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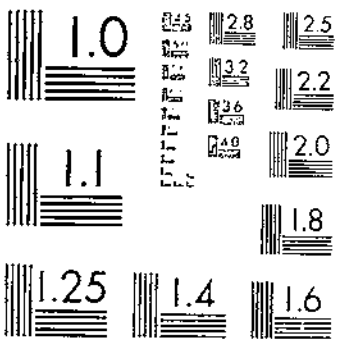

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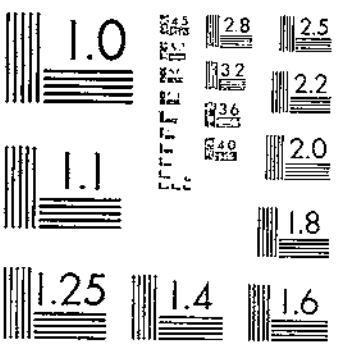

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CERCOSPORELLA FOOT ROT OF WINTER CEREALS

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[The Bureau of Plant Industry in cooperation with the Oregon, Washington, and Idaho Agricultural Experiment Stations]

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INTRODUCTION

Cercosporella foot rot of winter cereals, also known as the Columbia Basin foot rot, is a serious economic problem in certain prairie sections of Washington, Oregon, and Idaho (*28*).[2] In a recent report (*29*) it was shown that the disease is caused by *Cercosporella herpotrichoides* Fron. It is the purpose of this bulletin to summarize the results of investigations on this disease.

REVIEW OF LITERATURE

Cercosporella herpotrichoides Fron was named in 1912 from material gathered in northern France 3 years previously. Fron (*13*) considered that the fungus was the conidial stage of *Leptosphaeria herpotrichoides* DeNot. In 1914 Foëx (*5*) found conidia of *C. herpotrichoides* on wheat. In referring to Fron's belief that *C. herpotrichoides*

[1] The writers are indebted to A. G. Johnson, principal pathologist, Division of Cereal Crops and Diseases, Bureau of Plant Industry, and to H. P. Barss, formerly plant pathologist in charge of botany and plant pathology, Oregon Agricultural Experiment Station, for criticism of the manuscript. The field and station staffs of plant pathology, botany, and agronomy of the three experiment stations have rendered invaluable aid. Acknowledgments are due W. S. Nelson, of the Wasco County Chamber of Commerce, The Dalles, Oreg., for aid in the investigations.

[2] Italic numbers in parentheses refer to Literature Cited, p. 23.

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was connected with *L. herpotrichoides*, Foëx noted that this is not definitely known.

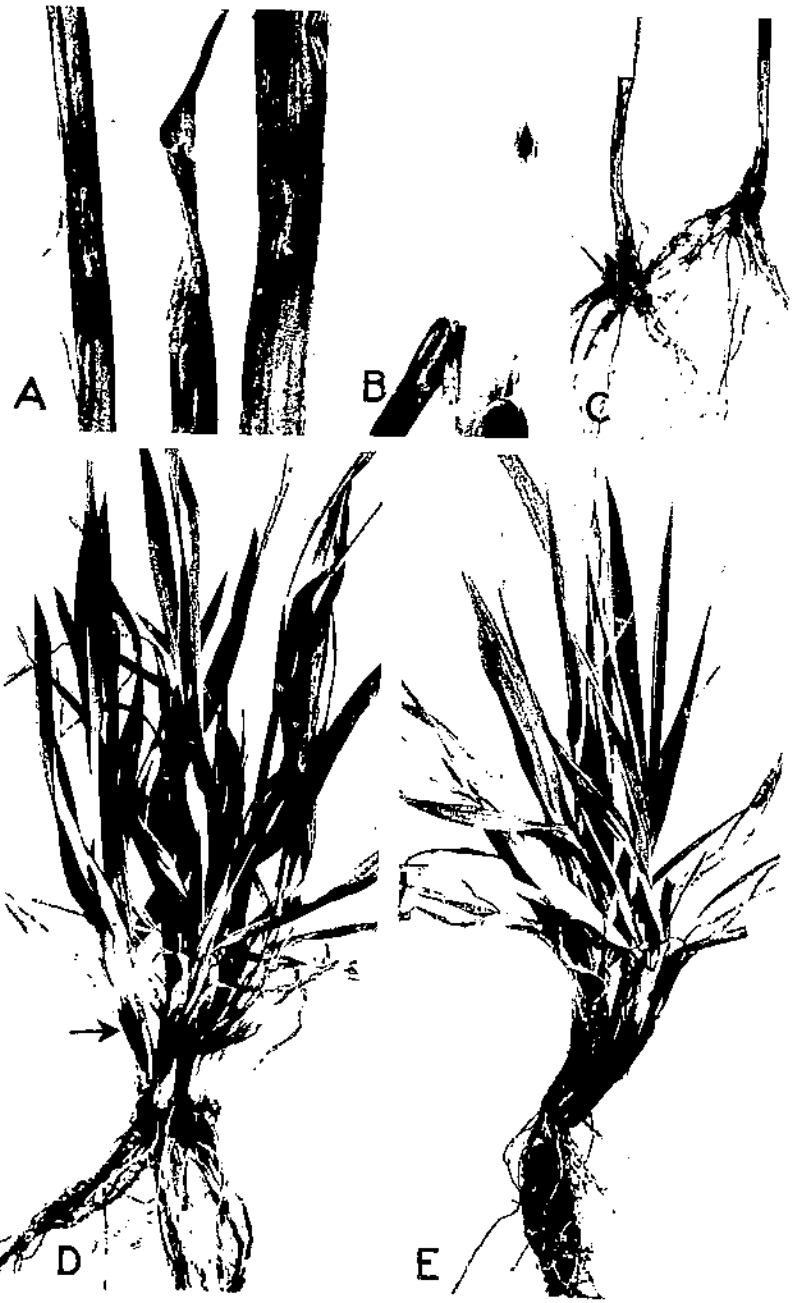
In 1919 Foëx (*6*) reported finding *Cercosporella herpotrichoides* at the end of March on wheat sown August 9 of the previous year and also on the sheaths of culms placed in the ground, as well as in stubble on which *Leptosphaeria herpotrichoides* and *Ophiobolus herpotrichus* (Fr.) Sacc. had been produced. He suggested that if *C. herpotrichoides* is the conidial stage of *L. herpotrichoides*, it must, without doubt, play a role in the propagation of the fungus, but again he pointed out that it had not been possible to establish the connection between these two forms. Foëx also reported that *C. herpotrichoides* was sometimes found on wheat culms that had produced perithecia of *L. herpotrichoides*, *O. graminis* Sacc., and *O. herpotrichus*, but he did not consider this association as proof of connection of this conidial form with any of these ascigerous forms.

From 1919 to 1924 Heald (*18*) consistently isolated a sterile fungus, now known to be *Cercosporella herpotrichoides*, several Fusaria, and a fungus referred to as *Wojnowicia graminis* (McAlp.) Sacc. and D. Sacc. from diseased culms of wheat grown near Spokane, Wash. Preliminary experiments conducted at Pullman, Wash., indicated that none of these fungi was pathogenic on wheat. The foot rot studied by Dana (*2*) was different from the one caused by *C. herpotrichoides*, as his studies were confined to western Washington, where this latter disease has thus far not been found.

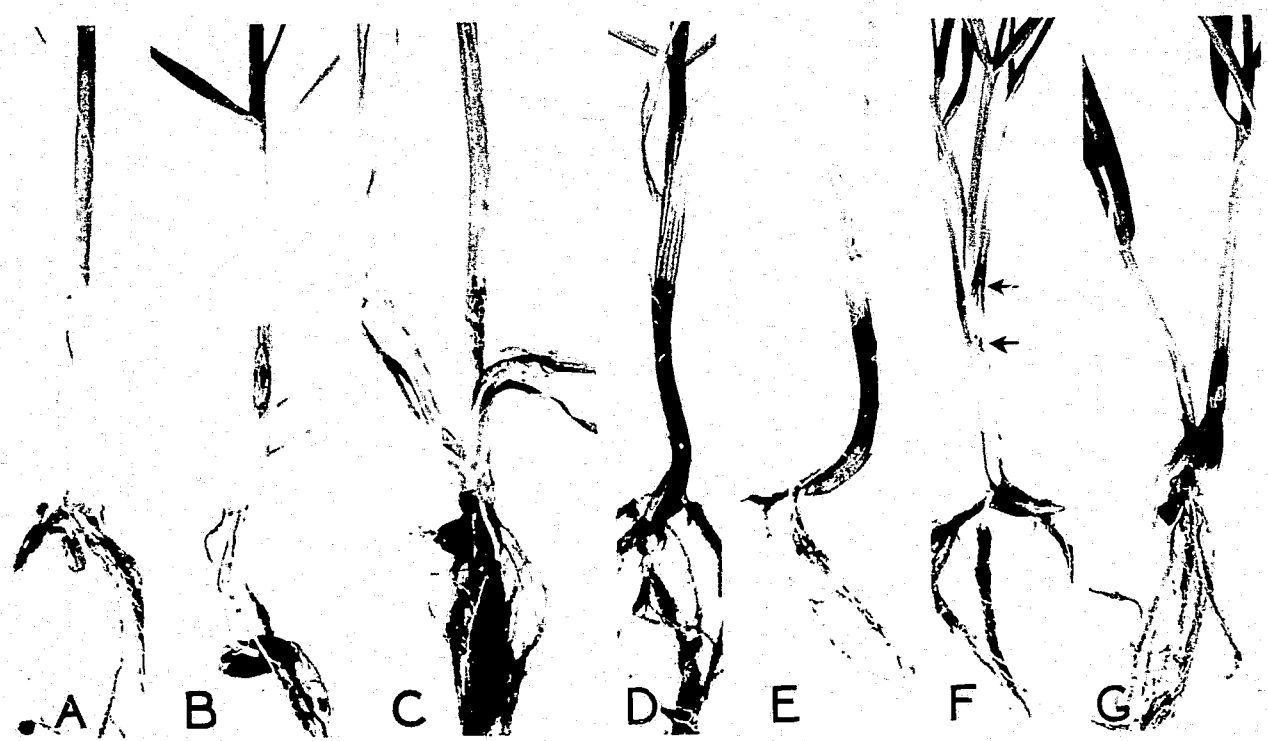
In 1925 some of the late-season symptoms were mentioned by McKinney (*23*), who briefly presented the results of the early cooperative experiments in the Pacific Northwest.

