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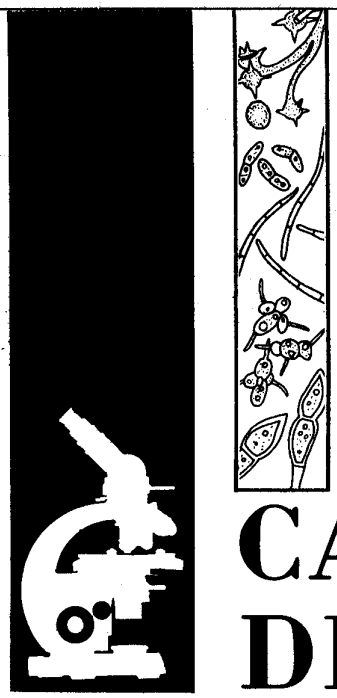

CANADIAN PLANT DISEASE SURVEY

EDITOR W.L. SEAMAN

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

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CONTENTS

"The Canadian Plant Disease Survey is a periodical of information and record on the occurrence and severity of plant diseases in Canada. It will also accept other original information such as the development of methods of investigation and control, including the evaluation of new materials. Review papers and compilations of practical value to phytopathologists will be included from time to time."

NATURAL INFECTION OF TOMATO FOLIAGE BY PLEOSPORA HERBARUM [1]

C.O. Gourley

Abstract

A severe foliage infection of tomato plants caused by Stemphylium botryosum Wallr. was found in a greenhouse in Nova Scotia. The symptoms of the disease were similar to those described on field tomatoes in Israel. The Nova Scotia isolate produced only sterile perithecia; however, based on its morphological similarity to an ascospore producing isolate from strawberry and on the perithecial primordia which formed in cultures, it was identified as the asexual form of Pleospora herbarum (Fr.) Rabh.

Introduction

In the fall of 1969 the foliage of tomato plants, cultivar Eurocross BB, became severely blighted in a greenhouse at Greenwich, Kings County, Nova Scotia. The disease was first thought to be early blight, caused by the fungus Alternaria solani (Ell. & Mart.) Jones & Grout. However, a Stemphylium species sporulated on leaf lesions in moist chambers, and it was the only fungus isolated from diseased leaves. Because of the perithecial primordia produced in cultures it was thought to be Stemphylium botryosum (Fr.) Rabh.

S. botryosum was reported by Rotem et al. (4) as the cause of a foliage disease of field tomatoes in Israel. Because the fungus attacked only tomato foliage they considered it a separate forma specialis, which they designated S. botryosum f. sp. lycopersici. Samuel (5) reported S. botryosum as the cause of leaf blight and fruit spot of tomatoes in greenhouses in South Australia. In Nova Scotia the disease occurred on the foliage but not on the fruit of tomato plants in the greenhouse.

A brief account of the symptoms of the disease, the conditions conducive to infection, and the isolation and identification of the fungus is reported.

Observations

Symptom development

The tomato plants were grown in a plastic-covered greenhouse in which centrally suspended ventilator fans forced air toward the end of the house. Leaf lesions appeared to have occurred first on plants beneath the fans and gradually spread outward in the direction of the forced air. At the time the plants were examined approximately 75% and 25% of the foliage was destroyed at the center and at the ends, respectively, of the central bed. Occasionally infected leaves were found on plants in the two side beds. It was not possible to determine the source of the initial inoculum.

Leaf lesions first appeared in the older, lower leaves as small chlorotic spots in which the centers soon became necrotic. Many such spots occurred in a single leaf and these often coalesced to form extended areas of infection (Fig. 1). As the lesions increased in size, they became concentrically ringed, and the brown, necrotic tissue became dry and often cracked. Individual lesions varied from 2 to 10 mm in diameter. Severely infected leaves turned brown and dropped. Foliage injury was most severe during the harvest period. No infections were found on the fruit.

Isolation and Identification of the Asexual Stage of the Fungus

Infected tissue was surface sterilized in 1:1000 $HgCl_2$ solution, rinsed in two changes of sterile water and plated on potato-dextrose agar (PDA). Severel surface sterilized diseased leaves were placed in large petri plate moist chambers.

All the isolations made from infected tissue yielded pure cultures of a fungus that developed conidia of the Stemphylium type. A pinkish color, characteristic of some isolates of S. botryosum (2), developed in the mycelium of the colonies growing on PDA. Cultures incubated on the laboratory bench sporulated concentrically. There were no differences in the morphology or the size (38-64 x 14-25 u) of conidia produced on leaf lesions in moist chambers and those produced on PDA. The swollen apices of the

[1] Contribution No. 1429, Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

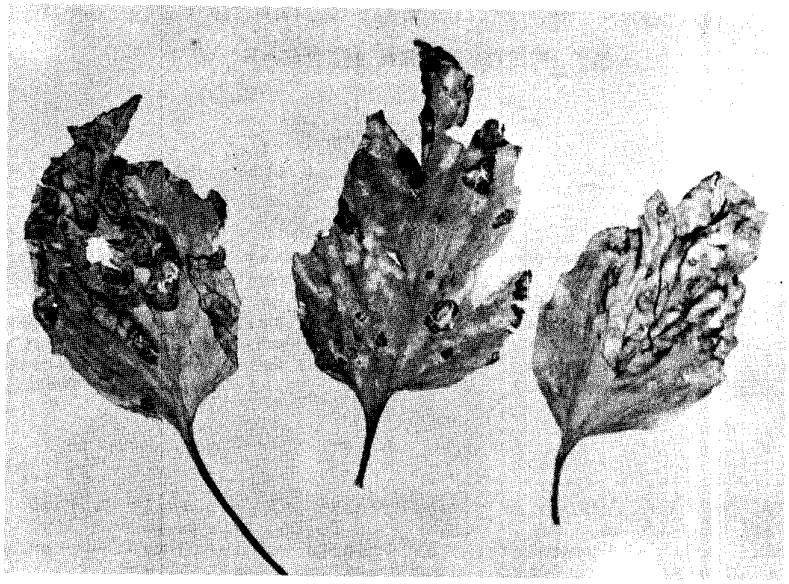

Figure 1. Lesions on tomato leaves caused by natural infection with *Stemphylium botryosum*. DAOM No. 137773.

sporophores, the conidia, and the perithecial primordia which formed in PDA cultures indicate that the fungus is S. botryosum (2, 6).

Sexual Stage

Numerous sterile perithecia formed in all PDA cultures of S. botryosum. Rotem et al. (4) showed that the perithecia which formed in cultures held for 2 weeks at 10, 15, or 20 C produced ascospores when exposed for an additional 14 days to 15, 20, and 25 C, respectively. However, with the Nova Scotia tomato isolate biweekly alterations of either upward or downward temperature shifts failed to induce the maturation of perithecia and the production of ascospores. Similarly an isolate of S. botryosum from strawberry, included for comparison, failed to produce ascospores. Neither constant temperatures nor various temperature shifts induced maturation of the sterile perithecia for either of these isolates in PDA cultures. When cultures containing immature perithecia were exposed to constant light, diffused daylight, alternate artificial light and dark periods, and constant darkness, no asci or ascospores were produced by either the tomato or strawberry isolates.

Sterile seedlings of three tomato cultivars, Eurocross BB, Stokesdale, and Viscount, were grown in test tubes as recommended by Neergaard (3), and eight seedlings of each cultivar were inoculated with each of the tomato and the strawberry isolates. Both isolates were pathogenic and perithecial primordia formed on the roots of all seedlings. These necrotic seedlings were held at 18 C and observed periodically. After 8 months, mature perithecia and ascospores of the strawberry isolate were found on the 'Stokesdale' and 'Viscount' seedlings. The tomato isolate did not

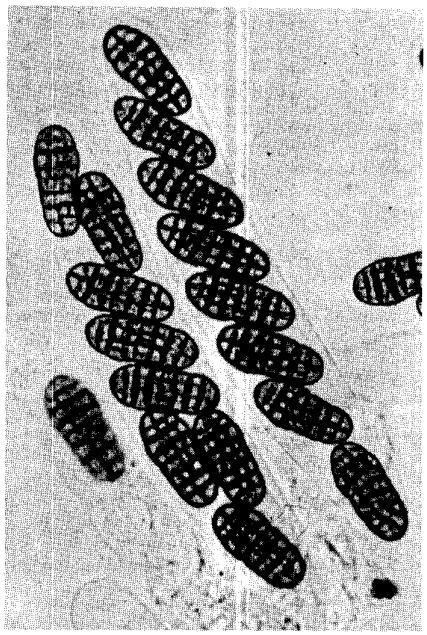

Figure 2. Ascospores of *Pleospora herbarum* produced by an isolate from strawberry, X 400. DAOM No. 137380.

produce mature perithecia on any of the seedlings. The ascospores of the strawberry isolate (Fig. 2) were typical of those described for Pleospora herbarum (Fr.) Rabh., and their size, 37.3 x 15.9 u, was within the range given for this species (2).

Discussion and conclusions

In Nova Scotia the symptoms of naturally infected tomato foliage in the greenhouse were similar to those of naturally infected tomato foliage in the field in Israel (4). S. botryosum is a weak, faculative parasite found ubiquitously on dead or weakened plant material. In Nova Scotia foliage infections were most severe during the harvest season. At this time plants are more vulnerable to weak parasites than at earlier growth stages. Rotem et al. (4) did not indicate the growth stage at which tomato plants were the most susceptible to natural infection under field conditions.

The morphology of S. botryosum isolates from tomato and strawberry were similar, and not unlike the isolates described as pathogenic on tomato foliage in Israel (4).

Rotem et al. (4) found that cultures of Stemphylium pathogenic on tomato produced sterile perithecia on nutrient media at constant temperatures between 5 and 30 C. They reported that a few isolates parasitic on tomato never produced perithecia, and that these were identical with the perithecia-producing isolates in their morphology and conidial dimensions. They did not find perithecia on naturally or artifically infected plants nor on their debris, regardless of the kind of treatment employed to stimulate their formation. Here the isolate of S. botryosum from diseased foliage of greenhouse grown tomatoes produced only sterile perithecia in culture and on artifically inoculated tomato seedlings. The morphologically similar isolate of S. botryosum from strawberry produced mature perithecia and ascospores of P. herbarum on tomato seedlings, but only after a long incubation period. Thus it is concluded that the tomato isolate is P. herbarum.

As far as the author is aware, P. herbarum has not heretofore been reported as the cause of a foliage disease of greenhouse grown tomatoes in Canada (1). The disease is not considered to be economically important because it occurred only on the foliage and its manifestation at a late stage of plant growth would have little effect on crop production.

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A HIGHLY VIRULENT STRAIN OF CUCUMBER MOSAIC VIRUS OCCURRING IN CUCUMBER IN EASTERN ONTARIO[1]

A.T. Bolton and V.W. Nuttall[2]

Abstract

A strain of cucumber mosaic virus (CMV) was isolated from Cucumis sativus L. at Ottawa, Ontario, that was more virulent than isolates of CMV 1 from Ontario, Maryland, and Wisconsin. The new strain also infected many cucumber varieties than were resistant to CMV 1. Other differences included the length of time the virus remained infective in cucumber and the concentration of the virus in cucumber as shown by assay tests on cowpea.

Introduction

Cucumber mosaic virus (CMV) has for years been one of the most destructive diseases of slicing and pickling cucumbers. Several strains of the virus have been reported by various workers, but the strain most commonly used in breeding for disease resistance has been referred to as cucumber virus 1, and varieties of cucumber described as mosaic resistant have, in most cases, been tested against this strain. Resistance to this strain does not, however, preculde susceptibility to other strains of cucumber mosaic virus. Porter (4) in 1931 described a strain that he referred to as cucumber virus 2 and reported that the cucumber variety Chinese Long, used as a resistant parent in much of the breeding work in North America, was susceptible to it. In 1934 Price (5) described two strains of CMV that produced yellow spots in tobacco leaves; one of the strains was capable of causing systemic infection in cowpea.

During the summer of 1966 fruits of the cucumber variety Armour growing at Ottawa became severely affected by cucumber mosaic. This variety had survived screening for CMV resistance using a strain of the virus obtained from Beltsville, Maryland, in 1965 and one isolated at Ottawa in 1962. Subsequently from 1966 to 1970, several reputedly highly tolerant cucumber varieties became severely infected with CMV in the Ottawa area. The work described in this paper was undertaken to determine if a strain of the virus was present in the Ottawa area that was different from the one isolated previously.

Materials and methods

In 1966 isolations were made from wilted 'Marketer' cucumbers (Cucumis sativus L.) growing in the field at Ottawa (isolate 0-66) and from severely infected plants in the greenhouse at the Ottawa Research Station (isolate C-66). The infectivity of this isolate was compared with that of the strain of CMV obtained from Beltsville, Md., in 1965 (isolate B-65); an isolation made from cucumbers at Ottawa in 1962 (isolate 0-62); and a strain of the virus identified as cucumber virus 1 obtained in 1966 from the University of Wisconsin (isolate W-1). Preliminary infectivity tests failed to show differences between 0-66 and C-66 and between B-65 and W-1; therefore isolates 0-66, 0-62, and W-1 were used in the experimental work reported here.

Inoculum was prepared by grinding systemically infected cucumber leaves and stems in 0.05 M phosphate buffer at pH 7.0 at a rate of 1 gram tissue to 10 ml buffer solution. The inoculum was rubbed lightly into cucumber cotyledons or leaves after dusting them with 600-mesh carborundum powder.

To test for rate of multiplication of the virus within cucumber plants, healthy Marketer plants were inoculated at the second true leaf stage and inoculum was prepared from them 3, 4, 6, 8, 10, 12, 16, 20, and 24 days later. This inoculum was rubbed onto cotyledons of 14-day-old Marketer plants and observations were made 14 days later.

Assay tests were made using the cowpea (Vigna sinensis Savi.) variety Dixielee. Cucumber plants of the variety Marketer were inoculated with each of the virus isolates in the usual manner. At various intervals inoculum was prepared from the infected plants and rubbed onto the leaves of cowpea plants that had been planted at intervals so that each inoculation was made on plants of the same age. Local lesions appeared about 48 hours after inoculation and were counted 4 to 6 days later.

