Diseases of forage grasses in humid temperate zones

S. W. BRAVERMAN, F. L. LUKEZIC, K. E. ZEIDERS, AND J. B. WILSON
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COVER PHOTOGRAPH: Red thread disease, Corticium fuciforme, on perennial ryegrass. Courtesy of C. J. O'Rourke.
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The authors wish to express their appreciation to the scientists and others who have provided photographs or otherwise contributed to the preparation of this publication. Dr. T. Tominaga, Sayama-Chi, Japan; Dr. T. Egli, CIBA-Geigy, Ltd., Basel, Switzerland; and Dr. D. Schmidt, Swiss Federal Research Station for Agronomy, Nyon, Switzerland provided photographs of bacterial diseases. Dr. C. J. O’Rourke, The Agricultural Institute, Dublin, Ireland; and Dr. P. Weibull, Landskrona, Sweden, provided photographs of fungus diseases. Photographs of the virus diseases are courtesy of Dr. P. L. Catherall, Welsh Plant Breeding Station, Aberystwyth, Dyfed County, Wales. Mrs. Teri-Anne Jordan assisted in preparation of the manuscript for the authors and editors.

Research reported in this publication is supported by funds from the Pennsylvania State Legislature, the United States Congress, and other government and private sources. Published by The Pennsylvania State Agricultural Experiment Station in cooperation with the U.S. Regional Pasture Research Laboratory, University Park, Pennsylvania, and agricultural experiment stations of the Northeast Region. Authorized for publication 21 November, 1985.

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FOREWORD

Continuing conversion of agricultural lands to non-agricultural use dictates the need for continued improvement in the quality and production of forage grasses in grassland agriculture. The identification of forage grass diseases as they occur in the field is of paramount importance prior to the recommendation of adequate control measures or to the development of resistant cultivars through forage grass breeding programs. It is also important to develop improved techniques for expediting disease resistance and to facilitate the incorporation of disease resistant germplasm into forage grasses. The value of these improvement programs cannot be overestimated.

This bulletin recognizes the fiftieth anniversary of the U.S. Regional Pasture Research Laboratory. The Laboratory is located on the campus of The Pennsylvania State University, University Park. The Pasture Laboratory staff works in close cooperation with Pennsylvania Agricultural Experiment Station workers on forage problems of mutual interest.

Charles R. Krueger
Associate Dean for Research and Associate Director
of the Pennsylvania Agricultural Experiment Station.
OVERVIEW
Forage grasses are utilized as pasture and hay and are a basic foundation of United States agriculture. These grasses support the production of beef and dairy cattle, each an annual multibillion dollar enterprise. The 1979 U.S. Department of Agriculture’s Agricultural Statistics (1979), indicated that there were 110.9 million head of cattle worth $44.7 billion feeding on over 100 million acres of pastureland in the United States. Recent developments in grassland agriculture have focused on improved management practices which include planting warm-season forage grass species, increasing yields, quality and nutrient content of the grasses, disease resistance, and other mechanisms of disease control.

The warm-season grasses include big bluestem (Andropogon gerardi Vitman), little bluestem (Schizachrium scoparium (Michx.) Nash), indiangrass (Sorghastrum nutans (L.) Nash) and switchgrass (Panicum virgatum L.). These major native grasses of North America are distributed throughout the central and, to a lesser degree, the eastern United States. They are characterized by drought tolerance and relatively short growing season (mid-May to mid-September). Cool-season grasses grow from early April to November. The peak-production period for warm-season grasses is the hot, often dry, months of July and August, a time when cool-season grasses produce little forage. For this reason, they are important supplements to the cool-season species for grazing and hay production on many farms.

Forage grasses are subject to a multitude of leaf, stem, floral, and root diseases. The leaf diseases include rusts, smuts, and leafspots, and cause the major reductions in yields, as harvested forage consists primarily of leaf material. Grass diseases may limit effective utilization of the grass species and affect herbage quality, digestibility, tillering, and root growth. Perennial grasses are usually prone to one or more diseases that, over an extended period, may weaken the plant. A weakened plant is less likely to survive winter-kill.

Resistance in certain cultivars offers an efficient approach to control of several of the more serious fungal, bacterial, and viral-caused diseases. Maintaining resistance in a cultivar may require continuous effort because disease resistance often loses effectiveness. Loss of effective resistance may be due to the acquisition of new virulent genes in the pathogen. It becomes imperative to seek new and better sources of disease resistance from as broad a germplasm base as possible. Resistance to many of the diseases of the Gramineae has been reviewed by Braverman (1967, 1986).

This bulletin describes control measures for common disease incitants on selected forage-, range-, and pasture-type grasses. The majority of the pathogens discussed are common in the United States. However, a few bacteria and viruses not known to occur in the United States are included because of their destructive potential, should the causal agents become established. Host susceptibility, favorable temperature and moisture, and suitable vectors for virus transmission are the major determinants of incidence, rapidity, and severity of disease development. Resistant cultivars offer a prime means of control, and are most effective when used with good farm management and agricultural practices. Tillage, sanitation, crop rotation, fertilization, and proper time of harvesting all influence development of diseases on farm crops.

Incitants discussed here are divided into the biotic agents—bacterial, fungal pathogens, and viral agents, and then selected abiotic agents, including environmental factors and nutritional disorders. Publications referenced for each disease are listed with the discussion; a complete bibliography is presented on page 38.

This publication has been written for growers, teachers, and researchers concerned with grassland production. The biotic and abiotic diseases which affect temperate forage grasses are described. Control measures are listed, if known. A glossary defines selected technical terminology.

Nematodes damage fine turf. However, to the authors’ knowledge there are no reports of such damage to range and pasture grasses.

Initially, the common name for each host species, followed by the Latin binomial, is used. Thereafter, only the common name is listed. Binomials not accompanied by an appropriate common name indicates none was identified.

Diseases are listed according to hosts on page 43.

Literature:
DISEASES INCITED BY BACTERIA

Symptoms caused by bacteria may be similar to those incited by fungi or viruses. However, symptoms characteristic of bacterial infections include watersoaked lesions on foliage in the early stage of disease development. When observed in transmitted light, such lesions appear to be greasy. Under suitable conditions, bacterial exudates may occur on the diseased plant parts (Figure 1).

Plant pathogenic bacteria are unicellular rods from 1 to 3 μm in length. They do not have well-defined nuclei or nuclear membranes. They do not form spores, but are covered with a slime which aids in survival under adverse conditions. Bacterial pathogens are most commonly spread by plant debris, water, and insects. Bacteria can persist on seeds, infected plants, plant debris, and soil. Free moisture is usually required for infection, and penetration of host tissue is through wounds or natural openings. Bacterial pathogens invade the vascular system or intercellular spaces in host tissue. Necrosis is commonly due to the effect of toxins or enzymes produced by the pathogen.

At present, the control measures on field crops are sanitation and use of pathogen-free seed. Little is known about resistance in varieties of forage grasses. Bactericidal sprays have been used effectively in control of some vegetable and ornamental crop diseases, but are too expensive for forage crop disease control.

Bacterial taxonomy, always in a state of flux, has undergone a large change in recent years. The proliferation of new techniques for determining genetic and phenotypic relationships along with the establishment of regional repositories for type cultures makes it convenient for scientists to compare their cultures with the original type culture. Such comparisons have shown that a large number of old species are phenotypically and genetically related, but have different host ranges. Thus, it is possible to group a number of closely related species under one species and create a new designation, “pathovar,” as a subgroup based on host range. Advantages of this new system become apparent upon examination of the recent literature on pseudomonads and xanthomonads. In the former, the species “syringae” includes all phytopathogenic pseudomonads that are oxidase negative and produce green fluorescent pigment. In the genus Xanthomonas, the species “campestris” consists of 123 pathovars grouped together because of their characteristic growth on a specific medium.

Study of this group of pathogens is not sufficiently complete to determine with certainty the existence of pathovars within the bacterial pathogens of forage grasses. Where the literature is clear that such a pathovar exists, we have used that designation. We have relied heavily on the work of others for descriptions of symptoms of these diseases on the small grains discussed herein.

Suggested reading:

Yellow Slime Disease of Orchardgrass (Dactylis glomerata L.) caused by Corynebacterium rathayi (Smith 1913) Dowson 942. Figure 2

Yellow slime disease, or bacteriosis, of orchardgrass occurs sporadically in Britain where it is introduced on imported seed. In Denmark and northern Germany, the disease is well established and can cause serious losses to seed growers. The bacteria also occur in the United States.

Symptoms: A yellow slime consisting of bacterial cells appears on the surface of the upper plant parts, especially the inflorescences. A dwarfing and distortion, through incomplete elongation of the upper internodes, is also evident. Because of the sticky slime, the stalks often form knee-shaped bendings due to elongation of the under part of the stuck stem, and the inflorescence may push out laterally. Infected parts dry prematurely. Vessels and parenchyma are invaded by bacteria (Bradbury, 1973a).

Very similar diseases have been reported on other grasses as caused by other species of Corynebacterium.

Etiology: The etiology is not fully known. Appar-
ently, wet periods in May and June favor development of yellow slime disease. The nematode *Anguina tritici* (Steinbuch 1799) Chitwood 1935, associated with spread of *Corynebacterium tritici* (Hutchison) Burkholder 1948 on wheat, may serve as a vector of *C. rathayi* to wheat (*Triticum aestivum* L.). But as this nematode does not go to *Dactylis*, Sabet (1954) suggested that an unknown nematode may serve as a vector. Bradbury (1973a) stated that direct plant to plant transmission seems very unlikely.

**Host Range:** The organism has been reported on orchardgrass, bermudagrass (*Cynodon dactylon* (L.) Pers.), and rye (*Secale cereale* L.) (Bradbury, 1973a).

**Literature:**

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**Bacterial Stripe of Sorghum** (*Sorghum bicolor* (L.) Moench = *S. vulgare* Pers.), caused by *Pseudomonas andropogonis* (Smith 1911) Stapp 1928 (*Pseudomonas stizolobii* (Wolf 1920) Stapp 1935). Figure 3

This bacterial species has a wide host range that includes gramineous, leguminous, and ornamental species. In 1981, *P. andropogonis* reportedly caused a bacterial dark spot of coffee in Brazil (Hayward, 1983). There is some evidence of pathogenic specialization, suggesting that listing of pathovars may become possible.

**Symptoms:** The bacteria cause red streaks and blotches on the leaves and sheaths. Lesions at first appear to be water-soaked dark green specks. Single lesions are only a few millimeters in diameter but may extend to appear later as long red streaks between the veins. When lesions coalesce, they may cover a large part of a leaf blade. The red coloring is not marginal but is continuous throughout the lesion. Bacterial exudate occurs on the undersurface of the leaves; when dry, the exudate appears as light red scales. Lesions are similar in form on all varieties, but may vary from deep reddish-brown or purple to brick red. Lesions on a few varieties are tan to brown, but show no reddening, suggesting that difference in color is due to the host.

**Etiology:** The bacteria enter through the stomata. Spread is probably by wind-driven rain.

**Host Range:** *Agropyron intermedium* (Host.) Beauv. is resistant to certain strains but susceptible to others (Tominaga, 1968a, 1971).

**Literature:**

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*Pseudomonas avenae* is the cause of bacterial leaf blight of oats (*Avena sativa* L.), bacterial leaf blight, stalk rot of maize (*Zea mays* L.) and teosinte (*Euchlaena mexicana* Schrad. (= *Zea mays* spp. *mexicana* (Schrad.) Illis)], and bacterial brown stripe of foxtail and other grasses (Bradbury, 1970b). The causal agent was named *Pseudomonas alboprecipitans* until Schaad et al. (1975) demonstrated that the criterion used to establish this species was not valid, and recommended the designation *Pseudomonas avenae*. Investigations by Tominaga (1971) and a summary of available data by Bradbury (1973b) suggest that *P. alboprecipitans* (*P. avenae*) is closely related to *P. setariae* (Okabe) Burkholder 1939; Tominaga (1971) considers them synonymous. In addition to their identical culture tests, Tominaga (1971) reported that two isolates of *P. alboprecipitans* were serologically identical to one of *P. setariae* and closely related to a second. Goto (1964) suggested that *P. setariae* was synonymous with *P. panicii* and *P. panicii-miliacei* (Ikata) Yaumati 1947.

In conclusion, under current rules and findings, *P. avenae* is the only valid name for bacteria still referred to in the literature as *P. alboprecipitans*, *P. panicii*, *P. panicii-miliacei*, and *P. setariae*.

**Symptoms:** These bacteria induce light brown spots and streaks of no particular size or shape (Bradbury, 1973b). The spots occur mostly on the blades and sheath, but the lesions may occur on any above-ground part of the plant. Depending on the host, these lesions may vary from indefinite light yellow areas to greyish-green withered spots. On oats, there is often a reddish tinge. On broomcorn millet (*Panicum miliaceum* L.), this organism causes narrow, brown, water-soaked streaks on leaves, sheaths, and culms. When the streaks coalesce, the tissue becomes
brown and translucent. Abundant exudate dries, forming shiny thin scales along the streaks. Similar lesions occur on the peduncles and pedicles of the panicle. In severe infections, the entire upper part of the plant is killed, and new shoots emerge at the base.

**Etiology:** The presence of bacterial exudates on the lesions suggests transmission by wind and rain (Bradbury, 1973a). The bacteria enter the plants through stomata and hydathodes (Bradbury, 1973a).

**Host Range:** These bacteria have been recorded as occurring on, or causing lesions when inoculated on, rice (*Oryza sativa* L.), foxtail millet (*Setaria italica* (L.) P. Beauv.), broomcorn millet, barley (*Hordeum vulgare* L.), *Aegopyron intermedium,* *A. tricophorum* (Link) Richt., rescuegrass (*Bromus unioloides* H.B.K. (= *B. catharticus* Vahl)), smooth bromegrass (*B. inermis* Leyss.), *B. marginatus* Nees ex Steud., yellow bristle-grass (*Setaria lutescens* Weigel), teosinte, meadow foxtail (*Alopecurus pratensis* L.), tall oatgrass (*Arrhenatherum elatius* (L.) Beauv. ex. J. & C. Presl.), oats, Japanese millet (*Echinochloa frumentacea* (Roxb.) Link), meadow fescue (*Festuca pratensis* Huds.), velvetgrass (*Holcus lanatus* L.), annual ryegrass (*Lolium multiflorum* L.), sorghum, and wheat.

**Literature:**


**Bacterial Leafblight of Foxtail** (*Setaria lutescens*)
caused by *Pseudomonas syringae* van Hall 1902.

This pathogen is quite cosmopolitan in its host range, producing symptoms that vary somewhat on different hosts.

**Symptoms:** Symptoms vary, depending on the hosts. Lesions on all hosts are usually round-oblung, linear to irregular, and of various sizes. The lesions are not usually limited by the vascular bundles. On foxtail, the spots are small and dark brown, surrounded by a narrow light green halo. On sorghum, sudangrass (*S. sudanense* (Piper) Stapf), and Johnson grass (*S. halepense* (L.) Pers.) (Kendrick, 1926), the spots are red, or light-centered with a red margin, depending on the variety. Some varieties have spots with dark brown borders. Lesions on pearl millet (*Pennisetum typhoides* (Burm.) Stapf & C.E. Hubb) are dark brown and have a slight halo.

**Etiology:** Infection is through stomata or wounds. The organism is at first intercellular, but soon causes a collapse of the tissues. It then becomes intracellular. The presence of a halo often reflects reaction to a toxin. The causal agent may overwinter in seed, on seed, in stubble, or in the soil.

**Host Range:** In inoculation trials, the following species were resistant to *P. syringae:* Triticeum vulgare Vill., oats, smooth bromegrass, tall oatgrass, orchardgrass, perennial ryegrass (*Lolium perenne* L.), *Phalaris* sp., *Agrostis* spp., timothy (*Phleum pratense* L.), meadow fescue (*Festuca pratensis*), and the following varieties of *Setaria lutescens:* Japanese, Siberian, common, Hungarian, and broomcorn.

**Literature:**


*Pseudomonas syringae* pv. *atrofaciens* occurs on wheat, barley, rye, and triticale (Dowson, 1957, Zillinsky, 1983). At this time, it has not been reported on forage grasses.

**Symptoms:** The pathogen attacks the leaves and inflorescences of the plant. The young lesions on inoculated leaves are small, dark water-soaked spots. These enlarge and elongate, turn yellow and finally turn light brown as the tissues dry. Unlike those produced by the black chatt pathogen, *Xanthomonas campestris* pv. *translucens* (Jones, Johnson & Reddy 1917) Dye 1978, the spots are not translucent nor do the bacteria exude in droplets to form encrustations. Infected glumes exhibit dull brownish black areas at the base (basal glume rot), sometimes extending over nearly the entire surface of the glume. The dark staining is more pronounced on the inner sides of glumes.
and lemmas, and the staining may extend to the rachis and kernels.

Etiology: Little is known about the disease cycle of this bacterium.

