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Compendium of Sweet Potato Diseases

C. A. Clark and J. W. Moyer

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Preface

Relative to its importance as a world food crop, very little investment has been made in sweet potato disease research. The monograph written by L. L. Harter and J. L. Weimer in 1929 has stood as the uncontested "bible" of sweet potato disease information. It is a monument to their accuracy, scope, and thoroughness that their work has held such a singularly lofty position. Since that time two plant pathologists have dedicated their careers to the study of sweet potato diseases. Weston J. Martin, at the Louisiana Agricultural Experiment Station, Louisiana State University, and Lowell W. Nielsen, at North Carolina State University, are cited numerous times throughout this book. It is only because of their efforts in identifying and, more importantly, developing practical control procedures for the diseases of sweet potato, that we could afford the time to undertake the task of compiling this compendium. It is therefore with great admiration and respect that we dedicate this book to them.

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Introduction

Importance and Utilization of Sweet Potato

Among the food crops of the world, sweet potato ranks seventh in production, based on weight, according to recent documents of the Food and Agriculture Organization. It ranks fourth in the tropics. Several attributes of sweet potato account for its prominence and the recent resurgence in interest in the crop. It withstands environmental extremes, such as droughts and typhoons, which few crops can tolerate. It quickly covers the ground, reducing the need for herbicides and cultivation. Insecticide and fungicide use is low, and sweet potato grows well with small supplements of nitrogen and in a wide range of soil pH without the addition of lime.

Sweet potato is versatile, and genotypes can be selected to fit the needs of a particular use or consumer group. Sweet potato storage roots are commonly consumed directly as food, and shoot tips are a minor food item. The edible roots are not stored for long in the tropics; the indeterminate growth of sweet potato and multiple plantings permit the harvesting of storage roots during most of the year, making them available year round with little use of storage. Both storage roots and vines are fed to animals. Storage roots are also used as a source of starch and for fermentation products, including wine, ethanol, lactic acid, acetone, and butanol.

Two broad categories of sweet potatoes are used for the production of storage roots for human consumption: the staple type and the dessert type. The staple type is most widely grown in the tropics. It generally has white to cream-colored flesh and a higher content of dry matter, starch, and protein than the dessert type. High dry matter and high starch contents are important for industrial uses and animal feed, and high protein content is needed for animal feed. The dessert type generally has orange flesh and a higher content of β -carotene and simple sugar than the staple type. Some cultivars are intermediate between these extremes. Less effort has been expended on developing cultivars for use as shoot tips, and thus few genotypes have been developed specifically for this purpose.

Sweet potato has been a staple in the diet of many societies. Diets vary tremendously from one growing region of the world to another. The highlanders in Papua New Guinea rely on sweet potato for 60–90% of their energy requirements and as a major source of protein. The People's Republic of China produces about 75% of the world's sweet potatoes, production of the crop being second only to that of rice in that country. Sweet potato is an important food crop in many countries throughout the tropics. In the United States it is produced for consumption as a supplemental vegetable and in many regions is associated strictly with holidays. Its use as food has declined in some countries as the affluence of consumers has increased. In Taiwan and Japan sweet potatoes for industrial uses and animal feed have greater markets than those sold for human consumption.

The utilization of sweet potatoes is affected by the availability

of storage for the roots. The quality of many cultivars, including taste, may improve with storage. Indeed, in the United States "cured" sweet potatoes command a premium price. Improved storage facilities and storage management methods are needed to serve the expanding urban markets in tropical growing regions.

Sweet potato is a high-energy food. Its storage roots have a total carbohydrate content of 25–30%, of which 98% is considered easily digestible. Sweet potato provides an estimated 113 cal/100 g, whereas white potato provides 75 cal/100 g. Despite the caloric difference, white potato may elevate blood sugar content more than sweet potato. Sweet potato is an excellent source of provitamin A carotenoids. An average serving of a dessert type provides 5,345 international units (IU) of vitamin A per 100 g, or 121% of the recommended dietary allowance. However, different types and cultivars vary in carotene content over the range of 0–8,000 IU/100 g. Sweet potato also provides a source of vitamin C (20–30 mg/100 g), potassium (200–300 mg/100 g), iron (0.8 mg/100 g), and calcium (11 mg/100 g). The amino acid content is relatively well balanced, with a higher percentage of lysine than rice or wheat but a somewhat limited content of leucine. However, like most other starchy root and tuber crops, sweet potato has a relatively low protein content, ranging from 2.5 to 7.5% of dry weight, depending on the genotype. A combination of legumes and sweet potato could combat protein-calorie malnutrition in some areas. Current studies of the role of vitamin A and fiber on long-range human health may further enhance the image of sweet potato.

The potential for industrial uses of sweet potato has not been extensively studied or exploited. There is great potential for breeding cultivars with higher yields and a higher percentage of dry matter. Initial efforts to use sweet potato as a source of starch in the United States were not economical, but in Japan it is used for industrial production of starch. It is a potential raw material for ethyl alcohol production: 100 kg of sweet potatoes can yield 14.5 L of ethanol, compared to 11.4 L for potatoes; 11.9 L for sugar beets; 17.6 L for wheat, barley, and oats; and 44.9 L for maize. Since sweet potatoes generally bring a relatively high price when marketed for human consumption, it is unlikely that they will be grown exclusively for ethanol production. However, they are potentially a much greater source of ethanol on an acreage basis, because they generally yield more than other crops. Perhaps the unmarketable portion of the crop, such as culls, jumbos, or even partially decayed storage roots, might be diverted to ethanol production. This potential has not yet been realized, perhaps because of the lack of genotypes selected specifically for ethanol production.

Historically, sweet potato used as animal feed has been a by-product of crops grown for human consumption. In the United States only sweet potatoes culled from grading lines or those left in the field after harvest have been fed to animals. However, in Asia their use as animal feed is increasing. Unfortunately, the roots contain sufficient amounts of trypsin inhibitor to require heat inactivation of this inhibitor before they are fed to animals in large quantities. Technologies are currently being

developed to produce dried sweet potato products suitable for both storage and subsequent use as animal feed.

The importance of sweet potato as a crop cannot be measured solely on the basis of past or present production and utilization statistics. Its potential as food, feed, and biomass for industrial purposes far exceeds its current utilization. It has one of the greatest potentials among the world's food crops for yield improvement through the development of new cultivars. Nevertheless, yields of sweet potato fell by 2% in Asia between 1963 and 1979, while yields of rice in the same region increased by 32%. Furthermore, existing technology has not been efficiently employed by sweet potato growers. When actual yields obtained by farmers are compared with recorded yields on experiment stations in the same region, it appears that yields could potentially be improved by as much as 428% in India, 600% in the Philippines, 146% in Nigeria, 75% in Japan, 115% in Korea, and 246% in the United States. Perhaps the developing international interest in sweet potato will stimulate new research aimed at realizing the potential of this crop. Much less research has been reported on sweet potato than on Irish potato, or white potato (*Solanum tuberosum*). For example, in 1984, the *Annual Review of Plant Pathology* listed 409 citations of research on potato but only 16 citations of research on sweet potato.

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The Sweet Potato Plant

The sweet potato, *Ipomoea batatas* (L.) Lam., is a dicotyledenous plant in the family Convolvulaceae, the morning glory family, which contains several important species of weeds and cultivated ornamentals. Certain moist-fleshed types of sweet potato are referred to in the United States as yams. The sweet potato is in reality not even closely related to the true yam, a monocot in the family Dioscoreaceae. There is a long list of common names used for the sweet potato in various regions of the world, but, interestingly, widely separated geographic groups use variations of one of three names: *batatas*, *kamote*, and *kumara*.

The sweet potato is thought to have originated in Central America or South America, in the region between the Yucatán peninsula of Mexico and the mouth of the Orinoco River, in Venezuela. It has been used by humans not only in this region but also on many of the islands in the South Pacific Ocean for at least 2,000 years. There has been considerable speculation as to how the plant was disseminated from South America to Polynesia. It has been suggested that Peruvian sailors riding the Humboldt Current carried it to the islands or that Polynesian traders were responsible for the dissemination. European explorers and traders are thought to be responsible for the movement of the sweet potato from the Caribbean region to North America and the Mediterranean basin. There are several possibilities for its introduction into Asia, where the vast majority of the world's sweet potatoes are now grown.

The sweet potato is a vegetatively propagated perennial plant grown as an annual. Because it does not have a definable maturity, it can be harvested following growing seasons of widely varying lengths.

The plant produces several different types of roots, which have been classified by several different schemes. Generally, the roots originate adventitiously from the vine or as a result of lateral branching of other roots. Young adventitious roots

arising from the internodal area are thin. Roots arising from nodes are thick. The thin roots usually differentiate into primary fibrous roots, which are heavily lignified and do not enlarge in diameter, or occasionally into pencil roots, which are less extensively lignified and enlarge by divisions in the vascular cambium to diameters of 5–15 mm. Thick roots tend to differentiate into either pencil roots or storage roots. The latter have little lignification and enlarge as a result of division in the vascular cambium and many anomalous cambia throughout the pith of the root. Storage roots have a pentarch or hexarch arrangement of vascular tissue and have a phellogen that produces a thin layer of periderm on the surface of the root. Storage roots are not tubers. Most are initiated at the first stem node below the soil line, to which they are attached by a stalk of thinner root.

The sweet potato plant is predominantly prostrate, with a vine system that expands horizontally very rapidly and develops a relatively shallow canopy. Considerable variation in branching pattern, internode length, and overall vine length exists among genotypes. Three general types have been recognized: bunch (bush), intermediate, and vining. The leaves of different genotypes vary widely in size, length of the petiole, and shape, from deeply indented or lobed to broad and entire. The shape and size of leaves may also vary considerably on a single vine.

Sweet potato flowers are complete, with a compound, superior pistil, five stamens attached to the corolla but not attached to each other, and petals united into a trumpet-shaped corolla. The corolla is usually white at the entire margins with a pink to purple throat. Seed are borne in a capsule and have a very hard seed coat. The seed do not have a strong physiological dormancy but are generally scarified mechanically or with acid to promote germination. Seedlings have characteristically bilobed cotyledons, similar to those of many morning glories.

Sweet potato is a hexaploid with 90 chromosomes. Most species of *Ipomoea* have 30 chromosomes, and there has been considerable speculation and controversy on the genetic origins of sweet potato. One suggestion is that it involved a cross between a tetraploid progenitor (possibly *I. trifida*) and a diploid.

Under the best circumstances sweet potatoes set relatively few viable seed. Many genotypes do not flower readily, if at all. Flowering can be enhanced by training the vines onto trellises or by grafting onto other *Ipomoea* spp. as rootstocks. *I. setosa* and *I. trichocarpa* are frequently used for this purpose. Some genotypes are sterile, producing defective pollen. Self-compatibility, self-incompatibility, cross-compatibility, and cross-incompatibility all exist in sweet potato; however, self-incompatibility is the predominant condition. Several cross-incompatibility groups exist among the available pool of sweet potato germ plasm. These factors, combined with the hexaploid constitution, make genetic studies difficult. Nevertheless, considerable improvements have been made in sweet potato through breeding.

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Cultivation and Storage

Sweet potato is the most widely adapted of the agriculturally important root crops native to the humid tropics. It can be

found in many tropic and subtropic regions of the world and is grown in many temperate regions as well. Large acreages are reported at northerly latitudes greater than 35°. Sweet potatoes require a minimum frost-free period of 120–150 days, a minimum average daily temperature of 24°C, and approximately 2 cm of moisture per week, uniformly distributed during the growing season. Production is favored by well-drained, fine, sandy loam soils. Sweet potatoes grow well over a wide range of soil pH (4.5–7.5). However, adjustment of the pH may be necessary in areas where soil acidity or alkalinity contributes to other problems. For example, soil acidity is a problem in soils having a potential for aluminum toxicity. Conversely, reduction of the pH to 5.2 may be necessary in fields with a history of soil rot, the disease caused by *Streptomyces ipomoeae*.

Fertilizer requirements for sweet potatoes are dependent on local soil type, previous condition of the soil, and environmental factors such as leaching of nutrients in areas of high rainfall. In general, nitrogen and phosphorus are used in relatively moderate amounts, compared to potassium. Local soils may require application of minor elements. Boron deficiency is common in some soils and, if not corrected by the addition of borax, may result in mild stunting and superficial necrosis on roots. Further details on nutritional requirements of sweet potatoes can be found in Part II, on noninfectious disorders.

Cultivation strategies vary in tropical regions, where plants are produced and maintained in the field throughout the year. In more temperate regions roots are stored during the winter months to serve as the source of “seed” for sprout production for the subsequent crop. Thus, production in temperate regions requires more extensive production inputs, such as storage facilities with the capacity to maintain a protected environment and additional land for the production of plants from roots.

Stems up to 30 cm long are used as the propagative organ. The stem pieces are often referred to as cuttings, slips, sprouts, or transplants. In tropical areas, where the crop is grown continuously throughout the year, stem cuttings are obtained directly from plants of the previous crop. In temperate regions the production cycle begins by planting roots adjacent to each other, covered with 2–5 cm of soil, in long, rectangular plots 60–80 cm wide, called beds. It is generally recommended that the roots be incubated for 2–4 weeks prior to bedding, at 25–30°C and 90% relative humidity, for maximum sprout production. Each root has the potential to produce many sprouts suitable for transplanting beginning 4–6 weeks after bedding. Roots continue to produce sprouts for several weeks under favorable conditions. The sprouting potential of individual roots is dependent on the genotype and root size as well as cultural conditions, including freedom from disease. Approximately 0.8–1.5 t of roots is required for producing enough transplants for each hectare of sweet potatoes. For comparison, the yield of U.S. No. 1 roots in the United States ranges from 10 to over 30 t/ha. This method of propagation requires a significant proportion of each crop for the production of the next crop.

Although considerable effort has been expended to improve the efficiency of propagation, practical alternatives have not yet been developed. For example, direct planting of root pieces has been little used, because of the tendency for proximal dominance of sprouting and the lack of uniform root production. Recent efforts to adapt rapid propagation techniques to commercial production have not yet become practical. Sweet potatoes are hexaploid and genetically complex. This results in an unacceptable level of phenotypic variability in true seed for commercial production. Propagation stock is most efficiently obtained in regions where the crop is produced the year round. Vines from one crop are immediately used to propagate the next crop, requiring no use of roots for subsequent crops. This may reduce the occurrence of diseases caused by rootborne pathogens, but foliar pathogens and viruses can be spread in this manner.

Sweet potato transplants are planted in rows 80–100 cm

apart, with 17–26 cm between plants. The rows may be prepared as level beds or raised beds, depending on local requirements. Raised beds are generally preferable to flat beds when it is necessary to improve drainage. Roots acquire or express the potential to become storage roots at the time of initiation. The reader is referred to an excellent treatment of the physiology of sweet potato yield by S. J. Kays. The roots develop to a harvestable size in 90–150 days, depending primarily on the genotype, with some variability attributed to environmental influences.

The harvesting of sweet potato roots has not yet been fully automated, particularly where they are to be stored and used for human consumption. In many areas of the world the roots are dug entirely by hand. In the United States they are dislodged from the soil by various implements, such as modified moldboard or disc plows and chain diggers. Hand labor is the predominant method of transporting the roots from the soil into containers for storage. The cultivars that now enjoy wide acceptance in the United States, and presumably cultivars in many other parts of the world, are susceptible to significant injury from handling during harvest. These injuries predispose the roots to many organisms that require wounds for infection. The most common among these are *Rhizopus* and *Fusarium* spp. Curing immediately following harvest and prior to storage reduces these diseases. Harvested roots are cured by promptly moving them into storage maintained at 30°C and 90% relative humidity for 5–7 days. This environment stimulates the rapid synthesis of a periderm layer on the root surface. The wounds are “healed” by the curing process and protected from invasion by pathogens. Roots should not be moved after placement in storage, to prevent further wounding of the periderm. They are held in storage at 16°C until marketed or used as a source of propagating material for the next cropping cycle. They should not be stored at temperatures less than 15°C.

In summary, sweet potato production can be divided into six major operations: the acquisition and production of vegetative propagation material, the planting of transplants, the growing of plants for root production, harvesting, curing, and storage. Disease management should be integrated into each of these operations.

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General Disease Control

Effective disease control for sweet potatoes is based on prevention. Most of the important diseases of sweet potatoes are caused by root pathogens or are capable of spreading systemically through the plant. Thus, once infection occurs and the disease can be detected, it is generally not possible to restore the health of an affected plant (or root). Further, the vegetative tissues used for propagation, whether roots or stems, provide a nearly perfect vehicle to perpetuate pathogens within the production cycle.

Selection of healthy propagating stock is the first consideration. This material should be carefully managed and a strict tolerance established for all pathogens. Planting stock should be produced and stored separately from commercial sweet potatoes. It should be inspected at each step of the production cycle to minimize the inadvertent introduction of pathogens. These inspections may also ensure cultivar purity or trueness to type. Diseased roots and plants should not be used. In areas with a high potential for root disease, vine cuttings and transplants should be removed from plant beds by cutting above the soil line rather than by pulling. In some instances it may also be necessary to apply fungicides as a prophylactic

measure. Severely infested fields should be avoided both for the production of propagation material and for commercial production. Losses due to diseases in commercial fields mildly or moderately infested with pathogens such as *Streptomyces ipomoea* and nematodes may be controlled by appropriate soil fumigants and the use of resistant cultivars.

Efforts to minimize losses caused by diseases during production may be useless if they are not accompanied by appropriate disease prevention measures at harvest. Careful handling and prompt curing are the primary disease prevention measures. Application of fungicides and bactericides at harvest is not as effective as proper curing to prevent diseases in storage. However, sweet potatoes are routinely treated with a fungicide prior to marketing to control *Rhizopus* spp., which can cause rapidly progressing decays. Where bacteria are a particular problem, calcium hypochlorite in the wash water can be effective. These efforts should be accompanied by generally accepted sanitation of farm equipment and storage bins and rooms. Adoption of these cultural practices effectively controls many sweet potato diseases and minimizes the need for pesticides.

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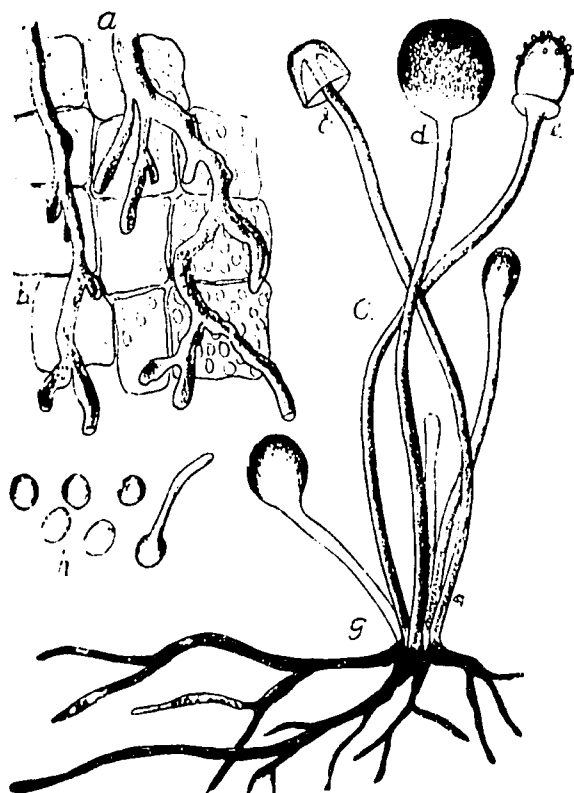


Fig. 1. Microscopic structures of *Rhizopus stolonifer*, the soft rot fungus, from infected sweet potato storage roots. (Reprinted, by permission, from Halsted, 1890)

Sweet Potato Diseases— Historical Perspective

Even though sweet potatoes were cultivated in many parts of the Americas and the Pacific region prior to the explorations by Europeans, the history of sweet potatoes in much of their present range is relatively recent. They are thought to have been introduced into Africa and most of Asia, where better than 90% of the world's sweet potatoes are grown today, after Columbus brought them to western Europe. Most of the information available concerning diseases of sweet potato and their control has been developed in the last 100 years.

Sweet potatoes are subject to diseases caused by fungi, bacteria, nematodes, viruses, actinomycetes, mycoplasmas, and abiotic factors. Although our current knowledge of sweet potato diseases results from contributions by many individuals, from a historical perspective some contributions are landmarks because they were innovative or monographic. The pioneering work of B. D. Halsted, published in 1890, clearly showed the relationship between certain fungi and the diseases they cause. This work included some of the earliest detailed drawings of plant-pathogenic fungi and their relationship to their hosts (Fig. 1). A period of intensive descriptive research aimed primarily at determining the etiology of sweet potato diseases led to the publication of a monograph by L. L. Harter and J. L. Weimer in 1929, which has served since then as the authoritative source of information on sweet potato diseases, especially those caused by fungi.

The suggestion that soil rot was caused by an actinomycete rather than a fungus was first made by J. F. Adams in 1929, and this etiology was firmly established by L. H. Person and W. J. Martin in 1940. The causal organism, *Streptomyces ipomoea*, is one of the two major actinomycete pathogens of plants. Bacterial soft rot also occurs on sweet potato, to a limited extent. However, bacterial diseases have not been of particular significance in the history of sweet potato culture. Sweet potato witches'-broom was studied in 1967 in the pioneering work of Y. Doi and coworkers, who first demonstrated the association of mycoplasmas with plant disease.

Viruses have been presumed for many years to cause several important diseases of sweet potato, but the first extensive characterization of a sweet potato virus was in research by J. W. Moyer and B. B. Cali on sweet potato feathery mottle virus in 1985. Although virus etiology is currently an area of considerable research activity, several sweet potato viruses have yet to be isolated and characterized. In this regard, the state of sweet potato research is many years behind that on the white potato, *Solanum tuberosum*.

Although only a small proportion of the world's sweet potato crop is grown in the so-called developed countries of the temperate zones, most of the published research on sweet potato diseases has originated in these countries. Consequently, many of the disease control procedures and disease-resistant cultivars have been developed in and are adapted to the temperate regions. Several factors contribute to this situation. Since sweet potatoes are not stored for long in the tropics and are propagated from vine cuttings without the need of sprouting roots, many of the most serious temperate-zone diseases are avoided. Secondly, there is very little reliable information on the geographic distribution of sweet potato diseases. More importantly, a greater financial commitment to research efforts has been made in the developed countries than elsewhere, and these research efforts have been organized across disciplinary lines for many years. For example, the National Sweet Potato Collaborators Group, formed in 1939 in the United States, promotes interdisciplinary research among plant pathologists, geneticists, breeders, entomologists, horticulturists, food scientists, physiologists, agricultural engineers, and others. Cooperative evaluations of breeding lines by members of this group have greatly facilitated the development of improved

cultivars, many with improved disease resistance. As a result of these efforts, yields of sweet potatoes in the United States increased markedly during the 1950s and 1960s, as higher-yielding cultivars with resistance to *Fusarium* wilt were brought into commercial production.

More recently, the development of active research programs on sweet potato at international research centers, such as the Asian Vegetable Research and Development Center, the International Institute of Tropical Agriculture, and the International Potato Center, and the publication of the proceedings of the first international symposium on sweet potato and *Sweet Potato Culture in China* have given increased impetus to international cooperation in sweet potato research.

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Part I. Infectious Diseases

Bacterial Diseases

Each of the four major diseases caused by prokaryotes—soil rot, bacterial stem and root rot, bacterial wilt, and a proliferation disease caused by a mycoplasma-like organism—has a restricted geographic distribution but can be very destructive.

Soil Rot (Pox)

Soil rot, also known as pox, *Streptomyces pox*, pit, or ground rot, is common in the major sweet potato production areas of the United States and Japan. It has not been reported in any other regions of the world. The disease may cause markedly lower yield and quality but does not appear to develop further in sweet potatoes in storage.

Symptoms

Symptoms on fleshy storage roots vary, depending on the time of initial infection and the cultivar. They may resemble those of circular spot (see Field and Storage Diseases). The most common symptom is the "scab" type of lesion on storage roots (Plate 1). These lesions are circular to somewhat irregular in outline and usually less than 5 mm deep. They are composed of dark brown to black, necrotic, corky tissue with frequent cracks radiating from the center. The lesions vary in diameter but are usually less than 3 cm. The remains of a necrotic feeder root may be found emerging from the center of a lesion. Lesions initiated early in the enlargement of a fleshy root (Fig. 2) often restrict enlargement at the point of infection, with the result that indentations form in the root or it acquires a dumbbell shape (Fig. 3A). In such cases the lesions may be obscured by the



Fig. 2. Soil rot (pox) lesions, produced by *Streptomyces ipomoea*, on fibrous feeder roots and fleshy storage roots at different stages of development. (Courtesy W. J. Martin)

development of adjacent healthy tissue. Occasionally, lesions appear to be partially healed, with islands of necrotic tissue separated by apparently healthy periderm (Fig. 4).

Soil rot of sweet potato causes what has been referred to as a rootlet rot, which can devastate the fibrous root system. The primary symptom on feeder roots is a dark black necrotic decay (Plate 2). The decay progresses slowly, but all tissues within the root are affected. During excavation of root systems for examination, most of the necrotic tissue commonly breaks off, leaving a necrotic stub at the end of the root. Lesions apparently can be initiated at any time during the growing season. Secondary effects from extensive rootlet rot include extreme stunting of vines (Fig. 5), lower yield, bronzing and chlorosis of foliage, premature flowering, and transient wilting.



Fig. 3. Storage roots with soil rot (pox) lesions, initiated by *Streptomyces ipomoea*. A, Lesions formed early in root development, which led to indentations and restrictions of the roots. B, Lesions with cracks radiating from their centers. (Courtesy C. A. Clark)

Lesions may also develop on portions of the vine in contact with the soil, but such lesions are not common or especially important.

Causal Organism

Streptomyces ipomoea (Person & W. J. Martin) Waksman & Henrici has been erroneously referred to in the literature as a fungus. It is a prokaryotic microorganism, belonging in the Actinomycetales. The initial determination of the etiology of soil rot proved difficult, because of the difficulty of isolating the causal organism from infected tissue; it grows slowly, and lesions are often invaded by other actinomycetes and bacteria. It is not isolated efficiently from lesions on storage roots, because these are generally contaminated with many secondary organisms. *S. ipomoea* is most easily isolated early in the growing season from near the margin of lesions on fibrous roots. Pieces of fibrous root cut about 1–2 mm above and below the transition between necrotic and healthy tissue are surface-sterilized in 0.525–1.05% sodium hypochlorite for 2–5 min. The tissue is then thoroughly ground with the end of a sterile glass rod in a sterile drop of water or buffered saline solution (pH 7) and either streaked on the surface of a suitable agar medium or suspended in cool molten agar and plated. The efficiency of isolation varies with the degree of contamination of the lesions by secondary organisms and with the medium. Several media can be employed, including the growth medium of Clark and Lawrence. Colonies producing the characteristic blue aerial mycelia can be transferred to slices of susceptible storage roots for presumptive identification based on their ability to produce necrotic lesions on the slices.

S. ipomoea is gram-positive, and the walls contain L-diaminopimelic acid, characteristic of streptomycetes. Spores are formed in short chains of the spirales type (Fig. 6A) from sporogenous hyphae produced on the aerial mycelium. The spore chains usually contain three to 10 spores in short spirals, hooks, or loops. On some media the spore chains may be compressed into balls (Fig. 6B), which superficially resemble sporangia produced in other genera of actinomycetes. Individual spores are oval to cylindrical, about $0.8\text{--}0.9 \times 0.9\text{--}1.8 \mu\text{m}$, with smooth walls. The sheath enveloping the spore chain is not readily broken, and thus the spores are not easily broken from the chains. On most media the substrate mycelia are white and give very young colonies a smooth, glistening surface. The aerial mycelia are initially white, becoming a distinctive blue and giving colonies a fuzzy appearance as they age. The mycelia are about $0.8\text{--}0.9 \mu\text{m}$ in diameter.



Fig. 4. Soil rot (pox) lesions, caused by the actinomycete *Streptomyces ipomoea*, on fleshy storage roots. Portions of some of the lesions have "healed," and healthy periderm has formed beneath the necrotic tissue. (Courtesy W. J. Martin)

S. ipomoea is aerobic, heterotrophic, and strongly oxidative. It grows relatively slowly in culture but is capable of growth on a wide variety of media. Typical aerial mycelia develop on a limited number of media, including yeast-malt agar, oatmeal agar, and salts-starch agar. Melanin pigments are not produced, but a diffusible, nonfluorescent, yellow to greenish yellow pigment is produced on certain media, especially potato-glycerine-peptone agar. The bacterium can use D-glucose, L-arabinose, D-xylose, D-inositol, D-mannitol, D-fructose, rhamnose, sucrose, and raffinose as carbon sources. It reduces nitrate to nitrite; it hydrolyzes gelatin and casein but does not hydrolyze cellulose or utilize it for growth. Although *S. ipomoea* reportedly does not produce the typical earthy odor of actinomycetes, some isolates may produce this characteristic aroma.

A method has been developed for rapid diagnosis of soil rot by an enzyme-linked immunosorbent assay that detects the pathogen in infected root tissue.

Disease Cycles and Pathogenesis

S. ipomoea is soilborne and persists in soil for many years in the absence of a sweet potato crop. The host range is probably limited to sweet potato and other members of the Convolvulaceae, including many common morning glory species, although there is little information concerning the role of the latter in the survival of the pathogen in the field. The pathogen may infect other plant species to a limited extent under artificial conditions. Considering the importance of the genus *Streptomyces* in soil, it is surprising that the behavior of plant-pathogenic *Streptomyces* spp. in soil has been so neglected. For example, soil rot is often severe in areas of fields where old barns once stood, and *S. ipomoea* is commonly grown in the laboratory on sterile horse manure, yet there is no information in the literature to indicate whether it is capable of saprophytically colonizing manure in natural field soils. There have been no reports to indicate what propagules of the pathogen survive in soil, but it is thought that other species of *Streptomyces* occur in soil primarily as dormant spores.

S. ipomoea is disseminated by the movement of soil (for example, on farming equipment or by erosion) and by infected or infested planting material. Dissemination may also occur through the digestive tract of livestock.

The pathogen does not directly penetrate the periderm of sound fleshy storage roots. It enters fibrous roots by directly penetrating the periclinal wall of epidermal cells or by penetrating between adjoining cells. It does not form appressoria or other specialized penetration structures. Instead, penetration occurs from short, lateral branches of hyphae that arise where surface hyphae are in contact with the root surface. Lesions on fleshy roots result from the growth of the pathogen from infected secondary fibrous roots into the fleshy roots.



Fig. 5. Vine growth of the susceptible cultivar Centennial (left) and the resistant cultivar Jasper (right) in a field with a heavy infestation of the soil rot pathogen, *Streptomyces ipomoea*, and soil pH near 7.0. (Courtesy C. A. Clark)



Fig. 6. Scanning electron micrographs of a spore chain (A; bar = 5 μ m) and a ball of spores (B; bar = 1 μ m) of *Streptomyces ipomoea* and an infected storage root parenchyma with hyphae of *S. ipomoea* within the tissue (C; bar = 5 μ m). (Courtesy C. A. Clark)

Whether the pathogen enters the fleshy roots through wounds such as those caused by insects is not known. Once within the host tissue, the pathogen ramifies the tissue mainly intracellularly. Hyphae penetrate cell walls directly by partial dissolution of the cell walls and by the growth of lateral branches (Fig. 6C). Limited numbers of spore chains have been observed within infected tissue late in the infection period.

Epidemiology

Three factors strongly influence the extent of disease development: soil pH, soil moisture, and crop rotation.

Soil pH is probably most important to disease development as well as geographic distribution of soil rot. The disease does not develop to any significant extent in soil with pH below 5.2, regardless of the amount of inoculum in the soil. As the pH is increased above 5.2, soil rot severity increases progressively. In areas where the natural soil is very acidic, the presence of *S. ipomoea* is often unrecognized until the pH is raised by liming. That soil rot has not been reported as a problem in the tropics may be due to the acidic nature of many tropical soils. There is one report of the occurrence of soil rot in Iowa in soil at pH 4.7.

The development of soil rot is favored by relatively dry soil. However, since lesions caused by *S. ipomoea* can develop throughout the growing season, the influence of dry soil on disease development depends in part on moisture distribution during the growing season. When dry conditions occur early in the growing season, yield is affected more than quality. When dry conditions occur after the initiation of storage roots, quality is lower, because of the increased incidence of lesions on the storage roots.

Soil rot is more severe if susceptible sweet potatoes are grown continuously in the same field. Unlike common scab of potato, caused by *S. scabies* (Thaxter) Waksman & Henrici, soil rot of sweet potato remains severe with continued monoculture rather than declining to intermediate levels. Rotations that exclude sweet potato for several years reduce soil rot severity to acceptable levels, but only in the first year in which sweet potato production is resumed. If it is also planted in the second year, soil rot may be severe.

Several species of morning glory that commonly occur as weeds have been infected with *S. ipomoea* experimentally, but their role in the survival of the pathogen in nature has not been determined.

Control

The most desirable means of controlling soil rot in the future will be the use of resistant cultivars. Several cultivars with resistance to the disease have been released in the United States since the introduction of the first such cultivar, Jasper. These cultivars have not yet been widely accepted, but they have been useful in problem areas. Their resistance is so great that a relatively normal crop can be produced even in the most severely infested fields. The resistance has been stable in the field, and races of the pathogen have not been found on resistant cultivars.

A combination of procedures is effective in reducing soil rot when resistant cultivars are not used or in augmenting intermediate levels of genetic resistance:

1. A low soil pH should be maintained. In many sweet-potato-growing areas the soil pH is low enough to be naturally unfavorable to the disease, and thus application of lime should be avoided. The use of sulfur to reduce soil pH effectively reduces soil rot, but this practice has not been widely adopted, because it is expensive and difficult to apply and requires time to adequately reduce soil pH.
2. Rotation with other crops reduces soil rot severity but does not entirely eliminate the pathogen.
3. Timely irrigation to reduce or eliminate periods with dry soil may also reduce the severity of the disease.
4. Soil fumigation (prior to planting) with fumigants containing chloropicrin is effective in reducing soil rot severity,

but in severe infestations losses may still occur.

Where there is good reason to believe that *S. ipomoea* has not yet been introduced into an area, care should be taken to prevent its introduction. Avoiding the movement of equipment, cattle, or any other potential carrier of soil from infested land into the area is important. Likewise, avoiding the use of mother roots or slips from fields infested with the pathogen is vital.

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Bacterial Stem and Root Rot

Economic losses due to bacterial stem and root rot were reported to occur for the first time in 1974 in Georgia, United States. The disease is also known as bacterial soft rot, bacterial wilt, bacterial wilt and root rot, or shell rot. To date it has only been reported in the United States, although the pathogen has a much wider geographic distribution.



Fig. 7. Bacterial root rot caused by *Erwinia chrysanthemi* on a storage root of the cultivar Jewel. (Courtesy C. A. Clark)

Symptoms

Brown to black, necrotic, water-soaked lesions on stems and petioles are initially similar in appearance to symptoms of Fusarium wilt (see Field and Storage Diseases). The lesions occasionally appear iridescent. Eventually, the stem may become watery and collapse, causing distal portions of the vine to wilt. Usually only one or two branches of the vine collapse, but occasionally the entire plant dies. Storage roots are affected in the field or, more commonly, in storage by a soft rot that turns diseased tissue light brown and watery (Plate 3 and Fig. 7). Lesions on storage roots often have a dark black margin and appear to be restricted. Some storage roots appear sound from the outside but are internally decayed (Fig. 8). Black streaks may appear in the vascular tissue of vines or storage roots. Mother roots often totally decay in plant beds, leaving only fibers and the periderm intact. The collapse of such roots often allows the covering soil to cave in, creating a crater on the surface of the bed.

Causal Organism

The pathogen, *Erwinia chrysanthemi* Burkholder, McFadden, & Dimock, is readily differentiated from other soft-rotting erwinias by its pattern of carbohydrate utilization, sensitivity to erythromycin, production of gas from glucose, higher optimum temperature for growth, and other characteristics. There is one report that *E. carotovora* subsp. *carotovora* (Jones) Bergey et al can infect sweet potato following artificial inoculation, but others have found that neither that subspecies nor *E. carotovora* subsp. *atroseptica* (van Hall) Dye decays storage roots.

E. chrysanthemi is widespread in warm climates. The species has an extensive host range, and several suggestions have been made for subspecific grouping based on host specialization and a few other phenotypic characteristics. Although isolates from sweet potato have not been used in extensive studies of host range, strains of the pathogen from sweet potato appear to be members of the largest, most diverse subgroup of *E. chrysanthemi*.

Disease Cycles and Epidemiology

Because bacterial stem and root rot has only recently been recognized and is restricted in distribution, relatively little research has been done on this disease. The pathogen invades the host primarily through wounds. It does not normally survive in soil except in association with crop debris or weed hosts or possibly in the rhizosphere of various plants. Sources of inoculum probably include infected mother roots, contaminated wash water, and harvesting equipment contaminated by infected roots and adhering infested soil.

Bacterial stem and root rot is a disease of warm, humid weather. Infections may remain latent at temperatures below 27°C, but symptoms may develop quickly with the advent of higher temperatures (30°C or higher).

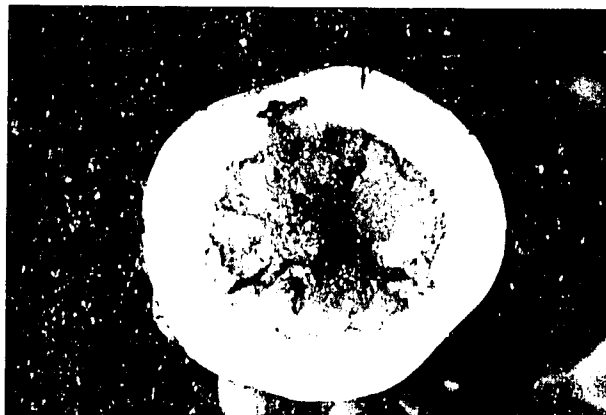


Fig. 8. Internal decay typical of that caused by *Erwinia chrysanthemi*, in a storage root with no external symptoms. (Courtesy G. Philley)

The use of dump tanks or flumes for handling storage roots after harvest favors the spread of *E. chrysanthemi* from infected roots to other roots during handling.

There are more questions than answers regarding the biology of this disease: symptomless infections of both storage roots and transplants may play a role in the transmission of *E. chrysanthemi*; the bacterium may survive and possibly multiply on the surface of mother roots in plant beds; it may infect both mother roots and slips pulled from them, through wounds incurred during pulling; lenticels might serve as an avenue of ingress for the pathogen; and the pathogen may be involved in the breakdown of sweet potatoes in waterlogged soils. Unfortunately, none of these suggestions has been adequately investigated.

Control

Several steps can be taken to reduce bacterial stem and root rot:

1. The incidence of wounding at any stage of production should be reduced.
2. Mother roots should be selected from fields free of the disease, and any roots that become infected during storage should be culled before bedding.
3. If bed covers or mulches are used, care should be taken to make provisions for gas exchange, because anaerobic conditions probably favor disease development.
4. Only vines cut above the soil surface should be used for transplanting.
5. The use of handling systems that do not involve immersion in water reduces the contamination of storage roots with the bacterium, but it also results in more wounding. Alternatively, if dump tanks or flumes are used in handling, the addition of chlorine to the water may be necessary to kill the bacterium.
6. Some cultivars, such as Centennial and Porto Rico, are less susceptible to *E. chrysanthemi* than others, and the disease rarely develops on them under field conditions. There are indications that the reactions of vines and storage roots may be inherited independently.

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Bacterial Wilt

Sweet potatoes are grown in many areas of the world where *Pseudomonas solanacearum* E. F. Sm. is an important pathogen of other crops. However, bacterial wilt of sweet potato, a disease caused by this bacterium, has been reported only in some regions of China, and only since the 1950s. In the regions of China where it does occur, it can be severe, causing yield reductions estimated from 30–40 to 70–80%. This disease, also known as blast, is the subject of quarantine regulations within China.

Symptoms

Symptoms of bacterial wilt may appear at any stage of sweet potato growth. Sprouts from diseased mother roots in plant production beds may wilt when they reach a height of about 15 cm. The base of the sprouts becomes water-soaked and then

turns yellowish brown to dark brown. Infected sprouts produce less latex, and the vascular bundles become brown from the base of the sprout progressively upward. Infected plants generally fail to develop roots and die within a few days after transplanting to the field. Healthy transplants may also become infected following transplanting to infested fields. The lower parts of their stems become water-soaked, turn yellowish brown, and develop vascular discoloration similar to that on sprouts. Once wounds incurred during transplanting have healed and roots have developed on plants in the field, wilting is not as great. However, lower leaves may yellow, vascular discoloration may develop in the stem, and the fibrous roots may become water-soaked and slough epidermis. Mildly infected storage roots may not have symptoms, although the fibrous roots emanating from them may become discolored and water-soaked. Yellowish brown, longitudinal streaks may develop in storage roots, and when infection is severe, grayish brown, water-soaked lesions may appear on the surface. Such roots usually decay entirely and develop a distinctive odor.

Causal Organism

At least four different bacteria have been named as the causal organism of bacterial wilt of sweet potato: *P. batatae* Cheng & Fan; *Xanthomonas batatas* Hwang, Chen, Hwang, Cheng, & Ho; *Bacillus kwangsinensis* Hwang, Chen, Hwang, Cheng, & Ho; and *P. solanacearum*. The recent characterization of isolates of the pathogen by L. Y. He and coworkers indicates that it is apparently a unique strain of *P. solanacearum* that has evolved in China. This strain is not pathogenic on tobacco; it causes typical symptoms in peanut and a number of other solanaceous crops, including tomato, potato, eggplant, and pepper. It reacts in cultural and physiological tests very similarly to strains of *P. solanacearum* from other parts of the world. The sweet potato strain has been assigned to pathogenicity group 2, race 1. Differential reactions have been observed on sweet potato cultivars, which suggest that there are two races within the strain.

Disease Cycles and Epidemiology

The bacterial wilt pathogen is both soilborne and carried in propagative material. Plants can thus become infected either by transmission from infected mother roots to sprouts or by the invasion of healthy transplants through wounds at transplanting. The bacterium can also be disseminated in the field by the movement of water or in composts incorporating infested plant debris.

Disease development is favored by warm, humid conditions (relative humidity greater than 85%). The pathogen grows at 20–40°C, with an optimum of 27–35°C. The disease is more serious in poorly drained clay loam soils than in well-drained sandy soils. Acid soils are more favorable for disease development than alkaline soils. Continuous cropping of sweet potato favors disease development, but flood cultivation (used in rice production) for 2 or 3 years reduces disease severity.

Control

Since the pathogen is restricted to certain areas within China, a quarantine has been established to prevent its dissemination to areas currently free of it. The quarantine prohibits the movement of storage roots and transplants from infested areas. It also requires isolation of livestock for several days before they are moved from a regulated area, to reduce the risk of dissemination of the pathogen in their manure.

A combination of other methods is recommended for areas where the disease is present:

1. *Resistance*. Although immunity has not been detected, several cultivars have resistance to at least one of the races of the pathogen.
2. *Sanitation*. Only disease-free storage roots should be used for propagation, and they should be bedded only in pathogen-free soil.
3. *Crop rotation*. The pathogen survives well in upland fields

for about 3 years and in flooded fields for about 1 year. Rotating with a flooded crop, such as paddy rice, is beneficial. Rotating with nonhosts, such as wheat, maize, or sorghum, is also of benefit.

4. *Avoidance.* Establishing or growing the crop during a cooler period of the year, where possible, reduces the effects of the disease on yield.

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Disease Caused by a Mycoplasmalike Organism

A disease typified by an excess proliferation of young shoots from leaf axils and dwarfing of subsequent growth was reported in the Ryukyu Islands in 1951. Since then similar diseases have been reported along the western rim of the Pacific Ocean from Korea to the Solomon Islands. The disease has been referred to by many common names: *ishuku-byo*, little leaf, and witches'-broom.

