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CANADIAN PLANT DISEASE SURVEY



EDITOR W.L. SEAMAN



RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE



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EDITOR W.L. SEAMAN, Research Station, Ottawa

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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."

ECONOMIC LOSS FROM MUMMY BERRY OF Highbush BLUEBERRY IN COASTAL BRITISH COLUMBIA, 1969¹

H.S. Pepin and H.N.W. Toms²

Abstract

Measurement of loss caused by mummy berry of highbush blueberry was made on the variety Rancocas for both ascospore and conidial infection. Using these data, the total loss for all varieties grown in British Columbia in 1969 was calculated to be \$66,462 or 8.14% of the value of the crop.

Introduction

Man's food supply is menaced by three main factors: bad weather, insect pests, and plant diseases. Although each alone can be destructive, they generally act in concert and for this reason it is often difficult to measure the loss caused by any one factor.

Highbush blueberry production in coastal British Columbia during 1969 was almost a classic example of the interaction of these three factors. Production was down 18% from the 1968 crop of 4,572,000 pounds (Personal communication, G.R. Thorpe, B.C. Department of Agriculture). Part of the loss was caused by blossom bud damage during an unusually severe winter, part by a heavy leafroller infestation and part by mummy berry, a fungus disease caused by Monilinia vaccinii-corymbosi (Reade) Honey.

This paper is an attempt to determine the portion of the total loss due to mummy berry.

Materials and methods

Seven fields of the highbush blueberry variety Rancocas were chosen from those of 122 growers as representative of the disease situation. Infection ranged from severe to trace amounts. Rancocas was chosen as a highly susceptible variety against which the susceptibility of other varieties could be measured (1) and because it is the most widely grown variety.

Bushes were chosen at random throughout each field and the numbers of flower cluster and leaf shoot infections per bush were recorded. Generally, ten bushes were counted per acre, although the number varied according to severity of infection. The data were averaged for each field.

During early July another sample of bushes was chosen at random in the same fields for counts of mummy berries on the bushes and on the ground beneath them. The average number of healthy berries per cluster for each bush was also recorded.

Samples of ripe berries were weighed and counted and the average weight per berry determined.

The average number of infected flower clusters multiplied by the average number of berries per cluster, times the average weight per ripe berry gives the average weight of berries per bush lost from ascospore infection. The average number of mummy berries per bush multiplied by the average weight per ripe berry gives the average weight of berries per bush lost from conidial infection. Adding the losses per bush from ascospore infection and from conidial infection together and multiplying by 800 (the average number of bushes per acre) gives the loss per acre in pounds of marketable fruit. The loss in pounds per acre was multiplied by \$0.20, the estimated final price per pound to the growers in 1969, to obtain a dollar value loss per acre.

On the basis of previous work (1), which compared the relative susceptibility of other varieties with 'Rancocas', the dollar value loss per acre for 'Rancocas' was used to calculate the total loss to the industry.

Results and discussion

Loss in the variety Rancocas due to mummy berry ranged from \$15.20 to \$227.25 per acre and averaged \$98.35 per acre (Table 1). Total loss for all farms for this variety was calculated to be \$21,265 based on a total Rancocas acreage of 216.22.

All the important highbush blueberry varieties grown in coastal British Columbia have been put into three classes of relative susceptibility to both ascospore and conidial infection (1). These are: resistant, moderately susceptible, and susceptible; moderately susceptible is rated as one-half

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that of susceptible. Approximately one-half the loss per acre was due to ascospore or flower cluster infection (Table 1), while the remaining loss was due to the formation of mummy berries by conidial infection. If a variety is classified as susceptible to conidial infection its susceptibility class is 1.0 and if classified as moderately susceptible, 0.5. Multiplying the susceptibility class by \$49.18, the loss per acre from conidial infection for Rancocas, gives the loss per acre from conidial infection for the variety.

However, a further complication appeared with ascospore infection. Not all ascospore infection results in flower cluster infection; leaf shoots may also be infected. The proportion of each type depends on the variety. Fortunately, data on these proportions are available (1). Therefore, to obtain the loss per acre from ascospore infection, \$49.18 was multiplied by 1.0 or 0.5, depending on the susceptibility class, times the percentage of flower cluster infections. Loss per acre from conidial infection added to loss per acre from ascospore infection gives the total loss per acre for the variety.

No attempt was made to estimate losses from leaf infection. Leaf shoots produce

flower cluster buds and losses from the failure to produce buds would not show up until the following season.

The calculation of variety loss (VL) may be summarized in the following formula:

$$VL = [(csc \times Rcl) + (asc \times fci \times Ral)] \times A$$

where csc = conidial susceptibility class
Rcl = Rancocas conidial loss
asc = ascospore susceptibility class
fci = flower cluster infection percentage
Ral = Rancocas ascospore loss
A = acreage

For example, for the variety Berkeley, csc = 0.5, Rcl = \$49.18, asc = 0.5, fci = 0.65 (1), Ral = \$49.18 and A = 33.88 (Table 2). Therefore, variety loss = $[(0.5 \times \$49.18) + (0.5 \times 0.65 \times \$49.18)] \times 33.88 = \$1,374.51$.

This procedure was followed for all varieties to obtain a grand total loss of \$66,462 for all growers (Table 2).

The total estimated value of the 1969 crop is \$750,000. The percentage loss to the industry is, therefore,

$$\frac{66,462}{750,000 + 66,462} \times 100 = 8.14\%$$

Table 1. Loss due to mummy berry disease in the highbush blueberry variety Rancocas on seven farms in 1969

Rancocas acreage	Fruit lost (lb) from			Loss*	
	Flower cluster infection	Mummies	Total	\$/acre	Total \$/farm
8.0	3179	5911	9090	\$227.25	\$1818.00
5.0	550	372	922	36.88	184.40
4.5	2360	622	2982	132.53	596.40
4.0	2853	998	3851	192.55	770.20
2.0	101	51	152	15.20	30.40
4.0	282	970	1252	62.60	250.40
5.0	127	409	536	21.44	107.20
Total 32.5	9452	9333	18,785	\$688.45	\$3757.00
Average/acre	291	287	578	\$ 98.35	

* Based on a price to the grower of \$0.20/lb.

Table 2. Loss due to mummy berry disease in highbush blueberry in British Columbia, 1969

Variety	Susceptibility rating			Loss per acre			Acres**	Total loss
	Flower cluster	Asco-spore	Conidia	Asco-spore	Conidia	Total		
Atlantic	0.00	0.0	0.0	0.00	0.00	0.00	0.93	\$ 0.00
Berkeley	0.65	0.5	0.5	15.98	24.59	40.57	33.88	1,374.51
Bluecrop	0.29	1.0	0.5	14.26	24.59	38.85	134.12	5,210.56
Blueray	0.59	1.0	0.5	29.02	24.59	53.61	11.13	596.68
Burlington	0.62	1.0	1.0	30.49	49.18	79.67	2.70	215.11
Charlotte	0.60	1.0	1.0	29.51	49.18	78.69	4.50	354.11
Collins	0.94	1.0	0.0	46.23	0.00	46.23	6.13	283.39
Concord	0.76	0.5	1.0	18.69	49.18	67.87	45.68	3,009.62
Coville	0.76	1.0	0.5	37.38	24.59	61.97	34.03	2,108.84
Dixi	0.42	0.0	0.0	0.00	0.00	0.00	97.43	0.00
Earliblue	0.23	1.0	1.0	11.31	49.18	60.49	15.71	950.30
Fraser	0.92	0.0	0.0	0.00	0.00	0.00	13.07	0.00
Grover	0.77	0.0	0.0	0.00	0.00	0.00	0.15	0.00
Jersey	0.74	0.5	1.0	18.20	49.18	67.38	83.77	5,644.42
Johnston	0.98	0.0	0.5	0.00	24.59	24.59	0.33	8.11
June	0.53	1.0	1.0	26.07	49.18	75.25	56.55	4,255.39
Pacific	1.00	0.0	0.5	0.00	24.59	24.25	0.01	0.25
Pemberton	0.58	0.5	0.0	14.26	0.00	14.26	57.43	818.95
Pioneer	0.67	1.0	0.5	32.95	24.59	57.54	4.65	267.56
Rancocas	*	1.0	1.0	49.18	49.18	98.35	216.22	21,265.24
Rubel	0.51	0.0	1.0	0.00	49.18	49.18	64.68	3,180.96
Stanley	0.12	1.0	0.0	5.90	0.00	5.90	37.43	220.84
Weymouth	0.63	1.0	1.0	30.98	49.18	80.16	66.17	5,304.19
Seedlings	0.50	0.5	0.5	24.59	24.59	49.18	38.14	1,875.73
Mixed Vars.	0.50	0.5	0.5	24.59	24.59	49.18	178.39	8,773.22
Scammell)								
Cabot)								
Phyllis)								
Ivanhoe)								
Evelyn)								
GN 87)	Susceptibility rating unknown, averaged for computation					49.18	13.30	654.09
Shirley)								
Herbert)								
Wareham)								
1613)								
Total							1,216.20	\$66,462.07

* Rancocas loss determined by count.

** Acreage based on 1968 survey by British Columbia Department of Agriculture.

This figure of 8.14%, based on measurement and calculation, although lower than previously published losses of 10% in 1965 (2) and 15% in 1966 (3), is probably not significantly different from the earlier figures even though the latter were only estimates.

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VERIFICATION OF WHEAT SPINDLE STREAK MOSAIC VIRUS AS A CAUSE OF MOSAIC OF WHEAT IN ONTARIO¹

J.T. Slykhuys² and Z. Polák³

Abstract

Wheat spindle streak mosaic was observed in 62 of 107 fields of winter wheat surveyed in Ontario in May and June 1969. About 37% of the wheat surveyed was infected, which is moderate in comparison with none to 65% for other years since 1960.

The presence of the virus in field collections of plants and soil was verified by manual, root soak, and soil transmission to wheat. In manual transmission tests, wheat spindle streak mosaic virus was differentiated from wheat streak mosaic virus and agropyron mosaic virus by producing spindle streak mosaic symptoms on wheat kept in a growth room at 8-12°C but not in a greenhouse at 18-25°C. Manual transmission was not as successful from plants collected and tested in mid-June as in May.

Filamentous particles were observed with difficulty by the leaf dip electron microscope technique in young plants with spindle streak mosaic symptoms.

Introduction

A mosaic disease caused by a soil-borne virus has been recognized on winter wheat in Ontario since 1957 (4). Although the disease was at first assumed to be caused by a strain of wheat (soil-borne) mosaic virus (1,2,3), the cause is now recognized to be a different virus with filamentous rather than tubular particles and is designated wheat spindle streak mosaic virus (WSSMV) (5).

Surveys were made in Ontario in 1969 to compare with those of previous years. Special emphasis was placed on using transmission tests to verify the identification of the causes of symptoms observed in the field.

Materials and methods

The first general survey was made between May 12 and 16 when most wheat in most districts was in the late tillering stage. Another survey was made during June 16-19 when most wheat was headed and in or

approaching flowering. In early May the presence of WSSMV was often evident from the road by a yellowish-brown discoloration in patches or throughout the field. Similar discolorations are sometimes caused by other factors, but on close examination the presence of WSSMV was indicated by a light green to yellow mosaic including spindle-shaped dashes parallel to the leaf axis. These markings could occur anywhere on the leaf from near the tip to the base. Sometimes dashes or blotches of tissue were grayish white and necrotic. During June the disease was identifiable principally by short, spindle-shaped streaks or light green to yellow mosaic on the youngest two or three leaves. The affected plants were usually only slightly stunted but had fewer tillers than mosaic-free plants.

Verification of the presence of WSSMV in field collections with mosaic symptoms was done by transmission from plant and soil samples and sometimes by electron microscopic examination of leaf dip preparations.

Manual transmission tests were done by grinding 1 g of leaf tissue with 3 ml of water and about 2 mg of celite, then rubbing the mixture on the leaves of 'Kent' winter wheat in the 2-3 leaf stage. Some plants were kept in a growth room at 8-12°C with about 1200 ft-c of light for 12-16 hr/day. Similarly inoculated wheat plants and 'Clintland 60' oat plants were placed in a greenhouse at about 18-25°C. The development of spindle streak mosaic symptoms on wheat kept up to 3 months at 8-12°C, but not on plants at 18-25°C, indicated the presence of WSSMV. The development of mosaic on wheat, but not on oats, at 18-25°C indicated the presence of agropyron mosaic virus (AMV),

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while symptoms on oats indicated wheat streak mosaic virus (WSMV).

A root-soak transmission test was done by lightly washing the roots of each diseased plant to free them from soil, then placing them in water in petri dish with 15-20 sprouted wheat seeds. After one day at 20C and another at 8-12C, the seedlings were transplanted into a pot of noninfective soil and grown at 8-12C for 3 months.

Soil infectivity tests were done by placing a 4-cm layer of test soil on top of a noninfective potting soil mixture in a 5-inch clay pot, then seeding 'Kent' wheat (15-20 seeds) in the test soil and maintaining at 8-12C for 3 months. When necessary a satisfactory test could be done by mixing a few grams of the test soil with the potting soil at seed level.

Results

Field surveys, 1969

Wheat spindle streak mosaic was observed in 62 of the 107 (58%) fields of winter wheat observed in May and June. The estimated percentages of diseased plants varied from trace to 20% in 14 fields, 21 to 80% in 11 fields and 81 to 100% in 37 fields. The mean infection for all fields was 37.6%.

The incidence of WSSM in 1969 was moderate in comparison with the incidence in other years in the decade (Table 1). There

were 6 years in which diseased plants were found in a higher percentage of fields and 5 in which the mean percentages of infected plants were higher.