Host Range: Oats, barley, and wheat are hosts, but occurrence of the pathogen on forage grasses has not been reported.

Literature:


Schaad and Cunfer (1979) examined the physiological, biochemical, serological, and pathological properties of several strains of Pseudomonas coronafaciens (Elliott 1920), P. coronafaciens pv. zeae (Riberio, Durbin, Arny & Uchytill 1977), P. coronafaciens subsp. atropurpurea (Stapp 1928), and Pseudomonas striafaciens. They concluded that the divisions of strains of P. coronafaciens and the separation of P. coronafaciens from P. striafaciens (Elliott 1927) was not tenable because of the minor differences in pathogenicity and symptomology. We follow their recommendations and list P. striafaciens as a synonym of P. syringae pv. coronafaciens.

Symptoms: P. syringae pv. coronafaciens causes a spot disease of the foliage, sheaths, and glumes of several Gramineae. On leaves and glumes, the spots are circular to elliptical water-soaked light olive-green spots with brown centers. Later they become linear and dark chocolate or purplish-brown to black. Depending on the bacterial strain involved, the spots may or may not be accompanied by yellow halos. The centers become slightly depressed. Lesions may coalesce and destroy the entire leaf. Upper nodes are sometimes killed, and panicles may wither and die (Bradbury, 1970a,c).

Etiology: Infection takes place through wounds and stomata and is at first intercellular and later intracellular in the parenchyma. The pathogen may be transmitted on seed and may overwinter in lesions on dead bromegrass (Bradbury, 1970a).

Host Range: The pathogen has been reported (Tominaga, 1968b) to be pathogenic naturally or when inoculated onto the following grass species: quackgrass (Agropyron repens L.), red oats (Avena byzantina K. Koch), rescuegrass, smooth bromegrass, Japanese chess (B. japonicus Thunb. ex Murr.), soft chess (B. secalinus L.), cheatgrass (B. tectorum L.), mountain brome (B. hookeriatus Thunb. (= B. carinatus Hoof & Arn.)), meadow fescue, Hordeum bulbosum L., H. stenostachys Godron, barley, annual ryegrass and perennial ryegrass, timothy, rye, wheat, and maize.

The following grass species have been reported to have resistance to certain strains of the pathogen: big quakinggrass (Briza maxima L.), rescuegrass, Bromus hordeaceus L., smooth bromegrass, foxtail chess (B. rubens L.), orchardgrass, tall fescue (Festuca arundinacea Schreb.), meadow fescue, annual and perennial ryegrasses, and reed canarygrass (Phalaris arundinacea L.), timothy, and Kentucky bluegrass (Poa pratensis L.) (Tominaga, 1968b).

Literature:

Bacterial Wilt of Orchardgrass, Annual Ryegrass, and Meadow Fescue caused by Xanthomonas campestris pv. graminis (Egli, Goto, & Schmidt 1975) Dye 1978. Figures 1, 9

Bacterial wilt of forage grasses is a relatively new disease, first described by Egli et al. (1975). They observed the bacterium at several locations in Switzerland, France, and Germany and discussed its disease-inciting potential. Later, Egli and Schmidt (1982) reported its occurrence in the United Kingdom, Belgium, and New Zealand, and that its host spectrum is relatively large. They listed species of Lolium, Festuca, Trisetum, Dactylis, Phleum, Poa, and Arrhenatherum as hosts.

Symptoms: The disease becomes most evident when the plants begin to head. The young leaves curl and
withers, and shoots become stunted or may die. Less severely infected plants continue to form shoots, but their emerging inflorescences are small and distorted. The older leaves often become chlorotic and later necrotic along the margin of the blades. Chlorotic and necrotic zones form along the vascular bundles and usually extend over the length of the leaves into the sheaths. During the colder seasons, young leaves may exhibit such discoloration.

Formation of bacterial droplets or slime on surfaces of diseased leaves has not been reported. If diseased stalks are cut off, yellow bacterial ooze may be observed hanging on the inside walls of the lumen. Low-power magnification of the edge of a cut infected leaf in water should show masses of bacteria streaming out of the vascular system. Bacteria have been observed in root and stalk sections.

**Etiology:** Although the etiology has not been reported, studies with artificial inoculation have demonstrated that the bacteria enter the plant through wounds, and may be spread by cutting knives. Mechanical harvesting is a likely mechanism for spread of the pathogen. The bacteria may overwinter in the roots, and seed transmission may also be a possibility.

**Host Range:** Egli et al. (1975) proposed naming the causal agent of bacterial wilt *Xanthomonas graminis* because of its specific host range. Dye (1978) and Dye et al. (1980), using the standards for naming pathohvars of phytopathogenic bacteria, placed this group of bacteria under *X. campestris* as a pathovar, i.e., *X. c. pv. graminis*. Additional research revealed that the agents causing bacterial wilt of forage grasses can be assigned to one of at least four intrasubspecific subdivisions, distinguished by their host-specific pathogenicity. Egli and Schmidt (1982) identified these four pathovars as: *X. campestris pv. graminis*, *pv. phlei*, *pv. pae*, and *pv. arrhenatheri* from *Phleum*, *Poa*, and *Arrhenatherum*, respectively. The host range of *pv. graminis* is relatively wide.

DeCleene et al. (1981) determined that 10 of 11 forage grass cultivars cultivated in Belgium were susceptible to *X. campestris pv. graminis*.

**Literature:**


**Translucent Leaf Stripe of Grasses and Cereals caused by *Xanthomonas campestris* pv. *translucens* and related pathohvars.**

This disease, also known as bacterial stripe and black chaff, is common on cereal crops and occurs in all cereal-growing regions of the world (Zillinsky, 1983). All above-ground parts of the plant may be affected, but the disease occurs most commonly on the leaves and glumes.

**Symptoms:** Leaf lesions first appear as small water-soaked areas which enlarge longitudinally, forming irregular translucent stripes which may extend the full length of the blade and sheath. The water-soaked lesions remain translucent for a long period before drying to a yellowish to brownish cast. Blotch-like lesions may also occur and may cause portions of the leaf to shrivel and become light brown. Under humid conditions, bacterial exudate appears as small turbid drops along the lesions and hardens into yellowish resinous granules or into a yellow crust. If the flag leaf is infected before the head emerges, the bacterial exudate may prevent the sheath from opening, thereby impeding emergence of the head. If they emerge, the heads may be bent or distorted and part of the seed blighted.

If the glumes and lemmas are affected, the disease is called black chaff, easily recognized by the dark, linear, water-soaked streaks. As the disease progresses, the lesions merge, producing a dark staining of the glumes, lemmas, and peduncles. Under suitable conditions for the pathogen, the kernels may become stained and shriveled.

Symptoms of black chaff may be confused with the brown necrosis caused by physiological reactions to certain environmental conditions and bacterial stripe caused by *Pseudomonas syringae* pv. *coronafaciens*. Therefore, isolation and growth on certain media are necessary for positive identification.

**Etiology:** *X. campestris pv. translucens* invades through the stomata, and spreads through the intercellular spaces of the parenchyma. The pathogen survives in its
host, and is seed-borne, but will not survive in the soil. During the growing season, local spread is by wind-blown rain and contact.

Host Range: While the symptoms and pathogenic characteristics of the four *X. campestris* (Pammel) Dowson 1939 pathovars are similar, their host ranges are different. The host range for each of the four pathovars is presented here. The listings, except for that of *X. campestris* pv. *translucens*, are from Bradbury (1984). Publications of Tominaga (1967, 1971) have been summarized for the host range of the latter:


*X. campestris* pv. *translucens*. Hosts: Japanese millet, orchardgrass, smooth bromegrass and quackgrass. Investigations completed by Tominaga (1967, 1971) showed that the following pasture grasses were resistant to the strains of the pathogen tested: red top (*Agrostis alba* L. = *A. gigantea* Roth), tall oatgrass, smooth bromegrass, *Bromus marginatus*, soft chess (*B. mollis* L.), tall fescue, meadow fescue, annual and perennial ryegrasses, and Kentucky bluegrass.


Literature:


DISEASES INCITED BY FUNGI

The fungi, an extremely heterogeneous group of lower plants, are devoid of chlorophyll. They exist either as saprophytes or parasites, with reproductive structures that usually bear well-defined spores. Vegetative growth is attained primarily by threadlike mycelia that are specialized hyphae. Reproduction is generally by means of spores that are specialized unisporous or multicellular propagative bodies. Fungus spores may be formed asexually or by the sexual process. Spores are microscopic in size, ranging from about 5 to 100 micrometers in length, depending on the species. They are dispersed by wind, splashing or flowing water, animal vectors, and machinery. Although infections of plants are often initiated from germinating spores, in some species infection may be initiated from mycelia from specialized vegetative resting structures such as small seed-like sclerotia.

RUSTS caused by Puccinia spp. and Uromyces dactyliidis Otth.

The rust fungi, worldwide in distribution, are highly specialized obligate parasites that cause diseases of high economic significance. They produce from one to five distinct spore forms. Autoecious rusts produce their spores on a single host or on two closely related hosts, and the heteroecious rusts produce spores on two unrelated hosts. Physiologic forms (formae speciales) of rust fungi are differentiated according to their ability to attack different host species.

Crown Rust caused by Puccinia coronata Cda. Figure 10

Crown rust is worldwide in distribution; specialized forms occur on many cultivated and wild grasses. Many physiological races also occur within formae speciales. In the northeastern United States, P. coronata is destructive on meadow fescue. The disease incitant causes considerable loss of foliage and a reduction in forage quality. Leaves of infected plants usually die. Buckthorns (Rhamnus cathartica L. and R. frangula L.) are alternate hosts of this heteroecious rust. Crown rust is also the most important grass rust in Great Britain, where it causes severe damage in perennial and annual ryegrasses and meadow fescue.

Symptoms: Scattered bright orange-yellow pustules (uredia) develop on the upper and lower leaf surfaces of grass hosts, but are found mainly on the upper side of the leaf. Uredia may also occur on leaf sheaths. In severe epiphytotic, the leaves turn pale yellow and wither completely. A powdery mass of urediospores forms in the uredia. During winter in milder climates, urediospores continue to develop alongside the black, linear telial stage which produces teliospores. Telia often form in rings around the uredia. In a heavy infestation, infected plants wither, turn yellow, and die from excessive loss of moisture.

Etiology: The fungus overwinters as teliospores. These germinate in the spring, producing basidiospores that infect both buckthorn species. Aeciospores are subsequently produced on the alternate host and are disseminated by wind and rain to a susceptible grass host. Uredia develop from the aeciospore infection in the host and the life cycle is complete with the subsequent production of teliospores. In milder climates, the fungus may overwinter as mycelium in the host or as uredial pustules that resume sporulation in the spring.


Control: Resistant cultivars offer an appropriate means to control crown rust in economically important Gramineae. Braverman (1967, 1986) recorded many cultivars, comprising several grass genera and species, developed specifically for crown rust resistance.

Literature:

Stem Rust caused by Puccinia graminis Pers. Figure 11

Stem rust (black stem rust), worldwide in distribution, is highly destructive on cereals and affects numerous grasses of economic importance by reducing
yield, quality of forage, and seed production. The fungus is heteroecious, alternating from grass to barberry (*Berberis vulgaris* L.) or *Mahonia* spp. The disease is most damaging in moderately humid areas.

**Symptoms:** The red rust, or uredial, stage is prominent on leaves and culms of grasses during the growing season. As infection progresses, the epidermis is ruptured by uredial pustules containing orange-red spore masses (urediospores). These spores may become wind-blown and spread to other plants, initiating infection. As the diseased plant matures, brown-black, oblong to elongated telia (black rust stage) develop in the uredia or in new sori on sheaths and culms. The telia give rise to black teliospores.

**Etiology:** The fungus overwinters as teliospores. In more mild climates, overwintering occurs as urediospores or teliospores on the host plant or as dormant mycelium. The teliospores germinate in the spring to produce basidiospores which subsequently reinfect the same host and eventually form uredia of this autoecious rust.

**Host Range:** The host range includes species of *Agropyron*, *Agrostis*, *Andropogon*, *Dactylis*, *Festuca*, *Lolium*, *Phleum*, *Poa*, and *D. glomerata* (Cummins, 1971).

**Control:** Braverman (1967, 1986) has summarized those genera and species in the Gramineae with reported resistance to stripe rust.

**Literature:**


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**Yellow Leaf Rust** caused by *Puccinia poae-nemoralis* Otth.

Yellow leaf rust attacks several grass genera but does not infect cereals. *P. poae-nemoralis* has several specialized forms, but only the form that parasitizes tall oatgrass has a pycnial and aerial stage on barberry.

**Symptoms:** The fungus produces round to oval, yellow-brown to red-brown uredial sori on the upper leaf surface and also on the leaf sheaths. The sori are covered by the epidermis and surrounded by chlorotic leaf tissue, and may be arranged in dense groups (Weibull, 1983). Telia, rarely found, are brown to black and covered by the epidermis. When present, they are found on the lower leaf surface.

**Etiology:** The fungus overwinters as urediospores. No aerial stage has been reported; teliospores germinate to produce basidiospores which subsequently reinfect the same host and eventually form uredia of this autoecious rust.

**Host Range:** The host range includes species of *Festuca* and *Poa* (Cummins, 1971).

**Control:** Braverman (1986) has listed a number of re-
sistant *P. pratensis* cultivars.

**Literature:**

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**Leaf Rust** caused by *Puccinia poarum* Niels. Figure 12

This fungus is primarily a pathogen on *Poa* spp., with an alternate host of cloverfoot (*Truslago farfara* L.), and is worldwide in distribution.

**Symptoms:** The rust produces oval to elongate orange-yellow uredia on the upper leaf surface and occasionally on the culms. The telia are black and abundant, and appear on both surfaces of the leaf.

**Etiology:** Aeciospores from cloverfoot are the main source of infection. Urediospores may be disseminated by wind to infect new plants. However, the uredial stage is brief, with telia appearing two to three weeks after an initial infection. The fungus overwinters as teliospores that germinate to form basidiospores in the spring, subsequently infecting cloverfoot.

A distinguishing feature of *P. poarum* is the production of two generations of aeciospores, especially in Ireland and Great Britain (O’Rourke, 1976). The first generation of aeciospores appears in late spring and the second in late summer. Urediospore infection occurs between each generation of aeciospores.

**Host Range:** The host range includes species of *Agrostis*, *Festuca*, *Phleum*, and *Poa* (Smiley, 1983).

**Control:** Braverman (1986) listed numerous Kentucky bluegrass cultivars with resistance to *P. poarum*.

**Literature:**

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**Uromyces Leaf Rust** caused by *Uromyces dactylidis* Otth.

The principal leaf rust of orchardgrass, caused by *U. dactylidis*, occurs in mid to late summer. In the northeastern United States, severe infection will reduce forage yields and quality (Kreitlow et al., 1953). The rust is heterocous, with crowfoot (*Ranunculus* spp.) as alternate hosts.

**Symptoms:** Yellowish-brown powdery uredia occur, primarily on the upper leaf surface. Telia form on the lower leaf surface or on culms and produce yellow-brown teliospores.

**Etiology:** The fungus overwinters as teliospores; these germinate to produce basidiospores. These spores are wind-blown onto crowfoot and develop into the pycnial and aecial stages. Aeciospores on crowfoot are disseminated by rain and wind to an appropriate host. Urediospores appear in midsummer.

**Host Range:** The host range includes *Dactylis glomerata* and species of *Agrostis*, *Festuca*, and *Poa* (Couch, 1973; Smiley, 1983).

**Control:** Braverman (1986) listed orchardgrass cultivars resistant to *U. dactylidis*.

**Literature:**

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**SMUTS** caused by *Ustilago* spp., *Urocystis* spp., *Entyloma* spp., and *Tilletia* spp.

The numerous smuts which occur on grasses infect the inflorescences, leaves, stems (culms), and caryopses. The spore-bearing sori are specific to certain morphological portions of the grasses. Smuts are worldwide in distribution and can be of major economic importance. If foliage is attacked, forage yield and quality are reduced. If inflorescences are infected, seed yield is reduced.

Smuts are divided into leaf, stem, and head smuts. Leaf smuts of grasses appear on the leaf blades and leaf sheaths. These smuts are divided on the basis of symptoms: stripe smut, flag smut, and
Spot smut. Sori of these smuts are linear and form long or short stripes between the leaf veins. The leaf epidermis ruptures, discharging the spores and shredding the leaves. Chlamydospores are formed in the shredded tissue. Sori in leaf spot smuts are covered by the epidermis and are more permanent.

In seedlings where crown tissue is invaded, infection is followed by a systemic infection of the primordia. In perennial grasses, sori may also appear on the leaf sheath. In a heavy infection, growth is retarded and inflorescence development is inhibited.

Etiology: The pathogen overwinters as dormant mycelium in plant debris or as chlamydospores in the soil or on the seed. In the spring, infection hyphae develop from the chlamydospores and penetrate the coleoptiles of young seedlings or tillers in older grasses. The host is systemically colonized. Cool, moist weather favors disease development. Masses of chlamydospores develop in the colonized foliage, and these spores eventually rupture the epidermis and are dispersed by wind and rain.

