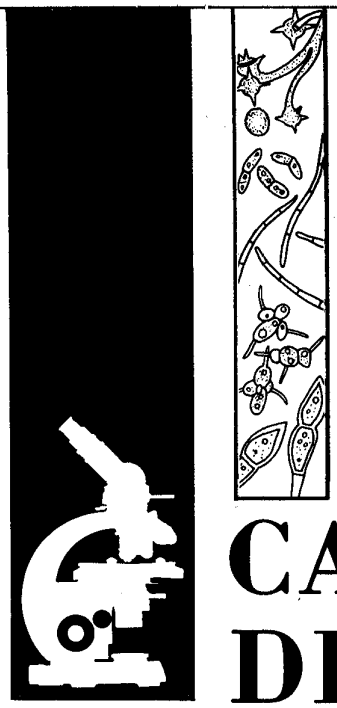
VOL.50, Nos.3–4, SEPT.– DEC., 1970



CANADIAN PLANT DISEASE SURVEY

EDITOR W.L. SEAMAN

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

VOL.50, Nos.3-4, SEPT.-DEC., 1970

CANADIAN PLANT DISEASE SURVEY

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

CONTENTS

See inside back cover for contents to Vol. 50, No. 4.

"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."

RESISTANCE OF TIMOTHY CULTIVARS TO HETEROSPORIUM PHLEI, DRECHSLERA PHLEI, AND FROST INJURY[1]

J.Drew Smith[2]

Abstract

Although none of 44 cultivars of timothy, Phleum pratense L. and P. nodosum L., showed complete resistance to Heterosporium phlei Gregory in a mild, artificially induced field epidemic of leaf spot disease, some appeared significantly less susceptible than others. The absence of complete mature plant resistance in eight strains was confirmed in growth chamber pathogenicity tests. There was no good correlation between frost injury and resistance to H. phlei. The abundance of overwintering inoculum on crop debris is perhaps more important as an epidemiological factor than frost damage. The destruction of this inoculum by hay removal or careful field burning and the use of less susceptible cultivars and fertilization are possible control measures requiring further testing. Natural infection with Drechslera phlei (Graham) Shoemaker was very light and was not correlated with resistance to H. phlei or with the other characters. There were good positive correlations between severity of frost injury and plant vigor, frost injury and earliness of heading, and between plant vigor and earliness of heading.

Introduction

Timothy, Phleum pratense L. and P. nodosum L., is grown primarily as a seed crop in Saskatchewan. Lack of resistance to drought and soil salinity mitigate against its common use for hay and pasture (13). A leaf spot of timothy caused by Heterosporium phlei Gregory (5) was observed causing severe damage to seed crops of the 'Climax' cultivar in the seed-growing district in northeastern Saskatchewan in 1967 and 1968 (unpublished data). Although timothy is generally regarded as winter hardy (13), early spring frosts may cause considerable leaf injury. Such injury and abundant carry-over of inoculum on the heavy straw from the previous crop were considered to be contributory factors in the 1967 epidemic in Saskatchewan. It was been reported that the disease reduced forage yields. Tsutomu and Takeshi (12), who controlled Heterosporium leaf spot by applications of a dithiocarbamate fungicide, found that the disease reduced crude protein in leaves by 26% and in whole shoots by 7%. Depressed yields of forage were ascribed to the disease in Nova Scotia (3).

Drechslera phlei (Graham) Shoemaker, syn. Helminthosporium dictyoides Drechsler var. phlei Graham (4), is the cause of a leaf spot (streak) of timothy in Europe and North America (4,10). The disease appears to be of little importance in Saskatchewan seed crops. However, heavy seed infection has been found in samples from eastern Canada, U.S.A., and Europe (11), so the disease is probably of greater importance where Saskatchewan timothy seed may be used.

This paper reports on the 1969 field reaction of 44 cultivars of P. pratense and P. nodosum from various countries to H. phlei, D. phlei, and frost. Cultivars were also rated for general vigor, abundance of seed heads, and earliness of heading to determine whether these characters were correlated with disease resistance.

Materials and methods

Plant material

After a health test (11), seed of cultivars (Table 1) with satisfactory germination was dressed with a captan dressing at 0.2% seed weight (8) to reduce the risk of introducing alien pathogens. A field test with six replicates was planted at Saskatoon on May 29, 1968. Seed was sown at 1 cm depth in rows 30 cm apart at the rate of 6.5 g/3 m row. Six infector rows of untreated 'Climax' were sown around the perimeter of each replicate.

Plot inoculation

All plants were mown with a sickle-bar mower on August 28, and the hay was left to serve as a substrate for H. phlei. Isolates of the fungus were cultured on potato dextrose agar for 3 weeks. A distilled water suspension containing 3.25 x 10 spores/ml was prepared and applied to the test area with a pneumatic sprayer on September 3 at the rate of 8 litres/400 m . The plots were sprayed in two directions to improve eveness of cover. Light rain followed the application of the inoculum, and the weather was

[1] Contribution No. 392, Research Station, Canada Department of Agriculture, Saskatoon, Saskatchewan.

[2] Plant Pathologist.

cool (max. 15C) and dull for the next 2 days. The pathogenicity of the inoculum used in the field was checked on timothy plants in an infection chamber.

Rating for disease, plant vigor, and heading

The leaf spots caused by H. phlei and D. phlei were rated on 10 plants per row; frost injury, general plant vigor, earliness of heading, and abundance of seed heads were appraised on a row basis. A rating scale of 0 to 4 was used, 0 indicating no disease and 4 very severe disease. A zero rating also denoted lowest frost injury, plant vigor, fewest seed heads, and latest heading, while 4 was the opposite extreme. All ratings were made between June 27 and 29, 1969 except for seed head abundance, which was scored on July 16.

Table 1. Ratings of 44 cultivars of timothy for resistance to Heterosporium phlei, Drechslera phlei, and frost injury and for plant vigor, earliness of heading, and abundance of seed heads

Cultivar or strain	Country of origin	Average rating* per plant or row					
		H. phlei	D. phlei	Frost injury	Plant vigor	Heading earliness	Heading abundance
S-50	U.K.	1.25	0.00	1.42	2.50	2.33	2.17
Timo	Holland	1.17	0.25	1.83	2.42	1.33	2.17
Vertas	Holland	1.08	0.33	2.83	1.58	0.25	1.00
9-S	Canada	1.00	0.25	1.92	2.67	2.50	1.50
Tiger	U.S.A.	1.00	0.50	2.33	1.75	1.00	0.67
Veng	Holland	0.92	0.67	2.58	1.92	1.00	1.00
Clair	U.S.A.	0.92	0.50	1.92	2.58	2.42	1.17
Lofar	Holland	0.83	0.33	2.08	2.42	1.58	2.50
CIV Tuss.	Holland	0.83	0.42	2.08	1.92	1.50	1.50
Champ	Canada	0.83	0.75	1.92	2.50	2.08	1.50
S-51	U.K.	0.83	0.58	2.75	2.00	0.92	1.17
Evergreen	Sweden	0.75	0.08	1.33	2.67	1.83	1.33
Bottnia 2	Sweden	0.75	0.25	1.92	2.25	2.08	1.67
Heidimij	Holland	0.75	0.33	2.42	2.00	0.42	1.67
32-4	France	0.75	0.33	2.75	1.50	0.58	1.67
Barenza early hay	Holland	0.75	0.50	2.16	2.33	2.42	2.33
S-48	U.K.	0.75	0.08	2.75	1.75	0.25	2.17
Taca Trif. S65	Sweden	0.67	0.25	1.58	2.58	2.75	2.17
Eskimo	Holland	0.67	0.42	2.00	2.16	2.25	2.00
Sceempter	Holland	0.67	0.08	2.67	1.58	0.08	2.33
CIV 34	Holland	0.67	0.50	1.75	2.58	2.83	0.83
Climax (U.S.A.)	Canada	0.67	0.58	1.42	2.58	2.42	1.00
Wisconsin T4	U.S.A.	0.67	0.33	1.50	2.58	2.33	1.83
Wisconsin T1	U.S.A.	0.67	0.33	1.33	2.67	2.25	0.67
Panther	U.S.A.	0.67	0.17	2.42	1.83	1.17	1.83
2-S	Canada	0.67	0.50	1.83	2.50	2.42	1.67
Topas Otofte	Denmark	0.58	0.25	1.50	2.58	2.00	3.00
Kahu	New Zealand	0.58	0.25	2.00	2.33	1.25	2.17
King	Holland	0.58	0.50	2.67	1.83	0.42	1.83
Samo	Holland	0.58	0.17	2.83	1.25	0.58	1.67
S-352	U.K.	0.58	0.50	1.50	2.75	2.75	1.33
Combi	Holland	0.58	0.42	2.33	2.08	0.58	1.67
Drummond	Canada	0.58	0.42	2.08	2.33	1.25	1.83
Bounty	Canada	0.58	0.58	1.83	2.42	2.00	1.00
Erecta RVP	Belgium	0.50	0.25	1.67	2.58	2.42	1.50
Barenza late hay	Holland	0.50	0.33	2.25	2.00	0.50	1.83
Jacoba	Holland	0.50	0.25	2.50	1.83	1.17	1.83
Climax (Can.)	Canada	0.50	0.42	1.33	2.75	2.50	1.50
T41	U.S.A.	0.50	0.17	1.83	2.58	2.17	1.00
Essex	U.S.A.	0.50	1.08	1.50	2.33	1.75	1.17
Common (U.S.)	U.S.A.	0.50	0.42	1.75	2.50	2.33	1.33
Labelle	Canada	0.50	0.42	1.58	2.58	1.83	0.83
L 84	Italy	0.50	0.50	2.00	2.58	2.42	1.17
Bero	Sweden	0.42	0.33	2.00	2.33	2.08	2.00
Grand Mean		0.70	0.35	2.01	2.26	1.66	1.59
L.S.D. 5%		0.35	0.37	0.64	0.59	0.69	1.03
1%		0.47	0.49	0.81	0.78	0.91	1.37
Mean of							
8 Canadian cultivars		0.67	0.49	1.74	2.54	2.13	1.35
16 N. American cultivars		0.67	0.40	1.78	2.44	2.03	1.28
28 Other cultivars		0.71	0.32	2.15	2.15	1.44	1.77

* All ratings were made on a 0 to 4 scale, where 0 was no disease, or no frost injury, lowest vigor, fewest seed heads, and 4 the extreme opposite. 0 was latest heading and 4 earliest.

Results and discussion

Timothy plants inoculated with the spore suspension and placed in an infection chamber showed leaf lesions in 6 days, which was also the time required for slight leaf spotting to develop in the field. Heterosporium leaf spots were easily found on many of the cultivars, but no D. phlei lesions were seen in 1968. Ratings of the test made in 1969 are given in Table 1.

Although none of the cultivars showed complete resistance to H. phlei, some were significantly less susceptible than others. There was little difference apparent in the reactions of cultivars of North American, Canadian, and other origins (Table 1). Tsutomu and Takeshi in Japan (12) found in one test that none of 12 cultivars showed great resistance to this pathogen, but 'Essex' was least susceptible. In another test, 'S-50' was the most severely spotted of seven cultivars. They found also that 'Climax' of Canadian and U.S. origin showed considerable differences in susceptibility. Similar results were obtained with these cultivars in the test reported here. Resistance to H. phlei was examined further by screening in growth chambers 500 plants of the Canadian 'Climax', 'Bounty' (1), and 'Champ' (2) cultivars and of five lines from the U.S.S.R. The plants were tested in flights, with uninfected survivors from earlier ones being reinoculated. This mitigated against disease escape. No cultivars remained free from disease although several clones of 'Bounty' were only slightly affected. Possibly these may form the basis of a resistant cultivar.

Leaf spot caused by D. phlei was quite light on the test plots in 1969. Cultivars of P. nodosum, 'S-50' and 'Evergreen', and 'S-48', 'Sceempter' and 'Essex' of P. pratense showed little or no infection. The inoculum for this infection may have come from the infector plants of 'Climax' since the seed of all test cultivars was treated and there were no other stands of timothy in the vicinity.