Transmission tests for WSSMV in field samples

Although the number of test plants that developed symptoms was variable, the combined results of the three types of transmission tests confirmed the presence of WSSMV in all Ontario fields from which plants with WSSM symptoms were collected (Table 2). The manual transmission tests indicated the presence of WSSMV in all plant samples with WSSM symptoms collected in May but failed for three of the four samples collected in June after the wheat was headed and had experienced very warm weather. The lack of mosaic development in oats kept at 18-25C after inoculation indicated that WSMV was not present in any of the Ontario samples tested. The development of symptoms in inoculated wheat kept either at 8-12C or 18-25C, but not in oats, confirmed the presence of AMV in one sample of wheat, No. 64, which had symptoms resembling agropyron mosaic. The root-soak method confirmed the presence of WSSMV in 8 of the 11 samples tested, and the soil infectivity test confirmed the presence of WSSMV in 13 of the 16 samples tested, including the four samples collected in June.

Observations of particles with the electron microscope

Although particles in leaf dip preparations from plants with symptoms of

Table 1. Incidence of wheat spindle streak mosaic in winter wheat in Ontario, 1960-69

Year	Total fields	No. fields according to % diseased plants				Fields with disease		Mean % infection
		0%	tr-20%	21-80%	81-100%	No.	%	
1960	43	25	5	4	9	18	72.0	24.7
1961	106	25	6	7	68	81	76.4	61.6
1962	50	29	9	5	7	21	42.0	19.4
1963	39	5	13	6	15	34	87.1	45.6
1964	33	25	3	1	4	8	24.2	13.3
1965	24	24	0	0	0	0	0.0	0.0
1966	41	12	4	6	19	29	70.7	50.0
1967	140	19	8	26	87	121	86.4	65.1
1968	96	33	11	6	46	63	65.6	47.4
1969	107	45	14	11	37	62	57.9	37.6

Table 2. Verification of WSSMV in plant and soil samples from Ontario fields in which mosaic-disease plants were found in 1969

Field No.	Location	Mosaic-diseased plants		Date samples collected	Manual transmission to			Infection from		Virus identified	
		Symptoms	%		Oats 18-25C	'Kent' wheat 18-25C	wheat 8-12C	Root of dis. plants	Field soil	WSSMV	Other
1(a)	Ottawa	WSSM		May 27			8/29	1/17	1/21	+	
1(b)	Ottawa	WSSM		June 20		0/11	2/28	2/24	7/27	+	
4	Pt. Colborne	WSSM	100	May 13	0/20	0/22	5/34	0/20	7/71	+	
5	Pt. Colborne	WSSM or AM	1 plt	May 13	0/18	0/15	1/28			+	
7	Pt. Colborne	WSSM	<1	May 13	0/26	0/14	1/27		0/41	+	
24	Talbotville	WSSM	75	May 13	0/21	0/15	5/26	2/16	2/54	+	
27	Blenheim	WSSM	75	May 13	0/29	0/17	6/32	1/17	12/18	+	
28	New Glasgow	WSSM	75	May 13	0/26	0/14	20/33		5/40	+	
30	Cedar Springs	WSSM	100	May 13	0/22	0/13	13/27	1/15	2/10	+	
47	Clandeboyne	WSSM	25	May 13	0/26	0/14	7/41	1/15	4/47	+	
49	Clandeboyne	WSSM or WSM?	<1	May 13	0/15	0/15	5/28	0/13	0/27	+	
51	Exeter	WSSM	90	May 13	0/15	0/10	9/27	0/15	0/16	+	
58	Tottenham	WSSM	50	May 13	0/20	0/12	9/24	2/16	5/65	+	
59	Bondhead	WSSM	100	May 13	0/29	0/13	1/21	2/14	2/13	+	
64	Renfrew	AM	1 plt	May 13	0/15	11/12	15/15			-	AMV
100	Clinton	WSSM	100	June 19		0/10	0/34		12/15	+	
101	Clinton	WSSM	75	June 19		0/9	0/22		1/12	+	
106	Newmarket	WSSM	100	June 19		0/11	0/37		5/16	+	
M4	WSMV control	WSM		May 14	25/25	15/16	11/29		0/27	-	WSMV
Ck	Potting soil mixture				0/25	0/16	0/15		0/27		

WSSM were more readily observed after shadowing with palladium than after staining negatively with phosphotungstic acid, they were generally much more difficult to find than particles of WSMV or AMV in plants infected with these viruses. Particles were rarely found in preparations from wheat collected in June. It was sometimes necessary to examine several grids prepared from a plant infected with WSSMV to find a few particles, which were usually partly obscured by non-specific, fine, granular material, but sometimes clumps of particles were found. The particles were thin (12-13 m μ) and flexuous and varied in length from 200 to >2000 m μ . Particles of WSMV and AMV were more readily found and were more distinctly visible, more uniform in size (19 x 650 - 750 m μ), but less flexuous than particles associated with WSSM (5,6). Although electron microscopy provided an additional means for indicating the presence of WSMV or AMV in mosaic diseased plants, the unreliability of this method for finding particles identifiable with WSSMV limited the usefulness of the leaf dip electron microscopic technique for routine determination of the presence of WSSMV.

Serology as an aid in distinguishing viruses causing mosaic symptoms in wheat

Microprecipitin tests with specific antisera (6) were useful for identifying WSMV and AMV in clarified juice from mosaic-diseased wheat plants. Sometimes the titre of AMV in field-collected plants was too low to detect the virus, but if young wheat plants were inoculated and the juice

extracted after 12-14 days at 18-25C, the virus was usually detectable in precipitin tests.

Although attempts were made to develop an antiserum by injecting rabbits with concentrates of particles from plants infected with WSSMV, subsequent use of serum from the rabbits in precipitin tests failed to demonstrate the presence of WSSMV in clarified juice from diseased plants.

Discussion

Although the manual transmission, root-soak transmission, and soil infectivity tests each yielded positive results for the presence of WSSMV in most samples, and the combined results confirmed its presence in all samples from fields in which WSSM was observed, the percentages of test plants infected by each method were variable. Manual transmission was more successful for samples collected in May than in June when the wheat was more mature. The small number of test plants infected in any of the tests by the root-soak method probably indicates that temperature conditions during the infection period were not optimum for the vector or the virus. The variable results from soil transmission tests may reflect differences in infectivity of the soils as well as failure to provide optimum environmental conditions for infection.

Symptoms produced by WSMV and AMV on some plants may sometimes be difficult to distinguish from WSSM in the field. However,

the presence of these viruses can be determined easily by the differences in host reactions, the development of symptoms in one to two weeks at temperatures too high for WSSM development, lack of soil transmissibility, differences in particle morphology, and reactions to specific antisera.

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ZOOSPORIC FUNGI ASSOCIATED WITH WHEAT SPINDLE STREAK MOSAIC IN ONTARIO¹

D.J.S. Barr² and J.T. Slykhuis³

Abstract

Polymyxa graminis Led. has been found frequently in Ontario on wheat plants infected with wheat spindle streak mosaic. Other wheat parasites, *Olpidium brassicae* (Wor.) Dang., *Rhizophydium graminis* Led., and *Lagena radiculicola* Vanterpool and Led., have also been found in Ontario and they are being investigated as possible vectors of the virus.

Introduction

Wheat spindle streak mosaic caused by a soil-borne virus (WSSMV) has been common on winter wheat in Ontario most years since it was first observed in 1957. It has been prevalent only in fields in which three or more preceding crops of winter wheat were grown at intervals of 5 years or less (5).

The soil relationships of WSSMV appear to be similar to those for wheat soil-borne mosaic virus (WMV). Infectivity is retained in soil stored dry at room temperature for several years, in fractions of soil that pass through sieves with 44 μ openings, and in organic fractions from soil. However, infectivity can be destroyed by heating at 52C or by treating with any of a number of fungicidal chemicals.

These characteristics are consistent with the possibility that the virus is harbored and transmitted by a root-invading zoosporic fungus, several of which have been observed on roots of Gramineae in Ontario:

Polymyxa graminis Led. (Plasmodiophorales) was discovered in 1929 during an investigation of certain root rots of wheat occurring in Ontario (3). There is good evidence that it is a vector of WMV in the U.S.A. (1,4).

Olpidium brassicae (Wor.) Dang. (Chytridiales) is a vector of several plant viruses, including tobacco necrosis, tobacco stunt, and lettuce big vein (7). It occurs in the roots of many plants in Ontario.

Rhizophydium graminis Led. (Chytridiales) was first described from material found in 1932 in roots of wheat grown in soil from the Central Experimental Farm, Ottawa (2). Unlike the other zoosporic fungi considered here, which are endobiotic, this fungus produces epibiotic zoosporangia and resting spores on root hairs and epidermal cells. Only delicate rhizoids penetrate the host cells. The epibiotic resting spores soon become separated from the roots leaving no observable bodies in the roots. Although this fungus has seldom been observed, it may be very common and possibly a vector.

Lagena radiculicola Vanterpool and Led. (Lagenidiales) was reported to be a parasite of wheat, barley, and *Zea mays* in Western Canada (9) and Ontario where it was also found on wild grasses (8). It produces endobiotic zoosporangia and resting spores.

Root examinations were made to determine if any of these fungi were commonly associated with wheat spindle streak mosaic in Ontario, and if they occurred on certain other common grasses.

Methods

Wheat (*Triticum aestivum* L.) plants showing symptoms of spindle streak mosaic were collected from the main winter wheat producing districts of Ontario during surveys in May and June 1969 (6), from field plots at Ottawa, and from growth rooms operated at 8-12C. Roots of various grasses, principally from the vicinity of Ottawa, were also examined.

The roots were washed and small portions mounted in water and examined with a phase contrast microscope. Fungi were identified by their characteristic zoosporangia or resting spores. As a further check on the presence of fungi, the plants were potted in washed white sand and incubated for 3-4 weeks by a south-facing window in a laboratory, or at 15-20C in growth chambers. These conditions encouraged rapid spread of *P.*

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graminis, O. brassicae and L. radiculicola and, in the absence of soil particles, the fungi were easily seen. This technique was not satisfactory for R. graminis which was seen only on roots of plants from soil.

Results

Zoosporangia and resting spores of P. graminis were found in the roots of spindle streak mosaic diseased wheat plants from 12 of 13 fields sampled in the counties of Carleton, Elgin, Kent, Middlesex, Simcoe, and Welland. P. graminis was also found in wheat seeded in infested soils from these counties and grown at 8-12C in a growth room. However, it was also found in mosaic-free wheat grown on land at Ottawa for which there was no record of a previous crop of wheat.

In host range tests, Hordeum vulgare L. 'Vantage', Bromus inermis Leyss. 'Lincoln', and Agropyron repens (L.) Beauv., which appear to be immune to WSSMV, became infected with P. graminis.

Olpidium brassicae was detected in roots of wheat from 5 of the 13 fields of WSSM-diseased wheat sampled, including fields in Carleton, Elgin, Kent, and Simcoe counties. It was also found in roots of mosaic-free wheat grown in soil collected from fields in which wheat has probably never been grown previously, and in roots of the grasses Poa pratensis L., Hordeum jubatum L., Phleum pratense L., Agrostis palustris Huds., and Agropyron repens.

Rhizophydium graminis was found on wheat that developed wheat spindle streak mosaic while growing at 8-12C in soil collected from a field in Brant county in which the disease was severe in 1968. The resting spores of this fungus were abundant on the surface of roots and root hairs 6 to 8 weeks after seeding, but later they were difficult to find. R. graminis was not found on roots of plants collected from the field but since the resting spores do not persist on the roots, failure to find them does not necessarily indicate that the fungus had not been present.

Lagena radiculicola was found on Agropyron repens at Ottawa, but it was not detected in roots of wheat infected with WSSMV.

Discussion

The similarities in transmission in soil and factors involved in the persistence and elimination of infectivity of soil indicate

that WSSMV and WMV may have a similar vector and vector relations. The evidence that WMV is transmitted by P. graminis (1,4) and the frequent finding of this fungus in roots of wheat infected with WSSMV indicate that it may be a vector of WSSMV. Proof that P. graminis is a vector may be achieved from experiments with unifungal cultures of this fungus grown on wheat roots in association with the virus, but the slow development of wheat spindle streak mosaic prevents rapid results. In the meantime, each of the zoosporic fungi known to infect wheat must be considered as potentially a vector.

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LOSSES FROM CEREAL DISEASES AND VALUE OF DISEASE RESISTANCE IN MANITOBA IN 1969¹

W.C. McDonald, J.W. Martens, G.J. Green, D.J. Samborski, G. Fleischmann, and C.C. Gill

Data on losses caused by plant diseases and on gains in productivity from the use of disease control measures developed through research are becoming increasingly important to agencies such as FAO, U.S.D.A. and C.D.A. for assessing research programs and setting priorities. The terms of reference of the C.D.A. Research Station, Winnipeg, include "to develop varieties of wheat, oats and barley suitable for production in the Prairie Provinces" and "to devise protective measures for the growing crops...against disease". To fulfill our objectives and assess the value of our research under these terms of reference we must have data showing the importance of various diseases and the gain in cereal production through the use of resistant varieties.

In the past, pathologists in Manitoba relied on limited field surveys and yield trials in experimental plots to assess the importance of the various diseases. This approach was successful and the most important diseases were given a high priority in establishing breeding programs for their control. However, except for rusts (2, 8, 13, 17), very little was published on the dollars or bushels lost on a provincial basis to justify the initiation of research on specific diseases.

Similarly, the obvious advantages of new, disease resistant varieties have been such that no need was felt to provide extensive data on their value to growers or to those providing funds for research. Some data are available on the value of rust-resistance in new varieties of wheat and oats (2, 13) but, with current attitudes towards budgeting for research programs, more data of this type will have to be provided for all diseases.

This paper reports estimates of losses caused by major diseases of wheat, oats and barley and the value of disease resistance obtained from an extensive survey of Manitoba in 1969.