Nearly 10 years after Heald and his coworkers first began to isolate the "sterile fungus", later shown to be *Cercosporella herpotrichoides*, Foëx and Rosella (*7*, *8*) isolated a similar organism in France which they termed "champignon x" (fungus x). They were able to induce the disease with this organism. They also found spores of *C. herpotrichoides* on artificially inoculated plants (*9*, *10*), but were unable to obtain the spores of the fungus in pure cultures. In 1931 (*11*) they stated that the fungus x apparently was the one responsible for most of the really serious cases of foot rot found in the preceding few years in the Departments of Seine-et-Oise and Eure-et-Loir, France. In 1931 Kerleroux (*20*) found that the fungus x attacked wheat plants both in the fall and spring, but more severely in the spring following a severe winter.

In 1929 and again in 1930 the senior writer obtained spores of *Cercosporella herpotrichoides* in pure culture from mycelium of the long-known but supposedly sterile fungus isolated from wheat culms collected in Oregon and Washington (*29*). Until he obtained what he at first assumed were *Cercospora* spores in the early fall of 1929, the original articles of Fron and Foëx had not been reviewed. While the article by the senior writer (*29*) was being prepared for publication the article of Foëx and Rosella (*9*) came to hand, and through the kindness of Doctor Foëx the macroscopic identity of the fungus in the United States and of that from France was determined. Since that date, typical spores of *C. herpotrichoides* Fron were obtained in pure culture transfers of an isolation of the so-called "fungus x" furnished to the senior writer. The pathogenicity of the *C. herpotrichoides* from the Columbia Basin was proved (*29*).



A, Eye-spot lesions on leaf sheaths from winter-wheat plants, High Prairie, Wash., April 3, 1930. ×2. *B*, Culm, with the sheaths pulled back, showing young lesion. ×2. *C*, Basal portions of old diseased wheat culms with sheaths removed. ×1. *D*, Young plants of Hybrid 128 club wheat affected with cercosporella foot rot, High Prairie, Wash., Mar. 24, 1931 (sown Sept. 6, showing start of infection at the ground line. ×2. *E*, The disease advanced further than in *D*, showing injury from ground line down to crown. ×2.



BASAL PORTIONS OF PLANTS INOCULATED AT SEEDING TIME WITH PURE CULTURES OF CERCOSPORELLA HERPOTRICHOIDES AND KEPT IN A COOL GREENHOUSE.

A, Hybrid 128, uninoculated check. ×⅗. *B*, Hybrid 143 wheat 3 months after inoculation, showing eye-spot lesion. ×1. *C*, Same as *A* but with outer leaf sheaths stripped back, showing *stromata* of the fungus between the sheaths. ×1. *D*, Alstroum spelt, a very susceptible host, with extensive lesion on base of culm. ×⅗. *E*, Alaska barley, showing long but superficial lesion at base of culm. ×⅘. *F*, Einkorn, a very resistant host, showing very small lesions at ground line as indicated by arrows. ×⅗. *G*, Lesions on Hybrid 128 wheat. ×⅗.

SYMPTOMS OF THE DISEASE

Cercosporella herpotrichoides attacks chiefly the basal leaf sheaths and internodal tissues of the culms at or near the soil line. The fungus does not attack underground portions of the host. In some cases brace roots are invaded for a short distance where they come in contact with infested soil, and occasionally the tissues of the basal nodes also are penetrated.

Unless otherwise stated, the symptoms of the disease were observed on susceptible winter wheat (*Triticum vulgare* Vill.).[3]

SYMPTOMS IN EARLY SEASON

In the Pacific Northwest most of the infection begins in late February or early to mid-March, depending on the season. The first lesions become evident on the outer leaf sheaths as pin-point, water-soaked areas. These enlarge vertically and when fully developed are 1 to 2 centimeters long and 3 to 6 millimeters wide. The lesions are elliptical and have a tendency to become ovate after the expanding lesion meets obstructing nodal tissue. In March and April the lesions when well developed are fawn colored to nearly white and are bordered by an amber-brown[4] to chestnut-colored area (pl. 1, *A*). In later stages, early May to harvest time, secondary fungi and the gradual nonparasitic necrosis of the sheath obscure the clear-cut outlines of these outer lesions. Foëx and Rosella (*8*) term the fully developed lesions "taches ocellées" (eye-spot lesions).

The fungus is able to penetrate into deeper tissue either directly from one sheath to the next (pl. 1, *D* and *E*) or through the rupture of the sheaths by crown roots.

In the early-season infections a visible dark-olive to black stroma develops at the ground line between each leaf sheath and over the eye-spot lesions. This stroma gives the diseased host parts a charred appearance (pls. 1 and 2). Various stages in the development of the disease on artificially inoculated plants are illustrated in plate 2.

The weaker tillers of a plant are the first to be attacked, and these become sources of infection for the stronger ones. The fungus reduces the number of tillers, and while the less-crowded survivors partially refill the vacancies a seriously diseased field has a distinctly thinner stand after heading time.

SYMPTOMS IN LATE SEASON

The disease usually is most conspicuous near the end of the growing season of the host. Diseased plants are characterized by an excessive number of dead, brown tillers. The culms are affected in the first or second internode with lesions that extend from the ground line upward for 1 or 2 inches (pl. 1, *B*, *C*). The culm lesions vary from almost white (ivory yellow) to black, depending on the extent of development of the stroma. The culm tissue in the lesions is firm and brittle, slightly to distinctly collapsed on one side of the stem, or later the lesion completely girdles the culm and is bordered by discolored to amber-brown, sometimes (later) xanthine-orange areas.

[3] According to the rules of botanical nomenclature the name of this species is *T. aestivum* L., but as *T. vulgare* is in general use among agronomists and cereal pathologists and geneticists, the writers give preference to that form.

[4] RIDGWAY, R. COLOR STANDARDS AND COLOR NOMENCLATURE. 43 p., illus. Washington, D.C. 1912.

The fungus spreads faster in the parenchyma in the lumen region than it does nearer the outer surface of the culm. During moist weather in late season (June to July) the lumen of a large percentage of the diseased culms becomes filled with wefts of smoke-gray mycelium.

After the heads become heavy with grain the diseased culms buckle at the lesions and fall. The percentage of culms that fall varies according to the variety, the succulence of the culm, the severity of infection, and the occurrence of beating storms. Inasmuch as the period of falling extends over a considerable time during which the wind may blow from various directions, the grain becomes badly crisscrossed as it falls, and it is difficult and often impossible to harvest it (pl. 3). If driving rains occur near harvest time, the grain in heavily infested fields lodges very badly.

In the sections of the cercosporella foot-rot area where grain grows very tall, as in the Grande Ronde Valley of Oregon, lodging often occurs in wheat not injured by foot rot. Whereas the diseased culms buckle at the point of injury, the nonparasitized lodged culms usually bend in an arc that involves several of the lower internodes. In some cases where the ground is very soft following rains, the nonparasitized lodged plants are partly uprooted. Wheat culms attacked by *Cercosporella herpotrichoides* are frequently weakened to a point where they scarcely react to geotropism, whereas the heads of nonparasitized lodged culms usually become at least partly erect by growing upright at the nodes.

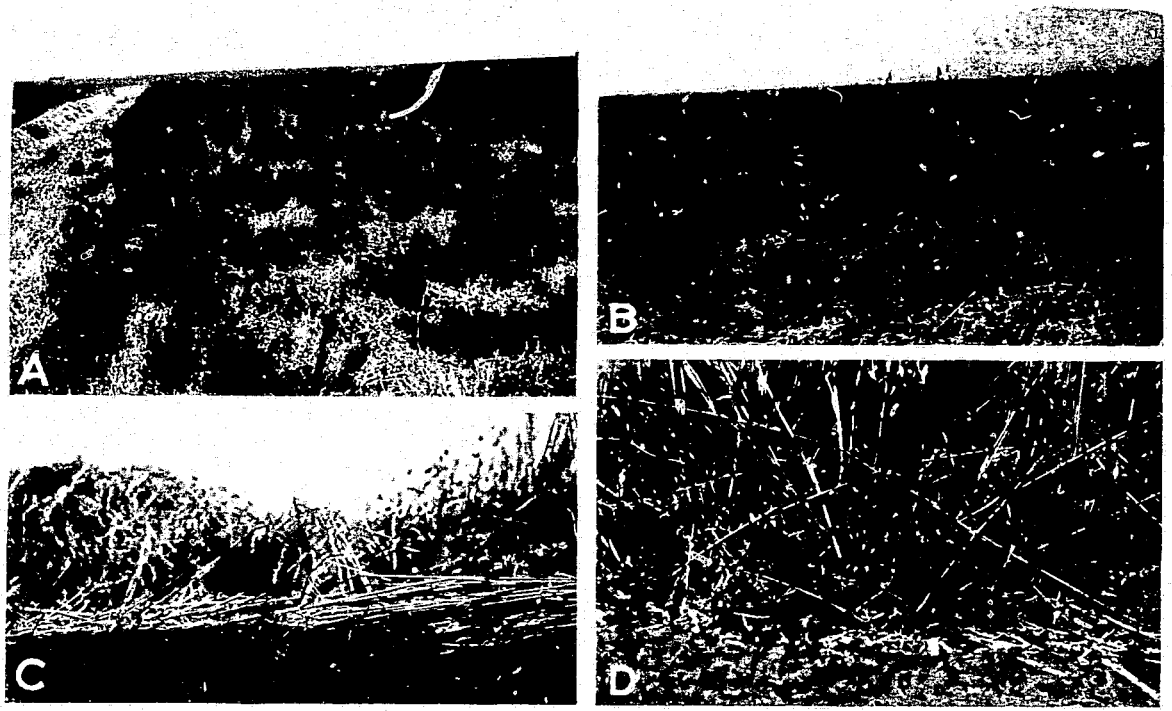
In severe cases, when infection starts early and continues throughout the rainy season, the culms may become stunted and fail to head. Saprophytic sooty molds may overrun entire plants, causing them to become fragile and to ripen prematurely.

The appearance of the diseased plants, as described above, contrasts rather sharply with that of plants affected with "take-all", a disease caused by *Ophiobolus graminis*.

Ophiobolus graminis attacks the underground portions of the wheat plants, progresses upward, and, as a rule, does not attack the inner leaf sheaths. The black lesions in take-all have a dark neutral-gray appearance when viewed through the thin sheaths, whereas those on cercosporella-infected plants appear charred.

