SCLEROTINIA INFECTION IN TWO KIWIFRUIT ORCHARDS IN THE BAY OF PLENTY

H.H. GOH and S.N. LYONS

Ruakura Agricultural Centre, Private Bag, Hamilton

SUMMARY

Incidence of *Sclerotinia sclerotiorum* infection on the buds, flowers and fruits were monitored from pre-blossom to harvest (late November to late April) in two adjacent kiwifruit orchards with different cultural practices. The levels of *Sclerotinia* infection in the two orchards were similar. Disease incidence and early fruit loss caused by *S. sclerotiorum* increased rapidly during late November to late February from 0 to 13% in Orchard A and 0 to 16% in Orchard B. The disease incidence after late February until harvest increased only gradually by 1 to 3%.

Keywords: disease monitoring, disease infection, *Sclerotinia sclerotiorum*, kiwifruit.

INTRODUCTION

Sclerotinia sclerotiorum (Libert) de Bary attacks a wide range of crops (Purdy 1979). On kiwifruit, *S. sclerotiorum* causes blossom rot and field rot of immature fruit (Pennycook 1982; 1985; Brook 1990). Under favourable moist conditions, white cottony mycelium and wet rot are observed on pre-blossom buds, flowers or fruitlets. The fungus forms sclerotia on the diseased parts which drop to the orchard floor where it continues producing more sclerotia. Overwintering sclerotia serve as a primary source of inoculum for subsequent infection during late spring to summer by producing apothecia and windborne ascospores.

Sclerotinia sclerotiorum reduces yields by causing fruit drop in the early season and increases rejection of fungal blemished or damaged fruit during packing. Information on the extent of immature fruit loss caused by this disease is scant. Pennycook (1985) estimated a 5% loss in the Te Puke district after a severe *Sclerotinia* infection period in late December 1980, but some growers have reported as much as 20% loss in some seasons. This disease has the potential to cause serious early fruit losses under favourable warm, moist environmental conditions during late spring to summer.

This paper reports on the monitoring of *S. sclerotiorum* infections in two adjacent kiwifruit orchards in the Bay of Plenty which have different cultural practices.

MATERIALS AND METHODS

Male and female vines from each kiwifruit block in two orchards were mapped. On 20 September 1991, 20 plots were marked in Orchard A and 14 plots in Orchard B with each plot consisting of four female vines. The two orchards with different cultural practices, such as summer pruning, low shelter belts, shelter height and density, and vine canopy, were monitored for disease infection.

Field assessment

Four shoots, each with five flower buds were tagged in each plot. The tagged shoots were assessed for *Sclerotinia* infection at two weekly intervals from pre-flowering until harvest (27 November 1991 to 30 April 1992). Fruit with shape and blemish defects were thinned by the orchardists at the end of January.

Stalks left on the vine by diseased flowers or fruitlets and shrivelled fruitlets were sampled on 9 January 1992 and plated directly, without surface sterilisation, onto potato dextrose agar (PDA) and incubated at 20°C under 12:12 L:D cycle to detect presence of *S. sclerotiorum*.

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Laboratory incubation

Four plant samples of flower buds, flowers, fruitlets and fruit from each plot were collected and placed in plix trays. Samples were placed on top of wet paper towels inside plastic bags and incubated at about 10°C to encourage fungal growth. Characteristic white cottony mycelial growth and presence of sclerotia of *S. sclerotiorum* on the plant materials were observed and recorded after 1 month. Cottony mycelium and sclerotia were isolated on PDA under aseptic conditions and incubated at 20°C to confirm identity of *S. sclerotiorum*.

RESULTS AND DISCUSSION

Development of *S. sclerotiorum* in both orchards was rapid during late November to February reaching 13% in Orchard A and 16% in Orchard B (Figure 1). From late February until late April the disease incidence for Orchard A increased to 14% and Orchard B to 19%. Early fruit loss followed the same trend, as the diseased fruit dropped after 1 to 2 weeks. The rapid increase in disease infection was attributed to germination of apothecia from overwintering sclerotia during the late spring and summer months (Manning 1991). In an orchard in the Bay of Plenty, apothecia were present during late October to mid-January, and by mid-February the apothecia had disappeared (Hoyte *et al* 1992). Both orchards had similar levels of *Sclerotinia* infection despite differences in cultural practices. This could be caused by cross infection between the adjacent orchards from disseminated airborne ascospores known to travel long distances (Abawi and Grogan 1979; Adams and Ayers 1979).

Fig. 1: Incidence of *Sclerotinia sclerotiorum* on flowers and fruits in two kiwifruit orchards in the Bay of Plenty.

The frequency of isolation of *S. sclerotiorum* from non-surface sterilised pedicels was 71% and shrivelled fruitlets was 50%. Other fungal contaminants isolated were species of *Cladosporium*, *Fusarium*, *Epicoccum*, *Penicillium*, *Alternaria*.

Symptoms and signs of *Sclerotinia* disease on incubated plant samples decreased from 22% in Orchard A and 12% in Orchard B on 27 November 1991 to no disease on 22 January 1992 (Figure 2). Samples collected after 22 January had no *Sclerotinia* infection. This showed that kiwifruit was susceptible to *S. sclerotiorum* during the pre-flowering bud, petal fall and early post blossom period coinciding with the production of apothecia and discharge of ascospores (Pennycook 1985; Hoyte *et al* 1992). During these periods, the presence of senescing flower parts assisted the

establishment of the ascospores (Pennycook 1985; Brook 1990). Control measures during this period proposed by Pennycook (1985) involved dislodging senescing female flower parts from newly set fruit with air-blast spray application, pruning male vines and removal of male flowers, and fungicide application to protect the fruitlets.

Fig. 2: Incidence of *Sclerotinia sclerotiorum* in field collected flowers and fruits incubated under humidity at 10°C.

Sclerotinia sclerotiorum is the cause of early immature fruit drop (Pennycook, 1982; 1985). Because of this and early fruit thinning later in the season, the importance of *S. sclerotiorum* in causing yield losses may be under-estimated by the kiwifruit industry. From our tagged samples, Orchard A had 14% *Sclerotinia* infection and 22% fruit thinning while Orchard B had 19% *Sclerotinia* and 30% fruit thinning.

Infected plant materials falling to the orchard floor will produce more overwintering sclerotia for next season infection. A severe disease epidemic may develop with greater cycling of sclerotial inoculum and ascospore loading during a warm, moist spring and summer resulting in higher disease incidence and early fruit loss. A greater understanding of the ecology and epidemiology of *S. sclerotiorum* on kiwifruit is important in order to develop suitable control measures using cultural and chemical (Pennycook 1985) and biological control agents (Goh and Lyons 1992).

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