

A Reexamination of Mango Decline in Florida

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ABSTRACT

Ploetz, R. C., Benschler, D., Vázquez, A., Colls, A., Nagel, J., and Schaffer, B. 1996. A re-examination of mango decline in Florida. *Plant Dis.* 80:664-668.

Decline syndromes are recognized in virtually all mango-production regions. During a study to clarify the cause(s) of this problem in Florida, we sampled symptomatic tissue from different sources of affected mango (*Mangifera indica*) trees. In laboratory assays, the fungi that were most often isolated included, alphabetically: *Alternaria alternata*, *Cladosporium* sp., *Colletotrichum gloeosporioides*, *Dothiorella dominicana*, *Fusarium* spp., *Lasiodiplodia theobromae*, *Penicillium* sp., *Pestalotiopsis* sp., and *Phomopsis* spp. The relative abundance of the species that were isolated varied by sample source and date. During artificial inoculation experiments on mango cv. Keitt, *A. alternata*, *C. gloeosporioides*, *D. dominicana*, *L. theobromae*, and two species of *Phomopsis* caused all or some of the following decline symptoms: bud necrosis, tip dieback, gummosis, and vascular discoloration. The data suggest that mango decline in Florida is a disease complex involving several different fungi. *D. dominicana* is reported here as a pathogen of mango for the first time in the United States.

Mango, *Mangifera indica* L., is an important fruit crop in Florida. Commercial production occurs mainly in Dade County, where approximately 1,000 ha are now grown.

One of the primary production constraints in the area is a disease syndrome known as mango decline (24). Symptoms that are associated with mango decline are diverse and include the following: dieback of terminal shoots with or without accompanying defoliation, gummosis on branches and scaffold limbs, vascular discoloration, marginal chlorosis and necrosis of leaves, foliar nutritional deficiencies, and root degeneration. Many of the most important commercial cultivars in the area, such as Tommy Atkins and Keitt, are affected, and fruit yields can be reduced by more than 50% (R. C. Ploetz, personal observations).

Despite the importance of mango decline and prior research on this problem, its etiology in Florida remains confused. Several different organisms have been associated with the syndrome. Ramos et al. (23) isolated fungi from mango trees that were affected by tip dieback. *Botryosphaeria ribis* Gross & Duggar (anamorph: *Fusicoccum* sp. Corda) and, less commonly, a *Diplodia* sp. caused tip dieback after artificial inoculation. Smith and

Scudder (26) associated a *Diplodia* sp. with dieback of mango but did not confirm the pathogenicity of the fungus experimentally. McSorley et al. (17) detected a nematode, *Hemicriconemoides mangiferae* Siddiqi, at low but consistent levels on declining trees and suggested that it might play a role in the development of decline; however, they did not conduct experiments to verify this hypothesis. The association of host nutrition with decline has also been investigated. Schaffer et al. (25) assessed foliar nutrient levels in trees with and without decline. Although analyses of these data using the Diagnostic Recommendation and Integrated System (DRIS) approach indicated that manganese and iron were limiting elements in affected trees, the authors did not determine whether these deficiencies actually caused mango decline.

In other mango-production regions, fungi have been indicated as causes of decline symptoms (2,8,17,28). The fungi are usually Ascomycetes or Coelomycetes, which also cause stem-end rots of mango fruit (11-14).

Unfortunately, the decline disorders are not as well-characterized as are the stem-end rots. One or two species are reported to cause decline symptoms in most locations, but there is little agreement among locations. Factors that contribute to the occurrence and prevalence of, and symptoms caused by, the various fungi are poorly understood (20).

The reports outside Florida and the confused status of decline research within the state indicated that a reexamination of this problem in Florida was needed. During this study, our objectives were to: (i) identify fungi that are associated with mango

decline in the important production areas of Dade County, and (ii) determine the relative virulence and symptoms caused by the isolated fungi. This paper expands upon a previously published, preliminary report (21).

