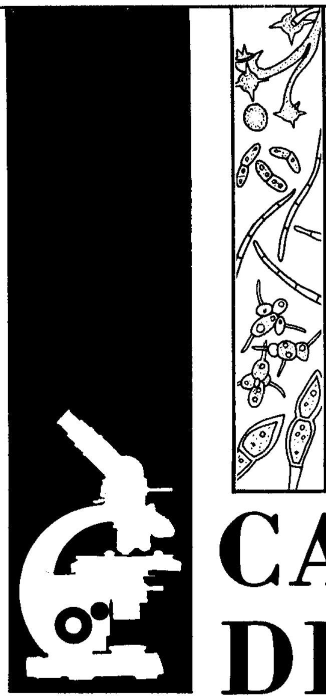
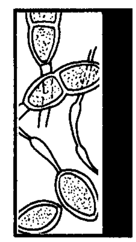
VOL.46, No.1, MARCH, 1966



CANADIAN PLANT DISEASE SURVEY



EDITOR: D.W. CREELMAN

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

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CANADIAN PLANT DISEASE SURVEY

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CONTENTS

"The Canadian Plant Disease Survey is a periodical of information and record on the occurrence and severity of plant diseases in Canada. It will also accept other original information such as the development of methods of investigation and control, including the evaluation of new materials. Review papers and compilations of practical value to phytopathologists will be included from time to time. It will not accept results of original research suitable for publication in more formal scientific journals".

CONTROL OF POTATO WART BY CHEMICAL TREATMENTS[1]

O. A. Olsen[2]

Abstract

Investigation of chemical control of wart in potatoes has been conducted in Newfoundland during the years 1958-62 and 1964-65 inclusive. The soil fungicide Vancide 51 at 400 lb/acre gave best results and was non-phytotoxic. Uracide and CP30249 were effective but somewhat phytotoxic. The herbicide dinoseb gave wart control at rates of application in the range used for weed control. The fumigants Vorlex and Trapex, containing methyl isothiocyanate were effective but strongly phytotoxic after a 4-week period between application and planting.

Introduction

Soil fungicide treatments were successfully used in Pennsylvania to eradicate the potato wart disease, according to Hartman (3). On an acre basis, 2,500-3,000 lb of copper sulfate were disced or dug into cultivated plots; bare areas near walks or foundations received a fall treatment of 10,000 lb in solution, and lawns, flower beds, shrubs and trees were treated with a 10% solution of 40% formaldehyde at the rate of 20 gal/100 ft^2. On some infested areas the soil was sterilized with 5 tons/acre of copper sulfate, followed by 5 tons of lime/acre one year later. Hartman also found that ammonium thiocyanate at 2,500-3,000 lb/acre eradicated the wart organism. Bell (2) had previously noted that ammonium sulfocyanate at 1,200-3,200 lb/acre gave wart control. In a few cases, where 2,000 lb/acre were applied to soil containing sods and trash, wart was not eradicated.

Roach et al. (6) found that the application of sulfur at 10 cwt/acre on sandy soil and 40 cwt/acre on clay give wart-free plots. Roach and Glynne (5) and Roach (4) concluded that the effect was due to acidified thiosulfate formed from sulfur.

Zakopal (7) reported that a 2% solution of the 25% sodium salt of dinitro ortho cresol at 10 liter/m^2 gave some control of wart without phytotoxicity. It has recently been claimed (1) that Nitraphen, a nitrous salt, has given complete control of wart when the soil is treated with a 1.5% solution.

Materials and methods

Studies on the chemical control of wart in naturally-infested field plots have been conducted in Newfoundland since 1958. During that period, 8 soil fungicides, 1 herbicide and 3 fumigants have been tested. Replicated field plots of one 30-ft, or two 15-ft rows side by side, separated by suitable guard rows, were used. The wart-susceptible cultivar, 'Arran Victory', was grown as the test plant in all cases. Soil fungicides were broadcast and dug in to a 3- or 4- inch depth and soil fumigants were injected 7 inches deep, 6 inches apart each way, with a hand applicator. A waiting period of 4 weeks elapsed between application of fumigants and planting to allow dissipation of toxic vapors. Planting was done immediately after the application of soil fungicides. The herbicide dinoseb was applied as a preemergence spray, and was also sprayed on the soil surface and dug in prior to planting.

Results were assessed in several ways. From 1958-1960 inclusive, wart disease indices and numbers of tubers set were used; from 1962-1965, the marketable and total yields and weights of warted cull potatoes were determined. Measuring wart development by a disease index instead of weight of warted cull gives higher readings for wart in the check plots where wart development on each infected tuber is nearly always greater than in treated plots. Weight of warted cull, on the other hand, is a more useful measure for practical purposes.

The soil fungicides used were: calomel (Calogreen 76.5%); bis ethyl xanthogen (Herbisan 58%); chloro (tolylsulfonyl) propionitrile (CP30249, 4 lb/gal); ethylene thiuram monosulfide (Amobam 50%); pentachloronitrobenzene (Terraclor, 75% WP); sodium dimethyldithiocarbamate (Vancide 51, 30%); tetrachlorotetrahydrothiophene dioxide (DAC-649, W50) and uracide (urea formaldehyde 85%). The fumigants were: mylone (Crag Mylone 50 D, Soil Kare 50); methyl isothiocyanate (Trapex 20%); and methyl isothiocyanate - chlorinated C_3 hydrocarbon mixture (Vorlex 100%). The herbicide was dinoseb (Sinox P.E. 3.6 lb/gal).

The wart disease index was obtained by grouping the potato tubers into five classes as follows: 0 = no wart; 1 = one or two small pustules, total diameter less than 1.0 cm; 2 = up to $\frac{1}{4}$ of tuber warted; 3 = $\frac{1}{4}$ to $\frac{1}{2}$ of tuber warted; 4 = $\frac{1}{2}$ to entire tuber involved.

$$\text{Disease Index} = \frac{1\,(\text{No. of class 1 tubers}) + 2\,(\text{No. of class 2 tubers}) + 3\,(\text{No. of class 3 tubers}) + 4\,(\text{No. of class 4 tubers}) \times 100}{\text{Total no. of tubers} \times 4}$$

[1] Contribution No. 10, Experimental Farm, Research Branch, Canada Department of Agriculture, St. John's West, Nfld.

[2] Plant Pathologist.

Table 1. Wart control obtained from soil fungicides.

Soil fungicide	Rate of application	Method of application	Year of test	Disease index	No. of healthy tubers	Market-able yield cwt/acre	Total yield cwt/acre	Warted culls cwt/acre	Total plus culls cwt/acre
Vancide 51	150 lb/acre	Broadcast	1958	* 4.8	193	Yields not measured		Culls not measured	
" "	300 lb/acre	"	"	2.6	207	" "	"	" "	"
" "	400 lb/acre	"	"	1.1	238	" "	"	" "	"
Check			"	14.8	148	" "	"	" "	"
Vancide 51	300 lb/acre	"	1959	5.9	162	" "	"	" "	"
" "	400 lb/acre	"	"	4.6	178	" "	"	" "	"
Check			"	20.5	142	" "	"	" "	"
Vancide 51	200 lb/acre	"	1960	0.8	169	126.0	161.0	" "	"
" "	300 lb/acre	"	"	0.6	170	140.0	177.2	" "	"
" "	400 lb/acre	"	"	0.5	157	135.0	165.4	" "	"
Uracide	150 gal/acre	"	"	2.0	204	182.4	221.7	" "	"
"	300 gal/acre	"	"	1.2	231	159.1	210.2	" "	"
CP30249	240 lb/acre	"	"	0.4	144	115.1	138.0	" "	"
Check			"	3.7	151	119.6	148.8	" "	"
Vancide 51	300 lb/acre	"	1962	(Disease index and number of tubers not determined.)		140.6	248.4	10.2	258.6
" "	400 lb/acre	"	"			156.6	258.7	11.5	270.2
CP30249	120 lb/acre	"	"			135.0	240.2	17.6	257.8
"	240 lb/acre	"	"			67.5	152.6	5.2	157.8
Check			"			103.4	164.8	72.9	237.7

* All figures are the mean of four replicates.

Table 2. Wart control obtained from soil fumigants.

Soil fumigant	Rate of application	Method of application	Year of test	Disease index	No. of healthy tubers 30' row	Market-able yield cwt/acre	Total yield cwt/acre	Warted culls cwt/acre	Total plus culls cwt/acre
Trapex	30 gal/acre	Injected	1960	*0.2	72	24.2	37.9	Culls not measured	
"	80 gal/acre	"	"	0.0	2	1.5	3.0	" " "	
Check			"	3.7	151	119.6	148.8	" " "	
Vorlex	40 gal/acre	"	1962	(Disease index not determined.)		76.6	161.8	1.3	163.1
"	70 gal/acre	"	"			5.6	20.5	0.4	20.9
Check			"			103.4	164.8	72.9	237.7

Table 3. Wart control obtained from the herbicide Dinoseb.

Treatment	Rate of application	Method of application	Year of test	Market-able yield cwt/acre	Total yield cwt/acre	Warted culls cwt/acre	Total plus culls cwt/acre
Dinoseb	5 lb/acre	Pre-emergence spray	1964	*165.6	215.2	13.8	229.0
"	10 lb/acre	" "	"	170.8	218.0	8.2	226.2
"	15 lb/acre	" "	"	148.4	193.4	12.8	206.2
"	30 lb/acre	" "	"	92.2	120.4	5.8	126.2
Check			"	156.8	199.8	31.8	231.6
Dinoseb	5 lb/acre	" "	1965	158.3	233.6	1.1	234.7
"	10 lb/acre	" "	"	179.3	259.1	1.0	260.1
"	20 lb/acre	Pre-plant, dug in	"	64.2	98.4	0.0	98.4
"	40 lb/acre	" " " "	"	27.0	51.4	0.0	51.4
Check			"	50.8	103.0	6.8	109.8

* All figures are the mean of four replicates.

Results and discussion

All of the following materials, at the rates of active ingredient shown, gave a statistically significant degree of control, but not enough for practical application: calomel at 147 lb/acre; Herbisan at 200 lb/acre; Amobam at 240 and 480 lb/acre; Terraclor at 188 and 262 lb/acre; DAC-649 at 100 and 150 lb/acre; and the fumigant mylone at 200, 300, and 400 lb/acre.

The results with Vancide 51, uracide, CP30249, Trapex, Vorlex and dinoseb, which gave a high degree of control, are listed in Tables 1 to 3.

From Table 1, it is evident that very good control of potato wart can be obtained with soil fungicides, but that relatively high rates of application are necessary.

From 1959-1960 inclusive, the wart disease index in the untreated controls varied from 20.5 to 3.7. This fluctuation was due to differences in soil moisture, to which wart development is quite sensitive. There was sufficient rainfall each year to produce a potato crop, but 1960 was too dry for good wart development. 1961 was so dry that wart did not grow enough to give results. In all trials, Vancide 51 gave the best results and had no toxic effects. Uracide is somewhat phytotoxic but the effect is counterbalanced by the nitrogen supplied by this compound. CP30249 was an effective fungicide but was phytotoxic at the 240 lb/acre rate.

The results obtained with Trapex and Vorlex are shown in Table 2. In spite of a four-week period between soil treatment and planting, emergence was prevented or the potatoes were badly stunted. In the surviving plants, wart control was very good. Since Trapex and Vorlex are active in cold soils, it is suggested that fall treatment would allow sufficient time for phytotoxic effects to disappear from the soil and still give effective fumigation.

Dinoseb, in preemergence applications, gave effective control as shown in Table 3. The rates of application required were considerably lower than those of any material previously tested. Poor weed control and rather dry field conditions may explain the low yield from the 1965 check plots. The quantity of warted cull potatoes was also low, but considerably higher than in the treated plots.

Literature cited

1. Anonymous. 1964. Nitraphen - A new chemical to control potato wart disease. Indian Potato Journal 6(2): 107.
2. Bell, R.H. 1935. Further notes on chemical sterilization as a means of eradicating potato wart disease from the soil. Jour. Econ. Ent. 28(3): 519-524.
3. Hartman, R.E. 1955. Potato wart eradication program in Pennsylvania. Amer. Pot. Jour. 32(9): 317-326.
4. Roach, W.A. 1930. Sulphur as a soil fungicide against the potato wart disease organism. Jour. Agr. Sci. 20(1): 74-76. (R. A. M. 9: 468-469. 1930).
5. Roach, W.A., and Mary D. Glynne. 1928. The toxicity of certain sulphur compounds to Synchytrium endobioticum, the fungus causing wart disease of potatoes. Ann. Appl. Biol. 15(2): 168-190.
6. Roach, W.A., Mary D. Glynne, W.B. Brierley, and E.M. Crowther. 1925. Experiments on the control of wart disease of potatoes by soil treatments with particular reference to the use of sulphur. Ann. Appl. Biol. 12: 152-190. (R. A. M. 4: 696-697. 1925).
7. Zakopal, J. 1950. The possibility of soil disinfection against potato wart disease, Synchytrium endobioticum (Schilb.) Perc. with a preparation containing 2,4-dinitro-orthocresol. (Czeck.) Sbor. Ceskoslov. Akad. Zem. 23 (1/2): 132-141. (Biol. Abstracts. 26: 941. 1952).

THE INTRODUCTION OF SOIL-BORNE ONION DISEASES INTO THE BRITISH COLUMBIA INTERIOR

G. E. Woolliams [1]

Abstract

Pink root and basal bulb rot of onion became established in the Okanagan Valley over 40 years ago; onion smut in 1945 at Kelowna; and white rot in 1964 at several locations. Circumstances under which these diseases became established indicate that the pathogens were brought into the Valley on seedling onion plants imported from Washington State. Pink root and basal bulb rot are now widely distributed throughout the irrigated sections of the British Columbia Interior; smut has become established throughout the Kelowna district.

Introduction

For over forty years, commercial onion growers in the semi-arid sections of B.C. have been importing onion seedlings each spring from Walla Walla, Washington. There is considerable evidence that with these plants they have also introduced the pathogens that cause the most serious diseases on onion in this region. These include the Fusarium spp. responsible for pink root, Fusarium oxysporum Schlecht. f. cepae (Hanz.) Snyd. & Hansen causing basal rot, Urocystis magica Pass. (U. cepulae Frost), causing onion smut, and Sclerotium cepivorum Berk., causing white rot.

The importations are fall-sown seedlings of an early-maturing strain of 'Sweet Spanish' onion. These imported plants have had a continuous demand among B. C. growers because under local soil and climatic conditions they grow well and provide an early cash crop normally harvested before the main crop of spring-sown onions is mature.

Pink root and basal bulb rot

There are two types of circumstantial evidence to suggest that the Fusarium spp. causing these diseases were introduced from Washington. Although onions have been grown in the B.C. Interior for over seventy years, pink root and basal rot were first recorded about the time the first importations of seedlings were made. Moreover, a proportion of the Walla Walla seedlings have shown pink root symptoms in each of several years that the author has inspected them, both at the time of importation and in the field on growing plants. The Fusarium type of pink root is due to F. solani (Mart.) Appel & Wr. Basal bulb rot is caused by F. oxysporum Schlecht. f. cepae (Hanz.) Snyd. & Hans. Both of these fungi occur at Walla Walla, Wash. (8) as well as in the Okanagan valley. No attempts have been made in British Columbia to discriminate Fusarium (F. solani) pink root from pink root caused by Pyrenochaeta terrestris (Hans.) Lorenz, J. C. Walker and Larson. Shaw (8) has reported the occurrence of P. terrestris in Washington. This is confirmed by isolation of the fungus by Duran (5) from the roots of onion plants collected at Walla Walla. Thus there are strong grounds for concluding that this fungus was also introduced into the Okanagan valley on imported plants and is responsible for at least part of the pink root disease found in onion crops produced in this area.

