Sour Rot of Peaches Caused by Monilia implicata and Geotrichum candidum

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ABSTRACT

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Losses due to sour rot caused by *Monilia implicata* and *Geotrichum candidum* were consistently greater on peaches experimentally waxed with 2,6-dichloro-4-nitroaniline (DCNA) and benomyl after hydrocooling in nonchlorinated water than on unwaxed peaches similarly hydrocooled. On peaches from a commercial packing shed with poor sanitation and grading practices, sour rot developed in three out of seven tests and affected 3.8 to 48.7% of the fruit. On peaches from a shed with good sanitation practices, sour rot developed in two of seven tests and affected less than 1% of the fruit. Frequency of isolation of organisms from infected fruit was 54.8% for *M. implicata* and 13.6% for *G. candidum*. On inoculated peaches, growth (lesion diameter) of *M. implicata* at 21 C was nearly three times as great as growth of *G. candidum*, but on artificial media it was significantly less than that of *G. candidum*. Concentrations of 1 or 10 μg/ml DCNA or benomyl (residues normally found on waxed peaches) did not affect, or inhibited only slightly, the growth of both organisms on artificial medium.

Additional key words: postharvest diseases, postharvest pathology, *Prunus* sp.

Sour rot, a postharvest disease of peaches [*Prunus persica* (L.) Batsch] caused by *Geotrichum candidum* Lk. ex Pers., was first reported in Chicago wholesale and retail markets by Burton and Wright (1). Bruised and injured peaches were particularly susceptible to sour rot which affected an estimated 3% of the peaches inspected. Infected peaches initially developed sunken, brown lesions, primarily around injured areas. The lesions then became covered with the characteristic white mycelium and arthospores of the pathogen and the fruit developed a yeastlike sour odor. *Geotrichum candidum* is a yeastlike fungus that causes watery soft rot of a variety of commodities such as melons (2), carrots (10), and tomatoes (6).

Sour rot has been considered a minor postharvest disorder of peaches but, as described in this report, can cause considerable losses in individual shipments. However, the incidence of sour rot in the field and market, the relationship of orchard and packing shed practices to incidence, and the complete identity of the pathogens is generally unknown. This report describes some factors that are related to the etiology of this disease in Georgia, and presents evidence that two organisms can incite sour rot.

MATERIALS AND METHODS

Incidence of sour rot was evaluated on peaches obtained from commercial packing sheds in Peach and Houston counties, Georgia, during 1974. Peaches were selected for uniformity of size and color and for freedom from bruises or blemishes. Postharvest hydrocooling and waxing treatments were applied with experimental equipment under simulated commercial conditions (8), or with commercial equipment in local packing sheds. Experimental fruits were hydrocooled for 20 min in nonchlorinated water at 1 C, and waxed against rotating brushes with an oil, petrolatum, and paraffin-base concentrate that contained 2,6-dichloro-4-nitroaniline (DCNA) at 5,000 μg/ml and benomyl [methyl 1-(butyl-2-carbamoyl) benzimidazolecarbamate] at 2,500 μg/ml. About 200 to 400 fruit constituted a treatment lot, and the treatments were replicated 10 times on different dates with different cultivars.

Samples of commercially-treated fruit were obtained from two local sheds. Fruits were sampled in each shed from different points along the packing line; namely, field bins, dump tank, hydrocooler, and waxer. In one shed, the fruits from the orchard were of good overall quality, were generally free of decay, and were hydrocooled in chlorinated water before introduction into the packing line. In the other shed, bins contained a considerable proportion of overripe fruit with split pits and decay, and fruits were dumped directly into nonchlorinated water that contained a noticeable amount of dirt and organic debris. About 8-9 kg of fruit (0.5 bushel) constituted a treatment lot, and samples were taken on seven separate occasions during the 1974 season.

Treated fruit were packed in lidded containers, and stored 5-14 days at 1 C and 75-80% relative humidity, then for 2-4 days at room temperature. Fruit were considered infected if typical sunken lesions covered with white,

floccose mycelium and arthospores (1) were present at any stage of development. Fruits also were rated for brown rot [caused by *Monilinia fructicola* (Wint.) Honey and *M. laxa* (Aderh. & Ruhl.) Honey], Rhizopus rot [caused by *Rhizopus stolonifer* (Ehr. ex Fr.) Vuill.], and miscellaneous rots [caused by *Botrytis cinerea* Pers. ex Fr., *Aspergillus niger* v. Tiegh, *Alternaria* sp., and *Penicillium* spp.] (5).