SYMPTOMS FROM SECONDARY INFECTIONS

The growing season in the Grande Ronde Valley of Oregon starts later in the spring than it does in the Columbia Gorge area of Oregon and Washington, and the rainy season also is considerably more protracted in the former area. As a result of these conditions, new infections which are in part secondary from spores, begin developing in the Grande Ronde Valley at any time up until harvest. These later lesions, which also have the eye-spot appearance, have vague to sometimes dark-brown borders. The primary stromatic tissue begins to form at the point where the fungus first enters the sheath and underlying culm. Since the fungus may become arrested by dry weather, it is not uncommon to find elliptical lesions with scarcely any bordering of discolored tissue and no trace of stromatic mycelia except possibly a pin-point spot at the point of invasion. If the culm ripens early because of dry weather, these lesions become somewhat enlarged through necrosis, resembling sun scald.



FIELD SYMPTOMS OF THE CERCOSPORELLA FOOT ROT OF WHEAT IN THE GRANDE RONDE VALLEY OF OREGON, 1928.

A, General view of a field of Hybrid 128 wheat showing the culms fallen in many directions, causing a tangled condition that increases the expense of harvest. *B*, View of badly fallen wheat, showing considerable areas flat; a few slightly diseased plants are still standing. *C*, Detailed view of culms at ground line showing how closely appressed the fallen grain frequently is. This makes harvest difficult and slows up the ripening process. *D*, Detailed view showing the beginning of falling.

SYMPTOMS ON WINTER BARLEY

Cercosporella foot rot appears on barley (*Hordeum vulgare* L.) somewhat later in the spring than it does on wheat. The fungus attacks the inner portion of the barley culms far more readily than it does the hard outer portions. Also, the fungus forms less stromatic tissue on barley than on wheat, and there is less collapse of tissues in the lesions near maturity. Secondary fungi are especially confusing in this host. In general, there is less buckling of the culms in barley than in wheat.

GEOGRAPHIC DISTRIBUTION AND ECONOMIC IMPORTANCE

In the United States, cercosporella foot rot occurs in three main areas, all within Washington, Oregon, and Idaho, as follows: The rolling prairie country lying at an elevation above 1,500 feet, adjacent to the Columbia Gorge in Wasco County, Oreg., and Klickitat County, Wash.; the relatively level Grande Ronde Valley, in eastern Oregon, surrounded by the Blue Mountains, and the adjacent prairie lands in Oregon and Washington; and in the Spokane Valley on gravelly prairie soil in Washington and Idaho and on fine sandy loam on adjacent prairie lands in Washington.

The counties in Washington, Oregon, and Idaho where cercosporella foot rot has been found, and the estimated reduction in yield of wheat, in bushels, are shown in table 1. The injury varies greatly from season to season. The seasonal severity of the disease was as follows: 1928, very severe; 1929, slight; 1930, moderate; 1931, moderate; and 1932, severe. In spite of the severity of the disease development in 1932, the total losses are noted as less or no greater than in either of the 2 preceding years. There are two reasons for this: (1) Many farmers discontinued the growing of wheat in the fall of 1931 because of the previous losses sustained and because of low prices, and (2) many farmers who continued to grow wheat used relatively resistant varieties and followed practices that reduced the development of the disease.

TABLE 1.—*Estimated reduction in yield of wheat caused by cercosporella foot rot in certain counties of Washington, Oregon, and Idaho, 1928–32* [1]

State and county	Estimated reductions in yield				
	1928	1929	1930	1931	1932
Washington:	*Bushels*	*Bushels*	*Bushels*	*Bushels*	*Bushels*
Klickitat	6,500	1,500	1,500	2,500	2,500
Spokane	16,000	2,000	4,000	11,000	10,500
Walla Walla	[2]			1,000	1,000
Oregon:					
Umatilla	[2]			[2]	[2]
Union	60,000	10,500	12,000	4,000	3,000
Wasco	12,000	4,000	3,500	2,500	2,000
Idaho:					
Kootenai			[2]	1,000	2,500
Total	94,500	17,500	21,000	22,000	21,500

[1] A serious outbreak of Cercosporella foot rot occurred in the spring of 1934, following the open winter of 1933–34. The loss in bushels is estimated as follows: Idaho, 155,000; Washington, 90,000; and Oregon, 30,000. Counties in Idaho in which the disease had not been previously known are Clearwater, Idaho, Latah, Lewis, and Nez Perce.

[2] Trace.

As already stated, the fungus causes a distinct reduction in the number of culms. In culms attacked early in the spring there is a sharp decrease in grain weight per head, as shown in table 2. The data for 1929 were taken from healthy and diseased culms of Hybrid 63 and Hybrid 143 grown in tangled fallen spots at Tygh Ridge, Oreg. The diseased heads were evidently smaller and in many instances contained shriveled grains. Later, however, there was no difference in percentage of germination in the grain from healthy heads and that from diseased heads. The 1930 data were taken from a field of Goldcoin wheat near Alicel, Oreg. The healthy check sample was from a small plot treated by being covered with straw and burned over immediately before seeding.

TABLE 2.—*Effect of cercosporella foot rot on weight of heads and grain in healthy and diseased wheat grown in fields of infested soil on Tygh Ridge, Wasco County, Oreg., and near Alicel, Union County, Oreg., respectively, 1929–30*

Year grown and variety	Average weight per head						Percentage of grain		Average kernels per head			Weight per 1,000 kernels		
	Total			Grain										
	Healthy	Diseased	Decrease	Healthy	Diseased	Decrease	Healthy	Diseased	Healthy	Diseased	Decrease	Healthy	Diseased	Decrease
1929	*Grams*	*Grams*	*Per cent*	*Grams*	*Grams*	*Per cent*			*Number*	*Number*	*Per cent*	*Grams*	*Grams*	*Per cent*
Hybrid 63	0.9244	0.6866	26.0	0.6675	0.4507	32.5	72.2	65.6	24.8	18.1	27.0	24.3	21.8	10.3
Hybrid 143	1.4879	.8023	46.0	1.0884	.5941	45.4	73.2	66.6	39.0	25.3	35.2	27.9	22.9	17.9
1930														
Goldcoin	2.2051	1.1112	49.6	1.5770	.7664	51.7	71.5	69.0	35.2	23.3	33.9	44.6	33.6	25.0

The depressing effect of a very severe attack of cercosporella foot rot on the yield of wheat is shown in table 3 from data obtained in Klickitat County, Wash. The inconsistent data on Hosar are explained by the extreme shattering that occurred in the healthy heads.

TABLE 3.—*Effect of a severe attack of cercosporella foot rot on the yield of several varieties of winter wheat grown in the field on High Prairie, Klickitat County, Wash., 1930–31*

Variety	C.I. no.[1]	Comparative resistance	Acre yield [2]	
			Healthy	Diseased
			Bushels	*Bushels*
Hosar	10007	Very resistant	[3] 29.0	30.6
Queen Wilhelmina	.....	Resistant	46.6	30.9
Hybrid 128	4512	Susceptible	53.3	23.3
Turkey	.....	Very susceptible	47.0	10.9

[1] C.I. refers to accession number of the Division of Cereal Crops and Diseases, formerly Office of Cereal Investigations.
[2] The comparative yields are based on square-rod areas in adjacent diseased and nondiseased portions of the same field.
[3] Yield reduced somewhat by shattering.

DISTRIBUTION ACCORDING TO SOIL TYPES

The distribution and the economic importance of cercosporella foot rot on the various types of soil in the Columbia Basin are shown in table 4. The specific names of the soils are given when known, e.g.,

Hunter's very fine sandy loam, otherwise the soils are listed by class, e.g., very fine sandy loam.

In general, the fine sandy loam and loam soils, which are warm and retain moisture fairly well and have a low hygroscopic moisture coefficient, are the ones that harbor the foot-rot fungus.

The principal exception to this is near Coeur d'Alene, Idaho, on cut-over land in Helmer silt loam. In this area the water table, which lies a few feet under the surface, counteracts the drying conditions that usually occur in heavy soil after the spring rains cease. The area involved on this soil is small, however, in comparison with the total area infested in the nearby prairie lands.

TABLE 4.—*Distribution and degree of severity of the cercosporella foot-rot disease on various types of soil in the Columbia Basin, and the estimated area infested*

Location	Degree of severity	Soil type	Estimated area infested with *Cercosporella*
			Acres
Grande Ronde Valley, Oreg.	Very severe	Alicel fine sandy loam	5,000
Do.	Severe	Alicel sandy loam	500
Elgin, Oreg.	Extremely severe	Very fine sandy loam	2,000
Tygh Ridge, Wasco County, Oreg.	Very severe	Fine sandy loam	2,000
High Prairie, Klickitat County, Wash.	Severe	do	1,500
Seven Mile Hill and vicinity, Wasco County, Oreg.	Moderate	Loam and gravelly loam	1,800
Friend, Oreg.	Trace	White sandy loam	100
Spokane Valley, Wash., and Idaho	Extremely severe	Garrison gravelly loam	6,000
Peone Prairie, Spokane County, Wash.	Very severe	Hunter's very fine sandy loam.	3,000
Peone and Pleasant Prairies, Spokane County, Wash.	Severe	Palouse fine sandy loam	400
Coeur d'Alene, Idaho	Very severe	Helmer silt loam	(1)
Columbia and Walla Walla Counties, Wash.	Moderate to slight	Very fine sandy loam	1,000
Umatilla County, Oreg.	Trace	Loam	(1)
Anatone, Wash.	Moderate	Sandy loam or loam [2]	2,500

[1] Scattered fields.
[2] Approximately Palouse series.

HOST RANGE AND VARIETAL SUSCEPTIBILITY

From 1925 to 1932 several hundred varieties and selections of cereals were studied on infested ground in an effort to find resistant varieties. Most of the material tested has been eliminated on account of susceptibility. Further study is being made of the more promising varieties. The technic of determining comparative resistance has gone through a gradual elaboration, until the following procedure is now used:

Plots are located on summer-fallowed land known to have been infested 2 years previously.

Varieties are sown early in September, usually in duplicate 3-row blocks. (Until 1932 at least 2 and usually 4 or 5 tests were located in widely scattered sections.)

Every fourth plot is sown to a check of Hybrid 128. This variety is commercially grown throughout the areas where cercosporella foot rot is found. It is of average susceptibility.

The injury from foot rot is estimated at harvest time by classifying the culms in several linear feet of the central row in each plot of each variety according to the degree of severity of injury, as shown in table 5.