MATERIALS AND METHODS

Decline surveys. Samples were taken from six different sources in Dade County in 1994 and 1995 (Table 1). Three commercial orchards (two of Tommy Atkins and one of Keitt) and two experimental orchards (one each of Tommy Atkins and a cultivar collection) were sampled, as well as potted plants of Tommy Atkins. The orchard trees and potted plants were grown on Turpentine rootstocks in a calcareous soil that predominates in production areas in Dade County (Krome very gravelly loam: Ruptic-Alfic Lithic Eutrocrepts; clayey, mixed hypothermic; pH 7.5; sand, silt, and clay 65, 25, and 10%, respectively).

Terminal branches were taken from symptomatic trees during different times of the year and assayed for fungi in the laboratory within 24 h. Bark was removed from samples, and chips of tissue, ca. 2 × 5 mm, were excised from the interface of symptomatic and nonsymptomatic tissue. Tissue pieces were surface-disinfested in 70% ethanol for 10 s and in 10% household bleach for 2 min, rinsed in sterile H₂O, blotted dry on sterile paper towels, placed in empty 9-cm-diameter petri dishes, and submerged in molten Difco potato-dextrose agar (PDA) that had been cooled to 45°C and amended with 100 µg of streptomycin sulfate, 50 µg of rifamycin, and 6 drops of danitol 2HEC miticide (Chevron Chemical Corp., San Francisco, CA) per liter.

Dishes were examined after 2 to 10 days of incubation in the laboratory under fluorescent light. Fungi that were isolated were grouped according to their appearance on the isolation medium, and the different colony types were recorded for future reference and species identification. Representative isolates from the different groups were single-spored prior to storage on filter paper, as described previously (6). Stored isolates were grown on PDA and oatmeal agar (OMA) under fluorescent light for as long as 5 weeks and examined microscopically for identification purposes.

Inoculation experiments. Three experiments were conducted to determine the virulence and symptoms caused by a representative subset of the isolated fungi

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Florida Agricultural Experiment Station Journal Series R-04887.

Accepted for publication 6 March 1996.

Table 1. Sources assessed and fungi recovered during mango decline surveys

No. ^v	Cultivar	Date	Trees ^w sampled	Samples ^x per tree	Incidence of recovery (%) ^y								
					Aa	Cl	Cg	Dd	F	Lt	Pe	Ph	Other
1	Various ^z	3 Sep 94	4	4	0	0	35	0	5	27	25	2	5
2	Tommy Atkins	5 Oct 94	5	16	0	0	4	8	12	25	4	16	16
3	Keitt	6 Oct 94	5	16	0	0	0	17	13	4	0	39	26
4	Tommy Atkins	6 Oct 94	5	16	0	0	43	2	10	0	7	7	31
5	Tommy Atkins	21 Nov 94	12	4	0	0	0	6	81	0	0	0	12
1	Various ^z	22 Dec 94	4	12	0	0	25	0	25	50	100	50	0
6	Tommy Atkins	27 Jan 95	6	8	0	0	48	48	33	17	67	0	0
6	Tommy Atkins	28 Feb 95	40	8	40	13	8	0	0	18	13	10	0

^v No. = different sources from which samples were taken. No. 5 samples were from potted plants maintained in a greenhouse, whereas the remaining samples came from orchards.

^w A variable number of symptomatic trees were sampled in surveys of each source.

^x From each sampled tree in a given source, a variable number of tissue samples was taken.

^y Numbers are the percentage of the total number of fungi recovered that are represented by a given fungal taxon; fungi were Aa = *Alternaria alternata*, Cl = *Cladosporium* sp., Cg = *Colletotrichum gloeosporioides*, Dd = *Dothiorella dominicana*, F = *Fusarium* spp., Lt = *Lasiodiplodia theobromae*, Pe = *Pestalotiopsis* sp., Ph = *Phomopsis* spp., and other = nonidentified or other fungi. Total percentages for a sample date/source exceed 100% when multiple fungi were isolated from tissue.

^z Diverse cultivars in a collection including Joe Welch, Ott, Tamaraca, and Turpentine.

