Epidemiology and management of *Sclerotinia sclerotiorum* (Lib.) de Bary in kiwifruit (*Actinidia deliciosa* (A. Chev.))

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Abstract

Kiwifruit (*Actinidia deliciosa* (A. Chev.) C. F. Liang et A. R. Ferguson, var. *deliciosa* cv. 'Hayward'), a valuable fruit grown in New Zealand orchards for export, is susceptible to the necrotrophic and cosmopolitan fungus S*clerotinia sclerotiorum* (Lib.) de Bary. Sclerotinia disease causes direct crop loss in the form of diseased fruitlets, scarring of fruit and field rot of fruit. Costs to the industry amounting to an estimated $5 million pa. are incurred through crop loss on vines or during grading and through the purchase and application of fungicide.

This study showed that 19% (range 0–53%) of 3–4 day-old kiwifruit petals were colonised by *S. sclerotiorum* ascospores arising from apothecia that were present within most orchards. Adhering floral tissues (AFT) were present on 38% of fruit 7–8 weeks after anthesis and 11% (range 0–43%) of these were colonised by *S. sclerotiorum*. The incidence of fruit with scarring was significantly higher on fruit with AFT than fruit without AFT. Removal of AFT 10–14 days after anthesis resulted in a 66% reduction in diseased fruitlets and a 85% reduction in fruit with scarring. Adhering floral tissues on fruit were therefore determined to be a major source of secondary spread.

An *in vitro* assay was developed in which freshly detached kiwifruit petals were inoculated with dry *S. sclerotiorum* ascospores in a settling chamber and incubated for 72 h. The number of discrete colonies which formed on selective agar medium, when macerated petal tissue was spread on the agar, effectively quantified petal colonisation. Colonisation of petals significantly increased with increasing flower age. Colonisation of petals incubated under static conditions within saturated salt chambers was highest between 18–27^{o}C and 90–100% relative humidity. Colonisation of petals in dynamically controlled environment chambers was inhibited by incubation in diurnally fluctuating temperature and relative humidity conditions typical of days with <5 mm rainfall during flowering. The minimum, maximum and optimum temperatures for mycelial growth on PDA and percentage germination of ascospores on water agar (5^{o}C, 34^{o}C and 23^{o}C respectively) were similar to those for the rate of colonisation of petals by ascospores at 100% RH.

Symptoms of diseased fruitlets, fruit scarring and field rot were reproduced in field inoculation experiments, provided free moisture was present for at least 9 h. Sclerotia

were extracted from diseased fruitlets and fruit with field rot from three vines, yielding 2.7 and 18.6 sclerotia per unit respectively. Diseased fruitlets produced significantly smaller sclerotia, which had a higher germination rate and produced smaller apothecia, compared with sclerotia from fruit with field rot. External damage on 39% of sclerotia from fruit with field rot and their closer contact with soil micro-organisms during formation and maturation are likely causes for these observed differences in germination rate. Thus, the type of symptom which develops on pistillate vines can affect the potential for ascospore production during subsequent seasons through the production of ecologically distinct populations of sclerotia.

During field studies in 18 orchards, positive relationships were shown between primary inoculum source (apothecial density) and primary infection (incidence of petal colonisation) and between both these factors and the incidence of diseased fruitlets and fruit with scarring. A conceptual model of inoculum production and disease development was constructed. This model highlights the importance of primary colonisation of floral tissues and showed that disease risk can be estimated from the apothecial density or the colonisation of petals by *S. sclerotiorum*. Model structures have also been developed for predicting disease risk and disease incidence. Disease management strategies are discussed, and if implemented, would rationalise decision processes and potentially reduce the costs of controlling sclerotinia. Decision support software could be developed to incorporate such models once they are validated and combined with further research to determine effective control measures. This software could then be integrated into a sclerotinia management system for kiwifruit in New Zealand.

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Table of Contents

Table of figures

Table of tables

Table of plates