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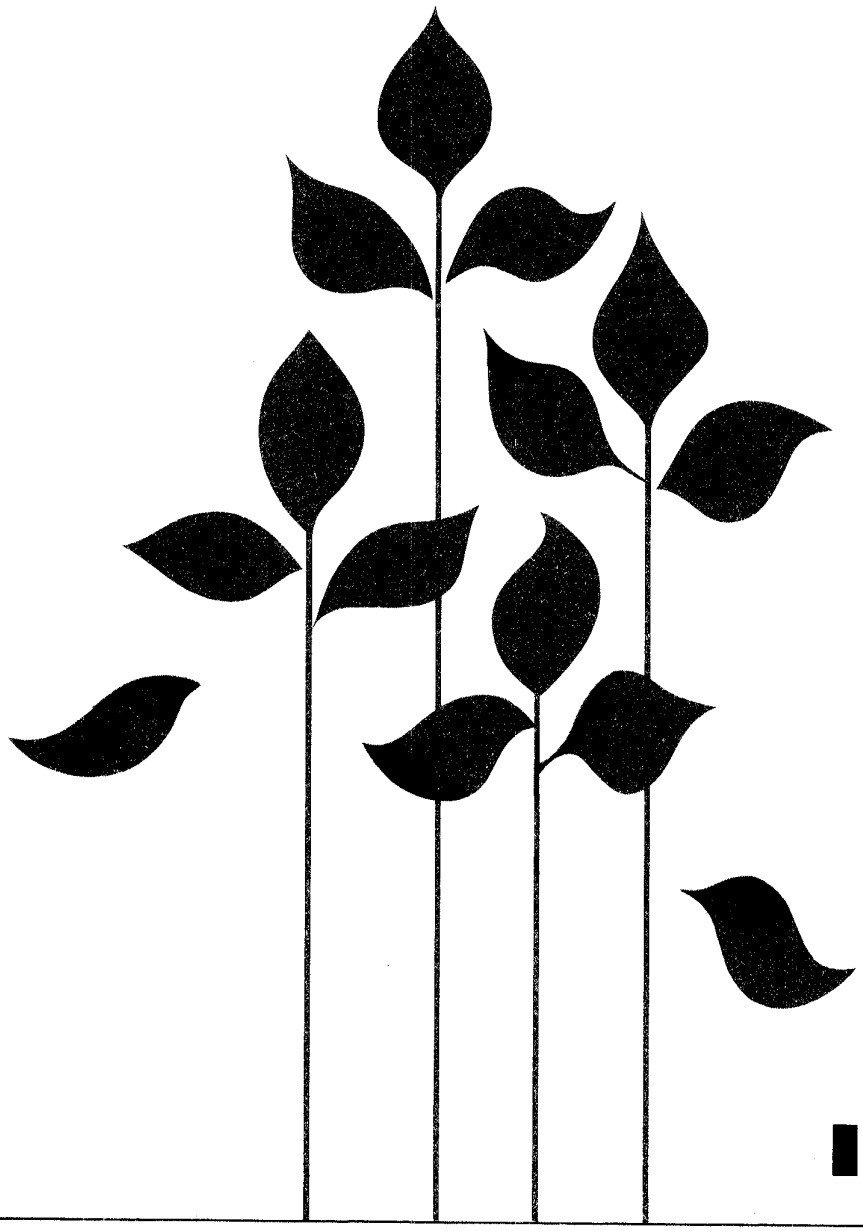

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Contents

The *Canadian Plant Disease Survey* is a periodical of information and record on the occurrence and severity of plant diseases in Canada and on the assessment of losses from disease. Other original information such as the development of methods of investigation and control, including the evaluation of new materials, will also be accepted. Review papers and compilations of practical value to plant pathologists will be included from time to time.

Canadian Plant Disease Survey est un périodique d'information sur la fréquence des maladies des plantes au Canada, leur gravité, et les pertes qu'elles occasionnent. La rédaction accepte d'autres communications originales notamment sur la mise au point de nouvelles méthodes d'enquête et de lutte ainsi que sur l'évaluation des nouveaux produits. De temps à autre, il inclut des revues et des synthèses de rapports d'intérêt immédiat pour les phytopathologistes.

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Snow mold control in turfgrasses with fungicides in Saskatchewan, 1971–74[1]

J. Drew Smith

Field tests were made from 1971 to 1974 to evaluate fungicides for the control of the nonsclerotial low-temperature basidiomycete (LTB), *Fusarium nivale, Sclerotinia borealis*, and a *Typhula* sp. (designated FW), which singly or in complexes cause snow molds of turfgrasses in Saskatchewan. Epidemics of the first three snow molds were started on domestic lawn-type turfs of *Poa pratensis* and of *P. pratensis* with *Festuca rubra* by inoculating them with cultures grown on sterile grain. Tests were also made on naturally infected fine turf of golf and bowling greens composed of *Agrostis* spp. with *Poa annua*. Infection levels varied from generally light in 1971-72, moderate in 1972-73, to very heavy in 1973-74. Quintozene, mercury chlorides, and chloroneb were usually the most effective control materials where the basidiomycetes LTB and *Typhula* FW were dominant or in disease complexes. In some tests, effective control of these pathogens also was achieved with phenyl mercuric acetate, two oxathiin derivatives, and chlorathalonil. A prehibernal attack of *F. nivale* was completely prevented by benomyl, dichlorophene, mercury chlorides, methyl thiophanate, quintozene, and the granular commercial products Bromosan and 3336 at half the usual fall dosage. Quintozene, chloroneb, phenyl mercuric acetate, and mercury chlorides also controlled snow mold where *F. nivale* was the pathogen predominant in a disease complex. Where *S. borealis* was the major cause, quintozene, was the most consistent and effective material but benomyl, methyl thiophanate, chlorothalonil, thiabendazole, phenyl mercuric acetate, and mercury chlorides also controlled the pathogen. Unless resistant *P. pratensis* cultivars are used, it is probable that mercury chlorides will be needed to control severe attacks of LTB. The occurrence of more than one pathogen in snow mold complexes, common in natural infections, makes accurate evaluation of fungicide effectiveness difficult.

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Des essais de terrain ont été effectués de 1971 à 1974 pour évaluer certains fongicides dans la lutte contre le basidiomycète psychrophile asclérotique (LTB), *Fusarium nivale, Sclerotinia borealis* et un *Typhula* sp. (désigné FW), qui, seuls ou en combinaison, causent des moisissures nivéales chez certaines graminées à gazon en Saskatchewan. À des fins d'essai, des foyers des trois premières moisissures ont été déclenchés sur des gazons d'habitation composés de *Poa pratensis* et d'un mélange de *P. pratensis* et de *Festuca rubra*, en les inoculant avec des cultures produites sur grain stérile. Des essais ont également été effectués sur du gazon fin et naturellement infecté de verts de golf et de parterres de boulingrins composés d'un mélange d'*Agrostis* spp. et de *Poa annua*. L'infection a varié de généralement légère en 1971-1972 à très prononcée en 1973-1974, en passant par modéréé en 1972-1973. En général, le quintozène, les chlorures de mercure, et le chloronèbe ont été les matériels de lutte les plus efficaces là où les basidiomycètes LTB et *Typhula* FW dominaient dans les cas d'infections combinées. Dans certains essais, l'acétate de phénylmercure, deux dérivés de l'oxathiine et le chlorathalonil ont pu combattre efficacement ces agents pathogènes. Une infection préhivernale de *F. nivale* a été totalement enrayée par le bénomyl, le dichlorophène, les chlorures de mercure, le thiophanate de méthyle, le quintozène et les produits granulaires commerciaux Bromosan et 3336, à raison de la moitié de la dose habituelle d'automne. Le quintozène, le chloronèbe, l'acétate de phénylmercure et les chlorures de mercure ont également détruit la moisissure nivéale là où *F. nivale* dominait dans les cas d'infections combinées. Là où *S. borealis* constituait le principal organisme pathogène, le quintozène s'est révélé le matériel le plus constamment efficace, mais le bénomyl, le thiophanate de méthyle, le chlorothalonil, le thiabendazole, l'acétate de phénylmercure, et les chlorures de mercure ont également donné de bons résultats. A moins d'utiliser des cultivars résistants de *P. pratensis*, il est probable que certains chlorures de mercure seront nécessaires pour combattre les infections graves de LTB. La présence de plus d'un agent pathogène dans les combinaisons de moisissures nivéales, généralisées dans les infections naturelles, rend difficile l'évaluation précise de l'efficacité des fongicides.

Only since 1970 have detailed studies been made on the causes and control of snow mold of turfgrasses in Saskatchewan. Until then it had been assumed that overwintering disease of turfgrasses was due to a low-temperature basidiomycete, LTB, and winter injury. The most commonly used snow mold fungicides on golf courses and lawns were based on inorganic and organic mercury compounds. However, in 1970-71 tests on domestic lawns where the dominant pathogen was the non-sclerotial low-temperature basidiomycete (LTB) and disease severity was generally light, quintozene, chloroneb, and an oxathiin derivative were as effective as the phenyl mercuric acetate used as a reference material (10); benomyl was not very effective and thiram ineffective (3).

Since 1970 surveys and taxonomic studies have been made on the causes of snow mold in turfgrasses, forage

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grasses, and cereals in parts of Manitoba, Saskatchewan, Alberta, British Columbia, and adjacent Idaho and Washington. These studies (3–6 and unpublished) indicated that the LTB often caused major damage to turfgrasses, even in years with below average or average snowfall (3–7); a sclerotial low-temperature basidiomycete, the SLTB (3, 6) was pathogenic towards turfgrasses; and an undescribed *Typhula* sp. at present designated FW was highly pathogenic on turf and forage grasses and on winter wheat and rye (6, 7). *Fusarium nivale* (Fr.) Ces. and *Sclerotinia borealis* Bub. & Vleug. were found for the first time on Gramineae generally. Descriptions of the symptoms and of these pathogens have been given elsewhere (3–6). A psychrophillic orange-colored, sclerotial fungus designated ORS and tentatively classified as a *Cephalosporium*, occurred on grasses, cereals, and forage legumes in the four western provinces of Canada and in Norway (Smith unpublished); Dr. G. N. Davidson (personal communication) has found it on a wide range of plant species in Alberta. It is antagonistic to several snow mold fungi at low temperature (6) although it is not an active plant pathogen.

Anomalies in the results of the 1970-71 tests of fungicides and the wide diversity of snow molds in different localities and on varying turf types suggested that the effectiveness of the different fungicides depended mainly on which pathogen was dominant. The prevalence of a pathogen might change from season to season according to microclimatic conditions (6). The effect of such changes may be minimized by inducing epidemics of particular pathogens by inoculation. This paper reports the results of tests on turf in Saskatchewan to find fungicides effective against the different pathogens. An attempt was made to find less toxic substitutes for mercurial fungicides.

Materials and methods

The effectiveness of fungicides in the control of different snow molds was examined in tests both on naturally infected golf course and bowling green turf in Saskatchewan and in plot tests at Saskatoon, Saskatchewan, where lawn turf was inoculated with specific pathogens.

Turf inoculation

All tests at the experiment grounds at Saskatoon in the 1971-72 and 1972-73 seasons used turf inoculated with a snow mold. Isolates of the LTB, *S. borealis*, and *F. nivale* obtained from severe outbreaks on turf in the province were grown on moist sterile grain, usually rye, in 1.14 liter (1 quart) milk bottles. Cultures of the LTB and *S. borealis* were incubated at approximately 6°C and *F. nivale* at approximately 15°C, and were harvested after 3 to 4 months growth, spread thinly on paper, dried at room temperature and screened through a 6-mm sieve. They were then refrigerated at −10°to −20°C until required. Evenness in distribution of inoculum on the turf was obtained by hand broadcasting portions of the total amount in several different directions. The inoculum was applied at rates of 1 to 2 kg/100 m^2 in August or September, several weeks before fungicides were used. In the case of test 4 in 1974, inoculum of *S. borealis* from cultures was supplemented with natural sclerotia of the same fungus which had been collected with a rotary carpet sweeper from a severe case of the disease on *Agrostis* turf.

Turf test plots

Turf of *Poa pratensis* L., Common, and *Festuca rubra*, Common, sown in fall 1968, i.e. mature turf, was used for LTB, *F. nivale* and *S. borealis* control studies in 1971 and 1972 (Table 2) and for one test with the LBT in 1973 (Table 4). In plots inoculated with *F. nivale* in 1971, turf of *P. pratensis* cv. Merion of similar age was employed. All turf was mowed in the year after seeding with a 1.2-m-wide underslung rotary mower. Later this was changed to mowing at 4 to 5 cm height with triple-gang reel mowers (each 1.2 m wide) drawn by a garden tractor. All turf was irrigated and maintained in a moderate state of fertility.

Tests on bowling and golf greens were on turf of different botanical compositions but composed mainly of various cultivars of bents, *Agrostis* spp., with varying proportions of annual bluegrass, *Poa annua* L., which is a normal invader of bent turf previously damaged by snow molds. In the 1971-72 (Table 2) and 1972-73 (Table 3) tests, plot areas of 1.52 X 1.52 m (25 ft^2) were treated with fungicide. The size was changed to 1.0 X 1.0 m in the 1973-74 tests. One test with granular materials in 1973-74 (Table 4, site 1) used plots 3.66 X 3.05 m. Treatments were replicated five times with the following exceptions: Test N had three replicates and the North Battleford test two replicates in 1972-73 (Table 3); at the Saskatoon site 1 in 1973-74 each treatment was replicated six times.

Fungicide applications

Fungicide sprays were applied in 107 ml water/m^2 (10 ml/ft^2) with 1-liter capacity pneumatic hand sprayers. Warm water was used in some late fall applications to prevent nozzle icing. Dry powders were applied with a dusting can or by hand after bulking with sand or compost. In both 1971 and 1972 a single application of material was made between mid-October and early November, before the development of a permanent snow cover. In 1973 the first fungicide application was made in September and the second in October to turf in all tests except the one at Lloydminster which received only the last. The first application was at half dosage. The common and product names, percent active ingredients, formulations, and sources of fungicides used are given in Table 1. Both systemic and nonsystemic fungicides were included.

Polyethylene covers

It is a practice on some golf courses in Saskatchewan to cover greens before winter with polyethylene sheeting to reduce desiccation during snow-free periods in spring and to promote a "greenhouse effect" which encour-

Table 1. Fungicides used in snow mold tests

Index no.	Product name	Active ingredient* % and formulation†	Source
1	Benlate	benomyl 50%, WP	Du Pont
2	Cad-Trete	thiram 75% + $CdCl_2.H_2O$ 8.3%, Gran	Cleary
3	CD/Urea	$CdCl_2.H_2O$ 10% + urea, Soln	Smith
4	Tersan SP	chloroneb 65%, WP	Du Pont
5	Demosan	chloroneb 7.5%, Gran	Du Pont
6	Chlorophenate	chlorophenate mixture 18%, Soln	Cleary
7	Daconil	chlorothalonil 75%, WP	Diamond-Shamrock
8	DCP	dichlorophene 50%, WP	Smith
9	BAS 3460F	2(methoxy carbonil)-benzimidazole 50%, WP	BASF
10	Calo-clor	Hg 36.5%, WP	Malinckrodt
11	Mersil	Hg 42%, WP	May & Baker
12	CCS	Hg 100%, P	Smith
13	BAS 3050	methyl benzoic acid anilide 75%, WP	BASF
14	Topsin M	methyl thiophanate 70%, WP	Nippon Soda
15	4222	polychlorphenate + quintozene** Gran	Cleary
16	PMA	phenyl mercuric acetate 10%, Soln	Smith
17	PMA-10	phenyl mercuric acetate 10%, Soln	Later
18	Terraclor	quintozene (PCNB) 75%, WP	Olin
19	4221	sodium polychlorphenate 4%, Gran	Cleary
20	Salan	salicylanilide 50%, WP	Smith
21	Mertect	thiobendazole 40%, Slurry	Merck
22	Tersan 75	thiram 75%, WP	Du Pont
23	4220	thiram + dyrene** Gran	Cleary
24	Arrest	thiram 50% + carbathiin 20% + oxycarbathiin 5%, WP	UniRoyal
25	Vitavax	carbathiin 75%, WP	UniRoyal
26	D-735	carbathiin 75%, WP	UniRoyal
27	Terrazole	5-ethoxy-3-trichloro methyl, 1,2,4 thiadiazole 95%, P	Olin
28	TCMTB	2 (thiocyanomethyl thio benzothiazole 40%, Slurry	Buckman
29	BAS 3201F	** 50%, WP	BASF
30	Bromosan	diethyl-4,4-0-phenylene bis (3-thioallophanate) 16.67% + thiram**, Gran	Cleary
31	Bromosan	diethyl-4,4-0-phenylene bis (3-thioallophanate) 16.67% + thiram 50%, WP	Cleary
32	CA 70203	** 20%, Soln	Cela
33	3336	diethyl-4,4'-0-phenylene bis (3-thioallophanate) 50%, Gran	Cleary
34	3336	diethyl-4,4'-0-phenylene bis (3-thioallophanate) 50%, WP	Cleary

* Where the common name of the active ingredient is not known or is inconveniently long the product name is used in the tables and text.

† WP = wettable powder, P = powder, Gran = granular, Soln = solution.

** Ingredient or % not available.

ages a more rapid recovery in spring. Two treatments in test 2 in 1971-1972 (Table 2) employed clear 4 mm polyethylene covers, either intact or perforated every 10 cm with 6 mm holes, to determine whether the amount of damage due to *F. nivale* snow mold was affected by this practice.

Rating of disease

An estimate was made of percentage of turf affected by disease in each plot. Usually this was done more than once, and occasionally three times. Only where there were considerable differences between ratings on different dates, resulting in different ranking of the fungicides, have these data been given in the tables.

Results and discussion

In evaluating the effectiveness of the fungicides in controlling turfgrass snow molds, consideration must be given to level of infection and to the identity of the pathogens causing disease, as indicated by those which predominated on dead or damaged grass at snow melt.