The pathogens were isolated from sour rot lesions by transfer of infected tissues from lesions to plates that contained peach-dextrose agar. Medium was prepared from 200 g ripened peach flesh, 20 g dextrose, and 15 g agar per liter. Plates with infected tissue were incubated at 27 C for 3 days. Fungi that grew out of tissues were classified according to genus. Pure cultures of representative fungal isolates were tested for pathogenicity to peaches by wound-inoculations on freshly-harvested fruit which then were held at room temperature and subsequently observed for lesion development. Only those cultures that produced typical sour-rot lesions were considered to be sour-rot pathogens. Inoculants were reisolated from inoculated tissues in fulfillment of Koch's postulate for proof of pathogenicity. Pathogenic cultures were maintained on potato-dextrose agar slants at 0 C.

Sour-rot pathogens were identified to genus and species with the aid of taxonomic descriptions (3, 4). Identities were considered definitive if all major

TABLE 1. Incidence of sour rot *(Monilia implicata, Geotrichum candidum),* brown rot *(Monilinia fructicola),* Rhizopus rot *(R. stolonifer),* and miscellaneous other postharvest decays on selected lots of peaches experimentally hydrocooled and waxed under simulated commercial conditions and held 7 days at 1 C, and then 2-4 days at 21 C[a]

Cultivar	Treatment date (1974)	Treatment[b]	Incidence of Brown rot (%)	Rhizopus rot (%)	Sour rot (%)	Misc.[c] rots (%)	Total decay (%)
Loring	9 Jul	nonwaxed	53.3	1.3	4.7	0	59.3
		waxed	2.6	0.5	8.3	0	11.4
Redglobe	10 Jul	nonwaxed	6.3	0.8	7.0	0	14.1
		waxed	1.8	0.6	13.1	1.5	17.0
Dixiland	15 Jul	nonwaxed	2.1	0.3	1.2	0.6	4.2
		waxed	0	0.3	12.0	0	12.3
Dixiland	16 Jul	nonwaxed	2.4	9.1	7.0	0	18.5
		waxed	0	0.5	16.7	0	17.2
Elberta	16 Jul	nonwaxed	0.5	19.2	14.0	0.3	34.0
		waxed	0	0.5	25.8	0	26.3
Redskin	2 Aug	nonwaxed	7.1	0.3	11.4	0	18.8
		waxed	0	0	3.3	0	3.3

[a]Data shown for the six of 10 experimental lots of fruit in which sour-rot incidence was over 1%.
[b]Peaches initially hydrocooled for 20 min in nonchlorinated water at 1 C, then nonwaxed or waxed with an oil, petrolatum and paraffin-base concentrate containing DCNA at 5,000 μg/ml and benomyl at 2,500 μg/ml.
[c]Miscellaneous rots caused by one or more species of *Botrytis, Aspergillus, Alternaria,* and *Penicillium.*

TABLE 2. Incidence of sour rot caused by *Monilia implicata* and *Geotrichum candidum* on peaches sampled at different points along the packing line in a commercial packing shed with poor sanitation and grading practices and held 7-14 days at 1 C, and then 2-4 days at 21 C

Cultivar	Sampling date (1974)	Sour rot on peaches per point along a packing line (%) Bin (check)	Dump tank[a]	Hydro-cooler[b]	Waxer[c]
Springold	3 Jun	0	18.5	6.2	9.8
Springold	4 Jun	0	0	0	0
Redcap	6 Jun	5.8	29.4	16.8	48.7
Coronet	14 Jun	0	0	0	0
Coronet	21 Jun	0	0	0	0
Coronet	26 Jun	0	0	0	0
Coronet	9 Jul	0	3.9	3.0	3.8

[a]Peaches dumped into and washed with nonchlorinated water.
[b]Hydrocooled for 20 min in water containing chlorine at 10-45 μg/ml (added as sodium hypochlorite) at 1 C.
[c]Waxed with an oil, petrolatum, and paraffin-based concentrate containing DCNA at 5,000 μg/ml, and benomyl at 2,500 μg/ml.

morphological characteristics of representative isolates corresponded to type descriptions.