(Table 2). Isolates of fungi were taken out of storage and grown for 1 week under fluorescent lights on PDA amended with 5 g of yeast extract per liter (PDYE). In all experiments, a small incision was made on each stem with a sterile scalpel two nodes below the terminal bud. A square of mycelium of the test fungus, 5 × 5 mm, was placed in each incision and sealed immediately with Parafilm. For control treatments, blocks of PDYE were used instead of mycelium.

Grafted plants were used in all experiments: Tommy Atkins scions on Turpentine rootstocks were used in an initial study, and Keitt on Turpentine plants were used in two remaining experiments. Plants were grown in 11-liter pots in the calcareous soil described above and were watered daily. Plants in the Tommy Atkins experiment were fertilized with soluble 20-20-20 fertilizer and a chelated iron product (Sequestrene 138 Fe, Ciba-Geigy Corp., Greensboro, NC) every 2 to 3 months. In the Keitt experiments, plants were fertilized monthly with a 6-6-6 granular fertilizer and a foliar application of a commercial minor element product (Fer-a-gro; A.F.E.C. Fertilizer Company, Homestead, FL). Every 75 days, these plants were also fertilized with Sequestrene 138 Fe.

In the Tommy Atkins experiment, 13 plants, each with multiple branches, were utilized. Sixty-four branches on the plants were selected and randomly assigned to eight treatments (Table 3). In each of the Keitt experiments, six treatments were tested (Table 4). Treatments were replicated six times, and individual plants were considered replications even though as many as five branches were treated on each plant. For analyses of variance, mean data for a plant were used. In all three experiments, Parafilm was removed from the plants after 2 weeks; and after 3, 16, and 5 weeks, the respective experiments were terminated.

After experiments were completed, 12 to 16 tissue samples from each inoculated

Table 2. Fungi used during inoculation studies on Tommy Atkins and Keitt mango

Isolate	Species	Source
JCM 24	<i>Alternaria alternata</i>	Tommy Atkins, experimental, orchard 6
BT1A-2	<i>Colletotrichum gloeosporioides</i>	Tommy Atkins, commercial, orchard 2
BT1C-1	<i>Dothiorella dominicana</i>	Tommy Atkins, commercial, orchard 2
JCM 13	<i>D. dominicana</i>	Tommy Atkins, experimental, orchard 6
BM7A-2	<i>Lasiodiplodia theobromae</i>	Keitt, commercial, orchard 2
BT2E-1	<i>L. theobromae</i>	Tommy Atkins, commercial, orchard 2
AC11D-2 ^z	<i>Phomopsis</i> sp.	Tommy Atkins, commercial, orchard 4
AC12C-2 ^z	<i>Phomopsis</i> sp.	Tommy Atkins, commercial, orchard 4
AC13G-1	<i>Fusarium</i> sp.	Tommy Atkins, commercial, orchard 4
AC12E-2	<i>Fusarium</i> sp.	Tommy Atkins, commercial, orchard 4

^z Based on the morphology of AC11D-2 and its formation of α - and β -conidia, this isolate may be *Phomopsis amraii*; whereas the morphology of AC12C-2 and its formation of only α -conidia suggest that it may be *Phomopsis mangiferae*.

branch were assayed for fungi as described above. In addition, the extent of vascular discoloration above and below the point of inoculation, and the development of the following symptoms, were recorded: necrosis = death of the terminal bud above the point of inoculation, dieback = progressive necrosis advancing in a basipetal fashion from the terminal bud, and gummosis = conspicuous discharge associated with inoculation. Completely randomized experimental designs were utilized in all experiments, and data were analyzed with statistical programs in PC versions of SAS (SAS Institute, Cary, NC).

RESULTS

Decline surveys. In general, salient features for the studied fungi were those published by Johnson (11) and Sutton (27). The synonyms listed below are those of Farr et al. (10), Johnson (11), and Sutton (27).

Despite the stringent disinfestation treatments that were employed prior to isolation, a diverse array of fungi were recovered from symptomatic tissue. In alphabetical order, the following fungi were isolated most often: *Alternaria alternata* (Fr.:Fr.) Keissl.; *Cladosporium* sp.; *Colletotrichum gloeosporioides* (Penz.)

