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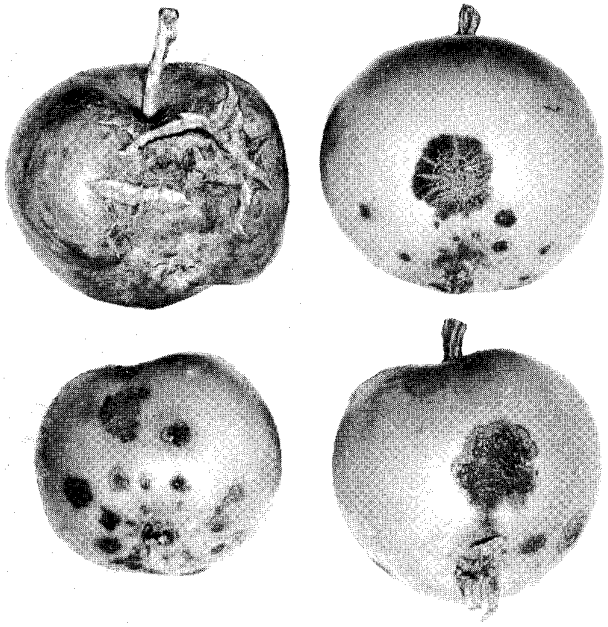

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CANADIAN PLANT DISEASE SURVEY

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OZONE DAMAGE TO TOBACCO IN CANADA[1]

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Abstract

Tobacco weather fleck has caused significant losses of flue-cured tobacco in southern Ontario since 1955. Fleck damage was greatest near the coast of Lake Erie and decreased progressively inland. Ozone has been shown to be one of the most important incitants of the fleck response in tobacco whereas parasitic fungi, bacteria and viruses were proven not to be implicated as causes of the disorder. The inherently susceptible variety White Gold, used in all tests, was rendered more susceptible by irrigation and nitrogen deficiency. It was also more susceptible during flowering and when producing lateral shoots. The degree and duration of stomatal opening were important factors in determining the amount of injury. Concentration of ozone in the air was shown experimentally to affect the speed of fleck response as well as the severity of symptoms. Statistically significant correlations between ozone concentrations and fleck damage were obtained from field data when the response of highly susceptible tissues only was considered. The merits of several visual rating methods are compared and discussed.

Introduction

Tobacco weather fleck has been a disorder of economic importance in Canada since 1955 (10, 14, 21). Its sudden appearance and increasingly wide distribution on this continent is associated with the general increase of air pollution one ingredient of which, ozone, has now been shown to be the causal agent (4, 5, 6, 8, 9, 13, 17, 20). Ozone was suggested to be the cause of weather fleck in Canada in 1957, and the Ontario Research Foundation measured air pollution in southern Ontario through the 1958 and 1959

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growing seasons (12). Fleck responses usually followed high ozone concentrations, but in general there was no significant correlation between ozone concentrations and fleck in the field (10). However, correlative information was obtained in ozone fumigation tests (8), and better field evidence was obtained with improved methods of measurement of both ozone and fleck. This causal aspect was studied in collaboration with the Meteorological Branch, Department of Transport and the Department of Health and Welfare (9, 11). Preventive measures were also intensively investigated (3, 20). This paper is presented as a background for further reports on the problem. The amount and distribution of damage in Canada, the nature of fleck, the involvement of ozone, the importance of stomata, cultural conditions and methods of rating will be discussed.

Materials and Methods

Seasonal Damage and Geographical Distribution

The severity of weather fleck damage in terms of the whole tobacco crop in southern Ontario was estimated by extension officers of the Ontario Department of Agriculture, graders of the Imperial Leaf Tobacco Co. of Canada Ltd., and research officers of the Canada Department of Agriculture. The individual estimates of leaf damage obtained by the graders for each year were totalled to provide an assessment of the extent of the overall damage. Accuracy in distinguishing this disorder from other leaf spots was increased by providing the graders with photographs of typical leaves affected with each type of spot for comparative use in the field. As a result, fairly reliable information was obtained as to the numbers of farms on which weather fleck occurred. This information was used to determine the percentage of the total acreage in each grader's territory occupied by crops showing weather fleck. The geographical distribution was obtained and compared from year to year. In addition to making a general survey of the extent of destruction of or partial damage to leaves the extension and research officers rated fleck damage at five specific locations. These were in a line from Port Burwell on Lake Erie (Fig. 1), inland. Five consecutive plants in each corner and in the center of a field at each location were rated according to the number of leaves affected and percent area of each leaf injured. In 1961, plants of the variety White Gold were grown to near maturity in pots at the Experimental Farm, Delhi, and ten were transplanted, Still potted, to each of five meteorological stations ranging inland from the lake. Individual fleck occurrences were rated at each station when possible.

Pathological Tests

For pathological tests tobacco plants, of the varieties White Gold, Hicks, and Delcrest, were collected from the Delhi and Harrow areas of southern Ontario as soon as flecking was observed. On each sampling date leaves were collected from (a) apparently healthy plants, (b) plants on which flecks had just begun to appear and (c) plants which had older lesions. The samples also included stalk and roots from both weather

flecked and healthy plants. Each sample was examined microscopically. Fresh sections were mounted in distilled water and examined for active fungus mycelium and living bacterial cells within the tissues. Sections were also stained in boiling lactophenol-cotton blue, cleared in lactophenol and examined for evidence of mycelium or bacterial cells within the flecked areas. Epidermal strips were also taken from fresh leaf samples and examined for crystalline inclusions indicative of virus infection.

Isolations were made from sections of leaves washed and plated on potato dextrose agar (P.D.A.) adjusted to pH 4.0 and P.D.A. containing 1/30,000 parts rose bengal and 5% oxgall. In other tests the tissue were surface-sterilized by treating for 1/2 to 1 minute in a 30% hypochlorite-alcohol solution, and washed 3 times in sterile water before plating. The organisms which grew on the agar plates were isolated and identified. The virus group was identified by symptoms on the leaves and by mechanical inoculation of several differential hosts: cowpea, N. glutinosa, N. samsum, and N. tabacum vars. Harrow Velvet, Delcrest, and White Gold.

Fleck Rating

A special site was selected each year within a mile of Lake Erie, and a small crop of the very susceptible White Gold variety of flue-cured tobacco was grown for the experimental study of weather fleck. The same site was used in both 1959 and 1960. The crop usually consisted of 5 ranges each having 52 rows of 24 plants, with north-south orientation of the rows. In addition, a row each of the Greek varieties Basma Serez and Ravici Drama was grown each year to indicate impending damage, since these two were the most susceptible to weather fleck, of a number of species of Nicotiana and 32 varieties of N. tabacum grown at the site in 1958.

In each experimental crop, a representative number of plants in randomly-distributed 10-plant sections of rows, was used for rating fleck damage. In the years 1959, 1960 and 1961, 50, 200 and 100 index plants, respectively, were used. All plants were topped in 1959, half of them in 1960 and none in 1961.

Several fleck rating methods were devised to appraise the severity of individual fleck occurrences for correlation with quantities of suspected casual agents. For reference purposes these are listed by number as follows:
1) Percent leaf-kill per plant. The percentage of dead tissue in the upper epidermis was estimated. Standard cards with known fractions blackened by flecks were prepared photographically. Each area of a given fleck density on a leaf was rated by matching with a card and an index for the leaf was assigned after totalling these ratings. The percentages of damage on all the leaves were summed and averaged for each plant, daily, to give the cumulative fleck rating. Fresh fleck was estimated by subtracting the previous day's rating.
2) Fleck index per plant. Fresh fleck damage, distinguishable in the early part of the day as blue-black spots in the mesophyll, was rated daily for each leaf according to an index ranging from 0 to 5. The indices were broadly

interpreted to include a composite evaluation of density of flecking and area of leaf involved. The maximum value of 5 was applied to leaves completely covered with dense flecking. This could recur more than once on a leaf. The totalled leaf indices for each day were divided by the number of plants.
3) Percent flecked area per plant. The percentage area encompassed by fresh weather flecks within each leaf was recorded. The data for each day were totalled and divided by the number of plants examined.
4) Number of leaves flecked per plant.
5) Percent plants flecked.
6) Maximum fleck index per leaf. The ten highest leaf fleck indices of the day were averaged.
7) Maximum number of leaves flecked per plant. The ten plants having the greatest number of freshly-flecked leaves on a given day were assessed for that day.
8) Percent leaf area severely flecked per plant. To assess the effects of cultural treatments on damage, accumulated fleck was rated for each leaf longer than 5 inches on each index plant at several times during growth and at the time of harvest. This rating procedure involved the assessment, on each leaf, of the percentage area that was lightly flecked and the percentage area that was severely flecked. The average density of the former was about one-fifth of the latter, so the total damage was expressed as severe fleck after appropriate computation.

Attempts were made to record the time course of development of fleck by time-lapse photography. Three camera units (19) were set up, each with a single susceptible leaf in view, before forecast fleck occurrences. The sequence of appearance of flecks were later examined in the developed film by means of an analysis projector.

Ozone Tests

Greenhouse-grown tobacco plants of the variety White Gold were experimentally fumigated with ozone. Ozone was generated by passing tank oxygen through a high voltage gap, after which it was admixed with carbon-filtered air being blown at 40 cu. ft. per min. into a 72 cu. ft.-glass-walled chamber in the greenhouse. About 25 pphm ozone was obtained in the chamber with an oxygen flow of 100 cc/min. and with a 15,000-volt transformer fed by a variable transformer set at 100 V.

Measurements of ozone polluting the air in the tobacco crop were made continuously in 1960 and 1961 in a field laboratory operated by the Canada Department of National Health and Welfare. A Kruger "72" Double Analyzer, which measured photometrically the iodine liberated by the oxidation of neutral potassium iodide, was used. The infiltration method used to measure stomatal opening has been described elsewhere (7).

Results

Seasonal Damage and Geographical Distribution

Estimates of the amount of tobacco damaged by weather fleck since 1955 are presented in Table 1. Damage was highest in 1955 and 1957, considerable damage occurred in 1961, and there was almost no loss in 1962.

Table 1. - Seasonal damage by tobacco weather fleck in Canada

Year	Damage to Tobacco (lb. x/1000)	
	Destroyed	Destroyed + Partial Damage (± 500)
1955	-	5,000
1956	1,012	3,500
1957	2,562	6,000
1958	-	2,500
1959	1,203	4,000
1960	432	4,000
1961	2,186	4,000
1962	2	500

The geographical distribution of the disorder varied considerably from year to year although some areas were regularly affected. Figure 1 comprises a series of maps of the tobacco-growing area of southern Ontario indicating the variation in the incidence of weather fleck from year to year. The circles, each centred approximately in the middle of a grader's territory, indicate the percentage of the territory's tobacco acreage in which weather fleck was observed. There were 100 to 150 farms in each territory. Figure 1 shows that the area to the southwest of Port Dover was the most severely and consistently affected. Crops close to Lake Erie were affected to a greater extent than those further inland.

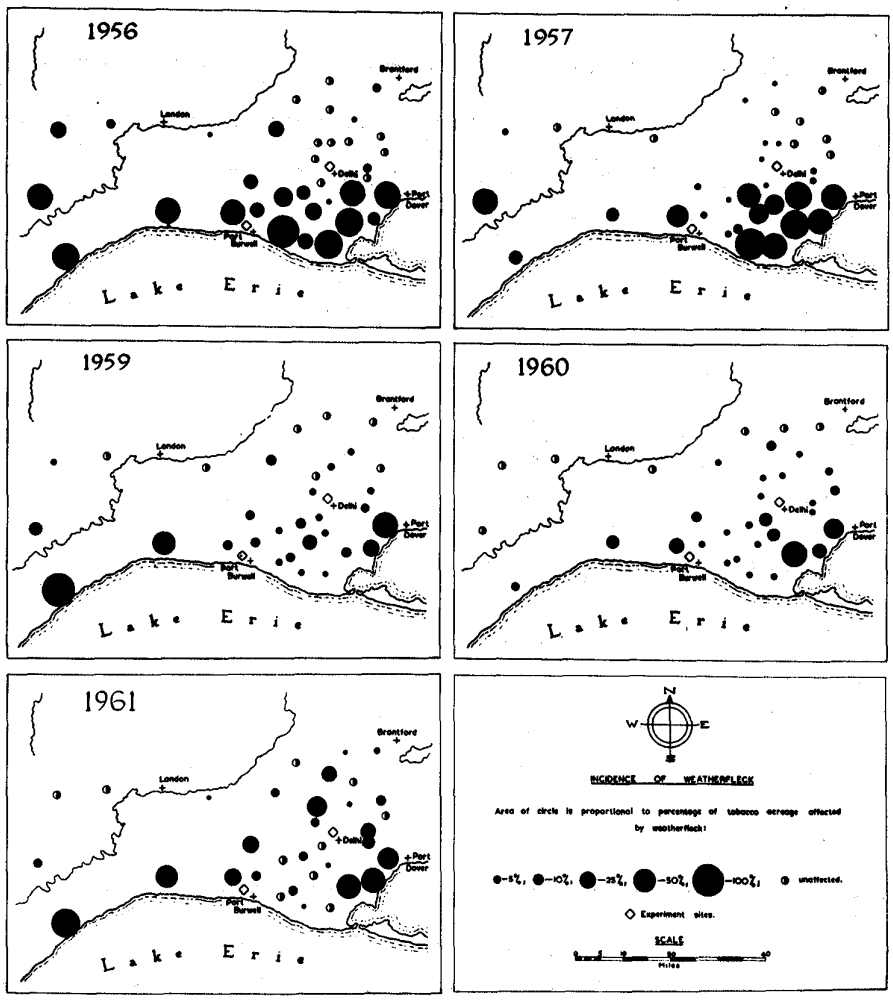

Figure 1. Geographical distribution of tobacco weather fleck in southern Ontario. Incidence of fleck represented as percentage of tobacco acreage in which fleck appeared.

The latter conclusion is substantiated by the data in Table 2. The data given for 1959 were obtained from partially-primed plants of the varieties Hicks and White Gold growing beside satellite meteorological stations (11). The data for 1960 were obtained by examining a large number of crops. Half of those closest to the lake consisted of the more tolerant variety Delcrest, but further inland a quarter to a tenth of the farms grew Delcrest. For this reason the damage was not as great close to the lake (Table 2, 1960 data). The damage from a single fleck occurrence in 1961 is also shown in Table 2. This involved the rating of 50 White Gold plants set out at the meterorological stations after growth to maturity in a greenhouse.

Damage to plants was usually heaviest on the south and west sides of fields and in outside rows. For example, 32 out of 38 growers describing north, south, east and west damage in 1958 reported most damage on the south or west sides of fields. This was statistically confirmed in 1960 (unpublished). The consideration of ozone as the damaging agent led to its identification in air meteorologically advected from the southwest (9, 10).

Table 2. - Decrease of fleck damage with increasing distance from Lake Erie.

1959		1960		1961	
Dist. (miles)	No. Flecked Leaves/Plant	Dist. (miles)	Av. Loss ($/acre)	Dist. (miles)	Attack of Aug. 12 (Fleck Index/Plant)
2	3.6	0 to 5	49	0.3	21
4	2.1	6 to 10	53	2	13
6	0	11 to 15	46	4	11
10	1.5	16 to 20	34	6	0
13	0.4	31 to 35	27	8 and 12	0

Non-Parasitic Nature

Intensive microscopic examinations, isolation experiments and tests for mechanically-transmissable viruses during several seasons proved that tobacco weather fleck is non-parasitic in origin. Flecks examined within 48 hours of their formation were mostly free of bacteria, fungi and viral crystalline inclusions. Some typical data are shown in Table 3. As the flecks become older bacteria and fungi probably increase the severity of leaf damage.

These organisms are however, assumed to be secondary. Isolation tests were made to determine whether some known plant pathogens were constantly associated with weather fleck. The results of one series of tests are given in Table 4. Many organisms known to be pathogenic to tobacco leaves were

Table 3. - Results of microscopic examinations of sections from flecked and non-flecked leaves showing relative proportions of fungi, bacteria and virus inclusions in the tissues.

Material	Number of Sections			
	Examined	+Fungi	+ Bacteria	+ Inclusions
Flecked Leaves				
a) Fresh Lesions	35	9	3	5
b) Old Lesions	35	21	12	6
Non-Flecked Leaves	25	5	1	3

Table 4. - Relative numbers of microorganisms isolated from tobacco leaves with and without weather fleck.

Material	Number			Parasites (Times Isolated)	Virus (Times Isolated)
	Isolations	Fungi	Bacteria		
Flecked Leaves					
A) Fresh Lesions	50	12	2	I Alternaria longipes (7) II Cercospora nicotianae (2) III Pseudomonas tabaci (1)	I Mosaic gp. (5) II Etch gp. (1) III Ringspot gp. (2) IV Potato Y (2) V Unk. mixt. (5)
B) Old Lesions	50	32	6	I (15) II (5) III (2)	I (3) II (2) V (10)
Non-Flecked Leaves	25	4	1	I (2)	I (3) II (1) IV (2) V (5)

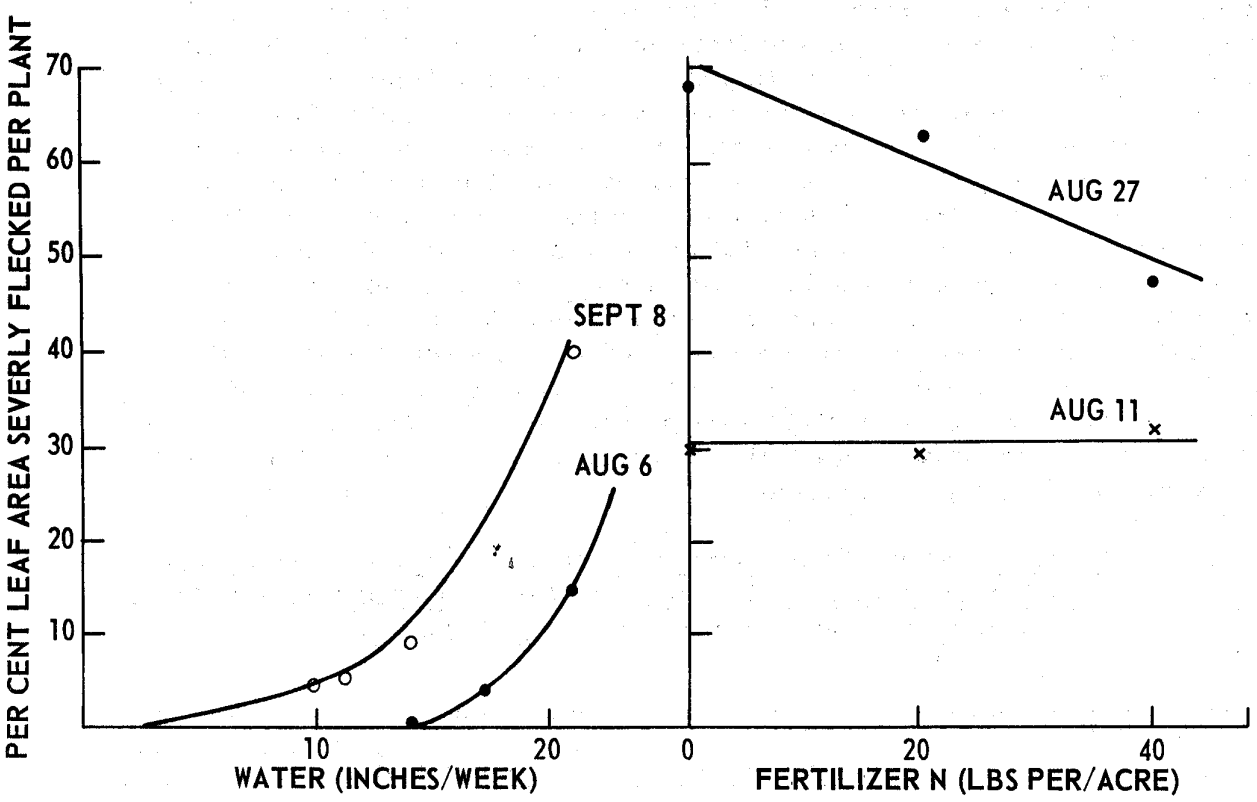

Figure 2. The effects of the amount of applied water (rainfall + irrigation), left side, and amount of fertilizer N applied with 120 lbs. P and 120 lbs. K, right side, on the amount of weather fleck accumulated on plants (by rating method number 8 in text).

associated with fleck-like areas, but many of the flecked areas also appeared to be free of microorganisms. None of the isolated organisms produced the typical weather-fleck symptoms when re-inoculated into tobacco leaves. Similarly, inoculation experiments with mechanically-transmitted viruses failed to produce these symptoms.

Influences of Cultural Practices

Information on flecking as a physiological disorder was obtained from experiments employing different cultural practices as well as from differences in varietal response (3). Experiments over 3 years revealed a direct relation between amounts of fleck and applied water (21). The relation was exponential (Figure 2). Nutrition experiments, involving different rates and proportions of commercial fertilizer ingredients, and also foliar sprays and drenches of a wide variety of elements, have established an inverse relation between applied nitrogen and amount of weather fleck (Figure 2). This effect became more pronounced later in the season. The prevention of floral development by the commercial practice of topping and suckering, or by topping and applying maleic hydrazide (MH_{30}), an inhibitor of lateral shoot growth, reduced the amount of weather fleck (Table 5). The treatments apparently acted by delaying the onset of leaf maturity (8). However, the maleic hydrazide treatment is known to adversely affect the quality of tobacco leaf, hence its use is undesirable as a control measure. The suppression of weather fleck by the foliar application of certain antioxidants (20) may also have some detrimental side effects.

