Canadian Plant Disease Survey

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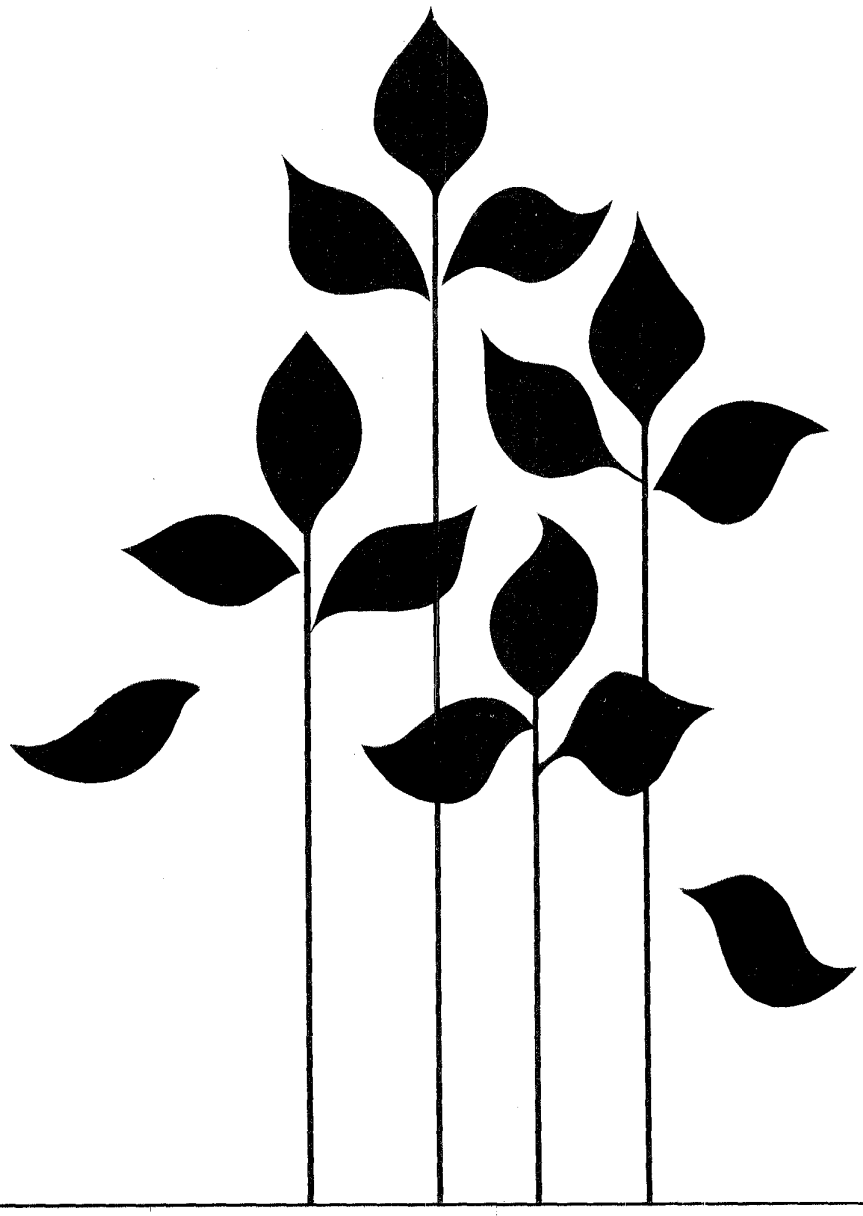

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The *Canadian Plant Disease Survey* is a periodical of information and record on the occurrence and severity of plant diseases in Canada and on the assessment of losses from disease. Other original information such as the development of methods of 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.

L'Inventaire des maladies des plantes au Canada 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 immediat pour les phytopathologistes.

Crown rust of oats in Canada in 1977

D.E. Harder[1]

Oat crown rust (*Puccinia coronata* f. sp. *avenae*) did not cause significant crop losses in western Canada in 1977. In Ontario crown rust was widespread, but there was a wide range in the severity of infection. Infections ranged from trace in some farm fields to very severe in others. The severity of infection was closely associated with the proximity to the oat field of European buckthorn (*Rhamnus cathartica* L.). Nearly all crown rust infections in Ontario appeared to originate locally from infected buckthorn. The occurrence of standard races of crown rust across Canada was determined. In eastern Canada race 210 predominated, while in western Canada races 326 and 295 predominated. Virulence combinations in the crown rust population were also determined used a set of 12 oat lines carrying single substituted genes (Pc) for crown rust resistance. The 242 isolates from eastern Canada and 190 isolates from western Canada comprised 38 and 31 virulence combinations, respectively. In eastern Canada virulence on genes Pc 35 and Pc 56 predominated, and in western Canada on genes Pc 40 and Pc 35. There has been little change from previous years in the level of virulence on the currently most resistant commercial oat cultivar Hudson, and no virulence was found on the gene combinations Pc 38-39 and Pc 55-56.

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La rouille couronnée de l'avoine (*Puccinia coronata* f. sp. *avenae*) n'a pas entraîné d'importantes pertes de rendement dans l'ouest du Canada en 1977. En Ontario, la maladie a été très répandue bien que son degré de virulence ait varié grandement allant de traces imperceptibles dans certains champs à de graves infestations dans d'autres. Le degré d'infection était étroitement lié à la présence voisine de nerprun cathartique (*Rhamnus cathartica* L.). Presque tous les cas de rouille couronnée trouvés en Ontario semblent avoir pour origine des nerpruns atteints. L'auteur a déterminé la fréquence des races courantes de rouille couronnée au Canada. Dans l'Est c'est la race 210 qui a prédominé tandis que dans l'Ouest, ce sont les races 326 et 295. Les combinaisons de virulence présentes au sein des populations de rouille ont aussi été établies à l'aide d'un ensemble de 12 lignées d'avoine portant des gènes simples substitués (Pc) pour la résistance à la rouille couronnée. Les 242 isolats trouvés dans l'est du Canada et les 190 isolats de l'Ouest comportaient respectivement 38 et 31 combinaisons de virulence. Dans l'est du pays, la virulence envers les gènes Pc 35 et Pc 56 a prédominé et dans l'Ouest, elle s'est surtout portée sur les gènes Pc 40 et Pc 35. Le degré de virulence sur Hudson, le cultivar commercial actuellement le plus résistant a très peu changé par rapport aux années précédentes et on n'a pas trouvé de signe de virulence sur les combinaisons de gènes Pc 38-39 et Pc 55-56.

Occurrence in western Canada

Oat crown rust caused by *Puccinia coronata* Cda. f. sp. *avenae* Eriks. did not cause significant damage to oat crops across western Canada in 1977. The first infections were observed by mid July, and conditions were favorable for rust development throughout most of the growing season. However, the air-borne influx of crown rust inoculum in 1977 was insufficient to generate an extensive epidemic.

Occurence in Ontario

A survey of the cereal growing areas of Ontario in late July of 1977 showed variable occurrence of crown rust. Infections ranged from trace amounts in some fields to near destruction of the oat crop in others. In some fields most of the oat plants were killed, and nearly all of the rust was in the telial stage. In Ottawa-Carleton, Lanark, Dundas, Grenville, and Frontenac counties infection was generally light to moderate, with occasional severe infections occurring where European buckthorn (*Rhamnus cathartica* L.) was found nearby. In the southern portions of Hastings, Northumberland-Durham, Ontario, and in York and Peel counties, infection was generally moderate to heavy. In some fields there was severe damage due to crown rust, despite generally dry conditions over much of this region. The heaviest infections were found near Nestleton Station, where buckthorn occurred extensively in woodlots and in hedgerows alongside farm fields. In Waterloo, Perth, Oxford, Middlesex, and Lambton counties, infection ranged from trace to moderately heavy. The trace infections occurred in relatively isolated fields where no buckthorn was found in the immediate vicinity. Moderate to moderately heavy infections occurred more generally in regions where there was a greater concentration of oat or barley-oat crops. In Peterborough county all fields examined were oat-barley mixtures, and the fields were quite isolated. Some fields had only traces of crown rust, while in others the oat component of the crop was destroyed by crown rust. The severe infections were associated with the proximity of buckthorn. A single large buckthorn shrub was sufficient to generate a moderately-severe infection in an adjacent field.

[1] *Contribution No. 837, Research Station, Agriculture Canada, Winnipeg, Manitoba, R3T 2M9*

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Table 1. Distribution of standard physiologic races of *Puccinia coronata* in Canada in 1977

Standard physiologic race	Eastern Canada		Western Canada	
	No. of isolates	% of isolates	No. of isolates	% of isolates
203	7	2.9	1	0.5
210	74	30.5	0	0.0
211	9	3.7	0	0.0
216	5	2.1	0	0.0
217	0	0.0	1	0.5
224	0	0.0	1	0.5
226	4	1.6	0	0.0
227	5	2.1	0	0.0
228	11	4.5	0	0.0
230	6	2.5	0	0.0
231	3	1.2	0	0.0
232	1	0.4	0	0.0
240	0	0.0	1	0.5
241	1	0.4	0	0.0
259	16	6.6	0	0.0
264	0	0.0	26	14.2
274	6	2.5	0	0.0
276	0	0.0	9	4.9
279	1	0.4	0	0.0
281	0	0.0	1	0.5
283	7	2.9	0	0.0
284	8	3.3	4	2.2
294	4	1.6	0	0.0
295	0	0.0	41	22.4
299	3	1.2	0	0.0
320	11	4.5	5	2.7
324	1	0.4	0	0.0
325	0	0.0	3	1.6
326	3	1.2	67	36.6
327	0	0.0	1	0.5
330	8	3.3	1	0.5
333	0	0.0	7	3.8
335	2	0.6	0	0.0
338	1	0.4	0	0.0
341	18	7.4	0	0.0
342	0	0.0	1	0.5
345	0	0.0	2	1.0
371	1	0.4	0	0.0
384	0	0.0	1	0.5
388	0	0.0	1	0.5
391	1	0.4	0	0.0
403	3	1.2	0	0.0
410	0	0.0	3	1.6
415	2	0.8	0	0.0
416	2	0.8	0	0.0
423	1	0.4	0	0.0
424	1	0.4	0	0.0
425	1	0.4	0	0.0
427	1	0.4	0	0.0
495	2	0.8	0	0.0
496	1	0.4	0	0.0
498	0	0.0	1	0.5
499	2	0.8	0	0.0
[a]1,3,4,5,6,8,9	2	0.8	0	0.0
[a]2,3,4,5,8,9,10	1	0.4	0	0.0
[a]2,3,5,6,8,10	0	0.0	2	1.0
[a]2,5,6,8,9	1	0.4	0	
[a]4,8,9,10	0	0.0	1	0.5
[a]8,10	0	0.0	1	0.5
TOTAL	243		183	

[a]Resistant standard differential varieties: 1 = Anthony, 2 = Victoria, 3 = Appler, 4 = Bond, 5 = Landhafer, 6 = Santa Fe, 8 = Trispernia, 9 = Bondvic, 10 = Saia

Table 2. Virulence combinations of *Puccinia coronata* on backcross lines of *Avena sativa* cv. Pendek containing single (Pc) genes for crown rust resistance

Virulence formula effective/ineffective Pc genes	Eastern Canada No. of isolates	Eastern Canada % of isolates	Western Canada No. of isolates	Western Canada % of isolates
35,38,39,40,45,46,47,48,50,54,55,56	48	19.8	34	17.9
38,39,40,45,46,47,48,50,54,55,56/35	41	16.9	12	6.3
35,38,39,45,46,47,48,50,54,55,56/40	7	2.9	71	37.4
35,38,39,40,46,47,48,50,54,55,56/45	12	5.0	1	0.5
35,38,39,40,45,47,48,50,54,55,56/46	1	0.4	7	3.7
35,38,39,40,45,46,47,50,54,55,56/48	1	0.4	0	0.0
35,38,39,40,45,46,47,48,54,55,56/50	11	4.5	5	2.6
35,38,39,40,45,46,47,48,50,55,56/54	1	0.4	2	1.0
35,38,39,40,45,46,47,48,50,54,55/56	22	9.1	3	1.6
38,39,45,46,47,48,50,54,55,56/35,40	3	1.2	8	4.2
38,39,40,46,47,48,50,54,55,56/35,45	5	2.1	0	0.0
38,39,40,45,46,47,48,54,55,56/35,50	6	2.5	3	1.6
38,39,40,45,46,47,48,50,54,55/35,56	24	9.9	1	0.5
35,39,40,45,46,47,48,50,54,55/38,56	0	0.0	1	0.5
35,38,39,46,47,48,50,54,55,56/40,45	8	3.3	0	0.0
35,38,39,45,47,48,50,54,55,56/40,46	0	0.0	6	3.1
35,38,39,45,46,47,48,54,55,56/40,50	2	0.8	5	2.6
35,38,39,45,46,47,48,50,55,56/40,54	0	0.0	5	2.6
35,38,39,45,46,47,48,50,54,55/40,56	3	1.2	1	0.5
35,38,39,40,47,48,50,54,55,56/45,46	1	0,4	0	0.0
35,38,39,40,46,47,48,54,55,56/45,50	2	0.8	0	0.0
35,38,39,40,46,47,48,50,54,55/45,56	6	2.5	0	0.0
35,38,39,40,45,47,50,54,55,56/46,48	0	0.0	1	0.5
35,38,39,40,45,47,48,54,55,56/46,50	0	0.0	2	1.0
35,38,39,40,45,47,48,50,55,56/46,54	1	0.4	0	0.0
35,38,39,40,45,46,47,48,55,56/50,54	0	0.0	4	2.1
35,38,39,40,45,46,47,48,54,55/50,56	6	2.5	0	0.0
35,38,39,40,45,46,47,48,50,55/54,56	1	0.4	0	0.0
39,45,46,47,48,50,54,55,56/35,38,40	0	0.0	1	0.5
39,40,45,46,47,48,50,54,55/35,38,56	1	0.4	0	0.0
38,39,46,47,48,50,54,55,56/35,40,45	1	0.4	0	0.0
38,39,45,47,48,50,54,55,56/35,40,46	1	0.4	3	1.6
38,39,45,46,47,48,54,55,56/35,40,50	1	0.4	1	0.5
38,39,45,46,47,48,50,55,56/35,40,54	1	0.4	4	2.1
38,39,45,46,47,48,50,54,55/35,40,56	3	1.2	1	0.5
38,39,40,46,47,48,50,55,56/35,45,54	1	0.4	0	0.0
38,39,40,46,47,48,50,54,55/35,45,56	6	2.5	0	0.0
38,39,40,45,47,48,50,54,55/35,46,56	1	0.4	0	0.0
38,39,40,45,46,47,48,54,55/35,50,56	6	2.5	0	0.0
38,39,40,45,46,47,48,50,55/35,54,56	1	0.4	0	0.0
35,38,39,47,48,50,54,55,56/40,45,46	2	0.8	0	0.0
35,38,39,45,47,48,54,55,56/40,46,50	0	0.0	1	0.5
35,38,39,45,47,48,50,55,56/40,46,54	0	0.0	1	0.5
35,38,39,45,46,47,48,54,55/40,50,56	2	0.8	0	0.0
35,38,39,40,47,48,50,55,56/45,46,54	0	0.0	1	0.5
35,38,39,40,46,47,48,54,55/45,50,56	1	0.4	0	0.0
38,39,45,47,48,54,55,56/35,40,46,50	0	0.0	1	0.5
38,39,45,47,48,50,54,55/35,40,46,56	0	0.0	1	0.5
38,39,45,46,47,48,54,55/35,40,50,56	0	0.0	2	1.0
38,39,40,45,47,48,54,55/35,46,50,56	0	0.0	1	0.5
38,39,47,48,50,54,55/35,40,45,46,56	1	0.4	0	0.0
38,39,47,48,54,55/35,40,45,46,50,56	1	0.4	0	0.0
TOTAL	242		190	

