

PFR SPTS No. 19579

Phosphite treatment by trunk injection in kauri

Horner I

June 2020



Confidential report for:

Confidential report for:

Ministry for Primary Industries

DISCLAIMER

The New Zealand Institute for Plant and Food Research Limited does not give any prediction, warranty or assurance in relation to the accuracy of or fitness for any particular use or application of, any information or scientific or other result contained in this report. Neither The New Zealand Institute for Plant and Food Research Limited nor any of its employees, students, contractors, subcontractors or agents shall be liable for any cost (including legal costs), claim, liability, loss, damage, injury or the like, which may be suffered or incurred as a direct or indirect result of the reliance by any person on any information contained in this report.

LIMITED PROTECTION

This report may be reproduced in full, but not in part, without the prior written permission of The New Zealand Institute for Plant and Food Research Limited. To request permission to reproduce the report in part, write to: The Science Publication Office, The New Zealand Institute for Plant and Food Research Limited – Postal Address: Private Bag 92169, Victoria Street West, Auckland 1142, New Zealand; Email: SPO-Team@plantandfood.co.nz.

CONFIDENTIALITY

This report contains valuable information in relation to the Phosphite treatment by trunk injection in Kauri programme that is confidential to the business of The New Zealand Institute for Plant and Food Research Limited and Ministry for Primary Industries. This report is provided solely for the purpose of advising on the progress of the Phosphite treatment by trunk injection in Kauri programme, and the information it contains should be treated as "Confidential Information" in accordance with The New Zealand Institute for Plant and Food Research Limited's Agreement with Ministry for Primary Industries.

PUBLICATION DATA

Horner I. June 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.

Report prepared by:

Ian Horner
Team Leader, Pathogen Biology & Ecology
June 2020

Report approved by:

Suvi Viljanen
General Manager Science – Bioprotection
June 2020

Contents

1	Introduction	1
1.1	Scope of this document.....	1
1.2	Kauri dieback background.....	1
1.3	What is phosphite?.....	1
1.4	What is the mode of action of phosphite?	2
1.5	Environmental impacts of phosphite	2
2	Kauri dieback/phosphite research results to date	4
2.1	<i>In vitro</i> trials of phosphite sensitivity	4
2.1.1	Glasshouse trials	4
2.1.2	Ricker trials.....	5
2.1.3	Trunk spray and low rate trials	6
2.1.4	Large trees	7
2.1.5	Timing trial.....	7
2.1.6	Kauri Rescue	8
2.1.7	Conclusions/general observations from trials to date	8
3	Research uncertainties/assumptions	10
4	Decision framework.....	13
4.1	Site selection	13
4.2	Tree selection.....	13
4.3	Deployment	13
4.4	Phosphite concentration and dose.....	15
5	Operational specification – Injection process	16
5.1	Equipment and preparation.....	16
5.2	Mixing of phosphite solution.....	16
5.3	Injection procedures.....	17
5.4	Clean-up.....	19
5.5	Safety	19
6	Monitoring.....	20
7	Hygiene requirements	24
8	Glossary of terms	25

9	Acknowledgements	25
10	References.....	26

1 Introduction

1.1 Scope of this document

The purpose of this document is to provide background information on phosphite, potential for treatment of kauri trees threatened by kauri dieback, research gaps and general guidelines on treatment and decision-making processes. It is the intention of the Kauri Dieback Programme to consider these guidelines to underpin best practice.

These guidelines are intended for Kauri Dieback Programme partners only and not for the general public. Currently, the Kauri Rescue™ Programme is planned to be the platform for the general public roll-out.

There is sufficient evidence from trials to be confident about the potential efficacy of phosphite trunk injection for kauri dieback control. However, phosphite is still considered experimental for treatment of kauri, and there are still unknowns regarding factors such as concentration, dose rates, the longevity of treatment, and appropriate doses for large trees or trees showing advanced decline. The recommendations in this guide are based on the best available experimental data, predominantly from Kauri Dieback Programme-sponsored research, but also with some reference to Kauri Rescue trials and commercial applications. This document should be updated as more information comes to hand.

1.2 Kauri dieback background

Kauri dieback, caused by *Phytophthora agathidicida* (Weir et al. 2015), is resulting in extensive decline and death of kauri trees in many parts of Auckland, Northland and Coromandel (Beever et al. 2009, 2010; Waipara et al. 2013). All ages of kauri trees appear to be susceptible to the pathogen, which causes root rot and bleeding cankers at the trunk base, leading to canopy thinning and eventually tree death. The pathogen is soil borne and the key to control is to keep the pathogen out of 'clean' forests. Distant spread could be by any movement of contaminated soil from an infected site, including on footwear, machinery, animals or plants. There will also be local passive spread via growth on roots, water runoff, and rain-splash. However, this is likely to be only a few meters per year.

Once *P. agathidicida* is in a forest, there are very few control options available for treatment of infected and threatened trees. To date, treatment with phosphite appears to be the most promising.

1.3 What is phosphite?

'Phosphite' is the anionic form of phosphonic acid (H_3PO_3). It is variously referred to as phosphite, phosphonic acid, phosphorous acid, or phosphonate. There are many commercial formulations of the product available internationally and in New Zealand, most being liquid formulations of a potassium salt of phosphonate. Phosphite has been used extensively for treating phytophthora diseases of many plant species, especially in horticultural systems, but

also in management of phytophthora diseases in forests and natural ecosystems (Smillie et al. 1989; Hardy et al. 2001; Garbelotto et al. 2007; Crane & Shearer 2014, Horner et al. 2015).

Phosphite can be applied as a trunk injection, foliar spray, trunk bark application or soil drench.

1.4 What is the mode of action of phosphite?

Phosphite is a systemic chemical that is translocated readily throughout the plant, in both the xylem and the phloem (Ouimette and Coffey 1989, 1990). In the xylem it moves with the mass sap/transpiration flow, and in the phloem it is translocated with photoassimilates in a source-sink relationship (Ouimette & Coffey 1990; Guest & Grant 1991; Jackson et al. 2000). It is not readily metabolised by the plant, and remains *in planta* until it is gradually diluted by leaf fall and bark shedding.

Phosphite appears to have a dual mode of action. Firstly, it directly inhibits mycelial growth and sporulation of oomycetes (*Phytophthora* and related organisms), as can be seen in *in vitro* tests with varying phosphite concentrations. *In vitro* trials with *P. agathidicida* showed a 50% reduction in growth at a phosphite concentration of 4.0 µg/ml (Horner & Hough 2013). But the control observed *in planta* is far greater than the concentration of phosphite in tissue would suggest, probably a result of stimulated plant defences (Smillie et al. 1989, Jackson et al. 2000, Hardy et al. 2001). Mechanisms such as increased soluble phenolic and phytoalexin production, and stimulated host defence enzyme activity are involved in this enhanced response (Nemestothy & Guest 1990; Jackson et al. 2000; Daniel & Guest 2006). In essence, phosphite application stimulates host defences. This means that disease control is possible at very low application rates, much lower than would be expected from direct fungicidal activity alone.

