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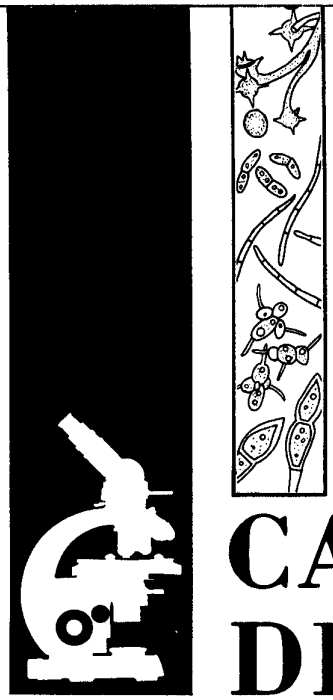
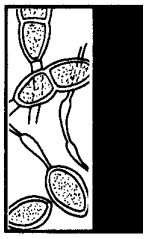


CANADIAN PLANT DISEASE SURVEY



EDITOR W.L. SEAMAN

Agriculture Canada

Research Branch

VOL.52, No.4, DECEMBER, 1972

CANADIAN PLANT DISEASE SURVEY

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

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CONTENTS

"The Canadian Plant Disease Survey is a periodical of information and record on the occurrence and severity of plant diseases in Canada. It will also accept other original information such as the development of methods of investigation and control, including the evaluation of new materials. Review papers and compilations of practical value to phytopathologists will be included from time to time."

LOSSES FROM CEREAL DISEASES IN MANITOBA IN 1971[1]

W.A.F.Hagborg, A.W.Chiko, G. Fleischmann, C.C.Gill,
G.J.Green, J.W.Martens, J.J.Nielsen, and D.J.Samborski

Introduction

It was pointed out previously by McDonald et al. (8) that data on losses caused by plant diseases have become increasingly important to agencies such as FAO, USDA, and CDA in the setting of priorities in research programs. The objective sought in a three-year study initiated by this station in 1969 was to appraise the degree of reduction of crop yield from various diseases and to place a monetary value on the gains achieved by growing the current resistant varieties. By following similar procedures in successive years in the same territory observations could be made on consistencies and discrepancies. To this end the disease loss study in cereal crops begun by McDonald et al. in 1969 (8) in Manitoba and in 1970 extending also over a part of Saskatchewan (9) was continued in the present study in 1971 in Manitoba only.

Methods

The procedure followed was essentially the same as in 1969 and 1970. Observations were made over the same six survey routes as previously followed in Manitoba. Because the 1971 provincial agricultural census was not available, the 1966 census (3) was again used to ascertain the number of survey sites required in each district, as described previously by McDonald et al. (8), and in general close agreement was achieved (Table 1).

The surveys were made from July 26 until August 10. Crop maturity varied from the heading to the dough stage but the data were adjusted to be equivalent to the milky-ripe stage for the conversion of disease incidence into estimated crop damage. To facilitate comparisons, the results in the 12 crop districts surveyed were grouped into five larger areas as in 1969 and 1970.

The methods of assessing losses from individual diseases were similar to those used previously (8, 9). Field readings of leaf rust of wheat and crown rust of oats, made as a percentage rating based on the modified Cobb scale, were adjusted according to the maturity of the crop. To those made before the milk stage 20% was added; from those made after the milk stage, 20% was subtracted. The adjusted reading was then referred to Chester's Table 4 (1) to find the estimated percentage loss.

The loss from leaf spotting diseases such as infection by bacteria, Septoria spp., Helminthosporium spp., and Rhynchosporium secalis, was derived from the formula

$$\frac{2/3 \text{ infection \% flag leaf} + 1/2 \text{ infection \% 2nd leaf}}{2}$$

This formula was adapted from the work of James et al. (7) with Rhynchosporium secalis infection in barley.

The potential average yield in each area was estimated by use of the formula

$$\frac{100 \times \text{mean yield}}{100 - \text{\% loss from all diseases}}$$

The loss in bushels was calculated by multiplying the mean percentage loss attributable to diseases by the potential yield per acre times the acreage.

The methods of assessing losses from individual diseases and of assessing gains from disease resistance were similar to those used previously (8). In the 1971 Western Wheat Cooperative Tests, the mean yields in cwt/acre from nine stations in the rust area (Glenlea, Morden, Portage la Prairie, Brandon, Indian Head, Melfort, Regina, and Saskatoon) were: 'Manitou', 32.1; 'Thatcher', 32.1; and 'Marquis', 27.7. The mean yields from ten stations in the adjoining rust free area (Edmonton, Evansburg, Beaverlodge, Kindersley, Scott, Acme, Lethbridge, and Swift Current) were: 'Manitou', 30.1; 'Thatcher', 30.4; and 'Marquis', 28.8. The gain in production from leaf rust resistance was calculated by subtracting the difference between the mean yields of 'Manitou' and 'Thatcher' in the non-rust area from the difference between the mean yields of 'Manitou' and 'Thatcher' in the rust area and expressing this quantity as a percentage of the yield of Manitou in the rust area.

A somewhat parallel calculation was made for the gain in production from stem-rust resistance. The difference between the mean yields of 'Thatcher' and 'Marquis' in the

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Table 1. Number of commercial farms in Manitoba with specific crops, number of survey sites in 1971 and percentage of farms surveyed by crop district

Area and crop district	Wheat			Oats			Barley		
	Number of farms*	Farms surveyed (no.)	(%)	Number of farms	Farms surveyed (no.)	(%)	Number of farms	Farms surveyed (no.)	(%)
East									
4	513	7	1.4	605	8	1.3	403	7	1.7
5	1750	14	0.8	1880	5	0.3	843	7	0.8
6	96			189			29		
12	600			728			321		
Total	2959	21	0.7	3402	13	0.3	1596	14	0.9
Central									
3	3723	18	0.5	3204	18	0.6	1649	18	1.1
Southwest									
1	1544	13	0.8	1207	12	0.9	586	5	0.9
2	2335	14	0.6	1913	13	0.7	994	10	1.0
7	1422	11	0.8	1299	10	0.8	632	12	1.9
Total	5301	38	0.7	4419	35	0.8	2212	27	1.2
West-central									
8	1541	13	0.8	1427	12	0.8	571	10	1.8
9	1360	10	0.7	1276	12	0.9	571	8	1.4
10	2543	20	0.8	1852	20	1.1	1465	18	1.2
14	599	4	0.7	630	3	0.5	273	3	1.1
Total	6043	47	0.8	5185	47	0.9	2880	39	1.4
Northwest									
11	1818	2	0.1	1489	4	0.3	810	5	0.6
13	932	7	0.8	656	6	0.9	702	6	0.9
Total	2750	9	0.3	2145	10	0.4	1512	11	0.7

* Based on 1966 farm census.

non-rust area was subtracted from the difference in the mean yields of 'Thatcher' and 'Marquis' in the rust area and this subtracted quantity was expressed as a percentage of the yield of Manitou in the rust area. This percentage was applied to the production of wheat "Manitou predominating) in Manitoba to find the gain in production attributable to the stem rust resistance in 'Manitou'.

For the probable losses that would be caused in susceptible cereals as compared with the resistant varieties sown in Manitoba, the methods of estimating used by McDonald et al. (8) were adopted.

Results

Wheat

Yield losses from the diseases of wheat that occurred in Manitoba in 1971 amounted to 6.4 million bushels or 8.8% of the potential yield without disease (Table 2).

Most of the wheat acreage surveyed was sown to varieties that were resistant to stem rust (Puccinia graminis Pers. f. sp. tritici Erikss. & Henn.) and losses from this source were of no significance.

Bacterial black chaff [Xanthomonas translucens f. sp. undulosa (Smith, Jones, & Reddy) Hagborg and X. translucens f. sp. cerealis Hagborg] caused a loss of 2.7 million bu or 3.7% of the potential production. Previously this disease had been included under leaf spots but in 1971 it was recorded separately. The high frequency of mid-season rains probably resulted in more bacterial infection than average. A particularly severe infection, not included in the disease loss survey, was noted in the second half of August in an area north and west of Portage la Prairie. In this district every one of 13 fields examined had bacterial black chaff present. Leaf area destruction ranged from 15% to 100% with a mean value of 65%. Glume discoloration was less destructive but in some fields was recorded on 100% of the heads.

Leaf rust was the second most important single disease, causing losses of 1.1 million bu or 1.6% of the potential production.

Leaf spots caused by Dreschslera tritici-repentis (Died.) Shoem., Bipolaris sorokiniana (Sacc. in Sorok.) Shoem., and Septoria avenae Frank. f. sp. tritici T.

Table 2. Yield losses from diseases in wheat in Manitoba, 1971

Area (crop districts)		Yield losses from: Leaf rust	Bacterial black chaff	Leaf spots	Viruses	Root rot	Total loss	Average yield (bu/ac)	Potential avg yield (bu/ac)	Acres ('000)	Potential production ('000 bu)
East	Range (%)	0-6	0-28	0	0	0					
(4, 5)	Mean (%)	0.8	5.6	0	0	0	6.4	27.4	28.7	357	10,246
	Bu* ('000)	82	574	0	0	0	656				
Central	Range (%)	0-12	0-13	0-35	0	0-1					
(3)	Mean (%)	2.5	1.7	4.9	0	0	9.1	28.9	31.8	499	15,868
	Bu ('000)	397	270	778	0	0	1444				
Southwest	Range (%)	0-8	0-20	1-20	0-tr†	0-6					
(1, 2, 7)	Mean (%)	1.8	4.6	3.7	0	0.3	10.4	29.0	32.4	637	20,639
	Bu ('000)	372	949	764	0	62	2146				
West-central	Range (%)	0-15	0-18	0-20	0	0-tr					
(8, 9, 10)	Mean (%)	1.4	3.0	2.3	0	0	6.7	29.9	32.0	543	17,376
	Bu ('000)	243	521	400	0	0	1164				
Northwest	Range (%)	0-3	0-12	0-20	0	0-tr					
(11, 13, 14)	Mean (%)	0.5	4.0	6.9	0	0	11.4	29.8	33.6	268	9,005
	Bu ('000)	45	360	621	0	0	1027				
Total ('000 bu)		1139	2674	2563	0	62	6437	28.9	31.7	2304	73,134
% of potential production		1.6	3.7	3.5	0	0.1	8.8				

* Bu = bushels

† tr = trace

Johnson, caused losses of 2.6 million bu or 3.5% of the potential production. No losses from smuts and virus diseases were recorded and the losses from root rot were light at 0.1%.

Oats

Losses from diseases in oats were 1.6 million bushels or 2.2% of the potential production for Manitoba. Stem rust was found in trace amounts at only two locations, but crown rust reduced the yield by 0.7 million bu or 0.9% of the potential production (Table 3).

Leaf spots, caused by Drechlera avenacea (Curt. ex Cke.) Shoem., Septoria avenae Frank f. sp. avenae Conners and Pseudomonas coronafaciens (Elliott) Stevens also reduced the yield by 0.7 million by or 0.9% of the potential production. Colletotrichum graminicola (Ces.) G. W. Wilson made an erratic appearance varying from absent to as high as 20% of the leaf area affected. Virus diseases, viz. barley yellow dwarf, oat blue dwarf, and aster yellows, caused an estimated 0.1 million bu loss (0.1%) and blast a 0.2 million bu loss (0.3%). No losses were recorded for smuts.

Table 3. Yield losses from diseases in oats in Manitoba, 1971

Area (crop districts)		Yield losses from: Crown rust	Leaf spot	Viruses	Blast	Total loss	Average yield (bu/ac)	Potential avg yield (bu/ac)	Acres ('000)	Potential production ('000 bu)
East	Range (%)	0-10	0-tr	0-tr	0-tr					
(4, 5)	Mean (%)	1.5	0	0	0	1.5	50.7	51.5	306	15,759
	Bu ('000)	236	0	0	0	236				
Central	Range (%)	0-10	0-tr	0-4	0					
(3)	Mean (%)	1.4	0	0.2	0	1.6	54.9	55.8	274	15,289
	Bu ('000)	214	0	31	0	245				
Southwest	Range (%)	0-3	0-10	0-1	0-5					
(1, 2, 7)	Mean (%)	0.6	0.3	0.2	0.1	1.2	51.6	52.2	337	17,591
	Bu ('000)	106	53	35	18	211				
West-central	Range (%)	0-14	0-10	0-0.7	0-10					
(8, 9, 10)	Mean (%)	0.7	0.5	0	0.3	1.5	50.8	51.6	344	17,750
	Bu ('000)	124	89	0	53	266				
Northwest	Range (%)	0	0-10	0-tr	0-5					
(11, 13, 14)	Mean (%)	0	5.7	0	1.4	7.1	45.7	49.2	197	9,692
	Bu ('000)	0	552	0	136	688				
Total ('000 bu)		680	694	66	207	1646	51.1	52.2	1458	76,081
% of potential production		0.9	0.9	0.1	0.3	2.2				

Table 4. Yield losses from diseases in barley in Manitoba, 1971

Area (crop districts)		Yield losses from: Viruses	Leaf spot	Thrips	Leaf rust	Stem rust	Smut	Root rot	Total yield loss	Average yield (bu/ac)	Potential avg yield (bu/ac)	Acres ('000)	Potential production ('000 bu)
East	Range (%)	0-1	0-13	0-1	0	0	0	0					
(4, 5)	Mean (%)	0.2	3.5	0.1	0	0	0	0	3.8	43.9	45.6	289	13,178
	Bu ('000)	26	461	13	0	0	0	0	501				
Central	Range (%)	0-2	0-15	0-1	0-tr	0	0-5	0					
(3)	Mean (%)	0.3	3.3	0.1	0	0	0.3	0	4.0	46.6	48.5	409	19,837
	Bu ('000)	60	655	20	0	0	60	0	793				
Southwest	Range (%)	0-3	0-30	0-5	0-tr	0	0-tr	0					
(1, 2, 7)	Mean (%)	0.3	3.3	0.4	0	0	0	0	4.0	46.7	48.6	585	28,431
	Bu ('000)	85	938	114	0	0	0	0	1137				
West-central	Range (%)	0-0.7	0-30	0-5	0-tr	0	0-13	0					
(8, 9, 10)	Mean (%)	0	2.8	0.4	0	0	0.8	0	4.0	47.3	49.2	482	23,714
	Bu ('000)	0	664	95	0	0	190	0	949				
Northwest	Range (%)	0-tr	0-27	0-tr	0	0	0-10	0-tr					
(11, 13, 14)	Mean (%)	0	9.6	0	0	0	1.4	0	11.0	41.9	47.1	361	17,003
	Bu ('000)	0	1632	0	0	0	238	0	1870				
Total ('000 bu)		171	4350	242	0	0	488	0	5250	45.6	48.1	2126	102,163
% of potential production		0.2	4.3	0.2	0	0	0.5	0	5.1				

Barley

Yield losses in barley from disease amounted to 5.3 million bu (5.1%) and from damage by thrips 0.2 million bu (0.2%, Table 4).

Of all diseases, leaf spots incited by Bipolaris sorokiniana, Drechslera teres (Sacc.) Shoem., Septoria passerinii Sacc., Puccinia hordei Otth., Rhynchosporium secalis (Oud.) Davis, and Xanthomonas translucens f. sp. hordei-avenae Hagborg were the most destructive, resulting in a loss of 4.4 million bu (4.3% of the potential yield). Smuts caused a loss of 0.5 million bu (0.5%) and viruses 0.2 million bu (0.2%). The virus disease most prevalent was barley stripe mosaic and in a special survey Chiko (2) found it in 34% of the fields of 2-rowed barley and 4.5% of the fields of 6-rowed barley.

Value of resistance

The resistance to stem rust, leaf rust, and loose smut in the varieties of wheat presently grown combined to make a

Table 5. Value of disease resistance in cereal varieties grown in Manitoba, 1971

Crop	Disease	Loss in* susceptible varieties (%)	Acreage of† resistant varieties (%)	Total** production ('000 bu)	Gain in production ('000 bu)	Price ($/bu)	Value ($000)
Common wheat	Stem rust	8.7	100	70,000	6,090	1.40	8,526
	Leaf rust	0.9	90		567		894
	Loose smut	1.3	100		910		1,274
	Total				7,567		10,694
Oats	Smut	1.2	99	80,000	950	0.50	475
Barley	Smut	2.1	60	100,500	478	0.75	358
Total					8,593		11,523

* See McDonald et al. (8) for method of determining loss in susceptible varieties.

† Seed Time and Harvest. Nos. 117, 119, 122. Federal Grain Ltd., Winnipeg, Manitoba.

** Manitoba Agriculture 1971 Yearbook.

substantial gain in wheat production in 1971 worth over $10 million (Table 5) and when this was added to gains from resistance to the smuts of oats and barley a total was reached of more than $11.5 million.

A summary of the losses from disease and the gains from disease resistance in the varieties grown for the 3-yr period is given in Table 6. It will be noted that the annual saving for the three years is estimated at over $11 million.

Table 6. Yield losses from diseases in wheat, oats and barley in Manitoba, 1969-71

Year	Wheat	Oats	Barley
		bushels	
1969*	3,344,600	5,259,100	4,073,200
1970†	2,837,400	9,906,900	3,992,500
1971	6,437,000	1,646,000	5,250,000
Total	12,619,000	16,812,000	13,315,700
Mean	4,206,333	5,604,000	4,438,567

* Data from McDonald et al. (8).

† Data from McDonald et al. (9).

Discussion

The present paper covers the last phase of a three-year study of disease losses by means of systematic plant disease surveys. Losses from the different diseases, especially the rusts, fluctuated from year to year suggesting that even greater fluctuations might occur in a longer study. Similarly the savings from resistance probably did not reach a maximum. Thus the yields of Marquis wheat in the Western Wheat Cooperative Tests in the "rust area" during the three years, viz. 18.1, 21.2 and 27.7 cwt/ac, did not suggest that rust was potentially severe in any of these years. The same opinion apparently was held by Green (4,5,6). Two noticeable changes in 1971 were the marked drop in virus diseases in barley and the appreciable loss from bacterial black chaff recorded when this disease was separated from other leaf spot diseases.

Satisfactory observations on ergot could not be made at the time of the general disease loss survey as ergot bodies were not evident at this date.

Two general conclusions appear to be obvious from the study. Losses from all diseases, reaching an annual average of 4.2 million bu of wheat, 5.6 million bu of oats, and 4.4 million bu of barley, are great enough to justify considerable research and extension work in their control; and, secondly, the average annual gain in production as a result of the use of the present disease-resistant varieties (Table 7) has amply justified the federal and provincial expenditures on research and extension in Manitoba thus far.

Table 7. Gain in productivity from the use of disease-resistant wheat, oats and barley varieties in Manitoba, 1969-71

Year	Wheat	Oats	Barley	Total
1969	$16,146,100	$ 411,800	$ 466,000	$17,023,900
1970	4,820,700	314,200	487,600	5,622,500
1971	10,694,000	475,000	358,000	11,523,000
Total	31,660,800	1,201,000	1,311,600	34,169,400
Mean	10,553,600	400,333	437,200	11,389,800

Acknowledgments

The authors are grateful for the participation of three visiting scientists, Dr. R. A. Fullerton[2], Dr. M. E. McDaniel[3], and Dr. D. E. Harder[4] in the survey.

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OCCURRENCE AND PATHOGENICITY OF GODRONIA CASSANDRAE F. VACCINII ON LOWBUSH BLUEBERRY IN NOVA SCOTIA[1]

C.L.Lockhart[2] and R.W.Delbridge[3]

Abstract

Cankers caused by Godronia cassandrae f. vaccinii were found on lowbush blueberries (Vaccinium angustifolium) in several areas of Nova Scotia and in one location in Prince Edward Island. An isolate of this fungus from lowbush blueberry was pathogenic on lowbush blueberry, highbush blueberry (V. corymbosum), and cranberry (V. macrocarpon).

In 1970, cankers caused by the fungus Godronia cassandrae (Peck) f. vaccinii Groves (stat. conid. Fusicoccum putrefaciens Shear) (1) were found for the first time in Nova Scotia on lowbush blueberry (Vaccinium angustifolium Ait.). Previously it was reported on lowbush blueberry in Quebec in 1968 (3) and in Michigan (4) in 1969. In Michigan isolates of G. cassandrae from Spiraea spp. and from V. angustifolium were pathogenic to highbush blueberry (4). In Nova Scotia this disease is recognized as a limiting factor in the production of highbush blueberries (2). This study reports on the occurrence of G. cassandrae on lowbush blueberry and on the pathogenicity of an isolate of this fungus from lowbush blueberry on lowbush blueberry, highbush blueberry (Vaccinium corymbosum L.), and cranberry (Vaccinium macrocarpon Ait.).

Isolations from lowbush blueberry

In April 1970, F. putrefaciens was isolated from cankers found on lowbush blueberry plants in a headland bordering a commercial highbush blueberry field at Sheffield in Kings County, N.S. (Figures 1 and 2). At Debert in Colchester County, cankers were found on 5% of the plants in hedgerows and in scattered areas of a poorly burned commercial lowbush blueberry field. Severely infected clones were found by the roadside near Musquodoboit, Halifax County, and on plants near a cranberry bog at Aylesford, Kings County. Recently G. cassandrae f. vaccinii was isolated from a sample of lowbush blueberry plants from a commercial field at Lewes, Prince Edward Island.

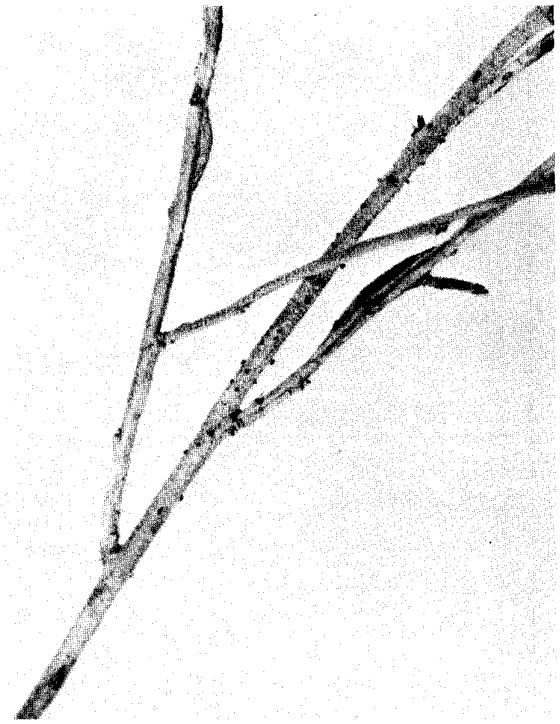

Figure 1. Three-year-old shoot of lowbush blueberry infected with *Godronia cassandrae f. vaccinii*.

Pathogenicity

An isolate of G. cassandrae f. vaccinii from a canker on the lowbush blueberries at Sheffield was used for all pathogenicity tests. The inoculum was grown on lowbush blueberry twigs that had been previously placed in test tubes containing water and sterilized in the autoclave. Twelve plants grown in pots in the greenhouse were wound inoculated by making an incision in the bark with a sterile scalpel and inserting conidia of F. putrefaciens scraped from a twig

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[2] Plant Pathologist, Research Station, Kentville.

[3] Plant Pathologist, Nova Scotia Department of Agriculture and Marketing, Kentville.

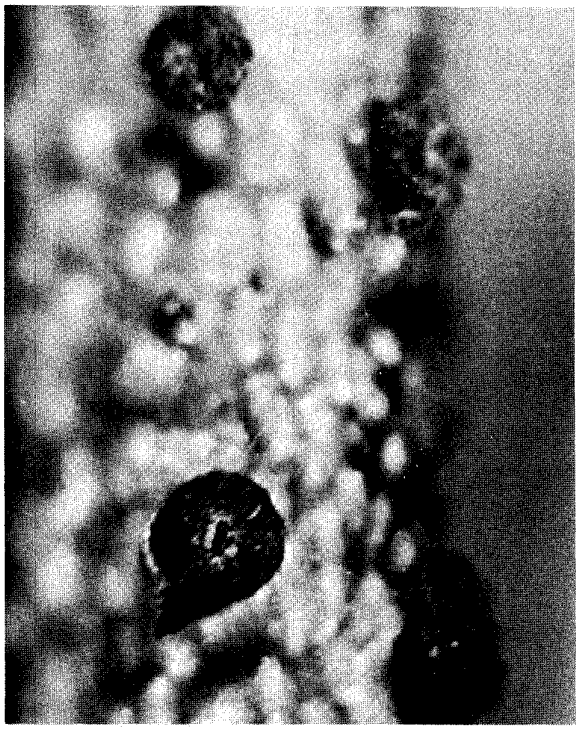

Figure 2. Pycnidia of *Fusicoccum putrefaciens*, the conidial state of *Godronia cassandrae* f. *vaccinii*, on lowbush blueberry.

