but more frequent applications were required to provide control, and it was clearly inferior to trunk injection.

There is less certainty about appropriate phosphite injection rates for large mature kauri trees. Trials established on 42 large (0.5–2.5 m trunk diameter) kauri trees in three forest sites (Cascades, Puketotara, Trounson) in 2016 used phosphite rates that were very conservative, given the immense size and age of the trial trees. Phosphite was injected at a 4% concentration, with injectors at 40-cm and 80-cm intervals around the trunk. These treatments provided significant trunk lesion healing compared to untreated controls, but healing was not as complete as that observed with ricker trees. Even following a second treatment application 3 years after the initial treatment, active lesions remained on nearly half the treated trees. Comparisons of the results from the 4%/40 cm treatment used in both the ‘Low-rate ricker’ and ‘Large tree’ trials showed that a low rate effective on small trees was less effective on large trees. This suggests that higher doses (per unit of trunk circumference) will be required in large trees, to get the same degree of healing as in ricker trees. The possibility of using sapwood cross-sectional area (rather than circumference) as the determinant of dose, especially for larger trees, is discussed. Such a measure predicts higher doses for bigger trees than using circumference and may address the deficiencies in large tree treatment identified in the current work. Further large tree trials testing higher rates are required, and an outline for these is provided. In the meantime, it is suggested that scaling up from rates for ricker trees, using trunk circumference as the determinant of dose, should be both safe and more effective than the rates used in the large tree trials to date. If anything, it could still be under-dosing large trees.

Many different tree health measures were used in the course of this work, including lesion measures (activity, advance and area), and canopy measures (health score, shoot growth and photograph comparisons over time). Lesion assessments were the most useful measure in the short term, providing useful results and discrimination between treatments within a year of application. Canopy measures were more informative long-term, with comparison between pretreatment canopy photographs and current canopy status proving to be the most accurate and least subjective measure of canopy changes following treatment.

Overall, this series of trials has provided overwhelming evidence for the efficacy of phosphite injection for the treatment and control of *P. agathidicida* infection in kauri. Lesion healing, cessation of tree decline, and eventual canopy recovery give confidence that phosphite injection is a useful tool for the management of kauri dieback.

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1 Introduction

Phytophthora agathidicida (formerly known as *Phytophthora* taxon Agathis or PTA) is a serious problem, killing kauri trees of all ages in forests in Auckland, Northland, and Coromandel (Beever et al. 2009). The pathogen seriously threatens this taonga tree and the ecosystem it supports. Treatment with phosphite (phosphorous acid, phosphonate) is one of the few potential options for treating infected or threatened trees. Phosphite is widely used internationally on a range of *Phytophthora* diseases on many plant species, including multiple horticultural crop and forest trees (Erwin & Ribeiro 1996: Crane & Shearer 2014). It works by a combination of direct suppression of *Phytophthora* and stimulation of the host-plant defences (Smillie et al. 1989; Marin et al. 2023). Its predominant use is in horticulture, with some use in forest systems (Crane & Shearer 2014). In New Zealand, phosphite is commonly used for *Phytophthora* control in avocados, apples, strawberries and other crops.

In vitro tests showed that *P. agathidicida* was very sensitive to phosphite, and glasshouse trials showed that phosphite injections could protect kauri seedlings from *P. agathidicida* (Horner & Hough 2011, 2013). Following the success of the glasshouse trials, forest trials were established to determine whether phosphite treatment had any efficacy against *P. agathidicida* in forest kauri trees, whether it could reduce the rate of symptom development in infected trees and/or improve the health of trees in advanced stages of infection, or potentially protect kauri trees from infection in the forest environment. It was also hoped to determine what concentrations and doses would be effective against the pathogen, yet safe for kauri. The first forest trials with phosphite on kauri were small pilot studies established in 2011. The first replicated forest trials were established in 2012 on young (ricker) trees in four forests. Further trials were established in 2016 to evaluate lower phosphite rates and trunk sprays on rickers in three forests, with a further three forest trials established the same year to determine phosphite efficacy on larger trees.

The expectation was that the trials would each take at least 5 years to give definitive results. That time period was considered necessary to achieve good discrimination of symptom development between treated and untreated trees, and to determine the longer-term robustness of any treatment effects. In 2022 it was decided to re-evaluate all the forest trials to investigate the long-term effect of treatment. The current report includes results from throughout the studies, including reassessments made in 2022, 6–10 years after initial treatment application. Parts of this work have been summarised in previous reports and publications (Horner et al. 2015, 2019, 2020a, 2020b, 2021). This report incorporates and supersedes these previous reports.

2 Phosphite rate pilot trials 2011

Before starting the main forest trials, it was necessary to determine the phosphite dose rates that were safe to use on forest kauri. There were no serious signs of phytotoxicity in glasshouse trials (Horner & Hough 2011). Although there were leaf scorch symptoms in branches within a few centimetres of the injection point in kauri seedlings injected with 150 mL/L (=15%) phosphite, leaves otherwise remained healthy. From these seedling trials, kauri was considered reasonably tolerant to phosphite treatment.

A range of rates have been used for trunk injection of phosphite into trees of various species suffering from *Phytophthora* infection. Tree canopy diameter or trunk circumference are often used for calculation of the volume of chemical that should be injected. For example, the label instruction for injection of Agrifos600® into avocado in New Zealand is 20 mL of 15% phosphite per 1 m canopy diameter. However, canopy calculations are difficult with kauri, especially in a forest situation. For all the current trials, trunk circumference was used to determine rates applied to individual trees.

2.1 Waipoua rate pilot trial

Healthy kauri ricker trees growing beneath a power line in Waipoua forest (Northland), destined to be felled within the next few years, were selected for the rate trial in November 2011. A rate of one 20-mL injection of 15% phosphite (=250 mL Agrifos600®/L) per 20 cm trunk circumference was used as a standard treatment. To determine the effects of extreme rates of phosphite, 20 mL of undiluted Agrifos600 (60% phosphite solution) per 20 cm trunk circumference was applied to other trees. This was to determine what might happen if people mistakenly applied undiluted phosphite to trees. In checks of these trees 2–3 weeks after treatment, no obvious symptoms were reported. On this basis, the initial treatment application in the main ricker trial sites at Huia and Whatipū proceeded, using 20 mL of 20% phosphite per 20 cm trunk circumference (see Section 3.1 below). However, subsequent checks of the Waipoua trees in late January 2012, 12 weeks after treatment, revealed several symptoms ranging from yellowing or browning of leaves to loss of many leaves and even some branchlets (Figure 1). With the branchlet loss, the normal abscission that occurs with kauri branches appeared to occur prematurely and rapidly in response to treatment with phosphite. These symptoms were noted with both the 15% and 60% injections. Further checks of the Waipoua trees in June 2012, 7 months after treatment, showed that many of the leaves and branchlets that were brown in late January had abscised and dropped. The abscission zones appeared normal (Figure 1). The remaining canopy on these trees appeared healthy and green. Further checks of these trees were made in January and June 2013 and annually thereafter until 2018. All trees showed signs of new growth over the 2012/13 and all subsequent seasons, indicating that the trees were able to survive and ultimately grow out of the initial toxic shock of extreme doses of phosphite (Figure 2). Apart from the initial response of leaf and twig loss, there was no sign of long-term adverse effects from the phosphite treatment on healthy trees. No bleeds or other trunk symptoms, such as those observed in Huia and Whatipū sites discussed below, were observed in any of the injected Waipoua trees. When the trees were checked in 2023, most had been felled because of their threat to power lines. The two remaining trees appeared healthy, with no sign of canopy or trunk problems.

Figure 1. Premature leaf browning and abscission of kauri branchlets following trunk injection with a high concentration (60%) of phosphite at the Waipoua pilot rate trial site, Northland. Similar symptoms also occurred with 15% and 20% solutions of phosphite.

Pre-treatment (November 2011).

7 months post-treatment (June 2012).

3 years 9 months post-treatment (August 2015).

Figure 2. Kauri trees before and after trunk injection with an extremely high concentration of phosphite at the Waipoua pilot rate trial site, Northland. In November 2011, the kauri on the right was injected with 60% phosphite solution; the tree on the left was left untreated. In the lower two photographs, taken 7 months and 3 years 9 months after the injection, note the thinner, but otherwise healthy canopy in the treated tree on the right.

2.2 Huia rate pilot trial

Following the observation of phytotoxicity symptoms in the Waipoua trees in late January 2012, a second pilot rate trial was established at the Huia site (Te Wao Nui ā Tiriwa/Waitākere Ranges), adjacent to the main trial area (see Section 3.1 below). None of the trees in this pilot study were showing kauri dieback symptoms. Trees were treated with a range of rates, as follows:

A. 15% phosphite, 20 mL per 20 cm trunk circumference
B. 10% phosphite, 20 mL per 20 cm trunk circumference
C. 15% phosphite, 20 mL per 30 cm trunk circumference
D. 20% phosphite, 20 mL per 40 cm trunk circumference
E. 7.5% phosphite, 20 mL per 20 cm trunk circumference
F. Untreated control.

Each treatment was applied to two trees. Photographs were taken of the tree canopies at the time of treatment application, and these were used as references for determining changes in canopy health in assessments made 6, 8, 12 and 20 weeks after treatment, and annually thereafter for 5 years. Leaf yellowing, browning and leaf drop were observed in many of the treatments (Table 1, Figure 3), although symptoms were not as severe as seen in the Waipoua trial. Even with the lowest concentration of phosphite applied (7.5%), one of the two trees showed slight yellowing and minor leaf drop, although the tree remained healthy.

By June 2013, 17 months after the initial treatment, and in all subsequent assessments, tree canopies looked green and healthy, regardless of the application rate (Figure 3). This suggests that although trees were initially adversely affected by the high rates, they recovered and continued to grow normally.

From 3 years post-treatment, bark flaking in line with injection points was noted in some trees, and in the final assessment of these trees (February 2017) minor trunk cracks or bleeding were noticed in most treatments (Table 1). This will be discussed further with the main ricker trial analysis below (Section 3). No formal assessments of tree health have been made in this pilot site since 2017, but brief checks of tree health in 2018, 2019 and 2022 indicated healing of the trunk cracks noted in earlier assessments.

2.3 Rate selection

The Waipoua and Huia pilot rate trials demonstrated that injection of high rates of phosphite into kauri trees may result in severe phytotoxicity symptoms. Symptoms include leaf yellowing, browning and abscission, and in some cases premature twig or lower branch abscission. But all the trees in these pilot rate trials appeared to recover, and within 2 years canopies were green and, in most cases showing signs of new growth. It must be noted that these rate trials were carried out on healthy trees. Responses to phosphite may differ on trees with severe symptoms of kauri dieback, as was observed in the main phosphite trials discussed below.

As a result of the Waipoua and Huia pilot rate trials, the lower rate of 7.5% phosphite was selected as the standard for the Northland sites and the remaining treatments of the Huia and Whatipū sites (see below). This low rate was selected as a precaution against causing severe phytotoxicity in trees. As will be discussed later, even the 7.5% concentration may be too high in some circumstances.

Figure 3. Phytotoxicity symptoms in kauri trees following trunk injection with phosphite, Huia pilot trial site, Waitākere Ranges. Clockwise from top left: Pre-treatment (January 2012), 12 weeks post-treatment (April 2012), 20 weeks post-treatment (June 2012), 72 weeks post-treatment (June 2013). The tree at the lower/centre of each picture was injected with 20 mL of 15% phosphite per 20 cm trunk circumference. The tree on the upper left of each picture was injected with 20 mL of 10% phosphite per 20 cm trunk circumference. Note the yellowing and browning of leaves in the photograph taken 12 weeks post-treatment, and the thinner, but otherwise healthy canopies after 20 and 72 weeks.

Table 1. A summary of notes on kauri tree canopy health 6, 8, 20, 50, 72 weeks and 5 years after trunk injection with various concentrations of phosphite at the Huia trial site. Tree canopies were compared with photographs taken on the day of treatment application. Notes on trunk symptoms are at the 5-year assessment only. 'In line' refers to in line with injection points. All notes refer to observations on two trees in each treatment.

	Phosphite (%)	Volume (mL) per cm trunk circumf.	6 weeks	8 weeks	20 weeks	50 weeks	72 weeks & 5 years	5 years (trunk)
A	15	20/20	Most leaves yellow, some brown, esp. on small shoots	Many leaves yellow or brown, some leaf drop	Thinner canopy. Remaining leaves green and healthy	Thinner canopy. Leaves green and healthy	Slightly thinner canopy. Leaves green and healthy	Slight swelling in line with injection
B	10	20/20	No change	1 tree slightly yellow, 1 tree very yellow, some brown leaves, minor leaf drop	Thinner canopy. Remaining leaves green and healthy	Slightly thinner canopy. Remaining leaves green and healthy on 1 tree, yellow on other	Slightly thinner canopy. Leaves green and healthy	Swelling in line. Minor crack in line (2/2 trees)
C	15	20/30	Similar to photograph, but slightly more yellow	Very yellow leaves and many brown. Some leaf drop	Thinner canopy. Remaining leaves green and healthy	Slightly thinner canopy. Leaves green and healthy	1 tree similar to photograph, other tree slightly thinner canopy. Leaves green and healthy	Swelling in line. Minor crack in line (1/2 trees)
D	20	20/40	Similar to photograph, but 1 tree slightly yellow	Some yellow and brown leaves, minor leaf drop	Thinner canopy. Remaining leaves green and healthy	Slightly thinner canopy. Leaves green and healthy	1 tree similar to photograph, other tree slightly thinner canopy. Leaves green and healthy	Swelling in line. Minor crack in line (1/2 trees)
E	7.5	20/20	Similar to photograph, but 1 tree slightly yellow	1 tree similar to photograph, other tree with many yellow or brown leaves	1 tree similar to photograph, other tree slightly thin canopy, leaves green/healthy	1 tree similar to photograph, other tree slightly thin canopy. Leaves green and healthy	1 tree similar to photograph, other tree slightly thinner canopy. Leaves green and healthy	Swelling in line. Minor crack in line (1/2 trees)
F	0	0	No change	No change	No change	No change	No change	Bark flaking

3 Ricker trials 2012

3.1 Methods

3.1.1 Trial site and tree selection

After consultation with the Department of Conservation (DOC), regional councils, and Mana whenua iwi/hapū, four sites were selected for the main trials testing phosphite efficacy. Trial sites were:

1. Huia dam, Te Wao Nui ā Tiriwa (Waitākere ranges), Auckland
2. Whatipū, Te Wao Nui ā Tiriwa (Waitākere ranges), Auckland
3. Raetea Forest, Mangamuka ranges, Northland
4. Omahuta Forest, Mangamuka ranges, Northland.

Both the Auckland sites were in naturally regenerating forest, in stands of kauri rickers (Figure 4). Both Northland sites were plantation kauri, planted by the New Zealand Forest Service in the 1950s and 1960s (Figure 4). All four sites had confirmed diagnoses of *P. agathidicida* in soil or trees, and subsequent checks showed widespread distribution within the soil at each site.

The trials were established January to March 2012. Only kauri trees showing symptoms consistent with *P. agathidicida* infection (lesions or bleeding sap at the base of the trunk, or thinning or yellowing canopies) were included in the trial. There were 51, 54, 15 and 42 trees in the trials at the Huia, Whatipū, Omahuta and Raetea sites respectively, a total of 162 trees. Most trees were at the ricker or advanced ricker stage, with girths ranging from 35 to 130 cm (Figure 5).

3.1.2 Pre-treatment tree assessments and trial design

Before treatments were assigned to trees, all potential trial trees were mapped, measured (girth), and canopy symptoms were scored on the Kauri Dieback Programme (KDP) 1–5 scale (1 = healthy, no signs of dieback, 2 = slight canopy thinning, 3 = thinning and some branch dieback, 4 = severe dieback, 5 = dead). Further refinement in canopy health scoring was made by breaking down categories to "+" or "-" (e.g. 3-, 3, 3+). For analysis purposes, 0.3 was added or subtracted from the numerical score so 3-, 3 and 3+ became 2.7, 3.0 and 3.3. Canopy photographs were taken from reference points for future comparisons of canopy, and the trunk base was photographed at cardinal points. At the start of the trial, most trees were showing moderate to advanced canopy symptoms, with canopy health scores mostly in the "3" categories, with fewer in the "1", "2" and "4" categories (Figure 6).

Basal trunk lesions (if present) were measured, noting position, height, and width. In many cases lesion margins were marked using a chinagraph pencil or paint pen for future reference and measurement. Lesion activity (i.e. whether they appeared fresh and active or dried and inactive) was noted. Once sufficient trial trees were identified, they were assigned into groups at each site, based on similarity of symptoms. This exercise was done away from the forest, using only the information previously collected for each tree. Trees within each group of 'similar' trees were then randomly assigned treatments, such that there was an even number of trees from each treatment in each disease severity group. This ensured that across each site, the 'average' disease severity of trees in each treatment at the start of the trial was similar.

3.1.3 Treatment application

Trees were injected with either a high (20% active ingredient (a.i.), Waitākere sites only) or low (7.5% a.i., all sites) phosphite concentration (Agrifos600®) (Table 2, Figure 7) using spring-loaded Chemjet® tree injectors (Figure 8) into a 6-mm diameter hole drilled 30–40 mm deep, 0.4–0.8 m above the ground. Both concentrations were applied at a dose rate of 20 mL of phosphite solution injected per 20 cm trunk circumference. Some trees were left untreated as controls. In January 2013, 10 to 12 months after the initial treatment, half the previously treated trees at each site were re-injected with a low (7.5%) concentration of phosphite, regardless of whether they were treated with the high or low rate at the start of the trial. Remaining injected trees and untreated controls were left untreated, making a total of five different treatments in the trial (Table 2).

All phosphite treatments were a single dose of 20 mL at 20-cm intervals around the trunk circumference, injected into the trunk 0.4 to 0.8 m above the ground. Following removal of the injector, the drilled hole was filled with silicon sealant. All five treatments were applied at Huia and Whatipū. Only treatments 1, 4 and 5 were applied at Omahuta and Raetea. There were no further applications of phosphite after January 2013.

Table 2. Treatments applied and number of trial trees on four sites for testing phosphite for control of *Phytophthora agathidicida* in kauri. Hu = Huia dam site, Waitākere Ranges; Wh = Whatipū site, Waitākere Ranges; Ra = Raetea site, Mangamuka Ranges; Om = Omahuta site, Mangamuka Ranges.

Treatment		Hu	Wh	Om	Ra	Total
1. Untreated control	-	11	12	5	14	42
2. ‘High Phos/low Phos’	20% phosphite (January 2012) +7.5% phosphite (January 2013)	11	11	-	-	22
3. ‘High Phos/nil Phos’	20% phosphite (January 2012)	9	11	-	-	20
4. ‘Low Phos/low Phos’	7.5% phosphite (March 2012) +7.5% phosphite (January 2013)	10	10	5	14	39
5. ‘Low Phos/nil Phos’	7.5% phosphite (March 2012)	10	10	5	14	39
Total		**51**	**54**	**15**	**42**	**162**

3.1.4 Post-treatment assessments

In January/February and June/July each year for 5 years post-treatment, and again 8 and 10 years post-treatment (February 2020 and March 2022, respectively), tree canopy and trunk health assessments were made. Binoculars were used to check for current season shoot growth which was scored on a scale of 0 (no growth), 0.5 (slight growth), 1 (good/normal growth) or 2 (vigorous growth). Tree canopy health and vigour were compared with those in the photographs taken at the start of the trial. The scoring system, based on the original photographs and comparing canopy health and density was: -2 = tree dead, -1 = substantially worse, -0.5 = slightly worse, 0 = similar, 0.5 = slightly better, 1 = substantially better. The subjective ratings of ‘substantially’ or ‘slightly’ different were based on whether the difference was immediately obvious (substantial) or whether the original photograph and current canopy had to be examined carefully, making comparisons down to the individual twig or leaf level (slight). Five, 8 and 10 years after treatment application, canopy health was re-scored on the same 1 to 5 scale used in pre-treatment assessments.

At each assessment time, the maximum width and height dimensions of lesions at the base of the trunk were re-measured. Where margins on particular lesions had been marked, any advance of the lesion margin was measured 6-monthly up until 2017. However, lesion margin mark assessments could not be done in 2020, as in the intervening 3-year period more than 90% of the markings indicating lesion margin had been lost through natural bark peeling, sap bleeding or fading. Lesion activity, as indicated by freshly oozing sap, was scored as follows:

0 = not active (all bleeds hard and dry, cannot dent with fingernail)
0.2 = probably not active (bleeds mostly hard and dry, but possible to make dent with fingernail)
0.5 = probably active (slightly soft bleeds, easily dented with fingernail, but not very sticky)
1 = active (soft and sticky bleeds, obviously recent)
2 = very active (copious, very soft and sticky ooze, pus-like).
Where lesions had dried up and lesion margins had cracked and peeled back, a score of -0.1 was assigned. Although the lesion activity scoring scale is slightly arbitrary, it allowed unbiased assessment of each lesion, and comparison across treatments. Either individual lesion scores or the maximum score for any tree at each assessment time were used in the final data analyses.

For analyses of basal lesion areas, a roughly triangular lesion shape was assumed, with lesion areas approximated by multiplying height × width × 0.5. Data for lesion area in 2017 or 2020 were expressed as percentages of the area pre-treatment in 2012. To avoid problems with either dividing by zero or taking the log of zero, 0.5 cm^2 (the smallest lesion size) was added to all the sizes. These data were then log transformed, and averages were calculated for each site-treatment-year combination. The means for 2017 and 2020 were compared between sites and treatments using analysis of variance on the log-transformed percentages.

Approximately 2.5 years after the first phosphite treatments, small cracks were noticed in the trunks of some trees, in line with injection points. From the 3-year assessment (February 2015) onwards, the extent, dimension and orientation of bark peeling, cracking or bleeding were recorded systematically, focusing on effects in line or associated with the injection points. Lesions associated with *P. agathidicida* infection were not considered as part of this scoring. To simplify the analysis, trunk responses were grouped into the following categories for each tree: 0 = no symptom, A = minor bark flaking, B = bark flaking all around base of trunk, C = bark flaking in line with injection, D = minor bleed, E = moderate bleed, F = major bleed. If the tree died, the most severe score recorded to date was retained for data analyses.

Figure 4. Trial sites for testing phosphite for control of *Phytophthora agathidicida* in kauri. Top left, Huia dam site, Waitākere Ranges; top right, Whatipū site, Waitākere Ranges; bottom left, Raetea site, Mangamuka Ranges; bottom right, Omahuta site, Mangamuka Ranges.

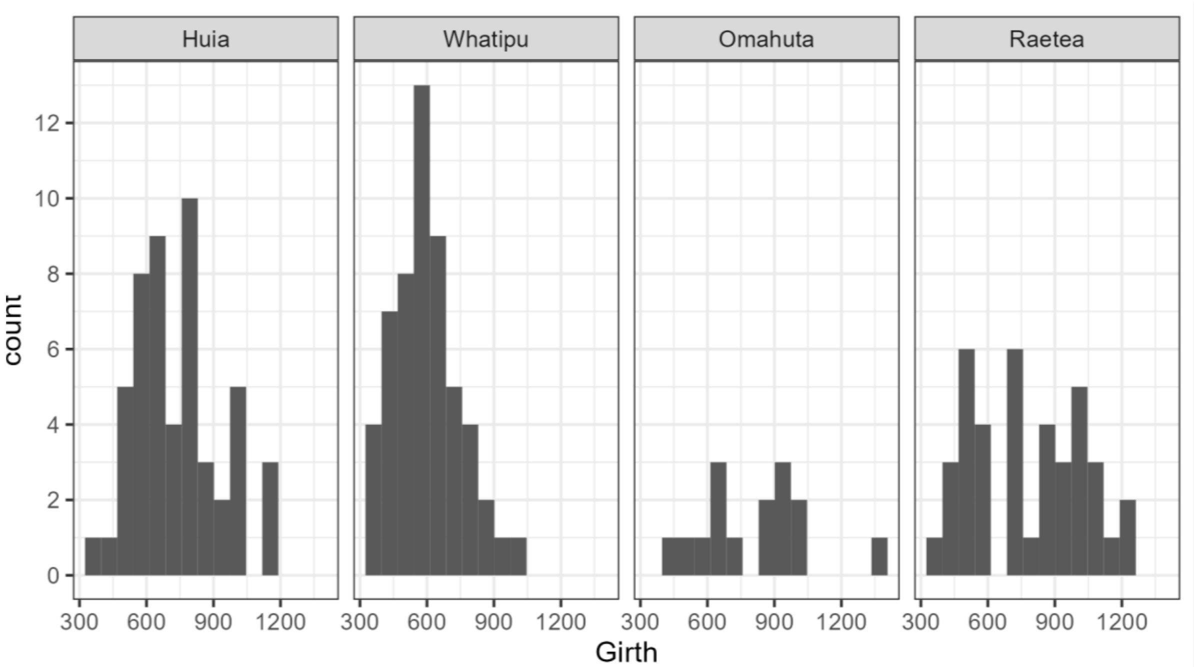


Figure 5. Range of kauri tree girths (mm) at the start of the 2012 ricker phosphite treatment trial. ‘Count’ = number of trees in each size class.

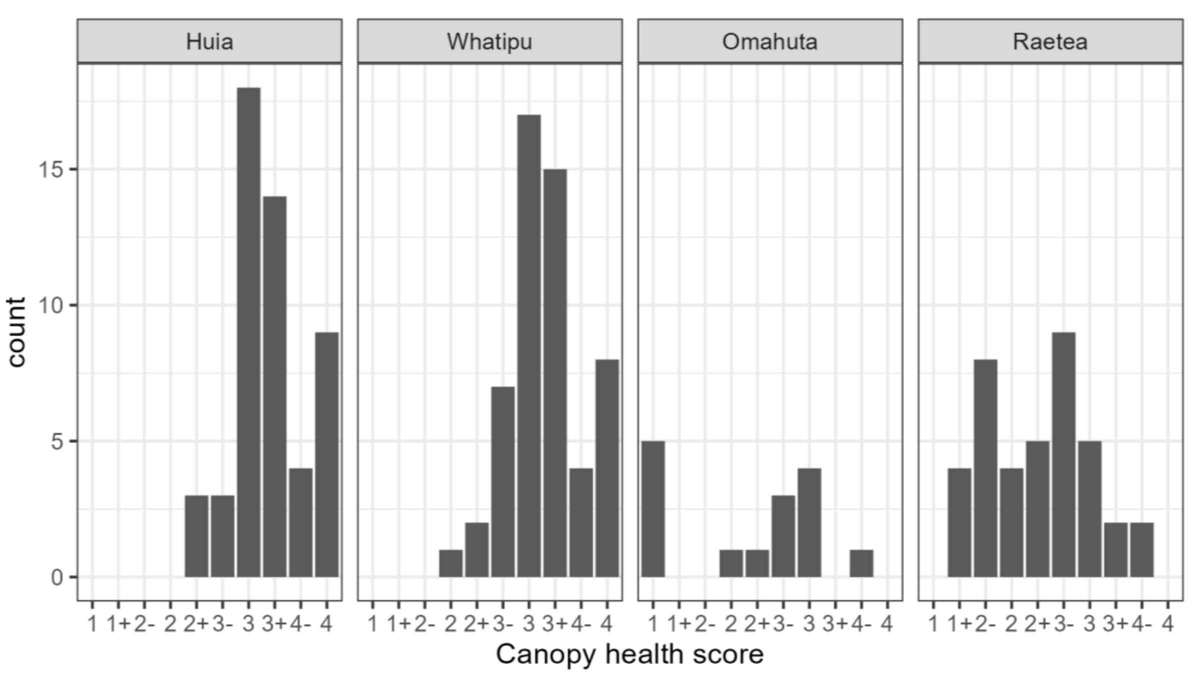


Figure 6. Pre-treatment canopy health of trial kauri trees, pooled over the four trial sites. Canopy scores: 1 = healthy signs of dieback, 2 = canopy thinning, 3 = thinning and some branch dieback, 4 = severe dieback, 5 = dead. ‘Count’ = number of trees in each health score category.

Figure 7. Trunk injection of a kauri tree with phosphite at the Huia trial site, Waitākere Ranges.

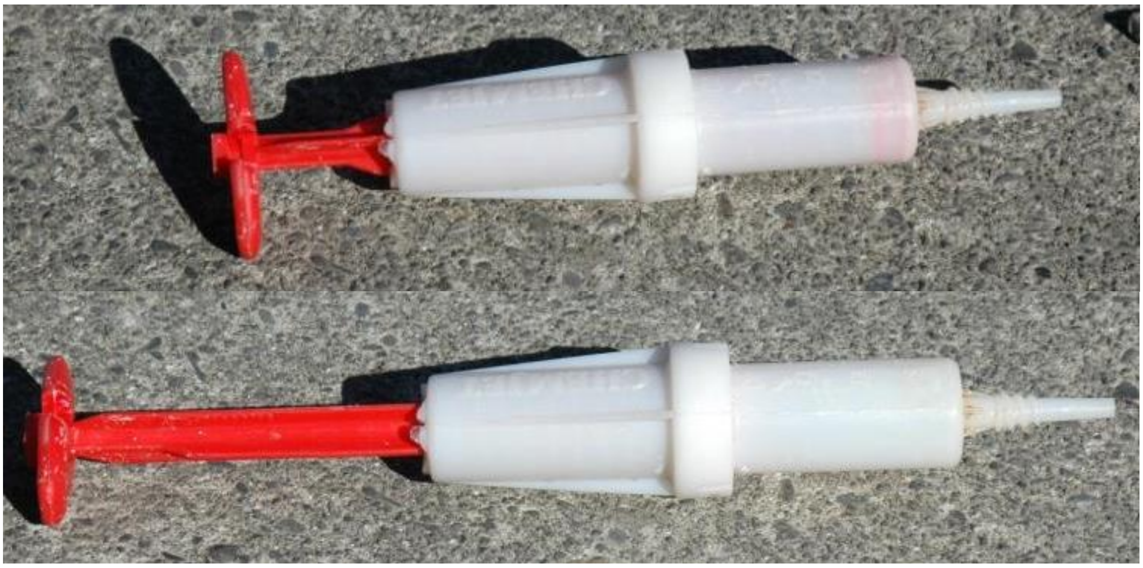


Figure 8. Chemjet® trunk injector used for injection of phosphite into kauri trees.

3.2 Results and discussion

3.2.1 Injection point healing

Most injection points healed well. Although the points sometimes bled for a few weeks, they usually healed well and within 6 months were generally hard and dry. Often the silicon plug was expelled as the tree oozed sap and healed over. After 2 to 3 years, there was often superficial bark cracking around the plug, with the bark eventually expelled and sealed beneath (Figure 9). Very occasionally, trees continued bleeding from injection wounds for more than a year (Figure 10). There was no obvious reason why some trees did this; most healed quickly and well.

Figure 9. Top left and centre: injection point healing, 3 years after phosphite trunk-injection of kauri trees. Note bark peeling around the injection point, with eventual expulsion of the plug (bottom left). Top right: natural sealing of unplugged injection hole, 30 min after injection (hole = 6 mm across).

Figure 10. Prolific bleeding from injection points (left), which occurred in about 3% of kauri trees. More typical bleeding is shown in the centre and right photographs.

3.2.2 Lesion assessments

At the start of the trial (pre-treatment 2012), lesions ranged from none present on some trees, to substantial active bleeding lesions on others. The percentage of trial trees with lesions present at the start was 93, 79, 54 and 33% of trees at the Omahuta, Raetea, Huia and Whatipū sites, respectively. The balanced method of treatment assignment across trees at the start of the trial, ensured that on average there were only small treatment differences in lesion presence or activity at the start.

3.2.2.1 Lesion activity

Within a year of treatment, there was an obvious difference in the lesion activity in treated and untreated trees, and this difference persisted for the duration of the trial. All lesions on treated trees, regardless of the phosphite dose applied, stopped advancing and dried up. In most cases there was bark cracking around the edge of the lesion and a peeling back of the diseased tissue (Figure 11). Bark behind this peeled back area was generally healthy, with no sign of lesion activity. At the 5-year assessment, old lesions on many of the treated trees had peeled back well below the initial margin, revealing healthy bark beneath, and this trend was reinforced at the 8- and 10-year assessments. Cracking and healing were also sometimes observed around lesions on untreated control trees, indicating that some untreated trees were attempting to repel the infection. However, in many such cases on untreated trees the lesion continued to advance beneath or around the peeled bark, with fresh sap oozing beyond the original canker margin (Figure 11). In many instances, lesions on untreated trees remained active and continued advancing with no signs of healing.

The trend in lesion healing noted at the 1-year assessment continued for the subsequent 9 years of assessments. In almost all phosphite-treated trees there was no further lesion activity noted after the 6-month assessment (Figures 12 and 13). Five years after the initial treatment, only two out of 120 phosphite-treated trees had "active" lesions; both were at the Raetea site, and both were heavily pig-damaged on the trunk and around the roots. At the 8- and 10-year assessments, with one exception, all trees recorded as having 'probably active', 'active' or 'very active' lesions were in the untreated control treatment, with no active lesions noted on any phosphite-treated trees (Figures 12 and 13). The one exception was a single tree noted with a 'probably active' lesion at Omahuta. Fitting a linear mixed effects model to lesion activity data, with fixed effects for date and treatment, and random effects for site, tree, site × date and site × treatment, indicated both the fixed effects (treatment and date) were significant. Splitting the treatment effect into a factor comparing the controls with all the other treatments, and a factor looking at differences among the phosphite treatments, the untreated control vs treatment effect and interaction with date was significant ($F = 13.6$ on 1 and 7 df, $p = 0.007$ for the main effect; $F = 13.9$ on 12 and 132 df, $p < 0.001$ for interaction with date). The differences among the various phosphite treatments were not significant ($F = 0.0$ on 3 and 10 df, $p = 0.989$ for the main effect; $F = 0.5$ on 36 and 172 df, $p = 0.991$ for the interaction with date).

Figure 11. Cracking and peeling of bark around margins of *Phytophthora agathidicida* lesions on kauri trunks. Top and bottom left: phosphite-treated trees, showing bark peeling around lesions, with healthy bark beneath. Bottom right: untreated tree, with bark peeling, but active *P. agathidicida* lesion on the new bark beneath.

