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The significance of *Citrus* spp. fruit as a pathway for the introduction or spread of *Elsinoë australis*, the organism that causes Sweet orange scab disease

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Executive Summary

This analysis considers scientific, expert, and empirical evidence regarding the significance of citrus fruit as a pathway for the introduction or spread of Sweet orange scab (SOS), a fungal disease of citrus caused by *Elsinoë australis* Bitancourt and Jenkins.

The analysis concludes that:

- Asymptomatic fruit is not a pathway for the introduction or spread of the disease.
- Symptomatic fruit that is commercially packed is not epidemiologically significant as a pathway if washed with a surface disinfectant and treated with fungicides during packing.
- Symptomatic fruit that is not washed and fungicide treated could be epidemiologically significant under unusual conditions.

The implementation of a disease management program reduces the incidence of SOS infected fruit in the field. Fruit without symptoms cannot transmit the disease. Commercial packinghouse procedures that include culling, washing with a surface disinfectant, and surface treatment with a fungicide-wax destroy fruiting bodies on symptomatic fruit and inhibit further sporulation. The potential for establishment is further reduced by the unique set of conditions that must be met for infection to occur during the 1.5 hours that any spores which may be produced will survive. Untreated fruit is not likely to be a pathway unless large quantities of highly symptomatic fruit are concentrated near susceptible host material under the proper conditions.

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I. Introduction

This document was prepared by the Plant Epidemiology and Risk Analysis Laboratory (PERAL) of the Center for Plant Health Science and Technology (CPHST), USDA Animal and Plant Health Inspection Service (APHIS), Plant Protection and Quarantine (PPQ) in partial response to the outbreak of Sweet orange scab disease in commercial citrus production areas of Texas, Louisiana, and Mississippi.

The objective of this document is to summarize the significance of citrus fruit as a pathway for the introduction or spread of Sweet orange scab. The focus is on fresh citrus fruit from infested areas that is commercially cultivated, harvested, packed, and distributed in the United States or for export from the United States. The analysis distinguishes between industry practices associated with fruit for retail consumption and shipments for processing (usually juicing). Non-commercial shipments of fresh fruit, dried or processed fruit, citrus leaves, flowers, and plants are beyond the scope of this analysis.

II. Characteristics of the organism

Sweet orange scab disease (SOS) is caused by the fungus *Elsinoë australis* Bitancourt and Jenkins (Ascomycetes, Dothideales) and its alternate state (anamorph) *Sphaceloma australis* Bitancourt and Jenkins. The organism has two stages: a sexual stage represented by the ascospores of *Elsinoë australis* and an asexual stage represented by the conidiospores of *Sphaceloma australis*. These two stages are produced at different times and under different conditions.

Elsinoë stage:

The sexual stage is characterized by the ascomata which is pulvinate, globose, dark, pseudoparenchymatous, multi-locular, up to 80-120 µm thick. Up to 20 asci are formed per locule. Asci are subglobose or ovoid, bitunicate, with the inner wall thickened at the top, 12-16 µm diameter, eight-spored. Ascospores are hyaline, ellipsoidal or oblong-ellipsoidal, with two to four cells, usually constricted at the central septum, 10-12 x 5-6 µm diameter (12-20 x 4-8 µm for *E. australis*). This stage is only known from Brazil (CABI, 2007; EPPO, 1997). The role of ascospores (sexual spores) in the infection process remains uncertain (Chung and Timmer, 2005).

Sphaceloma stage:

The asexual stage is characterized by the acervuli which are intra-epidermal or sub-epidermal, scattered or confluent, pseudoparenchymatous, produced in infected plant organs (fruit, leaves, twigs). Conidiogenous cells originate from the upper cells of the pseudoparenchyma or from the hyaline or pale-brown phialidic conidiophores, which have 2-4 septa. Conidia are hyaline, unicellular, ellipsoid, biguttulate, 5-10 x 2-5 µm.

Conidia are formed abundantly on wet scabs, in a nearly saturated atmosphere, between 20° and 28° (CABI, 2007; EPPO, 1997). Conidia are fragile and ephemeral, maintaining viability for approximately 1.5 hours. Lesions can produce spores for an extended period of time, but the number of spores released declines as lesions age (USDA, 2010).

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The determining factor in the development of disease is the presence of free water on the surface of pustules, since the conidia are produced in only 1-2 hours after a short rain and they need least 2-3 hours of continuous moisture to complete germination and infection of the tissues on a susceptible host (Hernandez and Mendes, 2003).

