



Control of *Phytophthora cinnamomi* by the fungicide phosphite in relation to *in planta* phosphite concentrations and phytotoxicity in native plant species in Western Australia

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Abstract. Low volume aerial phosphite applications has been used in recent years at rates of up to 24 kg ha⁻¹ to protect native plant species and communities threatened by *Phytophthora cinnamomi* while the recommended rate for spray to run-off phosphite application is 5 g L⁻¹. Phosphite uptake and *in planta* phosphite concentrations in native plant species may vary considerably between species and with application rate as may the effectiveness of disease control and the duration of this control. Phytotoxicity symptoms post-spray include foliar necrosis, defoliation, growth abnormalities and chlorosis, reduced root growth and reproductive effects and these may also vary considerably between species. Phytotoxicity symptoms increase with increasing application rate but are generally mild at recommended rates. However, a percentage of species in the plant communities assessed show greater sensitivity to phosphite. Aerial phosphite application rates for native plant communities aims to maximise *in planta* phosphite concentrations in *Phytophthora*-susceptible species for disease control while minimising phytotoxicity symptoms in the species present.

Introduction

The soil-borne plant pathogen *Phytophthora cinnamomi* Rands is recognised as a key threatening process to Australia's biodiversity (Commonwealth Environmental Protection and Biodiversity Conservation Act 1999). Its impact is particularly devastating in the southwest of Western Australia (Wills and Keighery 1994). The fungicide phosphite has been used regularly in horticulture and agriculture for the control of plant pathogens from the genus *Phytophthora*. Research in recent years has shown that application of the fungicide phosphite, by stem injection or foliar spray, can control *Phytophthora cinnamomi* Rands in a number of native species (Shearer and Fairman 1991; 1997a; 1997b; Komorek *et al.* 1997; Ali and Guest 1998; Aberton *et al.* 1999; Tynan *et al.* 2001, Wilkinson *et al.* 2001). The term phosphite refers to the salts of phosphonic acid (H₃PO₃). Phosphite is a systemic fungicide translocated both in the phloem and xylem (Ouimette and Coffey 1990). In the phloem it is translocated through the plant along typical source-sink pathways (Ouimette and Coffey 1990).

Phosphite exhibits a complex mode of action, acting directly on the pathogen and indirectly in stimulating host defence responses to inhibit pathogen growth (Guest and Bompeix

1990; Guest and Grant 1991). Increasing phosphite concentrations have been generally found to correlate with better disease control (El Hamalawi and Menge 1995) however the presence or absence of phosphite in stem tissue may not strictly correlate with inhibition of *P. cinnamomi* growth (Hardy *et al.* 2001a). The ability of phosphite to control *P. cinnamomi* in native plant species may vary considerably between species, season of application and community (Hardy *et al.* 2001a). Plant tissue phosphite levels and control of *P. cinnamomi* decline over time and this rate of decline varies between species. Phosphite application aims to achieve optimal *in planta* phosphite concentrations for the control of *P. cinnamomi* while minimising phytotoxicity symptoms in target plant species. Although phosphite is considered to have low phytotoxicity (Guest and Grant 1991), symptoms including foliar necrosis have been recorded in a number of native species following its application (Komorek *et al.* 1997; Aberton *et al.* 1999; Ali and Guest 1998; Pilbeam *et al.* 2000; Barrett 2001, Hardy *et al.* 2001b;). In evaluating fungicides it is important to balance phytotoxic effects with the degree of control provided. Current low volume aerial application rates in Western Australia range from 12 to 24 kg ha⁻¹ with disease control anticipated for approximately two years post-spray at these rates. The recommended rate for high volume

spray to run-off applications and for trunk injection is 5 g L⁻¹. This review investigates factors, which may influence the selection of appropriate phosphite application rates for use in native plant communities.

Phosphite concentrations and control of *Phytophthora cinnamomi*

Phosphite may act directly on *P. cinnamomi* to inhibit growth or alternatively indirectly to enhance host defence responses. The concentration of phosphite in the plant may determine the relative importance of each mode of action (Afek and Szejnberg 1989). At low levels phosphite may increase the host defence mechanisms but at higher levels act in a fungistatic mode. Mode of action may also be related to whether target plants have well developed or poor dynamic defence systems (Smillie *et al.* 1989). Control of *P. cinnamomi* in native plant species has been achieved with foliar phosphite concentrations of 4.24 and 17.7 µg g⁻¹ dry weight. These concentrations reduced colonisation by *P. cinnamomi* of inoculated *Adenanthos barbiger* and *Daviesia flexuosa* stems, respectively, following high volume application at 2 g L⁻¹ (Pilbeam *et al.* 2000).