Table 5. - Effects of manual and chemical sucker suppression, after topping plants of the White Gold variety, on the amount of weather fleck, as percent leaf area severely flecked (1958) and as the same per plant (1959).

Treatment	1958	1959	
	Port Burwell	Port Burwell	Delhi
None	217	26	6
Hand suckering	67	15	2
1.125 lbs. MH_{30}/acre 1 week before and at topping	2	11	0

Symptomatology

The symptom known as tobacco weather fleck (Figure 3) (1, 14, 21) can be produced by experimentally generated ozone (Figure 4) (4, 5, 6, 16, 20). However, the fleck symptom is not specific to ozone damage. A fleck response was elicited by sulfur dioxide (15, 22). We have unpublished data

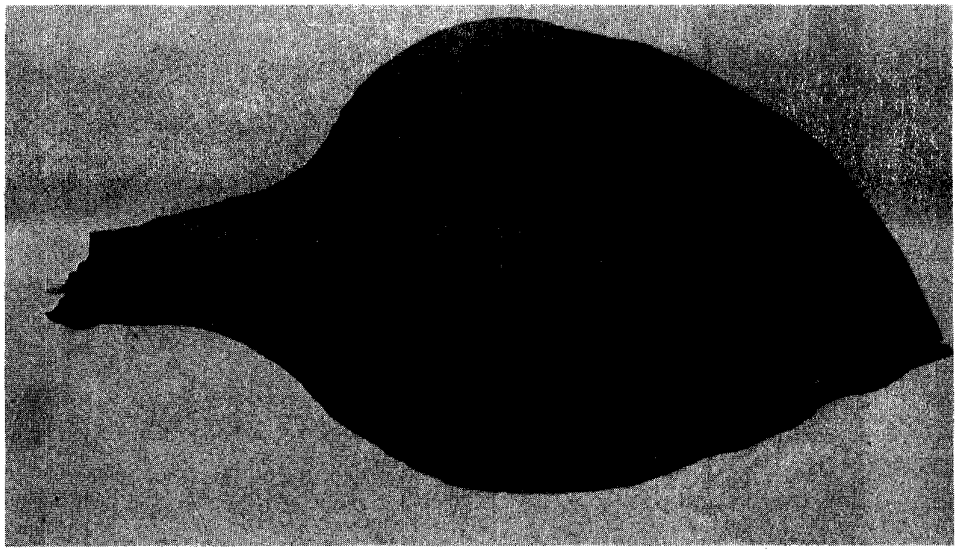

Figure 3. Tobacco weather fleck

confirming this (Figure 4). Tobacco leaves may also fleck as a result of the application of 0.01 M solutions of certain amino acids, particularly serine and alanine, or 0.1 M of alkaline ammonium acetate, either as a foliar spray or as a soil drench.

The histology of weather flecks in Canadian flue-cured tobacco has been described (14). Macroscopically the colour, size and pattern of flecks are variable. The primary lesion, consisting of a darkened (blue-green) group of palisade cells, extends to include the contiguous cells of the upper epidemis. The fleck changes through black to brown as autolysis and desication occur, and may be bleached in leaves that are sufficiently green. All colour conditions may be seen on one leaf.

Susceptibility changes occur as the leaf develops and are especially evident between the completion of expansion and the yellowing of the leaf (8). The largest individual flecks, up to 20 square mm, occur on the oldest susceptible leaves. An excessive dose of ozone produces large necrotic patches on the leaves as a result of fusion of numerous flecks (Figure 4, B).

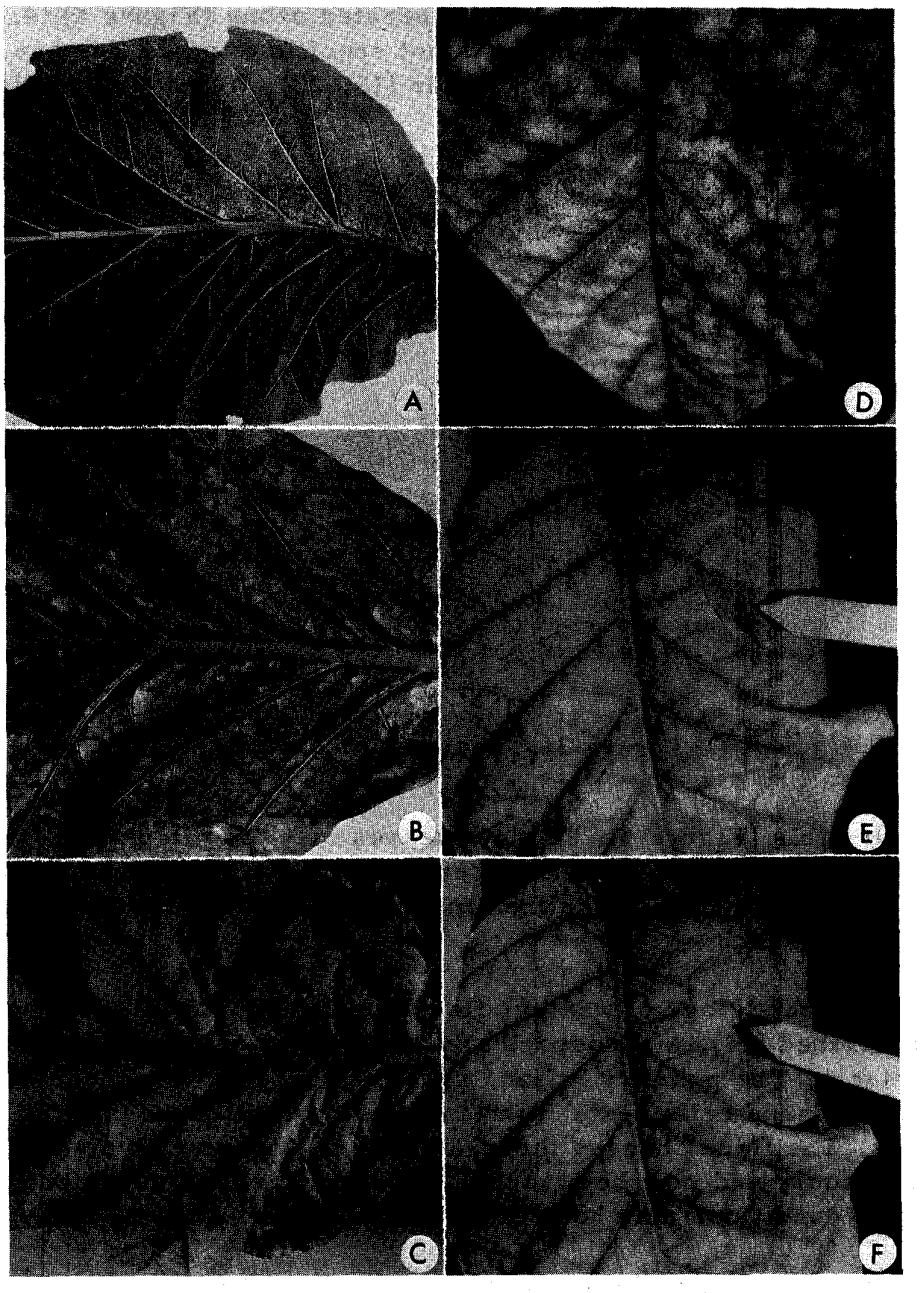

Figure 4. Damaged tobacco leaves from fumigation of greenhouse material with ozone or sulfur diozide. A) Bleached ozone flecks.B) Effect of ozone overdose. C) Ozone response of leaf having water deficit. D) E) and F) Leaves flecked by action of sulfur dioxide, showing (E and F) protective effect of overlying leaflet.

No leaf damage followed exposures to ozone under conditions inducing stomatal closure or blockage. A 6-hour fumigation with 45 pphm ozone in daylight damaged plants severely, but the same treatment in darkness was without effect. Similarly, two turgid plants that were treated with 45 pphm ozone for 6 hours developed 85% and 100% damage, respectively, whereas two wilted plants escaped injury. In the same test a wilted plant, watered before fumigation, recovered sufficient turgidity to suffer 30% damage. The flecks lined the midribs and veins of leaves under moisture-stress(Figure 4, C), and damage increased with leaf position from base to top of such a plant (Table 6).

Table 6. - Distribution of damage (Fleck Index) among the leaves of a turgid plant and a partially wilted plant fumigated with 45 pphm ozone for 6 hours.

Plant Condition	Leaf Number from Base												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Turgid	5	5	5	5	5	4	5	3	4	2	4	3	-
Partially Wilted	0	0	1	0	1	1	1	2	2	2	4	3	4

These symptoms differed from the turgid plant's more heavily-damaged mature leaves and intercostal leaf-damage. The protective effect of a leaf overlapping another, attributed to stomatal closure (4), was a common observation in the field and in experiments with ozone or sulfur dioxide (Figure 4, E and F). A plastic bag over a leaf or a tent over a plant (9) also afforded protection from air-polluting ozone. Using impervious coatings on leaf surfaces we observed that, as in natural weather flecking (4), experimentally-generated ozone entered the leaf primarily through the lower epidermis. Since lower guard cells were considerably more responsive to light and other stimuli than those in the upper epidermis (7) the accumulation of a damaging dose of ozone depended primarily on the lower epidermal stomata. Stomatal response to the microenvironment, including sunlight (Table 7), may be responsible for the occasionally-observed concentrations of weather fleck on one side of a row of tobacco plants.

Table 7. - Relation between damage (Fleck Index) by ozone and amount of leaf directly illuminated during fumigation with 35 pphm O_3 for 8 hours.

Leaf number from base	1	2	3	4	5	6	7	8	9	10
% Leaf in direct sunlight	100	0	100	0	100	100	0	100	50	50
Damage Index	2	1	4	0	1	4	0	4	0	2

There was a time lag between fumigation of the plants with ozone and the appearance of the fleck symptom (Figure 5). Flecking was characteristically first observed in the morning following the day of fumigation. Moreover, flecks appeared most quickly at the higher dosages. Figure 6 shows counts of fresh flecks in parts of a single leaf during three successive fleck occurrences of increasing severity. The data were obtained from a time-lapse photographic film. The fleck index per plant of 20 contiguous plants was 17, 18 and 20 on July 18, 19 and 23 respectively. The data show that the sooner the flecks appear the more damage to the plant. On days of heavy air pollution, very susceptible leaves developed visible flecks before dark. Statistical support for the time lag between dose and response was found in the correlation coefficients for weather fleck on ozone dose, for various time lags (Table 8). The effect of a day's pollution was measured routinely on the next day. In chamber experiments with greenhouse-grown tobacco plants the amount of ozone damage increased with the dosage until stomatal closure interfered. Typical results are given in Table 9. In agreement with observations on weather fleck, the time between removal from fumigation and the appearance of symptoms decreased with increasing dose.

Comparison of Methods for Rating Fleck

The correlations between methods of rating fresh weather fleck and ozone incidence are given in Table 10. The lack of any significant correlation in 1959 is attributable to inaccurate ozone and flecking data. The complexity of the rating of accumulated fleck, as percent leaf tissue killed, caused a large error, such that some of the values for fresh damage, obtained by subtracting the day to day cumulative values, were negative. Also, the changing susceptibility of the growing crop was not taken into account in 1959.

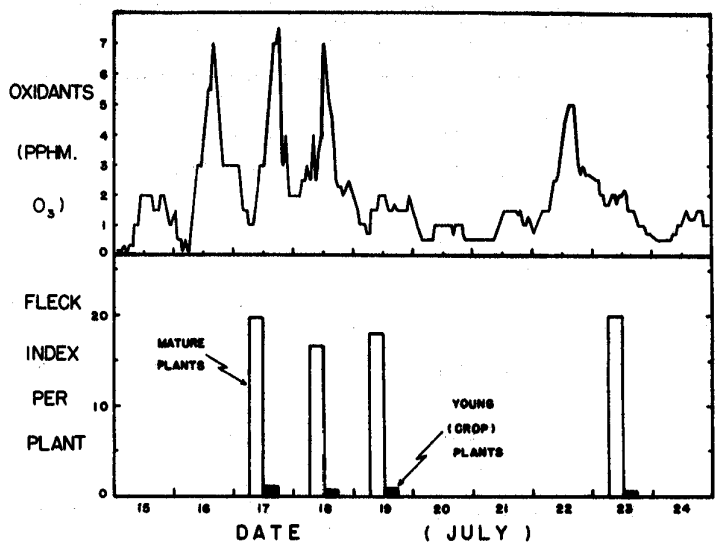

Figure 5. The weather fleck response of tobacco plants (histograms) in relation to high concentrations of ozone (upper curve). Data of 1960 at Port Burwell site.

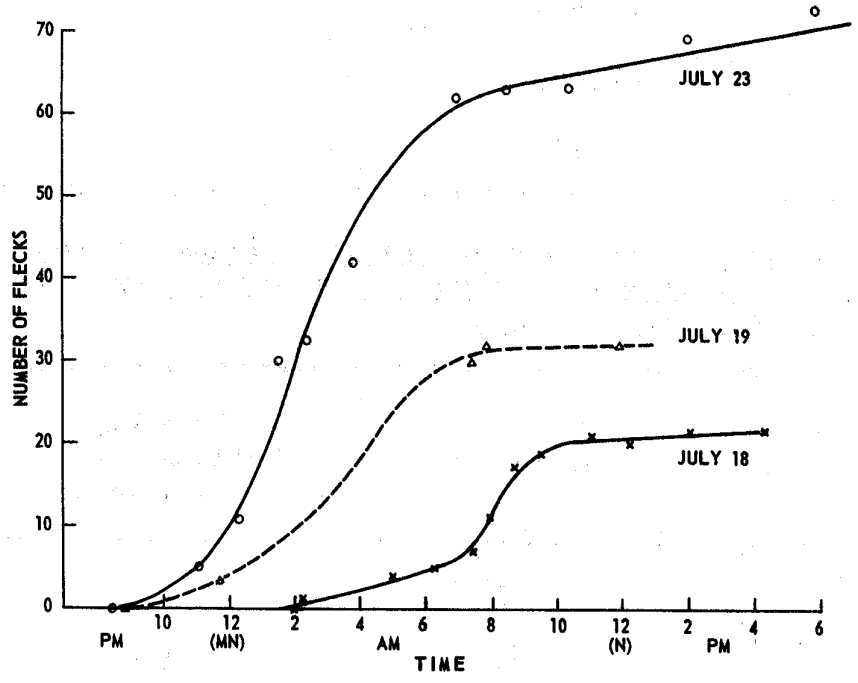

Figure 6. The time course of the appearance of weather flecks on parts of a single leaf subjected to three successive attacks of increasing severity. Data from time-lapse photography.

Table 8. - Correlation coefficients of daily weather fleck (Max. Fleck Index/-Leaf) on daily ozone dose (pphm-hours), for successive time lags from the day of fleck appearance. 1960.

Time Lage Ozone Dose to Fleck (days)	Correlation Coefficient	Degrees Freedom	Significance
5 before	+ 0.167	66	
4 "	- 0.273	67	* $0.01 < P < 0.05$
3 "	- 0.254	68	* "
2 "	- 0.076	69	
1 "	+ 0.648	70	*** $P < 0.001$
0	+ 0.262	71	* $0.01 < P < 0.05$
1 after	- 0.087	70	
2 "	- 0.247	69	* "
3 "	+ 0.008	68	
4 "	+ 0.009	67	
5 "	+ 0.145	66	

Table 9. - Effects of increasing time of exposure to 45 pphm ozone, on the damage (% destruction of plant) and on the time required for the first appearance of damage.

Time of Exposure (minutes)	% Damage	Time-lag End of Exposure to Visible Symptoms
30	3	1 day
75	70	1 hour
135	75	10 mins.
240	90	0 mins.

Table 10. - Correlations between data on weather fleck, rated daily by different methods, and daily ozone dose for three different seasons at experimental sites near Port Burwell.

Rating Method (see text)	Correlation Coefficients and Significance 1959	1960	1961
1) % Leaf Kill/Plant	- 0.106	--	--
2) Fleck Index/Plant	--	0.257*	0.245
3) % Leaf Area Fld./Plant	--	0.217	--
4) No. Leaves Flecked/Plant	0.103	0.293*	0.266
5) % Plants Flecked	0.007	0.410***	0.220
6) Max. Fleck Index/Leaf	--	0.468***	0.315*
7) Max. No. Leaves Fld./Plant	0.132	0.381***	0.262
Degrees Freedom	48	70	42

* $0.01 < P < 0.05$. ** $0.001 < P < 0.01$. *** $P < 0.001$.

Table 11 - Correlation coefficients and significance of the relationship between daily ozone doses and maximal fleck indices for two seasons with and without divisions into two time classes.

Date	Correlation Coefficient	d.f.	Significance
1960 Data			
July 12-Aug. 15	+ .814	33	*** $P < .001$
Aug. 16-Sept. 22(two points omitted)	+ .599	34	*** $P < .001$
Total (July 12-Sept. 22)	+ .648	70	*** $P < .001$
1961 Data			
Aug. 1-Aug. 18 (two points omitted)	+ .548	14	* $.01 P < .05$
Aug. 20-Sept. 13	+ .567	20	** $.001 P < .01$
Total (Sept. 1-Sept. 13)	+ .315	42	* $.01 P < .05$

Judging from the significance of correlation in 1960, the simplest methods for evaluating fleck (Table 10, methods 4 and 5) were as useful as the more detailed ones (Table 10, methods 2 and 3). There was no significant correlation of ozone with any of these sets of rating data in 1961. However, when the change in susceptibility of the crop was ignored and only the most susceptible leaves considered (Table 10, method 6), the correlation coefficients were improved to more significant levels. Further improvement of correlations resulted when on the basis of statistical and graphical examinations of the data, the seasons were divided into 2 time classes (Table 11).

The use of mature plants for rating individual fleck occurrences early in the season, as illustrated in Figure 5, is a practical way to evade the ontogenetic change in susceptibility of the crop. Very significant seasonal correlations were obtained in 1960 by rating July damage on a set of mature, greenhouse-grown plants transferred to the field in June.

Discussion

The first occurrence of weather fleck in epidemic proportions in 1955 resulted from increasing pollution of air, widespread use of susceptible tobacco varieties, changed cultural practices, and appropriate combinations of meteorological factors. A reversion to the more tolerant variety Delcrest may partly account for the subsequent decrease in severity, but the continued increase of air pollution has caused an increasingly wide distribution of damage. The latter effect has been especially evident in areas beyond those covered in Figure 1. The inland dilution of damage reflects the lifting and breaking of pollutant-retaining inversions, as a result of convection currents from the land (11). The geographical and local distributions of fleck damage thus appeared to result from the effects of meso-and micro-meteorological factors on the dissemination of the catalysts of ozone formation.

Daily ratings of weather fleck were required for the study of its relation to environmental factors. The rating methods used initially were cumulative; the day to day differences representing fresh flecking. However, a full index describing a severely-flecked leaf excluded a record of future attacks to that leaf, thus accentuating the exponential relationship between damage and visual rating (18). Therefore, an attempt was made to obtain daily estimates of the actual amount of dead tissue (method 1). This method was tedious, time-consuming and subject to errors that were on the same scale as the daily damage. These methods were abandoned in favour of rating fresh fleck only. A simple index, including severity and area-coverage (method 2), was applied and further simplifications proved to be still better measures of response to ozone doses. However, the most detailed method of rating fresh weather fleck, the leaf fleck index per plant, provided the best data on crop response from which to select maximal daily damage in order to evade the problem of changing susceptibility. The maximal fleck index, being the average index for the 10 most severely-

damaged leaves on each day, represented the most susceptible tissue only, the nature of which was assumed to be the same in that respect from beginning to end of the season. The best correlations between fleck and ozone dose were obtained with this expression of flecking when the seasons were divided into 2 time classes. A physiological basis is implied by the coincidence of the time classes with the vegetative and flowering phases of development of the crop.

The correlations obtained between rated damage and ozone dose, particularly in 1960, were surprisingly good considering the day to day anomalous effects wrought by internal factors of susceptibility (8) and by external micrometeorological factors (9), and also considering the non-linear relations between visual damage ratings and damage per se (18).

Substitution of the crop by successive sets of identically grown mature plants, in successive intervals of time through the season, would provide the ideal material for the rating of fleck occurrences. Some success was met with this method in 1960, but repetition in 1961 was not successful as the greenhouse-grown plants of that year were much more tolerant. The conditions of preliminary growth required for maximum susceptibility deserve to be worked out.