Elsinoe fawcettii, a closely related organism causing Sour orange scab on citrus, produces spindle-shaped conidia (2 -3 x 10 - 15 µm) on host tissue; *E. australis* does not. Both species propagate primarily via conidia, which are produced in acervuli on the edge of scab pustules (Chung, K-R., 2010). Conidia are capable of reproducing by budding (Bayer Crop Science, 2008).

Elsinoë australis is known to occur in tropical and subtropical regions with abundant rainfall in the summer, including Argentina, Bolivia, Brazil, India, South Korea, Paraguay, Uruguay, Cook Islands, and Fiji (Farr and Rossman, 2010; CABI, 2010, EPPO, 1997; Hyun, 2007). The disease was recently detected in the United States (Texas, Louisiana, and Mississippi).

III. Symptoms

SOS is primarily a disease of fruit (Bitancourt and Jenkins, 1937; Timmer et al., 2000), although leaves and stems are also infected (CABI, 2010). The susceptibility of citrus varies with the species and cultivar. Hosts of *Elsinoë australis* anamorph *Sphaceloma australis* include: *Citrus aurantiifolia*, *C. aurantium*, *C. deliciosa*, *C. hystrix*, *C. limon*, *C. limonia*, *C. paradisi*, *C. reticulata*, *C. sinensis*, *C. unshiu*, and *Fortunella margarita*, (Farr and Rossman, 2010; CABI, 2010, EPPO, 1997), *C. nobilis* (USDA-APHIS-PPQ, 1982), *Citrus natsudaidai* (Hyun et al. 2001).

Lesions on leaves of sweet orange are rare. It was once believed that the leaves are entirely free from the disease (Bitancourt 1937). As a result of a very thorough examination of a navel orange grove in Limeira, Brazil during one of the most severe attacks of the disease, a number of mildly scabbed leaves were found in the inner part of the tree (Bitancourt 1937). SOS leaf lesions are typically found on the lower surface, chiefly on the midrib. Recent samples in the United States exhibit lesions mainly on the margins of the upper side of leaves; lesions are relatively large and coalesced. They seldom form protuberant outgrowths as on the fruits, although in very young leaves they sometimes form funnel-like pockets similar to those produced in sour orange scab. The leaf lesions are smooth, and frequently have a somewhat glossy surface. Their color agrees with that of the fruit lesions, except that they may be bordered by a narrow brown line. The lesions, with the exception of those along the midrib, seldom attain 2mm in diameter. In general, the leaf lesions of sweet orange scab are smaller and less elevated than those of sour orange scab (Bitancourt, 1937).

Fruits are highly susceptible to *Elsinoë australis* during the six to eight weeks after 'petal fall,' (Timmer et al., 2000). Fruits are infected in the early stages of their development, grow misshapen and are subject to premature fall. On the rind of developed fruits, raised lesions are formed with different shape, size and color according to the species and cultivar affected. They appear as scattered protuberances, conical projections or crater-like outgrowths, or they coalesce to give scabby patches or extensive areas of fine eruptions. Typical lesions on young fruit are

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round to irregular, 2 to 6mm in diameter, raised and slightly convex (Bitancourt and Jenkins, 1937). Rind symptoms of initial infections typically display slightly raised and pink to light brown scab pustules. As the scab or pustules develop they become warty and cracked and yellowish brown to dark gray in color (Bitancourt and Jenkins, 1937; Timmer et al., 2000). Lesions appear as scattered protuberances, conical projections or crater-like outgrowths, or they coalesce to give scabby patches or extensive areas of fine eruptions. Scab lesions do not extend into the albedo (CABI, 2010).

Elsinoë australis generally forms larger, smoother, more circular scabs than *E. fawcettii* scabs, which are typically irregular, warty and deeply fissured. Both species form intra-epidermal or sub-epidermal pseudoparenchymatous acervuli from hyaline short-branched mycelium (Bayer Crop Science, 2010).



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Figure 1. *Elsinoë australis* lesions on the surface of orange fruit

IV. Epidemiology

Plant parts affected by *E. australis* are fruits, flowers and inflorescences, leaves, and stems (CABI, 2007, Bitancourt and Jenkins, 1937, Sivanesan and Critchett, 1998, Timmer et al., 2000). Symptom development starts a few days after infection and is dependent on environmental conditions that promote disease development, e.g. warm temperatures and moist plant tissues. The incubation period is at least 5 days (CABI, 2007, FDCA/DPI, 2003). Artificially inoculated seedlings develop scab symptoms after 7 to 14 days (Timmer, 1996).