Onion smut

The first record of the occurrence of onion smut in B. C. was made in 1947. During that season diseased plants were brought to the Plant Pathology Laboratory at Summerland for diagnosis. The owner of one diseased field reported that he had first observed the disease two years previously on a few plants. Since 1947 the disease has increased in prevalence in infested fields, and has spread to most other onion fields in the Kelowna district.

The following evidence indicates that onion smut was introduced to the Okanagan Valley on plants imported from Washington:

(1) Onion plants infected with smut have been intercepted by Plant Protection officials in shipments from Walla Walla destined for the Kootenay districts of British Columbia. It seems probable that the disease was not detected in plants imported into the Okanagan Valley because they were imported in such very much larger numbers that spot inspections only could be made, and the chances of detecting the disease were thereby reduced. The fact that the first occurrence of the disease was very much limited indicates that soil infestation with the pathogen came from only a few plants.

(2) Cultures of the pathogen secured from smutted plants grown in the Okanagan valley are compatible with cultures made from diseased specimens collected at Walla Walla (5).

(3) Disease control experiments, conducted independently at Kelowna and Walla Walla (6) have provided the same finding that captan is relatively ineffective against the disease, although this fungicide is effective against onion smut in many other regions. In these two districts hexachlorobenzene, one brand only, controls smut very effectively.

These facts suggest that the strain of the fungus in the Okanagan Valley plantings is the same as that in the Walla Walla district.

White rot

Onion white rot was found in the Okanagan Valley for the first time in 1964. Mr. E. M. King of the Horticultural Branch, B. C. Department of Agriculture, had examined fields of fall-planted onions at Walla Walla early in the 1964 season, and had been

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disturbed to learn that the white rot disease occurred extensively in Walla Walla plantings. Surveys were initiated during the summer in Okanagan Valley fields that had received recent shipments of planting stock from Walla Walla, and Mr. King's anxiety proved to be justified. White rot was found on a new farm, developed on virgin land within the last five years. The disease affected approximately 75% of the plants growing in a small section of a 35-acre field of 'Stockton Yellow Globe' onions. These had been fall-seeded in August, 1963, but the immediately preceding crop had been onion plants imported from Walla Walla.

Subsequent 1964 inspections in some Okanagan Valley fields that had been planted that year with seedling plants from Walla Walla disclosed traces of white rot infection in 15 of the 16 fields inspected.

In 1965 the field in which severe infection had been found in 1964 was seeded to alfalfa. However, white rot was found in two other fields on the same farm, in onion plants that had been imported as seedlings from California. Both fields in previous years had been planted with seedlings from Walla Walla. One of these fields had only a trace of infection. The disease was moderate to severe in patches throughout the second field.

Discussion

The accumulated evidence that these important soil-borne onion diseases have been introduced to the Okanagan Valley in seedling plants is a significant demonstration of the risks involved in wholesale movement of living plants from one region to another. Even the careful inspection given by regulatory officials during transshipment has not proved effective in preventing entry of pathogens causing these diseases. Once introduced they add seriously to the problems of onion culture.

Since their introduction, the fusaria causing pink root and basal bulb rot have become widely distributed throughout the irrigated sections of the B. C. Interior. They occur in most onion fields and often cause considerable losses. Basal bulb rot has become considerably more destructive in recent years, with the replacement of open pollinated varieties by hybrid varieties that are much more seriously affected. The incidence of the disease is as high as 20 - 50% in some of the hybrid varieties being grown.

Within the same region the more recently introduced onion smut pathogen has been restricted to one major onion-growing district, that surrounding Kelowna. It has become distributed generally through the onion fields of this district, and is quite destructive if not adequately controlled by seed treatment. There appears to be little hope of preventing its eventual spread to other onion growing districts of the region.

Although an eradication programme for white rot has been considered, it appears unlikely that eradication can be effected, especially because the acreages of many of the infested farms are too small to encourage or practise rotations of sufficient duration. Some hope can be derived from the reports that minor outbreaks of the disease in Canada have been observed at Steveston, B. C. (1) and Thetford Mines, Quebec (3), on garlic, and at Winnipeg, Man., (2) and Sherrington, Que., (4) on onion, and that the disease has not persisted in these districts. However, the history of white rot in the Walla Walla district suggests that the disease can become firmly established in the Okanagan Valley. It was first found in Walla Walla plantings in 1951 (6) and has increased steadily in importance until it has become a limiting factor in onion production there. Soil, climate, and cultural methods in the Okanagan Valley are very similar to those at Walla Walla.

The establishment of seedling propagation plantings in B. C. early in the history of the Province's onion industry would have minimized the chances of introducing these pathogens. Although climatic and economic factors discouraged domestic propagation, the evidence of disease introduction now accumulated indicates that more has been lost than gained from the practice of importing young plants. It provides strong justification for initiating local production of this planting stock.

Literature cited

1. Conners, I. L. and D. B. O. Savile. 1952. Canadian Plant Disease Survey 31: 50.
2. Creelman, D. W. 1960. Canadian Plant Disease Survey 39: 50.
3. Creelman, D. W. 1961. Canadian Plant Disease Survey 41 (2): 69.
5. Duran, Ruben. 1966. Correspondence.
4. Creelman, D. W. 1963. Canadian Plant Disease Survey 43 (3): 90.
6. Duran, Ruben and George W. Fischer. 1959. The efficacy and limitations of hexachlorobenzene for the control of onion smut. Plant Disease Reptr. 43 (8): 880-888.
7. Locke, Seth Barton. 1965. An improved laboratory assay for testing effectiveness of soil chemicals in preventing white rot of onion. Plant Disease Reptr. 49 (6): 546-549.
8. Shaw, Charles Gardner. 1958. Host fungus index for the Pacific Northwest I. Hosts. Washington Agric. Expt. Sta. Circular 335.

AN APPLE FRUIT DEFORMITY OF UNKNOWN ETIOLOGY[1]

T. R. Davidson and W. R. Allen [2]

Fluted, somewhat egg-shaped fruits of the 'McIntosh' variety were sent from Collingwood, Ontario to the Research Station at St. Catharines in August. Fluting was most pronounced at the blossom end but depressed lines often extended more than half way up the fruits (Fig. 1a). The stem cavity was full or even built-up rather than depressed. The similarity to 'flute fruit' (1) was so obvious that the orchard was visited in September.

Fluted and otherwise deformed fruits were observed on 6 varieties. 'Golden Delicious' was the least affected and showed only slight distortion, mostly at the blossom end and with the stem end normal (Fig. 1b). 'McIntosh', 'Cortland', and 'Jonadell' were all affected to about the same degree with severe distortion at the blossom end (Fig. 1c). Fluting extended 2/3 or more towards the stem end. Some 'McIntosh' fruits were flattened laterally and had a full stem cavity, thus making them somewhat egg-shaped. 'Red Delicious' showed pronounced fluting and distortion at the blossom end. Also, the stem cavity was often filled with a proliferation of the stem into a thick, knobby structure. 'Cox's Orange Pippin' was the most severely affected variety. Fruits were fluted at the blossom end and the stem ends of many were drawn out into short, thick, somewhat curved, pear-like necks. The stems were thickened as in 'Red Delicious' (Fig. 1d).

Some of the varieties listed were examined on two or even all three of the common rootstock types, standard, dwarfing and semi-dwarfing. Distorted fruits were found on all types. The condition, therefore, could not be correlated with rootstock. Also, the occurrence of the condition on nearly every tree of all varieties, on all rootstocks, ruled out the probability of a virus as being the cause. Spray injury was considered as a possible cause but the condition was observed in two adjacent orchards where different fungicides had been used. Also, one grower had used a chemical thinner but the other had not. The topography of these orchards was gently rolling, consisting of knolls with long slopes in many directions forming irregular, broad, shallow valleys. It was not possible by observation to determine where frost pockets might develop or what the air drainage might be. However, trees on the higher land were the least affected while those on the lower slopes carried the most deformed fruits.

Very similar conditions have been reported from British Columbia, New Brunswick and Nova Scotia this year. Affected orchards in British Columbia have a history of frost injury[a]. Damaged fruits from New Brunswick appeared similar to those from Collingwood. Deformed 'McIntosh' fruits with full stem cavities and an elongate pear-shape have been reported from Nova Scotia[b]. In Nova Scotia only the 'king' fruits were affected. No such correlation existed at Collingwood as all fruits in some clusters were distorted. The Nova Scotia report, which refers only to 'McIntosh', states that 19% of the crop was affected. In the orchards observed at Collingwood at least 50% of the 'Red Delicious' and 'Cox's Orange Pippin' fruits were deformed. Other varieties were affected to a lesser degree, probably in the 15 to 20% range. However, this condition was not reported from any other part of Ontario so the overall damage was minimal.

The observers were not able to determine the exact cause of this condition. However, the symptoms certainly suggest some type of hormone imbalance. Low-temperature injury at some very early stage in the development of the fruits, perhaps even in the bud stage, seems to be a plausible interpretation. Such injury could conceivably cause a disturbance in some hormone system and produce the symptoms seen. The differences in symptoms as seen in various regions could be caused by slight differences in the physiological time at which injury occurred.

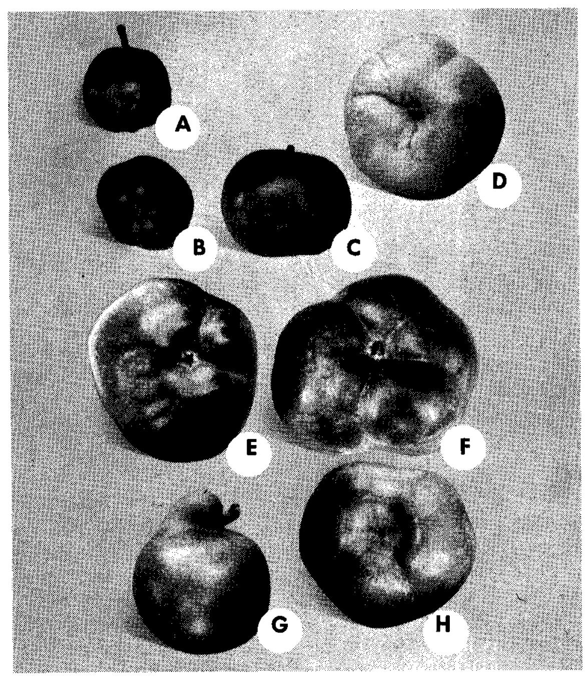

Fig. 1. A, B, 'Fluted McIntosh fruits from Collingwood, with normal fruit, C. D, Distorted Golden Delicious fruits. E, Distorted McIntosh fruits. F. Distorted Cortland fruits. G, Distorted Cox's Orange Pippin fruits. H, Distorted Jonadell fruits.

a Personal communication with Dr. M. F. Welsh.

b Dr. R. P. Longley, Press releases, Can. Dept. of Agriculture, Oct. 1965.

Literature cited

1. Welsh, M. F., and F. W. L. Keane. 1961. Diseases of apple in British Columbia that are caused by viruses or have characteristics of virus diseases. Can. Plant Dis. Surv. 41: 123-147

1 Contribution No. 117 from the Research Station, Canada Department of Agriculture, Vineland Station, Ontario.

2 Plant Pathologists.

WHEAT STREAK MOSAIC VIRUS IN CORN IN ONTARIO

Y. C. Paliwal, J. T. Slykhuis [1] and R. E. Wall [2]

During a survey in southwestern Ontario in August 1965, mosaic symptoms were observed on several corn (Zea mays L.) plants in each of several fields in Essex and Kent counties. At one location in Essex County, the incidence of plants with mosaic symptoms in two fields varied from 5% to more than 10%. Although some plants had only mild mosaic symptoms and were not stunted, others had pronounced yellowish streaks or chlorotic dots and dashes interspersed with oval to elliptical chlorotic rings on the leaves (Fig. 1) and were moderately stunted.

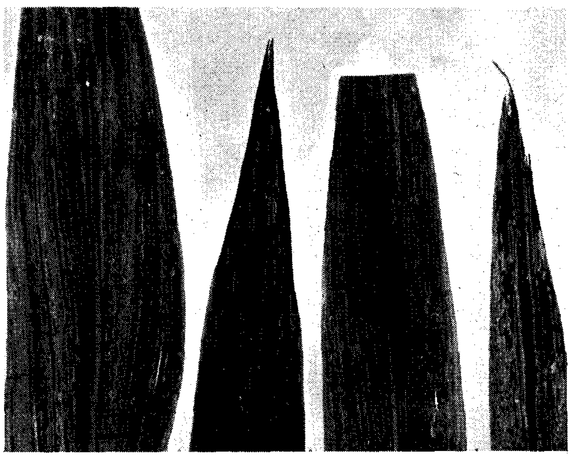

Figure 1. Symptoms caused by wheat streak mosaic virus on corn from Essex County, Ontario.

A virus was transmitted manually from mosaic-diseased corn plants to corn (inbred HY, Single Cross CH159 x CH3, and 'North Star' sweet corn) as well as to 'Kent' wheat. In tube precipitin and precipitin ring tests, the virus reacted positively with an antiserum prepared against wheat streak mosaic virus (WSMV) from Alberta. It was also transmitted by Aceria tulipae (K.) the vector of WSMV. The same virus was isolated from Setaria viridis (L.) Beauv. and Echinochloa crus-galli L. from the same corn fields. Although several species of perennial grasses in and around these corn fields were also tested, the virus was not detected in any. According to the farmer, the corn fields concerned had been planted near winter wheat. Probably the wheat harbored WSMV over the winter and served as the source of infection in the corn planted nearby.

Since red striping of pericarp of corn was widespread in southwestern Ontario in 1964 (1, 5) observations and tests were made to determine if the presence of WSMV was correlated with the presence of this condition. WSMV was isolated from immature kernels with red striped pericarp as well as from leaves of plants with mosaic symptoms. However, WSMV was also isolated from immature kernels without red striped pericarp from plants with mosaic symptoms on leaves. Red striped pericarp occurred in corn in many locations where WSMV was not found and on plants from which WSMV could not be isolated. Therefore, there was no indication that WSMV was a cause of the red striped pericarp.

WSMV, which is common on wheat in Alberta, was not detected on wheat in Ontario until 1964 (4), but was again found on several plants in Middlesex County in 1965. Although WSMV has been found in corn in Idaho (2) and Nebraska (3) and probably also in Ohio (6), this is the first record of its occurrence in corn in the field in Canada.

Agropyron mosaic virus (AMV) was suspected to be a cause of mild mosaic symptoms on corn and wild annual grasses closely associated with diseased Agropyron repens L. Although AMV was isolated from the A. repens and from Setaria glauca (L.) Beauv., a mild strain of WSMV but no AMV was isolated from the corn.

Wheat spot mosaic virus (WSpMV), which occurs associated with WSMV in Alberta and is transmitted by A. tulipae, the vector of WSMV, was readily transmitted to a number of inbred lines of corn. Although WSpMV has not been recognized in southwestern Ontario, it should not be ignored as a possible cause of leaf spotting of corn in areas where WSMV occurs.