As a result of many experiments in the early stages of this study a number of factors were tentatively excluded from a causal role in weather flecking. These included pathogenic microorganisms, certain classes of virus, soil type, natural gas and root exudation. However, it was early recognized that many factors are involved in the final expression of weather fleck, many of which are still not completely understood. These factors influence one or both of the two requirements, namely the susceptibility of the plants (8) and the toxicity of the air (9). Field and laboratory experiments strongly supported the thesis that ozone and its reaction products are the initiating agents of the weather fleck response. The concentration of ozone required to bring about the fleck response appears to be dependent on the physiological condition of the host and particularly of the leaf tissue. On one day a relatively low concentration of pollutant brought about injury while on another a much higher concentration was required. This cast some doubt on the causative role of ozone in the field. Nevertheless, with the use of especially defined parameters of dose and of damage, statistically good correlations have been obtained between ozone and weather fleck. Daily variations are attributable to the influences of micrometeorological factors on ozone flux (9) and stomatal opening. The importance of the latter has been unduly criticized (2). The variety of the tobacco and the nutrition and water economy of the plant are primary factors in the development of the physiological state of susceptibility (8).

Acknowledgements

The authors wish to thank Mr. A.F.W. Cole and Dr. Morris Katz, Director, Environmental Assessment, Department of National Health and Welfare for the use of data from continuous ozone measurements for the seasons of 1960 and 1961, and Mr. E.I. Mukammal and Dr. C.C. Boughner, Director, Climatology Division, Meteorology Branch, Department of Transport for associated meteorological studies. We gratefully acknowledge the work of Messrs. E.K. Walker, J.M. Elliot, and E.C. Birch, Department of Agriculture, on irrigation, nutrition and suckering experiments, respectively. Our thanks are also due to many others involved in this project for their collaboration and assistance.

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A SIMPLE DEVICE FOR EMULSIFICATION OF VIRUS PREPARATIONS WITH FREUND ADJUVANT

W.M. Haigh and J.H. Tremaine

The use of the Freund adjuvant to obtain high titered antisera with many plant viruses was described by Wetter (3) and Moorehead (2) but neither describes methods for emulsification of virus preparations with the adjuvant. A common method consists of placing the virus preparation and the adjuvant in a beaker and drawing and expelling them with a syringe until a suitable emulsion is obtained. Desjardins and Wallace (1) employed a vibrator-type mechanical shaker to obtain an emulsion. The present communication describes a simple method for emulsification employing a common laboratory stirrer. This device has been used in most serological work at this laboratory.

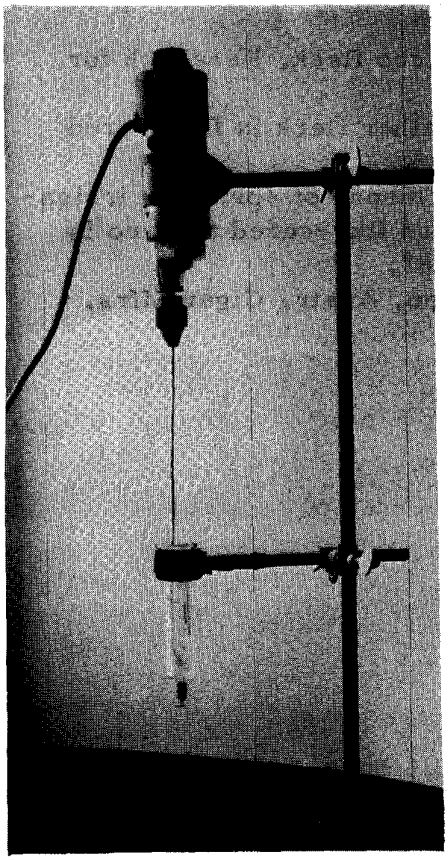

Fig. 1. Apparatus for emulsification of virus preparations with Freund adjuvant (see text for description).

A flexible shaft consisting of the handle of a test-tube brush was inserted into a piece of 4 mm-diameter, tygon tubing. The end of the tubing was sealed by heating under a low flame and pinching it with forceps. The shaft was inserted into a common laboratory stirrer, the end of shaft bent slightly, and inserted into a syringe (Fig. 1). Another short piece of tygon tubing was sealed in the same manner and placed over the tip of the syringe. The Freund adjuvant was placed in the syringe and the stirrer started. The adjuvant whirled up the sides of the syringe through the action of the shaft. The virus prepared was added so that it ran slowly down the side of the syringe while the stirrer was in operation. An emulsion formed immediately upon addition of the virus preparation but the emulsion was stirred for approximately three minutes. After stirring was completed the shaft was removed and emulsion clinging to it was wiped off with the finger and placed in the syringe. The plunger was placed in the syringe and the syringe inverted. The tygon tip was replaced with a needle and the syringe plunger pushed in to expel any excess air. Animals were injected directly with the same syringe to avoid handling the emulsion. The size of

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Canada Department of Agriculture, Vineland Station, Ontario, Canada.

syringe employed depended upon quantities of virus preparation available; generally a 10 ml-syringe was used with 1 cc of virus preparation.

The emulsions produced with this device are extremely stable and have been kept in the refrigerator for four weeks without visible signs of breaking. Some advantages are: use of common laboratory articles; its speed and the avoidance of excessive handling of emulsions.

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CANADA AGRICULTURE
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RELATION OF SPRING DROUGHT, SUMMER RAINS, AND HIGH FALL TEMPERATURES TO THE WHEAT STREAK MOSAIC EPIPHYTOTIC IN SOUTHERN ALBERTA, 1963

T.G. Atkinson[1] and J.T. Slykhuis[2]

Abstract

A severe epiphytotic of wheat streak mosaic on winter wheat developed during the fall of 1963 in areas of southern Alberta where a severe spring drought delayed the development of spring grains until after heavy rains fell late in June. The resulting predominance of late-maturing spring wheat and barley that harboured the virus and its vector (Aceria tulipae K.) until late September, together with record-breaking warm weather throughout September and October favored the exceptional spread and severe development of the disease even on winter wheat sown when normally recommended.

Introduction

The most severe outbreak of wheat streak mosaic ever known to occur in the winter wheat area of southern Alberta prior to freeze-up developed during the fall of 1963. This outbreak was restricted to the eastern portion of the winter wheat growing area. Surveys during October revealed that around Claresholm, Barons, Granum, Lethbridge, Wrentham, and Warner, wheat streak mosaic was more prevalent and severe than it had ever been at that time of year since this disease was first correctly diagnosed in 1952 (3). In contrast, no severely infected crops were found in the Spring Coulee, Cardston, Glenwood, or Pincher Creek districts (Fig. 1).

The development of the disease in the eastern districts can be described as a major epiphytotic. Most early-sown fields of winter wheat were uniformly yellowed by the disease in mid-October and many plants were dying by the end of the month (Fig. 2). Winter survival in such crops is expected to be low, and even surviving plants are not likely to set seed. Wheat sown during the first two weeks of September, normally the recommended period (1, 2, 4), was also severely infected and many fields will produce little or no grain.

This report describes the unique succession of unusual weather conditions, beginning in the spring and continuing into the fall, that determined the localization and allowed the development of this unprecedented epiphytotic.

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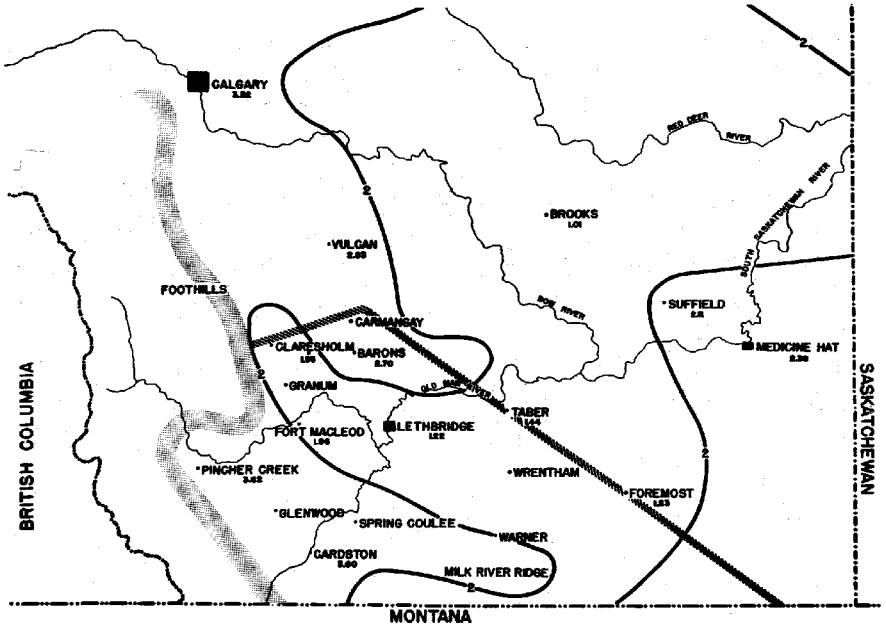

Fig. 1. Distribution of total precipitation, April 1-June 17, 1963, in southern Alberta. Winter wheat production is concentrated in the area bounded by the cross-hatched lines, foothills, and Montana border. Notice how the two-inch isohyet divides this area into eastern and western districts.

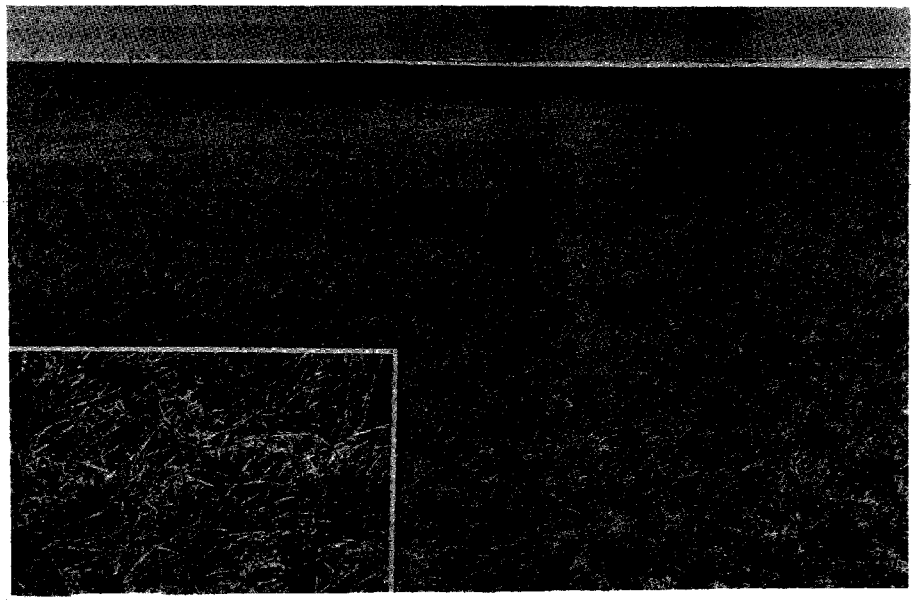

Fig. 2. Uniformly severe wheat streak mosaic symptoms in a field of winter wheat sown August 27, 1963; photographed October 18, 1963. The diseased wheat appears light in contrast to the unaffected wild oats in the center background. Insert shows portion of the same field.

Conditions Promoting the Epiphytotic

Effects of spring drought

The sequence of weather events that provided ideal conditions for the development of an epiphytotic began with a severe spring drought. Throughout all but the foothills area of southern Alberta, spring precipitation in 1963 was much below normal. In the most severely affected area, including the eastern portion of the winter wheat zone, precipitation between April 1 and June 17 totalled less than two inches (Fig. 1). Here, stands of spring-sown grains were extremely sparse and uneven because the soil was so dry that seedlings did not tiller and, in many instances, seed did not germinate. Even volunteer wheat was scarce. In the western part of the winter wheat zone, however, the germination and early development of spring grains followed a normal pattern.

Effects of summer rains

The effects of the severe drought were dramatically reversed by heavy rains on June 21, 22, and again a week later. During that period total precipitation over the winter wheat area averaged more than 4.5 inches. In the drought area, seed of spring wheat and barley that had lain dormant in the dry soil now germinated more than a month late, and the stunted plants in sparse stands tillered abundantly. In addition, late seeding of barley was common as farmers sought to take advantage of the renewed soil moisture. Above-normal precipitation throughout July and near-normal rainfall during August promoted the vigorous development of this new growth and, by reducing the effectiveness of summerfallow operations, permitted the extensive and profuse development of volunteer wheat.

Although surveys in mid-June had revealed an extremely low incidence of wheat streak mosaic throughout the area, these late-developing wheat and barley crops and volunteer plants became massive reservoirs of inoculum as the virus and its mite vector multiplied rapidly during the summer and spread throughout the area. Consequently, in the eastern zone where extensive acreages of wheat and barley did not mature until late in September, winter wheat sown during the first two weeks of September, the normally recommended time, became heavily infected.

Effects of a prolonged warm fall

At Lethbridge, the mean temperature for the month of September was 62° F, almost 10° higher than the 30-year normal. Unseasonably high temperatures continued in October both before and after the first killing frost on October 19, twenty-four days later than the 61-year average date. While this weather was ideal for harvesting the late-maturing crops, it allowed these potent sources of inoculum to be effective until they finally matured in late September. The prolonged period of exceptionally warm weather favored the continued multiplication and dispersal of viruliferous mites throughout the winter wheat fields and was responsible for the unprecedented development of symptoms prior to freeze-up.

Effectiveness of Control Measures

Wheat streak mosaic becomes a serious problem only if the virus and its mite vector are provided with a continuous supply of living host plants, principally spring and winter wheats. Because winter wheat plays a key role in the disease cycle, control recommendations call for the elimination of all sources of infection before the fall crop is sown (Fig. 3). This is achieved by seeding winter wheat only after all spring wheat and barley in the vicinity have matured and by destroying volunteer hosts on or near the field.

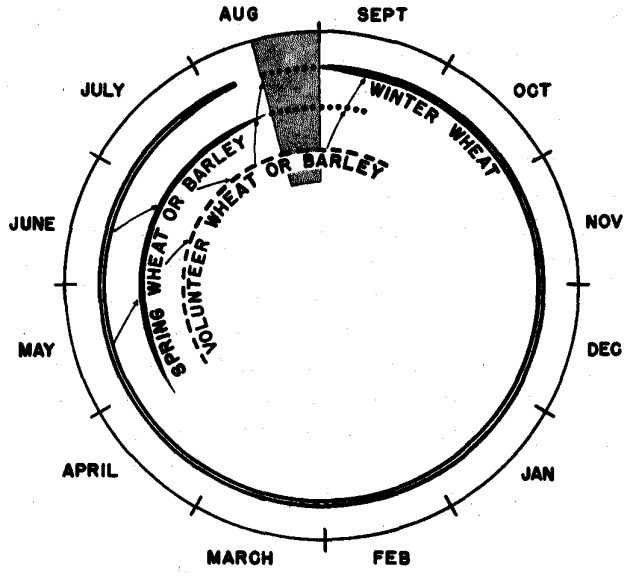

Fig. 3. Wheat streak mosaic disease cycle. Control depends upon preventing an overlapping sequence between spring hosts and winter wheat. Shaded area represents period during which effective control is normally achieved. Dotted lines indicate problems presented by early-seeded winter wheat and-or late-maturing spring wheat or barley. Arrows represent mite transfer of virus.

In southern Alberta, spring-sown crops normally mature by mid-August so infection from this source is avoided when winter wheat is sown during the first two weeks of September. This is the planting period recommended for obtaining the most winterhardy crops (1, 2). Control then depends upon the destruction of volunteer wheat or barley. Farmer experience in southern Alberta has strikingly demonstrated the effectiveness of these cultural control practices.

In 1963, the combination of late-maturing crops and a prolonged warm fall complicated effective control of wheat streak mosaic. However, farmers who heeded the special warnings and followed the recommendations

issued by the Lethbridge Research Station early in August generally avoided serious infection of their winter wheat. Although the problem of late-maturing spring crops was not as acute in the western as it was in the eastern portion of the winter wheat zone, farmers in the Spring Coulee-Cardston districts, who often seed earlier than recommended, undoubtedly avoided serious infection this year by uniformly delaying their seeding of winter wheat. In contrast, although similar conditions existed in the Barons district, some fields were severely infected because they were seeded before the end of August (Fig. 2). Around Lethbridge, where the epiphytotic was most severe, the only winter wheat that escaped serious infection was that sown after spring grains matured and away from diseased volunteer or early-sown winter wheat.

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YELLOW-LEAF CONDITION OF UNKNOWN CAUSE ON OATS IN ONTARIO

F.J. Zillinsky[1] and J.T. Slykhuis[2]

A severe leaf yellowing appeared in early June on oats in at least 12 counties in southwestern Ontario. In co-operation with staff of the Department of Crop Science, Ontario Agricultural College, Guelph, affected oat fields were examined on June 4 and 5. Tests were made to determine the cause, and some fields were re-examined during the last week of June and on July 22.

On June 4 and 5 the yellowing was severe in scattered, irregular patches varying from a small group of plants to extensive areas in oat fields seeded before May 1. Plants in the affected fields were in the 3 and 4 leaf stage. The most severely affected fields were entirely yellow except for isolated patches of green plants. The youngest leaves of the affected plants were the least chlorotic, being yellow to almost white toward the tips or in blotches and streaks, but green toward the base. Older leaves were progressively more severely chlorotic. On some plants the lowest leaves had dried to a light brown color. Darker water-soaked blotches were also present on some of the lower leaves. The symptoms resembled barley yellow dwarf on oats except that there was no bronze or reddish discoloration and leaves did not stand erect. Macrosiphum avenae (F.), one of the known important vectors of barley yellow dwarf virus in Ontario, was present in low numbers in most fields. The condition occurred on most oat varieties grown in the area, including Rodney and Garry, but no similar symptoms were found on barley even when mixed with oats in the same field. Halo blight symptoms occurred in some fields but they did not appear to be specifically associated with the severe leaf yellowing. There did not appear to be any correlation between the yellow leaf condition and the previous crop, cultural practices, topography, soil type, nematodes, fertilizers or herbicidal sprays.

Samples of diseased plants and aphids were collected and tests were made to determine if either barley yellow dwarf virus or bacteria were the cause of the leaf yellowing. Although barley yellow dwarf virus was transmitted by some of the aphids collected in the field, the virus was not transmitted by non-infective Macrosiphum avenae (F.), Rhopalosiphum padi (L.) or R. maidis (F.) fed on leaves with the yellow-leaf condition. Two types of bacteria were isolated from diseased plants[3]. One isolate induced halo-blight symptoms on oat plants, but neither isolate induced symptoms

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resembling the disease seen in the field.

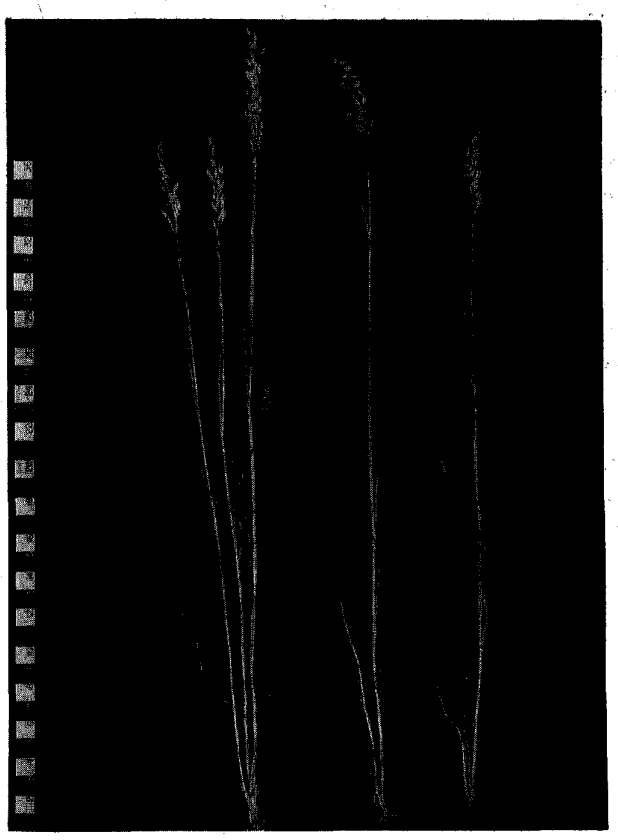

Figure 1. Mature oat plants showing effects of the yellow-leaf condition first observed in the 3-to 4-leaf stage. The first tiller, which was severely chlorotic on June 4, died, but the later tiller did not develop symptoms and grew to almost equal the tillers on adjacent non-affected plants (left).

When the oat fields were re-examined during the last week in June, yellow patches were no longer visible. The affected leaves and early tillers had withered and darkened but had become obscure under a growth of new green leaves. The new tillers were free from the chlorotic symptoms, but the affected plants were shorter and less vigorous than plants in other areas of the fields.

By July 22 when the oats had headed and were nearing maturity the early tillers on the affected plants were dead and dried out, but the later tillers appeared to be developing normally without the blasting which occurred on the early tillers (Fig. 1).

Discussion

Although the yellow-leaf condition on oats in Ontario was initially attributed to a strain of barley yellow dwarf virus, the inability to transmit the condition with three known vectors of the virus and the failure of symptoms to develop on new tillers during the warmer weather in June, discouraged this hypothesis. Halo blight and other mottle symptoms were present on many of the affected plants but none of the bacteria isolated from diseased plants appeared to have a direct relation to the yellow-leaf condition.

Similar conditions have been reported in the U.S.A. Yellowing symptoms that developed on winter oats in South Carolina, Georgia and Virginia soon after growth started in the spring were attributed to such varied causes as barley yellow dwarf virus, Helminthosporium sp., halo blight and weather conditions (1, 2, 3). In a personal communication, Dr. H.C. Murphy of Beltsville, Md. reported that an outbreak of leaf yellowing occurred on spring oats in Iowa in 1955 and was at first suspected to be caused by barley yellow dwarf, but virus transmission by aphids was not obtained. Halo blight was found among affected plants but was also ruled out and the cause was not determined.