Penz. & Sacc. in Penz. (teleomorph: *Glomerella cingulata* (Stoneman) Spauld. & H. Schrenk); *Dothiorella dominicana* Petr. & Cif. (synonym: *Fusicoccum aesculi* Corda; teleomorph, *Botryosphaeria dothidea* (Moug.:Fr.) Ces. & De Not.); *Fusarium* spp.; *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl. (synonyms: *Botryodiplodia theobromae* Pat., *Diplodia natalensis* Pole-Evans, *Diplodia theobromae* (Pat.) W. Nowell; teleomorph: *Botryosphaeria rhodina* (Cooke) Arx, synonym: *Physalospora rhodina* Cooke); a species each of *Penicillium* and *Pestalotiopsis*; and *Phomopsis* spp. Teleomorphs for the above fungi were detected neither prior to isolation nor after prolonged growth on PDA and OMA under fluorescent light. Considerable variation existed among sources and sample dates for the various fungi that were isolated (Table 1).

Dothiorella dominicana and *L. theobromae* were distinguishable on isolation media, and their cultural and microscopic features on PDA and OMA matched those reported earlier (11). No attempt was made to identify the species of *Cladosporium*, *Fusarium*, *Penicillium*, and *Pestalotiopsis* that were recovered, since their relatively low rates of recovery or minimal impact during the first inoculation experiment

indicated that they were probably not important agents in the development of mango decline.

The two species of *Phomopsis* that were isolated could not be easily distinguished in culture and were not identified conclusively. However, based on cultural and microscopic criteria (11), tentative identifications were made for the isolates that were used in inoculation tests.

On PDA, the AC12C-2 isolate of *Phomopsis* initially formed tan-to-buff, felt colonies that turned dark brown after 2 weeks. Mature, multilocular stroma, which

formed in the center of colonies after 2 weeks, produced only α -conidia, 2×6 to $8 \mu\text{m}$, on conidiophores $25 \mu\text{m}$ in length. All but the latter characteristic matched those of *P. mangiferae* Ahmad, which produces much shorter conidiophores, 5 to $8 \mu\text{m}$. Thus, it is not clear which species of *Phomopsis* is represented by AC12C-2. In contrast, the AC11D-2 isolate of *Phomopsis* also produced tan-to-buff colonies on PDA, which darkened with age, but formed α -conidia 2×6 to $8 \mu\text{m}$ and β -conidia 1 to $2 \times 25 \mu\text{m}$ in discrete pycnidia after 2 weeks. Since β -conidia have not

been reported for *P. mangiferae*, AC11D-2 could be *P. amraii* Srivastava. The latter determination awaits comparisons of AC11D-2 with bona fide specimens of the latter species.

Inoculation experiments. Representatives of the most prevalent taxa isolated during the first surveys were used in the first experiment on Tommy Atkins (Table 3). Gummosis (one plant) and minor vascular discoloration were the only decline symptoms that developed on plants inoculated with agar disks. Bud necrosis and gummosis were caused by all fungi except the AC13G-1 and AC12E-2 isolates of *Fusarium*. None of the plants inoculated with AC13G-1 developed these symptoms, and only one plant inoculated with AC12E-2 developed bud necrosis. Oddly, the latter isolate did cause significant vascular discoloration in inoculated plants. Relatively few plants developed tip dieback in this experiment. Each of the isolates used to inoculate plants was isolated after the experiment ended (data not shown).

Isolates that caused conspicuous external symptoms of decline and significant vascular discoloration on Tommy Atkins in the first experiment were used in subsequent tests on Keitt. *A. alternata* and *D. dominicana* were also tested due to their prevalence in latter surveys and their recognized roles as mango pathogens (22).

Since results from both experiments with Keitt were very similar, they were combined for statistical analyses (Table 4). Bud necrosis, tip dieback, and gummosis did not develop, and very minor vascular discoloration developed on plants inoculated with agar disks. *C. gloeosporioides*, *D. dominicana*, and *L. theobromae* were the most damaging of the fungi tested. Significant necrosis, gummosis, and vascular discoloration developed in plants inoculated with these fungi; *L. theobromae* also caused significant tip dieback. In contrast, *A. alternata* and the AC11D-2 isolate of *Phomopsis* were somewhat less damaging: both caused significant bud necrosis, and AC11D-2 also caused significant gummosis. Since defoliation and other foliar symptoms of decline were not consistently associated with inoculation, these results are not shown. As for the first experiment, the isolates of fungi that were used to inoculate plants were usually the only ones recovered from plants at the end of the experiments (Table 5).

