Sources of Inoculum and Infection Courts of *Diplodia gossypina* on Sweet Potato

Jing-Yi Lo and C. A. Clark

Graduate research assistant and professor, Department of Plant Pathology & Crop Physiology, Louisiana Agricultural Experiment Station, Louisiana State University Agricultural Center, Baton Rouge 70803-1720.
Accepted for publication 15 June 1988 (submitted for electronic processing).

ABSTRACT

Lo, J-Y., and Clark, C. A. 1988. Sources of inoculum and infection courts of *Diplodia gossypina* on sweet potato. Phytopathology 78:1442-1446.

Diplodia gossypina was isolated from the 1–2-cm base of sweet potato sprouts growing from mother roots that were bedded adjacent to diseased roots, bedded in infested soil, or inoculated with a conidial suspension before bedding. On established vines that were wounded, *D. gossypina* was recovered 1–2 cm above and 2–4 cm below the point of inoculation 2 mo after inoculation. The fungus was not isolated from the proximal or distal ends of storage roots harvested from field plots in which vine cuttings or established vines were artificially inoculated or when vine cuttings were planted in artificially infested soil. Storage roots harvested from field plots with artificially infested soil had higher disease incidence than roots from noninfested plots. Roots harvested from inoculated vine cuttings or established vines did not differ from the uninoculated control in disease incidence. Freshly harvested storage roots inoculated by placing artificially infested soil on cut ends developed significantly higher disease incidence (16%) than the control (1%). Conidia of *D. gossypina* survived in field soil for at least 1 yr. Survival of the conidia in soil was reduced by high soil moisture. Thus, the pathogen may survive in soil and infect storage roots through wounds made at harvest causing postharvest decay.

Additional keywords: Java black rot.

Java black rot of sweet potato (*Ipomoea batatas* (L.) Lam.) was first described by Clendenin in 1896 (4). Taubenhaus (14) gave the disease its name because he thought the fungus was introduced from Java to the United States. However, Harter et al (6) indicated that this disease was probably already as widely distributed as the sweet potato crop. It can be found in most sweet potato storage houses in the southern United States and is one of the most important postharvest diseases of sweet potato in the tropics. Historically, the causal fungus on sweet potato has been referred to by several names including: *Diplodia tubericola* (E. & E.) Taub., *Lasiodiplodia tubericola* E. & E., and *Botryodiplodia theobromae* Pat. Jones (8) summarized the pertinent literature on *Diplodia* and related genera and regarded these binomials and many others as synonyms of *Diplodia gossypina* Cke. *D. gossypina* has a host range of about 138 plant species in 58 families (8) and causes postharvest decay on many crops.

The modes of infection and the sources of inoculum for postharvest decays caused by *D. gossypina* have been studied on orange and mango. On orange, one-celled conidia from pycnidia produced in deadwood of the tree and two-celled conidia that survived on bark of deadwood were disseminated in water to necrotic tissue on the button (calyx and disc) of the immature fruit (2). Hyphae from these latent infections of the necrotic tissue invaded the mature fruit during button abscission, which usually occurred after harvest (3). On mango, the avenues of natural infection of uninjured fruits were the exposed surface of the attached pedicel or the stem-end scar when the pedicel was removed. Dead twigs and bark of the trees harbored the pathogen and served as sources of inoculum. When unripe fruit with and without pedicels were put in cellophane bags immediately after harvest, they remained free from infection at ripening time (11,12). In both cases, the sources of inoculum were airborne conidia.

Symptoms of sweet potato infection by *D. gossypina* usually begin at the ends of storage roots (5,10). Artificial inoculations of uninjured roots have not been successful (7,10). From inoculation experiments and field observations, Harter et al (6) concluded that this organism did not attack the vines in the field, and consequently it could not be transmitted through vines to storage roots. Jenkins's (7) observations suggested that all infections occurred at harvest and during subsequent handling.

Aderiye and Ogundana (1) reported that *D. gossypina* in infected yam leaves and stems could survive at 10, 30, and 60 cm depths in field soil for at least 4 mo. The pathogen survived in small pieces of infected yam tuber only 3 mo in nonsterile farm soil placed in petri dishes. There have been no reports on survival of conidia in field soil.

