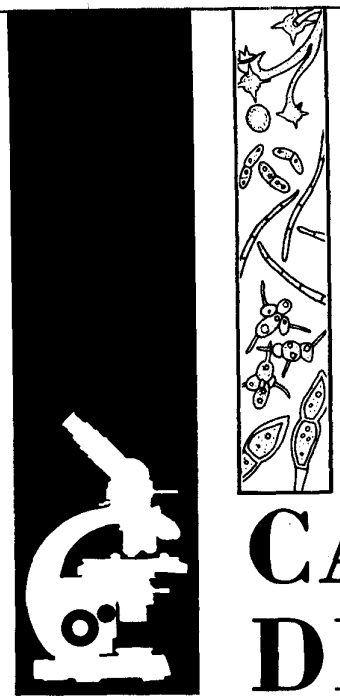
VOL.49, No.2, JUNE, 1969



CANADIAN PLANT DISEASE SURVEY

EDITOR W.L. SEAMAN

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

VOL.49, No.2, JUNE, 1969

CANADIAN PLANT DISEASE SURVEY

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

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."

PHYTOPHTHORA SYRINGAE FRUIT ROT OF APPLES IN NOVA SCOTIA[1]

R.G. Ross and C.O. Gourley[2]

Abstract

The fungus Phytophthora syringae Klebahn caused a fruit rot of stored 'McIntosh' and 'Red Delicious' apples in Nova Scotia in 1968. Nine strains of each of the two cultivars were all susceptible to the fungus. Infection appeared to occur when harvested apples were left in the orchard in boxes through a rainy period.

Introduction

In December 1968 a decay of stored 'McIntosh' and 'Red Delicious' apples was brought to the writers' attention. The decay has since proved to have been caused by the fungus Phytophthora syringae Klebahn. As far as the authors are aware, this fungus has not previously been reported in North America as a cause of decay of apple fruit in storage or on trees. In Europe P. syringae has been recorded as the cause of a collar rot of apple trees, a rot of fruit on the trees and a decay of apple fruit in storage (1, 2, 4, 5, 6, 7, 8, 9, 10). In North America it has caused a stem canker of nursery stock of heeled-in crab apple trees (11). The fungus isolated in Nova Scotia was sent to Dr. C. J. Hickman, London, Ontario, and Dr. D. L. McIntosh, Summerland, British Columbia, for confirmation of its identity. Both believed it to be P. syringae. The specimen has been filed in the National Mycology Herbarium, Plant Research Institute, Ottawa, Ont., as DAOM 127886.

Symptoms

The symptoms on the decayed portion of the apple fruit were very similar to those described by Ogilvie (5). On the cultivar McIntosh (Figs. 1 and 2) the decayed areas were light brown and on 'Red Delicious', dark brown. The margin was indefinite resembling a bruise, the rotted area was firm, and there were no superficial wounds. Often the entire fruit was decayed. The interior of the rotted tissue was elastic or stringy when teased with a scalpel, and the vessels throughout the flesh were a darker brown, giving a striated appearance. The fungus was not evident on the exterior of the fruit.

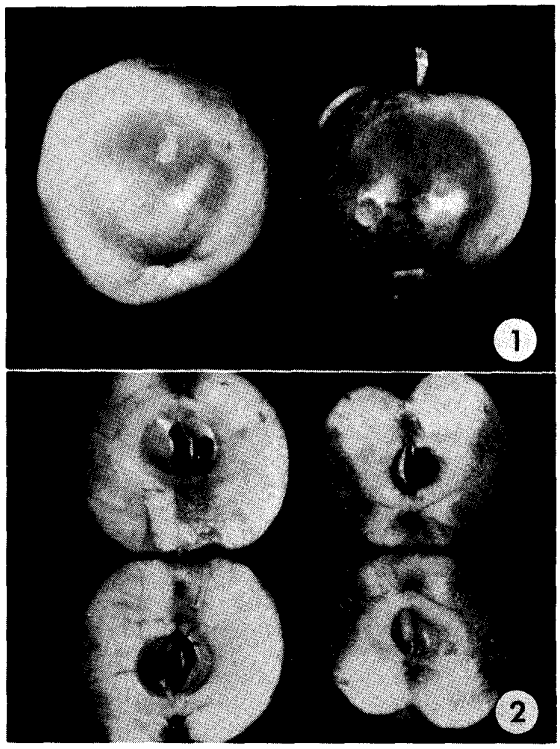


Figure 1. Phytophthora syringae rot of 'McIntosh' apple.

Figure 2. Cross section of 'McIntosh' apples rotted by *Phytophthora syringae*.

Experimental

The apples rotted by P. syringae (Fig. 3) were from a 10-year-old orchard located at the Canada Department of Agriculture Research Station, Kentville, N.S., in which the Rogers, Hamilton, N.J. 2, A11 Red, Generation Removed, Blackmac, Farley, W.L. Hamilton and Geneva strains of the cultivar McIntosh; and the Starking, Turner, Bridgeham, Tucker, Richared, Shotwell, Red King, Royal Red, and Vance strains of 'Red Delicious' are being evaluated. The planting consisted of columns of 'McIntosh' and 'Red Delicious' alternating in a 9 × 9 latin

[1] Contribution No. 1365, Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

[2] Plant Pathologists.

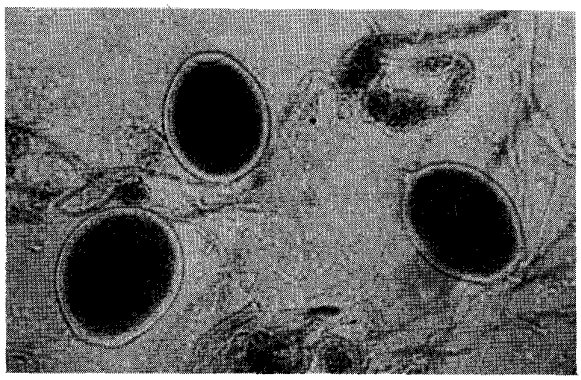


Figure 3. Sporangia of *Phytophthora syringae* (X600), DAOM 127886

square with the rows and trees 20 feet apart. In 1967 an experiment in which Alar (succinic acid 2,2-dimethyl hydrazide, 85%; Uniroyal (1966) Ltd., Elmira, Ont.) was applied on different dates was superimposed on the original design. In 1968 Alar was not applied. A sample of 100 apples from each tree was stored in a bushel picking box at 1C. In January 1969 these samples were examined for rot and the causal organism of each rotted area determined by isolation on potato dextrose agar (PDA).

In the cultivars McIntosh and Red Delicious, an average of 4.7% and 6.3%, respectively, of the apples were rotted by P. syringae. In both cultivars there were some samples that had no rot, whereas the maximum percentages of rotted fruit per sample were 12 and 34 for 'McIntosh' and 'Red Delicious', respectively. There was no evidence that P. syringae spread from apple to apple in storage. There was no indication that the previous Alar treatments had any effect on the intensity of rotting, and the data do not suggest that the strains varied in their susceptibility to P. syringae.

In the cultivar McIntosh the apples decayed by P. syringae were fairly evenly distributed throughout the orchard, but with 'Red Delicious' the average percentages of decayed apples were 7.1, 7.6, 11.4, 11.1, 7.9, 11.3, 0, 0.2, and 0.1 from rows 1 to 9, respectively. The apples from rows 1 to 6 of 'Red Delicious' were picked on October 19 and remained in the orchard in bushel boxes until October 23. Rows 7 to 9 were picked on October 22 and stored on October 23. Rainfall on October 20-21 was 4.6 inches. Similar harvest data are not available for 'McIntosh', except that picking began on October 3 and 1.54 inches of rain fell on October 4.

Similar symptoms were readily reproduced in 'Red Delicious' apples when they were inoculated below the peel with inoculum from an agar culture of P. syringae or from a decayed apple. Twenty-four apples were inoculated, using culture inoculum and an additional 24, with inoculum from a decayed apple. Twelve of the inoculated apples of each set were placed in a plastic bag and incubated at 20C, and 12 were similarly incubated at 1C. After 11 days at 20C the average diameters of the rotted areas on the apples inoculated from culture and from decayed apple tissue were 4.8 and 4.9 cm, respectively. At this time the apples at 1C showed no evidence of decay, but after 36 days the average diameters of the rotted areas were 4.9 and 4.5 cm, respectively. Isolations on PDA showed all rots to be caused by P. syringae.

At about monthly intervals during the growing season of 1969, soil samples were collected from under the canopy of 'McIntosh' and 'Red Delicious' trees and were saturated with water in shallow pans. Green apples or pears were placed on the wet surface as recommended by Klotz and DeWolfe (3) but none of the fruit became rotted by P. syringae.

Discussion

The literature suggests that outbreaks of fruit rot of apples caused by P. syringae are very sporadic. No explanation can be given for its sudden appearance in Nova Scotia. Inoculum for fruit infections by Phytophthora spp. is generally considered to come from the soil (10), and our results with the cultivar Red Delicious suggest this. Apples picked and left in the orchard in bushel boxes during a rainy period were heavily infected, whereas the disease was very rare on apples picked after the rain. This shows that most of the infection occurred after picking and suggests that the inoculum reached the fruit in water and soil particles splashed from the ground. Where the disease occurred, there was a large variation among trees in the number of decayed apples. The samples taken for storage may have been from apples subjected to different amounts of splashing from the ground. The apples may have been from boxes stacked at different heights on sod or on the 10-foot strip down each row where ground vegetation had been removed with herbicides.

According to Wormald (10) no attempts have been made to control Phytophthora apple fruit rots. The results here show the danger of leaving picked fruit in the orchard over wet periods where P. syringae is present.

Literature cited

1. Buddenhagen, I. W. 1955. Various aspects of Phytophthora cactorum collar rot of apple trees in the Netherlands. Tijdschr. Plantenziekten 61:122-129. (Rev. Appl. Mycol. 35:103. 1956).

2. Calhoun, J. 1938. Fungi causing rots of apple fruits in storage in Northern Ireland. Ann. Appl. Biol. 25:88-99.

3. Klotz, L. J., and T. A. DeWolfe. 1958. Techniques for isolating Phytophthora spp. which attack citrus. Plant Dis. Reptr. 42:675-676.

4. Lafferty, H. A., and G. H. Pethybridge. 1922. On a Phytophthora parasitic on apples which has both amphigynous and paragynous antherida; and on allied species which show the same phenomenon. Roy. Dublin Soc. Sci. Proc. 17:29-43. (Rev. Appl. Mycol. 2:181. 1922).

5. Ogilvie, L. 1931. A fruit rot of apples and pears due to a variety of Phytophthora syringae, p. 147-150. In Annu. Rep. Agr. and Hort. Res. Sta., Long Ashton, Bristol, for 1930.

6. Roosje, G. S. 1957. In Report for 1956 of the Experiment Station for Outdoor Fruit Culture. p. 37-46. (Rev. Appl. Mycol. 37:239. 1958).

7. Roosje, G. S. 1958. In Report for 1957 of the Experiment Station for Outdoor Fruit Culture. p. 14 (Rev. Appl. Mycol. 38:213. 1959).

8. Sewell, G. W. F., and J. E. Wilson. 1963. Branch Stem and collar rot of apple caused by Phytophthora species of the "cactorum group". Nature 200:1229.

9. Sewell, G. W., and J. E. Wilson. 1964. Death of maiden apple trees caused by Phytophthora syringae Kleb. and a comparison of the pathogen with P. cactorum (L. & C.) Schroet. Ann. Appl. Biol. 53:275-280.

10. Wormald, H. 1955. Diseases of fruits and hops. Crosby Lockwood and Son Ltd. London.

11. Young, R. A., and J. A. Milbrath. 1959. A stem canker disease of fruit tree nursery stock caused by Phytophthora syringae. Phytopathology 49:114-115. (Abstr.)

BACTERIAL BLISTER SPOT OF APPLE IN ONTARIO

B.N.Dhanvantari[1]

Abstract

This is the first report of bacterial blister spot of apple in Canada. The disease was found on the apple cultivar 'Mutsu' in Essex County and was characterized mostly by lesions with brown centers and dark-purple borders around the lenticels on the fruit and infrequently by elongated depressed areas on the twigs. Pseudomonas syringae was isolated from these lesions and its pathogenicity to apple was established.

Introduction

Blister spot of apple was first reported from Missouri in 1916 and described by Rose (8) who found that it was a bacterial disease and named the causal organism Pseudomonas papulans. He also isolated these bacteria from the bark of apple trees that showed symptoms of a disease he described as rough bark or scurfy bark. In 1931, Lacey and Dowson (5) reported a bacterial canker on seedling trees of several new varieties of apple in England; the disease was characterized by horseshoe-shaped or circular cracks up to one inch in diameter, raised blisters, and elongated depressed areas in the bark, and dead buds. They found that the organism they isolated was identical with P. papulans Rose, the cause of scurfy bark in the United States. Roberts (7) also isolated P. papulans from early lesions associated with a disease he had described as target canker and was of the opinion that measles, target canker, and rough bark of apple might prove identical, since their early symptoms were similar. Smith (10) found that blister spot of apple had been reported from Missouri, Arkansas, Indiana, Pennsylvania, Virginia, and Illinois but not outside the United States. Although he was able to induce cankers on apple twigs by inoculation, he was of the opinion that under natural conditions, the disease occurred only on apple fruits. He further stated that P. papulans could not be assigned to a specific rank, as it was similar in morphological, cultural, physiological, and pathological characteristics to P. syringae, and he considered it to be only a strain of the latter species.

Disease incidence, symptomatology, and isolations

During the summer and fall of 1968, it was noted that apples of the cultivar 'Mutsu', in an orchard in the Leamington areas of Essex County, Ontario, had conspicuous lesions around the lenticels (Fig. 1) that rendered them unmarketable. Almost every tree of 'Mutsu' was affected, but the condition was not found on other cultivars in the same orchard. A similar pattern of disease incidence was seed in two other orchards in Essex Co. with 'Mutsu'.

