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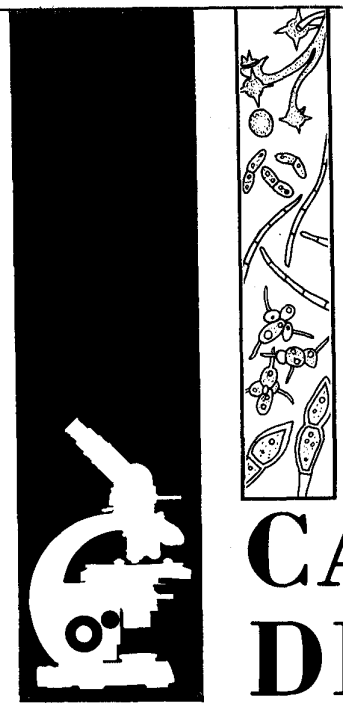

CANADIAN PLANT DISEASE SURVEY

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RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

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"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."

REACTION OF SUSCEPTIBLE AND RESISTANT TOMATO GENOTYPES TO TOBACCO MOSAIC VIRUS IN SOUTHWESTERN ONTARIO

L.F. Gates and C.D. McKeen[1]

Abstract

Tomato genotypes with resistance or tolerance to tobacco mosaic virus (TMV) were tested with 27 TMV isolates from Essex County, Ontario. Genotypes containing genes Tm-2^2 or Tm-2 resisted all the isolates. Those containing Tm-2^2 were agronomically better than those with Tm-2, and the performances of some cultivars with Tm-2^2 that have been released for commercial use were assessed. Although the resistance of these cultivars to TMV was usually satisfactory, small plants developed systemic necrosis when inoculated and kept in unusually hot greenhouse conditions.

Introduction

Because of the trend toward monoculture, greenhouse tomatoes (Lycopersicon esculentum Mill.) are usually infected with tobacco mosaic virus (TMV) in southwestern Ontario. Even after steaming or fumigating the soil, the reservoir of virus is not completely eliminated in root debris, so that the next crop becomes infected about 1 month after transplanting. Breeding for resistance has made progress in recent years and this work has been reviewed by Pelham (10). The gene Tm-1 gives a tolerance to the virus. The genes Tm-2 and Tm-2^2 cause a hypersensitive reaction to the virus, resulting in a form of resistance.

Selections of Lycopersicon species were used by McRitchie and Alexander (8) to group isolates of TMV found in Ohio into 4 classes or strains. Genotypes with Tm-2^2 resisted all four strains. More recently, a further TMV strain, V, was found to overcome the resistance of plants heterozygous for Tm-2^2 at 80-85°F, (27-29C), but not at 60° F(16C) (4).

Since several tomato genotypes with tolerance or resistance to TMV are being grown to a limited extent in Essex County, it was necessary to determine their reaction to a spectrum of local isolates. Genotypes containing Tm-1 and Tm-2 were kindly supplied by Dr. E.A. Kerr, Horticultural Research Institute of Ontario, Vineland Station, Ontario, and genotypes with Tm-2^2 by Dr. L.J. Alexander, Ohio Agricultural Research and Development Center, Wooster, Ohio. The Tm-1 used by Dr. Kerr was from J.M. Walter's material and appears to be the same as Tm-1 of Holmes and other workers (6). Tm-2^2 was derived by Alexander from Lycopersicon peruvianum (L.) Mill. P. I. 128650 (1). We are reporting the behavior of these genotypes when grown under normal spring and fall greenhouse cropping conditions.

Methods

Virus isolates

Twenty-two isolates were obtained from greenhouse tomato crops in Essex County and five from locally available seed. Isolates from seed were multiplied in burley tobacco; for other isolates the original tomato leaves were frozen until required. To inoculate plants, infected leaf tissue was ground in 0.1 M phosphate buffer of pH 7 and rubbed onto leaves powdered with carborundum dust. All soils, pots, and other containers for growing plants were steamed before use.

Preliminary tests with young potted plants

Sets of five seedlings of about 5 cm diameter, planted around the edges of pots 13 cm in diameter, were inoculated on the cotyledons and first true leaves. Symptomless plants were tested for virus by inoculation, as above, of leaves of Nicotiana glutinosa L., or N. tabacum L. cv. Samsun NN.

Tests under normal greenhouse crop conditions

Seeds were sown in flats and seedlings transplanted into pots 10 cm in diameter. Seedlings were transplanted into the greenhouse when 7 weeks old and about 20 cm tall. Plants were 45 cm apart in double rows 55 cm apart, with 75 cm between the pairs of rows. Normal greenhouse procedures were followed in growing the crops.

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Results

A. PRELIMINARY TESTS

The 27 TMV isolates were inoculated, usually individually, to 10 seedlings of each host genotype. All TMV isolates infected all plants of the susceptible cultivar Michigan-Ohio Hybrid, so that symptomless plants of other genotypes were unlikely to have escaped being exposed to infection.

One isolate induced very marked narrowing of the leaflets of Michigan-Ohio Hybrid, but the isolates differed more in symptom severity than in types of symptoms. The degree of multiplication in the plant cells seemed to be involved because isolates to which Lycopersicon peruvianum (L.) Mill. and Lycopersicon peruvianum var. humifusum C. H. Mull. reacted with mosaics tended to produce more necrosis on hypersensitive tomato genotypes than did isolates for which these plants were symptomless carriers.

(i) Reaction of genotypes containing Tm-2.

The three genotypes of Tm-2^2 used (Table 1, entries 1, 4, and 5) were homozygous or nearly so for this gene. They resisted all the virus isolates. Five hundred and sixty-seven seedlings gave resistant (hypersensitive) reactions, evidenced by lack of visible symptoms, by occasional local lesions without further symptoms, or, in eight seedlings only, by systemic necrosis. Eighteen plants of one genotype gave normal mosaics and presumably were segregates lacking the resistance gene. Symptomless plants, however, occasionally contained small amounts of TMV.

(ii) Reactions of genotypes containing Tm-2

Two genotypes of Tm-2 (Table 1) were tested. Plants showing the netted virescent character, which would be homozygous for Tm-2, were discarded. Seedlings groups consisted therefore of plants heterozygous for Tm-2 and plants lacking the resistance gene.

For all the virus isolates, each genotype contained some plants which gave resistant (hypersensitive) reactions. In all, 336 seedlings gave resistant reactions and 150 gave normal mosaics. Of the 336 hypersensitive plants, 67 showed systemic necrosis. Eighteen of the 27 virus isolates produced this symptom on occasion. Resistant symptomless plants occasionally contained small amounts of TMV.

(iii) Reactions of genotypes containing Tm-1

Eleven genotypes were tested with groups of virus isolates. All genotypes contained some plants that developed mottle or mild mosaic. Some genotypes reacted only in this way; others contained some plants that were symptomless carriers of the virus.

B. TESTS OF EXPERIMENTAL RESISTANT GENOTYPES UNDER NORMAL GREENHOUSE TOMATO CROP CONDITIONS

The genotypes listed in Table 1 were grown under fall and spring greenhouse conditions (see Methods and Table 1). The fall experiment was planted in the greenhouse on Aug. 21 and plants were inoculated with TMV on Aug. 23 with a typical local TMV isolate. Fruits were harvested between Oct. 29 and Dec. 5. In the spring experiment, plants were set in the greenhouse on Jan. 30 and inoculated on Feb. 20 with a mixture of the 27 local isolates of TMV used in the preliminary tests. Plants were harvested between April 29 and June 25. Plants in the check plots were not inoculated but in both experiments became naturally infected about 1 month after planting.

(i) Genotypes containing Tm-2^2

All except 3 of the 72 plants inoculated remained free from typical TMV symptoms and appeared essentially healthy. However, traces of TMV were occasionally detected in leaves, suckers, and fruit, and occasional fruit lesions and slight leaf mottle were noted. Yields were good (Table 1), but the fruit had somewhat greenish, ribbed shoulders.

Three inoculated plants developed systemic necrosis. The results of later experiments suggested that young plants inoculated during a period of hot weather are more likely to become systemically necrotic than young or older plants grown under cool conditions following inoculation.

(ii) Genotypes containing Tm-2

Fifteen of 24 plants were resistant (hypersensitive) and two more reacted with systemic necrosis. The other seven showed normal mosaics and presumably did not contain the resistance gene. Later all plants showed mild mosaic symptoms. Resistant plants had lesions on the fruits, which were hollow and of poor shape.

(iii) Genotypes containing Tm-1

Plants of 'Vendor', like the susceptible cultivars used as checks, showed mild to bright mosaics on the leaves and produced fruit which was of good appearance but contained much TMV.

C. TESTS OF COMMERCIALLY AVAILABLE RESISTANT AND SUSCEPTIBLE GENOTYPES

Plants of M-R9, M-R12, W-R25, Michigan-Ohio Hybrid, and Vendor were grown under normal fall and spring greenhouse conditions (see Methods and Table 2). M-R9 and M-R12 are cultivars containing Tm-2^2 released by Alexander (2, 3). Vendor contains Tm-1 (9).

Table 1. Yields of tomato genotypes with genes for resistance to tobacco mosaic virus when grown under normal greenhouse crop conditions* in fall and spring and inoculated with isolates of TMV from Essex County, Ontario

Resistance gene and genotypes		Total yield (g/plant)			
		Fall		Spring	
		Not Inoc.†	TMV**	Not Inoc.†	TMV††
Tm-2^2	66.2144	1630	1745	4897	4114
	64.2144-1^u	2170	1807	4658	5266
	64.2119-B2+	1934	1828	5207	5292
	66.2156	1577	2067	5045	4856
	64.2132-1^u	2014	1979	4411	3524
	66.2161	1852	1609	5198	4913
Mean		1863	1839	4903	4661
Tm-2	153-5	1466	1592	4814	3119
	167-7-10	1453	1616	3457	2607
Mean		1460	1604	4136	2863
Tm-1	Vendor	1424	1383	3547	3411
Susceptible checks					
	Vinequeen	1640	1597	3826	3721
	Michigan-Ohio Hybrid	1795	1733	4486	4717
Mean		1718	1665	4156	4219

* Temperatures (°C):

Fall			Spring			
Weeks of	Night	Day high (2-5 hr)	Weeks of	Night	Day high (2-5 hr)	Occasionally
Aug. 16	21-24	38	Feb. 5-Feb. 26	20	24-27	32
Aug. 23-Sep. 27	16-21	30-35	Mar. 5-Apr. 16	16-17	21-24	30
Oct. 4-Oct. 11	19	24-27	Apr. 23-June 18	16-18	24-30	32
Oct. 18-Dec. 6	19	19-21	June 25	19	35-38	

† Susceptible genotypes became naturally infected about 1 month after transplanting.

** Inoculated 2 days after transplanting with a typical local isolate.

†† Inoculated 3 weeks after transplanting with a mixture of 27 local isolates.

(i) Fall crop 1970

Plants were transplanted into the greenhouse on August 4 and inoculated on August 6 with a mixture of the 27 local isolates used previously. Eleven days later, inoculated plants of Vendor and the two susceptible varieties, Michigan-Ohio Hybrid and W-R25, showed mosaic symptoms, and 7 of the 40 plants of M-R12 had developed areas of systemic necrosis.

Unexpectedly by Sept. 3, 21 of the 40 inoculated plants of M-R9 and 29 of those of M-R12 had developed severe systemic necrotic reactions. These plants made little further growth and produced only unmarketable fruit with brown necrotic areas. This reaction seems to occur when several days of hot greenhouse conditions follow the inoculation of small plants. Noninoculated plants of susceptible cultivars showed clear mosaic from natural infection on Sept. 3, but noninoculated plants of M-R9 and M-R12 showed only local areas of necrosis and by October, except for one plant, appeared to be in good condition.

Yields of M-R9 and M-R12 in the noninoculated but naturally infected plots were similar to those of W-R25 and Michigan-Ohio Hybrid (Table 2). Inoculated plants of M-R9 and M-R12 that resisted systemic necrosis yielded well under reduced competition from the systemically necrotic plants. However, their fruits, especially those of M-R12, on occasion developed necrotic blotches. One truss on a naturally infected plant of M-R9 showed these blotches.

Young and old leaves of inoculated plants of M-R9 and M-R12 that resisted systemic necrosis contained traces of virus. Top shoots of the severely necrotic plants contained much virus.

Table 2. Yields of commercially available tomato cultivars with genes for resistance to tobacco mosaic virus when grown under normal greenhouse crop conditions* and inoculated with isolates of TMV from Essex County, Ontario

	Total yield marketable fruit (g/plant)					
	Fall 1970		Spring 1971			Fall 1971
Cultivar	Not Inoc.†	TMV§	Not Inoc.†	TMV**	TMV††	TMV§§
Resistant						
M-R9	2053	2513	5132	4992	4849	1283
M-R12	2553	2874	5242	5064	4918	1194
Non-resistant						
W-R25	2105	2171	4510	4014	4231	1595
Michigan-Ohio Hybrid	2219	2031	4841			1733
Vendor	1952	1924				2365

* Temperatures (°C):

Fall 1970

Weeks of	Night	Day high (2-5 hr)
Aug. 4-Aug. 26	16-21	35-38
Sept. 2-Sept. 9	16-21	30-32
Sept. 16-Sept. 23	19-21	27-30
Sept. 30-Oct. 15	17-19	24-27
Oct. 23-Dec. 4	17-18	19-21

Spring 1971

Weeks of	Night	Day high (2-5 hr)	Occasionally
Jan. 25-Feb. 25	19-21	24-27	30
Mar. 4-May 7	17-18	24-27	30
May 14-June 23	19-21	27-32	35

Fall 1971

Weeks of	Night	Day high (2-5 hr)
July 30-Aug. 20	18-22	38
Aug. 30-Sept. 29	18-22	27-35
Oct. 6-Oct. 27	17-20	22-24
Nov. 3-Dec. 1	18	18-21

† Susceptible cultivars became naturally infected about 1 month after transplanting.

§ Inoculated 2 days after transplanting with a mixture of 27 local isolates. Yields from plants without systemic necrosis.

** Inoculated 2 weeks after transplanting with the mixture of 27 local isolates or with sap from plants with systemic necrosis.

†† Inoculated 3 weeks after transplanting with the mixture of 27 local isolates or with sap from plants with systemic necrosis.

§§ Inoculated 4 weeks after transplanting with sap from plants with systemic necrosis.

(ii) Spring crop 1971

In the spring crop, cultivars W-R25, M-R9, M-R12, and Michigan-Ohio Hybrid were transplanted to the greenhouse on Jan. 25-27. Plots to receive virus were inoculated 2 weeks after transplanting, when about 30 cm high, or a week later when about 50 cm high. Some plots were inoculated with a mixture of the 27 isolates used previously; others with sap from severely necrotic shoots of M-R9 and M-R12 from the previous fall crop.

Inoculation of young plants of M-R9 and M-R12 with either virus inoculum had caused no systemic necrosis 1 month later, except in two plants of M-R12 inoculated 2 weeks after transplanting with sap from necrotic plants of M-R9 and M-R12. Apart from these two plants, the resistant cultivars showed no virus symptoms during the season, whereas W-R25 inoculated 2 weeks after transplanting showed good symptoms and retarded growth. Occasional fruit lesions were seen on M-R12 in April-June, and more developed on both resistant varieties when the temperatures rose later in the season.

Until April 20, W-R25 showed little yield loss from TMV infection, but its yield was eventually reduced, especially in plants inoculated early (Table 2).

(iii) Fall crop 1971

Plants were transplanted on July 30 and were inoculated with TMV on an upper leaflet on Sept. 1, using sap from necrotic leaves from the fall crop of 1970. Daytime high temperatures in September were 27-35° C compared with 38° C in August. Symptoms appeared 8 days after inoculation in the susceptible cultivars, but the resistant cultivars under these conditions showed no sign of the systemic necrosis that was so frequent in the fall crop of 1970.

Yields of marketable fruit of M-R9 and M-R12 were less than those of W-R25 and Michigan-Ohio Hybrid, and about half the yield of Vendor (Table 2). Total yields (marketable and unmarketable) of M-R9 and M-R12 were 2202 and 2452 g/plant respectively, compared with 2942 from Vendor, but in this experiment much fruit of the two resistant varieties was discarded because of blotchy ripening, hollowness, and blossom-end rot. Michigan-Ohio Hybrid and W-R25, with total yields of 2794 and 2523 g/plant respectively, suffered less from these disorders.

Discussion

The term "resistant" is used here to describe plants which remain as a whole free of nearly free from TMV after inoculation, although the cells whose hypersensitive reaction localizes the virus and prevents its spread are themselves susceptible. Plants which show mild or no symptoms, but which contain much virus, are considered "tolerant".

None of the 27 isolated from Essex County failed to elicit the resistance reaction of Tm-2 and Tm-2^2. Hence TMV strain IV of Alexander was not among these isolates. Local isolates would come into Alexander's classes I - III, corresponding to Pelham's 0 and 1 (0 = I + II) (11). Separation of our isolates into classes I, II and III is not possible since they were only tested in groups on genotypes with Tm-1, but the presence of symptomless carriers and of plants with mosaics suggests the presence of strains II and III. Two hundred and nine isolated collected by Fletcher and MacNeill (5) from tomato crops in southern Ontario came into Pelham's classes 0 and 1, mostly into class 0.

The gene Tm-2^2 showed most promise for TMV resistance and crop quality. However, the development of systemic necrosis in plants homozygous for this gene was a feature of the fall experiment of 1970. In this experiment plants about 20 cm high were inoculated and exposed for several days to temperatures of 35-38 C during the middle of the day. Cirulli and Alexander (4) suggested that the occasional appearance of systemically necrotic plants might be related to inoculum dose and incubation temperature. They did not specify that only their strain V is involved, though this strain was mentioned, and their high temperatures were 27-28°C. At 24-27°C and 27-35° C and with older plants, this necrosis did not develop in our experiments. The virus in our systemically necrotic plants merits study as a possible strain with type V characters, but it may well be that at 35° C the hypersensitive reaction fails to restrict TMV strains in general, as is the case in Nicotiana glutinosa.