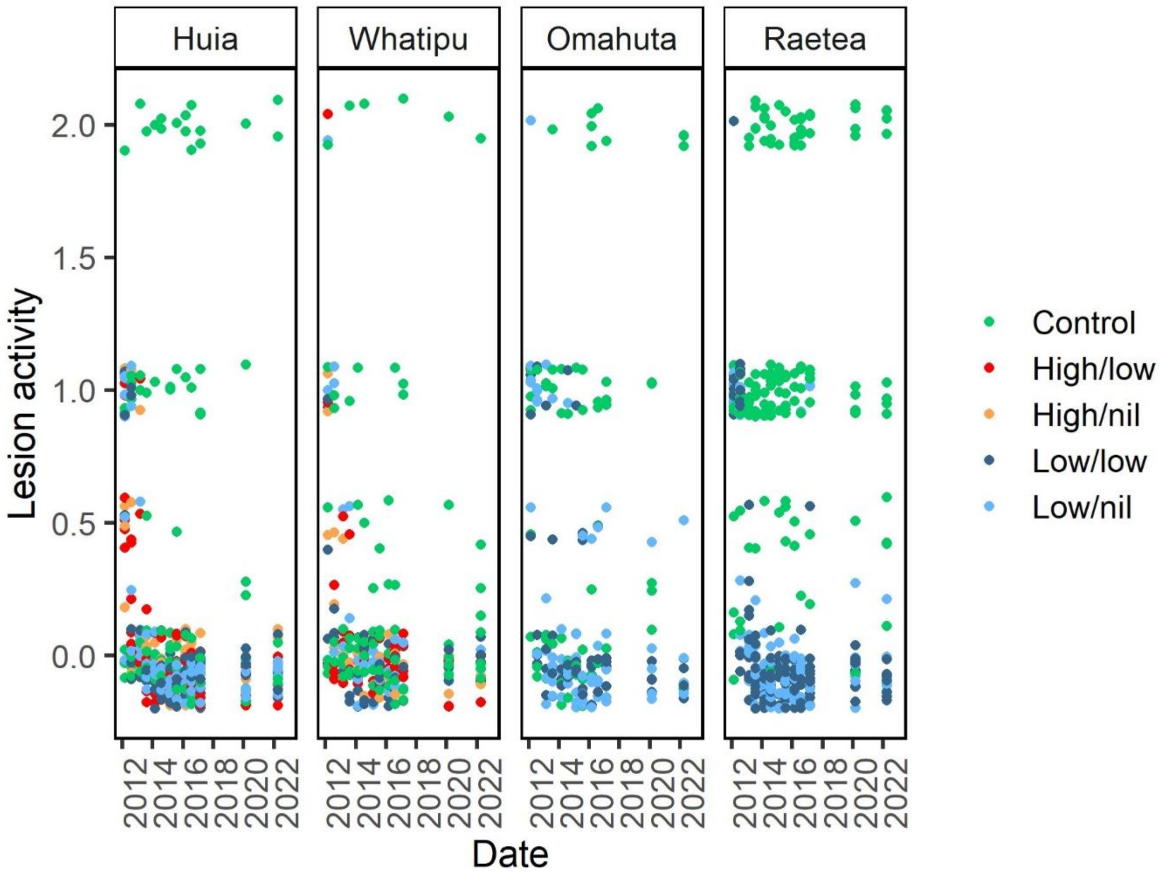


Figure 12. Lesion activity score in *Phytophthora agathidicida*-infected kauri trees on four sites assessed 6-monthly over 5 years following treatment with phosphite in early 2012, with further assessments in February 2020 and March 2022. Lesion activity was assessed as -0.1 = healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active. Data points are individual tree scores for all trees that had lesions, with the maximum score for each tree at each time-point plotted. Data points are plotted with a slight jitter to avoid overlaying. 'High' and 'Low' indicate the phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, nil = no treatment, Control = untreated.

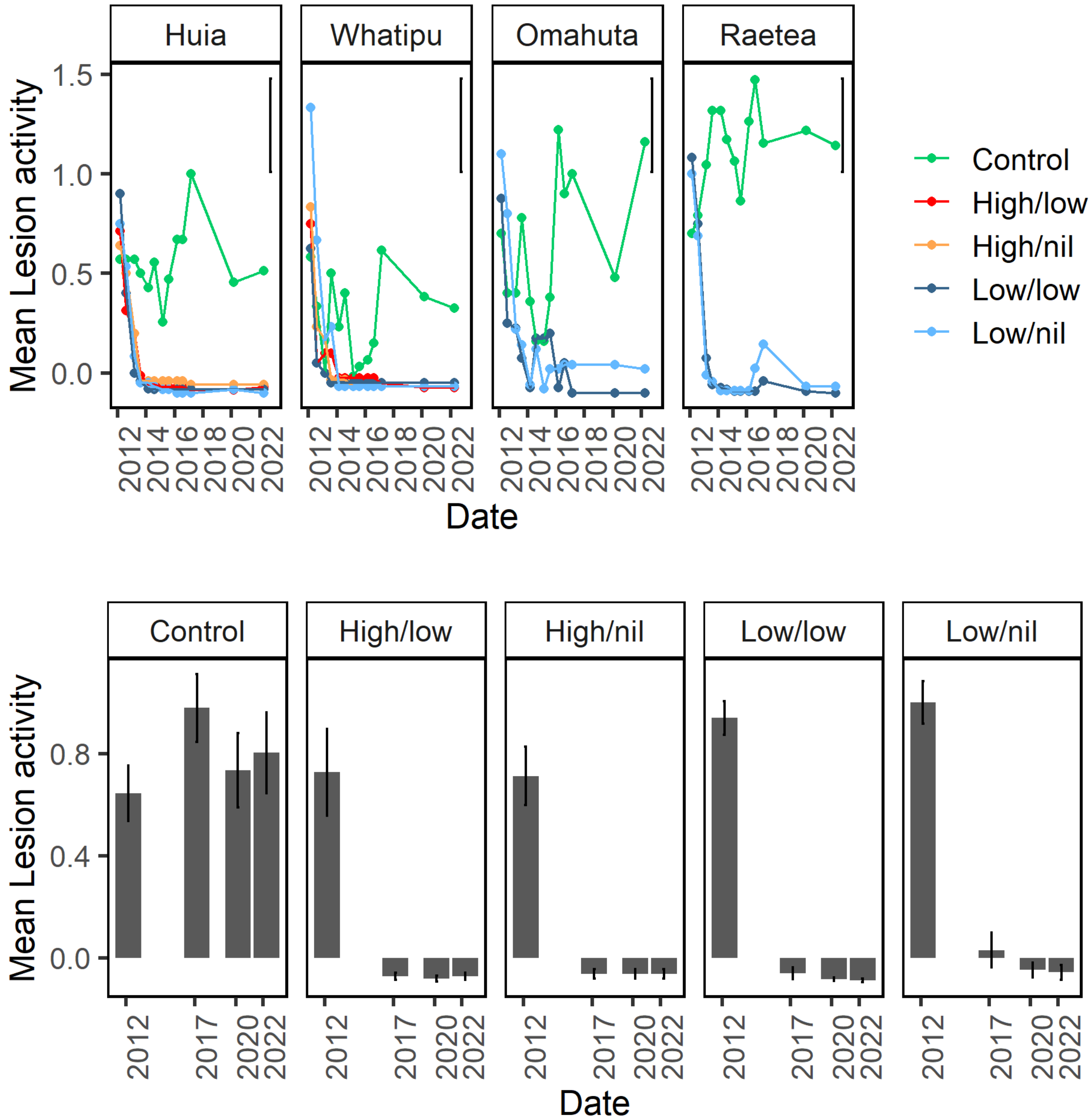


Figure 13. Mean basal trunk lesion activity score on *Phytophthora agathidicida*-infected kauri trees in four forest sites, following application of phosphite treatments in February 2012. Trees were assessed approximately every 6 months for the first 5 years, then re-assessed after 8 and 10 years (February 2020 and March 2022). Lesion activity was assessed as -0.1 = healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active. The bars in the top four (site) graphs indicate Least Significant Difference (LSD) (p = 0.05) between treatments in the final 2020 assessment. Lines on the lower graph (pooled data) indicate standard errors of the means. Lesion data used are for the most advanced/active lesion for any given tree. Only trees with above-ground trunk lesions were included in the analysis. The bar graph shows treatment means recorded pre-treatment and after 5, 8 and 10 years, averaged across all four sites. 'High' and 'Low' indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, nil = no treatment, Control = untreated.

3.2.2.2 Lesion expansion

Lesion expansion reflected the lesion activity scores, with continued extension of marked lesions in most control trees, and no or minimal lesion extension in treated trees (Figure 14). This was the case at all four sites. Within 3 or 4 years of treatment, mean lesion ‘expansion’ was trending ‘negative’ in most phosphite treatments. This reflected the lesion healing in all treated trees, with the peeling back of diseased bark revealing healthy bark beneath. After 5 years, the growth of marked lesions was significantly greater in untreated controls than in any of the phosphite injected treatments (F = 5.6 on 4 and 59 df, p < 0.001). There was no significant site effect (F = 0.8 on 3 and 59 df, *p* = 0.493) or site × treatment interaction (F = 0.8 on 8 and 59 df, *p* = 0.607). There were no significant differences among the various phosphite treatments. Marked lesion margin expansion could not be assessed after 8 years because of loss of the reference markings on bark. However, analysis of total lesion area showed the trend in bark healing (Figure 15), with a significant reduction in average lesion area in injection treatments on all sites.

In terms of lesion activity and expansion, the response to phosphite was similar in all the treatment regimes, i.e. high or low concentration, one application or two, on all four sites, i.e. there were no significant differences between any of the injection treatments. This contrasts to the significant differences from the untreated controls, already noted (Figures 13, 14 and 15).

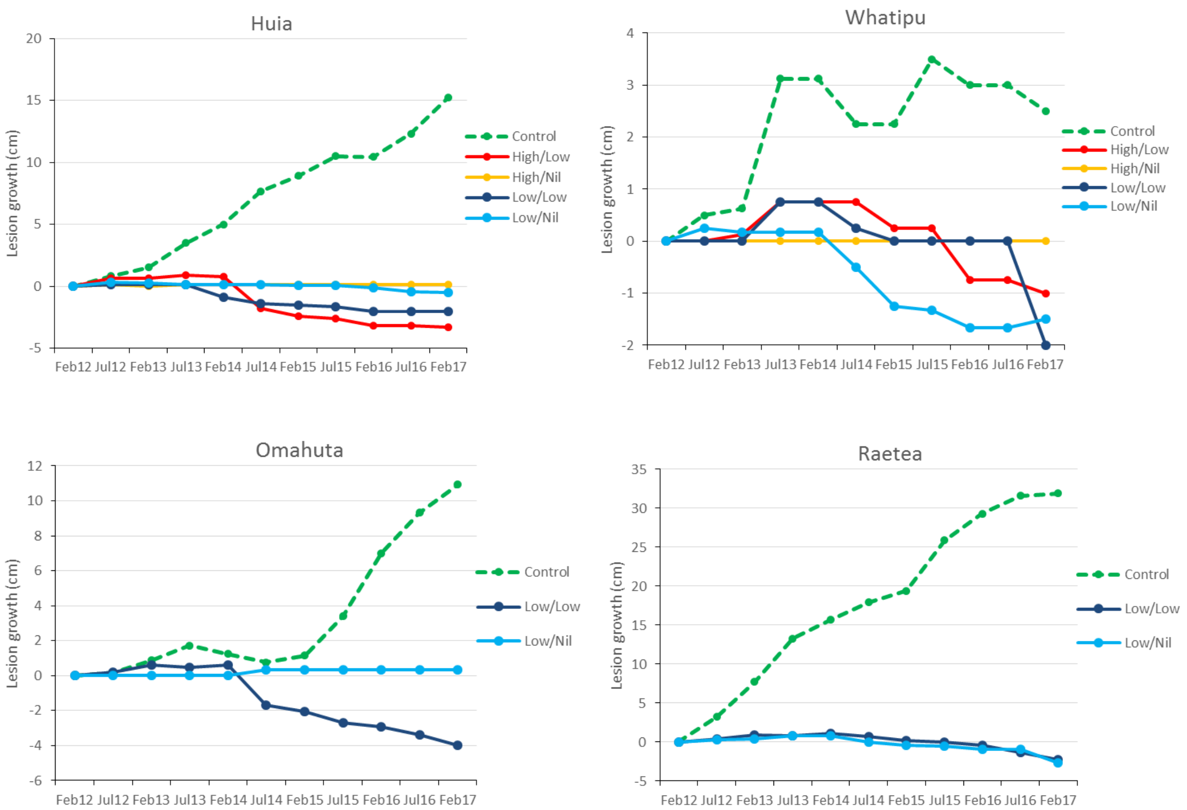


Figure 14. Mean basal trunk lesion advance on *Phytophthora agathidicida*-infected kauri trees in four forest sites. Vertical growth of tagged lesions was measured approximately every 6 months over a 5-year period, following application of phosphite treatments in February 2012. ‘High’ and ‘Low’ indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment in 2013, Control = untreated.

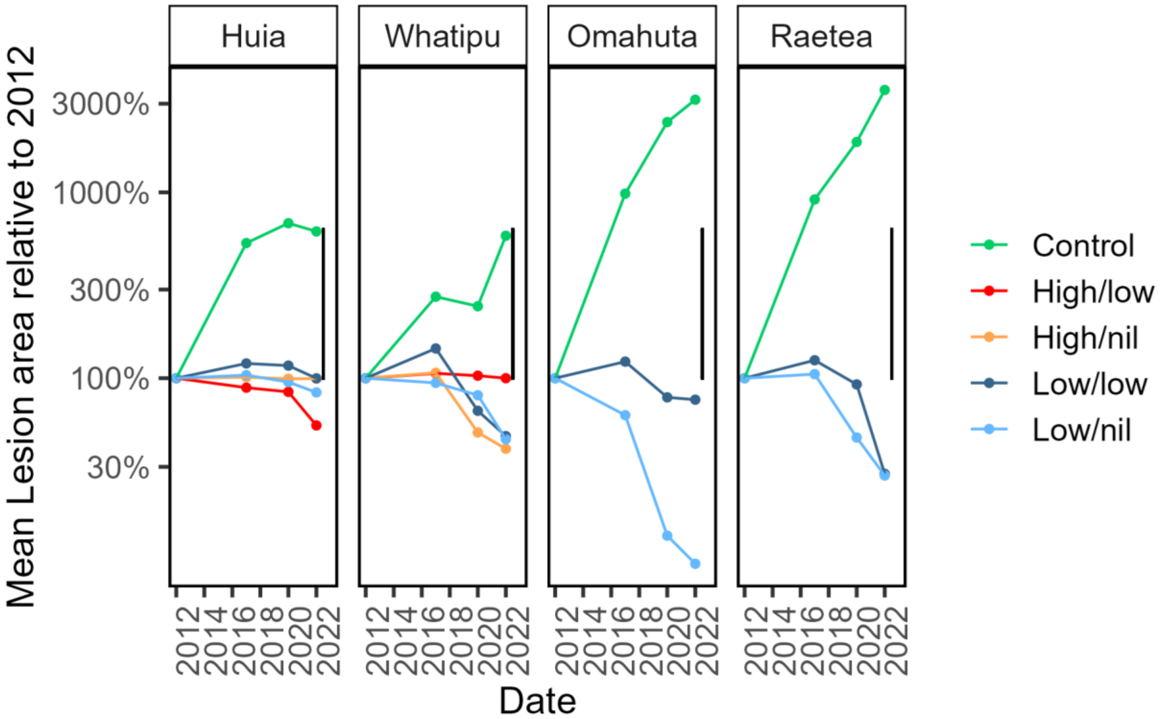


Figure 15. Mean basal trunk lesion area change on *Phytophthora agathidicida*-infected kauri trees in four forest sites, following application of phosphite treatments in February 2012. Approximate lesion area was calculated from lesion width and height assessments pre-treatment and after 5 and 8 years. In 2017 and 2020, lesion areas are expressed as percentages of the 2012 (pre-treatment) lesion area. '100%' indicates no change in lesion size, numbers less than 100% indicate a shrinkage of the lesion area and peeling back of diseased bark. 'High' and 'Low' indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment, Control = untreated. Bars indicate the least significant difference (95%) between treatment means.

3.2.3 Canopy health and shoot growth

The pre-treatment, 5-year, 8-year and 10-year canopy scores of all trees in the trial are shown in Figure 16. The change in canopy health can be most easily tracked in the canopy photograph comparisons at each 6-monthly assessment. The mean change for each treatment at each site at each assessment date is presented in Figure 17.

Fitting a linear mixed effects model to the photograph comparison ratings, with fixed effects for date and treatment, and random effects for site, tree, site × date and site × treatment, indicated there was a significant date effect (i.e. a change over time). The random effect for tree was large, indicating that a great deal of the variation in the data was due to differences in the initial tree health.

When we excluded trees which had died, and fitted a similar model, the date effect was significant (F = 7.4 on 11 and 106 df, $p < 0.001$), but there was also a significant date × treatment interaction (F = 2.0 on 44 and 104 df, $p = 0.002$). Data suggest the low dose treatment trees recovered to their initial canopy condition or improved, while the high dose and untreated control trees had stabilised or declined from their initial canopy condition. On average, there was a decline in canopy health in untreated control trees at all sites. This was most evident at Raetea, where every untreated tree declined in health (Figure 16).

The canopy health response to phosphite treatment varied at the different sites. At the Raetea and Omahuta sites, the canopy health of the majority of phosphite-treated trees either remained the same or improved slightly (Figure 16), although at Omahuta the “low/nil” treatment average (Figure 17) was heavily influenced by the fact that two of the five replicate trees died. Both these trees had advanced infections at the start of the trial. Apart from these two trees, most treated trees in the two Northland sites were showing good new shoot growth after 5, 8 and 10 years, and in many cases had begun to fill in canopies that had thinned earlier in the trial (Figure 28).

In contrast to the Northland sites, at Huia and Whatipū there was an initial decline in canopy health in the majority of trees treated with phosphite. This was despite lesion healing in all trees, as discussed above. This decline in canopy health was evident within a year of treatment application, and is assumed to be a phytotoxicity response. Symptoms were similar to those described in Section 2 above. There was leaf yellowing, browning and leaf drop in many trees, with twig abscission in some trees. This resulted in a thinning of the overall canopy, and a subsequent higher canopy score (Figure 16) or negative photograph comparison (Figure 17).

At Huia and Whatipū, by the 5-, 8- and 10-year assessments, most of the treated trees that survived were close to the canopy health score they exhibited at the start (Figure 16). This was not reflected in the mean photograph comparisons using the full data, which was heavily influenced by the trees that died (Figure 17). Removal of dead trees from the analysis demonstrated the partial recovery of some trees that initially suffered phytotoxicity symptoms (Figure 17, right hand graphs). This was more noticeable in trees treated with the low (7.5%) rather than the high (20%) phosphite concentration.

Shoot growth data showed similar trends to the canopy health and photograph comparison data, but give a little more insight (Figure 18). Trees that died biased the data, as obviously a dead tree records zero growth. When these were excluded (Figure 18 right hand graphs), a more reasonable interpretation of trends in growth could be made. At Huia, on average, remaining trees were growing well, but there was little difference in growth of treated and untreated trees. At the Whatipū site, with dead trees excluded there was a trend towards better growth in treated than in untreated trees. At the Raetea and Omahuta sites, there was significantly better shoot growth in trees injected with phosphite (low dose only) than in those left untreated (Figure 18).

Overall, canopy health scores did not necessarily correlate with or reflect lesion healing. It could be many years before lesion healing is reflected in substantial growth and canopy filling. But the 8- and 10 year assessments, especially at Omahuta and Raetea, were starting to show the long-term benefits of phosphite treatment, and the potential for addressing canopy decline and re-growing the canopy (Figure 28).

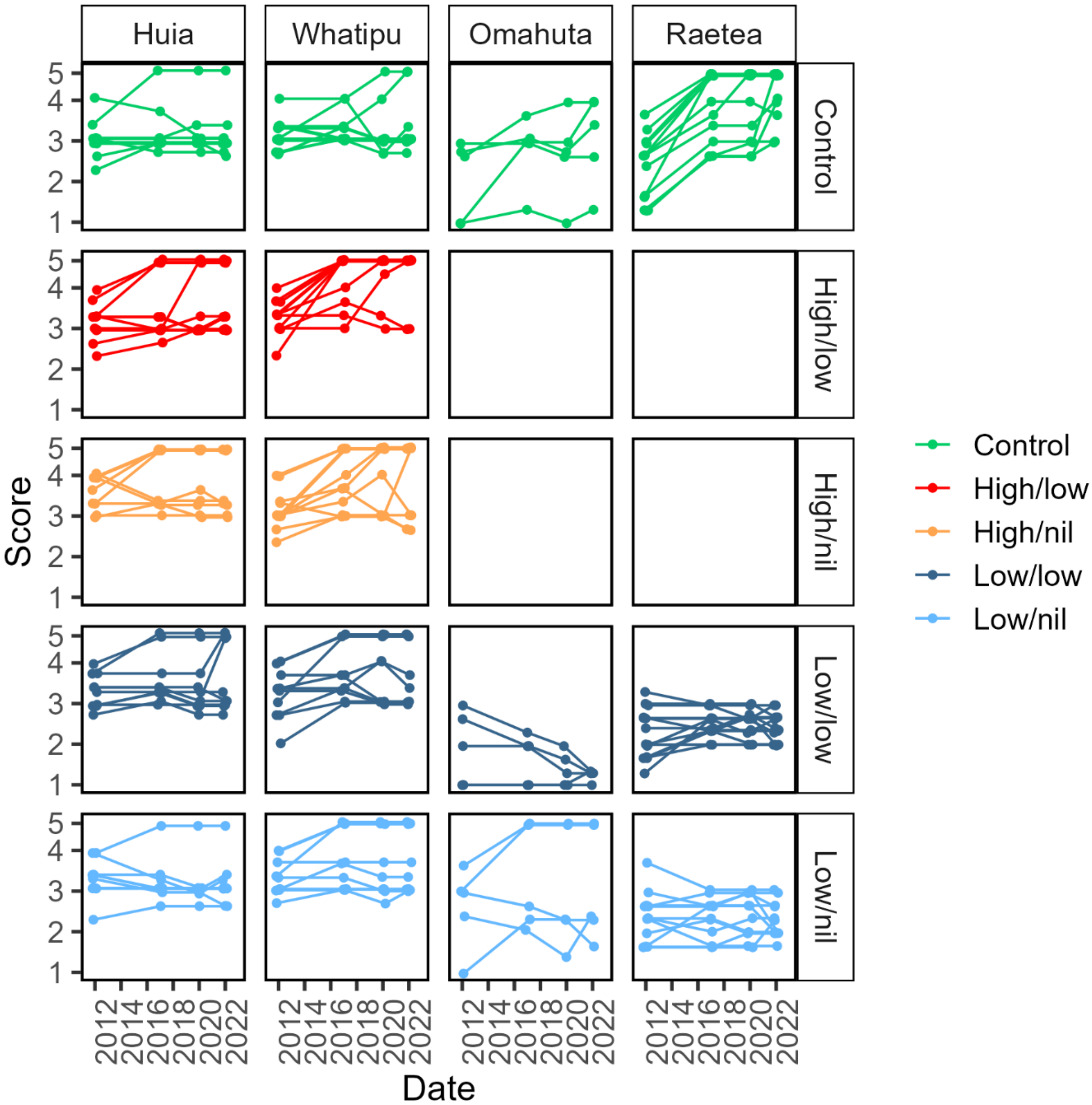


Figure 16. Kauri canopy health scores pre-treatment (February 2012), 5 years post-treatment (February 2017), 8 years post-treatment (February 2020), and 10 years post-treatment (March 2022) at four phosphite trial sites. Data points are individual trees and are plotted with a slight jitter to minimise overlap. Canopy scores: 1 = healthy, no signs of dieback, 2 = canopy thinning, 3 = thinning and some branch dieback, 4 = severe dieback, 5 = dead. 'High' and 'Low' indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment, Control = untreated.

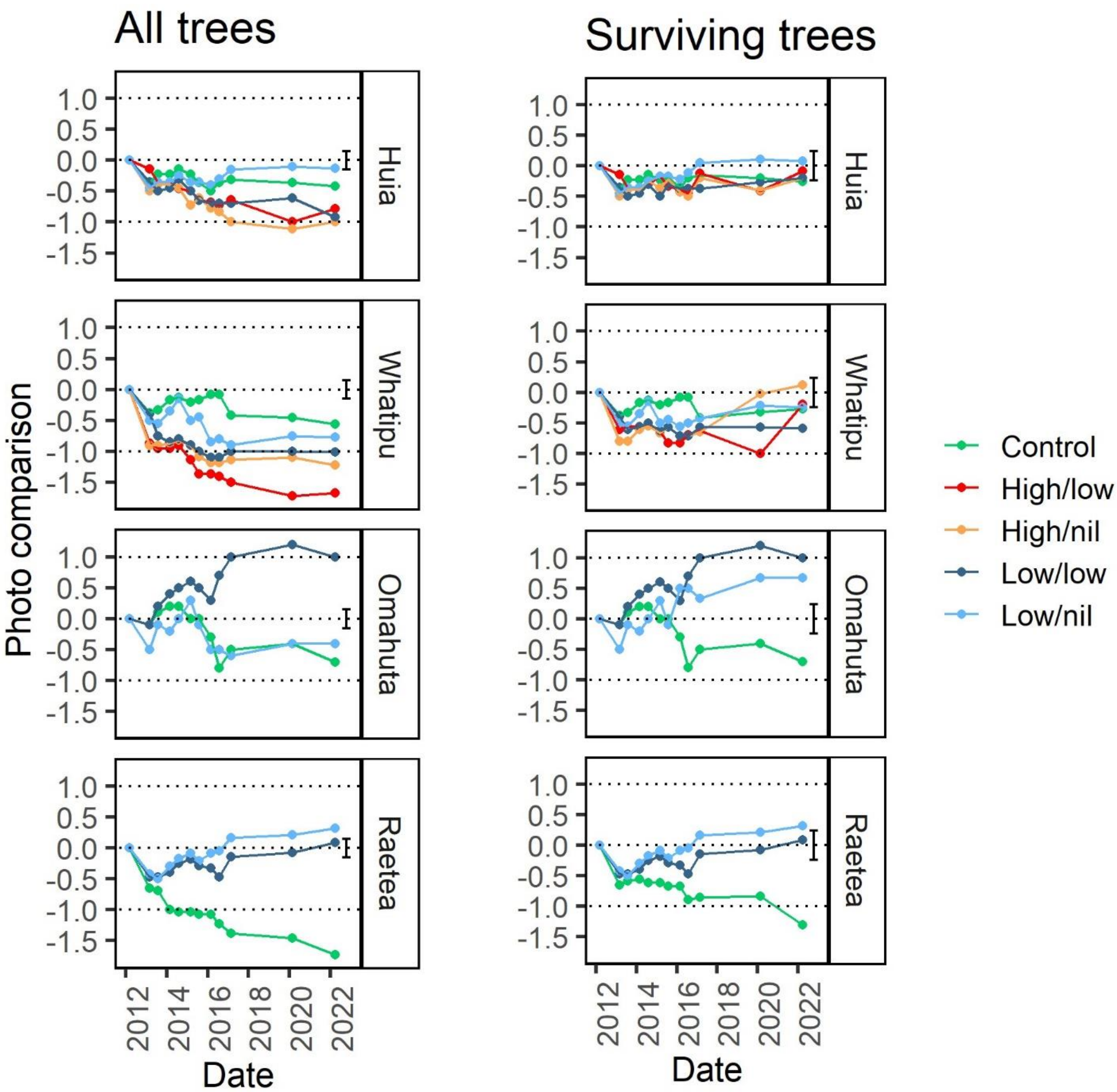


Figure 17. Comparison with baseline canopy photographs taken pre-treatment, in *Phytophthora agathidicida*-infected kauri trees in four forest sites. Every 6 months over a 5-year period (following application of phosphite treatments in February 2012), with further assessments after 8 and 10 years, comparisons of canopy density and health were made using the following scale: -2 = tree dead, -1 = substantially worse, -0.5 = slightly worse, 0 = similar, 0.5 = slightly better, 1 = substantially better. 'High' and 'Low' indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment, Control = untreated. Graphs on the left are mean scores of all trees, graphs on the right are mean scores after removing dead trees. Bars indicate Least Significant Difference (p<0.05) for comparisons between treatment means.

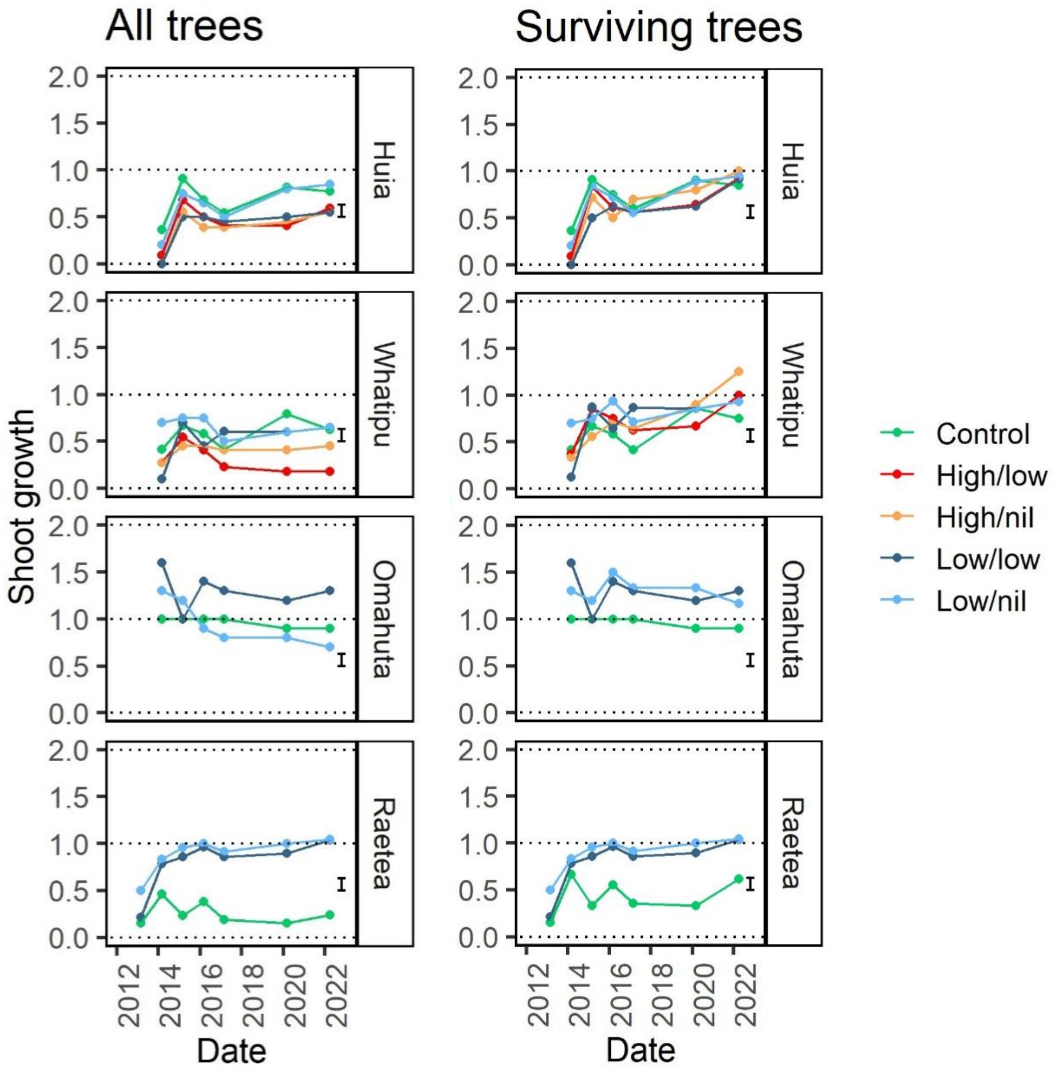


Figure 18. Mean shoot growth scores assessed in *Phytophthora agathidicida*-infected kauri trees in four forest sites. Every year over a 5-year period (following application of phosphite treatments in February 2012), with a final assessment after 8 years, shoot growth was assessed as 0 (no growth), 0.5 (slight growth), 1 (good growth) or 2 (vigorous growth). Data are means for each treatment at each site. 'High' and 'Low' indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment, Control = untreated. Graphs on the left are mean scores of all trees, graphs on the right are mean scores after removing dead trees. Bars indicate Least Significant Difference (p<0.05) for comparisons between treatment means.

3.2.4 Bark flaking and trunk cracking

Application of phosphite appeared to stimulate bark flaking in trees, particularly around *P. agathidicida* lesions and other injuries. This was apparent within a year of treatment application. After about 2 years, it was also noted that there was more prolific bark flaking in line with the injection points, often a metre or more above (but not below) the point (Figure 19). This appeared to be related to a slight swelling or more rapid growth of xylem or phloem in line with injections. This symptom was noticed at Huia, Whatipū and Raetea, but not Omahuta.

Just over 2 years after treatment, vertical bark cracking on the trunks was first noticed in some injected trees (even in trees that had only one treatment), usually in line with injection points. Again, this appeared to be related to a slight swelling or more rapid growth of trunk tissues in line with injections, and was often preceded by bark flaking in line. The cracking symptom was present only in trees at Huia and Whatipū, and not at either of the Northland sites. This cracking ranged from minor cracks and bleeds a few centimetres long, to sporadic deeper cracks and bleeds that could be 3 or 4 metres above the injection point (Figure 20). By the 5-year assessment (2017), the vast majority of these cracks had dried up and healed, but three trees at the Huia site still had active bleeds from cracks. Of these three trees, in the 2020 assessment, all bleeds had completely dried up and healed in one tree, with the remaining two trees still having active bleeds from cracks (although at a substantially lesser amount than previously). Apart from these two trees, there were no other active cracks or bleeds associated with injection points in any trial trees. By 2020, most signs of swelling or bark flaking in line with injection points were no longer obvious. A summary of the trunk symptom data up until 2017 is presented in Table 3 and Figure 21.

There is no obvious explanation for why many trees at Huia and Whatipū exhibited this cracking symptom, yet the trees in Raetea and Omahuta did not. Part of the reason could be the much higher phosphite concentration used in some treatments at Huia and Whatipū, but cracking was also noted in some trees in treatments of comparable lower dose (7.5%) to that applied in the two Northland sites.

The trunk cracking is not necessarily related to the disease status of the tree prior to injection. In the Huia rate pilot trial (Section 2.2 above), minor trunk cracks were observed in some trees that showed no sign of kauri dieback symptoms when injected. There was no obvious relationship of trunk cracking to measured pre-treatment parameters such as tree girth, canopy score or lesion activity, although these factors did correlate with mortality (Section 3.2.5 below).