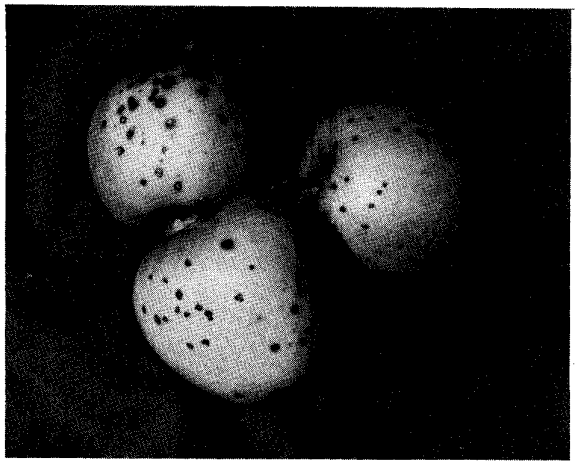


Figure 1. Blister spot on 'Mutsu' apple.

The lesions were always confined to the area around the lenticels. Incipient lesions appeared as water-soaked areas; mature ones were about 1-3 mm in diam., 1-2 mm deep, and had blistered brown centers with a dark-purple border. Some lesions were irregular in outline, appearing like minute infections of the apple scab fungus, Venturia inaequalis (Cke.) Wint., but mostly they were round. While the infections appeared to be uniform around the tree, the side of the fruit exposed to the outside had more spots. Twig infections were relatively infrequent and appeared as elongated, depressed areas, measuring 14-43 x 3-6 mm.

Microscopic examination of the fruit spots and twig cankers showed the association of bacteria with them. Isolations yielded bacteria that conformed in morphological, cultural, and biochemical characteristics to the description of Pseudomonas syringae van Hall as set out in Bergey's Manual of Determinative Bacteriology, 7th edition (2) with the exception

[1] Research Station, Canada Department of Agriculture, Harrow, Ontario.

that these isolates were lipolytic on Tween 80, according to the method of Sierra (9). These isolates produced round, dark-purple lesions 2-3 mm in diam. in 10-15 days when bacterial suspensions in sterile distilled water (75 Klett units) were inoculated into mature fruit of 'Mutsu' apples by needle-puncture. The bacteria were reisolated from such lesions. Inoculation of the non-wounded surface of the fruit was not successful. Leaf spots were produced on young leaves of 'Veecot' apricot (Prunus armeniaca L.) in 7 days when the plants were inoculated by atomizing the bacterial suspension under a pressure of 25 psi and held in a moist chamber for the following 48 hours. The last method has been used successfully to test the pathogenicity of a number of isolates of Pseudomonas occurring on stone fruits. These tests, it is thought, are sufficient to establish the pathogenicity of the apple isolates. Further work is under way to assess the factors contributing to the production of field symptoms.

Discussion

Lenticel spots of apple have been ascribed to diverse causes. In New Zealand, Brook (4) isolated from pre-harvest lenticel spots on 'Sturmer' and 'Golden Delicious' apples nine different fungi, among which Stemphylium botryosum Wallr. and Urocladium consortiale (Thum.) Simmons were predominant. He presumed that they were pathogenic because the lenticel spots were controlled by pre-harvest sprays with fungicides. In France, Bondoux (1) attributed lenticel spots on 'Golden Delicious' and 'Reinette du mans' apples to three pathogenic fungi, Trichoseptoria fructigena Maubl., Cylindrocarpon mali (Allesch) Wr. (stat. perf. Nectria galligena Bres.), and Gloeosporium perennans Zeller & Childs (Pezicula malicorticis). Miller and Rich (6) found grey to dark-brown lesions, 1-2 mm in diam. and 1 mm deep, around lenticels on 'McIntosh' apples in the valleys near New Haven, Connecticut. Because ozone is the air pollutant that most commonly causes damage to plants in Connecticut and also because the orchardist had noticed smog-like conditions in these valleys during periods of temperature inversions, they tested mature apples against ozone fumigation. Symptoms ranging from raised lenticels to small pits or larger depressed areas around lenticels occurred only when apples were exposed to ozone for at least 3 days; it should also be noted that the concentration of ozone used in their experimental set-up was 10 times the highest recorded for ambient air in New Haven and also that such symptoms could not be induced on 'Golden Delicious', which is a parent of 'Mutsu'. Lenticular spotting on apples was also reported to be caused by ammonia fumes (3) and hence it is possible that such symptoms may be induced by a variety of phytotoxic fumes as well as pathogenic micro-organisms. Because P. syringae was isolated from a large number of lenticel spots on apple fruits and from a few twig cankers, and because its pathogenicity was established by inoculation, the author is of the opinion that the disease reported hrer on the apple cultivar 'Mutsu' is not different from the blister spot of apple described by Rose (8) and Smith (10) in the United States.

This is the first report of bacterial blister spot of apple in Canada. So far as it can be ascertained by the author, this is also the first report of the fruit spot phase of this disease outside the United States.

Literature cited

1. Bondoux, P. 1967. Warm water and thiabendazole in curative treatment against lenticel parasites of apple. Rev. Appl. Mycol. 47: 2205.

2. Breed, R. S., E. G. D. Murray, and N. R. Smith. 1957. Bergey's Manual of Determinative Bacteriology. 7th ed. Williams and Wilkins Co., Baltimore. 1094 p.

3. Brennan, E., I. Leone, and R. H. Daines. 1962. Ammonia injury to apples and peaches in storage. Plant Dis. Reptr. 46:792-795.

4. Brook, P. J. 1968. Pre-harvest lenticel spot of apples. New Zeal. J. Agr. Res. 11:237-244.

5. Lacey, M. S., and W. J. Dowson. 1931. Studies in bacterioses. XVIII. A bacterial canker of apple trees. Ann. Appl. Biol. 18:30-36.

6. Miller, P. M., and S. Rich. 1968. Ozone damage in apples. Plant Dis. Reptr. 52:730-731.

7. Roberts, J. W. 1934. Apple target canker, measles and rough bark. Phytopathology 24: 16. (Abstr.)

8. Rose, D. H. 1917. Blister spot of apples and its relation to a disease of the apple bark. Phytopathology 7:198-208.

9. Sierra, G. 1957. A simple method for the detection of lipolytic activity of microorganisms and some observations on the influence of the contact between cells and fatty substrates. Antonie van Leeuwenhoek J. Microbiol. Serol. 23:15-22.

SEED TREATMENT FUNGICIDES FOR CONTROL OF CONIFER DAMPING-OFF: LABORATORY AND GREENHOUSE TESTS, 1967-68

L. W. Carlson and J. Belcher[1]

Abstract

Sixty-one seed treatment chemicals were tested in laboratory germination tests and 25 in greenhouse damping-off control tests. Effective control of preemergence damping-off was attained with 13 chemicals for jack pine, 15 for lodgepole pine, and 6 for white spruce. Post emergence damping-off was controlled effectively by 8 chemicals for jack pine, 9 for lodgepole pine, and 3 for white spruce. Two experimental fungicides, THC 324 and DHC 324, were effective in controlling postemergence damping-off of all three conifer species.

Introduction

In an earlier report (1) the inhibition of mycelium growth of isolates of Pythium sp., Rhizoctonia sp., and Fusarium sp. by 69 different seed-treatment chemicals was shown. Results for seed germination and damping-off control under greenhouse conditions were also given for a few of the 69 chemicals. To supplement the earlier tests, results of seed germination tests and damping-off control studies for the more active of the 69 chemicals are reported here. The species of conifers tested were jack pine (Pinus banksiana Lamb.), lodgepole pine (P. contorta Dougl. var. latifolia Engelm.), and white spruce (Picea glauca (Moench) Voss).

Materials and methods

General procedures for the germination tests and greenhouse damping-off control tests of the 61 chemicals (Table 1) were described in the earlier report (1). Lodgepole pine was substituted for red pine as a test species. The seeds were pelleted at the rate of 0.33 g of chemical per gram of seed. (In the earlier study [1], rates were 0.25 g and 1.0 g in the laboratory germination tests and 0.5 g and 2.0 g in those for damping-off control). One hundred air-dried seeds of each tree species were used in each treatment.

Sixty-one chemicals, earlier found to have a high degree of activity against Rhizoctonia sp., Pythium sp., or both, were screened for phytotoxic effects in laboratory germination tests. Twenty-five of the chemicals were then used in greenhouse damping-off control tests because of their non-phytotoxicity to at least one of the test species, or because they were standard treatments now in use (captan and thiram).

Results and discussion

Laboratory germination tests — Twenty-three of the 61 chemicals tested had no inhibitory effect on germination of any of the test species (Table 2) Captan and Arasan inhibited germination of all three species, and both chemicals inhibited germination of lodgepole pine and white spruce more than that of jack pine. Many of the other chemicals tested caused only small reductions in germination and may be used in future studies on soil treatments for damping-off control.

Greenhouse damping-off control tests — Preemergence damping-off was significantly reduced by 13 chemicals for jack pine, 15 for lodgepole pine, and 6 for white spruce (Table 4). Postemergence damping-off losses were significantly less with 8 chemicals for jack pine, 9 for lodgepole pine, and 3 for white spruce.

Six of the 23 chemicals selected for greenhouse tests were used on jack pine despite minor phytotoxic effects. Significant reduction of preemergence damping-off was observed with four of the six treatments, (nos. 39,42,51, and 52) and significantly less postemergence damping-off was also observed with four of them (nos. 7, 14, 39, and 42).

The most effective chemicals for control of preemergence damping-off were 66-S-2 for jack pine and white spruce and TMHC 175 for lodgepole pine. Others of high activity were KHC 324, 66-S-3, DHC 324, and Arasan for jack pine; 66-S-2, THC 324, DHC 324, and Arasan for lodgepole pine; and THC 324 and Arasan for white spruce. Postemergence damping-off was best controlled with Polyram ZMC5-80W for jack pine; Arasan for lodgepole pine; and Arasan and DHC 324 for white spruce. Other fairly effective chemicals were 6638 and DHC 324 for jack pine; THC 324 and 66-S-2 for lodgepole pine; and THC 324 for white spruce. Several of the chemicals tested, 66-S-2, THC 324, DHC 324, and BHC 324, were fairly active against both pre- and postemergence damping-off.

[1] Research Scientist and Research Technician, Department of Fisheries and Forestry, Canadian Forestry Service, 25 Dafoe Road, Winnipeg 19, Manitoba, Canada.

Table 1. Source and identity of seed treatment materials

Treatment number	Source*	Product and formulation	Chemical name or active ingredient
1	Stauffer	Captan 50% WP	captan
2	Diamond Shamrock	Daconil 2787 75% WP	tetrachloroisophthalonitrile
3	Diamond Shamrock	Daconil 2787 (35%) & captan (35%)	tetrachloroisophthalonitrile + captan
4	Naugatuck	Spergon 95%	chloranil
6	Naugatuck	Vitavax 75% (D735)	5, 6-dihydro-2-methyl-1, 4-oxathiin-3-carboxanilide
7, 9	Naugatuck	Numbered compounds	identity not available
10	Niagara	Phygon 50%	dichlone
11	Niagara	Polyram 80%	zinc activated polyethylenethiuram disulfide
14	Niagara	Polyram ZMCS 80W	identity not available
15	Dupont	Arasan 75%	thiram
16	Dupont	Manzate D 80%	maneb
18	Dupont	Fermate 76%	ferbam
19	Dupont	Demosan 65%	1, 4 dichloro-2, 5-dimethoxybenzene
20	Chemagro	4497 50%	bis (1, 2, 3, -trichloroethyl) sulfoxide
21	Chemagro	Dyrene 50%	2, 4-dichloro-6-(o-chloroanalino)-s-triazine
22	Chemagro	Dexon 50%	p-dimethylaminobenzenediazo sodium sulfonate
23	Chemagro	Bay 47531	dichlofluanid
24	Cyanamid	Cyprex 65%	dodine
25	Green Cross	DuTer 20%	triphenyl tin hydroxide
27	Green Cross	RD8684 + Cyprex 20%	identity not available + dodine
28	Green Cross	3944X	identity not available
29	Green Cross	Drillbox Lindasan	lindane 37.5% & captan 5%
30-32	Green Cross	Numbered compounds	identity not available
35	Green Cross	RD8684 + maneb (50%)	identity not available + maneb
36	Green Cross	RD8684 + captan (50%)	identity not available + captan
37-43	Green Cross	Numbered compounds	identity not available
44-48) 50-55) 57-59)	Morton	Numbered compounds	identity not available
60, 61	Chipman	Numbered compounds	identity not available
63	Chipman	66-S-2	zinc coordinated maneb 50%
64-66	Chipman	Numbered compounds	identity not available
67	Co-op	Hexa 40%	hexachlorobenzene
68	Dow	Dowicil 100 95%	1-(3 chloroallyl)-3, 5, 7-triaza-1-azoniaadamantane chloride
69-70	Hoechst	Numbered compounds	identity not available
71	Niagara	Polyram Seed Protectant 53.5%	zinc activated polyethylene thiuram disulfide

* Chemicals were supplied by: Stauffer Chemical Co. of Canada Ltd., Vancouver, B.C.; Diamond Shamrock Corp., Painesville, Ohio; Naugatuck Chemicals, Elmira, Ontario., Niagara Brand Chemicals, Burlington, Ont.; DuPont of Canada Ltd., Montreal, Que.; Chemagro Corp., Kansas City, Mo.; American Cyanamid, New York, N. Y.; Sherwin-Williams Co. of Canada Ltd. (Green Cross Products), Montreal, Que.; Morton Chemical Co., Woodstock, Ill.; Chipman Chemical Ltd., N. Hamilton, Ont.; Interprovincial Cooperatives Ltd., Winnipeg, Man.; Dow Chemical Co., Midland, Mich.; American Hoechst Corp., North Hollywood, California.