Control of *P. cinnamomi* in plant species in native plant communities following low volume aerial phosphite application has been achieved with *in planta* levels in the order of 5–50 µg g⁻¹ dry weight. However, the duration of control varied from one year at low levels to two years at higher concentrations (Komorek *et al.* 1997). Thus the longevity of disease control depends upon the initial phosphite concentrations achieved in plants post-spray. Faster growing young plants may lose their resistance to *P. cinnamomi* more quickly after phosphite application, presumably due to dilution of the chemical with increased plant biomass (Komorek and Shearer 1998). Low volume phosphite application at 12 kg ha⁻¹ to *Banksia telmatia* A S George seedlings resulted in leaf concentrations of 115.4 µg g⁻¹ two weeks post spray, one year later this had decreased to less than 7 µg g⁻¹ and then to a undetectable level at two years (Komorek *et al.* 1997).

In planta phosphite concentrations after low volume or spray to run-off applications may vary considerably between species sprayed at a fixed rate. Phosphite analysis of shoot samples from five shrubland species (*Adenanthos cuneatus* Labill, *Banksia coccinea* R.Br,

Jacksonia spinosa (Labill.) R.Br, *Lysinema ciliatum* R.Br., and *Melaleuca thymoides* Labill.) five weeks after low volume phosphite application at rates of 36, 72 and 144 kg ha⁻¹ showed a significant ($P < 0.01$) difference in *in planta* phosphite concentrations between species (Barrett 2001). *J. spinosa* had phosphite concentrations some 20 times higher than those of *A. cuneatus* at these rates. Similar differences in species uptake have been recorded in other native plant communities after high volume applications at recommended rates (Hardy *et al.* 2001b). For example the jarrah forest species *Leucopogon verticillatus* R.Br. had concentrations of 26 µg g⁻¹ compared with 299 µg g⁻¹ in *Hibbertia furfuracea* (DC) Benth after application at 5 g L⁻¹ (Tynan *et al.* 2001). Concentrations in *Daviesia decurrens* Meissner were generally 10 times higher than those of *Adenanthos barbigerus* Lindley sprayed at 2, 5 and 20 g L⁻¹ (Pilbeam *et al.* 2000). Comparatively high *in planta* phosphite concentrations and low phytotoxicity have been recorded in plants grown in glasshouse conditions compared with those of the same species growing in native vegetation (Hardy *et al.* 2001a). Stem phosphite levels in glasshouse grown *Banksia grandis* Willd. were 750 and 380 times greater than field-grown plants treated with 5 or 10 g L⁻¹, respectively (Wilkinson *et al.* 2001).

Colonisation by *P. cinnamomi* generally decreases with increasing phosphite application rate (Smith 1994; Wilkinson 1997; Jackson *et al.* 2000, Pilbeam *et al.* 2000; Barrett 2001). However, there is not always a clear relationship between application rate and disease control. In *A. barbigerus*, control of *P. cinnamomi* colonisation was similar when sprayed at 5 and 20 g L⁻¹ despite a 11-fold difference in *in planta* phosphite concentration while fungal colonisation decreased significantly as the application rate increased from 2 to 5 g L⁻¹ despite small differences in phosphite concentrations (Pilbeam *et al.* 2000). Control of *P. cinnamomi* in five native species, stem-inoculated and sprayed with 5 and 10 g L⁻¹ in a glasshouse study did not consistently correlate with phosphite concentrations in stems (Wilkinson *et al.* 2001). Extremely phytotoxic concentrations may also decrease the effectiveness of phosphite. For example, Shearer (pers. comm.) observed an increase in disease severity and plant mortalities in *B. coccinea* after trunk injection with 100 g L⁻¹ phosphite.

Similarly, colonisation of roots of *Xanthorrhoea preissii* Endl. by *P. cinnamomi* sprayed at 20 g L⁻¹ was more extensive than in plants sprayed with 5 g L⁻¹ while foliage showed extensive foliar necrosis (Pilbeam *et al.* 2000). It is possible that excessive phytotoxicity may inhibit phosphite translocation in the phloem (Groussal *et al.* 1986).

