Pathogens to be considered by NPDRS

Cronartium flaccidum (Alb. & Schwein.) G. Winter 1880 [1881] (Urediniomycetes, Uredinales)
≡ Sphaeria flaccida Alb. & Schwein. 1805
and other synonyms.
Alternate State (Anamorph): Peridermium cornui Rostr. ex Kleb. 1890
Disease: Scotch pine blister rust; resin canker
Substrate: Stems.
Host: Aecial host: *Pinus* spp.; telial host: Asclepiadaceae, Paeoniaceae, Scrophulariaceae. Occurs on commercially grown species of pine as well as many wild species.

Distribution: Europe especially northern Europe (Finland, Norway, Sweden), south to Italy, Asia (China, Korea, Japan).

Biology and Ecology: Heteroecious rust. Infection through foliage or stem wounds. The heteroecious form of *C. flaccidum* infects *Pinus* needles by airborne basidiospores in the late summer (July to September). The autoecious form directly infects *Pinus* needles or shoots by air- or insect-borne aeciospores through stem and needle wounds or nwounded tissues in the early summer (May to August). The rust overwinters as mycelia in the host shoot.

A latent period of several years may exist between infection and the appearance of aecia on the infected stem. This period is mostly 2-4 years for the autoecious form but may be several years longer for the heteroecious form. For several years after spermatization by insects, aecia are formed on the shoot in the early summer. In Finland, the first aecia mostly developed in 5- to 10-year-old shoots of *Pinus sylvestris*, and sporulation tended to continue for 1-2 years before the infected shoot bearing the lesion was killed.

Aeciospores are disseminated long distances and infect alternate host leaves. A week after the germination of aeciospores on alternate hosts, uredinia develop on the host leaves and urediniospores spread among the alternate hosts. Eight days after aeciospore germination (in June to August), hair-like telia start to develop on the alternate hosts. When the teliospores germinate, basidia develop on the telia and four basidiospores are formed that will re-infect *Pinus* needles. Distance from the alternate host negatively correlates with the degree of damage caused to species of *Pinus*.

Temperature and moisture affect both disease development and sporulation of the rust on hosts and alternate hosts. An increase in relative humidity during the summer may have increased rust epidemics in Greece. The beginning of disease development and uredinia formation are enhanced by high temperatures, whereas telia formation is enhanced by precipitation and slowed down by high

Pathways: Long-distance dispersal of *C. flaccidum* occurs by wind but also insects can transmit spores attracted by sugary exudate. This rust infects seedlings and becomes systemic through leaves, stems and the whole plant. The telial state could be introduced on herbaceous ornamental hosts. A period of years elapses between infection and appearance of the aecial state on an infected stem.

Control measures: *Cronartium flaccidum* has been controlled by intensive removal of diseased trees, but despite this severe epidemics regularly occur throughout its distribution range.

Economic data:

Elsinoë australis Bitanc. & Jenkins 1936
Alternate State (Anamorph):
Sphaceloma australis Bitanc. & Jenk. 1936
≡*Sphaceloma fawcettii* var. *viscosa* Jenk. 1933

Disease: sweet orange scab

Substrate: Fruit

Host: Most important on fruits of orange (*Citrus sinensis*) and mandarin (*C. reticulata*). It also infects lemons (*C. limon*), satsumas (*C. unshiu*), limes (*C. aurantifolia*), *Fortunella* and many other citrus species (Rutaceae).

Distribution: South America

Biology and Ecology: Inoculum for new infections consists of conidia, and possibly ascospores, from scabs formed on fruits. Conidia are formed abundantly on wet scabs, in a nearly saturated atmosphere, between 20 and 28°C. Germination of conidia and infection do not require rainfall, both processes being possible in the presence of free water from dew or fog. A wet period of 2.5-3.5 h is needed for conidial infection. The temperature range required for germination of conidia is 13-32°C, but infection does not take place below 14°C or above 25°C with an incubation period is at least 5 days.

The pathogen is able to survive in scab pustules on fruits remaining on the tree, providing the inoculum for the next season. Even in resistant cultivars, the fungus can survive on diseased shoots from susceptible rootstocks.

Dissemination of the pathogen is mostly by rain (or irrigation water), although insects and, to a certain extent, wind-carried water droplets containing spores may contribute to the spread of the pathogen. In international trade the pathogen can be carried on infected nursery stock, ornamental citrus plants, and fruits.

Pathways: Conidia develop on fruit but the pathogen may be latent in infected nursery stock.

Control measures: Citrus scab can be controlled using resistant cultivars and by fungicide applications both in the nursery and in the orchard. Protectant fungicides may be applied (copper, ferbam, thiram, difenoconazole and chlorothalonil have been used), or systemic fungicides (benomyl, carbendazim) before flushing and after petal fall. Benomyl-tolerant strains of the pathogen have been found. Crop sanitation, establishing citrus nurseries in dry areas or in greenhouses, and adoption of proper treatments, may help in production of rootstocks and budwood free from the pathogen. The usual procedures for importation of certified citrus planting material should be followed.

