CONTROL OF *PHYTOPHTHORA CINNAMOMI* IN *ERICA SESSILIFLORA* AND *ERICA DAVISII*

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ABSTRACT

A field trial was conducted to test the efficacy of two fungicides (metalaxyl and phosphorous acid) and supplementary organic matter for the control of *Phytophthora cinnamomi* root rot in *Erica sessiliflora* and *E. davisii*. Five treatments were applied: (1) untreated control, (2) metalaxyl, (3) phosphorous acid, (4) metalaxyl plus phosphorous acid and (5) organic soil amendment. Plants were assessed for plant health as an indication of possible *P. cinnamomi* infection. Phosphorous acid, applied alone or in combination with metalaxyl, resulted in a significant reduction in the number of diseased or dead *E. sessiliflora* plants compared with the untreated control plants ($P=0.011$ and $P=0.004$ respectively). The mean health index of phosphorous acid treated *E. davisii* plants was not different ($P>0.05$) to control plants. Results suggest that this species of *Erica* has some tolerance to *P. cinnamomi*. Metalaxyl applications alone or organic matter treatments did not reduce disease relative to controls in either species.

Keywords: *Erica davisii*, *Erica sessiliflora*, phosphorous acid, metalaxyl, organic matter, *Phytophthora cinnamomi*.

INTRODUCTION

The domestic cut flower and foliage industry in New Zealand has an estimated value of \$60 million annually of which \$39 million is from exports (FreshFacts 2006). Ericaceous species are generally grown for their foliage in New Zealand and are often planted in association with Proteaceae plants as they require similar growing conditions. The production of flowers and foliage requires ongoing attention to combating both pests and fungal diseases. Soil-borne pathogens are of particular concern and can cause considerable revenue losses for the industry. Growers' management strategies for *Erica* species are similar to those used for *Protea* species in order to maintain quality production.

Phytophthora cinnamomi Rands is a widely distributed plant pathogen in New Zealand and is thought to have been introduced centuries ago along with the kumura (*Ipomoea batatis*) (Podger & Newhook 1971). Extensive infection is typically reported in areas with poor soil drainage and an annual rainfall greater than 500 mm, whereby repetitive water logging may induce host stress by root anoxia (Messenger et al. 2000; Robin et al. 2001). A neutral to acidic pH of the soil, low nutrient status and low organic matter are known to provide favourable conditions for the pathogen (Messenger et al. 2000). The main growing region of Ericaceous plants in the Bay of Plenty, New Zealand, exhibits many of these characteristics, and plant losses from *P. cinnamomi* are high. The implementation of control measures in this area is essential to reduce levels of infection.

Control of *P. cinnamomi* is currently sought by a combination of husbandry practices and chemical applications, which provide the essential components of integrated control (Crous et al. 2004). In the past, infested areas were treated with the soil fumigant methyl bromide. However, methyl bromide is now banned from use as a soil fumigant because it depletes the ozone layer. Other chemicals are now more heavily relied upon, particularly the systemic chemicals metalaxyl and phosphonates (Crous et al. 2004).

Metalaxyl, applied as a soil drench, reduces soil populations of the pathogen and inhibits spore germination. The addition of systemic chemicals has a prophylactic effect on the plants once they are infected. These fungicides, however, cannot protect plants against *Phytophthora* when there are high inoculum loads and disease-conducive soil and weather conditions (Crous et al. 2004). Mulches have also been shown to be effective in decreasing the level of *P. cinnamomi* in the field by creating a soil environment suppressive to *Phytophthora* (Menge et al. 1994; Messenger et al. 2000). In New Zealand, metalaxyl, phosphorous acid and mulch are all currently employed for the control of *P. cinnamomi* but with varying degrees of success.

This paper reports on the effects of soil and foliar chemical treatments and enhanced organic soil matter to protect *Erica* plants from infection by *P. cinnamomi*.

MATERIAL AND METHODS

A field trial was conducted from May 2005 to December 2006 in the Bay of Plenty with two test species, *Erica sessiliflora* and *E. davisii*. The trial was planted in a site previously infested with *P. cinnamomi* and used commercially for growing *Erica* species. Clonally propagated plants, approximately 15 cm tall, were tested for the presence of *P. cinnamomi* prior to planting out. Plants were apparently uninfested prior to planting.