The virus that occurs in low levels on occasion in nearly resistant plants homozygous for Tm-2^2 also merits study. Cirulli and Alexander report a similar finding (4). It may be that the hypersensitive reaction is modified in older plants, permitting some virus development. The presence of some TMV in these plants increases the likelihood of development of strains unaffected by the resistance mechanism. Strain selections or strain changes in resistant tomatoes have been shown by MacNeill and Fletcher (7).

In Essex County the two commercial resistant varieties M-R9 and M-R12 if protected from early inoculation and high temperatures appear to resist TMV satisfactorily. To date, M-R12 has been found slightly more susceptible to systemic necrosis and fruit necrosis than M-R9. However, Vendor, Michigan-Ohio Hybrid, and Vinequeen give fruit of good quality even though foliage and fruit contain much TMV. Further study of resistant genotypes meeting the precise nutritional requirements of these cultivars is necessary to determine their usefulness to the greenhouse industry.

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DIDYMELLA STEM EYESPOT OF FESTUCA SPP. IN NORTHERN ALBERTA AND BRITISH COLUMBIA IN 1970 AND 1971[1]

J. Drew Smith[2] and C.R. Elliott[3]

Abstract

Infection of red fescue (Festuca rubra) with the stem eyespot fungus, Didymella festucae (imperfect state, Phleospora idahoensis), was generally light in 1970 but severe in 1971 in the main seed-growing areas of the Peace River region of Alberta and British Columbia. In 1969 the disease had been severe. Disease severity was reflected in seed yield and dockage at a major seed cleaning plant. Actual yields in a very severely infected area near Beaverlodge, Alberta, in 1971 were only 20-25% of the expected yield. Infected native sheep's fescue (Festuca ovina var. saximontana) found in areas of sandy soil within the main seed-growing region seems the most likely natural inoculum reservoir for infection of introduced fescues. However, the more ecologically adaptable introduced red fescue planted along roadsides in the western Canadian provinces may pioneer infection corridors. In a fertilizer test using nitrogen, phosphorus, potassium, and sulfur on red fescue on sulfur deficient soil, nitrogen exercised the greatest influence in reducing disease. In another test within a heavily infected red fescue field, litter removal was ineffective in reducing disease severity.

Introduction

Surveys of the stem eyespot disease of seed crops of red fescue, Festuca rubra L., caused by Didymella festucae (Weg.) Holm (2, 5, 6) (imperfect state, Phleospora idahoensis Sprague) were made in 1969 (3). Yield estimates indicated that considerable seed losses could result from severe infections (2, 3).

Results

1970 Survey

In early September 1970 inspections were made of red fescue in stubble or on roadsides at 23 locations from Grande Prairie in northern Alberta to Fort St. John, British Columbia, and thence to 75 km west of Dawson Creek, B. C. Except for two places west of Dawson Creek less than 35% of stems (stubble) were infected. At 15 points incidence was 1% or less. At six places there was no disease. Berkenkamp (1) found light infections in a survey of nine fields earlier in the season in this region.

1971 Survey

Between 24 and 29 June 1971, 77 seed fields of red fescue in the Peace River regions of Alberta and British Columbia were surveyed for the disease. An estimate was made of the percentage of infected culms on samples plucked at random on one or more transects of each field; the acreage involved was also estimated (Table 1). Approximately half of the 67 fields surveyed in Alberta were very severely infected, whereas only 1 of the 10 in British Columbia was in this category. In Alberta most of the heavily infected fields were in the region 38 km north and south of Highway 2 from Hythe to Grande Prairie. Little infection was found in fields north of Highway 49 from Donnelly to the British Columbia border.

A second short survey was made in early August from Grande Prairie to Goodfare, Alta., near the British Columbia border. Of a total of 11 fields examined, 10 (225 ha) showed 100% stem eyespot incidence and one (32 ha) showed 90%.

Didymella stem eyespot was also found on a seed crop of red fescue near Valleyview, 105 km east of Grande Prairie. It occurred at several roadside locations on that species and on native sheep's fescue (F. ovina L. var. saximontana (Rydb.) Gleason,) as far south as Whitecourt, 272 km southeast of Grande Prairie. A disease rating of 1% was recorded in a hay crop 112 km north of Dawson Creek, British Columbia, off the Alaska Highway. Infected sheep's fescue was found 8 km south of Grande Prairie and on the north

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bank of the Redwil r River due south of Beaverlodge. This is e southern part of the main red fescue s l-growing area.

The survey resu s indicate that the disease on seed crops is much more severe in 1971 than in 1970 id as severe as that reported in 1969 (3). This is reflected in the seed yield sta stics for the 3 years. Canadian seed product on of red fescue in thousands of metric ons (millions of lb. in brackets) was 5.1 (11 5) in 1969, 11.1 (25.0) in 1970, and 5.1 (11.5) in 1971 (data on production from Plant Products Division, Production & Marketing Branch, CDA, Ottawa). Most Canadian red fescue seed is produced in Alberta and practically all of the remainder is grown in British Columbia. Estimated yields in kg/ha (lb/ac in brackets) of creeping red fescue seed produced in 1969, 1970, and 1971 were respectively: Alberta 140 (125), 246 (220), 118 (105); and British Columbia 129 (115), 375 (335), and 196 (175). These yields have been calculated from data supplied by the Alberta, British Columbia, and Canada Departments of Agriculture. Dockage figures from a major red fescue seed cleaning plant at Beaverlodge showed an average cleanout of 25% in 1969, 18% in 1970, and 32% in 1971 (personal communication, N. Foster, Foster's Seed & Feed, Beaverlodge, Alta., Jan. 1972). This fluctuation coincides with trends in disease severity shown by the surveys and with seed yields. At Fort St. John, B. C., average cleanout was 20% in 1971 and this probably reflects the lower disease ratings in British Columbia (Table 1). An estimate of yield made at heading on 180 ha of red fescue in the very severely diseased area near Beaverlodge in 1971 was 250-300 kg/ha; however only 60 kg/ha of clean seed was harvested (personal communication, N. Foster, Jan. 1972). While there is no doubt that total production of red fescue is influenced by factors other than disease, it is our experience, and that of growers, that once seed heads have been formed yields can usually be accurately predicted.

The significance of the finding of infected native sheep's fescue in the main red fescue seed-growing area is that the native grass is the likely natural reservoir of inoculum which infects the introduced species. However, it does not invalidate the possibility that "seed" borne transmission also occurs. It seems probable that the incidence of the disease on the introduced red fescue on roadsides is related to the presence of adjacent infected native sheep's fescue. It is also likely that the sowing of a susceptible, ecologically adaptable species such as red fescue on the sides of highways through areas of light sandy soil which harbor infected F. ovina serves to develop "infection corridors". These may pioneer the spread of infection to disease-free areas where fescue seed production is contemplated (6). The mowers used on road verges may be potent instruments in short-distance disease transmission by infected stem fragments.

Effect of nitrogen, phosphate, potash, and sulfur fertilizers on the incidence and severity of the disease

Differences in the incidence and severity of stem eyespot were found in 1971 in a field test of red fescue designed to study the effects of fertilizers on seed yield.

Red fescue was seeded 20 June 1968 in 6-meter rows spaced 30 cm apart on a sulfur deficient, gray wooded sandy loam (Demmitt series) near Beaverlodge. Fertilizer treatments of P K S, N P K, and N P K S were applied at time of seeding at the following rates in kg/ha; N, 15; P, 20; K, 25; and S, 10. The fertilizer treatments were repeated in the autumns of 1969 and 1970 except for N which was applied at 30 kg/ha. Fescue shoots were clipped from all the rows on 28 June 1971 and 100 of the stems, drawn at random from each of the four replicates of each treatment, were rated for disease on the 0 to 3 scale used in a disease loss study (3). Treatment effects were determined by analysing infection index data calculated from the disease ratings (Table 2).

All fertilizer treatments lowered the eyespot index compared with no fertilizer. The decline was greatest where N P K S were applied, but the difference between the complete fertilizer and the N P K treatment was so small that it seemed that sulfur was not a major factor, even on this sulfur-deficient soil. On the other hand where N was omitted, as in the P K S treatment, the index increased sharply. It appeared from this that N was exercising a major influence on the disease index. The combination P K S reduced the disease index significantly but it was uncertain which of the nutrients had the greatest effect. Until further tests are made all that can be concluded is that nitrogen applications are likely to reduce the incidence of didymella stem spot.

Effect of mowing and litter removal

After harvest, in mid-September 1971, the effect of litter removal treatments on the disease were compared in a heavily infected commercial crop of red fescue near Beaverlodge. The previous crop has been heavily infected with stem eyespot. Treatments were replicated six times on 200-m^2 plots. Treatments following harvest were: 1) check, combined swath left on; 2) straw raked off; 3) and 4) mown at 5 cm, clippings returned (3) and removed (4); 5) and 6) mown at 10 cm, clippings returned (5) and removed (6).

None of these treatments had any noticeable effect on disease incidence or severity in stem samples collected on 28 June 1971. More than 90% of these were in the category rated most severe. The inoculum may have come from a heavily infected field that surrounded the test area or, if the treatments were inefficient in inoculum

Table 1. The incidence of infected culms in crops of red fescue in the Peace River region of northern Alberta and British Columbia in 1971

British Columbia			Alberta		
Infection (%)	Number of crops	ha*	Infection (%)	Number of crops	ha*
0	0	0	0	7	284
0-0.5	2	97	0-0.5	4	150
0.6- 25	6	151	0.6- 25	13	361
26- 50	1	65	26- 50	2	97
51- 90	0	0	51- 90	5	164
91-100	1	10	91-100	35	1183

* To convert hectares to acres multiply by 2.47.

Table 2. The effect of nitrogen, phosphorus, potassium, and sulfur fertilizers on didymella stem eyespot of red fescue

Treatment	Disease index* (avg of 4 replicates)
No fertilizer	65.7**
P, K, S	59.7
N, P, K	41.4
N, P, K, S	40.6

* Calculated from disease ratings on a 0 to 3 scale (3), where 0 is no disease and 3 the most severe category:

$$\text{Disease index} = \frac{(0 \times n_0) + (1 \times n_1) + (2 \times n_2) + (3 \times n_3) \times 100}{300}$$

** L.S.D. 5% 5.76
1% 8.28

removal, from within the plots. Further studies with isolated tests are needed to resolve these points.

Acknowledgments

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PREVALENCE, DISTRIBUTION, AND IMPORTANCE OF DWARF BUNT OF WINTER WHEAT IN ONTARIO 1970-71[1]

V.R. Wallen and A.B. Ednie[2]

Abstract

Field surveys in southwestern Ontario in 1970 and 1971 showed that dwarf bunt caused by Tilletia controversa was present primarily in Huron County. Disease incidence was low; less than 1% of the plants in affected fields showed symptoms of the disease. T. controversa was detected in samples of pedigreed seed produced in Ontario each year from 1960 to 1971.

Dwarf bunt caused by Tilletia controversa Kuhn was first detected in Ontario in 1952 (2,5). The disease caused immediate concern to growers of winter wheat in Ontario because the causal fungus is soil-borne as well as seed-borne. Concern was expressed that seed lots infected with the organism would infect fields formerly free of the organism, resulting eventually in the general distribution of the pathogen in winter wheat soils throughout the province.

Research during 1952-55 revealed that the fungus was present in soils of at least 16 counties in the western part of the province, but was most prevalent in the counties bordering or adjacent to Lake Huron (1). The level of field infection was low, with most fields having only a few bunted heads; however an occasional field was found to have as high as 25% of the heads affected. Although dwarf bunt was not observed in the field in several of the counties bordering Lake Erie, namely Essex, Kent, Elgin, Oxford, Norfolk, and Haldimand, spores of the fungus were detected in seed samples that had originated from fields in these counties. Approximately 2,500 seed samples were examined from 1953 to 1955 and spores of T. controversa were present in samples from counties where the disease had not been observed in the field. Although only the better grades of seed were examined, at least 10% of the samples were contaminated with T. controversa (1).

Following the discovery of dwarf bunt and the initial field and seed surveys for the disease, laboratory and field research revealed that for severe infections of dwarf bunt to occur an unusual combination of climatic factors is necessary. A correlation was shown to exist between temperature, light, and soil moisture and the severity of soil-borne dwarf bunt in field plots (1).

Wagner (6) in Germany using a pentachloronitrobenzene preparation indicated that dwarf bunt could be largely eliminated from field soil, and Holton and Jackson in USA (4) found that soil applications of Anticari, a chlorobenzene dust, significantly reduced dwarf bunt in the field. Fushtey (3) conducted extensive tests in Ontario on the control of both seed- and soil-borne inoculum of the pathogen. Tests over the period 1954-58 showed that mercury and chlorobenzene seed treatments were ineffective for the control of soil-borne inoculum but that chlorobenzene treatments were effective against seed-borne inoculum.

The use of effective seed treatment fungicides eliminated the danger of spread of the fungus from contaminated seed lots to fields free of dwarf bunt, and regulations for the treatment of pedigreed seed stocks were established. The disease was reported sporadically during the early 1960's in western Ontario and although no organized surveys for the disease were conducted it was thought to be of minor importance. In 1967, a number of samples of pedigreed winter wheat seed from the Hensall, Ont., area adjacent to Lake Huron were found to be infested with T. controversa. Again in 1968 and in 1969 samples of pedigreed seed from the same area were found to be infested with T. controversa.

The work reported here was the result of a preliminary survey and loss assessment conducted in 1970 in the Hensall area and a more extensive survey and loss assessment conducted in 1971 in southwestern Ontario. Although dwarf bunt surveys were made previously, no disease-loss evaluations had been attempted (1). A summary of dwarf bunt incidence in samples of winter wheat seed over the period 1960-1971 is also presented.

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Methods

In 1970, 25 fields of winter wheat ranging from 10 to 20 acres were selected at random within a 5-mile radius of Hensall, Ontario. Hensall is located approximately 30 miles north of London. Each field was surveyed for the presence of dwarf bunt in the following manner. Initially each field was inspected until dwarf bunt was discovered or until 25% of the field was inspected. If dwarf bunt was found, the incidence of the disease was assessed by examining plants at 10 sites along a diagonal of the field. Each site consisted of a 30 ft length of row containing an average of approximately 750 heads. In this manner approximately 7,500 heads were examined per field.

In 1971, 53 fields were selected at random throughout the winter wheat growing areas of southwestern Ontario (Fig. 1). Survey and assessment procedures were similar to those used in 1970 except that when dwarf bunt was located, six sites along a diagonal of the field were assessed for dwarf bunt. Each site was 30 ft long and 4 rows wide. Approximately 3,000 heads were scanned at each site for a total of 18,000 heads per field. In Select plots and small fields the number of heads assessed at each site was reduced to 1,000.

Seed infestation

In both years, spores from infected heads were examined microscopically to confirm the presence of *T. controversa*. Forty grams of 0.5 lb sample of seed were shaken for 3 min in 40 ml sterile distilled water containing 0.1% Tween 20. Two 20 ml aliquots of the supernatant were centrifuged for 10 min. The supernatant was discarded, and the pellets were resuspended in 0.1 ml of Shear's fluid and examined under the microscope for the presence of *Tilletia controversa*.

Results and discussion

The survey conducted in the Hensall area in 1970 demonstrated that dwarf bunt was present throughout the area, but at a level insignificant from a crop loss standpoint. Although 14 of the 25 fields were affected, no field showed an infection higher than 1% of the crop (Table 1).

In the 1971 survey (Table 1), 16 of 53 fields were affected by dwarf bunt. The highest level of field infection was less than 0.5% and most fields contained only trace amounts of the disease. The distribution (Fig. 1) of the disease was primarily confined to the more northerly area between the towns of Clinton and Exeter in Huron County. No infected fields were found in Kent and Elgin counties and only two in the northern part of Middlesex County.

The distribution of dwarf bunt in Ontario indicates that environmental conditions play an important role in the survival of *T. controversa*. Dwarf bunt is present in areas of western Ontario that receive the most snow, and although contaminated seed has undoubtedly been sown in the noninfested areas over a period of years, the fungus has not survived in soils where snow cover is minimal.

Table 1. Prevalence of dwarf bunt in winter wheat fields in southwestern Ontario in 1970 and 1971

Infection category (% infected heads/field)	No. of fields in each category	
	1970	1971
0	11	37
0.013-0.053	7	13
0.066-0.133	3	1
0.146-0.267	2	1
0.280-0.666	1	1
0.680	1	0

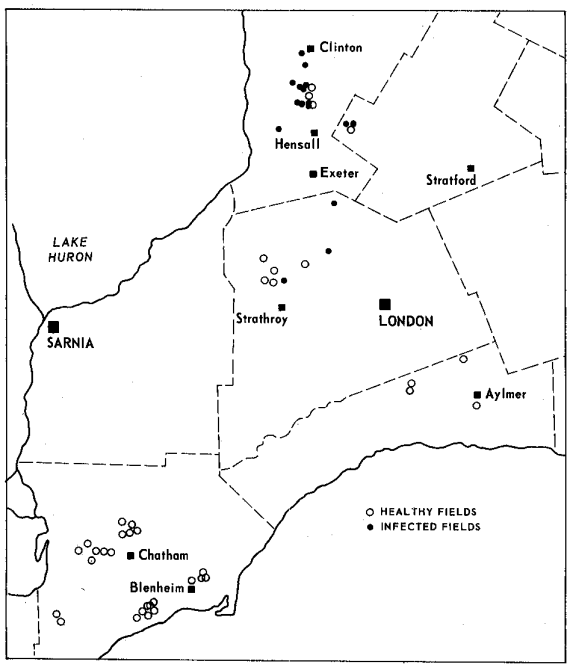

Figure 1. Incidence of dwarf bunt in fields of winter wheat in southwestern Ontario in 1971.

Table 2. Incidence of *Tilletia controversa* in samples of pedigreed winter wheat seed grown in Ontario, 1960- 1971

Year of production	Number of samples examined	Number of samples infested	% of samples infested
1960	12	12	100
1961	14	11	78
1962	12	4	33
1963	17	9	53
1964	21	5	24
1965	21	9	43
1966	39	6	15
1967	49	11	22
1968	36	11	31
1969	32	6	19
1970	21	8	38
1971	22	8	36

During the period 1960-71 pedigreed seed samples from southwestern Ontario were examined microscopically for spores of T. controversa. Although only a small number of samples were examined each year, the results (Table 2) show that dwarf bunt has been present predominantly in Huron County and to a lesser extent in western Perth and northern Middlesex counties during this period.