The distribution of crown rust in Ontario indicates that in 1977 buckthorn was responsible for most of the crown rust infection of oats. The very light infections that occurred in the absence of buckthorn suggests that there was little influx of inoculum from elsewhere. It is apparent that many growers are not aware of the association between crown rust on oats and buckthorn. It is most important that they be informed in those areas

Table 3. Distribution of virulence of isolates of *Puccinia coronata* in 1977 on the standard differential varieties, on backcross lines carrying single crown rust resistance (Pc) genes, and on Hudson. The trap nursery consisted of selected oat lines grown near Winnipeg, Manitoba

	Eastern Canada		Western Canada		Trap Nursery	
Variety or resistance gene	No. of virulent isolates	% of isolates	No. of virulent isolates	% of isolates	No. of virulent isolates	% of isolates
Anthony	78	32.1	176	96.2	76	98.7
Victoria	93	38.3	114	62.3	52	67.5
Appler	93	38.3	166	90.7	69	89.6
Bond	168	69.1	180	98.4	76	98.7
Landhafer	25	10.3	163	89.1	69	89.6
Santa Fe	14	5.8	163	89.1	69	89.6
Ukraine	240	98.8	175	95.6	74	96.1
Trispernia	0	0	44	24.0	17	22.1
Bondvic	0	0	49	26.8	17	22.1
Saia	22	9.0	14	17.6	8	10.4
Pc 35	104	43.0	40	21.0	30	35.3
Pc 38	1	0.4	2	1.1	0	0.0
Pc 39	0	0.0	0	0.0	0	0.0
Pc 40	36	14.9	114	60.0	33	38.8
Pc 45	47	19.4	2	1.1	2	2.4
Pc 46	8	3.3	28	4.7	24	28.2
Pc 47	0	0.0	0	0.0	0	0.0
Pc 48	1	0.4	1	0.5	0	0.0
Pc 50	38	15.7	25	13.2	13	15.3
Pc 54	6	2.5	17	8.9	12	14.1
Pc 55	0	0.0	0	0.0	0	0.0
Pc 56	85	35.0	11	5.8	15	17.6
Hudson	37	15.3	21	11.1		

where oats may be grown. Growers must either avoid planting oats near buckthorn or eradicate buckthorn from their farms. Buckthorn has become very widespread in Ontario, and complete eradication is not feasible. However, in Ontario severe crown rust infections were limited to fields in close proximity to buckthorn, thus removal of buckthorn from individual farm fields would be sufficient to greatly reduce damage.

Physiologic specialization

Isolates of crown rust from eastern Canada were obtained from uniform nurseries grown at McDonald College and Lennoxville in Quebec and Guelph, Ottawa, Appleton, and Sunbury, Ontario, and from farm fields in southern and eastern Ontario. In western Canada the isolates were obtained from field surveys in Manitoba and eastern Saskatchewan. Isolates were also obtained from a trap nursery consisting of selected oat lines, grown near Winnipeg, Manitoba.

In 1977 all crown rust collections were identified using the standard international differential varieties (3) and a series of backcross lines of *Avena sativa* L. cv. Pendek containing single genes (Pc) for crown rust resistance, derived from *A. sterilis* L.

From eastern Canada 243 isolates, comprising 40 races, were identified using the standard differential varieties (Table 1). There were no major changes in the main races since the previous assessment in 1975 (1). Race 210 has remained the predominating race, comprising 30.5% of the isolates. Race 341, which was not found in the previous survey (1), occurred fairly frequently at 7.4% of the isolates.

In western Canada 183 isolates comprised 25 races (Table 1). This represents relatively more races (race/isolate ratio of 0.13) as compared to the race/isolate ratio of 0.085 in 1975 (1). Since 1975 race 326 has become the predominating race, although this is probably due to a decrease in race 295, which predominated previously. Race 295 decreased from 35.5% of isolates in 1975 to 22.4% in 1977. Race 264 increased from 4.3% in 1975 to 14.2% in 1977. Races 264, 295, and 326 are quite closely related in their virulence on the standard differential varieties. Races 295 and 326 are differentiated by the variety Victoria, and the virulences of both races on this cultivar are variable, indicating considerable heterozygosity for virulence at the locus involved. Race 264 is separated from race 326 by the additional virulence of 264 on Trispernia and

Bondvic, but the reaction of these cultivars to races 295, 326, and 264 appears to involve a single locus, and the reaction is variable. The increase in virulence on Victoria, Trispernia, and Bondvic (Table 3) is reflected in the increase in prevalence of races 264 and 326. The relative frequency of virulence on the standard differential varieties of isolates obtained from the trap nursery was similar to that of isolates from the field survey (Table 3) in western Canada.

In addition to the races described in the international register of crown rust races (3) and more recent descriptions (Dr. M.D. Simons, personal communication), six new races of crown rust were found (Table 1).

Using the single Pc-gene lines of Pendek, 242 isolates comprising 38 virulence combinations were identified in eastern Canada, and 190 isolates comprising 31 virulence combinations in western Canada (Table 2). Compared to 1976 (2), there was a decrease in the number isolates avirulent on the twelve Pc-gene lines, with the largest decrease in eastern Canada. There was increased virulence on gene Pc 35 in eastern Canada, but decreased virulence on Pc 35 in western Canada. Virulence on gene Pc 40 increased across Canada, with the largest increase occurring in western Canada. There were also substantial increases in virulence on genes Pc 45, Pc 50, and Pc 56 in eastern Canada. There were no new combinations of virulence on the Pc-gene carrying lines which would be of concern to the rust resistance breeding program. At present combinations of genes Pc 38-39 and Pc 55-56 are of most interest to the breeding program at Winnipeg, and these gene combinations have remained highly effective.

The most crown rust resistant cultivar presently being grown in Canada is Hudson. Based on the number of isolates identified using the standard differentials, 15.3% and 11.1%, respectively, of isolates from eastern and western Canada were virulent on Hudson. This represents a decrease from 28% in 1975 (1) in eastern Canada and little change from 10% in 1975 in western Canada.

The distribution of standard races and virulence combinations in eastern and western Canada indicates that these regions are epidemiologically isolated from each other. In western Canada the crown rust inoculum originates from over-wintering rust in the southern United States, and arrives as showers of urediospores blown if from infected oats south of the United States-Canada boundary. The distribution in Canada indicates that these spore showers do not reach the areas surveyed in Ontario. In Ontario the primary crown rust inoculum appears to be largely derived from infected buckthorn, and this inoculum remains confined to the oat growing regions of Ontario. There is insufficient data from other regions of eastern Canada to evaluate the epidemiology of crown rust.

Acknowledgements

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

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Stem rust of wheat, barley and rye in Canada in 1977[1]

G.J. Green

Susceptible wheat varieties in experimental plots in the eastern prairies were severely infected by *Puccinia graminis tritici* in 1977 but resistant commercial varieties in farm fields were nearly free from infection. Stem rust was also prevalent on *Hordeum jubatum* in Manitoba and eastern Saskatchewan but much of this rust was *Puccinia graminis secalis*. There was little stem rust in the rust nurseries excepting those located in Manitoba and eastern Saskatchewan. Twenty-seven races of wheat stem rust were identified. Race C33 (15B-1L) declined in prevalence and race C53 (15B-1L) replaced it as the main race. Races C25 (38) and C57 (32) are moderately virulent on seedlings of the resistant commercial varieties Neepawa and Sinton, but they were not found in farm fields of these varieties. There was a marked increase in the prevalence of races virulent on resistance gene *Sr 17*.

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Dans les provinces de l'est, les variétés de blé sensibles ont été gravement atteintes de *Puccinia graminis tritici* en parcelles expérimentales en 1977, mais en culture commerciale les variétés résistantes ont été pratiquement indemnes. La rouille de la tige a aussi attaqué *Hordeum jubatum* au Manitoba et dans l'est de la Saskatchewan, mais en général elle appartenait au type *Puccinia graminis secalis*. Il y eu peu de rouille de la tige dans les pépinières d'observation sauf dans celles du Manitoba et de l'est de la Saskatchewan. Vingt-sept races de rouille de la tige du blé ont été identifiées. La race C33 (15B-1L) a perdu de l'importance et a cédé la première place à la race C53 (15B-1L). Les races C25 (38) et C57 (32) se sont montrées moyennement virulentes sur les plantules des variétés commerciales résistantes Neepawa et Sinton, mais en conditions de culture ordinaire à la ferme, on ne les a pas retrouvées. Il y en une nette recrudescence de la fréquence des races virulentes sur les lignées portant le gène de résistance *Sr17*.

Prevalence and importance in western Canada

Early in the spring of 1977 infections of wheat stem rust (*Puccinia graminis* Pers. f. sp. *tritici* Eriks. and E. Henn.) were heavy in the south-eastern United States but the rust developed slowly and infections in central and northern United States were light.

Urediospores produced in the south were carried into western Canada in early June. Stem rust was first observed on a susceptible wheat variety in experimental plots at Morden, Manitoba, on July 4. It developed rapidly on susceptible varieties and on August 8 the susceptible variety Klein Titan was severely infected (80%) at Brandon, Manitoba, and the plot was killed by stem rust as it turned color. On the same day at Indian Head, Saskatchewan, there was a 30% infection in a late plot of Klein Titan that, apparently, would become severely infected before maturity. There was abundant stem rust development on wild barley, *Hordeum jubatum* L., throughout Manitoba and in eastern Saskatchewan.

Despite favorable conditions for stem rust development resistant commercial varieties of wheat and barley were virtually free from stem rust.

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

Rust nurseries consisting of 15 varieties of wheat, 3 varieties of barley, one variety of rye, and one variety of triticale were planted by cooperators at 28 locations across Canada. The varieties grown (Tables 1 and 2) have been described in previous reports in this series. The cooperators harvested samples from the plots and sent them to the Winnipeg Research Station where rust assessments were made.

Wheat stem rust was present in nurseries from Creston, B.C., to La Pocatière, Que., but heavy infections occurred only on the susceptible variety Red Bobs at locations in the eastern prairies (Table 1). Most rust occurred at Brandon where Red Bobs was heavily infected and Lee, Frontana, Thatcher[6] X Transfer, and Mindum had light infections. The resistant commercial varieties Neepawa, Napayo, Sinton, Glenlea, Wascana, Macoun, and Wakooma showed only traces of rust at a few locations.

Stem rust occurred on barley and rye at 7 scattered locations (Table 2). Infections were light except at Glenlea, Manitoba, where a moderate infection on Montcalm barley was, apparently, caused by wheat stem rust, and at Guelph, Ontario, where Prolific rye was heavily infected. The triticale variety Rosner was nearly free from infection at all locations.

Physiologic specialization

Physiologic races were identified using previously described methods and materials (1). During the 1977 survey some differentials were replaced by lines of wheat carrying the required resistance gene in a more

[1] *Contribution No. 839, Research Station, Agriculture Canada, Winnipeg, Manitoba, R3T 2M9.*

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Table 1. Percent infection of stem rust (*Puccinia graminis* f. sp. *tritici*) on 15 wheat varieties in uniform rust nurseries at 11 locations* in Canada in 1977

	Common Wheat											Durum Wheat			
Location	Red Bobs	Lee	Pitic 62	Neepawa	Napayo	Sinton	Kenya Farmer	Glenlea	Exchange	Frontana	Thatcher^{6} × Transfer	Mindum	Wascana	Macoun	Wakooma
Creston, B.C.	tr**	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Indian Head, Sask.	60	10	tr	tr	tr	tr	tr	tr	tr	1	0	0	0	0	0
Durban, Man.	tr	tr	0	0	0	0	0	0	0	0	0	0	0	0	0
Brandon, Man.	60	5	1	tr	tr	tr	tr	1	tr	20	20	30	tr	tr	tr
Morden, Man.	70	tr	0	0	0	0	0	0	0	0	0	0	0	0	0
Glenlea, Man.	60	tr	tr	tr	tr	tr	tr	0	0	0	0	1	0	0	0
Thunder Bay, Ont.	5	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New Liskeard, Ont.	tr	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Appleton, Ont.	tr	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Normandin, Que.	tr	0	0	0	0	0	0	0	0	0	0	0	0	0	0
La Pocatiere, Que.	10	1	0	0	0	0	0	0	0	0	0	0	0	0	0

*No rust was observed in nurseries at 17 locations: Agassiz, B.C.; Beaverlodge, Lacombe, Edmonton and Lethbridge, Alta.; Scott and Melfort, Sask.; Guelph, Vineland, Sunbury, Ottawa and Kapuskasing, Ont.; Macdonald College and Quebec, Que.; Fredericton, N.B.; Kentville and Truro, N.S.
**tr trace

Table 2. Percent infection of stem rust (*Puccinia graminis*) on 3 varieties of barley, one variety of rye, and one variety of triticale in uniform rust nurseries at 7 locations* in Canada in 1977

	Barley			Rye	Triticale
Location	Montcalm	Conquest	Wpg-702-M7118-13	Prolific	Rosner
Creston, B.C.	tr**	0	5	tr	0
Brandon, Man.	10	0	0	tr	tr
Morden, Man.	tr	0	tr	5	0
Glenlea, Man.	30	0	0	tr	tr
Guelph, Ont.	0	tr	tr	80	0
Sunbury, Ont.	tr	0	0	10	0
Appleton, Ont.	tr	tr	0	20	0

*No rust was observed in nurseries at 20 locations: Agassiz, B.C.; Beaverlodge, Lacombe, Edmonton and Lethbridge, Alta.; Scott, Melfort and Indian Head, Sask.; Durban, Man.; Thunder Bay, New Liskeard, Vineland, Ottawa and Kapuskasing, Ont.; Macdonald College, Normandin, Quebec and La Pocatiere, Que.; Fredericton, N.B.; Kentville, N.S.
** tr = trace

susceptible background. The parentage and source of the new differentials are:

Sr 9b - PRELUDE*4/2/MARQUIS*6/K117A
P.L. Dyck, Agriculture Canada
Research Station, Winnipeg.

Sr 10 - MARQUIS*4/EGYPT NA95/2/W2691
R.A. McIntosh, University of
Sydney, Australia.

Sr 13 - PRELUDE*4/2/MARQUIS*6/KHAPSTEIN
P.L. Dyck, Agriculture Canada
Research Station, Winnipeg.