Symptoms

This disease has an exceptionally long incubation period in sweet potato, ranging from 50 to 186 days following graft transmission. The initial symptoms consist of vein-clearing followed by the development of new leaves that are distinctly smaller and more chlorotic than normal. The new growth is generally more erect, and there is a proliferation of axillary shoots, resulting in a bushy appearance (Plates 4 and 5). Individual leaves tend to roll up near the margins and to have a smooth outline. Latex is conspicuously absent from the roots and stems. Some infected plants do not survive until harvest or are at such a competitive disadvantage that they produce few harvestable roots. The root systems may be stunted and more branched than usual.

A wide range of incubation periods was observed in other *Ipomoea* spp. The disease is lethal in *I. ericolor*, with a relatively short incubation period of 35 to 49 days, making that species a candidate for an indexing host.

Causal Organism

A mycoplasmalike organism has been associated with this disease. Characteristic pleomorphic bodies ranging from 0.1 to

1.0 μ m in diameter, with a well-defined unit membrane, have been observed by several researchers in electron micrographs of sieve elements of infected plants. Both thermotherapy and chemotherapy cause remission of symptoms. Approximately 50% of the cuttings propagated from plants maintained at 38-39°C for up to 60 days remained symptomless for over 1 year. Cuttings treated with oxytetracycline also remained symptomless during a 2-year observation period. These characteristics, together with the inability to mechanically transmit the agent and the knowledge that it is vectored by a leafhopper, provide sufficient evidence to classify this disease among those caused by mycoplasmalike organisms.

Epidemiology and Control

The causal agent is spread by certain leafhoppers, such as *Orosius lotophagorum ryukyuensis* and *Nesophrosyne ryukyuensis*, and in infected planting material. In the Solomon Islands the disease causes the greatest losses in dry areas, which favor high leafhopper populations. However, it can be easily eradicated in the absence of its leafhopper vector. Disease control through control of the vector may be theoretically possible under well-defined circumstances, as the organism has a persistent relationship with the vector. This strategy may be of greatest value in maintaining healthy planting stock in the field.

The long incubation period increases the likelihood of spreading this disease when planting material is collected from sites where it is a problem. Additional caution is warranted since sweet potatoes are also a host of the leafhopper vector. Thus, it is possible to introduce eggs of the vector as well as the pathogen.

Sanitation has provided the best control to date. Planting material should be selected from a reliable source of healthy plants. When diseased plants are observed, they should be removed immediately and destroyed to reduce the opportunity for secondary spread. Wild *Ipomoea* spp. are susceptible to the organism and should be controlled wherever possible, especially near nursery stock used for planting material.

Although some progress has been made in developing resistant cultivars, they have not been widely accepted.

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Fungal Diseases

Sweet potatoes can be affected at different times during their production and marketing by many fungal diseases. Some pathogenic fungi interact with the crop through the entire cycle of vegetative propagation and affect it throughout its development. However, many fungal diseases are important only

during certain stages in the development of the crop. Accordingly, this section is divided into subsections concerning plant bed diseases; foliar diseases; field and storage diseases; storage rots; and flower, seed, and seedling diseases.

Plant Bed Diseases

Much of sweet potato production occurs in tropical regions of the world, where sweet potatoes are grown in the field all year around, and cuttings can be obtained from vines at any time. Problems specifically associated with the production of plants for transplanting are avoided under these conditions. In temperate and subtropical regions, however, sweet potatoes are propagated by bedding fleshy storage roots in the field, or occasionally in greenhouses or hotbeds, for the production of slips, or cuttings. The ability to efficiently produce healthy plants is vital to the success of sweet potato production in these areas.

Many of the diseases that occur on storage roots in the field or develop in storage may continue to develop in plant production beds. Diseases in this category are bacterial stem and root rot and soil rot (see Bacterial Diseases); black rot, foot rot, Fusarium root rot, Fusarium wilt, and scurf (see Field and Storage Diseases); dry rot, Java black rot, and Rhizopus soft rot (see Storage Rots); root knot (see Root-Knot Nematode); and virus diseases (see Virus Diseases). In addition, some pathogens, such as the reniform nematode (see Nematode Diseases), may be carried in soil adhering to sweet potatoes. Diseases that cause significant decay of fleshy storage roots may seriously limit plant production by rotting the mother roots, or seed roots, in plant beds. They may also reduce the vigor of sprouts and in a few instances kill some plants after the roots have sprouted, by progressing from the infected mother roots up into the sprouts. However, the plant production bed is most important in relation to these diseases because of the opportunity it provides for the transmission of pathogens from one crop to the succeeding crop. These pathogens cause greater losses in the field or in storage than in plant production beds and thus are discussed in greater detail in those sections of the compendium.

Two diseases of fungal etiology are most destructive in plant production beds, namely, sclerotial blight and Rhizoctonia stem canker. In addition, slime molds often make a dramatic appearance in plant beds and cause considerable concern,

although they are not thought to seriously affect plant production.

Sclerotial Blight

Sclerotial blight, also known as southern blight, southern stem rot, or bed rot, is one of two important sweet potato diseases caused by *Sclerotium rolfsii* Sacc. (The other, circular spot, is described in Field and Storage Diseases.) Sclerotial blight may be very destructive. It occurs almost exclusively in plant production beds and therefore is a problem only in temperate and subtropical production areas. The pathogen also has a restricted geographic distribution; it is not present in the cooler latitudes of the temperate zones.

Symptoms and Signs

Sclerotial blight usually does not appear in beds until after sprouts have emerged from the soil. The disease is usually first evident as a sudden wilting of the sprouts, soon followed by their death (Plate 6). The disease begins at isolated foci, which may expand at a rapid rate if conditions are favorable. Infections are initiated at or near the point where sprouts emerge from mother roots (Fig. 9), and necrotic lesions rapidly enlarge from this point up the sprout and down into the mother root. In some cases, infection is first noticed as plants are pulled. The plants break off very easily, and their bases are necrotic. In some cultivars with a degree of resistance to sclerotial blight, restricted circular lesions may be produced on the underground portions of the sprout, resembling circular spot lesions on fleshy storage roots. Disease development is arrested when plants from affected beds are transplanted to the field.

Diagnostic signs of the pathogen become evident soon after symptoms first appear. Typically, coarse, white mycelia of *S. rolfsii* cover the soil surface and, if the canopy is dense, may grow up and over sprouts. White mycelia also grow throughout the covering soil in the beds and over the surface of mother roots. Soon after the mycelia appear, sclerotia form in large numbers on the mycelial mat. The sclerotia are initially white, compact masses of hyphae, which gradually turn brown and take on the characteristic mustard seed appearance (Plate 7).

Causal Organism

The pathogen is commonly recognized as the anamorph, *S. rolfsii*. It is characterized by the production of rapidly growing, expansive fans of white hyphae. Aerial hyphae are abundant, and hyphae also frequently aggregate into rhizomorphic cords. At least two types of hyphae are produced by *S. rolfsii*. Coarse, straight, large-diameter hyphae with cells measuring $5-9 \times 150-250 \mu\text{m}$ have paired clamp connections at each septum (Fig. 10). Slender hyphae ($1.5-2.5 \mu\text{m}$ in diameter) are often observed entering the substrate; they grow irregularly and seldom have clamp connections.

After 4-7 days of mycelial growth, sclerotial initials develop from individual hyphae or groups of hyphae. The initials are spherical networks of interwoven hyphae, which appear white and fuzzy. As the sclerotium matures, at least three types of tissue are differentiated: the rind, or epidermis, two to four cell layers thick, on the outside of thick, flattened cells; a cortex of pseudoparenchymatous tissue, six to eight cell layers thick, made up of thin-walled, densely staining cells; and the medulla, at the center, composed of loosely arranged hyphae and intercellular air spaces. Mature sclerotia are smooth and spherical and range from light tan-brown to dark brown-black. They range from 0.5 to 5 mm in diameter, depending on the isolate and substrate, but are most commonly 1.0-1.5 mm in diameter.



Fig. 9. Hyphae of the sclerotial blight pathogen, *Sclerotium rolfsii*, on the surface of a sprouted mother root. The lower portions of the sprouts and the proximal end of the mother root are partially decayed by the pathogen. (Courtesy C. A. Clark)

The teleomorph, *Athelia rolfsii* (Curzi) Tu & Kimbrough, has rarely been observed in nature and has not been reported in association with sweet potato. However, it has been induced in culture, and isolates from sweet potato fruited. Basidia are produced on a hymenial layer that adheres loosely to the substrate. The hymenia, produced in patches 1–2 cm in diameter and 50–100 μm thick, are white to creamy white. Basidia, $5\text{--}6 \times 15\text{--}19 \mu\text{m}$, are produced in clusters and usually bear four basidiospores. The basidiospores are thin-walled and hyaline and measure about $3\text{--}5 \times 5.5\text{--}7 \mu\text{m}$.

Disease Cycles

S. rolfsii survives for years in soil and has an extremely broad host range, including both dicots and monocots. As a result, it is widely disseminated within the broad climatic zones in which it occurs. Sclerotia are the primary survival structures, and many factors affect their longevity.

No definitive study has been conducted to determine the exact nature of the relationship between sclerotial blight and circular spot. Since *S. rolfsii* also causes circular spot, and since roots with circular spot are frequently bedded for plant production, presumably such roots might serve as a source of inoculum for sclerotial blight. It is, however, extremely difficult to isolate *S. rolfsii* from circular spot lesions within only a few days after they are placed in storage. The possibility that roots or slips may be superficially contaminated by sclerotia has not been verified.

Epidemiology and Control

Temperature and moisture affect both the survival of *S. rolfsii* and pathogenesis. The fungus is restricted to warm climates, because of an inability of sclerotia to survive harsh winter temperatures. Where sclerotial blight occurs, it often does not appear until after the onset of warm, humid weather. Losses may be reduced by harvesting plants before infections occur or before the foci of infection enlarge. Many reports indicate that diseases caused by *S. rolfsii* are more severe under "wet" or under "dry" conditions; the only consistent observation is that either extreme of moisture stress apparently predisposes plants to infection by the pathogen.

One of the earliest observations of the incidence of sclerotial blight in sweet potato beds was that the disease becomes more severe when the canopy in the beds becomes dense and older leaves drop to the soil surface and collect there. This may influence disease development in several ways. Obviously, humidity is increased around the infection court, and disease development is favored by high humidity. Under some circumstances *S. rolfsii* seems to require a saprophytic food base before inciting infection, and it is possible that senescent leaves and other plant material serve as substrates. In other cases, sclerotia germinate in response to certain volatile chemicals, such as various alcohols, which commonly emanate from decaying plant tissues. Such volatiles may also stimulate subsequent mycelial growth. Senescent leaves, leaves injured by heat from plant bed covers, or decaying mother roots possibly serve as sources of stimulating volatiles in sweet potato beds.

Differences in incidence and severity of sclerotial blight have been observed among sweet potato selections and cultivars. The disease can, however, be relatively destructive on even the least susceptible selections.

An integrated program for the control of sclerotial blight combines the following steps:

1. *Site selection.* Locations that have not had sweet potatoes for at least 3 years should be chosen for plant production beds. It is also important to be sure that *S. rolfsii* has not been a problem on the rotational crops.

2. *Fungicidal treatment.* Dipping roots prior to bedding or, to a lesser extent, spraying roots laid out in beds with a protectant fungicide such as dichloronitroaniline can reduce the incidence of sclerotial blight. Because this fungicide is not systemic, it is thought to act primarily by protecting the points where sprouts emerge from mother roots, which are a favored

site for infection. The fungicide has limited effectiveness, however, when the fungus infects sprouts closer to the soil line.

3. *Removal of bed covers.* Beds are frequently covered with materials such as clear or black polyethylene sheeting to increase the soil temperature and thereby enhance sprouting. If such covers are left in place too long after the plants emerge, the foliage of emerging sprouts may be seriously injured by excess heat. The damaged foliage may then serve as a source of nutrients or stimulatory volatiles to increase sclerotial blight. Therefore, care should be taken to promptly remove bed covers as soon as the sprouts emerge from the soil.

4. *Cultivar selection.* The choice of a "less susceptible" cultivar may help reduce the incidence and severity of sclerotial blight.

5. *"Seed" selection.* No definite connection has been established between the incidence of circular spot on mother roots and sclerotial blight, but it seems prudent to avoid roots infected with circular spot for bedding.

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Fig. 10. Light micrograph of a hypha of *Sclerotium rolfsii*, showing the characteristic pattern of branching and a clamp connection at the septum. (Courtesy C. A. Clark)



Fig. 11. Unidentified slime mold growing on the surface of fleshy storage roots. (Courtesy W. J. Martin)

Taubenhaus, J. J. 1920. Recent studies on *Sclerotium rolfsii* Sacc. J. Agric. Res. 18:127-138.

Slime Molds

Several genera and species of slime molds occasionally grow on sweet potatoes, especially in plant production beds. The growth of these fungi is superficial and does not appear to damage the plants, except for detracting from their appearance and in some cases shading the foliage. However, their appearance usually attracts attention and concern from growers, because the molds may cover large areas of leaves, vines, and petioles. Slime molds appear on sweet potatoes in beds when the canopy has become dense and the weather has turned warm and humid.

Slime molds are generally first noticed when they have produced large masses of spores, which are usually brown. At this stage, workers reach into the beds for plants and find that their hands become covered with spores. Prior to that time, however, the growth may form a jellylike, slimy coating on plant surfaces, which may vary from white to yellow to purple, depending on the species involved.

Early reports identified *Fuligo violacea* Pers. and *Physarium plumbeum* Fries as species occurring on sweet potato. *Stemonitis* spp. are also common on sweet potatoes along the U.S. Gulf Coast (Plate 8).

Slime molds may also occur on the surface of roots in storage (Fig. 11).

Rhizoctonia Stem Canker

Rhizoctonia stem canker, also known as *Rhizoctonia* rot or *Rhizoctonia* sprout rot, has received little attention in the recent literature on sweet potato diseases. It was originally described on the stems of sweet potato plants in hotbeds throughout the United States. A small proportion of the plants developed an extensive decay of the underground portions, which resulted in secondary symptoms of foliar yellowing, stunting, and death or damping-off of sprouts in the plant beds. More commonly, sunken cankers appear on the stem, near the soil line, anywhere from the point of attachment to the mother potato to several centimeters above the soil line. These cankers are 2-6 mm in diameter and only occasionally girdle the stem.

Losses in plant beds are usually minimal, as relatively few plants are killed. In the field, plants that are initially healthy only rarely develop symptoms of the disease. Cankers on plants infected in plant beds commonly heal over, and the plants grow and yield as well as unaffected plants.

The stem canker pathogen, *Rhizoctonia solani* Kühn (teleomorph, *Thanatephorus cucumeris* (Frank) Donk), is a ubiquitous soilborne pathogen of many plants. The same fungus is also involved in the sweet potato rootlet rot complex (see Rootlet Rot). In addition, it is frequently isolated from apparently healthy stems following surface sterilization and is a common secondary invader of lesions induced by other pathogens. Under most circumstances, *R. solani* produces characteristic sterile mycelia, identified by their branching pattern. The hyphal branches arise at a distinctive angle and are constricted at the point of branching, and a new septum is laid down in the branch hypha near the branch point. There is one report of the formation of sclerotia of this fungus on infected tissue. The basidial stage has been observed occasionally in sweet potato beds, forming a white powdery growth on the surface of the soil and on the stems of sprouts.

The incidence of *Rhizoctonia* stem canker varies from year to year, and the factors responsible for this variation have not been determined. Although the disease is not presently considered important in commercial sweet potato production, it may cause subtle, undetected damage, since the underground portions of the plant are not closely examined unless a significant problem is observed.

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Harter, L. L. 1916. *Rhizoctonia* and *Sclerotium rolfsii* on sweet potatoes. Phytopathology 6:305-306.

Foliar Diseases

A variety of diseases that primarily affect foliage have been reported on sweet potatoes. Leaf and stem blights and leaf spots, for example, are not uncommon in the United States, and rust occurs occasionally. However, these foliar diseases have caused significant losses only in isolated instances and in the United States are not generally serious enough to warrant specific control measures. Of the fungi causing foliar diseases, only *Sphaceloma batatas*, which has not been reported in the continental United States, consistently causes economic losses.

Although many organisms have been associated with foliar diseases of sweet potato, details are presented only for diseases and causal organisms that have received the most attention. Organisms associated with foliar decays that are not discussed include *Botrytis cinerea* and *Choanephora cucurbitarum*.

There are several potential explanations for the apparent absence of significant foliar diseases on sweet potato. Sweet potatoes produce a dense canopy of leaves and stems under normal growing conditions. Defoliation experiments indicate

that the leaf area far exceeds that needed to produce an adequate yield. Thus, an abnormally high disease threshold may be necessary for yield to be adversely affected. Also, sweet potatoes contain high levels of phenolic compounds, phenol oxidases, latex, and phytoalexins, which contribute to disease resistance mechanisms. However, it is not known if these or other chemicals play a role in natural defense mechanisms in the foliage of sweet potatoes.

Selected Reference

Harter, L. L., and Weimer, J. L. 1929. A monographic study of sweet-potato diseases and their control. U.S. Dep. Agric., Tech. Bull. 99, 118 pp.

Alternaria Leaf Spot and Stem Blight

Several species of *Alternaria* have been associated with leaf spots on sweet potato that are characteristic of lesions caused by this genus. The disease is found on older leaves. The lesions are brown, with concentric rings and well-defined margins (Plate 9). The necrotic tissue may crack.

Alternaria spp. are easily isolated from the lesions; however, the disease is difficult to reproduce experimentally. In some instances the causal organism has been identified by species, but frequently it has been identified only by genus. The genus is most easily identified by its ellipsoidal conidia, which are light to olivaceous brown with many transverse and longitudinal septa and a beaklike structure on the distal end (Fig. 12). The exact shape and size of the conidia varies with the species. They are borne singly in some species and in chains in others.

Alternaria leaf spot is widespread. However, since it predominates on the oldest leaves, it has not been associated with reduced yields of storage roots. The most severe of the foliar diseases caused by *Alternaria* spp. is a newly recognized stem blight reported in Ethiopia. This disease is characterized

by small, gray to black, oval lesions on stems and petioles (Fig. 13). Humid weather is favorable for lesion enlargement, and the stems and petioles eventually become girdled. Dry weather results in a bleaching of the lesions. No information is currently available on control. However, differences in the reactions of several cultivars suggest that resistant cultivars may already be available.

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Cercospora Leaf Spot

Cercospora leaf spot of sweet potato, first described in Africa, is most prevalent in the warm and humid tropics and has been reported in Asia and South America as well as Africa. It is not generally a problem in the major sweet-potato-growing regions of the United States. Although the disease is widely distributed, it has only been a major problem in isolated situations.

Symptoms and Signs

Two similar types of lesions are associated with *Cercospora* leaf spot. Both are limited to approximately 8 mm in diameter. The lesions may be circular or somewhat angular and are sometimes delimited by veins. They can be uniformly brown or pale gray with light centers and dark borders (Plate 10). There is some evidence that the different symptoms may be due to the occurrence of more than one species of *Cercospora* on sweet potato.

Causal Organism

In the literature on sweet potato, the causal organism is usually referred to as *C. bataticola* Ciferri & Bruner. However, in the mycological literature it is referred to as *C. ipomoeae* Wint. This organism is characterized by colonies that grow on both adaxial and abaxial leaf surfaces and by hyaline conidia, with seven to 15 septa, borne on long, pale brown conidiophores. Although the common practice has been to name *Cercospora* spp. for the plant on which they were found, it is generally recognized that these species have broad host ranges.



Fig. 12. Ellipsoidal, septate conidia, typical of those produced by most *Alternaria* spp. (Courtesy C. A. Clark)



Fig. 13. Symptoms of stem blight associated with several *Alternaria* spp., with lesions on both the stem and the petioles. (Courtesy A. H. C. van Bruggen)

It has been suggested that this organism is probably synonymous with *C. apii* Fres.

C. timorensis Cooke has also been isolated from *Ipomoea* spp. in the tropics. It is distinct from the other *Cercospora* spp. in that it tends to grow primarily on the lower leaf surface, its conidia have fewer septations, and they are a darker, straw color. Lesions caused by *Cercospora* spp. are generally a solid brown.

Disease Cycle

Cercospora leaf spot is favored by hot, wet weather and is seldom observed during the dry season. However, the disease has been observed in dry regions of Peru. The organism colonizes leaf tissue and produces conidia on long conidiophores, which protrude from stomata. The conidia are disseminated by wind or splashing rain to other hosts. The organism may survive in plant debris or on alternate weed hosts.

Control

There are no reports of control measures specifically for *Cercospora* leaf spot of sweet potato. Presumably sanitation and removal of all material that might serve as a bridge between crops should help reduce the incidence of this disease. Resistant or tolerant cultivars may be available in regions where the pathogen persists from year to year.

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Phyllosticta Leaf Blight

Phyllosticta leaf blight is common in sweet potato fields in the United States and the Caribbean region. It is favored by humid conditions and is rare in arid climates. Although this is one of the most common leaf diseases of sweet potato, it is not considered to be a significant economic problem. No controls are warranted.

Symptoms and Signs

Leaf blight lesions are of irregular shape and may be up to 8 mm in diameter (Plate 11). The central portion of the lesion may vary from a light gray or tan to brown, and it usually has a dark brown or purple border. Pycnidia erupt through the lesion surface; these black fruiting bodies, usually clustered in the center of the lesion, are diagnostic signs of this disease.

Causal Organism

Phyllosticta batatas (Thuemen) Cooke is classified among the Sphaeropsidales. In earlier literature it was referred to as *Depazea batatas* and *P. bataticola*. The organism is disseminated by hyaline, unicellular conidia borne in pycnidia. The pycnidia are globose, 100-125 μ m in diameter, and are produced below the lesion surface (leaf epidermis). Chains of conidia are expelled through a beaklike structure, which protrudes through the surface of the lesion. The pathogen is thought to overwinter in decaying sweet potato leaves, as it is not known to infect any host other than sweet potato.

Septoria Leaf Spot

Septoria leaf spot is widely distributed in the United States, including Hawaii, and in the Caribbean region. Although it

probably occurs elsewhere, its distribution is not well documented. This may be attributed to the lack of a recognized impact on production.

Symptoms and Signs

This disease is similar to Phyllosticta leaf spot. Symptoms of Septoria leaf spot are small, white lesions (2-5 mm in diameter) with dark brown borders on the leaf surface. The lesions tend to be smaller than those caused by *Phyllosticta batatas*, as are the pycnidia, which may not be visible to the unaided eye. Usually only one or two pycnidia per lesion are formed.

Causal Organism

The causal organism, *Septoria bataticola* Taub., like *P. batatas*, is a member of the Sphaeropsidales. The pycnidia are dark, globose structures, 70-130 μ m in diameter. The short conidiophores produce filiform conidia, up to 60 μ m long, with three to seven septations. The conidia are disseminated by water, splashing, and insects.

Red Rust

Red rust is of minor importance. It is seldom observed on sweet potato, even on plants growing near other severely infected *Ipomoea* spp. The causal organism is disseminated as urediospores from these infected species to sweet potatoes growing nearby. The basidiospores infect pine, the alternate host. The aeciospores produced on pine serve to reinfect sweet potato.

The disease has been observed on many *Ipomoea* spp. throughout the western hemisphere. Reports suggest that it is most prevalent in the Caribbean region, but it also occurs in the United States as far north as New Jersey.

Causal Organism

Red rust in the Convolvulaceae is caused by *Coleosporium ipomoeae* (Schw.) Burrill, a heteroecious, macrocyclic rust belonging in the Uredinales. The uredial and telial stages are produced on *Convolvulus* hosts. The yellow-orange uredia may be up to 1 mm in diameter. The globose or ellipsoidal urediospores (16-28 μ m) are hyaline to pale yellow and have wartlike structures on their surface. The deep reddish orange telia are produced subepidermally around the uredia. The smooth, hyaline teliospores (60-140 \times 15-28 μ m) are clavate and have a thick wall and a foot cell at maturity. The basidiospores are only viable for short periods; they infect pine needles through stomata in the late summer or fall. The pycnial stage is produced on pine and is visible as a discrete spot up to 1 mm in diameter. Aecia, up to 2 mm high, 4 mm long, and 1 mm wide, are also borne on pine needles. The aeciospores are globose to ellipsoidal or cylindrical, up to 30 μ m in diameter and 45 μ m long. Like the urediospores, they have wartlike structures on either side.

In some instances red rust on sweet potato can be confused with white rust, caused by *Albugo*. They can be distinguished by inspecting for the presence of the telial stage. In addition, *Albugo* spores are smooth.

Control

Red rust of sweet potato has not been sufficiently severe to recommend controls, but fungicides may be necessary to protect ornamental pine species.

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Leaf and Stem Scab

Scab is the most severe of all the foliar diseases of sweet potato. It is common from Asia through the islands in the southern Pacific Ocean to Hawaii, and it has also been reported in Brazil. The disease is most severe where there is frequent fog, rain, or dew. Fields in areas where the pathogen has become firmly established may have to be abandoned because of the severity of the disease.

Symptoms and Signs

The first symptoms are small brown lesions on the veins of leaves. The lesions become corky in texture and result in a shrinkage of the veins, causing the leaves to curl (Plate 12). Stem lesions are slightly raised, with purple to brown centers and lighter brown margins (Plate 13). A scablike structure forms on stems as the lesions coalesce.

Field experiments have demonstrated losses greater than 50%. Lower yield is due primarily to the production of fewer edible roots, which indicates that the disease may interfere with the initiation of fleshy roots.

Causal Organism

Scab of sweet potato is caused by *Sphaceloma batatas* Saw. The ascigerous form of this organism, *Elsinoë batatas* (Saw.) Viégas & Jenkins, has also been observed in infected tissue. *S. batatas* grows slowly, and hyphae are sparse in diseased tissue. It is isolated from diseased tissues with some difficulty. Thin sections of lesions are excised from surface-sterilized, infected stems and leaves and incubated on onion agar. Hyphae grow into the agar in about 4 days. The fungus should be subcultured as soon as growth is apparent. The organism has also been isolated by baiting into drops of potato-dextrose agar on a glass slide. Pure cultures of the fungus can be grown on sweet potato agar, carrot agar, malt agar, yeast agar, or oatmeal agar. Maximum growth occurs at pH 6.0–8.5 and between 25 and 30°C with alternating periods of light and dark.

The host tissue is colonized by sparse mycelia that grow intercellularly and intracellularly. Colorless acervuli form beneath the epidermis. Microconidia and macroconidia are produced on conidiophores (6–8 µm). The microconidia are mostly spherical (2–3 µm in diameter), and the macroconidia tend to be ovoid (2.4–4.0 × 5.3–7.5 µm). Recent observations indicate that the microconidia enlarge in the presence of moisture to form the macroconidia, like those of *S. fawcettii* Jenkins.

The ascigerous stage, *E. batatas*, produces globose asci (10 × 15 µm) in a dark gray stroma below the epidermis. Each ascus contains four to six hyaline, septate ascospores (3 × 7 µm). Very little research has been conducted on *E. batatas*.

Control

Relatively little is known about controlling this serious disease. Research is currently under way to improve resistance in new cultivars. For example, numerous native and introduced cultivars have been evaluated at the University of the Philippines at Los Baños. Similar studies have been conducted at other research institutions.

A high degree of control of scab has been achieved with chlorothalonil. Mancozeb has been less effective.

In some areas infected plants are destroyed at harvest. Propagation material should be carefully inspected. Ideally, only material free of the disease should be used, and crop rotation should be practiced.

As wet, humid weather favors the disease, overhead irrigation should be avoided if possible.

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Fig. 14. Ruptured pustule produced by *Albugo ipomoeae-panduratae*, discharging sporangiospores on a leaf of *Ipomoea hederacea*. (Courtesy C. A. Clark)

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White Rust

White rust, or leaf mold, is a minor disease of sweet potatoes. It occurs in moist environments. The disease occurs on other *Ipomoea* spp. and can be severe in several of these hosts. Most improved cultivars have high levels of resistance; however, highly susceptible seedlings have been observed. Specific control recommendations are not currently necessary.

Symptoms and Signs

The first symptoms are distinct chlorotic lesions on the upper leaf surface (Plate 14) followed by destruction of the leaf surface. Infection may also spread to petioles and stems and result in distortion of these tissues. Signs of the disease are found on the lower leaf surface. White clusters of stalks bearing sporangia form beneath the lower epidermis. When the production of sporangia exceeds the capacity of the tissue, the epidermis ruptures, exposing the characteristic white pustules containing spores (Plate 15 and Fig. 14).

Causal Organism

White rust is caused by *Albugo ipomoeae-panduratae* (Schw.) Swingle, a member of the family Albuginaceae in the Peronosporales. Members of the Albuginaceae differ from the Peronosporaceae in that they bear sporangia in chains rather than singly or in clusters. The sporangia (12–22 × 10–21 µm) may produce biflagellate zoospores as well as germinate directly. Sporangial germination and subsequent infection are favored by cool nights. Infection of sweet potato leaves occurs either by the direct germination of sporangia or indirectly by zoospores. Members of this family typically colonize their hosts by intercellular hyphae, with individual cells being penetrated by a spherical haustorium.

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Field and Storage Diseases

Many of the most serious fungal diseases of sweet potato occur in the field. Most of these diseases also occur during all stages of the cycle of vegetative propagation of the crop.

Some of the pathogens that cause these diseases are transmitted primarily from infected seed roots to transplants and thus to plants in the field. Diseases of this type include black rot, foot rot, Fusarium root rot and stem canker, Fusarium wilt, and scurf.

In other cases, the pathogen is primarily soilborne, infecting plants in the field, but also causing a rot that continues to develop on fleshy storage roots in storage. Charcoal rot, surface rot, mottle necrosis, Phymatotrichum root rot, and violet root rot are in this group.

Rootlet rot, caused by several soilborne fungi, develops on fibrous roots in the field. In some cases it may lead to the development of mottle necrosis of storage roots, but otherwise it does not occur in storage.

Circular spot is caused by a soilborne pathogen and is common on storage roots in the field but does not develop further in storage under most circumstances.

Several genera of fungi reported to occur on sweet potato roots or vines in the field have not been studied extensively and are thus not covered in this compendium. These include *Sclerotinia*, *Macrosporium*, *Pyrenochaeta*, *Cylindrosporium*, *Schizophyllum*, *Hypochmus*, *Thielaviopsis*, and *Vacuulomyces*.

Black Rot

Historically, black rot has been one of the most significant diseases of sweet potato. Black rot is the most common name for this decay of fleshy roots and is also applied to the symptoms that occur on sprouts and vines in the field, which are also referred to as black shank or black root. The disease can cause severe losses in storage, in the plant bed, and in the field. The pathogen not only reduces the yield and quality of fleshy roots but also imparts a bitter taste that extends beyond the lesion. In 1929, L. L. Harter and J. L. Weimer stated that "black rot, while reported from other countries, is certainly much more destructive and prevalent in the United States." That situation has been almost entirely reversed since then. The disease is now relatively uncommon in the United States, because of the widespread implementation of an integrated control program, but is important elsewhere, especially in Southeast Asia, Oceania, and many countries in other regions.

Black rot of sweet potato has been extensively used as a model for the study of the physiology of disease and mechanisms of host specificity and is one of the most thoroughly characterized plant diseases in this regard.

Symptoms and Signs

Fleshy storage roots infected by the black rot pathogen develop a firm, black, dry rot befitting the name. Externally, the lesions appear gray to black when dry (Plate 16) and dark greenish black when moistened, and the periderm remains largely intact over the decayed cortical tissue. Lesions enlarge throughout storage but do not usually extend below the vascular ring unless they are invaded by secondary rotters (Plate 17). The lesions are usually centered on wounds, lenticels,

or lateral roots. The pathogen produces a fruity odor, both in infected tissue and in pure culture.

When infected sweet potatoes are bedded for plant production, symptoms develop on the plants in the bed and after transplanting to the field. Belowground portions of sprouts may rot from the point of attachment to the mother root or may have numerous cankers (Plate 18). The cankers are also dark black and sunken. Secondary symptoms in the bed vary, depending on the severity of infection, and may include stunting, wilting, yellowing, dropping of leaves, and in some cases the death of sprouts, much as with Fusarium wilt. When infected slips are transplanted to the field, the vine may survive and produce a crop even though the original stem may be rotted away below the soil line. These symptoms are similar to those produced by Fusarium stem canker. Daughter roots on infected vines often have lesions at harvest, which vary in size from specks to areas 1-5 cm in diameter.

The most diagnostic feature of black rot infection is the production of perithecia on the surface of infected tissue. The black perithecia have a distinctively long fimbriate beak, which may have a small, pink to cream-colored, viscous mass of ascospores at the tip.

Causal Organism

The pathogen is an ascomycete now known by the name of its teleomorph, *Ceratocystis fimbriata* Ell. & Halst. It was previously known by the synonym *Ceratostomella fimbriatum* (Ell. & Halst.) Elliott, *Ophiostoma fimbriatum* (Ell. & Halst.) Nannf., *Sphaeronema fimbriatum* (Ell. & Halst.) Sacc., and *Endoconidiophora fimbriata* (Ell. & Halst.) Davidson. The species contains many strains, which cause diseases of coffee, prune, almond, taro, and others in addition to sweet potato, but which are morphologically very similar. Although there is at least one report that strains of *C. fimbriata* from different hosts are interfertile, there is a high degree of host specificity. Strains from other hosts do not infect sweet potato, and vice versa, but sweet potato strains infect other Convolvulaceae, including many wild morning glory species.

C. fimbriata is normally homothallic, readily producing perithecia on diseased tissue and in culture on such common media as potato-dextrose agar. The perithecia are usually superficial or partially embedded in the substrate, black, and 140-220 μm in diameter, with long necks, up to 900 μm in length (Fig. 15A). Distinctive fimbriae are produced at the apex of the neck (Fig. 15B). The asci are globose, with a thin, fragile wall that disintegrates and releases ascospores within the perithecium. The ascospores, which are often referred to as hat-shaped, are exuded from the neck of the perithecium in a gelatinous matrix. They are elliptical (4.5-8 \times 2.5-5.5 μm), hyaline, nonseptate, and smooth, with a gelatinous sheath adhering to one side, giving the appearance of a brim (Fig. 15C).

Isolates of *C. fimbriata* from sweet potato produce two types of asexual spores. The cylindrical endoconidia (3-7 \times 7-37 μm) are produced abundantly on most substrates. They are hyaline to subhyaline, nonseptate, truncate, and single or in chains of up to 20 (Fig. 16A). They are produced within hyaline, phialidic conidiophores on aerial or subsurface hyphae; the conidiophores are 3-7 \times 35-172 μm , tapering toward the tip (Fig. 16B). The fungus also produces thick-walled conidia frequently known as chlamydospores, which are usually embedded in host



Fig. 15. Scanning electron micrographs of structures of the sweet potato black rot pathogen, *Ceratocystis fimbriata*. A, Perithecium (bar = 50 μ m). B, Fimbriae at the tip of the ostiole of a perithecium (bar = 50 μ m). C, Ascospores (bar = 0.5 μ m). (Courtesy Amanda Lawrence)

tissues. They are pale brown to olive brown and oval to globose (6–13 \times 9–18 μ m), have smooth to rough walls, and occur singly or in short chains, on simple or branched blastic conidiophores (Fig. 16C). Strains of *C. fimbriata* from other hosts also produce doliform endoconidia, but these have not been observed in isolates from sweet potato.

Disease Cycles

C. fimbriata is one of several pathogens of sweet potato that are chiefly transmitted in conjunction with the cycle of vegetative propagation of the host. Infected storage roots may escape detection at harvest or bedding, or new infections may occur during these processes, with the result that infected roots are then used for plant production. When such roots sprout in the beds, the fungus either colonizes the sprout directly from the mother root or infects the stem at other sites between the mother root and soil line. When slips are pulled for transplanting to the field, the infected underground portion of the stem carries the pathogen along with the plant.

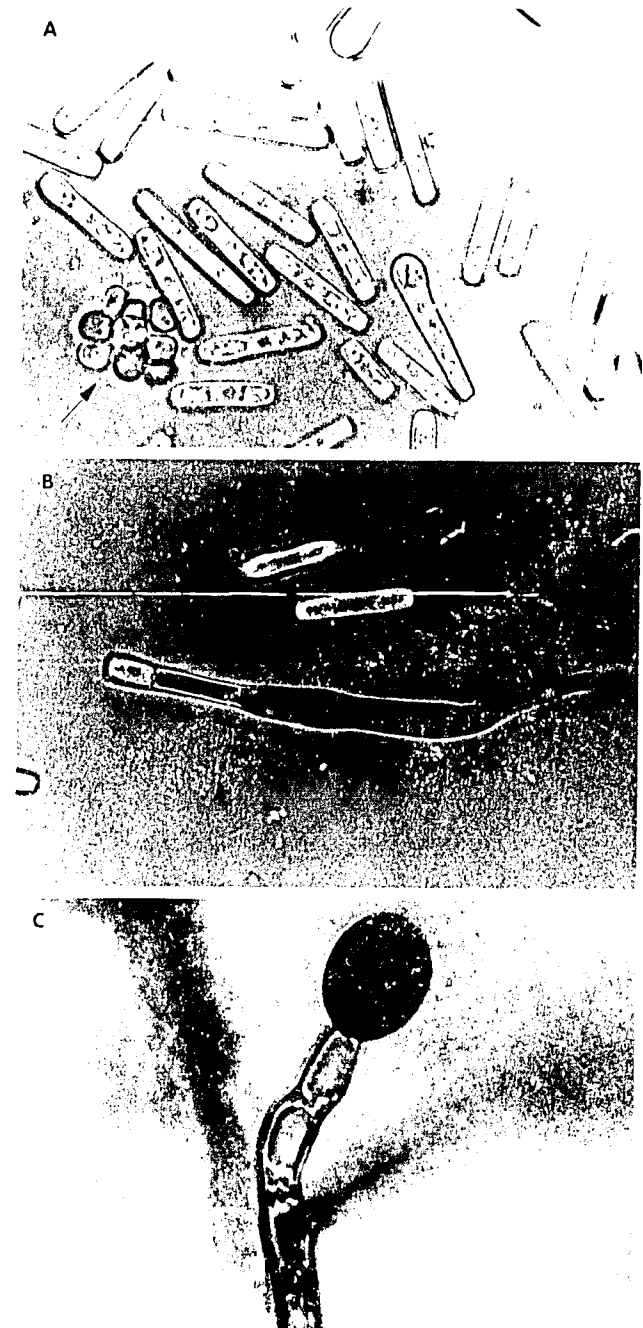


Fig. 16. Light micrographs of spores of *Ceratocystis fimbriata*. A, Endoconidia and ascospores (arrow). B, Phialide containing a maturing endoconidium. C, Chlamydospore. (Courtesy C. A. Clark)

C. fimbriata survives about 1–2 years in crop debris in the soil in the field. Presumably, the thick-walled chlamydospores serve as the primary survival structure in soil. There is some discrepancy in accounts of the ability of the pathogen to infect plants in the field without wounding. Some data suggest that soilborne inoculum is capable of infecting unwounded below-ground portions of the stem; however, there are also indications that the pathogen is not capable of invading unwounded fleshy storage roots. In Japan, *C. fimbriata* survives in sweet potato soils for several years, but the incidence of black rot is closely correlated with factors that cause wounds on fleshy roots in the field. The majority of such wounds were caused by field mice, which feed on healthy as well as infected parts of the roots. Chewing injury caused by crickets or wireworms or clawing injury caused by moles also contributed to increased black rot.

Black rot has been one of the most important diseases of sweet potato largely because of its ability to increase drastically during handling of the crop. The pathogen produces tremendous numbers of endoconidia and ascospores on infected tissue, which may contaminate the hands of workers, washing machines, storage containers, structures, etc. Whenever a crop is handled, there is considerable potential for contaminated items to serve as sources of inoculum for new infections. Tanks of water in which sweet potatoes are dipped during handling are potentially excellent reservoirs of inoculum. There is also evidence that, unlike most storage rots, black rot may spread in storage via insects, such as the sweet-potato weevil, *Cylas formicarius elegantulus* (Summers). When weevils were exposed first to infected storage roots and then to healthy storage roots, new infections developed around almost every puncture made by the weevils. They are likewise capable of transmitting the pathogen to vines in the field.

Epidemiology

Black rot can develop on sweet potatoes over a wide range of environmental conditions. Optimal temperatures for the infection of plants in the field are in the range of 23–27°C; infection can occur over the range of 10–34°C. In the plant production bed, infection is greater at 24°C than at 30 or 35°C. Roots presprouted at warm temperatures develop more black rot in beds than roots not presprouted. Infection increases with increasing soil moisture, up to about –0.1 to –0.2 bar, and then decreases; however, infection occurred both in saturated soil and in soil with barely sufficient moisture to support growth of the plants.

Control

The precipitous decline in the incidence of black rot in the United States has been due to the successful implementation of sanitation and chemical control. Because of the success of this approach, there has been little interest in developing resistant cultivars or of heat-treating propagating material, even though there is evidence to indicate that both could potentially contribute to disease control.

Successful control of black rot has relied on the following practices:

1. *Selection of seed roots.* Only sweet potatoes free of the disease should be selected for bedding for plant production.
2. *Fungicide treatment.* Seed roots should be treated with an effective fungicide, such as thiabendazole, to kill spores of the fungus contaminating the root surface. Available fungicides are not effective against established infections.
3. *Bedding site selection.* Sweet potatoes should not be bedded in sites that have been used to grow sweet potatoes within the last 3 years. In some cases fumigation of bedding soil with materials such as methyl bromide has been necessary as an extra precaution.
4. *Cutting of transplants.* It is critically important for transplants to be cut at least 2 cm above the soil line, to exclude infected underground portions of the stem.
5. *Rotation.* Sweet potatoes should not be planted in the same field more than once every third or fourth year.

6. *Handling.* It is not advisable to wash and package sweet potatoes from crops that show any signs of infection, as the incidence of the disease may increase drastically following this operation, and equipment may become contaminated. Some may be salvaged by processing in bulk.

7. *Curing.* Proper curing at 30–35°C and 85–90% relative humidity for 5–10 days immediately after harvest greatly reduces the incidence of infection through wounds incurred during harvesting.

8. *Decontamination.* Any equipment or materials that come in contact with an infected crop, e.g., washing machines, storage crates, or storage structures, should be decontaminated. Empty washing machines and crates can be sprayed with effective fungicides. Storage structures and crates can be fumigated with materials such as methyl bromide or chloropicrin to kill the fungus.

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Charcoal Rot

Charcoal rot routinely causes some loss of sweet potatoes in storage in tropical and subtropical areas, but seldom serious

losses. In early work it was mistaken for a stage of black rot (described above). It is also frequently confused with Java black rot (see Storage Rots). In most cases, the three diseases can be readily distinguished by symptoms and signs. Ironically, although the charcoal rot pathogen was previously referred to as *Sclerotium bataticola* because of its pathogenicity on sweet potato, there has been little research on the disease as it occurs on sweet potato.

Symptoms and Signs

Symptoms of charcoal rot develop primarily during storage. They begin as a reddish brown to brown, firm, moist rot, initially restricted to the cortex of the storage root. As the decay progresses, the pathogen crosses the vascular cambium, and the pith is progressively rotted. Two distinct zones become apparent within the infected tissue: the leading edge continues as a reddish brown decay, and a zone of black tissue develops behind the zone of active decay (Plate 19). The black tissue contains numerous microsclerotia of the pathogen, evenly dispersed throughout the tissue and visible with a microscope (Fig. 17). This zone of sclerotium production is often restricted to the cortex, even though the entire root may be rotted (Fig. 18). Although the lesions are sometimes restricted, charcoal rot usually consumes the entire root, which eventually dries, becoming hard and mummified. The periderm remains intact over the decayed root, and the external appearance of the lesions is not distinctive.

Affected roots are also frequently infected with *Fusarium* spp. In many cases it is not certain which fungus entered the root first. The symptoms produced are slightly different than those caused by either pathogen alone (see *Fusarium* Root Rot and Stem Canker and Surface Rot). Generally, the tissue infected with *Fusarium* spp. becomes lighter-colored and more ashen than tissue with typical charcoal rot, and the number of sclerotia produced by the charcoal rot pathogen is greatly reduced.