Dwarf bunt causes negligible losses to winter wheat crops at the present time. However the appearance of new races of the fungus could change the present situation, and the introduction of new winter wheat varieties with less resistance to dwarf bunt could also affect the severity of the disease. From this standpoint, it would be advantageous to screen breeding material, such as promising selections and varieties, on one or more of the farms where dwarf bunt is now indigenous in the soil.

As hexachlorabenzene seed treatment is effective in controlling seed-borne dwarf bunt (3), seed lots infested with the organism should be treated to prevent the spread of the disease to soils now free of the pathogen.

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SCREENING CRUCIFERS FOR GERMPLASM RESISTANCE TO CLUBROOT PLASMODIOPHORA BRASSICAE

Morgan S. Chiang and René Crête [1]

Abstract

A total of 334 varieties and lines of Brassica spp. were tested for resistance to Plasmodiophora brassicae race 6 under greenhouse conditions, and to races 6 and 2 under field conditions. Information on number of plants in each disease grade as well as disease indices from greenhouse and field tests for each entry are provided. Plant distribution in each disease grade is recommended as a guide in selecting germplasm resistance to clubroot rather than relying on disease indices alone. Some accessions and lines of cabbage showed a high degree of resistance to clubroot and are being used for breeding stocks.

Resumé

Un total de 334 lignées et variétés de Brassica spp. ont été soumises, en serre, à une infestation artificielle de la race 6 de Plasmodiophora brassicae et en plein champ, à une infestation naturelle des races 6 et 2 du même champignon dans le but de sélectionner du plasma germinatif résistant à la hernie. Ce plasma servira à des croisements ultérieurs de résistance.

Introduction

In 1967, the publication of a short paper (2) summarizing our results from screening tests of crucifers for resistance to clubroot proved to be useful in that many requests were received for the complete list of entries, including the distribution of disease grades for each entry, so that a more critical selection for developing resistant varieties could be effected. Since then more than 200 accessions, selections, lines, and varieties of crucifers were received from Plant Introduction Stations in the USA. In addition many breeding lines from institutions throughout the world were tested to select new clubroot resistant lines which could be used in our breeding program. The object of this report is to supply further information to those interested in searching for germplasm resistance to clubroot in crucifers, thus avoiding duplication or repetition of work.

Materials and methods

Greenhouse tests. In all greenhouse tests clubroot-free organic soil was infested with a suspension of resting spores of Plasmodiophora brassicae Wor. race 6. The technique of infestation was the same as described previously (2). Each plant was graded according to the four categories of disease severity used by Crête et al. (3).

Field tests. Field tests were conducted on mineral soil at L'Acadie, Que., and on well decomposed organic soil at Ste. Clotilde, Que. The soil at the L'Acadie farm was naturally infested with P. brassicae race 2 and possibly with race 3. At the Ste. Clotilde farm in the early part of the testing experiments race 6 was predominant with the presence of race 2; but gradually there seemed to be a shift in ratio between race 6 and race 2, with race 2 increasing in prevalence during the past few years. The procedures used in the field experiments were identical to those described elsewhere (2) except that in 1967 and thereafter grades of infection and disease indices were scored and calculated according to the method of Crête et al. (3) with the slight modification of increasing from four to five the grades of disease severity, as follows:

Grade	Disease severity (% of root system affected) 1966	1967-1971
0	0	0
1*	1- 29	1- 10
2	30- 59	11- 30
3	60-100	31- 60
4**		61-100

* Small nodules only.

** Partial to complete decay.

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Most of the Plant Introduction accessions and the commercial varieties were tested in 1966 using the four-grade classification, therefore for most entries column 4 in Table 1 is blank.

All materials tested and their origins are listed in Table 1.

Results and discussion

The distribution of test plants in four or five disease grades and the disease index of each entry from greenhouse and field tests are presented in Table 1.

Of the 334 entries, the following showed promise and are now used in our breeding program: PI 215513, PI 215514, PI 215515, PI 261643, PI 330389, 192264 A, 192261 G, 192264 B, 1922879 BC, 1922907 BC, 1922916 BC, Witte Kool, and 8-41. Entry 8-41 is a selection from 61-L-104, which was a hybrid of Wisc. 8351 x 1922-52-0, and is our most resistant line. It is immune to race 1, highly resistant to race 6, but susceptible to race 2 (1).

The disease indices obtained from greenhouse tests were, in general lower than those from field tests. This might be because in the field the roots remained in soil much longer (2 to 3 months) than in the greenhouse (45 days) and consequently more infected plants were decayed at the time of examination. Also under field conditions new physiological races of P. brassicae could have developed, possibly by mutation of the pathogen, particularly in the field of organic soil that has been planted with crucifers continuously for the past 15 years. This hypothesis is supported by the fact that the cabbage variety Badger Shipper was totally susceptible to the disease in this field in 1970 and 1971 trials; Badger Shipper is known for its resistance to race 6 (4, 5). However, with multiple races of the pathogen in the field, we are hopeful of selecting plant lines each with resistance to several races, and many plants have been selected under these conditions for further investigation.

One should keep in mind that the disease index of an entry is a weighted average of infection; therefore, a breeder should look at the frequency of plant distribution in each disease grade of the entry rather than depend only on the index. For example, PI 212971 from India had a disease index as high as 96% at the Ste. Clotilde Farm, and one may consider that this accession should be discarded. However by examining the plant distribution in the five grades, one finds that this entry might be as useful as an entry with a low disease index, because there were two plants showing small nodules only (grade 1); with further testing these two plants might be useful in developing a new resistant variety.

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Table 1. List of *Brassica* entries tested for resistance to clubroot, their scientific names and origin, number of plants in each disease grade and disease indices from greenhouse tests with race 6 of *Plasmodiophora brassicae* (figures in left hand column under each heading) and from field tests in mineral soil containing races 2 and 3 (figures underlined) and in organic soil containing races 6 and 2 (figures in parentheses)

Entry	Scientific name*	Origin	Disease grade 0	1	2	3	4	Disease indices
Plant introduction								
155061	1	Japan	1	0	0	0		0
156701	1	Japan	2 (0)	0 (0)	0 (0)	0 (1)	(10)	0 (98)
156702	1	Japan	1	0	0	1 (1)		50 (100)
163490	1	India	2	3	0	3		14
164275	1	India	15	0	1	6		30
164534	1	India	4	0	0	3		43
165054	1	Turkey	2	1	1	21		88
165067	1	Turkey	7	1	0	6 (6)		45 (100)
169039	1	Turkey	20	2	1	2		13
169040	1	Turkey	5	3	0	1 15 (29)		22 100 (100)
169041	1	Turkey	5	1	0	3 (9)		37 (100)
169046	1	Turkey	5	0	0	6		55
169047	1	Turkey	15	8	0	3 (24)		22 (100)
169048	1	Turkey	16	1	1	4 (43)		23 (100)
169049	1	Turkey	3	1	0	1		27
169051	1	Turkey	21	7	2	6 12 (41)		27 100 (100)
169052	1	Turkey	16	5	2	7 21 (40)		33 100 (100)
169053	1	Turkey	13	5	4	7 (10)		39 (100)
169055	1	Turkey	8	3	1	17 (8)		64 (100)
171523	1	Turkey	4	0	3	6 (9)		62 (100)
171529	1	Turkey	5	0	1	21		80
171530	1	Turkey	6	3	2	4 (9)		47 (100)
171531	1	Turkey	8	2	3	17 (9)		66 (100)
171532	1	Turkey	4	3	1	9 (8)		63 (100)
172743	1	Turkey	9	7	1	5 (8)		56 (100)
179188	1	Turkey	0	0	0	28		100
181720	1	Syria	4	0	1	6 (9)		61 (100)
181721	1	Lebanon	5	4	2	11		62
181722	1	Lebanon	6	6	2	18		65
182148	1	Turkey	8	3	5	12 (7)		58 (100)
182150	1	Turkey	2	3	2	6		64
194069	1	Germany	3	1	0	6 (7)		63 (100)
194070	1	Germany	2	0	2	9 (3)		80 (100)
194226	1	Germany	15	3	3	5 17 (23)		31 100 (100)
199787	1	Holland	18	4	1	8 11 (37)		32 100 (100)
204563	1	Turkey	7	4	4	5 (9)		45 (100)
204679	1	Turkey	8	7	3	5		41
211907	1	Afghanistan	8	4	1	14 (7)		59 (100)
212080	1	Afghanistan	0 (0)	2 (0)	1 (1)	4 (7)		86 (96)
212971	1	India	17 0 (0)	9 0 (2)	4 1 (0)	2 21 (31)		24 99 (96)
215513	1	Germany	23 2 (0)	2 6 (13)	1 0 (7)	1 3 (13)		9 45 (77)
215514	1	Germany	27 3 (0)	1 5 (9)	0 1 (4)	0 0 (4)		1 26 (57)
215515	1	Germany	36 14 (16)	1 15 (8)	0 0 (4)	0 29 (11)		1 17 (42)
222237	1	Iran	6	5	2	1		43
225855	1	Denmark	4	3	1	14		71
225856	1	Denmark	8	8	2	4		36
225858	1	Denmark	0	1	0	0		33
225860	1	Denmark	3	1	1	8		69
225861	1	Denmark	19	8	0	5		23
225862	1	Denmark	15	7	2	9 (9)		38 (100)
227012	1	Iran	4	4	4	8 (9)		60 (100)
227232	1	Iran	4	6	1	20 (3)		73 (100)
229470	1	Turkey	7	6	7	13 (4)		60 (100)
229747	1	Iran	9	1	1	14		60
230721	1	Holland	11	7	2	11 (6)		58 (100)
230722	1	Holland	9	5	4	9 (9)		49 (100)
232071	1	Holland	7	0	1	7		51
235041	1	Brazil	5	2	0	7 (7)		55 (100)
235042	1	Brazil	8	3	0	14 (6)		60 (100)
235043	1	Brazil	13	2	3	10		45
235044	1	Brazil	6	4	1	12		61
235045	1	Brazil	3	3	3	17		77
244986	1	England	7	6	3	11 (5)		56 (100)
244988	1	England	13	6	3	11		46
244990	1	England	2	3	3	16 (8)		79 (100)
244994	1	England	9 (0)	0 (0)	0 (1)	15 (10)		63 (97)
244996	1	England	1	2	1	15 (8)		86 (100)
244998	1	England	2	4	3	12		73
244999	1	England	5	6	3	21		71
245000	1	England	4	2	3	14		72
245001	1	France	1	0	0	2		67
245009	1	France	1	3	2	5		67
245010	1	France	10	6	0	8		42
245011	1	France	11	3	1	12 (9)		51 (100)
245012	1	France	10	3	1	15		58
245014	1	France	11	3	0	18		59
245015	1	France	0	2	1	7		83
245017	1	France	10	7	2	12 (9)		51 (100)
245018	1	France	1	3	0	5 (26)		67 (100)
245019	1	France	3	1	1	12 (8)		77 (100)
245020	1	France	9	11	1	3 11 (26)		31 100 (100)
245021	1	France	4	3	6	7 (9)		60 (100)

Entry	Scientific name*	Origin	Disease grade 0		1		2		3		4	Disease indices	
Plant introduction (cont'd)													
245022	1	France	9	(0)	4	(0)	1	(1)	5	(5)		37	(94)
245023	1	France	2		4		2		8	(7)		75	(100)
246046	1	Holland	14		4		1		10	(4)		41	(100)
246048	1	Holland	9		6		4		12			54	
246052	1	Holland	1		3		0		7			73	
246053	1	Holland	6		3		1		13	(8)		64	(100)
246054	1	Holland	10		2		0		8			48	
246055	1	Holland	6		2		2		10	(6)		60	(100)
246057	1	Holland	0	(0)	0	(0)	1	(3)	14	(13)		64	(94)
246063	1	Holland	13		3		2		9			42	
246064	1	Holland	10		1		1		8	(9)		45	(100)
246066	1	Holland	14		7		0		5			28	
246067	1	Holland	2		0		2		0			33	
246068	1	Holland	5		5		0		6			48	
246069	1	France	1		2		1		8			78	
246071	1	France	0		3		1		10			83	
246072	1	France	9		4		2		10			51	
246075	1	France	7		4		2		4			39	
246076	1	France	9		5		1		17			67	
246078	1	France	7		3		1		13			61	
246079	1	France	1		3		1		10	(7)		78	(100)
246080	1	France	3		4		2		18	(9)		77	(100)
246081	1	France	5		8		4		6			57	
246083	1	France	3	(1)	2	(0)	3	(0)	14	(7)		76	(88)
246084	1	Germany	0		4		0		10			81	
246085	1	Germany	1		2		1		14			85	
246087	1	Germany	5		5		1		15	(9)		67	(100)
246088	1	Germany	3	(0)	1	(0)	1	(1)	10	(4)		73	(93)
246090	1	Germany	13		1		2		0			10	
246093	1	Germany	7		4		1		8			50	
246094	1	Germany	12		5		3		12			49	
246096	1	Germany	3		2		1		12			74	
246098	1	Germany	8		12		5		7			45	
246100	1	Germany	10		3		1		19	(8)		63	(100)
246101	1	Germany	7		1		3		7	(9)		52	(100)
246102	1	Germany	16		6		2		8	(9)		35	(100)
246103	1	Germany	9		1		1		18	(7)		66	(100)
246104	1	Germany	2		2		1		5			63	
246109	1	Holland	9		1		2		12			57	
246110	1	Holland	18		4		0		2	13		14	100
246111	1	Holland	18		2		2		8	12 (30)		33	100 (100)
246113	1	Holland	3		4		5		16	(16)		74	(100)
246116	1	Holland	11		4		1		12	(6)		50	(100)
246117	1	Holland	13		7		4		8	(6)		41	(100)
246119	1	Holland	0		5		1		5	(3)		67	(100)
246565	1	England	12		7		2		9	(8)		42	(100)
246567	1	England	3		2		4		13			74	
246568	1	England	9		5		5		9			50	
249557	1	Thailand	6		3		1		8			54	
250419	1	Czechoslovakia	1		2		0		8			79	
250420	1	Czechoslovakia	2		5		1		8			65	
250421	1	Czechoslovakia	2		3		0		5			60	
250422	1	Czechoslovakia	4		3		1		5			51	
250423	1	Czechoslovakia	1		1		0		6			79	
250424	1	Czechoslovakia	13	(0)	7	(0)	2	(1)	5	(2)	(20)	32	(96)
255558	1	Yugoslavia	1		2		2		5			70	
255559	1	Yugoslavia	6		2		1		9			57	
255560	1	Yugoslavia	3		3		1		6			59	
255561	1	Yugoslavia	0		0		0		27			100	
255562	1	Yugoslavia	14	(0)	2	(0)	0	(1)	13	(1)	(45)	47	(98)
255581	1	Iran	3		1		0		3			48	
261600	1	Spain	14		2		0		18			77	
261601	1	Spain	12		8		2		18			55	
261602	1	Spain	3		4		0		11			69	
261603	1	Spain	0		5		2		24			77	
261604	1	Spain	8		5		3		5			41	
261605	1	Spain	8		8		0		13			54	
261642	1	Holland	25	(1)	7	(3)	1	(3)	2	(1)	(0)	14	(50)
261643	1	Holland	22	(4)	0	(12)	0	(4)	6	(6)	(6)	21	(45)
261757	1	Belgium	9	(0)	2	(0)	0	(2)	2	(1)	(36)	13	(97)
261758	1	Belgium	23		5		4		15		(48)	41	(100)
261769	1	France	22		11		0		11		(29)	33	(100)
261770	1	France	8		1		0		8		(34)	44	(100)
261771	1	France	16	(0)	3	(0)	0	(0)	2	(1)	(36)	14	(99)
261772	1	France	23		5		2		14			39	
261773	1	France	22		5		0		4		(42)	18	(100)
261774	1	France	5		3		3		9			60	
263058	1	USSR	12		4		1		5		(39)	32	(100)
263059	1	USSR	9		4		2		10			51	
263063	1	USSR	3		1		1		5			60	
263066	1	USSR	8		12		1		6			40	
263640	1	USSR	2		0		0		3			60	
263867	1	Greece	2		2		0		17			84	
269433	1	Pakistan	7		7		3		3			37	
273243	1	California	1		3		2		16			83	
274242	1	California	0		2		1		33			97	
275003	1	Bulgaria	0		1		1		17			95	

