The Role of *Colletotrichum* spp. in Postharvest Anthracnose of Citrus and Survival of *C. acutatum* on Fruit

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ABSTRACT

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Colletotrichum gloeosporioides causes postharvest anthracnose of citrus fruit and is a common saprobe in citrus groves, whereas *C. acutatum* infects flower petals and causes postbloom fruit drop (PFD). The purpose of this study was to determine whether *C. acutatum* caused anthracnose, survived as quiescent infections on fruit, and represented a threat for introduction of the PFD pathogen to disease-free areas. When Robinson tangerine fruit were artificially inoculated with both species and exposed to high levels of ethylene, only fruit inoculated with *C. gloeosporioides* developed postharvest anthracnose. However, *C. acutatum* was readily reisolated from inoculation sites on asymptomatic fruit. Navel oranges harvested from groves with severe PFD the previous spring developed low levels of anthracnose, but only *C. gloeosporioides* was isolated from lesions. Valencia orange fruit harvested at bloom during a PFD outbreak had high surface populations of *C. acutatum* which declined rapidly during 2 to 3 weeks of storage. Quiescent infections formed on artificially inoculated and naturally exposed fruit were reduced but not eliminated by washing, waxing, and fungicide treatment in the packinghouse. Since *C. acutatum* does not produce postharvest anthracnose, the risk of introduction of this pathogen to disease-free areas is minimal. However, if fruit were harvested during a PFD outbreak, *C. acutatum* might survive standard packinghouse treatment and shipment as quiescent infections.

Additional keyword: *Colletotrichum gloeosporioides*

Colletotrichum spp. cause anthracnose disease and postharvest decays on many tropical, subtropical, and temperate fruits (4,9,13). Many of these diseases are caused by *C. gloeosporioides* (13) but, in recent years, *C. acutatum* has been found to be an important pathogen of these fruits (4,8,9). This species has been reported on peaches, apples, and pecans (4) and on strawberries (8). On some hosts, more than one species cause disease. For example, *C. gloeosporioides*, *C. acutatum*, and *C. fragariae* all cause strawberry anthracnose (8).

On citrus, postharvest anthracnose of fruit is caused by *C. gloeosporioides* (Penz.) Penz. & Sacc. in Penz. (6,7). This species, which is a common saprobe in citrus groves, invades dead and senescent leaves, twigs, and fruit and produces acervuli with abundant conidia on dead tissues

of citrus. Conidia are splash-dispersed to living leaves, twigs, and fruit, where they germinate to produce appressoria and quiescent infections (6,14). Once the tissue dies, it is rapidly colonized and acervuli are formed, completing the life cycle.

When fruit with immature rinds with high numbers of appressoria of *C. gloeosporioides* are exposed to stress, the rind collapses and is colonized by the fungus, producing postharvest anthracnose (6,7). Early-season fruit is especially susceptible to anthracnose, and disease severity is greatly increased by exposure to high levels of ethylene for degreening. Washing of the fruit prior to degreening removes many appressoria and reduces disease incidence. Preharvest sprays of benomyl and postharvest fruit drenches with thiabendazole (TBZ) are effective in reducing the disease (6,10).

C. acutatum Simmonds causes postbloom fruit drop (PFD) of all citrus species as well as anthracnose of Key lime, on which it affects immature leaves, fruit, and flowers (3,5). In PFD, the fungus infects petals, producing lesions with acervuli and abundant conidia. Conidia are splashed from acervuli on petals to leaves, twigs, fruit, and calyces, where they germinate to form appressoria. Quiescent infections are formed on vegetative tissues in the grove

and the fungus is capable of surviving for months in that state (2,3,15). In contrast to *C. gloeosporioides*, *C. acutatum* does not colonize dead tissues of citrus in the field (3). Rather, in the presence of water, appressoria on the leaf surface splashed from flower petals germinate directly and produce a few conidia (2,12,15). These conidia are splash-dispersed to new flowers, which they infect, causing PFD.

