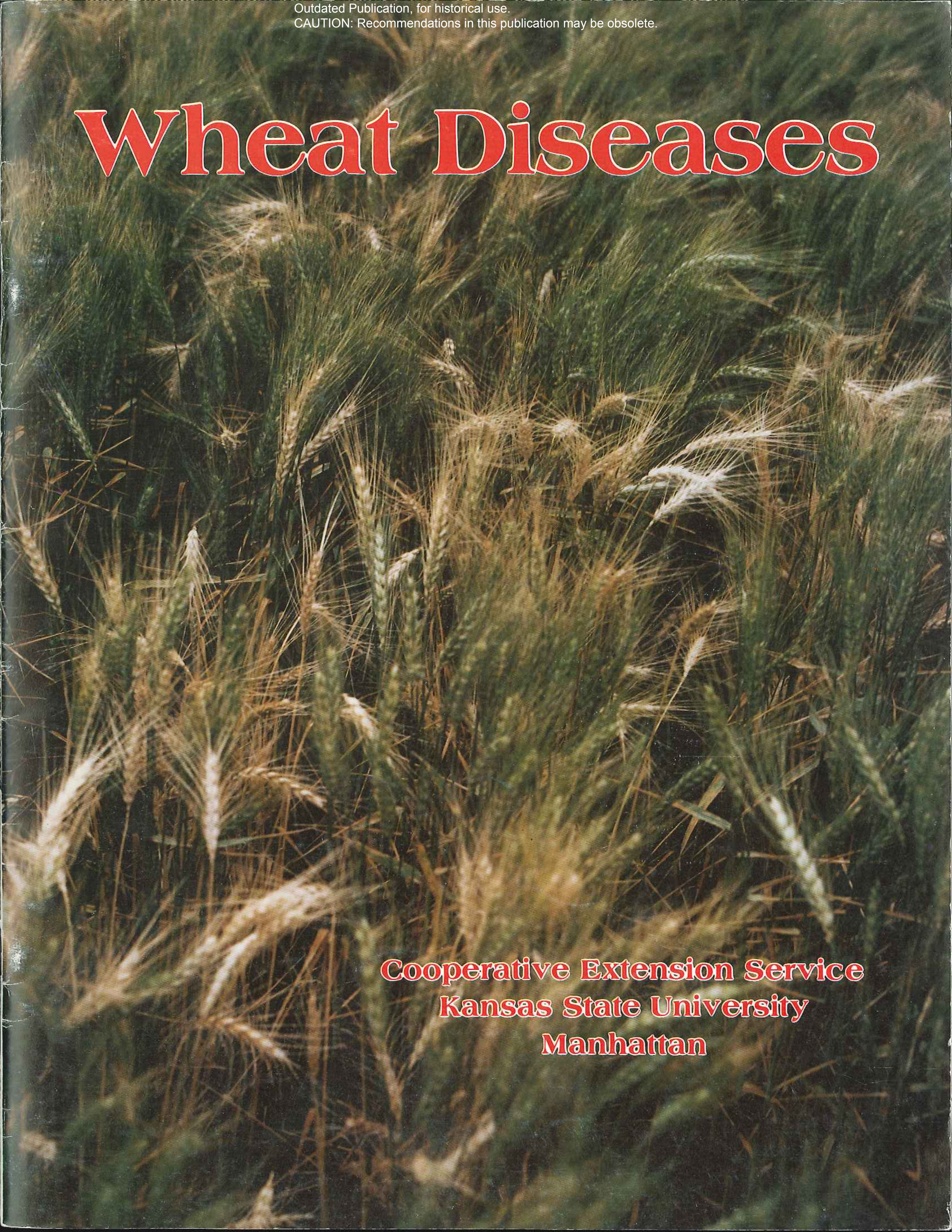
Wheat Diseases



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Wheat Diseases

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Introduction

Wheat diseases destroy 10 to 25 percent of our Kansas wheat and reduce quality of harvested grain. Estimated average annual percent losses from 1976-1983 were as follows.

Soil-borne mosaic	2.9
Wheat streak mosaic	1.8
Speckled leaf blotch	1.8
Tan spot	1.6
Leaf rust	1.5
Cephalosporium stripe	1.3
Take-all	1.2
Barley yellow dwarf	0.8
Powdery mildew	0.6
Winter injury/root and crown rots	0.6
Scab	0.3
Smuts	0.1
Bacterial leaf blight	trace
Stem rust	trace

Disease incidence and severity constantly change. In six of those eight years, a different disease caused the highest loss. Changing management practices, especially those pushing for maximum yields will increase disease pressure.

Every wheat grower should recognize and understand diseases in his field. This is the first necessary step in control. This bulletin describes and illustrates symptoms, details conditions for disease development and gives control measures. Specific varieties and chemicals which change yearly are not included. Current, regularly updated recommendations should be consulted for these.

Virus Diseases

Kansas wheat is affected by three important diseases caused by viruses. Viruses are so small they cannot be seen even with a light microscope and so must be studied by their effects on plants. They cause mosaic, yellowing, stunting and growth distortions. They multiply inside cells of their host and require living hosts for survival. Once inside cells they take command and divert energy from plant growth to virus reproduction. Insects and mites transmit some viruses when they feed on diseased then healthy plants. Organisms which transmit pathogens are called vectors.

Wheat Soil-Borne Mosaic

Soil-borne mosaic (SBM) is one to the most serious wheat diseases in eastern and central Kansas. It often causes losses up to 50% in affected fields planted to susceptible varieties and has caused an estimated average annual loss of 2.9 percent statewide since 1976.

The disease is most easily recognized in early spring by the distinct, irregularly shaped areas of yellow wheat. Once these areas appear there is no further spread to unaffected wheat plants **(Photo 1).** Affected plants are stunted and bronzy-yellow **(Photo 2).** Leaves are mottled with a yellow background and darker green islands giving the mosaic name to this disease **(Photo 3).**

1. Soil-borne mosaic. Early spring field pattern. (courtesy of Tom Sim).

2. Soil-borne mosaic. Yellow stunted plants.

3. Soil-borne mosaic. Leaf mosaic with "green islands".

Mosaic symptoms gradually fade away, but stunting often persists until maturity. Symptoms fade quickly with warm temperatures, but persist into April and May if temperatures are cooler than normal. The longer mosaic and stunting persist the greater the yield loss **(Photo 4).**

4. Greenbug spots are browner and less sharply defined than soil-borne mosaic.

Soil-borne mosaic is caused by the Wheat Soil-borne Mosaic Virus. It lives in association with a soil fungus vector *(Polymyxa graminis)* which enables it to survive and infect wheat plants. The fungus has a resting spore stage which persists in soil for many years. When adequate moisture and a host are available this spore germinates, swims to a wheat root, and invades a root hair carrying the virus with it. The virus multiplies and spreads in the plant diverting energy from growth. Plants are infected in the fall and occasionally shows symptoms then but the yellow areas are usually not obvious until late February to mid-March.

The swimming spore stage requires free moisture so SBM is most serious following wet fall weather. In dry areas it is usually found only in drainage channels and irrigated fields. In wet cycles small patches of SBM can be found in dry land wheat as far west as Highway 283. Serious losses requiring control are ordinarily limited to the state east of Highway 281. It became especially severe in central Kansas during the late 1960's and early 1970's. Rye, barley and some annual weedy bromes are hosts but are not important in the virus survival or spread.

Soil-borne mosaic is effectively controlled with resistant varieties **(Photo 5).** Some yield loss probably results in resistant varieties planted in infected fields but excellent yields are still achievable. Resistance depends on a single major gene with some minor genes but has been very stable and reliable. Mosaic symptoms in highly resistant varieties are probably caused by some other virus.

5. Soil-borne mosaic susceptible and resistant varieties.

Wheat spindle streak virus has been reported from Nebraska and tentatively identified in a few fields in Kansas. Symptoms of this virus are similar to SBM and it is carried by the same fungus.

There is no practical way known to rid the soil of the SBM virus. Rotation out of wheat to other crops for several years has not been effective.

Any field which has ever shown symptoms of SBM should be planted to a resistant variety. All varieties from the Kansas State University breeding program in recent years are highly resistant.

Adequate nitrogen fertility will help SBM diseased plants recover to some degree but it will not cure the disease.

Wheat Streak Mosaic

Wheat streak mosaic is a very serious disease in the western 1/3 of Kansas. It can cause near total yield loss over wide areas in epidemic years. Severity is extremely variable. Average annual estimated loss is 1.8 percent state-wide since 1976. In 1981 it was 7 percent.

Streak mosaic symptoms typically begin showing in early April as yellow stunted strips along field margins adjacent to where volunteer wheat grew the previous summer. This yellowing and stunting becomes more severe over the next several weeks and gradually spreads across the field **(Photo 6).** The earliest emerging wheat, often volunteer, is usually most severely affected **(Photo 7).**

Individual plants are yellow, stunted, wilted and tillers are often partially prostrate. Root development is weak **(Photo 8).**

Leaf mosaic begins in younger leaves as light green to yellow dashes which enlarge to give a streaked appearance to the leaf. Finally the whole leaf blade turns yellow. The younger a plant is when infected the more severe the symptoms and loss. Late infection causes obvious light and dark green streaks on flag leaves but not much stunting **(Photo 9).**

6. Wheat streak mosaic. Volunteer wheat in summer fallow is the source of virus and curl mites. Severity decreases with distance from volunteer.

