SDSU Extension

iGrow Soybean

BEST MANAGEMENT PRACTICES

Chapter 59:

Fungal and Fungal-like Diseases in Soybeans

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A number of foliar, stem, or root fungal diseases are found in South Dakota soybean fields. Under certain conditions, fugal and fugal-like diseases can produce substantial losses. The purpose of this chapter is to discuss soybean fungal disease characteristics, life cycles, and management. Fungal diseases discussed in this chapter include Phytophthora root and stem rot, white mold, stem canker, brown spot, charcoal rot, frogeye, leaf blight and purple seed stain, downy mildew, powdery mildew, brown stem rot, sudden death syndrome, anthracnose, soybean rust, and Cercospora blight. A management timeline which provides details on disease scouting is provided in Chapter 28, seed treatment information is provided in Chapter 8, and information on specific fungicide treatments and rates are available at https://extension.sdstate.edu/south-dakota-pest-management-guides.

Table 59.1. Key factors to control fungal problems.

1. Correct disease identification is a must in order to develop effective control strategies.
   a. Different problems require different treatments. (Table 59.2)
   b. Seed or foliar application fungicides can be used and fungicides are most effective when applied at an appropriate time. The selection of seed or foliar application should depend on the best option to control the targeted pathogen.
2. The risk of developing fungicide resistant pathogens can be reduced by:
   a. applying fungicides at recommended rates,
   b. applying appropriate fungicides only when the diseases are present, and
   c. by rotating pesticide chemistries.
3. A listing of registered fungicide products for soybean disease problems is available at https://extension.sdstate.edu/south-dakota-pest-management-guides. When using fungicides, read and follow label directions for approved uses.

Table 59.2. Foliar diseases and possible management approaches.

Disease	Rotations	Tillage	Variety Selection	Fungicide	Notes
Phytophthora	X		X	X	Can destroy entire fields.
White mold	X	X	X	X	Can reduce yields 4 bu/a for each 10% increase in incidence.
Stem canker	X		X	X	Northern stem canker can reduce soybean yields 50 to 80%.
Brown spot	X	X		X	Overwinters on infected leaf and stem debris.
Charcoal rot	X		X		Drought may increase.
Frogeye	X	X	X	X	Use clean seed.
Downy mildew	X	X	X		Use clean seed.
Powdery mildew			X	X	Temps above 86ºF halt disease.
Brown stem rot	X	X	X		Use shorter maturities.
Sudden death syndrome		X	X		Install drainage and delay planting.
Anthracnose	X	X			Use sed treatment (Chapter 8).
Soybean rust			X	X	Not typically found in South Dakota
Cercospora blight	X	X	X	X	Favored by high temperatures and can reduce soybean quality due to purple staining of the seed.

Table 59.3. Scouting guide for soybean diseases caused by fungi and fungal-like pathogens. Modified from Chapter 28.

Growth Stage	April 30	May 7	May 14	May 13	May 28	June 4	June 11	June 18	June 25	July 2	July 9	July 16	July 16	July 30	August 6	August 13	August 20	August 27	Sept 3	Sept 10
		P	VE	VC	V1	V3	V4	V6	R2		R3	R4	R5			R6		R7	R8	
Scout Phytophthora Root/Stem Rot	■	■	■	■	■															
Scout Damping off/Pythium Seed Rot	■	■	■	■	■	■														
Watch for root rots and Rhizoctonia	■	■	■	■	■	■	■													
Scout Soybean Mosaic Virus			■	■	■	■	■	■	■	■	■	■								
Scout Bean Pod Mottle Virus			■	■	■	■	■	■	■	■	■	■								
Scout Brown Spot			■	■	■	■	■	■	■	■	■	■	■	■	■	■				
Scout Downy Mildew					■	■	■	■	■	■	■	■	■	■	■	■	■	■		
Scout Cercospora Leaf Spot					■	■	■	■	■	■	■	■	■	■	■	■	■	■		
Scout Soybean Aphids					■	■	■	■	■	■	■	■	■	■	■	■	■	■		
Check roots for cysts							■	■	■	■	■	■	■	■						
Scout for Bacterial Leaf Blight							■	■	■	■	■	■	■	■	■	■				
Scout Phytophthora Root/Stem Rot (2nd event)							■	■	■	■	■	■	■	■						
Scout for Soybean Cyst Nematodes							■	■	■	■	■	■	■	■	■	■	■	■	■	■
Scout Anthracnose									■	■	■	■	■	■	■	■	■	■		
Scout Frogeye Leaf spot									■	■	■	■	■	■	■	■	■	■		
Scout Brown Stem Rot											■	■	■	■	■	■	■	■	■	■
Scout Sudden death Syndrome											■	■	■	■	■	■	■	■	■	■
Scout Schlerotinia Stem Rot (white mold)											■	■	■	■	■	■	■	■	■	■
Scout Pod and Stem Blight											■	■	■	■	■	■	■	■	■	■
Scout Northern Stem Canker											■	■	■	■	■	■	■	■	■	■
Scout Charcoal Rot														■	■	■	■	■	■	■

Phytophthora Root and Stem Rot

Extent of the problem

Phytophthora root and stem rot (PRR) in soybeans is one of the most destructive soybean diseases in South Dakota and the North Central region of the U.S. State wide, annual losses range from 4% to 6%. PRR has also been reported from Argentina, Australia, Brazil, Canada, the People's Republic of China, Hungary, Italy, Japan, and the countries composing the former Soviet Union.

Damage from PRR is directly related to annual weather patterns, especially by rainfall amounts and cycles. Fields or sites within fields that receive heavy rainfall or irrigation are susceptible to plant mortality from PRR with 100% yield loss in heavily affected field areas. PRR can destroy entire fields when the conditions are favorable (Draper and Chase, 2001; Dorrance et al., 2012; Schmitthenner, 2000)

Symptoms

PRR can appear in any stage of soybean development. Fields that should routinely be scouted include those that: 1) contain wet spots; 2) are poorly drained; 3) contain high clay contents; 4) have been in continuous no-till or soybean; 5) have a history of PRR; and 6) large weedy areas due to poor stand development. PRR symptoms change as the plant matures (Dorrance et al., 2012; Schmitthenner, 2000).