In late April 1972 infections resulting from inoculation with *F. nivale*, LTB, and *S. borealis* in tests 1 to 4 (Table 2) were only moderately severe. Inoculation was not effective in the establishment of a predominantly *F. nivale* infection in test 2 since the LTB also developed. The low level of *S. borealis* infection in test 5, as compared with its high incidence in test 6, may explain

Table 2. Snow mold fungicide tests on amenity turf, Saskatchewan, 1971-72

Fungicide or cultural practice	Index no. (Table 1)	Dosage (a.i.g/m^2)	Percent area of turf affected, April 1972* Research Station, Saskatoon Test 1	Test 2	Test 3	Test 4	Swift Current Test 5	Test 6
Benomyl	1	0.33		14	37ij	9ab	6	
Benomyl	1	0.65			34hi			
Cadmium chloride + urea	3	0.08			38j			
Cadmium chloride + urea	3	0.17			28g			
Chloroneb	4	1.40	6a		3ab		10	
Chloroneb	4	2.80			7bc			
Mercurous/mercuric chlorides	12	0.78	3a	0	8cd	9ab	22	
Mercurous/mercuric chlorides	12	1.56			2a			
Methylbenzoic acid anilide	13	0.08		8		25d		
Methyl thiophanate	14	0.08		16		7ab		
Methyl thiophanate	14	0.16			27g			
Phenyl mercuric acetate	16	0.07	4a	4	18f	6a	23	19c
Phenyl mercuric acetate	16	0.13			5abc			
Quintozene	18	0.98						11b
Quintozene	18	1.95	7a		7bc	8ab	7	8ab
Quintozene	18	3.90			2a	9ab		4a
Salicylanilide	20	0.87			18f			
Salicylanilide	20	1.74			32h			
Thiram	22	1.63		6	13e	17c		
Thiram	22	3.26			20f			
D-735	26	0.49		3	11de	15c		
D-735	26	0.98		3	5abc			
BAS 3201F	29	0.05		13		6ab		
CA 70203	32	0.02		13		14c		
Polyethylene unperforated				12				
Polyethylene perforated				15				
Check - untreated			20b	15	32h	29e	20	70d
Predominant pathogens** at snow melt			F.n.†	F.n.† & LTB	LTB	S.b.†	S.b.	S.b.

* Damage assessed 28 April 1972 at Saskatoon Research Station and 13 April 1972 at Swift Current Golf Club. Within tests, figures subtended by the same letters do not differ significantly at the 1% level as determined by Duncan's multiple range test.

** F.n. = *Fusarium nivale*, LTB = low temperature basidiomycete, S.b. = *Sclerotinia borealis*.

† Artificial inoculation.

the lack of significant differences between the treatments in test 5. This disease was successfully established by inoculation with natural sclerotia and those from grain cultures in test 4, the only occasion in several attempts (unpublished) when this was successful. Ascospores may be necessary for heavy infection of turf by this fungus, at least this seems to be the case in the infection of grass plants in infection chamber studies (K. Årsvoll, personal communication). In the 1971-72 season with comparatively light infections, one application of quintozene, chloroneb, phenyl mercuric acetate, or a mercurous/mercuric chloride mixture effectively controlled *F. nivale* and the LTB. D-735 and BAS 3201F also gave a fair control of the LTB. Phenyl mercuric acetate, methyl thiophanate, quintozene, benomyl, and inorganic mercury chlorides reduced severity in a moderate attack of *S. borealis* induced by inoculation (test 4). Quintozene was more effective than phenyl mercuric acetate in controlling a heavy natural infection of *S. borealis* on *Agrostis* spp. turf on a golf green in southern Saskatchewan. The polyethylene sheets covering plots appeared to have had little effect on the severity of disease. In practice, covers do not usually increase the severity of fusarium patch disease where they are used on golf greens composed on *Agrostis* spp. and *Poa annua*, compared with uncovered.

In April 1973, levels of infection were higher than in 1972 on both artificially inoculated turf on the Saskatoon test area (Figs. 1 and 2) and on golf course greens (Table 3). On sites A and B, where the turf was of the Merion cultivar of *P. pratensis*, a very high infection resulted from inoculation with the LTB. Variation in disease rating from plot to plot of treatments was more apparent in golf course tests than on the Saskatoon test area (vide footnote 1, Table 3). On the Saskatoon and Prince Albert golf courses, a low level of infection by *F. nivale* was noted before fungicide application in 1972.

In the 1972-73 tests a very heavy outbreak of the LTB on the very susceptible *P. pratensis* cultivar Merion (8) (Table 3, sites A and B) was not controlled by any fungicide other than inorganic mercury chlorides. These

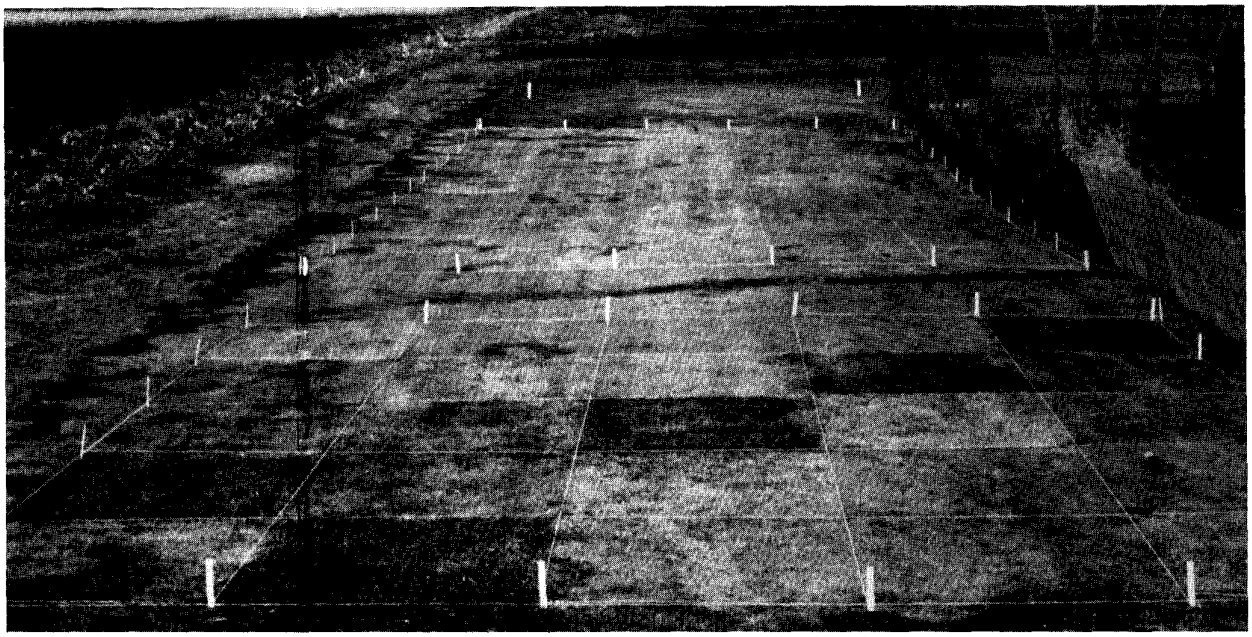

Figure 1. LTB test, Site A 1972-73. Only inorganic mercurials controlled heavy infection in April 1973.

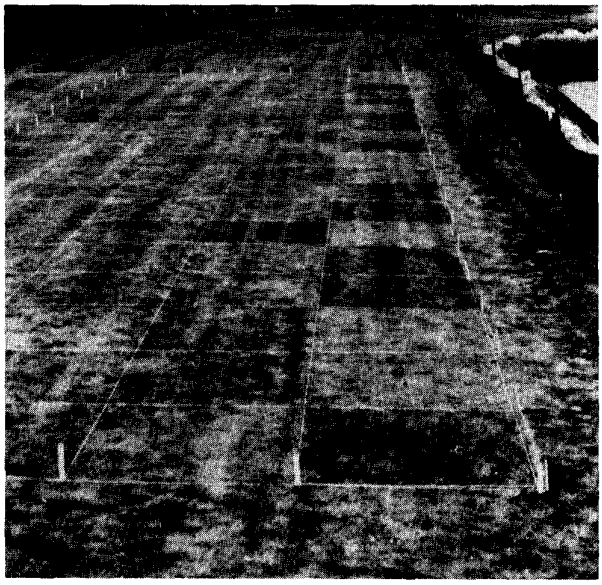

Figure 2. LTB test, Site D 1972-73. Inoculated plots showing effective control by some fungicides in April 1973.

materials were also the most effective in the test on site N with the less susceptible Park cultivar (8) in the same year. Where levels of infection were lower on turf containing the more resistant common Kentucky *P. pratensis* (8) (Table 3, sites D and E) than on the Merion turf several materials were effective against the LTB when applied only once in the fall. The most efficient were quintozene, chloroneb, and chlorothalonil although both thiabendazole and an oxathiin derivative (D-735) also considerably reduced disease severity. Inorganic mercuries, which have a fairly broad spectrum of effectiveness as fungicides, and to a lesser extent organic mercury compounds (1, 10) have been extensively used to control snow molds of turfgrasses in many

Table 3. Snow mold fungicide tests on amenity turf, Saskatchewan, 1972-73

			Percent area of turf affected on dates indicated in 1973*									
			Research Station, Saskatoon					North Battleford	Saskatoon		Prince Albert	Swift Current
	Index no.	Dosage	Site D	Site B	Site A	Site E	Site N	G. & C. C.	G. & C. C.		G. & C. C.	G. C.
Fungicide	(Table 1)	(a.i.g/m^2)	13/4	18/4	18/4	13/4	18/4	17/4	20/3	28/3	10/4	3/4
Benomyl	1	0.33	55de				28					
Benomyl	1	0.66	36bcd	85		26ab	13	14	24ab	25abc	25abc	14ab
Benomyl	1	1.32	41cd	79			40					
Chloroneb	4	1.69	17ab	89	80b			9	3a	2a	10a	14ab
Chloroneb	4	3.38	8a	88								
Chlorothalonil	7	1.95	15a	87		8a		3	14ab	20abc	66def	14ab
Chlorothalonil	7	3.90	4a									
MBC	9	0.33	54de	98								
MBC	9	0.66	78f			34b		6	36b	41bc	38abcd	26b
Mercurous/mercuric chlorides	10	0.78			7a		7					
Methyl thiophanate	14	0.46	49de	91								
Methyl thiophanate	14	0.92	63ef			28ab		9	26ab	30abc	52bcde	12ab
Phenyl mercuric acetate	16	0.07	48de	88	88b		47					
Phenyl mercuric acetate	16	0.13	57de			23ab	40	23	1a	1a	4a	
Quintozene (PCNB)	18	0.98	14a									
Quintozene (PCNB)	18	1.95	6a	87	78b			1	2a	0a	1a	4a
Thiabendazole	21	0.52					28	3			20ab	14ab
Thiabendazole	21	1.04	23abc			18ab	43		20ab	16ab		
Thiabendazole	21	1.56					23					
D735	26	0.98	23abc									
TCMTB	28	0.52		83		36b		6	26ab	31abc	84ef	17ab
CA 70203	32	0.13	82f									
CA 70203	32	0.26	57de	90		27ab		1	18ab	21abc	61cdef	18ab
Check - untreated			54de	85	79b	36b	37	21	38b	54c	92f	46c
Predominant pathogens** at snow melt			LTB†	LTB†	LTB†	LTB†	F.n. & LTB†	S.b.	F.n. & LTB	F.n. & LTB	F.n.,T.sp. & S.b.	S.b., LTB,F.n.

* Within tests, figures subtended by the same letters do not significantly differ as determined by Duncan's multiple range test; at the 5% level in the Saskatoon and Swift Current G. C. tests and at the 1% level in the others.

† Artificial inoculation with LTB only.

** LTB = low temperature basidiomycete, F.n. = *Fusarium nivale*, S.b. = *Sclerotinia borealis*, T.sp. = *Typhula sp.*

parts of the world for several decades (9). While they are ecologically undesirable, present substitutes are not entirely satisfactory, particularly where a severe attack by the LTB on susceptible cultivars such as Merion is concerned. Several *P. pratensis* cultivars in use are more susceptible to the LTB than is Merion (8). It seems probable that from the data obtained in tests A, B, D, E, and N the effectiveness of materials other than the mercuric chlorides would be adequate if more resistant turfgrass cultivars were used (8).

One of the factors complicating the evaluation of the effectiveness of fungicides in the 1973-74 tests was the much above-average depth and longer duration of the snow cover in that season. Winter snowfall at Saskatoon for 1973-74 was 1,704 mm and a continuous snow cover persisted for 170 days from 30 October to 17 April. This was much above the average snowfall for the previous 33 winters of 1,087 mm and 143 days snow cover (personal communication, Dr. J. Maybank, Physics Department, Saskatchewan Research Council, 19 August 1974). The level of infection with pathogens was high at snow melt in April 1974 (Table 4). In fall 1973, infections of *F. nivale* developed on some test areas after the first application of fungicide. In the Emma Lake test (Table 4), a sufficiently high incidence of disease developed for a reliable estimation of the protective effect of fungicides against *F. nivale* to be obtained before a persistent winter snow cover formed in late October.

In the 1973-74 tests quintozene, mercurous/murcuric chloride and chloroneb were overall the most effective materials in a year of prolonged, deep snow cover when the basidiomycetes *Typhula* (FW) and the LTB were prominent. However, in practice a greater reduction in infection than that achieved would be desirable. Control of a moderately severe, prehibernal attack of fusarium patch disease was obtained in the Emma Lake test (Table 4) by half dosage of many of the materials, including two granular products. The control of this

Table 4. Snow mold fungicide tests on amenity turf, Saskatchewan, 1973-74

Fungicide	Index no. (Table 1)	Dosage (g a.i./m^2)	Percent area of turf affected on dates indicated*: Emma Lake G. C. 11/10/73†	Emma Lake G. C. 9/5/74	Lloydminster G. C. 17/4/74	Prince Albert G. C. 8/5/74	Prince Albert National Park Turf nursery 26/4/74	Prince Albert National Park Bowling green 26/4/74	Saskatoon Research Station Site 1 20/4/74	Saskatoon Research Station Site 2 29/4/74
Benomyl	1	1.0	0a	96b	81b	87cde	95d	91e	76d	65bc
Cadmium complex	2	25.00**							76d	65bc
Chloroneb	4	1.95	7ab	52a	69b	66bc	48ab	31a		
Chloroneb	5	25.00**								42b
Chlorophenate	6	1.08				97e	58bc	65bcd		
Chlorothalonil	7	2.02	12b	93b	74b	74bcd	74bc	79bcde		
Dichlorophene	8	1.00	0a	91b	82b	96de	84cd	78bcde		
Dichlorophene	8	2.00			82b					
Mercurous/mercuric chlorides	1	1.08	0a	38a	17a	77bcde				
Methyl thiophanate	14	0.98	0a	95b	74b	92de	83cd	86cde		
Polychlorphenate + quintozene	15	25.00**							76d	65bc
Phenylmercuric acetate	17	0.05	5ab	85b		32a				
Quintozene (PCNB)	18	2.02	0a	52a	37ab	62b	26a	17a	30abc	13a
Sodium polychlorphenate	19	25.00**							58abcd	71c
Thiabendazole	21	1.00			81b	86cde	95d	91e		
Thiram + dyrene	23	25.00**							66bcd	67bc
Arrest	24	2.15			3a		79cd	55b		
Vitavax	25	1.50			19a		83cd	62bc		
Terrazole	27	0.98	12b	84b		94de	79cd	94e		
Bromosan	30	25.00**							26a	68bc
Bromosan	30	15.00**	0a	95b	81b	96de	86cd	78bcde		
Bromosan	31	1.02			67b	88de	79cd	78bcde		
3336	33	25.00**							19a	61bc
3336	34	1.0	0a	95b	82b	93de	84cd	89de		
Check - untreated			27c	93b	73b	98e	92d	91e	70cd	77c
Predominant pathogens § at snow melt			F.n.	T. FW	T. FW	S.b. & T. FW	T. FW	T. FW & S.b.	LTB & F.n.	LTB††

* Within tests, figures subtended by the same letter do not differ significantly at the 1% level using Duncan's multiple range test.

† Rated before snow cover between first and second applications of fungicide.

** g formulation/m^2

†† Artificial inoculation.

§ F.n. = *F. nivale*, T. FW = *Typhula* FW, S.b. = *S. borealis*, LTB = low temperature basidiomycete.

disease, common in wet, cold fall weather in western Canada (4, 5), and economy in fungicide usage were being attempted by half dosage. However, full dosage of fungicides in September as well as in the later application might have improved control of the *Typhula* snow mold in spring under particularly arduous conditions. In the Lloydminster test, the effectiveness of oxathiins plus thiram (2) and to a lesser extent a mercury chloride fungicide at half dosage was remarkable (Table 4). The only other material showing some control effectiveness here was quintozene. The mode of action of systemic fungicides such as the oxathiins (11) and the inorganic mercury chlorides is quite different (1); the action of the latter being mainly due to mercury in the vapor phase. Nevertheless, excellent control was achieved by both materials. Granular materials, with the exception of the commercial products 3336 and Bromosan, were not as effective as a quintozene spray against a complex of the LTB and *F. nivale*. A granular chloroneb fungicide showed some activity against the LTB (Table 4). The main advantage of granular materials appears to be in convenience of application in top dressings or by simple applicators and not in their efficiency of disease control.

Where *S. borealis* was the principal cause of snow mold in tests 4 and 5 in 1971-72 (Table 2) and in the North Battleford test in 1972-73 (Table 3), materials other than quintozene, viz benomyl, chloroneb, phenyl mercuric acetate, methyl thiophanate, chlorothalonil, thiabendazole, mercurous/mercuric chlorides, TCMTB, and CA 70203 gave some control. However, when *S. borealis* was associated with other pathogens in tests on golf greens at Prince Albert and Swift Current in 1972-73 (Table 3) and at the Prince Albert National Park Golf Club and bowling green in 1973-74 (Table 4), quintozene was generally the most effective. The systemic fungicide benomyl, which showed varying degrees of control where *F. nivale* was present, was ineffective

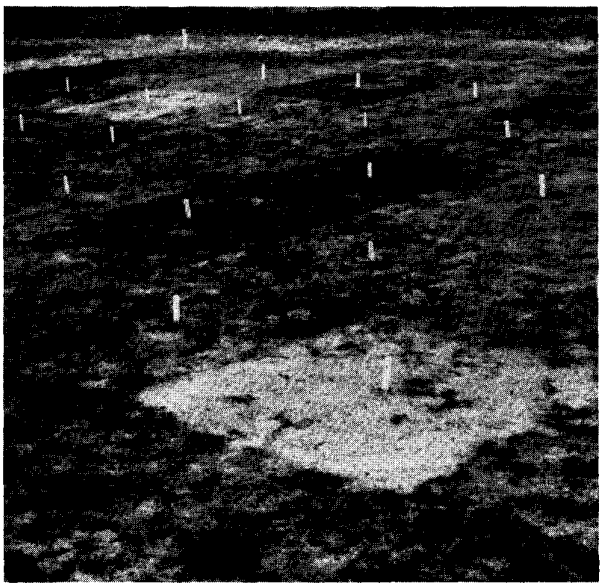

Figure 3. Turf nursery test, Prince Albert National Park, 1973-74. *Typhula* FW mycelium developed on thiabendazole plots in foreground and left rear.

against the LTB in all the 1971-72 tests and also in all tests in 1973-74 where snow cover was prolonged and infection level high. The systemic fungicide thiabendazole failed to reduce severe damage caused mainly by *Typhula* FW on the turf nursery test in 1973-74 and appeared to stimulate mycelial development of this fungus (Fig. 4). This effect was not seen with other materials. Chlorothalonil significantly reduced severity of infection in three and considerably reduced the areas of turf damaged in four of the eight 1972-73 tests (Table 3) but was effective in only one of the six tests in 1973-74 (Table 4). When infection levels were much higher than in the previous year, it appeared to have a wide spectrum of activity. Chloroneb gave effective control over a wide range of pathogens although it was not fully tested against *S. borealis*.