A common characteristic of all these conditions appears to be that they developed early in the spring. The yellow-leaf condition in Ontario was severe only on early-sown oats and developed during the 3- and 4-leaf stage. It failed to progress further even on affected plants when the weather became warm later in June, and it did not appear on oats sown after May 1. Regardless of the cause, it appears that temperature and possibly other environmental factors are of critical importance to the development of the disease.

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A SURVEY OF LEAF AND HEAD DISEASES OF BROMEGRASS IN SASKATCHEWAN, 1963

C. Noviello[1]

Brome, Bromus inermis Leyss., is the most important forage and pasture grass in Saskatchewan. A rough estimate would place the acreage at 1,000,000. A field survey of leaf and head diseases of bromegrass was carried out during the period May to September in 17 localities of the province and a total of 48 fields were examined. When necessary, diagnoses were confirmed by isolation, in pure culture, of the causal organism. No attempt was made to identify the virus diseases, undoubtedly present. The aim of the survey was to determine the most important brome diseases in the province.

Table 1. - Prevalence and severity of brome disease in 48 fields

Disease	S	M-S	M	L	Tr	Total
Selenophoma leaf spot	9	5	22	6	3	45
Leaf blotch	2	-	4	2	2	10
Bacterial blights	-	1	6	1	-	8
Scald	-	-	1	-	4	5
Ergot	1	1	-	1	1	4
Powdery mildew	-	-	-	-	2	2
Septoria leaf spot	-	-	-	2	-	2

S = severe; M-S = moderate to severe; M = moderate; L - Light; Tr = traces

SELENOPHOMA LEAF SPOT (Selenophoma bromigena) was the most prevalent disease. Only 3 fields out of the 48 inspected were found free of this disease. The three fields were all in the Nipawin area where the disease was found in trace amounts in two other fields. The disease appeared early in May and reached a peak in July. In 9 fields at 5 different localities (Melfort, Regina, Saskatoon, Unity, Zealandia) the disease was very severe and not only was the leaf blade found affected but also the sheath, stem, pedicels

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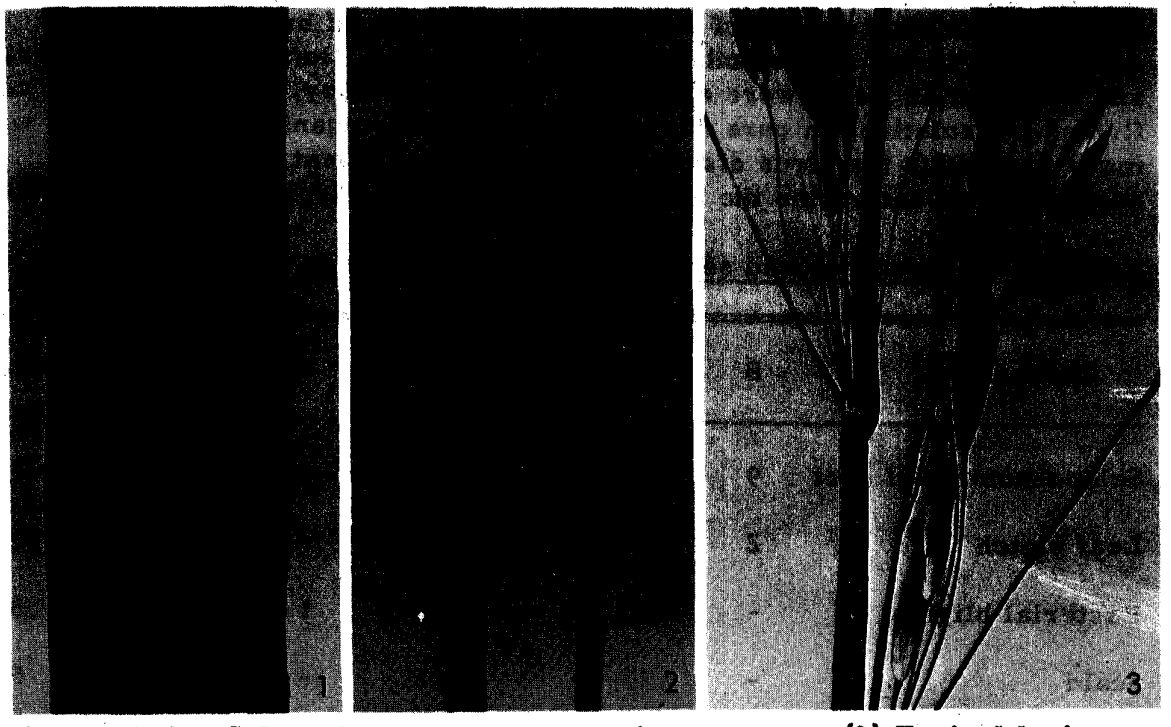
and panicles (Figures 1-3). A remarkable difference in susceptibility was observed among the bromegrass clones in the experimental plots at Saskatoon. Single-spore cultures of wild type isolates from different localities exhibited a great deal of variability in gross morphology. The disease affected the yield (hay quality and seed production) in a significant way and therefore has considerable economic importance. Burning or cutting the stubble early in the spring seems to be a good control measure.



Figures 1-3. Selenophoma symptoms on bromegrass. (1) Typical lesions on leaf blade showing pycnidia in rows.

(2) Lesions on sheath and leaf blade.

(3) Lesion on the inflorescence, especially on pedicels.

LEAF BLOTCH (Drechslera bromi) is a rather common disease of Bromus inermis and was observed in 10 fields at 5 localities (Chamberlain, Melfort, Nipawin, Regina, Unity), in varying degrees of intensity. First symptoms appeared early in June at the Melfort Experimental Farm and subsequently the disease was observed throughout the season.

BACERIAL BLIGHTS (Pseudomonas coronafaciens var. atropurpureum and Xanthomonas translucens f. sp. cerealis). Stripe blight incited by X. translucens f. sp. cerealis was observed in experimental plots at Saskatoon and in one field near Unity where plants were heavily infected. Light to moderate infections of bacterial blight incited by P. coronafaciens were observed in 6 fields at 4 different localities (Craik, Regina, Saskatoon, and Unity).

SCALD (Rhynchosporium secalis) was encountered in 5 fields at 3 localities (Nipawin, Saskatoon, Unity) but the disease was moderate in one field only (Saskatoon).

ERGOT (Claviceps purpurea) was encountered in 4 fields, of which one near Codette was very heavily infected.

POWDERY MILDEW (Erysiphe graminis). A very light infection was observed in two fields in the Unity area, late in July.

SEPTORIA LEAF SPOT (Septoria bromi) was encountered in light amounts in two fields at two localities (Prince Albert and Saskatoon).

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AIR-BORNE RUST INOCULUM OVER WESTERN CANADA IN 1963

G. J. Green[1/]

The amount of rust inoculum in the air over western Canada in 1963, relative to earlier years, was estimated by trapping urediospores on vaseline-coated microscope slides exposed in spore traps. The slides were exposed for 48-hour periods at Winnipeg, Morden and Brandon in Manitoba and at Indian Head, Regina and Saskatoon in Saskatchewan. The slides were examined at Winnipeg, excepting those exposed at Saskatoon which were examined at the Plant Pathology Section, Canada Department of Agriculture Research Station, Saskatoon. The number of spores caught on each slide is shown in Table 1.

The spore trappings indicate that the first spore shower, which deposited spores across Manitoba and southern Saskatchewan, began in Manitoba on June 6-7, but the finding of wheat leaf rust at Winnipeg on June 11 suggests that some spores were present about June 2-3. Another spore shower began on June 18-19 and deposited additional spores across Manitoba and southern Saskatchewan. Locally produced spores probably began to appear on the slides at about this time. Stem rust spores were scarcer during June and more abundant during July than for comparable periods in 1962. Leaf rust urediospores were more abundant throughout the growing season than in the 3 preceeding years.

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Table 1. Numbers of urediospores of stem rust and leaf rust caught on vaseline-coated slides exposed for 48 hour periods at three locations in Manitoba and three locations in Saskatchewan in 1963.

Date	Winnipeg		Morden		Brandon		Indian Head		Regina		Saskatoon [1]	
	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	0	0	0	0	0	0	0	0	0	2 [2]	0	0
May 31 - June 1	0	0	0	0	0	0	0	0	0	2	0	0
2-3	0	0	0	0	0	0	0	0	0	0	0	0
4-5	0	0	0	0	0	0	0	0	0	0	0	0
6-7	0	0	6	17	1	2	0	0	0	1	0	0
8-9	0	0	0	0	1	1	0	0	-	-	0	0
10-11	2	1	0	1	1	0	1	1	0	19	0	2
12-13	1	1	2	15	0	1	1	9	4	23	0	1
14-15	0	1	0	2	0	2	0	4	0	2	0	0
16-17	0	1	0	5	0	1	0	0	0	10	0	0
18-19	5	9	1	6	4	6	1	6	1	4	0	2
20-21	5	85	2	169	7	14	4	27	-	-	0	0
22-23	15	298	12	341	13	78	0	12	-	-	0	0
24-25	11	72	12	165	15	77	2	47	6	18	0	10
26-27	0	8	10	45	1	7	2	8	4	44	0	4
28-29	10	42	62	335	12	40	1	16	1	17	4	3
June Total	49	518	107	1101	55	229	12	130	16	140	4	22
June 30 - July 1	9	46	22	155	2	46	3	20	2	9	0	0
2-3	1	8	11	84	2	24	10	58	23	101	9	32
4-5	6	13	49	416	12	32	1	12	1	6	1	9
6-7	9	11	1	14	6	24	0	3	2	39	0	1
8-9	8	67	107	883	42	241	5	12	14	96	0	2
10-11	116	718	152	2014	35	225	7	224	38	874	1	16
12-13	16	77	39	979	21	107	14	398	49	1004	9	332
14-15	63	327	91	1322	44	141	14	213	97	709	5	172
16-17	42	508	77	1722	37	178	30	168	14	75	3	110
18-19	42	377	-	-	-	-	16	120	72	765	2	236
20-21	1	4	40	1740	-	-	234	2848	109	1793	55	4800
22-23	144	1439	202	1814	51	767	132	3708	496	8599	15	5275
24-25	37	705	6	89	15	434	47	2989	-	-	29	14125
26-27	41	284	23	327	49	1520	32	4037	0	0	13	4100
28-29	33	440	58	596	36	543	46	3962	188	10750	75	12425
30-31	80	618	232	2825	53	1731	36	5257	65	4766	50	8050
July Total	648	5642	1110	14980	405	6013	627	24029	1170	29586	267	49685
Aug. 1-2	95	340	109	1423	116	1511	7	3182	74	7725	225	5225
3-4	141	671	209	3028	63	1168	49	3541	109	7973	100	2175
5-6	144	1403	67	1319	18	209	25	2919	46	320	275	14375
7-8	66	521	169	623	146	1805	16	1237	51	4614	525	2950
9-10	25	86	95	663	46	192	62	192	58	2695	500	1750
11-12	174	1562	206	1402	234	1654	63	1425	-	-	675	1800
13-14	76	308	32	199	104	391	125	552	113	5879	1975	1250
15-16	56	194	74	406	175	866	72	338	18	2486	1025	1425
17-18	106	188	94	290	56	232	139	394	86	1305	-	-
19-20	167	338	49	431	40	84	132	640	44	1868	-	-
21-22	100	267	70	225	171	530	72	237	37	1601	-	-
23-24	248	387	40	209	72	259	35	151	39	1770	-	-
25-26	76	162	18	190	21	67	70	274	91	741	-	-
27-28	84	104	28	123	-	-	91	544	56	978	-	-
29-30	289	994	-	-	-	-	-	-	-	-	-	-
Aug. Total	1847	7525	1260	10531	1262	8968	958	15626	822	39955	5300	30950
Total	2544	13685	2477	26612	1722	15210	1597	39785	2008	69681	5571	80657

1/ Number of spores per slide. All others number of spores per square inch of slide.

2/ Caught May 29-30.

CROWN RUST OF OATS IN CANADA IN 1963

George Fleischmann [1/]

Disease development in western Canada

Crown rust of oats, Puccinia coronata Corda f. sp. avenae Erikss., was first found in western Canada in 1963 at Morden, Manitoba on July 2nd. The disease was present in trace amounts throughout the south-eastern part of Manitoba early in July. Favorable weather conditions permitted rapid rust development, and moderate to severe infection occurred on the lower leaves of oats in all fields examined south of Winnipeg on July 20th. The oat crop north of Winnipeg and as far west of the Red River Valley as Swift Current in Saskatchewan sustained only mild crown rust infection. The disease was general throughout all oat growing regions of the western rust belt by August 10th, but remained light (trace to 10%) in intensity in northern Manitoba and in Saskatchewan. A severe rust epiphytotic occurred in southern Manitoba where the intensity of infection ranged between 30 and 90% by August 20th, Figure 1.

Disease losses in western Canada

Quantitative and qualitative losses in oats due to crown rust were small despite the widespread distribution of the disease. In Saskatchewan and northern Manitoba this is ascribed to the very limited intensity of the disease on the oat crop. In southern Manitoba, however, where the crown rust epiphytotic was severe, most of the crop escaped serious damage because the plants had headed prior to the onset of heavy crown rust infection. This was clearly demonstrated by the severe losses suffered in late-sown fields in which the oats headed after initiation of the epiphytotic. Fortunately, late-sown fields were few in number this year.

Experiments are currently being conducted at the Research Station in Winnipeg to determine the degree of correlation which exists between the occurrence, duration and intensity of rust infections, and the yield and quality of oats. Once the tests have been completed, estimates of crown rust damage in terms of bushels loss per acre will accompany these reports. Suffice it to say this year that with the exception of late-sown fields in southern Manitoba crown rust did not significantly damage the western oat crop.

Disease ratings in crown rust nurseries

Uniform rust nurseries were grown at many localities across Canada. When the plants were approaching maturity a small sheaf was cut from each row in the nursery and sent to Winnipeg where the disease rating was assessed. The rust intensity ratings for each of the 10 oat varieties grown at each of the nurseries are presented in Table 1.

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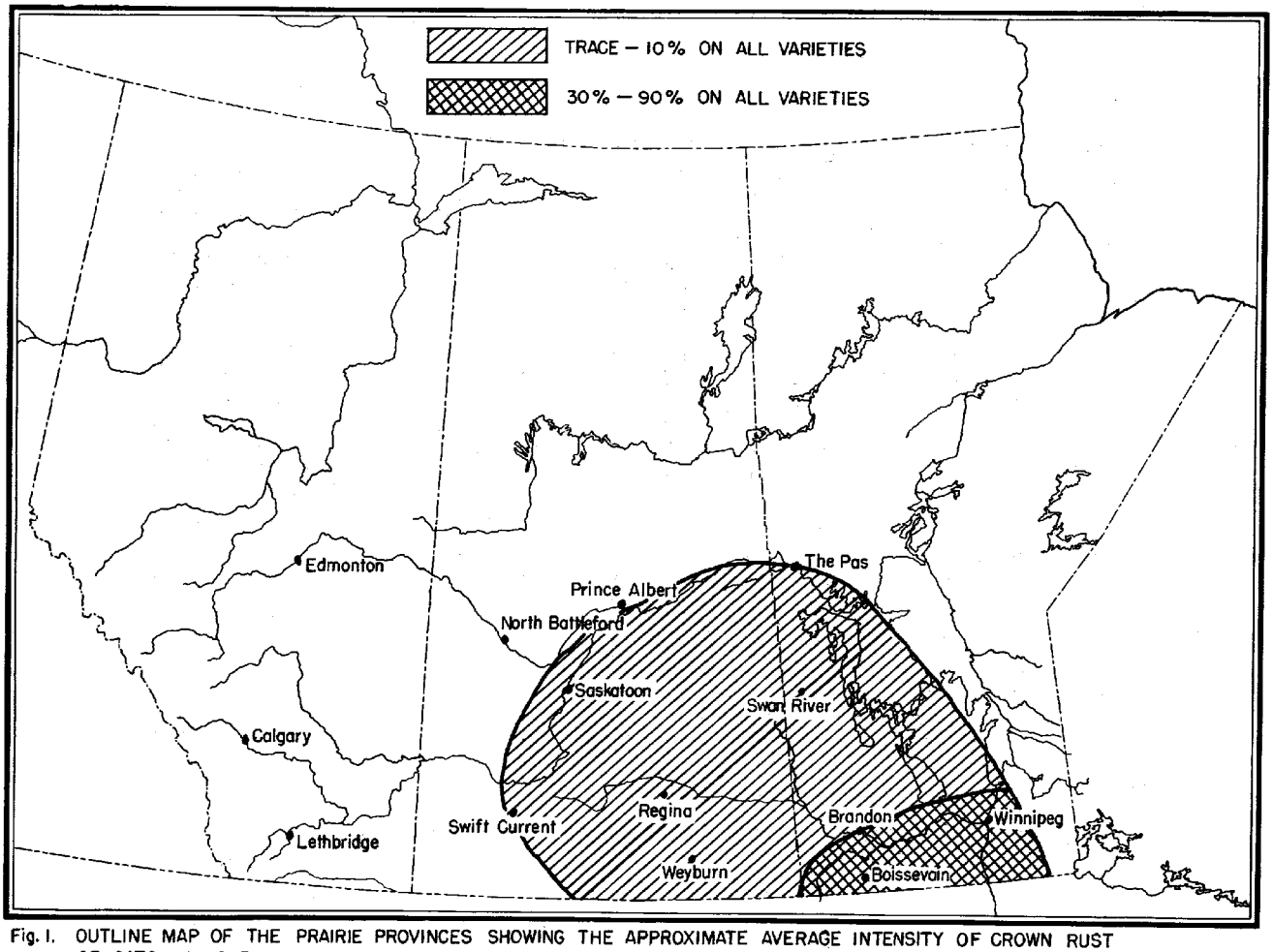

Fig. 1. OUTLINE MAP OF THE PRAIRIE PROVINCES SHOWING THE APPROXIMATE AVERAGE INTENSITY OF CROWN RUST OF OATS IN 1963.

Table 1. Per cent infection of crown rust of oats in 1963 on 10 oat varieties at 15 locations across Canada

Locality	Bond	Trispernia	Exeter	Garry	Clinton	Landhafer	Rodney	C I 4023	Ceirch du Bach	Saia
Melfort, Sask.	70	0	40	20	70	0	5	10	0	0
Indian Head, Sask.	70	tr	40	10	50	5	20	30	0	0
Brandon, Man.	70	tr	80	60	70	20	50	70	tr	tr
Morden, Man.	70	30R	80	80	80	50	80	70	20M	5
Glen Lea, Man.	5	tr	30	20	30	tr	10	5	tr	tr
Winnipeg, Man.	40	tr	40	30	50	2	30	10	tr	tr
Fort William, Ont.	50	0	40	30	20	tr	10	20	0	0
Guelph, Ont.	0	0	0	10	0	0	tr	0	0	0
Ottawa, Ont.	20	0	20	20	20	tr	20	20	0	0
Merrickville, Ont.	80	0	80	80	80	0	50	10	5	0
Alfred, Ont.	40	0	40	20	50	0	80	60	0	0
Appleton, Ont.	40	0	40	10	20	tr	20	10	0	0
Williamstown, Ont.	20	0	60	20	40	tr	40	20	0	0
Quebec City, Que.	80	0	60	50	80	0	40	50	0	0
Fredericton, N.B.	0	0	tr	0	0	0	0	0	0	0

Table 2. Distribution by geographic areas of physiologic races of Puccinia coronata avenae collected on oats in Canada in 1963

Physiologic race	Geographic areas								
	Manitoba	Sask.	Total isolates West	% of total isolates West	Que. & P.E.I.	Ontario	Total isolates East	% of total isolates East	Total isolates E. & W.
201	1	3	4	3.6	-	2	2	2.1	6
202	2	2	4	3.6	-	1	1	1.1	5
203	10	7	17	15.5	2	9	11	11.5	28
209	-	-	-	-	2	1	3	3.1	3
210	1	2	3	2.7	3	12	15	15.7	18
211	5	7	12	10.9	1	9	10	10.5	22
216	6	3	9	8.2	2	4	6	6.3	15
228	-	-	-	-	1	-	1	1.1	1
229	-	-	-	-	-	1	1	1.1	1
230	-	-	-	-	1	-	1	1.1	1
239	-	-	-	-	2	-	2	2.1	2
241	-	1	1	0.9	1	2	3	3.1	4
264	1	1	2	1.8	-	-	-	-	2
274	1	-	1	0.9	-	2	2	2.1	3
276	1	-	1	0.9	-	-	-	-	1
281	1	-	1	0.9	-	5	5	5.2	6
284	-	-	-	-	1	13	14	14.7	14
290	5	2	7	6.4	-	1	1	1.1	8
293	-	3	3	2.7	-	1	1	1.1	4
294	5	8	13	11.8	-	3	3	3.1	16
295	15	5	20	18.2	-	1	1	1.1	21
320	-	-	-	-	2	1	3	3.1	3
324	-	-	-	-	-	2	2	2.1	2
326	7	-	7	6.4	-	-	-	-	7
327	1	-	1	0.9	-	-	-	-	1
332	1	-	1	0.9	-	-	-	-	1
New races	2	1	3	2.7	-	7	7	7.3	10
Totals	65	45	110	100.0	18	77	95	100.0	205

The uniform oat nurseries in southern Manitoba were the most severely infected with crown rust. Oat varieties in eastern Saskatchewan were also rusted, but from western Saskatchewan to the Pacific no infection was detected on any of the 10 oat varieties grown. This nursery data corroborates the information from the field surveys indicating that a widespread crown rust epidemic occurred across the entire rust belt of Manitoba and Saskatchewan in 1963.