Economic data:

Guignardia citricarpa Kiely 1948 (Ascomycetes, Dothideales)
Alternate State (Anamorph): Phyllosticta citricarpa (McAlpine) Aa 1973
≡ Phoma citricarpa McAlpine 1899
≡ Phyllostictina citricarpa (McAlpine) Petr. 1953
= Phyllosticta circumsepta Sacc. 1914

The common endophyte *Guignardia mangiferae* was previously referred to as the non-pathogenic strain of *Guignardia citricarpa* but has now been demonstrated to be a distinct species. Historical literature must be interpreted with caution; reports on hosts other than *Citrus* spp. are presumably *Guignardia mangiferae*.

Disease: Citrus black spot.

Substrate: Fruit, leaves.

Host: Apparently restricted to *Citrus* spp. (Rutaceae). Reports on other hosts are doubtful and presumably refer to *Guignardia mangiferae*.

Distribution: Africa (South Africa), Asia, Australia, South America. The collection from North America (USA: FL) was reported on decaying leaves and is therefore presumably *Phyllosticta capitalensis*. Reports from other countries may refer to *Guignardia mangiferae.*

Biology and Ecology: Ascospores from infected, fallen leaves are the major source of inoculum. Sexual state development in decomposing leaves occurs from 40 to 180 days after leaf fall, depending on the frequency of wetting and drying. The optimum temperature for ascomata formation is 21-28°C and no fruiting bodies are formed below 7°C or above 35°C. Prolonged periods of wetness prevent fruiting since the leaves are rapidly colonized by competing saprobes. Once ascospores are mature, rainfall or irrigation may trigger their release. Ascospores are carried by wind throughout the canopy and long distances beyond.

When ascospores are deposited on fruit or vegetative tissues under moist conditions, they germinate to form appressoria. An infection peg penetrates the cuticle and epidermis to form quiescent infections on leaves or fruit. Quiescent infections on fruit develop to produce the typical black spot symptom after the fruit attains full size or becomes mature. Such infection on leaves seldom develops. However, the fungus colonizes the leaf as a saprophyte after the leaf dies and eventually forms pycnidia or pseudothecia.

The anamorph probably plays only a minor role in the disease cycle. Conidia produced on the leaves and fruit in the canopy are capable of infecting the leaves and fruit. However, conidia produced on dead leaves can only reach susceptible fruit and leaves by splash dispersal into the canopy. Conidia produced on fruit can be washed down through the canopy and infect leaves and younger fruit that are still at the susceptible stage.

In most subtropical citrus areas, leaf fall occurs just before or about the time of bloom. Fruit are susceptible for at least 4-5 months after petal fall. Ascospores are released whenever conditions are favorable during that time and produce the quiescent infections. Thus infections probably occur throughout spring until at least mid-summer whenever conditions are favorable. Fruit must be protected during that entire time to achieve a high degree of control.

Black spot has been present in Australia, South Africa and parts of Asia for many years. Most of the losses in Australia and South Africa have been due to the external blemishes that make fruit

unsuitable for the fresh market. Some losses to fruit drop occurred in years favorable for disease development and when fruit was held on the trees past peak maturity. Internal quality of fruit may also be affected. Latent infections are common on leaves and occasionally symptoms appear. No harmful effects from leaf infection have been reported.

Pathways: All plant parts affected including fruits, leaves and stems. This fungus is most likely to be brought into the U.S. on infected nursery stock.

Control measures: Exclusion: Efforts have been made to exclude black spot from areas where it is not currently present. Clearly, living or dead vegetative tissues represent a high risk for introduction since airborne ascospores can be produced on these tissues under favorable conditions. Living trees or even budwood could carry quiescent infections. The importance of dispersal by fruit has been questioned since the fungus produces only water-dispersed conidia on symptomatic fruit.

Sanitation: The removal of infected, off-season fruit may be useful to reduce conidial inoculum in some situations. Mechanical removal of leaf litter from the orchard floor reduces disease pressure and facilitates control, but is costly.

Host-Plant Resistance: Sour orange (*Citrus aurantium*) is one of the few species of citrus that is resistant to black spot. Grapefruit (*C. paradisi*) and lemons (*C. limon*) are the most susceptible, whereas some mandarins (*C. reticulata*) are more tolerant. Some attempts have been made to produce tolerant hybrids using sour orange as a source of resistance. It is unlikely that conventional breeding will produce commercially useful, tolerant cultivars in the foreseeable future. Genetic modification holds promise for developing resistant cultivars.