The charcoal rot pathogen does not normally attack other parts of the plant, but it has been reported on dead vines in the field (the vines had been killed by *Fusarium* wilt).

Causal Organism

The fungus that causes charcoal rot was once thought to be sterile and was named *Sclerotium bataticola* Taub. (syn. *Rhizoctonia bataticola* (Taub.) Butl.). It is now recognized, however, that the pathogen produces pycnidia, and it is now called *Macrophomina phaseolina* (Tassi) Goid. It commonly produces numerous sclerotia, sometimes referred to as microsclerotia, throughout infected tissue or in agar when grown on

common media, such as potato-dextrose agar. In tissue, the sclerotia are 100–1,000 μm in diameter, but in agar culture they are smaller, 50–300 μm in diameter. They are black, smooth, and hard, have a somewhat irregular surface, and are ellipsoidal to spherical. Their black exterior is made up of anastomosed black hyphae. Their interior is light to dark brown and consists of thick-walled hyphal cells.

Mycelia are sparse in culture, with few aerial hyphae produced in old cultures.

Pycnidia have not been reported on sweet potato, but they occur on other hosts and can be induced to form on certain natural media sterilized with chemicals or irradiation. Pycnidia are dark brown, immersed becoming erumpent, ostiolate, and 100–200 μm in diameter. The wall of the pycnidium is composed of several layers of pigmented, thick-walled cells. Short, obpyriform to cylindrical phialides (5–13 \times 4–6 μm) arise from a layer of hyaline cells. The conidia are hyaline and ellipsoidal to obovoid (14–30 \times 5–10 μm).

Disease Cycles

M. phaseolina is soilborne and survives as sclerotia in plant debris or free in the soil. It can be recovered from infected sweet potatoes long after they are first infected, but there has been no indication that the pathogen is carried in roots used for bedding. The pathogen has an extensive host range, including many crops commonly grown in rotation with sweet potatoes, such as soybean, cotton, sorghum, corn, and many other tropical crops. Inoculum in field soil may increase to a greater extent after some of these rotational crops than after sweet potato.

Like most storage rot pathogens of sweet potato, *M. phaseolina* requires a wound for entering a storage root. Thus, proper curing immediately after harvest effectively reduces the incidence of charcoal rot. Several reports indicate that pathogenesis by *M. phaseolina* is favored by relatively high temperatures, with an optimum of 29–31°C. The disease is more common in storage houses that are too warm or in crates located close to heaters. Likewise, infection is greater if harvested roots are scalded by sunlight before placement in storage. Heat stress predisposes plants in the field to the formation of lesions on stems, near the soil line, which may progress down into the storage roots.

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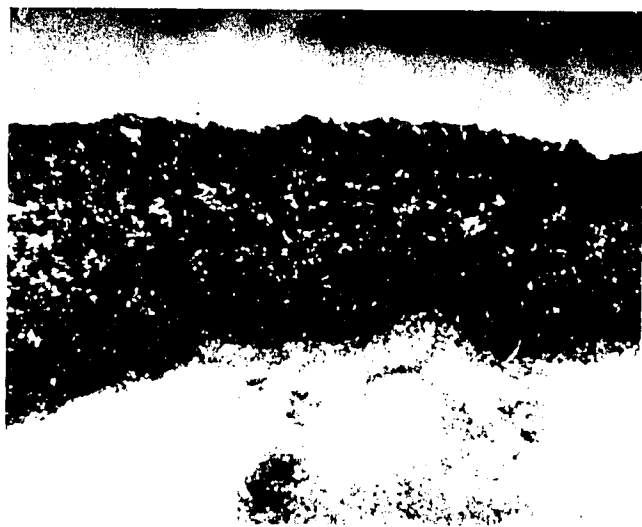


Fig. 17. Light micrograph of the cortex and a portion of the pith of a fleshy storage root infected with the charcoal rot pathogen, *Macrophomina phaseolina*. Sclerotia of the fungus can be seen embedded within the cortex. (Courtesy C. Pace)



Fig. 18. Cross section of a fleshy storage root with charcoal rot. The entire root is infected; sclerotia have formed primarily in the cortex. (Courtesy W. J. Martin)

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Circular Spot

Circular spot is the second of two diseases described in this compendium that are caused by *Sclerotium rolfsii* Sacc. Much of the information contained in the description of sclerotial blight (see Plant Bed Diseases) also applies to circular spot and is not repeated here.

Symptoms and Signs

Circular spot lesions occur only on the fleshy storage roots of sweet potato and have frequently been confused with lesions caused by the soil rot organism, *Streptomyces ipomoea* (see Soil Rot [Pox]). As the name implies, the lesions are circular, with sharply defined margins. The extent of lesion development varies considerably among sweet potato genotypes, but on the common commercial cultivars grown in the United States, the lesions usually average 1-2 cm in diameter (Plate 20). The surface of the lesion is brown, with a margin that may be slightly darker than the rest. The tissue within the lesion is yellowish brown to brown; it is soft and mushy when the root is first harvested, becoming mealy to leathery in texture as the lesion dries in storage. Generally, the lesions are shallow, 1-5 mm deep, and do not usually extend beyond the vascular cambium. However, on some particularly susceptible breeding lines, lesions have been observed to consume most of the root.

The lesions are initiated in the field and apparently develop only in the late stages of enlargement of the fleshy root. They

differ from soil rot lesions in that roots with circular spot are seldom, if ever, indented or constricted where the lesions form, nor do the lesions develop the healed appearance often associated with soil rot. If all else fails, circular spot can be distinguished by the bitter taste of affected roots, probably due to toxic compounds produced by the host in response to infection; soil rot lesions have an earthy flavor.

Circular spot lesions are not usually cracked at harvest, but they may crack from the center out as the lesions dry in storage. Typical patches of coarse white mycelia and brown sclerotia of *S. rolfsii* (see Plate 7) are occasionally observed on the soil surface in the field just prior to harvest, but it is more common to find severely affected roots with no sign of the pathogen evident. The pathogen apparently does not normally affect other parts of the plant.

Circular spot does not normally continue to develop in storage. In fact, once roots have been in storage for as little as a few days, isolation of the pathogen from lesions becomes difficult, if not impossible. Later in storage, an abscission layer develops around the lesions, which may be physically removed to expose a surface of wound periderm (Fig. 19). On rare occasions, when sweet potatoes are exposed to a saturated atmosphere or free moisture immediately after harvest, the lesions continue to enlarge, and large fans of the coarse, white mycelia of *S. rolfsii* grow out of them and cover the surface of surrounding roots (Fig. 20).

Disease Cycle

There has been little reported research on circular spot, despite its wide occurrence. The inoculum was reported to be soilborne. A connection between the use of plants from beds with sclerotial blight and the development of circular spot on daughter roots has not been firmly established. The incidence and severity of circular spot may vary significantly from field to field and from year to year. In some fields, the disease develops regularly to similar levels every year, while in other fields it may

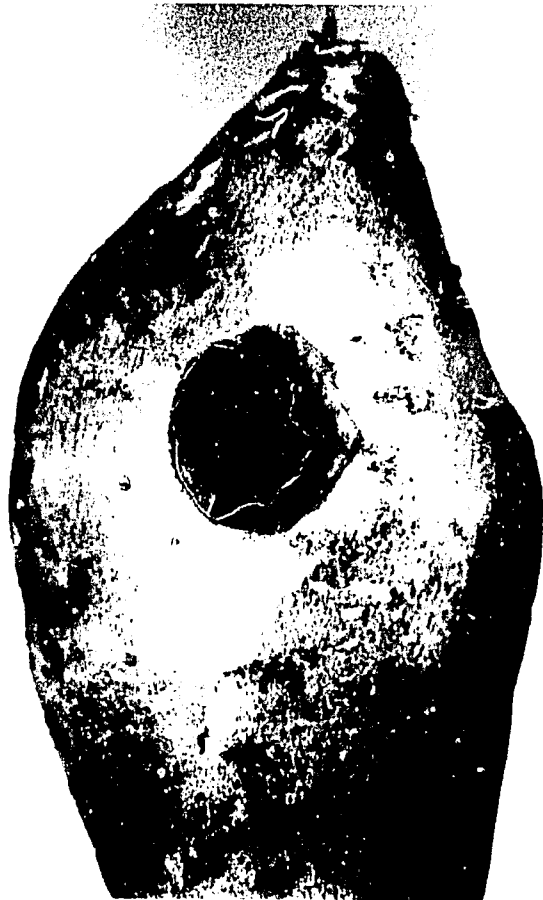


Fig. 19. Circular spot lesion on a fleshy storage root after several weeks in storage. At its margins, the lesion has begun to peel away from the root. (Courtesy C. A. Clark)



Fig. 20. Fleshy storage root infected with the circular spot pathogen, *Sclerotium rolfsii*. The root was placed in a moist chamber immediately after harvest to induce the fungus to grow out of the lesions. (Courtesy W. J. Martin)

never be a problem, and in still others it may vary from minor one year to severe the next. There is little available information to explain this variation.

Control

The primary method of control of circular spot is to avoid growing sweet potatoes in fields with a known history of problems with *S. rolfii*. Transplants should be free of the pathogen. Since the disease appears to be initiated late in the growing season, it may be possible to reduce the extent of disease development by harvesting the crop at the earliest opportunity.

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Foot Rot

Foot rot, also known as die-off, is of minor significance overall but may be very destructive in certain fields or storages when proper sanitation procedures are not followed.

Symptoms and Signs

Foot rot occurs in storage, plant reproduction beds, and the field. In beds, lower leaves of severely affected plants turn yellow, and the plants wilt and die. Plants affected less severely may have brown, necrotic lesions at or below the soil line or extending from the infected mother root up the stem. Such plants may escape detection and be planted in the field, where the disease continues to develop: lower leaves yellow, the vine wilts, and the plant may die (Plate 21) unless sufficient roots develop from nodes above the infection to sustain the plant. A dark brown canker may develop, girdling the vine at the soil line, and may rot off the lower portion of the vine. The canker may extend above and below the soil line and become covered with numerous small, black pycnidia at about the time the plant dies. The pathogen grows down the stem into the fleshy storage root and causes a slow decay of the proximal end of the root. This dark brown, dry, firm decay continues to develop slowly in storage but usually does not destroy the whole root. Frequently, pycnidia are revealed when the periderm is pulled off the surface of the lesion (Plate 22).

Causal Organism

Foot rot is caused by *Plenodomus destruens* Harter, a fungus closely related to and easily confused with *Phomopsis phaseoli*, the anamorph of the dry rot pathogen. *P. destruens* has no known ascigerous stage. It produces pycnidia readily, both in culture and on infected vines or roots. The nonstromatic pycnidia are round to irregular in shape (Fig. 21A). They have well-developed beaks and two walls (a well-developed, dark outer wall at the base and at the top and a hyaline layer within). Conidia within pycnidia produced in culture are one-celled, hyaline, oblong and sometimes slightly curved, biguttulate, and about $7-10 \times 3-4 \mu\text{m}$ (Fig. 21B). In addition, one-celled stylospores have been found in pycnidia produced on the host. They are hyaline, vary in length from 5 to $15 \mu\text{m}$, and are narrower than the conidia. The stylospores have not been observed to germinate.

Disease Cycle

Infection by the foot rot pathogen closely parallels the cycle of vegetative propagation of the sweet potato. *P. destruens* may overwinter in plant debris in the soil, but it does not persist much longer than over the winter in soil. Conidia may survive on the surface of fleshy roots in storage and germinate and infect wounded roots in storage or sprouts developing from the roots in beds. More commonly, the pathogen grows from

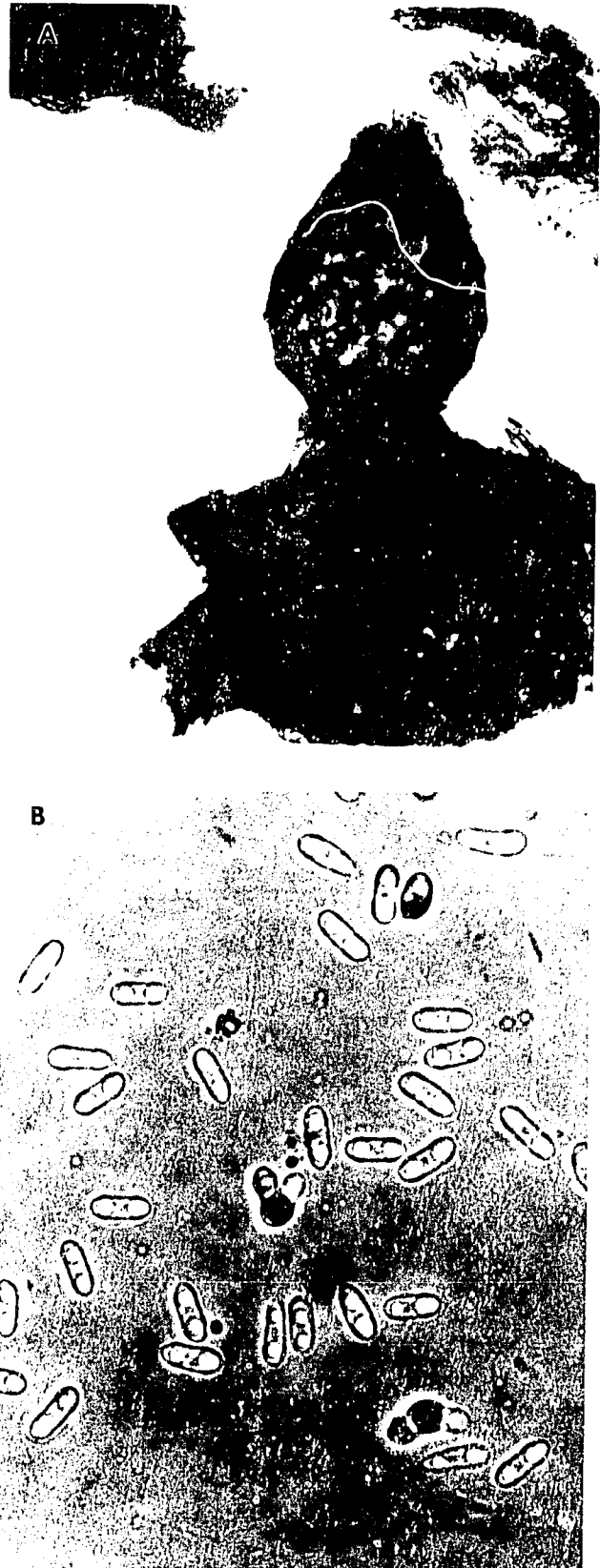


Fig. 21. Light micrographs of structures of the foot rot pathogen, *Plenodomus destruens*. A, Pycnidia. B, Biguttulate conidia. (A, courtesy G. W. Lawrence; B, courtesy C. A. Clark)

infected mother roots into daughter sprouts. When infected slips are transplanted to the field, the pathogen may grow from cankers on the vine down into storage roots. The only other known hosts of *P. destruens* are members of the Convolvulaceae.

Control

Foot rot is not generally a problem if proper sanitation procedures are routinely followed. Selection of disease-free roots for seed, the use of cut transplants rather than pulled slips, crop rotation in both plant beds and production fields, and the use of a fungicide such as thiabendazole on seed roots provide effective control of foot rot.

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Fusarium Root Rot and Stem Canker and Surface Rot

Sweet potato is affected by one or more decays caused by species of *Fusarium* that are cortical rotters requiring wounds for infection. These diseases are distinct from Fusarium wilt (described below), which is caused by a host-specific vascular pathogen. Surface rot is particularly common on storage roots, especially in areas where sweet potatoes are stored for any

length of time after harvest. Fusarium root rot is a serious disease of sweet potato in the southeastern United States. It has a root rot phase (also referred to as Fusarium root and stem canker, end rot, or Fusarium end rot) and a stem canker phase (once referred to as Fusarium foot rot).

Considerable research has been conducted concerning metabolites produced in *Fusarium*-infected storage roots by the interaction of host and pathogen. It has been suggested that these metabolites may be involved in reported poisonings of animals fed damaged sweet potatoes.

Symptoms and Signs

Surface rot is a postharvest disease of fleshy storage roots. It also occasionally occurs prior to harvest on roots that have been mechanically injured by growth cracks or by the feeding of nematodes, insects, or rodents. Surface rot lesions on fleshy roots are circular, light to dark brown, firm, and dry, and they usually do not extend into the root beyond the vascular ring (Plate 23). Externally, they usually appear solid brown (Fig. 22). When infected roots are stored for extended periods, the tissue within and around lesions dries and becomes shrunken, and the root eventually becomes hard and mummified.

Fusarium root rot may be very difficult to differentiate from surface rot on the basis of the external appearances of infected storage roots. In some cases, surface rot may be a preliminary stage of the more aggressive root rot. Root rot lesions on fleshy roots are circular and commonly exhibit concentric rings of light and dark brown (Fig. 23). They extend through the vascular ring, into the center of the root, and may eventually involve the entire root (Fig. 24); the root rot phase of the disease is differentiated from surface rot mainly by this characteristic.

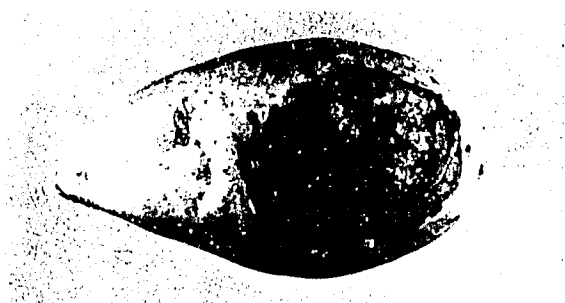


Fig. 22. Surface rot lesion, produced on a fleshy storage root by *Fusarium oxysporum*. (Courtesy G. W. Lawrence)



Fig. 23. Storage root with Fusarium root rot, caused by *Fusarium solani*. (Courtesy G. Philley)



Fig. 24. Cross section of a storage root with Fusarium root rot, containing characteristic lens-shaped cavities with mycelia of the pathogen on their inner surfaces. (Courtesy G. Philley)



Fig. 25. External and internal views of a storage root of the cultivar Jewel with Fusarium root rot, caused by *Fusarium solani*. Extramatrical growth of the pathogen is evident on the outer surface of the root, and lens-shaped cavities are visible within the lesion. (Courtesy C. A. Clark)

The tissue near the advancing margins of these lesions varies from orange to light brown, and the tissue nearer the origin is dark brown. The tissue at the lesion margins is more spongy and moist than either healthy tissue or older lesion tissue. Older lesion tissue is dry and pithy, and lens-shaped cavities occur in a radial pattern near the exterior. These cavities often have white hyphae growing on their inner surfaces (Fig. 24). As the lesions become large, the infected tissue becomes shrunk and dried. Eventually the roots become mummified. If the lesions develop rapidly or the roots are stored in a humid environment, extramatrical myphae and sporodochia may develop on the lesion surface, near the center or origin (Fig. 25). Frequently, root rot is initiated at either the distal or the proximal end of the fleshy storage root, a phase of the disease referred to as *Fusarium* end rot.

The stem canker phase of *Fusarium* root rot usually begins on sprouts from infected mother roots in plant beds, especially when several pullings of plants have been made. A dark brown to black decay begins at the base of the sprout, where it is connected to the mother root, and progresses up the stem (Plate 24). The necrosis often progresses slowly and may be restricted at nodes of the stem. The stem may also split for considerable distances above the necrotic tissue. When infected slips are transplanted to the field, the stem canker may continue to develop, stunt the vines, and reduce yields. Furthermore, the decay may spread from an infected vine into any its daughter roots.

Causal Organisms

Surface rot was originally attributed to *F. oxysporum* (Schlecht.) Snyd. & Hans., and for many years this organism was cited as the sole causal agent. It is morphologically indistinguishable from *F. oxysporum* f. sp. *batatas* (Wollenw.) Snyd. & Hans., the forma specialis that causes *Fusarium* wilt (described below), but is different in pathogenicity and other traits. Surface rot is also caused by some strains of *F. solani* (Sacc.) Mart. emend. Snyd. & Hans., the same fungus that causes *Fusarium* root rot. The name *F. solani* f. sp. *batatas* McClure was originally used in reference to the fungus that caused foot rot of sweet potato sprouts and vines. However, there is no indication that this organism is truly host-specific, and thus the designation as a forma specialis is questionable. The teleomorph *Hypomyces ipomoeae* (Hals.) Wollenw. is rare on infected stems in nature. In addition to these pathogens, *F. moniliforme* (Sheldon) Snyd. & Hans. is frequently isolated from healthy sweet potato tissue as well as tissue showing symptoms of the various *Fusarium* rots, but it is probably not a primary pathogen of storage roots.

The species of *Fusarium* pathogenic on sweet potato can be distinguished from each other primarily by the appearance and pigmentation of colonies and by the morphology of asexual spores. Both *F. oxysporum* and *F. solani* produce chlamydospores, microconidia, and macroconidia; *F. moniliforme* does not produce chlamydospores. The macroconidia of *F. oxysporum* are more than 4 μ m wide, whereas those of *F. solani* are less than 4 μ m wide (Fig. 26). Generally, the cortical-rotting *Fusarium* spp. that infect sweet potato are similar in their ecological and pathogenic characteristics and are therefore considered herein as a complex.

Disease Cycles

The species of *Fusarium* that cause disease of sweet potato are able to persist in soil for many years, primarily as chlamydospores. The strains of *F. oxysporum* and *F. solani* that cause surface rot and *Fusarium* root rot enter storage roots only through wounds that usually occur during harvesting. Surface rot or root rot then develops during storage, but the diseases do not spread to other roots in storage unless new wounds occur on them.

F. oxysporum does not spread from infected mother roots to sprouts produced on them, but *F. solani* can spread in this manner and thus causes stem canker. The pathogen is carried

with infected slips and continues to cause disease on below-ground portions of the stem and any fleshy daughter roots produced on infected plants. An important aspect of the stem canker phase of the disease is that the pathogen has been isolated from apparently sound tissue several centimeters above necrotic tissue of infected plants; thus, it is plausible that the pathogen can be transmitted on apparently healthy transplants cut from beds where the disease occurs.

Epidemiology

Little is known of the population dynamics of the sweet potato fusaria in soil. However, several factors affect the incidence of surface rot or root rot in storage and cause it to vary dramatically from year to year. Most act more by influencing the ability of the sweet potato to produce wound periderm than by directly affecting the activities of the pathogen. Surface rot is more prevalent during storage when sweet potatoes are mechanically damaged by any means; when the soil is wet and cold at harvest; when the soil is excessively dry prior to harvest, causing increased skinning of sweet potatoes as they are harvested; when sweet potatoes are exposed to high or low temperatures for extended periods after digging and prior to curing; or when conditions are favorable for desiccation of wounded tissue.

Very little is known about factors that influence the occurrence and severity of stem canker. The frequency of stem canker increases as additional pullings of slips are made from beds containing infected seed roots. Its effect on plant growth and yields of fleshy roots, like that of many other root and stem diseases, is aggravated by dry soil.

Control

Sanitation and proper handling of harvested storage roots are the most effective control procedures for both surface rot and *Fusarium* root rot.

There are indications that cultivars differ in their susceptibility to surface rot, but this has not been pursued, because the disease can be controlled by avoiding wounding during harvest and by immediate and proper curing of storage roots after harvest. Although surface rot usually is not a significant problem prior to harvest, the incidence of the disease in the field can be reduced by avoiding or controlling factors such as root-knot nematodes or insects that cause mechanical rupture of the periderm.

Control methods for surface rot also reduce *Fusarium* root rot on storage roots after harvest. Steps also must be taken to control the transmission of the pathogen to sprouts in plant beds. A combination of the following measures is necessary to achieve this end: careful selection of seed roots so that only disease-free roots are bedded for plant production; treatment of seed roots during the bedding process with an effective fungi-

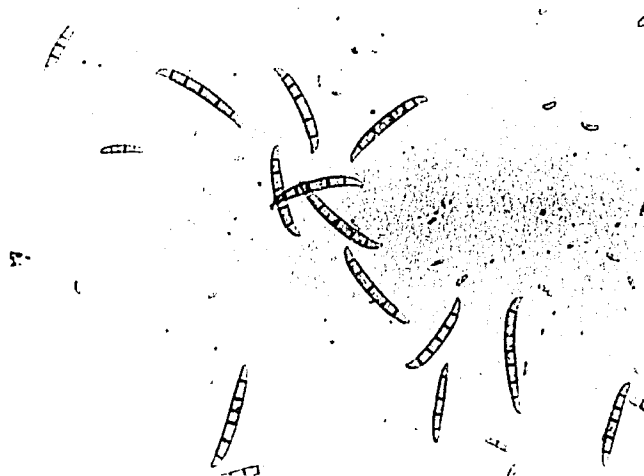


Fig. 26. Macroconidia of the *Fusarium* root rot pathogen, *Fusarium solani*. (Courtesy C. A. Clark)

cide, such as thiabendazole, to prevent new infections from developing on them; and bedding roots in sites free of the pathogen. To assure that stem canker is not transported to the field on infected transplants, it is important to use transplants cut above the soil line rather than pulled and to avoid taking more than two or three cuttings from a single bed.

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Fusarium Fibrous Root Rot

Recent reports from China have described a *Fusarium* disease that involves a decay of the fibrous feeder root system and sometimes also fleshy storage roots. When the feeder root system is extensively decayed, secondary symptoms may develop, including stunting of vines, leaf abscission, premature flowering, lower yield, or the death of entire plants. Storage roots that are produced are often small and cracked, and they may have long, brown-black lesions.

Several species of *Fusarium* have been associated with fibrous root rot in China. *F. solani* (Sacc.) Mart. f. sp. *batatas* McClure and *F. javanicum* Koord. are described as the most virulent.



Fig. 27. Sweet potato stem segments with the cortices removed. Healthy segments are on the left, and the segments on the right are from stems infected with the *Fusarium* wilt pathogen, *Fusarium oxysporum* f. sp. *batatas*, showing diagnostic vascular discoloration. (Courtesy C. A. Clark)

Fusarium fibrous root rot, as reported in China, apparently differs from surface rot and from *Fusarium* root rot and stem canker in that infection of fibrous roots commonly occurs in the field during the growing season without obvious prior injury to the root system. Isolates of *F. solani* from the United States that cause stem canker infect and cause limited necrosis on fibrous roots only if the roots are injured or originate from infected stem tissue. It will be of interest to determine whether the fibrous root rot that occurs in China differs from that in the United States because of differences in the strains of *F. solani* involved, the susceptibility of the genotypes of sweet potato grown in those regions, or environmental conditions prevailing in the field.

Resistant cultivars have been developed in China and provide the primary means of control of fibrous root rot.

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Fusarium Wilt

Fusarium wilt, also known commonly as stem rot or less commonly as vine wilt, blue stem, yellow blight, or yellows, was at one time the most serious disease of sweet potatoes in the United States and threatened the existence of the industry. However, it is a minor disease of little economic significance since the advent of resistant cultivars. In some countries the trend has been the opposite, with the introduction of susceptible cultivars increasing the incidence of the disease. *Fusarium* wilt has been reported in most areas where the crop is grown; it is more important in cooler temperate regions than in the tropics.

Symptoms and Signs

The most prominent symptoms of *Fusarium* wilt occur in the field just after the vines have started rapid growth. The usual sequence involves yellowing of older leaves followed by wilting, abscission of older leaves (Plate 25), and stunting of vine growth. In severe cases, the vine may turn tan to light brown, the pith within the stem may decay, and the plant may die. Discoloration of the vascular tissues of the stem or vine is an early and diagnostic symptom (Fig. 27) and may be accompanied by rupturing of the cortex of the stem; however, similar discoloration may be caused by *Erwinia chrysanthemi* (see Bacterial Stem and Root Rot). If the stem is cut in cross section or the cortex is peeled away, the xylem tissue is dark brown (or sometimes purple, if the stem segment is from below the soil line). Frequently, the symptoms are one-sided, with only a portion of the vascular ring discolored.

In some cases, infected plants survive and produce hills of sweet potatoes that appear healthy. However, if the vine and daughter roots are sliced longitudinally, the vascular tissue is revealed to be discolored continuously from the vine down into at least the proximal end of the daughter root. Infected storage roots store normally but, when bedded for plant production, give rise to sprouts that wilt either in the bed or when transplanted to the field. In such instances the sequence of symptoms is as described above.

The surface of vines killed by *Fusarium* wilt often has a pinkish extramatrical growth, consisting of numerous macroconidia and microconidia of the pathogen.

Causal Organism

Fusarium wilt of sweet potato is caused by *Fusarium oxysporum* Schlecht. f. sp. *batatas* (Wollenw.) Snyder & Hans.

This fungus is morphologically indistinguishable from *F. oxysporum*, which causes surface rot (see Fusarium Root Rot and Stem Canker and Surface Rot), but has a distinct pathogenicity. Much of the early research work on wilt referred to two species, *F. batatas* Wollenw. and *F. hyperoxysporum* Wollenw., which have since both been reduced to synonymy with *F. oxysporum* f. sp. *batatas*.

On common media, such as potato-dextrose agar, the pathogen resembles other forms of *F. oxysporum*. It produces a moderate amount of white aerial hyphae early in the development of a colony, but the colony has a slimy surface at later stages. A purple pigment, characteristic of the species, diffuses into the agar as the fungus grows.

Three spore forms are produced: microconidia, macroconidia, and chlamydospores. The hyaline microconidia ($2-3.5 \times 5-12 \mu\text{m}$) are produced from phialides. These spores collect in a drop of liquid at the end of a phialide, from which they are easily dispersed. They are predominantly aseptate; some are one-septate. The hyaline macroconidia ($3-4 \times 25-45 \mu\text{m}$) are readily produced and are also found in false heads on phialides. They are mostly three-septate; some have four or five septa (Fig. 28). Chlamydospores are produced either in the mycelia or by the differentiation of an internal cell of macroconidia. They are golden brown when mature, spherical, and $7-10 \mu\text{m}$ in diameter. The perfect stage is not known.

Disease Cycles

Infection occurs in several ways. The most common is infection from soilborne inoculum shortly after planting in the field. The fungus persists in the soil for many years as chlamydospores. It enters vascular wounds, such as those created by cutting the plants, or at fresh scars where leaves have detached from the stem. The pathogen apparently is not capable of entering through the callus that forms over wounds or through an intact root system. Symptoms generally are first apparent within 2-3 weeks after transplanting, but some new infections may appear throughout the growing season. The soil may also serve as a source of inoculum for the infection of storage roots at harvest or sprouts in plant production beds.

F. oxysporum f. sp. *batatas* can survive in the soil for extended periods in the absence of a suspect. Although it is often thought of as host-specific, it is capable of infecting other hosts, e.g., species of Convolvulaceae and two types of tobacco. Race 1 causes wilt in sweet potato and in burley tobacco, and race 2 causes wilt in sweet potato and in burley and flue-cured tobacco. There has been some suggestion that *F. oxysporum* f. sp. *nicotianae* (J. Johnson) Snyder & Hans., pathogenic on tobacco, is also capable of inducing wilt in sweet potato. There is a long list of plant species that become infected but show no symptoms, including several *Ipomoea* spp., which commonly occur as weeds, as well as cotton, tomato, okra, soybean, corn, cabbage, snap bean, Irish potato, watermelon, cowpea, sage, *Cassia* sp., and Mexican clover.

Fusarium wilt may also occur in association with the cycle of vegetative propagation of the sweet potato. Fleshy storage roots may become infected either as the result of growth of the fungus through the vascular system from infected vines or from soilborne inoculum when they are broken from the vine at harvest. In most cases the vascular system is discolored, but symptomless infections can occur in fleshy roots. Frequently, the fungus is present only near the proximal end of the fleshy root; since sweet potatoes display proximal dominance in sprouting, this is the area most suited for the subsequent infection of sprouts. When infected roots are bedded for sprout production, the pathogen grows through the vascular system from the mother root up into the sprout for a short distance and induces wilting, either of the sprouts in the bed prior to transplanting or of the vines in the field after transplanting.

The pathogen may be moved in infested soil, infected mother roots, infected slips or vine cuttings, or less frequently cattle, which can apparently pass viable propagules of the pathogen through their digestive tract after grazing on cull storage roots.

Epidemiology

Infection can occur whenever sufficient moisture is present to support sweet potatoes. However, symptoms are more severe and the effect on yield is greater when soil moisture is low.

The optimum temperature for Fusarium wilt is in the range of $28-30^\circ\text{C}$, but it can develop at the lowest temperatures that allow growth of the sweet potato. Although Fusarium wilt is prevalent in the cooler regions of sweet potato culture, it has been long recognized that high temperatures increase disease severity.

A significant factor governing the distribution of Fusarium wilt is the conduciveness of the soil. The disease does not occur to the same extent in certain soils as in others, apparently because of the presence of soil microorganisms that suppress disease development by reducing the rate of germination of chlamydospores and the extent of germ tube growth.

Control

The most effective and efficient means of control of Fusarium wilt is the use of resistant cultivars. Many cultivars with high levels of resistance have been released since the 1950s. The major source of resistance in the United States was the cultivar Tinian (PI 153655), discovered on Tinian Island during World War II. Although most such cultivars can be infected under artificially controlled conditions, even cultivars with intermediate levels of resistance are very useful under field conditions. This disease has become relatively unimportant in some countries, such as the United States, since the introduction of resistant cultivars, but care is needed to maintain resistance in cultivars released in the future. Although most resistance is derived from Tinian, no races of the pathogen have yet been recognized.

A combination of several practices can be employed to reduce Fusarium wilt in situations where resistant cultivars are not available:

1. *Rotation.* The pathogen population in the soil can be reduced but not eliminated by crop rotation.



Fig. 28. Light micrograph of microconidia and macroconidia of the Fusarium wilt pathogen, *Fusarium oxysporum* f. sp. *batatas*. (Courtesy C. A. Clark)

2. *Seed selection.* Seed roots should be taken only from fields where *Fusarium* wilt has not been a problem.

3. *Cutting of transplants.* Since the fungus is mostly limited to the basal portions of sprouts from infected mother roots, transmission of the pathogen can be reduced by cutting vines 2–5 cm above the soil rather than pulling slips. However, the pathogen has been found in vine cuttings, even in cultivars with a degree of resistance, and thus other control procedures should also be used.

4. *Fungicide treatment.* Dipping seed roots or vine cuttings (or both) in fungicides such as benzimidazole or thiabendazole reduces transmission of the pathogen through the vegetative propagation cycle.

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Fig. 29. Symptoms of mottle necrosis on storage roots. (Courtesy J. C. Bouwkamp)

Mottle Necrosis

Mottle necrosis, once referred to as white rot, has been observed in the United States since the early 1900s. Although it occasionally causes significant losses, it has not generally been considered important. Historically, it has been more important at temperate latitudes, such as in New Jersey, but losses have also occurred in subtropical areas, such as Louisiana.

Symptoms and Signs

Mottle necrosis refers to a decay of fleshy storage roots that develops in the field but does not cause further losses in storage. Three distinct patterns of symptoms are caused by the fungi responsible for this disease.

A marbled decay, which may resemble *Fusarium* root rot (described above), occurs at soil temperatures above 18–22°C. The external symptoms consist of slightly sunken brown spots, which vary from small and circular to relatively large spots that coalesce into an irregular outline (Fig. 29). When affected roots are sliced, islands and channels of dry, crumbly, dark gray to brown, necrotic tissue are seen in an interconnected, labyrinthine pattern (Plate 26). The extent of internal decay is unrelated to the extent of the development of surface lesions; small surface lesions may connect with extensive decay within the root, and vice versa.

The cheesy type of mottle necrosis may be confused with bacterial soft rot (see Bacterial Stem and Root Rot) or *Rhizopus* soft rot (see Storage Rots). It occurs more frequently when soil temperatures are below 18–22°C. Affected tissue has the consistency of soft cheese; it is the same color as healthy flesh or slightly grayer. Lesions are large and continuous, as opposed to the labyrinthine decay associated with the marbled type.

The band type of mottle necrosis is indistinguishable on the basis of symptoms from the ring rot reportedly caused by species of *Rhizopus* that also cause soft rot (see *Rhizopus* Soft Rot). Lesions are usually not sunken, unlike those associated with the marbled and cheesy types, but are shallow and usually restricted to the cortex by the vascular ring. The tissue is firm and chocolate brown. The lesions tend to extend more laterally than longitudinally from the point of origin, forming a band or ring.

Mycelia of the pathogen are distinctive and abundant within infected tissue, even in older areas of lesions, from which the fungus can no longer be isolated. The hyphae are nonseptate, and protoplasmic streaming is rapid in young hyphae. If infected tissue is cut and incubated in a humid environment, the cut surface is quickly covered with white hyphal growth, in 1–2 days. Sporangia and oogonia are not normally found in infected tissue.

Causal Organisms

Koch's postulate for mottle necrosis have been completed with at least two species of *Pythium*: *P. ultimum* Trow and *P. sclerotieichum* Drechsler. In addition, *P. aphanidermatum* (Edson) Fitzpatrick, isolated from other plants, can cause this disease in artificially inoculated sweet potato. Unidentified *Pythium* spp. that also cause mottle necrosis, distinct from *P. ultimum* and *P. sclerotieichum*, have been isolated. *Phytophthora* spp. have also been isolated, but pathogenicity tests have yielded conflicting results.

The frequency of isolation of the two primary pathogens, *P. ultimum* and *P. sclerotieichum*, from infected sweet potatoes varies from year to year, but generally *P. ultimum* is isolated more frequently. It also causes damping-off, rootlet rot, and soft rots on a wide variety of plants. *P. sclerotieichum* has only been found in nature in sweet potatoes with mottle necrosis or rootlets associated with infected fleshy roots.

Hyphae of *P. ultimum* are up to 11 µm wide and do not usually bear sporangia. The smooth-walled oogonia are globose, intercalary or sometimes terminal, and 20–24 µm in

diameter. Antheridia are mostly monoclinal and sacklike. Oospores are single, aplerotic, globose, and 17–20 μm in diameter.

P. scleroteichum produces hyphae 2.5–7 μm in diameter, bearing a moderate number of terminal, clavate appressoria 5–12 μm in diameter. Production of the sexual stage can be induced on special media with some difficulty. The smooth-walled oogonia are terminal or sometimes intercalary, thick-walled, and 21–27 μm in diameter. Antheridia are monoclinal, clavate, and crook-necked. Oospores are yellow, largely filling the oogonium, and 15–22 μm in diameter.

Disease Cycles and Epidemiology

Lesions of mottle necrosis are often centered at the point where feeder roots originate from the fleshy root, and these rootlets are usually infected by the causal fungi (see Rootlet Rot). It has therefore been presumed that the disease is initiated by growth of the pathogens from infected rootlets into the fleshy root. Within the fleshy root, the fungi rapidly ramify the tissue, both intercellularly and intracellularly. Cells of the host often become filled with hyphae, and cell walls are penetrated at right angles following the production of appressoria and narrowly constricted penetration pegs. The fungi can be readily isolated from water-soaked tissue at the margins of lesions by techniques appropriate to the isolation of *Pythium* spp. Mycelia in older portions of the lesions die rapidly, however, and the pathogens are not readily isolated from these regions or from fleshy roots that have been in storage for more than about 4 weeks.

Pythiaceae fungi are ubiquitous in soil, and rootlet rot (described below) frequently begins in plant production beds and continues in the field. Rootlet rot is much more common than mottle necrosis. Mottle necrosis is strongly influenced by environmental factors, e.g., soil texture, moisture, and temperature. The disease tends to be more prevalent in fields with an intermediate soil texture than in fields with particularly fine or coarse soils. The most serious losses to mottle necrosis have occurred in sweet potatoes harvested late in the growing season, especially after cool rainy periods. Although optimal temperatures for growth of the pathogens in culture are 25–32°C, the highest frequency of successful inoculation of fleshy roots is at lower temperatures (12–15°C). The marbled type of mottle necrosis occurs above 18–22°C, and the cheesy type below 18–22°C. The disease does not develop at the temperatures used for proper curing of sweet potatoes, and it does not usually develop after harvest.

Control

Since mottle necrosis is not normally encountered in commercial production of sweet potatoes, control programs are not well developed. Avoidance is the most effective means of control and can be accomplished by harvesting sweet potatoes early, before the occurrence of the cool, wet conditions that

favor the disease, and by crop rotation, to avoid a buildup of inoculum. In addition, it has been noted that cultivars differ in the extent of natural development of mottle necrosis in the field, even though all cultivars tested can be readily infected by artificial inoculation. The Jersey type of cultivars appear to be particularly susceptible.

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Phymatotrichum Root Rot

Phymatotrichum root rot, also known as cotton root rot or Texas root rot, is not important on sweet potato. The pathogen, *Phymatotrichum omnivorum* (Shear) Duggar, is restricted in its distribution to the southwestern United States and is destructive on many dicotyledenous crops and weeds in poorly drained black soils. The disease significantly affected sweet potato production many decades ago, but these soils are no longer considered suitable for commercial production, and the disease is now limited to home gardens.

Phymatotrichum root rot appears in circular spots in a field. Plants in the center of the spots often fail to produce any storage roots, and those near the margins of the spots have roots of various sizes and in various stages of decay. The decay is firm and dry and is initially associated with the surface of the root (Plate 27). It often progresses from one end of the root, and the interior of the root is not affected until the rot is quite advanced. The outside of the root may be dark brown to black at first, but as the decay progresses, the distinctive mycelia of the pathogen appear as white protuberances and brown to buff strands. The brown fungal strands run parallel to one another and branch at right angles. In many cases vines may grow normally, even though the root system is severely diseased. In some cases, however, the pathogen has been observed to progress 15–30 cm above the soil line within the vine, producing a brown discoloration. By the time infected sweet potatoes are dug, the entire belowground system of the plant may be seriously decomposed, and the fleshy storage roots may be hard, dry, buff to brown, and thoroughly mummified.

Rootlet Rot

The term *rootlet rot* refers to a disease complex involving several pathogens. With the exception of the soil rot pathogen, *Streptomyces ipomoea*, most of them are cortical-rotting fungi that are normally considered weak pathogens. The rootlet rot phase of soil rot is generally more dramatic than that caused by the other pathogens and is discussed in greater detail in the section on bacterial diseases (see Soil Rot [Pox]). Fusarium fibrous root rot (described above) is also more destructive.

Symptoms of rootlet rot are indistinct and may exhibit subtle variations depending on the primary pathogen involved. Secondary organisms also probably affect the appearance of affected rootlets. Generally, the decay on affected rootlets progresses several centimeters from their ends (Fig. 30). They may become light brown to dark black, and the cortex may be sloughed, leaving a relatively undamaged stele. *S. ipomoea* generally rots the stele as well as the cortex.

Pythium ultimum Trow., the same organism involved in the mottle necrosis complex (see Mottle Necrosis), was most

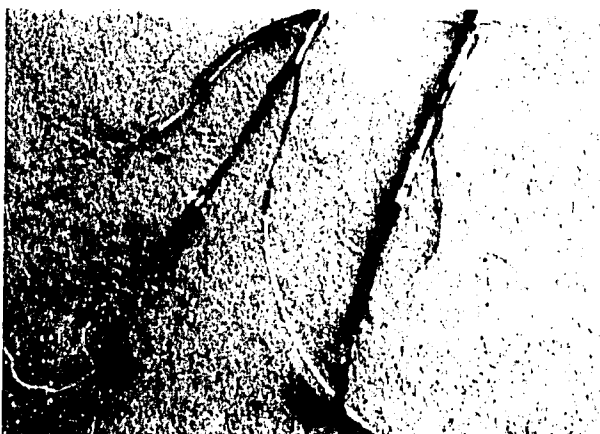


Fig. 30. Rootlet rot. (Courtesy J. C. Bouwkamp)

frequently associated with rootlet rot in the early studies of the disease. In addition, *Rhizoctonia solani* Kühn, which also causes a stem canker on sweet potato (see Rhizoctonia Stem Canker), has been isolated from many of the same lesions from which *P. ultimum* was isolated. *Fusarium solani* (Sacc.) Mart. emend. Snyder & Hansen, the causal organism of Fusarium root rot and stem canker (described above), has also been observed to cause lesions on fibrous feeder roots of sweet potato, especially when the roots are on stems with lesions of Fusarium stem canker.