1.5 Environmental impacts of phosphite

Phosphite is generally considered as having low environmental toxicity, with beneficial effects far out-weighing any potential negative effects. A 17-year study in Western Australia found that phosphite treatment had no adverse effects and a strong positive impact on species diversity (Barrett & Rathbone 2018). In kauri forest, the beneficial effect of phosphite in maintaining kauri tree survival will flow on to maintenance of habitat integrity and thus to species diversity and assemblages.

Trunk injection will have negligible negative impacts outside the treated trees. Oxidation of phosphite to phosphate *in planta* to ultimately be used as a source of phosphorous (P) is minimal (Guest & Grant 1991). The phosphite generally remains in the plant, eventually shed by leaf drop, bark shedding or plant senescence. In the leaf litter, residual phosphite will eventually be metabolised by microbial activity into phosphate and only then can it be used as a nutrient for plant growth.

The volumes of phosphite applied on a landscape scale, especially with application via trunk injection only, are minimal compared with the background amounts of P in plants, soil and microbes. Roughly 0.1 g P/m² would be applied in a typical injection treatment regime. This represents less than 0.05% of the amount of P already present in the top layers of a typical forest soil. It has been postulated that phosphite may have a negative impact on phosphorous-sensitive plants (Lambers et al. 2013), but there is no evidence for this in kauri. Phosphite

application in some species where phosphate is limited can lead to exacerbated symptoms of phosphate deficiency (Carswell et al. 1997).

Spray applications of phosphite over a forest could potentially have effects on non-target organisms and interactions between them, but these are largely unknown. Plant species show various sensitivities to phosphite foliar treatment. Foliar phytotoxicity (e.g. leaf tip necrosis, flower and fruit drop) has been reported in a number of horticultural, ornamental and native forest species (de Boer & Greenhalgh 1990; Pilbeam et al. 2000; Tynan et al. 2001), but mostly only when high rates are used. The potential impacts of spray application in kauri forest are unknown. However, glasshouse trials by Scott et al. (2016) with a number of common New Zealand native species indicate relatively low phytotoxicity.

2 Kauri dieback/phosphite research results to date

2.1 *In vitro* trials of phosphite sensitivity

In 2010/11, *in vitro* tests on sensitivity to phosphite were carried out by growing six isolates of *P. agathidicida* on V8-agar plates amended with various concentrations of phosphite. *P. cactorum* and *P. cinnamomi*, two *Phytophthora* species known to be controlled well by phosphite in orchard and forest situations, were included for comparison. *P. agathidicida* proved to be even more sensitive to phosphite than the other species, with hyphal growth suppression at very low phosphite concentrations (Figure 1, Horner & Hough 2011, 2013). EC₅₀ values (50% growth reduction) were 4, 25 and 38 mg/L for *P. agathidicida*, *P. cinnamomi* and *P. cactorum*, respectively. In addition, interference of oospore development of *P. agathidicida* was noted at phosphite concentrations as low as 5 mg/L.

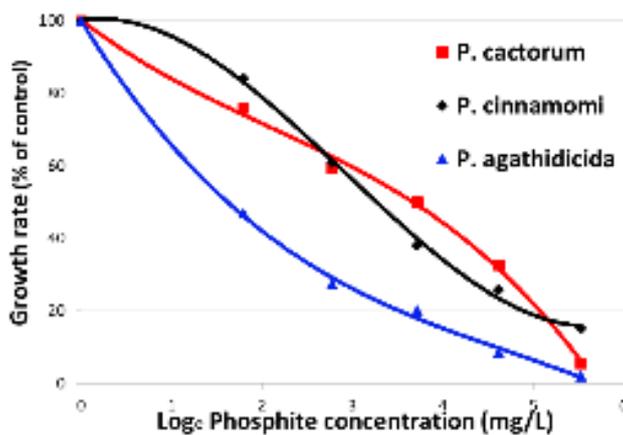


Figure 1. Growth of mycelial cultures of *Phytophthora cactorum*, *P. cinnamomi* and *P. agathidicida*, grown on V8-agar amended with various concentrations of phosphite.

2.1.1 Glasshouse trials

Following the *in vitro* trials, and before progressing to forest trials, phosphite efficacy was tested on *P. agathidicida*-inoculated kauri seedlings in the glasshouse. Two-year-old kauri seedlings were inoculated with *P. agathidicida* applied directly to trunk wounds or by soil application. Phosphite was applied as a foliar spray, as a trunk injection or as a soil drench, either 5 days before or 5 days after inoculation. All untreated control trees died, regardless of inoculation method. With phosphite injection, survival was 100% following *P. agathidicida* soil inoculation and 67% following trunk inoculation. The only trees that died following trunk injection were where the tree was inoculated before injection, and lesions had already nearly girdled the stem at the time of treatment. Foliar spray and soil drench-applied phosphite treatments were less effective than trunk injection, although some trees survived. Lesion extension (in stem-inoculated trees) and root health (in soil-inoculated trees) differed substantially between untreated trees and the various phosphite treatments (Figures 2 and 3).

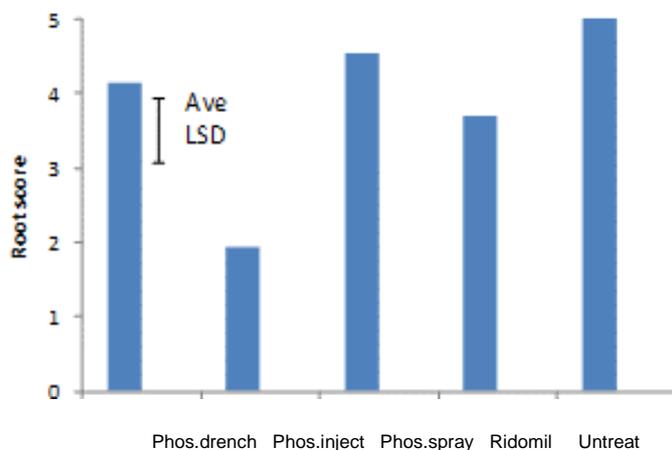


Figure 2. Average root disease score following treatment application, assessed 20 weeks after soil inoculation with *Phytophthora agathidicida*. ‘Root score’ is the average of root disease scored on a 0 to 5 scale, where 0 = all healthy, and 5 = all dead.

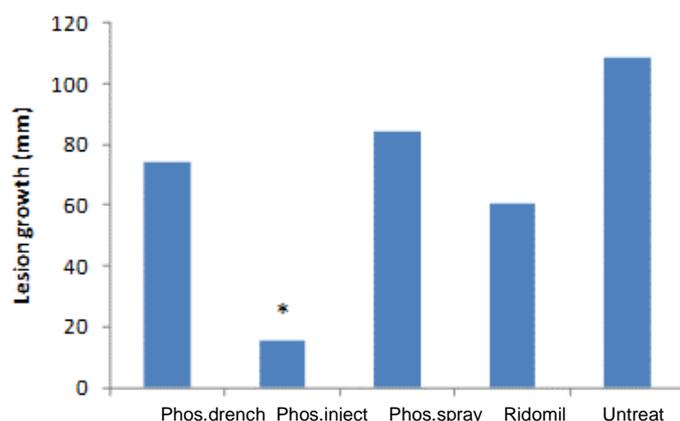


Figure 3. Average lesion extension upwards from the inoculation point, assessed 10 weeks after kauri trunk inoculation with *Phytophthora agathidicida*. The asterisk indicates lesion growth significantly ($p < 0.05$) less than in the untreated control.

