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PHOSPHITE AND ITS EFFECT ON LESION DEVELOPMENT AND PHENOLIC ACCUMULATION IN THE ROOTS OF CLONAL EUCALYPTUS MARGINATA, RESISTANT AND SUSCEPTIBLE TYPHOPTHORA CINNAMOMI, INOCULATED WITH PHOSPHITE TOLERANT AND SENSITIVE ISOLATES OF P. CINNAMOMI.

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INTRODUCTION
Phosphite inhibits the in vitro and in vivo growth of a number of Oomycetes, of which P. cinnamomi is particularly sensitive (1). However, P. cinnamomi isolates from the northern jarrah forest were found to vary significantly in their sensitivity to phosphite in vitro, with a 20 times difference between most and least tolerant isolates. The current study examined two isolates (most sensitive and most tolerant to phosphite) in order to determine the role of phosphite in protecting the roots of resistant and susceptible clonal jarrah from invasion by phosphite tolerant and sensitive P. cinnamomi isolates in the form of reduced lesion development and accumulation of phenolics.

MATERIALS AND METHODS
Resistant and susceptible clonal E. marginata plants were grown in aeroponics chambers to facilitate root growth. Fourteen days after foliar application of 0.5% and 1.5% phosphite, the roots were inoculated with a zoospore suspension of either the phosphite tolerant or sensitive P. cinnamomi isolate. The roots were harvested 4 days after inoculation. Lesion development and the accumulation of soluble phenolics were determined.

RESULTS AND DISCUSSION
Both the phosphite tolerant and sensitive P. cinnamomi isolates induced similar lesion development in the roots of non-phosphite treated E. marginata clones indicating a similar level of pathogenicity (Figure 1). Phosphite significantly restricted lesion development in the roots of P. cinnamomi resistant and susceptible clonal jarrah, with the reduction in lesion development proportional to an increase in phosphite concentration.

The phosphite sensitive isolate was unable to invade clones treated with 1.5% phosphite, however the phosphite tolerant isolate still induced small lesions (Figure 1). At 1.5% phosphite, the accumulation of phenolics remained similar to that of non-phosphite treated clonal plants (Table 1). In comparison, lesion development in the roots of plants treated with 0.5% phosphite was associated with an induction of phenolics. At 0.5% phosphite the sensitive isolate stimulated the defence mechanisms of both clones, however the tolerant isolate only stimulated phenolics in the resistant clone. The action of phosphite on the tolerant isolate was unable to stimulate phenolics in the susceptible clone.

CONCLUSION
The control of P. cinnamomi by phosphite in jarrah depends on a combination of the concentration of phosphite applied and the sensitivity of the pathogen to phosphite and natural resistance of host. When phosphite is applied at high concentrations it acts directly on the pathogen to inhibit mycelial growth, therefore restricting lesion development. At subtoxic concentrations, phosphite disrupts the fungal cell wall inducing a more rapid host defence response, thereby restricting lesion development.

REFERENCES