Early-season Phytophthora root and stem rot symptom may be found in areas with poor seed germination. Seed rot and decay can occur if infection occurs between seed swell and germination. Affected seeds may be very soft and brown, and often, individual seeds completely disintegrate. In severe cases, replanting may be necessary. Optimum temperatures for disease development range from 77°F to 86°F (25°C to 30°C) (Draper and Chase, 2001; Dorrance et al., 2007; Schmitthenner, 2000).

This disease also causes damping-off, which is most likely to occur several days prior to or after seedling emergence. Damping-off is marked by wilting and death of pre-emergent or emerging soybean seedlings. Affected areas of the stem may look water-soaked or bruised and often disintegrate. Symptoms include yellowing, wilting, and light brown-soft necrosis (decay and rot) of the stem. Affected stem areas may have a dark discoloring, and colonized seedlings may die. Plants typically are easily pulled from the ground due to root damage. The severity of damping-off may be variable depending on the amount of PRR resistance in the variety. Early-season PRR is promoted when flooding occurs within the first week following planting. Damping-off symptoms can develop 5-14 days after the soil has been saturated with water (Draper and Chase, 2001; Dorrance et al., 2007, 2012; Iowa State University Extension, 2010).

Stem and root rot midseason PRR symptoms can be observed around the first of July (Figs. 59.1 and 59.2). At this stage, PRR infection begins in the root system and progresses into the lower stem. Symptoms include dark-brown discoloration on the lower stem that advances from below soil level to several nodes on the stem. In severely affected plants, stem girdling, destruction of the lateral roots, and rot of the taproot can be lethal to severely affected plants. Leaf wilting followed by drooping of the petioles follows, and this wilting advances upward from the bottom of the plant. Leaves may develop a grayish cast and yellow spots prior to permanent wilting. Symptoms on older soybean plants are related to the susceptibility of the soybean cultivar. Cultivars with low tolerance may continue to die throughout the season; although, they were infected during early growth stages; (Draper and Chase, 2001; Dorrance et al., 2007; Iowa State University Extension, 2010; Schmitthenner, 2000).

Late in the growing season, areas within a row where plants have been killed by PRR may be hidden by healthy plants that have grown tall enough to disguise this problem (Fig. 59.3). Large areas in poorly drained fields where plants have died due to PRR may be covered by colonizing weeds. Thus, late-season PRR is often underestimated due to being concealed within the canopy or by weed growth (Draper and Chase, 2001).

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Figure 59.1. Areas of PRR-affected soybean plants within the rows of planted soybeans. (Photo courtesy of Martin Draper, USDA-NIFA)


Figure 59. 2. Dying soybean plants affected by PRR are seen between plants showing chlorotic leaves that may be a precursor to wilting. (Photo courtesy of Martin Draper, USDA-NIFA)


Figure 59.3. Poorly filled rows seen in the field plot above are due to early-season damping-off and plant death later in the season. Areas resembling this in production fields would likely be overgrown by weeds during the later season. (Photo courtesy of C.L. Strunk, SDSU Extension; and M.A.C. Langham, SDSU Plant Pathology)

Causal pathogen and life cycle

Phytophthora root and stem rot (PRR) is caused by Phytophthora sojae. Phytophthora is a member of the Kingdom: Stramenopila and the Phylum Oomycota. These organisms were traditionally classified as fungi, but recently, basic differences in their composition and genome have caused these organisms to be reclassified. They are now called fungal-like organisms. P. sojae has a large amount of genetic variability that forms a number of races (also called pathotypes). These races are identified by the combination of virulence and avirulence genes. Races 1, 3, 4, and 25 are the most common races in South Dakota (Draper and Chase, 2001; Iowa State University Extension, 2010).

P. sojae produces oospores (Fig. 59.4) and zoospores. Oospores are thick-walled spores that are produced through sexual reproduction. They can remain dormant in soybean residue or soil without a host for many years. The infection cycle begins when oospores germinate to form sporangia (the structure where zoospores are produced). Temperature is one of the principal triggers for this germination (Draper and Chase, 2001; Schmitthenner, 2000).

Zoospores are rapidly produced through asexual reproduction. P. sojae-infected soybean tissue produces sporangia and zoospores as long as moisture and temperatures remain favorable. Zoospores are unique in that they have flagella, which allow them to discover new sites to infect by swimming from one site to the next) (Draper and Chase, 2001).

A video of the zoospores swimming, produced by Dr. Thomas Chase, is available at http://www.youtube.com/watch?v=gDT5Pg3_nsM%20%20. In the soil, zoospores find soybean roots by following gradients of root exudates (daedzein, genestein, and other isoflavonoids) to the plant roots. Soil temperatures above 60°F promote germination and infection of P. sojae. Although capable of initial plant infection through the leaves, P. sojae seldom infects leaves because this mechanism requires that soil be splashed onto leaves (Draper and Chase, 2001; Dorrance et al., 2007; Schmitthenner, 2000).

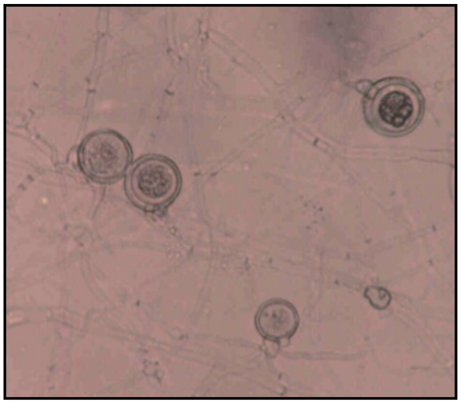

Figure 59.4. Oospores are the sexual spores formed by *Phytophthora sojae* and other related fungi. Their sturdy thickened cell wall structure helps these spores to survive adverse conditions in the soil in order to overwinter. (Photo courtesy of Martin Draper, USDA-NIFA)

Management

PRR requires a combination of crop management and disease monitoring to manage, particularly in low-lying fields or fields with compacted soils. In order to minimize PRR, a combination of management approaches is needed. (Draper and Chase, 2001).

