

# Java Black Rot of Okinawan Sweetpotato

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Sweetpotato is a very important food in Hawai'i and a multimillion dollar export crop for the state. Perhaps the most important and widely grown variety is known as Okinawan sweetpotato, which is popular because of its flavor and because the edible portion inside the swollen storage root is purple. Okinawan sweetpotato crops can be damaged by a variety of pests, including a number of postharvest diseases of the fleshy roots caused by plant-pathogenic fungi.

Several significant and common postharvest diseases of similar appearance caused by fungi present in the soil threaten to reduce Okinawan purple sweetpotato production and profits for Hawai'i growers, marketers, and exporters. The diseases may occur together on the same sweetpotatoes, or in the same harvested batch of sweetpotatoes, or their pathogens can occur together and infect plants within the same areas of sweetpotato fields.

The major postharvest diseases of the Okinawan purple sweetpotatoes in Hawai'i include Ceratocystis black rot, Java black rot, Rhizopus rot, and scurf. The fungal pathogens cause most damage after the crops are harvested, handled, processed, and marketed and/or exported. Java black rot can be one of the most destructive postharvest diseases of sweetpotato. Infected tuberous roots may be completely decayed within 3–5 weeks after their harvest.

This publication discusses Java black rot disease symptoms on the Okinawan purple sweetpotato in Hawai'i and describes integrated practices designed to minimize sweetpotato losses to farmers and marketers.

#### The host

Most varieties of sweetpotato (*Ipomoea batatas* (L.) Lam.; synonym, *Ipomoea fastigiata* Choisy; family, Convolvulaceae) are susceptible to the disease. The Hawaiian name for sweetpotato, 'uala, refers to red-fleshed types that originated in the American tropics and are considered Polynesian "canoe plant" introductions. The purple-fleshed Okinawan type is probably a relatively recent introduction.

Sweetpotato is a tuberous-rooted perennial. The top is herbaceous, and the stems form a prostrate, slender, running vine. Lateral stem branches are usually not themselves branched. The green or purplish leaves are ovate-cordate, borne on long petioles, palmately veined, and angular or lobed, depending on the variety.

## The pathogen

Diplodia gossypina Cooke is a fungus that causes diseases of many crops, and different names have been or are used for the pathogen on the different hosts. In the United States the preferred name for the pathogen's anamorph (asexual stage) was initially Diplodia tubericola. In other countries the preferred name was Botryodiplodia theobromae. Another synonym is Diplodia theobromae. No teleomorph (sexual stage) has been found associated with sweetpotato.

The pathogen host range includes up to 138 plant species in 58 plant families, and the general symptom is postharvest decay. Infection in sweetpotato is primarily through wounds in the tuberous root periderm (i.e., the "skin"), broken root tips damaged during harvest, holes



Infection usually begins from the tips of the sweetpotato, most commonly where it was detached from the vine.

All photos by S. Nelson

created by harvesting tools, abrasions from rough handling and packing containers, and chewing or boring by insects such as weevils.

#### **Disease symptoms**

On fleshy roots in storage, general symptoms are as follows:

- Internal rot and decay usually progress from one end or both ends of the sweetpotato, especially where roots are detached from vines and a wound is created for the pathogen to enter. Decay may be restricted to the terminal 1–2 cm of the root, or the decay may expand much farther into the root. Affected periderms have a brown to black discoloration that advances along the root. Underneath the diseased periderm inside the sweetpotato, the tissue eventually turns black. Infected sweetpotatoes may mummify, and the periderm may be wrinkled. The infected tissue is initially light to dark brown, later turning dark grey to solid black.
- Mummification: The decay is firm and moist in consistency until mummification sets in, and the diseased root dries out and becomes extremely hard.
- Exterior lesions: On exterior surfaces, lesions may appear raised, circular, and brown near their margins, but black within the centers, and the periderm stays relatively intact. Some lesions on the root sides may not cause much interior rot.



The external appearance of the periderm of affected sweetpotatoes includes a brown to black discoloration that advances along the long axis of the root (3–5 weeks post-infection).

### **Disease signs**

Signs of Java black rot disease (i.e., the visible presence of the pathogen) develop after infection and are diagnostic evidence of the disease. They include:

- Black, raised, stromatic masses of the pathogen: the fungus breaks through the periderm of infected roots.
   These pathogen masses have shapes of raised domes or cushions on the sweetpotato storage-root surface.
   The stromatic masses contain numerous pycnidia, both near the surface and/or entirely embedded within the stroma.
- Black powder created by the released spores: Numerous conidia (fungal spores, asexual) release as the stroma decomposes, forming a distinctive black powder which may coat the surface of nearby objects such



Dark grey to black fungal growth may develop on cut or broken ends of the root.