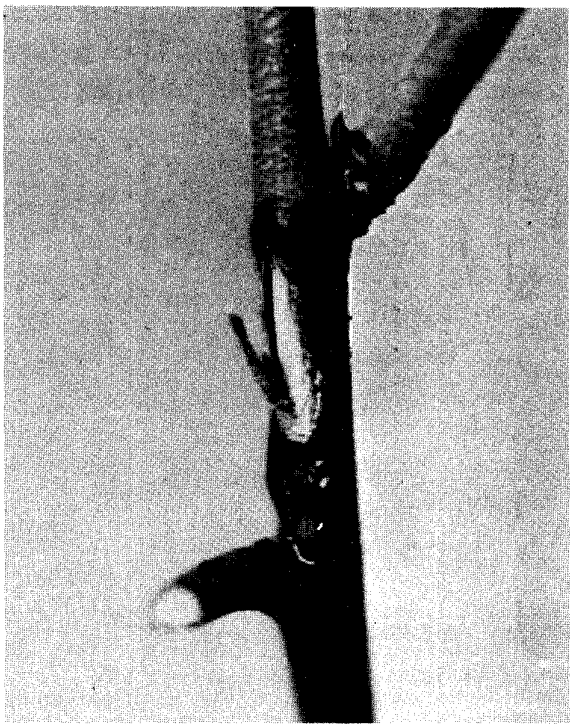

Figure 3. Canker produced on lowbush blueberry artificially inoculated with *Fusicoccum putrefaciens*.

culture. The incisions were wrapped with moistened cotton held in place with cellulose tape. One week after inoculation the cotton was removed. Controls consisted of incisions without inoculum.

Following inoculation, the plants were placed in a growth chamber (Controlled Environments Ltd., EY8VH) under a regime of 18 C days (16 hr) and 10 C nights (8 hr). Light in the day period was provided by six 40-W fluorescent bulbs and eight 100-W incandescent bulbs. Relative humidity was 92% during the day and 98% at night.

After 1 month, cankers were evident on the inoculated plants. After 2 months the plants were transferred to a greenhouse. Nine months after inoculation, cankers 4 to 9 cm long (Fig. 3) had developed. At this time, one of the cankers was dissected and yielded the fungus on isolation. The remaining plants were held for another 5 months, during which the cankers enlarged slightly. No fungus fruiting structures developed on the cankers. No cankers developed on the control plants and the incisions healed completely.

Nine highbush blueberry plants, 3 each of the cultivars Bluecrop, Blueray, and Earliblue were inoculated in the same manner as the lowbush blueberry plants. They were placed in the growth chamber for 6 months and then transferred outdoors in June. Cankers first became evident on Earliblue 8 months after inoculation. At 12 months cankers were 2 cm long on Earliblue and 1 cm long on Bluecrop, and the wood was discolored under these affected areas. No definite cankers developed on Blueray but the fungus was readily isolated from the inoculated areas on this cultivar and from the cankered areas on the other two cultivars.

Nine cranberry plants, cultivar Stevens, were inoculated in the same manner as the blueberry plants and held in a growth chamber for 7 months. By 1 month, swollen callus tissue had surrounded the incisions and by 7 months this tissue was 1 cm in length, but these areas did not appear to have healed. Pycnidia of F. putrefaciens developed on one infection site and the fungus was isolated from all the inoculated plants. The control incisions calloused over but did not yield the fungus on isolation.

Discussion

G. cassandrae f. vaccinii has been found to be confined to lowbush blueberries on headland areas, roadsides, or improperly burned lowbush blueberry fields. The

practice of burning commercial lowbush blueberry fields every two or three years, apparently has effectively controlled this disease. The findings from these experiments confirm those of Weingartner (4), indicating that infected lowbush blueberry plants serve as a reservoir of the fungus for this disease on highbush blueberry. Its status as a canker causing organism on cranberry is not known.

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EFFECTS OF NATURAL INFECTIONS OF CROWN RUST AND STEM RUST ON YIELD AND QUALITY OF OATS IN MANITOBA[1]

J.W.Martens, G. Fleischmann, and R.I.H.McKenzie

Abstract

Losses in groat yield and quality of oats caused by crown rust, Puccinia coronata Cda. f. sp. avenae Eriks., and stem rust, P. graminis Pers. f. sp. avenae Eriks. & E. Henn., in Manitoba were measured during 1965-1970. In representative commercially grown cultivars under field plot conditions, natural infections caused significant reductions in groat yield, ranging from 6.8% to 30% in most years.

Introduction

The FAO symposium on crop losses held in Rome in 1967 focused attention on the need for specific information on the economic importance of plant diseases (4). Data on losses due to specific diseases are important to plant breeders and pathologists if they are to make the most efficient use of limited resources. In Manitoba, losses caused by rust in oats have been monitored on a continuing basis since 1963. Annual rust loss experiments take into account the epidemiology of the pathogen in relation to the stage of development of the host. They also provide current data on the prevalent varieties and permit a data-based assessment of losses in commercial production.

Loss investigations prior to 1963 have been reviewed (2). In 1963 and 1964 groat yield was reduced by 15% on the average when the commonly grown cultivar Garry was subjected to natural epidemics of crown rust in the Red River Valley (2). At the flowering stage in late-seeded oats, infection levels of naturally occurring crown rust rated 30% reduced yield by 25%. Similar levels of infection at a later stage of development reduced yields by less than 10% (2). Yield data obtained in 1967 indicated that maneb, the rust protectant chemical used in these experiments, had no effect on yield in the absence of rust (5). In Ontario, Clark (1) obtained yield increases of about 20% by chemically protecting oats from natural crown rust infection. This paper presents data for 5 years on the effect of crown and stem rust of oats on the yield and quality of oats in Manitoba.

Materials and methods

A split-plot design, with maneb treatments for main plots and cultivars as subplot treatments, was used to assess the effect of stem rust and crown rust on yield and kernel characteristics. Six replicates were planted in 1965, 1966, and 1970 and 12 replicates in 1968 and 1969. Also two different seeding dates were used in 1965, 1966, and 1969. Five of 12 replicates in 1968 and in the late-seeded test of 1969 were discarded due to weather damage or virus infection. Each subplot consisted of 4 rows 5.6 m long with either 23- or 30-cm spacing between rows. Buffer plots were used to separate main plot treatments. In 1969, the plots were surrounded by solid-seeded oats to simulate field conditions.

The cultivars 'Victory' or 'Eagle' were used in each test; both are highly susceptible to crown rust and stem rust. 'Lodi' was used in the 1965 and 1966 tests to differentiate between crown and stem rust losses because it is susceptible to stem rust but was moderately resistant to crown rust in tests in 1965. Either 'Garry' or 'Kelsey' was used as a cultivar representative of commercial production although both are susceptible to crown rust and stem rust. Maneb (Dithane M-22, 80% maneb W.P., Rohm and Haas Co. of Canada, West Hill, Ont.) sprays were applied to half of the main plots weekly and after heavy rains, at the rate of 3 liters of 0.33% aqueous solution (w/v) per subplot beginning in mid-July. Yield data were obtained by harvesting all the plants in 5-m lengths of the two center rows of each subplot.

Results and discussion

Maneb gave excellent control of both rusts every year. In 1965 moderate to heavy infections of both crown rust and stem rust developed (Table 1), resulting in combined reductions in average groat yields for early

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Table 1. Severity of oat crown rust and stem rust, expressed as % area of flag leaf or culm affected, in untreated plots at various stages of plant development

Date	Oat cultivar	Early seeding			Late seeding		
		Plant stage	Crown rust %	Stem rust %	Plant stage	Crown rust %	Stem rust %
1965							
June 12		Seeded					
June 18					Seeded		
Aug. 3	Victory		2			tr	
Aug. 18	Garry		8			3	
	Lodi		1			tr	
	Victory		8			3	
Aug. 23	Garry		10	1		5	1
	Lodi		4	1		2	tr
	Victory		10	10		5	5
Aug. 30	Garry		30	2		15	2
	Lodi		5	2		3	1
	Victory		20	20		10	15
Sept. 8	Garry			5		40	10
	Lodi			8		10	15
Sept. 16	Victory	Harvested		40		40	40
Sept. 20	Garry	Harvested				50	10
	Lodi	Harvested				10	15
	Victory	Harvested				60	50
Sept. 21					Harvested		
1966							
May 31		Seeded					
June 9					Seeded		
Aug. 15	Eagle		1			1	
	Garry	Late milk	1		Early milk	1	
	Lodi		tr			tr	
Aug. 25	Eagle		50			20	
	Garry	Late dough	8		Late milk	3	
	Lodi		tr			1	
Aug. 31		Harvested					
Sept. 6	Eagle				Late dough	25	22
	Garry					10	5
	Lodi					1	4
Sept. 8					Harvested		
1968							
June 3					Seeded		
Aug. 2	Eagle					10	
	Kelsey				Early milk	1	
Aug. 28	Eagle					48	
	Kelsey					14	
Sept. 24					Harvested		
1969							
May 22		Seeded					
June 5					Seeded		
Aug. 15	Eagle	Early dough	12				
	Kelsey		5				
Aug. 25	Eagle					73	
	Kelsey					45	
Sept. 4		Harvested					
Sept. 12					Harvested		
1970							
June 23					Seeded		
Aug. 31	Eagle				Late milk	tr-3	tr
	Kelsey				Late milk	tr-3	tr
Sept. 10	Eagle				Dough	1	3
	Kelsey				Dough	1	3
Sept. 28	Eagle						38
	Kelsey						40

Table 2. Effect of natural infections† of crown rust and stem rust on groat yield in oats

Year and cultivar	Early seeding			Late seeding		
	Maneb-treated	Non-treated	% difference††	Maneb-treated	Non-treated	% difference
	Groats in kg/ha					
1965						
Garry	3627	3169	12.6*	3467	3130	9.7
Lodi	3316	3149	5.0*	3369	3269	3.0
Victory	3704	2956	20.2*	3157	2413	23.6**
1966						
Eagle	2915	2613	10.4*	1925	1501	22.0*
Garry	2205	2055	6.8*	1778	1599	10.0
Lodi	2477	2353	5.0*	1847	1839	0.3
1968						
Eagle				3403	2366	30.4**
Kelsey				3541	2616	26.1**
1969						
Eagle	3505	2603	25.7**	2822	1773	37.2**
Kelsey	3401	2493	26.7**	3585	2485	30.7**
1970						
Eagle				1961	1660	15.3*
Kelsey				2401	2220	7.4*
Mean all years	3144	2674	14.9	2771	2239	19.2

* and ** significant at the 5% and 1% levels, respectively.

† Stem rust did not develop in the 1966 early seeding or in the 1968 and 1969 plots; however, septoria leaf spot affected yield in nontreated plots in 1969. In 1970 the yield losses were caused primarily by stem rust.

†† Difference as % of treated.

Table 3. Effect of natural crown rust and stem rust infections on quality in oats expressed as thousand kernel weight and liter weight

Year and cultivar	Early seeding			Late seeding			Early seeding			Late seeding		
	Maneb-treated	Non-treated	% difference†	Maneb-treated	Non-treated	% difference	Maneb-treated	Non-treated	% difference	Maneb-treated	Non-treated	% difference
	Thousand kernel weight (g)						Liter weight (g)					
1965												
Garry	32.3	30.6	5.3**	31.4	29.6	5.7**	499	480	3.9**	480	454	5.4
Lodi	34.5	33.0	4.3**	33.4	31.0	7.2**	493	485	1.6**	479	461	3.8
Victory	32.3	29.4	8.8**	30.9	27.3	11.7**	513	472	8.0**	470	409	12.9**
1966												
Eagle	28.1	28.6	-1.7	27.8	26.0	6.5	502	483	3.8	463	455	1.4
Garry	29.5	29.3	0.7	29.5	30.6	-3.6	483	485	0.5	467	476	1.9
Lodi	33.0	33.1	-0.1	35.7	36.4	-1.9	485	480	1.1	454	461	1.4
1968												
Eagle				29.6	25.2	14.8**				454	428	5.9**
Kelsey				27.7	24.7	10.8**				454	427	6.2**
1969												
Eagle	27.0	24.5	9.2**	30.0	25.4	15.3*	404	381	5.7*	386	325	15.3**
Kelsey	25.6	23.6	7.8**	32.1	29.2	9.0*	423	394	7.0*	411	376	9.0**
1970												
Eagle				27.0	24.5	9.3*				396	377	4.9*
Kelsey				29.1	28.7	1.3				454	441	2.8*
Mean all years	30.3	29.0	4.3	30.3	28.2	6.9	475	457	3.7	447	424	5.1

* and ** significant at the 5% and 1% levels, respectively.

† Difference as % of treated.

and late seeded crops of 21.9%, 11.2%, and 4.0% for 'Victory', 'Garry' and 'Lodi', respectively (Table 2). Thousand kernel weights and liter weights were also reduced in most cases (Table 3). The loss in 'Garry' was 6% greater than that in 'Lodi', suggesting that about half the yield reduction was caused by crown rust.

In 1966 both rusts developed to significant levels only on 'Eagle', and in this cultivar significant reductions in yield occurred at both dates of seeding. The significant reduction in yield in early-seeded 'Garry' and 'Lodi' is surprising because of the light infections.

In 1968 Kelsey and Eagle were seeded on only one date, and crown rust caused an average reduction in yield of 28%. Thousand kernel weights and liter weights were reduced by an average of 12.8% and 6%, respectively.

Crown rust developed again in 1969 and overall average reductions in yield were approximately 30%, with significant adverse effects on both quality measurements. In that year a leaf spot, caused primarily by Septoria sp., developed on the plants, and unsprayed plots nearing maturity had infections that covered an average of 5.4% and 12% of the flag leaf area for the early seeded and late seeded plots, respectively. Since maneb also controlled this disease, not all of the yield loss in untreated plots is attributable to crown rust.

In 1970 very little crown rust developed, and the yield losses of 15.3% and 7.4% for 'Eagle' and 'Kelsey', respectively, must be attributed to the late infection of stem rust. These results confirm previous observations that stem rust can cause serious losses even if severe infections develop only after the milk stage (3).

These data clearly indicate that either or both of the oat rusts causes significant yield and quality reductions in oats in Manitoba in most years.

Acknowledgment

The statistical analyses were performed by R.J. Baker.

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COOPERATIVE SEED TREATMENT TRIALS - 1972[1]

J.T. Mills[2]

Abstract

Twenty-six seed treatment chemicals were tested for their efficacy in controlling bunt of wheat (Tilletia foetida), covered smut of oats (Ustilago kolleri), and covered smut of barley (U. hordei) and for their effects on the emergence of wheat, barley, oats, rye, flax, rape, and corn. The results show that UNI 2002 and BASF 3302 controlled the three smuts without reducing emergence. None of the treatments gave significantly increased emergence in all of the seven crops, but Panogen 15B, Manzate D, Polyram liquid, RHC 287, and BASF 3302 gave significant increases in four or five of them.

Introduction

In 1972, 26 seed treatment chemicals were tested for their efficacy in controlling common bunt of wheat [Tilletia foetida (Wallr.) Liro], covered smut of oats (Ustilago kolleri Wille), and covered smut of barley [U. hordei (Pers.) Lagerh.] and for their effects on the emergence of wheat, oats, barley, flax, rye, rape, and corn under Manitoba conditions.

Materials and methods

The tests were designed to test the efficacy of the chemicals upon the smuts and the effects on emergence of wheat, oats, barley, flax, rye, rape, and corn. Table 1 lists the chemical composition where available, the product name, and the source of the materials used. Panogen 15B was included as a standard.

Seed of 'Red Bobs' wheat (Triticum aestivum L.), 'Random' oats (Avena sativa L.), and 'Herta' barley (Hordeum distichon L.) were used in the smut and emergence tests. 'Noralta' flax (Linum usitatissimum L.), 'Cougar' rye (Secale cereale L.), 'Target' rape (Brassica napus L.) and cultivar CM7 field corn (Zea mays L.) were used for emergence tests. Many cracks were evident at the edges of the flax seed and also above the embryos of the rye seed.

One gram of the appropriate smut spores was added to each 200g of wheat, oats, and barley seed before treatment to ensure heavy infection. Each of the chemicals was applied to 200-g seed samples of the seven crops, at the rates of product suggested by the manufacturer, by hand-shaking the seed in a glass jar until the seed was uniformly covered. As recommended by the manufacturer, corn treated with DL plus was pretreated with a slurry of Captan 75W (1.00 oz in 1 pint water per bushel). Seed was removed from the jars after not more than 3 days, and lots of 200 seeds were packaged in paper envelopes. Envelopes that contained seed from the same treatment were stored in polyethylene bags at 15 C for not more than 4 weeks before seeding.

Tests were carried out at Brandon and at Morden, Manitoba. There were four replicates per location. Each plot replicate consisted of 200 seeds (100 for corn) planted in a row 12 ft long; all rows were planted 9 inches apart, and plots were arranged in a randomized block design. Emergence of all crops was recorded 3-4 weeks after seeding.

The percentage of smutty heads, based on counts of 500 heads per row, was recorded after the crop had headed. The results are given as means of eight replicates, four from each planting site. The "LSD-05" is based on an analysis of the means of the treatments for each station.

Results and discussion

Smut infection of untreated seed was 4% for barley and varied from 11% to 17% for wheat, and from 10% to 12% for oats. Some chemicals gave complete control of all smut diseases on wheat, oats, and barley; others controlled oat and barley smut but failed to control bunt, or they controlled bunt and barley smut but failed to control oat smut (Table 2). Treatments giving good control of

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Table 1. Seed treatment materials used in the cooperative tests

Treatment no.	Source*	Product name	Chemical name
1		Untreated check	
2	BASF	BASF 3270	2,5-dimethyl-3-furylamide (50%)
3	BASF	BASF 3302	BASF 3270 (50%) + maneb (32%)
4	Chipman	TF 3124	identity not available
5	Ciba-Geigy	NF 44	1,2-bis(3-methoxycarbonyl-2-thioureido) benzene (70%)
6	Interprovincial	Busan 30IP	2-(thiocyanomethylthio) benzothiazole (30%)
7	Du Pont	Benlate T	benomyl [methyl 1-(butylcarbamoyl)-2-benzimidazole carbamate] (30%) + thiram (30%)
8	Du Pont	Manzate D	maneb (80%)
9	Hoechst	Hoe 6053	2-methyl-5,6 dihydro-4-H-pyran-3-carboxylic anilide (75%)
10	Hoechst	Hoe 6053 + thiram	Hoe 6053 (75%) + thiram (75%)
11	Merck	Me 77	identity not available
12	Niagara	Polyram liquid	zinc activated polyethylene thiuram disulfide (22.5%)
13	Niagara	NIA 25050	identity not available
14	Nor-Am	Panogen 15B	methylmercuric dicyandiamide (3.7 oz/gal)
15	Nor-Am	SN 43410	identity not available
16	Nor-Am	SN 43493	identity not available
17	Rohm & Haas	RHC 287	identity not available
18	Rohm & Haas	RHC 288	identity not available
19	Uniroyal	UNI 2002	Vitavax + thiram
20	Uniroyal	UNI 2005	Vitavax + thiram
21	Uniroyal	UNI 2009	Vitaflo DB + lindane
22	Chipman	DL plus	diazinon 15%, lindane 25%, captan 15%
23	Chipman	B-3	diazinon 11%, lindane 16.6%, captan 33.5%
24	Chipman	Gammasan	lindane 75%, captan 10%
25	Ciba-Geigy	CGF 2590	NF 44 7%, captan 37%, lindane 10%
26	Ciba-Geigy	CGF 2610	NF 44 23%, diazinon 10%, lindane 16.7%
27	Ciba-Geigy	CGF 2620	NF 44 7%, lindane 37.7%
28		Untreated check	

* BASF Canada Ltd., Montréal, Québec; Chipman Chemicals Ltd., Hamilton, Ontario; Ciba-Geigy Canada Ltd., Montréal, Québec; Interprovincial Cooperatives Ltd., Winnipeg, Manitoba; E.I. Du Pont de Nemours & Co., Inc., Wilmington, Delaware; Hoechst Chemicals Canada Ltd., Montréal, Québec; Merck & Co., Inc., Rahway, New Jersey; Niagara Chemicals, Burlington, Ontario; Nor-Am Agricultural Products Inc., Woodstock, Illinois; Rohm & Haas Co. of Canada Ltd., West Hill, Ontario; Uniroyal Chemical Division, Elmira, Ontario.

the three smuts without reducing emergence were BASF 3302 (an unidentified wettable powder) and UNI 2002, a slurry containing vitavax and thiram.

Emergence of untreated cereal checks was: wheat, 69% and 77%; oats, 64% to 85%; and barley 92% (Table 2). Some seed treatments were phytotoxic: emergence of wheat, barley and oats was significantly reduced after treatment with BASF 3270, NF 44, Hoe 6053, Me 77 and NIA 25050. At Brandon, Hoe 6053 and, to a lesser extent, Hoe 6053 + thiram were associated with burnt foliage of oats and barley, probably due to the very high dosages used.

Emergence of untreated checks of the other crops was: flax, 36% to 62%; rye, 41% to 47%; rape 11% to 69%; corn, 52%. Rape flea beetle (Phyllotreta cruciferae Goeze) caused severe damage to emerging rape

Table 2. Effects of seed-treatment chemicals on smuts and emergence in wheat, oats, and barley

Treatment no.	Product name	Formulation*	Dosage (oz/bu)	Smutted heads (%)** Wheat BM	Oats BM	Barley BM	Emergence (%) Wheat BM	Oats B	Oats M	Barley BM
1	Untreated check			10.5	12.1	4.3	69.4	73.0	63.5	92.3
2	BASF 3270	WP	0.96		0.0			80.0	68.3	
			1.44			0.0				87.8
			1.80	0.0			69.1			
3	BASF 3302	WP	0.96		Tr			78.5	83.0	
			1.44			0.0				91.4
			1.80	0.0			79.4			
4	TF 3124	SL	0.50	0.6			77.4			
			1.00	0.0	1.8	0.0	78.8	77.3	63.8	87.3
			1.50		1.1	0.0		81.5	70.5	90.4
5	NF 44	WP	1.00	3.2	0.3	0.2	70.9	82.3	64.8	87.0
			1.50	1.9	0.2	0.6	70.3	75.3	58.5	88.4
6	Busan 30IP	SN	0.75	0.8	0.4	0.0	71.5	83.8	79.8	85.0
			1.00	0.3	0.0	0.1	71.9	78.3	77.0	85.8
7	Benlate T	WP	0.70	0.5			75.8			
			1.25		0.0	0.0		83.5	62.8	91.3
8	Manzate D	WP	0.70	0.1			85.3			
			1.25		0.1	0.2		81.8	82.5	93.2
9	Hoe 6053	D	4.00	0.0	0.0	0.0	64.9	70.0	66.5	90.3
10	Hoe 6053 + thiram	D	2.00 + 2.00	0.0	0.0	0.0	82.8	85.0	75.0	83.3
11	Me 77	SN	1.02		2.0			80.3	68.5	
			1.54			1.8				88.3
			1.92	0.7			70.0			
			2.04		0.3			80.3	66.8	
			3.08			0.6				90.1
			3.84	0.0			65.1			
12	Polyram liquid	SL	2.00	0.0	4.1	0.1	77.4	89.8	77.0	93.9
13	NIA 25050	WP	2.00	5.4	--***	0.2	49.9	92.8	62.0	81.4
14	Panogen 15B	SN	0.75	0.1	0.3	0.0	84.5	80.5	84.8	89.6
15	SN 43410	WP	2.00	0.3	0.0	0.0	77.5	81.5	76.0	90.7
16	SN 43493	WP	2.00	0.4	0.6	0.1	82.8	81.5	78.5	90.5
17	RHC 287	SL	1.00	0.1		0.0	82.6			89.5
			1.50		1.8			82.8	77.0	
18	RHC 288	SL	0.75	0.3		0.1	82.0			88.1
			1.00		5.9			89.0	75.8	
19	UNI 2002	SL	1.50	0.1	Tr	0.0	81.8	79.5	72.5	91.4
20	UNI 2005	SL	1.50	0.2	0.3	0.0	76.6	78.8	71.0	90.3
28	Untreated check			16.9	9.9	3.6	77.3	85.0	72.5	92.0
LSD .05				6.4	2.0	1.5	2.9	NS	1.4	1.1

* Formulation code: D = dust, SN = solution, SL = slurry, WP = wettable powder.

** See text.

*** Morden had complete smut control, Brandon not sown.

B = Brandon, M = Morden, BM = Brandon and Morden combined.

NS = not significant.

Tr = Trace.