Generally, North American cultivars were slightly superior to those from other sources in resistance to frost injury (Table 1). Canadian cultivars scored well.

There were no significant correlations between the ratings for the two diseases or between these and the other agronomic characters. There were good positive correlations between severity of frost injury and plant vigor, r = 0.659, and between frost injury and earliness of heading, r = 0.661. This indicated that those cultivars which produced early growth in spring or were early in heading were also susceptible to early spring frost damage. Canadian and U.S. cultivars showed greater plant vigor and were considerably earlier in heading out than those from other sources, but the former generally produced less abundant seed heads (Table 1). This may be related to the fact that many of the North American cultivars were late hay types (1,2,6). There was also a good correlation, r = 0.661, between plant vigor and earliness of heading.

The absence of correlation between frost injury and leaf spot ratings for H. phlei suggests that these are not linked epidemiologically, as was previously considered in the epidemics in northeastern Saskatchewan. The abundance of overwintering inoculum on crop debris may be the important factor in determining disease severity. The fungus overwinters as mycelium in tissues (12) and spores are produced sparingly on lesions. According to Horsfall (7) and Jacques (9), these will germinate at temperatures as low as 3C, while Narita and Sakuma found that spores may be produced at low temperatures and high humidity under snow (12). The same workers reported that sporulation was more abundant on parts of the plant deficient in magnesium and potassium than on normal parts. The disease was partly controlled by appropriate applications of fertilizer containing nitrogen, phosphate, and potassium. The burning of crop debris may be a means of controlling the disease, but its physiological effect on the plants is uncertain. Severe thinning has resulted in seed crops in Saskatchewan following burning, probably due to damage to growing points.

Acknowledgment

I am indebted to Dr. T. Ootaki, a Postdoctorate Fellow in Biology at the University of Saskatchewan, Saskatoon, for translation of Japanese papers.

Literature cited

1. Canada Department of Agriculture, Production and Marketing Branch, Plant Products Division. 1966. Description of variety, DV 1030: Bounty timothy. 6 p.

2. Canada Department of Agriculture, Production and Marketing Branch, Plant Products Division. 1967. Description of variety, DV 1075: Champ timothy. 6 p.

3. Creelman, D.W. 1956. The unusual occurrence of three leaf-spotting fungi on grasses in Nova Scotia. Plant Dis. Rep. 40:510-512.

4. Graham, J.H. 1955. Helminthosporium leaf streak of timothy. Phytopathology 45:227-228.

5. Gregory, C.T. 1919. Heterosporium leafspot of timothy. Phytopathology 9:576-580.

6. Hanson, A.A. 1965. Grass varieties in the United States. U.S. Dep. Agr. Handb. 170. 102 p.

7. Horsfall, J.G. 1930. A study of meadow-crop diseases in New York. Cornell Univ. Agr. Exp. Sta. Memoir 130. 139 p.

8. Jackson, N. 1958. Seed dressing trial. J. Sports Turf Res. Inst. 9:455-458.

9. Jacques, J.E. 1941. Studies in the genus Heterosporium. Contrib. Inst. Bot. Univ. Montreal 39:36-38.

10. Noble, Mary, and M.J. Richardson. 1968. An annotated list of seed-borne diseases. Commonw. Mycol. Inst. England. 191 p.

11. Smith, J. Drew. 1970. Seed-borne Drechslera phlei on Phleum spp. Can. J. Plant Sci. 50 (In press).

12. Tsutomu, S., and N. Takeshi. 1963. Timothy leaf spot caused by the fungus Heterosporium phlei Gregory. J. Hokkaido Nat. Agr. Exp. Sta. 7:77-90. (In Japanese)

13. University of Saskatchewan, Extension Division, Saskatoon. 1969. Guide to Farm Practice in Saskatchewan, 1969. 142 p.

INCIDENCE OF ARMILLARIA ROOT ROT IN BALSAM FIR INFESTED BY BALSAM WOOLLY APHID[1]

Janos Hudak and Pritam Singh[2]

Abstract

Studies in Newfoundland have shown that the incidence and intensity of armillaria root rot in balsam fir (Abies balsamea) varies directly with the level of damage caused by the balsam woolly aphid (Adelges piceae). This damage appears to predispose trees to infection by Armillaria mellea.

Introduction

Armillaria root rot, caused by Armillaria mellea (Vahl ex Fr.) Kummer, is an important root disease of a wide variety of tree species in both the temperate and tropical regions of the world. The pathogenicity and degree of parasitism of A. mellea and its role in the decadence of forest trees is not well understood, but the fungus is particularly destructive in plantations and to natural stands already weakened by other factors (3). Day (1) reported that oak trees weakened by drought or by defoliating or stem-boring insects were readily infected by A. mellea. Naumenko (2) and Staley (4) showed that the incidence of the disease in oak increased with the extent of defoliation caused by the gypsy moth, Porthetria dispar (L.), and the leaf roller, Argyrotoxa semipurpurana Kearf.

Armillaria root rot has been recorded in natural stands of native species and in plantations of exotic species in Newfoundland, but little is known about its status in stands of balsam fir, Abies balsamea (L.) Mill., infested by the balsam woolly aphid, Adelges piceae (Ratz.). The aphid is one of the most destructive forest insects in Newfoundland, where it has infested more than 6000 square miles of balsam fir forest during the last 40 years. The most commonly observed symptoms of aphid attack are inhibited shoot growth, distortion of twigs, or "gout", gradual thinning of the foliage, and dieback of the crown.

A survey of aphid-infested stands in southwestern Newfoundland showed that the disease was more prevalent in trees damaged by the aphid (5). This paper describes the relationship between the incidence of armillaria root rot and damage caused by balsam woolly aphid.

Materials and methods

Investigations were conducted in five merchantable, 60- to 70-year-old, aphid-damaged balsam fir stands located at Codroy Pond, St. Fintans, Flat Bay Brook, Southwest Brook, and Crabbes River, and in an undamaged stand near Corner Brook.

Damage was stratified into five classes and class limits were arbitrarily assigned within the range of 1 to 7, as follows: undamaged 1, light 1.5-3.0, medium 3.5-4.5, severe 5-6, and dead 7. Trees were allocated to appropriate damage classes by averaging the numerical indices of visual estimates obtained from the upper and lower halves of the crowns. In damaged stands, 10 trees over 3.5 inches dbh were sampled for each damage class along 20 randomly selected transects. In the undamaged stand, 40 trees were examined. Three primary roots were marked on each tree. The most northerly root was designated as I, and roots II and III were identified clockwise from the first. These roots were exposed and the proximal 3-foot sections were examined for characteristic symptoms and signs of the root rot: discoloration of the bark, resin exudate, and mycelial fans under the bark. The incidence of the disease was expressed as percentage of infected trees. The intensity of the disease was indicated by the percentage of roots infected and by the lineal extent of mycelial fans on the 3-foot sections.

Results

Armillaria root rot was present in all six stands (Table 1). Infection in undamaged trees averaged 2.5% and in damaged living trees 18.6%. However 36% of living and dead damaged trees were infected. The percentage of infected trees damaged by the aphid was similar irrespective of the location of stands.

The incidence of armillaria root rot increased with the level of aphid damage, averaging 4% in the light, 20% in the medium, 32% in the severe, and 88% in the dead classes (Table 1). The intensity of the disease showed a similar trend as indicated by the lineal extent of mycelial fans (Table 2). The increase in the incidence and intensity of the disease was gradual from the

---

[1] Contribution from Forest Research Laboratory, Canada Department of Fisheries & Forestry, P.O. Box 6028, St. John's, Newfoundland.

[2] Forest Pathologist and Mycologist, respectively.

light to the severe damage classes and rose sharply from the severe to the dead classes.

Table 1. Percent aphid-damaged and undamaged balsam fir trees* infected by Armillaria mellea in the six stands

Location of stand	Undamaged	Damage Class				Average
		Light	Medium	Severe	Dead	
Codroy Pond		0	20	30	80	32.5
St. Fintans		0	30	30	100	40.0
Crabbes River		10	30	40	70	37.5
Flat Bay Brook		0	0	30	90	30.0
Southwest Brook		10	20	30	100	40.0
Corner Brook (Control)	1					2.5
Average %	2.5	4	20	32	88	30.4
Average % (Light to Dead damage classes)				36		
Average % (Light to Severe damage classes)				18.6		

* The percentage is based on 10 trees in each damage class in a stand except in the control, where the number of trees was 40.

Table 2. Incidence and intensity of Armillaria mellea in balsam fir trees showing aphid damage

Aphid damage class	No. trees examined	Trees infected		No. roots examined	Roots infected		Avg lineal extent of mycelial fans	
		(No.)	(%)		(No.)	(%)	(inches)	(%)*
Undamaged	40	1	2.5	120	1	0.8	4.0	11.1
Light	50	2	4.0	150	3	2.0	10.3	26.8
Medium	50	10	20.0	150	11	7.3	13.0	36.3
Severe	50	16	32.0	150	22	14.6	22.9	63.7
Dead	50	44	88.0	150	104	69.3	27.8	77.3

* Based on 3-ft.-long portions of three roots/tree.

Conclusions

This study shows that armillaria root rot can affect undamaged as well as aphid-damaged trees, but the incidence of the disease was higher in the latter group. This indicates that aphid damage, including loss of foliage, is a primary factor predisposing trees to infection, but such damage is probably more important in influencing the rate of progress of the disease. Similar observations were made by Day (1), Naumenko (2), and Staley (4) on oak trees damaged by defoliating and stem-boring insects.

Armillaria mellea is common in most forest soils in Newfoundland and may be important in increasing stand decadence and mortality of trees damaged by the balsam woolly aphid. It is becoming more important as aphid infestations spread. The higher incidence of the disease in merchantable stands damaged by the insect shows that high priority must be given to salvaging severely damaged stands. There is also a need for more intensive study on the epidemiology of armillaria root rot in aphid-infested stands in all age classes and on all sites to determine appropriate management practices.

Literature cited

1. Day, W.R. 1927. The oak mildew Microsphaera quercina (Schw.) Burrill and Armillaria mellea (Vahl) Quél. in relation to the "Dying-back" of oak. Forestry 1:108-112.

2. Naumenko, I.M. 1949. The desiccation of the oak in Voronezh State Forest and in the Timber establishments of Voronezh Oblast. Publications of Voronezh State Forest No. III, 1949. (English translation from Usykhanie duba v Voronezhskom gosudarstvennom zapovednike i leskhozakh Voronezhskoi oblasti. Trudy Voronezhskogo gosudarstvennogo zapovednika, vypusk III, 1949).

3. Patton, R.F., and R. Vasquez Bravo, 1967. Armillaria root rot, Armillaria mellea (Vahl ex Fr.) Kummer. p. 36-38. In A.G. Davidson and R.M. Prentice [ed.] Important forest insects and diseases of mutual concern to Canada, The United States and Mexico.. Can. Dep. Forest. Rural Develop., Ottawa.

4. Staley, J.M., 1965. Decline and mortality of red and scarlet oaks. Forest Sci. 11(1):2-17.

5. Warren, G.L., 1966. Important forest diseases. In Annual report of the forest insect and disease survey, Newfoundland region. Can. Dep. Forest. Rural Develop., Forest. Branch, Ottawa. p. 9-17.

PLANT-PARASITIC NEMATODES FROM CANADA AND ABROAD, 1969

Robert Sewell[1]

During 1969, soil samples, plants, and other materials were submitted to the Nematology Section, Entomology Research Institute, for the extraction and identification of nematodes. Most of the samples came through the Plant Protection Division, Canada Department of Agriculture, being intercepted at airports and ports. Other agricultural agencies, scientists, local farmers, greenhouse operators and florists from across Canada also sent in material for identification and advice.