While some progress was made in the three seasons in evaluating the effectivness of fungicides against snow molds in tests on golf greens under playing conditions, the main difficulty is still related to the occurrence of complexes of pathogens. This problem was suggested by earlier work (4, 6). The balance of these pathogens shifts from year to year under the influence of climatic factors. It seems possible, by establishing test areas under uniform management practices, to develop test sites with specified pathogens dominant (7, 8). So far the LTB has been most amenable to this treatment but further attempts with *S. borealis* and *Typhula* FW appear warranted.

Acknowledgments

I am indebted to the management and staff of Elmwood Golf Club, Swift Current; Lloydminster Golf and Country Club; Saskatoon Golf and Country Club; Cooke Golf Club; Prince Albert; Emma Lake Golf Club; Prince Albert National Park for turf test facilities; to Drs. R. D. Tinline and W. L. Seaman for suggestions on presentation of data and to Mr. W. W. Reiter for valuable technical assistance.

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Air-borne rust inoculum over western Canada in 1975[1]

G. J. Green

There was less air-borne inoculum of *Puccinia graminis* and *P. recondita* over western Canada in 1975 than usual. Rust was widespread in the south but the late arrival of a small quantity of primary inoculum and unfavorable weather restricted rust development early in the season and reduced the number of urediospores found in spore traps.

Can. Plant Dis. Surv. 56:9-11. 1976

En 1975, il y avait moins de spores de *Puccinia graminis* et de *P. recondita* que d'habitude en suspension dans l'air au-dessus de l'ouest du Canada. La rouille était répandue dans le sud, mais l'arrivée tardive d'une faible quantité d'inoculum primaire et le mauvais temps ont restreint le développement de la rouille au début de la saison et réduit le nombre d'urédiospores dans les pièges à spores.

An estimate of the amount of air-borne rust inoculum over western Canada in 1975 was made using the same methods described in earlier reports of this study published annually in the *Canadian Plant Disease Survey*.

Urediospores of *Puccinis graminis* and *P. recondita* appeared on the slides later than usual (Table 1) despite widespread rust infections to the south. Leaf rust was first observed in the field on June 27 at Winnipeg about 10 days later than normal, suggesting that a spore

Table 1. Number of urediospores of stem rust and leaf rust per square inch (6.5 cm^2) observed on vaseline-coated slides exposed for 48-hour periods at three locations in Manitoba and three locations in Saskatchewan in 1975

Date	Winnipeg		Morden		Brandon		Indian Head		Regina		Saskatoon	
	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust
May 27-28	0	0	0	0	0	0	0	0	0	0	0	0
29-30	0	0	0	0	0	0	0	0	0	0	0	0
31- 1	0	0	0	0	0	0	0	0	0	0	0	0
May total	0	0	0	0	0	0	0	0	0	0	0	0
June 2- 3	0	0	0	0	0	0	0	0	0	0	0	0
4- 5	0	0	0	0	0	0	0	0	0	0	0	0
6- 7	0	0	0	0	0	0	0	1	0	0	0	0
8- 9	0	0	0	0	0	0	0	0	0	0	0	0
10-11	0	0	0	0	0	0	0	0	0	0	0	0
12-13	0	0	0	0	0	0	0	0	0	0	0	0
14-15	0	0	0	0	0	0	0	0	0	0	0	7
16-17	0	0	0	0	0	0	0	1	0	0	0	0
18-19	8	0	1	2	1	3	0	0	0	1	0	0
20-21	0	1	0	0	1	0	0	0	0	0	0	0
22-23	0	2	0	0	0	0	0	1	1	0	0	0
24-25	0	9	39	49	0	11	0	39	0	2	0	6
26-27	0	4	5	35	0	11	0	0	1	2	0	0
28-29	0	0	1	5	0	2	0	0	0	0	0	11
30- 1	0	0	1	1	0	0	0	2	4	2	0	13
June total	8	16	47	92	2	27	0	44	6	7	0	37

[1] *Contribution No. 714, Research Station, Agriculture Canada, 25 Dafoe Road, Winnipeg, Manitoba R3T 2M9*

Table 1. Cont.

Date	Winnipeg		Morden		Brandon		Indian Head		Regina		Saskatoon	
	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust
July 2- 3	0	1	0	1	0	1	0	1	0	2	0	0
4- 5	0	0	1	1	0	2	0	0	0	7	0	12
6- 7	0	6	-	-	0	2	0	11	0	15	0	19
8- 9	0	0	1	6	0	1	0	2	0	2	0	10
10-11	0	1	0	2	0	2	0	0	0	4	0	18
12-13	0	1	0	4	0	4	0	3	0	1	0	9
14-15	1	4	1	45	0	0	0	24	0	11	0	28
16-17	0	15	1	33	0	1	1	4	1	20	0	1
18-19	0	2	1	10	0	2	1	16	0	11	0	14
20-21	0	6	0	11	0	0	0	1	0	38	0	2
22-23	0	1	4	14	0	8	0	5	0	47	0	20
24-25	0	32	2	98	0	28	0	16	0	19	0	7
26-27	1	35	4	208	0	35	0	8	0	46	0	26
28-29	40	322	20	473	0	130	0	75	1	298	2	15
30-31	14	38	0	9	0	0	0	0	0	5	11	103
July total	56	464	35	915	0	216	2	166	2	526	13	284
Aug. 1- 2	0	56	1	27	4	71	0	5	0	4	12	120
3- 4	7	38	38	43	0	20	0	20	0	8	20	65
5- 6	87	544	52	558	6	54	2	66	7	307	35	217
7- 8	2	8	0	14	0	2	0	0	0	19	3	78
9-10	40	102	13	96	2	33	0	21	0	79	52	151
11-12	69	41	15	30	22	68	0	0	0	0	42	238
13-14	50	2	39	34	0	5	0	13	0	12	33	249
Aug. total	255	791	158	802	34	253	2	125	7	429	197	1118
1975 total	319	1271	240	1809	36	496	4	335	15	962	210	1439
1974 total	17	151	15	290	12	185	12	223	36	500	21	861

Table 2. Average number of urediospores of stem rust and leaf rust observed on slides exposed for 48-hour periods at six locations from July 1 to August 15 for the years 1965 to 1975

Year	Winnipeg		Morden		Brandon		Indian Head		Regina		Saskatoon		Average	
	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust
1975	14	57	9	82	2	21	1	13	1	43	10	64	6	47
1974*	1	4	1	7	1	5	1	5	1	12	1	47	1	13
1973	16	249	4	136	1	242	2	629	2	1449	7	179	5	481
1972	16	277	24	696	16	645	12	1515	23	6566	3	528	16	1705
1971	38	497	14	404	4	114	5	125	5	172	7	87	12	233
1970	56	252	73	649	12	235	8	173	12	480	2	197	27	331
1969	5	41	5	62	1	29	1	8	1	6	2	24	3	28
1968	3	225	5	219	1	47	1	24	1	23	1	15	2	92
1967	9	81	6	122	1	16	1	8	2	11	0	12	3	42
1966	23	145	17	239	4	11	13	702	6	618	3	1296	11	502

Table 2. Cont.

Year	Winnipeg		Morden		Brandon		Indian Head		Regina		Saskatoon		Average	
	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust
1965	20	331	74	951	43	659	364	2810	892	7526	92	3690	248	2661
1965-74 average	19	210	22	348	8	200	41	600	94	1686	12	608		

* July 1 to August 5

shower had occurred about June 15 but was not detected by the spore traps in Manitoba. The small amount of primary inoculum, its late arrival, and unfavorable weather for rust development in July seems to have reduced the amount of rust in western Canada early in the season and the numbers of spores on the slides (Table 2).

The averages presented in Table 2 are usually indicative of the amount of rust in western Canada each year but they can be misleading. The averages indicate that there was little stem rust development in 1974 and 1975 but in these years stem rust was common to the south and in western Canada by the end of August on susceptible *Hordeum jubatum* L. and on susceptible varieties in experimental plots. During the 11 year period 1965 to 1975 stem rust resistant varieties were grown in the main rust area of Manitoba and southeastern Saskatchewan, but prior to 1968 Thatcher, which is susceptible to race 15B of stem rust, was widely grown farther west in Saskatchewan. The planting of Thatcher could account for the relatively large numbers of stem rust spores caught at Saskatchewan locations in 1965. Thatcher is very susceptible to leaf rust as well but it was replaced after 1967 by Manitou and later by Neepawa, which were resistant to leaf rust. The planting of these varieties could account for the small numbers of leaf rust spores present in the years 1967 to 1969. In 1970, races with increased virulence on Manitou and Neepawa appeared and caused moderately severe infections which resulted in increased numbers of spores from 1970 to 1973. In 1974 and 1975 weather conditions were unfavorable for leaf rust development.

Leaf rust of wheat in Canada in 1975[1]

D.J. Samborski

Conditions were favorable for a leaf rust [*Puccinia recondita*] epidemic on the Prairies in the spring of 1975 since most of the acreage was planted to moderately susceptible varieties and leaf rust was widespread in the United States. However, very hot, dry weather during July delayed rust development and there was little damage from leaf rust in Canada in 1975. The leaf rust race survey showed that virulence on the *Lr2* locus for resistance was prevalent in the rust population. Three cultures virulent on Transfer (gene *Lr9*) were isolated from collections made in Manitoba and Saskatchewan.

Can. Plant Dis. Surv. 56:12-14. 1976

Au printemps 1975, les conditions étaient favorables à une épidémie de la rouille des feuilles [*Puccinia recondita*] dans les Prairies, puisque la plupart des superficies étaient ensemencées de variétés modérément sensibles et que la rouille était largement répandue aux Etats-Unis. Toutefois, le temps sec et très chaud qui a sévi au mois de juillet a retardé le développement de la rouille et a permis de réduire les dégâts. L'étude des races de la rouille des feuilles a montré que la population de rouille a été virulente sur les variétés possédant le gène de résistance (*Lr2*). Trois cultures virulentes sur Transfer (gène *Lr9*) ont été isolées de prélèvements effectués au Manitoba et en Saskatchewan.

Disease development and crop losses in western Canada

Most of the wheat acreage in the traditional rust area of Manitoba and Saskatchewan was planted to moderately susceptible varieties in 1975. Leaf rust [*Puccinia recondita*] was widespread in the United States in the spring and there was abundant inoculum to initiate an epidemic in western Canada. However, very hot, dry weather during July delayed rust development and there was little damage from leaf rust in 1975.

Leaf rust in the rust nurseries

Ratings of leaf rust intensity on 20 wheat varieties grown at nurseries across Canada are shown in Table 1. A number of varieties were essentially free of leaf rust but virulence has been found on all these bread wheat varieties but Agatha, which has gene *Lr19*.

Table 1. Percentage infection by *Puccinia recondita* on 20 wheat varieties in uniform rust nurseries at 12 locations in Canada in 1975

Location	Red Bobs	Lee	Pitic 62	Neepawa	Napayo	Sinton	Kenya Farmer	C.I.8154 x Frocor[2]	Glenlea	Norquay	Exchange	Frontana	Thatcher[6] x Transfer	R.L.4255	Agatha	Hercules	Mindum	Wascana	Macoun	Wakooma
Creston, B.C.	70	10	10	5	5	0	5	3	0	0	0	0	0	0	0	5	0	5	5	5
Lethbridge, Alta.	5	3	1	1	1	0	3	1	0	0	0	0	0	0	0	tr*	0	tr	tr	tr
Melfort, Sask.	70	50	20	40	40	tr	30	20	0	0	0	0	0	0	0	3	0	3	3	5
Indian Head, Sask.	5	1	tr	3	3	tr	tr	tr	0	0	0	0	0	0	0	tr	0	tr	tr	tr
Brandon, Man.	90	70	50	20	20	10	40	5	tr	tr	tr	5	tr	0	0	5	tr	5	5	5
Morden, Man.	10	3	tr	3	5	tr	tr	1	0	0	0	0	0	0	0	tr	0	tr	tr	tr
Glenlea, Man.	5	5	tr	3	5	tr,3	3	tr	0	tr	tr	tr	0	0	0	1	0	1	1	1
Thunder Bay, Ont.	30	5	5	5	5	0	5	3	0	0	0	0	0	0	0	3	0	3	3	3
Vineland, Ont.	20	1	tr	tr	tr	0	1	3	0	0	0	0	0	0	0	3	0	3	3	3
Sunbury, Ont.	10	1	1	tr	tr	0	tr	2	0	0	0	0	0	0	0	tr	0	tr	tr	tr
Québec, Qué.	80	5	40	3	5	tr	5	3	0	0	tr	0	0	0	0	20	0	20	20	20
Truro, N.S.	50	1	tr	3	3	0	1	1	0	0	0	0	0	0	0	tr	0	tr	tr	tr

* tr = trace

[1] *Contribution No. 712, Research Station, Agriculture Canada, Winnipeg, Manitoba R3T 2M9*

Table 2. Virulence of isolates of *Puccinia recondita* on backcross lines containing single genes for resistance to leaf rust in Canada in 1975

Resistance genes	No. of virulent isolates from:						Total no. of virulent isolates	% total isolates
	N.S.	Que. & Ont.	Man.	Sask.	Alta.	B.C.		
Lr1	2	0	4	2	1	0	9	2.8
Lr2a	0	0	18	11	18	0	47	14.4
Lr2c	6	3	19	11	18	4	61	18.7
Lr3	5	3	155	94	62	3	322	98.5
Lr3ka	5	0	1	0	0	0	6	1.8
Lr10	4	1	111	70	59	4	249	76.1
Lr16	0	0	1	0	0	0	1	0.3
Lr17	2	0	1	0	0	3	6	1.8
Lr18	4	3	17	5	1	1	31	9.5

Table 3. Virulence combinations of *Puccinia recondita* isolates on backcross lines containing single genes for resistance to leaf rust in Canada in 1975

Avirulence/virulence formula	No. of isolates from:						Total no. of isolates
	N.S.	Que. & Ont.	Man.	Sask.	Alta.	B.C.	
1,2a,2c,3ka,10,16,17,18/3	0	2	35	19	2	0	58
1,2a,2c,3ka,16,17,18/3,10	0	1	86	61	42	0	190
1,2a,2c,3ka,10,16,17/3,18	0	0	8	3	0	0	11
1,2a,3,3ka,10,16,17/2c,18	1	3	0	0	0	0	4
2a,2c,3ka,10,16,17,18/1,3	0	0	1	0	0	0	1
1,2a,2c,3ka,16,17/3,10,18	0	0	5	0	0	0	5
1,2a,2c,3ka,17,18/3,10,16	0	0	1	0	0	0	1
1,2a,3,3ka,16,17/2c,10,18	0	0	0	0	0	1	1
3ka,10,16,17,18/1,2a,2c,3	0	0	0	1	0	0	1
1,2a,16,17,18/2c,3,3ka,10	0	0	1	0	0	0	1
1,2a,3ka,16,18/2c,3,10,17	0	0	0	0	0	3	3
1,2a,10,16,17/2c,3,3ka,18	1	0	0	0	0	0	1
1,3ka,16,17,18/2a,2c,3,10	0	0	10	8	17	0	35
1,3ka,16,18/2a,2c,3,10,17	0	0	1	0	0	0	1
1,3ka,16,17/2a,2c,3,10,18	0	0	4	1	0	0	5
1,2a,16,17/2c,3,3ka,10,18	2	0	0	0	0	0	2
3ka,16,17,18/1,2a,2c,3,10	0	0	3	0	0	0	3
3ka,10,16,17/1,2a,2c,3,18	0	0	0	1	1	0	2
2a,16,18/1,2c,3,3ka,10,17	2	0	0	0	0	0	2

Physiologic specialization

Field collections of leaf rust were established on *Triticum aestivum* L. 'Little Club' wheat in the greenhouse and one single-pustule isolate was taken from each collection. Urediospores from the remaining pustules were collected and were bulked with other collections to give composites that were used to inoculate a group of highly resistant varieties of wheat.

A total of 327 cultures were established in 1975. Most of the collections were obtained from commercial fields of wheat varieties that do not possess any genes for seedling resistance.

The single-gene backcross lines used to study physiologic specialization in leaf rust were as previously decribed (1).

The distribution of virulence on the individual single-gene lines (Table 2) was similar to that obtained in 1974 (2). For the past 30 years, virulence on alleles of the *Lr2* locus has been very scarce in Manitoba and Saskatchewan. The presence of these genes in some wheat varieties presently being grown in the United States has led to increased incidence of virulence on *Lr2a* and *Lr2c*. Only one isolate virulent on *Lr16* was obtained in 1975. This was surprising since Selkirk was grown on 13.4% of the wheat acreage in Manitoba in 1975. This represents nearly 400,000 acres where virulence on *Lr16* would give a selective advantage and virulence on *Lr16* has been present for a number of years. In 1972, 125,000 acres of Agent wheat (*Lr24*) were grown in Oklahoma. This variety was lightly infected with a new virulent strain of leaf rust and virulence on *Lr24* was readily isolated in Manitoba that fall. This illustrates the profound effect on the Canadian leaf rust population of varieties grown in the south.

Nineteen virulence combinations were obtained in 1975 (Table 3). Only a few collections were obtained from eastern Canada and British Columbia. As a rule, the pattern of virulence in southern Alberta is markedly different from that obtained in Manitoba and Saskatchewan and resembles virulence patterns that occur on the west coast. In 1975, the rust population in southern Alberta was the same as that in Manitoba and Saskatchewan.

Composite collections of leaf rust were used to inoculate a number of highly resistant varieties of wheat. Three cultures virulent on Transfer (*Lr9*) were obtained from collections made in Manitoba and Saskatchewan. These are the first cultures virulent on *Lr9* to be isolated in Canada although virulence on *Lr9* in the Great Plains was first observed in North Dakota in 1973 (3).

Virulence on Agent (*Lr24*) was at a high level in 1975 but all isolates tested had a similar virulence pattern on other resistance genes. A backcross line containing gene *Lr21* was resistant to all cultures but there was considerable variation in rust development on this host. One isolate virulent on El Gaucho was obtained in 1975.

Acknowledgments

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Stem rust of wheat, barley, and rye in Canada in 1975[1]

G. J. Green

Wheat stem rust [*Puccinia graminis* f. sp. *tritici*] developed slowly in Canada in 1975 because of unfavorable weather in July. Rain in early August favored rust development in western Canada, and by mid-August stem rust was common on susceptible wheat varieties and wild grasses. Plots of susceptible varieties in Manitoba were severely attacked before harvest. There was less rust than usual in rust nurseries at 32 locations across Canada. There were no economically important changes in the physiologic races. Race C33(15B-1L) contined to predominate and races C25(38), C49(15), and C18(15B-1L) were fairly common. The wheat stem rust population exhibited the high level of variability observed in 1974. About twice as many races as usual were identified. If this level of variability continues, chances for the development of dangerous virulence combinations will increase.

