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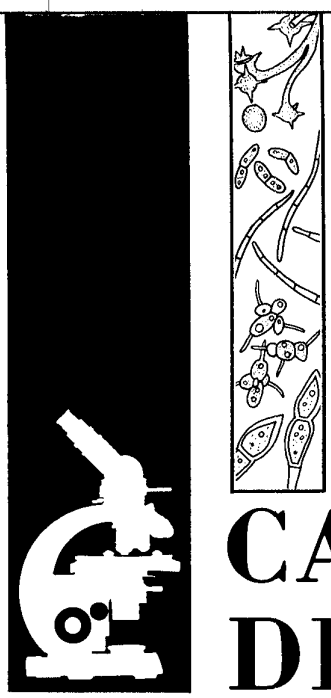
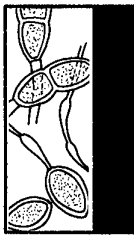


CANADIAN PLANT DISEASE SURVEY



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CANADIAN PLANT DISEASE SURVEY

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

CONTENTS

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

STRAWBERRY GREEN PETAL DISEASE IN QUEBEC AND THE MARITIME PROVINCES, 1971-72[1]

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Abstract

A coordinated strawberry green petal survey was made in first production year commercial fields of Redcoat, Sparkle and Cavalier in Quebec and the Maritime Provinces in 1971 and 1972. The general low level of infection (less than 3%) and low calculated losses, indicated that the disease was not of economic importance. The first reported presence of green petal in the Terrebonne-Deux Montagnes area of Quebec suggests a westward spread of the disease.

Résumé

Une enquête coordonnée sur la maladie du pétale vert du fraisier dans les plantations commerciales d'un an des variétés Red Coat, Sparkle et Cavalier a été poursuivie au Québec et dans les provinces maritimes en 1971 et 1972. Le peu d'infection généralement observée (moins de 3%) et le peu de pertes estimées ont mis en évidence la faible importance économique de cette maladie. Les premières observations du pétale vert du fraisier dans la région de Terrebonne-Deux-Montagnes laissent entrevoir que cette maladie se propage vers l'Quest du Québec.

Introduction

Green petal of strawberry, suspected of being caused by a Mycoplasma-like microorganism (4), was first reported in Canada in 1955 (1) and is now found generally distributed throughout the Maritime provinces and eastern Quebec. The prevalence of the disease varies from year to year and from area to area. In some years the levels of infection indicated the disease to be of major economic importance (5,6). Since annual strawberry production in these regions approaches 14 million quarts, a coordinated survey was conducted in 1971 and 1972 to determine the incidence and possible economic implication of green petal on the strawberry industry in Quebec and the Maritimes.

Methods

The survey was conducted at peak harvest time (between June 23 and July 13, depending on area) in commercial fields of strawberry (Fragaria chiloensis var. ananassa Bailey) in their first year of fruit production. Redcoat was the main cultivar surveyed and the cultivars Sparkle and Cavalier were also examined in areas where significant acreages were being grown. An attempt was made to survey no less than 10% of the acreage (with a minimum of 5 fields for each cultivar) of the first year fruiting fields of each cultivar in each area. The selection of areas and the random selection of fields were made by the cooperators in each province. Within a field, ten sampling sites, each consisting of 10 linear feet of matted row, were selected at random on a line running diagonally across the field. The number of infected plants was counted at each of the 10 sampling sites, and the total number of plants was counted at two of the sites to provide an average number of plants per site for the field. The prevalence of green petal in a region or province was expressed as a weighted mean % based on the acreage and

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Table 1. Strawberry green petal survey in Eastern Canada, 1971

Province	Area	Cultivar	First year acreage	Surveyed acreage	% Infection
Quebec	Terrebonne-Deux Montagnes	Redcoat	270	12.8	0.2
		Sparkle		6.0	0.1
	St. Hyacinthe	Redcoat	239	23.2	0.8
	Montmorency and Orleans Is.	Redcoat	500	26.2	0.1
	Bellechasse	Redcoat	190	13.2	0.3
New Brunswick		Redcoat		96.2	0
		Cavalier		29.5	0
		Sparkle		1.0	0
Nova Scotia	Annapolis	Redcoat	18.0	7.2	0.4
		Cavalier	1.6	0.7	0
		Sparkle	0.1	0.1	0
	Colchester-Pictou	Redcoat	9.0	7.0	0.9
		Cavalier	2.7	2.7	0.4
	Cumberland	Redcoat	19.0	16.0	0.6
		Sparkle	1.0	1.0	0
	Kings	Redcoat	10.0	2.8	0.4
		Cavalier	4.0	2.1	0.1
		Sparkle	3.7	1.9	0.7
	Yarmouth	Redcoat	2.2	1.2	0
		Cavalier	0.7	0.6	0
		Sparkle	0.5	0.4	0.2
Prince Edward Island		Redcoat	100.0	19.3	2.4
		Cavalier	17.0	6.0	1.9
		Sparkle	14.0	3.8	9.0

incidence of the disease in each field surveyed in the region (3), as follows:

$$\text{mean} = \frac{\Sigma\ (\%\ \text{diseased plants} \times \text{field acreage})}{\text{total acreage of fields}}$$

Yield losses were calculated on the basis that a plant showing symptoms of green petal bears no marketable fruit and that the percentage of infected plants is therefore directly related to fruit loss. Two methods were used in attempting to calculate losses due to green petal. In the first, the average percent infection for each province was calculated using the method of Grainger (3). The provincial production figures supplied by Statistics Canada were considered to be lower than potential production by an amount equal to the percentage green petal calculated for the province. In the second method, losses in the cultivar Redcoat were determined on a per acre basis for the various areas surveyed. These were calculated by multiplying the percentage infection for the area by the average yield per acre for that area. Average yield figures were based on yield data obtained from producers in each area.

Results and discussion

The results of the 1971 survey, summarized in Table 1, show a low level of green petal throughout the surveyed areas. In New Brunswick, for example, a few infected plants were observed in 8 of the fields surveyed but none were within the sampling sites. Of interest also, was the presence of green petal in the Terrebonne-Deux Montagnes area of Quebec. Although the percentage infection was low, the disease had not been reported previously in that area, indicating that the disease may be slowly spreading westward. Prince Edward Island is the only area in which the percentage infection might be considered significant but even there the most severely affected cultivar, Sparkle, was grown on a relatively small acreage.

Low levels of infection were again encountered in 1972 (Table 2). Green petal was recorded in all areas of Quebec surveyed and the highest percentage of infected plants was observed in the Terrebonne-Deux Montagnes area where the disease had been observed for

Table 2. Strawberry green petal survey in Eastern Canada, 1972

Province	Area	Cultivar	First year acreage	Surveyed acreage	% Infection
Quebec	L'Assomption	Redcoat	96	13.2	0.1
	Terrebonne-Deux Montagnes	Redcoat	232	31	2.2
	St. Hyacinthe	Redcoat	203	22	0.6
	Yamaska	Redcoat	106	13.3	1.5
	Montmorency	Redcoat	545	34.6	<0.1
	Bellechasse	Redcoat	248	10.9	0.6
New Brunswick	York	Redcoat		33.0	*
		Cavalier		2.5	0
		Sparkle		0.5	0
	Carleton	Redcoat		5.5	0
	Victoria	Redcoat		3.8	*
		Cavalier		0.8	0
	Madawaska	Redcoat		5.2	0
	Restigouche	Redcoat		6.0	0
	Gloucester	Redcoat		16.0	0
	Kent	Redcoat		4.5	0
	Westmorland	Redcoat		12	0
	Queens	Redcoat		28	0
		Cavalier		1.2	0
Nova Scotia	Annapolis-Kings	Redcoat	27.8	6.5	0.6
		Cavalier	4.7	1.6	0.5
		Sparkle	3.2	1.0	0.6
	Colchester-Pictou	Redcoat	9.5	5.0	0.3
		Cavalier	0.5	0.5	0
		Sparkle	2.0	1.0	3.0
	Cumberland	Redcoat	7.0	5.5	0
	Lunenburg	Redcoat	5.3	5.0	0.8
		Cavalier	0.3	0.3	0.9
		Sparkle	0.3	0.2	1.3
		Redcoat	2.9	2.5	1.6
		Cavalier	0.1	0.1	0
		Sparkle	0.9	0.5	0.9
Prince Edward Island		Redcoat	50.0	17.8	1.1
		Cavalier	8.5	4.1	0.5

* Green petal present in samples but percentage not calculated.

the first time in 1971. Although low numbers of infected plants were recorded in New Brunswick in 1972, the severe winterkill in many fields prevented the calculation of percentage infection. In Nova Scotia, green petal was recorded in all areas of the province except Cumberland County. Disease incidence in Prince Edward Island was down from that recorded in 1971. Severe winterkill in Eastern Canada in 1972 resulted in plant losses estimated at 50%, with losses in some fields reaching 90%. It is probable that this situation influenced the levels of green petal recorded in 1972.

Although the methods used for calculating losses in yield due to green petal might be considered approximations, they do serve to illustrate percent infection in terms of yield loss. In Table 3, for example, a 2.4% infection level in Prince Edward Island in 1971 resulted in a calculated loss of 41,311 quarts, and in Quebec in 1972 a level of infection of less than 1% resulted in a loss

Table 3. Estimated yield loss in strawberry production in Eastern Canada due to green petal disease in 1971 and 1972

Province and year	Total production ('000 qt)	Avg % infection	Potential total production without green petal ('000 qt)	Yield loss ('000 qt)
Quebec				
1971	7,830	0.3	7,854	24
1972	4,930	0.7	4,965	35
Nova Scotia				
1971	2,500	0.5	2,513	13
1972	1,200	0.5	1,206	6
Prince Edward Island				
1971	1,680	2.4	1,721	41
1972	270	1.1	273	3

Table 4. Estimated per acre losses in the cultivar Redcoat due to green petal in 1971 and 1972

Province and area	Avg yield (qt/acre)	Loss in yield (qt per acre) in: 1971	1972
Quebec			
St. Hyacinthe	6,500	53.3	38.4
Terrebonne-Deux Montagnes	7,000	13.3	156.8
L'Assomption	6,042	12.5	5.9
Yamaska	5,938		88.5
Montmorency	3,750	4.2	1.9
Bellechasse	3,750	10.0	22.1
Nova Scotia			
Annapolis-Kings	8,280	29.2	48.0
Colchester-Pictou	7,000	60.0	19.6
Lunenburg	9,000		72.0
Yarmouth	7,750	0	122.5
Prince Edward Island	4,200	100.8	44.5

of 33,753 quarts. Table 4 illustrates the variability in losses per acre in the various areas surveyed. In the Terrebonne-Deux Montagnes area of Quebec, for example, calculated losses in 1972 were 157 quarts per acre while in Montmorency losses were negligible. Loss calculations on an area basis serve to illustrate the importance of the disease to the economy of specific areas and in this respect are probably more meaningful than are figures for provinces as a whole.

The low level of green petal observed in 1971 and 1972 and the losses sustained suggest that this disease is not of major importance in the production of strawberries in Quebec and the Maritime Provinces. Several workers (2,6) have observed higher levels of infection in the cultivar Sparkle than in the cultivar Redcoat in commercial plantings. Since Sparkle has now been largely supplanted by Redcoat and other cultivars, this change in cultivars may have contributed to a lower disease incidence. It may well be, however, that a combination of factors such as availability of disease inoculum, leafhopper populations, susceptible cultivars, and climatic conditions might produce an optimum set of conditions whereby a high level of infection could occur, as has been reported in the past.

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SERIOUS DAMAGE CAUSED BY STALACTIFORM BLISTER RUST AND WESTERN GALL RUST TO A LODGEPOLE PINE PLANTATION IN CENTRAL ALBERTA

J.M. Powell and Y. Hiratsuka[1]

Abstract

Two pine stem rusts, stalactiform blister rust (Cronartium coleosporioides Arth.) and western gall rust (Endocronartium harknessii (J. P. Moore) Y. Hiratsuka), caused severe damage to lodgepole pine (Pinus contorta Doug. var. latifolia Engelm.) grown for Christmas trees and ornamental trees in a tree farm in central Alberta. Stalactiform blister rust killed over 80% of the young trees in the nursery area and a third of the transplanted stock in one area. Numerous new galls of western gall rust appeared in the fall of 1972, having originated from 1971 infections. A survey showed that 63% of the 6- to 12-year-old lodgepole pine were infected, with an average of 28 galls per infected tree. Because of the damage caused by the two pine stem rusts, the operator of the tree farm suffered severe financial loss.

Résumé

Les deux Rouilles-tumeurs Cronartium coleosporioides Arth. et Endocronartium harknessii (J. P. Moore) Y. Hiratsuka causèrent de sérieux dommages au Pin lodgepole (Pinus contorta Dougl. var. latifolia Engelm.) cultivé pour les arbres de Noel et ornementaux dans une ferme forestière en Alberta central. La première tua plus de 80% des jeunes arbres de la pépinière et un tiers des plants dans un certain secteur de la ferme. De nombreuses tumeurs de la deuxième apparurent à l'automne de 1972, résultats d'infections survenues en 1971. D'après un inventaire, 63% des Pins lodgepoles âgés de 6 à 12 ans furent infectés (moyenne de 28 tumeurs par arbre infecté). Tous ces dommages causèrent de sérieuses pertes financières à l'exploitant.

Introduction

During recent years, there have been several reports of significant localized damage by pine stem rusts in Canada and especially by the western gall rust, Endocronartium harknessii (J. P. Moore) Y. Hiratsuka (Carlson 1969, Forbes et al. 1970, 1972, V. Hildahl 1972 personal communication, Ives et al. 1969, 1971, Molnar et al. 1970). Up to 70% to 100% of the trees were reported to be infected in some localities in the Prairies (Ives et al. 1969, 1971, V. Hildahl 1972 personal communication), and a Scotch pine Christmas tree plantation in New Brunswick was abandoned because of the frequency and intensity of the western gall rust (Forbes et al. 1972).

In the summer of 1972, the attention of the Canadian Forestry Service was drawn to a pine stem rust problem on a tree farm, after several infected trees were noted among stock for distribution to the public in Edmonton. The tree farm, near Mackay, Alberta, covers 80 acres and is planted with nearly 100,000 trees. Over 30,000 of these trees are pines, mainly lodgepole pine, Pinus contorta Dougl. var. latifolia Engelm., with smaller plantings of Scotch pine, P. sylvestris L. The trees were grown for Christmas tree production, but in recent years an increased portion has been sold for the ornamental tree market mainly in the city of Edmonton. The trees, up to about 20 years in age, had been planted on agricultural land, surrounded by stands of native forest trees. To investigate and assess the situation, four visits were made to the tree farm by personnel of the Canadian Forestry Service.

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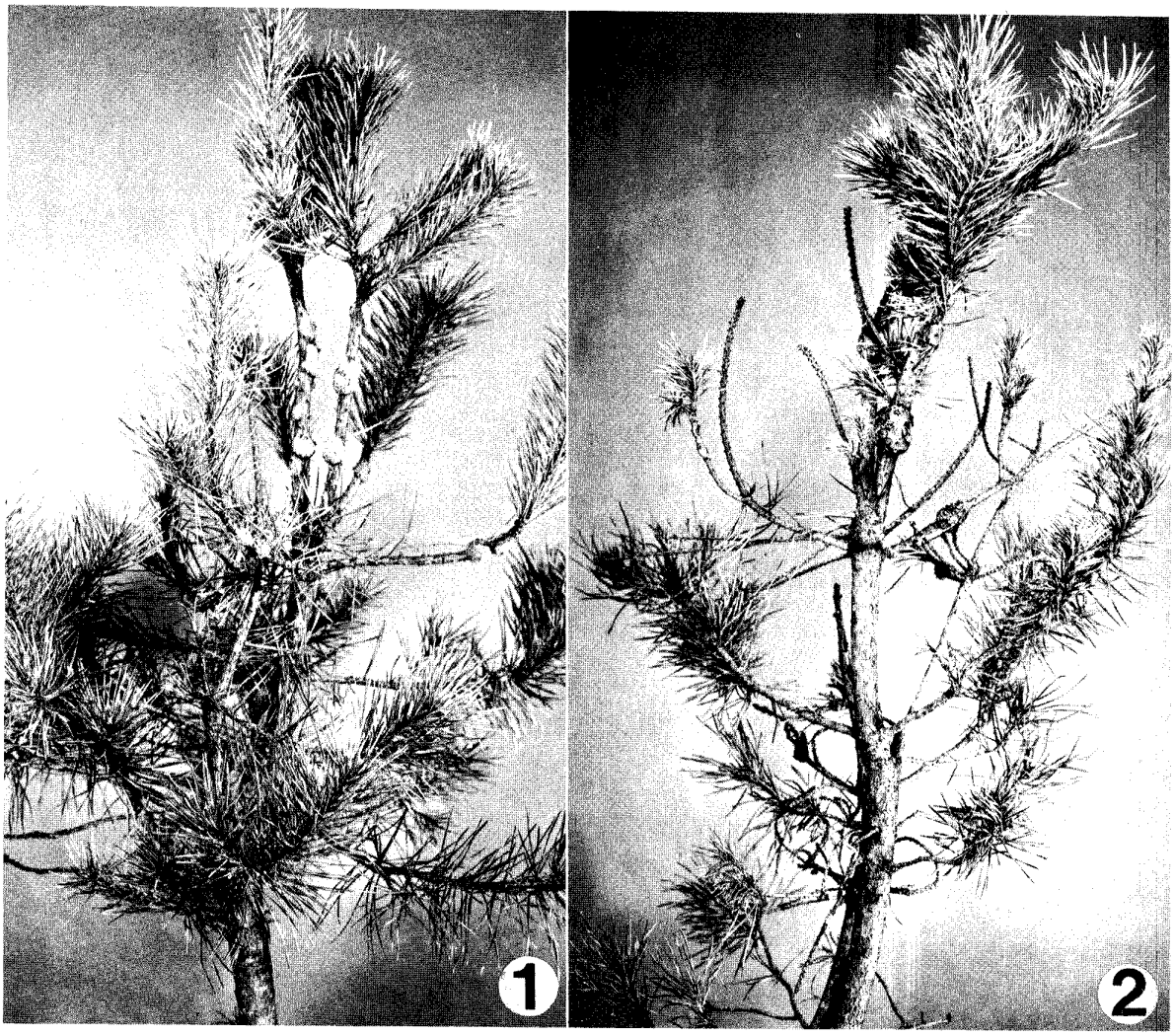


Figures 1 & 2. Top portions of 8-year-old lodgepole pine trees with 1) many multiple galls on 2nd-year shoots; 2) four 5-year-old galls that have caused some branch mortality and reduction of growth; a few new galls are also present.