Table 2. Germination in seed germinator of conifer seeds pelleted with seed treatment chemicals

	Treatment and product	Germination (%)		
		Jack pine	Lodgepole pine	White spruce
1	Captan 50WP	76	52	55
2	Daconil 75WP	86*	38	43
3	Daconil + captan (35-35)	77	62	49
4	Spergon 95%	89*	46	80*
6	Vitavax 75%	17	20	2
7	6638	80	70*	59
9	D-735-10D	92*	65*	15
10	Phygon 50%	88*	3	11
11	Polyram 80%	67	39	40
14	Polyram ZMCS 80%	72	52	70*
15	Arasan 75%	78	34	50
16	Manzate D 80%	60	22	15
18	Fermate 76%	75	48	14
19	Demosan 65%	42	39	27
20	4497 50%	0	0	46
21	Dyrene 50%	19	28	2
22	Dexon 50%	1	1	0
23	Bay 47531	50	39	1
24	Cyprex 65%	2	0	3
25	DuTer 20%	4	9	0
27	RD 8684 + Cyprex	24	6	3
28	3944X	30	17	0
29	Drillbox Lindasan	85*	26	53
30	MHC 223	95*	74*	73*
31	TMHC 175 (2)	93*	77*	67*
32	TMHC 2222	89*	75*	77*
35	RD 8684 + maneb 50%	55	14	36
36	RD 8684 + captan 50%	77	53	44
37	KHC 324	89*	76*	61
38	MHC 324	83	64	54
39	PHC 324	75	67*	46
40	XHC 324	66	32	45
41	BHC 324	91*	72*	79*
42	DHC 324	82	73*	78*
43	THC 324	92*	81*	74*
44	EP 277 50%	5	10	10
45	EP 277 A liquid	2	0	3
46	EP 279 50%	0		
47	EP 279 A liquid	0		
48	EP 293 50%	0	0	0
50	EP 301B 50%	51	59	59
51	EP 301C	77	54	72*
52	EP 301D	83	69*	72*
53	EP 301E	86*	92*	69*
54	EP 302B	37	27	2

Table 2 (Con't)

Treatment and product	Germination (%)		
	Jack pine	Lodgepole pine	White spruce
55 EP 302C	60	30	43
57 EP 305	0	0	0
58 EP 306 75%	5	1	0
59 EP 308	73	39	20
60 65-S-1	91*	50	54
61 54-S-7	79	64	64
63 66-S-2	92*	78*	81*
64 66-S-3	90*	77*	82*
65 66-S-4	70	51	46
66 66-S-6	70	45	20
67 Hexa	93*	66*	68*
68 Dowicil 100 95%	1	58	34
69 2844	0	1	8
70 2874	0	0	2
71 Polyram S. P.	79	63	79*
Untreated control	89	80	83

* Statistically not different from the untreated control at the 5% level.

Table 3. Seed-treatment chemicals not inhibiting conifer seed germination under laboratory conditions

Conifer	Number of chemicals	Treatment number
Jack pine, lodgepole pine, and white spruce	9	30, 31, 32, 41, 43, 53, 63, 64, 67
Jack pine and lodgepole pine	2	9, 37
Jack pine and white spruce	1	4
Lodgepole pine and white spruce	2	42, 52
Jack pine, alone	4	2, 10, 29, 60
Lodgepole pine, alone	2	7, 39
White spruce, alone	3	14, 51, 71
Total	23	
Jackpine, total	16	2, 4, 9, 10, 29, 30, 31, 32, 37, 41, 43, 53, 60, 63, 64, 67
Lodgepole pine, total	15	7, 9, 30, 31, 32, 37, 39, 41, 42, 43, 52, 53, 63, 64, 67
White spruce, total	15	4, 14, 30, 31, 32, 41, 42, 43, 51, 52, 53, 63, 64, 67, 71

Table 4. Effect of seed treatment on preemergence and postemergence damping-off of conifer seedlings in natural soil in the greenhouse

	Treatment and product	Emergence (%)			Postemergence damping-off (%)		
		Jack pine	Lodgepole pine	White spruce	Jack pine	Lodgepole pine	White spruce
1	Captan	53	69*	40	68	58*	40
2	Daconil	54	-†	-	96	-	-
4	Spergon	77*	-	29	75	-	28
7	6638	60	44	-	17*	67	-
9	D735-10D	63	59*	-	58	80	-
10	Phygon	33	-	-	89	-	-
14	Polyram ZMCS 80W	63	-	60*	11*	-	31
15	Arasan	78*	74*	67*	77	44*	24*
29	Drillbox Lindasan	76*	-	-	83	-	-
30	MHC 223	58	64*	58*	49	54*	42
31	TMHC 175	64	82*	-	59	72	-
32	TMHC 2222	67*	73*	40	57	56*	68
37	KHC 324	81*	59*	-	49	76	-
39	PHC 324	70*	61*	-	35*	77	-
41	BHC 324	74*	70*	46	31*	57*	38
42	DHC 324	78*	74*	55	25*	60*	24*
43	THC 324	66	75*	68*	43*	46*	25*
51	EP 301C	73*	-	49	52	-	33
52	EP 301D	68*	56	40	55	59*	40
53	EP 301E	66	67*	-	61	72	-
60	65-S-1	72*	-	-	45*	-	-
63	66-S-2	83*	81*	73*	36*	46*	53
64	66-S-3	80*	67*	59*	61	63	48
67	Hexa	55	64*	31	70	79	47
71	Polyram S.P.	-	-	55	-	-	41
	Untreated control	47	39	35	68	79	50

†Indicates that the treatment was not included in the greenhouse test because of its phytotoxicity to the conifer in the seed germination test at the treatment rate of 0.33 g of chemical/g of seed.

* Significantly different from the untreated control at the 5% level.

The general performance of these chemicals was better than Captan and in some cases better than Arasan. In these tests Arasan performed better than Captan.

Seed treatment chemicals that were effective in these tests had in earlier laboratory bioassay tests demonstrated high activity against Rhizoctonia sp. and Fusarium sp. and variable activity against Pythium sp. It is possible that combinations of the better chemicals may give even better control and this approach will be considered in future studies. The continued use of thiram and captan as standard seed treatments for conifer seedling damping-off seems to be in question. The better chemicals mentioned above will undergo further greenhouse and field testing in order to find a satisfactory replacement for captan or thiram, which are still the best chemicals available commercially.

Literature cited

1. Belcher, J., and L. W. Carlson. 1968. Seed-treatment fungicides for control of conifer damping-off: Laboratory and greenhouse tests, 1967. Can. Plant Dis. Surv. 48:47-52.

SCALE ROT TESTS OF HARDY HYBRID LILIES[1]

J. Drew Smith[2] and Edward A. Maginnes[3]

Abstract

Twenty-two Lilium cultivars and one species showed differences in susceptibility to scale rot pathogens Cylindrocarpon radicicola Wr. and Colletotrichum dematium (Pers. ex Fries) Duke in a pot test. In another pot test the same 22 cultivars and 2 Lilium species showed differences in susceptibility to the scale rot pathogen Fusarium oxysporum Schlecht f. lilii Imle.

F. oxysporum caused more rot than the other pathogens; only the cultivar 'Rose Cup' showed considerable resistance to this fungus. Several of the test entries consistently showed resistance to the other rot organisms. Further breeding work should be directed particularly towards obtaining resistance to fusarium scale rot.

Introduction

Scale and bulb rots were reported by Smith and Maginnes (5) to be limiting factors in the successful cultivation of hardy hybrid lily cultivars in Saskatchewan, particularly in heavy soils. By means of a greenhouse scale test in 1966 (5) differences in resistance to the fungal pathogens Cylindrocarpon radicicola Wr., Colletotrichum dematium (Pers. ex Fries) Duke and Rhizoctonia solani Kuhn were demonstrated in six cultivars from the collection of Patterson Hybrids at the Department of Horticulture, University of Saskatchewan at Saskatoon (7). Resistance to scale rot caused by Fusarium oxysporum Schlect f. lilii Imle was not tested in 1966. This pathogen is a common cause of severe basal rot of lilies in Saskatchewan (5) and elsewhere in North America (2). This paper reports the results of further scale tests on a wider range of lily cultivars and species in 1967 and 1968.

Materials and methods

Test 1. In 1967, 19 Patterson, 1 Preston and 2 Skinner Hybrids (4), and a parental species 'Willmott' (Lilium davidi var. willmottiae) (8) (Table 1) were included in a 10-week test for resistance to scale rot caused by C. radicicola and C. dematium. The test fungi were isolated from diseased lily bulbs.

Test 2. In 1968, scales of the lilies as in Test 1 (1967) and bulblets of the prairie lily, Lilium philadelphicum L. var. andinum[4] (Nutt.) Ker. were included in a 12-week test for resistance to F. oxysporum f. lilii. A mixture of three isolates of the fungus from diseased lily bulbs was used as inoculum.

In both tests the methods of pathogen culture, soil and pot sterilization, pot inoculation, and scale preparation were the same as in the test already reported (5), but the scales were not wounded. Ten scales of one cultivar or species were planted in a 6-inch pot; in the case of the prairie lily, six bulblets were planted per pot. Each cultivar or species was replicated four times in inoculated and check treatments. The severity of scale rot was assessed using a 0 to 4 rating (5). Individual scale or bulblet ratings were converted to a pot index figure.

Results and discussion

Test 1. Although only "clean", undamaged scales were used, some scale rot appeared in all the cultivars grown in the uninoculated soil (Table 1). Most of this rot was due to Penicillium species, which frequently develop on bulbs in low temperature storage (2, 5). Occasionally scales showed insect damage and bacterial soft rot; these were recorded as rotted. The presence of rot on scales in uninoculated soil suggests that either the preliminary wash and dip in hypochlorite solution did not control incipient rot or that other rotting organisms were introduced in the top watering of the pots.

There was significantly more scale rot in Cylindrocarpon- than in Colletotrichum-inoculated soil (Table 1). Some of the cultivars were consistent in their reaction to the pathogens. 'Fuchsia Queen',

---

[1] Contribution No. 347, Research Station, Canada Department of Agriculture, Saskatoon, Saskatchewan.

[2] Plant Pathologist, Research Station, Canada Department of Agriculture, Saskatoon, Saskatchewan.

[3] Assistant Professor, Department of Horticulture, University of Saskatchewan, Saskatoon, Saskatchewan.

[4] This lily, the floral emblem of Saskatchewan, flourishes on the lighter soils of the open woodland and prairie. It was tested as a possible parental disease resistant species.

Table 1. The reaction of scales of 22 Lilium cultivars and 1 species to Cylindrocarpon radicicola and Colletotrichum dematium

Parental component†	Cultivar	Average rot rating*			
		Overall	Check	Cylindrocarpon	Colletotrichum
	Willmott	1.05 a**	0.92 ab	1.22 a	1.00 a
W	Rose Cup	1.29 ab	1.00 abc	1.35 abc	1.52 bcdefg
	Maxwill	1.30 ab	1.15 bcd	1.60 abcde	1.15 abc
W	Lemon Queen	1.31 ab	1.05 abc	1.27 ab	1.60 cdefgh
W	Apricot Glow	1.34 abc	1.15 bcd	1.75 bcde	1.12 ab
WM	White Gold	1.34 abc	1.07 abc	1.57 abcde	1.37 abcdefg
W	Red Torch	1.39 abc	1.00 abc	1.95 defg	1.22 abcd
WM	Burnished Rose	1.43 abc	1.37 bcde	1.55 abcde	1.35 abcdef
W	Pink Charm	1.47 abc	1.25 bcde	2.00 cdefgh	1.17 abc
?	Crimson Queen	1.50 abc	1.42 bcde	1.47 abcd	1.60 cdefgh
WM	Rose Queen	1.51 abc	1.30 cde	1.95 defgh	1.27 abcde
W	Lillian Cummings	1.59 bcd	1.52 cde	1.75 bcde	1.50 abcdefg
W	Rosalind	1.60 bcd	1.47 cde	1.55 abcde	1.77 efghjk
?	Primrose Lady	1.60 bcd	1.25 bcde	2.07 efgh	1.47 abcdefg
?	Crimson Beauty	1.65 bcd	0.92 ab	2.30 ghj	1.72 defghj
W	Edith Cecilia	1.72 bcd	1.50 cde	1.77 bcdef	1.87 ghjk
WM	White Princess	1.77 bcde	1.60 de	1.87 defg	1.82 fghjk
W	Bronze Queen	1.86 cdef	1.50 cde	1.85 cdefg	2.22 jk
W	Dunkirk	2.04 defg	0.62 a	3.25 l	2.25 k
W	Rose Dawn	2.07 defg	1.47 cde	2.67 jk	2.05 hjk
W	Orchid Queen	2.25 efg	2.10 e	2.45 hjk	2.22 jk
W	Fuchsia Queen	2.32 fg	2.02 e	2.75 jk	2.17 jk
W	Jasper	2.44 g	2.25 e	2.82 kl	2.25 k
Mean rot		1.65	1.34	1.64	1.95

* 0 was no visible rot and 4, complete rot.

** Duncan's multiple range test (1) at the 5% level of significance.

† W = 'Willmott' (L. davidi var. willmottiae) (8); M = 'Maxwill' (L. X 'Maxwill') (3); ? = Parentage uncertain.

'Jasper' and 'Dunkirk' were consistently very susceptible to both pathogens. 'Willmott' was consistently resistant. 'Dunkirk', the cultivar least affected by rot in the uninoculated soil, and 'Crimson Beauty'[5] were particularly susceptible to C. radicicola. In both of these cultivars it was necessary to use small inner scales for the tests, since the outer scales of the bulbs showed severe storage rot. This may have affected their reaction in the test.

The ranking of the cultivars 'Dunkirk' and 'Jasper', as very susceptible, 'Lillian Cummings' and 'Crimson Queen'[6] as intermediate, and 'Burnished Rose' and 'Apricot Glow' as less susceptible (Table 1) was in agreement with previous test results (5).

Although 'Willmott' was outstanding in its resistance to scale rot and 'Maxwill' reasonably rot resistant, cultivars derived from these parents (Table 1) showed a wide range of rot reaction. The parentage of many of the cultivars is known to be complex at least on one side. We have not yet fully determined the rot resistance of all parents, but the test method which has been developed is probably suitable for this task.

[5] Name changed to 'Cardinal Beauty' in 1969 (6).

[6] Name changed to 'Tiger Queen' in 1969 (6).

Test 2. After 12 weeks, scales of all cultivars showed rot in uninoculated soil (Table 2). The severity of this rot was of the same order as that in uninoculated soil in the previous test (Table 1). Complete bulblets of the prairie lily, however, showed no rot in uninoculated soil.