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En 1975, la rouille de la tige du blé [*Puccinia graminis* f. sp. *tritici*] a connu une croissance lente au Canada en raison du mauvais temps qui a sévi au mois de juillet. Au début d'août, la pluie a favorisé le développement de la rouille dans l'ouest du Canada et, à la mi-août, le champignon était très répandu chez les variétés de blé et les graminées sauvages sensibles. Au Manitoba, certaines parcelles de variétés sensibles ont été gravement atteintes avant la récolte. La rouille était moins fréquente que d'habitude dans les parcelles d'étude sur la rouille à 32 endroits au Canada. Aucune modification économiquement importante n'a été observée chez les races physiologiques. La race C33 (15B-1L) a continué de dominer et les races C25(38), C49(15) et C18(15B-1L) étaient assez répandues. La population de rouille de la tige du blé a présenté le maximum de variabilité observé en 1974. Environ deux fois plus de races que d'habitude ont été identifiées. Si ce niveau de variabilité persiste, un accroissement des possibilités de développement de combinaisons virulentes est à prévoir.

Prevalence and importance in western Canada

Wheat stem rust [*Puccinia graminis* Pers. f. sp. *tritici* Eriks. and E. Henn.] was widespread in the United States by the end of July but infections were light. It was first observed in western Canada at Morden, Manitoba, on July 11, about 2 weeks later than usual. Hot, dry weather in July delayed rust development but rain in early August increased the rate of spread and by mid-August it was common on susceptible wild barley (*Hordeum jubatum* L.) throughout Manitoba and south-eastern Saskatchewan. Plots of susceptible wheat varieties at Morden and Portage la Prairie, Manitoba, were severely attacked before harvest. There was no rust on resistant varieties in farm fields.

Barley fields in the rust area of western Canada showed only traces of stem rust. Most barley varieties are resistant to wehat stem rust but they are susceptible to rye stem rust. An investigation of stem rust infections on wild barley indicated that rye stem rust developed too late in the season to cause damage.

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

Uniform rust nurseries consisting of 20 varieties of wheat, three varieties of barley, one variety of rye, and one variety of triticale were planted by cooperators at 32 locations across Canada in 1975. Most of the varieties used in the nurseries were discussed in a previous report (1). In 1975 the new commercial varieties Sinton, Macoun, and Wakooma were added.

Stem rust was less prevalent than usual in the nurseries. Moderate infections of wheat stem rust developed on the susceptible variety Red Bobs at only 4 of the 32 nursery locations (Table 1). Most rust occurred in the traditional rust area of Manitoba and eastern Saskatchewan, and at nearby Thunder Bay in northwestern Ontario. The small amount of stem rust in most nurseries seems to have been caused mainly by unfavorable weather early in the growing season.

The commercial varieties and sources of stem rust resistance planted in the nurseries continued to show high resistance (Table 1). The variety Pitic 62, which was susceptible in earlier years, was also resistant, indicating that races such as C35(32-113) have become less prevalent. The varieties Lee and Mindum are susceptible to the predominant race C33(15B-1L) but they were not severely infected.

Barley and rye were infected at 12 locations (Table 2) indicating that rye stem rust [*P. graminis* f. sp. *secalis*] was more widely distributed than wheat stem rust, especially in eastern Canada. The levels of infection in the nurseries suggest that rye stem rust was responsible for much of the infection on barley, although wheat stem rust probably contributed at the locations in western Canada.

Physiologic races

Wheat stem rust was widespread in western Canada in 1975 and 332 isolates were identified. Thirty-five

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Table 1. Percent infection of stem rust *(Puccinia graminis f. sp. tritici)* on 20 wheat varieties in uniform rust nurseries at 9 locations* in Canada in 1975

	Common wheat															Durum wheat				
Location	Red Bobs	Lee	Pitic 62	Neepawa	Napayo	Sinton	Kenya Farmer	C.I.8154 X Frocor[2]	Glenlea	Norquay	Exchange	Frontana	Thatcher[6] X Transfer	R.L.4255	Agatha	Hercules	Mindum	Wascana	Macoun	Wakooma
Creston, B.C.	30	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Melfort, Sask.	60	40	0	0	0	0	0	0	0	0	0	10	5	5	tr**	0	5	tr	0	0
Indian Head, Sask.	0	0	0	0	0	0	0	0	0	0	0	0	tr	0	0	0	0	0	0	0
Durban, Man.	5	tr	0	0	0	0	0	0	0	0	0	0	tr	0	0	0	tr	0	0	0
Brandon, Man.	20	tr	tr	0	0	0	0	0	0	0	0	tr	tr	tr	tr	tr	40	0	0	0
Morden, Man.	tr	0	0	0	0	0	0	0	0	0	tr	0	0	tr	0	0	0	0	0	0
Glenlea, Man.	60	1	tr	tr	tr	tr	1	tr	tr	tr	tr	tr	tr	tr	tr	tr	10	tr	tr	tr
Thunder Bay, Ont.	60	10	0	0	0	0	0	0	0	0	0	tr	10	5	5	0	15	0	0	0
Guelph, Ont.	60	tr	0	0	0	0	0	0	0	0	0	tr	5	tr	5	0	tr	0	0	0

* No rust was observed in nurseries at 23 locations: Agassiz, B.C.; Beaverlodge, Lacombe, Edmonton and Lethbridge, Alta.; Scott, Sask.; New Liskeard, Vineland, Sunbury, Appleton, Ottawa, Kemptville and Kapuskasing, Ont.; Macdonald College, Normandin, Lennoxville, Quebec and La Pocatière, Que.; Fredericton, N.B.; Kentville and Truro, N.S.; Charlottetown, P.E.I.; St. John's West, Nfld.

** tr = trace

Table 2. Percent infection of stem rust *(Puccinia graminis)* on three varieties of barley, one variety of rye, and one variety of triticale in uniform rust nurseries at 12 locations* in Canada in 1975

	Barley			Rye	Triticale
Location	Montcalm	Conquest	Wpg.-702-M.7118-13	Prolific	Rosner
Creston, B.C.	20	tr	0	tr	0
Melfort, Sask.	tr**	0	0	tr	0
Durban, Man.	0	0	0	10	0
Brandon, Man.	tr	0	5		0
Glenlea, Man.	10	tr	tr	0	tr
Thunder Bay, Ont.	0	0	0	tr	0
Guelph, Ont.	0	0	0	50	0
Sunbury, Ont.	5	0	0	25	0
Appleton, Ont.	0	0	0	10	0
Kemptville, Ont.	tr	0	0	tr	0
Macdonald College, Que.	0	0	0	tr	0
Lennoxville, Que.		tr	0	50	

* No rust was observed in nurseries at 19 locations: Agassiz, B.C.; Beaverlodge, Lacombe, Edmonton and Lethbridge, Alta.; Scott, Sask.; Morden, Man.; New Liskeard, Vineland, Ottawa and Kapuskasing, Ont.; Normandin, Quebec and La Pocatière, Que.; Fredericton, N.B.; Kentville and Truro, N.S.; Charlottetown, P.E.I.; St. John's West, Nfld.

Table 3. Distribution by provinces of physiologic races of *Puccinia graminis f. sp. tritici* on wheat, barley, and grasses, and frequency of isolation of *P. graminis f. sp. secalis* from barley and grasses in 1975

Virulence formula and (race) number	Virulence formula (effective/ineffective host genes)	Number of isolates from: Que.	Ont.	Man.	Sask.	Alta.	B.C.	Total number of isolates	Percent of total isolates
C 4 (23)	5,6,8,9a,9e,11,17,22,Tt1,Tt2/7a,10,15					1		1	0.3
C10 (15B-1)	6,7a,8,22,GB/5,9a,9b,9d,9e,10,11,13,14,15,17,Tt1,Tt2			1	1			2	0.6
C16 (39)	6,7a,8,9e,11,17,22,Tt2/5,9a,10,15						2	2	0.6
C17 (56)	6,8,9a,9b,9d,9e,11,13,17,22,Tt2/5,7a,10,14,15		1					1	0.3
C18 (15B-1L)	6,8,9a,9b,13,15,17,22,Tt2/5,7a,9d,9e,10,11,14,Tt1		2	6	5			13	3.9
C19 (38)	6,7a,9e,10,11,22,Tt2/5,8,9a,15,Tt1				1			1	0.3
C22 (32)	9a,9d,9e,13,22,Tt1,Tt2/5,6,7a,8,9b,10,11,14,15,17			2	1			3	0.9
C23 (38)	9a,9e,11,22,Tt1,Tt2/5,6,7a,8,10,15				1			1	0.3
C25 (38)	9a,9e,22,Tt1,Tt2/5,6,7a,8,10,11,15		2	20	2			24	7.3
C25 (38)	9a,9e,22,Tt1,Tt2/5,6,7a,8,10,11*,15			1				1	0.3
C27 (59)	6,8,9e,11,17,22,Tt1,Tt2/5,7a,9a,10,15						1	1	0.3
C32 (32)	9a,9b,9d,9e,11,13,22,Tt2/5,6,7a,8,10,14,15,17		1		1			2	0.6
C33 (15B-1L)	6,9a,9b,13,15,17,22,Tt2/5,7a,8,9d,9e,10,11*,14,Tt1		2	22	6			30	9.0
C33 (115)	6,9a,9b,15,17,22,Tt2/5,7a,8,9d,9e,10,11,Tt1			1		1		2	0.6
C33 (15B-L)	6,9a,9b,13,15,17,22,Tt2/5,7a,8,9d,9e,10,11,14,Tt1		4	173	33	1		211	63.6
C35 (32-113)	9d,9e,10,11,13,17,22,Tt2/5,6,7a,8,9a,9b,14,15,Tt1				2			2	0.6
C41 (32-113)	9d,9e,10,13,17,22,Tt2/5,6,7a,8,9a,9b,11,14,15,Tt1			1				1	0.3
C42 (15)	6,8,9a,9b,11,13,15,17,22,Tt2/5,7a,9d,9e,10,14,Tt1			1				1	0.3
C49 (15)	6,9a,9b,11,13,15,17,22,Tt2/5,7a,8,9d,9e,10,14,Tt1		1	13	2			16	4.8
C50 (15B-5)	7a,8,22/5,6,9a,9b,9d,9e,10,11,13,14,15,17,Tt1,Tt2			3				3	0.9
C53 (15B-1L)	6,9a,9b,13,15,22,Tt2/5,7a,8,9d,9e,10,11,14,17			1				1	0.3
C57 (32)	9a,9d,9e,22,Tt1,Tt2/5,6,7a,8,9b,10,11,13,14,15,17			1	1			2	0.6
C59 (31)	9d,9e,13,22,Tt1,Tt2/5,6,7a,8,9a,9b,10,11,14,15,17			3	1			4	1.2
C61 (38)	6,7a,9e,10,11,22,Tt2/5,8,9a,15,17,Tt1			1				1	0.3
C63 (32)	7a,9d,9e,10,11,13,17,22,Tt2/5,6,8,9a,9b,14,15,Tt1			3	1			4	1.2
C65 (38)	6,8,9e,11,17,22,Tt1,Tt2/5,7a,9a,10,15				1	1		2	0.6
Total wheat stem rust isolates			13	253	59	4	3	332	100
Rye stem rust isolates		1	6	85	14	11		117	

* Intermediate infection type; see text

isolates were from plots of the susceptible variety Klein Titan grown at Morden and 35 isolates were obtained from the susceptible variety Marquis at each of the locations Morden, Portage, and Brandon, Manitoba. Over 150 of the 253 Manitoba isolates were obtained from susceptible varieties at these three locations. The remaining isolates were from wild barley, and it is evident (Table 3) that wheat stem rust and rye stem rust were about equally prevalent on this host in Manitoba.

Physiologic races were identified by methods already described (1). A wheat line carrying resistance gene *SrTt1* was added to the differential hosts. When effective this gene produced a mesothetic reaction. It is included in the formulas in Table 3 for those races that produced clear infection types.

Twenty-six races were identified including two strains of race C25(38) and three of race C33 (Table 3). The common strain of race C25(38) is virulent on varieties with resistance gene *Sr11* but a new strain that produces an intermediate infection type was found. A strain of race C33(15B-1L) was separated from the common strain by the same intermediate virulence on *Sr11*. A third strain of race C33 was distinguished by its avirulence on the "standard" differential Marquis. Consequently it was not a "standard" race 15 but race 115.

Race C33(15B-1L) continued to predominate as it has since 1970. The three variants mentioned above comprised 73% of the isolates in 1975 (Table 3) and 63% in 1974. Race C25(38) was next in prevalence. It is avirulent on Marquis and Selkirk but is moderately virulent on seedlings of the widely grown varieties Manitou, Neepawa, and Napayo. Race C49(15), third in order of prevalence, resembles race C33(15B-1L) except that it is avirulent on resistance gene *Sr11*. Race C18(15B-1L) decreased from 11.2% of the isolates in 1974 to 3.9% in 1975.

No new races were identified in 1975 and only four of the races found were not present in 1974. The identification of races C10(15B-1), C17(56), and C50(15B-5) is noteworthy. Race C10(15B-1), the original 15B of 1950, was not found from 1964 to 1972 but it has been found in trace amounts each year since 1973. Race C17(56), the most important race in North America before 1950, has been found rarely since 1969. Race C50(15B-5), a widely virulent race that threatened Selkirk when it was widely grown, was found for the first time since 1960.

In 1975 the wheat stem rust population again exhibited the greatly increased variability observed in 1974. The reduced number of isolates in 1975 probably was

Table 4. Percent of total isolates and races avirulent on single identified resistance genes in 1975 and (1974)

Resistance gene	Avirulent isolates (%) 1975 (1974)	Avirulent races (%) 1975 (1974)
Sr 5	0.3 (0.2)	4 (3)
Sr 6	85.8 (88.7)	58 (72)
Sr 7a	3.9 (4.1)	23 (22)
Sr 8	7.8 (20.7)	35 (44)
Sr 9a	93.1 (91.3)	58 (53)
Sr 9b	93.6 (85.2)	56 (50)
Sr 9d	6.4 (7.7)	50 (44)
Sr 9e	16.0 (16.3)	65 (63)
Sr10	2.7 (7.5)	19 (31)
Sr11	10.5 (11.3)	52 (34)
Sr13	97.6 (90.2)	81 (75)
Sr14	0 (0.2)	0 (3)
Sr15	82.5 (82.2)	27 (28)
Sr17	90.2 (86.1)	63 (56)
Sr22	100.0	100 (100)
SrTt2	98.5 (99.5)	92 (97)

responsible for the identification of a slightly smaller number of races (26) than in 1974 (32). In earlier years about 12 races were found. The greatly increased number of races in the last 2 years suggests that factors such as new selection pressures are influencing rust evolution. The increased variability is disturbing from the practical point of view because, if continued, it could result in new and more virulent rust variants that would threaten resistant commercial varieties.

The finding of rust variants that have intermediate virulence on resistance gene *Sr11* produced a nomenclatural problem. The genetic basis of the intermediate condition is uncertain and until there is a better understanding of what happened an asterisk placed beside the gene in question (Table 3) is being used to indicate an intermediate interaction.

The percentages of races and isolates avirulent on the identified resistance genes have not changed appreciably in the last year (Table 4). The small increase in virulence on *Sr8* was caused by the increase of race C33. The genes conferring resistance to most isolates and races were *Sr22, SrTt2* and *Sr13*. Although *Sr6, Sr9a* and *Sr9b* conferred resistance to the predominant races and to most isolates, over 40% of the races were virulent on them.

Thirty-two highly resistant varieties were inoculated with 11 composite collections of urediospores from all isolates identified. Most results resembled those of earlier years with many varieties being resistant (Table 5), but some reactions are noteworthy. The varieties Chris, Mida-McMurachy-Exchange II-47-26, Frontana-K58-Newthatch II-50-17, and Sinton showed large pustules, as they have in previous years. However, a few large pustules were observed on Era, Glenlea, and Webster for the first time since composite urediospore collections came into use. Preliminary results with isolates from these large infections indicate they are virulent on seedlings of these varieties.

Table 5. Infection types produced on 32 resistant varieties by 11 composite collections of urediospores from 332 isolates of wheat stem rust collected in 1975

	Lowest	*Highest*
Agatha	0	2
Agent	0	2
Chris	0	; to 3+
C.I.8154 X Frocor[2]	0	; to 2
D.T.411	0	;
Era	0	; to 3+
Esp 518/9	0	; to 2
Glenlea	0	; to 3+
Hercules	0	1
Frontana-K58-Newthatch II-50-17	0	4
Kenya Farmer	0	2 to 3+
Macoun	;1	1
Mida-McMurachy-Exchange II-47-26	0	; to 4
Norquay	0	2
N.D.499	0	; to 2
N.D.506	0	2±
(P X Mq[8])6 X (Rsc X Etoile de Choisi)	0	2
Romany	0	;1
Rosner triticale	0	;
R.L.4308	0	; to 4
R.L.4311	0	; to 2+
R.L.4320	0	; to 4
R.L.4326	0	2 to 3
R.L.5405	0	2
Sinton	0	; to 4
St464	0	;1
Tama	0	; to 2
T[6] X (Rsc X Etoile de Choisi)	0	2
Wascana	;1	1+
Wakooma	;1	1+
Webster	0	2 to 3+
WRT240 (Manitou with rye translocation)	0	;

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Crown rust of oats in Canada in 1975[1]

D. E. Harder

Oat crown rust [*Puccinia coronata* f. sp. *avenae*] did not cause significant crop losses in western Canada in 1975. There was no increase from 1974 in virulence on the currently most resistant commercial oat (*Avena sativa*) cultivar Hudson and on the important resistance genes *Pc38* and *Pc39*. The occurrence of standard races of crown rust across Canada was determined. In western Canada races 295 and 326 predominated, while in eastern Canada race 210 predominated. Virulence combinations in the crown rust population were also determined using a set of 12 oat lines carrying single substituted genes (*Pc*) for crown rust resistance. The 333 isolates from western Canada and 56 isolates from eastern Canada comprised 18 and 40 virulence combinations respectively. There was little change from 1974 in the levels of virulence on the *Pc* genes except for some increase in virulence on lines with genes *Pc50* and *Pc56*.

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En 1975, la rouille couronnée de l'avoine [*Puccinia coronata* f. sp. *avenae*] n'a pas causé de pertes importantes de récoltes dans l'ouest du Canada. Par rapport à 1974, on n'a constaté aucun accroissement de virulence sur le cultivar Hudson normalement le plus résistant de l'avoine commerciale (*Avena sativa*) ni sur les principaux gènes de résistance, soit Pc38 et Pc39. La fréquence des races courantes de rouille couronnée au pays a été déterminée. Dans l'Ouest, les races 295 et 326 ont dominé, comparativement à la race 210 dans l'Est. Certaines combinaisons de virulence chez les populations de rouille couronnée ont également été déterminées au moyen d'une série de 12 lignées d'avoine porteuses d'un seul gène substitué différent (*Pc*) de résistance à la rouille. Les 333 isolats provenant de l'Ouest et les 56 de l'Est comprenaient respectivement 18 et 40 combinaisons de virulence. On a constaté peu de changement, par rapport à 1974, du degré de virulence sur les lignées possédant le gène Pc, sauf un léger accroissement sur celles possédant les gènes Pc50 et Pc56.

