PRINCIPAL BACTERIAL AND FUNGAL DISEASES OF SWEET POTATO AND THEIR CONTROL

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Many of the bacterial and fungal diseases of sweet potatoes have been known for almost 100 years (17) and several monographs and reviews have been written in the USA over that period of time (6,7,23,35,53,56,57,81,84).

The relative importance of bacterial and fungal diseases of sweet potato is highly dependent on two factors: i) the type of sweet potato production system, and ii) the intended utilization of the crop. Generally, sweet potatoes are produced in one of two systems. In warm tropical climates they are usually grown year round and therefore may be propagated using vine cuttings taken from fields already in production. In subtropical and temperate regions, they are propagated from transplants produced from storage roots which must be preserved during the cool season and sprouted prior to transplanting. Many important bacterial and fungal diseases are root diseases, and thus increase to much greater extent when storage roots are involved in the propagation cycle.

Sweet potatoes are utilized in diverse ways in different regions of the world and their use influences regional perceptions of the relative importance of prevalent diseases. Where sweet potatoes are grown primarily for foliage or vine tips, foliar diseases may be regarded as more important than root diseases. Where sweet potatoes are grown for storage roots, the perceived importance of foliar and root diseases is reversed.

Fungal and bacterial diseases affecting storage roots are important because they affect yield, esthetic quality, storage life, and nutritional value of storage roots. Fungal pathogens in particular can induce sweet potato storage roots to produce an array of furanoterpenoid phytoalexin-like compounds which may be toxic to animals (9,48,70). Because of esthetic requirements and potential detrimental health effects, the amount of root disease that can be tolerated on sweet potato is much lower than it is for nonroot crops which generally require multiple infections by root pathogens before the crop is economically affected.

BACTERIAL DISEASES

There are four major diseases caused by prokaryotes each of which occurs only in a restricted geographic region.

Bacterial Soft Rot

Bacterial soft rot of sweet potato was first reported in the southeastern United States in 1977 (47). It is caused by Erwinia chrysanthemi and affects both vines and storage roots (47,74). A soft, moist decay resembling Rhizopus soft rot turns affected storage root tissue light brown. In storage, the lesions have a dark brown to black margin and appear to be restricted, but in plant production beds, some roots are totally decayed leaving only residual fibers and periderm. Sprouts may become infected from partially decayed mother roots in beds or vines may become infected during or after transplanting. The pith of the vine is quickly decayed and the hollowed out vines become extensively necrotic, wilt and sometimes die.

Bacterial Wilt

Although sweet potatoes are grown in many regions of the world where the causal organism of bacterial wilt, Pseudomonas solanacearum, occurs on other crops, the disease has been reported on sweet potato only in The People's Republic of China (35). Apparently Chinese strains of the bacterium are unique in their pathogenicity to sweet potato (26). The bacterium induces vascular discoloration and wilting of vines in the field and can invade storage roots and cause them to breakdown.

Soil Rot or Pox

Soil rot, caused by the host-specific, soil-borne actinomycete, Streptomyces ipomoea, has been reported only in the USA (72). It causes severe losses under conducive field conditions but does not affect roots in storage. Symptoms are similar to those reported from China for Fusarium root rot, including extensive necrosis of feeder roots and scab-like lesions on the storage roots. The pathogen invades the storage roots through feeder roots and does not penetrate the periderm directly. When storage roots are infected early in development, subsequent enlargement at the site of infection is prevented. This leads to the development of indentations or constrictions in the fully grown root. The disease develops only in soils with pH of about 5.2 or higher although the pathogen may persist in more acid soils without causing disease (52,71). Disease development is also much greater in dry than in moist soil (73).

Witches' Broom

Witches' broom, also known as little leaf or Ishuku-byo, is caused by a mycoplasma-like organism (13,29,78). It was first reported in 1947 and now occurs in southeast Asia and Oceania.