Chemical Control: A number of fungicides such as copper products, dithiocarbamates, benzimidazoles and strobilurins are effective against black spot. However, resistance has developed to benzimidazoles in many areas and there is a potential problem with strobilurins. Fungicides must be chosen carefully to minimize the possible development of resistance. For many years, protective products such as the coppers and dithiocarbamates were the basis for the control program. Subsequently mid-summer postinfectional applications of benzimidazoles were sufficient for control in many areas. However, with the development of resistance, many growers have returned to the use of protectant sprays or combinations of systemic and protectant products.

Spore trapping and rainfall and dew measurements have been helpful in determining the timing of ascospore release and the need for fungicide applications in South Africa. In Brazil, infections seem to occur to varying degrees throughout the susceptible period and fruit must be protected from petal fall to mid-summer.

Postharvest: Fruit from black spot-infested groves often bear quiescent infections that may later develop into black spot lesions in transport or at the final destination. Fruit produced for the fresh market should be refrigerated and kept as cold as possible to slow development of the lesions. Preharvest sprays of benzimidazole fungicides are effective in preventing or delaying symptom expression during transport or storage. Postharvest applications of fungicides are generally less effective in preventing symptom development. However, treatment with guazatine or imazalil, hot water or waxing decreased the viability of the pathogen in black spot lesions. Postharvest waxing also decreases the manifestation of symptoms following postharvest storage.

Economic data:

Harpophora maydis (Samra, Sabet & Hing.) W. Gams 2000
≡ Cephalosporium maydis Samra, Sabet & Hing. 1963
[≡ Acremonium maydis (Samra, Sabet & Hing) ???, never published in this genus, although name is used by EPPO]
Ascomycetes, Incertae sedis, related to *Gaeumannomyces graminis*, cause of take all disease of wheat

Disease: Late wilt of corn, black bundle disease.

Substrate: Infects seedlings through roots or mesocotyl, occurs on seeds, leaves and stems.

Host: *Zea* (Poaceae)

Distribution: Asia (India), Africa (Egypt), also reported from Hungary.

Biology and Ecology: The disease is primarily soilborne and may infect maize through the roots or mesocotyl. *H. maydis* was detected in 42 out of 43 seed samples in Egypt, at infection levels up to 11%. Seed infection was induced in plants inoculated at planting time detected *H. maydis* in a higher percentage of white maize seeds (1-9%) than in yellow cultivars (1-3%) in Hungary. The fungus was detected in different ear parts, i.e., ear branch, cob, seeds, ear husks and silk of naturally infected maize cultivars. It was mostly manifested in the branch followed by cob, seeds, husks and silk. *H. maydis* is internally and externally seedborne in maize as it was detected at higher levels in the embryo and in both the endosperm and coat of the 13 tested seed samples, with the exception of seeds of cv. Amon in which the fungus was confined to the seed coat.

Seed rot and reduced emergence resulted from infesting soil with *H. maydis*. This effect has not been demonstrated with natural seedborne inoculum.

Disease Impact: This is a late-season disease of widespread incidence and severity in Egypt, with 100% infection reported in some fields.

Pathways: This pathogen is soilborne, with sclerotia surviving on maize debris; it could also enter on infected germplasm. It may also be seedborne as this fungus causes a seed rot and reduced emergence, although natural seedborne inoculum has not been demonstrated.

Control measures: Seed treatment with carbendazim or captan gave effective control of late wilt of maize in India. Apparently some genetic resistance exists. No data on disease control.

Economic data:

Monilinia fructigena Honey ex Whetzel 1946 (Ascomycetes, Helotiales)
Alternate State (Anamorph): Monilia fructigena Pers.:Fr. 1801
≡ Oospora fructigena (Pers.:Fr.) Wallr. 1833
≡ Sclerotinia fructigena (Pers.:Fr.) J. Schröt. 1893
≡ Acrosporium fructigenum (Pers.) Pers. 1822
= Oospora candida Wallr. 1833
= Torula fructigena Pers. 1796
= Oidium fructigenum Kunze & J.C. Schmidt 1817
= Oidium wallrothii Thüm. 1875

Disease: Brown fruit rot.

Substrate: Primarily fruits, rarely blossoms and twigs. Overwinters in mummified fruits.

Host: *Malus* spp., *Pyrus* spp., *Prunus* spp. and other Rosaceae, also reported on *Vitis vinifera* (Vitaceae).

Distribution: Europe, Asia (Japan, China). Also reported in South America (Brazil, Uruguay), North America (MD). Although reported from MD, the fungus did not spread and was eradicated. Reports in Japan probably refer to *Monilia polystroma* Leeuwen 2002, a recently described taxon.

Biology and Ecology:

The conidia of *M. fructigena* are dry air spores. The spores are not actively discharged but are set free by air currents and wind. Also, except when mummified fruit have fallen to the ground, infected fruits and peduncles are in positions that are well placed for efficient take-off and aerial dispersal of the spores they bear. Rain splashes are also important as a means of liberating spores. Aerial dispersal results in the spread of spores over a wide area, whilst water splash dispersal brings about only short-range dissemination, mainly to other parts of the same tree or, in some instances, between adjacent trees. Animals are important vectors of this fungus, either incidentally or because of complex adaptations. Almost any insect has the potential to pick up and carry spores from sporulating mycelium to healthy, susceptible tissues.