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[1] Contribution No.303, Research Station, Canada Department of Agriculture, Ottawa, Ontario K1A 0C6.

[2] Present Address: Research Station, Canada Department of Agriculture, Harrow, Ontario.

Results

Each of the three stains 0-66, 0-62, and W-1 caused severe symptoms, including stunting and mottling, on the susceptible Marketer cucumber. There were distinct differences among the isolates in the degree of stunting, the amount of yellowing, and the mosaic pattern on the leaves. 0-66 caused very severe stunting but limited leaf chlorosis whereas 0-62 produced definite yellow blotches on the leaves. W-1 infection resulted in vein clearing and mottling of the type generally described for the disease on cucumbers. All three isolates caused the fruits to become extremely warty accompanied by yellow mottling.

Strain 0-66 produced mild mottling and moderate stunting of plants of the resistant cucumber variety Niagara, but symptoms on the fruits were severe. No symptoms were observed in this variety after inoculation with 0-62 or W-1. The variety Armour became slightly mottled and severely stunted after inoculation with 0-66, whereas W-1 caused mild mottling and 0-62 did not produce symptoms in this variety. Both 0-66 and W-1 caused severe fruit symptoms in Armour. The cucumber varieties Chinese Long and Tokyo Long Green were resistant to all three isolates.

In testing for reaction to infection by the various isolates it was observed that the time interval between inoculation of cucumber and attempts to recover the virus for assay affected the inoculum infectivity. When sap was taken from Marketer plants 16 days after inoculation, 0-66 and W-1 produced severe symptoms in cucumber while only very mild symptoms appeared in those inoculated with 0-62. Plants that had been infected for a shorter period of time were then used for inoculum production, with the result that severe symptoms were produced with 0-66 and 0-62 and mild symptoms with W-1.

Four experiments run over a period of 7 months indicated that isolates 0-66 and W-1 were most infective 12-16 days after inoculation and that 0-62 was most infective 8 days after inoculation (Table 1). There was also some variation among the strains in the time interval during which the plants remained infective.

The differences among the three strains in time required to reach maximum infectivity and in length of the infectivity period were demonstrated in an assay for virus titer using Dixielee cowpea as the local lesion host (Table 2). The concentration of 0-66 in sap from Marketer cucumber was considerably higher than that of the other two strains and it remained high over a longer period (Fig. 1). Isolate 0-62 reached maximum titer in 9 days and W-1 in 17 days after inoculation. Inoculum of 0-66 and W-1 prepared 24 days after inoculation did not produce local lesions in cowpea, but inoculum of 0-62 prepared 29 days after inoculation was infective. In four assays conducted over a period of 14 months under slighly different environmental conditions, these differences remained consistent (Table 2).

Table 1. Time required by three CMV isolates to produce severe symptoms in Marketer cucumber

Days after inoculation	Severity ratings* for virus isolates		
	0-66	0-62	W-1
3	+	-	-
4	+	-	-
6	+	+	-
8	++	+++	+
10	+++	+	++
12	+++	+	++
16	+++	+	+++
20	-	+	+
24	-	+	-

* Severity ratings: - = no infection to +++ = severe symptoms.

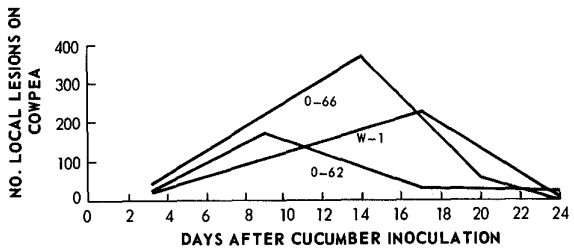

Figure 1. Pattern of infectivity of three isolates of cucumber mosaic virus as determined by assay on 'Dixielee' cowpea.

Discussion

Considerable variation in the incubation time of CMV in cucumber before the appearance of CMV symptoms has been reported. According to Kooistra (2) symptoms of cucumis virus 2 appeared 12-14 days after inoculation. Linnasalmi (3) reported that the mottling symptoms of CMV appeared 10-14 days after inoculation, and symptoms of cucumber green mottle mosaic usually could be observed 3 weeks after inoculation. According to Doolittle (1) symptoms of CMV appeared 6 days after inoculation. Porter (4) found that

Table 2. Recovery of CMV isolates from Marketer cucumber at various times after inoculation as expressed by the number of local lesions produced in Dixielee cowpea

CMV isolate	Days after cucumber inoculation	Average no. of lesions per cowpea leaf				
		Test 1	Test 2	Test 3	Test 4	Average
0-62	3	22	12	17	10	15.2
	4	38	52	50	29	42.2
	6	65	96	88	58	64.8
	9	187	150	192	163	173.0
	11	147	121	207	105	145.0
	14	45	67	82	110	76.0
	17		20	17	41	26.0
	20		18	10	26	18.0
	24		22	19	25	22.0
0-66	3	36	20	28	51	33.7
	4	51	73	49	46	53.8
	6	147	122	96	96	115.2
	9	268	148	163	195	193.5
	11	264	282	217	256	254.7
	14	387	415	326	352	370.0
	17		198	302	216	238.7
	20		78	37	51	55.3
	24		2	0	6	2.6
W-1	3	12	19	22	9	15.5
	4	52	23	17	26	29.5
	6	41	27	19	22	27.2
	9	102	111	86	94	98.2
	11	155	129	180	99	140.7
	14	196	217	224	168	201.2
	17		196	271	221	229.3
	20		97	154	137	129.3
	24		3	17	2	7.3

symptoms of CMV1 and CMV2 were produced 4 to 6 days respectively after inoculation.

In the present investigation there was little variation in incubation period within a single strain in spite of the fact that there were variations in environmental conditions during the course of the experiments. There were, however, definite differences in the pattern of infectivity between different strains.

Sill and Walker (6) reported difficulty in producing local lesions in the cowpea variety Black when the inoculum was obtained directly from severely infected cucumber plants, and they found it necessary to use tobacco plants as sources of assay inoculum. Sill and Walker (7) later attributed the lack of production of local lesions in cowpea to the presence of a virus inhibitor in cucumber. In the present study, using the cowpea variety Dixielee, no difficulty was encountered in obtaining local lesions using the sap directly from cucumber plants. The virus titer of sap from plants infected with strain 0-66 was much greater and remained so for a longer time than that of sap from plants infected with either isolate W-1 of cucumber virus 1 or isolate 0-62. It is possible that the inhibitor present in cucumber is less effective against 0-66 than it is against the other strains.

It seems quite evident that 0-66 is a strain of CMV distinct from cucumber virus 1 represented in the experimental work by W-1. The method by which 0-66 became widespread in the Ottawa area is not known. In the field this strain caused a high incidence of rapid

wilting in susceptible varieties such as Marketer, Highmoor, and Straight Eight. The virus isolated from wilted or severely stunted plants in the area from 1966 to 1970 in all cases proved to be of the 0-66 type. Attempts to produce the rapid wilting symptoms in the greenhouse and in growth rooms were unsuccessful so there is no proof that this is a typical symptom of infection with this particular strain. Strain 0-66 failed to cause systemic infection in Chenopodium amaranticolor and did not produce symptoms of any kind in Phaseolus vulgaris. The fact that moderate to severe symptoms appeared on fruits of varieties such as SMR58, Triumph, Saticoy MR, Hiyield MR, Challenger, Gemini, and Spartan Dawn at the Ottawa Research Station is reason for some concern. If these varieties are resistant in other areas in North America, strain 0-66 probably exists only in the Ottawa area; but if this strain is widespread these and many other supposedly resistant varieties will become infected.

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EFFECT OF TRANSPLANTING TOBACCO SEEDLINGS IN PEAT POTS ON PLANT VIGOR AND ON SUSCEPTIBILITY TO THIELAVIOPSIS ROOT ROT[1]

S.K. Gayed

Abstract

Seedlings of tobacco (Nicotiana tabacum cv. Hicks Broadleaf) grown in steam-sterilized soil were transplanted directly from the seedbed or in peat pots into a poorly drained field of Granby sandy loam that was heavily infested with Thielaviopsis basicola. Transplanting shock caused a marked delay in growth of transplants from the seedbed as compared with those in peat pots; four weeks after transplanting vigor was correlated with size of pot. However, at harvest there were no differences among treatments in yield or quality of tobacco or in damage from black root rot, indicating that resistance to the disease was not influenced by the relative vigor of the seedlings during the active phase of infection.

Introduction

Black root rot of tobacco caused by the soil-borne facultative parasite Thielaviopsis basicola (Berk. and Br.) Ferr. is a major disease of flue-cured tobacco in Ontario. Black necrotic lesions are formed on roots of infected plants and under severe conditions the plants are stunted resulting in a reduction of yield and quality. The disease is usually most severe in relatively fine-textured soils under poorly drained conditions.

Tobacco is commonly grown from seed in a 2-inch deep bed of steam-sterilized muck soil over coarse sand in an unheated greenhouse for 6-8 weeks prior to transplanting to the field. Transplants always suffer from shock due to root injury and to the sudden change of environment, resulting in a growth lag which lasts for several days. As transplanting tobacco seedlings in peat pot eliminates root injury and decreases the severity of transplanting shock, it was thought that vigorously growing tobacco plants set out in peat pots might resist infection with black root rot.

The aim of the experiments reported here was to compare the subsequent vigor and susceptibility of seedbed and peat pot seedlings transplanted to a field highly infested with black root rot.

Materials and methods

Experiments were carried out in 1966 and 1967 in Granby sandy loam soil in a poorly drained field in which tobacco had been grown for more than 13 consecutive years. The soil and cultural conditions favored a high inoculum potential of T. basicola and severe root infection.

In both years, tobacco (Nicotiana tabacum L. cv. Hicks Broadleaf) seedlings were grown for 4 weeks in seedbeds before transplanting to peat pots. Three weeks later, potted and seedbed seedlings of the same age were transplanted to the field. Steam-sterilized muck was used as a growth medium in both the seedbeds and the peat pots. The experiment in 1966 was comprised of two unreplicated treatments, each consisting of three rows 42 inches apart with approximately 20 plants spaced 24 inches apart in each row. These plots received seedbed seedlings and seedlings raised in 1.75 inch peat pots. The experiment in 1967 was comprised of four treatments in triplicate with plants spaced 18 inches apart in rows 42 inches apart. The treatments consisted of seedlings raised in a seedbed or in 1.75, 2.25 or 3 inch peat pots. The size of these peat pots represents a volume of 1:2:4 respectively. Transplants were set in the field during early June in both years. Transplants from the seedbed and those in 1.75 inch pots were similar in size and were slightly smaller than those in the larger peat pots.

Relative growth in the field was rated 4, 6, and 8 weeks after transplanting in 1966 and 4 weeks after transplanting in 1967 using a scale of 1 to 10, where 10 represents best growth. The height of aerial growth 4 weeks after transplanting was measured only in 1967. Nine weeks after transplanting, leaves at the lower-most stem positions started to ripen and were harvested and flue-cured. In the succeeding 4 weeks the remaining leaves were similarly harvested and cured. The cured leaves were weighed to determine yield

[1] Contribution No. 70, Research Station, Canada Department of Agriculture, Delhi, Ontario.

in lb per acre and were graded (Ontario Farm Products Grades and Scale Act) to determine the grade index in cents per lb.

On completion of harvest the fourth, seventh, tenth, and sixteenth plants in each row were dug from the soil, and the roots were thoroughtly washed and rated for disease lesions using a scale of 0.5, with 0 representing no lesions and 5, complete covering of the root with lesions.

Results and discussion

In the 1966 experiment, the growth rate of tobacco plants raised from seedlings transplanted in 1.75 inch peat pots was greater than that of transplants from the seedbed. The difference in vigor was pronounced 4-6 weeks after transplanting but gradually decreased as the plants advanced towards maturity (Fig. 1). At the end of the season, the root lesion ratings of plants originating from the seedbed and from peat pot transplants were similar and averaged 2.7 on the 0-5 scale.

In the 1967 experiment, the growth of tobacco plants transplanted in peat pots was significantly greater 4 weeks after transplanting than the growth of those from the seedbed (Table 1). At this time plant vigor was greater in the larger peat pots and was almost twice that of plants transplanted from the seedbed. At harvest, however, there were no differences in the size of tobacco plants in any of the plots. Similarly, there were no significant differences in yield or quality among the treatments. This disparity in the relative growth of tobacco plants early in the season and at harvest indicates that the stresses to which seedbed transplants had been subjected and which were responsible for a growth lag for several days in the field delayed the maturity of seedbed transplants. Therefore, the plants from the seedbed were still in the active growth phase while peat pot transplants were approaching maturity. Also there were no significant differences among the treatments in the disease lesion ratings, which in 1967 averaged 3.0 (Table 1).

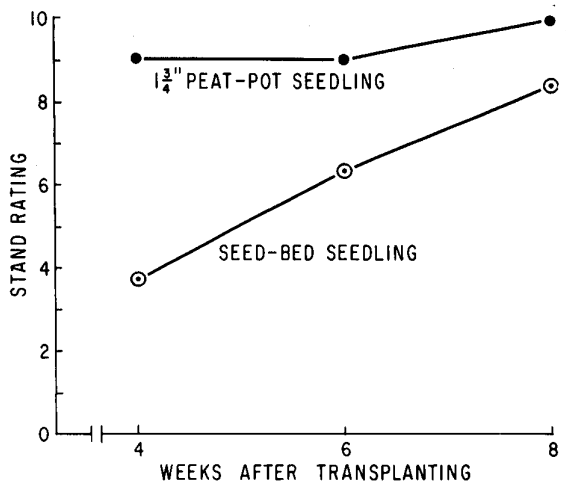

Figure 1. Rating of relative growth of tobacco plants in the field 4, 6, and 8 weeks after transplanting directly from the seedbed or in 1.75 inch peat pots; 1966 data. Stand rating: 10 =best growth.