Proliferation of axillary shoots and stunting of vine growth and leaf enlargement are the most pronounced symptoms. Etiology of the disease can be confirmed by observation of the characteristic pleomorphic bodies in the phloem (13,33,86). Geographic distribution of the disease has been correlated with the range of the leafhopper vectors, Orosius lotophagorum ryukyuensis and Nesophrosyne ryukyuensis (89). The disease has a very long latent period which makes detection difficult.

FUNGAL DISEASES

Most of the diseases of sweet potatoes caused by fungi have been recognized for many years (17). A number of these are foliar diseases. Some are quite common and others have been observed only in certain geographic areas, but with the exception of vine and leaf scab, they have not been observed to cause sufficient damage to the plant to warrant control efforts. Included in this group are: rust, caused by Coleosporium ipomoeae; white rust, caused by Albugo ipomoeae-panduranae; and leaf spots caused by; Alternaria spp., Cercospora spp., Phyllosticta batatas, and Septoria bataticola.

Several fungal diseases, particularly black rot, foot rot, and scurf are very efficiently transmitted through the storage root propagation cycle but do not persist many years in soil. These pathogens have narrow host ranges, however, Convolvulaceous weeds become infected following artificial inoculation (11). Whether such weeds play an important role in pathogen survival between sweet potato crops has not been determined. Many fungi can occasionally cause storage rots, especially under adverse conditions (24), but those discussed below are the most common.

Black Rot

Black rot, caused by Ceratocystis fimbriata, was once one of the most economically significant diseases of sweet potato in the USA (83). It no longer causes significant losses in the USA but remains a serious problem in other regions of the world. Strains of the same fungus cause disease on other hosts such as taro, almond, cacao, and others, but the strains seem to be very host specific. A black, dry rot is produced on the storage roots which is often restricted to the cortex. Black sunken cankers are produced on sprouts below the soil line. Perithecia of the causal fungus are sometimes produced on the surface of infected tissue and the tissue has a characteristic fruity odor.

In the USA, it has been determined that C. fimbriata does not persist in soil for more than 1-2 years in the absence of a susceptible host. The pathogen is very efficiently transmitted from infected mother roots to the underground portions of sprouts and from infected sprouts to the succeeding crop of storage roots

(15). Chewing insects and rodents may also transmit the pathogen (16) and spores may also be carried in wash water.

Charcoal Rot

Although charcoal rot may appear on some storage roots prior to harvest, it is primarily a postharvest disease (83). It is caused by Macrophomina phaseolina, a warm weather pathogen with a very broad host range. It produces a firm decay of the storage roots which first turns the tissue reddish-brown and then black as sclerotia of the pathogen are produced within the tissue. Often the sclerotia are produced only in the cortex of the storage root even though the whole root is affected.

Circular Spot and Sclerotial Blight

Sclerotium rolfsii, a pathogen of many crops grown in warm climates, causes two distinct diseases of sweet potato that occur at different stages in the development of the crop. Circular spot develops on storage roots in the field just prior to harvest. The lesions are seldom more than 1-2 mm deep but may be from a few mm to a few cm in diameter with a well-defined margin (41). The lesions cease development when the roots are harvested, and within a few days after harvest it is very difficult to isolate the pathogen from them. When harvested roots are placed in a high-humidity environment immediately after harvest, mycelia of S. rolfsii often grow out. Eventually, the root produces an abscision zone around the lesion. There is still doubt about the role of circular spot lesions on mother roots as potential sources of inoculum for sclerotial blight which develops in plant production beds.

Sclerotial blight first appears as wilting of the sprouts at about the time they are ready for transplanting (19,27). Necrotic lesions girdle the sprouts at the soil line and they then quickly desiccate and die. Expanding, circular foci of infection can destroy large areas of the bed. Coarse white mycelia of the fungus, and later sclerotia, appear on the soil surface around the plants and if the mother roots are unearthed, the mycelia can be seen on the surface of the roots and at late stages of infection the mother root may be extensively rotted with minimal discoloration of the tissue. Cultivars vary in reaction to sclerotial blight and those with a degree of resistance may develop restricted lesions on sprouts below the soil line rather than the more aggressive blight. Volatile chemicals emanating from mother roots with any of several storage rots stimulate germination of sclerotia of S. rolfsii and may increase disease incidence.