PFD is known only in the Americas (11), but there is concern that the pathogen could be introduced into other citrus areas on fruit shipped to those regions. If *C. acutatum* were able to infect fruit and produce postharvest anthracnose and acervuli on the fruit surface, the risk of introducing the pathogen to new areas would be increased. The goal of this study was to determine the ability of *C. acutatum* to cause anthracnose and to survive on the surface of harvested fruit as quiescent infections.

MATERIALS AND METHODS

Artificially inoculated fruit. Fruit used in experiment 1 were from 7-year-old Robinson tangerine trees which had been sprayed with benomyl at 1.2 g a.i./liter, using about 5 liters/tree, on 2 July 1996 to suppress natural infection by *C. gloeosporioides*. Fruit used in experiment 2 were also Robinson tangerines, but had not received a preharvest benomyl spray. In experiment 3, the fruit used were from 7-year-old Robinson tangerines, navel oranges, and Ruby Red grapefruit trees. The Robinson tangerines and navel oranges in this experiment received preharvest benomyl sprays as described above, but the grapefruit received no preharvest benomyl applications.

Fruit for inoculation was harvested from the grove, washed thoroughly with soap and water, and scrubbed by hand with a brush to remove as many naturally formed appressoria as possible. Fruit was disinfested for 2 min in 10% household bleach (0.05% Na hypochlorite), then rinsed with sterile distilled water, and dried. For each treatment, about 15 to 20 fruit were placed in each of 3 plastic containers. Fruit was supported on a metal rack and the bottom of the container was filled to a 2-cm depth with water. An isolate of *C. gloeosporioides* from dead citrus leaves from Arcadia, Florida, and an isolate of *C. acutatum* from diseased flower petals from Vero Beach, Florida, were grown on potato-dextrose

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agar (PDA). Conidia were harvested from the plates using sterile distilled water, washed three times by centrifugation, and diluted to 10^5 /ml. Two separate drops of a conidial suspension were placed on marked areas on each fruit. Fruit in experiments 1 and 3 were inoculated without wounding. In experiment 2, half of the fruit inoculated with each species were wounded by puncturing the rind at the inoculation site with a needle to a depth of about 2 mm. The plastic containers were closed and incubated at 23 to 25°C for 48 h. After incubation, representative fruit were examined microscopically. In all cases, both species had formed numerous appressoria by that time. Fruit were inoculated during the time of year when they were most susceptible to anthracnose; those in experiment 1 on 11 September 1996, those in experiment 2 on 25 September 1996, and those in experiment 3 on 9 October 1996.

After inoculation, fruit were incubated for 72 h at 25°C at close to 100% relative humidity (RH) with 50 µl/liter ethylene. After removal from the ethylene chamber, fruit were incubated 2 more days at 25°C in experiment 1. In experiment 2, fruit were washed and waxed in a standard packing-house operation, then incubated for 2 more days at 25°C. In experiment 3, fruit were washed and waxed using 2,000 µg/ml TBZ in the wax in a standard packinghouse operation, then incubated 2 days at 25°C.

Inoculated sites on all fruit were examined for the presence of anthracnose le-

sions. Fruit were rated as positive for anthracnose when sunken, necrotic lesions about the size of water droplets were produced. Fruit rated as negative did not differ from the non-inoculated controls. Isolations were made from each inoculated site using the selective medium for *Colletotrichum* spp. developed by Agostini and Timmer (1), consisting of PDA plus copper hydroxide (42 mg Cu/liter) and streptomycin (300 mg/liter). In experiment 1, where fruit was not washed and waxed, rind pieces were surface-disinfested for 2 min in 10% household bleach (0.05% Na hypochlorite) and rinsed in sterile distilled water prior to plating. In experiments 2 and 3, rind pieces were plated directly without disinfestation.