Host Range: Stripe smut occurs on red top, creeping bentgrass (Agrostis alba Huds.), colonial bentgrass (A. tenuis Sibth.), orchardgrass, timothy, and Kentucky bluegrass. It is less widely distributed on species of Agropyron, Bromus, Elymus, Festuca, Lolium, and reed canarygrass (Fischer, 1953; Smiley, 1983).

Control: Braverman (1986) listed several cultivars of grass genera and species developed for resistance to stripe smut. Fungicide seed treatment and crop rotation are useful control practices.

Literature:

Flag Smut caused by Urocystis agropyri (Preuss) Schroet.

Flag smut infects a wide range of the Gramineae and is worldwide in distribution. The disease is usually not destructive.

Symptoms: Symptoms induced by U. agropyri are similar to those of stripe smut, but the disease is manifest especially in the upper leaves of infected hosts. According to O’Rourke (1976), diseased plants are stunted, and production of total dry matter and inflorescence is markedly reduced. Sori, which develop beneath the epidermal cells in the leaf, rupture and release powdery masses of spore balls. Chlamydospores of flag smut may be distinguished from stripe smut spores with a microscope; flag smut produces spore balls in the smut sori. The spore balls consist of one to four dark, smooth, spores surrounded by smaller, hyaline to pale brown, sterile cells (Kreitlow et al., 1953). Leaf sheaths may also be infected.

Etiology: The etiology of flag smut is similar to that of stripe smut. Infection occurs in coleoptiles of young seedlings or in underground lateral buds of mature plants. The host is systemically colonized, and sori eventually erupt through the epidermis and disperse by wind and rain.

Host Range: Flag smut infects species of Agropyron, Agrostis, Bromus, Elymus, Pileo, Poa, orchardgrass and red fescue (Festuca rubra L.) (Fischer, 1953; Smiley, 1983).

Control: Some systemic fungicides provide an effective control of flag smut in breeding stock nurseries (O’Rourke, 1976). Several Kentucky bluegrass cultivars are noted by Braverman (1986) as resistant.

Literature:
Leaf Spot Smuts (Blister Smuts) caused by Entyloma spp.

Leaf spot smuts are the third category of the leaf smuts. They are caused by Entyloma spp. and are common on many grasses. A representative smut of this group, *E. dactylis* (Pass.) Cif., is worldwide in distribution. These smuts are differentiated from *Tilletia* and *Urocystis* in that sori of *Entyloma* in the leaf forms discolored spots that are frequently light in color, hence the common name “white smuts.” Aerial conidia may be formed on the surface of the spots, giving the spots an appearance similar to that of powdery mildew.

Symptoms: Sori are formed on the leaves and less frequently in the floral bracts. Fruiting structures resemble tar-like angular to oblong spots. The epidermis subsequently in the floral bracts. Fruiting structures resemble the spots an appearance similar to that of powdery mildew.

Etiology: *Entyloma* bluster smuts are not systemic in the host. According to Smiley (1983), most of the infection results from sporidia spreading on the leaves. Sporidia are disseminated by rain, leaf-to-leaf contact, and by movement of equipment and animals.

Host Range: The fungus occurs on species of *Agrostis, Festuca, Phleum,* and *Poa* (Smiley, 1983).

Literature:

Culm Smuts caused by Ustilago spp.

These smuts, also called sheath smuts, are found in western North America, South America, Europe, North Africa, and Asia. Dickson (1956) reports four species: *U. sphenazzii* (Hirschh.) Fischer (=*U. hypodytes* Aucht.) appears to be the most ubiquitous.

Symptoms: Smut sori are superficial on the internodes of the culms and occasionally on aborted inflorescences. Naked linear dark brown to black sori are covered by leaf sheaths. Infections also occur in the crown-bud primordia, and sori appear for two to three seasons following infection. Mycelium will also persist in stolons and crowns of perennial grasses.

Control: Some selections of crested wheatgrass (*Agropyron cristatum* (L.) Beauv.) apparently are resistant (Dickson, 1956).

Literature:

Head Smuts caused by species of Ustilago, Sorosporium, and Sphacelotheca.

These smuts, most abundant in western North America, form sori in grass inflorescences. Head smuts (loose smuts) produce spores in the total inflorescence, or just in the floral bracts and ovaries. They are differentiated from kernel smuts, in which sori form only in the ovaries.

These three genera of the Ustilaginaceae are differentiated by these characteristics: *Ustilago*—sori naked or without an enclosing membrane and forming black, dusty masses at maturity; *Sorosporium*—sori generally dusty and without an enclosing membrane; *Sphacelotheca*—sori generally in the inflorescence, replacing the kernels; and with a false external membrane.

*Ustilago bulbata* Berk. causes common head or ear smut in a variety of grasses in the western United States and in drier climates; it has also been found in the central and eastern United States. Sori form in the spikelets, involving all or part of the floral bracts, and are enclosed in the epidermal membranes of the floral structure. The loose to semi-covered spore mass is dark brown to purple-black. The powdery mass of spores that replaces the kernels is usually covered by the glume; glumes are rarely destroyed completely. Seedling infection occurs in perennial grasses, and the disease may survive in the host for years.

Host Range: Species of *Agropyron, Bromus, Elymus,* and *Festuca* are common hosts (Fischer, 1953).

Control: Several rescuegrass cultivars resistant to head smut have been developed; mountain bromegrass 'Bromar' is also resistant.

*Ustilago avenae* (Pers.) Rostr. causes a loose smut of tall oatgrass, generally wherever the grass is grown. The seeds are replaced by black, compact sori which eventually become powdery. These sori replace the floral parts.

Literature:

Kernel (Covered) Smuts caused by Tilletia spp.

Some kernel smuts caused by species of *Tilletia* infect
only the ovary of the respective host. These smuts are common in western North America, the intermountain states, and in similar climatic areas in other countries. The smut sorus form in the ovaries and assume the general shape of the caryopsis.

**Symptoms:** This smut affects the floral parts of the host. Diseased seed remains green longer than healthy seed, and when mature is somewhat darker brown than healthy seed. Eventually, only the pericarp of diseased seeds remain, with their interiors completely replaced with brown, powdery spore masses.

**Etiology:** The smut fungus survives as chlamydospores on the seed and overwinters in this stage. In the spring, chlamydospores germinate concurrently with the seed. Infection hyphae eventually form and penetrate a seedling. The fungus grows within the host until floral formation, at which stage the flower tissue is colonized, and subsequently the entire pericarp of the seed is replaced by smut spores.

**Host Range:** *Agrostis* spp. are hosts (Fischer, 1953).

**Literature:**

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**Powdery Mildew caused by Erysiphe graminis DC. ex Merat.** Figure 13

Powdery mildew occurs on most Gramineae, wherever they may be growing. The disease is economically important in grass nurseries, grass breeding stocks, and expansion plantings, but not so in pastures. In Great Britain, Davies et al. (1970) have shown that powdery mildew reduces the yield and quality of ‘Lior’ annual ryegrass.

*E. graminis*, an obligate parasite on grasses, includes many specialized varieties and races. Few are restricted to a single grass species. Several races may attack a single plant species, and thus provide an excellent opportunity to form new races of the fungus.

**Symptoms:** The fungus attacks the aerial parts of the plant, but generally only the foliage is injured. The mildew first appears as oblong, irregular, white, powdery blotches on the upper surface of the leaf. Orange-yellow areas develop beneath the white blotches. The powdery mycelial colonies enlarge, and may coalesce until the total leaf surface is covered with grayish-white mycelium. Eventually, the leaf yellows and turns brown. The powdery tufts of mycelium support numerous conidiophores from which conidia are formed in long chains. Symptoms are less evident on grasses grazed or cut closely. Cleistothecia may develop and appear in the powdery mycelial tufts as tiny, round brown-black fruiting structures.

**Etiology:** The severity of this powdery mildew depends primarily upon climatic conditions. Powdery mildew appears during cool and somewhat cloudy conditions. Couch (1973) has shown that in the United States four conditions are essential for maximum mildew development: (i) reduced air circulation; (ii) high atmospheric humidity; (iii) low light intensity, and (iv) air temperature about 18 C.

The pathogen overwinters as cleistothecia and mycelia on living host tissue; the cleistothecia are probably of secondary importance (Kreitlow et al., 1953; Weibull, 1978b). New infections are caused by conidia from overwintering host plants. Additional infections may occur, when cleistothecia containing ascospores may develop and serve to maintain the fungus in hot weather or under conditions not favorable for production of mycelia and/or conidia. Conidia will germinate over a wide temperature range. Free moisture is a deterrent to the germination of conidia.

**Host Range:** Most species of the Gramineae are mildew susceptible. The disease is of economic significance in *Poa, Festuca, Dactylis*, and *Bromus*. Susceptible species were tabulated by Sprague (1950).

**Control:** Sulfur dusts, cycloheximide, and systemic fungicides will control this fungus in seed crops and in the greenhouse (O’Rourke, 1976). Chemical control is impractical on grazing lands, and resistant cultivars offer the best means of control in these plantings. Braverman (1986) has listed numerous grass species reported to be powdery mildew resistant, along with cultivars developed specifically for mildew resistance.

**Literature:**
Ergot caused by *Claviceps purpurea* Tul. Figure 14

Ergot affects worldwide the inflorescence of numerous cereals and grasses of economic importance in temperate zones. Specialized races occur, particularly in Japan. Many *Claviceps* species have been described on the basis of host specialization, rather than distinct morphological characteristics. The economic importance of *C. purpurea* is two-fold: affected plants produce ergots (sclerotia) instead of seed, thereby reducing yield, and the alkaloidal substances in the ergots are toxic to livestock or humans feeding on the infected grains. According to O'Rourke (1976), ergot-infested grain when ingested may cause two distinct syndromes—gangrene of the extremities and stimulation of the nervous system, causing convulsions. Ergot-infested grain will cause abortion in cows and ewes (Carr, 1971).

**Symptoms:** The disease first appears as a sticky "honeydew" ooze on the young ovary. The sugary ooze attracts insects and also forms a substrate for other microorganisms (Cunfer, 1976). This primary infection and subsequent development of a sclerotium preempts the ovary. The most noticeable phase of the disease is the appearance of horny, curved, dark olive to olive-purple sclerotia that project from the inflorescence. A sclerotium is generally two to three times the length of the seed and may be corrugated longitudinally. Transverse or longitudinal cracks with reddish margins may occur, exposing the light-colored interior. Sclerotia produce a conspicuous odor (Walker, 1970). The sclerotia remain attached to the plant until it is mature. Sclerotia infecting large-seeded grass genera such as *Arrhenatherum*, *Bromus*, *Festuca*, and *Lotium* may be as long as 20 mm. Narrow, smaller sclerotia (up to 10 mm long) are confined to *Dactylis*, *Phleum*, and *Phalaris*, which are grasses with smaller seeds. Unfortunately, sclerotia are usually harvested with the seed.

**Etiology:** Ergot sclerotia fall to the soil when the host plant matures, and overwinter in soil or in stored grain (Kreitlow et al., 1953). In the spring, the sclerotia germinate to form minute mushroom-like stalks with rounded heads in which perithecia develop. Arrival of the wind-blown ascospores must occur at full floral development of the host plant. It is believed that insects transmit the ascospores during pollination. Spores infect the young ovaries of the grass flowers by direct penetration, which eventually produces the "honeydew" ooze from this primary infec-

tion. The ooze contains millions of tiny conidia of *Sphacelia segetum* Lev., which is the asexual stage of the fungus. The inoculum is transmitted by insects and water to healthy flowers. The sclerotia eventually develop below the tiny mass of *Sphacelia* spores, mature, and complete the cycle.

**Host Range:** Ergot infects a multitude of genera in the Gramineae. Most of the known hosts have been reported by Sprague (1950).

**Control:** The use of ergot-free seed provides disease control, for it eliminates a potential inoculum source. Ergots can be separated from seed by floating them off in brine. Crop rotations of two to three years between susceptible crops and legumes or non-susceptible hosts, mowing of roadside grasses, and cleaning borders and headlands prior to formation of the "honeydew" stage will reduce the secondary inoculum.

**Literature:**

**Blind Seed** caused by *Gloeotinia temulentana* (Prill & Del.) Wilson, Nobel & Gray (Syn. *Phiala temulentana* Prill & Del.).

Blind-seed disease may cause considerable losses in seed production. Perennial ryegrass is the most common host, but annual ryegrass and some other grass genera of economic importance are also susceptible. The disease is endemic in nearly all places where ryegrass is grown for seed—Europe, Oregon (United States), Australia, and New Zealand—especially true in regions where the summers are cool, moist and early. An early infection destroys the embryo, while a later infection results in the typical blind seed which fails to germinate. According to O'Rourke (1976), there is conflicting evidence on the possible effects of blind seed ingested by grazing animals. Feeding in-
fected seeds to sheep in New Zealand apparently caused no ill effects. In France, however, ill effects on animals were attributed to the fungus.

**Symptoms:** The fungus, prevalent in cool, wet growing seasons, infects the developing embryo, which then becomes a partially shrunken rusty-brown Caryopsis rather than a healthy seed. Infected seeds are indistinguishable from healthy seeds but can be detected when glumes are removed.

**Etiology:** The fungus overwinters in infected seeds in the soil and produces above-ground apothecia coincident with the developing ryegrass florets. Cool, damp soil favors an increase in apothecial development by *G. temulenta*, and also increases the length of time which infflorescences remain open. Ascospores are wind-blown and gain entry to an open floret just below the stigma, completing the primary infection. Within two weeks of ascospore germination, abundant macroconidia are produced in a pinkish, slimy matrix on the seed. The conidia are cylindrical, slightly lunate, and hyaline. Microconidia are dispersed by rain, wind-driven water droplets, and by physical contact as inflorescences brush against each other. Macroconidia, functioning in a manner similar to the ascospores, set up secondary infections. Microconidia also occur in pink sporodochia on the seeds. An early infection destroys the embryo, preventing seed formation and producing the typical "blind seed."

**Host Range:** The disease is prevalent on annual and perennial ryegrasses and species of *Agrostis, Dactylis, Festuca, Phleum, Poa*, and *Secale*.

**Control:** Pathogen-free seed appears to be a promising approach to control of blind seed disease. Apparently, the fungus will not survive two years in storage. Hot-water treatment provides some control. Resistant cultivars also present a reasonable approach to control. Tetraploid cultivars are more susceptible than the early maturing selections. Wright and Breese (1966) and Wright (1967) reported that blind seed disease was under phylogenetic control and designed a backcross program aimed at introducing resistance into 'S.24.' Breeding for disease escape, rather than for direct resistance to the pathogen, has been investigated (Wright, 1967). According to O'Rourke (1976), the very early and very late flowering types generally escape infection by the fungus.

**Literature:**


**Leaf Streak** caused by *Scolecorichium graminis* Fckl.

Leaf streak, also known as brown stripe, brown streak, or brown leaf blight, is widely distributed on more than 150 grasses in the temperate zones of the United States, Europe, and South America (Braverman, 1958). The pathogen also attacks cereals and is frequently reported on rye. Brown stripe is present throughout the growing season, but is particularly evident during mid-summer and autumn when leaves and culms are maturing. Severe foliar attacks before maturity cause withering and dying of leaves and reduction in the quality of forage (Kreitlow et al., 1953). Morphological variants of the fungus occur. Braverman (1958) and Graham et al. (1963) have shown that infection by conidia from orchardgrass and tall oatgrass is restricted to the original host species, whereas the causal agent from timothy is pathogenic to both orchardgrass and timothy.

**Symptoms:** Initial foliar lesions appear as tiny, elliptical, purplish-brown water-soaked spots with dark brown borders visible on both leaf surfaces. Subsequent necrosis of adjacent tissue forms elongated brown streaks. Shape, color, and size of the developing lesions depend upon the host species and the age of the plant. Centers of the older lesions usually are ashy gray to almost white. In a moist atmosphere, dense parallel tufts of conidiophores and conidia develop in older lesions. At the close of the growing season, small subepidermal black stromata form in a parallel arrangement. This is the distinguishing feature of leaf streak. Symptoms are similar on all hosts.

**Etiology:** *Scolecorichium graminis* overwinters as stromata in debris and as mycelium in leaf tissue. In spring, the stromata rupture and release conidiophores and conidia, sources of primary inoculum on new spring growth. The conidia are disseminated primarily by rain.

**Host Range:** *Scolecorichium graminis* attacks a multitude of grasses. Smooth brome grass is one of the few economic grasses resistant to the fungus. A complete list of hosts has been recorded by Sprague (1950).

**Control:** Disease resistant cultivars offer a practical means for controlling the disease. Several workers have studied the resistance of orchardgrass to *S.
graminis, and numerous cultivars with varying degrees of resistance have been developed (Braverman, 1986).

**Literature:**

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**Leaf Scald** caused by *Rhynchosporium secalis* (Oud.) Davis and by *R. orthosporum* Caldwell.