Observations and discussion

During the two early summer visits, a small percentage of the older lodgepole pine was found to be infected by the western gall rust, and a small area of the younger lodgepole pine was heavily infected by the stalactiform blister rust, Cronartium coleosporioides Arth. The remaining trees in the adjacent nursery area from which the younger, 5- to 7-year-old stock had been transplanted were also heavily infected by stalactiform rust. It was recommended that the nursery area be cleared out and that all planted trees with stalactiform blister rust cankers should be removed, and further suggested that chemical control should be carried out on the surrounding healthy trees in August to prevent reinfection from the alternate host, Indian paintbrush, Castilleja spp., which was abundant and heavily infected in the plantation area. It was also recommended that the trees with western gall rust galls on the main stem be cut out, and branches with galls be pruned.

A re-examination on September 21 and again on October 20, of areas where stalactiform blister rust infected trees had been. showed that many infected trees remained despite the removal of over 1,000

Figure 3. Top portion of an 8-year-old tree with one 6-year-old main stem gall which has caused major reduction in growth and some branch mortality. Note new multiple galls on several of the branches.

trees, or nearly a third of the original plantings in this block of the plantation. Most of the infections occurred near the base of the tree. It was established through aging the cankers on ten infected trees that most of the infections had occurred in 1967 before transplanting. A visual check of the small remaining area in the nursery showed over 80% of the trees were already dead, and more than half of the remaining living trees had signs of active stalactiform blister rust cankers.

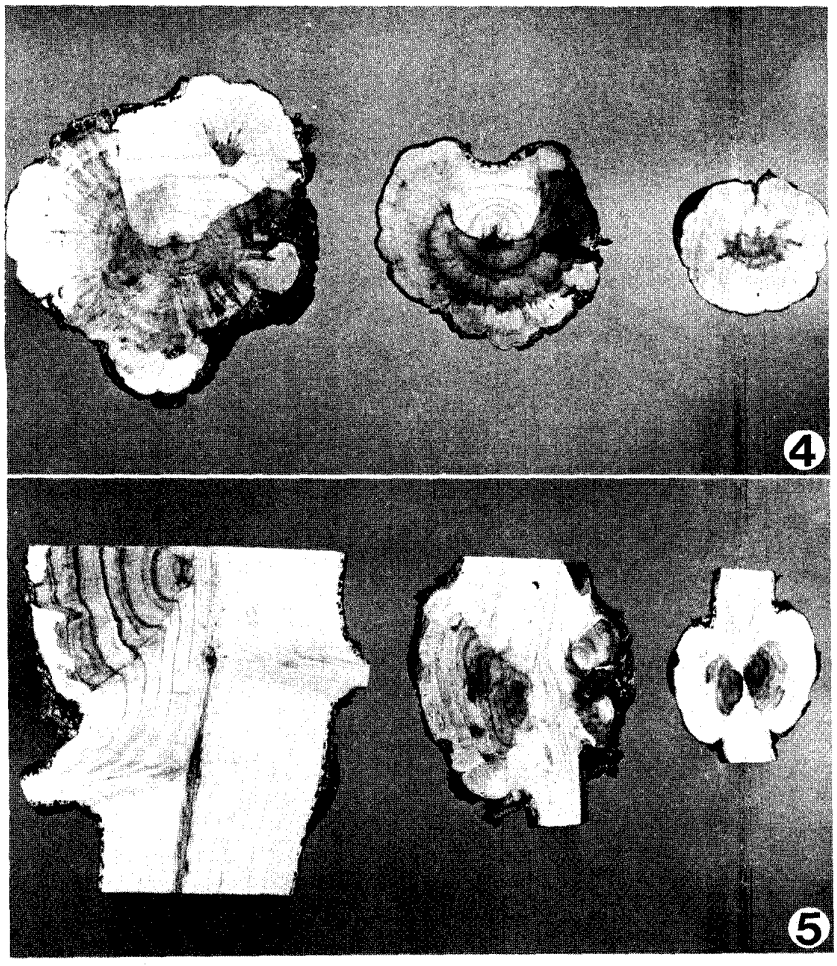
Over the summer there had been a spectacular change in the status of infections by the western gall rust, although some pruning had been carried out. Where before there were only a few infected trees in the 10- to 15-year-old and virtually none in the 8-year-old lodgepole pines, the rust was now very much in evidence with new galls developing on the main stems and branches (Figs. 1 to 3). On some leaders or branches up to ten galls were developing in a row on 1-year-old tissue. Attempts were therefore made on October 20, and subsequently in the laboratory, to obtain some quantitative data of the incidence and intensity of the rust and to find out the ages of infections.

The incidence of the rust was surveyed in two age groups of lodgepole pine (6-8 and 10-15-year-old) by tallying a total of one hundred trees in random rows from each planting block. To determine intensity and to find out the possible year of infection all galls that could be found on each tree were counted and aged. The number of galls occurring on the main stem were recorded separately. The age of the galls was assumed to equal the ages of the host tissues and was estimated by counting branch whorls as suggested by Peterson (1971), who found that in almost all cases infection occurs on the current-year shoots. The rust condition of these surveyed blocks appeared to be typical of the lodgepole pine blocks, although in some older blocks more older galls were in evidence, and the younger blocks planted near the edge of the plantation had less infection.

Percentages of trees infected in the older and younger age class were 57 and 69 respectively. In the older age class there was an average of 37 galls per infected tree and in the younger age class 20 galls (Table 1). On one tree 322 galls were counted and several others in the older age class had

Table 1. Number of lodgepole pine trees infected by western gall rust in two age classes and the number of galls in each age class surveyed in the field

Age class of trees	Number of trees sampled	Number of infected trees	Number and percentage of galls in each estimated year of infection									Total number of galls
			1971	1970	1969	1968	1967	1966	1965	1964	1963	
10-15 years	100	57	2037	7	7	8	18	1	2	2	1	2083
6-8 years	100	69	1391	0	0	1	1	1	0			1394
Total	200	126	3428	7	7	9	19	2	2	2	1	3477
%		63	98.6	0.2	0.2	0.3	0.6	<0.1	<0.1	<0.1	<0.1	



Figures 4 and 5. Sections of three galls with stained and deformed tissues used to determine the year of infection; 4) transverse sections; 5) longitudinal sections.

over 100 galls per tree. The percentage of galls occurring on the main stem was 2.7% in the older age class and 7.2% in the younger class. Of the total number of galls in the two age classes, 98.6% of the galls originated in 1971, percentages in other years were very small with the highest occuring in 1967 (0.5%) (Table 1).

To evaluate the field survey results, 599 galls were collected from the infected blocks for aging in the laboratory. This collection of galls was not a wholly random sample since increased emphasis was given to collecting older galls to help establish whether earlier "wave years" of infection had occurred. The galls were cut transversely (Fig. 4), or longitudinally (Fig. 5), for counting of the annual xylem rings to establish the first year of infection. Of the sample galls, 86.1% originated in 1971, 4.8% and 4.7% respectively in 1970 and 1967, and only 1% or less in the other years.

The two methods of sampling indicate that a wave year of infection occurred in 1971, some infection occurred in each of the other years with a slightly higher incidence in 1967. Peterson (1971) showed that wave years of infection occurred with western gall rust, although his data did not show such a marked year of infection as reported above. In his study the peak year of infection accounted for only 25% to 70% of the total infections.

Although the western gall rust had not yet killed the infected trees, the rust drastically restricted growth, causing the trees to become stunted and malformed (Fig. 3). Consequentely most of the heavily infected trees had lost their commercial value as ornamentals or Christmas trees. It was recommended that the owner should try to eliminate all trees with multiple galls or main stem galls and prune trees with only a few branch galls if the shape of the tree is not drastically altered. Some of these trees may be satisfactory for the Christmas tree market and thus will enable the operator to obtain some return on his investment. At last report the operator had cut or pulled out 3,500 of the trees heavily infected with western gall rust and estimated approximately the same number remained to be cut.

Conclusion

The two stem rusts were responsible for severe damage to lodgepole pine on a tree farm in central Alberta and caused substantial financial loss to the operator. Both stalactiform blister rust and western gall rust should be considered as dangerous biological agents capable of threatening the successful intensive cultivation of lodgepole pine in Alberta, especially in areas close to native stands.

Nursery stock showing a few rust infected seedlings should be destroyed and the remaining stock checked carefully for several years after being planted out, as incipient infections are probably present in a number of seedlings not showing signs of infection. Pruning of branch cankers and galls should be considered when trees in a plantation are of special value.

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GRAY SPECK OF OATS IN WESTERN CANADA[1]

W.A.F. Hagborg[2]

Attempts are being made to determine if resistance to gray speck caused by manganese deficiency is a necessary characteristic in new varieties of oats being developed for western Canada. The current concern is that certain new varieties of outstanding merit in other respects have been found to yield less than gray-speck resistant varieties on some soils.

It has been found (12, 29, 30, 33) that gray speck of oats may be due to an absolute manganese deficiency or to a deficiency of available manganese because of inorganic chemical fixation or biological fixation. This paper presents previously unpublished results of experiments on the identification and control of manganese deficiency in oats and includes a review of the literature pertaining to the occurrence of gray speck in western Canada and to the appraisal of oat varieties for resistance.

Establishment of the occurrence of gray speck in western Canada

Gray speck symptoms were observed in experimental plots and farmers' field in Manitoba for several years before diagnosis of the condition was confirmed experimentally (14). The determinative work of MacLachlan (25, 26) in Ontario suggested the desirability of similar studies here, and the presence of manganese deficiency in a Manitoba soil was established in 1944. Earlier attempts using field soils in greenhouse tests had failed to evince a response to manganese because of the presence of available manganese in the 6-inch clay pots used in the experiments. However deficiency symptoms were produced when procedures were followed to remove any traces of exchangeable manganese from the pots. They were washed thoroughly, steeped for 18 hr in 0.1 N NaCl followed by four successive steeps of 6, 15.5, 8, and 2 hr in tap water. A test of the last steep failed to show the presence of chlorine, suggesting that no manganese chloride remained in the pots which were then rinsed in running tap water, wiped with clean cheese-cloth and allowed to dry.

In the experiment, two soils were used: Gilbert Sandy Loam (10), (pH 6.8) from a field in which gray speck had been observed in SW 7 Township 25 Range 22 W near Gilbert Plains, Man.; and a potting soil (pH 7.5) containing a mixture of Red River clay and sand; the mixture was believed not to be deficient in available manganese. Later, the Soils Department, University of Manitoba, found the sand loam soil to be deficient in manganese, low in phosphates but not in potash. The potting soil was low in potash but not in phosphate. Unfortunately its manganese content was not determined.

Twelve pots of each soil for each of four treatments were randomized on a greenhouse bench. One treatment had 0.1 g $MnSO_4.4H_2O$ per pot mixed with the soil before sowing and 20 ml of aqueous solutions $MnSO_4.4H_2O$) (0.2 g) added 40 days after sowing. A[2] second treatment had no addition to the soil, but the plants were sprayed with 1% $MnSo_4.4H_2O$ (=0.68 $MnSO_4$) 34 days after sowing; the plants in a third treatment were sprayed with distilled water 34 days after sowing, a fourth treatment was left undisturbed. The pots were sown with seed of the susceptible variety Richland (37) that had been treated for 10 min by immersion in water at 57 C to kill any halo blight bacteria, Pseudomonas coronafaciens (Elliott) Stevens, that might be present. To establish that halo blight was not being confused with gray speck, 34-day-old plants in six pots in each treatment were inoculated with Ps. coronafaciens by pricking the crowns with a flamed and cooled nichrome needle dipped in inoculum. The plants in the remaining six pots of each treatment were wounded in the same fashion but not inoculated. Plants were examined for halo-blight 13 days later. Observations on gray speck were made 62 days after sowing.

The results of the test (Table 1) indicated a marked deficiency of available manganese in the field soil from Gilbert Plains, and a slight deficiency in the potting soil.

In Europe Steenbjerg and Boken (31) in pot experiments with Victory oats established that reducing agents applied to soil on which oats suffered from severe gray speck resulted in improved yields. Consequently a greenhouse pot experiment with soil from Gilbert Plains was done with the reducing agent quinhydrone. Additions of manganese sulfate and manganese chloride were included as treated controls and the effect of sodium as an exchange ion was determined by the inclusion of treatments with sodium sulfate and sodium chloride (Table 2). Four replicates of each treatment were sown with Richland oat seed that had been steeped at 62 C for 1 min then at 57 C for 10 min followed by cooling in cold water to prevent any possible infection by halo blight. The soil was almost neutral (pH 7.6). The pots were randomized on a greenhouse bench, and 4 weeks

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Table 1. Proportion of Richland oat plants developing halo blight and gray speck in a greenhouse test

Soil	Treatment	Plants developing: Halo blight	Plants developing: Gray speck
Field soil	Plants inoculated with *Ps. coronafaciens*		
	Mn applied to soil	16/17	1/17
	Mn sprayed on foliage	16/17	0/17
	Water sprayed on foliage	15/17	7/17
	Untreated	16/16	9/16
	Plants not inoculated		
	Mn applied to soil	0/18	0/18
	Mn sprayed on foliage	0/18	2/18
	Water sprayed on foliage	0/18	16/18
	Untreated	0/18	14/18
	% of plants developing symptoms of gray speck: Mn treated	4	
	untreated	67	
Potting soil	Plants inoculated with *Ps. coronafaciens*		
	Mn applied to soil	11/18	0/18
	Mn sprayed on foliage	18/18	0/18
	Water sprayed on foliage	18/18	0/18
	Untreated	18/18	3/18
	Plants not inoculated		
	Mn applied to soil	0/18	0/18
	Mn sprayed on foliage	0/18	1/18
	Water sprayed on foliage	0/18	1/18
	Untreated	0/18	3/18
	% of plants developing symptoms of gray speck: Mn treated	1	
	untreated	10	

Table 2. Severity of gray speck in plants of Richland oats 4 weeks after sowing in pots of field soil amended with various chemicals

Amendment and rate (g/6-inch pot of soil)		No. of plants in each severity category: Severe	Moderate	Slight	Trace	Nil
$MnSO_4 \cdot H_2O$	0.076	0	0	2	0	10
$MnCl_2 \cdot 4H_2O$	0.089	0	2	2	1	9
$Na_2SO_4 \cdot 10H_2O$	0.156	2	3	3	1	3
NaCl	0.059	2	6	3	0	1
Quinhydrone	1.767	0	0	0	0	12
Nil	0	2	5	4	0	1

after sowing the plants were examined for the presence and severity of gray speck (Table 2).

Dilution plate isolations of bacteria were made from the roots of 10 gray speck affected plants and from the roots of 10 apparently healthy plants. It was noted that the washed root mass of the healthy plants was approximately three times as great as that of the plants showing symptoms of gray speck. In both healthy and diseased plants the colony counts from individual roots ranged from just over 100 to "infinite" and were preponderantly over 500 per root, there being no correlation between bacterial count and treatment.

The same pots of soil were re-sown with Ajax oats and the plants harvested 13 weeks later. The plants were shaken to free the roots of most of the soil, then the above-ground parts of the plants were clipped off at the crown; the roots were allowed to soak for 3 hr in water, then washed in running water and allowed to soak 2 hr more. They

Table 3. Weight of oven-dried roots of Ajax oat plants grown in re-sown pots of field soil that had been amended before seeding a previous crop of oats (Table 2)

Amendment and rate (g/6-inch pot)		Replicates* 1	2	3	4	Total
$MnSO_4 \cdot H_2O$	0.076	0.500	0.615	0.350	0.260	1.725**
$MnCl_2 \cdot 4H_2O$	0.089	0.650	0.475	0.300	0.203[a]	1.628**
$Na_2SO_4 \cdot 10H_2O$	0.156	0.099	0.119	0.324	0.069	0.611
NaCl	0.059	0.237	0.389	0.239	0.225	1.090
Quinhydrone	1.767	0.754	0.600	0.355	0.415	2.124**
None	0	0.305	0.140	0.260	0.185	0.890

* 3 Plants/replicate, except [a] 2 plants adjusted to a 3-plant basis (3/2 x 0.135 = 0.203).

** Significantly different from control (P = <0.01).

were then washed in running water and spread to dry on filter paper placed on a pad of newspaper. After air-drying for 48 hr they were oven-dried at 105 C and weighed. Plants grown in soil amended with manganese sulfate, manganese chloride, and quinhydrone reducing compound all gave significantly higher root weights than the control (Table 3).

In 1945, paired plots of oat strain R.L. 1273 were grown on four farms at Gilbert Plains, Man., and on one farm each at Brokenhead, East Selkirk, Fort Garry, Hazelridge, Meleb, and Oakbank plants in one plot at each location were sprayed with 0.65% $MnSO_4.H_2O$. With the exception of those at Gilbert Plains all of the fields had been considered unproductive for unknown causes by Agricultural Representatives of the Manitoba Department of Agriculture.

At Gilbert Plains on Gilbert Sandy Loam soil, plots in one field showed a yield increase from spraying of 255% (P = < 0.01), but in another field on the same farm (SW 7-25-22) there was no response. In a field at NW 10-26-22, also on Gilbert Sandy Loam soil, the yield increase from spraying was 35% (P = < 0.01), and in a field at SW 8-25-22 on Dutton Clay Loam the yield increase was 16% (P = < 0.01). In a fifth field, at SW 27-25-22, also on Dutton Clay Loam, there was no response to the manganese sulfate spray.