Materials and methods

Six survey routes, two for each crop, were mapped to cover all of the crop districts in Manitoba (Fig. 1). The acreage of each crop in each crop district was obtained (3, 4, 5)

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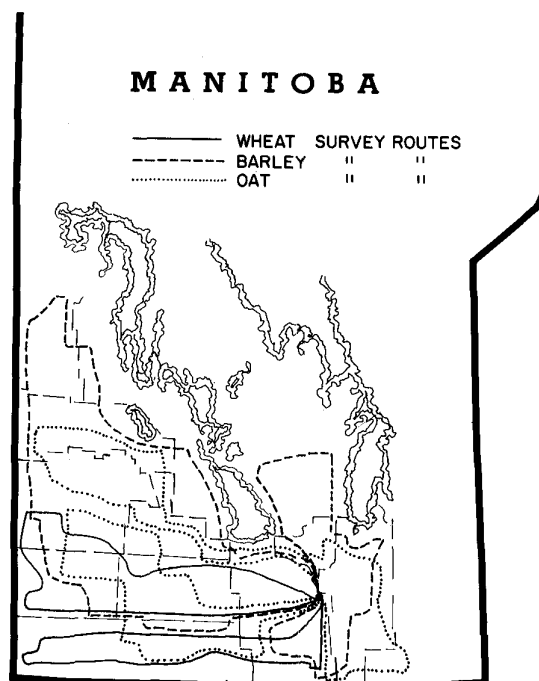


Figure 1. Map of the agricultural area of Manitoba showing survey routes.

and each pair of routes was designed to pass through the crop districts in which over 75% of the particular crop was grown. The length of the routes varied from 600 to 1100 miles.

The optimum number of survey sites in each crop district was arbitrarily set at 1% of the farms growing the specific crop (Table 1). The number of farms, rather than the number of acres in a district, was used because management practices such as seed treatment, crop rotation, and other disease control measures should be uniform on a particular farm regardless of the size of fields. It was assumed that a sample of this size would represent fields differing in variety, date of seeding, incidence of disease and environmental conditions. The sites were marked on the route maps at 15- to 20- mile intervals and the field closest to each site on the route was surveyed. Although each route was designed to survey a specific crop, some sites for the other two crops were also included. In districts where a specific crop was highly concentrated, the

Table 1. Number of farms with specific crops, number of survey sites, and percentage of farms surveyed in Manitoba

Area and crop district	Wheat				Oats				Barley			
	No. of farms ¹	Farms surveyed		No. of farms ¹	Farms surveyed		No. of farms ¹	Farms surveyed		No. of farms ¹	Farms surveyed	
		Number	%		Number	%		Number	%		Number	%
East												
4	513	5		605	10		403	5				
5	1750	16		1880	15		843	11				
6	96	1		189	1		29	0				
12	600	3		728	6		321	3				
Total	2959	25	0.9	3402	32	0.9	1596	19	1.1			
Central												
3	3723	23	0.6	3204	27	0.8	1649	16	1.1			
South-west												
1	1544	13		1207	10		586	6				
2	2335	12		1913	14		994	11				
7	1422	11		1299	13		632	8				
Total	5301	36	0.7	4419	37	0.8	2212	25	1.1			
West-central												
8	1541	15		1427	14		571	8				
9	1360	14		1276	16		571	10				
10	2543	19		1852	18		1465	15				
14	599	1		630	2		273	0				
Total	6043	49	0.8	5185	50	1.0	2880	33	1.1			
North-west												
11	1818	11		1489	10		810	12				
13	932	6		656	5		702	6				
Total	2750	17	0.6	2145	15	0.7	1512	18	1.2			
Total		150			161			111				

¹ Yearbook of Manitoba Agriculture, 1968.

number of sites was less than 1% because disease incidence on cereals is fairly consistent over wide areas and it is unnecessary to select sites less than 15 miles apart. Disease incidence in each field was assessed on 25 plants, one collected every 2 paces along a traverse 50 yards long and 50 yards in from the edge of the field. Disease ratings and information on stage of growth, location, etc. were recorded on crop-specific survey forms.

The surveys were completed during the period of July 30 to Aug. 12 when most of the crop was in the soft dough stage. For the final analysis of the data, the 14 crop districts were grouped into five areas based on previous knowledge of the general distribution of diseases.

The range and mean percentage loss for each disease were determined and the potential average yield in each area was found by multiplying the average yield by 100 and dividing by 100 minus the percentage loss from all diseases. The loss in bushels from individual diseases was calculated by multiplying the mean percentage loss from a disease by the potential yield and by the acreage.

The methods of assessing losses from individual diseases were based on published data, where available:

Smuts - The percentage of main tillers destroyed was used as the percentage yield reduction (10). Where less than 1% of the crop was affected the incidence was rated as

trace and was not included in the estimate of loss. The value of smut resistance in cereals was calculated by multiplying the total actual production by the percentage of the acreage sown to resistant varieties and by the percentage smut in susceptible varieties. The percentage loose smut in common wheat was estimated at 1.3%, the average incidence of loose smut in susceptible durum wheat during the past 6 years. The percentage smut in oats and barley was estimated from data obtained in years prior to the widespread use of smut resistant varieties. In oats the average incidence of all smut was 1.2% during the 8 years, 1947-1954, and in barley it was 2.1% during the 16 years, 1951-1966.

Rusts - Losses from rust were estimated by relating the percentage infection observed in the field to yield losses sustained from similar levels of infection in experimental trials (6, 7, 15). The gain in wheat production from the use of stem-and leaf-rust resistant varieties was obtained by comparing the average yield of 'Manitou' (resistant to stem rust and leaf rust), 'Thatcher' (susceptible to leaf rust) and 'Marquis' (susceptible to stem rust and leaf rust) in the 1969 Western Wheat Co-operative Tests. The mean yields in cwt/acre from the four stations in the rust area of Manitoba (Winnipeg, Morden, Portage la Prairie and Brandon) were: 'Manitou', 24.0; 'Thatcher', 21.5; and 'Marquis', 18.1. The mean yields from the four stations in the adjoining rust-free area of Saskatchewan (Indian Head, Yorkton, Melfort, and Regina) were: 'Manitou', 25.3; 'Thatcher', 25.3, and 'Marquis', 24.1 cwt/acre. The gain in production from leaf rust resistance was calculated from the difference in yield between 'Manitou' and 'Thatcher' as a percentage of the yield of 'Manitou'; and from stem rust resistance, the difference between 'Thatcher' and 'Marquis' yields as a percentage of 'Manitou' less 4.7%, the percentage yield advantage of 'Manitou' and 'Thatcher' under rust-free conditions. The acreage in the North-west area was not included in calculating the gain in production because of the low incidence of rust.

Leaf spots - Disease incidence was recorded on the flag and second leaves only and a formula developed for scald of barley (11) was used to calculate yield losses from all leaf spot diseases on all crops. Although assessment formulae have not been developed for all diseases, we believe that this formula gives a close approximation of losses from leaf spots on cereals based on previous observations (1, 9, 14, 19). Where possible, losses were attributed to specific diseases but, as they usually occurred as a complex, the total loss from all leaf spot diseases was used in the final estimation.

Virus diseases - By comparing the yield from individual plants that were moderately infected with barley yellow dwarf with that

from adjacent apparently healthy plants of barley and oats, it was shown that the disease caused a 65% decrease in yield per plant (Gill and Martens, unpublished data). The percentage of infected plants observed in a field was multiplied by 65% to obtain the percentage yield loss from barley yellow dwarf.

Thrips - Infestations of thrips (*Limothrips denticornis* Hal.) were rated as severe, moderate or light and the yield loss was assessed at 10, 5, or 1%, respectively, based on results from North Dakota (18). In severe infestations, all plants in a crop exhibited complete chlorosis of the flag leaf and often also of the sheath. Mature thrips and larvae were abundant beneath the sheath.

Results

Wheat

Losses from the major diseases of wheat that occurred in Manitoba in 1969 amounted to 3.4 million bu or 5% of the potential yield without disease (Table 2); and the value of rust and smut resistance was estimated at 11.6 million bu or \$16.3 million (Table 5).

Over 99% of the wheat acreage in Manitoba was sown to 'Manitou' or 'Selkirk' so that losses from stem rust caused by *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn. were practically nil, and losses from leaf rust caused by *Puccinia recondita* Rob. ex Desm. were mainly confined to late-sown fields of 'Selkirk' and to the few fields of susceptible varieties still grown. The potential severity of the leaf rust epidemic was shown by a field of 'Thatcher' in the West-central area in which a loss of 20% from leaf rust was estimated. A final yield of 18 bu/ac was reported by the grower of this crop compared to yields of over 30 bu/acre reported for 'Manitou' grown in adjacent fields.

All of the common wheat varieties grown in Manitoba are resistant to loose smut caused by *Ustilago tritici* (Pers.) Rostr., and only trace infections have been recorded for the past 5 years. The incidence of loose smut on susceptible durum varieties was 0.8%.

Leaf spots caused losses estimated as high as 26% in individual fields and they were the major factor contributing to the total yield loss. The results substantiate observations on the increased prevalence of these diseases in western Canada and North Dakota (16) during the past few years. It was difficult to distinguish in the field between symptoms caused by the three major leaf-spotting fungi, *Drechslera tritici-repentis* (Died.) Shoem., *Bipolaris sorokiniana* (Sacc. in Sorok.) Shoem. and *Septoria avenae* Frank f. sp. *triticea* T. Johnson. Collections of infected leaves were incubated in moist chambers and usually more than one of the pathogens were isolated.

Table 2. Yield losses from disease in wheat in Manitoba, 1969

Area (Crop Dist.)		Yield losses from			Total	Average yield ¹ (bu/ac)	Potential av. yield (bu/ac)	Acres ¹ (000)	Potential production (000 bu)
		Leaf rust	Leaf spot	BYDV					
East (4, 5, 6, 12)	Range (%)	0-10	0-20	0-3		19.3	20.4	384	7833.6
	Mean (%)	3.33	2.08	0.25	5.66				
	bu (000)	260.9	162.9	19.6	443.4				
Central (3)	Range (%)	T ² -10	0-20	0-7		18.4	19.4	395	7663.0
	Mean (%)	1.22	3.57	0.44	5.23				
	bu (000)	93.5	273.6	33.7	400.8				
South-west (1, 2, 7)	Range (%)	0-10	0-20	0-7		27.6	28.8	732	21081.6
	Mean (%)	1.42	2.28	0.39	4.09				
	bu (000)	299.4	480.7	82.2	862.3				
West-central (8, 9, 10, 14)	Range (%)	0-20	0-26	0-1		28.3	29.7	692	20552.4
	Mean (%)	2.37	2.29	0.12	4.78				
	bu (000)	487.1	470.6	24.7	982.4				
North-west (11, 13)	Range (%)	0-2	T-15	0-3		31.3	33.5	297	9949.5
	Mean (%)	0.47	5.94	0.18	6.59				
	bu (000)	46.8	591.0	17.9	655.7				
Total (000 bu)		1187.7	1978.8	178.1	3344.6	25.5	26.8	2500	67080.1
% of potential yield		1.77	2.95	0.27	4.99				

¹ Personal communication, M. Daciw, Statistics Branch, Manitoba Department of Agriculture.

² T = trace.

Losses from barley yellow dwarf were not great except in late-sown fields in the southern part of the province.

A loss of 5% was estimated from root rot (*B. sorokiniana*) in the one field in which the incidence was recorded as being greater than trace.

Oats

Losses from diseases in oats amounted to 5.3 million bu or 7% of the potential yield and was attributed mainly to an epidemic of crown rust (*Puccinia coronata* Cda.) that occurred on the one-third of the oat acreage that was sown late in the southern part of the province (Table 3). Smut-resistant varieties occupy 99% of the acreage of oats in Manitoba and the increase in production through their use amounted to 832,000 bu or \$411,800 (Table 5).

Losses from barley yellow dwarf virus on late-sown oats were much less than on late-sown barley because the predominant virus strain in 1969 was carried by *Rhopalosiphum maidis* Fitch, which prefers barley to oats as a host.

Leaf spot diseases caused losses in individual fields but in most fields the incidence was rated as trace. *Drechslera avenacea* (Curt. ex Cke.) Shoem. and *Septoria avenae* Frank f. sp. *avenae* were isolated alone or in combination from infected leaves.

Barley

Losses caused by the major diseases and by thrip damage in barley were estimated at 4.1 million bu or 8.7% of the potential yield (Table 4) and the gain in production from the use of smut-resistant varieties amounted to 621,300 bu or \$466,000 (Table 5). About one-

Table 3. Yield losses from disease in oats in Manitoba, 1969

Area (Crop Dist.)		Yield losses from			Total	Average yield ¹ (bu/ac)	Potential av. yield (bu/ac)	Acres ¹ (000)	Potential production (000 bu)
		Crown rust	Leaf spot	BYDV					
East (4, 5, 6, 12)	Range (%)	T ² -40	0-15	0-40		37.8	41.3	367	15,157.1
	Mean (%)	3.13	1.75	3.72	8.60				
	bu (000)	474.4	265.2	563.8	1303.4				
Central (3)	Range (%)	T-60	0-7	0-10		38.8	44.4	291	12,920.4
	Mean (%)	10.56	1.22	0.78	12.56				
	bu (000)	1364.4	157.5	100.8	1622.8				
South-west (1, 2, 7)	Range (%)	0-40	0-14	0-10		51.0	55.2	331	18,271.2
	Mean (%)	6.08	1.11	0.43	7.62				
	bu (000)	1110.9	202.8	78.6	1392.3				
West-central (8, 9, 10, 14)	Range (%)	0-20	0-20	0-7		49.7	52.0	387	20,124.0
	Mean (%)	1.90	1.88	0.68	4.46				
	bu (000)	382.4	378.3	136.8	897.5				
North-west (11, 13)	Range (%)	0-5	0-2	0-1		52.4	52.7	154	8,115.8
	Mean (%)	0.33	0.13	0.07	0.53				
	bu (000)	26.8	10.6	5.7	43.1				
Total (000 bu)		3358.9	1014.5	885.7	5259.1	45.3	48.8	1530	74,588.5
% of potential yield		4.50	1.36	1.19	7.05				

¹ Personal communication, M. Daciw, Statistics Branch, Manitoba Dept. of Agriculture.