Table 3. Effects of seed-treatment chemicals on emergence in flax, rye, rape, and corn

Treatment no.	Product name	Formulation*	Flax Dosage (oz/bu)	Flax Emergence (%) B	Flax Emergence (%) M	Rye Dosage (oz/bu)	Rye Emergence (%) BM	Rape Dosage (oz/bu)	Rape Emergence (%) B	Rape Emergence (%) M	Corn Dosage (oz/bu)	Corn Emergence (%) BM
1	Untreated check			61.8	40.0		46.8		69.3	11.0		51.5
2	BASF 3270	WP	1.68	55.0	36.8	1.68	36.8	1.50	72.3	8.0	1.68	42.8
3	BASF 3302	WP	1.68	64.8	64.8	1.68	55.3	1.50	69.3	34.3	1.68	66.0
4	TF 3124	SL†	4.00	53.8	52.5	0.50	47.4					
						1.00	51.6					
5	NF 44	WP	2.00	52.0	39.8							
6	Busan 30IP	SN	0.75	55.0	43.8	0.50	27.5	1.50	73.3	16.8	1.12	75.8
			1.00	56.5	55.8	0.75	30.5				1.40	71.8
7	Benlate T	WP	2.50	55.8	70.3	0.70	48.9	2.50	78.0	49.8	2.00	36.8
8	Manzate D	WP	2.50	54.3	32.0	0.70	59.1	2.50	71.8	33.3	2.00	54.1
9	Hoe 6053	D	4.00	61.3	61.0	4.00	39.9	4.00	63.8	50.8		
10	Hoe 6053 +		2.00 +			2.00 +		2.00 +				
	thiram	D	2.00	50.8	37.0	2.00	64.1	2.00	62.0	13.0		
12	Polyram liquid	SL				2.00	54.9	3.00	65.3	48.5	2.00	58.5
14	Panogen 15B	SN	1.50	61.8	63.0	0.75	72.0	1.00	67.0	32.8	1.50	70.6
15	SN 43410	WP	1.12	55.0	40.5	2.00	46.9	1.00	78.3	37.8	1.12	75.0
16	SN 43493	WP	1.12	52.3	40.5	2.00	39.5	1.00	81.3	10.5	1.12	61.1
17	RHC 287	SL	3.00	53.3	50.3	1.00	60.5	1.00	70.5	15.0	1.00	78.4
18	RHC 288	SL	2.00	58.0	55.0	0.75	57.9	0.75	71.3	35.3	0.75	48.9
19	UNI 2002	SL	4.00	51.5	59.5	1.50	50.3				1.50	48.6
20	UNI 2005	SL	4.00	54.3	62.0	1.50	55.4				1.50	70.6
21	UNI 2009	D						24.0	75.5	29.8		
22	DL plus +										2.00 +	
	captan**	D									1.00	66.3
23	B-3	D									3.00	73.3
24	Gammasan	D						8.0	72.8	44.5		
25	CGF 2590	D						75.0	62.3	6.0		
26	CGF 2610	D									3.00	73.1
27	CGF 2620	D						75.0	69.3	39.8		
28	Untreated check			44.5	36.0		41.1					51.9
LSD .05				2.3	2.2		3.9		NS	2.1		2.6

* Formulation code: D = dust, SN = solution, SL = slurry, WP = wettable powder.

† 50:50 Mixture of ethylene glycol and water.

** Captan applied as a pretreatment.

B = Brandon, M = Morden, BM = Brandon and Morden combined.

NS = not significant.

seedlings at Morden. None of the treatments gave significantly increased emergence of flax, rape, corn, and rye, but Polyram liquid and Panogen 15B gave significantly increased emergence in three of them. A significant decrease in emergence in three crops was evident with BASF 3270 (Table 3).

Acknowledgments

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INTERACTIONS AMONG BIOTIC VARIABLES AFFECTING COCHLIOBOLUS SATIVUS AS A PATHOGEN OF CEREALS[1]

J.T.Mills

Abstract

Present knowledge of seedling blight and common root rot diseases of cereals caused by Cochliobolus sativus is reviewed with particular reference to etiology and control. Interactions known to occur between C. sativus and other fungi are described and possible interactions between the pathogen and faunal and viral variables discussed. It is suggested that all variables that may affect disease incidence first be examined and then narrowed down to specific modifying factors. The use of selective chemicals may assist in determining how biotic variables modify common root rot.

Introduction

Cochliobolus sativus (Ito & Kurib.) Drechsl. ex Dastur [conidial state, Helminthosporium sativum Pamm. King & Bakke, syn. Bipolaris sorokiniana (Sacc. in Sorok) Shoem.] is the main causal organism of four important diseases of cereals: black point (kernel smudge), seedling blight, leaf blotch, and common root rot. It is the least specialized of the virulent and prevalent Helminthosporium species present on graminaceous hosts (11). Fusarium culmorum (W. G. Sm.) Sacc. and other Fusarium spp. (20) are often associated with C. sativus in common root rot. Black point cannot be controlled; seedling blight can be partially controlled with seed treatment fungicides; but attempts to control common root rot, studied for 40 years in Canada, have been less successful. Studies on common root rot are extremely difficult because of the existence in soil of numerous complex interrelationships among biotic and abiotic variables. This report summarizes present knowledge of the diseases, with emphasis on biotic and abiotic interactions, and proposes possible areas for future research.

THE MICROFLORA OF SEEDS AS RELATED TO THE SEED-BORNE PATHOGEN

C. sativus, with Alternaria sp. and other "field fungi", is a common component of the air-spora which invade developing heads of cereal plants (22, 25). The invasion is mainly dependent on the weather during the time the kernels are developing and maturing (13). In the swath, other fungi, including Trichothecium sp. and Streptomyces spp. ("harvest fungi") (31) and Penicillium spp. ("storage fungi") (8) also may infest the seeds. The bacteria, yeasts, fungi, nematodes, and viruses known to occur in barley have been listed by Pepper & Kiesling (33). Many fungi could directly or indirectly affect C. sativus on the grain (Fig. 1). Interrelations occurring among C. sativus and other fungi, insects, mites, and environmental variables in stored grain have been investigated by Sinha et al. (39).

Machacek et al. (24) found that seed samples heavily infested with C. sativus originated mainly in the Maritime Provinces, Quebec and Manitoba. C. sativus usually disappears from seed of wheat and barley within 3 years, but may survive in heavily infested samples as long as 9 years (23) as mycelium in the pericarp (36).

THE INFESTED SEED IN SOIL

Barley seed infested with C. sativus has dormant mycelium in the lemma, palea, pericarp, and lodicules and ungerminated spores between the lemma, palea, and pericarp (25). Because seed-borne C. sativus invades the plumule and radicle while they are still under the hull, in heavily infested seed the pathogen is soon well established. If such seed is sown, seedling blight may develop, with brown streaks on the coleoptiles and leaf sheaths, and may cause the death of seedlings. Such infested seeds have an attendant microflora the components of which are dependent on the age of the seed and its history, i.e. the growing and storage environments. The fungi isolated from seed freshly sown in soil are seed fungi. Both Christensen (9) and Mead (26) found that the soil microflora does not have a marked effect on seedling blight arising from naturally infected barley seed. The fungi isolated from freshly produced roots of wheat and barley in normal soil are soil fungi, e.g.

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[1] Contribution No. 544, Research Station, Canada Department of Agriculture, Winnipeg, Manitoba R3T 2M9.

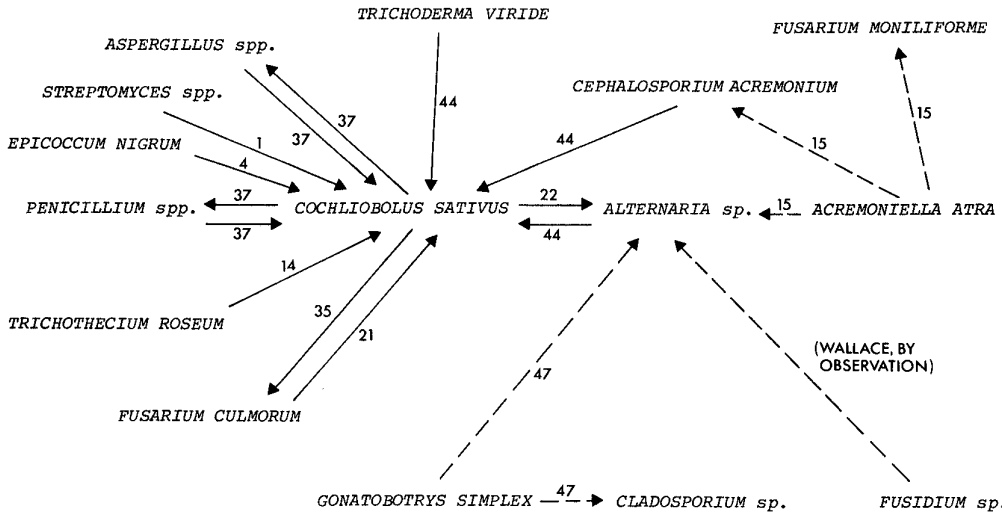

Figure 1. Possible interactions among *Cochliobolus sativus* and other fungi on cereal seed and in soil; numbers refer to Literature cited; ——→ = inhibition, e.g. *Streptomyces* spp. are known to inhibit C. *sativus*; – – –→ = dependence, e.g. *Gonatobotrys* is dependent on *Alternaria* sp. for growth.

Pythium, Fusarium, Cylindrocarpon species, and are not seed fungi (34). However, it appears that seed-borne fungi can reduce the coleoptile streaking caused by C. sativus (10). When surface-sterilized wheat seeds were inoculated serially with C. sativus, Epicoccum nigrum Lk. and Alternaria tenuissima (Fr.) Wilt., streaking caused by C. sativus was reduced by the antagonistic action of E. nigrum on the pathogen and by stimulation of the host by A. tenuissima. Csuti et al. (10) also found that the fungus which is applied first to the host dominates fungi inoculated later, which remain secondary.

Volatile fungicides, e.g. Panogen PX, have been tested (45) as seed treatments but they have not given complete control of the pathogen probably because of their inability to affect deep-seated infections. A more promising approach is the use of selective systemic fungicides that can kill the pathogen without altering possible beneficial effects to the seedling from Alternaria spp. and other fungi.

SOIL-BORNE C. SATIVUS & F. CULMORUM AND COMMON ROOT ROT

Fusarium culmorum is commonly associated with C. sativus in common root rot in Canada.

In certain areas, for example northwest Alberta, F. culmorum is the dominant pathogen. This fungus, which has the dual role of crop pathogen and post-harvest colonizer of cereal stubble, is prevalent in the top 6 inches of the soil and has pronounced competitive saprophytic ability (3).

C. sativus occurs widely in soil or on stubble as overwintering conidia which are kept dormant by the action of soil fungistasis (40). Conidia germinate under the influence of exudates from the crown, the subcrown internode, and possibly the crown roots (S.H.F. Chinn, personal communication). Chinn has found that conidia more than 1/8 inch away from the crown will not germinate. On the Canadian prairies the subcrown internodes usually become infected in May or early June; infection subsequently extends to the crown where sporulation occurs by late July or early August and conidia drop to the soil and are later mixed by cultivation (S.H.F. Chinn, personal communication). Plants affected by root rot occur at random throughout the crop. In the post-heading phase of plant growth, premature bleaching of the ears and straw can occur. At Saskatoon Chinn (7) has studied invasion of wheat seedlings by fungi other than C. sativus. Possibly symptomless invasion of healthy plants, e.g. by Penicillium spp., interferes with subsequent invasion by C. sativus.

Control has been attempted with seed fungicides, by fungicidal pellets placed near the subcrown internode (29), with various soil amendments, and by certain cultural practices, e.g. incorporation of straw into soil. These attempts have met with little or no success.

Butler (3) has presented information on microfloral antagonism which has explained disease anomalies but this has achieved little in terms of disease control. He lists 846 references on root rots of which three are concerned with interrelations between root rot and insects, and one with nematodes. It is as if there has been an unwritten

agreement to investigate root rots solely in terms of fungi. Insects, mites, nematodes, and other microfaunal components may affect indirectly or directly root rot incidence. These components, most abundant in the top 6 inches of the soil, are mostly microfloral grazers and some are known to feed and breed on Alternaria and other fungi (30).

VIRUSES AND COMMON ROOT ROT

Conceivably, root invasion by C. sativus and F. culmorum and the random distribution of common root rot across a field might be related to a virus infection, with or without evident symptoms, that predisposes plants to root rot infection. Some examples of plant viruses which may affect the incidence of common root rot are as follows:

Soil-borne viruses -- Wheat soil-borne mosaic virus (WMV) and oat mosaic virus (OMV) are known to have a serious effect on yields of cereals in the USA (19) but have not been reported in Canada. In Ontario, wheat spindle streak mosaic virus (WSSMV) is known to cause significant losses in yield of winter wheat (41). Similar agents may be involved in the transmission of the three viruses. It has been suggested that the root-inhabiting fungus Polymyxa graminis Ledingham could be the vector of WMV and WSSMV (41). Once it was thought that Helminthosporium spp. were associated with transmission of the rosette form of WMV (27).

Seed-borne viruses -- Chiko (5, 6) found seed-borne barley stripe mosaic virus (BSMV) in 22% and 34% of the 2-row barley fields surveyed in Manitoba in 1970 and 1971, respectively. The incidence of diseased plants in these fields generally varied from a trace to 5% but in certain fields up to 50% of the plants were affected. Apparently no determinations have been made to check whether BSMV is a predisposing factor in common root rot.

Aphid-transmitted viruses -- Smith (42) suggested that aphid-transmitted barley yellow dwarf virus (BYDV), widespread on the Canadian prairies, could be a predisposing factor in common root rot. This hypothesis was based on New Zealand data indicating increased pathogenicity of Fusarium and Rhizoctonia root rotting fungi on wheat plants previously infected with BYDV. Scott (38) studied the interactions between BYDV and C. sativus in oats and durum wheat in Illinois in laboratory and field experiments. He found that root rot symptoms were more severe under field than under laboratory conditions. In BYDV-infected plants root rot symptoms were detectable earlier and were more severe than in virus-free plants. Similarly more fungi were isolated from BYDV-infected plants.

Leafhopper-transmitted viruses and mycoplasmas -- Aster yellows causal agent (AYCA) and oat blue dwarf virus (OBDV) occur on wheat, oats, and barley in Canada and can result in significant yield losses. The aster leafhopper (Macrosteles fascifrons Stål) is the chief vector of AYCA and the only known vector of OBDV. No determinations have been made to check whether AYCA or OBDV are predisposing factors to common root rot.

Mite-transmitted viruses -- Wheat streak mosaic virus (WSMV) is transmitted by the wheat curl mite (Aceria tulipae Keifer) and is found only in the winter wheat growing areas of Ontario, Alberta, and Saskatchewan.

In summary, there is considerable evidence for increased susceptibility of virus-infected plants to root rot (12, 32, 43, 46). Some of the virus and mycoplasma diseases occurring on cereals in Canada are of widespread occurrence in wheat, barley and oats (OBDV, BYDV, AYCA), others occur in restricted areas or on particular cereals (WSMV, WSSMV, BSMV). No determinations have been made to check whether any of these diseases are predisposing factors in common root rot of cereals in Canada.

SOIL MICROFAUNAL COMPONENTS AND COMMON ROOT ROT

Evidence for the involvement of microfauna in the common root rot disease syndrome has been presented by Hanson et al. (18) who found that the bluegrass billbug, Calendra parvula (Sphenophorus parvulus Gyllendal), was widely distributed in eastern North Dakota, Nebraska, Minnesota and other north-central States of the United States. This weevil attacks the lower internodes of stems and sometimes the roots of spring wheat, barley, and grasses, weakening the plants and making them more vulnerable to attack by root rot organisms. Also, it provides avenues of entrance for these pathogens, and both adults and larvae of the insect carry fungi and bacteria in and on their bodies. So far there have been no reports of involvement of this insect with common root rot on the Canadian prairies.

Burrage and Tinline (2) at Saskatoon treated wheat seed with gamma BHC, aldrin, and heptachlor and found in field tests that root rot frequently was greater in plants from untreated seed than in those from seed treated with most of the insecticides tested, possibly because of damage to the untreated plants by wireworms. However, wireworms, while locally abundant, are not a problem in most fields on the Canadian prairies (S.H.F. Chinn, personal communication).

Some microfaunal types are found in most wheat fields, for example, Collembola, mites, and nematodes. They are mobile, have a discontinuous distribution in soil, and can increase quickly to large numbers if a suitable food source is present (28). Interactions that could occur between microfloral and microfaunal components in a

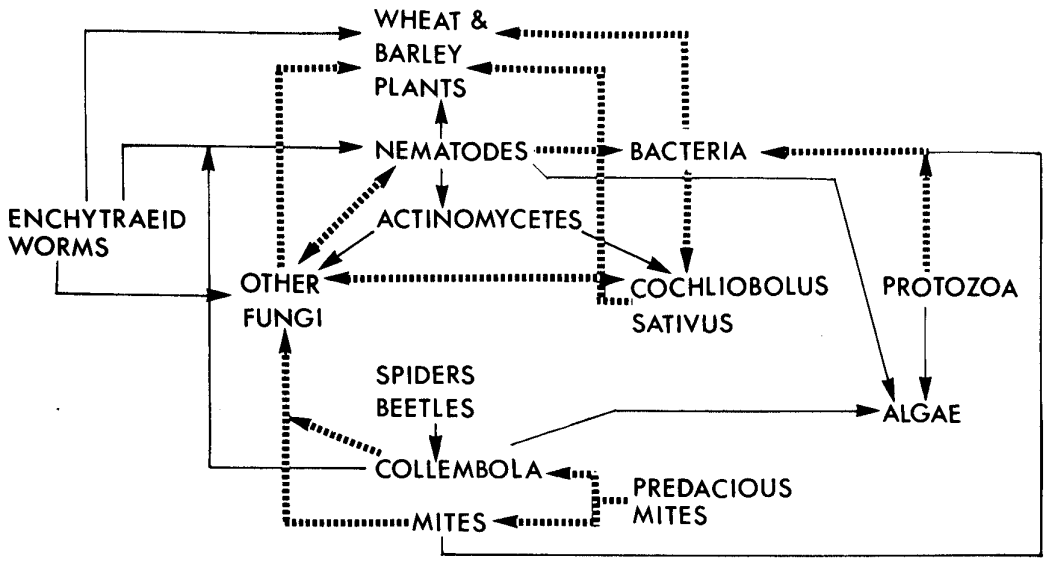

Figure 2. Possible interactions among *Cochliobolus sativus* and other biota. Solid lines indicate inhibition, broken lines indicate dependence.

soil infested with C. sativus are depicted in Fig. 2. Possibly C. sativus is affected indirectly; for example microfaunal components may eat fungi or bacteria antagonistic to C. sativus. Also, some nematodes eat bacteria and thus could interfere with bacterial lysis of C. sativus spores. In addition, some nematodes penetrate roots and thus allow fungi antagonistic to C. sativus to enter, excluding the pathogen by physical occupation of space. A further consideration is the dispersal of spores by mites and Collembola. Collembola feed on fungi and bacteria including components antagonistic to the pathogen. They migrate vertically downwards under adverse weather conditions, e.g. drought, and flourish in moist soil (17). In years of high rainfall there is a lower incidence of common root rot and this may be related in some way to the soil microfaunal components remaining near the surface.

CONTROL OF COMMON ROOT ROT OF CEREALS WITHIN THE TOTAL SOIL ECOSYSTEM

The total soil ecosystem and the abiotic and biotic factors operating within it, are depicted in Fig. 3. The phases in the life cycle of C. sativus related to the

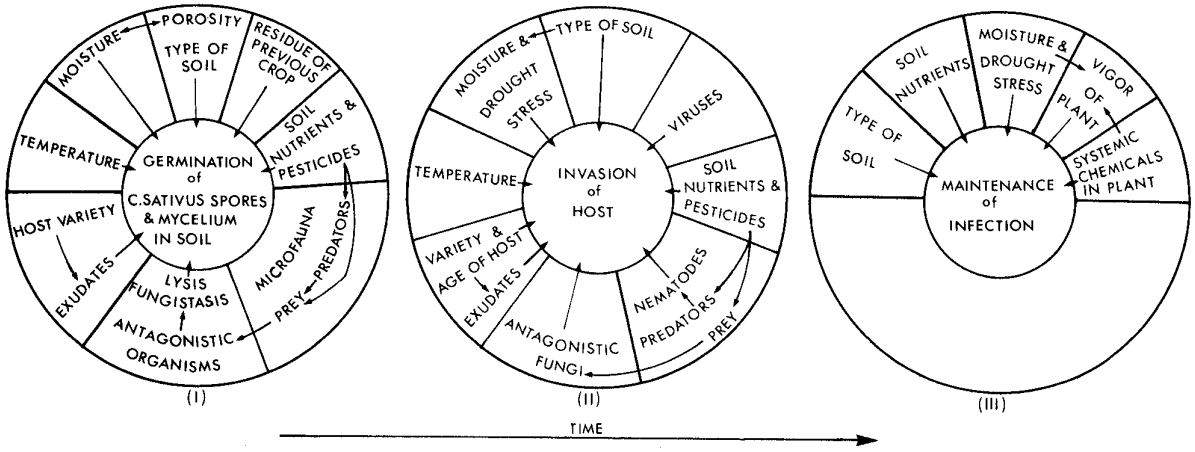

Figure 3. Most important variables affecting *Cochliobolus sativus* in soil and development of root rot in cereal plants with time.

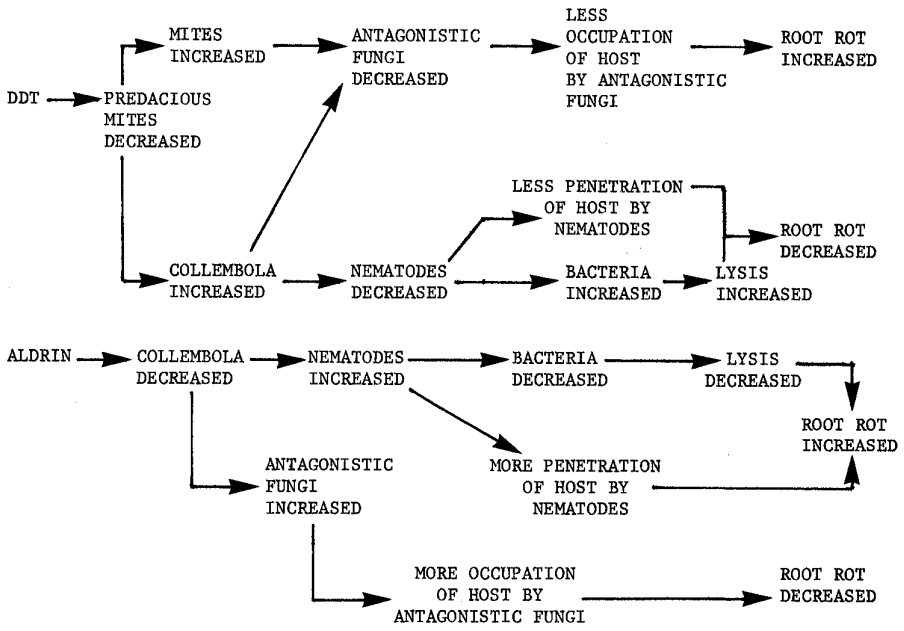

Figure 4. Theoretical pathways showing how the insecticides DDT and aldrin could affect incidence of root rot of cereals.

development of common root rot in soil are probably governed by only a few of these factors which vary with time. Control of common root rot can best be achieved at the stage of invasion of the host (Fig. 3 ii). We need to concentrate on the modifying factors and interactions among them, so that any recommended management practices will not indirectly favor the fungus. Fig. 4 shows two examples of how pesticide management practices may affect root rot via several possible pathways. Selective and wide-spectrum pesticides have already been used (16) for sorting out causal agents of soil-borne plant diseases. Each part of each interaction in the pathways could be monitored in the laboratory, e.g. changes in microfaunal populations. Similarly increases in Streptomyces when plants are sprayed with 2, 4-D, could be checked out using media selective for Streptomyces. Once validated in the laboratory, interactions may be studied in barley or wheat fields with a known history of severe root rot. Replicated strips of barley or wheat could be treated with pre-emergence or post-emergence herbicides, insecticides, acaricides, and fungicides. Microfloral and microfaunal populations and virus occurrence should be monitored at intervals, and root rot at maturity. In this way, involvement of microfauna and viruses in root rots may be determined. This information may lead to a practical means of control. Once the disease is controlled successfully, treatments can be modified where necessary to nullify possible harmful ecological effects. If microfauna and viruses are not found to be involved then we can look at the other (Fig. 3 ii) modifying variables.