Foot Rot

Foot rot, caused by Plenodomus destruens, occasionally causes serious losses in isolated fields but is not a limiting factor to sweet potato production. The most commonly observed symptom is the

occurrence of necrotic lesions which often girdle the vines in the field at or just below the soil line (18). The pathogen may grow down the vine into the storage roots and cause a slowly progressing decay which does not usually destroy the entire root even in storage. The pathogen readily spreads from infected mother roots to sprouts. Pycnidia of the fungus are commonly produced near the surface of infected vines and just beneath the periderm of storage roots.

Fusarium Rots

Surface rot is a long-known disease which affects sweet potatoes only in storage (22,54). It is caused by cortical-rotting strains of Fusarium oxysporum, a soil-borne, wound pathogen distinct from the Fusarium wilt pathogen. Symptoms usually develop following harvest and consist of a brown, dry rot restricted to the cortex of the storage roots. Symptoms may occur in the field if growth cracks develop in the storage roots. Disease severity is affected by conditions in the field leading up to harvest, and dry weather, which favors 'skinning' of the roots during harvest, may lead to an increase in disease incidence (61).

Fusarium solani has been reported in the USA to cause a complex syndrome which manifests itself in different ways depending on the cultivars and environmental conditions involved. It can cause a surface rot which is difficult to distinguish from that reported for F. oxysporum (8). On some cultivars, it more commonly enters the storage root from one of the ends causing an end rot (8). Recently, some strains of F. solani have been shown to cause a more aggressive root rot that frequently penetrates the vascular ring, causing a dry rot which can slowly consume much of the root (65). It is characterized by the presence of lens-shaped cavities which often have mycelial growth on their inner surfaces. In addition, some of these strains are also capable of moving from infected mother roots into the sprouts and growing in advance of macroscopic symptoms (55,58). In sprouts and vines, these strains cause a stem canker that includes necrosis and splitting of the stem. Infection can apparently occur either through the storage root propagation cycle or as a result of invasion of harvest associated wounds from soil-borne inoculum.

Fusarium solani has also been described as the cause of a root rot in China which differs from that described in the USA in that it involves extensive feeder root necrosis (28,39).

Fusarium Wilt

Fusarium wilt, also commonly known as stem rot, is caused by Fusarium oxysporum f. sp. batatas (21). Symptoms on sweet potato may appear on sprouts in the plant beds or more commonly shortly after the transplants begin growing in the field. Symptoms include yellowing of the lower leaves, pronounced vascular discoloration, wilting and sometimes stem necrosis and death of the plant.

Surviving plants may produce daughter roots with discolored vascular tissues that serve as a source of inoculum for the next crop, or the roots may become infected during harvest (60).

The pathogen is relatively specific with regard to the hosts in which the wilt syndrome is induced, which include: sweet potato, related Ipomoea spp., and certain genotypes of tobacco (2,11). However, the pathogen can also colonize other plants without inducing wilt. It also persists in the soil for many years in the absence of a sweet potato crop. The disease was once especially serious in many sweet potato growing areas in the USA and Japan. However, wilt has been relatively unimportant in many parts of South and Central America. In some cases, biologically suppressive soils may limit the distribution and severity of the disease (77).

The disease is no longer considered to be a limiting factor in some areas where it once threatened the economic survival of the sweet potato industry due primarily to the use of resistant cultivars and also to the use of crop rotation and sanitation procedures (62).

Java Black Rot

The name for this disease is a misnomer as the disease is widely distributed, especially in the warmer areas of the world (12,32,83,87). It generally only affects the storage roots following harvest and is caused by Diplodia gossypina (syn. = Diplodia tubericola, Botryodiplodia theobromae et al.). Symptoms often progress from the end(s) of the storage root and involve the entire root. The decay is firm, at first reddish-brown then turning black. The root desiccates and becomes very hard. When infection occurs through wounds on the side of the storage root, lesions are often restricted and develop a black center surrounded by a wide brown zone (69). At advanced stages of infection, the disease is readily recognized by the black stromatic domes which erupt through the periderm of the root. The stroma contain pycnidia with many 1 or 2-celled conidia.