CYST-FORMING NEMATODES (Genus Heterodera)

Heterodera trifolii Goffart, 1932 (Oostenbrink, 1949) was intercepted in soils containing Spirea sp. from Holland, Oxalis sp. from Ireland, rosemary house plants from Italy and Portugal, and nursery stock from New Jersey; it was also found in a survey of potato fields carried out in the Montreal, Toronto, and Vineland areas. Heterodera avenae Wollenweber, 1924 (Filipjev, 1934) was encountered in soil during the cyst survey of the Toronto and Vineland areas, and from soils supporting Spirea sp. and tulip from Holland, and Rosa sp. from England. Heterodera schachtii Schmidt, 1871, the sugar beet nematode, was associated with house plants, potatoes, peppers, and Lilium sp. from Italy, and tomatoes from Portugal. Heterodera bifenestra Cooper, 1955 was intercepted in soil on ornamentals from Italy. Heterodera weissi Steiner, 1949 was discovered in soils associated with Oxalis sp. from Holland and ornamentals from Yugoslavia and Tennessee. Heterodera cacti Filipjev and Schuurmans-Stekhoven, 1941 was intercepted in soils associated with cauliflower from the United States, and Crassula sp. from Europe, and from the cyst survey carried out in the Toronto area. Heterodera cruciferae Franklin, 1945 was reported from soil about the roots of Ranunculus sp. and begonia from Portugal, and Crassula sp. from Europe. Heterodera humuli Filipjev, 1934, the hop cyst nematode, was found in close association with grape cuttings from Italy and oleander from Portugal. Heterodera fici Kirjanova, 1959 was detected in shipments of fern and house plant from Italy, tomato plants from Portugal, and asparagus from Greece. Heterodera punctata Thorne, 1928 was found in soil with Spirea sp. from Holland, and from soil samples from surveys made in Newfoundland and British Columbia.

ROOT-KNOT (Genus Meloidogyne)

Meloidogyne hapla Chitwood, 1949, the northern root-knot nematode was found on Rosa sp. from Pasadena, California; clematis, and Rosa sp. from Holland; Rosa multiflora from France, Oregon, and Belgium; horseradish from Ancaster, Ontario; and in soil from lilac from Iowa. Meloidogyne incognita (Kofoid and White, 1919) Chitwood, 1949 was found on tomato plants from Georgia and in greenhouse soil from the CDA Research Station, Kentville, Nova Scotai; also in a soil sample from Macdonald College, Quebec, and from soil on honeysuckle rose from Tennessee. Meloidogyne javanica (Treub, 1885) Chitwood, 1949, the Javanese root-knot nematode, was removed from the roots of tomato plants from Georgia, U.S.A.

ROOT-LESION NEMATODE (Genus Pratylenchus)

Pratylenchus crenatus Loof, 1960 was found in soils associated with an unidentified herbaceous plant from England, and with red and pin oak from Pennsylvania. Pratylenchus penetrans (Cobb, 1917) Filipjev and Schuurmans-Stekhoven, 1941 was found on a herbaceous plant from England, on oak from Illinois, on red clover from Prince Edward Island, on lily from Holland, and on juniper from Pennsylvania. Pratylenchus convallariae Seinhorst, 1959 and Pratylenchus fallax Seinhorst, 1968 were discovered on lily-of-the-valley pips from Holland.

SPIRAL NEMATODES (Genera Helicotylenchus and Rotylenchus

Helicotylenchus pseudorobustus (Steiner, 1914) Golden, 1956 was found on house plants from Portugal and in soil associated with orange trees from Florida. Helicotylenchus digonicus Perry, 1959 was detected in soil taken from the roots of Acer sp., rosemary, and begonia from the United States, Italy, and Russia, respectively. Helicotylenchus platyurus Perry, 1959 was found on blueberry from Aylen Lake, Ontario, and in soil from the roots of juniper from Pennsylvania. Helicotylenchus californicus Sher, 1966 was reported on begonia from Russia. Helicotylenchus dihystera (Cobb, 1893) Sher, 1961 was found on begonia from Italy, and on Mimosa sp. from New York. Nematodes identified as Helicotylenchus sp. were reported from a farm field in Ancaster, Ontario, in which horseradish was cultivated. Hoplolaimus galeatus (Cobb, 1913) Sher, 1963 was intercepted in soil on red oak, pin oak, and juniper from New York. Scutellonema brachyurum (Steiner, 1938) Andrassy, 1958 was intercepted in soil from around fruit trees from Israel.

[1] Nematology Section, Entomology Research Institute, Canada Department of Agriculture, Ottawa.

STUNT NEMATODES (Genus Tylenchorhynchus)

Tylenchorhynchus dubius (Butschli, 1873) Filipjev, 1936 was discovered on strawberry from the Central Experimental Farm, Ottawa, and on begonia from Russia. Tylenchorhynchus maximus Allen, 1955 was discovered in grasses from a golf course at Glenlea, Manitoba. Tylenchorhynchus martini Fielding, 1956 was reported on soil around orange trees from Florida.

PIN NEMATODES (Genus Paratylenchus)

Paratylenchus projectus Jenkins, 1956 was removed from soil around heather and ornamentals from Belgium and Poland.

RING NEMATODES (Genus Criconemoides)

Criconemoides ornatum Raski, 1958 was intercepted on oak from Pennsylvania. Criconemoides curvatum Raski, 1952 was intercepted from fruit trees, rosemary, and orange trees from Israel, Italy, and Florida, respectively. Criconemoides xenoplax Raski, 1952 was found on oak from Illinois.

Several Criconemoides spp. (possibly new species) were identified from Mimosa sp. from New York, begonia from Italy, and strawberry from Ottawa, Ontario.

MISCELLANEOUS (Tylenchids)

Psilenchus hilarulus de Man, 1921 was found on Mimosa sp. from New York. Ditylenchus destructor Thorne, 1945, the potato-rot nematode, was identified on iris bulbs from New Jersey.

DORYLAIMIDS

Xiphinema americanum Cobb, 1913 was recorded on Acer platanoides and on juniper from Illinois and Pennsylvania. Xiphinema bakeri Williams, 1961 was found in a soil sample submitted from British Columbia.

PLANT-PARASITIC NEMATODE GENERA ASSOCIATED WITH CROPS IN ONTARIO IN 1969

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

The Ontario Nematode Diagnostic and Advisory Service processed 786 soil samples in 1969. This is an increase of 78% or 344 more samples than were handled in 1968. The samples originated from 28 crops (Table 1) representing a cross section of plant production in greenhouses and fields throughout the province. As in previous years the majority of the samples were from the tobacco areas, with only 17% of the samples pertaining to other crops.

The marked increase in the number of samples processed by the diagnostic service during the past year was due to the submission of 530 soil samples by agricultural chemical companies, who conducted demonstration plots and field tests on proven and candidate nematicides. Although the total number of samples diagnosed in 1969 was high, the number of soil samples submitted by growers decreased to 250, a 43% decrease from the previous year. Soil samples from tobacco growers decreased from 269 in 1968 to 122 in 1969, a decrease of 55%. Similarly there was a decrease of 37% in the number of samples submitted by fruit growers.

There is no doubt that the decline in the number of soil samples submitted by growers is directly related to the recent upsurge in the practice of pre-plant soil fumigation. This is particularly true in the tobacco area, where in 1969 the acreage being treated with nematicides increased by 100% to include approximately 50% of the total acreage planted to tobacco. Pre-plant soil fumigation is also rapidly becoming a standard practice for fruit growers.

The general extraction techniques (1,2) used in previous years have been found to be satisfactory for diagnosing large numbers of soil samples. However, it has been found that accuracy can be increased by removing as much of the root tissue as possible from the soil sample, and by placing emphasis on proper mixing of the soil before removing the subsample for nematode extraction. Standardization of processing techniques and of environmental conditions under which nematode extraction occurs has increased both the capacity and quality of the diagnostic service.

Pratylenchus spp. (root-lesion nematodes) were present in 17 of the 28 crops sampled. This genus is associated with most of the nematode problems in Ontario crops. The level of infestation of the root lesion nematode P. penetrans (Cobb, 1917) Filip. & Stek., 1941 in the tobacco area has remained relatively stable over the past 5 years: 1621,1055,1256,1069, and 1522 nematodes per lb of soil for the years 1965 through 1969, respectively (3,4,5,6). It is apparent that annual variation in winter kill and general weather conditions does not significantly alter the background level of the nematode population.

Paratylenchus spp. (pin nematodes) were found in 17 crops, 16 of which also contained root lesion nematodes. The average number of pin nematodes per pound of soil was 554, and of root lesion nematodes, 661. This close association suggests that the pin nematode may be an additional factor in crop losses due to root-lesion nematodes.

Meloidogyne hapla (northern root knot nematode) was associated with lettuce, tomatoes, wheat, rye, strawberries, sweet cherries, tobacco, roses, and vinca.

Heterodera avenae (oat-cyst nematode) was found in oats and tobacco. Although the oat cyst nematode was not found in soil samples from corn fields in 1969, information from previous years (3,4) suggests that this nematode is becoming a problem in corn production in the areas where this pest is prevalent. Only by following a specified rotation can the oat cyst nematode be controlled in this situation. Such a rotation should allow at least 5 years between oat crops, with corn not directly following oats.

Literature cited

1. Townshend, J.L. 1963. A modification and evaluation of the apparatus for the Oostenbrink direct cottonwool filter extraction method. Nematologica 9:106-110.

2. Fenwick, D.W. 1940. Methods for the recovery and counting of cysts of Heterodera schachtii from soil. J. Helminth. 18:155-172.

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.

[1] Horticultural Research Institute of Ontario, Vineland Station, Ontario.

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

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. 46:144-145.

5. 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.

6. Olthof, Th.H.A., J.L. Townshend, J.W. Potter, and A Cornelisse. 1969. Plant-parasitic nematode genera associated with crops in Ontario in 1968. Can. Plant Dis. Surv. 49:54-55.

Table 1. Plant parasitic nematode genera associated with Ontario crops in 1969

Crop	Praty-lenchus	Paraty-lenchus	Meloido-gyne (larvae)	Xiphi-nema	Tylencho-rhynchus	Helicoty-lenchus	Hoplo-laimus	Hetero-dera (larvae)	Cricone-moides	Dity-lenchus
Asparagus (1)†										
Barley (3)	200/1**	1300/1				1750/1	50/1			
Cherries (sour) (4)	367/3	400/3								
Cherries (sweet) (4)	700/4	1350/3	10/1	50/1					50/1	
Corn (5)	380/5	283/3		500/1						
Cucumbers (2)										
Garden (1)	700/1	350/1								
Geraniums (1)										
Grapes (3)		10/1								
Greenhouse flowers (1)										
Impatiens (1)										
Lettuce (1)	30/1	10/1	30/1							
Mushrooms (4)	10/2					20/1				
Oats (3)	183/3	1100/1			50/1			483/3		
Onions (2)										10/1
Peaches (15)	1417/13	580/12		50/4	100/3				100/1	
Potting soil (1)										
Roses (15)	725/2	100/1	100/1			40/2				
Rubber plants (1)										
Rye (23)	706/16	1298/11	400/1	50/1	178/9		100/1			
Shrubs (5)	1175/4	50/2				925/2				
Squash (2)	1075/2	50/1			200/1					
Strawberries (10)	1450/9	150/2	600/1					50/1		
Tobacco (122)	1522/94	180/32	250/3		194/31	100/1	175/2			
Tobacco R* (530)										
Tomatoes (6)	103/3	1200/1	1150/3	70/2		925/2				
Vinca (1)			1000/1							
Wheat (19)	493/14	1000/10	263/4	155/2	163/12					
Total samples (786)										

† Number of soil samples processed.
* Samples from nematicide trials - averages are not included because chemical treatments render them invalid.
** Average number of nematodes per 1b of soil/number of samples containing the nematode.