Both *Rhynchosporium secalis* and *R. orthosporum* occur on many cereal and grass hosts; the diseases which they incite are also referred to as leaf blotch and leaf streak. Both species are widely distributed in North America, Europe, and Asia. In the United States, Elliott (1962) recorded the destructive nature of *R. orthosporum* on orchardgrass with disease peaks in April-May and July-August. Latch (1966) showed that *R. orthosporum*-infected perennial ryegrass was unpalatable to sheep in New Zealand, while in the United States Gross et al. (1975) noted that *R. secalis* reduced the digestibility of smooth bromegrass in grazing animals. In the Nordic countries, *R. orthosporum* is quite common on orchardgrass, meadow fescue, red fescue, timothy, and Kentucky bluegrass, while *R. secalis* is common on wheatgrasses and to a lesser extent on bromegrasses and annual and perennial ryegrasses (Weibull, 1978c).

**Symptoms:** Leaf scald (*R. secalis*) appears as dark, bluish-gray water-soaked blotches which become light gray with darker brown margins. The lesions may extend up to 30 mm and constrict and shred the leaf blades. The centers of the leaves are gray and become covered with conidia. While *R. secalis* forms irregularly shaped lesions on the upper leaf surface and sheaths, lesions on the under surface frequently show pointed terminals, relating to an overall diamond-shape appearance (O’Rourke, 1976).

Lesions produced by *R. orthosporum* on grass leaves and sheaths are similar to those caused by *R. secalis*. O’Rourke (1976) has noted that the scald-like blotches produced by *R. orthosporum* are usually more diffuse than the lesions produced by *R. secalis*, but not as diamond-shaped on the under-leaf surface. Lesions on the lower leaf surface are chocolate-brown at the margin and pale in the center. Sprague (1950) described *R. orthosporum* on orchardgrass as “white stripe,” since mature lesions become elongated and off-white. Lesions caused by either *Rhynchosporium* species may coalesce, causing death of the leaves and reduction in quality of the forage. Conidia of the two species differ in shape; those of *R. secalis* have characteristic beak, while conidia of *R. orthosporum* are slightly narrower and symmetrical.

**Etiology:** Both species overwinter as dormant mycelium on dead leaves and crop residue. New lesions may form throughout the winter in milder climates. Conidia develop during cool, moist spring weather and are readily disseminated by wind and rain to healthy leaves. New spores will be produced on older leaves as long as conditions are favorable. Cool weather favors development of the disease, although conidia may germinate in temperatures up to 28°C. Seed transmission occurs to a limited extent in *R. secalis*.

**Host Range:** Sprague (1950) has recorded the range of hosts.

**Control:** Eliminating crop residues, rotating crops, and seedling resistant cultivars will help to control leaf scald. Use of resistant cultivars was reported by Braverman (1986).

**Literature:**

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**Mastigosporium Leaf Fleck** caused by *Mastigosporium* spp. Figure 15
Species of *Mastigosporium* infect several valuable forage grasses, including timothy, perennial ryegrass, and orchardgrass in Europe, New Zealand, and North America. *M. rubricosum* (Dearn. & Barth.) Sprague is common on orchardgrass in Ireland throughout the year (O’Rourke, 1976). The disease has been reported to reduce orchardgrass yields and forage quality. O’Rourke (1976) indicates that a 10 percent level of infection reduces orchardgrass yield by 30 percent and its water-soluble carbohydrate content by 50 percent.

**Symptoms:** The disease first appears as elliptical water-soaked spots on both surfaces of the leaf. These darken to purplish-brown flecks with bright orange-tan margins. As the flecks enlarge, the centers become pale and support the growth of many conidia. Marginal lesions may cause localized constrictions of the leaf blade (O’Rourke, 1976). Lesions may also coalesce to form irregular blotches on as much as one-half of the leaf surface.

**Etiology:** The disease is favored by cool, damp weather, and its effects are most prominent in early spring and autumn. When climate favors, conidia are borne on short conidiophores that continuously carry new conidia and are dispersed by water. The pathogen persists in leaf tissue during summer and winter and produces conidia when conditions are favorable. Makela (1970) in Finland reported a stromatic resting stage of *M. rubricosum* and other *Mastigosporium* species. Such a stage is probably necessary to insure the pathogen’s survival during long cold winters.

**Host Range:** In addition to the grasses already mentioned, *M. rubricosum* has been reported on *Agrostis* spp. (Sprague, 1950).

**Control:** Several cultivars have been developed for resistance to Mastigosporium leaf fleck (Braverman, 1986). Additional control measures include cutting or grazing the foliage and prompt removal of infected leaves (O’Rourke, 1976).

**Literature:**

**Spermospora Leafspot** caused by *Spermospora* spp.

Fungi of this genus cause a leafspot disease on various grasses in North America, Ireland, and presumably Europe. Several species have been described, of which *S. lolii* MacGrlve & O’Rourke may cause moderate damage in ryegrasses and fescues.

**Symptoms:** Elliptical or irregular foliar lesions (depending upon the host) may vary from 4 mm to 10 mm in length and are gray-brown or reddish-brown. Discoloration spreads beyond the necrotic tissues (O’Rourke, 1976). Necrotic spots are amphigenous and may be concentrated at the leaf margins, presenting a scald effect. Mature lesions on annual and perennial ryegrasses and fescues may become bleached in their centers, giving an eyespot appearance. The centers bear whitish masses of conidia.

**Etiology:** Presumably, *S. lolii* conidia overwinter on diseased tissue and provide the primary source of inoculum in the spring. Spread of the pathogen by wind-borne or water-disseminated conidia continues throughout the growing season. In milder climates, *S. lolii* may produce conidia throughout the year.

**Host Range:** Sprague (1950) has listed several grasses as hosts to *Spermospora* spp.

**Control:** According to O’Rourke (1976), frequent cutting restricts build-up of the disease by removing infected leaf material.

**Literature:**

**LEAFSPOTS** caused by *Drechslera* spp. and *Bipolaris* spp. (Syn. *Helminthosporium* spp.).

Many graminicolous species of *Drechslera*, formerly classified in the genus *Helminthosporium* Link, cause leafspots, net blotches, leaf blotches, and occasionally foot rot and turf decay on a wide range of Gramineae in the temperate areas of North America, Europe, and Japan. Separation into two genera is based primarily upon the manner of spore germination (Shoemaker, 1959). Those species formerly in *Helminthosporium* whose conidia germinate from all cells were placed in *Drechslera* (Shoemaker, 1959, 1962), and those whose conidia germinate primarily from end cells were placed in *Bipolaris*. Species of
these two genera exhibit a wide range of host specificity, from D. poae (Baudys) Shoem., that incites melting out of bluegrass, to H. sativum Pam., King & Bakke (= Bipolaris sorokinianaun (Sacc. ex. Sorok.) Shoem.), that causes leafspot/blotch on many grass species.

The perfect (sexual) stages of D. dactylidis Shoem. (Pleospora phaeocomes (Rab.) Wint.), D. bromi (Died.) Shoem., (Pyrenophora bromi (Died.) Drechs.), D. siccans (Drechs.) Shoem. (P. lolii Dovaston), and H. sativum (Cochiobolus sativus (Pleospora phaceospora (Coch.) Shoem.). Figure 16

have been described. Conidia of these and other species (for which a perfect stage has not yet been reported) serve as primary sources of inoculum. With the exception of D. catenaria (Drechs.) Ito, there is similarity in the etiology of diseases caused by Drechslera spp. and H. sativum. This characteristic is recognized in the discussions that follow. Conidia form on leaves and are disseminated by wind and water droplets, infecting nearby plants. Early spring infections, particularly evident in D. catenaria, D. dictyoides, D. poae, D. siccans, and H. sativum, may be caused by planting infected seed. Brown leafspot caused by D. bromi is most prominent during cool, wet weather of the spring and fall months.

Descriptions, host range, and recommended control measures for seven Drechslera spp. and H. sativum follow.

**Literature:**


**Brown Leafspot** caused by Drechslera bromi.
Figure 16

Smooth bromegrass is very susceptible to brown leafspot. The fungus usually attacks this host wherever smooth bromegrass is grown in the temperate areas of the United States and in Canada. The fungus apparently is restricted to species of Bromus.

**Symptoms:** The disease first appears as small, dark brown, oblong spots on the foliage which develops in the spring. As the growing season progresses, the spots become dark purplish-brown, elongate, and are surrounded by a yellow band or halo (Kreitlow et al., 1953). Lesions may coalesce, forming large yellow to brown areas. Lesions may envelop the entire leaf.

**Host Range:** The fungus is restricted to smooth bromegrass and several annual and perennial Bromus spp.

**Control:** Resistant cultivars offer partial means of controlling the disease (Braverman, 1986). Berg et al. (1983) studied inheritance of leafspot resistance in smooth bromegrass and demonstrated that lesion size is regulated by multiple genes and that susceptibility to the fungus may be dominant or epistatic to resistance. For these reasons, they concluded that it will be difficult to develop populations with high levels of resistance to the pathogen.

**Net Blotch** caused by Drechslera dictyoides (Drechs.) Shoem. Figure 17

Net blotch, one of the most common foliar diseases of tall and meadow fescue, is prevalent in the eastern and southeastern United States and widespread in the Nordic countries.

**Symptoms:** Foliar lesions, present throughout the growing season, first appear as scattered, dark brown spots on the upper and lower leaf surfaces. When the disease is severe, symptoms will be seen on the leaf sheath. Lesions expand to about 15 mm long and up to 10 mm wide. The larger lesions often develop into extensive lateral and transverse brown threads, creating the net-like total withering of the leaf. Lesions usually remain small when the host is growing rapidly. If grass growth is slow, lesions may spread across the leaf blade and cause an early senescence and yellowing of the leaf tip. This yellowing is characteristic of a heavy infection (Weibull, 1978a). The net blotch symptom is most evident from spring to autumn.

**Host Range:** The fungus is widespread on annual and perennial ryegrasses, meadow fescue, and to a lesser extent on tall fescue. D. dictyoides f. sp. dictyoides (Braverman & Graham) Shoem. occurs primarily on meadow and tall fescues. D. dictyoides f. sp. perenne (Braverman & Graham) Shoem. occurs mainly on annual and perennial ryegrass (Braverman and Graham, 1960).

**Control:** Braverman (1967, 1986) has summarized the reported resistance of meadow and tall fescues and annual and perennial ryegrasses to net blotch.
Brown blight caused by *Drechslera siccans*.

Brown blight is a common foliar disease on annual and perennial ryegrasses in the eastern United States and in Europe. In Ireland, the disease is most prevalent on ryegrasses and meadow fescue. *D. siccans* is fairly common in the Nordic countries.

**Symptoms:** Symptoms caused by *D. siccans* resemble those incited by *D. dictyoides*. However, characteristic net markings are either very faint or lacking on the leaves. The fungus produces numerous dark brown spots, of various shapes, which may coalesce to form large mottled discolored areas. More mature lesions show ash-gray centers and a yellowing of surrounding tissue (O’Rourke, 1976). If the infection is severe, the leaves become yellow at the tip. The blade and sheath gradually wither and die (Kreitlow et al., 1953).

**Host Range:** Brown blight, common on annual and perennial ryegrasses, is less common on meadow fescue and orchardgrass.

**Control:** Several perennial ryegrass cultivars have been developed specifically for resistance to *D. siccans* (Braverman, 1986).

**Literature:**


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Leafspot caused by *Drechslera catenaria*.

*Drechslera* leafspot is not as common as tawny blotch (*Stagonospora foliicola* (Bres.) Bubak), but it is destructive and capable of severely damaging reed canarygrass stands.

**Symptoms:** The initial foliar symptoms appear as tiny, dark green spots surrounded by a chlorotic halo. Mature lesions are elongated and reddish-brown, often with light-buff-colored centers. As lesions increase in size, they often coalesce to form large areas of necrotic tissue on susceptible clones (Zeiders, 1976).

**Etiology:** There is evidence suggesting that pathogenesis by *D. catenaria* is accomplished primarily by secretion of a toxic substance(s) which precedes mycelial colonization of the tissue. The fungus overwinters as mycelium in tissue infected the previous year. *D. catenarium* may also be seed-borne.

**Host Range:** The fungus is common on reed canarygrass, woodreed (*Cinna arundinacea* L.), and to a lesser extent on orchardgrass and bentgrass.

**Control:** Reed canarygrass genotypes with good resistance to *D. catenaria* exist (Zeiders, 1976). These clones could be used in breeding programs designed to increase disease resistance.

**Literature:**

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Leafspot/Blotch caused by *Drechslera dactylidis*.

This disease may occur on orchardgrass from May to October in the northeastern and north central United States, and sometimes causes serious damage.

**Symptoms:** In the field, the disease appears as marginal, light brown, irregular leaf lesions up to 15 mm long and 5 mm wide. Scattered conidiophores develop on the necrotic tissue, and black perithecia of *Pleospora phaeocomes* form underneath the epidermis (Graham, 1955a). In artificial inoculations, young lesions develop as yellow to orange, round or oblong areas 1 to 4 mm in diameter. When infection is heavy, mature lesions may coalesce and envelop the entire leaf. The causal fungus is unique in that it regularly produces a certain proportion of curved, branched, or three-pointed spores in culture and on infected tissue (Zeiders, 1980). This characteristic is important in the laboratory diagnosis of the disease.

**Host Range:** *D. dactylidis* has a very narrow host range among forage grasses. Orchardgrass is the most important host.

**Control:** Disease severity can be reduced by crop ro-
tation, timely (early) cutting to prevent a build-up of inoculum, and development of resistant cultivars.

**Literature:**

**Drechslera Leaf Streak** caused by *Drechslera phlei* (Graham) Shoem.

This leaf streak disease of timothy was first observed in central Pennsylvania and subsequently was reported to occur throughout the northeastern United States (Graham, 1955b).

**Symptoms:** The disease is characterized by irregular light brown, necrotic streaks with conspicuous chlorotic borders. The necrotic areas, which are 1 to 5 mm wide, are often marginal and may extend the entire length of the leaf blade (Graham, 1955b). Often, lesions may coalesce, thereby causing a browning of most of the leaf. If the chlorotic symptom is lacking, the disease is difficult to distinguish from leaf streak. However, under moist conditions, the leaf streak disease is characterized by parallel rows of dark tufts of conidiophores of the causal fungus on infected leaves.

**Host Range:** The fungus is limited to timothy and other *Phleum* spp.

**Control:** Resistant cultivars offer a means of reducing severity of Drechslera leaf streak of timothy (Braverman, 1986).

**Literature:**

**Bipolaris Foot Rot, Leaf Blight, and Seedling Blight** caused by *Bipolaris sorokinianum* (sexual stage: *Cochliobolus sativus*).

The disease incitant causes a foot rot, leaf blight, and seedling blight on a wide range of grasses in North America. While the pathogen is of significant economic importance on wheat, barley, and oats, it is capable of causing moderate to severe damage on cool- and warm-season grasses.

**Symptoms:** The predominant symptom on grasses is either a leafspot or leaf blight. Lesions caused by *B. sorokinianum* are elongated, dark to purple, and measure 2 to 5 mm. Older lesions may become light-colored in the center. As disease severity increases with time, lesions often coalesce, causing foliage to become blighted. This reduces forage quality. The disease is most severe on switchgrass during the
warmest part of the summer.

**Host Range:** The fungus attacks a multitude of the Gramineae as noted by Sprague (1950).

**Control:** Resistant cultivars offer the best means of reducing the severity of *B. sorokinianum* (Braverman, 1967, 1986).

**Literature:**


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**Spot Blotch of Switchgrass** caused by *Bipolaris sorokinianum* (*Helminthosporium sativum*).

Spot blotch, causing moderate to severe damage, was the most prevalent disease on leaves of switchgrass in Pennsylvania during a 5-year study (Zeiders, 1984).

**Symptoms:** Lesions caused by *B. sorokinianum* are elongated (1 to 1.5 by 3 to 5 mm), dark to purple. Older lesions may become light-colored in the center. As disease severity increases, lesions often coalesce and leaves become blighted, reducing quality of the forage. The early symptoms of spot blotch, tiny dark elongated spots, begin to appear in mid to late June. The disease often becomes severe from mid-July through September.

**Etiology:** Spores (conidia) of *B. sorokinianum* are produced on dead infected leaf tissue and disseminated by air currents to new leaves, causing new infections. New disease lesions can be seen about 48 hours after inoculation. The severity of spot blotch is related to the duration of high relative humidity and leaf wetness.

**Control:** Resistant cultivars appear to be the best means of reducing the severity of spot blotch. Zeiders (1984) noted wide variability in reaction of 11 switchgrass varieties and strains to spot blotch. Damage ranged from mild to severe. The Ky-729 strain showed the most resistance, and three other strains were moderately resistant. These differences provide evidence of genetic variability for resistance to spot blotch.