Table 3. Distribution by provinces of physiologic races of *Puccinia graminis* f. sp. *tritici* on wheat, barley, and grasses, and of *Puccinia graminis* f. sp. *secalis* on barley and grasses in 1977.

Virulence formula and (race number)	Virulence formula [1] (effective/ineffective host genes)	Number of isolates from:					Total number of isolates	Percent of total isolates
		P.Q.	Ont.	Man.	Sask.	B.C.		
C1 (17)	5,6,7a,9a,9b,9d,9e,10,11*,13,17,Tt2/8,14,15,Tt1			1			1	.2
C10 (15B-1)	6,7a,8/5,9a,9b,9d,9e,10,11,13,14,15,17,Tt1,Tt2			4			4	.6
C15 (11-32)	6,7a,9d,9e,10,13,17,Tt2/5,8,9a,9b,11,14,15,Tt1				1		1	.2
C16 (39)	6,7a,8,9e,11,Tt2/5,9a,9b,10,13,14,15,17,Tt1			1			1	.2
C17 (56)	6,8,9a,9b,9d,9e,11,13,17,Tt1,Tt2/5,7a,10,14,15	1					1	.2
C18 (15B-1L)	6,8,9a,9b,13,15,17,Tt2/5,7a,9d,9e,10,11,14,Tt1			4			4	.6
C25 (38)	9a,9d,9e,Tt1,Tt2/5,6,7a,8,9b,10,11,13,14,15,17	1		76	50	2	129	19.7
C33 (15B-1L)	6,9a,9b,13,15,17,Tt2/5,7a,8,9d,9e,10,11,14,Tt1	1		126	48		175	26.8
C33 (15B-1L)	6,9a,9b,13,15,17,Tt2/5,7a,8,9d,9e,10,11*,14,Tt1			5	4		9	1.3
C35 (32-113)	9d,9e,10,11,13,17,Tt2/5,6,7a,8,9a,9b,14,15,Tt1			1			1	.2
C40 (32-113)	6,9d,9e,10,13,17,Tt2/5,7a,8,9a,9b,11,14,15,Tt1			1			1	.2
C41 (32-113)	9d,9e,10,13,17,Tt2/5,6,7a,8,9a,9b,11,14,15,Tt1			4			4	.6
C43 (32)	6,7a,8,9d,9e,11,Tt2/5,9a,9b,10,13,14,15,17,Tt1	1		9			10	1.5
C44 (15B-1L)	6,9a,9b,13,17,Tt2/5,7a,8,9d,9e,10,11,14,15,Tt1			1			1	.2
C46 (15B-1L)	6,8,9a,9b,13,15,Tt2/5,7a,9d,9e,10,11,14,17,Tt1			1			1	.2
C49 (15)	6,9a,9b,11,13,15,17,Tt2/5,7a,8,9d,9e,10,14,Tt1	1	1	33	26		61	9.3
C53 (15B-1L)	6,9a,9b,13,15,Tt2/5,7a,8,9d,9e,10,11,14,17,Tt1	1		158	38		197	30.1
C53 (15B-1L)	6,9a,9b,13,15,Tt2/5,7a,8,9d,9e,10,11*,14,17,Tt1			6			6	.9
C56 (38-151)	6,7a,8,9b,9d,9e,10,11,Tt2/5,9a,13,14,15,17			1			1	.2
C57 (32)	9a,9d,9e,Tt1,Tt2/5,6,7a,8,9b*,10,11,13*,14,15,17			12	1		13	1.9
C58 (29)	5,9a,9b,9d,9e,11,13,15,Tt1,Tt2/6,7a,8,10,14,17			1			1	.2
C61 (38)	6,7a,9b,9d,9e,10,11,13,Tt2/5,8,9a,14,15,17,Tt1			6	3		9	1.3
C62 (39)	6,8,9b,9d,9e,11,13,Tt1,Tt2/5,7a,9a,10,14,15,17					2	2	.3
C63 (32-113)	7a,9d,9e,10,11,13,17,Tt2/5,6,8,9a,9b,14,15,Tt1			1	1		2	.3
C66 (15)	6,9a,9b,11,13,15,Tt2/5,7a,8,9d,9e,10,14,17,Tt1			12	4		16	2.4
C71 (172)	6,9a,9b,9d,9e,10,11,13,Tt1,Tt2/5,7a,8,14,15,17			1			1	.2
C72 (39)	6,9a,9b,9d,9e,11,13,Tt1,Tt2/5,7a,8,10,14,15,17					1	1	.2
Total wheat stem rust isolates		6	1	465	176	5	653	100
Rye stem rust isolates			2	177	113	1	293	

* Intermediate infection type
[1]/ All races were avirulent on resistance genes *Sr22, Sr24, Sr26, Sr27, Sr29,* and *Sr30.*

Sr 14 - W2691*2/KHAPSTEIN
R.A. McIntosh, University of Sydney, Australia.

Sr 15 - PRELUDE*2/NORKA
P.L. Dyck, Agriculture Canada Research Station, Winnipeg.

Sr 17 - PRELUDE/8*MARQUIS*2/2/ESP518
P.L. Dyck, Agriculture Canada Research Station, Winnipeg.

Sr Tt1 - PRELUDE*4/MHLII.64.62.1.3.18
P.L. Dyck, Agriculture Canada Research Station, Winnipeg.

Three lines with resistance genes *Sr 27, Sr 29* and *Sr 30* were used as differentials for the first time. The parentage and source of each line is:

Sr 27 - WHEAT-RYE-TRANSLOCATION-238-5
University of Minnesota

Sr 29 - PRELUDE/8*MARQUIS/2/ETOILE DE CHOISI
P.L. Dyck, Agriculture Canada, Research Station, Winnipeg.

Sr 30 - WEBSTER
P.L. Dyck, Agriculture Canada, Research Station, Winnipeg

A relatively large number of isolates (653) was identified but most of them were from Manitoba and Saskatchewan. Many of the collections from these two provinces (227 from Manitoba and 136 from Saskatchewan) were obtained from plots of the susceptible variety Klein Titan at Morden, Portage, and Brandon, Manitoba and at Indian Head and Regina, Saskatchewan. The other collections were mainly from wild barley. A few were from susceptible varieties in experimental plots. The absence of collections from Alberta confirms rust nursery results indicating that stem rust was scarce on the western prairies.

Table 4. Percent of total isolates and races avirulent on single identified resistance genes in 1977 and 1976

Resistance gene	Avirulent isolates % 1977 (1976)	Avirulent races % 1977 (1976)
Sr5	0.4 (0)	8 (0)
Sr6	76.9 (94.7)	76 (74)
Sr7a	4.1 (0.2)	24 (4)
Sr8	3.6 (4.1)	28 (30)
Sr9a	94.8 (94.4)	64 (65)
Sr9b	74.8 (93.4)	68 (52)
Sr9d	27.4 (5.2)	60 (26)
Sr9e	27.4 (6.3)	60 (52)
Sr10	3.2 (4.5)	32 (17)
Sr11	16.5 (18.3)	52 (48)
Sr13	75.9 (98.2)	80 (61)
Sr14	0 (0)	0 (0)
Sr15	72.0 (93.0)	36 (40)
Sr17	40.0 (79.7)	44 (57)
SrTt1	22.9 (2.3)	28 (35)
SrTt2	99.4	96

Twenty-seven races were identified in 1977 (Table 3) including two new virulence combinations (C71 and C72). This is the fourth consecutive year that the stem rust population of western Canada has shown broad variability.

The trends in race prevalence noted in 1976 were continued in 1977. Race C33 (15B-1L) was displaced as the predominant race for the first time since 1970. It comprised 28.2% of the isolates and was displaced by race C53 (15B-1L) (31.1% of the isolates). Race C25 (38) increased to 19.8% of the isolates and race C49 comprised 9.3%. Other less prevalent but not uncommon races were C43 (32), C57 (32), C61 (38) and C66 (38) (Table 3). Race C53 resembles race C33 except that it is virulent on *Sr 17*, and race C49 resembles race C33 except that it is avirulent on *Sr 11*. The three races appear to be equally aggressive. Many collections contained both race C33 and race C53, and race C49 frequently occurred with them. The similarities of the three races and their chronological relationships suggest that races C53 and C49 are mutants of race C33. Race C25 is a different type of race as indicated by its formula and by its avirulence on resistance gene *Sr 7b* that is carried by Marquis. However, except for avirulence on gene *Sr 7b* it closely resembles race C57. They appear to be related and they are the most threatening of the races found in 1977. Both are moderately virulent on Manitou, Neepawa and Sinton. They were not found in farm fields of these varieties in 1977 nor did they attack adult plants of these varieties vigorously in a preliminary greenhouse test.

One of the most important results of the 1977 survey was the finding that the number and prevalence of races virulent on *Sr 17* had increased (Table 4). Canadian commercial varieties do not depend on *Sr 17* for their resistance and they are resistant to races virulent on *Sr 17*.

The use of plots of Klein Titan to trap wheat stem rust was a good method for determining the prevalence of the main races. However, four races were isolated from Klein Titan that were not isolated from wild barley and 9 races not found on Klein Titan were isolated from wild barley. These results are consistent with those of the past two years. They show that collections from plots of Klein Titan reliably indicate the main races present and their prevalence, but they do not reveal all of the minor races.

In total there were less than half as many rye stem rust isolates as wheat stem rust isolates, but when the collections from the wheat variety Klein Titan are ignored, there were 240 wheat stem rust isolates and 293 rye stem rust isolates. Many collections from *H. jubatum* contained both wheat stem rust and rye stem rust. Collections from *H. jubatum* made in October in central Saskatchewan were mostly rye stem rust. The ratio of rye stem rust to wheat stem rust in collections from wild barley was greater in 1977 than in 1976.

Evidently there are many constantly changing minor races in the stem rust population and many of them are never detected in the race survey. Fourteen of the races identified in 1977 were not found in 1976 and 7 of the races found in 1976 were not identified in 1977. It is uncertain whether such changes result from genetic variability or from inadequate survey methods, but it seems reasonable to believe that these minor races lack aggressiveness and do not threaten commercial varieties, although they may carry dangerous virulence combinations.

After several years of stability, the virulence of the population on some identified resistance genes changed in 1977. Although the proportion of races avirulent on resistance gene *Sr 6* was about the same, the prevalence of virulent races increased by about 18% (Table 4). The

Table 5. Adult plant reaction of five wheat varieties to six stem rust races collected in 1977

Race	Variety				
	Marquis	Sinton	Prelude-*Sr6*	Neepawa	Manitou
C25 (38)	MR*	MR	S	R	MS
C41 (32)	S	M	MR	R	MR
C43 (32)	S	R	MS	MR	R
C53 (15B-1L)	S	R	R	R	R
C57 (32)	S	R	S	MR	MR
C66 (15)	S	R	R	R	R

* S - susceptible; MS - moderately susceptible; M - mesothetic; MR - moderately resistant; R - resistant

precentage of races and isolates avirulent on genes *Sr 9d* and *Sr 9e* increased sharply. The greatest change was a 40% increase in the number of isolates and a 13% increase in the number of races virulent on resistance gene *Sr 17*.

A group of highly resistant varieties, essentially the same as those used in 1975 (2), were inoculated with composite collections of urediospores from each 1977 isolate. Varieties that were resistant to all bulked collections were: C.I. 8154 X Frocor[2], Waldron, Agatha, Tama, Romany, Esp 518/9, R.L. 5405, N.D. 499, N.D. 506, St 464, Coulter, Hercules, Wascana, Wakooma, and Macoun. The varieties Norquay and Glenlea were resistant to most bulk collections but occasionally they had type 3 infections that were presumed to result from high temperature. Varieties that had infections ranging from flecks to type 4 were: Mida-McMurachy-Exchange II-47-26, Frontana-K58-Newthatch II-50-17, Kenya Farmer, R.L. 4314, Chris, Era, and Bonny. Frontana-K58-Newthatch II-50-17, Era, and Bonny were resistant to all bulk collections in 1976. Isolates from the type 4 infections on varieties in the last group were identified as race C25 (38). Evidently this race is one of the most threatening of those found in Canada in 1977.

Adult plant reactions

The adult plant reactions of the widely grown commercial varieties Sinton, Neepawa, and Manitou and the check varieties Marquis and Prelude-*Sr 6* to races C25 (38), C41 (32), C43 (32), C53 (15B-1L), C57 (32) and C66 (15) were tested in a preliminary greenhouse trial.

The results (Table 5) indicate that Neepawa and Sinton are fairly resistant to the cultures used in the test. Manitou is resistant to all races except C25 (38) and this race is not fully virulent on it. The results indicate that the races used in the test do not seriously threaten the most widely grown Canadian stem rust resistant varieties. However, both adult plant and seedling reactions suggest that races such as C25 (38), C41 (32), and C57 (32) would not have to change much to attain virulence on one or more of these varieties.

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Air borne rust inoculum over western Canada in 1977[1]

G.J. Green

Early in the 1977 growing season southerly winds brought the usual number of stem rust urediospores into western Canada. Stem rust spore counts increased rapidly later in the season and greatly exceeded the 11-year mean. Many more leaf rust urediospores than usual were carried into western Canada early in the season but leaf rust spore counts increased slowly later in the season, and the mean number of spores present was less than the 11-year mean.

Can. Plant Dis. Surv. 58:3, 49-50. 1978

Au début de la saison de végétation de 1977, les vents du sud ont amené dans l'ouest du Canada le nombre habituel d'urédospores de la rouille de la tige. Les numérations des spores ont augmenté rapidement par la suite et ont dépassé de beaucoup la moyenne de 11 ans. Par ailleurs l'afflux d'urédospores de la rouille de la feuille en début de saison a été beaucoup plus important que d'habitude dans l'ouest du Canada, mais les numérations de spores n'ont augmenté que lentement par après, si bien que le nombre moyen de spores a été inférieur à la moyenne de 11 ans.

The relative numbers of air-borne urediospores over western Canada in 1977 were estimated by the methods described in earlier reports published annually in the Canadian Plant Disease Survey (1) except that in 1977 the spore trap slides were coated with silicone oil instead of vaseline.

The number of stem rust spores carried into western Canada from the south during June (Table 1) was about the same as in 1976 but there were many more leaf rust spores than in 1976 (1). As the season progressed the rate of increase in the numbers of leaf rust spores was slower than usual and the total numbers of spores on the slides at most locations were similar to 1976 (Table 1). In contrast, the numbers of stem rust spores increased rapidly and greatly exceeded the numbers reported for 1976. The mean number of leaf rust spores observed per slide (48-hour exposure) was much less than the 11-year mean (1966-76) but the mean number of stem rust spores greatly exceeded the 11-year mean (Table 2), especially over the eastern prairies.

The failure of leaf rust spore counts to increase at the usual rate in 1977 was probably caused by unfavorable weather conditions, especially by hot weather early in the growing season that restricted leaf rust development in farm fields. Stem rust was scarce in farm wheat fields but it was abundant in oat fields. The relatively large numbers of stem rust spores observed in 1977 probably originated in heavily infected oat fields.