1. **Selecting tolerant or resistant varieties** is the first practice to utilize in PRR management. Tolerance and race-specific resistance options are available. **Tolerance** is a partial solution and does not eliminate the risk of PRR. Tolerance delays disease onset, severity, and losses. This approach is typically effective across many races, and it generally more effective at late rather than early growth stages. **Race-specific resistance** targets a specific P. *sojae* race. Two mechanisms of race-specific resistance exist (incompatible interaction and root resistance). When incorporated into a soybean cultivar, this resistance provides complete resistance against a specific P. *sojae* race. However, prior to deploying race-specific resistance, the P. *sojae* race in a producer's field should be identified. Additionally, fields may have multiple P. *sojae* races present. Producers should also be alert for signs of PRR in a resistant variety that has previously performed well. This could signal the development of a P. *sojae* race adapted to the resistance gene used in that variety (Draper and Chase, 2001; Dorrance et al., 2012; Iowa State University Extension, 2010; Schmitthenner, 2000). Consult SDSU Extension for available information of the current status of race-specific resistance.

2. **Fungicide seed treatments** can reduce PRR. Details on fungicide seed treatments are available in Chapter 8. Generally: 1) metalaxyl and related fungicides, such as mefanoxam, are effective against Phytophthora; 2) seed treatments are more effective at lower rates and more cost effective than band applications over the row; and 3) band applications of metalxyl have been highly effective when soybeans are planted in wide rows on compacted soil (Draper and Chase, 2001). Fungicide recommendations can be obtained at https://extension.sdstate.edu/south-dakota-pest-management-guides or by checking with SDSU Extension personnel.

3. **Use crop rotation.** Crop rotation can assist in PRR management. Continuous soybeans increase Phytophthora inoculum in the soil and promote the development of new races. Extended rotations into corn, small grains or other non-host crops may reduce PRR levels in severely affected soybean fields (Draper and Chase, 2001; Dorrance et al., 2012; Iowa State University Extension, 2010.

4. **Improve soil drainage, aeration, and structure.** Water is a vital part of the Phytophthora's life cycle. Thus, decreasing the amount of standing water in fields with a history of PRR is an important part of

control. Additionally, P. sojae infection is often higher in compacted, fine-textured, and poorly drained soils (Draper and Chase, 2001).

5. **Manage tillage practices.** Reduced tillage practices including no-till can maintain high concentrations of oospores in the soil zone where the new soybean plants are establishing roots. Additionally, no-till soils retain moisture and dry out more slowly, and moisture promotes the disease risk. However, deep tillage with heavy equipment or working waterlogged soils can increase compaction and water retention. Ridge tillage and other similar tillage practices are preferred for drying wet soils (Draper and Chase, 2001; Iowa State University Extension, 2010).

6. Manage residual nitrogen. High residual nitrogen level, particularly where swine manure has been injected into fields (especially in the spring), may increase the severity of PRR when soil moisture is adequate for infection (Draper and Chase, 2001).

White Mold (Sclerotinia Stem Rot)

White mold, caused by Sclerotinia sclerotiorum, is a chronic problem disease that can severely damage soybeans, sunflowers, dry beans and other legumes, and many other dicotyledonous crops. In soybeans, white mold has been estimated to cause a 4 bu/ha yield reduction for every 10% increase in disease incidence. White mold damage may be moderate when disease incidence is less than 20%. Moderate damage is attributed to the soybean plants' ability to compensate for the damaged plants. In South Dakota, soybeans grown in the eastern region are more at risk that soybeans grown in western regions (NCSRP, 2012).

Symptoms

S. sclerotiorum may first be identified when apothecia have formed (Fig. 59.5). Apothecia are vase or cuplike structures that emerge from the overwintered sclerotia (Figs. 59.5 and 59.6). Since these structures are formed from fungal tissues, they are referred to as signs rather than symptoms. If a field is being scouted for the apothecial stage of white mold, this should be timed to coincide with canopy closure and it should target poorly drained areas with high relatively humidity (NCSRP, 2012).

Infected plants may appear wilted during the reproductive growth stages. S. sclerotiorum progresses from the young pods to the nodes and to the stem. It may girdle the stem, which disrupts the transport of water and nutrients to the leaves. As the fungus invades the stem, plant tissues die (necrosis) and the necrosis progresses upward from the point of infection. Inspection of the lower stem may reveal a bleached area starting from a leaf axil and extending in each direction. Leaves and stems of plants affected by white mold may turn brown.

In fields with thick canopy growth, infected plants may be hidden, and the necrotic leaves and stems may only be found when the thick canopy is pushed open. When necrotic plants are closely examined, lesions and thick mats of white fungal mycelium may be found (Fig. 59.7). As the infection progresses (Fig. 59.8), dark pellet-like structures (sclerotia) will be formed on the mats. Sclerotia are important for overwintering, and they can remain viable in the soil or debris for many years. Sclerotia may also form in the pith of infected soybean stems and may be found only when they are split open (Fig. 59.9) (Iowa State University Extension, 2010; NCSRP, 2012; Westphal et al., 2006).

Figure 59.5. Apothecia is forming in a petri dish from a sclerotium produced by *S. sclerotiorum* to demonstrate how the apothecia is produced from the sclerotia and the number of apothecia than can be produced by one sclerotium.
(Photo courtesy of Martin Draper, USDA-NIFA)

Figure 59.6. Apothecia are forming at the soil surface in the center of this photograph. These apothecia will produce thousands of ascospores to start the infection cycle in the spring. (Photo courtesy of H.F. Schwartz, Colorado State University, Bugwood.org)

Figure 59.7. Abundant white mycelial growth is a distinctive characteristic of white mold. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.8. Sclerotia are the hard dark structures forming on the infected plant stem. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.9. Sclerotia can be seen forming in the pith of infected soybean stems. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Causal pathogen and life cycle

Sclerotinia sclerotiorum is the fungus that causes white mold. *S. sclerotiorum* belongs to the Kingdom Fungi and the Phylum Ascomycota. *S. sclerotiorum* overwinters in the soil by producing hardened, dark, pellet-like structures called sclerotia. These structures are composed of tightly compacted fungal mycelium covered with a dark black covering (rind). Sclerotia can be found in crop debris or can fall to the soil during harvesting. Sclerotia can survive in the soil for many years (NCSRP, 2012).