The objectives of this study were to determine the infection courts of *D. gossypina* on sweet potato and the sources of inoculum.

MATERIALS AND METHODS

Inoculum preparation. *D. gossypina* was isolated from infected storage roots collected at the Burden Research Plantation in Baton Rouge. Isolates originating from single-conidium transfers were tested for virulence on slices of sweet potato storage roots. The most virulent isolate, N-2, was maintained on silica gel (13). For vine and storage root inoculation, 6-mm-diameter mycelial disks were cut from the edge of a 2-day-old culture growing on potato-dextrose agar (PDA). Conidial suspensions were prepared with conidia collected from artificially inoculated storage roots incubated at room temperature (24 ± 2 C) for 1 mo and adjusted to 10^4 conidia/ml of distilled water. For mother root bedding, vine cutting planting, and storage root inoculation, natural field soil was infested with an aqueous conidial suspension to give a density of 10^4–10^5 conidia per gram of dry soil. Oat seeds used to infest field soil were saturated with distilled water and autoclaved at 121 C in autoclavable plastic bags for 1 hr on each of 3 consecutive days. Mycelial disks of isolate N-2 were placed in the oat seed substrate and incubated at 28 C for 2 wk. The colonized oat seeds were incubated in trays covered loosely with plastic bags at room temperature for an additional month to produce conidia. Field soil was infested by mixing infested oats (30 g equivalent before autoclaving) in the upper 15 cm around one sweet potato vine. All inoculation experiments were conducted with the sweet potato cultivar Centennial.

Isolation. Vines with leaves and roots removed, and proximal or distal ends of storage roots cut at harvest, were washed with running tap water to remove adhering soil. Segments (1-cm length of vines and 3-mm thickness of ends) were then cut from the tissue and surface sterilized with 1% sodium hypochlorite for 1 min and placed on PDA to isolate *D. gossypina*.

Transmission from mother roots to sprouts. Mother roots in field plant production beds in 1984 and 1985 were treated in one of four ways: drenching with thiabendazole (TBZ, Mertect 340F [42.28% a.i.], 237 ml/28.39 L of H_2O); placing adjacent to Java

black rot-infected roots; bedding in soil infested with *D. gossypina* (10^5 conidia per gram of dry soil); or inoculating skin-wounded (1-cm^2) roots with one drop of conidial suspension and incubating in a moist chamber overnight at room temperature before bedding. Each treatment had 20 mother roots in each of three replicate plots. All of the initial and regrowth sprouts were pulled, 10 sprouts sampled at random from each plot were cut at 1-cm intervals from the base for a distance of 15 cm, and the segments were processed for isolation of *D. gossypina*.

Transmission from vines to daughter roots. To investigate whether the pathogen could infect vine cuttings (sprouts cut above the soil line) during transplanting to the field and subsequently infect daughter roots, three treatments were compared in the greenhouse: healthy vine cuttings (indexed negative for *D. gossypina* by isolation) were planted in autoclaved soil; vine cuttings were dipped in a conidial suspension for 1 hr and planted in autoclaved soil, and healthy vine cuttings were planted in autoclaved soil infested with 2×10^5 conidia per gram of dry soil. Each treatment had 10 replicate vine cuttings each planted in a 25-cm-diameter pot. After 4 mo, storage roots were harvested by carefully washing the soil out of the pots to prevent wounding, and isolations were attempted immediately from vines below the soil line and proximal ends of the storage roots. The roots were then dipped in a mixture of thiabendazole (Mertect 340F 35 ml/7.57 L of H_2O) and dichloronitroaniline (Botran 75WP, 120 g/7.57 L of H_2O) to prevent infection through wounds at harvest. Percent of storage roots with Java black rot was subsequently determined after 4 mo of storage in paper bags at room temperature. A second experiment was conducted with established vines. Healthy vine cuttings planted in autoclaved soil were wounded with a scalpel 1 cm above or below the soil line 1 mo after planting in the greenhouse. Each of 10 wounded stems was inoculated with conidial suspensions, mycelial disks, or was left uninoculated. Isolations were attempted from stem segments and proximal ends of storage roots after harvest.