Growth of fungi was determined by daily measurements of colony diameters on potato-dextrose and peach-dextrose agar plates and by measurement of lesion development on wound-inoculated peaches held under polyethylene liners to maintain high humidity. Growth studies were conducted at 21, 12.7, and 4.5 C. Plates were spot-inoculated by mass transfer of the organism from stock cultures. Peaches were inoculated by breaking the surface of the skin with the tip of a transfer needle previously smeared in a stock culture of the pathogen. Growth curves were plotted and regression coefficients calculated using data averaged from three replicated tests with four isolates of each organism inoculated on agar plates or on 10 individual fruit. In addition, growth was similarly determined for mixed cultures and infections by superimposing inoculations with different organisms. Colony growth also was determined at 21 C on peach-dextrose agar amended with 0, 1, 10, 100, and 1,000 μg DCNA or benomyl/ml. Serial logarithmic dilutions of the fungicides were prepared in acetone and diluted into warm, sterilized agar at the rate of 1 ml acetone solution per 100 ml of medium (to yield desired final concentrations). Percentage inhibition of growth was obtained from colony diameters measured 3-8 days after plates were inoculated. Inhibition of growth by the fungicides was calculated for four isolates each of *M. fructicola* and *R. stolonifer*.

RESULTS

Incidence on experimentally treated fruit.—The incidence of sour rot on experimentally treated fruit was over 1% in six of 10 tests. In these tests, sour rot accounted for from 10-50% of the total decay of hydrocooled, but unwaxed peaches, and for over 75% of the total decay of waxed peaches (Table 1). The wax treatments effectively controlled brown rot and Rhizopus rot, but were ineffective against sour rot. In most tests, waxing did not markedly reduce the net losses due to decay because of the high levels of sour rot that developed on waxed fruit.

Incidence on commercial fruit.—Fruit obtained from the commercial packing shed with good sanitation and grading practices generally was free of sour rot. Sour rot developed on fruit in only two of seven tests and then incidence was less than 1% (data not shown). In the packing shed with poor sanitation practices, however, sour rot was a major cause of losses on three of the seven sampling dates (Table 2). On 6 June, for example, fruit sampled from the field bins developed 5.8% sour rot. Incidence was generally severe (over 16%) on samples from the dump tank, hydrocooler, and waxer. As with the experimentally treated fruit, the wax-fungicide treatment controlled decay due to brown rot and Rhizopus rot (9), but did not control sour rot.

Isolation of pathogens.—Of the organisms isolated from 186 samples of sour-rot lesions, 54.8% were *M. implicata* Gilman and Abbott and 13.6% were *G. candidum* (Table 3). Miscellaneous organisms such as *Monilinia, Fusarium, Alternaria,* and unidentified species of fungi constituted the remainder. In two of the five sampling occasions, *G. candidum* could not be isolated from sour-rotted tissues.

Symptoms.—Isolates of *M. implicata* and *G. candidum* tested on inoculated peaches caused softened, brown lesions that eventually developed a vinegarlike odor. Infections by *M. implicata* developed rapidly and produced a creamy exudate with mycelia and spores. *G. candidum* infections developed slowly and were covered by white arthospores of the fungus. Mixed infections developed rapidly and produced a creamy white exudate.

Lesion development on inoculated fruit.—Lesion diameters on peaches inoculated with isolates of *M. implicata, G. candidum,* or both, increased linearly with time when held at 21, 12.7, and 4.5 C (Fig. 1). Growth of *M. implicata* was greater than that of *G. candidum* at all temperatures. Sample regression coefficients (b) of *M. implicata* which developed at 21 and 12.7 C were 8.4900 and 1.3678, respectively; these were more than double those of *G. candidum* at the corresponding temperatures (b = 3.0100 and 0.5705, respectively). At 4.5 C, b for *M. implicata* was 0.2675, six times that of *G. candidum* (0.0440).