In Ontario fields, the critical period for infection of tobacco by T. basicola begins at tranplanting and extends through June and early July. This observation is supported by evidence that younger tobacco plants are more susceptible to the disease than older ones (5) and that low soil

Table 1. Relative growth in the field of transplants from the seedbed and from peat pots and the subsequent yield and grade index of cured leaves and incidence of black root rot lesions on the roots, 1967

Source of transplant	Rating* of relative growth July 7	Shoot length July 7 (inches)	Yield (lb/acre)	Grade index (cents/lb)	Root lesion rating†
1¾ inch peat pot	6.4	9.4	441	40.2	3.0
2¼ inch peat pot	7.3	10.6	362	39.4	3.0
3 inch peat pot	9.1	13.0	350	42.9	3.0
Seedbed	4.4	6.7	431	40.9	3.0
L.S.D. 0.05	0.45	1.45	N.S.	N.S.	N.S.
0.01	0.69	2.20			

* 1 = very poor growth; 10 = best growth.

† 0 = no lesions; 5 = lesions covering root surface.

temperatures favor the development of the disease (4). In the present study, the more vigorous growth of peat pot transplants compared to those from the seedbed was apparent for up to 6 weeks after transplanting, the period during which most infections occur. However, the difference in early vigor was not reflected either in yield or in the root rot severity ratings of mature plants. Therefore, the general rule (2) that high host vigor usually increases host resistance to facultative parasites did not apply in this situation. Apparently the initially higher growth rate of seedlings in peat pots did not influence the chemical factor believed to be associated with resistance to black root rot of tobacco (1,3,6).

Acknowledgments

Thanks are due to Mr. D. A. Brown and Mr. G. Paloots for their technical assistance.

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FURTHER OBSERVATIONS ON THE EFFECTS OF ORCHARD FUNGICIDES ON STORED McINTOSH APPLES[1]

R.G. Ross and C.A. Eaves

Abstract

Sprays of captan, dodine, dichlone and phenylmercuric acetate were applied to McIntosh apples in two tests of 4- and 5-years duration. After 170 days storage, fruit sprayed with phenylmercuric acetate had a higher acid content than fruit sprayed with other fungicides. There were no differences among fungicide treatments in fruit firmness or sugar content. The best control of storage scab caused by Venturia inaequalis was obtained with dodine and the best control of Gloeosporium album rot with captan.

Introduction

Fungicides have usually been tested on apples to control diseases in the orchard and those that develop in storage. However, little attention has been given to their effects on quality of stored fruit. Eaves et al. (3) found that stored McIntosh apples from trees sprayed with phenylmercuric acetate (PMA) had higher acid contents than did fruit receiving other fungicides. They did not obtain differences in fruit firmness with the five fungicides used. Earlier studies showed that some fungicides increased the sugar content of apples (6). Garman et al. (5) in a comparison of several pesticides reported a lower acid content in fresh fruit receiving dichlone-lead arsenate sprays while captan-lead arsenate sprays tended to increase the acid content. They also found that apples sprayed with glyodin had a higher sugar content than apples sprayed with other fungicides.

Results of two tests, from 1959 to 1962 and from 1964 to 1968, on the long-term effects of fungicides on the performance of mature McIntosh apple trees have already been published (7, 9). This paper provides additional information from these two tests on the effects of these fungicides on storage rots and storage scab, acidity, firmness, and sugar content of stored McIntosh apples.

Materials and methods

The McIntosh apples used were treated as described in previous publications (7, 9). The treatments and rates of application per 100 gallons of water are listed in Table 1.

Treatments were randomized and applied to plots, each consisting of a row of six trees, in each of 4 blocks. Eight to 10 sprays, consisting of about an equal number of precover and cover sprays, were applied each year. At harvest 1 bushel of fruit obviously free of apple scab and insect injuries was picked at random from a tree in each plot, stored immediately at 0 C, and removed for examination after about 170 days. The number of fruit showing storage scab caused by Venturia inaequalis (Cke.) Wint. and rot was recorded and the cause of each rot was identified from fungal fruiting structures on the rotted areas, or after isolation on an agar medium. Ten apples from each sample were tested for hardness with a Magness-Taylor pressure tester. Titratable acidity was determined as before (4) and percent soluble solid was obtained with a refractometer and expressed as sucrose. Duncan's Multiple Range Test (2) was used to test for significant differences.

Results and discussion

In two tests on the effects of fungicidal sprays on stored McIntosh apples there were no significant differences between treatments in firmness of fruit or in fruit sugar content. No differences in firmness of fruit were obtained in an earlier test (3) which included two of the fungicides, PMA and captan, used here. In the 1959-1962 experiment the acid content of apples sprayed with the PMA-captan schedule was significantly higher ($P < 0.01$) than that of apples receiving the other treatments (Table 2). This confirms earlier findings (3) in which it was shown that precover sprays of PMA were associated with a high acid content of the fruit. With the other fungicides, captan, dodine, and dichlone, the acid content did not differ in the tests beginning in 1959 and 1964. Garman et al. (5) found that apples sprayed with dichlone-lead arsenate had a lower acid content, and fruit sprayed with captan tended to be more acid

[1] Contribution No. 1414, Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

Table 1. Fungicide treatments applied to McIntosh apple trees in precover and cover sprays during 1959-62 and 1964-68

Treatment*	Chemical name	Rate (lb formulation/100 gal water) Precover	Cover
1959-62			
Captan 50-W	captan 50%	2.0	1.5
Cyprex 65-W	dodine 65%	0.75	0.75
Phygon XL	dichlone 50%	0.5	0.25
Erad - Captan 50-W	PMA† 10%	0.5 pint	0.0
	captan 50%	0.0	1.5
1964-68			
Captan 50-W	captan 50%	2.0	1.5
Cyprex 65-W	dodine 65%	0.5	0.5
Phygon XL -	dichlone 50%	0.5	0.0
Captan 50-W	captan 50%	0.0	1.5
Captan 50-W -	captan 50%	2.0	0.0
Phygon XL	dichlone 50%	0.0	0.25

* Sources of fungicides: Stauffer Chemical Co., New York, N.Y. (Captan 50-W); Cyanamid of Canada Ltd., Rexdale, Ont. (Cyprex 65-W); Naugatuck Chemicals, Elmira, Ont. (Phygon XL); Green Cross Products, Montreal, Que. (Erad Eradicant Fungicide).

† PMA = phenylmercuric acetate (Hg equivalent of formulation, 6%).

Table 2. Effect of orchard fungicidal sprays on the acid content of McIntosh apples stored at 0 C for 170 days

	Malic acid (%)				
Fungicide	1959	1960	1961	1962	Mean*
Dodine	0.29	0.39	0.33	0.34	0.34 a
Captan	0.33	0.38	0.30	0.33	0.33 a
Dichlone	0.32	0.41	0.37	0.33	0.36 a
PMA - captan	0.35	0.47	0.43	0.41	0.42 b

* Numbers followed by the same letter do not differ significantly at the 1% level.

Table 3. Percentage of apple fruits affected by storage scab following preharvest treatment with fungicides and storage at 0 C for 170 days

Fungicide	1959	1961	1962	Mean*
Dodine	0.2	1.7	0.3	0.7 a
Captan	19.0	19.9	5.6	14.8 b
Dichlone	46.6	19.5	16.4	27.5 b
PMA - captan	15.6	6.2	0.7	7.5 b

* Numbers followed by the same letter do not differ significantly at the 1% level.

than apples receiving other fungicides.

Storage scab appeared only on the 1959, 1961, and 1962 crops and control with dodine was significantly better ($P < 0.01$) than that with the other fungicides (Table 3). The incidence of early and late scab in the orchard for these years has already been reported (9). There was a positive correlation between the amount of late scab in the orchard and that in storage. In a previous test (8) cover sprays of dodine at the rate of 0.75 lb of 65% formulation per 100 gal were effective in preventing storage scab but sprays at a 0.5 lb rate were relatively ineffective. That test, which showed that regular cover sprays of dichlone and captan did not give good control of storage scab, agrees with the results in Table 3.

Rots in storage were caused almost entirely by Penicillium spp., Gloeosporium album Osterw., and Botrytis cinerea Pers. Analyses of the data on total fungal rots showed no significant differences among treatments. However, in the 1959-1962 test, there was a significant difference ($P < 0.05$) between treatments in rots caused by G. album (Table 4). The full schedule of captan gave the best control followed by PMA with captan

Table 4. Percentage of apple fruits affected by *Gloeosporium album* in storage following preharvest treatment with fungicides, 1959-1962

Fungicide	1959	1960	1961	1962	Mean*
Dodine	1.7	12.5	3.6	2.3	5.0 b
Captan	1.2	3.2	2.2	0.8	1.8 a
Dichlone	3.2	10.6	4.5	2.3	5.1 b
PMA - captan	0.4	7.7	3.8	1.8	3.4 ab

* Numbers followed by the same letter do not differ significantly at the 5% level.

Table 5. Percentage of apple fruits affected by *Gloeosporium album* in storage following preharvest treatment with fungicides, 1964-1968

Fungicide	1964	1965	1966	1967	1968	Mean*
Captan	3.2	0.0	1.5	0.2	0.3	1.0 a
Dodine	5.6	1.2	3.4	0.0	1.3	2.3 a
Dichlone - captan	3.4	0.3	1.7	1.0	0.1	1.3 a
Captan - dichlone	4.0	0.2	1.7	0.3	0.6	1.4 a

* Numbers followed by the same letter do not differ significantly at the 1% level.

in the cover sprays. From 1964 to 1968 the differences among treatments were not significant for G. album but when captan was used in the spray schedule, the incidence of rotting by this organism was less than with dodine (Table 5). Control of G. album probably accounted for the good control of fungal rots obtained with PMA and captan in a previous test (3). In Nova Scotia G. album causes the only apple storage rot of any significance where infection occurs while fruit is on the trees. It has been suggested that control with PMA may be due either to a reduction in the amount of inoculum in the orchard or to physiological changes in the fruit as indicated by the high acid content (3). Subsequent work (1) has shown that it is probably due to a reduction in the inoculum.

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SURVEY OF PEACH CANKER IN THE NIAGARA PENINSULA DURING 1969 AND 1970[1]

W.C. James and T.R. Davidson[2]

Abstract

The incidence and severity of peach canker incited by the fungi Leucostoma cincta and L. persoonii were assessed on 2,000 peach trees in 93 orchards in the Niagara Peninsula, Ontario, in July 1969. Ninety-eight percent of the trees were cankered and, on average, approximately 30% infection (percentage circumference affected) was recorded for trunk, crotch, and scaffold branches, with 3 to 4 cankers on a 5-ft length of bearing limb. The orchards were revisited in July 1970 and the same trees showed an increase in infection of 10, 7, and 14% on the trunk, crotch, and scaffold branches, respectively, and an increase of 3 cankers per 5-ft of bearing limb. In addition 10% of the bearing area had been removed by the grower because of canker, and this alone was equivalent to a loss of approximately 1 million dollars in 1970. It is also probable that canker decreases fruit production by decreasing tree longevity, but this cannot be estimated from the results.

Introduction

The peach crop is the third most valuable fruit crop in Ontario. In 1969 the 10,000 acres of peach orchards produced 85 million pounds of fruit worth approximately $9 million (3). Peach canker is caused by the fungi Leucostoma cincta (Fr.) Höhn., (Valsa cincta Fr.), imperfect state Cytospora cincta Sacc; and Leucostoma persoonii (Nits.) Höhn. (Valsa leucostoma (Pers.)Fr.), imperfect state Cytospora leucostoma (Pers.) Sacc. (2, 5, 7). The disease is recognized as one of the most serious disorders of peach trees in the Niagara Peninsula, the main peach growing area in Ontario. During the period 1912-1917 McCubbin reported on the incidence of the disease in commercial orchards (2) and later Willison (6, 8) contributed much to our understanding of the disease by studying its development in experimental orchards. Programs to control the disease have been unsuccessful and the present investigation was undertaken to monitor the level and development of peach canker in a large number of commercial orchards selected at random in the peach growing area of Ontario.

Materials and methods

The sample used for this survey was based on a sampling scheme designed by the Dominion Bureau of Statistics to estimate prior to harvest the expected yield for the peach (Prunus persica (L.) Batsch) crop of Ontario. A sample of 93 orchards (Figure 1) was selected in proportion to orchard size, and 250 trees were selected in proportion to the number of bearing trees per orchard. The trees chosen were marked with paint so that they could be located easily in successive seasons. For our purposes, a further 1,750 trees approximately (again in proportion to the number of bearing trees but with a maximum of 50 trees per orchard) were selected in the immediate vicinity of the original 250 trees chosen (see example, Figure 1), thus making approximately 2,000 trees available for examination in the disease survey in 1969.

The disease assessment method involved estimates of the damage caused by canker. These estimates included the percentage circumference affected on the whole length of the trunk, crotch, and all scaffold branches. In addition estimates were made of the percentage of the crotch affected and the number of cankers on a 5-ft length of a 2- to 3-inch diameter bearing limb chosen at random. Where there were two or more cankers on the trunk or scaffold branches, the percentage of the circumference diseased was calculated as shown in Figure 2. The trees were classified by age into 5 groups of 5 years within the range 1 to 25 years, and a sixth group was included for trees from 26 to 50 years of age. A map of each orchard was made to facilitate locating the marked trees on the second visit in 1970, and schematic drawings of each tree were made on a short record form with details of cultivar, age, and disease assessments.

In 1970 the orchards were revisited and

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[1] Contribution No.298, Ottawa Research Station, Canada Department of Agriculture, Ottawa, Ontario K1A 0C6.

[2] Plant pathologists, Ottawa Research Station and Vineland Station respectively.