DISEASES OF RAPE AND OTHER CRUCIFERS IN SASKATCHEWAN IN 1969[1]

G. Allan Petrie[2] and T. C. Vanterpool[3]

Staghead or white rust caused by Albugo cruciferarum S.F. Gray was the most damaging disease of turnip rape (Brassica campestris L.) encountered in 1969 in Saskatchewan, particularly in the northwestern part of the province. During the August-September survey, the proportion of plants in each field having terminal stagheads (systemic infections) was recorded. Approximately 40 uniformly-distributed fields were examined. On an area basis, the results were as follows:

Area	% of plants per field having terminal stagheads
Central Saskatchewan (Zealandia, Delisle)	1 to 5
Rosthern	5 to 10
Meota-Turtleford	10 to 20
Meadow Lake	up to 50

In rape plots of the Biology Department, University of Saskatchewan, the seed yield of individual plants with systemic Albugo infections was reduced on an average by 60%. Using this figure as a basis, yield reductions in 1969 due to this disease would have ranged from less than 1% to approximately 30% (in one field), with the average for western Saskatchewan as a whole being less than 10%.

Albugo cruciferarum was collected at Saskatoon on Capsella bursa-pastoris (L.) Medic. on May 16. Heavy infections had developed on this host in the field by June 10. Only the conidial state was found, no stagheads being observed. In early June, traces of white rust occurred on Descurainia sophia (L.) Webb, but at no time during the spring or summer were Albugo infections observed on Sisymbrium loeselii L., numerous specimens of which were examined. On June 21, the first observations of white rust on rape were made in the plots of the Biology Department. At about this time, abundant leaf infections were found on specimens of Lepidium sp. at Saskatoon.

As in the case of staghead, a south-to-north gradation in severity of ringspot (Mycosphaerella brassicicola (Duby) Lind.) was noted. The disease started to become prevalent north of Meota and was most widespread in the Meadow Lake area. It severely damaged individual plants in a few fields near Turtleford. Once again it was found on Capsella bursa-pastoris, collections being made near St. Walburg, Goodsoil, and Dorintosh.

Observations indicated that alternaria black spot caused by Alternaria brassicae (Berk.) Sacc. and A. raphani Groves & Skolko is definitely increasing in severity. For example, 100% of the plants in a rape field near Delisle were infected, and 80% infection was recorded in a field near Duck Lake. Severe blighting of individual plants occurred in many instances. Several weed hosts of A. brassicae were identified. Plants of Descurainia sophia adjacent to the heavily infected Delisle field were severely spotted. The fungus was also isolated from extensive black stem lesions on Sisymbrium altissimum L. and S. loeselii collected at Saskatoon, and from Thlaspi arvense L. obtained from northwestern Saskatchewan. In addition, A. brassicae was isolated from numerous 1968 rapeseed samples from the three prairie provinces, with Alternaria raphani also being obtained from many of these. Several instances of the occurrence of the latter on Thlaspi were recorded.

The brassica strain of Leptosphaeria maculans (Desm.) Ces. & de Not. (Petrie and Vanterpool 1965) was encountered in several areas where it has not hitherto been found. These included the Aylsham and Meadow Lake regions. It was isolated from stem material from five of six rape fields located in an area immediately west of Meadow Lake. In these fields from 10 to 20% of the plants were lesioned. The pathogen was also isolated from 1968 rapeseed samples from the Viking and Penhold regions of Alberta, from the Darlingford and Homewood areas of Manitoba, and from the Melfort area in Saskatchewan. One factor which perhaps contributed to the prevalence of L. maculans and other rape diseases near Meadow Lake is the rape-barley-rape rotation practised by many farmers in the area.

Fusarium foot rot of rape caused by a complex of Fusarium species was worthy of note in several parts of Saskatchewan. It was judged to be a potentially troublesome disease. Near Vonda a field having 10% severely infected plants was examined. Due to the earliness of infection, the reduction

---

[1] Contribution No. 378, Research Station, Canada Department of Agriculture, Saskatoon, Saskatchewan.

[2] Plant Pathologist, Saskatoon.

[3] Professor Emeritus, Biology Department, University of Saskatchewan, Saskatoon.

in yield of the field was approximately 10%. Herbicide damage may have been a contributing factor. In CDA rape plots at Saskatoon, foot rot was prevalent, and near Donavon infection in the order of 1% was recorded in several fields. Fusarium was also collected on specimens of Thlaspi arvense near St. Walburg.

Sclerotinia stem rot declined in severity in 1969 compared to previous years. Sclerotinia sclerotiorum (Lib.) de Bary was isolated from two 1968 rapeseed samples obtained from Manitoba. The fungus was carried in or on the seed itself.

During the summer months, a species of Selenophoma was isolated from the aerial parts of the following plants: Arabis sp., Brassica campestris L. (var. 'Echo'), Descurainia sophia, Linum (?) lewisii Pursh, Melilotus officinalis (L.) Lam., Sisymbrium altissimum, Sisymbrium loeselii, Sonchus arvensis L., (Fckl.) Petr. It is distinct from S. linicola Vanterpool, which was isolated from the same collection of wild flax.

Colletotrichum dematium (Pers. ex Fr.) Grove was found on Capsella bursa-pastoris and Thlaspi arvense near St. Walburg. We have reported the occurrence of this fungus on Lepidium sp. and Descurainia sp. (Petrie and Vanterpool, 1965).

Re-examination of a collection made in the Biology plots at Saskatoon in September, 1968, revealed the occurrence of fasciation on a few rape plants, the short fasciated branches being completely covered with Erysiphe polygoni DC.

Literature cited

1. Petrie, G.A., and T.C. Vanterpool. 1965. Diseases of rape and cruciferous weeds in Saskatchewan in 1965. Can. Plant Dis. Surv. 45:111-112.

BRIEF ARTICLES

ISOLATION OF GIBBERA COMPACTA FROM CRANBERRY AND THE EFFECT OF MOISTURE AND TEMPERATURE ON ASCOSPORE[1] DEVELOPMENT

C.L. Lockhart

Initial attempts in 1966 to isolate Gibbera compacta (Pk.) Shear from characteristic black spots, described by Bain (1), on cranberry leaves using 2% Cl as a sterilizing agent were unsuccessful. When 0.5% Cl (one part Javex to 9 parts water) was used in 1968, G. compacta was isolated from 12.5% of the leaf spots. No fungi were isolated from 54.1% of the diseased sections and 33.4% of them produced other fungi, some of which were known pathogens of the cranberry.

In 1966, G. compacta was rarely isolated from speckle spots on cranberry fruit when 70% (v/v) ethyl alcohol was used as a sterilizing agent (3) but in 1968, using 0.5% Cl it was isolated from 8.2% of the spots. Carlson and Boone (2) had similar results in isolating G. compacta from fruit using strong and weak sterilizing methods. Gourley and Harrison (3), who used 70% ethyl alcohol as a sterilizing agent in their fruit rot investigations, did not report the presence of G. compacta.

Table 1. Effect of temperature and moisture on development of ascospores of Gibbera compacta in infected cranberry leaves held in moist chambers on the surface of wet filter paper or submerged in water

Treatment	Days in moist chamber	% asci with ascospores at 10 C	15 C	22 C
Surface	0	0	0	0
Submerged	0	0	0	0
Surface	6	0	5	25
Submerged	6	0	10	75
Surface	8	0	10	40
Submerged	8	1	15	75
Surface	14	1	15	60
Submerged	14	2	20	80

Samples of leaves, in which asci but not ascospores were apparent in the perithecia of G. compacta, were placed in large petri plates on moistened filter paper or on filter paper flooded with water (2 cm deep). In the latter, filter paper was placed on top of the leaves to keep them submerged. Leaves of both treatments were held at 10C, 15C and 22C for 2 weeks with periodic examinations of ascospore development. Ascospores formed and matured faster in the flooded treatment and at the higher temperatures (Table 1). In the normal practice of flooding cranberry bogs for frost and insect control, the development of ascospores of G. compacta would be stimulated if the water was warm when the bogs were flooded.

Literature cited

1. Bain, H.F. 1926. Cranberry disease investigations on the Pacific Coast. . U.S. Dep. Agr. Bull. 1434.

2. Carlson, L.W., and D. W. Boone. 1966. A berry speckle disease of cranberry and its control. Plant Dis. Rep. 50:539-543.

3. Gourley, C.O., and K.A. Harrison. 1969. Observations on cranberry fruit rots in Nova Scotia 1945-55. Can. Plant Dis. Surv. 49:22-26.

[1] Contribution No. 1379, Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

VARIATION IN ISOLATES OF FUSARIUM SOLANI F. PISI COLLECTED FROM PROCESSING PEAS IN ONTARIO[1]

A.T. Bolton, A.G. Donaldson, and V.W. Nuttall

Abstract

A survey and collection of plants in fields of processing peas (Pisum sativum) in Ontario yielded both Fusarium solani f. pisi and F. oxysporum f. pisi. Preliminary tests demonstrated differences in pathogenicity among isolates of F. solani f. pisi. Some correlation was found between highly pathogenic isolates and severity of disease in the pea fields.

Introduction

For several years, isolates of Fusarium solani (Mart.) App. & Wr. f. pisi (F.R. Jones) Snyd. & Hansen from the pea disease nursery at the Ottawa Research Station have shown considerable variation in pathogenicity

[1] Contribution No. 256, Research Station, Canada Department of Agriculture, Ottawa, Ontario.

when used to test peas for root rot resistance. In 1967, a survey was made in several areas in Ontario where processing peas were being grown. A collection of diseased pea plants from these areas yielded cultures of both F. solani f. pisi and F. oxysporum f. pisi (Linford) Snyd. & Hansen. Although all isolates recorded as F. solani f. pisi completely fitted the description of this fungus given in the literature, differences in pathogenicity varied from inability to produce symptoms to complete destruction of 'Progress No. 9' seedlings in 14 days.

Materials and methods

All isolates of F. oxysporum f. pisi and F. solani f. pisi were monospored before pathogenicity tests were made. Seedlings of the root rot susceptible 'Progress No. 9' pea (Pisum sativum L.) variety and of a root rot tolerant selection, 'M-129', were inoculated with each isolate. Ten-day-old plants grown in sterile sand were dug up, the roots trimmed to 2 inches and immersed in a spore-mycelium suspension for 2 hours prior to replanting in sterile soil. All disease ratings were made 14 days after inoculation.

Table 1. Incidence of Fusarium solani f. pisi in Ontario pea fields and pathogenicity rating of isolates in greenhouse tests

Field number	Location (county)	Pea variety	% plants affected	Rating†
1	Prince Edward		tr*	3
3	Prince Edward		tr	2
4	Prince Edward		tr	3
5	Prince Edward	Perfection	tr	2
6	Prince Edward		30	4
8	Prince Edward		15	1
9	Prince Edward		3	4
10	Prince Edward		15	5
11	Prince Edward		tr	3
13	Prince Edward		tr	0
14	Northumberland	Perfection	tr	0
15	Northumberland	Freezer 69	8	2
16	Northumberland		tr	4
17A	Northumberland	Perfection	tr	5
17B	Northumberland	Perfection	tr	3
19	Northumberland	Pride	tr	0
20	Northumberland	Spright	tr	3
22	Middlesex		tr	3
23	Middlesex		tr	0
24A	Middlesex	Venus	10	5
24B	Middlesex	Venus	tr	3
25	Middlesex	Spright	20	4
26	Middlesex	Alpine	tr	5
28	Huron	Perfection	tr	4
29	Perth	Delmar 16	tr	3
31	Huron	Perfection	tr	3
32	Huron	Perfection	20	5
35	Carleton**	Wisconsin 183	5	5
37	Carleton	Perfection WR	tr	3
38	Carleton	New Season	tr	2
39	Carleton	New Wales	tr	2

† 0 = no symptoms to 5 = death of the plants.
* tr = trace.
** Carleton Co. isolates were from plots at the CDA Research Station, Ottawa.

Results and discussion

Of 31 specimens collected, 16 yielded F. solani f. pisi alone, 2 yielded F. oxysporum f. pisi alone, and 13 yielded both species. Several different cultural types of F. solani f. pisi were observed, as well as several that showed differences in pathogenicity when inoculated onto pea seedlings under controlled conditions in the greenhouse. The results of the survey and of preliminary pathogenicity tests are given in Table 1.