Literature cited

1. Cuddy. T.F. and V.R. Wallen. 1965. Seed borne diseases of corn in 1964 and their effect on germination. Can. Plant Disease Survey 45: 33-34.
2. Finley, A. M. 1957. Wheat streak mosaic, a disease of sweet corn in Idaho. Plant Disease Reptr. 41: 589-591.
3. How, Shao Chung. 1963. Wheat streak mosaic virus on corn in Nebraska. Phytopathology 53: 279-280.
4. Slykhuis, J. T. 1964. Noteworthy and new records of grass viruses in Canada. Can. Plant Disease Survey 44: 242-243.
5. Wall, R. E. and C. G. Mortimore. 1965. Red striped pericarp of corn. Can. Plant Disease Survey 45: 92.
6. Williams, E., L.J. Alexander and H.A. Runnels. 1965. A virus isolated from red streaked corn grain. Phytopathology 55: 1083 (Abstr.).

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CAS INUSITÉ DE POURRITURE SÈCHE DE LA POMME DE TERRE AU QUÉBEC

H. Généreux et C. Aubé [1]

Résumé

Une pourriture sèche a été observée sur le semis 'B3048-18' de pomme de terre à l'automne de 1964. Des organismes tels que Cephalosporium acremonium Corda, Rhizoctonia solani Kühn, Fusarium oxysporum Schlecht., et Fusarium avenaceum (Fries) Sacc. ont été isolés des tubercules malades. Les essais de pathogénicité faits en serre et en champ on donné des résultats négatifs sauf dans certains cas, en serre, où les divers organismes ont été utilisés en mélange.

Abstract

A dry tuber rot of the potato seedling 'B3048-18' was noticed in the fall of 1964 at digging time. Organisms like Cephalosporium acremonium Corda, Rhizoctonia solani Kühn, Fusarium oxysporum Schlecht., and Fusarium avenaceum (Fries) Sacc. were isolated from diseased tubers. Pathogenicity tests carried out in the field and greenhouse gave negative results, except in a few cases, in the greenhouse, where the organisms were inoculated in various combinations.

Introduction

Chaque année, diverses pourritures s'attaquent aux pommes de terre soit à la récolte ou en entrepôt. La pourriture sèche des tubercules a été observée en entrepôt dans plusieurs pays, notamment au Canada (1), aux Etats-Unis (2), et en Grande Bretagne (3). Cette pourriture est surtout causée par Fusarium sambucinum Fckl. f. 6 Wr. et Fusarium coeruleum (Lib.) Sacc.

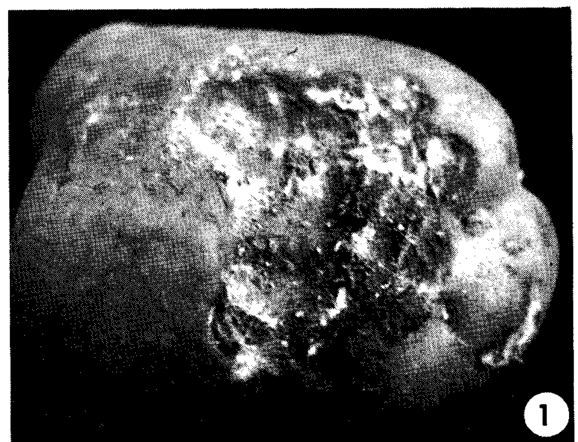
Au Québec, cette maladie cause parfois des dommages à la variété Keswick par suite de blessures mécaniques. A l'automne de 1964, on observa de la pourriture sur les tubercules du semis 'B3048-18', issu du Maine et cultivé à La Pocatière sur sol sablonneux. En effet, 25% des tubercules montraient des symptômes de pourriture sèche à la récolte.

Cette étude a pour but de décrire les symptômes de la maladie et de fournir le résultat des essais de pathogénicité poursuivis en serre et en champ.

Description des symptômes

A la surface des tubercules (Fig. 1), la maladie se reconnaît à des taches de teinte brunâtre, d'étendue et de forme variables, marquées parfois de zones déprimées d'environ $\frac{1}{4}$ de pouce de profondeur. A l'emplacement de ces taches, un mycelium blanc se développe plus tard en entrepôt.

Une coupe des tubercules (Fig. 2), vis-à-vis les taches montre une zone corticale atteinte de pourriture sèche de couleur variant du brun au blanc. On remarque, en dessous de la chair desséchée, une zone molle noirâtre d'apparence aqueuse.



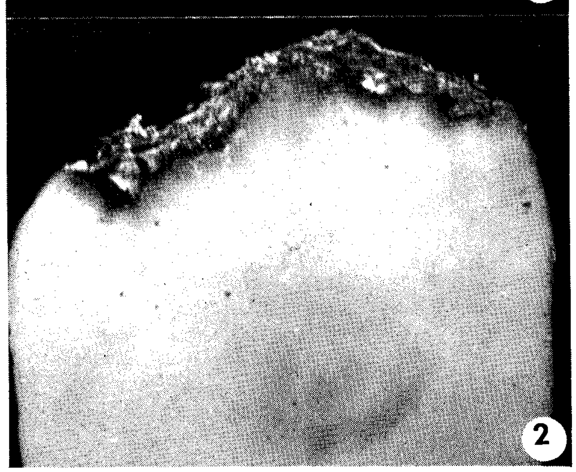


Fig. 1. Surface d'un tubercule atteint de pourriture sèche.

Fig. 2. Coupe d'un tubercule dont la zone corticale est affectée de pourriture sèche.

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Isolation des microorganismes

L'isolation des champignons de tubercules malades a été faite à partir de zones desséchées, puis, des parties adjacentes d'apparence brunâtre. Les tissus malades ont été sectionnés en morceaux de $\frac{1}{4}$ de pouce, désinfectés durant 2 minutes dans une solution à 3% d'hypochlorite de sodium et déposés dans des boîtes de Petri sur de la gélose au pommes de terre enrichie de sucrose (2%). Ainsi, cent boîtes de Petri ont été préparées le 8 juin 1965 et les divers champignons étaient repiqués, dès leur croissance, sept jours plus tard sur un milieu de culture identique.

Quatre champignons ont été isolés et identifiés comme suit: Cephalosporium acremonium Corda, Rhizoctonia solani Kühn, Fusarium oxysporum Schlecht., et Fusarium avenaceum (Fries) Sacc.

Tests de pathogénicité

Des essais de pathogénicité ont été poursuivis en 1965 afin de déterminer la cause de cette pourriture sèche. Des fragments de la variété 'Montagne Verte' et du semis 'B3048-18' ont été plantés en serre et en champ après trempage durant deux minutes dans une suspension d'inoculum (1000 spores/cc) de chaque isolat ou en combinaison. Une fois inoculés, les fragments ont été plantés, soit en serre à raison d'un fragment par pot, soit en champ après avoir été déposés dans le sillon.

Résultats

A l'automne de 1965, aucun des tubercules inoculés et plantés en plein champ n'a causé de symptômes de pourriture sèche à la récolte. En serre, chaque tubercule a été examiné afin de déceler la présence de pourriture ou de taches brunâtres en surface. Chaque isolat des quatre champignons, inoculé individuellement, n'a causé aucun symptôme. Par contre, divers mélanges d'isolats ont donné des résultats positifs, soit: C. acremonium en mélange avec les trois autre organismes; C. acremonium mélangé à R. solani et F. oxysporum; C. acremonium combiné avec R. solani et F. avenaceum et enfin, R. solani en mélange avec F. oxysporum et F. avenaceum. Dans chaque cas, un tubercule a montré des symptômes de la maladie. Les organismes inoculés ont pu être réisolés des tubercules malades.

Des tubercules du semis 'B3048-18', gravement atteints de pourriture sèche, et, plantés en serre, ont donné une récolte exempte de maladie. Des tubercules sains du même semis, plantés en champ en 1965, ont produit une récolte saine.

Discussion

L'isolation de R. solani, C. acremonium, F. oxysporum et F. avenaceum de tubercules atteints de pourriture sèche apparait assez inusitée pour une telle maladie. Par contre, ce phénomène peut s'expliquer du fait que ces organismes sont très communs dans le sol.

Les essais de pathogénicité faits en serre et en champ ont démontré que les quatre organismes, pris isolément, n'ont pu causer la maladie. Ces résultats paraissent décevants du point de vue pathologique; par contre, ils nous permettent de postuler que cette maladie pourrait se manifester occasionnellement à la faveur de conditions écologiques spéciales, Ces facteurs sont sans doute importants dans le comportement de cette maladie, car la récolte issue du semis 'B3048-18' était saine en 1965 alors qu'elle était affectée dans une proportion de 25% en 1964. Comme les températures moyennes du sol et de l'air ambiant étaient quasi identiques au cours des deux saisons de végétation, il faudrait attribuer l'apparition de la maladie au taux de précipitation et au pourcentage d'humidité du sol qui a prévalu en 1964. Au cours de cette saison, la précipitation fut supérieure à la normale en juin et juillet, mais inférieure en août et septembre alors qu'en 1965, la précipitation était inférieure en juin, juillet et août et supérieure en septembre. D'autre part, le pourcentage moyen d'humidité du sol en 1964 était inférieur à celui de 1965.

Les rares cas de pourriture observés au cours des essais en serres indiquent que les quatre organismes, pris isolément, n'ont causé aucun symptôme et qu'une fois combinés, la maladie est apparue. Ces résultats indiqueraient que des organismes auraient un effet synergitique. Nonobstant les résultats obtenus dans cette étude, nous considérons que le semis 'B3048-18' devrait être éprouvé pour sa résistance au pourridié fusarien.

Bibliographie

1. Robinson, D.B. and G.W. Ayers. 1953. Fusarium storage rot of potatoes in Prince Edward Island. Can. Jour. Agric. Sci. 33:566-571.
2. Cassell, R.C. 1945. Fusarium rots and storage diseases of potatoes in New York. Plant Disease Reptr. 29: 337-342.
3. McKee, R.K. 1952. Dry rot diseases of the potato. 11. Fungi causing dry rot of seed potato in Britain. Ann. Appl. Biol. 29: 38-43.

PATHOGENICITY OF FUSARIUM SPECIES FROM ALSIKE CLOVER

Claude Aubé [1]

Abstract

Three pathogenic species of Fusarium were obtained from alsike clover affected by root rot. Of these, F. oxysporum Schlecht. appeared most commonly followed by F. culmorum (W. G. Sm.) Sacc. and F. avenaceum (Fr.) Sacc. On the other hand, their pathogenicity was not related to their frequency since F. avenaceum was more virulent to alfalfa and alsike clover than the other two species isolated, and F. culmorum was the most virulent on ladino clover. Red clover was relatively non-susceptible to the Fusarium species. The significance of the disease distribution in Quebec is discussed and its occurrence in fields planted with red clover shows that it should receive more attention with respect to the competition of plant species. The outbreak of root rot in Quebec is significant if alsike is to be kept in the mixture with timothy and red clover. The need for further investigations along this line is suggested.

Résumé

Trois espèces de Fusarium pathogènes ont été isolés de plants de trèfle alsike malades. F. oxysporum Schlecht. a été isolé le plus souvent, suivi de F. culmorum (W. G. Sm.) Sacc. et de F. avenaceum (Fr.) Sacc. Cependant, la pathogénicité de ces isolats fut sans rapport avec leur fréquence, i. e., F. avenaceum fut plus pathogène à la luzerne et au trèfle alsike que les deux autre espèces, tandis que F. culmorum le fut plus au trèfle ladino. Le trèfle rouge semblait, par contre, résistant au Fusarium isolés du trèfle alsike. L'auteur traite de l'importance de la distribution de cette maladie et émet l'hypothèse que sa présence dans les champs de trèfle rouge peut être due à la compétition qui pourrait exister entre celui-ci et le trèfle alsike. L'éclosion de cette maladie au Québec est importante si on continue de semer cette plante en mélange avec le mil et le trèfle rouge. L'auteur suggère donc que l'on devrait porter plus d'attention à ce problème.

Introduction

Alsike clover, Trifolium hybridum L. is an important forage legume in Quebec, although its usefulness is limited by a lack of persistance of stands. This plant is commonly sown in mixture with red clover and timothy in the proportions of 8 lb timothy, 5 lb red clover, and 2 lb alsike clover per acre. During a disease survey made throughout the province of Quebec in the summer of 1965, alsike clover plants were observed to be dying following attack by Fusarium spp. Specimens of wilted plants were collected from 23 farm fields. Diseased and dead plants have been found in most of the regions where alsike clover is grown. This disease of alsike clover has not heretofore been reported in Canada. This paper presents the results of the survey and of pathogenicity tests of the isolates on forage legumes.

Materials and methods

Isolates of Fusarium were identified according to Gordon's system (3). Eight percent of the isolates (3 isolates) were identified as Fusarium avenaceum (Fr.) Sacc., 19% (8 isolates) as Fusarium culmorum (W. G. Sm.) Sacc., and 73% (26 isolates) as Fusarium oxysporum Schlecht. All the isolates were kept on potato sucrose agar.

The legumes studied in experiments on the host range and pathogenicity included alfalfa (Medicago sativa L. var. 'Vernal'), alsike clover (Trifolium repens L. var. 'Pilgrim'), and red clover (Trifolium pratense L. var. 'Dollard'). Twenty plants of each species were inoculated with each isolate of Fusarium.

The Fusarium isolates were grown on a sterilized 3:1 mixture of soil and sand plus 10% by weight of corn meal, in Erlenmeyer flasks. They were allowed to grow at room temperature for two weeks before being used to inoculate the plants. Sterile distilled water was added, and the cultures were gently shaken. The resulting suspension contained spores, some mycelial fragments, and a few soil particles.

Inoculation consisted of dipping the roots of the seedlings for 10 minutes in the spore suspension containing approximately 100,000 cells per ml. All the experiments were terminated 60 days after transplanting.

Method of assessing plant damage

The disease severity rating was based on the damage caused to tap root and secondary roots and recorded as follows: 0 = no root discoloration, no disease in this case; 1 = trace of root browning, mostly on secondary roots; 2 = moderate root browning, considerable necrosis on secondary roots; 3 = severe rotting of secondary and tap roots; 4 = severe rotting of the entire root system; 5 = death of the plant. The disease rating presented in Table 1 is the mean rating of 20 plants per isolate, i. e. these data represent the mean disease rating of 60 plants in the case of F. avenaceum, 160 plants in the case of F. culmorum, and 520 plants in the case of F. oxysporum. The percent plants killed in the final recording is also based on the same number of plants.