In plots at Oakbank, Man. (SW 7-25-22) on Marquette Clay to Heavy Clay Loam soil, a yield increase of 101% (P = < 0.01) was obtained, while at Brokenhead, East Selkirk, Fort Garry, Hazelridge, and Meleb there was no response to spraying with manganese sulfate.

Wherever a substantial increase in yield was obtained a noticeable improvement was observed in the vigor and height of the plants treated with manganese sulfate.

Also in 1945, a seed steep with 23% manganese sulfate for 20 min followed by covering for 6 hr (moist) gave a yield increase of 55% in the field (P = < 0.05) at Gilbert Plains, Man. (16). Later Berkenkamp and McBeath (4) obtained some control of gray speck by pelleting seed with manganese phosphate.

In July 1945, Mr. B. Peturson drew to my attention 12 plots of Tama oats affected by gray speck at the University of Manitoba in Fort Garry. The plants were 18 inches tall and had reached the early shotblade stage, considered a late stage for a response to Mn spray. However one guard row of each plot of Tama was sprayed with 1% $MnSO_4$, the other left unsprayed. Spraying resulted in a mean increase in yield of 30% (P = < 0.01). This experiment demonstrated that a deficiency of available manganese occured in the soil type on which the moderately resistant variety Exeter was selected.

Varietal resistance

Although manganese appears to be an essential element for all oat varieties, differences in varietal resistance to gray speck have been reported by several investigators (1,2,9,11,28,32,37,38) but not always consistently. Inconsistencies may be due in part to differences in the standards used by different investigators and in part to soil heterogeneity with respect to manganese availability. It is not unusual to find the same variety with widely different severities of gray speck in different replicates of the same test. For this reason, the maximum severity found may be more reliable than the mean of all readings. Perhaps an even more reliable indication would be dry weight of roots produced in carefully controlled comparisons of different varieties grown in a uniformly-mixed soil deficient in available manganese. Dry weight of roots appeared to be a useful criterion in determining the effect of quinhydrone and might prove useful in varietal comparisons.

The original plot-by-plot data gathered at Oakbank on percentage of leaf area destroyed by gray speck and on reduced vigor, and summarized in part previously (37 Table 1), were re-examined. Of 44 varieties tested 2 years or more during 1947, 1948, and 1949, 8, (Bambu, Benton, Exeter, Landhafer, Larain, Nakota, Santa Fe, and Sixty Day) had maximum single-plot ratings of 30% or less, and 2 of these (Landhafer and Santa Fe) had maximum single-plot ratings of 15%.

Table 4. Chemical content, basis dry matter, of varieties differing in degree of gray speck development when grown on a neutral Red River clay soil deficient in available manganese at Oakbank, Man., 1945

Variety	Gray speck (%)	Ash (%)	K (%)	Ca (%)	Mg (%)	P (%)	Fe (ppm)	Mn (ppm)
Black Mesdag	6.0	10.4	3.34	0.34	0.25	0.26	160	9.5
Ajax	7.3	8.3	2.68	0.26	0.18	0.30	130	9.5
Beaver	18.3	11.6	4.29	0.37	0.32	0.39	140	10.0
Gopher	20.0	11.5	3.95	0.38	0.35	0.40	160	8.0
Tama	31.7	11.6	3.34	0.38	0.30	0.30	205	10.5
Trispernia	76.7	14.4	4.30	0.29	0.33	0.36	160	11.0

Chemical content of oat tissues

In the 1945 varietal test of resistance to gray speck reported by Welsh et al. (37), samples of plant material from two resistant, two intermediate, and two susceptible varieties were submitted for analysis to Division of Chemistry, Science Service, CDA, Ottawa, Ontario. Only minor differences in manganese content were found (F. B. Johnston, personal communication) even though there were very marked differences in the field readings for gray speck (Table 4). These data suggest that plants resistant to gray speck do not accumulate a high content of manganese. More recent data (R. I. H. McKenzie, personal communication) indicate that the manganese content of oat plants varies widely in different soils regardless of variety (Table 5 and 6) and tends to be low in fields suspected of gray speck proneness, such as those at Glenlea, Man. Low manganese content appeared both in a resistant variety, Rodney, and a susceptible variety, O. T. 187 (Tables 5, 6). A low content of manganese was also found by Leach et al. (24) in several samples of Tama oats grown at Oakbank by W. A. F. Hagborg. It seems quite possible that plant sampling for manganese content may be a useful method of surveying for gray speck-prone soils.

Table 5. Manganese content* (ppm), basis dry matter, in flag leaves of oat plants collected at 3 locations in Manitoba in 1972

Variety	Glenlea	Portage la Prairie	Brandon
Rodney	13.0	96.2	117.0
		86.8	84.0
O.T. 186	8.2		
O.T. 187	8.0	53.5	56.0
	8.0	47.2	96.1
	15.3		
	8.0		
Frazer		110.0	79.1
		92.6	80.1

* Determinations by G. Racz, Soil Science Department, University of Manitoba, by atomic absorption spectrometry (Perkin-Elmer Model 303).

Summary of reports of gray speck in western Canada

Gray speck was first reported in western Canada by Hagborg (13,14,16) at Winnipeg and Gilbert Plains in Manitoba. Later it was found at Swan River, Ethelbert, Erickson, and Oakbank (15), Portage la Prairie and Elm

Table 6. Manganese content* (ppm) in seed samples of two lines of oats grown in the Co-operative Oat Test in 1972

Variety	Glenlea, Man.	Portage la Prairie, Man.	Brandon, Man.	Indian Head, Sask.	Edmonton, Alta.
Rodney	16.2	43.0	39.0	53.5	31.2
O.T. 187	4.7	32.0	36.5	42.5	19.7

* Determinations by R.E. Smith, Soils Section, CDA Research Station, Winnipeg, Man., by atomic absorption spectrometry (Perkin-Elmer, Model 303).

Creek (23), St. Norbert (17), Balmoral, Mesieres, and Prawda (35), Starbuck and Oak Bluff (18), and Glenlea (20). A sample of typical gray speck was also received from the Peguis Indian Reserve, Hodgson, Man. in 1969.

Vanterpool (34) in 1949 first reported gray speck in Saskatchewan at Spalding and confirmed the response of oats to manganese treatment on soil from that location. Subsequently Vanterpool and Samborski (36) reported work with Mn amendments to the same soil. In 1953 and 1962 Vanterpool (36, 8) again reported gray speck at Spalding. Manganese deficiency in soils at Kinistino, Sask., was evidenced by the discovery of marsh spot in peas (Pisum sativum L.) (19). Samples of peas with the marsh spot syndrome were also received from Aylesham, Sask., and Sperling, Man.

Henry (23) first reported gray speck in Alberta in 1951 at Edmonton and in the Peace River District. In 1959 Campbell (5) reported it from 29 of 76 fields of oats surveyed in the northern part of the province. Two years later Campbell and Horricks (6) reported it in 26 fields in the north, central, and foot-hill areas of the province and, also in 1961, Campbell and Skoropad (7) confirmed that manganese deficiency was the cause of the disease and reported evidence of some factors influencing the availability of manganese in Alberta soils. Still later the disease was also found at High Prairie, McGrath, and in southern Alberta (8), Lacombe and Red Deer (3), Crooked Creek (22), La Crete, Fluffton and Two Hills (21), and Thorhild and Redwater (20).

Although gray speck is evidently widespread in Manitoba and Alberta, its importance is diminished by the fact that it occurs in patches even at the many known locations.

Discussion

Whether the abundant data on varietal reaction to the Scolecotrichum or gray speck disease reported by Nilsson-Ehle (27) in 1908 applies to our presently known gray speck disease is somewhat uncertain. It was not until 1928 that Samuel and Piper (29) showed that gray speck was a manganese-deficiency disease due probably to a deficiency of available manganese in soil. They pointed out that some kinds of plants either require less manganese or else have a greater ability to absorb manganese. They did not mention varietal differences within the cereals but stressed the difference between rye (resistant) and the other cereals. Gray-speck resistant oat varieties do not have a lower manganese content than susceptible varieties grown on the same soil (Table 4), so there should be no fear by nutritionists that widespread use of gray-speck resistant varieties would fail to supply sufficient manganese. Susceptible varieties are also low in manganese when grown on soils low in available manganese.

The question of whether or not strains of oats developed for release as commercial varieties in western Canada should have gray-speck resistance is a matter requiring the appraisal of several factors. Obviously resistance is a desirable character, but lack of gray-speck resistance may not be sufficiently important to prevent the release of new varieties with other desirable characteristics such as resistance to stem rust.

From the data reviewed in this paper it is obvious that soils lacking available manganese are widespread in Manitoba and Alberta and are present locally in Saskatchewan. To date there is insufficient data from the prairie provinces on gray speck to arrive at a firm conclusion as to the need for resistance to it in a new variety. It would appear that a thorough survey, possibly in cooperation with Agricultural Representatives, in which tissue samples are collected and analyzed by atomic absorption spectrophotometry would provide the required data for estimating the extent of the problem and the probable monetary loss associated with releasing a variety lacking resistance to gray speck. Comparisons could then be made with potential losses from susceptibility to other diseases such as stem rust.

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BOTRYTIS FABAE AND ASCOCHYTA FABAE ON BROAD BEANS IN NOVA SCOTIA[1]

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Abstract

Chocolate spot [Botrytis fabae] and leaf and pod spot [Ascochyta fabae] were found on plants of tickbean, Vicia faba var. minor, cultivar Maris Bead, for the first time in Nova Scotia. B. fabae has not heretofore been reported in North America. It occurred primarily on the foliage and had no apparent effect on yield of spring-grown tickbeans. The effect of A. fabae was most severe on the seed and it may be the greater threat to bean production.

Introduction

The broad bean (Vicia faba L.) is a cool-season plant widely grown as a field crop in Great Britain and Europe where the gross margin of profit per acre approximates that of barley (3). Mazagan, windsor, English bean and horse bean are names frequently used for this plant. The tickbean cultivar Maris Bead, one of the small seeded varieties of horse bean, Vicia faba L. var. minor (Peterm.)Beck., has recently been intoduced for commercial production in Nova Scotia.

In August 1970, the leaves, pods, and stems of tickbean plants in Annapolis and Kings counties, Nova Scotia, were severely spotted. Botrytis fabae Sard. was the dominant organism isolated from leaf spots and Ascochyta fabae Speg. fruited on pod lesions. These fungi were determined to be the cause of specific infections on leaves, pods, and stems.

B. fabae, has not heretofore been reported in North America. It has been recorded previously from Africa, Asia, Australasia, Europe, including Great Britain, and South America (2). B. fabae and A. fabae probably came to Nova Scotia via tickbean seed imported from England. Since 1970 varying intensities of leaf and pod spot occurred on tickbeans in commercial fields. A specimen of B. fabae has been filed in the National Mycological Herbarium, Plant Research Institute, Ottawa, Ontario, as DAOM 137145. Specimens of A. fabae on leaves and pods were filed as DAOM 142288 and DAOM 142290, respectively.

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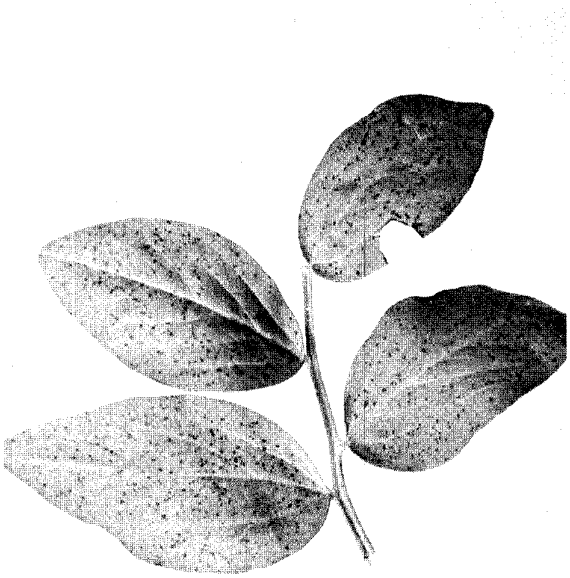
Symptoms

Chocolate spot disease caused by B. fabae occurs on leaves, pods and stems. Leaf lesions vary from small reddish brown spots (Fig. 1) to conspicuous well defined lesions with reddish brown margins and tan colored centers (Fig. 2). Later these lesions become entirely reddish brown. Under favorable conditions the disease becomes aggressive and lesions may coalesce causing blackening and partial defoliation. On pods, spots may be merely brown markings or may be similar to those on the leaves. On stems, infections may be similar to those on leaves (Fig. 3) or they may occur as streaks.

A. fabae occurs on leaves, pods and stems. Few leaf lesions were seen and then only on plants in areas where the disease was severe on the pods. Leaf spots were slightly sunken, circular to irregular, and up to 8 mm in diameter. Most spots had definite reddish brown margins with paler centers (Fig. 5). At a later stage spots often coalesce forming irregular patches and turning almost black. Lesions may be dotted with numerous pycnidia which exude spores in tendrils during damp weather.

The intensity of A. fabae infection was greater on pods than on leaves or stems. Pod lesions are similar to those on the leaves except they are more sunken and often larger. These spots become black, coalesce and bear abundant pycnidia which exude spores in tendrils (Fig. 6). The mycelium often grows through the pod onto the seed (Fig. 7). On mature seed infected areas may be circular or irregular and dark brown to black in color (Fig. 8). Seed from pods which become infected early in the season may be black and shrivelled.

Stem lesions are similar to those on leaves except they are more deeply sunken and often somewhat elongated (Fig. 9). They may coalesce to form extended areas of infection and in severe attacks may weaken the stem and kill the plant. Few pycnidia formed in stem lesions.



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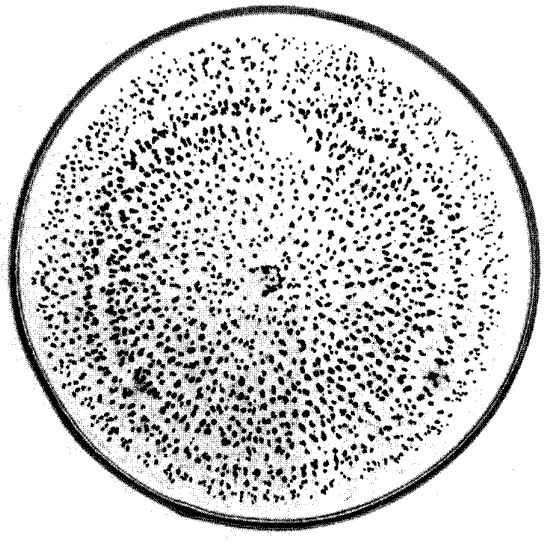

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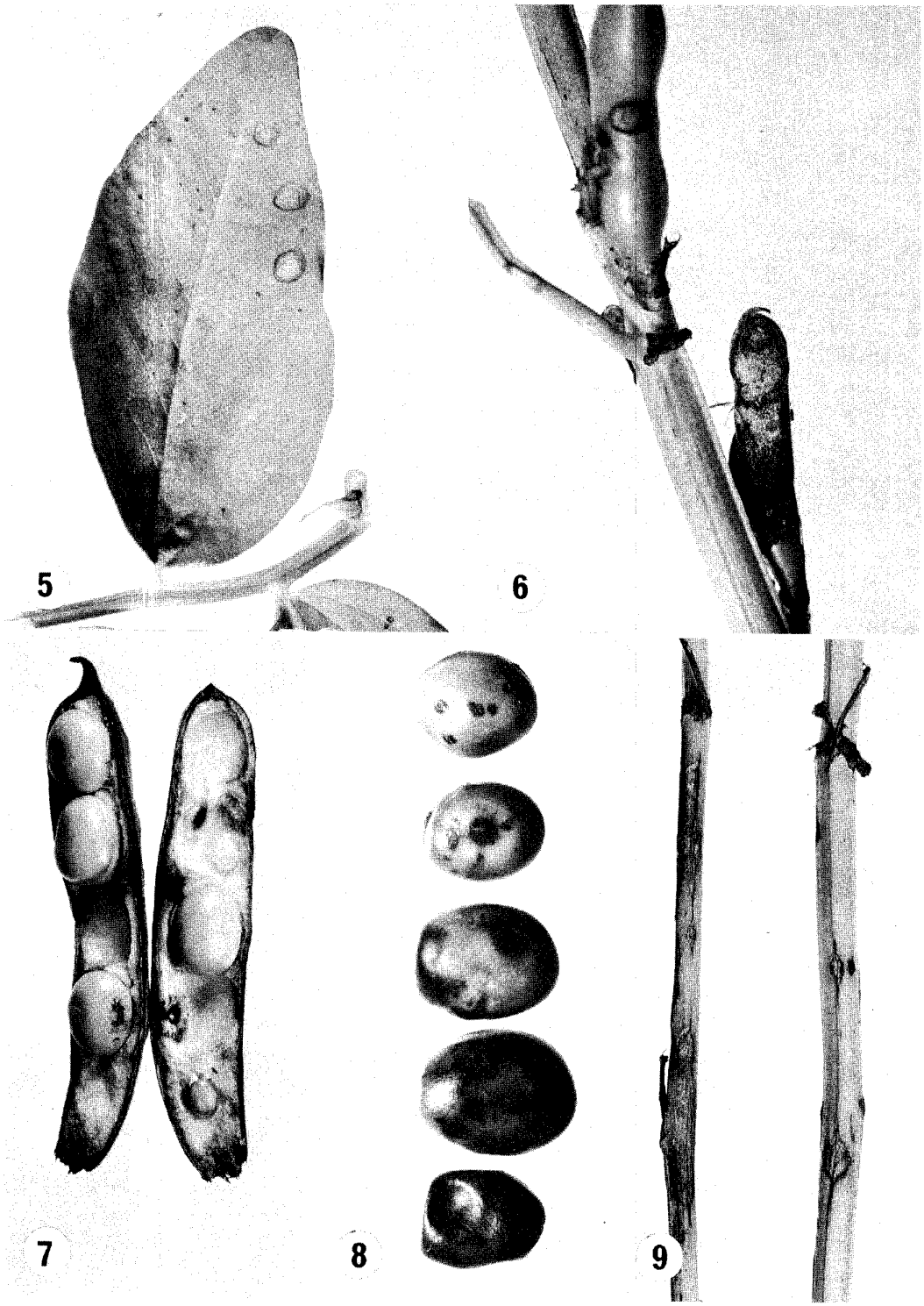
Figures 1–4. *Botrytis fabae* on tickbean cv. Maris Bead; 1) small lesions on leaf; 2) leaf lesions with dark margins and tan centers; 3) lesions on stem; 4) sclerotia in culture.