Where the discolored periderm is peeled away, the tissue beneath it appears black. Over time, as the potato mummifies, the periderm wrinkles.

as other sweetpotatoes or storage crates or boxes.

- Fungal growth at cut ends of sweetpotatoes: Dark grey to black colored fungal growth can develop on the cut ends of affected sweetpotatoes.
- Spores of the pathogen: microscopically, the conidia are initially single celled and without color, about 11–14 X 18–20 micrometers in size. Later conidia become two-celled and dark brown.

Several other soil-borne, fungal diseases of Okinawan sweetpotatoes that occur in Hawai'i may be confused



About 3–5 weeks after harvest, the interior decay of roots is first light to dark brown in color, and turns dark grey to solid black as the decay progresses. The decay is initially firm and moist in consistency. Where the periderm is peeled away (lower photo), discolored, blackened tissues beneath it are revealed.

with the initial symptoms of Java black rot. They may occur together on the same sweetpotatoes infected by *D. gossypina*, or even in the same lesions. The similar diseases are Ceratocystis black rot, Rhizopus soft rot, and diseases caused by *Fusarium* sp.

## Dispersal of the pathogen

The pathogen spreads between locations by the following means:

- water running through fields carrying soil particles
- infested sweetpotato wash water
- sweetpotato weevils and cockroaches that carry the pathogen from infected to healthy roots in storage and, since they are capable of creating wounds, may

- effectively introduce the fungus into sweetpotatoes
- soil on equipment and tools, tractors, truck tires, shovels, handling baskets, crates, shoes, hands
- infected sweetpotatoes.

#### Infection

Infection occurs only through wounds usually created during harvesting or handling, initiated by inoculum (e.g., fungal spores or mycelium) existing in the field soil or on soil particles on dirty crates or dirty tools. Infections can occur rapidly, within 6 hours. The pathogen does not infect properly cured sweetpotatoes unless they are injured after curing and then exposed to the pathogen. However, when the sweetpotato roots are washed, sorted, and packaged for shipment to markets, or when they are handled for sowing for plant production, many new wounds may be created, and the fungal conidia produced in the primary cycle of infection may be spread over the sweetpotato root surface and enter wounds, holes, or cracks. The resultant secondary cycles of infection are often far more destructive than the primary cycle.

The conidia of *D. gossypina* may survive in the soil for several years. The fungus may also survive in small culled roots or vines of sweetpotato left in the field and infested after harvest, or in debris of sweetpotato or other susceptible crops.

The optimum range for infection and development of Java black rot is 68–86°F (20–30°C). The disease develops over a wide range of relative humidity levels.

## **Effects of curing sweetpotatoes**

Okinawan purple sweetpotatoes usually are not cured in Hawai'i, although the practice of curing would help control this and several other postharvest diseases. Curing is done to promote wound healing on sweetpotatoes before they are washed, handled, packed, transported, and marketed. However, the optimal conditions for Java black rot are similar to those recommended for curing sweetpotatoes. Yet, immediate curing of freshly harvested roots does reduce the incidence of the disease on other types of sweetpotatoes if the curing and storage conditions are carefully controlled. Curing should be done only once. If sweetpotato roots are cured again after they have been stored for 5-8 months, to promote the healing of wounds inflicted during packing, more Java black rot disease may occur than by returning the potatoes to the recommended storage temperature, which is approximately 60°F (16°C). Since conditions for infec-



Black stromatic masses of the fungus break through the periderm of infected roots and take the shape of domes or cushions on the root surface. These stromatic masses contain numerous pycnidia, both near the surface and entirely embedded within the stroma.

tion by *D. gossypina* and conditions for wound healing in fleshy roots are so similar, inoculum potential is very important in determining the incidence of infection and disease development.

In storage, the fleshy roots may become more susceptible to Java black rot over time. Sweetpotatoes stored for 5–8 months are more susceptible to infection than those freshly harvested. Those handled after storage for marketing or bedding are exposed to higher inoculum densities due to conidia having been produced from primary infections, and they are also more susceptible.

Chilling injury to the sweetpotatoes increases their susceptibility if they are subsequently returned to higher temperatures. However, *D. gossypina* does not develop rapidly at recommended storage temperatures, and its development is negligible at temperatures that induce chilling injury. Adequate gas exchange is also important to retarding the development of the disease and the roots having been exposed to flooding in the field or to poor ventilation in storage are more susceptible to the disease.