Table 1. Number of urediospores of stem rust and leaf rust per square inch (6.5 cm^2) observed on silicone oil-coated slides exposed for 72-hour periods at three locations in Manitoba and three locations in Saskatchewan in 1977

	Winnipeg		Morden		Brandon		Indian Head		Regina		Saskatoon	
Date	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust
June 1-4	0	0	0	2	0	1	0	1	0	3	0	1
June 4-7	0	0	0	0	0	1	0	0	0	1	1	6
June 7-10	0	1	1	4	0	2	2	5	0	7	0	2
June 10-13	0	3	0	3	1	5	0	4	0	4	0	11
June 13-16	0	1	1	2	0	4	0	0	0	4	0	0
June 16-19	0	4	0	8	0	0	0	14	2	10	0	4
June 19-22	0	1	1	5	0	7	1	5	1	13	1	23
June 22-25	0	5	1	11	1	8		0	1	18	0	13
June 25-28	0	9	1	8	1	8	0	2	1	7	0	5

(continued)

[1] *Contribution No. 840, Research Station, Agriculture Canada, 195 Dafoe Road, Winnipeg, Manitoba, R3T 2M9*

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Table 1. Number of urediospores of stem rust and leaf rust per square inch (6.5 cm^2) observed on silicone oil-coated slides exposed for 72-hour periods at three locations in Manitoba and three locations in Saskatchewan in 1977 (concluded)

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
June 28-1	2	6	0	5	1	12	0	0	3	20	0	21
June total	2	30	5	48	4	48	3	31	8	87	2	86
July 1-4	1	5	8	15	2	6	2	11	2	33	1	23
July 4-7	0	4	2	14	0	8	0	8	1	15	0	23
July 7-10	2	6	6	33	0	0	0	1	2	14	7	63
July 10-13	7	21	4	15	0	8	0	3	0	3	4	34
July 13-16	13	28	45	125	0	11	8	38	1	9	0	33
July 16-19	54	138	56	180	4	15	2	7	8	110	2	84
July 19-22	12	73	40	145	10	43	3	45	5	113	2	30
July 22-25	67	145	38	139	14	84	2	57	1	221	7	26
July 25-28	53	122	115	100	19	27	12	46	9	186	2	7
July 28-31	122	81	100	115	3	2	1	1	23	470	5	141
July total	331	623	414	881	52	204	30	217	52	1174	30	464
Aug. 31-3	184	87	29	33	11	10	3	7	7	253	15	37
Aug. 3-6	122	18	224	156	69	131	0	0	19	162	0	3
Aug. 6-9	181	46	155	108	133	163	22	54	43	487	0	0
Aug. 9-12	394	142	359	144	120	171	131	159	21	691	0	11
Aug. 12-15	276	104	251	61	138	79	64	99	40	627	5	49
Aug. 15-18	249	117	190	58			53	120	50	263	2	28
Aug. 18-21	308	121	169	56	42	16	56	98	36	352	45	604
Aug. total	1714	635	1377	616	513	570	329	537	216	2835	67	732
1977 Total	2047	1288	1796	1545	569	822	362	785	276	4096	99	1282
1976 Total	50	1056	95	1124	35	888	19	774	39	2174	157	4284

Table 2. Mean number of urediospores of stem rust and leaf rust observed on slides exposed for 48-hour periods at six locations in western Canada from July 1 to August 15 in the years 1966 to 1976 and in 1977

Location	Stem rust		Leaf rust	
	1966-76 mean	1977 mean	1966-76 mean	1977 mean
Winnipeg	16.6	66.1	170.5	45.3
Morden	14.7	63.6	242.4	61.5
Brandon	4.0	23.2	127.6	33.7
Indian Head	4.2	11.1	294.2	23.8
Regina	5.1	8.1	861.5	150.8
Saskatoon	3.6	2.2	232.5	25.1

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Stem rust of oats in Canada in 1977[1]

J.W. Martens

Stem rust (*Puccinia graminis* f. sp. *avenae*) was first found on oats (*Avena sativa*) in Manitoba on July 11. Light infections were observed in Manitoba and eastern Saskatchewan by late July and these developed explosively, resulting in very heavy infections and the most severe crop losses in several decades. Race C10 (U.S. race 31) was the predominant race in both eastern and western Canada comprising 89% of the 319 isolates identified. Virulence on the differential host Rodney 0^2 x C.I. 9139 was observed for the first time in isolates of both races C10 and C23 (U.S. race 61).

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C'est le 11 juillet que sont apparus les premiers symptômes d'infection de l'avoine par la rouille de la tige (*Puccinia graminis* f. sp. *avenae*) au Manitoba. De faibles infections ont été observées vers la fin de juillet dans cette province et dans l'est de la Saskatchewan; puis l'infestation a pris une tournure explosive et a entraîné les pertes culturales les plus importantes qu'on ait subies depuis plusieurs décennies. La race C10 (race 31 aux États-Unis) a été le pathotype dominant dans l'est et dans l'ouest du Canada comptant pour 89% des 319 isolats identifiés. Cette année pour la première fois, des isolats des deux races C10 et C23 (race 61 aux États-Unis) ont manifesté de la virulence sur l'hôte différentiel Rodney 0^2 x C.I. 9139.

Prevalence and crop losses in western Canada

Stem rust of oats (*Avena sativa* L.) caused by *Puccinia graminis* Pers. f. sp. *avenae* Eriks. and E. Henn. was first observed in southern Manitoba on July 11, 1977. Light infections were observed in Manitoba and eastern Saskatchewan by late July and favourable climatic conditions resulted in explosive disease development, very heavy infections and the most severe crop losses in several decades. About one-third of the oat crop in the rust area escaped significant damage due to early planting. The estimated losses for the remainder of the crop ranged from 5% to nearly 100% and averaged about 35% for a total loss of 385,000 tonnes.

The commonly grown cultivars Harmon, Kelsey, Random, Rodney and Terra are susceptible to stem rust, but Hudson, which comprised 12.5% and 4.2% of the hectarage in Manitoba and Saskatchewan, respectively, is moderately resistant to this disease.

Physiologic specialization

Rust isolates obtained from wild oats (*A. fatua* L.), commercial oats and rust nurseries grown across Canada were established on the susceptible cultivar Victory and virulence combinations were determined by the infection types produced on seedlings of "Rodney 0" single-gene backcross lines (Table 1). The oat line Rodney 0^2 x C.I. 9139 (Pg X), an undetermined genotype thought to have Pg 12 plus one or more other resistance genes, was used as a supplementary differential. One field culture (race C10) from Saskatchewan was virulent on Pg (X). Races C10 (U.S. 31) and C23 (U.S. 61) continued to predominate in western Canada and comprised about 90% and 9% of all isolates (Table 1), respectively. This is similar to results obtained in the United States (3) where C10 and C23 comprised 95% and 3%, respectively, of the isolates identified. Only one other race (C2) was found in western Canada, possibly because the great race C10 epidemic everwhelmed any other races that may have been present.

In eastern Canada, where race C9 (U.S. 87) traditionally predominates (1, 2) C10 was also the most common race, comprising 84% (vs 13% for C9) of all isolates identified in 1977. Although the rust populations of eastern and western Canada are usually distinct, it appears that the epidemic in the south central states and western Canada may have affected the rust population of eastern Canada in 1977.

Virulence on resistance conferred by gene Pg 8 increased sharply in eastern Canada (84% vs 35% for 1976) while that on Pg 9 and Pg 13 (Table 2) decreased, due to the shift from race C9 to race C10. The increased virulence on resistance conferred by genes Pg 2 and Pg 4 in western Canada is also attributable to the increased prevalence of race C10.

In an effort to detect the evolution of new virulence combinations in the rust population, a natural-infection trap nursery consisting of breeding lines and various other genotypes was planted at Glenlea, Manitoba. The isolates obtained from this material (Table 3) were less variable than in previous years (2). Probably the race C10 epidemic masked the presence of other virulence combinations. This nursery produced only one race (C1) not found in the field survey (Table 1) but it also produced several isolates of race C10 and one of C23 virulent on Rod 0^2 x C.I. 9139 (Pg X). However, these isolates are not virulent on Pg 9 and Pg 13 resistance and so present no immediate threat to the breeding program.

[1] *Contribution No. 841, Research Station, Agriculture Canada, Winnipeg, Manitoba, R3T 2M9*

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Table 1. Virulence combinations of oat stem rust field isolates on backcross lines with single-gene resistance to stem rust in Canada in 1977

Designation	Avirulence/virulence formula (Pg gene)	No. of isolates from: Ont.	Man.	Sask.	Total isolates	Percentage of total isolates
C2	1,2,4,8,13/3,9		3		3	0.9
C8	3,8,13/1,2,4,9	1			1	0.3
C9	8,13/1,2,3,4,9	4			4	1.2
C10	9,13/1,2,3,4,8	27	182	76	285	89.3
C23	2,4,9,13/1,3,8		9	17	26	8.1
TOTAL		32	194	93	319	

Table 2. Frequency of virulence in the oat stem rust population on various types of resistance in eastern and western Canada in 1977

Source of isolates	Percentage of isolates virulent on cultivars with the following genes for resistance: Pg 1	Pg 2	Pg 3	Pg 4	Pg 8	Pg 9	Pg 13	Pg (X)	Total no. isolates	Mean virulence capability*
East	100	100	97	100	84	16	0	0	32	4.9
West	99	90	100	90	99	1	0	0.3	287	4.8

*Mean virulence capability = No. of isolates virulent on Pg 1 + Pg (X)/total no. of isolates.

Table 3. Virulence combinations of oat stem rust isolates obtained from a trap nursery at Glenlea, Manitoba in 1977 on backcross lines with single-gene resistance to stem rust

Designation	Avirulence/virulence formula	No. of isolates	% of total
C1	1,2,3,4,8/9,13	7	3.1
C2	1,2,4,8/3,9,13	10	4.8
C10	9,13/1,2,3,4,8	207	91.
C23	2,4,9,13/1,3,8	3	1.3
TOTAL		227	

Acknowledgements

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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Leaf rust of wheat in Canada in 1977

D.J. Samborski[1]

Leaf rust of wheat was first found in Manitoba on June 17. However, subsequent development was slow and leaf rust caused little damage to wheat in 1977. The leaf rust race survey was carried out with 18 backcross lines with single genes for resistance as differential varieties. Lines with resistance genes *Lr 11, Lr 16, Lr 19* and *Lr 21* were resistant to all isolates in 1977 and only one isolate was virulent on *Lr 9*. Thirty-one virulence combinations on 13 genes for resistance were identified in 1977. A culture virulent on Tobari was isolated for the first time in Canada.

Can. Plant Dis. Surv. 58:3, 53-54. 1978

Le premier cas de rouille des feuilles du blé au Manitoba a été relevé le 17 juin 1977. L'évolution ultérieure a toutefois été lente et les dégâts causés par cette maladie ont été minimes. L'auteur a mené une étude des races de la rouille des feuilles sur 18 lignées de rétrocroisements à gènes de résistance simples utilisées comme variétés réactifs. Les lignées possédant les gènes de résistance *Lr 11, Lr 16, Lr 19* et *Lr 21* ont résisté à tous les isolats en 1977 et seul un isolat a manifesté de la virulence envers le gène *Lr 9*. Trente et une combinaisons de virulence sur 13 gènes de résistance ont été identifiées en 1977. Un pathotype virulent sur la variété Tobari a été isolé pour la première fois au Canada.

Disease development and crop losses in western Canada

Leaf rust of wheat (*Puccinia recondita*) was first found in Manitoba on June 17 and was widespread by early July in Manitoba and eastern Saskatchewan. However, subsequent development was slow and leaf rust caused little damage in wheat in 1977. The bread wheat varieties Neepawa, Napayo and Manitou were moderately susceptible while Sinton was resistant and Glenlea was highly resistant to leaf rust. All commercial durum varieties grown in Canada were resistant to leaf rust.

Physiologic specialization

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

A total of 203 cultures was established in 1977. These single pustule isolates were used to inoculate 18 backcross lines with single genes for resistance that served as differential varieties (1, 2, 3). Genes *Lr 11, Lr 16, Lr 19* and *Lr 21* were resistant to all isolates of leaf rust in 1977 and only one isolate was virulent on *Lr 9* (Table 1). Virulence on *Lr 9* occurs largely in eastern Canada (1) and few collections were obtained from this area. In 1965, over 50% of leaf rust isolates were virulent on *Lr 16*. At that time the variety Selkirk, which possesses *Lr 16*, occupied most of the wheat acreage in Manitoba and eastern Saskatchewan. Varieties with adult plant resistance derived from Frontana have largely replaced Selkirk in this area and virulence on *Lr 16* has also drastically declined.

A marked increase in virulence on *Lr 17* was observed in collections from Manitoba and Saskatchewan. Virulence on *Lr 17* was not obtained from this area in 1976 (1) but about 15% of the isolates were virulent in 1977 (Table 1). This may be a consequence of random fluctuations in virulence or may result from varietal changes in the United States. Gene *Lr 17* is not present in any Canadian wheat variety.

Thirty-one virulence combinations on thirteen genes for resistance were identified in 1977 (Table 2). Most of the isolates from the Canadian prairies combine virulence on *Lr 3, Lr 10* and *Lr 14a*. Although only a small number of collections were obtained from eastern Canada, most of the isolates represented different virulence combinations. For example, nine different virulence combinations were identified from the twelve isolates obtained from Quebec.

Composite collections of leaf rust were used to inoculate a number of highly resistant varieties of wheat. A number of single pustule isolates that developed on these varieties were studied but most of these were similar to cultures already described (Table 2). One isolate was virulent on Tobari (avirulence/virulence formula B, 3ka, 10, 17, 24, T/1, 2a, 2b, 2c, 3, 14a, 18). This is the first culture isolated in Canada with virulence on Tobari.

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I am grateful for the assistance given by co-operators in the care of the rust nurseries. Mr. W. Ostapyk performed the technical aspects of the program.

Table 1. Virulence of isolates of *Puccinia recondita* on backcross lines containing single genes for resistance to leaf rust in Canada in 1977.

Resistance genes	No. of virulent isolates from:					Total no. of virulent isolates	% virulent isolates
	Alta.	Sask.	Man.	Ont.	Que.		
Lr 1	0	18	24	3	5	50	24.7
Lr 2a	1	13	13	3	1	31	15.3
Lr 2b	1	13	13	7	2	36	17.7
Lr 2c	1	17	17	9	8	52	25.6
Lr B	0	0	2	6	7	15	7.4
Lr 3	3	93	80	12	9	197	97.1
Lr 3ka	0	4	2	5	7	18	8.9
Lr 9	0	0	0	0	1	1	0.5
Lr 10	1	69	54	9	5	138	68.0
Lr 11	0	0	0	0	0	0	0.0
Lr 14a	3	93	82	8	5	191	94.1
Lr 16	0	0	0	0	0	0	0.0
Lr 17	0	15	18	0	2	35	17.2
Lr 18	1	7	8	3	5	24	11.8
Lr 19	0	0	0	0	0	0	0.0
Lr 21	0	0	0	0	0	0	0.0
Lr 24	0	8	8	1	1	18	8.9
Lr T	0	4	4	5	3	16	7.9

Table 2. Virulence combinations of *Puccinia recondita* isolates on backcross lines containing single genes for resistance to leaf rust in Canada in 1977.