Crown rust incidence in eastern Canada, as indicated by its prevalence in uniform oat nurseries, remained much the same as in previous years. Most severe infections again occurred in buckthorn-riddled southeastern Ontario. The buckthorn hedgerows between fields, along roadsides and in wood-lots in the Merrickville, Kemptville, Appleton, Williamstown area provide large quantities of aeciospores, which in years of favorable environmental conditions, initiate early and severe crown rust epidemics in neighbouring oat fields.

Distribution of physiologic races

Thirty-three physiologic races of crown rust were identified from 205 isolates made in Canada this year. The Victoria-virulent races 216 and 274, which have been increasing annually since 1957, were greatly reduced in eastern Canada (28% in 1962 to 8% in 1963) and in the west (41% in 1962 to 9% in 1963). In the east these two races were replaced by races common to the region, namely 203, 210, 211, and 284, but in western Canada a dramatic shift occurred towards races virulent on the Landhafer and Santa Fe sources of crown rust resistance. Last year the Landhafer - Santa Fe virulent races comprised about 10% of the western crown rust population, but this year the same group of races headed by 294 and 295 constituted 52% of the isolates identified in the west. This alarming change has undermined the value of the current crown rust resistance breeding program in western Canada in which the Landhafer and Santa Fe sources are being widely used. Nearly every isolate made this year, regardless of race, was able to attack the commercial oat varieties Rodney and Garry.

A number of new physiologic races of crown rust were discovered during the 1963 survey. These, along with the new races identified last year, will be subjected to further testing prior to their submission for race number assignment. One interesting feature of the new races found in western Canada is that all are virulent on Landhafer and Santa Fe.

Acknowledgements

The author is grateful for assistance given by the co-operators in the care of the rust nurseries and the collection of rust specimens. All those who forwarded crown rust material to Winnipeg, and the many workers in eastern Canada who aided me on my survey through Ontario this summer are also thanked. Mr. W.L. Timlick performed the technical operations requisite to the identification of the physiologic races.

STEM RUST OF OATS IN CANADA IN 1963

G.J. Green [1/]

Prevalence and importance in western Canada

Stem rust of oats (Puccinia graminis Pers. f. sp. avenae Erikss. & Henn.) was first found in western Canada on July 4. It developed slowly, mainly because the varieties Rodney and Garry were resistant to the predominant race, but it was widespread. By early August moderate infections were present on wild oats (Avena fatua L.) throughout Manitoba and infections of 10 to 20 per cent occurred in fields of the variety Rodney. Infections were lighter to the west but traces of rust spread to southwestern and north-central Saskatchewan.

Losses occurred only in south-eastern Manitoba where a few late fields of the variety Rodney were attacked by race 7A.

Incidence in the rust Nurseries

There was little oat stem rust in the nurseries west of Manitoba but severe infections occurred on the susceptible variety Bond in the nurseries from Brandon, Man., eastwards to Fort William, Ont. (Table 1). In these nurseries Garry was nearly free from infection and Rodney had light infections. In eastern Canada the severe infections on Garry and Rodney as well as on Bond in several nurseries were probably caused by race 6A which has predominated in the barberry areas of eastern Canada since 1957. The most resistant variety in the nurseries, C.I. 4023, was lightly infected at Kemptville and Appleton, Ont.

Distribution of physiologic races

Race 6F predominated in western Canada for the third consecutive year. Isolates from susceptible varieties (Table 3) demonstrate its predominance more clearly than the isolates from all sources (Table 2), including resistant varieties. Race 7A, the only other race that occurred commonly in the west, was isolated frequently from the variety Rodney that is susceptible to it but resistant to 6F.

A few cultures of the very dangerous races 6A and 6AF were identified for the first time in western Canada (Table 2). Both races can attack all oat varieties licensed in Canada. For the last 5 years race 6A has severely damaged Rodney and Garry in the barberry areas of eastern Canada. If it becomes prevalent in the prairie provinces it could severely damage all the varieties now grown there. Resistance gene F protects

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Table I. Per cent infection of stem rust of oats (Puccinia graminis avenae) on 10 varieties of oats in 24[1/] uniform rust nurseries in Canada in 1963.

Locality	Bond	Trispermia	Exeter	Garry	Clinton	Landhafer	Rodney	C.I. 4023	Ceirch du Bach	Sala
Agassiz, B.C.	0	t[2/]	0	0	0	0	0	0	0	0
Creston, B.C.	t	1	t	0	0	1	0	0	1	0
Melfort, Sask.	3	0	0	0	0	0	0	0	0	0
Indian Head, Sask.	2	t	2	0	20	t	1	0	t	0
The Pas, Man.	2	t	t	0	t	t	t	0	0	0
Brandon, Man.	60	50	10	t	20	10	1	0	5	0
Morden, Man.	60	-	10	0	50	-	1	t	-	0
Glenlea, Man.	1	t	2	t	10	0	0	t	t	0
Winnipeg, Man.	60	t	40	t	30	5	5	t	20	0
Fort William, Ont.	60	10	20	0	40	30	t	0	30	0
Kapuskasing, Ont.	1	0	t	0	3	t	0	0	t	0
St. Catharines, Ont.	0	0	0	t	1	0	0	0	0	0
Kemptville, Ont.	60	-	70	70	50	t	70	10	20	t
Ottawa, Ont.	30	-	30	20	30	-	30	t	-	0
Verner, Ont.	0	t	t	0	0	t	0	0	t	0
Appleton, Ont.	50	70	50	70	70	60	80	20	30	t
Alfred, Ont.	1	-	1	t	t	0	t	0	0	0
Williamstown, Ont.	5	0	t	1	10	0	5	t	t	0
Macdonald College, Que.	10	5	40	5	2	10	40	t	t	0
Lennoxville, Que.	5	t	t	1	5	0	t	0	-	0
La Pocatiere, Que.	50	t	20	40	50	t	20	t	t	0
Normandin, Que.	t	t	0	0	0	t	0	0	0	0
L'Assomption, Que.	20	30	10	5	5	-	10	t	-	0
Quebec, Que.	70	1	20	20	5	1	50	0	-	-

1/ No rust was observed in nurseries grown at Saanichton, B.C., Beaverlodge, Edmonton, Lethbridge, and Lacombe, Alta., Scott, Sask., Guelph, Ont., Fredericton, N.B., Kentville, Nappan, Brule, and Boulardarie, N.S., and St. John's West and Doyles, Nfld.

2/ t = trace

Table 2. Distribution by provinces of physiologic races of Puccinia graminis f. sp. avenae identified in Canada in 1963.

Race	Virulence Formula Effective/ Ineffective Host Genes	Province					Total Isolates	Per cent of Total Isolates
		Que.	Ont.	Man.	Sask.	B.C.		
1	ABDEF/	-	-	-	-	1	1	.6
2	ABDF/E	-	-	-	-	2	2	1.2
4A	EF/ABD	-	2	-	-	-	2	1.2
6	BF/ADE	-	1	-	-	-	1	.6
6A	F/ABDE	15	28	2	-		45	26.5
6F	B/ADEF	-	12	25	12	-	49	28.8
6AF	/ABDEF	-	2	4	-	-	6	3.5
7A	AF/BDE	2	4	25	7	-	38	22.3
8	BDF/AE	-	-	-	-	1	1	.6
8A	DF/ABE	2	8	-	-	-	10	5.9
10A	DEF/AB	-	1	-	-	-	1	.6
11A	DEF/AB	-	-	1	-	-	1	.6
13A	EF/ABD	5	8	-	-	-	13	7.6
Total Isolates		24	66	55	21	4	170	

Table 3. Distribution by provinces of physiologic races of Puccinia graminis f. sp. avenae collected on susceptible varieties of cultivated oats and wild oats in Canada in 1963.

Race	Province					Total Isolates	Per cent of Total Isolates
	Que.	Ont.	Man.	Sask.	B.C.		
1	-	-	-	-	1	1	1.8
2	-	-	-	-	2	2	3.6
4A	-	1	-	-	-	1	1.8
6A	6	5	-	1	-	12	21.8
6F	-	6	15	6	-	27	49.1
7A	-	1	4	2	-	7	12.7
8A	1	1	-	-	-	2	3.6
13A	3	-	-	-	-	3	5.5
Total Isolates	10	14	19	9	3	55	

against race 6A. Race 6AF is potentially more dangerous than race 6A. It was first identified among isolates from an artificially-inoculated barberry plant in the greenhouse at Winnipeg and then was found, late in 1962, in the field in eastern Canada by F.J. Zillinsky and P. Dyck. It is virulent on varieties

carrying all the resistance genes currently identified, but a search for new sources of resistance has revealed that several varieties, notably Rosen's Mutant and Arkansas 674, are resistant to some cultures.

The race distribution in eastern Canada is unchanged from 1962. Race 6A, and the related races 13A and 4A continue to predominate. Two isolates of race 6AF were identified.

Acknowledgements

The author is grateful for the assistance of many cooperators in the care of the rust nurseries and the collection of rust specimens. Mr. J.H. Campbell, Mr. T. Watson and Mr. S. Nichol assisted in identifying physiologic races and in making disease ratings in the rust nurseries.

STEM RUST OF WHEAT IN CANADA IN 1963

G.J. Green [1/]

Prevalence and importance in western Canada

Wheat stem rust (Puccinia graminis Pers. f. sp. tritici Erikss. & Henn.) was present throughout most of the cultivated areas of Manitoba. Saskatchewan and Alberta, but it did little damage. It was first observed in Manitoba on June 17 and, by early August, susceptible varieties in experimental plots throughout that province were severely infected. The resistant varieties Selkirk and Pembina, which occupy nearly all the commercial wheat acreage in the rust area of Manitoba and southeastern Saskatchewan, were nearly free from infection. The durum wheat variety Ramsey and the predominant barley variety Parkland had only traces of rust. Light infections occurred on Thatcher wheat throughout Saskatchewan. In Alberta, infections on Thatcher were light, but some fields of susceptible varieties, such as Marquis and Red Bobs, were damaged.

Incidence in the rust nurseries

Wheat stem rust was present in nurseries from Lethbridge, Alta., eastwards to Quebec, Que., (Table 1). The most severe infections on susceptible varieties occurred in nurseries from Melfort, Sask., eastwards to Fort William, Ont. The comparatively light infections on the varieties Lee, Mindum and Thatcher indicate that race 15B, which can attack them, was not as common as races such as 56 that cannot attack them. The scarcity of rust on the varieties McMurachy and Selkirk indicate that little, if any, of the rust present could attack varieties carrying the gene Sr6 that protects Selkirk from race 15B. Race 17A, making its first appearance in Canada, probably contributed to the light infections on the durum variety Ramsey at Glenlea and Brandon, Man. Kenya Farmer has been resistant in nurseries across Canada since 1954.

Stem rusts of barley and rye in the rust nurseries

Stem rust infections on the barley varieties in the nurseries generally paralleled those on the wheat varieties. Presumably most of the stem rust on barley was wheat stem rust except at Creston, B.C., and Appleton and Williamstown, Ont., where infections of stem rust on barley were accompanied by heavy stem rust infections on rye and light infections on wheat. At these locations rye stem rust (P. graminis Pers. f. sp. secalis Erikss. & Henn.) probably attacked the barley.

Stem rust of rye occured sporadically in nurseries in all regions except the Maritime Provinces.

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Table 1. Per cent infection of stem rust of wheat (Puccinia graminis f. sp. tritici) on 12 wheat varieties in 24 1/ uniform rust nurseries in Canada in 1963.

Locality	McMurachy	Lee	Kenya Farmer	Red Bobs	Marquis	Mindum	Thatcher	Selkirk	Thatcher^6 x Transfer	Exchange	Frontana	Ramsey
Lethbridge, Alta.	0	t	0	40	40	t	t	0	0	5	0	0
Lacombe, Alta.	0	0	0	1	t	0	0	0	0	0	0	0
Scott, Sask.	0	t	0	40	20	1	t	0	t	1	0	0
Melfort, Sask.	0	t	0	70	70	t	3	0	0	20	0	0
Indian Head, Sask.	0	t	0	80	80	20	3	0	5	70	0	0
The Pas, Man.	0	2	t	50	60	t	t	0	30	30	t	0
Brandon, Man.	--	20	1	80	80	40	5	0	20	90	30	5
Morden, Man.	t	20	t	70	70	5	5	0	20	40	t	0
Glenlea, Man.	0	t	0	70	60	10	t	0	t	20	0	20
Winnipeg, Man.	t	10	t	80	90	10	10	t	10	80	5	t
Fort William, Ont.	0	t	0	70	70	2	3	0	20	30	t	0
Kapuskasing, Ont.	0	t	0	5	5	0	0	0	0	t	0	0
Kemptville, Ont.	0	t	0	10	20	0	0	0	0	t	0	0
Ottawa, Ont.	0	0	0	10	5	0	0	0	0	t	0	0
Verner, Ont.	0	1	0	10	5	t	2	0	t	5	0	t
Appleton, Ont.	0	0	0	20	5	0	0	0	0	0	0	0
Alfred, Ont.	0	0	0	40	10	0	0	0	0	0	0	0
Williamstown, Ont.	0	0	0	1	1	0	0	0	0	0	0	0
Macdonald College, Que.	0	t	0	10	10	0	0	0	0	t	0	0
Lennoxville, Que.	0	0	0	t	t	0	0	0	0	0	0	0
La Pocatiere, Que.	0	0	0	40	10	0	0	0	0	t	0	0
Normandin, Que.	0	0	0	2	1	0	0	0	0	0	0	0
L'Assomption, Que.	0	0	0	t	t	0	0	0	0	0	0	0
Quebec, Que.	0	0	0	3	3	0	t	0	t	0	0	0

1/ No rust was observed in nurseries grown at Saanichton, Agassiz, and Creston, B.C., Beaverlodge and Edmonton, Alta., St. Catharines and Guelph, Ont., Fredericton, N.B., Kentville, Nappan, Brule, and Boulardarie, N.S., and St. John's West and Doyles, Nfld.

Distribution of physiologic races

Race 56 continued to be the most common race of wheat stem rust in Canada (Table 2) but its predominance was threatened, especially in western Canada, for the first time since 1958. Race 15B-1L (Can.) which had been increasing gradually since its discovery in 1956 increased sharply to 41.9 per cent of the isolates from all sources. A few isolates of several new races were obtained. The most important of these were biotypes of race group 11-32; 9 of the 20 isolates of this race group attacked seedlings of the varieties Selkirk and Pembina. The 20 isolates were separated into 7 subraces (Table 4). The most threatening of these seems to be the same as race 11F or 32B of the Cooperative Rust Laboratory, St. Paul, Minnesota. This subrace can attack seedlings of Selkirk, Golden Ball, and the Marquis lines carrying genes Sr6 to Sr11, but its pathogenicity on adult plants of varieties such as Selkirk and Pembina has not yet been determined. Naming the other subraces of the 11-32 group does not seem practicable at this time. Race 17A, which can attack Lee and Golden Ball, has not been found previously in Canada. It can attack the durum variety Ramsey which has been cultivated in the rust area of western Canada for several years.

The rank of the races for prevalence is not changed when only isolates from susceptible hosts are considered (Table 3) but it is evident that race 56 was more predominant than is indicated by the data in Table 2.

The reaction types produced by the races and subraces identified in 1963 on 3 supplementary differential host varieties and backcross lines of Marquis carrying genes Sr6 to Sr11 appear in Table 4. Most of the Marquis lines again served as good differential hosts except for races producing a mesothetic reaction on Marquis. No races avirulent on Marquis were identified in 1963. The Marquis^{10} -Sr11 line produced clearer reactions than Lee in the susceptible class. For example, Lee produces a 2+ or intermediate reaction with race 15B-4 (Can.) but Marquis^{10} -Sr11 produced reaction type 3+. Lee seems to have other genes, lacking in Marquis^{10} -Sr11, that influence reaction to some Canadian races. A virulence formula for each race is given in Table 4.

A high proportion of the stem rust collections from barley and wild barley (Hordeum jubatum L.) included rye stem rust (Table 5). Evidently rye stem rust was common in Ontario, Manitoba and Saskatchewan in 1963.

Table 2. Distribution by provinces of physiologic races of Puccinia graminis f. sp. tritici collected on wheat, barley and grasses in 1963.

Race [1]	Province						Total No. of Isolates	Per cent of Total Isolates
	Que.	Ont.	Man.	Sask.	Alta.	B.C.		
11-32 Group	-	4	9	7	-	-	20	5.5
15B-1 (Can.)	-	-	1	-	-	-	1	.3
15B-1L (Can.) [2]	2	7	59	80	4	-	152	41.9
15B-4 (Can.)	-	-	2	3	-	-	5	1.3
17A	-	1	9	6	1	-	17	4.7
18	-	-	-	-	-	1	1	.3
29-1 (Can.)	-	-	-	1	-	-	1	.3
56	8	36	43	66	13	-	166	45.7
Total No. of Isolates	10	48	123	163	18	1	363	

[1] Races 15B-1L (Can.) and 15-4 (Can.) appear to be equivalent to races 15B-2 and 15B-3, respectively, of the Cooperative Rust Laboratory, St. Paul, Minnesota.

[2] From Que., Ont., Man., Sask., and Alta., 2, 4, 38, 37 and 3 cultures, respectively, attacked Marquis^{6}-Sr 7 and Marquis^{4}-Sr 10. The other cultures of race 15B-1L (Can.) were avirulent on these varieties.

Table 3. Distribution by provinces of physiologic races of Puccinia graminis f. sp. tritici collected on barley, wild barley and susceptible varieties of wheat in 1963.

Race	Province						Total No. of Isolates	Per cent of Total Isolates
	Que.	Ont.	Man.	Sask.	Alta.	B.C.		
11-32	-	4	3	4	-	-	11	4.6
15B-1L (Can.)	2	3	28	39	1		73	30.7
17A	-	-	3	3	-	-	6	2.5
18	-	-	-	-	-	1	1	.4
56	7	36	39	56	9	-	147	61.8
Total No. of Isolates	9	43	73	102	10	1	238	

Table 4. Infection types produced on supplemental host varieties of wheat and backcross lines of Marquis wheat with substituted genes for resistance by races and subraces of stem rust in 1963

Race	Host Variety										Virulence Formula (Effective/ Ineffective Host Genes)
	Lee	Golden Ball	Yuma	Marquis^{6}-Sr6	Marquis^{6}-Sr7	Marquis^{6}-Sr8	Marquis^{6}-Sr9a	Marquis^{6}-Sr9b	Marquis^{4}-Sr10	Marquis^{10}-Sr11	
15B-1 (Can.)	3	2	;	;	23^{CN}	2	4-	3+	3+	3+	6, 7, 8/9a, 9b, 10, 11
15B-1L (Can.)	3+	2	3^{C}	;	23^{CN}	2	1+	2	X	3+	6, 7, 8, 9a, 9b, 10/11
15B-1L (Can.)	3+	2	3^{C}	;	3+	2	2	2	3+	4-	6, 8, 9a, 9b/7, 10, 11
15B-4 (Can.)	2+	4	;	;	23^{CN}	2	3+	3+	3+	3+	6, 7, 8/9a, 9b, 10, 11
17A	3	4	;1	;	23^{CN}	4	2	2	X-	3+	6, 7, 9a, 9b, 10/8, 11
18	1	2	;	;	3+	2	4-	2+	3+	1	6, 8, 9b, 11/7, 9a, 10
29-1 (Can.)	1	4	;	3+	4	3+	2	2	3+	1	9a, 9b, 11/6, 7, 8, 10
56	1	2	;	;	4	2	2	2	4[1]	1	6, 8, 9a, 9b, 11/7, 10
11(5)[2]	1	2	;	3+	23^{CN}	1+	3+	3+	3+	1	7, 8, 11/6, 9a, 9b, 10
11(1)	1	2	;1	;	23^{CN}	3+	3+	3±	;1	1	6, 7, 10, 11/8, 9a, 9b
11(1)	1	3	;	;	23^{CN}	2	3	3	3	1	6, 7, 8, 11/9a, 9b, 10
11(1)	1	3+	;1	3+	3±	2	3+	3+	3+	1	8, 11/6, 7, 9a, 9b, 10
11-32(5)[3]	3	2	;1	;	X	X	X	X	;1	X	
11F-32B(3)[5]	3+	3+	;	3+	3+	3+	3+?[4]	3+	4	3+	/6, 7, 8, 9a?, 9b, 10, 11
11-32(4)[3]	12	2	;1	;	23^{CN}	23^{CN}	23^{CN}	X	X-	12	

1/ In Report No. 18 the reaction of Marquis-Sr10 was shown as type 2 in error.
2/ Number of isolates in brackets.
3/ Produced a mesothetic reaction on Marquis. The effect of the resistance genes in the Marquis backcross lines was difficult to assess.
4/ Reaction unstable; sometimes type 2.
5/ Seems equivalent to race 11F or 32B of the Cooperative Rust Laboratory, St. Paul, Minnesota.