DISCUSSION

D. dominicana is reported here as a pathogen of mango for the first time in the United States. Although reports of *L. theobromae* on mango in the United States have not been made, its teleomorph, *B. rhodina*, was reported from Florida in 1960 (3). Voucher specimens of JCM 13 and BM7A-2, isolates of *D. dominicana* and *L. theobromae*, respectively, have been de-

Table 3. Symptom development on inoculated plants of Tommy Atkins mango^x

Treatment ^y	Symptom development ^z			
	Necrosis (no.)	Dieback (no.)	Gummosis (no.)	Vascular discoloration (mm)
Control	0	0	1	1.6 c
<i>Colletotrichum gloeosporioides</i> , BT1A-2	3 ns	1 ns	2 ns	14.3 a
<i>Fusarium</i> sp., AC13G-1	0 ns	0 ns	0 ns	6.9 a-c
<i>Fusarium</i> sp., AC12E-2	1 ns	0 ns	0 ns	13.9 ab
<i>Lasiodiplodia theobromae</i> , BT2E-1	2 ns	0 ns	3 ns	2.0 c
<i>L. theobromae</i> , BM7A-2	2 ns	0 ns	4 ns	5.5 bc
<i>Phomopsis</i> sp., AC11D-2	2 ns	1 ns	3 ns	10.0 a-c
<i>Phomopsis</i> sp., AC12C-2	4 *	3 ns	1 ns	5.9 a-c

^x Tommy Atkins scions were grafted on Turpentine rootstocks and grown in native calcareous soil in pots. Treatments were replicated eight times on individual branches that were randomly chosen on 13 plants. A completely randomized experimental design was used.

^y One 5×5 mm piece of mycelium from a yeast-extract-amended potato-dextrose agar (PDYE) culture of a given fungus was placed in a small incision two nodes beneath a terminal node. Pieces of PDYE were used for the control treatment.

^z Data under necrosis, dieback, and gummosis are the number of branch terminals in a treatment that developed a given symptom: necrosis = death of the terminal bud above the point of inoculation, dieback = progressive necrosis advancing in a basipetal fashion from terminal bud above point of inoculation, and gummosis = conspicuous cloudy or clear discharge associated with inoculation. Data in these columns are significantly different from those for the control treatment when followed by an *, according to Fisher's exact test at $P \leq 0.05$. Vascular discoloration = the mean total distance above and below the point of inoculation for a given treatment; means in these columns are not significantly different if followed by the same letter, according to DMRT at $P \leq 0.05$.

Table 4. Symptom development on inoculated plants of Keitt mango^y

Treatment	Symptom development			
	Necrosis (%)	Dieback (%)	Gummosis (%)	Vascular discoloration (mm)
Control	0 c	0 b	0 c	0.2 b
<i>Alternaria alternata</i> , JCM 24	44 ab	4 ab	4 bc	9.0 ab
<i>Colletotrichum gloeosporioides</i> , BT1A-2	36 ab	4 ab	17 ab	21.4 a
<i>Dothiorella dominicana</i> ^z	66 a	10 ab	50 a	19.1 a
<i>Lasiodiplodia theobromae</i> , BM7A-2	56 a	12 a	37 a	21.7 a
<i>Phomopsis</i> sp., AC11D-2	19 b	0 b	20 ab	5.1 b

^y Keitt scions were grafted onto Turpentine rootstocks and grown in native calcareous soil in pots. Treatments were replicated six times in each of two experiments, and individual plants were considered replications. Two to five branches were treated on a given plant. For ANOVAs, mean values for a replication were used. Data are combined means for both experiments. Necrosis, dieback, and gummosis are percentages of branch terminals in a given treatment that developed a given symptom: necrosis = death of the terminal bud above the point of inoculation, dieback = progressive necrosis advancing basipetally from the terminal bud located above the point of inoculation, and gummosis = conspicuous cloudy or clear discharge associated with inoculation. Vascular discoloration = the mean total distance above and below the point of inoculation. Numbers within a column are not significantly different according to DMRT, $P \leq 0.05$, if followed by the same letter. Percentage data were arcsine square root transformed prior to ANOVA.