Isolates recovered were tentatively identified to species by colony morphology and growth rate. Species identification was confirmed by transferring all cultures to PDA plates containing 10 µg/ml benomyl. *C. gloeosporioides* from citrus is highly sensitive to benomyl and does not grow at that concentration, whereas *C. acutatum* grows readily, albeit at a slower rate than on unamended media (11).

Natural infections from fall-harvested fruit. Fruit from navel orange groves heavily infested with PFD the previous spring were harvested in Arcadia on 17 September 1996 and in Lake Wales on 25 September 1996. All fruit were harvested from the vicinity of persistent calyces that characteristically remain on the tree as a

result of postbloom fruit drop. About 400 to 600 fruit were placed in ethylene chambers at close to 100% RH. Half of the fruit were exposed to 10 µl/liter and half to 50 µl/liter ethylene for 48 h. After removal from the ethylene chamber, fruit were washed and waxed in a standard packing-house operation. Waxed fruit were then incubated at 21°C for 2 to 3 weeks, examined periodically, and the number of fruit affected by postharvest decays recorded. Isolations were made on the selective medium for *Colletotrichum* spp. (2) from all fruit with anthracnose symptoms. Isolates were identified as previously described.

Natural infections in spring-harvested fruit. Valencia oranges were harvested on 31 March 1997 and again on 18 April 1997 from a grove severely affected by PFD near Fort Pierce, Florida. Mature fruit were harvested from portions of trees with currently affected flowers, or near clusters of persistent calyces indicating a recent attack by *C. acutatum*. Fruit from the first harvest were maintained in a screenhouse exposed to ambient conditions for the first 11 days, then transferred to a storage room at 21°C and close to 100% RH to prevent dehydration. Fruit from the first harvest were sampled 2, 11, and 19 days after collection for determination of surface propagule densities. Fruit from the second harvest were stored in the screenhouse under ambient conditions until sampled 5 days later to determine surface propagule densities.

For determination of surface propagule densities, on each sample date 40 fruit were scrubbed by hand using a brush with a small amount of water, and rinsed using 50 or 100 ml tap water per fruit. One-tenth ml aliquots of the rinse suspension was spread on each of 5 plates for each fruit. The medium used was that of Agostini and Timmer (2), to which benomyl had been added at 10 µg a.i./ml. Benomyl eliminates the growth of *C. gloeosporioides* except for occasional resistant isolates but allows growth of *C. acutatum* (11). In some cases, dodine (400 µg/ml) and tetracycline (100 µg/ml) were added to the above medium, which slowed the growth of *C. acutatum* but eliminated many fungal and bacterial

Table 1. Inoculation of Robinson tangerines with *Colletotrichum gloeosporioides* (C.g.) and *C. acutatum* (C.a.) to determine the ability of these two species to cause anthracnose and form quiescent infections on fruit (experiment 1)

Treatment ^b	Inoculation sites with lesions (%)	Reisolation (%) ^a			
		Lesions		No lesions	
		C. g.	C. a.	C. g.	C. a.
<i>C. gloeosporioides</i>	48.7 ± 4.1	97.2 ± 2.8	0.0	77.7 ± 9.3	0.0
<i>C. acutatum</i>	0.0	...	...	0.0	92.2 ± 1.1
Control	0.0	...	...	1.1 ± 1.1	0.0

^a Fruit not washed or waxed prior to reisolutions. Rind pieces disinfested with 10% household bleach (0.05% Na hypochlorite).

^b Three replicate humid chambers with 15 fruit each were used for each treatment with two inoculation sites per fruit; means ± standard error.

^c No lesions.

