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INTRODUCTION

In the first 1-3 years temporary ponding of water can occur in rehabilitated bauxite mines in the south west of Western Australia. If P. cinnamomi is present, infection at the collar of E. marginata (jarrah) can occur. Under similar conditions, motile zoospores of P. cinnamomi have been shown to invade jarrah through apparently non-wounded periderm (1).

This study examines the disease development of P. cinnamomi in jarrah over a 12 month period. The role of isolate virulence and season of inoculation were examined in the survival of seedlings, and the persistence of the isolates.

MATERIALS and METHODS

Jarrah seedlings (17 months old) were inoculated in winter (June 1995) or spring (September 1995) in a recently rehabilitated mined area near Dwellingup (100 km SE of Perth), Western Australia.

Ponding was simulated by constructing a watertight receptacle around the lower stem of each seedling. Seedlings were inoculated by placing motile zoospores of either a highly virulent (HVI) or a moderately virulent (MVI) isolate of P. cinnamomi (2) into the receptacle. Inoculum and receptacle were removed 2 weeks later. Controls were sham-inoculated with sterile de-ionised water. The pathogen was recovered from harvested seedlings (3, 6 and 12 months after each inoculation) by plating the tissue onto a Phytophthora selective medium, and/or by baiting and re-plating the tissue when initial plating was unsuccessful.

RESULTS and DISCUSSION

Exposure of the non-wounded lower stem to motile zoospores of the HVI resulted in the death of 7 out of 51 seedlings, while inoculation with the MVI resulted in the death of only 1 out of 54. Of the 7 deaths that resulted from inoculation with the HVI, 5 of those seedlings were inoculated in spring. All deaths occurred between 3 to 6 months after inoculation.

The proportion of seedlings from which both isolates were recovered decreased with increasing time after inoculation (Figure 1).

There is evidence that both isolates went into dormancy within the host tissue, over the summer of 1996 (when xylem pressure potentials of -2.5 MPa were measured in inoculated as well as control seedlings). Firstly, recoveries of the isolates 12 months after the spring inoculation (September 1996) were slightly higher (but not significantly) than at the 6 month harvest (March 1996) (Figure 1). Secondly, baiting and re-plating of the harvested tissue (when initial plating was unsuccessful) yielded positive results only for seedlings harvested after the summer of 1996, before the winter rains (in 1996 rainfall was below average, until June). This suggests that the pathogen became dormant, and baiting of the tissue harvested in March and early June (dry period), or natural rehydration of the tissue in planta (after rain) probably helped break this dormancy.

Overall, recoveries of the pathogen were higher from winter inoculated than from spring inoculated seedlings (ANOVA, p = 0.04). The HVI was similarly virulent whether seedlings were inoculated in winter or spring. The MVI was recovered from a high proportion of winter inoculated seedlings, while recoveries from spring inoculated seedlings were consistently low (Figure 1).

![Figure 1. Proportion of jarrah seedlings from which the HVI and the MVI of P. cinnamomi was recovered.](image-url)