Rootlet rot was first observed in plant production beds. It has also been found on fibrous roots at all stages of plant growth and in widely scattered geographic locations. Pythium rootlet rot is more common in plant beds where the soil is sandy, where sweet potatoes have been bedded repeatedly, and where the soil is wet. Although Pythium rootlet rot was found on all cultivars observed, apparent differences in the severity of the disease were noted.

After virus diseases, diseases of the fibrous root systems of sweet potato have been the most neglected. In light of recent findings with many other crops, to which root-nibbling pathogens such as *Pythium* spp. and others have been found to be more damaging than previously thought, a reevaluation of the importance of rootlet rot of sweet potato seems to be in order.

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Scurf

Scurf, once known as soilstain, is common in the United States, many of the islands of the Pacific, and Japan. Damage from the disease is primarily cosmetic. It may also result in increased shrinkage due to water loss in storage.

Symptoms and Signs

Symptoms of scurf are restricted to the underground portions of the plant. Dark brown to black spots develop on fleshy storage roots during the growing season (Plate 28). The color of the lesions is dependent in part on the color of the periderm, with copper-skinned sweet potatoes usually having brown lesions, and red-skinned cultivars having almost black lesions. The spots slowly enlarge and may coalesce, until the entire surface of the root is discolored. Characteristically, the scurf pathogen and symptoms it induces are restricted to the



Fig. 31. Hill of sweet potatoes infected with scurf, caused by *Monilochaetes infuscans*. The infection spread from the stem of the original slip to the proximal ends of the daughter storage roots. (Courtesy W. J. Martin)

periderm and do not directly affect the cortex or underlying tissues of the storage root. Affected tissue can be readily scraped off the root. Severely affected sweet potatoes may also develop small cracks as they shrink in storage.

When infected sweet potatoes are used for plant production, the enlarging lesions spread to the sprouts, causing a superficial discoloration similar to that produced on the mother root (Plate 29). Likewise, when infected slips are transplanted to the field, the pathogen spreads to the daughter roots (Fig. 31). For this reason, lesions on storage roots are more frequent on the proximal ends of the roots. Similar superficial symptoms are also produced on fibrous roots.

Causal Organism

The pathogen, *Monilochaetes infuscans* Ell. & Halst. ex Harter, is a very slow-growing fungus with no known sexual stage. It can be induced to sporulate on infected tissue by incubation in a moist chamber. The sporulation is usually sparse, and magnification is required for viewing the simple dematiaceous conidiophore with an unbranched chain of hyaline conidia borne on it (Fig. 32).

The fungus can be difficult to isolate from infected tissue. It has been isolated by the following procedure. Infected storage roots are first thoroughly washed in tap water. An infected region of the root is swabbed with cheesecloth saturated with 0.525% sodium hypochlorite for 1-2 min. A small piece (1 mm²) of infected periderm is removed from near the margin of a lesion and aseptically transferred to a solidified drop of suitable agar on a sterile microscope slide. This is incubated in a petri dish in a moist chamber and examined daily with a microscope until the fungus has grown out sufficiently to be transferred. *M. infuscans* grows on most common media, forming dark gray to black colonies that are raised and domelike. Sporulation is greater on green pea agar than on other media on which it has been grown. White mutant sectors develop frequently in cultures of the fungus and are apparently avirulent.

Hyphae of the fungus are initially hyaline and become dematiaceous. They ramify within the periderm of the host and penetrate cell walls following the production of a simple appressorium and a penetration peg (Fig. 33). Conidiophores are usually unbranched, brown, and connected to the host tissue by an enlarged, bulbous basal cell (Fig. 32A). They are usually 40-300 μ m long and occasionally contain two or three enlarged, bulbous cells in a chain. The conidia are initially hyaline and may turn light brown with age. They are 4-7 \times 12-20 μ m, nonseptate, and formed in chains of 10-25 spores, which may curl (Fig. 32B). The chains of conidia readily break up on wetting.

Disease Cycles and Epidemiology

Most scurf infections result from the use of infected propagating material. The pathogen also survives in soil for 1-2 years, depending on the soil type. Disease severity is greater and persistence of the pathogen longer in fine-textured soils and when the organic matter content of the soil is high. The use of animal manures in fields or beds may increase the incidence of scurf, and the fungus grows well and sporulates on extracts from manure.

M. infuscans is capable of infecting several *Ipomoea* spp. It is not known to infect any other crop.

The pathogen invades only the periderm of the fleshy storage root.

Scurf lesions continue to enlarge when sweet potatoes are placed in storage, and new lesions may appear if a high relative humidity is maintained. The optimum temperature for disease development is approximately 24°C, but scurf can develop to a lesser extent over a wide range of temperatures. Disease development is greatest when soil moisture is neither excessive nor limiting to plant growth.

Control

Although variation in the relative susceptibility of sweet potato breeding lines and cultivars has been observed, the levels

of resistance are not sufficient for commercial control. Resistance is related in part to the length of the slender attachment root between the stem and the storage root proper. The longer it is, the lower the probability of the spreading of the fungus from the slip to the daughter root.

Hot-water treatments of seed roots or slips (or both) has potential for reducing scurf but is not widely practiced, because

it is difficult to control the water temperature, and because alternative control procedures are effective but not as potentially damaging as hot water.

Sanitation procedures help to keep sweet potatoes free of scurf, and a combination of sanitation and fungicide applications is recommended. The following combination of practices should be followed in controlling the disease:

1. Only scurf-free sweet potatoes should be used as seed roots.
2. Seed roots should be treated with an effective fungicide, such as thiabendazole.
3. Seed roots should be bedded only in soil free of the pathogen.
4. Transplants should be cut at least 2 cm above the soil line.
5. When the disease has been a problem, it may be advisable to dip slips in a fungicide such as thiabendazole or dichloronitroaniline, as well as cutting the transplants above the soil line. Fungicide treatment of infected roots is only partially effective.
6. Sweet potatoes should be rotated with other crops in a 3- to 4-year rotation.

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Fig. 32. Light micrographs of structures of the scurf fungus, *Monilochaetes infuscans*. A, Cross section of an infected storage root, with two conidiophores arising from the periderm. B, Conidiophore and chain of conidia produced in culture. (Courtesy G. W. Lawrence)



Fig. 33. Scanning electron micrograph showing penetration of the cell wall or a periderm cell by the scurf fungus, *Monilochaetes infuscans*, which has formed an appressorium above the cell wall. (Courtesy G. W. Lawrence)

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Violet Root Rot

Most reports of violet root rot of sweet potato have come from Southeast Asia. It can cause serious losses of sweet potato in some regions of China, Japan, Korea, and Taiwan in years that favor disease development.

Symptoms and Signs

Violet root rot develops only in the field. Decay of fibrous roots progresses from the tips and eventually may destroy the entire root system. Foliage of severely infected plants becomes chlorotic, and older leaves may senesce and abscise prematurely. Storage roots usually decay from the distal end toward the proximal end. Decaying roots have an alcohol odor, similar to that associated with souring (see Disorders Caused by Adverse Environment in the Field) or *Rhizopus* soft rot (see Storage Rots). The entire root is usually rotted but remains held together by a thick mantle of hyphae over its outer surface. This coarse network of hyphae is at first white, gradually becomes pink to brown, and eventually develops the characteristic violet for which the disease is named, as the velvetlike fruiting bodies of the pathogen are formed. Sclerotia are frequently formed on the base of the stem, near the soil line, and purplish brown mycelial mats, about 300–500 cm in diameter, may develop on the soil surface surrounding diseased plants. Hymenia of the fungus were not observed on these mats, although they are produced on many surfaces, including stones, dead branches, soil masses, and even concrete.

Causal Organism

Violet root rot is caused by the basidiomycete *Helicobasidium mompa* Tanaka. This pathogen has an extensive host range, including sugar beet, soybean, potato, cotton, peanut, and woody plants such as mulberry, tea, apple, plum, and grape. The fungus produces sclerotia that are flat to spherical but generally irregular in shape, violet on the outside to white inside, with dimensions of 0.5–3.0 × 1–5 mm. Basidia are hyaline, cylindrical to clavate, commonly curved, three-septate, and 6–7 × 25–40 μm, with up to four sterigmata, bearing basidiospores. The basidiospores are ovoid to kidney-shaped, tapering to the point of origin from the sterigma, and 6.0–6.4 × 16–19 μm. Basidiospores and hyphal cells arising from germinating sclerotia are binucleate. Germ tubes from the basidiospores are multinucleate.

Disease Cycles and Epidemiology

H. mompa is soilborne and survives at least 4 years in soil, primarily as sclerotia, but also as mycelia, rootlike mycelial strands, or sclerotia attached to debris from infected crops. Basidiospores are not important in the field. Infected mother roots are not thought to be an important source of inoculum. The pathogen can be dispersed in irrigation water and in infested manure but is dispersed most commonly by the

movement of infested soil, e.g., by erosion associated with heavy rain or by adhering to transplants from infested nurseries.

The pathogen grows on the outside of the sweet potato during the early part of the growing season and forms infection cushions in the middle lamella of the periderm. Later in the season, hyphae penetrate from the infection cushions into the parenchyma of the storage root and cause the root to decay. *H. mompa* produces pectolytic enzymes. A heat-stable, nonvolatile toxic principle has also been associated with the fungus. Resistant cultivars of sweet potato restrict the development of mycelia of the fungus by forming a wound cork layer.

Violet root rot is most severe in wet growing seasons, especially if high-moisture conditions occur late in the season. Basidiospores of the pathogen germinate over the range of 17–35°C; mycelial growth occurs from 8 to 35°C. The optimum for both processes is 27°C. Disease severity is also favored by continuous sweet potato culture, poor soil drainage, nutrient deficiency, and low soil pH.

Control

A combination of several cultural practices reduces the development of violet root rot:

1. **Sanitation.** When infected plants are found, the infected material should be collected and either sterilized with a suitable chemical or destroyed by burning and deep burial. Care should be taken to avoid introducing infected plants or infested crop debris or soil into fields used for sweet potato production. Infested fields should not be used for sweet potato beds or for nurseries for other vegetable or fruit tree crops, since the pathogen may be disseminated with the transplants. Caution should also be exercised to reduce the spread of the pathogen in infested manure, on implements, on animals, or on workers or their clothing.
2. **Crop rotation.** Violet root rot is increased by continuous culture of sweet potato and also by rotation with mulberry or tea. Rotation for more than 3 years with graminaceous crops reduces the disease. Rotation with rice is especially effective in poorly drained infested fields.
3. **Soil management.** Practices that increase soil fertility and improve soil structure, e.g., the application of organic fertilizers, not only improve the water relations of sweet potato, by increasing aeration in the root zone, but also reduce the incidence of violet root rot.
4. **Cultivar selection.** The use of early-maturing cultivars enables growers to harvest their crop before the disease becomes severe.

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Storage Rots

Postharvest diseases of sweet potato are referred to as storage rots even though they may include diseases that also occur in the field or on sweet potatoes in transit or in the market. Storage rots are most important in the temperate zone, where sweet potatoes are stored for several months before being sold for consumption or bedded for plant production. In more tropical regions, where they are harvested all year, little consideration is given to storage rots, because few people attempt to store sweet potatoes for long periods of time. Several rapidly developing postharvest diseases prevent successful storage in the tropics. The development of a practical system for storing sweet potatoes in the tropics might help to provide a stable supply, facilitate trade, and make land available for other uses.

The vast majority of sweet potato storage rots are caused by fungi, the exceptions being bacterial soft rot (see Bacterial Stem and Root Rot) and brown ring (see Nematode Diseases). Some of the most important storage rots are caused by fungi that attack the plant at any time in its cycle of vegetative propagation. These diseases, described in the preceding section, include black rot, charcoal rot, foot rot, *Fusarium* root rot and stem canker, mottie necrosis, *Phymatotrichum* root rot, and surface rot (see Field and Storage Diseases).

Other storage rots, described in this section, are caused by fungi that infect sweet potatoes primarily through wounds inflicted during harvest. Although they occasionally infect the plant at other stages in its life cycle, they are most destructive in storage. *Rhizopus* soft rot is the most destructive of these, because it occurs wherever sweet potatoes are grown and decays whole roots quickly. It can therefore cause significant losses in both tropical and temperate growing regions. Java black rot is common in warmer regions, and dry rot occurs in temperate regions.

The other storage rots covered in this section occur primarily in temperate areas when sweet potatoes have been predisposed by unusually low temperatures. They are not normally considered economically significant diseases. This group includes *Alternaria* rot, blue mold rot, and gray mold rot. Punky rot, the remaining disease described in this section, may be more a laboratory artifact than a naturally occurring storage rot.

Other genera of fungi (*Aspergillus*, *Epicoccum*, *Pestalotia*, *Pleospora*, *Rosellinia*, *Schizophyllum*, and *Sclerotinia*) have occasionally been associated with decaying sweet potatoes but have not been shown to cause serious losses.

In addition to reducing the supply of food and disrupting the economics of growing sweet potatoes, storage rots pose another problem. Animals are often fed fleshy storage roots that remain in the field after harvest or are culled during grading before or after storage. Many of the organisms that cause storage rots simultaneously induce sweet potato to produce a group of related furanoterpenoid compounds. These compounds may serve a role like that of phytoalexins. The major furanoterpenoid component, ipomeamarone, causes liver toxicity, and several minor components, the ipomeanols, induce lung edema in mammals. Although the validity of these claims has been questioned, it seems prudent to avoid feeding animals infected sweet potatoes when possible.

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Rhizopus Soft Rot

Rhizopus soft rot, often referred to simply as soft rot or, when the infection is restricted, as ring rot or collar rot, is one of the most ubiquitous and costly fungal diseases of sweet potato. It is most prevalent in temperate and subtropical growing regions but is also common in tropical growing regions.

Symptoms and Signs

Symptoms develop only after fleshy roots have been harvested or occasionally in the field following flooding. Affected roots rapidly turn soft, moist, and stringy (Plate 30), much like those infected with the bacterial soft rot pathogen (see Bacterial Stem and Root Rot). The infection commonly progresses from one or both ends of the root, although it is occasionally initiated elsewhere. The color of the infected tissue is not significantly altered, but a pronounced odor is produced, reminiscent of the odors of alcohol and yeast associated with fermentations. As with bacterial soft rot, the odors from affected sweet potatoes attract fruit flies, which reproduce in the decaying tissue and become abundant in the storage area. The entire root is generally rotted, and the parenchyma is liquefied within a few days, but the periderm and fibers of the root are not extensively degraded. At low temperatures (2–5°C), where the



Fig. 34. Fleshy storage root infected with the soft rot pathogen, *Rhizopus stolonifer*. "Whiskers," consisting of mycelia and sporangia of the pathogen, have emerged from several places on the root. (Courtesy W. J. Martin)

predominant pathogens are *Mucor* spp., the decay progresses more slowly and may have a starchy odor.

Ring rot, or collar rot, resulting from infection of the fleshy root at one or more points between the ends, is essentially indistinguishable from the ring rot attributed to the mottle necrosis pathogen (see Mottle Necrosis). The infection progresses as a ring around the root. Several rings have been observed to occur on the same root. In most cases the infected

tissue dries, leaving a brown, relatively superficial necrotic ring. In some cases the entire root is quickly rotted, as with the more typical soft rot.

A diagnostic feature of *Rhizopus* soft rot, by which it can be easily distinguished from bacterial soft rot and other storage rots, is the development of so-called whiskers of the causal fungus through breaks in the periderm or through lenticels (Fig. 34). The whiskers are tufts of wide, coenocytic hyphae with numerous sporangiophores and sporangia (Fig. 35). These mycelia and sporangia strongly adhere to the surface of roots in close contact with infected roots in storage. The superficial fungal growth may develop naturally and can be induced by incubating infected roots in a moist chamber. The pathogen also produces stolons and rhizoids (types of specialized hyphae) on media and in infected tissue. Stolons arch over the surface of the substrate, rhizoids (rootlike hyphae) grow into the substrate at each point of contact, and sporangiophores rise above the substrate.

Causal Organisms

Several species in two genera of the Zygomycetes, often known as common bread molds, are capable of causing soft rot of sweet potato. *Rhizopus stolonifer* (Ehr. ex Fr.) Lind has been most commonly referred to in recent plant pathology literature, but *R. nigricans* Ehr. is the synonym preferred by taxonomists. The *R. nigricans* species complex causes the most soft rot at 6–22°C. *R. oryzae* Went & Prinsen-Geerligs is the predominant pathogen above 30°C. Both species are active between 22 and 30°C. In addition to *Rhizopus* spp., which are by far more commonly associated with soft rot, several *Mucor* spp., including *M. racemosus* Fres., *M. piriformis* Fisher, and *M. circinelloides* Van Tieghem, can infect sweet potato, at lower temperatures (2–5°C) than those at which *Rhizopus* is active. This description concentrates on the most commonly encountered and extensively studied, *R. nigricans*.

Disease Cycles

The disease cycle of soft rot is presented in Fig. 36. The causal fungi are ubiquitous, and spores are in the air constantly. In addition, inoculum can survive in crop debris in the soil and to some extent on contaminated equipment and crates. *Rhizopus* spp. require wounds for infection of sweet potato roots, and when either airborne sporangiospores or infested soil comes in contact with a wound, the spores germinate and hyphae enter the root. The pathogens rapidly decay the entire fleshy root, and mycelia grow out through breaks or natural openings in the periderm and produce sporangia. The sporangia (Fig. 35A) may subsequently rupture and release numerous sporangiospores (Fig. 35B) into the air, which may land on neighboring healthy sweet potatoes. If properly cured, neighboring roots do not become infected. However, when roots are washed, graded, and repackaged for shipment to market or bedded for plant production, new wounds are created, and secondary infections may be numerous.

The sexual stage of *R. nigricans* requires the presence of compatible mating types. When hyphae of compatible mating types grow in close proximity, branches called progametangia grow toward one another, make contact, and form gametangia, which fuse to form a zygote. The zygote differentiates into a zygospore, which overwinters in decaying tissue and soil. It germinates by forming a germ tube, which differentiates into a sporangiophore. The sporangiophore bears a sporangium, which releases sporangiospores into the air.

In addition to requiring wounds for infection, *Rhizopus* spp. also require the presence of dead host tissue. Thus, when sporangiospores from a pathogenic isolate are placed on wounds made by a clean cut or scrape, the resulting incidence of infection is low. However, if the spores are first incubated in a nutrient solution, if pectolytic enzymes are added to the inoculum, or if the wounds are made by striking the root with a hard object, the incidence of infection is much higher. Natural infections frequently originate at the ends of roots, because as

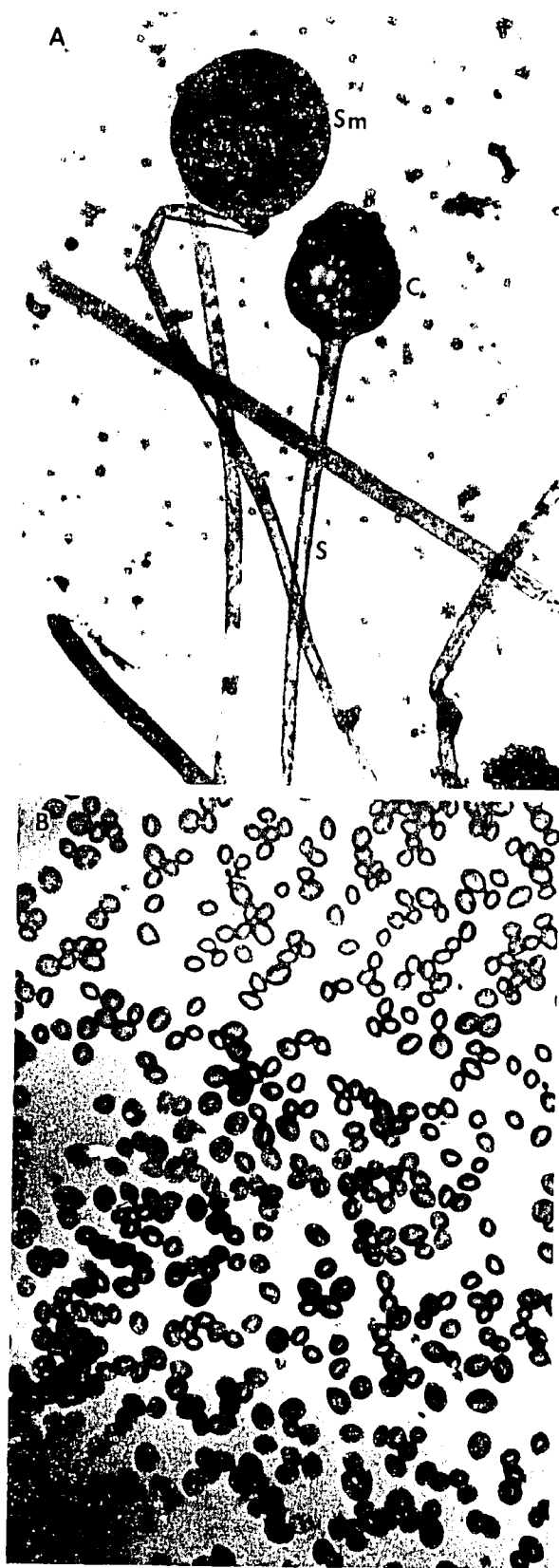


Fig. 35. Light micrographs of structures of the soft rot fungus *Rhizopus stolonifer*. A, Sporangiophores (S), sporangium (Sm), and exposed columella (C). B, Sporangiospores. (Courtesy C. A. Clark)

Color Plates



1. Soil rot (pox) lesions, caused by *Streptomyces ipomoea*, on fleshy storage roots of clone L4-89. (Courtesy W. J. Martin)



2. Rootlet rot caused by *Streptomyces ipomoea* on fibrous roots. (Courtesy W. J. Martin)



3. Bacterial root rot caused by *Erwinia chrysanthemi* on a storage root of the cultivar Jewel. (Courtesy C. A. Clark)



4. Vine affected with witches'-broom (right) and a healthy vine. (Courtesy F. W. Zettler)



5. Proliferation of axillary shoots associated with witches'-broom. (Courtesy F. W. Zettler)



6. Area in a plant production bed affected by sclerotial blight, caused by *Sclerotium rolfsii*. (Courtesy W. J. Martin)



7. Hyphae and sclerotia of *Sclerotium rolfsii* on the surface of the soil and the base of vines in a plant production bed affected by sclerotial blight. (Courtesy C. A. Clark)



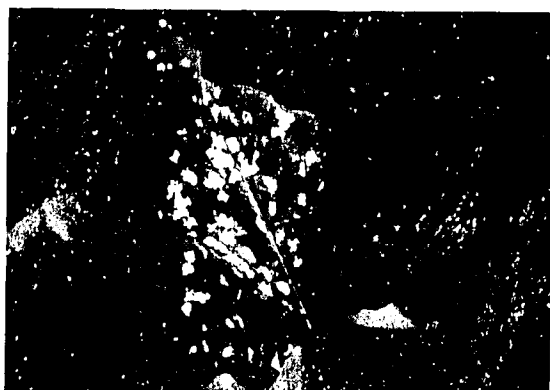
8. *Stemonitis* spp. on the surface of stems and leaves from a plant bed. (Courtesy C. A. Clark)



9. Light brown lesions with darker brown concentric rings, characteristic of leaf symptoms caused by *Alternaria* spp. (Courtesy G. W. Lawrence)



10. Small, dark brown lesions, characteristic of leaf infections by *Cercospora ipomoeae*. (Courtesy C. Martin)



11. Light gray lesions with dark brown borders and black specks (pycnidia) near the lesion center, diagnostic symptoms and signs of leaf blight caused by *Phyllosticta batatas*. (Courtesy W. J. Martin)



12. Leaf symptoms caused by *Elsinoë batatas*, typically including venal necrosis on the underside of leaves and severe leaf distortion. (Courtesy R. Gapasin)



13. Petiole and stem lesions caused by *Elsinoë batatas* in the early stages of disease development. (Courtesy R. Gapasin)



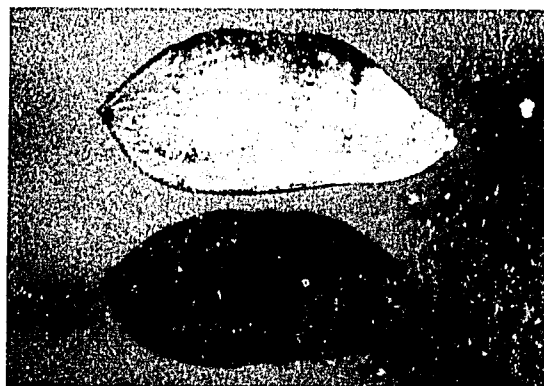
14. Early stages of white rust, caused by *Albugo ipomoeae-panduratae*. The first symptoms are distinct chlorotic lesions on the upper leaf surface. (Courtesy W. J. Martin)



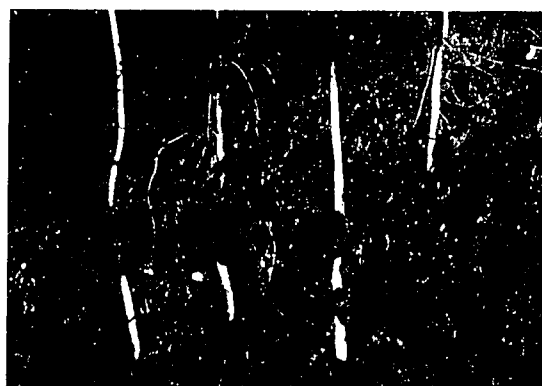
15. Late stages of the white rust syndrome. Pustules containing white sporangiospores develop in lesions on the lower leaf surface. (Courtesy K. Pohronezny)



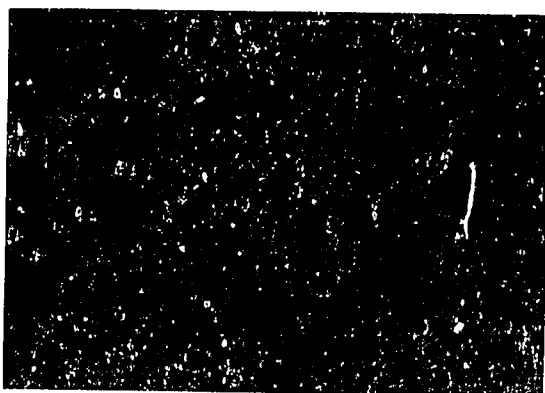
16. Fleshy storage roots with black rot lesions (left and right), caused by *Ceratocystis fimbriata*, and uninfected roots (center). (Courtesy W. J. Martin)



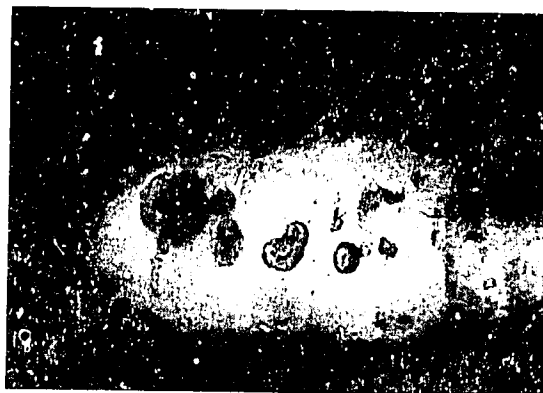
17. External and internal views of a fleshy storage root infected with the black rot fungus, *Ceratocystis fimbriata*. (Courtesy C. A. Clark)



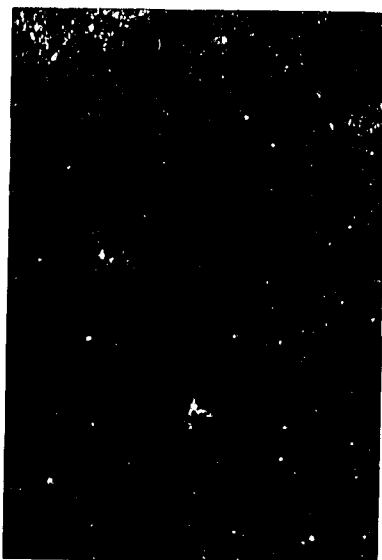
18. Sunken cankers caused by the black rot fungus, *Ceratocystis fimbriata*, on sweet potato slips. (Courtesy W. J. Martin)



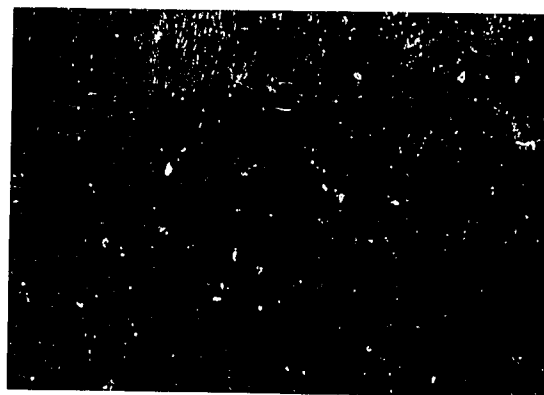
19. Internal views of a fleshy storage root with charcoal rot, caused by *Macrophomina phaseolina*. (Courtesy G. W. Lawrence)



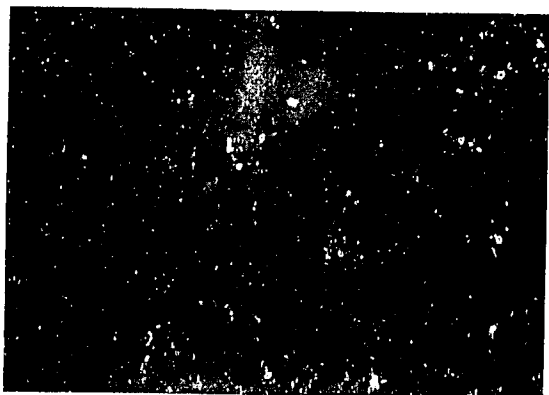
20. Circular spot lesions, caused by *Sclerotium rolfsii*, on a fleshy storage root of the cultivar Porto Rico. (Courtesy W. J. Martin)



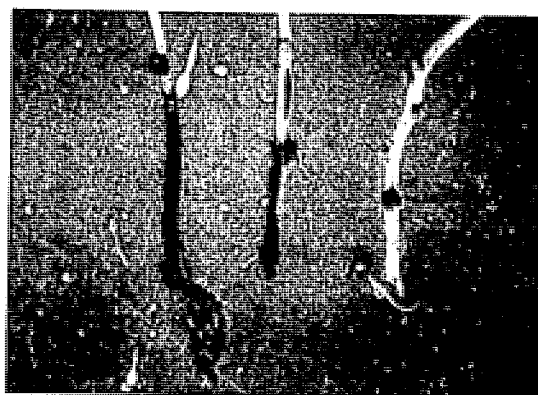
21. Wilting symptoms induced in the field by the foot rot fungus, *Plenodomus destruens*. (Courtesy C. A. Clark)



22. Fleshy storage root with foot rot, caused by *Plenodomus destruens*. A portion of the periderm is peeled back to reveal pycnidia of the pathogen. (Courtesy C. A. Clark)



23. Cross sections of storage roots of the cultivar Jewel with symptoms of surface rot (left) and Fusarium root rot (right). (Courtesy C. A. Clark)



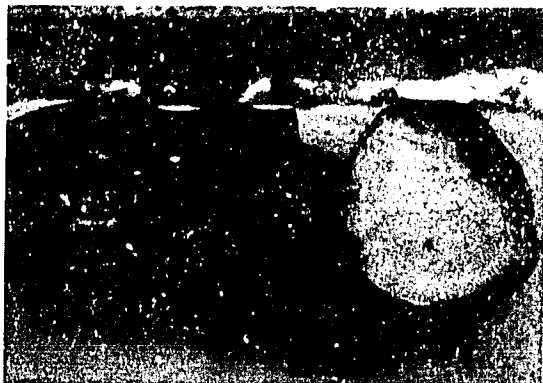
24. Stem canker phase of Fusarium root rot, caused by *Fusarium solani*, on the cultivar Jewel. (Courtesy J. W. Moyer)



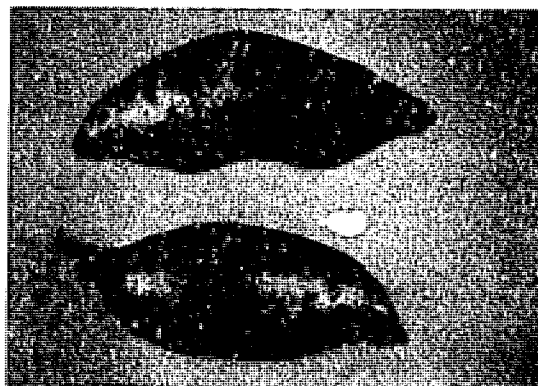
25. Symptoms of Fusarium wilt, caused by *Fusarium oxysporum* f. sp. *bafatas*, on clone L4-73 in the field. (Courtesy W. J. Martin)



26. Mottle necrosis in a storage root. (Courtesy J. C. Bouwkamp)



27. Internal and external views of a fleshy storage root with Phymatotrichum root rot, caused by *Phymatotrichum omnivorum*. (Courtesy G. Philley)



28. Symptoms of scurf, caused by *Monilochaetes infuscans*, on fleshy storage roots. (Courtesy G. W. Lawrence)



29. Scurf, caused by *Monilochaetes infuscans*, on an infected mother root and the sprouts produced from it. (Courtesy G. W. Lawrence)



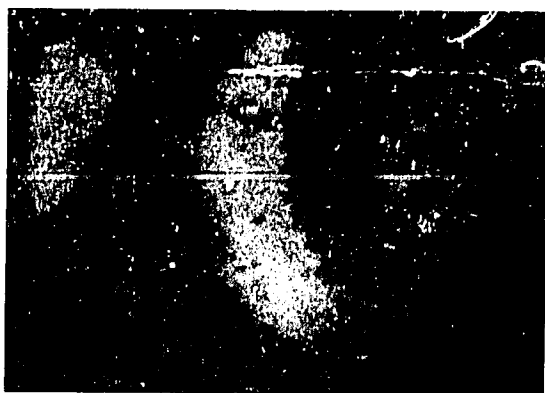
30. Internal and external views of a fleshy storage root infected with *Rhizopus stolonifer*. (Courtesy G. W. Lawrence)



31. Internal and external views of storage roots with Java black rot, caused by *Diplodia gossypina*, at different stages of disease development. (Courtesy J.-Y. Lo)



32. Restricted end lesion induced by *Diplodia gossypina* on a fleshy storage root. (Courtesy C. A. Clark)



33. Symptoms of dry rot, caused by *Diaporthe phaseolorum*, on fleshy storage roots. (Courtesy W. J. Martin)



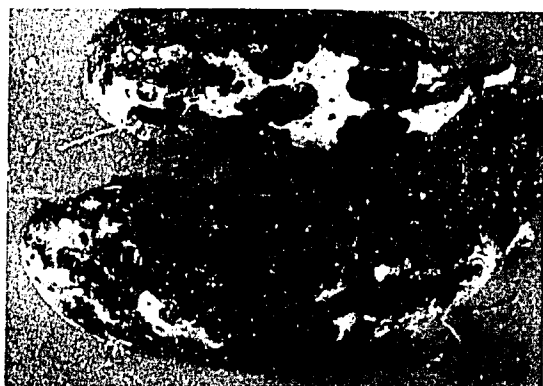
34. Punky rot, caused by a *Trichoderma* sp., on a fleshy storage root. (Courtesy W.-S. Wu)



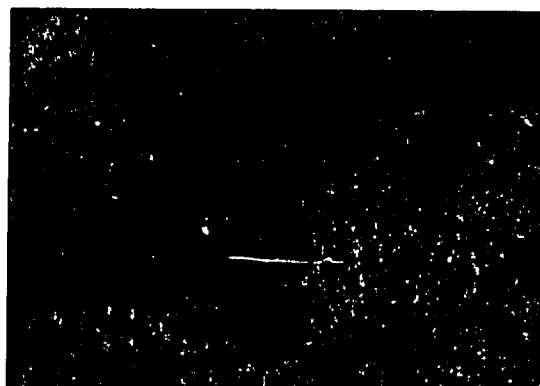
35. Blue mold, caused by *Penicillium* spp., on fleshy storage roots. (Courtesy W. J. Martin)



36. Galls and egg masses produced by *Meloidogyne incognita* on fibrous feeder roots. (Courtesy W. J. Martin)



37. Cracking of fleshy storage roots associated with injury by the root-knot nematode *Meloidogyne incognita*. (Courtesy G. W. Lawrence)



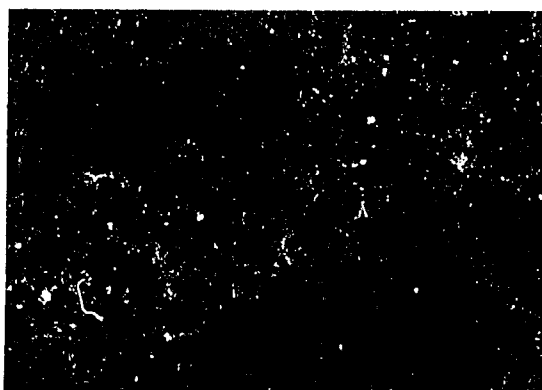
38. Subcortical lesions caused by root-knot nematodes, *Meloidogyne* spp., on storage roots. The lesions were not evident until the roots were peeled for canning. (Courtesy W. J. Martin)



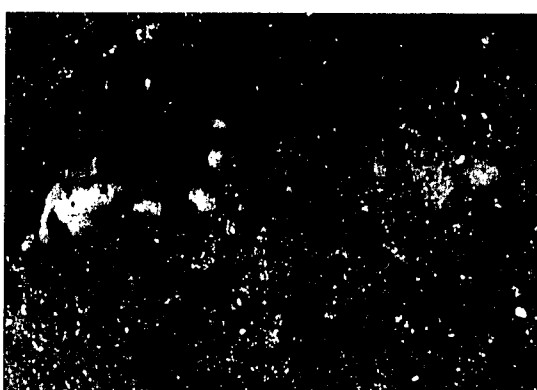
39. Cracking of fleshy storage roots associated with injury by the reniform nematode, *Rotylenchulus reniformis*. (Courtesy C. A. Clark)



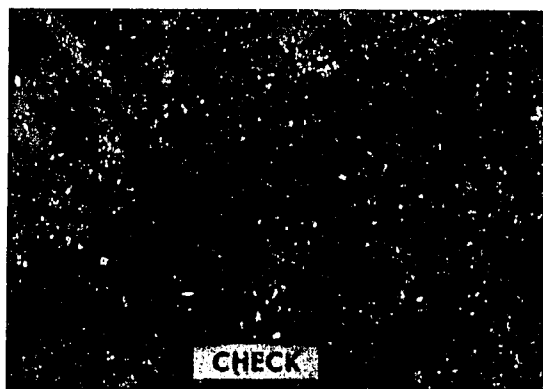
40. Lesions induced by the root-lesion nematode, *Pratylenchus colleeae*, on fleshy storage roots of the cultivar Norin No. 2. (Courtesy H. Kawagoe and K. Nakasono)



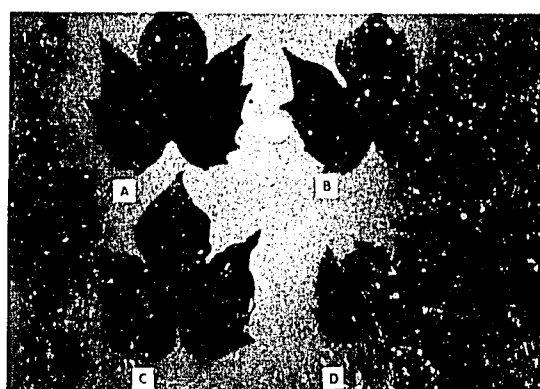
41. Leaf symptoms induced by sweet potato feathery mottle virus, including chlorotic patterns along veins (feathering) and interveinal chlorotic spots. (Courtesy J. W. Moyer)



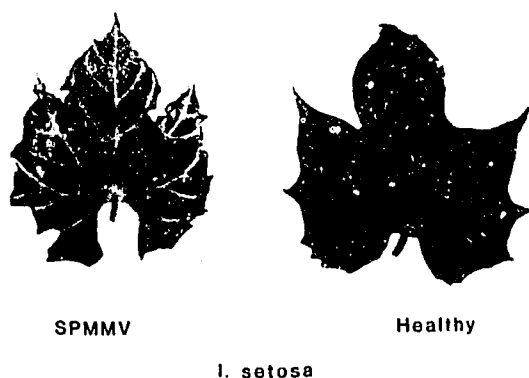
42. External root necrosis induced by the russet crack strain of sweet potato feathery mottle virus in a Jersey-type storage root. (Courtesy J. W. Moyer)



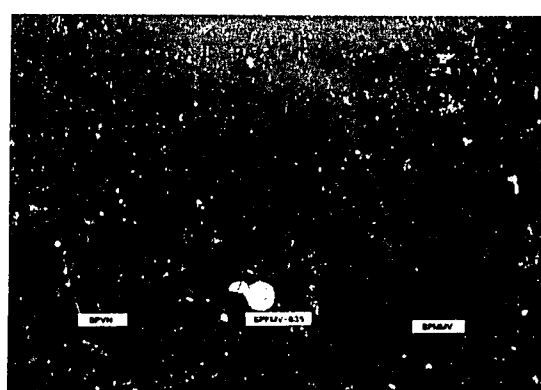
43. Internal root necrosis typical of internal cork disease. The orange-fleshed "check" is a root of a tolerant cultivar graft-inoculated from the same source used for the symptomatic roots. (Courtesy J. W. Moyer)



44. Development of symptoms of sweet potato feathery mottle virus in the indicator host *Ipomoea setosa*. Leaf A is from a healthy plant. Leaf B exhibits veinal chlorosis, usually the first symptom observed. Veinal chlorosis on subsequent leaves (C and D) is progressively restricted, until symptomless leaves are produced. (Courtesy J. W. Moyer)



45. Distinct veinal chlorosis induced in *Ipomoea setosa* by sweet potato mild mottle virus (left). (Courtesy J. W. Moyer)



46. Range of symptoms induced in *Nicotiana benthamiana* by (left to right) sweet potato latent virus (formerly known as sweet potato virus N, SPVN), sweet potato feathery mottle virus, and sweet potato mild mottle virus. (Courtesy J. W. Moyer)



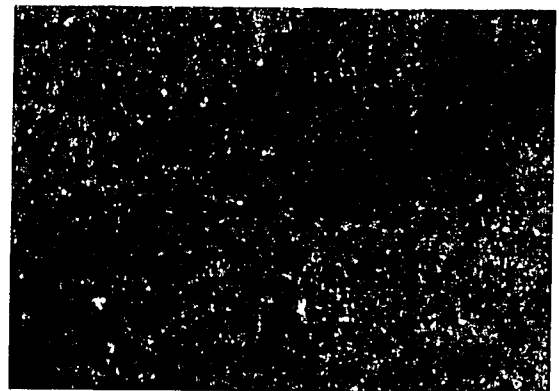
47. Symptoms of the sweet potato virus disease complex in clone TIB 8, with small, chlorotic, misshapen leaves at the tip of the vine (right), and a plant infected only with the sweet potato feathery mottle component of the complex (left). (Courtesy J. W. Moyer)



48. *Ipomoea setosa* graft-inoculated with (left to right) sweet potato feathery mottle virus, a whitefly-transmitted agent, and both agents, which in combination cause the sweet potato virus disease complex. (Courtesy J. W. Moyer)



49. Symptoms of sweet potato leaf curl disease. (Courtesy S. Green)



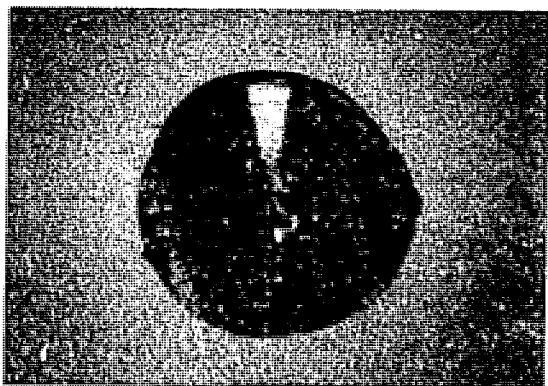
50. Chlorotic and distorted leaves symptomatic of chlorotic leaf distortion of sweet potato. (Courtesy C. A. Clark)