The best time to detect disease in the field is during early spring when new tissues and fruit are forming. Symptoms generally develop one week after tissue infection (USDA, 2010). There is a dramatic increase in the resistance of the leaves and fruits to infection in later stages (INTA, 2010). Once established in an area, *E. australis* can spread readily to nearby hosts in the natural environment with adequate rainfall, temperatures, and inoculum. Long distance dissemination of

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E. australis is most likely through the movement of infected nursery stock, including budwood (CABI, 2010).

Leaves are susceptible to infection when young (flush stage), primarily in the early spring as they emerge from the bud and 'petal fall' commences; thereafter they become immune to infection (Timmer et al., 2000). When lesions do develop on leaves, they typically remain small (2 mm diameter) (Bitancourt and Jenkins, 1937). A protuberance is formed on leaf tissue where the infection develops due to induction of cell hyperplasia, and on the opposite side, a depression is formed. In addition to the pustules, the leaves exhibit distortions, but if infections develop close to when the leaf becomes resistant or immune, the pustules are smaller and no distortion of the leaf blade occurs (Hernandez and Mendes, 2003). There is a dramatic increase in the resistance of the leaves and fruits to infection in later physiological stages (INTA, 2010).

During the six to eight weeks after 'petal fall,' fruits are highly susceptible to *E. australis* (Timmer et al., 2000, Chung and Timmer 2005) or up to 12 weeks after petal fall in Brazil (Hardy, 2004; Prates, 2007). Infected fruit readily express symptoms after infection but tissue susceptibility decreases rapidly as fruit mature (USDA, 2010). Fruits infected in the very early stages of their development, grow misshapen and are subject to premature fall (CABI, 2007).

Conidia dispersal is dependent on rain and/or overhead irrigation (CMI, 1998; EPPO1997; Timmer et al., 2000), and may be dispersed over short distances by droplets in air (Chung and Timmer, 2005). The spores can be spread while adhering to windblown water droplets, but dispersal is mostly within the tree canopy of origin (Kucharek and Whiteside, 200). The dispersal gradient is therefore short. Based on a study conducted by Whiteside (1975), the estimated spread rate for *E. fawcettii* in Florida groves was no more than 100 to 300 meters per year.

Elsinoë australis overwinters in the tree canopy on limbs and fruit that were infected during the previous season. The pathogen will survive if existing scab pustules retain fruiting bodies (EPPO/CABI, 1997). Conidia are formed abundantly on wet scabs (CABI, 2006). Conidial germination can occur between 13-32°C, and infection between 14-25°C (EPPO/CABI, 1997) with an optimum temperature between 24-29°C (Bitancourt and Jenkins, 1937). Germination of conidia and infection do not require rainfall, however, a minimum period of wetness, via dew, fog, or other high moisture conditions of 2.5 - 3.5 hours, is necessary for conidial infection (CABI, 2006; EPPO/CABI, 1997). When these favorable conditions are present, infection and further conidial production may occur within 4-6 hours, as is the case with the closely related species *Elsinoë fawcettii* (Whiteside, 1975). Once infected, *Elsinoë* spp. reproduces rapidly inside the lesions and initiates new infection if environmental conditions are conducive and if susceptible hosts with young tissues are available. Infection by *Elsinoë* spp. occurs mainly during the spring flush and sporadically during the summer flushes (Chung, 2010).

A climate risk model developed by Magarey using NAPPFAST (model output available on NAPPFAST) examined the likely areas that *Elsinoë australis* could establish in the United States. The pathogen requires moderately warm temperatures (4-25°C) and a minimum leaf wetness period of 2.5-3.5 hours for infections to occur. The model assumes the presence of inoculum. The infection model parameters used for *E. australis* were $T_{min} = 7^{\circ}C$, $T_{max} = 32^{\circ}C$, $T_{opt} = 26$, $W_{min} = 2.5h$, precipitation requirement = 2.0mm. (Thayer et al., 2003).