Infection of storage roots from soilborne inoculum at harvest. The following treatments were compared in field plots for subsequent infection by *D. gossypina* and incidence of Java black rot in storage roots in 1983 and 1984: vine cuttings planted in noninfested soil; vine cuttings planted in soil infested with oat seeds colonized by *D. gossypina* 1 mo after planting; vine cuttings dipped in a conidial suspension and planted in noninfested soil; vines established in noninfested soil and inoculated with a conidial suspension placed on wounds made above the soil line 1 mo after planting; and vines established in noninfested soil and inoculated with mycelial disks on wounds made above the soil line 1 mo after planting. Treatment of vine cuttings by dipping in a conidial suspension was omitted in 1983 and treatment of established vines by inoculating with a conidial suspension was omitted in 1984. Each treatment had four replications with 15 plants per plot in a randomized complete block design. Isolations were attempted immediately after harvest from proximal ends of 10 storage roots per plot in 1983. Thirty storage roots were collected at random from each plot and stored in a paper bag at ambient temperature and humidity (approximately 24–30 C, variable RH) in a storage building. In 1984, isolations were attempted immediately after harvest from the proximal and distal ends of 10 storage roots per plot. Half of the roots harvested from one plot were stored in a paper bag under ambient conditions and half were stored in another paper bag under conditions recommended for curing and storing (immediately after harvest, roots were placed at 30–32 C, 85–95% RH for 10 days, followed by storage at 13–16 C, ambient humidity). Disease incidence (percent of storage roots infected) was surveyed once a month for 4 mo.

To determine effectiveness of different inocula at harvest, freshly harvested storage roots from the field were cut at one end and inoculated with a conidial suspension, mycelial disk, infested soil (10^4 conidia per gram of dry soil), or infected tissue (1 cm of sweet potato stem infected with *D. gossypina* for 1 mo) in 1983 and 1984. Roots were stored in paper bags under ambient conditions. Each treatment had 25 roots in each of three replications. Disease incidence was surveyed once a month for 4 mo.

Survival of *D. gossypina* in soil. The number of pathogen propagules per gram of dry soil was detected by dilution plating on semi-selective media. A semi-selective medium (SM) was prepared with culture filtrates of *D. gossypina* grown in sweet potato-dextrose broth (SPDB) as a basal medium. Sweet potato-dextrose broth was prepared by steaming 200 g of peeled sweet potato in 1 L of distilled water, filtering through cheesecloth, adding 10 g of dextrose and autoclaving at 121 C for 15 min. A 6-mm-diameter mycelial disk of *D. gossypina* was transferred to SPDB (100 ml/500-ml flask), and the cultures were incubated 5 days on a platform shaker at room temperature. The culture was filtered through cheesecloth and the semi-selective medium was prepared with 960 ml of the filtrate, 18 g of agar, 33 mg of rose bengal, and 1 g of sodium propionate. SM was steamed 30 min to melt the agar, filtered through cheesecloth, dispensed to bottles, and autoclaved 15 min at 121 C. When cooled at 50–55 C, the following were added separately to 1 L of medium: streptomycin sulfate, 100 mg; penicillin G, 50 mg; oxgall, 1,000 mg; and pyrogallol, 50 mg. A modification of the above semi-selective medium (MSM) employed sweet potato broth (2 g of sweet potato storage root steamed in 100 ml of distilled water) instead of the culture filtrate of *Diplodia* in SPDB as the basal medium, in addition to the other ingredients listed above, and was autoclaved only 10 min at 121 C without prior steaming.

To determine how long the conidia of *D. gossypina* can survive in nonautoclaved field soil, Olivier silt loam soil was air-dried and passed through a 850-μm-opening sieve to remove large pieces of organic matter. The soil was then sprayed with a conidial suspension and mixed uniformly until the soil water content reached field capacity. A 15-cm-diameter clay pot was filled with the infested soil, covered with fine nylon mesh, and buried in the field with the top of the pot 2 cm below the soil surface. Densities of viable propagules were measured 1 wk later and then monthly with SM the first year (November 1982–July 1983) and MSM when repeated in the second year (September 1984–November 1985). Three randomly collected subsamples totaling 10 g were combined from each sample. A 10-fold dilution series was prepared and 1 ml from 10^{-2}, 10^{-3}, and 10^{-4} dilution was spread on each of five plates. plates.