In the spring, *S. sclerotiorum* produces sexual spores by forming an abundance of ascospores in vase or cup-like structures called apothecia (Figs. 59.25 and 59.26). This plays an important role in white mold as the apothecia provide ascospores for the primary inoculum to restart each year's disease cycle. The ejection of these ascospores, which adhere to the dying flower petals attached to young pods, begins the infection phase of white mold. Ascospores may also infect nearby plants or be wind-blown to nearby fields. Apothecia develop when the soil has been continuously moist for 10-14 days. Dense canopies, high moisture levels, and irrigation before flowering favor apothecia development (NCSRP, 2012; Staton, 2012; Westphal et al., 2006).

Management

Soybean white mold incidence is favored by dense soybean canopies, rain events during flowering, high humidity and fogs, and extended dew periods. Other risk factors that should be considered are a history of white mold in the field, rotation history, pockets of poor drainage, air movement barriers, early canopy closure, narrow rows, excessive plant nutrition, and weed management. Managing white mold combines variety selection with minimizing the impact of high moisture levels and heavy rainfall in the soybean canopy and reducing the sclerotia number and spread (NCSRP, 2012).

1. **Select a tolerant soybean variety is a good first step.** No soybean varieties, with complete resistance to white mold, are available, but tolerant varieties are often effective in managing white mold and increasing yields. Additionally, selecting soybean varieties that are short and do not lodge easily will also improve white mold management. Susceptible varieties should be avoided in poorly drained fields with a white mold history (NCSRP, 2012).

2. **Manage the canopy coverage and row spacing.** Tight canopy coverage in soybeans slows down moisture evaporation, drying of wet soils, and increases the relative humidity within the canopy. Thus, production practices that allow more air movement through the canopy reduce the potential for white mold development. Wider row spacing is a very effective method for opening the canopy to air movement and decreasing white mold risk (NCSRP, 2012; Westphal et al., 2006).

3. **Use an effective crop rotation.** Continuous soybean production systems increase the potential for white mold. Rotation with crops that are not susceptible to white mold (such as corn, wheat, or other cereals) is needed to decrease the white mold potential. Typically, 2-3 years of rotation is recommended before returning to soybeans, particularly in severely infected fields. Additionally, weed control of dicotolydenous plants (such as lambsquarters and pigweed) that are alternate hosts for S. sclerotiorum is critical during nonsoybean years (Iowa State University Extension, 2010; NCSRP, 2012; Westphal et al., 2006).

4. **Avoid excessive moisture.** Moisture promotes white mold infection. Thus, excessive irrigation should be avoided until after flowering (the prime infection period) (NCSRP, 2012).

5. Consider tillage. When buried deep in the soil, sclerotia can survive up to seven years. However, only sclerotia that are buried within two inches of the soil surface can germinate and produce ascospores. Thus, burying the sclerotia by tillage can be effective. Subsequent tillage, however, can bring buried sclerotia to the surface. In no-till situations, the sclerotia tend to remain close to the surface, but following soybeans in rotation with a non-host crop allows a large number to germinate during the non-host years (NCSRP, 2012; Westphal et al., 2006).

6. **Minimize the spread of disease by field equipment and seed.** White mold can be spread between fields by seed (Figs. 59.10 and 59.11), equipment, manure, and soil movement. Sclerotia present in soybean stems, debris, or seed can be trapped on harvest combines and other equipment. Because combines can move sclerotia from infected to non-infected fields it is recommended to harvest infected fields after non-infected fields. After harvesting infected areas, it is recommended to clean the combine, other equipment used in the field during harvest, and any seed from that field. Seed cleaning is critical because sclerotia and soybean seeds have similar size and shape, and sclerotia mixed in with soybean seeds can initiate infections in new fields (NCSRP, 2012; Westphal et al., 2006).

7. **Fungicide can be used to help control the disease.** In cases of severe white mold incidence or fields with a history of white mold, fungicide usage may be desired. Complete control of white mold is not possible, but fungicides may reduce the white mold incidence. A challenge with effective control is getting the fungicide to penetrate into the dense soybean canopy. Penetration can be improved by increasing the spray volume, reducing the groundspeed, and using 40 PSI nozzle pressure and a flat fan nozzle that produces fine to medium drops (Staton, 2012). Contact SDSU Extension plant pathologist and pathology field specialists for currently labeled fungicide recommendations, spraying hints, and fungicide rates.

8. **Biocontrol may be an option.** Some antagonistic fungi are available that may colonize and reduce sclerotial number in soil (Iowa State University Extension, 2010; Westphal et al., 2006).

Figure 59.10. Sclerotia forming in the seedpod can lead to the sclerotia being included in harvested seed or caught in farm machinery. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.11. Sclerotia mixed in with harvested seed can be a source of inoculum for white mold if these seed are used for planting. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Stem Canker

Northern stem canker was a major disease in the Midwest through the 1950s and 1960s, but the use of less susceptible cultivars has curbed this disease. However, since 2000, a resurgence of this disease has been found in South Dakota, Wisconsin, Minnesota, and Iowa. Northern stem canker can reduce soybean yields 50% to 80% (Chase, 2012; Hadi, 2012).

Symptoms

Early-season symptoms occur only rarely and are typically small reddish lesions on cotyledons. This early infection may spread into the stem and cause seedlings to die. Seed germination rate can be reduced and seedling decay and death can occur if infected seeds are planted.

In most cases stem canker symptoms can be observed in the mid to late August). The most prominent symptoms are reddish-brown lesions on one side of a leaf petiole branch base or stem nodes. As the disease develops, the lesion becomes sunken, elongates, and turns dark gray-brown (sometimes with reddish margins) (Fig. 59.12). A distinctive characteristic of the lesions is the presence of green stem tissue above and below the canker. Brown discolorations, which can be seen when the stem is split, can develop inside the stem (Chase, 2012; Hadi, 2012; Iowa State University Extension, 2010).