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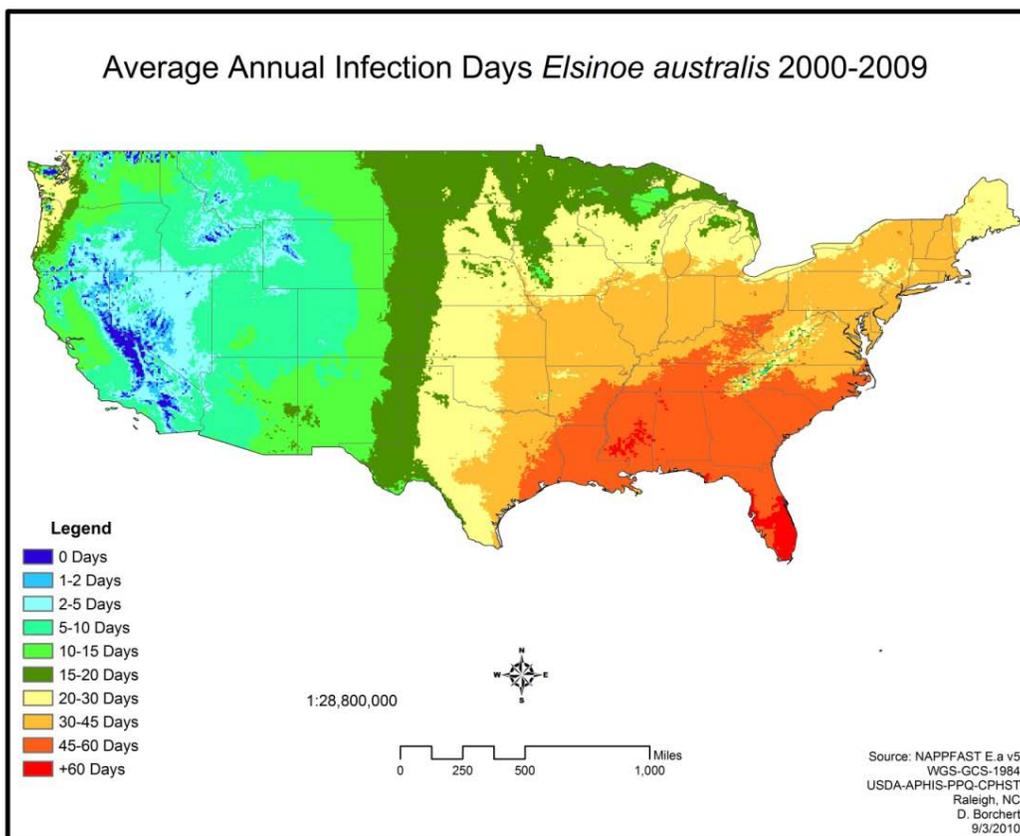


Figure 2. Average annual infection period of *E. australis* for ten years (2000-2009).

Because of the critical role of moisture for spore production and infection to occur, the disease is unlikely to become established or spread in areas with limited annual rainfall (less than 1300mm), warm temperatures (mean monthly temperature above 24°C) and dry climates like Arizona and California (EPPO/CABI, 1997). The disease is more likely to become established in the humid southeastern United States, particularly Florida, which has the proper environmental conditions (Bushong and Timmer, 2000; EPPO/CABI, 1997). Suitable microclimates may occur in limited areas within generally unsuitable regions. While it may be more likely for establishment to occur under these unique circumstances, it is unlikely that spread will occur due to the lack of contiguous conditions in the surrounding area.

V. The significance of asymptomatic fruit as a pathway

The incubation period (from spore infection to symptom expression) could be up to fourteen days and fruit is only susceptible to infection for up to eight weeks after petal fall. As a result, any fruit that is infected will express symptoms during the first 10 weeks of development. Since all citrus requires more than 3 months for fruit to develop and mature, infected fruit will show symptoms well before they are harvested. If mature fruit does not show scab symptoms at harvest, it is not infected and therefore has no possibility to be a source of inoculum for the spread of SOS. Thus, asymptomatic fruit is not a pathway for the introduction or spread of SOS.

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VI. The significance of symptomatic fruit as a pathway

Early infected susceptible fruit (up to 6 -8 weeks of age) readily express symptoms after infection. Knowing that the development period (from spore infection to symptom expression) could be up to 14 days, all fruit infected at susceptible stages will show symptoms by the time they are harvested. Symptoms are variable and may be more or less evident depending on the variety of citrus and environmental conditions. Severely diseased fruit is likely to be left in the field during harvest or culled in the packinghouse due to appearance. Lightly infected fruit or fruit having atypical lesions such as those often associated with injuries and other blemishes which do not strongly affect appearance, are more likely to escape culling. Thus it is highly likely that infected fruit will be found in commercially packed shipments. The outstanding question is whether this fruit is able to serve as the mechanism for transmission of the disease to susceptible hosts leading to establishment in a new area.

