Disease identification and management are integral parts of South Dakota’s wheat production. Diseases can affect agronomic traits (such as growth and stand) and reduce yield. They also contribute to inferior seed, lower grain quality, and market rejection due to mycotoxin concentrations. The purpose of this chapter is to discuss how to recognize wheat diseases and possible management options.

Scouting and control principles for diseases

Fungal, bacterial and viral pathogens cause critical diseases that reduce South Dakota wheat yields. The first step in diagnosing a problem is recognition. Thus, crop scouting is critical to assess the actual risk of a particular disease in the field. Scouting is the basis for integrated disease management and provides the information needed for when and where to apply chemical, cultural or biological control measures (Chapter 21). A wheat disease scouting calendar (Table 23.1), which includes a summary of management considerations, is located at the end of the chapter.

Fungicides are well-known methods of disease control (Osborne and Stein 2009; Ruden and Osborne 2011). Wheat fungicide management is most economical when:

1. Fungicides are used in response to actual disease risk rather than as a prescriptive application without a risk assessment; and
2. Fungicides are used as part of an integrated disease management strategy and not when they are the only control method.
Well-adapted disease resistant varieties (Hall et al. 2011) should be combined with good cultural practices such as crop rotation, disease-free seed, and optimal planting dates. Foliar fungicides are effective only in managing diseases caused by fungi and do not offer direct protection against bacterial or viral diseases, nematodes, or abiotic stresses.

To maximize the return on a fungicide application, select the least expensive fungicide with the highest efficacy on the target disease (when warranted). The North Central Regional Committee on Management of Small Grain Diseases, a working group of plant pathologists from north central universities, has developed an efficacy rating of commonly used fungicides based on field trials conducted across multiple years and locations. A summary of the latest efficacy rating trial (conducted in 2010) is given in Table 23.2, which is shown at the end of the chapter.

Foliar fungicide applications on wheat are most profitable if timed to protect the flag leaf, as the flag leaf contributes up to 75% of a producer's grain yield. In cases where disease pressure is high early in the growing season, it may be necessary to apply a fungicide before flag leaf emergence for early season disease suppression. The decision to apply a foliar fungicide depends on several factors including:

1. Favorability of the environment to disease development (disease risk).
2. Susceptibility of the variety planted to disease.
3. Fungicide application cost.
4. Yield saved due to fungicide application.
5. The market price of wheat.

Wise use of fungicides should also include rotation of the chemical class utilized in order to limit the development of pathogen strains with resistance toward a particular class of fungicide.

**Fungal Diseases**

There are many fungal diseases that can and do attack wheat, but only a few are routinely responsible for major economic losses. Foliar fungal diseases of primary concern include: tan spot, powdery mildew, stem rust, leaf rust, stripe rust, Stagonospora (Septoria) leaf blotch, Fusarium head blight or scab, and root rot diseases.

**Tan spot**

*Symptoms:* Infection may occur on all above-ground plant parts, but symptoms are most commonly found on leaves. The symptoms start as small brown freckles that grow into oval or lens-shaped lesions (⅛ to ½ inch long and ¼ to ⅛ inch wide) with prominent yellow halo and tan to dark brown centers (Fig. 23.1). Spots may coalesce forming larger necrotic areas (Fig. 23.2), and the leaves may eventually wither. Tan spot is usually more severe on lower leaves and then progresses upward.
**Causal pathogen:** Tan spot is caused by the fungus *Pyrenophora tritici-repentis*. There are eight recognized races of *P. tritici-repentis*. Each race produces different host-selective toxins (HST) on susceptible wheat varieties and is often geographically distinct in distribution.

**Life cycle:** The causal pathogen of tan spot overwinters as pinhead-sized fruiting bodies called pseudothecia on crop residues in the field. During wet periods in the spring, ascospores are produced and released as primary inoculum. Infection occurs first on the lower leaves and spreads upward. As the disease progresses, conidia are produced on infected tissues and spread by wind for secondary infection. Kernels can be infected if heavy infection occurs on the flag leaves. Infected kernels can sometimes show red smudges on the surface. Damp (frequent rains) moderate temperatures (68–82.4°F, 20–28°C) are favorable for disease development. Infected plants usually produce smaller kernels. Yield loss can reach 50% in highly susceptible varieties.

**Management:**

1) Wheat varieties resistant to tan spot are available and effective in managing the disease.

2) Reduced tillage cropping system stands have an increased tan spot risk due to the pseudothecia on stubble and debris.

3) Removal or destruction of residues known to be infected by tan spot is effective in decreasing the tan spot risk in subsequent wheat seasons.

4) Crop rotations can reduce the inoculum pressure from tan spot in a given field. Rotate wheat with broad leaf crops since these crops are known as non-hosts of *P. tritici-repentis*. Corn is not a tan spot host, but planting wheat on corn residue may increase the risk of Fusarium head scab.