TABLE 5.—*Degree of severity and percentage of culm injury caused by cercosporella foot rot on various selections of cereals studied from 1925 to 1932*

Degree of severity	Percentage of injury	Description
None	0	No foot rot.
Slight	5	Young eye-spot lesions not penetrating more than a few cells deep.
Moderate	10	Lesions penetrating one fourth to one half way through culm, usually a young developing lesion.
Severe	30	Slightly less than one half way through culm (in extended lesions) to nearly or entirely through culm, but tissue not extensively collapsed.
Very severe	60	Rotted clear through the culm, tissue collapsed; plant evidently will not recover.
Dead [1]		Culms dead and charred by disease, tillers small. When a large number of tillers are dead and diseased the percentage above 40 should be added to the total percentage of injury to approximate the actual percentage of reduction in yield.

[1] Previous to 1932, dead culms were not considered in the estimate except in cases where over 67 percent were dead. The percentage above this figure was added to the total percentage of injury.

The percentage of injury is determined by multiplying the percentage of diseased culms by the average rating on the severity of injury of the diseased culms. The calculated percentage of injury so estimated when checked with yield data has been found to be reasonably accurate.

The relative susceptibility of varieties is determined by direct comparison with the nearest Hybrid 128 check (considered as 100), taking into account observable abrupt changes in soil infestation, influence of adjacent varieties of guard rows, etc.

A summary of the reaction to cercosporella foot rot of certain key varieties and selections is given in table 6. Most of the varieties and selections of the several cereals tested are not listed.

TABLE 6.—*Reaction of certain varieties of cereals and grasses to cercosporella foot rot*

Genus, species, and variety	C.I. no.	Severity rating [1]
Avena sativa (oats):		
Winter Turf (Gray Winter)	1670	0–10
Hordeum vulgare (barley):		
White Club		[2] 30–50
Tennessee Winter		80–90
Alaska		90–100
Secale cereale (rye):		
Rosen		0–10
Union Beardless		30–60
Aegilops cylindrica		10–30
Aegilops ovata		30–50
Aegilops triuncialis		60–70
Triticum monococcum (einkorn)	2133	0–10
Triticum dicoccum (emmer):		
Black Winter	2337	[3] 10–30
Triticum spelta (spelt):		
Alstroum	1775	130–140
Triticum compactum (club wheat):		
Little Club	4066	60–70
Red Chaff sel.	10072	60–70
Fortyfold × Hybrid 128	8251	90–100
Hybrid 128	4512	100
Hybrid 143	4100	100
Hybrid 63	4510	100–110
Jenkin	5177	100–110
Fortyfold × Hybrid 128 (Nursery no. 942)		100–110
Fortyfold × Hybrid 128 (Nursery no. 945)		100–110
Albit	8275	100–110
Triticum vulgare (common wheat):		
Queen Wilhelmina (Holland)	11380	60–90
Ruston	5208	60–70
Hohenheimer (glabrous chaff)	11458	60–70
Hohenheimer (pubescent chaff)	11459	70–80
Federation	4734	70–80
Ridit	6703	90–100
Wheat-rye	8899	90–100
Purplestraw	1915	90–100
Dawson	3342	90–100
Pacific Bluestem	4067	90–100
Triplot	5108	100–110
Rio	10001	100–110
Harvest Queen	3314	100–110
Turkey × Minessa	8035	110–120
Kharkof	8249	110–120
Turkey	4129	110–130
Goldcoin	4156	110–120
Golden	10033	110–120
Moskin	6088	120–130
Jones Fife	4408	120–130
Kanred	5146	120–130
Oro	8229	120–130
Mahmudi × Minturki	8215	120–130
Hussar	4813	120–130
Cooperntorka	8801	120–130
Stoyanitchka	8802	130–140

[1] Compared with Hybrid 128 checks rated as 100.
[2] Sometimes more resistant.
[3] Sometimes less resistant.

The relative susceptibility of varieties varies somewhat from year to year. For instance, if the rainfall in the period from March to June is heavy the moderately susceptible club wheats become considerably more susceptible. As a rule the Turkey types have been the most susceptible of the wheats, although some of the soft-red and white varieties, including Jones Fife and Goldcoin, are nearly as severely injured.

Susceptibility in a variety usually is associated with one or several of the following characters: Deep-seated compact crowns, abundant tillering, small culms in compact crowns, tall straw, and moderately early maturity.

Resistance in a variety usually is associated with one or several of the following characters: Shallow-seated relatively open crowns, sparse tillering, coarse straw and tough leaf sheaths, short straw, spring habit, and very late maturity.

Hohenheimer (glabrous and pubescent) and Queen Wilhelmina (known also as Holland) are good examples of very late-maturing resistant varieties. The resistance shown by fall-sown spring wheats seems to be a matter of aeration. The stands of these varieties usually are thinned by winter injury, the plants tiller poorly, and therefore conditions around the crowns are drier and not so favorable for the development of the disease. For example, Hybrid 143, a club wheat of spring habit, is resistant to foot rot under average semiarid conditions, but becomes very susceptible when grown in heavy stands under humid conditions.

In the fall of 1930 and in the late winter and spring of 1931 periodic sowings of spring and winter wheats and a winter barley were made in infested soil on Seven Mile Hill, Wasco County, Oreg., and the percentage of injury was determined for each variety by the method previously described. The results are given in table 7.

There was no foot rot in the series sown in the spring and but slight injury in the series sown in February. This agrees with field observations, that spring-sown grain escapes the disease.

In general, the study of varietal resistance shows that none of the adapted commercial wheat varieties is very resistant, but the results suggest the hope that resistant varieties may be developed by breeding and selection.

Extensive search has been made for *Cercosporella herpotrichoides* on various wild grasses growing near infested wheat fields, but so far without definite results. In June of the years 1929, 1931, and 1932, lesions resembling those on wheat were found on *Agropyron inerme* (Scribn. and Smith) Rydb. Attempted isolations from such lesions did not yield the fungus, but there is need for further study of this grass, especially on specimens collected earlier in the season.

PATHOLOGICAL HISTOLOGY

Studies on the histology of foot-rot-infected wheat and barley plants were conducted at Corvallis, Oreg., in 1930–31. With the exception of the early writings of Fron (*13*) and a recent note by Foëx and Rosella (*12*), there is no published information on the pathological histology of this disease.

TABLE 7.—*Cercosporella injury in varieties of spring and winter wheat and a winter barley sown in infested soil in the fall, late winter, and spring at Seven Mile Hill, Oreg., 1930–31*

Crop and variety	C.I. no.	Growth habit	Reaction to cercosporella foot rot	Injury when grain was sown—		
				Sept. 5, 1930	Feb. 20, 1931	Apr. 10, 1931
Wheat:				*Percent*	*Percent*	*Percent*
Minhardi×Minturki	8215	Winter	Susceptible	20.4	1.2	0
Goldcoin	4156	do	do	19.6	.9	0
Hybrid 128	4512	do	do	18.0	.7	0
Purplestraw	1915	Spring	do	17.5	.8	0
Federation	4734	do	Resistant	9.5	.9	0
Huston [1]	5208	do	do		.2	0
Average				17.0	.78	0
Barley:						
O.A.C.[2] no. 7	2814	Winter	Resistant	7.9	1.0	0

[1] Winter-killed when sown Sept. 5; data from this variety not included in average.
[2] O.A.C. = Oregon Agricultural College.

METHODS AND MATERIALS

Most of the study was made on field-infected material killed and fixed in chromo-acetic fixative, dehydrated and infiltrated with paraffin after the n-butyl alcohol method, as used by Zirkle (*30*). Because *Cercosporella herpotrichoides* and associated fungi are usually mingled in the tissues of field-infected plants, supplementary studies were conducted with wheat artificially inoculated in the greenhouse with *C. herpotrichoides*. Where possible, fresh material from infected host tissues grown both in field and greenhouse was studied.

The n-butyl alcohol method usually, though not always, aided in softening the highly silicified and brittle foot-rot-invaded culms. As a further aid, the paraffin blocks were soaked in water for long periods.

Erythrosin in clove oil was the most satisfactory stain for routine laboratory studies. For photomicrographic work, three stain combinations were used: Ehrlich's haematoxylin, safranin and fast green, and Haidenhain's iron alum-haematoxylin.

OBSERVATIONS

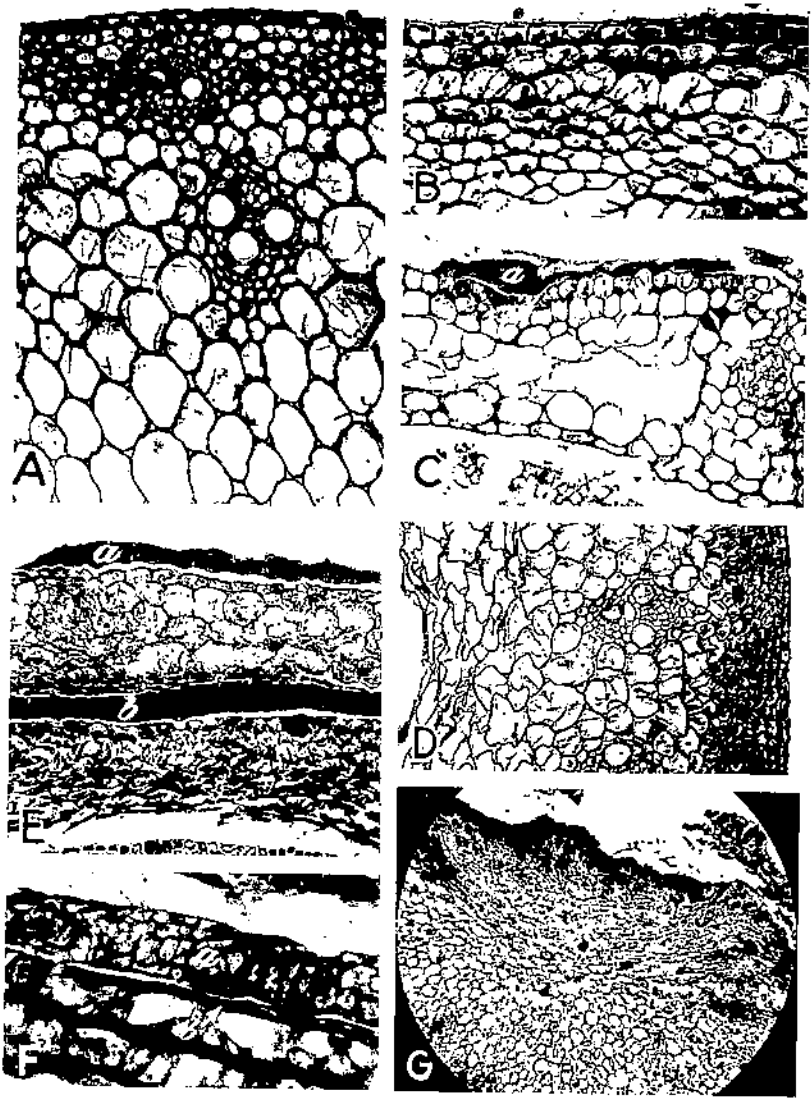
PLANTS INOCULATED IN THE GREENHOUSE

In young plants from seed inoculated at time of sowing the penetration of the outer sheaths, which in these cases frequently are coleoptiles, follows the formation of slight to substantial stromata. Since inoculum is abundant the chances for excess development of stromata are increased. In those cases in the greenhouse where there were secondary infections from spores of *Cercosporella herpotrichoides* the infections took place through stomata and formed eye-spot lesions, and stromata developed later.