Table 2. Reaction of scales of Lilium cultivars and 1 species and bulblets of another species to Fusarium oxysporum f. lilii

Cultivar or species	Average rot rating* Check	Fusarium
Rose Cup	1.2 bc**	2.9 a
Apricot Glow	1.3 bc	3.3 ab
Crimson Queen	1.3 bc	3.3 ab
Burnished Rose	1.2 bc	3.5 ab
Edith Cecilia	1.2 bc	3.5 ab
Lillian Cummings	1.7 bc	3.6 ab
Rosalind	1.3 bc	3.6 ab
Bronze Queen	1.1 bc	3.7 b
Crimson Beauty	1.4 bc	3.7 b
Orchid Queen	1.0 b	3.7 b
Pink Charm	1.8 c	3.7 b
Primrose Lady	1.3 bc	3.8 b
Fuchsia Queen	1.4 bc	3.8 b
White Gold	1.5 bc	3.8 b
Dunkirk	1.1 bc	3.9 b
Lemon Queen	1.5 bc	3.9 b
Red Torch	1.6 bc	3.9 b
Rose Queen	1.6 bc	3.9 b
Willmott	1.1 bc	3.9 b
White Princess	1.4 bc	3.9 b
Jasper	1.2 bc	4.0 b
Maxwill	1.2 bc	4.0 b
Rose Dawn	1.1 bc	4.0 b
L. philadelphicum var. andinum	0.0 a	4.0 b

* 0 was no visible rot and 4, complete rot.

** Duncan's multiple range test (1) at the 5% level of significance.

F. oxysporum f. lilii caused moderate to severe rotting of lily scales and bulblets of the test entries, and in all it prevented bulblet formation. The scales or bulblets of four test entries were completely rotted. Only 'Rose Cup' appeared to show some worthwhile resistance. This variety also showed overall rot resistance (Table 1). Neither 'Willmott' nor 'Maxwill' were resistant to F. oxysporum.

These results indicate that in breeding new hardy lily varieties particular attention should be paid to resistance to F. oxysporum. Several cultivars appeared to possess some resistance to the other pathogens, but only 'Rose Cup' showed any degree of resistance to F. oxysporum f. lilii. It may be profitable to test the resistance of the parents of this cultivar. Because fungicidal bulb dips and soil drenches were not effective in controlling scale rot (unpublished results), breeding for resistance may offer a practical means of control.

Literature cited

1. Duncan, D. B. 1955. Multiple range and multiple F tests. Biometrics 11:1-42.
2. McWhorter, F. P. 1963. Non-virus diseases of lilies (Lilium species). North Amer. Lily Soc. Yearbook, p. 54-88.
3. Skinner, F. L. 1967. Horticultural horizons. Manitoba Dep. Agr. and Conserv. 157 p.
4. Slate, G. L. 1963. Lily species and hybrids. North Amer. Lily Soc. Yearbook, p. 17-53.
5. Smith, J.Drew, and Edward A. Maginnes. 1966. Scale rot test for hybrid lilies. Can. Plant Dis. Surv. 46:92-94.
6. The Royal Horticultural Society. 1969. The International Lily Register, 1969. Roy. Hort. Soc., London. 141 p.
7. University of Saskatchewan, Department of Horticulture. 1967. Hardy lilies. Univ. Saskatchewan, Saskatoon. 2 p. (mimeo).
8. Woodcock, H. B. D., & W. T. Stearn. 1950. Lilies of the world. Country Life Limited., London, 431 p.

A SYSTEMIC FUNGICIDE FOR CONTROL OF VERTICILLIUM WILT IN STRAWBERRIES[1]

C.L. Lockhart[2], A.A. MacNab[3], and Bart Bolwyn[4]

Abstract

The systemic fungicide Benlate controlled wilt of strawberry caused by Verticillium dahliae when applied to the planting hole at planting time. It was less effective when plant roots were dipped in the fungicide solution prior to planting, and it was ineffective as a foliar spray. Soil applications of the fungicides Lanstan and Lannate did not control verticillium wilt, and Lannate was phytotoxic.

Introduction

In Nova Scotia epidemics of verticillium wilt occur occasionally in strawberry plantings. In 1964 Gourley and MacNab (2) reported that Verticillium dahliae Klebahn attacked new plantings of strawberries in the late summer and fall. In 1966 verticillium wilt killed 50% of the strawberry plants in a new planting. Because Benlate was found to have systemic properties which protected cotton plants from verticillium wilt (1), experiments were made with Benlate and with the fungicides Lannate and Lanstan[5] to control verticillium wilt in strawberry plants.

Materials and methods

A greenhouse trial with 'Redcoat' strawberry plants was made to evaluate methods of applying Benlate (1-[butylcarbamyl]-2-benzimidazole carbamic acid, methyl ester) to control verticillium wilt. The experiment was repeated four times. Each of seven treatments was applied to five strawberry plants, each of which was in a 4-inch clay pot. Soil inoculum consisted of potting mixture (1 part soil, 2 parts peat, 1 part sand) infested with cornmeal-sand cultures of V. dahliae prior to planting. Benlate at the rate of 4 lb 50% WP/100 Imp. gal water plus Surfactant F at 4 oz/100 Imp. gal were applied as follows:

Soil drench - 8 fluid oz added to the surface of the soil in each pot at planting;

Soil drench - 8 oz added to the surface of the soil in each pot 7 days after planting;

Foliar spray - plants sprayed to run-off at planting;

Foliar spray - plants sprayed to run-off 7 days after planting;

Root dip - roots dipped in the fungicide solution prior to planting;

Soil mix - 8 oz mixed with the soil of each pot prior to planting;

Control - no fungicide.

Three months after planting, the plants were examined for symptoms of verticillium wilt and two sections of one petiole from each plant were plated on potato dextrose agar (PDA).

In 1967 three fungicide chemicals were evaluated in a field trial for the control of verticillium wilt in 'Redcoat' strawberry. In this particular field 50% of the plants had been killed by Verticillium in 1966. The experiment was laid out with 100 plants/plot, with the plants 2 ft apart and the rows 5 ft apart, using a randomized block of four replicates. The following treatments were applied on June 12 and 13, 1967:

Lanstan 20% G (1-chloro-2-nitropropane) at the rate of 45 lb active ingredient/acre

---

[1] Contribution No. 1329, Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

[2] Plant Pathologist, Research Station, Kentville, Nova Scotia.

[3] Plant Pathologist, Nova Scotia Department of Agriculture. Present address, Department of Plant Pathology, Cornell University, Ithaca, New York.

[4] Plant Pathologist, Nova Scotia Department of Agriculture. Present address, Ontario Department of Agriculture, Ridgetown, Ontario.

[5] Benlate and Lannate were obtained from Dupont of Canada Ltd., Toronto, Ontario, and Lanstan from Niagara Brand Chemicals, Burlington, Ontario.

broadcast on a 2-ft-wide strip down the center of the row and rototilled to a depth of 6 inches the day before planting;

Benlate 4 lb 50% WP/100 Imp. gal plus Surfactant F, 4 oz/100 Imp. gal, using 8 oz in each planting hole immediately prior to setting the plant;

Lannate, 90% (methyl0-[methylcarbamyl] thiolacelohydroxamate), at 1.0 lb 90%/ acre, using 8 oz/plant in the planting hole at planting;

Control - no treatment.

On October 13, the plants were examined for symptoms of verticillium wilt, and petiole sections from 30 mother plants from each treatment in each block were plated on PDA. On October 23, counts were made of the number of runner plants on all mother plants.

In 1968, Benlate was used in a field trial on randomized plots of the same size as those used in 1967. Benlate was applied at rates of 1, 2, and 4 lb 50% WP/100 Imp. gal, plus Surfactant F at rates of 1, 2, and 4 oz/100 Imp. gal, respectively. Eight ounces of fungicide solution were added to the planting hole at planting on May 29, 1968. Control plants received 8 oz of water per plant. On October 2 the plants were examined for wilt, the number of runners was counted, and sections of the petioles were plated, as previously described.

Results and discussion

Benlate was effective in controlling verticillium wilt when applied to the soil but was not effective as a foliar spray (Table 1-3). In the greenhouse Benlate applied as a soil drench up to 7 days after planting controlled verticillium wilt. In the field it was more effective in 1967 than in 1968 in controlling wilt. During the 1967 test period there was above-normal rainfall and the fungicide may have been more active than in the abnormally dry summer of 1968. In 1967 plants treated with Benlate produced more runner plants than the controls (Table 2), but this difference was not evident in the 1968 test (Table 3).

Lanstan and Lannate were not effective in controlling verticillium wilt, and Lannate was phytotoxic at the rate used.

Because infected strawberry plants do not always show wilt symptoms (Tables 2 and 3), diagnosis should be based on isolation of the pathogen from the petioles.

Table 1. Effect of Benlate on control of verticillium wilt of 'Redcoat' strawberry plants in the greenhouse

Treatment*	Visible symptoms (%)	Pathogen isolated (%)
Soil drench at planting	0	0
Soil drench 7 days after planting	0	0
Foliar spray at planting	10	25
Foliar spray 7 days after planting	5	20
Preplant root dip	0	10
Mixed with potting soil before planting	0	0
Control	10	30

* Benlate was used at 4 lb 50% WP/100 Imp. gal with Surfactant F at 4 oz/100 Imp. gal; soil applications were made using 8 fluid oz of formulation/ pot.

Table 2. Effect of three fungicide treatments on control of verticillium wilt of 'Redcoat' strawberry plants in the field - 1967

Treatment*	Visible symptoms (%)	Pathogen isolated (%)	No. of runner plants/100 mother plants
Benlate	0	0	668
Lanstan	5.8	13.3	487
Lannate	0.4	17.5	108
Control	10.5	10.8	334

* Benlate, 4 lb 50% WP/acre, 8 oz added to planting hole; Lanstan, 45 lb active ingredient/acre, tilled into 2 ft wide row; Lannate, 1.0 lb 90%/acre, 8 oz added to planting hole.

Table 3. Effect of Benlate on control of verticillium wilt of 'Redcoat' strawberry plants in the field - 1968

Benlate (lb 50% WP /acre)	Visible symptoms	Pathogen isolated	No. of runner plants/100 mother plants
4	0	11.7a*	457
2	0	44.5b	472
1	0	41.4b	459
Control	0	48.4b	446

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

The results suggest that it may be feasible to control verticillium wilt of strawberries by adding Benlate to the planting water.

Literature cited

1. Erwin, D. C., Henry Mee, and J. J. Sims. 1968. The systemic effect of 1-(butylcarbamoyl)-2-benzimidazole carbamic acid, methyl ester, on verticillium wilt of cotton. Phytopathology 58:528-529.

2. Gourley, C. O., and A. A. MacNab. 1964. Verticillium dahliae and Gliocladium roseum isolation from strawberries in Nova Scotia Can. J. Plant Sci. 44:544-549.

COOPERATIVE SEED TREATMENT TRIALS - 1969[1]

H.A.H. Wallace[2]

Abstract

Sixty-eight seed treatment chemicals were tested for their efficacy in controlling bunt of wheat (Tilletia foetida), covered smut of oats (Ustilago kolleri), covered smut of barley (U. hordei), seedling blight of barley (Cochliobolus sativus), and seed rot of flax caused by a complex of seed- and soil-borne microorganisms. Oat smut was difficult to control and the best chemical for control of seedling blight was only partially effective. As expected, the systemic fungicides usually controlled smut diseases. The value of maneb, and to a lesser extent thiram, as broad-spectrum fungicides, is indicated.

Introduction

In 1969 sixty-eight seed treatment chemicals were tested for their efficacy in controlling common bunt of wheat caused by Tilletia foetida (Wallr.) Liro, covered smut of oats caused by Ustilago kolleri Wille, covered smut of barley caused by U. hordei (Pers.) Lagerh., seedling blight of barley caused by Cochliobolus sativus (Ito & Kurib.) Drechsl. ex Dastur, and seed rots of flax caused by a complex of soil- and seed-borne microorganisms.

Materials and methods

Clean seed of 'Red Bobs' wheat (Triticum aestivum L.), naturally smutted seed of 'Vanguard' oats (Avena sativa L.), and naturally smutted seed of 'Plush' barley (Hordeum vulgare L.) were used. One gram of the appropriate smut spores was added to each 200 g of seed to ensure heavy infection. 'Herta' barley, 100% naturally infected with C. sativus, was used for the seedling blight test; and 'Linott' flax (Linum usitatissimum L.) was used for the seed rot test.

The experiment was divided into two sections for convenience (Series A and B). The source, product name, and chemical name, where available, of the treatment materials are listed in Tables 1 and 2. Res-Q and Panogen 15B (Series A) and Agrox NM and Mergamma NM (Series B) were included as standards. Each chemical was applied to 100 g of seed, or to 200 g of seed if the rate (Tables 3 and 4) was less than 1 oz per bushel, by shaking the seed in a glass jar until the seed was uniformly covered. Seed was removed from the jar after not more than 3 days, and samples of 200 seeds in paper envelopes were stored in polyethylene bags at 15C for not more than 4 weeks before seeding.

Both series of tests were carried out at Brandon and Morden, Manitoba. Each plot replicate consisted of 200 seeds planted in a row 12 ft long; all rows were planted 9 inches apart, and plots were arranged in a randomized block design. Emergence of barley infected with C. sativus and of flax was recorded 6 - 8 weeks after seeding. Disease ratings of the emerged barley plants were made at the same time by examining 100 plants from each row and rating them on a 0 - 5 scale:

$$\text{Disease rating percentage} = \frac{\text{average of numerical ratings of individual plants} \times 100}{5}$$

The percentage of smutty heads, based on counts of 200 heads per row, was recorded after the crop had headed (when infection was very heavy, assessments were based on 100 heads). The results are given as means of eight replicates, four from each planting site. The "LSD-05" is based on an analysis of the means of the treatments for each station.

Results and discussion

Smut infection of untreated seed varied from 21% to 31% for wheat, 13% to 18% for oats, and 7% to 12% for barley. Some chemicals gave complete control of all smut diseases (Tables 3 and 4); many others controlled bunt and barley smut but failed to give good control of oat smut. BEI 16 was an exception since it gave poor control of bunt but controlled smuts of oats and barley well. Emergence of untreated flax ranged from 56% to 64%. Less than half of the seed treatment chemicals increased emergence. Emergence from the untreated diseased barley seed ranged from 57% to 60%, and generally seed treatment, except when phytotoxic, increased emergence.

Generally, treatments of the TN-702 series were phytotoxic to wheat, barley, and flax when applied at 8 oz per bushel. BEJ 15 and BEJ 16 were phytotoxic to flax.