8. Wheat streak mosaic. Fall infected plants are stunted and prostrate.

7. Wheat streak mosaic. Early volunteer strip is severely diseased and later seeded wheat is unaffected.

9. Wheat streak mosaic. Mosaic pattern on flag leaves from late infection.

Cool, moist spring weather reduces severity and hot, dry weather increases it.

Streak mosaic is caused by the Wheat Streak Mosaic Virus (WSMV) which is carried from diseased to healthy plants by the microscopic wheat curl mite, *Aceria tulipae*—the vector. The virus survives only on green living tissue of cultivated grass crops or some wild grass hosts. When infected plants die, the virus can no longer be spread from the dead tissue. This precarious existence then requires either perennial grass hosts or alternating between summer and winter annuals.

Curl mites are extemely small, less than 1/100

10. **Wheat curl mite adults and eggs through a microscope. (courtesy of Tom Harvey).**

11. **Wheat curl mite. Scanning electron micrograph. (courtesy of Merton Brown).**

inch long, requiring a microscope for observation **(Photos 10 and 11).** The life cycle, from egg through two larval stages to the adult and egg, can be completed in seven to ten days. Under ideal conditions one adult could theoretically produce several million offspring in 60 days. They reproduce most rapidly at 75 degrees through 80 degrees fahrenheit, very slowly at 48 degrees fahrenheit, and essentially stop at 32 degrees fahrenheit. However, all stages can survive for at least three months at near freezing temperatures and several days at 0°F so they have little trouble living over Kansas winter. They revive and can reproduce during warm days in mid-winter. Hot, dry summer weather slows reproduction but they survive our hottest weather if adequate green wheat or grass is available for food. Their major weakness is that in summer they die very quickly when deprived of food. In the laboratory without food and water, curl mites live less than 8 hours at 75 degrees fahrenheit and between 1 and 2 days at 36 degrees fahrenheit.

Curl mite larvae acquire the wheat streak mosaic virus within a few minutes when feeding on sap of infected plants. They retain the ability to transmit the virus to healthy plants for at least a week following acquisition.

They crawl very slowly and depend almost entirely on wind for movement from plant to plant. After landing on a suitable host they crawl to the youngest unrolled leaf in the whorl, attach themselves to the upper surface by an anal sucker and begin to feed and reproduce. Their feeding prevents the leaf from unrolling normally so tightly rolled leaf edges result. Heavy populations cause enough leaf rolling that expanding leaves and heads may be trapped; this is a good indication in the field of heavy infestation.

As wheat plants mature, the curl mites migrate to the last remaining green tissue including the ripening grain. When grain is shattered before harvest, especially as a result of hail, the mites attached to grains can sometimes survive long enough to move to the sprouting seedling. If mites are carrying the virus, these new volunteer seedlings are infected very early.

This succession of events results in our most severe epidemics. The most devastating streak mosaic often follows the exact path of hail near maturity the previous year.

Without early emerging volunteer wheat the virus and mites die quickly following wheat maturity. Hot, dry July weather reduces mites and virus drastically, but enough survive on grasses so they are not eliminated. Wet summer weather permits volunteer wheat to grow and the earlier it emerges and the curl mites become established the higher the population will be to blow into fall seeded wheat.

Curl mites crawl to upper leaf tips, especially when plants are drouth stressed, release their attachment and are easily carried by wind. They drop on newly emerged wheat only by chance and this is why such huge populations are necessary for epidemic development. They can be carried several miles by wind but severe infection usually develops only within one-fourth to one-half mile of volunteer fields. The furthest spread is typically to the northeast of volunteer in the direction of prevailing winds. This pattern of mite dispersal explains why streak mosaic appears first, and is most severe, along the field edge next to volunteer and decreases with distance. Epidemics sometimes occur over wide areas and fields are uniformly infected with streak mosaic. We do not know what conditions permit this.

Warm weather in October and November extends the period when mites move and reproduce and increases streak mosaic severity. There is increasing evidence that warm weather in February and March can result in further spread of the mites and virus and cause significant losses. But this has not been carefully documented.

Many other mites and insects have been

tested but the wheat curl mite is the only vector found.

Wheat is the preferred host for curl mites and streak mosaic virus. Other small grains; oats, barley and rye, are slightly susceptible but do not show obvious symptoms nor are they damaged significantly. Triticale reactions have not been extensively tested and probably vary with each cross.

A few corn hybrids are quite susceptible to the virus and many hybrids support high populations of the mites under husks during late summer. Occasionally severe streak mosaic is found adjacent to late maturing corn which was still green when seeded wheat emerged. But most corn matures early enough that the mites die before seeded wheat emerges. This probably prevents serious epidemics from occurring where wheat is planted in dry corners of irrigated corn circles.

Sorghums are immune to WSMV and very poor curl mite hosts. Proso millet *(Panicum miliaceum)* is a good host for both mites and virus and it would be a serious source of the virus if it were widely grown.

Some grasses are hosts, but very few permit increase of both virus and mites sufficient for serious epidemics. The winter annuals, goat grass and weedy bromes, can support both but do not stay green through the summer long enough to be important.

Summer annuals such as sandbur, crabgrass, barnyardgrass, stinkgrass, witchgrass, and foxtails have been proved susceptible so are potential oversummering hosts. Western wheatgrass, buffalograss and smooth brome are perennials immune to the virus but can support mite populations. Hairy grama, Canada wildrye, Virginia wildrye and bermudagrass are perennials which can host curl mites and WSMV. All these grasses theoretically could be important in streak mosaic epidemics but we rarely see this happen. These grasses are reservoirs for the long time survival of mites and virus but apparently do not build these up in high enough amounts at the right time. Low levels of streak mosaic can be found in most fields, most years. Severe outbreaks are almost always traceable to volunteer wheat.

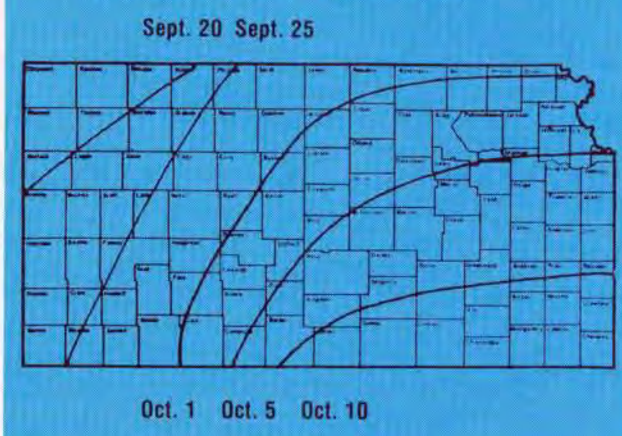
Streak mosaic epidemics and high losses are most likely with one or more of the following conditions:

1. Hail near maturity which shatters viable grain resulting in early volunteer.
2. Wet July which encourages volunteer.
3. Cool summer for high curl mite survival.
4. Warm, dry fall which increases mite reproduction and movement.
5. Early wheat seeding.
6. Warm late winter weather permitting further infection.
7. Hot, dry late spring which stresses disease weakened plants.

Control of wheat streak mosaic is achieved by managing volunteer wheat and planting time to reduce populations and activity of wheat curl mites to a minimum when seeded wheat emerges. These controls are essential following hail storms which shatter viable seed.

1. Prevent volunteer from growing or kill it 2 or 3 weeks before seeded wheat emerges. This can be done with either tillage or herbicides but must be very thorough since a few live plants or green tissue can support enough virus carrying mites to infest a lot of seeded wheat. Pasturing off volunteer wheat is ineffective since enough green tissue remains for mite survival. Killing volunteer after seeded wheat has emerged is too late—infection has already occurred even though symptoms may not become obvious until spring. Chemical control of curl mites in volunteer wheat has not proved effective.
2. Plant late. The Hessian fly-free date has been adopted as the standard for wheat streak mosaic. This varies from Sept. 15 in extreme northwest to Oct. 13 in southeast.

Figure 1
Approximate safe dates to plant wheat to reduce wheat streak mosaic.



These are only approximations based on normal temperatures. The dramatic reduction in streak mosaic with later planting has been documented many times with research and field observations.

In South Dakota the effect of planting date on streak mosaic was studied ten years. In four of those years streak mosaic was severe, in three it was moderate, and three it was very low.

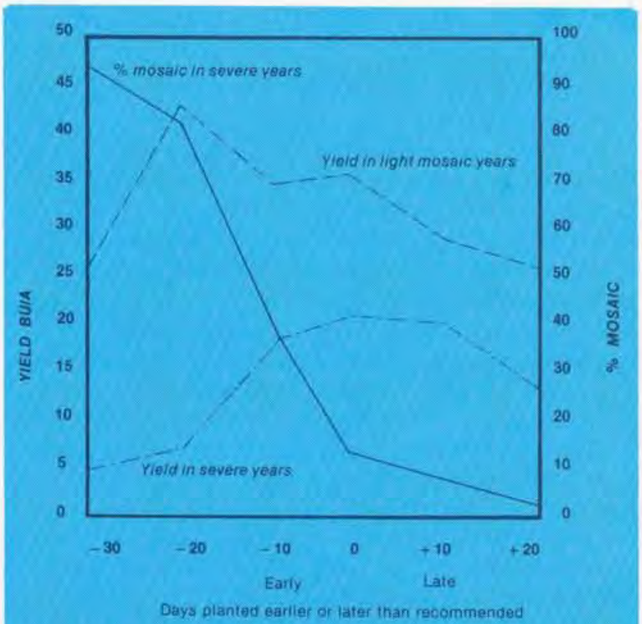


Figure 2. Planting date, streak mosaic severity and wheat yield.