PLANTS GROWN IN THE FIELD

EARLY-SEASON DEVELOPMENT

The fungus gains entrance into the outer leaf sheath of the host either directly through the epidermal cells or, more commonly, through stomatal openings and usually precedes its invasion by forming a



CROSS SECTIONS THROUGH LESIONS AT BASE OF WHEAT PLANTS.

A, Section of wheat culm in late June, showing late-season invasion of fungi into culm. The host cells are only slightly injured, the xylem elements being especially resistant. ×120. *B*, Young lesion in culm showing characteristic development of a late-season infection in the Grande Ronde Valley of Oregon. Note thickening of outer cell walls. ×200. *C*, Infection of leaf sheath in the field, High Prairie, Wash., in early April. The fungus formed a stroma *a*, on the outside of the sheath and penetrated through the outer cell layer and again started stromal formation *b*, on reaching the inner side of the cell cavity. ×120. *D*, Culm tissue severely infected in early June with foot-rot fungi. The outer cells are nearly filled with hyphae. The larger vessels were free of mycelia, while the fungus had penetrated to the culm lumen. The stromatic layer on the outside of the culms was torn away in the process of sectioning, together with the remnants of the sheaths. ×85. *E*, Cross section of young tiller in early spring, showing invasion of the two outer leaf sheaths by foot-rot fungi. The sheaths are partly broken down. Note the prominent fungus stromata *a* and *b*. The third sheath has not yet been invaded; the deeper lying sheaths are always resistant to the fungal invasion because they are in a more vigorous condition. ×120. *F*, Stroma *a*, on outside of second leaf sheath, showing cells of macrophyplae, and epidermis showing thickened outer cell walls *b*. ×325. *G*, General view of cross section of shrunken lesion showing collapsed cells. ×35. Note that the larger elements of the xylem remain partly intact.

stroma on the outer surface of the host (fig. 1 and p', 4). The stromata range in size from a few cells to a mass measuring several millimeters in diameter. The individual cells of the stroma are closely packed, polygon-shaped, heavy-walled, and are olive-brown. The stroma of *Cercosporella* differs markedly from the rhizomorphlike filaments of *Ophiobolus graminis* illustrated by Kirby (*21*).

The stroma on the outer surface continues to grow after the fungus invades the host and usually becomes very extensive through sapro-

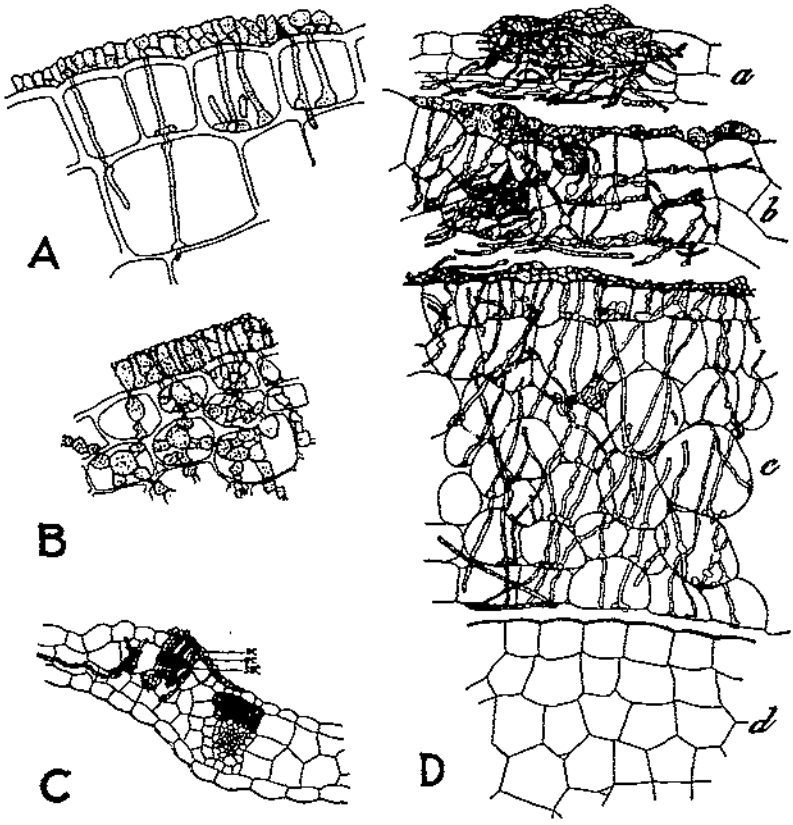


FIGURE 1.—Cross sections through lesions at base of wheat plants: *A*, Young infection in outer leaf sheath, showing direct penetration of the outer cells following the formation of a stroma. Note the slight constriction of the hyphae in passing through the cell walls of the host and the tendency to expand on reaching the inner walls. ×675. *B*, Detail of older infection in leaf sheath, showing increased septation and hyphal development. Shows detail of the passage of the fungus through the thickened cell walls. ×675. *C*, Detail of very young infection in leaf sheath in March, showing penetration through stomatal opening. Note strands passing through stomatal opening (*sc*), fungi in guard cells (*gc*) and stromatic mass completely filling substomatal cavity (*sac*). ×190. *D*, Portion of a cross section at base of a young wheat tiller 7 weeks after the wheat had been inoculated with *Cercosporella herpotrichoides* at sowing time, in the greenhouse. The fungus has penetrated through the leaf sheaths (*a*, *b*, and *c*) and is about to attack the inner sheath *d*. Note the fungus stromata, the bulbous enlargements on some of the hyphae within the cells, and the thickenings of the cell walls in certain of the infected areas, especially beneath stromata. This section happens to be taken so that no well-developed veins are shown. ×500.

phytic development, producing the charred appearance previously mentioned.

When the fungus penetrates directly through the epidermal cell, the penetrating hyphae are reduced in diameter, but after reaching the cell cavity they increase their diameters again. The writers have not

observed the exaggerated intracellular development shown by Fron (*13*).

Wherever the fungus is retarded by contact with resisting cell walls it produces accumulations of mycelial tissue in the form of bulbous cells and various types of macrohyphae. Frequently these fill the whole cell cavity (fig. 1 and pl. 4). In the stomatal invasions the fungus accumulates a stroma in the substomatal cavity, invades the guard cells, and eventually pushes its way into the adjacent mesophyll cells (fig. 1, *C*).

The host cells in the outer sheaths develop heavily thickened walls beneath the stroma and in cells that are being invaded (fig. 1 and pl. 4, *F*). No lignitubers were seen, such as described by Fellows (*4*) for *Ophiobolus graminis*, but in some cases cell walls were thickened to a point where they approximated one third of the width of the cell cavity.

The fungus is able to penetrate the inner chlorenchyma tissue of the outer leaf sheaf rather readily, but it is held in check for a time by the external wall of the inner epidermal layer, as shown by the accumulation of hyphae present at this point in young lesions. After forcing its way into the space between sheaths, the fungus develops another stroma which is usually larger than that on the outer sheath and consists of cells (macrohyphae) larger than those of the first-formed stroma (fig. 1, *D*, and pl. 4, *E*).

MIDSEASON DEVELOPMENT

When the conditions are favorable for the fungus, it continues to penetrate radially, forming a series of stromata between the leaf sheaths (pl. 4, *E*). While the culm with the delicate head has usually grown past the line of attack by the time the inner sheath is reached, instances have been observed during periods of very damp and cool spring weather in which the fungus killed the entire tiller before heading time. In any season some of the lateral culms, growing less vigorously than the main culm because of food limitations, are invaded rapidly and frequently fail to recover. In figure 1, *D*, the fungus is illustrated as ready to enter the inner sheath before the bud has forced itself above the danger zone. At best, in such a case, the resulting head would be considerably stunted by the fungus.

LATE-SEASON DEVELOPMENT

In its progress through the leaf sheath the fungus tends to grow radially and oblique-radially. After a difficult passage has been made through the epidermal cells of the culm proper, the fungus progresses vertically almost as rapidly as horizontally in the vertically elongated cell tissue immediately beneath the epidermis. There is a tendency for young diseased culms to form a "water-soaked" halo, owing to the fact that the fungus advances faster in the above-mentioned subepidermal elongated cells than in the epidermal cells.

When the parenchyma cells are reached in the interior of the culm the fungus again resumes a steady inward march through the less-resistant cells therein encountered. There is very little hyphal constriction in the invasion of parenchyma tissue.

After the initial horizontal growth into the inner parenchymatous tissue, the fungus progresses vertically for considerable distances.

Various other fungi also invade the lumen area at the same time, the mixed infection sometimes extending several inches up the culm. Such affected culms frequently are lead color, especially when *Cercosporella herpotrichoides* and associated fungi fill the hollow internodes with cottony gray wefts of vegetative hyphae.

In cases of long-extended infection in which the lesions shrivel and the culms fall, the cell contents break down and all but the heavier xylem elements become shrunken and collapse (pl. 4, *G*). The tissue throughout the diseased portion is heavy-walled and brittle. Davidson and Phillips (*3*) recently indicated that excessive lignin development tends to increase the brittleness of culms. The writers have observed that diseased tissue was much more difficult to section than corresponding healthy tissue, and when such diseased tissues were sectioned without special softening measures they broke into small fragments because of their brittleness.

The fact that the xylem elements resist the fungus saves much of the crop from complete destruction. Since the roots are not attacked, the plants, if they reach heading, are seldom killed, because a sufficient number of xylem elements survive to feed the aerial parts of the plants.

THE PATHOGENE

TAXONOMY

In 1931 the senior writer (*29*) accepted *Cercosporella herpotrichoides* as the correct name of the Columbia Basin foot rot, and in the brief literature reviewed on pages 51 to 52, *Cercosporella* is cited by all investigators with the exception of Foëx and Rosella (*8*) who, in 1929, gave *Cercospora herpotrichoides*. Their later reports refer to *Cercosporella herpotrichoides*. While there may be some question of including this species under the genus *Cercosporella*, the writers are accepting this classification for the present.

