DISEASES OF FRUIT

J. O. WHITESIDE

INTRODUCTION

Some diseases are important only as a rind blemish and these need be of concern only to producers of fruit for the fresh market. Examples of such diseases are greasy spot rind blotch (Mycosphaerella citri) and melanose (Diaporthe citri). These diseases are particularly important on those cultivars which fetch a better price on the fresh market than for processing and for those cultivars that cannot be disposed of easily or profitably for processing.

Alternaria brown spot (Alternaria citri), which mostly affects Dancy tangerines and Minneola tangelos, is an example of a disease that can cause fruit drop and consequent loss of yield, as well as a rind blemish. Scab (Elsinoe fawcettii) is regarded mostly as a cosmetic problem, but on some cultivars the resulting fruit distortion is so severe that juice yields may be reduced. Therefore, scab still needs to be controlled on such highly susceptible cultivars as Temples and lemons, even if the crop is destined for processing.

There is also the type of disease that causes a rapid rotting of the fruit. Most of the disease in this category, including stem-end rots, sour rot, green mold, and center rot cause rotting of the fruit only after harvesting. They will not be discussed in this presentation. Brown rot caused by Phytophthora spp. is an example of a disease that can cause rotting of fruit on the tree, but, fortunately, this is only a very localized problem in Florida.

Fruit are susceptible to attack by most pathogenic fungi only during certain periods in their development. Only young fruit are susceptible to infection by the melanose and scab pathogens and the rind becomes immune to any further attack by these organisms at about 3 months after petal fall. It takes longer for the rind to become resistant to Alternaria brown spot but the risk of infection is generally over by the time the fruit are 4 months old. Brown rot is an example of a disease that attacks fruit only after they reach a certain stage of development, and usually not until 4-5 months prior to harvest. With greasy spot rind blotch, the time of infection is related mostly to the abundance of the inoculum supply and the required climatic conditions for infection, and this restricts infection to the summer.

This presentation describes the more serious fungal diseases of fruit that occur in Florida citrus groves. Information is given on each causal agent and its life cycle, the salient features of disease epidemiology, and the current preventative and control practices. A summary of the relative importance of fungal diseases on the fruit of different citrus cultivars is shown in Table 1.

Melanose

Symptoms. Visual symptoms develop about 1 week after infection. The flavedo tissue is killed for up to six cells deep and the dead cells become impregnated with reddish-brown gum. At first the affected areas shrink to form slightly sunken

Table 1. The occurrence and severity of fungal diseases on the fruit of different citrus cultivars.

Cultivar	Disease	Occurrence	Importance as a blemish	Probable or known effects on total yield
Orange	Melanose	Common	Moderate	None
	Scab	Rare	Moderate	None
	Greasy spot rind blotch	Common	Minor	None
Grapefruit	Melanose	Common	Major	None
	Scab	Localized	Moderate	None
	Greasy spot rind blotch	Common	Major	None
Temple	Melanose	Common	Moderate	None
	Scab	Common	Major	Minor
Murcott	Melanose	Common	Moderate	None
	Scab	Common	Moderate	None
Dancy tangerine	Melanose	Common	Minor	None
	Alternaria brown spot	Common	Major	Major
Minneola tangelo	Melanose	Common	Minor	None
	Alternaria brown spot	Common	Major	Major
Orlando tangelo	Melanose	Common	Minor	None
	Scab	Localized	Major	None
Lemon	Melanose	Common	Major	None
	Scab	Common	Major	Minor

lesions. Later, a periderm develops and this produces a corky tissue which ruptures the cuticle and lifts the dead cells above the fruit surface. The final diameter and height of lifting of a melanose pustule depends on the age of the fruit when infected. Infection shortly after petal fall results in relatively extensive and raised pustules whereas infection nearer to the time of rind immunity results in the formation of pustules that are only slightly raised and that detract little from fruit appearance unless they are very numerous. A tear-streaked melanose pattern is produced when spore-laden water flows over the rind in definite paths. When numerous and close enough, the pustules coalesce to form extensive reddish-brown areas which crack as the fruit enlarges to produce a symptom called mudcake melanose.

The only other blemish that might be confused with melanose is that caused by rust mites. The two blemishes can be similar in color but melanose produces a roughened condition whereas rust mite injury is generally smooth.