² T = trace.

third of the loss occurred in the eastern area, where a severe epidemic of barley yellow dwarf reduced yields of late-sown barley. Most of the loss from smut diseases occurred on susceptible 2-rowed varieties where the incidence of loose smut caused by *Ustilago nuda* (Jens.) Rostr. and of seedling-infecting smuts (*U. nigra* Tapke and *U. hordei* (Pers.) Lagerh.) as high as 9 and 12%, respectively. No loose smut and only a small percentage of seedling-infecting smuts was recorded on 6-rowed varieties. Leaf-spotting diseases were most severe on the late-sown crops in the eastern area where spot blotch (*B. sorokiniana*) and net blotch (*Drechslera teres* [Sacc.] Shoem.) predominated. Only trace infections of root rot (*B. sorokiniana*) were observed except in one field in the north-west area where the loss was estimated at 20%.

Damage from thrips was a major cause of yield losses in the western areas of the province where most of the crop was seeded early. Two factors contributed to the lower infection in the eastern and central areas.

About one half of the barley acreage was sown late in these areas and Post (18) has reported that damage is less severe on late-sown crops. Also, 'Herta' barley was sown on over 35% of this acreage and it is resistant to thrip damage (H. P. Richardson, unpublished data).

Discussion

The estimates of disease loss and the value of resistance, calculated from 1969 survey data are believed to be conservative, for several reasons. The estimates were based on the incidence of the major diseases of the three crops and only their effect on yield was considered. Decrease in the quality of diseased crops also has a significant bearing on their value but estimating this type of loss from disease, or the gain from resistance, was beyond the scope of a survey of this scale. Stem rust of wheat, which has the potential of causing major losses in the absence of resistant

Table 4. Yield losses due to diseases in barley in Manitoba, 1969

Area (Crop Dist.)		Yield losses from					Average yield ¹ (bu/ac)	Potential av. yield (bu/ac)	Acres ¹ (000)	Potential production (000 bu)
		BYDV	Leaf spot	Leaf rust	Thrips	Smut				
East (4, 5, 6, 12)	Range (%)	T ² 57	0-30	0-T	0-10	0-9				
	Mean (%)	19.89	7.76	0	0.53	0.79	28.97	36.5	204	7446.0
	bu (000)	1481.0	577.8	0	39.5	58.8	2157.1			
Central (3)	Range (%)	0-14	T-15	0-17	0-T	0-12				
	Mean (%)	2.06	1.88	1.06	0	0.81	5.81	27.8	217	6032.6
	bu (000)	124.3	113.4	63.9	0	48.9	350.5			
South-west (1, 2, 7)	Range (%)	0-3	T-7	0-3	0-10	0-1				
	Mean (%)	0.38	1.10	0.12	1.24	0.04	2.88	41.7	279	11634.3
	bu (000)	44.2	128.0	14.0	144.3	4.7	335.2			
West-central (8, 9, 10, 14)	Range (%)	0-6	0-27	0-4	0-10	0-T				
	Mean (%)	0.39	3.36	0.73	2.46	0	6.94	44.3	329	14574.7
	bu (000)	56.8	489.7	106.4	358.5	0	1011.4			
North-west (11, 13)	Range (%)	0-7	0-6	0	0-10	0-T				
	Mean (%)	0.37	0.68	0	2.16	0	3.21	39.9	171	6822.9
	bu (000)	25.2	46.4	0	147.4	0	219.0			
Total (000 bu)		1731.5	1355.3	184.3	689.7	112.4	4073.2	35.4	1200	46510.5
% of potential yield		3.72	2.91	0.40	0.48	0.24	8.76			

¹ Personal communication, M. Daciw, Statistics Branch, Manitoba Department of Agriculture.

² T = trace

varieties, did not appear in Manitoba until late in the season. It is difficult to place a true value on rust resistance today as practically all of the wheat crop in Manitoba and throughout the Mississippi Valley is resistant. Under these conditions the build-up of inoculum is severely restricted and reaches significant levels much later in the season than previously when only susceptible varieties were grown. Other diseases of major importance in some years, such as aster yellows (20), septoria leaf blotch (9), and stem rust of barley and oats were also absent. The relatively low average percentage of smut in susceptible varieties that was used to calculate the value of smut-resistant varieties does not give a true indication of the value of smut resistance. Seed-treatment fungicides also contribute to the control of smut and were responsible for the lower incidence of the disease recorded prior to the introduction of resistant varieties, compared to the levels reported in earlier years (10).

It should be emphasized that the value placed on new varieties reflects only the increased yield attributable to their resistance to specific diseases. It does not include the value of their increased productivity through improvements in yielding

ability, adaptability, quality, resistance to lodging or other agronomic characteristics.

The methods used for this survey are open to criticism by those who desire greater statistical accuracy in data collecting. The size of the sample examined at each site was admittedly small but we believe that, as there is more variability in cereal crops as to variety, management and other factors between farms than there is between areas in a field, it is better to obtain smaller samples from many sites rather than larger samples from a few. The methods of relating disease intensity to yield losses were selected from published reports but differences in varieties or weather conditions as they affect disease development and plant maturation could change the intensity-loss ratios. A compromise is required between the degree of precision desired and the availability of resources for surveying. We believe that the methods used were adequate for the purpose but admit that the total loss from diseases was underestimated. The use of Co-operative Test data to obtain yield comparisons of resistant and susceptible varieties gives an underestimate of the value of disease resistance in years when much of the crop is seeded late because of wet spring weather.

Table 5. Value of disease resistance in cereal varieties grown in Manitoba in 1969

Crop	Disease	Loss on ¹ susceptible vars. (%)	Acreage of ² resistant vars. (%)	Total production ³ (000 bu)	Gain in production (000 bu)	Price ³ (\$/bu)	Value (\$000)
Wheat	Stem rust	9.5	99.6	54418.6	5169.8	1.40	7237.7
	Leaf rust	10.4	99.6	54418.6	5659.5	1.40	7923.3
	Loose smut	1.3	95.5	63704.8	790.9	1.40	1107.3
	Total				11620.2		16268.3
Oats	Smut	1.2	99.0	69327.5	823.6	0.50	411.8
Barley	Smut	2.1	69.7	42447.5	621.3	0.75	466.0
Total					13065.1		17146.1

¹ See text for methods of determining loss on susceptible varieties.

² Seedtime and Harvest, Nos. 74, 75, 76. Federal Grain Ltd., Winnipeg, Man.

³ Personal communication, M. Daciw, Statistics Branch, Manitoba Dept. of Agric.

As the Co-operative Tests are seeded as early as possible, there is less loss from disease than on crops that are planted later and are exposed to the full effects of late-season inoculum.

Despite these limitations, the data accumulated on disease losses are believed to be reasonably accurate although conservative, and indicate the importance of cereal diseases and the value of disease resistance in Manitoba in 1969.

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DISEASES OF CEREALS IN THE MARITIME PROVINCES IN 1969¹

H. Winston Johnston²

Many cereal diseases occur in the Maritime Provinces in endemic proportions and periodically become epidemic causing severe yield decreases as occurred in 1967 (4). In the summer of 1969 a survey was carried out to identify and assess the occurrence and severity of cereal diseases in order to establish priorities as to their economic importance. This report contains the results of a thorough survey of Prince Edward Island, and a partial survey of north-eastern Nova Scotia and southern New Brunswick. Fungal diseases were identified on the basis of both symptoms and characterization on the causal organism. The only virus disease reported herein is barley yellow dwarf on oats and barley; no attempt was made to locate or identify other virus diseases.

Several diseases were found to be widespread in all cereal growing areas while others occurred less frequently (Table 1). Some diseases were found in isolated instances and caused unknown but probably insignificant yield decrease. Resistance to specific diseases was present in some cultivars and produced variations in disease intensity and distribution.

Barley

The most widespread barley diseases found were the leaf blotch and root rot incited by *Cochliobolus sativus* (Ito and Kurib.) Drechs. ex Dastur. These diseases were found in all areas examined. Barley yellow dwarf virus infections were severe in localized areas, notably eastern Nova Scotia. Severe losses were usually restricted to a particular grower and it is believed that cultural practices, especially late seeding and lack of insecticide applications, may be the cause of the excessive yield loss. In Nova Scotia, where barley was severely damaged, yields as low as 12 bushels per acre were recorded while a few miles distant, yields were more than four times this level. Ergot incited by *Claviceps purpurea* (Fr.) Tul. was more prevalent in New Brunswick and Nova Scotia than in Prince Edward Island.

Oats

The most prevalent oat disease was leaf blotch incited by *Septoria avenae* Frank. This disease was present in all surveyed areas as reported earlier by Hamilton (1). Leaf blotch incited by *Helminthosporium*

Oats

The most prevalent oat disease was leaf blotch incited by *Septoria avenae* Frank. This disease was present in all surveyed areas as reported earlier by Hamilton (1). Leaf blotch incited by *Helminthosporium avenae* Eidam also occurred, in most instances concurrent with *Septoria* leaf blotch, and the influence of *H. avenae* on the overall yield reductions is unknown. Red leaf, caused by the barley yellow dwarf virus, was found to be severe on oats in localized areas of Prince Edward Island.

Rye

Rye is not extensively cultivated in the Atlantic area. Ergot incited by *Claviceps purpurea* and leaf spotting by *Cochliobolus sativus* were the most commonly occurring diseases recorded.

Wheat

Powdery mildew of wheat incited by *Erysiphe graminis* DC. f. sp. *tritici* Em. Marchal was very prevalent in Prince Edward Island and in areas where wheat was observed in Nova Scotia and New Brunswick. 'Opal' wheat, a newly introduced high yielding variety, was observed to be quite susceptible to head blights incited by *Fusarium* spp. Such head blights were more severe in New Brunswick than in the other two provinces. Loose smut incited by *Ustilago tritici* (Pers.) Rostr. occurred in all 'Opal' fields examined; incidence was approximately 0.1%. This is considered to be a low incidence of smut, but it should be noted that all other wheat varieties grown in the Maritimes are resistant to loose smut. Leaf rust incited by *Puccinia recondita* Rob. usually does not occur in the Maritimes until after flowering, as was observed on 'Opal', and it is not known how serious a disease this is under these conditions.

Several diseases not mentioned in this report but reported earlier (2,3) were probably localized in distribution and were not observed. Although this report does not present any definite quantitative evidence as to yield decreases caused by the various diseases, *Septoria avenae*, *Cochliobolus sativus* and *Erysiphe graminis* are considered to be the most important pathogens, based on prevalence alone, and they may cause the greatest yield losses.

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Table 1. Distribution of cereal diseases in the Maritime Provinces in 1969

Crop	Disease	Pathogen	Distribution*		
			P. E. I.	N. S.	N. B.
Barley	Spot blotch	<u>Cochliobolus sativus</u> (Ito and Kurib.) Drechs. ex Dastur.	1	1	1
	Net blotch	<u>Pyrenophora teres</u> (Died.) Drechs.	2	2	
	Speckled leaf blotch	<u>Septoria passerinii</u> Sacc.	3	3	
	Scald	<u>Rhynchosporium secalis</u> (Oud.) J. J. Davis	3	3	3
	Stripe	<u>Helminthosporium gramineum</u> Rabh.	3		
	Leaf rust	<u>Puccinia hordei</u> Oth.	2	2	
	Stem rust	<u>Puccinia graminis</u> Pers.	3		
	Loose smut	<u>Ustilago nuda</u> (Jens.) Rostr.	3	3	3
	Covered smut	<u>Ustilago hordei</u> (Pers.) Lagerh.	3	3	
	Ergot	<u>Claviceps purpurea</u> (Fr.) Tul.	3	2	2
	Common root rot	<u>C. sativus</u> <u>Fusarium</u> spp.	1	1	1
	Yellow dwarf	Barley yellow dwarf virus	2	2	2
	Nutritional disorders	Unbalanced fertilization	3	3	3
	Non-parasitic	Non-parasitic brown spot**	3		
Oats	Speckled leaf blotch	<u>Septoria avenae</u> Frank.	1	1	1
	Leaf blotch	<u>Helminthosporium avenae</u> Eidam	1	1	1
	Crown rust	<u>Puccinia coronata</u> Cda. f. sp. <u>avenae</u> Eriks. & E. Henn.	3	3	

Table 1 (Cont'd.)

Crop	Disease	Pathogen	Distribution*		
			P.E. I.	N. S.	N. B.
Wheat	Stem rust	<u>Puccinia graminis</u> Pers. f. sp. <u>avenae</u> Eriks. & Henn.	2	3	
	Covered smut	<u>Ustilago kolleri</u> Wille	2	2	2
	Root rot	<u>Fusarium</u> spp.	1	1	1
	Yellow dwarf	Barley yellow dwarf virus	2	2	2
	Greyspeck	Manganese deficiency	2		
	Blast	Unknown	1	1	1
	Spot blotch	<u>C. sativus</u>	1	1	1
	Speckled leaf blotch	<u>Septoria avenae</u> Frank F. sp. <u>triticea</u> Th. Johnson	3	3	3
	Mildew	<u>Erysiphe graminis</u> DC. f. sp. <u>tritici</u> Em. Marchal	1	1	1
	Glume blotch	<u>Septoria nodorum</u> ** Berk.	3	3	
	Leaf rust	<u>Puccinia recondita</u> Rob.	1	1	1
	Stem rust	<u>Puccinia graminis</u> Pers. f. sp. <u>tritici</u> Eriks & E. Henn.	3	3	3
	Loose smut	<u>Ustilago tritici</u> (Pers.) Rostr.	1	1	1
	Scab	<u>Fusarium</u> spp.	1	1	1
	Root rot	<u>Fusarium</u> spp. <u>C. sativus</u>	1	1	1
	Head discoloration	<u>Alternaria</u> spp. <u>C. sativus</u> <u>E. graminis</u>	1	1	1
	Nutritional disorders	Unbalanced fertilization	3	3	3
Rye	Spot blotch	<u>C. sativus</u>	1	1	1
	Ergot	<u>C. purpurea</u>	1	1	1
	Scab	<u>Fusarium</u> spp.	1	2	

* 1 - Found in all areas examined.