---

[1] Contribution No. 393, Research Station, Canada Department of Agriculture, Winnipeg 19, Manitoba.

[2] Plant Pathologist.

Table 1. Seed treatment materials used in the cooperative test (Series A)

Treatment no.	Source*	Product name	Chemical name
1		Untreated check	
2	Green Cross	Res-Q	hexachlorobenzene (20%) + captan (20%) + maneb (15%)
3	Nor-Am	Panogen 15B	methylmercuric dicyandiamide
4 - 6	Green Cross	"TD-"	identity not available
7	Green Cross	Ascurit	identity not available
8 - 13	Green Cross	"SWF-"	identity not available
14 - 23	Nor-Am	"EP-"	identity not available
24 - 33	Niagara	"BE-"	identity not available
34 - 39	Interprovincial	TCMTOB	2-(thiocyanomethylsulfinyl) benzothiazole
40 - 43	Interprovincial	TCMTB	2-(thiocyanomethylthio) benzothiazole
44 - 53	Hoechst	"28--"	identity not available
54	Hopkins	WOM-DB	identity not available
55 - 58	Aagrunol	"11--"	identity not available
59	Dupont	Manzate D	maneb (80%) + zinc
60		Untreated check	

* Green Cross Products, Montreal, Quebec; Nor-Am Agricultural Products Ltd., Woodstock, Illinois; Niagara Brand Chemicals, Burlington, Ontario; Interprovincial Cooperatives Ltd., Winnipeg, Manitoba; Canadian Hoechst Ltd., Montreal, Quebec; Hopkins Agricultural Chemical Co., Madison, Wisconsin; Aagrunol Chemical Works, Groningen, Holland; E. I. Dupont de Nemours and Co., Inc., Wilmington, Delaware.

Table 2. Seed treatment materials used in the cooperative test (Series B)

Treatment no.	Source*	Product name	Chemical name
61		Untreated check	
62-68	Chipman	"TF-"	identity not available
69	Dupont	Benlate	benomyl (methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate (50%)
70	Dupont Dupont	Benlate Arasan 70-S	benomyl (50%) thiram (70%) + methoxychlor (2%)
71	Dupont Chipman	Benlate Agrox NM	benomyl (50%) maneb (37.5%)
72	Dupont	Arasan 70-S	thiram (70%) + methoxychlor (2%)
73	Chipman	Agrox NM	maneb (37.5%)
74-83	Merck	"TN-702-"	identity not available
84	Uniroyal	Vitavax 40S	5,6-dihydro-2-methyl-1,4-oxathiin-3-carboxanilide

Table 2 (Continued)

Treatment no.	Source*	Product name	Chemical name
85-87	Uniroyal	Vitavax 100, 101	Vitavax + maneb
88-90	Uniroyal	Vitavax 200, 201	Vitavax + zineb
91	Uniroyal	Vitavax 300	Vitavax + maneb + zineb
92-93	Chemagro	Bay 78175	N, N^1 - dipropyl- N, N^1 - (dichlorofluoromethyl thio) sulfamide
94-95	Chemagro	Chemagro 5506	2-((1, 2, 2-trichloroethyl) dithio) propriona-mide
96	Uniroyal	Vitavax 75W	Vitavax
97	Green Cross	Res-Q Dual	hexachlorobenzene (16%), maneb (12%), cap-tan (16%), lindane (30%)
98	Rohm & Haas	Dithane M45	zinc coordinated maneb (80%)
99	Chipman	Mergamma NM	maneb (37.5%) + lindane (18.75%)
100		Untreated check	

* Chipman Chemicals Ltd., Hamilton, Ontario; E. I. Dupont de Nemours & Co., Inc., Wilmington, Delaware; Merck & Co., Inc., Rathway, New Jersey; Uniroyal (1966) Ltd., Elmira, Ontario; Chemagro Corporation, Kansas City, Missouri; Green Cross Products, Montreal Quebec; Rohm & Haas Co. of Canada Ltd., West Hill, Ontario.

† In treatments 70 and 71, the seed was treated twice, once with each fungicide at the rates indicated in Table 4.

Table 3. Results of cooperative seed treatment trials (Series A)

Treatment no.	Product name	Formu-lation *	Dosage (oz/bu)	Smutted heads (%)** Wheat	Oats	Barley	Barley seedling blight** Emergence (%)	Disease rating (%)	Flax Dosage (oz/bu)	Emergence (%)
1	Untreated check			21.88	18.64	7.22	58.4	23.8		56.2
2	Res-Q	WP	1.00	0.00					4.00	66.7
			2.00		1.94	0.00	64.0	20.1		
3	Panogen 15B	Sn	0.75	0.40	0.00	0.00	70.9	9.6	1.50	65.2
4	TD5124 A	WP	2.00	11.17	4.42	6.77	60.8	16.2	4.00	42.7
5	TD5124A + charcoal	WP	2.00	13.50	9.68	10.20	57.1	18.3	4.00	45.1
6	TD5056	WP	2.00	0.24	7.14	0.00	60.4	23.0	4.00	53.9
7	Ascurit	D	2.00	0.00	7.64	0.74	55.9	18.2	4.00	54.6
8	SWF 780	D	1.00	0.39					4.00	62.9
			2.00		0.61	0.16	65.9	20.8		
9	SWF 2250	D	1.00	0.00					4.00	62.1
			2.00		2.22	0.00	63.4	19.0		
10	SWF 2260	D	1.00	0.09					4.00	58.5
			2.00		3.32	0.30	66.3	23.6		
11	SWF 2270	D	1.25	0.00					5.00	64.1
			2.50		2.34	0.15	63.0	19.7		
12	SWF 2280	D	1.00	0.09					4.00	66.1
			2.00		4.74	0.04	65.6	19.9		
13	SWF 2290	D	1.25	0.00					5.00	63.5
			2.50		2.23	0.00	60.3	19.6		
14	EP-411C	L	0.67	0.19	0.06	0.08	59.2	23.7	2.50	57.8
15	EP-411C	L	1.25	0.08	0.06	0.08	66.1	20.7	5.00	48.4
16	EP-342-A	WP	1.00	0.13	13.05	4.55	61.5	26.0	2.00	50.8

Table 3. (Concluded)

Treatment no.	Product name	Formulation *	Dosage (oz/bu)	Smutted heads (%)**			Barley seedling blight**		Flax	
				Wheat	Oats	Barley	Emergence (%)	Disease rating (%)	Dosage (oz/bu)	Emergence (%)
17	EP-342-A	WP	2.00	0.21	14.24	8.08	51.0	22.8	4.00	52.0
18	EP-476	L	2.00	0.73	5.71	4.40	58.9	17.9	4.00	47.7
19	EP-473-B	L	5.00	19.81	8.72	5.67	53.1	17.6	5.00	53.7
20	EP-477	L	8.00	7.65	6.76	4.60	57.1	22.2	8.00	60.5
21	EP-371-A	WP	2.00	0.00	0.00	0.00	63.9	18.9	4.00	63.9
22	EP-439-B	WP	2.00	0.13	3.74	0.70	58.1	14.1	4.00	61.3
23	EP-458-A	WP	2.00	0.14	12.10	0.53	58.0	22.3	4.00	59.1
24	BEJ 11	D	3.00	4.12	3.00	0.49	55.5	24.3	3.00	52.9
25	BEJ 11	D	6.00	2.50	3.05	0.30	56.6	23.1	6.00	49.9
26	BEJ 12	L	3.00	1.75	1.98	0.44	64.9	20.1	3.00	55.6
27	BEJ 12	L	6.00	0.10	0.40	0.15	63.8	20.2	6.00	61.7
28	BEI 24	D	1.00	0.04	0.06	0.00	70.8	15.2	2.00	56.3
29	BEI 24	D	2.00	0.00	0.00	0.00	66.6	9.3	4.00	54.0
30	BEJ 14	D	1.00	0.00	3.68	0.00	61.8	19.4	2.00	63.8
31	BEJ 14	D	2.00	0.15	3.50	0.04	64.7	19.9	4.00	59.4
32	BEJ 15	L	6.00	0.70	0.10	0.68	51.9	10.3	6.00	34.4
33	BEJ 16	L	6.00	8.38	0.11	0.00	63.4	23.6	6.00	34.7
34	TCMTOB (2%)	D	1.00	11.70	11.33	6.08	60.9	24.1	2.00	55.2
35	TCMTOB (2%)	D	2.00	17.88	9.17	2.88	51.9	20.0	4.00	58.3
36	TCMTOB (10%)	D	1.00	3.49	3.20	1.07	61.0	22.7	2.00	60.9
37	TCMTOB (10%)	D	2.00	2.57	0.79	0.44	62.9	20.6	4.00	62.3
38	TCMTOB (2%)	L	0.75	8.12	10.09	7.45	57.4	25.2	1.50	60.1
39	TCMTOB (2%)	L	1.50	8.76	3.75	3.07	57.6	19.6	3.00	57.7
40	TCMTB	L	1.00	9.75	3.02	5.79	60.4	23.7	2.00	50.8
41	TCMTB	L	2.00	4.94	1.74	2.96	61.9	27.5	4.00	44.3
42	TCMTB	D	1.00	19.23	10.14	5.28	58.2	18.5	2.00	58.0
43	TCMTB	D	2.00	11.70	8.52	9.96	58.1	20.4	4.00	50.5
44	2988	D	2.00	0.67	0.23	0.04	63.6	22.3	2.00	60.6
45	2988	D	4.00	0.10	0.00	0.00	64.2	27.2	4.00	47.6
46	2988	D	6.00	0.04	0.00	0.00	59.9	21.6	6.00	49.0
47	2988	D	8.00	0.00	0.00	0.00	58.3	20.5	8.00	48.6
48	2989	D	2.00	12.30	0.00	0.04	63.6	19.4	2.00	49.0
49	2989	D	4.00	7.57	0.06	0.00	58.9	16.4	4.00	53.5
50	2989	D	6.00	3.09	0.00	0.00	61.8	18.8	6.00	52.8
51	2981	D	2.00	0.16	0.00	0.00	67.1	19.0	2.00	44.9
52	2981	D	4.00	0.04	0.00	0.00	66.9	12.5	4.00	55.6
53	2981	D	6.00	0.00	0.00	0.00	67.0	8.9	6.00	50.5
54	W-O-M-DB	D	2.00	0.04	0.35	0.04	65.8	15.5	4.00	62.8
55	1813-V25	D	2.00	14.10	1.57	4.77	61.9	19.9	4.00	48.8
56	1813-V25	D	4.00	12.30	0.55	2.53	60.7	22.6	8.00	49.3
57	1813-V10	D	2.00	20.10	6.38	6.76	61.6	21.4	4.00	51.8
58	1813-V10	D	4.00	14.40	2.44	4.59	61.8	21.1	8.00	55.1
59	Manzate D	WP	2.00	0.00	0.04	0.00	70.1	13.0	4.00	65.7
60	Untreated check			31.07	17.19	11.44	57.1	23.4		57.8
LSD	(.05)			5.91	4.44	3.22	5.7	5.9		9.3

* Formulation code: D = dust; L = liquid; Sn = solution; WP = wettable powder

** See text

Table 4. Results of cooperative seed treatment trials (Series B)

Treatment no.	Product name	Formulation*	Dosage (oz/bu)	Smutted heads (%)**			Barley seedling blight**		Flax	
				Wheat	Oats	Barley	Emergence (%)	Disease rating (%)	Dosage (oz/bu)	Emergence (%)
61	Untreated check			29.06	13.25	11.04	60.1	22.0		64.5
62	TF15-69	D	2.00	0.00	0.46	0.00	65.8	15.8	4.00	66.0
63	TF16-69	D	2.00	0.00	0.72	0.00	65.8	18.8	4.00	70.5

Table 4. (Con't)

Treatment no.	Product name	Formulation*	Dosage (oz/bu)	Smutted heads (%)** Wheat	Oats	Barley	Barley seedling blight** Emergence (%)	Disease rating (%)	Flax Dosage (oz/bu)	Flax Emergence (%)
64	TF17-69	D	2.00	0.00	0.39	0.08	68.1	19.9	4.00	73.9
65	TF20-69	D	2.00	23.25	16.35	15.63	57.1	26.5	4.00	57.8
66	TF21-69	D	2.00	0.00	0.00	0.04	56.5	33.0	4.00	56.1
67	TF22-69	D	2.00	0.06	0.89	0.00	65.4	16.7	4.00	68.3
68	TF23-69	D	2.00	0.18	1.19	0.26	70.4	14.9	4.00	69.7
69	Benlate	D	2.00	0.00	0.00	0.00	55.4	31.5	4.00	58.8
70	Benlate + Arasan 70-S	SL	2.00 1.00	0.00	0.00	0.00	56.6	25.4	4.00 2.00	66.8
71	Benlate + Agrox NM	D	2.00 1.00	0.00	0.00	0.43	69.6	28.0	4.00 2.00	70.3
72	Arasan 70-S	WP	1.00 2.00	0.26	1.85	0.38	61.5	21.1	4.00	70.0
73	Agrox NM	D	1.00 2.00	0.00	0.96	0.04	68.4	18.0	4.00	71.6
74	TN-702-50-3	L	8.00	0.00	0.00	0.00	60.7	12.0	8.00	37.4
75	TN-702-50-5	L	4.00	0.00	0.00	0.00	61.7	11.6	4.00	41.1
76	TN-702-50-6	L	4.00	0.06	0.00	0.00	66.8	16.1	4.00	43.9
77	TN-702-50-6	L	8.00	0.00	0.23	0.00	59.0	11.9	8.00	33.9
78	TN-702-50-7	L	8.00	0.04	0.00	0.16	61.1	20.7	8.00	38.9
79	TN-702-50-8	L	8.00	1.20	2.79	2.30	58.6	27.3	8.00	52.0
80	TN-702-50-9	D	2.00	0.94	5.69	2.95	54.5	24.6	4.00	62.3
81	TN-702-50-9	D	4.00	0.00	2.53	0.79	59.3	29.0	4.00	52.8
82	TN-702-50-11	L	8.00	0.04	0.00	0.08	52.4	7.7	8.00	26.3
83	TN-702-50-12	L	8.00	0.40	1.81	0.10	54.1	25.7	8.00	51.6
84	Vitavax 40S	L	3.80	0.00	0.00	0.00	78.7	21.1	3.80	62.7
85	Vitavax 100	D	3.60	0.08	0.00	0.00	84.0	15.9	6.00	70.9
86	Vitavax 101	D	4.00	0.00	0.00	0.00	81.3	10.0	4.00	68.9
87	Vitavax 101	D	8.00	0.00	0.00	0.00	78.1	9.2	8.00	73.7
88	Vitavax 200	D	3.50	0.00	0.00	0.00	83.1	14.9	3.50	72.7
89	Vitavax 201	D	4.50	0.00	0.00	0.00	80.4	11.1	4.50	66.3
90	Vitavax 201	D	9.00	0.00	0.00	0.00	80.4	11.2	9.00	69.9
91	Vitavax 300	D	4.00	0.00	0.00	0.00	79.3	12.2	4.00	75.3
92	78175	WP	2.00	0.59	3.58	3.52	55.9	23.6	4.00	52.9
93	78175	WP	4.00	0.14	1.70	3.05	57.9	26.2	8.00	55.6
94	5506	WP	1.00	0.35	0.23	0.08	64.7	20.5	2.00	61.0
95	5506	WP	2.00	0.21	0.00	0.00	60.8	72.0	4.00	21.3
96	Vitavax 75W	D	2.00	0.23	0.00	0.00	65.9	75.8	4.00	14.6
97	Res-Q Dual	D	1.25 2.50	0.00	4.64	0.20	70.1	67.3	5.00	23.8
98	Dithane 45	D	2.00	0.00	0.69	0.00	68.1	71.4	4.00	23.8
99	Mergamma NM	D	2.00	0.00	0.54	0.14	74.4	76.9	4.00	22.7
100	Untreated check			29.75	15.77	12.17	62.8	57.8		24.9
LSD	(.05)			3.48	4.19	1.91	8.3	4.8		