Avirulence/virulence formula	No. of isolates from:					Total no. of isolates
	Alta.	Sask.	Man.	Ont.	Que.	
1, 2a, 2b, 2c, B, 3ka, 10, 17, 18, 24, T/3, 14a	1	5	6	1	2	15
1, 2a, 2b, 2c, B, 3ka, 17, 18, 24, T/3, 10, 14a	0	53	40	2	0	95
1, 2a, 2b, 2c, B, 3ka, 10, 18, 24, T/3, 14a, 17	0	1	1	0	0	2
1, 2a, 2b, 2c, B, 3ka, 10, 17, 24, T/3, 14a, 18	1	1	0	0	0	2
1, 2a, 2b, 2c, B, 3ka, 10, 17, 18, T/3, 14a, 24	0	5	7	0	0	12
2a, 2b, 2c, B, 3ka, 10, 17, 18, 24, T/1, 3, 14a	0	1	0	0	0	1
1, 2a, 2b, 3, 3ka, 10, 14a, 17, 24, T/2c, B, 18	0	0	0	1	0	1
1, 2a, 2b, 2c, B, 3ka, 17, 18, T/3, 10, 14a, 24	0	1	0	1	0	2
1, 2a, 2b, 2c, B, 3ka, 17, 24, T/3, 10, 14a, 18	0	1	0	0	0	1
1, 2a, 2b, 2c, B, 17, 18, 24, T/3, 3ka, 10, 14a	0	0	0	1	0	1
1, 2a, 2b, 2c, 10, 17, 18, 24, T/B, 3, 3ka, 14a	0	0	0	0	2	2
2a, 2b, 2c, B, 3ka, 17, 18, 24, T/1, 3, 10, 14a	0	0	2	0	0	2
2a, 2b, 2c, B, 3ka, 10, 18, 24, T/1, 3, 14a, 17	0	6	8	0	0	14
2a, 2b, B, 3ka, 14a, 17, 18, 24, T/1, 2c, 3, 10	0	0	0	0	1	1
1, 2a, 2b, 3, 3ka, 14a, 17, 24, T/2c, B, 10, 18	0	0	0	0	2	2
2a, 2b, 2c, B, 3, 10, 17, 18, T/1, 3ka, 14a, 24	0	0	0	0	1	1
2a, 2b, 2c, B, 3ka, 17, 18, T/1, 3, 10, 14a, 24	0	2	1	0	0	3
1, 2a, 2b, 10, 14a, 17, 24, T/2c, B, 3, 3ka, 18	0	0	0	0	1	1
B, 3, 3ka, 10, 14a, 18, 24, T/1, 2a, 2b, 2c, 17	0	0	0	0	1	1
1, B, 3ka, 17, 18, 24, T/2a, 2b, 2c, 3, 10, 14a	1	8	5	0	0	14
2a, 2b, 3, 3ka, 18, 24, T/1, 2c, B, 10, 14a, 17	0	0	2	0	0	2
1, B, 3ka, 17, 18, 24, T/2a, 2b, 2c, 3, 10, 14a	0	0	0	3	0	3
1, 2a, 3, 3ka, 14a, 17, 24/2b, 2c, B, 10, 18, T	0	0	0	1	0	1
B, 3ka, 10, 17, 24, T/1, 2a, 2b, 2c, 3, 14a, 18	0	1	1	0	0	2
2a, 10, 14a, 17, 18, 24/1, 2b, 2c, B, 3, 3ka, T	0	0	0	3	0	3
2a, 2b, B, 14a, 18, 24/1, 2c, 3, 3ka, 10, 17, T	0	0	0	0	1	1
1, 2a, 10, 14a, 17, 24/2b, 2c, B, 3, 3ka, 18, T	0	0	0	0	1	1
1, 2a, 2b, 14a, 17, 24/2c, B, 3, 3ka, 10, 18, T	0	0	0	1	0	1
B, 3ka, 10, 24, T/1, 2a, 2b, 2c, 3, 14a, 17, 18	0	4	5	0	0	9
2a, 2b, B, 18, 24/1, 2c, 3, 3ka, 10, 14a, 17, T	0	4	2	0	0	6
2a, 2b, 14a, 17, 24/1, 2c, B, 3, 3ka, 10, 18, T	0	0	0	0	1	1

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Announcement

The composition of the Editorial Board of the Canadian Plant Disease Survey has recently been changed.

It has been brought to the attention of the new editorial board that a number of articles reporting incidental disease observations of crops in Canada for 1974 were never published. As these papers were unduly delayed and in the opinion of the board not now timely, we have decided with regret to no longer consider them for publication.

However, as the authors spent considerable time and effort in the preparation of the material and to afford them some accreditation, the titles are listed below. Anyone interested in obtaining information on disease incidence for 1974 may contact the various authors directly.

V. R. Wallen
Chairman, Editorial Board

Plant diseases in central and northern Alberta, 1974.

B. Berkenkamp
Research Station, Agriculture Canada, Lacombe, Alberta

Plant diseases in southern Alberta, 1974.

F.R. Harper
Research Station, Agriculture Canada, Lethbridge, Alberta

Plant diseases in British Columbia, 1974.

H.S. Pepin and D.L. McIntosh
Research Stations, Agriculture Canada, Vancouver and Summerland

Plant diseases in Manitoba, 1974.

J.W. Martens
Research Station, Agriculture Canada, Winnipeg, Manitoba

Plant diseases in Newfoundland, 1974.

M.C. Hampson
Research Station, Agriculture Canada, St. John's West, Newfoundland

Plant diseases in Nova Scotia, 1974.

C.O. Gourley
Research Station, Agriculture Canada, Kentville, Nova Scotia

Plant diseases in eastern Ontario, 1974.

R.V. Clark
Research Station, Agriculture Canada, Ottawa, Ontario

Plant diseases in the Niagara Peninsula, Ontario, 1974.

T.R. Davidson
Research Station, Agriculture Canada, Vineland Station, Ontario

Plant diseases in southwestern Ontario, 1974.

J.H. Haas and S.K. Gayed
Research Stations, Agriculture Canada, Harrow and Delhi, Ontario

Plant diseases in Prince Edward Island, 1974.

L.C. Callbeck
Research Station, Agriculture Canada, Charlottetown, P.E.I.

Relève de quelques maladies des plantes dans la province du Québec, 1974.

J. Santerre[1], D. Chez[4], J.P. Dubuc[1], H. Généreux[2], J. Laganière[3], J. Lu[4], G. Pelletier[5], C. Richard[1], J. Ringuet[4], et P.O. Thibodeau[4]
Agriculture Canada: [1] Station de recherches, Ste-Foy; [2] Ferme expérimentale et [3] Certification des pommes de terre, la Pocatière; [4] Ministère de l'Agriculture du Québec: Complexe scientifique, Ste-Foy; [5] Université Laval, Ste-Foy, Québec

Etat phytosanitaire des productions végétales au sud-ouest du Québec, 1974.

R. Crête
Station de recherches, Agriculture Canada, C.P. 457, St-Jean, Québec

Plant diseases in Saskatchewan, 1974.

R.J. Ledingham
Research Station, Agriculture Canada, Saskatoon, Saskatchewan

A search for leaf roll virus that does not cause diagnostic symptoms in potato foliage

R.H.E. Bradley[1]

Potatoes from various parts of New Brunswick, Canada, were tested for leaf roll virus by means of aphid vectors and the indicator plant *P. floridana*. Only moderate strains of the virus were recovered, always from plants that developed clear symptoms of leaf roll. There was no conclusive evidence that any of the potatoes were infected by mild strains that do not cause leaf rolling in potatoes. When Kennebec plants from various sources were inoculated with a moderate strain of the virus, all of them became infected showing that they were not already infected by mild strains that protect against stronger ones. Mild strains of leaf roll were not recovered from supposedly infected tubers obtained from western Canada. Nor was the virus recovered from these same clones during concurrent tests made in western Canada by one of those who made the original diagnosis of infection some years ago. Possible reasons from these unexpected results are mentioned. In any event there is no reason to believe that mild strains of leaf roll are common in New Brunswick.

Can. Plant Dis. Surv. 58:3, 56-60. 1978

La réaction au virus de l'enroulement des pommes de terre provenant de diverses régions du Nouveau-Brunswick a été mesurée en utilisant les pucerons comme agents de transmission et *P. floridana* comme plante indicateur. On n'a pu trouver que des souches de virulence moyenne et toujours sur des plants qui présentaient des symptômes nets d'enroulement. Il ne semble pas que les plants aient été infectés par des souches bénignes qui ne provoquent pas l'enroulement chez la pomme de terre. Quand on a inoculé des plants de la variété Kennebec provenant de diverses sources avec une souche modérée du virus, tous les plants ont par la suite été virosé ce qui montre qu'ils n'étaient pas déjà infectés d'une souche bénigne les protégeant des souches plus virulentes. On n'a pas isolé de souches bénignes dans des tubercules prétendument infectés provenant de l'ouest du Canada, pas plus qu'on en a retrouvé dans ces mêmes clones au cours d'essais simultanés effectués dans l'ouest du pays par l'un des chercheurs qui avait établi le premier diagnostic d'infection, il y a quelques années. Plusieurs raisons plausibles sont proposées pour expliquer ces résultats imprévus. Quoi qu'il en soit, rien ne permet de croire que les souches faibles de l'enroulement soient courantes au Nouveau-Brunswick.

Introduction

Wright and MacCarthy (10) reported that they recovered leaf roll virus (LRV) from potatoes that did not develop diagnostic symptoms of leaf roll in the field or greenhouse. Their first recoveries were made from Kennebec potatoes grown near the west coast of Canada. Apparently these isolates were designated as LRV because: (a) they were obtained from potato, (b) transmissible via aphids, and (c) in the host plant *Physalis floridana* Rybd. they caused symptoms like those that Webb *et al.* (9) had described for mild strains of LRV. But these three characteristics in common are not sufficient to establish a strain relationship. Moreover, the Kennebec isolates differed from previously described strains of LRV (2, 9) in that they did not cause leaf roll symptoms in potato plants nor protect them from virulent LRV (11). So it may be premature to designate these isolates as LRV. Assuming that they are viruses, I shall refer to them simply as mild aphid-borne potato virus (MAPV). The term mild LRV will be used here for isolates having the characteristics of the mild strains described by Webb *et al.* (9).

Later Wright *et al.* (11) recovered MAPV from 25% of plants of 16 potato varieties obtained from various sources in Canada and the United States, recoveries being made from all the varieties except 3 of which only a total of 10 plants were tested. From this it appeared that MAPV may be widespread in North America. If so, and it does not cause diagnostic foliar symptoms in other areas, presumably it will spread undetected and eventually become endemic. An endemic potato virus is potentially troublesome even though it does not cause obvious disease symptoms. Especially vulnerable would be an area like eastern Canada where most of the country's seed potatoes are grown and large quantities are exported to many countries.

Inevitably the work by Wright and colleagues caused concern in other potato growing areas. Perhaps nowhere was the impact greater than in the Eastern Canadian province of New Brunswick, because Kennebec is widely grown there and appears to be very tolerant to MAPV (12). To determine if MAPV was already endemic in New Brunswick, MacKinnon (5) tested various potatoes that were at hand but found none infected. That was in 1970 and shortly thereafter the incidence of leaf roll in New Brunswick increased to the point where there was widespread fear of an impending epidemic. In trying to account for this unexpected increase, some people

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postulated on the basis of the work by Wright *et al.* (10, 11, 12) that the New Brunswick clones of Kennebec may be infected by LRV that does not cause symptoms in that variety but causes leaf roll when transmitted via aphids to other locally grown varieties. The previous tests by MacKinnon had not been extensive enough to preclude this possibility, and since he no longer worked in the province I was asked to check New Brunswick potatoes further for strains of LRV that do not cause diagnostic foliar symptoms.

Materials and methods

The potatoes for testing were grown in a screened greenhouse, singly in pots of fertile soil. Unless otherwise stated they were from tubers of the 1973 New Brunswick potato crop, the various sources being given later. Most of the testing was carried on during the winter, spring, and summer of 1974 and 1975. After each tuber was marked with an identification number, a small piece was removed from the seed end for planting and the rest stored so that additional plants could be grown from it for retesting if necessary. Except where otherwise stated each plant was tested for LRV and MAPV soon after emergence and before any foliar symptoms had developed. An attempt was made to transmit virus from each plant by means of aphids of *Myzus persicae* (Sulz.) to seedlings of *P. floridana* in the cotyledon stage.

The method of testing was essentially the same as that described originally by MacCarthy (3) and also used by Wright *et al.* (10, 12). Like others who have used this method, I modified it slightly from time to time to better suit local conditions; but each change was first checked to be sure that it did not interfere with the test by visibly affecting the test seedlings. Initially the test seedlings were grown individually in small pots, but mostly they were grown in 9 inch diameter bulb pots, about 12 evenly spaced seedlings per pot. The *P. floridana* seed used was derived from the same line as that used by Wright *et al.* (10, 12).

Experience showed that with aphids from my culture of *M. persicae*, which was reared on rape, the nymphs remained and fed on the test seedlings much better than the apterous adults used by others (3, 10, 12). So for the most part my tests were made with nymphs. To avoid transferring them twice by hand, each potato was infested with 2 or 3 vigorously reproducing apterous adults, which would give birth to 30 or more nymphs within about 3 days. About a week after the potato was infested, from 4 to 6 of the largest nymphs were transferred to each of 4 test seedlings. Following this the potato was freed of aphids by spraying it with a 1:500 aqueous solution of Black Leaf 40 (nicotine sulphate) and returned to the glasshouse for the periodic observation of disease symptoms.

Once infested each seedling was covered with a small transparent cylinder to confine the aphids, which were allowed 2 days to feed at about 20°C. After this the seedlings were freed of aphids and maintained about a month in a growth cabinet at 27°C, 84% R.H., and 1200 ft-c of fluorescent light for 16 hr each day (Cabinet Model 60 made by Controlled Environments Ltd., Winnipeg, Manitoba). When aphids were removed from each seedling by means of a small brush as recommended by MacCarthy (3), some of them were invariably missed and reproduced before being detected. This led to the growth cabinet becoming infested and there were a few accidental transmissions of LRV to healthy controls at the start of the work. To prevent this from happening all seedlings were subsequently freed of aphids by spraying them with a 1:500 solution of Black Leaf 40, which did not visibly affect the test seedlings; and there was no further evidence of accidental transmissions of virus.

