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Phosphite for control of kauri dieback: final report (Executive summary only)

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EXECUTIVE SUMMARY

Phosphite for control of kauri dieback: final report

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Plant & Food Research: Havelock North

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Kauri dieback, caused by *Phytophthora agathidicida*, is resulting in decline and death of kauri trees in many parts of Auckland, Northland and Coromandel. Of a very small number of potential control options, phosphite (phosphorous acid, phosphonate) is probably the most promising. Following success with phosphite to control *Phytophthora* lesion growth on inoculated seedlings in glasshouse trials, forest trials were established on four *P. agathidicida*-infected sites in early 2012. Sites were at Huia and Whatipu in the Waitakere Ranges (Auckland) and Raetea and Omahuta in the Mangamuka Ranges (Northland). A total of 163 trial trees across the four sites were all showing symptoms of kauri dieback (canopy thinning/ dieback and/or basal trunk lesions) at the start of the trial.

Baseline assessments of canopy health, trunk lesions and tree size were made before assigning trees to treatment regimes in a manner that evenly spread trees of different starting disease severity across the different treatments. In January or March 2012, trees were injected with either a 20% phosphite solution (Huia and Whatipu only), a 7.5% phosphite solution (all sites) or left untreated. One year later, half of the previously treated trees were re-injected, this time with a 7.5% solution, regardless of the first treatment. Every 6 months for a 5-year period following the first treatment, all trees were assessed for canopy symptoms, lesion activity and lesion expansion.

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. 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 six out of 14 untreated control trial trees became girdled by lesions and died, and the remainder were in decline with active lesions, while lesions on all 24 treated trees healed and trees appeared healthy after 5 years.

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. Mortality was greatest in the trees with the most advanced canopy scores at the start of the trial, and in smaller trees.

The phytotoxicity symptoms observed, particularly at the Huia and Whatipu sites, 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, masked the rapid and long-lasting lesion healing that occurred. Phosphite treatment prevented the expansion of *P. agathidicida* lesions in all treated trees (including those that subsequently died) demonstrating the potential to either protect trees from *P. agathidicida* or restore them to health. Slightly lower phosphite rates are still likely to provide disease control, while reducing the detrimental phytotoxicity effects. The 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 trees in *P. agathidicida*-infected stands if no treatment is made.

The 20% phosphite rate is certainly considered too high for treatment of kauri. Even the 7.5% rate is possibly too high. Trials established in 2016 using 4% phosphite have to date shown no phytotoxicity symptoms.

Until more information is available, a phosphite concentration of 4–6% is recommended, with 20 ml of this solution injected at intervals of at least 20 cm around the trunk circumference. The required frequency of re-treatment is still unknown, but results from this trial indicate that the effect lasts for 4–5 years. Re-treatment every 3 or 4 years may be appropriate for long-lasting control.

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A copy of the final report will be made available on the website https://www.kauridieback.co.nz at a later date.