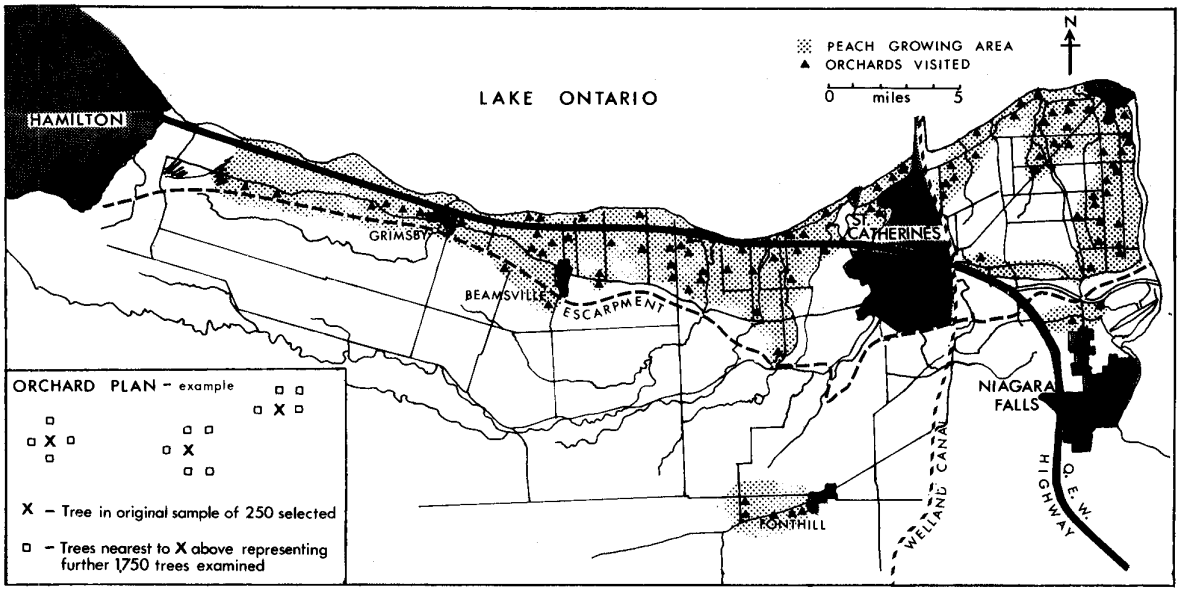

Figure 1. Location of peach growing areas and orchards surveyed for canker, with example of sampling scheme used for selecting trees within orchards.

the same detailed assessments were made on the 227 trees remaining from the original sample of 250 to determine the increase in canker, if any; note was also made of scaffold or bearing branches that had been removed by the grower because of canker. The additional 1,750 trees examined in detail in 1969 were checked only to determine the number and position of trees replaced since 1969. Both surveys were conducted during the latter part of July.

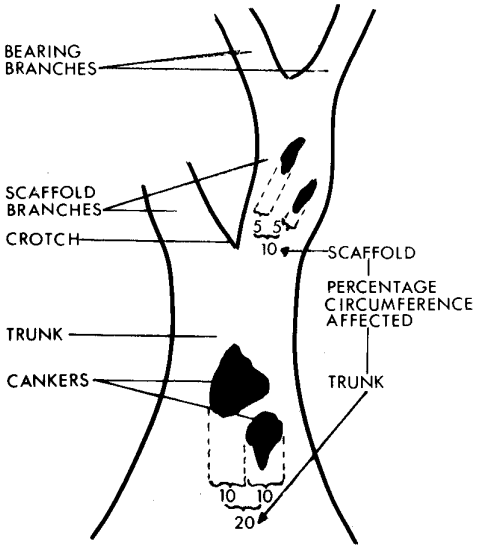

Figure 2. Diagrammatic representation of the method used for assessing canker severity.

Results

1969 Survey

A total of 1,969 trees were examined and 98% of them were affected with canker. On average approximately 30% of the circumference of the trunk, crotch, and scaffold branches was affected, and 3 to 4 cankers were found on a 5-ft length of bearing limb (Table 1). After the 1969 season 9% of the trees were removed. The average amount of infection on the trunk, crotch, and scaffold was higher on the trees that were removed after the 1969 season than in the 91% trees remaining, but the number of cankers per 5-ft of bearing limb was the same (Table 1). The average age of the trees that were removed was 16 years compared with 13 years for the trees that remained, but the age difference was not responsible for the difference in canker between the two groups of trees (Table 2). Within both age groups 1 to 20 and 21 to 50 years the trees that were removed generally had higher infection ratings than those that remained; the exception was in the number of cankers on the bearing limb which was approximately the same for the two age groups.

Of the 1,969 trees examined in 1969, the variety Jubilee was the most prevalent and accounted for 38% of all trees checked; the other varieties in decreasing order of importance were Elberta 33%, Veteran 18%, Redhaven 9%, and Loring 1%; other varieties represented the remaining 1%. No differences were noted among the disease assessments recorded for the above varieties.

Table 1. Average amount of canker on peach trees examined in 1969

Trees examined in 1969	No. of trees	Percentage circumference affected and corresponding standard error			No. of cankers per 5-ft length of bearing limb
		Trunk	Crotch	Scaffold	
All trees examined	1969	29 ± 0.6	26 ± 0.8	29 ± 0.5	3.7 ± 0.06
Trees remaining after 1969 season	1796	27 ± 0.6	24 ± 0.8	27 ± 0.5	3.6 ± 0.06
Trees removed after 1969 season	173	43 ± 2.1	37 ± 3.0	35 ± 1.8	3.7 ± 0.26

Table 2. Canker severity expressed as the percentage of the total number of trees in each disease category within two age groups; data for trees remaining and trees removed after assessment in 1969; all data recorded in 1969

Disease assessment method and disease category	Trees remaining after 1969		Trees removed after 1969	
	Age group (yr) 1-20	21-50	Age group (yr) 1-20	21-50
Percentage circumference of trunk affected				
0 to 50%	85	83	65	63
over 50%	15	17	35	37
Percentage crotch affected				
0 to 66%	83	85	75	72
over 66%	17	15	25	28
Percentage circumference scaffold affected				
0 to 50%	89	75	82	65
over 50%	11	25	18	35
No. of cankers per 5-ft length of bearing limb				
less than 5 cankers	69	47	73	60
5 cankers or more	31	53	27	40
Number of trees in each age group	1453	238	130	43

Approximately 50% and 20% of all trees examined were free from canker on the crotch and trunk, respectively, whereas only 10% of the trees were free from canker on the scaffold and bearing branches (Table 3). In approximately one out of eight trees the entire crotch areas was affected, but disease assessments of 100% were not noted for any trunk or scaffold branch. The data did not lend itself to a correlation analysis of age and amount of canker but the figures in Tables 2 and 3 show that in general the older trees had more disease than the younger ones. The increase of disease with age is most apparent for canker recorded on bearing limbs (Table 3); 45% and 3% of the 1- to 5-yr old trees and 0 and 5-6 cankers respectively, compared with 2% and 35% of 26- to 50-yr old trees. The same trend to more disease on older trees can be detected for disease assessments on the trunk, crotch, and scaffold. The indication that canker seems to increase rapidly with age up to approximately 15 years and then stabilizes is generally true for all the disease assessments recorded.

Table 3. Canker severity expressed as the percentage of trees in each disease category within various age groups; data for all trees examined in 1969

Disease assessment method and severity category		Age group of trees (yr) 1-5	6-10	11-15	16-20	21-25	26-50	All groups
Percentage circumference of trunk affected	0	51	20	12	8	10	12	20
	1- 10	11	21	18	19	19	14	18
	11- 25	9	15	16	13	14	14	13
	26- 50	21	30	35	37	36	41	33
	51- 75	4	10	12	15	14	13	11
	76- 99	4	4	7	8	7	6	5
	100	0	0	0	0	0	0	0
Percentage circumference of crotch affected	0	64	48	47	47	46	54	50
	1- 33	11	19	25	28	25	14	21
	34- 66	9	13	11	11	11	17	12
	67- 99	5	4	4	2	5	5	4
	100	12	6	13	12	13	10	13
Percentage circumference of scaffold branches affected	0	37	10	4	3	3	1	10
	1- 10	19	22	15	9	6	10	15
	11- 20	16	17	18	13	10	15	15
	21- 30	12	20	21	15	19	17	18
	31- 50	11	25	29	38	30	35	28
	51- 70	4	6	10	17	24	17	12
	71-100	1	0	2	4	7	4	2
No. of cankers on 5-ft length of bearing limb	0	45	12	3	2	1	2	10
	1- 2	38	27	21	13	11	12	22
	3- 4	13	38	37	34	33	38	33
	5- 6	3	16	28	31	35	35	24
	7- 8	1	6	8	16	17	8	9
	9- 10	0	1	3	2	3	4	2
	11- 12	0	0	0	1	0	1	0
	13- 14	0	0	0	1	0	0	0
No. of trees in each age group		280	539	357	407	218	163	

1970 Survey

The survey was repeated in 1970 to estimate the increase in canker since 1969. The variability in the disease assessments recorded in 1969 suggested that a sample of approximately 250 trees was adequate to detect an increase of 10% canker on the trunk or scaffold branches or an increase of one canker on a 5-ft length of bearing limb. Of the total sample of 1,969 trees examined in 1969 only 1,796 (91%) remained in 1970, and similarly of the original sample of 250 trees selected in 1969, only 227 (91%) remained in 1970. The increase in canker between 1969 and 1970 is shown in Table 4 and is based on the 227 trees examined in both years. The average percentage of trunk, crotch, and scaffold affected by canker increased from 33 to 44, 28 to 35, 31 to 45, respectively, and the number of cankers on 5-ft of bearing limb increased from 3.9 to 6.6. Records for the same 227 trees showed that the bearing area had been reduced by 10.1% due to the removal of scaffold or bearing limbs.

Table 4. Increase in canker severity from 1969 to 1970 in the 227 trees examined in both years

Canker assessment method	Increase in % circumference affected*
Trunk	10.6 ± 0.9
Crotch	7.0 ± 1.0
Scaffold	14.3 ± 0.8
Bearing limb	2.7 ± 0.16†

* Percentage and standard error.

† Average number of cankers per 5-ft length of bearing limb.

Discussion

One of the main purposes for conducting disease surveys using standardized disease assessment methods is to try to obtain records which are quantitative rather than qualitative, so that the importance of the disease under study can be established. However, only rarely is it possible to estimate how much real damage has been caused by disease, and perennial crops present a more difficult problem than annual crops because the effect of disease on the crop may not be manifest in the year the disease is recorded (1). Also, in the peach crop commercial practices such as thinning of fruit make the task of relating disease level to the yield of fruit per tree problematic. Disease surveys are important because they monitor the level and development of disease as it occurs in commercial crops where crop management and conditions are often different to those found in experiments.

The effect of canker on yield cannot be estimated in one simple measurement because some of the effects of the disease are direct and others indirect. For example, the 10% decrease in bearing area due to the removal of cankered scaffold and large bearing branches is a direct effect which is easily measured and is equivalent to a loss of approximately $1 million. On the other hand the decrease in tree longevity which undoubtedly is to some extent due to canker is indirect and difficult to measure. Although the data in Table 2 cannot be cited as evidence that all the trees removed were replaced because of canker (a large proportion were removed because of age), it is more than coincidence that the levels of canker on the trunk, crotch, and scaffold branches are much higher for the trees that were removed than for the remaining trees. However, it is interesting to note that the data for the number of cankers on bearing branches are the same for trees remaining and for those removed (Tables 1 and 2). The removal of badly cankered limbs in conjunction with a program to allow new and healthier bearing limbs to develop results in the number of cankers per 5-ft of bearing limb remaining constant. This suggests that cankers on trunk, crotch, and scaffold are the criteria that growers use for deciding whether a tree should be removed or not and that the number of cankers on a bearing branch has little significance in this decision. Canker can accelerate the tree replacement rate through decreasing the longevity of trees but it is not possible to estimate what proportion of the 9% replacement rate reported for this survey was due to canker.

The higher level of disease on the older trees may be due to their greater susceptibility or it may merely reflect the longer exposure period to disease, compared with the younger trees. Disease seemed to increase rapidly with age up to approximately 15 years and then stabilize; this may be the result of grower practice which allows disease to develop for a period of time until the canker level is unacceptable, resulting in a continuous program of removing cankered branches over a few years and finally replacing the tree. It should be noted that the results of the present survey reflect grower practice to a great extent and this may explain why no differences in disease assessments were recorded for the different varieties examined. However, it cannot be concluded that the varieties did not differ in their susceptibility to canker because the practice of replacing trees and removing cankered wood will tend to eliminate any varietal differences. The present results therefore do not conflict with the findings of Weaver (4) who reported a negative correlation between canker and rate of defoliation and on this basis classed the varieties Elberta and Redhaven resistant and moderately susceptible, respectively.

The survey reported here has shown that 98% of the trees in the commercial peach orchards of the Niagara Peninsula are affected by canker and that the data on average infection (Table 1) represents a high level of canker on the trunk, crotch, scaffold, and bearing branches. By making consecutive assessments in 1969 and 1970 on the same trees it was shown that there was a substantial increase of disease after one year (Table 4). The increase in disease between 1969 and 1970 cannot be used to project the levels of disease for a period of years because climatic and other factors affect its development, and consequently the increase will vary from year to year (8). However, the level of canker in the orchards in 1969 combined with the increase in 1970 provides evidence that canker is a very serious problem and should command a high priority in research.

Acknowledgments

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MICROFLORA OF BUCKWHEAT SEED, CHANGES IN STORAGE AND EFFECT OF SEED TREATMENTS ON SEEDLING EMERGENCE[1]

J.T. Mills and H.A.H. Wallace[2]

Abstract

The microfloral components of 37 lots of buckwheat (Fagopyrum sagittatum) seed harvested in Manitoba were determined after 7 days incubation. Field fungi predominated, with a high incidence of Botrytis sp. Storage of 11 tough and damp lots of buckwheat for 390 days in sealed jars in an unheated storage shed resulted in decreased germination, decreased infection with field fungi, including Botrytis, increased infection with storage molds, and increased moisture content. In contrast, the one dry sample stored for the same period showed increased germination, reduced Botrytis, increased Cladosporium, no storage molds, and decreased moisture content. Emergence was significantly decreased for seed with high Botrytis levels at Brandon, Manitoba, and was not improved with seed treatment. No phytotoxic symptoms were observed with 14 seed treatment chemicals in 1970 field trials and, with the possible exception of Manzate 200 at 2.60 and 5.20 g/kg, emergence was not reduced by any of the 10 treatments used in 1971.