Entry	Scientific name*	Origin	Disease grade 0		1		2		3		4		Disease indices	
Plant introduction (cont'd)														
275004	1	Bulgaria	3	(0)	5	(0)	5	(0)	18	(1)	(16)		82	(99)
275005	1	Bulgaria	0		1		1		28				97	
275006	1	Bulgaria	0		0		0		34				100	
275007	1	Bulgaria	0		0		0		34				100	
277278	1	Bulgaria	1		0		1		27				95	
280065	1	Scotland	0		0		0		30				100	
280066	1	Scotland	0		1		0		19				97	
280067	1	Scotland	0		0		0		24				100	
280068	1	Scotland	0		1		1		35				97	
281547	1	Japan	0		1		0		31				98	
281548	1	Japan	1		2		1		32				93	
281549	1	Japan	2		1		2		14				82	
281550	1	Japan	0		1		0		34				98	
281551	1	Japan	0		0		0		30				100	
281552	1	Japan	0		0		0		35				100	
287535	1	Japan	0		1		0		26				98	
288229	1	Egypt	0		0		1		26				99	
288230	1	Egypt	0		1		0		25				97	
288231	1	Egypt	0		0		0		31				100	
289697	1	Australia	1		0		0		21				95	
294591	1	Denmark	0		2		0		33				96	
296130-A	1	USA	3		10		2		15				66	
296132	1	USA	0		0		0		34				100	
296133	1	USA	6		5		3		19				69	
296134	1	USA	1		1		0		3				67	
296135	1	USA	0		0		1		25				96	
296135	1	USA	0		1		1		25				96	
296136	1	USA	1		0		0		28				97	
296209	1	USA	16		6		0		9				35	
296330	1	S. Africa	9		2		0		14				59	
296331	1	S. Africa	8		6		1		6				41	
302442	1	Taiwan	5		6		0		16				67	
303629	1	S. Africa	11		9		0		14				50	
320069	1	India									19	(8)	100	(100)
320913	1	Hungary	8	(18)	5	(0)	5	(0)	4	(2)	29	(23)	70	(57)
320914	1	Hungary	12	(0)	10	(0)	6	(1)	3	(1)	18	(33)	53	(98)
329199	1	Holland	4		8		0		0		1	(9)	23	(100)
330389	1	Norway	8	(1)	0	(2)	4	(2)	4	(0)	11	(7)	59	(71)
330392	1	Norway	2	(0)	2	(0)		(1)		(1)	1	(4)	30	(88)
330393	1	Norway	7	(0)	1	(0)	0	(0)	0	(1)	0	(10)	3	(98)
343500	1	USSR	2		3		0		0		2	(8)	39	(100)
343515	1	USSR	1		0		1		2		2	(8)	67	(100)
343516	1	USSR	2		6		0		1		5		52	
343560	1	USSR ex. Bulg.	2		0		1		2		0	(10)	40	(100)
343569	1	USSR ex. Ger.	1		1		0		0		6	(14)	78	(100)
343619	1	USSR ex. India									5		100	
343622	1	USSR ex. Italy	1		1		0		1		1		50	
343625	1	USSR ex. Japan	0	(0)	0	(0)	1	(0)	0	(2)	5	(10)	92	(96)
343628	1	USSR ex. Japan	0	(0)	3	(0)	0	(0)	0	(1)	4	(10)	68	(98)
343630	1	USSR ex. Japan	3		4		0		1		3	(11)	43	(100)
343631	1	USSR ex. Japan	4		1		1		2		4	(15)	52	(100)
343633	1	USSR ex. Neth.	0		0		1		2		14	(19)	97	(100)
343634	1	USSR ex. Neth.	0		3		1		2		4	(6)	68	(100)
343635	1	USSR ex. Neth.	22		5		6		2		8		32	
343636	1	USSR ex. Neth.	1		0		0		0		7	(6)	88	(100)
343638	1	USSR ex. Neth.	0		0		0		0		7	(10)	100	(100)
343639	1	USSR ex. Neth.									11	(6)	100	(100)
206942	2	Turkey	8		0		2		20				71	
209751	2	Netherlands	0		2		0		0				33	
209756	2	Netherlands	2		2		1		2				47	
209757	2	Netherlands	2		0		1		2				53	
212592	2	Afghanistan	1		1		0		0	(5)			2	(100)
213209	2	Italy	2		1		1		9				77	
217517	2	Pakistan	5		5		0		14				65	
232070	2	S. Africa	8		4		2		12	(32)			56	(100)
277273	2	India	4		10		2		9	23 (42)			55	100 (100)
236257	3	Japan	1		0		0		1				50	
236258	3	Japan	1	0	1	0	0	1	0	7			17	96
236259	3	Japan	7	1	3	2	0	1	4	10			36	78
257229	5	Thailand	1		3		0		26				90	
273640	5	Ethiopia	2		0		0		24				92	
Plant breeder														
TK 499-2023	1	Norway	24		24		2		0		0	(47)	14	(100)
K 504-2024	1	Norway	22	(0)	26	(0)	0	(2)	0	(5)	0	(38)	14	(95)
K 512-2004	1	Norway	2	(0)	11	(0)	5	(0)	5	(8)	13	(36)	61	(96)
K 512-2028	1	Norway	16	(0)	27	(0)	5	(3)	0	(12)	0	(28)	19	(90)
K 512-2034	1	Norway	6	(0)	26	(0)	4	(0)	1	(15)	3	(41)	28	(97)
K 512-2041	1	Norway	4	(0)	31	(0)	3	(1)	0	(10)	0	(32)	24	(93)
TK 704-2006	1	Norway	0	(0)	5	(0)	4	(1)	16	(8)	21	(36)	79	(94)
K 707-2008	1	Norway	3	(0)	14	(0)	13	(2)	11	(3)	1	(41)	46	(96)
K 707-2044	1	Norway	12	(0)	17	(0)	2	(7)	0	(15)	0	(20)	17	(83)
K 707-2047	1	Norway	16	(0)	18	(0)	1	(1)	1	(3)	2	(44)	22	(97)
K 716-2013	1	Norway	14	(0)	26	(1)	2	(2)	1	(13)	0	(29)	19	(89)
K 716-2022	1	Norway	36	(0)	10	(1)	0	(2)	0	(6)	0	(37)	5	(93)

Entry	Scientific name*	Origin	Disease grade 0		1		2		3		4		Disease indices	
Plant breeder (cont'd)														
K 716-2026	1	Norway	17	(0)	24	(0)	3	(0)	0	(9)	1	(36)	19	(90)
K 732-2014	1	Norway	21	(0)	25	(2)	0	(0)	0	(3)	0	(41)	14	(95)
K 873-2018	1	Norway	38	(0)	0	(0)	0	(0)	0	(3)	0	(42)	0	(98)
K 873-2025	1	Norway	13	(0)	18	(0)	3	(6)	1	(5)	0	(34)	19	(91)
192261 G	1	Wisconsin	5	(0)	2	(10)	0	(1)	0	(16)			1	(85)
192262 L	1	Wisconsin	2		8	(27)	0	(2)	0	(0)			27	(36)
192264 A	1	Wisconsin	5	(1)	12	(21)	0	(5)	0	(1)			24	(40)
192265 B	1	Wisconsin	17	(2)	7	(23)	0	(6)	1	(5)		(1)	13	(37)
1922879 BC	1	Wisconsin	8	(1)	11	(8)	0	(1)	25	(3)		(28)	55	(99)
1922907 BC	1	Wisconsin	8	(2)	13	(13)	0	(6)	8	(4)		(29)	43	(55)
1922916 BC	1	Wisconsin	5		7		2		5				46	
Badger Shipper	1	Wisconsin	253	24 (0)	6	11 (1)	0	3 (1)	0	0 (4)	9	(55)	1	29 (96)
Badger Inbred #1	1	Wisconsin		(0)		(1)		(1)		(5)		(32)		(94)
Badger Inbred #7	1	Wisconsin		(0)		(0)		(2)		(1)		(31)		(96)
Badger Inbred #9	1	Wisconsin										(29)		(100)
Badger Inbred #10	1	Wisconsin		(0)		(1)		(3)		(6)		(24)		(89)
Badger Inbred #12	1	Wisconsin										(35)		(100)
Sanibel	1	Wisconsin		(21)		(2)		(1)		(1)		(36)		(62)
Junior	1	Morden, Man.	1		3		4		28			(32)	88	(100)
Little Leaguer	1	Morden, Man.	0		2		2		33			(35)	95	(100)
Pee Wee	1	Morden, Man.	0		1		3		31			(41)	95	(100)
Witte Kool	1	Holland	28	(1)	0	(14)	0	(10)	0	(8)		(10)	0	(57)
8-41 (61-L-104)	1	St. Jean, Que.	24	(31)	11	(18)	0	(0)	0	(0)			11	(12)
63-L-101	1	St. Jean, Que.	27	1	11	5	0	1	0	23			10	84
60-305	1	St. Jean, Que.	10		3		1		5	(41)			35	(100)
60-309	1	St. Jean, Que.	16		6		1		8				34	
60-331	1	St. Jean, Que.	5		4		0		5	(45)			45	(100)
60-332	1	St. Jean, Que.	9		3		1		12	(46)			54	(100)
Md. 8	1 X 2	Maryland		(0)		(4)		(6)		(17)				(78)
Md. 16	1 X 2	Maryland		(1)		(15)		(1)		(22)				(71)
Md. 17	1 X 2	Maryland		(1)		(6)		(2)		(21)				(81)
Md. 19	1 X 2	Maryland		(1)		(7)		(3)		(13)				(72)
Md. 21	1 X 2	Maryland		(3)		(4)		(4)		(26)				(89)
Wild Cabbage	5	England	1		3		3		2		1		48	
Commercial varieties														
Baby Head	1	Stokes	10		0		0		12			(42)	55	(100)
Badger Ballhead 14	1	Asgrow	3		1		0		4				70	
Badger Ballhead Y.R.	1	Asgrow	4		2		5		19				77	
Badger Ballhead Y.R.	1	Sem. Supér.†	9		1		1		7				44	
Badger Market	1	Letherman's	0		0		0		9				100	
Badger Market Y.R.	1	Asgrow	8		3		4		5				100	
Bonanza	1	Twilley's	29		1		0		1			(33)	4	(100)
Copenhagen Market	1	Sem. Supér.	4		1		0		2				33	
Copenhagen Market No. 86	1	Asgrow	6		3		0		7				50	
Danish Ballhead	1	Asgrow	16		0		2		18				52	
Danish Ballhead Tall Stemmed	1	Sem. Supér.	1		0		0		2				67	
Earlihead	1	Sem. Supér.	6		1		1		10				61	
Early Greenball	1	Stokes	7		0		1		13				65	
Earlygreen Ballhead	1	Stokes	12		4		1		1				46	
Early Jersey Wakefield	1	Vaughan's	0		0		1		26				99	
Early Marvel	1	Stokes	10		2		1		6				39	
Early Wonder	1	Sem. Supér.	0		0		0		3				100	
Extra Early 2	1	Sem. Supér.	1		3		0		6				70	
F_1 Hybrid Cabbage No. 18		Sakara	3		3		0		7				61	
F_1 Hybrid Cabbage No. 21	1	Sakara	12		2		1		11				47	
F_1 Hybrid Cabbage No. 26	1	Sakara	3		6		1		13				68	
Ferry's Round Dutch	1	Twilley's	35	(0)	2	(0)	0	(0)	0	(1)		(31)	2	(99)
Golden Acre Elite #3072	1	Sem. Supér.	1		0		0		3			(40)	75	(100)
Golden Acre Y.R.	1	Harris	6		3		1		17				69	
Golden Acre No. 84	1	Harris	0		0		1		10				97	
Greenback	1	Stokes	11		1		1		18				61	
Houston Evergreen	1	Stokes	10		3		1		14			(90)	56	(100)
Pennstate	1	Sem. Supér.	6		5		0		7	(35)			54	(100)
Red Acre	1	Sem. Supér.	5		0		0		2			(38)	29	(100)
Rest. Detroit Y.R.	1	Asgrow	7		0		1		14				67	
Savoy Cabbage Atlas	1	Sem. Supér.	8		2		1		11				56	
Savoy Cabbage Early	1		8		0		0		7				47	
Savoy Cabbage Wrener Kapuziner	1	Sem. Supér.	9		3		1		16				61	
Savoy Iron Head	1		5		2		1		8				58	
Viking Extra Early Strain	1	Stokes	8		1		1		13				61	
Wisconsin All Season Y.R.	1	Letherman's	1		1		0		20				92	
Wisconsin Ballhead Y.R.	1	Vaughan's	0		1		1		37				97	
Wisconsin Hollander Y.R.	1	Sem. Supér.	8		1		0		10				54	
Cottager	4	Sem. Supér.	8		2		1		2				26	
Dwarf Thousand-Headed	4	Sem. Supér.	4		4		0		8				58	
Green Asparagus Kale	4	Sem. Supér.	5		0		0		0				0	
Hungary Gap	4	Sem. Supér.	11	(30)	4	(0)	0	(0)	0	(4)			9	(12)
Marrow Stem Green	4	Sem. Supér.	9		1		1		3				29	
Rape Kale	4	Sem. Supér.	22		0		2		0				1	
Tall Green Kale	4	Sem. Supér.	1	(0)	0	(1)	0	(0)	0	(24)			0	(97)
Thousand-Headed	4	Sem. Supér.	2		1		0		2				47	
Variegated Kale	4	Sem. Supér.	10	(23)	3	(0)	0	(1)	3	(3)			1	(14)

* 1 = *B. oleracea* L. var. *capitata* L.
2 = *B. oleracea* L. var. *botrytis* L.
3 = *B. oleracea* L. var. *viridis* L.
4 = *B. oleracea* L. var. *acephala* DC.
5 = *Brassica* sp.

† Sem. Supér. = Semences Supérieures.

LOSSES FROM FOLIAGE DISEASES OF FORAGE CROPS IN CENTRAL AND NORTHERN ALBERTA IN 1971

B. Berkenkamp[1]

Abstract

An extensive survey of foliar diseases of alfalfa, clover (red, alsike, sweet, and white), brome, timothy, and fescue was made in central and northern Alberta in 1971, using methods similar to those followed in a 1970 survey. The estimated avg loss was 6.8% or $5.9 million in 1971.

Introduction

Until 1970, no extensive survey of forage crop diseases had been conducted in Canada with an objective of estimating disease losses. In order to estimate losses, it was essential to determine the species composition of the hay grown and the intensity of diseases on each species. A survey was conducted in central and northern Alberta in 1971, using the same methods as in 1970 (1) to allow comparison of disease prevalence and intensity from year to year.

Materials and methods

Fields in Census Divisions (C.D.) 8-15 in central and northern Alberta were surveyed between July 8 and August 24. C.D. 9 was not sampled because of limited cultivation of forage crops. Shoots of 4,850 forage plants from 315 fields were examined and the severity of diseases estimated. Methods of sampling, estimation of disease severity, and estimation of losses have been described (1). The Disease Index was based on percent area of the plant affected and was multiplied by 0.25 to give percent loss.

Results

Four diseases found in 1971 but not in 1970 were assessed as follows: pepper spot of red clover and sooty blotch of white clover were estimated as percentage of foliage area affected, gray stem canker of sweet clover as percentage of stem area affected, and whitehead (silver top) of brome as percentage of heads killed. One disease reported in 1970, downy mildew of sweet clover, was not found in 1971.

The intensity and distribution of diseases on the various species are shown in Table 1. The number of fields affected and the disease index, which is an average of all fields sampled, are shown by C.D. The number of fields sampled and the estimated acreage in each C.D. is indicative of the prevalence of each forage species.

The largest increases in disease intensity over 1970 were found in three Census Divisions: C.D. 11, primarily on red and alsike clovers; C.D. 14, in which a limited number of samples were collected; and C.D. 15, which experienced a very wet season and high disease levels in most species.

A comparison of the percentage loss for each disease and species in 1970 and 1971 for the entire area surveyed is shown in Table 2. Some of the diseases remained relatively constant, especially those of brome and timothy, but most showed an increase. Losses from Stagonospora of red and alsike clovers were greatly increased, as were losses from some other diseases of clovers. A tenfold increase was found in diseases of fescue, probably due to a very wet year in the fescue area (southern C.D. 15). Some areas in C.D. 15 were also found showing flooding injury.

Losses by C.D. and total for the area surveyed are shown in Table 3. Data for yield, production, and farm value of forage was supplied by the Alberta Marketing and Statistics Branch (H.H. Bryce, personal communication). The estimated average loss was up from 5.65% in 1970 (1) to 6.8% in 1971.

Forage yields in 1971 were estimated at 1.81 tons/acre compared with 1.85 tons/acre in 1970, and the total loss was 369.3 and 258.3 thousand tons for 1971 and 1970 respectively. Acreage was up 117 thousand in 1971, and although the value of hay decreased from $18.00 to $16.00 per ton, the estimated disease loss value increased from $4.66 to $5.91 million.

Literature cited

1. Berkenkamp, B. 1971. Losses from foliage diseases of forage crops in central and northern Alberta in 1970. Can. Plant Dis. Surv. 51:96-100.

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Table 1. Incidence and severity of foliage diseases of forage crops in central and northern Alberta, 1971

1. ALFALFA (*Medicago sativa* L.)

Census Division	Acres grown ('000)	No. fields sampled	Diseases* assessed** Yellow leaf blotch	Black stem	Stagon-ospora	Pepper spot	Downy mildew	Common leaf spot
8	141.0	25	23/ 9.03	18/2.63	13/0.23	2/0.28	4/0.23	22/ 3.57
10	133.4	38	32/ 8.41	33/1.84	12/0.11	8/0.71	2/0.02	32/ 3.34
11	237.6	37	37/14.38	31/2.28	17/0.19	7/0.64	0/0	34/ 8.62
12	130.9	22	11/ 3.17	14/1.86	17/1.50	0/0	1/0.01	22/11.15
13	120.5	21	18/11.10	19/5.84	12/0.54	1/0	0/0	20/19.90
14	14.0	3	3/13.97	3/4.47	0/0	1/0.33	0/0	1/ 1.50
15	158.5	16	15/24.89	15/8.69	3/0.02	1/0.03	0/0	13/10.99
Total	935.9	162	139/11.24	133/3.31	74/0.38	20/0.36	7/0.04	144/ 8.51

* Causal fungi: Yellow leaf blotch, *Leptotrochila medicaginis* (Fckl.) Schuepp; black stem, *Ascochyta imperfecta* Pk.; stagonospora, *Leptosphaeria pratensis* Sacc. and Briard; pepper spot, *Pseudoplea trifolii* (Rostr.) Petr.; downy mildew, *Peronospora trifoliorum* de Bary; common leaf spot, *Pseudopeziza trifolii* f. sp. *medicaginis-sativae* Schmiedeknecht.