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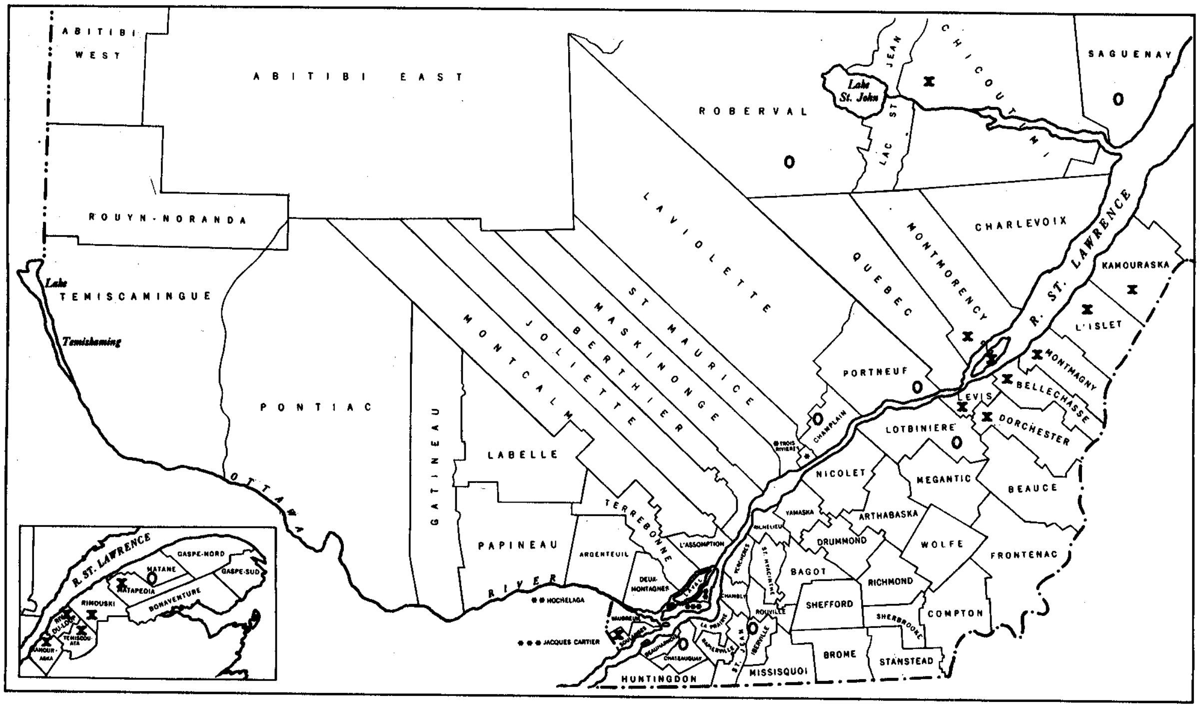

Figure 1. Map of the province of Quebec showing the distribution by counties of alsike clover and its disease caused by *Fusarium* spp.

O: alsike clover not attacked by *Fusarium* spp.
X: alsike clover attacked by *Fusarium* spp.

Results

Figure 1 shows that alsike clover plants attacked by Fusarium spp. were found only from Levis to Bonaventure counties on the south shore of the St. Lawrence River and in two other counties, Chicoutimi and Montmorency on the north shore. In Montmorency the disease was found on Orleans Island.

The results presented in Table 1 indicate that the four plant species so far tested show varying degrees of susceptibility to attack by Fusarium spp. F. avenaceum is the most pathogenic to alfalfa followed by F. culmorum and F. oxysporum in that order. Alsike clover is more susceptible to F. avenaceum than to F. culmorum and F. oxysporum. Although the data obtained with F. culmorum and F. oxysporum do not lend themselves to statistical analysis, the differences do not appear to be particularly significant. Ladino clover is quite susceptible to attack by F. culmorum followed in that respect by F. avenaceum and F. oxysporum. Whereas no plants of red clover were killed by any of the Fusarium spp. tested the disease intensity was higher following inoculation with F. avenaceum than with the other two Fusarium spp.

Discussion

Although root rot ranks among the most important diseases of forage legumes, the Fusarium spp. inciting it are generally considered as weak pathogens. They cause little damage to vigorously growing plants, but are extremely destructive to plants lacking vigor. Under field conditions plants become predisposed to attack in many ways. In Quebec, root reserves are largely consumed during the long winter dormancy, so that the plants are less able to resist attack. In forage legume growing areas, fungus pathogens, moisture, drought, and most important, excessive grazing in the fall, predispose plants to attack. The Fusarium spp. were isolated from alsike clover collected in regions where the rainfall and consequently the soil moisture is relatively high.

Table 1. Virulence of three Fusarium isolates on four species of legume seedlings as indicated by disease severity ratings and percent plants killed.

Isolate and plants tested	Disease severity rating	Percent plants killed
F. avenaceum		
Alfalfa	3.8	76.2
Alsike clover	2.9	55.5
Ladino clover	1.4	13.3
Red clover	1.3	0.0
F. culmorum		
Alfalfa	2.4	41.1
Alsike clover	2.0	31.1
Ladino clover	2.3	40.0
Red clover	0.4	0.0
F. oxysporum		
Alfalfa	1.8	16.2
Alsike clover	2.1	33.8
Ladino clover	0.7	8.7
Red clover	0.6	0.0
Check		
Alfalfa	0.8	0.0
Alsike clover	0.6	0.0
Ladino clover	0.2	0.0
Red clover	0.2	0.0

The distribution of the disease on alsike clover is very interesting since it was found only in an area extending from Levis to Bonaventure and in Chicoutimi. Generally, diseases caused by Fusarium are classed as high soil temperature diseases because the pathogen is most active during the warmest weeks of the growing season. In the regions where the disease was noticed, the temperature is relatively low as compared to that of the Montreal area where the disease was not found. On the other hand, the rainfall is quite high in the Lower St. Lawrence and in the Lake St. John areas where the disease was observed; we would be inclined to think that this factor might have contributed to the development of the disease if we consider the heavy incidence of alfalfa root rot caused by F. avenaceum in 1964 when the soil moisture was very high (1). Since alsike clover is sown in mixture with timothy and red clover, could these two plants weaken alsike clover through competition, rendering it more susceptible to attack by microorganisms? We are inclined to think so since agronomists generally contend that it is difficult to keep alsike clover stands for many years in Quebec.

Fusarium isolates from alsike clover differed in virulence; Chi (2), using Fusarium isolates from red clover has recorded similar findings. On the average, of all the isolates tested, F. avenaceum was the most pathogenic to alfalfa and alsike clover, and F. culmorum the most pathogenic to ladino clover. Furthermore, the isolates of F. oxysporum, which were the most numerous, were less pathogenic to alsike than the isolates of F. avenaceum and had about the same virulence as F. culmorum. A singular lack of specificity was shown by the isolates of Fusarium obtained from alsike clover since they attack roots of M. sativa, T. repens and T. pratense. However, the present results indicate that the use of red clover in mixture with alsike clover may have certain limitations although the strains of Fusarium attacking alsike clover do not appear very virulent to red clover in the present studies. These results open up a field for further investigations of the competition of the two clover species with the emphasis on red clover weakening alsike clover.

The discovery of Fusarium spp. on alsike clover in eastern Quebec may mean that the disease has been missed in earlier surveys since it has not been reported previously. However, if that plant is to be kept in our mixtures in the future, it is urgent to look for varieties resistant to this disease. Furthermore, if alsike clover proves to be unsuitable to our regions due to climatic factors governing the occurrence of this disease, is it wise to retain it any longer in our forage crop?

Literature cited

1. Aubé, C. 1965. Studies on the biology of Verticillium and Fusarium species and effects of environment on development of alfalfa wilt and root rot. XIII. Fusarium rot of alfalfa. Ph. D. Thesis, McGill University, Montreal, Quebec.
2. Chi, C. C. 1965. Pathogenicity of Fusarium species from red clover. Can. Plant Dis. Surv. 45: 3-7.
3. Gordon, W. L. 1952. The occurrence of Fusarium species in Canada. II. Prevalence and taxonomy of Fusarium species in cereal seed. Can. J. Botany 30: 209-251.

TWO MECHANICALLY TRANSMISSIBLE VIRUSES IN CLOVER IN ALBERTA

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Abstract

Two viruses affecting clover in Alberta were tentatively identified as bean yellow mosaic virus and pea streak virus. They were found causing loss in clover breeding stocks, but they did not reduce winter hardiness. Pea streak virus was found to be widely distributed in the clover growing area, but neither virus appears to cause economic loss to commercial production at this time.

Introduction

Economic losses in clovers due to viruses have been reported throughout the world. In 1963 serious damage to seed production, presumably due to virus infection was noted in the field and greenhouse at Lacombe but symptoms were not widespread in commercial fields that year. In 1964 and 1965 surveys were conducted through central Alberta to determine the extent and distribution of viruses infecting clover. This report summarizes a study of the effect of viruses on overwintering of clovers and assesses their economic significance.

Materials and methods

Hosts listed in Table 1 were used to identify the viruses. The length and shape of the virus particles were determined by Dr. M.J. Pratt, C.D.A., Research Station, Vancouver. Infected plant material collected during surveys were inoculated into Chenopodium amaranticolor, Gomphrena globosa, and Vicia faba. Inoculum was prepared by grinding infected plant material in mortars with the addition of distilled water. The juice was then rubbed on the leaves of test plants, which had been dusted with carborundum or celite, with cotton pads. Symptoms on these three test plants and on clovers were considered sufficient for survey identification.

In the spaced-planted (2 x 3 ft) clover nursery, naturally-infected and healthy plants were labelled late in the fall of 1963 and 1964. The following spring the numbers of dead and live plants were recorded for the healthy and virus-infected groups.

Results and discussion

Two viruses were tentatively identified as bean yellow mosaic virus (BYMV), and pea streak virus (PSV) on the basis of host reaction and particle size, (BYMV - 7500 A, PSV - 6200 A). Inclusion bodies as described by McWhorter (4) were observed in Vicia faba inoculated with BYMV. The identifications were not verified by serological studies. Both viruses were found in red and alsike clovers (Trifolium pratense and T. hybridum). In addition, unidentified symptoms were occasionally found in white clover (T. repens), and sweet clovers (Melitolus officinalis and M. alba). The variability of the symptoms in red clover reported by Diachun and Henson (1) was confirmed (Fig. 1 - A). Clover plants infected with PSV showed very faint chlorotic leaf streaks (Fig. 1 - C). Symptoms were found both early and late in the season and appeared to be latent during mid-season.

Since it has been shown that some viruses reduce winter hardiness (2, 3), a total of 2275 plants in 1963 - 1964 and 1180 in 1964 - 1965 were examined for the effect of virus infection on overwintering. No significant differences in survival between healthy and infected plants were found. However these were spaced plants in a vigorous condition not subject to competition and these observations may not be too applicable to field conditions.

The surveys in 1964 and 1965 showed that these viruses were widely distributed throughout the clover growing area of central Alberta (Fig. 2). Virus symptoms were also observed both years in the Peace River area but inoculations were not made.

Viruses were found in 15% - 20% of the fields examined. In all cases the proportion of infected plants was very low. Newly-established stands were not found to be infected and infection increased with the age of the stands. Pea streak virus was found to be more prevalent than BYMV and occasionally plants infected with both viruses were found.

The surveys indicate that these viruses are, at present, not economically important in clover production.

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Literature cited

1. Diachun, S. and L. Henson. 1956. Symptom reaction of individual red clover plants to yellow bean mosaic virus. Phytopathology 46: 150-152.
2. Goth, R. W. and R. D. Wilcoxson. 1962. Effect of bean yellow mosaic on survival and flower formation in red clover. Crop Sci. 2: 426-429.
3. Oshima, N. and M. F. Kernkamp. 1957. Effects of viruses on overwintering of red clover in Minnesota. Phytopathology 47: 26.
4. McWhorter, F. P. 1957. Chlorazol fast pink b and trypanblue tests for virus and other proteinaceous inclusions. Stain Techn. 32: 135-138.

Table 1. Symptoms observed on test plants inoculated with viruses from clover.

Host	Symptoms	
	BYMV	PSV
Trifolium pratense	Variable mosaic [a]	Faint leaf streak [a]
Trifolium hybridum	Variable mosaic	Faint leaf streak
Vicia faba	Systemic mosaic [a]	Necrotic rings [a]
Gomphrena globosa	None	Necrotic local lesions [a]
Chenopodium amaranticolor	Blotch or None	Chlorotic local lesions [a]
Pisum sativum		
'Perfected Wales'	Faint mosaic	Wilt and death
'Wisconsin Perfection'	None	Streak
Phaseolus vulgaris	Interveinal chlorosis	None
Glycine max	None	Chlorosis and stunting
Nicotiana tabacum	None	None

[a] Symptoms shown in Figure 1.

Necrotic rings [a]

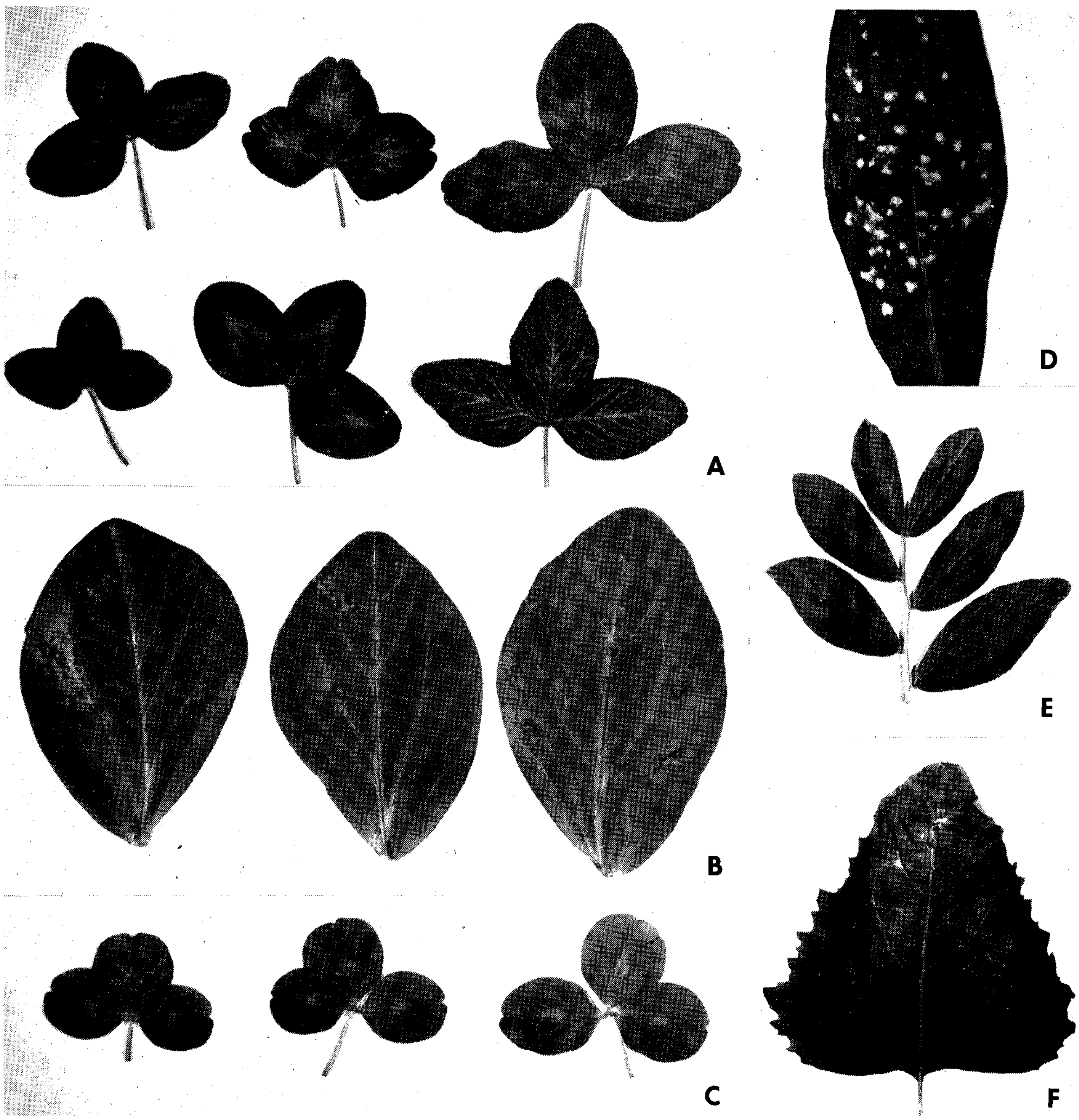

Figure 1. Virus symptoms on various host plants: A – *Trifolium pratense* infected with BYMV, B – *Vicia faba* infected with PSV showing necrotic rings, C – *Trifolium pratense* infected with PSV, D – *Gomphrena globosa* infected with PSV showing necrotic local lesions, E – *Vicia faba* infected with BYMV showing mosaic symptoms, F – *Chenopodium amaranticolor* infected with PSV showing chlorotic local lesions.