Introduction

Buckwheat, Fagopyrum sagittatum Gilib., is an important special crop in Western Canada. The number of hectares sown to buckwheat in Manitoba has increased from 20,250 (50,000 acres) in 1968 to 32,400 (80,000 acres) in 1970 (6), constituting over 50% of the total Canadian crop. Also in 1970, for the first time Saskatchewan and Alberta both grew over 8,100 ha (20,000 acres). Most of the grain is exported to Japan where the flour is used for making noodles, pancakes, and other edible products (1). The hulls are used in the packing industry and for filling pillows. Diseases of buckwheat have not been reported commonly (4,10) and the seed is not treated in Canada. However, reports of wilting in buckwheat at Morden, Manitoba, in 1969 and requests from farmers for suitable fungicides prompted a study of the seed microflora of buckwheat with particular reference to possible pathogens and efficacy of seed treatments. The interrelationships between the microfloral components, storability, and germination was also studied by using naturally damp buckwheat seed.

Materials and methods

Thirty seven lots of buckwheat seed produced in 1969 were received from southern Manitoba in January 1970 (Tables 1,2, and 4). Twelve lots (Table 1, nos. 1 to 12) grown at Carman, Morden, and Winkler were received from Federal Grain Co., and the remainder were from the CDA Research Station, Morden. Lots 1 to 3 and 6 to 10 were of common buckwheat; lots 4, 5, and 11 to 20 were of the cultivar Tokyo, and the remainder of other cultivars (Table 2). Lot 4 had been dried on the farm and lots 17 to 19 were harvested from wilted plots. Moisture contents were determined by AACC method 44-18(2) on duplicate 10-g samples of lots 1 to 12 (Table 1) on arrival. Lots 1 to 12 were then stored in tightly sealed 0.5-liter glass jars, 225 g per jar, in an unheated storage shed; moisture contents were again determined after 390 days. The range in official temperatures, over the storage period 7 January 1970 to 1 February 1971 was -38 to +36 C. Microfloral components present on seed of lots 1 to 37 were determined on receipt (0 days) and for lots 1 to 12 again after 390 days storage. To determine the microflora on the seed a No. 3 Whatman filter paper disc (9 cm) in a petri dish was moistened with 5 ml distilled water and 25 seeds placed on it in a circular pattern near the periphery. There were four replicates each of 25 seeds. The plates were exposed to daylight for 7 days at room temperature (17-24 C) after which the microflora of each seed was examined microscopically. Germination was determined from the same plates after 7 days and the results subjected to an analysis of variance.

The source, formulation, and composition of the 14 seed treatment chemicals used in 1970 are given in Table 3. Each chemical was

[1] Contribution No. 498, Research Station, Canada Department of Agriculture, Winnipeg, Manitoba.

[2] Plant Pathologists.

applied to 200 g of seed at the indicated dosage and shaken well in a 1-liter glass jar. The jars were kept sealed for 2 days to allow the vapor, if any, to act and then lots of 120 seeds were package in envelopes. Envelopes that contained seed from the same treatment were then placed in polyethylene bags and stored at 15 C until seeding 7-8 days later. There were two field tests in 1970 with treated seed; one was to determine possible phytotoxicity (Table 3) by using seed with low (0-3%) Botrytis, content, and the other was to measure control of seed-borne Botrytis sp. infection (Table 4) by using seed with high and low levels of the pathogen. Both tests were sown at Brandon on 5 June 1970. The single-row plots were 3.66 m (12 ft) long, 22.8 cm (9 inches) apart and were replicated four times. One hundred and twenty seeds were sown in each row. The plants were pulled 7 days after seeding, emergence was recorded and results from all replicates were subjected to analysis of variance.

In 1971 there was one field test with treated diseased seed. The variety used, CD 7274, from Morden, had 15% Botrytis infection. The source, formulation and composition of the 10 chemicals used are given in Table 5. Twenty-two days after treatment four replicates of each treatment (200 seeds per row) were sown at Morden and Brandon on 19 and 20 May, respectively, and emergence was determined after 25 days.

Table 1. Microflora, germination, moisture content, and grade of 12 lots of buckwheat seed after 0 and 390 days storage

	% microfloral components* arranged in groups**																	
	Field						Harvest		Storage					Other				
Lot no.	Alt. sp.	Bot. sp.	Clad. sp.	Epi. sp.	Fus. sp.	Gon. sp.	Ceph. sp.	Strep. spp.	Asp. cand.	Asp. vers.	Asp. other	Pen. blue	Pen. other	Rhiz. sp.	Bact.	Germination (%)	Moisture content (%)	Grade†
0 days storage																		
1	89	37	31	3	0	0	0	2	0	0	0	0	0	0	0	82	16.1	T
2	92	5	15	5	0	1	0	7	0	0	0	0	0	0	0	92	15.0	T
3	95	8	19	9	0	0	0	4	0	0	0	0	1	0	0	89	16.9	T
4	73	52	14	1	1	0	3	1	0	0	0	0	0	0	0	86	11.2	S
5	85	14	31	3	1	1	3	9	0	0	0	0	0	0	0	74	16.8	T
6	93	1	17	1	0	0	0	8	0	0	0	0	0	0	0	91	16.1	T
7	93	22	42	3	0	5	6	31	0	0	0	0	0	0	0	68	16.5	T
8	92	30	20	5	0	2	1	8	0	0	0	0	0	0	0	78	18.6	D
9	96	2	24	1	0	0	0	3	0	0	0	0	0	0	0	73	17.9	D
10	95	13	15	0	1	3	0	12	0	0	0	0	0	0	0	80	16.4	T
11	89	34	25	4	0	4	4	6	0	0	0	0	0	0	0	82	16.2	T
12	93	1	23	0	1	1	0	12	0	0	0	0	0	0	0	77	17.0	T
390 days storage																		
1	0-††	0-	0-	0	0	0	1	13+	0	0	0	0	8+	0	20+	26-	17.3	D
2	0-	0	0-	0	0	0	0	2	1	0	0	0	61+	0	1	78	15.7	T
3	0-	0-	0-	0-	0	0	0	67+	1	0	3	0	4	1	17+	24-	17.7	D
4	71	13-	54+	1	0	0	0	2	0	0	0	0	0	0	0	92	9.8	S
5	0-	0-	3-	0	1	0	2	18	0	0	0	1	5	0	0	17-	18.0	D
6	1-	0	0-	0	0	0	0	7	0	0	0	0	1	0	0	69	16.8	T
7	9-	0-	3-	0	6+	0	9	79+	7+	13+	0	0	62+	0	0	0-	17.1	D
8	25-	0-	0-	0	3	0	3	5	69+	35+	0	53+	34+	0	25+	0-	19.4	D
9	0-	0	0-	0	1	0	0	7	0	0	0	0	0	1	0	44-	18.1	D
10	0-	0-	45+	0	0	0	0	34+	1	0	0	1	11+	0	0	55-	16.1	T
11	0-	0-	0-	0	0	0	0	11	0	0	8+	0	7+	2	0	53-	18.2	D
12	0-	0	0-	0	0	0	0	28+	0	0	8+	0	4	0	2	63	18.0	D

* Based on four replicates each of 25 seeds: Alt. = *Alternaria*; Bot. = *Botrytis*; Clad. = *Cladosporium*; Epi. = *Epicoccum*; Fus. = *Fusarium*; Gon. = *Gonatobotrys*; Ceph. = *Cephalosporium*; Strep. = *Streptomyces*; Asp. cand. = *Aspergillus candidus*; Asp. vers. = *A. versicolor*; Asp. other = *Aspergillus*, other species; Pen. blue = *Penicillium*, tall blue species; Pen. other = *Penicillium*, other species; Rhiz. = *Rhizopus*; Bact. = bacteria.

** See text for definitions of groupings of microorganisms associated with seed at various stages.

† Grade: S = standard (<14.8% moisture); T = tough (14.9-17.0% moisture); D = damp (>17.0% moisture).

†† + and - indicates an increase and a decrease ($P < 0.05$), respectively, compared with the corresponding value at 0 days.

Results and discussion

Microflora, germination, and moisture content of untreated seed

Components of the microflora present on untreated seed lots (Table 1 and 2, nos. 1 to 12 and 17 to 37) are listed in groups, namely: "field", "harvest", "storage", and "other". The "field" group includes those fungi that appear whilst the crop is developing in the field (3), and the "storage" groups include those fungi and bacteria that normally become apparent during storage (3). The "harvest" group includes those fungi and bacteria that generally appear before or at time of harvest in the period between the occurrence of "field" and "storage" fungi (7). "Other" microflora are those that are not in the field, harvest, or storage groups but appear when seed is plated on moist filter paper (7). In all lots at 0 days field fungi predominated, particularly Alternaria, Botrytis, and Cladosporium. The harvest microflora consisted of Cephalosporium and Streptomyces, but representatives of storage and other groups were almost entirely absent. Germination on filter paper ranged from 21 to 98% (Tables 1 and 2). After 390 days storage most field fungi had disappeared from all tough and damp samples, but not from the dry sample (lot 4). Streptomyces spp. (harvest), Penicillium spp. (storage), and bacteria (other) greatly increased, except in lots 4, 6, and 9 (Table

Table 2. Microflora and germination of 21 lots of buckwheat from CDA Research Station, Morden, Manitoba

Lot no.	Cultivar	% microfloral components* arranged in groups**													
		Field								Harvest				Other	
		Alt. sp.	Bot. sp.	Clad. sp.	Epi. sp.	Fus. sp.	Gon. sp.	Paec. sp. 1	Paec. sp. 2	Ceph. sp.	Pap. sp.	Strep. spp.	Trich. sp.	Rhiz. sp.	Germination (%)
17	Tokyo	90	1	29	1	2	2	1	1	1	0	16	12	0	82
18	Tokyo	90	1	21	1	0	2	0	0	5	0	11	3	0	79
19	Tokyo	88	1	32	3	0	0	0	0	0	2	55	4	0	75
20	Tokyo	93	4	25	0	0	2	0	0	0	0	9	1	0	94
21	CD 1356-40-3	98	3	33	1	0	1	0	0	3	6	15	2	0	85
22	CD 1356-42-3	97	1	18	2	0	2	0	0	0	1	21	0	0	92
23	CD 1370-61-4	93	0	20	0	0	5	0	0	2	2	12	0	0	87
24	CD 5852	92	1	13	2	1	5	3	1	4	0	21	0	0	78
25	CD 6183	94	4	22	4	0	12	1	0	4	0	15	0	0	82
26	CD 7269	92	1	22	7	0	0	1	0	0	0	3	1	0	93
27	CD 7271	94	0	28	1	0	5	0	0	1	3	20	0	0	80
28	CD 7272	93	12	28	2	0	8	0	2	0	0	37	4	0	88
29	CD 7274	98	0	33	1	0	3	0	0	0	0	10	0	1	89
30	CD 7464	89	7	17	1	0	7	3	2	6	3	28	2	0	85
31	CD 8217	97	1	33	4	0	3	0	0	4	0	15	8	0	85
32	Jap. B + O 61-7	93	1	27	1	0	8	0	0	1	3	8	2	0	94
33	Jap. B + O 61-15	85	3	25	3	0	5	1	1	5	5	45	1	1	21
34	Jap. B + OI R-5	88	5	31	5	0	4	0	0	1	1	8	4	0	93
35	Jap. B + OI R-13	92	1	15	1	0	9	0	0	3	1	21	5	0	88
36	Pennquad	95	3	46	1	0	11	1	3	6	2	19	21	0	64
37	Silverhall 24	95	4	35	2	0	7	0	1	4	3	22	1	0	58

* Based on four replicates each of 25 seeds; Paec. sp. 1 = *Paecilomyces* (large spores); Paec. sp. 2 = *Paecilomyces* (small spores); Pap. = *Papularia;* for other abbreviations see footnote to Table 1.

** See text for definitions.

1). The exceptions were apparently due to dry seed (lot 4), to a low proportion of unsplit hulls (lot 6), or to unknown factors (lot 9). Germination of lots 1 to 12 after 390 days storage ranged from 0 to 92%; with the exception of lot 4, percentage germination had decreased from the values at 0 days. In lots 7 and 8 the decrease was from 68% and 78% to 0, respectively. These were the only samples infested with Aspergillus candidus Link and A. versicolor (Vuill.) Tiraboschi. Most split hulls occurred in lot 1 and the least in lot 6. Over the 390-day period moisture contents in most lots increased, probably due to respiration of the microflora and of the grain itself; exceptions were lots 4 and 10. The maximum moisture increase, 2%, occurred in lot 11.

Efficacy of seed treatments on diseased seed

At Brandon in 1970, emergence of untreated seed with low levels of Botrytis sp. ranged from 76 to 82%; for seed with high levels of Botrytis emergence ranged from 67% to 76% (Table 4.) Emergence of the four lots of lightly infested seed was significantly greater than that of infested lots, but it was not increased by seed treatment. Emergence in the test using lightly (3%) infested treated seed (Table 3) was 87% in the untreated control and among the fungicide treated lots ranged from 78 to 92%. There were no differences in emergence between treatments ($P < 0.05$) and no phytotoxicity was apparent. Germination of seed treated with Panogen PX, Manzate D, or Arasan 75 was not reduced in the laboratory on filter paper (8).

Emergence in the 1971 field experiment from seed with medium (15%) infection was 53% in the control and according to treatment ranged from 48 to 56% (Table 5). No phytotoxic symptoms were apparent even at the rate of 8.60 g/kg of Benlate T. Manzate 200 at both 2.60 and 5.20 g/kg rates was associated with reduced emergence ($P < 0.05$).

Microfloral components present on freshly harvested buckwheat are predominantly field fungi, similar to those found on wheat, barley, and oats of the same age. However, Botrytis sp., a possible pathogen, occurs on buckwheat but rarely on cereals (5). Botrytis sp. has not been recorded on buckwheat previously (4, 10) in Canada or the U.S.A. Damp and tough buckwheat in sealed storage deteriorates, moisture and storage fungi increase, and, as with cereals, viability falls (9). If the buckwheat is dried to a lower moisture level, as the 11.2% in lot 4, viability is maintained and storage fungi are not apparent. In field tests, compared with lightly infested seed, heavy infestation with Botrytis sp. reduced emergence ($P < 0.05$), and emergence was not improved by seed treatment. The fungicides, with the possible exception of Manzate 200 in the 1971 trial, showed no evidence of adversely affecting emergence of buckwheat.