* Formulation code: D = dust; L = liquid; SL = slurry; WP = wettable powder
** See text

Among the better all-round treatments were Panogen 15B (mercurial), EP 371A (unidentified), Manzate D (80% maneb), and the Vitavax formulations that contained maneb and thiram. However, many other chemicals gave results that are not statistically different from the above ($P > 0.05$).

Acknowledgments

The writer thanks members of the staff of the Morden Research Station and the Brandon Experimental Farm for their cooperation and assistance, and Dr. R. J. Baker for the statistical analyses.

PLANT-PARASITIC NEMATODE GENERA ASSOCIATED WITH CROPS IN ONTARIO IN 1968

Th.H.A.Olthof, J.L.Townshend, J.W.Potter[1], *and A.Cornelisse*[2]

The Ontario Nematode Diagnostic and Advisory Service processed 442 soil samples for growers in 1968, as compared to 724 samples in 1967. The decline of 39% was mainly due to a drop of 42% in the number of tobacco soil samples received, which reflected a combination of weather conditions unfavorable for showing nematode-induced plant damage and the increasing practice of regularly fumigating the soil without having a nematode assessment carried out. The samples originated from 29 crops covering the spectrum of agricultural activities in southern Ontario. Actively moving nematodes and cysts were recovered from the soil with techniques described earlier (1).

Root-lesion nematodes, Pratylenchus spp., were present in 20 of the crops sampled. The average number per pound of soil was considerably smaller than in previous years (1, 3, 4). Pin nematodes, Paratylenchus spp., were present in most samples of forced winter rhubarb, with populations ranging from 600 to 13,200 per pound of soil. Work is in progress on the association of this nematode with yield.

Populations of dagger nematodes, Xiphinema spp., and ring nematodes, Criconemoides spp., were again fairly low in 1968. As pointed out earlier (1), this may be due to the rather inefficient extraction technique in use.

The number of samples containing spiral nematodes, Helicotylenchus spp., was very much smaller than in 1967, whereas stunt nematodes, Tylenchorhynchus spp., occurred more commonly in 1968. The reason for this shift is not known. Only two samples were received this year infested with the root-knot nematode, Meloidogyne spp. This also contrasts sharply with 1967, when 35 samples contained this nematode.

The oat cyst nematode, Heterodera avenae Wollenweber, was found in barley and corn, and the sugar beet nematode, H. schachtii Schmidt, in rhubarb and spinach. The lance nematode, Hoplolaimus spp., was encountered once in tobacco near Simcoe, Ontario. The bulb and stem nematode, Ditylenchus dipsaci (Kühn) Filipjev, was found parasitizing onions in muck soil in the Leamington Marsh, Ontario. This nematode has been shown to be a serious threat to onions in southwestern Ontario (2). An unidentified species of Ditylenchus (not D. dipsaci or D. myceliophagus) damaged mushrooms in a single house in southern Ontario. The nematode probably originated from the sawdust insulation, as large numbers were recovered from it and the heaviest damage occurred in the top trays.

As in previous years, tobacco roots included with samples were rated visually for severity of black root rot caused by the fungus Thielaviopsis basicola (Berk. & Br.) Ferr. Of 151 samples rated black root rot was absent in 33, while 45 showed a trace infection, 32 were lightly infected, 29 moderately and 12 severely. The incidence and severity of the disease was considerably less than in 1967.

Literature cited

1. Olthof, Th. H. A., J. L. Townshend, J. W. Potter, and H. D. Francis. 1967. Plant-parasitic nematode genera associated with crops in Ontario in 1967. Can. Plant Dis. Surv. 47:110-111.

2. Sayre, R. M., and W. B. Mountain. 1962. The bulb and stem nematode (Ditylenchus dipsaci) on onion in southwestern Ontario. Phytopathology 52:510-516.

3. Townshend, J. L. 1965. Plant parasitic nematode genera associated with crops in Ontario in 1964 and 1965. Can. Plant Dis. Surv. 45: 131-133.

4. Townshend, J. L., Th. H. A. Olthof, and J. E. Staples. 1966. Plant parasitic nematode genera associated with crops in Ontario. Can. Plant Dis. Surv. 46:114-145.

[1] Nematologists, Research Station, Canada Department of Agriculture, Vineland Station, Ontario.

[2] Technician, Horticultural Research Institute of Ontario, Ontario Department of Agriculture and Food, Vineland Station, Ontario.

Table 1. Plant parasitic nematodes associated with Ontario crops in 1968

Crop	Praty-lenchus	Paraty-lenchus	Xiphi-nema	Cricone-moides	Helicoty-lenchus	Tylencho-rhynchus	Meloido-gyne larvae	Hetero-dera larvae	Hoplo-laimus	Dity-lenchus
Apples (2)*	150/2**	0/0	50/2	0/0	0/0	150/2	0/0	0/0	0/0	0/0
Barley (3)	200/3	275/2	50/1	0/0	0/0	0/0	0/0	2600/2	0/0	0/0
Beets (1)	400/1	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Buckwheat (4)	188/4	150/1	50/1	0/0	0/0	100/3	0/0	0/0	0/0	0/0
Carrots (4)	200/2	100/2	0/0	0/0	0/0	0/0	3000/1	0/0	0/0	0/0
Cabbage (2)	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Corn (6)	930/5	150/2	0/0	0/0	0/0	200/1	0/0	833/3	0/0	0/0
Cherries (sweet) (10)	985/10	183/3	0/0	0/0	0/0	200/1	0/0	0/0	0/0	0/0
Cherries (sour) (2)	3800/1	200/2	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Cauliflower (1)	350/1	50/1	20/1	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Hay (1)	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Lettuce (4)	1000/1	0/0	0/0	0/0	0/0	300/1	0/0	0/0	0/0	0/0
Mushroom (4)	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Onion (3)	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	2600/1
Parsnips (1)	1500/1	0/0	200/1	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Peat moss (2)	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Peaches (29)	977/24	872/18	413/4	150/3	0/0	392/6	0/0	0/0	0/0	0/0
Potatoes (10)	729/7	30/2	0/0	0/0	0/0	83/3	0/0	0/0	0/0	0/0
Radish (1)	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Rhubarb (18)	786/14	5807/15	10/1	0/0	0/0	0/0	0/0	6721/7	0/0	0/0
Roses (12)	353/6	200/2	0/0	700/1	0/0	2260/2	0/0	0/0	0/0	0/0
Strawberries (14)	422/9	412/5	35/2	0/0	0/0	167/3	0/0	0/0	0/0	0/0
Soybean (1)	50/1	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Shrubs (28)	216/7	125/4	100/7	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Spinach (6)	50/1	100/4	0/0	0/0	0/0	0/0	0/0	867/3	0/0	0/0
Tomatoes (2)	0/0	0/0	0/0	0/0	0/0	0/0	50/1	0/0	0/0	0/0
Turf (1)	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Tobacco (269)	1069/220	209/66	37/16	50/2	47/3	169/31	0/0	0/0	50/1	0/0
Wheat (1)	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Total (442)	717/320	242/129	97/36	300/6	47/3	402/53	1525/2	2755/15	50/1	2600/1

* Number of soil samples processed.

** Average number of nematodes per lb of soil/number of samples containing the nematode.

THE SEASONAL OCCURRENCE OF FUNGAL AND BACTERIAL DISEASES OF CRUCIFERS IN ONTARIO IN 1967 AND 1968[1]

A.A. Reyes[2]

This paper reports on the time of year at which fungal and bacterial diseases of crucifers were observed in Ontario in 1967 and 1968. It is intended as an aid for those wishing to schedule field trips to collect or observe diseases of crucifers.

The 1967 survey covered all of the main vegetable growing areas of the province. The over-all prevalence of diseases for that year has been reported (1, 2, 3). The 1968 survey covered only the southern Ontario counties listed by Reyes, et al. (2). The survey methods used in 1967 and 1968 were the same.

The results of the 1967 and 1968 surveys are presented in Table 1. Only seedling diseases were observed in May; most root diseases (yellows, clubroot, etc.) were first observed in June; foliar diseases in July; and downy and powdery mildews in September. The total number of diseases observed was the same in 1967 and 1968. There were twice as many fungal diseases as bacterial diseases, a relationship that was previously reported (2). Generally, fungal diseases appeared earlier in the season (May or June) than bacterial diseases (July). The number of diseases progressed gradually until a peak was reached which coincided with the onset of host maturity in September. The greatest prevalence of diseases did not occur during the periods of maximum temperature (June, July, August) or precipitation (June).

There were variations in the time of occurrence of seedling diseases in the four regions but the number of observations was too limited to allow conclusive interpretation.

No attempt was made to establish a correlation between the types and severity of diseases during successive growth stages of a particular plant, since few fields were visited more than once during the year.

The cooperation and assistance of Fruit and Vegetable Extension Specialists of the Ontario Department of Agriculture and Food is gratefully acknowledged.

Literature cited

1. Reyes, A. A., G. H. Comly, L. F. Mainprize, D. A. Pallett, C. A. Warner, and W. A. Willows. 1968. Fungal and bacterial diseases of crucifers and cucurbits in Western Ontario in 1967. Can. Plant Dis. Surv. 48:95-96.

2. Reyes, A. A., J. R. Chard, A. Hikichi, W. E. Kayler, K. L. Priest, J. R. Rainforth, I. D. Smith, and W. A. Willows. 1968. A survey of diseases of vegetable crops in Southern Ontario in 1967. Can. Plant Dis. Surv. 48: 20-24.

3. Reyes, A. A., R. W. Daniels, E. N. Estabrooks, C. C. Filman, L. F. Mainprize, W. M. Rutherford, C. A. Warner, and H. M. Webster. 1968. A survey of fungal and bacterial diseases of vegetable crops in Eastern and Central Ontario in 1967. Can. Plant Dis. Surv. 48:53-55.

---

[1] Contribution No. 182. Research Station, Canada Department of Agriculture, Vineland Station, Ontario.

[2] Research Scientist, Research Station, Canada Department of Agriculture, Vineland Station, Ontario.

Table 1. Observations on the seasonal occurrence of crucifer diseases in Ontario during 1967 and 1968

Crop	Disease and pathogen	Occurrence					
		May	June	July	August	September	October
1967 (whole province)							
Brussels sprouts	Black rot (Xanthomonas campestris)					Sev.[a] 1/2[b](C)[c]	
Cabbage	Black rot (X. campestris)					Sev. 4/8 (C)	Sl. 1/7 (W)
	Clubroot (Plasmodiophora brassicae)		Tr. 1/8 (C)			Tr. 1/2(S), tr. 1/8(C), sl. 1/2(S), mod. 1/7(W)	
	Damping-off (Fusarium spp.)			Tr. 1/2(C)			
	Drop (Sclerotinia sclerotiorum)		Tr. 2/4 (S)				
	Leaf spot (Alternaria brassicae)					Sev. 3/8(C)	
	Wirestem (Rhizoctonia solani)		Tr. 1/2(W), sl. 1/2(W)				
	Yellows (Fusarium oxysporum f. conglutinans)		Tr. 1/8(W), tr. 1/2(S), sl. 1/8(C), sl.-sev. 3/8(W)				
Cauliflower	Bacterial leaf spot (Xanthomonas sp.)					Mod. 3/4(C)	
	Black rot (X. campestris)			Tr. 2/7(S)	Mod. 1/7(S)	Mod. 3/4(C)	
	Clubroot (P. brassicae)					Tr. 1/7(S), sl. 1/7(S), mod. 1/7(S)	
	Damping-off (Pythium spp., R. Solani, Fusarium spp.)		Tr. 1/5(W), tr. 2/4(S)				
	Drop (S. sclerotiorum)		Tr. 1/1(S), sl. 1/5(W)	Tr. 1/7(S)			
	Leaf spot (Alternaria brassicae)				Sl. 1/4(C)	Tr. 1/2(S), sev. 1/2(S)	
	Leaf spot (cause unknown, bacteria isolated)					Mod. 1/1(S)	
	Root rot (Fusarium spp.)				Tr. 1/1(S)		
	Wirestem (R. solani)		Tr. 1/1 (S)	Tr. 1/7(S)			
	Yellows (F. oxysporum f. conglutinans)				Sl. 1/5(W)		
Turnip	Clubroot (P. brassicae)					Mod. 1/4(C)	
1968 (southern Ontario)							
Broccoli	Clubroot (P. brassicae)					Sev. 1/1	
Brussels sprouts	Black root (X. campestris)						Mod. 1/1
Cabbage	Bacterial soft rot (Erwinia carotovora)			Tr. 1/3			
	Black rot (X. campestris)			Tr. 1/3		Mod. 1/4	
	Clubroot (P. brassicae)				Tr. 1/5, sl. 2/5, mod. 1/5		
	Damping-off (Fusarium spp., R. solani, Pythium spp.)	Tr. 1/2					
	Downy mildew (Peronospora parasitica)					Tr. 1/4	Mod. 1/2
	Drop (S. sclerotiorum)			Tr. 1/3			Tr. 1/2
	Wirestem (R. solani)	Tr. 1/2		Tr. 1/3			
	Yellows (F. oxysporum f. conglutinans)				Sev. 1/5	Sl. 1/4, mod. 1/4	Tr. 1/2
Cauliflower	Bacterial soft rot (E. carotovora)					Tr. 3/9, sl. 1/9	Tr. 1/2
	Black rot (X. campestris)					Tr. 3/9, sl. 1/9, mod. 1/9, sev. 2/9	Sev. 2/2
	Leaf spot (Alternaria brassicae)					Tr. 3/9	Tr. 1/2
	Leaf spot (cause unknown, bacteria isolated)					Tr. 2/9, sl. 1/9, sev. 2/9	Sl. 2/2
	Wirestem (R. solani)	Tr. 1/1	Tr. 1/1				
Pak-choi[d]	Clubroot (P. brassicae)			Mailed specimen			
Radish	Downy mildew (P. parasitica)					Sl. 1/2, mod. 1/2	
Turnip	Clubroot (P. brassicae)		Tr. 1/2, sl. 1/2		Sev. 1/1		
	Powdery mildew (Erysiphe polygoni)					Tr. 1/1	

[a] Tr. (trace) = 1-10% of plants affected in the field; sl. (slight) = 11-30%; mod. (moderate) = 31-60%; sev. (severe) = 61-100%.

