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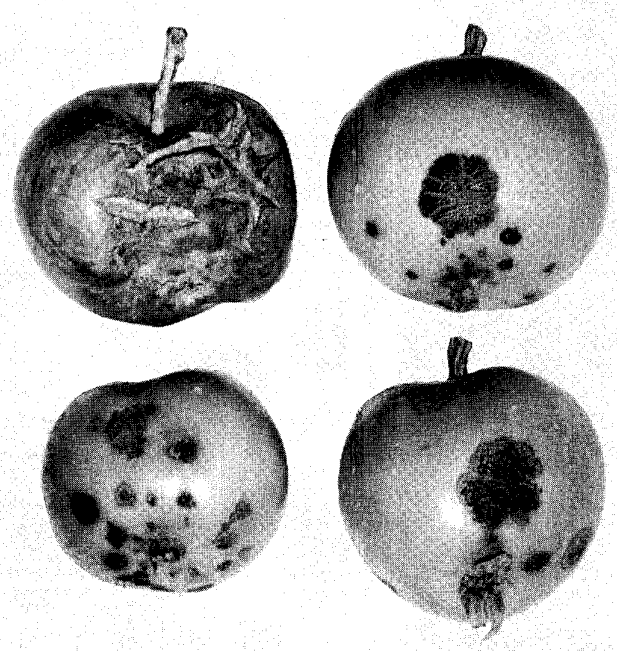
Canadian Plant Disease Survey

Compiled and Edited by D. W. Creelman



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CANADIAN PLANT DISEASE SURVEY

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CONTENTS

VIRUS DISEASES OF STRAWBERRIES IN EASTERN CANADA[1]

A. T. Bolton

Introduction

During 1961 and 1962 a survey was made of the viruses occurring in strawberries in eastern Canada. Strawberry plants were collected from fields in Ontario, Quebec, New Brunswick, Nova Scotia, and Prince Edward Island. Most of these plants were collected from fields where certified virus-tested stock had not been used.

The strawberry plants were tested for virus content by grafting to the East Malling strain of Fragaria vesca, to seedlings of Alpine F. vesca and to Miller's F. vesca.

The results of the survey are described in this paper.

Materials

Strawberry plants were collected at random in fields (Fig. 1), placed in plastic bags, and mailed to the laboratory. They were then planted in pots and allowed to become well established. Four leaves from each plant were grafted to the East Malling strain of Fragaria vesca. Leaves were also grafted to seedlings of Alpine F. vesca and to Miller's latent free F. vesca.

The commercial variety Empire was used for determining the presence of veinbanding virus. In all cases symptoms showed on the indicator plants during the fifth week after grafting and these were read after seven weeks.

The descriptions of symptoms of virus diseases by Mellor and Fitzpatrick (4) were used in identifying most of the viruses found during the survey.

Survey Results

Plants from Ontario included the varieties Premier, Valentine, Sparkle, Louise, Mackenzie, and Senator Dunlap. The varieties Senator Dunlap and Sparkle were collected in Quebec, New Brunswick, Nova Scotia, and Prince Edward Island.

All Senator Dunlap plants collected in Quebec, New Brunswick, Nova Scotia, and Prince Edward Island produced typical mottle symptoms on the indicator plants. Twelve of 22 plants of this variety from Ontario were infected with mottle virus. These 12 plants were from 4 locations scattered throughout the province. Of the 10 plants collected in Ontario that did not have mottle, 9 were infected with veinbanding and 3 with a veinbanding-latent C complex.

One hundred and twenty Sparkle plants taken from 36 locations in the five provinces contained latent A virus. Thirty-two, representing 3 locations in Prince Edward Island, 2 locations in Nova Scotia, 1 location in New Brunswick, 1 location in Quebec, and 7 locations in Ontario were infected with mottle virus. Of the 88 plants remaining, 70 from 18 locations in Ontario and 8 from 2 locations in Nova Scotia caused symptoms of the veinbanding-latent C complex. Ten plants, representing 5 locations in Ontario, were infected with veinbanding.

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[1]Contribution No. 127 from the Genetics and Plant Breeding Research Institute, Canada Department of Agriculture, Central Experimental Farm, Ottawa, Canada.

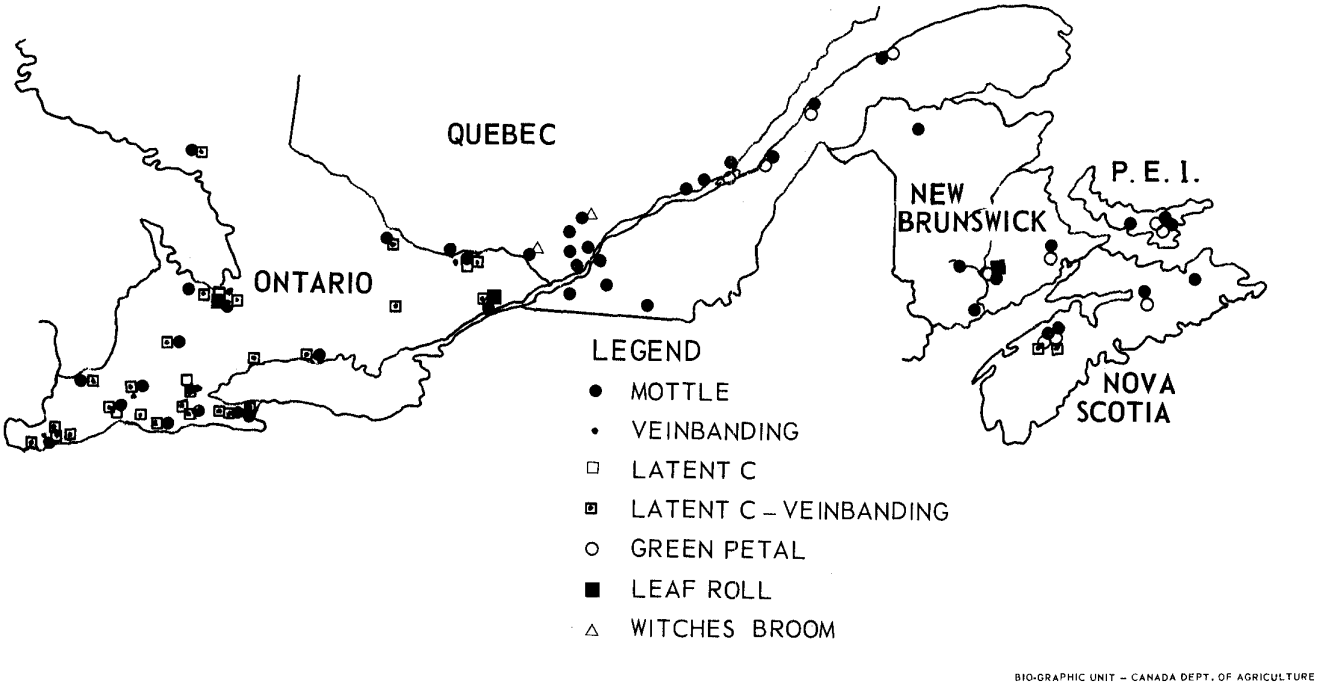

Figure 1. Map showing location of strawberry plant collections and the virus diseases occurring on them.

All plants of the Premier variety collected in Ontario caused symptoms of virus disease. Seventy-two per cent of the plants were infected with the veinbanding-latent C complex. Twelve per cent contained mottle as well as the veinbanding-latent C complex, 10 per cent contained veinbanding alone, and 6 per cent latent C alone.

Valentine collected from 5 locations in Ontario was infected with mottle, veinbanding, and latent C combined while Mackenzie plants from 3 locations in Ontario were infected by the veinbanding-latent C complex.

Symptom Expression

Although most of the viruses present in the affected plants were identified as mottle, veinbanding, latent C, or a combination of these, there were cases when the indicators exhibited atypical symptoms.

Mottle

East Malling F. vesca was used for comparing mottle symptoms in all varieties. All plants containing mottle virus caused similar symptoms on the indicator with the exception of Sparkle taken from one locality in New Brunswick. These plants caused a very severe distortion of the leaves as well as the typical mottling symptoms resulting in a condition similar to curly-dwarf mottle

described by Frazier and Posnette (3). Proliferation of the crown was accompanied by both upward and downward curling and twisting of the leaves (Fig. 2). In addition, several necrotic spots were observed at an early stage of infection. These symptoms appeared 21 days after grafting. Since the plants failed to produce symptoms on the indicators after a heat treatment of 100°F for 24 days, it was concluded that they had been infected by a severe strain of the mottle-latent A complex.

Veinbanding and latent C

Infection by the veinbanding virus was widespread in Ontario in all strawberry varieties, and usually occurred in combination with latent C virus. Veinbanding was found on Sparkle plants in Nova Scotia, but was not encountered in Prince Edward Island, New Brunswick or Quebec. Symptoms of veinbanding virus alone occurred on East Malling F. vesca as more or less continuous yellow streaks along the veins, especially along the midrib, accompanied by moderate downcurling of the leaflets (Fig. 3). Where veinbanding occurred alone in the plants, there was no observable variation in symptoms expressed by different varieties from different localities.

Veinbanding in association with latent C was found in the majority of plants from Ontario. Symptoms produced on East Malling F. vesca by these plants were similar to those produced by veinbanding alone. In the commercial varieties Empire and Redcoat containing latent A, however, the symptoms were considerably more severe than those caused by veinbanding. The veinbanding - latent C complex from Premier, in which it produced no symptoms, produced a much more severe condition in these two commercial varieties. Leaf distortion and epinasty were greatly increased, and petioles and runners became necrotic within a short time (Fig. 4 and Fig. 5). Most of the infected Empire plants died within two months of the appearance of symptoms. The effect on the runner plants was also much more pronounced (Fig. 6).

Latent C alone was encountered in a few of the Premier plants examined. Early symptoms produced by this virus alone on East Malling F. vesca consisted of severe epinasty of the newly formed leaves and petioles (Fig. 7). Symptoms became much more severe as the disease reached chronic proportions causing dwarfing of the leaves and proliferation of the crown. In plants that did not contain latent A, no definite symptoms appeared.

Green Petal

Green petal was observed in all fruiting commercial plantations visited in Prince Edward Island and Nova Scotia. A small amount of infection by this virus was observed in New Brunswick and eastern Quebec. The disease was not observed in western Quebec nor in Ontario. The virus was readily transmitted by grafting to both indicator plants and commercial varieties. In the absence of floral structures, the disease was indistinguishable from witches' broom.

Witches' Broom

Plants showing witches' broom symptoms were found in all five provinces. The disease was severe in two plantations of Senator Dunlap in Quebec and in one plantation of Premier in eastern Ontario. The disease was transmitted by grafting to indicator plants and commercial varieties. Symptoms consisted, in all cases, of dwarfing of the leaves and proliferation of the crown.

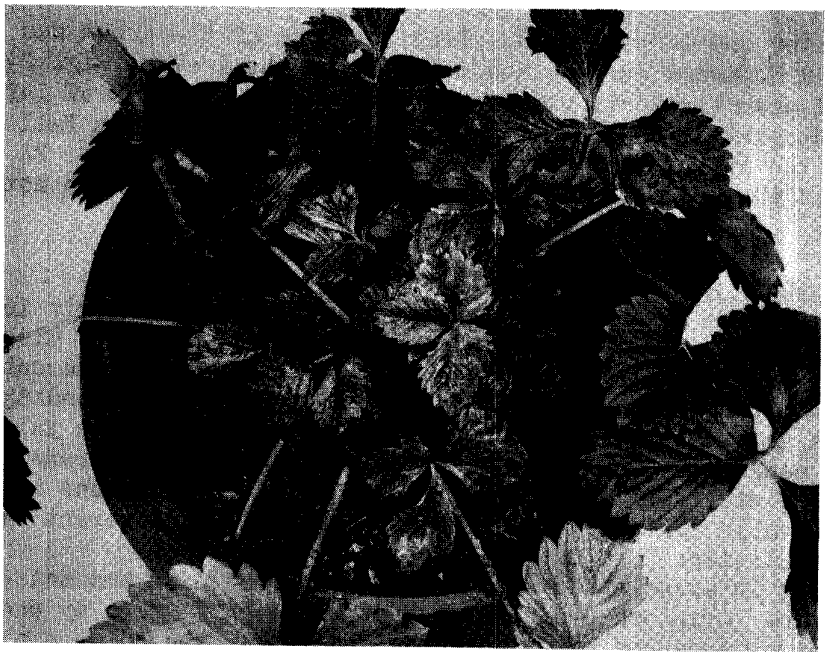

Figure 2. Mottle symptoms obtained after grafting Sparkle from N.B. to East Malling *F. vesca*.

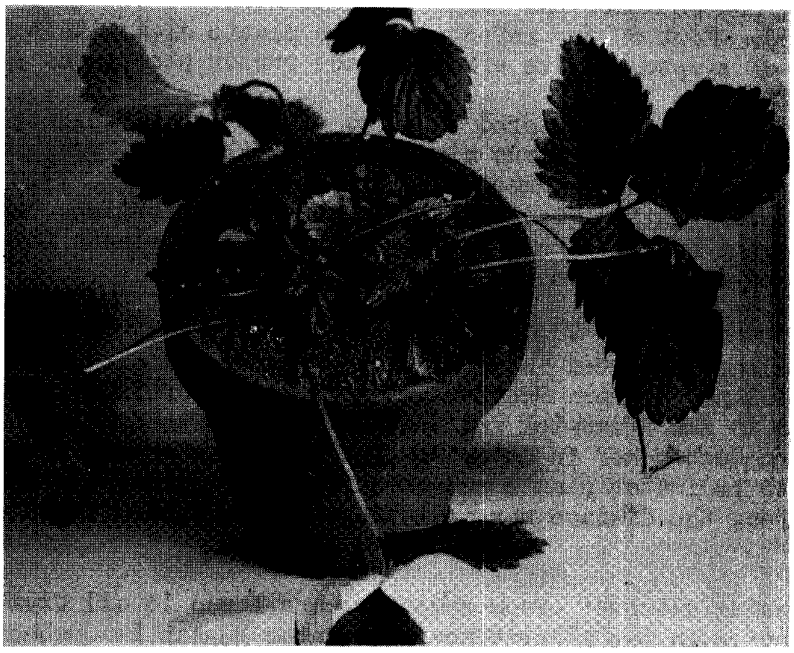

Figure 3. Veinbanding virus symptoms on East Malling *F. vesca*.

Figure 4. Veinbanding – latent C virus symptoms on the variety Redcoat.