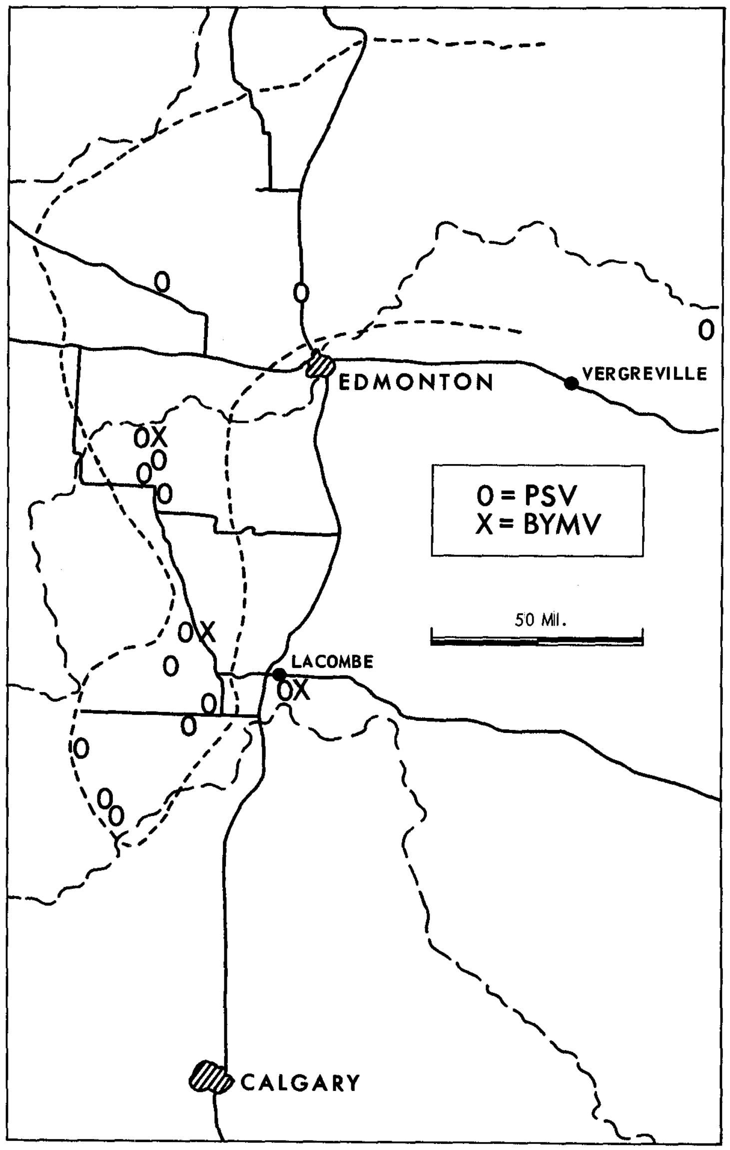

Figure 2. Distribution of viruses in central Alberta. Dotted line generally outlines the clover growing area.

VIRUS DISEASES OF CEREALS AND VECTOR POPULATIONS IN THE CANADIAN PRAIRIES DURING 1965[1]

C. C. Gill and P. H. Westdal[2]

Introduction

These observations are based mainly on weekly surveys during spring and summer in south-central Manitoba, supplemented by surveys to Saskatchewan, Alberta, and other areas of Manitoba in August.

Aster yellows virus

The six-spotted leafhopper, Macrosteles fascifrons (Stål), the chief vector of aster yellows virus (AYV), was much less abundant than usual. The spring migration of leafhoppers into Manitoba was somewhat smaller than in recent years and there was a very small summer population. Six % of the leafhoppers, mainly migrants, tested in early June were carrying AYV. In late June and early July the figure was 2%.

Although the percentage of viruliferous individuals in the population was high, particularly early in the season, there was only a trace of aster yellows in barley in southern Manitoba because of the small population of leafhoppers. A survey late in the season also indicated only a trace of AYV on barley in Saskatchewan and Alberta.

Barley yellow dwarf virus

Populations of aphid vectors for this virus were very low during the early part of the season, and appeared to consist almost exclusively of migrant English grain aphids, Macrosiphum avenae (Fabricius). During the second week of May, this species was found in fields of winter rye before the spring cereals had germinated. Population numbers rose, chiefly by local increase, from one per 50 sweeps to about 10 per 50 sweeps by the middle of June, and thereafter, assisted by additional migrants, at an increasingly rapid rate. By the end of July, counts for this species on spring cereals were as high as 410 per 50 sweeps, equivalent to an average of 7 per plant on oats in head.

The first corn leaf aphids, Rhopalosiphum maidis (Fitch), and rose grass aphids, Metopolophium dirhodum (Walker), were found on barley at the end of the first week in July, and the first greenbugs, Schizaphis graminum (Rondani), and cherry oat aphids, Rhoalosiphum padi (Linnaeus), on oats during the third week of July.

The aphid populations reached their peak density by the end of July and the first week in August, and then declined rapidly to relatively low numbers by the end of August. During the period of highest aphid populations, the English grain aphid was the dominant species on wheat and oats and frequently also on barley. The corn leaf aphid was occasionally the most numerous species on barley. Populations of greenbugs and rose grass aphids were very low by comparison, and the cherry oat aphid was scarce.

Eleven % of the English grain aphids sampled from winter rye during May and June were carrying barley yellow dwarf virus. No virus was transmitted by limited numbers of English grain aphids collected from spring cereals in June and early July. Four % of the corn leaf aphids collected from barley during July were found to be carrying a weak strain of the virus.

Successive batches of oat seedlings, exposed for weekly intervals in the field as bait plants for the aphids, showed a sharp peak in the rate of infection for the virus at a time that coincided with that for peak densities in aphid populations, namely during the last week in July and the first week in August.

There was generally only a trace of the disease in most of the 70 commercial fields of spring cereals examined. However, in one area east of Riding Mountain, from Dauphin to Neepawa, where crops were not as advanced as in other areas, aphid populations were exceptionally heavy, and disease incidence on oats and barley was occasionally as high as 90%.

Oat blue dwarf virus

This disease was first reported in Canada in 1964 from an experimental plot near Winnipeg (Can. Plant Dis. Surv. 45: 45). This disease was probably present in Manitoba previous to this, since it was reported earlier from Minnesota (1, 3). During 1965, a few very stunted plants with short, dark-green leaves and considerable blasting of the florets were found in an oat field near Swan River, Manitoba. Transmission of the virus was obtained by the six-spotted leafhopper, Macrosteles fascifrons (Stål), onto oats, where symptoms typical of the virus developed. The disease appeared to be rare in Manitoba during 1965, but diseased plants may be readily overlooked because they are often obscured by taller, healthy plants.

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1 Contribution No. 208 from Canada Department of Agriculture Research Station, Winnipeg, Manitoba.

2 Virologist and Entomologist, respectively.

Wheat striate mosaic virus

A few diseased plants were found in one wheat field in the extreme southern part of Manitoba near Morden. No further examples of this disease were seen in numerous other wheat fields. The identity of this disease was confirmed by transmission of the virus by the painted leafhopper, Endria inimica (Say) (4) to test wheat seedlings. The very low incidence supports previous observations (2) and the findings in a recent biological study of the vector (5) that this disease may never be severe in the eastern Canadian prairies.

Barley stripe mosaic virus

This disease was not observed in any of the numerous commercial fields examined. However, the virus was isolated from barley in experimental plots at Winnipeg and Portage la Prairie where infection was occasionally very high. These isolates caused a severe disease on 'Minter' and 'Selkirk' wheat and on 'Parkland' and a black-hulless barley, following mechanical inoculations in the greenhouse. Systemic infection was also obtained on 'Golden Bantam' sweet corn, and localized infection on 'Klein Wanzleben' sugar beet. No infection was obtained on 'Clintland' or 'Rodney' oats, 'Betzes' barley or 'Bloomsdale Savoy' spinach.

A virus disease of oats

A disease not previously described from Manitoba was found in one field of 'Russell' oats near St. Pierre in August, 1965. Diseased plants occurred most commonly near the margin of the field though isolated plants with symptoms were also scattered within the field. The crop was in the green, headed stage when the disease was first observed.

Symptoms consisted of necrosis on leaf blades and sheaths and a green mosaic of non-necrotic areas. The necrosis and green mosaic were apparent on leaves of all ages. Affected plants showed noticeable stunting and blasting of the florets.

When test seedlings of 'Russell' oats at the two-leaf stage were inoculated with sap from crushed leaves of the diseased plants, systemic symptoms developed nine days after inoculation at about 20°C. Initially, chlorotic lines of variable length, parallel with the veins, occurred on young leaves. Later this chlorosis developed into an irregular mottle, and with further aging, necrotic lines and irregularly shaped necrotic areas appeared on the mottled leaves. Necrosis became more extensive as the plant aged, and dark-green islands often remained in the middle of extensive necrotic areas. Infected plants showed no tendency to recover from symptoms.

All of 10 varieties of oats mechanically inoculated with the virus were susceptible, but none of 14 barley and 14 wheat varieties showed symptoms and no virus was recovered from the inoculated plants. The total infection rate on oats, using the rubbing method with corundum powder and 1% K_2HPO_4 averaged 88% for 10 plants of each of the 10 varieties inoculated. No transmission was obtained from oats to oats with the six-spotted leafhopper, the painted leafhopper, or with 5 species of aphids that commonly infest the small grains.

Although numerous oat fields in Manitoba and eastern Saskatchewan were examined during the season, no other examples of this disease were found. Work is now proceeding to characterize the virus.

Literature cited

1. Banttari, E. E. and M. B. Moore. 1959. The cause and transmission of blue dwarf of oats and of two kinds of dwarfing in barley. (Abstr.) Phytopathology 49: 533.
2. Hagborg, W. A. F. 1963. Wheat striate mosaic, a sword of Damocles hanging over the western wheat grower - or not? Can. Plant Dis. Survey 43: 194-195.
3. Moore, M. B. 1952. The cause and transmission of blue dwarf and red leaf of oats. (Abstr.) Phytopathology 42: 471.
4. Slykhuis, J.T. 1953. Striate mosaic; a new disease of wheat in South Dakota. Phytopathology 43: 537-540.
5. Westdal, P. H. (unpublished).

AIR-BORNE RUST INOCULUM OVER WESTERN CANADA IN 1965

G. J. Green[1]

A relatively large number of urediospores of the cereal rusts were caught in 1965 on vaseline-coated slides exposed in spore traps at three locations in Manitoba and at three locations in Saskatchewan (Table 2).

Rust spores from the south were present over Western Canada during May and the first half of June but the widespread and severe leaf rust epidemic that developed in the region was probably initiated by the heavy spore shower of June 15-18 that deposited spores on most of Western Canada. On June 17 and 18, 1,793 leaf rust spores per square inch of slide were caught at Regina. A second heavy, widespread spore shower occurred between June 23 and June 30. Leaf rust infections were especially heavy in western Saskatchewan where the susceptible variety 'Thatcher' predominates. As a result, the total numbers of leaf rust spores caught at Brandon, in western Manitoba, and at the three Saskatchewan locations were greater than in any year since 1960 (Table 1). Most of the spores caught after mid-July probably were produced locally.

Spores of stem rust, although not as numerous as those of leaf rust, were also present in large numbers (Table 2). Spore showers of primary inoculum from the south coincided with those of leaf rust but stem rust development was restricted by the resistance of the commonly grown varieties. The large number of stem rust spores caught at Regina in late July and August probably originated in south-western Saskatchewan where susceptible varieties of durum wheat were heavily rusted.

Table 1. Total numbers of urediospores of stem rust and leaf rust caught in spore traps in Western Canada from 1960 to 1965.

Year	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
1960	1,719	1,295	677	1,708	223	546	49	2,087	49	3,674	0	10,277
1961	88	153	109	212	24	80	27	71	37	101	8	246
1962	782	1,563	2,236	6,282	1,640	2,972	789	1,874	3,000	4,840	198	2,498
1963	2,544	13,685	2,477	26,612	1,722	15,210	1,597	39,785	2,008	69,681	5,571	80,657
1964	12,827	15,041	18,578	14,780	16,439	12,797	3,798	6,918	8,632	42,129	132	531
1965	4,943	9,811	5,362	25,978	2,698	16,981	10,559	66,730	31,635	227,576	1,927	77,502

Acknowledgements

Slides were exposed at Morden, Brandon, Indian Head and Regina by Canada Department of Agriculture staff at these locations. The data for Saskatoon was supplied by the staff of the Canada Department of Agriculture Research Station, Saskatoon. Slides from spore traps at all locations, excepting Saskatoon, were examined at Winnipeg by Mr. N. Enns.

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Table 2. Numbers of urediospores of stem rust and leaf rust per square inch caught on vaseline-coated slides exposed for 48-hour periods at 3 locations in Manitoba and 3 locations in Saskatchewan in 1965.