The glasshouse trials demonstrated the potential of phosphite for control of kauri dieback, and gave the confidence to progress to forest trials to test the efficacy on infected trees.

2.1.2 Ricker trials

In early 2012, trials were established in four kauri ricker stands naturally infested with *P. agathidicida*. Sites were located at Huia and Whatipu (Waitakere ranges, Auckland) and Raetea and Omahuta Forests (Mangamuka Ranges, Northland). More than 160 trees were selected for the trial, with girths from 40 to 120 cm and all showing symptoms of *P. agathidicida* infection (canopy thinning and/or trunk cankers/bleeds). Baseline assessments were made of canopy health, trunk lesion size and activity, and baseline photographs were taken of the canopy and lower trunks for future comparisons. Trees were injected with 7.5% or 20% phosphite (Agriphos600®), 20 ml per 20 cm trunk circumference. Untreated control trees were included for comparison. Half of the treated trees were retreated after 1 year. Assessments of canopy health, lesion activity and lesion expansion, and comparisons with baseline canopy photographs were made 6 monthly for 5 years, with a further assessment after 8 years (Horner et al 2017 and November 2020).

Results clearly demonstrated that phosphite suppresses *P. agathidicida* infections. All basal trunk lesions on phosphite-treated trees dried up and healed, in most cases within 1 year of treatment application. There was cracking around lesion margins and a peeling back of

diseased bark, revealing healthy bark beneath. In the vast majority of cases these lesions remained inactive throughout the 5 years of the trial and were still inactive in an assessment done 8 years after treatment (Figure 4). In contrast, many lesions in untreated control trees remained active and continued to expand, in some cases ringbarking trees and causing tree death. The contrast was most noticeable at the Raetea site where 7 of 14 untreated control trial trees became girdled by lesions and died, and the remainder showed canopy decline and/or continued lesion activity. In contrast, lesions on all 24 treated trees healed and trees appeared healthy after 8 years, in most cases with strong new shoot growth (Horner et al. November 2020).

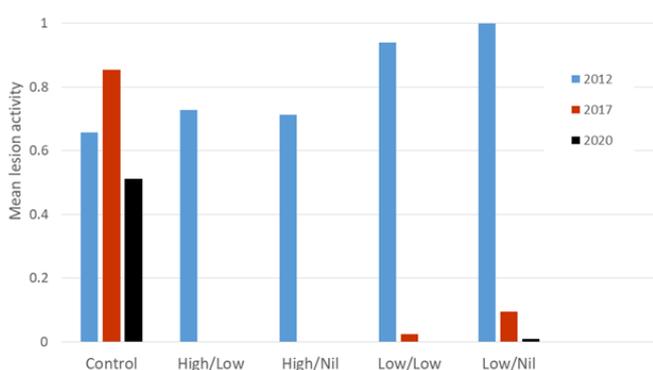


Figure 4. Mean basal trunk lesion activity score, on *Phytophthora agathidicida*-infected kauri trees in four forest sites, assessed before application of phosphite treatments in February 2012, and after 5 and 8 years, averaged across all four sites. ‘High’/‘Low’ indicate phosphite application rate in February 2012 and February 2013, respectively. High = 20% phosphite, Low = 7.5% phosphite, Nil = no treatment.

Phytotoxicity following treatment was a problem, particularly on the two Waitakere sites, suggesting that the rates applied were probably too high. There was leaf yellowing, canopy thinning and premature branch abscission in some cases, particularly with the 20% rate. A few trees at the Huia and Whatipu sites also showed bark cracking in line with injection points, first noticed 2 or 3 years post-treatment. Although these cracks generally dried up and healed, their presence is a concern. The phytotoxicity at Huia and Whatipu almost certainly contributed to accelerated mortality of some treated trees, with mortality greatest in the trees with the most advanced canopy scores (thinner canopies) at the start of the trial, and in smaller trees.

2.1.3 Trunk spray and low rate trials

Following phytotoxicity symptoms in earlier trials, new trials testing substantially lower phosphite injection rates were established across three naturally infested forest sites in 2016 (Horner et al. Aug. 2020). Trunk sprays of phosphite were included as a potential alternative application method. Injection treatments were applied only once, spray treatments were re-applied after 2 years.

Three years after initial treatment, no injected trees had died. Two of the 12 trees have died in each of the untreated control and trunk spray treatments. No canopy phytotoxicity symptoms had been noted in any treatment, and although some trees in all injection treatments showed minor cracks/bleeds associated with injection points, all these appeared healed and dry after 2–3 years.

Lesion activity and expansion was substantially lower in all phosphite injection treatments than in untreated controls (Figure 5). 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. There

were no obvious differences in lesion healing among the various injection concentrations and doses.

Trunk sprays with phosphite were not as effective as injection treatments, but still provided some control in terms of lesion healing, particularly following a second treatment after 2 years (Figure 5).

The trials demonstrated that a concentration of 4% phosphite injected into trunks, even at an increased spacing of 40 cm, is effective at healing *P. agathidicida* lesions in kauri ricker trees, without causing significant phytotoxicity. It is not yet known whether these lower concentrations and doses provide similar longevity of efficacy to those of higher doses.

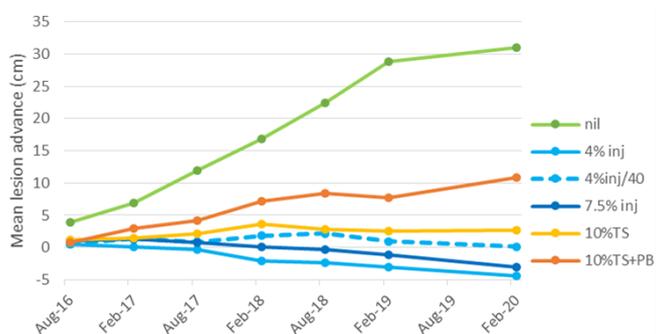


Figure 5. Mean basal trunk lesion expansion on *Phytophthora agathidicida*-infected kauri trees in three forest sites, following application of various phosphite treatments in February/March 2016. Expansion of pre-marked lesions was recorded approximately 6-monthly for 4 years. Lesion growth averaged across all monitored lesions at all sites. Negative values reflect lesion healing and shrinking. 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.