2. RED CLOVER (*Trifolium pratense* L.)

Census Division	Acres grown ('000)	No. fields sampled	Diseases* assessed** Powdery mildew	Northern anthracnose	Black stem	Black-stem leaf spot	Stagon-ospora	Pepper spot
8	72.6	15	2/0.37	9/ 4.79	9/1.73	0/0	14/20.16	0/0
10	2.2	1	0/0	1/ 0.10	1/0.90	0/0	0/0	0/0
11	58.9	11	6/9.70	6/11.66	8/3.05	2/ 0.85	11/29.13	2/1.93
12	17.6	5	2/8.22	4/ 8.12	5/1.28	0/0	5/46.68	0/0
13	61.8	12	0/0	9/10.31	10/1.35	2/11.42	10/39.97	1/0.26
14	8.0	2	0/0	2/28.45	2/3.80	0/0	2/26.90	0/0
15	133.8	14	3/6.25	8/15.06	13/4.61	0/0	13/41.92	0/0
Total	354.9	60	13/4.01	39/10.54	48/2.59	4/ 2.44	55/32.94	3/0.40

* Causal fungi: Powdery mildew, *Erysiphe polygoni* DC. ex Mérat; northern anthracnose, *Kabatiella caulivora* (Kirchn.) Karak.; black stem, *Ascochyta meliloti* (Trel.) Davis; stagonospora, *Stagonospora recedens* (O. Massal.) Jones and Weimer; pepper spot, *Pseudoplea trifolii* (Rostr.) Petr.

3. ALSIKE CLOVER (*Trifolium hybridum* L.)

Census Division	Acres grown ('000)	No. fields sampled	Diseases* assessed** Powdery mildew	Black stem	Stagon-ospora	Pepper spot	Rust	Sooty blotch
8	63.1	18	4/ 7.22	5/0.09	17/23.49	4/1.78	0/0	6/4.72
10	1.2	1	1/18.30	0/0	1/31.70	0/0	0/0	0/0
11	72.9	16	8/24.78	4/0.44	16/33.51	1/1.64	2/0.38	2/3.85
12	6.3	1	0/0	1/0.70	1/72.60	0/0	0/0	0/0
13	65.0	15	5/ 8.20	7/1.05	15/27.46	1/1.80	0/0	6/2.61
14	10.0	3	0/0	1/0.37	3/56.43	0/0	0/0	0/0
15	102.9	15	8/15.15	3/0.10	15/34.21	1/1.44	0/0	0/0
Total	321.4	69	26/12.97	21/0.40	68/31.27	7/1.55	2/0.09	14/2.69

* Causal fungi: Powdery mildew, *Erysiphe polygoni* DC. ex Mérat; black stem, *Ascochyta melitoti* (Trel.) Davis; stagonospora, *Leptosphaeria pratensis* Sacc. and Briard; pepper spot, *Pseudoplea trifolii* (Rostr.) Petr.; rust, *Uromyces trifolii* (Hedw. f. ex DC.) Lév.; sooty blotch, *Cymadothea trifolii* (Pers. ex Fr.) Wolf.

** Number of fields affected/disease index.

Table 1 (Cont'd.)

4. SWEET CLOVER (*Melilotus alba* and *M. officinalis* L.)

C.D.	Acres grown ('000)	No. fields sampled	Diseases* assessed** Black stem	Downy mildew	Stagonospora	Gray stem canker
8	1.6	2	0/0	0/0	2/0.11	0/0
10	8.4	3	2/0.33	0/0	3/1.17	0/0.10
11	0	0	0/0	0/0	0/0	0/0
12	12.7	2	1/0.45	0/0	2/6.10	0/0
13	7.6	1	0/0	0/0	0/0	0/0
14	0	0	0/0	0/0	0/0	0/0
15	29.5	3	2/0.10	0/0	2/0.77	0/0
Total	59.8	11	5/0.20	0/0	9/1.66	1/0.03

* Causal fungi: Black stem, *Ascochyta meliloti* (Trel.) Davis; downy mildew, *Peronospora trifoliorum* de Bary; stagonospora, *Leptosphaeria pratensis* Sacc. and Briard; gray stem canker, *Ascochyta caulicola* Laub.

5. WHITE CLOVER (*Trifolium repens* L.)

C.D.	Acres grown ('000)	No. fields sampled	Diseases* assessed** Pepper spot	Stagonospora	Rust	Sooty blotch
8	2.5	2	1/30.0	1/1.0	2/ 9.0	1/ 8.0
10	0	0	0/0	0/0	0/0	0/0
11	0	0	0/0	0/0	0/0	0/0
12	0	0	0/0	0/0	0/0	0/0
13	9.6	2	1/ 3.0	2/9.0	1/27.0	1/24.0
14	0	0	0/0	0/0	0/0	0/0
15	0	0	0/0	0/0	0/0	0/0
Total	12.1	4	2/16.5	3/5.0	3/18.0	2/16.0

* Causal fungi: Pepper spot, *Pseudoplea trifolii* (Rostr.) Petr.; stagonospora, *Leptosphaeria pratensis* Sacc. and Briard; rust, *Uromyces trifolii* (Hedw. f. ex DC.) Lév.; sooty blotch, *Cymadothea trifolii* (Pers. ex Fr.) Wolf.

6. BROME (*Bromus inermis* Leyss.)

C.D.	Acres grown ('000)	No. fields sampled	Diseases* assessed** Brown leaf spot	Selenophoma	Scald	Whitehead
8	19.7	8	8/ 5.60	1/0.01	0/0	1/1.25
10	86.9	26	26/ 8.01	18/0.56	3/0.05	1/0.38
11	38.7	12	10/ 6.97	3/0.07	2/0.02	1/0.83
12	16.5	3	3/ 9.83	2/0.73	1/0.03	0/0
13	69.8	15	15/10.86	5/0.91	5/0.11	1/0.67
14	12.8	2	2/ 8.65	0/0	0/0	0/0
15	110.4	12	12/ 8.21	8/0.55	7/0.33	0/0
Total	354.8	78	76/ 8.27	37/0.49	18/0.09	4/0.51

* Causal fungi: Brown leaf spot, *Drechslera bromi* (Died.) Shoem.; selenophoma, *Selenophoma bromigena* (Sacc.) Sprague and Johnson; scald, *Rhynchosporium secalis* (Oud.) J.J. Davis.; whitehead, *Fusarium poae* (Pk.) Wr.

7. TIMOTHY (*Phleum pratense* L.)

C.D.	Acres grown ('000)	No. fields sampled	Diseases* assessed** Eyespot	Leaf streak
8	109.5	33	32/0.39	33/ 3.60
10	5.8	2	1/0.02	2/ 2.10
11	36.4	15	15/0.29	15/ 6.21
12	5.7	2	2/0.35	2/21.00
13	63.0	19	19/0.66	19/10.37
14	19.2	4	4/0.29	4/ 3.92
15	54.9	9	9/0.23	9/ 5.77
Total	294.5	84	82/0.40	84/ 6.22

* Causal fungi: Eyespot, *Heterosporium phlei* Gregory; leaf streak, *Drechslera phlei* (Graham) Shoem.

8. FESCUE (*Festuca rubra* L.)

Census Division	Acres grown ('000)	No. fields sampled	Diseases* assessed** Brown stripe	Stem eyespot
8	0	0	0/0	0/0
10	1.0	1	1/ 5.00	0/0
11	3.1	1	1/ 5.00	0/0
12	0	0	0/0	0/0
13	0	0	0/0	0/0
14	0	0	0/0	0/0
15	80.3	9	9/28.36	9/36.29
Total	84.4	11	11/24.11	9/29.69

* Causal fungi: Brown stripe, *Passalora graminis* (Fckl.) Höhn; stem eyespot, *Phleospora idahoensis* Sprague.

** Number of fields affected/disease index.

Table 2. Percentage losses from diseases of forage crops in central and northern Alberta, 1970-71

Forage species	% of forage crops grown 1970	1971	Disease	Causal fungus	Loss (%) 1970	1971
Alfalfa (*Medicago sativa*)	45.5	38.3	Yellow leaf blotch	*Leptotrochila medicaginis*	2.87	2.81
			Black stem	*Ascochyta imperfecta*	1.45	0.83
			Stagonospora	*Leptosphaeria pratensis*	0.11	0.10
			Pepper spot	*Pseudoplea trifolii*	0.06	0.09
			Downy mildew	*Peronospora trifoliorum*	0.02	0.01
			Common leaf spot	*Pseudopeziza trifolii* f. sp. *medicaginis-sativae*	1.30	2.13
				TOTAL	5.81	5.97
Red Clover (*Trifolium pratense*)	16.5	14.5	Powdery mildew	*Erysiphe polygoni*	3.04	1.00
			Northern anthracnose	*Kabatiella caulivora*	1.11	2.63
			Black stem	*Ascochyta meliloti*	0.64	0.65
			Stagonospora	*Stagonospora recedens*	2.17	8.23
			Black stem leaf spot	*Ascochyta meliloti*	0.32	0.61
			Pepper spot	*Pseudoplea trifolii*		0.10
				TOTAL	7.28	13.22
Alsike clover (*Trifolium hybridum*)	8.7	13.1	Powdery mildew	*Erysiphe polygoni*	3.54	3.24
			Black stem	*Ascochyta meliloti*	0.18	0.10
			Stagonospora	*Leptosphaeria pratensis*	2.83	7.82
			Pepper spot	*Pseudoplea trifolii*	0.06	0.39
			Rust	*Uromyces trifolii*	0.24	0.02
			Sooty blotch	*Cymadothea trifolii*	0.18	0.67
				TOTAL	7.03	12.24
Sweet clover (*Melilotus alba* and *M. officinalis*)	3.0	2.4	Black stem	*Ascochyta meliloti*	0.28	0.05
			Downy mildew	*Peronospora trifoliorum*	0.01	
			Stagonospora	*Leptosphaeria pratensis*	0.06	0.41
			Grey stem canker	*Ascochyta caulicola*		0.01
				TOTAL	0.35	0.47
White clover (*Trifolium repens*)	0.2	0.5	Pepper spot	*Pseudoplea trifolii*		4.12
			Stagonospora	*Leptosphaeria pratensis*	0.01	1.25
			Rust	*Uromyces trifolii*	0.01	4.50
			Sooty blotch	*Cymadothea trifolii*		4.00
				TOTAL	0.02	13.87

Table 2 (Cont'd.)

Forage species	% of forage crops grown 1970	1971	Disease	Causal fungus	Loss (%) 1970	1971
Brome (*Bromus inermis*)	11.0	14.5	Brown leaf blotch	*Drechslera bromi*	2.56	2.07
			Selenophoma leaf spot	*Selenophoma bromigena*	0.02	0.12
			Scald	*Rhynchosporium secalis*	0.02	0.02
			Whitehead	*Fusarium poae*		0.13
				TOTAL	2.60	2.34
Timothy (*Phleum pratense*)	9.5	12.0	Purple spot	*Heterosporium phlei*	0.11	0.10
			Leaf streak	*Drechslera phlei*	1.46	1.55
				TOTAL	1.57	1.65
Fescue (*Festuca rubra*)	3.2	3.4	Brown stripe	*Passalora graminis*	0.24	6.03
			Stem eyespot	*Phleospora idahoensis*	0.91	7.42
				TOTAL	1.15	13.45
Other	2.3	1.1				

Table 3. Losses from foliage diseases of forage crops in Alberta Census Divisions 8 to 15, 1971

Census Division	No. of fields sampled	Acreage of forage crops ('000)	Yield (tons/acre)	Loss (%)	Actual production ('000 tons)	Potential production ('000 tons)	Loss ('000 tons)	Loss* ($'000)
8	47	410	1.91	4.55	783.1	820.4	37.3	596.8
10	54	240	1.60	2.59	384.0	394.2	10.2	163.2
11	61	450	1.91	8.72	859.5	941.6	82.1	1313.6
12	31	196	1.80	5.45	352.8	373.1	20.3	324.8
13	53	399	1.80	7.97	718.2	780.4	62.2	995.2
14	8	64	1.80	5.77	115.2	122.3	7.1	113.6
15	61	686	1.86	10.52	1276.0	1426.1	150.1	2401.6
Total	315	2445	1.81	6.80	4488.8	4858.1	369.3	5908.8

* Based on a farm value of $16 per ton of forage.

OCCURRENCE IN WESTERN CANADA OF COLLECTIONS OF LOOSE SMUT, USTILAGO AVENAE, VIRULENT ON OAT VARIETIES WITH RESISTANCE FROM VICTORIA[1]

J. Nielsen

Many widely grown Canadian varieties of oats have been resistant to loose smut and covered smut caused by Ustilago avenae (Pers.) Rostr. and U. kolleri Wille. Resistance in these varieties was derived from the variety Victoria (C.I. 2401), which comes from Argentina and was first used in the 1920s because of its resistance to rust. Later it was realized that it also carried resistance to all known races of the loose and covered smut fungi, and for decades Victoria and its derivatives were used as sources of resistance to smut in the breeding of improved oats. This resistance is based on one or two dominant genes (Murphy and Coffman 1961; Cherewick and McKenzie 1969).

In 1942 Reed and Stanton reported the first collection of loose smut with virulence on Victoria. By 1966 this virulence had been found in 15 states of the U.S.A., and races of this type predominated in the South Central and South Atlantic states (Holton 1967a).

Another common source of resistance to smut was the variety Clinton (C.I. 3971). Again, extensive use over a wide area screened the population of the parasite effectively for virulence on Clinton. Finally races of loose smut were found in the North Central region of the U.S.A. that are virulent on both Victoria and Clinton (Holton 1967b).

With the gradual spread of races of these types in the U.S.A. it was apparent that it would be only a matter of time until the natural spread of inoculum or the importation of infected seed would bring them to Canada. Annual surveys in Manitoba and eastern Saskatchewan and virulence tests of field collections did not reveal virulence on Victoria or Clinton until 1969. In 1970, 225 fields of oats were inspected (McDonald et al. 1971) and 6 fields were found to have a trace infection of smut, one of covered smut and five of loose smut. Spores collected from these fields were used to inoculate by the standard partial vacuum method seed of the variety Kelsey, which has the Victoria resistance. Three of the collections, all of loose smut, were found to infect Kelsey; percentage infection of Kelsey for the three samples was 48, 63, and 76%.

Spores of collection 04-70 (originally collected at Buchanan, Sask.) were taken from Kelsey and used to inoculate the standard differential varieties (numbers 1-10, Holton and Murphy 1966), supplemented with three varieties (numbers 11-13) that had been added by Cherewick (1958) and with the variety Markton (number 14). In this set of differentials Atlantic possesses the Victoria resistance, and Clintland possesses the Clinton resistance. The reaction of these differentials to collection 04-70 is presented in Table 1.

In 1971 trace amounts of loose smut were detected in 4 of 123 fields of oats surveyed in Manitoba (Hagborg et al., in preparation). Spores collected from the four fields were tested for virulence on Kelsey. One of the collections, 02-71, had a low level of virulence with 2% infection. This type of virulence has been found previously in Western Canada (Cherewick 1958, and unpublished data). The other three collections were highly virulent on Kelsey with 81, 84, and 72% infection. Spores from Kelsey of collection 04-71 (originally collected at Binscarth, Man.) were used to inoculate the differential varieties. The pattern of virulence was found to be identical with that of the 1970 collection 04-70 (Table 1).

Table 1. Reaction of differential varieties to two collections of loose smut

	Differential variety	% infection after inoculation with collection: 04-70	04-71
1	Anthony	81	92
2	Black Diamond	0	4
3	Victory	63	56
4	Gothland	0	0
5	Monarch	0	0
6	Camas	0	0
7	Black Mesdag	0	0
8	Atlantic	73	80
9	Fulghum	0	5
10	Clintland	74	24
11	Nicol	0	0
12	Beacon	79	52
13	Mabel	0	0
14	Markton	0	0

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The origin of this race with Victoria and Clinton virulence is unknown since its pattern of virulence on the differentials has not been reported before. A relationship with the race that was accidentally introduced at the Experimental Farm at Regina, Sask., in 1967 with a sample of Lodi oats from Wisconsin has to be ruled out. That race was virulent on 12 of the differential varieties (Cherewick and McKenzie 1969, and unpublished data), whereas the present race is virulent on only five. Natural hybridization could not have brought about such a discrepancy within this short time.

At present, provincial recommendations of varieties of oats for Manitoba and Saskatchewan list varieties with Victoria resistance (Fraser, Garry, Harmon, Kelsey, Rodney, Russell, and Sioux) as "resistant" to smuts. If the level and incidence of infection in fields increase these recommendations will have to be changed and periodic seed treatment of these varieties recommended. A prediction that this will in fact happen would be rather uncertain at the present time. Under similar circumstances, it was predicted that there would be an increase in area and intensity of infection by a race of loose smut of wheat with virulence on Thatcher and its derivatives (Nielsen 1969). Surveys of the last 3 years confirmed that the area in which this race occurs did in fact increase, but the level of infection is still only a trace. This example illustrates that tests of the reaction of varieties to smut by artificial inoculation will reveal the maximum infection physiologically possible. It does not take into account any field resistance by morphological characteristics nor the influence of climatic conditions on host or parasite. In the present case, it is interesting to note the prevalence of Victoria virulence in the South Central and South Atlantic regions of the U.S.A. (Holton 1967a). Holton explains this by the predominance there of varieties with Victoria resistance. It may well be, however, that some genotypes of the parasite may be better adapted than others to certain environmental conditions.

Considering the possible spread of virulence on Victoria and Clinton in Canada the breeder will ask what to use in future as a source of resistance to the smuts. The list published by Holton and Murphy (1966) shows that "there is ample potential for the development of smut resistant varieties for all areas of adaptation." Varieties with Markton background appear particularly promising.

The author would very much appreciate receiving field collections of smuts of oats to keep a continued vigilance for new, potentially threatening genes for virulence.

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SQUASH MOSAIC VIRUS IN MUSKMELON SEED DISTRIBUTED COMMERCIALLY IN ONTARIO

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Abstract

The occurrence of a virus similar to the ringspot strain of squash mosaic virus is reported in muskmelon (Cucumis melo L.). The virus was found in seedlings grown in the greenhouse from seed of the cultivar Iroquois obtained commercially in Canada. Evidence of transmission of the virus through the roots of neighboring plants was obtained in a greenhouse test.

Introduction

In May 1971 a pronounced mosaic symptom was observed in muskmelon (Cucumis melo L. cv. Iroquois) seedlings that had reached the first true-leaf stage of development. The plants originated from California seed obtained from a commercial seed company in Ontario and planted 28 April 1971 in 3-inch peat pots containing sterilized sandy loam at the Horticultural Research Institute of Ontario, Vineland Station, Ontario. Three weeks after seeding, mosaic symptoms and distortion similar to hormonal injury were noticed on the first true leaves of some seedlings but not on the cotyledons. Many of the affected plants were stunted (Figure 1). A count of the plants from this seed lot indicated that 38 of 480 (7.9%) were affected. The absence of insects on any of these plants and the relatively high percentage of diseased seedlings suggested that the condition was due to a seed-borne virus. Since there has been no report of the seed transmission of viruses cucurbits in Canada (1, 6), an investigation was initiated to identify the causal agent and to verify its seed transmission.

