

Soybean Diseases

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Soybean diseases can be a significant economic factor in Arkansas. On average, diseases reduce yields in the state by an estimated 10%, although in individual fields and with certain diseases, losses may be much higher. Because soybean pathogens are common statewide, accurate disease identification and an awareness of the potential for disease losses are essential for the continued success of Arkansas soybean production. The following symptom descriptions and color photographs of common diseases in Arkansas should help you identify and manage soybean diseases before they become a yield-limiting problem.

Bacterial Diseases

Bacterial Blight

Bacterial blight is the most common bacterial disease of soybeans and occurs in all soybean-producing regions of the world. Although this disease is of limited importance in most Arkansas production areas, it is one of the first leaf spot diseases to appear on young plants. Bacterial blight has been reported to cause significant yield reductions on susceptible cultivars under heavy disease pressure.

Bacterial blight is primarily a leaf disease, but symptoms can occur on stems, petioles and pods. Leaf symptoms begin as small, angular, translucent, water-soaked yellow to light-brown spots. As the spots age, their centers darken to a reddish-brown, become sunken and are surrounded by a water-soaked margin bordered by a yellowish-green halo (Figure 11-1). The halo is more noticeable on the upper leaf surface. When environmental conditions favor disease development, spots may enlarge and merge to form large, irregular, necrotic areas. These large dead areas of the leaf often fall out or tear away after strong winds and beating rains, giving leaves a ragged appearance. When disease is severe, premature defoliation may occur.

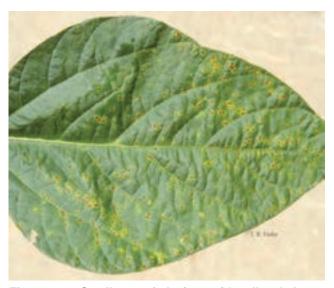


Figure 11-1. Small necrotic lesions with yellow halos caused by bacterial blight. (Photo by T. R. Faske)

Leaf symptoms of bacterial blight may resemble those of the fungal disease brown spot. However, bacterial blight moves upward within the canopy rapidly whereas upward movement is slow for brown spot. A simple test for bacterial blight is to hold infected leaves to the light; bacterial blight spots will be translucent.

Bacterial blight is caused by the bacterium *Pseudomonas savastanoi* pv. *glycinea* which overwinters in seed and infected soybean debris. Lesions develop on cotyledons from infected seed and can spread to cause secondary infection on leaves. The pathogen enters the plant through stomata or wounds. The bacterium is spread from infected tissue by wind-blown rain and during cultivation or spraying when the foliage is wet. Seeds can be infected through the pods during the growing season. Windy, cool, rainy weather at temperatures of 75° to 79°F favor the development of bacterial blight whereas hot, dry weather suppresses its development.

Management of this disease is primarily dependent on cultivars that are resistant to bacterial blight. Cultural management practices consist of planting high-quality, disease-free seed and using tillage practices that lead to rapid decomposition of crop residue. Narrow row widths and high plant populations should be avoided in fields with a history of bacterial blight.

Bacterial Pustule

Bacterial pustule has been reported worldwide. In Arkansas, bacterial pustule is not as common as another bacterial disease, bacterial blight, and is of minor importance because of the availability of highly resistant cultivars.

In susceptible cultivars, early symptoms are characterized by small yellow-green spots with elevated reddish-brown centers that are most conspicuous on upper leaf surfaces (Figure 11-2). As these leaf spots mature, a small, slightly raised, pale-colored pustule develops at the center of each lesion that is most noticeable on lower leaf surfaces. Leaf lesions vary from very small specks to large, irregular, mottled necrotic areas depending on the susceptibility of the cultivar and environmental conditions. Diseased leaves develop a ragged appearance when the necrotic areas are torn away by stormy or windy weather. Severe infection often results in premature defoliation that may decrease yield by reducing seed numbers and size.



Figure 11-2. Small, reddish brown lesions caused by bacterial pustule. (Photo by C. Coker)

Symptoms of bacterial pustule may resemble those of bacterial blight, and it is common for both diseases to occur together. Pustule formation and the absence of a water-soaked appearance during the early stages of lesion development (before the leaf spots turn yellow) distinguish bacterial pustule from bacterial blight.

Bacterial pustule is caused by *Xanthomonas* axonopodis pv. glycines that overwinters in infested seed and soil on crop residue. The bacteria spread from crop residue or nearby diseased plants by splashing water, windblown rain and during cultivation when the foliage is wet. The bacterium enters the plant through stomata and wounds. Disease development occurs during warm (86° to 91°F), wet weather conditions.

Management of this disease is primarily dependent on the use of cultivars that are resistant to bacterial pustule. Cultural practices include planting high-quality, disease-free seed and using tillage practices that hasten rapid decomposition of crop residue. Cultivation when foliage is wet should be avoided to reduce disease spread.

Fungal Diseases

Anthracnose

Anthracnose occurs worldwide and reduces plant stand, seed quality and yield by 16% to 26% in the U.S. Anthracnose is also an important disease in Arkansas with pod infections contributing to a greater impact on yield loss than stem or petiole infections.

Soybeans are susceptible at all stages of development. Pre- and post-emergence damping-off occurs when infected seeds are planted. On emerging seedlings, dark brown, sunken lesions develop on the cotyledons. These lesions can extend along the stem when conditions favor disease development, causing one or both cotyledons to become water soaked, wither and abscise from the stem. Under severe disease development, numerous small lesions may kill young plants.

Foliar symptoms often occur at early reproductive growth stages with irregular-shaped brown lesions that develop on stems, petioles and pods. Premature defoliation may occur throughout the canopy on maturing plants when anthracnose lesions girdle the leaf petiole, resulting in a shepherd's crook symptom (Figure 11-3). Early-season infection of pods or pedicels can result in fewer and smaller seed or no seed development.