Figures 5–9. *Ascochyta fabae* on tickbean cv. Maris Bead; 5) lesions on leaf; 6) lesions on pods; 7) infected seed; 8) lesions on dried beans; 9) lesions on stems.

Fungus morphology and cultural characteristics

B. fabae sclerotia were numerous in agar culture (Fig. 4), discrete or sometimes confluent and they were within the diameter range of 1-1.7 mm given by Ellis (2). His conidial dimensions of 14-29 x 11-20 µm (mostly 16-25 x 13-16 µm) coincide with measurements of conidia for our isolate. No perfect stage is know.

A. fabae grew well in agar culture but produced few pycnidia. On sterilized bean seed this fungus grew luxuriantly and produced abundant pycnidia which spored profusely (Kentville Plant Pathological Specimen 2811). Our observations agree with those of Beaumont (1) who gives pycnidial dimensions on the host plant as being mostly 120-150 m (range 80-200 µm), ostiole 30-50 µm, pycnospores 15-18 x 4-5 µm (range 12-23 x 4-6 µm) with rounded ends, 1 septate, rarely continuous, not constricted, hyaline. In culture pycnospores were 14-23 x 3-6 µm, mostly 18-20 µm, and commonly showed a proportion of 2- or 3-septate spores, not generally found on the natural host.

Epidemiology and control

Weather conditions determine the severity of attack of B. fabae and A. fabae on broad bean (3). Cool, moist conditions in the early part of the growing season are most favorable for development and spread of these diseases. In dry seasons infections may be confined to the lower parts of the plant. There are no resistant cultivars and there is no satisfactory seed treatment. In England, B. fabae seldom does much damage to spring beans but winter beans may be completely killed (3). In Nova Scotia no spring-grown tickbean plants were free of chocolate spot and the disease did not appear to have any apparent affect on plant growth. The fungus persist from season to season in old bean haulm and on the seed. A. fabae is seed borne and the only satisfactory method of control is the use of clean seed.

Survey

On August 28, 1972, a random selection of fields of tickbeans grown from three grades of seed were surveyed for A. fabae infection (Table 1). Because infections were numerous on the pods and scanty on the foliage the survey was limited to determining the intensity of pod infection. The pods on at least 20 plants per field were examined for disease. The number of pods per plant ranged from 0 to 39, and averaged 11. Imported seed was not free of A. fabae infection because the disease appeared in plants grown from basic seed in fields where broad beans had never been grown previously. The percentage of basic seed plants per field having infected pods ranged from 15 to 40, and the percentage of infected pods ranged from 1 to 6. The number of A. fabae infected plants increased rapidely in each crop grown and seeded in succession from basic seed.

Table 1. Intensity of *Ascochyta fabae* infection on Maris Bead tickbeans

Bean grade seeded*	Acreage surveyed	Percent infected	
		Plants	Pods
Basic	12	21	3
Commercial	8	57	16
Commercial (+)	25	75	36

* Basic - seed imported from England; Commercial - local seed from basic stock; Commercial (+) - local seed from commercial stock.

On September 20, the survey was repeated on the same fields examined on August 28. The amount of A. fabae on plants and pods did not differ for the two dates. On September 20 many plants had matured and turned black which made it difficult to see disease symptoms, whereas on August 28 the infections contrasted well with the green color of the plants.

Conclusions

The cool, moist climate of Nova Scotia provided ideal conditions for development and spread of B. fabae and A. fabae diseases of tickbean. Commercial growers have not been concerned about these diseases but they do recognize the importance of disease-free seed.

Beaumont (1) stated: "A. fabae, although widely distributed throughout the world, rarely causes appreciable economic damage". However, it may be the more damaging of the two diseases in Nova Scotia because infections were most severe on pods and seeds.

This report extends the host range of both pathogens to Nova Scotia and that of B. fabae to North America.

Acknowledgment

The authors are indebted to Mr. A. T. Lightfoot for the photographs that appear in this paper.

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DISEASES OF BRASSICA SPECIES IN SASKATCHEWAN, 1970-72

II. STEM, POD, AND LEAF SPOTS[1]

G. Allan Petrie[2]

Abstract

Alternaria black spot [Alternaria brassicae and A. raphani] was the principal stem and pod spot of Brassica species from 1970 to 1972. Average severity indices for the disease increased by a factor of four during the 3-year period in both rape (Brassica napus) and turnip rape (B. campestris). The average severity ratings for B. campestris were almost double those for B. napus each year. Ringspot [Mycosphaerella brassicicola] and pod drop (a pedicel rot) were not diseases of major importance. However, pod drop was considered potentially serious, as it caused entire pods to be lost or fail to fill normally; Alternaria alternata and Cladosporium sp. were isolated from infected pedicels.

Introduction

The diseases to be dealt with in this paper are alternaria black spot, ringspot, and "pod drop". Alternaria black spot is caused by Alternaria brassicae (Berk.) Sacc. and A. raphani Groves & Skolko. Mycosphaerella brassicicola (Duby) Lind. is responsible for ringspot. The cause of pod drop has now been identified with certainty.

Methods

Procedures employed, including the method of calculation of the disease severity index (DSI) have been described in an earlier paper (3). Locations of fields inspected appeared in Figures 1 and 2 of that publication. Severity classes used for alternaria black spot and ringspot have been defined in Tables 1 and 2, respectively, of this paper. In 1972, percentages of pods exhibiting pod drop symptoms were determined on plants from certain fields to indicate potential yield reductions attributable to infections of the severity usually encountered. Meaningful estimates of reductions in yield caused by Alternaria and Mycosphaerella cannot be made at present.

Results and discussion

Alternaria black spot.

In many fields, infection of high percentages of plants was coupled with very low severity indices (Tables 3 and 4).

Table 1. Disease severity classes used for alternaria black spot

Severity class	Percentage of surface area of plant covered by lesions
0	0
TR*	<1
1	1 - 10
2	11 - 30
3	>30

* Plants in this class were considered healthy for purposes of calculating the disease severity index.

However, in 1971 and 1972, the mean DSI values for black spot were double those of the preceding year in both Brassica napus L. and B. campestris L. (Table 3). A DSI rating of 17 for a field represents the equivalent

Table 2. Disease severity classes used for ringspot

Severity class	Percentage of surface area of plant covered by lesions
0	0
1	1 - 20
2	21 - 40
3	41 - 60
4	61 - 80
5	81 - 100

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Table 3. Prevalence, incidence, and severity of alternaria black spot and ringspot in Saskatchewan from 1970 to 1972

	Disease	*Brassica napus* 1970	1971	1972	*Brassica campestris* 1970	1971	1972
No. of fields sampled		16	18	19	24	51	19
% of fields having the disease	black spot	100	100	100	100	100	100
	ringspot	94	94	50	88	96	94
% of plants per field diseased (avg)	black spot	90	94	93	82	97	99
	ringspot	56	27		43	79	
Avg DSI* (%)	black spot	5	10	18	8	18	32
	ringspot	7	2		9	9	

* DSI = disease severity index.

Table 4. Percentages of fields with alternaria black spot in each of a number of disease severity categories

Mean DSI	*Brassica napus* 1970	1971	1972	*Brassica campestris* 1970	1971	1972
0 - 5	75	33	28	58	28	0
6 - 10	13	22	11	17	8	0
11 - 20	13	28	22	8	20	11
21 - 30	0	16	16	4	20	28
31 - 40	0	0	16	13	24	39
41 - 50	0	0	6	0	2	22

of up to 10% of the surface area of 50% of the plants covered with lesions. Arbitrarily, all fields of B. campestris and B. napus with DSI values of 17 or more have been listed in Tables 5 and 6, respectively, along with their locations.

The uniformity and severity of infection in many fields examined in 1971 and 1972 was striking. The abundance of spores on plants at harvest time in northern Saskatchewan was vividly demonstrated in 1972. In August a 12.5-g sample of a dark powder scraped from a swather was received from the Meadow Lake area. Upon examination, it was found to consist almost entirely of conidia of A. brassicae. Those working in fields around the northern community attributed cases of skin and eye irrigation to this source. The prevalence of black spot in crop districts 5B, 8, and 9 has also been reflected in high levels of seed-borne Alternaria (unpublished data).

It would appear from the data in Tables 3 and 4 that Brassica napus and B. campestris differ considerably in susceptibility to black spot. Reports in the literature have indicated that B. hirta Moench and B. napus are considerably more resistant to Alternaria brassicae than is B. campestris (1, 2). Observations of leaf infections on plants in varietal tests in the field lend support to these reports, as do infection studies (unpublished data). Nevertheless, factors other than inherent resistance or susceptibility may offer a partial explanation for the discrepancies in DSI values between species in Tables 3 and 4. These include differences in geographical area of cultivation. There is a great preponderance of B. campestris acreage in northern districts due to the earlier maturation of this species.

Ringspot

The 1970 and 1971 data for this disease are summarized in Tables 3, 7, and 8. Average severity indices were low for both Brassica species. In 1972, ringspot was generally inconspicous and only abbreviated notes were taken. In B. napus, infections rated "trace" occurred at one to a few out of 10 sampling sites per field. In fields of B. campestris, the disease was recorded at almost all sampling sites but still rated "trace" or "trace to slight". No ringspot was seen on B. hirta. The severity index of the only field of B. juncea (L.) Coss examined (field 7, 1971) was 5, with 57% of the plants infected.

Often during the 3-year period, young infections on lower parts of stems were the only indications of the presence of ringspot. These linear, tan-colored lesions were unlike typical older lesions caused by the fungus and could easily go unnoticed.

Table 5. *Brassica campestris* fields most heavily affected by alternaria black spot

Field no.	DSI	Crop District	Locality
	1970		
17	32	8A	Nipawin
23	18	9A	Meath Park
30	32	9B	St. Walburg
33 - 35	19 - 33 (avg 27)	9B	Meadow Lake
	1971		
11	24	9B	Maidstone
19, 59, 67	27 - 33 (avg 31)	8B	
20, 22, 31, 32, 33, 36	19 - 35 (avg 26)	5B	
24, 26, 27, 28	17 - 36 (avg 26)	9B	North Battleford to St. Walburg
39, 40, 41, 43, 44	29 - 37 (avg 33)	9A	Prince Albert to White Fox
52, 55	24, 35	8A	Melfort, Tisdale
61, 63, 64, 65, 66	23 - 43 (avg 33)	9A	West of Prince Albert
	1972		
13 - 15, 19 - 24	22 - 48 (avg 33)	5B	
30 - 35	30 - 45 (avg 38)	9B	Meadow Lake

Table 6. *Brassica napus* fields most heavily affected by alternaria black spot

Field no.	DSI	Crop District	Locality
	1970		
18	20	8A	Nipawin
	1971		
47	24	8A	Nipawin
48	20	8A	Nipawin
68	23	8B	St. Benedict
71	22	8B	Humboldt
	1972		
10	22	8B	Annaheim
17	17	5B	Preeceville
18	26	5B	Stenen
25	28	9A	Fielding
26 - 29	20 - 43 (avg 33)	9B	North Battleford to St. Walburg
43	34	9A	Shellbrook
44	20	9A	MacDowall

In July, 1972, the white leaf spot (Cercospora) stage of Mycosphaerella, first observed by Vanterpool (4, and personal communication), was seen frequently on Brassica breeding lines at Saskatoon. The spots were usually 1-2 cm in diameter. Large numbers of 0- to 3-septate Cercospora spores were washed free in water mounts. Colonies identical to those isolated from Mycosphaerella stem lesions developed in spore dilution plates.

Pod drop

Pod drop was first observed by the author in July 1970 on turnip rape from the Rosetown area of Saskatchewan. Symptoms consisted of unusual black lesioning of the pedicel just at and below the point of attachment to the

Table 7. Percentages of fields with ringspot in each of a number of disease severity categories

Mean DSI	*Brassica napus*		*Brassica campestris*	
	1970	1971	1970	1971
0 - 5	38	94	54	24
6 - 10	31	0	17	43
11 - 20	31	6	17	29
21 - 30	0	0	8	4
31 - 40	0	0	4	0

pod. The infections probably started from diseased petals. In instances in which the blackening extended for several mm down the pedicels, the pods often had failed to fill normally. Pods were frequently missing. Gaps in the inflorescence may result from 2,4-D-induced flower drop or from lack of pollination on calm days. However, loss of pods due to the pod drop disease may be distinguished by the characteristic blackening at the tips of the pedicels.

Pod drop was recorded in 13% of the 1970 fields, 9% of the 1971 fields, and 18% of those sampled in 1972. Infections were plentiful in 1971, but in each of the other 2 years the disease was abundant only in one field. All of the fields affected were of Brassica napus, with the exception of one of B. juncea. Taking this into consideration, the disease was detected in 31%, 28%, and 42% of the B. napus fields examined in 1970, 1971, and 1972, respectively.

In 1970, several Brassica introductions at Saskatoon had many examples of this type of infection. Alternaria alternata (Fries) Keissler and a species of Cladosporium were isolated. In 1971, many platings of material with pod drop symptoms were made. Cladosporium and A. alternata were the only fungi obtained consistently. Only the

Table 8. Fields of *Brassica napus* and *Brassica campestris* most heavily infected with ringspot*

Field no.	DSI	Crop District	Locality
	Brassica napus		
	1970		
4	11	9A	Albertville
5	11	9A	Spruce Home
13	16	8A	Zenon Park
20	14	8A	Valparaiso
27	11	9B	Cavalier
	1971		
68	14	8B	St. Benedict
	Brassica campestris		
	1970		
16	29	8A	Carrot River
17	18	8A	Nipawin
23	19	9A	Meath Park
29	26	9B	Turtleford
32	14	9B	Makwa
33	36	9B	Meadow Lake
34	11	9B	Meadow Lake
	1971		
4	10	7B	Landis
11, 13, 27, 28, 30	10 - 15 (avg 13)	9B	
17, 20, 21, 32, 36	10 - 16 (avg 12)	5B	
19, 59, 67	12 - 23 (avg 18)	8B	
45	10	8A	Nipawin
39, 41, 42, 43, 44, 61, 66	13 - 29 (avg 17)	9A	

* Only fields having DSI values of 10 or more have been listed. A value of 10 is the equivalent of up to 20% of the surface area of 50% of the plants covered with lesions.

latter grew from 1972 collections. In all these attempts, A. brassicae was never isolated and A. raphani was found only rarely.

Percentages of infected pods were determined on plants from a number of representative sampling sites from several fields. On an average, from 5% to 13% infected pods per plant were found. The highest percentage was 33. There were up to 67% infected pods per branch, and an average of from 50% to 75% of the branches per plant had diseased pods.

Of the three diseases considered, only alternaria black spot was of major significance. Pod drop, however, is thought to be potentially the most devastating of any new disease problem to appear on this crop in recent years.

Acknowledgments

The author gratefully acknowledges the technical assistance of Miss Marjorie M. Smith and Mr. George Cornwell.

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DISEASES OF BRASSICA SPECIES IN SASKATCHEWAN, 1970-72
III. STEM AND ROOT ROTS[1]

G. Allan Petrie[2]

Abstract

Yield losses caused by stem rot fungi collectively were of little significance in a vast majority of the fields of Brassica campestris and B. napus sampled during the 3-year survey period. Nevertheless, the prevalence and incidence of footrot substantially increased. In approximately one-third of the fields of B. napus examined in 1972, over 50% of the plants had small basal lesions. Fusarium roseum 'Acuminatum' and Rhizoctonia solani were the principal pathogens isolated from footrot lesions. Sclerotinia stem rot decreased in importance, occurrring in 40% of the 1970 fields and 18% of the 1972 fields.

Introduction

The diseases to be considered are footrot, which has been attributed to Fusarium spp. and Rhizoctonia solani Kühn, and sclerotinia stem rot caused by Sclerotinia sclerotiorum (Lib.) de Bary. Leptosphaeria maculans (Desm.) Ces. & de Not., the fungus responsible for blackleg, will be considered part of the footrot complex in this report. Most of the survey data for blackleg have already been published (3).

Duczek and Morrall have published the results of an extensive survey of sclerotinia stem rot conducted in Saskatchewan in 1970 (1).

Methods

Techniques employed in field sampling and calculation of the disease severity index (DSI) have already been presented in detail (4). Disease severity classes for footrot and sclerotinia stem rot are defined in Table 1. Isolations were made routinely from field collections as described in an earlier paper (3). Methods used for seedling pathogenicity tests were also much the same as those previously described at length (2).

Results and discussion

The data for sclerotinia stem rot are in Tables 2 and 3. Those for footrot appear in Tables 2, 4, and 5. The geographical distribution of fields having the two diseases may be found by consulting Figure 1 in addition to the tables.

Table 1. Disease severity classes used for footrot and sclerotinia stem rot

Severity class	Description of symptoms: Footrot	Description of symptoms: Stem rot
0	No disease	
TR*	Discrete lesion near ground level 1 cm diam	
1	Lesion up to a few cm long but stem not girdled	Up to ¼ of stem rotted
2	Lesion up to several cm long girdling stem	From ¼ to ½ of stem rotted, plant ripening prematurely
3	Stem girdled, plant stunted, ripened prematurely, seed set reduced substantially	Over ½ of stem rotted, pronounced premature ripening, seed set substantially reduced

* Plants in this class were considered healthy when the disease severity index was calculated.