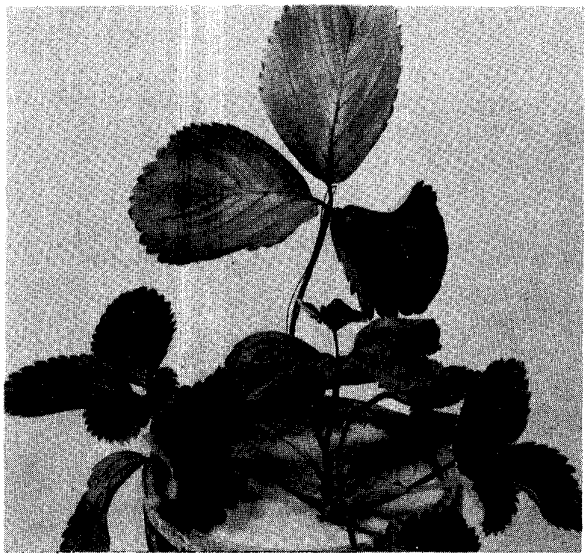

Figure 5. Veinbanding – latent C symptoms on the variety Empire.

Figure 6. Veinbanding – latent C symptoms on runner plants of Redcoat.

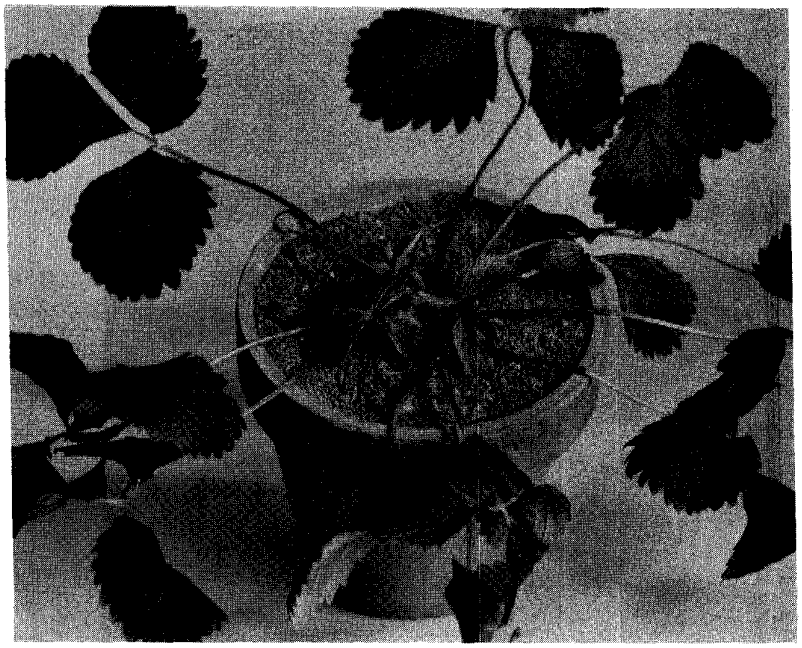

Figure 7. Latent C symptoms on East Malling *F. Vesca.*

Figure 8. Symptoms of strawberry leaf roll on the variety Sparkle.

Leaf Roll

Typical leaf roll symptoms were found in two plantations in Ontario and at one location in New Brunswick. The disease was severe in the Ontario plantations, and no fruit developed. The plants were dwarfed and the leaves severely rolled downward. Chlorotic streaks and powder-fine specks were present on the leaves (Fig. 8). The virus was transmitted to several commercial varieties by grafting, and typical symptoms appeared on all plants in 15-20 days.

Discussion

The important virus diseases occurring on strawberries in eastern Canada are veinbanding, mottle, latent C, and green petal.

Veinbanding, although symptomless on Premier and Senator Dunlap, produced severe symptoms in the presence of latent A on most other commercial varieties, especially Redcoat and Empire. In the presence of both latent A and latent C infection with veinbanding can be lethal. Because of this, it is preferable to use latent A-free plants for setting out new plantations. In several cases within the last five years, symptoms of the latent A-veinbanding complex have been observed on Sparkle, Redcoat, Empire, and Guardsman in Ontario. Yields of such infected plants have been reduced by as much as 50%. Veinbanding on Redcoat and Guardsman, in the absence of latent A, had very little effect on vigor and yield in greenhouse experiments.

Latent C virus occurring in the absence of veinbanding, but in the presence of latent A, did not appear to reduce vigor in commercial varieties. This virus does, however, increase the symptom expression of the veinbanding-latent A complex. The combination of veinbanding and latent C in the absence of latent A, as it occurs in most Premier plants in Ontario, has a marked effect on the general vigor. This reduction in vigor is most pronounced under conditions adverse for plant growth. The Premier plants which proved to contain latent C alone were collected from plantations showing exceptional vigor, and it was apparent that the virus had little effect on this variety. Redcoat and Guardsman, artificially infected with latent C in the absence of other viruses, showed no loss of vigor. When latent C was transmitted from these plants to latent A-free F. vesca no symptoms were produced. However, when leaves from East Malling F. vesca were grafted onto these latent C-infected F. vesca plants, symptoms appeared which were typical of those described by Mellor and Fitzpatrick (4) for the latent C-latent A complex, and by Demaree and Marcus (2) for type 2 virus.

From the results of this survey and preliminary greenhouse investigations, it appears that the veinbanding-latent A or veinbanding-latent C-latent A complexes are of major concern in Ontario.

Green petal virus is of great importance to strawberry growers in Nova Scotia, Prince Edward Island, New Brunswick, and eastern Quebec. The disease was not observed on strawberries west of Quebec City. This distribution agrees with that given by Chiykowski (1) for the strawberry green petal virus causing phyllody of clover.

Crinkle virus, described by Zeller and Vaughan (6) in Oregon and by Prentice (5) in Great Britain, was not encountered in the survey. There was no evidence that any of the components of yellows virus disease, as described by Mellor and Fitzpatrick (4) in British Columbia, are present on strawberries in eastern Canada.

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A SURVEY OF GREEN PETAL VIRUS IN NEW BRUNSWICK AND SOME EFFECTS OF BARRIERS ON SPREAD[1]

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Abstract

A 3-year survey of green petal disease in 22 strawberry fields in central New Brunswick showed only light infections in scattered areas. Barrier crops around small plots did not prevent spread of aster yellows virus into China asters and carrots, but 30-foot barriers of grain and timothy or interplantings of China asters and carrots showed promise of reducing spread of green petal into strawberries.

Introduction

Green petal disease of strawberry (6) has been increasingly troublesome in experimental plots at the Canada Department of Agriculture Horticultural Substation, McDonald's Corner, New Brunswick, since 1953 (3). In 1960, a plot containing more than 20 varieties was ploughed under because a large percentage of the plants was infected. Growers in the area appeared to experience little trouble during this time, although the extent and economic importance of the disease in commercial fields were not known. Aster yellows disease, meanwhile, was commonly present in almost all of the home gardens in the area. The relationship of this virus to that which causes green petal is not well understood, although strawberry green petal and clover phyllody diseases have been shown to be caused by the same virus (2, 4). Frazier and Posnette (4) suggest that green petal may be a strain of aster yellows virus but differences in symptoms on several hosts imply a distinction between it and both the eastern and western aster yellows viruses. Chiykowski (2), meanwhile, has transmitted green petal virus from strawberry to China aster with Macrosteles facifrons (Stal) producing symptoms typical of clover phyllody virus infection.

In 1961 we began a 3-year survey to determine the extent of green petal disease in some of the more important commercial strawberry-growing areas of the province. At the same time, we started a trial at the Substation to learn whether or not border crops or fallow had any effect on spread of both green petal and aster yellows viruses. Our results follow.

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[1]Contribution No. 160, Research Station, Canada Department of Agriculture, Fredericton, New Brunswick.

[2]Pathologist and Physiologist, respectively.

[3]Pathologist, Plant Protection Branch, New Brunswick Department of Agriculture.

Figure 1. Healthy strawberry plant on left and one infected with green petal virus on right.

Materials and Methods

The areas surveyed included fields in Carleton, York, Sunbury, Queens, and Kings Counties in central New Brunswick. The survey began each year in June, when most varieties were in full bloom; at which time symptoms of green petal virus are easily recognizable. During the 3 years, 1961-1963, only first- or second-year fruiting fields were examined. Incidence was rated by recording all clones of each variety that showed symptoms (Fig. 1), and by counting the infected plants in each clone.

The field trial on spread of green petal and aster yellows viruses into plots surrounded by different barriers began in early spring 1961 at McDonald's Corner Substation and continued until after harvest 1963. The trial consisted of 8 plots, each 10 rows wide. Four plots were planted with 3 rows each of the varieties Sparkle and Senator Dunlap and 4 half-rows each of China asters and carrots. The other 4 plots were each planted with alternate rows of the 2 strawberry varieties. Distance between rows was 5 feet and plants in a row were set one and one-half feet apart, except carrots, which were sown continuously. Ten strawberry plants or 5 China asters and continuous carrots made a row. Incidence of green petal infections was rated in the same manner used in the survey. Incidence of aster yellows was recorded by percentage of infected China asters and carrots. Each plot was completely surrounded by one of the barriers shown in Table 2. The strawberry mother plants, obtained from Norfolk Farms Limited, Vittoria, Ontario, were certified virus-free. The carrot seed was sown directly in the rows each year, but the China asters were started in a greenhouse and placed

the trial when the transplants were about 4 inches high. Good growth was ained each year in the test plants as well as in the barrier crops. The rye reached about 5 feet in height and the timothy 3 feet or more. Continuous cultivation of the fallow prevented any weeds from growing. No insecticides were applied to the trial nor were any leafhopper counts made.

Results and Discussion

The 3-year survey showed that green petal virus was not a serious economic problem in any of the fields examined (Table 1). Although only light infections were found in scattered areas, more diseased plants were found in 1961 than in either 1962 or 1963. Most infections were found in Sparkle and none in the varieties Paymaster, Redcoat and Senator Dunlap in any of the years.

Table 1. Numbers of strawberry clones found infected with green petal virus in New Brunswick fields from 1961 to 1963, inclusive.

Varieties	Number of fields surveyed	Approximate acreage surveyed	Number of infected clones[1]		
			1961	1962	1963
Catskill	3	3	0	2	1
Cavalier	4	5	1	0	0
Grenadier	2	4	1	1	1
Paymaster	1	1	0	0	0
Redcoat	5	8	0	0	0
Sparkle	5	8	7	2	0
Senator Dunlap	1	1	0	0	0
Mixed	1	1	1	0	0

[1]Each infected clone contained 1-7 diseased plants.

The effects of barriers on spread of both green petal and aster yellows viruses into small plots are shown in Table 2. None of the surrounding crops nor fallow prevented spread of aster yellows into China asters and carrots, but 30 feet of either grain or timothy appeared to almost completely stop spread of green petal into strawberries. The relatively high rate of spread of aster yellows virus is further evidence that this virus differs from that which causes green petal disease. Green petal infections were found only in 1962, and except for 1 clone in plot 3, all were in plots 2 and 6. The spread of this virus generally was not extensive but we were surprised to find none in plots 1 and 5. These latter plots contained China asters and carrots and plots 2 and 6 did not, which may have resulted in leafhoppers preferring to feed on these hosts rather than on the strawberries.

Barrier crops have been used to advantage in preventing spread of some aphid-borne viruses. For example, Broadbent (1) reduced incidence of cauliflower mosaic in cauliflower seedlings with barriers of barley and wheat. Similarly, Jenkinson (5) decreased spread of the same virus into broccoli seed beds with barrier crops of kale or barley when diseased plants were only 5 yards away. And, Simons (7) obtained significant decreases of spread of vein-banding virus into peppers by the use of sunflower barrier plants. Our results with one of two leafhopper-borne viruses show further promise of a similar means of control.

Table 2. Percentage spread of green petal and aster yellows viruses into small plots

Plot No.	Crop or Fallow Surrounding Plot	% Clones[1] with green petal virus						% Plants with aster yellows virus[2]			
		Sparkle			S. Dunlap			China asters		Carrots	
		1961	1962	1963	1961	1962	1963	1961	1962	1961	1962
1	15 ft. fallow and 15 ft. oats or rye[3]	0	0	0	0	0	0	55	50	5	10
2	Same as plot 1	0	8	0	0	4	0	-	-	-	-
3	30 ft. oats or rye	0	0	0	0	1.7	0	40	10	12	9
4	Same as plot 3	0	0	0	0	0	0	-	-	-	-
5	30 ft. fallow	0	0	0	0	0	0	90	10	7	14
6	Same as plot 5	0	8	0	0	4	0	-	-	-	-
7	30 ft. timothy and clover	0	0	0	0	0	0	40	35	5	8
8	Same as plot 7	0	0	0	0	0	0	-	-	-	-

[1] Plots 2, 4, 6, and 8 were each planted with 50 mother plants of Sparkle and 50 of S. Dunlap in 1961. The remaining plots contained 30 mother plants of each variety. One of 5 diseased plants in each infected clone..

[2] Plots 1, 3, 5, and 7 were planted each year with 20 China asters and 4 half-rows of carrots.

[3] Oats surrounded plot in 1961 and fall rye in 1962 and 1963.

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THE CONTROL OF STRAWBERRY FRUIT ROT IN COASTAL BRITISH COLUMBIA

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Abstract

In a 2-year study several fungicides, including organic and inorganic compounds along with calcium and boron additives, were tested for control of gray-mold fruit rot of strawberries. Folpet, captan and thiram proved the most effective of the materials tested. In the first fruiting year marketable yields were increased by 96 to 119 per cent over the untreated plants by a spray schedule consisting of four sprays at approximately 10-day intervals beginning at first bloom. A single application of captan at full bloom resulted in only 37 per cent increase in sound fruit. In the second fruiting year yields in sprayed plots were increased by only 48 to 62 per cent even though the spray schedule continued through harvest. It is suggested that this apparent decrease in response was the result of an improved sanitary condition in the plot area. The addition of calcium chloride to the captan schedule appeared to have no beneficial effect, while the addition of boron resulted in a significant reduction in yield. The holding quality of the fruit was improved by the field sprays of folpet, thiram and captan. Fruit quality was affected slightly by captan, which reduced the sugar and total acid content of the fruit. Boron sprays tended to increase ascorbic acid content while calcium chloride decreased it. Polyram 80 (zinc activated poly (ethylene thiuram disulphide)) reduced ascorbic acid content.

Introduction

Gray mold of strawberries, caused by Botrytis cinerea (Pers.) Fr., causes a serious fruit rot most years in coastal British Columbia. The current control program is based upon protecting the blossom parts through the bloom period, the recommendation being that strawberries should be sprayed or dusted with captan at least three times, starting when the first blossoms emerge. This recommendation was based primarily on work conducted at Oregon State University (7,9). There has been no local investigation within recent years on the control of this fungus and, therefore, no factual evidence with which to convince the grower that the currently recommended control measures are necessary or even effective. Thus an experiment was started in 1962 to obtain information on the effectiveness of several fungicides including organic and inorganic compounds, along with boron and calcium additives, for the control of strawberry fruit rot. This paper reports the results obtained in the 2-year study.