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THREE NEW HOST RECORDS FOR DWARF MISTLETOES IN BRITISH COLUMBIA

R.B.Smith, J.A. Baranyay, and E.V.Morris[1]

While the signs and symptoms of dwarf mistletoe are fairly conspicuous, some host-parasite combinations occur so infrequently in British Columbia that new ones are still being discovered. Three new combinations are discussed in this note.

1. LARCH DWARF MISTLETOE (Arceuthobium laricis [Piper] St. John) ON PONDEROSA PINE (Pinus ponderosa Laws.)

Hawksworth and Wiens (2) reported that this combination was first observed in 1911 in Montana. They examined four other collections and rated ponderosa pine as an occasional host for larch mistletoe, i.e. 5% to 50% of trees infected when within 20 ft of heavily infected western larch (Larix occidentalis Nutt.), the primary host. In Canada, the first collection was made in 1970, 12 miles west of Creston, alongside Hwy. 3 (Fig. 1,A). This was a single main-stem infection on a 4 ft ponderosa pine located under an overstory of severely mistletoe-infected western larch. No other ponderosa pines were in the immediate area. Two more collections of this combination were made in 1972, one 8 miles south of Creston along Dodge Creek Trail (Fig. 1,B), the other at the junction of Hwys. 3A and 6 near South Slocan (Fig. 1,C). Although stands in both locations were dominated by infected western larch, they also contained numerous ponderosa pines. At the Dodge Creek site, only 2 infections were noted on one ponderosa pine, whereas 8 to 10 dead infections and several live ones were present on the infected ponderosa pine at South Slocan. As the neighboring uninfected ponderosa pine trees at South Slocan appeared equally exposed to larch dwarf mistletoe seed, the degree of susceptibility of ponderosa pine to larch dwarf mistletoe probably varies greatly among individual trees. All infections examined had caused well-defined, localized swellings with no branch proliferation or production of witches' brooms.

Ponderosa pine is also attacked by lodgepole pine dwarf mistletoe (Arceuthobium americanum Nutt. ex Engelm.) in British Columbia (3).

2. LARCH DWARF MISTLETOE ON GRAND FIR (Abies grandis [Dougl.] Lindl.)

Hawksworth and Wiens (2) list a collection representing this combination made in 1915 in Oregon and four others from western United States. In Canada, the first collection was made in 1971 at the junction of Hwys. 3A and 6 near South Slocan (Fig. 1,D). The stand contained mistletoe-infected larch overstory with grand fir in the understory. A second collection was made in 1972 on Dodge Creek Trail about 7 miles south of Creston (Fig. 1,E). Though lacking aerial shoots, this one consisted of a well-defined swelling with numerous basal cups, indicating the presence of dwarf mistletoe. The parasite was assumed to be larch dwarf mistletoe because it was abundant on the surrounding western larch trees and was the only one observed in the area. Some branch proliferation but no actual development of witches' brooms was noted on the infected grand fir branches. The combination is listed as rare in the United States (less than 5% of trees infected within 20 ft of heavily infected western larch) (2), and our surveys confirm this same status in Canada.

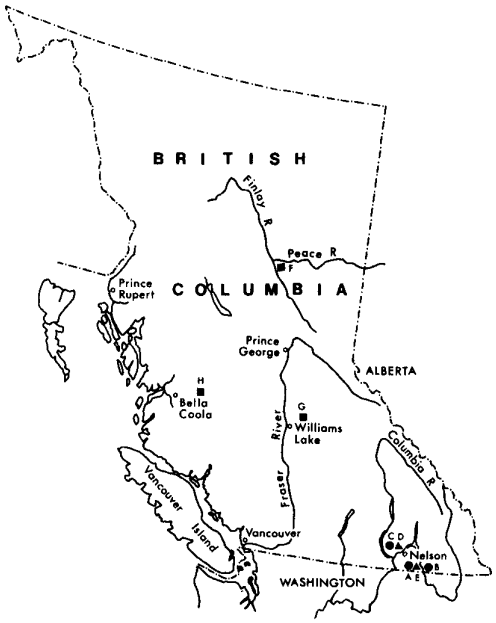

Figure 1. Locations of new host records for dwarf mistletoe in British Columbia; ● = larch dwarf mistletoe on ponderosa pine; ▲ = larch dwarf mistletoe on grand fir; ■ = lodgepole pine dwarf mistletoe on white spruce.

Grand fir is also occasionally attacked by Douglas-fir dwarf mistletoe (Arceuthobium douglasii Engelm.), in British Columbia (4).

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3. LODGEPOLE PINE DWARF MISTLETOE (Arceuthobium americanum) ON WHITE SPRUCE (Picea glauca [Moench] Voss)

This combination is commonly found in Alberta where white spruce occurs under heavily mistletoe-infected lodgepole pine (1). The infections are systemic, the witches' brooms small and compact, and the aerial shoots are generally sparse. In British Columbia, this combination appears less frequently. In 1970, the first collection was made at mile 65 on the Parsnip Forestry Road (35 miles north of MacKenzie) (Fig. 1,F). The infected tree had numerous brooms but no localized branch swellings. Only one of the brooms had aerial shoots. As is often the case in Alberta, surrounding white spruce trees were not infected. A second collection was made 13 miles south of Horsefly along Moffat Creek in 1972 (Fig. 1,G). The infected tree was severely broomed and aerial shoots were abundant. Three nearby white spruce were uninfected. A third collection was made about 13 miles east of Anahim Lake (Fig. 1,H). Two infected white spruce, both with several witches' brooms, were found within the understory of a lodgepole pine stand severely infected with dwarf mistletoe. About 30 other spruce examined in the same stand were uninfected. Thus, while the combination is rare in terms of number of trees infected, individual white spruce may be severely damaged.

In British Columbia, lodgepole pine dwarf mistletoe is the only mistletoe recorded in nature on white spruce.

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ADDITIONAL COLLECTIONS OF TUBERCULINA MAXIMA ON PINE STEM RUSTS IN WESTERN CANADA

J.M.Powell[1]

Abstract

The purple mold Tuberculina maxima Rost. is recorded for the first time in Saskatchewan on a pine stem rust (Cronartium comptoniae Arth.), constituting a range extension eastward of 9 degrees of longitude. The mold was also recorded on the western gall rust, Endocronartium harknessii (J. P. Moore) Y. Hiratsuka, for the first time in Alberta. A further collection in Alberta on the stalactiform blister rust, Cronartium coleosporioides Arth., considerably extended the range on this host.

A compilation of the collections of the purple mold Tuberculina maxima Rost. occurring on pine stem rusts in western Canada was recently published (1). During 1972, collections contributing important range extensions were made on three host species and these are reported to update our knowledge of the distribution of this fungus which has potential as a biological control agent (2).

Cronartium comptoniae Arth., sweetfern blister rust.

A collection of T. maxima on this rust was obtained from Twin Lake, about 30 miles northeast of La Ronge, Saskatchewan, on jack pine, Pinus banksiana Lamb. =P. divaricata (Ait.) Dumont, on July 18, 1972 (CFB 20334). This is the first record of T. maxima on a pine stem rust in Saskatchewan, and therefore extends the known range of T. maxima in Canada about nine degrees of longitude eastward.

Endocronartium harknessii (J. P. Moore) Y. Hiratsuka, western gall rust.

A collection of T. maxima on this rust was made in a private garden in Edmonton on a lodgepole pine, Pinus contorta Dougl. var. latifolia Engelm., on June 1st, 1972 (CFB 20335). The tree was about 10 years old and the gall six years old. This tree, however, had been obtained from a local nursery, and it was ascertained that the infected tree had come that spring from a tree farm near Mackay, Alberta. It was obvious that the gall was infected by T. maxima prior to the time of shipping, since 55% of the actively sporulating 2 1/2-inch-diameter gall was covered by purple spores of T. maxima. Visits to the tree farm showed much of the lodgepole pine stock to be heavily infected by E. harknessii and steps are now being taken to eliminate the stock. Evidence of T. maxima was found on a few E. harknessii galls when a small portion of the lodgepole pine stock was examined at the tree farm in mid-October. This is the first report of T. maxima on galls of E. harknessii in Alberta, and only the second in Canada (1).

Cronartium coleosporioides Arth., stalactiform blister rust.

During the mid-October visit to the tree farm near Mackay, Alberta, T. maxima was also found on stalactiform blister rust cankers on lodgepole pine, P. contorta var. latifolia. This is a range extension northwards of about 150 miles on this pine stem rust.

As indicated in the earlier paper (1) the real distribution of T. maxima probably closely approximates the range of the pine stem rusts, at least in western Canada. Intensive surveys in other areas of Canada may show T. maxima to be present there also.

Acknowledgment

I wish to thank E. J. Gautreau and N. W. Wilkinson of the Northern Forest Research Centre, Edmonton, for collecting two of the specimens reported here.

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CONTROL OF STORAGE DISEASES OF CARROTS BY WASHING, GRADING, AND POSTHARVEST FUNGICIDE TREATMENTS[1]

C.L.Lockhart[2] and R.W.Delbridge[3]

Abstract

Carrots washed and graded before being stored for 15-16 weeks at 32 F (0 C) and 95-100% relative humidity had significantly less decay than carrots stored directly from the field. Treating washed carrots with sodium orthophenyl phenate, by spraying in 1970 and by flooding in 1971, gave a further significant decrease in decay. A postharvest spray rinse of dichloran was effective in one year but not in the other. In 1971-72, thiabendazole gave significantly better control than sodium orthophenyl phenate. Chlorine did not control storage decay. Botrytis cinerea was the predominant rotting organism.

Introduction

In Nova Scotia the storage life of carrots in commercial jacketed cold storage is limited by rots caused mainly by Botrytis cinerea Pers. Losses of carrots after 4-5 months in jacketed cold storage have ranged from 5% to 80%, with the average loss being about 30% (2,3). Losses varied considerably in carrots from different fields, and carrots harvested late in the season usually had more storage decay than those dug early. Van den Berg and Lentz (3) tested several storage environments and found that 32-34 F and 98-100% relative humidity (R.H.) was optimum for control of decay and quality in carrots. They later reported that Sclerotinia sclerotiorum de Bary did not survive or grow in this environment but that some strains of B. cinerea were not inhibited (4).

Hoadley (1) found that dipping unwashed carrots in Dowicide A prior to storage reduced the incidence of decay. In preliminary work we found that washing carrots in water before storage was as effective as some fungicide dip treatments in controlling decay in carrots held at 32 F for 3 months. The effects of washing, grading, and postharvest fungicide treatments on the decay of carrots in a commercial jacketed cold storage are presented in this paper.

Materials and methods

In September 1970, carrots (Daucus carota L. var. sativa DC, cultivar Nantes) machine harvested from field no. 1, Sawler Gardens Limited, Berwick, Nova Scotia, were washed, spray rinsed, and graded in a commerical operation. The prestorage treatments were as follows:

1. Field run control - carrots as received from the field.
2. Washed control - carrots from conveyor following grading.
3. Botran (50% dichloran, Upjohn Company, Kalamazoo, Michigan, USA), 2 lb/100 gal water, sprayed at 200 p.s.i. onto carrots on conveyor belt following rinse.
4. Dowicide A (97% sodium orthophenyl phenate, Dow Chemicals of Canada Limited, Sarnia, Ontario), 0.5 lb/100 gal, applied as in No. 3.
5. Chlorine (Javex) 25 ppm in rinse water.
6. Chlorine (Javex) 50 ppm in rinse water.
7. Chlorine (Javex) 100 ppm in rinse water.

Each treatment was applied to three 18-bushel bulk bins of carrots. The carrots were held for 16 weeks in a jacketed cold storage at 32 F (0 C) and 95-100% R.H., and then sorted into No. 1's, culls, and rots and weighed. Isolations were made onto potato dextrose agar from the rots in a 10 lb sample of carrots from each treatment.

In September 1971 carrot cultivars Touchon and Long Type Nantes from fields no. 1 and no. 18, respectively, were used in a test for control of storage rots. Both fields had received a field spray of maleic hydrazide for sprout control. The prestorage operation was the same as in 1970

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Table 1. Percentage of carrots in different grades following storage at 32°F and 95-100% relative humidity for 15-16 weeks

Prestorage treatment	1970-71			1971-72					
	Field no. 1			Field no. 1			Field no. 18		
	#1	Culls	Rots	#1	Culls	Rots	#1	Culls	Rots
Field run, unwashed	48	22	30	30	16	54	43	22	35
Washed and graded	92	2	6	89	1	10	88	2	10

The 1970-71 and 1971-72 samples are based on 54 and 72 bushels of carrots, respectively.

except that the grower replaced the spray rinse with a flood rinse containing Dowicide A. The treatments were as follows:

1. Field run control - carrots as received from the field.
2. Washed control - carrots washed and graded.
3. Botran (50% dichloran), 2 lb/100 gal of water, sprayed at 200 p.s.i. onto carrots on conveyor belt following washing.
4. Mertect 460 (60% thiabendazole, Merck and Company Inc., Rahway, New Jersey, U.S.A.), 1.12 lb/100 gal, applied as in No. 3.
5. Dowicide A (97% sodium orthophenyl phenate), 0.5 lb/100 gal, applied as a flood rise.

Each treatment was applied to four 18-bushel bulk bins of carrots from each field. The carrots were held in the jacketed cold storage for 15 weeks at 32 F and 95-100% R.H. and then sorted into No. 1's, culls, and rots, and weighed. The rot types were identified visually.

Results and discussion

There was significantly less storage decay in carrots that had been washed, graded, and culled before storage than in those stored directly from the field (Table 1). Less than 1% of the carrots were culled out during grading because of field rots, except in field no. 1 in 1971 when 10% of the carrots had field rots. Removing this source of infection, the cracked and broken carrots and adhering soil, obviously accounts for the decrease in storage decay. The high incidence of storage rots in field run control carrots from field no. 1 in 1971-72, compared with those of 1970-71, was due to heavy rains that occurred in mid-August and flooded some parts of the field for 10 days. Field rots were mainly due to B. cinerea except in those from the flooded field where bacterial rots predominated, followed by rots caused by B. cinerea and Fusarium spp.

Prestorage fungicide treatments of washed and graded carrots significantly decreased storage decay (Table 2). Botran was not effective in 1970-71 but it significantly decreased rots the following year. Sodium orthophenyl phenate and thiabendazole significantly decreased decay in the years they were used. The chlorine treatments were not effective and there was an indication that the higher concentrations may have increased the amount of decay. The causal organisms of the storage rots with their percent occurrence were as follows: B.

Table 2. Percentage rots in carrots stored at 32°F and 95-100% relative humidity for 15-16 weeks

Prestorage treatment	Field no. 1		Field no. 18
	1970-71	1971-72	1971-72
Field run, unwashed	28.9a*	54.5a	35.4a
Washed and graded	6.1 b	9.9 b	10.3 b
Chlorine 25 ppm	7.8 b	**	
Chlorine 50 ppm	11.6 b		
Chlorine 100 ppm	15.8 b		
Botran	6.3 b	1.4 d	1.1 d
Dowicide A	2.6 c	3.6 c	3.5 c
Thiabendazole		1.4 d	1.2 d

* Letters indicate treatments which do not differ significantly in Duncan's Multiple Range groupings at the 5% level.

** Blank spaces indicate no evaluation.

cinerea 51%, bacteria 20%, Alternaria sp. 9%, S. sclerotiorum 7%, Penicillium, Rhizopus, and Fusarium spp. 13%.

Prestorage washing and grading of carrots followed by fungicide treatments markedly reduced losses from storage decay in a jacketed cold storage. The heavy losses in field run carrots reported here were also encountered by van den Berg and Lentz (3). The rot control with sodium orthophenyl phenate agrees with that obtained by Hoadley (1). The grower who cooperated in these tests obtained excellent control of storage decay in carrots with a prestorage flood of sodium orthophenyl phenate in the washing and grading operation. Removal of soil and unmarketable carrots before storage also allowed more efficient use of cold storage facilities. It should be noted that when carrots are to be washed and graded before storage, a field spray of maleic hydrazide is required for sprout control. Normally sprouts developing during storage are removed by the post-storage washing operations.

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A QUALITATIVE SURVEY OF DISEASES OF SOME SPECIALTY CROPS IN SASKATCHEWAN IN 1970 AND 1971: SUNFLOWER, SAFFLOWER, BUCKWHEAT, LENTIL, MUSTARDS, AND FIELD PEA

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Abstract

This survey of diseases of sunflower (Helianthus annuus), safflower (Carthamus tinctorius), buckwheat (Fagopyrum esculentum), lentil (Lens culinaris), mustards (Brassica hirta 'Yellow' and B. juncea 'Oriental' and 'Brown'), and field pea (Pisum sativum var. arvense) was undertaken to identify potential disease problems on crops which could become more important in Saskatchewan. The two most widespread severe disorders were blight of field pea caused by Ascochyta pinodes, and herbicide damage of sunflower. Other noteworthy diseases recorded were alternaria leaf spot of safflower, aster yellows of buckwheat, a late root rot complex and alternaria black spot of the mustards, fusarium root rot of field pea, and sclerotinia stem rot of sunflower, lentil, the mustards, and field pea.

Introduction

In recent years there has been considerable interest in Saskatchewan in crop diversification because of a large oversupply of hard red spring wheat, and a consequent rural economic slump. While the main result has been larger acreages of coarse grains, such as barley, and oilseeds, such as rapeseed (Duczek and Morrall 1971), diversification has also led to increased acreages of several specialty crops (Table 1). With those such as sunflower and field pea, there has simply been renewed interest in a crop grown for a number of years on the prairies, while with others, such as lentil and buckwheat, the crops are relatively new to the province.

The diseases that affect these specialty crops seem to be well known in only some instances. Some idea of the extent of knowledge can be obtained by examining entries in sources such as Conners (1967), the USDA Index of plant diseases (1960) and the host-pathogen index of Review of Applied Mycology (C.M.I., 1968). There have been, at best, only sporadic records of diseases on the crops in Saskatchewan (Conners 1967). Clearly, if these crops are to become established in the province, cognizance must be taken of their diseases, and any serious threats to production should be identified so that attempts at control may be initiated.

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Though acreages may be small in Saskatchewan (Table 1) compared with those of cereals and rapeseed, the crops are of great significance to individual growers. Moreover, it is conceivable that one or more of them could, with changing markets and requirements, show a dramatic increase in acreage such as occurred with rapeseed in the last 10 years. Interest on the part of plant breeders will be a factor in this connection. Another factor may be the extent of new irrigation farming in areas such as the Outlook district; irrigation will, of course, also be important with respect to crop pathology.

This paper is the report of a preliminary survey of the diseases of six specialty crops in Saskatchewan. The work was done over a 2-year period, in 1970 and 1971, but fieldwork was much more extensive in 1971. The crops included were buckwheat (Fagopyrum esculentum Moench), sunflower (Helianthus annuus L.), safflower (Carthamus tinctorius L.), lentil (Lens culinaris Medik.), field pea (Pisum sativum var. arvense L.) and the mustards Brassica hirta Moench 'Yellow', and B. juncea (L.) Coss 'Oriental' and 'Brown'.

Methods

The survey was, of necessity, qualitative. However, this was not considered to be a serious disadvantage since the main purpose was to identify diseases and form an initial impression of their present and potential importance. The numbers of fields of each crop that were visited are shown in Table 1. The approximate locations

Table 1. Estimates of acreage* of six specialty crops and rapeseed in Saskatchewan in the period 1945-1971, and number of fields of each specialty crop surveyed in 1970 and 1971

Crop	Acreage ('000)					No. of fields surveyed	
	Avg 1945-49	Avg 1963-67	1969	1970	1971	1970	1971
Rapeseed	40	480	1,000	2,200	2,750	NA†	NA
Mustards	0	60	180	120	175	1	18
Sunflower	0	10	0	3	65	4	25
Safflower	-**	-	-	40	24	5	7
Buckwheat	0	0	10	25	11	10	4
Field pea	7	2	2	2.5	2.5	2	12
Lentil	-	-	-	1.5	5.6	1	4

* Acreage data compiled from Canada Year Books, 1952-53, 1961, and 1970-71 and Saskatchewan Department of Agriculture Crop Reports, 25 November 1970 and 9 August, 4 October, and 22 November 1971.

** - = No record.

† NA = not applicable.

of fields are illustrated in Figure 1. Visits were made either between July and October 1970 or between August and September 1971. The majority of visits were deliberate, in response to information about field locations received from agricultural representatives. However, some fields were found by accident in connection with other work (Duczek and Morrall 1971), or during vacation trips. Because of this, and also because of limitations of time, it was impossible to attempt quantitative assessments of the diseases observed, based on systematic sampling in several parts of each field. Furthermore, several of the visits were made late in the season when swathing or combining of the crops were complete. At those times the recognition of diseases depended upon the examination of stubble, or thin strips of standing crop at the edges of fields or near power poles. Nevertheless, wherever feasible, observations in fields were based on an examination of plants in two separate parts, or while walking up to about 400 meters through the crop. In this latter case, particular attention was paid to covering both low and higher areas when the topography was clearly uneven. During the survey many specimens were taken to the laboratory for microscopic examination, isolation work, or both, to aid in the identification of pathogens. All isolations were made on potato dextrose agar (PDA).

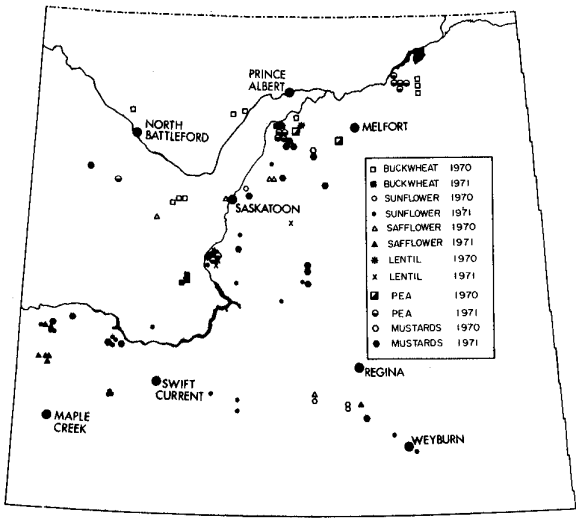

Figure 1. Map of southern and central Saskatchewan showing the approximate locations of the fields surveyed.

Results and discussion

Table 2 presents a summary of diseases encountered in the survey and the numbers of fields in which they were found. New Canadian and Saskatchewan records are indicated with asterisks. The remainder of the text consists of an elaboration of, and comment on, information in Table 2.

Table 2. Diseases of specialty crops in Saskatchewan in 1970 and 1971

Crop	Disease and principal causal pathogen(s)	Number and percentage of fields examined where disease was found			
		1970		1971	
		(No.)	(%)	(No.)	(%)
Sunflower	Herbicide injury	0	0	22	88
	Hail damage	0	0	3	12
	Basal stem rot *(Sclerotinia sclerotiorum)*	1	25	6	24
	Head rot (**Rhizopus* sp., *S. sclerotiorum*, **Mucor spinosus*)	0	0	8	32
	Root rot (*Fusarium* spp., *Rhizoctonia* sp., etc.)	1	25	18	72
	Leaf spots	0	0	16	64
Safflower	Leaf spot *(Alternaria carthami)*	2	40	7	100
	Rust *(Puccinia carthami)*	0	0	2	29
	Root rot (***Fusarium* spp.)	3	60	2	29
Buckwheat	**Aster yellows (Mycoplasma)	3	30	3	75
	**Stem rot *(Botrytis cinerea)*	3	30	1	25
	*Root rot (*Fusarium* spp. & *Botrytis* sp.)	5	50	3	75
Lentil	Sclerotinia stem rot *(Sclerotinia sclerotiorum)*	1	100	1	25
	*Botrytis stem rot (*Botrytis* sp.)	0	0	1	25
	*Root rot (*Fusarium* spp., *Rhizoctonia* sp.)	0	0	3	75
Mustard ('Yellow')	Late root rot complex (*Fusarium* spp., *Rhizoctonia* sp.)	0	0	12	86
	Black spot *(Alternaria* spp.)	0	0	9	64
	Aster yellows (Mycoplasma)	0	0	5	36
	Sclerotinia stem rot *(Sclerotinia sclerotiorum)*	1	100	3	21
	Ring spot *(Mycosphaerella brassicicola* [Duby] Oud.)	0	0	1	7
Mustard ('Brown' and 'Oriental')	Late root rot complex *(Fusarium* spp., *Rhizoctonia* sp.)			3	75
	White rust *(Albugo cruciferarum* S.F. Gray)			2	50
	Black spot *(Alternaria* spp.)			2	50
	Aster yellows (Mycoplasma)			1	25
	Sclerotinia stem rot *(Sclerotinia sclerotiorum)*			1	25
	Downy mildew *(Peronospora parasitica* [Pers. ex Fr.] Fr.)			1	25
Field pea	Ascochyta blight *(Ascochyta pinodes)*	2	100	10	84
	Bacterial blight *(Pseudomonas pisi)*	0	0	2	17
	Sclerotinia stem rot *(Sclerotinia sclerotiorum)*	1	50	7	58
	Root/foot rot *(Fusarium* spp., *Rhizoctonia* sp., *Botrytis* sp.)	0	0	8	67
	Powdery mildew *(Erysiphe polygoni)*	1	50	0	0
	Downy mildew (***Peronospora viciae* [Berk.] Casp.)	0	0	1	8

* New Canadian record.