The fungus overwinter mainly in or on diseased mummified fruit either attached to the tree or on the ground. Other infected tissues on trees such as twigs, peduncles and cankers on twigs or branches could also serve as primary inocula. In the spring or early summer when temperatures, day-length and relative humidities are suitable for sporulation, tufts of conidiophores form sporodochia on the surface of the mummified fruit and infected tissues bear chains of asexual spores (conidia). This is the *Monilia* stage of the fungus. Initial infection is always via wounds, usually scab lesions or sites of insect damage, but subsequent spread by contact between adjacent fruit is possible. After initial penetration of fruits there is active mycelial growth, and the hyphae in the outer tissues of the fruit become closely interwoven to form a stroma. Fruits may become infected at harvest time and then fruit rots develop during the postharvest period. The mycelia survive long periods of adverse environmental conditions within mummified fruits, twigs, cankers and the other infected tissues. When conditions become favourable (after a dormant period), spores are produced on infected tissues and the fungus is dispersed and propagated. Thus a new cycle of infection is started which coincides with early spring growth of host plants.

Apothecia are produced in spring on mummified fruit that have overwintered on the ground. Mummified fruits that remain on the tree do not produce apothecia. The liberation of ascospores normally coincides with the emergence of young shoots and blossoms of plants. When spores

alight on susceptible tissues under favorable environmental conditions infections are initiated. In addition microconidia are produced in abundance within small cavities and on the surfaces of mummified fruit.

Although *Monilinia fructigena* causes significant losses both before and after harvest, it is not easy to assess the overall losses it causes in a country, or on a worldwide scale. Losses are highly visible to the grower, but are rarely worth the implementation of specific control measures in their own right. Early-maturing cultivars are most affected, but the majority of diseased fruits are those that would in any case be rejected for other reasons such as bruising, or bird and insect damage.

Monilinia fructigena is less damaging than *M. fructicola* or *M. laxa*, although it occasionally causes economically important losses of apple and plum fruits in Europe, particularly in hot and humid summers.

Pathways: This fungus affects all plant parts and could enter the U.S. on infected nursery stock.

Control measures: Losses from *M. fructigena* are rarely worth specific control measures in their own right. Apart from avoiding very susceptible cultivars in disease-prone districts, few control measures are specifically aimed at this fungus. By reducing the amount of inoculum produced, fungicides used for the routine control of foliar diseases provide an element of incidental control that in practice is often sufficient. Prospects for resistance breeding are limited at least in apple since most resistant cultivars are of the cider type with fruit characteristics unsuitable for dessert use. However, much research has been done in Europe in breeding for resistance, particularly in Eastern Europe.

Economic data:

Peronosclerospora sacchari (T. Miyake) Shirai & Hara 1927 (Oomycetes, Sclerosporales)
≡ Sclerospora sacchari T. Miyake 1912 [1911]

Disease: Downy mildew of sugarcane.

Substrate: Leaves; seed-borne.

Host: Principal host: sugar cane, *Saccharum officinarum*; also corn, *Zea mays* and other Poaceae.

Distribution: Asia (India, China, Japan, Papua New Guinea, Philippines, Taiwan, Viet nam), Australia, South America (Argentina, Venezuela). Reports from Central America and Africa (Nigeria) are unsubstantiated.

Biology and Ecology:

Conidia of *Peronosclerospora sacchari* released from diseased sugarcane plants in maize fields is recognized as the primary inoculum source of the pathogen for maize. Shedding of conidia lasted 7 h, starting 5-6 h after sugarcane leaves were exposed to high humidity; the fungus regained the ability to shed conidia after c. 10 h. A diurnal sporulation cycle was observed. Most conidia were produced on younger leaves and none on red discolored tissues. On rainy days there was no sporulation.

Oospores of *P. sacchari* are produced in both maize and sugarcane. They also can infect sugarcane plants under glasshouse conditions, but their role as an inoculum source under natural conditions has not been demonstrated. Sugarcane downy mildew developed on maize in seedlings grown in autoclaved soil from seeds obtained from infected plants, but these were freshly harvested seeds that had not been dried or stored. No transmission of *P. sacchari* occurred from maize seeds dried to less than 20% moisture. The only important means of 'seed' transmission of *P. sacchari* is through infected sugarcane sets. Conidia of *P. sacchari* that are produced in abundance on maize are considered to be a source of inoculum for sugarcane grown in the vicinity of maize fields.