To determine the effect of soil condition on survival of *D. gossypina*, artificially infested sandy loam soil was air dried in 12 15-cm-diameter clay pots in the greenhouse. Six pots each were remoistened to field capacity, three were flooded, and three were left dry. A sweet potato vine cutting was planted in three pots of remoistened soil, and the remaining pots were left fallow (second test only). Experiments were conducted from September 1984 to June 1985 and repeated from July to November 1985; the number of viable pathogen propagules per gram of dry soil was monitored monthly on MSM. Three random subsamples totaling 10 g were collected from 2 cm below the soil line from each pot.

RESULTS

Transmission from mother roots to sprouts. Sprouts did not develop disease symptoms in the plant production beds in either year. Results were similar both years, and the 1985 data are presented here. *D. gossypina* was isolated from 13–30% of the initial sprouts at 1 cm from their attachment to the inoculated mother roots (1-cm segments). Only 1-cm segments from mother roots placed adjacent to diseased roots had significantly higher isolation frequency (30%) than those from thiabendazole-treated mother roots (3%). *D. gossypina* was recovered from few of the 2–6 cm segments and none of the sprout segments above 7 cm. Fewer infections were detected in the regrowth sprouts than in the initial sprouts (Table 1).

Transmission from vines to daughter roots. In a preliminary experiment, data were collected only on disease incidence and were similar to the data reported here. The pathogen was recovered after harvest from stem segments up to 6–9 cm from the base of plants inoculated with a conidial suspension or planted in infested soil in the greenhouse, but greatest recovery (80–90%) was from the 1-cm segment (Table 2). The pathogen was isolated from 0–2% of

proximal ends, which were usually at 5–10 cm distance from the base of plants. The conidial density in infested soil was 8.5×10^3 conidia per gram of dry soil when the storage roots were harvested. None of the storage roots showed Java black rot symptoms after 4 mo of storage.

Most established vines that were inoculated in the greenhouse with conidial suspensions or mycelial disks did not develop symptoms, but several developed restricted cankers at the site of inoculation. No discoloration was found in tissue above or below these cankers. The pathogen was recovered from all points of inoculation and a high percentage of stem segments 1 cm from the point of inoculation. However, the pathogen was not recovered more than 2 cm above or 4 cm below the point of inoculation after 2 mo. Stems inoculated above or below the soil line showed similar patterns of pathogen distribution (Table 3). The pathogen was isolated from proximal ends of 1/15, 0/28, and 0/15 of storage roots harvested from the vines in one experiment at 2 mo after inoculating with conidial suspensions, mycelial disks, or leaving uninoculated, respectively, and 1/39, 0/29, and 0/44 of storage roots in another experiment at 4 mo after inoculation.

TABLE 1. Percentage of surface-sterilized sprout segments of sweet potato from which *Diplodia gossypina* was isolated 2 and 4 mo after various treatments at bedding

Sprouts[a]	Treatment[b]	Distance from mother roots (cm) 1	2	3	4	5	6	7–15
Initial	I	3	0	0	0	0	0	0
	II	30	3	0	3	0	0	0
	III	13	10	0	0	0	0	0
	IV	23	0	0	0	0	0	0
LSD	($P = 0.05$) = 21							
Regrowth	I	0	0	0	0	0	0	0
	II	3	0	0	0	3	0	0
	III	10	3	0	0	0	3	0
	IV	10	0	0	0	0	0	0
LSD	($P = 0.05$) = 14							

[a] Initial and regrowth sprouts were pulled 2 and 4 mo after bedding, respectively.
[b] I = mother roots in beds drenched with thiabendazole (237 ml/28.39 L H_2O), II = mother roots placed adjacent to diseased roots, III = mother roots bedded in infested soil (10^5 conidia per gram of dry soil). IV = mother roots inoculated with conidial suspension (10^4 conidia/ml) before bedding.