It appears from this survey that F. solani f. pisi is present in most fields in Ontario where processing peas are grown. In most cases, where a highly pathogenic isolate was obtained, the losses due to the disease were greater than in areas where a moderate or weak pathogen was isolated. F. solani f. pisi and F. oxysporum f. pisi occurring together in the plants did not always produce more extensive damage than either species alone. Since type of soil, moisture, and general environmental conditions were quite variable, it is impossible to determine the actual effect of both species within a single plant. Generally, where soil was poorly drained and where rainfall had been heavy during the early summer, damage caused by root rot was severe. Further investigations to determine pathogenic variation in isolates of F. solani f. pisi are in progress.

PHYTOPHTHORA ROOT ROT OF ALFALFA IN ONTARIO IN 1969[1]

C.C. Chi[2]

In Canada phytophthora root rot of alfalfa (Medicago sativa L.) caused by Phytophthora megasperma Drechs. was first observed in Ontario in the Ottawa Valley in 1964 (1). The disease is one of the highly destructive maladies of alfalfa (1,3,4). In August 1969 a limited survey was made in Ontario and southern Quebec to determine the prevalence of the disease.

Samples were taken at random in alfalfa fields where the stand had been thinned, or in areas with plants showing yellow discoloration. The roots of suspected plants were examined, and root tissues with disease symptoms were plated on an agar medium selective for Phytophthora and Pythium (2), using the procedures described previously. (1).

Phytophthora root rot was found in alfalfa fields in the 19 Ontario and two adjacent Quebec counties surveyed (Table 1). The disease occurred in low areas, on slopes where drainage was poor, and in areas where

[1] Contribution No. 257 Research Station, Canada Department of Agriculture, Ottawa, Ontario.

[2] Plant pathologist.

water collected. Damage varied from slight in some fields to very severe in others. Usually only small areas in a field were affected, but in several cases the stand in an entire field was severely thinned by the disease.

Table 1. Phytophthora root rot of alfalfa observed in Ontario and southern Quebec in 1969

Province and county	Incidence of P. megasperma
Ontario	
Carleton	22/26*
Durham	5/5
Frontenac	5/5
Grenville	3/3
Halton	8/8
Hastings	1/3
Lanark	5/7
Leeds	5/5
Northumberland	3/3
Ontario	3/5
Oxford	1/3
Peel	7/7
Perth	2/2
Peterborough	2/5
Renfrew	11/11
Victoria	3/3
Waterloo	4/5
Wellington	8/9
York	5/6
Quebec	
Argenteuil	7/9
Papineau	8/12

* No. of fields from which the fungus was isolated/no. of fields sampled.

Pathogenicity tests of the Phytophthora isolates obtained revealed that all are very pathogenic to 'Vernal' alfalfa. The fungus killed 2-month-old plants in 2 weeks at 28C in a greenhouse under wet soil conditions.

The present survey indicated that phytophthora root rot is present in most of the soils in Ontario where alfalfa is grown. The disease becomes epidemic when the soil remains excessively wet during periods of prolonged heavy rainfall. Under favorable moisture conditions, the fungus may invade alfalfa plants and cause damage in a very short time. It is a potentially serious alfalfa problem in some areas, especially where the soils are poorly drained.

Literature cited

1. Chi, C.C. 1966. Phytophthora root rot of alfalfa in Canada. Plant Dis. Rep. 50:451-453.

2. Eckert, J.W., and P.H. Tsao 1962. A selective antibiotic medium for isolation of Phytophthora and Pythium from plant roots. Phytopathology 52:771-777.

3. Irwin, D.C. 1954. Root rot of alfalfa caused by Phytophthora cryptogea. Phytopathology 44:700-704.

4. Frosheiser, F.I. 1967. Phytophthora root rot of alfalfa in Minnesota. Plant Dis. Rep. 51:679-681.

FIRST RECORD OF WHITE ROT OF ONION IN COASTAL BRITISH COLUMBIA

D.J. Ormrod and J.F. Conroy[1]

White rot caused by Sclerotium cepivorum Berk. has not previously been observed on onions in coastal British Columbia, although it was seen in a small planting of garlic (Allium sativum L.) in 1951 (2) and has more recently been observed on onions (Allium cepa L.) in the southern Interior of the province (1,3). It occurs commonly in the onion growing regions of the western United States.

In June 1970 a grower in the Cloverdale muck vegetable growing area observed a disease in one field of bulb onions (Allium cepa L.) which proved to be white rot. Examination of the field showed the disease to be apparent in two small patches totalling approximately 1/4 acre.

Inquiries relative to the possible source of inoculum revealed that the field was also seeded to onions in 1969 but had not grown onions before that. Prior to 1967, the farm was used for livestock and hay production for many years. In 1965, fruit and vegetable waste and trimmings from a major Vancouver supermarket chain were used on the farm as a source of feed for cattle. Green bunching and bulb onions grown in the western United States were included in this refuse and it appears likely that some white rot infected onions were present.

The field in which the disease was detected in 1970 is the same field which received the majority of the refuse as evidenced by the presence of elastic bands and other non-decomposed packaging material.

Seed has been ruled out as a possible source of inoculum as other fields planted with the same seed in 1969 and 1970 were not affected.

Literature cited

1. British Columbia Department of Agriculture. 1970. Vegetable production recommendations, 1970. Victoria, B.C. 41p.

[1] Plant Pathologist and Horticulturist, respectively, British Columbia Department of Agriculture, Surrey, B.C.

2. Conners, I.L. 1967. An annotated index of plant diseases in Canada. Can. Dep. Agr. Pub. 1251. 381p.

3. Creelman, D.W. 1967. Summary of the prevalence of plant diseases in Canada in 1965 [1966]. Can. Plant Dis. Surv. 47:31-67.

NEWLY RECORDED FUNGI FROM COLONIAL BENTGRASS IN COASTAL BRITISH COLUMBIA

D.J. Ormrod, E.C. Hughes[1], and R.A. Shoemaker[2]

During a routine laboratory examination of a turf disease specimen some difficulty was experienced in detecting a known pathogen which might account for the visual symptoms of damage.

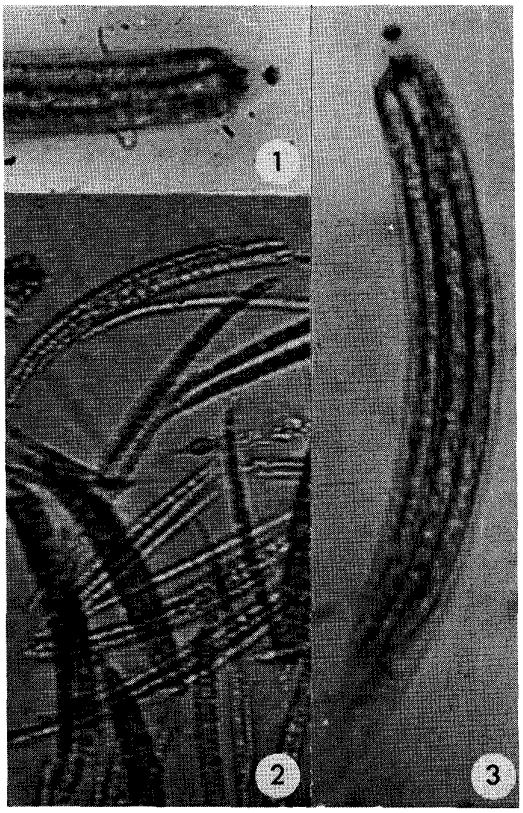
The over-all appearance of the disease, in a home lawn in Delta, B.C., corresponded to ophiobolus patch (Gaeumannomyces graminis (Sacc.) v. Arx & Olivier ≡ Ophiobolus graminis Sacc.), a disease which is common in Western Washington and which is believed to be common in the Fraser Valley of British Columbia, although not officially recorded (1,4). The symptoms of the disease were the same as Ophiobolus patch, namely depressed, circular areas of straw colored grass ranging from a few inches to several feet in diameter with the centres of the larger patches eventually filling in with coarse grasses and broad-leaved weeds (2).

Detailed laboratory examination of the samples received in June, 1970 failed to reveal the presence of G. graminis. As a result, further samples were obtained in July and August, at which times information on the history of the lawn was obtained.

The lawn was established as part of a new landscape project in the spring of 1969. A mixture of Kentucky bluegrass (Poa pratensis L.), creeping red fescue (Festuca rubra L.), and 'Highland' colonial bentgrass (Agrostis tenuis Sibth.) was used. Balanced fertilizer, hydrated lime, and sugar beet seed cleanings, as a mulch, were added and the lawn grew well until September of 1969 when the disease first appeared. No fungicides were applied and by the summer of 1970 large areas of the lawn were virtually destroyed.

During the laboratory examination, it was found that bentgrass comprised over 90% of the turf, the other grasses having been decimated by the close-mowing regime. Thus, the disease was primarily affecting Highland colonial bentgrass (Agrostis tenuis Sibth). Kentucky bluegrass and creeping red fescue were among the species which recolonized the centers of the patches.

In the last sample, after many fruitless searches, G. graminis was found. It matched the description given by Dickson (3) (Figs. 1-3).



Figures 1-3. *Gaeumannomyces graminis*. Figures 1 and 3. Asci stained with ink to show apical ring, DAOM 19596, X1000. Figure 2. Asci and ascospores, DAOM 133679, X ca. 450.

Various other fungi were found in the samples. Two, in particular, were noted because they had not been seen previously in numerous investigations of this kind over the past few years. These have been identified as Leptosphaerulina australis McAlpine, DAOM

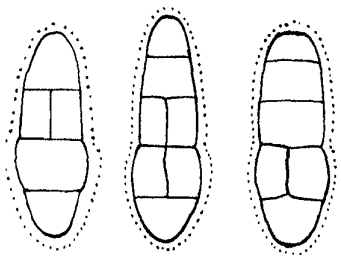


Figure 4. *Leptosphaerulina australis*, DAOM 116550, ascospores, X1000.

---

[1] Plant Pathologist and Field Crops Specialist, respectively, British Columbia Department of Agriculture, Surrey, B.C.

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

130612 (Fig. 4), and Robillarda agrostidis Sprague (Mycologia 31:47. 1939), DAOM 130613 (Fig. 5). This is the first Canadian record

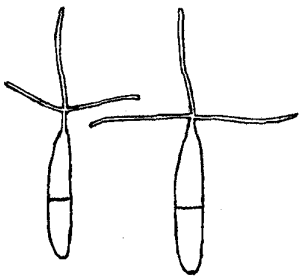


Figure 5. *Robillarda agrostidis*, DAOM 130613, conidia, X1000.

of the latter and the first published record of the Leptosphaerulina although there are Canadian herbarium specimens: DAOM 116550, bowling green grass (? Agrostis tenuis Sibth.), Kamloops, B.C., H.S. Pepin, 27 July 1967. DAOM 124546 isolated from Bromus inermis Leyss., leafspot, Saskatoon, Sask., J.D. Smith, 28 Sept. 1968.

The role of these two fungi in the overall disease syndrome is unknown. Robillarda agrostidis is listed as causing leaf rot of colonial bentgrass in Oregon (5). As the seed used on this lawn was grown in Oregon, the source of inoculum would appear to be self-evident.

Literature cited

1. Conners, I.L. 1967. An annotated index of plant diseases in Canada. Can. Dep. Agr. Publ. 1251. 381 p.

2. Couch, H.B. 1962. Diseases of turf grasses. Reinhold Publication Corp., New York. 289 p.

3. Dickson, J.G. 1947. Diseases of field crops. McGraw Hill Book Co., New York. 429 p.

4. Toms, H.N.W. 1964. Plant diseases of Southern British Columbia - a host index. Can. Plant Dis. Surv. 44:143-225.

5. U.S. Department of Agriculture. Agricultural Research Service, Crops Research Division. 1960. Index of plant diseases in the United States. U.S. Dep. Agr. Handbook 165. 531 p.