** New Saskatchewan record.

Sunflower

There was a striking increase in sunflower acreage in Saskatchewan from 1970 to 1971 (Table 1). The majority of fields in 1971 were on dry land in the southern part of the province, but a few in the Outlook district, 60 miles south of Saskatoon, were on irrigated land (Fig. 1). The most prominent damage was caused by herbicide drift which was found in 88% of the fields inspected in 1971. Rough visual estimates of the percentages of plants affected by drift were made in each field, although no account was taken of severity, which is known to vary from plant to plant due to genetic factors. Of 22 fields in which damage was observed 10 were estimated to have only a trace, 3 were estimated to have 1% of the plants affected, 2 estimated to have 5% affected, 4 estimated to have 10% affected, and one each estimated to have respectively 25%, 30%, and 90% affected. The field with 90% was one where it was known that cereals nearby had been sprayed by airplane early in the growing season. It is difficult to relate these estimates of damage to yield losses because of the variable severity on individual plants. The most severely affected plants clearly produced no seed, but less severe damage was manifest by slight malformation of leaves and probably only slight yield reduction. However, it was clear that substantial yield losses were incurred in some fields due to herbicide drift. In view of the frequency of this damage, herbicide drift may be one of the major problems of sunflower production in Saskatchewan. Control will depend not only on greater care in spraying operations by farmers, but also, perhaps, on the development of lines with greater tolerance to herbicides.

Sclerotinia basal stem rot caused by Sclerotinia sclerotiorum (Lib.) de Bary was found in both 1970 and 1971. A single heavy infection found in 1970 in a small irrigated field has been reported elsewhere (Duczek and Morrall 1971). In 1971 infections were found in both irrigated and dryland fields; rough visual estimates were that two fields had about 1% infection and four had only traces. S. sclerotiorum is undoubtedly a potential threat to sunflower production in Saskatchewan, especially on irrigated land, but the presence of the disease depends on local inoculum, which had not had time to build up in the areas where sunflower was grown in 1970-71. As indicated in Table 1, sunflower is relatively new to Saskatchewan and the crop is not yet grown in areas where Sclerotinia is common on other crops (Duczek and Morrall 1971).

It was of interest that where head rot of sunflower (McDonald 1967) was found, S. sclerotiorum was associated with the condition only in one plant. Usually the decayed heads yielded Rhizopus sp. on isolation and contained abundant sporangia of Rhizopus in vivo. One representative Rhizopus culture was identified as R. arrhizus Fischer. Isolation from a rotten head in one field yielded Mucor spinosus van Teigh. In all, head rot was found in 32% of fields surveyed in 1971 but never in greater than trace amounts. However, the latter part of July and August prior to the survey was relatively hot and dry in most of south-central Saskatchewan and these conditions would not have favored the disease.

Other sunflower diseases found relatively frequently in 1971 (Table 2) were a rather nondescript root rot complex and several types of leaf spotting. The etiology of these remains obscure and further work seems warranted. The root rot was usually manifest as brownish stem discoloration, necrotic leaves, and decayed roots, the affected plants being generally scattered throughout a field but occasionally occurring in patches. Such patches were sometimes full of weeds. In some cases the diseased patches were around the perimeter of the field, and in one they were at the bottom of gullies in a hilly field. Affected plants in the patches ranged up to 50%, but elsewhere in the fields, or in fields with only scattered affected plants, the disease was usually only in trace amounts. Isolations from the bases of the stems and the roots of affected plants yielded a variety of fungi and some bacteria but not in a consistent pattern. Nevertheless, the most common isolates were species of Fusarium and Rhizoctonia, which were sometimes associated together and sometimes alone. Hence, these organisms may be etiologic agents of the disease although the limitations of using only PDA to make isolations must be recognized. Drought may also be a factor since the disease was never found in irrigated fields. Verticillium spp. were not isolated from any diseased plants.

The leaf spotting referred to above varied from small black polygonal spots to large spreading brown lesions, and even yellowish lesions in a few cases. Isolation yielded a variety of organisms, but again with no consistent pattern; Fusarium spp., Alternaria alternata (Fries) Keisslers, Rhizopus spp., and even Helminthosporium sativum P.K. & B. [Bipolaris sorokiniana (Sacc. in Sorok.) Shoem., stat. perf. Cochliobolus sativus (Ito & Kurib.) Drechs. ex Dastur] were among the organisms obtained. Many of the isolates were perhaps growing saprophytically on moribund tissue. In most of the fields where leaf spots were seen, infections were in trace amounts, but in four it was estimated visually that 5-10% of the plants were affected, although plants that were affected usually had many spots on each leaf. These facts suggest that much of the spotting may have been physiological or even genetic.

Three well-known sunflower diseases on the prairies (McDonald 1967), leaf mottle (Verticillium albo-atrum Reinke & Berth.), rust (Puccinia helianthi Schu.), and downy

mildew (Plasmopara halstedii [Farl.] Berl. & de Toni) were not encountered during this survey. While it is possible that they were overlooked due to inadequate sampling techniques or inexperience on the part of the observers, it seems more likely that they were absent or very rare. All three depend on local sources of inoculum, which would have been absent in those parts of Saskatchewan where sunflower was grown, except where wild sunflower occurred. However, these diseases will probably appear if sunflower continues to be grown in the same areas and inoculum has time to build up and if climatic conditions are favorable.

Safflower

The survey for diseases of safflower was limited to a few fields in both years (Table 1) and unfortunately most of the fields examined in 1971 were in one limited area in the southwest of the province (Fig. 1). No severe diseases were found either year. The most frequent was alternaria leaf spot caused by Alternaria carthami Chowdhury (Table 2), which varied in intensity from trace to moderate; even with moderate infections not all plants in the field had lesions. This disease and rust caused by Puccinia carthami Cda., which was found in only trace amounts in two fields in 1971, are two of the potentially more destructive diseases of safflower in Saskatchewan. With climatic conditions favorable to their spread and sufficient primary inoculum (for example, seed-borne teliospores of the safflower rust fungus), they could be responsible for substantial yield reductions. The only other disease found in the survey was a root rot, causing necrosis of above-ground plant parts, that was present in trace amounts in about half of the fields. Isolations from such plants consistently yielded Fusarium spp.

Buckwheat

Fewer fields of buckwheat were surveyed in 1971 than in 1970, but this was less a reflection of the decline in acreage (Table 1) than of chance in finding fields. Fields examined in 1971 were in quite different areas from those seen in 1970 (Fig. 1). Only three diseases were found (Table 2) and these were in trace and slight amounts. Aster yellows would probably have been detected in more fields in 1970 if some had not already been swathed or combined when examined. In both 1970 and 1971 some aster yellows infections were estimated at 1% of the plants in the field. The same applied with botrytis stem rot, a disease which has been reported in Manitoba to be seed borne and a potential problem (J.T. Mills, personal communication). Representative isolates were identified as belonging to both Group A and Group B of Botrytis cinerea Pers. (Morgan, 1971). It appeared that Botrytis infected the stems late in the growing season, giving rise to grayish discoloration and wilting of the plant above the infected zone. Moist conditions in late summer might be expected to result in the disease becoming a serious problem through large-scale infection. The majority of fields visited also contained traces of plants suffering from a root/basal stem rot. Isolates from discolored tissue invariably consisted of Fusarium spp. and also occasionally Botrytis sp.

Lentil

There is no mention of lentil in Conners' book (1967); hence, the only published Canadian record of a disease on this crop is one of Sclerotinia sclerotiorum in an earlier paper from our laboratory (Duczek and Morrall 1971). Very few fields of lentil were surveyed as the crop is not easy to find. Three of the four fields in 1971 were on irrigated land. Few diseases were found (Table 2) and those were in only trace to slight amounts; however, the presence of two stem rots should be noted since both have the potential to become serious on irrigated land if inoculum builds up. The botrytis stem rot was characterized by grayish lesions covered with abundant conidia and conidiophores of Botrytis sp. On isolation the fungus proved to sporulate very poorly in culture and it was not possible to identify it to species. However, its cultural characteristics were quite distinct from those of the B. cinerea cultures obtained from buckwheat. As on other crops referred to in this paper, traces of root rot were found in some lentil fields, and isolations from the roots gave Fusarium spp. and in one case Rhizoctonia sp.

Mustards (Yellow, Brown, and Oriental)

The diseases of the various mustards grown on the prairies are well known, and they have been included in several surveys by Petrie and Vanterpool (1965, 1966, 1968). Furthermore, in 1970 and 1971 comprehensive and quantitative surveys of Brassica diseases in Saskatchewan were done by Petrie (personal communication). These will be published in future. Hence, no discussion of mustard diseases will be attempted here. Indeed, the only reason for including the diseases of these species in Table 2 of this paper is that many of the fields visited were in parts of the province not included in Petrie's surveys because cruciferous crops are not common there (Fig. 1).

Field pea

Field pea has been grown in Saskatchewan for many years but the acreage has remained more or less constant (Table 1). New uses for peas currently being investigated in the College of Agriculture, University of Saskatchewan, could lead to an expansion of the acreage. To date, the crop has been grown primarily in two rather limited areas in north-central Saskatchewan where rainfall is adequate; however, the implementation of irrigation in the Outlook district has recently resulted in some fields there too (Fig. 1). Among the 14 fields surveyed

(Table 1), 5 in 1971 consisted of mixed stands with other crops; 3 with oats, 1 with barley, and 1 with rapeseed.

Clearly the most serious disease of field pea was ascochyta blight, which was found in all but two fields. It is noteworthy that the only fields in which it was not found were two in the Outlook district in which bacterial blight caused by Pseudomonas pisi Sackett was found. In all cases the two blight diseases were confirmed by making isolations from diseased leaves, stems, and pods. The isolates of Ascochyta were identified as A. pinodes L.K. Jones. Although only visual estimates were made, the severity of both blight diseases was clearly considerable. In most cases 100% of the plants in the field had lesions, especially on the leaves. With ascochyta blight usually the lower leaves, stems, and pods were most severely diseased, and in several of the fields they were completely covered by lesions. Amounts of disease on the upper portions of the shoots ranged from slight to severe. It was clear that the loss of photosynthetic surface from ascochyta blight must have caused considerable yield reductions, although these could not be quantified. In addition, in fields where pod lesions were severe, it is likely that a large proportion of the seed would be infected by the fungus. Especially if pea acreage increases in Saskatchewan in the future, ascochyta blight will require considerable attention from pathologists and growers.

Another potentially serious problem in pea production is sclerotinia stem rot, which was found in about 50% of the fields in this survey. Where present it was in trace or slight amounts (meaning that it was probably present on 1% or less of the plants). However, field peas are mainly being grown in areas where another host of Sclerotinia sclerotiorum, rapeseed, is widely grown. In view of the ability of the sclerotia of this fungus to survive in soil more than 1 year (McDonald 1967, Morrall unpublished), inoculum could build up and lead to serious infestations and losses in future years, if climatic conditions were favorable. The only other significant disease of pea that was found was root rot caused mainly by Fusarium spp. but sometimes by Rhizoctonia sp. Isolation in one case also gave Botryis sp. Infections of root rot, when present, were in trace amounts in all except one field where a severely diseased patch occurred at a low-lying end of the field. A contributing factor in this case appeared to be temporary waterlogging of the soil at an earlier date.

Acknowledgments

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FOLIAGE DISEASES OF ALFALFA IN NORTHERN SASKATCHEWAN; A NOTE ON THE 1972 SURVEY AND THE DIFFERENTIAL REACTIONS OF NINE VARIETIES[1]

Howard Harding[2]

Abstract

Disease surveys in 1972 showed yellow leaf blotch [Leptotrochila medicaginis] to be causing serious losses in 15 of 44 alfalfa (Medicago sativa) fields. Black stem [Phoma medicaginis] and common leaf spot [Pseudopeziza trifolii f. sp. medicaginis-sativae] each caused losses in four of the fields. In experimental plots, nine varieties of alfalfa were scored for their reaction to natural infection by these three diseases and by downy mildew. The varieties Ferax, Ranger, and Vernal seemed the most susceptible to yellow leaf blotch. Rambler and Rhizoma seemed the least susceptible to this disease but Rhizoma was extremely susceptible to common leaf spot.

Introduction

Previous surveys (1,2) have shown wide ranges in the incidence of the three primary foliage diseases of alfalfa (Medicago sativa L.), yellow leaf blotch [Leptotrochila medicaginis (Fuckl.) Schuepp], black stem [Phoma medicaginis Malbr. & Roum.], and common leaf spot [Pseudopeziza trifolii f. sp. medicaginis-sativae Schmiedeknecht]. These differences do not appear to be related to geographic location, indicating that management practices differ widely or that there are varietal differences in reaction to each of these diseases. As in prior years, a disease survey was conducted in 1972. However, as it is usually not possible to determine on field surveys the varieties being grown, a variety trial was set up at Saskatoon to determine whether varietal differences in reaction to foliage diseases could be detected.

Materials and methods

The 1972 alfalfa disease survey was conducted in mid-July. A total of 44 fields in northern Saskatchewan were visited. The area covered was approximately the same as described previously (2) with many of the fields being located close to alfalfa dehydration plants. Sampling was started about 50 m from the edge of the field and 6-10 samples were taken at about 10-m intervals. Samples were brought back to the laboratory for examination. Disease severity was rated as trace, slight, moderate, or severe.

The variety trial was conducted on the CDA plots at the Research Station Farm, Saskatoon. Seed of nine varieties (Table 2) was obtained from Dr. D. H. Heinrichs, CDA Research Station, Swift Current. Individual plants were grown in plastic "split-tube" containers and were transplanted to the field in June 1971. Four replicated plots of each variety were used. Each plot comprised 126 plants planted in six rows 45.7 cm apart with 21 plants per 610 cm row. Paths 152 cm wide surrounded each plot. The plants were first cut, to a height of 30 cm, in late May 1972. This allowed a build-up of crop debris and presumably a build-up of natural inoculum. Plots were rated for yellow leaf blotch, common leaf spot, black stem, and downy mildew [Peronospora trifoliorum de Bary] in late July 1972. Rating of severity was done as previously described (1) on a scale of 1-10.

Results

The results from the 1972 disease survey are shown in Table 1. Yellow leaf blotch caused serious loss in 15 of the 44 fields while common leaf spot and black stem were serious problems in four fields each. There appeared to be little difference in the severity of each disease with different geographic location. Downy mildew again seemed to be causing real losses in the Shell Lake-Parkside area.

In the experimental plots at Saskatoon, yellow leaf blotch was recorded as early as July 1971. No other foliage diseases were evident in that season. However, sufficient natural inoculum was apparently present, as by mid-July 1972 all four foliage diseases were conspicuous. July was one of the coolest and wettest on record and this

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Table 1. Alfalfa disease survey, 1972

Disease	Disease category and number of fields in each category			
	Trace	Slight	Moderate	Severe
Yellow leaf blotch	21	8	10	5
Common leaf spot	35	5	4	0
Black stem	25	15	4	0

doubtlessly encouraged the build-up of inoculum. The disease ratings for yellow leaf blotch, common leaf spot, and black stem are shown in Table 2. The varieties Ferax, Ranger, and Vernal appeared to be the most susceptible to yellow leaf blotch, while Rambler and Rhizoma seemed to be the least susceptible. However, Rhizoma was very heavily infected with common leaf spot; Beaver, Ferax, Grimm, and Ranger also appeared to be quite susceptible to this disease. Black stem was less evident, although Ferax and Grimm were more heavily infected than the other varieties. The reaction to downy mildew was more difficult to assess as the disease was not distributed evenly within the plots. Generally, however, Ladak appeared to be the most susceptible and Grimm and Rhizoma the least susceptible.

Table 2. Reaction of nine alfalfa varieties to natural infection with three foliage diseases, Saskatoon, 1972

Variety	Disease and disease rating* (avg of four replicates)		
	Yellow leaf blotch	Common leaf spot	Black stem
Beaver	4	6	2
Ferax	8	6	5
Grimm	5	6	4
Iroquois	5	3	2
Ladak	4	4	3
Rambler	2	2	2
Ranger	8	6	3
Rhizoma	2	8	2
Vernal	6	4	2

* 0 = no disease, 10 = severe disease

Discussion

Apparently some of the differences in field survey results can be attributed to varietal differences. It seems obvious that more detailed information on the varieties grown would be of great value. Yellow leaf blotch still appears to be the most serious foliage disease of alfalfa in northern Saskatchewan. This may be due partly to an apparent increase in the acreage of Vernal which seems rather susceptible to the disease. A variety such as Rhizoma apparently has some resistance to the disease but its susceptiblility to common leaf spot would necessitate selections being made very carefully if attempts were made to use this variety in breeding programs. However, it seems probable that an examination of a wider range of genotypes should provide useful resistance to each of the major foliage diseases of alfalfa.

Acknowledgment

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SCREENING OF POTATO FUNGICIDES IN 1972[1]

L.C.Callbeck[2]

Introduction

Weather conditions in Prince Edward Island were generally unfavorable to the development and spread of the potato late blight fungus, Phytophthora infestans (Mont.) de Bary, during the growing season of 1972. Consequently, growers experienced no difficulty in protecting their plantings from attack and experimental work with the disease was hampered.

Materials and methods

In the following list of the fungicides selected for screening in 1972, the description of each is arranged in order of trade name or code number, guaranteed active ingredient, source, and dosage rate per acre in terms of formulated products.

1. Bravo 6F. 6.0 lb/U.S. gal. tetrachloroisophthalonitrile. Diamond Shamrock Canada Ltd., Willowdale, Ontario. 1.0 U.S. pint/acre.

2. Dithane M-45 80W. 80% zinc coordinted maneb. Rohm and Haas Company of Canada Limited, West Hill, Ontario. 1.5 lb/acre.

3. Bravo 6F + Dithane M-45. 0.5 U.S. pint + 1.0 lb/acre.

4. Bravo 6F + Dithane M-45. 1.0 U.S. pint + 1.0 lb/acre.

5. CGF 2630. Confidential mixture and dosage. Ciba-Geigy Canada Ltd., Montreal, Quebec.

6. Difolatan 4.8F. 4.8 lb/Imp. gal. N-(1,1,2,2,-tetrachloroethylsulfenyl)-cis-Δ-cyclohexene-1,2-dicarboximide. Chevron Chemical (Canada) Limited, Oakville, Ontario, Canada. 1.0 Imp. qt/acre.

7. Du-Ter 50WP. 50% fentin hydroxide. Philips-Duphar, Amsterdam, Holland. 10.0 oz/acre.

8. Kocide 101 WP. 83% cupric hydroxide. Kennecott Copper Corporation, Houston, Texas, U.S.A. 1.0 and 2.0 lb/acre.

9. Liro-Martin 45.5W. 34% maneb, 11.5% fentin acetate. Ciba-Geigy Canada Ltd., Montreal, Quebec. 1.8 lb/acre.

10. Manzate 200 80W. 80% zinc coordinated maneb. DuPont of Canada Limited, Montreal, Quebec. 1.5 lb/acre.

11. Polyram 80W. 80% zinc activated polyethylene thiuram disulfide. Niagara Brand Chemicals, Burlington, Ontario, Canada. 1.5 lb/acre.

The plots were planted on June 6 on land that had received a dressing of manure in the fall and a broadcast application of 1,400 pounds per acre of 6-12-12 + 1.2 Mg fertilizer on May 31. Each plot was 4 rows wide by 50 feet long and 50 seed pieces of the variety Green Mountain were planted in each row. Single rows of the same variety were planted as borders and as buffers between plots. The 13 treatments were randomized and replicated in five ranges.

The plants in all rows were sprayed at times appropriate for insect control with endosulfan.

A tractor-sprayer unit, the 4-row boom of which carried four nozzles per potato row,

Table 1. Percentage defoliation

Treatment	Sept. 7	Sept. 15	Sept. 18	Sept. 22
Bravo 6F(1 pt)			Tr*	Tr
Bravo 6F(0.5 pt) + Dithane M-45(1.0 lb)				Tr
Bravo 6F(1.0 pt) + Dithane M-45(1.0 lb)				Tr
CGF 2630	1.5	2.5	5	15
Difolatan 4.8F			Tr	0.5
Dithane M-45				Tr
Du-Ter			Tr	1
Kocide 101(1.0 lb)	2	3	6	11
Kocide 101(2.0 lb)	0.5	1.5	3	6
Liro-Matin				Tr
Manzate 200			Tr	Tr
Polyram			0.5	2
Check	20	35	80	98

* Tr = trace.

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two being above the plants and two on drop pipes, was used to apply the fungicidal mixtures. The applications were made on July 13 and 24, August 1, 9, 18, 29, September 7 and 15, the mean interval being 9.3 days.

Late blight disease was introduced by sprinkling a few plants in each of the border and buffer rows with a water suspension of spores of the race complex 1, 2, 3, 4, 5, 6, 7, 8, 9 in the evening of July 28. A light mist wetted the foliage immediately after the inoculation. Early in the following morning, the foliage being damp, a similar dissemination of spores was made. Both inoculations were successful, lesions being found on August 1.

Because the weather was commonly unfavorable for the fungus, the disease progressed very slowly. The unsprayed check plots were only 20% defoliated on September 7 (Table 1), at which time most of the treated plots were still free of disease. On September 22, when the experiment was terminated by the application of diquat top killer, the check plots showed a mean defoliation of 98% but the means of the treated plots were in the low range of traces to 15%.

Defoliation readings, recorded by means of the British Mycological Society Key (1), were taken at regular intervals and the mean readings for four dates, expressed as percentages, are shown in Table 1.

The plots were harvested and the tubers graded and examined for late blight rot on October 18 and 19. The data are given in Table 2.

Results and discussion

Under the conditions of low disease activity, six spray treatments allowed only trace amounts of late blight to develop. These were Bravo 6F, Dithane M-45, and two mixtures containing both these fungicides, Liro-Matin, and Manzate 200. Mean defoliations allowed by Difolatan 4.8F, Du-Ter, and Polyram were 0.5%, 1.0%, and 2.0%, respectively. The relatively greater defoliations show in Table 1 for Kocide 101 and the confidential fungicide CGF 2630 suggest that these products would be inadequate under blight epidemic conditions.

Phytotoxic reactions were observed in plots sprayed with Du-Ter and with Kocide 101 after the area was subjected to a storm with very strong winds early in September. The plants in the plots that had been sprayed with either of these fungicides showed a scorching of leaf tips and margins, the extent of which injury made subsequent late blight defoliations difficult to estimate. The same type of injury in plots sprayed with Du-Ter has been reported previously (2).

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Table 2. Effects of treatments on yield and rot

Treatment	Total (bu/acre)	Small (bu/acre)	Rot (bu/acre)	No. 1 (bu/acre)	Rot (%)
Bravo 6F(1 pt)	591.4	45.8	0.0	545.6	0.0
Bravo 6F(0.5 pt) + Dithane M-45(1.0 lb)	597.7	45.3	0.4	552.0	Trace
Bravo 6F(1 pt) + Dithane M-45(1.0 lb)	619.5	44.4	0.0	575.1	0.0
CGF 2630	546.9	43.6	10.3	493.0	1.9
Difolatan 4.8F	582.5	43.3	0.0	539.2	0.0
Dithane M-45	582.8	50.1	1.5	531.2	0.2
Du-Ter	570.9	47.3	0.2	523.4	Trace
Kocide 101(1.0 lb)	538.1	48.6	7.0	482.5	1.3
Kocide 101(2.0 lb)	549.1	46.2	3.3	499.6	0.6
Liro-Matin	548.4	48.8	0.2	499.4	Trace
Manzate 200	591.8	49.7	1.1	541.0	0.2
Polyram	564.3	40.5	1.7	522.0	0.3
Check	521.2	44.7	17.4	459.1	3.3
LSD 0.05	33.1			35.2	
LSD 0.01	44.1			46.9	

A SEPTORIA DISEASE OF KOELERIA MACRANTHA IN ALBERTA AND SASKATCHEWAN[1]

K.A.Pirozynski[2] and J.Drew Smith[3]

Abstract

A fungus regularly associated with extensive sheath and culm blackening of Koeleria macrantha, June grass, and collected from widely distributed locations in native pasture in Alberta and Saskatchewan is described as Septoria andropogonis J.J. Davis forma specialis koeleriae. Severe infection may reduce seed fertility in this grass and reduce its competitive ability. S. calamagrostidis (Lib.) Sacc. f. koeleriae (Cocc. & Mor.) Sprague is newly recorded in Canada, and S. quinqueseptata Sprague is discussed.