**Literature:**

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**Eyespot** caused by *Heterosporium phlei* Gregory. Figure 18

Eyespot, a common foliar disease of timothy, was first described in New York State and since has been reported throughout North America, Europe, and Japan. The disease probably occurs wherever the crop is grown. Roberts et al. (1955) described the destructive nature of eyespot to timothy in New York. Sakuma and Marita (1961) reported that eyespot reduced leaf crude protein by 26 percent.

**Symptoms:** *Heterosporium phlei* occurs on both leaf surfaces as small, light-colored, oval lesions with pale grayish-tan centers surrounded by a narrow purple border, which eventually fades to brown. The disease may also be referred to as purple spot (O'Rourke, 1976). When lesions are abundant, the intervening tissue frequently becomes yellow; affected leaves turn brown and wither prematurely (Kreitlow et al., 1953).

**Etiology:** The fungus probably overwinters in dead infected leaf tissue. Although eyespot is more prevalent during the summer months, it may be found on green tissue during the spring and early autumn months. Primary infection in the spring probably occurs from conidia disseminated by wind and rain. However, it is difficult to find spores on diseased leaves in the field (Kreitlow et al., 1953). Conidia germinate over a wide temperature range, which may account for the occurrence of *H. phlei* on leaf tissue over a long growing season.

**Host Range:** Eyespot is confined to *Phleum* species. In Europe, it is found on timothy and *P. bertoloni* DC.; in North America the host range includes alpine timothy (*P. alpinum* L. (= *P. pratense*)), *P. phleoides* (L.) Karst., and *P. nodosum* L. as well.

**Control:** Resistant cultivars, noted by Smith (1970) and Hanson (1972), offer some control of *H. phlei*.

**Literature:**


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**STAGONOSPORA Diseases of Forage Grasses**

*Stagonospora* species cause diseases on a number of forage grasses. Important diseases are purple leafspot of orchardgrass and tawny blotch of reed canarygrass. These two diseases and a less important disease of smooth bromegrass are described in some detail.

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**Purple Leafspot of Orchardgrass caused by Stagonospora arenaria Sacc.**

Purple leafspot of orchardgrass occurs throughout the eastern United States. Lesions appear on leaves as soon as new growth begins and continue to develop in the summer and fall except during periods of prolonged hot, dry weather. The disease reaches its peak either shortly before or at time of heading. Leaves wither and turn brown when heavily attacked by the fungus, reducing the nutritive value of the forage. Infections progress rapidly in the fall and new lesions can be found until late November.

**Symptoms:** The lesions usually appear as small, somewhat elongate, blackish-brown to deep purple spots. When lesions are abundant they coalesce, causing the leaf to turn brown and die. Frequently the browning develops at the tip of a leaf or along the margin in long brown streaks. Small golden-brown bodies, the pycnidia of the fungus, develop within the dead areas of a leaf.

**Etiology:** Infection occurs from spores that overwinter in pycnidia in dead stems and leaves. Upon emerging from the pycnidia they are disseminated by spattering rain or in wind-blowed fragments of dead plants. Pycnidia usually form in rows in the dead parts of leaves.

**Host Range:** A list of susceptible species has been compiled by Sprague (1950).

**Control:** Breeding for resistance is a practical means of reducing disease severity. Although immune plants have not been found, resistance to purple leafspot can be effectively increased by inoculation and selection in the greenhouse (Zeiders et al., 1984).

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**Leafspot of Smooth Bromegrass caused by Stagonospora bromi A.L. Sm. & Ramsb.**

Leafspot caused by *S. bromi* does not achieve economic importance in all years, but it is reported to be a destructive parasite of smooth bromegrass and other species.

**Symptoms:** Leafspots on smooth bromegrass are dark brown, elongated, and often pointed at the ends. The inner portions become light tan as the lesion expands. Pycnidia develop within the infected tissue. Although similar in appearance to brown spot caused by *Drechslera bromi*, lesions are less angular in outline and may be distinguished by the light central area and the pycnidia in the infected tissue (Zeiders and Graham, 1962).

**Etiology:** The etiology of *S. bromi* is similar to that of *S. arenaria*.

**Host Range:** The reported hosts are smooth bromegrass and *Bromus* species (Sprague, 1950).

**Control:** In screening tests at the U.S. Regional Pasture Research Laboratory, selections of smooth bromegrass varied from very susceptible to resistant in reaction to *S. bromi* (Zeiders and Graham, 1962).

**Literature:**


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**Tawny Blotch caused by Stagonospora folicola.**

Tawny blotch is probably the most important disease of reed canarygrass in the temperate zones of the world. Reed canarygrass is often grown as a cover crop on areas that are spray-irrigated with effluent from sewage treatment plants.

**Symptoms:** Young lesions are small, elongated, and purple; as they enlarge they become dark brown to reddish-brown, usually with pointed ends and buff centers. If numerous, the lesions often coalesce to form large areas of dead tissue. Lesions are present
on infected plants throughout the growing season. Leaves, leaf sheaths, and culms may be attacked by *S. foliicola* (Zeiders, 1975).

**Etiology:** In the spring, pycnidia of *S. foliicola* develop on diseased leaves infected the previous year. The crumplent pycnidia are scattered over the dead leaves and are not confined to the diseased tissue. Spores are released by rupture or disintegration of the pycnidial wall. These spores comprise the inoculum that causes new infections in the spring. They are probably disseminated from plant to plant by rain and wind-blown fragments of dead plant parts.

**Host Range:** The fungus is confined to *Phalaris* spp.

**Control:** Disease severity can be significantly reduced by mowing the stand three times a year; preventing build-up of disease. Inoculation experiments and field evaluations have revealed strong resistance to tawny blotch in some genotypes. It should be possible to develop varieties with good resistance to this disease (Zeiders and Sherwood, 1977a, b).

**Literature:**


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**Anthracnose of Grasses**

caused by *Colletotrichum graminicola* (Ces.) Wils.

Anthracnose, one of the most common and widely distributed diseases of forage grasses, is particularly noticeable in mid-summer to early fall. Anthracnose occurs on practically all of the cultivated warm- and cool-season forage grasses in humid areas of the northern United States. Sudangrass is attacked in mid-summer at the height of vegetative growth. *C. graminicola* is primarily a high temperature organism (optimum 28°C), which accounts for its association with maturity of grass in mid-summer (Kreitlow et al., 1953). Bruehl and Dickson (1950) found some host specificity among isolates. In general, isolates from warm-season grasses, such as *Sorghum* spp., were more pathogenic at higher temperatures. If conditions are favorable for development of the disease, the fungus causes stunting, wilting, and sometimes death of seedlings. Although relatively few seedlings may be killed, the root system is usually damaged and yield is reduced, even though plants appear to recover as the season progresses. In older plants, the culm or leaf sheath is attacked, and the fungus may spread into the crown and roots. Perennial grasses, so infected, frequently die out in the second or third years. Death of plants occurs more rapidly in areas of low soil fertility. Early attacks cause a general reduction in vigor, shriveling of seed, premature ripening, or death of the plant (Kreitlow et al., 1953).

**Symptoms:** Lesions that develop on the sheath or stem are usually light tan with a darker border of red or brown. Black acervuli of the fungus usually develop within the bleached center of a lesion or on leaf blades of dead plants, particularly when moisture is plentiful.

**Etiology:** Infection occurs from diseased seed or from spores and mycelia that develop saprophytically on old crop residues. Infection in mature culms is common in the vicinity of nodes. After the pathogen is established and lesions develop, secondary spread occurs from spores or mycelia.

**Host Range:** A lengthy list of Gramineous hosts has been compiled by Sprague (1950).

**Control:** General control measures include crop rotation, particularly to avoid a sequence of closely related crops; timely cutting or grazing; maintaining soil fertility; plowing under all plant residues; and development of resistant varieties (Kreitlow et al., 1953).

**Literature:**


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**SEPTORIA LEAFSPOTS** of Forage Grasses

Numerous species of *Septoria* Fr. em. Sacc. cause leaf blotch and leafspots on grasses and cereals throughout the temperate and subtropical zones. These irregular blotches are straw-colored to brown, with dark brown to black pycnidia occurring in the older portions of the lesions. The blotches are often similar to those produced by *Stagonospora* or *Ascochyta*; morphological distinctions between the three genera on
the grasses are not clearly defined. *Septoria* species are generally more aggressively parasitic on grasses than are those of closely related genera. Infected grasses may be the source of *Septoria* inoculum from which cereal crops become infected.

In *Septoria*, pycnidia are subepidermal, slightly erumpent, and are formed in the older portions of the blottches or spots on the leaves, culms, and inflorescences of the host. The pycnidia are lens-shaped, brown to black, ostiolate, and parenchymatous.

The etiologies of the *Septoria* diseases discussed here are basically similar. Spores overwinter in pycnidia on dead leaves and are dispersed via wind or rain, and infect healthy plants. Germination will occur over a wide temperature range. Under moist conditions, spores are extruded in a gelatinous mass from the ostiole and are disseminated to adjacent healthy leaves by rain and wind-borne infected plant parts.

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**Leafspot of Bromus Species** caused by *Septoria bromi* Sacc.

*S. bromi* is widely distributed on *Bromus* species and may cause moderate to severe damage on smooth brome grass.

**Symptoms:** Leafspots are elongate to elliptical, tan to brown, with infected leaves often yellow, eventually becoming brown (Sprague, 1950). The disease occurs early in spring, decreases in prevalence during summer, and reappears again in autumn.

**Control:** Some reduction in disease incidence may be achieved by removing or plowing-under plant residues.

**Literature:**

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**Speckled Leaf Blotch** caused by *Septoria tritici* var. *lolicola* Sprague & A.G. Johnson.

Although *Septoria* species are common on many grasses and occur worldwide, damage caused by this pathogen is most severe on the ryegrasses.

**Symptoms:** The earliest symptoms usually appear as diffuse oval lesions along the leaf margins. Pycnidia are prominent and numerous within the lesions, usually aligned in rows parallel to the leaf veins. They are most numerous on the upper surface of the leaf (O’Rourke, 1976). Pycnidia are globose to sub-globose and are light brown.

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**Host Range:** The fungus is restricted to species of *Lolium*.

**Control:** There are no reports of cultivars with resistance to *S. tritici* var. *lolicola*.

**Literature:**

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**Leafspot/Blotch** caused by *Septoria bromi* (Sacc.) var. *phalarica* Sprague.

This disease is not as common or prevalent on reed canary grass as is tawny blotch, but has the potential to cause serious damage. The pathogen is confined almost entirely to reed canary grass.

**Symptoms:** The young lesions are narrow, water-soaked or dark gray spots up to 4 mm in length, surrounded by a yellow halo. Mature lesions are buff-colored, 2 to 4 mm long, and often confined between veins of the leaf. Lesions may coalesce to form large buff-colored necrotic areas. The disease may occur from May to October.

**Etiology:** Pycnidiospores, produced in globose or bulb-shaped pycnidia, are disseminated by wind and wind-driven rain to adjacent plants.

**Host Range:** The pathogen is confined to reed canary grass.

**Control:** Zeiders (1979) identified genotypes with moderate resistance to *S. bromi* var. *phalarica* that could be used in breeding resistance into a new cultivar.

**Literature:**

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**Ascochyta Leafspot** caused by *Ascochyta sorghi* Sacc. (Syn. A. graminicola Sacc.).

*A. sorghi* is probably the most widely distributed of several *Ascochyta* species that cause leafspot diseases on forage grasses.

**Symptoms:** *A. sorghi* attacks a wide range of the Gramineae and causes brownish-tan lesions, up to 20 mm in length, with brown to reddish margins on leaves and culms. Light brown lens-shaped-to-globose ostiolate pycnidia form in groups within the
leaf lesions and then become erumpent.

**Etiology:** The fungus overwinters as mycelium in diseased plant tissue. In late spring and early summer, globose pycnidia form in infected tissue. Pycnidiospores are extruded, disseminated by wind and wind-blown rain, and infect other plants.

**Host Range:** The host range of *A. sorghi* has been recorded by Sprague (1950).

**Control:** The tetraploid 'Sabrina,' *Lolium x hybridum* Hausskn., has resistance to *A. sorghi* (Braverman, 1986).

**Literature:**

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**Leafspot** caused by *Ascochyta brachypodii* (perfect state: *Didymella sp.*).

Ascochyta leafspot was the most important disease on three perennial warm-season grasses (big bluestem, little bluestem, and indiangrass) in Pennsylvania over a five-year period (Zeiders, 1982). The disease also occurs on these and several other species in the western United States.

**Symptoms:** The disease, caused by *A. brachypodii*, is characterized by reddish-brown elongated spots or blotches with tan centers, 2 to 6 by 1 to 1.5 mm in dimension, on leaves and leaf sheaths. In advanced stages, symptoms are sometimes streak-like.

**Etiology:** Symptoms of the disease caused by *A. brachypodii* usually appear in mid to late June. Infection increases in severity until about October 1 on grasses not cut or grazed. The fungus overwinters as mycelium in infected leaves. In late spring and summer, brown, globose, beaked pycnidia develop in this tissue. Spores (conidia) of *A. brachypodii* are produced in these fruiting bodies and then extruded through the pycnidial beak in a viscous mass. Other plants are infected by spores disseminated by wind and wind-blown rain. Zeiders (1982) found the severity of Ascochyta leafspot to be related to the duration of high relative humidity and leaf wetness in the growing area.

**Host Range:** Additional hosts of *A. brachypodii* include maize, sudangrass, and oats (Zeiders, 1982).

**Control:** The severity of the disease can be reduced by cutting or grazing the grasses at least twice per growing season to prevent disease build-up. Differences in susceptibility of big bluestem varieties to *A. brachypodii* in the field have been observed (Zeiders, 1982). In Pennsylvania, severity of leafspot on the cultivar NY-1145 was consistently less severe than on cultivars Kaw or Pawnee (Zeiders, 1982).

**Literature:**

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**Leafspot of Smooth Bromegrass** caused by *Selenophoma bromigena* (Sacc.) Sprague & A.G. Johnson. Figure 19

*Selenophoma* leafspot is an important disease that is general in distribution, appearing on smooth bromegrass wherever it is grown (Allison, 1945). Severe leaf infection may almost completely defoliate the plant. *S. donacis* (Pass.) Sprague & A.G. Johnson is an important pathogen of timothy.

**Symptoms:** Soon after new spring growth, chlorotic oblong lesions 8 to 15 mm in length appear on the lower leaves of infected plants. After about 7 days, the light-brown lesions have a light-red border and are dry at the center. After about 12 to 14 days, numerous pycnidia appear in these dried areas. The infected leaves turn yellow and often die. Almost total defoliation of severely infected plants is common. Ordinarily, infection is confined to localized spots on the leaves, but if the environment is cool and damp, infected areas may coalesce and cover large portions of the leaf surface. At such times the pathogen often spreads to the sheath, stem, rachis, panicle, and glumes; severe attacks stunt growth and may kill the plants.

**Etiology:** Presumably, the fungus overwinters as pycnidia in infected tissue. Pycnidiospores ooze from the pycnidium during the spring and are spread to adjacent plants by wind-driven rain. Penetration of the host tissues is direct.

**Host Range:** Smooth bromegrass, mountain bromegrass, fringed brome (*B. ciliatus* L.), Canada brome (*B. purgans* L.), and *B. arvensis* L. are hosts (Sprague, 1950).

**Control:** Smooth bromegrass plants resistant to *S. bromigena* are reported to be common; thus, selection for resistance and breeding offers a means of reducing disease severity. Specialized races of *S. bromigena*
have also been observed.

**Literature:**

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**Rhizoctonia Blight** caused by *Rhizoctonia solani*
Kuehn (perfect state: *Thanatephorus cucumeris* (Frank) Donk).

*Rhizoctonia solani*, a soil-borne organism, is particularly abundant in acid soils. The pathogen has a wide host range among the forage grasses and is severe on smooth bromegrass, orchardgrass, and tall fescue. It is important also in subtropical climates. The fungus may attack all parts of the plant—roots, crowns, leaves, and stems—but damage usually becomes most evident after stands are well established. The fungus causes damping-off and root rots of many grasses and is most destructive on seedlings or older weakened plants. In addition to causing damping-off and root and crown rots, *R. solani* causes a foliar blight that is extremely destructive to dense stands during hot, humid weather.

**Symptoms:** Large, irregular, tan-to-whitish blotches with narrow, dark brown borders form on leaves and leaf sheaths. The blotches often coalesce and girdle the stems. Under warm, moist conditions in dense stands, the fungus spreads rapidly by leaf-to-leaf contact, producing large patches of grayish matted dead tissue covered with a web of mycelium. Even so, the crowns of mature plants often survive.

**Etiology:** *R. solani* produces small, tan to brown sclerotia on dead infected tissues, enabling the fungus to survive for many years in the soil. The sexual stage of the fungus, *Thanatephorus cucumeris* (Frank) Donk, has been reported to occur on tall fescue as effuse white patches of mycelium, usually on the lower surface of the leaf (Luttrell, 1962).