The long-term effect of these cracks is yet to be determined. Although they generally healed, some of the severe cracks left wounds that could be a source of weakness or entry point for other pathogens.

Figure 19. Kauri bark flaking in line running up from injection points, 3 years after injection with a phosphite solution.

Figure 20. Moderate (left) and major (centre and right) kauri trunk cracks and bleeds in line with injection points, 2.5 to 3 years after trunk injection with a phosphite solution.

Table 3. Kauri trunk symptoms at four phosphite trial sites, 5 years after trunk injection with various phosphite treatments. ‘Bark flaking ‘in line’ and ‘bleeds’ or ‘cracks’ all refer to symptoms noted in line with the injection points. Data are the number of trees in each category, with the most severe trunk symptom noted for each tree. ‘High’ and ‘Low’ indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment.

Site	Treatment	Total tree number	Normal/ minor bark flaking	Bark flaking in line	Minor bleed or crack	Moderate bleed/crack	Major bleed/crack
Huia	Control	11	11				
	High/Low	11	2		8		1
	High/Nil	9	3		3	1	2
	Low/Low	10	2	1	4	2	1
	Low/Nil	10		4	2	1	3
Whatipū	Control	12	12				
	High/Low	11	5	4	1		1
	High/Nil	11	4	4	2	1	
	Low/Low	10	2	7		1	
	Low/Nil	10	1	4	5		
Omahuta	Control	5	5				
	Low/Low	5	5				
	Low/Nil	5	5				
Raetea	Control	14	14				
	Low/Low	14	9	5			
	Low/Nil	14	9	5			

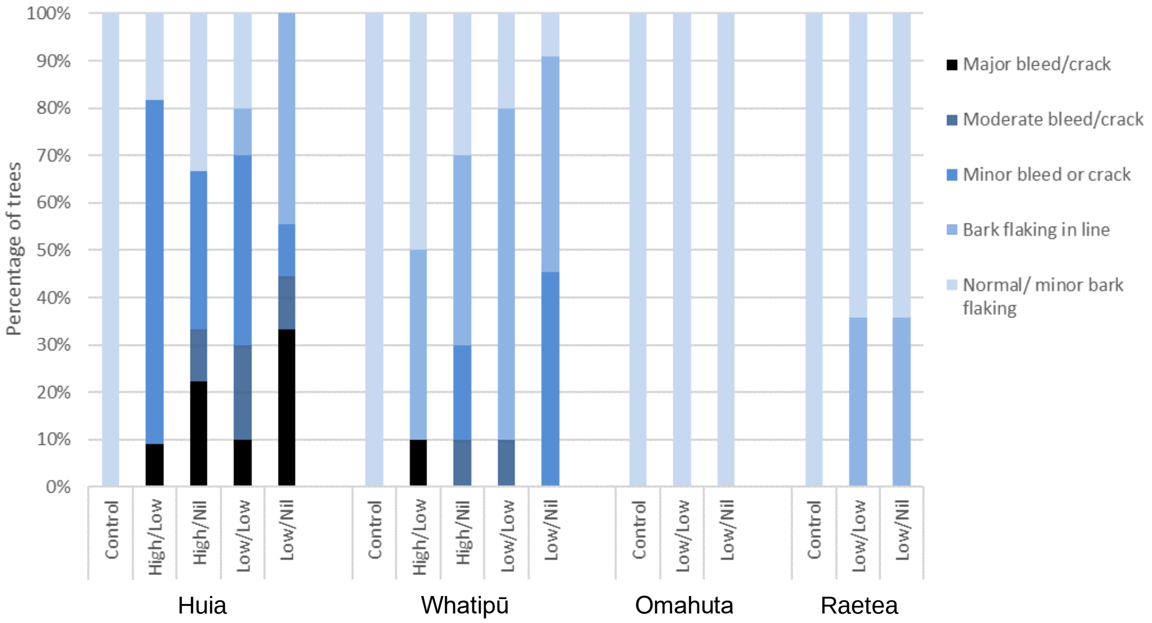


Figure 21. Trunk bark symptoms following phosphite injection treatments of *Phytophthora agathidicida*-infected kauri trees at four forest sites. Data are expressed as the proportion of trees in each trunk symptom category, with only the most severe symptom noted with 5 years on each tree recorded. ‘High’ and ‘Low’ indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment.

3.2.5 Tree mortality

The pattern of tree mortality and treatment responses varied at the different sites (Table 4 and Figure 22). This was supported by applying a binomial generalised linear model to the mortality data, showing in 2022 there were significant differences in mortality between sites (deviance = 9.3 on 3 df, $p = 0.026$) and between treatments (deviance = 15.2 on 4 df, $p = 0.004$), but also a very significant site × treatment interaction (deviance = 32.7 on 8 df, $p < 0.001$).

At Raetea, 10 years after the start of the trial, eight out of 14 of the untreated control trees had died, all six remaining control trees were showing a noticeable canopy decline, and five of these six had active basal bleeds. In contrast, none of the phosphite-injected trees died, and all appeared healthy at the end of the trial. In all the untreated control trees that died, death was preceded by rapid advance and expansion of basal trunk lesions.

At Omahuta, two trial trees died and both were in the ‘Low/nil’ treatment. Both these trees had substantial active lesions at the start of the trial and were showing canopy dieback symptoms.

At Huia, only one out of 11 untreated control trees died, yet 13 out of 40 treated trees died; the majority of these were treated with the higher (20%) phosphite concentration, or received two applications of the lower (7.5%) concentration. Only one tree at Huia died where a single application of the low (7.5%) concentration was used — a comparable result to the untreated control. At Whatipū there was a similar trend to that seen at Huia, with 22 out of 42 treated trees dying, with the highest mortality rate in trees treated with the high (20%) concentration of phosphite.

Testing each site individually, differences in mortality between treatments were significant at Whatipū (deviance = 14.1 on 4 df, $p = 0.007$) and Raetea (deviance = 22.3 on 2 df, $p < 0.001$). By testing the treatment effect, handling the site × treatment interaction as random noise, there was no significant difference between the treatments (F = 1.0 on 4 and 8 df, $p = 0.470$). This over-simplified analysis ignores the fact that we believe different factors are contributing to mortality at the different sites (discussed below).

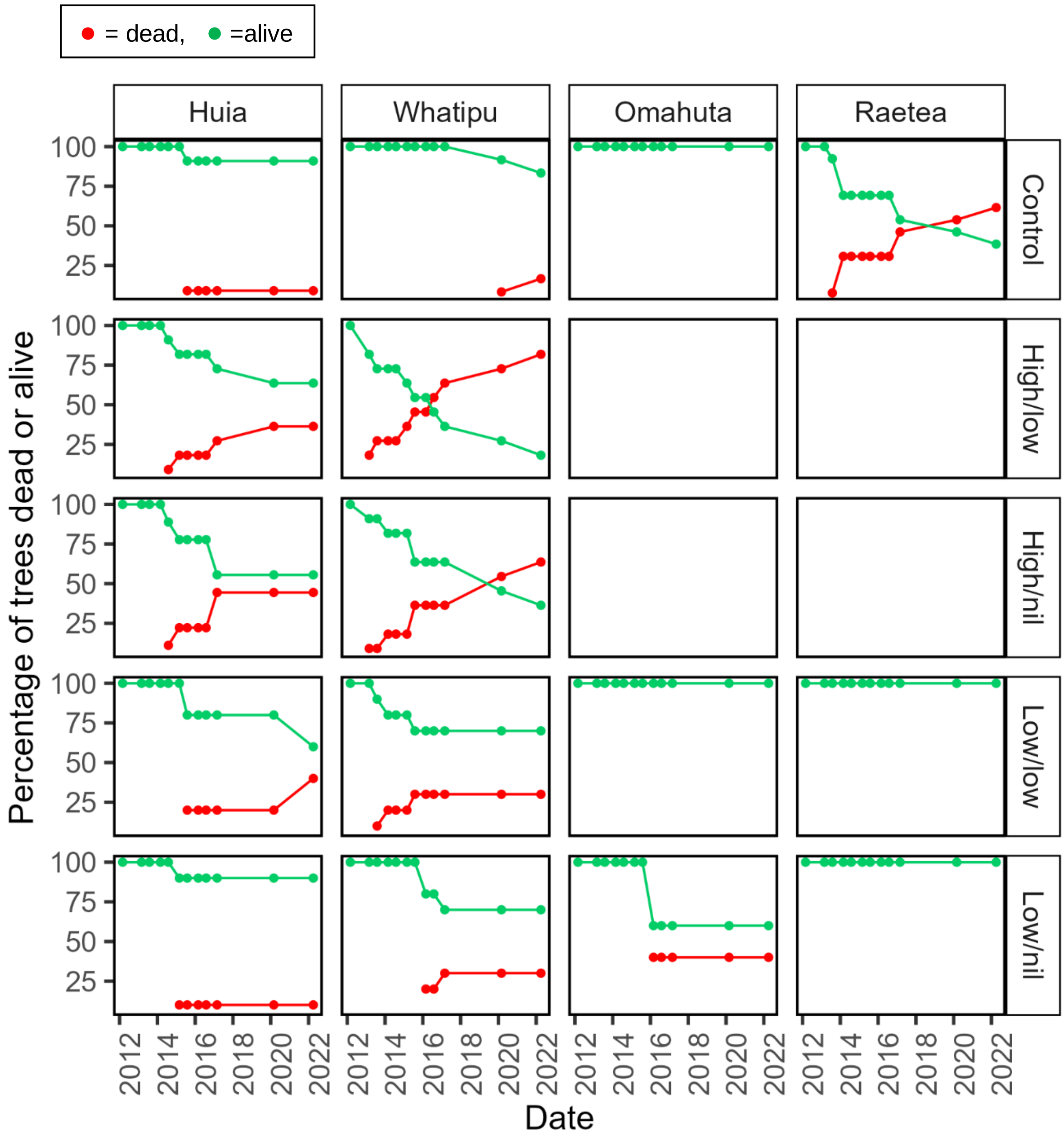


Figure 22. Proportion mortality of *Phytophthora agathidicida*-infected kauri trees on four sites, treated with various phosphite applications, assessed 6-monthly for 5 years following treatment in February 2012, with further assessments after 8 and 10 years (March 2022). Data are the proportion dead (red dots) or alive (green dots). 'High' and 'Low' indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment, Control = untreated control trees.

Table 4. Kauri tree mortality totals at each phosphite trial site, assessed in March 2022, 10 years after treatment application. Data are the number of dead/total trees treated. ‘High’ and ‘Low’ indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment, Control = untreated control trees. Letters represent statistical significance groupings (p = 0.05) within (but not between) sites. There were no significant differences in mortality between treatments at the Huia and Omahuta sites.

Treatment	Huia	Whatipū	Omahuta	Raetea
Control	1/11	2/12 a	0/5	8/14 b
High/Low	4/11	9/11 b	-	-
High/Nil	4/9	7/11 b	-	-
Low/Low	4/10	3/10 a	0/5	0/14 a
Low/Nil	1/10	3/10 a	2/5	0/14 a

Possible reasons for mortality

The causes of mortality appear to differ at the different sites. At Raetea, in most untreated control trees there was a rapid advance of *P. agathidicida* trunk lesions, in many cases resulting in ringbarking and tree death. After 5 years, six of the 14 untreated control trees had died, and the remaining eight trees were in decline. Ten years after the start of the trial, eight out of 14 of the untreated control trees had died, all six remaining control trees were showing a noticeable canopy decline, and five of the six remaining trees had active basal bleeds. In contrast, in all phosphite-treated trees, the lesions quickly stopped spreading and dried up, and all trees survived. At the end of 5 years all treated trees were looking healthy and showing signs of new growth. This trend continued, and at the 8- and 10-year assessments most treated trees were showing good canopy growth, and none had active lesions, either new or old. The phytotoxicity symptoms noted at other sites were relatively minor at Raetea. Thus, at the Raetea site it is reasonable to attribute tree deaths to advancing *P. agathidicida* infections and trunk girdling, and survival of treated trees to the protection and healing stimulated by phosphite.

At Omahuta, no phytotoxicity symptoms were noticed on any trees, yet two trees in the lowest phosphite regime died. Both these trees had substantial lesions and dieback symptoms at the start (pre-treatment) and died within 2–3 years.

At Huia and Whatipū, although all basal trunk lesions stopped spreading and dried up in all treated trees, the apparent phytotoxic effect of phosphite appeared to accelerate the decline of some trees. The higher mortality in the 20% phosphite treatment, particularly at Whatipū where the difference was statistically significant, suggests it is a direct dose response. There was also some mortality in the lower (7.5%) phosphite treatment, although numbers were not significantly different from the untreated control. This lower rate was also used in the Northland sites, without any noticeable adverse effects. There are no obvious reasons for this different response between sites, although there are many variables, including tree, soil, and climatic factors that may differ between the sites. In subsequent trials using the 7.5% concentration on kauri rickers in three sites (at Huia and Arapohue, Section 4 below), there was no mortality of treated trees, compared with 42% mortality of untreated trees).

To determine factors that might contribute to, or be associated with, the tree mortality in response to treatment, mortality was plotted against several variables. These included site, treatment, tree girth, initial lesion activity and starting canopy health (Figures 23 to 26).

Focusing on Huia and Whatipū where the apparent treatment-induced deaths occurred, most of the trees that died had active lesions at the start of the trial (p = 0.034, Figure 24). Trees with a more severe canopy disease score at the start of the trial had significantly (p = 0.001) higher mortality than trees with lower initial canopy scores (Figures 25 and 26). This is most easily seen in Figure 26, which plots mortality against girth and canopy score for the combined data. There is a grouping of deaths (red dots) in the lower right of the graph, i.e. the smallest girths and highest starting canopy disease scores. Earlier assessments suggested that smaller trees were more likely than larger trees to die following treatment, but there was not a significant trend in the 2022 assessments (p = 0.157).

The higher treatment-induced mortality in trees at more advanced stages of infection at the start of the trial suggests that sicker trees are less able to tolerate the phosphite treatment. This could simply be a factor of higher toxicity resulting from a higher concentration of the chemical in trees with a reduced canopy, perhaps accentuated in smaller trees with a reduced volume of wood. These factors will be discussed further in the General Discussion (Section 6.1.2).

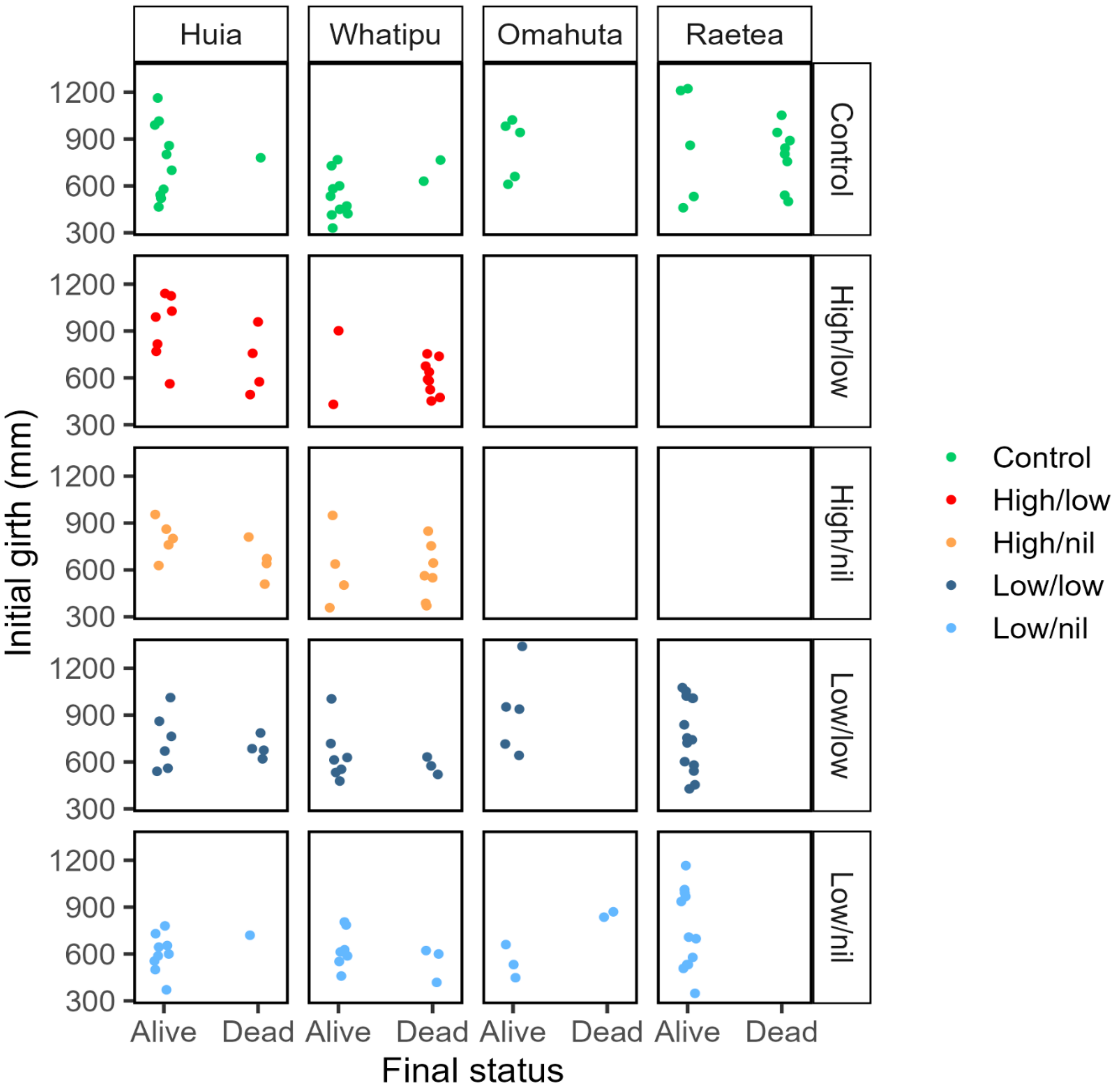


Figure 23. Relationship of trunk girth to mortality of *Phytophthora agathidicida*-infected kauri trees at four sites, assessed 10 years after treatment with various phosphite applications in February 2012/13. ‘High’ and ‘Low’ indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment.

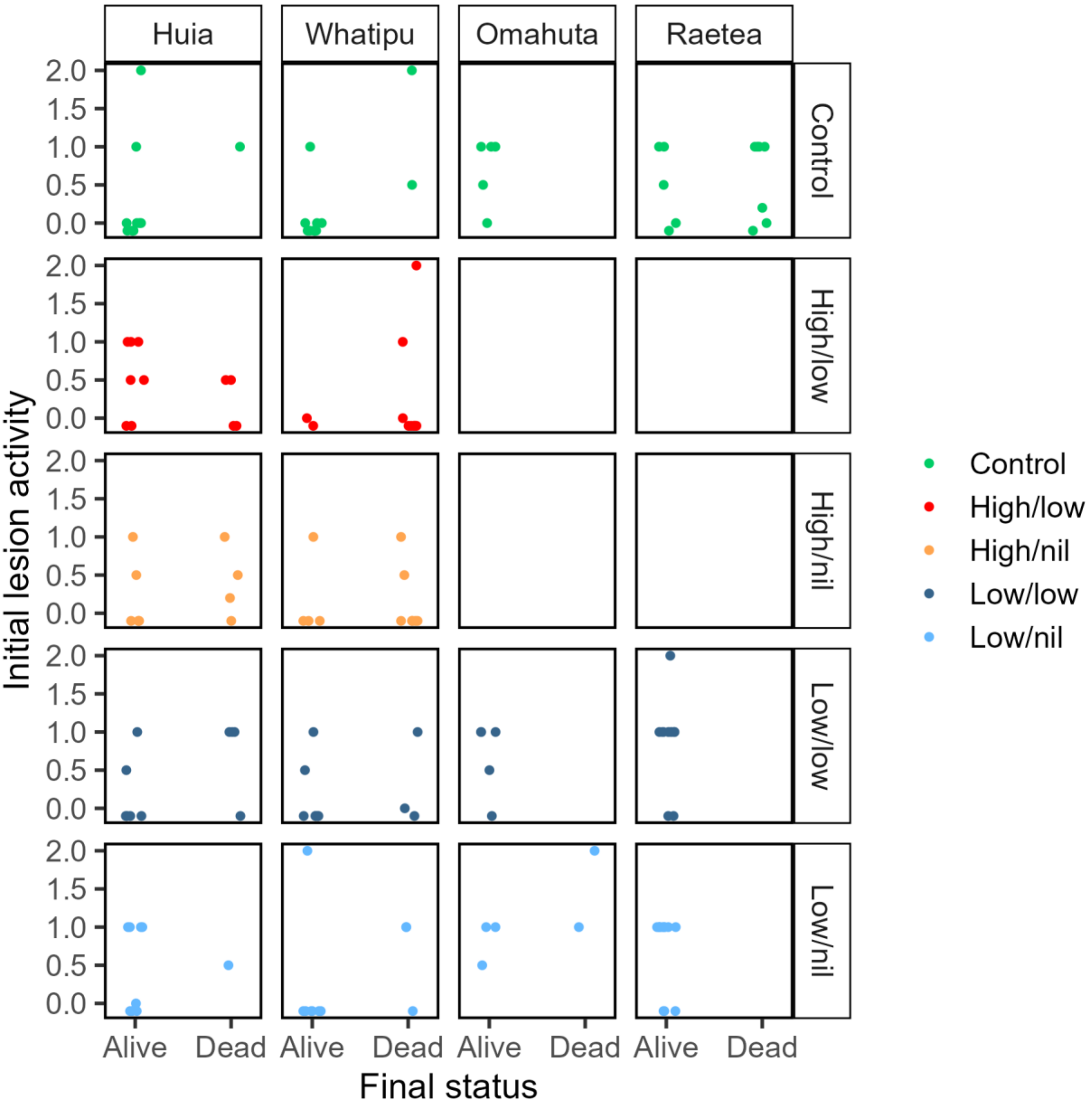


Figure 24. Relationship of initial lesion activity to subsequent mortality of *Phytophthora agathidicida*-infected kauri trees at four sites, assessed 10 years after treatment with various phosphite applications in February 2012/13. 'High' and 'Low' indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment. Lesion activity scoring: -0.1 = no lesion, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active.

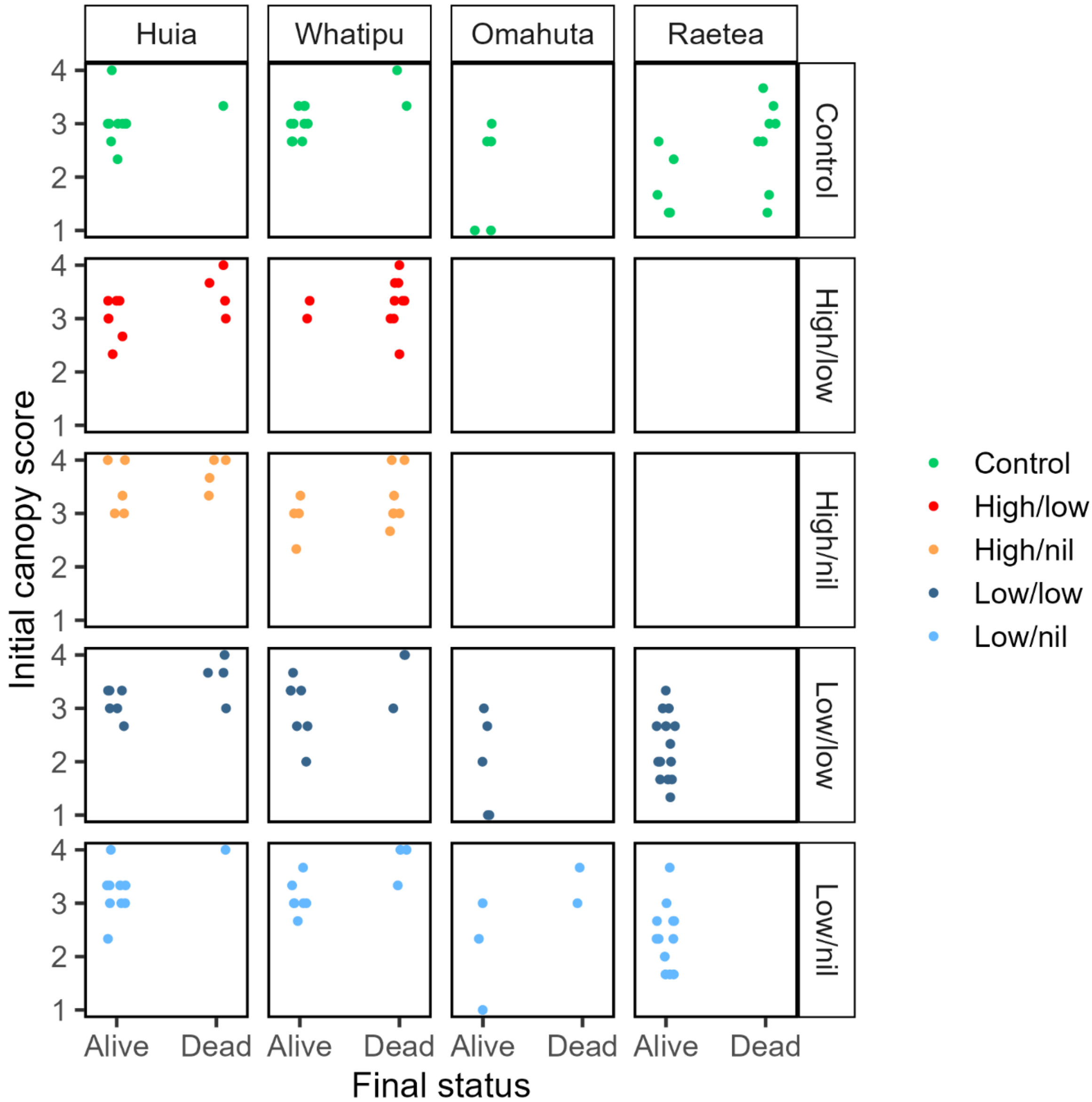


Figure 25. Relationship of pre-treatment canopy score to mortality of *Phytophthora agathidicida*-infected kauri trees at four sites, assessed 10 years after treatment with various phosphite applications in February 2012/13. Canopy was scored on a scale where 1 = healthy, 2 = canopy thinning, 3 = thinning and some branch dieback, 4 = severe dieback, 5 = dead. 'High' and 'Low' indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment.

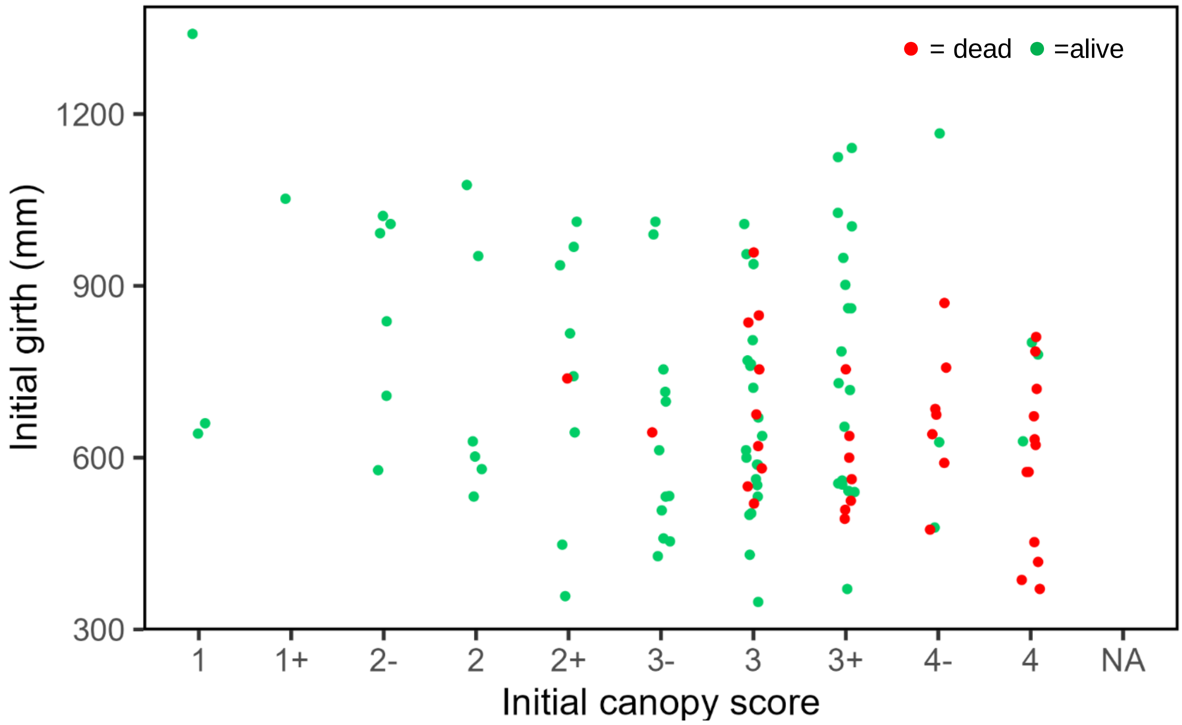


Figure 26. Mortality or survival of *Phytophthora agathidicida*-infected kauri trees in relation to initial trunk girth or canopy score, recorded 10 years after injection with phosphite in 2012. Data are for all phosphite treatments over four trial sites, with untreated control trees removed. Each datum represents a single tree. Green dot = alive, red dot = dead in 2022. Canopy scores: 1 = healthy, no signs of dieback, 2 = canopy thinning, 3 = thinning and some branch dieback, 4 = severe dieback, 5 = dead.

3.2.6 Miscellaneous observations

Raetea site

The results at the Raetea site warrant special mention and indicate the potential of phosphite as a control method. Lesions have dried up and healed on all phosphite-treated trees, and all these trees now appear relatively healthy and are putting on new growth. This includes a couple of trees deemed to be almost girdled and near death at the start of the trial (Figure 28). At this site, *P. agathidicida* lesions seemed particularly aggressive, as reflected in lesion growth in untreated trees (Figure 14, Figure 27). Most untreated trees declined rapidly, a majority died and those still alive continue to decline. Within the same stands as the trial trees, there were also a number of trees that were not included in the trial because they did not have basal trunk lesions or other symptoms when the trial was established. Ten years later, many of these trees now have substantial active lesions, and others are already dead. Within this site, with such aggressive disease progression, the positive response to phosphite seems greatest.

Root grafting

Root grafting among adjacent kauri trees has been previously recorded (Hillary 1944; I.L. Barton pers. comm. in Ecroyd 1982; Bader & Leuzinger 2019), and there is evidence of it occurring in our current trial. At the Huia and Whatipū sites in particular, trial stands were of quite closely spaced ricker trees. Many untreated control trees were within 3–5 m of injected trees and could potentially have been linked to these trees via root grafting. Evidence of this was seen in one instance at Whatipū, where an untreated tree was within 2 m of a tree injected with 20% phosphite. Within a few months of treatment, the injected tree canopy showed substantial leaf yellowing and leaf thinning, presumably a phytotoxic response. The adjacent untreated tree also showed leaf yellowing and leaf thinning, but not as extreme as the injected tree, suggesting that phosphite may have transferred between the trees. Both trees subsequently recovered. Such connections could have partially compromised the trial if untreated control trees acquired a small dose of phosphite. However, the difference in lesion growth in treated and untreated trees suggests this was not a major factor.

Pigs

Pig damage was an issue at the Raetea site throughout the duration of the trial. It appeared that pigs targeted infected trees, gouging at lesions with their tusks, rooting around the base and in some cases creating wallows next to infected trees (Figure 29). There can be little doubt that such activity would spread the pathogen, but it also further compromises trees already suffering from pathogen attack.

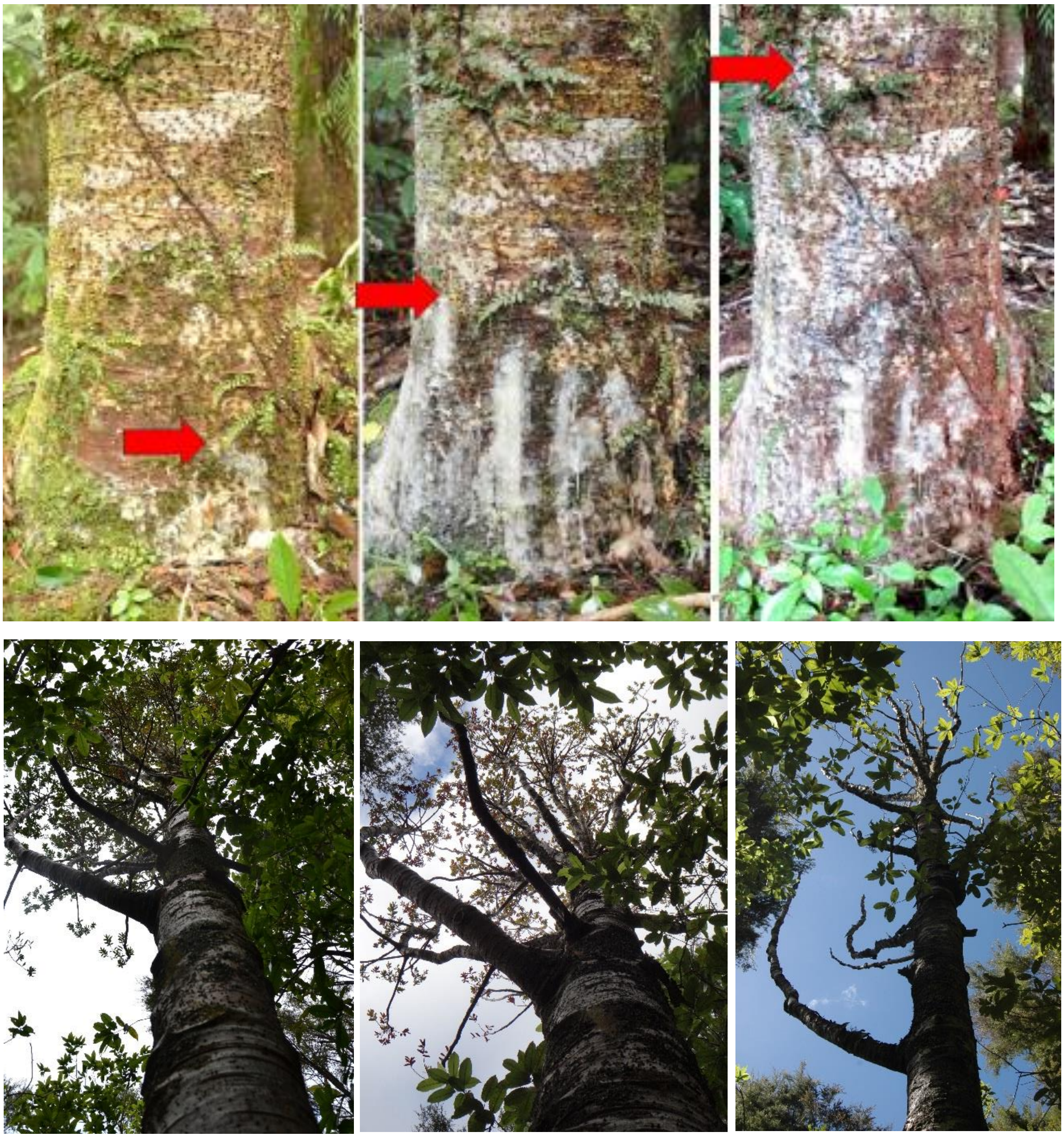


Figure 27. Top: rapid advance of a *Phytophthora agathidicida* lesion on an untreated control kauri tree in the Raetea trial site. Top left, March 2012; top centre June 2013; right January 2014. The arrows indicate the upper extent of the lesion. Bottom: canopy decline on the same tree, left, March 2012; centre, January 2014; right, February 2017.