At advanced stages of disease development, near soybean maturity (R7-R8), black fungal fruiting bodies called acervuli that produce minute black spines (setae) are abundant and randomly distributed on infected tissue. Setae are diagnostic of anthracnose and may be seen with a good hand lens or a dissecting microscope (Figure 11-4). In contrast, the fungal



Figure 11-3. Shepherd's crook caused by anthracnose canker on a petiole. (Photo by T. R. Faske)

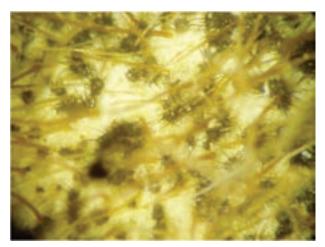


Figure 11-4. Acervuli of Colletotrichum truncatum on soybean pod. (Photo by A. Greer)

fruiting bodies of another common pathogen on pods and stems, *Diaporthe phaseolorum*, which causes pod and stem blight, do not contain setae and are often arranged in rows.

Anthracnose is caused by *Colletotrichum trunca-tum* and several related species, which overseason as mycelium on crop residue or in infected seed. Infected seed may result in damping-off, or seedlings may become infected and colonized by the fungus without symptom development until early reproductive stages. Warm, wet weather favors stem and pod infection whereas dry weather suppresses disease severity.

Management of anthracnose includes the use of high-quality, disease-free seed and tillage or rotation practices that reduce soybean residue. Applying a fungicide between beginning pod development (R3) and initial seed formation (R5) can be effective at suppressing anthracnose. Fungicide seed treatments are also effective at minimizing the effects of anthracnose on seedlings.

Brown Spot

Brown spot or Septoria leaf spot has been reported throughout the southern U.S. soybean-growing region. This disease can cause premature defoliation that contributes to yield losses when susceptible cultivars are planted and conditions favor disease development. This disease is of minor importance in Arkansas because it rarely causes significant yield losses.

Irregular, small brown leaf spots vary in size from a small speck to 1/5 inch in diameter and develop on the upper and lower leaf surfaces. Adjacent leaf spots may coalesce resulting in irregular-shaped blotches. The lesions gradually darken to blackish brown and often develop a yellow halo around the leaf spot (Figure 11-5). Additionally, irregular-shaped brown lesions with undefined margins may form on the stem, petioles and pods. Though leaf spots are generally confined to the lower canopy, the disease may progress to the upper canopy under favorable environmental conditions. As the plant nears maturity, severely infected leaves appear rusty brown and may drop prematurely.



Figure 11-5. Brown lesions with yellow halo caused by brown spot. (Photo by T. R. Faske)

A visual examination of infected leaves by holding them to light reveals that brown spot leaf lesions are dark and opaque. In contrast, bacterial blight lesions, which may appear similar to the casual observer, are translucent.

Brown spot is caused by the fungus *Septoria glycines*, which overwinters on crop residue and in infected seed. Initial infections develop on cotyledons and leaves from conidia (spores) discharged from pycnidia, which are flask-shaped fruiting structures

that form on crop debris. Conidia germinate on leaf surfaces and enter the plants through stomata. Secondary infection occurs as conidia are dispersed upward in the canopy by wind or splashing rain on leaves, petioles, stems and pods.

Infection and disease development may occur at any time during the season. Optimum conditions for disease development are warm (79° to 83°F), wet weather. Hot, dry weather conditions, on the other hand, suppress disease development. Consequently, brown spot is most severe when soybeans are planted early, particularly after extended periods of rainfall, where soybeans are grown continuously in the same field, or when the crop is planted in poorly drained fields.

Soybean cultivars vary in susceptibility to brown spot, so planting a less susceptible, adapted cultivar will suppress this disease. Cultural practices that may help minimize brown spot include planting high-quality seed, rotating with non-host crops (corn, cotton, rice or grain sorghum) for 2 years and implementing tillage practices that reduce crop residue on the surface of the field. In rare situations where brown spot is severe enough to pose a threat to yield, and where yield potential is high and conditions favor continued disease severity, a fungicide application timed between beginning pod fill (R3) and initial seed formation (R5) can be effective in minimizing yield loss.

Cercospora Leaf Blight and Purple Seed Stain

Cercospora leaf blight and purple seed stain are caused by the same fungal pathogen. Disease development typically occurs late in the growing season from beginning of seed development through pod fill. Though both diseases have been reported in all soybean-growing regions of the U.S., yield losses are higher in southern states.

The first visible symptom of Cercospora leaf blight is a light purple discoloration on the upper leaf surface. This discoloration can deepen and expand to cover part or the entire upper leaf surface, giving a leather appearance, sometimes mistaken for sunburn. Numerous infections cause rapid necrosis of leaf tissue resulting in defoliation, starting in the upper canopy. Lesions on petioles or stems are reddish purple and several millimeters in length (Figure 11-6). Infected petioles remain attached to the plant that has been defoliated by Cercospora leaf blight (Figure 11-7).



Figure 11-6. Purple discoloration of a soybean leaf caused by Cercospora leaf blight. (Photo by C. Coker)



Figure 11-7. Cercospora leaf blight on stems and petioles with accompanying defoliation. (Photo by C. Coker)

Purple seed stain is characterized by irregular light to dark purple blotches on the seed that may cover much or even all of the seed coat. Infection can lower seed quality, germination and seedling vigor. Prolonged delay of harvest may contribute to a higher frequency of seed infection and discoloration.

The fungus *Cercospora kikuchii* is the causal agent of both diseases that overwinter in crop debris and infected seed. The pathogen produces a light-activated plant toxin called cersposporin, which is suspected of contributing to the reddish-purple discoloration of diseased tissue. Spores produced on infected debris are dispersed by wind or rain onto nearby soybean plants. Infection and disease development are favored by extended periods of high humidity and warm weather (82° to 86°F).

Disease management strategies include planting high-quality, disease-free seed, using tillage practices

that hasten decomposition of crop residue, growing the least susceptible cultivars that are adapted for the area, crop rotation with non-host crops such as corn, cotton, rice or sorghum, and timely harvest. Fungicides applied when weather conditions favor disease may suppress disease severity.