The trial utilised a randomised block design with five treatments replicated four times. The treatments were (1) untreated control, (2) metalaxyl (25 g ai/kg; Ridomil®) incorporated into the soil at 50 g/m², (3) phosphorous acid (400 g ai/litre; Foli-R-Fos®) applied at 5.3 ml/litre to foliage to the point of runoff, (4) metalaxyl followed by phosphorous acid (applied as in (2) and (3)), and (5) organic amendment (composted pine bark obtained from Mt Maunganui wharf) applied to the soil at 10 litres/m².

The organic amendment and metalaxyl were incorporated into the soil with a handheld rotary hoe to a depth of approximately 15 cm prior to planting of the *Erica* species in May 2005. Phosphorous acid was applied to the foliage at monthly intervals from August 2005 until April 2006. Phosphorus acid was not applied during winter because of the risk of phytotoxicity when the chemical is applied in cooler, wetter conditions.

The trial area (48 m × 0.6 m) consisted of four blocks, each containing five treatment plots. Each plot (2.4 m × 0.6 m) contained 36 plants comprising four rows (of six plants) of *E. sessiliflora* (24 in total) and two rows (of six plants) of *E. davisii* (12 in total). The six plants (four *E. sessiliflora* and two *E. davisii*) at the interface between plots were used as buffers to minimise treatment contamination between plots. After final assessments of results, the six buffer plants were consistent with results obtained for the treatment within the plot and were included in the analysis.

As the two plant species responded differently to the treatments, they were assessed in different ways. The *E. sessiliflora* plants were generally assessed monthly on the basis of whether the plant was 'dead or diseased' or 'healthy'. Initially all dead or very unhealthy plants were removed and taken back to the laboratory to be tested for the presence of *P. cinnamomi*. Roots of the plants were surface sterilised and plated onto selective media and the identity of the fungus was confirmed as *P. cinnamomi* by morphological means. It was necessary to take plants that were very unhealthy for testing as it is very difficult to directly isolate *P. cinnamomi* from dead plant tissue. Confirmation was required as to whether the health of the plants was associated with *P. cinnamomi* infection. Plants that were 'very unhealthy' were included in the final dead plant score (hereafter called 'dead' plants). As the trial progressed and the number of dead plants increased, only randomly selected plants were chosen to confirm the presence of the pathogen.

The *E. davisii* plants did not show symptoms until after a severe frost in July 2006. After the frost the *E. davisii* plants were assessed three times: 28 October 2006, 1 December 2006 and 21 December 2006. The trial was terminated at the time of the last assessment. A scoring index that assessed the overall plant health was used: 0= healthy (no dead stems visible); 1= 1-24% stems dead; 2= 25-49% of the stems dead; 3= 50-74% of the stems dead; 4= 75-99% of the stems dead; 5= plant dead. The mean index of overall plant

health within each plot at each assessment was calculated by averaging the assessments for each plant. The number of plants with foliage (plants with less than 25% of stems dead) and the number of dead plants was collated at the final assessment.

Data analysis

Differences in the cumulative number of dead *E. sessiliflora* plants between treatments were tested using a one-way ANOVA followed by a two-tailed Dunnett test comparing treatments with the controls to locate significant differences (Payne et al. 2007). The proportion of the cumulative number of dead plants was arcsin-transformed to meet the test assumptions (homogeneity of variances) for ANOVA.

For *E. davisii*, plots of the mean index of overall plant health were calculated and means were separated using a one-tailed Dunnett test comparing with controls (SAS Institute Inc. 2004).

RESULTS

Erica sessiliflora

Dead and symptomatic plants were observed in the trial plot by December 2006, 7 months after the trial inception. Plants tested were confirmed to be infected with *P. cinnamomi*. At the end of assessments in September 2006 there were 215 dead plants from a total of 384 plants. The total number of dead plants for each treatment was: 83 for the untreated control, 49 for the 'metalaxyl only', 15 for the 'phosphorous acid only', 19 for the 'metalaxyl followed by phosphorous acid' and 49 for the 'organic amendment'. The proportion of the cumulative number of dead plants for the entire trial for all treatments differed significantly from the control ($P=0.017$). The Dunnett test indicated a significantly lower number of dead plants for both the phosphorous acid treatments ('phosphorous acid only' and 'metalaxyl followed by phosphorous acid', $P=0.034$ and 0.0124 , respectively) compared with the untreated control (Fig. 1). The proportion of dead plants in the phosphorous acid treatments was one-third of the other treatments (Fig. 1). The proportion of dead plants in the 'metalaxyl only' and 'organic amendment' treatments was not significantly different ($P>0.05$) from the untreated control.