Two sets of controls were included in all tests: one was healthy seedlings to check for accidental transmissions; the other was to show whether the aphids being used and the method would result in a high level of virus transmission. About one seedling out of 5 was left as a healthy control. Initially every second seedling of this group was infested with about 5 nymphs from the aphid culture on rape, and the remaining healthy controls were left uninfested. After some weeks of testing when no differences were observed between the infested and uninfested controls, subsequently all of them were left uninfested. For the second set of controls, several seedlings were infested each week with nymphs from potatoes known to be infected by LRV. The number of nymphs per seedling, the time they spent on the source of virus and test seedlings, and the test method were the same as those described above.

Results

Growth and appearance of the P. floridana test seedlings

When young seedlings of *P. floridana* are infected by mild LRV (9) or by MAPV (10), the symptoms that have been described are very mild consisting mainly of a slight upward rolling of the leaves, which may also show a mild chlorosis and epinasty and there may also be slight twisting of the petioles. Apparently these symptoms are so mild that they are difficult to diagnose unless the test seedlings grow uniformly and well and otherwise appear healthy. During my tests more than 5000 test seedlings were used and for the most part their growth was excellent and uniform. Occasionally a seedling died for no apparent reason while it was infested by aphids or within a few days of being placed in the growth cabinet; and now and then a seedling developed a slight chlorosis and was slightly stunted. But unless otherwise stated both of these anomalies occurred as often in healthy controls as in the test seedlings and could not therefore safely be attributed to virus infection. Whenever more than one of a set of four test seedlings did not grow well that test was repeated in its entirety, either by using the same potato plant while it was still young or by growing another plant from the original tuber. Also for some unknown reason sometimes an entire pot of seedlings

including the controls became slightly chlorotic. Since these seedlings were unsuitable for showing the symptoms expected of virus infection, these tests also had to be repeated.

Although the incidence of the above growth anomalies was low, it increased the difficulty of diagnosing infections by both mild LRV and MAPV. Therefore, early in the work I decided that if there was any suggestion whatsoever that a test might be positive it was repeated over and over until there was no doubt about the result.

Potatoes from the New Brunswick elite potato seed farm at Bonaccord

Tubers from the provincial seed farm were tested first because at the time it seemed important to determine if mild LRV or MAPV was endemic in the main source of Elite seed for the province. Since the lowest grade of seed grown there is Elite II, it was tested initially on the assumption that the highest level of virus infection ought to occur in the lowest grade of seed. Single plants were grown from each of 850 tubers -- 400 of Kennebec and 450 of Netted Gem (Russet Burbank). Soon after emergence 135 of the Kennebec plants and 50 of the Netted Gem were selected at random for aphid transmission tests to *P. floridana*. The results were as follows: 2 potatoes proved to be infected by a moderate strain of LRV, both being of the variety Kennebec; and there was no conclusive evidence that any of the remaining 183 potatoes were infected by an aphid-borne virus that causes symptoms in *P. floridana*. Several of the initial tests were repeated for various reasons given in the preceding section. In one of the initial tests all 4 seedlings became slightly chlorotic with mild rolling of the lower leaves, rather like the symptoms expected of either mild LRV or MAPV. Furthermore, none of the nearby controls developed these symptoms. Thus it appeared that the test was positive. As soon as this was observed, attempts were made to transmit virus via aphids from each of the 4 test seedlings showing symptoms to healthy test seedlings, the methods and numbers of nymphs used being the same as that for the tests from potato. Also, the original potato plant was retested twice before it was 40 cm high, and a second plant grown from the original tuber for retesting at emergence. Despite repeated attempts to transmit virus via aphids from the original 4 test seedlings and from the original potato and another from the same tuber, none of the *P. floridana* seedlings used developed any virus-like symptoms. Nor did the potato plants themselves develop any disease symptoms. Thus it could not be confirmed that this potato, which was Kennebec, was infected by a mild aphid-borne virus.

All 400 plants of Kennebec and 450 of Netted Gem were observed periodically for symptoms of leaf roll until they were mature and dying. The readings were as follows:

Leaf Roll Symptoms	*Kennebec*	*Netted Gem*
Typical	5	3
Atypical	10	7
None	385	440

Of the potatoes that developed typical leaf roll, 2 of the Kennebec had been tested by means of aphids and *P. floridana* at emergence and both had given an unmistakable positive test. The remaining 6 potatoes that developed typical leaf roll were tested when about 20 cm high and each also gave a positive test, the symptoms on *P. floridana* corresponding to those that Webb *et al.* (9) described for moderate strains of LRV.

The 17 potatoes that were diagnosed as showing atypical leaf roll developed a variety of symptoms. These ranged from very weak plants with all leaves rolled upwards to unstunted plants with slight upward rolling of the lower leaves. As soon as any symptoms suggestive of leaf roll were observed on these potatoes, each was tested for virus by means of aphids and *P. floridana*. Also the 10 potatoes that developed the least severe symptoms and therefore the ones judged to be the most likely to be infected by mild LRV or MAPV, were given additional tests. For these a second plant was grown from the original tuber, an aphid transmission test to *P. floridana* carried out at emergence, and this test repeated from one to 4 times during the first month of growth. None of the 10 additional potatoes grown from the original tubers developed any leaf rolling as the first plants had done. And none of the aphid transmission tests from the original potatoes showing symptoms or the second plants without symptoms gave any evidence that the tubers were infected by LRV or MAPV.

Throughout the tests of Elite II seed from the provincial farm, the method used gave excellent transmission of LRV from the potatoes found to be infected and from known sources of LRV used as controls. In all 68 *P. floridana* seedlings were infested with nymphs from potatoes known or later found to be infected by LRV, and all but 3 of these seedlings developed unmistakable symptoms of leaf roll.

When no evidence was obtained that tubers from the 1973 crop of the provincial seed farm were infected by mild LRV or MAPV, somewhat different samples of tubers were tested the following two years. Some potatoes are rejected every year at the farm because of mild to moderate rolling of the leaves, this being recorded as 'suspicious leaf roll'. If mild LRV or MAPV occurred on the farm, it seemed more likely to be found in such plants once no evidence of either virus had been found in random tuber samples. So tubers were obtained for testing from plants diagnosed as 'suspicious leaf roll' either during the growing season in New Brunswick or during the checking of these stocks each winter in Florida. Of the tubers tested, 15 were of Kennebec, 5 were of Netted Gem, and there was one each of Red Pontiac, Fundy, and Belleisle. When plants were grown from these tubers in the greenhouse at Fredericton, all of them appeared healthy and showed no symptoms of leaf roll whatsoever. And when the plants were tested by means of aphids and *P. floridana*, none of the test seedlings developed any symptoms suggestive of leaf roll. Thus there was no reason to

believe that any of these rejected potatoes were infected by mild LRV or MAPV.

Kennebec seed potatoes from commercial growers

Samples of Kennebec tubers were obtained from each of 18 seed growers scattered throughout the main potato growing area. The numbers of samples that were of Class Elite III, Foundation, and Certified were 4, 9 and 5 respectively. From 20 to 40 tubers were selected at random from each sample for testing, the total being 460. When single plants were grown from each tuber and tested at emergence by using *P. floridana*, 3 of the potatoes were found to be infected by a moderate strain of LRV judging from the symptoms in *P. floridana* (9). No conclusive evidence was obtained that any of the remaining 457 potatoes were infected by mild LRV or MAPV. As the potato plants grew and were observed for foliar symptoms, 3 developed typical leaf roll symptoms, these being the same plants that had been found infected by the *P. floridana* test. Of the remaining potato plants, 429 showed no symptoms of leaf roll whatsoever, and 28 were diagnosed as atypical leaf roll. These latter plants developed symptoms ranging from slight rolling of lower leaves to severely stunted plants whose leaves developed various degrees of rolling. Although these 28 plants had been tested at emergence and none found to be infected, each one was retested when about 20 cm high using *P. floridana* and again no evidence was obtained that any was infected by an aphid-borne virus that causes symptoms in *P. floridana*. Furthermore a single tuber was saved from each of the 28 atypical leaf roll plants and a plant grown from each tuber for retesting the following year. Not only was no evidence obtained that any of these second generation plants was infected by mild LRV or MAPV, but all of the tubers except 2 produced healthy appearing plants. The 2 exceptions developed atypical leaf roll, but further tests of these for aphid-borne virus gave only negative results.

Although there was no evidence that any of the potatoes tested thus far were infected by either mild LRV or MAPV, it is possible that they were infected by strains that are so mild they do not cause symptoms in either potato or the indicator plant *P. floridana*. One means of testing for such strains would be to determine if the potatoes are protected from infection by known strains of LRV. Protection would be evidence of mild LRV; but lack of it would not prove the absence of symptomless strains of LRV or of MAPV (10). A test for protection was made by growing a second plant from each of the first 5 tubers selected from each of the 16 samples of Kennebec seed from commercial growers. Soon after emergence each plant was infested for 2 days with from 5 to 10 nymphs that had been reared on potatoes infected by moderate LRV. Nearly all of the 80 plants infested this way developed clear symptoms of leaf roll as they matured. Furthermore, when 2 tubers were saved from each of the 80 plants and replanted the following year, all 160 of the tubers gave rise to plants that developed typical leaf roll. Thus there was no evidence that any of the potatoes were protected from infection by LRV.

Progeny of potatoes from which MAPV had been recovered

As the testing progressed and MAPV was not recovered, one could not ignore the possibility that the method being used might not be suitable under my conditions. With the intention of checking this point, I requested MAPV infected tubers from Dr. H.R. MacCarthy of the Vancouver Research Station where the original work on this virus was done. He kindly supplied one tuber from each of 6 clones of Netted Gem that had been found to be infected some years previously. Since the original diagnosis, these clones had been maintained in the field and were tested for MAPV from time to time. The virus had been recovered during each of these periodic tests except the final one made a few months prior to my request. In view of this unexpected result, Dr. MacCarthy decided to make further tests using tubers of the same clones that were forwarded to me. Later he informed me that despite repeated tests he had failed to recover MAPV from any of the clones sent to me and from other supposedly infected clones as well. In the meantime I too had tried repeatedly and failed to recover MAPV from any of the Vancouver clones. Initially 2 plants were grown from each of the 6 tubers, one being kept for the observation of disease symptoms and the other tested for MAPV by means of aphids and *P. floridana* seedlings. One plant from each clone was tested at emergence, again when about 20 cm high, and again at flowering. Furthermore tubers were saved from each of the plants observed for symptoms and plants grown from these for testing at various stages of growth the following year. More than 100 *P. floridana* seedlings were infested with aphids from plants derived from the Vancouver clones of Netted Gem without obtaining any conclusive evidence of MAPV or any other aphid-borne virus that causes symptoms in *P. floridana*. The 6 potato plants observed for disease symptoms did not appear as vigorous as did those of New Brunswick clones of Netted Gem and at first this was looked upon as possibly being mild symptoms caused by MAPV or another virus. But other than this no confirmation of a virus in the Vancouver clones was obtained.

Miscellaneous tests

The breeding of new varieties of potato in Canada is mainly carried out at the Fredericton Research Station with the seedlings being grown in isolation at a substation. At the time of my tests for MAPV and mild LRV, there was a minor outbreak of leaf roll in the potato seedlings being grown at the substation, and there was concern that this might stem from mild strains of LRV like those reported by Wright *et al.* (10, 12). In view of this I was asked to test samples of the seedlings from LRV that does not cause diagnostic symptoms in potato foliage. Accordingly 10 tubers were tested from seedling lines that had been maintained at the substation for some years on the assumption that these lines were the

most likely to be infected. A single test of plants derived from each tuber gave no indication whatsoever that they were infected by MAPV or mild LRV. At about the same time as these 10 tubers were tested, another 30 tubers were sent for similar testing to Dr. N.S. Wright of the Vancouver Research Station; 10 of these tubers were from the older seedlings lines like those tested by me and 20 were of the cultivar Kennebec that had been propogated for some years at the potato seedling substation. Subsequently Dr. Wright reported that his tests gave no evidence that any of the 30 tubers were infected by mild LRV.

Discussion

Had MAPV been recovered during this work as there was every reason to expect (10, 12), it could have been characterized further until it was shown whether it is related to LRV or not. The failure to recover it from supposedly infected western clones in itself shows a need for further study, especially since similar results were obtained during concurrent tests in western Canada where the original diagnosis of infection was made. Why the virus could not be detected in the western clones cannot be explained, but there are several possibilities based on what has been reported for other plant viruses. For example, maintaining the virus for some years in potatoes reproduced vegetatively may have caused it to become further attenuated until it no longer causes visible symptoms in the indicator plant *P. floridana*; or the virus may have ceased to be transmissible via its vector as has been reported for some other aphid-borne viruses that have been maintained a long time without being transmitted by their vectors (e.g. 1, 7, 8).

Had MAPV been recovered from the western clones, my failure to recover it from New Brunswick potatoes would have been convincing evidence that it is not endemic in New Brunswick. But without any positive tests to confirm the test method that was used, one could postulate that MAPV had been endemic in New Brunswick for some time and that it cannot now be detected by the *P. floridana* test just as was found with the supposedly infected western clones. If such mild strains do occur, some way will have to be found to detect them before one can determine if they are important enough to justify control measures; and presumably they would be widespread in potatoes of most countries just as was found with other latent potato viruses such as X and S.

A more likely interpretation of my results is that neither MAPV nor mild LRV are common in New Brunswick. All of the isolates made during my tests caused clear symptoms of leaf roll in potatoes, and similar results were reported some years ago by Webb *et al.* (9), who tested 36 isolates of LRV from eastern Canada. If a mild strain of LRV that does not cause leaf roll symptoms in potato did occur in New Brunswick, possibly it would be controlled under our conditions. Unless our varieties were completely tolerant to such strains, they would affect plants enough that they would appear abnormal, and such plants would be removed during roguing of seed potatoes where the practice is 'if in doubt pull it out'. In a major seed growing area like New Brunswick where aphid-borne viruses spread slowly, the roguing of infected plants from the highest grades of seed usually suffices to give practical control throughout the crop. Certainly plants showing atypical leaf roll symptoms would be removed during roguing, though on the basis of my tests these are rarely infected by LRV.

Finally, my results are similar to those reported by Manzer *et al.* (6), who carried out a similar research at about the same time in the adjoining state of Maine. They found that aphid transmission tests to *P. floridana* gave a few more positive readings than did field readings of leaf roll symptoms in potato foliage. They concluded that the higher readings with *P. floridana* probably resulted from mild LRV like that reported by Wright *et al.* (10, 12). But the symptoms that they obtained in *P. floridana* were often questionable, so they cautioned that their tests with *P. floridana* probably overestimated the incidence of LRV. Nor did they confirm that the *P. floridana* showing questionable symptoms were in fact infected by an aphid-borne virus by transmitting it via aphids to other *P. floridana* seedlings. In any event, the searches in both Maine and New Brunswick gave no reason to believe that mild strains of LRV are common in these important seed growing areas.