Lesion development of combined infections by *M. implicata* and *G. candidum* at 21 and 4.5 C were not significantly different from those caused by *M. implicata* alone. At 12.7 C, however, lesion development for the combined infection (b = 1.4625) was significantly ($P \leqslant 0.05$) greater than that for *Monilia* (b = 1.3678).

Colony diameter on artificial media.—On peach- and potato-dextrose agars, the rates of growth of *G. candidum,* as measured by colony diameters at 21 C, were significantly greater than those of *M. implicata.* Regression coefficients of colony diameters on days were

TABLE 3. Incidence of *Monilia implicata* and *Geotrichum candidum* isolated on potato-dextrose agar from sour-rot lesions on peaches

Cultivar	Lesions sampled (no.)	Organisms isolated (no.)	Percentage of isolates per pathogen		
			M. implicata (%)	*G. candidum* (%)	Misc.[a] (%)
Coronet	36	39	48.7	7.7	43.7
Junegold	24	34	41.2	14.7	44.1
Junegold	36	38	84.2	0	15.8
Loring	54	68	26.5	45.6	27.9
Dixiland	36	41	73.2	0	26.8
Average			54.8	13.6	31.6

[a]Miscellaneous isolates were species of *Monilinia, Fusarium, Alternaria, Rhizopus,* unidentified fungi, and bacterial contaminants.

10.4503 for *G. candidum* and 1.2000 for *M. implicata* on peach-dextrose agar (Fig. 2). Growth curves on potato-dextrose agar were sigmoidal for *G. candidum,* and rates were maximum between days 2 and 4. Curves on peach-dextrose agar were linear throughout the 8-day test.

Effect of fungicides on growth.—*Monilia implicata* and *G. candidum* grew on peach-dextrose agar amended with DCNA or benomyl at ratios as high as 1,000 μg/ml (Table 4). Conventional wax-fungicide treatments leave a residue of both fungicides of 1-10 μg/g on peaches (9). At concentrations of 1-10 μg/ml, of either fungicide, growth was unaffected or only slightly inhibited compared to the checks. At the range of 100 to 1,000 μg/ml, DCNA was more toxic to *G. candidum* than to *M. implicata,* and benomyl was more toxic to *M. implicata* than to *G. candidum.*

The brown rot fungus, *M. fructicola,* was 100% inhibited by DCNA at 1 μg/ml or by benomyl at 10 μg/ml. *Rhizopus* isolates were unaffected by benomyl at any concentration tested, as has been previously reported (7), but were 100% inhibited by DCNA at 1 μg/ml.

DISCUSSION

Sour rot is a postharvest disorder of peaches that has caused significant losses in individual shipments. The incidence is associated with poor sanitation in the packing shed. In some cases, fruit waxed with the commonly-used

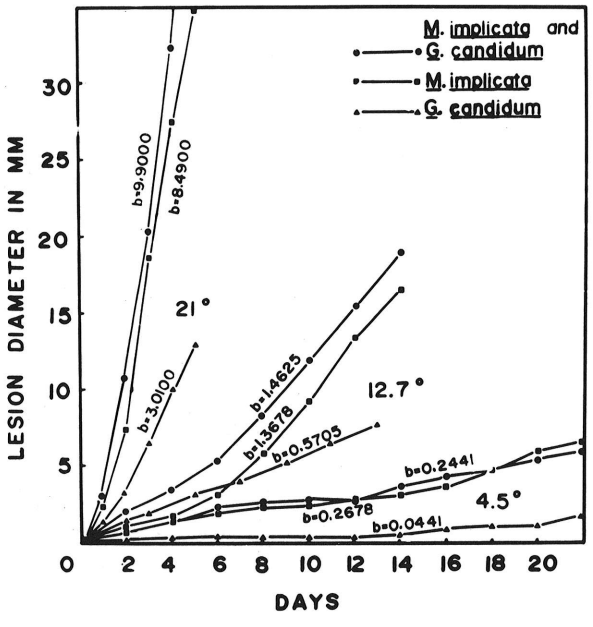

Fig. 1. Lesion development at 21, 12.7, and 4.5 C on peaches inoculated with *Monilia implicata, Geotrichum candidum,* or with both organisms. Each point represents average lesion diameters of 10 inoculated peaches per test, replicated three times. Regression coefficients (*b*), derived from data of each curve represented, are shown.