Table 5. Number of collections of stem rust on barley and wild barley, and the number of isolates of Puccinia graminis f. sp. secalis obtained from them in 1963.

Province	Number of Collections	Isolates of P. gr. secalis
Ontario	11	8
Manitoba	36	6
Saskatchewan	50	10

Acknowledgments

The author is grateful for the assistance of many cooperators in the care of the rust nurseries and the collection of rust specimens. Mr. J.H. Campbell, Mr. T. Watson and Mr. S. Nichol assisted in identifying physiologic races and in making disease ratings in the rust nurseries.

LEAF RUST OF WHEAT IN CANADA IN 1963

D.J. Samborski [1/]

Disease development in western Canada

Leaf rust of wheat (Puccinia recondita Rob. ex. Desm.) was first found at Winnipeg, Manitoba, on June 11, and on June 12, a trace of leaf rust was found in early fields of Selkirk wheat in the Red River Valley. This was an earlier than usual occurrence of leaf rust. However, the moderate resistance of Selkirk and Pembina delayed rust development, and severe infections did not develop until after heading. This is usually too late to cause large losses but this year hot dry weather in Manitoba during July and the leaf rust attack caused defoliation before the kernels were fully formed. Continued hot dry weather matured the crop rapidly leaving little time for the grain to fill. These conditions caused a greater reduction in yields and grades than could have been anticipated from leaf rust alone.

By early August, leaf rust infections were severe in much of Saskatchewan. However, leaf rust developed later than in Manitoba and early-seeded wheat probably did not suffer any appreciable damage. Late fields undoubtedly were damaged to some extent but favourable temperature and moisture conditions prevented any serious yield loss even in late fields.

Leaf rust in the rust Nurseries

Severe infections of leaf rust occurred at nurseries throughout the prairie provinces (Table 1). Selkirk, Lee and Kenya Farmer were severely attacked, but on these varieties rust development is delayed, usually until after heading. Thatcher^6 x Transfer, which contains the gene from Aegilops umbellulata for leaf rust resistance, was highly resistant at all locations in Canada.

Exchange and Frontana were resistant to leaf rust at all nurseries. These varieties have adult plant resistance to leaf rust and are being used in current breeding programs, both in Canada and the United States.

Distribution of physiologic races

Nine races of wheat leaf rust were isolated in the 1963 race survey (Table 2). Race 15 was the most prevalent race in Canada and was markedly predominant in the prairie provinces. This situation has existed for a number of years. However, in recent years, evolutionary changes leading to increased virulence on commercial varieties have occurred within the race 15 population. The latest shift in virulence is occurring with respect to the commercial wheat varieties Selkirk and Pembina.

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Table 1. Per cent infection of wheat leaf rust (Puccinia recondita) in 1963 on 12 wheat varieties in uniform rust nurseries at 34 1/ locations in Canada

Locality	McMurachy	Lee	Kenya Farmer	Red Bobs	Marquis	Mindum	Thatcher	Selkirk	That^{6} x Trans.	Exchange	Frontana	Ramsey
Saanichton, B.C.	70	15	25	70	60	0	70	0	0	t	0	t
Agassiz, B.C.	t	0	0	t	t	0	t	0	0	0	0	0
Creston, B.C.	100	20	20	100	100	20	100	50	0	0	0	0
Lethbridge, Alta.	85	65	60	90	90	t	90	65	t	0	t	5
Lacombe, Alta.	65	50	50	70	70	2	70	30	0	1	t	2
Scott, Sask.	80	40	40	90	80	t	90	30	0	t	0	3
Melfort, Sask.	100	80	50	100	100	t	100	80	0	3	t	t
Indian Head, Sask.	100	70	70	100	100	0	100	70	0	t	0	0
The Pas, Man.	90	75	75	95	90	15	95	70	0	t	5	5
Brandon, Man.	--	80	80	90	80	t	90	70	0	2	t	5
Morden, Man.	80	80	70	80	70	5	80	70	0	t	t	t
Glenlea, Man.	80	60	70	90	80	t	100	50	0	2	0	t
Winnipeg, Man.	90	80	60	100	90	t	100	70	0	0	0	0
Fort William, Ont.	90	60	50	80	80	3	90	60	0	1	0	0
Kapuskasing, Ont.	15	1	2	20	15	t	15	5	0	0	0	t
St. Catherines, Ont.	t	t	0	0	0	0	t	0	0	0	0	0
Guelph, Ont.	85	20	20	85	70	15	85	45	0	0	0	0
Kemptville, Ont.	85	60	40	80	75	t	80	15	0	5	t	t
Ottawa, Ont.	70	5	3	70	60	1	70	3	0	0	0	0
Verner, Ont.	60	10	10	60	60	--	60	5	0	0	--	0
Appleton, Ont.	20	3	2	20	20	1	20	2	0	0	0	0
Alfred, Ont.	60	1	1	60	60	t	60	1	0	0	0	0
Williamstown, Ont.	80	40	30	90	80	10	80	30	0	20	2	t
Macdonald College, Que.	20	1	1	20	20	0	20	t	0	0	0	0
Lennoxville, Que.	15	5	8	20	20	2	25	t	0	0	0	2
La Pocatiere, Que.	15	1	10	15	10	--	--	--	--	--	--	--
Normandin, Que.	90	35	15	90	90	10	90	30	0	0	0	0
L'Assomption, Que.	20	1	1	20	15	0	25	t	0	t	0	0
Quebec, Que.	60	5	10	75	75	5	70	10	0	0	0	1
Fredericton, N.B.	t	0	0	t	t	0	0	0	0	0	0	0
Kentville, N.S.	15	t	2	15	15	t	15	0	0	0	0	0
Nappan, N.S.	t	t	t	t	t	0	t	0	0	0	0	0
Brulé, N.S.	t	0	0	t	t	0	t	0	0	0	0	0
St. John's West, Nfld.	10	t	t	10	10	0	10	0	0	0	0	0

1/ No wheat leaf rust was observed in nurseries from Edmonton and Beaverlodge, Alta., Boulardarie, N.S.

Table 2. Distribution by provinces of physiologic races of Puccinia recondita isolated in Canada in 1963.

Race		Province						Total Isolates	Per Cent of Total Isolates
UN		Que.	Ont.	Man.	Sask.	Alta.	B.C.		
1	1	--	--	--	2	--	--	2	0.8
2	15	6	20	82	50	13	--	171	66.8
3	58	5	34	--	--	--	--	39	15.2
3	161	--	--	--	1	--	10	11	4.3
5	5	--	2	5	9	2	--	18	7.0
6	105	--	1	--	--	--	--	1	0.4
9	9	--	--	1	1	--	--	2	0.8
10	11	--	7	--	1	--	--	8	3.1
13	35	--	4	--	--	--	--	4	1.6
Total No. of Isolates		11	68	88	64	15	10	256	100.0

Table 3. Frequency distribution of the 1963 leaf rust isolates from the prairies in pathogenicity classes designated by infection types produced on Selkirk wheat.

Province	;1^{-}	1-2	2^{+}	2^{++}	3	3^{+}4	Total
Man.	2	46	12	10	6	12	88
Sask.	0	39	11	6	5	3	64
Alta.	0	13	0	0	2	0	15
Total	2	98	23	16	13	15	

Selkirk and Pembina have the same seedling genes for leaf rust resistance; of these, the L gene conditioning a fleck and type 1 reaction, and the E gene which conditions a type 1 to 2 reaction, are the most important. It can be seen from Table 3 that nearly all isolates from the Prairies can attack the L gene. The interesting feature shown in Table 3 is the lack of a clear distinction between virulence and avirulence on the E gene. The reaction type 1 to 2 is typical for cultures with avirulence to this gene and type 3 to 4 would be expected from virulent cultures. It is possible that reaction types 2+, 2++ and 3 are conditioned by cultures which are heterozygous for virulence, with virulence being incompletely dominant. The influence of modifying genes can lead to a range of reaction types with different heterozygous cultures.

The NA 61 races isolated in Canada in 1963 are shown in Table 4. Most of the isolates were identified as NA 61-14 which attacks three of the four supplementary differentials. It is obvious that this group of supplementary differentials is no longer adequate and should be changed in the near future.

Table 4. Distribution by geographic areas of NA 61 races of *Puccinia recondita* isolated in Canada in 1963.

Geographic Area	Number of isolates of indicated NA 61 races						
	1	3	4	5	7	12	14
B.C.		8					2
Alta.							15
Sask.		2		1		6	55
Man.		2				2	84
Ont.	7	5	4		11		41
Que.					1		10
Total Isolates	7	17	4	1	12	8	207

Bulked collections of leaf rust uredospores from each area were used to inoculate a group of highly resistant wheat varieties which included Exchange, Agrus, Transfer, Klein Lucero, Aniversario, Africa 43, Klein Titan and Maria Escobar. A small sporulating pustule was observed on Transfer after inoculation with a bulked collection from Saskatchewan. Spores were transferred to Little Club wheat and the culture was tested again on Transfer, producing a 1$\frac{+}{4}$ reaction. There is only one other recorded instance of a culture being isolated capable of even limited sporulation on Transfer. This culture was isolated at Winnipeg in 1960

(reaction type $1^{\pm}$ on Transfer) and was found to be heterozygous for virulence on the Transfer gene for resistance. It is reasonably certain that the culture obtained in 1963 is also heterozygous for virulence at this locus.

Acknowledgements

The author is grateful for the help of cooperators who sent rust collections to Winnipeg for identification and to Mr. W. Ostapyck for his technical help in the identification of races.

CO-OPERATIVE SEED TREATMENT TRIALS -- 1963 [1]

J.E. Machacek and H.A.H. Wallace [2]

Twenty-nine seed treatment materials were tested in 1963 against common bunt of wheat (mixed Tilletia foetida (Wallr.) Liro and T. caries (D.C.) Tul.), oat smut (mixed Ustilago avenae (Pers.) Rostr. and U. kolleri Wille), covered smut of barley (U. hordei (Pers.) Lagerh.), and against seed rot of flax caused by a complex of soil-borne and seed-borne micro-organisms.

Materials and Methods

Kinds of seed used in trials

Wheat bunt trials - Variety Red Bobs. Seed artificially contaminated (1:200, by weight) with mixed spores of Tilletia tritici and T. foetida.

Oat smut trials - Variety Vanguard. Seed naturally contaminated by loose and covered smut.

Barley smut trials - Variety Plush. Seed naturally contaminated by covered smut.

Flax seed-rot trials - Variety Redwood. About 50% of seeds cracked during threshing.

Fungicides

The 29 seed-treatment materials received for testing and brief statements on their nature and source are listed below with designating numbers 2 to 30 inclusive. Those numbers 2 to 12 are dusts, and the remainder are liquids. The majority of the materials contain organic mercury compounds.

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[1] Contribution No. 150 from the Canada Department of Agriculture Research Station, Winnipeg, Manitoba.

[2] Principal Plant Pathologist and Associate Plant Pathologist, respectively, Plant Pathology Laboratory.

Treatment No.	Description of Products
1	Check - Seed not treated.
2	A powder containing 3.2% mercury as ethylmercuric p-toluene sulfonanilide. Obtained from E.I. du Pont de Nemours, Wilmington, Delaware.
3	A powder containing 0.8% mercury as ethylmercuric p-toluene sulfonanilide. Obtained from E.I. du Pont de Nemours, Wilmington, Delaware.
4	A mercurial powder of undisclosed composition. Obtained from E.I. du Pont de Nemours, Wilmington, Delaware.
5	A powder containing 12.5% "Diazinon" and 37.5% captan. Obtained from Chipman Chemicals, Winnipeg, Manitoba.
6	A powder containing 50.0% tetrachloronitroanisole. Obtained from Pittsburgh Plate Glass Company, Mooretown, New Jersey.
7	A powder containing 5.0% mercury as mixed phenylmercuric acetate and ethylmercuric chloride. Obtained from Allied Chemical Services, Calgary, Alberta.
8	A powder containing 60.0% captan and 15.0% dieldrin. Obtained from Stauffer Chemical of Canada, Vancouver, B.C.
9	A powder containing 40.0% dieldrin and 2.0% mercury as mixed phenylmercuric acetate and ethylmercuric chloride. Obtained from Allied Chemical Services, Calgary, Alberta.
10	A powder containing 70.0% p-dimethylaminobenzene-diazo sodium sulfonate. Obtained from Chemagro Corporation, Kansas City, Missouri.
11	A powder containing 35.0% p-dimethylaminobenzene-diazo sodium sulfonate and 35.0% trichlorodinitro-benzene. Obtained from Chemagro Corporation, Kansas City, Missouri.
12	A powder containing 70.0% trichlorodinitrobenzene. Obtained from Chemagro Corporation, Kansas City, Missouri.

Treatment No.	Description of Products
13	A liquid containing 25% heptachlor and 0.37% mercury as methylmercuricdicyandiamide. Obtained from Morton Chemical Corporation Co., Woodstock, Illinois.
14	A liquid containing 5.0% mercury as ethylmercuric hydroxide. Obtained from Green Cross Products, Winnipeg, Manitoba.
15	A liquid containing 4.2% mercury as methylmercuric 8-hydroxyquinolinate. Obtained from Seventy Seven Oil Company, Lethbridge, Alberta.
16	A liquid containing 1.5% mercury as methylmercuric nitrile. Obtained from Morton Chemical Company, Woodstock, Illinois.
17	A liquid containing 40.0% aldrin and 0.44% mercury as methylmercuric 8-hydroxyquinolinate. Obtained from Shell Oil Company, Toronto, Ontario.
18	A liquid containing 30.0% aldrin. Obtained from Morton Chemical Company, Woodstock, Illinois.
19	A liquid containing 7.8 oz./gal. of mercury as methylmercuric dicyandiamide. Obtained from Seventy Seven Oil Company, Lethbridge, Alberta.
20	A liquid containing 1.5% mercury as methylmercuric benzoate. Obtained from Morton Chemical Company, Woodstock, Illinois.
21	A liquid containing 1.25% mercury as methylmercuric 8-hydroxyquinolinate. Obtained from Morton Chemical Company, Woodstock, Illinois.
22	A liquid containing 1.5% mercury as methylmercuric nitrile. Obtained from Chipman Chemical Company, Winnipeg, Manitoba.
23	A liquid containing 1.5% mercury as methylmercuric dicyandiamide. Obtained from Chipman Chemical Company, Winnipeg, Manitoba.
24	A liquid containing 2.5 oz./gal. of mercury as methylercuric dicyandiamide. Obtained from Morton Chemical Company, Woodstock, Illinois.

Treatment No.	Descriptions of Products
25	A liquid containing a mixture of heptachlor and pentachloronitrobenzene. Obtained from Morton Chemical Company, Woodstock, Illinois.
26	A liquid containing 1.5% mercury as methylmercuric benzoate. Obtained from N.V. Aagrunol Chemical Works, Holland.
27	A liquid containing 1.75% mercury as methylmercuric benzoate. Obtained from N.V. Aagrunol Chemical Works, Holland.
28	A liquid containing 2.1% mercury as mixed ethylmercuric 2,3-dihydroxypropyl mercaptide and ethylmercuric acetate. Obtained from E.I. du Pont de Nemours, Wilmington, Delaware.
29	A liquid containing 30.8% technical aldrin and 8.43% mercury as phenylmercuric acetate. Obtained from Gallowhur Chemicals Canada Ltd., Montreal, Quebec.
30	A liquid containing 2.25% mercury as mixed methylmercuric 2,3-dihydroxypropyl mercaptide and methylmercuric acetate. Obtained from E.I. du Pont de Nemours, Wilmington, Delaware.

Experimental Results

The field data collected in 1963 are summarized in Table 1. One product, Drinox, was unsatisfactory for the control of all smut diseases tested, and two products, Diazinon-Captan Dual Purpose Seed Dressing and Pentadrin, gave only moderate control. In general, TCNA (50% powder), Captan-Dieldrin 60-15 and Gallodual were not quite as effective as some of the other seed dressings for controlling smut diseases. Although Chemagro 2635 70% gave inadequate control of bunt and depressed flax germination, and Dexon 70% WP inadequate control of oat and barley smuts, the combined product Dexon 35% + Chemagro 2635 35% gave satisfactory results for all crops. Flax germination was considered satisfactory when it exceeded 70% and moderately satisfactory within the range 65 to 70%. Four products, TCNA, Chemagro 2635 70%, Drinox and Pentadrin decreased the germination.

Table 1. Co-operative Seed Treatment Trials - 1963 (Summary of Data from 6 Stations for Wheat, 11 Stations for Oats, 13 Stations for Barley, 9 Stations for Flax).

Treatment No.	Abbreviated Name	Dose (oz./bu.)				Smut (%)			Germination %
		Wheat	Oats	Barley	Flax	Wheat	Oats	Barley	Flax
1	Check (dry, untreated seed)	0.00	0.00	0.00	0.00	18.7	8.0	23.4	60.3
2	Ceresan M	0.50	0.50	0.50	1.50	0.0	0.0	0.5	69.3
3	Ceresan M-DB	2.00	2.00	2.00	4.00	0.0	0.0	0.6	65.7
4	DuPont 1966	0.50	0.50	0.50	1.50	0.0	0.0	0.2	72.2
5	Diazinon-Captan	(4 oz./100 lb. seed)				3.6	2.0	2.9	68.4
6	TCNA (50% powder)	1.50	1.50	1.50	1.50	0.2	1.9	3.7	55.2
7	ACS Mercury ST.	0.50	0.50	0.50	1.50	0.0	0.0	0.1	66.9
8	Captan-Dieldrin 60-15	1.00	1.00	1.00	1.00	1.1	1.2	0.8	68.9
9	ACS AM Dual Purpose**	2.00	1.40	1.40	5.00	0.2	0.1	0.4	64.0
10	Dexon 70% WP	1.00	1.00	1.00	1.00	0.0	2.3	15.7	73.4
11	Dexon 35% + Chemagro 2635 35%	1.00	1.00	1.00	1.00	0.0	0.0	0.3	72.1
12	Chemagro 2635 70%	1.00	1.00	1.00	1.00	4.1	0.0	0.2	55.3
13	Pandrinox	2.12	2.12	2.12	4.00	0.0	0.0	0.1	67.0
14	Sandoz Tillex	0.75	0.75	0.75	1.50	0.0	0.0	0.1	64.9
15	Seventy Seven (P.C.P. 8239)*	0.75	0.75	0.75	1.50	0.0	0.0	0.0	75.0
16	EP-208 Liquid	0.75	0.75	0.75	1.50	0.0	0.0	0.2	73.7
17	Shell AM Dual Purpose**	2.00	1.40	1.40	5.00	0.0	0.1	7.4	64.1
18	Drinox	4.00	4.00	4.00	4.00	22.7	7.7	24.0	56.4
19	Seventy Seven (P.C.P. 8542)*	0.75	0.75	0.75	1.50	0.0	0.0	0.3	72.8
20	EP-209 Liquid	0.75	0.75	0.75	1.50	0.2	0.0	0.0	69.8
21	EP-202 Liquid	0.75	0.75	0.75	1.50	0.0	0.0	0.6	70.7
22	Agrosol (1962)	0.75	0.75	0.75	1.50	0.0	0.0	0.2	70.0
23	Agrosol (SB)	0.75	0.75	0.75	1.50	0.0	0.0	0.3	73.8
24	Panogen 15B	0.75	0.75	0.75	1.50	0.0	0.0	0.2	70.5
25	Pentadrin	3.00	3.00	3.00	7.50	1.7	1.6	13.9	56.1
26	Aabiton LSV-150	0.75	0.75	0.75	1.50	0.0	0.0	2.3	69.5
27	Aabiton LS-175	0.75	0.75	0.75	1.00	0.0	0.0	0.1	72.5
28	Ceresan 75	0.75	0.75	0.75	1.50	0.0	0.0	0.4	69.7
29	Gallodual	2.44	1.90	1.90	6.66	0.1	1.7	0.9	65.2
30	Ceresan L	0.75	0.75	0.75	1.50	0.0	0.0	0.1	74.2
	Least Sign. Difference					6.3	1.0	3.3	5.4

* Dilute according to Manufacturer's directions before use.
** Dosage is based on seeding rate.

Acknowledgements

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CANADA AGRICULTURE
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WHEAT STRIATE MOSAIC, A SWORD OF DAMOCLES HANGING OVER THE WESTERN WHEAT GROWER-OR NOT?

W.A.F. Hagborg [1/]

In a paper entitled "Wheat striate mosaic, a virus disease to watch on the prairies" Slykhuis (1) expressed concern regarding the danger of damage to the western wheat crop from a severe attack of wheat striate mosaic. In early July, 1961, he found striate mosaic at a low rate of infection (less than 1%) in "almost all wheat fields examined on a route from Carlyle, Sask., through Brandon to Winnipeg, Man". In greenhouse transmission tests with the painted leafhopper, Endria inimica (Say), he established that Selkirk red spring wheat and many durum wheat varieties were very susceptible to this disease.