**Host Range:** Sprague (1950) has compiled an extensive list of gramineous hosts of the fungus.

**Control:** The pathogen is soil-borne, attacks a wide range of hosts, and persists in the soil indefinitely. Timely cutting or grazing of the grass is effective in curtailing the spread of Rhizoctonia blight, since its development is retarded when exposed to direct sunlight and good air drainage. Crop rotation is ineffective. Parasitic races of *R. solani* exist, but attempts to identify germplasm for forage grasses with good field resistance have met with little success (Luttrell, 1962).

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**Literature:**

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**Take-all:** caused by *Gaumannomyces* (*Ophiobolus*) *graminis* (Sacc.) Arx & Oliver. Figure 20

*Gaumannomyces graminis*, the causal agent of the take-all and white diseases of cereals, parasitizes a large number of forage and turf grasses as well. Take-all disease is widely distributed in western Europe, Ireland, and Great Britain. In the United States, the disease occurs mainly in the Pacific Northwest (O'Rourke, 1976).

**Symptoms:** The fungus attacks the roots, crown, and basal culm tissues of cereals and grasses. Infection is not as prominent in the grasses. Under moist conditions leaf color fades and bleaching of leaves and culms follows. The main roots, crown, and culm (particularly the tissue between the leaf sheath and culm base), show a distinct rot and dark runner hyphae that form a mat of thick-walled coarse mycelia. Under dryer conditions, plant tillering is reduced and the mycelial mat is less pronounced.

**Etiology:** The pathogen is soil-borne in a rather direct association with culms and roots of grasses. Infection occurs from mycelia in the crop residue; the mycelia invade the roots, culm, and sheath. Slender infection hyphae grow from the runner hyphae into the root tissues as far as the stele (O'Rourke, 1976). Perithecia form on the dark fungal mats between the leaf sheaths and the culm base. In a moist environment, asci subsequently develop and mature ascospores are discharged and initiate new infection sites. Grasses are important economic hosts of *G. graminis*, as they provide the inoculum sources for the infection of cereal hosts. Each is required for the continued spread and survival of the pathogen.

**Host Range:** In Great Britain and Ireland, the fungus is prevalent on *Agrostis*, *Agropyron*, *Festuca*, *Lolium*, and *Poa* spp. (O'Rourke, 1976). While *G. graminis* readily attacks wheat, barley, and rye, oats are nearly immune. However, oats are very susceptible to *G. graminis* var. *avenae* Turner. Brooks (1965) found that pathogen survival was optimum on orchardgrass, red fescue, and annual and perennial ryegrasses. He also noted survival on *Arrhenatherum* sp. and *Agrostis* sp.

**Control:** Brooks (1965) noted that tall oatgrass was the least susceptible of 15 grasses when inoculated ar-
tificially with G. graminis, but the most susceptible when the inoculum source was G. graminis var. avenae. Nilsson (1969) reported that orchardgrass showed resistance to G. graminis var. avenae, but was susceptible to G. graminis. Perennial ryegrass was resistant to G. graminis. In general, specific resistance to take-all disease by economically important grasses will vary within a country and from country to country.

Nilsson (1969) noted that perennial ryegrass was resistant to take-all, but documented conflicting reports of resistance to the disease in other Lolium spp. However, Walker (1975) reviewed the take-all diseases of the Poaceae and indicated that Lolium spp. were resistant.

Nitrogen in ammonia compounds in a slightly acid soil appears to reduce the incidence of take-all disease (O’Rourke, 1976).

**Literature:**

**Red Thread** caused by Corticium fuciforme (Berk.) Wakef. Figure 21

Red thread, or pink patch, is common on turfgrass species in areas of low soil fertility. The disease is particularly noticeable on red fescue and perennial ryegrass.

**Symptoms:** The disease first appears as small irregular reddish or pinkish patches of infected plants; the infection may expand to several meters. The pinkish discoloration is due to (i) anthocyanins produced in affected grass foliage and (ii) to a pink, slimy mycelial growth. The fungus produces pinkstromata on dried-out leaves which, in turn, fade to a tan to reddish color.

**Etiology:** In dry weather, stromata break off from the diseased tissue. Stromata may survive under this condition up to two years, and can be wind-blown, disseminating the pathogen to initiate new areas of infection. Basidia are produced on infected host tissues and on the stromata. Fungal spread within a diseased area is by mycelial growth from plant to plant. According to Couch (1973), basidiospores are not important in the spread of this fungus. The disease is most evident in autumn, particularly during periods of low temperature and high humidity.

**Host Range:** In Britain, the disease is widespread on red fescue, Agrostis spp., perennial ryegrass, and annual meadowgrass (Poa annua L.) (O’Rourke, 1976).

**Control:** Red thread is prevalent in soils of low fertility, thus application of nitrogenous fertilizers reduces disease incidence (O’Rourke, 1976). However, excessive nitrogen fertilization may enhance Fusarium patch disease. Several cultivars of perennial ryegrass have been tested for resistance to C. fuciforme (Braverman, 1986).

**Literature:**

**Snow Molds** caused by Typhula incarnata Lasch. ex Fr., T. ishikariensis Imai, T. itoana Imai, Fusarium nivale (Fr.) Ces, and Sclerotinia borealis (Bubak & Vleugel) Kohn.

Several species of Typhula, Fusarium, and Sclerotinia are the causal agents of a ubiquitous disease in temperate, cool regions of the world. The disease is quite severe in regions where winters are cold and snow cover predominates. F. nivale usually requires a snow cover of at least 60 days; T. incarnata needs 90 days; T. ishikariensis requires about 120 days, and severe damage results with a snow cover in excess of 150 days. S. borealis needs at least 180 days' snow cover to cause extensive damage. The disease caused by F. nivale and T. incarnata can also occur without snow cover (Weibull, 1979b, 1983).

**Symptoms:** Fusarium patch caused by F. nivale (Microdochium nivale (Schaffn.) Booth) appears on snow-free areas as small, circular water-soaked patches, becoming yellow to orange-brown. The small patches often coalesce, and under moist conditions a fringe of pale pink mycelium is visible; this mycelium tends to mat down the aerial plant parts. The disease progresses under prolonged snow cover, with new infections showing an abundant aerial mycelium which may turn pink, orange, or beige if exposed to light.
At times, minute gelatinous masses of conidia may be seen on the matted leaves. The dead tissue, after drying, forms a thick pink mat, providing the name "pink snow mold." The pink color distinguishes this disease from other low-temperature maladies, such as those caused by *Typhula* spp. and *Sclerotinia* sp. (Weibull, 1979b).

*Typhula* blight or gray or speckled snow mold, caused by *T. incarnata*, is widespread and may severely damage grasses. Although fungal attacks will occur on grasses without snow cover, damage is most severe after at least 90 days of cover. The disease first appears as small patches, 2 to 5 cm in diameter, which at first light are yellowish-brown to a grayish-white. Under snow, the diseased patches coalesce to a considerable size. Initially, a white-grayish mycelium develops on the grass substrate. Pink to pinkish-orange followed by reddish-brown to dark brown sub-globose sclerotia develop on or within infected tissue (Weibull, 1979b).

Gray or speckled snow mold, also called *Typhula* blight, is caused by *T. ishikariensis* and is a major disease on overwintering grasses with prolonged snow cover. Much more virulent than *T. incarnatum* (Weibull, 1979b), *T. ishikariensis* becomes a major incitant of snow mold on grasses covered 150 days or longer. Symptoms of this snow mold are bleached patches of dead or dying plants sparsely covered with a grayish-white mycelium. The decaying tissue is speckled with numerous dark, globose to slightly flattened sclerotia.

A third causal agent of the *Typhula* speckled snow mold or snow scald is *T. itoana*. The fungus is prevalent on grasses snow-covered during winter and early spring. As the snow melts, those grasses attacked by the fungus appear as flattened mats of bleached dead foliage and mycelium. Numerous brown sclerotia develop on the dead leaves and grass stems. *T. itoana* is apparently the most widely distributed of the three incitants of *Typhula* snow molds.

*Sclerotinia* snow mold is also a major disease on overwintering grasses. It is restricted to those regions of extreme cold where snow cover lasts for at least 180 days. The disease causes severe damage to grasses. Symptoms first appear, after snow melt, as bleached patches of dead and/or dying plants. Diseased areas are covered with grayish-white mycelium. Elongated and flattened grayish-white sclerotia form on or within the infected tissue and blacken upon maturity.

**Etiology:** Fusarium patch disease may occur throughout the year, while pink snow mold occurs only under a snow cover or very shortly after a thaw. Both Fusarium patch and pink snow mold are favored by cool, moist conditions. The conidial stage of *F. nivale* is the primary inoculum source. The disease is favored by cool, moist weather and high relative humidity. Conidia and mycelia produced under these climatic conditions invade healthy tissue. Perithecia of *M. nivalis* are abundant on cereals, but rarely observed on grasses in the field and do not form in cultures of *F. nivale* isolated from grass. Chlamydo­spores are absent (Weibull, 1979b).

The etiologies of the three diseases caused by *Typhula* spp. are similar. Each fungus produces sclerotia that remain dormant on the soil surface during the summer months. As temperatures become cooler and moist conditions prevail, the sclerotia germinate and produce one to three sporophores that bear basidiospores. While the basidiospores may cause infections, the most infections are from mycelial growth into weakened tissues. Sclerotia buried in the soil may survive for several years.

*Sclerotinia borealis* produces sclerotia that are dormant during the summer months, and become a source of inoculum in autumn. Elongated ascii, having eight ascospores, develop during moderately low temperatures and high moisture. Ascospores discharge onto surrounding foliage and are the main inoculum source. Infection may also occur by direct mycelial growth in weakened host tissues. The sclerotia may survive burial in the soil for years.

**Host Range:** Snow molds occur on a wide variety of grasses and are particularly prevalent on *Agrostis* spp., *Festuca* spp., and *Poa* spp. During periods of prolonged snow cover, *Lolium* spp., timothy, and *Agropyron* spp. may be extensively damaged.

**Control:** Good management practices offer a measure of control for snow mold-susceptible grass species. Cutting low in autumn to reduce excessive top growth, avoiding heavy top dressings or winter mulches, and eliminating heavy or late nitrogen applications may be effective. Abundant late-season nitrogen contributes to a dense host growth and creates a microclimate favorable to fungal development. Good drainage is important and fungicide applications may be advisable.

Resistant cultivars offer a good measure of snow mold resistance in forage grasses (Abe and Matsumoto, 1981; Duich et al., 1972; Funk et al., 1969, 1973, 1974; Jamalainen, 1974; O’Rourke, 1976; Schmidt, 1976a, b; and summarized by Braverman, 1967, 1986).

**Literature:**


Disease resistance in cool season forage, range and turf grasses II. Bot. Rev. 52:1-112.


DISEASES INCITED BY VIRUSES

Viruses are submicroscopic infective entities that reproduce only in living cells. When purified, many viruses form crystals of characteristic sizes and shapes permitting classification. Dimensions of virus particles are measured in millimicrons; the particles may be rigid or flexuous rods, spheres, or pinwheels.

Symptoms of virus infections include a wide range of host reactions, some similar to those incited by bacteria or fungi. Two distinct symptoms commonly encountered in the Gramineae are a mosaic (or mottle) and a yellowing of leaf tissues. In the former, chlorophyll development is patchy, causing an unevenness in green coloring of the foliage and formation of a yellow and green mosaic or mottle pattern. In leaves that yellow, there is a nearly uniform reduction of chlorophyll, with little or no mosaic pattern. Other symptoms encountered in the Gramineae include: veinbanding and veinclearing, streak, necrosis, and tissue malformations. Quite often, virus-infected plants are stunted (Walker, 1970).

Several methods of virus transmission are found in the Gramineae: mechanical, seed, insects, mites, nematodes, and soil (Slykhuis, 1976).

Many viruses infecting the Gramineae, such as barley yellow dwarf virus, have a broad host range while the host range of others may be restricted to a few species (Rochow, 1970; Catherall, 1971b). With some viruses, the host range is dependent upon the strain of the virus (Catherall, 1971b).

Literature:

Ryegrass Mosaic. Figures 22, 26

Ryegrass mosaic virus (RMV) causes a light green to yellow mosaic on Lolium spp. and has been reported from North America and Europe (Slykhuis, 1972). In Great Britain, RMV is probably the most damaging disease of annual and perennial ryegrasses (Heard et al., 1974). The virus infects only members of the Poaceae and naturally occurs on annual and perennial ryegrasses, although several additional genera have been infected by mechanical inoculation.

Ryegrass mosaic virus is transmitted by the eriophyid mite, Acar tus hystricis (Neipa), and by manual sap inoculation to other species of the Poaceae. All instars of A. hystricis transmit RMV, but lose their infectivity within 24 hours after removal from a virus source (Slykhuis, 1972).

RMV is readily transmitted by sap inoculation in the ryegrasses. The virus is not seed- or soil-borne; nor has transmission through dodder (Cuscuta spp.) been reported. In the field, RMV is probably introduced and spread within the crop by mites and by grazing.

Symptoms: RMV is systemic and causes pale green streaks in ryegrass foliage, most evident in the upper leaves of the flowering stem. On older leaves, the streaks become yellow or brown and distinct. On annual ryegrass, the entire lamina occasionally becomes dark brown. In perennial ryegrass, the virus reduces tillering; in late stages of infection, the plants are also stunted. In general, annual ryegrass is more susceptible and more severely affected than perennial ryegrass.

Control: The severity of RMV in Great Britain and Europe has stimulated progress toward producing disease resistant cultivars in both species (Braverman, 1986). Resistance in the ryegrasses is polygenic (controlled by more than one gene). Interspecific crosses between perennial ryegrass and annual ryegrass combine the longevity of perennial ryegrass and the yield of annual ryegrass. Resulting lines are intermediate in reaction to RMV (Braverman, 1986).

Host Range: In addition to infections of annual and perennial ryegrass, RMV has been transmitted mechanically to orchardgrass, meadow fescue, annual meadowgrass, Kentucky bluegrass, rough bluegrass (Poa trivialis L.), smooth bromegrass, colonial bentgrass, Bromus arvensis L., and B. sterilis L. (Mulligan, 1960).

Literature:

Cocksfoot Mottle. Figure 23

Cocksfoot mottle virus (CfMV) causes a severe mottling and “dying out” of orchardgrass. The virus, not reported outside of central and southern England (Catherall, 1971a), infects only a few species in the Poaceae.

CfMV is transmitted by adults and larvae of a Chrysomelid beetle, Oulema melanopus L. The adults are the more efficient vectors, and may be infective up to two weeks after acquiring the virus from infected tissue. CfMV is readily transmitted by sap inoculation in the orchardgrasses (Catherall, 1971a).

The virus is not seed-borne; transmission by dodder has not been tested. CfMV may be spread in the field by mowing implements and (presumably) by grazing animals.

Symptoms: Symptoms of CfMV are similar to those of cocksfoot streak virus (CSV). The virus is systemic, and the most striking symptoms occur in spring and early summer. Young leaves display a yellow streaking or mottling which often becomes white or necrotic as the leaf matures. Infected leaves die prematurely; severely infected tussocks appear flattened, with young mottled tillers standing erect among a mass of streaked, yellow, and dying leaves. Infected plants occasionally flower, but set few viable seed. Although infected plants will form a few new tillers, the plants from these tillers are stunted and mottled and do not flower (Catherall, 1971b; Serjeant, 1964, 1967).

Control: Considerable variation in genotype response exists in orchardgrass. 'Conrad' and 'Cambria,' released by the Welsh Plant Breeding Station, Aberystwyth, Wales, are highly resistant. The Japanese cultivar Okamidorio is also CfMV resistant. One hybrid, D. mariana Borill x D. glomerata, has shown good CfMV resistance (Wilkins, 1977).

Host Range: The virus infects orchardgrass, other Dactylis spp., wheat, oats, and barley (Catherall, 1971a). Several additional species were identified as hosts in inoculation studies (Catherall et al., 1977).

Literature:

Literature.

Literature.

Literature.

Literature.

Literature.

Cocksfoot Streak.

Cocksfoot streak virus (CSV) is an aphid-stylet-borne (nonpersistent) virus which infects the Gramineae in Britain. It is also widespread in Germany, France, The Netherlands, and Sweden. According to Catherall (1971b), a similar (but possibly distinct) virus, orchardgrass mosaic, occurs in North America. While in Britain, CSV is restricted to Dactylis and Lolium spp., the virus has been transmitted to a multitude of grasses. Many are symptomless carriers.

CSV is transmitted primarily by five species of aphids: Myzus persicae (Sulz.); Macrosiphum euphorbiæ (Thos.); M. avenae (F.); Hyalopteroides humulis (Wlk.), and Metopolophium dirhodum (Wlk.). The aphids become infective shortly after feeding on infected hosts, but the virus persists for less than one hour. With difficulty, CSV can also be transmitted mechanically by sap inoculation. However, this mode of transmission is probably of minor consequence in spreading the virus.