51. Sweet potato infected with sweet potato feathery mottle virus and an ilarviruslike agent. (Courtesy J. W. Moyer)



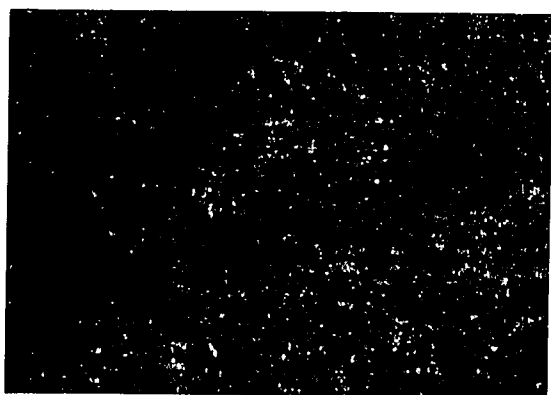
52. Mericlinal mutation of skin color in a storage root. (Courtesy L. H. Rolston)



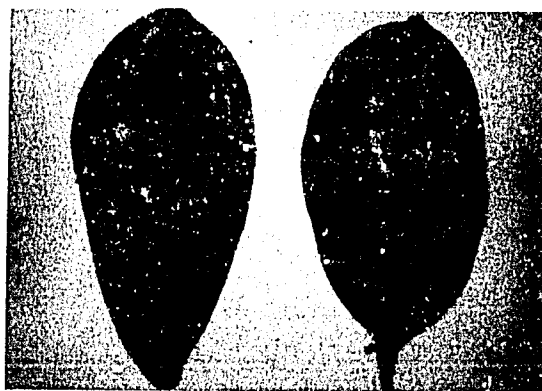
53. Storage root with a sectorial mutation of flesh color. (Courtesy C. A. Clark)



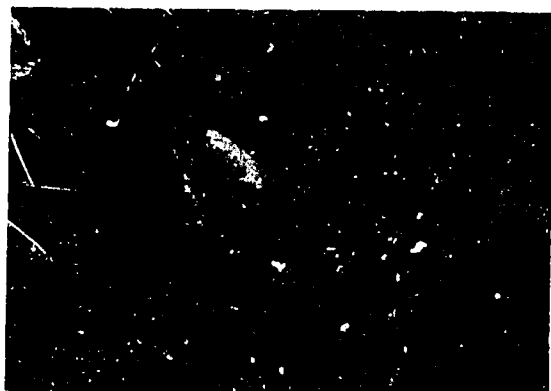
54. Intumescences on the adaxial (upper) leaf surface of sweet potato grown in the greenhouse. (Courtesy C. A. Clark)



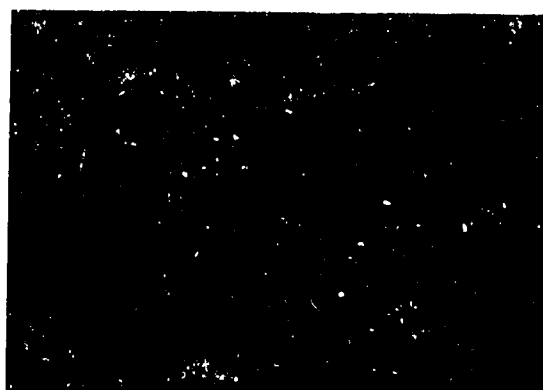
55. Internal breakdown and discoloration in a storage root of the cultivar Jasper exposed to cold during enlargement in the field. (Courtesy C. A. Clark)



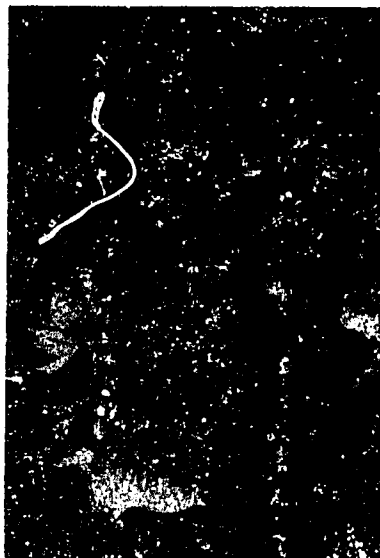
56. Skinned storage roots of the cultivar Jewel. The root on the right was not cured and is discolored and shrunken around the injury. The root on the left was properly cured immediately after skinning. (Courtesy C. A. Clark)



57. Plant grown in soil in which an unregistered tank mix of a nematicide and herbicide was incorporated prior to transplanting (right) and a plant grown in untreated soil. (Courtesy T. J. Monaco and C. E. Motsenbocker)



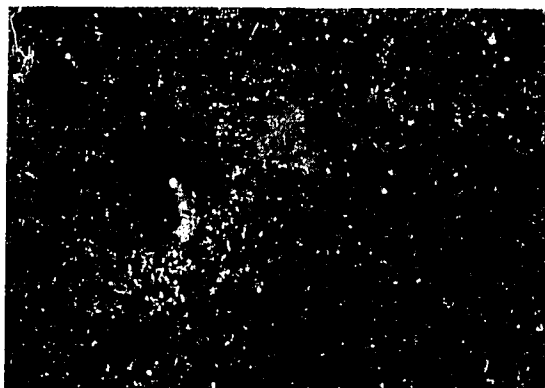
58. Veinal chlorosis and leaf necrosis resulting from a foliar application of diuron, a photosynthetic inhibitor. (Courtesy T. J. Monaco and C. E. Motsenbocker)



59. Interveinal chlorosis and leaf necrosis from a foliar application of bentazon. (Courtesy T. J. Monaco and C. E. Motsenbocker)



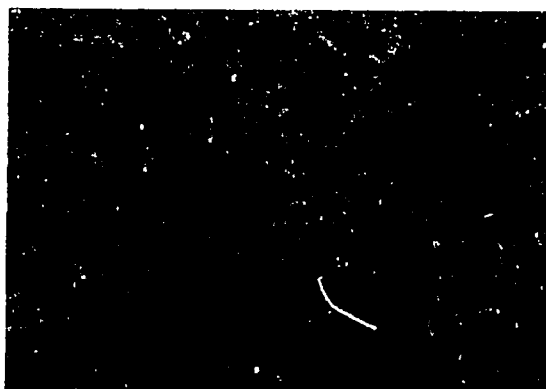
60. Petiole twisting caused by chloramben, a growth regulator; the injury is quickly outgrown and does not affect quality or yield. (Courtesy T. J. Monaco and C. E. Motsenbocker)



61. Yellowing and malformation of new leaves from foliar contact with glyphosate. (Courtesy T. J. Monaco and C. E. Motsenbocker)



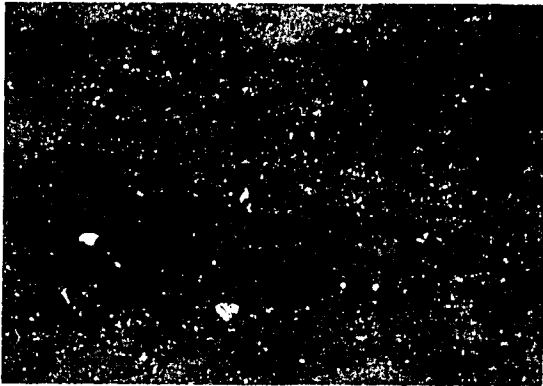
62. Inhibition of root development by a dinitroaniline herbicide incorporated into the soil prior to transplanting. (Courtesy T. J. Monaco and C. E. Motsenbocker)



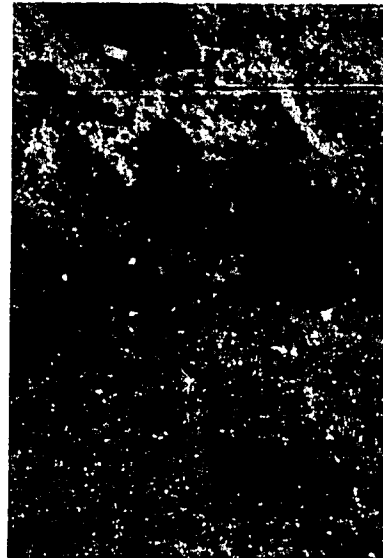
63. Plants grown in soil treated with a dinitroaniline herbicide (left) and a plant grown in untreated soil. (Courtesy T. J. Monaco and C. E. Motsenbocker)



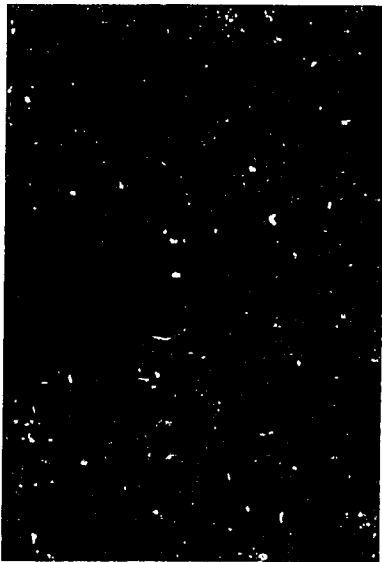
64. Off-shape and cracked roots (upper row), with injuries typical of damage caused by dinitroaniline, acetanilide, and thiocarbamate herbicides. Drought, excessive fertilizer, weed competition, and other stresses can also cause this effect. (Courtesy T. J. Monaco and C. E. Motsenbocker)



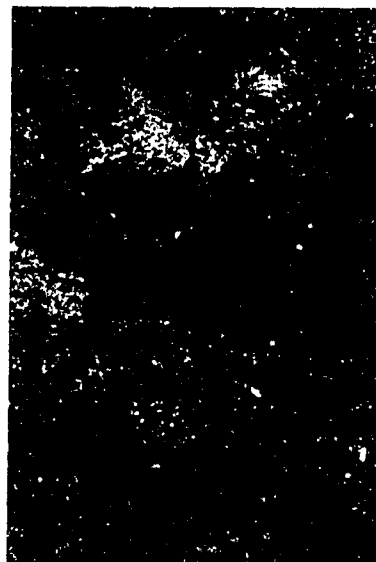
65. Leaf crinkle, leaf mottling, and yellowing of new leaves as the result of foliar contact with a sulfonylurea herbicide. (Courtesy T. J. Monaco and C. E. Motsenbocker)



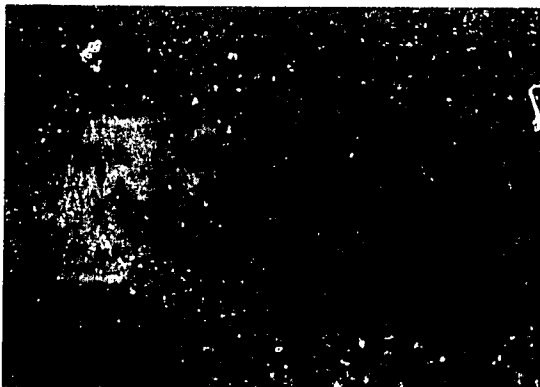
66. Leaf curling, leaf crinkle, and anthocyanin development from foliar contact with an imidazolinone herbicide. (Courtesy T. J. Monaco and C. E. Motsenbocker)



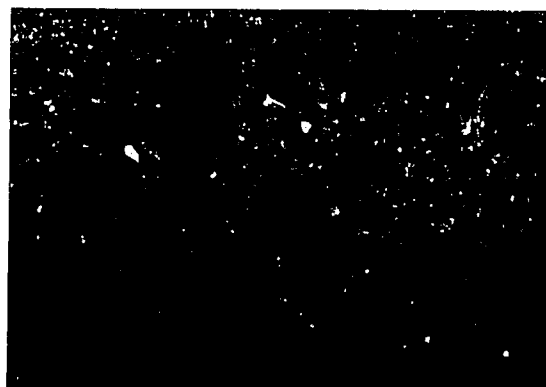
67. Stunting, leaf crinkle, and loss of apical meristem resulting from excessive dosage of DCPA, a cell-division-inhibiting herbicide. (Courtesy T. J. Monaco and C. E. Motsenbocker)



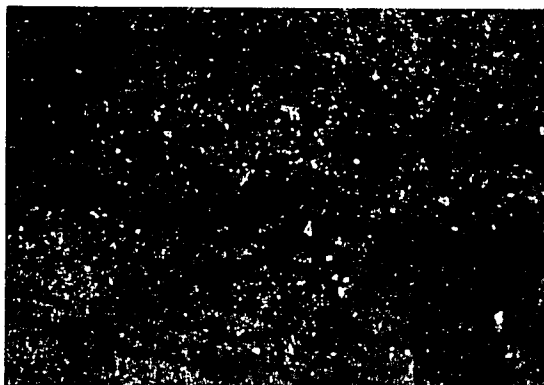
68. Rapid foliar necrosis resulting from foliar contact with contact herbicides, such as diphenyl ethers, bipyridiliums, and organic arsenicals. (Courtesy T. J. Monaco and C. E. Motsenbocker)



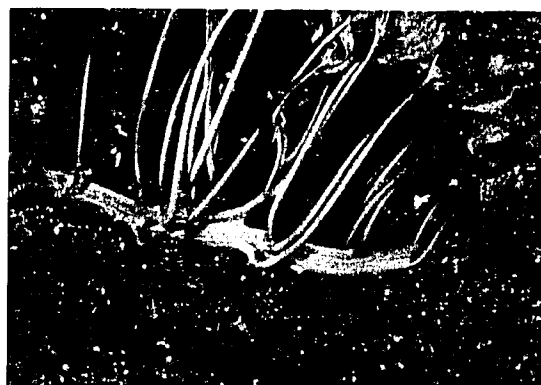
69. Internal discoloration of a storage root chilled during storage. (Courtesy L. G. Wilson)



70. Storage roots of the cultivar Travis with blister due to boron deficiency. (Courtesy C. A. Clark)



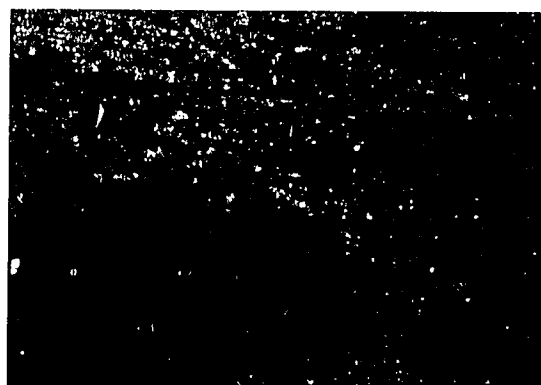
71. Distal end rot on a fleshy storage root. (Courtesy W. J. Martin)



72. Vine with flat fasciation. (Courtesy C. A. Clark)



73. Subcutaneous roots in a storage root. (Courtesy W. J. Martin)



74. Storage root with alligator skin. (Courtesy G. W. Lawrence)

they are detached from the vines, the slender attachment root is often twisted, and much of the tissue at the wound site is damaged.

R. nigricans secretes considerable amylase, pectinase, and cellulase. The pectolytic enzymes are elaborated in infected tissue and kill the tissue in advance of the fungal hyphae, which invade only dead tissue. The root tissue is extensively macerated and quickly becomes liquefied.

Epidemiology

R. nigricans is usually encountered at the temperatures at which sweet potatoes are commonly stored, and its epidemiology has therefore been studied more extensively than that of *R. oryzae*, which is more common in tropical areas, where roots are exposed to higher temperatures. The optimum temperature for growth of *R. nigricans* in culture is 28°C, but the optimum temperature for the production of pectolytic enzymes and infection of sweet potatoes is 20°C.

Relative humidity influences the initial stages of infection. At 23°C, the optimum relative humidity for the initiation of soft rot is in the range of 75–84%. Few infections occur at 93–99% relative humidity, but once an infection has been established it continues to enlarge at relative humidities of 50–100%.

Environmental factors also affect the formation of wound periderm, which effectively excludes the pathogen from roots. Thus factors that favor the rapid formation of wound periderm, such as proper curing conditions, can significantly reduce the occurrence of soft rot. Extended exposure after digging and prior to curing may result in heat damage from direct sunlight, which makes storage roots more susceptible to the disease. Chilling also predisposes them to the disease, even when they are subsequently returned to a temperature more suitable for storage. Likewise, the longer they are stored after harvest, the more susceptible they become. Soft rot is especially destructive when sweet potatoes from storage are washed, packed, and shipped to market during cold weather, unless proper controls are used.

Control

Since it is not practically possible to prevent contact between *Rhizopus* spp. and sweet potatoes, the most important control of soft rot is to reduce the available sites for infection. Care in handling during harvest is important in reducing unnecessary wounding. However, since infection is often initiated at the ends of roots, which of necessity are wounded by removal from the vine, proper curing immediately after harvest is essential. Furthermore, whenever roots are handled during the storage period, such as during washing, grading, and packing for market, new wounds are created and must be protected. Recuring is one possible approach to this problem, but it may lead to a greater incidence of Java black rot, or the roots may have to be shipped to market before they can be properly cured.

For this reason, a protective fungicide, 2,6-dichloro-4-nitroaniline (DCNA), is routinely applied to roots as they pass across grading lines in the United States. This reduces the incidence of soft rot in marketed sweet potatoes. However, wounds created after the application of the fungicide are not protected. Dipping seed roots or root pieces in DCNA or spraying DCNA over the tops of seed roots in plant production beds effectively reduces the occurrence of the disease in the beds.

All cultivars of sweet potato compared for their reaction to soft rot have been susceptible to varying degrees, and thus the development of resistant cultivars has not been pursued.

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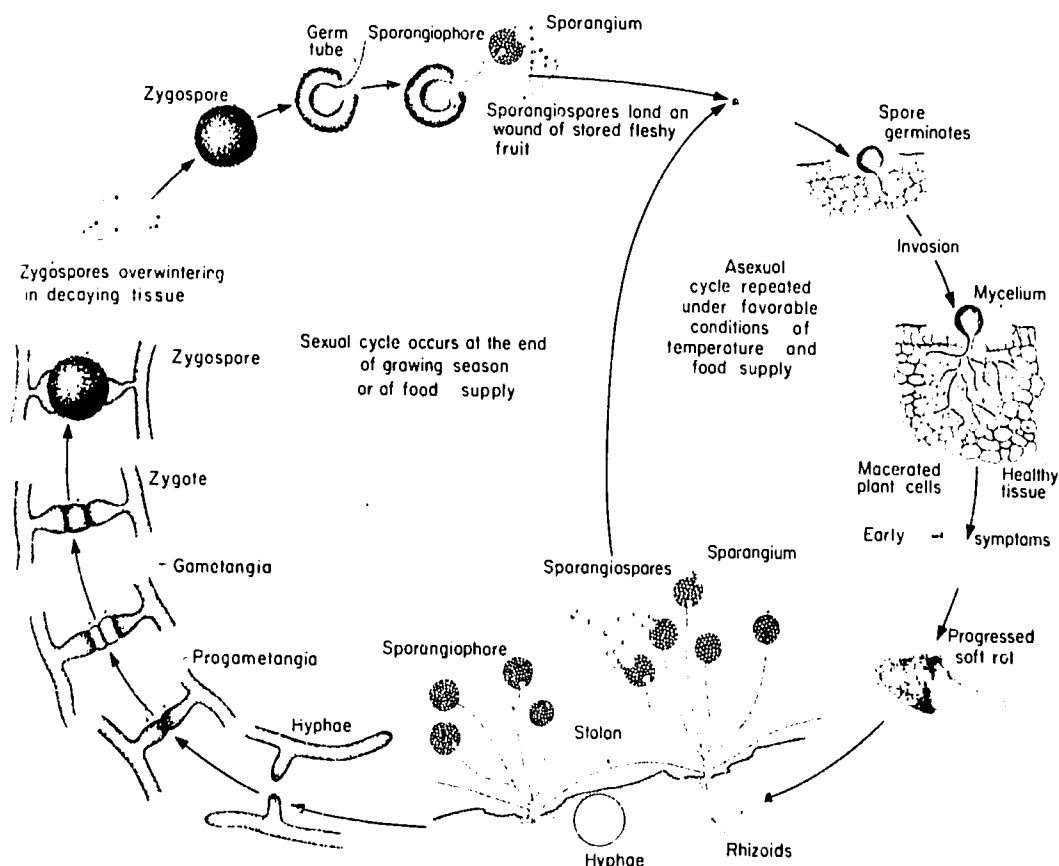


Fig. 36. Life cycle of the soft rot pathogen *Rhizopus stolonifer*. (Reprinted, by permission, from Agrios, 1978)

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Java Black Rot

Java black rot can be one of the most destructive postharvest diseases of sweet potato. It was given this name because it was first observed on fleshy storage roots imported to the United States from Java. The disease is found wherever sweet potatoes are grown but is more serious in warmer regions.

Symptoms and Signs

Java black rot is most frequently observed on fleshy roots in storage, usually progressing from one end or both ends of the root. Affected tissue is first brown to reddish brown and turns solid black as the decay progresses (Plate 31). When partially decayed roots are cut open and exposed to the air, the apparently healthy tissue surrounding the lesions gradually turns brown. The decay is firm and moist in consistency. Infected roots are often completely decayed within 2 weeks and subsequently dry out, becoming mummified and extremely hard. However, lesions may cease enlargement, and decay in storage is commonly restricted to the terminal 1-2 cm of the root (Plate 32). Several pathogens may induce such symptoms, but when the Java black rot pathogen is involved, the tissue at the center of the lesion is usually solid black. The few lesions initiated on the sides of the root are usually restricted before rotting much of it. They are generally circular, brown near the margin, and black in the center, with the periderm intact (Fig. 37).

The disease has not been observed on the fibrous feeder root system, but sprouts produced in plant beds infested with the pathogen sometimes develop a decay from the point where they arise from the mother root. The tissue turns black, and a firm, moist decay develops for a few centimeters from the point of attachment.

During the early stages of development, or when development is restricted, Java black rot can be confused with black rot, charcoal rot, and to a lesser extent *Fusarium* rots (see Field and Storage Diseases). Signs of the pathogen, which develop at later stages of infection, provide diagnostic evidence of the disease. 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 (Fig. 38). These stromatic masses contain numerous pycnidia, both near the surface and entirely embedded within the stroma (Fig. 39A). Many conidia are released as the stroma breaks down with age. The conidia and fragments of stromatic tissue form a black powder, which may coat the surface of any nearby objects, including neighboring roots or crates. When viewed microscopically, the conidia are at first one-celled, hyaline, and $11-14 \times 18-20 \mu\text{m}$, and as they "mature" they become two-celled and turn dark brown (Fig. 39B).

Causal Organism

Plant pathologists in different regions of the world have used several names for the anamorph of the Java black rot fungus. In the United States it has most frequently been called *Diplodia tubericola* (Ell. & Ev. apud Clendenin) Taub. In other countries the preferred name is *Botryodiplodia theobromae* Pat. Other synonyms include *D. theobromae* (Pat.) Nowell and *Lasiodiplodia tubericola* Ell. & Ev. apud Clendenin. One report indicated that strains may be specialized to certain hosts, but it has been generally recognized that this fungus causes diseases on many crops, with different names used for it on different hosts. Jones concluded that isolates from different hosts were indistinguishable and should be considered synonyms of the earliest valid name, *D. gossypina* Cooke. Two teleomorphs



Fig. 37. Restricted lateral lesion induced on a fleshy storage root by the Java black rot pathogen, *Diplodia gossypina*. (Courtesy C. A. Clark)

have been suggested, *Botryosphaeria quercuum* (Schw.) Sacc. and *Phyalospora rhodina* Cooke, but since no teleomorph has been found associated with sweet potato, *D. gossypina* appears to be the most acceptable name.

Disease Cycles and Pathogenesis

Primary infections of Java black rot are initiated by soilborne inoculum. Conidia of *D. gossypina* survive free in the soil for several years. The fungus may also survive in small cull roots or vines of sweet potato, left in the field and infested after harvest, or in debris from other susceptible crops, such as infected cotton bolls. The fungus is not capable of penetrating unwounded periderm of the root. Infested soil adheres to the broken ends of sweet potatoes at harvest, and the pathogen subsequently enters through the ends or through wounds created elsewhere on the root during handling.

Conidia deposited on the surface of crates survive from one season to the next and serve as an additional source of inoculum. Wounds inflicted on roots as they are placed in crates during harvesting may serve as infection courts.

Because the periderm is an effective barrier to penetration of the root by *D. gossypina*, the pathogen does not spread on properly cured roots in storage, despite the abundance of inoculum, unless they are freshly injured. However, when roots are washed, sorted, and packaged for shipment to market, or when they are handled for bedding for plant production, many new wounds are created, and conidia produced in the primary cycle of infection are spread over the root surface. The resultant secondary cycle of infection is often far more destructive than the primary cycle.

When infected roots are bedded, or when roots become infected in plant beds, the sprouts produced on them may also become infected. The pathogen grows slowly from mother roots into the basal portion of sprouts, seldom advancing more than 2–5 cm from the mother root by the time the sprouts have grown

sufficiently for transplanting. Infected tissue can be avoided if vines are cut above the soil line, but if slips are pulled, some infected tissue is carried with them. Using infected slips does not appear to significantly affect the growth of vines in the field or the transmission of the pathogen to daughter roots, but a low level of infection occasionally results.

D. gossypina is generally considered to be airborne on other crops. However, when artificially inoculated into wounded sweet potato vines, it grows no more than 2–5 cm from the site of inoculation over the course of a growing season and causes only a limited infection. Thus, although a low incidence of infection may result from airborne inoculum or from the use of infected transplants, it appears negligible relative to infection from soilborne inoculum or, more importantly, secondary disease cycles during handling in storage.

Limited studies indicate that *D. gossypina* produces pectolytic and cellulolytic enzymes near the margins of lesions. The infection results in a reduction of the carbohydrate, protein, and lipid contents of the tissue and the production of substantial concentrations of furanoterpenoid compounds (including ipomeamarone), similar to phytoalexins.

Epidemiology

D. gossypina is a warm-temperature pathogen; the optimum range for the development of Java black rot is 20–30°C. The activity of the pathogen declines sharply above this range. The disease develops over a wide range of relative humidities. Conidial germination and initial infection can occur in less than 6 hr, limiting the pathogen's exposure prior to entering the root.

Fleshy roots become more susceptible to Java black rot with time in storage. Those stored for 5–8 months are more



Fig. 38. Fleshy storage root with Java black rot, caused by *Diplodia gossypina*. Domes of stromatic tissue have broken through the periderm in several places. (Courtesy C. A. Clark)

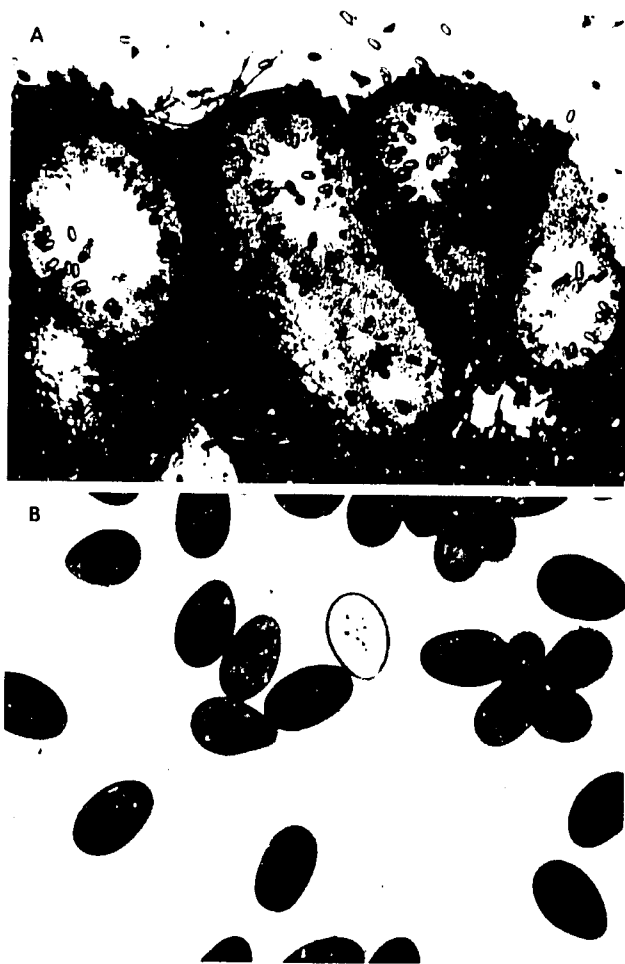


Fig. 39. Light micrographs of structures of the Java black rot pathogen, *Diplodia gossypina*. A, Pycnidia embedded within stromatic tissue of the fungus near the surface of an infected storage root. B, Hyaline one-celled and dematiaceous two-celled conidia. (Courtesy C. A. Clark)

susceptible than freshly harvested roots. Those handled after storage for marketing or bedding are exposed to higher inoculum densities due to conidia produced from primary infections, and they are also more susceptible.

Optimal conditions for Java black rot are similar to those recommended for curing sweet potatoes. Despite this, immediate curing of freshly harvested roots reduces the incidence of the disease, provided that conditions are carefully controlled. However, curing roots again after they have been stored for 5-8 months, to promote the healing of wounds inflicted during packing, may result in more Java black rot than returning them to recommended storage temperatures (approximately 16°C).

Chilling injury to roots 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. Gas exchange is also important to the development of the disease; roots exposed to flooding in the field or poor ventilation in storage are more susceptible.

Since conditions for infection by *D. gossypina* and conditions for wound healing by fleshy roots are so similar, inoculum potential is very important in determining the incidence of infection. Little is known regarding factors that affect the survival and density of the pathogen in soil or on infested equipment, such as crates.

Certain insects, including sweet-potato weevils and cockroaches, may carry *D. 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.

Control

Control of Java black rot requires integrating several practices. Prior to harvest, any previously used storage containers should be washed and disinfested. Where possible, sweet potatoes should be harvested before exposure to flooding or cold in the field. Care should be taken to minimize wounding during harvesting, and immediately upon being harvested, roots should be properly cured and then stored at about 16°C.

When sweet potatoes are removed from storage for bedding, infected roots should be culled, and the remaining roots should be dipped in a suspension of an effective fungicide, such as thiabendazole. When they are removed from storage for washing, sorting, and packing for market, they should be handled quickly, since this is a weak point in the control program for the disease. Roots should not be recured but should be held as close to 16°C as possible until consumed. Treating them 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. Dichloronitroaniline is less effective than thiabendazole but, at allowable concentrations, may slightly reduce disease incidence.

Although *D. gossypina* is not thought to be transmitted to any significant extent on infected transplanting stock, it is prudent to use vines cut above the soil line for transplanting. In addition, rotation with crops not usually infected by the pathogen reduces the density of soilborne inoculum in both the field and plant beds.

Preliminary investigations indicate significant differences in the susceptibility of sweet potato cultivars and breeding lines to Java black rot, especially when they are evaluated after several months of storage. This potential control mechanism has not been exploited but offers hope for improved control of the disease.

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Dry Rot

Dry rot is common in stored sweet potatoes but does not generally cause serious losses, and little research has been reported. It may be found in plant production beds and in the field, but to a much lesser extent than in storage. Dry rot may be difficult to distinguish from foot rot (see Field and Storage Diseases), caused by *Plenodomus destruens*, not only because symptoms are similar but also because the causal fungi are closely related and produce similar conidia.

Symptoms and Sign.

Dry rot symptoms are most frequently observed on fleshy roots in storage. The older literature indicates that the entire root may be rotted in a few weeks, but the decay is more often limited to one end of the root (Plate 33). The decay progresses from the end and causes the root to shrink and wrinkle. The lesion may be light to dark brown on the outside and dark brown to black on the inside. The decayed tissue is firm and dry and eventually becomes mummified. If the decay progresses sufficiently, pycnidia formed beneath the periderm break through it and become visible as minute, black, raised bodies on the surface of the root.

If infected roots are bedded for plant production, the sprouts produced from them may become infected. A reddish brown to black decay, similar to that caused by *Ceratocystis fimbriata* (see Black Rot), develops at the base of the sprout, and pycnidia may occasionally develop as the decay progresses.

Symptoms were also reported in the older literature to occur on vines, where numerous pycnidia are produced. However, this may be the result of saprophytic colonization of senescent vines; because of its saprophytic ability, the pathogen may also occur as a secondary invader.

Causal Organism

Plant pathologists have for many years referred to the causal fungus of sweet potato dry rot as the teleomorph *Diaporthe batatas* (Ell. & Halst.) Harter & Field. However, since the same fungus also causes pod and stem blight of soybean and pod blight of lima bean, taxonomists have concluded that the

valid name for the teleomorph is *D. phaseolorum* (Cooke & Ell.) Sacc. The anamorph is *Phomopsis phaseoli* (Desm.) Sacc. The ascigerous stage has been produced in culture with isolates from sweet potato but has not been observed on naturally infected sweet potato. Pycnidia occur separately in stem or vine tissue and are embedded in a stroma in root tissue. They are globose, $60\text{--}130 \times 60\text{--}110 \mu\text{m}$, with a short neck. The conidia are hyaline, one-celled, somewhat oblong to ellipsoidal, usually biguttulate, and $6\text{--}8 \times 3\text{--}5 \mu\text{m}$.

Epidemiology

Like most storage rot pathogens, *D. phaseolorum* requires a wound for infection. On leguminous hosts it commonly produces latent infections, which do not become apparent until the infected tissue approaches senescence. Whether this can occur on sweet potato is unknown. Infection of sprouts in plant beds is greater at 30°C than at 24 or 32°C .

Control

Dry rot has not caused sufficient losses to warrant control. However, practices used for control of other sweet potato pathogens also appear to reduce the incidence of this disease. These include careful handling to avoid wounding roots at harvest, immediate curing of harvested storage roots, and treatment of seed roots with an effective fungicide to limit the spread of infection from roots to sprouts in plant beds. Casual observations suggest that cultivars differ in their susceptibility to the disease.

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Punky Rot

Punky rot, caused by *Trichoderma koningii* Oud., which affects only the fleshy root of sweet potato, has not often been found in nature. However, the causal fungus has frequently been isolated from sweet potatoes with various other fungal

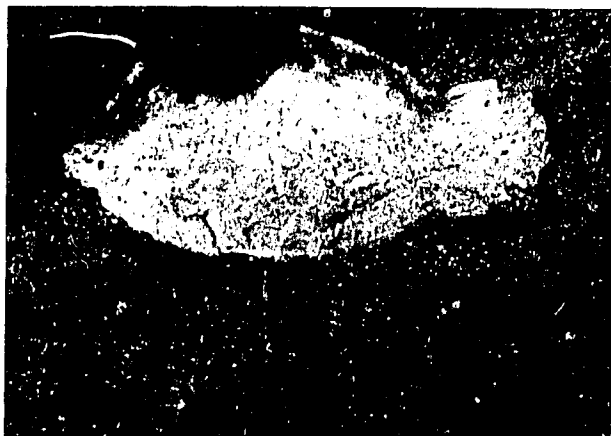


Fig. 40. Internal and external views of a storage root with symptoms of punky rot, after artificial inoculation with *Trichoderma* spp. (Courtesy C. A. Clark)

storage rots, such as soft rot or ring rot (see *Rhizopus* Soft Rot), *Fusarium* root rot (see Field and Storage Diseases), or Java black rot (described above), and on occasion from apparently healthy tissue. In fact, the original description of punky rot resulted from research on the cause of ring rot, which is due to infection by *Rhizopus* spp. When pure cultures of *T. koningii* originally isolated from ring rot infections were inoculated back into fleshy roots, a fairly vigorous rot distinct from the original ring rot was produced. Viewed externally, such artificially induced lesions are circular, light brown, and often wrinkled (Fig. 40). The flesh is firm, water-soaked, brown at the center of the lesion, and black at the margins. White mycelial growth may appear on the surface of the root when the decay is advanced, and masses of green spores may later appear on the mycelia (Plate 34). Mycelia have also been observed to grow over the surface of a root without entering until they encounter a wound that allows them to penetrate the root.

Since *Trichoderma* spp. are hyperparasites of many plant-pathogenic fungi, *T. koningii* may occur in decaying sweet potatoes more as a parasite of the primary storage rotters than as a pathogen in its own right.

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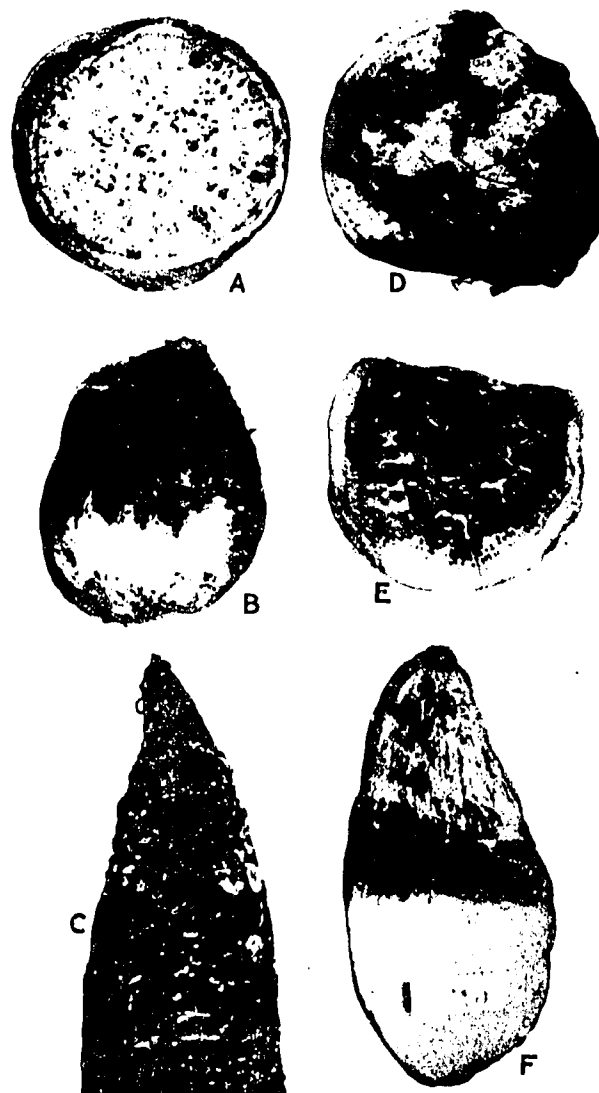


Fig. 41. Sweet potato storage rots caused by *Mucor racemosus* (A), *Alternaria* spp. (B), *Penicillium* spp. (C), *Botrytis cinerea* (D), *Epicoccum* sp. (E), and *Fusarium oxysporum* (F). (Reprinted, by permission, from Harter et al, 1918)

Alternaria Rot

Alternaria rot is neither a common nor an important storage rot of sweet potatoes. *Alternaria* spp. have been isolated from naturally infected fleshy roots on only a few occasions, generally when the roots had been held at low temperatures. The disease is a moist, firm rot that turns the affected tissue first light brown and then darker brown (Fig. 41B).

It has not been determined whether any relationship exists between the species of *Alternaria* that cause leaf spot or the more aggressive shoot blight (see Alternaria Leaf Spot and Stem Blight) and the species associated with Alternaria storage rot.

Selected Reference

Harter, L. L., Weimer, J. L., and Adams, J. M. R. 1918. Sweet potato storage rots. J. Agric. Res. 15:337-368.

Blue Mold Rot

Blue mold rot, caused by *Penicillium* spp., is not an important disease of sweet potatoes. It occurs exclusively on stored fleshy roots but is not normally found on those stored at or above the recommended 16°C. Occurrence of the disease is generally a reliable indication that the roots have been injured by chilling. *Penicillium* spp., which are more saprophytic than pathogenic, may also occur as secondary invaders, especially

following the development of soft rot.

Tissue decayed by *Penicillium* spp. is firm, dry to slightly moist, and brown. The signs of the pathogen are diagnostic and consist of cushions of white mycelia on the outer surface of the root (Fig. 41C), which eventually become covered with masses of blue to bluish green spores (Plate 35).

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Daines, R. H. 1970. Effects of temperature and a 2,6-dichloro-4-nitroaniline dip on keeping qualities of 'Yellow Jersey' sweet potatoes during the post storage period. Plant Dis. Rep. 54:486-488.

Harter, L. L., Weimer, J. L., and Adams, J. M. R. 1918. Sweet potato storage rots. J. Agric. Res. 15:337-368.

Gray Mold Rot

Gray mold rot occurs occasionally on sweet potatoes held at low temperatures and is not normally significant. It is caused by *Botrytis cinerea* Pers. ex Fr., which causes postharvest decays, petal blights, and leaf spots on many vegetable and ornamental crops. The decay produced on fleshy roots progresses slowly, turning the affected tissue gray and giving it a soft, watery consistency (Fig. 41D) and a starchy odor.

Selected Reference

Harter, L. L., Weimer, J. L., and Adams, J. M. R. 1918. Sweet potato storage rots. J. Agric. Res. 15:337-368.

Floral, Seed, and Seedling Diseases

Since sweet potatoes are universally produced by vegetative propagation, diseases associated with the flowers, botanically true seed, and seedlings are not of direct economic significance. For this reason, very little research has been conducted on diseases associated with the sexual reproduction cycle. However, many programs throughout the world are engaged in improving sweet potatoes by breeding and obviously must be able to make crosses and produce and germinate true seed. In addition, the possibility of developing methods for commercially propagating sweet potato from true seed planted directly in the field has aroused some interest.

A disease of pollen mother cells can cause tetrads to appear as dyads or polyads, with an increased number of microcytes and pollen grains that are fused, misshapen, or enlarged. However, the anthers appear normal. *Fusarium moniliforme* (Sheldon) Snyd. & Hans. has been associated with these symptoms, but Koch's postulates have not been satisfied, and the fungus is frequently isolated from apparently healthy vines following surface sterilization. In a separate study, however, application of a systemic fungicide to flowering vines in the nursery resulted in increased pod set, number of seed per pod, and proportion of healthy seed.

"Healthy" true seed is generally selected in breeding programs by floating the seed in water. Seed that sinks is considered potentially viable, and seed that floats is discarded as inviable. Etiological studies are lacking, and no precise cause or causes have been defined for the conditions under which seed

floats or subsequently rots. Floating could result from genetic aberrations of sweet potato, microbial activity, or environmental factors. However, the fact that systemic fungicides may sometimes increase the proportion of healthy seed indicates the possible involvement of fungi.

Even though seeds are normally planted in greenhouse beds containing soil that has been either pasteurized or sterilized, seed rots and damping-off have been observed. *Rhizoctonia solani* Kühn causes the greatest losses; *Pythium* spp., *Fusarium* spp., *Sclerotium rolfsii* Sacc., and soft-rot bacteria cause similar problems. Older seedlings are often susceptible to stem rots caused by *Sclerotinia* spp., *Fusarium* spp., and *S. rolfsii*. *Meloidogyne* spp. infect small seedlings and cause severe stunting and even death. Considering the array of diseases that can affect sweet potato seedlings under the relatively controlled conditions of the greenhouse, it seems likely that many more may be encountered if true seed are planted directly in the field.

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Nematode Diseases

Many genera of plant-parasitic nematodes are associated with sweet potato in the field, but only *Meloidogyne* (see Root-Knot Nematode) and *Rotylenchulus* (see Reniform Nematode) have been studied in association with the plant to any significant extent.

Two other genera, *Pratylenchus* (see Lesion Nematode) and *Ditylenchus* (see Brown Ring), are migratory endoparasites that evoke diagnostic symptoms on sweet potato and, although found only occasionally, can reduce the quality or yield of infected plants.

Several genera of migratory ectoparasites have been associated with stunting and reduced vigor of sweet potatoes in the field but have not been sufficiently studied to reliably determine the extent of their influence on growth and yield. These include the genera *Belonolaimus* (see Sting Nematode), *Trichodorus* (see Stubby-Root Nematode), and *Tylenchorhynchus*.

Helicotylenchus spp. (see Spiral Nematode) have been repeatedly associated with sweet potato production throughout the world and reproduce well on sweet potato, but they do not appear to damage the crop.