2.1.4 Large trees

Early forest trials were carried out only on trees in the 'ricker' size range, mostly up to about 35 cm trunk diameter. To determine rates that might be required to treat very large trees, in late 2016 trials were established in three infected forest sites (Cascades – Waitakere, Puketotora – Kerikeri, and Trounson Kauri Park) with focus on trees from 0.6 to 2.5 m trunk diameter (Horner et al. April 2020). An extremely conservative approach was taken with doses for the very big trees, because of the phytotoxicity noted in earlier trials and the unknowns of scaling up. Phosphite concentrations of 4% were used, with the low dose rate of one 20-ml injector every 40 or 80 cm. Although there was some lesion healing on most phosphite-injected trees, the healing was not complete in many cases, and certainly not the near total healing seen with rickers. In light of this trend early in the trial, a second dose of the original low rates was applied 2–3 years after the initial treatment. Early results show that this second dose stimulated improved lesion healing in most trees. In the concurrent 'Trunk spray and low rate trial', a rate of 4% phosphite injected every 40 cm around the trunk was effective at stopping lesion activity in rickers. This same dose (based on trunk circumference) in the large tree trial was not fully effective, suggesting that these large trees need a higher dose to facilitate total lesion healing and a different formula may be required. Given that there have been no obvious phytotoxicity symptoms noted to date, higher dose rates or higher frequency of application should be considered.

2.1.5 Timing trial

To address the question of the optimal season or timing to inject trees, a trial was carried out at the Huia dam site to investigate uptake time for the 20 ml solution from the injector. The same group of 20 trees were injected with water in spring, summer, autumn and winter, at different times of the day (early morning, midday, late afternoon) and in dry and wet weather conditions. The time for the syringe to empty was measured. Surprisingly, season had little impact on uptake time. Uptake was slightly slower in spring than in the other seasons, but otherwise uptake at different times of the day, weather conditions and season was remarkably similar. Results suggest that trees could potentially be treated at any time of the year – uptake will be similar whatever the season.

The work has not been repeated using phosphite injections and measuring differential efficacy with different seasonal application. Information on this is likely to come from Kauri Rescue trials, as trees are injected throughout the year, and any gaps in efficacy relating to season of application will become apparent over time.

2.1.6 Kauri Rescue

Kauri Rescue™ is a Citizen Science project that commenced in 2017 and involves working with private landowners to treat and monitor kauri trees suffering from kauri dieback disease on their own properties. Landowners are provided with materials, advice and any community support required to assess the health of their trees, treat them and monitor effectiveness of treatments. All data are centrally collated so that contributions from multiple landowners add to the pool of knowledge, accelerating development of treatment tools for kauri dieback, while helping landowners protect their own trees. At the time of writing this document (June 2020), there are approximately 80 participant landowners, with a total of approximately 2000 trees treated. This provides a dataset with a huge range of locality, topography, vegetation type and soil type. This, combined with many different tree sizes, stages of decline, timing of treatment, phosphite concentration and dose, provides the opportunity, over time, to investigate many factors that may influence treatment efficacy. Most treated trees are in the 'ricker' size category. To date, treatments have focused on either 4% or 6% phosphite, injected at either 25- or 40-cm spacings around the trunk. Early results suggest each of these rates and doses have some efficacy, aligning with observations in the low-dose trials outlined in Section 2.1.4. It is still too early in the trial to look for associations with some of the potential co-factors noted above.

2.1.7 Conclusions/general observations from trials to date

There are still many unknowns around treatment with phosphite, which are outlined in the following section. However, the trials to date clearly indicate that phosphite is a useful tool for the treatment of kauri trees infected by *P. agathidicida*, and potentially for protecting trees immediately challenged by the pathogen. Trunk injection of infected trees halted lesion expansion in almost all cases, and often led to healing and sloughing off of lesions, revealing healthy bark beneath.

The first ricker trials demonstrated the potential efficacy of phosphite, but also some of the risks of treatment, with high concentrations (7.5% and 20%) causing phytotoxicity and accelerating the demise of some trees, especially those already showing advanced symptoms. These trials have also given some insight into the potential longevity of the treatment effect, with minimal reactivation of lesions 8 years post-treatment, although it should be noted that new colonisation will most likely be subterranean and perhaps not noticed for a number of years.

Subsequent trials with 4% phosphite have demonstrated that this lower concentration is still effective at stopping lesion expansion, at least in ricker trees, although it is still too soon to know if the longevity of efficacy and eventual canopy recovery is comparable to that achieved with higher concentrations.

Phosphite injection clearly has a systemic effect, with healing of lesions some distance from the injection point. With the movement of phosphite from the trunk injection point into the canopy then subsequent redistribution throughout the tree, we can reasonably assume that subterranean lesions on the trunk base and main root system will also be healed. This systemic nature of phosphite makes it a very powerful tool.

The mechanism of control is not known for kauri, but evidence suggests that it is predominantly through stimulation of host defences rather than fungicidal activity. The effect at very small concentrations in the tree is far greater than would be expected from fungicidal activity alone. The stimulation of host defences is seen in the accelerated sloughing of kauri bark following treatment, not just around lesions, but also remote from the lesions.

3 Research uncertainties/assumptions

Although the basic efficacy of phosphite injection treatment of kauri is established, there are still many gaps in our knowledge, and this should be considered in deployment. These knowledge gaps should not paralyse operational adoption of phosphite, but should add a layer of caution and signal the need to adapt protocols as more information emerges.

Current knowledge gaps include:

Dose for big trees: Effective and safe doses for big trees are yet to be determined. Rates trialled to date have been too low to stimulate total lesion healing, but higher rates have yet to be tested. A second low-dose treatment may improve efficacy, but more time is needed to determine this.

Longevity of treatment: It is not known how long treatments remain effective. It is currently assumed that it is at least 3 years, perhaps significantly longer.

Low-dose efficacy and longevity: Low-dose treatments have to date appeared effective at healing lesions in ricker trees, but the longevity of these treatments and long term tree health improvement in comparison to higher dose treatments is as yet unknown. Lower doses were not as effective in large trees.

Retreatment regimes: Required re-treatment regimes for long-term control are unknown. Currently the assumption is a 3- to 6-year interval may be appropriate, based on overseas experience treating other species, and the apparent long-term protection in the original ricker trials.

Roots: Are roots healed/protected by trunk injection? It is assumed that following trunk injection, root lesions will heal, and feeder roots will have some protection from *P. agathidicida* attack. But this has not been directly investigated.

State of inoculum within treated trees: Following effective treatment, is *P. agathidicida* eradicated from within the tree, effectively contained (e.g. by gum) or just temporarily suppressed?

How soon after treatment does recolonisation of roots occur? Inoculum will remain in the soil and in plant debris. It is not known how soon after treatment that recolonisation and re-establishment in the root system can occur. Current assumptions are that it may be a few years, but with no direct evidence to back this up.

Phytotoxicity: Factors contributing to phytotoxicity (leaf yellowing or loss, twig drop, trunk cracking) are still largely unknown. Is it simply the concentration or dose of phosphite, or do other factors influence the outcome? Site factors, application season, tree health, canopy volume, etc. could all have an effect.

Tree health at time of treatment: How does initial tree health influence efficacy of treatment, and potential phytotoxicity responses. At what point is a tree too far gone to treat? Should trees with advanced symptoms be given a lower dose?

Concentrations and doses: Is it better to give fewer injections of higher concentration, or more injections with more dilute concentration? Does it matter? Current recommendations are based on research with other species and early kauri trials, but there are many possible regimes.