Figure 1. Healthy seedling of 'Iroquois' muskmelon (right) and one infected with squash mosaic virus (left). Note the pronounced chlorotic mottle on the upper leaf and the extreme distortion and narrowing of the lower ones.

Experimental and discussion

The reactions of Vigna sinensis Savi (cowpea cv. Black-eye), Nicotiana glutinosa L., Citrullis vulgaris Schrad. (watermelon cv. Market Midget), and Cucurbita pepo L. (pumpkin cv. Small Sugar) after inoculation with sap from infected muskmelon indicated that the virus was similar to the ringspot strain of squash mosaic virus (4). No reaction occurred on cowpea or N. glutinosa, but local lesions appeared on the cotyledons of watermelon and a systemic ringspot and mottle on pumpkin. In our tests, sap extracted from infected muskmelon retained infectivity after 10 min at 60° C but not at 65° C. These results also agree with those reported for squash mosaic virus (3), which has been reported to be seed-transmitted in California (5).

To assure that the seed, and not accidental contamination of the seedlings, was responsible for the infection, seed of the same cultivar was again obtained from the same supplier. Two lots of 200 seeds were

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selected at random from the source material, one was treated with 10% trisodium phosphate for 15 min while the other was untreated. Both lots were sown in sterilized sandy loam (10 seeds per 5 inch clay pot) and placed in a small, screened greenhouse compartment. To further guard against insect infestation, each pot was covered with a clean polyethylene bag which was removed only after the cotyledons and first true leaves had expanded. Of 192 seedlings that emerged after 3 weeks from untreated seed, 11.9% were virus-infected; whereas 6.7% of 177 seedlings from treated seed shown virus symptoms. These results suggest that the virus was within the seed since it was not eliminated by the treatment with trisodium phosphate, a chemical reported to eliminate tobacco mosaic virus from tomato seed coats but not from its endosperm (8).

Circumstantial evidence for what might be assumed to be mechanical inoculation by root contact or natural root grafting was also obtained. Thirty peat pots, each containing one infected and one apparently healthy seedling at the first leaf stage, were divided into three groups. In one group, the infected seedlings were decapitated with scissors at the soil surface. Prior to decapitation no contact occurred between infected and symptomless seedlings because of adequate spacing. In the second group, the infected seedlings were completely enclosed in a polyethylene bag to prevent contact with the stem and leaves of the neighboring healthy plant. In the remaining group, the infected plants were untouched. A control group of 10 pots in which both seedlings were apparently healthy was maintained under the same greenhouse environment. Six weeks after seeding all the plants in this latter group remained healthy. However, 6 of the 10 originally symptomless seedlings in pots with decapitated infected plants became infected. Of the 10 plants grown adjacent to bagged, infected plants 7 showed virus symptoms. In the pots where healthy plants were not prevented from contacting infected plants, 9 of the 10 seedlings became infected. These results could be attributed mainly to transmission by root contact.

The significance of seed-borne infection and transmission by root contact in outbreaks of squash mosaic virus in commercial muskmelon plantings in Ontario is not known. However, seed transmission could facilitate the initial introduction of the virus. The subsequent spread of the virus would depend mainly on the presence of Diabrotica beetles and the grasshopper Melanoplus differentalis, the reported vectors (2,7). But, as indicated above, transmission by root contact may also be important. In areas of California where cucumber beetles occur (2), squash mosaic virus is prevalent in commercial muskmelons and other cucurbits and persists in a relatively high percentage of the seed. Therefore, seed imported from these areas could serve as a primary source of inoculum for subsequent infection of muskmelon and other cucurbits in Canada.

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SEED PRODUCTION IN THE VIRUS INDICATOR PLANT SCOPOLIA SINENSIS

R.E. Hanneman, Jr. and R.P. Singh[1]

Abstract

A simple and effective means of producing seed of Scopolia sinensis Hemsl., a local lesion host of potato spindle tuber metavirus, is described. Pollinations made the day before or the day of flower opening resulted in good fruit and seed set, while pollinations made at the time of anthesis resulted in poor fruit set and almost no seed set. Pollen viability declined rapidly with storage. Freshly harvested seed germinated well. No natural seed dormancy was observed.

Introduction

Previous reports (4,5) from this laboratory have shown that several species of Scopolia are indicator plants for potato spindle tuber metavirus[2] (PSTM). Scopolia sinensis Hemsl. was shown to develop local lesions with both mild and severe strains of PSTM (5) and appeared to be a suitable host for quantitative assay during purification studies (6).

The need for a reliable means of seed production from S. sinensis has been made evident by the demand for this plant as an indicator for PSTM. Workers desiring to employ this plant have found difficulty in propagating it vegetatively and have often expressed a desire for seed.

In our work, a collection of S. sinensis seed obtained from the Netherlands was the only seed which was found to be suitable for the indexing of PSTM. Seed of this particular lot was not available in sufficient quantities to permit continuous use of seedlings; therefore, the plants had to be multiplied by cuttings. Attempts to produce seed in the greenhouse failed and only a few seeds were obtained from plants grown in the field.

A survey of literature revealed that Scopolia species typically have low fertility because of several inherent abnormalities in the male and female gametophytes (1,2) or because of an early shedding of flowers (3). Modification of environmental conditions (3) or use of growth stimulants (2,3) failed to increase flower retention and thus seed production has been erratic and unpredictable.

Here we report a simple procedure which lends itself readily to the production of seed in the greenhouse.

Materials and methods

Vigorous plants of S. sinensis which had previously been multiplied vegetatively were selected for use in seed production. They were grown in a greenhouse during the winter and were subjected to an 18-hr day with supplemental fluorescent lighting of 600-700 ft-c. Temperatures were maintained at 21-24° C. These conditions promoted flower initiation.

Pollination was done at the time of anthesis or 1 to 3 days prior to anthesis. Flowers open approximately 1 day before anthesis. When buds were used, the upper portion of the corolla was removed to expose the stigma. No emasculation was done. Pollination was accomplished by removing a dehiscent anther from a flower of the same or preferably another plant and rubbing it repeatedly across the stigmatic surface until the stigma was well covered with pollen. Pollen was applied only once. Each pollinated flower was tagged and dated.

The viability of stored pollen was investigated. Dehiscent anthers were collected in small gelatin capsules and placed in sealed containers with anhydrous calcium chloride as a drying agent. These containers were placed in a refrigerator (4°C). Pollen viability was judged by stainability with acetomcarmine and by fruit set.

Fruits were harvested 6-8 weeks after pollination. A sample of seed was used for germination tests immediately after harvest.

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[2] In view of the low-molecular weight nature of the spindle tuber agent and its other properties (reference 6), Singh and Clark have proposed the term "metavirus" to describe the infectious agent of spindle tuber disease. The details of the proposal will appear elsewhere.

Results

Table 1 summarizes the data collected on fruit and seed set in flowers pollinated before and at anthesis. Fruit set was most successful in flowers pollinated the day before or the day of opening (pre-anthesis). Fruits from pre-anthesis pollinations were larger and contained more seeds than those from anthesis pollinations. The flowers pollinated at anthesis set only a few fruits, most of which were seedless.

It was noted from the examination of ovaries from unsuccessful pollinations that often the only ovules found to be developing from anthesis pollinations were located in the upper portion of the ovary, while those developing from pre-anthesis pollinations occurred over the entire placental surface. In an attempt to enhance the success of fertilization, styles of anthesis stage flowers were cut in half and pollinated on the cut surface; however, developing ovules were still found primarily in the upper portion of the ovaries. The "cut style" approach was found to be ineffective.

Stored pollen lost its viability very rapidly. Stainability fell from more than 90% on the first day to about 70% by the third day of storage. Pollen stored for several weeks was still capable of inducing fruit set; however, to ensure success, use of pollen which has been stored more than a few days is not recommended.

Seed requires 6 to 12 days to germinate. About 75% of seeds planted immediately after harvest germinated. Seed of this plant does not seem to exhibit dormancy.

The resultant seedlings produced numerous local lesions in response to inoculation with PSTM, thus retaining the characteristic of the parental line.

Discussion

Why seed production should fail when flowers are pollinated at the time of anthesis is an open question. When a flower first opens, the anthers lie below the stigma but by the time of anthesis, the stamens have elongated, placing the anthers above the stigma. The time between the opening of the flowers and anthesis is about one day. This suggests that pre-anthesis pollination might be the natural mode of pollination in the wild where visitation by insects would be the most likely mechanism of pollination. The lack of successful seed production at anthesis may simply be a reflection of an effective mechanism which promotes cross pollination by taking advantage of a time differential between female receptivity and anthesis.

The method herein described to obtain seed set from S. sinensis is simple and requires no unusual techniques or facilities. Its implementation should make seed production possible wherever this plant will flower.

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Table 1. Fruit and seed set in *Scopolia sinensis* plants pollinated at different stages of flower development[1]

Flower stage when pollinated	No. of flowers pollinated	No. of fruit	No. of fruit harvested	No. of seeds	No. of seeds/fruit (avg)	No. of seeds/fruit (range)
Pre-anthesis[2]	132	59	19	548	29	0-95
Anthesis	60	7	6	2	0.3	0- 2

[1] Data collected during January and February 1972.

[2] 1-3 days before anthesis.

DISEASES OF RAPESEED IN CENTRAL AND NORTHERN ALBERTA IN 1971

B. Berkenkamp[1]

Abstract

A survey of the diseases of rapeseed in central and northern Alberta was conducted by sampling 84 fields between 8 July and 19 August 1971. The average intensities of the diseases were: white rust, 0.3% of leaf area affected; staghead, 1.2% heads affected; gray leaf spot, 0.2% leaf area affected; alternaria pod spot, 0.1 spots per pod; ringspot, 0.1% stem affected; and root rot, 11.7% roots and crowns showing symptoms. Intensity of the diseases varied throughout the season depending on the nature of the pathogens and the growth stage of the plant.

Introduction

Rapeseed has become a major crop in Alberta, increasing from 0.6 million acres in 1966 to 2.3 million acres in 1971. Rapeseed diseases have received careful attention in Saskatchewan (3, 4, 5) but have been essentially ignored in Alberta. This survey was undertaken to determine the incidence and severity of rapeseed diseases in central and northern Alberta, where about 80% of the rapeseed in the province is produced.

Materials and methods

Rapeseed fields in Census Divisions (C.D.) 8 to 15 were sampled between July 8 and August 19. The number of fields sampled in each C.D. was related to the intensity of rapeseed cultivation, i.e. approximately 1% of the number of farms growing rapeseed in 1971. Ten plants were selected at 2-pace intervals diagonally across each field, starting 10 paces from the edge. The plants sampled were read by estimating the leaf area affected by leaf pathogens, the stem area affected by ringspot caused by Mycosphaerella brassicicola, the average number of spots per pod with alternaria pod spot, the percentage of heads deformed by staghead, and the percentage affected with root rot. Average disease indices were based on all the fields sampled in the particular census division, including healthy fields.

Results and discussion

The intensity of diseases of rapeseed in Census Divisions 8 to 15 are shown in Table 1. There was insufficient acreage in C.D. 9 and C.D. 14 to warrant sampling. The foliar phase of white rust caused by Albugo cruciferarum S.F. Gray occurred in most fields (69%) but appeared to cause relatively minor loss. However staghead, the flowering shoot phase of white rust, was probably the most serious disease of rapeseed in Alberta in 1971, occurring in 24% of the fields examined. Harper (F. R. Harper, personal communication) has developed a method of determining the loss in yield from staghead. Using this method he found yield losses of up to 13% from this disease in southern Alberta. Average loss in central and northern Alberta was 1.2%.

Gray leaf spot caused by Alternaria brassicae (Berk.) Sacc. was the most prevalent disease found this year (93% of the fields sampled). Although gray leaf spot has caused serious losses in wet years (2), losses in 1971 were minor. Only 7% of the fields sampled were affected with the pod spot phase.

Ringspot caused by Mycosphaerella brassicicola (Fr.) Lindau, previously known only in moist coastal areas, was reported in Saskatchewan in 1960 (5). It appears late in the season and in moist years can be widespread; however, this year only 7% of the fields sampled were diseased. This disease apparently causes only limited losses due to its late season appearance when the crop is ripening.

Root rots of rapeseed have received very little attention; in this survey all plants with symptoms of diseases of the crown and root, however slight, were included in the root rot category. Stem blight caused by Sclerotinia sclerotiorum (Lib.) de Bary and fusarium foot rot (Fusarium spp.) have been reported but few estimates of intensity, distribution, or loss are available (1). Fly larvae were found consuming rapeseed roots in some of the fields, but they were not identified and the damage was not estimated.

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Table 1. Distribution and intensity of rapeseed diseases in Alberta Census Divisions 8 to 15, 1971

C.D.	Fields assessed		Disease index (avg)					
	No.	Date (approx.)	White rust	Staghead (%)	Gray leaf spot	Alternaria pod spot	Ringspot	Root rot (%)
8	14	July 13	0.4	0	0.2	0	0	10.7
10	17	July 28	0.4	1.8	0.2	0.1	0	5.3
11	6	July 22	<0.1	0	0.1	0.4	0	3.3
12	7	Aug. 12	<0.1	1.4	0.6	0.9	0.1	37.1
13	15	July 20	0.1	0	0.2	0	0	3.3
15	25	July 27	0.4	2.3	0.3	0	<0.1	16.6
Total or avg	84		0.3	1.2	0.2	0.1	<0.1	11.7

The range in sampling dates was unavoidable because of the extensive travel involved. The dates of assessment had a definite effect on the intensity reported for some of the diseases. For example, in the earliest sampling (C.D. 8, July 13) very little staghead, alternaria pod spot, and ringspot were found compared with the last sampling (C.D. 12, August 12), in which high levels of the late appearing diseases and lower levels of early disease, such as white rust on foliage, were encountered. Therefore, the crop should be sampled several times through the season, or disease intensity - host growth stage relationships established. The leaf diseases must be assessed before leaves become senescent and drop, and the flowering head and pod diseases must be read after they develop later in the growth cycle.

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SOUTHERN LEAF BLIGHT OF CORN IN SOUTHWESTERN ONTARIO IN 1971

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Abstract

In laboratory tests, Helminthosporium maydis race T in naturally infected corn debris survived 11 weekly cycles of thawing and freezing, the total tested. In the field the fungus survived much better in corn residues above the ground surface than in residues partly or wholly buried. The fungus overwintered from 1970 to 1971 in most corn fields. Infected seeds were present in 22% of samples of commercial seed from the 1970 seed crop. Because about 70% of seed for the 1971 corn crop contained the Texas (T) factor for male sterility that is associated with susceptibility to H. maydis race T, early and severe blight seemed possible in 1971.

Leaf lesions were first seen in late June 1971 in fields near elevators, and eight such fields were sprayed. Infection was occasionally heavy near corn cribs. The disease became general in late July, but remained light in most fields through August and September.

In growth cabinet tests at spring temperatures, few seedlings emerged from severely infected seed and the fungus took 4 weeks to appear above ground on the bases of the seedlings. These factors may have reduced disease from infected seed in the field. Dry weather and cool nights probably restricted field spread from infected debris. During the summer, day temperatures were ideal for the fungus, but rainfall was abnormally low for much of the season. At night, when leaves would be wet from dew, temperatures were average but were too low for maximum fungus growth. In southwestern Ontario, unusually wet days or unusually warm nights would be needed for the disease to develop at its full potential rate.

Introduction

Southern leaf blight of corn (Zea mays L.) caused by Helminthosporium maydis Nisikado and Miyake (Bipolaris maydis (Nisikado) Shoem., stat perf. Cochliobolus heterostrophus Drechs.), occurred in Ontario in 1970 from Essex and Kent counties, where most plants were infected, northward to Bruce and Grey counties and eastward to the Ottawa Valley (3). The race of H. maydis involved (race T) is especially able to attack corn plants that contain the Texas (T) factor for male sterility, as did most corn hybrids grown in this area in 1970. It was unavoidable that again in 1971 some 70% of corn seed contained this factor. In 1970 the pathogen reached Ontario late in the season from areas to the south, and caused little damage. For 1971, however, the possibility of its overwintering locally posed the threat of early outbreaks and more severe damage. Overwintering of the fungus on corn residues in the field and on seed from the 1970 seed crop was studied, together with the possibility of spore production on seedlings from infected seed. The incidence of the disease in southwestern Ontario in 1971 is summarized.

Methods

To determine survival of H. maydis on corn debris, the debris was placed on and between 20 corn seedlings containing T cytoplasm. Pots of seedlings about 15 cm tall were enclosed in plastic bags closed by cotton wool plugs and kept in a greenhouse at about 24 C. The bags were rumpled daily to spread any spores to the leaves. After 8-9 days, lesions were counted.

To indentify race T of the pathogen, seedlings of single crosses 29N and 29T with normal and T cytoplasm respectively (kindly supplied by Dr. G. Scheifele, P-A-G Seeds, W. R. Grace & Co., Champaign Research Station, R. R. 1, Champaign, Illinois 61820) were used as described above.

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Table 1. Survival of *Helminthosporium maydis* in field residues of corn subjected to weekly cycles of freezing and thawing[a]

Material and conditions while thawed	Number of weekly cycles[b] 1-4	5-8	9-11
2°C, kept wet			
leaves & husks	78	62	29
stalks	17	12	6
kernels	50	17	1
21°C, kept wet			
leaves & husks	100+	100+	100+
stalks	41	36	5
kernels	19	32	55
21°C, allowed to dry to about 15% moisture, rewetted before freezing			
leaves & husks	100+	100+	100+
stalks	39	28	10
kernels	100+	100+	42

[a] Average numbers of lesions per set of corn seedlings in plastic bag in test described under Methods.