In the absence of any control measures, it is reasonable to assume that some infected fruit will be shipped to areas where suitable conditions exist and susceptible hosts are available (see Figure 1). Lesions on fruit that has been harvested will be at least several months old, and removed from the natural wetting cycles in the field that are required to stimulate sporulation. Because acervuli are surface structures, those that are present and active at the time of harvest are likely to be damaged or destroyed in the packing process. This would be expected to substantially reduce the quantity of spores which may be produced by lesions on infected fruit. An increase in sporulation would require the requisite temperatures and wetting events to stimulate acervula/conidia formation. Although germination of conidia and infection do not require rainfall, a minimum wet period, via dew, fog, overhead irrigation or other high moisture conditions of 2.5 - 3.5 hours between 13-32°C is necessary for conidial infection (CABI, 2010; EPPO, 1997). When these favorable conditions are present, infection and further conidial production may occur within 4-6 hours as is the case with the closely related species *Elsinoë fawcettii* (Whiteside, 1975).

The primary window for infection is the early spring, when the tree puts out new shoot growth and 'petal fall' commences. Leaves are susceptible as they emerge from the bud but become immune to infection as they mature and harden. Fruits are highly susceptible only during the six to eight weeks after petal fall. Asynchrony of the harvest/shipping season and the flush/flowering season make it unlikely that susceptible fruit host material will be available at the time most fruit is moving in commerce. It is recognized however that flushes may occur throughout the growing season depending on temperature, moisture, and fertility. Plants in nurseries are often forced to flush continuously throughout the growing season and may therefore be in a susceptible stage throughout the year. Likewise, the harvest season for fruit can vary widely depending on the variety and growing conditions; and citrus fruit may also be stored for short periods which further extends the time when fruit are available for distribution. Although unlikely under most circumstances, susceptible foliage could be available in citrus growing areas where off-season flushing occurs.

For infected fruit to transmit *E. australis* to susceptible hosts, they must be taken or placed in the vicinity of growing plants under the conditions of temperature and wetness which would allow for conidia production and infection. The infectious material and the susceptible host must be close enough to allow for the movement of a sufficient number of viable conidia from the infected fruit to the susceptible host in water droplets. Considering that conidia are relatively

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fragile and survive less than two hours, the conditions for successful transmission from individual lots of fruit for consumption are highly unlikely except under the most contrived circumstances.

One such scenario would be the shipment of fresh bulk fruit that is severely diseased (minimal field selection, minimal culling, and no treatment) to a citrus packaging or processing facility where large quantities of fruit may be concentrated outdoors nearby citrus nurseries, groves, or ornamental plantings. Under ideal conditions of temperature, moisture, and wind, sporulation may be stimulated and inoculum spread to susceptible plants.

VII. Control measures

The analysis above assumes that commercially produced fresh fruit has not undergone any treatment either in the field or packinghouse. It is assumed that infection of the fresh fruit is likely if fruit originates in areas where *E. australis* occurs but the magnitude of the hazard depends in large part on the proportion of infected fruit. The incidence of infected fresh fruit depends primarily on the citrus species and variety, environmental conditions, and field management. This section describes common control practices.

There are several effective field management programs that reduce the incidence of citrus scab, including SOS, in the field. Copper-based fungicides may be used to control citrus scab, but may not be as effective in heavily infected groves (Timmer and Chung, 2007b). Garrán (2006) indicates that the number of fungicide applications to control citrus scab will depend on the susceptibility of the crop and the field history. In areas with old leaves that are heavily infested three are sprays recommended for citrus scab control. If there is little carryover of the disease from one season to the next then only two sprays are typically necessary (Timmer and Chung, 2007). In countries where SOS is endemic, two fungicide sprays are applied; one at 2/3rd petal fall followed by a second application 2 to 3 weeks later (Chung and Timmer, 2005). On susceptible cultivars three spray treatments are advised (flower formation, petal fall, and fruit formation) while on less susceptible cultivars two are recommended (petal fall and fruit formation). Fungicides such as carbendazim, benomyl¹, thiophanate methyl, pyraclostrobin, trifloxystrobin, azoxystrobin are efficacious in controlling citrus scab followed by ziram, ferbam, mancozeb, and difenoconazole with intermediate efficacy and cupric-base products with low efficacy.