5) Planting pathogen-free seeds is recommended. Seedlings growing from infected seeds have reduced vigor. If seeds infected with the pathogen are planted, seed treatment can reduce the risk of seedling infection.

6) Foliar fungicides that are effective in suppressing tan spot are available. However, application of foliar fungicide is not always profitable. In situations where wheat is planted on the previous year's infected residue, a susceptible variety is used. When spring weather is warm and rainy, foliar fungicide may or may not be economically beneficial.

**Powdery mildew**

**Symptoms:** Powdery mildew-infected plants show numerous raised, white, powdery spots on the aerial plant surface (Fig. 23.3). These spots are, in fact, the vegetative strands (mycelium) and spore masses of powdery mildew colonies. As the colonies age, the spots may turn grey with cleistothecia (structures that produce sexual spores and help the pathogen survive during the winter) visible on them (Fig. 23.4). Powdery mildew is prevalent in the lower canopy and humid environments.
Causal pathogen: Powdery mildew on wheat is caused by *Blumeria graminis* f. sp. *tritici*. Several races of this pathogen exist.

Life cycle: Powdery mildew pathogen survives the winter in plant debris left on the field. Ascospores (spores formed in the cleistothecia) are produced in the spring to act as primary inoculum. The fungus produces structures (haustoria) that directly penetrate the plant tissue while maintaining a network of fine white mycelial filaments and spore producing structures on the plant surface. Conidia, a type of asexual spores, are produced on the surface of infected plants throughout the growing season, facilitating the disease spread. Temperatures between 60–70° F (15.5–21.1° C) with damp weather are favorable for powdery mildew buildup. Disease development is retarded when temperatures are higher than 77° F (25° C). High planting rate elevates the humidity within the lower canopy and increases disease development.

Management:

1) Deploying wheat varieties with resistance toward powdery mildew is critical. Wheat varieties differ significantly in their reactions to powdery mildew.

2) Since *B. graminis* overwinters on crop residues in the field, reduced tillage practices may increase the risk of powdery mildew infection. Destruction of volunteer wheat, tillage and crop rotations reduce the risk of powdery mildew infection.

3) Over fertilization with nitrogen may increase the risk of powdery mildew. Plants with increased nitrogen are more susceptible to powdery mildew. Nitrogen promotes tiller formation and, inadvertently, produces a favorable environment for powdery mildew development. Balanced fertilization regimes with proper levels of N, P and K should be utilized.

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**Figure 23.3.** Numerous raised powdery spots characteristic of powdery mildew on wheat. (Image: Clemson University - USDA Cooperative Extension Slide Series, Bugwood.org)

**Figure 23.4.** Cleistothecia (the overwintering structure) produced on mature powdery mildew colonies. (Image: Department of Plant Pathology Archive, North Carolina State University, Bugwood.org)
4) Foliar fungicides that are effective in suppressing powdery mildew are available. However, application of foliar fungicide is not always profitable. Conditions where wheat is planted on the previous year’s infected residue, a susceptible variety is used and the weather is mild and humid are favorable for the disease and should be considered when making a decision on fungicide application.

RUSTS ON WHEAT

Leaf rust, stem rust, and stripe rust affect wheat in South Dakota. These rusts have very complex life cycles requiring five spore stages. **Teliospores** overwinter on plant debris and soil and begin this complex life cycle in the spring when they germinate. **Basidiospores** (sporidia) are produced on germinating teliospores which then infect the leaves of the alternate host. Soon after very small pycnial pustules occur. Pyenia produce **pycniospores** and receptive hyphae. Once receptive hyphae of a pyenia are fertilized by pycniospores of the opposite mating type, mycelia grow through the alternate host leaf producing **aecia** (cluster cups) on the underside of the leaf. Aecia produce **aeciospores**, which are windborne and capable of infecting wheat plants. On wheat, aeciospores germinate and penetrate into the plant tissue. In a week or two, infecting mycelium starts to produce uredinia which bear **urediospores**. Urediospores are the only rust spores that are able to re-infect wheat plants throughout the growing season. As the plants mature, telia start to develop and teliospores form within the telia. Unfortunately, rust urediospores can also initiate infections on wheat when aeciospores are not present.

**Leaf Rust**

*Symptoms:* Leaf rust pustules are orange to dark reddish-brown, raised, powdery, small (usually 1 mm or less in size), oval shaped, often found on the upper leaf surface (Fig. 23.5). Thousands of spores (contained in each pustule) are then dispersed by the wind. Using your finger you can distinguish rust pustules from other leaf spot diseases by rubbing (smearing) the colored spores on the leaf surface.