As previously stated, *Cercosporella herpotrichoides* was originally considered connected with *Leptosphaeria herpotrichoides*. While the two fungi occur together in foot-rot infested wheat in Europe, only negative information has been obtained on their connection. In the United States McKinney reported *Leptosphaeria* on wheat infected with *C. herpotrichoides* near Spokane, Wash. (*23*). *Leptosphaeria* has not been found since, although most of the surveys have been conducted in other areas than the one in which *C. herpotrichoides* was originally found. The cultures of *C. herpotrichoides* studied by the writers were distinctly different from the culture of *L. herpotrichoides* sent by Henry and referred to by Henry and Foster (*19*). Naturally diseased stubble was kept under observation on the ground at Corvallis, Oreg., and in the field at other points, but numerous examinations failed to disclose an ascomycete that might be the ascigerous stage of *C. herpotrichoides*.

All attempts to produce an ascigerous stage of *Cercosporella herpotrichoides* in pure culture have been negative. Numerous strains of the fungus were grown in pure culture at Corvallis, Oreg., during the winter of 1929–30 on various media, and parallel series were stored (1) in the laboratory at approximately 20° C., (2) in the ice box at approximately 12°, and (3) out of doors at fluctuating winter temperatures. The media used were as follows: Potato-dextrose agar (500 isolations); corn-meal agar; corn meal in flasks; wheat heads in test tubes; wheat

straw in test tubes; wheat kernels in flasks; oat and barley kernels in flasks; synthetic media of standard and special formulas; *Melilotus* stems, with water, also with 1-percent glycerine solution; gelatin; unpolished rice; and filter paper in various nutrient solutions and in water.

MORPHOLOGY

The hyphae of *Cercosporella herpotrichoides* are of two sorts: (1) Rapidly growing vegetative hyphae, which are pale chalcedony yellow to olive-brown in color and have narrowly cylindrical cells; and (2) stromatic hyphae with moderately to very thick walls (macrohyphae) and frequently polygonal cells that form charred masses on the outside of culms or sheaths or in the cells of infected culms.

The conidiophores are simple or sometimes branched. In some cases they are swollen at the base and elongated, but usually they are short, erect, subhyaline to hyaline, and rise from macrohyphal subicula. The large ends of the obclavate spores are attached terminally or subterminally and may occur singly or sometimes in pairs. The spores

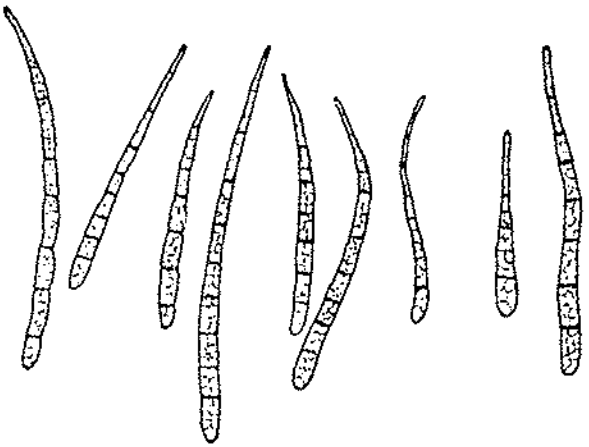


FIGURE 2.—Spores of *Cercosporella herpotrichoides* from culms of foot-rot-infected wheat grown at High Prairie, Klickitat County, Wash., April 1931. ×750.

(fig. 2) are somewhat curved and are 2 to several septate, chiefly 5 to 7 septate. They differ in size, ranging from 1.5μ to 3.5μ by 30μ to 80μ. Most of the spores are 40μ to 60μ long.

PHYSIOLOGY

ISOLATION STUDIES

Numerous pure-culture isolations from diseased tissues have been made on potato-dextrose agar at intervals since 1923. Previous to 1930, the isolations were obtained in June and July, after the disease was well advanced. The data for 1929 are presented in table 8. They show that *Cercosporella herpotrichoides* was by far the most frequently isolated fungus. Isolations from the young lesions yielded *C. herpotrichoides* in a higher percentage of cases than those from the older lesions, but there was not very much difference in the relative number of fungi in different parts of the lesions. The surface stroma was composed almost exclusively of *C. herpotrichoides*.

TABLE 8.—*Kinds and number of fungi isolated from young and old lesions and from various locations in lesions in wheat culms from fields near Imbler, Oreg., and in the vicinity of The Dalles, in Oregon and Washington, during June and July 1929*

Fungus	Cultures obtained from—			Total	Cultures obtained from locations in lesions as indicated					
	Young lesions	Old lesions			At or near margin	Center	Beyond visible margin	General	Surface stroma	Total
		Culms not buckled	Culms buckled							
Cercosporella herpotrichoides	*Number* 95	*Number* 178	*Number* 159	*Number* 432	*Number* 221	*Number* 158	*Number* 48	*Number* 23	*Number* 13	*Number* 463
Wojnowicia graminis	5	15	28	48	22	18	8	6	0	54
Fusaria spp.	8	30	30	77	25	37	13	4	2	81
Miscellaneous [1]	5	12	6	23	5	8	2	2	0	17
Total	113	244	223	580	273	221	71	35	15	615

[1] Includes unknown organisms that occurred frequently enough to warrant retention. Bacteria, common molds, and various nonseptate fungi were discarded.

During the season of 1930 isolations were made in March and April, and the results, shown in table 9, indicate that *C. herpotrichoides* was the primary invader.

As the season advances there is a tendency for secondary fungi to obscure the action of *C. herpotrichoides* and this is especially true following heavy rains in June. One of the most common symptoms at this time is the lead-colored stem induced by the various fungi browning the cells of the lumen. Besides the usual *Fusaria* and *Wojnowicia graminis* (fig. 2, *A*), several nonseptate fungi and a Rhizoctonialike fungus accompany *C. herpotrichoides* in these late-season infections.

TABLE 9.—*Kinds and number of fungi isolated from lesions on culms and sheaths of wheat taken from fields in the vicinity of The Dalles, in Oregon and Washington, during March and April 1930*

Date of isolation	Culm or sheath	Culms or sheaths infected by—			
		Cercosporella	*Wojnowicia*	*Fusaria*	Miscellaneous fungi
		Number	*Number*	*Number*	*Number*
Mar. 18 to 21	Culm	17	0	1	1
Mar. 28	do	10	0	0	0
Do	Sheath	7	4	7	18
Mar. 31	Culm	29	0	3	7
Apr. 4 to 7	do	01	9	1	0
Apr. 7	Sheath	24	0	2	3
Apr. 22 and 23	Culm	83	7	3	0
Apr. 25 to 30	do	41	6	3	3
Apr. 29 and 30	do	5	5	4	0

In some cases, *Wojnowicia graminis* alone seemed to extend the lesions caused by *C. herpotrichoides* in the lumen region and produced long discolored internal lesions.

The latter fungus is readily isolated from lesions on overwintering or overwintered stubble in the field.

CULTURAL CHARACTERS

VEGETATIVE GROWTH

The colonies of *Cercosporella herpotrichoides* on potato-dextrose agar [5] are, as previously described (*29*), at first hemispherical mounds of smoke-gray to light grayish-olive velvet-nap mycelium with paler margins. About a week after the fungus is transferred and incubated at 20° C., hyphae begin to grow out from the mounds of mycelia over the surface of the medium. As compared with associated fungi, *C. herpotrichoides* is slow-growing. At a temperature of 21°, colonies of *Wojnowicia graminis* in several dozen petri-dish cultures averaged 74 millimeters in diameter in 11 days, whereas corresponding colonies of *C. herpotrichoides* averaged only 31 millimeters in diameter. The cultures produce small spherical sclerotial aggregates of dark macrohyphae that superficially resemble fruiting bodies.

Wojnowicia graminis differs from *C. herpotrichoides* in forming a looser growth which is olive-brown to olivaceous-black on potato-dextrose agar.

SPORULATION

The appearance of fruiting cultures was briefly mentioned in an earlier article (*29*). The fungus was grown in flasks on sterilized corn meal, and after the usual hemispherical mounds had developed in the laboratory the cultures were placed outdoors in the late fall in temperatures fluctuating between −4° and 16° C. A darker growth immediately started, the mycelium lost its velvety nap, became looser and coarser, and varied in color through several shades of gray to fuscous, with an ultimate tendency to be lighter in color. The coarsening proceeded variably; sometimes loose coremiumlike strands were formed, or more commonly the hyphae gradually clumped together into gray to black microsclerotia or flat sporodochia as the fungus progressed across the surface of the medium. The extreme condition occurred as slimy masses of flesh-pink to carrot-red conidia similar to Sherbakoff's pseudopionnotes (*27*). All of these types were found together or separately. Sometimes fans of growth spread out from the margin of the colony. These were frequently pseudopionnotes, or more commonly they were olive-green to black, flat, stromatic, or chlamydosporelike growths resembling saltants, such as have been observed in *Ascochyta pinodella* (*22*). Spores are produced in tremendous quantities in the pseudopionnotes and on sporodochia. Some cultures produced so little vegetative mycelium that they resembled colonies of bacteria rather than those of fungi. In fact, they were considered as contaminated with bacteria until examination proved the slimy masses were quantities of spores.

Sporulating cultures change the color of the corn meal from a characteristic light maize yellow to a bright lemon chrome. This chromatic development starts at the advancing edge of the colony and gradually extends deep into the medium.

Cultures left outdoors for some weeks and returned to a warm temperature (20° C.) revert to the compact mycelium with velvety nap and tend to lose their chromatic proclivities.

SPORE GERMINATION

The spores studied were obtained from viable sporulating pure cultures. At a temperature of 20° C., germ tubes appear first from the

[5] Difco potato-dextrose agar, pH 5.5, was used.

end cells and later from any cell in the spore (fig. 3). Spores germinate readily, if fresh, either in potato-dextrose agar or in Ranker's medium (*26*), but growth is slow. In one series the germ tubes were only twice the length of the spores at the end of 36 hours, and after 45 hours the mycelia were only 10 times as long as the original spore. After 3 days the colonies were visible to the naked eye, and after 5 days they had developed sufficiently to be recognized as typical *Cercosporella* colonies.

