SEPTORIA LEAF BLIGHT IN YOUNG *EUCALYPTUS NITENS* PLANTATIONS

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Short-rotation stands of *Eucalyptus nitens* have been established in parts of New Zealand in order to provide a hardwood fibre component for quality paper production. However, young plantations growing in warmer, more humid parts of the country are seriously affected by a leaf blight disease caused by *Phaeophleospora eucalypti*. Studies were undertaken to determine how the disease develops, as a basis for possible management or control options. It was found that *E. nitens* produces foliage throughout most of the year, but with a brief lull in winter. New leaves appearing in spring are susceptible for only a short period, whereas conidia are produced on infected leaves throughout the growth season, while still retained. Under suitable conditions, and with adequate starting inoculum, an epidemic appears to develop as successive sets of leaves emerge, become infected, and continue releasing conidia, resulting in a sustained increase in available inoculum. Research is underway to define environmental criteria for the recognition of disease-prone sites where susceptible eucalypt stock should be replaced by resistant material in future plantations.

DOTHISTROMIN TOXIN AND NEEDLE BLIGHT OF PINES

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Pinus radiata is an important New Zealand plantation species. Young trees are very susceptible to Dothistroma (red band) needle blight caused by the fungus *Dothistroma pini*. There are currently serious outbreaks of Dothistroma needle blight overseas (e.g. in Canada and England) and similar outbreaks could occur here. A major concern is that the dothistromin toxin produced by the fungus could be hazardous to forestry workers under certain conditions, since it is structurally similar to the aflatoxin precursor, versicolorin. The current control method is fungicide drift spraying but there is pressure on forest industries to find alternatives. Development of new, effective, methods of disease control could depend on whether dothistromin toxin is a pathogenicity or virulence factor. In order to determine this, dothistromin (*dot*) genes were cloned and toxin-deficient mutants generated by targeted gene replacement. Pathogenicity testing of these mutants is in progress. Four clustered dothistromin (*dot*) genes have been characterised so far and are homologous to aflatoxin genes. One is a major facilitator superfamily transporter (*dotC*) and is a potential control target. Further elucidation of the biosynthetic pathway of the toxin could lead to discovery of other potential molecular targets for disease control.