*Causal pathogen:* Leaf rust on wheat is caused by *Puccinia triticina* (*P. recondita* f. sp. *tritici*).

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**Figure 23.5. Leaf rust pustules on wheat.**
(Image: Ida Paul, Small Grain Institute, Bugwood.org)
**Life cycle:** Leaf rust has a complex life cycle involving five different spore types and two types of host: wheat as the primary host and meadow rue (*Thalictrum* sp.) as the alternate host. *Puccinia triticina* overwinters on infected wheat in the southern states and Mexico, and the urediospores, one of the spore types produced by leaf rust pathogen, are carried northward by the wind. Upon alighting on wheat leaf surface, moisture is required for urediospores to germinate and infect wheat leaves. If environmental conditions are favorable, a new generation of urediospores may be produced every 7 to 14 days. Light rain, high humidity, or heavy dew and temperatures ranging between 59° and 77° F (15–25° C) are ideal for rust development. Leaf rust continues to spread by means of wind-blown urediospores (from plant to plant and from field to field) until the wheat matures. *Puccinia triticina* does not survive the winter in the northern latitudes, and for the disease to start in any given year, new inoculum must be introduced from the southern latitudes.

**Management:**

1) Fungicide application is recommended to control leaf rust if the disease is established in the crop canopy and weather favors rust development prior to heading.

2) Many wheat varieties are resistant to the various rusts (Hall et al. 2011); however, the development of new races may break a variety's resistance.

http://pubstorage.sdstate.edu/AgBio_Publications/articles/EC774-11.pdf

**Stem Rust**

**Symptoms:** Stem rust appears as dark orange to brick-red, raised, powdery pustules with ragged edges. Lesions are often large and can be found on stems and leaf sheaths (Fig. 23.6).

**Causal pathogen:** Stem rust is caused by *Puccinia graminis* f. sp. *tritici*.

**Life cycle:** Stem rust has a complex life cycle which requires a susceptible host (wheat and some varieties of barley, oats, rye, wild barley, and goat grass) and an alternate host (common barberry, *Berberis vulgaris*). It also produces five different spore types, produced in five different fruiting bodies, in order to complete its life cycle. Of these fruiting bodies and spore types, spermagonia (pycnia) containing spermatia (pycniospores) and aecia containing aeciospores are found on the alternate woody host. Uredia (which produce urediospores) and telia (which produce teliospores) are found on wheat or other grassy hosts. Stem rust overwinters as teliospores in colder climates and urediospores on fall-planted wheat in warmer climates. In South Dakota, wind-blown urediospores are carried upward from the southern latitudes and serve as primary inoculum for wheat infection. Stem rust infection can occur between 65–85° F (18.3–29.4° C) when free moisture is available (dew, light rain, humidity).

*Figure 23.6. Stem rust on wheat.*
(Image: William M. Brown Jr., Bugwood.org)
Management:

1) Plant resistant cultivars (Hall et al. 2011). Resistance is the primary means of control for stem rust. Remember, the emergence of new races require constant vigilance.

http://pubstorage.sdstate.edu/AgBio_Publications/articles/EC774-11.pdf

2) Eradicate woody hosts.

3) Fungicide application is usually not necessary or cost effective as long as resistant cultivars are used. If a new race is identified, than a fungicide application will be warranted.

Stripe Rust

Symptoms: Small, yellow-orange pustules arranged in rows (stripes) on leaves of wheat (Fig. 23.7). Rows of pustules often resemble sewing machine stitches.

Causal pathogen: Stripe rust on wheat is caused by Puccinia striiformis f. sp. tritici.

Life cycle: Cool, wet weather favors stripe rust development. Rapid disease development occurs between 50° and 60° F (10–15.5 C) when moisture is available, while temperatures over 68° F (20° C) for several days in a row inhibits disease development. Stripe rust needs a green host (wheat, perennial grassy weeds) to survive and has been known to overwinter under snow cover as dormant mycelium. Stripe rust spores are blown into South Dakota from neighboring wheat-producing states to the south.

Management:

1) The use of cultivar resistant varieties is the main means of stripe rust control (Hall et al. 2011). http://pubstorage.sdstate.edu/AgBio_Publications/articles/EC774-11.pdf

2) Control volunteer wheat and grassy weeds in order to eliminate the green living bridge.

3) The use of fungicides is recommended for stripe rust control (Ruden and Osborne 2011). http://pubstorage.sdstate.edu/AgBio_Publications/articles/FS917.pdf

4) Fungicides effective for wheat leaf rust should be effective for stripe rust control.