DISEASES AND OTHER FACTORS AFFECTING AVERAGE YIELDS OF BARLEY IN MANITOBA, 1954-1968[1]

W.C.McDonald[2]

Abstract

In 11 of the 15 years under consideration, diseases were responsible for decreasing average yields of barley considerably below those expected under prevailing conditions of management and weather. Epidemics of stem rust occurred in 6 of the 15 years, and in 1954 alone the loss due to stem rust was over $9 million. Significant losses from one or more of the foliage diseases occurred in 10 of the 15 years and the total loss amounted to over $21 million. Virus diseases decreased yields in at least 4 of the 15 years, and the total loss from aster yellows alone was over $8 million. Annual losses from smut have been limited by the use of seed treatment chemicals. Nevertheless losses over the 15-year period exceeded $7 million. The use of varieties developed during this period that are resistant to rust and smut eliminated much of this loss and resulted in a gain of nearly $8 million. Good management practices appeared to be more important than weather or varieties in achieving a high average yield of barley in Manitoba.

Introduction

The acreage of barley grown in Manitoba decreased by 77% from 1954 to 1962 and many reasons were proposed for farmer disillusionment with this crop. One of the factors believed to be a primary cause of low yields was the effect of diseases. However, although the prevalence of diseases was reported yearly in the Canadian Plant Disease Survey, no estimates of actual losses were made. In this paper losses from diseases and gains from the use of resistant varieties are estimated from an analysis of yield data for the period 1954-1968. The effects of diseases and other factors on the yearly variations in yield are also discussed.

Methods

Comparative yield data for the three most popular varieties grown in Manitoba from 1954 to 1968, Montcalm, Parkland and Conquest, were obtained from reports on the Western Cooperative Barley Tests. Each variety represented a group of commonly grown varieties with similar yields and disease susceptibility. 'Montcalm', susceptible to all diseases, represented 'OAC 21'; 'Parkland', resistant to stem rust, represented 'Vantage' and 'Herta' (although it is not rust resistant, Herta's yield has been similar to that of 'Parkland' in the rust-free years since it became popular in 1965); and 'Conquest', resistant to stem rust and smut, represented 'Keystone'. The yields are averages of the four test locations in Manitoba: Winnipeg, Portage la Prairie, Morden, and Brandon. Since 'Montcalm' was not included in the cooperative tests after 1964, the yield of 'OAC 21' was substituted from 1965 to 1968.

Data on the Manitoba average yield, total acreage, and price were obtained from the Yearbook of Manitoba Agriculture 1968 (3), and the percentage of the acreage sown to varieties susceptible to stem rust and smut, to varieties resistant to stem rust, and to varieties resistant to both stem rust and smut was provided by the former Line Elevators Farm Service, Winnipeg. The yields from the cooperative tests were reduced by a conversion factor calculated by summing the products of test yield x acreage of each variety, and dividing that sum by the total Manitoba production. The potential yield of a variety was calculated by multiplying the converted yield by 100 and dividing by 100 minus the percentage loss from all diseases. Total yield losses from disease and gains from resistance were based on the acreage of susceptible or resistant varieties.

The loss from stem rust was assumed to be the difference in yield between 'Montcalm' and the resistant varieties 'Parkland' and 'Conquest', minus an allowance for the inherent advantage in yielding ability of the latter two. The inherent advantage in yield was calculated by comparing the average increase, in rust-free years, of 'Parkland' over 'Montcalm' as a percentage of 'Parkland' yield, and similarly the average increase of 'Conquest' over 'Montcalm' as a percentage of 'Conquest' yield. These calculations showed that 'Montcalm' yielded 8% less than 'Parkland' and 13% less than 'Conquest' in the absence of diseases to which the latter two varieties are resistant.

---

[1] Contribution No. 449, Research Station, Canada Department of Agriculture, Winnipeg 19, Manitoba.

[2] Plant pathologist.

The yearly average percentage of smut in barley in Manitoba is recorded in the Proceedings of the Manitoba Agronomists Conference and these figures were used to calculate losses on susceptible varieties. In 1965 the acreage of smut resistant varieties began to increase with a concomitant reduction in the average percentage of smut. Therefore, to assess the losses on susceptible varieties and the gains from resistance an average of the percentages of smut in the previous 11 years was used.

No data are available on the percentage loss caused by foliage or virus diseases in Manitoba during this period but reports on the prevalence and severity of these diseases in epidemic years appear in the Canadian Plant Disease Survey. Results from experiments using fungicidal sprays (1), varietal comparisons (4), or inoculations (2) showed that leaf spot diseases can cause

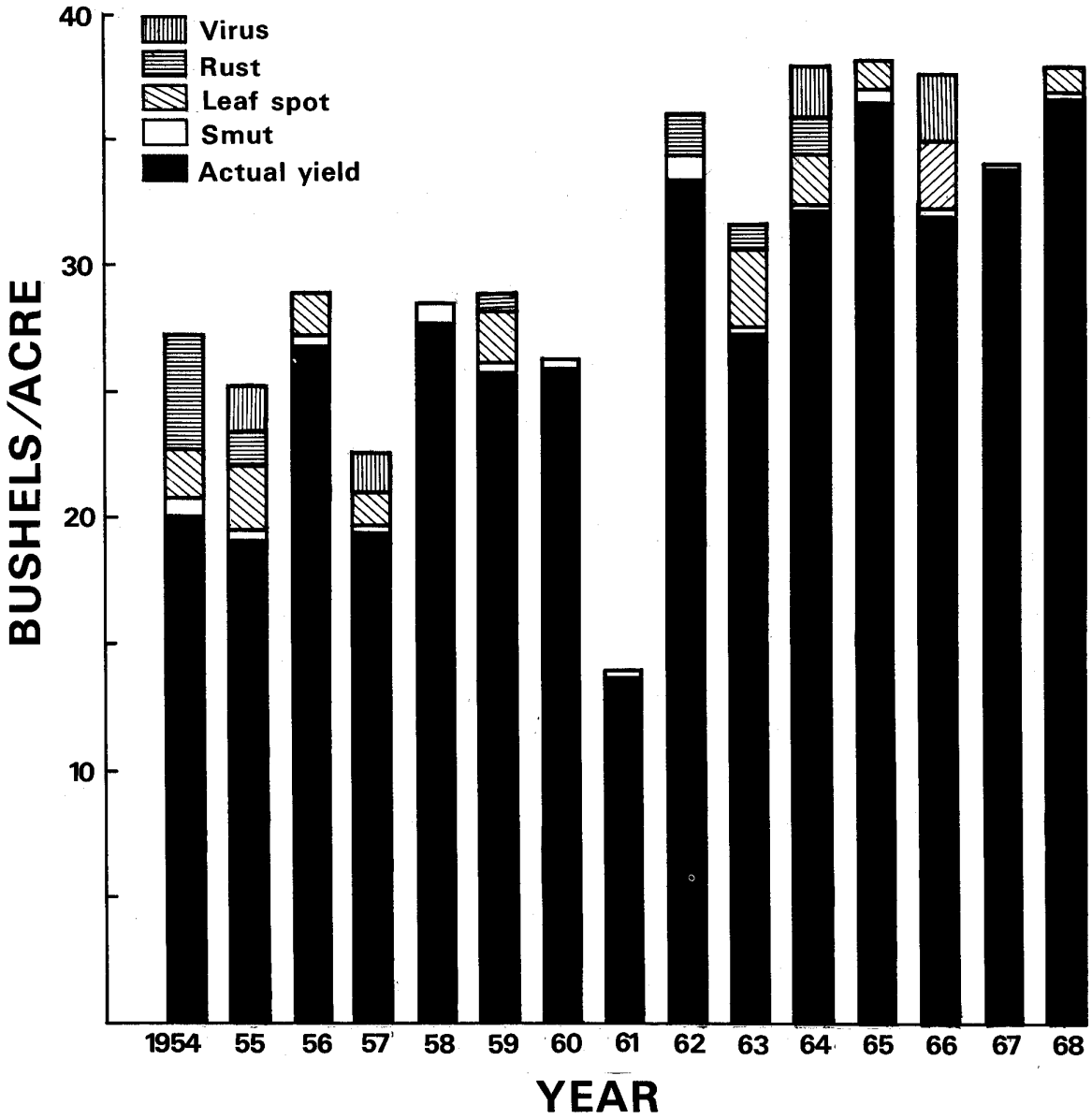


Figure 1. The effect of diseases on barley yields in Manitoba, 1954–68 The solid portion of a bar represents the actual average yield; the area above the solid portion represents the estimated yield loss from diseases. The top of a bar, therefore, represents the estimated potential average yield in the absence of disease.

Table 1. Yield, acreage, and loss from diseases of barley in Manitoba, 1954-68

			1954	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964	1965	1966	1967	1968
Test yield -	Montcalm	bu/ac	47.3	54.2	61.1	52.3	62.9	61.7	55.7	43.1	55.0	41.9	52.6	62.3	49.6	64.1	68.4
	Parkland	bu/ac	64.1	63.5	68.7	61.9	71.4	71.1	61.7	45.5	68.0	48.8	68.6	68.0	52.4	67.7	70.5
	Conquest	bu/ac										55.6	77.2	72.3	56.5	69.3	77.2
Acreage	Montcalm	%	93	89	88	83	57	46	45	42	41	34	28	21	12	7	4
	Parkland	%	7	11	12	17	43	54	55	58	59	58	57	54	46	22	23
	Conquest	%										8	15	25	42	71	73
Conversion factor			2.424	2.891	2.305	2.780	2.394	2.578	2.261	3.248	1.876	1.715	2.031	1.855	1.681	2.017	2.046
Manitoba average yield		bu/ac	20.0	19.1	26.9	19.4	27.8	25.9	26.1	13.7	33.4	27.4	32.2	36.6	32.0	34.0	36.8
Converted yield	Montcalm	bu/ac	19.5	18.7	26.5	18.8	26.3	23.9	24.6	13.3	29.3	24.4	25.9	33.6	29.5	31.8	33.4
	Parkland	bu/ac	26.4	22.0	29.8	22.3	29.8	27.6	27.3	14.0	36.2	28.5	33.8	36.7	31.2	33.6	34.5
	Conquest	bu/ac										32.4	38.0	39.0	33.6	34.4	37.7
Disease loss	Montcalm	%	27.8	25.1	6.8	14.0	2.3	13.7	1.3	2.6	15.2	19.7	28.0	4.8	15.8	1.8	4.8
	Parkland	%	9.6	18.4	6.8	14.0	2.3	8.5	1.3	2.6	3.0	10.8	10.9	4.8	15.8	1.8	4.8
	Conquest	%										10.0	10.0	3.0	14.0	0.0	3.0
Potential yield	Montcalm	bu/ac	27.0	25.0	28.4	21.9	26.9	27.7	24.9	13.6	34.6	30.4	36.0	35.3	35.0	32.4	35.1
	Parkland	bu/ac	29.3	26.9	32.0	25.9	30.5	30.1	27.7	14.4	37.4	31.9	37.9	38.5	37.0	34.2	36.2
	Conquest	bu/ac										36.0	42.2	40.2	39.1	34.4	38.9
Average disease loss		bu/ac	7.2	6.1	2.0	3.2	0.7	3.1	0.3	0.4	2.8	4.3	5.8	1.7	5.7	0.2	1.3
Total acreage		000ac	2202	2090	1548	1704	1584	1270	930	655	629	584	497	601	875	970	1170
Total disease loss		000bu	15,820	12,823	3,108	5,381	1,036	3,976	319	239	1,770	2,525	2,886	995	4,947	170	1,542
Price		$/bu	0.92	0.94	0.82	0.79	0.81	0.78	0.84	1.05	1.00	0.92	1.02	1.05	1.10	0.91	0.85
Total disease loss		$000	14,555	12,054	2,549	4,251	839	3,101	268	251	1,770	2,322	2,944	1,045	5,442	155	1,311

about 20% loss in yield when severe. On this basis, losses in years when the diseases were widespread in farm fields were conservatively estimated as follows: severe, 10%; moderate to severe, 7%; moderate, 5%; and light to moderate, 3%. Similarly, no data are available on the extent of the damage caused by aster yellows and barley yellow dwarf on barley prior to 1964. However, epidemics of aster yellows on other crops occurred in 1955 and 1957 and it is assumed that barley was affected also. In 1966, a loss of 7% from this disease on barley was estimated by Westdal (5), so this figure was used as an estimate of the loss in the other 2 years.