[b] Number of fields in which the disease was found/number of fields inspected in the region during the month.

[c] C = Central Ontario, E = Eastern, S = Southern, and W = Western.

[d] Brassica chinensis L.

INCIDENCE AND EFFECTS OF WHEAT SPINDLE STREAK MOSAIC IN ESSEX AND KENT COUNTIES, ONTARIO, 1967-68

L.F. Gates[1]

Abstract

Symptoms of wheat spindle streak mosaic were present in an average of 49.6% of the plants in winter wheat fields surveyed in Essex and Kent counties, Ontario, in 1967 and 1968. It is estimated that the average loss in grain yield was 5% in both years.

A mosaic disease of winter wheat caused by a soil-borne virus has been recognized in southern Ontario since 1957 (1-4). This disease, characterized by a mosaic which includes light green to bright yellow lens-shaped spots or short streaks, and straw-colored to light brown necrotic blotches, is now designated wheat spindle streak mosaic (4). In recent years this mosaic has been widespread in southwestern Ontario, showing especially in the unusually cold springs of 1967 and 1968, when it imparted an overall brownish discoloration to many fields. Surveys of this disease were made in Essex and Kent counties, Ontario, in 1967 and 1968.

Surveys

Surveys were made during May, when the bright yellow spotting on the spring foliage showed most clearly. In randomly selected fields, counts were made on enough 1-yard or 1-foot lengths of row to arrive at a consistent estimate of the proportion of infected plants.

About one half the number of fields of wheat examined in 1967 and about one quarter of those examined in 1968 were completely infected (Table 1). In Essex Co. only a small proportion of fields was completely free from disease. Heavily infected fields were found in all areas visited in both counties. The overall infection for 119 fields examined in the two seasons was 49.6%.

Estimates of loss caused by the disease

Fields were selected in May near Harrow which showed areas where the leaves were discolored by the disease, interspersed with areas of green, symptomless plants. Six pairs of plots were staked in each of four fields in each season. One plot of each pair was in an area where essentially all plants were infected, and the other was in an area as close by as possible where the plants showed no symptoms. Each plot was 0.84 m^2, and care was taken to avoid poorly drained areas, thinly drilled rows, and any other obvious difference between the two areas except for the presence or absence of the disease symptoms. Ripe heads were collected from the plots, counted and threshed, and the grain was weighed.

These comparisons showed reductions in grain yield caused by the disease in all eight fields, significant at or close to $P = 0.05$ in five of them, resulting in an average loss of 13% in grain (Table 2). The disease reduced the number of heads per m^2 by an average of 6.1%; this effect reached significance at $P = 0.05$ for only one of the individual fields, but it was consistent.

This estimate of losses from disease assumes that other adverse conditions are not consistently associated with the disease, and obvious effects of this type were avoided when staking the plots. However, in field F in 1968 the stand grew less vigorously on the diseased areas, and allowed the development of weeds which probably accentuated yield loss. If this field were excluded, the figures from the other seven fields indicate an average yield loss of 10.1% due to the disease.

This method of estimating loss also assumes that the symptomless plants were not infected, presumably because the vector or virus had not reached their part of the field. If they were infected and damaged, but for some reason did not show symptoms, the comparisons made would underestimate the effects of the disease.

If the figure of 10.1% yield loss from complete infection is used, an average of 49.6% infected plants would result in a loss of 5.0% per year in yield in Essex and Kent counties in 1967-68. The combined wheat acreage of Essex and Kent is about 70,000 and at an average yield of 45 bushels per acre this loss would represent 2.2 bushels per acre over the two counties as a whole. In fields with all plants infected, loss due to the virus would be about 4.5 bushels per acre.

Acknowledgments

This work was carried out in association with Dr. J.T. Slykhuis, whose advice is gratefully acknowledged.

[1] Plant Pathologist, Research Station, Canada Department of Agriculture, Harrow, Ontario.

Table 1. Incidence of wheat spindle streak mosaic in Essex and Kent counties in 1967 and 1968

Year	County	Number of fields examined	Number of fields with: No disease	Up to 50% diseased plants	51-95% diseased plants	All plants infected	Average infection for all fields (%)
1967	Essex	29	2	9	4	14	66.2
1968	Essex	35	3	15	7	10	52.2
	Kent	55	20	14	6	15	39.1

Table 2. Comparison of areas of healthy plants with areas of plants infected with wheat spindle streak mosaic

Year and field	Number of heads/m^2			Yield of grain (g/m^2)			
	Healthy	Infected	Reduction (% of healthy)	Healthy	Infected	L. S. D. (P = 0.05)	Reduction (% of healthy)
1967 A	402	395	1.8	360	323	14	10.4
B	421	402	4.6	368	322	90	12.8
C	431	393	8.7	420	335	68	20.1
D	492	464	5.5	445	414	60	7.1
1968 E	579	560	3.3	452	427	39	5.7
F	536	460	13.9	365	233	67	35.9
G	434	422	2.8	396	356	44	9.9
H	475	441	6.9	421	396	27	5.9
Mean	471	442	6.1	403	351	31	13.0

Literature cited

1. Slykhuis, J. T. 1960. Evidence of soil-borne mosaic of wheat in Ontario. Can. Plant Dis. Surv. 40:43.
2. Slykhuis, J. T. 1961. The causes and distribution of mosaic diseases of wheat in Canada in 1961. Can. Plant Dis. Surv. 41:330-343.
3. Slykhuis, J. T. 1967. Virus diseases of cereals. Rev. Appl. Mycol. 46:401-429.
4. Slykhuis, J. T. 1969. Factors determining the development of wheat spindle streak mosaic caused by a soil-borne virus in Ontario. Phytopathology. (In press)

SEED INFECTION OF BARLEY BY COCHLIOBOLUS SATIVUS AND ITS INFLUENCE ON YIELD[1]

R.V. Clark[2] *and V.R. Wallen*[3]

Abstract

Yield losses from planting barley seed heavily infected with Cochliobolus sativus were not significant at the normal planting rate. Seed infection reduced seedling emergence by up to 38% in the field, but the reduction in number of plants per row had to be more than 50% of the control before significant yield decreases occurred. Treatment of the seed with mercury fungicides eliminated the reduction in emergence caused by C. sativus and also provided an additional increase, due possibly to protection from other soil microorganisms, but there was no significant improvement in seed yields. Treatment of the seed improved kernel weight slightly. Only a few plants grown from infected seed became infected by the causal organism under normal greenhouse conditions. When they were grown under conditions of high humidity, however, almost as many plants were infected as there were infected seeds. Disease development on plants in high humidity was controlled by treatment of the infected seed with mercury fungicides.

Introduction

In 1967 a severe infection of spot blotch and head blight caused by Cochliobolus sativus (Ito and Kurib.) Drechs. ex Dastur (imperfect state Bipolaris sorokiniana (Sacc. in Sorokin.) Shoemaker (syn: Helminthosporium sativus PK & B) occurred on the barley crop in Prince Edward Island. There was considerable concern about what effect the heavily infected seed from this crop might have on the subsequent crop if it were used for planting. Some years ago Greaney and Wallace (1) and Machacek et al. (2) reported that seed infection by this fungus lowered germination but had little effect on yield. They found that chemical seed treatments improved germination of infected seed but had little influence on yield.

The availability of this heavily infected barley seed provided an opportunity to establish further the relationship between yield losses and seed infection by C. sativus as well as to determine the benefits obtained from treating this seed with fungicides.

Materials and methods

The barley (Hordeum vulgare L.) variety Herta was used in all experiments. Heavily infected seed was obtained from Charlottetown, Prince Edward Island, relatively healthy seed from Saskatoon, Saskatchewan, and commercial grade seed from Ottawa, Ontario. The amount of internal infection by C. sativus was determined by plating aseptically 100 surface sterilized (10 minutes in a Javex solution adjusted to 2% available chlorine as sodium hypochlorite) seeds from each sample on malt agar. Fungus colonies were identified after 10 days' incubation in the dark at 23-24 C.

In field tests, 4-row plots with 100 seeds (unless otherwise specified) per 3-m long rows were planted for each treatment and replicated four times. Seedling emergence was recorded approximately 1 month after seeding. At maturity, the plants were removed from 30 cm at the ends of the two centre rows of each plot and the remainder of the plants in these rows were harvested for seed yield and 1000-kernel weight determinations. Threshing was done with a cyclone thresher.

In greenhouse tests, the seed was planted in steamed soil in flats, 100 seeds per flat. The plants were grown at 25-27 C, in one instance using normal humidification and in another instance using a "mist-bed" where the humidity was maintained at 95-100%. Emergence was recorded 1 month after seeding, and approximately 3 weeks later all plants were harvested and classified as being healthy or infected by C. sativus.

Three chemical fungicides[4] were used to treat the seed at the following rates per 45.5 kg (100 lb) seed: Ceresan M (7.7% ethyl mercury p-toluene sulfonanilide), 42 g (1½ oz); Liqui-San 10L (1.97% methyl mercury 2-3 dihydroxypropylmercaptide and 0.42%

---

[1] Contribution No. 227, Research Station, Ottawa; and No. 665, Cell Biology Research Institute.

[2] Research Station, Canada Department of Agriculture, Ottawa, Ontario.

[3] Cell Biology Research Institute, Canada Department of Agriculture, Ottawa, Ontario.

[4] Ceresan M and Benlate were supplied by Dupont of Canada Ltd., Toronto, Ontario and Liqui-San 10L by Green Cross Products, Montreal Quebec.

methyl mercury acetate), 63 g (2¼ oz), and Benlate (50% benomyl [1-(butyl carbamoyl)-2-benzimidazole carbamic acid, methyl ester]), 112 g (4 oz). Each fungicide was added to a 115 g sample of seed in a 1-litre widemouth Erlenmeyer flask, which was rotated for 5 minutes on a machine designed to give good coverage of the seed. The treated seed was left in the stoppered flasks for at least 48 hr before planting.

Results

Field tests

The percentage of seeds infected by C. sativus in the Prince Edward Island sample ranged from 97 to 100 and in the Saskatchewan sample from 2 to 4. The commercial seed from Ottawa was divided into two groups of large or normal sized seed and small seed. The group of large seed was 60% infected by C. sativus and the small 55%. Seed from the four samples was treated with the mercury fungicide Liqui-San 10L, the non-mercury systemic fungicide Benlate, and with a combination of the two applied at the same rate as used individually.

Emergence of heavily infected untreated Prince Edward Island seed was 38% lower than that of untreated seed from Saskatchewan (Table 1). Treatment of the seed with Liqui-San 10L increased the emergence of all samples significantly and of the heavily infected seed by 100%. Benlate did not improve emergence over the controls. When Benlate was combined with Liqui-San 10L, emergence of the Saskatchewan seed was somewhat better than with Liqui-San 10L alone; emergence of the other samples was similar to that with Liqui-San alone. A significant interaction occurred between the seed treatments and the seed sources indicating that the emergence of the seedlings from the various sources did not follow the same pattern with all the fungicides.

Comparable differences in seed yields were not found. There was only a small difference in yield between the heavily and the lightly infected untreated seed. In most instances treatment of the seed with chemicals gave no improvement in yield. Neither Liqui-San 10L nor Benlate improved the yield of any of the seed samples. Liqui-San 10L combined with Benlate gave a small improvement in yield with the Saskatchewan and Prince Edward Island samples.

The data on 1000-kernel weights were not consistent but there was an indication that seed treatment increased them.