TABLE 2. Frequency of isolation of *Diplodia gossypina* from stems and proximal end of roots of sweet potato plants grown from inoculated vine cuttings in the greenhouse

Treatment	Isolation of *D. gossypina* (%)[a] Stem segment (cm) 1	2	3	4	5	6	7	8	9	10	Proximal end of root
Healthy vine cuttings in autoclaved soil	0	0	0	0	0	0	0	0	0	0	0
Healthy vine cuttings in infested soil[b]	80*	20	40*	30	0	20	20	10	10	0	2
Inoculated vine cuttings[c] in autoclaved soil	90	10	20	20	10	10	0	0	0	0	0

[a] Data were analyzed by chi-square analysis and numbers in the same row followed by an asterisk (*) were not significantly different ($P = 0.05$).
[b] Soil infested with 2×10^5 conidia per gram of dry soil.
[c] Vine cuttings dipped in conidial suspension (10^4/ml) for 1 hr before planting.

Infection of storage roots from soilborne inoculum at harvest. *D. gossypina* was not isolated from proximal ends of freshly harvested storage roots from the 1983 field plots. Incidence of Java black rot was not significantly different between the storage roots harvested from uninoculated controls and those harvested from inoculated vine cuttings or established vines, but storage roots from infested soil treatments had significantly higher disease incidence. In 1984, *D. gossypina* was not recovered from proximal or distal ends of freshly harvested storage roots. When the storage roots were stored in ambient conditions, only the storage roots harvested from infested soil had significantly higher disease incidence than the control. However, when the storage roots were cured before storage, disease incidence did not significantly differ among treatments (Table 4).

Storage roots inoculated with infested soil at harvest developed significantly higher ($P = 0.05$) Java black rot incidence (16%) than the control (1%). Disease incidence was not significantly different ($P = 0.05$) in storage roots inoculated with conidial suspensions, mycelial disks, or infected tissue (13, 11, and 5% of the roots, respectively).

Survival of *D. gossypina* in soil. In preliminary studies, when densities greater than 10^3 conidia per gram of dry soil were sampled, both SM and MSM had 90% recovery efficiency, but when densities were below 10^3 conidia per gram of dry soil, the recovery efficiencies of SM and MSM were 10 and 50%, respectively. In the first study, viable propagules of *D. gossypina* could be detected for 240 days, after which the density dropped below the threshold of detection (10^2 cfu per gram of dry soil) and other fungi quickly covered the medium. In the second study, propagule density was 1.6×10^3 cfu per gram of dry soil after 410 days, when sampling was terminated (Fig. 1).

The pathogen population in moistened soil artificially infested and left fallow or planted with sweet potato vines did not change over 4 mo (slope = 0). The pathogen population in dried fallow soil declined slowly (slope = −0.81), but in flooded fallow soil it quickly dropped (slope = −2.69). The results for the second test are presented in Figure 2.

DISCUSSION

Primary infection of sweet potato storage roots occurs through wounds incurred during harvest primarily from soilborne

TABLE 3. Frequency of isolation of *Diplodia gossypina* from surface-sterilized vine segments at various distances from the point of inoculation

Treatment[a]	Recovery of *Diplodia gossypinia* (%)[b] Below (cm) 5	4	3	2	1	Point of inoculation 0	Above (cm) 1	2	3	4
Inoculated above soil line										
Control	0	0	0	0	0	0	0	0	0	0
Conidia	0	0	10	20	100*	100*	80*	0	0	0
Mycelia	0	10	20	40	60	100*	70*	10	0	0
Inoculated below soil line										
Control	0	0	0	0	0	10	0	0	0	0
Conidia	0	0	0	20	70*	100*	60	10	0	0
Mycelia	0	30	30	40	40	100*	80*	0	0	0

[a] Vines were wounded with a scalpel at either 1 cm above or below the soil line 1 mo after planting. The wounds were either left uninoculated (control) or inoculated with a suspension of 10^4 conidia/ml (conidia) or mycelial disks from 2-day-old cultures on PDA (mycelia).
[b] Data were analyzed by chi-square analysis and numbers in the same row followed by an asterisk (*) were not significantly different ($P = 0.05$).

inoculum. Populations of *D. gossypina* added to natural field soil declined slowly for the first year and thus could persist in the soil between successive crops of sweet potato. This contrasts with the infection cycle for *D. gossypina* reported on other crops where infection usually results from dissemination of airborne conidia (2,3,11,12).