Ten year study by Wayne S. Gardner, South Dakota State University.

This study clearly demonstrates several important points:

1. Streak mosaic is most severe with early planting.
2. Late planting decreases streak mosaic dramatically.
3. Streak mosaic causes severe yield loss.

The search for streak mosaic resistant varieties has been long, difficult and disappointing. No adapted varieties with acceptable quality and good streak mosiac resistance are available. Some sources of resistance have been unstable with warm fall weather or have been linked to poor milling and baking quality. Present research is directed toward combining low levels of resistance to both the mites and the virus from several sources.

At present, no variety can be planted early next to infected volunteer wheat and consistently escape serious disease.

Barley Yellow Dwarf

Barley yellow dwarf (BYD) can be found in low amounts every year but only occasionally (1959, 1976) has caused serious losses in the past thirty years. Distribution is erratic but BYD is usually more prevalent in eastern Kansas.

Stunting and yellowing are the most obvious symptoms. Stunted plants often appear in spots up to several feet in diameter with those in the center most severe and decreasing toward the perimeter **(Photo 12).** This corresponds to establishment, reproduction and spread of virus carrying aphids. Scattered individual plants may also be affected.

12. Barley yellow dwarf. Stunted area and yellow leaf tips.

Symptoms are usually noticed in late spring at about jointing but late infection may show only yellow tipped flag leaves. Leaf yellowing begins at the leaf tip and along the margins and progresses toward the middle and base with the midrib remaining green longest. There may be some blotchy appearance but no distinct mosaic. With cooler than normal temperatures flag leaves may have reddish-purple tips **(Photo 13).** Sometimes leaves may be darker green than normal and stiff with some distortion. Root systems are reduced, heading is sometimes prevented, winter kill increased and yields reduced. Symptoms are more pronounced under cool temperatures, long days and abundant sunshine. Fall infection causes much more serious stunting and loss than spring infection.

13. Barley yellow dwarf. Typical yellowing and purpling from flag leaf tips.

Barley and oats are more susceptible than wheat and exhibit more distinctive symptoms. Barley is severely stunted and in the advanced stage bright yellow. In oats the disease is called red leaf because susceptible varieties turn a brilliant reddish-brown.

Symptoms in wheat overlap with wheat streak mosaic, soil-borne mosaic, crazy top, environmental stresses and nutrient deficiencies, making BYD difficult to diagnose.

The disease is caused by the Barley Yellow Dwarf Virus (BYDV). It is transmitted only by aphid feeding **(Photo 14).** Wheat, oats, barley, rye, corn, sorghum, brome, tall fescue, bluegrass, bermudagrass, little bluestem, and a large number of other perennial and annual grasses are hosts to the virus. Many of these do not show symptoms when infected but are constant, ready sources of the virus.

BYDV is transmitted by several aphids. Greenbugs, oat bird cherry aphids, corn leaf aphids and English grain aphids are the most common vectors. Aphids acquire the virus by feeding on diseased plants and after a one to two day latent period, can transmit the virus to a healthy plant by feeding. After acquisition, aphids are able to transmit the virus as long as they live. Symptoms can appear one to three weeks after transmission but are usually not obvious in the fall.

With plentiful virus hosts, abundant aphid vectors and susceptible wheat, it is surprising that we do not have serious epidemics more often.

Control of BYD is difficult. It is not practical to try reducing or eliminating the virus since it is present in so many different plants and can be carried long distances by aphid vectors. With several species of aphids involved, the problem becomes even more complex. Insecticide conrol of vectors might help but the unpredictable nature of aphid dispersal and disase development make it impossible to know when application is necessary or effective.

14. Aphid vectors of barley yellow dwarf virus. Light colored aphid is a greenbug and dark one is an oat bird cherry aphid. (courtesy of Dell Gates).

Varieties differ in susceptibility but none are highly resistant.

Planting late reduces the most serious fall infection and since it also reduces wheat streak mosaic, it is a practical control.

Virus diseases sometimes look alike. But they are quite different in when they develop and how symptoms show and persist in the field. The table on the following page compares these similarities and differences.

Table 1. Wheat Virus Disease Comparisons

	SOIL-BORNE MOSAIC	WHEAT STREAK MOSAIC	BARLEY YELLOW DWARF
WHEN SYMPTOMS APPEAR	1-2 weeks after spring growth begins. Rarely in fall.	4-6 weeks after spring growth begins. On volunteer in fall.	6-8 weeks after spring growth begins.
PATTERN IN FIELD	Distinctly affected areas often in wetter spots.	Most intense along edge near volunteer wheat and diminishing with distance. Spread in the direction of prevailing wind.	Random circular spots or 2-6 feet in diameter.
STUNTING	Severe—but may partially recover with warm weather.	Severe and persists to maturity	Some—often most severe in center of spots.
LEAF MOTTLING AND COLOR	Mosaic of pale yellow leaves with darker green islands of color.	Bright yellow with streaked patern. Mosaic most prominent on upper leaves.	Leaf tips bright yellow or reddish-pruple. Flag leaf symptoms most distinct.
OTHER SYMPTOMS	Poor roots. Increased winter-kill.	Prostrate tillers. Curling and trapping of leaves. Wilted, drouthy. Poor roots.	Increased winter-kill. Blackened glumes. Poor roots.
TEMPERATURE EFFECTS	Warm—symptoms fade. Cool—symptoms persist and stunting permanent.	Symptoms increase as weather warms.	Cool—more reddish purple. Warm—more yellow.
VECTORS	Soil inhabiting fungus.	Wheat curl mite.	Several aphids.
SOURCE OF VIRUS	Fungus in soil.	Early volunteer wheat, and some grasses.	Many native and introduced grasses.
CONDITIONS FAVORING DISEASE	Wet soil in fall. Cool temperatures in April.	Early volunteer from hail at harvest. Volunteer left until after seeded wheat emerges. Warm fall.	Many aphids in fall. Cool spring.
WHERE SERIOUS	Eastern 2/3 of Kansas	Western 1/2 of Kansas	Erratic
CONTROL	Resistant varieties.	Destroy volunteer. Late planting.	Late planting.

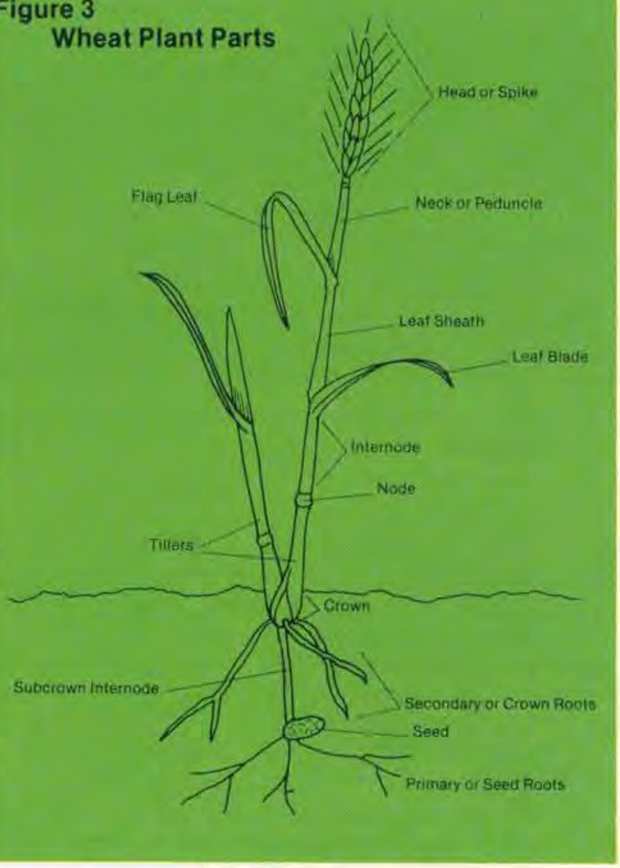
Root and Crown Rots

Some root and crown rots develop in every wheat field. The extent of damage may range from a few roots killed to weakened plants and occasionally extensive areas of dead plants. It is difficult to diagnose and measure losses since we do not know at what level these rots reduce yield or interact with other problems. Symptoms often overlap and are interrelated with those caused by winter injury, drouth, drowning or herbicides.

These rots are caused by a complex of soil inhabiting fungi which can survive for long periods in varied weather conditions, cropping sequences, tillage practices and soil types. The take-all fungus is an exception and is covered in a separate section. Often two or more fungi can be found in a field or even within the same plant. Several different fungi cause root and crown rots.

Helminthosporium sativum and several *Fusarium* species are both seed and soil-borne. One or more species of *Rhizoctonia* causes sharp eyespot. Several species of *Pythium* rot roots in cool wet soils.

Figure 3
Wheat Plant Parts



Seed Rots and Seedling Blights

Good quality seed planted in a good seedbed with optimum temperatures for quick germination is seldom seriously hurt by seed rots or seedling blights. But with less than ideal conditions, several fungi may rot the seed before germination, or kill or stunt seedlings before or soon after emergence. Invaded tissue is brown and soft. Brown or black color on the sub-crown internode is the best indicator **(Photo 15).** This is most prominent where the seed is attached but may extend up to the crown. The crown may also be affected and secondary roots killed. Poor stands and weakened plants result. Stand losses in Kansas are seldom serious enough to require replanting. Losses are worse in cool, wet falls, so are most likely in eastern Kansas.