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Methods

Siletz strawberries were planted in a Lynden silt loam at the Small Fruits Substation, Abbotsford, British Columbia in 1961. The experiment was laid out in a randomized block design with four replications. Each plot consisted of a single 50-foot row. The plants were grown by the matted row system and good grower practices were followed in establishing the plantation. The planting was sprinkler-irrigated when necessary. In the spring of 1962 the entire planting was cleaned up by removing the old dead leaves and cultivating lightly. A lime-sulphur spray, 1½ gal/100 gal was applied March 22 to all plots. On May 7 the planting received an application of 6-30-15 fertilizer at 500 pounds per acre.

1962 Trial

Treatments were applied as outlined in Table 1. The nickel chloride 10 and 30 lb/a and the mixture of nickel chloride 10 pounds plus potassium chloride 200 lb/a were applied with the fertilizer on May 7.

Control of pre-harvest infection was determined by weighing all infected berries from each plot at each picking. The 1962 crop was picked on July 5, 12, 19 and 25. In addition to weighing the infected fruit, the weights of marketable and cull fruit were also recorded. The size index of sound berries from each plot was determined at each picking. The effect of treatment on post-harvest fruit rot was determined from a random sample of one pound of sound berries picked on July 5 from each plot in each replicate. The berries were stored in shipping crates in a common storage shed and the percentage of sound berries was determined at 24, 48, and 90 hours after harvest.

1963 Trial

Folpet, captan and thiram were again tested in 1963, but instead of discontinuing the sprays a week before harvest, as in 1962, the treatments were continued through the picking period. Polyram 80 (zinc activated poly (ethylene thiuram disulphide)) 2 lb/a and copper 53 at 6 lb/a were added to the test. Since the variety Siletz is a relatively soft-fruited variety, boron and calcium were tested with the object of increasing the firmness of the fruit. Boron was applied as a fertilizer (Tronobar (14% boron) 35 lb/a) and as a spray (Solubor (20.5% boron) 2.5 and 25 lb/a) on May 3. Calcium was applied as a 0.4% calcium chloride spray on May 15, 24, 27 and 31 during bloom. Boron and calcium sprays were also combined with a full captan schedule. The complete spray schedules are listed in Table 3.

The 1963 crop was picked on June 17, 25, July 4 and 10. Data were recorded as in 1962 with the refinement that the effect of treatment on the post-harvest fruit rot was determined from the complete crop and not just a random sample of one picking. The percentage of sound berries was determined at 24, 48 and 72 hours after harvest.

Fruit Quality

Samples of fruit were quick-frozen and later analyzed in the laboratory for acidity, sugar and ascorbic acid as a measure of quality.

Results and Discussion

Of the 1962 treatments the folpet and thiram spray schedules gave the best control of gray mold as reflected in the increase in yield of sound fruit (Table 1). However, the results obtained from the lime-sulphur plus captan and the captan schedules were not significantly different from those obtained by the folpet and thiram schedules. The increased marketable yield for these four spray schedules ranged from 96 to 119 per cent over the unsprayed plots. This result is in agreement with Horn (3) who reported no significant difference between thiram, folpet and captan in rot control.

The single application of captan at full bloom resulted in only 37 per cent increase of sound fruit over the unsprayed plot. The additional three captan sprays resulted in a further 59 per cent increase in sound fruit. Thus, evidence is provided supporting the desirability of the full spray schedule. The fungicides dichlone, Dyrene and N-3684 (di-n-butyltin dimesylate) did not appear to be of sufficient efficacy to be considered further. The inorganic salts were but little better than no treatment.

Fruit size was affected by treatment; the trend was for fruit to be larger from plots treated with the more efficient fungicides. This result is in accord with findings of Powell (6) who reported that strawberry plants had benefitted nutritionally from captan and that fruit size was increased.

The data on post-harvest control of fruit rot show that there was little effect of treatment in the first 24 hours of storage (Table 2). The value of the folpet, thiram and captan spray schedules was, however, evident after 48 hours storage. The beneficial effect was still evident after 90 hours storage. Powelson (7) found that three pre-harvest applications of captan reduced rot incidence in the field, and also the amount of latent infection of marketable fruit. Powell (6) also reported that captan delayed the development of post-harvest rot.

The 1963 results again showed that folpet, captan and thiram were the most effective fungicides for fruit rot control (Table 3). There was not as great a difference in marketable fruit yields between treated and untreated plots as in 1962. It is well known that the severity of gray mold infection varies greatly with seasonal conditions. However, the weather pattern was not too different for the two years of this experiment and sprinkler irrigation was applied only as required. It is suggested, therefore, that much of the reduction in fruit rot in the second year can be attributed to an improved sanitary condition in the plot area as a result of the previous year's spray applications and the fact that all diseased berries were removed at each picking. It would be expected that the source of inoculum would be reduced significantly by such practices. Miller and Waggoner (5) on studying the dispersal of spores of Botrytis cinerea among strawberries reported that most spores were caught in periods of high humidity, regardless of rain or time of day. Only occasional spores were caught under other conditions. They suggested that most infections by B. cinerea originate from nearby primary inoculum and that microclimate afforded by dense strawberry foliage is more important than environmental conditions above the plants in determining incidence of

Table 1. Influence of various treatments on pre-harvest fruit rot of Siletz strawberries - 1962

Treatment (active ingredient) (sprays in 100 gal. water)	Dates of Application*	Rotted fruit	Sound fruit (mkt. yield)	Increase over unsprayed	Size index
		lb/plot	lb/plot	%	gm/25 fruit
Folpet 2½ lb/a	May 16,29, June 11,19	13.6 a	53.9 a	119	210 a
Thiram 2 lb/a	May 16,29, June 11,19	19.0 b	53.9 a	119	198 ab
Lime-sulphur 1½ gal + captan 1½ lb/a	May 16 May 29, June 11,19	22.0 bc	50.5 ab	104	197 ab
Captan 1½ lb/a	May 16,29, June 11,19	21.0 bc	48.1 ab	96	206 a
Dichlone ½ lb/a	May 7,29, June 11,19 July 10	19.9 bc	45.9 b	85	193 abc
Dyrene 1½ lb/a	May 16,29, June 11,19	20.9 bc	43.9 b	78	195 abc
Captan 1½ lb/a	May 29	28.3 e	33.8 c	37	192 abc
Nickel chloride 30 lb/a	May 7	29.6 e	29.6 cd	19	181 bc
N-3684 (di-n-butyltin-dimesylate) 2 lb/a	May 16,29, June 11,19	23.6 cd	27.4 cd	11	186 bc
Nickel sulphate 2 lb/a (spray)	May 7,29, June 11,19	24.0 cd	26.6 d	7	179 bc
Nickel chloride 10 lb + potassium chloride 200 lb/a	May 7	27.5 de	26.2 d	7	182 bc
Nickel chloride 10 lb/a	May 7	26.9 de	25.8 d	4	181 bc
Unsprayed	-	28.3 e	24.3 d	0	177 c
Mean		23.43	37.68		190.6
S.E. Mean		1.26	2.23		5.75

Means not followed by the same letter are significantly different at the 5% level. (Duncan's Multiple Range test)

*May 16 - first floom, May 29 - full bloom, June 11 - after full bloom, June 19 - last spray before harvest. (first fruit ripe June 27)

Table 2. Influence of various pre-harvest treatment schedules on the post-harvest fruit rot of Siletz strawberries - 1962

Treatment (active ingredient) (sprays in 100 gal. water)	Dates of Application*	Percent sound fruit hours after picking 24	48	90
Folpet 2½ lb/a	May 16,29, June 11,19	88	70	9
Thiram 2 lb/a	May 16,29, June 11,19	91	62	11
Lime-sulphur 1½ gal/100 gal. + captan 1½ lb/a	May 16 May 29, June 11,19	79	67	8
Captan 1½ lb/a	May 16,29, June 11,19	74	72	8
Dichlone ½ lb/a	May 7,29, June 11,19, July 10	81	42	2
Dyrene 1½ lb/a	May 16,29, June 11,19	90	63	2
Captan 1½ lb/a	May 29	-	-	-
Nickel chloride 30 lb/a	May 7	78	29	2
N-3684 (di-n-butyltin dimesylate) 2 lb/a	May 16,29, June 11,19	83	24	1
Nickel sulphate 2 lb/a (spray)	May 7,29, June 11,19	74	27	0
Nickel chloride 10 lb + potassium chloride 200 lb/a	May 7	75	25	0
Nickel chloride 10 lb/a	May 7	80	25	0
Unsprayed	-	74	15	0

*See footnote Table 1

Table 3. Influence of various treatment schedules of pre-harvest fruit rot of Siletz strawberries - 1963

Treatment (active ingredient) (Sprays in 100 gal. water)	Dates of Application*	Rotted fruit lb./plot	Sound Fruit (Mkt. Yield) lb./plot	Percent Increase over unsprayed	Size Index gm/25 fruit
Folpet 2½ lb/a	May 15,24,31, June 7,14,21,29, July 5	10.8 a	67.4 a	62	256 a
Captan 1½ lb/a	May 15,24,31, June 7,14,21,29, July 5	9.9 a	62.9 a	51	252 a
Thiram 1½ lb/a	May 15,24,31, June 7,14,21,29, July 5	8.8 a	61.6 ab	48	261 a
Calcium chloride 0.4% + captan 1½ lb/a	Calcium chloride May 15,24,27,31, captan as above	10.8 a	60.5 ab	45	244 a
Boron spray ½ lb + captan 1½ lb/a	Boron May 3, captan as above	9.5 a	54.3 bc	30	253 a
Boron spray 5 lb/a + captan 1½ lb/a	Boron May 3, captan as above	8.7 a	50.4 cd	21	247 a
Polyram 80W 2 lb/a	May 15,24,31, June 7,14	19.8 bc	45.3 de	9	238 a
Copper 53 6 lb/a	May 15,24,31, June 7,14,21,29, July 5	18.5 b	42.2 ef	1	238 a
Unsprayed	---	22.9 bc	41.7 ef	0	238 a
Calcium chloride 0.4%	May 15,24,27,31	20.7 bc	40.6 ef	0	241 a
Boron fertilizer 5 lb/a	May 3	23.1 c	38.2 ef	0	243 a
Boron spray 5 lb/a	May 3	23.0 bc	35.6 f	0	239 a
Mean		15.55	50.6	-	245.7
S.E. Mean		1.90	2.69		6.44

Means not followed by the same letter are significantly different at the 5% level. (Duncan's Multiple Range test).

*May 15 first bloom, May 24-31 full bloom, June 17 first picking.

Table 4. Influence of various pre-harvest treatment schedules on the post-harvest fruit rot of Siletz strawberries - 1963

Treatment (active ingredient) (sprays in 100 gal. water)	Dates of Application*	Percent sound fruit hours after picking (means of 4 harvests)		
		24	48	72
Folpet 2½ lb/a	May 15,24,31, June 7, 14,21,29, July 5	97	77	48
Captan 1½ lb/a	May 15,24,31, June 7,14,21,29, July 5	96	67	45
Thiram 1½ lb/a	May 15,24,31, June 7,14,21,29, July 5	95	87	54
Calcium chloride 0.4% + captan 1½ lb/a	Calcium chloride May 15,24,27,31; captan as above	96	77	48
Boron spray ½ lb/a + captan 1½ lb/a	Boron May 3, captan as above	96	79	45
Boron spray 5 lb + captan 1½ lb/a	Boron May 3, captan as above	97	76	42
Polyram 80W 2 lb/a	May 15,24,31, June 7,14	91	56	15
Copper 53 6 lb/a	May 15,24,31, June 7,14,21,29, July 5	92	37	8
Unsprayed	—	91	52	10
Calcium chloride 0.4%	May 15,24,27,31	92	50	13
Boron fertilizer 5 lb/a	May 3	92	45	8
Boron spray 5 lb/a	May 3	93	47	6

*May 15 - first bloom, May 24 - 31 full bloom, first picking June 17.

Table 5. Influence of various treatment schedules on total acid, sugar and ascorbic acid content of Siletz strawberries - 1963

Treatment/a*	Total Acidity (% citric)	Treatment/a*	Sugar (%)	Treatment/a*	Ascorbic acid (mg./100 gm)
Polyram 80W 2 lb	.95 a	Boron spray 5 lb	8.31 a	Boron spray 5 lb	35.63 a
Copper 53 6 lb	.95 a	Calcium chloride 0.4%	8.27 ab	Unsprayed	34.12 ab
Thiram 1½ lb	.94 a	Polyram 80W 2 lb	8.20 ab	Boron spray 5 lb + captan	34.02 ab
Boron fertilizer 5 lb	.94 a	Copper 53 6 lb	8.20 ab	Boron spray ½ lb + captan	33.96 ab
Calcium chloride 0.4%	.94 a	Thiram 1½ lb	8.18 ab	Thiram 1½ lb	33.27 abc
Folpet 1½ lb	.93 ab	Boron fertilizer 5 lb	8.18 ab	Boron fertilizer 5 lb	32.95 abc
Boron spray 5 lb	.93 ab	Unsprayed	8.13 ab	Folpet 1½ lb	31.85 bcd
Calcium chloride + captan	.93 ab	Folpet 1½ lb	8.12 ab	Copper 53 6 lb	31.72 bcd
Unsprayed	.92 ab	Boron spray 5 lb + captan	8.09 ab	Captan 1½ lb	31.42 bcd
Captan 1½ lb	.92 ab	Calcium chloride + captan	8.05 ab	Calcium chloride .04%	31.19 bcd
Boron spray ½ lb + captan	.90 b	Captan 1½ lb	7.99 ab	Polyram 80W 2 lb	30.98 cd
Boron spray 5 lb + captan	.90 b	Boron spray ½ lb + captan	7.89 b	Calcium chloride + captan	29.84 d
Mean	.93		8.14		32.58
S.E. Mean	.01		0.12		0.88

Means not followed by the same letter are significantly different at the 5% level (Duncan's Multiple Range Test).

*Treatment schedules as Tables 3 and 4

gray mold. Jarvis (4) states that the primary aim in control must be the eradication of the over-wintering stages of the pathogen and the reduction in potential sites for saprophytic colonization at the time of flowering and fruit development. He further states that good plantation hygiene is therefore of great importance, and must include attention to many details such as weed control and the removal of strawberry debris. There would appear to be no advantage to lengthening the spray schedule by spraying during harvest. Yields of sound fruit from plots treated with Polyram 80 (zinc activated poly (ethylene thiuram disulphide)) at 2 lb/a were not different from the untreated plots. Stall (8) found Botrytis more prevalent on tomato plants sprayed with zinc sulfate or nabam-zinc sulfate than on unsprayed plants. Cox and Winfree (1) found the same effect with gray mold on strawberries and suggested that an excess of zinc in the plant tissue might increase susceptibility to the disease.