Introduction

Specimens of Koeleria macrantha (Ledeb.) Spreng. (= K. cristata (L.) Pers.), June grass, showing extensive blackening of lower sheaths and culms were collected in several locations in Alberta and Saskatchewan in the fall of 1970 and 1971. K. macrantha is a long-lived bunch grass found from Ontario to British Columbia in native pastures from the short grass prairie to the open woods of the northern boreal forest (1). Although rarely dominant, it is a useful, palatable constituent of native grazings in western North America (1). Recent studies in the stem eyespot disease of Festuca rubra L. caused by Didymella festucae (Weg.) Holm (9,10) suggested that heavy fungal infections of the sheath and culm may reduce seed yield. The disease on K. macrantha was first noticed because of the pronounced blackening of culms similar to that produced by D. festucae on F. rubra. The disease may reduce seed yield, seed fertility, which is reported to be low in K. macrantha (1), or both. Since there is little stooling in the species this may be a factor controlling its competitive ability. Although collections were widely distributed in the two provinces, all specimens were collected in habitats at the moister end of the ecological range for the species, in seasonally dry, shallow ditches or on drainage slopes. In these situations this grass was the dominant member of the Gramineae.

The fungus

The Septoria sp. that was invariably associated with the culm and leaf sheath blackening is here referred to as S. andropogonis J.J. Davis forma specialis koeleriae. Septoria calamagrostidis (Lib.) Sacc. forma koeleriae (Cocc. & Mor.) Sprague, and several saprobic fungi, namely, Mycosphaerella tassiana (de Not.) Johanns., Platyspora permunda (Cke.) Wehm., and Stagonospora graminella Sacc., were also found on some of the specimens.

Two species of Septoria are known to occur on Koeleria, S. koeleriae Cocc. & Mor. and S. quinqueseptata Sprague. S. koeleriae was described by Cocconi and Morini (2) from K. gerardii (Vill) Shinners (=K. phleoides (Vill.) Pers.) in Italy. Sprague (5) reduced it to a form of S. calamagrostidis (Lib.) Sacc., and recorded it as occurring on K. macrantha in the western United States. We have identified this fungus, S. calamagrostidis f. koeleriae, on one of the Saskatchewan specimens (vide infra). It does not appear to have been previously reported from Canada, though the type form occurs in Alaska (3). S. calamagrostidis f. koeleriae is readily recognized by the acicular, obscurely septate conidia, which are only about 1.25 µm wide (Fig. 2C). S. koeleriae Cocc. & Mor. var. macrocarpa Rayss (4) on K. gerardii in Israel and S. koeleriae var. koeleriae vallesianae Unamuno (11) on K. vallesiana (All.) Bertol. in Spain hardly justify varietal distinction.

The second species, S. quinqueseptata, was described by Sprague (5) from a poor specimen on Sphenopholis obtusata (Michx.) Scribn. In the type specimen, which we have examined (Mandan, N. Dak., J.T. Sarois, July 31, 1915, USDA 80732 in BPI), the conidia are stouter than those of S. calamagrostidis f. koeleriae, 50-70 x 2-2.5 µm, and usually distinctly 5-septate (Fig. 2B). As already pointed out by Sprague (6,7,8) this fungus may only represent a form of S. andropogonis

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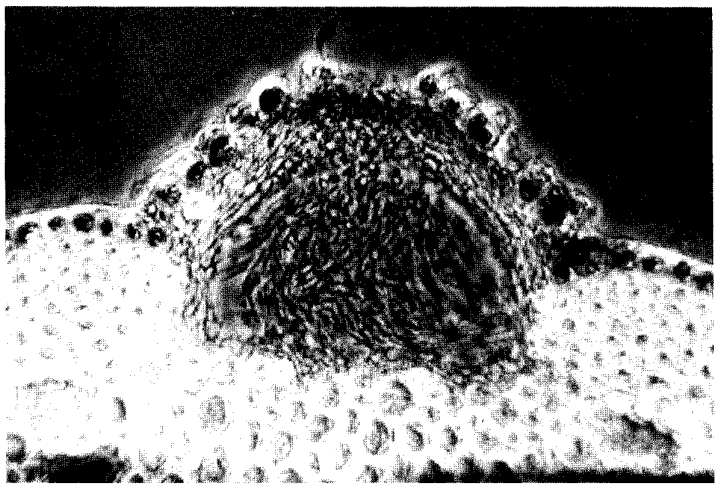

Figure 1. *Septoria andropogonis* f. sp. *koeleriae*, vertical section of pycnidium, from DAOM 138178a, X 500; note fungal hyphae in the host epidermis.

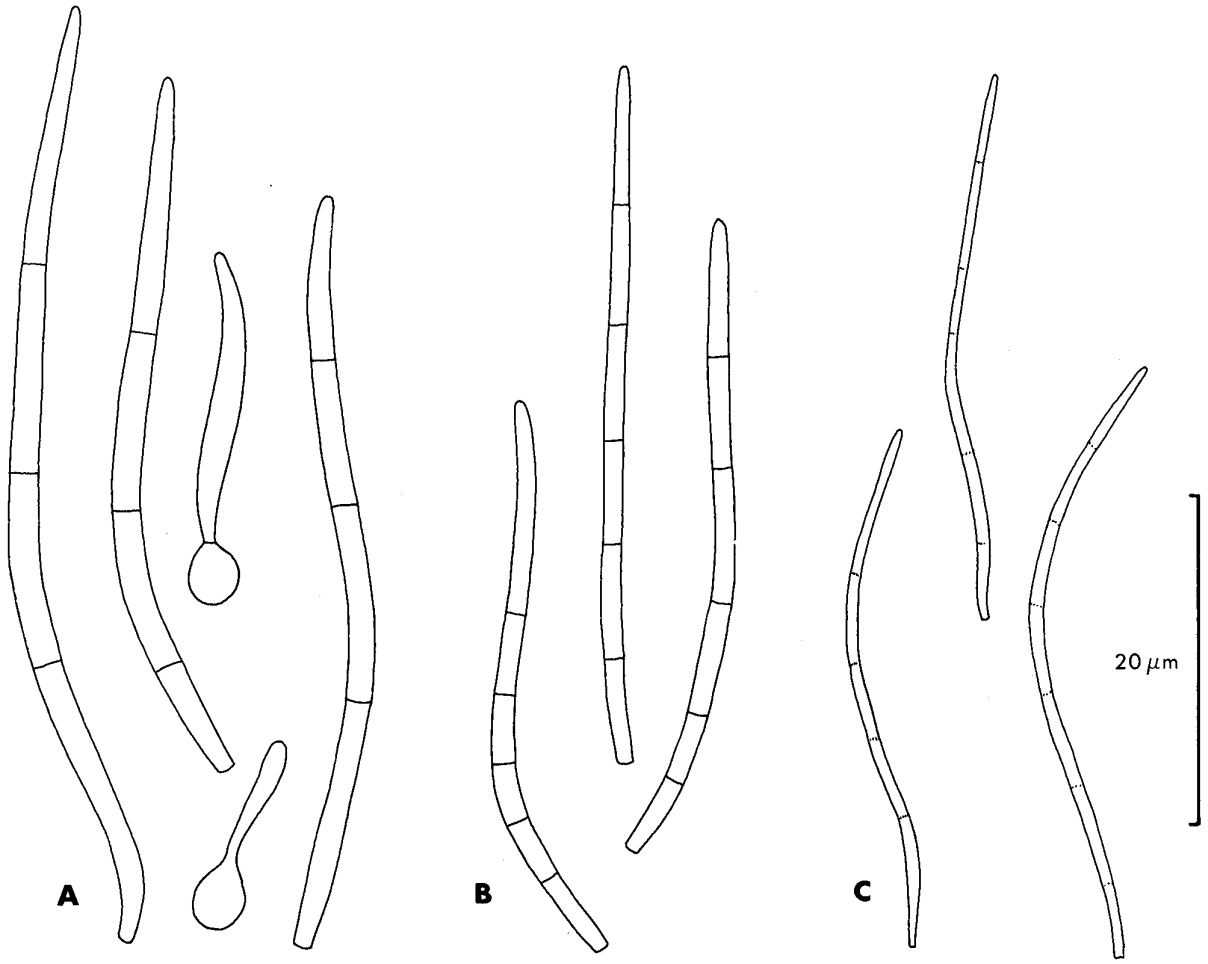

Figure 2. A) *Septoria andropogonis* f. sp. *koeleriae*, conidiogenous cells and conidia, from DAOM 138176; B) *Septoria quinqueseptata*, conidia, from type; C) *Septoria calamagrostidis* f. *koeleriae*, conidia, from DAOM 133191.

J.J. Davis. The species was subsequently recorded on K. macrantha, also at Mandan, North Dakota, by Sprague (7) and stated to be doubtfully parasitic and possibly representing an accidental development on this grass. We are of the opinion that the Sprague collection on Koeleria is not S. quinqueseptata, but is very probably the same as our Canadian fungus only less well developed. The Canadian material is considered to represent a form of S. andropogonis, and we propose to designate it as S. andropogonis J.J. Davis f. sp. koeleriae.

The fungus resembles the type form in globose shape of the conidiogenous cells, and in the shape of the conidia, which are also 3-septate but somewhat longer, up to 90 µm but mostly 50-75 µm (Fig. 2A). It differs in symptoms, which are manifested by conspicuous blackening of culms and leaf sheaths, and which are due to extensive development of brown mycelium in the host epidermis (Fig. 1).

Specimens examined (all on K. macrantha collected by J. Drew Smith): 1) Pipestone Creek, Alberta, 13 Sept. 1970 (DAOM 133189); 2) Pipestone Creek/Wapiti R., Alberta, 16 Sept. 1970 (133190a, with Mycosphaerella tassiana); 3) Meath Park, Sask., 26 Sept. 1970 (133191a, with Septoria calamagrostidis f. koeleriae); 4) Canwood, Mt. Nebo, Sask., 30 Sept. 1970 (138175a, with Stagonospora graminella); 5) Pike Lake, Sask., 16 Nov. 1970 (138176a, with Platyspora permunda); 6) Dundurn, Sask., 21 Nov. 1970 (138177); 7) Candle Lake, Sask., 14 June 1971 (138178a, with Mycosphaerella tassiana).

Acknowledgments

We are indebted to the curator of herb. BPI for the loan of specimens, and to Dr. R.A. Shoemaker for the photomicrograph.

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WINTERKILL PATTERNS OF FORAGE CROPS AND WINTER WHEAT IN P.E.I. IN 1972[1]

Michio Suzuki[2]

Abstract

Prolonged subfreezing soil temperatures during the winter months of 1972 resulted in one of the most severe plant winterkills in the history of Prince Edward Island. Red clover (Trifolium pratense) sustained the greatest loss, followed by alfalfa (Medicago sativa), winter wheat (Trifolium aestivum), and orchardgrass (Dactylis glomerata). Birdsfoot trefoil (Lotus corniculatus) and bromegrass (Bromus inermis) sustained less damage. The hardiest crop was timothy (Phleum pratense) but even this crop did not escape damage. The lethal damage to individual crop species occurred at different times. Red clover plants were killed in February or earlier, orchardgrass in April, and birdsfoot trefoil in May. Winter wheat and bromegrass lost vitality gradually throughout the winter and were dead by spring. Despite the severe winter conditions, very few cases of plant damage caused by frost-heaving were observed.

Climatic conditions

Air temperature at Charlottetown, Prince Edward Island during the winter months of 1972 was slightly below the average for the past 63 years (Fig. 1), but the soil temperature was much below the average for the past 12 years (Fig. 2). The low soil temperature resulted from the abnormally high rainfall during January and the first two weeks of February with 52% and 86%, respectively, of the total precipitation falling as rain during these periods. The resulting absence or lightness of snow cover meant that many areas of P.E.I. were exposed to cold air temperatures or were covered with ice sheets. On February 9 the soil temperature reached record lows of -12.8, -8.9, -6.7, 0 and +2.2 C at depths of 5, 10, 20, 50 and 100 cm, respectively, and the top 50 cm of soil remained frozen until May, being subject to occasional alternate freezing and thawing in March and April. Under these circumstances, one of the most severe winterkills in the history of P.E.I. occurred in 1972.

Extent of damage

Surveys were conducted in June to determine the extent of damage to forage legumes, grasses, and winter wheat. The number of surviving and dead plants was counted with each of ten 1 ft^2 areas chosen at random in each field, and the percent survival for individual crops was estimated at various locations of P.E.I.

Damage to red clover (Trifolium pratense L.) and alfalfa (Medicago sativa L.) was great in most areas except in a few fields situated close to coastal waters. Birdsfoot trefoil (Lotus corniculatus L.) was damaged to a lesser extent. It was noted that damage to the legumes grown in mixtures with grasses, particularly those protected by oat stubble or other companion crop, was much less than in pure stands of legumes.

Among grass species, timothy (Phleum pratense L.) was the hardiest, while orchardgrass (Dactylis glomerata L.) and winter wheat (Triticum aestivum L.) were severely damaged. Winterkill of bromegrass (Bromus inermis Leyss.) in poorly drained fields was also extensive.

There was also a great loss of strawberry plants, almost all mother plants being killed. Daughter plants survived in a few fields.

The extent of winterkill varied considerably, being dependent upon location, drainage conditions, soil type, plant species, cultivar, age, and management regime.

Winterkill patterns

In the spring, patches of winterkill with various sizes and shapes appeared in many fields. Almost all plant species within patches were either killed or severely injured, making the boundaries of each patch

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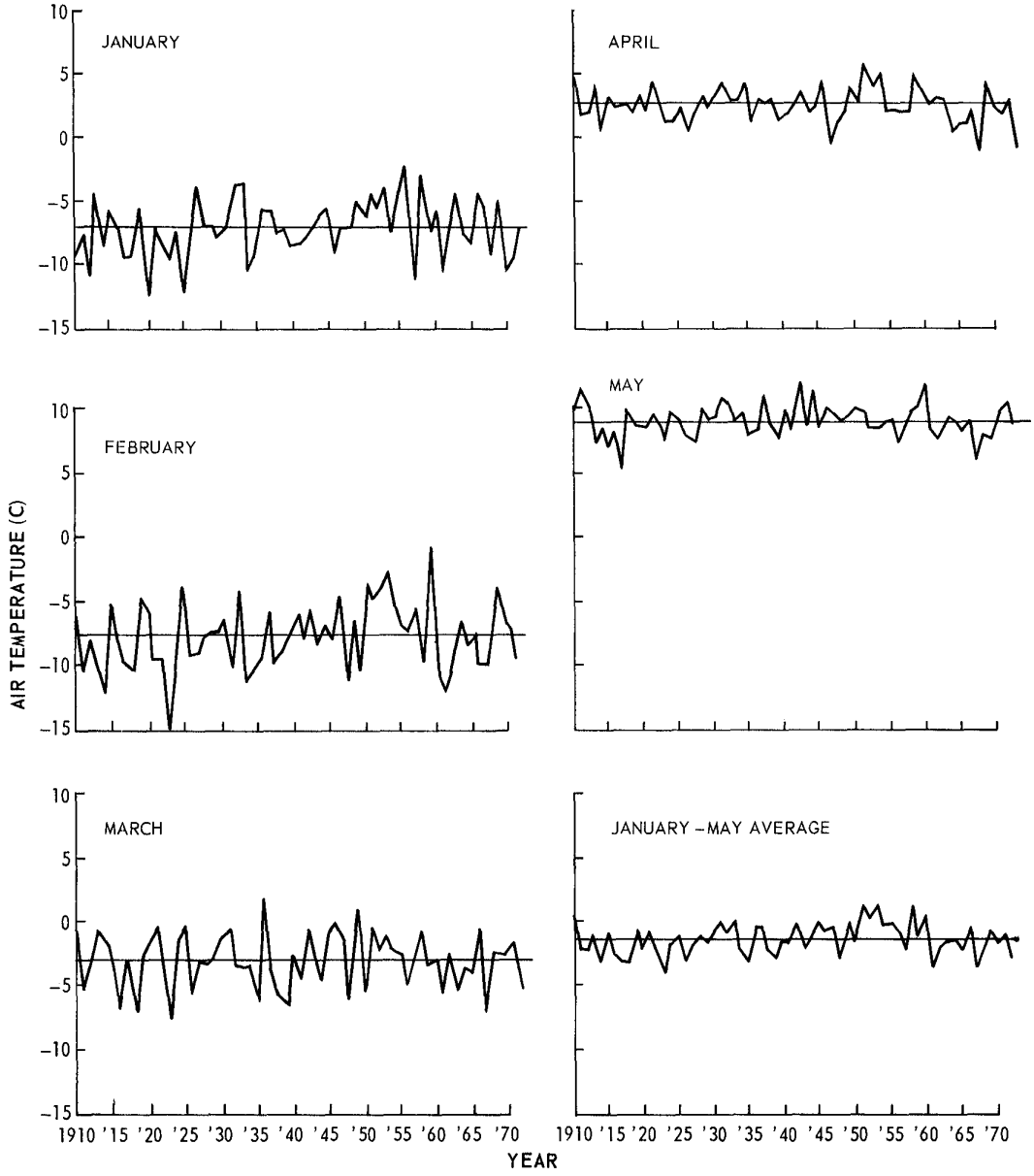

Figure 1. Monthly averages of air temperature at Charlottetown, P.E.I., from 1910 to 1972. Straight lines indicate the 63-yr. averages.

distinct. Other winterkill patterns, such as partial or complete disappearance of legume stands from a grass-legume mixture, but without the formation of characteristic patches, which often occurred in the past, were observed all over P. E. I. There were, however, very few cases of winter injury due to frost-heaving, one of the major causes of winterkill in the past.

In order to determine the time of lethal damage to the individual crop species, with the exception of timothy, which had good survival, sample plants were dug out of the soil during the period from February 17 to May 26 and taken to a greenhouse. Sampling procedures were attended with the difficulties of reaching fields and of digging root systems from frozen soil. In the present survey, sampling was carried out, using pickaxes, at regular time intervals during the season in selected fields. The use of a tractor equipped with a soil-core sampler for digging as well as a torch for defrosting soils was unsuccessful. Further studies to develop and evaluate more

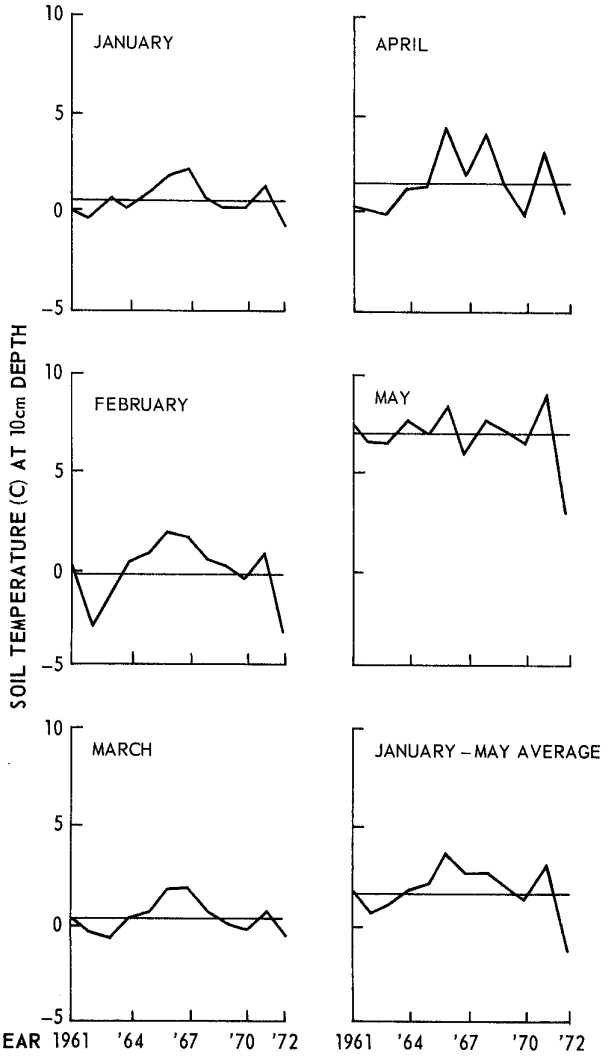

Figure 2. Monthly averages of soil temperature at Charlottetown, P.E.I., from 1961 to 1972. Straight lines indicate the 12–yr averages.

efficient equipment for the purpose are required.

The sample plants were grown in the greenhouse under day and night temperatures of 18C and 13C, respectively. Three weeks after transplanting, the number of surviving and dead plants was counted.

Since judgement of injury or death of plants in the field by means of visual observation was not reliable, the vitality of fresh sample plants was examined by staining crown and root tissues with triphenyltetrazolium chloride (TTC) (1). Staining degrees that reflect plant vitality were scored as 3, 2, and 1 for dark red, red, and pink color, respectively. A dead plant was not stained and was scored as 0 (Table 2). It was found that red clover plants were killed or severely injured in February or before. The tap roots of red clover sampled on February 17 were spongy and many of them were rotted. Alfalfa and birdsfoot trefoil plants that survived through February maintained high vitality of crown and root tissues until the third weed of March. Approximately 40% of the alfalfa plants observed in the fields started new top growth during the last week of April, but the root system became spongy, with several incidents of severe root rots, and failed to support the top in May. New shoots of birdsfoot trefoil did not appear above the ground until May, thus receiving less damage than alfalfa.

Orchardgrass was killed in April or earlier. Vitality of grass species in February and March was lower than that of alfalfa and birdsfoot trefoil, but bromegrass lived longer than these legumes. Winter wheat lost vitality gradually throughout the winter and was dead by June.

Table 1. Winter survival of forage legumes, grasses, and winter wheat in 1972

Crop	Survival (%) Range	Mean	Plant age[1] (years)	Cultivar[2]	Pure stand or[3] mixture with	Location[4]	No. of fields observed
Alfalfa	0-42	22	1-4	Narragansett	Timothy	Q-S	6
				Saranac	Bromegrass	Ch	4
				Alfa	Pure	Q-N	2
				Iroquois	Orchard grass		
Birdsfoot trefoil	0-65	37	1-3	Leo	Timothy	Ch	4
				Empire	Pure		
				Viking			
Red clover	0-93	20	1-2	Lakeland	Timothy	Q-S	10
				Unidentified[5]	Pure	Q-N	8
						Ch	4
						K-N	4
						K-S	4
						P-S	4
						P-N	4
Timothy	72-93	83	1-5	Climax	Pure	Q-S	10
				Champ	Red clover	Q-N	8
				Unidentified[5]	Alfalfa	Ch	4
					Birdsfoot trefoil	K-N	4
						K-S	4
						P-S	4
						P-N	4
Bromegrass	15-88	71	1-3	Redpatch	Alfalfa	Ch	6
				Saratoga	Pure		
Orchard grass	0-52	26	1-2	Tardus II	Pure	Ch	3
				Erode	Alfalfa	Q-S	1
Winter wheat	0-36	24		Yorkstar		K-N	9
				Genesee		Ch	3
				Talbot			
				WW1001-1			

[1] One-year-old plants tended to be more susceptible, but not always.

[2] Listed in the order of dominant cultivars in the fields, not including ones in variety trial plots.

[3] Listed in the order of more common practice in the field. Pure stands of legumes suffered most; there was little difference among components of mixtures.

[4] Ch within 5 miles of Charlottetown; Q Queens County except Ch; N North; K Kings County; P Prince County; S South

[5] Information was not available from the farmers, but fields were assumed to be planted mostly to a mixture of 'Climax' timothy and 'Lakeland' red clover.

Table 2. Vitality of field plants sampled during the winter and spring of 1972

Crop		Sampling date: Feb. 17	March: Early	March: Late	April: Early	April: Late	May: Early	May: Late
Alfalfa	% survival[1]	94	98	72	47	26	0	0
	Vitality[2]	2.8	2.8	1.8	1.6	1.5	0	0
Birdsfoot trefoil	% survival	93	96		75		26	0
	Vitality	2.4	2.0		2.0		1.6	0
Red clover	% survival	46	15	0				
	Vitality	1.7	0.9	0				
Bromegrass	% survival			91		78	72	
	Vitality			1.5		1.4	1.2	
Orchard grass	% survival		88		28		0	
	Vitality		1.9		0.9		0	
Winter wheat	% survival	85		71	69		44	32
	Vitality	1.8		1.2	1.2		1.1	0.9

[1] Percent survival of plants removed to the greenhouse.