^z Two different isolates of *D. dominicana*, BT1C-1 and JCM 1, respectively, were used in the first and second experiment on cv. Keitt. Since results with the two isolates were very similar, they were combined for statistical analyses.

posited in the American Type Culture Collection (Rockville, MD) under the accession numbers ATCC 200137 and ATCC 200136.

At least six different fungi, *A. alternata*, *C. gloeosporioides*, *D. dominicana*, *L. theobromae*, and two species of *Phomopsis*, caused symptoms of mango decline in the present study. Although the different taxa varied in virulence, they were all able to cause at least one symptom of mango decline (Tables 3 and 4). The most virulent of these fungi, *C. gloeosporioides*, *D. dominicana*, and *L. theobromae*, caused most of the symptoms that have been previously associated with mango decline. Interestingly, the bud necrosis, tip dieback, and gummosis symptoms that the latter fungi caused were usually indistinguishable. Only when *C. gloeosporioides* sporulated on inoculated branches was it possible to identify the pathogen that caused symptoms on these plants. Far less damage occurred on inoculated Tommy Atkins plants in the first experiment than on Keitt plants in the latter two experiments (Tables 3 and 4). Although the data for each cultivar cannot be compared statistically, the observed differences may reflect inherent differences in the susceptibility of Tommy Atkins and Keitt to these pathogens. Alternatively, it is possible that unknown abiotic factors influenced disease development in the different experiments.

Symptoms of marginal scorching and mineral deficiency on foliage and defoliation were not consistently associated with inoculation with any of the tested fungi, but have been reported previously as symptoms of mango decline (24). The latter symptoms may indicate the presence of factors that predispose the host to decline development, e.g., nutritional deficiencies, rather than the disease syndrome itself. Deficiencies of manganese and iron were previously associated with mango decline symptoms in Florida and Israel (15,25). Some of the decline symptoms reported in the past may be solely or primarily indications of nutritional deficiencies in this host.

Considerable variation existed among sources and sample dates for the various fungi that were isolated (Table 1). For

example, although *A. alternata* was the most prevalent fungus recovered during a thorough survey of an experimental field of Tommy Atkins (source 6, 28 Feb 1995 sample date), it was not recovered in a preliminary survey of the same field conducted 1 month earlier, nor was it recovered from any of the other sources that were sampled. Understanding the factors that influence such variability may be critical to understanding the role these fungi play in the decline syndrome.

L. theobromae is a common and widespread cause of decline symptoms on mango. It caused a serious dieback of mango in the Jaipur district of India in the 1940s (28), and in the Sonsonate area of El Salvador and in Egypt (1). It was associated with a trunk canker disease of mango in the Dutch East Indies (present day Indonesia) (19) and Malaysia (16), and also caused a gummosis and dieback of mango in Puerto Rico (2).

D. dominicana is also an important decline agent. Johnson et al. (12) indicated that it was the primary cause of mango twig dieback in Australia. After observing its teleomorph, *B. dothidea*, in the litter beneath trees, they felt that the ascospores it produced represented a significant source of inoculum. Darvas (7) indicated that *D. dominicana* was associated with branch dieback in South Africa, but did not observe *B. dothidea*.

Several different factors have been reported to predispose mango to disease caused by the above fungi. For example, in El Salvador *L. theobromae* was viewed as a weak parasite that caused damage to mango only after long periods of drought (1). In Puerto Rico, the same fungus damaged mango trees that had been sun-scorched (2), and in Indonesia sun scorch, tar, and tanglefoot all predisposed mango trunks to cankers caused by this fungus (19).