Occurrence in western Canada

Oat crown rust caused by *Puccinia coronata* Cda. f. sp. *avenae* Eriks. did not cause significant losses in oat crops in most localities in 1975. Buckthorn (*Rhamnus cathartica* L.) infection was light in the spring of 1975, and general infection of oats did not occur until mid July. Although the early season weather was favorable for rust development, much of July and August was very warm and dry, limiting rust spread. In a few localities isolated showers occurred which allowed crown rust to attain moderately severe levels in some late sown fields.

Physiologic Specialization

All isolates of crown rust from eastern Canada were obtained from uniform rust nurseries grown at Lennoxville, La Pocatière, L'Assomption, and Macdonald College, Quebec; and Elphin, Appleton, Brantford, Vineland, Guelph, and Ottawa, Ontario. In western Canada the isolates were obtained from field surveys throughout Manitoba and in eastern Saskatchewan.

In 1975 all crown rust collections were assessed using the "standard" differential oat varieties to monitor evolutionary changes in virulence (Table 1). The 40 isolates obtained from eastern Canada comprised 17 "standard" races, giving a race/isolate ratio of 0.425. As in the last report (2) race 210 predominated in eastern Canada, and increased to 42.5% of isolates as compared to 26% in 1973. Insufficient collections were obtained from eastern Canada to reliably assess the occurrence of the less common races.

In western Canada 282 isolates comprised 23 races giving a race/isolate ratio of 0.085. The difference in race/isolate ratios between eastern and western Canada may be a reflection of the difference in numbers of isolates, but the widespread occurrence of *R. cathartica* in parts of eastern Canada may also be a factor. As in 1973 (2) race 295 predominated, but decreased slightly to 35.5% of isolates. Race 326 increased from 21% in 1973 to 32.6% in 1975. Race 335 increased in prevalence from 2.9% in 1973 to 8.5% in 1975.

All crown rust collections were also tested on a series of backcross lines of *Avena sativa* L. cv. Pendek containing single genes for crown rust resistance derived from *Avena sterilis* L. (Table 2). In 1975 there were a total of 44 virulence combinations, 18 from eastern Canada (race/isolate ratio of 0.32), and 40 from western Canada (race/isolate ratio of 0.12). In 1975 there was relatively little change in the number of isolates from both eastern and western Canada that were avirulent on the single *Pc*-gene lines. As in 1974 (1) virulence predominated on genes *Pc35* and *40*, either singly or in combinations, in western Canada, and on *Pc35* in eastern Canada (Table 3). There was increased virulence on gene *Pc50* and on the newly isolated gene *Pc56* in eastern and western Canada, but the levels of virulence

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Table 1. Distribution of physiologic races of crown rust in Canada in 1975

Physiologic race	Eastern Canada		Western Canada	
	No. of isolates	% of isolates	No. of isolates	% of isolates
203	0	0.0	10	3.6
209	0	0.0	2	0.7
210	17	42.5	1	0.4
211	1	2.5	0	0.0
212	0	0.0	1	0.4
216	0	0.0	5	1.8
226	1	2.5	3	1.1
228	3	7.5	0	0.0
231	3	7.5	2	0.7
232	1	2.5	0	0.0
237	0	0.0	2	0.7
259	1	2.5	0	0.0
263	0	0.0	3	1.1
264	0	0.0	12	4.3
281	1	2.5	0	0.0
284	1	2.5	8	2.8
294	1	2.5	2	0.7
295	0	0.0	100	35.5
297	1	2.5	0	0.0
299	1	2.5	1	0.4
320	2	5.0	5	1.8
322	1	2.5	1	0.4
325	0	0.0	3	1.1
326	2	5.0	92	32.6
330	2	5.0	1	0.4
335	0	0.0	24	8.5
345	0	0.0	1	0.4
410	0	0.0	1	0.4
427	1	2.5	2	0.7
Total	40		282	

Table 2. Virulence combinations of *Puccinia coronata* on backcross lines containing single *(Pc)* genes for resistance

Virulence formula effective/ineffective *Pc* genes	Eastern Canada		Western Canada	
	No. of isolates	% of isolates	No. of isolates	% of isolates
35,38,39,40,45,46,47,48,50,54,55,56/	20	35.6	70	21.0
38,39,40,45,46,47,48,50,54,55,56/35	7	12.4	35	10.5
35,39,40,45,46,47,48,50,54,55,56/38	0	0.0	3	0.9
35,38,39,45,46,47,48,50,54,55,56/40	1	1.8	62	18.5
35,38,39,40,46,47,48,50,54,55,56/45	1	1.8	0	0.0
35,38,39,40,45,47,48,50,54,55,56/46	1	1.8	6	1.8
35,38,39,40,45,46,47,48,54,55,56/50	0	0.0	17	5.1
35,38,39,40,45,46,47,48,50,55,56/54	0	0.0	9	2.7
35,38,39,40,45,46,47,48,50,54,55/56	7	12.4	4	1.2
39,40,45,46,47,48,50,54,55,56/35,38	0	0.0	1	0.3
38,39,45,46,47,48,50,54,55,56/35,40	0	0.0	26	7.8
38,39,40,46,47,48,50,54,55,56/35,45	2	3.6	4	1.2
38,39,40,45,47,48,50,54,55,56/35,46	1	1.8	5	1.5
38,39,40,45,46,47,48,54,55,56/35,50	2	3.6	14	4.2
38,39,40,45,46,47,48,50,55,56/35,54	1	1.8	8	2.4
38,39,40,45,46,47,48,50,54,55/35,56	3	5.4	3	0.9
35,39,40,46,47,48,50,54,55,56/38,45	0	0.0	1	0.3
35,38,39,46,47,48,50,54,55,56/40,45	0	0.0	1	0.3

Table 2. (Cont.)

Virulence formula effective/ineffective *Pc* genes	Eastern Canada No. of isolates	Eastern Canada % of isolates	Western Canada No. of isolates	Western Canada % of isolates
35,38,39,45,47,48,50,54,55,56/40,46	0	0.0	2	0.6
35,38,39,45,46,47,50,54,55,56/40,48	0	0.0	1	0.3
35,38,39,45,46,47,48,54,55,56/40,50	0	0.0	11	3.3
35,38,39,45,46,47,48,50,55,56/40,54	0	0.0	11	3.3
35,38,39,45,46,47,48,50,54,55/40,56	1	1.8	6	1.8
35,38,39,40,47,48,50,54,55,56/45,46	2	3.6	0	0.0
35,38,39,40,46,47,48,50,55,56/45,54	2	3.6	0	0.0
35,38,39,40,45,47,48,54,55,56/46,50	0	0.0	1	0.3
35,38,39,40,45,46,47,48,55,56/50,54	0	0.0	2	0.6
35,38,39,40,45,46,47,48,54,55/50,56	0	0.0	1	0.3
38,39,46,47,48,50,54,55,56/35,40,45	0	0.0	1	0.3
38,39,45,47,48,50,54,55,56/35,40,46	2	3.6	1	0.3
38,39,45,46,47,48,54,55,56/35,40,50	1	1.8	7	2.1
38,39,45,46,47,48,50,55,56/35,40,54	0		3	0.9
38,39,40,46,47,48,50,54,55/35,45,56	1	1.8	3	0.9
38,39,40,45,47,48,50,54,55/35,46,56	1	1.8	0	0.0
38,39,40,45,46,47,48,55,56/35,50,54	0	0.0	2	0.6
35,38,39,46,47,48,54,55,56/40,45,50	0	0.0	1	0.3
35,38,39,46,47,48,50,55,56/40,45,54	0	0.0	2	0.6
35,38,39,45,46,47,48,55,56/40,50,54	0	0.0	2	0.6
35,38,39,40,47,48,50,55,56/45,46,54	0	0.0	2	0.6
35,38,39,40,46,47,48,55,56/45,50,54	0	0.0	1	0.3
38,39,47,48,50,54,55,56/35,40,45,46	0	0.0	1	0.3
38,39,46,47,48,50,55,56/35,40,45,54	0	0.0	1	0.3
38,39,45,47,48,54,55,56/35,40,46,50	0	0.0	1	0.3
38,39,45,46,47,48,55,56/35,40,50,54	0	0.0	1	0.3
Total	56	100.0	333	100.0

Table 3. Distribution of virulence of isolates of *Puccinia coronata* in 1975 on the standard differential varieties and on backcross lines carrying single crown rust resistance genes

Variety or resistance gene	Eastern Canada No. of virulent isolates	Eastern Canada % of isolates	Western Canada No. of virulent isolates	Western Canada % of isolates
Anthony	15	37.5	274	97.2
Victoria	6	15.0	108	38.3
Appler	7	17.5	259	91.8
Bond	29	72.5	270	95.7
Landhafer	5	12.5	240	85.1
Santa Fe	4	10.0	215	76.2
Ukraine	40	100.0	270	95.7
Trispernia	0	0.0	16	5.7
Bondvic	0	0.0	16	5.7
Saia	3	7.5	2	0.7
Pc35	19	33.9	104	31.2
Pc38	0	0.0	5	1.5
Pc39	0	0.0	0	0.0
Pc40	4	7.1	136	40.8

Table 3. (Cont.)

Variety or resistance gene	Eastern Canada		Western Canada	
	No. of virulent isolates	% of isolates	No. of virulent isolates	% of isolates
Pc45	5	8.9	15	4.5
Pc46	6	10.7	20	6.0
Pc47	0	0.0	0	0.0
Pc48	0	0.0	1	0.3
Pc50	4	7.1	61	18.3
Pc54	3	5.4	43	12.9
Pc55	0	0.0	0	0.0
Pc56	13	23.2	11	3.3

on the other genes remained relatively constant across Canada. Gene *Pc55*, another recently isolated gene, provided resistance against all crown rust isolates in Canada in 1975. There was no change in virulence on lines with genes *Pc38* and *Pc39*, which are presently being used to provide crown rust resistance in the oat breeding program at Winnipeg, thus these genes continue to be effective resistance sources. There was also no increase in virulence on the presently most resistant commercial cultivar, Hudson.

Acknowledgements

The cooperators who cared for nurseries and submitted rust collections from the various locations in Canada are thanked. Mr. W. L. Timlick carried out all technical operations.

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Stem rust of oats in Canada in 1975[1]

J. W. Martens and R. I. H. McKenzie

Stem rust [*Puccinia graminis* f. sp. *avenae*] was first found on oats (*Avena sativa*) in mid July. Light infections developed on late fields in Manitoba and eastern Saskatchewan. Races C10 and C23 continue to predominate in western Canada with 55% and 36% of all isolates. In eastern Canada, the predominant race was C9. A culture of race C9 with virulence on resistance gene *Pg13* was again found in both eastern and western Canada.

Can. Plant Dis. Surv. 56:23-24. 1976

La rouille de la tige [*Puccinia graminis* f. sp. *avenae*] a été observée pour la première fois chez l'avoine (*Avena sativa*) à la mi-juillet. De légères infections se sont développées dans certaines plantations tardives au Manitoba et dans l'est de la Saskatchewan. Les races C10 et C23 ont continué de dominer dans l'Ouest avec 55 et 36% respectivement de tous les isolats. Dans l'Est, la race C9 a prédominé. Une culture de la race C9 virulente sur les variétés possédant le gène Pg13 de résistance a également été observée dans l'est et l'ouest du Canada.

Prevalence and crop losses in western Canada

Stem rust of oats caused by *Puccinia graminis* Pers. f. sp. *avenae* Eriks and E. Henn. was first found in southern Manitoba on July 10, somewhat earlier than normal. Light infections developed throughout most of Manitoba and eastern Saskatchewan but they caused no crop losses except in small areas in central and eastern Manitoba where a few late fields with infections exceeding 50% of the stem area were observed in late August.

Uniform rust nurseries

Rust nurseries comprising the oat, *Avena sativa* L., cultivars Fraser, Hudson, Rodney, C.I. 3034, C.I. 4023, C.I. 9139, R.L. 2924, R.L. 2925, R.L. 2926, and R.L. 2970 were grown at 31 locations across Canada. Trace to moderate infections of rust were observed at Fredericton, N.B.; Lennoxville, Que.; Guelph and Sunbury, Ont.; and Brandon, Man. No rust infections were observed in nurseries grown at St. John's West, Nfld.; Charlottetown, P.E.I.; Kentville, and Truro, N.S.; Macdonald College, Normandin, La Pocatière and Québec, Qué.; Appleton, Kapuskasing, Kemptville, New Liskeard, Ottawa, Thunder Bay, and Vineland, Ont.; Durban and Morden, Man.; Indian Head, Melfort and Scott, Sask.; Beaverlodge, Edmonton, Lacombe and Lethbridge, Alta., and Agassiz and Creston, B.C.

Physiologic races

Field collections were established on the oat cultivar Victory and physiologic races were identified by the infection types produced on seedlings of 'Rodney 0' single-gene lines as indicated in Table 1. A supplementary set comprising the cultivars C.I. 9139 (unknown genotype) and R.L. 2926 (*Pg 13*) was also included in the study. All 229 field cultures were avirulent on C.I. 9139 resistance, but two cultures of C9 from Ontario and one C9, two C28, and one C29 from Manitoba were virulent on *Pg 13* resistance. Races C10 and C23 continued to predominate in western Canada and comprised 55% and 36% of all isolates, respectively. This represents a resurgence of race C23 from a low of 7% in 1974 to its former levels (2). Numerous rare and several new races also appeared in western Canada. The avirulent race C26 (*Pg 1, 2, 3, 4, 8 9, 13*/) first found in experimental plots in 1973 (1) was isolated from a field collection made at Poplar Point, Man., and the discovery of races C28 and C29, both with virulence on *Pg 13*, adds to the growing list (1) of races with virulence on this source of resistance. It is interesting that virulence on *Pg 13* has developed in spite of the fact that no commercial oats are grown that possess this resistance gene. With the exception of race C9, none of the races so far identified presents a threat to the cultivar Hudson, which combines resistance conferred by genes *Pg 2, Pg 4* and *Pg 9*. The frequency of virulence on lines carrying single resistance genes (Table 2) has in most cases declined from the previous year (2), but the appearance of virulence on *Pg 13* in field cultures in western Canada is cause for concern.

[1] *Contribution No. 716, Research Station, Agriculture Canada, Winnipeg, Manitoba R3T 2M9.*

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

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Table 1. Distribution of physiologic races of oat stem rust in Canada in 1975

Race no.	Virulence formula (effective/ineffective *Pg* host genes)	No. of isolates from: N.S. + N.B.	Qué.	Ont.	Man.	Sask.	Total isolates	Percentage of total isolates
C 1	1,2,3,4,8/9				1		1	0.4
C 4	1,4,8,9/2,3			1			1	0.4
C 5	4,9/1,2,3,8					1	1	0.4
C 6	1,8/2,3,4,9			1			1	0.4
C 7	1/2,3,4,8,9			1			1	0.4
C 8	3,8/1,2,4,9	1		4			5	2.2
C 9	8/1,2,3,4,9			9	1		10	4.4
C10	9/1,2,3,4,8	1		3	86	28	118	51.5
C14	8,9/1,2,3,4	1			6		7	3.0
C17	1,3,8/2,4,9			1			1	0.4
C19	1,2,4,8,9/3					2	2	0.9
C21	1,8,9/2,3,4	1					1	0.4
C23	2,4,9,13/1,3,8		1		61	13	75	33.0
C24	1,2,8/3,4,9,13				1		1	0.4
C26	1,2,3,4,8,9,13/				1		1	0.4
C28	3,8,9/1,2,4,13				2		2	0.9
C29	3,9/1,2,4,8,13				1		1	0.4
Total		4	1	20	160	44	229	

Table 2. Frequence of virulence in the oat stem rust population on various types of resistance in eastern and western Canada in 1975

Source of isolates	Percentage of isolates virulent on cultivars with the following genes for resistance: *Pg1*	*Pg2*	*Pg3*	*Pg4*	*Pg8*	*Pg9*	*Pg13*
East	80.0	96.0	76.0	92.0	24.0	68.0	8.0
West	97.5	61.2	97.5	61.2	92.1	1.4	1.4

Yield loss conversion factors for fusarium root rot of pea[1]

P.K. Basu[2], N.J. Brown[2], R. Crête[3], C.O. Gourley[4], H.W. Johnston[5], H.S. Pepin[6], and W.L. Seaman[2]

A method for relating the severity of root rot caused by *Fusarium solani* f. sp. *pisi* to yield losses in green pea (*Pisum sativum*) was developed from experimental plots at Ottawa and from commercial crops in five provinces. On the basis of 3 years results with several pea cultivars, severely affected plants showed an average yield loss of 57%. Moderately affected plants showed an average yield reduction of 35% in experimental plots, but a similar severity-loss relationship for the moderate severity level was not confirmed in commercial fields. It is suggested that a conservative estimate of yield loss in growers' fields may be obtained by multiplying the percentage of severely affected plants by a factor of 0.57.

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On a mis au point une méthode permettant d'établir un rapport entre la gravité du pourridié fusarien causé par *Fusarium solani* f. sp. *pisi* et les baisses de rendement des petits pois (*Pisum sativum*) à partir de parcelles expérimentales à Ottawa et de cultures commerciales dans cinq provinces différentes. D'après les résultats de trois ans obtenus de plusieurs cultivars de pois, les plants gravement atteints ont montré une baisse de rendement moyenne de 57%. Les plants moyennement touchés ont montré une baisse moyenne de 35% dans les parcelles expérimentales, mais il a été impossible de confirmer un rapport gravité de la maladie-baisse de rendement semblable pour une infestation modérée dans les cultures commerciales. Toute porte à croire qu'il est possible d'obtenir une estimation prudente des baisses de rendement des plantations commerciales en multipliant le pourcentage de plants gravement atteints par un facteur de 0.57.

In Canada *Fusarium solani* f. sp. *pisi* is considered to be the primary causal agent of root rot of green pea (*Pisum sativum* L.), although *F. oxysporum* f. sp. *solani* and species of *Ascochyta, Rhizoctonia*, and possibly *Aphanomyces* also may be involved (1,3,4,5,10,11). Attempts to establish a relationship between levels of *Fusarium* propagules in soil and disease development have been inconclusive (2,8,9). Johnson (8) developed a useful method for estimating the hazard of planting peas in infested soil but it could not be used for predicting yield losses. In some fields, root rot has caused partial (9) to complete loss of pea crops (1,4,11), but it has also been shown experimentally that pea plants can recover from the disease when root development continues beyond the zone of infested soil (3). In our previous survey (1), no consistent relationship was found between the percentage of plants affected by root rot and the bulk yield of shelled peas from growers' fields.

The aim of the present work was to find if a consistent relationship between the severity of root rot and yield loss could be established by comparing the yield of healthy plants with that of diseased plants in field plot experiments and in commercial fields.