Causal Organism and Its Life Cycle. The spore-producing structures of Diaporthe citri, the pycnidia and perithecia, are formed on dead wood, mostly on twigs that have died within the previous year or two. No inoculum is ever produced on the melanose pustules themselves. Essentially, D. citri is a saprophytic fungus that completes its life cycle on dead wood. Infection of rind to create melanose can be regarded as a hypersensitive reaction in which the host rapidly seals off the invaded area by forming a barrier of cork tissue.

Asexual spores (conidia) are produced in greater abundance than sexual spores (ascospores). Conidia are dispersed by water, whereas ascospores are airborne, the later being responsible for long-distance dispersal. Generally, heavy infection is traceable to pycnidiospore-laden dead twigs located above the fruit. If, however, there is much dead wood remaining on or beneath the trees or there is citrus brushwood nearby, enough ascospores may be released to cause a problem.

Epidemiology. Disease severity is determined (1) by the amount of dead wood in the tree canopy, (2) by the amount of dead wood or brush left in a grove after hedging or topping operations, and (3) by the number of infection days that occur during the 3-month period of rind susceptibility.

A key feature in melanose epidemiology is the long period of continuous wetting needed for spore germination and host penetration. For example, at 15 C the rind surface would have to remain wet for 18-24 hr for infection to begin. Even at 25 C the minimum wetting period for infection is still 8-10 hr. Therefore there is little risk of infection in Florida citrus groves unless fruit stay wet for long periods and the temperature remains high at the same time. Infection is mostly confined to those times when rain falls in the afternoon and the fruit remain wet overnight.

Melanose becomes more prevalent as trees grow older because of a tendency for more twig dieback to occur on older trees. Melanose is troublesome on young trees only if they lack vigor or have been injured by a freeze.

Control. Mostly, copper fungicides are still used to control melanose. They are effective when applied postbloom but have little or no value if applied before petal fall, their action being essentially a protectant one on the fruit surface itself. If only a single treatment is to be applied, it is best delayed until late April or early May. This is because there is relatively little risk of infection before this time, April generally being a very dry month. If the single copper treatment is applied too soon after petal fall, its protectant value will be lost through fruit enlargement and erosion long before the fruit rind becomes resistant to attack. A better assurance of melanose control is provided by two copper treatments, one during the first 3 weeks after petal fall followed by another about 4 weeks later.

Another fungicide that is currently used to control melanose is captafol. Postbloom treatments of this material may cause rind injury, especially on grapefruit. Therefore, it is normally used only before petal fall and to supplement a postbloom copper treatment. Unlike copper compounds, captafol can reduce the inoculum supply. Furthermore, it can be redistributed from deposits on bark and leaves onto newly exposed fruit. A bloom treatment is therefore useful for preventing melanose on fruit if any infection days should occur before a postbloom copper treatment is applied.

Scab

Symptoms. A scab pustule is comprised of a thin layer of fungal material plus dead host cells, called a stroma, and an underlying pathogen-induced swollen living host tissue called a hyperplasia. The volume of hyperplasia, and hence the height of the scab pustule above the normal rind contour varies greatly according to the age of the fruit at time of infection and the cultivar. More hyperplasia is formed on younger fruit than on older fruit. Pustules are more raised on Temple and lemons than on grapfruit or Murcott fruit. They are especially large on Orlando tangelos. If numerous and concentrated enough, the pustules coalesce to form extensive scabby or scurfy areas which may crack into platelets as the fruit expands. Delayed coloring of rind in the vicinity of scab pustules is characteristic of the disease. When first formed, the scab stromata range from pink to light-brown. Later, they become yellowish- or grayish-brown or even black.

The blistered type of scab pustule is so distinctive it can hardly be confused with any other disease symptom. The flat, scurfy type of confluent scab could, however, be confused with windscar. Scurfy scab can be distinguished from windscar markings by the usual presence of at least some discrete, circular, typical scab pustules that have not coalesced, on the periphery of the blemished area. Any islands of windscar outside the main scurfy area will be elongated or running in lines. Frequently, windscar and scab appear on the same parts of a fruit. They both tend to occur where a leaf blade touched the surface of the fruit when it was still young. Movement of a leaf against the rind surface by wind results in windscar. Under calm conditions the same leaf would tend to hold water between itself and the rind surface, thus favoring more scab infection at this location.

Causal Organism and Its Life Cycle. The only known sources of inoculum are asexual spores produced on the scab pustules themselves. Elsinoe fawcettii produces two types of conidia; hyaline colorless conidia and spindle-shaped colored conidia. The latter are produced in fewer numbers than the hyaline conidia and they have no direct infecting ability; however, when they are wetted they bud off hyaline conidia which can cause infection. Colored conidia are of interest mainly because they can be liberated by wind as well as by water and can withstand limited desiccation, thus permitting more distant dissemination.