Decline syndromes similar to those described on mango are known on apple, citrus, and peach (4,5,9,18,29). The associated fungi are not aggressive pathogens, and the hosts are most apt to develop symptoms if they have been affected by cold temperatures, drought, or mechanical injury. These stresses, nutritional defi-

ciencies, and other factors may predispose mango to decline development in Florida.

L. theobromae, *D. dominicana*, and other stem-end rot pathogens of mango fruit are endophytes in mango (14). These fungi occur in healthy-appearing tissue and can colonize inflorescence and fruit tissues without inducing symptom development. Although their endophytic habit helps explain their distribution in mango-production areas around the world, additional work is needed to determine how and if they interact with abiotic factors that have been previously associated with symptoms of mango decline. We are now investigating these factors with the hope that mango decline might be managed in the future by controlling the most important predisposing factors.

ACKNOWLEDGMENTS

We thank Greg Johnson for confirming the identities of *L. theobromae* and *D. dominicana* and Zaragoza Alegria for technical assistance. This work was supported by an allocation for research on tropical fruit from the State of Florida.

LITERATURE CITED

1. Acuña, H. E., and Waite, B. H. 1977. La muerte regresiva del mango (*Mangifera indica* L.) en El Salvador. Proc. Am. Soc. Hortic. Sci., Trop. Reg. 21:15-16.
2. Alvarez-García, L. A., and López-García, J. 1971. Gummosis, dieback, and fruit rot disease of mango (*Mangifera indica* L.) caused by *Phylospora rhodina* (B. & C.) Cke. in Puerto Rico. J. Agric. Univ. Puerto Rico 55:435-450.
3. Anonymous. 1960. Index of Plant Diseases in the United States. U.S. Dep. Agric., Agric. Handb. No. 165.
4. Brown, E. A., II, and Britton, K. O. 1986. *Botryosphaeria* diseases of apple and peach in the southeastern United States. Plant Dis. 70:480-484.
5. Brown, E. A., II, and Hendrix, F. F. 1981. Pathogenicity and histopathology of *Botryosphaeria dothidea* on apple stems. Phytopathology 71:375-379.
6. Correll, J. C., Puhalla, J. E., and Schneider, R. W. 1986. Identification of *Fusarium oxysporum* f. sp. *apii* on the basis of colony size, virulence, and vegetative compatibility. Phytopathology 76:396-400.
7. Darvas, J. M. 1993. *Dothiorella dominicana*, an important mango pathogen in South Africa. Acta Hortic. 341:321-328.
8. Das Gupta, S. N., and Zaccariah, H. T. 1945. Studies in the diseases of *Mangifera indica*. Part V. On the die-back of mango trees. J. Ind. Bot. Soc. 24:101-108. (Abstr. in Rev. Appl. Mycol. 25:267-268).
9. Davis, R. M., Farrald, C. J., and Davila, D. 1987. Botryodiplodia trunk lesions in Texas citrus. Plant Dis. 71:848-849.
10. Farr, D. F., Bills, G. F., Chamuris, G. P., and Rossman, A. Y. 1989. Fungi on Plants and Plant Products in the United States. American Phytopathological Society, St. Paul, MN.
11. Johnson, G. I. 1994. Stem-end rot. Pages 39-41 in: Compendium of Tropical Fruit Diseases. R. C. Ploetz, G. A. Zentmyer, W. Nishijima, K. Rohrbach, and H. D. Ohr, eds. American Phytopathological Society, St. Paul, MN.
12. Johnson, G. I., Cooke, A. W., Mead, A. J., and Wells, I. A. 1991. Stem end rot of mango in Australia: Causes and control. Acta Hortic. 219:288-295.
13. Johnson, G. I., Mead, A. J., Cooke, A. W., and

Table 5. Recovery of fungi from inoculated plants of Keitt mango

Treatment	Fungi isolated (%) ^a				
	Aa	Cg	Dd	Lt	Ph
Control	1	2	2	2	1
<i>Alternaria alternata</i>	19	0	2	0	2
<i>Colletotrichum gloeosporioides</i>	0	24	1	0	0
<i>Dothiorella dominicana</i>	0	0	22	0	0
<i>Lasiodiplodia theobromae</i>	0	0	2	41	1
<i>Phomopsis</i> sp.	0	0	0	0	32

^a Data are mean percentages from two experiments on Keitt. A total of 12 to 16 tissue pieces were excised from above and below each point of inoculation and surface-disinfested before plating on potato-dextrose agar (PDA). Numbers are the percentage of tissue pieces that yielded the indicated species. Isolated fungi were Aa = *Alternaria alternata*, Cg = *Colletotrichum gloeosporioides*, Dd = *Dothiorella dominicana*, Lt = *Lasiodiplodia theobromae*, and Ph = *Phomopsis* sp.