Toxins produced by the pathogen are translocated to the leaves where it causes an interveinal yellowing that progresses to death of the leaves. Leaves remain attached to the plant after death. As the lesion progresses, the stem can be girdled which cuts off water transport to the leaves. This in turn causes the soybean plants to wilt. Stem girdling has a large impact on yield because it affects pod fill and yield. Early occurrence of stem girdling reduces yield, and late occurrence reduces seed size and weight. Close examination of the lesion may reveal tiny dark black dots that are spore-producing structures (perithecia) for the pathogen (Fig. 59.13). Plants that are dead or have the upper portion killed become visible after the R3 stage. These distinctive plants with their attached blackened leaves can be identified when scouting (Chase, 2012; Hadi, 2012).

Figure 59.12. Stem canker presents as an elongated lesion with a gray-brown center and a reddish-brown edge. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.13. Stem canker lesions form dark black perithecial clusters that look like dots in the lesion in order to produce ascospores. (Photo courtesy of Martin Draper, NIFA)

Causal pathogen and life cycle
Stem canker belongs to a complex of diseases referred to as the *Diaporthe/Phomopsis* complex. This *Diaporthe* (sexual stage)/*Phomopsis* (asexual stage) complex of pathogens contains *Diaporthe phaseolorum* var. *caulivora* (causal agent of Northern stem canker); *Diaporthe phaseolorum* var. *meridionalis* (causal agent of Southern stem canker); *Diaporthe phaseolorum* var. *sojae* (*Phomopsis sojae*, causal agent of soybean pod and stem blight); and *Phomopsis longicolla* (causal agent for Phomopsis seed decay of soybean) (Chase, 2012).

Diaporthe belongs to the kingdom Fungi and the Phylum Ascomycota in the Class Pyrenomycetes. *Diaporthe* produces clusters of long-necked, black fruiting bodies called perithecia (Fig. 59.13). Inside the perithecia, sac-like structures called asci produce ascospores. Unlike the apothecia produced by *S. sclerotorium*, these ascospores are not open to the air and are exuded from the long neck of the perithecia. *Diaporthe* also produces areas of mycelium called stroma. Both the perithecia and stroma are able to survive for long periods in the soil. *D. phaseolorum* overwinters typically in infested plant debris and seed. Seed infection rate is estimated to be 10-20%. Infected seed can cause reduced germination, seedling decay, and seedling mortality (Chase, 2012; Hadi, 2012).

In the spring, the pathogen on last season's residue produces spores that are carried by wind or rain to the plant surface. Infection occurs during the soybean vegetative stages, although, symptoms do not occur until reproductive stages. Infection of soybean at the V3 growth stage correlates with the highest disease severity. High rainfall and temperatures during the vegetative growth stages favors stem canker infection. Once infected, the damage can be increased by dry weather during the reproductive stages (Chase, 2012; Hadi, 2012).

Management

Due to *D. phaseolorum's* ability to survive for extended periods of time in the soil, fields with a history of stem canker are at high risk for future outbreaks. Stem canker can be managed through accurate diagnosis and a combination of control measures (Chase, 2012).

1. Use resistant or tolerant soybean varieties. Soybean varieties with resistance or tolerance to stem canker are available and should be selected particularly in fields with a history of stem canker problems (Chase, 2012; Hadi, 2012).

2. Crop rotation. Avoid planting soybeans in fields with a history of high stem canker incidence. The risk of this disease is increased by a continuous soybeans rotation or planting alternative hosts (such as alfalfa and other weeds). Thus, weed control should be maintained during the soybean-free rotation periods (Chase, 2012; Grau, 2006).

3. Tillage and cultural practices. Infected soybean residue can allow *D. phaseolorum* to survive in no-till or conservation tillage fields. Although not appropriate in all production systems, deep plowing after harvest can help manage stem canker by burying soybean residue contaminated with stem canker. Seed should be planted into a warm, fertile, well-prepared seedbed to establish seedlings with vigorous growth. Increased infection has been associated with low fertility (particularly K). Thus, maintaining good soil fertility is a cultural practice that can decrease stem canker incidence (Chase, 2012; Grau, 2006; Hadi, 2012).

4. Clean seed should be planted. As *D. phaseolorum* is a seed-borne pathogen, clean high-quality seed should be used when possible. Seed should be well cleaned to eliminate infected pieces of debris and certified to have a high germination rate. Seed treatment with fungicide will reduce stem canker, but it will not completely eliminate it (Chase, 2012; Grau, 2006; Hadi, 2012).

5. Consider applying foliar fungicides during the vegetative stages. However, this may not always be economically beneficial (Hadi, 2012).

6. Obtain an accurate diagnosis. Stem canker may be confused with white mold, PRR, or other late-season diseases. Thus, accurate diagnosis can promote accurate management (Chase, 2012).

7. Avoid delayed harvest. Soybeans in infected fields should be harvested as soon as mature. Rain-delayed harvests often allow infection to spread into seeds throughout the plant (Chase, 2012).

Brown Spot (Septoria Leaf Spot)

Brown spot is found in almost every South Dakota soybean field. Typically it is found on older leaves in the lower canopy. This disease may move up throughout the plant if the weather is warm and moisture is available. As the season progresses, infected leaves may turn rusty brown or yellow and fall off prematurely (Fig. 59.14). Brown spot is often found on the same plants as bacterial blight and is commonly misidentified as bacterial blight or soybean rust.

Symptoms

Irregular, dark brown lesions (spots) (Fig. 59.14) that vary in size are often observed on both the upper and lower leaf surfaces but are often observed first in the lower canopy (Fig. 59.15). Lesions merge together to form irregularly-shaped blotches (Fig. 59.16). Browning may occur along leaf vein or leaf edges and infected leaves can turn rusty brown or yellow and fall off the plant prematurely.

Figure 59.14. Irregular, dark brown lesions associated with brown spot infection in soybeans. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.15. Brown spot is mainly found on older leaves lower in the canopy. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.16. Brown spot infection showing lesions merging together and leaves turning yellow. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Causal pathogen and life cycle

Brown spot is caused by the fungus *Septoria glycines* and may be called Septoria leaf spot. *Septoria glycines* overwinters on infected leaf and stem debris from the previous soybean crop. Splashing rain and wind carry the spores or conidia from the soil surface to the soybean plant. Fruiting bodies develop in lesions on infected cotyledons and unifoliate leaves. Spores are developed and provide inoculum for later infections. Warm (60°F to 85°F), moist weather is optimal and favors the spread of infection and disease development. A leaf wetness period of six or more hours is required for infection. Hot, dry weather typically halts the spread of this disease.