Sclerotinia stem rot decreased in prevalence from 1970 to 1972, most notably in Brassica campestris L. fields. In both this species and B. napus L. lesions were usually relatively small, occurring high up on the stems and probably resulting from ascospore infections. The average loss in yield was clearly much less than 1% in each of the 3 years.

The prevalence and incidence of footrot increased during this period, particularly in fields of B. napus (Table 2). In 5 of a total of 19 inspected in 1972, over 50% of the plants were infected. All five occurred

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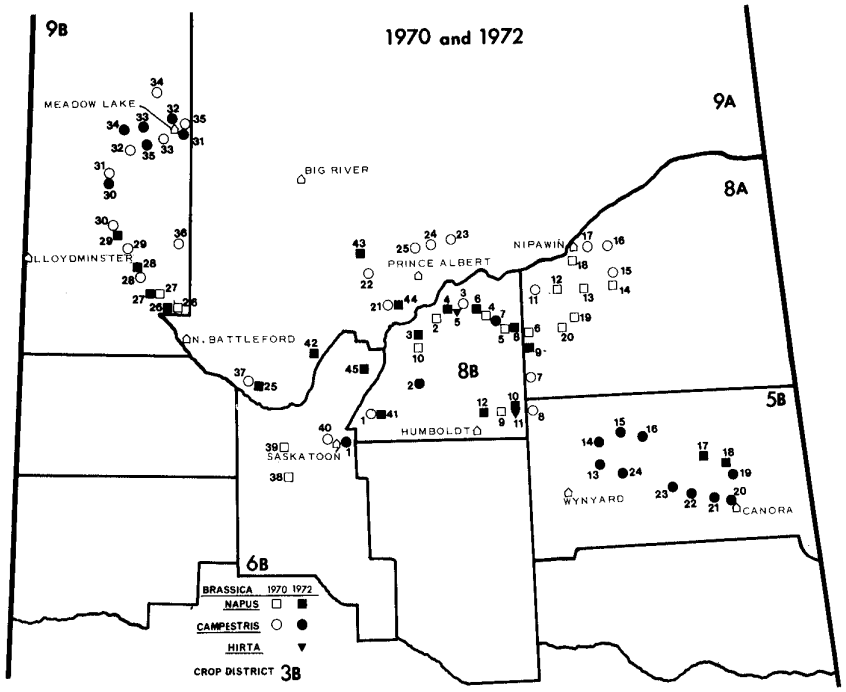

Figure 1. Commercial fields of *Brassica napus*, *B. campestris*, and *B. hirta* sampled during disease surveys conducted in Saskatchewan in 1970 and 1972.

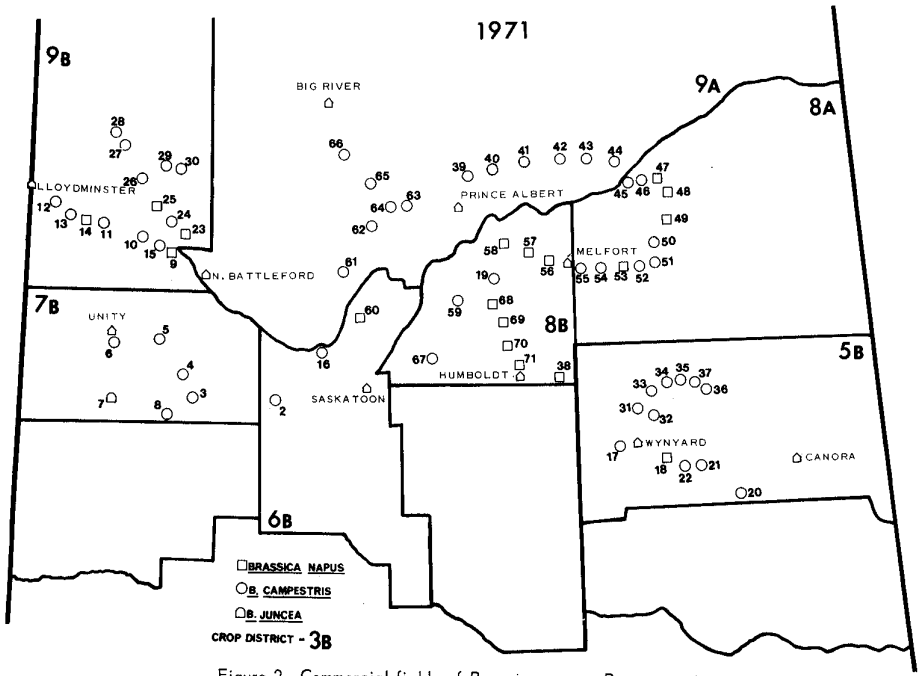

Figure 2. Commercial fields of *Brassica napus*, *B. campestris*, and *B. juncea* sampled in Saskatchewan during the 1971 disease survey.

Table 2. Prevalence, incidence, and severity of footrot and sclerotinia stem rot in Saskatchewan, 1970-1972

		Brassica napus			*Brassica campestris*			All fields*		
	Disease	1970	1971	1972	1970	1971	1972	1970	1971	1972
No. of fields		16	18	19	24	51	19	40	70	40
% of fields having the disease	footrot	88	83	100	58	82	94	70	83	97
	stem rot	50	39	32	33	12	6	40	19	18
% of plants per field diseased (avg)	footrot	6	21	25	1	7	4	3	11	14
	stem rot	4	1	<1	<1	<1	<1	2	1	<1
Avg DSI (%)	footrot	1	3	7	<1	1	1	<1	2	4
	stem rot	2	1	<1	<1	<1	<1	<1	<1	<1

* Including *Brassica hirta* and *Brassica juncea* fields (Figs. 1 and 2).

Table 3. Incidence and relative severity of sclerotinia stem rot in Saskatchewan from 1970 to 1972. Infection levels in individual fields*

Brassica napus						*Brassica campestris*					
Field no.	Crop District	% of plants infected	% of sites with plants infected	Highest % infection at any site	DSI	Field no.	Crop District	% of plants infected	% of sites with plants infected	Highest % infection at any site	DSI
					1970						
4	8B	1	17	8	1	3	8B	2	20	8	1
5	8B	17	100	32	8	11	8A	1	20	4	<1
12	8A	7	40	23	4	21	9A	2	17	12	2
13	8A	4	60	12	2	23	9A	1	20	4	<1
18	8A	3	40	8	2	24	9A	1	17	4	1
19	8A	2	40	4	1	30	9B	1	20	3	1
26	9B	2	40	4	1	33	9B	1	20	4	<1
27	9B	21	100	52	11	40	6B	1	20	3	<1
					1971						
14	9B	2	10	20	1	16	6B	1	10	10	1
25	9B	1	11	10	<1	24	9B	8	60	20	3
56	8B	5	50	10	2	42	9A	1	10	10	<1
57	8B	10	60	30	5	55	8A	1	10	10	<1
58	8B	4	30	20	3	61	9A	1	13	10	<1
60	6B	1	10	10	1	66	9A	1	13	10	<1
68	8B	1	10	10	1						
					*1972***						
3	8B	<1				34	9B	3	10	30	<1
8	8B	2	10	20	<1						
9	8A	<1									
10	8B	<1	10	10	<1						
12	8B	2	10	20	<1						
45	6B	<1									

* Fields having no infection are not listed.

** In fields 3, 9, and 45, no infected plants occurred in the samples pulled but one or two were observed elsewhere in each field.

in crop district 8B (Table 4 and Fig. 1). Nevertheless, overall yield losses were not substantial, as infection usually consisted of a small lesion from 0.5 to 1.5 cm long and less than 1 cm wide near soil level.

Observations made in experimental plots and survey results would appear to indicate differences in susceptibility of Brassica species to foot rot. In the only field of B. juncea (L.) Coss (field 7, 1971), over 55% of the plants had footrot symptoms. The average severity index was 16. These figures are considerably higher than those for B. campestris fields in the same area. In 1970 the percentage of footrot-infected plants in plots of B. juncea at Saskatoon was noticeably greater and the symptoms more fully expressed than in adjacent plantings of other Brassica spp. One might conclude from the survey results that varieties of B. napus are more susceptible than those of B. campestris to both foot rot and sclerotinia stem rot. Greenhouse inoculation experiments have shown differences in the rates at which different Brassica species succumb to stem rots (unpublished data). However, the results to date do not support the conclusion that B. napus is more susceptible than B. campestris. Footrot generally appears to develop rather late in the season. For this reason the earlier-maturing B. campestris may

Table 4. Prevalence and relative severity of footrot in Saskatchewan in 1970 and 1972. Infection levels in individual fields*

Brassica napus						*Brassica campestris*					
Field no.	Crop District	% of plants infected	% of sites with plants infected	Highest % infection at any site	DSI	Field no.	Crop District	% of plants infected	% of sites with plants infected	Highest % infection at any site	DSI
				1970							
2	8B	23	100	44	5	3	8B	1	20	4	<1
4	8B	11	83	16	3	11	8A	4	40	14	<1
5	8B	5	67	12	1	15	8A	1	20	4	<1
6	8A	19	100	26	1	17	8A	2	40	4	<1
9	8B	3	40	7	1	22	9A	7	67	23	4
10	8B	3	40	10	1	24	9A	1	17	8	<1
12	8A	19	100	35	1	29	9B	1	20	4	1
13	8A	6	60	15	<1	30	9B	2	40	7	1
14	8A	1	20	4	<1	31	9B	1	20	7	<1
18	8A	3	40	12	<1	32	9B	1	20	4	<1
19	8A	1	20	4	1	33	9B	1	20	4	<1
20	8A	1	20	4	<1	34	9B	7	80	11	1
26	9B	2	20	8	1	37	9A	1	40	3	<1
38	6B	<1				40	6B	4	60	10	<1
				1972							
3	8B	29	90	60	10	2	8B	7	60	20	2
4	8B	52	100	100	13	7	8B	7	50	20	2
6	8B	74	100	100	27	13	5B	1	10	10	1
8	8B	52	100	80	17	14	5B	2	30	8	1
9	8A	29	90	54	4	15	5B	8	60	33	3
10	8B	69	100	100	20	16	5B	4	50	11	2
12	8B	57	100	82	18	19	5B	2	20	14	1
17	5B	4	40	10	1	20	5B	4	50	8	1
18	5B	1	10	10	<1	21	5B	4	40	23	1
25	9B	6	30	33	2	22	5B	10	60	31	4
26	9B	1	10	9	<1	23	5B	1	10	11	<1
27	9B	3	20	17	<1	24	5B	6	50	18	2
28	9B	2	20	8	<1	31	9B	1	10	9	<1
29	9B	4	40	10	1	32	9B	5	40	20	2
42	9A	1	8	10	<1	33	9B	2	30	8	1
43	9A	10	70	27	2	34	9B	2	20	8	1
44	9A	17	88	30	4	35	9B	2	30	8	1
45	6B	<1			<1						

* Fields having no infection are not listed.

Table 5. Prevalence and severity of footrot in Saskatchewan in 1971. Infection levels in individual fields*

Field no.	Crop District	% of plants infected	% of sites with plants infected	Highest % infection at any site	DSI	Field no.	Crop District	% of plants infected	% of sites with plants infected	Highest % infection at any site	DSI
					Brassica napus						
9	9B	2	10	20	<1	57	8B	4	40	10	1
14	9B	42	90	70	8	58	8B	52	90	90	11
18	5B	8	30	40	2	60	6B	28	100	60	2
23	9B	5	30	30	<1	68	8B	28	90	70	4
25	9B	3	11	30	<1	69	8B	29	90	50	2
38	8B	36	100	100	5	70	8B	39	90	80	8
49	8A	31	90	90	4	71	8B	46	100	90	7
56	8B	16	80	40	<1						
					Brassica campestris						
3	7B	7	40	30	1	32	5B	20	60	60	5
4	7B	2	20	10	1	33	5B	6	50	20	<1
5	7B	14	70	30	2	34	5B	14	80	50	3
6	7B	10	70	20	1	36	5B	3	10	30	1
8	7B	10	60	30	1	37	5B	1	10	10	<1
10	9B	6	40	20	1	39	9A	3	10	30	1
11	9B	20	80	60	5	43	9A	4	38	10	1
12	9B	8	40	30	2	44	9A	4	25	20	2
13	9B	1	10	10	<1	46	8A	2	20	10	1
15	9B	2	20	10	<1	51	8A	1	10	10	<1
16	6B	4	30	20	3	52	8A	11	80	20	3
19	8B	13	70	60	3	54	8A	5	30	30	1
20	5B	7	50	20	3	55	8A	15	70	40	2
21	5B	6	20	40	2	59	8B	14	70	50	3
24	9B	18	50	70	3	61	9A	9	56	30	<1
26	9B	7	60	20	2	62	9A	6	50	20	1
27	9B	1	10	10	<1	63	9A	11	60	30	2
28	9B	4	30	20	2	64	9A	3	30	10	1
29	9B	7	40	30	<1	65	9A	10	60	20	2
30	9B	14	60	40	1	66	9A	31	88	50	7
31	5B	16	70	40	5	67	8B	12	60	30	1

* Fields having no infection are not listed.

Table 6. Relative proportions of the principal fungi obtained upon plating stem bases with footrot symptoms*

Year	No. sampled		Percentage of total stems yielding						
	Stems	Fields	*Fusarium*	*Fusarium* & *Rhizoctonia*	*Rhizoctonia*	*Fusarium* &/or *Rhizoctonia*	*Leptosphaeria maculans*	*Sclerotinia sclerotiorum*	*Alternaria alternata*
1970	37	12	87	54	57	89	16	3	57
1971	179	27	84	36	40	88	18	1	68
1972	181	27	85	40	48	93	4	1	54
Avg	397	66	85	40	45	90	11	1	60

* The *Fusarium* cultures were almost all *F. roseum* 'Acuminatum'. Those of *Rhizoctonia* were *R. solani*.

Table 7. Results of a representative seedling pathogenicity test in which isolates of *Fusarium roseum* and *Rhizoctonia solani* from footrot-infected plants were compared

Species	No. of isolates	Disease severity index (%)*			
		Brassica napus var. Zephyr		*Brassica campestris* var. Span	
		Avg DSI	Range in DSI's	Avg DSI	Range in DSI's
Fusarium roseum	23	33	3-100	29	3-70
Rhizoctonia solani	17	93	73-100	97	83-100

* Calculated according to the formula used for field survey material (4) with the exception that severity classes 1, 2, and 3 equalled 0-25%, 26-50%, and 50-100% of a seedling destroyed, respectively.

tend to escape the disease. Many Sclerotinia infections seen in late summer also were small and apparently of recent origin.

Basal segments of approximately 400 stems with footrot symptoms from 66 fields were plated. The principal fungi obtained in culture are shown in Table 6. Fusarium occurred in 85% of the stems. All of the considerable number of isolates identified to species belonged to F. roseum Lk. emend. Snyder & Hansen, and all but a very few could be further classified as F. roseum 'Acuminatum'. Rhizoctonia solani was the second most common pathogen but was isolated from only 45% of the lesions. Leptosphaeria maculans was found in 11% of the platings. Sclerotinia was isolated infrequently from stems having typical footrot symptoms (Table 6), showing that the two stem rots can be reliably distinguished by appearance. The rather frequent occurrence of Alternaria alternata (Fries) Keissler suggests, perhaps, a role of some importance as a secondary invader. Nematodes were found in 6% of the isolations in 1971, occurring in significant amounts in material from only two of the 27 fields.

The results of a representative seedling pathogenicity test comparing isolates of Fusarium roseum and Rhizoctonia solani from footrot-infected plants are summarized in Table 7. The Fusarium cultures were generally considerably less virulent than those of Rhizoctonia, with severity ratings exhibiting a great deal more variation.

Acknowledgments

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A KEY TO STANDARDIZE THE DESCRIPTION OF GROWTH STAGES IN TURNIP RAPE, BRASSICA CAMPESTRIS

F.R. Harper[1]

Abstract

The growth of plants of Span rape (Brassica campestris) in a greenhouse under supplemental light was recorded periodically by photography. Six growth stages were recognized: 0 = preemergence, 1 = seedling, 2 = rosette, 3 = stem elongation, 4 = flowering, and 5 = seed ripening. Brief descriptions of the stages are given.

Introduction

The division of the growth cycle of crop plants into readily recognizable stages that can be identified by a number or a standard term has been described for small grains, corn, cotton, and tobacco (1). These standardized descriptions of growth stages have proved useful to plant pathologists, entomologists, and agronomists who wished to relate their observations to the developmental morphology of the crop rather than to a time period.

No standard description of the development of turnip rape (Brassica campestris L.) or other closely related species is available. Turnip rape has become an important oilseed crop on the Canadian prairies. A considerable amount of research is being carried out on the crop and its diseases and pests. The present study was undertaken to identify and describe the easily recognized stages in the development of the rape plant as integral part in the development of methods for assessing disease losses.

Materials and methods

Turnip rape cv. Span was grown from November to January and again from April to June in a greenhouse maintained at 20°C in 15 cm clay pots filled with a 3:1:1 soil-sand-peat potting mixture. The plants were thinned to one per pot in the seedling stage, spaced on a greenhouse bench and supplied with supplemental illumination for 8 h per day. When flowering commenced, pollen was collected daily and all open flowers were manually pollinated by means of a camel-hair brush.

Growth was recorded on 35 mm film every 3 to 7 days during the growth cycle. A reference scale was included in each frame. Line drawings were prepared by tracings from the projected slides. Stems developing from axillary buds were not depicted.

Results and discussion

The several easily recognized morphological stages in the growth of B. campestris are depicted in Figure 1. Each stage is described in terms of the main stem.