In another test, seed from Prince Edward Island and Saskatchewan were combined in different proportions so that two intermediate levels of infection of approximately 33% and 66% could be compared with the original samples. Seed of the four infection levels was treated with Liqui-San 10L and Ceresan M. Ceresan M was found by Mills and Wallace (3) to be the most effective chemical seed treatment for controlling C. sativus on barley in the laboratory. The difference between the emergence of the heavily and the lightly infected seed samples (Table 2) was similar to that in the previous test. At the 33% and 66% infection levels emergence was intermediate. Both chemical seed treatments improved the emergence of all levels of infected seed and the emergence

Table 1. Seedling emergence, seed yield and 1000-kernel weight of barley grown in the field from Cochliobolus sativus infected seed from three sources and treated with two fungicides

	Emergence (Plants/row)				Seed yield (g/plot)				1000-kernel weight (g)			
Seed source	Control	Liqui-San	Benlate	Liqui-San & Benlate	Control	Liqui-San	Benlate	Liqui-San & Benlate	Control	Liqui-San	Benlate	Liqui-San & Benlate
Saskatchewan	71.2	84.0	71.0	93.2	376.5	370.0	356.5	399.2	37.9	43.0	42.0	41.2
P. E. I.	44.2	88.0	45.5	86.5	366.0	333.5	289.0	398.2	40.4	39.7	39.2	42.8
Ottawa #1[1]	62.0	90.5	52.0	89.2	396.5	396.0	337.5	380.5	39.5	40.5	39.9	39.5
Ottawa #2[2]	65.7	88.0	67.5	87.7	394.5	358.0	359.7	373.0	41.0	39.3	41.7	42.5
L. S. D. (1%)												
seed sources			13.2				N. S.				N. S.	
fungicides			13.2				N. S.				0.9	

[1] large seed

[2] small seed

of Prince Edward Island seed was increased to the same level as the Saskatchewan seed. There were no significant differences in seed yields among the various infection levels or between treated and untreated seed. Yield from the heavily infected seed was 11% lower than that from the lightly infected seed. Both seed treatments reduced yields at the two lower levels of infection and increased yields at the two higher levels. In general, yields were better with Ceresan M than with Liqui-San 10L. There was an indication that Liqui-San 10L increased the 1000-kernel weight of the subsequent crop.

A third field test was carried out to determine if different numbers of plants per row as well as various levels of seed infection would have any effect on seed yields. This was done by planting untreated and Ceresan M treated seed of the same infection levels as in the previous experiment at 5-, 10-, 15- and 20-cm spacings within the 3-m-long rows. This meant that rows at 10-, 15- and 20-cm spacings had approximately 75%, 50%, and 25% as many plants as rows with seed placed at 5-cm spacings (Table 3). The relative emergence was about the same as in the previous two experiments but, due to the increased size of the experiment, variablility was greater. Seed yields were also more variable and there was a greater reduction in the yield from the highly infected seed compared with the lightly infected seed. Treatment of the seed, however, gave no significant improvement in yield. Seed spacing per row and the resulting number of plants per plot did influence yields. Plots planted with untreated seed containing 33% and 98% infection and treated seed with 98% infection and spaced 15 cm apart yielded significantly less than those planted 5 cm apart. There was no significant yield difference between 5- and 10-cm spacings. Therefore there had to be more than a 50% reduction in the number of plants per row before significant yield losses were observed. A significant interaction occurred for yields between seed spacings and infection levels, indicating that significant losses did not occur at all infection levels. However there was no significant third order interaction. The kernel weight data were again inconclusive, but there was a trend toward increased kernel weight as the number of plants per row decreased.

Greenhouse tests

Samples of the same seed that was used in the first two field experiments were grown in the greenhouse. Seedling emergence was similar to that found in the field tests (Tables 4 and 5), except that overall emergence was considerably higher in the greenhouse than in the field and there was less difference between the highly infected Prince Edward Island seed and the lightly infected Saskatchewan seed. The mercury fungicides increased emergence of all samples, while Benlate slightly increased that of two samples but gave a lower emergence with the other two. At the normal humidity level very few C. sativus infected plants were found, even from the heavily infected seed (Table 4). In the greenhouse "mist-bed" (Table 5), where the relative humidity was maintained at approximately 95%, more than 90% of the plants from the heavily infected untreated seed were infected by C. sativus. The lower infection levels also produced considerable disease. Plants from the treated seed were relatively free of symptoms and developed about the same amount of disease as those grown in conventional greenhouse conditions.

Discussion

Infection of barley seed by C. sativus causes a considerable reduction in seedling emergence especially if the percentage infection is high. The fact that reduction in emergence can be eliminated by the treatment of the seed with mercury fungicides agrees with the findings of Greaney and Wallace (1). Emergence in all seed samples, including those lightly infected, was improved by treatment. The lower emergence in the untreated seed, however, was not followed by a comparable reduction in seed yield, and conversely the increased emergence of the heavily infected treated seed did not give a significant increase in yield. The reliability of yield data from small plots is sometimes questionable. Our yield data had a relatively low coefficient of variation (12.5%, Tables 1 and 2) and thus should be reliable. Our results agree with those found earlier by Machacek et al. (2), and there seems to be no question that the use of seed heavily infected by this fungus has little influence on the eventual yield of the crop.

There appears to be little practical value in treating barley seed infected with C. sativus with chemical fungicides. The greenhouse tests showed that under normal conditions of humidity (Table 4) very few of the seedlings from the heavily infected untreated seed were infected by the fungus. Furthermore, even when emergence was reduced in the field, it had to be reduced by more than 50% before there was a significant drop in yield (Table 3); and, on the average, there was no yield benefit from treating the seed. There was a benefit from seed treatment when the humidity in the greenhouse was maintained at a high level (Table 5), as most of the young seedlings from the treated seed were not infected by C. sativus, whereas the majority of those from the untreated seed were infected. Occasionally, unusual weather conditions of high relative humidity, dew point, and temperature occur, as in the Maritime Provinces in 1967. In this situation, treatment of the seed with chemicals might have prevented the extensive build-up of the disease that occurred by crop maturity. Such a heavy seed infection would appear to be due primarily to weather conditions rather than to a heavy inoculum load on the seed. The progeny of plants grown from heavily infected seed in these tests were found to be relatively disease-free and the seed produced by them was as disease-free as that produced by plants from clean seed.

Table 2. Seedling emergence, seed yield, and 1000-kernel weight of barley grown in the field from seed infected with four levels of Cochliobolus sativus and treated with two fungicides

Infection level (%)	Emergence (plants/row)			Seed yield (g/plot)			1000-kernel weight (g)		
	Control	Ceresan M	Liqui-San	Control	Ceresan M	Liqui-San	Control	Ceresan M	Liqui-San
2	81.5	86.6	88.0	497.5	491.2	450.5	39.0	37.3	40.8
33	69.8	81.3	86.2	509.7	469.0	456.5	39.3	39.1	38.1
66	61.4	95.7	85.7	439.7	566.7	462.2	37.8	38.1	39.6
98	52.8	85.9	85.5	442.7	468.7	491.5	36.8	36.8	40.0
L. S. D. (1%)									
infection levels		7.5			N. S.			N. S.	
fungicides		7.5			N. S.			N. S.	

Table 3. Seedling emergence, seed yield, and 1000-kernel weight of barley seeded in the field at 5-, 10-, 15-, and 20-cm distances within the row with Ceresan M-treated and untreated seed with four levels of Cochliobolus sativus

Seed treatment	Infection level (%)	Emergence (plants/row)				Seed Yield (g/plot)				1000-kernel weight (g)			
		5 cm	10 cm	15 cm	20 cm	5 cm	10 cm	15 cm	20 cm	5 cm	10 cm	15 cm	20 cm
Untreated	2	54.8	27.5	16.8	12.6	503.5	456.7	356.7	425.2	38.2	36.8	37.4	37.8
	33	48.7	25.4	17.0	12.5	513.7	469.0	347.0	323.2	38.6	37.9	38.5	39.2
	66	43.2	19.5	15.1	11.2	400.7	330.0	428.0	283.0	38.4	37.3	38.8	40.8
	98	37.5	17.6	13.4	9.8	504.2	384.7	333.5	294.7	37.0	35.1	39.3	38.9
Treated	2	54.7	26.8	18.0	13.3	463.0	378.7	411.5	375.5	34.4	34.5	38.0	39.4
	33	50.8	23.1	16.5	12.6	454.2	504.5	366.5	383.7	33.6	37.9	35.9	38.6
	66	48.9	24.6	15.7	11.6	398.2	380.7	468.5	342.0	33.7	34.7	40.0	40.2
	98	42.9	21.8	13.9	9.2	444.0	383.0	297.5	284.0	36.8	40.1	40.2	37.0
L. S. D. (1%)													
treatments				14.0				N. S.				N. S.	
infection levels				14.0				N. S.				N. S.	
seed spacings				14.0				150.0				3.4	

Table 4. Seedling emergence and infection of barley grown at normal greenhouse humidity from Cochliobolus sativus-infected seed from three sources and treated with two fungicides

Seed source	Emergence (plants/flat)				Infected plants (%)			
	Control	Liqui-San	Benlate	Liqui-San & Benlate	Control	Liqui-San	Benlate	Liqui-San & Benlate
Saskatchewan	88.5	94.5	92.2	94.5	2.2	4.2	10.9	5.5
P. E. I.	72.7	90.2	57.5	91.5	9.6	6.9	26.5	10.9
Ottawa #1[1]	74.2	93.0	68.5	94.5	9.7	9.6	12.0	10.5
Ottawa #2[2]	82.0	93.0	83.2	92.5	10.0	5.9	17.3	8.6

[1] large seed

[2] small seed

Table 5. Seedling emergence and infection of barley grown in a high humidity greenhouse from seed with four levels of Cochliobolus sativus and treated with two fungicides

Infection level (%)	Emergence (plants/flat)			Infected plants (%)		
	Control	Liqui-San	Ceresan M	Control	Liqui-San	Ceresan M
2	92.5	97.7	96.7	7.8	2.0	1.2
33	87.5	96.5	95.2	33.0	3.7	5.4
66	81.5	94.5	96.2	56.9	8.8	8.1
98	67.0	94.5	97.5	92.9	10.4	12.2

The mercury fungicides effectively increased emergence and prevented seedling infection from seed-borne inoculum. The manufacturer of Benlate does not recommend this chemical for the control of C. sativus, and our findings support this conclusion. The suggestion that it be used with another seed dressing appears satisfactory, as the combination gave the highest average emergence, seed yield, and 1000-kernel weight (Table 1).

Acknowledgments

We wish to thank DuPont of Canada Ltd., Toronto, Ontario and Green Cross Products, Montreal, Quebec, for experimental samples of fungicides; and T. F. Cuddy, Plant Products Division, Ottawa, and J. D. E. Sterling, Research Station, Charlottetown, for providing seed samples. The technical assistance of Mrs. J. Jeun, J. H. Clark, and R. Courdin is appreciated.

Literature cited

1. Greaney, F. J., and H. A. H. Wallace. 1943. New and standard seed treatments in the control of certain seed-borne diseases of wheat, oats and barley. Phytopathology 33:1064-1070.

2. Machacek, J. E., H. A. H. Wallace, H. W. Mead, and W. C. Broadfoot. 1954. A study of some seed-borne diseases of cereals in Canada III. Effect of rate of seeding, percentage of infested kernels, and weeks on the yield of plots sown with treated and untreated seed. Can. J. Agr. Sci. 34:240-251.

3. Mills, J. T., and H. A. H. Wallace. 1968. Determination of selective action of fungicides on the microflora of barley seed. Can. J. Plant Sci. 48:587-594.

BRIEF ARTICLE

MYCODIPLOSIS (DIPTERA, CECIDOMYIIDAE) FEEDING ON CLOVER RUST (UROMYCES TRIFOLII) SPORES

B. Berkenkamp[1]

During forage crop disease surveys, rust caused by Uromyces trifolii (Hedw. f. ex. DC.) Lév. was occasionally found on alsike clover (Trifolium hybridum L.), red clover (T. pratense L.), and white clover (T. repens L.). Small dipterous larvae were regularly found feeding on uredospores on rusted plants, usually on the lower surface of the leaves and occasionally on infested petioles and stems. Rust and associated larvae were found between July 7 and September 6 each year 1964 through 1968 in various areas throughout central and northern Alberta.

Larvae were collected and reared on rust-infected greenhouse plants, or detached leaves in the laboratory. In the field the larvae migrated downward to pupate and probably pupate in the soil. Since larvae of unknown ages were collected, the time required for completion of the life cycle was not established. Eggs, pupae and adults were not seen in the field, and the pupae and adults described were reared from larvae. The largae were 1.80 × .39 mm, had fourteen segments, and were bright orange in color. Pupae measured 1.60 × 0.48 mm, and adults were 1.45 mm long with legs up to 2.28 mm long.

Larvae and adults were identified as Mycodiplosis impatientis Felt (Cecidomyiidae) by Raymond J. Gagné of the Systematic Entomology Laboratory, U.S. Department of Agriculture, National Museum, Washington, D.C. This species was originally reared and described from Aecidium impatientis on stems of touch-me-not (Impatiens) (1).

Literature cited

1. Felt, E.P. 1908. Studies in Cecidomyiidae II, p. 307-422. In 23rd Report of the state entomologist 1907. New York State Mus. Bull. 124.

[1] Plant Pathologist, Research Station, Canada Department of Agriculture, Lacombe, Alberta.

BOOK REVIEW

PLANT PATHOLOGIST'S POCKETBOOK

Commonwealth Mycological Institute (Compiler). Commonwealth Mycological Institute, Kew, Surrey, England. iv + 267 p. 1968. Price 30 shillings or Can. $3.90.

Dr. G.C. Ainsworth, Director of the C.M.I., remarked in the preface to this book that the contributions made to it by the staff of that institution, and by outside specialists, were planned to fill a need for a wide-ranging reference miscellany for plant pathologists. In such a wide conspectus of plant pathology, it is not surprising that full justice could not be done to many of the topics. The selection of these and the emphasis placed on each have clearly been the compilers' dilemma.

The contents, under 19 main headings, range from fungus diseases, plant quarantine, plant pathogenic nematodes, and weeds, to presentation of results. The index is adequate since the contents table is sufficiently detailed. There are excellent sections on methods and techniques, and formulae (with most of the standard and a few novel prescriptions and "wrinkles"), which will probably be used more than most of the other information. The section on some common plant diseases is least regarded by this reviewer since there is only a passing reference to diseases of grasses, but then it may be of considerable interest to those pathologists whose interests are less parochial. The tables in this section waste a considerable amount of valuable space and a running account of the more important diseases of major crops would have sufficed. This space would have been profitably employed in expanding lists and references to diseases by region and crop, select bibliographies to each section, and addresses of organizations. Plant pathologists with their major training in mycology in the majority should welcome the sections on bacteria, viruses, nematodes, and insects, since they suggest how to handle and describe these often unfamiliar pathogens. The introductions to these and other sections are very readable and succintly bring one up-to-date. This is a very useful handbook (it is hardly a pocket book) for the individual plant pathologist, especially when working away from a good library. Very few serious slips or printing errors were found.

J. Drew Smith
Research Station
Canada Dept. Agric.
Saskatoon, Sask.