Initial symptoms of CSV on orchardgrass and ryegrass are pale green or yellow streaks on the young leaves. As infection progresses, the streaks increase in intensity and eventually spread throughout the host. Infected plants are not stunted to any extent, but vegetative tiller production is usually reduced by 40 percent. Seed yield is slightly reduced (Catherall, 1971b).

Control: Orchardgrass cultivars resistant to the virus have not been found (Catherall, 1971b).

Host Range: In Britain, CSV infects only orchardgrass and ryegrass; in Germany it has been transmitted to several grasses, some of which are symptomless carriers (Catherall, 1971b).
Cocksfoot Mild Mosaic. Figure 25

Cocksfoot mild mosaic (CMM) has been reported in Germany and England and infects only species in the Gramineae. In orchardgrass, CMM causes a diffuse mild mosaic or mottle, and at times may induce a strong chlorotic streaking (Huth and Paul, 1972). Lolium perisicum Boiss. & Hohen ex Boiss. and foxtail millet are additional diagnostic hosts. Perennial ryegrass and meadow fescue, while not susceptible to the type strain of CMM, are susceptible to a different virus strain. CMM is transmitted by Myzus persicae, but transmission by seed or by dodder has not been reported.

CMM and Phleum mottle viruses have distinct host ranges, but are serologically related. CMM does not infect timothy, and Phleum mottle virus does not infect orchardgrass.

Literature:

Agropyron Mosaic.

Agropyron mosaic virus (AMV), which causes a mosaic in the leaves of many Gramineae, has been reported in the United States, Canada, and Europe. According to Slykhuis (1973), only species of the Gramineae have been found to be systemically infected, although the virus does produce local lesions in Chenopodium quinoa Willd., a dicotyledonous species.

AMV is transmitted by an eriophyid mite, Abacerus hystrich, and also is sap transmissible. The virus persists in the rhizomes of its respective hosts, but is not seed-borne. Transmission by dodder has not been reported. In the field, AMV is most likely spread by mowing implements and presumably by the trampling of virus-infected foliage by grazing animals.

Symptoms: Systemically infected species in the Gramineae develop a light green to yellow mosaic and striping on the foliage. Some stunting is evident.

Host Range: In North America, wheat appears to be highly susceptible to the virus. Several Agropyron species are moderately susceptible. They include A. elongatum (Host.) Beauv., beardless wheatgrass (A. interme (Scribn. & Smith) Rydb.), A. intermedium (Host.) Beauv., quackgrass, and fescue. Wild ryegrass and annual ryegrass are less susceptible. A majority of the reported hosts are found in Europe (Slykhuis, 1973).

Literature:

Bromegrass Mosaic.

Bromegrass mosaic virus (BMV) causes a mild mosaic in most species of Gramineae in the United States and Europe. The monocotyledonous host range is large, but the dicotyledonous host range consists of a few genera in five families (Bancroft, 1970). BMV survives in air-dried leaf tissue for one year or longer.

Schmidt et al. (1963) reported transmission of BMV by two nematodes — Xiphinema paraelongatum Atherr. (= X. diversicaudatum (Micoetzky) Thorne) and X. coxi Tarjan — in plants grown in a greenhouse, but field transmission of the virus by those nematodes has not been reported. The virus is also transmitted by animals (McKinney, 1953). BMV is not seed-borne (Lane, 1974). Its transmission by dodder has not been reported. Attempts to transmit BMV by selected insects have been unsuccessful (Lane, 1974).

Symptoms: Bromegrass mosaic virus causes a chlorotic mottling and striping, and brown necrotic stripes and blotches. Culms are shortened and the inflorescences are frequently sterile. Occasionally, excessive tillering may occur.

Although BMV is widely distributed on smooth bromegrass, the virus has a broad host range in the Gramineae. This is based on mechanical inoculations including other grasses. The disease is generally mild and of little economic importance. The virus appears to lack the appropriate insect vector (Gibson and Kenten, 1978).

Literature:

Barley Yellow Dwarf. Figure 24

Barley yellow dwarf virus (BYDV), which causes a stunting and chlorosis of a wide range of monocotyledonous species, is worldwide in distribution (Rochow, 1970; Panayotou, 1982). While BYDV is probably more widespread than any other virus infecting cereals, Doodson (1967) concludes that a large reservoir of the virus exists in England and Wales.

Barley yellow dwarf virus is transmitted by about 14 species of aphids, including Macrosiphum avenae, Rhopalosiphum maidis F., R. padi (L.), and Schizaphis graminum.

The virus is not seed-borne. However, BYDV has been recovered by aphids feeding on dodder established on infected barley (Rochow, 1970).

Symptoms: Barley yellow dwarf virus is systemic; however, some infected grasses show no symptoms. In perennial ryegrass, infected foliage is characterized by yellow or red leaf discolorations and/or stunting. Catherall (1963) and Doodson (1967) have emphasized the importance of infected perennial ryegrass as a reservoir of virus-infected material from which growing cereal crops could become infected by migrating aphids.

Control: Breeding for BYDV resistance in perennial ryegrass not only serves to develop a virus-free cultivar(s) but also to reduce a potential BYDV reservoir for future cereal crop infections. BYDV resistance in Lolium perenne is a complex problem (Catherall and Wilkins, 1977) due in part to the lack of symptom expression in susceptible cultivars and excessive tillering to compensate for plant dwarfing due to infection.

Host Range: The virus infects a wide range of monocotyledonous species (Rochow, 1970).

Literature:


Phleum Mottle.

Catherall (1966) proposed the name phleum mottle virus (PMV) for a Gramineae-infecting virus distinct from other grass-infecting viruses. The physical properties of PMV confirm that it belongs in the southern beak mosaic group of beetle-transmitted plant viruses (Walters, 1969). PMV is reported to occur only in central and southeastern Britain. PMV includes five distinct strains, as follows: PM (phleum mottle); HTM (holcus transitory mottle); FM (festuca mottle); CMM (cocksfoot mild mosaic), and BSM (brome stem leaf mottle).

PMV is transmitted by two species of cereal leaf beetle, Lema melanopa L. and L. lichens Weise. Adult beetles of either species are more efficient vectors than larvae. Some beetles retain the PMV virus for as long as two weeks after feeding on an infected plant. Attempts to transmit PMV with insects other than beetles, including aphids, were unsuccessful.

The virus is readily sap-transmissible and can be spread by farm machinery and presumably by grazing animals.

Symptoms: Initial infection appears as pale green or yellow streaks that form at the base of new growth and gradually extend to the leaf tip. Streaks coalesce to form a noticeable mottle, eventually covering the entire leaf (Catherall, 1970). Infection reduces the number of tillers and fresh green weight, but apparently does not affect plant height or number of inflorescences.

Control: Resistance to PMV in P. pratense cultivars has not been reported. Alpine timothy (P. alpinum L.) and P. commutatum Guad (= P. alpinum) do show resistance to PMV (Catherall, 1970).

Host Range: Suscepts include Agrostis spp., Bromus spp., orchardgrass, meadow fescue, Lolium persicum, reed canarygrass, timothy, and Poa spp.

Literature:
MINERAL DEFICIENCIES

This section describes characteristic effects on plants of various mineral disorders but does not attempt to be comprehensive. For a more extensive coverage of minerals and their role in plant growth and composition, the reader is referred to the volumes edited by C. Bould, E.J. Hewitt, and P. Needham titled "Diagnosis of Mineral Disorders in Plants," Volume 1, "Principles," and Volume 2, "Vegetables," published by Chemical Publishing, N.Y., 1984, from which the following information has been summarized.

Macronutrient Deficiencies

Calcium. In plant tissue, the concentration of calcium is important to growing points and young leaves and is usually related to the distribution of specific symptoms, but does not entirely explain the appearance of others. In cereals and grasses, emerging young leaves remain trapped in subtending leaves. Leaves which have emerged remain rolled, chlorotic, and have circular constrictions a few centimeters behind the apex. The distal portion wilts and withers. Death of the stem apex occurs in calcium-deficient plants, but multiple apical shoots or axillary nodal shoots may follow to fill the void.

Magnesium. Magnesium is very important in the makeup of chlorophyll. Therefore, magnesium deficiency is most frequently indicated first by the loss of chlorophyll. As a rule, the chlorosis usually appears first in the oldest leaves and is progressive. In several species, the chlorosis is generally interveinal within a persistent green margin of the leaf.

Symptoms of magnesium deficiency may be difficult to distinguish from the symptoms of potassium deficiencies at certain stages of plant growth. The principal distinction is that magnesium deficiency affects first the oldest leaves, while potassium deficiency usually is first noticed in younger leaves.

Nitrogen. Nitrogen-deficient plants are much smaller than normal, but the root growth can be extensive and root length may increase to compensate for the absence of N. The angle between petioles and stems is more acute. Tilling is suppressed, bud production or expansion is decreased, and flowering is delayed. Bud dormancy can be prolonged. Foliage is pale green and leaf senescence is accelerated. Depending upon species, leaves can develop purple, red, or orange anthocyanin tints in addition to the yellowishness caused by the loss of chlorophyll. Interveinal areas and older leaves are first to express symptoms. Leaf bases and stems become red-purple.

Phosphorus. Symptoms of phosphorus deficiency frequently resemble those of nitrogen deficiency. There is a diminutive or spindly habit, suppression of tilling, acute leaf angles, decreased size and numbers of flowers, prolonged dormancy, and early senescence. The leaves lack luster; leaf color changes, but may be either paler or darker than normal. Again, depending on species, leaf color may range from deep purple to red, or be absent.

Potassium. Potassium deficiency frequently appears as short telescoped shoots, caused by a shortening of stem internodes. Apical dominance of the viable terminal bud is sometimes suppressed, causing excessive basal shoots or tillers. Severe deficiency causes death of the terminal bud and a typical dieback. Strong light intensity accentuates and weak light often reduces or eliminates these conditions. Leaf scorch, preceded by irregular marginal or interveinal chlorosis, appears first in the oldest leaves. Scorching may be pale brown to almost black. Chlorosis almost always occurs and first appears in oldest leaves, which often curve downwards or become convex.

Sulphur. Decreased leaf size, red or purple pigmentation, and general chlorosis — symptoms of sulphur deficiency — resemble those of nitrogen. However, there are important differences. Usually young leaves are more sensitive to sulphur deficiency than older ones. In sulphur deficient soils, new leaves are frequently uniform golden yellow and stiff and erect.

Micronutrient Deficiencies

Chlorine. Little has been reported in the literature as to the effect of chlorine deficiency on plants in the Gramineae. Chlorine deficiency has been reported to limit growth and cause wilt, chlorosis, and prominent raised veins and clubbed tips of roots in dicotyledons. Deficiency will keep barley leaves rolled, as does copper deficiency.

Copper. In many plant species, the young leaves are most severely affected. Leaves are often rolled or curled, and may be coiled in a spiral which may reverse direction along its length. These leaves are often white. The emerging leaves may be trapped in the subtending leaf, producing symptoms known as
white tip or wither tip. Floral meristems are sensitive to copper deficiency. Head formation is suppressed, and the grains are shrevled or the glumes do not fill. Often the pollen cells are sterile.

Iron. Iron deficiency is first apparent as chlorosis of rapidly expanding leaves. Chlorosis is usually interveinal and produces a contrasting tramline effect in the leaves. The glumes may be more chlorotic than the flag leaf. In cereals and grasses, bleached or brown lesions develop more frequently in the interveinal areas and leaves collapse transversely.

Manganese. A variety of symptoms (including some form of chlorosis) are produced by manganese deficiency. Older leaves are usually affected first. Chlorosis is usually interveinal and produces a bold pattern of dark green major veins which contrasts with the fine reticulate pattern observed with iron deficiency. Manganese deficiency is also distinguished from iron deficiency symptoms in leaves by the appearance of varied but characteristic necrotic spotting or lesions. In the Gramineae, these lesions vary in appearance from dark brown spots along interveinal areas to chlorotic beading. In oats, the necrotic lesions are elongated, generally ivory to pale brown with “blue-green or grey” halo areas, and often coalesce into broad lesions that may collapse leaves transversely.
ICE AND WATER DAMAGE

Ice and water damage prevail in areas with alternate freezing and thawing and in maritime locales of prolonged high humidity. Such areas may be characterized by poor soil drainage or the soil may be frozen, thereby preventing or substantially reducing water flow. Damage increases during prolonged ice cover.

**Symptoms:** Small differences in topography influence the extent and severity of damage. The grass, usually green after initial ice melt in the spring, soon becomes dark brown and subsequently light brown upon drying. At this stage, the crowns and roots have been damaged and the entire plant begins to rot (Weibull, 1979c). Species forming rhizomes or stolons may regenerate later in the growing season to fill in killed patches. While ice and water damage may affect any grass species, cultivars differ in susceptibility. Kentucky bluegrass is tolerant and perennial ryegrass is sensitive, for example.

**Literature:**
BIBLIOGRAPHY


———, C.I. Kado, and D.R. Sumner. 1975. Synonymy of \textit{Pseudomonas avenae} Manns 1905 and
Morphological definitions are based in part on Kreitlow, J.W., et al., 1953, and O'Rourke, C.J., 1976.

**ABIOTIC** [A disease] not caused by a biological agent.

**ACERVULUS** (i). A small, open fruiting body that has ruptured the host epidermis and consists of a mass of hyphae bearing conidiophores and conidia.

**AECIOSPORE**. A spore formed in chains within the aecium of a rust.

**AECIUM** (A). A cup-shaped fruiting structure characteristic of the rusts in which aeciospores are borne.

**ALTERNATE HOST**. Either of two unrelated plant species necessary for certain rust fungi to complete their life cycle.

**AMPHIGENOUS**. Making growth all around or on two sides.

**APOTHECIUM** (A). An open cup or saucer-shaped fruiting body whose concave surface is lined with asci.

**AsaGERous**. Having asci.

**AscocARP**. The ascus-bearing structure of the Ascomycetes.

**AscosPORE**. One of normally eight spores borne in an ascus.

**Ascus** (I). A sac-like structure in which normally eight ascospores are formed as a result of the sexual process.

**ASEXUAL REPRODUCTION** Reproduction without fusion of gametes.

**AUTOECIOUS**. Referring to a parasite which completes its life cycle on one host.

**BACTERIUM** (A). A single-celled microscopic organism lacking a well-defined nucleus or nuclear membrane.

**BASIDIOSPORE**. A spore produced at the apex of a basidium.

**BASIDIUM**. A fruiting structure which bears the basidiospores.

**BIOTIC** [A disease] caused by a biological agent.

**BLIND SEED**. Sterile seed.

**CHLAMYDOSPORE**. A thick-walled asexual resting body formed by the rounding up of a mycelial segment.

**CHLOROTIC**. Deficient in chlorophyll.

**CLEISTOTHECIUM** (A). A fungal fruiting body, containing asci, which has no specific opening (e.g., in Erysiphaceae).

**CONIDIUM** (A). An asexual spore.

**CONIDIOPHORE**. A specialized branch of the mycelium bearing cells from which conidia are borne.

**CULM**. The stem of a grass.

**CUTICLE**. The outermost layer of leaf tissue.

**DISEASE**. An interaction between a causal agent and host which alters the morphological and physiological development of the host.

**DISEASE DEVELOPMENT**. The sequence of events from time of infection to symptom expression.

**DISEASE INCITANT**. An abiotic or biotic entity which may cause disease.

**ENDEMIC**. The occurrence of a disease from year to year in moderate to severe form.

**EPiphytic**. A large-scale disease outbreak in plants.

**FORMA (AE) SPECIALIS (ES)**. A subdivision within a species, distinguished by physiological traits rather than by morphological characteristics.

**GERMINATION**. The process by which a hypha emerges from a spore.

**GUTTATION**. The process of the escape of liquid water from uninjured plants.

**HAUSTORIUM** (A). A specialized mycelial branch, especially one within a living cell of a host.

**HETEROECIOUS**. Referring to a parasite that completes its life cycle on unrelated hosts.

**HOST PLANT**. A plant morphologically and physiologically altered by a disease incitant.

**HOST RANGE**. Those plant species known to be susceptible to a pathogenic organism.

**HYDATHEOD**E. A pore-like structure in a plant leaf through which guttation occurs.

**HYPHA** (AE). A single strand or strands of mycelium.

**INFECTION**. The establishment of a disease incitant within a host.

**LESION**. An area of tissue showing disease symptoms.

**LIFE CYCLE**. A sequential series of forms and relationships assumed by an organism from a primary stage to a resumption of that stage.