Vine cuttings, usually cut more than 15 cm distance from mother roots, are not an important means of transmission of *D. gossypina* to storage roots, even when they arise from infected mother roots; since the pathogen was isolated mainly from the base of sprouts and was not efficiently transmitted to storage roots. Infected sprouts appeared vigorous and did not show symptoms when pulled from the seed bed. Perhaps the pathogen does not extend to the sprouting end of the mother root until the sprouts develop their own fibrous feeder roots and independently absorb water and nutrients from the soil. However, some sprouts may have died before emergence if the proximal end of the mother root was infected before sprouts could root. Thiabendazole did not entirely prevent infection by *D. gossypina* in the seed bed and thus Java black rot may be a problem in sprout production. Reduced incidence of infection of regrowth sprouts compared to initial sprouts may have resulted from decay of the mother roots bearing infected sprouts after the first pulling. Soilborne inoculum of the pathogen caused infection at the base of vine cuttings through the cuts, but infection did not spread to daughter storage roots. The pathogen also failed to infect the storage roots through the periderm, distal ends, or attached fibrous roots before harvest.

The pathogen was not efficiently transmitted from inoculated vines to storage roots. Infected vines grew well in greenhouse and field conditions, but whether the yield of storage roots from these plants was affected, especially under water stress conditions, remains to be determined.

Failure to isolate *D. gossypina* from proximal or distal ends of storage roots freshly harvested from differently treated field plots indicates that latent infections had not developed before harvest. The incidence of Java black rot in field studies increased only in plots where soil was artificially infested with the fungus. Similarly, storage roots inoculated immediately after harvest by placing infested soil on wounds developed significantly higher disease incidence. When sweet potato storage roots are harvested, wounds always occur at the proximal ends as a result of detaching the roots from the vines and usually distal ends of the roots also break. Because infection did not occur through intact surfaces (7,10), and symptoms usually first appeared at the ends of storage roots (5,10),

TABLE 4. Effect of inoculation methods and storage conditions on incidence of Java black rot caused by *Diplodia gossypina* on sweet potato storage roots 4 mo after harvest from field plots

	Storage roots with Java black rot (%)		
	1983	1984	
Inoculation method	Not cured[a]	Not cured[a]	Cured[a]
Vine cuttings planted in noninfested soil	1	1	0
Vine cuttings planted in infested soil[b]	12	20	4
Vine cuttings inoculated with conidial suspension[c]	...[f]	3	2
Established vines inoculated with conidial suspension[d]	2	...	...
Established vines inoculated with mycelial disk[e]	1	9	9
LSD ($P = 0.05$)	6	10	

[a] Not cured = ambient temperature and humidity inside storage building. Cured = recommended condition (cured at 30–32 C, 85–95% RH for 7–10 days, stored at 13–16 C, ambient humidity).

[b] The soil was infested 4 wk after planting by incorporating *D. gossypina*-infested oats into the upper 15 cm.

[c] Vine cuttings were inoculated immediately before planting by dipping them in a suspension of 10^5 conidia of *D. gossypina* per milliliter.

[d] A drop of 10^4 conidia per milliliter of *D. gossypina* was placed on a wound made on vines with a scalpel 1 mo after planting.

[e] A 6-mm-diameter mycelial disk from the margin of a 2-day-old PDA culture of *D. gossypina* was placed on a wound made on vines with a scalpel 1 mo after planting.

[f] Not tested.