Table 3. Seed treatment materials, dosages, and emergence of buckwheat in 1970 field trial

Product name	Source*	Formulation**	Chemical name	Dosage (g product/kg)	Mean emergence (%)
Untreated					86.9
Arasan 42-S	Dupont	SL	thiram 42%	2.60	91.5
Arasan 70-S	Dupont	SL	thiram 70.0% + methoxychlor 2.0%	1.69	83.4
Arasan 75	Dupont	D	thiram 75.0%	1.69	89.6
Ceresan M	Dupont	D	ethyl mercury p-toluene sulfon-	0.65	89.8
Ceresan M	Dupont	D	anilide 7.7%	1.30	84.8
Manzate D	Dupont	D	maneb 80.0%	1.30	84.8
Manzate D	Dupont	D	maneb 80.0%	2.60	87.3
Manzate 200	Dupont	D	mancozeb (coordination product of zinc ion and maneb) 80.0%	2.60	84.7
Res-Q	Green Cross	D	hexachlorobenzene 20.0% + captan 20% + maneb 15.0%	1.30	88.6
Hoe 2981	Hoechst	WP	identity not available	1.30	86.1
Hoe 2981	Hoechst	WP	identity not available	2.60	79.8
TCMTB	Interprov.	D	2-(thiocyanomethylthio) benzo-thiazole 10.0%	3.12	84.7
Polyram 53.5	Niagara	D	zinc activated polyethylene thiuram	1.30	88.6
Polyram 53.5	Niagara	D	disulfide 80.0%	2.60	86.3
BEJ 15	Niagara	L	identity not available	2.60	78.4
Panogen PX	Nor-Am	D	methylmercuric dicyandiamide 0.9%	2.60	88.4
Panogen 15B	Nor-Am	L	methylmercuric dicyandiamide 3.7 oz/gal	0.98	86.5
Captan 50WP	Stauffer	WP	captan 50.0%	1.30	87.2
Captan 50WP	Stauffer	WP	captan 50.0%	2.60	87.2

* E. I. Dupont de Nemours & Co., Inc., Wilmington, Delaware; Green Cross Products, Division of CIBA Co. Ltd., Montréal, Québec; Hoechst Chemical Co., Montréal, Québec; Interprovincial Cooperatives Ltd., Winnipeg, Manitoba; Niagara Brand Chemicals, Burlington, Ontario; Nor-Am Agricultural Products Ltd., Woodstock, Illinois; Stauffer Chemical Co. of Canada Ltd., Montréal, Québec.

** D = dust, WP = wettable powder, L = liquid, SL = slurry.

Table 4. Effect of seed treatments on emergence* of buckwheat from seed infested with low and high levels of *Botrytis* sp.; 1970 field trial

Sample no.**	Botrytis (%)	Treatment†				Sample mean	Group mean††
		Check	Res-Q	Panogen PX	Arasan 75		
14	1	79.4	77.2	74.8	72.6	76.0	
13	2	75.9	75.5	80.1	76.5	77.0	
15	3	75.9	80.1	78.0	81.1	78.8	
16	3	81.9	77.8	80.2	74.0	78.5	77.6
8	30	71.1	69.9	71.5	74.0	71.6	
11	34	71.4	74.7	76.1	75.5	74.4	
1	37	67.4	69.7	72.2	62.2	67.9	
4	52	75.5	71.8	72.2	76.3	73.9	72.0

* Means of 4 replicates.

** Sample nos. 4, 11, and 13 to 16 were of the cultivar Tokyo; nos. 1 and 8, common buckwheat.

† Dosages as Table 3.

†† Avg of sample means for samples having low and high levels of *Botrytis* infestation.

Table 5. Seed treatment materials, dosages, and emergence of buckwheat in the 1971 field trial

Product name	Source*	Formulation**	Chemical name	Dosage (g product/kg)	Mean† emergence (%)
Untreated					52.8
Agrox NM	Chipman	D	37.5% maneb + 10.0% hexachlorobenzene	2.60	50.8
Agrox NM	Chipman	D	37.5% maneb + 10.0% hexachlorobenzene	5.20	50.6
Arasan 75	Dupont	D	75.0% thiram	1.70	54.5
Arasan 75	Dupont	D	75.0% thiram	3.40	53.7
Benlate	Dupont	D	50.0% methyl 1-(butylcarbamoyl)-2-	2.60	53.5
Benlate	Dupont	D	benzimidazole carbamate	5.20	52.4
Benlate T	Dupont	D	Benlate + thiram	4.30	55.1
Benlate T	Dupont	D	Benlate + thiram	8.60	51.4
Manzate D	Dupont	D	80.0% maneb	2.60	52.5
Manzate D	Dupont	D	80.0% maneb	5.20	54.9
Manzate 200	Dupont	D	80.0% mancozeb (coordination product	2.60	48.4-
Manzate 200	Dupont	D	of zinc ion and maneb)	5.20	48.9-
Panogen PX	Nor-Am	D	0.9% methylmercuric dicyandiamide	2.60	56.3
Panogen PX	Nor-Am	D	0.9% methylmercuric dicyandiamide	5.20	54.0
Captan 50	Stauffer	WP	50.0% captan	2.60	53.1
Vitavax 75	Uniroyal	D	75.0% 5,6-dihydro-2-methyl-1,4-oxathiin-	1.75	54.5
Vitavax 75	Uniroyal	D	3-carboxanilide	3.50	51.0
Vitaflo DB	Uniroyal	D	Vitavax 40% W/W + thiram 40% W/W	3.30	51.6
Vitaflo DB	Uniroyal	D	Vitavax 40% W/W + thiram 40% W/W	6.60	52.5
LSD (0.05)					3.7

* Chipman Chemicals Ltd., Hamilton, Ontario; E. I. Dupont de Nemours & Co., Inc., Wilmington, Delaware; Nor-Am Agricultural Products Ltd., Woodstock, Illinois; Stauffer Chemical Co. of Canada Ltd., Montréal, Québec; Uniroyal Ltd., Elmira, Ontario.

** D = dust, WP = wettable powder.

† - indicates a significant decrease compared with the control at the 0.05 level.

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BARLEY STRIPE MOSAIC VIRUS IN MANITOBA IN 1971[1]

Arthur W. Chiko

Abstract

In 1971, barley stripe mosaic virus was detected by infectivity and serological assays in 34.0% and 4.5%, respectively, of the 2- and 6-row barley fields surveyed in southeastern Manitoba. The incidence of plants with barley stripe mosaic symptoms in these fields varied from a trace to about 20%. At present, the predominant source of this virus in Manitoba is probably infected seed of the 2-row barley variety 'Herta'.

Introduction

In 1970, barley stripe mosaic virus (BSMV) was detected in 22% and 4%, respectively, of the fields of 2-row barley (Hordeum distichum L. emend. Lam.) and 6-row barley (H. vulgare L. emend. Lam.) surveyed in southern Manitoba (3). A survey was conducted in Manitoba in 1971 with the objectives of intensifying the search for BSMV in 2-row barley, determining if the virus was again more prevalent in 2-row than in 6-row barley, and identifying the most commonly infected variety or varieties.

Materials and methods

The 1971 survey for BSMV, conducted from July 5 to 15, was confined to southeastern Manitoba, since about 65% of Manitoba's 1970 2-row barley crop was grown in this region (1). Fields of 2- and 6-row barley in the early tillering to milky ripe stage were inspected at intervals of about 5 and 15 miles, respectively, along eight preselected survey routes totalling about 1400 miles. Fields were sometimes inspected at shorter or longer intervals for reasons discussed previously (3).

A sample was collected in each field where plants with suspected symptoms of barley stripe mosaic (BSM) were detected. Each sample consisted of 2-4 apical leaves from one tiller of each of three plants with symptoms. The day after collection, each sample was ground in a sterile mortar with 1 ml of distilled water, and a group of 14-16 'Black Hulless' barley test plants in the 2-leaf stage was inoculated with the crude extract. Seven to 10 days after inoculation, the presence or absence of symptoms was recorded and the third leaf was detached from each of three diseased test plants in each group. The three leaves were combined and juice was extracted with a plier-type press. Each sample of juice was tested undiluted against undiluted BSMV antiserum using the Ouchterlony double diffusion method (4) and the final results of this test were recorded 1 week later. The diffusion medium was prepared with 0.5% Ionagar No. 2 and 0.2% sodium azide in distilled water. Procedures for storing field-collected leaf samples and for growing and inoculating test plants were similar to those described previously (3). The BSMV antiserum was the same as that prepared and used by the author in 1970 (3).

Results and discussion

The presence of BSMV in each field-collected leaf sample was demonstrated by transmission of the virus to 'Black Hulless' barley and by its subsequent reaction with BSMV antiserum. Extract from healthy 'Black Hulless' plants (control) did not react with this antiserum.

BSMV was detected in 49 of 144 (34.0%) fields of 2-row barley and in 2 of 44 (4.5%) fields of 6-row barley surveyed. The incidence of plants with BSM symptoms in both fields of 6-row barley was a trace, whereas in fields of 2-row barley the approximate incidence was a trace in 40 fields, 1-2% in 6 fields, 5% in 2 fields, and 20% in 1 field. BSMV was also detected in a 2-row selection and in the 6-row variety 'Keystone' in barley breeding plots near Winnipeg. The incidence of diseased plants was about 1% and 2%, respectively.

Symptoms of BSM in 2-row barley were generally more pronounced in 1971 than in 1970. Brown stripes, sometimes present in the form of a V or W, were generally observed on lower leaves of diseased plants. Mosaic symptoms, which were rarely detected in 1970, were frequently observed on upper leaves. In a few fields, however, faint brown stripes on lower leaves were the only apparent symptoms of infection.

[1] Contribution No. 501, Research Station, Canada Department of Agriculture, Winnipeg, Manitoba R3T 2M9.

Although the percentage of 2-row barley fields in which BSMV was detected was 12% higher in 1971 than in 1970 (3), this difference is not considered to be indicative of the rate of virus spread. It was probably due mainly to the detection of more trace infections in 1971, because of either more pronounced symptom development or a more intense and protracted search for BSM in this type of barley, or both. Some trace infections, however, were detected only after extensive searching and, therefore, the 1971 estimate for the percentage of 2-row barley fields with BSMV-infected plants may still be conservative.

'Herta', a variety licensed for sale in Canada in 1956, comprised at least 94% of the 2-row barley acreage in Manitoba in 1970 (1) and about 73% in 1971 (2). It thus seems probable that in 1970 this was the variety most frequently infected with BSMV. In 1971, growers of 15 of 49 (30.6%) of the 2-row barley fields known to contain BSMV-infected plants were personally interviewed and each reported that the infected variety was 'Herta'. Although the possibility of other 2-row barley varieties being infected with BSMV cannot be discounted, there can be little, if any, doubt that at present the predominant source of the virus in Manitoba is infected seed of 'Herta' barley. No evidence has been obtained that 'Fergus', a variety licensed in 1968 and grown on about 22% of the 2-row barley acreage in Manitoba in 1971 (2), has become infected with BSMV under natural conditions.

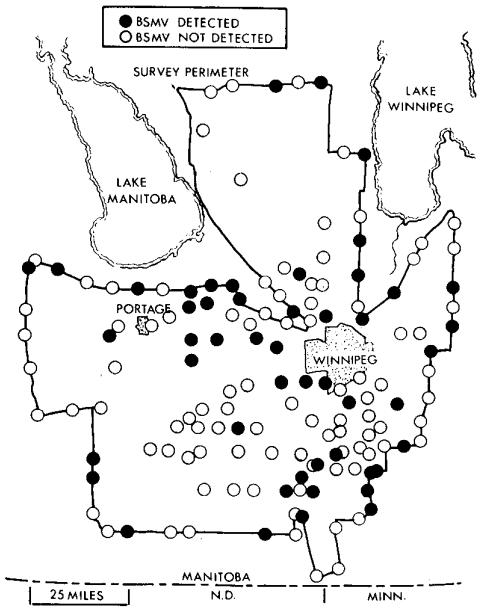

Figure 1. Distribution of barley stripe mosaic virus in fields of 2-row barley in southeastern Manitoba in 1971.

BSMV was present in 2-row barley fields throughout the region surveyed, but it appeared to be more common in some areas than in others (Fig. 1). This may indicate that some seed sources of 'Herta' are almost or completely free of the virus or, alternatively, that 'Fergus' was the predominant 2-row barley variety in areas where BSM was not detected.

In greenhouse (3) and field (Chiko, unpublished) tests, 'Herta' and 'Fergus' barley appeared to be equally susceptible to BSMV. Therefore, even if 'Fergus' or another susceptible 2-row variety eventually replaces 'Herta', the virus could become a serious problem in the production of 2-row barley in Manitoba.

Acknowledgments

I am grateful to Mr. B. E. Halstead for providing technical assistance and to Mr. R. J. Cheale for preparing the illustration.

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EFFECT OF PLANTVAX EMULSIFIABLE CONCENTRATE ON STEM AND CROWN RUSTS OF OATS[1]

J.W. Martens, G. Fleischmann, and R.I.H. McKenzie

Abstract

Plantvax E. C. failed to provide effective control of either crown rust or stem rust in oat plots artificially inoculated with these rusts.

The periodic shortage of genetic resistance to stem rust, Puccinia graminis Pers. f. sp. avenae Eriks. and E. Henn., and crown rust, P. coronata Cda. f. sp. avenae Eriks., in oats (Avena sativa L.) had led to the search for chemicals that can be used as stopgap control measures until resistant varieties are developed. Maneb has proven highly effective in the control of both rusts (Fleischmann et al. 1968), but it has poor tenacity and it is subject to weathering effects and so must be applied several times during the growing season, particularly after rains. The systemic oxathiin derivatives showed some promise of controlling the wheat leaf and stem rusts (Hagborg 1970; Rowell 1967, 1968) but field trials on oat rusts (Hagborg, unpublished data) with the original formulation were disappointing. Hagborg (1971) found that a more water soluble emulsifiable concentrate (E.C.) formulation of Plantvax was more effective against wheat rusts, and further tests with oat rusts seemed desirable.