[b] Weekly cycle: freeze 1 day, thaw 6 days.

Results

Overwintering of the fungus on corn debris

In laboratory tests on infected corn debris, the fungus survived 11 weekly cycles (the total tested) of freezing for 1 day and thawing for 6 days (Table 1). It survived better when kept at 21° C for each 6-day period than at 2°C, and its survival was not affected when the debris was allowed to dry during each 6 days at 21° C to about 15% moisture. In a test after the 8th cycle, the fungus gave more and larger lesions on plants with T cytoplasm than on plants with N cytoplasm.

Table 2. Overwinter survival[a] outdoors in Essex County of *Helminthosporium maydis* in field residues of corn wrapped in open-weave terylene cloth, 1970-71

Overwintering site	Sampling dates Dec. 9	Jan. 13	Feb. 25	Apr. 14
45 cm above ground	100+	71	100+	78
At ground level	90	57	34	34
10 cm below ground	70	-[c]	20	12
Check[b]	100+	100+	100+	100+

[a] Avg number of lesions on corn seedlings, see Methods for description of test; avg of 3 locations.

[b] Dried infected residues sealed in plastic bags kept outdoors 45 cm above ground.

[c] Not sampled - ground frozen.

Survival in the field

Infected leaves, stalks, and ears were wrapped separately in open textured terylene cloth and overwintered outdoors 45 cm above ground, half-buried in soil, and 10 cm below ground at each of three locations near Harrow (Table 2). The fungus survived equally well on the three types of plant material. Samples which were sealed dry in plastic bags 45 cm above ground survived without evident deterioration. The fungus survived much better in debris wrapped in cloth at 45 cm above ground than when the debris was half-buried or buried 10 cm deep.

Naturally overwintering debris collected

Table 3. Occurrence of *Helminthosporium maydis* in residues in growers' fields in Essex and Kent counties, 1971

	Period Late March - early April	Late April - early May	Mid May - early June	Mid June - early August
Essex County				
No. of fields in which *H. maydis* was detected[a]	25/30	23/31	12/38	22/35
Avg no. lesions per positive test[b]	46	14	24	10
Tests in which T cytoplasm was attacked more than N cytoplasm	8/8	14/14	5/6	6/7
Kent County				
No. of fields in which *H. maydis* was detected[a]	15/18	10/15		
Avg no. of lesions per positive test[b]	34	18	No survey in this period	
Tests in which T cytoplasm was attacked more than N cytoplasm	5/6	1/2		

[a] No. positive/no. tested.

[b] Tested on corn seedlings as described under Methods.

each month from December to June at and above the soil surface in six fields near Harrow gave the following average numbers of lesions when tested on corn seedlings as described: Dec., 24; Jan., 33; Feb., 37; March, 19; April, 4; May, 28; June, 14.

Overwintering occurred in most fields in Essex and Kent counties (Table 3), and in nearly all cases that were checked the fungus attacked plants with T cytoplasm more readily than plants with N cytoplasm.

Seed infection

In 1970 the fungus invaded the tip kernels of many ears in seed fields. Seeds from fields with badly infected ears, graded as for commercial use, were examined for infection. Untreated or surface-sterilized seeds were germinated at 21-27° C in paper towels, sand, or soil (details in Table 4).

All commercial grades contained infected seeds, though less than rejected grades. Smaller "flat" grades, which would come from nearer the ear tips, contained more infected seeds than larger "flat" or "round" grades.

Southern leaf blight of corn in southwestern Ontario in 1971

Corn seedlings emerged mainly in the middle two weeks of May in Essex County. No seedlings infected with H. maydis were seen until June 3, when 1 of 30 unthrifty seedlings collected from several fields where corn was growing after corn was infected below ground.

Leaf lesions were found in Essex County on June 24-25 on lower leaves of plants in 9 fields adjacent to elevators. In 3 of these, only single lesions were found. In areas of the other fields up to 10% of plants had 1-5 lesions on lower leaves. One of these fields was ploughed and 5 were sprayed, as described later. By mid-July, occasional lesions (up to 2% of plants with 1-3 lesions) were found in 5 other fields close to elevators, in 9 of 23 fields where corn had been grown the previous year, along the edges of 2 fields where the adjacent field had been planted with corn the year before, and in 2 of 8 fields which had not grown corn in the previous year. In the last week of July and the first week of August, 26 out of 34 fields surveyed had blight. In 16 fields, lesions occurred only on 3-15% of the leaves below the ear. Eight fields had occasional lesions on a few upper leaves, and in 2 fields lesions occurred on many upper leaves. In late September, the disease had become more general on upper leaves. In 9 of 23 fields surveyed the disease was not seen, but in 12 fields the upper leaves had up to 50 lesions per leaf and in 2 fields 200-300 lesions per leaf. Infection of the ear husks was occasional and usually penetrated only one or two husk layers.

Table 4. Infection with *Helminthosporium maydis* in seeds from fields with badly infected ears, graded as for commercial use

Seed source and treatment	% infected seeds
Seed sources[a]	
Sample A - rejected seed	41
- commercial grades:	
medium flats (18-20)	17
medium flats (22-24)	10
large flats	6
medium rounds (18-20)	8
medium rounds (22-24)	7
Sample B - rejected seed	28
- commercial seed (ungraded)	8
Sample C - ears sorted before shelling	13
Sample D - ears sorted before shelling	2
Seed treatments[b]	
Javex	21
Mercuric chloride	10
Captan dust	12
None	14
Germination methods[c]	
Paper towels	16
Trays steamed sand	9
Trays steamed soil	16

[a] Means for all seed treatment and germination methods. Sieve sizes in brackets in 1/64 inch (0.40 mm).

[b] Means for all samples and germination methods. Javex: soak 1 minute in Javex (5.25% active Cl), rinse twice. Mercuric chloride: soak 3 minutes in 95% ethanol, then in 0.1% mercuric chloride for 15 minutes, 5 rinses.

[c] Means for all samples and seed treatments. Paper towels were folded around seed and enclosed in plastic bags closed with cotton wool plugs. Trays had glass covers sealed with cotton wool between cover and tray.

Blight was identified in Kent County on June 26 at the Ridgetown College of Agricultural Technology in plots in which quantities of dried infected leaves were spread on the soil as a source of Phyllosticta maydis Arny and Nelson. Apparently the inoculum source also contained H. maydis. These plots were ploughed on June 28 to reduce the chance of early spread into other corn plots at the College.

A survey of corn fields near grain elevators in Kent County on June 28-29 revealed 7 fields with blight infections on

the lower leaves of plants growing close to the elevators. On July 3, 12 fields in Kent County were found to contain southern leaf blight, 10 next to elevators and 2 near cob dumps.

Surface-sterilized seed had about the same percentage of infection as untreated seed, suggesting that the fungus usually penetrates the seed. The fungus was most readily detected by germinating seeds in moistened paper towels. In tests in sand or soil the fungus showed at the bases of the seedling stems, but was often obscured by other fungi.

These samples, though from unusually severely affected fields, indicated that commercial seed from other fields would probably contain infected kernels. Of 1129 samples of commercial seed examined from January to March 1972 by the Plant Products Seed Testing Laboratory in Toronto, 22% contained infected seeds (Mr. P. Grainger, personal communication). The infection level was generally 0.5-2%, though occasional samples contained 10-20% infected seed.

Seedling infection and appearance of the fungus above ground

To determine how often infected seeds gave rise to infected seedlings, and how readily the fungus appeared above ground to produce spores for further spread, infected seeds were planted 5 cm deep in steamed field soil in the greenhouse or in a growth cabinet. Temperatures were 20° C by day and 13° C at night, to approximate day air temperatures and night soil temperatures in May in Essex County.

For six samples of commercial seed with a 20% estimated average infection with *H. maydis* planted in the growth cabinet average seedling emergence was 76%. Under these temperature conditions *H. maydis* appeared on the tips of the coleoptile sheaths, just above the soil surface, on 11% of the plants about 4 weeks after emergence. In the greenhouse, in similar tests under less consistently humid conditions, no *H. maydis* was observed above ground. Seeds infected to the extent that 50% of the surface of each seed was blackened were also planted in the greenhouse. Only 3-4% emerged, and no *H. maydis* was seen on these plants in the next 5 weeks.

At 16-17° C, corn and *H. maydis* grew at about half their rates of growth at 25-28° C, and the fungus took about twice as long to induce the collapse of 10-cm-segments of seedling stalks. At 11° C, both corn and fungus grew very slowly.

In early September the disease was noted as far east as Wellington County. It developed in August and September in the Ottawa Valley (2), Quebec, and Prince Edward Island (W. E. Sackston and H. W. Johnston, respectively, personal communications).

Fields sprayed for blight control

It was decided to spray 5 of the fields in Essex County that had blight on June 24-25 because they might provide an early source of spores for surrounding corn. In the first week of July and in mid July these fields were sprayed with zineb at 3.5 lb active ingredient/acre (3.9 kg/ha) plus Triton B1956 at 1/3 oz formulation/acre (23 g/ha) in 35 gallons (160 liters) water. Rain (up to 2 cm) occurred on July 4-5 and 23-25.

In late June and early July, the number of lesions multiplied 3-5 times per week in these fields. At the second spraying there were up to 20 lesions per leaf at the bottom 45 cm of the plant. On Aug. 9 there were 20-30 lesions per leaf at ear level, and 1-8 on upper leaves, about ten times as many as in Essex County in general. At this time, about 3 weeks after mid-silk, there was a potential for damage, but dry weather and cool nights restricted the disease. The disease increased in early September, and up to 200 lesions per leaf damaged some of the upper leaves, but this was too late to affect yield appreciably. There was no apparent spread of the disease to neighboring fields during the season.

In Kent County, blight did not develop as rapidly as in Essex County. On July 9 a few lesions were noted in the upper leaves in two fields and considerably more in a third field where air movement was reduced by tall weeds. These three fields were sprayed on July 14 and 23 with maneb at 2.5 lb a.i./acre (2.8 kg/ha). By mid-August there was no longer a threat of an epidemic, and spraying was discontinued.

Cribs as sources of infection

Although leaf infection was noted on two occasions in 1971 near cribs in Essex County, no serious infections resulted from this association. In Kent County, two fields near cribs from which corn had been removed in June had severe outbreaks of blight in the dozen or so rows next to the cribs were corn handling equipment had been standing in the new crop. Sections of 20 rows in each field were ploughed down on July 10 and 12. When growers were advised of this development, samples of blight infections came in from widely scattered farms in Kent County.

The widespread overwintering of the fungus on corn residues in southwestern Ontario and its presence in 20% of commercial seed samples made an early outbreak of blight seem very possible in 1971. Growers were advised to plant hybrids with normal (N) cytoplasm, to plough under corn residues, to avoid growing corn after corn, to handle stored corn as little as possible in the growing season, to plant early using a Vitavax-thiram seed dressing, and to minimize stress on the plants by sound cultural practices.

Table 5. Weather records, Harrow (Essex County) and Ridgetown (Kent County)[a], summer 1971

Period		Soil temp 10 cm depth[b] (°C)	Air temperature (°C)				Rainfall			
			Average daily maximum		Average daily minimum		Days with rain		Total rain (cm)	
		Harrow	*Harrow*	*Ridgetown*	*Harrow*	*Ridgetown*	*Harrow*	*Ridgetown*	*Harrow*	*Ridgetown*
May	1-10	10	17	18	6	5	2	1	1.1	0.1
	11-20	13	22	22	9	8	4	2	0.7	0.1
	21-31	13	18	19	7	7	2	3	2.5	2.8
June	1-10	16	23	24	13	10	4	3	2.0	1.6
	11-20	19	26	26	16	15	4	1	3.0	0.6
	21-30	22	28	29	18	17	3	4	2.7	1.4
July	1-10	23	28	28	18	18	2	1	0.5	0.7
	11-20	22	26	26	15	14	2	1	0.2	0.1
	21-31	22	25	25	16	16	6	4	4.5	3.2
Aug.	1-10	21	26	27	15	15	2	1	2.9	5.5
	11-20	21	26	26	16	16	1	2	0.1	4.6
	21-31	21	26	25	16	14	1	4	1.5	5.6
Sept.	1-10	22	27	27	20	19	2	0	1.3	0.0
	11-20	20	22	22	14	13	4	6	2.7	3.5
	21-30	17	21	21	11	11	3	2	2.4	0.9

[a] Ridgetown is 70 miles east of Harrow.

[b] Average of readings at 8 a.m. and 5 p.m., in soil with sod cover.

Except for one plant, infection of seedlings was not seen even when they grew through corn residues. The low rate of emergence of seedlings from severely infected seeds, and the 4 weeks required at spring temperatures for the fungus to appear above ground at the base of the plant, may have reduced the likelihood of disease originating from infected seed. Dry weather and cool nights probably restricted spread from debris.

Discussion

The disease was first seen near elevators, as it often was in the U.S. corn-growing areas. Growers and elevator operators were asked to avoid or reduce movement and shelling of corn until mid-August and to avoid movement of cobs and corn debris. The eight most affected fields were sprayed as described above.

Although the disease became widespread in late July and early August, infection was very light and no spray recommendations were made. At this time a further 2-3 weeks were needed for 70% yield dry matter and 2 more weeks for 90% dry matter to be obtained (1). Experiments at Harrow (unpublished) indicated that loss of leaves below the ear leaf during the first 2-3 week period would decrease yield at the most by 15%. Therefore, unless the weather favored rapid disease spread, spraying was likely to be uneconomic. In fact, dry weather and cool nights delayed the disease, which did not become general on upper leaves until late September.

The weather, as recorded at Harrow and Ridgetown (Table 5), evidently was important in restricting blight development. Rainfall at Harrow in May, July, and August was only about 70% of the long-term averages, while at Ridgetown rainfall in May, June, and July was only about half the long-term averages. Average temperatures were usually within 1-2 C of the long-term averages, except in early September. Because the widespread establishment of new infections would require moisture from rain or dew, night temperatures during periods of dew probably limited the development of the fungus. At Ridgetown, rainfall totalled 15.7 cm in August, but night temperatures were low. The general summer night low temperatures of 15-18 C are those which allowed the fungus only half its maximum growth rate on agar, and this limitation would presumably operate in any normal season in this area. In addition, low soil temperatures and low night air temperatures in May and early June would restrict the development of H. maydis from infected seeds and debris.

Maximum day temperatures of 23-28 C are ideal for the fungus through June-early September, but these days in 1971 were mostly dry.

It would seem therefore that for southwestern Ontario a period of unusually wet days or of unusually warm nights would be needed for the disease to develop at its full

potential rate. It was during unusually hot and humid weather, particularly at night, in August 1970 and early September 1971 that the disease became general in this area.

The fungus overwintered from fall 1970 to spring 1971 in many areas of the United States in addition to the southeastern states (4, 5), and it evidently survives a wide range of winter conditions. It is therefore likely to overwinter again in southwestern Ontario, and the use of hybrids with normal (N) cytoplasm is recommended for the future. Because some lesions occurred in 1971 on plants with normal cytoplasm, efforts to prevent overwintering of inoculum are needed to reduce the chance of development of fungus races able to attack severely plants with normal cytoplasm. It was the general experience that the fungus overwintered best in corn residues above the soil surface (4, 5), and the destruction of such debris by deep ploughing is therefore of great importance.

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SOUTHERN LEAF BLIGHT OF CORN IN EASTERN ONTARIO IN 1971[1]

A.T. Bolton and W.L. Seaman

Abstract

In 1971 southern leaf blight of corn was not found in eastern Ontario until the first week of September. Although the disease then became widespread throughout the area, damage to the rapidly maturing crop was negligible. Race T of Helminthosporium maydis was isolated from infected plants and from overwintered corn debris. The primary inoculum affecting corn crops in the surveyed area apparently originated locally from corn residue on the surface of the soil.

Introduction

In eastern Ontario in 1970 southern leaf blight of corn was first observed in mid September (Gates et al. 1971). Although the disease caused very little damage in the few fields affected during September and October, it was suggested that an earlier and more severe outbreak could occur in 1971 if inoculum overwintered in this area. Therefore corn fields at various locations were examined frequently during the 1971 growing season.

Observations

Blight-like symptoms were reported on corn from Renfrew County in August, but the occurrence of southern leaf blight in eastern Ontario was not confirmed until September 6, when Helminthosporium maydis Nisikado (stat perf. Cochliobolus heterostrophus (Drechs.) Drechs.) race T was isolated from corn growing near a crib site in Dundas County about 30 miles south of Ottawa (Fig. 1). Most plants in this field were infected, and 10% to 60% of the leaf area of infected plants was necrotic. A week later in Renfrew County the pathogen was isolated from a field where approximately 2% of the plants exhibited light infection. During the 2-week period following the first positive identification of the disease, leaf symptoms appeared in 26 of 56 fields examined, and by the last week of September southern leaf blight was widespread throughout the major corn growing area. At this time most of the crop was nearing maturity and overall the disease had little effect on yield. However in a few fields of very late corn that was still quite green the disease spread rapidly, involving a high percentage of all leaves and

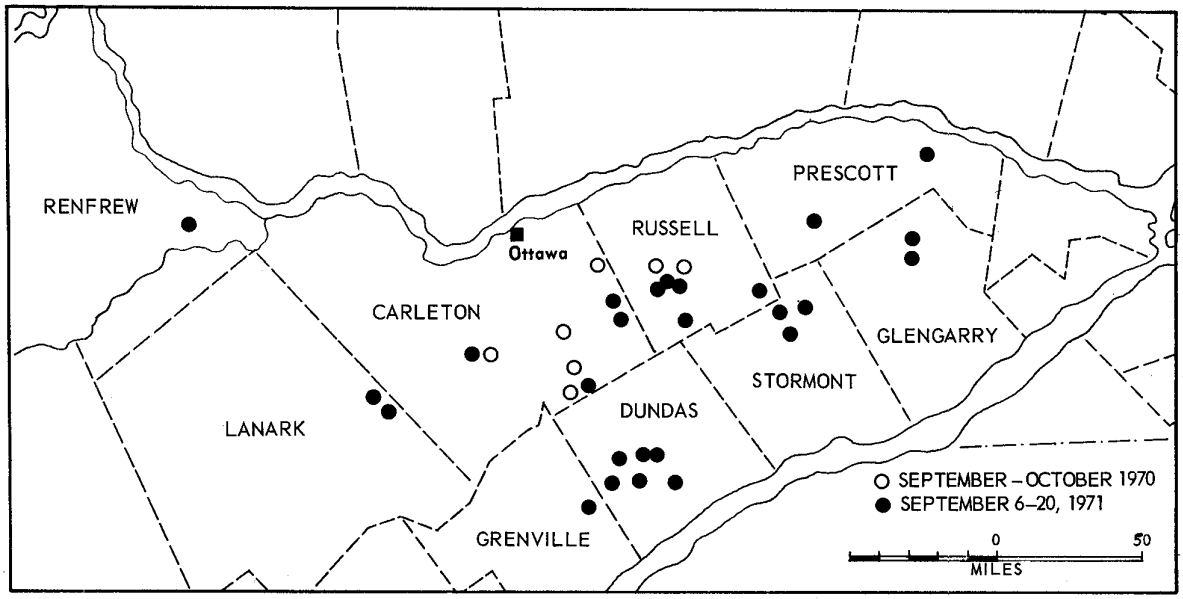

Figure 1. Distribution of southern leaf blight of corn in nine eastern Ontario counties in 1970 and 1971. In late September & October 1971 blight became generally distributed throughout the area.