The duration of control of lesion growth in five native species in a glasshouse trial sprayed at 5 and 10 g L⁻¹ ranged from 6 to 18 months indicating that a single plant species cannot be used to determine the time for reapplication of phosphite (Wilkinson *et al.* 2001). Foliar application at 5, 10 and 20 g L⁻¹ to species growing in two native plant communities that were stem-inoculated post-spray, reduced colonisation for between 5 and 24 months depending on species and rate (Hardy *et al.* 2001a). Loss of control was associated with a marked decline in phosphite concentration between 6 to 12 months post-spray. After high volume phosphite application of 10 g L⁻¹ in autumn to *B. grandis* growing in jarrah forest, in planta phosphite was not detectable after 12 months although *P. cinnamomi* was controlled up to 24 months (Tynan *et al.* 2001). However, as the limit of phosphite detection was less than 50 µg g⁻¹, low levels of phosphite may still have persisted. At 12 months post-spray, phosphite was detected in inoculated stems of *Leucopogon verticillatus* growing in the same plant community but *P. cinnamomi* growth was no longer contained (Tynan *et al.* 2001). After phosphite application to *B. grandis* in spring at 10 and 20 g L⁻¹, phosphite was detected for up to 24 months post-spray but *P. cinnamomi* was only contained at 20 g L⁻¹. In contrast, trunk injection at 50, 100 and 200 g L⁻¹ to naturally growing wound inoculated plants of *B. grandis* and *E. marginata* resulted in disease control for up to four years (Shearer and Fairman 1997b). Phosphite applied at 6 g L⁻¹ controlled *P. cinnamomi* for at least two years in *Xanthorrhoea australis* R.Br. growing in infested vegetation. Percentage survival of *Sphenotoma* sp. Stirling (P G Wilson 4235) growing in infested vegetation following aerial application at 24 kg ha⁻¹ remained higher than in non-sprayed plants for up to three years post-spray (Barrett unpublished).

Phosphite concentrations and phytotoxicity symptoms in vegetative growth

Phytotoxicity symptoms may also vary considerably between species and application rate (Barrett 2001). In a study of 207 species sprayed at rates ranging from 24 to 144 kg ha⁻¹, plant families sensitive to phosphite included the Epacridaceae, Myrtaceae, Anarthriaceae, Proteaceae and Papilionaceae. However, trends within families and genera were not consistent (Barrett 2001). At the operational rate of 24 kg ha⁻¹, 92% of species showed either no symptoms or only mild symptoms over a 15-month study period. In a study of 18 plant species in the Northern Sandplains of Western Australia most species were unaffected by an application of 5 g L⁻¹ but applications of 10 and 20 g L⁻¹ caused damage to as much as 50 to 70% of foliage (Hardy *et al.* 2001a). Application of 5 g L⁻¹ generally caused less than 25% damage to the canopy in most species in *E. marginata* forest (Tynan *et al.* 2001). Necrosis of leaves was observed in 9 of 36 native plants in Victoria sprayed at 6 g L⁻¹ but in none sprayed at 2 g L⁻¹ (Aberton *et al.* 1999). High volume foliar application of phosphite at 5 g L⁻¹ caused burning of leaf tips in *Xanthorrhoea minor* R.Br. and *X. australis* (Ali and Guest 1998).

Recovery from foliar necrosis and defoliation also varies with application rate and species. At 7 months and 2 years after application, there was very little evidence of gross phytotoxicity symptoms remaining in Northern Sandplain species sprayed at 10 and 20 g L⁻¹. Following aerial application at rates above 24 kg ha⁻¹, *Nuytsia floribunda* rapidly shed and replaced foliage within a few months whereas other species had not fully replaced necrotic foliage by 15 months (Barrett 2001). Leaf regeneration in defoliated stems of *Banksia marginata* Cav. sprayed at 6 g L⁻¹ did not occur for up to two years post-spray (Aberton *et al.* 1999). Phosphite stimulated new growth in individuals of the resprouter species *A. barbigerus*, *D. decurrens* and *X. preissii* with high phytotoxicity ratings particularly at application rates of 5 and 20 g L⁻¹ (Pilbeam *et al.* 2000).

Plant deaths were recorded in *Astroloma xerophyllum* (DC) Sond and *Trymalium ledifolium* Fenzl sprayed at 5 and 10 g L⁻¹ (Hardy *et al.* 2001a). Phosphite applied at 5 and 10 g L⁻¹ also killed individuals of the annuals *Pterocheata paniculata* F. Muell. Ex. Benth., *Podotheca gnaphalioides* R. A. Graham and *Hyalosperma cotula* Benth with up to 60% and

90%, respectively of *P. gnaphalioides* plants dying (Fairbanks *et al.* 2001). The incidence of plant deaths in species from four plant communities sprayed at 24, 36 and 48 kg ha⁻¹ ranged from 0 to 10% (Barrett 2001).