Materials and methods

Disease severity rating and yield

Preliminary attempts to measure the severity of pea root rot in greenhouse-grown plants showed that a quantitative estimation of the percentage of the root system damaged was not feasible. In this study severity was judged primarily on the length of the brown to black discoloration of the lower stem and primary root region (epicotyl and hypocotyl). Field grown plants were dug carefully at a stage of development suitable for processing, and the following root rot categories were adopted to classify them according to the overall disease symptoms:

Root rot categories

0 No discoloration of roots, plants apparently healthy but may have few (4 to 6) lower leaves chlorotic or dry due to natural senescence

1 A trace to 2 cm brown to black discoloration of the lower stem and tap root region, lateral roots not discolored, plants apparently healthy

2 Up to 4 cm brown to black discoloration of the lower stem and tap root region, lateral roots not discolored, plants apparently healthy

3 Up to 5 cm brown to black discoloration of the lower stem and tap root region, lateral roots turning brown; plants showing slight yellowing of leaves apart from normal senescence

4 Six cm or more brown to black discoloration of the lower stem and tap root region, most lateral roots decayed, most leaves yellowed; plants often stunted, wilted, moribund, or killed

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Table 1. Yield[a] of Jade pea plants grown in root rot infested field plots, 1972; plants grouped in five severity categories

	Root rot severity category[b]				
Plot no.	0	1	2	3	4
1	4.16	3.21	2.42	1.14	1.56
2	6.30	4.05	5.00	2.82	2.46
3	4.96	3.99	2.77	3.06	1.76
Mean yield[c]	4.85	3.67	3.05	2.48	1.84
Yield loss (%)[d]		24.00	37.00	49.00	62.00
Plants/category (%)[e]	50.10	24.10	3.10	3.40	19.30

[a] Avg yield of shelled green peas (g/plant), oven-dry basis.

[b] 0 = healthy, 4 = severe root rot.

[c] Means underscored by the same line are not significantly different by the Duncan's multiple range test at P = 0.05.

[d] Based on the mean yield of plants in healthy (0) category.

[e] Based on 1,677 plants sampled from the 3 plots.

Yield of shelled peas from plants of each root rot category was expressed as the average oven-dry weight of seeds per plant unless otherwise stated. The yield loss in the diseased categories was based on the yield of healthy plants.

Field plot experiments

At Ottawa the relationship between root rot severity and yield loss was studied during 1972-74 in a 0.28 ha field that had been artificially infested in 1971 with propagules of *Fusarium solani* (Mart.) Sacc. f. sp. *pisi* (Jones) Snyder and Hansen. The viability of the pathogen was checked annually in spring and fall by a dilution plate method (12). The field was subdivided into plots and pea seed obtained from commercial processors or seed companies was sown at the usual spacing, 5 cm × 18 cm, with a grain drill in early June each year. Plants were harvested when most of the green pods were filled, 60 to 70 days after planting. Earlier studies (P.K. Basu, unpublished) indicated that it was not possible to maintain *Fusarium*-free plots within the test area to serve as controls because of cross contamination. Hence, plants without root rot symptoms were used as controls.

In 1972 the cultivar Jade was grown in 3 large plots, each 12.8 m x 66.1 m. In each plot 50 consecutive plants of a row were sampled at each of 10 equally spaced sites located along a W pathway, discarding a 2-m-wide border; the plants were rated for root rot using the five categories described. In 1973 the cultivars Jade and Thomas Laxton were grown in a randomized block design with six replicated plots. A total of 300 plants (10 per site) were sampled from each plot at 30 predetermined sites; 10 of the sites were selected along a W, 10 on a diagonal, and 10 at random. These plants were placed in three instead of five root rot categories by combining categories 1 to 3. In 1974, 10 pea cultivars, Anoka, Charger, Dark Skin Perfection, Jade, Mars, Nugget, Trojan, Venus, Asgrow XPF 3007, and Asgrow 4683 were grown with four replications of each in a randomized block design. A stand count was made after emergence. At harvest, the number and yield of all plants showing symptoms of root rot category 4 in each plot were recorded. The percentage and yield of plants belonging to categories 0 (healthy) and 1-3 (moderately affected) were estimated from 2-3 groups of 100 plants taken from one-half of each plot, divided diagonally; plants from the other half of each plot provided the bulk fresh weight yield of shelled peas.

Sampling in growers' fields

Based on previous records (1), root rot affected fields were chosen for sampling in 1972, 1973, and 1974 in the main pea growing areas of British Columbia, Ontario, Quebec, Nova Scotia, and Prince Edward Island. Samples were taken when the crop was ready fo machine-harvesting for freezing or canning. In 1972 a systematic sampling procedure was adopted; 100 successive plants in a row were removed from each of 10 sites located equidistantly along the arms of a W pattern (1) in each field. Plants were placed in the appropriate root rot categories, and the average seed yield (oven-dry basis) per plant was recorded for each category. In 1973 and 1974, yield comparisons were based on a paired sample of 100 severely affected (root rot 4) and 100 "healthy" (root rot 0-3) plants from each field. Usually the severely affected plants were found in distinct patches containing yellow to brown plants; the apparently healthy plants were taken from nearby green areas.

Results

Field plot experiments

In the 1972 experiment with the cultivar Jade, plants of root rot categories 1, 2, 3, and 4 yielded 24%, 37%, 49%, and 62% less, respectively, than the plants classed as healthy (Table 1). These results suggested that there was a direct correlation between yield loss and root rot severity. However, the mean yields of adjacent disease categories were not significantly different. In these samples the numbers of plants in each category were not equivalent, and hence the values of categories 2 and 3 may have received undue weight because they contained relatively few plants. These results further indicated that mildly to moderately affected plants may be grouped into one category (root rot 1-3), thus eliminating many borderline plants and saving considerable time in judging severity.

In the 1973 experiment with the cultivars Jade and Thomas Laxton (Table 2), significant yield differences occurred among the three (0, 1-3, and 4) root rot categories. Plants in the latter two categories showed losses of 26% and 58% in Jade and 45% and 69% in Thomas Laxton. The loss values of the most severely affected plants in both 1972 and 1973 were similar, e.g. 62% and 58% in Jade and 69% in Thomas Laxton

Table 2. Yield[a] of Jade and Thomas Laxton pea plants grown in root rot infested field plots, 1973; plants grouped in three severity categories[b]

	Jade			Thomas Laxton		
	Root rot severity category			Root rot severity category		
Plot no.	0	1-3	4	0	1-3	4
1	2.82	2.78	1.67	2.75	1.92	1.68
2	3.97	3.15	1.84	4.94	2.84	1.74
3	4.40	3.04	1.57	5.67	3.13	1.76
4	5.16	3.80	2.06	2.92	2.17	1.01
5	4.63	3.33	1.77	2.78	3.20	1.55
6	5.16	3.30	2.03	9.20	2.16	0.94
Mean yield[c]	4.36	3.23**	1.82**	4.71	2.57*	1.45*
Yield loss (%)[d]		26.00	58.00		45.00	69.00
Plants/category (%)[e]	9.70	65.10	25.20	6.60	56.50	36.90

[a] Avg yield of shelled green peas (g/plant), oven-dry basis.

[b] 0 = healthy, 4 = severe root rot.

[c] Mean yields indicated by * and ** are significantly different at P - 0.05 and 0.01, respectively.

[d] Based on the mean yield of plants in the healthy (0) category.

[e] Based on 1,800 plants of each cultivar from the 6 plots.

(1973 only). Whether or not a common loss factor could be applied to all cultivars was considered further the following year.

In the 1974 field plot experiment, losses were estimated for 10 pea cultivars on the basis of yield per plant and percentage of plants in the three root rot categories (Table 3). In all cultivars the yield of healthy plants was significantly higher than that of severely affected ones; the yield of moderately affected plants (root rot 1-3) was significantly different from that of healthy plants in 6 of 10 cultivars and from that of severely affected plants in 3 of 10 cultivars (Table 3). The yield loss per plant within the moderately infected group ranged from 6.6% to 48.8% with an average of 27.4% ± 2.6%. Per plant yield loss of severely infected plants ranged from 25% to 66.6% with an average of 44.9% ± 1.8%. The standard error values, 2.6 for the moderate and 1.8 for the severe categories, were less than 10% of the respective grand means (Table 3, cols. 4 and 5). The low SE values afforded some justification for averaging the loss values of different cultivars to obtain a common loss factor for the two levels of disease severity. In this experiment, the majority of the plants belonged to the moderately diseased category (62.1%) followed by those in the healthy (32.7%) and those in the severely diseased (5.2%) categories. The percent yield loss per plant of a category, expressed as a fraction, multiplied by the percentage of plants in that category resulted in a loss value for the category; the sum of losses from the disease categories represented the total loss for each cultivar. On average, the yield loss for all cultivars was estimated at 19.9% (Table 3). However, despite an overall loss of about 20%, the average bulk fresh weight of shelled peas from the plots was 5042 kg/ha (over 2 tons/acre), which was comparable to the average pea

Table 3. Estimated yield losses in 10 pea cultivars grown in root rot infested field plots, 1974, plants grouped in three severity categories; 0, 1-3 and 4

Cultivar	Avg yield (g) per plant[a]			Yield loss (%) per plant[b]		% of total plants in each category			Yield loss (%) per category		Total loss (%) per cultivar
	0	1-3	4	1-3	4	0	1-3	4	1-3	4	
Anoka	8.4	4.3	4.1	48.8 p	51.2 pqr	19.1	70.4	10.5	34.3	5.4	39.7
Charger	2.4	1.9	1.8	20.8 qrs	25.0 rst	37.4	59.2	3.4	12.3	0.9	13.2
Dark Skin Perfection	2.0	1.6	1.4	20.0 qrs	30.0 st	41.2	56.0	2.8	11.2	0.8	12.0
Jade	4.6	2.8	2.6	39.1 pqr	43.5 qrs	35.7	60.2	4.1	23.5	1.8	25.3
Mars	3.0	2.5	1.0	16.6 rs	66.6 p	21.5	70.5	8.0	11.7	5.3	17.0
Nugget	1.3	1.2	0.9	7.7 s	30.8 rst	42.0	53.5	4.5	4.1	1.4	5.5
Trojan	5.9	3.8	2.0	35.6 pqrs	66.1 p	30.5	65.5	4.0	23.3	2.6	25.9
Venus	5.0	3.3	2.4	34.0 pqrs	52.0 pqr	33.0	62.5	4.5	21.2	2.3	23.5
XPF 3007 (Asgrow)	4.2	2.3	1.8	45.2 pq	57.1 pq	29.3	63.5	7.2	28.7	4.1	32.8
#4683 (Asgrow)	1.5	1.4	1.1	6.6 s	26.6 st	37.7	59.3	3.0	3.9	0.8	4.7
Grand mean	3.8	2.5	1.9	27.4 ± 2.6[d]	44.9 ± 1.8[d]	32.7	62.1	5.2	17.4	2.5	19.9

[a] Based on oven-dry weight of seed from a total of 400 plants in each severity category; figures underscored by the same line are not significantly different by the Duncan's multiple range test at P = 0.05.

[b] Values followed by the same letters in each column are not significantly different by the Duncan's multiple range test at P = 0.05.

[c] Based on 2-3, 100-plant samples in each of 4 replications.

[d] Standard error of the grand mean.

Table 4. Yield[a] of pea plants grown in 10 commercial fields affected by root rot, 1972; plants grouped in five severity categories

Field no.[b] and Cultivar	Root rot severity category[c]				
	0	1	2	3	4
1 Early Sweet	0.44	0.66	0.50	0.46	0.19
2 Early Sweet	2.62	3.68	3.31	2.61	1.33
3 Lark	0.06	0.17	0.14	0.18	0.07
4 Lark	0.12	0.28	0.21	0.37	0.26
5 Lark	0.51	0.90	0.87	0.76	0.39
6 Lark	0.67	0.57	0.60	0.48	0.34
7 Delmar	0.05	0.50	0.77	0.84	0.28
8 Delmar	0.68	0.84	1.00	0.58	0.15
9 Mars	1.35	1.25	1.37	1.14	0.52
10 Pride	1.57	1.28	1.22	1.33	0.93
Mean yield	0.81	1.01	1.00	0.88	0.45
Yield loss (%)[d]					51.00
Plants/category (%)[e]	5.40	11.30	25.90	22.10	35.30

[a] Yield of shelled green peas (g/plant), oven-dry basis.

[b] Fields 1-8 and 9-10 were in Ontario and Nova Scotia, respectively.

[c] 0 = healthy, 4 = severe root rot.

[d] Based on the combined average yield of root rot categories 0 to 3 (underscored), which were not significantly different by the Duncan's multiple range test at P = 0.05.

[e] Based on a total of 9,815 plants from 10 fields.

yield in growers' fields reported earlier (1). The actual fresh bulk yields (kg/ha (over 2 tons/acre), which was comparable to the average pea yield in growers' fields reported earlier (1). The actual fresh bulk yields (kg/ha) of the 10 pea cultivars at Ottawa were: Anoka, 3933; Charger, 6688; Dark Skin Perfection, 6550; Jade, 5139; Mars, 3679; Nugget, 5897; Trojan, 5944; Venus, 5388; Asgrow XPF 3007, 4343; and Asgrow 4683, 2802. These fresh weights were on average five times greater than the corresponding oven-dry seed weight (1 kg of freshly shelled peas yielded approximately 200 g dry matter). It is also noteworthy that the population of *Fusarium solani* in the field plots ranged from 400 to 1600 propagules per gram of soil. Similar levels of inoculum were observed in root rot affected fields elsewhere (2,8).

Samples from growers' fields

During these studies, samples were also taken from growers' fields to determine the per plant yield loss associated with different root rot categories.

In 1972 plants were placed in five root rot categories but there were no significant differences among the mean yields of categories 0 to 3, (Table 4). However, plants in the severely affected category (root rot 4) showed a significant yield loss of 51%. In these fields, healthy plants constituted only 5.4% of the total number of plants sampled; therefore the estimate of yield loss for plants in the severe category was based on the combined yield of categories 0 to 3. It was evident that securing sufficient numbers of healthy plants in some root rot affected fields posed a problem. Therefore in 1973 and 1974, yield data were obtained for equal numbers of plants in only two severity categories (root rot 0-3 and 4), constituting paired samples of apparently healthy and severely affected plants (Tables 5 and 6). These data illustrate the range of yield and corresponding yield losses for several pea cultivars grown in different regions of Canada. It should be noted that the yield of the same cultivar varied from field to field even

Table 5. Yield loss from severe root rot (category 4) based on yield of apparently healthy plants (categories 0-3) in 31 commercial pea fields, 1973

Field no.[b] and cultivar or line	Avg seed yield[a] (g/plant, oven-dry)		
	Apparently healthy (categories 0-3)	Severe root rot (category 4)	Yield loss (%) in severe category
1 Venus	1.12	0.50	55.4
2 Venus	0.80	0.53	33.8
3 Venus	0.76	0.28	63.2
4 Venus	0.68	0.36	47.1
5 A-45 (Asgrow)	0.75	0.11	85.3
6 A-45 (Asgrow)	0.81	0.24	70.4
7 A-45 (Asgrow)	0.75	0.54	28.0
8 A-45 (Asgrow)	0.77	0.44	42.9
9 Scout	0.79	0.73	7.6
10 Scout	0.76	0.62	18.4
11 Dark Skin Perfection	0.81	0.60	25.9
12 Dark Skin Perfection	0.66	0.36	45.5
13 Dark Skin Perfection	0.85	0.46	45.9
14 Early Sweet	0.91	0.07	92.3
15 Early Sweet	0.57	0.19	66.7
16 Early Sweet	0.31	0.12	61.3
17 394 (Asgrow)	0.36	0.32	11.1
18 394 (Asgrow)	0.51	0.33	35.3
19 4683 (Asgrow)	0.48	0.18	62.5
20 4683 (Asgrow)	0.65	0.17	73.8
21 4683 (Asgrow)	0.60	0.30	50.0
22 4683 (Asgrow)	0.60	0.26	56.7
23 4683 (Asgrow)	0.93	0.81	12.9
24 Lilaska	0.54	0.20	63.0
25 Lilaska	0.55	0.15	72.7
26 Early Sweet	1.19	0.46	61.3
27 Early Sweet	0.94	0.36	61.7
28 Nugget	0.64	0.44	31.3
29 Nugget	0.58	0.23	60.3
30 Pride	0.88	0.14	84.1
31 Pride	1.25	0.35	72.0
Mean yield and standard error	0.74 ± 0.04	0.35 ± 0.03	
Mean yield loss (%) calculated from mean yield difference[c]			52.7

[a] Based on avg yield of 100 plants in each of the two categories (0-3 and 4).

[b] Fields 1-13, 14-23, 24-27 and 28-31 were in British Columbia, Ontario, Quebec and Nova Scotia, respectively.

[c] Mean yield difference was significant by paired *t* test at P = 0.01.

Table 6. Yield loss from severe root rot (category 4) based on yield of apparently healthy plants (categories 0-3) in 36 commercial pea fields, 1974

Field no.[b] and cultivar or line		Avg seed yield[a] (g/plant, oven-dry)		
		Apparently healthy (categories 0-3)	Severe root rot (category 4)	Yield loss (%) in severe category
1	Dark Skin Perfection	0.71	0.53	25.4
2	Dark Skin Perfection	0.83	0.11	86.7
3	Dark Skin Perfection	0.61	0.27	55.7
4	Scout	1.35	0.28	79.3
5	Scout	1.05	0.21	80.0
6	Scout	1.46	0.10	93.2
7	Scout	1.55	0.38	75.5
8	Scout	3.30	0.28	91.5
9	Scout	1.46	0.31	78.8
10	Scout	2.53	0.62	75.5
11	Scout	1.62	0.34	79.0
12	4683 (Asgrow)	1.47	0.42	71.4
13	4683 (Asgrow)	0.72	0.13	81.9
14	4683 (Asgrow)	1.01	0.53	47.5
15	4683 (Asgrow)	0.86	0.22	74.4
16	Trumpet	1.04	0.62	40.4
17	Trumpet	0.42	0.18	57.1
18	A-45 (Asgrow)	0.72	0.31	56.9
19	A-45 (Asgrow)	1.14	0.48	57.9
20	Early Sweet	1.38	0.60	56.5
21	Early Sweet	1.07	0.43	59.8
22	Early Sweet	0.93	0.32	65.6
23	Dash	0.54	0.33	38.9
24	Lark	0.96	0.21	78.1
25	Medalis	1.02	0.44	56.9
26	Medalis	1.00	0.30	70.0
27	Early wilt resistant Perfection	1.79	0.93	48.0
28	Early wilt resistant Perfection	0.51	0.31	39.2
29	Sparkle	2.04	1.17	42.6
30	Sparkle	1.36	1.04	23.5
31	Sparkle	2.68	0.68	74.6
32	Sparkle	1.69	1.39	17.8
33	Anoka	4.89	4.29	12.3
34	Anoka	2.52	0.78	69.0
35	Anoka	3.19	0.58	81.8
36	Anoka	1.25	0.51	59.2
Mean yield and standard error		1.46 ± 0.15	0.57 ± 0.11	
Mean yield loss (%) calculated from mean yield difference[c]				60.9

[a] Based on avg yield of 100 plants in each of the two categories (0-3 and 4).