Stagonospora (Septoria) leaf blotch

Symptoms: Infected leaves develop dark tan, linear to irregular-shaped lesions (Fig. 23.8). Dark brown fruiting bodies called pycnidia develop later on the lesions. Lesions will often develop a yellow halo, but are distinguishable from tan spot by the presence of pycnidia. On mature leaves, the lesions may coalesce forming larger brownish dead areas (Fig. 23.9). The glumes may be infected and produce purple brown blotches with ash grey areas (Fig. 23.10). This phase of the disease is usually called glume blotch.

Causal pathogen: Stagonospora leaf blotch is caused by the fungus Stagonospora nodorum which was formerly known as Septoria nodorum.
**Life cycle:** The pathogen causing Stagonospora leaf blotch survives the winter as mycelia or fruiting bodies (either pycnidia or pseudothecia) on crop residues. In the spring, the fungal fruiting bodies produce spores that are carried by wind and rain-splashed onto plant tissues. After infecting the tissues, the fungal pathogen produces conidia that can infect adjacent plants (Fig. 23.11). Temperatures ranging between 68–80.6°F (20–27°C) together with 6–16 hours of high humidity are favorable for the pathogen’s development. The conidia produced on leaves may be splashed to heads, which lead to head infection especially during wet summers.
Management:

1) Plant varieties with moderate resistance.
2) Similar to tan spot and powdery mildew, the pathogen causing Stagonospora leaf blotch survives the winter on crop residues. Thus, burying the inoculum into the soil by tillage decreases the risk of the disease.
3) Continuous wheat crop rotations allows for the buildup of inoculum in a given field. A three-year crop rotation with two years of non-cereal crops is recommended.

**Fusarium head blight or scab**

*Symptoms:* In infected plants, spikelets on the whole or parts of the head appear bleached (Fig. 23.12). Pink or orange-red spore masses are sometimes visible at the base of the glumes, especially during long periods of high humidity. Purple-black perithecia, fruiting bodies of the pathogen, can sometimes be seen on older scab infections. Scab infection is favored by warm, wet weather for two weeks before flowering and continuing through the flowering period.

*Causal pathogen:* The fungus, *Fusarium graminearum*, causes head blight. Apart from wheat, this fungal pathogen also infects sorghum, oats, barley and corn. The pathogen causes stalk rot on sorghum and ear rot on corn.

*Life cycle:* *Fusarium graminearum* overwinters on small grains and corn residues in the field. In the spring and summer, the fungi can continue to multiply on the above-ground residues. Spores produced are rain-splashed or carried by wind on to the head. Spores can also be blown from long distant sources. The spores landing on wheat heads may germinate and grow through the anthers into the glumes. Critical time of infection occurs between the start of flowering and the hard dough of kernel development.

Prolonged periods of high humidity (2–3 days) coupled with temperatures ranging between 75° and 85°F (23.9–29.4°C) are favorable for disease development. Infection may also occur under lower temperatures, if the period of high humidity exceeds three days. Scabby wheat may have elevated levels of the mycotoxin, deoxynivalenol (DON). This toxin may have negative impact on livestock performance (Box 23.1, at end of chapter).
ROOT ROT DISEASES

There are a number of pathogens that can cause root rot problems in South Dakota wheat. These include Common root rot complex, Fusarium foot rot, and Take-all. More than one root rot pathogen is often found infecting the same plant. Stressed plants are more susceptible to root rot diseases.

Common root rot symptoms: Infections may take place at any plant growth stage. Seedlings will often display dark brown lesions on the roots, subcrown internodes, crowns, and lower leaf sheaths (Fig. 23.13). Chocolate-brown leaf spots are often found on lower leaves. Surviving seedlings may be wilted, stunted, and chlorotic. Sterile white heads may result from premature death of the plant when infection is severe. Common root rot is favored by dry, droughty conditions.

Fusarium foot rot symptoms: Roots are often brown, the subcrown internode is discolored, and a chocolate brown or reddish brown lesion often extends up the plant stem (Fig. 23.14). A pink, cottony fungal growth may sometimes be found in the interior of the lower stem when it is split open. Fusarium foot rot also produces sterile white heads and premature plant death. The severity of this disease is also worsened by prolonged drought and dry conditions.

Management:

1) Wheat varieties differ in their response toward Fusarium graminearum. No varieties are immune, but some are moderately resistant. Choosing varieties with some levels of resistance should be the first management option in areas with scab history.

2) Since the pathogen survives on crop residues on the soil surface, tillage should reduce the risk of scab inoculum carrying over from the previous year. Crop rotation with broad leaf crops is also efficient in reducing the inoculum buildup in a given field. The highest risk of scab infection occurs in continuous wheat cropping or in wheat planted on previous year's corn residue.