Growth abnormalities, in particular rosetting of foliage, a reduction in leaf size, chlorosis and stunted or spindly growth were recorded after low-volume phosphite application at rates ranging from 24 to 144 kg ha⁻¹ (Barrett 2001). Of 207 species assessed, 32 % showed growth abnormalities and 36 % chlorosis. At the lowest rate applied of 24 kg ha⁻¹, the incidence of growth abnormalities ranged from 2 to 11 % in four plant communities. Growth abnormalities were more apparent in members of the plant family the Proteaceae while there was a trend towards a greater incidence of growth abnormalities in communities growing on low nutrient deep sandy soils (Barrett 2001). This may be due to an inverse relationship between plant nutritional inorganic phosphate levels and phosphite uptake (Carswell *et al.* 1996). Further research may reveal whether soil or plant nutrient levels influence phosphite uptake and metabolism.

Phytotoxicity symptoms general show a linear relationship with application rate. *In planta* phosphite concentrations in nine species sprayed at 36, 72 and 144 kg ha⁻¹ were significantly correlated with phytotoxicity symptoms (Barrett 2001). Even at the extremely phytotoxic rate of 144 kg ha⁻¹ (six times the recommended rate) certain species such as *A. cuneatus* showed minimal symptoms and relatively low phosphite concentrations while *J. spinosa* showed necrosis of more than 80% of canopy cover. Similarly, phosphite concentrations in the jarrah forest species, *A. barbiger* and *D. decurrens* showed a positive correlation with phytotoxicity symptoms after high volume application at 2, 5 and 20 g L⁻¹ (Pilbeam *et al.* 2000). Mild phytotoxicity was evident in *A. barbigerus* and *D. decurrens* with foliar dry weight phosphite levels in the order of 10 µg g⁻¹ however more severe symptoms occurred with concentrations in the order of 100 µg g⁻¹ or higher (Pilbeam *et al.* 2000). Komorek *et al.* (1997) reported severe phytotoxic symptoms in *Lambertia multiflora* Lindl. following low volume phosphite application at 18 and 36 kg ha⁻¹ that resulted in phosphite levels in plant tissues several weeks post-spray ranging from a few hundred to several thousand µg g⁻¹.

Mild phytotoxicity symptoms occurred with *in planta* phosphite concentrations in the order of 100 µg g⁻¹ in *D. tenuifolia*, *A. cuneatus* and *M. spathulata* sprayed at 36 L ha⁻¹, the severity of symptoms increased above this concentration (Barrett 2001). In *B. coccinea*, concentrations of up to 200 µg g⁻¹ resulted in mild symptoms in the majority of individuals (Barrett 2001). In contrast, shoots of *Corymbia calophylla* Lindley grown in a glasshouse showed only mild phytotoxicity (0 to 20% necrosis of the canopy) despite *in planta* shoot phosphite concentrations of 731 µg g⁻¹ in plants sprayed at the 'phytotoxic' rate of 96 kg ha⁻¹ (Barrett 2001). High phosphite concentrations of 1534 µg g⁻¹ were recorded in fine root material although shoots showed minimal foliar necrosis in glasshouse grown *C. calophylla* after low volume application of 24 kg ha⁻¹ (Barrett 2001). This data suggests that water deficit and osmotic stress may affect phosphite uptake in native vegetation and exacerbate foliar necrosis. Phosphite concentrations in necrotic foliage of *Eucalyptus redunca* were approximately four times higher than that of healthy foliage (Barrett 2001). Excessive foliar necrosis in target species is likely to result in direct loss of phosphite through defoliation or phosphite retention in necrotic foliage. In either case, a reduced quantity of phosphite may be available for translocation to root tissues.

Investigation of the relationship between phosphite phytotoxicity and selected plant characteristics showed that plant height, leaf shape, leaf hairs, the distribution and position of stomata relative to leaf surface and the presence of oil glands influenced phytotoxicity ratings (Barrett 2001). Growth form, leaf size, leaf orientation, fire response and the position of veins relative to the leaf surface were not related to phytotoxicity symptoms. This suggests that phosphite uptake may be influenced by species specific macroscopic and microscopic plant characteristics and this may explain some of the variation in *in planta* phosphite concentrations recorded in native species. Ultra-microscopic characteristics such as wax composition may also influence pesticide retention and uptake, as may cuticle thickness or a combination of all these factors.