[b] Fields 1-11, 12-24, 25-26, 27-28, and 29-36 were in British Columbia, Ontario, Quebec, Nova Scotia, and Prince Edward Island, respectively.

[c] Mean yield difference was significant by paired t test at $P = 0.01$.

in the same geographic region, indicating the influence of local climatic and edaphic conditions. Also, the critical timing of assessment in relation to maturity of the crop presented some practical problems in conducting such a wide ranging survey and this may have contributed to some of the variation observed. However, yield variation expressed as standard error was less than 20% of the mean yield for each category of plants in both years, and thus the derivation of a mean yield loss value for different cultivars seems reasonable, provided we allow for about 20% error due to natural variability.

Loss factors

An average loss value of 55% was obtained for severely affected plants in the 3-year field plot experiments with the cultivar Jade. In 11 pea cultivars tested similarly in field plots the average loss for that severity category was 58.4%. Similar results were obtained in commercial fields, where the average per plant yield loss for the severely affected category was 51%, 52%, and 60.9% in 1972, 1973, and 1974, respectively. Based on the results of the experimental and field observations, a loss factor of 0.57 seems appropriate for estimating the effect of severe root rot on pea yield.

Plants classed as moderately affected by root rot (categories 1-3) showed an overall average yield loss of 34.7% as compared to the yield of healthy plants (category 0) in experimental field plots. However, in commercial fields a comparable estimate of loss for moderate levels of root rot was not obtained. In those fields very few healthy (category 0) plants appeared in the samples and they were combined with those of categories 1 to 3 in evaluating the loss due to severe disease.

On the basis of these results we propose, as a working model, the use of a loss factor of 0.57 for estimating yield loss from this disease; i.e. % yield loss % severely affected plants × 0.57. Until further information is available on the effects of moderate levels of root rot on pea yield in growers' fields, this formula should provide a useful, conservative estimate of yield loss in cultivars of green pea presently grown in Canada.

Discussion

Difficulties involved in studying yield losses from root diseases have been recognized (7) and there are few examples (6) of well defined methods for measuring disease severity, for producing controlled epidemics, and for relating severity ratings to yield loss. We have chosen what is in effect a critical point system (7) in which disease assessment is made only once, as close to harvest as possible. In this study the effects of early or late infections and of climatic or edaphic factors on pea yield were not specifically considered, and there are obvious dangers in drawing conclusions from samples collected from a wide geographical area involving different pea cultivars. For the purpose of this study it was assumed that green pea producing areas share similar cultural methods and that the pea cultivars commonly grown do not differ appreciably in symptom expression and in yield response to this disease.

In this work we have attempted to relate various levels of root rot severity to pea yield to provide a working model for estimating loss in commercial crops. Percent yield loss for the severe disease level was reasonably consistent over a 3-year period in both growers' fields and experimental plots. Although moderate levels of root rot showed a substantial yield loss (35%) in experimental plots, similar detectable loss could not be confirmed in the commercial crops sampled. Consequently a conservative estimate of yield loss due to root rot over a large area can be obtained by multiplying the percentage of severely affected plants by a factor of o.57.

The percentage of severely affected plants can be estimated by suitable sampling procedures (1) or by other methods. Since severely affected plants often appear in visually discernible patches in a field, aerial photography may be useful in determining the area of crop severely affected by the disease.

These results clearly indicate that severe levels of fusarium root rot result in a large measurable loss in yield of green peas. Reducing the incidence of severely affected plants in infected fields by the development of resistant cultivars or by chemical or cultural means, should have a significant effect in increasing yield.

Acknowledgments

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Feathery mottle virus of sweet potato in Ontario

W.G. Kemp[1] *and G.H. Collin*[2]

The occurrence of a virus similar to feathery mottle virus in certain transmission characteristics, particle morphology, and size is reported in sweet potato (*Ipomoea batata*) for the first time in Canada. The virus was found in both experimental and commercial plantings in Norfolk County, Ontario, and is widespread in the cultivars Jewel, Nemagold, Baker, Puerto Rico, and Redmar.

Can Plant Dis. Surv. 56:33-34. 1976

La fréquence d'un virus semblable à celui de la marbrure duveteuse, en ce qui concerne certaines caractéristiques de transmission, la morphologie et la taille, est signalée pour la première fois au Canada chez la patate douce (*Ipomoea batata*). Le virus a été observé dans certaines plantations expérimentales et commerciales du comté de Norfolk (Ontario) et est largement répandu dans les cultivars Jewel, Nemagold, Baker, Puerto Rico et Redmar.

Research to improve the quality and yields of sweet potato (*Ipomoea batata* (L.) Poir), a crop of limited acreage but of high dollar value per acre in Ontario, was initiated in 1972 by the Horticultural Research Institute of Ontario at Simcoe, Ontario. One of the principal problems encountered was an apparent virus disease in all the experimental plantings that often reached an incidence of 100%. The disease was equally prevalent on farms in the Simcoe region. Our concern was that material produced as selected seed roots from affected cultivars in the experimental plots and later released to the Ontario grower might further spread the disease and affect marketable yield and quality in subsequent crops. This paper briefly reports experiments conducted to determine the transmissibility, the viral nature, and the identity of the virus associated with this disease condition.

Symptoms

Primary symptoms of the disease in most of the sweet potato cultivars observed in the field consist of small faintly chlorotic spots and sometimes rings randomly scattered over the younger leaves. These spots gradually enlarge and later become diffuse. Vein-clearing and vein-banding may develop subsequently and as the affected leaves age they may become mildly chlorotic and mottled. Under summer conditions the foliage symptoms are usually masked; definite symptoms can only be distinguished on the lower, shaded leaves (Fig. 1A). The cultivar Nemagold, however, frequently exhibits purple rings on the lower, older leaves.

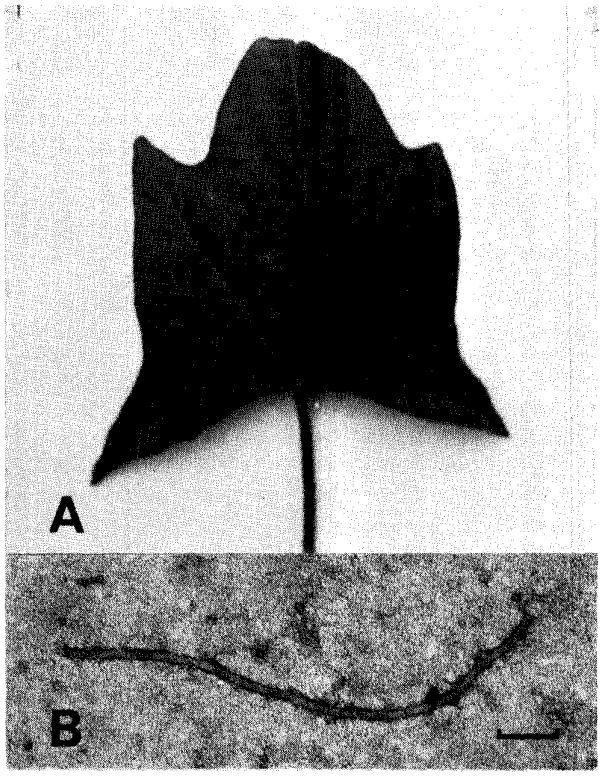

Figure 1, A) Sweet potato leaf (cv. Nemagold) naturally infected with feathery mottle virus. B) Flexuous rod-shaped virus particle (ca. 850 nm) in negatively stained leaf dip from infected Nemagold sweet potato. Bar length, 0.1 mμ.

Experimental and discussion

When inoculum was prepared with 0.1M DIECA, symptoms appeared on the first true leaves of the test plants (*Ipomoea nil* cv. Scarlet O'Hara) as early as 14 days after mechanical inoculation. In this test, all 35 inoculations resulted in visible symptoms within 4

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weeks. Moreover, symptoms induced in the Scarlet O'Hara seedlings after inoculation with sap in 0.1M DIECA from infected leaves of the sweet potato cultivars Jewel, Nemagold, Baker, Puerto Rico, Redmar, and B18 were indistinguishable from each other.

No virus-like symptoms were produced on the cultivar Scarlet O'Hara mechanically inoculated at the cotyledonary stage with inoculum prepared in tap water or in 0.1M cysteine hydrochloride at ca. 2 ml/g infected leaf. Twenty-five seedlings inoculated with sap prepared either way failed to develop symptoms after a 4–6 week period.

All 19 healthy 14-day-old Scarlet O'Hara seedlings were successfully infected when young apical shoots from *I. batata* cv. B18 and Redmar with pronounced symptoms were grafted onto their stems and bound with latex rubber strips. The incubation period of the virus in this plant varied from 14 to 28 days, with most of the plant showing definite symptoms within 18 days. The first symptoms consisted either of small yellow spots or vein-clearing on leaves that had not yet reached full size. Three of the 19 grafts failed to unite and the scions wilted and died within 3 days; however in each case the receptor seedling became infected. This suggested that contact only was required to transmit the virus from scion to stock. The symptoms induced on Scarlet O'Hara seedlings after mechanical inoculation with sap and by grafting were indistinguishable.

The possibility that the virus was transmitted by aphids was also investigated. On 3 occasions, summer populations of the aphid (*Myzus persicae* [Sulz]), collected from infected sweet potatoes in the field, failed to transmit the virus to Scarlet O'Hara when 10 aphids were caged on each healthy seedling immediately after collection. In two greenhouse experiments *M. persicae* raised on healthy cabbage seedlings were used in aphid transmission tests. The aphids were removed from the seedlings, starved by holding on moist filter paper for 3 to 4 hours, and then placed on detached leaves of infected sweet potatoes. After feeding for 1 to 2 minutes they were transferred in groups of 5 to 10 seedlings of *I. nil*. About 15 hours later, the aphids were either killed with a malathion spray or removed by hand. None of the plants exposed to feeding aphids developed visible virus symptoms. Neither was virus detected in the test plants when they were indexed by mehcanical inoculation to *I. nil* seedlings.

Attempts to determine the thermal inactivation, dilution end-point and longevity of the virus in sweet potato sap expressed in 0.1M DIECA were unsuccessful.

Crude extracts from sweet potato leaves with chlorotic spots, rings, and vein-banding symptoms were examined under a Phillips 201 electron microscope. With both the epidermal-strip and leaf dip techniques (2), involving negative staining with 2% phosphotungstate, only long, flexuous rods (ca. 850 nm) were observed (Fig. 1B). No virus-like particles were observed in preparations from symptomless, apparently healthy plants of the same cultivar.

The data presented here indicate that the disease in sweet potato in Ontario is virus induced. Similarities in symptomatology, certain transmission characteristics, and particle morphology and size strongly suggest that the chlorotic spotting, vein-banding, and, in some instances, the purple ring symptoms on sweet potato were caused by feathery mottle virus (FMV) previously described in the USA (1); however our failure to achieve virus transmission by aphids is at variance with that report. While there is no previous report that this virus occurs in Canada, our observations indicate that FMV may be widely distributed in all commercial sweet potato cultivars in Ontario. Moreover, the disease appears to be of economic importance in the successful production of this crop.

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Godronia canker of highbush blueberry restricted by suspected winter sun scald injury[1]

C. L. Lockhart and F. R. Forsyth

In 1-year-old shoots of the highbush blueberry (*Vaccinium corymbosum*) cultivar Jersey, which is highly susceptible to godronia canker [*Godronia cassandrae* f. sp. *vaccinii*], cankers and pycnidia of the pathogen developed only on areas of the stem that did not show injury from winter sun scald.

Can. Plant Dis. Surv. 56:35. 1976

Sur les pousses d'un an du cultivar Jersey du bleuet en corymbe (*Vaccinium corymbosum*), très sensible au chancre causé par *Godronia cassandrae* f. sp. *vaccinii*, le chancre et les pycnides de l'agent pathogène ne se sont développés que dans les zones de la tige non endommagées par l'insolation en hiver.

The highbush blueberry *Vaccinium corymbosum* L., cv. Jersey has been reported to be the cultivar most susceptible to stem canker caused by *Fusicoccum putrefaciens* Shear (*Topospora myrtilli* (Feltg.) Boerema), stat. perf. *Godronia cassandrae* Peck f. sp. *vaccinii* [(Groves) Boerema & Verhoeven] (2). In mid June, 1975, it was observed that canker development on this cultivar was restricted by areas of suspected winter sun scald injury and this observation is reported here.

In July suspected winter sun scald injury (1), consisting of tan to brown longitudinal areas running almost the full length of the south side of 1-year old blueberry shoots, was frequently observed on several plants of the cultivar Jersey. The planting also contained the cultivars Blueray, Bluecrop, Coville, Berkeley, and Burlington, but none of these showed winter injury.

Many of the winter sun scald injured shoots were also infected with godronia cankers. Where the cankers were adjacent to areas injured by winter sun scald, the pattern of canker development was invariably that shown in Figure 1. Cankers developed normally on healthy tissue and were restricted to these areas by the winter sun scald injured areas. In the field, no canker or *Fusicoccum* pycnidia were found on the injured areas. However, when infected stems were held in moist chambers in the laboratory, a few scattered pycnidia of *F. putrefaciens* developed on the injured areas adjacent to cankered tissues.

This interesting observation and the fact that godronia cankers did not develop in winter sun scald injured tissue may be important in studying the development of this disease.

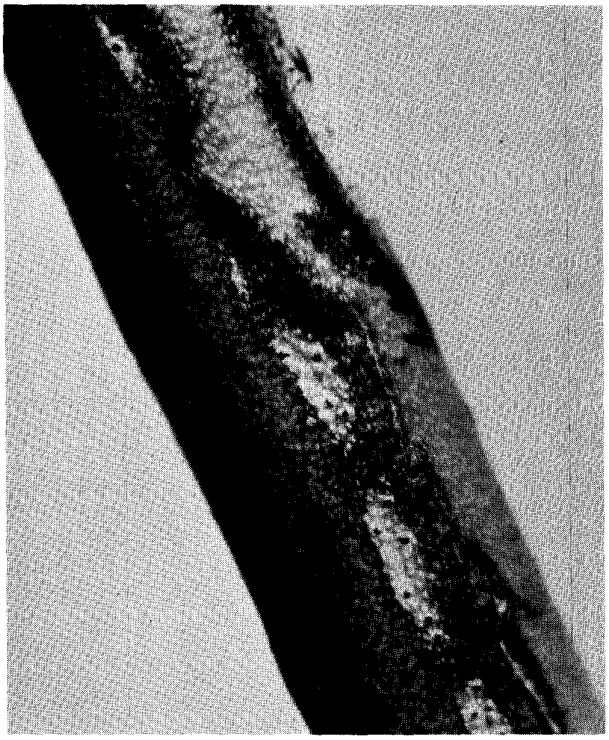

Figure 1. Godronia cankers restricted by suspected winter-injured area. The dark area on the left is the winter injury and the oblong light areas with dark curved borders on one side are cankers incited by *Godronia cassandrae*.

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Distribution, severity, and relative importance of leaf spot diseases of wheat in western Canada in 1974[1]

A. Tekauz

The leaf spot diseases spot blotch [*Bipolaris sorokiniana*], tan spot [*Drechslera tritici-repentis*], and speckled leaf blotch [*Septoria avenae* f. sp. *triticea*] caused insignificant damage to wheat (*Triticum aestivum* and *T. durum*) in western Canada in 1974, although infections were widespread. Spot blotch and tan spot occurred commonly in Manitoba and Saskatchewan but were rare in Alberta. Speckled leaf blotch, the least common disease, was found in all three provinces. Artificial inoculations indicated that spot blotch symptoms were distinguishable from those of tan spot and speckled leaf blotch, but that symptoms of the last two diseases were difficult to differentiate. Tan spot and speckled leaf blotch killed infected leaves within 21 days. The widespread distribution and potential for foliar damage of *D. tritici-repentis* indicate that it is the most important leaf spot pathogen of wheat in western Canada.

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En 1974, l'helminthosporiose [*Bipolaris sorokiniana*], la tache helminthosporienne [*Drechslera tritici-repentis*] et la tache septorienne [*Septoria avenae* f. sp. *triticea*] *ont causé des dégâts négligeables au blé dans l'ouest du Canada, même si les infections étaient largement répandues. L'helminthosporiose et la tache helminthosporienne étaient fréquentes au Manitoba et en Saskatchewan, mais ont été rarement observées en Alberta. La tache septorienne, maladie la moins courante, a été constatée dans les trois provinces. Des inoculations artificielles ont révélé que les symptômes de l'helminthosporiose se distinguaient de ceux de la tache helminthosporienne et de la tache septorienne, mais qu'il était difficile de différencier ces deux dernières maladies, lesquelles détruisent les feuilles infectées en moins de 21 jours. La large distribution de la tache helminthosporienne et les possibilités de dégâts foliaires montrent que c'est la plus importante tache des feuilles du blé dans l'ouest du Canada.*

The fungi *Bipolaris sorokiniana* (Sacc. in Sorok.) Shoem., *Drechslera tritici-repentis* (Died.) Shoem., and *Septoria avenae* Frank f. sp. *triticea* T. Johnson have been associated with the leaf spot disease complex on wheat in Manitoba and Saskatchewan in recent years (1,7,8). These organisms cause the foliar diseases spot blotch, tan spot or yellow leaf blotch, and speckled or septoria leaf blotch, respectively. In disease loss surveys in 1969, 1970, and 1971, the various diseases were recorded as a single entity, "leaf spots", because of similar field symptoms. Leaf rust caused by *Puccinia recondita* Rob. ex Desm. was not included in the leaf spot category. Yield losses from leaf spots, as a percentage of potential production, were calculated to be 3.0% in Manitoba in 1969 (7), 3.2% in Manitoba, and 4.2% in Saskatchewan in 1970 (8), and 3.5% in Manitoba in 1971 (1). These were the largest or second largest components of the total yield loss in the three years.

In Alberta in 1970, wheat leaf diseases caused by *Septoria* sp. and by *Erysiphe graminis* DC. ex Mérat were found, but losses were unimportant (9). Studies in North Dakota showed that in some years *Helminthosporium sativum* (syn. *Bipolaris sorokiniana*) and *S. avenae* f. sp. *triticea* caused severe leaf spot damage to wheat (2) and that *Pyrenophora trichostoma* (Fr.) Fckl. (perfect stage of *D. tritici-repentis*) caused the most prevalent foliar disease in the region and accounted for significant yield losses (3,4).

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Because leaf spot diseases of wheat have been prevalent and caused significant losses in recent years, this study was initiated in 1974 to assess the present importance of these diseases in western Canada and to elucidate techniques for their detection and identification.

Materials and methods

The first of two disease surveys was carried out from 27 July to 3 August 1974. It encompassed wheat growing regions in the southern and central portions of the three prairie provinces and extended from Winnipeg, Manitoba westward to Calgary, Alberta. The second survey, on 26 and 27 August extended from 30 miles north to 60 miles south of Winnipeg. Wheat fields were examined and sampled for diseases at 5-20 mile intervals along the routes.