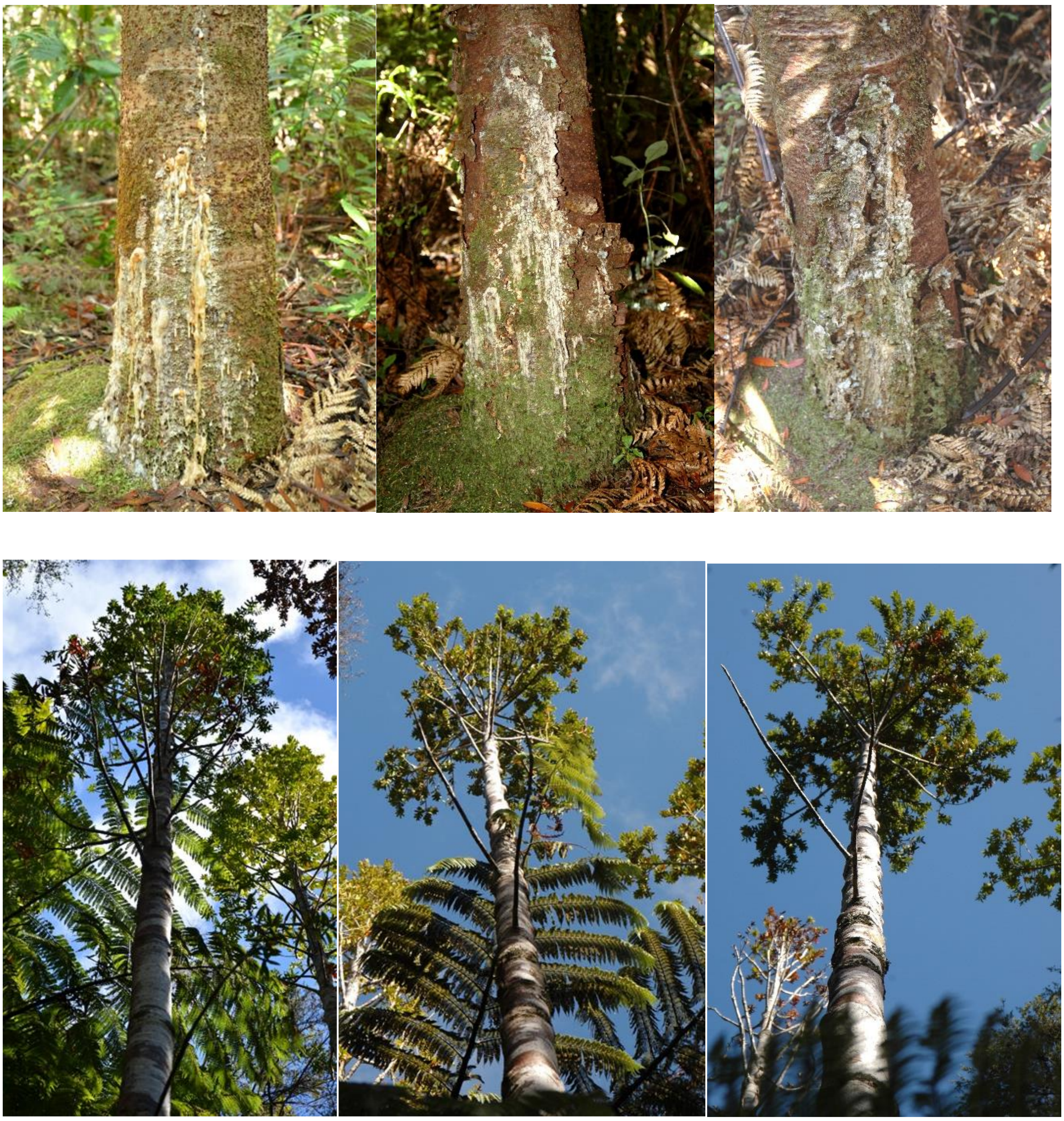


Figure 28. Top: Healing of a *Phytophthora agathidicida* lesion on a kauri tree injected with 7.5% phosphite in the Raetea trial site. Left, March 2012 (on the day of injection), showing a very active bleeding lesion (there was a similar lesion on the other side of the trunk); centre, February 2017, showing a dried up lesion and peeling of bark with healthy bark beneath; right, February 2020, continued lesion healing and peeling. Bottom: Canopy of the same tree on the same dates. Note the initial decline in canopy density, but very good new shoot growth and denser canopy by 2020. Note also the declining canopy of an untreated tree in the background to the left.

Figure 29. Left: Pig wallow at the base of a *Phytophthora agathidicida*-infected kauri tree at the Raetea trial site. Right: trunk damage from pig tusks, and mud rubbed onto the trunk of a nearby infected tree.

4 Low rate and trunk spray ricker trial 2016

The ricker trials outlined in Section 3 above provided very promising results, showing cessation of lesion expansion in treated trees and evidence for excellent control of *Phytophthora agathidicida* within trees (Horner et al. 2015). However, there were also some detrimental effects, with foliar phytotoxicity in some treated trees. In addition, there were some trunk symptoms, such as cracking, which appeared to be associated with injection points. Application concentrations in the early trials were probably too high (20% and 7.5% phosphite), and this may have contributed to the observed phytotoxicity, particularly on trees with advanced kauri dieback symptoms.

The ‘Low rate and trunk spray ricker trial’ was established to investigate the efficacy of lower concentrations and doses of phosphite, to determine if phytotoxicity symptoms could be reduced while still providing adequate disease control. In addition, trunk sprays were also included to determine if topical application and absorption through the bark could provide disease control while avoiding invasive injection. Such treatments have been tried with other species (such as apple, avocado and oak) and, while not always as effective as trunk injection, still had a positive effect on *Phytophthora* spp. control (Garbelotto et al. 2007).

4.1 Methods

4.1.1 Trial site and tree selection

The trials were established on three sites where *P. agathidicida* had been confirmed by soil testing: Huia Dam (Waitākere Ranges) — adjacent to the previous long-term ricker trial (Figure 4), and two farm blocks at Arapohue, near Dargaville (Figure 30). Trees in the trial were mostly at the advanced ricker and early mature stage, ranging in size from 20–70 cm trunk diameter (Figure 31). All trial trees showed symptoms of kauri dieback at the start of the trial, including basal trunk lesions. Canopy scores ranged from 1 to 4 (Figure 32). The trials were established in March 2016, and continued for 6 years.

Figure 30. Arapohue1 trial site for testing low-rate phosphite injection and trunk sprays for control of *Phytophthora agathidicida* in kauri.

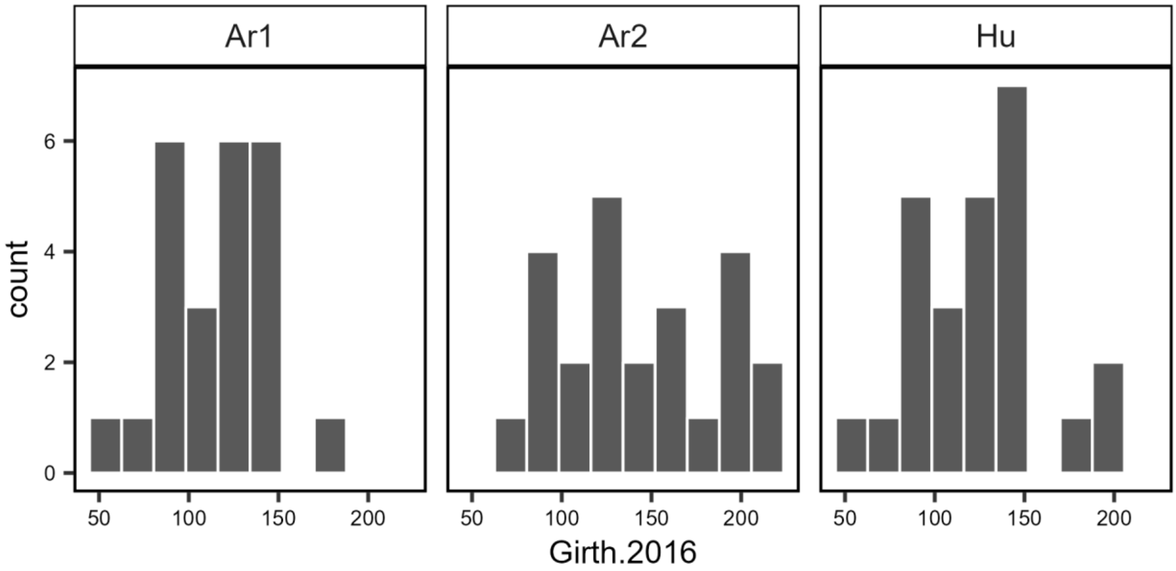


Figure 31. Range of kauri tree girths (cm) across three forest sites at the start of the 'low rate and trunk spray ricker' phosphite treatment trial. Ar1, Ar2 and Hu refer to the Arapohue 1 & 2 and Huia sites, respectively. 'Count' = number of trees in each size class.

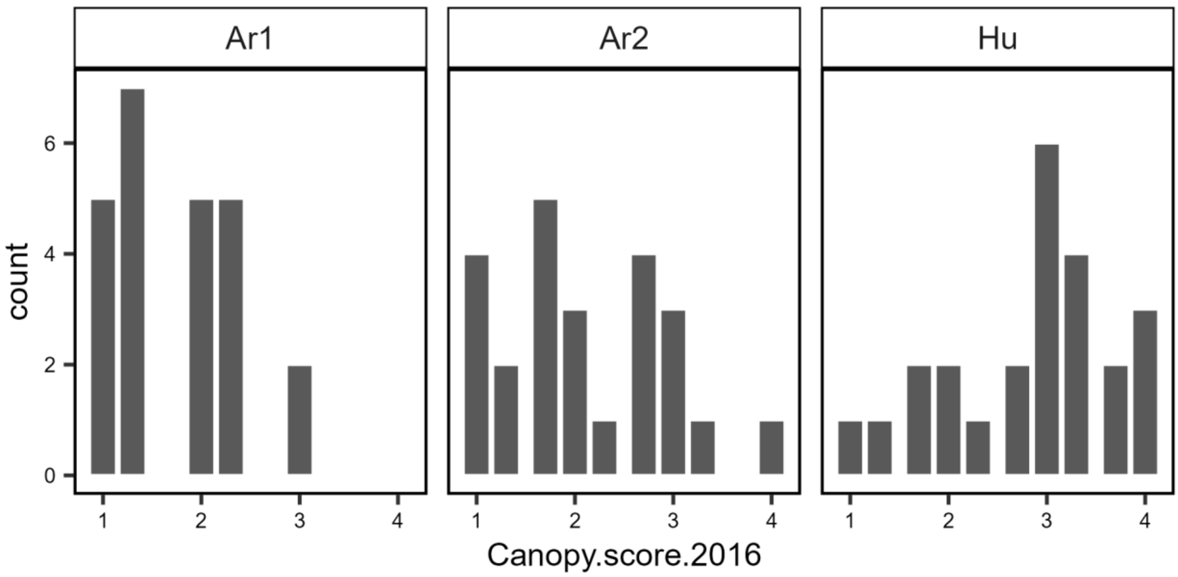


Figure 32. Range of kauri tree canopy scores across three forest sites at the start of the 'low rate and trunk spray ricker' phosphite treatment trial. Scores were on a 1–5 scale where 1=healthy and 5=dead (see Section 3.1.2). Ar1, Ar2 and Hu refer to the Arapohue 1 & 2 and Huia sites, respectively. 'Count' = number of trees in each health score category.

4.1.2 Pre-treatment assessments and trial design

Before treatment selection, baseline assessments were made on various tree growth and health parameters. These included tree girth, canopy health score, canopy colour and shoot growth, plus trunk lesion size and activity, using methods outlined in Section 3.1.2 above. Selected basal trunk lesion margins were marked for subsequent measurement of expansion, and canopy photographs were taken for later comparison. At each site, potential trial trees were assigned into groups based on similarity of disease parameters such as lesion activity and canopy symptoms, then within each grouping, trees were randomly assigned to the various treatments. This ensured a relatively even and unbiased distribution of disease symptoms across treatments.

There were a total of 72 trees, 24 on each site. The trial was evenly balanced, with four replicate trees of each treatment on each site.

4.1.3 Treatments selection and application

The rationale of treatment selection for this trial was to include the lowest concentration from previous trials (7.5%) as the ‘high’ injection rate for this trial, to include injections with a lower phosphite concentration (4%), plus the 4% concentration at a lower dose (i.e. one 20-mL injection every 40 cm around the trunk, rather than every 20 cm). The trunk sprays were included to test this application method, with or without the bark penetrant recommended by the phosphite suppliers. Thus, treatments were:

1. Untreated control;
2. 7.5% phosphite trunk injection, 20 mL every 20 cm;
3. 4% phosphite trunk injection, 20 mL every 20 cm;
4. 4% phosphite trunk injection, 20 mL every 40 cm;
5. 10% phosphite trunk spray with bark penetrant (Pentrabark™);
6. 10% phosphite trunk spray without bark penetrant.

Agrifos600® (Key Industries) was the phosphite formulation used for all applications. All treatments were applied in March 2016. Injection followed the method outlined in 3.1.3 above, except that drill-holes were not filled with silicon sealant following injection. Trunk sprays were applied to the lower 2 m of the trunk, using a hand mister. Volumes were carefully measured, so that equivalent total volumes of phosphite were applied in injection and spray treatments (based on trunk girth). Trunk spray treatments were re-applied in March 2018, February 2020, and March 2022, but all other trees were left untreated at these times. In the March 2018 re-spray application, Pentrabark was again used as the penetrant in Treatment 5. For the 2020 and 2022 trunk applications this was changed to Infiltrate Bark Penetrant™ (Renovo Technologies), following a recommendation from Key Industries.

4.1.4 Post-treatment assessments

Approximately every 6 months for 3 years, tree health plus lesion expansion and activity were measured, with assessments in August 2016, February/March 2017, August 2017, February/March 2018, October 2018 and February/March 2019. Further assessments were made in February 2020, and March/April 2022, 2 and 4 years after the initial treatment, respectively.

Shoot growth was recorded in the summer assessments only, on a 0–2 scale, where 0 = no growth, 0.5 = some, but poor growth, 1 = good (‘normal’) growth and 2 = vigorous growth. The overall canopy health was scored on a modification of the 1–5 canopy health scoring scale adopted by the KDP (see section 3.1.2). At each 6-monthly assessment, the state of the canopy viewed from a fixed photo point was compared to a photo of the canopy taken pre-treatment in 2016 (see 3.1.4).

Basal trunk lesions were measured (maximum width and height), their orientation (side of tree) noted, and a judgement was made on the activity of the lesion using the scale described in Section 3.1.4.

Selected points on some of the original lesions were marked in 2016, using a paint-pen to mark the lesion boundary and reference points 10 and 20 cm beyond. Any change in the lesion boundary was measured at each 6-monthly assessment, and lesions were re-marked if necessary. In some cases, lesion healing and bark peeling (with healthy bark below) occurred, in which case the lesion advance was recorded as a negative number.

Signs of phytotoxicity in the canopy (e.g. leaf yellowing/twig drop), and trunk cracks or bleeds associated with injection points (as observed in earlier trials) were noted at each assessment.

4.2 Results and discussion

4.2.1 Injection point healing

In contrast to earlier trials, injection holes were not sealed with silicon in this trial. Observations showed that trees usually naturally plugged the hole with resin within an hour or so of injection (Figure 9), and the holes generally healed very well.

4.2.2 Lesion activity and expansion

At the start of the trial (February 2016), lesion activity was similar across all treatments (Figures 33 and 34), reflecting the balanced way in which treatments were assigned. In untreated control trees, average lesion activity dropped slightly initially, but overall did not change significantly throughout the 6-year assessment period, with many active lesions remaining. In contrast there was a significant decline in lesion activity across all trunk injection treatments, first noted at the first 6-monthly assessment (Figure 33), and the trend continued throughout the trial (Figure 35). This pattern of reduced lesion activity in injected trees was reflected in the lesion advance measurements, with minimal lesion growth and, within a year or two, a decline in lesion advance measurements (Figure 36) and lesion area (Figure 37). This ‘negative’ lesion growth reflected lesion healing and peeling in many trees. Even the lowest concentration of 4% at the reduced dose of one injector every 40 cm (instead of the standard 20 cm) appeared to provide control. It should be noted that in some untreated control trees there were also observations of a few individual lesions stopping growth, then healing and peeling back. However, on average, untreated tree lesion growth continued throughout the 6-year duration of the trial. The apparent tailing off of lesion advance in untreated control trees in year 4 (Figure 36) reflects the death of some trees, with no subsequent expansion of their lesions.

There is no clear discrimination between the various injection treatments, with no significant difference between them in terms of lesion activity or expansion. It must be noted that there was still a low amount of activity (or probable activity) noted in some lesions on some injected trees after 4 years, and this increased after 6 years, but the amount of activity was substantially lower than in untreated trees (Figures 34 & 35, Table 5).

After one application, the trunk spray treatment *without* Pentrabark provided some control, with a reduction in average lesion activity. There was a dramatic peeling of bark where the trees had been sprayed, which helped with this healing (Figure 42). However, lesion healing was inconsistent, with lesions on some trees remaining active and spreading. In February/March 2018, 2 years after the initial application, trunk sprays were re-applied to trees. Six months later, this second spray appeared to have helped heal most lesions, with average lesion activity scores in spray treatments lower than those observed before the second treatment, and lesion expansion almost halted (Figures 33, 36 and 37). Results across the 6 years of the trial suggest that regular (2-yearly) trunk-spray application (without penetrant) may help suppress trunk lesion expansion.

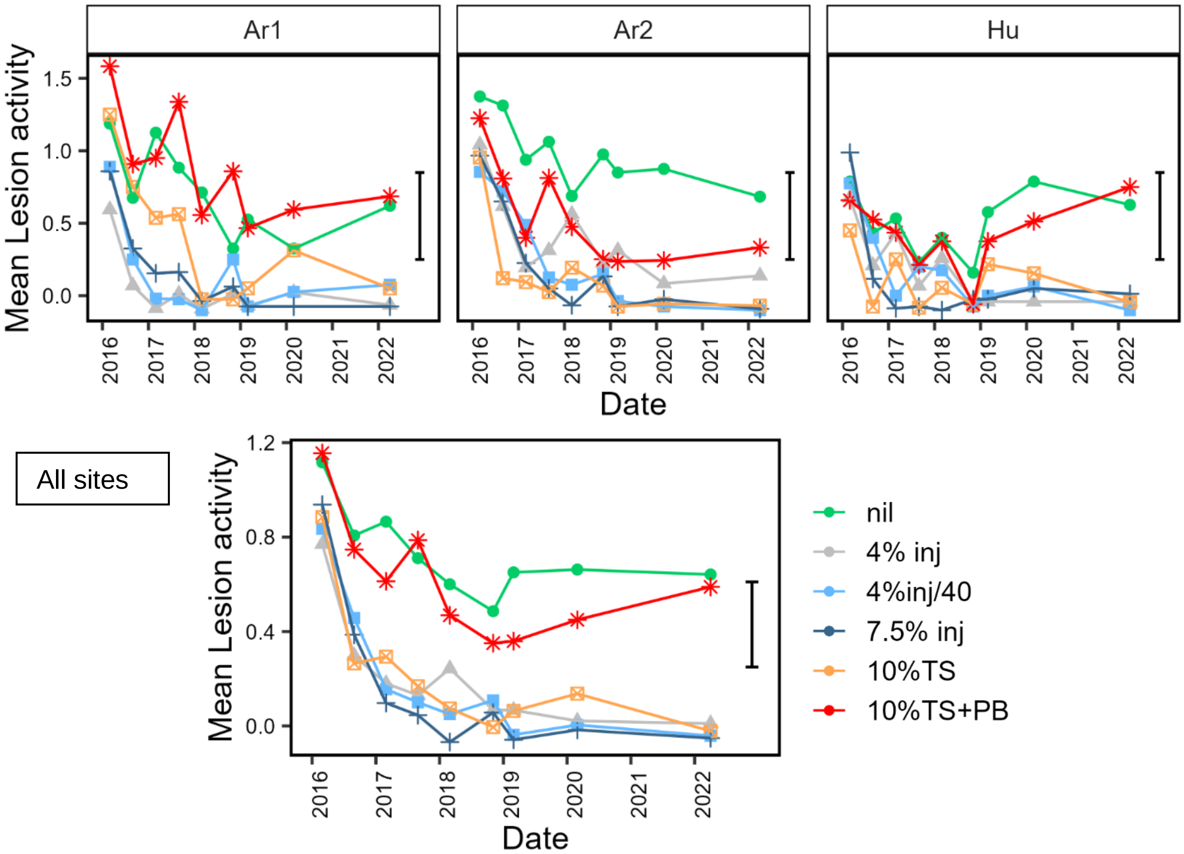


Figure 33. Mean basal trunk lesion activity scores on *Phytophthora agathidicida*-infected kauri trees in three forest sites (Arapohue 1 & 2 and Huia). The top graphs are mean data for individual sites, the lower graph for all three sites combined. Data were collected pre-treatment and for 6 years post-treatment from trees treated with various phosphite formulations. Lesion activity was assessed as -0.1 = healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active. All treatments were applied in February/March 2016. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk sprays were re-applied in February/March 2018, February 2020 and March 2022. Bars are least significant difference (p = 0.05).

The trunk-spray with the penetrant additive was less effective than the formulation without the additive, with lesion activity similar to that in untreated control trees (Figure 33, Table 5) and lesion expansion mid-way between untreated control and injection treatment (Figures 36 & 37, Table 6). Following the second application of this treatment, new information from the Agrifos supplier indicated problems with the Pentrabark surfactant with this particular formulation of phosphite, hence the change in the surfactant (to Infiltrate Bark Penetrant) in the year 4 and year 6 trunk spray applications. However, there was no evidence of an improvement in lesion activity or expansion following this change.

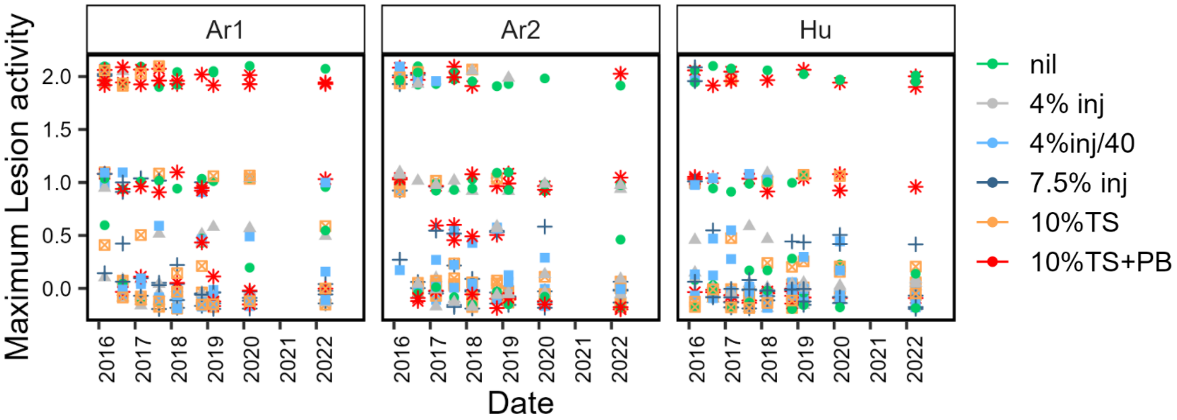


Figure 34. Basal trunk lesion activity scores on *Phytophthora agathidicida*-infected kauri trees in three forest sites (Arapohue 1 & 2 and Huia). Data are for the maximum lesion activity score for individual trees at each site. Data were collected pre-treatment and for 6 years post-treatment of trees with various phosphite formulations. Lesion activity was assessed as -0.1=healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active. All treatments were applied in February/March 2016. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk sprays were re-applied in February/March 2018, February 2020 and March 2022. Bars are least significant difference.

Table 5. Maximum lesion activity in kauri trees in assessments made in 2022, 6 years after treatment with various phosphite formulations. Data are across all trees on all three sites. All treatments were applied in February/March 2016. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Percentage figures for treatments are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk sprays were re-applied in February/March 2018, February 2020 and March 2022. 'Percentage analyses' are on the percentage of trees with at least one 'very active', 'active' or 'probably active' lesion in the 2022 assessment. Fitting a binomial generalised linear model to this showed a significant treatment effect (deviance =28.3 on 5 df,, $p < 0.001$) but no significant site or site × treatment effects (deviance = 2.7 on 2 df, $p = 0.266$, and deviance = 12.1 on 10 df, $p = 0.276$). LSD = least significant difference: Treatments which do not have a letter in common were significantly different ($P < 0.05$).

Treatment	Maximum lesion activity, by tree					Percentage analyses on active lesions		
	0 (not active)	0.2 (probably not active)	0.5 (probably active)	1 (active)	2 (very active)	Back transformed Mean	Back transformed 95% confidence interval	LSD Letter Group
nil	2	1	1	4	4	75%	44% - 92%	A
4% inj	9	0	1	2	0	25%	8% - 56%	BC
4%inj/40	10	1	0	1	0	8%	1% - 42%	C
7.5% inj	11	0	1	0	0	8%	1% - 42%	C
10%TS	10	1	1	0	0	8%	1% - 42%	C
10%TS+PB	4	0	0	3	5	67%	37% - 87%	AB

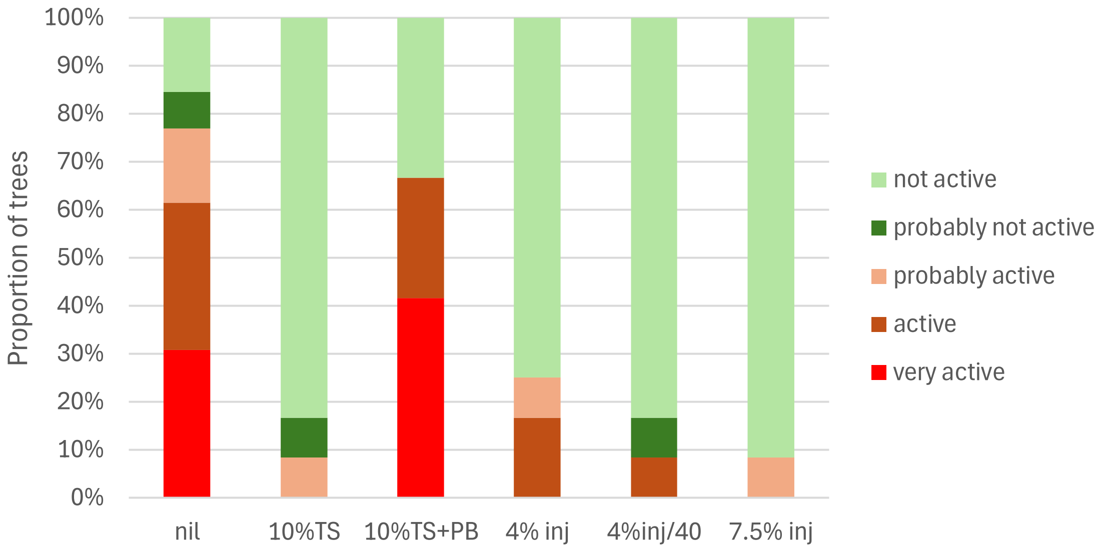


Figure 35. Maximum basal trunk lesion activity scores on *Phytophthora agathidicida*-infected kauri trees in three forest sites. Data are the proportion in various lesion activity categories, using the maximum score for each tree, assessed 6 years after initial application of various phosphite treatments in February/March 2016. Data are pooled across three sites. Lesion activity was assessed as 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active. All treatments were applied in February/March 2016. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk sprays were re-applied in February/March 2018 and February 2020.

Table 6. Analysis of the mean of lesion advance in cm/year (slope calculations) in *Phytophthora agathidicida*-infected kauri trees across three trial sites. Pre-marked lesion expansion was recorded approximately 6-monthly for four years following treatment with various phosphite applications. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Treatment percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm.

	nil	4% inj	4% inj/40	7.5% inj	10%TS	10%TS+PB
Slope Estimate	4.2	-0.9	-1.6	-1.9	-0.7	4.0
Standard Error	2.1	1.7	1.6	1.6	1.7	2.1
Least significant difference	4.8					

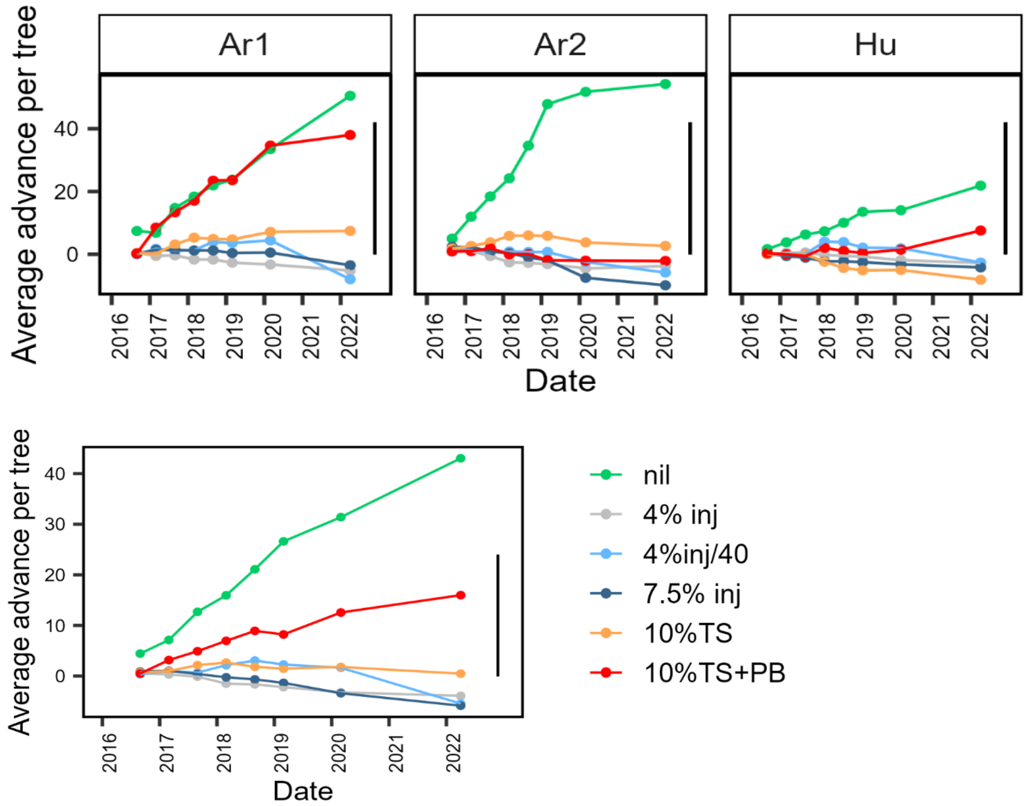


Figure 36. Mean basal trunk lesion advance on *Phytophthora agathidicida*-infected kauri trees in three forest sites (Arapohue 1 & 2 and Huia), following application of various phosphite treatments in February/March 2016. Expansion of pre-marked lesions was recorded approximately 6-monthly for 4 years, and again after 6 years. Negative values reflect lesion healing and shrinking. Line slope calculations and analysis are presented in Table 6 above. The upper graphs are data for individual sites, the lower graphs for combined data. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk spray treatments were re-applied in February/March 2018 and February 2020. The error bar is least significant difference (LSD) for 2022 data.

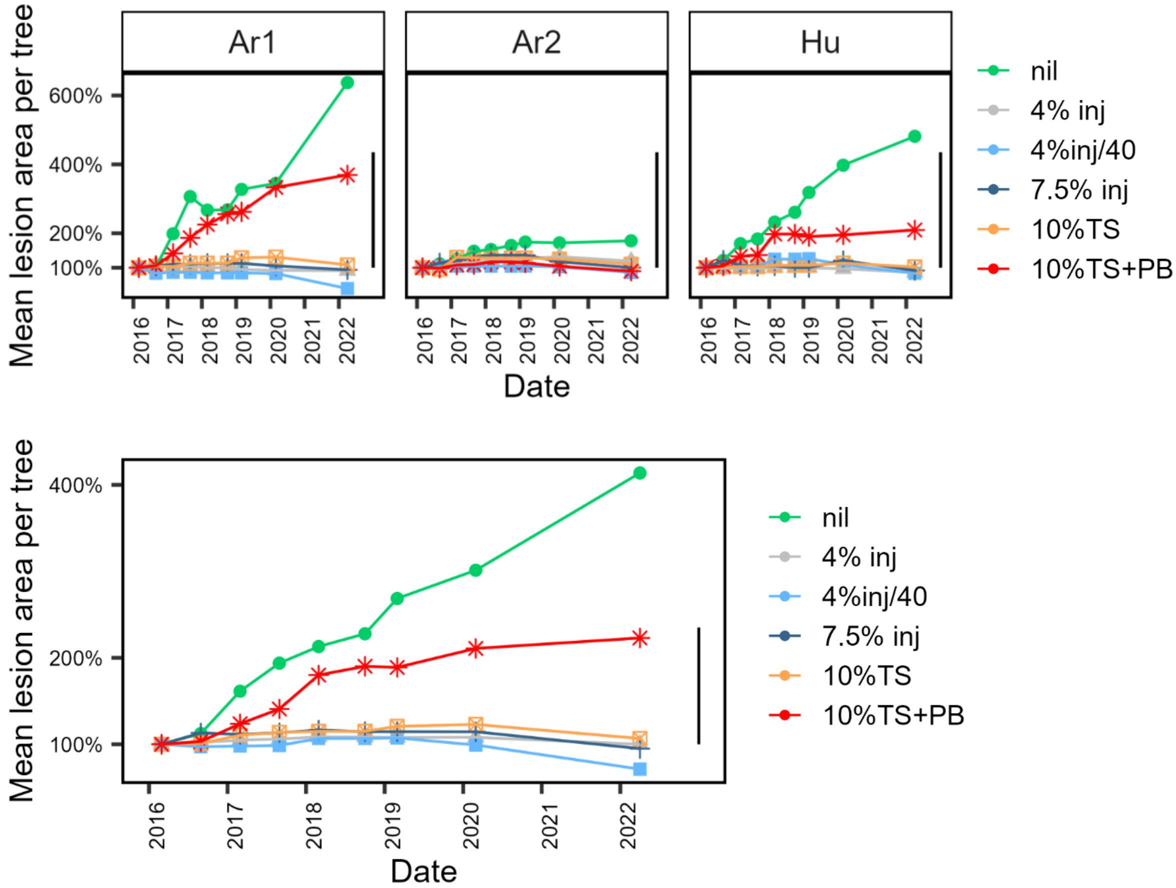


Figure 37. Mean basal trunk lesion area on *Phytophthora agathidicida*-infected kauri trees in three forest sites (Arapohue 1 & 2 and Huia), following application of various phosphite treatments in February/March 2016. Data are lesion areas recorded approximately 6-monthly for 4 years, and again after 6 years, expressed as a percentage of the pre-treatment (2016) lesion area. Values below 100% reflect lesion healing and shrinking. The upper graphs are data for individual sites, the lower graphs for combined data. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Percentage figures for treatments are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk spray treatments were re-applied in February/March 2018 and February 2020. The error bar is least significant difference (LSD) for 2022 data.

