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CROP DISEASES IN 1996 - REVIEW AND UPDATE

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Diseases cause losses to our crops every year, and 1996 was no exception. Many of the disease problems this year were associated with the wet spring. Cooler-than-normal summer temperatures also influenced diseases, some positively, some negatively. In corn, the most troubling disease problems were seedling diseases and early-season crown rot. Some foliar diseases appeared late in the season, causing losses in some fields. Gray leaf spot did not develop into a widespread problem as it did in 1994 and 1995, but this disease remains an important issue. Common rust and Stewart’s wilt reached damaging levels in some fields. There were more notable disease problems in soybeans in 1996. Rhizoctonia root rot killed seedlings and stunted older plants in many fields. White mold was the most serious problem of the year, appearing most severely in the northern half of the state. Fields with soybean cyst nematode commonly displayed obvious symptoms of stunting and yellowing, indicating that losses to this pest were higher than usual. Wheat and barley growers in Iowa suffered from two diseases as a result of the spring weather. Scab occurred on wheat throughout southern Iowa and on barley in NE Iowa. Ergot, a somewhat rare disease in Iowa, caused serious problems in NE Iowa barley.

This paper will examine the occurrence of these diseases in 1996 in Iowa and, where appropriate, provide new information related to their biology and management. Diseases covered in detail elsewhere in the proceedings will not be emphasized here.

Corn Diseases

Seedling Diseases and Crown Rot
Seedling diseases caused stand reduction and replanting costs in many Iowa corn fields in 1996. Although much of the acreage was planted in a timely manner, emergence was very slow due to persistent moisture and cool soils. This is illustrated by the crop report of May 13, which showed that 83% of the acreage had been planted, but only 6% had emerged. Corn seed that remains in the ground for a long time before emerging is very vulnerable to attack by soilborne pathogens.

Although seedling diseases did occur fairly extensively, it appeared that ultimate corn plant emergence was better than might be expected considering the long delay. This was an observation shared by a number of Extension Specialists, producers, and agricultural industry professionals. There can be much speculation about the reason for this phenomenon, but I am not aware of specific data that might explain it. We did not conduct seed treatment field trials in 1996. One possible explanation is the widespread use of a Captan/Apron combination of seed treatment fungicides. A shift toward this combination has been occurring in the seed industry, and 1996 was the first year that the majority of corn seed was treated with Captan/Apron instead of Captan alone. Apron (active ingredient: metalaxyl) is very effective against Pythium, which is among the most important seedling pathogens of corn. In previous seed treatment trials, we have observed only slightly better performance by Captan/Apron compared to Captan alone.

In 1996, some commercially sold corn seed was treated with a new fungicide, Maxim, in combination with Apron. Maxim has a similar spectrum of activity to Captan, but it is used at a much lower rate. In some cases, Maxim/Apron has been shown to be superior to Captan/Apron. In limited seed treatment
trials in Ames in 1995, we did not observe significant differences between these two treatments. It is anticipated that a greater proportion of corn seed sold in 1997 will be treated with Maxim/Apron.

Management recommendations for corn seedling diseases in 1997 remain the same as in previous years: plant high quality fungicide-treated seed in a well-prepared seedbed when soil temperature is greater than 50° F and soil moisture is adequate but not excessive. As with most diseases, crop rotation reduces the risk of severe disease. Reducing the amount of surface residue also decreases the risk of seedling disease. If a choice of seed treatment chemicals is available, there may be some advantage to planting seed treated with Capitan/Apron or Maxim/Apron instead of Capitan alone. Planter-box treatments usually contain Capitan plus an insecticide. Under most conditions, you will not see an added benefit to additional Capitan on already-treated corn seed. The primary benefit of planter-box treatments is insect control.

Some fungal pathogens that cause seedling blight can continue to plague corn plants as they develop and mature. *Fusarium* species can invade the crown of the plant (the base of the stalk), where they may do no damage or they may stunt or kill the plant by rotting the growing point and/or the root/stalk interface. Development of these infections is usually a result of an interaction between some environmental stress and the *Fusarium* infection. Most crown rot problems occur in corn-on-corn fields. Crown infections can later lead to *Fusarium* stalk rot, although most stalk rot infections are initiated later in the season. Managing crown rot depends on following seedling disease management recommendations, especially in terms of crop rotation. Cultivation may have some value in helping plants recover from crown rot infections. Hybrids differ in their tendency to suffer from crown rot, but there are little or no data available.

**Gray Leaf Spot**
Following the 1995 outbreak of gray leaf spot, there were high levels of inoculum in many areas of southern Iowa. The disease appeared fairly early in 1996; it was present in corn-on-corn fields around Ames as early as July 8. Given favorable weather conditions, the disease might have been very widespread and severe in 1996. However, the disease did not develop rapidly until mid-September, and this delayed development substantially reduced the impact of the disease. The result was that some fields had severe disease, but the problems were not nearly as widespread as in 1995. Weather in 1996 was somewhat cooler than optimum for gray leaf spot, especially at night when leaf wetness is present. The cooler weather partially explains reduced disease severity, but we have much to learn about predicting this disease. Temperature and precipitation comparisons for SE Iowa showed that 1996 was very similar to 1994, but gray leaf spot was much more severe in 1994. The crop was considerably later in 1996, and this also influences disease development. A key factor in gray leaf spot development is humidity, which is not reported for most of the state’s weather stations.