Downy Mildew

Downy mildew is distributed worldwide and is frequently observed in Arkansas, but rarely causes yield losses. Under extremely favorable environmental conditions, the disease may become severe enough to cause premature defoliation contributing to lower seed quality and reduced seed size.

Early symptoms of downy mildew may occur on young plants, but the disease does not become widespread in a field until late vegetative or early reproductive growth stages. Downy mildew appears on the upper leaf surface of young leaves as pale green or light yellow lesions (Figure 11-8). Lesion size and shape depend on leaf age. Older lesions may turn grayish brown to dark brown with yellow-green margins. On lower leaf surfaces, when conditions



Figure 11-8. Upper (light green spots) and lower (grayish-beige downy tufts) leaf surface of soybean leaves with downy mildew. (Photo by T. R. Faske)

favor disease development, lesions are covered with grayish tufts of fungal hyphae and spores. Severely infected leaves turn from yellow to brown and prematurely drop. In some regions, although rarely in Arkansas, infected seeds may produce systemically infected seedlings. Systemically infected plants remain stunted, and grayish tufts are commonly observed on the underside of leaves. With systemic infection, light green areas appear at the base of young leaves and spread along the vascular system infecting the entire plant.

Downy mildew is caused by Pernospora manshurica which overwinters in leaf debris and less often on seeds. The fungus survives as oospores (thick-walled resting spores) that germinate and infect seedlings the following spring. Once infection has occurred, sporangia (infectious spores) are produced on newly infected leaves from sporangiophores (spore stalks). These fungal structures make up the tufts of down-like growth that can be seen on the lower leaf surface, hence the name downy mildew. Sporangia are dispersed by wind to infect other plants. Disease development is favored by cool (68° to 72°F) temperatures and high humidity. Temperatures above 86°F halt fungal sporulation. Soybean leaves become more resistant as they age, and although lesions may increase in number, they decrease in size on older leaves.

Management practices for downy mildew include use of resistant cultivars, fungicide seed treatment, crop rotation with something other than soybeans for a year and crop residue destruction.

Frogeye Leaf Spot

Frogeye leaf spot is a common fungal disease in Arkansas. If not managed properly, severe yield losses can occur on a susceptible cultivar when conditions favor disease development.

Leaf spots are circular to angular in shape and range from 1/32 to 6/32 inch in diameter (Figure 11-9). Leaf symptoms begin as dark brown, watersoaked spots and mature into lesions with tan or brown centers and a narrow reddish-brown to purple margin. Older lesions are translucent and have whitish centers containing black dots (stromata). In severely infected plants, several lesions may coalesce into larger irregular-shaped spots. When



Figure 11-9. Frogeye leaf spot lesions on a soybean leaf. (Photo by T. R. Faske)

leaves are heavily infected (> 30% severity), they may wither quickly and prematurely shed, a condition called blighting.

Stem and pod symptoms are less common, but may appear late in the growing season with prolonged conditions that favor disease development. Stem lesions are elongated whereas pod lesions are circular. Mature lesions are slightly sunken with light gray centers and brown borders. The fungus can grow through the pod into the seed. Infected seeds may be gray to brown in color.

The causal agent is a fungal pathogen, *Cercospora sojina*, which overwinters on soybean residue and seed. Conidia (spores) produced on crop residue are dispersed by splashing rain or wind onto soybean plants. Warm (81° to 85°F), wet weather (e.g., heavy dew) conditions favor infection and disease development. Symptoms become visible 7 to 14 days after infection. Infection can occur at any stage of development, but young leaves are more susceptible than older leaves. Five known races of this pathogen have been reported in the U.S., and in the South, races at any location may change dramatically from year to year. Reaction of cultivars varies from highly resistant to susceptible.

Host resistance is the most effective and economical management practice for frogeye leaf spot. Single dominant genes Rcs1, Rcs2 and Rcs3 confer resistance to races, 1, 2 and 2 + 5, respectively. Rcs3 is the only gene with multi race resistance, and it confers resistance to all races that are known to occur in the U.S. Fungicides can be effective in managing the disease and are most effective when applied preventatively to protect new growth when conditions favor disease development. The most favorable environmental conditions often occur from full bloom to beginning seed (R2 to R5) in Arkansas. Strobilurin-resistant populations of C. sojina were confirmed in 2012 in several Arkansas counties, thus all strobilurin fungicides (FRAC 11) are not effective on these resistant populations of frogeye leaf spot. Triazole fungicides (FRAC 3) are effective on the strobilurin-resistant strains of C. sojina. Cultural management practices consist of planting high-quality, disease-free seed and implementing tillage practices that improve crop residue decomposition.

Aerial Blight

Aerial blight, also called aerial web blight or Rhizoctonia foliar blight, is a common disease on soybeans in the rice-growing regions of the U.S. and along the Gulf Coast. When the disease occurs in rice, it is referred to as sheath blight. This disease can cause significant yield loss in both soybeans and rice. Extensive yield losses (40% to 50%) have been reported in soybeans when conditions favor disease development.

Foliar symptoms often occur during late vegetative growth stages on the lower portion of the plant following canopy closure. Initially leaf symptoms appear as water-soaked, grayish-green lesions that turn tan to brown at maturity (Figure 11-10). The pathogen may infect leaves, pods and stems in the lower canopy. Reddish-brown lesions can form on infected petioles, stems, pods and petiole scars. Long strands of web-like hyphae can spread along affected tissue (Figure 11-11), and small (1/16 to 3/16 inch in diameter) dark brown sclerotia form on diseased tissue (Figure 11-12).



Figure 11-10. Water-soaked, greenish lesions caused by aerial blight on soybean leaves. (Photo by M. Emerson)



Figure 11-11. Web-like hyphae of *Rhizoctonia solani* spreading along the stem of soybean. (*Photo by M. Emerson*)



Figure 11-12. Mature sclerotia of *Rhizoctonia solani* on soybean petiole. (*Photo by M. Emerson*)

Aerial blight is caused by a fungus, *Rhizoctonia solani* AG1-IA, which overwinters as sclerotia in soil or plant debris from the preceding crop. During warm, wet weather, mycelium spread extensively on the surface of plants forming localized mats of "webbed" foliage. Spread from these localized areas can be rapid when conditions favor disease (high RH and 77° to 90°F). Because this pathogen also causes sheath blight of rice, soybean fields that follow rice with a history of sheath blight are likely to have high incidence of aerial blight.