The addition of calcium chloride to the captan schedule appeared to have no beneficial effect, while the addition of boron resulted in a yield reduction. These treatments had no apparent effect upon fruit firmness. Eaves and Leefe (2) reported that highly significant increases in firmness of Sparkle strawberries were associated with calcium sprays.

Fruit quality was affected to some extent by the treatments. Captan tended to reduce the sugar and acid content of the fruit (Table 5). Boron sprays tended to increase ascorbic acid content while calcium chloride caused a reduction. Of the fungicides only Polyram caused a significant reduction in the ascorbic acid content. The holding quality of the fruit was proved again in 1963 by the field sprays (Table 4).

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THE CONTROL OF STORAGE ROTS OF RASPBERRIES WITH SULFUR DIOXIDE[1]

C. L. Lockhart and D. L. Craig

Abstract

Fumigating red raspberries with 0.5 per cent SO_2 for 20 minutes controlled storage rots for three days without causing off flavors. Fumigated raspberries were lighter in color and more attractive than untreated ones. The varieties Trent, Canby, Willamette and Carnival were firmer but Viking, Malling Promise and Early Red were usually softer following fumigation. One per cent SO_2 caused excessive bleaching and off flavors.

Introduction

The shelf life of red raspberries is very short and the consumer must receive them within 24 hours after picking to obtain good quality berries free of rots. However, according to Wright et al (4) shelf life can be prolonged by cold storage. They were able to keep raspberries 5 to 7 days at 31 to 32°F and at 85 to 90 per cent relative humidity. In Nova Scotia the low annual production (45,000 quarts) is spread among many growers in widely separated areas and it is usually not feasible to maintain local cold storage facilities. Obviously, other less expensive means of controlling rots are needed. Recently, Capellini et al (1) in New Jersey found that rots of Red Latham raspberries were controlled with SO_2 treatments. Such treatments are inexpensive, safe and present no residue problem.

Materials and Methods

In 1963 both field run and selected raspberries were fumigated immediately after picking with SO_2 in a 36 cu. ft. wooden chamber equipped with an air circulating fan, described by Sanford (3). Varieties treated were: Trent, Willamette, Carnival, Viking, Early Red, Canby and Malling Promise. The raspberries had received a dormant Bordeaux 5-10-100 spray on May 6 and a spray of ferbam at 2 lb. per 100 gallons on June 17, 1963. Pint boxes of field run berries or boxes containing 50 selected berries were placed on the floor of the fumigation chamber. Prior to each fumigation, the temperature and relative humidity of the treatment chamber was recorded. A weighed amount of SO_2 was released into the fumigation chamber through a flowmeter to give a concentration of 0.5 or 1.0 per cent. The berries were exposed to SO_2 for 20 minutes with the circulation fan operating in the chamber and, before opening the chamber, a sample of its atmosphere was withdrawn and analysed by Ruck's (2) method to verify the SO_2 concentration. Treated and untreated raspberries were stored in crates at 32, 52 or 72°F

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[1]Contribution No. 1168 from the Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

for two and three days and then examined for appearance and rots. Isolations from unknown rots and discolored drupelets were made on potato dextrose agar. Four boxes of berries were usually used for each treatment but for some varieties it was necessary to treat a lesser number due to lack of fruit.

Results and Discussion

Fumigation with 0.5 per cent SO_2 for 20 minutes gave good control of storage rots of carefully selected berries stored at 72°F for three days and of field run berries stored for two days at 72°F. The latter showed considerable breakdown when stored for three days (Table 1 and 2). Lesser amounts of raspberries of other varieties gave similar results. Selected raspberries which had and had not been treated with SO_2 showed no evidence of breakdown after three days storage at 32°F. The importance of selecting good berries for storage or shipment to market is obvious.

Raspberries fumigated with 0.5 per cent SO_2 had no off flavor and were lighter in color and more attractive than untreated ones. Treated raspberries of the varieties Trent, Canby, Willamette and Carnival were firmer, and Viking, Malling Promise and Early Red were usually softer than the controls. Capellini *et al* (1) found softening and bleaching of Red Latham raspberries treated with 0.5 per cent SO_2. Both treated and untreated Early Red berries crumbled quite readily. All varieties showed considerable bleaching and off flavor when fumigated with 1.0 per cent SO_2. Tests were carried out at temperatures ranging from 70 to 85°F and at relative humidities ranging from 53 to 69 per cent, but these factors had no noticeable influence on the results.

Table 1. Per cent rots on field run Trent raspberries after exposure to 0.5 per cent SO_2 for 20 minutes (Mean values for 4 boxes).

Days in storage	Temp. °F	Total rots		Botrytis		Others ***	
		T*	C**	T	C	T	C
2	32	1.9	8.6	1.9	7.3	0	1.3
2	52	4.0	16.4	2.6	12.5	1.4	3.9
2	72	23.6	88.3	23.3	88.3	0.3	0
3	32	18.3	31.5	17.1	30.1	1.2	1.4
3	52	23.5	25.4	23.3	24.2	0.2	1.2
3	72	89.9	97.9	78.0	93.4	12.0	4.5

* Treated with SO_2

** Untreated

*** Includes *Cladosporium* sp, *Penicillium* spp and *Rhizopus* sp.

Table 2. Per cent rots on selected raspberries stored at 72°F after exposure to 0.5 per cent SO_2 for 20 minutes (Mean values for 4 boxes).

Variety	Days in storage	Total rots		Botrytis		Penicillium		Cladosporium		Rhizopus	
		T*	C**	T	C	T	C	T	C	T	C
Trent	2	2.5	54.0	0.5	2.0	0	5.5	2.0	5.5	0	41.0
Willamette	2	1.5	59.0	0.5	2.5	0	1.0	1.0	15.5	0	30.0
Carnival	2	3.5	38.5	0	2.5	1.0	1.0	2.0	28.0	0.5	7.0
Carnival	3	0	22.5	0	5.0	0	5.5	0	11.5	0	0.5
Willamette	3	3.5	39.5	0.5	4.5	0.5	5.5	2.5	27.0	0	2.5
Viking	3	0	31.0	0	5.0	0	6.5	0	17.0	0	2.5

* Treated with SO_2

** Untreated

Table 3. Occurrence of microorganisms in per cent from discolored drupelets of raspberries

Isolates	Varieties			
	Trent	Early Red	Carnival	Willamette
Botrytis cinerea	0	16.6	14.2	39.3
Cladosporium herbarum	4.2	0	25.0	11.6
Penicillium spp	0	4.2	0	4.5
Rhizopus sp	0	0	25.0	3.6
Undetermined fungi	8.2	4.1	0	0
Bacteria	0	0	3.5	10.7
Yeast	4.2	16.6	0	0.9
Sterile	83.4	58.5	32.3	29.4

Discolored drupelets, light red to whitish in color and often slightly shrunken, were found on both untreated and treated raspberries. In a first series of isolations these were all sterile. In later isolations from untreated berries, a high percentage of Trent and Early Red were sterile but a large number of discolored drupelets of Carnival and Willamette contained organisms (Table 3). Fumigation with SO_2 or storage temperature had little effect on the incidence of discolored drupelets. The drupelet injury was thought to be caused by an insect puncture that occurred prior to picking. The higher incidence of microorganisms isolated from Carnival and Willamette may have been due to periods of wet weather prior to the picking date of these varieties.

It is concluded that first grade red raspberries fumigated 20 minutes with 0.5 per cent SO_2 can be safely handled for three days at 72°F without a significant loss of berries from decay.

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CANADA AGRICULTURE RESEARCH STATION,
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CONTROL OF GLOEOSPORIUM ALBUM ROT AND STORAGE SCAB OF APPLES WITH ORCHARD FUNGICIDES[1]

R. G. Ross

Abstract

A regular orchard spray program of Delan gave excellent control of a storage rot of apples caused by Gloeosporium album and storage scab of apples caused by Venturia inaequalis. Dodine was effective for storage scab but not for storage rot. Folpet usually gave better control of rot caused by G. album than did captan but was not consistently effective against storage scab.

Introduction

In 1960, Ross and Lockhart (4) reported that excellent control of a storage rot of apples, caused by Gloeosporium album Osterw., was obtained with a regular orchard spray program of captan followed by 2 late cover sprays of captan or a mixture of captan and zineb. Late cover sprays of zineb or a mixture of captan and zineb controlled storage scab caused by Venturia inaequalis (Cke.) Wint. Recently workers in England (1,2,3) have found that dormant applications of mercury fungicides give some control of Gloeosporium spp. A fungicide is needed that would control both apple scab and Gloeosporium storage rot without applying extra cover sprays.

Each year apples from apple scab fungicide plots are placed in storage to assess the effect of sprays on the incidence of storage rots and storage scab. The results for 1961 and 1962 are given in this paper.

Materials and Methods

The orchard used in previous work (4) was divided into 3 blocks with the treatments randomized in each block. The sprays were applied dilute with a hand gun and the trees were sprayed to run-off. In 1961, 4 pre-cover (May 15, 23-24, June 1, 12-13) and 3 cover (June 21-22, July 3, 17-18) sprays were applied except in a dodine treatment which received 6 sprays at 12-day intervals ending on July 10. In 1962, 5 pre-cover (May 7, 16, 24-25, June 7, 14) and 3 cover (June 26, July 5, 18) sprays were applied with the 12-day dodine treatment consisting of 7 sprays ending on July 20.

The fungicides used were:

Captan (Captan 50-W), N-(trichloromethylthio) - 4 - cyclohexene - 1, 2 - dicarboximide, 50% (Stauffer Chemical Co., New York, N.Y.)

Dodine (Cyprex), n-dodecyl guanidine acetate, 65% (Cyanamid of Canada, Ltd., Toronto, Ont.)

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[1]Contribution No. 1171 from the Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

Folpet (Phaltan 50-W), N-(trichloromethylthio) phthalimide, 50% (California Chemical Co., Richmond, California.)

Glyodin (Crag Fruit Fungicide 341), 2-heptadecyl -2-imidazoline acetate, 34% (Green Cross Insecticides, Montreal, Que.)

Delan, 2,3-dinitrilo 1, 4 -dithioanthroquinone, 75% (Green Cross Insecticides, Montreal, Que.)

One bushel of unblemished apples of each of the varieties McIntosh and Cortland from each plot were stored at 32°F. The McIntosh were stored for about 6 months and the Cortlands for about 5½ months before examining for fruit showing Gloeosporium rot and storage scab. In calculating the per cent fruit with rot or scab all affected fruits were included irrespective of the number of infections. Identification of the causal organisms of nonsporulating lesions was made from isolations on PDA.

Results and Discussion

The results for 1961 are in Table 1 and those for 1962 are in Table 2. There was considerable variation between different replicates of some treatments so rather large differences are required for significance. This suggests that larger samples would be required to give more uniform data. Nevertheless, Delan gave the best and most consistent control of Gloeosporium fruit rot. In 1962, varying the rate of application of Delan made little difference in the control obtained. Dodine did not consistently give good control of rots and the results with folpet varied although it was usually more effective than captan. A regular captan schedule, without extra cover sprays, is usually regarded as being somewhat effective (4) against Gloeosporium storage rot of apples but in these tests it was always less effective than Delan.

Storage scab was much more severe in 1962 than in 1961. In both years Delan gave excellent control. In general, dodine, particularly at the 3/4-1/2 lb. rate, also gave good control; this agrees with previous results (4). Folpet was effective against storage scab in 1961 but not in 1962. Captan did not give good control of storage scab in either year.

Delan was the only fungicide that consistently gave good control fo both storage scab and Gloeosporium rot. If it becomes accepted as an apple fungicide, extra cover sprays should not be necessary for the control of these diseases.

Table 1. Effect of orchard fungicides on G. album rot and storage scab of apples - 1961

Fungicide and rate per 100 gallons		Per cent rot and scab			
		McIntosh		Cortland	
Pre-cover	Cover	Rot	Scab	Rot	Scab
Dodine, 3/4 lb.*	Dodine, ½ lb.*	6.1 ab	2.6 a	8.9 c	0.0
Dodine, 3/4 lb.	Dodine, ½ lb.	12.0 b	0.3 a	4.3 abc	0.1
Dodine, ½ lb.	Dodine, ¼ lb.	6.6 ab	13.2 ab	3.4 ab	2.9
Dodine, ¼ lb. + glyodin, 1 pint	Dodine, ¼ lb. + glyodin, 1 pint	7.1 ab	6.9 ab	8.5 c	0.6
Dodine, ¼ lb. + captan, 1 lb.	Dodine, ¼ lb. + captan, 3/4 lb.	3.9 ab	0.6 a	6.6 bc	0.2
Folpet, 2 lb.	Folpet 1½ lb.	1.6 a	1.4 a	5.7 bc	0.6
Delan, 2 lb.	Delan, 1½ lb.	1.2 a	0.0 a	2.4 a	0.0
Captan, 2 lb.	Captan, 1½ lb.	5.6 ab	23.6 b	5.1 bc	2.6

* Sprayed every 12 days.

The small letters in Tables 1 and 2 indicate Duncans' multiple range groupings, treatments with the same letter do not differ significantly at the 5% level. There are no significant differences in columns without small letters.

Table 2. Effect of orchard fungicides on G. album rot and storage scab of apples 1962.

Fungicide and rate per 100 gallons		Per cent rot and scab			
		McIntosh		Cortland	
Pre-cover	Cover	Rot	Scab	Rot	Scab
Dodine, 3/4 lb.*	Dodine, ½ lb.*	12.3	0.6 a	6.8	0.8 a
Dodine, 3/4 lb.	Dodine, ½ lb.	8.6	8.1 abc	4.7	1.7 a
Folpet, 2 lb.	Folpet, 1½ lb.	4.9	46.4 cd	3.4	34.6 b
Folpet, 1 lb.	Folpet, 1 lb.	5.7	40.2 bc	1.9	36.2 b
Delan, 2 lb.	Delan, 1½ lb.	2.0	0.0 a	2.0	0.4 a
Delan, 1 lb.	Delan, 3/4 lb.	1.3	1.1 ab	0.8	0.0 a
Delan, ½ lb.	Delan, ½ lb.	2.6	0.0 a	1.5	0.0 a
Captan, 2 lb.	Captan, 1½ lb.	8.1	46.6 cd	7.0	27.1 b

* Sprayed every 12 days.