We conducted foliar fungicide trials for gray leaf spot control in hybrid corn for the second year in 1996. Reduced severity of the disease compared to 1995 was evident in the disease ratings. At Crawfordsville, diseased leaf area for the ear leaf reached only about 15% in non-sprayed controls by September 20. In 1995, the non-sprayed control plants had up to 45% diseased leaf area by September 5. Even with reduced disease severity in 1996, we did observe significant increases in yield of susceptible hybrids if gray leaf spot was controlled by a fungicide. In one field, yields of non-sprayed controls averaged 152 bu/acre, while those receiving a single spray of Tilt at tasseling averaged 169 bu/acre. However, this does not necessarily mean a 17 bu increase, because the Least Significant Difference for the treatments in this experiment was 15 bu/acre. Similar results were obtained in another field with a susceptible hybrid. However, when fungicides were applied to a moderately resistant hybrid, there were no significant differences in yield. This result demonstrates the value of hybrid selection for reducing this disease.
In 1996, the Iowa State University Corn Yield Test collaborated with the Plant Pathology Department to evaluate corn hybrids for gray leaf spot reactions. All hybrids entered in ISU Corn Yield Test Districts 5, 6, and 7 were evaluated for gray leaf spot severity in early September. Each hybrid was evaluated at two or three sites. Hybrids were rated at sites in Adams, Boone, Cedar, Henry, Mahaska, Madison, and Poweshiek Counties. Results of these evaluations will be published as a separate supplement to the ISU Corn Yield Test Results.

**Stewart’s wilt**

Winter weather is often used as a predictor of Stewart’s wilt severity, which depends on survival of the disease’s vector, the corn flea beetle (*Chaetocnema pulicaria*). This insect feeds on the leaves of corn, small grains, and grasses. *Erwinia stewartii*, the bacterium that causes Stewart’s wilt, overwinters in the gut of the adult flea beetles, and when the beetles survive the winter in greater numbers, the incidence and severity of Stewart’s disease are higher. Occurrence of Stewart’s disease consequently can be predicted by considering the mean temperatures for December, January, and February. If the sum of the mean temperatures for these three months exceeds 90, severe Stewart’s disease can be expected in susceptible genotypes. If the sum is between 80 and 90, the risk is moderate to severe, and if the sum is 70-80, the risk is moderate. Last winter was sufficiently cold that we predicted little or no Stewart’s wilt in Iowa in 1996. The temperature sum for Iowa last winter was less than 80 for the whole state, except the extreme SE corner, where the sum reached 82. Nevertheless, flea beetle populations were high in some fields. As the season progressed, leaf damage from Stewart’s wilt was evident across the southern 1/3 of the state. In some fields, up to 50% of the leaf tissue was killed prior to denting. At this level of disease, some yield loss probably occurred.

This is the second consecutive year in which Stewart’s wilt reached damaging levels in some fields. Last year, it was a more significant factor because the previous winter was warmer. In addition to causing losses in yield, in 1995 it occurred in some seed production fields and interfered with the export of seed. In 1996, I did not receive any reports of severe outbreaks in seed corn.

Stewart’s wilt control is usually not needed in field corn. Most hybrids have sufficient partial resistance that losses do not occur in most years. If early-season flea beetle populations are high enough 5 or more per plant prior to stage V5 for field corn and 2 or more per plant for seed corn), a foliar insecticide application is warranted (Rice, 1995). In addition, a systemic insecticidal seed treatment (Gaucho) has been shown to reduce flea beetle feeding and Stewart’s wilt in young corn plants (Munkvold et al., 1996). The cost of this product will probably prevent its use on field corn, but it may be a practical alternative for seed corn and sweet corn.

**Soybean Diseases**

**Rhizoctonia root rot**

Rhizoctonia root rot is caused by the fungus *Rhizoctonia solani*. It can attack plants at any growth stage, but most of the damage is done to seedlings. The first noticeable symptom is wilting and death of plants. Often there is a reddish-brown lesion at the soil line. On older plants, the lesion may be absent, but the entire root system is decayed and reddish-brown.

In 1996, *Rhizoctonia* was widespread on seedlings. This can be partially explained by the cool wet spring, but the predominance of *Rhizoctonia* over *Pythium* and *Phytophthora* this year is not fully understood. *Rhizoctonia* is more common in lighter soils, compared to the heavier soils that favor *Pythium* and *Phytophthora*.

*R. solani* differs from most fungi because it does not produce spores for dissemination and infection.
Plants are infected when the roots contact the fungus directly in the soil. The fungus develops a network of weblike strands of mycelium, drawing nourishment from crop residue. The structural integrity of the mycelium is important for the fungus to be able to infect plants. Because of this unique characteristic, tillage can reduce the impact of the disease by disrupting the mycelial structure.