3) Seed treatment reduces the incidence of wheat scab due to usage of infected seeds (Ruden and Osborne 2011); however, seed treatment does not reduce the risk of subsequent scab infection. http://pubstorage.sdstate.edu/AgBio_Publications/articles/FS949.pdf

4) Foliar fungicide application at flowering (when the anthers are still yellow) can be effective in reducing wheat scab and DON. Use the wheat scab risk assessment tool to make a fungicide application decision. http://www.wheatscab.psu.edu/riskTool_2011.html

Figure 23.12. Bleached spikelets characteristic of Fusarium head blight or scab. (Image: Mary Burrows, Montana State University, Bugwood.org)
**Take-all symptoms:** Roots and crowns appear as a shiny black color (Fig. 23.15 and Fig. 23.16). White heads are often observed. In contrast to Common root rot and Fusarium foot rot, Take-all is favored by poorly drained wet soils. Take-all also tends to be more severe near the field edge where it uses grassy weeds for alternate hosts.

![Figure 23.13. Chocolate-brown lesions of common root rot located on roots and lower stems.](Image: Mary Burrows, Montana State University, Bugwood.org)

**Causal pathogens:** *Bipolaris sorokiniana* causes common root rot complex of wheat. This fungus can also infect most small grains and numerous grasses. Take-all is caused by *Gaumannomyces graminis* var. *tritici*. Fusarium root rot (dryland root rot) is caused by several different species of *Fusarium* spp.

![Figure 23.14. Brown discoloration of crown and roots observed on Fusarium infected wheat.](Image: Ernesto Moya, Bugwood.org)

![Figure 23.15. Shiny black discoloration on roots and crowns of wheat infected with Take-all.](Image: Mary Ann Hansen, Virginia Polytechnic Institute and State University, Bugwood.org)
Life cycle: These diseases are soil borne. The fungus spores persist in soil on old stubble and root debris.

Management:
1) Crop rotation with a broadleaf crop.
2) Plant varieties with moderate resistance.
3) Use fungicide treated seed.

BACTERIAL DISEASE
The main bacterial disease that attacks South Dakota wheat is black chaff, or bacterial leaf streak. Just a reminder, foliar fungicides do not offer protection against bacterial diseases.

**Black chaff/Bacterial leaf streak**
*Symptoms:* The disease earns its name, black chaff, from the darkened glumes of infected plants. This symptom is easily confused with glume blotch caused by glume infection of *Stagonospora nodorum*. Plants with black chaff/bacterial leaf streak, however, show a diagnostic sign of cream to yellow ooze (bacterial exudates) on the plant surface especially in humid weather. The ooze starts as viscous liquid. The bacterial ooze may appear as distinct droplets or a thin sheet of exudates. The bacterial ooze later dries and appears as shiny areas on the plant surface. Small water-soaked spots or streaks usually appear on infected leaves. The lesions may enlarge, usually yellowish in color and elongate in shape (Fig. 23.17). As they developed, the lesions become necrotic (Fig. 23.18).
Causal pathogen: Black chaff/bacterial leaf streak on wheat is caused by the bacterium *Xanthomonas campestris* pv. *translucens*.

Life cycle: The bacteria that causes bacterial leaf streak can be introduced via infected seeds, which are estimated to be the highest factor in bacterial leaf streak introduction into wheat fields with no history of the disease. *Xanthomonas campestris* pv. *translucens* can also survive as epiphytes on volunteer wheat due to its large host range that includes grassy weeds. Alternatively, the pathogenic bacteria can survive on crop debris in the field. However, bacterial survival decreases when the debris decomposes.

Inoculum in crop debris is splashed during rainfall onto healthy tissues. The bacteria enter the host plant through stomatal openings or wounds. Droplets of bacterial ooze during high humidity periods may act as inoculum for secondary infection. The disease can develop under a relatively wide range of temperature (59–86°F or between 15 and 30°C) and humidity levels. Infection is enhanced under frequent rainfall.

Management:
1) Usage of certified, pathogen-free seeds is the primary option to manage bacterial leaf streak.
2) Controlling volunteer cereal and grassy weeds around a wheat field reduces the disease pressure on the field.
3) There are currently no wheat varieties with high levels of resistance against bacterial leaf streak.
4) Susceptibility varies among wheat varieties and usage of highly susceptible varieties in fields with history of bacterial leaf streak should be avoided.

VIRAL DISEASES

In South Dakota, the two main viral diseases that attack wheat are *Wheat streak mosaic virus* and *Barley yellow dwarf virus*. Depending upon the year and stage of growth, wheat streak can cause a 10 to 80% loss in yield, while barley yellow dwarf can cause a 10 to 40% yield reduction. Just to reiterate, *foliar fungicides are not effective and do not offer protection against viral diseases.*
**Wheat Streak Mosaic Virus (WSMV)**

*Symptoms:* The major symptoms of wheat streak mosaic are stunted plants with mottled and streaked leaves. Light green and dark green or yellow and green mosaics that coalesce into streaks are characteristic of this disease (Fig. 23.19). In severe reactions, streaks may become necrotic as disease progresses. Other symptoms include:

1) Spreading rather than erect growth (prostrating).
2) Delayed heading.
3) Reduced tillering.
4) Sterility or poor seed set.
5) Poor grain fill.
6) Reduced yields.