[1] Contribution No. 314, Research Station, Canada Department of Agriculture, Ottawa, Ontario K1A 0C6

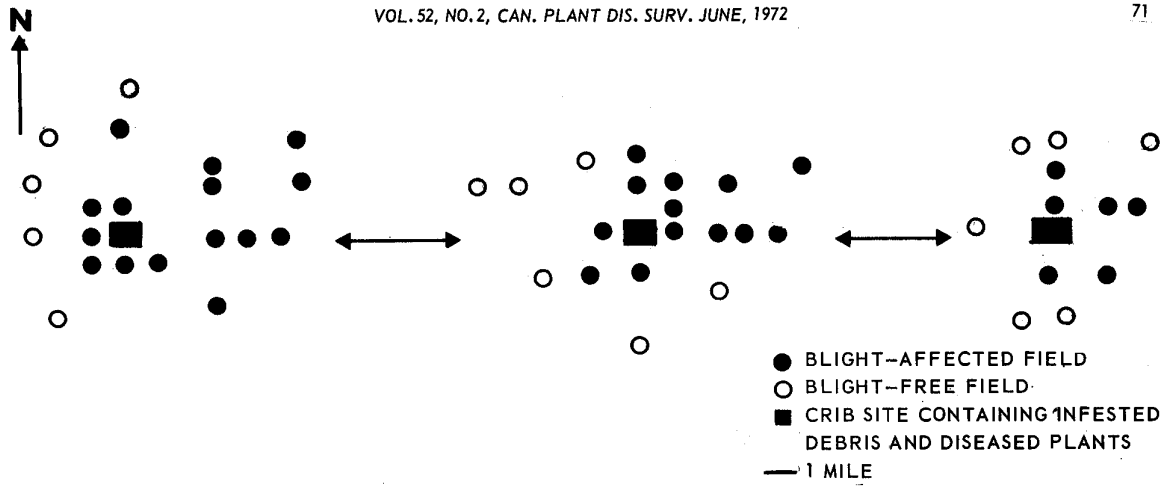

Figure 2. Distribution of corn fields affected by southern leaf blight in the vicinity of crib sites containing overwintered corn debris at three locations in eastern Ontario, October 1971.

cobs on less mature plants.

To determine the source of the inoculum responsible for infection, samples of overwintered corn residue were collected and plated on potato dextrose agar. H. maydis was isolated from kernels, shelled cobs, husks, sheath tissue, and stalks from three locations; in each case the infested residue was collected from the surface of the soil in the vicinity of cribs holding corn harvested in 1970. Volunteer corn plants growing among the debris and plants nearby in adjoining fields were severely affected by blight; many were stunted and showed 80-90% necrosis of the leaf area.

Examination of corn fields in the vicinity of these inoculum sources disclosed a heavily infested zone near the cribs. Although infection was found in nearby fields in all directions, the disease was most prevalent in fields to the east of the cribs (Fig. 2).

Discussion

Weather conditions during early summer 1971 were not conducive to the development and spread of southern leaf blight in eastern Ontario. Rainfall was light and there were few periods in July when corn leaves remained wet for more than a few hours at temperatures above 65° F (18° C). Similarly in central Ontario lengthy cool periods in July and August suppressed development of the disease, and blight was not found in the Guelph area until September 16, following the first period of weather conditions favoring blight multiplication (Gillespie 1972).

Although blight indices based on temperature and leaf wetness (Gillespie 1972) were not calculated for eastern Ontario, three potentially favorable periods for blight development occurred during August. At Ottawa the total rainfall for the month, 5.05 inches, occurred during Aug. 9-14, Aug. 19-22, and Aug. 27-30; undoubtedly much of the development of blight near crib sites and the subsequent spread of the disease evident in early September occurred during these periods.

The fact that H. maydis overwintered successfully on corn residue in this area and the pattern of disease development in fields surrounding crib sites suggest that primary inoculum was produced locally in 1971.

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SURVEY FOR SOUTHERN LEAF BLIGHT OF CORN IN QUEBEC IN 1971

W.E. Sackston and J.W. Sheppard[1]

Abstract

Helminthosporium maydis was identified for the first time in Quebec on August 5, 1971, in Iberville County. By harvest time southern leaf blight was widespread throughout the corn growing area of Quebec, but the distribution pattern was uneven, and severe leaf symptoms developed in only a few fields in September and early October. Cob infection was not found and little or no reduction in yield was expected.

Introduction

Southern leaf blight of corn attributed to race T of Helminthosporium maydis Nisikado was reported in Ontario in 1970, with significant infections in fields in the Ottawa Valley in mid-September to mid-October (Gates et al. 1971). A one-day survey from Macdonald College to the Deschambault Experimental Station in mid-September, 1970, revealed miscellaneous lesions (including frost damage) on corn leaves in the limited part of Quebec traversed, but H. maydis was not found (unpublished).

In 1971 a systematic survey was made of the main corn area of Quebec. With the advice of Dr. R. I. Brawn, formerly of the Department of Agronomy, and with the help of Agronomes of the Quebec Department of Agriculture and Colonization, staff at CDA Research Stations, seed merchants, and cooperating farmers, locations were chosen where fields were sown with hybrids with Texas male-sterile (T) cytoplasm. Some of the fields were sown with mixtures containing approximately 50% normal (N) cytoplasm seed.

Methods

Twenty-four "stations" were located by August 12 (although station 22 was first visited September 16). The stations, consisting of farms on which one or more fields of corn were grown, were revisited at approximately 2-week intervals. In addition to the regular stations, 33 other farms were visited, most of them only once, to check on patterns of southern leaf blight in other areas. Surveys were terminated October 17.

Samples were collected wherever leaf lesions were found and were incubated in moist chambers. Disease intensity and distribution were estimated and recorded in the field, but all diagnoses were based on the results of microscopic examination of incubated tissues. Only a few samples were surface-sterilized and plated. It is quite probable that some lesions were not identified because of the failure of the pathogen to sporulate on them.

Observations and discussion

The first positive identification of H. maydis in Quebec was from a single lesion on one leaf of 'Dekalb 007' collected August 5 at station 5 in Iberville County. There were several hybrids and a corn variety test plot on this farm. On August 11, H. maydis was identified on leaves collected in Deux Montagnes Co., from a variety purchased at a premium price as N cytoplasm seed.

When station 5 was revisited August 16, about 2% of the plants had one lesion on the bottom leaf. At a second station in Iberville Co. on the same date about 5% of the plants had 10 to 12 spots per leaf on all five leaves below the cob, and 1 or 2 spots on the first leaf above the cob on scattered plants. No lesions were found on the first visit there August 5.

Traces to 2% of the plants had up to 5 spots on one or two lower leaves on the second visit to fields in Iberville, Napierville, and Chateauguay counties August 17 to 26, although no lesions were found earlier. The first lesions in the Plant Pathology plots at Macdonald College (Jacques Cartier Co.) were collected August 20.

On August 27, two field in Deux Montagnes Co. had scattered lesions on the leaves below the cob, and 1 or 2 lesions on the cob leaf on 5% to 20% of the plants. In one field 75% of the plants had about 20 lesions on the bottom leaf, and from 1 to 3 lesions on all leaves above the cob. The kernels were in the "blister" stage.

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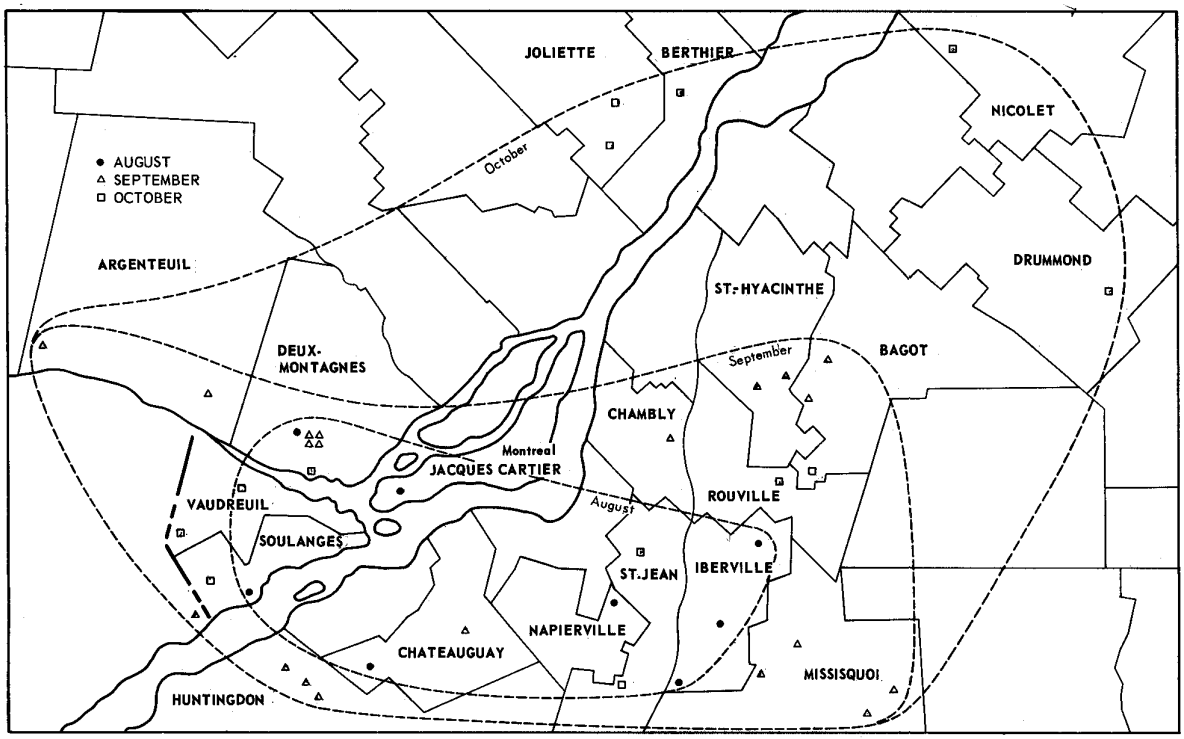

Figure 1. Distribution of southern leaf blight of corn in Quebec, August 1 – October 15, 1971. Blight was also detected in Pontiac Country (not shown) September 4.

In Vandreuil Co. at station 10, where corn had been grown for 6 or 7 years in various fields, about 3% of the plants had one or two lesions on lower leaves, August 31.

On September 11, a few lesions were found on about 5% of the plants in a field in Huntingdon Co. In a nearby field there were many lesions on the lower leaves and 3 to 10 lesions on the cob leaves of 75% of the plants. The kernels were in the dent stage. On the same date a few plants in a field in Bagot Co. had up to 10 spots per bottom leaf; and about 65% of the plants in a field in Mississquoi Co. had lesions on the lower leaves. A few spots were found in a field in Pontiac Co., September 4. About 50% of the plants had numerous spots on the cob leaf in a field in Argenteuil Co., September 16.

Light infections of southern leaf blight were found just before harvest (October 14-16) in fields in Berthier, Joliette, Nicolet, and Drummond counties.

The disease was widespread in the entire corn area by harvest time. However the distribution pattern was erratic. Some fields of T cytoplasm corn remained relatively or entirely free of southern leaf blight throughout the season, although infection appeared early and developed rapidly in other fields in the same area. For example, blight appeared August 20 and developed rapidly on plants in one experiment in the Plant Pathology plots at Macdonald College, but it was not identified in a nearby plot or in differential varieties until October 28. In the College farm fields the disease was not seem until early October.

Although the disease developed rapidly, damage is believed to have been negligible, at worst. At Station 2, Iberville Co., blight severity increased from 4 to 5 spots per bottom leaf on 1% of the plants, August 17, to 10 spots on the cob leaf on 75% of the plants, September 9. However infection reached this severity in only a few fields even by early October. Plants were in mid to late silk August 17, and kernels were beginning to dent by September 9.

In four fields in late September and early October, lesions were observed on the husks, but not on the cobs themselves.

The most significant finding of the survey is that southern leaf blight was present and widespread in Quebec by the end of the season. Presumably there will be some overwintering of the fungus, to provide inoculum early in the season in 1972. Development of the disease in 1972 will depend largely on weather conditions. If they are favorable to the pathogen (high temperatures and high humidity), the disease could start earlier and develop faster than in 1971 and might induce some loss of yield in susceptible T cytoplasm corn. Seed of N cytoplasm corn should be in fairly good

supply in 1972, and growers who can obtain it at reasonable prices might be well advised to do so. All growers are advised to cultivate their fields thoroughly to cover any diseased corn debris as well as possible before the new crop emerges.

Acknowledgments

The authors are grateful to all those who assisted them in locating fields of known 'T' cytoplasm corn. The survey was made possible by a grant from the Quebec Agricultural Research Council.

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PARATYLENCHUS PROJECTUS IN ALFALFA FIELDS OF CENTRAL AND NORTHERN ALBERTA[1]

G.R. Webster[2] W.R. Orchard[3] and E.J. Hawn[4]

Abstract

Paratylenchus projectus has been detected in considerable numbers in certain soils of Alberta. It appears to be associated with a disease of alfalfa in central and northern Alberta. Twenty-three percent of the soils analyzed contained more than 4,000 nematodes/kg of soil. Most of the high counts were from the Dark Gray Luvisolic soils where, of the areas examined, alfalfa "sickness" was the most prevalent. Further studies are in progress.

Introduction

Paratylenchus projectus Jenkins (4) was first detected in soils of Alberta by the late W. R. Orchard in 1970 and identified by Dr. L. Y. Wu. The occurrence of this nematode appears to be associated with a widespread disease of alfalfa (*Medicago sativa* L.) in central and northern Alberta. Symptoms of the disease, called "alfalfa sickness" (7), are poor plant growth interspersed with irregularly shaped patches of healthy growth. Affected plants are short, spindly, yellowish-green in color, and poorly nodulated. Additions of N, P, K, and micronutrients have not materially improved the growth of diseased plants (5, 7, 8). Genetic selection against the toxic soil condition has proved futile (3.) Beneficial effects from treating "sick" soils with steam and with Vapam (metam, sodium) have been reported (7). Research on the effects of root temperature and leachates from "sick" soils on growth of alfalfa (5, 7) has indicated that a toxic agent, probably biologic in origin, may be at least partially responsible for the disease. Particular interest in nematodes of the genus *Paratylenchus* was stimulated by Orchard, who during the period 1962 to 1969 examined alfalfa plants and soil adhering to their root systems and consistently found appreciably higher counts of *Paratylenchus* in soils from areas of poor growth as compared to areas of good growth. There were a few nematodes of the genera *Tylenchus* and *Tylenchorhynchus* in all samples. He recommended that a survey be conducted to determine the magnitude of infestation of *Paratylenchus* in central and northern Alberta. This paper presents a summary of the counts that were obtained in 1970.

Materials and methods

Alfalfa plants and soil adhering to their root systems were collected from fields selected at random and from experimental plots, giving a total of 43 locations. The distribution gave a fairly broad coverage of the central and the Peace River areas of Alberta. Sampling was done during June and September 1970. Excess soil was trimmed from the root system leaving approximately 300 g of soil which was washed from the roots in about 6 liters of water; the suspension was allowed to settle for a few seconds and the supernatant passed through a 60-mesh Endecott sieve. The material on the sieve was rinsed and the entire supernatant passed through 100, 200, 325, and 400-mesh sieves and the screenings from the latter two were placed in Baermann funnels for roughly 16 h. (1). Counts of *Paratylenchus* were based on morphological characteristics (6).

Results and discussion

Counts of *Paratylenchus* varied widely from field to field, covering a range from zero to greater than 7,000/kg soil. Twenty-three per cent of the soils contained more than 4,000 *Paratylenchus projectus*/kg of soil. Most of the high counts were from the Dark Gray Luvisolic soils in areas west and north of Edmonton where of the areas examined alfalfa "sickness" was the most prevalent.

Since alfalfa, oats, barley, sweet clover, brome, and orchard-grass are hosts of *P. projectus* (2), it would appear that extensive research is warranted to determine the implications of its presence. Field

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Table 1. Counts of *Paratylenchus projectus* in alfalfa fields of central and northern Alberta

Count range*	Percentage of samples in each range
0	6.8
1- 500	20.9
500- 1,000	13.9
1,000- 2,000	13.9
2,000- 3,000	9.3
3,000- 4,000	11.8
4,000- 5,000	9.3
5,000- 6,000	4.7
6,000- 7,000	4.7
7,000-40,000	4.7

* Average numbers of nematodes in soil washed from root systems of three to five alfalfa plants per field or plot, expressed as numbers per kg of oven-dry soil.

surveys were continued in 1971 to further determine its distribution and experiments are in progress to determine its role in the alfalfa "sickness" disorder. The presence of other parasitic nematodes were also recorded in the 1971 survey.

Acknowledgments

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