Optimal Treatment timing: Does the seasonal timing of treatment make a difference to either efficacy or phytotoxicity responses? There is no evidence for this, but it has not been systematically investigated.

Phosphite formulation: Various commercial phosphite products are available. Do different formulations of phosphite differ in efficacy on kauri? Can efficacy be improved with additives? To date only Agri-fos 600 has been tested in formal kauri forest trials. Internationally, other phosphite formulations have been used successfully.

Drench or spray application: Soil drenches with phosphite had some efficacy in glasshouse trials, but less effective than injection. It is assumed that soil drenches in the forest would be logistically difficult and unlikely to be effective because of deep litter layers, difficulty reaching the target roots, complex microbial interactions and unknown environmental impacts.

Trunk spray application: Trunk sprays have proven partially effective at trunk lesion healing, but not as well as injection. It is not known whether topical trunk application stimulates genuine systemic responses throughout the trunk and root system, or is just local activity.

Foliar spray application: Foliar application of phosphite has not yet been tested in field situations with kauri. Glasshouse trials with kauri showed some activity, though inferior to trunk injection. Multiple off-target effects of spray application in a forest situation would need to be considered before deployment, including environmental, social and cultural concerns.

What percentage of trees should be treated? There have been no studies of the optimal treatment pattern across sites, i.e. whether all trees should be treated, or whether just a percentage of trees is sufficient. Will root grafting mean that neighbours of treated trees get some protection, or will it just dilute the effective concentration in the treated tree, making the treatment less effective?

Should smaller trees be treated, and if so, how? What are the impacts of only treating trees large enough to be injected? How can smaller/sapling trees be treated (e.g. foliar or trunk spray, micro-injection)?

Barrier deployment: Deployment of phosphite as a barrier treatment has potential to substantially reduce spread and potentially contain the pathogen on some sites, but multiple factors need to be considered and systematic research is required.

Preventative treatment: Should phosphite be used to treat healthy trees, as a preventative treatment? If so, what criteria or protocols should be employed, e.g. proximity to symptomatic trees or confirmed sites. Current recommendations are to treat only symptomatic trees or immediately adjacent asymptomatic trees. Most of the formal scientific trials have been with symptomatic trees, although Kauri Rescue treatments extend to adjacent asymptomatic trees.

Risks to asymptomatic trees: What are the risks of treating asymptomatic trees as a prophylactic treatment? Phytotoxicity is assumed to be the main risk, although healthy trees appear to have a greater tolerance.

Alternate hosts: It is expected that many trees other than kauri are potential hosts of *P. agathidicida*, harbouring the pathogen. When treating infested stands, should these alternate hosts also be treated, and what impact will this have on pathogen spread and control?

Non-target impacts: What impacts will injection of kauri have on other species and the environment? It is assumed that trunk injection, and thus restriction of most of the phosphite

within the treated tree, will result in minimal impacts outside the tree (other than the targeted effect of prolonging kauri tree survival)

Physiological responses in kauri: The impact of phosphite treatment on biochemical and physiological responses in kauri are unknown.

Mycorrhizae: The impact of phosphite trunk injection in kauri on mycorrhizal colonisation or function is unknown.

Resistance: Frequent and continued use of fungicides can lead to a build-up of resistance to that chemical within pathogen populations. Some chemicals are more prone to this effect than others. Slight shifts in population susceptibility to phosphite have been noted with some *Phytophthora* populations, primarily in horticultural settings with frequent applications. It is not anticipated that this will be an immediate threat to phosphite use in kauri forest, given the scale, timing and treatment regimes being recommended.

Impact of site factors: 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.

Some of the questions outlined above will be addressed over time by analysis of Kauri Rescue data where trees are being treated in multiple different situations, and also in formal trials. It will also be useful to keep records if agencies or land managers roll out phosphite on a large scale. With the number of uncertainties remaining around phosphite treatment, every opportunity to gather more information around treatment successes and problems in various situations should be taken. This should be backed up by dedicated controlled trials, where research trials are specifically targeted at addressing specific questions.

4 Decision framework

4.1 Site selection

Only trees on sites with confirmed *P. agathidicida* infection should be treated.

Appropriate consultation with landowners, land managers and mana whenua should precede any applications, with all applicable approvals in place before commencing any treatment.

4.2 Tree selection

When deciding whether or not to treat, consider the potential benefits (e.g. lesion healing and tree survival) versus the potential risks (e.g. phytotoxicity or accelerated decline of trees with advanced infections).

In confirmed infected sites, consider treatment of:

- trees with canopy symptoms consistent with kauri dieback (canopy thinning, yellowing, branch dieback)
- trees with basal trunk lesions consistent with kauri dieback (lesions/sap bleeds contiguous, or almost contiguous, with the ground)
- non-symptomatic trees immediately adjacent to symptomatic trees (assessment of 'at risk' non-symptomatic trees to treat should consider site factors including proximity to symptomatic trees, slope, water drainage, vector pathways etc., and will vary from site to site). Decisions on distance will be subjective, until more information on local spread, disease latency, local vectors etc. is available.

Do not inject:

- trees less than 7 cm diameter
- dead trees – they are unlikely to take up the phosphite, and it will not be translocated to the required (infected) portions of the tree
- healthy trees remote from any obvious threat of infection (taking into consideration site factors including proximity to symptomatic trees, slope, water drainage, vector pathways etc., which will vary from site to site).

4.3 Deployment

Timing: As yet there is no evidence that any particular season is better or worse than any other for injecting trees, but this aspect has not been formally investigated. Most of the formal trials had application between November and March. Over the next few years, results from Kauri Rescue trials should give some insight into whether season of application influences efficacy.

Pattern of treatment: There have been no studies of the optimal treatment pattern across sites, i.e. whether all trees should be treated, or whether just a percentage of trees is sufficient. The deployment strategy, concentration and dose, criteria for treating/not treating a given tree etc. should be determined in advance of commencing treatment at a site.

Barrier treatment: Deployment of phosphite as a barrier treatment, effectively isolating the pathogen within a non-susceptible barrier, could potentially reduce spread and contain the pathogen on some sites. However, many unknowns remain and multiple factors need to be considered (e.g. kauri tree size and density, alternative hosts, slope, vectors etc.). Systematic and long-term research is required (discussed more fully in Horner (2016)).

4.4 Phosphite concentration and dose

Phosphite is likely to be effective across a range of concentrations (a.i./ml) and doses (number of injectors per cm of trunk circumference). Trees with advanced canopy symptoms are more likely to show phytotoxicity, so doses should be reduced.

Rickers (DBH <50 cm diameter): Early ricker trials indicated that phosphite concentrations of 20% and 7.5% often caused phytotoxicity, so lower concentrations are recommended, at least until more information is available. Current evidence suggests that phosphite concentrations from 4 to 6% are safe yet effective for trunk injection. Higher concentrations could cause phytotoxicity symptoms including leaf yellowing, some leaf or branchlet loss, and trunk cracking.

Current recommendation for ricker trees: one 20-ml injection of 4–6% phosphite per 20–40 cm trunk circumference. For trees with substantial canopy thinning (3 or 4 on the canopy scale), injectors should be spaced at 30–40 cm. A retreatment interval of 5 years is recommended, although evidence for this is currently lacking.

