CHAPTER 5

GENERAL DISCUSSION

The possible spread of citrus black spot from South Africa to Mediterranean countries was investigated. Existing evidence indicates that citrus fruit are susceptible to infection by ascospores form the teleomorphic state, *Guignardia citricarpa* Kiely, for 3-4 months after petal drop. Thereafter the pathogen lies dormant until maturity sets in (Wager, 1953; Kotzè, 1963; McOnie, 1964). Spells of wet weather are necessary for ascospores to germinate and infect the young fruit and leaves. Data on the prerequisites for *Phyllosticta citricarpa* (McAlp.) Van der Aa conidia to incite disease in citrus fruit or leaves are vague and inconclusive.

CBS lesions first appeared on naturally infected symptomless Valencia fruit after 5-7 days of incubation under optimal conditions (27 °C, high humidity and continious fluorescent lighting). Pycnidia containing viable conidia were visible after 9-13 days under optimal conditions. Unlike CBS lesion development and condium and spermatium production in culture, (Brodrick & Rabie, 1970) light had no effect on condial germination by *P. citricarpa*, while the optimal germination temperature, 22 °C, corresponded with the optimal temperature for conidium production in culture. During the 30-40 days it takes for shipment and marketing of fruit conditions for CBS lesion development are suboptimal and it is doubtful if new lesions with viable conidia could develop in this period. Wager (1948) showed that lesion development at 4.4 °C was significantly slower than at 29.4 °C. According to him, *P. citricarpa* conidia do not appear to have a long live-span but the present results showed a tendency for conidia to stay longer viable at cooler storage temperatures (4.5 °C and 11 °C). Artificial inoculation with *P. citricarpa* conidia proved unsuccessful at 0.5 °C on wounded and unwounded fruit.

A combination of packhouse treatments and storage of fruit eliminated *P. citricarpa* conidial inoculum on CBS fruit, although mycelium in the periphery of viable lesions survived. Fruit are exported under cool (10 – 11 °C) conditions (Venter & Cook, 1998). There is thus little opportunity for pycnidia with viable conidia to develop from surviving mycelium on packhouse-processed fruit during shipment and marketing.

Existing packhouse procedures and the fungicides utilised in South Africa, with the exception of imazalil sulphate, reduced viable mycelium and eliminated conidia of *P. citricarpa* from fruit. Combinations of factors are employed in packhouses to reduce decay (hurdle technology) and it is therefore unlikely that imazalil sulphate will be applied as the only postharvest treatment in a packhouse, and the inability of the compound to control conidial germination is no reason for concern. Chorine in the receiving bins is sufficient for reducing the *P. citricarpa* condial inoculum on fruit. The warm water bath and subsequent postharvest chemical treatment will eliminate the remaining condial inoculum and reduce viable mycelium significantly. The only CBS lesion type capable of producing viable conidia in quantities sufficient to cause infection were red margin hard spots (Chapter 2, Fig.9). Mycelium in the periphery of lesions showing red active growth (freckle spots, virulent spots and red margin hard spots), unlike conidial inoculum present in these lesions, can survive the packhouse treatments.

It was further demonstrated in artificial and natural inoculation studies that *P. citricarpa* conidia could not infect healthy mature packhouse-treated oranges. This is in accordance with findings of Wager (1953) that infected CBS fruit cannot transfer the disease to healthy mature oranges. A low percentage infection occurred on wounded fruit artificially inoculated with *P. citricarpa* conidia and stored for 4 weeks at 25°C and high humidity. These conditions do not occur under export conditions and there is therefore little chance of cross-contamination between infected and clean fruit.

Even though remote possibility exists for *P. citricarpa* conidia on CBS-infected fruit to infect wounded citrus fruit during shipment, the requirements for the onset of a CBS epidemic is not met. The disease must spread from the rind of imported infected fruit to

intact citrus leaves in recipient countries free of the disease. This step is crucial because the teleomorphic state, *G. citricarpa*, develops only on pre-infected decaying citrus leaves on the orchard floor (Kiely, 1948; McOnie,1964; Kotzé, 1981). The presence of a summer rainfall climate is a further prerequisite for the onset of a CBS epidemic (Kotzé, 1981; Schutte, 1996). Mature intact Valencia leaves could not be inoculated through artificial inoculation with *P. citricarpa* conidia (Chapter 2). According to Wager (1948), young citrus leaves are susceptible to infection by *P. citricarpa* conidia, but this statement is contradicted in findings where he had sprayed leaves with viable *P. citricarpa* conidial suspensions during the first few months after petal drop without achieving infection of mature or young leaves (Wager, 1953).

It is undeniable that in the past 40 years citrus fruit infected with CBS were exported to European countries where the disease is absent. During this time no transference of the disease occurred. Results made available through this study demonstrated that for the CBS pathogen to spread from infected fruit to orchards where the disease is absent, is highly unlikely if not impossible.

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