Several species of *Hoplolaimus* (lance nematode) are associated with sweet potato, but little is known of the effects of most of them on growth and yield. However, *H. columbus* Sher, which occurs in the southeastern United States, neither reproduces well on sweet potato nor causes significant reduction in yield.

Finally, several genera of plant-parasitic nematodes, mostly migratory ectoparasites, have been found in association with sweet potato, but data concerning their effects are lacking. These include *Aphelenchoides*, *Aphelenchus*, *Criconea*, *Criconea*, *Hemicycliophora*, *Longidorus*, *Paratylenchus*, *Quinisulcius*, *Radopholus*, *Scutellonema*, *Tylenchus*, and *Xiphinema*. Further research is needed to determine the effects of the ectoparasitic genera on yield and their population dynamics on sweet potato and to develop integrated programs for their control, where necessary.

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Root-Knot Nematode

The root-knot nematode is one of the most destructive nematodes on sweet potato, because it is widely distributed, and because it reduces both yield and quality of fleshy storage roots.

Symptoms and Signs

The most conspicuous and diagnostic symptom of root knot on most crops is round to spindle-shaped swellings (galls) on roots, for which the disease is named. Such galls occur on infected fibrous roots of sweet potato (Plate 36). They are often smaller (1-2 mm in diameter) than galls on the roots of many other plants. In fact, their size varies significantly among cultivars of sweet potato, and in many cases they are too small to distinguish visually.

Egg masses are often observed on the surface of galls and on roots even when galls are not visible. The egg masses vary from a translucent white to a golden brown and are approximately 0.5-1 mm in diameter (Fig. 42). When galls or segments of roots bearing egg masses are teased apart, the females (Fig. 43A and B) and the individual eggs that make up the egg mass (Fig. 43B and D) can be observed with low magnification.

Infected root systems may show different patterns of necrosis. Resistant reactions, small necrotic flecks, or lesions develop when roots are infected by certain species or populations of root-knot nematodes. In such cases, the nematodes usually fail to produce egg masses or mature. Large portions of the root system may also become necrotic as a result of the activities of secondary organisms, especially during late stages of the growing season.

Secondary symptoms may result from infection of the feeder root system. Infected feeder root systems are usually shorter and have fewer secondary roots and root hairs. Additional symptoms, such as reduction in vine growth, yellowing, flagging (transient wilting of foliage), or abnormally abundant production of flowers, may result from the loss of vigor of the root system.

The most dramatic symptom on fleshy roots is the occurrence



Fig. 42. Galls produced by *Meloidogyne incognita* and egg masses of the nematode on fibrous feeder roots. (Courtesy C. A. Clark)

of longitudinal cracks (Plate 37), similar to growth cracks. Cracks initiated early in the growing season are long and deep and have periderm over much of the exposed surface. Those initiated later in the growing season are generally shorter and shallower and have necrotic margins as a result of the activity of secondary organisms. However, cracks may be induced by many biotic and abiotic agents, and cultivars vary considerably in their tendency to crack in response to the root-knot nematode. Occasionally, bumps or blisters form on the surface of infected fleshy roots (Fig. 44).

A more consistent feature of root knot is the presence of the nematode within fleshy roots. As centripetal enlargement of the root continues following infection, the root tissue envelops the developing nematodes, and a layer of cork tissue is usually

produced around them (Fig. 45). Such pockets may contain nematodes in various stages of development. They are often associated with cracks in the root but may also be found within roots with no external symptoms. In the latter case the brown-black cork tissue becomes apparent when the roots are cut open for cooking or peeled in preparation for commercial canning (Plate 38).

Causal Organisms

Several species of *Meloidogyne* infect sweet potato, including *M. arenaria* (Neal 1889) Chitwood 1949, *M. hapla* Chitwood 1949, *M. incognita* (Kofoid & White 1919) Chitwood 1949, and *M. javanica* (Treub 1885) Chitwood 1949. Each species is an obligate parasite with an extensive host range. *M. arenaria*

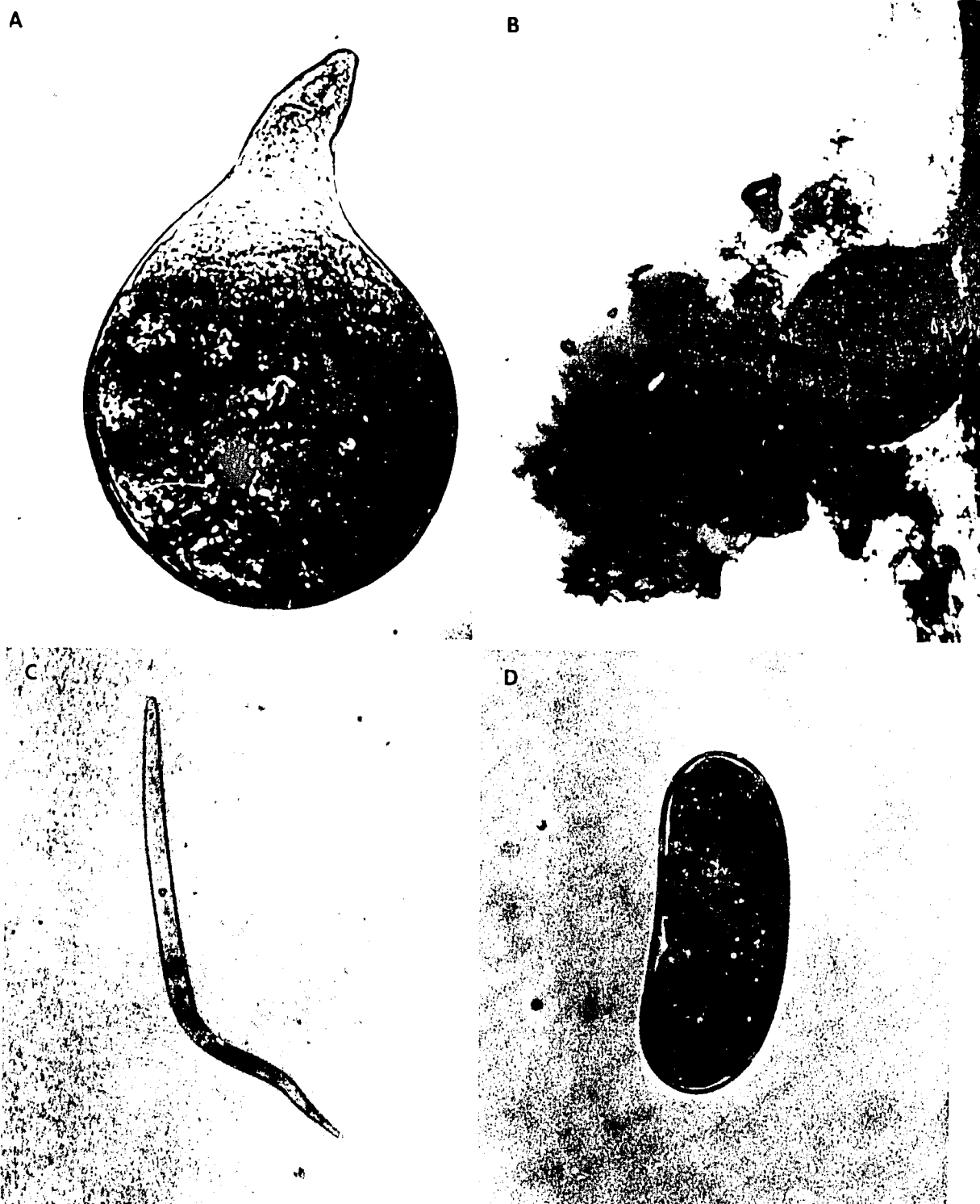


Fig. 43. Light micrographs of the root-knot nematode *Meloidogyne incognita*. A, Mature female. B, Mature female and egg mass in an infected fibrous root. C, Juvenile. D, Egg. (A and B, courtesy G. W. Lawrence; C and D, courtesy C. Pace)

induces necrosis on root systems and does not develop to maturity within roots. *M. hapla* (also known as the northern root-knot nematode) normally occurs at latitudes cooler than those at which sweet potatoes are most frequently grown, but *M. incognita* (also called the southern root-knot nematode) and *M. javanica* are often found on sweet potato in the field. Because *M. incognita* is most frequently associated with sweet potato, it has been more extensively studied. The remainder of this account therefore deals primarily with this species.

Although it usually reproduces parthenogenetically, *M. incognita* is a complex species with considerable variation, especially in characteristics such as host range and virulence. The morphological traits that distinguish it from other species of the genus are larval body length (its mean length is 0.376 mm), location of the female excretory pore opposite the stylet knobs, perineal patterns, male head shape, and stylet morphology. In addition, the ability to produce galls and egg masses on plants of the North Carolina differential host test is used to distinguish this species and its four host races from the other three common plurivorous species. All four of the host races may occur on sweet potato. Races 1 and 3 are most commonly associated with the crop in the United States. However, populations of each race vary considerably in their virulence on different clones of sweet potato.

Disease Cycles

Meloidogyne spp. are soilborne, and disease cycles are initiated primarily from eggs and juveniles surviving in the soil. Since fleshy roots of sweet potato are commonly infected and used as seed, they serve as an additional source of inoculum. Infective juveniles may migrate from infected seed roots to the fibrous roots of sprouts, which are then transplanted to the field.

Shortly after the eggs are deposited into the gelatinous matrix

of the egg mass by the female, they begin to differentiate into first-stage juveniles. The first-stage juvenile molts within the egg, to give rise to a second-stage juvenile, which hatches through a hole made in the end of the egg by repeated thrusts of the stylet. The second-stage juvenile (Fig. 43C) is the infective stage. After hatching, it may move randomly within the egg mass or soil until it comes near an infection court, where it may respond positively to stimuli from the root. There are three major infection courts or points of entry: the tips of young fibrous roots, ruptures where lateral fibrous roots emerge from fleshy roots, and cracks in fleshy roots.

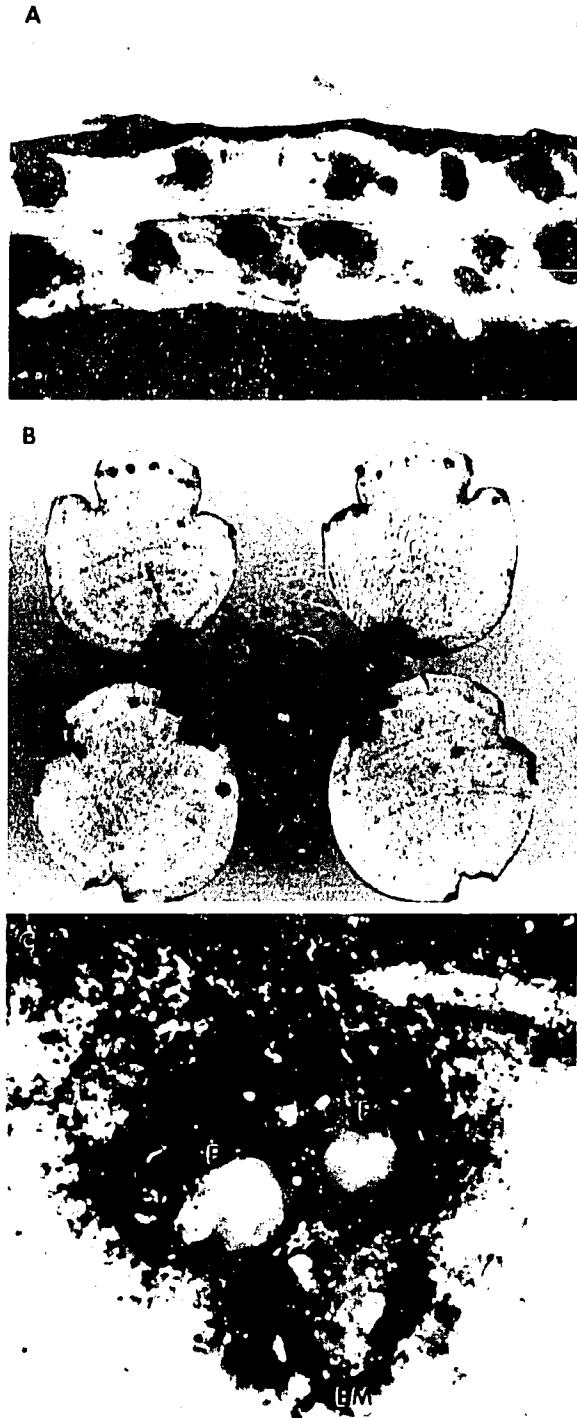


Fig. 45. Root-knot nematodes, *Meloidogyne* spp., within fleshy storage roots. **A**, Longitudinal section of the distal end of a storage root in which numerous females are embedded. **B**, Cross sections of fleshy storage roots with several embedded females. **C**, Light micrograph of an infected storage root, with two females (F) and an egg mass (EM). (A, courtesy G. Philley; B and C, courtesy G. W. Lawrence)



Fig. 44. Storage root with blisterlike bumps or protuberances caused by root-knot nematodes, *Meloidogyne* spp. (Courtesy G. Philley)

Once inside, the juveniles migrate intracellularly and intercellularly through the tissue of the root until they come to the eventual feeding site, where they become sedentary and initiate the formation of hypertrophied, multinucleate giant cells. The site of feeding depends in part on the location of the infection court, with giant cells being formed in vascular or cortical parenchyma or secondary cambial zones. As a developing nematode continues to feed at a site, the surrounding root tissues hypertrophy. The nematode enlarges, undergoes three additional molts, and differentiates into a mature female entirely embedded within the tissue of the root. The female deposits eggs into a mass connected to her posterior. Egg masses commonly contain 500–1,000 eggs and are usually deposited within fleshy roots and on the outer surface of fibrous roots. The generation time for *M. incognita* is estimated to be about 35 days.

Epidemiology

The damage to sweet potatoes by the root-knot nematode is directly related to the size of the initial population of the nematode. Thus factors that affect its survival in the soil between crops have an important bearing on the extent of damage, by influencing nematode densities at planting time. Winter temperatures influence the geographic distribution and the survival rate of *Meloidogyne* spp. *M. hapla*, the northern root-knot nematode, is restricted to areas that are generally cooler than those where sweet potato is commonly grown. On the other hand, damage due to *M. incognita*, the southern root-knot nematode, may be significantly reduced because of its inability to survive cold winter weather. Excess soil moisture also reduces nematode populations. The cropping sequence can also affect the initial population, since some species and cultivars of crops and weeds allow substantial population development and others do not.

Climatic conditions during crop development affect damage by imposing stress on the plants. Root knot is more severe in sandy-textured soils. Evidently this is related to the size of pore spaces and the greater mobility of the nematode in water in larger pore spaces. However, it may also be related to the ability of the root system to take up water and nutrients. Damage due to the nematode is more severe under conditions of moderate drought, because of the combined effects of the reduced availability of water and the reduced capacity of infected roots to take up water. When soil moisture is maintained at an adequate level for the growth of sweet potato during the growing season, the nematode may have less effect on the growth or yield of the crop.

Soil moisture is critical in determining the extent of the cracking of fleshy roots. There is evidence that fluctuations in soil moisture may induce growth cracks in sweet potatoes in the absence of the nematode, and cracking does not always occur, even when root-knot infestations are severe. Apparently, however, the nematode predisposes sweet potatoes to an increased incidence of growth cracks. The greatest incidence of cracking occurs when soil moisture is limiting early and abundant later in the growing season. This apparently causes a rapid centripetal enlargement of the fleshy root, and cracks develop at weak points in the root cortex, such as nematode infection sites.

Infected propagating material is an important source of inoculum in previously uninfested fields. The use of transplants grown from infected seed roots also contributes to the initial nematode population during the current growing season, but the amount of reproduction from that source alone is not thought sufficient to substantially limit yield or affect the quality of fleshy roots.

Populations of *M. incognita* in the soil around sweet potato plants decline during the first 4–8 weeks after planting, presumably because most larvae have penetrated roots but have not yet reproduced. Populations are maximal about 12–16 weeks after planting. In some years, populations on some cultivars may then decline prior to harvest, especially if the

fibrous roots become necrotic. Following harvest, detectable populations further decline. Normal sampling procedures, however, are inadequate to detect nematodes embedded within storage roots, eggs free in the soil, or egg masses released into the soil from fibrous roots.

Considerable genetic variability exists among populations of *M. incognita* associated with sweet potato. In particular, several populations are able to reproduce extremely well on cultivars considered resistant to root knot.

Interactions with other organisms also affect the extent of damage to sweet potato by the root-knot nematode. As mentioned above, root-knot infection may predispose fibrous and fleshy roots to decay by secondary invaders, presumably common soilborne fungi. *M. incognita* competes on sweet potato with the reniform nematode, *Rotylenchulus reniformis* (described below). These nematodes cause no more damage together than singly, but when either gains a competitive advantage (because of a higher initial population, more favorable soil conditions, etc.) it tends to dominate. The interaction of *M. incognita* with other nematodes commonly associated with sweet potato has not been studied.

Control

Before control procedures for root knot are undertaken, it is advisable to assay soil samples for the presence of *Meloidogyne* in the field in question, to determine whether there is a potential problem. This is best done at the end of the growing season, when nematode populations can be more precisely measured, to avoid fields known either from previous experience or from soil sampling to harbor a serious infestation.

Control procedures are primarily designed to reduce the initial nematode population. When this is impractical, other procedures may be useful. Hot-water treatments reduce nematode populations on or in seed roots and slips, but considerable damage to the plants may result, and the practice has not been widely adopted. The most successful programs for producing sweet potatoes in infested fields include a combination of the following practices:

1. *Crop rotation.* In some regions populations of *Meloidogyne* are significantly reduced by a cropping sequence including nonhost crops, such as winter wheat, followed by a root-knot-resistant cultivar of another crop, such as soybean. Many other sequences employing nonhosts and resistant cultivars are potentially useful.

2. *Selection of nematode-free propagating material.* Seed roots should be carefully selected to ensure that propagating material is free of the nematode, and they should be bedded only in uninfested soil. The use of uninfected transplants or vine cuttings for transplanting to the field is essential. Cutting vines or slips above the soil line greatly reduces the chances of transmitting the root-knot nematode as well as other soilborne pests.

3. *Nematicide treatment.* Treatment of soil in plant production beds or production fields can be effective in reducing existing populations of the nematode. Several approaches are effective: preplant fumigation of the soil with volatile nematicides, such as dichloropropene or methyl bromide; incorporation of nonfumigant nematicides, such as ethoprop or fenamiphos, into the soil; and, under experimental conditions, the use of systemic nematicides, such as oxamyl, in transplant water or for the treatment of seed roots or slips.

4. *Use of resistant cultivars.* Considerable effort has been expended to develop sweet potato cultivars resistant to root knot. A number of cultivars released in the United States with varying levels of resistance have proven useful in controlling the disease. Breeding lines have even greater levels of resistance than the currently available cultivars. However, resistant cultivars are not immune and may be severely affected in infestations with large nematode populations. Additionally, there is evidence to suggest that the use of resistant cultivars may select pathotypes that are more virulent on those cultivars, such as the "resistance-breaking races" of *M. incognita*. Some

breeding lines have a high degree of resistance to this pathotype as well as the common populations of *M. incognita*. Such resistance may eventually prove useful as a primary means of root-knot nematode control on sweet potato, but until that approach is more thoroughly proven, resistant cultivars should be regarded as only one component of an integrated control program. Crop rotation should reduce the danger of selecting new pathotypes.

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Reniform Nematode

The reniform nematode, a destructive pathogen of sweet potato, affecting both the yield and the quality of the crop, has been recognized as serious for only about 30 years. It is primarily tropical and subtropical, also occurring in some temperate areas, but is not distributed as widely as the root-knot nematode. It occurs in western and northern Africa, India, many island nations of the Caribbean region, the southeastern

United States, and several areas of the Pacific region, including Fiji, Hawaii, Japan, and the Philippines.

Symptoms and Signs

The reniform nematode may have profound effects on sweet potato, but the symptoms are not as distinctive as those caused by many other pathogens, and its effects have not always been recognized. The nematode infects the fibrous root system at any stage of development. It does not produce galls or other distinctive symptoms. The roots may be shorter and have fewer secondary roots when nematode densities are high, but when densities are low, root growth may actually be stimulated early in the season. Later in the growing season, the fibrous roots may become necrotic. Secondary symptoms resulting from damage to the fibrous root system include stunting of vines, yellowing of foliage, and transient wilting.

Fleshy storage roots produced in soil infested with the reniform nematode are often severely cracked and distorted. The cracks are predominantly of the deep, healed-over type (Plate 39), which develop early during the enlargement of the root, and thus the pattern of cracking differs slightly from that due to the root-knot nematode.

The only reliable method for diagnosing reniform nematode damage on sweet potato is to detect the presence of the nematode itself. It produces egg masses on the surface of infected fibrous roots, but they are difficult to see with the naked eye, since they are smaller than those of the root-knot nematode, and soil has a greater tendency to adhere to them. The reniform nematode is best observed in the fibrous root system by clearing and staining roots suspected of harboring it and then inspecting them at a magnification of 10-50X. The kidney-shaped body of the female can be seen outside the roots, with the head embedded near the stele (Fig. 46). Egg masses with adhering soil may envelop the posterior of the female. Juveniles (Fig. 47) are usually present in the soil surrounding infected plants and may reach densities as high as 100,000 juveniles per 500 cm³ of soil. The nematode has not been reported within enlarged fleshy roots of sweet potato, but it has



Fig. 46. Light micrograph of an immature female reniform nematode, *Rotylenchulus reniformis*. (Courtesy C. A. Clark)

been observed in small storage roots prior to significant centripetal enlargement.

Causal Organism and Disease Cycles

The reniform nematode, *Rotylenchulus reniformis* Linford & Oliveira 1940, is an obligate, sedentary semiendoparasite with an extensive range of dicotyledonous hosts. The species includes populations that are bisexual and amphimictic and populations that are parthenogenetic and lacking males. Variation within the species has not been extensively studied, but there may be one or more biotypes or races that infect certain monocots, such as day lily or sugarcane.

Juveniles of the reniform nematode are differentiated within the egg and undergo one molt before the second-stage juveniles hatch. Three additional molts occur without feeding while the juveniles are free in the soil. Only the females feed on plant roots. The females complete differentiation and maturation after assuming a sedentary position within the root and may be fertilized by the males, which remain in the soil. Eggs are deposited in gelatinous matrices around the posterior of the female, which contain approximately 50–100 eggs. The generation time is estimated to be 18–29 days.

When the females penetrate sweet potato roots, they pass through the cortex and feed in the endodermis. A single endodermal cell is converted to a giant cell, and pericycle cells in a sheath immediately adjacent to it also become hypertrophied. Phloem and cortical parenchyma cells next to the infection site also become enlarged, and the cambium and xylem vessels appear pressed together. Most of the female's body protrudes from the root.

Between crops, the reniform nematode survives primarily in the soil, where very high population densities (50,000–100,000 cm⁻¹) commonly occur. It has also been shown to withstand desiccation to a greater extent than many plant-parasitic soilborne nematodes, including *Meloidogyne* spp. Because it has not been found within fleshy storage roots, the possibility that it may be carried on or within seed roots has not been investigated. In this regard, two possibilities appear worthy of future investigations: whether the nematode survives in the soil adhering to seed roots, or whether it is carried in association with infected fibrous roots attached to seed roots. It can also be carried to the field in infected feeder roots on slips produced in infested beds.

Epidemiology

Damage by the reniform nematode is greatest when population densities are high at planting time and increase during the growing season. The focus of epidemiology and control studies

of the nematode concerns factors affecting the initial population at planting.

The tropical and subtropical distribution of *R. reniformis* is due largely to its inability to survive cold winters. Survival within its range is also affected by the extremes of winter temperatures.

Unlike the root-knot nematode, which is favored by sandy-textured soils, the reniform nematode is better adapted to somewhat finer-textured soils. However, the damage to sweet potato, like the damage from the root-knot nematode, is enhanced by moderate drought stress during the growing season, which occurs more frequently in sandier soils. The reniform and root-knot nematodes have a competitive interaction on sweet potato, causing essentially the same damage whether in combination or alone. However, the reniform nematode has been observed in many surveys to eventually predominate.

Control

The use of a fallow period has been suggested as a means to reduce populations of *R. reniformis* in infested fields. However, the length and nature of management of the fallow period could be critical, since the nematode can occur in unusually high numbers and is capable of surviving many adversities.

Hot-water treatment eliminates the reniform nematode on infected slips but has not been widely adopted in commercial culture of sweet potato, because of the reduced vigor of the slips and the difficulty of maintaining uniform treatment of a large number of plants.

Cultivars and breeding lines of sweet potato have been evaluated for their reaction to the reniform nematode in efforts to identify sources of resistance. Unfortunately, although differences occur in the extent of nematode reproduction on some selections, many of those that support the least reproduction are apparently hypersensitive and are the most seriously damaged under field conditions. Useful resistance has not yet been found in sweet potato.

Before initiating a management program, it is advisable to assay soil samples to determine if potentially damaging nematode infestations are present. Fields in which severe infestations are identified should be avoided. Control of the reniform nematode on sweet potato is more difficult than control of the root-knot nematode, because of the lack of resistant cultivars, and because population densities are often higher. It thus requires adherence to an integrated program of management practices including the following:

1. *Nematicide treatment.* Treatment of infested soil with nematicides is relied on more extensively with the reniform nematode than with the root-knot nematode, because there are fewer suitable alternatives. Several nematicide treatments are effective in reducing reniform nematode populations to 75–95% of the initial level, but the surviving populations may still cause damage. Several methods of treatment have proven effective: preplant fumigation of soil with volatile nematicides, such as dichloropropene or methyl bromide; incorporation of nonfumigant nematicides, such as ethoprop or fenamiphos, into the soil; and, under experimental conditions, the use of systemic nematicides, such as oxamyl, in transplant water or for treatment of slips.

2. *Use of nematode-free slips.* To avoid introducing the nematode into previously uninfested fields, and to reduce the initial population at transplanting, only transplants free of the nematode should be used. This can be accomplished by using vine cuttings or slips cut above the soil line rather than pulled slips for transplanting. Bedding seed roots only in nematode-free soil or treating beds with nematicides is also advisable, since the nematode might also be carried in soil with the transplants.

3. *Crop rotation.* The effect of cropping sequence has not been studied as extensively with the reniform nematode as with the root-knot nematode but should be considered a potential control tactic. The use of nonhost crops (such as some of the

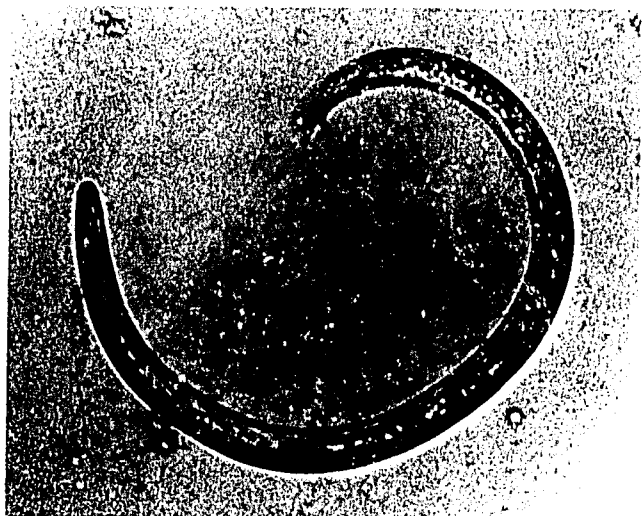


Fig. 47. Juvenile of *Rotylenchulus reniformis*. (Courtesy C. A. Clark)

graminaceous crops), resistant cultivars of crops such as soybean or cotton, or clean fallow should help reduce reniform nematode populations.

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Lesion Nematode

The lesion nematode, or meadow nematode, *Pratylenchus* spp., causes a disease of sweet potato known in some regions as root lesion or nematic root rot. Various species of *Pratylenchus* have been associated with sweet potato. In Japan the principal species is *P. coffeae* (Zimmerman 1898) Goodey 1951. In the United States *P. brachyurus* (Godfrey 1929) Filipjev & Schuurmans Stekhoven 1941 is the most common. In the United States *Pratylenchus* is not often associated with sweet potato, and greenhouse tests indicate that the nematode does not reproduce well on it. However, cultivars relatively susceptible to *P. coffeae* were grown in Japan during the 1950s. These cultivars supported increased multiplication of the

nematode in the field. Greater emphasis has been placed on the study of *P. coffeae* than on other *Pratylenchus* spp., since it has been considered to have caused significant losses of sweet potatoes in Japan, especially in the volcanic ash soils of southern Kyushu and Nagasaki. Breeding programs in Japan include screening for resistance to this pathogen.

Symptoms and Signs

When sweet potato is planted in infested fields, the nematode typically causes small, necrotic root lesions (Fig. 48A), from which its common name is derived. Nematodes in various stages can be viewed within roots or teased out of them (Fig. 48B). Fibrous root necrosis may lead to some stunting of vines and a significant reduction in the quality of fleshy storage roots. Secondary fungi and bacteria may invade lesions incited by the nematode and increase the extent of necrosis or decay. Small, brown to black, necrotic lesions are also produced on storage roots, which make the roots unmarketable (Plate 40).

Causal Organism and Disease Cycles

The lesion nematode is a migratory endoparasite. Juveniles and young adults enter roots and then continue to move intercellularly and intracellularly through the cortex, feeding on many different parenchyma cells within the colony or nest area. The root cells fed upon by the nematode become brown, granular, and necrotic. *P. coffeae* first enters sweet potato fibrous roots in or above the root-hair zone. The juveniles mature and differentiate within the root, and adult females deposit eggs singly or in small groups within the tissues of the root. The nematodes may leave the roots as secondary bacteria and fungi invade the lesions. The generation time for *P. coffeae* in sweet potato varies from 30-40 days at 25-30°C to 50-60 days at 20°C. Two generations may develop on weeds or other crops prior to planting sweet potato, and three may develop during the growing season for sweet potato in southern Kyushu, Japan. Generally, populations are highest on sweet potato just



Fig. 48. A, Hill of sweet potatoes infected with the root-lesion nematode *Pratylenchus coffeae*. B, Root-lesion nematodes teased from infected roots. (Courtesy T. Nishizawa)

prior to harvest and are lowest during the middle of the growing season and midwinter. Any stage of the nematode can overwinter, but adults rarely survive.

Epidemiology and Control

Generally, *Pratylenchus* spp. reproduce to a greater extent on monocots than on dicots. However, *P. coffeae* has an extensive host range that includes many dicots. Problems with root lesion are often greater when sweet potato crops follow monocots such as upland rice, wheat, or maize or dicots such as sweet potato or soybean. Conversely, peanut crops result in significantly lower populations of *P. coffeae*.

The severity of damage due to root lesion is favored by soil temperatures of 25–30°C, soil moisture content of 60–80%, sandy soil, and excess nitrogen fertilization. Severity is lower following the cultivation of paddy rice or increased fertilization with potassium or manure.

P. coffeae can also be controlled by the use of resistant cultivars and fumigants.

Selected References

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Brown Ring

Brown ring is usually attributed to the stem and bulb nematode, *Ditylenchus dipsaci* (Kühn 1857) Filipjev 1936. However, recent taxonomic studies suggest that in China *D. destructor* Thorne 1945 may be the dominant pathogenic species on sweet potato. The disease was reported to cause a serious loss of sweet potatoes in storage in New Jersey in the 1930s, but it has not been reported as a serious problem since then.

The disease was first discovered when seed roots failed to sprout. Symptoms initially consist of scattered sunken areas on storage roots. When they are cut open, a brown to brownish black layer is revealed in the cortex beneath the periderm. The nematode is confined to the affected cortical tissue. Eventually, the entire root becomes decayed, and nematodes and mycelia of secondary fungal invaders spread throughout it. The periderm then shrinks and becomes crinkled. The disease is primarily a storage disorder rather than a field disease, and the nematode is not capable of infecting growing vines.

The development of brown ring is favored by temperatures of 22–27°C, which are higher than those recommended for storage of sweet potatoes (13–19°C). Additionally, cultivars vary in their susceptibility to the nematode, with the Jersey types being more susceptible than others. The failure of the disease to recur since the initial report may be due in part to the use of less susceptible cultivars and to storage of sweet potatoes at recommended temperatures. The disease can be reduced by thorough weed control and by selection of seed roots free of the pathogen.

Selected References

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Spiral Nematode

The spiral nematode, *Helicotylenchus* spp., has been found in association with sweet potato in the field more frequently than any of the other genera of ectoparasitic nematodes. *H. dihystra* (Cobb 1893) Sher 1961 is most frequently associated with sweet potato. This species is a resident of soils throughout the world and has an almost unlimited host range. It reproduces rapidly on sweet potato in the field.

However, when the spiral nematode is the primary plant-parasitic nematode associated with the crop, nematicide treatments have failed to significantly affect yields or quality of sweet potatoes. The only report on the effect of different population densities of *Helicotylenchus* spp. indicated that low densities stimulated root growth but higher densities had no effect on the growth of fibrous roots or the weight of fleshy storage roots. It appears that the spiral nematode is not a serious pathogen of sweet potato, despite its frequent association with the crop.

Selected Reference

- Lopez, E. A., Gapasin, R. M., and Palomar, M. R. 1981. Effects of different levels of *Helicotylenchus* nematode infestation on the growth and yield of sweet potato. Ann. Trop. Res. 3:275-280.

Sting Nematode

A sting nematode, *Belonolaimus longicaudatus* Rau 1958, causes significant stunting of sweet potato vines, some of which may die prematurely, and lower yields of storage roots. The etiology has been confirmed by greenhouse inoculation studies and by controlling damage with nematicides in the field. Although the damage can be serious in infested fields, the distribution of the nematode is restricted to a very limited acreage of very sandy soils in the southeastern United States, and thus it is only occasionally encountered on sweet potato.

Severely affected fibrous root systems may appear similar to those damaged by *Trichodorus* spp. and *Paratrichodorus* spp. (see Stubby-Root Nematodes). The feeder roots are short and often swollen just behind the root tips. New roots may proliferate above feeding sites. Minute, discolored, shrunken lesions may develop at feeding sites.

The nematode resides in the soil and can be successfully controlled with nematicides.

Selected Reference

- Graham, T. W., and Holdeman, Q. L. 1953. The sting nematode *Belonolaimus gracilis* Steiner: A parasite on cotton and other crops in South Carolina. Phytopathology 43:434-439.

Stubby-Root Nematodes

The stubby-root nematodes *Paratrichodorus christiei* (Allen 1957) Siddiqi 1974, *P. minor* (Colbran 1956) Siddiqi 1974, and *Trichodorus* spp. have been associated with sweet potatoes in several surveys. These nematodes are ectoparasites found in sandy soils in many regions of the world. Although they are most damaging on monocots, they have extensive host ranges that include many dicots. They normally feed near the tips of fibrous roots, causing a cessation of root elongation. Affected root systems are shortened and have fewer lateral roots, and the roots are swollen at the ends. There is usually no necrosis. It has been suggested that stubby-root nematodes reduce both growth and yield of sweet potato.

Populations of the nematodes in the soil can be reduced by

fumigation, and *P. minor* reproduces more on some cultivars than others. However, there has been insufficient study of these nematodes on sweet potato to reliably determine what effect different populations have on its growth, yield, and quality.

Selected Reference

Roberts, P. A., and Scheuerman, R. W. 1984. Field evaluation of sweet potato clones for reaction to root-knot and stubby root nematodes in California. *HortScience* 19:270-273.

Virus Diseases

The diseases caused by viruses are probably the most poorly understood of all the sweet potato diseases. Viruses have been isolated from virtually all sweet potatoes grown in the absence of a virus-indexing program. Many of the diseases of a suspected viral etiology have merely been shown to be insect- or graft-transmissible (or both). The word *virus* was often appended to a disease name prior to a thorough attempt at biologically or biochemically comparing the causal agent with those previously described, resulting in considerable synonymy in the literature on sweet potato viruses. More recently antisera and diagnostic host ranges have become available for some of the more common viruses and should be used in the future to confirm diagnoses.

Epidemiology and Control

The most common source of sweet potato viruses is infected planting material. Viruses are spread from one cropping cycle to the next by sprouts produced from diseased roots and by the practice of taking vine cuttings directly from the previous crop. Plants with severe or acute virus diseases can occasionally be detected visually and easily avoided. However, virus infections may not be confidently diagnosed by visual inspection at all stages of the production cycle.

Some of the viruses have insect vectors. Thus, to prevent the introduction of viruses into sweet potatoes raised as "healthy" (virus-indexed) plants, they should be grown in an area free of inoculum sources and isolated from commercial production. In general, efforts to control the spread of viruses by controlling the vectors have not been successful.

One method of control is to plant material that is free of known viruses. Such healthy plants may be selected or produced by meristem or shoot tip culture. It is sometimes also necessary to subject plants to chemotherapy or thermotherapy prior to in vitro culture, to obtain the required number of healthy plants. Plants obtained from in vitro culture must first be tested (indexed) to identify those that are free of the known viruses and then to ensure that they are horticulturally true to type. It is necessary to confirm the absence of known viruses with a biological or biochemical assay designed specifically for the virus of interest. This is always true for the certification of plants to meet quarantine regulations established to prevent the international movement of viruses.

The development of resistant or tolerant cultivars is the other most viable alternative for control. When breeding programs are conducted in areas where the pressure from virus diseases is high, sensitive genotypes tend to be discarded early in the selection process as offtypes, or because of their lack of vigor or poor performance. Care should be taken when cultivars are developed in one area for use in another geographic region or country, because of differences in local viruses or virus strains and their vectors. Two examples of cultivar improvement utilizing natural virus disease pressure are the introduction of tolerance into breeding material for internal cork disease in the United States and for the sweet potato virus disease complex in Nigeria.

Selected References

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Phytopathology 65:769-773.

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Sweet Potato Feathery Mottle Virus

Sweet potato feathery mottle virus (SPFMV) is found nearly everywhere sweet potatoes are grown. Many strains have been identified, and it has been referred to by many names in different parts of the world. Some of the synonyms include russet crack virus, sweet potato virus A, sweet potato ringspot virus, sweet potato leaf spot virus, and probably internal cork virus. The ubiquitous nature of SPFMV has hindered the identification of many other viruses whose presence has been indicated by preliminary tests. Current virus transmission procedures and common host ranges have made it difficult to obtain cultures of potentially new viruses not contaminated with SPFMV.

Symptoms

The range of symptoms associated with infection by SPFMV are as much influenced by host genotype and environment as by virus strain or isolate. Leaf symptoms may consist of the classic irregular chlorotic patterns (feathering) associated with the leaf midrib and faint to distinct chlorotic spots, which may or may not have purple-pigmented borders (Plate 41). The symptoms are mainly on older leaves. Electron micrographs of infected leaves may reveal cytoplasmic pinwheel inclusions characteristic of potyvirus infections (Fig. 49). In addition, many plants tend to have symptomless infections, depending on the growth stage, genotype, and degree of stress, or when grown in vitro.

Sweet potato genotypes sensitive to virus infections also exhibit external and internal root symptoms. The russet crack strain (SPFMV-RC) causes annular necrotic lesions, which girdle the fleshy and fibrous roots of Jersey-type sweet potatoes (Plate 42). The same strain only causes foliar symptoms on other genotypes. Internal cork is also thought to be caused by

FMV; however, many genotypes do not develop necrotic lesions inside the root (Plate 43). The severity of internal cork symptoms in roots increases in sensitive genotypes stored for extended periods of time above 25°C.

Causal Agent

SPFMV has many of the biological and biochemical properties of a potyvirus. However, the virion is approximately 350 nm long, or about 100 nm longer than a typical potyvirus. The virion contains a single, plus-sense strand of RNA with a molecular weight of approximately 3.7×10^6 , which is consistent with its length. The RNA is encapsidated in a

repeating monomeric polypeptide with a molecular weight of 3.8×10^5 . Pinwheel inclusions, formed by nonstructural viral proteins, have been observed in infected cells (Fig. 49).

The virus is aphid-transmitted in a nonpersistent manner. It is most reliably isolated from sweet potatoes by grafting to *Ipomoea setosa* (Plate 44). It can be mechanically transmitted to *I. nil* from symptomatic tissue in phosphate buffer containing 0.05M sodium diethyldithiocarbamate. Most strains infect a wide range of *Ipomoea* spp. Many strains cause local lesions on *Chenopodium amaranticolor* and *C. quinoa*. Some strains infect *Nicotiana benthamiana*, a good propagative host for purification. Some strains do not induce prominent symptoms until after two or more passages through *N. benthamiana*.

Control

Sensitive cultivars may suffer significant yield losses.

However, cultivars released in the United States during the past 20 years appear to have a high level of tolerance to U.S. strains of SPFMV. It should be assumed that most plants in commercial production are infected with the virus. In addition, virus-indexed plants may be rapidly inoculated by the aphid vector when they are grown near sources of inoculum, such as infected sweet potato plants and various wild *Ipomoea* spp.

The general virus control strategies cited above are appropriate for SPFMV.

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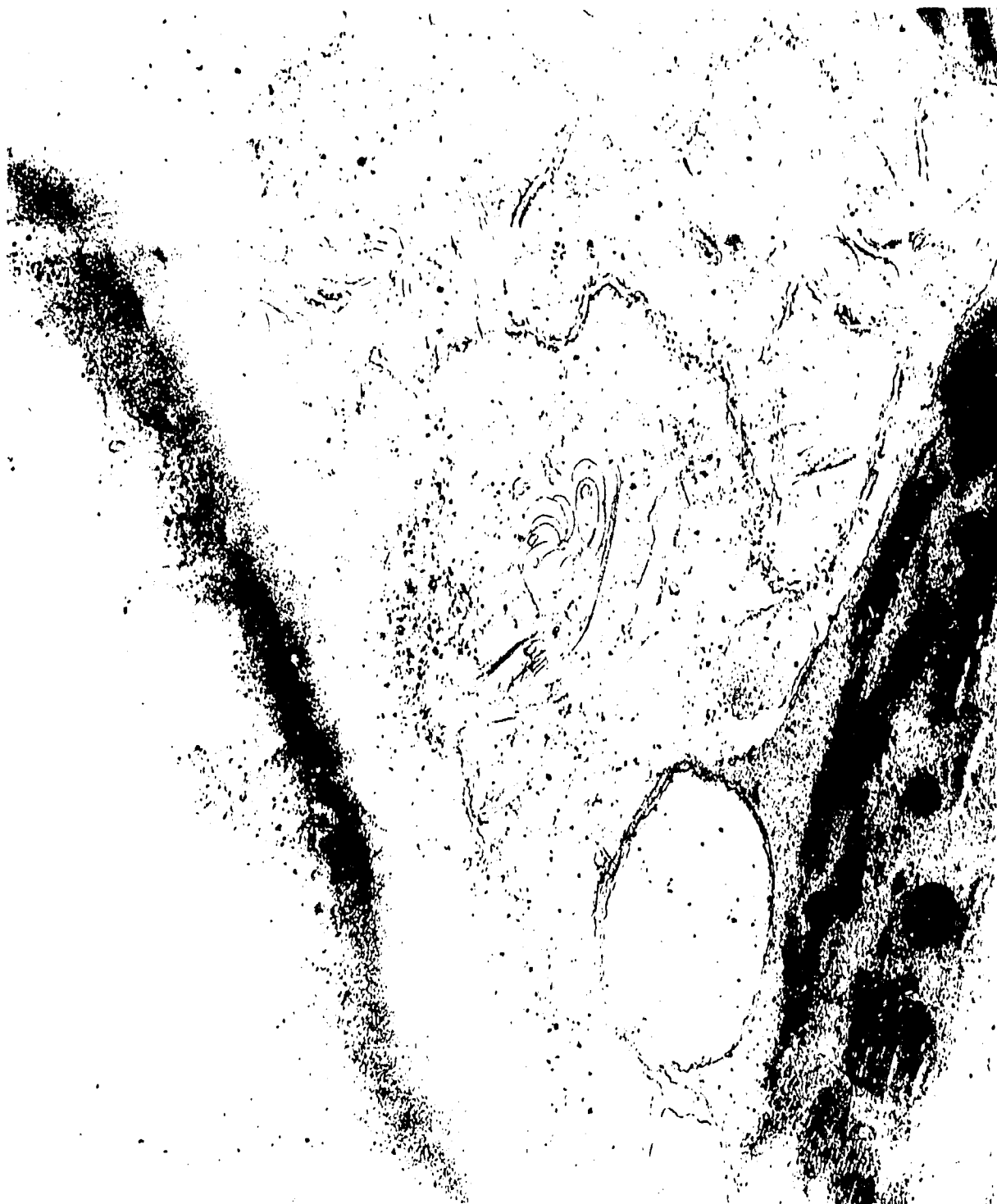


Fig. 49. Transmission electron micrograph of leaf tissue of *Ipomoea nil* cv. Scarlett O'Hara with cytoplasmic pinwheel and laminar inclusions associated with infection by sweet potato feathery mottle virus. (Courtesy Amanda Lawrence)

mottle virus. *Phytopathology* 71:302-305.