Large trees (DBH >50 cm diameter): Less is known about effective concentrations and doses for large trees. Concentrations of 4% phosphite with doses of 20 ml per 40 or 80 cm trunk circumference were safe (no obvious phytotoxicity) but not fully effective at lesion healing. However, higher concentrations and doses have not yet been tested on large trees, so a conservative approach is recommended.

Current recommendation for large trees: one 20-ml injection of 4% phosphite every 40-cm trunk circumference. This 40-cm spacing is likely to be decreased (i.e. dose increased) in future once efficacy and safety tests have been completed. In the meantime, a repeat application after 1 or 2 years should be considered if complete lesion healing is not apparent within a year or two of treatment.

5 Operational specification – Injection process

5.1 Equipment and preparation

- Battery-operated drill with 5–6 mm diameter drill-bit
- Spring-loaded 20-ml injectors (e.g. Chemjet®)
- Phosphite (e.g. Agrifos®600)
- Measuring cylinders and containers for mixing phosphite and loading syringes
- Tape measure for tree girth
- Tags for labelling trees
- Protective equipment (gloves, goggles)
- Water for mixing phosphite solutions and for rinsing hands etc.
- Hard brush for cleaning syringe tip
- Silicone sealant for sealing holes (optional).



5.2 Mixing of phosphite solution

Ideally, the phosphite should be diluted fresh from the concentrate each day, using clean water. Once diluted, efficacy is likely to decline after 48 h. ‘Hard’ or alkaline water will reduce product longevity and should be avoided if possible. Most town water supplies should be suitable (even if chlorinated), or rainwater could be used.

Dilution of phosphite concentrate

Product used	Desired phosphite concentration		
	4%	5%	6%
60% phosphite (e.g. Agrifos600)	1 part phosphite, 14 parts water	1 part phosphite, 11 parts water	1 part phosphite, 9 parts water
40% phosphite	1 part phosphite, 9 parts water	1 part phosphite, 7 parts water	1 part phosphite, 5.7 parts water

5.3 Injection procedures

- Measure tree girth (at chest height) by placing a measuring tape around the trunk, and calculate the number of injectors required, dependant on the dose chosen (e.g. one injector per 20 or 40 cm circumference). Note that a hand-span width is roughly 20 cm. Do not inject trees less than 20 cm girth.
- Load the injector by firmly holding the barrel, immersing the tip in the phosphite solution and pulling the plunger out fully. Twist the plunger slightly to lock in place. Load all injectors for a tree before starting to drill.
- Drill holes to 4.5–5 cm depth on a slight downward angle, and immediately screw in the loaded injector. Hold the barrel, not the plunger. Screw in until there is a tight fit. Release the lock, to allow the spring to slowly force the solution into the tree. Do not push the plunger in, the spring will force the phosphite into the tree automatically.
- Remove the injector as soon as possible after the injector is completely empty and removed. This usually takes from 3 to 10 minutes. If injectors are left in too long, kauri sap can enter the injector tip or chamber, reducing its longevity.
- Post injection, holes can be sealed by smearing a small dab of silicon sealant across the hole entrance. But don't fill the entire hole with silicon. Alternatively, leave the holes open – in most cases the tree's own sap will plug the hole.



Notes and tips:

- The injection height up the trunk is not critical, but between knee and chest height is usually most convenient and comfortable.
- The injection angle should be slightly downward, to aid complete emptying of the syringe.
- Injectors should be evenly spaced around tree. An exception would be where dead areas or large lesions need to be avoided.
- Avoid dead areas. Do not drill directly into lesions, dead areas of wood, cracks or immediately above such areas. Sap flow in such areas will be impeded or non-existent, and there will be inadequate uptake. Preferably move injectors to healthy wood to the side of lesions, rather than above. If forced to inject above lesions, go as far away as possible (preferably >50 cm).
- Use each hole only once.
- If syringes completely stop, or do not empty within about 20 minutes, remove carefully and re-inject a fresh point at least 8–10 cm away. Estimate the total volume not taken up by a given tree and re-inject 20-ml doses accordingly, to avoid over-dosing.

- If trees are being reinjected, e.g. a few years after initial treatment, the injection points should be laterally well away from the initial points, and not immediately above or below those initial points. Where possible, re-injection points should be mid-way between the initial points (which are usually visible for a few years).

5.4 Clean-up

Syringes should be cleaned in warm soapy water, at least daily. This will prolong their life. Soaking the syringe tip in methylated spirits for a few minutes will help unclog blocked tips, or remove persistent gum residues. Immerse the tip only, not the whole syringe, then rinse in clean water.

There is no need to clean or sterilise syringes between trees, but debris (if present) should be brushed off.

5.5 Safety

Regional Councils, the Department of Conservation (DOC), the Ministry for Primary Industries (MPI) and other agencies have existing protocols for working safely in the bush, handling chemicals, working alone etc., and these should be followed.

All operators should read and follow label instructions, and refer to the Material Safety Data Sheet (MSDS) sheet for whatever product they are using. For example, the Agri-fos 600 MSDS can be obtained here:

https://keyindustries.co.nz/Portals/0/PDFs/Key%20Agrifos_SDS%202015.pdf

Gloves and eye protection should be worn during phosphite mixing and injection operations.

6 Monitoring

Collection of individual tree data is not essential to the large-scale treatment of forests, and it will have no direct effect on the success or failure of treatment. However, over time it will help to fine-tune procedures and improve treatment efficacy. Some uncertainties remain around phosphite treatment. Systematic gathering of data during treatment operations, with subsequent monitoring, will add to the pool of knowledge and help to elucidate some of the outstanding questions. It will also help to gauge the success of any operations.

The time required to collect data is typically longer than the time to treat a tree, depending on the number of parameters measured. The amount of data collected will depend on factors such as budget and/or time available, the number of trees to be treated, and the goals of the agency/group doing the treatment. Therefore, a full set of data variables and consistent measuring and recording of each should be developed as a national phosphite monitoring form, which can be reduced for different situations. Some key parameters need to be assessed from all treated trees and a random selection of untreated comparison trees if any useful long-term treatment information is to be gained.

Basic site and tree-health parameters should be recorded pre-treatment or at the time of treatment. This will form the baseline for comparison and assessment of treatment effects in future years. At a minimum, initial pre-treatment measurements should include:

- A tree identifier, including a unique number and GPS reference. Trees should also be tagged.
- Trunk girth (or DBH/diameter at breast height)
- Canopy health score (using the KDP 1–5 canopy scoring scale, Figure 6)
- Trunk lesion presence (see notes and typical pictures below, Figure 7)
- Trunk lesion activity (see below for scale and how to assess).

Additional useful pre-treatment parameters include:

- Extent of lesions – maximum height from ground, percentage of circumference at ground level
- Canopy growth (vigour of current season growth, on a 0 to 2 scale where 0=no growth, 1=some growth, 2=vigorous growth)
- Canopy photo at a fixed (referenced) photo point
- Canopy colour
- Trunk cracks (prior to injection)
- Site parameters including slope, aspect, vegetation type.