#### Integrated pest management practices

**Choice of planting site.** Avoid planting sweetpotatoes in poorly drained, heavy soils; avoid high-rainfall areas.

Plant disease-free materials. The best practice for



Signs of the pathogen develop at later stages of infection and are diagnostic evidence of the disease (about 3–5 weeks after infection). Black stromatic masses of the fungus break through the periderm of infected roots and take the shape of domes or cushions on the root surface. These stromatic masses contain numerous pycnidia, both near the surface and entirely embedded within the stroma.

planting is to use sweetpotato vines cut above the soil line for transplanting (avoid planting vines with their roots and soil attached to them).

**Crop rotation and cropping cycle.** Rotate sweetpotatoes with crops not usually infected by the pathogen; do not grow sweetpotatoes in the same field for more than 2 or 3 years without rotating to other crops that do not host the pathogen.

**On-time harvest.** Where possible, sweetpotatoes should be harvested before exposure to flooding in the field.

**Harvesting practices.** Minimize wounding of sweetpotatoes during harvesting; use clean baskets for transporting the potatoes; use clean potato digging equipment.

**Field sanitation.** Clean up all sweetpotato plant parts after harvest; do not allow them to remain in the field.

**Storage containers.** Prior to harvest, any previously used storage containers should be washed and disinfested.

**Postharvest handling.** Minimize sweetpotato injury and wash them with clean water. When sweetpotatoes are removed from storage for washing, sorting, and packing for market, handle them quickly. Slow handling of the sweetpotatoes is a weak point in disease control programs. Sweetpotatoes should not be re-cured after washing and packing, but should be held as close to 60°F as possible until they are consumed.

**Curing and storage.** For best control of postharvest diseases of sweetpotatoes, cure them immediately after



Weevil holes in a sweetpotato. Certain insects, including sweetpotato weevils and cockroaches, may carry *Diplodia gossypina* from infected to healthy roots in storage and, since they are capable of creating wounds, may effectively introduce the fungus into a suitable infection court for the pathogen.



Gray and black fungal mycelium can develop with a few days after slicing open a diseased sweetpotato.

harvest and then refrigerate or store them at a cool temperature. To cure the potatoes, hold them at 86–95°F (30–35°C) at 85–95% relative humidity for 5–10 days and then after processing refrigerate them at approximately 60°F (16°C).

**Fungicides after harvest.** Treating potatoes with an effective fungicide immediately after handling reduces subsequent development of the disease, but few effective fungicide treatments are available for roots destined for human consumption. Botran 75W is less effective than thiabendazole but, at allowable concentrations, it may

Table 1. Some fungicides registered in Hawaii for application to sweetpotatoes (*Ipomoea batatas*) or to sweetpotato cultivation soils for management of Java black rot and some other soilborne diseases.\*

Product name, application	Active ingredient(s)	Formulation
Botran 75-W (postharvest application to non-stored commodity)	Dicloran (75%)	Wettable powder
HTH Calcium Hypochlorite Granular 75 Granular Chlorinator by HTH (many other products with same active ingredient are available) (postharvest application to non-stored commodity)	Calcium hypochlorite (78%)	Granular
Maxim 4SF (seed treatment)	Fludioxonil (40.3%)	Soluble concentrate
Mertect 340-F (root dip\sweetpotato sprouts)	Thiabendazole (42.3%)	Flowable concentrate
Telone (postharvest application to non-stored commodity) (restricted-use pesticide)	1,3-Dichloropropene (82.9%), Chloropicrin (14.9%)	Solution, ready to use

<sup>\*</sup>Source: Hawaii Pesticide Information Retrieval System (HPIRS). Always follow pesticide label instructions and allowances. Some products may require a pesticide applicator's license issued by the state of Hawai'i.

slightly reduce disease incidence.

**Transport.** Protect sweetpotatoes in carboard boxes from rainfall (keep boxes dry); cover transport trucks with waterproof plastic tarps.

**Marketing.** Avoid enclosing diseased sweetpotatoes in unventilated plastic bags in markets; use perforated plastic bags or open bins or boxes for display and marketing; keep sweetpotatoes in markets at about 60°F if possible.

Retention of samples after handling. Retain some potatoes in storage boxes to observe them for symptom development after their harvest and processing; submit disease samples for diagnoses to the CTAHR Agricultural Diagnostic Service Center (via Cooperative Extension Service offices) to confirm the disease identity and to obtain disease management recommendations.

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