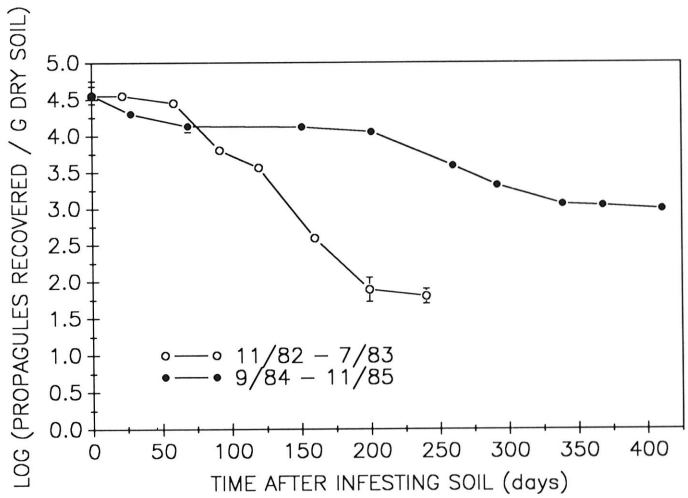

Fig. 1. Survival of *Diplodia gossypina* in pots buried in the field following artificial infestation of the nonautoclaved field soil as determined by dilution plating on semiselective media.

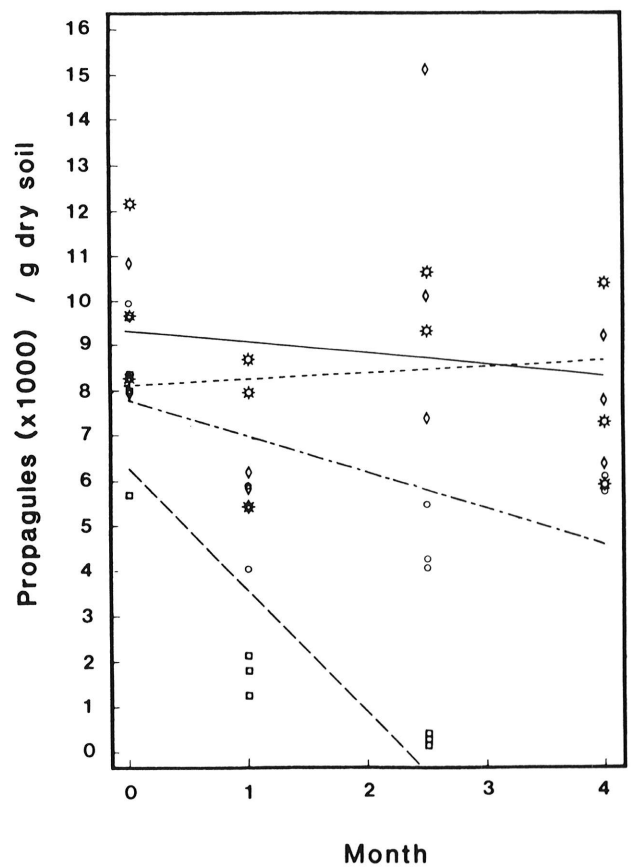

Fig. 2. Effect of soil conditions on survival of *Diplodia gossypina* in artificially infested soil over a 4-mo period from July to November 1985 in the greenhouse. Infested soil (*) was moistened to field capacity and a sweet potato vine cutting was planted (PR > F = 0.54); infested soil (◇) was moistened to field capacity and left fallow (PR > F = 0.81); infested soil (○) was moistened to field capacity and left fallow (PR > F = 0.04, $R^2 = 0.34$, $Y = 7.83 - 0.81X$); or infested soil (□) was flooded and left fallow (PR > F = 0.002, $R^2 = 0.77$, $Y = 6.27 - 2.69X$). The 4-mo sample for the flooded, fallow treatment was not included because it was below the threshold of detection.

it is likely that inoculum in soil adhering to these wounds incites infection. This is further supported by the fact that washing storage roots out of pots containing infested soil and dipping immediately in thiabendazole and dichloronitroaniline prevented infection. Furthermore, in one experiment, curing immediately after harvest, which enhanced the process of suberization and wound periderm formation, decreased disease incidence on storage roots harvested from infested soil.