15. Seedling blight. Dark colored lesions are evidence of fungus attack.

Planting good seed with the best possible germination conditions will usually prevent significant seed rot and seedling blight. Seed treatment fungicides protect germinating seedlings long enough for them to get established and will improve stands. This protection does not last long enough to prevent later stages of root and crown rot.

Winter Injury/Crown Rot

When wheat plants die during the winter, or soon after they resume spring growth, it is usually from a combination of cold injury and root and crown rots working together **(Photos 16 and 17).** Plants in low vigor are more easily injured by cold and this in turn makes them more vulnerable to attack by rot pathogens. Crown tissue is brown, soft and mushy. Secondary roots may be rotted off **(Photo 18).** Some injury is evident on most wheat plants at the end of a Kansas winter. Given decent growing conditions, plants with adequate vigor form new crown roots and recover.

The following conditions are often associated with winter injury/crown rot:

1. Poor root establishment.
2. Loose dry seedbed.

16. **Winter injury/crown rot is often most prominent on terrace tops where snow blows off.**

17. **Winter injury/crown rot. Wheat may make some spring growth then die in irregularly shaped patches.**

18. **Winter injury/crown rot. Crowns and secondary roots are brown and soft.**

3. Warm period followed by a sudden temperature drop.
4. Lack of snow cover during coldest weather.
5. Winter tender varieties.

Some of these conditions are obviously uncontrollable. But plantng winter hardy varieties in a good firm seedbed early enough for vigorous establishment will reduce the winter injury/crown rot complex.

Common or Dry Land Foot Rot

Common or dry land foot rot encompasses a complex of rots caused by *Helminthosporium* and *Fusarium* species. Some infection can be found in most wheat plants but it becomes severe only with drouth stress and consequently is worst in western Kansas. It is impossible to tell where the drouth stress ends and foot rot begins. Plants are stunted in patches and tillering is reduced **(Photo 19).** The crown is dark brown on the outside and dirty brown internally **(Photo 20).** Discoloration does not extend far into the roots so they appear fairly normal. If drouth persists, plants ripen prematurely and show "whiteheads". *Fusarium* sometimes invades the lower internode causing a medium brown coloration and a pink color on lower nodes **(Photo 21).**

19. **Common or dry land foot rot. Affected areas are stunted and drouthy.**

Control of common foot rot requires a combination of moisture conservation practices, especially tillage to maintain proper mulch and adequate weed control. Early planting and excessive nitrogen both promote heavy fall growth and use up moisture, so they should be avoided. Balanced fertility helps promote root regrowth. Rotating out of small grains would help reduce common foot rot but is seldom practical where this disease is most prevalent.

20. Common or dry land foot rot. Crown is dark brown on the outside and dirty brown internally.

21. Common or dry land foot rot caused by the fungus *Fusarium.*

Sharp Eyespot and Strawbreaker

Sharp eyespot is named for the conspicuous lesions which develop on the lower internode. These lesions are lens-shaped, up to an inch long, with brown borders and light tan centers **(Photo 22).** These lesions resemble strawbreaker which is rare in Kansas. Strawbreaker lesions are similar in size and shape except there is usually some black carbon-like material in the center of the lesion and the straw collapses, is brittle and breaks there **(Photo 23).**

22. Sharp eyespot lesion with tan center and brown border.

23. Strawbreaker lesion with black carbon-like center and weakened straw.

Affected tillers are usually scattered and ripen prematurely. Sharp eyespot causes little loss here and no controls have been developed **(Photos 24 and 25).**

24. Hessian fly can cause straw to break and lodge similar to foot rots. Look for the "flax seed" just above nodes. (courtesy of Ken Oppenlander)

25. Fuzzy white growths on roots in dry soil are normal root hairs and not fungus.

Root and Crown Rot Control Summary

1. Crop rotation or summer fallowing where feasible will give some control.
2. Maintain maximum vigor by planting good seed at the right time in a well prepared seedbed.
3. Apply phosphorus where soil tests indicate and avoid excessive nitrogen.
4. Adapted varieties do not show much difference in resistance.
5. Seed treatment fungicides give early protection but do not last long enough to prevent spring root and crown rots.

Take-All Root Rot

"Take-all" is the name given to a root and crown rot of wheat which occurs world wide. It is most severe in eastern and central Kansas, but also develops in irrigated continuous wheat in the west. It is not serious in summer fallowed fields.

Losses are quite erratic from year to year. Mild infections go unnoticed but probably cause some loss in many fields every year. Severe infections can cause total crop falure.

The most obvious symptoms are patches (rarely whole fields) of stunted, light colored, prematurely dead plants **(Photo 26).** These plants pull

26. Take-all root rot dead spots.

out of the soil easily and roots are rotted off **(Photo 27).** Roots and crown are blackened and, with wet soil, the lower stem internode turns shiny black **(Photo 28).** This black color is the surest diagnostic symptom to compare with other root and crown rots which cause a brown discoloration. However, with dry soil during heading, stem blackening may not develop, but the disease still can be causing significant loss. All tillers in infected plants are killed, usually without lodging. Grain is shriveled if it develops at all. Tillering is reduced. Cephalosporium stripe also causes whiteheads and stunting in a similar pattern but roots, crowns and lower stems are not rotted and black.

27. **Take-all root rot. Plants pull up easily because roots are destroyed.**

28. **Take-all root rot. Roots, crown and lower internode are black.**

With drouth stress in April, plants in take-all affected areas are stunted, have yellow lower leaves and may never develop heads. If spring moisture is adequate these affected patches are not obvious until after heading.

Take-all is caused by the fungus *Gaeumannomyces graminis (Ophiobolus graminis)* which probably was present on native grasses in Kansas prairie soils before cultivation and has found wheat a compatible host. It survives in the soil by colonizing undecayed, infected crowns. It competes poorly with other soil organisms for food and has no resistant resting stage. Infection occurs through roots in contact with old infected crop residue.

Take-all affects many cultivated and wild grasses. Wheat and barley are severely affected. Corn, sorghum, oats and rye are resistant and probably do not contribute to build-up in rotation with wheat. Soybeans can host the fungus but this has not been observed in Kansas. Cultivated smooth brome *(Bromus inermis)*, annual wild bromes such as Japanese brome *(B. japonicus)*, cheat *(B. secalinus)* and downy brome *(B. tectorum)* and wheat grasses *(Agropyron spp.)* are all very susceptible and are important in build-up and carry over of the take-all fungus in soil. Severe take-all often develops in wheat following brome grass or alfalfa infested with weedy annual bromes.

Other grasses reported infected with take-all include little barley *(Hordeum pusillum)*, foxtail barley *(H. jubatum)*, wild rye *(Elymus spp.)*, tall fescue *(Festuca elatior)* and foxtail or bristlegrass *(Setaria spp.)* but their importance as hosts in Kansas is unknown. All weedy grasses should be considered potential hosts.

The take-all fungus grows best at 50-60 degrees fahrenheit. Infection is favored by relatively wet soils but maximum losses occur under drouth stress between jointing and maturity. Adequate moisture at this time permits the plant to develop new roots or survive with remaining roots.

Neutral to slightly alkaline soils (pH 6.6-7.6) are most conducive for take-all development. It is usually suppressed in acid soils below pH 6.0, so liming acid soils can increase take-all. Loose, light textured soils favor take-all. Heavy, compacted, high organic soils suppress it.

Soil fertility affects take-all directly and indirectly. Adequate phosphorus and nitrogen permit the plant to develop a vigorous root system, replace rotted roots and survive. Other fertilizer effects, especially nitrogen, are extremely variable. Fall applications of NO_3-N have increased severity and reduced yields, but spring top dressing with nitrogen reduces take-all losses. Plowing under green or barnyard manure also reduces take-all.

Oregon researchers recommend ammonium chloride (NH_4 CL) and/or potassium chloride (KCL) fertilizer to reduce take-all; however, this has not been proved effective here.

Crop sequences, rotations and residue management affect take-all severity. Rotation out of wheat to a non-host crops for 1 or 2 years will reduce severity to a negligible level the first year back into wheat. Severity then may increase and be higher by the third or fourth year than occurred previously, then level off and gradually decline to a moderate severity under continuous wheat. This phenomenon, now known as "take-all decline", was noted in the 1930's and 1940's by Hurley Fellows in Kansas and has since been reported from many places in the world. The exact mechanism of this decline is unknown but it is apparently caused by antagonism of soil organisms which build up under continuous wheat. Researchers in the Pacific Northwest have isolated bacteria which suppress take-all when coated on seed. This is a promising control but requires further development and testing in Kansas before it can be recommended.

Take-all decline complicates crop rotation decisions. Maximum decline requires continuous cropping to wheat affected with take-all. But decline is unpredictable and sometimes develops slowly or not at all. Take-all losses will be minimal if wheat is planted not more than 2 consecutive years in rotation with 1 or more years of corn, sorghum, soybeans or alfalfa in which weedy grasses are controlled. However, rotating to another crop, then coming back into wheat 3 or more years consecutively may be disastrous since the decline factor disappears more quickly and builds up more slowly than the take-all fungus. So unless a grower follows a regular rotation out of wheat he may have lower long term loss in continuous wheat.

The effect of residue management on take-all varies, but it usually increases under reduced tillage which slows decay of crown tissue where the fungus survives.

Late planting sometimes reduces take-all severity.