Date	Winnipeg		Morden		Brandon		Indian Head		Regina		Saskatoon	
	Stem Rust	Leaf Rust	Stem Rust	Leaf Rust	Stem Rust	Leaf Rust	Stem Rust	Leaf Rust	Stem Rust	Leaf Rust	Stem Rust	Leaf Rust
May Total	3	11	2	14	3	6	5	8	0	0	0	0
June 1-2	0	0	0	0	0	1	0	0	3	0	0	0
3-4	0	0	2	3	1	0	0	2	0	1	0	0
5-6	0	1	0	0	0	0	0	1	11	1	0	0
7-8	1	2	2	3	0	2	0	0	0	0	0	0
9-10	1	1	0	2	0	0	1	0	0	0	0	0
11-12	1	4	1	6	1	5	4	12	8	27	0	0
13-14	3	5	1	12	0	3	2	2	2	3	0	0
15-16	2	11	1	13	0	9	5	29	202	442	0	5
17-18	21	42	11	109	8	22	96	131	1,134	1,793	2	6
19-20	4	11	0	27	11	18	2	7	3	6	0	0
21-22	0	6	1	13	2	5	0	1	0	0	0	0
23-24	0	9	1	12	0	5	55	223	309	1,013	0	4
25-26	23	14	15	32	11	41	120	134	11	43	0	3
27-28	4	8	15	80	1	4	0	2	0	7	0	0
29-30	0	3	0	1	0	1	0	1	599	1,194	--	--
June Total	60	117	50	313	35	116	285	545	2,282	4,530	2	18
July 1-2	0	1	2	16	2	21	2	4	2	6	0	0
3-4	0	1	0	0	0	13	15	8	46	157	0	3
5-6	1	10	1	25	0	8	7	49	93	137	0	17
7-8	0	11	2	9	0	16	0	50	4	52	0	10
9-10	0	5	1	17	0	161	8	125	36	233	0	6
11-12	1	15	5	124	4	111	50	130	11	238	0	3
13-14	0	20	2	90	5	147	84	114	21	239	0	40
15-16	1	11	2	18	1	12	214	430	14	1,603	0	530
17-18	0	7	5	70	5	74	28	183	14	2,841	150	3,075
19-20	7	49	0	2	11	22	1	52	0	302	50	2,425
21-22	0	1	2	84	0	12	42	4,707	28	4,515	50	6,250
23-24	0	1,456	0	851	0	2,138	28	12,475	127	11,307	25	9,200
25-26	0	12	7	162	0	354	0	1,055	84	2,405	50	8,250
27-28	0	8	0	247	0	42	141	6,104	464	13,347	75	6,250
29-30	0	274	7	731	56	1,969	113	4,951	239	9,521	25	4,700
July Total	10	1,881	36	2,446	84	5,100	733	30,437	1,183	46,903	425	40,759
July 31 - Aug. 1	0	316	0	162	57	2,264	70	5,203	155	8,115	125	5,875
2-3	0	0	0	1,392	0	999	183	4,374	394	11,317	50	9,650
4-5	56	323	225	4,120	70	1,210	84	5,471	141	19,952	275	13,000
6-7	55	520	0	2,208	13	619	83	2,855	422	3,480	75	1,175
8-9	14	450	0	63	28	900	212	4,229	1,688	32,155	250	3,500
10-11	253	3,868	661	6,793	56	1,091	788	3,910	3,592	25,137	725	3,525
12-13	7	77	633	3,558	211	774	6,158	7,994	12,798	22,411	--	--
14-15	56	176	155	1,139	478	2,208	63	155	142	3,619	--	--
16-17	408	380	295	1,252	21	345	84	70	139	1,392	--	--
18-19	138	407	239	394	3	21	98	197	829	5,429	--	--
20-21	91	141	394	478	57	56	294	295	705	18,143	--	--
22-23	900	211	2,279	1,336	148	175	577	548	703	8,720	--	--
24-25	107	145	253	225	759	689	12	15	1,744	4,247	--	--
26-27	155	282	42	42	154	127	182	183	1,019	4,079	--	--
28-29	197	98	98	43	451	196	578	213	2,813	7,454	--	--
30-31	2,433	408	--	--	70	85	70	28	886	493	--	--
Aug. Total	4,870	7,802	5,274	23,205	2,576	11,759	9,536	35,740	28,170	176,143	1,500	36,725
TOTAL	4,943	9,811	5,362	25,978	2,698	16,981	10,559	66,730	31,635	227,576	1,927	77,502

CROWN RUST OF OATS IN CANADA IN 1965[1]

George Fleischmann[2]

Disease development and yield losses in Western Canada

Crown rust of oats, Puccinia coronata Cda. f. sp. avenae Erikss., was first found near Morden, Manitoba on July 13, 1965. By early August, trace amounts of the disease occurred as far west in southern Saskatchewan as Weyburn, and as far north as Melfort. Except for southern Manitoba, no appreciable increase in the incidence of crown rust was detected in Western Canada until the end of the season. Moderately severe infections of crown rust (50% to 80% intensity) developed in late-sown fields of 'Rodney' and 'Garry' oats in southern Manitoba by mid-August.

Losses due to crown rust were negligible in most oat fields in Western Canada during 1965. Even in the Red River Valley of southern Manitoba, crown rust developed too late in the growing season to cause important losses, except in occasional late-sown fields.

Table 1. Percent infection of crown rust on 10 oat varieties at 10 locations across Canada.

Locality	Bond	Trispernia	Exeter	Garry	Clinton	Landhafer	Rodney	C. I. 4023	Ceirch du Bach	Saia
Indian Head, Sask.	tr*	0	0	tr	tr	tr	tr	0	0	0
Brandon, Man.	10	0	50	20	25	tr	20	30	tr	0
Morden, Man.	50	tr	70	80	70	5	60	30	tr	0
Glenlea, Man.	40	1	60	50	60	10	50	50	1	0
The Pas, Man.	0	0	0	0	0	tr	0	0	0	0
Guelph, Ont.	0	0	0	tr	tr	0	tr	tr	0	0
St. Catharines, Ont.	tr	tr	0	0	0	0	0	0	0	0
Kemptville, Ont.	50	0	50	20	50	0	30	10	0	0
Merrickville, Ont.	50	40	50	60	50	20	40	50	50	10
Macdonald College, Que.	tr	0	tr	0	0	0	0	0	0	0

*tr - trace infection, less than 1 percent.

Disease ratings in the crown rust nurseries

Crown rust intensity readings on oat varieties grown at nurseries across Canada are presented in Table 1. Nurseries in which no crown rust was found, and those in which rust intensity could not be estimated because of the poor condition of the leaves, were omitted from Table 1.

Crown rust was not observed on any of the oat varieties grown at 9 of the 10 nurseries in British Columbia, Alberta, and Saskatchewan. Only traces of crown rust were detected on some varieties grown at Indian Head, Saskatchewan and The Pas, Manitoba. Nurseries in the Red River Valley of Manitoba, particularly at Glenlea and Morden, were heavily infected. Results from the crown rust nurseries agree with the survey data and with the seasonal rust reports with respect to the distribution and intensity of crown rust in 1965.

Readings from nurseries in Eastern Canada indicated that crown rust infection was very light in 1965, apart from the buckthorn - infested area of southeastern Ontario. Crown rust was not found on any of the eight nurseries grown in the Maritimes, and except for traces on a few varieties at Macdonald College, it was not detected in Quebec. Severe crown rust infection occurred on oats in the Kemptville and Merrickville nurseries in eastern Ontario which are in the centre of dense buckthorn infestations. Even the varieties 'Trispernia' and 'Ceirch du Bach' were heavily infected at Merrickville. Crown rust infections were very light in all other regions of Ontario. Traces of infections were found on a few varieties from the Guelph and St. Catharines nurseries, while no crown rust appeared in the other nurseries in Ontario.

1 Contribution 207 from the Canada Department of Agriculture Research Station, Winnipeg, Manitoba.

2 Plant Pathologist, Canada Department of Agriculture Research Station, Winnipeg, Manitoba.

Table 2. Distribution of physiologic races of Puccinia coronata avenae collected on oats in Canada in 1965.

Physiologic race	Number isolates west	% of all isolates west	Number isolates east	% of all isolates east	Total isolates east and west
201	2	1.2	-	-	2
202	3	1.9	-	-	3
203	5	3.1	2	10	7
210	4	2.5	4	20	8
211	10	6.2	2	10	12
213	8	5.0	-	-	8
216	1	0.6	-	-	1
226	1	0.6	1	5	2
230	1	0.6	-	-	1
231	3	1.9	-	-	3
274	12	7.4	-	-	12
279	12	7.4	-	-	12
281	-	-	1	5	1
283	1	0.6	-	-	1
284	3	1.9	1	5	4
293	5	3.1	1	5	6
294	19	11.8	3	15	22
295	5	3.1	-	-	5
297	-	-	1	5	1
299	1	0.6	-	-	1
320	1	0.6	-	-	1
324	-	-	1	5	1
326	7	4.3	-	-	7
327	2	1.2	-	-	2
330	2	1.2	1	5	3
333	1	0.6	-	-	1
338	39	24.2	-	-	39
339	5	3.1	-	-	5
341	1	0.6	2	10	3
New races	4	2.5	-	-	4
Totals	158		20		178

Distribution of physiologic races

A total of 178 crown rust isolates, comprising 32 physiologic races, were identified in 1965. Very few of these cultures originated in Eastern Canada. As far as could be determined from the few isolates available, there was little change in the composition of the crown rust population in the east.

Races virulent on the varieties 'Landhafer' and 'Santa Fe' again made up more than half the crown rust population in Western Canada in 1965. As indicated in Table 2, the most frequently occurring biotype was race 338 (resistance formula 1, 8, 9, 10). It comprised one-quarter of the isolates identified from Manitoba and Saskatchewan. In contrast to the situation in 1964 (1), no races were identified in 1965 which attacked the differential varieties 'Trispernia' and 'Bondvic'. Highly virulent races like 264 and 276, which attack the differential varieties 'Trispernia', 'Bondvic', 'Landhafer', and 'Santa Fe', and which constituted 14% of the crown rust population of Western Canada in 1964, were not isolated this year.

Three races, with previously undescribed virulence combinations on the crown rust differential oat varieties, were isolated in 1965. Their resistance formulae are: 1, 2, 6, 7, 8, 9, 10; 1, 4, 8, 9, 10; and 1, 6, 8, 9, 10.

Acknowledgements

I am indebted to Dr. D. J. Samborski for the identification of all crown rust isolates collected in 1965 during my absence abroad. I am also grateful for assistance given by the cooperators in the care of rust nurseries and the collection of rust specimens. Mr. W. L. Timlick performed the technical operations requisite to the identification of the isolates.

Literature cited

1. Fleischmann, G. 1965 Crown rust of oats in 1964. Can. Plant Dis. Survey 45: 15-18.

STEM RUST OF OATS IN CANADA IN 1965

J. W. Martens and G. J. Green[1]

Disease development and crop losses in Western Canada

Stem rust of oats (Puccinia graminis Pers. f. sp. avenae Erickss. & Henn.) developed too late in the season to cause significant losses in Western Canada in 1965. The first traces were observed on wild oats in Manitoba and western Saskatchewan July 30. The rust did not build up very rapidly so that most of the oats reached maturity with relatively little stem rust infection. However, late fields in the Red River Valley of Manitoba had infections of up to 60% by the beginning of September. These fields undoubtedly suffered a considerable reduction in yield from rust infection, in addition to that caused by frost. Disease development was apparently delayed by the fact that the earliest spore showers consisted largely of race C5(6F) to which the bulk of the western oat population was resistant.

Uniform rust nurseries

Rust nurseries consisting of ten varieties (Table 1) were grown at 36 locations across Canada. The nurseries were planted, cared for and harvested by cooperating University and Canada Department of Agriculture personnel and then sent to Winnipeg for disease rating. No rust was observed on nurseries in Alberta. Infections were generally absent or light in British Columbia, Saskatchewan and Manitoba. Infections in the nurseries at Glenlea and Winnipeg, Manitoba were heavier than in most oat fields in the Red River Valley because they were planted late. Heavy infections were common in the barberry area of Ontario. Rust infections were light or absent east of Ontario.

Table 1. Percent infections of stem rust of oats (Puccinia graminis f. sp. avenae) in 10 varieties of oats in 14[a] uniform rust nurseries in Canada in 1965.

Locality	Bond	Trispernia	Landhafer	Ceirch du Bach	Saia	Exeter	Clinton	Rodney	Garry	C. I. 4023
Creston, B. C.	0	tr[b]	tr	tr	0	0	0	0	0	0
Melfort, Sask.	10	tr	2	5	0	2	5	0	0	tr
Brandon, Man.	5	0	tr	0	0	1	1	0	tr	0
Glenlea, Man.	60	5	tr	tr	tr	30	60	20	2	2
Morden, Man.	1	0	0	0	0	1	10	tr	0	0
The Pas, Man.	0	0	tr	0	0	0	0	0	0	0
Winnipeg, Man.	30	tr	tr	tr	0	20	50	5	tr	tr
Alfred, Ont.	2	1	tr	0	0	tr	tr	tr	0	0
Appleton, Ont.	80	80	60	50	30	70	70	80	70	30
Ft. William, Ont.	tr	tr	0	tr	0	0	0	0	0	0
Kemptville, Ont.	70	0	10	0	0	70	50	50	15	1
Merrickville, Ont.	60	60	30	40	5	60	60	60	40	30
Ottawa, Ont.	50	30	20	30	0	25	30	40	30	0
Macdonald Coll., P. Q.	0	tr	tr	5	tr	15	tr	10	tr	0

a No rust was observed in 22 other nurseries located at Saanichton and Agassiz, B. C., Edmonton, Beaverlodge, Lacombe, and Lethbridge, Alta., Indian Head and Scott, Sask., Guelph, Kapuskasing, St. Catharines, Williamstown and Verner, Ont., L'Assomption, Lennoxville, Normandin and Quebec, P. Q., Kentville and Nappan, N. S., Fredericton, N. B., Charlottetown, P. E. I., and Doyles, Nfld.

1 Plant Pathologists, Canada Department of Agriculture, Research Station, Winnipeg, Manitoba.

Physiologic race identification and distribution

Physiologic races were identified using a host set consisting of 'Richland' (gene A), 'Rodney' (gene B), 'Minrus' (gene D), 'Jostrain' (gene E), 'Eagle^2 x C.I. 4023' (gene F) and 'C.I. 5344-1' (gene H). The race designations used (Table 2) follow the system initiated by Green (1, 2).

Table 2. Virulence formulae and numbers for races of oat stem rust identified in Canada from 1963 to 1965.

Formula No.	Formula (Effective/Ineffective Host Genes)	Race
C1	ABDEF/H	1
C2	ABDF/EH	2
C3	AF/BDEH	7A-12A
C4	BDFH/AE	8
C5	BH/ADEF	6F
C6	DF/ABEH	8A-10A
C7	D/ABEFH	8AF
C8	EF/ABDH	4A
C9	F/ABDEH	6A-13A
C10	H/ABDEF	6AF
C11	DF/ABE	8A
C12	DH/ABEF	8AF
C13	BF/ADEH	6
C14	FH/ABDE	6A
C15	BDF/AEH	8
C16	ABF/DEH	7
C17	DEF/ABH	11A
C18	ABFH/DE	7
C19	ABDFH/E	2

A supplementary set of 'Rosen's Mutant', 'Saia' and 'C.I. 3034' was also used. Reactions of 'Rosen's Mutant' were usually similar to but more resistant than those of 'C.I. 5844-1'. Nineteen of the 72 isolates avirulent on 'Rosen's Mutant' were moderately virulent on 'C.I. 5844-1'. Most of these 19 isolates were races C3(7A-12A) and C6(6A-13A). 'C.I. 3034', which has adult plant resistance to race C10(6AF), was susceptible to all isolates of this race in the seedling stage. It reacted like 'Minrus' to most isolates. 'Saia' was resistant to all isolates identified in 1965.

Races C10(6AF), C3(7A-12A) and C5(6F) accounted for 50%, 26% and 22%, respectively, of all isolates from Manitoba and Saskatchewan in 1965 (Table 3). Race C10, which can attack all commercially grown oat varieties, continued to increase in prevalence. It increased from 5% of all isolates from Manitoba and Saskatchewan in 1963 to 41% in 1964 to 50% in 1965.

The data in Table 4 indicate that the early incoming inoculum was not predominantly C10 as the above race distribution might suggest. The first isolates were obtained mostly from plants with no

Table 3. Distribution by provinces of physiologic races of Puccinia graminis f. sp. avenae identified in Canada in 1965.