Management

1. **Utilize crop rotation.** This allows time for soybean debris to be broken down and fruiting bodies to degrade.

2. **Employ tillage practices.** Bury residue for faster debris decay.

3. **Use fungicides.** Fungicides applied to South Dakota soybeans between bloom and pod fill can reduce disease severity and may increase yields if brown spot has been a problem. *The R3 (pod set) growth stage is recommended for fungicide application.* Brown spot has been shown to reduce yield by two to four bushels. It is a coin flip on whether it is deemed economically feasible to manage this disease.

Charcoal Rot

Symptoms

Charcoal rot can develop in South Dakota when the weather has been hot and dry. The most evident symptom of charcoal rot is a gray speckling within the lower stem (Fig. 59.17). This gray speckling is microsclerotia which are very small black structures. Early in the season symptoms may be difficult to distinguish. A reddish streak may be observed along the hypocotyl. This disease may also kill the growing point at the tip of the stem resulting in "twin stems" (two stems instead of one). Later in the season, infected plants will lose vigor and may die prematurely. Leaflets often turn yellow, then die and shrivel but often remained attached to the plant. Seedlings can become infected early, but the fungus grows slowly until the weather becomes hot and dry after flowering occurs.

Figure 59.17. Charcoal rot infection on soybeans. Note the gray discoloration observed on the root tissues. (Photo courtesy of Martin Draper, USDA-NIFA, Bugwood.org)

Causal pathogen and life cycle

Charcoal rot is caused by *Macrophomina phaseolina*. This fungus can survive in dry soil as microsclerotia for two years or longer, but is short-lived (7-8 weeks) in wet soil. The fungus can sustain itself in the soil by growing on available nutrients and plant debris. Soil populations build quickly when soybeans are grown continuously.

Management

1. **Utilize crop rotation.** This helps prevent the build-up of microsclerotia in the soil.

2. **Follow recommended plant populations.** Densely planted soybeans will compete for moisture under drought conditions which favor disease development.

3. **Select resistant varieties.** Plant varieties that are not highly susceptible to charcoal rot. Some resistance sources have been identified.

Frogeye Leaf Spot

Symptoms

Lesions (spots) are small, gray spots with reddish-brown to purple borders (Fig. 59.18). Smaller lesions may grow together to form larger, irregular spots on the leaves (Fig. 59.19). In severe cases, premature leaf drop may occur. Lesions may also occur on the pods. Lesions on pods are reddish-brown, and may appear shrunken and are often circular to elongate in shape.

Figure 59.18. Frogeye leaf spot lesions showing reddish-brown to purple borders. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.19. Small lesions growing together to form larger, irregular lesions. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Causal pathogen and lifecycle

Frogeye leaf spot is caused by *Cercospora sojina*, and is a close relative of the fungus that causes Cercospora leaf spot and purple seed stain. *Cercospora sojina* survives on crop residue left on the soil surface. Rain splashing will carry spores from plant residues to young soybean leaves. Symptom development will occur in 7 to 12 days after inoculation. Conidia will develop from these lesions and easily spread to other areas on the plant, or also spread to surrounding plants and fields. This disease is considered a polycylic disease, which means the number of lesions will continue to increase as long as conditions are favorable for disease development. Warm, humid, wet weather favors this disease's development.

Management

1. **Plant resistant varieties.** Single gene resistance has been effective in managing Frogeye leaf spot.

2. **Plant clean, pathogen-free seed.** Infected seed may contain higher levels of inoculum.

3. **Practice crop rotation.** Rotating away from soybeans helps to reduce the inoculum level in the field.

4. **Consider residue management.** Burying residues will help reduce the amount of inoculum in the field.

5. **Utilize fungicide applications.** Fungicide timing is most effective at the R3 stage of growth.

Cercospora Leaf Blight and Purple Seed Stain

Cercospora leaf blight is becoming more common in the North Central region. Yields may be reduced, while the fungus, which causes Cercospora blight, can also affect the value of the crop as it is often downgraded due to the seed quality from the color variation (purple seed stain).

Symptoms

The leaf blight may start at the beginning of seed set. A dark, reddish-purple bronzing may be observed on infected leaves (Fig. 59.20). Lesions vary in size and may be on both the leaf surface or leaf underside. Red-brown spots may develop on leaves, stems, pods, and may merge together to form large lesions.

Infected soybean seed may appear healthy or have pink to purple spots that range in size from small specks to large blotches that cover the entire seed surface (Fig. 59.21). Discoloration often extends from the hilum. Soybean yield is typically not affected by the seed stain phase of this disease, but the value of the crop is typically downgraded due to the seed quality and color variation. Infected seeds typically have higher protein content and lower oil content compared to healthy seed. Germination and seedling emergence is also reduced when compared to healthy seed. Infected cotyledons often shrivel, turn purple, and drop.

Figure 59.20. Dark reddish-purple bronzing observed on soybean leaves infected with Cercospora leaf blight. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.21. Infected soybeans displaying the purple seed stain. (Photo courtesy of Adam Sisson, Iowa State University, Bugwood.org)

Causal pathogen and life cycle

Cercospora leaf blight of soybean is caused by the fungus *Cercospora kukuchii*, a close relative of the frogeye leaf spot pathogen, *Cercospora sojina*. *Cercospora kikuchii* overwinters in infected seeds and plant residue. Spores are formed on the surface of the residue during periods of warm (75°F to 80°F), humid weather. Wind and rain move the spores to new soybean tissue where infection occurs. Seeds become infected when the fungus invades the pod and grows through the upper vein. The hilum and eventually the seed coat become infected.

Management

1. **Plant clean, pathogen-free seed.** Infected seed may contain higher levels of inoculum.

2. **Plant resistant varieties.** Use varieties with some resistance to this disease if they are available.

3. **Practice crop rotation.** Rotating away from soybeans helps to reduce the inoculum level in the field.

4. **Consider residue management.** Burying residues will help reduce the amount of inoculum in the field.