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The effect of weeds on the value of rotation as a practical control for Verticillium wilt of potato

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In microplot tests in Ontario rotation of potatoes with barley or fallow proved ineffective in controlling Verticillium wilt caused by *Verticillium dahliae* or *Verticillium albo-atrum*. Several weed species were excellent hosts of *V. dahliae*, and microsclerotia were produced on their roots within 2 weeks of inoculation. In soil samples averaging 1.3 microsclerotia of *V. dahliae* per g of soil 80-90% of the Kennebec potato test plants became infected. It is suggested that variations in the number of viable microsclerotia in soil at the start of a rotation experiment, the effectiveness of the weed control program, and the weather during the experiment could account for the controversy surrounding the value of rotation as a means of control. The authors suggest that in Ontario rotation would be of no value in controlling Verticillium wilt of potato.

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Au cours d'essais en micro-parcelles effectués en Ontario, l'alternance de la sole de pomme de terre avec l'orge ou avec une jachère s'est révélée impuissante dans la lutte contre la flétrissure verticillienne causée par *Verticillium dahliae* ou *Verticillium albo-atrum*. Plusieurs espèces de mauvaises herbes constituaient d'excellents hôtes de *V. dahliae* et leurs racines produisaient des microslérotes moins de deux semaines après l'inoculation. Dans les échantillons de sols possédant en moyenne 1.3 microsclérotes de *V. dahliae* par gramme de sol, 80 à 90% des plants d'essai de pommes de terre de la variété Kennebec ont été atteints. Il semblerait que des variations du nombre de microsclérotes dans le sol au début de l'expérience de rotation, l'efficacité du programme de désherbage et les conditions météorologiques prévalaient au cours de l'expérience pourraient partiellement expliquer la polémique sur la valeur de la rotation comme moyen de lutte. Les auteurs estiment qu'en Ontario, la rotation n'aurait aucune valeur pour la lutte contre la flétrissure verticillienne de la pomme de terre.

In Ontario Verticillium wilt caused by *Verticillium albo-atrum* Reinke & Berth and *Verticillium dahliae* Kleb. is a serious disease of potato, tomato, eggplant, strawberry, maple, and other hosts. The two pathogens are soil-borne fungi that attack their hosts through the root system. Another important source of inoculum is infested potato seed pieces. The latter may be the major recurring source of inoculum of *V. albo-atrum*, particularly in areas where potatoes are rotated with non-hosts such as grains or corn (13).

Verticillium wilt is difficult to control; resistant varieties, crop rotation, and soil fumigation with high-value crops are the three most commonly recommended practices. Currently there is no variety of potato recommended for Ontario which can be considered resistant and, until very recently, fumigation was considered uneconomical. This situation has changed in the past few years and fumigation is currently being seriously considered. However, because of our short growing season, fumigation must be done in the fall following harvest and in many years this may not be practical.

There is considerable controversy in the literature regarding the usefulness of rotation as a means of control. Results have been conflicting and in many cases difficult to explain (11, 12, 16, 18). Some of these difficulties may be due to the presence of broadleafed weeds in the treatment areas (13). Brown and Wiles (2) state "Adequate control of the weed hosts would also be required where crop rotation was used to reduce the inoculum potential of the fungus." Unfortunately in most cases with weed hosts there are no observable symptoms, with the possible exception of stunting, to indicate infection (18).

The effectiveness of rotation is also affected by the number of viable microsclerotia present in the soil and by the number of microsclerotia per gram of soil required for infection of a particular crop. Reports of these numbers vary widely, from 0.3 to 3000 per g of soil, depending upon the crop, the workers involved, and the method of estimating the number of microsclerotia present (1, 6, 8).

This paper reports results of a crop rotation experiment on the control of Verticillium wilt of potato caused by *V. albo-atrum* and *V. dahliae*, of studies on the number of microsclerotia of *V. dahliae* per gram of soil required for infection of potato plants, and on the effects weed hosts may have on the survival of *V. dahliae*.

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Materials and methods

Crop rotation

Potatoes were grown in the field in a range of 24 microplots constructed of reinforced concrete. Each plot was 1.2 x 1.2 x 0.8 m deep, sunk in the ground with the tops 10 cm above the soil surface to prevent soil water from washing into the plots from surrounding areas or adjacent plots. The plots were filled with sandy loam to within 10 cm of the top. Nine Kennebec potato seed pieces inoculated by dipping them into a water suspension of spores and mucelium of *V. dahliae* or *V. albo-atrum* were planted in each of the plots the first year. The potato tops were left on the surface of the soil over winter and were incorporated into their respective plots the following spring. After the first year, the potato seed pieces were washed prior to planting but were not inoculated. The following rotations were utilized: 1) one year fallow followed by two years potatoes, 2) one year barley followed by two years potatoes, 3) two years barley followed by one year potatoes, and 4) potatoes three years. A record was kept each year of the percent wilt present whenever a susceptible crop was grown. There were three randomized replications for each treatment. Sections from near the soil surface of each potato stem taken in early August were placed on moist filter paper in petri dishes and examined for conidiophores 1 week later (14).

Number of microsclerotia of V. dahliae required for infection of potato

Little information is available on the number of microsclerotia (MS) of *V. dahliae* per gram of soil necessary for infection and for subsequent development of symptoms in potato. To obtain microsclerotia for soil infestation studies, *Verticillium dahliae* was cultivated at 25°C in the dark for 4 weeks on a glucose-KNO_3 mineral salts medium (9) modified by the addition of 5μg/litre biotin for maximum microsclerotia production.

Microsclerotia free from mycelium and conidia were obtained by using a modification of Congly's method (4, 5). The number of microsclerotia used to infest the soil was determined by weight. Two, 5, 8, 10, 20, 40, 70 and 100 microsclerotia were mixed with a small amount of soil in a beaker and then added to a weighed air-dried sterile potting soil sample and mixed thoroughly. For every experiment, a representative soil sample was collected from each treatment mixture prior to planting to determine by three sampling techniques the number of viable MS present in the soil. The potato plants were grown under short day conditions (10 h light - 14 h dark) in environmental chambers at 22 ± 2°C. Light intensity at plant height was 16,000 lux provided by fluorescent and incandescent lamps (3). The plants were examined for symptoms 60 days after planting. The three soil isolation techniques used were: a) *Datura* root bioassay (7), b) soil washing (7), and c) soil dilution (15). Method B was modified by using a soil sample size of 1.0 g rather than 0.1 g as used by Evans (7). Method C was modified by reducing the quantity of agar in the medium by 6.3 g/litre to prevent gelling at 40-42°C; 1.0 g of sieved (1.0 mm) soil sample was added to 400 ml of cooled (40°C) agar, shaken to disperse the soil, and then poured into 20 plates. The soil sample was not diluted with water prior to use. Method C proved to be most reliable for the recovery of MS added to the soul but even it was quite variable.

Effect of weeds on Verticillium dahliae survival

Because of the erratic results obtained from the crop rotation experiments, various weeds were examined as potential hosts of *Verticillium*. In several experiments the weeds were grown in pots in the greenhouse or light chamber and inoculated by pouring over the soil a spore-mycelial suspension of *V. dahliae*. Complete plants were harvested weekly for 3 weeks starting 2 weeks after inoculation. The roots were washed and 100 cm of the smaller roots placed on water agar and examined for microsclerotia 1 week later (6). Representative cross sections of the stems and petioles were placed on moistened filter paper in petri dishes. These sections were examined periodically with the dissecting microscope for conidiophores of *Verticillium* (14).

Results

Rotation

Table 1 summarizes the results obtained from the various cropping sequences used during 1970-73. The results are difficult to explain and do not follow any readily apparent pattern. There is an indication that *V. dahliae* survives better, i.e. maintains a higher inoculum level in the absence of potatoes than *V. albo-atrum* but it is obvious from the data that other complicating factors were operating throughout the experiment. In 1974 eggplant (*Solanum melongena* L.) was planted in all of the plots and was 100% infected by mid August. It is also interesting to note that stunting of eggplant occurred only in the *V. dahliae* plots, possibly indicating that the plants were infected and colonized sooner than those grown in the *V, albo-atrum* plots.

Weed hosts

Table 2 indicates our results from many different tests over a 2-year period. In all of the weed species inoculated with *V. dahliae* the pathogen was isolated more often from the cortical tissue of the roots than from aboveground parts. Frequently microsclerotia were produced in the root tissue harvested at the first sampling date 2 weeks after inoculation; with some of the weed hosts the fungus was isolated only from the roots, never from the above ground parts. With the exception of groundsel (*Senecio vulgaris* L.), the weed hosts generally did not exhibit symptoms. Several cruciferous weeds proved to be infected and colonized by *V. dahliae*.

Table 1. Effect of cropping practices on survival of 2 species of *Verticillium* in a 4-year rotation in microplots

Crop sequence*	%potato plants infected†							
	Verticillium dahliae				*Verticillium albo-atrum*			
	1970	1971	1972	1973	1970	1971	1972	1973
1	100		40	70	100		6	49
2	100		55	56	100		>1	33
3	100			79	100			63
4	100	73	53	97	100	37	31	53

*1 - Potatoes, fallow, potatoes, potatoes
*2 - Potatoes, barley, potatoes, potatoes
*3 - Potatoes, barley, barley, potatoes
*4 - Potatoes each year
†Figures are means of 3 replications of 9 Kennebec potato plants/rep. The microplots were infested in 1970 with inoculated seed pieces of Kennebec potato; plant remains were incorporated into the soil each year. Infection was determined by isolation.

Table 2. Recovery of *Verticillium dahliae* from weed hosts grown in infested soil in pot tests

Weed host	No. of plants affected			No. of plants tested
	Root	Stem	Petiole	
Senecio vulgaris L.	19	11	11	20
Chenopodium album L.	29	22	17	29
Thlaspi arvense L.	18	8	7	18
Amaranthus retroflexus L.	22	5	5	22
Solanum nigrum L.	6	1	6	20
Malva pusilla Sm.	2	3	7	22
Malva neglecta Wallr.	2	0	2	16
Capsella bursa-pastoris (L.) Medic.	3	0	0	14
Cichorium intybus L.	1	0	0	2
Portulaca oleracea L.	18	12	sessile	27
Medicago lupulina L.	12	1	0	23

Table 3. Relationship between soil inoculum density and Verticillium wilt index of potatoes grown in microplots in 1975

Plot no.	Inoculum density (no. colonies/100 cm *Datura* root)	Inoculum density (MS/g soil)*	Wilt index†
85	7	5.35	1.88
86	7	5.35	2.00
87	1	1.27	2.38
88	3	2.63	2.13
89	1	1.27	2.14
90	8	6.03	2.13
91	1	1.27	1.83
92	8	6.03	2.75
93	1	1.27	2.13
94	6	4.67	2.75
95	2	1.95	2.13
96	23	16.23	2.88

* Converted according to Evans' regression equation ($Y = 0.59 + 0.68X$).
† Wilt index on a 1-5 scale, 1 = no symptoms, 5 = plant dead.

Number of microsclerotia required for infection

The threshold level of infection (defined as the minimum number of MS recovered per gram of soil in which potatoes became infected) was 1 MS/g soil; at this level the wilt index averaged 1.54 on a scale of 1 to 5 (1 - no symptoms, 5 - plant dead). Isolations from the microplots in 1975 also indicated a threshold level of infection of approximately 1 MS/g, with wilt indices ranging from 1.83 to 2.38 (Table 3).

Discussion

Rotation has been proposed for many years as a control for Verticillium wilt. The results have been variable, generally disappointing, and usually unpredictable. Many of the rotation experiments were done under circumstances in which even with the best of care some mixing of the soil from one plot to another would be expected. While this was impossible under our set up, our results would still indicate that rotation would be ineffective as a means of control.

We suspected that part of the problem of variability in our rotation study could be traced to inadequate weed

control. While every effort was made to eliminate weeds by hand, some were missed or grew for 2-3 weeks before removal. At that time, we did not appreciate the fact that microsclerotia could be produced on the roots of 2-week-old seedlings. When these weeds were pulled many of the smaller roots would be left in the soil and these could have produced sufficient microsclerotia to account for the variability encountered.

In *V. dahliae*, microsclerotia are the principal, if not the only, propagules that persist in the soil and ensure the survival of this fungus. The microsclerotia develop in infected moribund potato or other host tissue, including weeds, above or below ground and are returned to the soil when this tissue is incorporated in the normal tillage procedures (5, 10, 17). The fact that the wilt index did not increase at the same rate as the number of viable MS recovered (Table 3) suggests that 1 MS/g soil would be sufficient to infect almost 100% of the potato crop with almost the same wilt index as from much larger numbers, e.g. 16, of MS/g soil. The percentage of plants infected varied from 84 to 92 in these plots in 1975 and the previous crop history or the previous percentage of plants infected did not appear to have any effect on this.

The variability in the percent infection encountered from year to year in treatment 4 (Table 1) (potatoes each year) suggests that the number of viable microsclerotia necessary to achieve any given percentage of infection varies from one year to the next. Huisman and Ashworth (10) noted a reduction in the MS in cotton soils which was independent of the crop grown and they suggest that soil temperature-moisture interactions could be involved. The fact that the environment affects not only the expression of symptoms but also the survival of the microsclerotia and probably the actual infection of the plant itself must be considered when attempting to correlate number of microsclerotia per gram of soil, percentage of plants infected and severity of symptoms produced. It may very well be that in experiments where rotation gave control the actual number of MS/g soil was low or larger numbers of MS were required for infection and symptoms to be expressed under the particular condition existing at the time. Rotation under these circumstances could well lower the number of MS/g soil down to a point where the effect could be measured on the percentage of plants infected in subsequent crops.

While Verticillium has not been reported from cultivated crucifers in Ontario our results Table 2; penny cress (*Thlaspi arvense* L.) and shepherd's purse (*Capsella bursa-pastoris*) (L.) Medic. suggest that infection of field grown crucifers is a distinct possibility. In addition, field collected *Sisymbrium officinale* (L.) Scop (hedge mustard) from potato soils yielded cultures of *V. dahliae* although the plants were showing no symptoms.

It is the authors' opinion that with MS numbers running as high as 78/g soil in our major potato growing area in Ontario (unpublished results) and with the prevalence of weed hosts in the alternate crops grown, rotation may be valuable as an agronomic practice, but it will do little or nothing to control Verticillium wilt. The particular crop involved in the rotation (as long as it is a non-host) is not as important as the weed control which to be effective must be as close to 100% as possible.

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Peach X-disease in southwestern Ontario

B. N. Dhanvantari[1] *and F. Kappel*[2]

Peach X-disease, first observed in a few peach orchards in southwestern Ontario in 1971, has since spread, affecting more than 50% of the 140 orchards surveyed in Essex County and five out of eight orchards surveyed in Kent and Elgin counties. The survey conducted in 1977 covered nearly 70% of peach trees grown in southwestern Ontario. Typically, less than 2% of the trees were infected in an affected orchard, although there were cases of higher incidence. The disease was found on most of the popular peach cultivars. Mature trees appeared to be more subject to the disease; it was rare among trees newly-planted or under four years old. Infected chokecherry trees, wild reservoir host of the disease agent, were found along the fence lines adjacent to many of the affected orchards.