![Figure 23.19. Yellow streaking and mottling symptoms associated with Wheat streak mosaic virus (WSMV) infection on Lyman wheat.](Image: Marie A.C. Langham, South Dakota State University)

*Causal pathogen:* The pathogen causing this disease is *Wheat streak mosaic virus* (WSMV). WSMV is transmitted from plant to plant by the wind-blown wheat curl mite (*Aceria tosichella* Keifer) (Fig. 23.20). Both the mites and virus survive winters on seeded and volunteer winter wheat and perennial grasses.

![Figure 23.20. Wheat curl mite, the vector of Wheat streak mosaic virus.](Image: Frank Peairs, Colorado State University, Bugwood.org)

*Life cycle:* Infection typically takes place in the fall, but disease expression is often not observed until the spring. Mites and virus survive the winter in the crown of winter wheat and other perennial grasses. Wheat streak mosaic can also infect oats, barley, corn, sorghum, millets, and many other grass species. Symptoms are often found first along the edge of a field or in patches near wheat volunteers. WSMV is the most important endemic (always here, but varies in amount) viral disease in South Dakota.
Management:

1) Avoid early planted winter wheat. Winter wheat planting should be delayed until after mid to late September for optimum WSMV control.

2) Volunteer grasses should be controlled at least two weeks before planting a new crop.

3) Early planted spring wheat is at less risk (mite populations increase with warmer temperatures) than late-planted spring wheat.

4) Use wheat cultivars with the most tolerance/resistance available in your area.

Barley yellow dwarf/Cereal yellow dwarf

Symptoms: The symptoms and severity of the disease are dependent on the age of the wheat when infected. In drought situations, infected wheat seedlings grow to only $\frac{1}{3}$ or $\frac{1}{2}$ of their normal size; the leaves suffer from chlorosis, and the heads may not be completely filled. If infection occurs after tillering, stunting is minimal or not present. Depending on the anthocyanin level of the wheat variety, the infected leaves turn bright yellow or reddish purple color (Fig. 23.21). Symptomatic plants can either appear singly or in circular groups within the field (Fig. 23.22). Plants infected by BYDV/CYDV have stunted root growth and, consequently, the impact of BYDV/CYDV infection is exacerbated in drought years.

Causal pathogen: Viruses causing barley yellow dwarf/cereal yellow dwarf are Barley yellow dwarf virus (BYDV) and Cereal yellow dwarf virus (CYDV). There are several distinct strains of these viruses: BYDV-PAV, BYDV-MAV and CYDV-RPV. Each strain is optimally transmitted by distinct aphid species and their geographic distribution varies. In South Dakota, BYDV-PAV is the predominant species causing barley yellow dwarf.

Over 100 species in the grass family are affected by the disease, including many common grain crops such as barley, wheat, oats, sorghum, rye, triticale, corn, and rice and numerous wild grasses. The wide host range provides many potential hosts for the virus in the absence of cultivated commodities. Wild annuals, perennial grassy weeds, volunteer cereals and neighboring cultivated grain crops may act as alternative hosts for the virus.

Life cycle: BYDV and CYDV are transmitted exclusively by aphids. A number of aphid species can transmit these viruses from plant to plant; two of the most important are bird cherry-oat aphid and English grain aphid. The winged form of bird cherry-oat aphid has a pale to dark green abdomen. The non-winged form is broadly oval and olive to greenish black in color with brownish coloration on the rear end of the body.
Both forms of bird cherry-oat aphid have black tailpipes (cornicles) and dark cauda (tail). The non-winged form of English grain aphid is medium sized, broadly spindle shaped, yellowish green to dirty reddish brown in color, with black tailpipes and pale cauda. The winged form of English grain aphid is similar in color with distinct dark markings across the back. Bird cherry-oat aphid is known to migrate in early fall while English grain aphid usually migrates in spring or summer.

The aphid vectors overwinter in the southern parts of the country and migrate northward in summer and early fall. Migration seems to be encouraged by cool (50° to 68° F or between 10° and 20° C) humid weather. Migrating aphids may introduce the virus or transmit the virus from infected perennial weeds to wheat. Infected wheat acts as inoculum source for viral spread in the field.