2 - Found in the majority of areas but not necessarily in all fields.

3 - Traces found in some areas.

** tentative identification.

Acknowledgments

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DISEASES OF ALFALFA AND OTHER FORAGE LEGUMES IN SASKATCHEWAN IN 1968 AND 1969¹

Howard Harding²

Introduction

Approximately 1,642,000 acres of alfalfa are grown in Saskatchewan (1). Of this, some 677,000 acres are devoted to alfalfa and alfalfa-grass hay production, 960,000 acres to pasture, and about 5,000 acres to alfalfa seed production. Most of this acreage is located in the north-eastern part of the cultivated area of the province.

Materials and methods

The survey was devoted primarily to leaf and stem diseases of alfalfa. However, an attempt was also made to assess the occurrence of other diseases of alfalfa, sweet clover and red clover. The surveys were conducted in mid-June and mid-August in 1968 and in mid- to late-August in 1969. Representative fields in the north eastern crop district (Fig. 1) were scored for the percentage of plants affected and disease severity. Rating was done subjectively on a scale of 1-10 where 1 represents a trace of the disease and 10 a severe outbreak.

Results

Leaf and stem diseases of alfalfa

In 1968 (Table 1) the most obvious disease was black stem, caused by *Phoma*

medicaginis Malbr. & Roum. It was found in all fields with most fields showing slight to moderate infection on about 50% of the plants. Stem blackening was the most noticeable symptom of the disease but leaf spotting was abundant also. Common leaf spot, caused by *Pseudopeziza trifolii* f. sp. *medicaginis-sativae* Schmiedeknecht, was found in most fields but only in slight amounts. Yellow leaf blotch, caused by *Leptotrochila medicaginis* (Fckl.) Schüepp, was noted only occasionally and did not seem to be of any importance.

In 1969 (Table 2) the picture was quite different. Black stem was rarely found in more than slight amounts and the incidence of leaf spotting was higher than that of stem blackening. Yellow leaf blotch was very common and appeared to be causing serious defoliation at some locations. Common leaf spot again appeared innocuous with the possible exception of a large acreage at location 12 and a breeder seed plot of variety Grimm at Saskatoon.

Table 1. Alfalfa disease survey 1968

Location No.	Black Stem		Common Leaf Spot	
	% Plants affected	Disease rating*	% Plants affected	Disease rating*
1	75	4	25	1
2	50	2	25	4
	25	7		
3	25	2	25	2
	25	5		
4	50	5	15	2
6	75	4	5	2
7	5	1	0	
8	75	2	5	2
8	100	2	25	2
9	50	4	25	2
10	75	5	25	1
10	50	2	25	2
	25	7		
12	100	2	25	5
14	75	4	25	2
18	75	3	25	1

* Where 0 = no disease and 10 = severe disease.

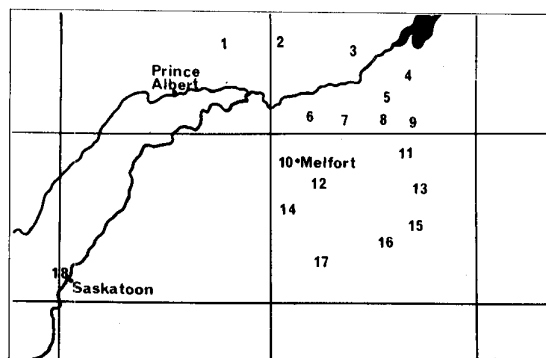


Figure 1. Locations of fields surveyed in alfalfa disease survey in 1968 and 1969.

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² Plant Pathologist.

Table 2. Alfalfa disease survey 1969

Location No.	Black Stem		Yellow Leaf Blotch		Common Leaf Spot	
	% Plants affected	Disease rating	% Plants affected	Disease rating	% Plants affected	Disease rating
5	5	1	75	4	0	
9	50	1	75	4	25	1
10	50	1	50	4	0	
			25	7		
10	25	1	100	4	0	
	75	5				
10	75	4	75	7	5	1
10	25	1	25	1	0	
11	5	1	0		0	
12	50	1	15	2	50	2
					25	4
13	50	1	75	3	0	
			15	7		
14	50	1	75	2	50	2
	5	5	25	5	5	5
15	5	1	5	1	0	
16	25	1	50	2	0	
17	25	1	50	2	50	1
18	75	2	5	2	75	2

The difference between the two years probably reflects the different climatic conditions (Table 3). The summer of 1968 was unusually cool and wet, while in 1969 many fields of alfalfa showed signs of stress due to lack of moisture.

Other diseases

Bacterial wilt, caused by Corynebacterium

insidiosum (McCull.) Jensen, seems well controlled by the use of resistant varieties and was rarely seen. In 1968 a few infected plants in an old stand of Grimm alfalfa were noted at location 8. Also in 1968 crown bud rot, caused by a combination of Rhizoctonia solani Kuhn, Fusarium roseum Lk., and Phoma medicaginis, was recorded in fields of alfalfa under irrigation in south-western Saskatchewan.

Other forage legumes

In both 1968 and 1969 the sweet clover fields surveyed seemed relatively disease-free. Gray stem canker, caused by Ascochyta caulicola Laub., was commonly found but usually in slight amounts.

The most obvious disease of red clover in 1969 was powdery mildew, caused by Erysiphe polygoni DC ex Merat. Most plants were moderately infected, some severely. In both 1968 and 1969 small amounts of northern anthracnose, caused by Kabatella caulivora (Kirchn.) Karak., and black stem were recorded on red clover.

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Table 3. Mean temperature and total rainfall recorded at Melfort, April-September 1968 and 1969

Month	Temperature (°F)		Rainfall (inches)	
	1968	1969	1968	1969
April	38	40	0.42	0.24
May	49	49	1.26	0.57
June	58	54	1.18	0.68
July	60	62	2.85	3.05
August	56	64	3.34	1.23
September	53	53	1.49	1.70

THREE DISEASES OF CORN (ZEA MAYS) NEW TO ONTARIO: CRAZY TOP, A PHYLLOSTICTA LEAF SPOT, AND EYESPOT

L. F. Gates and C.G. Mortimore¹

CRAZY TOP (*Sclerophthora macrospora* (Sacc.) Thirum., Shaw and Narasimhan)

This downy mildew has been reported by Conners (1) to occur on wheat (*Triticum aestivum* L.) in Canada, but it has not been recorded on corn (*Zea mays* L.) in this country. The only previous occurrence of crazy top in Ontario known to the authors was in 1946, when it was seen in one field in Kent County by Mr. Earl Thompson of Pioneer Seed Company (personal communication). However, in 1968 the disease occurred unexpectedly in several areas of Essex and Kent Counties. In an August survey of 22 fields near Harrow known to have had flooded areas in the spring, 5 fields contained infected plants. In all, 10 cases were reported in 1968 in a 6-mile radius of Harrow, and 2 cases were reported in Kent County.

Infected plants were stunted, tillered, and often crooked, with disorganized vascular systems. Leaves were thickened, leathery, and corrugated, with a characteristic striping of light green lines, which were frequently longer near the midrib than at the edges of the leaf (Fig. 1A). The tassels, or parts of the tassels, developed leafy proliferations of the floral parts, and some plants developed multiple ears which were small and largely barren (Fig. 1B).

Invasion of the plant is reported to occur when the soil is flooded or waterlogged after seed germination and before the plants are about 6 inches high (6). In the Harrow area, where most corn is sown during the second and third weeks of May, 4.01 inches of rain were recorded on May 26-27, and many fields were flooded. The disease was clearly associated with the flooded areas; infected plants

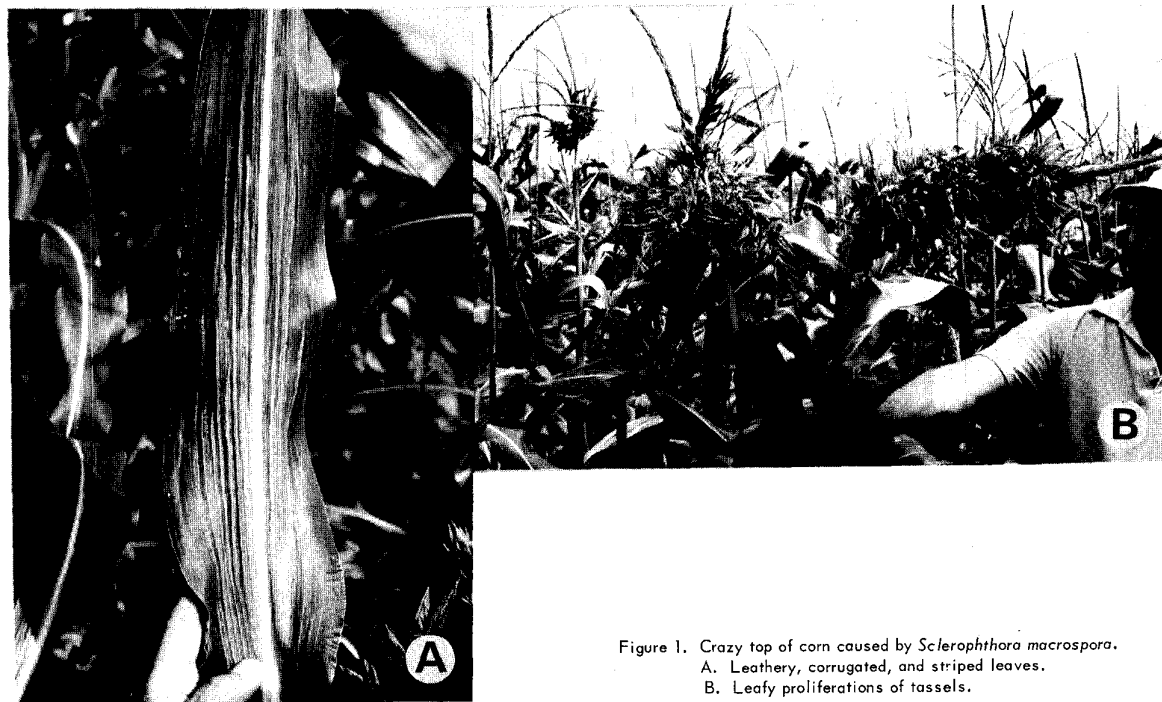


Figure 1. Crazy top of corn caused by *Sclerophthora macrospora*.
A. Leathery, corrugated, and striped leaves.
B. Leafy proliferations of tassels.

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occurred around patches of flood-damaged corn or along areas where surface-water had flowed. Usually only a few infected plants were found, but some fields had areas with many infected plants. Crazy top was unusually prevalent in Indiana and in some adjacent states in 1968 under similar weather conditions (8).

It is of interest that the disease should appear in so many fields in areas where it has not been recorded before. Possibly, long-dormant oospores were the source of inoculum, but since the flooded areas frequently adjoined grass edges spores may have floated in from perennial grass hosts.

Some plants were seen in which leaf symptoms were recognized but the tassels were not affected, and it is possible that under wet conditions corn plants are invaded to a limited extent more frequently than observations of typical symptoms would suggest.

LEAF SPOT CAUSED BY A *PHYLLOSTICTA* SP.

In 1967 this disease was noted on corn in southwestern Ontario between Brantford and Chatham. In 1968 it was observed in the same general area and also in Essex County, and in 1969 was seen from Essex County to Northumberland County. Usually scattered infections occur mainly on the lower and middle leaves in August and September, but on occasions the spots coalesce and severely damage the leaves. Most of 56 hybrids were affected in a performance trial near Brantford in 1969.