There is little resistance to *R. solani* in soybeans, but some cultivars are less susceptible than others. Plant the least susceptible and best-adapted cultivar. Rotating with poor or non-host crops such as corn or grain sorghum for 2 years and avoiding narrow row widths and high plant populations are good management practices. When aerial blight is present in highly susceptible cultivars and environmental conditions are favorable for disease, fungicides should be applied.

Soybean Rust

Soybean rust (SBR) was first reported in 1902 in Japan and has been an important disease in Asia and South America for many years. SBR recently became an important disease in the continental U.S. It was first reported in 2004 in Louisiana and was confirmed in eight other states including Arkansas the same year. Since 2004, SBR has been observed on average three of every five years in Arkansas. Yield losses (30% to 80%) have been reported in other countries; however, yield losses in the U.S. have remained low due to early detection and timely application of fungicides.

SBR (also called Asian soybean rust or Australasian soybean rust) is caused by the fungus Phakopsora pachyrhizi, which requires a living host to survive. Typically, symptoms are observed first on the leaves in the lower canopy at or after flowering (R1 to R3). Lesions appear as small (2-5 mm), tan or reddish-brown, angular spots on leaves. Lesions are often observed first at the base of the leaflet near the petiole. Volcano-shaped pustules (uredinia, Figures 11-13 and 11-14) can be observed within the lesion on the underside of the leaf. When pustules are mature, they rupture and exude spores (urediniospores) that cause new infections. Pustules can be observed in the field with a 20x hand lens (Figures 11-13 and 11-14), but may be misdiagnosed as bacterial pustule by untrained observers. As the disease progresses and secondary infections occur,



Figure 11-13. Soybean rust pustule (yellow arrows) development at no magnification (right) and 10x and 50x magnification (left). (*Photo by T. R. Faske*)



Figure 11-14. Numerous soybean rust pustules on the lower leaf surface of a soybean leaf. (Photo by T. R. Faske)

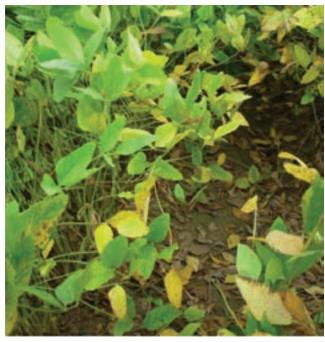


Figure 11-15. Defoliation of soybean leaves caused by soybean rust. (Photo by M. Emerson)

leaves begin to turn yellow and defoliate (Figure 11-15). Severely diseased plants may completely defoliate resulting in fewer and smaller seeds.

Soybean is the most important agronomic host of P. pachyrhizi, but the fungus can parasitize several other members of the Fabaceae (legume) family, including kudzu and common bean. Soybean rust does not overwinter in Arkansas, so each year new infectious spores must be disseminated from Gulf Coast states where it overwinters mainly on kudzu. Soybeans are susceptible to rust at any stage of development, but are most susceptible during the early reproductive stages. Conditions that favor disease are extended periods of leaf wetness over a wide range of temperatures (61° to 82°F). Temperatures above 86°F retard disease development. Infection can occur within 6 to 12 hours under optimum conditions, and new spores can be produced within 7 to 10 days after infection. A single uredinium can continue to produce spores for a 3-week period. Thus, when conditions favor disease, there is a high potential for spore production and secondary infection.

Management of SBR relies mainly on fungicides. Although fungicides are effective at managing SBR, both the type of fungicide applied and timing of application are critical in disease management. Strobilurin fungicides are effective as protectants and should be applied prior to disease presence whereas triazole fungicides, which are systemic, can be effective after disease has been observed in the field.

Triazoles, however, are also most effective when applied prior to disease development. Although data is limited in Arkansas, a fungicide applied after 10% disease incidence in the lower canopy under favorable environmental conditions in a South American field trial did not completely control rust. Since rust must be reintroduced each year into the state, early detection is crucial to management. An ongoing service provided to soybean growers in Arkansas is a network of sentinel plots and regular inspection of kudzu, and early-planted commercial fields to detect initial infections and provide early warning of disease presence in the state. Details of rust movement through the U.S. can be found on the IPM PIPE website or Arkansas Row Crops Blog. This early warning system allows timing of fungicides in high-risk fields for maximum effectiveness. In general, this will include a fungicide application during the early stages of reproduction (R1 to R3) and a second application made 14 to 21 days after first application. Applying a fungicide after the R6 growth stage may not provide a significant economical return; however, untreated fields may supply spores to later-planted soybeans in the area. A management tactic that appears to be effective in minimizing losses from SBR is simply planting early to avoid infection by late-season dissemination of spores from surrounding states or areas. Conversely, producers planting lateseason soybeans or double-crop soybeans should budget for a fungicide application.

Target Spot

Target spot is a foliar disease that has been reported in all soybean-growing regions of the U.S. Yield losses of 18% to 32% have been reported on susceptible cultivars in some areas of the country when conditions favored disease for a prolonged period of time, but this disease rarely causes significant yield losses in Arkansas.

Leaf lesions are reddish-brown, round to irregular-shaped spots that range in size from 3/8 to 5/8 inch in diameter. Lesions are frequently surrounded by a yellowish-green halo. Larger spots on leaves often develop diagnostic zonate patterns, hence the common name target spot (Figure 11-16). Infected areas on stems and petioles are dark brown and range from specks to elongated lesions. Lesions on pods are typically small (1/32 inch), circular purple or black spots with brown margins.

Target spot is caused by the fungus *Corynespora cassiicola* that overwinters on crop debris. Initial infections require high humidity (> 80%) or free moisture. Dry weather conditions suppress disease development.