The fungus is soilborne and infects storage roots through wounds incurred during harvest. Stressed roots are particularly susceptible (1). Susceptibility of the storage roots increases significantly during storage as does the availability of inoculum. Thus the greatest losses to this disease often occur following handling of stored sweet potatoes (40).

Rhizopus Soft Rot

Several different species of Rhizopus can be associated with soft rot but in temperate and subtropical areas the predominant pathogen is R. stolonifer (syn. = R. nigricans) and in tropical areas R. oryzae predominates (25,37,88). Storage roots are very rapidly destroyed by a soft, watery decay that consumes the entire

root. Whiskers', consisting of mycelia, sporangiophores and sporangia of the causal fungus commonly appear as the infection progresses. Storage roots affected by Rhizopus soft rot also give off a distinctive alcohol-like odor.

Although Rhizopus spp. are the most common storage rot pathogens, they are fastidious about the type of wound required for infection. Infection occurs only when wounds kill surrounding tissue, or when exogenous nutrients or pectolytic enzymes are added to the inoculum (79). Only the harvested storage roots are affected, but the losses have been tremendous in some circumstances. Roots are predisposed to the disease by prior chilling or exposure to direct sunlight.

Scurf

Scurf is a superficial disease of storage roots and below-ground portions of the stem that is caused by Monilochaetes infuscans (20,34,85). Affected roots have dark brown lesions, resembling a stain, which are restricted to the periderm. Lesions continue to enlarge and may coalesce and spread from mother roots onto sprouts produced on them and from there to daughter roots. When infected roots are incubated in a moist chamber, the fungus produces simple, dematiaceous conidiophores from a bulbous basal cell and a curved chain of hyaline, single-celled conidia. Although the flesh of the storage root is not directly affected, severely diseased roots are much more subject to water loss and shrinkage during storage (14).

Vine and Leaf Scab

Also known as bud atrophy, this disease caused by Elsinoe batatas (anamorph = Sphaceloma batatas), may sufficiently affect yield of sweet potato to justify control efforts. Round to elliptical lesions first appear on leaf veins, petioles or stems as yellowish-brown spots which become gray-brown and finally corky (7,30). Symptoms are more pronounced on young shoots and leaves and on particularly susceptible genotypes the disease may cause stunting of the shoot system and distortion of the leaves.

The disease is restricted in its geographic distribution to southeast Asia and many of the Pacific islands (7,57). It is most common and severe in areas and seasons that are rainy.

Violet Root Rot

Helicobasidium mompa causes a serious root disease of sweet potato and many other crops in some parts of southeast Asia (82). Feeder roots are extensively decayed and the storage roots are often totally decayed prior to harvest. Storage roots begin decaying from the distal end and develop an odor similar to that associated with Rhizopus soft rot. The decayed roots are held together by a thick mantle of coarse mycelia over their surface.

The mycelia are at first white, becoming pink, brown and finally violet with a velvet-like texture as a hymenial layer with basidia and basidiospores is formed. Brown sclerotia may form on the stem near the soil line and a mat of purple-brown mycelia may grow over the surrounding soil (35).

The fungus persists in soil for several years as sclerotia, mycelial strands or in debris. The disease is most severe in wet soils, especially when saturation occurs late in the growing season.