During the leaf infection process, conidiophores appeared on the surface of maize leaves 5 h after inoculation; mature conidia were discharged after incubation for 8 h. Periodic epidemics of sugarcane downy mildew have occurred in Taiwan. The damage to maize crops is usually greater than that to sugarcane. In Fiji, sugarcane downy mildew at one time caused major losses sugarcane-growing areas, but these were reduced to very low levels by the release of resistant cultivars coupled with an intensive system of disease control.

Pathways: The pathogen occurs on all plant parts including seeds.

Control Measures: Control measures include removal of infected maize and other hosts within at least 42 m of newly sown seed (outside the range of conidial dissemination); rogueing of any infected plants from adjacent fields; sowing at the same time within an area to prevent cross-infection during different stages of growth; removal and burning of infected plants to prevent the provision of sources of primary infection; and using seed only after drying to 13-14% moisture.

Resistant maize hybrids have given give good control of sugarcane downy mildew.

Fungicides (e.g. metalaxyl and mancozeb), vegetable oil, and calcium hypochlorite sprays

controlled systemic infection by *P. sacchari* and increased yield of maize. Chloroneb was effective as an in-furrow application. Seed treatments with metalaxyl gave 100% control of disease development in the field and had no adverse effect on germination. Chloroneb also gave good control.

The fungicidal activity of acylalanine against *P. sacchari* in sugarcane was fully effective only in the main crop: the disease reappeared in the first and subsequent ratoons. The expense of using this compound as a preplant dip and foliar spray on the susceptible cultivar Q 90 was only justified in the absence of a resistant cultivar of equal agronomic qualities

Economic data:

Phytophthora kernoviae Brasier, Beales & S.A. Kirk 2005 (Oomycetes, Pythiales)

Disease: On beech, associated with bark necrosis and canker and bleeding stem lesions, especially on aerial stems. Pathogenicity confirmed by inoculation studies. Also associated with shoot dieback, foliar necroses and wilting of rhododendron. Previously reported as *Phytophthora* taxon C.

Substrate: Bark, leaves, shoots.

Host: *Fagus sylvatica* (beech), *Quercus robur* (oak) (Fagaceae); also *Liriodendron tulipifera* and *Michelia doltsopa* (Magnoliaceae), *Rhododendron ponticum* and other members of the Ericaceae.

Distribution: Europe (UK), at present restricted to local woodlands in Cornwall plus single nearby locations.

Biology and Ecology: This species infects foliage and shoots of understory plants from which it spreads aerially to attack the inner bark of tree stems causing bleeding lesions. Leaf infection results in blackening of the leaf petiole, often extending into the base of the leaf or causing necrosis of the entire leaf. Both young and old leaves affected equally, often falling within weeks of infection. Also, causing dieback of shoots and cankers, girdling the stem with leave wilt above the lesion. This species is self-fertile in culture, apparently an inbreeding species.

Pathways: Adapted for splash or wind dispersal. Four invasive species of *Phytophthora* in the UK appear to have been introduced by the international nursery trade.

Control: None known at present.

Economic data:

Literature:

Beales, P.A., Lane, C.R., Barton, V.C., and Giltrap, P.M. 2006. *Phytophthora kernoviae* on ornamentals in the UK. Bull. OEPP/EPPO Bull. 36: 377-379.

Brasier, C.M., Beales, P.A., Kirk, S.A., Denman, S., and Rose, J. 2005. *Phytophthora kernoviae* sp. nov., an invasive pathogen causing bleeding stem lesions on forest trees and foliar necrosis of ornamentals in the UK. Mycol. Res. 109: 853-859.

Phytophthora quercina T. Jung 1999 (Oomycetes, Pythiales)

Disease: Implicated in European oak decline, associated with other *Phytophthora* spp.

Substrate: Roots, rhizosphere soil.

Host: *Quercus* spp. (Fagaceae).

Distribution: Asia (Turkey), Europe (Austria, Belgium, France, Germany, Hungary, Italy, Sweden, United Kingdom).

Biology and Ecology: This relatively recently described species of *Phytophthora* is an oak-specific, fine root pathogen that appears to have been introduced and then spread rapidly. Two subgroups can be distinguished suggesting introduction of isolates of two different genetic backgrounds. This species is predominantly homothallic with occasionally outcrossing and presence of long-lived soil-borne oospores. Pathogenicity may be correlated with acid forest soils and restricted water availability resulting in stress-induced susceptibility of seedlings or a more aggressive pathogen. No significant effects have been found to occur on above-ground growth. This species has spread to northern European countries and Turkey since its original discovery in central and southern Europe in 1999.

Pathways: Appears limited to species of *Quercus* especially roots.

Control measures: None known.