Figure 11-16. Single target-patterned leaf spot surrounded by a yellowish-green halo caused by target spot. Other smaller lesions are caused by frogeye leaf spot. (Photo by T. R. Faske)

Typically, this disease is managed by using highyielding soybean cultivars, managing surface crop residue and avoiding soybean monoculture. Fungicides are rarely justified economically.

Charcoal Rot

Charcoal rot is a root and stem disease that commonly occurs in hot, dry weather conditions. This disease is most severe when plants are stressed from lack of moisture or nutrients, at excessive plant populations, or where soil compaction, other diseases or nematodes or improperly applied pesticides impair root development.

Charcoal rot symptoms typically appear as soybeans approach maturity. The earliest symptoms are smaller than normal sized leaves, which become chlorotic, then turn brown, but remain attached to the petiole giving the entire plant a dull greenish-yellow appearance. In many cases, these plants wilt and die. At early reproductive stages, a light gray to silver discoloration of the sub-epidermal tissue develops on taproot and lower part of the stem (Figure 11-17).

At advanced stages of disease development, near soybean maturity (R6-R7), the lower stem epidermal tissue is often shredded in appearance and exhibits an ashy-gray discoloration. Removal of epidermal tissue reveals numerous small, charcoal-black fruiting bodies (microsclerotia) embedded in the lower stem and taproot (Figure 11-18). Microsclerotia are often so numerous they resemble charcoal dust, hence the name of the disease.

The disease is caused by the soilborne fungus *Macrophomina phaseolina* which can infect more than 500 plant species. The pathogen overwinters as

sclerotia in soil or in crop debris, and these sclerotia can remain viable for at least 2 years. Infection can occur at seedling stage of development (2 to 3 weeks after planting), but symptoms remain latent unless the plants undergoes environmental stress during reproductive stages of growth. The optimal growth of the fungus is 82° to 93°F. Planting late-season or double-crop soybeans may encourage greater charcoal rot severity.



Figure 11-17. Discoloration of a lower soybean stem by charcoal rot. (Photo by T. R. Faske)

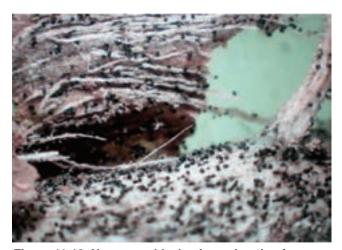


Figure 11-18. Numerous black microsclerotia of *Macrophomina phaseolina* on soybean. (Photo by A. Greer)

Currently, there are no commercially available resistant cultivars or fungal practices that effectively suppress charcoal rot. Fields with a history of severe charcoal rot should be rotated for 1 to 2 years with non-host crops (cereals). Avoiding excessive seed rates and maintaining adequate soil fertility to maintain healthy, vigorous plants reduces losses by this disease. The best way to avoid issues with charcoal rot is to limit drought stress during the reproductive stages of growth. Production systems like no-till that conserve soil moisture may also reduce losses by charcoal rot.

Pod and Stem Blight

Pod and stem blight is a common disease in all soybean-growing regions in the U.S. This disease can cause reduced seed quality and yield losses on susceptible cultivars when conditions favor disease development.

Infection may occur early in the season without any definite visible lesion development on leaf, stem, petiole or pod (Figure 11-19). Late in the season (R7), small, black, flask-shaped fungal fruiting bodies called pycnidia occur in linear rows on lower stems, petioles and pods, confirming early-season infection (Figure 11-20). Seeds within pods containing pycnidia are usually infected, thus reducing seed quality. Occasionally, bright red to brown lesions develop on cotyledons or hypocotyl (at or near soil line) of seedlings from infected seed.

Pod and stem blight is caused by a fungal pathogen, *Diaporthe phaseolorum* var. *sojina*, which overwinters in infected seed or on crop residue.



Figure 11-19. Pod and stem blight on soybean stem and pods. (Photo by A. Greer)



Figure 11-20. Pod and stem blight fruiting structures on soybean pod. (*Photo by A. Greer*)

Infection occurs when spores are splashed onto plants from crop residue or nearby diseased plants. Systemic, asymptomatic infection occurs throughout the growing season during prolonged warm (> 69°F), wet weather conditions. Such conditions favor seed infection and pycnidia development on maturing plants. Delayed harvest contributes to a higher incidence of infected seeds.

Management of pod and stem blight includes the use of high-quality, disease-free seed. Crop rotation to crops other than soybeans and tillage practices that hasten crop residue decomposition are helpful. Genetic resistance is available, and the least susceptible cultivars should be used in fields with a history of pod and stem blight. Timely fungicide applications during pod development (R3) and seed formation (R5) can be effective in suppressing disease development. Fungicide seed treatments can be effective at suppressing infection by pod and stem blight.

Stem Canker

Stem canker has been divided into two groups (northern and southern stem canker). Southern stem canker was first reported in 1973 in the South and by 1984 had been detected in all southern states. Stem canker can be one of the most destructive soybean diseases. Yield losses in susceptible cultivars can approach 90% under the right environmental conditions. The frequency and severity of stem canker outbreaks in Arkansas have been erratic and unpredictable from year to year, but stem canker is found somewhere in the state just about every year.

Leaf symptoms are characterized by yellowing and browning of the tissue between the main veins (Figure 11-21) that occur during the reproductive stages of development. Although leaf symptoms are somewhat diagnostic, they may resemble symptoms of sudden death syndrome or stem-boring insects, so diagnosis is based on both leaf symptoms and the presence of the characteristic stem cankers. Stem cankers are tan-brown lesions (cankers) with dark red-purple margins on the lower stem. Cankers first appear as small reddish-brown lesions on the main stem at a lower node. As the disease develops, the cankers enlarge and may extend for several inches along the main stem or up lateral branches (Figure 11-22). The lesions rapidly become definite, but the slightly sunken cankers rarely girdle the stem complexly. The cankers generally run along one side of the stem with adjacent stem tissue remaining green. Lengthwise sections cut through stems of symptomatic plants will show internal brown discoloration of the pith in the canker area.