4.2.3 Canopy health and shoot growth

Canopy score

Canopy scores for all trees assessed pre-treatment in 2016 and again 6 years post-treatment are shown in Figure 38. Across the trial the majority of trees had increasing canopy scores over the 6 years, i.e. trees were declining and the canopy disease symptoms appeared worse. However, there were differences between sites and between treatments. Looking at the proportion of trees which stayed the same or got better, using a binomial generalised linear model, there were significant differences between sites (9/24 at Huia and Arapohue2; 2/24 at Arapohue1; p = 0.008), and between treatments (7/12 for 4%/40, 6/12 for 7.5%, 3/12 for 4%, 2/10 for 10% TS and 10% TS+PB, 0/12 for nil; p = 0.002). The treatment pattern was similar across sites (interaction p = 0.516). Thus, decline was greatest in the untreated controls, followed by the trunk spray treatments, with the injection at 7.5% and 4%/40 showing the most trees with stable or improved canopies.

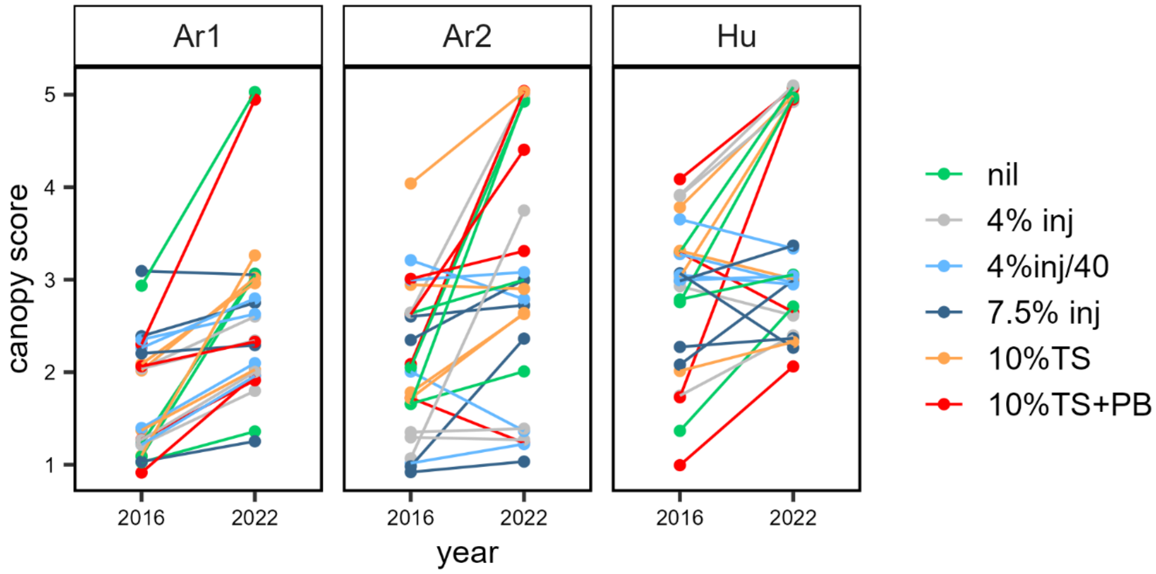


Figure 38. Change in canopy health score in kauri trees, recorded pretreatment and again after 6 years, following treatment with various phosphite applications in February 2016. The three graphs are individual tree data at each of the three trial sites (Arapohue 1 & 2 and Huia). Canopy health was scored as 1 = healthy, no signs of dieback, 2 = canopy thinning, 3 = thinning and some branch dieback, 4 = severe dieback, 5 = dead. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Treatment percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk spray treatments were re-applied in February/March 2018 and February 2020.

Photo comparison

Six-monthly assessments of canopies using baseline photographs for comparison indicated that there was a steady significant decline in mean canopy health in untreated control trees across the trial (Figure 39). In contrast, 4- and 6-years after treatment in the 7.5% and 4%/40cm injection treatments, mean canopy health was the same or better than it had been pre-treatment, i.e. there had been no decline. After 6 years there was also significantly poorer mean canopy health in the trunk spray treatments than in the best injection treatments, although the decline was not as great as in the untreated controls. As noted for the shoot growth analysis below, results were heavily influenced by tree death.

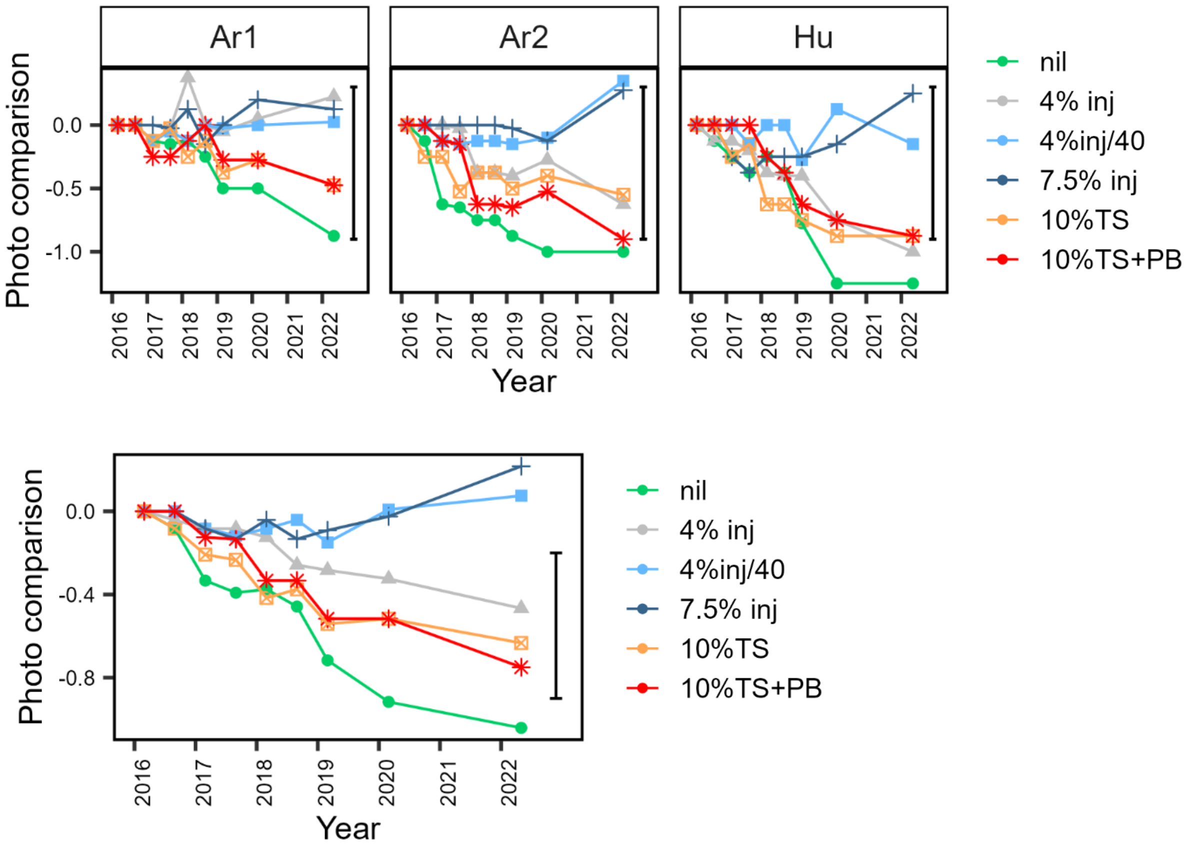


Figure 39. Mean canopy photo change in kauri trees, recorded in 6-monthly assessments for 4 years and again after 6 years, following treatment with various phosphite applications in February 2016. The top three graphs are mean data at each of the three trial sites (Arapohue 1 & 2 and Huia), the lower graph is data pooled across three trial sites. The scoring system comparing canopy health and density with that in the original photographs was: -2 = tree dead, -1 = substantially worse, -0.5 = slightly worse, 0 = similar, 0.5 = slightly better, 1 = substantially better. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Treatment percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk spray treatments were re-applied in February/March 2018 and February 2020. Bar = Least significant difference (LSD) $p = 0.05$.

Shoot Growth

Pre-treatment (2016) shoot growth scores were similar across all treatment groups (Figure 40), but over time the mean growth scores significantly declined in the untreated control and trunk spray treatments (Figure 40, Table 7). This was heavily influenced by the tree deaths that occurred, with such trees recording shoot growth scores of ‘0’. However, excluding the trees that died, average shoot growth scores were still significantly better in injected than in untreated or trunk-sprayed trees (Figure 41, Table 7).

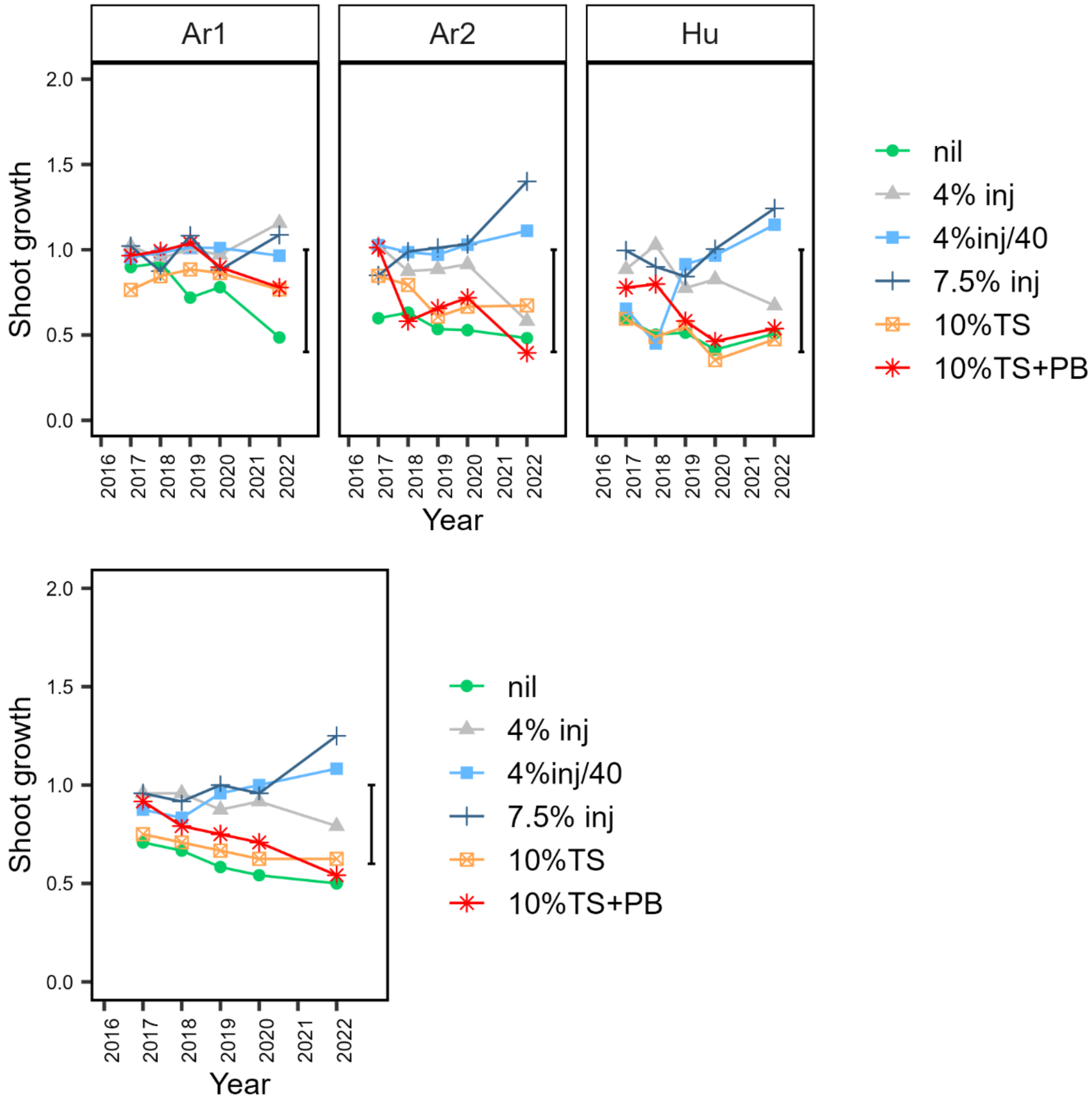
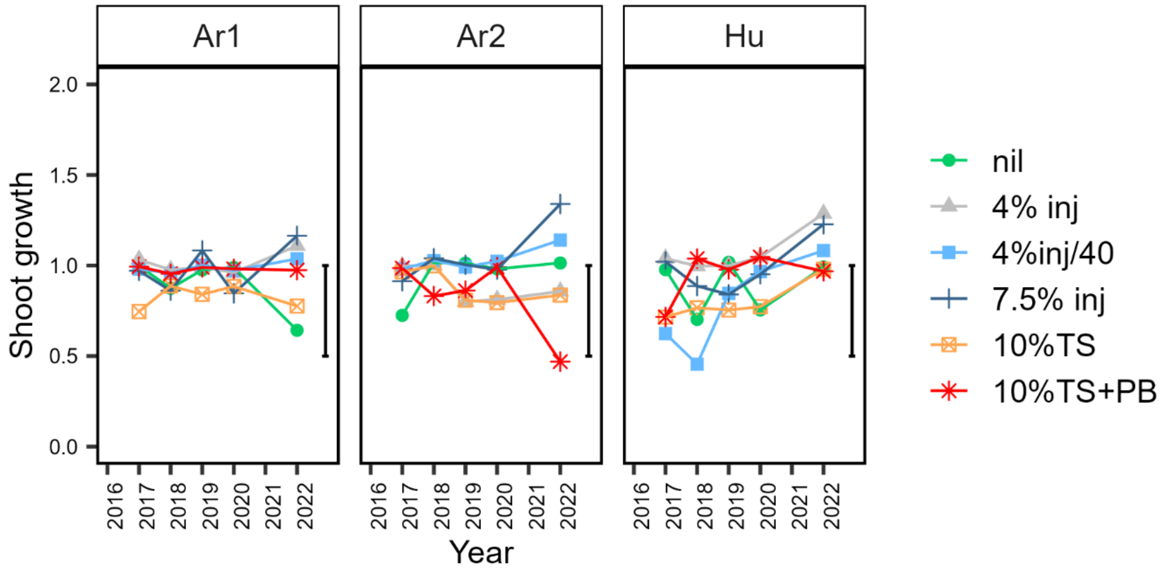


Figure 40. Mean shoot growth score of kauri trees recorded annually (February/March) for 6 years following treatment with various phosphite applications. The top three graphs are mean data for all trees at each site (Arapohue 1 & 2 and Huia), and the lower graph is data pooled across all three trial sites. Shoot growth was recorded on a 0–2 scale, where 0 = no growth, 0.5 = some but poor growth, 1 = good (‘normal’) growth and 2 = vigorous growth. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Treatment percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk spray treatments were re-applied in February/March 2018 and February 2020. The bar is the least significant difference (LSD) at 95%.



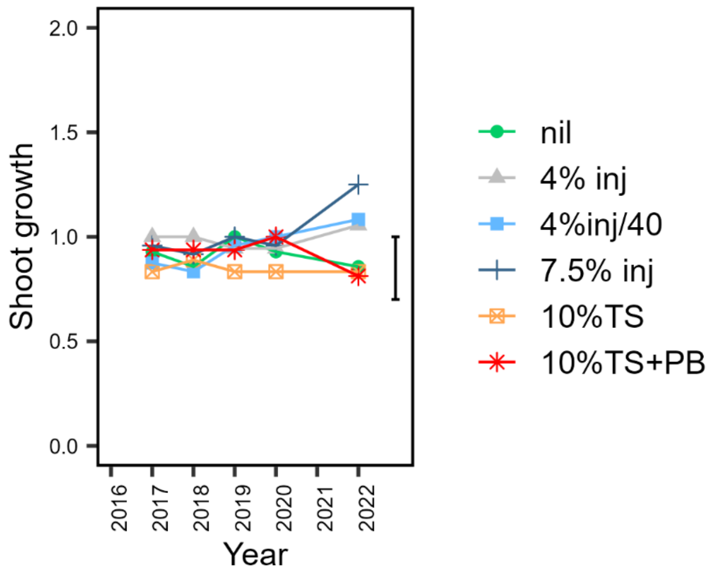


Figure 41. Mean shoot growth score (surviving trees only) of kauri trees recorded annually (February/March) for 6 years following treatment with various phosphite applications. The top three graphs are mean data for all surviving trees at each site (Arapohue 1 & 2 and Huia), and the lower graph is data pooled across all three trial sites (surviving trees only). Shoot growth was recorded on a 0–2 scale, where 0 = no growth, 0.5 = some but poor growth, 1 = good ('normal') growth and 2 = vigorous growth. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Treatment percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk spray treatments were re-applied in February/March 2018 and February 2020. The bar is the least significant difference (LSD) at 95%.

Table 7. Mean shoot growth score of kauri trees assessed in 2022, 6 years after treatment with various phosphite applications. Data are pooled across three trial sites, with separate analyses for 'all trees' and surviving (live) trees. Shoot growth was recorded on a 0–2 scale, where 0 = no growth, 0.5 = some but poor growth, 1 = good ('normal') growth and 2 = vigorous growth. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Treatment percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk spray treatments were re-applied in February/March 2018 and February 2020. LSD = least significant difference: Treatments which do not have a letter in common were significantly different (P < 0.05).

	All trees			Live trees		
Treatment	Mean	SE (fitted)	LSD letter group	Mean	SE (fitted)	LSD letter group
Nil	0.50	0.14	c	0.89	0.12	b
4% inj	0.79	0.14	bc	1.07	0.11	ab
4% inj/40	1.08	0.14	ab	1.08	0.09	ab
7.5% inj	1.25	0.14	a	1.25	0.09	a
10% TS	0.63	0.14	c	0.86	0.11	b
10% TS+PB	0.54	0.14	c	0.83	0.12	b
LSD	0.38			0.31		
ANOVA	F	p		F	p	
Site (2 and 54 df)	0.5	0.617		1.0	0.371	
Treatment (5 and 54 df)	5.1	<0.001		2.8	0.031	
Site x Treat (10 and 54 df)	0.5	0.903		0.9	0.540	

4.2.4 Phytotoxicity

To date, none of the canopy phytotoxicity symptoms such as the leaf yellowing and canopy thinning noted in the earlier ricker trials have been observed in this low-rate ricker trial, even with the 7.5% injection. In assessments made 1, 1.5 and 2 years after treatment, minor 'stretch marks' were noted in the trunks of almost half the injected trees, seemingly in line with injection points. These were noted with both 7.5% and 4% phosphite concentrations. These marks were less obvious in the 2.5-, 3-, 4- and 6-year assessments. Eighteen months after treatment, small bleeds in line with injection points were observed in about half the trees in both the 7.5% and 4% injection treatments at the Huia site, and one minor bleed was noted in one of the Arapohue trees injected with 7.5% phosphite. All these bleeds appeared dry and healed in all subsequent assessments.

In some of the 'trunk spray' trees there was prolific peeling of bark in the sprayed zone, first noted in the 6-month assessment (Figure 42). This was not just around lesion margins, but extended throughout the zone that had been sprayed. In some cases the peeling was of bark that would not normally be expected to peel as rapidly, although there appeared to be healthy bark below. By 1.5–2 years post-treatment almost all this bark had shed and trunks appeared normal and healthy. There was only a small amount of bark peeling following the second spray application of phosphite after 2 years.

Figure 42. Peeling of kauri bark stimulated by a trunk-spray application of phosphite.

4.2.5 Mortality

Four years after initial treatment, 15 of the 72 trial trees had died, with deaths of 5/12 'untreated' control trees and 7/24 'trunk spray' (+/- penetrant) trees (Table 8, Figure 43). Only three out of 36 trunk-injected trees died (all in the 4%/20 cm treatment). Statistical analysis fitting a binomial generalised linear model showed survival was significantly better in the 4%/20cm and 7% injected treatments than in untreated controls. Survival following trunk-spray application was midway between that of the best injection treatment and untreated trees, as was the 4% injection treatment (Table 8). The proportion dead varied near-significantly between sites (8/24 at Huia, 2/24 at Aropohue1, 5/24 at Aropohue2; p = 0.059).

Table 8. Mortality of *Phytophthora agathidicida*-infected kauri trees recorded in March 2022, 6 years after treatment with various phosphite applications. Data are from three trial sites (Arapohue 1 & 2 and Huia), with analysis on the pooled data. The letters adjacent to the death percentages indicate significance groupings of means (at $p = 0.05$, based on a pairwise likelihood ratio test); percentages which do not have a letter in common were significantly different. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Treatment percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk spray treatments were re-applied in February/March 2018 and February 2020.

Treatment	Hu	Ar1	Ar2	Number dead/total	Percent dead
Nil	2/4	1/4	2/4	5/12	42% b
4% inj	2/4	0/4	1/4	3/12	25% b
4% inj/40	0/4	0/4	0/4	0/12	0% a
7.5% inj	0/4	0/4	0/4	0/12	0% a
10%TS	2/4	0/4	1/4	3/12	25% b
10%TS+PB	2/4	1/4	1/4	4/12	33% b

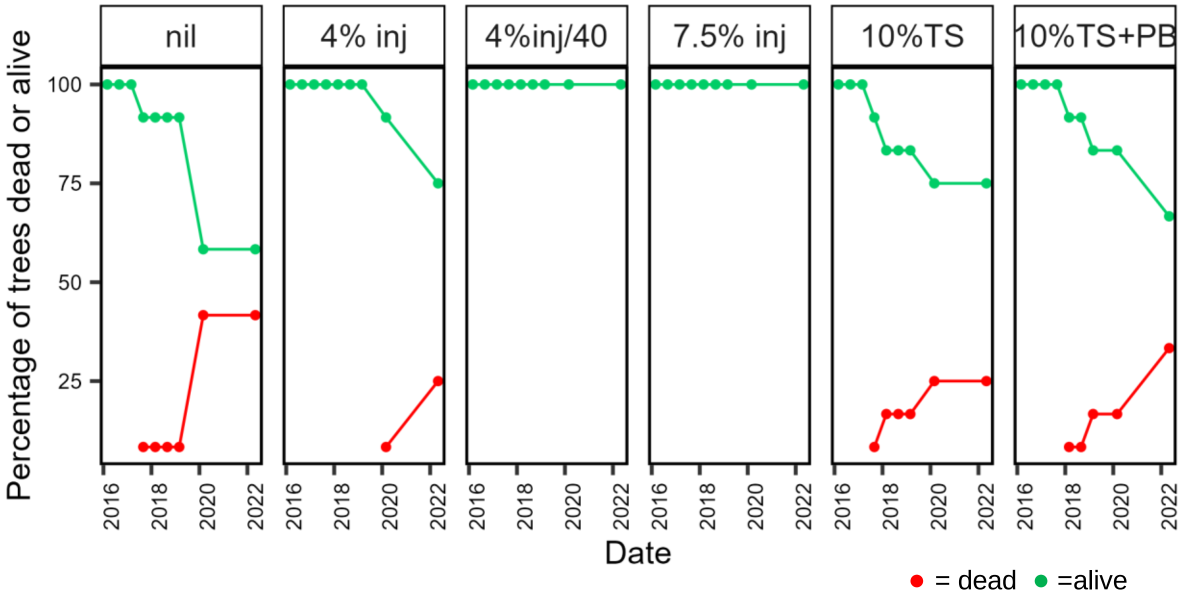


Figure 43. Percentage mortality/survival of *Phytophthora agathidicida*-infected kauri trees recorded for 6 years following treatment with various phosphite applications. Data are pooled across three trial sites. TS = trunk spray, PB = Pentrabark™, inj = trunk injection. Treatment percentage figures are phosphite concentrations. 4%inj/40 = 4% phosphite, 20 mL injected every 40 cm around the trunk. Both other injection treatments were 20 mL every 20 cm. Trunk spray treatments were re-applied in February/March 2018 and February 2020.

4.3 Discussion – low rate and trunk spray trial

From this study, there is evidence that a concentration of 4% phosphite can be effective at healing or containing *P. agathidicida* lesions on ricker to early mature kauri, without causing significant phytotoxicity effects. There is also evidence that the lower rate of one injector every 40 cm around the trunk provides control for a period of at least 4 years. There was a hint of a loss of control after the 6-year assessment, with a few active or semi-active lesions appearing in both the 4% and 7.5% treatments.

The trunk-spray application of phosphite was not as effective as trunk injection, but still had some benefits, particularly in terms of lesion healing. The trunk spray without the penetrant generally gave better results than when the penetrant was added. Evidence to date suggests that trunk application needs to be repeated on a regular basis (e.g. every 2 years) to provide adequate lesion healing. There is no information on the systemic movement beyond the area where the spray was applied, and it cannot be assumed that the treatment is efficacious beyond the application zone. The fact that lesion activity scores improved following regular trunk spray application, but that canopies still declined over time suggests local healing of lesions from the topically applied phosphite, but not the systemic healing such as that presumed to be occurring with the injected treatments. Trunk spray application may be a useful supplement to help to rapidly heal superficial above-ground trunk lesions.

5 Large tree trial 2016

Forest trials established in 2012, testing phosphite for kauri dieback control, provided promising results, with demonstration of a curative effect (Horner et al. 2015; Section 3 above). However, these trials were all carried out with trees in the 'ricker' size class, mostly 15–35 cm trunk diameter, with no testing on larger trees. Before any future deployment to treat moderate-sized trees or large iconic trees, and to allow informed decisions to be made, information on safe and effective treatment regimes for large trees was required. Doses based on trunk girth have previously been used to calculate required phosphite volumes. But with giants such as kauri, scaling up from rickers to trees with girths of 5–15 m may be difficult. Earlier trials also indicated some problems with phytotoxicity, particularly with higher phosphite rates, so it was very important that effects on larger trees were assessed before widespread release of the treatment. A balance must be struck between rates sufficient to suppress the disease, yet still safe for the tree.

In 2016, new trials were established on large kauri trees to help to determine appropriate treatment regimes, with emphasis on phosphite rates and doses lower than those used in previous trials. This section summarises trial protocols, and results of 6-monthly assessments made on all trial trees from February 2016 to February 2022, and supersedes the previous report in September 2020 (Horner et al. 2021).

5.1 Methods

5.1.1 Trial sites and tree selection

Three sites were selected for the trials: Puketotara Road, between Kerikeri and Ōkaihau in Northland; Trounson Kauri Park in Northland; and the Cascades in Te Wao Nui ā Tiriwa (Waitākere Ranges), Auckland. The Puketotara block is on private farmland, Trounson Kauri Park is under DOC and Te Roroa jurisdiction, and the Cascades are under Auckland Council and Te Kawerau ā Maki jurisdiction.

All 42 trees in the trial were in the mature stage. At Puketotara, the nine trial trees ranged in size from 0.4 to 1.1 m trunk diameter. At Trounson, the 15 trial trees ranged from 1.0 to 2.1 m trunk diameter, and the 18 trial trees at the Cascades ranged from 0.6 to 2.4 m diameter (Figure 44). All trial trees showed symptoms of kauri dieback at the start of the trial, including basal trunk lesions.

5.1.2 Pre-treatment assessments and trial design

Before treatment, baseline assessments were made on various tree growth and health parameters. These included trunk girth, canopy health score, canopy colour, plus trunk lesion size and activity (see section 3.1.2 above and 5.1.4 below for scoring scales). Canopy scores of trial trees ranged from 1 to 4 on the KDP canopy scoring scale (Figure 45). Canopy photographs were taken for later comparison, and selected basal trunk lesion margins were marked for subsequent measurement of expansion (Figure 46).

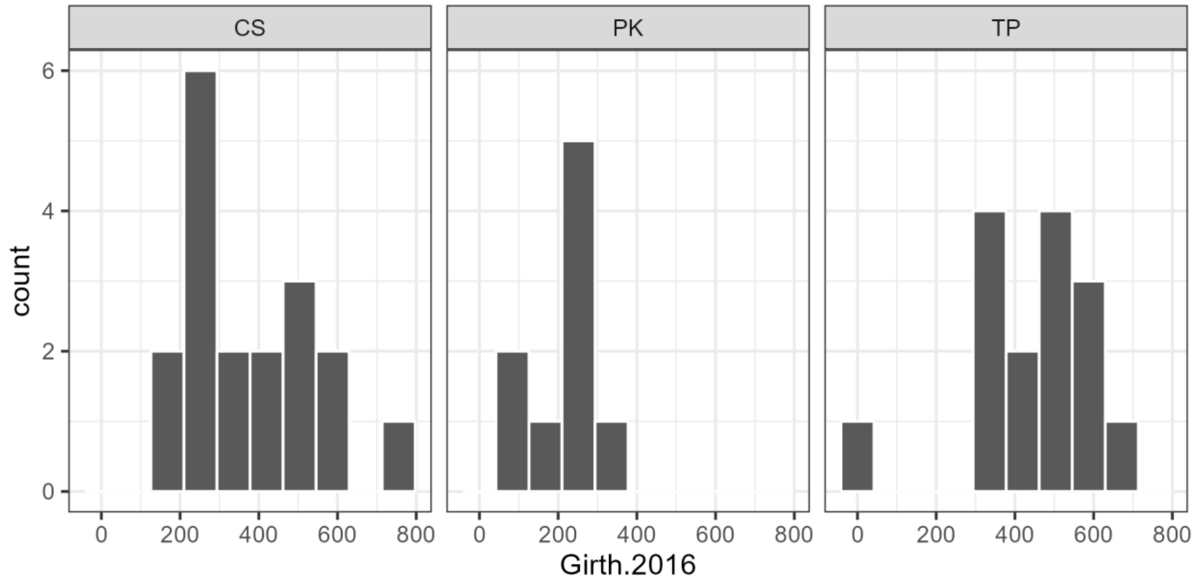


Figure 44. Trunk girth (cm) of all kauri trees used in the large tree phosphite trial across Cascades (CS), Puketotara (PK) and Trounson Park (TP) trial sites. Measurements were taken pre-treatment in 2016. 'Count' = number of trees in each size class.

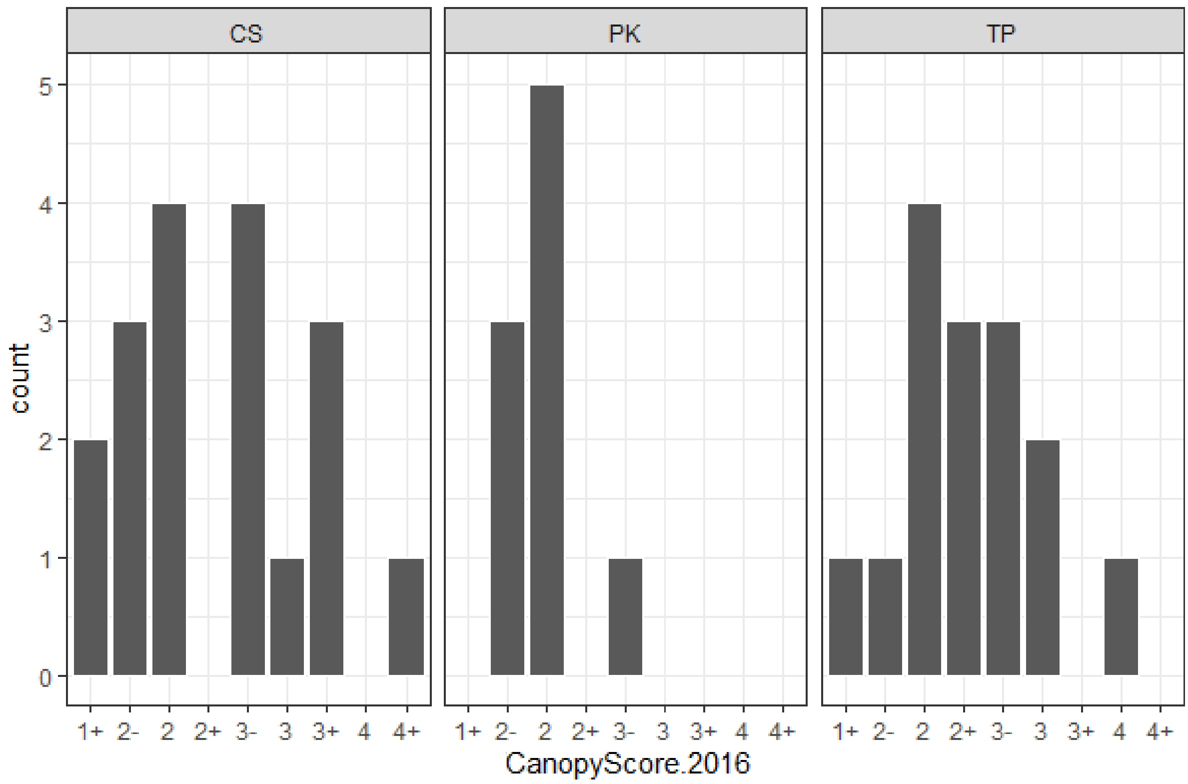


Figure 45. Pre-treatment canopy disease scores of all kauri trees used in the large tree phosphite trial, across Cascades (CS), Puketotara (PK) and Trounson Park (TP) sites. The overall canopy health was scored on a modification of the 1–5 canopy health scoring scale adopted be the Kauri Dieback Programme (1 = healthy crown – no visible signs of dieback, 2 = foliage/canopy thinning, 3 = thinning and some branch dieback, 4 = severe dieback, 5 = dead), with '+' and '-' scores used to further discriminate between categories. 'Count' = number of trees in each canopy score category.