Barrier zones around export groves help to reduce the spread of the pathogen from one registered grove to another. Copper oxychloride sprays during new spring growth and flowering protect the young leaves and fruit by preventing the spores from germinating and causing infection (Timmer et al., 2000; Whiteside, 1975). Other chemical treatments may be used in addition to copper, benomyl, fenbuconazole, and azoxystrobin. All are effective as protectant and post-infection sprays for management of citrus scab (Bushong and Timmer, 2000). Products such as thiophanate methyl, azoxystrobin, trifloxystrobin, pyraclostrobin, ferbam, and copper fungicides are registered in Florida for citrus scab and presumably would be effective for SOS management (<http://edis.ifas.ufl.edu/CG020>).

¹ Benomyl is no longer listed for use as a post-harvest treatment for citrus fruit.

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Post-harvest treatments are effective in reducing the viability of pycnidiospores in lesions on symptomatic fruit with Citrus black spot, another fungal disease causing lesions on citrus fruit (Korf et al., 2001). Routine packinghouse treatments such as chlorine dips, warm water bath, or chemical tank dip (1000 µg/ml guazatine, 503 µg/ml imazalil sulphate, 500 µg/ml 2,4-D sodium salt) and combinations of these treatments reduce the viability of the conidia to zero on fruit kept at 4.5°C-25°C (Korf et al., 2001). In addition, wax treatments will further reduce the viability of conidia (Korf et al., 2001; Seberry et al., 1967).

Imazalil (IMZ) and thiabendazole (TBZ) are systemic fungicides that also have a trans laminar effect as post-harvest surface treatments for several fungi including *Diplodia*, *Alternaria*, *Fusarium*, *Botrytis*, *Molinia*, *Rhizoctonia*, and apple scab among others (Ware and Whitacre, 2004). Thiabendazole has a range of activity similar to that of benomyl (Ware and Whitacre, 2004).

Studies on the distribution of postharvest fungicides on/in citrus fruit showed that residues are predominantly found on the surface of the fruit and in the peel and only small amounts migrate into the flesh (Friar and Reynolds, 1994). Systemic fungicides (benomyl, carbendazim) are commonly used before flushing and after petal fall (González, 1980; Reddy et al., 1983; Canteros 1998). Benomyl has been used for the control of common scab. Benomyl was highly effective as a protectant against citrus scab and as an eradicant of the fungus in old scab lesions (Whiteside, 1977), but was retired due to the development of resistant strains (Whiteside, 1980). Benomyl is still effective in many locations and is still recommended for scab control (Knapp 2000).

The systemic fungicides, IMZ and TBZ penetrate the rind of the fruit. Brown et al. (1983) conducted a comparative study of residues and efficacy of IMZ applied in either water or water-based resin solution wax using a non-recovery spray application to oranges revolving on horsehair brushes saturated with the treating solutions. Their results indicated that applications of IMZ in water apparently resulted in better penetration of the fungicide into the uninjured exocarp than when applied with water-based wax; additionally fruit treated with IMZ in water were less susceptible to infection through post-treatment injuries than fruit treated with IMZ in wax. Moreover, levels of IMZ were consistently higher from non-recovery water applications than from wax applications. Residues from water treatments were also enhanced by increased time on the brushes. Injured rind contained higher residues of IMZ than uninjured tissue and residues on fruit washed after IMZ treatment were reduced only slightly.

Cabras et al., (1999) studied the effect of concentration, temperature, and length of treatment with IMZ and TBZ applied to citrus fruit. Their results indicate that IMZ had a great persistence during storage when applied separately, and >83% of active ingredient was present after 9 weeks of storage. IMZ residues increased with dip length, doubling when dip time increased from 0.5 to 3 min. In contrast, TBZ residues did not change with the different dip times. Taking into account the facts that the residues of TBZ and IMZ in citrus fruit are retained mostly by the peel portion (Brown et al., 1983; Tadeo et al., 1988) and that 50% of IMZ and 10% TBZ are absorbed by the peel and not removed by home washing (Cabras et al., 1999).

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La fuente et al. (1987) evaluated the amount of TBZ residue in citrus fruit using two different methodologies. The amount of TBZ residues found in Washington navel oranges varied depending upon the part of the fruit. Higher concentration were found in the peel (2.8 mg/kg after two days of storage and 2.1 mg/kg after 30 days of storage); in the albedo and pulp the concentration varied from 0.1 mg/kg after two days of storage to 0.09 mg/kg after thirty days of storage at 5C. A similar response was obtained in Clementines.