Figure 11-21. Leaf symptom of southern stem canker showing yellowing and browning between the main leaf veins. (Photo by Kim Rowe)



Figure 11-22. Stem canker on main stem and lateral branches. (Photo by Kim Rowe)

The fungus *Diaporthe phaseolorum* var. *meridionalis* overwinters mainly in infested stem debris and may survive up to 14 months in soil. Susceptible plants can be infected at any stage of development, although infection generally occurs during the vegetative stages. Severe disease strongly correlates with prolonged rainy periods and temperatures from 70° to 85°F during early vegetative stages. Infection occurs when spores are splashed onto wet foliage during rainy weather. Stem canker can be extremely severe in susceptible cultivars, and yield loss can be extensive. High levels of resistance to this disease are available, and resistant cultivars are the primary means of control.

Stem canker is more severe with continuous soybean production and in no-till planting regimes. Soybean fields should be scouted each year during the reproductive stage to determine if stem canker is present. Small areas of stem canker in a field in one year may result in widespread disease the following year unless a resistant cultivar is selected.

Sudden Death Syndrome

Sudden death syndrome (SDS) was first reported in Arkansas in 1971 and since then has been found in most major soybean production regions of the U.S. This disease is often observed in well-managed, high-yield potential, irrigated fields growing under optimal conditions. Yield losses range from slight to 100% depending on the time of infection, cultivar susceptibility and disease severity.

Symptoms are most pronounced at mid-reproductive stages of development. Initial foliar leaf symptoms are scattered, chlorotic blotches between the main leaf veins that become necrotic, leaving mid-vein and major lateral veins green (Figure 11-23). Severely infected leaves detach from the petiole while the petioles remain green and attached to the stem long after leaf defoliation (Figure 11-24). Leaf symptoms of SDS may be confused with those seen with stem canker because they look so similar. However, with stem canker, leaflets remain attached to the petiole on plants after they die. In addition to leaf symptoms, flower and pod abortion, which is associated with the greatest yield losses, are symptoms of SDS.

Although there are no external symptoms of SDS on stems in contrast to visible stem lesions with stem canker, the vascular tissue of SDS-infected plants is



Figure 11-23. Chlorotic and necrotic blotches between central leaf veins on plants infected with sudden death syndrome. (Photo by T. R. Faske)



Figure 11-24. Green petioles without leaves remain attached on plants severely infected with sudden death syndrome. (Photo by T. R. Faske)

gray to brown on plants expressing foliar symptoms. The pith (central portion of the stem) in infected plants, however, remains white or slightly cream colored. Vascular discoloration often extends up the stem progressing farther on plants expressing higher disease severity.

Sudden death syndrome is caused by a soilborne fungus, Fusarium virguliforme, which overwinters as thick-walled spores (chlamydospores) in soil or on crop residue. Infection may occur early as seedlings development, but symptoms are not visible until plants have reached mid-reproductive stages of development. Symptoms are most severe at 68° to 77°F. Hot, dry weather appears to slow SDS although severe disease has been reported under these conditions. Disease development can be especially severe in fields that are also infested with soybean cyst nematodes, and disease is most problematic in cultivars that are susceptible to both the fungus and the nematode. Sudden death syndrome is usually most severe in saturated soils and is often most severe near the header pipe in furrow-irrigated fields or in lowlying areas in fields that are prone to standing water. Other factors that increase disease severity are high fertility and soil compaction.

Management options are limited for SDS, and foliar fungicides are not effective at suppressing this disease. Currently, there are no highly resistant cultivars available to producers, but some soybean cultivars are less susceptible to SDS. Delayed planting of fields with a history of SDS may be beneficial if saturating rains do not occur during early reproductive stages. Cultural practices that improve field drainage and crop rotation (2 years) with a non-host crop for soybean cyst nematodes may reduce severity of SDS.

Phytophthora Root Rot

Phytophthora root rot has been reported in all major soybean-producing areas of the U.S. and is common in Arkansas. This disease is most severe in poorly drained soils that remain wet for several days. Plant stand losses and 100% yield reductions can occur on highly susceptible soybean cultivars.

Symptoms may be found at any stage of soybean development, and severity is dependent on soybean susceptibility. Pre- and post-emergence damping-off occurs when soils remain saturated for several days after planting. On susceptible, intolerant cultivars, stems of older seedlings may appear water soaked and leaves may become chlorotic (Figure 11-25). Generally, these plants wilt and die rapidly. Where Phytophthora rot is present, soybean plants may die throughout the season (Figure 11-26). Symptoms include yellowing between leaf veins and margins and chlorosis of upper leaves followed by wilting. Leaves often remain attached to the dead plant. Because this is a soilborne pathogen, foliar symptoms are the result of a compromised root system. Root symptoms include roots that are discolored, with rotted lateral roots. Severe infection results in a girdling stem lesion (Figure 11-27) that may progress up the stem as high as 10 nodes before the plant wilts and dies on highly susceptible cultivars. On less susceptible cultivars, stem lesions may not girdle the stem, thus the plant does not wilt. Some cultivars may be susceptible, but highly tolerant, and although root systems are discolored and rotted, plants may remain alive. These plants can be stunted and slightly chlorotic, with stem lesions that develop along only one side of the stem.



Figure 11-25. Soybean seedlings infected with Phytophthora root rot. (Photo by J. Rupe)



Figure 11-26. Phytophthora root rot in soybean field. (*Photo by J. Rupe*)



Figure 11-27. Lesion caused by Phytophthora root rot on soybean stem. (Photo by J. Rupe)

The causal agent, *Phytophthora sojae*, overwinters in the soil or on crop residue as oospores (thick-walled resting spores). Oospores can remain viable for several years in the soil in the absence of soybeans. Oospores germinate at cool temperatures (< 60°F) and infect soybeans directly, or they can produce zoospores that are motile. Zoospores cause the primary infection in the spring. Flooding rains shortly after planting result in the most severe disease development. Increased disease severity has been observed with reduced tillage practices (especially no-till) and monocropping of soybeans.