Economic data:

Literature:

Cooke, D.E.L., Jung, T., Williams, N.A., Schubert, R., Bahnweg, G., Oswald, W., and Duncan, J.M. 1999. Molecular evidence supports *Phytophthora quercina* as a distinct species. Mycol. Res. 103: 799-804

Jung, T., Cooke, D.E.L., Blaschke, H., Duncan, J.M., and Oswald, W. 1999. *Phytophthora quercina* sp. nov., causing root rot of European oaks. Mycol. Res. 103: 785-798

Pseudocercospora angolensis (T. Carvalho & O. Mendes) Crous & U. Braun
= *Cercospora angolensis* T. Carvalho & O. Mendes
= *Phaeoramularia angolensis* (T. Carvalho & O. Mendes) P.M. Kirk
= *Pseudophaeoramularia angolensis* (T. Carvalho & O. Mendes) U. Braun

Disease: Phaeoramularia fruit and leaf spot of citrus

Substrate: Leaves, stems and fruit.

Host: All species of *Citrus*, reported on *Citrus aurantiifolia* (lime), *Citrus aurantium* (sour orange), *Citrus limon* (lemon), *Citrus reticulata* (mandarin), *Citrus sinensis* (navel orange), *Citrus* x *paradisi* (grapefruit). Smooth lemon reported to be relatively resistant.

Distribution: Central to southern Africa as far north as Ethiopia, recently discovered in Swaziland and South Africa; Yemen. The disease appears to be restricted to the humid tropics in Africa, between altitudes of 80 and 1500 m.

Biology and Ecology: The disease is favored by prolonged wet weather conditions followed by dry spells coupled with moderately cool temperatures of 22-26°C. At the onset of the rainy season, new disease-free leaves are formed while on older leaves varying numbers of non-sporulating lesions are present. These lesions begin to sporulate after a further 3-5 weeks and new symptoms appear on young leaves 2-3 weeks later.

Long-distance dispersal of the fungus is by windborne conidia; within the tree it is primarily by rain-splash or raindrops carrying conidia. Undoubtedly humans mediate in the dissemination of the disease through inadvertent movement of infected plant material and/or fruits from infected areas. Because leaf lesions produce more conidia than similar lesions on fruit it is most likely that they constitute the main source of infection during disease spread in infected areas.

Survival mechanisms are unknown; the fungus probably survives as dormant lesions on infected material until the onset of conditions conducive to sporulation.

Disease Impact: *Pseudocercospora angolensis* often causes a significant disease of citrus, the most devastating effect being the premature abscission of young fruit and leaves and the development of fruit lesions that render the fruit unmarketable. A yield loss of 50-100% is not uncommon in disease-affected areas. The loss of leaves can have a significant debilitating effect on the tree that is likely to affect subsequent fruit yields.

Pathways: This fungus is most likely to be brought into the U.S. on infected fruit or infected nursery stock.

Control measures: The most effective fungicides of a range that were tested on fruit and leaf spot of citrus in Cameroon were copper oxide and benomyl. Others found to be effective were mancozeb, tridemorph, triadimenol, captafol. Treatment with benomyl, alternated with copper-based fungicides, is effective. Sprays are applied a week after the onset of rains and every two weeks. A further three applications with copper-based fungicides followed by benomyl should be made when the fruits are the size of golf balls.

Economic data:

Pseudopezicula tracheiphila (Müll.-Thurg.) Korf & W.Y. Zhuang 1986 (Ascomycetes, Helotiales)
≡ Pseudopeziza tracheiphila Müll.-Thurg. 1903
Alternate State (Anamorph): Phialophora tracheiphila (Sacc. & D. Sacc.) Korf 1986
≡ Botrytis tracheiphila Sacc. & D. Sacc. 1906

Disease: Angular leaf scorch disease of grape(Rotbrenner).

Substrate: Leaves, associated with vascular tissue.

Host: *Vitis* spp., *Parthenocissus* spp. (Vitaceae).

Distribution: Europe, Asia (Jordan, Turkey), Africa (Tunisia). Collections in North America and possibly also in South America (Brazil) are referred to *Pseudopezicula tetraspora* (Korf et al. 1986).

Biology and Ecology: This disease causes significant losses in some years mainly in Germany & eastern France. Symptoms are lesions on leaves, that are initially yellow or bright red to reddish brown. Later, a reddish brown necrosis develops. Early infections generally result in less loss than late infections. Inflorescences can be attacked before or during flowering causing them to rot or dry out. High levels of infection during flowering can lead to severe yield losses of up to 90%. Heavy rainfall and prolonged periods of leaf wetness favor infections. This species has been confused with *Pseudopezicula tetraspora* Korf et al., cause of angular leaf spot of grape that occurs in North and South America.

Pathways: This fungus is most likely to be brought into the U.S. on infected nursery stock.

Control: This disease can be controlled with systemic fungicides.

Economic data:

Pyrenochaeta glycines R.B. Stewart 1957 (Ascomycetes, Pleosporales)
≡ Dactuliochaeta glycines (R.B. Stewart) G.L. Hartm. & J.B. Sinclair 1988
Phoma glycinicola Gruyter & Boerema 2002
Dactuliophora glycines C.L. Leakey 1946

Disease: Soybean red leaf blotch. Causes leaf spot and leaf abscission in susceptible varieties of soybean.