As a result of his findings, Slykhuis inferred that the disease might have been severe in previous years and escaped observation. He said, "It cannot be stated categorically that wheat striate mosaic has or has not caused serious losses in Canadian crops ...".

The position he has taken seems untenable. Striate mosaic has been observed in durum wheat plots and fields in Manitoba for several years but always in low intensities. Even when only a trace of the plants are infected its characteristics are sufficiently distinctive to make it noticeable. It seems improbable that a severe infection could have escaped the observations of plant pathologists in their annual plant disease surveys.

In 1962, as the result of a survey of Manitoba and Saskatchewan, Slykhuis (2) stated, "There was no evidence of wheat striate mosaic in any of the areas of southeastern Saskatchewan and southern Manitoba where it occurred in trace amounts in nearly all fields examined in 1961".

Sylkhuis (3) exhibited continuing apprehension about the potential destructiveness of wheat striate mosaic on the prairies in a paper given at the Annual Meeting of the Canadian Phytopathological Society in June, 1963. He discussed a number of factors that affect the development of the disease, but did not mention what seems to be a very significant factor in relation to the epidemiology of the disease, namely, the rate of reproduction of the vector. Preliminary results of a life history study (4) indicate a relatively long period from egg to adult and a low reproductive potential. It has yet to be shown that the population dynamics of Endria inimica can meet the heavy demands required for wheat striate mosaic to become a destructive disease in the short growing period characteristic of the prairies of Western Canada.

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Whether or not wheat striate mosaic constitutes a potential threat, selection for resistance to it may be prudent in the cereal breeding program. At present there appears to be insufficient grounds for regarding resistance to striate mosaic as essential in a new variety although, admittedly, it would be a desirable characteristic to incorporate.

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OVERWINTERING OF RYE STEM RUST IN MANITOBA

G.J. Green [1/]

In the spring of 1951, Johnson and Green (1) observed the overwintering of rye stem rust (Puccinia graminis Pers. f. sp. secalis Erikss. & Henn.) at Winnipeg, Manitoba, (about 50° N.) on Agropyron repens (L.) Beauv. and its subsequent spread to new growth. No other report was found of the reestablishment of overwintered stem rust so far north. On August 15, 1963, circumstantial evidence for the overwintering of rye stem rust on A. repens and its reestablishment was found at two locations near Minitonas, Manitoba, over 300 miles northwest of Winnipeg, and a short distance north of the 52nd parallel.

In the course of a rust survey, an area about 20 feet square in a corner of a barley field was found to carry an 80 per cent infection of stem rust. The remainder of the field had only traces of rust. A patch of A. repens growing in the headland at this corner of the field was severely attacked by stem rust. In a second field of mixed barley and oats the barley plants in a localized area along one side of the field was severely infected, as was a heavy stand of A. repens in the adjacent headland. The remainder of the field was nearly free from rust.

These circumstances indicated that the rust had spread from A. repens to the barley. The severity of the infections on A. repens indicated that rust development had commenced early in the season. It was unlikely that the infections were initiated, as is usual, by air-borne inoculum from the south because other infection centers were not found in the barley. Infection tests in the greenhouse showed that rust collected on A. repens at both locations and on barley at one location attacked rye but not wheat. The evidence indicates that rye stem rust overwintered on A. repens at these two locations north of the 52nd parallel.

Literature Cited

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

SCREENING OF POTATO FUNGICIDES IN 1963[1]

L.C. Callbeck[2]

The relative efficiencies in the control of potato late blight, Phytophthora infestans (Mont.) de Bary, of the several fungicides listed below were studied in a Screening Test at Charlottetown in 1963.

1. Bayer 47531 50W - N -(dichlorofluoromethylthio)-N, N-dimethyl-N-phenylsulphamide. 1.3 pounds/80 Imperial gallons. Chemagro Corporation, Latham, New York, U.S.A.

2. Bordeaux mixture - 8-4-80 formula. Included annually as a standard fungicide.

3. Carbane - Polyethylene bis-thiuram sulphide (12%) + copper oxy-chloride (36%). 2.5 lb./80 gal. Procida, Neuilly sur Seine, France.

4. Dithane M-22 - Manganese ethylene bisdithiocarbamate. (maneb). 1.0 lb./80 gal. Rohm and Haas Company of Canada Limited, West Hill, Ontario.

5. Dithane M-45 - Zinc ion and maneb. Mn, 16%; Zn, 2%. 1.0 lb./80 gal. Rohm and Haas Company of Canada Limited.

6. Difolatan 80W - N-(1, 1, 2, 2, -tetrachloroethylsulphenyl)-cis-Δ^4-cyclohexene-1, 2-dicarboximide. 1.0 lb./80 gal. Ortho Agricultural Chemicals Limited, Oakville, Ontario.

7. DuTer - Triphenyl tin hydroxide (20%). 0.75 lb./80 gal. Philips-Duphar, Amsterdam, Holland.

8. F-100 - Confidential product. 1.0 lb./80 gal. Green Cross Products, Montreal.

9. Hortocritt - Ethylene thiuram monosulphide. 2.5 lb./80 gal. S.I.A.P.A., Rome, Italy.

10. Miller 658 - Copper-zinc-chromate. 2.0 lb./80 gal. Miller Chemical and Fertilizer Corporation, Baltimore, U.S.A.

11. Polyram 80W - Zinc activated polyethylene thirame disulphide. 1.5 lb./80 gal. Two forms of this product were used: (a) the current form on the market and (b) a milled form. Both forms were submitted by Niagara Brand Chemicals, Burlington, Ontario.

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2/ Plant Pathologist.

12. TC-90 - 48% copper salts of fatty and rosin acids. 1.7 qt./80 gal. Tennessee Corporation, Atlanta, Georgia, U.S.A.

13. Trimanoc - Confidential product. 1.25 lb./80 gal. Fabrick van Chemische Producten, Holland, and supplied by Green Cross Products of Montreal.

The plots were planted on June 4, exactly 45 seed pieces of the Green Mountain variety being dropped in each 45-foot row. Each plot was 4 rows wide x 45 feet long and 15 plots, or one for each treatment, were set out in each of 4 ranges. Single rows of potatoes were planted as borders and buffers. These rows were not sprayed, their purpose being to equalize the epidemic over the area. They were inoculated with the late blight fungus by sprinkling them with a water suspension of spores on the evening of August 8. Lesions were observed in all border and buffer rows on August 13.

The fungicides were applied on July 19, 30, August 8, 20, 28, September 2, 12; the mean interval being 9.2 days. The machine employed was a tractor-sprayer unit which delivered approximately 120 gallons per acre at a pressure of 375 pounds per square inch. The boom carried 4 nozzles per potato row; 2 being directly over the plants and 2 being on drop pipes. Insects were controlled by spraying all rows with endosulfan, three applications being made during the season.

The weather in 1963 was favorable for the development and spread of late blight. Rain was recorded in each week of the July-September period, the total precipitation being 14.56 inches or approximately 4.0 inches above normal. There were also several periods of extremely high relative humidity which favored sporulation of the fungus. Three of these periods of abundant sporulation were accompanied by heavy rains which, in turn, caused heavy losses by washing spores into the soil where they established infection on many tubers. In the period of very high humidity and rapid sporulation of August 20-25, rain fell every day, the total for the six days being 2.24 inches. Of this amount, 1.18 inches fell on August 24. In a similar period, August 29-31, 1.44 inches of rain were recorded. A third period of very active sporulation occurred in the first week of September and inoculation of additional tubers was assured by a heavy rain of 1.89 inches on September 6.

Because of the weather conditions described above, losses caused by the rotting of tubers were very high. Losses in the treated plots ranged from 3.9 to 29.0 per cent. In the unsprayed check plots 34.6 per cent of the crop was spoiled by rot.

Under the influence of the high relative humidity and frequent rains, the disease spread out rapidly from the inoculated buffer rows. The unsprayed check plots were 50 per cent defoliated by September 3 and from a trace to 10 per cent defoliation had occurred in the treated plots. Beginning on that date defoliation readings were taken at regular intervals, and mean defoliations, expressed as percentages, are given for selected dates in Table 1.

Table 1. Percentage of defoliation

Treatment	Sept. 6	Sept. 12	Sept. 16
Dithane M-45	3	10	15
Polyram (milled)	3	11	15
Hortocritt	3	14	18
Bordeaux	5	14	22
Bayer	7	16	22
Difolatan	3	14	22
F-100	4	16	22
Dithane M-22	5	18	24
DuTer	4	17	25
Trimanoc	7	20	27
Polyram (current)	8	23	30
Carbane	5	25	35
Miller 658	17	70	85
TC-90	23	72	87
Check	70	100	100

Table 2. Effect of treatments on yield* and rot

Treatment	Total bu/ac	Smalls bu/ac	Rot bu/ac	No. 1 bu/ac	Tuber % Rot
Difolatan	460.3	55.2	18.0	387.1	3.9
DuTer	474.5	52.8	36.0	385.7	7.6
Bayer	466.8	48.2	39.6	378.0	8.5
Dithane M-45	475.7	49.7	55.9	369.6	10.8
F-100	484.1	55.2	64.8	364.1	13.4
Dithane M-22	449.5	47.3	48.7	353.5	10.8
Bordeaux	437.5	51.6	34.8	351.1	8.0
Trimanoc	444.5	50.4	46.8	347.3	10.5
Polyram (milled)	462.5	45.1	78.5	338.9	17.0
Carbane	433.2	51.6	54.5	327.1	12.6
Hortocritt	418.8	51.1	49.7	318.0	11.9
Polyram (current)	417.1	49.2	72.7	295.2	17.4
Miller 658	400.8	37.7	91.9	271.2	22.9
TC-90	393.6	49.2	114.0	230.4	29.0
Check	334.8	56.9	115.9	162.0	34.6
S.D. 5%	42.6			66.4	10.5
S.D. 1%	57.0			88.9	14.0

*Arranged in descending order of No. 1 tuber yields.

The test was terminated on September 18, 106 days after planting and 6 days after the last fungicide treatment, by spraying the plants with Reglone. The tubers were dug, graded, examined for blight rot, and weighed on October 3. The data are given in Table 2.

EXPERIMENTAL FARM,
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STRAIGHT VS. SPLIT POTATO BLIGHT SPRAY SCHEDULES[1]

A Progress Report

L.C. Callbeck[2]

In regions where potato late blight, Phytophthora infestans (Mont.) de Bary, is often a serious threat to the health of the crop near the end of the growing season, many growers employ a split schedule of spraying. Under such a program a carbamate fungicide is used for the early-and mid-season sprays and a copper fungicide is used for the late-season sprays. By and large, the growers who follow this practice are of the opinion that the change to a copper fungicide will provide a greater measure of protection against losses from late blight tuber rot. This somewhat popular grower theory is being tested experimentally at Charlottetown and this paper is a report on the progress that has been made up to and including 1963.

The first field test was conducted in 1953. In that year four treatments were included as follows: (1) Bordeaux mixture all season, (2) nabam + zinc sulphate all season, (3) a split schedule of the nabam spray followed by Bordeaux mixture for the late-season sprays, (4) unsprayed check.

The experiment was laid out in a Latin square, the plots of which were each 4 rows wide by 50 feet long. Separating the plots from one another, and bordering the area, were single rows of potatoes. These rows were not sprayed, their purpose being to equalize the late blight epidemic over the experimental area. The Green Mountain variety, which is very susceptible both to disease of the foliage and to rot of the tubers, was used. Planting was on June 4.

The fungicides were applied with a tractor-sprayer unit which delivered approximately 120 gallons per acre at a pressure of 375 pounds per square inch. The boom carried four nozzles per potato row, two being above the plants and two being on drop pipes.

The sprays were applied on July 20, 30, August 10, 20, 27, September 3, 10, giving a mean interval of 8.7 days. In the split schedule, the change from nabam to Bordeaux was made on August 27, at which time the unsprayed check plots had reached a defoliation of 40 per cent. During the season insects were controlled by spraying all rows with an insecticide mixture as conditions required.

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[2] Plant Pathologist.

The weather during July and August of 1953 was very conducive to the development and spread of late blight. The border and buffer rows were inoculated by sprinkling them with a water suspension of late blight spores on the evening of August 12 and the epidemic built up evenly and rapidly, the unsprayed plots showing a defoliation of 45 per cent at the end of the month. From the 27th of August, the date on which the change to Bordeaux was made in the split schedule until the experiment was terminated on September 17 by spraying the plants with a sodium arsenite top killer, there were nine days of recordable precipitation, the total fall being 2.06 inches. Of this amount 1.0 inch fell on September 14-15. There were also several periods of light mist and drying conditions were extremely poor throughout most of this period.

The results of two defoliation readings are given in Table 1, in which it is indicated that a straight programme is superior to a split one in so far as the fungicides used in this test are concerned. It is suggested that Bordeaux mixture superimposed on nabam is not so effective as when built up on itself. In the laboratory an artificial weathering process with water removed three times the amount of copper from glass slides on which Bordeaux had been sprayed over a deposit of nabam + zinc sulphate than from slides carrying Bordeaux mixture only.

The plots were harvested on October 14, or 27 days after top killer had been applied. It was found (Table 1) that the treatments followed the same order in respect to losses from tuber rot as for the decimations of the foliage, the crop from the split schedule plots suffering the greatest loss.

Because of the pressure of other projects the study on the split schedule was abandoned for several years; but in 1961, at the request of a number of grower groups from Prince Edward Island to Manitoba, it was resumed. The field test was laid out and conducted in the same manner as in 1953 except that the now more popular maneb replaced the nabam + zinc sulphate (zineb) mixture.

The season of 1961, however, was unusually warm and dry, and no blight developed. The unsprayed check plots, probably because they alone were not damaged by the passage of the equipment through them, gave the highest yield at 410.3 bushels per acre. The maneb-treated plots were second with 404.1 bushels. The Bordeaux plots, with 381.0 bushels per acre, had the lowest yield, and this may be a reflection of copper phytotoxicity in a season of high temperature and little rain. The plots on the split schedule, having less time for copper injury and Bordeaux interference with transpiration to effect the yield, placed between the plots on straight maneb and Bordeaux schedules, the yield being 397.5 bushels per acre.

Table 1. Foliage and tuber data, 1953

Treatment	% Defoliation Sept. 3	% Defoliation Sept. 15	Total bu./ac.	Rot bu./ac.	No. 1* bu./ac.	% Rot
Bordeaux	0	7	352.6	4.6	320.4	1.3
Nabam	2	9	420.7	6.3	389.9	1.5
Split Schedule	3	16	390.9	14.8	352.8	3.8
Check	65	100	291.5	16.6	241.8	5.7
S.D. 5%			31.2		31.3	2.8
S.D. 1%			44.9		45.0	-

*Total yield less small tubers and rotted tubers.

Table 2. Foliage and tuber data, 1962

Treatment	% Defoliation Sept. 4	% Defoliation Sept. 17	Total bu./ac.	Rot bu./ac.	No. 1* bu./ac.	% Rot
Bordeaux	20	40	352.6	42.9	261.8	12.2
Maneb	9	16	434.5	58.8	330.6	13.5
Split Schedule	15	37	372.4	66.6	257.4	17.9
Check	85	100	189.8	40.2	99.0	21.2
S.D. 5%			31.8		43.6	
S.D. 1%			45.7		62.7	

*Total yield less small tubers and rotted tubers.

Table 3. Foliage and tuber data, 1963

Treatment	% Defoliation Sept. 3	% Defoliation Sept. 16	Total bu./ac.	Rot bu./ac.	No. 1 bu./ac.	% Rot
Bordeaux	2	45	465.2	48.0	378.0	10.3
Maneb	3	30	474.7	86.4	355.1	18.2
Split Schedule	3	36	445.0	70.2	330.8	15.8
Check	44	100	360.5	174.2	147.2	48.3
S.D. 5%			29.7		60.9	11.5
S.D. 1%			42.6		87.2	16.6

The seasons of 1962 and 1963 were wet and humid and severe blight epidemics developed in both years. During the July-September period of 1962 a measurable amount of rain fell on 44 days to give a total of 16.38 inches, approximately 6.0 inches above normal for the region. The same period in 1963 was almost as wet, 14.56 inches of rain being recorded.

In both years insects were controlled by spraying all rows with Thiodan, three applications being given in each season.

The plots in 1962 were planted on June 5 and the eight spray dates were July 19, 29, August 6, 14, 21, 28, September 4, 10, the mean interval being 7.6 days. In the split schedule the change from maneb to Bordeaux was made on August 21. The first blight lesions, the result of natural inoculation, were observed in unsprayed plots near the end of July. These plots and all buffer rows were completely defoliated by the beginning of September. The data of the defoliation readings are given in Table 2.

The top killer Reglone was applied on September 21 and the tubers were lifted and examined on October 5. The data on yields and tuber rot are shown in Table 2.

In 1963 the planting date was June 3. The fungicides were applied on July 19, 30, August 8, 20, 28, September 5, 12, dates which gave a mean interval of 9.2 days. For the split schedule the shift from maneb to Bordeaux was made on September 5, on which date the unsprayed check plots had reached a defoliation of nearly 50 per cent, and about 4 per cent of the leaves in the maneb-treated plots were showing late blight lesions.

Reglone was applied on September 18 and harvesting was done on October 3. The data for 1963 are presented in Table 3 and a three-year summary (1961 results excluded) of the experiment is contained in Table 4

Table 4. Three-year summary of straight vs. split test.

Treatment	Total bu./ac.	Small bu./ac.	Rot bu./ac.	No. 1 bu./ac.	% Rot	% Defoliation [1]
Bordeaux	390.1	38.2	31.8	320.1	8.2	1.6
Carbamate	443.4	34.3	50.5	358.5	11.4	12
Split Schedule	402.8	38.6	50.5	313.7	12.5	18
Check	283.9	40.9	77.0	166.0	27.1	100 approx.

[1] Means calculated at time check plots were 97 to 100% defoliated.

The results obtained in these first years of study suggest that a straight schedule of a carbamate or of Bordeaux mixture is slightly superior, in controlling foliar blight, to a split schedule of these fungicides in which a carbamate is used for the early-and mid-season sprays and Bordeaux is used for the late-season sprays. It is indicated, too, that the split schedule results in fewer bushels of saleable potatoes, caused, in part, by a higher percentage of loss from late blight tuber rot.

As stated under the title, this paper is a progress report, a statement which implies that the evidences are neither complete nor conclusive and that further studies are to be made and reported on. In the future some changes may be introduced. For example, it has been suggested that a fifth treatment be added. This treatment would be a variation of the split schedule in that the carbamate would be used for the early sprays only, both the mid-season and late sprays being Bordeaux mixture. It might be desirable, too, to introduce a proprietary copper fungicide in place of Bordeaux mixture.

EXPERIMENTAL FARM,
CANADA AGRICULTURE,
CHARLOTTETOWN, P.E.I.

BACTERIAL BLIGHT IN REGISTERED FIELD BEAN CROPS IN SOUTHWESTERN ONTARIO, 1963

M.D. Sutton and V.R. Wallen[1]

During the summer of 1963 twenty fields of registered beans were inspected. Each field was inspected three times during the first week of July, August and September respectively. All the fields examined were located in southwestern Ontario, the principal bean-growing region of Canada. The purpose of the three inspections was to observe and compare the incidence and development of bacterial blight in bean fields grown from seed from three foundation seed sources. Of the 20 fields, 7 fields were grown from foundation seed imported from Michigan 8 fields were grown from foundation seed produced at the Ridgetown Agricultural School, Ontario; and 5 fields were seeded with the growers' own 1962 foundation seed. The number of fields inspected of each of the varieties was: Michelite '62, 2; Michelite, 3; Sanilac, 10; Seaway, 4 and Saginaw 1.

The first inspection was made to determine the prevalence of seed-borne infection in the bean crop at the seedling stage and to distinguish, if possible, the symptoms of halo blight (Pseudomonas phaseolicola) from both common blight (Xanthomonas phaseoli) and fuscous blight (Xanthomonas phaseoli var fuscans). At this time, the bean plants either were just emerging or at a very early stage of development. No recognizable symptoms of the bacterial blights were observed.

During the second inspection the 7 fields grown from Michigan seed showed no visible symptoms of infection by any of the bacterial-blight pathogens. These fields included 2 each of Michelite '62, Sanilac and Seaway and 1 of Saginaw. Of eight fields seeded with Ridgetown seed, 1 of 3 fields of Michelite and 3 of 5 fields of Sanilac showed a trace of blight infection. In 5 fields sown with the growers' own 1962 foundation seed, a trace of blight was found in 2 of 3 fields of Sanilac while no infection was observed in 2 fields of Seaway. The seed-borne nature of the blight infections was apparent by the production of discrete loci of infection in scattered areas of the fields. On the basis of symptomatology all the infections were considered to be caused by the fuscous and common blight pathogens. Laboratory examination of infected leaves from many fields revealed the presence of X. phaseoli var fuscans but not X. phaseoli. No halo blight symptoms were observed.

The third inspection was made in September to the spread of infection in the fields before the crop had fully matured. In this survey pod infection was the criterion for disease diagnosis. Only those fields in which infection had previously been noted were found to have a trace of pod infection.

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In marked contrast to the extensive spread of blights observed in this area in 1961 and 1962 the incidence of blights in the fields inspected was notably slight.