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La virose X du pêcher, dont on a constaté les premiers cas en 1971 dans quelques vergers du sud-ouest de l'Ontario, s'est répandue depuis, touchant plus de 50 % des 140 vergers étudiés dans le comté d'Essex et cinq vergers sur huit dans les comtés de Kent et d'Elgin. L'étude menée en 1977 portait sur près de 70 % des pêchers cultivés dans le sud-ouest de l'Ontario. En règle générale, moins de 2 % des arbres étaient atteints dans chaque verger infesté, bien qu'on ait relevé des cas de fréquence plus élevée. La virose attaquait la plupart des cultivars couramment en usage. Les arbres plus âgés semblaient plus exposés à la maladie que les arbres nouvellement plantés ou âgés de moins de quatre ans lesquels étaient rarement atteints. Des cerisiers à grappe infectés, hôtes sauvages du virus, poussaient le long des clôtures bordant nombre de vergers infestés.

In Ontario, X-disease in peach (*Prunus persica* (L.) Batsch) was first observed in the Niagara Peninsula in 1941 (1). It was also reported from Michigan in the same year and from Ohio in 1944 (1). By then, it had spread from Massachusetts to Wisconsin and had become a major disease of peach in New York. It had been considered a virus disease but has since been shown to be due to a mycoplasma-like organism (3,5). Sour cherry (*P. cerasus* L.) and sweet cherry (*P. avium* L.) are the other important economic hosts of this disease. Eastern chokecherry (*P. virginiana* L.) is the most important wild reservoir host and the disease is spread from it to stone fruits by a number of leaf hopper species (2,8). A detailed illustrated review of peach X-disease has been published recently (1). X-disease symptoms were observed, for the first time in Essex County, in a few peach orchards, in 1971. Chokecherry trees with the disease symptoms were present in adjacent fence rows. Preliminary surveys from 1971 to 1976 showed that X-disease appeared to be gradually spreading to other orchards in the same area. County-wide surveys were made in Essex and Kent in 1977 to assess its incidence in individual orchards and the extent of its distribution in the main peach growing areas of southwestern Ontario.

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Survey

The X-disease survey was made in July and August when the symptoms were more distinct. Care was taken to distinguish X-disease symptoms from those produced by nitrogen deficiency, *Leucostoma* canker and bacterial spot (*Xanthomonas pruni*). Nitrogen deficiency produced reddish necrotic spots and shot-holes bounded by veins. Although bacterial spot caused shot-holes, chlorosis and defoliation, leaf spots and shot-holes were relatively regular and smaller; the leaves did not curl up and defoliate in the manner of X-disease. Leaves on branches with canker were underdeveloped, chlorotic and drooping. The leaf symptoms used to identify X-disease included blotchy, irregular, water-soaked areas across the veins turning pale yellow and red before leaving shot-holes; leaves curling up and dropping, leaving a large number of leaf scars and only a few terminal leaves. Generally, only part of a tree was thus affected, contrasting with the normal appearance of the rest of the tree in midseason. X-disease is known to cause dieback of branches in the following year but it was hard to distinguish it from that induced by winter injury and canker. Infected chokecherry was recognized by its premature fall coloration contrasting with the surrounding greenery.

The survey covered 140 farms in Essex County, 8 large farms in Kent County and one in Elgin County, altogether comprising nearly 70% of the peach trees grown in southwestern Ontario.

Results

In Essex County, X-disease was found on 76 farms, being about 54% of those surveyed. All of them were

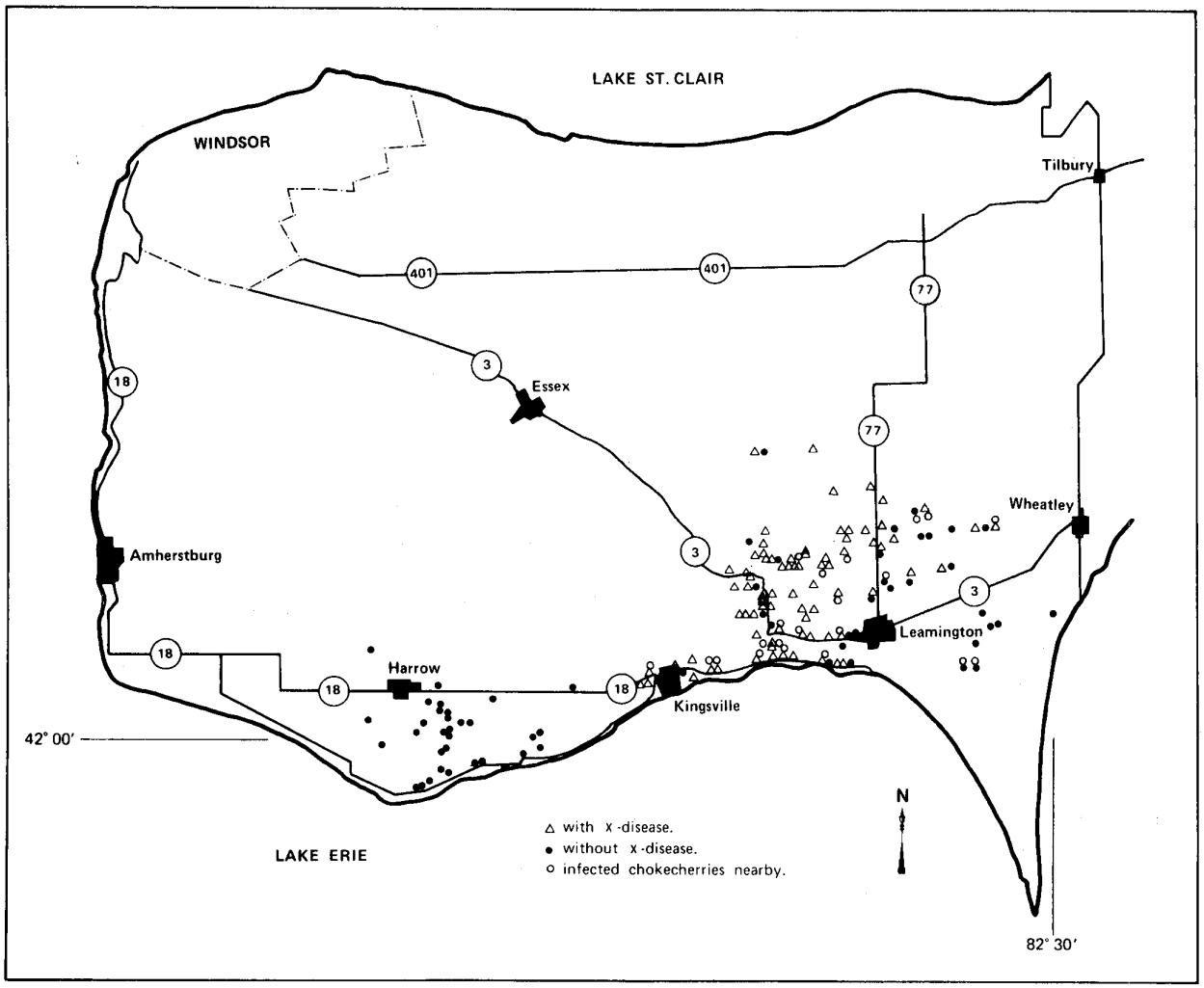

Figure 1. Distribution of peach X-disease in peach orchards of Essex County.

located in the southeastern part of the county between Essex, Kingsville and Wheatley (Fig. 1). As shown in Table 1, the disease incidence was one percent or less on 45 farms, and ranged up to 10% on 28 farms; three farms were severely affected with 13%, 23%, and 67% of the trees infected respectively. X-diseased chokecherry trees were present near, or had been recently removed from 42% of the affected orchards, and apparently healthy chockcherry trees were present near 16 other orchards.

In Kent County, 8 farms with 8845 trees were surveyed. Five farms were affected with the disease incidence ranging up to two percent. The total disease incidence for all the trees was less than one percent. Diseased chokecherry trees were found near three of the affected farms.

In terms of age groups, it was found that the disease incidence was relatively higher among peach trees 4 to 10 years old, and very few of those less than 4 years were affected (Table 2). The total disease incidence was 1.16% in Essex County and 0.91% in Kent County.

X-disease distribution in an affected peach orchard and its relation to diseased chokecherry trees is shown in Figure 2. Generally, the infected peach trees appeared to be mature and in clusters. The distance between affected orchards and infected chokecherry trees in the adjacent fence lines or wood lots varied from 20 to more than 1000 feet. There were also instances where infected chokecherries could not be found. The disease was found in at least 24 peach cultivars, including most of the popular ones (Table 3).

Discussion

Ever since X-disease was first observed in Essex County peach orchards in 1971, an epidemic seemed imminent since it is disseminated by several leaf hopper vector species; coincidentally the persistent contact insecticide DDT had been withdrawn from use on peach in 1969. It

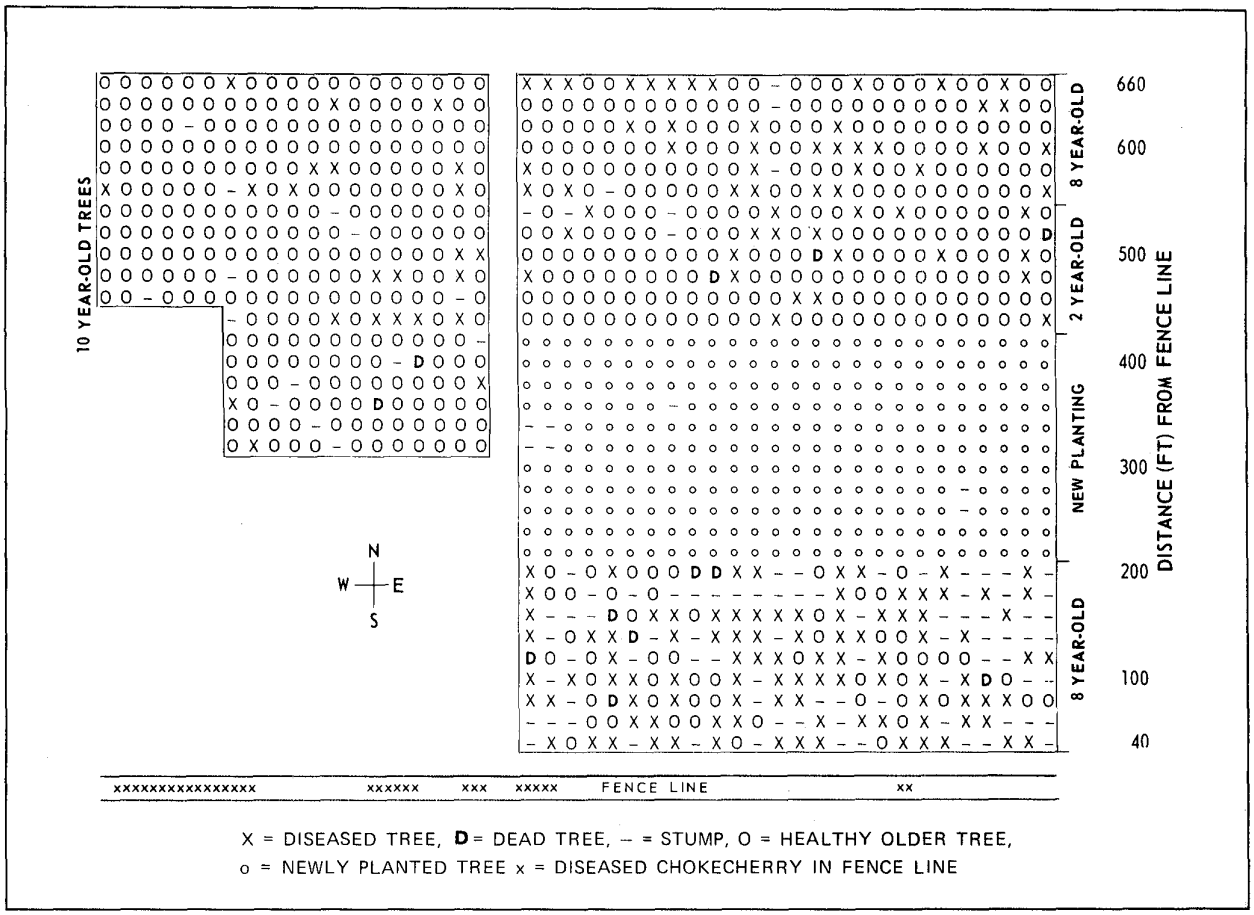

Figure 2 — Distribution of X-disease in an Essex County peach orchard

Table 1. Incidence of X-disease in peach orchards of Essex County fruit farms in 1977

Farms	Number	% of surveyed farms	% of affected farms
Surveyed	140		
With X-disease	76	54.28	
With up to 1% disease incidence	45	32.14	59.21
With 2 to 10% disease incidence	28	20.00	36.84
With >10% disease incidence	3	2.14	3.94
With X-diseased chokecherries	21	22.86	42.10
From which X-diseased chokecherries were recently removed	11		
With healthy chokecherries	16	11.43	

Table 2. Age groups, number and percent of peach trees affected by X-disease in Essex and Kent county fruit farms in 1977

Age (years)	Essex			Kent*			Total % of X-diseased trees
	Trees	X-diseased trees No.	%	Trees	X-diseased trees No.	%	
1-3	17,978	14	0.08	1060	0	0	0.07
4-10	101,454	1429	1.41	6767	81	1.19	1.39
>10	27,647	262	0.95	1018	0	0	
Total	147,079	1705	1.16	8845	81	0.91	1.14 (For 155 924 trees)

*Includes one farm from Elgin County

Table 3. A list of peach cultivars on which X-disease was found in southwestern Ontario in 1977

Babygold 5	Glohaven
Babygold 7	Golden Jubilee
Babygold 8	Harbinger
Canadian Harmony	Harbelle
Candor	Harbrite
Cresthaven	Harken
Earliglo	Loring
Earlired	Madison
Early Elberta	Olinda
Elberta	Redhaven
Envoy	Reliance
Garnet Beauty	Sunhaven

is significant that about the same time there was a resurgence of the disease in the Niagara Peninsula (W.R. Allen and T.R. Davidson, personal communication) and Michigan (A.L. Jones, personal communication) where it continues to be a problem. There is reason to believe that the disease is new to Essex and Kent counties since many of the same orchards had been regularly visited by plant pathologists and extension personnel over the years. X-disease is reported to occur among chokecherries even in remote areas where stone fruits are not grown (1). In Canada, chokecherry is transcontinental in distribution and is commonly found in rich moist soils, in open situations on cleared land bordering wooded areas (4). It is possible, then, to speculate that X-disease spread to southwestern Ontario as a disease of the wild chokecherry and has since been affecting the peach orchards here. Eradication of infected chokecherries has been shown to be of critical importance to the disease control among stone fruits (1). The task requires a great deal of concerted effort on the part of the growers and adjacent woodland owners; and it assumes monumental proportions considering the wide distribution of the eastern chokecherry.

It is intriguing to note that, typically, only 1-2% of peach trees in a given orchard are effected, whereas the disease is epidemic in three orchards. This puzzling situation will only be clarified through definitive studies on the host-range and transmission of X-disease. For the present, however, short-term measures of therapeutic remission of the disease in peaches with a tetracycline antibiotic have been successful (6,7).

Acknowledgements

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