Table 2. Inoculation of Robinson tangerines with *Colletotrichum gloeosporioides* (C. g.) and *C. acutatum* (C. a.) to determine the ability of these two species to cause anthracnose and form quiescent infections on fruit (experiment 2)

Species	Fruit treatment ^b	Inoculation sites with lesions (%)	Reisolation (%) ^a			
			Lesions		No lesions	
			C. g.	C. a.	C. g.	C. a.
<i>C. gloeosporioides</i>	Non-wounded	52.2 ± 6.8	97.0 ± 3.0	0.0	81.3 ± 9.2	0.0
	Wounded	62.6 ± 3.2	98.2 ± 1.8	0.0	97.9 ± 2.1	0.0
<i>C. acutatum</i>	Non-wounded	3.3 ± 3.3	100.0	0.0	0.0	66.3 ± 11.7
	Wounded	5.7 ± 2.2	100.0	0.0	0.0	90.0 ± 3.6
Control	Non-wounded	0.0	...	...	14.2 ± 3.8	0.0
	Wounded	0.0	...	...	14.7 ± 6.7	0.0

^a Fruit was washed and waxed in a standard packinghouse operation prior to reisolutions, but received no fungicide treatment.

^b Three replicate humid chambers with 15 fruit each were used for each species and for each fruit treatment with two inoculation sites per fruit; means ± standard error; fruit was wounded by puncturing with a needle to a depth of 2 mm.

^c No lesions.

contaminants, thus facilitating the reading of the plates. Plates were incubated at 27°C for 7 to 10 days.

The fruit not used for surface propagule density determinations were washed and waxed in a standard packinghouse operation using 2% sodium orthophenylphenate (SOPP) in the wash water and 2,000 µg/ml each TBZ and imazalil in the wax 11 days after picking in the first harvest and 5 days after picking in the second harvest. Fruit from the second harvest were then stored for 5 weeks at 10°C and an additional 2 weeks at 20°C. After storage, fruit were examined for postharvest decays and isolations were made from any fruit on which *Colletotrichum* spp. had developed.

The 40-fruit sample from the second sampling date of the first harvest (11 days), as well as the 40-fruit sample from the second harvest (5 days), which had been washed and waxed in a standard packinghouse operation with wash water containing 2% SOPP and wax containing 2,000 µg/ml each of TBZ and imazalil, were sampled for latent infections. Forty 3-mm-diameter rind plugs were cut from each of the fruit. Rind plugs were placed flavedo-side down, 5 plugs/plate, on the selective media described above without surface sterilization. Plates were incubated for 7 to 10 days at 27°C. *C. acutatum* was usually distinguishable from *C. gloeosporioides* directly on the isolation plates by colony size and morphology. Where doubt existed, the growth rate on PDA and the conidial morphology were examined in comparison with known isolates for final identification.

RESULTS

Artificially inoculated fruit. In experiment 1, *C. gloeosporioides* caused anthracnose lesions on about half of the inoculated sites after 7 days, but *C. acutatum* produced none (Table 1). *C. gloeosporioides* was reisolated from virtually all of the fruit with anthracnose and from many of the inoculated fruit which formed no lesions. Despite the fact that *C. acutatum* produced no anthracnose lesions, it

was reisolated from most of the inoculation sites. *C. gloeosporioides* was isolated from a low percentage of control fruit.

In experiment 2, *C. gloeosporioides* again caused anthracnose lesions on over half of the fruit and was usually reisolated from most inoculated fruit whether or not lesions were formed (Table 2). A low percentage of fruit inoculated with *C. acutatum* developed anthracnose lesions. However, all of the isolates from these lesions were *C. gloeosporioides*. *C. acutatum* was again reisolated from a high percentage of fruit without lesions inoculated with that species. Although fruit in this experiment were washed and waxed and fruit in experiment 1 were not, reisolation of *C. acutatum* was generally lower in experiment 2 than in experiment 1. Fruit inoculated with *C. gloeosporioides* had slightly higher levels of anthracnose on wounded than on non-wounded fruit. Recovery of both species on asymptomatic fruit was also slightly higher on wounded than on non-wounded fruit. No anthracnose lesions developed on control fruit, but *C. gloeosporioides* was isolated from a low percentage of the fruit.