5. **Utilize a chemical/biological control.** Use a seed treatment fungicide. Foliar fungicides may be applied during early pod stages R3-R5 to help prevent blight and pod infections.

Downy Mildew

Symptoms

Early infection on the upper surface of the leaves appears as pale green to light yellow spots which enlarge into bright yellow spots (Fig. 59.22). The underside of the leaf will appear gray and fuzzy (Fig. 59.23). The centers of the spots turn brown and are bordered by yellow margins. Infected seeds have a dull white appearance and are partially or completely covered in a coating of fungal spores.

Figure 59.22. Early infection of downy mildew. Note pale green lesions on upper surface of soybean leaf. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.23. Underside of soybean leaf showing downy mildew infection. Note the gray and fuzzy appearance. (Photo courtesy of Virginia Tech Plant Pathology Archive, Virginia Polytechnic Institute and State University, Bugwood.org)

Causal pathogen and life cycle

Downy mildew is caused by the fungus, *Peronospora manshurica*. There are 33 reported races of this fungus, and this number is projected to increase as more research is done. *P. manshurica* overwinters in crop residues and on the surface of seed. Wind and rain move the spores on the plants causing infection which can spread quickly throughout the field during periods of cool, wet, or humid weather.

Management

1. **Plant resistant varieties.** Use varieties with some resistance to this disease.
2. **Plant clean, pathogen-free seed.** Infected seed may contain higher levels of inoculum.
3. **Practice crop rotation.** Rotating away from soybeans helps to reduce the inoculum level in the field.
4. **Consider residue management.** Burying residues will help reduce the amount of inoculum in the field.

Powdery Mildew

Symptoms

White, powder-like patches form on all plant parts (Fig. 59.24). Patches continue to enlarge and merge until plant parts are covered. Different cultivars may develop different symptoms such as chlorosis or yellowing, leaf scorching, rusty colored patches, and premature leaf drop. Damage is caused by the fungal utilization of nutrients produced by the plant and by thick mats of fungal mycelium blocking the sunlight reaching the leaf and reducing photosynthesis.

Figure 59.24. White, powder-like patches observed on soybeans infected with powdery mildew. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Causal pathogen and life cycle

Powdery mildew is caused by the fungus *Microsphaera diffusa*. Infection occurs when conidia (asexual spores) land, germinate, and penetrate the epidermal cells. The conidia form germ tubes and attach to the cells via an anchorage structure. An infection peg forms under the anchorage structure and penetrates the epidermis. This allows the first feeding structure (haustoria) to form. The rest of the fungus body (mycelium) grows over the epidermal cells. Chains of conidia soon develop and become wind-borne, start new infections, and repeat the disease cycle until soybean plants mature. Cool weather (65°F to 76°F) favors disease development while temperatures above 86°F halt the growth and reproduction of the fungus. Black sexual fruiting bodies are sometimes produced in mildew colonies late in the fall. It is believed that the sexual spores are released in the spring and serve as a means of primary infection.

Management

1. **Plant resistant varieties.** Use varieties with some resistance to this disease if they are available

2. **Utilize fungicide applications.** Foliar fungicides may be used to control powdery mildew, but they haves not been very economical.

Brown Stem Rot

Brown stem rot is often confused and misdiagnosed with another soybean disease called Sudden Death Syndrome. A great diagnostic check for brown stem rot of soybean involves splitting the lower stem of affected plants and checking for brown discoloration. Healthy plants will have white pith.

Symptoms

The most common symptom of brown stem rot is the brown to reddish- brown discoloration of the stem pith (Fig. 59.25). In severe infections, the outside base of the stem can have a greasy appearance. When present, foliar symptoms consist of wilting, chlorosis, and tissue browning between veins. Brown interveinal tissue surrounded by yellow to green tissue along the veins represents typical foliar symptomatology.

Figure 59.25. Brown stem rot brown discoloration of stem pith. (Photo courtesy of Martin Draper, USDA-NIFA, bugwood.org)

Causal pathogen and life cycle

Brown stem rot is caused by *Phialophora gregata*. *Phialophora gregata* survives in crop residues left of the soil surface. The brown stem rot fungus produces spores that germinate, invade soybean roots, and progresses into the vascular systems of soybean seedlings. Cool weather (60°F to 80°F) is needed during pod development for disease infection. After pod formation, symptoms of brown stem rot can be found in affected plants.

Management

1. **Practice crop rotation.** Rotating away from soybeans helps to avoid build-up of brown stem rot fungus levels in the field.

2. **Plant resistant varieties.** Use varieties with some resistance to this disease if they are available.

3. **Select the best cultivars.** Plant cultivars with shorter relative maturity.

4. **Consider residue management.** Deep burying of residue reduces the survival of the fungus but this practice should also be used with long crop rotation sequences.

5. **Manage soil fertility.** Maintain optimum soil fertility and pH for soybean production.

Sudden Death Syndrome (SDS)

Sudden death syndrome (SDS) is often confused with brown stem rot as foliar symptoms often resemble one another. When diagnosing Sudden Death infection, be sure and look at the pith color. SDS-infected soybeans will have a white or slightly cream pith color while brown stem rot infected soybeans will have a brown pith color. Disease development is favored by cool, wet soils. There is discussion that fields infected with soybean cyst nematode should be scouted for sudden death syndrome. To date, SDS has not been officially identified in South Dakota.

Symptoms

Symptoms first appear as small chlorotic spots on soybean leaves. Spots coalesce (come together) until the entire leaf is chlorotic. Chlorosis of the leaves continues until necrosis occurs which gives the leaves a fired look (a very bold and striking difference in color) (Fig. 59.26). Leaves appear tattered as the necrotic tissue gives way (falls off) from the leaf. Leaf veins typically remain green, and the leaves often drop off the plant leaving the petioles still attached to the stem. The foliar symptoms develop very rapidly. Roots are often rotted and may have a blue-colored fungus (Fig. 59.27) on them. When roots are split, the pith remains white to a slightly creamy white color.