Materials and methods

The oat cultivar Eagle was planted on June 3, 1970, in a randomized 8-replicate experiment; each plot consisted of 4 rows 5.6 m long, spaced 30 cm apart. Four buffer rows of the cultivar Pendek were planted between plots. The plants were inoculated at the 5-6 leaf stage by injecting 1 or 2 plants at each end of each buffer row with a water suspension containing urediospores of both stem rust (race 1) and crown rust (races 264, 295, and 326). The test plots of Eagle were infected by secondary spread of rust from the artificially inoculated buffer rows1 between plots. Dithane M22 (maneb, 80% w/w; Rohm & Haas Co. of Canada Ltd., West Hill, Ont.) was applied weekly or after heavy rains at the rate of 3 liters of 0.33% active solution (w/v) per plot for each application. Plantvax E.C. (5,6-dihydro-2-methyl-1,4-oxathiin-3-carboxanilide-4, 4-dioxide, 12% w/v, Uniroyal (1966) Ltd., Chemical Division, Elmira, Ontario) was applied twice, 10 and 24 days after inoculation, at the rate of 3 liters of 0.1% active emulsion (w/v) per plot for each application. The two center rows of each plot were harvested for yield and quality determinations.

Results

The five maneb treatments gave almost complete control of both stem rust and crown rust (Table 1). Severe infections of both rusts developed in both the control and the Plantvax E.C. treated plots, although the latter showed slight retardation of rust development. The Plantvax E.C. treatment also increased the yield and quality measurements slightly, but the benefits did not economically justrify the effort and expense of treatment, particularly in view of the heavy rates of chemical used. Chemical control of rusts in commercial oat fields must await the development of a more effective compound.

Table 1. Effect of Plantvax E.C. and Dithane M-22 on rust infection, and on yield and quality of oats inoculated with stem rust and crown rust

Category	Control	Plantvax E.C.	Dithane M-22
Stem rust			
% infection			
August 10	67.5	51.3	0
August 19	95.0	90.0	0
Crown rust			
% infection			
August 10	57.5	37.5	0
August 19	72.5	51.3	tr
Yield (kg/ha)	1592	2057	3687
% loss	56.8	44.2	
Thousand kernel wt (g)	17.0	17.1	28.4
% reduction	40.2	39.8	
Liter weight (g)	340	381	494
% reduction	31.2	22.9	
Hull content (%)	38.7	34.7	23.9
% increase	61.9	45.2	

[1] Contribution No. 496, Research Station, Canada Department of Agriculture, Winnipeg, Manitoba R3T 2M9.

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AERIAL PHOTOGRAPHY AS A SURVEY TECHNIQUE FOR THE ASSESSMENT OF BACTERIAL BLIGHT OF FIELD BEANS[1]

V.R. Wallen and H.R. Jackson [2]

Abstract

Aerial infrared false-color photographs were used to assess the incidence of seed-borne bacterial blight caused by Xanthomonas phaseoli in 34 bean (Phaseolus vulgaris) fields in southwestern Ontario in 1968. The percentage of the crop area affected by blight was determined from the photographs by a scanning technique. Techniques for separating the colors in the 3-layer emulsion and reproducing them as separate black and white positives are discussed and illustrated. In some fields up to twice the number of infected plants were detected by infrared photography as were found by ground surveys carried out the same day. The incidence of blight was correlated with the pedigree of the seed used in planting the fields, ranging from zero in many fields of Breeder seed to more than 50% in some fields of Commercial seed.

Introduction

The detection of plant diseases by aerial photography has been attempted by a number of investigators and shows promise as a technique for surveys and for studies on epiphytology (1,2,3,4,5). Most of this work has involved the photography of experimental field plots in an attempt to correlate photographic patterns of disease with ground truth observations.

Conventional ground surveys for plant diseases, as part of a national crop disease loss program, are carried out under the limitations of the manpower available, and they provide estimates subject primarily to the expertise of the individual and the methods used in sampling the crop and assessing the disease. Aerial photography would solve an important problem by providing a survey technique that could cover vast areas in a short time and visibly portray affected areas.

In Canada, approximately 98% of the white bean (Phaseolus vulgaris L.) crop is grown in southwestern Ontario. Breeder seed for this crop is usually grown in California and Idaho, where arid conditions should assure freedom from bacterial blight caused by Xanthomonas phaseoli (E.F. Sm.) Dows. Breeder seed is distributed by the Canadian Seed Growers Association to approximately 25 growers who produce approximately 1 to 2 acres each of Select beans. This crop is inspected twice during the growing season by officers of the CDA Plant Products Division and the plot is given Select status if it is free from bacterial blight. Select seed is then used to produce approximately 1,000 acres of Foundation, followed by approximately 5,000 acres of Certified seed. Despite stringent regulations and inspections, bacterial blight is often present in Breeder seed and the levels usually increase during production of the other seed grades; in most years commercial crops, which are produced from Certified seed, are infected to varying degrees.

Because the disease produces small foci of infection that orginate from infected seed, bacterial blight of field bean was selected as a model for studies on the feasibility of using aerial photography for plant disease detection. Another important consideration in choosing the white bean crop is that at the time of inspection the plants provide a complete canopy of foliage with no visible ground showing on the photographs to interfere with disease interpretation.

The optimum time for photography occurs within a 3 to 5 day period when the plants have reached maximum growth and chlorophyll content. For white beans in southwestern Ontario this is usually between August 12 and 17 in the Hensall area and slightly later in the Chatham area. At this time of year chlorophyll content reaches a maximum and a sharp contrast between healthy and diseased tissues can be noted. Soon afterward senescence of the crop begins and differentiation of blight symptoms becomes difficult. Because little secondary spread of the pathogen occurs before August 12, initial seed-borne infections can be located and the disease condition of the original seed determined.

[1] Contribution No. 299, Ottawa Research Station, Canada Department of Agriculture, Ottawa, Ontario, K1A 0C6.

[2] Ottawa Research Station and Scientific Information Section, Research Branch, Canada Department of Agriculture, Ottawa, Ontario.

Materials and methods

Kodak Ektachrome Infrared Aero Film 8443 was used in conjunction with a yellow (Zeiss B) filter and processed as a positive from which reversal prints were made. A Zeiss camera having a 12 inch focal length lens was used throughout the study and exposure was f. 5.6 at 1/300 second. The altitudes of flight were 4,350 feet and 9,150 feet to produce scales of 1:3600 and 1:8400 respectively (6). In the flight area a total of 809.93 acres, including 34 bean fields ranging in size from 2.4 to 80.52 acres, were photographed.

To derive the percentage infection in each field after disease interpretations had been made from the photographs, we used a drum scanner method (7) which measures, depending on the photographic scale, areas of the film corresponding to field areas of from 1.4 to 7.8 sq ft. The areas of diseased and healthy foliage in each field were then recorded on magnetic tape and the percentage area affected by blight calculated. The IR photos were taken August 12, 1968, and ground surveys of the fields photographed were made during the period July 28 to August 19.

Results and discussion

The area of study near the town of Hensall, Ontario, is illustrated in Figure 1 (see map, inside back cover). In this 7 sq mile area the chief agricultural crops are white beans, cereals, and corn. In Figure 1, the map (left) shows the location and acreage of the bean fields examined and includes the percentage of plants affected by bacterial blight in each field as determined from the infrared (IR) photographs; on the right is a black and white translation reduced from the original false color photographs of the area taken at a scale of 1/8400. These photographs have been assembled and matched for "best fit" and are a pictorial representation only of the terrain; measurements should not be taken from them. Of the 34 fields in the flight path, 26 were infected. The highest infection in any field was 52.10%. The total area affected was 33.5 acres or 4.1% of the crop.

The black and white translations referred to should not be compared with monochrome copies from "normal" color prints or transparencies because the color values are not represented by gray levels to which we are accustomed. Furthermore this should not be confused with original panchromatic aerial photography. The value of false color images lies in the fact that reflected infrared radiation is recorded by a layer in the emulsion sensitized to wavelengths between 0.7 and 0.9μ. To incorporate the IR-sensitive layer and maintain a three-layer system, the blue wavelengths, which can be image-degrading, are eliminated by filtration during the original exposure. Therefore green, red, and infrared radiation is reproduced as blue, green, and red, respectively, hence the term "false color", and it is not really significant that high IR reflection is reproduced as "red" image. Normal color emulsions reproduce a scene containing blue, green, and red in the same colors that produced them during exposure. Although a black and white reproduction represents blue, green, and red in a false color original, the silver densities in the copy should not be evaluated in the same way as copies from normal color because the colors in the IR film used are not true to those in the original scene. However, the color separation procedure uses filters to isolate those colors contained in the three layers of the emulsion and reproduces them as separate black and white negatives (Figures 2 and 3). Blue, green, and red in false color transparencies and prints are produced by yellow, magenta, and cyan dye-layers which are formed during processing.

In any positive color material, the yellow dye absorbs blue from white light, magenta absorbs green, and cyan absorbs red. This system produces the color red in those areas of high IR reflection because the density of the cyan layer is inversely proportional to the exposure that produced it. In other words, high infrared reflection results in a low dye concentration in that sensitivity layer. For example, when the transparency is viewed, the yellow and magenta dyes in the green and red sensitivity layers absorb blue and green, allowing the remaining component of white light - red - to be seen. Red is absorbed by cyan but because it is of low concentration its filtering or absorbing capability is reduced.

To separate the dye layers, the transmitted light from transparencies is recorded separately on continuous-tone black and white emulsions through blue, green, and red filters, thus producing silver densities that can be related to the original color and its position in each of the three sensitivity layers.

At this stage in development of the technique, it is necessary to conduct extensive ground truth studies to identify certain characteristics on the photographs. However, we have found it necessary in many cases to return and reassess fields after seeing the photographs because the photography showed more disease foci than were originally noted visually in the field. In one field (Figure 2), only about 50% of the foci shown by the IR film were detected by ground truth observations made on the day the photographs were taken. Two to three days after the photographs were taken, additional disease foci appeared which correlated with the foci indicated on the photographs. It has been postulated that IR photography detects chlorophyll breakdown in the mesophyll tissues prior to the appearance of visible symptoms of infection (2). In

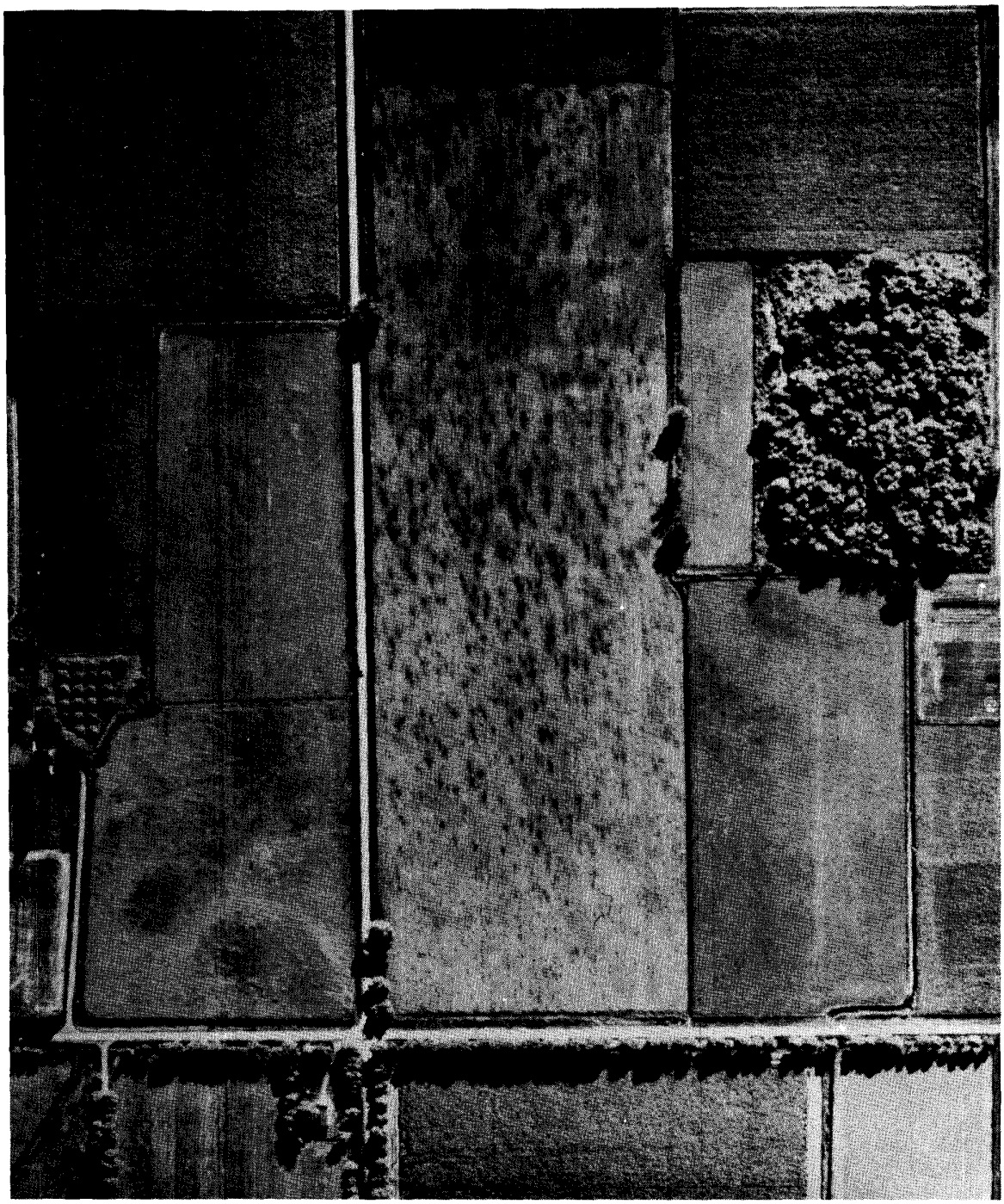

Figure 2. Enlarged black-and-white continuous tone separation from Kodak Ektachrome Infrared Aero film 8443 false color positive print. This reproduction shows only the red filter separation of the cyan dye layer. Dark foci in the center field indicate chlorophyll breakdown in bean plants affected by bacterial blight. Approximate scale: 1/2930.