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- Yang, I. L. 1972. Transmission of the feathery mottle complex of sweet potato in Taiwan. *Taiwan Agric. Q.* 8:123-134.

Sweet Potato Vein Mosaic Virus

Sweet potato vein mosaic virus (SPVMV) has been reported only in Argentina. The common name for the disease is *batata crespa*. Direct comparison of the particle morphologies indicated that SPVMV, with a modal length of 761 nm, was significantly shorter than sweet potato feathery mottle virus. SPVMV is also nonpersistently transmitted by aphids. However, the virus has not been purified, and consequently antiserum is not available for comparison of this virus with other known potyviruses or for assays of sweet potatoes from other countries.

Symptoms

The leaves on infected plants exhibit a general chlorosis, with a diffuse mosaic in interveinal areas. They are often distorted with distinct vein clearing. The plants have shortened internodes, resulting in overall stunting. The stunting is also reflected in a reduction of the size and number of roots. No other symptoms have been reported on roots.

The known host range of SPVMV and strain C of sweet potato feathery mottle virus is limited to the Convolvulaceae.

Selected References

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- Nome, S. F., Shalla, T. A., and Petersen, L. J. 1974. Comparison of virus particles and intracellular inclusions associated with vein mosaic, feathery mottle, and russet crack diseases of sweet potato. *Phytopathol. Z.* 79:169-178.

Sweet Potato Latent Virus

Sweet potato latent virus (SPLV), initially designated sweet potato virus N, has been reported only in Taiwan. The host range includes many species in the Convolvulaceae, *Chenopodium* spp., and some *Nicotiana* spp., such as *N. benthamiana*. As the name suggests, many sweet potato cultivars infected by SPLV do not have obvious foliar symptoms.

Causal Agent

The virus is a long, flexuous rod, approximately 700-750 nm in length, and has a capsid protein with a molecular weight of 3.6×10^4 . Inclusion proteins typical of potyviruses have also been associated with infections by SPLV. However, all attempts at aphid transmission and whitefly transmission have been unsuccessful. Reciprocal serological tests have demonstrated that SPLV and sweet potato feathery mottle virus are not serologically related (J. W. Moyer, unpublished data). Thus, definitive classification of SPLV awaits further characterization.

Selected Reference

- Liao, C. H., Chien, I. C., Chung, M. L., Chiu, R. J., and Han, Y. H. 1979. A study of sweet potato virus disease in Taiwan. I. Yellow spot virus. *J. Agric. Res. China* 28:127-138.

Sweet Potato Mild Mottle Virus

Sweet potato mild mottle virus (SPMMV) was isolated in East Africa from sweet potatoes exhibiting leaf mottling, dwarfing, and poor growth. It was referred to as SPV-T in preliminary reports and closely resembles virus B, also isolated from sweet potatoes in that region of Africa. SPMMV is the most thoroughly described of the whitefly-transmitted agents reported affecting sweet potatoes.

Symptoms

A series of sweet potato clones mechanically inoculated with SPMMV expressed a range of symptoms as well as susceptibility to infection (i.e., inoculation efficiency). Responses ranged from leaf mottling and stunting to symptomless infections in susceptible clones. However, none of the symptoms was diagnostic. In addition, several clones were not infected after repeated inoculations.

The host range of SPMMV has been demonstrated to include 45 species in 14 plant families. *Chenopodium quinoa* is a good local lesion host. Symptoms in *Ipomoea setosa* infected with SPMMV (Plate 45) are similar to those induced by sweet potato feathery mottle virus. SPMMV can be purified from *Nicotiana tabacum*, which exhibits chlorotic or necrotic local lesions followed by systemic vein clearing, netting, leaf mottle, and mosaic. Young leaves may show moderate to severe epinasty 3-5 weeks after inoculation. Other useful propagative hosts include *N. benthamiana*, *N. glutinosa*, and *N. clevelandii*. *N. benthamiana* is more sensitive to infection by SPMMV than to infection by sweet potato feathery mottle virus or sweet potato latent virus (Plate 46).

Causal Agent

Virions of SPMMV are flexuous rods approximately 950 nm long in the presence of 0.05 M MgCl and between 800 and 900 nm in 0.04 M ethylenediaminetetraacetic acid. The coat protein has a molecular weight of 3.8×10^4 . The nucleic acid has not yet been characterized. Pinwheel inclusions have been observed in infected cells. The virus is transmitted by the whitefly *Bemisia tabaci*, but not by several aphid species. The available biological and biochemical characteristics are somewhat conflicting for classification of SPMMV as a potyvirus. Further characterization is required for proper classification of this virus.

SPMMV has biological properties similar to those of sweet potato yellow dwarf virus, another whitefly-transmitted agent, isolated from sweet potatoes in Taiwan. Sweet potato yellow dwarf virus and other whitefly-transmitted agents, isolated from sweet potato in Nigeria, Israel, Taiwan, and the United States, are described in the following sections. They have one or more properties by which they differ from SPMMV; some are not mechanically transmitted, some have narrow host ranges, and some cause diseases in which no virions have been identified.

Selected References

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Sweet Potato Yellow Dwarf Virus

Sweet potato yellow dwarf virus (SPYDV) was recently described in Taiwan. Its characteristics are similar to those of sweet potato mild mottle virus, but neither virus is adequately characterized, nor have direct comparisons been made to determine their relationship.

Symptoms

Symptoms on sweet potato plants infected with SPYDV consist of mottling, chlorosis, and dwarfing. Expression of the symptoms was favored by poor soil fertility and low temperatures. The root systems of infected plants were poorly developed, and the fleshy roots were not marketable.

The disease is frequently observed in combination with sweet potato feathery mottle virus. Symptoms of the combination in *Ipomoea setosa*, in addition to general stunting, may include general chlorosis; small, distinct chlorotic spots; and veinal chlorosis.

The host range of SPYDV extends beyond the Convolvulaceae to include *Chenopodium* spp., *Gomphrena globosa*, *Sesamum orientale*, *Datura stramonium*, and *Cassia occidentalis*.

Causal Agent

SPYDV, like sweet potato mild mottle virus, is a long, flexuous rod; however, it is only 750 nm in length. The capsid protein has a molecular weight of 3.3×10^4 . The nucleic acid has not been characterized. The virus can be transmitted mechanically and by the whitefly *Bemisia tabaci*. In vitro properties indicate that SPYDV is present in higher concentrations and is more stable than most viruses with a flexuous-rod morphology. The dilution end point is between 10^{-6} and 10^{-7} , and the thermal inactivation point is between 85 and 90°C.

Selected Reference

- Chung, M. L., Hsu, Y. H., Chen, M. J., and Chiu, R. J. 1986. Virus diseases of sweet potato in Taiwan. Pages 84-90 in: Plant Virus Diseases of Horticultural Crops in the Tropics and Subtropics. IFRC Book Series, No. 33. Food and Fertilizer Technology Center for the Asian and Pacific Region, Taipei, Taiwan.

Diseases Caused by Other Whitefly-Transmitted Agents

Several diseases of sweet potato have been attributed, at least in part, to an agent transmitted by *Bemisia tabaci*. In each of these diseases, the agent transmitted by the whitefly has not been mechanically transmitted, nor has a virion been identified.

The sweet potato virus disease (SPVD), which was described in Nigeria, is one of the most thoroughly investigated. It is due to the synergistic interaction of a strain of sweet potato feathery mottle virus (SPFMV) and an unidentified whitefly-

transmitted agent. Symptoms caused by either agent alone are relatively mild or nonexistent in sweet potato and *Ipomoea setosa*. The SPVD syndrome in sweet potato consists of various combinations of leaf strapping, vein-clearing, puckering, and stunting. In the indexing clone TIB 8, developed at the International Institute of Tropical Agriculture, and the U.S. cultivar Porto Rico, the symptoms appear in young leaves 2-4 weeks after graft inoculation. The youngest leaves have a distinct chlorotic mottle and are fan-shaped, and the plants are stunted (Plate 47). *I. setosa* infected with SPFMV or the whitefly-transmitted agent alone exhibit mild or no symptoms, but doubly infected plants are severely stunted (Plate 48). Yield losses up to 80% have been reported in Nigeria. Tolerant clones have been developed by the International Institute of Tropical Agriculture. Jewel, the predominant cultivar grown in the United States, was also tolerant when evaluated for symptoms in greenhouse trials.

Diseases similar to SPVD, designated Georgia mosaic and yellow dwarf, have been reported in the United States. Sweet potato vein-clearing virus, reported in Israel, also induces similar symptoms. Agents associated with each of these diseases have components that are transmitted by the whitefly but not mechanically. In addition, the diseases occurred in areas where SPFMV or an SPFMV-like virus was also found. The whitefly-transmitted agent associated with these diseases likewise has not been identified.

Sweet potato leaf curl disease is also caused by an agent transmitted by *B. tabaci*. Short, rod-shaped particles found in the cytoplasm of phloem cells were associated with this disease. The causal agent has not been identified. Leaf crinkling and upward rolling or curling of the leaves are the characteristic symptoms (Plate 49). The symptoms were most prominent on young plants. Similar diseases have been described in Taiwan and in Japan.

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Caulimo-like Virus

A virus graft-transmitted from sweet potato, with some properties like those of caulimoviruses, has been provisionally designated sweet potato caulimovirus (SPCV). It was first isolated from sweet potatoes from Puerto Rico and has since been isolated from them in Madeira, New Zealand, Papua New Guinea, and the Solomon Islands.

Symptoms

Diagnostic symptoms have not been associated with sweet potatoes infected by this virus. Early symptoms on *Ipomoea setosa* include chlorotic flecks along the minor veins and interveinal chlorotic spots. These may develop into a general chlorosis resulting in wilting and premature death of the leaves.

Subsequent leaves develop symptoms characteristic of sweet potato feathery mottle in *I. setosa*.

Causal Agent

Virions associated with SPCV were typical of caulimoviruses. Some of the inclusions were similar to the fibrillar ring inclusions induced by geminiviruses. However, the SPCV inclusions were not observed in the nuclei of infected cells. Serological tests failed to reveal any serological affinities with selected caulimoviruses, and SPCV was not transmitted in a semipersistent manner by *Myzus persicae* or *Aphis gossypii*. Definitive classification of this virus will require elucidation of additional characteristics.

Selected Reference

Atkey, P. T., and Brunt, A. A. 1987. Electron microscopy of an isometric caulimo-like virus from sweet potato (*Ipomoea batatas*). J. Phytopathol. 118:370-376.

Other Viruslike Diseases of Unknown Etiology

Many disease syndromes typical of those with a viral etiology have been described on sweet potatoes. The name *mosaic* has been used to designate several of them. However, sweet potato mosaic disease, described in Taiwan, is an apparently distinct disease. It causes mottling, distortion, and rugosity of leaves and stunted or dwarfed plants. The symptoms are most prominent during the cooler months. The causal agent was graft-transmitted to *Ipomoea setosa*, but the disease could not be induced by reciprocal grafts. As a result of the use of tolerant or resistant cultivars, this disease is of little economic importance.

Chlorotic leaf distortion is another sweet potato disease of unknown etiology. It was recently described in Louisiana (C. A. Clark, unpublished data) and has been observed throughout the southeastern United States. The diagnostic symptom is a bright chlorosis of the young leaves on infected plants (Plate 50). Symptoms appear during bright sunny periods, go into remission during prolonged cloudy periods with low light intensity, and return after a few sunny days. Preliminary

investigations indicate that meristem tip culture can be used to obtain plants free of the disease agent.

Selected Reference

Chung, M. L., Hsu, Y. H., Chen, M. J., and Chiu, R. J. 1986. Virus diseases of sweet potato in Taiwan. Pages 84-90 in: Plant Virus Diseases of Horticultural Crops in the Tropics and Subtropics. FFTC Book Series, No. 33, Food and Fertilizer Technology Center for the Asian and Pacific Region, Taipei, Taiwan.

Other Viruses Isolated from Sweet Potato

At least three viruses isolated from sweet potato have broad host ranges and are associated with diseases of many other crops. In some instances, it was not possible to infect sweet potato with the pure virus isolate.

Tobacco mosaic virus has been isolated from sweet potatoes exhibiting chlorotic patterns. Infections by this virus have only been reported from the United States.

Cucumber mosaic virus has been isolated from sweet potato, first in the United States and more recently in Ghana (J. C. Thouvenel, personal communication) and Israel (G. Loebenstein and J. Cohen, personal communication).

A virus serologically related to tobacco streak virus has been isolated from sweet potatoes originating in Guatemala. Source plants exhibited a severe chlorosis and stunting (Plate 51). Sweet potato feathery mottle virus was also present in those plants.

Several *Ipomoea* spp. are susceptible to tobacco ringspot virus. However, it has not been recovered from sweet potatoes.

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Part II.

Noninfectious Disorders

Sweet potato may be injured by many commonly encountered environmental factors in the field or in storage. Although noninfectious injury differs from infectious disease in that it is not progressive, the symptoms of noninfectious disorders can easily be confused with those of many infectious diseases.



Fig. 50. Leaf distortion caused by a mutation. (Courtesy W. J. Martin)



Fig. 51. Leaf of the cultivar Centennial with variegation caused by a mutation. (Courtesy C. A. Clark)

Somatic Mutations

L. L. Harter, in 1926, described a single plant of sweet potato bearing both yellow- and red-skinned storage roots. When these roots were used for propagation, they bred true for yellow or red skin. Since then it has been recognized that sweet potato has an unusually high rate of somatic mutation, even as great as 20% in the cultivar Jewel. Generally, the incidence of mutations is higher in storage roots than in the rest of the plant, and propagation from vine cuttings rather than from storage roots appears to reduce the incidence of mutations. Reversion of mutations to the original type is negligible.

The most frequent mutations in sweet potato affect the color of storage root skin, storage root flesh, vines, petioles, or leaf veins; affect leaf shape (Fig. 50) or the pattern of leaf venation; and cause leaf variegation similar to that observed on many different plants (Fig. 51). Some mutations cause severe distortion of leaves (Fig. 50) similar to that associated with some virus diseases (see Sweet Potato Vein Mosaic Virus) or growth regulators (see Herbicide Injury). These abnormalities are not transmitted by grafting but are perpetuated by clonal propagation. Mutations may appear either as bud sports, in which the entire plant displays the altered trait, or as chimeras, in which only a portion of the tissue is altered. Root chimeras include periclinal chimeras, in which the entire periderm is changed without a change in the underlying tissue; mericlinal chimeras, in which only a portion of the periderm is changed (Plate 52); sectorial chimeras, in which a wedge-shaped section of the flesh is changed (Plate 53); and heteroclinal chimeras, in which scattered sections of the flesh are changed.

Early genetic improvements in sweet potato came from the selection of mutations to improved types. On the other hand, the high rate of mutation in commercial cultivars was an important consideration in the development of foundation seed programs in the United States. In addition to providing seed roots relatively free of disease, these programs involve rigorous selection to reduce the incidence of undesirable mutations.

Selected References

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- Miller, J. C. 1930. Further studies of mutations of the Porto Rico sweet potato. *Proc. Am. Soc. Hortic. Sci.* 33:460-465.

Disorders Caused by Adverse Environment in the Field

Environmental and cultural stresses during the growth of storage roots in the field can directly and indirectly have a negative influence on both the size and the number of roots. Poor root quality at harvest, associated with such characteristics as cracks, poor shape, premature sprouting, or altered culinary characteristics, may be due to unfavorable environmental conditions. Such conditions predispose storage roots to infection by weak pathogens. The precise cause of the abnormality is often difficult to determine.

There are significant interactions between genotypes and their environment, which complicate diagnosis. For example, some cultivars tend to produce spherical or knobby storage roots in compact soils or soils high in clay but might produce the preferred prolate roots in coarse, sandy soils. Other cultivars produce prolate roots in fine-textured soils but excessively elongated roots in coarse-textured soils.

Souring

Souring of harvestable roots is caused by their being in water-saturated soils for prolonged periods prior to harvest. Sweet potato roots sustain a high rate of metabolic activity. When soils are saturated with water, the exchange of oxygen and carbon dioxide is inhibited, and roots become asphyxiated. Ethanol and carbon dioxide accumulate in such roots. The result is a high percentage of roots that decay during curing, and surviving roots undergo a greater amount of shrinkage. Excessive soil moisture may also reduce quality factors such as carotenoid pigments, dry matter content, and baking quality.

Although most sweet potato genotypes are adversely affected by excessive soil moisture, some are more tolerant of wet soils, with less shrinkage and less decay during both short- and long-term storage. In comparisons of cultivars sensitive to flood damage and those tolerant to it, differences in pH, soluble potassium, and rates of accumulation of carbon dioxide have been correlated with differences in responses to excess water stress. An alternative hypothesis has received greater acceptance, namely, that tolerance to flood damage is due to less ethanol production. However, it remains to be demonstrated that the amount of ethanol present in roots of sensitive genotypes is sufficiently high to cause the degree of injury associated with flooding. In addition, the variation in ethanol among genotypes has not been significantly correlated with the amount of flooding damage. More recent evidence suggests that the genotypic differences may reflect differences in the ability of the storage root to metabolize ethanol during and immediately following flooding.

Selected References

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- Ton, C. S., and Hernandez, T. P. 1978. Wet soil stress effects on sweet potatoes. *J. Am. Soc. Hortic. Sci.* 103:600-603.

Water Blisters

Following extended periods of flooded soil, storage roots may develop small, raised bumps at the lenticels, sometimes

called water blisters. The bumps are white at first and turn brown to black after the roots are harvested. They often develop prior to souring (described above), which occurs if soil saturation persists. The condition is similar in symptomatology (and in the environmental factors that initiate it) to lenticel proliferation in Irish potato. Cultivars differ in their tendency to develop water blisters.

Intumescence

Intumescence, also known as tumefaction, is a proliferation of callus tissue on the surface of leaves of certain cultivars. Intumescences are white at first, granular, and less than 1-2 mm high (Plate 54). They usually form simultaneously and are scattered uniformly over the leaf surface, but sometimes they form preferentially over the major veins. They occur on either surface of the leaf but are more prevalent on the upper surface. Within a few days they turn brown and dry and collapse to a fraction of their original size, giving the leaf surface a rough, sandpapery appearance.

No pathogen has been associated with intumescence. It develops spontaneously under conditions of reduced transpiration, which may occur with high humidity and low light intensity. High temperatures may also be necessary. The disorder is noticed in the field most often early in the growing season when the weather first becomes warm and humid. It may develop more frequently in greenhouses than in the field, because of the high humidity common in greenhouses. Intumescence may be pronounced on plantlets growing in tissue culture, especially on leaf surfaces in contact with the walls of the culture vessel. It is not a threat to continued growth.

Storage Root Cracking

Storage roots can crack or split in the field, at harvest, or even during storage in response to many diverse factors. Growth cracks, which develop during root enlargement in the field, are probably the most frequently encountered. These are mostly longitudinal and are more common on large than on small storage roots. They vary in depth, depending on when they are initiated, and are superficially indistinguishable from cracks associated with the root-knot nematode (Plate 37) or the reniform nematode (Plate 39; see Nematode Diseases). Cracking can also originate from soil rot (pox) lesions (see Bacterial Diseases).

At harvest, growth cracks may be covered with periderm and appear partially healed. However, depending on the timing of harvest in relation to cracking, the tissue surrounding a crack may be necrotic, because of invasion by secondary decay microorganisms, or the crack may expose sound internal flesh tissue.

The incidence of growth cracking is highly variable. It appears to result when the subcortical tissue expands more rapidly than the overlying cortex and periderm. Although it has been suggested that boron deficiency or excessive nitrogen or lime can lead to cracking, results regarding the causal role of these factors are conflicting. A stronger association has been observed between fluctuations in soil moisture and cracking. For example, the incidence of cracking is high when prolonged periods of dry soil conditions are followed by continued wet soil conditions. In addition, low temperatures (12-16°C) during the period when storage roots are being set can also lead to

increased cracking. Storage roots produced in cool or dry soil often have thin cortices, which probably split more readily in response to increased pressure from underlying tissue. Cracking is more common when sweet potatoes are planted in the same field successively for several years. Cracking is probably a response to any number of interacting variables that induce increased pressure from within upon the cortical tissues of the storage root. Rapid changes in turgor pressure might be involved, but this has not been studied in sweet potato.

Healing of cracks on storage roots is favored by warm, moist soil and delayed by cool, dry soil.

Storage roots sometimes crack during digging, with a popping sound. These cracks are often referred to as air cracks. The phenomenon occurs when sweet potatoes are dug from warm, moist soil and exposed to much cooler air. Apparently, the cortex of the root contracts more rapidly than the inner tissue in response to the rapid drop in temperature upon exposure to the colder air.

Short longitudinal splits sometimes develop in roots during storage. These are usually shallower than growth cracks or air cracks and often occur near the end of the root. The cause of these splits is not known.

Selected References

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- Scott, L. E. 1949. Factors associated with cracking of sweet potatoes. *Trans. Peninsula Hortic. Soc.* 39:37-40.

Chilling and Freezing Injury in the Field

Sweet potatoes originated in the tropics, and warm days and nights are optimal for production. For maximal production, the mean daily temperature should be above 22°C. Sweet potato foliage is very sensitive to frost. Production may be significantly restricted if plants are subjected to cool weather and frost soon after planting. Conversely, frost at or near harvest may have a negligible effect on roots if soil temperatures remain above 10-12°C. Roots should never be left in the field overnight after digging and prior to storage and curing, even during warm weather. Lower temperatures significantly reduce root quality and storage life. Soil temperatures below 10°C may cause internal breakdown in storage roots (Plate 55), similar to the breakdown that occurs during storage (see Disorders Caused by Adverse Environment in Storage). Additional information is provided in the sections on chilling injury in storage and on storage diseases.

Skinning

The most frequently observed blemish on sweet potato storage roots in fresh marketing is skinning, or surface abrasion. Sweet potatoes are much more easily damaged by

abrasion than are many other vegetables. Sometimes the root periderm may come off under minimal finger pressure. When a root is first skinned, the symptoms and cause are obvious. However, the skinned area may change significantly during postharvest handling, especially under dry conditions. If the root is not properly cured immediately after harvest, the skinned area becomes dark and sunken and may be surrounded by a narrow brown border (Plate 56). The latter condition can easily be confused with symptoms of surface rot or *Fusarium* root rot (see Field and Storage Diseases). This is complicated by the fact that skinning also provides an avenue for the entrance of many storage rot pathogens, especially *Fusarium oxysporum* and *F. solani*. Thus, the incidence of surface rot and that of *Fusarium* root rot are greatly increased by skinning.

Skinning occurs during both harvest and the handling involved in packing and shipping sweet potatoes to market. Successful growers and shippers have adapted their operations to minimize this injury. Workers wear soft gloves and are encouraged to handle sweet potatoes like eggs. Gently placing storage roots directly into containers rather than throwing them into piles in the field or throwing them into containers is important.

Dry soil prior to and at harvest greatly increases the likelihood of skinning, because hard, dry clods cause greater injury than loose, crumbly soil, and because dry conditions alter the physiology of the storage root, making the periderm easier to rub off. Skinning is most likely when the periderm is thickest. When the soil is dry at harvest, growers either stop harvesting until more favorable conditions occur or irrigate before continuing the harvest. If the roots must be harvested, it is sometimes possible to "set" the skin by removing the vines several days before digging. When roots are badly skinned during harvest, they should be marketed immediately. Any degree of skinning can lead to an unattractive sweet potato; it is therefore vital to cure roots immediately after harvest, to prevent skinned areas from becoming discolored or sunken.

Skinning also occurs as sweet potatoes are washed and packed for marketing. This problem is not as great with roots that have been properly cured and stored for several weeks, because the skin is set on cured roots and is more resistant to skinning. When it is necessary to market sweet potatoes early in the storage period, it is sometimes possible to set the skin by lengthening the curing period and reducing the humidity for the last few days of curing.

Selected Reference

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Sunscald

Sunscald is rare on sweet potato plants growing in the field. Sweet potatoes are relatively resistant to the combined effects of heat and sunlight. In fact, they require relatively high heat for maximal growth. Injury on young plants exposed to strong sunlight and high temperatures (over 38-40°C) occurs mostly on the stem near the soil line. Sunscalded stems turn gray to brown and become constricted. On older plants, leaves become brown and desiccated, and growing points become necrotic and die.

In contrast to growing plants, storage roots left exposed to the sun after they are dug are commonly damaged by sunscald. Many harvesting systems involve removing storage roots from the soil and leaving them on the soil surface. This facilitates drying and removal of soil from the roots, which are picked up in a second step of the harvest operation. However, when roots are left in bright sun for as little as 30 min, serious sunscald can result. They become soft near the surface, and within a few days their exposed surfaces may turn purplish brown. The incidence of storage rots, especially *Rhizopus* soft rot, surface rot,

Fusarium root rot, and charcoal rot, is greatly increased when sunscalded roots are stored.

In some areas, sweet potatoes left in containers to dry for brief periods in the field are covered with vines to prevent sunscald of the roots.

Selected Reference

Miller, C. A., and Kimbrough, W. D. 1948. Effect of length of exposure to sun on keeping of sweet potatoes. Proc. Am. Soc. Hort. Sci. 52:322-324.

Lightning Damage

Lightning damage on sweet potato is dramatic, but the symptoms may be misleading if not observed soon after the lightning has struck. Plants quickly collapse and die in a circle around the strike (Fig. 52). Necrotic lesions may then develop on the vines and petioles of plants at the margin of the circle. These lesions resemble fungal infections, and many different saprophytic fungi, such as *Curvularia* spp., may be isolated from them. However, they do not enlarge beyond their initial size, and secondary lesions do not occur. The surviving plants eventually resume normal growth.

Herbicide Injury

by T. J. Monaco and C. E. Motsenbocker

Relatively few herbicides are registered for weed control in sweet potatoes, and, if applied properly, they seldom cause injury to the crop for which they are registered. Herbicide injury to this crop is usually caused by misuse of products; soil residues, or carryover of residual herbicides; spray or vapor drift; contamination of sprayers; contamination of fertilizers; and miscellaneous factors.

Misuse or misapplication of herbicides leading to crop damage involves overdosage, improper timing of application, and application of unregistered products. Since so few herbicides are registered for use in sweet potatoes, growers are tempted to try other products, often leading to damage. Some growers apply herbicide in a mixture with other pesticides, such as soil insecticides or nematocides (Plate 57). In rare instances these untested mixtures may have disastrous results on the formation and shape of roots.

Soil residues, or carryover, of herbicides used for weed control in corn and soybeans are a potential cause of damage to sweet potatoes grown in rotation with these crops. Herbicides in the s-triazine class (e.g., atrazine and simazine), used in corn, are relatively persistent and, in the case of overdosage or overlapping of spray patterns, can damage sweet potatoes grown the following season. Herbicides in the imidazolinone and sulfonylurea families, recently approved in soybeans, now pose a problem for all horticultural crops grown in rotation. If compounds such as imazaquin (Scepter) or chlorimuron (Classic) are used in soybeans, one or more growing seasons must pass before sweet potatoes are planted.

It is a common practice for tobacco growers to apply herbicides to more acreage than needed prior to transplanting their tobacco. In most instances this land is also suitable for sweet potato production, and on occasion sweet potatoes are planted in these areas after application of the tobacco herbicide, which often leads to crop damage.

Vapor drift is another cause of herbicide injury to crops. Volatile compounds such as 2,4-D and dicamba (Banvel), applied to adjacent agronomic crops, can release vapors injurious to sweet potatoes. The amount of vapor drift from these materials depends on the formulation (i.e., esters of 2,4-D are more volatile than salts), air and soil temperature, soil

moisture, and wind movement. In general, high temperatures, wet soil, and windy conditions increase the potential for drift from these materials.

Drift of spray droplets from the intended target to adjacent nontargeted areas is another method by which herbicides can injure sweet potatoes. This damage can be entirely prevented by proper application techniques. Herbicides should never be applied under extremely windy conditions. Relatively low operating pressures (less than 40 psi) and nozzles that produce coarse sprays (with few fine or small droplets) should be used, and the boom should be maintained at the proper height for the nozzle type. Aerial application of herbicides poses far more danger of drift than application with ground equipment.

Contaminated sprayer equipment is occasionally associated with herbicide damage to sweet potato. When contamination occurs, it is generally associated with "all-purpose" sprayers. This is particularly critical in the use of growth-regulator herbicides, such as 2,4-D or dicamba, or in a switch from atrazine for corn or imazaquin for soybeans to a pesticide for sensitive sweet potatoes. Contamination can be prevented by proper cleaning and rinsing.

Contamination of fertilizers by herbicides also can lead to crop damage. In very rare instances, this contamination occurs during manufacturing and causes widespread damage. More commonly it occurs when fertilizer is stored in close proximity to leaking herbicide containers or bags. Fertilizers and pesticides should never be held in common storage or be in close proximity.

Miscellaneous factors that affect the degree of injury include variation among cultivars in their tolerance for herbicides, adverse weather conditions, and the use of the wrong herbicide. When new cultivars of sweet potato are released, registered herbicides should be applied on a small scale to confirm tolerance. Weather conditions can markedly influence herbicide behavior and, in extreme cases, lead to a loss of selectivity or tolerance by the crop. Precautionary statements on the label should be checked carefully to avoid application during climatic conditions unfavorable for the performance of the product. One of the inexcusable causes of crop injury is the harmful application of compounds by growers who carelessly pick up the wrong herbicide container.

Diagnosis of herbicide injury to any crop is complex, and the injury can be confused with the effects of other stress factors, such as infectious diseases, air pollution, other pesticides, nutrient imbalances, and adverse weather conditions. However, some general characteristics are often associated with herbicide injury to crops. With overlapping of spray patterns or inadequate tank agitation, the injury may occur as bands or strips through a field. Injury from soil-applied compounds may correspond with changes in soil type and topography. Also, similar injury symptoms on susceptible weeds in or adjacent to a field can confirm that a herbicide was involved. Injury from spray or vapor drift is generally most severe in areas of the field close to the site of application.

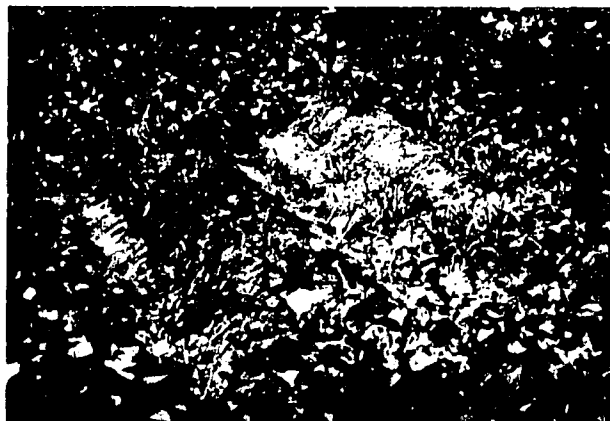


Fig. 52. Circular area in a field where plants were killed by lightning. (Courtesy C. A. Clark)

To further complicate diagnosis, the similarity of symptoms within herbicide families makes it extremely difficult to distinguish between injuries caused by different members of a family (Table 1). Additionally, a given herbicide can cause a

range of symptoms, and these vary with the dose and method of exposure.

Table 1 lists symptoms caused by various herbicides on sweet potatoes (see also Plates 58–68). Assessing the severity of

TABLE 1. Symptoms and Control of Herbicide Injury to Sweet Potatoes

Herbicide	Symptoms	Control	Remarks
Photosynthetic inhibitors <i>s</i> -Triazines Atrazine (AAtrex) Simazine (Princep) Cyanazine (Bladex) <i>as</i> -Triazines Metribuzin (Lexone, Sencor) Substituted ureas Diuron (Karmex) Linuron (Lorox) Substituted uracils Bromacil (Hyvar) Terbacil (Sinbar) Miscellaneous Bentazon (Basagran)	These compounds produce chlorosis (yellowing) of foliage followed by necrosis (browning) and eventually death of the plant. Soil uptake of <i>s</i> -triazines generally causes interveinal chlorosis initially. Soil uptake of ureas and uracils causes veinal chlorosis. Chlorosis can be followed by necrosis and dieback of the crop. Older leaves generally show the injury first. Foliar contact as the result of drift causes general chlorosis and rapid necrosis or blackening of leaves (Plates 58 and 59).	Observe rotational restrictions for these products. Avoid overdosage and apply uniformly. Do not spray these materials on adjacent crops in windy weather.	Carryover is common with <i>s</i> -triazines, ureas, and uracils. Persistence of these materials is enhanced by drought and cool conditions.
Pigment inhibitors Clomazone (Command) Norflurazon (Solicam) Amitrole (Amino Triazole)	These compounds cause a loss of chlorophyll, leading to bleaching, or whitening of foliage. Root uptake of norflurazon can cause complete bleaching of foliage. Foliar contact with amitrole causes complete bleaching of subsequently formed foliage. Sweet potatoes are not affected by clomazone.	Observe rotational restrictions with norflurazon. Avoid overdosage. Do not spray these materials in close proximity to sweet potatoes in windy weather.	Clomazone is the only pigment inhibitor ordinarily used on row crops near sweet potatoes. It is a volatile herbicide and causes nontarget effects due to vapor drift. However, sweet potatoes are very tolerant to it, and foliar or root contact does not cause any damage.
Growth regulators Phenoxys 2,4-D (numerous trade names) 2,4-DB (Butyrac, Butoxone) Benzoics Dicamba (Banvel) Chloramben (Amiben) Pyridines Picloram (Tordon) Triclopyr (Garlon)	Phenoxys, benzoics, and pyridines cause epinastic responses in sweet potatoes, especially from foliar contact. Initial symptoms are leaf petiole twisting followed by stem bending; leaf "feathering" and cupping; stem enlargement; misshapen roots; and, in some instances, death. Responses occur at very low dosages (in order of decreasing activity: pyridines, benzoics, phenoxys). Root uptake of phenoxys usually produces mild symptoms, whereas root uptake of benzoics and pyridines causes symptoms similar to those due to foliar contact. Chloramben, unlike other benzoics, causes mild symptoms, consisting of petiole bending (Plate 60), which is transient and does not affect quality or yield.	Avoid spraying phenoxys, benzoics, and pyridines on adjacent land in windy weather. Always use salt or amine formulations of 2,4-D in row crops. Do not apply these herbicides on land adjacent to sweet potatoes when extremely hot weather prevails.	Spray drift and vapor drift are the common causes of injury from these herbicides. Salt or amine formulations of 2,4-D are essentially nonvolatile and are the safest to use in adjacent row crops. Chloramben is registered for weed control in sweet potatoes in the United States.
Amino acids Glyphosate (Roundup) Sulfosate (Touchdown) Glufosinate (Ignite)	Glyphosate and sulfosate cause bright yellowing on new growth of sweet potatoes as the result of foliar contact. Malformation of new growth also occurs, followed by stunting and, in severe cases, death (Plate 61).	Avoid applying amino acid herbicides in adjacent areas in windy weather.	Spray drift is the major cause of injury to sweet potatoes. None of these herbicides has any activity as a soil residue.
Phenoxypropionates Diclofop (Hoelon) Fluazifop (Fusilade) Haloxyfop (Verdict) Fenoxaprop (Whip) Cyclohexanediones Sethoxydim (Poast) Clethodim (Select)	Sprays of phenoxypropionates and cyclohexanediones can result in foliar burning caused by adjuvants added to the spray mix.	Avoid applying phenoxypropionates and cyclohexanediones in hot and humid weather.	These materials rarely pose a threat to sweet potatoes when applied on adjacent crops. Direct application on sweet potatoes can result in contact injury, which is generally temporary. Fluazifop is registered for control of annual grasses in sweet potatoes in the United States.

(continued on next page)

damage on the basis of foliar symptoms can be misleading. For example, root development was adversely affected in the plant grown in treated soil in Plate 57, but vines in that field showed no abnormal growth or injury and were actually more vigorous

than those growing in untreated soil. Likewise, the off-shape roots shown in Plate 64 came from vines that were not severely damaged. It is essential to recognize that very few of the compounds listed in Table 1 are registered in the United States

Table 1. (continued)

Herbicide	Symptoms	Control	Remarks
Cell division inhibitors	These herbicides interfere with cell growth and development by interfering with mitosis. The primary symptoms are cessation of shoot and root growth, resulting in overall stunting of the plant.	Observe rotational restrictions, especially for the sulfonylureas and imidazolinones, used on soybeans. Avoid overdosage of long residuals, such as the sulfonylureas, imidazolinones, and dinitroanilines, by applying uniformly and (when recommended) incorporating into the soil evenly.	Carryover of sulfonylureas and imidazolinones can pose problems for all vegetable crops. Avoid land treated the prior year with the soybean herbicide imazaquin. Carefully follow replanting restrictions. Oryzalin and vernolate were registered for use in sweet potatoes in the United States but were withdrawn following injury to sweet potatoes in the form of misshapen roots and reduced yields. The injury from oryzalin was associated with unusually wet conditions.
Dinitroanilines Ethalfuralin (Sonalan) Oryzalin (Surflan) Trifluralin (Treflan) Pendimethalin (Prowl)	Dinitroanilines can cause root malformation, resulting in misshapen, unmarketable roots. Affected plants are stunted, and foliage may be dark green (Plates 62 and 63).		
Acetanilides Alachlor (Lasso) Metolachlor (Dual)	Acetanilides can cause stunting; crinkled or rough leaves; and misshapen, cracked roots. Thiocarbamates can cause stunting and misshapen roots (Plate 64).		
Thiocarbamates EPTC (Eptam) Butylate (Sutan) Vernolate (Vernam)			
Sulfonylureas Chlorimuron (Classic) Chlorsulfuron (Oust)	Sulfonylureas and imidazolinones cause stunting and general yellowing from soil uptake. Foliar contact causes yellowing and distortion of new growth, cessation of terminal growth, and a dwarfing effect (Plates 65 and 66).		
Imidazolinones Imazaquin (Scepter) Imazapyr (Arsenal) Imazethapyr (Pursuit)			
Acetamides Diphenamid (Enide) Napropamide (Devrinol)	Acetamides can cause slight stunting. These herbicides cause no significant injury on sweet potatoes.		Diphenamid is registered for weed control in sweet potatoes in the United States.
Miscellaneous DCPA (Daathal)	DCPA can cause stunting (Plate 67). The injury is usually temporary.		DCPA is registered for weed control in sweet potatoes in the United States.
Cell disruptors and cell membrane disruptors	Foliar contact can cause rapid necrosis and desiccation. These compounds are generally referred to as contact herbicides and produce what is known as a burning effect (Plate 68). They do not move much in plants; injury is only on treated plant tissue.	Avoid application on adjacent crops in windy weather. Use a drift control agent to minimize the possibility of off-target movement. Use low pressures and nozzles that produce coarse sprays to minimize drift.	Spray drift is the cause of injury to sweet potatoes from these herbicides.
Bipyridiliums Paraquat (Gramoxone) Diquat (Diquat)	Paraquat and diquat produce symptoms within hours, more rapidly than the others.		
Diphenyl ethers Acifluorfen (Blazer) Oxyfluorfen (Goal) Fomesafen (Reflex) Lactofen (Cobra)	Diphenyl ethers produce rapid necrosis and also affect subsequent growth, causing leaf crinkle and distortion.		
Organic arsenicals MSMA (Bueno)	MSMA is similar to the bipyridiliums in that only contacted foliage is damaged, with little or no effect on subsequent growth.		

for weed control in sweet potatoes. Injury from these products results from misuse, misapplication, and other errors committed by applicators. Symptom descriptions are provided as an aid to diagnosis and are not intended to imply that damage from the proper use of these herbicides occurs with any frequency.

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Disorders Caused by Adverse Environment in Storage

Temperature extremes during storage are an important cause of losses following harvest. Hardcore and sprouting can be attributed directly to the occurrence of temperatures that are too low or too high, respectively. Other problems, such as an unusually high incidence of rotting, may be only indirectly linked to the storage environment. For example, a slightly elevated storage temperature (20–25°C) by itself may have little effect on the storage life of roots, but losses may increase significantly at 20–25°C when root-rotting organisms are present. Excessive heat during storage may also result in premature sprouting and the formation of internal cork.

Chilling Injury and Hardcore

Sweet potatoes are extremely sensitive to temperatures below 13°C during storage, even though their average freezing temperature (–1.7°C) is relatively low. Under optimal conditions, roots are cured for 5–7 days at 30°C and then stored at 15°C until they are consumed. The differential between damaging and optimal storage temperatures is slight. Symptoms of chilling injury in storage include an increase in the frequency of roots with decay, flavor changes, internal discoloration (Plate 69), poor sprout production by seed roots, and tissues that remain hard after cooking. The disorder in which roots remain hard after cooking is referred to as hardcore. Unlike sound roots, chilled storage roots do not exude latex when cut. The duration of chilling, as well as the actual temperature, is an important determinant of the extent of injury.

Hardcore is the most easily recognized disorder attributable to the exposure of sweet potato roots to low temperatures. The cold is thought to modify pectic substances in the middle lamella, so that the tissue remains rigid during cooking. This disorder significantly reduces the marketability of sweet potatoes. The treatment of hardcore emphasizes prevention, since affected roots cannot be detected prior to sale and cooking.

Hardcore develops in roots exposed to 1.5°C for as little as 1 day or 10°C for at least 3 days. Cured roots are less sensitive to chilling (less likely to develop hardcore), and they exhibit a greater tendency to recover from the injury, especially if stored for several days at 21°C following exposure to chilling temperatures. Roots exposed to low temperatures late in the storage season are less likely to recover than roots exposed early in the growing season. There are also differences in the incidence and severity of hardcore among cultivars. Tests for

hardcore resistance should be made by chilling roots at 2°C for 7 days and then maintaining them near 24°C for 2 days. They should then be cooked and rated for incidence and severity of the disorder.

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Internal Breakdown (Pithiness)

Externally sound storage roots that have been stored for several months may feel spongy when squeezed and may become significantly lighter in weight. When such roots are cut open, the flesh has areas of white, dry, spongy tissue interspersed within the normal tissue. Parenchyma cells turn white (because of infiltration of the cells with air), desiccate, and



Fig. 53. Internal breakdown (pithiness) in a storage root stored for several months under warm, dry conditions. (Courtesy L. H. Rolston)

separate, creating large intercellular spaces or even large cavities scattered throughout the flesh. Long, continuous air channels may extend the length of the root, or the entire core of the root may become hollow (Fig. 53).

This internal breakdown, often referred to as pithiness, is usually associated with storage of roots in a warm, low-humidity environment for an extended period. However, the disorder can occur under any circumstances in which storage roots lose weight faster than volume. Although root volume can decrease during the first 2-3 weeks after harvest, especially during curing, it does not change greatly during long-term storage. Weight is lost in storage, both because of loss of water from the roots and because of respiration. Both kinds of loss are increased by wounding, insufficient curing, high temperatures (above 16-20°C), and low humidity during storage. Internal breakdown may also occur at harvest or during storage of roots exposed to low soil temperatures (5-10°C) prior to harvest (see Chilling and Freezing Injury in the Field).

Roots with internal breakdown usually also develop long sprouts during storage. Whether premature sprouting is a cause or an effect of internal breakdown or merely tends to occur under the same conditions as this disorder is not known.