Results and discussion

Effect of disease incidence on average yield

The potential average yield of barley in Manitoba in the absence of major disease epidemics is shown in Fig. 1, and the losses caused by disease are tabulated in Table 1. In 11 of the 15 years, diseases were responsible for decreasing the average yields considerably below those expected under the prevailing conditions of management and weather.

Major epidemics of stem rust (Puccinia graminis Pers.) occurred in 1954, 1962, and 1964, and less severe epidemics in 1955, 1959, and 1963. In 1954, when over 90% of the acreage was sown to susceptible varieties, the total loss was over $9 million and the gain from growing 'Vantage' was over $680,000 (Table 2). By 1964, only 28% of the acreage was sown to susceptible varieties and the loss was $874,000 compared to a gain of $1.7 million (Table 2) from the use of resistant varieties. Although rust does not appear every year, severe losses occur in epidemic years and rust resistance is mandatory for successful barley production in Manitoba.

The annual loss from smut diseases of barley caused by Ustilago nuda (Jens.) Rostr., U. nigra Tapke, and U. hordei (Pers.) Lagerh. varied little from 1954 to 1964 and averaged 1.8%. This comparatively low figure reflects the use of seed treatment chemicals for control, because losses as high as 12% still occur in fields of susceptible varieties grown from untreated seed. Losses over the 15-year period amounted to over $7 million, and with the introduction of smut-resistant varieties the gain has been $1.3 million.

Losses from foliage diseases include those caused by the three main diseases that occur in Manitoba; net blotch (Pyrenophora teres [Died.] Drechsl.), spot blotch (Cochliobolus sativus [Ito & Kurib.] Drechsl.), and septoria leaf blotch (Septoria passerinii Sacc.). Significant losses occurred from one or more of these diseases in 10 of the 15 years, and the total loss amounted to over $21 million.

Virus diseases decreased yields in at least 4 of the 15 years. Total losses from aster yellows were estimated to be over $8 million for the three major epidemics reported in 1955, 1957, and 1966. A loss from barley yellow dwarf was recorded only in

Table 2. Value of resistance to stem rust and to smut in barley varieties grown in Manitoba, 1954-68

Year	Yield loss on susciptible varieties (%)	Yield of resistant varieties (bu/ac)	Yield increase from resistance (bu/ac)	Acreage of resistant varieties (000 ac)	Increase in production (000 bu)	Price ($/bu)	Value of resistance ($000)
Stem rust							
1954	18.2	26.4 (P)*	4.8	154	739.2	0.92	680.1
1955	6.7	22.0 (P)	1.5	230	345.0	0.94	324.3
1959	5.2	27.6 (P)	1.4	686	987.8	0.78	770.5
1962	12.2	36.2 (P)	4.4	371	1639.8	1.00	1639.8
1963	8.9	28.5 (P)	2.5	338	858.5	0.92	789.8
		32.4 (C)	2.9	47	134.5	0.92	124.6
1964	17.1	33.8 (P)	5.8	283	1635.7	1.02	1668.4
		38.0 (C)	6.5	75	487.5	1.02	497.3
						Total	6494.8
Smut							
1963	0.8	32.4 (C)	0.3	47	12.2	0.92	11.2
1964	0.9	38.0 (C)	0.3	75	25.5	1.02	26.0
1965	1.8	39.0 (C)	0.7	150	105.0	1.05	110.3
1966	1.8	33.6 (C)	0.6	368	224.5	1.10	247.0
1967	1.8	34.4 (C)	0.6	685	424.7	0.91	386.5
1968	1.8	37.7 (C)	0.7	854	580.7	0.85	493.6
						Total	1274.6

* P = Parkland, C = Conquest.

1964. Losses from this disease probably occurred in other years but the effects of the disease were not recognized by those surveying for barley diseases prior to 1964.

Effect of weather conditions on average yield

Weather conditions varied between extreme drought in 1961 to excessive moisture in 1968 and accounted for some of the variations in average yield experienced during this period. Hot, dry weather reduced yields in 1957, 1961, 1963, and 1967, whereas cool, wet weather and absence of severe epidemics of disease contributed to near-record average yields in 1965 and 1968. Although weather conditions affected yearly fluctuations in yield, they are not believed to have contributed to the marked increase in average yield evident during the period after 1961. The Manitoba average yield for the period 1962 to 1968 was 30% higher than for the period 1954 to 1960, but the average yield of 'Parkland' in the cooperative tests was 3% lower. If weather was a factor it should have influenced the yield of 'Parkland', which was grown under uniform, optimum management conditions during the same periods.

Effect of varieties on average yield

From 1954 to 1960, varieties susceptible to stem rust and smut were grown on over 45% of the acreage in Manitoba, and from 1962 to 1968 the use of higher yielding, disease resistant varieties increased to 96% of the acreage. The increased yield from these varieties contributed significantly to the higher Manitoba average yields obtained during the latter period but does not account for all of the increase. The average yield of barley in Manitoba from 1962 to 1968 was 30% higher than in the period 1954-60. However, the Manitoba average yield and the yield of 'Montcalm' differed by only 11% for the period 1962-68. The yield difference between the Manitoba average, which reflects the acreage and yield of new varieties, and 'Montcalm' should have been greater if varietal improvement was mainly responsible for the better performance of barley in recent years.

Effect of management on average yield

Good management practices such as the use of quality seed of recommended varieties, early seeding, seed treatment where necessary, weed control, soil testing, and the optimum use of fertilizers have been strongly recommended to obtain increased yields of barley. It appears from the analysis of these data that improved management has been the main factor in achieving higher average yields in recent years. The conversion factor, which relates the yields obtained under optimum management conditions in experimental plots and the Manitoba average yield, was considerably smaller in each of the years 1962 to 1968

than in any year previous to 1961 (Table 1). As disease, weather, and varieties have no bearing on this figure, it must be concluded that better management of barley has decreased the difference between yields in farm fields and those in experimental tests. The acreage of barley dropped from a peak of 2,365,000 acres in 1953 to 497,000 acres in 1964, the lowest acreage since 1914. Possibly only those farmers who were using the best management practices and obtaining satisfactory yields continued to grow barley, and the higher Manitoba average yield reflects the yields obtained by only the best growers.

Conclusions

Estimates of losses from diseases and gains from resistance are only as accurate as the data on which they are based. In most studies, as in this one, the lack of data on disease incidence has been the limiting factor. Information is available on the yield losses caused by specific diseases under experimental conditions, but the results of these studies must be correlated with extensive surveys over all of the area involved to determine the prevalence of each disease. Recognizing this limitation, I have been as conservative as possible in extrapolating losses from these data.

Losses were assessed only in years when epidemics of specific diseases were known to occur. Losses in other years or from other diseases such as root rot, seedling blight, ergot, and bacterial blight, for which adequate data on incidence were not available, were not included. Average losses varied from 0.3 bu/ac in 1960 to 7.2 bu/ac in 1954 and the total loss for the 15-year period amounted to $52,846,000. These losses reflect estimates of decreased yield only, and no attempt was made to assess the decrease in value of the crop resulting from the effects of diseases on quality.

It should be emphasized that the total gain of $7,769,400 (Table 2) from the use of resistant varieties does not reflect the total value of these varieties to the economy. This figure only represents the value of their disease resistance and does not include the value of their inherent yield advantage over older varieties. To obtain estimates of their true value, similar studies would have to be made in Saskatchewan and Alberta where 75% of the barley in Western Canada is grown.

The results substantiate conclusions reached previously by those working on barley improvement in Manitoba. Extension services must place greater emphasis on promoting good management practices, and research programs must place greater emphasis on developing high yielding varieties with resistance to foliage diseases as well as to rust and smut, and on developing control measures for virus diseases through varietal resistance or other methods.

Literature cited

1. Buchannon, K.W., and H.A.H. Wallace. 1962. Note on the effect of leaf diseases on yield, bushel weight and thousand-kernel weight of Parkland barley. Can. J. Plant Sci. 42:534-536.

2. Green, G.J., and V.M. Bendelow. 1961. Effect of speckled leaf blotch, Septoria passerinii Sacc., on the yield and malting quality of barley. Can. J. Plant Sci. 41:431-435.

3. Manitoba Department of Agriculture. Yearbook of Manitoba Agriculture, 1968. Queen's Printer for Province of Manitoba.

4. McDonald, W.C., and K.W. Buchannon. 1964. Barley yield reductions attributed to net blotch infection. Can. Plant Dis. Surv. 44:118-119.

5. Westdal, P.H. 1968. The experimental approach in assessing disease losses in

ELYMANA VIRESCENS, A NEWLY DESCRIBED VECTOR OF WHEAT STRIATE MOSAIC VIRUS[1]

R.C.Sinha

Abstract

Wheat striate mosaic virus (WSMV) was shown to be transmitted by the leafhopper Elymana virescens (F.). However, fewer adult E. virescens became inoculative (23%) after an acquisition access period of 3 days than did Endria inimica (Say) (90%) - a known vector of the virus. In addition, the minimum incubation period of WSMV was much longer in E. virescens (15-18 days) than in E. inimica (4 - 6 days). Wheat (Triticum durum Desf.) plants that became infected after being inoculated by viruliferous E. virescens showed typical bacilliform WSMV particles (about 260 X 80 mu in size) both in the cytoplasm and in the nucleus of the parenchymatous leaf cells.

Introduction

Up to the present study the leafhopper Endria inimica (Say) was the only vector reported to transmit the North American wheat striate mosaic virus (WSMV) (7). The virus is transmitted in a persistent manner and has been shown to multiply in the leafhopper vector (5). This communication reports the transmission of WSMV by Elymana virescens (F.) and the efficiency of this newly described vector to transmit the virus as compared to that of E. inimica.

The particles of WSMV are bacilliform in shape, about 260 X 80 mu in size, and can easily be identified in infected plants (3). For this reason, ultrathin sections of wheat (Triticum durum Desf. cv. Ramsey) leaves that became infected after being inoculated by viruliferous E. virescens, were also examined in an electron microscope.

Materials and methods

The virus was maintained in wheat plants infected by means of viruliferous leafhoppers, E. inimica. Colonies of virus-free leafhoppers were reared and maintained on healthy wheat plants. Adult E. virescens collected from the Central Experimental Farm, Ottawa, were maintained on healthy barley (Hordeum vulgare L.) plants. Although WSMV has not been reported from the Ottawa area, samples of field-collected leafhoppers were tested in groups on healthy wheat plants (1 week each on four successive plants) to ensure that the leafhoppers were not carrying WSMV. None of the test plants developed symptoms of WSMV.

"Exposed" leafhoppers were obtained by caging virus-free insects on WSMV-infected wheat plants. During the incubation period of WSMV in the insects, groups of exposed leafhoppers were maintained on wheat plants and were transferred to new plants every 3 or 4 days (5). Not all exposed insects transmitted the virus when tested singly for their ability to do so; those that did have been referred to as "inoculative". During the acquisition access or test feeding periods, leafhoppers were held in a growth room with a night-day temperature range of 19-21 C and 10,000 lux of light for 16 hours a day. After each test feeding, the plants were sprayed with nicotine sulphate and were held in a greenhouse for at least 4 weeks to observe possible symptom development.

For electron microscopy, wheat leaves were processed and sectioned following the procedure described earlier (6) and examined in a Siemens Elmiskop I.