Stage 0 (preemergence) comprises the period of development from the start of inhibition, through elongation of the seedling axis, to the emergence of the cotyledons from the soil.

Stage 1 (seedling) extends from emergence of the cotyledons from the soil to the unfolding of the first true leaf which is normal in appearance. Frequently the first-formed true leaf, and occasionally the second one, partially expands and quickly becomes senescent.

Stage 2 (rosette) begins when the first normal leaf is unfolded and terminates when the stem begins to elongate. Four to seven petiolate leaves unfold at this stage. Stem length remains essentially unchanged although stem girth increases. The cotyledons and frequently the first one or two true leaves are senescent by the end of Stage 2.

Stage 3 (stem elongation) commences with elongation of the internodes of the expanded leaves and ends when the first flower opens. The remaining (sessile) leaves of the main stem unfold and the internodes elongate to near-maximum. The inflorescence enlarges and the rachis elongates, separating the pedicels of the first few flowers. The main stem reaches 30 to 60 percent of its maximum length by the end of Stage 3.

Stage 4 (flowering) is signaled by the opening of the first flower on the terminal raceme of the main stem and ends with

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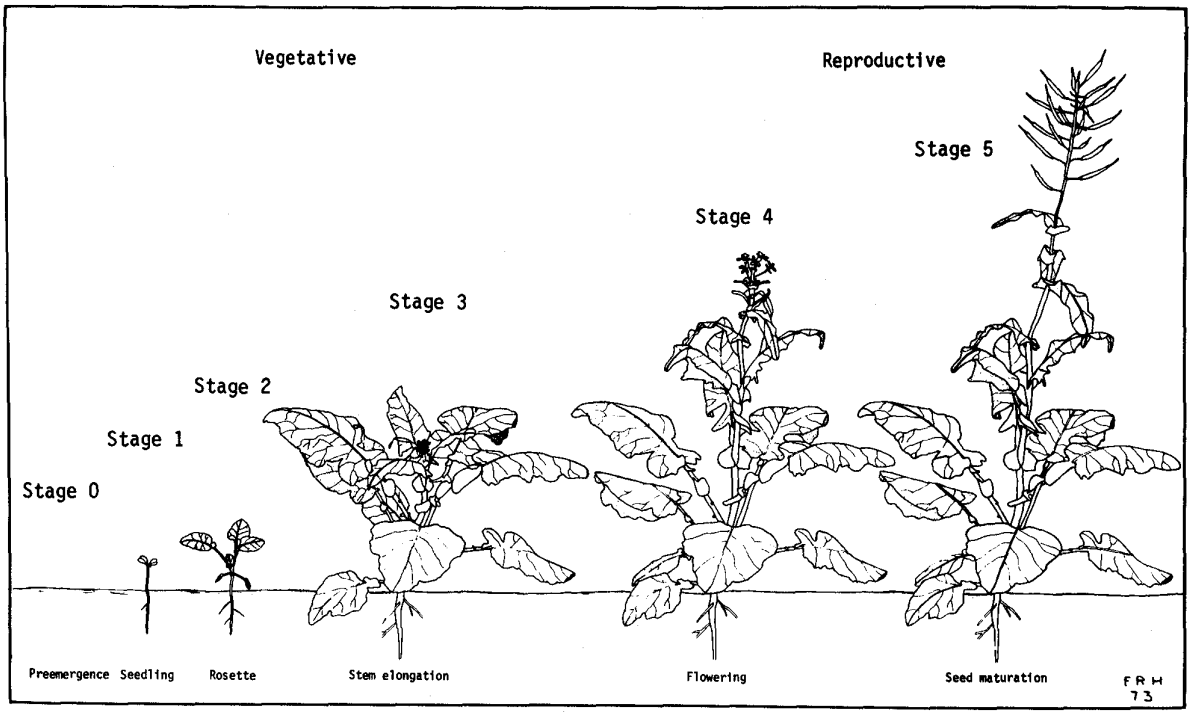

Figure 1. Stages in the growth of turnip rape (*Brassica campestris* L.)

incipient petal fall of the last flower on this raceme. Flowering progresses in the typical racemose pattern from base to apex. There is a moderate increase in plant height from elongation of the rachis of the inflorescence and from elongation of the internodes separating the uppermost leaves on the main stem. Axillary buds of the upper leaves, and occasionally some of the lower ones, become active and produce a stem, one to four sessile leaves and a terminal inflorescence. When environmental conditions are favorable, the racemes on the axillary stems will continue to flower for some time after flowering has finished on the main stem.

Stage 5 (seed ripening) begins with visible elongation of the ovary and incipient petal fall by the last-formed flower on the inflorescence of the main stem. It ends when all seeds of the plant have attained their maximum size and mature color. By the start of the seed-ripening stage, the siliques produced by the first-formed flowers have almost completed their elongation, their beaks are clearly discernable and their girth is approaching maximum. During this stage the siliques attain maximum size and change from green, through yellow to brown. The seeds achieve their final size and develop their mature reddish-brown color. Finally the siliques separate along the sutures and the seed is released. In western Canada the rape crop is generally swathed when 20 to 25 percent of the seeds have begun to turn from green to brown (2).

Duration of growth stages

The length of each growth stage in B. campestris is greatly influenced by temperature, moisture, and other factors in the environment of the plant. However, a rough approximation of the duration of the several stages, based on greenhouse and field observations is:

Stage	Description	Duration in days
0	Preemergence	4 to 6
1	Seedling	4 to 6
2	Rosette	18 to 25
3	Stem elongation	4 to 7
4	Flowering	7 to 14
5	Seed maturation	highly variable

Development of axillary stems

A secondary stem may emanate from any axillary bud on the main stem. The buds of the sessile upper leaves generally become active first with the development of a stem, one to four leaves, and a terminal inflorescence. However, in exceptional circumstances, for example, where stands are sparse or flea beetles have caused early, severe damage, the buds of the lower, petiolate leaves may also develop into flowering branches. Tertiary stems bearing

inflorescences occasionally develop from the axillary buds of the sessile leaves on the secondary stems.

Addendum

Since this manuscript was submitted for publication; another key to the growth stages of rape has been published (3).

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AERIAL SURVEY FOR BACTERIAL BLIGHT, 1970[1]

V.R. Wallen[2], D. Galway[2], H.R. Jackson[3], and L.E. Philpotts[4]

In 1968, the first plant disease survey of field beans by aerial infrared photography in Canada was conducted (7) as part of a national crop loss assessment program to relate the intensity of disease in crops to actual losses in the field. The survey revealed that with adequate ground truth and the ability to relate ground truth observations to film records, bacterial blight could be identified by means of the patterns of foci in the field; based on film records the % area affected in each field could be determined by a drum scanner technique (8).

To date, the best method of controlling bacterial blight in Canada is to introduce each year breeder seed stocks from Idaho and California. The resulting Select plots are usually free from common blight but sometimes contain traces of infection that are usually detected by plant pathologists during crop inspections. Any affected plots are discarded for future pedigreed seed stocks. However, despite stringent regulations, a gradual buildup of infection takes place in the following generations of seed crops produced in southwestern Ontario, and new breeder seed must be imported each year to keep infection at as low a level as possible.

Aerial photography is being used to ascertain the general incidence of bacterial blight in the field bean crop and to monitor the effectiveness of the program of importing disease-free breeder seed. In 1970, certain Select, Foundation, and Certified crops in two areas of southwestern Ontario near Hensall and Chatham were surveyed by aerial photography and by direct inspection on foot. Bacterial blight is also being used as a model in an attempt to develop better plant disease survey techniques.

Methods

During August 1970, extensive ground-truth studies were carried out in bean fields under two flight paths. Forty-two fields of white beans (Phaseolus vulgaris L.) in the Chatham area and forty fields in the Hensall area were examined prior to August 10 in the first survey and prior to August 24 in the second survey for the presence of bacterial blight. As bacterial blight is seed-borne, producing foci of varying size based upon secondary spread of the pathogen, field infection varies throughout the fields and no set pattern of field survey and no realistic estimate of disease percentage are possible. The survey here was conducted to obtain a visual knowledge of the location and approximate size and severity of infection foci so that this information could be correlated with aerial photographs for disease interpretation. Select plots of 1 to 2 acres were completely surveyed and larger fields were surveyed until the disease pattern in the field was evident. Diagnosis was confirmed by sending leaf samples from affected plants to the Ottawa laboratory where the causal organisms, Xanthomonas phaseoli (E.F. Sm.) Dows. and Xanthomonas phaseoli var. fuscans (Starr. & Burkh.), were isolated and identified (9). The first survey was conducted to locate initial foci and the second to indicate the extent of secondary infection.

Kodak Ektachrome Aero Film 8443 was used in conjunction with a Zeiss B (yellow) filter and processed as a positive from which reversal prints were made. A Zeiss camera with a 12-inch focal length lens and an exposure of 5.6 at 1/300 of a second was used. Flights were made on August 16 and 23 at two altitudes to produce scales of 1:3600 and 1:9600.

The photography of the Hensall area was chosen for a more detailed study and after disease interpretations were made field infection percentages were determined by the drum scanner method (8).

Results and discussion

There was excellent correlation between the ground truth survey and the aerial photography survey. In both the Hensall and Chatham areas all affected fields detected by ground truth and confirmed by laboratory identification of the organism were similarly detected on film by infrared aerial color photography (Table 1). Two fields detected on film but not by ground survey contained 0.1% and 0.4% infection. Otherwise the only differences were in the amounts of infection detected in the fields. Field notes taken during the ground truth survey did not indicate as much infection as was shown by photography. Fields 1, 8, and 19 in the Hensall area showed, respectively, 37%, 21%, and 30% infection by aerial photography (Fig.

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Table 1. Incidence of bacterial blight of field beans in the Hensall and Chatham areas, 1970

Area	No. fields surveyed	Acreage	No. fields affected		Causal organism*	
			Ground truth survey	Aerial IR photography	*X. phaseoli*	*X. phaseoli* var. *fuscans*
Chatham	42	836.21	18	18	14	7
Hensall	40	882.7	29	29	16	14

* In four fields both *X. phaseoli* and *X. phaseoli* var. *fuscans* were isolated.

1), but did not appear to be infected to this extent by visual observations.

X. phaseoli and X. phaseoli var. fuscans were isolated from infected leaf samples in approximately equal numbers from the Hensall area and in a ratio of 2 to 1 in the Chatham area (Table 1). This is in contrast to the epiphytotic years of 1961-62 when X. phaseoli var. fuscans was the predominant causal organism (10).

In 1970, 72.5% of the 40 fields in the Hensall area were affected with bacterial blight. The extent of infection ranged from less than 0.1% in some fields to 37% in others. On an area basis 57.9 acres of a total 882.7 acres were affected (Fig. 1). In 1968, 76.5% of the fields or 33.5 out of 809.93 acres were affected. In 1970 in the Chatham area, 42.9% of the fields were affected. It would appear from these figures that there was little difference in the overall infection level between 1968 and 1970, ranging from 4% to 6% of the crop. This level of infection occurred despite the importation of Breeder seed. It will be some time before the full effects of the program are known.

Beginning in 1957, the variety Sanilac was introduced because of its resistance to anthracnose, and subsequently a steady increase in the incidence of bacterial blight occurred in Ontario until in 1961 and 1962 it reached epiphytotic proportions. This increase coincided with the increasing acreage of the blight-susceptible variety Sanilac and a decline in acreage of the anthracnose-susceptible variety Michelite (1,2). In 1963, bacterial blight was at a low level primarily because of unfavorable conditions for blight development and spread, despite a high level of seed infection (3). In 1964, essentially the same situation prevailed because of a lack of seed-borne inoculum, and only 3 of 27 registered fields inspected showed symptoms of bacterial blight (4). In 1965, the plan to import Breeder seed free from bacterial blight was initiated. In that year, infection was found in 80% of the fields originating from Ontario-grown Seaway seed, but in only 20% of the fields originating from Michigan-grown Foundation Seaway seed (9). In 1966, none of the plots originating from imported Breeder seed were affected, and low infection levels were recorded in Registered and Certified crops (5). However, in 1967, 16 of the 24 Select plots produced from imported Breeder seed were affected by bacterial blight (unpublished data). This unusually high incidence of blight in imported seed resulted from a blight epiphytotic that flared up in Idaho the previous year. The infection in the Select plots in 1967 resulted in infection in all registered fields inspected in 1968 (6). Despite the high level of seed infection in 1968, the 1969 crop was relatively free from blight because of extremely dry growing conditions. It seems obvious that if bacterial blight is to be maintained at a low level or eliminated, Select seed plots must be kept free of bacterial blight by careful inspection and roguing. The major changes in blight incidence since the beginning of the program are a reduction in the percentage of plants affected and a reduction in pod infection. By 1970, fewer foci were present in the fields initially and, while secondary spread and infection resulted in leaf infection, the incidence of pod infection was much less, and yield was not affected. However, the 4% to 6% crop infection figures appear high, and should be reduced through a vigorous program of continually monitoring Select plots for disease.

Prior to 1968, fields were surveyed and rated primarily for the presence or absence of bacterial blight together with estimates of disease severity. Aerial photography can be used successfully to determine the percentage of field infection and to aid in determining not only year-to-year changes in incidence of disease, but also the practical success or failure of long range control measures.

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Figure 1 AERIAL SURVEY FOR BACTERIAL BLIGHT
Nominal scale of figure 1:19,200 ▶
NAPL Roll No. A30287, frames 7 to 37

YIELD LOSSES IN CRANBERRY IN NOVA SCOTIA, 1969-72[1]

C.L. Lockhart, I.V. Hall, and R.A. Murray[2]

Abstract

Over a 4-year period, 1969-72, in two cranberry (Vaccinium macrocarpon) bogs in Nova Scotia the percentage of fruit lost at harvest ranged from 1.9 to 5.7 from green berries, 1.4 to 4.3 from small berries, and 1.2 to 16.3 from decay. Sterile breakdown accounted for 21% of the loss from decay in 1970 and 46% in 1972. These data are discussed in relation to cultural practices.

Introduction

Field decay losses in cranberries in Massachusetts were 2.2 to 3.89% in 1917 (1) and 5.2% in 1969 (7) and were as high as 50% in New Jersey in 1940 (8). Gourley and Harrison (4) reported that fruit rots seldom occur under field conditions in Nova Scotia.

This report presents data yield losses at harvest in two bogs that accounted for more than half of the cranberry production in Nova Scotia during 1969-72.

Materials and methods

Through the cooperation of a local grower we obtained information for the crop years 1969-72 on the total yield of freshly harvested cranberries, the quantity of marketable fruit packed, and the amount of fruit lost during cleaning and because of greenness, small size, and decay (Table 1). The cranberries (Vaccinium macrocarpon Ait.) were a native selection and came from a total of 14 acres in two bogs.

In 1970 and 1972, 5 kg samples of cranberries that had been discarded by the packing crew were examined and the decayed berries picked out. A section of each decayed berry was plated on potato-dextrose agar (PDA) to identify any fungi and bacteria present (Table 2).

Results and discussion

Over the 4-year period, 1969-72, the average loss of cranberries at harvest time from decay was slightly larger than the combined average losses from green and small fruit and from cleaning (Table 1). The highest loss from decay occurred in 1969 and the lowest in 1972; in those years the percentages of berries marketable were 79.5% and 91.9%, respectively.

The average losses of cranberries from decay in Nova Scotia (Table 1) were similar to those reported for the Cape Cod area in Massachusetts (7). The increased use of

Table 1. Yield losses in two cranberry bogs in Nova Scotia, 1969-72

Year	Total yield (lb/acre)	Marketable yield (%)	% fruit loss at harvest from				lb fruit cleaned/hr
			Green	Small	Decay	Cleaning	
1969	53,412	79.5	2.2	1.4	16.3	0.6	19.5
1970	66,500	84.5	5.7	2.3	7.5		67.6
1971	48,390	88.6	1.9	4.3	5.2		98.8
1972	85,195	91.9	2.9	3.5	1.2	0.5	84.3*
Avg	63,374	86.1	3.1	2.8	7.5	0.5	67.5

* In 1972 the time required for placing fruit in cold storage was included in the cleaning time.

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Table 2. Incidence of fungi isolated from decayed cranberry fruit at harvest

Fungus	Percent incidence in 1970	Percent incidence in 1972
Acanthorhynchus vaccinii Shear	0	1
Diaporthe vaccinii Shear	0	3
Godronia cassandrae Pk. f. *vaccinii* Groves	21	21
Glomerella cingulata-vaccinii Shear	0	6
Guignardia vaccinii Shear	0	8
Monilinia vaccinii-corymbosi (Reade) Honey	25	0
Penicillium spp.	21	11
Pullularia pullulans (de Bary) Berkh.	4	0
Sporonema oxycocci Shear	0	4
Unidentified fungus	8	0
Sterile breakdown	21	46

fertilizer for high production, especially those containing a high percentage of N, will result in heavier vine growth, and growers may be faced with an increased incidence of field rots. In years in which prolonged wet periods are forecast during the bloom period growers should consider the use of a fungicide. In Wisconsin, Carlson and Boone (2) reported that rots can be controlled by spraying with fungicides at 10-day intervals from mid-July to mid-August. Ferbam (76% WP) at the rate of 6 lb per acre was applied during bloom to the two Nova Scotia bogs in 1969 on July 2, 19, and 31. There were 4 days of wet weather on July 11-14 and the plants were largely unprotected for most of this wet period. Cross (3) states that fungicides applied during the bloom period were the only ones to have given significant reduction in the number of rotted berries, both field and storage rots.

Berries from the 1971 and 1972 crops were much easier to clean than those from the 1969 crop (Table 1). The higher percentage of rots in 1969 was largely responsible for increased cleaning time. The organisms isolated from decayed berries are shown in Table 2. These organisms are all generally associated with decay of cranberries (4,6).

The increased incidence of sterile breakdown in 1972 can be associated with several frosts in late September and in October. Frosts occurred on September 21, 22, 24, and 29 and October 3 and 4, with three heavy frosts of 23°F (-5.0° C) in mid October; in 1970 the same periods were relatively free from frosts.