Substrate: Living leaves.

Host: *Glycine* spp. and *Neonotonia wightii*, a perennial relative of soybeans (Fabaceae).

Distribution: Africa (widespread), Asia (India-questionable), South America (Bolivia-one report).

Biology and Ecology: This pathogen causes lesions on the foliage, petioles, pods and stems of soybeans throughout the growing season. These can develop into necrotic tissue with a shot-hole appearance and the plants may defoliate prematurely and senesce 5-10 days before normal maturation, causing up to 75% defoliation, contributing to yield loses. Pycnidia form within the blotches. During lesion enlargement, sclerotia develop usually on lower leaf surfaces. With the increase in production of soybeans in southern Africa, the incidence of this disease has increased. This disease frequently develop on soybeans planted into newly cleared land suggesting that this fungus may also infect other hosts. The fungus is apparently not internally seedborne although incidental transmission may occur through seed lots especially those contaminated with plant debris or soil. Soilborne sclerotia may be rain-splashed onto leaf surfaces where they germinate and infect the host. Infection occurs and symptoms appear in 2-7 days. Most commercial cultivars of soybean grown in the U.S. in 1987 were susceptible.

Pathways: Infected plant material or seedlots with debris.

Control: No commercial control for this disease is known, although fungicide sprays reduced disease progress. In Zambia where the disease occurs, fentin acetate or benomyl were effective in controlling the disease.

Economic data:

Literature:

Hartman, G.L., Datnoff, L.E., Levy, C., Sinclair, J.B., Cole, D.L., and Javaheri, F. 1987. Red leaf blotch of soybeans. Pl. Dis. 71: 113-118

Thekopsora areolata (Fr.:Fr.) Magnus 1875 (Urediniomycetes, Uredinales)
Variant spelling Thecopsora areolata (Fr.:Fr.) Magnus
≡ Xyloma areolatum Fr.:Fr. 1817
≡ Melampsora areolata (Fr.:Fr.) Fr. 1849
≡ **Pucciniastrum areolatum** (Fr.:Fr.) G.H. Otth 1863 (This is the name used by CABI)
≡ Sclerotium areolatum (Fr.:Fr.) Fr. 1822
= **Pucciniastrum padi** (Kunze & J.C. Schmidt) Dietel 1897 (This name is also used)
plus many additional synonyms
Alternate State (Anamorph):
Aecidium strobilinum (Alb. & Schwein.:Fr.) Reess 1869
plus many additional synonyms

Disease: cherry spruce rust, spruce blister rust

Substrate: Heteroecious rust.

Host: Produces aecia on spruce cone scales (*Picea* spp., Pinaceae) and telia on leaves of *Prunus* spp. esp. *P. padus* and *P. serotina* (Rosaceae).

Distribution: Europe incl. Russia, Asia, Caribbean (Dominican Republic).

Biology and Ecology: *Thekopsora areolata* is a heteroecious rust that produces uredinia and telia on *Prunus* and some other members of the Rosaceae. Aecia are produced on cones of *Picea* and sometimes on young stems where the rust can cause twisting and distortion. According to one source, this pathogen is of economic importance only in seed orchards where it caused failure of the seed crop. The aecia develop in summer and mature on fallen cones in the same years; their spores germinate the following May and then infect the leaves of *Prunus*, on which they produce uredospores in the summer and teliospores in the fall. Norway spruce shoots especially terminal ones, often become infected mycelium and assume an S-shape and undergo necrosis, although aecia are few or absent on the shoots. The rust causes a serious shot-hole disease on plums.

Pathways: Not much information but obviously this fungus is seedborne on spruce.

Control measures: None were found in the literature except for biological control with *Trichoderma*. Although common, apparently this is not a serious disease.

Economic data:

***Heterodera latipons*- The Mediterranean Cereal Cyst Nematode**

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Binomial: *Heterodera latipons* Franklin, 1969

Later named as *Bidera latipons* (Franklin) Krall & Krall, 1978; *Ephippiodera latipons* (Franklin) Shagalina & Krall, 1981. However, these two names have **not** been widely used.

Disease information; Description; History:

Stunted, chlorotic wheat plants with cyst nematodes on the roots were sent in 1960 from Libya to England, where Hesling (1965) indicated that they represented a new species that was formally described by Franklin (1969). When compared to other cyst nematode species, there is nothing remarkable about the life cycle of this species. *Heterodera* latipons will thrive in warmer and drier climates that the more common cereal cyst nematode (*Heterodera avenae*) will not tolerate. Symptoms include stunting, chlorosis, and yield loss (as high as 50%); examination of the roots reveals brownish nematode cysts distributed along the roots.

Substrate (e.g. leaves, vascular tissue, etc.)