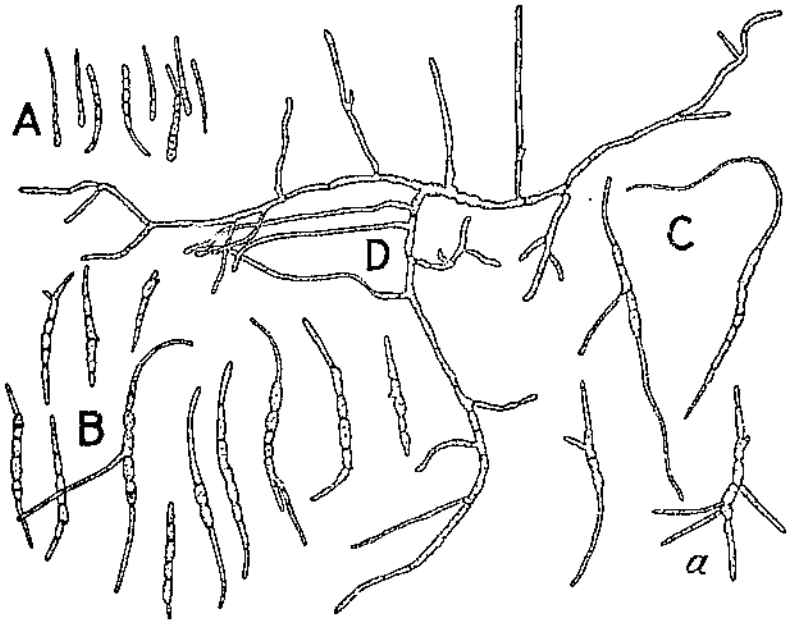


FIGURE 3.—Stages in the germination and early growth of spores of *Cercosporella herpotrichoides* obtained from pure cultures on sterile corn meal and germinated at 20° C. in potato-dextrose agar (pH 5.5). × 420. *A*, Ungerminated spores as taken from corn-meal culture. *B*, Germinating spores 18 hours after transfer to potato-dextrose agar. *C*, Germinating spores 24 hours after transfer to potato-dextrose agar. The spore shown at *a* has several secondary conidia that germinated immediately and lost their identity. *D*, Growth 42 hours after the spore had been transferred to potato-dextrose agar and just before the colonies became visible to the naked eye. Note increase in size of original spore over that of spores shown in *A*. The 6 original cells lie just above the letter *D*. The central cross walls had ruptured by this time.

PATHOGENICITY

ARTIFICIAL INOCULATION STUDIES

As already stated, in order to obtain infection by pure culture inoculation it is necessary to grow the wheat under cool moist conditions. The negative results of previous years may be attributed to the fact that the inoculated plants were kept in greenhouses where the temperatures averaged too high and the humidity was too low.

The result of preliminary inoculation work was briefly reported previously (*29*). Since that time additional and more extensive inoculation studies in the greenhouse and in the field at Corvallis, Oreg., and in fields adjacent to the foot-rot areas have proved that the fungus is a destructive parasite. An example of the severity of attack is shown in table 10. Cereals were sown in Amity silt loam at Corvallis in the fall of 1930, preceding a mild winter. Plants were inoculated in February by laying the oats-barley inoculum at the base of the culms, the soil being too moist to handle at the time. A strip

of grain through the plot was inoculated, and by harvest this grain and a considerable quantity on an adjacent area had nearly all fallen, and almost 100 percent of the culms were diseased. A parallel uninoculated series grown about 200 feet away produced no disease.

The resistance of spring-sown grain has already been discussed in part under host range. Most of the experimental field inoculations on spring-sown grain have been unsuccessful. Spring grain inoculated with pure cultures of *Cercosporella herpotrichoides* in late March was free from disease except in one instance where a 2½-percent infection was obtained at High Prairie, Wash. The winter grain surrounding the plots showed 50 to 100 percent of disease by culm count, and when inoculum was added to some of this naturally infected winter grain it was so severely injured that most of it failed to head.

TABLE 10.—*Result of inoculation on fall-sown cereals in the field at Corvallis, Oreg., on Feb. 4, 1931, with pure cultures of Cercosporella grown on sterile oats and barley kernels*

[Records taken June 25, 1931]

Variety [1]	C.I. no.	Fallen culms	Relative severity of foot rot	Culms			
				Total	Healthy	Diseased	
		Percent		*Number*	*Number*	*Number*	*Percent*
Hybrid 128 wheat	4512	60	Severe	59	3	56	94.9
Hosar wheat	10067	90	do	134	0	134	100.0
Queen Wilhelmina wheat	11389	35	do	122	0	122	100.0
Wheat×rye-wheat	8800	100	do	90	0	90	100.0
Alstroum spelt	1773	93	do	120	0	120	100.0
Wheat×wheat-*Aegilops*		80	do	116	0	116	100.0
Hohenheimer (pubescent) wheat	11459	75	do	85	0	85	100.0
Aegilops triuncialis		50	Moderate	108	0	108	100.0
Einkorn	2433	0	Slight	104	4	107	96.4
Black Winter emmer	2337	45	Moderate	104	0	104	100.0
Hybrid 128 wheat	4512	95	Very severe	110	0	110	100.0
Minhardi×Minturki wheat	8215	100	do	97	0	97	100.0

[1] The varieties are listed in the order in which they were sown in the field.

INOCULATION WITH NATURALLY INFESTED SOIL

Soil from fields known to be infested with *Cercosporella herpotrichoides* was transported to Corvallis, Oreg. Seed of Hybrid 128 wheat was sown in boxes containing this soil and incubated out of doors. Cercosporella foot rot was readily produced.

After preliminary work had shown that the disease would develop under these conditions, wheat was sown in heavy silt loam at Corvallis, and covered with 2 inches of soil from a diseased field in Klickitat County, Wash. As early as November there were indications of injury, and by February, after an open winter, characteristic eye-spot lesions were photographed. Isolations were positive. In June a count disclosed that all varieties in the plot were almost 100 percent diseased and that 70 percent of the culms had fallen and were only two-thirds as tall as those of the checks. All the diseased plants were a peculiar dull green-yellow (pl. 5).

On the basis of these results, studies of varietal resistance were started at Corvallis during 1931–32 and are still in progress. In these studies the grain was sown in 16-foot rows in Newberg sandy loam soil, and each row was inoculated with a shovelful of infested soil from Klickitat County, Wash. Infections resulting in an average injury of 30 percent were obtained the first season.

The disease also develops on wheat grown in naturally infested soil in the greenhouse when temperature and humidity are favorable.

ROD-SQUARE PLOTS OF SEVERAL VARIETIES OF WHEAT AT CORVALLIS, OREG., JUNE 15, 1931.

A, Uninoculated. *B*, Inoculated at seeding time by covering seed with soil from High Prairie, Wash., infested with *Cercosporella herpotrichoides* showing 100 percent of cercosporella foot rot, severe stunting, and many fallen culms.

DISSEMINATION AND OVERWINTERING

Field studies on the role of the spores in the dissemination of *Cercosporella herpotrichoides* have not progressed beyond the preliminary stages.

During the early spring, spores are common on diseased plants in the field, sometimes being produced in great abundance. These spores doubtless have much to do with the sudden increase of infection after heavy rains in the spring. The writers have had difficulty in greenhouse experiments in keeping uninoculated check plants free from infection when placed near inoculated plants. Spores are carried by splashing water. Uninoculated checks kept on benches other than those on which the inoculated plants were kept rarely became infected. There was some evidence that the spores were air-borne in a few cases. Foëx and Rosella (*11*) state that there is evidence that the spores may be air-borne. However, this point needs further study.

During the winter of 1930–31 wheat plants 2 months old were inoculated with spores from a pure culture of *Cercosporella* on corn meal and incubated in a moist cool greenhouse. Typical eye-spot lesions developed in 4 weeks.

Spores kept indoors a few days germinate sparsely and in many cases fail to germinate at all, which suggests that they are not long-lived.

As previously mentioned, the fungus survives the winter on diseased stubble standing or lying in the field. Attempts to isolate the fungus directly from the soil have been unsuccessful.

CONTROL MEASURES

A brief summary of the data on control of cercosporella foot rot is presented in the following paragraphs.

CHEMICALS

Numerous chemicals have been tried for controlling the soil-borne infestation of *Cercosporella herpotrichoides*. While some of these have caused material reduction in the severity of the foot rot, none has given promise of commercial success. The chemicals tried most extensively were organic mercury dusts, alum, aluminum sulphate, mercuric chloride, copper sulphate, flowers of sulphur, bordeaux mixture, and borax.

FERTILIZERS AND AMENDMENTS

There are many references to the use of commercial fertilizers for controlling foot-rot diseases of cereals. Hamblin (*17*) suggested the use of superphosphate at the rate of 156 pounds per acre for control of the Helminthosporium disease in Australia. Martinet (*24*) and Guerrapain and Demolon (*15*) cautioned against the application of excessive quantities of nitrogen, and Gaudineau and Guyot (*14*), in studying the complex of foot rots in northern France, found that lime, iron sulphate, sulphur, superphosphate, and sodium nitrate had no appreciable effect on the disease or diseases. The nitrate fertilizers, however, did not increase the severity of the disease, as had been suggested. Parisot (*25*), who apparently holds that nutrition is the entire cause of soil-borne foot-rot diseases, found that phosphate fertilizers tended to stabilize the growth of the host plant and to reduce foot rot (piétin) in general. According to Parisot,

the heavy application of nitrogen and potassium fertilizers, which tends toward a sudden revival of growth, increased foot rot. Biffen (*1*) reported that a foot rot possibly caused by *Leptosphaeria herpotrichoides* was especially bad on ammonium-sulphate plots. Guyot (*16*) found that nitrate of soda increased foot rot more than the increased yield warranted. She suggested spring application as the safer method. An application in October of approximately 134 pounds per acre (150 kilos per hectare) of potassium nitrate and ammonium phosphate increased foot rot only slightly and increased yield considerably. Foëx (*6*) recommended nitrate of soda and also iron sulphate at the rate of approximately 888 pounds per acre (1,000 kilos per hectare) for controlling foot rot. He mentioned that in this case *Cercosporella herpotrichoides* Fron was found associated both with *L. herpotrichoides* and *Ophiobolus graminis*.

Experiments conducted in the field with standard commercial fertilizers applied in the fall have been repeated many times in all of the major cercosporella foot-rot areas in the Pacific Northwest. The results will not be given in detail. No fertilizers have consistently shown any value in reducing the severity of the foot rot. On the contrary, heavy applications in the fall of fertilizers rich in nitrogen increase the disease. Spring applications of commercial fertilizers have not had any appreciable effect in reducing the foot rot.