CONTROL

Preventing infection is the key to control of bacterial and fungal sweet potato diseases since few effective therapeutic techniques are available to treat infected tissue. This is achieved by preventing contact between the pathogen and the crop and is accomplished on an international scale by regulatory controls designed to prevent introduction of pathogens into regions where they have not been previously detected (66). Several important bacterial and fungal sweet potato diseases appear to be geographically restricted. However, there has been a lack of organized effort involving plant pathoglogists to determine the true distribution of sweet potato diseases. Reports of diseases such as bacterial soft rot and circular spot/sclerotial blight imply a far more restricted distribution than the known ranges of the pathogens which cause them. In other cases, it is difficult to determine whether the diseases reported in different regions are the same. An example is Fusarium root rot as reported from the USA and China (8,28,39,65). Differences in disease occurrence in different regions may be due to use of different sweet potato genotypes which may differ in relative susceptibility or symptom expression for many diseases. Some of these questions might be resolved by increased interaction among plant pathologists in different areas of the world.

Generally, bacterial and fungal sweet potato pathogens have not been considered as important as viruses in regulation of international exchange of germplasm. This is because these pathogens are not known to be carried in true seed and in most cases can be detected as contaminants in tissue culture. They are also more readily eliminated by meristem-tip culture than most viruses and thus procedures routinely used for virus elimination are also effective for bacteria and fungi. However, it should be recognized that several very destructive bacteria and fungi such as E. batatas, H. mompa, P. solanacearum, S. ipomoea, and the witches' broom MLO, are currently restricted in their geographic distribution. It will continue to be necessary to exchange germplasm only in the form of true seed or meristem-derived tissue cultures to confine these pathogens as well as the viruses.

Bacterial and fungal diseases are in general most successfully controlled by an integrated program of sanitation, rotation, curing and fungicide application. In addition, some practices are designed for specific diseases. For example, particular advantage can be taken of the fact that certain important root pathogens (C. fimbriata, P. destruens, and M. infuscans) have limited persistence in soil and a restricted host range. They cause greatest losses when associated with the storage root cycle of propagation, and breaking this cycle is an important aspect of their control.

An important sanitation measure is the use of mother roots as free of disease as possible. Mother roots should be chosen from a crop with few disease problems to begin with, and they should be sorted before they are bedded to cull roots with any symptoms. In addition, by harvesting mother roots only under weather conditions that minimize subsequent storage rot development and by immediately curing roots after harvest, it is possible to limit entry of pathogens into the cycle during storage.

Crop rotations which include several years of nonhost crops between sweet potato crops are very effective in reducing soil-borne inoculum of pathogens such as C. fimbriata, P. destruens, and M. infuscans to levels below the threshold necessary for infection. Inoculum potentials of Fusarium spp., S. ipomoea, and D. gossypina are also reduced even though these pathogens persist for many years.

The intact periderm of the sweet potato storage root is a very effective barrier to the known sweet potato pathogens with the exception of M. infuscans. However, storage roots must be broken from the vine at harvest, and they are also easily injured during the harvest process. Thus all storage roots are wounded and infection sites are available for the many wound pathogens which can infect them. Curing sweet potatoes immediately after harvest by placing them in conditions of 85-90% RH and 28-30 C for 5-10 days effectively promotes the formation of wound periderm and thus protect wounds from invasion by a number of different pathogens (36,56). Subsequent storage at 15-16 C also helps reduce storage rot problems.

Fungicide use on sweet potatoes is limited, but it is important for control of certain diseases. Treatment of storage roots with dichloronitroaniline after washing and grading is a very effective and commonly used measure in some areas to control Rhizopus soft rot (43). Treatment of mother roots just prior to bedding with fungicides such as thiabendazole or benomyl combined with dichloronitroaniline is an important aspect of control of black rot, foot rot, Fusarium rots, Java black rot, Rhizopus soft rot, sclerotial blight, and scurf (4,45,46). Transplants have also been dipped in fungicides to control Fusarium wilt when susceptible

cultivars are grown (63). Fumigation with formulations containing chloropicrin has also been used to reduce the inoculum potential of S. ipomoea.

Heat therapy has been used on transplants and/or mother roots to reduce several soil-borne diseases (44,64). However, it also reduces survival of the sweet potatoes even under precisely controlled conditions. Thermotherapy is an effective control for witches' broom (6).