**MACROCONIDIUM** (A). The larger conidium of a fungus which also has microconidia.

**MICROCONIDIUM** (A). The smaller conidium of a fungus which also has macroconidia.

**MORPHOLOGY**. A study dealing with the form and structure of organisms.

**MYCELium** (A). The vegetative body of the fungus comprising a mass of hyphae.

**NECROTIC**. Dead or dying.

**OBLIGATE PARASITE**. An organism that grows only on its host and which usually cannot be cultured on artificial media.

**OSTIOLE**. An opening from which spores extrude from an ascigerous or pycnial fruiting body.

**PARASITE**. An organism which derives its nourish-
ment from another organism, the host.
**PATHOGEN.** A disease-causing organism.
**PATHOVAR.** A specialized variety of a pathogenic organism based on host range or its characteristic growth on a specific culture medium.
**PENETRATION.** An initial invasion of a host by a disease incitant.
**PERITHECIUM (A).** A round-oval, flask-shaped fruiting structure containing asci and an apical aperture.
**PHYSIOLOGIC RACE.** A pathogen similar in morphological characteristics but differing in ability to parasitize certain varieties of the host.
**POLYGENIC.** Having more than one gene.
**PRIMARY INOCULUM.** The initial infective disease-inciting agent (pathogen) causing infection in the host plant.
**PYCNIDIUM (A).** A small globose or flask-shaped fruiting body containing asexual spores.
**PYCNIDIOSPORE(s).** A spore or spores developed in a pycnidium.
**SAPROPHYTE.** An organism which derives its nourishment from dead organic matter.
**SCLEROTIUM (A).** A firm-bodied resting structure formed by certain fungi consisting of a mass of hyphae surrounded by a darker outer ring.
**SECONDARY INOCULUM.** Spores or infective bodies produced after the host has been colonized.
**SECONDARY INFECTION.** An infection resulting from inoculum produced by the pathogen during primary infection or during the production of secondary inoculum.
**SETA (AE).** A small, slender, usually rigid bristle or hair.
**SEXUAL REPRODUCTION.** Reproduction requiring nuclear fusion and meiosis.
**SORUS (A).** A spore-containing body of rusts and smuts which become exposed at maturity upon rupturing the epidermis of the host.
**SPORE.** A specialized cell(s) adapted for dissemination and capable of germinating to perpetuate the species.
**SPORIDIUM (A).** One of several spores borne on a specialized hypha (promycelium) which develop from teliospores of rusts and smuts.
**SPORDOCHIUM.** A compact conidial body; mass of sporophores.
**STRAIN.** An organism with similar morphologic characteristics to another but with different physiologic characters.
**STROMATIC BODY (STROMA).** A mass of vegetative hyphae in or on which fruiting structures develop.
**SUBSTRATE.** The base upon which an organism derives its nourishment.
**SYMPTOM.** The expression by the host resulting from the host-pathogen disease-incitant interaction.
**SYSTEMIC.** Spread throughout the host tissues; not localized or confined within specific boundaries.
**TELIOSPORE.** A spore produced by rusts and smuts which germinates to form a hypha (promycelium) on which sporidia are abstricted.
**TELIUM (A).** A spore-bearing body or sorus in which teliospores are produced.
**TUSSOCK.** A compact tuft (clump) of a grass.
**UREDIIUM (A).** A spore-bearing body or sorus in which urediospores are produced.
**UREDIOSPORE (s).** A spore characteristic of the rust fungi capable of initiating secondary infection by reinfecting the same host.
**VECTOR.** A living organism capable of transmitting a disease incitant.
**VIRUS.** A submicroscopic infective entity which can reproduce only in living cells.
**YELLOWS.** A foliage condition caused by destruction of the chlorophyll.
DISEASES INDEXED BY HOST

**Bentgrass and Red Top**
- Anthracnose 23
- Ascochyta leafspot (A. sorghi) 24
- Bipolaris foot rot 20
- Blister smuts (leafspot smuts) 12
- Cocksfoot mottle 31
- Covered smuts (kernel smuts) 12
- Crown rust 8
- Drechslera leafspot 20
- Ergot 14
- Kernel smuts (covered smuts) 12
- Leaf rust 10
- Leafspot smuts (blister smuts) 12
- Mastigosporium leaf fleck 12
- Phleum mottle 33
- Powdery mildew 13
- Red thread 27
- Rhizoctonia blight 26
- Stem rust (black stem rust) 8
- Stripe rust (yellow rust) 9
- Take-all 26
- Uromyces leaf rust 10

**Crested wheatgrass**
- Yellow leaf rust 9

**Agropyron species**
- Agropyron mosaic 32
- Anthracnose 23
- Ascochyta leafspot (A. sorghi) 24
- Bacterial brown stripe 3
- Bipolaris foot rot 20
- Brown spot/halo blight 5
- Crown rust 8
- Ergot 14
- Halo blight/brown spot 5
- Head smuts (loose smuts) 12
- Loose smuts (head smuts) 12
- Powdery mildew 13
- Rhizoctonia blight 26
- Snow molds 27
- Stem rust (black stem rust) 8
- Stripe rust (yellow rust) 9
- Uromyces leaf rust 10

**Bluegrass**
- Agropyron mosaic 32
- Anthracnose 23
- Ascochyta leafspot (A. sorghi) 24
- Bipolaris foot rot 20
- Brown spot/halo blight 5
- Crown rust 8
- Ergot 14
- Flag smut 11
- Leaf rust 10
- Leaf spot (Drechslera catenaria) 19
- Leaf spot (Drechslera dactylidis) 19
- Leaf streak (Scolecotrichum) 15
- Mastigosporium leaf fleck 16
- Net blotch 18
- Phleum mottle 33
- Powdery mildew 13
- Red thread (red fescue) 27
- Rhizoctonia blight 26
- Ryegrass mosaic 30
- Snow molds 27
- Stem rust (black stem rust) 8
- Stripe rust (yellow rust) 9
- Take-all (red fescue) 26
- Uromyces leaf rust 10
- Yellow leaf rust 9

**Orchardgrass**
- Anthracnose 23
- Ascochyta leafspot (A. sorghi) 24
- Bipolaris foot rot 20
- Blind seed 14
- Brown blight 19
- Cocksfoot mild mosaic 32
- Cocksfoot mottle 31
- Cocksfoot streak 31
- Ergot 14
- Flag smut 11
- Leaf blight (Rhynchosporium) 16
- Leaf scald 16
- Leaf spot (Drechslera catenaria) 19
- Leaf spot/blotch (Drechslera dactylidis) 19
- Leaf streak (Rhynchosporium) 16
- Leaf streak (Scolecotrichum) 15
- Mastigosporium leaf fleck 16
- Powdery mildew 13
- Phleum mottle 33
- Purple leafspot 22
- Rhizoctonia blight 26
- Ryegrass mosaic 30
- Snow molds 27
- Stem rust (black stem rust) 8
- Stripe rust (yellow rust) 9
- Stripe smut 11
- Translucent leaf stripe 6
- Uromyces leaf rust 10
- Yellow leaf rust 9

**Fescues**
- Agropyron mosaic (red fescue) 32
- Anthracnose 23
- Ascochyta leafspot (A. sorghi) 24
- Bacterial brown stripe 3
- Bipolaris foot rot 20
- Blister smuts (leafspot smuts) 12
- Brown blight (meadow fescue) 19
- Brown spot/halo blight 5
- Crown rust 8
- Ergot 14
- Halo blight/brown spot 5
- Leaf blight (Rhynchosporium) 16
- Leaf rust 10
- Leaf scald 16
- Leaf spot (Drechslera dactylidis) 19
- Leaf streak (Rhynchosporium) 16
- Leaf streak (Scolecotrichum) 15
- Mastigosporium leaf fleck 16
- Powdery mildew 13
- Phleum mottle 33
- Purple leafspot 22
- Rhizoctonia blight 26
- Ryegrass mosaic 30
- Snow molds 27
- Stem rust (black stem rust) 8
- Stripe rust (yellow rust) 9
- Stripe smut 11
- Translucent leaf stripe 6
- Uromyces leaf rust 10
- Yellow leaf rust 9

Yellow slime 2
Reed canary grass
Anthracnose 23
Crown rust 8
Ergot 14
Drechslera leaf spot (D. catenaria) 19
Leaf spot blotch (Septoria bromi) 24
Phleum mottle 33
Powdery mildew 13
Rhizoctonia blight 26
Stripe smut 11
Tawny blotch 22

**Phalaris spp.**
Anthracnose 23
Ascochyta leaf spot 24
Crown rust 8
Ergot 14
Rhizoctonia blight 26
Tawny blotch 22

**Annual rye grass**
Bacterial wilt 5
Take-all 26

**Perennial rye grass**
Red thread 27

**Annual and perennial ryegrasses**
Agropyron mosaic 32
Anthracnose 23
Bacterial brown stripe 3
Barley yellow dwarf 33
Bipolaris foot rot 20
Blind seed 14
Brome grass mosaic 32
Brown blotch 19
Brown spot/halo blight 5
Cocksfoot streak 31
Crown rust 8
Ergot 14
Halo blight/brown spot 5
Leaf blotch (Rhynchosporium) 16
Leaf scald 16
Leaf streak (Rhynchosporium) 16
Rhizoctonia blight 26
Selenophoma leaf spot (S. bromigena) 25
Septoria leaf spot (S. bromi) 24
Stagonospora leaf spot (S. bromi) 22
Translucent leaf stripe 6

**Bromus spp.**
Ascochyta leaf spot (A. sorghi) 24
Bacterial brown stripe 3
Bipolaris foot rot 20
Brome grass mosaic 32
Brown leaf spot 18
Brown spot/halo blight 5
Cocksfoot mottle 31
Crown rust 8
Ergot 14
Flag smut 11
Halo blight/brown spot 5
Leaf blotch (Rhynchosporium) 16
Leaf scald 16
Leaf streak (Rhynchosporium) 16
Rhizoctonia blight 26
Selenophoma leaf spot (S. bromigena) 25
Septoria leaf spot (S. bromi) 24
Stagonospora leaf spot (S. bromi) 22
Translucent leaf stripe 6

**Bromus spp.**
Ascochyta leaf spot (A. sorghi) 24
Bacterial brown stripe 3
Bipolaris foot rot 20
Brome grass mosaic 32
Brown leaf spot 18
Brown spot/halo blight 5
Cocksfoot mottle 31
Crown rust 8
Ergot 14
Flag smut 11
Halo blight/brown spot 5
Leaf blotch (Rhynchosporium) 16
Leaf scald 16
Leaf streak (Rhynchosporium) 16
Rhizoctonia blight 26
Selenophoma leaf spot (S. bromigena) 25
Septoria leaf spot (S. bromi) 24
Stagonospora leaf spot (S. bromi) 22
Translucent leaf stripe 6

**Sorghum**
Bacterial stripe 3
Bacterial brown stripe 3
Bacterial leaf blight 4
Powdery mildew 13

**Sudangrass**
Anthracnose 23
Ascochyta leaf spot (A. sorghi) 24
Bacterial stripe 3
Bacterial leaf blight 4
Ergot 14
Powdery mildew 13

**Tall oat grass**
Bacterial brown stripe 3
Barley yellow dwarf 33
Bipolaris foot rot 20
Blackish-brown stripe 5
Brome grass mosaic 32
Brown leaf spot 18
Brown spot/halo blight 5
Ergot 14
Flag smut 11
Halo blight/brown spot 5
Leaf blotch (Rhynchosporium) 16
Leaf scald 16
Leaf streak (Rhynchosporium) 16
Rhizoctonia blight 26
Selenophoma leaf spot (S. bromigena) 25
Septoria leaf spot (S. bromi) 24
Stagonospora leaf spot (S. bromi) 22
Translucent leaf stripe 6

**Tall oat grass**
Bacterial brown stripe 3
Barley yellow dwarf 33
Bipolaris foot rot 20
Blackish-brown stripe 5
Brome grass mosaic 32
Brown leaf spot 18
Brown spot/halo blight 5
Ergot 14
Flag smut 11
Halo blight/brown spot 5
Leaf blotch (Rhynchosporium) 16
Leaf scald 16
Leaf streak (Rhynchosporium) 16
Rhizoctonia blight 26
Selenophoma leaf spot (S. bromigena) 25
Septoria leaf spot (S. bromi) 24
Stagonospora leaf spot (S. bromi) 22
Translucent leaf stripe 6

**Wild ryegrass**
Agropyron mosaic 32
Anthracnose 23
Ascochyta leaf spot (A. sorghi) 24
Bacterial brown stripe 3
Bipolaris foot rot 20
Cocksfoot mottle 31
Crown rust 8
Ergot 14
Flag smut 11
Halo blight/brown spot 5
Leaf blotch (Rhynchosporium) 16
Leaf scald 16
Leaf streak (Rhynchosporium) 16
Rhizoctonia blight 26
Selenophoma leaf spot (S. bromigena) 25
Septoria leaf spot (S. bromi) 24
Stagonospora leaf spot (S. bromi) 22
Translucent leaf stripe 6

**Wild ryegrass**
Agropyron mosaic 32
Anthracnose 23
Ascochyta leaf spot (A. sorghi) 24
Bacterial brown stripe 3
Bipolaris foot rot 20
Cocksfoot mottle 31
Crown rust 8
Ergot 14
Flag smut 11
Halo blight/brown spot 5
Leaf blotch (Rhynchosporium) 16
Leaf scald 16
Leaf streak (Rhynchosporium) 16
Rhizoctonia blight 26
Selenophoma leaf spot (S. bromigena) 25
Septoria leaf spot (S. bromi) 24
Stagonospora leaf spot (S. bromi) 22
Translucent leaf stripe 6

**Warm-season grasses**

**Big bluestem**
Ascochyta leaf spot (A. sorghi) 24
Bacterial brown stripe 3
Bacterial leaf blight 4
Powdery mildew 13

**Big bluestem**
Ascochyta leaf spot (A. sorghi) 24
Bacterial brown stripe 3
Bacterial leaf blight 4
Powdery mildew 13

**Warm-season grasses**

**Big bluestem**
Ascochyta leaf spot (A. sorghi) 24
Bacterial brown stripe 3
Bacterial leaf blight 4
Powdery mildew 13

**Warm-season grasses**

**Big bluestem**
Ascochyta leaf spot (A. sorghi) 24
Bacterial brown stripe 3
Bacterial leaf blight 4
Powdery mildew 13

**Warm-season grasses**
**Little bluestem**
Ascochyta leafspot (*A. sorghi*) 24

**Switchgrass**
Spot blotch 21

**Indiangrass**
Ascochyta leafspot (*A. sorghi*) 24
ILLUSTRATIONS

Fig. 1. Cells of Xanthomonas campestris pv. graminis exuding from the vascular system of annual ryegrass (Lolium multiflorum Lam.).

Fig. 2. Yellow slime, Corynebacterium rhizohni, on orchardgrass.

Fig. 3. Bacterial stripe, Pseudomonas andropogonis, on sudangrass.

Fig. 4. Halo blight, Pseudomonas avenae, on oats.

Fig. 5. Brown stripe, Pseudomonas avenae, on mountain bromegrass.

Fig. 6. Halo blight, Pseudomonas syringae pv. coronafaciens, on mountain bromegrass.

Fig. 7. Halo blight, Pseudomonas syringae pv. coronafaciens, on annual ryegrass (early spring symptoms).

Fig. 8. Halo blight, Pseudomonas syringae pv. coronafaciens, on annual ryegrass (late summer symptoms).

Fig. 9. Bacterial wilt, Xanthomonas campestris pv. graminis, on annual ryegrass.

Fig. 10. Crown rust, Puccinia coronata, on perennial ryegrass.

Fig. 11. Stem rust, Puccinia graminis, on perennial ryegrass.

Fig. 12. Stem rust, Puccinia poae, on Kentucky bluegrass.

Fig. 13. Powdery mildew, Erysiphe graminis, on orchardgrass.

Fig. 14. Ergot, Claviceps purpurea, on orchardgrass.

Fig. 15. Mästigospórium leaf fleck, Mastigospórium spp., on orchardgrass.

Fig. 16. Brown leafspot, Pyrenophora tritici, on smooth bromegrass.

Fig. 17. Net blotch, Drechslera dictyoides, on annual ryegrass.

Fig. 18. Eyespot, Heterosporium pâlici, on timothy.

Fig. 19. Selenophoma leafspot, Selenophoma donatus, on timothy.

Fig. 20. Take-all, Gaëumannomyces graminis var. avenae, on perennial ryegrass.

Fig. 21. Red thread, Corticium fasciforme, on perennial ryegrass.

Fig. 22. Ryegrass mosaic virus on annual ryegrass, mild strain.

Fig. 23. Cocksfoot mottle virus on orchardgrass.

Fig. 24. Barley yellow dwarf virus on annual ryegrass (yellowing and dwarfing).

Fig. 25. Cocksfoot mild mosaic virus on orchardgrass.

Fig. 26. Ryegrass mosaic virus on annual ryegrass (severe strain).
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Fig. 15. Mastigosporium leaf fleck, *Mastigosporium* spp., on orchardgrass.
Fig. 16. Brown leafspot, *Pyrenaphora bromi*, on smooth bromegrass.

Fig. 17. Net blotch, *Drechslera dictyoïdes*, on annual ryegrass.
Fig. 18. Eyespot, *Heterosporium phlei*, on timothy.

Fig. 19. Selenophoma leafspot, *Selenophoma donacis*, on timothy.
Fig. 20. Take-all, *Gaumannomyces graminis* var. *avenae*, on perennial ryegrass.

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