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BIPOLARIS SOROKINIANA ON SNAP BEANS IN NEW BRUNSWICK[1]

K. M. Graham[2], R. A. Shoemaker[3], and S. R. Colpitts[4]

Abstract

A leaf and pod spot caused by Bipolaris sorokiniana (Sacc. in Sorok.) Shoemaker was found in 400 acres of snap beans near East Florenceville, New Brunswick, during 1963. An isolate of the pathogen caused crown rot symptoms on certain varieties of barley and oats, crops which are grown frequently in rotation with potatoes and beans. The source of inoculum for the outbreak was probably adjacent cereals or cereal stubble. The disease is not considered important at the present time because pod symptoms disappear during the blanching process.

Introduction

In September, 1963, snap bean plantations encompassing some 400 acres in Carleton County, New Brunswick, showed extensive spotting of the pods. The lesions (Fig. 1) were lenticular, crateriform, brown to black in color, and varied in size from 1-5 mm. They apparently began as black pinpoints surrounded by a watersoaked halo. Inspectors mistook them for symptoms of bacterial blight or anthracnose, although ooze and spores were not present. Leaf lesions were evident in July as inconspicuous reddish brown pinpoints which enlarged in August and September to gray, circular spots with black margins. Sometimes the centres dropped out to produce a shothole effect. Stem lesions were similar to those in pods.

The fungus isolated from the pod lesions matched stock cultures of Bipolaris sorokiniana (Sacc. in Sorok.) Shoemaker (=Helminthosporium sativum Pamm., King & Bakke), the conidial state of Cochliobolus sativus (Ito & Kurib. in Kurib.) Drechs. in Dastur.

Although processors feared that the disease would cause considerable economic loss, it proved to be of little importance. Pod symptoms disappeared during the blanching process which precedes freezing of the product. A brief investigation of the disease was undertaken in anticipation of possible future complications.

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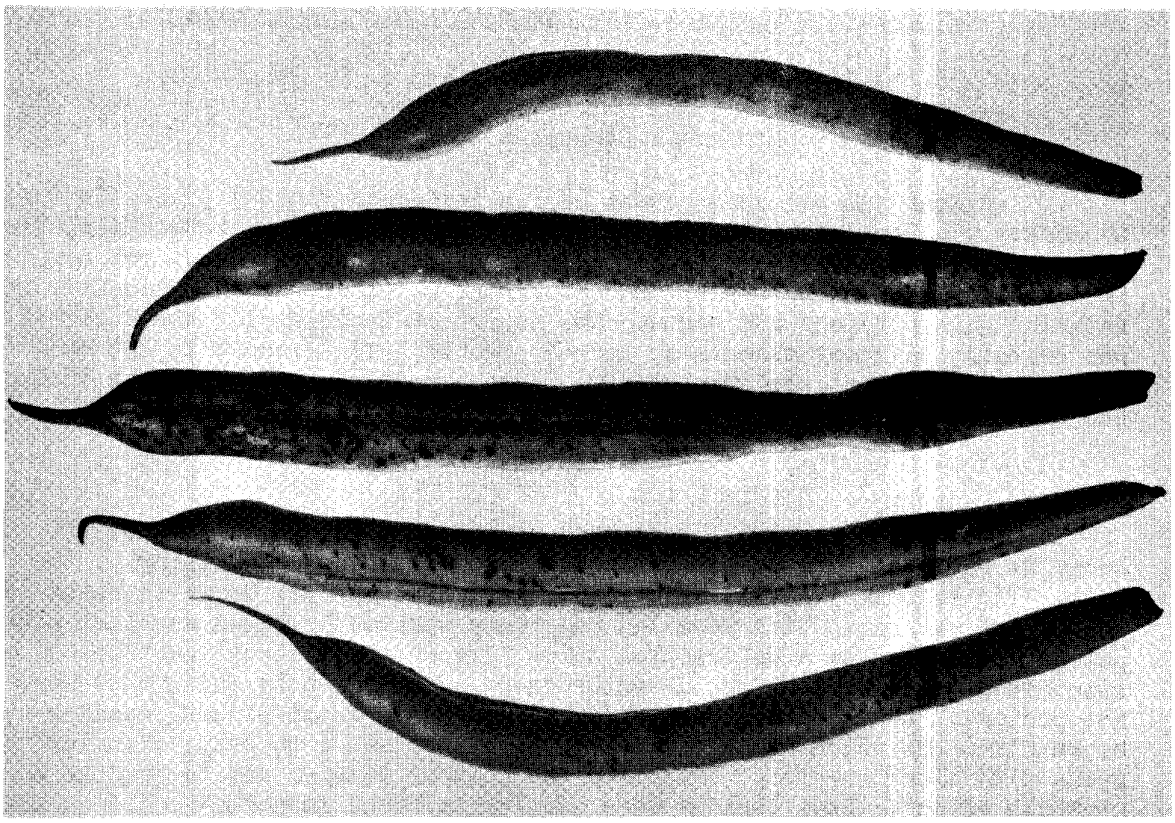

Figure 1. Symptoms of pod spotting due to *Bipolaris sorokiniana* on Tendergreen snap beans.

Methods

The fungus was propagated on 2% PDA and on corn meal sand. The latter was prepared by adding water to the medium, composed of 10% corn mean and 90% washed white sand, until the mixture was crumbly but not wet. It was then dispensed into 250 ml wide-mouth Erlenmeyer flasks at the rate of 200 cc per flask. The medium was then sterilized in an autoclave for 1 hour at 15 lbs pressure. The fungus was incubated for two weeks in the flasks and then added to sterilized greenhouse compost soil at the rate of 25 cc per pound of soil.

In an attempt to reproduce symptoms of the disease, three bean varieties, Tendercrop, Kinghorn, and Bush Blue Lake were inoculated with a spore suspension of the isolated fungus and put in a moist chamber for 24 hours at 60-65°F. A few representative varieties of oats, barley, wheat and rye were also inoculated. 100 seeds of each test variety were planted in separate pots of infested soil, with similar plantings of 100 seeds in noninfested sterilized soil to serve as controls. One month after planting the seedlings were dug and examined for crown rot.

Results

Symptoms on the green-podded snap bean varieties Tendercrop and Bush Blue Lake were similar to those on field-collected samples of the variety Tendercrop. No symptoms could be detected on the yellow-podded variety Kinghorn, and it is presumed to be resistant.

The barley varieties Charlottetown 80 and Parkland, and the oat varieties Abegweit, Fundy, and Russell all showed symptoms of crown rot, while Sangaste rye and Selkirk wheat were free of disease (Table 1). Victoria oats was not attacked. On the basis of these pathogenicity tests the identification of the pathogen as Bipolaris sorokiniana is confirmed.

Table 1. Pathogenicity of Bipolaris sorokiniana on certain bean and cereal varieties.

Crop	Variety	Infection
Beans	Tendercrop	+
	Bush Blue Lake	+
	Kinghorn	-
Barley	Charlottetown 80	+
	Herta	-
	Parkland	+
Oats	Abegweit	+
	Ajax	-
	Fundy	+
	Garry	-
	Russell	+
	Victoria	-
Rye	Sangaste	-
Wheat	Selkirk	-

Discussion

The New Brunswick disease is considered quite distinct from a leaf and pod spot of snap beans described by Winstead and Hebert (8) in Pender County, North Carolina. The pathogen in that case was Bipolaris victoriae (Meehan and Murphy) Shoemaker (= Helminthosporium victoriae Meehan and Murphy), and the source of inoculum was traced to an adjacent field of Victorgrain oats heavily infested with Victoria blight. Furthermore, leaf lesions of the North Carolina disease were small, narrow, black streaks on veins.

Spurr and Kiesling (7) were able to produce symptoms similar to the local disease on beans with Bipolaris sorokiniana. Their investigation was prompted by finding the pathogen on wheat which had followed beans in rotation. They were also able to produce lesions and sporulation on cowpeas, cucumbers, pumpkins, peas, sunflowers, and tomatoes.

Renfro (6), working in Minnesota, found that isolates of Bipolaris sorokiniana from stems of alfalfa and yellow sweet clover were pathogenic to oats, wheat, and barley, and he suggested that the fungus was part of the black stem disease complex of forage legumes in the north-central area of the U.S.A.

The local disease closely resembled, in some respects, a leaf spot of snap beans (haricots) described by J. A. Meyer (2) from Westmeerbek, Belgium. Spots on the leaves were reddish brown, with the centres becoming light gray with gray-brown borders as they enlarged. Eventually the centres fell out and left the leaf perforated. The fungus sporulating on the gray central portion was determined as Drechslera siccans (Drechs.) Shoemakes, and the author linked its occurrence on beans with the presence of ryegrass, (Lolium sp.), another susceptible host, in an adjacent pasture.

Olive, Bain, and Lefebvre (3) found a leaf spot of cowpeas and soybeans at locations in Louisiana, North Carolina, and South Carolina. They attributed the disease to a new species, Helminthosporium vignae Olive, Bain, and Lefebvre. However, Jones (1) considered that this fungus was really Corynespora cassiicola (Burk. et Curt.) Wei, a cotton pathogen that had been present in the Mississippi Delta and probably in other cotton-growing areas of the southern U.S.A. for 25 years previous to 1961. He demonstrated that his isolates could attack both cowpea and soybean and presumed the source of inoculum to be cotton.

Evidence from the literature indicates that legumes, particularly beans, are secondary or "catch-all" hosts for various species of Bipolaris, Drechslera, and Corynespora which propagate on cereals and occasionally on dicotyledonous plants. In the bean-growing area of New Brunswick, the rotation commonly practised is grass-oats-potatoes-beans. Fields of oat and barley varieties susceptible to Bipolaris sorokiniana were found interspersed throughout the area, often close to bean fields. Orlob and Bradley (5) in their 1960 survey observed Bipolaris sorokiniana on 6-rowed barley but gave no locality. Although cursory observations in September, 1963 did not indicate that Bipolaris leaf spot was prevalent on cereals, inoculum could have been generated from crop debris or stubble left from previous crops.

Orlob and Bradley (5) also found Drechslera avenacea (Curtis ex Cooke) Shoemaker on oats and D. teres (Sacc.) Shoemaker, on barley. Orlob (4) also noted D. tritici-repentis (Died.) Shoemaker on quackgrass, D. bromi (Died.) Shoemaker, on smooth bromegrass, D. dictyoides (Drechs.) Shoemaker, on fescue, and D. poae (Baudys) Shoemaker (=D. vagans (Drechs.) Shoemaker), on bluegrass. All of these grasses can be found along roadsides and in headlands and pastures in the bean-growing area and the species of Drechslera found on them could be implicated in the outbreak of leaf spot on beans. Checks on their pathogenicity might be worthwhile.

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BARLEY YIELD REDUCTIONS ATTRIBUTED TO NET BLOTCH INFECTION

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There are few reports in the literature on the effects of leaf diseases of barley, although it is generally accepted that such diseases reduce yield and quality. A comparison of yields from experimental plots protected by fungicides with those from untreated plots has shown that up to 20% reduction occurred when the plants were infected with one or more leaf diseases (1). In 1963 an epidemic of net blotch, caused by Drechslera teres (Sacc.) Shoem., occurred in Manitoba and provided an opportunity to assess, over a wide area, yield losses attributable to this disease.

Heavy infections of net blotch were observed during a survey in August, 1963, in an area extending from Winnipeg, Manitoba, northwest to Melfort, Saskatchewan. Earlier in the year observations in breeding plots at Winnipeg indicated that the variety Betzes was extremely susceptible to this disease while Herta was relatively resistant. The two varieties could be readily distinguished in experimental plots at Melfort and elsewhere on the basis of their net blotch reaction. In variety trials in Manitoba in 1960, 1961, and 1962, when only trace to moderate infection of net blotch was recorded, Betzes generally outyielded Herta. Data from these trials were compared with that obtained in 1963, an epidemic year, to determine what effect that severe infection of net blotch had on the yields of the two varieties. The data were analyzed by the "t" test for paired comparisons.

Betzes yielded less than Herta at all stations in 1963 whereas it generally yielded more in each of the 3 previous years (Table 1). This is presented as evidence of the destructiveness of a severe epidemic of net blotch. No factors other than the extreme susceptibility to this disease were apparent to account for the reduced yield of Betzes. Only trace to light infections of other diseases were recorded in the area.

Weather conditions were correlated with the prevalence of net blotch in each year. Precipitation is one of the most important factors because periods of high humidity are required for the development of an epidemic. In 1960 adequate moisture early in the season was followed by drought in July and net blotch infections were reported to be moderate. In 1961 dry conditions prevailed throughout the growing season and net blotch infection was trace to light. In 1962 rainfall was above normal but net blotch was only trace to moderate, probably because of the reduction of inoculum the previous two years. In 1963 average moisture and above average temperatures prevailed in the area and net blotch infection was severe throughout the region.

Betzes has been removed from the list of barley varieties recommended for Manitoba because of its extreme susceptibility to net blotch.

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Table 1. A comparison of the yields of the barley varieties Betzes and Herta at 9 locations in Manitoba and Saskatchewan, 1960-1963

Location	Bet.	Her.	Diff.	Bet.	Her.	Diff.	Bet.	Her.	Diff.	Bet.	Her.	Diff.
Winnipeg[1]	80	80	0	56	58	-2	-	-	-	74	85	-11
Hargrave	70	63	7	24	22	2	63	56	7	52	58	- 6
Mountain Road	41	43	-2	30	26	4	56	55	1	37	42	- 5
Dauphin	69	50	19	89	70	19	77	76	1	36	50	-14
Grandview	65	56	9	24	21	3	45	50	-5	36	55	-19
Roblin	48	45	3	17	13	4	58	54	4	43	57	-14
Durban	73	75	-2	30	27	3	95	98	-3	52	63	-11
The Pas	78	66	12	-	-	-	81	68	13	63	75	-12
Melfort, Sask.	55	62	-7	30	26	4	26	26	0	66	76	-10
Mean Difference			4.3			4.6			2.3			-11.3
t		1.59			2.13			1.11			8.04	
t .05		2.31			2.36			2.36			2.31	

[1] Data for Winnipeg and Melfort were obtained from the Report on Cooperative Two-rowed Barley Test compiled by Dr. S.A. Wells, Lethbridge, Alberta; the remainder of the data appeared in the report of the Manitoba Zonation Trials for 1963 compiled by Mr. G.M. Young, University of Manitoba.

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SOME RECORDS OF KNOWN AND SUSPECTED PLANT-PARASITIC NEMATODES ENCOUNTERED IN CANADA IN 1963

B. E. Hopper[1]

Root-knot Nematodes

The peanut root-knot nematode, Meloidogyne arenaria (Neal, 1889) Chitwood, 1949, was intercepted on Sansevieria sp. from Florida, U.S.A. Two possible cases of this nematode were recorded on strawberry from Delaware, and Philadelphus sp. from Tennessee, U.S.A.