No adapted varieties have significant resistance. Some experimental seed treatment chemicals show promise but are not yet approved.

Control Summary

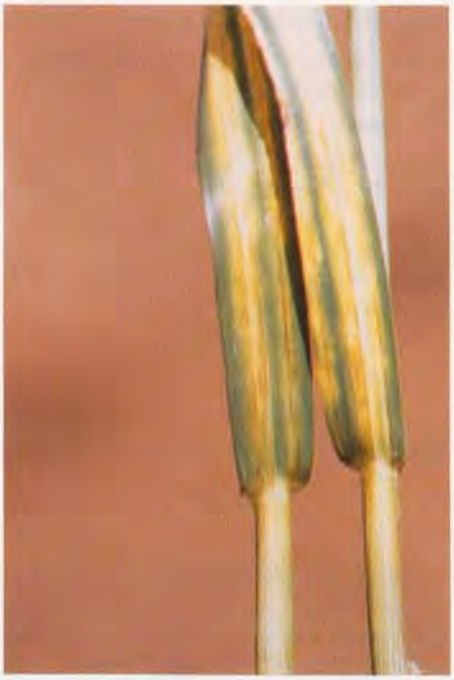
1. Summer fallow where feasible.
2. Rotate at least 1 year to corn, sorghum, oats, soybeans or alfalfa so that wheat or barley are not grown more than 2 years consecutively.
3. Don't plant wheat immediately following brome or alfalfa in which weedy grasses were prevalent.
4. Supply adequate fertility, especially phosphorus and potash where soil tests indicate a need. Split applications of nitrogen with part top dressed in spring may be better than total fall aplications.
5. Control weedy grasses, especially annual bromes, in crops preceding wheat.
6. Plant late—at least after the Hessian fly-free date.

Cephalosporium Stripe

Cephalosporium stripe was identified in Kansas in 1972 but has probably been here much longer. It is now recognized as one of our major diseases, causing an estimated average annual yields loss of 1.3 percent. Losses are erratic from year to year and field to field but often are more than 50 percent in severely affected fields. Losses are significant only in continuous cropped wheat and so are most serious in central Kansas.

Cephalosporium stripe is the only true vascular disease of wheat. The causal fungus plugs the vascular system and probably produces a toxin. The most prominent symptoms are broad (1/8" wide) yellow leaf stripes which continue the full length of the leaf blades, down the leaf sheaths and into the nodes **(Photo 29).** In the center of the yellow stripes the affected vascular bundle shows as a brown line **(Photo 30).** A longitudinal cut through the node will expose the brown vascular bundle which positively distinguishes this from any other disease **(Photo 31).** Tissue below nodes is sometimes discolored but other conditions can cause similar darkening so this is not a dependable symptom. Bottom leaves are affected first and stripe development progresses up leaf by leaf. Leaves with prominent stripes die within a few days and the leaf above then develops stripes. Maximum losses result when flag leaves become striped and die before grain fill. Diseased tillers head out but die prematurely several inches shorter than normal and are called "whiteheads".

29. Cephalosporium stripe. Leaves develop broad yellow stripes and die from the bottom of the plant up.



30. Cephalosporium stripe. Leaf stripes extend into the leaf sheath. Dark brown lines are the affected vascular bundles.

31. Cephalosporium stripe. Brown vascular bundle in a node.

Grain in these is shrivelled and light weight. These whiteheads intermingled with taller green tillers give the ripening field a ragged uneven appearance **(Photo 32).** Lodging sometimes follows but this is not consistent.

32. Cephalsporium stripe causes uneven height between diseased and healthy tillers.

Cephalosporium stripe is caused by the fungus *Cephalosporium gramineum*. Wheat is the preferred host but barley, rye and oats are also affected. Several grasses including downy brome *(Bromus tectorum)* are also hosts but it is not known whether they are important in Kansas epidemics.

The fungus survives only in undecayed stubble above ground or within the top 3 inches of soil but not in roots. Spores are produced on straw in very high numbers in the fall and winter. Freezing and thawing increases infection by breaking roots to permit spore entry and also causing roots to leak nutrients which increase spore germination. Spring seeded grains are not affected. Wet soil increases severity. Acid soils (around pH 5.0) are most conducive to Cephalosporium stripe. This may explain why it has become more severe in recent years where long time use of nitrogen fertilizers has lowered soil pH to this range.

After the fungus enters the vascular system it multiplies, spreads and severely impedes transport of water and nutrients. After the infected plant dies the fungus survives until the straw decays. It has no resistant forms for persisting in soil and so depends on having a susceptible host every year. This requirement limits Cephalosporium stripe to continues wheat production fields with heavy stubble carryover. Tillage which leaves stubble on or above the soil surface increases the disease.

Recognizing the disease and determining severity are necessary for control decisions. Losses can be estimated at early heading by counting the percent of tillers infected when flag leaf stripes are obvious. The percent yield loss will be about 1/2 of the percent tillers infected. (Example: 40 percent tillers infected will reduce yields about 20 percent). If losses are 5-10 percent in the current year, important decisions must be made that summer about cropping sequence and stubble management to prevent serious losses next year. Rotation to any crop but small grains will reduce severity to a low level in the following wheat crop. We never see significant Cephalosporium stripe following corn, sorghum, soybeans or alfalfa. Summer fallow is not an effective control in the Pacific Northwest but it works well in Kansas. Burning stubble gives good control if it is done with a hot fire to get a complete burn. But this is a poor practice because of increased erosion, water loss, nutrient and organic matter destruction, smoke pollution and fire danger. Plowing or disking under stubble reduces the disease severity especially if continued several years. This works only if there is enough summer moisture for fast stubble decay. The increased energy costs and erosion make this a poor choice for long time control but it may be necessary occasionally.

The effect of different stubble management on Cephalosporium stripe was studied 3 years at Hesston by Bill Bockus. **(Table 2)** Stubble treatments were: 1. Burned and disked after harvest, then disked as needed before seeding. 2. Moldboard plowed after harvest then disked as needed before seeding. 3. Disked after harvest and as needed before seeding. 4. Chopped with a rotary mower then disked as needed. 5. No till-direct drilled in stubble. **(Table 2)**

Late planting reduces Cephalosporium stripe some years but the disease control benefit may be negated by lower yields just from late planting. There is some difference in variety reaction but none are highly resistant.

Cephalosporium Stripe Control Summary

1. Rotate out of wheat at least one year to any crop except small grains.
2. Summer fallow where feasible.
3. Disk or plow under stubble in July where erosion is not a threat.
4. Burn stubble after harvest only if:
   a. Disease is severe in a field.
   b. Wheat is the only crop choice for next year.
   c. Summer fallow is not feasible.
5. Plant a week later than normal.

Diseases Affecting the Head or Grain

Smuts

Two smuts affect Kansas wheat—loose smut and bunt. Before the development of fungicide seed treatments and resistant varieties, they caused severe losses but in recent years have been effectively controlled. Relaxation of controls would permit losses to increase again. Smut reduces yield by replacing grain with smut spores. So losses are directly proportional to percent heads affected. If 10 percent of the heads are smutted, the yield will be reduced 10 percent. Bunt contaminated grain also is difficult to market, especially in international trade.

Other smuts, dwarf bunt, karnal bunt and flag smut, found elsewhere, have not been detected in Kansas.

Loose Smut

Loose smut is very obvious, especially as heads emerge from the boot. All parts of the head, except the rachis, (central stem) are converted to a mass of dusty, black spores **(Photo 33).** These spores are soon dislodged by wind and rain

Table 2.

Stubble Treatment	Percent Cephalosporium Stripe				Yield* Bu/A 3 Yr. Avg.
	1980	1981	1982	Avg.	
1. Burn	18	17	3	13	19
2. Plow	39	30	4	24	19
3. Disk	42	34	13	30	18
4. Chop	53	40	18	37	17
5. No-till	55	54	29	46	12

*Low yields were caused by other diseases and downy brome.

33. Loose smut soon after emergence from the boot.

(hence the name "loose smut") so that near maturity the smutted stems become less obvious. Wheat and barley are both susceptible.

Loose smut is caused by the fungus *Ustilago tritici*. It is seed-borne within the embryo. The smut fungus grows inside the germinating seedling, keeps pace with the developing plant, invades the developing head and replaces the flower parts with masses of smut spores. These spores mature just as normal plants flower. They are carried by wind to the opening flowers, germinate and penetrate into the developing embryo. Infected seed appears normal but if planted, will produce smutted heads the following year. Rainy, cool weather at flowering increases infection. Ordinarily wheat flowers remain open only a few minutes and this limits the infection period. Conditions which increase this open time will also increase loose smut infection. Hybrid wheat flowers remain open longer to increase cross pollination and this may consequently increase their loose smut susceptibility.

Since infected seed appears normal and infection is deep in the embryo, loose smut control is difficult. Prior to development of systemic fungicides, hot or cold water soaks were used. Systemic seed treatment fungicides became available about 1970, are highly effective and now universally used. Consult current publications for specific up-to-date recommendations.

Seed certification regulations limit the percent loose smut permitted. Seedsmen should routinely treat seed with systemic fungicide to control loose smut. However, with current levels of disease and variety resistance this is probably not necessary for grain production. No varieties grown widely in Kansas now are very susceptible, so incidence above 1 percent in a field is rare. If it begins to exceed this level, control in grain production might pay.