Epidemiology. Hyaline conidia are produced only when scab pustules are wetted by rain, dew, or overhead sprinkler irrigation for a minimum of 1-2 hr. They can survive in the pustule for only a day or two after they have been formed. They are dispersed by water and require only 2.5 hr continuous wetting to germinate and cause infection. Thus, even if there are no conidia present on the scab pustules to begin with, it is possible for a new crop of conidia to be produced, splash-dispersed, and cause infection all within 5 hr. In Florida, durations of rainfall are usually too short to promote the development of a new crop of conidia as well as to disperse them. Therefore, infection severity depends mostly on how many conidia are already present on the scab pustule when rainfall begins. This in turn depends on how many spores developed on the pustules during the previous 1-2 days with dew or previous rainfall. Overhead irrigation, when running for long periods, greatly increases the risk of scab attack, because it more certainly will promote production of conidia as well as their dispersal and germination.

Temperature does not have a major impact on the severity of scab. Infection occurs at about the same rate at all temperatures from 20 to 28 C and is only appreciably slower if the temperature drops below 15 C.

Control. The most effective fungicide currently available for scab control is captafol and one treatment with this material at bloom will usually suffice. However, if the disease has been previously severe it is advisable to apply an extra treatment before shoot emergence, to prevent infection of the spring growth flush and a possible buildup of inoculum which would increase disease pressure on the fruit. A postbloom copper fungicide treatment, as timed for melanose control, helps to protect the fruit from scab but it is generally insufficient on its own to provide acceptable scab control. Benomyl is not used much now for scab control because of fungal tolerance problems.

Overhead irrigation can greatly increase the potential for scab attacks. This risk is reduced if the sprinklers apply the necessary amount of water within a few hours and the fruit has a chance to dry off before sundown.

Greasy Spot Rind Blotch

Symptoms. Damage to rind by M. citri is restricted to stomata and nearby cells and has been observed only on sweet orange and grapefruit. On fruit rind, stomata occur only in those parts of the epidermis that are between the major oil glands. Thus, no greasy spot induced lesions appear over the oil glands themselves. On oranges the infection causes death of only the guard cells and a few substomatal cells. The black specks thus formed are therefore very small and they detract little from the appearance of the fruit. On grapefruit rind the injury is more noticeable; the lesions range in size from minute specks to larger areas of dead tissue which can be extensive enough to coalesce with lesions formed at neighboring stomata. The lesions are pink at first and later become brown or black. They are not noticeably sunken. Retention of green color in living cells adjacent to the necrotic spots is characteristic of the disease, particularly early in the season.

Greasy spot rind blotch can be readily distinguished from melanose because melanose pustules are raised and are located over the oil glands as well as between them. It is difficult, particularly on grapefruit, to distinguish greasy spot rind blotch from injury caused by rust mites. On grapefruit rind, rust mite injury is often concentrated in those areas of the epidermis between the oil glands. The general effect of such localized injury is macroscopically similar to that caused by greasy spot rind blotch. However, with a 10X hand lens, necrotic specks caused by M. citri are seen to have a distinct and continuous demarcation between them and surrounding healthy tissue whereas the areas damaged by rust mite have a diffuse margin.

Causal Organism and Its Life Cycle. Inoculum consists of ascospores released from fallen decomposing citrus leaves. Ascospores are carried by air currents to the rind surface. Germ tubes formed by the ascospore can invade stomata immediately or first produce a branching epiphytic growth on the rind surface. The chances for abundant stomatal penetration is greatly increased by the epiphytic mycelial growth because all hyphal tips on it have the potential for infection.

After entering the outer stomatal chamber, the hypae form a thick-walled, multicellular structure, an appressorium, which soon fills most of the chamber. Shortly after an appressorium forms, a hyphal thread grows from it through the stomatal pore into the substomatal cavity. From there it grows very slowly and for a limited distance intercellularly into the flavedo. After a delay of at least two months, cells in the vicinity of internal hyphae begin to die. After the necrotic specks first appear, they increase very little in area because lateral growth of M.

citri in the flavedo is very limited

Epidemiology. Ascospores are more abundant in June and July than at other times of the year. Minimum requirements for infection at temperatures above 20 C are 6 hr of near 100% relative humidity or wetness. It takes much longer for infection to occur at lower temperatures. Favorable conditions for infection seldom occur in the daytime. It is normally only at night and from June to September that long enough periods of high humidity or wetting occur to promote infection. Furthermore, little infection is likely to occur after August because the inoculum supply has usually diminished by this time.