There was a total of 42 trial trees (nine at Puketotara, 15 at Trounson and 18 at the Cascades). This is double the number that was proposed in the initial trial outline, thereby providing a more robust dataset. At each site, trees were divided equally among the three treatments. To ensure a relatively even distribution of disease symptoms across treatments, at each site trees were placed into groupings based on disease parameters such as lesion activity and canopy symptoms before random assignment of the various treatments within each grouping.

Figure 46. Baseline paint marks used to indicate *Phytophthora agathidicida* lesion margins on kauri tree trunks before treatment application. Marks were drawn 1 cm beyond the visible margin, with reference marks 10 and 20 cm from that margin, to help where advance was rapid or interpretation confounded by sap flow.

5.1.3 Treatment selection and application

The determination of phosphite concentration and doses for the large trees was difficult. With trunk girth being the main determinant of dose, and with no international experience with treating trees of such size, a very conservative approach was taken. This decision was in part influenced by previous experiences with phytotoxicity. The selected phosphite concentration of 4%, with injector frequency of one every 40 cm, corresponded to the lowest rate and dose used in the concurrent ‘Trunk spray and low-rate trial’ (Section 4 above) (Figure 47). We also included another treatment with an even lower dose of one injector every 80-cm girth. Although this dose may be too low to provide adequate long-term control, there was the opportunity to observe effects over the first year or two, then make another application if deemed appropriate. This was subsequently carried out, as noted below.

Treatments used in the trial were:

1. Untreated control
2. 4% phosphite trunk injection, 20 mL every 40 cm around the trunk circumference
3. 4% phosphite trunk injection, 20 mL every 80 cm around the trunk circumference.

Agrifos600® (Key Industries) was the phosphite formulation used for all applications. The 4% refers to 4% active ingredient (a.i.), i.e. a 1:14 dilution of Agri-fos®600. Injection followed the method outlined in 3.1.3 above, except that drill-holes were not filled with silicon sealant following injection.

Treatments were applied at the Puketotara site in March 2016 and at the Trounson and Cascades sites in November 2016. The delayed start at Trounson and the Cascades related to delays in obtaining permits. Treatment 3 applications were repeated at the Puketotara site in March 2018, and in the Trounson and Cascades sites in March 2019. Following discussions with the KDP Planning & Intelligence team, Treatment 2 was re-applied at all sites in June 2019. Re-treatment injection points were placed mid-way between previous injection points.

5.1.4 Post-treatment assessments

Post-treatment tree health and lesion expansion/activity was assessed approximately every 6 months for four years. Assessments were made in August 2016 for the Puketotara site and February/March 2017, August 2017, March 2018, October 2018, February/March 2019, August 2019, February 2020 and August 2020 for all three sites. The later-than-planned assessment in October 2018 was because of delays in gaining permission to access sites in the Waitākere Ranges with the Controlled Area Notice. A final assessment was made in March/April 2022, approximately 6 years after the start of the trial.

Current season shoot growth was recorded (summer assessments only) on a 0–2 scale (see Section 3.1.4). Canopy colour was recorded as the dominant colour of leaves, noted as green, yellow, brown or canopy dead. The overall canopy health was scored on a modification of the 1–5 canopy health scoring scale adopted by the KDP (see Section 3.1.2). At each 6-monthly assessment, the state of the canopy viewed from a fixed photographic point was compared with a photograph of the canopy taken pre-treatment in 2016 (see Section 3.1.4).

Basal trunk lesions were measured (maximum width and height), their orientation (side of tree, compass bearing) noted, and a judgement was made on the activity of the lesion, scored as described in Section 3.1.4. Selected points on some of the original lesions were marked in 2016, using a paint-pen to mark the lesion boundary and reference points 10 and 20 cm beyond (Figure 46). Any change in the lesion boundary was measured at each 6-monthly assessment, and lesions were re-marked if necessary. In some cases, lesion healing and bark peeling (with healthy bark below) occurred, in which case the lesion advance was recorded as a negative number.

Any signs of phytotoxicity in the canopy (e.g. leaf yellowing/twig drop), and trunk cracks or bleeds associated with injection points were noted at each assessment.

Figure 47. Trunk injection of a large kauri tree at Trounson Kauri Park, with one injector every 40 cm around the trunk circumference.

5.2 Results and discussion

5.2.1 Injection point healing

As in the low-rate trial (Section 4), injection holes were not sealed with silicon in this trial. In most cases the trees naturally plugged the hole with resin within an hour or so of injection (Figure 9), and the holes generally healed very well. A few injection holes in one tree on each of the Puketotara and Cascades sites failed to fill with resin. In both cases, trees were at the advanced stages of infection at the time of injection. It was also observed that these trees had been very slow to take up the phosphite injection, with some injectors not emptying fully.

5.2.2 Lesion activity and expansion

5.2.2.1 Lesion activity

Trunk lesions caused by *P. agathidicida* were assessed 6-monthly throughout the trial. Over the first two years of the trial, it was noted that neither of the phosphite treatments led to complete healing of the lesions at any of the sites. Phosphite treatments were re-applied to all treated trees between 2 and 3 years after the initial treatments. Within 2 months of the second treatment, there was a noticeable decline in lesion activity, and improved healing in treated trees at all sites (Horner & Arnet 2019). In the assessment made 8 months after the second application, the initial improvements were reinforced. In the February/March 2021 assessment, 2-plus years after the second treatment, lesion activity remained low in most treated trees (Figure 48). Analysis of variance on the final time-point data (2022) for maximum lesion activity per tree indicated a significant difference between treatments, with significantly lower lesion activity in phosphite-treated trees than in untreated trees ($p = 0.001$), although a number of lesions in treated trees remained active. There was no significant difference between sites, and no significant site × treatment interaction ($p = 0.96$ and 0.64 respectively). Analysing mean lesion activity per tree gave a similar result (Figure 49).

As noted above, in untreated control trees, on average, lesion activity remained higher than in treated trees. However, in the August 2020 assessment there appeared to be a reduction in average lesion activity in untreated trees, especially at the Puketotara site, and this remained the case in the 2021 assessment. Reasons for this are unclear, but it is possible that the very dry preceding summers may have contributed to lesions drying up. The 2022 assessment showed a return to earlier activity levels.

Even though there was a very significant improvement in lesion healing following phosphite injection, it is concerning that in 2022 almost half of the phosphite-treated trees had at least one active or semi-active lesion (Figure 48, Table 9), although still trending less than the untreated control ($p = 0.057$).

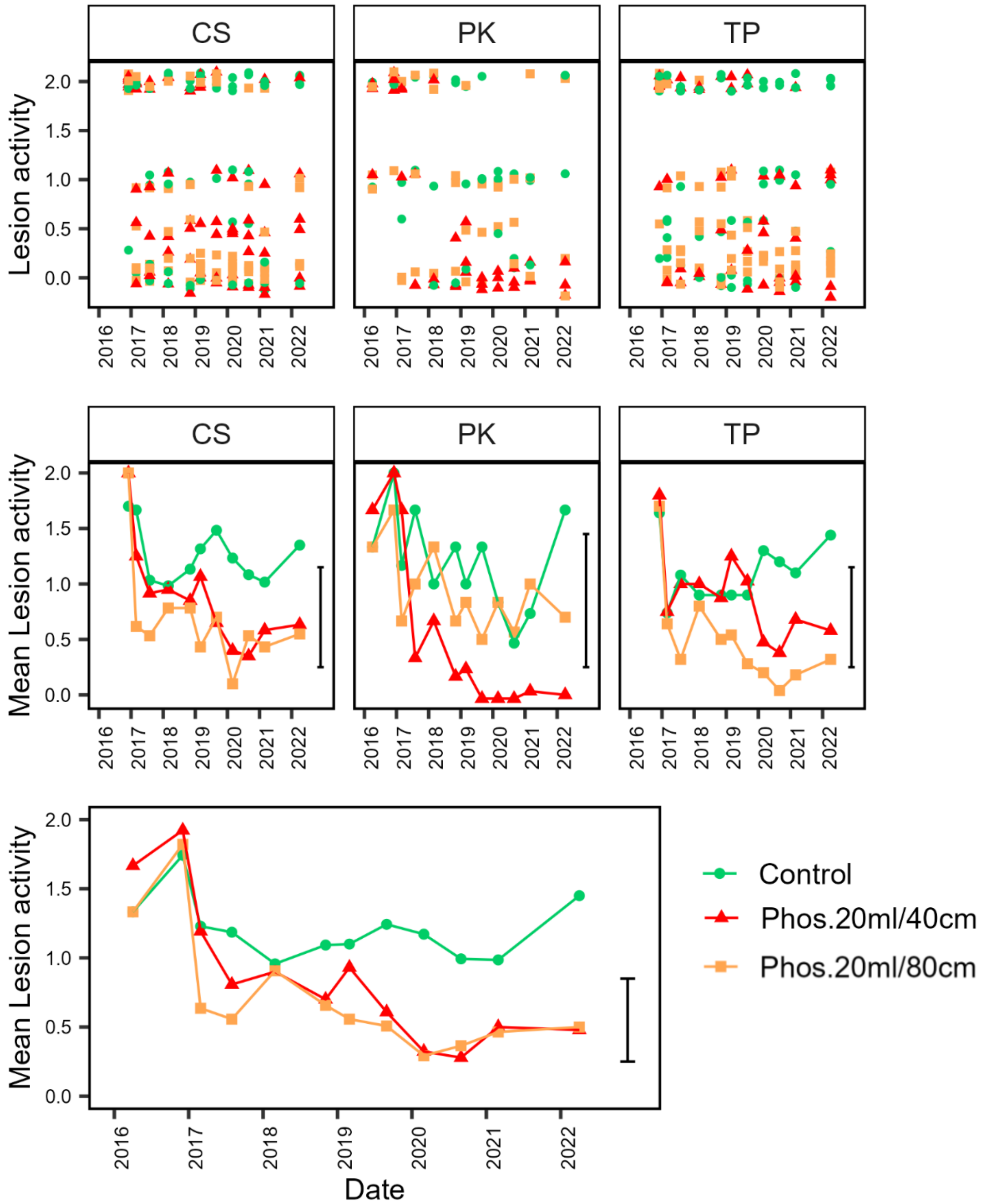


Figure 48. Maximum *Phytophthora agathidicida* lesion activity scores in kauri trees injected with phosphite or left untreated, assessed 6-monthly over 6 years on three sites. All plots are based on the maximum lesion score recorded for each tree at each time-point. Top: Individual tree scores, where each point represents the maximum lesion score across all lesions on a tree. Middle: Mean of maximum lesion score for all trees within a treatment on each of three sites (CS=Cascades, PK=Puketotara and TP=Trounson Park sites). Lower: Mean of tree maximum data across all three sites. Phosphite injections were applied in March 2016 (Puketotara site) or November 2016 (Cascades and Trounson sites). A 4% phosphite solution was applied as one 20-mL injection every 40 cm or one injection every 80 cm around the trunk circumference. Phosphite was re-applied to trees in the low dose treatment (4% injection every 80 cm) at the Puketotara site in March 2018 and Cascades plus Trounson sites in February/March 2019. The higher-dose phosphite treatment (4% injection every 40 cm) was re-applied on all sites in June 2019. Lesion activity scoring: -0.1 = healed and peeled back, 0=not active, 0.2=probably not active, 0.5=probably active, 1=active, 2=very active. The bar indicates average least significant difference (p=0.05) for the 2022 data.

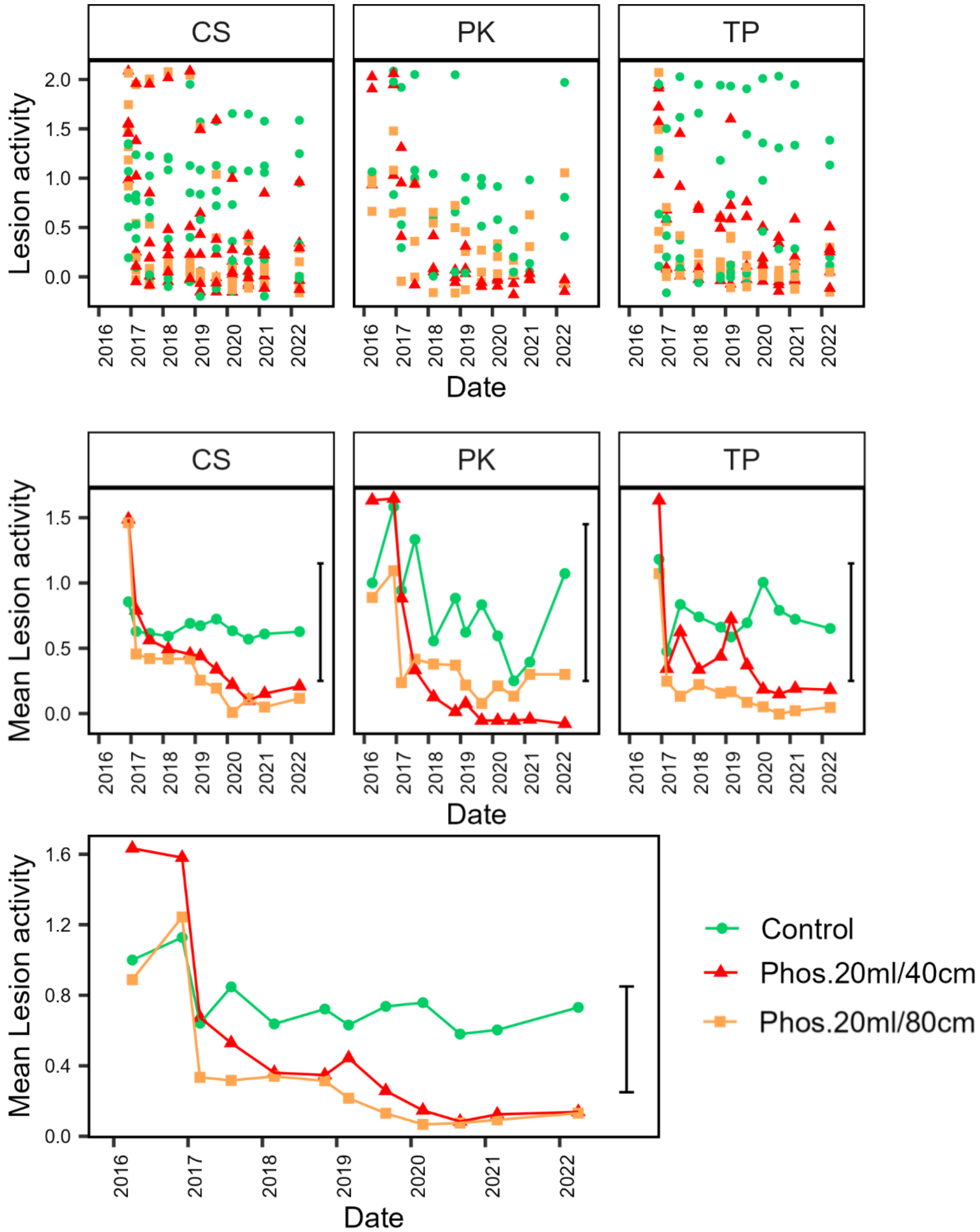


Figure 49. Mean *Phytophthora agathidicida* lesion activity scores in kauri trees injected with phosphite or left untreated, assessed 6-monthly over 6 years on three sites. All plots are based on the mean of all lesion scores recorded for each tree at each time-point. Top: Individual tree scores, where each point represents the mean lesion score across all lesions on a tree. Middle: Mean of lesion scores for all trees within a treatment on each of three sites (CS=Cascades, PK=Puketotara and TP=Trounson Park sites). Lower: Mean of all lesion activity scores across all three sites. Phosphite injections were applied in March 2016 (Puketotara site) or November 2016 (Cascades and Trounson sites). A 4% phosphite solution was applied as one 20-mL injection every 40 cm or one injection every 80 cm around the trunk circumference. Phosphite was re-applied to trees in the low dose treatment (4% injection every 80 cm) at the Puketotara site in March 2018 and Cascade plus Trounson sites in February/March 2019. The higher-dose phosphite treatment (4% injection every 40 cm) was re-applied on all sites in June 2019. Lesion activity scoring: -0.1 = healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active. The bar indicates average least significant difference (p=0.05) for the 2022 data.

Table 9. Number of *Phytophthora agathidicida*-infected kauri trees with at least one active or semi-active bleed, 6 years after being injected with phosphite or left untreated. Data are pooled across three sites (14 trees total in each treatment). Phosphite injections were applied in March 2016 (Puketotara site) or November 2016 (Cascades and Trounson sites). A 4% phosphite solution was applied as one 20-mL injection every 40 cm or one injection every 80 cm around the trunk circumference. Phosphite was re-applied to trees in the low dose treatment (4% injection every 80 cm) at the Puketotara site in March 2018 and Cascade plus Trounson sites in February/March 2019. The higher-dose phosphite treatment (4% injection every 40 cm) was re-applied on all sites in June 2019.

Treatment	Number of trees with active or semi-active lesions in 2022
Untreated control	11/14
4% Phos.20ml/40cm	7/14
4% Phos.20ml/80cm	5/14

It appeared that lesion healing was not as good in the really big trees, compared with the smaller trees within this trial. To test this, 2022 lesion activity scores were plotted against trunk girth (Figure 50). For the untreated control there was no relationship (r = 0.12 p = 0.678), with a similar result for the 4% phosphite and 40 cm spacings (r = 0.13, p = 0.656). However, with the lowest dose of phosphite 4% phosphite and 80 cm spacings, the relationship between tree size and lesion healing was close to significant (r = 0.43, p = 0.121). This suggests that perhaps at the lower limits of phosphite dose (i.e. 4% at 80 cm) the rate calculation based on circumference was not sufficient to provide control in the bigger trees. This idea will be explored further in the general discussion (Section 6).

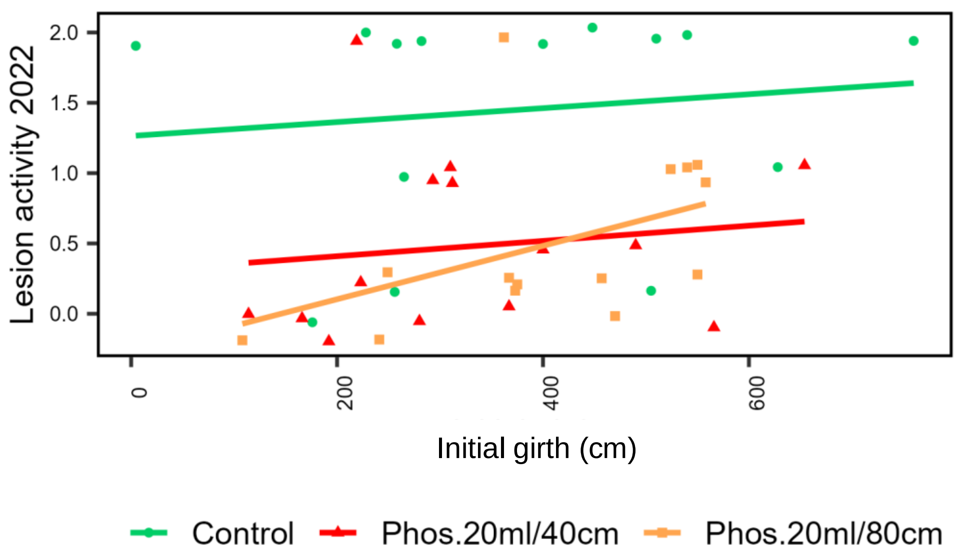


Figure 50. *Phytophthora agathidicida* lesion activity scores plotted against trunk girth, in kauri trees injected with phosphite or left untreated. Points represent individual trees across three sites, using maximum lesion activity score in 2022 versus tree girth (cm) measured pre-treatment. Phosphite injections were applied in March 2016 (Puketotara site) or November 2016 (Cascades and Trounson sites). A 4% phosphite solution was applied as one 20-mL injection every 40 cm or one injection every 80 cm around the trunk circumference. Phosphite was re-applied to trees in the low dose treatment (4% injection every 80 cm) at the Puketotara site in March 2018 and Cascades plus Trounson sites in February/March 2019. The higher-dose phosphite treatment (4% injection every 40 cm) was re-applied on all sites in June 2019. Lesion activity scoring: -0.1 = healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active.

5.2.2.2 Lesion area

Throughout the trial, basal trunk lesion width and height were measured at each assessment. For analysis purposes, lesion area was calculated as 0.5 × height × width, i.e. assuming a triangular lesion shape. While this is a crude approximation of lesion area, it was deemed an unbiased way of determining lesion size for comparisons between treatments.

Measurement of trunk lesions on many of the large trees in this trial proved very problematic, much more difficult than in previous trials with smaller trees. Lesion margins were often poorly defined and confounded by periodic peeling back of very large sheets of bark. There were also some instances where lesions 'popped up' some distance from previously noted lesions. In some cases, these appeared to be a result of spread of the pathogen beneath the bark surface, not apparent until it burst through. The baseline assessments of lesion margin in some cases clearly underestimated the full extent of the lesion. This led to difficulty in interpretation of lesion extent in the field, and some highly variable data, which confounded simple analysis.

All trees had at least one basal trunk lesion at the start of the trial. Some trees had multiple lesions, and there was a very wide range of lesion sizes. Across treatments, many lesions expanded in size, while some shrank, often reflecting healing and/or bark peeling.

To determine if there were patterns across treatments, lesion area at each assessment was expressed as a percentage of the initial lesion area (Figure 51). There was wide variation in the data, and no clear pattern across treatments. Analysis of variance on the last time point (plus last-observations-carried-forward from dead trees) indicated a significant difference between sites (p = 0.018) but there was no significant treatment, or site × treatment interactions (p = 0.54 and 0.12, respectively).

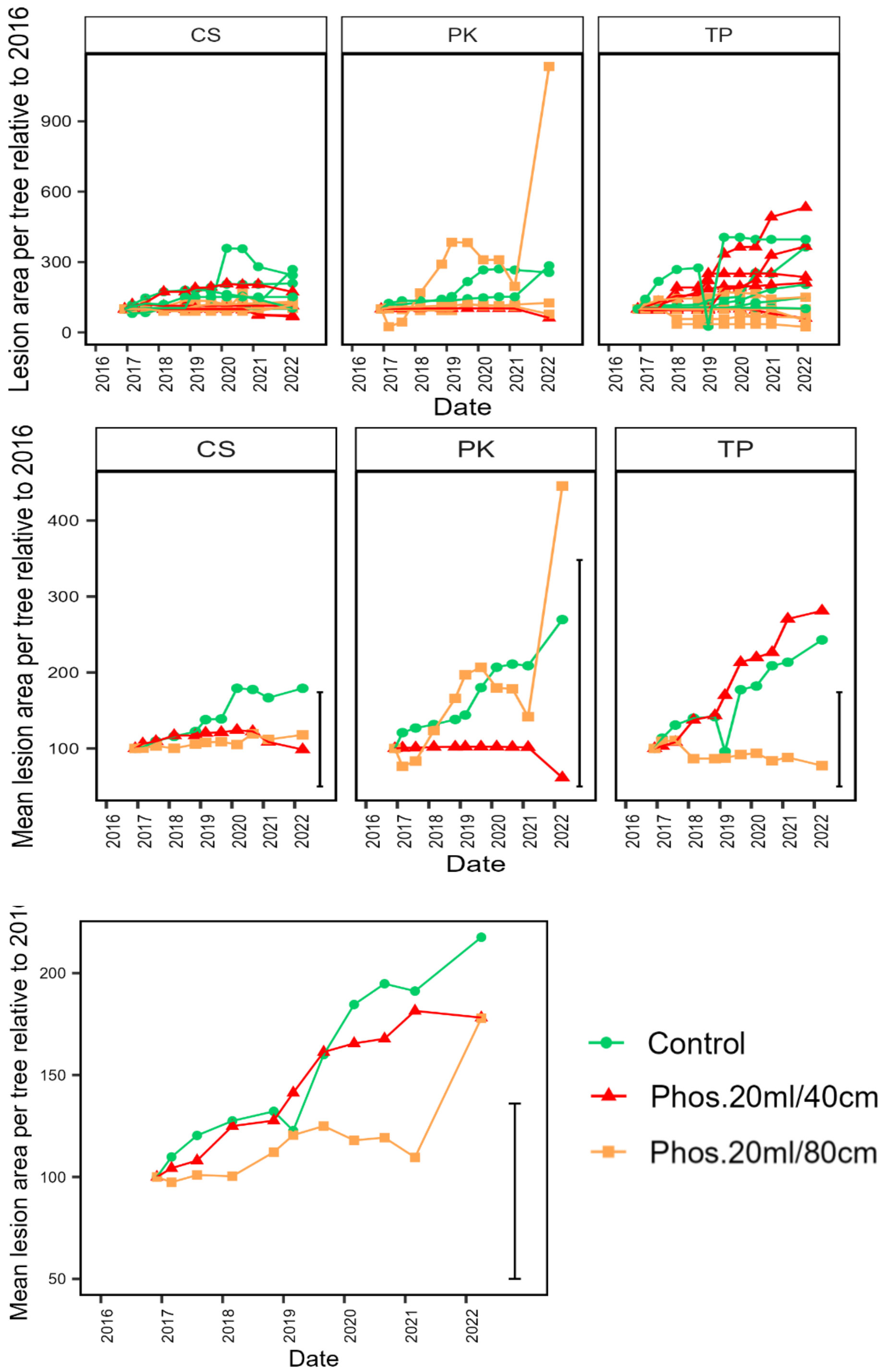


Figure 51. Total trunk lesion area expressed as a percentage of pre-treatment (2016) lesion areas, for kauri trees infected with *Phytophthora agathidicida* across three sites, recorded 6-monthly following treatment with phosphite in 2016. Top: mean data for each tree at each site; Middle: mean data by treatment and site. Bottom: Mean data across all three sites. Bars indicate the least significant difference (LSD, p=0.05) for the final assessment (2022). Sites were Cascades (CS), Puketotara (PK) and Trounson Park (TP). Phosphite injections were applied in March 2016 (Puketotara site) or November 2016 (Cascades and Trounson sites). A 4% phosphite solution was applied as one 20-mL injection every 40 cm or one injection every 80 cm around the trunk circumference. Phosphite was re-applied to trees in the low dose treatment (4% injection every 80 cm) at the Puketotara site in March 2018 and Cascades plus Trounson sites in February/March 2019. The higher-dose phosphite treatment (4% injection every 40 cm) was re-applied on all sites in June 2019.

5.2.2.3 Lesion advance

Before treatment application in 2016, points on the margins of some lesions on most trees were marked using a paint pen (Figure 46). At each 6-monthly assessment, the lesion at each marked point was measured, using the paint mark as a reference point. These assessments faced similar problems to those noted above, with measurement sometimes confounded by major bark shedding or by emergence of lesions beyond points expected from a progressive spread. This led to widely variable data, which precluded sensible statistical analyses. To simplify interpretation, the proportion of trees where lesions, on average, either remained the same or shrank, was calculated (Table 10). Analysis using a binomial generalised linear model on these data indicated a significant treatment effect (p = 0.010), with significantly more trees where lesions did not advance in the phosphite treatments than in the untreated controls. There was also a near-significant site × treatment interaction (p = 0.066, with a bigger difference between treatments at Puketotara and Trounson than at the Cascades), but no significant overall site difference (p = 0.27).

Table 10. Proportion of kauri trees where *Phytophthora agathidicida* lesion margin progression was either zero or was negative (lesion margin retreated), in a comparison of baseline (2016) and final (2022) assessments of specific marked points. Trees at three sites were treated with one of two phosphite treatments or left untreated. Data are the number or trees where lesion margins improved or remained unchanged / total number of marked trees in each treatment.

Site	Control	Phosphite 20 mL/40 cm	Phosphite 20 ml/80 cm
Cascades	2 / 4	3 / 5	3 / 5
Puketotara	1 / 3	3 / 3	2 / 2
Trounson	0 / 4	2 / 5	5 / 5
Total	3/11	8/13	10/12

5.2.3 Canopy health and shoot growth

5.2.3.1 Canopy score

Trial tree canopy scores were assessed pre-treatment and again 5.5–6 years post-treatment. Individual tree data are shown in Figure 52. In the untreated controls, across all three sites, most untreated trees showed higher canopy disease score after 5.5–6 years than at the start of the trial, i.e. there had been a decline in overall canopy health/density. In contrast, among treated trees, many either remained the same or improved slightly in health (although some trees still declined).

When trees were categorised as remaining the same or improving in canopy health, versus declining, there was clear discrimination between treated and untreated trees (Table 11). A binomial generalised linear model analysis on these data indicated a significant treatment effect (p = 0.030), with significantly more trees in decline in untreated controls than in phosphite-injected treatments. There was a significant overall site difference (p = 0.024), and no significant site × treatment interaction (p = 0.116).

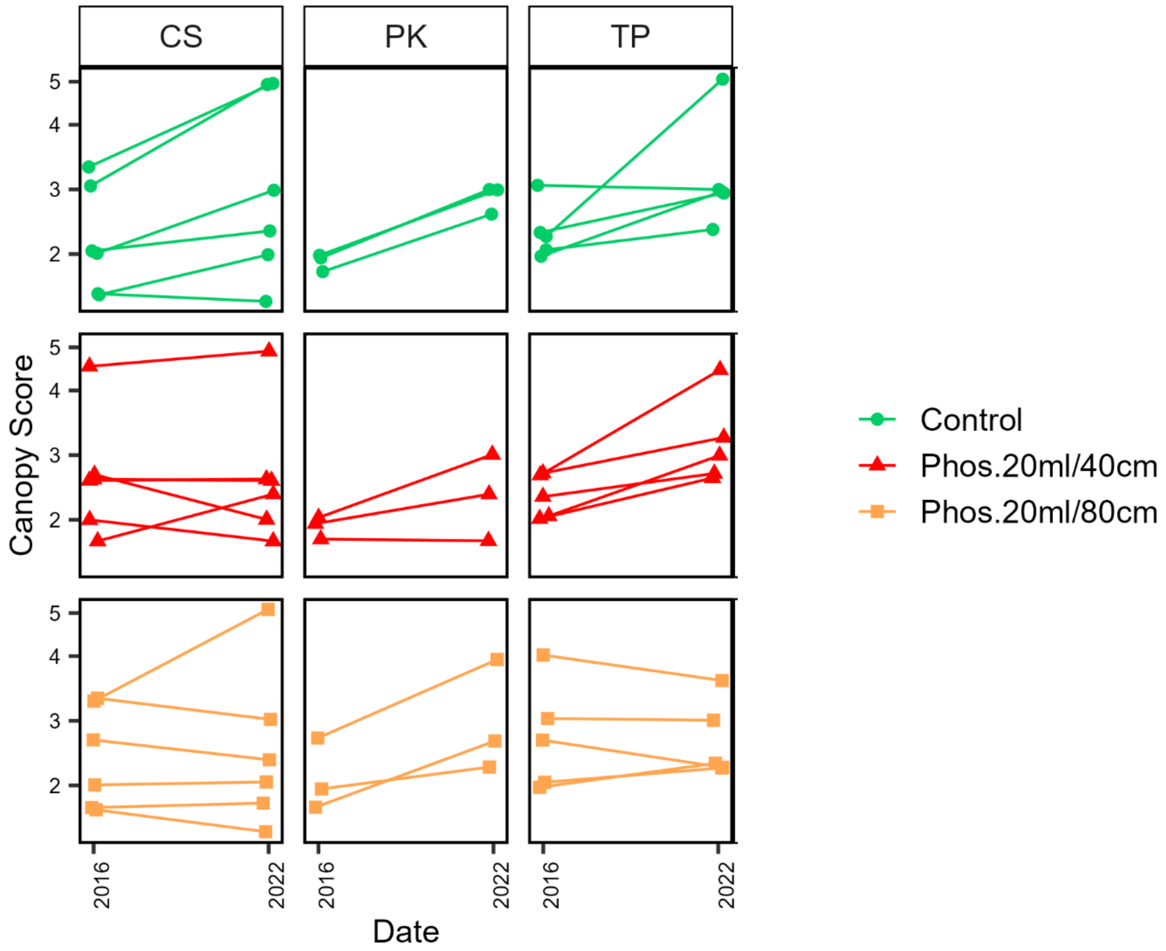


Figure 52. Canopy disease scores of all kauri trees used in the large-tree phosphite trial assessed pre-treatment (2016), and 5.5–6 years post-treatment (2022). The overall canopy health was scored on a modification of the 1–5 canopy health scoring scale adopted by the Kauri Dieback Programme (1 = healthy crown – no visible signs of dieback, 2 = foliage/canopy thinning, 3 = thinning and some branch dieback, 4 = severe dieback, 5 = dead). In 2016 a 4% phosphite solution was applied as one 20-mL injection every 40 cm or one injection every 80 cm around the trunk circumference. Sites were Cascades (CS), Puketotara (PK) and Trounson Park (TP).

Table 11. Number of trees with the same or improved canopy scores in assessments made 5.5–6 years post-treatment (2022), compared with pre-treatment assessments (2016). Trees were either injected with phosphite or left untreated.

Site	Control	Phosphite 20 mL/40 cm	Phosphite 20 mL/80 cm	Total
Cascades	1 / 6	4 / 6	5 / 6	10/18
Puketotara	0 / 3	1 / 3	0 / 3	1/9
Trounson	1 / 5	0 / 5	3 / 5	4/15
Total	2 / 14	5 / 14	8 / 14	

5.2.3.2 Photographic comparison

Individual and mean scores for canopy photographic comparisons are plotted in Figure 53. These data reflect comparisons of canopy health and vigour with those in photographs taken pre-treatment. Analysis of variance was used to compare photographic comparison scores in February 2021 by site and by treatment. On average, there was a decline in canopy health throughout the trial across all treatments (Figure 53, lower). As noted for shoot growth (below), there was an overall trend for slightly better canopy comparison scores in treated trees than in untreated control trees. However, neither site, treatment, nor their interaction, was statistically significant (p=0.918, 0.144 and 0.369 for site, treatment and interaction, respectively). The overall result was strongly influenced by trees that died, contributing to the average decline in tree health across treatments, and again, the plot for Puketotara is heavily influenced by one of the three replicate trees in the ‘4% injection every 80 cm’ treatment declining rapidly in health from the start of the trial. As can be seen from the upper graph in Figure 53, canopies of most trees across all treatments remained in similar health throughout the trial.