Fungicidal seed treatment is another way to control Rhizoctonia on seedlings. Some commercial seed treatments, such as Agrosol, Rival, and Vitavax are more effective against Rhizoctonia (Lipps and Labarge, 1994).

White mold and Soybean Cyst Nematode
Both of these diseases were more severe than usual in 1996. White mold, favored by cool weather during flowering, was severe in Iowa in 1992, 1994, and 1996, especially in northern Iowa. Soybean cyst nematode damage is typically more severe in dry years, and some areas of the state had prolonged dry periods in 1996, resulting in substantial cyst nematode symptom expression. Because these diseases are covered elsewhere in the proceedings, I will not explore them further here.

Small Grain Diseases

Scab
Wheat scab (pathogen: Gibberella zeae) caused extensive damage to winter wheat in southeast and south-central Iowa and in barley in northeast Iowa in 1996. It is caused by the same fungus that infects corn stalks and ears. Cool, wet conditions during flowering are very favorable for the development of scab. Symptoms of scab are premature death of wheat or barley heads. All or part of the affected heads appear bleached at first, sometimes with pink or salmon-colored fungal mycelium visible. Often the heads become colonized by saprophytic fungi such as Cladosporium and Alternaria, turning the heads black. This is known as sooty head mold. Any problem that causes premature plant death can result in sooty head mold, so these blackened heads are not a reliable indication of scab. “Scabby” heads may not contain any grain, or the grain will be shriveled and very lightweight. Both yield and quality can be drastically reduced.

Another serious consequence of scab infection is the production of mycotoxins by the fungus. G. zeae can produce both deoxynivalenol (DON or vomitoxin) and zearalenone. These compounds are toxic to humans and animals. DON, which causes feed refusal by livestock, is usually much more common than zearalenone in scabby wheat. If DON concentrations are too high, wheat cannot be used for human consumption. Recent outbreaks of scab in Minnesota and North Dakota have been devastating to the wheat industry, and there have been reports of animal health problems caused by scabby wheat used for pet food. In 1996, feed refusal problems were reported in swine operations where barley was being fed.

Scab is more severe when small grains are planted in heavy corn residue, so the risk of scab can be reduced by rotating with a crop other than corn. Tillage of the corn residue will help to reduce inoculum levels. Wheat and barley varieties vary in susceptibility to scab, and plant breeders at several universities and seed companies are working toward the development of more resistant varieties. Foliar fungicides have not consistently controlled scab, although seed treatment fungicides can reduce its effect on germination.

Ergot
This disease is caused by the fungus Claviceps purpurea. It can infect any of the small grain crops, but rye is by far the most susceptible. It also can infect many forage grasses. The disease is fairly rare in Iowa because rye is not widely grown. In 1996, many fields of barley grown in northeast Iowa were infected by this fungus. It is estimated that 200,000 bu of barley were affected. Ergot reduces grain
yields, but the most important effect is the production of toxic alkaloids.

Symptoms are obvious on rye but more subtle on barley and wheat. As the head develops, the grain is replaced by a dark fungal structure known as a sclerotium. On rye, the sclerotia protrude out from the head and may be an inch long. On wheat and barley, the sclerotia are usually less than ½ inch long and do not obviously protrude. These sclerotia develop as a result of infection of the florets by airborne spores. Infection takes place during flowering and soon a sticky liquid appears on the surface of infected florets. Later the sclerotia develop. The sclerotia drop to the ground during harvest or are picked up by the combine. In the spring, sclerotia on the surface germinate and produce another structure, called a stroma, which in turn produces the spores. The spores are spread by wind, rain, and insects. After the initial infections, more spores are produced on the heads and the disease spreads as long as there are open florets available. Cool, wet weather promotes sclerotial germination and infection by spores, but also prolongs the susceptible flowering period. Fields that flower unevenly, produce abundant tillers, or have a high percentage of sterile flowers will suffer more infection.

Livestock that eat the sclerotia mixed with feed can display a number of symptoms related to constriction of blood vessels; convulsions and death also can occur. The reduction in circulation can result in lameness and gangrene. Other effects include loss of appetite and decreased milk production. In Iowa this year, at least 700-900 cattle were reportedly affected by ergot poisoning. Only a few deaths were reported. Symptoms can occur in dairy cows if they consume grain that is greater than 0.1% ergot by weight. One sample from Iowa this year was reported to contain 4.5% ergot, and a number of samples from problem herds tested in the range of 0.1-0.4% ergot.

Because this disease is rare on small grains in Iowa, no control is usually warranted. However, this year, it may be necessary to take some precautions. Fields that had ergot problems in 1996 should not be planted to a small grain crop next year. Soil should be tilled before planting another small grain crop. If sclerotia are buried they do not survive more than a year, so tillage can reduce the chance of infection in nearby fields as well. Controlling grass weeds and cutting grass hay prior to flowering will reduce the chances of ergot infection. Contaminated grain should not be used for seed. Commercial seed cleaning can remove nearly all the sclerotia.