FIGURE 1: The mean proportion of dead *Erica sessiliflora* plants (%) treated with metalaxyl, phosphorous acid, metalaxyl followed by phosphorous acid, organic amendment and an untreated control plot control. Values are the total number of dead plants in each plot at the end of the assessment period expressed as a proportion of the total number of plants per plot ($n=24$) and are presented as treatment means $\pm$ standard error of the mean (SEM). Significant differences between the control and treatments are indicated by * ($P<0.05$) according to the Dunnett test.

Erica davisii

Before the July 2006 frost, no plant death was observed in any of the 240 *E. davisii* plants and all plots scored 0 (healthy) in the mean plant health index. After the frost, there was a gradual decline of the health of the plants, characterised by tip death that progressed down the stems. This pattern is characteristic of *P. cinnamomi* infection. Most (95%) of the plants taken back to the laboratory for diagnosis were infected by *P. cinnamomi*. At the final assessment only 34 plants had foliage of saleable quality and 13 were dead. There was a progressive decline in plant health (Fig. 2). No significant differences ($P>0.05$) were found between the overall mean plant health of the untreated control plots and any of the treated plots, at any of the three assessment dates (Fig. 2).

FIGURE 2: Mean index of overall plant health (0=healthy to 5=dead) in *Erica davisii* treated with metalaxyl, phosphorous acid, metalaxyl followed by phosphorous acid, organic amendment and an untreated control plot at three separate assessment times.

DISCUSSION

The fungicide phosphorous acid gave *E. sessiliflora* plants some protection from *P. cinnamomi* infection. Soil-incorporated metalaxyl or organic matter gave no protection from *P. cinnamomi* infection in either plant species and these treatments cannot be recommended as integrated control strategies for the disease. The result for these *Erica* species is contrary to other reports that phenylamides, which includes metalaxyl, are the most effective chemicals generally for control of *P. cinnamomi* (Hardham 2005).

Previous studies have shown that phosphorous acid provides protection from *P. cinnamomi* by inducing resistance in the host (Guest & Grant 1991; Crous et al. 2004; Hardham 2005). Based on these results, the use of phosphorous acid on *E. sessiliflora* can be recommended to prevent infection by *P. cinnamomi*. In this trial, phosphorous acid was not applied during winter because of grower experience of phytotoxicity (R. Allen, pers. comm.) with this chemical during cooler, wetter conditions. The chemical has previously been reported to cause foliage damage in some species and careful monitoring of dosage rates and application times is required in sensitive plants (Barrett et al. 2002; Hardham 2005). High soil moisture is a key factor in the establishment, spread and longevity of *P. cinnamomi* (Robin et al. 2001; Hardham 2005), thus it is possible that the use of the fungicide over the winter in New Zealand would provide some protection to the plants during this period. It would be beneficial to know whether

the application of phosphorous acid every month of the year would result in greater survival of *E. sessiliflora* plants.

Prior to the frost there were no above-ground symptoms of *P. cinnamomi* in any of the *E. davisii* plants suggesting that *E. davisii* may exhibit a degree of tolerance to infection by *P. cinnamomi*. However, neither the fungicides nor the organic amendment inhibited symptom expression in plants by *P. cinnamomi* after they were damaged by frost. Crous et al. (2004) proposed that even plants that are resistant or tolerant to *P. cinnamomi* may still become infected with the fungus. The results of this study suggest that the plants of *E. davisii*, although symptomless, were infected by *P. cinnamomi* and that their tolerance was lowered by physiological stress caused by the frost, allowing the disease to progress.

Further work on different species of *Erica* and their potential tolerance to *P. cinnamomi* should be investigated. The use of disease-tolerant species could offer another alternative for the management of the disease in the Bay of Plenty area.

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