Lesions are narrow, about 4-6 times as long as wide, and many fall typically within the range 1-4 cm x 0.2-0.5 cm; they are fawn-colored, frequently with a dark border (Fig. 2A). Pycnidia 80-150 μ in diameter develop, often in rows between the veins (Fig. 2B). Spores produced on leaves or on agar culture media are unicellular, ellipsoid to cylindrical, contain two or more oil-

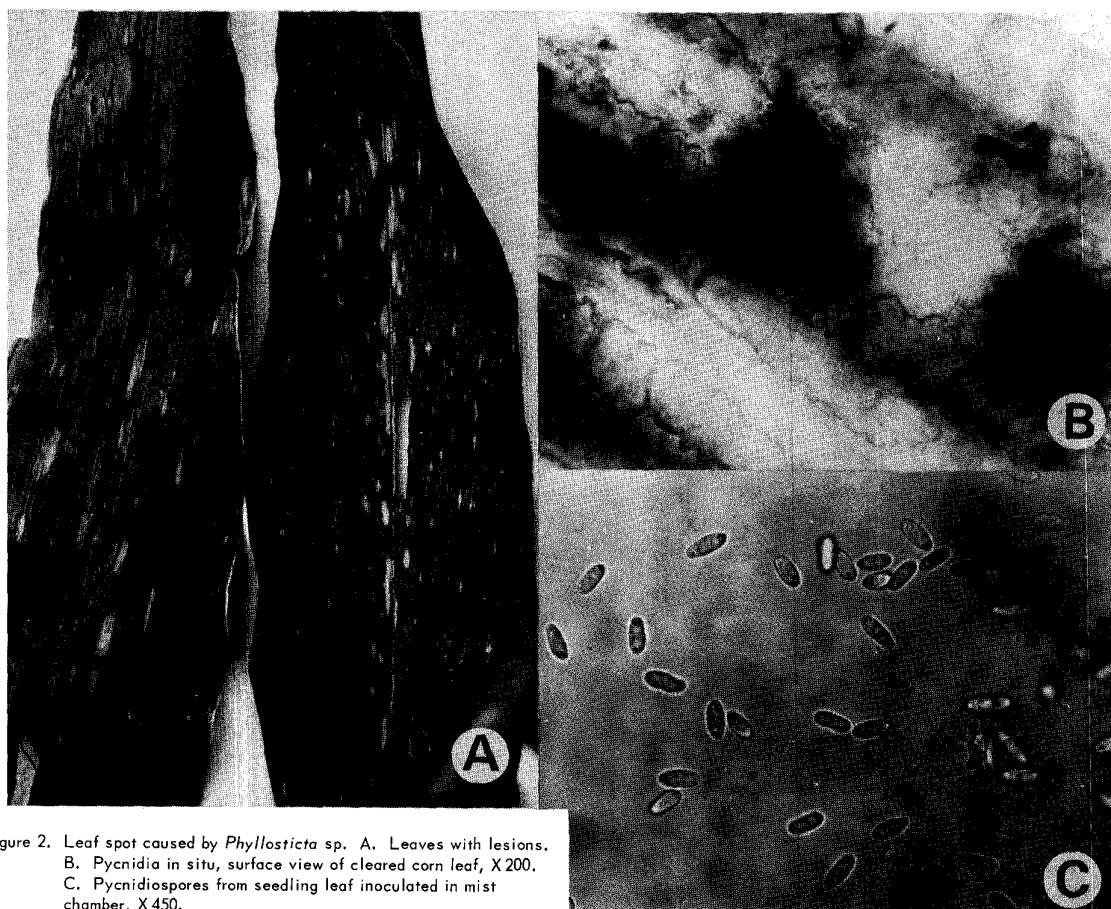


Figure 2. Leaf spot caused by *Phyllosticta* sp. A. Leaves with lesions. B. Pycnidia in situ, surface view of cleared corn leaf, X200. C. Pycnidiospores from seedling leaf inoculated in mist chamber, X450.

drops, and are generally $9.6-14.4 \mu \times 3.2-5.3 \mu$ (Fig. 2C). These are larger than the spores described by Stout for *Phyllosticta zeae* (4), though they match in size the spores of *Ascochyta zeae*, which Stout describes as "obscurely uniseptate, the septum often apparently lacking". Cross-walls have only occasionally been seen in our material.

The wide distribution of this disease suggests that it is normally present at levels that are not noticed, but that conditions have favored its appearance in 1967, 1968, and 1969. The fungus behaves as a weak pathogen in that, after artificial inoculations of greenhouse corn plants, it infects leaves of young seedlings and older leaves of larger plants, but mature leaves in good condition are attacked only slowly. In the field the fungus attacks mainly older and senescing leaves. In the last 3 seasons, conditions in some areas of southwestern Ontario have been somewhat adverse for corn due to dry periods in the late summer, sometimes following heavy spring rains, which seem to have restricted root development. These conditions have resulted in shortages of moisture and nutrients in late summer, with a consequent early maturation and

senescence of leaves that would favor unusually widespread attack by the fungus. In 1968, leaf spots due to species of *Phyllosticta* were also widespread in several states of the U.S.A. (3,5). The use of large populations of plants places greater stress on individual plants, also favoring the disease. Corn monoculture, which is becoming common in southwestern Ontario, is likely to increase pathogen inoculum. Experiments at Harrow indicate that stalk rot resistance and yield depend largely on the integrity of the leaves above the ear, and so far in Ontario these leaves generally are only lightly affected by the disease.

EYESPOT (*Kabatiella zeae* Narita and Hiratsuka).

This disease was observed on corn in performance trials near St. Thomas and Brantford in 1967-69. Its incidence has been light, except on occasional leaves.

Spots are circular, brown, 1-2 mm in diameter, including a dark brown or reddish brown margin (Fig. 3A). Sometimes a translucent ring surrounds the spots. Conidiophores about 30μ long emerge in

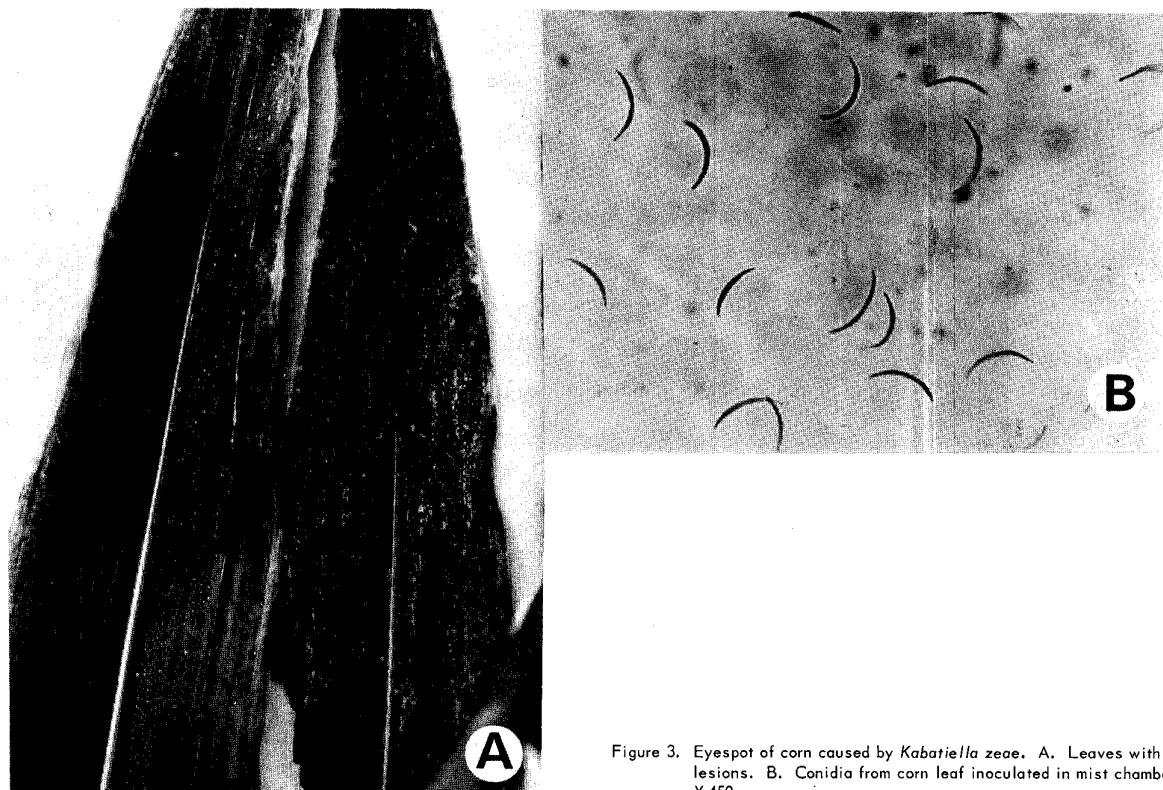


Figure 3. Eyespot of corn caused by *Kabatiella zeae*. A. Leaves with lesions. B. Conidia from corn leaf inoculated in mist chamber, X 450.

groups through the stomata and produce distinctive sickle- or crescent-shaped spores which measure $16-29 \mu \times 2.2-3.2 \mu$ (Fig. 3B). The original description of this disease and pathogen was made in Japan (2), and it was recorded over a wide area of north-central U.S.A. in 1968 (5,7).

The fungus appears to be a weak pathogen, only slowly attacking corn leaves after artificial inoculation in a mist chamber, and attacking only older leaves in the field. Lesions sometimes occur along folds in the leaves along which spores have evidently been washed by rain or dew.

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NEMATODES ASSOCIATED WITH TOMATO AND CUCUMBER GREENHOUSE SOILS IN ESSEX COUNTY, ONTARIO

P. W. Johnson and L. W. D. Boekhoven¹

Abstract

Six stylet bearing nematode genera, *Aphelenchoides*, *Aphelenchus*, *Helicotylenchus*, *Meloidogyne*, *Pratylenchus* and *Tylenchus* were detected in tomato and cucumber greenhouse soils in Essex County. The root-knot nematode, *Meloidogyne* sp., detected in approximately 42% of the samples, was the most economically important nematode encountered. The lesion nematode, *Pratylenchus* sp., occurred in 10% of the samples and appears to be of only minor importance. The spiral nematode, *Helicotylenchus* sp. was found in only one sample and poses no problem in the greenhouse. *Aphelenchoides*, *Aphelenchus* and *Tylenchus*, predominantly known as fungus feeding genera, are of no economic importance.

Introduction

Tomato (*Lycopersicon esculentum* Mill.) and cucumber (*Cucumis sativa* L.) are the most important greenhouse vegetable crops grown in southwestern Ontario. Two crops per year are usually grown, with tomato and cucumber frequently being rotated. Many growers have found nematodes to be a recurring problem in their houses. A survey of greenhouses in Essex County was conducted in 1969 to ascertain the extent of the nematode problem.

Methods

Soil samples were obtained in July, August and September of 1969 from 97 greenhouses throughout Essex County, representing approximately 25% of the vegetable-growing operations. Greenhouses were chosen randomly with no previous knowledge of their cropping history or nematode problems.

In each house six soils cores were taken at random with a 2.54 cm diameter soil tube from two depths, 0-15 and 15-30 cm. Samples from the same depth in each house were combined and thoroughly mixed, and two 50 g sub-samples from each were extracted, using a modified Baerman pan method (3). Stylet bearing nematodes in the extract were counted and identified to genus.

Cropping and treatment histories over the past 3 years were obtained for each house at the time of sampling. The data were compiled according to the crops that were grown in the previous 3 years, i.e. cucumbers only, tomatoes only, both cucumbers and tomatoes.

Results and discussion

Frequency of occurrence

Six stylet bearing nematode genera, *Aphelenchoides* Fisher, 1894, *Aphelenchus* Bastian, 1865, *Helicotylenchus* Steiner, 1945, *Meloidogyne* Goeldi, 1887, *Pratylenchus* Filipjev, 1936 and *Tylenchus* Bastian, 1865, were found in the extracts.

The root-knot nematode, *Meloidogyne* sp. was the most commonly encountered nematode of economic importance. It occurred in 42% of the 0-15 cm samples and in 38% of the 15-30 cm samples (Table 1). The lesion nematode *Pratylenchus* sp. occurred in 10% and 6% of the samples, respectively; while only one specimen of the spiral nematode *Helicotylenchus* sp. was isolated, from one sample at the 15-30 cm depth. The latter nematode does not appear to pose any problem in area greenhouses.

The most frequently encountered nematode was *Tylenchus* sp., which occurred in 46% of the 0-15 cm samples and 38% of the 15-30 cm samples. It was not possible to establish the *Tylenchus* sp. extracted from the soil samples on tomato or cucumber plants in steam sterilized soil. It was therefore probably a fungus-feeding species like most other members of the genus (1,2).

Aphelenchoides sp. and *Aphelenchus* sp. were infrequently isolated and like *Tylenchus* sp., are probably fungus feeders.

Population density

The root-knot nematode, *Meloidogyne* sp. had the greatest population density with an overall mean in infested samples of 245 and 142 nematodes/100 g of soil at the 0-15 and 15-30 cm depths, respectively (Table 2). This coupled with its high frequency of occurrence and severe damage to both tomato and cucumber makes *Meloidogyne* sp. the most economically important nematode in area greenhouses.

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Table 1. Occurrence of six nematode genera in tomato and cucumber greenhouse soils

Crop and depth of soil sample	No. of samples	Percentage of samples containing the indicated genus					
		<u>Aphelenchoides</u>	<u>Aphelenchus</u>	<u>Helicotylenchus</u>	<u>Meloidogyne</u>	<u>Pratylenchus</u>	<u>Tylenchus</u>
Cucumber (3 yr)							
0 - 15 cm	7	0	0	0	57	14	14
15 - 30 cm	7	0	0	0	57	0	14
Tomato (3 yr)							
0 - 15 cm	21	10	19	0	33	14	43
15 - 30 cm	21	5	10	0	33	10	33
Tomato and cucumber							
0 - 15 cm	69	7	1	0	44	9	51
15 - 30 cm	69	6	0	1	38	6	42
Total occurrence							
0 - 15 cm	97	7	5	0	42	10	46
15 - 30 cm	97	5	2	1	38	6	38

Table 2. Population density of five nematode genera in samples of 97 tomato and cucumber greenhouse soils

Crop and depth of soil sample	<u>Aphelenchoides</u>			<u>Aphelenchus</u>			<u>Meloidogyne</u>			<u>Pratylenchus</u>			<u>Tylenchus</u>		
	No. of samples	Range	Mean	No. of samples	Range	Mean	No. of samples	Range	Mean	No. of samples	Range	Mean	No. of samples	Range	Mean
Cucumber (3 yr)															
0 - 15 cm	0			0			4	2-286	87	1			1	1	6
15 - 30 cm	0			0			4	2-156	44	0			1		3
Tomato (3 yr)															
0 - 15 cm	2	2-10	6	4	2-15	7	7	2-5000	1168	3	22-460	177	9	1-164	50
15 - 30 cm	1		1	2	1-6	4	7	2-2500	507	2	2-94	48	7	1-82	23
Tomato and cucumber															
0 - 15 cm	5	1-581	168	1		4	30	1-670	51	6	2-8	4	35	1-200	37
15 - 30 cm	4	4-74	38	0			26	1-504	59	4	1-50	16	29	1-210	25
Total mean density**															
0 - 15 cm	7		122	5		7	41		245	10		56	45		39
15 - 30 cm	5		30	2		4	37		142	6		28	37		24

* Range and mean number of nematodes/100 g samples.