Experiments were conducted also in the greenhouse. Various quantities of different commercial fertilizers were added to Newberg sandy loam soil in cubic-foot boxes and 12-inch glazed drainage tiles, the checks being left without fertilizer. Half of the containers were not inoculated, and the other half were inoculated with *Cercosporella herpotrichoides* at seeding time. All were sown to Hybrid 128 wheat. In the soils to which lime or potash was added there was considerable reduction in the percentage of injury from foot rot, and in the soil to which superphosphate was added there was only 5 percent injury in comparison with 68 percent in the untreated checks. This did not agree with the results obtained in the field. The soil used in the greenhouse tests, however, was strongly acid, whereas the soils in the field tests were neutral or only slightly acid. This suggests that on strongly acid soils superphosphate might be of some value in controlling foot rot. In the United States, however, there are no strongly acid soils now known to be infested with *C. herpotrichoides*.

BURNING THE STUBBLE AND HEATING THE SOIL

At La Grande, Oreg., in 1921, 52 percent of cercosporella foot rot was found in a portion of a field from which the stubble had been burned after harvest. An unburned check area showed 54 percent. This indicated little effect from the first season's burning at least.

In September 1929 1 square rod in an infested field near Alicel, Oreg., was covered with a bale of straw and burned over. On the following day Goldcoin wheat was sown on this plot with a hand seeder, and within a few days the same variety was sown on the surrounding commercial field. Early in July 1930 the burned-over plot showed 13.1 percent of foot rot and the surrounding unburned field 69.9 percent.

In order to determine whether the presence of ashes on the surface of the soil was in any way responsible for the decrease in the severity of the disease in the burned portion, an experiment was conducted in

an infested field of Hybrid 63 on Tygh Ridge, Oreg., in the fall of 1930. Straw ashes were mixed into the soil between the wheat plants just after they had emerged. Data taken in July 1931 showed 23.2 percent of injury from foot rot where ashes had been added and 23.5 percent in adjacent untreated portions. It is evident, therefore, that the straw ashes did not reduce foot rot.

On the plot near Alicel in 1929–30 the fire no doubt destroyed the fungus at the surface of the soil, and because of the shallow seeding there probably was no mixing with subsurface-infested soil. The cumulative effect of burning year after year should be studied.

EFFECT OF HARROWING

Field observations have shown that harrowing in the spring increases foot rot slightly if followed by rainy weather; when followed by dry weather, harrowing aids in aerating the topsoil and there is a reduction in the amount of foot rot. It is not desirable to harrow too early in the spring when the soil is most moist and when the likelihood of immediate rains is greater.

TIME OF SEEDING

Evidence not presented here has shown clearly that, other things being equal, the earlier the grain emerges in the fall the greater is the amount of disease that may be expected.

DISTANCE BETWEEN ROWS AND USE OF DEEP-FURROW DRILLS

All experiments show that increased aeration reduces the amount of cercosporella foot rot. During seasons of excessive rainfall, when the growth of wheat is rank, there is no appreciable difference in severity of foot rot in rows 8, 10, 12, or even 14 inches apart. In seasons of reduced rainfall wider spacing decidedly reduces foot rot because of increased aeration. Growers in the Grand Ronde Valley of Oregon are taking advantage of this and are seeding with every other drill row blank. They are getting splendid results in controlling the disease in years when the precipitation is not excessive.

The deep-furrow drill has no value in controlling foot rot. In an experiment conducted on Seven Mile Hill, Oreg., four varieties were sown on four dates in the fall of 1928. In July 1929 the sowings averaged 16 percent of foot rot in the standard drill rows and 17.4 percent in the deep-furrow drill rows. A similar experiment conducted the same year at Imbler, Oreg., gave 76.2 and 78.7 percent of foot rot, respectively, for the two sowings.

RESISTANT VARIETIES

Information on varietal resistance is presented earlier in this bulletin. Hybrid 128, while moderately susceptible, is the most satisfactory wheat to grow on soil infested with *Cercosporella herpotrichoides*. A number of varieties have shown less injury from cercosporella foot rot than has Hybrid 128, but they have not been tested sufficiently to warrant recommendation for commercial growing.

CROP ROTATION

The writers have made general observations on the influence of crop rotation on the severity of cercosporella foot rot, but no actual rotation plots have been available for critical study.

Fortunately, foot rot occurs in areas having slightly more annual precipitation than much of the wheatland in the Pacific Northwest. For that reason, crop rotation can be practiced to some extent on most of the foot-rot-infested land.

SPOKANE RIVER VALLEY AND ADJACENT PRAIRIES

On Peone Prairie the lower ground in fields can be sown to spring grain or legume crops with success (pl. 6). Foot rot, once destructive, is now of comparatively minor importance over much of this locality. In Spokane Valley proper the problem is more acute, and rotation in the rocky soil is not so practicable. Where possible, spring grains, field peas, or sweetclover should be sown.

UNION COUNTY, OREG.

Spring-sown cereals, such as Federation wheat, oats, and barley, may be grown on some of the land in Union County, near Elgin, and to a smaller extent on Sand Ridge, near Imbler and Alicel. Alfalfa, sweetclover, and other legumes (except vetch) should replace winter wheat on certain fields in the Sand Ridge area. Fall-sown White Club (Winter Club) barley will come through a majority of the winters yielding a good crop of grain on foot-rot-infested ground.

COLUMBIA GORGE DISTRICT

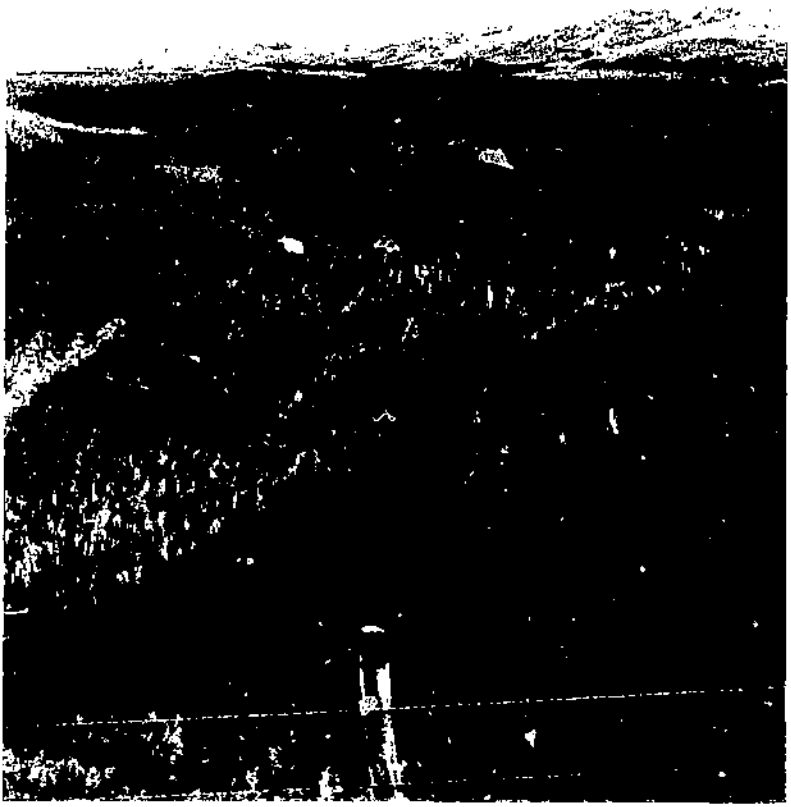
Crop rotation is more difficult in the slightly drier Columbia Gorge district. On Seven Mile Hill, Wasco County, Oreg., leguminous crops do fairly well and spring grains can be grown to some extent. These crops are profitable only in seasons when moisture is relatively abundant. However, they cannot entirely supersede winter wheat. On Tygh Ridge, also in Wasco County, in another strictly winter-wheat area, the worst spots of foot-rot-infested soil could well be held over until spring and sown to Federation wheat. On High Prairie winter barley is worth a trial on foot-rot-infested ground. Alfalfa and sweetclover should be used on spots that are chronically infested with the foot rot. The occasional seeding of the usually very badly infested hay strip with some legume is a good practice.

SUMMARY

A foot-rot disease of winter wheat and winter barley caused by the fungus *Cercosporella herpotrichoides* Fron is important in certain portions of the Columbia Basin in Oregon, Washington, and Idaho. The disease is also known to occur in France.

The fungus attacks the base of tillers in early spring, forming elliptical eye-spot lesions on the leaf sheaths. It penetrates into inner tissue, forming lesions in the first or second basal internode of the developing culms. Stromata are formed on the lesions, giving them a charred appearance. The lesions later shrivel, and the culms may buckle and fall under the weight of the maturing head, especially in the case of wheat. *Cercosporella herpotrichoides* usually does not cause severe stunting of the attacked plants nor does it attack the roots of such plants, two characteristics that distinguish it from *Ophiobolus graminis*.

The degree of injury caused each year by *Cercosporella herpotrichoides* varies considerably with seasonal climatic conditions, crop practices, and the application of control measures. The loss by this



METHOD OF CONTROL PRACTICED ON PEONE PRAIRIE, SPOKANE COUNTY, WASH., 1932.

Cercosporella-infested soil in the depression is sown to spring wheat and the uninfested soil on the higher ground is sown to winter wheat.

foot rot is due to reduction in the number of culms, decrease in head size and grain weight, and increased difficulty of harvesting the fallen, irregularly ripening grain. *C. herpotrichoides* is mainly confined to fine or very fine sandy loam prairie soils.

Many varieties of wheat, barley, and rye, and a number of grasses are susceptible to *Cercosporella herpotrichoides*, whereas oats are nearly immune under artificial inoculation conditions. No wild hosts are known. No wheat varieties of commercial importance were found to be resistant to the disease.

The fungus enters either directly through the cell walls of the host or through stomatal openings. It invades intracellularly and forms external and internal stromata on the host. Histological studies of the disease are discussed on pages 9 to 13.

It is shown that the primary invader in the early spring in the plants infected with foot rot is *Cercosporella herpotrichoides*. The conidial stage, which is the only known spore stage, develops on corn-meal cultures incubated under favorable conditions for temperature and humidity. It occurs on infected plants in the field in early spring. The sporulating cultures are fully described. The conidia germinate slowly and after several days produce the smoke-gray colonies characteristic of *C. herpotrichoides*.

Under conditions at Corvallis, Oreg., cereals were severely injured by cercosporella foot rot in naturally infested soil in a cool-temperature greenhouse held at 4° to 20° C. Similar results were obtained in localities in eastern Oregon.

The fungus lives over winter on infected stubble. Spores transmit the disease, although their importance is not known.

Crop rotation, resistant varieties, and time of seeding are important factors in the control of the disease. Fertilizers and chemicals have little value in controlling the disease in the field.

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