There is a delay of several weeks after ascospores germinate on the rind surface and before the resulting epiphytic growth has grown sufficiently to substantially increase its infection potential. This is because only limited extension of hyphae occurs each night the temperature-humidity or wetness regimes are favorable for pathogen growth. This is an important consideration in the timing of a single spray treatment for the control of greasy spot rind blotch. It means that spraying is best delayed until July even though the ascospores start to reach the rind surface earlier than this. A treatment applied in July can eradicate the epiphytic growth before much stomatal penetration has occurred and provide protection against ascospores that have still to be released from the fallen leaves.

The time taken for greasy spot rind blotch symptoms to appear varies greatly. The symptoms can appear as early as September but are not generally noticeable this early unless the fruit has been degreened with ethylene. At other times, the symptoms may not appear until late fall or winter. The infection can even remain latent throughout the life of the fruit. Growth of M. citri into the flavedo does not inevitably cause death of the adjacent cells. The rind has to be predisposed to fungal infection in some unknown manner for injury to occur. Some grapefruit groves are afflicted by greasy spot rind blotch every year; others are seldom affected, even when greasy spot is severe on leaves.

Control. Greasy spot rind blotch is best controlled by a copper fungicide treatment applied in July. A postbloom copper treatment sometimes helps to control the disease but it never does this as well as a copper spray applied in the summer. Spray oil seldom provides acceptable control of greasy spot rind blotch, not even in those groves or years when it controls greasy spot well on leaves. Oil has even been known to increase greasy spot rind blotch, particularly if applied in August. It may do this by weakening the substomatal cells and rendering them more prone to injury by M. citri.

Alternaria Brown Spot

This disease first appeared in Florida in 1974. It soon spread to all citrus-growing areas, but has caused economic losses of fruit only on Dancy tangerines and Minneola tangelo trees. In some groves, the disease continues to be a problem, but in others it has been kept under reasonably good control by appropriate grove management and spraying procedures.

Symptoms. The symptoms appear first as small black sunken spots. Infected fruit may soon abscise, particularly if very young. If the fruit do survive long enough, a wound periderm forms beneath the lesion and this lifts the dead tissue above the rind surface in a manner similar to melanose. However, unlike melanose pustules, the pustule caused by Alternaria brown spot dislodge easily from the rind

leaving a pockmark. At most infection points, the self-healing process is complete and all that is left on the fruit at maturity is a superficial grade-reducing blemish. Sometimes, the pustules first appear inactive and later produce a slowly extending necrotic area around them, forming a so-called brown spot symptom. When this happens while the fruit is still green, there is much chlorosis around the lesion. If it happens closer to maturity, there is premature coloring of the fruit. Most fruit with reactivated lesions drop before normal picking time.

Causal Organism and Its Life Cycle. The name given to the causal fungus is Alternaria citri, which is the same name as that given to the two fungi that, respectively, cause Alternaria brown spot of rough lemon and center rot (black rot) of harvested fruits. Nevertheless, the fungus that causes Alternaria brown spot on Dancy tangerine and Minneola tangelo is pathogenically distinct from these other fungi.

The pathogen overwinters on lesions formed the previous growing season on leaves and stems and mostly on the latter because infected leaves usually abscise prematurely. Spores are dispersed by wind as well as by a water-splashing action.

Epidemiology. Infection seems to occur mostly when leaves or fruit are wetted by rain or overhead irrigation. Apparently, dew in itself is not very conducive to infection. In years when there is no rain during the emergence of the spring growth flush, this flush escapes attack unless the trees receive overhead irrigation. If infection does occur on the spring growth flush, this leads to greatly increased inoculum pressure on the fruit.

Overstimulation through heavy fertilization and frequent irrigation promotes lush shoot growth which is more predisposed to attack. Hedging and topping can also increase the risk of disease attack. Partly, this is due to the promotion of lush shoot growth and partly it is due to interference in the normal pattern of shoot emergence during the 4-month period of fruit susceptibility. Under normal conditions, there is a period of 2 months or more between the emergence of the spring growth flush and the emergence of the next growth flush. This reduces the possibility for pathogen buildup on foliage during much of the critical period of fruit susceptibility. After pruning, new shoots emerge more or less continuously, so that some susceptible shoot growth is present wherever an infection period occurs.