In experiment 3, anthracnose lesions developed on Robinson tangerines inoculated with *C. gloeosporioides*, but not on navel

oranges or grapefruit (Table 3). None of the citrus species inoculated with *C. acutatum* developed anthracnose lesions. *C. gloeosporioides* was isolated from all fruit with anthracnose lesions, from 53 to 78% of sites inoculated with this species, and from 4 to 38% of the control fruit. *C. acutatum* was isolated from 51 to 78% of the inoculated sites. *C. gloeosporioides* was not recovered from any of the fruit inoculated with *C. acutatum* even though it was recovered from the controls, especially on Robinson tangerines and grapefruit. Recovery was somewhat lower in this experiment, where fruit was washed, waxed, and treated with TBZ, than in experiment 2, where fruit was only washed and waxed.

Natural infections from fall-harvested fruit. In fruit collected from two navel orange groves infested with PFD, relatively little anthracnose developed and no differences were observed between fruit degreened at 10 or 50 ppm ethylene. In navel oranges harvested from the grove near Arcadia, there were large losses from stem-end rot and oleocellosis. Only 35 of 564 fruit developed anthracnose-like symptoms, and *Colletotrichum* spp. were recovered from only 16 of the 35. Of 466 fruit harvested from the grove in Lake Wales, only 9 developed anthracnose;

Table 4. Recovery of *Colletotrichum acutatum* from naturally infested Valencia oranges harvested during an outbreak of postbloom fruit drop

Sample time (days postharvest)	Propagules/fruit in wash ± SE ^a	Fruit isolations (% positive)	
		Fruit ^b	Rind pieces ^c
First harvest 31 Mar 97			
2	7513 ± 2103	NT ^d	NT
11	580 ± 153	10	0.31
19	40 ± 21	NT	NT
Second harvest 18 Apr 97			
5	23 ± 7	15	0.44

^a Average number of propagules of *C. acutatum* recovered per fruit in a surface wash ± the standard error of the mean, prior to washing, waxing, and fungicide treatment.

^b Percentages of fruit with at least one isolation from the 40 3-mm diameter rind plugs plated per fruit; fruit washed and waxed with 2,000 µg/ml each of thiabendazole and imazalil in the wax before isolations.

^c Percentage of isolation of the total number of rind plugs.

^d NT = Not tested.

Table 3. Inoculation of Robinson tangerines, navel oranges, and Ruby Red grapefruit with *Colletotrichum gloeosporioides* (C. g.) and *C. acutatum* (C. a.) to determine the ability of these two species to cause anthracnose and form quiescent infections on fruit (experiment 3)

Citrus species ^b	Fungal species ^b	Inoculation sites with lesions (%)	Reisolation (%) ^a			
			Lesions		No lesions	
			C. g.	C. a.	C. g.	C. a.
Robinson tangerines	<i>C. gloeosporioides</i>	55.0 ± 10.4	100.0	0.0	74.4 ± 17.9	0.0
	<i>C. acutatum</i>	0.0	...	...	0.0	63.3 ± 6.0
	Control	3.3 ± 3.3	100.0	0.0	22.6 ± 2.6	0.0
Navel oranges	<i>C. gloeosporioides</i>	0.0	...	...	53.8 ± 13.8	0.0
	<i>C. acutatum</i>	0.0	...	...	0.0	51.3 ± 4.3
	Control	6.7 ± 3.5	100.0	0.0	3.2 ± 2.9	0.0
Red grapefruit	<i>C. gloeosporioides</i>	0.0	...	...	78.1 ± 10.4	0.0
	<i>C. acutatum</i>	0.0	...	...	0.0	78.1 ± 9.4
	Control	2.1 ± 2.1	0.0	0.0	38.0 ± 9.1	0.0

^a Fruit was washed and waxed in a standard packinghouse operation prior to isolations; wax contained thiabendazole at 2,000 µg/ml.

^b Three replicate humid chambers with 20 fruit each were used for Robinson tangerines; four replicates of 20 fruit each were used for navel oranges; and four replicates of 16 fruit each were used for Ruby Red grapefruit; two inoculation sites per fruit were used in all cases; means ± standard error.

^c No lesions.