**Management:**

1. Planting date management is important in managing the yellow dwarves. Planting winter wheat after the migration season of bird cherry-oat aphid may reduce the risk of early BYDV/CYDV infection.
2. Even though highly resistant varieties are not available, wheat varieties vary in their response to BYDV/CYDV infection. Usage of varieties with low susceptibility against BYDV/CYDV is recommended.
3. Since perennial grassy weeds and volunteer wheat can act as virus reservoir for fall and spring infection, destruction of these plants around a wheat field will reduce the risk of BYDV/CYDV infection.
4. Fungicide has no effect on virus infection and development inside the wheat plant.
5. Insecticide seed treatment or foliar insecticide application can decrease aphid population and activity on wheat, thus reducing the spread of the disease within a field. However, usage of these chemical options is only economically feasible in years with high BYDV/CYDV severity. Since it is difficult to forecast the risk of BYDV/CYDV infection, it is similarly difficult to predict whether seed treatment or foliar insecticide will be profitable in a given year.

**Box 23.1. Mycotoxin**

Mycotoxin concentration can limit end-use or reduce profits due to dockage or rejection at the point of sale. In the case of wheat, *Fusarium* spp. produces the mycotoxin, deoxynivalenol (DON), otherwise known as vomitoxin. FDA animal feed guidelines are shown as follows:

1. Cattle and chickens – 10 ppm not to exceed 50% of the diet.
2. Swine – 5 ppm not to exceed 20% of the diet.
3. All other animals – 5 ppm not to exceed 40% of the diet; can reduce weight gain and feed refusal at lower levels.
4. Human consumption – FDA recommendation < 1 ppm.
Additional information and references


Acknowledgements

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Table 23.1. Wheat disease scouting calendar for South Dakota. This chart is adapted from the 2011 field crop pest calendar published by SDSU Extension.

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<thead>
<tr>
<th>Disease</th>
<th>Wheat stage</th>
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<td>Fall</td>
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<td>Tan spot</td>
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<td>Powdery mildew</td>
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<td>Stagonospora leaf blotch</td>
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<td>Glume blotch</td>
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<tr>
<td>Stripe rust</td>
<td></td>
</tr>
<tr>
<td>Stem rust</td>
<td></td>
</tr>
<tr>
<td>Fusarium head blight / wheat scab</td>
<td></td>
</tr>
<tr>
<td>Root rot diseases</td>
<td></td>
</tr>
<tr>
<td>Black chaff / bacterial leaf streak</td>
<td></td>
</tr>
<tr>
<td>BYDV</td>
<td></td>
</tr>
<tr>
<td>Winter wheat</td>
<td></td>
</tr>
<tr>
<td>Spring wheat</td>
<td></td>
</tr>
<tr>
<td>WSMV</td>
<td></td>
</tr>
<tr>
<td>Winter wheat</td>
<td></td>
</tr>
<tr>
<td>Spring wheat</td>
<td></td>
</tr>
</tbody>
</table>

1. Effective in-season management options for diseases such as black chaff/bacterial leaf streak, root rot complex, WSMV or BYDV are either not available or are not consistently profitable. Any decision regarding disease management should be taken before planting. Examples of management options for these diseases typically include planting date management, selection of resistant varieties, usage of disease-free seed, crop rotation and, in the case of root rot complex, fungicide seed treatment. Details on each option are discussed under individual disease sections.

2. At tillering (Chapter 3), scout for early season leaf diseases (tan spot, powdery mildew, stagonospora leaf blotch and stripe rust).
   a. Light infection is normal and tolerable.
   b. Economic treatment using early rates of fungicides is warranted only when all of the following conditions are met.
      i. The field contains heavy stubble from previous wheat crop.
      ii. The variety seeded is susceptible or moderately susceptible.
      iii. There has been abundant rainfall.

Early application of fungicides is typically conducted at half the recommended rates. This increases the risk of fungicide resistance development among the field pathogen population. To prevent fungicide resistance development, early applications of strobilurin fungicides (e.g., Headline®, Quadris®, etc.) should not be followed by flag leaf applications with products with the same class of active ingredients (Table 23.1).
3. At flag leaf emergence, check for leaf spot/blotch and rust diseases.
   a. Check flag leaf and two leaves below for signs of leaf spots/blotch, leaf and stripe rust.
   b. If rust or heavy leaf spots (10% leaf area on 1st leaf below flag or 20% leaf area on 2nd leaf below flag) are present, consider fungicide application.

*Note that varieties resistant to leaf and/or stripe rusts only need protection for leaf spot/blotch.*

4. After head emergence, check for flowering stage. Stage of anther development is important in the timing of some fungicide applications and for checking if flower development was harmed by unfavorable weather conditions.
   a. Monitor the weather forecast at this time. Wet soils, heavy dew, frequent rainfall, and warm temperatures favor scab infection.
   b. If favorable weather conditions prevail and susceptible varieties are planted, consider scheduling fungicide application (Table 23.1) near or at the peak of flowering (when the anthers are still yellow).