Figure 59.26. Chlorosis and necrosis displaying the fired look (very bold and striking color) associated with sudden death syndrome in soybeans. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.27. Blue fungus is often observed on roots infected with sudden death syndrome. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Causal pathogen and life cycle
Sudden death syndrome is caused by the soilborne fungus *Fusarium solani f. sp. glycines*. The fungus overwinters as chlamydospores in crop residues or in the soil. If soybean cyst nematodes are found, the field should also be checked for SDS. The fungus can also be found in soybean cyst nematode cysts. SDS fungus is found in the soil and can be also found in soybean cyst nematode cysts but does not mean the disease (SDS) will develop. When the soil is wet, the fungus produces a toxin which invades the roots of the plant. These toxins are then translocated to the leaves.

Management
1. **Delay planting date.** Cool, wet soils make young soybean plants vulnerable to sudden death syndrome infection.

2. **Utilize tillage practices.** Compacted soils which retain water and restrict root growth of plants promotes SDS development. Correcting soil drainage and compaction with the use of tillage helps reduce the risk of SDS.

3. **Plant resistant varieties.** Some of the SDS-resistant varieties also have resistance to soybean cyst nematode (SCN).

Anthracnose
Symptoms
Warm, moist weather conditions favor the development of anthracnose. Symptoms of anthracnose include reddish veins, rolled leaves (Fig. 59.28), dark blotches on stems (Fig. 59.29), pods (Fig. 59.30), and leaf petioles. Sunken, dark brown lesions may appear on the cotyledons of soybean seedlings. Later in the season, a hand lens may be used to observe black spines on the infected areas. Premature defoliation may also occur. Infected seed may have no symptoms while others may develop brown or gray areas with black specks.

Figure 59.28. Anthracnose-infected soybean plants displaying rolled leaves. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.29. Dark blotches displayed on soybean stems infected with anthracnose. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Figure 59.30. Anthracnose infection on soybean pod. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Causal pathogen and life cycle

The primary pathogen that causes anthracnose in the Midwest is the fungus *Colletotrichum dematium* var. *truncatum*. It overwinters in crop residues and infected seeds. This disease overwinters in infested crop debris and is spread by conidia (spores). It has also shown to be seedborne.

Management

1. **Practice crop rotation.** Rotating away from soybeans helps to reduce the inoculum level in the field.
2. **Consider residue management.** Burying residues will help reduce the amount of inoculum in the field.
3. **Plant clean, pathogen-free seed.** Infected seed may contain higher levels of inoculum.
4. **Utilize fungicide seed treatments.** Use of a fungicide seed treatment may be beneficial for reducing disease infection.

Soybean Rust

Soybean rust has not been found in South Dakota. This disease dies back every year to the freeze line in the United States (U.S.) and causes significant yield loss in unmanaged soybean fields in the southern U.S. Foliar fungicides are labeled for disease control here in South Dakota for if and when we get this disease.

Symptoms

Lesions first appear as small, chlorotic, irregularly-shaped spots which turn to tan or brown or reddish in color as the disease progresses (Fig. 59.31). Lesions are usually confined to veins or close to the veins. Soybean rust pustules are typically observed on the underside of the leaf (Fig. 59.32) but may also occur on stem, pods, and petioles. The fungus produces spores in cone-shaped pustules (Fig. 59.33). Soybean rust causes premature defoliation and yield loss.

Figure 59.31. Soybean rust observed on soybean leaves. (Photo courtesy of Florida Division of Plant Industry Archive, Florida Department of Agriculture and Consumer Sciences, Bugwood.org)

Figure 59.32. Soybean rust pustules shown under magnification. (Photo courtesy of Reid Frederick, USDA Agricultural Research Service, Bugwood.org)

Figure 59.33. Cone-shaped rust pustules. (Photo courtesy of Daren Mueller, Iowa State University, Bugwood.org)

Causal pathogen and life cycle
Soybean rust is caused by the fungus Phakopsora pachyrhizi and Phakopsora meibomiate. Development of soybean rust is favored by prolonged periods of leaf wetness with temperatures between 46°F to 82°F. Rust pustules appear on the leaf surface 9 to 10 days after infection. Spores are easily spread by the wind and splashing rain. Rust pathogens are considered obligate pathogens because they survive on living plant material. Soybean rust is not able to overwinter in the Midwest. The disease dies back to the freeze line in the southern United States. It overwinters on kudzu that is not killed back by freezing, and it can produce spores for the primary inoculum that infects southern soybeans. This pathogen travels by wind currents and has to be reintroduced each year to the Midwest.

Management
1. **Use foliar fungicides.** The first fungicide application should be made before rust appears or shortly after onset of the disease. Scouting and application is beneficial through the R1-R5 soybean growth stages.

2. **Plant resistant varieties.** Use varieties with some resistance to this disease if they are available.

Fungicide Treatments
Information on the effectiveness of different products on foliar diseases is available in https://extension.sdstate.edu/south-dakota-pest-management-guides and Giesler and Gustafson (2008) (http://ianrpubs.unl.edu/epublic/live/g1862/build/g1862.pdf). Always read and follow label directions for approved uses of these products, and check with the South Dakota Department of Agriculture for up-to-date state product registration information.

To reduce the risk of resistance development, fungicides with different modes of action should be used. The following fungicide information has been modified from Larry Osborne at http://www.sdaba.org/agronomyconference/pdfs/FungicidesOsborne.pdf.

Strobilurin fungicides
- High risk for fungicide resistance.
- Products include: AproachTM (picoxystrobin); Evito® (fluoxastrobin); Headline® (pyraclostrobin).
- Quadris®(azoxystrobin); VertisanTM (penthiopyrad).

Triazole fungicides
- Broad spectrum and may be effective against rusts.
- High risk of resistance.
- Products include Alto® (cyproconazole); Domark® (tetraconazole); Folicur® & other generics (tebuconazole); Laredo® (myclobutanil), and Tilt® (propiconazole).

Strobilurin + Triazole premixes
- Evito T® (fluoxastrobin + tebuconazole); Stratego® (propiconazole, trifloxystrobin); Stratego YLD® (prothioconazole, trifloxystrobin); Quilt® (azoxystrobin, propiconazole); Quilt Xcel® (azoxystrobin(2x), propiconaxole), Priaxor® (pyraclostrobin, fluxapyroxad).

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