[2] Vitality index (max. 3.0) for suviving plants = $\frac{3A + 2B + C}{A + B + C}$

where: A = number of very active plants stained dark red with triphenyltetrazolium chloride (TTC);
B = number of active plants stained red with TTC;
C = number of weak plants stained pink with TTC.

AIR-BORNE RUST INOCULUM OVER WESTERN CANADA IN 1972[1]

G.J. Green[2]

The number of air-borne urediospores in Western Canada in 1972 was assessed by exposing vaseline-coated microscope slides for 48-hour periods at six locations in Manitoba and Saskatchewan. The slides were placed in spore traps that held the vaseline-coated surface at 45° from the vertical. The slides were prepared at Winnipeg, except those exposed at Saskatoon, and were mailed to and from each location protected by a wooden frame and carefully wrapped in paper. Precautions were taken to prevent urediospore contamination of the slides. The number of urediospores caught during each exposure was determined by microscope examination of the slides at Winnipeg. Slides exposed at Saskatoon were prepared and examined by the staff of the Agriculture Canada Research Station, Saskatoon, Saskatchewan.

Air-borne urediospores of stem rust and leaf rust were present in Manitoba and Saskatchewan during May (Table 1) and traces of leaf rust were found in early sown wheat fields in the Red River Valley of Manitoba on June 5, about two weeks earlier than usual. Most wheat varieties are resistant to stem rust and, although air-borne inoculum had been relatively plentiful, it was not observed on susceptible varieties until July 17.

Urediospores, especially leaf rust spores, were more abundant in Saskatchewan as the season progressed. Leaf rust spores greatly outnumbered stem rust spores because the main varieties grown in the rust area are resistant to stem rust and moderately susceptible to leaf rust. A late but severe

Table 1. Number of urediospores of stem rust and leaf rust per square inch observed on vaseline-coated slides exposed for 48-hour periods at three locations in Manitoba and three locations in Saskatchewan in 1972

Date	Winnipeg		Morden		Brandon		Indian Head		Regina		Saskatoon	
	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust
May 19-20	0	1	0	1	0	1	1	2	0	0		
21-22	0	3	0	1	0	1	0	1				
23-24	1	1	0	1	0	0	0	2	0	0	0	0
25-26	0	1	0	4	0	0	0	1	0	1	0	0
27-28	1	1	0	12	0	1	1	0	1	1	0	0
29-30	1	1	0	1	0	1	1	2	0	12	0	0
31- 1	1	2	0	1	1	1	0	1	1	4	0	0
May Total	4	10	0	21	1	5	3	9	2	18	0	0
June 2- 3	0	1			0	1	1	1	1	6	0	0
4- 5	0	0			1	0	1	5	1	11	0	0
6- 7	1	2	0	9	0	4	1	19	0	2	0	0
8- 9	1	4	0	3	1	7	1	7	1	9	0	0
10-11	1	19			1	6	0	12	1	18	0	3
12-13	0	3	2	21	0	4	2	11	1	9	0	7
14-15	1	4	1	6	0	17	1	3	1	9	0	5
16-17	0	9	10	66	3	16	1	10	3	48	0	40
18-19					2	9	1	14	0	9	0	34
20-21	0	1	2	2	0	1	1	5	0	5	0	14
22-23	1	9	0	33	4	25	17	136	47	281	0	19
24-25	0	6			2	13	0	7	2	19	0	10
26-27	7	25	0	75	0	4	2	8	0	9	0	34
28-29	2	15	4	29	2	16	2	10	4	31	0	11
30- 1	1	10	1	7	1	21	0	2	1	3	0	25
June Total	15	108	20	251	17	144	31	250	63	469	0	202

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[2] Plant Pathologist

Table 1 (ctd.)

Date	Winnipeg		Morden		Brandon		Indian Head		Regina		Saskatoon	
	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust
July 2- 3	1	5			1	3	0	0	0	0	0	0
4- 5	1	2	4	21	0	6	1	5	1	11	0	12
6- 7	0	3	2	1	13	5	0	4	2	29		
8- 9	4	29	0	1	19	101	5	86	16	258	0	16
10-11	8	59	4	98	0	3	0	28	1	35	0	25
12-13	1	24	9	75	3	51	1	35	3	45	0	41
14-15	0	5	1	41	5	76	0	26	2	17	0	13
16-17	1	39	2	12	5	114	1	50	1	131	0	28
18-19	5	20	0	12	1	15	0	49	0	49	0	55
20-21	1	150	11	219	8	68	0	25	1	8	0	17
22-23	0	161			2	86	0	61			0	9
24-25	1	80	1	142	13	97	2	315	1	124	5	109
26-27	1	20	0	45	0	20	0	11			0	18
28-29	1	107	4	172	1	99	1	192	11	448	5	159
30-31	1	222			6	459	1	132	4	584	4	378
July Total	26	926	38	839	77	1,203	12	1,019	43	1,739	14	880
Aug. 1- 2	4	244	14	434			14	304	1	1,161	1	355
3- 4	16	1,006	99	2,815	16	1,918	45	7,330	23	12,197	4	1,508
5- 6	43	578			19	897	40	5,700	40	11,317	1	803
7- 8	5	37	0	125	12	2,480	21	4,679	46	30,258	6	2,202
9-10	150	1,381	53	2,527	56	2,146	30	3,565	108	29,379	8	3,418
11-12	28	483	209	5,090	45	2,944	35	4,046	105	28,851	16	1,454
13-14	77	1,446			113	1,959	68	6,685	97	16,419	6	468
15-16	26	145	22	250	13	558	11	424	88	1,396	1	49
17-18	29	218	72	286	6	250	44	1,081	277	4,006	14	400
19-20	33	189	90	394	4	111	396	4,716	213	9,880	62	1,158
21-22	46	208	376	484	21	272	18	180	127	2,220	0	17
23-24	49	81	80	111	29	101	45	155	158	1,466	27	30
25-26			162	203	39	158	70	177	262	5,422	45	66
27-28	260*	294*			45	357	401	1,498	1,436	8,286	56	318
29-30	1,405	2,089	2,688	3,578	227	529	647	3,546	1,930	18,884	88	268
31- 1	288	435	810	1,227	475	2,663	861	2,574	1,044	4,765	34	86
Aug. Total	2,459	8,834	4,675	17,524	1,120	17,343	2,746	46,660	5,955	185,907	369	12,600
1972 Total	2,504	9,878	4,733	18,635	1,215	18,695	2,792	47,938	6,063	188,133	383	13,682
1961-71 Average	3,377	9,439	3,657	14,126	2,492	7,673	1,776	13,661	4,460	43,260	827	19,269

* 4-day exposure.

epidemic of leaf rust that developed on these varieties accounts for the large number of leaf rust urediospores observed on the slides. The oat rusts did not develop sufficiently to produce many spores.

The total number of stem rust spores in 1972 was slightly larger than the 1961-71 average and the 1972 total of leaf rust spores greatly exceeded the average (Table 1).

STEM RUST OF WHEAT, BARLEY, AND RYE IN CANADA IN 1972[1]

G.J.Green[2]

Prevalence and importance in Western Canada

Stem rust spores (*Puccinia graminis* Pers.) were found in spore traps in southern Manitoba and Saskatchewan during May and early June. Apparently the spores were carried into Western Canada earlier than usual because stem rust was more widespread in the United States than in the preceding 8 years. Despite the early presence of inoculum, stem rust was not found in Manitoba until July 17, about 2 weeks later than usual. Development was slow, but before the end of the growing season stem rust (*P. graminis* Pers. f. sp. *tritici* Eriks. and E. Henn.) was present on susceptible wheat varieties (*Triticum aestivum* L.) and wild barley (*Hordeum jubatum* L.) across Western Canada. However it was not observed on resistant commercial varieties that occupy most of the wheat acreage in the rust area, and losses were insignificant.

The bread wheat varieties Manitou, Neepawa, and Selkirk, the utility wheat variety Glenlea, and the durum wheats (*T. durum* Desf.) Stewart 63, Hercules, and Wascana continue to show good resistance. The Mexican variety Pitic 62, which was severely attacked by stem rust in 1970 and 1971, was moderately infected in test plots.

Stem rust of wheat, barley, and rye in the rust nurseries

Uniform rust nurseries were planted by cooperators at 29 locations across Canada in 1972. The wheat varieties grown included the susceptible Red Bobs and Mindum; Lee, which is selective for all strains of the "standard" race 15B (C9, C10, C11, C18, C26, C33, C38, C44, C46, C47, C48, and C50); Pitic 62, which is selective for certain strains in the "standard" race group 11-32-113 (C35,

Table 1. Percentage infection of stem rust *(Puccinia graminis* f. sp. *tritici)* on 18 wheat varieties in uniform rust nurseries at 16 locations* in Canada in 1972

	Common wheat													Durum wheat				
Location	Red Bobs	Lee	Pitic 62	Selkirk	Manitou	Neepawa	Kenya Farmer	Napayo	6 Thatcher x Transfer	Exchange	Frontana	R.L. 4255	Glenlea	Mindum	Stewart 63	Hercules	Wascana	D.T. 316
Creston, B.C.	tr**	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Edmonton, Alta.	10	tr	tr	0	0	0	0	0	tr	0	tr	1	0	0	0	tr	0	0
Lacombe, Alta.	10	1	0	0	0	0	0	0	0	0	0	0	0	tr	0	0	0	0
Scott, Sask.	tr	0	0	0	0	0	0	0	0	0	0	0	0	tr	0	0	0	0
Melfort, Sask.	1	tr	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Indian Head, Sask.	30	50	0	0	0	0	0	0	tr	0	tr	0	0	tr	0	0	0	0
Brandon, Man.	50	tr	0	0	0	0	0	0	0	0	0	0	0	tr	0	0	0	0
Durban, Man.	40	10	5	0	0	0	0	0	5	5	tr	10	0	10	tr	0	0	0
Morden, Man.	40	5	1	0	0	0	0	0	50	1	tr	40	0	25	0	0	0	0
Glenlea, Man.	20	5	1	0	tr	tr	tr	tr	tr	tr	5	tr	tr	5	0	tr	0	0
Thunder Bay, Ont.	50	1	0	0	0	0	0	0	0	30	tr	0	0	0	0	0	0	0
New Liskeard, Ont.	50	0	0	0	0	0	tr	0	0	tr	5	0	0	tr	0	0	0	0
Guelph, Ont.	30	tr	5	0	0	0	0	0	tr	0	tr	5	0	tr	0	0	0	0
Appleton, Ont.	20	0	0	0	0	0	0	0	0	0	tr	0	0	0	0	0	0	0
Ottawa, Ont.	70	0	tr	0	0	0	0	0	tr	0	0	0	0	0	0	0	0	0
Kentville, N.S.	20	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

* No rust was observed in nurseries at 13 locations: Agassiz, B.C.; Beaverlodge and Lethbridge, Alta.; Kemptville and Vineland, Ont.; La Pocatière, Quebec, Macdonald College and Normandin, Quebec; Truro, N.S.; Fredericton, N.B.; Charlottetown, P.E.I.; and St. John's West, Nfld.

** tr = trace.

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C41, C51, and C52); the resistant commercial varieties Selkirk, Manitou, Neepawa, Napayo, Glenlea, Stewart 63, Hercules, and Wascana; and the resistant test varieties Kenya Farmer and D.T. 316. The cooperators harvested the nurseries at an appropriate time and sent the sheaves to Winnipeg where rust infection was assessed and collections were made for race identification.

Wheat stem rust was more widespread and infections were more severe than in 1971. It was found in 16 nurseries in 1972 (Table 1) and in 10 in 1971. Evidently, severe infections would have developed in Western Canada if commercial varieties had been susceptible to the prevalent races.

The nurseries also included the barley (Hordeum vulgare L.) variety Montcalm, which is susceptible to wheat stem rust and rye stem rust (P. graminis Pers. f. sp. secalis Eriks. and E. Henn.); the barley varieties Parkland and C.I.10644, and the rye (Secale cereale L.) variety Prolific, which are resistant to wheat stem rust and susceptible to rye stem rust. These differences in reaction and the early maturity of barley compared with rye, account for the different amounts of rust on the barley and rye in the nurseries (Table 2).

Table 2. Percentage infection of stem rust *(Puccinia graminis)* on three varieties of barley and one variety of rye in uniform rust nurseries at 11 locations* in Canada in 1972

	Barley			Rye
Location	Montcalm	Parkland	C.I. 10644	Prolific
Agassiz, B.C.	0	0	0	20
Creston, B.C.	40	20	tr**	80
Brandon, Man.	1	tr	tr	70
Durban, Man.	0	0	0	20
Morden, Man.	tr	tr	tr	60
Thunder Bay, Ont.	5	0	0	tr
Kemptville, Ont.	0	0	0	50
Guelph, Ont.	10	0	0	20
Appleton, Ont.	5	40	20	70
Ottawa, Ont.	0	0	0	40
Kentville, N.S.	0	0	0	tr

* No rust was observed in nurseries at 18 locations: Edmonton, Beaverlodge, Lacombe and Lethbridge, Alta.; Scott, Melfort and Indian Head, Sask.; Glenlea, Man.; New Liskeard and Vineland, Ont.; La Pocatière, Quebec, Macdonald College and Normandin, Que.; Truro, N.S.; Fredericton, N.B.; Charlottetown, P.E.I.; and St. John's West, Nfld.

** tr = trace.

Rye stem rust has become prevalent in recent years but it was less prevalent in 1972 (11 nurseries) than in 1971 (14 nurseries). It was present in all Manitoba nurseries but not in Saskatchewan nurseries. Presumably it was carried into Manitoba by southerly winds but developed too slowly to infect rye in Saskatchewan.

Physiologic races

Physiologic races were identified by six "standard" differential hosts (T. aestivum 'Marquis' and 'Reliance'; T. durum 'Arnautka' and 'Mindum'; T. monococcum L. 'Einkorn', and; T. dicoccum Schrank 'Vernal') and by the formula method. The formulas were determined using the identified resistance genes Sr5, Sr6, Sr7a, Sr8, Sr9a, Sr9b, Sr9d, Sr10, Sr11, Sr13, and Sr14 that had been backcrossed into lines of Marquis. Marquis is known to carry at least three resistance genes, the main one for Canadian races being Sr7b. The variety Norka carries Sr15, but there was evidence for a second gene in this variety. A line of Chinese Spring carrying Sr16 from Thatcher and a selection of Renown carrying Sr17 were also used. Both lines are believed to carry additional resistance genes. The additional genes complicate race identification but they do not appear to cause important errors.

Several cultures identified in earlier years were retested and the genes found in recent years were placed in their formulas (Table 3).

Eight new virulence combinations (C45 to C52) were described in 1972 (Table 3). Formula C45 is for the old race 56A that differs from race C17(56) by being virulent on Sr6. Formula C50 is for the old race 15B-5 (Can.) that is virulent on both Sr6 and Golden Ball. Races 56A and 15B-5 have not been found in the field for many years but they are used often in experimental work at Winnipeg. Five interesting new races (C46, C47, C48, C49, and C51) were discovered in 1972. Race C52(32-113), called C35"S" in 1971, is like C35 but is virulent on Renown (Sr17) and Selkirk.

Most of the resistance genes used are good differentials but Sr15 and Sr16 have serious disadvantages. Both are influenced greatly by temperature and possibly by other environmental factors. Their resistance breaks down completely at times making differentiation between certain races possible only when the environment is favorable. At Winnipeg, the difficulties occur mainly during late spring, summer, and early fall when, despite evaporative cooling, greenhouse temperatures may be high. The resistant reactions are usually distinct at temperatures about 21°C (70°F).

Norka (Sr15) produced infection type 2+ or 3- with cultures of race C14 (38) instead of the usual ; to 2 or X- that resemble the resistant reaction described by Watson

Table 3. Formula (physiologic race) numbers, virulence formulas, and infection types produced on four wheat varieties by stem rust races found in Canada to 1972

Formula and (race) number	Virulence formula		Infection type on*			
	Effective genes	Ineffective genes	Sk	Mit	Np	Ptc62
C1(17)	5,6,7a,9a,9b,9d,10,11,13,17	8,14,15,16	;	0	;	;
C2(17A)	5,6,7a,9a,9b,10,13	8,11,14,15,16				
C3(29-4)	5,6,9a,11	7a,8,9b,10				
C4(23)	5,6,11,17	7a,15,16	;	;	;	;
C5(29-1)	5,9a,9b,9d,11,16	6,7a,8,10,13,14,15,17,GB*	3+	;	1	;
C6(29-2)	5,9a,9b,11,GB	6,7a,8,10				
C7(48)	5,11,GB	6,7a				
C8(48A)	5,11,16	6,7a,15,GB				
C9(15B-1L)	6,7a,8,9a,9b,10,13,15	5,9d,11,14,16	;	;	;	2
C10(15B-1)	6,7a,8,GB	5,9a,9b,9d,10,11,13,14,15,16,17	;	;	;2	1
C11(15B-4)	6,7a,8	5,9a,9b,9d,10,11,13,14,15,16,17,GB	;	;	2	;
C12(11)	6,7a,9a,9b,10,11	5,8				
C13(32,113)	6,7a,9d,10,11,13	5,8,9a,9b,14,15,16				
C14(14,38)	6,7a,10,11,15,16	5	;	;	;2	;
C15(11,32,113)	6,7a,10	5,8,9a,9b,11				
C16(39)	6,7a,11	5,10,15,16	;	;	;	;
C17(11,56)	6,8,9a,9b,9d,11,13,17	5,7a,10,14,15,16	;	;	;	;
C18(15B-1L)	6,8,9a,9b,13,15,17	5,7a,9d,10,11,14,16	;	;	;1	;
C19(10,38)	6,9d,10,11	5,7a,15,16				
C20(11,87)	7a,8,9d,11,13	5,6,9a,9b,10,14,15,16,17	3+	12	2	2
C21(32)	9a,11	5,6,7a,8,9b,10				
C22(32)	9a,9d,13,16	5,6,7a,8,9b,10,11,14,15,17	3+	23	12	2
C23(38)		5,6,7a,10,15,16				
C24(17)	5,7a,9a,9b,10	6,8,11				
C25(38)		5,6,7a,10,11,15	2	3+	3±	2
C26(15B-4)	6,7a,8,9b,13,15	5,9a,9d,10,11,14,16				
C27(33,59)	6,11,17	5,7a,10,15,16	;	;	1	1
C28(18,54)	6,8,9b,9d,11	5,7a,9a,10				
C29(17)	5,6,7a,9a,9d,10,11	8,9b				
C30(29)	9a,9b,9d	5,6,7a,8,10,11				
C31(27)	5,6,7a,10,11					
C32(32)	9a,9b,9d,11	5,6,7a,8,10				
C33(15B-1L)	6,9a,9b,13,15,17	5,7a,8,9d,10,11,14,16	;	;	;2	;
C34(32)	6,7a,9a,9b,9d,11	5,8,10,13,14,15,16				
C35(32-113)	9d,10,11,13,17	5,6,7a,8,9a,9b,14,15,16	;	3±	3±	3+
C36(48)	5,6,7a,11,16	10,15	;	;	;	;
C37(15)	6,8,9a,9b,11,13	5,7a,9d,10,14,15,16	;	;	1	;
C38(15B-1L)	6,8,9a,9b,13,17	5,7a,9d,10,11,14,15,16	;	;	;	1
C39(32-113)	6,9d,10,13,17	5,7a,8,9a,9b,11,14,15	;	;		;
C40(32-113)	6,9d,10,13,17	5,7a,8,9a,9b,11,14,15,16	;	;	2	2,;
C41(32-113)	9d,10,13,17	5,6,7a,8,9a,9b,11,14,15,16	;	3±	23	3+
C42(15)	6,8,9a,9b,11,13,15,17	5,7a,9d,10,14,16	;	;	;	;

Table 3 (ctd.)

Formula and (race) number	Virulence formula		Infection type on*			
	Effective genes	Ineffective genes	Sk	Mit	Np	Ptc62
C43(32)	6,7a,8,9d,11,16	5,9a,9b,10,13,14,15	;	;	;	;
C44(15B-1L)	6,9a,9b,13,17	5,7a,8,9d,10,11,14,15,16	;	;	2	;
C45(56A)	8,9a,9b,9d,11,13,17	5,6,7a,10,14,15,16	;	;	;	;
C46(15B-1L)	6,8,9a,9b,13,15	5,7a,9d,10,11,14,16,17	;	;	;2	;
C47(15B-1L)	6,9a,9b,10,13,17	5,7a,8,9d,11,14,15,16	;	;	;1	;
C48(15B-1L)	6,8,9a,9b,17	5,7a,9d,10,11,13,14,15,16	;	;	12	;
C49(15)	6,9a,9b,11,13,15,17	5,7a,8,9d,10,14,16	;	;	;1	;
C50(15B-5)	7a,8	5,6,9a,9b,9d,10,11,13,14,15,16,17	4	;1	x-	1+
C51(32-113)	9d,10,13	5,6,7a,8,9a,9b,11,14,15,16,17	3+	23	23	3+
C52(32-113)	9d,10,11,13	5,6,7a,8,9a,9b,14,15,16,17	3+	3±	3±	3+

* Sk = Selkirk, Mit = Manitou, Np = Neepawa, Ptc62 = Pitic 62, GB = Golden Ball.

and Luig (1). Norka seems to carry a resistance gene in addition to Sr15 that confers moderate resistance. When effective, Sr16 produces a 3- infection type.

The varieties Selkirk, Manitou, Neepawa, and Pitic 62 are important commercial varieties in Western Canada and their infection types with most races are recorded in Table 3. The variety Yuma is not shown although it has been a helpful differential. It is resistant to all races excepting cultures of race 15B designated 15B-IL. In recent years, its reaction to certain races has been obscure and variable. It is more resistant to the recently occurring strains of race 15B-IL than to earlier cultures.

The only races virulent on the important Thatcher derivatives Manitou and Neepawa (Table 3) are the rare race C25(38) and certain members of the "standard" race 11-32-113 complex (C20, C22, C35, C41, C51). They have not attacked these varieties in the field.

Seventeen races were identified in 1972, when rust was relatively prevalent, compared with 12 in 1971, when rust was not prevalent. There were no important changes in race distribution in 1972 (Table 4). Race C33

Table 4. Distribution by provinces of physiologic races of *Puccinia graminis* f. sp. *tritici* collected on wheat, barley, and grasses in 1972, and frequency of isolation of *P. graminis* f. sp. *secalis* from barley and wild grasses

Virulence formula (race) number	Virulence formula (effective/ineffective host genes)	Number of isolates from:								Total number of isolates	Percent of total isolates
		P.E.I.	N.S.	Que.	Ont.	Man.	Sask.	Alta.	B.C.		
C14(38)	6,7 ,10,11,15,16/5	1	2		9	3	9		1	25	8.9
C17(56)	6,8,9a,9b,9d,11,13,17/5,7a,10,14,15,16		3							3	1.2
C18(15B-1LX)	6,8,9a,9b,13,15,17/5,7a,9d,10,11,14,16				1	9	17	4		31	11.0
C22(32)	9a,9d,13,16/5,6,7a,8,9b,10,11,14,15,17						1			1	0.3
C25(38)	15/5,6,7a,10,11				1		1			2	0.7
C33(15B-1L)	6,9a,9b,13,15,17/5,7a,8,9d,10,11,14,16				19	45	81	16		161	57.1
C35(32-113)	9d,10,11,13,17/5,6,7a,8,9a,9b,14,15,16			1	5	5	21	3		35	12.4
C36(48)	5,6,7a,11,16/10,15								1	1	0.3
C41(32-113)	9d,10,13,17/5,6,7a,8,9a,9b,11,14,15,16					2	1			3	1.2
C42(15)	6,8,9a,9b,11,13,15,17/5,7a,9d,10,14,16						1			1	0.3
C44(15B-1L)	6,9a,9b,13,17/5,7a,8,9d,10,11,14,15,16					3	2			5	1.8
C46(15B-1L)	6,8,9a,9b,13,15/5,7a,9d,10,11,14,16,17						5			5	1.8
C47(15B-1L)	6,9a,9b,10,13,17/5,7a,8,9d,11,14,15,16				1					1	0.3
C48(15B-1L)	6,8,9a,9b,17/5,7a,9d,10,11,13,14,15,16						1			1	0.3
C49(15)	6,9a,9b,11,13,15,17/5,7a,8,9d,10,14,16						1			1	0.3
C51(32-113)	9d,10,13/5,6,7a,8,9a,9b,11,14,15,16,17					1				1	0.3
C52(32-113)	9d,10,11,13/5,6,7a,8,9a,9b,14,15,16,17				2	1	2			5	1.8
Total wheat stem rust isolates		1	5	1	38	69	143	23	2	282	100.0
Rye stem rust isolates					3	60	37		2	102	

Table 5. Distribution by provinces of physiologic races of *Puccinia graminis* f. sp. *tritici* collected on susceptible varieties of wheat and susceptible wild grasses in 1972

Virulence formula (race) number	Virulence formula (effective/ineffective host genes)	Number of isolates from						Total number of isolates	Percent of total isolates
		N.S.	Ont.	Man.	Sask.	Alta.	B.C.		
C14(38)	6,7a,10,11,15,16/5	1	3	2	7			14	7.7
C17(56)	6,8,9a,9b,9d,11,13,17/5,7a,10,14,15,16	3						3	1.6
C18(15B-1LX)	6,8,9a,9b,13,15,17/5,7a,9d,10,11,14,16		1	4	14	1		20	11.1
C33(15B-1L)	6,9a,9b,13,15,17/5,7a,8,9d,10,11,14,16		8	34	66	3		111	61.7
C35(32-113)	9d,10,11,13,17/5,6,7a,8,9a,9b,14,15,16		1		13			14	7.8
C36(48)	5,6,7a,11,16/10,15						1	1	0.6
C41(32-113)	9d,10,13,17/5,6,7a,8,9a,9b,11,14,15,16			1	1			2	1.1
C42(15)	6,8,9a,9b,11,13,15,17/5,7a,9d,10,14,16				1			1	0.6
C44(15B-1L)	6,9a,9b,13,17/5,7a,8,9d,10,11,14,15,16			2	1			3	1.6
C46(15B-1L)	6,8,9a,9b,13,15/5,7a,9d,10,11,14,16,17				5			5	2.8
C48(15B-1L)	6,8,9a,9b,17/5,7a,9d,10,11,13,14,15,16				1			1	0.6
C51(32-113)	9d,10,13/5,6,7a,8,9a,9b,11,14,15,16,17			1				1	0.6
C52(32-113)	9d,10,11,13/5,6,7a,8,9a,9b,14,15,16,17			2	2			4	2.2
Total isolates		4	13	46	111	4	2	180	100.0

(15B-IL) continued to predominate at about the same level as in 1971. It does not threaten the resistant varieties grown in Western Canada. Three races C14(38), C18(15B-ILX) and C35(32-113) were moderately prevalent. Race C14 more than doubled its prevalence in 1971; race C18 recovered from near extinction in 1971; and race C35 was identified about half as many times as in 1971. Race C35 is virulent on Pitic 62 and moderately virulent on seedlings of Manitou and Neepawa. Reduced planting of Pitic 62 probably accounts for its reduced prevalence.