Colletotrichum spp. were recovered from all of those fruit. All 25 isolates recovered from fruit from both groves were identified as *C. gloeosporioides*.

Natural infections in spring-harvested fruit. High populations of *C. acutatum* were recovered from washings of mature Valencia oranges harvested during active infections on the bloom (Table 4). However, those populations on untreated fruit held after harvest decreased about 10-fold for each 8- to 9-day period on fruit of storage. The number of propagules in washings were of the same order of magnitude at the third sample time of the first harvest as on those of fruit from the second harvest. Thus, populations on the fruit in the grove appeared to decline at the same rate as those on stored fruit. No anthracnose developed on any of these fruit.

C. acutatum was isolated from rind plugs from fruit that had been washed with SOPP and treated with imazalil and TBZ. This species was isolated from only about 10 to 15% of the fruit, then only from 1 to 2 plugs per fruit with less than 1% of the plugs yielding *C. acutatum* (Table 4).

Fruit from the second harvest which had been stored for 7 weeks showed some stem-end rind breakdown due to desiccation. *Colletotrichum* spp. colonizing affected areas of 21 of these fruit were isolated. All of the isolates recovered were identified as *C. gloeosporioides*.

DISCUSSION

In contrast to *C. gloeosporioides*, *C. acutatum* does not cause postharvest anthracnose on citrus. Even when high concentrations of conidia of *C. acutatum* were placed on localized wounded areas of the rind of Robinson tangerine, the most susceptible variety (6,7), and the fruit treated with high concentrations of ethylene, little anthracnose developed. *C. gloeosporioides* was isolated from those few lesions formed, probably as the result of natural infections. There were low levels of natural infections of *C. gloeosporioides* in all three experiments. The application of benomyl to some of the trees the previous summer probably reduced the number of quiescent infections. Thorough washing of the fruit prior to inoculation probably removed many appressoria from the surface, thus further reducing natural infection (6,7).

More than half of the Robinson tangerines inoculated with *C. gloeosporioides* developed anthracnose. No anthracnose

developed on navel oranges or grapefruit as a result of inoculation. These varieties are less susceptible to anthracnose than Robinson tangerines and their rinds were probably sufficiently mature by the time the fruit was harvested to be resistant to disease development (6,7). Sometimes *C. gloeosporioides* was recovered from a low to moderate percentage of the control fruit, but was not detected in fruit inoculated with *C. acutatum*. Recovery of *C. gloeosporioides* was probably prevented because the large amount of inoculum of *C. acutatum* suppressed the natural development of *C. gloeosporioides*.

When the navel oranges were harvested in the fall from groves which had severe PFD the previous spring, little anthracnose developed, even under favorable conditions. The failure to isolate *C. acutatum* from those anthracnose lesions provides further evidence that this species does not cause postharvest anthracnose.

High numbers of propagules of *C. acutatum* were recovered from the surface of Valencia orange fruit harvested during an active PFD outbreak. The number of propagules declined rapidly without any treatment, suggesting that many of those propagules were conidia rather than quiescent infections formed from appressoria. However, quiescent infections were formed in high numbers on inoculated fruit, and some apparently also were formed on naturally exposed fruit. These infections appeared to be reduced but not eliminated by washing, waxing, and fungicide treatment. However, after long-term storage, only *C. gloeosporioides* was isolated from damaged fruit.

The possibility of transport of *C. acutatum* to areas free of PFD on commercial citrus fruit appears to be slight. Only in the case of Valencia oranges or grapefruit which might be harvested during bloom would there be any chance of contaminating the fruit. Many propagules would be removed or killed by normal washing, waxing, and fungicide treatment in the packinghouse. Quiescent infections would probably decline with time on fruit as they do on leaves in the grove (2), further reducing risk. Since appressoria of *C. acutatum* germinate directly without invasion of tissue to produce a few conidia (3,12,15), rather than forming acervuli with abundant conidia, there would be little risk of spread even if fruit with quiescent infections were introduced to disease-free areas.

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