Formula No.	Race	B. C.	Sask.	Man.	Ont.	Que.	No. of Isolates	Percent of Total Isolates
C1	1	1	0	0	0	0	1	.7
C3	7A-12A	0	4	16	0	0	20	14.7
C5	6F	0	7	10	0	0	17	12.5
C6	8A-10A	0	0	0	4	3	7	5.1
C8	4A	0	0	0	7	1	8	5.9
C9	6A-13A	0	0	0	34	5	39	28.8
C10	6AF	0	4	34	0	0	38	27.9
C14	6A	0	0	0	2	0	2	1.5
C16	7	0	0	1	0	0	1	.7
C17	11A	0	0	0	1	0	1	.7
C19	2	2	0	0	0	0	2	1.5
Total		3	15	61	48	9	136	100.0

Table 4. Distribution by provinces of physiologic races of Puccinia graminis f. sp. avenae collected on varieties of cultivated oats with no rust resistance and wild oats in Canada in 1965.

Formula No.	Race	B. C.	Sask.	Man.	Ont.	Que.	No. of Isolates	Percent of Total Isolates
C3	7A-12A	0	0	2	0	0	2	10
C5	6F	0	5	7	0	0	12	60
C8	4A	0	0	0	1	0	1	5
C9	6A-13A	0	0	0	1	0	1	5
C10	6AF	0	0	3	0	0	3	15
C16	7	0	0	1	0	0	1	5
Total		0	5	13	2	0	20	100

rust resistance and they consisted largely of race C5(6F). However, the predominant host populations of 'Rodney' which is resistant to C5 and 'Garry' which is resistant to C5 and C3 in the west exerted a great selection pressure in favor of C10 so that it increased rapidly. Early spore showers of race C10 could cause very serious losses in the immediate future.

In Ontario and Quebec race C9(6A-13A) comprised 68% of all isolates with C6(8A-10A) and C8 (4A) accounting for most of the remainder. All commercial oat varieties are susceptible to race C9. The absence of races C3, C5, and C10 from Eastern Canada in 1965 suggests that the origin of the primary inoculum in this area was different from that in Western Canada. Barberry was probably an important source of primary inoculum in Eastern Canada.

Several previously undescribed physiologic races were isolated from oats in 1965. These include C14 = FH/ABDE and C17 = DEF/ABH from Ontario, C16 = ABF/DEH from Manitoba and C19 = ABDFH/E from British Columbia.

Acknowledgements

The authors are grateful for the technical assistance of J.C. Anderson, the rust nursery cooperators and those who submitted rust specimens from various parts of Canada.

Literature cited

1. Green, G.J. 1963. Stem rust of oats in Canada in 1963. Can. Plant Dis. Survey 43: 173-178.
2. Green, G.J. 1965. Stem rust of oats in Canada in 1964. Can. Plant Dis. Survey 45: 19-22.

STEM RUST OF WHEAT, RYE AND BARLEY IN CANADA IN 1965

G. J. Green[1]

Prevalence and importance in Western Canada

Wheat stem rust (Puccinia graminis Pers. f. sp. tritici Erikss. & Henn.) was widely distributed in Western Canada in 1965 and severe infections developed on susceptible varieties in the three Prairie Provinces. It was first found at Morden, Manitoba, on June 17. Development was slow until mid-July, but by the end of July there was abundant infection on susceptible varieties and wild grasses in much of Western Canada. Fields of susceptible varieties were severely infected before harvest, which was later than normal.

Fortunately, the acreage of susceptible varieties was small and the overall loss was small. The resistant varieties 'Selkirk', 'Pembina', 'Ramsey' and 'Stewart 63' that predominate in the main rust area of Manitoba and eastern Saskatchewan were nearly free from stem rust, but fields of the 15B susceptible variety 'Lee' had moderately severe infections. Stem rust infections were light on the variety 'Thatcher' that predominates in central and western Saskatchewan and in Alberta. Susceptible varieties of durum wheat, mostly 'Pelissier' and 'Stewart', sown in southwestern Saskatchewan were severely infected and suffered heavy losses. Some fields were reported to have been destroyed. The severe infections on susceptible varieties indicate that the resistance of the predominant varieties in Western Canada again prevented widespread and severe stem rust losses.

The small amount of stem rust on 'Thatcher', the main variety in Western Canada, was unexpected. 'Thatcher' is susceptible to the biotypes of race 15B found in earlier years and race 15B predominated in 1965. It appears that, in the field under the relatively cool conditions that prevailed in 1965, 'Thatcher' is resistant to the biotype of race 15B now prevalent. This biotype, C18(15B-1L (Can.)), was first found in 1962 and has quickly increased to predominance. It was mainly responsible for the severe damage to susceptible durum wheat in southwestern Saskatchewan and to susceptible varieties in Manitoba.

Stem rust of wheat in the rust nurseries

Uniform rust nurseries that included the 13 varieties shown in Table 1, were grown at 37 locations across Canada in 1965. The nurseries were planted and cared for by Canada Department of Agriculture and University personnel. A small sheaf was cut from each row of each nursery before the plants matured and sent to Winnipeg where disease ratings were made.

Severe infections developed in nurseries from Alberta to Quebec on the susceptible varieties 'Red Bobs' and 'Marquis'. The severe stem rust epidemic seemed to be confined to central Canada, since most nurseries in British Columbia and all of those located east of Quebec were free from infection.

In most nurseries, the variety 'Lee' was more severely infected than 'Thatcher'. Both varieties were considered susceptible to race 15B, but 'Thatcher' appears to be less susceptible than 'Lee' to the strain of 15B now prevalent. The widely grown variety 'Selkirk' was lightly infected at only a few locations, and the recently released variety 'Manitou' had only traces of rust at a few locations. The new durum wheat variety 'Stewart 63' had only a trace of rust at one location.

Stem rust of barley and rye in the rust nurseries

The barley variety 'Montcalm' is susceptible to both wheat stem rust and rye stem rust (P. graminis Pers. f. sp. secalis Erikss. & Henn.), and it was infected at the same locations as susceptible varieties of wheat (Table 2). The smaller percentages of rust on 'Montcalm' than on wheat is attributable to the earlier maturity of barley. The barley varieties 'Parkland' and 'C.I. 10644' are resistant to wheat stem rust, but susceptible to rye stem rust. They appear to have matured before heavy infections of rye stem rust could develop on them.

Rye stem rust was widely distributed on 'Prolific rye' (Table 2). The heaviest infections were at locations in Eastern Canada where barberry occurs, and at Creston, B. C. The reason for the heavy infection of rye stem rust at Creston is not known. Creston is isolated from the prairie region by mountains, and in past years physiologic race surveys of wheat stem rust have indicated that inoculum is brought into the Creston area from the south. There is no comparable information to indicate the source of inoculum of rye stem rust.

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Table 1. Percent infection of stem rust of wheat (Puccinia graminis f. sp. tritici) on 13 wheat varieties in uniform rust nurseries at 29[1] locations in Canada in 1965.

Locality	Common Wheat										Durum Wheat		
	Red Bobs	Marquis	Lee	Thatcher	Selkirk	McMurachy	Kenya Farmer	Manitou	Exchange	Frontana	Mindum	Ramsey	Stewart 63
Creston, B.C.	1	tr[2]	0	0	0	0	0	0	0	tr	0	0	0
Edmonton, Alta.	50	20	5	tr	0	0	0	0	5	0	5	0	0
Lacombe, Alta.	5	1	2	tr	0	0	0	0	0	0	tr	0	0
Lethbridge, Alta.	10	tr	0	0	0	0	0	0	0	0	tr	0	0
Scott, Sask.	25	5	35	2	0	tr	tr	0	2	tr	45	tr	0
Melfort, Sask.	80	45	25	20	0	3	25	0	35	10	75	0	tr
Indian Head, Sask.	40	5	2	tr	tr	0	0	0	-	-	5	0	-
Brandon, Man.	80	20	60	5	0	20	tr	0	10	tr	10	tr	0
The Pas, Man.	50	15	10	5	5	0	0	0	tr	tr	1	0	0
Morden, Man.	70	60	60	5	0	tr	tr	0	20	0	20	tr	0
Winnipeg, Man.	90	90	40	10	tr	tr	1	0	50	10	50	1	0
Glenlea, Man.	80	50	40	10	0	tr	0	0	1	tr	30	0	0
Fort William, Ont.	65	50	40	25	0	0	0	0	4	0	10	0	0
Kapuskasing, Ont.	5	tr	0	0	0	0	0	0	0	0	tr	tr	0
St. Catharines, Ont.	-	1	0	1	0	0	0	0	1	0	5	1	0
Guelph, Ont.	50	50	15	5	tr	0	10	0	2	tr	45	2	0
Kemptville, Ont.	25	30	0	tr	0	0	0	0, 30*	1	5	10	5	0
Merrickville, Ont.	60	40	5	5	0	tr	tr	0	5	0	7	5	0
Appleton, Ont.	70	100	30	5	0	40	25	tr	35	35	80	30	0
Williamstown, Ont.	tr	tr	0	0	0	0	0	0	0	0	0	0	0
Alfred, Ont.	tr	0	0	0	0	0	0	0	tr	0	0	0	0
Verner, Ont.	80	60	1	5	0	0	0	0	1	tr	10	1	0
Ottawa, Ont.	70	70	50	10	0	5	tr	0	20	10	40	30	0
Macdonald Coll., Que.	40	15	20	tr	0	tr	tr	0	35	tr	40	tr	0
Lennoxville, Que.	0	5	2	0	0	0	tr	0	0	0	8	0	0
La Pocatière, Que.	60	80	20	10	0	5	0	tr	20	10	20	tr	0
L'Assomption, Que.	tr	2	0	0	0	0	0	0	tr	0	tr	0	0
Normandin, Que.	1	1	tr	0	0	0	tr	0	0	tr	5	1	0
Quèbec, Que.	20	5	0	tr	0	tr	0	0	0	tr	10	tr	0

1 No rust was observed in 8 other nurseries located at Saanichton and Agassiz, B.C., Beaverlodge, Alta., Nappan and Kentville, N.S., Fredericton, N.B., Charlottetown, P.E.I., and Doyles, Nfld.

2 tr = trace infection

* Segregating

Table 2. Percent infection of stem rust (Puccinia graminis) on three varieties of barley and one variety of rye in uniform rust nurseries at 22[1] locations in Canada in 1965.

Locality	Barley: Montcalm	Barley: Parkland	Barley: C.I. 10644	Rye: Prolific
Creston, B.C.	1	tr	1	60
Edmonton, Alta.	10	5	0	1
Lacombe, Alta.	1	0	0	0
Scott, Sask.	tr	0	0	0
Melfort, Sask.	tr	0	0	0
Indian Head, Sask.	tr	0	0	5
Brandon, Man.	10	0	0	5
The Pas, Man.	tr	tr	tr	tr
Morden, Man.	50	tr	tr	tr
Winnipeg, Man.	30	tr	tr	tr
Glenlea, Man.	10	tr	tr	10
Fort William, Ont.	tr	tr	0	tr
Guelph, Ont.	15	tr	0	80
Kemptville, Ont.	50	5	1	20
Merrickville, Ont.	30	tr	0	50
Appleton, Ont.	25	10	5	70
Verner, Ont.	tr	0	0	0
Ottawa, Ont.	20	tr	0	10
Macdonald Coll., Que.	5	0	0	15
Lennoxville, Que.	tr	0	0	0
La Pocatière, Que.	tr	0	0	5
L'Assomption, Que.	5	5	2	0

[1] No rust was observed in nurseries located at Saanichton and Agassiz, B.C., Beaverlodge and Lethbridge, Alta., Kapuskasing, St. Catharines, Williamstown and Alfred, Ont., Normandin and Québec, Que., Nappan and Kentville, N.S., Fredericton, N.B., Charlottetown, P.E.I., and Doyles, Nfld.

Distribution of physiologic races

In 1965, 15 physiologic races were identified on the differential host varieties 'Marquis', 'Reliance', 'Arnautka', 'Mindum', 'Einkorn' and 'Vernal' of the set described by Stakman et al. (3). Lines of 'Marquis' wheat carrying single substituted resistance genes (2) were used to classify cultures into 15 virulence formulas. The 23 virulence combinations described in 1964 (1) and seven new combinations appear in Table 5. In this report, races are designated by the formula number followed by the physiologic race number in parentheses.

The main feature of the physiologic race distribution in Canada in 1965 was a continuation of the trend of increasing prevalence of race C18(15B-1L (Can.)), and of decreasing prevalence of races C17 (56) and C9(15B-1L (Can.)). Race C18(15B-1L (Can.)) increased from 31.3% of the isolates in 1964 to 53% in 1965 (Table 3), whereas, race C17(56) decreased by 6.9% to 15% of the isolates, and race C9(15B-1L (Can.)) decreased from 15.4% to 1.9%. These changes probably had practical importance, because the variety 'Thatcher' that predominates in Saskatchewan and Alberta appears to have some resistance to race C18(15B-1L (Can.)). It was not seriously affected by stem rust in 1965 when susceptible durum wheat varieties in southwestern Saskatchewan were heavily rusted. In the main rust area of Manitoba and eastern Saskatchewan, where 'Selkirk' and 'Pembina' predominate, the changes had no significance.

There was a sharp increase, mainly in Ontario and Quebec, of races C1(17) and C2(17A), and the new virulence combinations C24(17) and C29(17) were found. There is no obvious reason for the increase of races C1(17) and C2(17A) as they do not threaten

Table 5. Virulence formulas, formula numbers, and physiologic race numbers of cultures identified in 1964 and 1965.

Formula Number	Virulence Formula (Effective/Ineffective Host Genes)	Physiologic Race
C1	5, 6, 7, 9a, 9b, 10, 11/8	17
C2	5, 6, 7, 9a, 9b, 10/8, 11	17A
C3	5, 6, 9a, 11/7, 8, 9b, 10	29-4 (Can.)
C4	5, 6, 11/7	23
C5	5, 9a, 9b, 11/6, 7, 8, 10, GB[1]	29-1 (Can.)
C6	5, 9a, 9b, 11, GB/6, 7, 8, 10	29-2 (Can.)
C7	5, 11, GB/6, 7	48
C8	5, 11/6, 7, GB	48A
C9	6, 7, 8, 9a, 9b, 10/5, 11	15B-1L (Can.)
C10	6, 7, 8, GB/5, 9a, 9b, 10, 11	15B-1 (Can.)
C11	6, 7, 8/5, 9a, 9b, 10, 11, GB	15B-4 (Can.)
C12	6, 7, 9a, 9b, 10, 11/5, 8	11
C13	6, 7, 10, 11/5, 8, 9a, 9b	32, 113
C14	6, 7, 10, 11/5	14, 38
C15	6, 7, 10/5, 8, 9a, 9b, 11	11, 32, 113
C16	6, 7, 11/5	39
C17	6, 8, 9a, 9b, 11/5, 7, 10	11, 56
C18	6, 8, 9a, 9b/5, 7, 10, 11	15B-1L (Can.)
C19	6, 10, 11/5, 7	10
C20	7, 8, 11/5, 6, 9a, 9b, 10	11, 87
C21	9a, 11/5, 6, 7, 8, 9b, 10	32
C22	9a/5, 6, 7, 8, 9b, 10, 11	32
C23	/5, 6, 7	38
C24	5, 7, 9a, 9b, 10/6, 8, 11	17
C25	/5, 6, 7, 10, 11	38
C26	6, 7, 8, 9b/5, 9a, 10, 11	15B-4 (Can.)
C27	6, 11/5, 7, 10	33, 59
C28	6, 8, 9b, 11/5, 7, 9a, 10	18, 54
C29	5, 6, 7, 9a, 10, 11/8, 9b	17
C30	9a, 9b/5, 6, 7, 8, 10, 11	29

[1] GB indicates the reaction of the variety Golden Ball

Table 3. Distribution by provinces of physiologic races of Puccinia graminis f. sp. tritici collected on wheat, barley and grasses in 1965.