Fourteen other races occurred in trace amounts and some are worthy of note. The old and well-known race C17(56) was found three times in Nova Scotia. It had not been identified in 1971, for the first time since 1931, but apparently it has persisted in the east although not in Western Canada. The new races (C46, C47, C48, C49, C51, and C52) are variants in the "standard" race complexes 15B-IL and 11-32-113.

The distributions of races isolated from all sources (Table 4) and from susceptible, non-selective, hosts (Table 5) are similar. A few rare races (C22, C25, C47, and C49) were obtained only from selective hosts.

Changes in the percentage of isolates avirulent on the identified resistance genes were small (Table 6). The avirulence of the rust population on Sr8 had decreased sharply in 1971, but genes Sr6, Sr9a, Sr9b, Sr13, Sr15, and Sr17 continue to provide resistance to most isolates.

All cultures were bulked to make 10 composite collections which were used to inoculate 26 highly resistant varieties. Varieties resistant to all composite collections were: C.I. 8155, St464, WRT 240 (Manitou with rye translocation), Agent, Tama, Esp. 518/9, Inia 66, Saric 70, Era, D.T. 332, D.T. 350, D.T. 317, Stewart[8] x R.L. 5244, and Marquis[6] x (Stewart[8] x R.L. 5244). The last two have resistance from T. monococcum at the tetraploid and hexaploid levels, respectively. Important varieties showing susceptible or moderately susceptible type pustules were: Mida-McMurachy-Exchange II-47-26, Frontana-K58-Newthatch II-50-17, Chris, Glenlea, Kenya Farmer, C.T. 615, and Timgalen. C.T. indicates a variety from the Western Canadian Cooperative Test; D.T.

Table 6. Percent of total isolates avirulent on single identified resistance genes and number of avirulent races in 1971 and 1972

Resistance gene	Avirulent isolates (%) 1972 (1971)	Number of avirulent races 1972 (1971)
Sr 5	0.3 (1.6)	1 (2)
Sr 6	83.2 (68.8)	11 (7)
Sr 7a	9.2 (0.8)	2 (1)
Sr 7b	9.2	2
Sr 8	14.5 (3.2)	5 (2)
Sr 9a	74.3 (65.6)	10 (4)
Sr 9b	74.0 (65.6)	9 (4)
Sr 9d	17.2 (31.2)	6 (3)
Sr 10	25.0 (32.0)	6 (4)
Sr 11	25.2 (30.4)	7 (5)
Sr 13	89.8 (96.8)	13 (7)
Sr 14	0.0 (0.0)	0 (0)
Sr 15	79.4 (59.2)	6 (2)
Sr 16	9.5 (0.0)	3 (0)
Sr 17	87.7	11

indicates a variety from the Durum Test. No new races were isolated from the susceptible pustules.

Acknowledgments

The valuable contributions of cooperators across Canada who cared for rust nurseries and sent rust samples for identification are gratefully acknowledged. Mr. J. H. Campbell was responsible for the technical aspects of the survey.

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LEAF RUST OF WHEAT IN CANADA IN 1972[1]

D.J.Samborski

Disease development and crop losses in Western Canada

Wheat leaf rust caused by Puccinia recondita Rob. ex Desm. was unusually widespread in 1972 extending into the Peace River area of Alberta. However, appreciable damage was limited to Manitoba and southeastern Saskatchewan. Leaf rust was first observed in the Red River Valley of Manitoba on June 5. It developed slowly but by August 8 moderately heavy infections were present in the most advanced fields and by the end of August severe infections were general. Early sown fields escaped with little damage, but late sown fields suffered losses estimated at up to 10%. The average loss in Manitoba and southeastern Saskatchewan was estimated at less than 5% of the potential yield.

Leaf rust in the rust nurseries

Ratings of leaf rust intensity on 18 wheat (Triticum aestivum L.) varieties grown at nurseries across Canada are shown in Table 1. The severe infections observed at Lacombe and Edmonton are most unusual and indicate the widespread occurrence of leaf rust in Western Canada in 1972.

Physiologic specialization

In 1972, as in previous years, field collections of leaf rust were established on 'Little Club' wheat in the greenhouse and one single-pustule isolate was taken from each collection. Most of the collections in Manitoba and Saskatchewan were obtained from commercial fields of 'Manitou' or 'Neepawa'. These varieties do not possess any seedling genes for leaf rust resistance.

Table 1. Percentage infection by *Puccinia recondita* on 18 wheat varieties in uniform rust nurseries at 21 locations in Canada in 1972

Location	Lee	Pitic 62	Selkirk	Red Bobs	Manitou	Neepawa	Kenya Farmer	C.T. 432	Hercules	Mindum	Stewart 63	D.T. 316	Wascana	Exchange	Frontana	Tc^6 x Transfer	R.L. 4255	Glenlea
Creston, B.C.	0	0	5	70	tr*	tr	0	0	40	tr	0	20	10	0	0	0	0	0
Edmonton, Alta.	40	45	40	85	65	65	40	70	20	5	10	30	20	0	tr	0	0	0
Lacombe, Alta.	60	60	60	80	65	60	50	50	15	5	10	40	10	0	5	0	0	0
Lethbridge, Alta.	0	5	tr	20	tr	5	tr	5	tr	0	0	tr	0	0	0	0	0	0
Indian Head, Sask.	20	5	10	60	25	30	25	40	tr	0	0	tr	5	0	tr	0	0	0
Scott, Sask.	tr	0	tr	15	15	15	3	3	tr	0	0	0	0	0	0	0	0	0
Melfort, Sask.	5	5	10	25	15	10	10	5	0	0	0	5	0	0	0	0	0	0
Brandon, Man.	50	25	45	90	65	65	25	60	10	0	0	15	5	0	0	0	0	0
Durban, Man.	50	40	30	90	50	50	20	40	40	tr	tr	5	40	0	0	0	0	0
Morden, Man.	60	50	50	90	60	60	40	50	20	3	tr	40	25	0	0	0	0	0
Glenlea, Man.	30	20	20	40	30	20	5	20	5	tr	tr	3	1	tr	tr	0	tr	tr
New Liskeard, Ont.	10	25	25	80	65	65	50	70	35	tr	tr	40	35	0	0	0	0	tr
Kemptville, Ont.	0	tr	0	80	0	0	tr	0	0	0	0	0	0	0	0	0	0	0
Thunder Bay, Ont.	0	tr	5	30	10	10	0	25	0	0	0	0	0	0	0	0	0	0
Guelph, Ont.	50	25	0	85	15	15	40	tr	tr	2	5	25	25	0	0	0	0	tr
Ottawa, Ont.	5	5	5	50	15	15	20	25	25	tr	tr	25	25	0	0	0	0	tr
Appleton, Ont.	0	0	0	50	tr	5	tr	5	tr	0	0	tr	0	0	0	0	0	0
Vineland, Ont.	tr	tr	tr	80	0	tr	10	5	0	0	0	5	0	0	0	0	0	0
Macdonald College, Que.	0	0	0	30	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Kentville, N.S.	40	20	20	75	15	30	35	40	15	0	0	10	0	0	0	0	0	tr
Fredericton, N.B.	5	tr	15	80	0	0	tr	tr	0	0	0	0	0	0	0	0	0	0

* tr = trace.

Eight single-gene backcross lines were used to study physiologic specialization in leaf rust. The distribution of virulence on the individual single-gene lines (Table 2) is

[1] Contribution No. 563, Research Station, Agriculture Canada, Winnipeg, Manitoba R3T 2M9.

Table 2. Virulence of isolates of *Puccinia recondita* on backcross lines containing single genes for resistance to leaf rust in Canada in 1972

Resistance genes	No. of virulent isolates from:						Total no. of virulent isolates	% total isolates
	Maritimes	Que. & Ont.	Man.	Sask.	Alta.	B.C.		
Lr 1	5	5	0	1	0	0	11	6.5
Lr 2A	1	2	0	1	0	0	4	2.4
Lr 2D	1	9	0	1	1	7	19	11.2
Lr 3	5	10	69	55	17	7	163	96.4
Lr 10	5	9	28	25	5	7	79	46.7
Lr 16	0	0	3	4	1	0	8	4.7
Lr 17	1	1	1	0	0	7	10	5.9
Lr 18	0	7	19	12	1	0	39	23.1

Table 3. Virulence combinations of *Puccinia recondita* isolates on backcross lines containing single genes for resistance to leaf rust in Canada in 1972

Avirulence/virulence formula	No. of isolates from:						Total no. of isolates
	Maritimes	Que. & Ont.	Man.	Sask.	Alta.	B.C.	
1,2A,2D,10,16,17,18/3	1	4	26	26	11	0	68
1,2A,2D,16,17,18/3,10	0	0	20	14	3	0	37
1,2A,2D,10,16,18/3,17	0	0	1	0	0	0	1
1,2A,2D,10,16,17/3,18	0	0	14	3	1	0	18
2A,2D,16,17,18/1,3,10	4	2	0	0	0	0	6
1,2A,16,17,18/2D,3,10	0	0	0	0	1	0	1
1,2A,10,16,17/2D,3,18	0	1	0	0	0	0	1
1,2A,3,16,17/2D,10,18	0	5	0	0	0	0	5
1,2A,2D,17,18/3,10,16	0	0	3	3	1	0	7
1,2A,2D,16,17/3,10,18	0	0	4	8	0	0	12
1,2A,2D,10,16/3,17,18	0	0	1	0	0	0	1
10,16,17,18/1,2A,2D,3	0	1	0	0	0	0	1
1,2A,16,18/2D,3,10,17	0	0	0	0	0	7	7
3,16,18/1,2A,2D,10,17	1	0	0	0	0	0	1
10,16,17/1,2A,2D,3,18	0	0	0	1	0	0	1
2A,16,17/1,2D,3,10,18	0	1	0	0	0	0	1
16,18/1,2A,2D,3,10,17	0	1	0	0	0	0	1

similar to the distribution obtained in 1970 and 1971 (1). Most of the isolates were virulent on gene Lr3.

Seventeen virulence combinations were obtained in 1972 (Table 3). The majority of isolates were virulent on only gene Lr3 or on genes Lr3 and Lr10. Only 12 isolates were virulent on more than three of the single-gene lines.

Composite collections of leaf rust were used to inoculate a number of highly resistant varieties. A number of virulent isolates were obtained and further testing indicated that 'Agent', 'Wanken' and 'Preska' all possessed one identical gene for resistance, presumably derived from Agropyron elongatum. This gene is also present in 'Timpaw' which has an additional gene or genes conditioning a moderate level of

resistance to leaf rust. Other studies showed that the variety 'Glenlea' possesses only gene Lr1 conditioning seedling resistance to leaf rust. However, field reactions indicate that this variety must also have genes for adult plant resistance. The variety 'Waldron' has several genes for seedling resistance, probably including genes Lr2A and Lr10.

The identification of genes in these varieties is based on patterns of rust reactions on the varieties and on the single-gene lines. Positive identification would require conventional genetic studies.

Acknowledgments

I am grateful for assistance given by cooperators in the care of the rust nurseries and the collection of rust specimens. Mr. W. Ostapyk performed the technical work of the program.

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STEM RUST OF OATS IN CANADA IN 1972[1]

J.W.Martens

Prevalence and crop losses in Western Canada

Stem rust of oats caused by Puccinia graminis Pers. f. sp. avenae Eriks. and E. Henn. was first found at St-Joseph, Emerson and Horndean in southern Manitoba on July 31. Light infections occurred throughout most of Manitoba and eastern Saskatchewan, but the disease developed too late in the season to cause crop losses, except in the case of a few late fields in western Manitoba and northeastern Saskatchewan, where infections of up to 30% were observed.

Uniform rust nurseries

Oat stem rust infections were light or absent in rust nurseries grown at 27 locations across Canada (Table 1). Rust was observed in only five nurseries and more than trace infections occurred only at Morden, Manitoba. These readings are indicative of the very low levels of rust incidence throughout most of the country.

Identification and distribution of physiologic races

Physiologic races were identified by the methods used in previous years (1). In addition to the oat (Avena sativa L.) varieties with the genes listed in Table 2, a supplementary set consisting of 'Kyto' (pg 12). 'Saia' and R.L. 2926 (pg 13) was used. All 150 cultures identified were avirulent on the supplemental set. The race distribution in Western Canada (Table 2) has developed into the simplest since 1942, when two races comprised 97% of the population. In 1972, two races, C10 and C23, comprised 99% of all isolates from this area. The rapid increase of race C23 since it first appeared in 1969 to 10%, 22%, and 46% of all isolates in Canada in 1970 (2), 1971 (3) and 1972, respectively, is surprising. This race is avirulent on both Pg 2 and Pg 4 resistance which are present, singly or in combination, in most of the oat cultivars grown in Canada. If only collections from hosts with no resistance are considered, it comprised 68% of the population in 1972; this race is obviously highly successful in competition with race C10. The once dominant races C3 and C5 have disappeared, apparently not because of resistance in the host population, but for other reasons.

The frequency of virulence in the oat stem rust population on resistance conferred by genes Pg 2, Pg 4, pg 9 and pg 13 has declined significantly (Table 3) for the first time in 4 years. The almost complete absence of virulence on genes pg 9 and pg 13 is encouraging since these genes are the main components of the current multi-gene-resistance cultivar breeding program.

Table 1. Percentage infection of oat stem rust on 12 cultivars in the uniform rust nurseries* at 5 locations in Canada in 1972

Location	Bond	Trispernia	C.I. 4023	Saia	Rodney ABDH	C.I. 3034	Rodney	Harmon	R.L. 2924	R.L. 2925	R.L. 2926	R.L. 2970
New Liskeard, Ont.	0	tr**	0	0	tr	0	tr	0	0	0	tr	tr
Ottawa, Ont.	0	0	0	0	0	0	tr	0	tr	0	0	0
Durban, Man.	0	0	0	0	tr	0	0	0	0	0	0	0
Morden, Man.	10	tr	15	0	tr	0	40	15	20	10	0	30
Lacombe, Alta.	0	0	0	0	0	0	0	tr	0	0	0	0

* No rust was observed in 22 other nurseries grown at St. John's West, Nfld.; Charlottetown, P.E.I.; Fredericton, N.B.; Kentville and Truro, N.S.; Macdonald College, Normandin, Quebec, and Ste-Anne de la Pocatière, Qué.; Appleton, Guelph, Kemptville, and Thunder Bay, Ont.; Brandon, Man.; Indian Head, Melfort and Scott, Sask.; Beaverlodge, Edmonton, and Lethbridge, Alta.; and Agassiz and Creston, B.C.

** tr = trace infection.

[1] Contribution No. 553, Research Station, Agriculture Canada, Winnipeg, Manitoba, R3T 2M9.

Table 2. Distribution of physiologic races of oat stem rust in Canada in 1972

Race no.	Virulence formula (effective/ineffective Pg host genes)	No. of isolates from: Ont.	Man.	Sask.	Total isolates	Percentage of total isolates
	A. Combined isolates from all hosts					
C 9	8/1,2,3,4,9	1			1	0.7
C 10	9/1,2,3,4,8		54	25	79	52.7
C 20	/1,2,3,4,8,9		1		1	0.7
C 23	2,4,9/1,3,8	1	20	48	69	46.0
Total		2	75	73	150	
	B. Isolates from cultivated oats with stem rust resistance					
C 9		1			1	2.0
C 10			35	12	47	96.0
C 20			1		1	2.0
C 23						
Total		1	36	12	49	
	C. Isolates from wild oats and cultivars with no stem rust resistance					
C 9						
C 10			19	13	32	31.7
C 20						
C 23		1	20	48	69	68.3
Total		1	39	61	101	

Table 3. Frequency of virulence in the oat stem rust population on various types of resistance in western Canada in 1972

Percentage of isolates virulent on cultivars with with following genes for resistance							Total no. isolates	Mean* virulence capability
Pg 1	*Pg 2*	*Pg 3*	*Pg 4*	*pg 8*	*pg 9*	*pg 13*		
100.0	54.0	100.0	54.0	100.0	0.7	0.0	148	4.09

* Mean virulence capability = no. of isolates virulent on *Pg 1* + ... *pg 13*/ total no. of isolates.

Acknowledgments

The assistance of cooperators who cared for rust nurseries and submitted rust collections from various parts of Canada is gratefully acknowledged. Peter K. Anema performed the technical operations necessary for the identification of physiologic races.

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CROWN RUST OF OATS IN CANADA IN 1972[1]

D.J.Samborski and R.I.H.McKenzie

Disease development and crop losses in Western Canada

Oat crown rust caused by Puccinia coronata Cda. f. sp. avenae Eriks. was first found in Manitoba on July 6. By the end of August, a light infection of crown rust was present throughout Manitoba and southeastern Saskatchewan, but damage to the crop was negligible.

Uniform rust nurseries

Ratings of crown rust intensity on 12 oat (Avena sativa L.) varieties grown in nurseries across Canada are presented in Table 1. Crown rust was noted, or could be estimated, in only 8 of the nurseries and infections were generally light. The lines containing crown rust resistance genes Pc 38 (R.L. 2924) and Pc 39 (R.L. 2925) were not attacked by crown rust at any of the locations. However, other larger plots of these two lines showed trace infections at Glenlea, Manitoba, on R.L. 2924 but not on R.L. 2925.

Physiologic specialization

The frequency of occurrence and distribution of 24 physiologic races of crown rust identified from 133 Canadian isolates is presented in Table 2. In 1972, as in 1971, race 295 was predominant in Western Canada (1). Only 12 isolates were established from collections in Eastern Canada but these isolates comprised 9 physiologic races.

Table 2. Distribution of physiologic races of crown rust in Canada in 1972

Physiologic race	West		East	
	No. of isolates	% of all isolates	No. of isolates	% of all isolates
203	16	13.2	0	0.0
209	0	0.0	1	8.3
210	2	1.6	3	25.0
216	11	9.0	0	0.0
226	1	0.8	0	0.0
241	2	1.6	0	0.0
259	1	0.8	0	0.0
276	1	0.8	2	16.7
295	56	46.2	1	8.3
320	1	0.8	0	0.0
326	13	10.7	0	0.0
327	1	0.8	0	0.0
333	2	1.6	0	0.0
335	2	1.6	0	0.0
341	0	0.0	1	8.3
345	2	1.6	0	0.0
360	2	1.6	0	0.0
367	1	0.8	0	0.0
409	0	0.0	1	8.3
415	4	3.3	1	8.3
427	1	0.8	0	0.0
446	1	0.8	0	0.0
1,2,3,10	1	0.8	1	8.3
1,2,3,6,8,9,10	0	0.0	1	8.3

Table 1. Percentage infection of crown rust on 12 oat varieties at 8 localties in 1972

Location	OT 187	Trispernia	C.I. 4023	Saia	Rodney ABDH	C.I. 3034	Rodney	Harmon	R.L. 2924	R.L. 2925	R.L. 2926	R.L. 2970
Glenlea, Man.	0	0	0	0	0	tr	tr	0	0	0	0	0
Morden, Man.	5	0	20	0	20	5	10	20	0	0	10	10
Brandon, Man.	20	0	55	0	70	5	80	65	0	0	20	25
Kemptville, Ont.	25	20	30	0	30	25	70	30	0	0	0	30
Guelph, Ont.	0	0	0	0	5	0	5	5	0	0	0	0
Ottawa, Ont.	25	5	40	0	20	25	50	20	0	0	0	0
La Pocatière, Que.	tr*	0	5	0	0	0	10	5	0	0	tr	tr
Kentville, N.S.	10	tr	15	0	0	tr	30	25	0	0	0	20

* tr = trace.

[1] Contribution No. 562, Research Station, Agriculture Canada, Winnipeg, Manitoba R3T 2M9.

Table 3. Virulence of isolates of *Puccinia coronata* on backcross lines containing single genes for resistance to crown rust in Canada in 1972

Resistance genes	Total no. of virulent isolates	% total isolates
Pc 35	32	24.0
Pc 38	1	0.8
Pc 39	0	0.0
Pc 40	13	9.9
Pc 45	5	3.8
Pc 46	2	1.5
Pc 47	5	3.8
Pc 48	0	0.0
Pc 49	22	16.6
Pc 50	28	21.1

In 1972, all isolates of crown rust were tested on a new set of crown rust differentials based on single resistance genes from Avena sterilis L. backcrossed into 'Pendek' (2). The distribution of virulence on the individual single-gene lines is shown in Table 3. Crown rust in 1972 had a low frequency of virulence on the majority of the new resistance genes.

The single-gene lines were used to classify cultures into virulence formulas. Twenty-one virulence combinations were obtained in 1972 (Table 4). Seventy-seven percent of the isolates were either avirulent on all 10 differentials or virulent on only one, and only 8.0% of the isolates were virulent on more than two of the new differentials.

Acknowledgments

We are grateful for assistance given by cooperators in the care of the rust nurseries and the collection of specimens. Mr. W. L. Timlick performed the technical work of the program.

Table 4. Virulence combinations of *Puccinia coronata* isolates on backcross lines containing single genes for resistance to crown rust in Canada in 1972

Virulence formula (effective/ineffective host genes)	No. of isolates	% total isolates
35,38,39,40,45,46,47,48,49,50/	62	46.6
38,39,40,45,46,47,48,49,50/35	14	10.5
35,39,40,45,46,47,48,49,50/38	1	0.8
35,38,39,40,46,47,48,49,50/45	1	0.8
35,38,39,40,45,47,48,49,50/46	2	1.5
35,38,39,40,45,46,48,49,50/47	5	3.8
35,38,39,40,45,46,47,48,50/49	2	1.5
35,38,39,40,45,46,47,48,49/50	16	12.0
38,39,40,46,47,48,49,50/35,45	1	0.8
38,39,40,45,46,48,49,50/35,47	1	0.8
38,39,40,45,46,47,48,50/35,49	3	2.3
38,39,40,45,46,47,48,49/35,50	6	4.5
35,38,39,45,46,47,48,50/40,49	6	4.5
35,38,39,40,45,46,47,48/49,50	1	0.8
38,39,40,46,47,48,49/35,45,50	1	0.8
38,39,40,45,46,47,48/35,49,50	4	3.0
35,38,39,46,47,48,50/40,45,49	1	0.8
35,38,39,45,46,48,50/40,47,49	1	0.8
35,38,39,45,46,48,49/40,47,50	1	0.8
38,39,45,46,48,50/35,40,47,49	2	1.5
35,38,39,46,47,48/40,45,49,50	1	0.8

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