** Total mean density based on total number of positive samples.

The lesion nematode, Pratylenchus sp. had population means of 56 and 28 nematodes/100 g of soil, from infested samples, at the 0-15 and 15-30 cm depths, respectively. Its lower population density compared with Meloidogyne sp., plus its lower frequency of occurrence and less severe damage to tomato and cucumber make Pratylenchus sp. of only minor importance in area greenhouses.

The treatment histories of the houses samples showed that 89% of the growers had applied a nematocide at least once in the past 3 years and 49% had applied a nematocide every year. This, coupled with the high frequency of nematode occurrence in this survey, suggests that additional research may

be required in the area of greenhouse nematode control.

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A NEWLY RECORDED VIRUS DISEASE OF SUGAR BEET IN BRITISH COLUMBIA

H. R. MacCarthy¹

Abstract

During a field study of virus vectors a mild virus disease of sugar beets (*Beta vulgaris*) was encountered. About 60% of the plants were infected. On the evidence of its relations with the aphid vector, *Myzus persicae* (Sulz.), and of its plant host range and symptoms, the virus was identified as beet western yellows. This disease has become widespread in the U.S. northwest within the last 12 years.

Introduction

During a study of the epidemiology of potato leaf roll virus (PLRV) (13) it was found that several hundred acres of sugar beets (*Beta vulgaris* L.) grown for seed were a prime local source of potential vectors of this virus, i.e. winged green peach aphids, *Myzus persicae* (Sulz.). The beet fields were closely grouped about the main, southerly, mouth of the Fraser River. The beet seed is normally sown in August and the young plants are left in the fields to overwinter. In this mild coastal climate they resume growth early in spring and the seed is harvested in July and August. A backlog of seed is stored to allow for unusual winters when, as in 1968-69, most of the plants are killed by frost.

The upshot of this work was to establish the presence in sugar beets of a virus disease not hitherto recorded in Canada.

Materials and methods

During the summer of 1968 aphids collected from sugar beet plants in the field were given test feedings on *Physalis floridana* Rydb. to ascertain if any were viruliferous for PLRV. The technique has been described elsewhere (4,5).

To determine whether the cause of symptoms that developed on *P. floridana* was a mild strain of PLRV, all apparently infected plants were re-colonized with non-viruliferous *M. persicae*. Late instar nymphs or apterous adults were used. After 5 days the aphids were transferred in groups of five to the sprouts of 35 virus-free (8) 'Netted Gem' or 'White Rose' potatoes (*Solanum tuberosum* L.). After 24 hr the aphids were removed, tested for infectivity on *P. floridana*, and destroyed. The potato plants

were kept free from aphids in a greenhouse for 9 to 15 weeks. Stem tips from each were then indexed for PLRV on *P. floridana* by aphid transfers, as described (4,5).

To help identify the virus from sugar beet, plants representing 12 species and seven families were tested for susceptibility. The plants tested were *Chenopodiaceae*: garden beet (*Beta vulgaris* L. 'Detroit Dark Red'), spinach (*Spinacea oleracea* L. 'King of Denmark'), *Chenopodium amaranticolor* Coste & Reyn.; *Compositae*: zinnia (*Zinnia elegans* Jacq. 'Cactus-flowered Mix'), lettuce (*Lactuca sativa* L. 'Pennlake'); *Leguminosae*: pea (*Pisum sativum* L. 'Lincoln'); *Cruciferae*: broccoli (*Brassica oleracea* var. *italica* Plenck 'Italian Green Sprouting'), cauliflower (*Brassica oleracea* var. *botrytis* L. 'Snowball'), Chinese cabbage (*Brassica pekinensis* Rupr. 'Petsai'); *Caryophyllaceae*: mouse-ear chickweed (*Cerastium vulgatum* L.); and *Polygonaceae*: sheep sorrel or sourgrass (*Rumex acetosella* L.). Groups of 15 plants of each species were grown in soil in 5-inch clay pots. Ten plants of each group were infested at the first true leaf stage by three viruliferous aphids, two were fed upon by non-viruliferous aphids and three were kept as aphid-free controls. Test transmissions were made subsequently by allowing clean aphids to feed for 3 to 5 days on excised tips of the plants and then on *P. floridana* indicators.

Seventy seedlings of shepherd's purse, *Capsella bursa-pastoris* (L.) Medic., and 15 of *Claytonia perfoliata* Donn. were tested as indicators. Sugar beet plants from the field were tested in November, 1968, and in March, 1969.

Results

A virus was transmitted to *P. floridana* from 49 of 83 mature field-grown sugar beet plants tested in mid-August, 1968. The symptoms of stunting and mild chlorosis (Fig. 1) were indistinguishable from those caused

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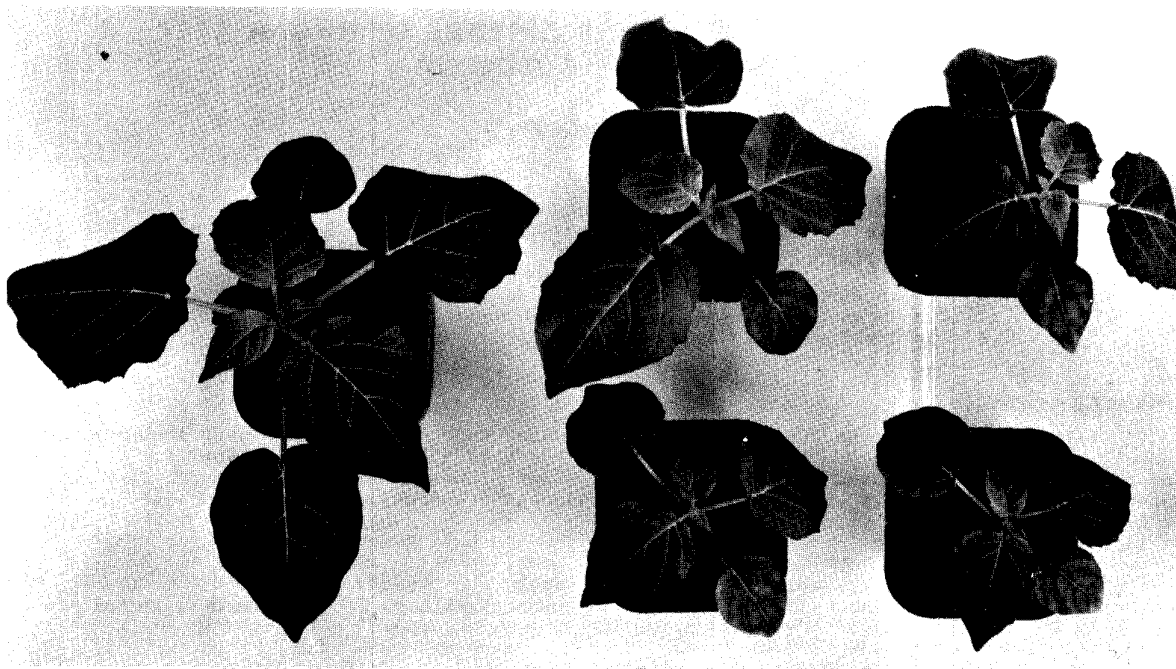


Figure 1. Symptoms in *Physalis floridana* of a virus from sugar beet. Left: healthy plant. Right: four infected plants.

on *P. floridana* by mild PLRV (12) and by mild chlorosis virus (MCV) (6). A third possibility was beet western yellows virus (BWV), but at that time the susceptibility of *P. floridana* to this virus was unknown.

The virus from sugar beet was transmitted readily by *M. persicae* but was not rub-transmissible, nor was it seed borne in sugar beet or *P. floridana*. All attempts to transmit the virus to potato with aphids proven to be viruliferous were unsuccessful. A total of 180 *P. floridana* indicators were used to index the 35 inoculated potatoes.

From the host range data given in Table 1 the virus was identified as almost certainly BWV. This identity is supported by other evidence, such as successful transmission of the virus into and from radish (*Raphanus sativus* L.). In addition the symptoms produced on garden beet, zinnia, and shepherd's purse agree with published descriptions; also the virus was retained for life once an aphid was viruliferous; and maximum transmission rates were obtained with acquisition feedings exceeding 48 hr and transmission feedings of 24 hr. All these characteristics agree with those reported by Duffus for BWV (1,2). The virus was not lost by nymphal aphids when they molted.

Shepherd's purse was inferior to *P. floridana* as an indicator. *M. persicae* failed to pick up the virus from several plants of shepherd's purse that had the characteristic symptoms (1) of stunting and swollen, purplish leaves. *C. perfoliata* was also inferior to *P. floridana*, both in ease of handling and in production of positive symptoms.

All six seedling sugar beet plants indexed in late November, 1968 were infected, and of 54 plants indexed in March, 1969, 33 (61%) were infected.

Discussion

On the basis of symptomatology, host range, aphid transmission, and geographic distribution, there is little doubt but that the sugar beet virus discussed here is beet western yellows virus. This is the most common of the components comprising the beet virus yellows complex. Since 1960 Duffus has clarified the host, vector, and economic relations of BWV in California in a series of papers, and Russell (7) in England almost simultaneously did the same for sugar beet mild yellowing virus. The two appear to be very closely related.

Table 1. Transmission by Myzus persicae of a virus from sugar beet to various test plants and from test plants to Physalis floridana under greenhouse conditions, using a 5 day acquisition access time and a 24 hr inoculation access time with three aphids per plant

Test plant	No.* infected	Leaf symptoms on test plant	Transmission** to <u>Physalis floridana</u>
Garden beet	10	Rolling, twisting, yellowing	30
Spinach	10	None	29
<u>Chenopodium amaranticolor</u>	7	None	13
Zinnia	10	Faint yellow mottle	27
Lettuce	9	None	19
Pea	8	None	19
Broccoli	7	None	9
Cauliflower	6	Some faint yellow mottle	9
Turnip	5	None	8
Chinese cabbage	1	None	1
Mouse-ear chickweed	0	None	0
Sheep sorrel	0	None	0

* Number of plants infected of 10 inoculated.

** Number of plants infected of 30 inoculated.

The spread of the disease from California and the problems associated with it in seed and sugar crops of beets have been recorded by Duffus in Oregon (2), by Hills et al. in Arizona (3), by Wallis in Washington (10,11), and by Simpson in Idaho (9). It has become widespread in the northwest only in the last 12 years (10). It is almost certain that, although not previously recorded in British Columbia, the disease has been present here for several years.

BWYV is of considerable economic importance. It is the cause of June yellows of lettuce and radish yellows in California (1,2). It has been shown to reduce the yield of lettuce seed by about 40% (3), the yield

of sugar beet seed by 13-15% (2,3) and the yield of sugar by 19% (2). These losses are about doubled when BWYV is combined with the more damaging beet yellows virus.

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BRIEF ARTICLES

EFFECT OF POSTHARVEST INFECTION OF POWDERY MILDEW ON YIELD OF THE STRAWBERRY CULTIVAR NORTHWEST

Jack A. Freeman¹ and H. S. Pepin²

Powdery mildew caused by *Sphaerotheca macularis* (Wallr. ex Fr.) P. Magn. is usually present during the months of August and September on strawberry plants grown in the Pacific Northwest. 'Northwest', the most important cultivar grown in the area, is particularly susceptible to the disease.

There are no published data on the effect of postharvest mildew on the following year's yield and hence no real evidence that postharvest mildew is harmful. Growers, following current recommendations (1), carry out a rigorous and expensive control scheme involving the application of sulfur at intervals of 14 days throughout the period of most severe infection. The purpose of this investigation was to determine whether postharvest spraying for powdery mildew control in 'Northwest' strawberries is necessary.

Methods

Various fungicide treatments were evaluated at Abbotsford, B.C., in 1967 for the control of postharvest infection of powdery mildew on the Northwest cultivar (2). The experiment was laid out in a randomized block designed with six replicates. Each plot consisted of a single 30-ft row. The 1967 season was particularly favorable for the development of this disease, with unsprayed foliage showing over 90% infection (Table 1). The most effective treatment of those tested was benomyl at 0.25 lb/acre applied five times at 14-day intervals beginning July 21. This treatment reduced the average mildew infection, based on percentage of leaf area affected, to as low as 19%. The other treatments gave control somewhere between the two extremes. Therefore, the 1967 treatments provided an ideal set of plots with plants showing a full range of infection.

Results

Since the sprays usually recommended for mildew and fruit rot control were applied in

the spring, it was felt that any yield reduction in the plots would be due to mildew infection the previous summer. Since these reductions did not occur (Table 1), the results suggest that spraying for postharvest powdery mildew control on 'Northwest' strawberries is an unnecessary practice in the Pacific Northwest.

Table 1. Effect of postharvest infection of powdery mildew in the strawberry cultivar 'Northwest' on the following year's crop

Fungicide applied in 1967	Rate (lb/acre)	Number of sprays*	Powdery mildew rating Sept. 20, 1967 (%)**	Total yield 1968 (lb/plot)
Unsprayed			91 a [†]	38.6 a
Benomyl	0.25	1	90 a	38.1 a
Benomyl	0.25	3	65 b	34.1 a
Benomyl	0.25	5	19 c	36.9 a
Sulfur	3.6	3	73 ab	39.3 a
Sulfur	3.6	5	25 c	39.0 a
Dinocap	0.75	3	61 b	34.4 a
Dinocap	0.75	5	32 c	36.9 a

* Sprays applied as follows in 1967:

- 1 - July 21;
- 3 - July 21, Aug. 3, Aug. 18;
- 5 - July 21, Aug. 3, Aug. 18, Sept. 1, Sept. 15.

** Percentages were transformed for statistical analysis.

[†] Means not followed by the same letter are significantly different at the 5% level (Duncan's Multiple Range Test).

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ROOT ROT OF PEAS IN PRINCE EDWARD ISLAND IN 1969¹

H. Winston Johnston and J.A. Cutcliffe²

Production of vegetables for processing has become an important segment of agriculture in Prince Edward Island. The major crop produced for this rapidly expanding industry is peas. Land area devoted to this crop has steadily increased during the past ten years and over 5,500 acres were grown for freezing and canning in 1969.