5.2.3.3 Shoot growth

Individual and mean scores for shoot growth are plotted in Figure 54. Analysis of variance was used to compare shoot growth in March 2022 by site and by treatment. Although there was an overall trend for slightly better growth in treated trees, neither site, treatment, nor their interaction was significant (p=0.324, 0.136 and 0.526 for site, treatment and interaction, respectively). The mean plot for Puketotara in Figure 54 (Middle) is heavily influenced by one of the three replicate trees in the ‘4% injection every 80 cm’ treatment declining rapidly in health from the start of the trial.

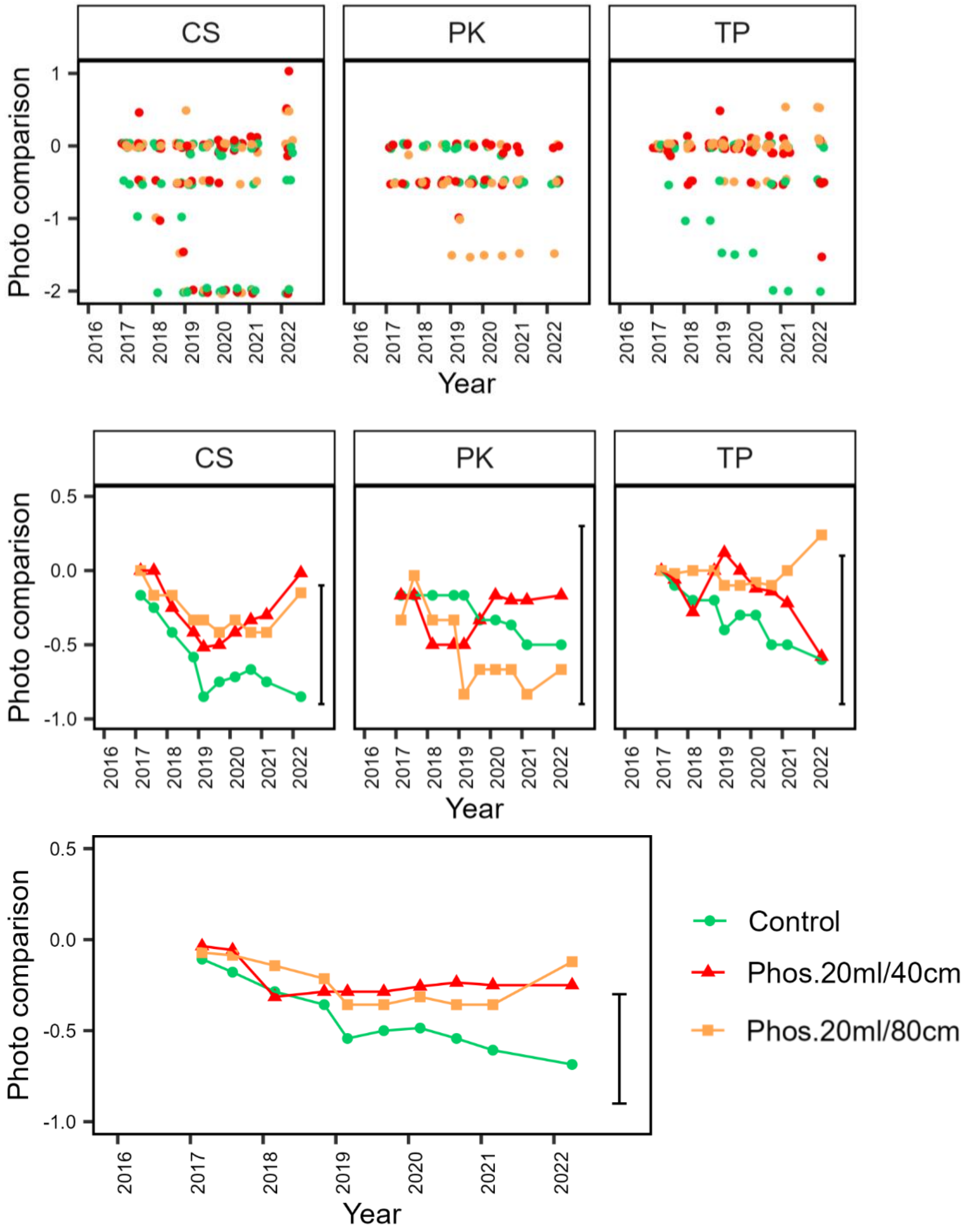


Figure 53. Canopy photographic comparison scores in *Phytophthora agathidicida*-infected kauri trees on three sites, assessed 6-monthly following phosphite injection treatment. Top: data for each tree at each site; Middle: mean data by treatment and site. Bottom: Mean data across all three sites. Phosphite injections were applied in March 2016 (Puketotara site, PK) or November 2016 (Cascades, CS and Trounson, TP sites). A 4% phosphite solution was applied as one 20-mL injection every 40 cm or one injection every 80 cm around the trunk circumference. Phosphite was re-applied to trees in the low dose treatment (4% injection every 80 cm) at the Puketotara site in March 2018 and Cascades plus Trounson sites in February/March 2019. The higher-dose phosphite treatment (4% injection every 40 cm) was re-applied at all sites in June 2019. Data were generated by comparing canopy health and density (from a fixed photographic point) with that in pre-treatment photographs using the following scale: -2 = tree dead, -1 = substantially worse, -0.5 = slightly worse, 0 = similar, 0.5 = slightly better, 1 = substantially better. Bars indicate the least significant difference (LSD, $p = 0.05$) for the final assessment (2022).

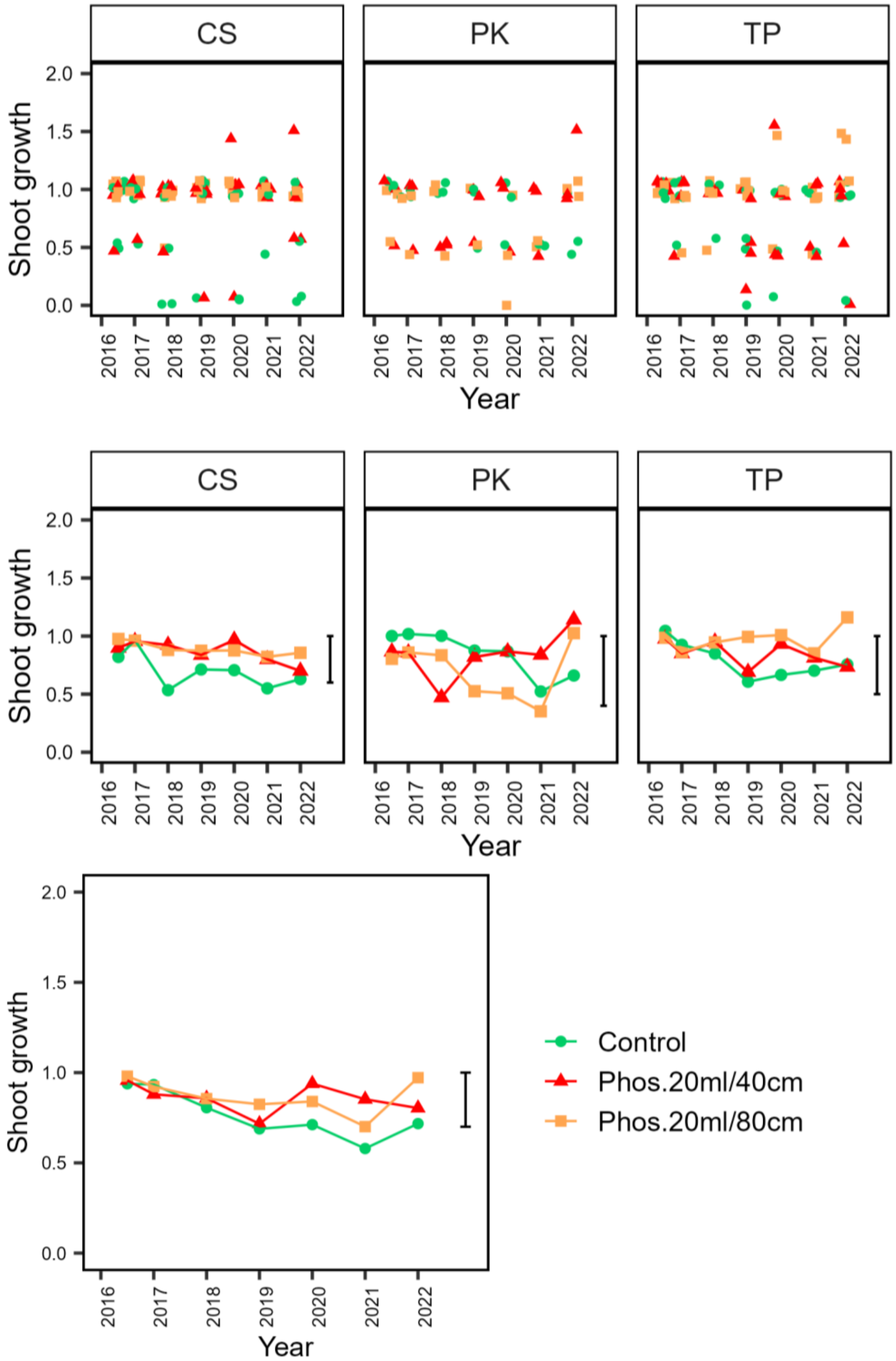


Figure 54. Shoot growth scores in *Phytophthora agathidicida*-infected kauri trees on three sites, assessed annually in February/March, following phosphite injection treatment. Top: individual tree data at each site; Middle: mean data by treatment and site. Bottom: Mean data across all three sites. Phosphite injections were applied in March 2016 (Puketotara site, PK) or November 2016 (Cascades, CS and Trounson, TP sites). A 4% phosphite solution was applied as one 20-mL injection every 40 cm or one injection every 80 cm around the trunk circumference. Phosphite was re-applied to trees in the low dose treatment (4% injection every 80 cm) at the Puketotara site in March 2018 and Cascades plus Trounson sites in February/March 2019. The higher-dose phosphite treatment (4% injection every 40 cm) was re-applied at all sites in June 2019. Shoot growth was recorded on a 0–2 scale, where 0 = no growth, 1 = good ('normal') growth and 2 = extremely vigorous growth. The bar indicates average least significant difference (p = 0.05) for the 2022 data.

5.2.4 Phytotoxicity

Five-and-a-half years after treatments were first applied at the Trounson and the Cascades sites, and 6 years after the initial Puketotara treatment, no signs of phytotoxicity in the canopy (e.g. leaf yellowing or sudden loss) had been noted at any stage. Minor trunk cracks or bleeds above injection points and possibly associated with them, were noted in two trees at Trounson and one tree at each of the Puketotara and Cascades sites. All these trees were in the ‘4% phosphite trunk injection, 20 mL every 40 cm’ treatment, and all such cracks/bleeds were dry and apparently healed in assessments made in February/March 2021 and March 2022.

5.2.5 Mortality

At the Cascades site, two out of six untreated control died, as did one of the six trees in each of the injected treatments (Table 12). One untreated control tree at Trounson steadily declined over the first 3 years then died. One treated tree (20 mL phosphite/80 cm spacing) at Puketotara showed substantial canopy decline over the first 3 years (from a ‘3-’ to a ‘4’ on the KDP canopy scoring scale) but stabilised in the 4- and 6-year assessments, with new growth in 2022. All trees that died had starting canopy scores of 3 or 4 (Figure 52). Analysis using a binomial generalised linear model on mortality data indicated no significant site or treatment differences (p = 0.23 and 0.49 respectively), and no significant site × treatment interaction (p = 0.90).

Table 12. Kauri tree mortality 5.5–6 years after phosphite treatment at three sites. Data are the numbers dead in 2022/numbers treated in 2016.

Site	Control	Phosphite 20 mL/40 cm	Phosphite 20 mL/80 cm
Cascades	2 / 6	1 / 6	1 / 6
Puketotara	0 / 3	0 / 3	0 / 3
Trounson	1 / 5	0 / 5	0 / 5
Total	3/14	1/14	1/14

5.3 Discussion – large tree trial

This trial has shown that phosphite has potential for treatment of large kauri trees infected by *P. agathidicida*, and for stimulating healing of lesions. There was a highly significant reduction in lesion activity in phosphite-injected trees, and slightly better canopy health scores in treated than in untreated trees. However, results indicate that a single application of the relatively low rates tested in this trial is insufficient to completely suppress *P. agathidicida* lesion growth in large trees (>50-cm diameter). The conservative and cautious approach with treating these big trees, to avoid phytotoxicity, stretched the lower limit too far. The highest rate used in this trial (20 mL of 4% phosphite injected every 40 cm) is substantially lower than the 20 mL of 7.5% or 20% phosphite injected every 20 cm in the earlier ricker trials (Section 3 above). While phytotoxicity symptoms seem to have been avoided with these low rates, and there has been significant lesion healing in most treated trees, the healing has not been complete. A second dose of the same low rates, 2–3 years after the initial treatment, improved the tree response, although a number of trees remained with active bleeds. At the 5- and 6-year assessments there appeared to be a slight increase in lesion activity in some trees, suggesting a waning of control.

Given that there have been no obvious phytotoxicity symptoms noted to date, higher dose rates or higher frequency of application should be considered (see plan for large tree trials, Section 8).

In the concurrent 'Trunk spray and low rate trial' (Section 4), a rate of 4% phosphite injected every 40 cm around the trunk was effective at stopping lesion activity in rickers (up to 40-cm diameter) (Horner et al. 2020b). This same dose (based on trunk circumference) in the current large-tree trial was not fully effective, suggesting that these large trees may need a higher dose to facilitate total lesion healing, and a different formula may be required for calculating dose (see Section 6.2). The trend towards poor control in the biggest trees would support this concept.

6 General discussion

6.1 Conclusions from across all trials

6.1.1 Efficacy

Overwhelming evidence from forest trials on 'ricker' sized kauri trees suggests that trunk injection with phosphite suppresses the activity of *P. agathidicida* within infected trees. The best evidence for this is the differential activity and spread of basal trunk lesions in phosphite-treated versus untreated trees. On phosphite treated trees at all four 2012 ricker trial sites and all three 2016 ricker trial sites, all lesions stopped expanding and appeared to heal. In most instances, the bark around the margin of lesions peeled back, revealing healthy bark beneath. In general, lesions in untreated control trees remained active and continued to expand, in some cases ringbarking trees.

Whether the treatment is sufficient to save trees already heavily infected and then ultimately restore them to good health will become more apparent in future years. However, there is a growing body of evidence from these trials indicating that many trees with declining canopies or aggressive basal trunk lesions can be turned around, and after an initial decline in canopy they can regrow and re-establish a healthy canopy.

6.1.2 Phytotoxicity

The potential phytotoxicity of phosphite was a major factor in early trials where high phosphite concentrations were used. The 20% phosphite concentration was certainly phytotoxic on kauri, with canopy thinning and bark-cracking symptoms, and accelerated demise of some trees. There was less evidence for significant phytotoxicity with the 7.5% phosphite concentration, although there was still some canopy yellowing/thinning and minor trunk symptoms in some trees on some sites. But in the majority of trials where 7.5% was injected, minimal phytotoxicity symptoms were observed. The trials established in 2016 testing the 4% phosphite concentration showed minimal phytotoxicity symptoms with this rate. This has more recently been backed up by Kauri Rescue Trust™ trials, where 4% and 6% concentrations were used, with no significant observations of phytotoxicity across a wide variety of sites (M. Barton pers. comm.). All trials to date on rickers suggest that phosphite at 4% is sufficient to stimulate lesion healing in *P. agathidicida*-infected kauri.

The phytotoxicity observed with the 20% rate of phosphite appeared to be correlated with pretreatment tree health. Trees with advanced canopy decline (3 or more on the KDP scale) were most likely to be detrimentally affected by phosphite, in some cases showing accelerated mortality. In contrast, healthy trees seem capable of recovering from the toxic effects of very high doses of phosphite, as demonstrated in the Waipoua pilot rate trial where uninfected healthy trees survived and re-grew following a 60% phosphite injection, although there was initially significant defoliation and premature branch abscission. The fact that trees with higher canopy disease scores have thinner canopies means that the phosphite could potentially be concentrated and therefore more phytotoxic. Reducing the dose on trees with high canopy disease scores could help lessen the phytotoxicity, though it could also potentially reduce disease control efficacy.

There also appeared to be a relationship between tree size and phytotoxicity, with most accelerated deaths occurring in the smallest trees. It is possible that the dose calculation based solely on the trunk circumference over-estimates the dose required for the smallest trees (see discussion below, Section

6.2). The phytotoxicity symptoms observed, particularly at the Huia and Whatipū sites in the 2012 trial, in part masked the very impressive disease control observed in all treated trees. The canopy thinning and thus higher canopy symptom score, and accelerated demise of a few severely infected trees following treatment, overshadowed the rapid and long-lasting lesion healing that occurred. Phosphite treatment prevented the expansion of *P. agathidicida* lesions in all treated trees in this trial (including those that subsequently died), demonstrating the potential to either protect trees from *P. agathidicida* or restore them to health. Any concerns about phytotoxicity must be put into perspective. The relatively low risk of enhanced decline of severely diseased trees must be balanced against the lesion healing and long-term improvement in health of the majority. The alternative is, almost inevitably, the death of most kauri trees in *P. agathidicida*-infected stands if no treatment is undertaken.

A current PhD research project is investigating physiological responses in kauri following treatment with phosphite (Arnet unpub.). This should give some insight into potential phytotoxicity effects.

6.1.3 Concentration/Dose

Phosphite concentrations (the percentage of phosphite active ingredient in the injection) of 20%, 7.5% and 4% have been investigated in various trials described in this report. Doses (number of injectors per unit of trunk circumference) of most formulations have been one 20 mL injector per 20 cm of trunk circumference, with the 4% concentration also investigated at the lower doses of one injector every 40 cm or 80 cm. In rickers, all these concentrations and doses (except 4% at 80 cm, which wasn't investigated on rickers) were effective at halting *P. agathidicida* lesion spread and stimulating bark flaking and healing around the lesion. As discussed earlier, the 20% concentration caused significant phytotoxicity and should be discarded as an option, but concentrations between 4% and 7.5% should be considered generally safe and effective. At 4%, even where spacing was increased to one injector every 40 cm, lesion healing was still apparent in all treated rickers. However, this lower dose was not fully effective on larger trees (>50 cm trunk diameter), with only partial healing of lesions and continued lesion activity in many trees. It is also possible that dose calculations based entirely on trunk circumference underestimate the dose required for very big trees (see discussion below, Section 6.2). To date, higher concentrations and doses have not been tested on large trees, but such trials are very important.

6.1.4 Tree size and treatment efficacy

Throughout the trials, there was a suspicion that phosphite at a given dose was not as effective on large trees as it was on rickers. The fact that the '4% at 40 cm' dose completely stopped lesions on all ricker trees in the 2016 trial, but the same dose failed to work completely in the large tree trial (even after a second treatment) would support this. We have already shown a trend within the large trees trial, with more lesion activity in larger, rather than smaller trees 6 years after initial treatment. In Figure 55 (top two graphs), data for the '4% phosphite at 40 cm' dose from both 2016 trials are combined, plotting lesion activity in 2022 against initial trunk girth. Although the trials were on different sites, they were in similar forests, set up at the same time, using the same dose and same techniques of treatment and assessment. Results show a slight trend for mean 2022 lesion activity ($r = 0.29$, $p = 0.156$), with a stronger trend when plotting maximum lesion activity for each tree ($r = 0.36$, $p = 0.075$). That is, lesion activity post-treatment was greater in larger than in smaller trees.

The main bias in this assessment is that the trees in the large tree trial were in fact treated twice (at the start and after 2–3 years), while in the other trial (i.e. most of the smaller trees), trees were only treated once at the start. If anything, this bias would suppress the trend noted above, as the largest trees in the comparison had two treatments. To overcome this bias, the comparison was repeated, using data collected 2 years post-treatment, before the second application was made (Figure 55, bottom two graphs). This analysis shows a strong trend, with significantly greater lesion activity post-treatment in the larger trees. For mean activity data in 2019, r= 0.43, p = 0.033, and for maximum lesion activity for each tree r = 0.49, p = 0.013.

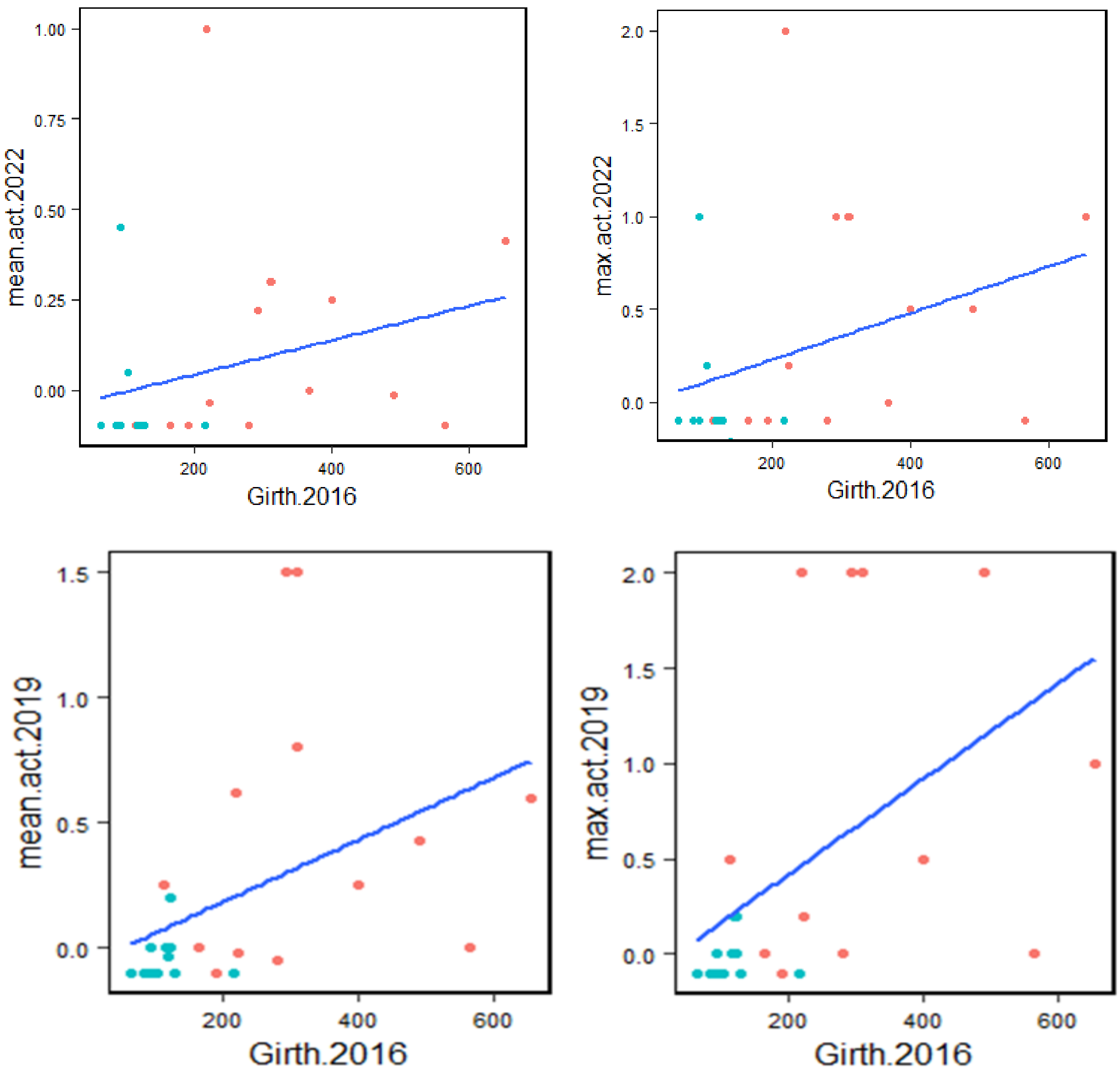


Figure 55. *Phytophthora agathidicida* lesion activity scores in plotted against trunk girth, in kauri trees injected with phosphite (4% injection, 20 mL every 80 cm) in 2016. The top two graphs are lesion data from 2022, and the lower two graphs are lesion data from 2019. Data points represent individual trees across six sites, using each tree's lesion activity score in 2022 versus tree girth (cm) measured pre-treatment. Blue dots are from trees in the low-dose trial (Section 4) and red dots are from the large tree trial (Section 5). Left hand graphs plot lesion activity score mean for all lesions on each tree, and the right hand graphs are the maximum lesion score for each tree (note the different scales). Phosphite was re-applied to trees in the large tree trial in June 2019 (after collection of the data in the lower two graphs). Lesion activity scoring: -0.1=healed and peeled back, 0=not active, 0.2=probably not active, 0.5=probably active, 1=active, 2=very active.

The trend is an interesting one and worthy of further consideration. It does suggest the large trees might require larger doses (based on circumference) than ricker trees. This gives us some confidence in at least directly scaling up from doses safe and effective for smaller trees (i.e. 4–7.5% phosphite at 40–20 cm spacings). If anything, this could still be under-dosing really big trees (>1 m diameter).

6.1.5 Longevity

The longevity of phosphite treatment efficacy and the required frequency of re-treatment for long-term control are yet to be determined. However, we are starting to see some useful trends to help make decisions. It seems that the concentration or dose will influence the duration of treatment effectiveness (Figures 56 and 57, Tables 13 and 14). In the extreme case, in the 2012 ricker trial, all trees treated with the 20% phosphite concentration remained free of active or semi-active basal trunk lesions for at least 10 years post-treatment. Similarly, in the same trial, 96% of trees treated with 7.5% phosphite remained free of active trunk lesions 10 years post-treatment. Further support of this observation comes from the 2016 low-rate ricker trial, where only one of the 12 trees (8%) treated with 7.5% phosphite had active or semi-active trunk lesions 6 years after treatment. Lower phosphite concentrations seem to have less longevity of effectiveness. In the same trial (2016 low-rate ricker), the effect of the 4% phosphite concentration did not appear to last as long as the 7.5% rate, with 25% of trees having at least one active or semi-active lesion after 6 years. However, this still compared favourably to untreated controls, which had 77% of trees with active lesions. These results align with observations made in Kauri Rescue Trust trials (Kauri Rescue Trust 2024), where new active lesions were noted in some trees treated with 4% phosphite after 3–5 years. The loss of control was less when 6% phosphite was used. In these same Kauri Rescue trials, the loss of control was much more noticeable where the lower dose (i.e. one injector every 40 cm) was used, although surprisingly this did not occur in our trials.

In the large tree trial where phosphite rates were obviously lower than optimal, after an initial decline in lesion activity following treatment, there was an increasing number of trees with active lesions in years 5 and 6, just 2–3 years after the second phosphite application. This further supports evidence that there is reduced longevity of control at lower phosphite rates.

It should be noted that new colonisation and lesion development is most likely to be initially subterranean and perhaps not noticed for a number of years, as lesions slowly extend along roots and to the lower trunk. Thus, by the time above-ground active lesions are noticed, there could potentially be substantial subterranean colonisation of roots. Therefore, the retreatment interval should be less than the time to observations of significant numbers of active above-ground lesions.

An interval of about 3 years between injections may be appropriate for treatments of 4% to 5% phosphite but potentially longer intervals may be possible if 6% to 7.5% concentrations are used. There is a trade-off between using higher rates to increase longevity of effectiveness, but potentially risking phytotoxicity if rates are too high, although phytotoxicity has been minimal at rates lower than 7.5%. Until long-term trials and observations testing various re-treatment regimes are done, there cannot be certainty in determining optimal intervals for retreatment.

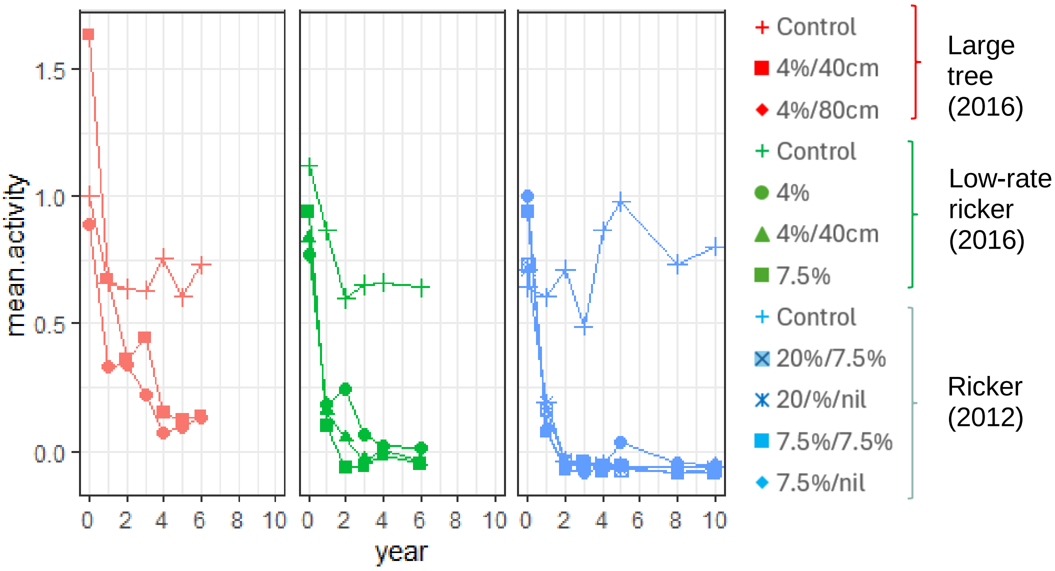


Figure 56. Summary of mean lesion activity scores across all trials testing trunk injection of phosphite in *Phytophthora agathidicida*-infected kauri trees. Data are means of lesion activity scores of all trees across all sites within each of three different trials (Large Tree trial 2016 = red, Low-rate Ricker trial 2016 = green, Ricker trial 2012 = blue. Year (Y) 0 is pre-treatment, other years are years post-treatment. In the Ricker 2012 trial, some treatments were re-applied after 1 year, and in the Large tree 2016 trial, treatments were re-applied between Y2 and Y3. Otherwise, all treatments were applied just once, at Y0. Percentage figures in the legend are the concentration of phosphite. All injections were at 20-cm spacings unless otherwise stated in the legend. Lesion activity was assessed as -0.1=healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active.

Table 13. Summary of mean lesion activity scores across all trials testing trunk injection of phosphite in *Phytophthora agathidicida*-infected kauri trees. Data are means of lesion activity scores of all trees across all sites within each of three different trials. Year (Y) 0 is pre-treatment, other years are years post-treatment. In the Ricker 2012 trial, some treatments were re-applied after 1 year, and in the Large tree 2016 trial, treatments were re-applied between Y2 and Y3. Otherwise, all treatments were applied just once, at Y0. Percentage figures are the concentration of phosphite. Injections were at 20-cm spacings unless otherwise stated in the treatment list. '-' = data not collected. Lesion activity was assessed as -0.1=healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active.

Trial	Treatment	Y0	Y1	Y2	Y3	Y4	Y5	Y6	Y8	Y10
Ricker. 2012, 4 sites.	Control (nil/nil)	0.65	0.60	0.71	0.49	0.87	0.98	-	0.73	0.80
	20%/7.5%	0.73	0.16	-0.05	-0.05	-0.05	-0.07	-	-0.08	-0.07
	20%/nil	0.71	0.19	-0.04	-0.05	-0.05	-0.06	-	-0.06	-0.06
	7.5%/7.5%	0.94	0.07	-0.07	-0.04	-0.08	-0.06	-	-0.08	-0.09
	7.5%/nil	1.00	0.09	-0.07	-0.08	-0.07	0.03	-	-0.05	-0.06
Low rate Ricker. 2016, 3 sites.	Control (nil)	1.12	0.87	0.60	0.65	0.66	-	0.64	-	-
	7.5%	0.94	0.10	-0.07	-0.06	-0.02	-	-0.05	-	-
	4%	0.77	0.18	0.24	0.07	0.02	-	0.01	-	-
	4%/40cm	0.84	0.16	0.05	-0.04	0.00	-	-0.04	-	-
Large tree. 2016, 3 sites.	Control (nil)	1.00	0.66	0.64	0.63	0.76	0.60	0.73	-	-
	4%/40cm	1.63	0.67	0.36	0.44	0.15	0.12	0.14	-	-
	4%/80cm	0.89	0.33	0.34	0.22	0.07	0.09	0.13	-	-

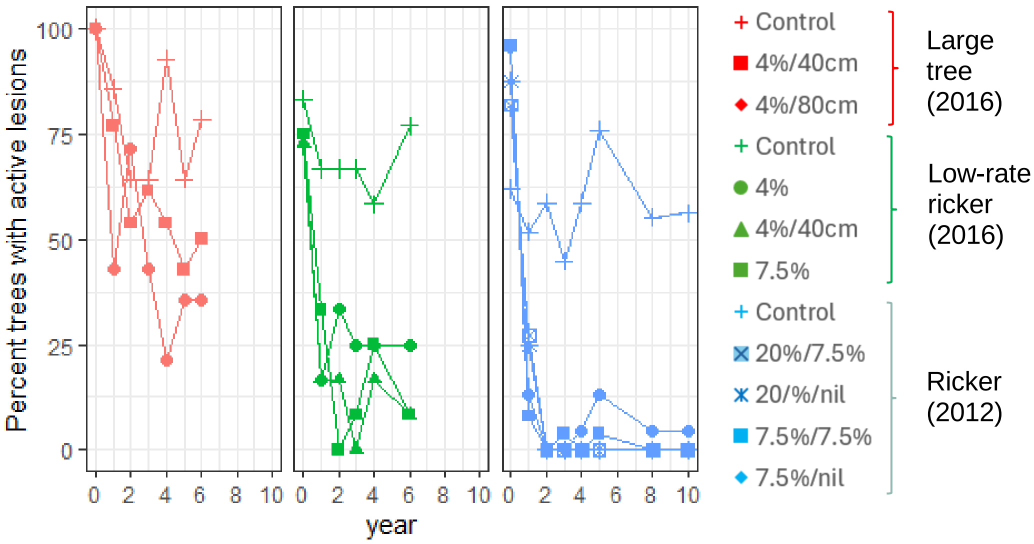


Figure 57. Summary of percentage of trees with active or semi-active basal lesions across all trials testing trunk injection of phosphite in *Phytophthora agathidicida*-infected kauri trees. Data are means of lesion activity scores of all trees across all sites within each of three different trials (Large Tree trial 2016 = red, Low-rate Ricker trial 2016 = green, Ricker trial 2012 = blue. Year (Y) 0 is pre-treatment, other years are years post-treatment. In the Ricker 2012 trial, two treatments were re-applied after 1 year, and in the Large tree 2016 trial, all treatments were re-applied between Y2 and Y3. Otherwise, all treatments were applied just once, at Y0. Percentage figures in the legend are the concentration of phosphite. All injections were at 20-cm spacings unless otherwise stated in the legend. Lesion activity was assessed as -0.1 = healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active.