The marked decrease in yield (Table 1) for 1971 was due to a heavy frost on June 27. In one of these bogs the irrigation system failed completely resulting in extensive injury to growing shoots (5) and to developing blooms.

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FIREBLIGHT IN SOUTHERN ONTARIO IN 1972

J. Dueck and H.A. Quamme[1]

Abstract

Observations for fireblight infection were made in 25 pear and apple orchards in southern Ontario in 1972. The symptom most commonly observed was twig blight, which in the Harrow area was not preceded by blossom blight. Bacteria from infected shoots were isolated on a selective medium to confirm visual identification of symptoms. The incidence of blight was generally light; however, in 8 of the orchards damage of economic significance occurred. Frequency of blighted shoots appeared to be more related to age of orchard, soil fertility, variety, and presence of inoculum than to local climatic conditions.

Fireblight caused by Erwinia amylovora (Burr.) Winsl. et al. is the most potentially destructive disease of pear and apple in southern Ontario. The occurence of the disease is sporadic and unpredictable, but each year a few orchards have a serious outbreak. Occasionally the disease becomes widespread. In 1972, observations were made on 25 pear (Pyrus communis L.) and apple (Malus pumila Mill.) orchards in the Harrow, Arkona, Cedar Springs, Simcoe, and Vineland areas (Fig. 1). Nine orchards in the Harrow area (Essex County) were observed throughout the growing season, and the other orchards

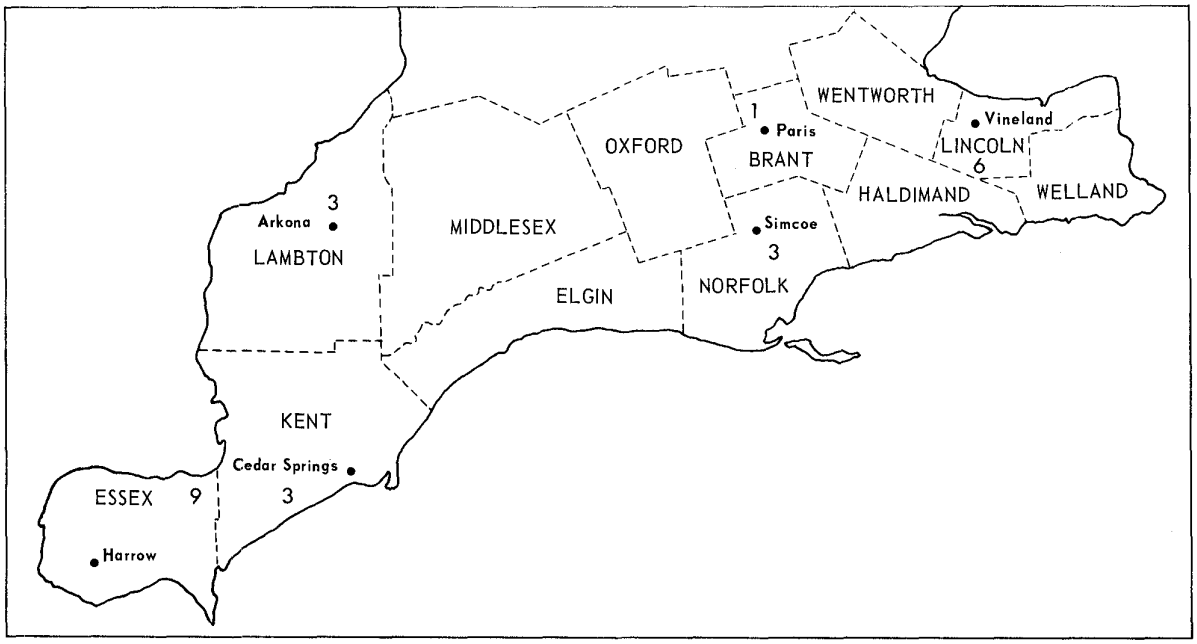

Figure 1. Location and number of orchards observed for fire-blight severity in southern Ontario, 1972.

were visited once in late July, August, or September.

Identification of fireblight was made by visual recognition of macroscopic symptoms on the trees, by the appearance of bacterial colonies isolated from infected shoots on Crosse and Goodman's selective medium (1,2), and by subsequent pathogenicity tests of single colonies on immature Bartlett pear fruitlets (Fig. 2) or on succulent shoots of potted Bartlett pear trees in the greenhouse.

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[2] Selective medium: sucrose 400 g, Difco nutrient agar 30g, crystal violet solution (0.1% W/V in absolute ethanol) 2 ml, 950 ml water, 1% actidione 5 ml (added to cool autoclaved medium).

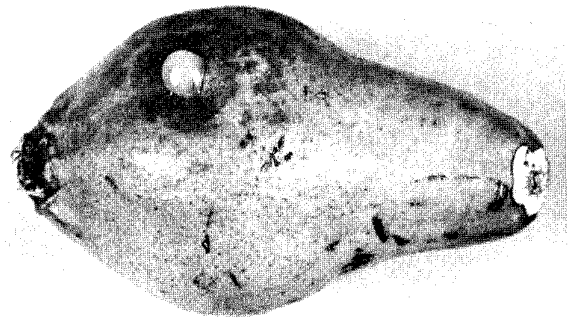
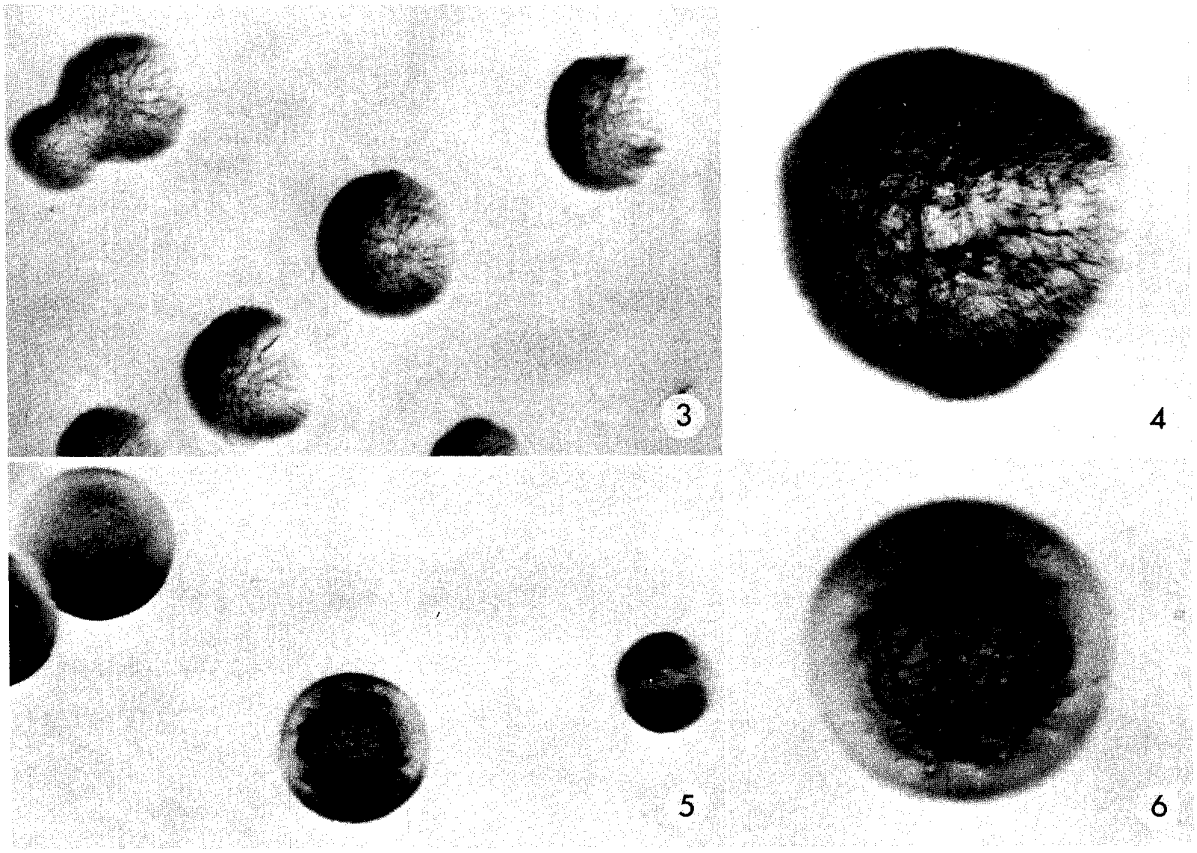


Figure 2. Bacterial ooze and watersoaking in immature Bartlett pear fruit 4 days after inoculation by stabbing with a dissecting needle dipped in a pathogenic colony of *Erwinia amylovora* and incubation in a moist chamber at 21°C.

Observations

On the selective medium colonies of E. amylovora isolates from both apple and pear were convex, round and had a smooth margin (Figs. 3-6). They appeared uniquely striated when viewed with transmitted incandescent light through a stereoscopic dissecting microscope after 48-72 h of incubation at 28 C (Figs. 3-6). Colonies of some of the isolates obtained from pear were more cone shaped than convex, had darker striations in the center, and were not striated at the margins (Figs. 5,6). The cratered appearance of colonies described by Goodman (2) was observed only in very young colonies. Colonies identified as E. amylovora on the selective medium were invariably pathogenic.

In Essex county the symptom most commonly observed is twig blight, with the characteristic production of bacterial ooze, followed by necrosis. Bacterial strands described by Ivanoff and Keitt (3) are occasionally found on infected shoots in the greenhouse (Fig. 7) but have not been observed in the field. Apparently they are not important for dissemination of bacteria in this area. Trunk cankers known to occur in 'Magness' pear (3) have been observed only in a few seedling trees in the progeny test orchard at the Research Station, Harrow. In pear, fruit may also be infected, both in the very immature stage and late in the season. Late-season fruit infection occasionally



Figures 3-6. Colonies of *Erwinia amylovora* on Crosse and Goodman's crystal violet selective medium after 48-72 hr incubation: 3,4) isolates from apple; 5,6) isolates from pear. (Enlarged)

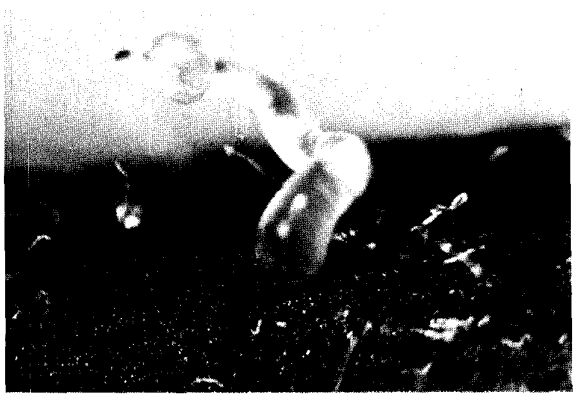


Figure 7. Crystalline bacterial strands on infected Bartlett pear shoot. Strands were highly soluble in water but insoluble in 95% ethanol and in 3% glutaraldehyde.

occurs in the absence of twig infection. Blossom blight is not a serious problem and in 1972 was observed on only one pear tree. The absence of blossom blight may be due to a low level of inoculum in spring. In an artificial inoculation experiment 99 of 100 blossom clusters became infected in 4 to 5 days when atomized with a 20-Klett-unit suspension (ca. 10^8 cells/ml) of E. amylovora during full bloom. Ninety-two of the infections developed into twig blight. These results suggest that environmental factors are not limiting to infection if inoculum is present.

The incidence of blight in the Harrow area was generally light throughout the season, with some infection occurring in 6 of the 9 orchards visited. Four of these plantings, consisting respectively of Lodi apple, Bartlett, Clapp's Favourite and Gifford pear, had damage to scaffold branches in 5% to 10% of the trees. Twig blight first appeared in the second week of June. In the Lodi orchard which had a history of fireblight epidemics, two severely blighted trees were found on July 19 in a row where no control measures had been taken. Blight continued to progress and new infections occured in these and adjacent trees through August, although growth had stopped in mid-July. Apparently in a highly susceptible variety such as Lodi, infection can occur after terminal growth has ceased. In the same orchard in a block of Bartlett pears also with a perennial blight problem, excellent control was obtained by applying streptomycin sulfate (100 ppm) in two sprays during the bloom period and one post-bloom spray.

Orchards in the Arkona area were visited because of a particularly serious outbreak of blight in a 5-yr-old high density trellised stand of Golden Delicious apples on East Malling IX rootstock. In the block of approximately 3 acres, all trees were infected, with many infections extending into 3- and 4-year wood (Fig. 5). Red Delicious trees interspersed at regular intervals in the stand showed only a trace of infection. The block was surrounded with Bartlett pear trees of which less than 25% were blighted, and only a few had damaged scaffold branches. In an adjacent nursery containing three apple varieties on the east side of the infected Golden Delicious block and surrounding pear trees, nearly all Idared trees, approximately 25% of Spartan, and less than 5% of Red Delicious trees were infected. The severity of blight decreased with increasing distance from the pear trees. Although the Bartlett pear trees were not seriously affected, they apparently served as a source of inoculum for the succulent young apple trees in both the Golden Delicious block and the nursery.

In a second orchard in the Arkona area, 75% to 100% of Idared, Lodi, Quinte, Tallman's Red, and Tydeman's Red apple trees were infected. While the incidence of blighted shoots was high, infection had not progressed into the scaffold branches because of continuous pruning of newly infected shoots. In the same orchard, Red Delicious and McIntosh trees were free of blight. A third orchard, predominantly Bartlett pear, had only a trace of fireblight.

All orchards visited in the Cedar Springs area consisted of blocks of Bartlett pear and several varieties of apples, whereas near Simcoe only apple orchards were visited. There was a low incidence of fireblight in both areas.

An orchard near Paris had a block of Bartlett pear surrounded by blocks of several varieties of apples. The pear trees were severely blighted, with nearly all trees showing some level of infection. Where infection was most severe, adjacent apple trees of the resistant variety McIntosh had a low level of infection in 1- and 2-year wood. In a 3-yr-old planting of Idared apples also adjacent to the pears, more than 50% of the trees were infected. As in the Arkona orchard, the pear trees appeared to be the source of inoculum.

Two of six pear orchards visited in the Vineland area had moderately severe blight problems. In one planting of Bartlett, 75% of the trees were infected, most of them into 2- and 3-year wood. In a second orchard 25% of Bartlett and Bosc trees were infected. Entire trees had been destroyed and the grower was having difficulty establishing replacement trees because of fireblight. In a third orchard of 6-year-old Bartlett pear a uniform infection of 5-6 shoots on every tree was observed. In the other orchards in the area zero to light infection was observed.

In general, the incidence of fireblight in 1972 was not critical. In most cases it could probably have been controlled with 4 to 6 appropriately timed applications of streptomycin sulfate and by pruning out

infected branches. It was difficult to associate any effect of climatic factors with frequency of occurrence of fireblight. Other factors affecting susceptibility such as age of orchard, soil fertility practices, varieties, and presence or absence of inoculum appeared to be of greater importance. It was apparent, however, that favorable conditions for development of serious blight problems could arise in all pear and apple growing areas of southern Ontario.

Because of its sporadic occurrence, an extensive survey would be required to accurately estimate losses due to fireblight. However, the disease can spread at an explosive rate from apparently minor infections to destroy entire orchards of susceptible pear and apple varieties. Furthermore, growers have been reluctant to use streptomycin in a control program. Instead, orchards are fertilized inadequately to reduce the amount of susceptible succulent growth. As a result, productivity is low and fruit size and quality are adversely affected. The threat of fireblight also deters growers from putting in new plantings of susceptible, but otherwise desirable varieties. Thus it is imperative to consider the disease more than in terms of current year's losses.

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RUSTS THAT PASS IMPORT INSPECTION[1]

D.B.O. Savile

The problem of invisibly infected or contaminated plant materials passing even meticulous import inspections is widely recognized for, e.g., virus diseases of bulbous plants. Recent experiences suggest that more attention should be directed to some rusts which are not visible in shipments of dormant material.

A familiar example of seed contamination is the repeated introduction into North America of Puccinia carthami with seeds of safflower, Carthamus tinctorius. The occurrence of several morphologically distinguishable biotypes, both in Canada and in United States, shows that the pathogen was introduced repeatedly from various sources (5). Examination of seed from infected crops under the dissecting microscope reveals many teliospores adhering to the oily seed coat; and such seeds planted in an isolated garden produced seedlings with pycnia (3). In poorly cleaned seed lots infection probably also results from the presence of rusted leaf fragments; but the spores on the seed coat defy detection by the most careful naked-eye inspection. The recent occurrence of Puccinia helianthi in New Zealand (specimen received from Miss J. M. Dingley) may have originated from either included rusted fragments or from spores on the seed coats. Some biotypes of P. helianthi have moderately firm teliospore pedicels, but in others the pedicels are fragile and irregularly deciduous. Surface sterilization should clearly be practised when seed of such crops is imported into disease-free areas.

Chrysomyxa spp. overwinter as dormant mycelium in evergreen leaves, especially of various Ericaceae, without any symptoms. The mycelia give rise to uredinia and /or telia in the spring. Only by growing imported stock under quarantine can such infections be detected. The introduction of Chrysomyxa ledi var. rhododendri on Rhododendron in coastal Oregon or Washington about 20 years ago presumably resulted from the importation of such invisibly infected planting stock.

Various species of Melampsora that attack willows and poplars have the ability to persist on these plants in the absence of the aecial host. Urediniospores enter the young winter buds before they are sealed by the hard and resinous scales. Mycelium penetrates the embryonic leaves, grows with them when the buds break in spring, and the leaves open bearing "instant uredinia", which allow a rapid build-up of the rust. Melampsora populnea (M. aecidioides) has long been recognized to winter in this manner, and it was presumably introduced into North America (1) (Rhode Island, Colorado and the Pacific states) in planting stock of Populus alba L. with such dormant infections. It is also recognized (4) that most of the arctic and subarctic willow rusts of the Melampsora epitea complex winter chiefly or solely by this means, which has occasionally been seen also in related temperate rusts.