Resistance is the most economical management tactic, but resistant genes in soybeans are only effective on specific races of the pathogen. Few soybean cultivars are resistant to all known races. An alternative to race-specific resistance is tolerant cultivars, which are effective against all races. Tolerant cultivars tend to sustain growth and yield even after infection, although the level of tolerance that is expressed in a

cultivar is highly dependent on both the amount of inoculum (pathogen) that is present and the favorability of the environment for disease development. Tolerant cultivars also contribute to a higher level of inoculum (oospores) for the following season, thus soybean monoculture should be avoided where Phytophthora rot is known to occur. Seed treated with the fungicide metalaxyl is effective in suppressing seedling infection. No foliar fungicide provides good suppression of Phytophthora root rot after the plants have emerged.

Southern Blight

Southern blight is considered a minor disease of soybeans in Arkansas. Typically, this disease occurs on isolated plants scattered through a field. Rarely does yield loss exceed 1% in fields affected by southern blight in Arkansas.

Symptoms can occur at any time during the season from seedlings to mature plants. The disease generally is most visible in plants during midreproductive stages. Seedling infection results in preor post-emergence damping-off. Later in the season, entire plants may become yellow and wilt, with leaves turning brown and often remaining attached to the plant (Figure 11-28). A dark brown lesion that girdles the stem occurs at the soil surface. This lesion is generally accompanied by the development of conspicuous white, fanlike mats of fungal mycelium that form on the base of the stem, on leaf debris and on the soil surface around infected plants (Figure 11-29). Numerous small, round fungal bodies that are about the size of mustard seeds (called sclerotia)



Figure 11-28. Yellow and wilted soybean plants infected with southern blight. (Photo by T. R. Faske)

Figure 11-29. White fungal mats of southern blight beginning to develop around soybean stems. (Photo by T. R. Faske)



Figure 11-30. Fungal hyphae of *Sclerotium rolfsii* on soybean plant with immature sclerotia developing above the soil line. (*Photo by M. Emerson*)

form on these fungal mats and on the lower stem (Figure 11-30). Initially, sclerotia are yellow-tan then progress to a reddish-brown color and finally dark brown at maturity.

The causal agent is the soilborne fungus *Sclerotium rolfsii* which has a host range of more than 200 plant species. This pathogen overwinters as sclerotia that can remain viable 3 to 4 years near the soil surface. Disease development is favored by hot (77° to 95°F), humid weather conditions, hence the name southern blight.

All soybean cultivars are susceptible to southern blight. Crop rotation with corn, grain sorghum or wheat for 2 years can be beneficial at reducing survival and buildup of sclerotia in the soil. Deep cultivation to bury sclerotia in the soil reduces sclerotia longevity and may be an option in certain farming systems.

Viral Diseases

Soybean Mosaic Virus

Soybean mosaic virus (SMV) occurs in all soybean production areas of the world. Yield loss ranges from 8% to 35% with a high of 94% in some production systems. Though symptoms can vary among soybean varieties, a green-yellow mosaic pattern is the most common (Figure 11-31). At advanced stages a yellow-brown mosaic pattern is often observed, often followed by premature defoliation (Figure 11-32). Infected seeds are mottled brown or black; however, other diseases may cause seed discoloration, thus a laboratory assay is necessary to verify SMV infection. Yield and quality losses are related to smaller seed size with lower germination rate than healthy seeds. The yield losses caused by SMV infection in Arkansas have not been thoroughly documented.



Figure 11-31. Mosaic pattern on infected soybean. (*Photo by J. Zhou*)



Figure 11-32. Leaf yellowing at late infection stage. (*Photo by J. Zhou*)

SMV may be introduced into a virus-free region by planting infected seed. The pathogen is spread from plant to plant by aphids. The soybean aphid, Aphis gylcines, the most common SMV vector, is the only aphid species that can establish colonies on soybeans. Once an aphid feeds on an infected soybean plant, it only takes a short time (seconds to a few minutes) for the insect to acquire the virus. As the virus-carrying aphids move and feed on healthy plants, the virus will be spread around. In the absence of soybeans the virus can overwinter on a wide range of hosts from five plant families (Bean, Amaranth, Passionflower, Figwort and Nightshade). The ability of the soybean aphid to overwinter in Arkansas and alternative host species of importance for SMV in Arkansas are not known. Chemical control of the soybean aphid is not recommended because some insecticides may increase the movement of the vector in the field, which facilitates further dissemination of the virus.

It is important to use virus-tested seeds to minimize incidence of this disease. Resistant cultivars have been widely used, and planting SMV-resistant soybean cultivars is the most economical practice to manage the disease. Several resistance genes have been identified and are effective against some, but not all, virus strains. Based on the differential reactions on a set of soybean cultivars, SMV has been classified into numerous strains. In the U.S, nine strains, G1-G7, G7a and C14, are currently recognized. Additional strains have been identified in other countries (Canada, China, Japan and South Korea), including isolates that overcome all known resistance to the virus. In Arkansas, only highyielding cultivars with resistance to most (or all) SMV strains, such as Ozark, USG 5002T and USG 5601T, are widely used in controlling SMV. An additional management tactic is avoiding late planting to minimize aphid transmission at an earlycrop growth stage.

Bean Pod Mottle Virus

Bean pod mottle virus (BPMV) is another important virus of soybeans. It was first reported in Arkansas in 1951 and is now prevalent in all production areas in the U.S. Yield reduction ranges from 10% to 60% depending on variety and geographic area, with highest yield reduction occurring when the virus infects plants early in the season. The yield loss in Arkansas is not known at this time.

Symptoms on infected soybeans may vary depending on the variety. Foliage symptoms range from mild chlorotic mottling in the upper canopy

(Figure 11-33) to puckering and severe mosaic (Figure 11-34) in lower leaves. Acute symptoms develop on young leaves. "Green stem" caused by delayed maturity due to BPMV infection is often observed in the field close to harvest season (Figure 11-35). Mottling of the seed coat is another prominent symptom, but this symptom is not a reliable predictor of BPMV infection because soybeans infected with soybean mosaic virus (SMV) also exhibit similar symptoms.