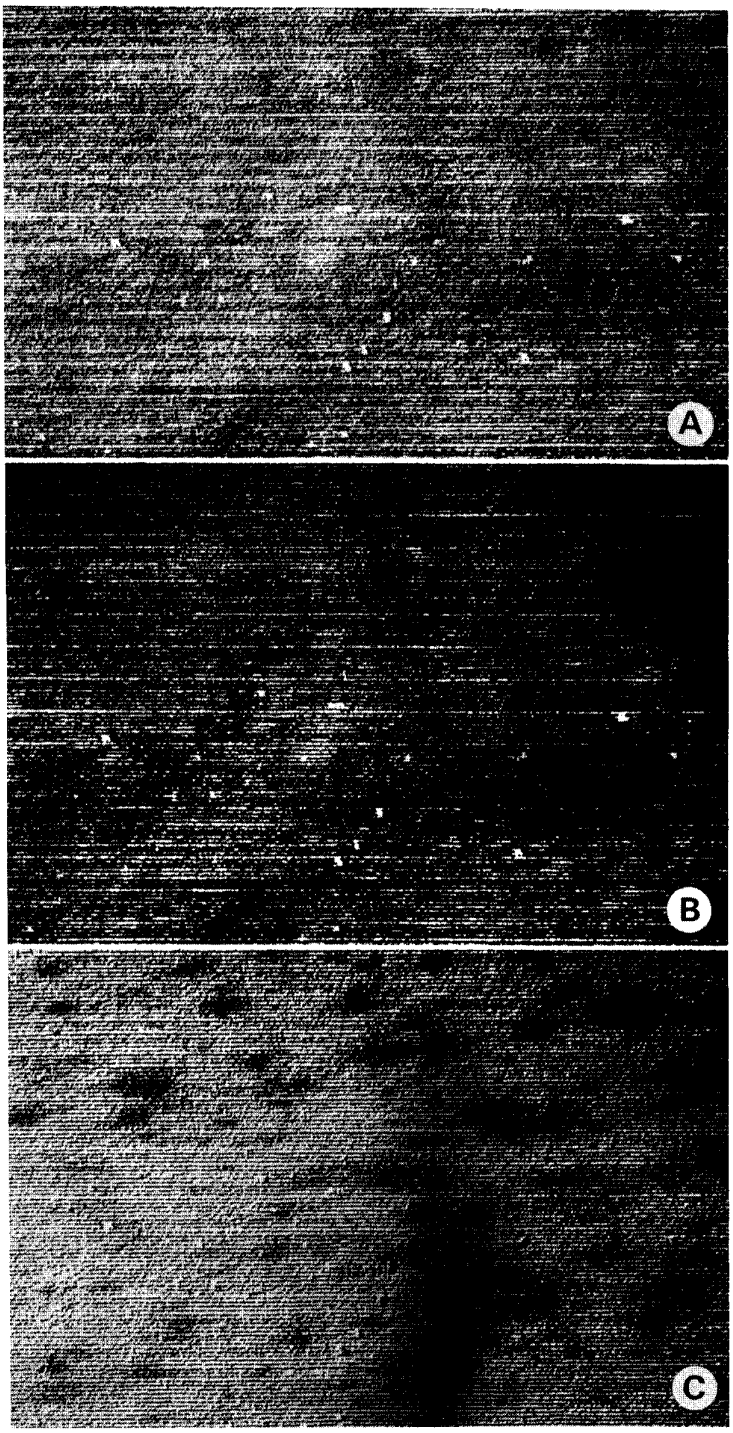

Figure 3. Enlargement of a portion of the same infected field as in Figure 2 but taken from an original camera-exposed false color transparency showing information contained in each sensitivity layer when separated by filters: A) blue, Wratten 47; B) green, Wratten 58; and C) red, Wratten 25. Note at this magnification that individual rows and plants are readily observed. Approximate scale: 1/960.

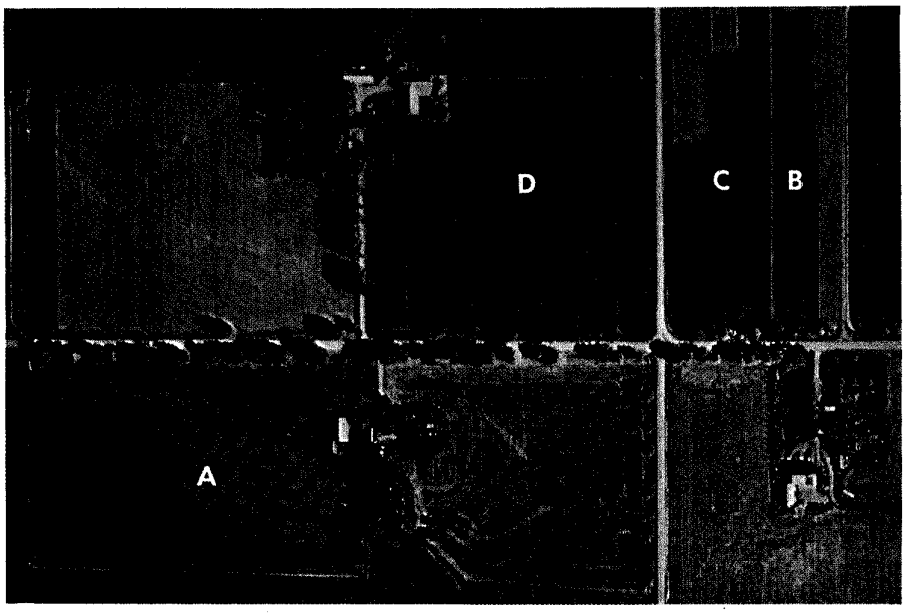

Figure 4. Three-color reproduction from Kodak Ektachrome Infrared Aero film 8443 false color, photographed at an original scale of 1/8400. The photograph depicts four bean fields originating from seed of different pedigree: A) non-pedigreed seed; B) Breeder seed; C) and D) Select seed. The numerous dark areas in A) represent foci of bacterial blight. Approximate scale: 1/7635.

this particular field, it was noted (Figure 2) that fewer foci were present in the area near the crossroads. This area is 2 to 4 ft higher than other areas of the field. Morning dews and fog dissipate earlier in this area and conditions ark not as favorable for development of bacterial blight as in other areas of the field.

In this survey by aerial photography, it was possible to ascertain on a single day the amount of infection arising from seed-borne inoculum in the 34 fields. By conventional means the survey would have taken one man at least 2 weeks and the results would have provided only an estimate based upon sampling. By aerial photography the entire crop was visibly displayed.

From the seed growers' standpoint, this technique displays the importance of using disease-free seed. Figure 4 depicts several fields of various seed pedigree: A) a field that was sown with non-pedigreed seed that was heavily infected with bacterial blight; the seed had been multiplied on this farm for 5 years; B) a Select plot, disease-free; C) and D) Foundation fields, also disease-free, produced from the previous year's Select plot.

It is apparent that in order to use IR aerial photography successfully in its present state of development a number of conditions of crop and disease must be present. The crop must form as complete a chlorophyll canopy as possible so any breakdown in chlorophyll can be detected as a lack of IR reflectance. Large or numerous patches of exposed soil in a field tend to interfere with disease interpretation because of a merging of soil and disease patterns. Also the disease must be present on the leaves photographed because diseased leaves that are covered by an upper canopy of healthy green foliage cannot be detected. Crops such as field beans, corn, and potatoes provide the necessary canopy in mid-to-late season.

Acknowledgment

The authors thank the members of the Cartography Section, Soil Research Institute, Canada Department of Agriculture for assistance in preparing Figure 1.

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CONTROL OF TYPHULA SNOW MOLD ON COLD-STORED STRAWBERRY RUNNER PLANTS[1]

C.L. Lockhart

Abstract

Demosan (chloroneb) was the only fungicide of nine tested that gave satisfactory control of mold in cold-stored strawberry plants that had been inoculated with Typhula sp. Captan, quintozene, and two mercury-containing fungicides also controlled the mold but, unlike Demosan, were phytotoxic at the rates used.

Introduction

Losses of cold-stored strawberry plants due to a Typhula sp. have been reported in Nova Scotia (1, 2). Average plant losses are generally less than 2%, but nursery growers have lost up to 26% of their cold-stored plants (2).

The effect of several post-harvest fungicide treatments on control of Typhula sp. in cold-stored plants and on plant survival are reported in this paper.

Materials and methods

Strawberry plants, cultivar Redcoat, cleaned and ready for storage were obtained from a commercial nursery. Nine fungicides either alone or in combination were used in these tests. The name, active ingredient, formulation, and source of each of the fungicides are as follows:

Bay 33172, 2-(2 furyl)-benzimidazole 50%, WP. Chemagro Corporation, Kansas City, Missouri.

Daconil 2787, tetrachloroisophthalonil 75%, W.P. Diamond Alkali Co., Cleveland, Ohio.

Benlate, benomyl [methyl 1-(butyl carbamoyl)-2 benzimidazolecarbamate] 50%, W.P. Dupont of Canada Ltd., Toronto, Ontario.

Demosan, chloroneb (1, 4-dichloro-2,5-dimethoxy-benzene) 65%, W.P. Dupont of Canada Ltd., Toronto, Ontario.

Thylate, thiram 65%, W.P. Dupont of Canada Ltd., Toronto, Ontario.

Puratized Agricultural Spray, phenylmercury triethanol-ammonium lactate 7.5%, Sn. Gallowhur Chemicals Canada Ltd., Montreal, Quebec.

Erad Eradicant Fungicide, phenylmercuric acetate 10%, Sn. Green Cross Products, CIBA Co. Ltd., Montreal, Quebec.

Captan, captan 50%, WP. Green Cross Products, CIBA Co. Ltd., Montreal, Quebec.

Terraclor, quintozene 75%, WP. Olin Agricultural Division, Little Rock, Arkansas.

The fungicides used as dusts were prepared by diluting the wettable powder products with talc to obtain the concentration of active ingredient listed in Table 1.

Eight bundles of 25 plants were dipped or dusted with each fungicide material at the rates shown in Table 1. For each dip treatment, the bundles of plants were dipped in the fungicide for 1 minute and the excess liquid was allowed to drain off before inoculating with Typhula. For the dust treatments, each bundle of plants was opened up and the dust was applied using a jar with a perforated cover. Four bundles of plants from each treatment were then inoculated b- placing 25 g of pulverized cornmeal-sand cultures of Typhula around the crowns and tops of the roots. The remaining four bundles of plants from each treatment were not inoculated. Controls consisted of water-dipped or untreated plants. The plants were stored at -1.1 C in polyethylene bags tied at the top with a wire tag. After 6 months the plants were removed from storage and observations on mold development were recorded as % plants affected. A sample of 25 plants from most treatments was planted in soil in the greenhouse and observations on appearance and survival were recorded.

[1] Contribution No. 1375, Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

Table 1. Effect of post-harvest fungicide treatments on the development of mold on *Typhula*-inoculated strawberry plants and on the survival and growth of the plants in the greenhouse following storage for 6 months at -1.1 C

Fungicide	Rate (lb formulation/ 100 gal)	Post-harvest treatment	Mold (%)	Survival* (%) I	Survival* (%) NI	Growth of plants** I	Growth of plants** NI
Bay 33172	2	Dip	100				
Bay 33172	4	Dip	100				
Benlate	3	Dip	100				
Benlate 12.5% active		Dust	100	88	100	3	4
Captan	2	Dip	100				
Captan	4	Dip	Trace†	80	96	2	2
Captan 7.5% active		Dust	100	52	100	2	4
Daconil	1	Dip	100				
Daconil	2	Dip	100				
Daconil	4	Dip	100				
Daconil	8	Dip	100				
Demosan	2	Dip	25	100	100	5	5
Demosan	4	Dip	0	100	100	5	5
Demosan 8.4% active		Dust	7	100	100	5	5
Puratized Agr. Spray	1/2 pt	Dip	Trace†	84	100	3	3
Puratized Agr. Spray	1 pt	Dip	0	88	100	3	3
Erad	1/2 pt	Dip	0	0	0	0	0
Terraclor	4	Dip	7.5	100	100	2	2
Terraclor	8	Dip	Trace	100	100	1	1
Terraclor + Captan	2 + 2	Dip	10	100	100	3	4
Terraclor + Captan	4 + 4	Dip	Trace	100	100	2	3
Terraclor + Captan	8 + 8	Dip	Trace	100	100	1	1
Thylate	2	Dip	100	100	100	3	4
Thylate 7.5% active		Dust	100	88	100	3	4
Control		Water dip	100	52	100	1	4
Control		None	100	72	100	2	5

* I indicates inoculated with *Typhula* and NI, not inoculated.

** Rating of plant growth: 0 = no growth, 1 = very poor, 2 = poor, 3 = fair, 4 = good, 5 = excellent. Absence of a rating for survival or growth indicates that because of extensive mold development the plants were not included in the greenhouse test.

† Roots dark when removed from storage.

Demosan was the only fungicide that gave satisfactory control of Typhula in inoculated cold-stored strawberry plants and had no phytotoxic effects (Table 1). Demosan was most effective for mold control at 4 lb per 100 gal. Although there was 25% mold on plants dipped in Demosan at 2 lb per 100 gal and 7% mold on plants dusted, these plants showed 100% survival and excellent growth in the greenhouse. Captan at the 4 lb rate, Puratized Agricultural Spray, Erad, and Terraclor controlled Typhula but were phytotoxic. Captan at the 4 lb rate caused the roots to become black, while the mercury fungicides and Terraclor retarded plant growth. Bay 33172, Benlate, Daconil, and thiram did not control Typhula.

Both the dip and dust treatments with Demosan gave satisfactory control of Typhula mold on cold-stored strawberry runner plants, but this fungicide is not registered in Canada for use on strawberry plants.

Acknowledgment

The author thanks G. L. Moody for technical assistance.

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AUTHOR INDEX TO VOLUME 51

V.R. Wallen and H.R. Jackson. Aerial photography as a survey technique for the assessment of bacterial blight of field beans. Can. Plant Dis. Surv. 51: 163–169, 176.

Figure 1. Crop disease survey map (left) and uncontrolled strip mosaic (right) of black-and-white translations from false color infrared photographs showing the location and acreage of bean fields and incidence of bacterial blight, Hensall, Ontario, August 1968.