Virulence Formula Number	Physiologic Race Number	Province							Number of Isolates	Percent of Total Isolates
		P. E. I.	Que.	Ont.	Man.	Sask.	Alta.	B. C.		
C1	17	1	4	36	3	2	1	0	47	12.6
C2	17A	0	3	12	1	2	0	0	18	4.8
C9	15B-1L (Can.)	0	0	1	1	4	1	0	7	1.9
C11	15B-4 (Can.)	0	0	0	0	1	0	0	1	0.3
C13	113	0	0	1	0	0	0	0	1	0.3
C17	56	0	1	10	9	20	16	0	56	15.0
C18	15B-1L (Can.)	0	5	30	37	91	35	0	198	53.0
C20	11	0	0	2	3	1	1	0	7	1.9
C20	15-87	0	1	6	1	1	1	0	10	2.7
C22	32	0	0	2	0	0	2	0	4	1.0
C24	17	0	0	3	0	1	0	0	4	1.0
C25	38	0	0	5	3	3	2	0	10	3.5
C27	33	0	0	0	0	0	0	1	1	0.3
C27	59	0	0	0	0	0	0	1	1	0.3
C28	18	0	0	0	0	0	0	2	2	0.5
C28	54	0	0	0	0	0	0	1	1	0.3
C29	17	0	0	1	0	0	0	0	1	0.3
C30	29	0	0	0	0	0	0	1	1	0.3
Total Isolates		1	14	109	58	126	59	6	373	100.0

resistant commercial varieties.

There was very little difference between the distribution of races isolated from all varieties (Table 3), and the distribution of the races isolated from susceptible varieties (Table 4). Evidently the isolates from selective, resistant varieties did not influence the results of the survey in any important way.

In 1965, the recently released variety 'Manitou' was inoculated with all isolates. No isolate produced type 4 infections on 'Manitou', but races C20(11 and 15), C22(32) and C25(38) produced the most susceptible reactions. These races are not new, and they did not increase in prevalence in 1965. Race C25(38) is of interest because, as shown below, it is more virulent on seedlings of certain highly resistant varieties than most other races.

Variety	Infection Type
Kenya Farmer	2
Mayo 54	2 to 3
Mida-McMurachy-Exchange II-47-26	1 to 3
Crim	0
Chris	; to 3

Table 4. Distribution by provinces of physiologic races of Puccinia graminis f. sp. tritici collected on wild barley and susceptible varieties of wheat and barley in 1965.

Virulence Formula Number	Physiologic Race Number	Province P. E. I.	Que.	Ont.	Man.	Sask.	Alta.	B. C.	Number of Isolates	Percent of Total Isolates
C1	17	1	0	31	2	0	0	0	34	14.1
C2	17A	0	1	9	0	0	0	0	10	4.2
C9	15B-1L (Can.)	0	0	1	1	3	0	0	5	2.1
C13	113	0	0	1	0	0	0	0	1	0.4
C17	56	0	0	9	8	17	12	0	46	19.1
C18	15B-1L (Can.)	0	4	16	35	46	18	0	119	49.4
C20	11	0	0	2	3	0	1	0	6	2.5
C20	15-87	0	0	3	1	0	0	0	4	1.7
C22	32	0	0	0	0	2	1	0	3	1.2
C24	17	0	0	1	0	1	0	0	2	0.8
C25	38	0	0	1	0	2	1	0	4	1.7
C27	33	0	0	0	0	0	0	1	1	0.4
C27	59	0	0	0	0	0	0	1	1	0.4
C28	18	0	0	0	0	0	0	2	2	0.8
C28	54	0	0	0	0	0	0	1	1	0.4
C29	17	0	0	1	0	0	0	0	1	0.4
C30	29	0	0	0	0	0	0	1	1	0.4
Total Isolates		1	5	75	50	71	33	6	241	100.0

Adult plant tests were carried out with 'Manitou', and its parent variety 'Thatcher' to determine the significance of the variable results with seedlings. Infection types on adult plants of 'Manitou' ranged from 1 to 3 with two cultures of race C25(38), but on the average the variety was considered to be moderately resistant (Table 6). Two cultures of race C22(32) were slightly more virulent on 'Manitou' than was race C25(38).

The varieties 'Selkirk' and 'Stewart 63' also were inoculated with all isolates. 'Stewart 63' was resistant to all isolates, but races C20(11 and 87) and C22(32) were virulent on seedlings of 'Selkirk'. They are not new races and they have not increased in prevalence.

Isolates obtained from rust collected at Creston, B. C., were distinct from those obtained from other parts of the country. They were identified as the new formulas C27, C28 and C30, and corresponded roughly to races 33 and 59, 18 and 54, and 29 respectively (Table 3).

Table 6. Estimated mean infection types on the upper two sheaths of four adult plants of the wheat varieties 'Manitou' and 'Thatcher' inoculated with seven cultures of stem rust.

Formula No.	Physiologic Race	Culture No.	Infection Type Manitou	Infection Type Thatcher
C18	15B-1L (Can.)	86-65	Fleck	3-
C2	17A	327-65	0	0
C22	32	306-65	2	2+
C22	32	247-65	2+	2+
C25	38	188-65	2	2+
C25	38	124-65	1+	2+
C17	56	92-65	Fleck	1+

Composite urediospore collections of all isolates in groups of about 20 were used to inoculate 20 highly resistant varieties that included 'Kenya Farmer', 'Mayo 54', 'Mida - McMurachy - Exchange II-47-26', 'Frontana-K58-Newthatch II-50-17', 'Crim', 'Justin', 'ND 264', 'Chris', 'C.T. 261', 'St 464', and C.I. 8155'. A few large pustules developed on the varieties 'Mayo 54', 'Mida-McMurachy-Exchange II-47-26', 'Frontana-K58-Newthatch II-50-17', and 'Chris'. Single pustule isolates from these varieties were identified as race C25(38).

Literature cited

1. Green, G. J. 1965. Stem rust of wheat, rye and barley in Canada in 1964. Can. Plant Dis. Survey 45: 23-29.
2. Green, G.J., D.R. Knott, I.A. Watson, and A. T. Pugsley. 1960. Seedling reactions to stem rust of lines of Marquis wheat with substituted genes for rust resistance. Can. J. Plant Sci. 40: 524-538.
3. Stakman, E.C., D.M. Stewart, and W.Q. Loegering. 1962. Identification of physiologic races of Puccinia graminis var. tritici. U.S.D.A. Bureau of Entomology and Plant Quarantine Bull. E-617 (Revised).

LEAF RUST OF WHEAT IN CANADA IN 1965[1]

D. J. Samborski [2]

Disease development in Western Canada

Leaf rust was severe in much of the wheat growing area of Western Canada. Wheat leaf rust appeared early in Manitoba (June 7), but developed slowly because the predominant varieties, 'Selkirk' and 'Pembina', are moderately resistant in early stages of growth. It developed rapidly after mid-July and severe infections were common throughout the province before harvest. Data from a yield trial at Winnipeg, in which plots of wheat were protected from rust with a fungicide, indicate that late fields of 'Selkirk' suffered a 20% reduction in yield as a result of leaf rust infection in 1965.

Leaf rust appeared later in Saskatchewan but developed rapidly after mid-July. Infections were very heavy on 'Thatcher' and 'Canthatch' but developed a little late to cause maximum losses. It is estimated that losses averaging 20% occurred with 'Thatcher' and 'Canthatch' in south-central Saskatchewan. Severe leaf rust infections occurred in some parts of Alberta but no estimate of yield loss is available.

Table 1. Percent infection of leaf rust of wheat (Puccinia recondita) in 1965 on 15 wheat varieties in uniform rust nurseries at 28[a] locations in Canada.

Locality	Lee	Thatcher	Selkirk	Red Bobs	Manitou	Marquis	Kenya Farmer	McMurachy	Ramsey	Mindum	Stewart 63	D. T. 184	Thatcher^6 x Transfer	Exchange	Frontana
Creston, B. C.	tr	80	20	80	tr	70	tr	80	0	tr	0	0	0	0	tr
Edmonton, Alta.	60	80	60	80	5	70	40	80	0	0	0	0	0	0	0
Lacombe, Alta.	0	2	0	3	0	2	0	2	0	0	0	0	0	0	0
Lethbridge, Alta.	7	25	5	25	2	20	10	20	0	0	0	0	0	0	0
Indian Head, Sask.	25	50	25	50	10	50	20	65	0	tr	0	0	0	0	0
Scott, Sask.	35	70	40	80	15	65	30	70	0	0	tr	0	0	10	0
Melfort, Sask.	70	90	60	90	5	80	60	90	0	0	0	0	0	0	0
Brandon, Man.	80	90	60	80	1	-	30	100	tr	0	tr	tr	0	tr	tr
The Pas, Man.	60	80	90	80	10	80	1	90	tr	0	tr	0	0	0	0
Morden, Man.	40	90	70	60	2	80	5	90	tr	0	0	tr	0	0	0
Winnipeg, Man.	40	80	50	80	10	80	30	80	0	0	0	0	0	0	0
Glenlea, Man.	50	80	50	80	5	70	60	80	0	0	0	0	0	tr	0
Verner, Ont.	30	80	20	80	3	80	30	80	0	5	0	1	0	0	0
Williamstown, Ont.	3	40	3	40	tr	40	3	40	0	3	0	0	0	0	0
Alfred, Ont.	3	50	3	50	0	50	3	50	0	2	0	0	0	0	0
Kapuskasing, Ont.	1	20	1	20	0	20	1	20	0	0	0	0	0	0	0
Kemptville, Ont.	15	60	15	70	5	70	10	60	0	tr	0	0	0	0	0
Fort William, Ont.	30	60	35	65	15	55	30	65	tr	0	0	tr	0	0	0
Guelph, Ont.	30	85	20	80	15	80	30	75	tr	tr	tr	0	0	tr	0
Ottawa, Ont.	60	80	30	85	3	70	60	80	0	3	0	0	0	1	0
Appleton, Ont.	50	80	40	80	tr	70	40	80	0	10	0	0	0	0	0
Merrickville, Ont.	tr	50	tr	50	tr	40	tr	50	0	tr	0	0	0	0	0
St. Catharines, Ont.	tr	20	tr	-	tr	25	tr	25	0	0	0	0	0	0	0
La Pocatière, Que.	20	50	25	60	5	55	25	50	5	5	5	tr	0	0	0
Québec, Que.	10	45	5	45	10	45	10	60	0	5	0	0	0	0	0
Macdonald College, Que.	15	40	15	60	tr	45	15	40	0	tr	0	0	0	0	0
Lennoxville, Que.	30	65	25	65	15	65	35	70	tr	0	0	0	0	0	0
Normandin, Que.	40	80	10	40	5	70	40	80	3	5	5	5	0	0	0

[a] Wheat leaf rust was not found on nurseries from Saanichton, B.C., Agassiz, B.C., Beaverlodge, Alta., L'Assomption, Que., Nappan, N.S., Kentville, N.S., Fredericton, N.B., Charlottetown, P.E.I., Doyles, Nfld.

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Leaf rust in the rust nurseries

Severe infections of leaf rust occurred in nurseries from Quebec to British Columbia, (Table 1). No leaf rust was observed in nurseries in the Maritime Provinces. A considerable amount of leaf rust was recorded on 'Selkirk' wheat; this was partly due to the increased prevalence of virulent strains of leaf rust. 'Manitou', a newly released variety, showed good resistance and the rust infections observed on this variety were of the resistant or the moderately resistant type. The durum varieties 'Ramsey', 'Mindum', 'Stewart 63' and 'D. T. 184', and the common wheats 'Thatcher^{6}' x 'Transfer', 'Exchange', and 'Frontana', were resistant at all locations. The reading of 10% on 'Exchange' at the Scott nursery probably resulted from a breakdown of resistance to leaf rust which may occur in this variety at an advanced stage of maturation.

Table 2. Distribution by geographic areas of physiologic races of Puccinia recondita isolated in Canada in 1965.

Race	Geographic Area						Total Isolates	Percent of Total Isolates
	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia		
1						1	1	0.3
5	2	3	10	10	3		28	6.9
9			1		3		4	0.9
15	12	45	85	111	46	3	302	75.3
35	9	3					12	3.0
58	23	10		1			34	8.5
126		1					1	0.3
140				1			1	0.3
161		6			1	11	18	4.5
	46	68	96	123	53	15	401	100.0

Table 3. Distribution by geographic areas of NA65 races of Puccinia recondita isolated in Canada in 1965.

Race	Geographic Area						Total Isolates
	Que.	Ont.	Man.	Sask.	Alta.	B. C.	
1			1		3	2	6
3	13	25	1	16	7	9	71
7	9	3					12
9	13	23	65	74	42	1	218
10		12	29	32	1	3	77
13				1			1
19	11	5					16

Table 5. Reaction of 'Selkirk' wheat to race 15 of Puccinia recondita.

Geographic Area	Number of isolates producing indicated reaction types				
	0;	1	2	2^{+}-2^{++}	3-4
Que.	1	5	4	1	1
Ont.	13	3	9	9	11
Man.	1	3	29	27	25
Sask.	14	6	38	23	30
Alta.	6	18	15	6	1
B. C.	0	0	0	0	3
Total Isolates	35	35	95	66	71

Table 4. Percent of isolates of Puccinia recondita studied in Canada in 1965 virulent on each of the NA65 differential wheat varieties.

Geographic Area	Percent of isolates virulent on:				
	Dular	Waban	Lee	Sinvalocho	Exchange
Quebec	23.9	19.5	28.3	71.7	0.0
Ontario	7.4	4.4	51.5	48.5	12.0
Manitoba	0	0	97.9	2.1	30.2
Saskatchewan	0	0	87.0	13.0	26.1
Alberta	0	0	81.0	13.2	1.9
British Columbia	0	0	26.7	60.0	20.0

Distribution of physiologic races

Nine races of wheat leaf rust were isolated in the 1965 race survey (Table 2). Race 15 constituted 75% of the isolates in Canada and 90% of the isolates in the three Prairie Provinces. In addition, 8% of the isolates from the prairies were race 5 which is very similar to race 15 differing only by virtue of additional virulence on 'Malakof' wheat.

A somewhat greater level of variability in the rust population can be demonstrated by the use of supplementary differential wheat varieties (Table 3). However, most of the isolates from the prairies again fall into one or two races, distinguished largely by virulence or avirulence on 'Exchange' (Table 4).

It appears that the leaf rust population in the spring wheat area is remarkably homogeneous. The commercial variety, 'Selkirk', which derives its leaf rust resistance from 'Exchange' has been the main rust resistant variety in the past ten years and evolutionary changes in leaf rust have been limited to the origin and spread of strains virulent on 'Selkirk'. The present status is shown in Table 5. The 0; and type 2 reactions are conditioned by two independent resistance genes in association with avirulent isolates of race 15. The isolates producing 2^{+} to 2^{++} reactions are probably heterozygous for virulence with respect to the gene conditioning the type 2 reaction. It is not known whether the type 1 reaction results from a separate gene for resistance or is a modified reaction of one of the other two genes.