Rainfall during June, July and August in the area was but 50 to 60 per cent of that for the same three months of 1961 and 1962. This fact undoubtably affected the development and spread of the blight pathogens which require conditions of high humidity for multiplication and rainfall for their distribution through the field. Temperatures for the region in 1963 were normal, as they were in 1961 and 1962 when bacterial blight was epidemic in the area. Although 1963 was a poor year for the comparison of blight development, field examination indicated that imported Michigan seed produced blight-free crops. As mentioned above, some fields produced from other sources were infected. Laboratory examination of samples of the growers' own foundation seed indicated that 2 samples of Sanilac and 1 sample of Seaway were infected with the fuscous blight pathogen. Under conditions of low rainfall and normal temperatures only 1 field of Sanilac grown from seed known to be infected showed visible symptoms of infection. Breeder seed from Michigan is produced under rigid inspection in the blight-free arid areas of Idaho. This seed is then grown 1 year in northern Michigan and inspected at regular intervals by a plant pathologist. Only seed from disease-free fields is marketed as foundation seed. Ridgetown breeder's seed and the growers' own foundation seed is produced in the blight-infected area of southwestern Ontario. As a consequence, a trace of blight in either breeder or foundation seed can increase in subsequent generations of registered seed to a serious level unless the number of generations from foundation seed is limited.

While examining the 20 fields other diseases were noted. Six fields showed a trace of bean rust, Uromyces phaseoli var typica; 3 fields of Sanilac, 2 fields of Seaway and 1 field of Saginaw. The field of Saginaw was also severely attacked by Sclerotinia wilt. The habit of growth of this variety, particularly in shaded areas where high humidity prevails, tends to promote infection by Sclerotinia sclerotiorum. Two fields of Michelite were moderately infected with the soil-borne root rot pathogen Fusarium solani f. phaseoli.

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SOYBEAN DISEASES IN ONTARIO, 1963

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In 1963 pod and stem blight (Diaporthe phaseolorum var. sojae (Lehman) Wehm.) was less prominent than in 1962 on all soybean varieties and experimental lines grown at the Central Experimental Farm, Ottawa. Symptoms were confined to stem and branch tissues near the base of the plant and occurred primarily on the early maturing varieties Comet and Merit. Only traces of the pathogen were evident in Hawkeye, Blackhawk and Harosoy plants, many of which were not fully mature by October 15. Lincoln plants were still green on Oct. 20 and appeared free from disease. Symptoms of stem canker (D. phaseolorum var. caulivora Athow & Caldwell) were absent from all varieties and lines examined.

At the Western Ontario Agricultural School and Experimental Farm, Ridgetown, on September 17, pod and stem blight had developed only on the varieties Merit and Chippewa. Stem canker symptoms were observed on less than 1% of Harmon, Hawkeye and Ford plants while up to 10% of Lincoln plants were infected.

Stems of mature Merit plants at the Research Station, Harrow, were moderately affected by pod and stem blight on September 18, but pods were free from symptoms. Stem canker was observed on a number of plants of Lincoln and Clark. In a commercially-produced field of Lincoln in the Harrow area, approximately 1% of the plants exhibited symptoms of stem canker. Affected plants were mature, showed typical stem lesions at one or more lower nodes, and had a higher proportion of unfilled pods than healthy green plants. Neither pod and stem blight nor stem canker was observed in 15 commercial fields of Harosoy examined in the area, September 17-19.

Diaporthe was not isolated from surface sterilized seeds obtained in September from affected Lincoln, Merit or Clark plants grown at Harrow. Seeds harvested in October from pod and stem blight affected plants grown at Ottawa were also free from Diaporthe. Movement of the pathogen into the seed was apparently affected by the unusually dry weather that prevailed while the crop matured. Plants similarly affected with pod and stem blight at Ottawa in 1962 produced seed containing up to 13% internal infection by Diaporthe. Seed produced commercially in Ontario in 1962 had a generally low incidence of Diaporthe, whereas serious germination problems associated with Diaporthe infection were encountered in seed produced in southern Indiana and Illinois for 1963 planting.

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Corynespora cassiicola (Berk. & Curt.) Wei was reported for the first time in Canada from roots of soybean plants collected at the Harrow Research Station in September, 1963 and was subsequently found on soybean roots from experimental plots at Ottawa (2) and Ridgetown. In the southern United States, this fungus causes target spot of soybeans and has become prevalent in southern producing areas on susceptible soybean varieties and other crops since it was first reported in 1945. A root rot of soybean caused by this fungus was described in Nebraska and did considerable damage under conditions of cool, moist weather early in the growing season (1). Progress of the disease was apparently arrested with the onset of warm summer weather.

In Ontario foliar symptoms characteristic of target spot were not observed, and no assessment could be made of root damage, because of the lateness of the season when the fungus was found. C. cassiicola was prevalent on the roots of all varieties and lines examined at Ottawa but was not found to be seed-borne. Plants were available from only one field of commercially produced soybeans at the time the fungus was first observed and these were free from C. cassiicola. Therefore, although the fungus has been demonstrated in experimental plots at three locations in Ontario, no information is yet available on its distribution in commercial plantings. Since the fungus is seed-borne in the southern United States, it may have been introduced in seed imported for experimental purposes at the three locations.

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SUNFLOWER DISEASES IN MANITOBA IN 1963

by J.A. Hoes and E.D. Putt[1]

Sunflowers were planted on 33,000 acres in Manitoba in 1963, mainly in the Red River Valley. This was the highest acreage since 1958. The weather was normal in the spring, followed by above-normal precipitation until July 25 and below-normal precipitation for the remainder of the growing season. Temperatures were well above-normal during July and August and into late October, allowing the crop to mature well. Yields were high, being 890 pounds per acre for the hybrid varieties Admiral and Advent, 850 for Mennonite and 1,000 for Peredovik, a recently-imported Russian variety which is promising because of high yield and high oil content.

Fourteen fields of hybrid varieties, 19 fields of Mennonite and 15 fields of Peredovik were examined for disease on August 27 and September 17. Mr. Peter Bergen, Co-op Vegetable Oils, Ltd., Altona, Manitoba, assisted in the survey.

Leaf mottle (Verticillium albo-atrum) was widespread, though in general yield reductions due to this disease were only small. This was in contrast to 1962 when severe losses were suffered due to Verticillium wilt. Disease severity in 1963 was slight (trace - 10% infected plants) in 36 fields of all varieties including all fields of Peredovik. It was moderate (15-40%) in one field of Admiral and four fields of Mennonite, and severe (50-75%) in four fields of hybrid varieties and one field of Mennonite. In one severely-infected field of Admiral the disease caused an appreciable loss of 25% in yield. The limited damage by Verticillium in 1963 was possibly due to the above-average or high precipitation along with average temperatures up till mid-June, a condition that resulted in vigorous plant growth.

Rust (Puccinia helianthi) occurred in varying degrees of pustule density (trace - 60%) on 80-100% of the plants in all fields of Mennonite and Peredovik. Some rust was found in one of the 14 fields of hybrid material. The prolonged hot weather in July and August seemed to arrest rust development.

Downy mildew (Plasmopora halstedii) occurred sporadically. It affected 1% or less of the plants in 14 fields and was absent in 32 fields of all varieties. In two fields, one each of Mennonite and Peredovik 30% of the plants were prematurely killed and yield loss was considerable.

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Sclerotinia wilt. (S. sclerotiorum) affected a trace to 2% of the plants in 17 fields of all varieties. In two fields 5% of the plants suffered from this disease.

Septoria leafspot (S. helianthi) and aster yellows affected up to 1% of the plants in, respectively, five and seven fields of all varieties.

A serious disorder, which at first was of doubtful etiology, was observed in a small field of a Russian variety near Morden. On July 18, when the condition was first observed, the plants were about six feet tall and near flowering. The condition was limited to an area of about 25 x 30 feet and all plants within it were affected, the plants at the centre of the area being the most seriously damaged. Their stems were brown, somewhat desiccated and bent double at the centre so that the head rested on the ground. The pith was at first discolored and water-soaked. Sometimes the pith was lacking and the stem hollow. The midrib and adjacent interveinal tissue of the leaves was necrotic. The leaves did not die as rapidly as might have been expected considering the gross symptoms of the stem, suggesting that the vascular system had not been seriously affected. Towards the periphery of the area only the upper portions of the plants were affected; the stems were flattened, wrinkled, brown, twisted and weakened, allowing the heads to hang down but most of the leaves were healthy. The disease did not spread and plants at the edge of the area usually recovered. Sackston (1) has described a disorder of sunflower with similar symptoms. Young (2) described ribbon stem of tomato with symptoms similar to those outlined above. Ribbon stem was attributed to lightning injury. Dr. M.L. Kinman of Texas A. and M. College, College Station, Texas, when visiting the area on September 4, thought the symptoms were typical of lightning injury as observed by him in other crops. The occurrence of a severe electrical storm in the neighborhood of the affected field just prior to discovery of the disease supports the conclusion that the disorder in sunflowers here described was caused by lightning.

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EXPERIMENTAL FARM,
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RAPE DISEASES IN SASKATCHEWAN IN 1963

T.C. Vanterpool[1]

The above-average rainfall during the 1963 growing season favored the growth of oil-rape and also its diseases. However, the increase in yields from the abundant moisture more than counterbalanced the increased losses from disease in comparison with average years. The estimated rape acreage in the province in 1963 was 232,000 with a yield of 260,000,000 pounds or 1121 pounds per acre. This is the highest yield on record for the province.

It is suspected that the late rains, especially in northern areas, caused an increase in seed-borne fungi over the average incidence. Consequently it would be advisable to make germination tests on rape seed intended for seeding. If germination is below 80% the application of a fungicidal seed dressing is recommended.

Mention should be made of the gradual increase in the acreage of commercial mustard grown in the province. There were 80,000 acres sown with an average yield of 960 pounds per acre. Mustard is susceptible to many of the diseases found on rape. To date only slight amounts of Alternaria, Albugo, Sclerotinia, Fusarium and bacterial diseases have been found on mustard but it is to be expected that as the acreage increases, especially in northern regions, the diseases on this crop will also increase. Such was the story with rape after its introduction.

Aster yellows virus. Traces could be found in most fields examined just before harvest. One field with as high as 5 per cent of the plants affected in some areas was found near Spalding. This is the highest incidence of AYV since the epidemic of 1957.

White rust and staghead (Albugo cruciferarum). The conspicuous stage of this disease and the only one observed by most farmers is the deformation or hypertrophy of the tips of the flower stalks as maturity approaches. To use the name "White rust" for this stage of the disease is confusing to growers and it has been found more convenient when talking with them to refer to the enlarged stem tips as the "staghead" disease. The combined name "white rust and staghead" is proposed for use of the disease on rape in Saskatchewan.

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The white rust and staghead disease is well established in the relatively moist rape-growing areas of the parkbelt. Its severity in a field depends largely on the crop sanitation and rotation practices of the individual grower. The moist conditions in 1963 accounted for its more general prevalence. Five fields were found with from 3 to 10 per cent infection in scattered areas (Valparaiso, Tisdale, Nipawin, Spalding, Meadow Lake). One field at Glaslyn showed a slight infection of the late, green, side branches and none on the more mature stems.

Black and grey spot (Alternaria brassicae and A. raphani). Stem lesioning was conspicuous in some northern fields. The disease extended further south than usual and was slight in some areas of the University plots at Saskatoon. The pods and stems in northern swathed fields showed more evidence of Alternaria lesioning and discoloration than those in standing fields. This increase in Alternaria on rape in the swath during moist weather is probably due mainly to the saprophytic A. tenuis, rather than to the parasitic species A. brassicae and A. raphani. Butler (Fungi and Disease in Plants, Calcutta, 1918, P. 303) has observed that in rape the seed matures normally in the stack, and the fungus (A. brassicae) ceases to grow after the plants are cut. He also states that in Europe losses in affected rape-seed may be avoided by early harvesting. It would seem that early swathing of heavily infected fields in the northern disticts in Saskatchewan would be beneficial, especially in decreasing the amount of seed-borne infestation by Alternaria spp. Plating out of 1963 rape-seed from Dorintosh, Meadow Lake, North Battleford, Nipawin, Carrot River and Melfort showed that all samples were carrying unusually high percentages of A. brassicae. A. raphani was less commonly isolated. There is some evidence that in drier years, for example, 1961, A. raphani is more common than A. brassicae. Fungicidal seed treatment of low-germinating 1963 rape-seed samples from northern districts should be beneficial. Alternatively, rape-seed of good germinatibility from drier prairie districts could be used for seeding purposes without fungicidal dressing.

Basal stem rot (Sclerotinia sclerotiorum). This disease occurs regularly in moist years in northern areas. Several fields with trace infections were recorded; two had 1-5 per cent and one 5-20 per cent. The occurrence of this disease on rape is probably higher than records indicate since large numbers of plants with symptoms of basal stem blight do not always show internal sclerotia. It is felt that the absence of sclerotia in the presence of suspected symptoms in a particular field may indicate late infection.

Black blight or ring spot (Mycosphaerella brassicicola). This disease has become so widespread that most fields in the north and north east show grey to bluish-grey stubble instead of a clean, pale tan colour after cutting. This difference between southern and northern rape fields is conspicuous. There is some indication that the effects of black blight on the

plants are not as severe as the extensive black discoloration of the stems and pods indicates. For instance, early harvested plots of Arlo (Polish type) were heavily discoloured, whereas plots of late-maturing Golden and Nugget (Argentine type), which were still quite green, showed slight amounts of the disease. This suggests that the disease develops rather late in the life of the plant; it probably does not suggest that the Polish type is more susceptible than the Argentine type because heavily diseased fields of Argentine are commonly observed. Timeliness of rainfall may be a contributing factor. This disease is more regular in appearance and intensity on the rape crop in the parkbelt than any other disease.

Blackleg (Phoma lingam). Two severe infections (up to 10%) were located this summer, one at Annaheim and one north of North Battleford. This indicates that blackleg is more prevalent than our surveys have hitherto indicated. The disease is being studied because of its serious potential. Phoma lingam, which is highly pathogenic on rape, was isolated by G.A. Petrie from penny-cress Thalspi arvense L. in a rape field at Saskatoon. This finding is of considerable interest not only because of the general prevalence of penny-cress, but because it is a winter annual it may prove to be more serious as an overwintering host than annual hosts such as the various mustard weeds.

Powdery mildew (Erysiphe polygoni). No powdery mildew was collected on rape during the surveys in northern areas. During early October, however, two late plots of rape at Saskatoon became heavily infected. In one of these fruit bodies thought to be cleistothecia were observed with the naked eye. Surprisingly, on microscopic examination they were found to be mature pycnidia of the mildew parasite Ampelomyces quisqualis Ces. (syn. Cicinnobolus cesatii de Bary). This hyperparasite has previously been reported on Podosphaera oxyacanthae in Manitoba and Saskatchewan and on Microsphaera in Manitoba (Bisby, G.R. The Fungi of Manitoba and Saskatchewan, 1938. p. 132). It has not previously been observed on the rape mildew in Saskatchewan. The Ampelomyces had almost completely suppressed the production of conidia by the Erysiphe. In the other rape plot, about one mile from the first, no Ampelomyces was detected and the Erysiphe was producing copious conidia. The Ampelomyces was readily obtained in pure culture where it fruited readily.

Root rot (Fusarium spp.). In experimental work on rape, conducted by a graduate student in the greenhouse, about two dozen pots of rape died because of a severe root and basal stem rot when the plants were coming into flower. The soil was the usual greenhouse mixture. The fungi most commonly isolated, and which proved to be moderately pathogenic on rape seedlings, were later identified by Dr. C. Noviello, Canada Agriculture Research Station, Saskatoon, as Fusarium acuminatum, F. solani and F. equiseti. Isolations and pathogenicity tests were made on two separate occasions. F. poae has previously been found associated with root rot of rape. Present evidence indicates that Fusarium root rot of rape is usually caused by the combined action of two or more species. To date, the wilt-producing species F. oxysporum f. conglutinans has not been detected on rape in Saskatchewan.

OCCURRENCE OF LAWN DISEASE AT SASKATOON, 1963

C. Noviello[1]

Although the survey was carried out mainly at Saskatoon, several lawn samples were received from different localities in Saskatchewan. Isolations on different artificial media were made throughout the survey. The diseases that were observed in the following chronological sequence were:

SNOW MOLD (low-temperature basidiomycete). At the first spring thaw several lawns were found affected. With a few exceptions damage was moderate and nearly all the affected lawns recovered with good care and warmer weather.

BLIGHT. In late April and early May dead patches of variable size were observed on several lawns. Isolations yielded the following organisms: Pythium sp., Bipolaris sorokiniana, Drechslera poae, Fusarium roseum "culmorum", F. roseum "acuminatum", and F. roseum "equiseti". The blight etiology was rather controversial because more than one of the above-mentioned organisms was isolated from the same dead patch and because winter injuries could not be excluded.

HELMINTHOSPORIUM LEAF, CROWN, AND ROOT DISEASES. The most outstanding feature of the lawn disease picture was the widespread occurrence of leaf, crown, and root lesions on Poa pratensis incited by Helminthosporium spp. The diseases became first evident in May and then developed throughout the summer and even into the fall. Damage was sometimes severe and several lawns had to be reseeded or resodded. The Merion variety of bluegrass, generally considered to be resistant, was severely affected by both leaf spot and foot rot.

FAIRY RINGS, caused mainly by Marasmius oreades and Lepiota naucina, was another common but not very serious disease in Saskatoon. The unusually rainy season might explain the light damage.

ANTHRACNOSE (Colletotrichum graminicola) was widespread and sometimes a very serious problem. The disease was first noticed in July and reached a peak late in August. One lawn was so badly damaged that it had to be reseeded. The disease was observed almost exclusively on Festuca rubra.

LEAF RUST (Puccinia poae-nemoralis) was observed on Poa pratensis during August in several lawns, but the disease was rated severe in one lawn only. Generally, damage was very light. Pycnidia of Darluca filum were found consistently in uredia.

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POWDERY MILDEW (Erysiphe graminis). Late-season infection by powdery mildew was encountered on many lawns, particularly in shaded areas. The disease was prevalent on Merion bluegrass, but damage was light.

ASCOCHYTA LEAF SPOT. This disease was observed in August but was without consequence as only a few infected leaves were collected in three lawns.

SEPTORIA LEAF SPOT. A trace infection was recorded on Poa pratensis in one lawn, late in August.

ASTER YELLOWS AND LEAFHOPPER DAMAGE IN MANITOBA IN 1963

P.H. Westdal[1] and H.P. Richardson[1]

Aster yellows was more severe in Manitoba in 1963 than it has been in any year since the severe outbreak in 1957. There was nearly 100 per cent loss in untreated lettuce, about 33 per cent loss in celery and carrots and about 5 per cent loss in onions. Many other vegetables and ornamentals were infected to varying degrees. Field crops, however, were not as severely infected. The disease was present in all fields of barley and flax examined and infection ranged from a trace to 5 per cent. A trace of infection occurred in buckwheat, sunflowers and rape.

In 1963, the six-spotted leafhopper arrived earlier, in larger numbers and with a higher proportion of viruliferous leafhoppers than in any year since 1957. By mid-May it was possible to predict that there would be severe damage from aster yellows.

Following the initial migration on April 27, conditions in the spring and in the early summer were favorable for rapid multiplication of the insect and of the virus. The leafhopper population reached a peak in early July (3000 to 4000 per 100 sweeps) and aster yellows symptoms were common on susceptible crops and weeds. These extremely high populations were comparable to those reported in 1957 but in 1963 the percentage of viruliferous leafhoppers was considerably lower.

Actual destruction of plants in the field by the feeding of leafhoppers was recorded for the first time in 1963. Vast numbers of leafhoppers, driven out of late summer fallow by cultivation, destroyed the margin of an adjoining field of oats.

Experiments were conducted at Portage la Prairie and Winnipeg to determine the efficacy of 8 different insecticides for the control of the six-spotted leafhopper and aster yellows on flax and barley in field plots. Bayer 39007 (o-isopropoxyphenyl methylcarbamate), a systemic carbamate, applied in a granular formulation at the time of seeding reduced aster yellows infection in flax from 14.8 per cent to 2.6 per cent and in barley from 11.0 per cent to 1.3 per cent.

In order of effectiveness, on barley, the remaining chemicals, malathion emulsion concentrate, Bayer 39007 emulsion concentrate, Thimet granular (O, O-diethyl S [(ethylthio) methyl] phosphorodithioate), E.I. 47-470 granular (2-(diethoxyphosphinylimino) -4-methyl-1, 3-dithiolane), Delnav

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emulsion concentrate (2, 3- p - dioxanedithiol S, S - bis (O, O-diethyl phosphorodithioate), E.I. - 031 granular (2-(diethyoxyphosphinylimino)-1, 3-dithiolane), and Thiodan emulsion concentrate (6, 7, 8, 9, 10, 10-hexachloro-1, 5, 5a, 6, 9, 9a-hexahydro-6, 9 methano - 2, 4, 3-benzodioxathiepin 3-oxide also significantly (5% level) reduced aster yellows infection. In barley the reduction in infection, with Bayer granular, was accompanied by a 35.0 per cent increase in seed yield. This suggests that some late infection by aster yellows, which may have reduced seed yield, was undetected or that pests other than leafhoppers and diseases other than aster yellows were controlled. The latter part of this speculation, in particular, warrants further investigation.

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