Treatment records should include:

- Concentration of phosphite
- Number of injectors used (or dose calculation e.g. one injector every 20 cm)
- Weather conditions during treatment (approximate temperature, precipitation).

Post-treatment records should include the tree parameters measured pre-treatment:

- Canopy health score

- Trunk lesion presence
- Trunk lesion activity
- Extent of lesions – maximum height from ground, percentage of circumference
- Canopy growth (vigour of current season's growth, if assessments are made in summer)
- Canopy colour
- Trunk cracks or other signs of phytotoxicity.

The timing frequency of post-treatment monitoring or assessments will depend entirely on resources available and the aims of the exercise. Provided the initial assessments are thorough, the interval to reassessment is not important. Treatment effects in terms of lesion healing should be apparent within a few months, and well entrenched after a year. An assessment after 1 year could determine general treatment efficacy, with a follow-up in Years 3, 4 or 5 to determine longer-term impacts on lesion healing and overall tree health. If more sensitive data are needed, e.g. to assess the relative performance of different treatment regimes, annual assessments would be beneficial to avoid missing subtle differences in treatments. If comparisons between treatment regimes are required, it is helpful but not essential to leave random untreated control trees or areas for comparison.

Monitoring data will only be useful if the pre-treatment data collection is robust, so if possible it is worth putting in the resources up front to allow flexibility in assessments later.

Whether to collect data on all trees or just on a selection again depends entirely on the aims of the work and the resources available. It will be a balance of the need to collect data versus the need to treat more trees. There should always be data collection on at least a percentage of trees, at the very least to facilitate assessment of the benefits of any operation. A sample size can be calculated depending on the expected effect size (odds ratio) of the variables to be measured to ensure sufficient power to detect a true difference between means or proportions if one exists.

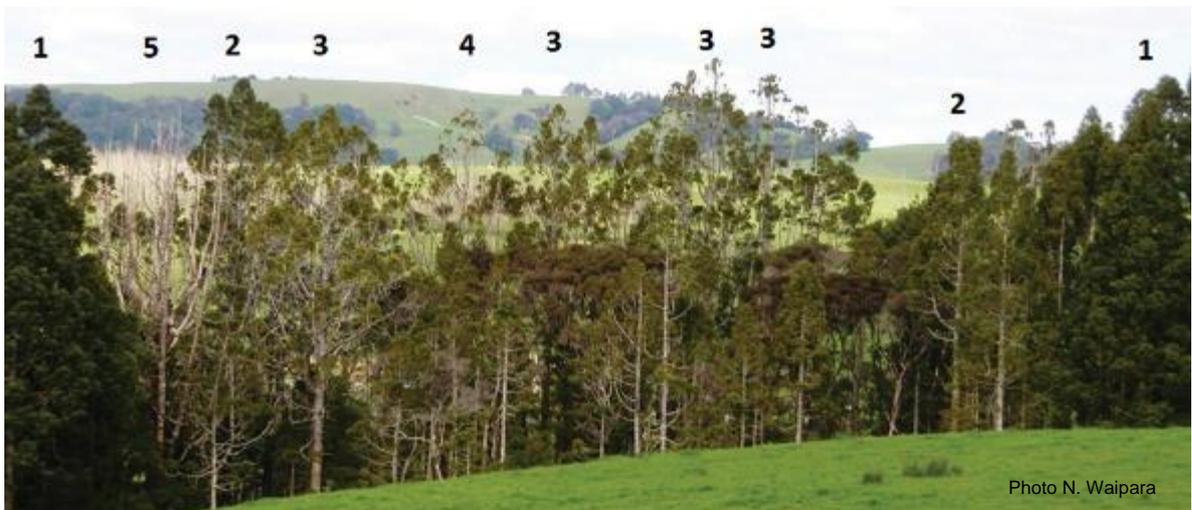


Photo N. Waipara

Figure 6. Canopy health scoring: 1 = Healthy crown – no visible signs of dieback, 2 = Foliage/canopy thinning, 3 = Some branch dieback, 4 = Severe dieback, 5 = Dead.

Lesion assessments

Kauri trunk lesions are dead or diseased areas of bark, usually associated with bleeding of sap. Not all lesions are caused by *Phytophthora* attack. Lesions not associated with the ground are unlikely to be caused by *P. agathidicida*. Only lesions that are close to or contiguous with the ground should be included in this assessment.

Typical lesions caused by *P. agathidicida* are shown in Figure 7. Such lesions, if associated with nearby canopy symptoms and a positive soil test can be reasonably assumed (in most cases) to be caused by *P. agathidicida*. Typical lesions healed following phosphite treatment are shown in Figure 8.

Lesion activity assessment:

Assessing lesion activity is a subjective judgement of how recently a given lesion has been active. The simplest method is based on a touch test to categorise a given lesion into one of three categories: *Active* is soft and sticky, *Not Active* is hard and dry (cannot be dented with a fingernail), *Semi-active* is not sticky, but soft enough to be dented by a fingernail.



Figure 7. Typical kauri trunk lesions caused by *Phytophthora agathidicida*.



Figure 8. Healing of typical kauri trunk lesions following treatment with phosphite. Top: cracking and peeling back of bark around lesions. Bottom: sequence of the same tree with an active lesion pre-treatment (left) and lesion peeling and healing 5 years (centre) and 8 years (right) post-treatment.

7 Hygiene requirements

Strict hygiene protocols should be followed to prevent spreading kauri dieback before, during and after treatment operations. Regional Councils, DOC, MPI and other agencies have their own protocols for working in kauri dieback areas, and these should be strictly followed. The general principles are outlined on the Kauri Dieback Programme website: <https://www.kauridieback.co.nz/media/1857/2018-kauri-dieback-hygiene-procedures.pdf>.

However, because of the specific targeting of kauri dieback-infected areas in any treatment programme, it will not be possible to follow the general principles of staying on the track and avoiding working around kauri. Therefore, special care needs to be taken to **minimise** spread of soil within infected sites, and to **prevent** spread between sites. A few strategies are listed here:

- Avoid working in wet or muddy conditions
- Wear footwear that does not easily accumulate soil, or wear over-booties that can be changed frequently
- Minimise contact of equipment with soil, for example by using a groundsheet for gear or using a rope/carabiner to hang gear in trees
- When treating within an infected area, commence work on the healthy margins and work towards more heavily diseased areas
- Always thoroughly clean footwear and equipment in contact with soil between sites or stands
- Thoroughly clean equipment, clothing etc. at the end of each day.

There is no need to sterilise the injection syringes between trees, but debris (if present) should be brushed off. However, they should be thoroughly cleaned at the end of each day.

8 Glossary of terms

Canopy symptoms: Yellowing of leaves, canopy thinning or dieback.

Canopy rating: Rating scale used to measure the health of the canopy of kauri.

DBH: Diameter of trunk at breast height

Girth: circumference of trunk, measured at chest height

Kauri dieback: Name of the disease associated with dieback or death of kauri, caused by the pathogen *Phytophthora agathidicida*

KDP: Kauri Dieback Programme

Lesion: canker or diseased tissue in the bark and sometimes wood of a trunk or branch

Phytotoxicity: Adverse reaction in a plant following treatment with a chemical agent, e.g. leaf scorching, yellowing, thinning etc.