In each field, leaf spot disease incidence and severity was observed along a 10-m diagonal transect, commencing 10 m from the edge of the road. Disease severity ratings were based on the number and size of lesions on the upper two leaves of plants using a scale with the values 0, 1, 2, 3, and 4. Qualitatively, these numbers correspond to no infection, light, moderate, heavy, and very heavy infections respectively, but they also agree closely with the quantitative values 0, 1, 5, 25, and 50% of total leaf area affected, as illustrated by James (5). In addition, the value trace (tr) was used for very light infections when the number of lesions averaged less than one per leaf on the plants examined.

Usually no attempt was made to distinguish between diseases. A total disease rating for leaf spots was recorded along with the stage of growth and the type of wheat grown (common, *Triticum aestivum* L., or durum, *T. durum* Desf.).

When leaf spot symptoms were present, a number of flag and second leaves were collected for isolation and identification of the pathogen(s). If symptoms were present on the lower leaves only, these leaves were collected for isolation of pathogenic species although the disease severity rating of the field was zero.

Fungi were isolated from lesions by placing surface sterilized sections of infected leaf tissue in petri dish moist chambers, incubating for 48 to 96 h at 20°C to induce sporulation, and transferring the conidia produced to plates of 10% V8-juice agar with a sterile needle. Identification of isolates was based on published morphological and cultural characters (6,10,11). A maximum of three separate attempts was made to isolate pathogenic species from each collection. Negative results or the presence only of known saprophytic species, resulted in a change in the leaf spot severity rating to zero.

Pathogenicity of isolated cultures was tested by inoculation of healthy wheat seedlings. Five to eight seeds of the common wheat cultivars Glenlea, Manitou, and Napayo were planted in three clumps per 15 cm clay pot. They were kept in a growth cabinet at 17 – 22°C and a 16-h photoperiod. The plants were inoculated at the three- to four-leaf stage, incubated for 24 h at 20°C and 100% R H, and checked for disease development 7 days after inoculation. Inoculum of *D. tritici-repentis* was prepared by homogenizing a 7-day-old single spore culture grown on 10% V8-juice agar with 100 ml sterile distilled water in a Waring Blendor. *Septoria avenae* f. sp. *triticea* cultures were of mass conidial origin and were grown in 40 ml potato-sucrose liquid medium for 7 days, made up to 100 ml with water and homogenized as above. One drop of Tween 20 per 50 ml of suspension was added to all inocula used. Plants were inoculated by dipping each clump of leaves into the inoculum suspension in a 250 ml graduated cylinder and swirling the contents for approximately 15 sec. Inoculum of *B. sorokiniana* was prepared by washing the conidia and conidiophores from the surface of a 7-day-old culture growing on 10% V8-juice agar, and adjusting the concentration to produce an aqueous suspension of 10^4 propagules per ml. This was sprayed on the plants at a rate of about 7 ml suspension per pot with a DeVilbis atomizer fitted to an electric pump. Symptoms were recorded 7 days after inoculation and some plants were kept for an additional 14 days to observe disease development.

Results

Manitoba, Saskatchewan, and Alberta survey

The route followed, some points of reference, and the location of the 141 wheat fields sampled for this survey are shown in Fig. 1. Plants in most fields were in flower or in the milky-to soft dough stages of growth. Seventy-nine locations (56%) were rated disease-free, that is, showed no disease on the upper two leaves. Symptoms of leaf rust, powdery mildew, or leaf spots were found in the remaining 62 (44%) fields. Leaf rust was found in trace amounts in 30 fields (21% of the total, 48% of those diseased) throughout the surveyed region. Powdery mildew was seen only in Alberta, where disease severity in the nine fields involved was in the tr–1 range with one field rated at 1–2, that is light to moderate infection. The lower leaves on most plants were more heavily infected. Leaf spots were seen at 43 (31% of total, 69% of those with disease) of the sampled locations. Typically the lesions were small (1–3 mm long), roughly oval in shape, and they ranged in color from light tan to dark brown. There was little or no chlorosis evident in most cases. In a few instances, minute pycnidia were clearly visible in the lesions, and these were rated for disease severity as *Septoria* sp. symptoms. Only trace amounts were recorded. Overall, leaf spot severity was rated as very light (tr) at all locations other than one field in west-central Saskatchewan where a light to moderate (1–2) infection was found.

Isolations made from the 43 collections with leaf spot symptoms yielded cultures of *D. tritici-repentis* from 23 (54%), *B. sorokiniana* from 16 (40%) and *S. avenae* f. sp. *triticea* from 9 (21%). The distribution is shown in Fig. 1 A, B, and C. In addition, *D. tritici-repentis* was isolated from lower leaves from two fields in south-central Saskatchewan, and *B. sorokiniana* and *S. avenae* f. sp. *triticea* from one field west of Yorkton. There was no difference in leaf spot incidence or severity on common and durum wheat. Thirty-one of the 141 fields sampled were planted to durum cultivars.

D. tritici-repentis was isolated from leaf spots in collections from all three Prairie Provinces but was found only once in Alberta. The greatest concentration of this pathogen was in central Saskatchewan from Saskatoon to the Alberta border. Eight out of nine fields sampled in this 150 mile section were infected, and one of these had the highest leaf spot severity found in the survey. *B. sorokiniana* was not found in Alberta, but was widespread in the other two provinces. *S. avenae* f. sp. *triticea* was isolated less frequently than the other two pathogens, but was found in all three provinces primarily along the northern transect of the survey route.

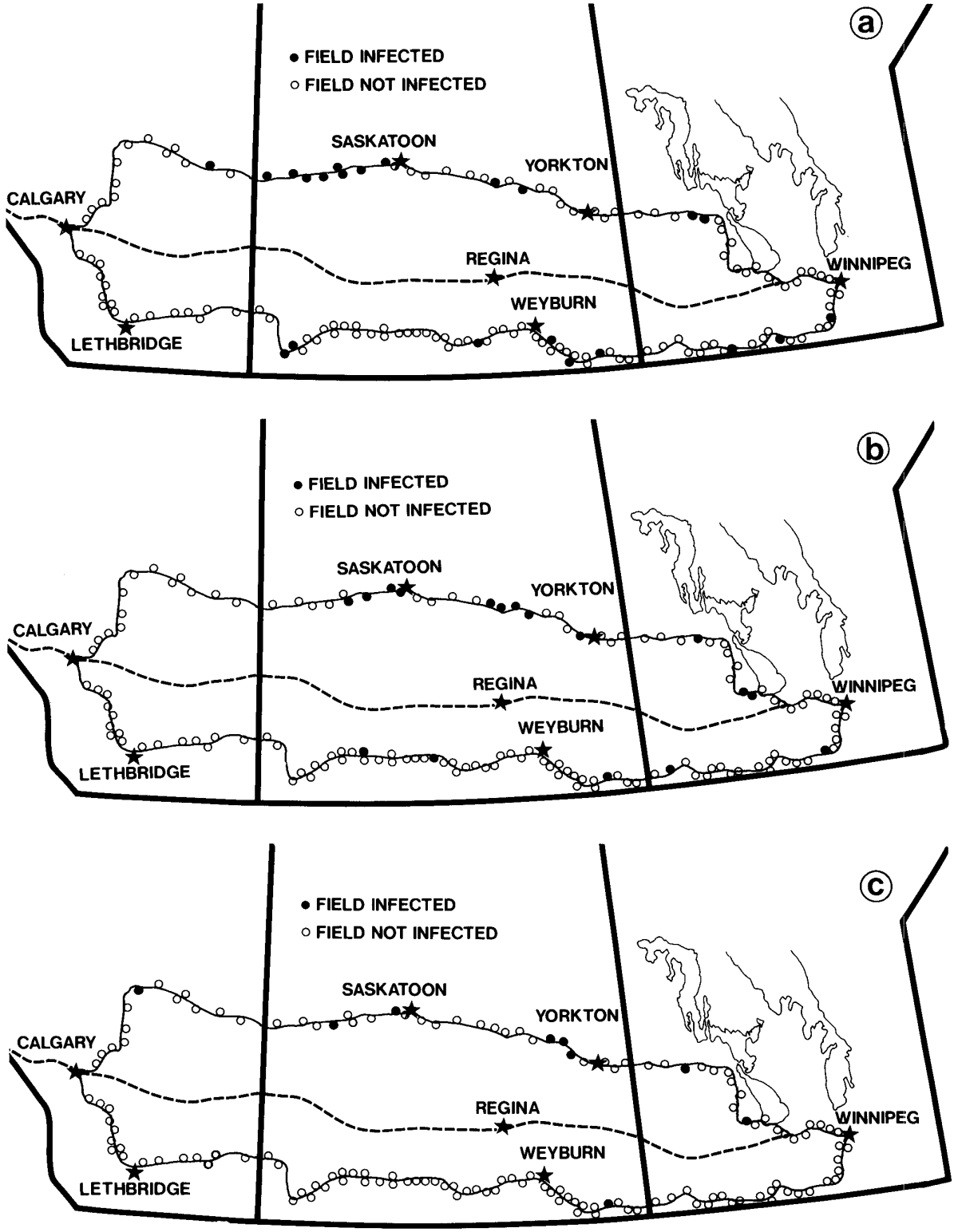

Figure 1. Distribution of wheat fields infected with A) tan spot [*Drechslera tritici-repentis*]; B) spot blotch [*Bipolaris sorokiniana*] and C) speckled leaf blotch [*Septoria avenae* f. sp. *triticea*] on the Canadian Prairies during 1974.

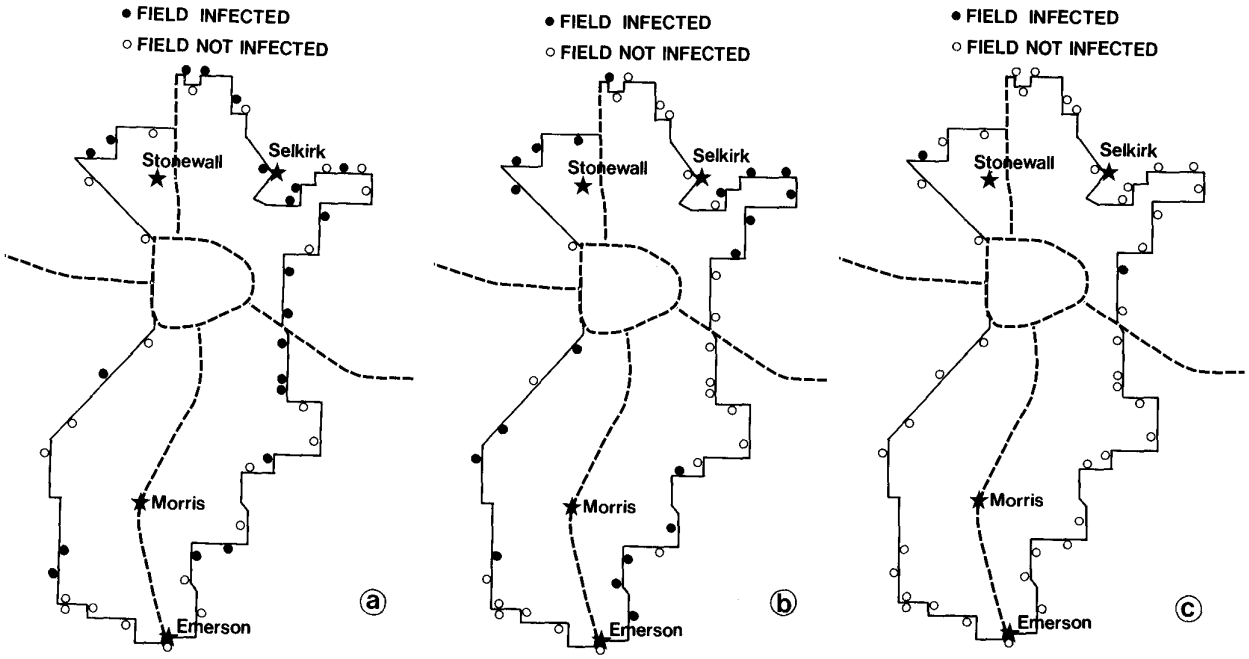

Figure 2. Distribution of wheat fields infected with A) tan spot [*Drechslera tritici-repentis*]; B) spot blotch [*Bipolaris sorokiniana*] and C) speckled leaf blotch [*Septoria avenae* f. sp. *triticea*] in the Winnipeg, Manitoba, region during 1974.

Winnipeg region survey

The locations of the 43 wheat fields sampled are shown in Fig. 2. The plants were all at the milky-to soft dough stage. Foliar diseases were found in all fields, with leaf spots present in 39 (91%) and leaf rust in 36 (84%). Leaf rust incidence was moderate at many locations and heavy at a few others. Leaf spot severity was very light to light (tr-1) in most fields, but light to moderate (1–2) in a few cases. *D. tritici-repentis* was isolated from 22 (56%) of the 39 collections showing leaf spot symptoms, *B. sorokiniana* from 25 (64%) and *S. avenae* f. sp. *triticea* from 2 (5%). The first two pathogens were found throughout the surveyed region but *Septoria* was rare (Fig. 2, A, B, C).

B. sorokiniana sporulated profusely in moist chambers within 48 h. Sporulation of *D. tritici-repentis* was variable; usually small numbers of spores were produced but in a few collections sporulation was heavy. One of the heavily sporulating isolates was from the field in Saskatchewan with the highest leaf spot severity. Sporulation on agar medium was proportionate to that on incubated leaf material. Transfer of *D. tritici-repentis* by the single spore method from either leaf lesions in moist chambers or culture plates was difficult because the conidia shrivelled quickly when the petri dish tops were lifted and the cultures exposed to ambient laboratory air. The conidia either fell from the conidiophores or became undistinguishable from them so that finding and transferring single spores was difficult, particularly from poorly sporulating cultures. Pycnidia of *S. avenae* f. sp. *triticea* were present on infected leaf material after 72 to 96 h. They were light reddish-brown, and some exuded pink-colored gelatinous droplets containing conidia of the pathogen.

Abundant symptoms were produced on wheat seedlings inoculated in the laboratory with all three pathogens. Lesion development after 7 days was similar to that found on the top leaves of mature plants in field surveys. Spot blotch lesions were small, irregularly oval, dark brown with a lighter center, and surrounded by a slight chlorotic zone (Fig. 3). Symptoms of tan spot and speckled leaf blotch were difficult to distinguish from each other. Lesions of both diseases were light tan in color, often with a darker center, irregularly oval to pointedly elongate, and often with distinct chlorotic zones extending in a longitudinal direction (Fig. 3). About 21 days after inoculation, lesions caused by *B. sorokiniana* were little changed from those described above. Their development seemed arrested, and unaffected leaf parts remained green in color. In contrast, the leaves infected with the other two pathogens became chlorotic and by 21 days after inoculation were shrivelled and necrotic. This process was often more rapid in leaves inoculated with *D. tritici-repentis*. Only a few leaves of the plants infected with *S. avenae* f. sp. *triticea* developed pycnidia after 21 days in the growth cabinet.

Discussion

In 1974 conditions for disease development were poor across the prairies for pathogens requiring frequent or prolonged periods of precipitation and high humidity. In

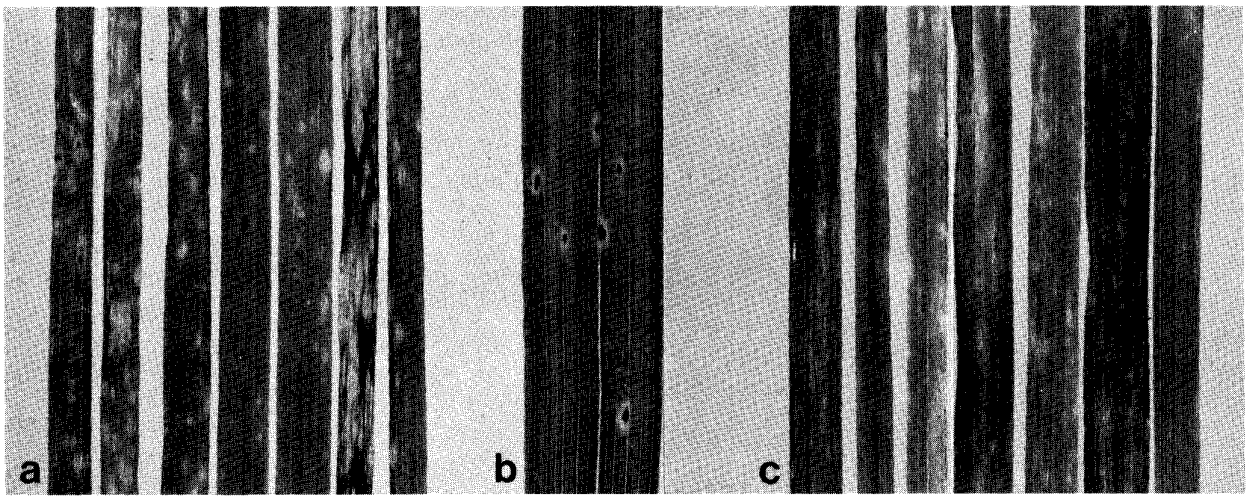

Figure 3. Symptoms produced by A) *Drechslera tritici-repentis*; B) *Bipolaris sorokiniana*; and C) *Septoria avenae* f. sp. *triticea* on wheat leaves 7 days after inoculation.

several regions, little or no rain fell for the first 2 months of the growing season and consequently disease incidence was low. During the second survey, later in the growing season, infections were more common but severity remained low. Rainfall in the Winnipeg region in the latter part of August presumably favored infection, but little disease development was evident at the time of this survey. Fungal leaf spots likely did not cause appreciable reductions in wheat yields on the prairies during 1974. It is likely that with more favorable environment conditions for disease development and/or more extensive surveys of the crop regions, particularly in Alberta, the distribution and severity of this group of pathogens would be greater than that found in 1974.

Leaf spot symptoms were readily induced on potted wheat plants using the methods of inoculation described. Inoculation by the dipping method, as carried out for *D. tritici-repentis* and *S. avenae* f. sp. *triticea*, was quite tedious but necessary because preliminary tests had shown inoculum made up primarily of mycelial fragments was reliable only if applied in this manner. Inoculum consisting largely of conidia, such as that of *B. sorokiniana*, could be applied successfully by the spraying method.

Symptoms produced by *B. sorokiniana* were distinct from those of the other two leaf spot pathogens. It is possible that familiarity with symptoms would permit field identification of spot blotch. It is unlikely, however, that field symptoms of tan spot and speckled leaf blotch could be reliably distinguished. Although the occurrence of pycnidia in tan-colored lesions would indicate infection by *Septoria*, pycnidia are not always present. Septoria lesions with pycnidia are often found in abundance very late in the growing season on senescent wheat foliage, but most develop too late to cause appreciable yield losses.

The sparse sporulation of many isolates of *D. tritici-repentis* and the difficulty at times in recognizing the conidia suggests that this pathogen may have been underestimated in the past. This study indicates that because of its widespread distribution in two of the prairie provinces in 1974 and its potential to cause severe damage to young foliage *D. tritici-repentis*, the incitant of tan spot, is the most important leaf spot pathogen of wheat in western Canada.

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