Bunt

Bunt (also called stinking smut or covered smut) is not obvious in the field until harvest. Affected plants are slightly stunted and heads slightly greener and more open than normal but the glumes remain intact. Smut balls develop in place of the grain and are wrinkled, dull gray to brown, and about the same size but rounder than normal kernels **(Photo 34).** During threshing these smut balls often break open releasing millions of black, dusty spores which adher to the surface of normal kernels **(Photo 35).** If bunt is heavy, a black

34. Bunted head with glumes removed to show bunt balls which have replaced the kernels.

35. Bunt. Smut balls are shorter than normal kernels, gray colored and break open releasing black spores.

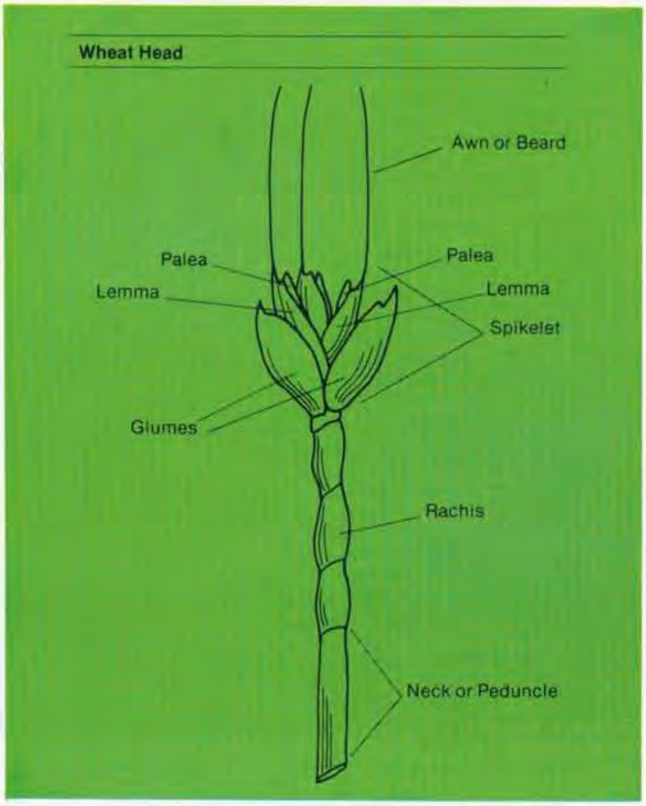
dust cloud may be released from the combine during threshing. A distinct fishy odor can often be detected (hence the name "stinking smut").

Losses from bunt in Kansas were as high as 10 percent in 1926 but have been very low in recent years. However, farmers who save their own seed several years and neglect fungicide seed treatment still report loss up to 50 percent.

When seed with bunt spores on the surface is planted the spores germinate along with seed, penetrate into the seedling and grow with the plant. The fungus invades the developing grain transforming it into smut balls. Cool fall temperatures increase seedling infection. Bunt spores do not survive in Kansas soils as they do some places so this makes control much easier. Spores on the seed surface are easily killed by fungicide seed treatments. Consult up-to-date literature for current, specific chemicals rates and efficacy.

Early planting will reduce bunt infection but will also increase some insects and several diseases and so is not recommended. None of the currently grown varieties are highly susceptible.

Is it necessary for a farmer to treat seed he is saving from his own planting every year? Probably not. But, if he does not treat, seed should be carefully monitored and treated when even traces of bunt are detected. Seed treatment fungicides are relatively cheap insurance. Seed certification regulations set strict limits on bunt and require fungicide treatment if even trace levels are detected.



Problems Resembling Smuts

Wheat heads and grain are sometimes discolored by several minor diseases or environmental conditions. It is important not to confuse these with smuts.

Ergot occasionally infects wheat and resembles bunt. But ergot bodies (sclerotia) are larger, protrude from the glumes and are hard throughout **(Photo 36).**

Black point results from infection of grain by several fungi during rainy weather. The superficial dark color is most prominent at the embryo end and along the crease **(Photo 37).** These fungi may reduce germination so it is best to avoid using black-pointed grain for seed or at least treat it with a fungicide.

36. Ergot sclerotia are larger than bunt balls, hard and protrude from the glumes.

37. Black point. Dark discoloration is most often on the germ end and along the crease.

Basal glume rot, caused by a bacterium, is rarely found in Kansas **(Photo 38).**

38. Basal glume rot.

Melanism or brown necrosis describes a darkening of heads of certain varieties under some environmental conditions **(Photo 39).** Barley

39. Melanism or brown necrosis.

yellow dwarf virus can cause this discoloration in some cultivars.

Sooty molds are saprophytic fungi which grow on heads, turning them a dirty gray **(Photo 40).** This is most likely following rainy weather, especially when plants die prematurely.

40. Sooty molds.

Scab

Scab (also called head blight) occurs in low levels most years but epidemics are erratic. It was severe in northeast Kansas in 1982. The last previous epidemic occurred in 1957. In fifty years of records scab was severe only seven years. It usually decreases in severity from northeast to southwest Kansas. Direct yield losses from scab are usually minor but can be 25-40 percent in severely diseased fields. Of greater concern is the ability of the causal fungi to produce mycotoxins, especially in cool, wet weather. At high enough levels these toxins may be harmful when consumed by humans or livestock, especially pigs.

Scab results from infection of individual spikelets at or soon after flowering when they are most susceptible. These spikelets are killed and the fungus then may girdle the rachis so that the head dies above that. A distinct salmon-pink ring of fungus develops at the base of the glumes **(Photo 41).** Occasionally, the head is covered with pink fungus. Early infected kernels are killed before filling. Later infection causes shrivelled, pink kernels or near normal sized chalky white kernels called "tombstones" **(Photo 42).** If scab-infected wheat is planted, seedling blight may ensue and reduce stands.

Scab is caused by one or more species of the fungus Fusarium, most commonly *Fusarium graminearum (Gibberella zeae).* These fungi can cause root, stalk and ear rots on wheat, corn, sorghum and many other crops. Old references noted an increase of scab in wheat following corn. Fusariums survive as saprophytes on decaying crop residue and develop long-lived resting spores.

41. Scab. A salmon-pink ring may develop at the base of the glumes.

42. Scab. Infected kernels are pink or chalky white.

This assures an ample source of infection whenever the right weather occurs.

Several days of continuous rain and temperatures about 70 degrees fahrenheit at and following flowering permit the most rapid buildup. Susceptibility decreases rapidly as grain develops.

Since scab is so sporadic, there has not been much research on control. Crop rotations in which wheat does not follow corn or sorghum may reduce scab. But this has not been proven with precise research and benefits of planting wheat after corn or sorghum will probably outweigh any scab increase over the long run. Stubble management will have little influence on scab development the following year. Fungicides applied at heading can reduce scab but would rarely pay. Differences in severity among varieties have been noted but this may reflect the coincidence of ideal weather at flowering rather than real resistance.

Leaf and Stem Diseases

Wheat leaf spots are caused by a diverse group of fungi and bacteria. These pathogens produce microscopic spores which are carried by wind and rain. Most require moisture for dispersal and infection so epidemics are very dependent on weather and, consequently, difficult to predict. Severe losses occur only when these diseases develop early enough to kill the top two leaves before grain fill. Cool weather not only favors the pathogens but slows wheat development and permits the diseases to do more damage.

Speckled Leaf Blotch

Speckled leaf blotch is our most common wheat disease. It can be found from November until maturity over most of the state. It kills some lower leaves in fall and again in spring but usually does not progress up the plant enough to cause serious loss. When conditions are right, and flag leaves are killed before grain fill, it causes serious losses. In 1983 it caused an estimated loss statewide of 5.8 percent. The eight year average is 1.8 percent.

The earliest symptoms are light green, irregularly shaped leaf spots. These quickly turn yellow then tan and tissue dies **(Photo 43).** The most distinctive signs are the brown to black specks visible without magnification, which develop quickly in the dying tissue **(Photo 44).** These are the spore bearing structures of the causal fungus *Septoria tritici*. Heavy infection kills the whole leaf. Glumes and awns are sometimes infected.

43. Speckled leaf blotch.

44. Speckled leaf blotch lesions showing the brown to black fungus fruiting bodies.

The fungus survives the summer on infected leaves and straw and in volunteer. Spores are extruded in sticky masses and are carried short distances by wind and splashing rain to newly emerging wheat. Spore production, germination and infection can occur over a wide temperature range from a low of 40 degrees fahrenheit up. The optimum is between 60 and 70 degrees fahrenheit. Free water and/or high humidity are necessary for most of the disease cycle. Leaves must be wet at least six hours for infection to develop but maximum infection follows 35 hours wetting and 48 hours more of high humidity. Severe epidemics follow weather with below normal temperatures and frequent rainfall in May and early June.

Fall infection, winter survival of the fungus in leaves, and an early start in the spring permits several cycles to develop, so great quantities of the fungus spores are often present when flag leaves emerge. Flag leaf infection depends on cool, moist weather occuring at that time. Clean tillage and crop rotations may reduce early infection and might be expected to reduce final severity but are not effective. Little difference in the amount of flag leaf infection is evident under different tillage practices and cropping sequences.

Short statured wheats are more vulnerable to speckled leaf blotch than tall wheats, because infection can spread more rapidly up plants with closely spaced leaves. Heavy fertilization increases susceptibility. Therefore, this disease will probably increase under present trends of intensive production.

There is some difference in susceptibility among varieties but none are highly resistant. Fungicides applied at heading can give good control and increase yields. But the erratic nature of the disease, necessity for aerial application and low wheat prices at present do not make this an economically attractive control.