It is now painfully clear that this ability to overwinter in dormant buds is shared by other Melampsora spp. on poplars. In February 1972 Dr. John Walker sent me specimens of Melampsora medusae, which had suddenly appeared on Populus deltoides Bartr. in New South Wales. In March 1973 Mr. D. T. Hartigan sent me specimens, also from New South Wales, of a rust on Lombardy poplar, P. nigra var. italica Muenchh., which proves to be the European M. larici-populina.

Melampsora medusae is endemic to North America, but has recently been reported in Europe, perhaps first in 1943 (2, p. 132). M. larici-populina is endemic to Europe, but we have Japanese specimens on P. nigra var. italica. These movements of both rusts, and their recent introduction into New South Wales, almost certainly result from the practice of shipping cuttings of many clonal selections to many countries for experimental planting, a practice that I understand to be widespread.

The only practical means of curtailing these dangerous introductions seems to be to limit shipments to small quantities that can be grown in post-entry quarantine with rigid inspection of every leaf.

As I write this note, word comes from Dr. Walker that Melampsora medusa and M. larici-populina have just been discovered in New Zealand. It is conceivable, as Dr. Walker suggests, that inoculum was blown across the Tasman sea; but this is a minimum distance of 1100 miles to South Island, with the coast guarded by mountains, and ca. 1300 miles to North Island. I suspect that both rusts were introduced with planting stock, as in New South Wales. Possibly reports of the rusts in Australia speeded their discovery in New Zealand.

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BARLEY STRIPE MOSAIC IN THE CANADIAN PRAIRIES IN 1972[1]

Arthur W. Chiko

Abstract

In Alberta, Saskatchewan, and southeastern Manitoba, respectively, barley stripe mosaic (BSM) was detected in 42.9%, 11.1%, and 15.3% of the 2-row barley (Hordeum distichum) fields and in 16.7%, 5.1%, and none of the 6-row barley (H. vulgare) fields surveyed in 1972. The incidence of affected plants in these fields varied from a trace to 10%. In Alberta and Saskatchewan, the disease was encountered primarily in southern areas. The percentage of 2-row barley fields in which BSM was detected in southeastern Manitoba in 1972 declined markedly from the previous year. This was probably due to decreased planting of 'Herta' barley, the variety most commonly infected with barley stripe mosaic virus in this province, and increased planting of 'Fergus' barley, a newer 2-row variety which in 1971 appeared to be virus-free. In 1972, however, a trace of BSM was detected in two fields of 'Fergus' and thus even a complete change-over to this variety in Manitoba will probably only temporarily control the disease in 2-row barley.

Breeder seed of 18 barley varieties, including those grown most commonly in Canada, was sown in a field plot and the progenies were examined periodically for BSM symptoms. The disease was detected only in 'Compana' barley, in which 1% of the plants were affected.

Introduction

In 1971, the 2-row barley (Hordeum distichum L. emend. Lam.) crop in Manitoba consisted almost entirely of two varieties. 'Herta', released in 1956, comprised about three-quarters of the crop and 'Fergus', released in 1968, most of the remainder (2). In a survey conducted in Manitoba in 1971 (5), barley stripe mosaic (BSM) was found to occur commonly in growers' fields of 'Herta' but there was no evidence of the disease in fields of 'Fergus'. In recent years, BSM has been detected in only an occasional field of 6-row barley (H. vulgare L. emend. Lam.) in Manitoba (5).

BSM has been reported in growers' fields in Alberta and Saskatchewan for many years but the proportion of fields in which the disease was observed was either unspecified or generally low (4). In 1967, the disease was not detected in Saskatchewan but was relatively common in some fields of 2-row barley near Lethbridge, Alberta (7). Since 1967, there have been no reports of BSM in Saskatchewan or Alberta.

In 1953, McKinney (12) suggested that the presence of barley stripe mosaic virus (BSMV) in barley seed might account for the gradually declining yields exhibited by some varieties in the United States. In mechanical inoculation tests, yield reductions in BSMV-infected barley plants as high as 64% (9) and 90% (12) have been reported. Timian (15) has recently demonstrated that yield losses in 'Kindred' barley were approximately equal to the percentage of infected seeds up to about 30%; higher percentages of infected seeds did not result in addition yield reductions. In North Dakota, where BSM was common in the 1950's and early 1960's, the disease resulted in yield losses in barley as high as 3.5% per year, equivalent to about $3 million (15). In Montana, where BSM was also common for many years, the disease accounted for yield losses to barley of about $3.1 million in 1964 (14).

Yield reductions due to BSM in 'Herta' barley in Manitoba have probably generally been light, because in most fields examined less than 1% of the plants have been affected. Previous experience with the disease in North Dakota and Montana, however, suggested that it might become a serious problem in Manitoba and possibly elsewhere in the Canadian prairies. Theoretically the yield-reducing potential of BSM is greatest when the virus is established in breeder seed of a variety, because all pedigreed seed lots could be contaminated. However, there have been no previous reports on the status of BSMV in breeder seed of barley varieties grown in Canada.

To obtain current information on the distribution and intensity of BSM in the Canadian prairies, in 1972 a survey for the disease was conducted in Manitoba, Saskatchewan, and Alberta. In addition,

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breeder seed of 18 barley varieties grown in Canada was evaluated for the presence of BSMV.

Materials and methods

The survey for BSM in Manitoba, conducted from June 20-29, 1972, was confined to the same southeastern area in the province surveyed the previous year (5). Fields of 2- and 6-row barley in the early tillering to watery ripe stage were examined at intervals of about 5 and 15 miles, respectively, along preselected routes totalling about 1250 miles.

In Saskatchewan and Alberta, the survey for BSM was conducted from July 6-12, 1972, along a preselected route of about 1600 miles. The route passed throught crop districts in which about 85% and 60% of the 1972 barley crop in these respective provinces was grown (3). Fields of either 2- or 6-row barley in the early tillering to milky ripe stage were examined at intervals of about 10 miles.

Breeder seed of 18 barley varieties was evaluated for BSMV infection by sowing samples in a field plot at Winnipeg on May 26, 1972, and examining the progenies for BSM symptoms periodically until July 17, when plants of all varieties had developed heads. Varieties of 6-row barley examined included 'Bonanza', 'Brock', 'Conquest', 'Galt', 'Gateway 63', 'Jubilee', 'Keystone', 'Olli', 'Paragon', 'Parkland', and 'Trent' while 2-row varieties included 'Betzes', 'Centennial', 'Compana', 'Fergus', 'Hannchen', 'Herta', and 'Palliser'. Breeder seed of 'Olli', 'Trent', 'Fergus', and 'Brock' consisted of 89, 100, 132, and 138 separate lines, respectively, whereas breeder seed of the remaining varieties was each maintained in single lots. For breeder seed of each variety consisting of separate lines, 250 seeds of each line were sown, and for each variety maintained as single lots, 1000 seeds were sown. Seeds were sown with a power seeder in 4.3 m rows spaced 0.3 m apart at a rate of about 58 seeds per m.

Leaf samples were collected from plants with suspected symptoms of BSM in both the field plot and growers' fields, assayed for infectivity on 'Black Hulless' barley test plants, and extract from test plants that developed symptoms was tested serologically against BSMV antiserum (5). Samples collected in Manitoba were tested for infectivity the same or following day, whereas those collected in Saskatchewan and Alberta were tested 3-8 days later. A sample was considered to be infected with BSMV only if extract from infected test plants reacted with BSMV antiserum.

Additional tests were conducted to detect BSMV in breeder seed of 'Herta' barley. Seeds were planted in a 4:1 mixture of soil and sphagnum peat moss in both a greenhouse and growth cabinets at about 27° C. One random sample of 1048 seeds were sown in a growth cabinet and samples of 1048 of the thinnest seed (screened to less that 2 mm diam) were sown in both a greenhouse and a growth cabinet. Seedlings in growth cabinets were provided a 15 hr photoperiod of 2800-3600 ft-c with fluorescent and incandescent lights, and those in a greenhouse were provided supplemental fluorescent light from 6 am to 9 pm daily. Seedlings were examined periodically for symptoms until they reached the 2-3 leaf stage, 11-13 days after seeding. At this time, juice was extracted from each seedling exhibiting chlorosis, necrosis or stunting, and was tested serologically against BSMV antiserum (5).

Table 1. Occurrence of barley stripe mosaic in fields of 2- and 6-row barley in the Canadian prairies in 1972

Province	Type of barley	Fields		
		No. examined	No. with BSM*	% with BSM
Alberta	2-row	21	9	42.9
	6-row	24	4	16.7
Saskatchewan	2-row	27	3	11.1
	6-row	39	2	5.1
Manitoba	2-row	118	18	15.3
	6-row	39	0	0.0

* BSMV transmitted to 'Black Hulless' barley and reacted with BSMV antiserum.

Results and discussion

In 1972, BSM was detected more frequently in Alberta than in Saskatchewan or Manitoba (Table 1). In Alberta and Saskatchewan, the disease was more common in fields of 2-row barley than in fields of 6-row barley; in Manitoba it was detected only in fields of 2-row barley. In 2-row barley, BSM affected 5% and 10% of the plants in 2 fields in Alberta and 2-10% of the plants in 8 fields in Manitoba. In other fields where the disease was observed in either 2- or 6-row barley, only a trace of the plants were affected.

Germination of breeder seed of 18 barley varieties evaluated for the presence of seed-borne BSMV in the field varied from 36% to 73% and averaged 58%. BSM was detected only in 'Compana' barley, in which 1% of the plants were affected. In 1972, 'Compana' comprised only about 1% of the total barley acreage in the Canadian prairies and was grown primarily in southern Alberta and southwestern Saskatchewan (3). In southern Alberta, BSM was recognized in growers'

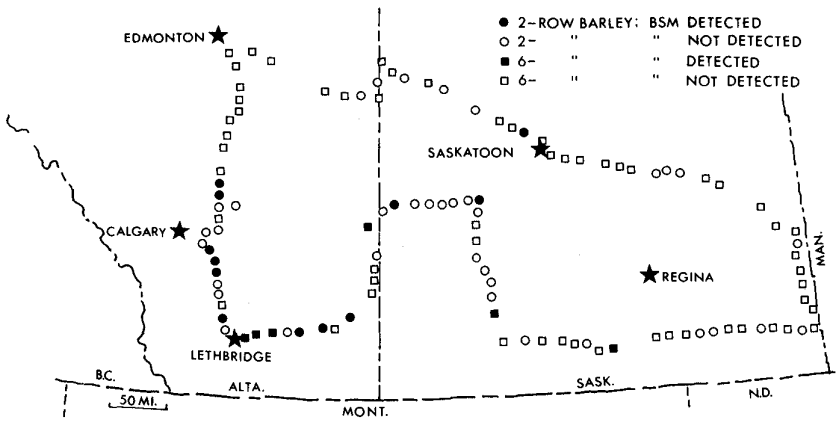

Figure 1. Distribution of barley stripe mosaic in fields of 2- and 6-row barley in Alberta and Saskatchewan in 1972.

fields of 'Compana' as early as 1953 (4). In Montana, where 'Compana' has been widely grown, BSMV was detected in 54.8% of 219 seed lots of this variety in 1954 and 1955; in seed lots containing the virus, an average of 9.2% of the seeds were infected (1).

Germination of a random sample of breeder seed of 'Herta' barley sown in a growth cabinet was 96%. Germination of seeds thinner than 2 mm diam from the same source was 79% in a growth cabinet and 83% in a greenhouse. None of the seedlings developed characteristic BSM symptoms and extract of each seedling that appeared in any way abnormal failed to react with BSMV antiserum.

Although larger samples of breeder seed might have resulted in detection of BSM in barley varieties other than 'Compana', the virus is probably either not present or very rare in breeder seed and rare or absent in other classes of pedigreed seed (i.e. select, foundation, registered, and certified) of varieties grown commonly in Canada. The failure to commonly detect BSM at high levels in growers' fields in Canada also supports this conclusion.

In Alberta and Saskatchewan, BSM was encountered primarily in southern areas and most commonly in southern Alberta (Fig. 1). The exact reason for the apparent localization of the disease is unknown. However, development of BSM symptoms is enhanced by high light intensities and high temperatures (10, 11, 13) and either or both of these factors may be more favorable for symptom expression in southern than in northern areas. In northern areas, symptoms of the disease could conceivably be masked.

Varieties of 2- and 6-row barley in fields where BSM was detected in Saskatchewan and Alberta in 1972 were not identified. Since BSMV was present in breeder seed of 'Compana' barley, the virus probably occurred in most fields of this variety. However, because 'Compana' was grown on a relatively small proportion of the acreage of 2-row barley in these provinces, it is improbable that it was the only 2-row variety affected with BSM in grower's fields.

In southeastern Manitoba, the percentage of 2-row barley fields in which BSM was detected in 1972 declined markedly from 1971 (Fig. 2-A). This was attributed to a reduction in acreage of 'Herta' barley and an increase in the acreage of 'Fergus' barley in 1972 (Fig. 2-B). During the winter of 1972, Manitoba Department of Agriculture extension workers provided information to growers of the potential problem with BSMV in seed of

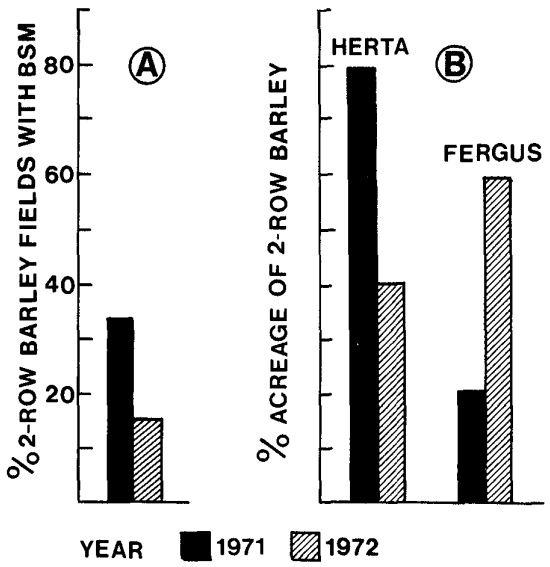

Figure 2. A) Percentage of 2-row barley fields in which barley stripe mosaic was detected in southeastern Manitoba in 1971 and 1972.
B) Percentage of acreage of 2-row barley occupied by the varieties 'Herta' and 'Fergus' in southeastern Manitoba in 1971 and 1972. Acreages were obtained from barley variety surveys (2, 3) in Manitoba crop reporting districts 3, 4, 5, 6, and 12.

'Herta' and of the probability that seed of 'Fergus' was nearly or completely virus-free. This action was probably mainly responsible for the pronounced differences in acreages of the two varieties grown in Manitoba in 1971 and 1972.

The origin of BSMV in growers' fields of 'Herta' barley in Manitoba remains to be determined. If the virus originated in breeder seed of 'Herta', it may be present in such a low percentage of seed that the increase plot would have to be inspected to detect the virus. There may, however, be at least three additional avenues by which the virus could become established in growers' fields. First, the virus might occasionally be transmitted from one barley field to another by pollen (8). Second, wild grasses, some of which have been reported to be susceptible to BSMV (16), might serve as reservoirs of the virus in nature. The virus might be transmitted mechanically from wild grasses to barley, either by natural contact or by machinery during cutting operations along edges of fields. Third, growers might inadvertently contaminate virus-free seed lots with infected ones.

In Manitoba, traces of 6-row barley plants were sometimes noted in fields of 2-row barley where BSM was detected in 1971. The 6-row barley contaminants were sometimes also affected with the disease. It thus seemed possible that BSMV might have been introduced into seed lots of 'Herta' barley by accidental mixing with seed of an infected contaminant. In 1972, all 2-row barley fields examined for BSM in Manitoba, were also inspected for the presence of contaminating crop plants but the data obtained did not support the aforementioned hypothesis. Contaminating 6-row barley and common wheat (Triticum aestivum L.) plants were observed in only 4 fields and 1 field, respectively, of 18 fields in which BSM was detected in 2-row barley. Some wheat contaminants were also infected with BSMV, while infected 6-row barley contaminants were noted in 3 of the fields.

In North Dakota and Montana, BSM has been controlled by different procedures. Barley seed certified to be free of BSMV was initially made available to growers in North Dakota in 1958 and new virus-free varieties with improved agronomic characteristics were released in 1961 and 1964. Growers changed rapidly to the new varieties and, consequently, BSM has not been detected in this state since 1966 (15). In Montana, complete varietal changes have not been made but pedigreed barley seed has been tested for BSMV for many years; no infection has been allowed in foundation and registered seed and a maximum of 5% infection has been allowed in certified seed (6). Since implementing this program, the incidence of the virus in Montana has declined to the point where it was not detected in any certified seed samples tested in 1970 (D. J. Davis, personal correspondence).

Because of the probable absence or low incidence of BSMV in pedigreed seed of barley varieties grown commonly in Canada, an extensive seed certification program to control the virus in this country is presently unwarranted. However, until virus-free seed of 'Compana' barley is made available, the use of this variety should be discouraged. Growers encountering a high incidence of BSM in fields of most other varieties, could probably avert or minimize yield losses simply by planting registered or certified seed. Contacts with Manitoba growers of 'Herta' barley known to be infected with BSMV tend to support this view. Of 15 such growers interviewed in 1971, none reported using pedigreed seed to grow this variety (Chiko, unpublished).

In 1972, two farms in Manitoba, where BSM was detected in 'Herta' barley the previous year, were revisited. In place of 'Herta', both growers had planted 'Fergus' in 1972. One field of 'Fergus' on each farm was thoroughly inspected and several plants affected with BSM were detected in each field. Therefore, even if growers in Manitoba switch completely from 'Herta' to 'Fergus', it seems evident that this action will not completely eradicate the virus from fields of 2-row barley in this province.

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