Table 14. Summary of percentage of trees with active or semi-active basal lesions across all trials testing trunk injection of phosphite in *Phytophthora agathidicida*-infected kauri trees. Data are means of lesion activity scores of all trees across all sites within a trial. Year (Y) 0 is pre-treatment, other years are years post-treatment. In the Ricker 2012 trial, two treatments were re-applied after 1 year, and in the Large tree 2016 trial, all treatments were re-applied between Y2 and Y3. Otherwise, all treatments were applied just once, at Y0. '-' = data not collected. Lesion activity was assessed as -0.1 = healed and peeled back, 0 = not active, 0.2 = probably not active, 0.5 = probably active, 1 = active, 2 = very active.

Trial	Treatment	Y0	Y1	Y2	Y3	Y4	Y5	Y6	Y8	Y10
Ricker. 2012, 4 sites.	Control (nil/nil)	62%	52%	59%	45%	59%	76%	-	55%	56%
	20%/7.5%	82%	27%	0%	0%	0%	0%	-	0%	0%
	20%/nil	88%	25%	0%	0%	0%	0%	-	0%	0%
	7.5%/7.5%	96%	8%	0%	4%	0%	4%	-	0%	0%
	7.5%/nil	96%	13%	0%	0%	4%	13%	-	4%	4%
Spray & low rate. 2016, 3 sites.	Control (nil)	83%	67%	67%	67%	58%	-	77%	-	-
	7.5%	75%	33%	0%	8%	25%	-	8%	-	-
	4%	75%	17%	33%	25%	25%	-	25%	-	-
	4%/40cm	73%	17%	17%	0%	17%	-	8%	-	-
Large tree. 2016, 3 sites.	Control (nil)	100%	86%	64%	64%	93%	64%	79%	-	-
	4%/40cm	100%	77%	54%	62%	54%	43%	50%	-	-
	4%/80cm	100%	43%	71%	43%	21%	36%	36%	-	-

6.1.6 Timing of application

The optimal timing of phosphite application on kauri is not known. Most of the treatment applications in the trials discussed in this report were in the period from January to March, with no comparison of treatment dates. To our knowledge no such trials have been done. There may be some useful information with Kauri Rescue Trust files, as they have properties treated at various times throughout the year (except perhaps the wettest winter months). But the information would be largely anecdotal, as systematic comparative trials on the same property have not been done.

The only study we know of looking at injection timing was by Horner (2018), and that study was looking only at injection uptake time in different seasons, time of day and weather conditions. Few differences were noted, except for slightly quicker uptake time in the spring than in other seasons. These trials were on healthy trees using water injection, so there is no information available on efficacy.

6.1.7 Parameters – what measurements were most useful?

Several different tree health parameters were measured during these trials, with various degrees of usefulness. The canopy measures were not particularly useful for assessing short-term changes, especially with big trees. This is because of long lag times from potentially turning around disease development in the roots and lower trunk, to improved growth of the canopy. Improvements (if any) are slow, although declines can be rapid. Assessments of shoot growth were very subjective, and often difficult to carry out from the ground, especially with tall trees and poor background light conditions, although this measure was useful in showing an eventual turnaround in tree health in treated trees in the 2012 ricker trial. Similarly, canopy colour assessment was very subjective, very dependent on current light conditions, and variable from tree-to-tree. This measure was not used at all in final analyses. Scoring canopy health on the KDP 1–5 scale was again quite subjective, with subtle changes difficult to detect, and variable assessments by different people (or even the same person at different times). Allowing ‘plus’ or ‘minus’ scores (or half scores as done by Kauri Rescue) helps with discrimination and accuracy, and over time this assessment was useful at detecting large changes in tree health. The most accurate and least subjective canopy measure in the field was the photographic comparison, where current observations of a portion of the canopy were compared with a baseline photograph from a fixed point. This could detect relatively subtle changes in canopy health and density but was reliant on good photographs at the start and clearly marked photo points. It was also complicated by in-filling of the understorey over time, blocking the view. In general, canopy measures are likely to be more useful for assessing much longer-term treatment effects. In the future, aerial measurements of canopy spectral reflectance to calculate vegetation indices may allow for the detection subtle canopy changes which are less subjective.

Lesion assessments were probably the most accurate and useful measure of tree response to treatment. Lesion activity scoring was relatively straightforward, with tree responses measurable within a few months of treatment, and giving good discrimination of treatment effects. Methods of lesion assessment such as measuring the advance of marked lesions, used effectively in trials with ricker trees, were not as useful with the large trees. Measurements of lesion dimensions and advance of the lesion margin were often problematic with these large trees, in part because the lesion margins were often poorly defined, and there was sub-bark movement of lesion fronts, and periodic peeling of large sheets of bark. Lesion dimension measures gave poor discrimination between treatments with large trees.

Although lesion assessments generally proved very useful for discriminating between treatments, lesion scoring is limited to infected trees which have above ground trunk lesions. Infected kauri that do not yet have above ground lesions are difficult to assess for the change in health in response to phosphite treatment.

Another possible measure of tree health is the moss and lichen score. This measure assumes that the amount of moss and lichen on a kauri trunk reflects tree health, as infected trees with slower growth stop shedding bark and thus accumulate lichen, moss and other epiphytes on trunks. As tree health improves (e.g. following treatment), natural bark shedding increases, reducing the accumulation of bark epiphytes. Although not used in the trials discussed in this report, Kauri Rescue has started using the moss and lichen score in tree health assessments (Kauri Rescue 2024). It is potentially a useful long-term measure of changes in tree health.

6.2 Alternative phosphite dose calculation for kauri

Based on the results from these trials, there has been considerable discussion about an alternative dose calculation for large trees, not necessarily relying solely on trunk circumference (Horner & Arnet unpublished). A synopsis of those discussions is presented here, as it is very relevant to recommendations for treatment of large trees and for future large tree trials.

Key points:

- Evidence from the large tree trial – low rates and doses that worked on rickers were not sufficient on larger trees. The larger the tree, the less lesion healing.
- Current dose calculations based on circumference seem appropriate for mid-range rickers, i.e. (20–40 cm trunk diameter), but perhaps underestimate what is needed for larger trees.
- Evidence from the ricker trial – at higher phosphite concentrations, there was potentially overdosing of smaller trees more than larger trees within the trial, as evidenced by slightly higher mortality post-treatment (especially with the more diseased trees).

The current dose calculation is based on trunk circumference (e.g. one 20 mL injection every 20 cm), but is this appropriate? What if another measure of tree size was used to calculate dose? Possibilities include continuing with trunk circumference, using canopy area, volume, or leaf-area index, using trunk cross-sectional area, using sapwood cross-sectional area, using a combination of measures. Pros and cons of these are considered in Table 15.

We feel that sapwood area could be the best option for large trees. Exploring the sapwood concept a bit further, the graph below (Figure 58) compares doses for a range of tree sizes, calculated based on the trunk circumference versus sapwood area. The sapwood area for trees of different diameter was calculated from the empirical data of Karikala (2015), who showed that although sapwood thickness varied among trees, for kauri there was no clear increase in sapwood thickness with increasing tree size. Sapwood depth averaged approximately 10–12 cm over a wide range of tree sizes. The key assumption for calculating the curve for required sapwood dose (based on work in this report) was that the dose used on a 30–40 cm diameter tree is appropriate, as it was safe and effective. Thus, any cross-over point in the circumference line and the sapwood area curve should be at 30–40 cm diameter. The phosphite volume used at this tree size was then extrapolated to larger trees by keeping the ratio between volume of phosphite to unit of sapwood cross-sectional area constant.

Table 15. Consideration of various potential methods for determining required phosphite dose in large kauri trees.

Dose predictor	Pros	Cons
Trunk circumference	Easy to do, used for many tree species internationally. Has proven useful for rickers and advanced rickers.	Perhaps not appropriate for very big and very small trees. Large kauri are substantially bigger than most trees that have been phosphite-treated internationally
Canopy volume, area, or leaf area index	Probably an accurate estimate of the tree's capacity to safely absorb phosphite. A true canopy volume score would allow for trees with thinner canopies to get a lower dose. LiDAR could be used to calculate leaf area index accurately and repeatedly. Data are widely available in New Zealand to 1 m spatial resolution. Likely reflects root volume.	Difficult to estimate, often can't see clearly from the ground, highly variable, canopy thinning, much guesswork. LiDAR would require technical knowledge or the development of an intuitive software for end users. Maybe only useful for research purposes.
Trunk cross-sectional area	Accurate estimate of tree's mass, easy to calculate.	Exponential increase in dose as tree size increases. Most of the trunk volume (heartwood) is probably inert, not transporting sap. Would probably over-estimate the number of phosphite injections required.
Sapwood cross-sectional area	Relatively easy to estimate. Related to circumference and trunk cross-sectional area, but could better reflect the in-tree dilution of phosphite to include where it might be dispersed. Empirical sapwood data have been collected for kauri, making it easy to estimate from trunk diameter measurements (Karikala 2015).	Unknown if sapwood area is an appropriate estimate for the functional tree volume, and thus the amount of phosphite to be injected into a large tree. Using 20 ml injections could result in injections closer than every 20 cm for large trees
Root volume	This is where much of the phosphite is likely transported and needed.	Very difficult to estimate. No empirical data. Probably reflects canopy volume, so that would be a better measure.
Complex combination of above	Could be the most effective way to calculate safe and effective doses of phosphite. Includes all meristematic tissue to where phosphite will be preferentially transported.	Too difficult for landowners (and probably most others) to calculate themselves without further extensive research.

The sapwood cross-sectional area curve in Figure 58 shows that if using circumference there is potentially an underestimation of doses required for really big trees, with the underestimation increasing with increasing tree size.

If sapwood is indeed the best predictor of required dose, the graph in Figure 58 (zoomed in inset) suggests there is potentially a slight overdosing of small trees (less than about 20 cm diameter). This is consistent with the slight trend observed towards more phytotoxicity in the smallest trees in the 2012 ricker trial with high concentrations of phosphite.

Currently, this discussion is mainly speculation, but based on hints from the trials covered in this report. We present it here to stimulate further discussion and to look at another way of calculating dose. One thing it suggests is that it should be safe to scale up to higher doses than have previously been used in big trees without a significant risk of adverse effects. For now, we suggest carrying on with circumference as the estimator of dose (i.e. number of injectors) while being aware that this is potentially underestimating what is required for big trees. Once higher doses on large trees have been tested, there will be useful empirical data to determine if a variation of sapwood area would be a better estimate of dose, or whether circumference should remain as the main determinant.

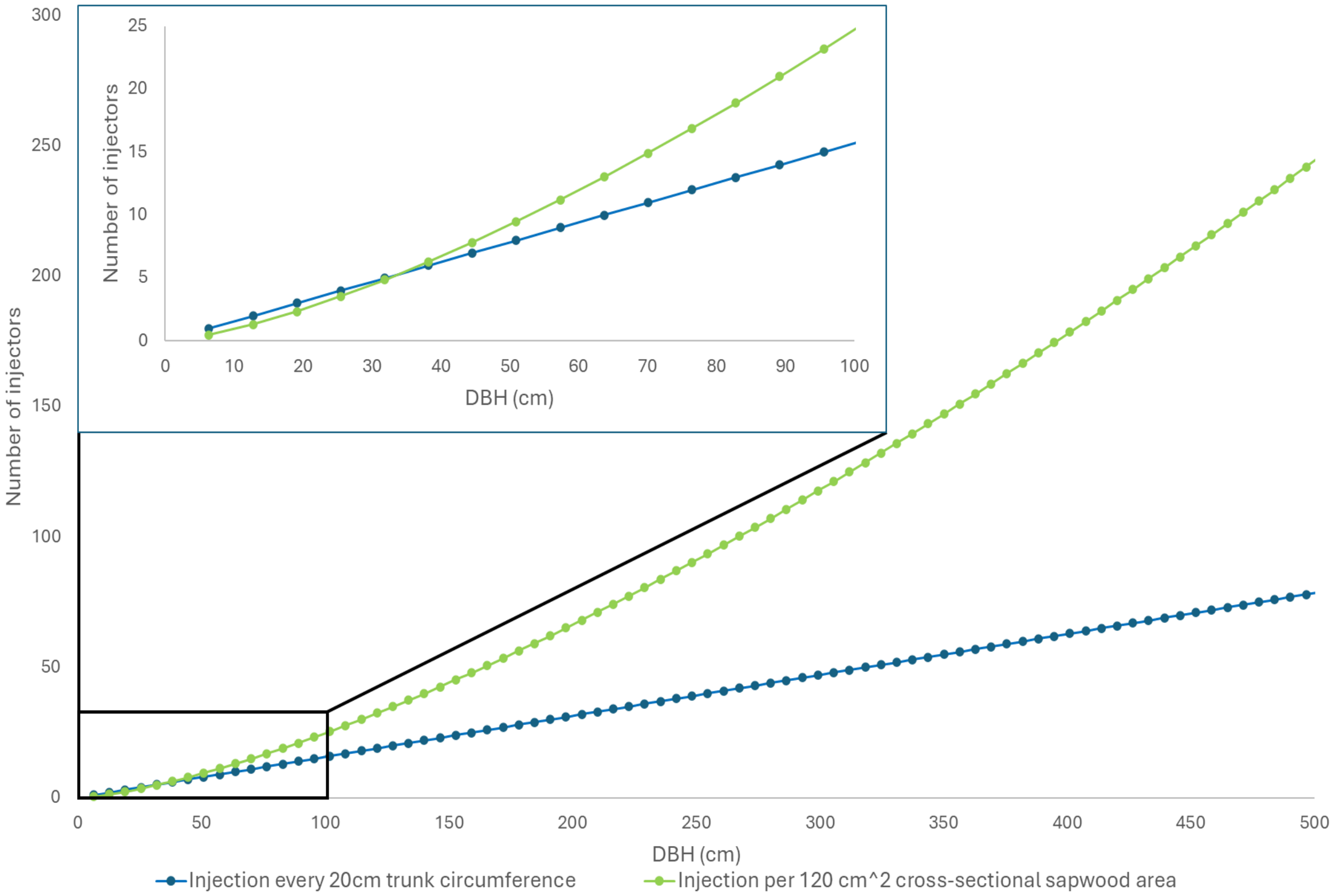


Figure 58. Phosphite dose for kauri trees at 20-cm spacings based on trunk circumference (blue) and dose every 120 cm^2 of cross-sectional sapwood area (green). The cross-over point for the two methods of dosing was set to be at 30–40 cm DBH (trunk diameter at breast height), a size where treatments are known to be safe and effective, and values for dose prediction extrapolated for smaller and larger trees. The insert on top left zooms in on the kauri ranging from 6–100 cm DBH.

7 Unknowns and future work

Trials with phosphite have provided sufficient information for confidence in treating kauri trees infected or threatened by *P. agathidicida.* But there are still questions that need to be addressed, building on the results and observations to date. There is confidence in concentrations and doses required for effective treatment of rickers. However, for large trees there is insufficient evidence for appropriate rates. The only trials to date indicated that 20 mL of 4% phosphite injected every 40 cm around trunks is partially effective but insufficient to completely heal trunk lesions on many large trees. Both higher phosphite concentrations and higher doses or dose volumes need to be investigated (see Section 8), or possibly repeated application of lower doses.

There is also a need to determine the longevity of treatment effects and appropriate regimes for repeated phosphite applications, and how this might relate to phosphite rates applied. This was discussed in Section 6.1.5, but there can only be confidence in retreatment regimes if variable retreatment programmes are established in a systematic and controlled way and observed in the long term. The Kauri Rescue programme is starting to look at retreatment of some trees, and their fate will be followed in the future. Although the retreatment is not being done in a systematic way, observational data coming from this programme will be useful in the future.

It is unknown whether roots are healed or protected by trunk injection. Although it is assumed that following trunk injection, root lesions will heal, and feeder roots will have some protection from *P. agathidicida* attack, this has not been directly investigated.

Assuming roots have some protection from colonisation or lesion development following phosphite injection, it is still not known how soon after treatment recolonisation of roots occurs. Inoculum will remain in the soil and in plant debris, and therefore remain available for infection, even though inoculum levels may be reduced in treated areas. Current assumptions are that it may be a few years before re-establishment of the pathogen in the root system, but there is no direct evidence to back this up.

There is also a need to investigate differential dose rates relating to canopy density rather than just tree girth. To avoid phytotoxicity, trees with significantly reduced canopy, as a result of dieback or other causes, may require lower doses than would otherwise be calculated by trunk girth alone. Some evidence from previous trials suggests that trees with thinner canopies are more likely to decline following treatment, but it is unclear whether that is reflecting increased phytotoxicity because of reduced canopy volume, or simply that the trees were more severely infected. Remote sensing including LiDAR data may provide a more accurate way to appropriately dose kauri trees with a declining canopy.

It is unknown whether there is an optimal time or season for trunk injection. Does the seasonal timing of treatment make a difference to either efficacy or phytotoxicity responses? Sap movement in many species changes with season and could potentially influence phosphite uptake and redistribution. There is no evidence for this, but it has not been systematically investigated.

The effect of site factors such as soil type, nutrition, and microclimate on phosphite efficacy or phytotoxicity is not known. To date, the treatment has proven effective on a range of soils and sites.

Deployment of phosphite as a preventative or barrier treatment (rather than solely as a curative treatment for already-diseased trees) has the potential to substantially reduce spread and contain the pathogen on some sites, but multiple factors need to be considered and systematic research is required. Related to this could be treatment of species other than kauri, which could be harbouring or proliferating the pathogen without overtly showing disease symptoms.

Given the large amount of work that has gone into establishing and maintaining the trials discussed in this report, there is merit in ongoing monitoring of these trees by doing brief disease assessments periodically (e.g. 2–3 yearly). Such assessments might give more insight into very long-term effects, plus potentially give options for retreatment programmes.

8 New large tree trials

Background:

The 2016 large tree trials discussed in this report were established to determine the efficacy of phosphite to control kauri dieback in trees greater than 50 cm trunk diameter. The aims were to find safe and effective dose rates, and to determine if rates effective on smaller trees could be scaled up to larger trees. Trials were on three *Phytophthora agathidicida*-infected sites: Cascades (Waitākere), Puketotara (near Ōkaihau) and Trounson Kauri Park. Phosphite rates selected were relatively low (a 4% phosphite trunk injection, 20 mL every 40 cm around the trunk circumference; or a 4% phosphite trunk injection, 20 mL every 80 cm around the trunk circumference). These conservative rates reflected a desire to avoid the phytotoxicity symptoms that had been observed in some earlier trials with high rates. However, results showed that the injection rates were too low. Although there was healing or partial healing of lesions in most treated trees, a significant number of trees still had active trunk lesions in assessments made in 2020 and 2022, despite reapplication of all treatments in 2018.

Rationale/considerations for further trials:

- 4% phosphite with 20 mL injected at 40 cm spacings around the trunk resulted in only partial lesion healing in large trees. This relatively low rate was more effective in trials with rickers, suggesting that higher rates might be required for big trees, and that with low doses direct scaling up using circumference might underestimate doses required for really big trees.
- Scaling up directly from circumference calculations in ricker trials suggest that higher doses than those previously tested could be used safely.
- Calculations based on cross-sectional sapwood area suggest that, if anything, circumference-based calculations might underestimate the doses required for bigger trees, and that scaling up from rates determined in smaller trees should be safe and probably effective (see Section 6.2).
- 6% and 7.5% phosphite at 20 or 25 cm spacings have proven effective and safe on rickers in Kauri Rescue and The New Zealand Institute for Plant and Food Research Ltd trials. Scaling these rates up to larger trees seems reasonable. However, rates higher than 4% at 40 cm spacings have not yet been trialled on large kauri trees, so this work is highly experimental and should proceed with caution, using a conservative approach.
- Repeat applications could be used, if needed
- The relationship between canopy volume/leaf area index and tree size (and therefore optimal dose) should be considered, or at least estimated in future trials.
- For now, tree circumference should continue to be used for calculating the higher doses required, but results may provide information for an improved model in the future.

Treatment options:

The number of treatment options should be minimised to avoid over-complicating any trials. Suggested treatments for consideration are:

- 4% phosphite trunk injection, 20 mL every 40 cm around the trunk circumference (comparable to highest rate in the previous trial)
- 6% phosphite trunk injection, 20 mL every 40 cm around the trunk circumference
- 6% phosphite trunk injection, 20 mL every 30 cm around the trunk circumference (equivalent to 20 mL of 4% phosphite every 20 cm of trunk circumference)
- 6% phosphite trunk injection, 20 mL every 20 cm around the trunk circumference
- Untreated control.

Inclusion of an untreated control treatment needs to be considered carefully. Although from a scientific research viewpoint it would be desirable to have an untreated control treatment, the ethics of this need to be considered. As in human medical trials, once it is known that a treatment is beneficial, it is unethical to withhold it from any of the trial subjects. Observations over the past 20 years suggest that most infected trees will decline and die. The real comparison required in future trials is between different rates of application (both concentration and dose), to see which, if any, provide complete healing of lesions, and which have the greatest longevity of any apparent control. We have shown in previous trials that 4%/40 cm is partially but not always fully effective on large trees, so this is probably an acceptable low-rate option for comparison – we are not leaving big trees to an inevitable decline and death and have the option of giving a second dose in the future. This repeated treatment at a lower rate might prove a useful long-term regime.

Repeat treatments across the trial after 2–3 years should be considered, but a decision on this should not be made until at least 18 months after the initial application, and healing to date and phytotoxicity symptoms (if any) are taken into consideration.

There should be a minimum of 12 replicate trees in each treatment, though many more than this would be preferable given the range of situations and tree variability anticipated. Assignment of treatments to individual trees needs to be carefully and systematically done to minimise bias – simple random allocation in a non-structured way will not be optimal.

Another consideration could be to apply further treatments to the trial trees in the 2016 trial. It is now 5 years since their last treatment, and many are in decline. Retreatment of these trees could give some insight into benefits from repeated low-dose treatment.

Potential trial sites:

Logistically, it would be preferable to have trial trees spread over just two or three sites, although it is more important to ensure suitable and sufficient trees are selected, regardless of the site. Consultation with appropriate local authorities, including iwi, councils, DOC etc., is the necessary early step in site selection. A few potential trial sites and trees have been considered, with three sites standing out as possibilities:

Cascades -Te Wao Nui ā Tiriwa/Waitākere Ranges (Te Kawerau A Maki, Auckland Council). This area has long-established areas of kauri dieback, with hundreds of trees affected, many with 0.5–2.5 m trunk diameter. Phosphite has already been used on selected trees in the area, and both Te Kawerau A Maki and Auckland Council are supportive of further treatment and trial work.

Waipoua – (Te Roroa, DOC). There are many stands of very big trees in Waipoua that are threatened by kauri dieback. Te Roroa have already phosphite-treated selected trees in a couple of areas and are supportive of further treatment and study of appropriate doses for really big trees.

Trounson Kauri Park – (Te Roroa, DOC). This is the site of one of the previous large tree trials, but still has many large symptomatic trees that have not been treated, mostly within a well-defined and easily accessible area. Te Roroa are interested in a treatment programme to try to save infected trees and reduce spread of the pathogen.

Existing trials:

Two trials have been recently established looking at phosphite treatment of large kauri trees. The first is around the Lower Te Piringa Track area at the Cascades. The Kauri Rescue Trust, in collaboration with Te Kawerau A Maki and Auckland Council have treated more than 50 large, threatened kauri trees, ranging in size from 0.6 to 2.5 m trunk diameter. The four injection regimes suggested above have been applied randomly to trees. Although there is not yet a plan or funding for ongoing assessments, baseline measurements have been taken, allowing future comparisons of treatment efficacy. This treatment programme/trial is an early part of a planned wider treatment roll-out in the area and could be used as a pilot for future trials.

The second current trial is part of Matthew Arnet's PhD thesis research, looking at physiological responses of kauri to phosphite treatment. In Waipoua forest, 24 kauri between 1.0 and 2.0 m trunk diameter (DBH), 12 infected by *P. agathidicida* and 12 not infected, will be treated or not with 6% phosphite at 20 cm spacings (our highest rate suggested above). Physiological responses, including sap flow and water storage following injection (planned for June 2024) will be monitored for at least 1 year. This trial could potentially be monitored in the long term (if resources were available), providing useful data on treatment of large trees with a phosphite rate three times higher than rates previously tested.

Timeline:

Work on new, systematic trials could potentially commence in spring/summer 2024/25, with final tree selection and baseline monitoring in spring and first treatments applied over summer.

Monitoring of canopy health, lesion activity etc. should be 6-monthly for the first 2 years, then annually for at least 5 years, preferably much longer.

Re-application of treatments should be considered after 18 months to 2 years, depending on the amount of lesion healing observed in assessments at that time. Throughout the trial, retreatment should be considered as necessary, based on lesion expression and overall tree health. Ideally, if there are sufficient trees in the trial, trees could be randomly re-treated or not, to evaluate different long-term management regimes.

Key Milestones:

November 2024 – Finalise trial trees and treatment selection
March 2025 – Complete initial treatment application
Thereafter, 6-monthly assessments and reporting (winter and summer).

9 Recommendations for phosphite treatment of kauri

These recommendations are based on results obtained from the trials established in 2012 and 2016 discussed in this report, with some added insight from observations from Kauri Rescue Trust's work. Recommendations are likely to be modified as more information becomes available. The trials summarised in this report demonstrate that phosphite is definitely effective at suppressing *P. agathidicida* activity in kauri trees and is an effective and safe control tool. The phytotoxicity observed at some sites in the 2012 ricker trial demonstrated that very high concentrations (e.g. 20%) should not be used. Concentrations of 7.5% and lower generally avoided significant phytotoxicity symptoms, and should be considered safe, although this could still be a concern on highly symptomatic trees. Only trees showing symptoms of kauri dieback, or those adjacent to infected trees, should be treated. A basic summary of recommendations follows. More detail is provided in the "Phosphite Treatment by Trunk Injection Guide" (Horner 2024).

Basic premise:

Phosphite has the potential to prevent *P. agathidicida* infections establishing and spreading within kauri trees.

Phosphite doses must be sufficient to suppress the pathogen and/or stimulate tree defence responses.

If phosphite doses are too high, phytotoxicity may occur.

If phosphite dose is too low, it may not be sufficient to suppress lesion spread, or may provide only short-lived control.

A balance must be struck to find doses suppressive to disease development, yet safe for the tree.

Trees with advanced infections are likely to be more sensitive to phosphite, and thus more likely to decline rapidly or show phytotoxicity symptoms following treatment.

Recommended treatment regime:

For rickers, up to about 70 cm DBH

Phosphite concentration: Phosphite at 4–6% active ingredient, potentially up to 7.5%, is recommended.

Application rate: (i.e. injector spacing) A 20-mL injector every 20–40 cm of trunk circumference.

Higher phosphite concentrations and lower injector spacings (within the recommended range), are likely to provide the best control and longevity of effect.

Timing: To date there is no evidence that any particular season or time of day is better or worse than any other for applying phosphite.

Frequency: No more than once every 3 years (for rickers), unless very low doses are used.

For Large trees**:** There is still no good empirical data on effective doses for treatment of large trees. Injection of 20 mL of 4% phosphite every 40 or 80 cm is too low for effective and lasting control, but higher concentrations and doses have not yet been tested on big trees. However, evidence suggests that the range of rates listed above for rickers should be safe, and if anything may be less than what is required for complete lesion healing on very big trees. Until more empirical data are available for very large trees, close follow-up monitoring should be carried out wherever higher concentrations and doses are used.

Tree health: Lower rates are recommended for trees with advanced symptoms or those with a very sparse canopy, by either reducing the concentration or giving a lower dose (i.e. wider spacing between injectors).

Deployment: Phosphite should be used on trees showing early stages of infection, and ideally on trees immediately threatened (adjacent to diseased trees) but not yet showing above-ground symptoms. Trees showing advanced symptoms could still be treated, as some could potentially be saved. But there is also the chance of accelerating the demise of trees with very advanced symptoms.

In the long term, deployment of phosphite as a barrier treatment should be considered. By treating a band of trees in advance of the disease front, there is the potential not only to protect those treated trees, but also to slow down the natural spread of the pathogen. Barrier application of phosphite has been successfully deployed in *P. cinnamomi*-infected natural forest in Western Australia (Shearer et al. 2004; Glenn Tuffnell pers. comm.). The potential for deployment of phosphite barrier treatment in kauri forests has been reviewed by Horner (2016).

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11 References

Bader MK-F, Leuzinger S 2019. Hydraulic coupling of a leafless kauri tree remnant to conspecific hosts. iScience 19: 1238–1243.

Beever RE, Waipara NW, Ramsfield TD, Dick MA, Horner IJ 2009. Kauri (*Agathis australis*) under threat from *Phytophthora*? In: Phytophthoras in Forests and Natural Ecosystems. Proceedings of the Fourth Meeting of IUFRO Working Party S07.02.09. General Technical report PSW-GTR-221, USDA Forest Service, Albany, CA, USA. Pp. 74–85.

Crane CE, Shearer BL 2014. Comparison of phosphite application methods for control of *Phytophthora cinnamomi* in threatened communities. Australian Plant Pathology 43: 143–149.

Ecroyd CE 1982. Biological flora of New Zealand 8. *Agathis australis* (D. Don) Lindl. (Araucariaceae) Kauri. New Zealand Journal of Botany 20: 17–36.

Erwin DC, Ribeiro OK 1996. Phytophthora diseases worldwide. Phytophthora diseases worldwide. APS Press, The American Phytopathological Society: 562 pp.

Garbelotto M, Schmidt DJ, Harnik TY 2007. Phosphite injections and bark application of phosphite + Pentrabark™: control sudden oak death in coast live oak. Arboriculture & Urban Forestry 33(5): 309–317.

Hillary J 1944. The vegetation of the Waitākere Ranges. Bulletin of the Wellington Botanical Society 9: 7–8. Cited in Steward GA and Beveridge AE 2010. A review of New Zealand kauri (*Agathis australis* (D.Don) Lindl.): its ecology, history, growth and potential for management for timber. New Zealand Journal of Forestry Science 40: 33–59.

Horner IJ, Arnet MA 2024. Best Practices Guide for Phosphite Injection of Kauri. A Plant & Food Research report prepared for: MPI/Tiakina Kauri. Milestone No. 98630. Contract No. 41623. Job code: P/383003/01. PFR SPTS No. 25848.

Horner I, Carroll E, Arnet M, Hedderley D, Horner M 2020a. Phosphite for control of kauri dieback: ricker trials, 8-year report. A Plant & Food Research report prepared for: Auckland Council. Milestone No. 88379. Contract No. 38248. Job code: P/345171/01. PFR SPTS No. 20159. December 2020

Horner IJ, Arnet MJ, Carroll EG, Horner MB 2021. Large-tree phosphite treatment trial for kauri dieback control. A Plant & Food Research report prepared for: Ministry for Primary Industries. Milestone No. 66666. Contract No. 33447. Job code: P/345160/05. PFR SPTS No. 21642.

Horner I, Arnet M, Horner M 2020b. Trunk sprays and lower phosphite injection rates for kauri dieback control – year 4. A Plant & Food Research report prepared for: Auckland Council. Milestone No. 87476. Contract No. 38244. Job code: P/345172/01. PFR SPTS No. 19916.

Horner I 2020. Phosphite treatment by trunk injection in kauri. A Plant & Food Research report prepared for: Ministry for Primary Industries. Milestone No. 84468. Contract No. 37705. Job code: P/345168/01. PFR SPTS No. 19579.

Horner I, Arnet M 2019. Phosphite large tree treatment trials: brief report August 2019. A Plant & Food Research report prepared for: Ministry for Primary Industries. Milestone No. 66662. Contract No. 33447. Job code: P/345160/05. SPTS No. 18505.

Horner I 2018. Phosphite toxicity and impact: an investigation of seasonal variability in injection uptake. A Plant & Food Research report prepared for: The Ministry for Primary Industries. Milestone No. 75505. Contract No. 33010. Job code: P/345160/01. SPTS No. 17188.

Horner I 2016. Phosphite Barriers for Kauri Dieback – Scoping Exercise. A Plant & Food Research report prepared for The Ministry for Primary Industries. Milestone No. 66367. Contract No. 66379. Job code: P/345160/03. SPTS No. 13757.

Horner IJ, Hough EG, Horner MB 2015. Forest efficacy trials on phosphite for control of kauri dieback. New Zealand Plant Protection 68: 7–12.

Horner IJ, Hough EG 2013. Phosphorous acid for controlling *Phytophthora* taxon Agathis in kauri: glasshouse trials. New Zealand Plant Protection 66: 242-248.

Horner IJ, Hough EG 2011. Phosphorous acid for controlling *Phytophthora* taxon Agathis in kauri. PFR Client report prepared for MAF Biosecurity. PFR SPTS No. 5802.

Karikala C 2015. Sapwood area and density of temperate kauri forest species. Bachelor of Science (Honours) dissertation, Biological Sciences, University of Auckland.

Kauri Rescue Trust 2024. Kauri Rescue Citizen Science Evaluation of Kauri Dieback Treatment Tools. Report to Ngā Rākau Taketake, New Zealand Biological Heritage National Science Challenge. www.kaurirescue.org.nz

Marin MV, Baggio JS, Melo PP, Peres NA 2023. Phosphite is more effective against Phytophthora Crown Rot and Leather Rot caused by *Phytophthora cactorum* than *P. nicotianae*. Plant Disease 107: 1602-1608

Shearer BL, Crane CE, Fairman RG 2004. Phosphite reduces disease extension of a *Phytophthora cinnamomi* front in Banksia woodland, even after fire. Australian Plant Pathology 33: 249–254. doi:10.1071/ap04002

Smillie RH, Grant BR, Guest D 1989. The mode of action of phosphite: evidence for both direct and indirect modes of action on three *Phytophthora* spp. in plants. Phytopathology 79: 921–926

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