Tan Spot

Tan spot is a very common leaf spot disease especially in continuous wheat. It is often confined to lower leaves and not conspicuous. Significant losses are erratic since they occur only when flag leaves are infected. The eight year average estimated annual loss to tan spot is 1.6 percent but it was as high as 3.5 percent in 1980. In many years tan spot is mixed with leaf rust and speckled leaf blotch and it becomes impossible to estimate how much loss each contributes. Experimental plots using fungicide have demonstrated 10-25 percent losses from severe tan spot.

Tan spots first show as small, 1/8 inch diameter lens or diamond shaped spots on leaves. A yellow border or halo soon develops around the tan center **(Photo 45)**. In young, actively growing leaves these spots remain about the same size. Then as leaves mature and begin to decline the spots expand to irregular shapes but the darker center where infection started is usually still obvious and the yellow halo becomes less prominent **(Photo 46)**. Severely affected leaves die prematurely and fungus spores are produced on

45. Tan spot lesions usually have a brown center and yellow halo.

46. Tan spot lesions expand to irregular shapes as leaves mature.

these dying leaves. Lower (older) leaves are infected first and with proper conditions infection progresses up to flag leaves **(Photo 47).**

47. Tan spot. Infection starts on lower leaves and progresses up the plant.

Tan spot is caused by the fungus *Pyrenophora trichostoma (Helminthosporium tritici-repentis).* This fungus also causes leaf spots on some other small grains and many native and introduced grasses. As wheat plants mature and die, the fungus invades the straw where it survives the summer. It is destroyed quickly if stubble is worked into the soil and decomposition begins. Two types of spores are produced, ascospores and conidia. In the fall black, raised, spore-bearing bodies develop on straw and ascospores mature inside **(Photo 48).** Mature ascospores are released in highest numbers in March and April following rain. These ascospores are not carried far by wind and rain so primary infection from this source is limited to fields with old straw on the soil surface. Conidia are dispersed longer distances by wind. They develop at temperatures between 50 and 75 degrees fahrenheit on old straw in the fall and may cause some infection then. Conidia are also produced on dying leaves from spring infection and probably account for severe epidemics which reach flag leaves. Moisture is required for both spore release and infection. The length of time which leaves must be wet for infection to occur varies from 12 hours for some susceptible varieties up to 24 hours or longer for more resistant ones. Leaf spots are visible within three to seven days after infection.

Control of tan spot is difficult. Crop rotation or clean tillage will prevent early spring infection from ascospores. But since conidia are produced on old straw, dying lower leaves and infected grasses and can be carried long distances by wind, there may be plenty of spores from outside the field to infect flag leaves. In years of severe tan spot the highest disease levels are in reduced tillage fields and areas of continuous wheat production. But lower levels can be found in clean tilled fields isolated from any old stubble source. Crop rotation and clean tillage then, may reduce the speed of disease build-up and final severity but are not reliable controls.

There is some difference in variety reaction but none now planted in Kansas are very resistant. Genes for resistance have been identified but await incorporation into adapted varieties.

Fungicide control may become feasible but the presently approved materials are not very effective in controlling this fungus. The present trend toward reduced tillage for soil, water and energy conservation will require genetic or chemical controls of tan spot for maximum yields.

48. Tan spot. Black structures in the straw on the soil surface develop and release ascospores.

Rusts

Wheat in Kansas is affected by three different rust diseases. Leaf rust is the most common and causes some loss every year. Stem rust has the potential for causing more serious loss than leaf rust in a severe year but occurs more sporadically. Stripe rust develops only in cool moist springs and is not found at all in most years.

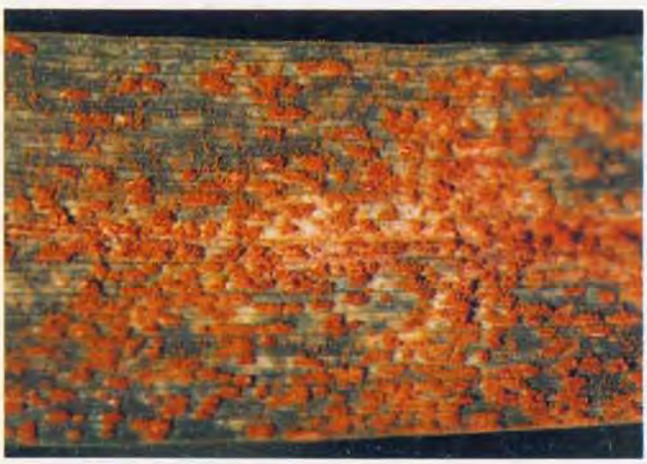
Rusts have developed hundreds of different races which can only be differentiated by their ability to infect a certain variety with matching genes for susceptibility. When a variety with genes for resistance to the prevalent rust races is widely planted, other races which are virulent on that variety increase. So a new variety may be resistant the first few years but later be susceptible because the rust population has changed. This makes breeding for resistance a never-ending battle to keep one jump ahead of the rusts.

Wheat rusts have alternate hosts on which other stages of the fungus life cycle develop. But none of these are necessary for rust survival nor important in Kansas epidemics.

Leaf Rust

Leaf rust causes some loss in Kansas every year. Early planted wheat may be heavily infected in the fall. But the major loss results from flag leaf infection before grain fill is complete. Leaf rust is most severe in eastern Kansas and generally decreases toward the west but this varies with weather. Losses also are extremely variable. Yields of highly susceptible varieties may be reduced 25 to 40 percent but usually losses are below 10 percent. Leaf rust is very democratic; it takes 1, 2 or 3 bushels an acre from wide areas. Estimated average annual loss from 1976-1983 was 1.5 percent but in 1983 it was 2.5 percent and was 15 percent as recently as 1974.

Leaf rust pustules (uredia) develop primarily on the upper surface of leaf blades. Pustules are round to oval, about 1/50 inch in diameter, light red, and filled with spores (urediospores) **(Photo 49).** These spores are easily dislodged by wind or



49. **Leaf rust. Pustules (uredia) and spores.**

rubbing and accumulate as red dust on hands, clothing and machinery. Rust increases water loss from leaves so they die prematurely.

Another stage (telia) develops on leaf blades and especially leaf sheaths near maturity. These are dark gray to black shiny smooth bumps and are not nearly as obvious as the uredia **(Photo 50).**

Leaf rust is caused by the fungus *Puccinia recondita* f. sp. *tritici*. Wheat is the main host but some isolates will also infect barley and goat grass. These other hosts play a minor role in rust

50. **Leaf rust. Telia on maturing leaves.**

epidemics. Urediospores are carried hundreds of miles by wind and survive to cause infection. This ability becomes very important in epidemic development. The leaf rust fungus survives in green wheat tissue. It winters in the south, moves north in spring and summer as wheat develops, then blows back south infecting volunteer wheat and early seeded wheat along the way. In Kansas it oversummers on volunteer wheat, infects fall seeded wheat and often survives the winter here when infected leaves remain green in protected places.

Early seeded wheat can be heavily infected with many leaves killed. This reduces forage yields for grazing, root growth and general vigor. However, conditions for severe fall rust are also optimum for good wheat establishment so we never see serious winter kill from rust.

Spring infection first show any time from mid-March to early May and can come either from spores produced on overwintering tissue or blown in from southern states. Unlike speckled leaf blotch or tan spot which start on lower leaves and gradually spread up the plant, leaf rust may develop first on upper leaves from wind deposited spores.

The optimum temperature for sporulation and infection is approximately 70 degrees fahrenheit and infection can occur within four hours if leaves are wet. New pustules erupt within seven to ten days. Rust development is slowed drastically with cooler temperatures or a shorter leaf wetting period. At 60 degrees fahrenheit a wetting period of 8-10 hours is necessary. Very wet conditions reduce spore dispersal. Epidemics rarely develop if daytime temperatures are below 60 degrees fahrenheit.

Since maximum leaf rust loss results when the upper two leaves are killed early, the faster an epidemic develops the greater the loss. Loss can be roughly predicted by matching severity on the flag leaf with growth stage. **(Table 3)**

Table 3

		% Severity on flag leaf				
		10%	25%	40%	65%	100%
	Milk	2	5	8	14	20
% yield	Soft Dough	1	3	4	7	10
loss at	Hard Dough	1	1	2	3	5

(Example: If leaf rust severity is 40% on the flag leaf at soft dough stage, a 4% yield loss is likely.)

This loss prediction assumes normal weather from the time severity and growth stage are estimated until maturity. Actual losses will be more with wet weather and less with dry weather and the error will be greatest on earlier predictions.

Leaf rust has been most effectively controlled by developing and planting resistant varieties. Research in Kansas began about 1901 and leaf rust resistance is a major component of high yielding varieties. The changing rust population requires a constant search for new genes and their incorporation into new varieties. When selecting resistant varieties be sure to use current information.

Planting late will reduce fall infection and possibly overwintering of the fungus but may have little effect on spring rust severity. Late planting delays maturity, permitting more time for spring epidemic development.

Fungicides are used in some states to control leaf rust and other diseases. The unpredictability of epidemics, the necessity for precise timing of applications, and economics make fungicide control unattractive now. (See discussion following leaf diseases).