Prior to 1969, disease losses were relatively minor and were usually caused by *Ascochyta* spp. (1). In 1969, fields were observed that lacked uniformity of stand and had areas which could easily be distinguished by a general lack of vigor. Plants in these areas exhibited symptoms of severe root rot and from their roots a biotype of *Fusarium oxysporum* was isolated. Soil dilution plate counts revealed from 2,000 to 3,000 *Fusarium* propagules per gram of undried soil from areas where plants exhibited slight to severe symptoms, respectively.

The disease symptoms were confined to about 600 acres near Bedeque in south-central P.E.I. The most severe losses occurred in one 70 acre field where yields obtained were 50% of the average for the area. The estimated overall yield reduction for the 600 affected acres was 20%. Peas have been produced in this area for a longer period than in other areas of the Province and during the past 4 to 5 years some of these fields have been cropped to peas continuously. There was no evidence of root rot in other pea producing areas where there are very few fields that have had more than 3 crops of peas during the past 10 years.

Presumably, the lack of crop rotation has enhanced the inoculum potential of the causal organisms. To minimize losses from root rot organisms, it appears that peas can no longer be produced under a monoculture cropping system in Prince Edward Island.

Acknowledgments

The authors are indebted to Dr. O.T. Page and Mr. A.A. Qureshi of the University of New Brunswick for confirming the identity of the *Fusarium* isolate.

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² Plant Pathologist and Horticulturist, respectively.

CHAR SPOT ON WHEATGRASSES¹

J. Drew Smith²

Char spot caused by *Septogloeum oxysporum* (Sacc.) Bromm. and Rouss. has been found on wheatgrasses, *Agropyron* spp., in Saskatchewan every year since 1965. Not until 1969 was it seen in sufficient amounts for comparison to be made of the disease reactions of strains. In a dryland test at Saskatoon on 17 July, ratings were made on 14 strains of slender wheatgrass (*A. trachycaulum* (Link) Malte) and on two strains of crested wheatgrass (*A. cristatum* [L.] Gaertn.). The design of the test was a 4 x 4 balanced lattice with 6-row plots 20 ft. long. Ratings were made on 10 tillers at random in the middle rows of each plot (Table 1).

Table 1. Disease ratings for char spot on strains of *A. trachycaulum* and *A. cristatum*

Species and strain	Source	Average rating*/plant	
<u>A. cristatum</u>			
'Summit'	Sask.	0.15	a [†]
'Fairway'	Sask.	0.16	
<u>A. trachycaulum</u>			
1587	Sask.	0.27	b c d c
1439	Sask.	0.29	
1710	U. S. S. R.	0.35	
1632	Sask.	0.37	
1142	Sask.	0.47	
1708	Hungary	0.48	
1358	Sask.	0.51	
1554	Sask.	0.52	
1181	Sask.	0.53	
'Primar'	U. S. A.	0.66	
'Revenue'	Sask.	0.72	
1466	Sask.	0.74	
1617	Sask.	0.77	
1294	Sask.	1.34	

* On a 0-4 scale where 0 is no disease and 4 very severe disease.

† Duncan's multiple range test at the 5% level of significance.

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² Plant Pathologist.

None of the wheatgrass strains were severely affected by the disease (Table 1). Both crested wheatgrass varieties 'Summit 1' and 'Fairway', showed very slight infection, considerably less than the slender wheatgrass varieties 'Primar' and 'Revenue'. Strain 1294 was significantly less resistant than the latter two varieties. 'Revenue', scheduled to be released in 1969-70 by the Canada Department of Agriculture, had been selected for a much higher leaf-to-stem ratio and in vitro digestibility than 'Primar'³. These differences apparently were not reflected in disease resistance.

The 'Revenue' variety was also grown under irrigation at Saskatoon for seed production. Leaves were heavily damaged by *S. oxysporum*. Ratings were made on 110 tillers at random in a 1-acre plot on the same day as those on the replicated dryland test. The average rating was 2.77 (Table 1) on the irrigated plot. Irrigation may prove useful in the evaluation of the new wheatgrass varieties for resistance to this disease by accentuating differences in resistance.

³ By W.L. Crowle of the Saskatoon Research Station to whom I am indebted for information.

SNOW MOLD ON LAWNS IN SASKATOON¹

J. Drew Smith²

Snow mold caused by an unidentified low-temperature basidiomycete was common on domestic lawns in Saskatoon following snow melt in the spring of 1969. In some severe cases, killed areas of grass were still visible in fall. A survey was made of the incidence of the disease on domestic front lawns in early May 1969 along streets where the age of lawns could be estimated. Certain environmental factors also were noted. Most of the lawns were composed of bluegrass, *Poa pratensis* L., alone or of a mixture of bluegrass and creeping red fescue, *Festuca rubra* L. subsp. *rubra*.

Nine hundred and thirteen lawns were rated, which was about a 4% sample of front lawns in the city. Of these, only 12% were free from symptoms of the disease (Table 1). On lawns sown in 1968, the disease occurred less frequently and was much less severe than on older lawns. This suggests either that new plants are intrinsically less susceptible to the disease, or that the inoculum of the fungus had not built up to the same extent in

the soil, on natural turfgrass litter, and on clippings in younger lawns. There was little apparent difference in incidence or severity of the disease in lawns in the other two age categories. Heavy tree shading favored the disease. Lawns associated with luxury housing had above-average incidence with a higher proportion in the very severe category. Possibly this was related to higher usage of nitrogenous fertilizer and irrigation water, producing a succulent susceptible growth (2) and to the more common use of a pure sward of the susceptible 'Merion' cultivar of *P. pratensis* (3). This cultivar is used because it forms a very attractive dark green turf under conditions of high fertility; however, it appears more susceptible to snow mold than common Kentucky bluegrass. The disease can be effectively controlled with inorganic or organic mercury fungicides applied before snowfall (1 and unpublished) but these rarely seem to be employed on lawns in the city.

Table 1. Incidence of snow mold on lawns in Saskatoon, 4 May 1969

Age or environment of lawn	Number of lawns in each rating category*					Average rating
	None	Slight	Mod.	Mod-severe	Very severe	
Sown 1968	46	27	7	1	0	0.5
2nd to 10th year	20	74	89	101	29	2.0
Older than 10 years	47	138	133	164	37	2.0
Heavily tree-shaded	9	62	87	130	28	2.3
Open	38	76	46	34	9	1.5
Luxury landscaping	0	13	41	56	21	2.6

* Rating scale: Slight = 0-10% of lawn area affected; Mod. = 11-25%; Mod.-severe = 26-50%; Very severe = 51-100%.

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² Plant Pathologist.

INCIDENCE OF COMMON AND FUSCOUS BLIGHTS OF FIELD BEANS IN SOUTHWESTERN ONTARIO - 1969

V.R. Wallen¹

In 1969, a total of 68 fields of beans were inspected in two areas of southwestern Ontario. In addition, 21 select plots grown from Idaho- or California-produced seed were also inspected. The two areas were located near the town of Hensall, 30 miles north of London, and near Chatham, approximately 50 miles west of London. The select plots were scattered throughout Ontario, as far north as Goderich and as far south as Blenheim.

In addition to field surveys, the 68 fields and some of the select plots were photographed aurally on IR color 8443 film. Samples of infected leaves and pods were taken for laboratory analysis to determine the causal organism in each infected field.

Three inspections were made in most fields, except select plots, over the period July 29 to September 10. Most fields in the Hensall area were harvested by September 10, while harvest was just initiated on this date in the Chatham area.

Hensall experienced a very dry year throughout the growing season, resulting in little spread of blight, although infection foci were present on July 29. Chatham experienced very wet conditions prior to planting, which was delayed considerably. However dry conditions prevailed after this until harvest. In general, blight was more general, although not severe, in the Chatham area. In Hensall, some fields contained a high percentage of infected seeds in the stock used for planting purposes, but environmental conditions in this area were not conducive to extensive spread of the pathogen.

Of the 68 fields inspected, 48 (70%) contained infection foci of blight. All fields inspected in the Chatham area were infected with blight, whereas 23 of the 43 fields inspected in the Hensall area were infected with bacterial blight. Of the 21 select plots inspected 4 contained blight.

Both the fuscous blight pathogen, *X. phaseoli* var. *fuscans* (Burkh.) Starr & Burkh., and the common blight pathogen, *Xanthomonas phaseoli* (E.F. Sm.) Dows., were isolated from infected leaves, pods, or seeds. In many cases both pathogens were isolated from material from the same field. Common blight was more prevalent in the Chatham area where this pathogen was isolated from 19 fields; six fields were infected with the fuscous blight organism. In the Hensall area, the common blight organism was isolated from 21 fields. Of the four select plots affected, the fuscous blight organism was isolated from all four and the common blight organism from three.

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ERRATA

OBSERVATIONS ON CRANBERRY FRUIT ROTS IN NOVA SCOTIA, 1945-55

C.O. Gourley and K.A. Harrison

Volume 49, No. 1, page 26, column 2, last line: delete Can.

COOPERATIVE SEED TREATMENT TRIALS - 1969

H.A.H. Wallace

Volume 49, No. 2, page 53: The figures in column 8 for treatment nos. 95 to 100 and LSD should appear in column 11, those in column 11 in column 9, and those in column 9 in column 8. The corrected table appears below.

Table 4. (Con't)

Treatment no.	Product name	Formulation*	Dosage (oz/bu)	Smutted heads (%)**			Barley seedling blight**		Flax	
				Wheat	Oats	Barley	Emergence (%)	Disease rating (%)	Dosage (oz/bu)	Emergence (%)
64	TF17-69	D	2.00	0.00	0.39	0.08	68.1	19.9	4.00	73.9
65	TF20-69	D	2.00	23.25	16.35	15.63	57.1	26.5	4.00	57.8
66	TF21-69	D	2.00	0.00	0.00	0.04	56.5	33.0	4.00	56.1
67	TF22-69	D	2.00	0.06	0.89	0.00	65.4	16.7	4.00	68.3
68	TF23-69	D	2.00	0.18	1.19	0.26	70.4	14.9	4.00	69.7
69	Benlate	D	2.00	0.00	0.00	0.00	55.4	31.5	4.00	58.8
70	Benlate + Arasan 70-S	SL	2.00	0.00	0.00	0.00	56.6	25.4	4.00	66.8
71	Benlate + Agrox NM	D	2.00	0.00	0.00	0.43	69.6	28.0	4.00	70.3
72	Arasan 70-S	WP	1.00	0.26					2.00	
			2.00		1.85	0.38	61.5	21.1	4.00	70.0
73	Agrox NM	D	1.00	0.00						
			2.00		0.96	0.04	68.4	18.0	4.00	71.6
74	TN-702-50-3	L	8.00	0.00	0.00	0.00	60.7	12.0	8.00	37.4
75	TN-702-50-5	L	4.00	0.00	0.00	0.00	61.7	11.6	4.00	41.1
76	TN-702-50-6	L	4.00	0.06	0.00	0.00	66.8	16.1	4.00	43.9
77	TN-702-50-6	L	8.00	0.00	0.23	0.00	59.0	11.9	8.00	33.9
78	TN-702-50-7	L	8.00	0.04	0.00	0.16	61.1	20.7	8.00	38.9
79	TN-702-50-8	L	8.00	1.20	2.79	2.30	58.6	27.3	8.00	52.0
80	TN-702-50-9	D	2.00	0.94	5.69	2.95	54.5	24.6	4.00	62.3
81	TN-702-50-9	D	4.00	0.00	2.53	0.79	59.3	29.0	4.00	52.8
82	TN-702-50-11	L	8.00	0.04	0.00	0.08	52.4	7.7	8.00	26.3
83	TN-702-50-12	L	8.00	0.40	1.81	0.10	54.1	25.7	8.00	51.6
84	Vitavax 40S	L	3.80	0.00	0.00	0.00	78.7	21.1	3.80	62.7
85	Vitavax 100	D	3.60	0.08	0.00	0.00	84.0	15.9	6.00	70.9
86	Vitavax 101	D	4.00	0.00	0.00	0.00	81.3	10.0	4.00	68.9
87	Vitavax 101	D	8.00	0.00	0.00	0.00	78.1	9.2	8.00	73.7
88	Vitavax 200	D	3.50	0.00	0.00	0.00	83.1	14.9	3.50	72.7
89	Vitavax 201	D	4.50	0.00	0.00	0.00	80.4	11.1	4.50	66.3
90	Vitavax 201	D	9.00	0.00	0.00	0.00	80.4	11.2	9.00	69.9
91	Vitavax 300	D	4.00	0.00	0.00	0.00	79.3	12.2	4.00	75.3
92	78175	WP	2.00	0.59	3.58	3.52	55.9	23.6	4.00	52.9
93	78175	WP	4.00	0.14	1.70	3.05	57.9	26.2	8.00	55.6
94	5506	WP	1.00	0.35	0.23	0.08	64.7	20.5	2.00	61.0
95	5506	WP	2.00	0.21	0.00	0.00	72.0	21.3	4.00	60.8
96	Vitavax 75W	D	2.00	0.23	0.00	0.00	75.8	14.6	4.00	65.9
97	Res-Q Dual	D	1.25	0.00					5.00	70.1
			2.50		4.64	0.20	67.3	23.8		
98	Dithane 45	D	2.00	0.00	0.69	0.00	71.4	23.8	4.00	68.1
99	Mergamma NM	D	2.00	0.00	0.54	0.14	76.9	22.7	4.00	74.4
100	Untreated check			29.75	15.77	12.17	57.8	24.9		62.8
LSD	(.05)			3.48	4.19	1.91	4.8	6.9		8.3

* Formulation code: D = dust; L = liquid; SL = slurry; WP = wettable powder

** See text