For *Citrus* fruit, residue quantification has been performed predominantly on the rind (flavedo + albedo) and pulp expressing the active ingredient concentration on a whole-fruit basis. These studies evidenced that nearly all of the fungicide resided in the rind, either when applied as non-recovery spray (NRS) in water-based resin solution wax or by immersing fruit in heated mixtures (Schirra et al, 1997).

In the packinghouse, fruit must be treated with at least one of the treatments listed in 7 CFR 305.11. Routine packinghouse treatments such as washing, brushing, surface disinfesting with at least one of the treatments listed in 7 CFR 305.11, waxing, and fungicide treatment are used to reduce postharvest infection of several pathogens and saprophytes. These practices directly or indirectly will affect any SOS symptomatic fruit that could have escaped the culling process. The physical action of the brushes to remove any soil particle present in the surface of the fruit would have a deleterious effect on scab lesions, exposing the fungal tissue that will produce the spores. The use of chlorine or SOPP will further eliminate any spore that could have been present in the lesion. The use of fungicides (IMZ or TBZ) will prevent any surviving fungal structure from sporulating, and the addition of wax would further discourage the development of living structures on the surface of the fruit. It is therefore highly unlikely that SOS symptomatic fruit which has been treated with a surface disinfestant and then treated with a fungicide/wax mixture will be capable of producing spores capable of initiating infection.

A survey of packinghouse treatments used in the State of Texas found: Dump Sprayers & Washer Sprayers: Agcor 310 Chlorine(12.5% w/sodium hypochloride); Decco 103 Cleaner (Acidic Cleaner); high pressure washer system; Freshgard 71(12% W/Sodium Hypochloride) – Liquid Chlorine Bleach; Sulfuric acid 9%; or Peraclean Acid; Carnauba premoium Wax at ambient room temperature; IMZ or TBZ (Lone star packinhouse TX); Dump Sprayers Line: Chlorine 200 ppm at buffer pH 7.0; High pressure washer system with Chlorine 200 ppm at buffer pH 7.0; Pre-drier brushes; Peraclean 85 ppm; wax system (Pearl Luster or EU) at ambient temperature; IMZ 2000 ppm or TBZ 3500 (Rio Queen packinghouse, TX; and Edinburg Citrus Association, TX).

VIII. Summary

SOS is primarily a fruit problem and most important because scabby lesions make fruit less attractive. Growers will likely apply field treatments where scab (either SOS or Sour orange scab) is problematic enough to affect the quality of harvested fruit. This would be especially true for fruit destined to fresh markets. Field control may be reduced for growers supplying processing markets due to less concern about the appearance of fruit and therefore less justification for the added expense and effort unless fruit drop becomes a significant factor.

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Where effective disease management strategies are used in groves, experience shows that the incidence of symptomatic fruit is significantly reduced. Combining field controls with field selection to avoid highly infected fruit and culling in the packinghouse to remove the scabbiest fruit, ensures that normal industry practices that will limit the number of highly infected fruit in commercial shipments. Although some small proportion of symptomatic fruit may be expected to arrive in areas suitable for establishment, transmission from asymptomatic fruit would be impossible and the combination of specific conditions required for transmission from symptomatic fruit are so unlikely as to be negligible for small lots of fruit for consumption. In any case, if fruit is packinghouse treated with a surface disinfectant to destroy existing acervuli and spores, and then treated with a fungicide-wax to prevent further development or sporulation, there is little likelihood that symptomatic fruit is even able to produce viable conidia.

The following summarizes this analysis:

- Asymptomatic fruit is not a pathway for the introduction or spread of the disease.
- Symptomatic fruit that is commercially packed is not epidemiologically significant as a pathway if washed with a surface disinfectant and treated with fungicides during packing.
- Symptomatic fruit that is not washed and fungicide treated could be epidemiologically significant under unusual conditions.

As noted above, the movement of large quantities of severely diseased, untreated fruit into suitable areas near hosts with susceptible tissue growth should be avoided because of the potential for a high concentration of inocula to be generated and distributed in the event temperature, moisture, and wind conditions become suitable. Such a scenario may be unlikely but could be imagined for routine shipments of bulk fruit to processing facilities in citrus growing areas.

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