Phosphite: Anionic form of phosphonic acid (H₃PO₃). Also referred to as phosphonic acid, phosphorous acid or phosphonate. Used to control diseases caused by *Phytophthora* and related organisms.

Ricker: pole stage of a kauri tree. Larger than a sapling, but not adult form. Phase where the tree has a conical canopy form, before establishing the main structural branch system. Trunks usually about 10–40 cm diameter

9 Acknowledgements

Preparation of this document was funded by MPI on behalf of the Kauri Dieback Programme. The recommendations are based on the best available experimental data, predominantly from Kauri Dieback Programme-sponsored research, but also with some reference to Kauri Rescue trials and commercial applications. Multiple individuals, agencies, landowners, iwi and hapū have contributed to this work over the past 10 years, and their input is gratefully acknowledged.

10 References

- Barrett S, Rathbone D 2018. Long-term phosphite application maintains species assemblages, richness and structure of plant communities invaded by *Phytophthora cinnamomi*. *Austral Ecol* 43: 360–374. DOI: [10.1111/aec.12574](https://doi.org/10.1111/aec.12574).
- 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.
- Beever RE, Tsai S, Waipara NW, Dick MA, Ramsfield TD 2010. Pathogenicity of *Phytophthora* taxon *Agathis* (PTA). Fifth Meeting of the International Union of Forest Research Organisations (IUFRO) Working Party 7.02.09, *Phytophthoras in Forests and Natural Ecosystems*; March 2010. Rotorua, New Zealand. p. 2 (Abstract only).
- Carswell MC, Grant BR, Plaxton WC 1997. Disruption of the phosphate-starvation response of oilseed rape suspension cells by the fungicide phosphonate. *Planta* 203: 67–74.
- Crane CE, Shearer BL 2014. Comparison of phosphite application methods for control of *Phytophthora cinnamomi* in threatened communities. *Australas Plant Pathol* 43: 143–149.
- Daniel R, Guest D 2006. Defence responses induced by potassium phosphonate in *Phytophthora palmivora*-challenged *Arabidopsis thaliana*. *Physiol Mol Plant Pathol* 67: 194–201.
- De Boer RF, Greenhalgh FC 1990. Efficacy of potassium phosphonate in controlling *Phytophthora* root rot of subterranean clover and ornamental plants in Victoria. *Australas Plant Pathol* 19: 124–125.
- Garbelotto M, Schmidt DJ, Harnik TY 2007. Phosphite injections and bark application of phosphite + Pentrabark™: control sudden oak death in coast live oak. *Arboric Urban For* 33 (5): 309–317.
- Guest DL, Grant B 1991. The complex action of phosphonates as antifungal agents. *Biol Rev* 66: 159–187.
- Hardy G, Barrett S, Shearer BL 2001. The future of phosphite as a fungicide to control the soilborne pathogen *Phytophthora cinnamomi* in natural ecosystems. *Australas Plant Pathol* 30: 133–139.
- Horner IJ, Hough EG July 2011. Phosphorous acid for controlling *Phytophthora* taxon *Agathis* in kauri. PFR Client report prepared for MAF Biosecurity. PFR SPTS No. 5802.
- Horner IJ, Hough EG 2013. Phosphorous acid for controlling *Phytophthora* taxon *Agathis* in kauri: glasshouse trials. *N Z Plant Prot* 66: 235–241.
- Horner IJ, Hough EG, Horner MB 2015. Forest efficacy trials on phosphite for control of kauri dieback. *N Z Plant Prot* 68: 7–12
- Horner IJ. November 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 I, Hough E, Horner M. August 2017. Phosphite for control of kauri dieback: final report. A Plant & Food Research report prepared for: Ministry for Primary Industries. Milestone No. 66652. Contract No. 33430. Job code: P/345160/06. SPTS No. 15425.

Horner IJ, Arnet MJ. April 2020. Phosphite large tree treatment trials: brief report April 2020. A Plant & Food Research report prepared for: Ministry for Primary Industries. Milestone No. 66663. Contract No. 33447. Job code: P/345160/05. SPTS No. 19316.

Horner I. June 2020. Phosphite treatment by trunk injection in kauri. A Plant & Food Research report prepared for: Ministry for Primary Industries. Milestone No. 87476. Contract No. 38244. Job code: P/345172/01. PFR SPTS No. 19579.

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

Jackson TJ, Burgess T, Colquhoun I, Hardy GESTJ 2000. Action of the fungicide phosphite on *Eucalyptus marginata* inoculated with *Phytophthora cinnamomi*. Plant Pathol 49: 147–154.

Lambers H, Ahmedi I, Berkowitz O, Dunne C, Finnegan P, Hardy G, Jost R, Laliberté E, Pearse S, Teste F 2013. Phosphorus nutrition of phosphorus-sensitive Australian native plants: threats to plant communities in a global biodiversity hotspot. Conserv Physiol. DOI: 10.1093/conphys/cot010.

Nemestothy GS, Guest DI 1990. Phytoalexin accumulation, phenylalanine ammonia lyase activity and ethylene biosynthesis in fosetyl-Al treated resistant and susceptible tobacco cultivars infected with *Phytophthora nicotianae* var. *nicotianae*. Physiol Mol Plant Pathol 37: 207–219.

Quimette DG, Coffey MD 1989. Comparative antifungal activity of four phosphonate compounds against isolates of nine *Phytophthora* species. Phytopathology 79: 761–767.

Quimette DG, Coffey MD 1990. Symplastic entry and phloem translocation of phosphonate. Pestic Biochem Physiol 38: 18–25.

Pilbeam RA, Colquhoun IJ, Shearer B, Hardy GESTJ 2000. Phosphite concentration: its effect on phytotoxicity symptoms and colonisation by *Phytophthora cinnamomi* in three understorey species of *Eucalyptus marginata* forest. Australas Plant Pathol 29: 86–95.

Scott P, Bader MK-F, Williams NM 2016. Foliar phosphite application has minor phytotoxic impacts across a diverse range of conifers and woody angiosperms. Physiol Plant 158: 124–134.

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.

Tynan KM, Wilkinson CJ, Holmes JM, Dell B, Colquhoun IJ, McComb JA, Hardy GESTJ 2001. The long-term ability of phosphite to control *Phytophthora cinnamomi* in two native plant communities of Western Australia. Aust J Bot 49: 761–770.

Waipara NW, Hill S, Hill LMW, Hough EG, Horner IJ 2013. Surveillance methods to determine tree health, distribution of kauri dieback disease and associated pathogens. N Z Plant Prot 66: 235–241.

Weir BS, Paderes EP, Anand N, Uchida JY, Pennycook SR, Bellgard SE, Beever RE 2015. A taxonomic revision of *Phytophthora* Clade 5 including two new species, *Phytophthora agathidicida* and *P. cocois*. *Phytotaxa* 205 (1): 021–038.



DISCOVER. INNOVATE. GROW.