5. Three weeks after flowering, check for scab development.
   a. Greater than 10% disease may translate into elevated toxin levels in grain.
   b. Explore insurance and/or marketing options, may require pre-harvest testing.
Table 23.2 Efficacy of fungicides for wheat disease control based on appropriate application timing.\(^1\)

<table>
<thead>
<tr>
<th>Fungicide</th>
<th>Powdery mildew</th>
<th>Tan spot/ Stagonospora leaf blotch</th>
<th>Leaf rust</th>
<th>Stripe rust</th>
<th>Stem rust</th>
<th>Fusarium head scab</th>
<th>Harvest restriction (days before harvest)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active ingredient (Product example)</td>
<td>Rate/ A (fl. oz)</td>
<td>Applied at flag leaf (Feekes 8-9)</td>
<td>Applied between emergence and flowering</td>
<td>Applied at flowering (Feekes 10.51)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Class: Strobilurin – high risk fungicide resistance</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Azoxystrobin Quadris 2.08 SC</td>
<td>6.2 – 10.8</td>
<td>F</td>
<td>VG – E</td>
<td>E</td>
<td>E</td>
<td>VG</td>
<td>NL</td>
</tr>
<tr>
<td>Fluoxastrobin Evito 480 SC</td>
<td>2.0 – 4.0</td>
<td>G</td>
<td>–</td>
<td>VG</td>
<td>–</td>
<td>–</td>
<td>NL</td>
</tr>
<tr>
<td>Pyraclostrobin Headline SC</td>
<td>6.0 – 9.0</td>
<td>G</td>
<td>VG – E</td>
<td>E</td>
<td>E</td>
<td>G</td>
<td>NL</td>
</tr>
<tr>
<td><strong>Class: Triazole – medium risk of fungicide resistance</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Cyproconazole Alto 100 SL</td>
<td>3.0 – 5.5</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>30 days</td>
</tr>
<tr>
<td>Metconazole Caramba 0.75 SL</td>
<td>10.0 – 17.0</td>
<td>VG</td>
<td>VG</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>G</td>
</tr>
<tr>
<td>Propiconazole Tilt 3.6 EC</td>
<td>4.0</td>
<td>VG</td>
<td>VG</td>
<td>VG</td>
<td>VG</td>
<td>VG</td>
<td>P</td>
</tr>
<tr>
<td>Prothioconazole e Proline 480 SC</td>
<td>5.0 – 5.7</td>
<td>–</td>
<td>VG</td>
<td>VG</td>
<td>–</td>
<td>VG</td>
<td>G</td>
</tr>
<tr>
<td>Tebuconazole Folicur 3.6 F</td>
<td>4.0</td>
<td>G</td>
<td>VG</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>F</td>
</tr>
<tr>
<td>Prothioconazole e + Tebuconazole Prosaro</td>
<td>6.5 – 8.2</td>
<td>G</td>
<td>VG</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>G</td>
</tr>
<tr>
<td><strong>Class: Triazole + Strobilurin</strong></td>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Metconazole + Pyraclostrobin TwinLine 1.75 EC</td>
<td>7.0 – 9.0</td>
<td>G</td>
<td>VG – E</td>
<td>E</td>
<td>E</td>
<td>VG</td>
<td>NL</td>
</tr>
<tr>
<td>Propiconazole 11.7% + Azoxystrobin 7.0% Quilt 200 SC</td>
<td>14.0</td>
<td>VG</td>
<td>VG</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>VG</td>
</tr>
<tr>
<td>Propiconazole 11.7% + Azoxystrobin 13.5% Quilt Xcel 2.2 SE</td>
<td>14.0</td>
<td>–</td>
<td>VG*</td>
<td>VG</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Propiconazole 11.4% + Trioxystrobin 11.4% Stratego 250 EC</td>
<td>10.0</td>
<td>G</td>
<td>VG</td>
<td>VG</td>
<td>VG</td>
<td>VG</td>
<td>NL</td>
</tr>
<tr>
<td>Propiconazole 22.6% + Trioxystrobin 22.6% Absolute 500 SC</td>
<td>5.0</td>
<td>G</td>
<td>–</td>
<td>E</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

\(^1\)This table is based on the work of North Central Regional Committee on Management of Small Grain Diseases (last update on April 2011), and is provided only as a guideline. Pesticide applicators are responsible to read and follow all current label directions. No endorsement is intended for any of the listed products. We assume no liability resulting from the use of these products.

\(^2\)Other products containing the same active ingredients may also be labeled in some states.

**Efficacy categories:**

- NL = Not labeled and Not Recommended
- P = Poor
- F = Fair
- G = Good
- VG = Very Good
- E = Excellent
- -- = Insufficient data to make statement about the efficacy of this product.