Discussion

This review demonstrates the wide variation in initial phosphite uptake, phosphite decline post-spray, control of *P. cinnamomi* and

the duration of this control which may occur between species and plant communities. Phytotoxicity symptoms may similarly vary between species. While there is not always a clear relationship between *in planta* phosphite concentrations and disease control, in general control increases with increasing application rate. *Phytophthora*-susceptible target species such *Dryandra* and *Banksia* may have relatively low phosphite uptake compared with other members of the same plant community. To achieve optimal *in planta* phosphite concentrations for control of *P. cinnamomi* with aerial or high volume phosphite application in such species, plant health may be compromised in other non-target species. More frequent phosphite application at lower rates may optimise *in planta* phosphite concentrations while minimising phytotoxicity. However cost of application increases with more frequent application while potential phytotoxic effects on plant reproduction (Barrett 2001; Fairbanks 2001) may be exacerbated by, for example, annual application. Phosphite applied at current recommended rates may result in mild symptoms in the majority of species however some may show more severe symptoms. A test application with a hand-held sprayer prior to application is recommended to assess the range in sensitivity of the species present to phosphite and to assist in the selection of an appropriate rate. Timing of application should consider soil moisture levels and temperature to avoid undue osmotic stress.

Short-term phytotoxicity in non-target species may be acceptable as long as there are no long-term consequences for plant health and reproductive ability. Current aerial applications on the south coast of Western Australia target plant communities containing declared rare flora, which are 'critically endangered' or 'endangered' as a direct result of *P. cinnamomi* (Barrett pers. comm.). Management options are limited at the majority of these sites as most or all populations of these taxa are currently infested by the pathogen. Aerial phosphite application may maintain viable populations or allow time for other management actions to be undertaken such as the collection of seed or other germplasm. While enhancing plant survival is the primary consideration, phytotoxicity must not compromise disease control, cause plant death, or reduce the seed store. Monitoring of target species is recommended to ensure that seed banks and the maintenance of viable populations are not compromised. Further

research is required to determine the impact of repeated spraying in terms of phytotoxicity, the long-term efficacy of phosphite in enhancing plant survival and to develop optimal application methodologies in terms of plant phenology and environmental conditions. Research into its mode of action may hopefully lead to other more effective treatment options in the future. In conclusion, phosphite application remains the only method currently available to control *P. cinnamomi* in native plant communities, in particular those that are uniformly infested and contain threatened species. It can continue to be a valuable tool in the overall management of *P. cinnamomi* provided it is used with caution.

References

- Aberton MJ, Wilson BA and Cahill MJ (1999) The use of potassium phosphonate to control *Phytophthora cinnamomi* in native vegetation at Anglesea, Victoria. *Australasian Plant Pathology* **28**, 225-234.
- Ali Z and Guest DI (1998) Potassium phosphonate controls root rot of *Xanthorrhoea australis* and *X. minor* caused by *Phytophthora cinnamomi*. *Australasian Plant Pathology* **27**, 40-44.
- Ali Z, Smith I and Guest DI (2000) Combinations of potassium phosphonate and Bion (acibenzolar-S-methyl) reduce root infection and dieback of *Pinus radiata*, *Banksia integrifolia* and *Isopogon cuneatus* caused by *Phytophthora cinnamomi*. *Australasian Plant Pathology* **29**, 59-63.
- Barrett S (2001) Phytotoxic effects of phosphite in native plant communities in southern Western Australia. PhD Thesis, Murdoch University.
- Carswell MC, Grant BR, Theodorou ME, Niere JO and Plaxton WC (1996) The fungicide phosphonate disrupts the phosphate-starvation response in *Brassica nigra* seedlings. *Plant Physiology* **110**, 105-110.
- El Hamalawi ZA and Menge JA (1995) Methods of fosetyl-Al application and phosphonate levels in avocado tissue needed to control stem canker caused by *Phytophthora citricola*. *Plant Disease* **79**, 770-778.
- Fairbanks MM, Hardy G E StJ, Mc Comb JA (2001) The effect of phosphite on the sexual reproduction of some annual species of the jarrah forest of south-west Western Australia. *Sexual Plant Reproduction* **13**, 315-321.
- Guest D and Bompeix G (1990) The complex mode of action of phosphonates. *Australasian Plant Pathology* **19**, 113-114.