Stem Rust

Stem rust is a potentially serious disease in Kansas but has been effectively controlled in recent years. It caused an estimated 38 percent loss in the U.S. in 1916 and 4 percent loss in Kansas in 1962. The most recent significant occurrence was a 0.5 percent loss in 1974. Early maturing, resistant varieties have reduced it to a minor disease. Stem rust is similar in many ways to leaf rust so descriptions will compare them. Pustules (uredia) develop on leaves, especially on leaf sheaths, necks, glumes and awns. These pustules are approximately 1/16 × 1/4 inch, much larger than leaf rust, dark red, considerably longer than wide, and are often evident on both upper and lower leaf surfaces. Pustules raise and break the epidermis making them rough to touch **(Photo 51).** As wheat matures pustules turn black (telial stage) but remain rough. Old timers called this "black rust."

51. Stem rust. Uredia on a leaf sheath.

Stem rust greatly increases water loss from leaves and stems so they die prematurely. Shrivelled, light weight grain results.

Stem rust is caused by the fungus *Puccinia graminis* f. sp. *tritici*. Wheat is the most common host but some isolates also attack barley, rye, oats, goat grass and wild barley. The sexual stage occurs on common barberry where new races may also develop. An extensive program to eradicate susceptible barberry in the north central stages is nearly complete. Barberries probably do not contribute to stem rust epidemics in Kansas.

Epidemic development is similar to leaf rust except that stem rust requires about 5 degrees warmer temperatures. Stem rust rarely builds up here in fall, and seldom, if ever, overwinters in Kansas. The first pustules are detected about three to four weeks later than leaf rust—late April to late May. This warmer temperature requirement and resulting later development often permits wheat to mature before the disease really gets started. Late maturing wheat is most vulnerable.

Effective control thus has been achieved by a combination of early maturity along with other types of resistance used in most of our popular varieties. Fungicide control would not be indicated with our present situation.

Stripe Rust

Stripe rust, sometimes called yellow rust, is important in higher elevations with cool moist climates. It usually occurs only in trace amounts in Kansas and is included here only for comparison. The causal fungus is *Puccinia striiformis*.

Pustules are light yellow and are arranged in distinct, straight sided stripes about 1/16″ wide and of irregular length **(Photo 52).** Heavily invaded leaves die prematurely.

52. Stripe rust.

Bacterial Leaf Blight

Bacterial leaf blight is common but usually unrecognized. Distinctive symptoms are transitory. Losses have not been documented but are probably comparable to other leaf diseases when flag leaf tissue is killed before grain fill is complete.

The first symptoms are tiny water-soaked leaf spots. These quickly enlarge and coalesce to form irregularly shaped gray-green areas. These stages may last only a few hours so are easily missed. Affected areas quickly fade to off-white or tan and do not enlarge further **(Photo 53).** This dead tissue is often invaded by saprophytes so within a few days dead tissue becomes a dirty gray and all distinctive symptoms are obscured. Symptoms are most prominent on flag leaves. Leaves often bend or roll at the point of most extensive invasion.

53. Bacterial leaf blight.

Table 4. Rusts Compared

	LEAF RUST	STEM RUST	STRIPE RUST
Pustule color	light red	dark red	yellow
Pustule arrangement	single	single	close together in stripes
Pustule shape	round to oval	oblong	round
Pustule size	medium	largest	smallest
Temperature requirements	60-75°F	65-80°F	55-70°F
Prevalence	every year	trace	rare
When first appears—Fall	4-5 leaf stage	4-5 leaf stage	never
Spring	Mar 15-May 1	Apr 15-May 30	erratic
Where pustules develop	leaf blades	leaves, stems, heads	leaves, heads

Bacterial leaf blight is caused by the bacterium *Pseudomonas syringae*. It is very common and causes various diseases on many different plants.

The disease strikes quickly, usually at about boot stage and over wide geographic areas. Then it stops just as suddenly. The exact conditions for epidemic development are unknown. Moisture is necessary for infection and symptoms develop a day or two after a storm. Wind-driven rain is important is dispersing the pathogen and possibly in making the wheat plant more susceptible.

No controls for bacterial leaf blight have been developed. Incidence and severity to not appear to be related to crop sequences or residue management. Varieties derived from Scout are most severely diseased but others with diverse backgrounds are also susceptible.

The bacterium causing black chaff occasionally infects leaves, causing yellow to brown lesions **(Photo 54).**

54. Black chaff leaf lesions.

Powdery Mildew

Powdery mildew is a common and obvious wheat disease. But losses from it are probably not as extensive as severity of symptoms indicate. Losses are difficult to assess but have been estimated below 1% state-wide in recent years. Severity appears to be increasing, probably due to more susceptible varieties and increased nitrogen fertilization. It is most severe in southeast Kansas and decreases toward the northwest.

Powdery mildew is easy to identify. The first symptoms are light green flecks on the leaves. Very soon white cottony tufts of fungus develop. These tufts spread over the leaf and turn to a dirty gray mat **(Photos 55 and 56).** Dusty white spores are produced in great numbers and cover leaves and soil. Affected leaves turn yellow and die. Under some conditions, distinctive spots of dark green persist in yellowing leaves. The disease increases respiration and water loss and decreases chlorophyll; consequently, photosynthesis declines.

55. Powdery mildew.

56. Powdery mildew.

Powdery mildew is caused by the fungus *Erysiphe graminis* f. sp. *tritici*. Closely related forms cause powdery mildew on many grasses. These forms are closely adapted to their specific hosts so it is doubtful if there is much cross-infection from other crops and grasses to wheat.

The fungus survivies on dead wheat plants and within infected leaves. Fall infection is reported some places but it is seldom seen in Kansas. The first appearance is usually in spring at about jointing, but occasionally earlier. Lower leaves are infected first and it progresses up the plant leaf by leaf as long as conditions for spore production and infection persist.

Powdery mildew thrives in cool damp conditions. Optimum temperature is 68 degrees fahrenheit with relative humidity near 100%. But heavy rain actually reduces it. Spores survive about 2 days at 70°F and considerably longer at cooler temperatures. This permits some long distance dispersal by wind. Some infection can occur as low as 40°F. Epidemic development stops abruptly about 75-80°F. This often prevents severe loss since infection of the top two leaves is necessary for significant yield effect and it is usually warm enough by the time these leaves emerge to prevent extensive infection. Powdery mildew is most prevalent in low lying, thickly planted fields protected from wind and heavily fertilized with nitrogen. Only slight differences in fertility or plant density can cause drastic differences in disease severity within a field.

Since high fertility and thick planting are necessary for maximum yields, cutting back on these to control powdery mildew is not practical. There is no clear indication that cropping sequences or residue management affect powdery mildew severity.

Resistant varieties have been developed in areas where powdery mildew is more serious. Our present varieties range from moderately resistant to very susceptible with none highly resistant.

Fungicides highly effective against powdery mildew have been developed. But the erratic nature of our epidemics and low wheat prices presently make regular use of funigicides uneconomical.

We are left with no practical, economical controls.

Fungicide Control of Leaf Diseases

Fungicides are used regularly for leaf disease control in some places where these diseases are consistently severe. In Kansas experiments, fungicides occasionally give economically acceptable control. But it would not pay often enough for routine use and predicting when it would pay is not possible. In a six year study (1975-1981) at Clay Center, Nebraska, two applications of mancozeb increased yields an average of only 1.3 bu/A. It will first be economically feasible on special high value, high yield fields such as for seed increase. But practical use of fungicides for grain production in Kansas awaits some combination of one or more of the following developments.

1. Higher wheat prices.
2. More effective fungicides.
3. Cheaper fungicides relative to wheat prices.
4. More flexible application methods and timing so ground equipment can be used before jointing (possibly with nitrogen top dressing or spring herbicide) and the fungicide must last long enough to protect flag leaves to maturity.

Miscellaneous Problems

57. Freeze may kill only part of the head.

58. Freeze kills tender flowers and they turn yellow to brown within a few days.

59. Hail or strong wind can bend the leaf sheath and trap beards so that the head pushes out sideways.

60. Hail broke the rachis killing part of the head.

61. Color banding is caused by frost when tender new leaves are emerging.

62. Crazy-top occurs in low wet areas and causes yellow stunted plants with thick leathery leaves.

63. Crazy-top. Some infected plants develop coarse distorted heads.

Definitions

Bacteria—Microscopic, single celled plants which reproduce by dividing.

Disease—Any alteration in a plant that interferes with its normal structure, function or economic value.

Fungus (plural—fungi)—Plants with thread-like vegetative growth which do not have chlorophyll.

Host—The living plant in which a parasite lives.

Incubation period—Time from infection to symptom development.

Infection—The establishment of a parasite within a host.

Inoculate—To transfer inoculum to a host under conditions for infection.

Inoculum—That portion of a pathogen that can be transferred to a host and cause infection.

Mosaic—A leaf pattern of light and dark green or yellow on leaves often caused by viruses.

Necrosis—Death. Necrotic means dead and usually discolored.

Parasite—An organism that subsists on a living host, usually causing disease.

Pathogen—Something causing disease.

Pathogenic—Ability to cause disease.

Resistance—The ability to remain disease-free to some degree—a relative term.

Saprophyte—An organism which subsists on dead organic matter.

Spore—The reproductive unit or "seed" of a fungus.

Sporulation—Producing spores.

Susceptible—Tendency to become diseased. Inability to resist disease.

Symptom—Evidence of disease.

Vascular system—Ducts through which water, minerals and food materials move through the plant.

Vector—An organism which transmits a pathogen.

Virus—Submicroscopic particle which reproduces only inside host cells and causes disease.

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