RESISTANCE - PRESENT USE AND FUTURE POTENTIAL

Fusarium wilt, which once threatened the continued existence of the sweet potato industry in the southeastern USA, is now controlled almost exclusively by the use of resistant cultivars (53,68,80). Resistant cultivars are also available which can be used to control soil rot under commercial growing conditions (51,59). Some degree of variation in the reaction of commercial cultivars and advanced breeding lines has also been recognized for nearly all the other major bacterial and fungal diseases of sweet potato including: Fusarium root rot (8,10,65,76), surface rot (75), bacterial soft rot (47,74,Clark, unpublished data), circular spot, sclerotial blight, vine and leaf scab, Java black rot (31), bacterial wilt (35), scurf (50,67), violet root rot (35), witches' broom, black rot (5,42,67), and Rhizopus soft rot (31). However, resistance to these diseases has not been commercially exploited either because the level of resistance is not sufficient or the resistance has not been combined with suitable horticultural quality (49). There has been some suggestion that Fusarium root rot and bacterial soft rot problems may have arisen in the southeastern USA in part due to the release of new cultivars that were more susceptible to these diseases than their predecessors (8,10,47,65,74).

It appears that virtually all the major bacterial and fungal diseases of sweet potato could be the subject of projects to improve the levels of resistance in commercial cultivars by traditional breeding approaches. Many such projects would be primarily of local interest, depending on the diseases which cause the greatest problems in a given region. Examples might include: soil rot in the USA, vine and leaf scab and witches' broom in southeast Asia and Oceania or bacterial wilt and violet root rot in China. The success of such projects would depend on access to resistant germplasm through germplasm collections as well as subsequent enhancement of locally-adapted cultivars.

Identifying sources of resistance to different diseases may be important even for those diseases such as Fusarium wilt, which are now satisfactorily controlled with resistance. The commercial cultivars of sweet potato currently used in the USA have resistance

to Fusarium wilt originally derived from the same source, Tinian (80). That new races of F. oxysporum f. sp. batatas have not yet been observed is fortuitous, but the possibility that new races may appear should not be overlooked.

General 'storagability' has been a criterion of selection in most of the US sweet potato breeding programs for many years. The most widely adopted cultivars, such as 'Jewel' and 'Centennial', seldom totally fail to keep in storage. However, the individual components affecting storagability of different lines are not always identified. The most widely used cultivars, even when cured, develop certain specific storage rots, such as Fusarium root rot and Java black rot, that can limit their storage in some regions. Storage characteristics have not been considered important in the tropical growing areas, in part because sweet potatoes are harvested year round, but also in part because many of the local cultivars do not store well under the prevailing conditions. The availability of cultivars that could be stored for at least a limited time could provide greater flexibility and economic opportunities for sweet potato growers and reduce losses that occur even in the brief time between harvest and consumption as practiced in the tropics.

Resistance has been observed to many individual storage rots, but to be commercially useful a cultivar should have broad spectrum resistance to the whole complex of storage rots. Furthermore, storage rot resistance that improved storagability in situations where curing cannot be practiced would have even greater value. It thus appears that the greatest potential benefit could be obtained from a program aimed at developing lines with broad-spectrum storage rot resistance effective under adverse conditions. While this would necessarily include screening of germplasm for reaction to individual storage rots, it should go beyond that and involve more basic studies of factors that might influence the incidence of the entire storage rot complex. Included might be studies of: the role of latex constituents and oxidized latex in protection of wounded ends of storage roots; factors influencing the rate of wound periderm formation; the involvement of ethylene in wound healing and disease resistance in storage roots; the role of furanoterpenoid phytoalexin-like compounds in storage rot resistance; the influence of duration of storage on susceptibility; the relationship of sugar, organic acid, and dry matter content to storage rot susceptibility; the role of lignified barriers in storage rot resistance; and the influence of storage insects, such as weevils, on storage rot incidence. Each of these factors might be studied with regard both to variation among sweet potato genotypes and with the possibility of developing more generally applicable screening methods for evaluation of resistance to the storage rot complex.

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