Cysts may persist in soil for several years. Juvenile nematodes emerge from eggs, penetrate roots, and establish permanent feeding sites, where females produce hundreds of eggs within their bodies, which eventually turn into the cysts.

Pathogen Distribution (include references):

Algeria (Bekal et al., 1997), Bulgaria (Stoyanov, 1982), Canada (Prince Edward Island; Sewell, 1977), Cyprus (Philis, 1988), Czech Republic (Sabová et al., 1988), France (Rivoal et al., 2003), Greece (Mulvey, 1972), Iran (Sturhan, 1996), Israel (Franklin, 1969), Italy (Tacconi, 1976), Japan (Momota, 1979), Jordan (Al-Abed et al., 2004) Libya (Hesling, 1965), Poland (Mulvey, 1972), Syria (Bekal et al., 1997), Russia (Subbotin et al., 1998), Spain (Romero, 1980), Tunisia (Swarup and Sosa-Moss, 1990), Turkmenistan (Sturhan and Wouts, 1995), and Turkey (Rumpenhorst et al., 1996).

Commodity Host:

The host range of *H.* latipons has not been studied adequately. The nematode infects many cereals and grasses, including barley, wheat, oat, and rye. Two reports of association with sugarbeet and carrot soils exist, but it is unclear if cysts were observed on roots in these studies.

Host Resistance identified? (include genotypes if known)

Unfortunately, resistance to *H. latipons* has not been adequately studied; in non-wheat hosts, it has not been studied. Resistance has been detected in *Aegilops tauschii* ssp. e*usuarrosa* var. *meyeri* AUS 18913, *A. longissima*, *A. geniculata*, *Triticum ovatum*, T. *variabile*, and *T. aestivum* AUS 4930. Additionally, the nematode species appears to contain pathotypes that vary in virulence toward hosts of different genetic composition. Resistance of U.S. cultivars to the species has not been the subject of specific investigations.

Vector and vector distribution

As with most cyst nematodes, long-distance dissemination of *H. latipons* occurs via movement of soil or plant roots from infested areas to noninfested areas.

Pathways of introduction:

The most obvious pathway for introduction of *H. latipons* into the United States is through movement of soil attached to root crops, plant materials, vehicles, or other machinery.

Control measures:
Nematicide expense plus the limited host range indicates that rotations with non-hosts would be the most effective means of control. Unfortunately, the survival of nematodes in the soil is unknown. If analogies to other cyst nematodes can be drawn, the rotations could be five years or longer.

Economic data:

Commodity Host annual value: $
Crop yield losses in native range: $
Export value; Losses due to export disruptions: $
Economic Impact of Regulatory actions: $

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Apple Proliferation Disease

Michael Berg and Jacqueline Fletcher

Description

Apple proliferation, also known as apple witches' broom, is a serious disease of apple trees caused by phloem inhabiting bacteria. The disease was first reported in Italy and has significant economic importance throughout Europe. The causal agent, *Candidatus* Phytoplasma mali, is transmitted through grafting or by phloem feeding insects, most likely leafhoppers or psyllids. *Cacopsylla picta* has been shown to transmit the apple proliferation phytoplasma in Europe. Apple proliferation has only recently been considered an important disease of fruit trees in the U.S., although pear decline, which is caused by a closely related phytoplasma, *Candidatus* Phytoplasma pyri, has been reported in North America since the 1950s.

Symptoms

Symptoms of the apple proliferation disease are diverse and may include the premature development of axillary buds to produce a proliferation of secondary shoots ('witches' broom) near the apex of the main shoot. Leafs with enlarged stipules as well as irregularly serrated, small, and chlorotic leafs are typical for the disease. They often emerge earlier and may exhibit early autumn color (lilac or purplish red). Fruits are smaller, incompletely colored and less flavorful. Development of the root system is repressed leading to reduced overall growth and vigor. Loss in crop production has been reported to be as high as 60-70%. With severe infection the trees may die.

Causal agent

Candidatus Phytoplasma mali is a phytoplasma, which is a distinct group of wall-less, non-helical plant pathogenic bacteria of the Class Mollicutes. Phytoplasmas, which remain uncultured, are associated with diseases in about 1000 plant species. The apple proliferation phytoplasma is closely related to the causal agent of pear decline (PD), European stone fruit yellows (ESFY) and peach yellow leaf roll (PYLR), the latter of which is found in western North America. The phloem inhabiting phytoplasmas often cause diverse symptoms seemingly associated with plant hormone or nutrient imbalance. Detection and characterization of phytoplasmas relies on DNA technology (PCR and RFLP).

Control

Currently, the use of healthy material for grafting and resistant rootstocks provides the most practical and efficient method to control the disease. Symptoms such as early autumn color and small fruits should be monitored and diseased trees eradicated. Since root-to-root transmission has been discussed, replacement of infected trees should be delayed for at least a year. Insect control has also been considered for disease control. However, *Cacopsylla* has proven to be resilient as insecticide treatments were ineffective.