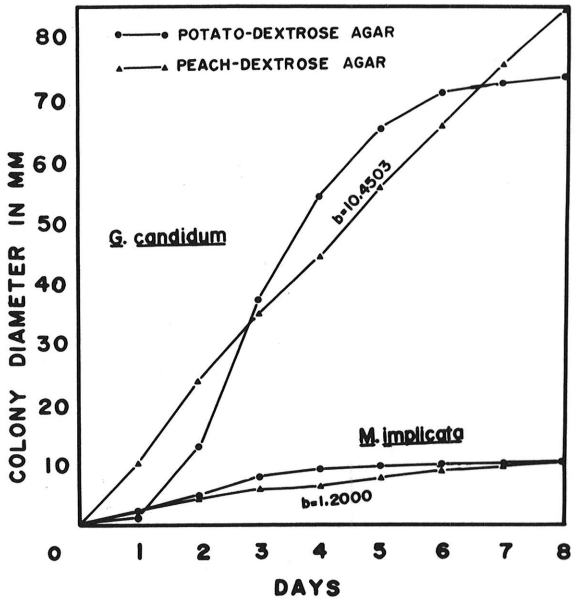

Fig. 2. Growth of *Monilia implicata* and *Geotrichum candidum* on peach-dextrose agar and on potato-dextrose agar at 21 C. Each point represents average values for three replicated tests with four strains of each organism.

TABLE 4. Inhibition of colony diameter at 21 C of four decay-causing fungi on peach-dextrose agar amended with logarithmic dilutions of benomyl or DCNA

Amendment		Inhibition per pathogen			
Fungicide	Concentration (μg/ml)	*Monilia implicata* (%)	*Geotrichum candidum* (%)	*Monilinia fructicola* (%)	*Rhizopus stolonifer* (%)
DCNA	1	0[a]	5.3	19.9	100
	10	5.9	13.7	100	100
	100	8.6	16.7	100	100
	1,000	29.6	41.5	100	100
Benomyl	1	0	1.3	100	0
	10	0	4.7	100	0
	100	49.4	13.1	100	0
	1,000	64.2	28.6	100	0

[a]Percentages based on average inhibition of growth of four strains or isolates of each organism during days 3-8 of growth.

organic fungicides, DCNA and benomyl, had more sour rot than unwaxed fruit. This was due to the selective action of the fungicides against competing decay-causing organisms such as *R. stolonifer* and *M. fructicola*. Residues of DCNA and benomyl, at the concentration range present on commercially-treated fruit (1-10 μg/ml) did not significantly affect the growth of the organisms. Those fungicides applied via hydrocoolers and waxers, however, can adequately control the major postharvest rots, such as brown rot and Rhizopus rot (7). Chlorination of dumping tanks and of hydrocoolers (if DCNA and benomyl are used in a wax applicator) appears to be important in the reduction of sour rot (J. M. Wells, *unpublished*) since chlorine, a broad-spectrum fungicide, is effective against all decay-causing organisms.

Geotrichum candidum has been reported previously as an incitant of sour rot on peaches (1). In the present investigation, *M. implicata* generally was isolated with *G. candidum* from infected tissues. Inoculation studies further showed that *M. implicata* was the more aggressive pathogen of the two, even though its growth was restricted on artificial media. Undoubtedly, sour-rot infections caused by *G. candidum* alone occur in commercially-handled fruit. However, *M. implicata* has been the primary pathogen in sour rot infections on fruit originating from the Georgia peach-growing areas during the recent years. To my knowledge, there are no previous reports of *M. implicata* causing a rot on stone fruits.

Development of sour rot lesions on fruit inoculated with both *M. implicata* and *G. candidum* and held at 12.7 C was significantly more rapid than on fruit inoculated with *M. implicata* alone. At the lower storage temperature (4.5 C), or at ripening temperatures (21 C), the mixed infections were no more severe than infections by *M. implicata*. The ideal temperature for shipping commercially-packed peaches is close to 1 C. In many cases, however, fruit is shipped at higher temperatures; some approach incubation conditions optimal for mixed sour-rot infections. Proper precooling of peaches during packing and transit temperatures close to 1 C should be an integral part of the control of sour rot on peaches.

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