- Dean, J. R. 1991. Mango stem end rot pathogens - Infection levels between flowering and harvest. *Ann. Appl. Biol.* 119:465-473.
14. Johnson, G. I., Mead, A. J., Cooke, A. W., and Dean, J. R. 1992. Mango stem end rot pathogens - Fruit infection by endophytic colonisation of the inflorescence and pedicel. *Ann. Appl. Biol.* 120:225-234.
 15. Kadman, A., and Gazit, S. 1984. The problem of iron deficiency in mango trees and experiments to cure it in Israel. *J. Plant Nutr.* 7:282-290.
 16. Lim, T. K., and Khoo, K. C. 1985. Diseases and disorders of mango in Malaysia. Tropical Press, Kuala Lumpur, Malaysia.
 17. McSorley, R., Parrado, J. L., and Goldweber, S. 1980. Observations on a mango decline in South Florida. *Proc. Fla. State Hortic. Soc.* 93:132-133.
 18. Menge, J. A. 1988. *Dothiorella gummosis*. Page 14 in: *Compendium of Citrus Diseases*. J. O. Whiteside, S. M. Garnsey, and L. W. Timmer, eds. American Phytopathological Society, St. Paul, MN.
 19. Muller, H. R. A. 1940. Overzicht van de belangrijkste Mangga-ziekten in Nederlandsch Indië. (English abstr.) *Rev. Appl. Mycol.* 19:355.
 20. Ploetz, R. C., and Prakash, O. Foliar, floral and soilborne diseases of mango. In: *The Mango*. R. E. Litz, ed. CABI, Wallingford, UK. In press.
 21. Ploetz, R. C., Benschler, D., and Vázquez, A. 1995. Fungi associated with mango decline in Florida. (Abstr.) *Phytopathology* 85:1150.
 22. Ploetz, R. C., Zentmyer, G. A., Nishijima, W., Rohrbach, K., and Ohr, H. D. 1994. *Compendium of Tropical Fruit Diseases*. American Phytopathological Society, St. Paul, MN.
 23. Ramos, L. J., Lara, S. P., McMillan, R. T., and Narayanan, K. R. 1991. Tip dieback of mango (*Mangifera indica*) caused by *Botryosphaeria ribis*. *Plant Dis.* 75:315-318.
 24. Schaffer, B. 1994. Decline. Page 43 in: *Compendium of Tropical Fruit Diseases*. R. C. Ploetz, G. A. Zentmyer, W. Nishijima, K. Rohrbach, and H. D. Ohr, eds. American Phytopathological Society, St. Paul, MN.
 25. Schaffer, B., Larson, K. D., Snyder, G. H., and Sanchez, C. A. 1988. Identification of mineral deficiencies associated with mango decline by DRIS. *HortScience* 23:617-619.
 26. Smith, P. F., and Scudder, G. K. 1951. Some studies of mineral deficiency symptoms in mango. *Proc. Fla. State Hortic. Soc.* 64:243-248.
 27. Sutton, B. C. 1980. *The Coelomycetes*. Commonw. Mycol. Inst., Kew, Surrey, England.
 28. Verma, O. P., and Singh, R. D. 1970. Epidemiology of mango dieback caused by *Botryodiplodia theobromae* Pat. *Ind. J. Agric. Sci.* 40:813-818.
 29. Whiteside, J. O. 1988. *Diplodia*-associated diseases. Page 14 in: *Compendium of Citrus Diseases*. J. O. Whiteside, S. M. Garnsey, and L. W. Timmer, eds. American Phytopathological Society, St. Paul, MN.