The northern root-knot nematode, Meloidogyne hapla Chitwood, 1949, was found on intercepted plant material from several areas in the United States: on rose from California and Texas, on strawberry from Alabama and Delaware, on honeysuckle from Alabama, on Weigela sp. from Alabama and Tennessee, on Spiraea sp. from New York, and on Deutzia sp. from Tennessee. It was intercepted on rose from Holland and Belgium and on Clematis sp. from Holland. From Ontario, the nematode was recorded on Philadelphus sp. from Cooksville, on parsnip from North Gower, on Berberis thunbergii from Brown's Line, on Berberis sp. from Guelph, Cyclamen sp. from Burlington, and on Deutzia lemoinei compacti from Glen Williams.

The southern root-knot nematode, Meloidogyne incognita (Kofoid and White, 1919) Chitwood, 1949, was found on interceptions of Lonicera sp. from Tennessee, Caladium sp. from Illinois, tomato from Georgia, Sansevieria sp. from Florida, and Hydrangea sp. and Forsythia sp. from Alabama, U.S.A. It was also found on Coleus sp. from Saskatoon, Sask., on Impatiens sp. from Edmonton, Alberta, on Peperomia sandersii from Dundas, Ont., Cyclamen sp. from Toronto, Ont., and Alternanthera sp. from Ottawa, Ont.

The Javanese root-knot nematode, Meloidogyne javanica (Treub, 1885) Chitwood, 1949, was found on eight occasions on tomato plants from Georgia, U.S.A.

In addition, Meloidogyne spp. were recorded on rose from England, on Lonicera sp. from Ohio, U.S.A., and tomato from Georgia, U.S.A.

Cyst-forming Nematodes

The oat cyst nematode, Heterodera avenae Wollenweber, 1924, was encountered from Holland in soil associated with Picea albertiana conica, P. kosteri, P. alba conica, Picea sp., Juniperus squamata meyeri, Juniperus sp., Taxus cuspidata hicksii, Taxus sp., Thuja occidentalis pyramidalis, T. occidantalis compacta, Thuja sp., Pinus mughus, Pinus sp., Prunus triloba, Caragana sp., Acer sp. and Euonymus alatus. From Belgium it was found in soil associated with Hydrangea sp., Caragana sp. (sent via Holland), Laurus sp., rose, cedar, tuberous begonia and other various trees and shrubs. In addition it was found from Philodendron sp., from Switzerland, Acer sp. from West Germany, soil from any army vehicle from France, and in soil surveys in Nova Scotia, Quebec and Ontario.

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The cactus cyst nematode, Heterodera cacti Filipjev and Schuurmans-Stekhoven, 1941, was tentatively identified from cactus soil from Switzerland and from soil from the rear fender of an improperly washed used car from Germany.

The cabbage cyst nematode, Heterodera cruciferae Franklin, 1945, was found in grape rooted cutting soil from the U.S.A.

The fig cyst nematode, Heterodera fici Kirjanova, 1954, was found from Ficus carica soil from Italy and tentatively from Spirea sp. soil from Belgium (sent via Holland).

The pea cyst nematode, Heterodera goettingiana Liebscher, 1892, was found in soil from Italy and in Thuja lutea soil from Holland.

The hop cyst nematode, Heterodera humuli Filipjev, 1934, was found in soil associated with Picea omorika, Picea albertiana, Picea sp., Thuja elegantissima, Thuja sp., Acer sp., Pinus mughus, Clematis sp., Pelargonium sp., juniper and Taxus cuspidata from Holland and Hydrangea sp. from Belgium. It was tentatively recorded from cactus, Euonymus sp., and ornamental cutting soil from Italy, Euphorbia sp., soil from Switzerland and Asparagus soil from France. In addition numerous tentative identifications were recorded from Holland and Belgium.

The grass cyst nematode, Heterodera punctata Thorne, 1928, was found from Holland in soil associated with Picea pungens, Picea conica, Picea albertiana conica, Picea kosteri, Tilia cordata, Viburnum opulus nanum, Pinus mughus, Rhododendron molle, Rhododendron sp. Syringa sp., Juniperus pfitzeriana, Juniperus sabina, Juniperus sp., Thuja rosenthalii, Thuja elegantissima, Thuja sp., Daphne sp. and possibly Taxus cuspidata nana and Taxus sp. It also was numerous in samples originating in Belgium associated with azalea, Hydrangea sp., Philadelphus sp., Weigela sp., Lonicera sp. Laurus sp., and rose. In addition the nematode was found associated with clover from England, soil in Newfoundland (survey), Cyclamen sp. from Germany, carnation from the United Kingdom, Sansevieria sp., Kafir lily and Christmas cactus from Greece, Philodendron sp. from Switzerland, Viburnum sp. from West Germany, and soil taken from two cars, both from Europe.

The golden nematode, Heterodera rostochiensis Wollenweber, 1923, was found in a soil survey in Newfoundland. It was intercepted in soil associated with heather from England, Taxus sp. from Holland, Hydrangea sp. from Belgium, Asparagus sprengeri from Poland, shamrock from Ireland, flowering cherry from Belgium and Cedrus sp. from Germany.

The sugar-beet nematode, Heterodera schachtii Schmidt, 1871, was found in lily-of-the-valley soil from Germany.

The clover cyst nematode, Heterodera trifolii Goffart, 1932, was intercepted on shipments with soil of Picea albertiana conica, Picea kosteri, Picea sp., Pinus mughus, Thuja occidentalis pyramidalis, T. o. compacta, T. rosenthalii, Juniperus squamata meyeri, J. sabina, J. pfitzeriana, J. glauca, Juniperus sp., Daphne sp., Taxus sp., Rhododendron sp. and Hydrangea sp. from Holland; Berberis sp., Hydrangea sp., Acer platanoides, Acer sp., Betula sp. Laurus sp., rose, azalea, Narcissus sp., and various trees, shrubs and ornamentals from Belgium, clover from England, variegated geranium, Sempervivum sp., and Aspidistra sp. from Italy, Cotoneaster sp. and Tilia sp. from West Germany, Euphorbia sp. from France, carnation soil from the United Kingdom, Coleus sp. from Portugal, and in soil surveys in the Provinces of Prince Edward Island, Quebec, New Brunswick, Ontario, British Columbia and Newfoundland.

Cysts identified only as Heterodera sp., were encountered in soil from shipments of Pinus mughus, Pinus nigra austriaca, Chrysanthemum sp., Thuja rosenthalii, Thuja occidentalis pyramidalis, Thuja occidentalis compacta, Picea kosterii, Acer sp., Taxus sp., Rhododendron sp., Hydrangea sp., Juniperus squamata meyeri, Clematis sp., and Fagus sp., from Holland, Lonicera sp., Cotoneaster sp., Forsythia sp., Robinia pseudoacacia, Quercus rubra, rose, Acer sp., azalea and Hydrangea sp. from Belgium, Tradescantia sp. and Pothos sp. from Switzerland, Viburnum lantana and Juniperus sp. from West Germany, Chrysanthemum sp., Sempervivum sp., mint, cactus, Aspidistra sp., and oleander from Italy, lily and myrtle from Poland, Sansevieria sp., Kafir lily and Christmas cactus from Greece, cactus from England, Chrysanthemum sp. from Yugoslavia, cactus from France, and from soil collected from cars (4) sent from Germany. In Canada, Heterodera spp. were found in soil surveys from the Provinces of Nova Scotia, Prince Edward Island, New Brunswick, Quebec, Ontario, and Newfoundland. In addition, cysts were found associated with Hydrangea sp. and tomato soil from Sherbrooke, Que., and alfalfa soil from St. Antoine de Lilly, Que.

Root-lesion Nematodes

Pratylenchus convallariae Seinhorst, 1959, was found in evergreen soil from Holland.

Pratylenchus crenatus Loof, 1960 was found in soil around roots of azalea from England, evergreens from Belgium, Picea sp. from Holland, dahlia from Portugal, and strawberry from Cobden, Ontario.

Pratylenchus neglectus (Rensch, 1924) Filipjev and Schuurmans-Stekhoven, 1941 was found in soil around roots of Thuja sp. from Holland and strawberry from Cobden, Ontario. The species was tentatively identified from rose soil from Richmond, Indiana.

Pratylenchus penetrans (Cobb, 1919) Filipjev and Schuurmans-Stekhoven, 1941 was found in soil around roots of Helleborus sp., Cytisus sp., Weigela sp., Spirea sp., Pinus sp., and evergreens from Holland, hydrangea from Belgium, cactus and ivy from Italy, begonia from Portugal, and Betula sp. from Tennessee, U.S.A. In addition, it was recorded in strawberry soil from Cobden, Ont., and apple seedling soil from a nursery in Carlisle, Ont.

Pratylenchus pratensis (de Man, 1880) Filipjev, 1936 was found in soil around roots of Cytisus sp. from Holland and fern from Portugal.

Pratylenchus thornei Sher and Allen, 1953 was tentatively identified from soil around the roots of an ornamental shrub from Italy.

Pratylenchus sp. was found in soil associated with the roots of cotoneaster, evergreens, Cytisus sp., lilac, Thuja sp., Buxus sp., and Picea glauca from Holland, hydrangea and Buxus sp. from Belgium, Helleborus sp. and ornamental shrubs from Italy, Colocasia esculenta from Portugal, dahlia tubers from Greece and soil from Pakistan.

Stunt Nematodes

Tylenchorhynchus acti Hopper, 1959 was found in soil associated with the roots of Juniperus sp. and evergreens from Holland.

Tylenchorhynchus brevidens Allen, 1955 was found in association with Acer sp. from Holland, cactus, ivy and sage cuttings from Italy and apple seedling soil from Carlisle, Ont.

Tylenchorhynchus bursifer Loof, 1959 was found in shipments with soil of rhododendron, Picea sp. and Malus profusum from Holland.

Tylenchorhynchus capitatus Allen, 1955 was found in soil associated with roses from Newark, New York.

Tylenchorhynchus claytoni Steiner, 1937 was intercepted in soil around roots of rhododendron, cotoneaster, hyacinth bulbs, evergreens, Thuja sp., Euonymus sp., Picea sp. and Pinus sp. from Holland, oleander cuttings and succulent plants from Italy and azalea from Lynden, Washington.

Tylenchorhynchus dubius (Buetschli, 1873) Filipjev, 1936 was found in the soil around hydrangea from Belgium.

Tylenchorhynchus maximus Allen, 1955 was found in association with tobacco and potato from Cobden, Ont., Poa pratensis from Waterdon, Ont., and soil from Merrickville, Ont.

Tylenchorhynchus spp. were also found in soil from hydrangea from Belgium, cactus, catalpa cuttings, and ornamental shrubs from Italy, soil from Pakistan and Jamaica, Acer sp. from Holland, rye from Cobden, Ont., and lily bulbs from Toronto, Ont.

Spiral Nematodes

Helicotylenchus spp. were found in soil associated with roots of cactus, ivy and Euonymus japonicus from Italy, Juniperus sp. and Picea sp. from Belgium, Colocasia esculenta from Portugal, Buxus sp., Acer sp., and Dieffenbachia omoena from Holland, apple from Merrickville, Ont., Alternanthera sp. from Ottawa, Ont., and lily bulbs from Toronto, Ont.

Rotylenchus goodeyi Loof and Oostenbrink, 1958 was found in Pinus sp. soil from Austria.

Rotylenchus robustus (de Man, 1876) Filipjev, 1936 was found in soil associated with roots of primrose plants from England, Weigela sp. from Holland and herbaceous plants from Italy.

Rotylenchus uniformis (Thorne, 1949) Loof and Oostenbrink, 1958 was found in association with Thuja pyramidalis, Adrus libani, azalea and hydrangea from Belgium, and Thuja sp., lilac, Acer sp., Euonymus sp., Juniperus sp., Picea sp., Koster blue spruce and Pinus sp. from Holland.

Scutellonema brachyurum (Steiner, 1938) Andrassy, 1958 was found in evergreen soil from Holland.

Ring Nematodes

Criconemoides xenoplax Raski, 1952 was found in lilac soil from Holland.

Criconemoides sp. was found in Helleborus sp. soil from Italy.

Pin Nematodes

Paratylenchus nanus Cobb, 1923, was found in Ontario from soil associated with Poa pratensis and tobacco.

Paratylenchus veruculatus Wu, 1962 was found in azalea soil from England.

In addition, Paratylenchus spp. were found in soil associated with begonia from Germany, evergreens from Holland, herbaceous plants from Italy and iris rhizomes from Clarkson, Ont.

Other Tylenchids

Anguina graminophila (Goodey, 1933) Christie, 1959 was found on Calamagrostis canadensis in Rupert and St. Martin, Que.

Hexatylus sp. was found in apple seedling soil from Carlisle, Ont.

Rotylenchulus sp. was found in Helleborus sp. soil from Italy.

Boleodorus sp. was found in Berberis sp. soil from Holland.

Ditylenchus dipsaci (Kuhn, 1857) Filipjev, 1936 was found in iris bulbs from Washington, U.S.A.

Species of the genera Aglenchus (Andrassy, 1954) Meyl, 1961, Tylenchus (Cephalenchus) Goodey, 1962, Filenchus (Andrassy, 1954) Meyl, 1961, Lelenchus (Andrassy, 1954) Meyl, 1961, Neoditylenchus Meyl, 1961, Psilenchus de Man, 1921, Tetylenchus Filipjev, 1936, and Tylenchus Bastian, 1865, were also found in association with soil and plants imported from abroad and from some areas in the United States and Canada.

Aphelenchids

Aphelenchoides parietinus (Bastian, 1865) Steiner, 1932 was found in soil supporting hydrangea from Belgium, primrose, Picea sp., ornamentals, grass, lichens, moss and soil from Austria, azalea from Portugal and Thuja sp. and Picea nidiformis from Holland.

Aphelenchoides subtenuis (Cobb, 1926) Steiner and Buhrer, 1932 was identified tentatively in soil about the roots of hydrangea from Belgium and ornamental shrubs and sage cuttings from Italy.

Aphelenchoides spp. were recorded from soil supporting various conifers, roses and herbaceous plants from Belgium, Holland, Portugal and Italy. From the United States it was recorded from California, Indiana, Massachusetts and Michigan. It also was found in soil from Cobden and Clarkson, Ontario.