In the field, conidia could survive in soil for at least 1 yr. In the greenhouse, the pathogen population in soil did not change during the 4-mo period when sweet potato vines were growing but was affected by soil moisture and decreased quickly when soil was flooded. An unusually heavy rainfall in Baton Rouge during spring 1983 may explain why the pathogen population quickly dropped below the detection threshold in the first field study.

Many storage roots left in the field plots were found to be infected by *D. gossypina* after harvest and contained conidia after 50 days. Because the fungus does not sporulate on plants in the field during the growing season, the observation that storage roots left in the field after harvest can become infected by *D. gossypina* (Lo, *unpublished*) and produce large numbers of conidia before winter may explain how soil in the field becomes infested. The semiselective media did not reliably detect the pathogen in samples of natural soil (Lo, *unpublished*). We speculate that this is because the distribution pattern of conidia in field soil is clustered, depending on where infected roots are located.

Conidia were produced on infected storage roots after only 1 mo in storage. They were deposited as visible, black, powdery masses on crates, and after 8 mo 7% of conidia recovered from the surface of used crates germinated (Lo, *unpublished*). Because the type of wound appears to have little influence on the probability of infection (7), the conidia on contaminated crates may serve as an additional source of inoculum when the storage roots were bruised and skinned on the sides of crates. Additional research is needed to address this possibility. Research is also needed to determine if insects such as cockroaches can transmit the pathogen in sweet potato storages and to further examine the influence of environment on survival of *D. gossypina* in soil.

The low incidence of infection reported in these studies is similar to the incidence of primary infection observed in commercial sweet potato storages. However, storage roots become progressively more susceptible to infection by *D. gossypina* the longer they are stored (9). Thus secondary cycles of infection, which occur when stored roots are handled for marketing or bedding, may be far more serious than the primary cycle. The factors affecting the development of primary cycles may be more important in determining the amount of inoculum available for secondary infections than in direct effects on loss of sweet potatoes.

LITERATURE CITED

1. Aderiye, B. I., and Ogundana, S. K. 1984. Survival of *Botryodiplodia theobromae* in yam tissues. Nova Hedwigia 40:171-176.
2. Brown, G. E. 1971. Pycnidial release and survival of *Diplodia natalensis* spores. Phytopathology 61:559-561.
3. Brown, G. E., and Wilson, W. C. 1968. Mode of entry of *Diplodia natalensis* and *Phomopsis citri* into Florida oranges. Phytopathology 58:736-739.
4. Clendenin, I. 1896. A new sweet potato disease. Bot. Gaz. 21:92.
5. Harter, L. L. 1955. Sweet potato diseases, Java black rot. USDA Agric. Farmer's Bull. 1059:21.
6. Harter, L. L., Weimer, J. L., and Adams, J. M. R. 1918. Sweet potato storage rots. J. Agric. Res. 15:337-368.
7. Jenkins, P. D. 1981. Differences in the susceptibility of sweet potatoes (*Ipomoea batatas*) to infection by storage fungi in Bangladesh. Phytopathol. Z. 102:247-256.
8. Jones, J. P. 1977. The current taxonomic status of *Diplodia gossypina*. Mycotaxon 6:24-26.
9. Lo, J-Y. 1986. Sources of inoculum and factors favoring infection of sweet potato by the Java black rot pathogen, *Diplodia gossypina* Cke. Ph.D. dissertation, Louisiana State University, Baton Rouge. 68 pp.
10. Palomar, M. K., Solis, A. D., and Bandala, H. S. 1980. Sweet potato tuber rot disease in the Philippines. Ann. Trop. Res. 2:111-121.
11. Pathak, V. N., and Srivastava, D. V. 1967. Mode of infection and prevention of Diplodia stem-end rot of mango fruits (*Mangifera indica*). Plant Dis. Rep. 51:744-746.
12. Pathak, V. N., and Srivastava, D. N. 1969. Epidemiology and prevention of Diplodia stem-end rot of mango fruits. Phytopathol. Z. 65:164-175.
13. Perkins, D. D. 1962. Preservation of Neurospora stock cultures with anhydrous silica gel. Can. J. Microbiol. 8:591-594.
14. Taubenhaus, J. J. 1913. The black rots of the sweet potato. Phytopathology 3:159-166.