At harvest, storage roots have a certain proportion of air-filled intercellular space, also referred to as internal gas content. The actual amount of space varies among cultivars, but it is apparently a stable trait for each cultivar and is relatively unaffected by environment during growth or in storage. The amount of intercellular space increases during storage in direct proportion to the loss of dry matter. The internal gas content can be determined by measuring the change in weight and volume of roots after vacuum infiltration while the roots are submerged in water. The increase in weight not accounted for by the increase in volume is assumed to be the weight of water filling voids in the tissue. Pithiness first appears when intercellular space exceeds 12% of the root volume.

Increases in intercellular space have been associated with

decreases in dry matter content but not with loss of water during storage. Cultivars also differ in their rates of respiration, weight loss, volume loss, and water loss. When these differences are imposed on the differences in initial internal gas content at harvest, an extreme range in the tendency to develop pithiness in storage is observed among cultivars.

Proper control of curing and the storage environment, coupled with the selection of cultivars that store well, minimizes internal breakdown. Curing roots at 29°C and 80-90% relative humidity, to promote the rapid formation of wound periderm, reduces both decay and water loss during storage. However, recommended curing conditions also favor high respiratory activity and thus loss of dry matter. Therefore, curing should not be extended any longer than is necessary for wound periderm to form and for the skin to become resistant to skinning (see Disorders Caused by Adverse Environment in the Field), because intercellular space continues to increase as curing continues. After curing, roots should be stored at 15-16°C and 80-90% relative humidity.

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Nutrient Deficiencies and Toxicities

The soils used to grow sweet potatoes are often of relatively low native fertility or subject to rapid losses of some nutrients by leaching. Thus nutrient deficiencies are encountered frequently. Toxicities are also possible in very acidic soils, due to an excessive availability of certain elements, such as aluminum and manganese.

Symptoms of some nutrient imbalances are similar in gross appearance to symptoms caused by some viruses (see Virus Diseases). Also, competitive uptake of cations can cause confusing patterns of nutrient imbalance symptoms. For example, high concentrations of potassium can reduce the uptake of calcium or magnesium, and in extreme cases excess potassium may cause symptoms of magnesium deficiency. Conversely, high levels of calcium, magnesium, or sodium may reduce the uptake of potassium, and high concentrations of sodium can induce deficiencies of calcium, magnesium, and potassium. As a consequence, symptomatology alone is not reliable for diagnosis of nutrient imbalances. Tissue analysis is essential for obtaining an accurate determination of which minerals are truly deficient or present in toxic quantities. The implications of competitive uptake should also be kept in mind in the selection of fertilizer formulations, as certain carriers may affect the uptake of other elements.

Some general precautions should be considered when corrective measures for nutrient imbalances are being developed. Lime is often used in acid soils to improve overall nutrient availability and plant growth. However, sweet potatoes are subject to serious damage by *Streptomyces*

ipomoea when the soil pH is above approximately 5.2 (see Soil Rot [Pox]). The damage caused by this pathogen generally far exceeds the potential benefits of liming. Liming is not recommended where *S. ipomoea* occurs. Fertilizer recommendations can only be relied on when based on knowledge of the local soil and the sweet potato cultivars being grown. For this reason, recommendations on fertilization are not included in this compendium.

There is relatively little published information on the effects on sweet potatoes of excesses of individual salts. It appears that the major required cations—calcium, potassium, magnesium, and sodium—may vary in the severity of the effect they induce, but they produce similar symptoms. The symptoms generally include chlorosis of terminal leaves followed by necrosis, stunting of the vine, abscission of older leaves, and in severe cases death of the plant.

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Nitrogen

Sweet potatoes usually require moderate amounts of nitrogen in the presence of other macronutrients and micronutrients for good vine growth and optimum yield of roots of marketable size and shape. When sweet potato plants are nitrogen-deficient, the leaves become uniformly light green, and vine growth is poor. Older leaves become red at the margins, and the laminae turn yellow, then develop a reddish tint, and eventually turn brown. Vines on older plants may develop a reddish color. Petioles are shorter than normal. Symptoms of nitrogen deficiency generally progress from the base of the plant toward the vine tips. Older leaves often abscise prematurely, but similar abscission of older leaves may also occur during rapid enlargement of storage roots of plants not under nutrient stress. Some root diseases may also induce premature leaf abscission. In the later stages of nitrogen deficiency, the younger leaves near the vine tip are small and either darker or lighter green, depending on the cultivar. Storage roots grown in a soil low or deficient in nitrogen may have abnormal skin color; e.g., white-skinned cultivars may develop tan skins. Fibrous roots are usually not visibly affected by nitrogen deficiency.

Excess available nitrogen may cause sweet potatoes to produce vine growth at the expense of storage roots, or flowering in breeding nurseries may be limited, despite the luxurious development of vines.

Phosphorus

Normally, sweet potato uses phosphorus efficiently and can extract sufficient amounts of it from soils well supplied with that element. However, phosphorus can be unavailable for plant use in some fine-textured (clay) soils. Leaves of sweet potatoes grown in phosphorus-deficient soil are dark bluish green with a purple cast over the veins on the abaxial surface and on the petioles. During later stages, both vine and leaf growth are restricted, and older leaves abscise. Storage roots are often small and irregularly shaped. Any purple pigmentation present in the storage roots becomes intensified by the deficiency.

Phosphorus in sufficient quantities increases the length of storage roots. When high concentrations of phosphorus are available, the thickness of the cambial zone may increase, and there is a corresponding increase in the number of secondary xylem vessels.

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Potassium

Sweet potatoes have a high demand for potassium, which is required for the enlargement of storage roots. The shape and solids content of storage roots are also affected by this mineral. Restricted vine growth, shortened internodes, and smaller leaves are the earliest symptoms of potassium deficiency. Leaf blades are darker green, especially at the margins, and petioles shortened and less pigmented. Small, shiny, brown patches may appear on the underside of leaves as a result of the rupture of cells beneath the epidermis. Interveinal chlorosis and bronzing occur on older leaves, beginning at the lamina tip and extending around the margins to the base. Chlorosis and bronzing may approach the midrib, and the leaves may become puckered. Severe deficiencies may result in general chlorosis of leaves, with a dark green band at the leaf base and along the secondary veins. The chlorotic areas eventually become brown and necrotic and may drop out, giving the leaf a tattered appearance. Very few storage roots form under conditions of extreme potassium deficiency, and those that do form are long and spindly. The deficiency has also been associated with greater weight loss and surface rot of roots in storage (see *Fusarium Root Rot and Surface Rot*). Increasing the availability of potassium tends to cause an increase in the carotene content of storage roots.

Potassium toxicity is not generally a problem with sweet potatoes. However, high concentrations of potassium can reduce the solids content of storage roots, which can lead to reduced dry matter content and reduced firmness in canned products.

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Calcium

Symptoms of calcium deficiency first appear well into the growing season, as a retardation of plant growth. Small chlorotic patches appear on the surfaces of leaves; these leaves eventually become necrotic and develop a leathery texture if the deficiency is not corrected. New leaves on calcium-deficient plants are light green and become reddish, while leaves near the base of the plant turn brown, and petioles develop little pigmentation. Thin lateral branches with small yellow leaves may then develop. As with many nutrient deficiencies, older leaves often abscise. Apical meristems may cease activity and finally die; this leads to the development of axillary buds, which subsequently follow the same pattern of deterioration. Fibrous roots produced by calcium-deficient plants are often soft and discolored; storage roots are soft, very small, and misshapen. The effect of calcium deficiency on the ability to produce storage roots varies, depending on the cultivar, from negligible effect to total failure to produce roots.

Magnesium

Symptoms of magnesium deficiency begin as an interveinal chlorosis progressing from the margins to the midrib of older

leaves. The veins remain dark green, but, since magnesium is highly mobile in the plant, the new leaves appear normal for a time after the onset of visible symptoms in older leaves. New stems are light blue-green. Later in the season, vine growth is retarded, internodes are shorter, and leaves are smaller. Young leaves curl up at the margins, and interveinal chlorosis eventually develops on both old and young leaves, sometimes accompanied by a pink cast. Chlorotic portions of leaves may become necrotic if the deficiency is not corrected. Generally, magnesium deficiency does not affect root enlargement.

Sulfur

In vine growth and root production, sweet potatoes grown in sulfur-deficient soils are often similar to those grown in sulfur-sufficient soils, and symptoms of the deficiency may be subtle. Leaves on deficient plants may become pale green to yellow. Veins may be bordered by green even after the interveinal areas become chlorotic. Symptoms appear on younger leaves before they appear on older leaves. Leaves may remain small, and older leaves may turn purple along the margins. Fibrous root development is extensive, and storage roots may become rounded.

Iron

Symptoms of iron deficiency can be dramatic. The lamina of young leaves may turn very pale yellow to almost white very early in leaf development. The veins themselves remain light green. Vines are markedly stunted, and plants may totally fail to produce storage roots. When storage roots are produced, their skin color may be altered.

Boron

Boron deficiency occurs most frequently in sweet potatoes grown in coarse-textured soils. Symptoms on vines and foliage first appear late in the growing season, when leaves become mottled at the margins. Terminal growth of the vines is distorted and restricted, and internodes are shorter. Marginal leaf burn and premature abscission of leaves and curling of petioles also occur. Stunting and distortion of the terminals may follow, similar to that caused by chlorotic leaf distortion (see Other Viruslike Diseases of Unknown Etiology).

Symptoms of boron deficiency in storage roots are diagnostic and have been referred to as internal brown spot and blister. Internal brown spots are similar in appearance to symptoms associated with internal cork (see Sweet Potato Feathery Mottle Virus). They may occur anywhere within the storage root but are most common near the vascular ring. The spots are brown, vary in size, and have indefinite margins. In some cultivars, blister symptoms also develop at the surface of the storage root. The blisters are purplish brown (Plate 70) and thus resemble scurf lesions (see Scurf); unlike scurf lesions, however, they are raised and are often (but not always) associated with internal brown spots. Blister symptoms may develop in storage even when symptoms are not evident at harvest.

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Manganese

The first symptom of manganese deficiency occurs on young sweet potato leaves, which turn pale yellow. Interveinal chlorosis then develops from the base to the apex of these leaves, but the veins remain dark green. Internodes are shorter and leaves smaller, but in some cases manganese-deficient plants may produce more top weight. Overall, the symptoms are similar to iron chlorosis but less severe. No pronounced effects on storage roots have been reported.

In certain acid soils, such as the Mississippi terrace soils of the southern United States, manganese toxicity may be an important limiting factor in sweet potato production. The most common symptom of excess manganese is interveinal chlorosis, which may occasionally be followed by necrosis. Symptoms appear early in the growing season and are most evident on fully expanded leaves, being less pronounced on both young expanding leaves and older leaves. Plants tend to recover from the initial symptoms and may be indistinguishable later in the season from plants growing with sufficient manganese. Symptomatic plants have greatly elevated levels of manganese in the leaves.

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Aluminum

Toxic concentrations of aluminum occur in soils with low pH, especially in the bauxitic soils of the tropics. As with many other crops, the toxic effects of aluminum generally appear in roots before any effect is evident on top growth. Fibrous roots become thickened and stunted and fail to branch normally. Fibrous roots of some sensitive sweet potato selections may turn brown when exposed to high levels of aluminum. Root tips and lateral roots are particularly sensitive. There is little indication in the literature of injury to leaves, petioles, or stems. However, leaf and petiole size and overall top weight are apparently reduced when sweet potatoes are grown in acid soils with high aluminum content. Cultivars vary in their tolerance to this mineral.

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Part III. Disorders of Unknown Etiology

Sweet potato is subject to several disorders for which a causal factor has not been clearly demonstrated. Two of them, distal end rot and false broomrape, have been described. There may be other disorders of unknown etiology that have been observed but not described; these are not discussed in this work.

Distal End Rot

Distal end rot is a disorder of unknown etiology that develops during curing or storage of storage roots. It was first reported to be peculiar to the cultivar Acadian, grown in Louisiana; it developed on up to 30% of the roots of Acadian. The decay develops slowly after harvest and is restricted to the distal end of the root, usually not affecting more than a third of the root (Plate 71). The symptoms are similar to the restricted lesions of Java black rot or charcoal rot. However, no causal organism has been isolated from affected roots.

Selected Reference

Martin, W. J. 1958. Distal-end rot of sweetpotatoes of the Acadian variety. Plant Dis. Rep. 42:983.

False Broomrape

False broomrape is a disorder that has been studied primarily on tobacco in the southeastern United States. It does not appear to cause significant loss on sweet potato but has been observed on sweet potato plants growing in infested potting soil. Subsequent artificial inoculations with ground tissue from tobacco tumors showed that some cultivars of sweet potato are susceptible to false broomrape. Tumors that resemble clusters of buds develop from underground nodes. Cultivars vary greatly in the extent of tumor development, with some producing tumors less than 3 g in fresh weight and others producing tumors in excess of 100 g. Transmission efficiency from sweet potato to sweet potato was very low; transmission to tobacco was much more efficient.

The causal agent of false broomrape has not been isolated or characterized. It persists for several years in infested soil and frozen tumor tissue. The agent has certain properties that suggest it may be a bacterium, such as sedimentation characteristics and an inability to pass through filters that retain bacteria. However, inoculations with plant-pathogenic bacteria known to produce hypertrophies, e.g., *Agrobacterium tumefaciens* and *Rhodococcus fascians* (syn. *Corynebacterium fascians*), failed to reproduce the symptoms.

Research not directly related to false broomrape has shown that certain plant-pathogenic bacteria not normally associated with sweet potato, e.g., *Pseudomonas syringae* pv. *phaseolicola*, can induce hypertrophy of the vascular and anomalous cambia of storage roots following artificial inoculation. These hypertrophies have not been directly compared to false broomrape.

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Fasciation

Vines of sweet potatoes sometimes develop fasciation in plant beds or in the field. This disorder is also known as flat fasciation, because affected vines become extremely wide but remain flat in cross section (Plate 72). In addition, a ring fasciation has been described, in which the stem is enlarged into a tubular shape. Flat fasciation is far more common than the ring form. Fasciated vines appear as if five to 10 normal vines were fused together. Elongation of the vine is restricted, but plants normally remain green and alive and continue to grow until harvest. The effect on the yield or quality of storage roots has not been determined. The disease is found in many fields, but the incidence is normally very low. Genotypes differ in their relative susceptibility to fasciation.

Subcutaneous Roots

In some regions, a small percentage of storage roots develop a network of dark brown lines just beneath the periderm, similar in size, shape, and arrangement to a fibrous root system (Plate 73). The cause and development of this symptom have not been extensively studied. Symptomatic storage roots are noticed most often after extended storage, but it may be that the symptoms develop earlier in the storage period. The cause of the disorder is unknown.

Alligator Skin

Occasionally, storage roots badly disfigured by a disorder affecting the periderm and cortex are discovered at harvest. The entire surface of the root has a pattern of purplish brown to black areas of corky tissue, separated by longitudinal grooves covered with normal periderm (Plate 74). The resulting pattern of bumps and grooves resembles the pattern on an alligator's skin. The internal tissue of the root is not affected. The occurrence of the disorder is erratic, and losses in some fields are occasionally large, but overall the losses are not significant. The cause of the disorder is not known.

Glossary

C—Celsius

cal—calorie

cm—centimeter (1 cm = 0.01 m = 0.3937 inch)

g—gram (1 g = 0.3527 ounce; 453.59 g = 1 pound)

ha—hectare (1 ha = 2.471 acres)

kg—kilogram (1 kg = 2.205 pounds)

L—liter (1 L = 1.057 quarts liquid [U.S.])

m—meter (1 m = 39.37 inches)

mg—milligram (1 mg = 0.001 g)

min—minute

mm—millimeter (1 mm = 0.001 m = 0.03937 inch)

μm—micrometer (1 μm = 10⁻⁶ m)

nm—nanometer (1 nm = 10⁻⁹ m)

psi—pounds per square inch

t—metric ton (1 t = 1.102 short tons = 2,205 pounds)

abaxial—directed away from the axis of a leaf; pertaining to the lower surface of leaves

abscission (v. abscise)—separation of flowers, fruit, or leaves from plants

acervulus (pl. acervuli)—saucer-shaped fungal fruiting structure bearing conidiophores and conidia

acid soil—soil with an acid reaction (pH less than 7)

actinomyce—any of the filamentous bacteria in the class Actinomycetes

acute—developing suddenly; severe, with reference to disease symptoms

adaxial—directed toward the axis of a leaf; pertaining to the upper surface of leaves

adventitious—arising or occurring sporadically or in an unusual place

aerospore—dehiscent, binucleate, dikaryotic spore produced in an aecium

aecium (pl. aecia)—the cup-shaped sorus of rust fungi, which produces chains of aeciospores

aerobic—requiring the presence of elemental oxygen for survival

alkaline soil—soil with a basic reaction (pH greater than 7)

amphimictic—capable of reproducing sexually and producing fertile offspring

anaerobic—living and surviving in the absence of elemental oxygen

anamorph—the asexual or imperfect thallus of a fungus; the binomial of a fungus in its asexual stage

anastomosed—fused together, with reference to hyphae joined together to form a network

antheridium (pl. antheridia)—male sexual organ in pythiaceae fungi

anthocyanin—blue, purple, red, or pink water-soluble flavonoid pigment in cell sap

antibody—a protein formed in the blood of a living animal in response to the injection of a foreign substance, such as a virus or viral protein

antiserum—blood serum containing antibodies, separated from the other components of the blood

aphid—any of the small, sucking, homopterous insects of the family Aphididae, living on plant juices, of which numerous species are capable of transmitting viruses

apical—pertaining to the apex, or tip

apterotic—bearing oospores that do not fill the oogonium, in pythiaceae fungi

appressorium (pl. appressoria)—swelling on a germ tube or hypha, especially for attachment to a host in an early stage of infection

ascigerous—bearing asci; pertaining to the phase in the life history of certain fungi that culminates in the formation of ascospores

ascocarp—the sexual fruiting body (ascus-bearing organ) of ascomycetes

ascomycete—any of the fungi in the class Ascomycetes, which produce

sexual spores (ascospores) within an ascus

ascospore—spore borne in an ascus by free cell formation

ascus (pl. asci)—sac-like or clavate structure containing ascospores (typically eight) and borne in an ascocarp

aseptate—without cell cross-walls

asexual—vegetative; without sexual organs, sex cells, or sexual spores, as in the imperfect stage of a fungus

avirulent—unable to cause disease

axillary—pertaining to or placed within an axil, the angle formed by a leaf petiole and the stem

bacterium (pl. bacteria)—typically, a single-celled microorganism lacking chlorophyll and increasing by simple cell division

basidiocarp—the sexual fruiting body of basidiomycetes

basidiomycete—any of the fungi in the class Basidiomycetes (including smut and rust fungi), characterized by septate mycelium, sometimes with clamp connections, and forming sexual spores (basidiospores) on a basidium

basidiospore—sexual spore produced on a basidium

basidium (pl. basidia)—short, club-shaped, haploid promycelium produced by basidiomycetes (including smut and rust fungi) and bearing basidiospores

biflagellate—having two flagella

biguttulate—having two globules or vacuoles

binucleate—having two nuclei

biotype—group of organisms belonging to the same species but differing in biochemical, physiological, or behavioral properties from other members of the species; group of individuals with like genetic makeup

blade—the flat or expanded part of a leaf or petal

blastic—pertaining to a type of conidium formation characterized by a marked enlargement of a recognizable conidial initial before the initial is delimited by a septum

blight—any sudden, severe, and extensive spotting, discoloration, wilting, or destruction of leaves, flowers, stems, or entire plants, usually affecting young, growing tissues

broadleaf, broad-leaved—pertaining to a plant (other than a grasslike plant) with a flat leaf

calyx—the outermost whorl of a flower; sepals, collectively

cambium (pl. cambia)—lateral meristem, occurring in the shape of a cylinder (vascular cambium) in sweet potato storage roots or in strips scattered through the root (anomalous cambia)

canker—necrotic, localized diseased area

capsid—protein coat of a virus particle

carbohydrate—food compound composed of carbon, hydrogen, and oxygen, with hydrogen and oxygen in the ratio of 2:1

cellulolytic—pertaining to an enzyme capable of dissolving cellulose, the fibrous substance that is the main component of plant cell walls

chimera—plant having several tissues or tissue layers that differ in genetic constitution

chlamydospore—thick-walled or double-walled, asexual resting spore formed from hyphal cells (terminal or intercalary) or by a transformation of conidial cells

chlorosis (adj. chlorotic)—failure of chlorophyll development, caused by disease or nutritional disturbance (e.g., lack of iron, zinc, or magnesium); fading of the green color of a plant to light green, yellow, or white

circulative—pertaining to viruses that accumulate within or pass through the gut of insect vectors before being transmitted to plants

clamp connection—hyphal protrusion formed at cell division and connecting the newly divided cells in basidiomycetous fungi

clavate—club-shaped, narrowing in the direction of the base

clone -- vegetatively (asexually) propagated plant derived from a single original plant or plant part
coenocytic -- aseptate, pertaining to mycelium with nuclei embedded in the cytoplasm without being separated by cross-walls
columella -- the central, sterile part of the sporangium in some fungi
concentric -- pertaining to circles with a common center but different diameters
conidiogenous -- producing and bearing conidia
conidiophore -- simple or branched, fertile hypha on which conidia are produced
conidium (pl. conidia) -- asexual spore borne at the tip or side of a specialized hypha (conidiophore)
corolla -- the petals of a flower, collectively
cortex (pl. cortices; adj. cortical) -- the tissue between the epidermis and the phloem in stems and roots; a thick outer covering
cotyledon -- leaf formed within a seed; primary embryonic leaf
cultivar -- cultivated variety
culture -- artificial growth and propagation of organisms on nutrient media or living plants
cytoplasm -- living protoplasm in a cell, excluding the nucleus

damping-off -- rapid, lethal decline of germinating seed or seedlings before or after emergence
dematiaceous -- pigmented darkly
desiccate -- to dry out
diagnostic -- pertaining to a distinguishing characteristic important for identification of a disease or some other condition
dicot (adj. dicotyledonous) -- plant having two cotyledons
dieback -- progressive death of shoots, leaves, or roots, beginning at the tips
dilution end point -- the point at which infectivity or other activity is lost because of dilution
diploid -- having a double set of chromosomes per cell
dissemination -- spread of infectious material (inoculum) from a diseased to a healthy plant
distal -- far from the point of attachment or origin
doliform -- barrel-shaped
dwarfing -- underdevelopment of a plant or plant organs, caused by disease, faulty nutrition, unfavorable environmental conditions, etc.

ectoparasite -- parasite living on the exterior of its host
ELISA -- see enzyme-linked immunosorbent assay
endoconidium -- conidium formed inside a hypha
endodermis -- the innermost tissue of the cortex
endoparasite -- parasite living inside its host
enzyme-linked immunosorbent assay -- an extremely sensitive serological test for virus antigens or other antigens
epidemic -- general and serious outbreak of disease (used loosely of plants)
epidemiology -- the study of factors influencing the initiation, development, and spread of infectious disease
epidermis -- the surface layer of cells of leaves and other soft parts of plants
epinasty -- downward curvature of a leaf, leaf part, or stem due to rapid expansion of the upper surface
eradicate -- to destroy or remove a pest or pathogen after disease has been established
erumpent -- bursting or erupting through the substrate surface
etiology -- the causes or origins of a disease or abnormality
exudate -- liquid (ooze) excreted or discharged from diseased tissues or from roots and leaves

facultative parasite -- saprophytic organism capable of parasitism
fasciation -- malformation of stems, buds, or other plant structures, in which affected parts appear to be fused together
fibrous -- containing, consisting of, or resembling fibers
fibrous root -- thin, heavily lignified root that functions in the uptake of water and nutrients
fibrovascular -- having or consisting of fibers and conducting cells (such as vessels)
filamentous -- threadlike
filiform -- threadlike
fimbria (pl. fimbriae) -- fringe or border of hairs or fibers
flagellate -- having a flagellum or flagella
flagellum (pl. flagella) -- hairlike, whiplike, or tinsel-like appendage of a motile cell (bacterium or zoospore), providing locomotion and similar to but longer than a cilium
flexuous -- having turns or bends, winding
foliar -- pertaining to leaves
forma specialis (pl. formae speciales) -- a taxonomic category within a

species, differentiated on physiological or pathological characteristics but not differentiable on morphological characters
fruiting body -- any of various complex, spore-bearing structures of fungi (e.g., apothecia, ascocarps, basidiocarps, perithecia, pycnidia)
fumigant -- vapor-active (volatile) disinfectant that kills microorganisms and other pests
fungicide -- a substance that kills fungi; sometimes, broadly, a substance that inhibits the growth of fungi or spore germination
fungus (pl. fungi) -- spore-producing plant lacking chlorophyll, often causing diseases of higher plants
fusiform -- spindle-shaped, narrowing toward the ends

gall -- abnormal swelling or localized outgrowth, often roughly spherical, produced by a plant as a result of attack by a fungus, bacterium, nematode, insect, or other organism
gametangium -- cell or organ that produces gametes (sex cells) or contains nuclei that act as gametes
gene -- the smallest functional unit of genetic material on a chromosome, bearer of a hereditary trait
genetic -- relating to heredity; pertaining to heritable characteristics as influenced by germ plasma
genome -- set or group of chromosomes
genotype -- the genetic constitution of an individual or group; class or group of individuals sharing a particular genetic makeup
genus (pl. genera) -- group of related species
germ plasma -- material capable of transmitting heritable characteristics sexually or asexually
germ tube -- hypha produced as an outgrowth of a spore wall or cytoplasm
germinate -- to begin growth (of a seed, spore, sclerotium, or other reproductive body)
giant cell -- multinucleate cell formed by the disintegration of cell walls (also called a syncytium, in nematode infections)
girdle -- to circle and cut through
globose -- almost spherical
globular -- almost spherical
gram-negative -- pertaining to bacteria that stain red when stained by Gram's technique
gram-positive -- pertaining to bacteria that stain violet when stained by Gram's technique

hardpan -- compacted soil layer that roots cannot penetrate
haustorium (pl. haustoria) -- hyphal branch specialized for absorbing food, produced by a parasite, especially within a living host cell, and usually associated with obligate parasites such as rust fungi
herbaceous -- nonwoody, with reference to a plant or plant part
herbicide -- chemical agent that kills herbaceous plants; broadly, chemical agent that limits the growth of herbaceous plants
heteroecious -- pertaining to a rust fungus requiring two unrelated hosts for completion of its life cycle
heterotrophic -- obtaining nourishment from outside sources
hexaploid -- having six sets of the basic number of chromosomes per cell
hexarch -- having six radiating vascular strands
homothallism (adj. homothallic) -- condition in which sexual reproduction can occur without the interaction of two different thalli, both sexes being present in the same mycelium
host -- living plant attacked by or harboring a parasite and from which the invader obtains part or all of its nourishment
host range -- collectively, the plants attacked by a given pathogen
hyaline -- transparent or nearly so; translucent; colorless
hymenium -- the spore-bearing layer of a fungal fruiting body
hyperparasite -- organism that is parasitic upon another parasite
hyperplasia -- overdevelopment of tissue (e.g., swelling, galls, or witches'-broom)
hypertrophy -- excessive growth caused by an abnormal increase in the number of cells in tissues or organs
hypha (pl. hyphae) -- tubular filament of a fungal thallus or mycelium, the basic structural unit of fungi

imperfect stage -- the asexual part of the life cycle of a fungus, when asexual spores (such as conidia) or no spores are produced
in vitro -- in glass, on artificial media, or in an artificial environment
inclusion body -- virus-induced structure formed in the cytoplasm or nucleus of cells of infected plants
indeterminate -- having an edge not well defined (of fruiting bodies, leaf spots, etc.); continuing growth indefinitely (of plants or conidiophores)
indicator plant -- plant that reacts to a pathogen (especially a virus) or an environmental factor with specific symptoms, used to identify the pathogen or determine the effects of the environmental factor

indigenous native to a country or region
infection process by which a pathogen enters and parasitizes a host
infection court site in or on a host where infection can occur (e.g., root, stem, leaf, pod, seed)
infection peg very fine tubular extension of a hypha, thrust through the cuticle or wall of a host cell
infectious capable of spreading disease from plant to plant
infestation attack by animals, especially insects or nematodes; aggregation of inoculum or other organisms on a plant surface
injury result of the transitory operation of an adverse factor such as insect feeding, chemical action, or unfavorable environmental conditions
inoculate to place inoculum in an infection court
inoculum pathogen or its parts capable of establishing a live colony when transferred to a favorable location
intercalary cell cell between two others
intercellular between or among cells
internode the area between two nodes on a stem
intracellular within or through a cell or cells
isolate pure microbial culture, separated from its natural origin

lamella (pl. lamellae) thin layer of cells, scales, or gills in plants
lamina (pl. laminae) the blade of a leaf
larva (pl. larvae) juvenile; growth stage between embryo and adult
latent present but not manifested or visible
latex white, rubberlike substance exuded from cut parts of sweet potato plants
lenticel pore in the bark of woody stems and other plant parts that permits exchange of gases
lesion well-marked, localized diseased area; wound
lignification process by which constituents of cell walls are converted into lignin, thereby forming woody tissue
local lesion host host (usually of a virus) responding to infection by developing lesions at the site of infection
locule cavity, especially in a stroma

macerate to cause to become softened and disintegrated as by steeping or soaking in fluid
macroconidium the larger, generally more diagnostic conidium of a fungus (e.g., *Fusarium*) that also has microconidia; long or large conidium
macrocyclic pertaining to the life cycle of a rust fungus in which all five spore states are produced
macroscopic visible with the naked eye
mechanical transmission, mechanical inoculation spread or introduction of inoculum to an infection court (especially, a wound) by hand, accompanied by physical disruption of host tissue
meristem (adj. meristematic) plant tissue that functions principally in cell division and differentiation, consisting of a mass of growing cells capable of frequent cell division
meristem tip culture excision and culture of the dome of cells at the growing tip of a plant, from which plants are regenerated and then tested for the presence of pathogens
microbial pertaining to or relating to microbes, or microorganisms
microconidium the smaller conidium of a fungus (e.g., *Fusarium*) that also has macroconidia; small conidium, often acting as a spermatium
microflora the composite of microscopic plants at a site
microsclerotium (pl. microsclerotia) microscopic, dense aggregate of darkly pigmented, thick-walled hyphal cells
microscopic too small to be seen except with the aid of a microscope
midrib the central, thickened vein of a leaf
MLO see mycoplasma-like organism
molt to shed a cuticle or body encasement (such as the outer sheath of a nematode) during a phase of growth
monoclinous having the oogonium and its antheridium on one hypha
monocot plant having one cotyledon
morphology the study of the form and structure of organisms
mosaic disease symptom characterized by a mottling of foliage or variegated patterns of dark green and light green to yellow, caused by disarrangement or unequal development of the chlorophyll content
mother root in sweet potato culture, a storage root planted in a bed for the purpose of producing sprouts for transplanting
motile exhibiting or capable of movement
mottle disease symptom characterized by irregular patterns of light and dark areas on leaves
mummify to dry and shrivel up
mutation abrupt heritable change in an individual
mycelium (pl. mycelia) mass of hyphae constituting the body (thallus) of a fungus
mycoplasma any of a class of pleomorphic prokaryotic micro-

organisms lacking cell walls
mycoplasma-like organism any of various microorganisms found in plant phloem that have not yet been cultured on artificial media but otherwise resemble mycoplasmas

necrosis (adj. necrotic) death of plant cells, usually resulting in a darkening of tissue
nematicide chemical agent that kills nematodes
nematode any of the threadlike, round, usually soilborne worms of the order Nematoda, of which several of microscopic size attack sweet potatoes
node joint of a stem; site of attachment of a leaf or leaves
nonpersistent virus virus that remains infectious within its insect vector for only a short time and may be transmitted immediately after acquisition
nymph juvenile insect resembling the adult

obligate parasite organism that lives only as a parasite and has not been cultured on laboratory media
obovoid inversely ovoid, broad at the upper end and narrow at the base
obpyriform shaped like an inverted pear
olivaceous colored olive green
oogonium (pl. oogonia) one-celled female sexual organ of some fungi, which produces one or more eggs (oospheres)
oospore thick-walled resting spore produced from an oosphere by fertilization or parthenogenesis
ostiolate having an ostiole
ostiole pore; opening in the papilla or neck of a perithecium or pycnidium, through which spores are freed
overwinter to survive the winter
ovoid egg-shaped

parasite organism living in or on another living organism (host) and obtaining food from it
parenchyma physiologically active plant tissue composed of thin-walled cells that often store food and usually retain meristematic potentials
parthenogenesis (adj. parthenogenetic) development of an egg (female gamete) into a new individual without fertilization by a sperm (male gamete)
pathogen (adj. pathogenic) organism or agent that causes disease in another organism
pathology the study of diseases and their control
pectolytic pertaining to an enzyme capable of dissolving pectin, the substance that normally cements plant cells together
pentarch having five radiating vascular strands
perennial a plant naturally persisting vegetatively for more than one year or growing season
perfect stage the state in the life cycle of a fungus in which sexual spores (ascospores or basidiospores) are formed after nuclear fission
pericycle layer or layers of cells between the phloem and the endodermis of roots and stems, giving rise to branch roots
periderm thin layer of cork cells produced on the surface of storage roots
perithecium (pl. perithecia) flask-shaped or subglobose, thin-walled fungal fruiting body (ascocarp) containing asci and ascospores, the latter being expelled or otherwise released through a pore (ostiole) at the apex
persistent virus circulative virus that remains infectious within its insect or other vector for long periods and is transmitted in salivary fluids
pest any organism that injures plants or plant products
petiole stalk of a leaf
pH measure of acidity (pH 7 is neutral; below pH 7, acidic; above pH 7, alkaline)
phellogen cork cambium
phialide end cell of a conidiophore or a conidiophore of fixed length with one or more open ends through which conidia develop in succession from the apex toward the base
phloem food-conducting tissue in plants
photosynthesis manufacture of carbohydrate food from carbon dioxide and water in the presence of chlorophyll (or chlorophylls), using light energy and releasing oxygen
physiological race subdivision within a species, comprising organisms that differ from other members of the species in virulence, symptom expression, biochemical and physiological properties, or host range, but not in morphology
phytoalexin any of a group of chemical substances produced by plants to combat infection

- phytotoxic**—harmful to plants
- pith**—soft, spongy tissue in the center of certain plant stems and storage roots
- pleomorphic**—having various shapes, of nonconstant form
- plurivorous**—living upon several hosts
- pollen** (v. pollinate) male sex cells produced by flowering plants
- potyvirus** any of the type of plant viruses similar to potato virus Y, having long, flexuous, rod-shaped particles (700–950 nm) and containing single-stranded RNA
- predispose** to make prone to infection and disease
- primary infection** the first infection by a pathogen after a resting or dormant period
- primary inoculum** inoculum that initiates rather than spreads or magnifies disease
- prokaryotic** (n. prokaryote) without internal membrane-bound organelles
- propagule** any of an organism's parts capable of initiating independent growth
- protectant** an agent, usually a chemical, that tends to prevent or inhibit infection
- protein** any of numerous complex, organic, nitrogenous substances built up from amino acids and constituting a major part of the organic materials in living protoplasm
- proximal** near the origin or point of attachment
- pseudoparenchyma** (adj. pseudoparenchymatous) aggregate of closely interwoven hyphae forming a definite body
- pustule** small, blisterlike, frequently eruptive spot or spore mass
- pycnidiospore** conidium produced within a pycnidium
- pycnidium** (pl. pycnidia) asexual, flask-shaped or globose fungal fruiting body containing conidia (pycnidiospores)
- race**—see physiological race
- ramify**—to branch; to separate or split into branches or constituent parts; to send forth branches or extensions
- reniform** kidney-shaped
- resistance**—ability of a host to overcome, suppress, prevent, or impede the activity of a pathogen
- respiration** series of chemical reactions whereby living protoplasm produces energy from oxygen, carbohydrate, and fat
- rhizoid** a rootlike hypha
- rhizomorphic** pertaining to specialized mycelium in which several strands of hyphae are twisted together to appear rootlike
- rhizosphere** the soil microenvironment near a living root, where the microflora is frequently richer than that in soil away from roots
- ribonucleic acid** any of a number of nucleic acids containing ribose, uracil, guanine, cytosine, and adenine and associated with the control of cellular chemical activity and protein synthesis (the type of nucleic acid present in most plant viruses)
- ring spot** disease symptom characterized by yellowish or necrotic rings surrounding green tissue, as in some virus diseases
- RNA**—see ribonucleic acid
- rogue**—to remove and destroy by hand individual plants that are undesirable because of infection or insect infestation or for some other reason
- rootlet** small root
- rugose** wrinkled
- sanitation** destruction of infected and infested plants or plant parts
- saprophyte** (adj. saprophytic)—organism that feeds on dead organic matter
- sclerotium** (pl. sclerotia)—hard, frequently rounded, usually darkly pigmented resting body of a fungus, composed of a mass of specialized hyphal cells and capable of remaining dormant for a long period and germinating when favorable conditions return, to produce a stroma, fruiting body, mycelium, or conidiophores
- secondary infection** infection resulting from the spread of infectious material produced after a primary infection or another secondary infection without an intervening inactive period
- secondary root** root developed from another root, as opposed to a primary root
- seed root** storage root planted for the purpose of producing transplants (sometimes referred to as seed)
- senescence** decline or degeneration with maturation, age, or disease stress
- sepal**—one of the modified leaves of a calyx
- septate** (n. septation)—having septa, or cross-walls
- septum** (pl. septa) cross-wall in a hypha
- serology**—the study, detection, and identification of antigens, antibodies, and their reactions
- shoot**—a stem with its leaves
- shoot tip culture** excision and culture of the meristematic dome with accompanying leaf primordia from the growing tips of plants
- shot hole**—disease symptom in which small, round fragments drop out of leaves, which then appear shot-riddled
- sign**—indication of disease from direct visibility of the pathogen or its parts (spores, mycelium, exudate, or fruiting bodies)
- slip**—sprout pulled from a bedded mother root for transplanting
- somatic** relating to the body, especially body cells as distinguished from germ plasma
- sorus** (pl. sori)—compact fruiting structure, especially the spore mass in rust and smut fungi; occasionally, a group of fungal fruiting bodies
- sp.** (pl. spp.)—species, the taxonomic classification subordinate to genus and higher than race, strain, and variety (sp. is used following a genus name to refer to an undetermined species; spp. is used following a genus name to refer to several species without naming them individually)
- sporangiophore** differentiated hypha that bears a sporangium
- sporangiospore** spore that develops in a sporangium
- sporangium** (pl. sporangia)—saclike or flasklike structure whose contents are converted into asexual spores (sporangiospores, zoospores)
- spore**—one- to many-celled reproductive body (in fungi and other lower plants) that can develop into a new plant
- sporodochium** (pl. sporodochia)—conidial spore mass supported by a cushion-shaped mass of short conidiophores interwoven on a stroma
- sporogenous** pertaining to reproduction by spores
- sporophore** structure bearing a spore
- sporulate**—to produce spores
- sprout**—shoot that develops from a bedded mother root
- stele** (adj. stelar)—the central column of sap-conducting tissues in stems and roots
- stem cutting**—portion removed from a stem or vine for propagating the plant (also called vine cutting)
- sterigma** (pl. sterigmata)—small, usually pointed hyphal branch or structure supporting a spore (conidium, basidiospore) or sporangium
- sterile**—free from contaminant organisms; incapable of propagation; infertile
- stolon**—modified stem arising from the base of certain plants and capable of producing a new individual; runner hypha, produced by certain fungi
- stoma, stomate** (pl. stomata, stomates)—regulated opening (pore) in plant epidermis, controlled by guard cells, for passage of gases and water vapor
- storage root**—the fleshy root of the sweet potato, which enlarges by division of cells in the vascular and anomalous cambia (sweet potatoes do not produce tubers)
- strain**—biotype; race; organism or group of organisms differing in minor respects from others of the same species or variety
- stroma** (pl. stromata; adj. stromatic, stromatal)—compact mass of specialized hyphae (with or without host tissue or substrate), sometimes resembling a sclerotium in form, in or on which fruiting bodies or spores (or both) are produced
- stunted**—unthrifty; reduced in size and vigor
- stylet**—stiff, slender, hollow feeding organ of plant-parasitic nematodes
- stylospore**—elongated pycnidiospore
- suberin**—waxy, water-impermeable substance associated with corky tissue deposited in or on plant cells
- subhyaline**—almost colorless
- substrate**—substance or object on which a saprophytic organism feeds and develops
- sunscald**—injury of plant tissues “burned” or scorched by excess sun and other unfavorable conditions
- susceptible**—lacking resistance; prone to infection; subject to attack and infection by a pathogen
- symptom**—indication of disease by a reaction of the host (compare *sign*)
- symptomatology**—the study of disease symptoms
- syncytium** (pl. syncytia)—multinucleate mass of protoplasm resulting from fusion of protoplasts and surrounded by a common cell wall
- systemic**—pertaining to chemicals, pathogens, or single infections that spread generally throughout a plant body instead of remaining localized
- teleomorph**—the sexual or perfect thallus of a fungus; the binomial of a fungus in its sexual stage
- teliospore**—thick-walled resting spore produced by some fungi, notably rust and smut fungi, and germinating to form a basidium
- telium** (pl. telia)—sorus that produces teliospores
- tetraploid**—having four sets of chromosomes per cell
- thallus** (pl. thalli)—fungus body

thermotherapy—the practice of growing plants at elevated temperatures prior to selecting tissue for the culture of plantlets for pathogen testing, frequently used in addition to meristem tip culture to increase the efficiency of obtaining plants free of known pathogens

tissue—group of cells, usually of similar structure, that perform the same or related functions

tolerant—capable of sustaining disease without serious damage or yield loss

toxicity—capacity of a substance to produce injury

toxin—poison produced by a living organism

translocation—movement by water, minerals, food, or pathogens within a plant

translucent—so clear that light can pass through

transmission—spread of a pathogen (especially a virus) from plant to plant

transpiration—loss of water vapor from aerial parts of plants, chiefly through stomata in leaves

transplant (v.) to remove a plant from one site and replant it at another; (n.) a plant removed from one site for replanting at another

true seed—seed resulting from sexual fusion of gametes (in contrast to seed roots, which are produced asexually)

truncate—ending abruptly, as though with the end cut off

tuber—short, thickened, fleshy, underground stem, usually borne at the end of a stolon (potatoes produce tubers; sweet potatoes produce storage roots)

turgor—internal fluid pressure of living cells

urediospore—binucleate, dikaryotic, asexual, one-celled repeating spore of rust fungi, borne in a uredium

uredium (pl. uredia)—the fruiting body (sorus) of rust fungi, which produces urediospores

variety—group of closely related plants of common origin within a species, differing from other members of the species in certain minor details

vascular—pertaining to conductive tissue (xylem and phloem)

vascular ring—circular arrangement of vascular strands within a stem or storage root

vector—agent (insect, mite, animal, human, etc.) able to transmit a pathogen

vegetative—pertaining to somatic or asexual parts of a plant; not involved in sexual reproduction

veinclearing—disappearance of the green color in or along leaf veins

viable (n. viability)—able to germinate (with reference to seeds, spores, sclerotia, etc.)

vine—long, slender stem that trails along the ground or climbs by winding around a support

vine cutting—transplant taken by cutting a portion of a vine growing either in the field or on a mother root in a plant bed

virescence (adj. virescent)—state or condition of turning green

virion—complete virus particle

virulence (adj. virulent)—degree or measure of pathogenicity; relative capacity to cause disease

viruliferous—carrying a virus (applied particularly to insect vectors)

volunteer—self-set plant, seeded by chance

water-soaking—disease symptom in which plants appear wet and dark or develop wet-locking lesions, which are usually sunken and transparent

wilt—lack of freshness or drooping of leaves from lack of water due to an inadequate water supply or excessive transpiration; a vascular disease that interrupts the normal uptake and distribution of water

witches'-broom—disease symptom characterized by an abnormal, massed, brushlike development of many weak shoots arising at or close to the same point

xylem—water-conducting tissue in plants

zonate—marked with zones; targetlike

zoospore—asexually produced fungal spore with cilia or flagella, capable of locomotion in water

zygote—sexually produced cell formed by the union of two gametes

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