Aphelenchus avenae Bastian, 1865 was found in soil supporting cactus, ivy, ornamental shrubs and succulent plants from Italy, azalea and primrose from England, begonia from Germany, lilac, Picea sp., Acer sp., Weigela sp., Pinus mughus and evergreens from Holland and amaryllis from Portugal. In North America it was detected in shipments of soil supporting Clematis paniculata from Massachusetts, Hedera sp. from Washington, strawberry from Manitoba and Croft lily, apple seedlings, strawberry, potato, tobacco, iris and Alternanthera sp. from areas in Ontario.

Aphelenchus spp. were found in soil supporting hydrangea from Belgium, catalpa from Italy, dahlia from Greece and Portugal, Dieffenbachia amoena from Hong Kong, hyacinth, Koster blue spruce and Thuja sp. from Holland, rose from New York, Viburnum opulus from Tennessee, azalea from Washington and Ligistrum sp. from Manitoba.

Paraphlenchus sp. was found in soil in association with the roots of Picea sp. from Holland and iris (rhizome type) from a Clarkson, Ont., nursery.

Seinura sp. was recorded from samples of soil supporting azalea from Belgium, Dieffenbachia omoena and evergreens from Holland, caladium bulbs and ferns from Portugal, oleander cuttings from Italy, and Croft lily bulbs from a nursery in Thornhill, Ont.

Dorylaimids

Diphtherophora sp. was found in ornamental shrub soil from Italy and in soil from around roots of apple trees in Merrickville, Ont.

Trichodorus primitivus (de Man, 1880) Micoletzky, 1922 was found in Acer sp. soil from Holland. Specimens of Trichodorus sp. were recovered from soil associated with the roots of azalea from England and Belgium, Picea sp. from Holland and Hedera sp. from Michigan, U.S.A.

Triplonchium sp. was found in evergreen soil from Holland and in soil from around roots of apple trees in Merrickville, Ont.

Tylencholaimellus striatus Thorne, 1939 was found in soil supporting Helleborus sp., Taxus sp., and evergreens from Holland, and azalea from Lynden, Washington. Specimens of Tylencholaimellus sp. were also associated with Thuja sp. and Weigela sp. from Holland.

Tylencholaimus sp. was found in ornamental shrub soil from Italy and Pinus sp. soil from Austria. The latter record was tentatively identified as T. steckii.

Xiphinema americanum Cobb, 1913 was found in soil associated with the roots of Betula sp. from Tennessee, U.S.A., and apple seedlings, strawberry and lily bulbs from Ontario. Xiphinema sp. was also found in Helleborus sp. soil from Italy.

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APPARENT REDUCTION OF LITTLE CHERRY DISEASE SPREAD IN BRITISH COLUMBIA[1]

Jack M. Wilks and Maurice F. Welsh[2]

During the years 1933 to 1948 the virus that causes little cherry disease was distributed very rapidly through the Kootenay region of British Columbia. All sweet and sour cherry trees in most Kootenay districts became infected within this 15-year period. The virus was transmitted very efficiently within orchards and the disease made sudden appearances in districts at distances of 50 miles from the nearest known source of infection.

Extension of disease spread to the remaining Kootenay districts, to other fruit-growing regions of British Columbia, and to neighboring parts of the United States was anticipated. There was particular concern for the Okanagan Valley, where the bulk of British Columbia's cherry crop is grown. This area is connected to the Kootenays by 2 highways, each about 300 miles long, traversing 2 mountain ranges.

The disease has not appeared in the Okanagan Valley, and there has been no evidence of spread from the Kootenays to other fruit-growing regions of the Pacific Northwest. The several reported appearances of little cherry disease in other widely separated regions (7, 5) can be more logically ascribed to spread of the virus from ornamental flowering cherry to sweet or sour cherry (8).

Meanwhile, within the Kootenay region the rate of spread of little cherry virus appears to have subsided since 1948. This assessment is supported by records of spread within orchards and by data on district-to-district spread.

Spread within experimental plantings

The rapid rate of spread pertaining during the early history of the disease was demonstrated in the isolated healthy orchard used for transmission tests in 1943 (3). In that year 20 sweet cherry trees were bud-inoculated. In 1944, 16 of these trees displayed little cherry symptoms. In 1945, the other 4 inoculated trees displayed symptoms, and 20 of the 24 uninoculated trees in the orchard were also diseased.

By contrast, spread has been slow in an experimental orchard of 52 Bing trees established in 1956 (Table 1). Sixteen of the trees have been inoculated during the years 1956 to 1963; seventeen trees have become naturally infected, and nineteen trees remained healthy in 1963. This orchard is surrounded by infected commercial orchards.

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[2]Plant Pathologists.

Table 1. Spread of little cherry virus disease in an experimental orchard near Creston, B.C.

Year	Trees Showing Symptoms		Trees Not Showing Symptoms
	Inoculated	Natural Infection	
1958	8	0	44
1959	4	1	39
1960	2	4	33
1961	0	7	26
1962	1	5	20
1963	1	0	19
Total	16	17	

Spread within commercial orchards

Surveys for little cherry disease in commercial orchards have also indicated contrasting patterns of spread during the two periods. In 1946, 155 infected trees were found in a survey of 5 selected orchards in the Creston district (2). In 1947, 568 additional trees in these orchards were showing symptoms. By 1949, essentially all the cherry trees in this, and most other Kootenay districts were infected and, in newly planted orchards, young trees often produced little cherry symptoms the first year that they fruited.

During the last 10 years, however, despite recommendations discouraging new plantings of sweet cherry in the Kootenay region, a limited acreage has been replanted with young cherry trees. Most of these trees remain healthy, although neighboring sources of infection are abundant.

Spread to new districts

Surveys have shown a similar depression of the spread of the disease to new districts. By 1949 the disease had reached all Kootenay districts except several communities on the Arrow Lakes, at the western edge of the Kootenay fruit-growing region (Fig. 1). Surveys in that season indicated that all cherry trees were diseased as far west as New Denver. All orchards were healthy in Nakusp, on Upper Arrow Lake, 30 miles west of New Denver, but most trees were diseased in Makinson and Burton, on South Arrow Lake, 18 and 23 miles southwest of Nakusp. No diseased trees were found in the Fauquier-Needles and Edgewood districts,

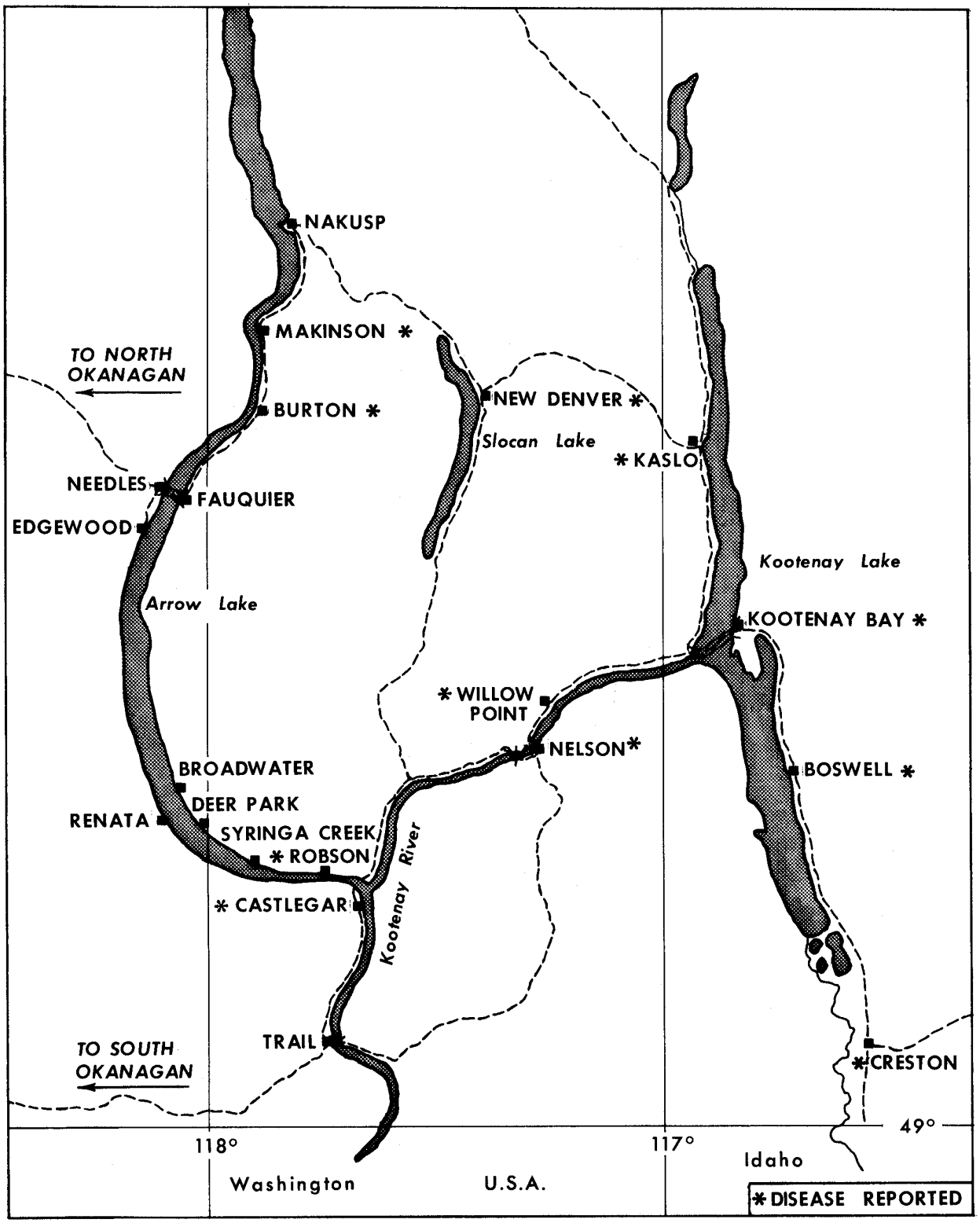

Figure 1. Distribution of little cherry virus disease in the Kootenay and Arrow Lakes districts of British Columbia.

14 and 20 miles southwest of Burton. A survey in 1960 disclosed no spread of the disease in the intervening 11 years into the Nakusp, Needles-Fauquier or Edgewood plantings. At the south end of the Arrow Lakes, by 1949, the disease had affected all trees as far west as the Robson district. There were no diseased trees at Syringa Creek, 9 miles west of Robson, or in the Renata, Deer Park and Broadwater districts, approximately 10 miles northwest of Syringa Creek. Subsequent surveys to 1962 have failed to demonstrate any spread into these 4 districts.

Thus, the disease spread during a 15-year period from its point of first discovery at Willow Point, into all trees of all orchards to points 70 miles southeast; 30 miles northeast, 40 miles west, and 65 miles northwest, yet it has not gained entry to any additional districts since 1949.

Discussion

It seems necessary to assume that during the recent period of reduced spread, vector activity has diminished. This could be reduction in vector efficiency of the species responsible for earlier rapid spread. It could also result from the disappearance of one or more efficient vectors, as reported for mosaic of sweet potatoes in Georgia (4).

The virus causing little cherry disease has been transmitted in screenhouse and orchard tests (9) by the leafhopper, Macrosteles fascifrons (Stal). There have also been isolated experimental transmissions by Scaphytopius acutus (Say) and Psammotettix lividellus (Zett.). Orchard populations of M. fascifrons have been high in most seasons but transmission tests have indicated that its vector efficiency is so low that its ability to effect epidemic spread of the disease is hard to envisage. All of these leafhoppers were collected and tested after 1949 when the disease was no longer spreading rapidly. Thus, the collections were not necessarily representative of the orchard fauna during the period of rapid disease spread.

The populations of leafhoppers in orchards and the rate of spread of the disease have been reduced experimentally by the application of 2 sprays per year of DDT and sulphenone (10). These and other insecticides have been introduced into spray programs for apple and pear in Kootenay orchards. They should have effected some reduction of leafhopper populations on interplanted cherry trees. However, the cherry plantings in Arrow Lakes districts that have remained free from the disease are not associated with sprayed pome fruit plantings, and have themselves received no insecticide sprays.

The drop in rate of disease spread coincided with the occurrence of the most severe winter in the history of British Columbia fruit growing. On January 25, 1950 temperatures in all Kootenay districts dropped to -15°F or lower (1, 6) and thousands of fruit trees were killed. It is tempting to speculate that this uniquely rigorous winter eliminated the most effective vector or vectors from all Kootenay districts, although relatively inefficient vectors such as M. fascifrons survived to effect further slow spread of the disease.

The little cherry disease has been mentioned frequently in scientific and popular literature as an example of the epidemic proportions that can be reached by tree fruit virus diseases. Thus a report of the altered rate of spread appears to be pertinent. The cherry plantings of the Kootenays remain under surveillance, so that any return toward the former rate of spread can be detected promptly.

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COMMENTS ON "YELLOW-LEAF CONDITION OF UNKNOWN CAUSE ON OATS IN ONTARIO"

Geo. W. Robertson[1]

In their article entitled "Yellow-leaf condition of unknown cause on oats in Ontario", Zillinsky and Slykhuis (2) do not discuss cold temperature injury as a possible cause of discoloration. They do hint, however, that "temperature and possibly other environmental factors are of critical importance."

It was pointed out that the affected crops were in the 3-to 4-leaf stage, presumably having been planted prior to May 1. Crops sown after May 1, according to the authors, were unaffected. Since it takes about a month, depending on soil temperature, for seed to germinate and the young plants to reach the 3-to 4-leaf stage, it is apparent that many crops sown late in April would be reaching this stage about the last week in May. Stem elongation and primordia development would be getting underway and the plant would be entering the period of grand growth. At this stage the plant might be undergoing an increasing susceptibility to low-temperature damage, having lost its earlier hardiness.

Abnormally cold weather occurred in western Ontario during the last week of May and lasted for three days. Lowest minimum temperatures, between 25 and 30°F occurred on the 24th (Table 1). Such low temperatures late in May are usually accompanied by clear skies and light winds. These conditions favor strong radiational cooling of the soil and vegetation surfaces and the latter may fall several degrees below the minimum temperature observed in the thermometer shelter. Air drainage would also be quite pronounced resulting in cold air being trapped in low areas, in flat areas with no drainage outlet, and in areas sheltered or protected by trees and hedges. Variations in physical characteristics and wettness of the soil would also affect the degree of radiational cooling. These factors would contribute to a spotty distribution of cooling at leaf level and temperatures might vary up to 10 to 15 degrees colder than the minimum temperatures recorded in the thermometer shelter.

Whether temperatures, ranging up to 20 degrees below freezing, would cause the yellowing of oat leaves may be open to argument. The date of occurrence is about right and the interval between the occurrence of the cold temperatures and the reported inspection of yellowing is not unduly long. The manifestation of a suspected cold effect might not be noticed for several days after the occurrence of the cause. Furthermore, the authors do not say when the manifestation was first observed, but it must have occurred several days before their inspection of the area on 4 June.