Seed contamination is not important in BPMV epidemiology. The virus is primarily transmitted by the bean leaf beetle (*Cerotoma trifurcata*). The virus has been found in overwintered bean leaf beetle adults which may survive in grass, leaf litter or even rocks and colonize soybeans as seedlings emerge in the spring. Because most flight events of beetles are limited to about 30 meters, it is likely that BPMV spread is restricted within and between fields. Other



Figure 11-33. Mottling on BPMV-infected soybean leaves. (*Photo by J. Zhou*)



Figure 11-34. Puckering and rugosity of soybean leaves. (Photo by J. Zhou)



Figure 11-35. "Green stem" caused by BPMV infection on soybean plant. (*Photo by R. Valverde*)

than overwintering beetles and infected seeds, alternate hosts in the field can also serve as an inoculum source for disease development. Leguminous hosts including cowpea and some bean species sustain virus replication, as well as *Demodium* species which are natural hosts for BPMV.

Soybean cultivars with BPMV resistance are not available. Consequently, elimination of alternative hosts and vector control are important for disease management. Insecticides targeted at emerging overwintered beetles (F_0) and the first seasonal generation (F_1) population of *C. trifurcata* can reduce vector populations throughout the growing season, provide limited reduction in virus incidence and improve both yield and seed coat color. In addition, it is also advisable to delay soybean planting so that the early-season mortality of beetles increases and thereby reduces vector populations.

Synergistic Interaction of SMV and BPMV -

A synergistic interaction between SMV and BPMV may lead to severe symptoms and yield losses. Co-infection with both viruses may result in yield reduction ranging from 66% to 86% compared with 8% to 35% when plants are infected with SMV alone

or 10% to 60% with BPMV alone. Symptoms in plants infected with both viruses include severe dwarfing, foliar distortion, leaf necrosis and mottling. Seed coat mottling may also occur, but this symptom is not diagnostic. The effect of co-infection on yield and the degree of seed transmission depends on virus strain, cultivar and time of infection by either virus.

Tobacco Ringspot Virus

Tobacco ringspot virus (TRSV) on soybeans can have a severe impact on seed yield and quality. Yield reduction ranges from 25% to 100% due to reduced pod set and seed formation with lower protein and oil content in infected seeds.

The most distinct symptom of TRSV infection on soybeans is bud necrosis (Figure 11-36) and excessive growth of leaves and buds (Figure 11-37). Virus



Figure 11-36. Bud necrosis caused by TRSV infection on soybean. (Photo by J. Zhou)



Figure 11-37. Excessive growth of buds on soybean. (Photo by I. E. Tzanetakis)

infection causes leaves to be thicker and darker in color. Stems of infected soybeans remain green for 1 to 2 weeks longer than healthy ones, and the pith of stems and branches of infected plants may exhibit brown discoloration. Infected plants are generally stunted and have a low seed formation rate. Pods are usually undeveloped or aborted because insufficient pollen is produced for fertilization. This in turn may cause production of a proliferation of new buds and pods leading to "green bean syndrome."

Seed transmission is the most important mode for long-distance dissemination of the virus, and the infection rate is much higher when soybeans are infected before flowering. The virus can invade the embryo where it remains viable for at least 5 years. TRSV is also mechanically transmissible and can be transmitted by the dagger nematode (*Xiphinema americanum*).

TRSV triggers symptomatic and asymptomatic infection on a wide host range including vegetables, ornamentals and common weed species. The elimination of indigenous weeds in soybean fields, such as Palmer amaranth (*Amaranthus palmeri*) and lambsquarter (*Chenopodium album*), is important for disease control. Given that no resistance for TRSV has been reported in soybeans, it is critical to use virus-free seeds when planting. Minimize dagger nematodes, which are efficient vectors of the virus, through tillage practices.

Soybean Vein Necrosis Virus

Since the first report in 2008, soybean vein necrosis virus (SVNV) has been reported in all soybean-producing areas in the U.S. Yield loss estimates for this virus are still under investigation.

Typically, symptoms of SVNV start as vein clearing along the main veins (Figure 11-38), with veins yellowing and finally becoming necrotic as the season progresses (Figure 11-39). Clearing or lesions may occur on one or multiple areas of the affected leaves, and severely affected leaves die off. Early infection is usually detected by mid-June in the southcentral and eastern states, but timing of the first symptoms may vary depending on cultivar and local weather pattern. The distinction between infected and non-infected plants may be difficult due to the absence of well-defined lesion edges in early infections. Unlike other diseases that cause foliar lesions such as frogeye leaf spot or bacterial blight, lesions caused by SVNV expand from main veins to the



Figure 11-38. Early symptom of SVNV on soybean leaf. (Photo by J. Zhou)



Figure 11-39. Typical lesion of SVNV on soybean leaf. *(Photo by J. Zhou)*

surrounding areas of the blade. Symptom intensities vary among cultivars. Mild infections cause thread-shaped vein clearing whereas severe infections result in purple or dark brown lesions expanding to the majority of the leaf blade. Disease symptoms are more evident higher in the canopy because newly emerged leaves are preferential feed sites of the virus vector, the soybean thrips.

SVNV is transmitted by the soybean thrips (Figure 11-40), probably the most abundant thrips species in soybean fields. An indigenous weed species commonly found in soybean fields, Ivyleaf morning glory (Ipomoea hederacea Jacq), may function as a virus reservoir. Two other legume species, cowpea and mungbean, can also be infected by SVNV. Because the soybean thrips is a common pest in different legume species, it is highly possible this virus is a new threat not only to soybeans but also other legumes. Virus infection can occur at any time during the growing season, but symptoms usually become most visible after flowering. Cool temperature favors symptom development, and a mild winter followed by a warm spring may promote vector proliferation.

Control of soybean thrips is critical in lowering SVNV incidence, and control of Ivyleaf morning glory in fields. At this point, other cultural practices that are effective in lowering SVNV incidence are unknown, and the identification of resistance and development of resistant cultivars are still underway.



Figure 11-40. Soybean thrips. (Photo by M. E. Rice)