- Guest D and Grant BR (1991) The complex mode of action of phosphonates as antifungal agents. *Biological Review* **66**, 159-187.
- Groussal J, Delrot S, Caruhel P and Bonnemain J-L (1986) Design of an improved exudation method for phloem sap collection and its use for the study of phloem mobility of pesticides. *Physiologie Vegetale*. **24**, 123-133.
- Hardy GE StJ, Dell B and Colquhoun I (2001a) The potential of the fungicide phosphite to control *Phytophthora cinnamomi* in native plant communities associated with mining. Report M280. Minerals and Energy Research Institute of Western Australia. Minerals House, Perth, Western Australia
- Hardy GE StJ, Barrett, S and Shearer BL (2001b) The future of phosphite as a fungicide to control the soilborne plant pathogen *Phytophthora cinnamomi* in natural ecosystems. *Australasian Plant Pathology* **30**, 133-139
- Jackson TJ, Burgess T, Colquhoun I and Hardy GE StJ (2000) Action of the fungicide phosphite on *Eucalyptus marginata* inoculated with *Phytophthora cinnamomi*. *Plant Pathology* **49**, 147-154.
- Komorek B, Shearer BL, Smith BJ and Fairman R (1997) The control of *Phytophthora* in native plant communities. Final Report to the Threatened Species and Communities Unit, Environment Australia. Dept. of Conservation & Land Management, Perth.
- Komorek B and Shearer BL (1998). Refinement of techniques and identification of resources for the long term control of *Phytophthora* with phosphonate. In 'Control of *Phytophthora* and *Diplodina* canker in Western Australia'. Final Report to the Threatened Species and Communities, Biodiversity Group, Environment Australia' (Ed. D. Murray) pp.21-32. (Department of Conservation and Land Management: Perth.)
- Quimette DG and Coffey MD (1990) Symplastic entry and phloem translocation of phosphonate. *Pesticide Biochemistry and Physiology* **38**, 18-25.
- Pilbeam RA, Colquhoun IJ, Shearer BL and Hardy GE StJ (2000) Phosphite concentration: its effect on phytotoxicity symptoms and colonisation by *Phytophthora cinnamomi* in three understorey species of *Eucalyptus marginata* forest. *Australasian Plant Pathology* **29**, 86-95.
- Shearer BL and Fairman R G (1991) Control of *Phytophthora* species in native communities with phosphorous acid. In 'Proceedings of the Conservation Biology in Australia and Oceania Conference'. p. 72. (University of Queensland: Brisbane).
- Shearer BL and Fairman R G (1997a) Phosphite inhibits lesion development of *Phytophthora cinnamomi* for at least four years following trunk injection of *Banksia* species and *Eucalyptus marginata*. in 'Proceedings of the 11th Biennial Conference of the Australasian Plant Pathology Society'. p.181. (Australasian Plant Pathology Society: Perth).
- Shearer BL and Fairman RG (1997b) Foliar application of phosphite delays and reduces the rate of mortality of three *Banksia* species in communities infested with *Phytophthora cinnamomi*. In 'Proceedings of the 11th Biennial Conference of the Australasian Plant Pathology Society'. p. 180. (Australasian Plant Pathology Society: Perth).
- Smith BJ (1994) Effects of phosphonic acid and sodium silicate on lesion development of *Phytophthora cinnamomi* and histological responses in host species endemic to Western Australia. Honours Thesis, University of Western Australia, Perth.
- Tynan KM, Wilkinson CJ, Holmes JM, Dell B, Colquhoun IJ, McComb JA and Hardy GE StJ (2001) The long-term ability of phosphite to control *Phytophthora cinnamomi* in two native plant communities of Western Australia. *Australian Journal of Botany* **49**, 761-770.
- Wilkinson C (1997) The efficacy of phosphite *in vitro* and *in planta* in controlling the growth of *Phytophthora cinnamomi* and the production of sporangia and zoospores from lesions in *Banksia grandis* and *Eucalyptus marginata*. Honours Thesis, Murdoch University.
- Wilkinson CJ, Holmes JM, Dell B, Tynan KM, McComb JA, Shearer BL, Colquhoun, IJ and Hardy GE StJ (2001) Ability of phosphite applied in a glasshouse trial to control *Phytophthora cinnamomi* in five plant species native to Western Australia *Australasian Plant Pathology* **30**, 343-351.
- Wills RT and Keighery GJ (1994) Ecological impact of plant disease on plant communities. *Journal of the Royal Society of Western Australia*. **77**, 127-133