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Minimum Temperatures - Southern Ontario - Spring 1963[1]

	May													June		
	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2
Chatham	49	48	49	45	39	32	38	35	40	51	57	54	51	52	52	58
Ridgetown	48	47	48	46	38	31	35	37	40	51	56	53	51	51	53	60
New Glasgow	48	47	48	43	37	35	33	34	38	50	54	53	50	49	47	57
Wallacetown	48	48	48	44	39	36	32	33	37	49	55	53	51	49	45	57
St. Thomas	49	48	48	39	37	36	29	34	38	45	56	52	51	45	48	54
Port Burwell 1.	52	50	48	44	41	36	30	34	38	45	56	53	52	45	46	59
2.	50	50	48	41	37	35	29	35	37	45	56	54	52	46	47	58
3.	51	49	48	38	38	34	28	31	35	43	57	53	52	45	46	58
4.	52	50	48	39	39	34	28	34	37	45	57	53	52	45	46	55
6.	52	50	49	45	41	37	30	34	40	46	56	53	52	45	46	58
Delhi	52	49	47	34	38	33	25	32	35	41	55	53	51	46	45	52
Simcoe	52	51	43	42	35	32	20	36	41	44	54	55	50	47	50	58
Hagersville	52	50	49	39	43	33	28	34	39	41	51	55	52	46	46	52
Oil Springs	47	47	48	37	38	31	31	33	37	43	56	50	50	48		
London	48	49	42	38	35	32	29	35	38	45	56	51	50	44	49	56
Woodstock	51	48	48	38	38	33	28	32	37	40	55	53	49	46	47	55
Brantford	52	49	49	36	40	35	28	33	39	41	54	56	52	46	46	50
Centralia	45	48	43	42	34	34	34	39	41	47	51	49	48	48	50	58
Stratford	49	46	45	40	37	31	28	34	37	44	51	51	46	44	47	56
Kitchener	52	47	47	41	37	32	30	35	40	43	53	52	46	47	50	53
Galt	53	49	47	38	40	34	28	33	38	41	54	54	49	49	48	51
Guelph	52	47	45	36	36	31	26	33	38	40	52	52	45	43	48	51

[1]From Monthly Record Meteorological Observations in Canada, Meteorological Branch, Department of Transport, May, June 1963.

Damage by cold temperature to seedlings of cultivated crops has been discussed by Sellschop, Makkink and Baier (1). In their paper they review other reports of damage and describe a technique for producing similar symptoms under controlled conditions. It appears that the published reports on this topic deal mainly with heat-loving crops such as corn, sugar cane and sorghum.

Whether or not cold temperatures had anything to do with the yellow-leaf condition, the fact still remains that temperatures were unseasonably cold. Because of the possible variability of temperatures at plant level they might have resulted, either directly or indirectly, in very patchy damage to the crop which may have been at a stage when plants were susceptible to cold injury. The authors obviously did not suspect low temperature as a cause of injury.

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RÉSUMÉ OF DATA ON BLACK STEM OF ALFALFA CAUSED BY ASCOCHYTA IMPERFECTA PECK[1]

H. W. Mead[2]

Introduction

Black stem of alfalfa and other forage legumes caused by Ascochyta imperfecta Peck is world-wide in distribution, including Europe (6), U.S.S.R. (34), and Argentina (48). It is particularly destructive in north temperate regions and has been extensively studied in the United States and Canada. During investigations on this disease the writer has reviewed published information from many sources and herein presents a brief résumé.

Description of the Disease

Black stem of alfalfa was first described by Stewart et al. (52). Subsequent descriptions by various authors (7, 10, 11, 22, 38, 54, 55) have added more details. In general, the symptoms are as follows: all above-ground parts —stems, petioles, leaves, inflorescence and seeds— may be affected. Dark brown to black lesions occur on the stems, petioles and peduncles and irregular brown to purple spots on the leaves and pods. The buds, crowns and upper parts of the roots also may be rotted (10, 19, 26). Symptoms vary with the host, the isolate, and the conditions under which the disease develops.

Damage to the Host

Lesions on the crown cause weakening and frequently death of the plants (10), and new shoots may be killed in the spring. The stems may be girdled and killed. Severe infection of the petioles and leaves causes defoliation, and girdling of peduncles and pedicels may cause flower drop. Infected pods frequently contain shrivelled seeds; these seeds, and also those that appear normal, frequently carry the fungus as mycelium on the seed coat (27). Seeds from severely infected plants germinate poorly (22) and the resulting seedlings may be blighted (27).

Environmental Relationships

Black stem develops most rapidly at cool temperatures and in the presence of free moisture from dew and rain on the plants (10, 31, 47, 48). This moisture is necessary for release of spores from pycnidia and for infection of stem and leaf tissues. Hot, dry weather tends to suppress the disease. The optimum temperature for disease development in the field (about 15° C) is considerably lower than that for growth on agar (20-22° C); the important factor for field infection is thought to be free moisture (31).

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The Black Stem Complex

Blackening of the stem and spotting of the leaves of alfalfa may be caused in varying degrees by at least 7 pathogens (20, 21, 44). They are Ascochyta imperfecta Peck, Phoma trifolii Johnson and Valleau, Cercospora zebrina Pass., Colletotrichum trifolii Bain and Essary, C. destructivum O'Gara, Stemphylium botryosum Wallr., and Pseudomonas medicaginis Sackett. Helminthosporium sorokinianum Sacc. ex Sorokin has also been isolated from legumes (40). The symptoms caused by these pathogens are confusing and frequently indistinguishable in the field at a given time because they are dependent on the weather, stage of growth of the host, and genetic constitution of the host. The relative importance of these pathogens varies from year to year and also from season to season (18). However, Ascochyta imperfecta is the most widely distributed and prevalent of these organisms during cool, moist weather.

Ascochyta imperfecta Peck

Description

This fungus was first described in 1912 from collections made in New York (35). It is an imperfect fungus of the order Sphaeropsidales. The pycnidia are submerged, ostiolate, globose, averaging 234 μ in diameter, and dark brown to black in color. The spores are hyaline, oval or cylindrical with rounded ends, straight or slightly curved, and predominantly non-septate. They vary in length from 4.3 to 13.6 μ (50). No perfect stage is known (10, 11, 51).

Genetics and cytology

Heterokaryosis and parasexuality have been observed in A. imperfecta. Using ultraviolet light as a mutagen, auxotrophic, morphological and antibiotic-resistant mutants were isolated from a wild type strain (49). This writer observed that, as hyphae from single spores of paired isolates approached each other on thin films of agar, anastomosis occurred between them. Nuclei were observed in the bridging hyphae. The cells of the hyphae were one- to many-nucleate.[1]

Life history

The fungus persists as mycelium and pycnidia on crop residue. Mature pycnidia are rarely found on the current season's growth, but are present on stubble and fallen leaves in the spring. They occur also on dead stems during the growing season. Infection of new shoots occurs as they grow through residue or stubble (12, 36). The spores which ooze from the pycnidia are spread by water, wind and insects (36, 39, 46). The resulting lesions are at first small but they coalesce on the stems to form large brown to black areas. The cycle is completed by maturation of pycnidia on crop residue during the winter. An exception to this cycle occurs in Alaska where mature pycnidia were found in August (12). The fungus is seed-borne (10, 24, 27) and it also persists in soil. It has been isolated from alfalfa fields 3 or more years old to a depth of 6 inches.

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[1] H. W. Mead. Unpublished data.

In cereal fields it occurred commonly the first year after alfalfa sod was plowed but was not obtained the second or third year. It was not obtained from soil of cereal rotations or from virgin prairie and woods (10). It will persist in dry stem material up to 10 years and on alfalfa seed for 3 years (10, 29, 38).

Taxonomy

Ascochyta imperfecta Peck
(Phoma medicaginis Malbr. and Roum.)
(Diplodina medicaginis Oud.) (8, 12)

This fungus is now widely known as Phoma herbarum var. medicaginis West. ex. Rab. (15), although some workers prefer to retain A. imperfecta (13).

Host-parasite relations

Spore germination begins in water in about 8 hours and is almost complete in 21 hours (46). Penetration is generally direct (2) but may also occur through stomata (50). Appressoria nearly always form as swellings on hyphal tips. The highest spore germination, the longest germ tubes, the most appressoria and the most penetration per 100 germ tubes occur on the most susceptible hosts (2). The pathogen has been found to be at first intercellular, then intracellular in dead and dying cells of leaves; in the stems, hyphae were inter- and intracellular in living cells. It was usually found in the cortex of the stem but in later stages around and between vascular cells and into the pith (50). These workers reported that the xylem was not invaded, but another (23) stated that 42 per cent of the root steles carried A. imperfecta.

Growth characteristics

Isolates of A. imperfecta differ in cultural characteristics, color, rate of growth, production of pycnidia, and septation (10, 13, 14, 28). These differences often are associated with origin (10, 13). Growth on potato dextrose agar is slow at 5-9° C, increases up to 22° C and falls off after 24° C (10, 14, 31, 36, 38). Differences in rates of growth have been shown by chromatographic analysis to be related to carbohydrate metabolism (14). The differences occurred between 24 and 72 hours; some isolates grew steadily, some lagged. Isolates responded differentially to amino acids, sugars and nitrogen sources in dry weight of mycelium and appearance of colonies (28).

Sporulation

Under natural conditions pycnidia rarely form on lesions during the growing season but are found in abundance on overwintered crop residue (29, 36, 38). When leaves or portions of lesioned stems were placed in a moist chamber, pycnidia formed within 48 hours[1]; this occurred also on infected seed (22). In laboratory studies (29) pycnidia formed on synthetic media and on sweet clover stems over a range of 20-80° F; the optimum was 65-75° F. Relative humidity was a controlling factor, development being best at 80-100 per cent, sparse at constant 100 per cent. On the synthetic

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[1] H. W. Mead, unpublished data.

media pycnidial formation was affected by the kind and concentration of sugars, balance of sugar and nitrogen, and, in the early stages the kinds of amino acids in the medium. Sporulation of A. imperfecta was increased by irradiating cultures with near ultraviolet light (25).

Pathogenicity

Isolates of A. imperfecta differ in pathogenicity among themselves and on different leguminous hosts (7, 12, 32). Inoculation of detached leaves of 10 legumes demonstrated clearly that there was specificity among 50 isolates. This was interpreted to mean that parasitic strains existed among these isolates (32, 37).

Pathogenicity tests have generally been conducted on plants of various ages, but several workers have used detached leaves (2, 32, 56). The effect on infection of age of the hosts has been investigated. Results have been inconclusive except in one set of experiments where it was shown that the most severe infection occurred on the youngest leaves (46).

Inoculum for pathogenicity tests is usually grown on agar or in liquid media for 2-3 weeks and scrapings from the agar or dilutions of the liquid in water are sprayed on the hosts. Addition of surfactants and stickers has been found to increase infection (5, 12, 38, 42, 53, 56). The same effect was obtained by homogenizing agar cultures in water and using the mixture as inoculum (56). Recently, successful growth and retention of viability of the fungus on wheat and barley kernels has been demonstrated (45).

Several factors influence infection. Most workers have found that constant free moisture on the plants for 2-3 days after inoculation was essential, and frequent moistening of inoculated plants after the incubation period increased infection (4, 46). Pre-wetting of plants also induced severe infection (46). Dense spore suspensions were more effective as inoculum than dilute mixtures (3, 5), and addition of nutrients, such as dextrose and asparagine to inoculum, and wounding of tissues, also increased infection (33, 46). Under greenhouse conditions, temperatures of 20-24° C are best for symptom expression (4, 10, 41, 53).

Rating of disease on inoculated plants has been done most frequently by means of a numerical scale related to number and size of lesions, and area of tissue destroyed (7, 32, 46, 56).

Host range

The following legumes have been shown to be susceptible to A. imperfecta in varying degrees under field and greenhouse conditions (10, 12, 18, 22, 32, 37, 51, 54):

Medicago sativa, M. falcata, M. lupulina, M. ruthenica
Melilotus alba, M. officinalis
Trifolium hybridum, T. pratense and 20 other spp.
Pisum sativum, Vicia faba, V. americana, V. cracca
Astragalus cicer, Coronilla varia, Hedysarum coronarium
Lathyrus corniculatus, L. sylvestris, L. tenius, L. uliginosis
Phaseolus vulgaris, P. aureus, P. calcaratus; Stizolobium derringianum.
Pisum sativum, Clitoria sp., Cajanus cajan, Arachis hypogaea

Control of black stem of alfalfa

Sources of resistance

Plant breeders have shown that Medicago dzawkhetica, M. suffructicosa, M. marina, M. ruthenica, and some hybrids of M. sativa and M. dzawkhetica are more resistant to A. imperfecta than common varieties of alfalfa (16, 37, 42, 43). Selected diploid clones of M. sativa and M. falcata were more resistant than selected tetraploid clones (17). A recent notice (9) reported the release of 3 clones of M. tunetana, highly resistant to A. imperfecta and Pseudoplea medicaginis. Resistant plants have also been found within M. sativa and M. falcata.[1]

Nature and inheritance of resistance

Physical and physiological characteristics: glossy, hairless leaves of Ladak were difficult to wet and were more susceptible than hairy types (7, 37). No indication was found of stimulatory substances on susceptible plants, or inhibiting substances on resistant plants (2).

Desirable varieties of alfalfa are tetraploid and because of this, inheritance of resistance to A. imperfecta is complex. Work with tetraploid alfalfa showed that hybridization and selection were effective in raising resistance levels (37). A study of diploid alfalfa indicated that resistance was determined by dominant and recessive genes, the former more prominent, and also by epistatic gene action (53). No studies to date have demonstrated a definite factorial basis for inheritance of resistance.

Chemical control

Black stem of alfalfa was reduced from 25 per cent infection to trace infection in the field by weekly heavy applications of Maneb and Dyrene (1). The authors stated that "..... as applied in these tests, the fungicides would probably not be economical, and the possible effects of residues on hay were not known." Extensive field tests with many fungicides and other chemicals failed to control the disease in Minnesota (22) and in Saskatchewan[2].

The fungus was destroyed on alfalfa seed by treating with such fungicides as Ceresan, Arasan, Spergon, and Semesan (10, 27).

Crop rotation

Since about 2 years is required to build up inoculum in alfalfa fields, rotation with non-legume crops has been recommended (22).

Burning

Destruction of inoculum by spring burning of alfalfa fields has reduced infection by A. imperfecta (22, 57).

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[1] R. K. Downey and H. W. Mead. Unpublished data.

[2] H. W. Mead. Unpublished data.

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