Control. Dancy tangerine fruit are inherently small and to increase fruit size growers frequently resort to such practices as heavy fertilization, frequent irrigation, and hedging. These practices promote a type of shoot growth, and perhaps fruit rind, that is more susceptible to Alternaria brown spot. Furthermore, if the irrigation is overhead, it greatly increases the chances of attack. Moderation is therefore needed in the use of those cultural practices that are likely to favor the disease.

The most essential part of the spray program consists of postbloom applications of copper fungicide. The first application is applied in late-April or early-May and a second is applied about 1 month later. In some years, two such sprays may suffice. In other years, an additional spray may be required in late June

Sometimes, Alternaria brown spot begins to attack the fruit before May, especially in groves receiving overhead irrigation. To guard against such early infection, a spray of captafol needs to be applied at bloom. This is particularly

desirable if the disease has appeared on the spring growth flush. Alternatively, or additionally, captafol can be applied routinely late dormant to prevent infection of the spring growth flush.

Brown Rot

In Florida, brown rot is a very localized disease, but in those groves where it does occur, it can recur year after year. Most of the affected groves are located in Hardee County and on the East Coast. The disease can be severe in years when there are unusually long durations of rainfall and wetting caused by slow-moving tropical depressions or hurricanes. Early maturing cultivars can be infected as early as August. Late maturing cultivars like Valencias seldom become infected because they are not susceptible until the winter and spring when the climate is prevailingly dry.

Symptoms. The rot first appears as a light brown discoloration of the rind and the fruit soon drop. The disease can be localized in a grove with the presence of brown rot being indicated by quantities of fallen fruit under some trees or parts of a tree. Brown rot produces a characteristic odor by which the disease can often be detected in an affected grove. If exposed to high humidity, the decaying portion of a fruit becomes covered with white growth of the causal fungus.

Causal Organism and Its Life Cycle. Phytophthora parasitica, which is ubiquitous in Florida citrus groves, can cause brown rot, but usually it infects only fruit that are close to or in contact with the soil. All serious outbreaks of brown rot in Florida citrus groves have been due to Phytophthora citrophthora. This species has a restricted occurrence in Florida, which is one of the reasons why brown rot epidemics are so uncommon.

Epidemiology. To initiate rind infection, zoospores have to be splashed from the soil onto low-hanging fruit. Further spread higher up into the canopy depends mostly on fruit to fruit spread. For this to occur, the sporangia have to develop on the infected fruit and then release zoospores which can be splashed higher up the canopy. A long period of continuous wetting of diseased fruit is required for sporangial development. A minimum of about 24 hr continuous wetting is needed for production of sporangia by P. parasitica but only 10 hr is needed for P. citrophthora.

Control. Where brown rot is a persistent problem, a precautionary copper fungicide treatment may have to be applied as early as August or September but only on early- or mid-season cultivars. A second treatment may be needed if the year is unusually wet.

Sooty Blotch and Fly Speck

The blemishes these diseases cause are due to the dark appearance of the causal fungi themselves, there being no destruction of the rind cells. Sooty blotch caused by Gloeodes pomigena consists of dark strands of fungal mycelium that are firmly attached to the rind cuticle. Fly speck, caused by Leptothyrium pomi consists of black pycnidia of this fungus. Neither sooty blotch nor fly speck can be effectively removed by washing the fruit in the packinghouse. These diseases become important only in years with exceptionally high summer rainfall. A single copper treatment applied in July usually provides adequate control.

REFERENCES

Knorr, L. C. 1973. Citrus diseases and disorders. Univ. Presses of Florida, Gainesville. 163 pp.

Whiteside, J. O. 1970. Factors contributing to the restricted occurrence of citrus brown rot in Florida. Plant Dis. Reptr. 54:608-612.

Whiteside, J. O. 1977. Behavior and control of greasy spot in Florida citrus groves. Proc. Intern. Soc. Citriculture 3:981-986.

Whiteside, J. O. 1978. Epidemiology and control of citrus scab in Florida. Proc. Intern. Soc. Citriculture 200-204.

Whiteside, J. O. 1979. Alternaria brown spot of Dancy tangerines and its control Proc. Fla. State Hort. Soc. 92:34-37.

Whiteside, J. O. 1980. Timing of fungicide spray treatments for citrus melanose control. Proc. Fla. State Hort. Soc. 93:21-24.

Whiteside, J. O. 1981. Evolution of current methods for citrus scab control. Proc. Fla. State Hort. Soc. 94:5-8.

Whiteside, J. O. 1983. Timing of spray treatments for citrus greasy spot control Proc. Fla. State Hort. Soc. 96:17-21.