Results and discussion

To determine the percentage transmission by E. virescens and to compare it with E. inimica, adult leafhoppers of both species were caged for various periods on the same WSMV-infected plants. After the acquisition access period, the exposed leafhoppers of the two species were maintained separately for 2 additional weeks on healthy plants and the surviving insects were then tested singly for 2 weeks for their inoculativity on wheat seedlings. The combined results of two such experiments with each acquisition access period showed (Table 1) that, in each case, fewer E. virescens transmitted the virus than did E. inimica. These results indicate that E. virescens is a less efficient vector of WSMV as compared to E. inimica.

Although the symptoms produced on wheat plants by inoculative E. virescens were typical of WSMV, attempts were made to transmit the virus from such infected plants to healthy wheat seedlings by using E.

[1] Contribution No. 687, Cell Biology Research Institute, Research Branch, Canada Department of Agriculture, Ottawa, Ontario.

Table 1. Transmission of wheat striate mosaic virus by Elymana virescens as compared with Endria inimica after different acquisition access periods

Acquisition access period (in days)*	Transmission by	
	Elymana virescens	Endria inimica
1	0/36 (0%)	18/30 (60%)
2	1/30 (3%)	29/36 (81%)
3	9/43 (23%)	37/41 (90%)

* After each acquisition access period, leafhoppers were maintained for 2 additional weeks on healthy plants and then were tested singly for 2 weeks for their inoculativity on healthy wheat seedlings. Numerator is the number of insects that became inoculative; denominator is the number tested.

inimica. Groups of 20 healthy adult E. inimica were caged for 3 days on the infected plants and were then tested singly for their inoculativity as described above. The results of two such experiments showed that 26/31 (84%) E. inimica became inoculative.

To obtain further evidence that the symptoms produced on wheat plants by the viruliferous E. virescens were indeed caused by WSMV, ultrathin sections of infected leaves were examined in the electron microscope. Typical WSMV particles were found in both cytoplasm and nucleus of the parenchymatous cells (Fig. 1).

The minimum incubation period of WSMV in E. inimica has been reported to be between 4 and 6 days and the maximum between 24 and 28 days (7). To determine the incubation period of the virus in E. virescens, 50 adult leafhoppers were given an acquisition access period of 3 days and were then transferred singly to wheat seedlings every 3 or 4 days, up to 28 days after the start of acquisition.

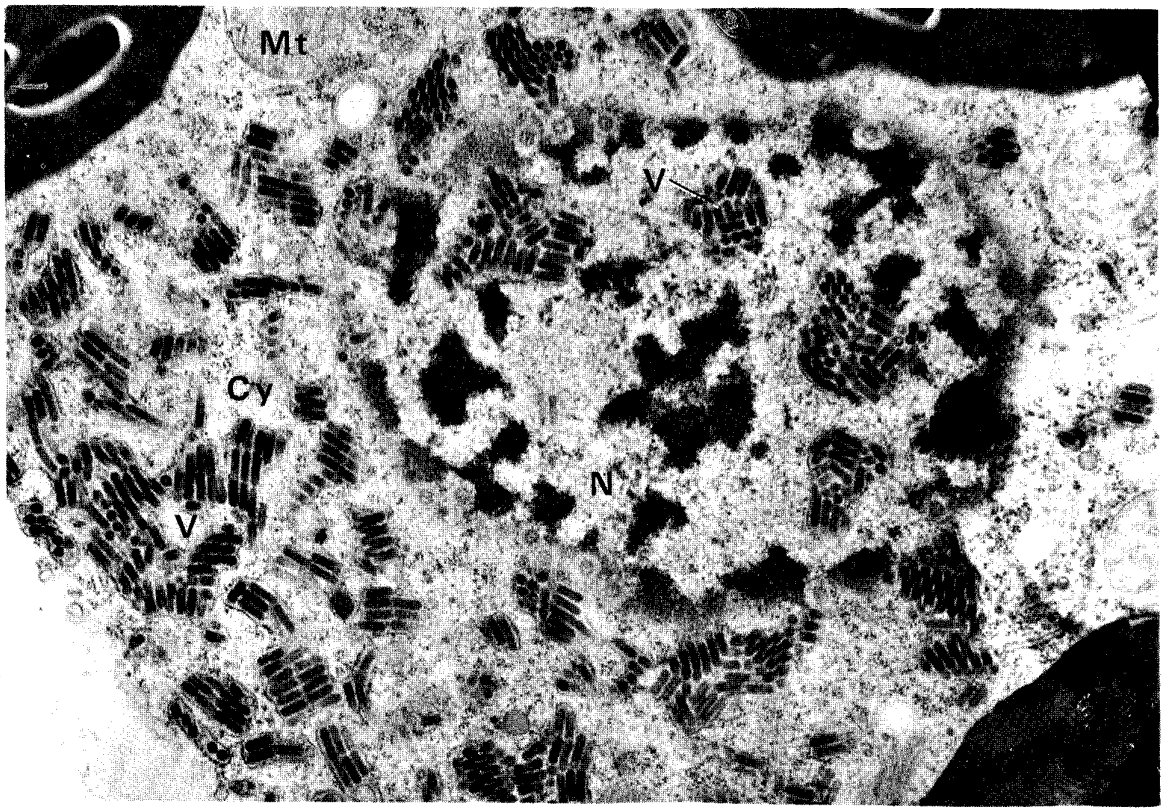


Figure 1. Electron micrograph of a section through a parenchymatous cell of a diseased wheat leaf. The plant had become infected by wheat striate mosaic virus after being inoculated by a viruliferous *Elymana virescens*. x 20,000. Note that the bacilliform virus particles (V) are present in both cytoplasm (Cy) and nucleus (N) of the cell. Mt: mitochondria.

A similar experiment was carried out with E. inimica. The results with E. inimica confirmed the earlier report by Slykhuis (7) regarding the incubation period of WSMV in this vector. The results with six E. virescens that became inoculative and survived for 28 days are given in Table 2.

Table 2. Transmission of wheat striate mosaic virus by inoculative Elymana virescens at different times after the start of a 3-day acquisition access period

Leafhopper number	Days from start of acquisition access period to test feed*				
	4 to 15	18	22	25	28
1	-**	-	-	+	+
2	-	-	+	+	+
3	-	+	+	+	+
4	-	-	+	+	+
5	-	-	-	-	+
6	-	-	+	+	-

* After the acquisition access period of 3 days, leafhoppers were transferred singly to wheat seedlings every 3 or 4 days. Transmission results are given only for those leafhoppers that became inoculative and survived for at least 28 days.

** - = no symptoms on test plants; + = test plant became infected.

The minimum incubation period of WSMV in E. virescens was between 15 and 18 days and the maximum was between 25 and 28 days. The longer minimum incubation period of WSMV in E. virescens could result because of the slower movement of the virus from the gut to the hemolymph and then to the salivary glands (4) and/or the virus may multiply at a slower rate in E. virescens than in E. inimica.

It is noteworthy that E. virescens is also capable of transmitting the causal agent of aster yellows (1). It will be of interest, therefore, to study the interactions in E. virescens between WSMV and the agent of aster yellows disease, which is suspected to be caused by a Mycoplasma sp. (2).

Acknowledgment

I am thankful to Mr. William Bell for technical assistance.

Literature cited

1. Chiykowski, L.N., and R.C. Sinha. 1969. Comparative efficiency of transmission of aster yellows by Elymana virescens and Macrosteles fascifrons and the relative concentration of the causal agent in the vector. J. Econ. Entomol. 62:883-886.

2. Doi, Y., M. Teranaka, K. Yora, and H. Asuyama. 1967. Mycoplasma - or PLT group-like microorgansims found in the phloem elements of plants infected with mulberry dwarf, potato witches' broom, aster yellows, or Paulownia witches' broom. Ann. Phytopathol. Soc. Japan 33:259-266.

3. Lee, P.E. 1964. Electron microscopy of inclusions in plants infected by wheat striate mosaic virus. Virology 23:145-151.

4. Sinha, R.C. 1968. Recent work on leafhopper transmitted viruses. Advan. Virus Res. 13:181-223.

5. Sinha, R.C., and L.N. Chiykowski. 1967. Multiplication of wheat striate mosaic virus in its leafhopper vector Endria inimica. Virology 32:402-405.

6. Sinha, R.C., and Y.C. Paliwal. 1969. Association, development, and growth cycle of Mycoplasma-like organisms in plants affected with clover phyllody. Virology 39:759-767.

7. Slykhuis, J.T. 1963. Vector and host relations of North American wheat striate mosaic virus. Can. J. Botany 41:1171-1185.

PYTHIUM INTERMEDIUM, A NEWLY RECOGNIZED PATHOGEN OF CONIFEROUS SEEDLINGS IN CANADA

D. Hocking[1]

Abstract

Pythium intermedium de Bary caused up to 50% mortality in 2- to 3-week-old container-grown seedlings of *Pinus contorta* Dougl. var. *latifolia* Engelm., *Picea glauca* (Moench) Voss var. *albertiana* (S. Brown) Sarg., *Picea engelmannii* Parry, and *Pseudotsuga menziesii* (Mirb.) Franco. It killed 3-week-old aseptic seedlings of all species *in vitro* within 3 days. This is the first report of this fungus as a pathogen of conifers in North America.

Introduction

Damping-off is a continuing problem in the raising of container seedlings for reforestation. Unusual devastation occurred during April 1969 in the greenhouses at the Provincial Tree Nursery, R.R. 6, Edmonton, Alberta. Mortality was widespread in all coniferous species being grown, reaching 40 to 50% by the 3rd week after emergence. This note reports on the pathogenicity of the organism principally responsible and previously unreported as a pathogen of conifers in North America.

Materials and methods

Seedlings of *Pinus contorta* Dougl. var. *latifolia* Engelm., *Picea glauca* (Moench) Voss var. *albertiana* (S. Brown) Sarg., *Picea engelmannii* Parry, and *Pseudotsuga menziesii* (Mirb.) Franco., were grown in the greenhouse on non-sterilized, locally dug sphagnum peat in split plastic tubes (3/4 inch diam. by 3 1/2 inches long), packed in plastic flats holding 220 tubes. Seeds were sown by a vacuum seeder, which deposited 2 to 8 seeds per tube. Irrigation for about 10 sec every half hour was by automatic mister, which had the additional function of reducing temperature during sunny periods and therefore tended to water the seedlings to excess. Temperature controls were set at 70F (21C), upon reaching which an extraction fan would cut in; however temperatures exceeding 100F (38C) occurred for periods up to 1 hour or so almost daily. Thus, many conditions predisposing the seedlings to epidemic damping-off existed: overcrowding, water-logged organic soil, high humidity, high and periodically extreme temperatures, and inoculum in non-sterile seeds or substratum.

Soon after emergence, about 2 weeks after sowing, damping-off was observed, and isolations were started. At the end of each week, for 3 weeks, mortality was assessed on two full flats of each species of tree (Table 1).

Results and discussion

Isolations.-Each week, 20 damped-off seedlings of each species were collected from various flats and placed individually in separate vials. They were individually washed in sterile water, surface-sterilized in a 1:1 mixture (v/v) of ethanol and aqueous-saturated $HgCl_2$, and plated on 2% agar in tap water. Isolations were made from hyphal tips or spores growing on or out of the seedling fragments.

Most isolates from samples taken in the 1st week were pure cultures of *Pythium intermedium* de Bary. In the 2nd and 3rd weeks, *Pythium intermedium* was still the most common isolate. Other fungi, mostly *Fusarium* spp., also occurred, but only irregularly and

Table 1. Mortality of coniferous seedlings from damping-off at different seedling ages (1-3 weeks from emergence)

Species	1 week		2 weeks		3 weeks	
	Number sampled	Mortality (%)	Number sampled	Mortality (%)	Number sampled	Mortality (%)
Pinus contorta	760	28	678	44	683	58
Picea glauca	598	6	635	27	667	41
Picea engelmannii	616	19	763	38	706	50
Pseudotsuga menziesii	410	2	470	21	588	27

[1] Forest Research Laboratory, Canadian Forestry Service, Department of Fisheries and Forestry, Edmonton, Alberta.

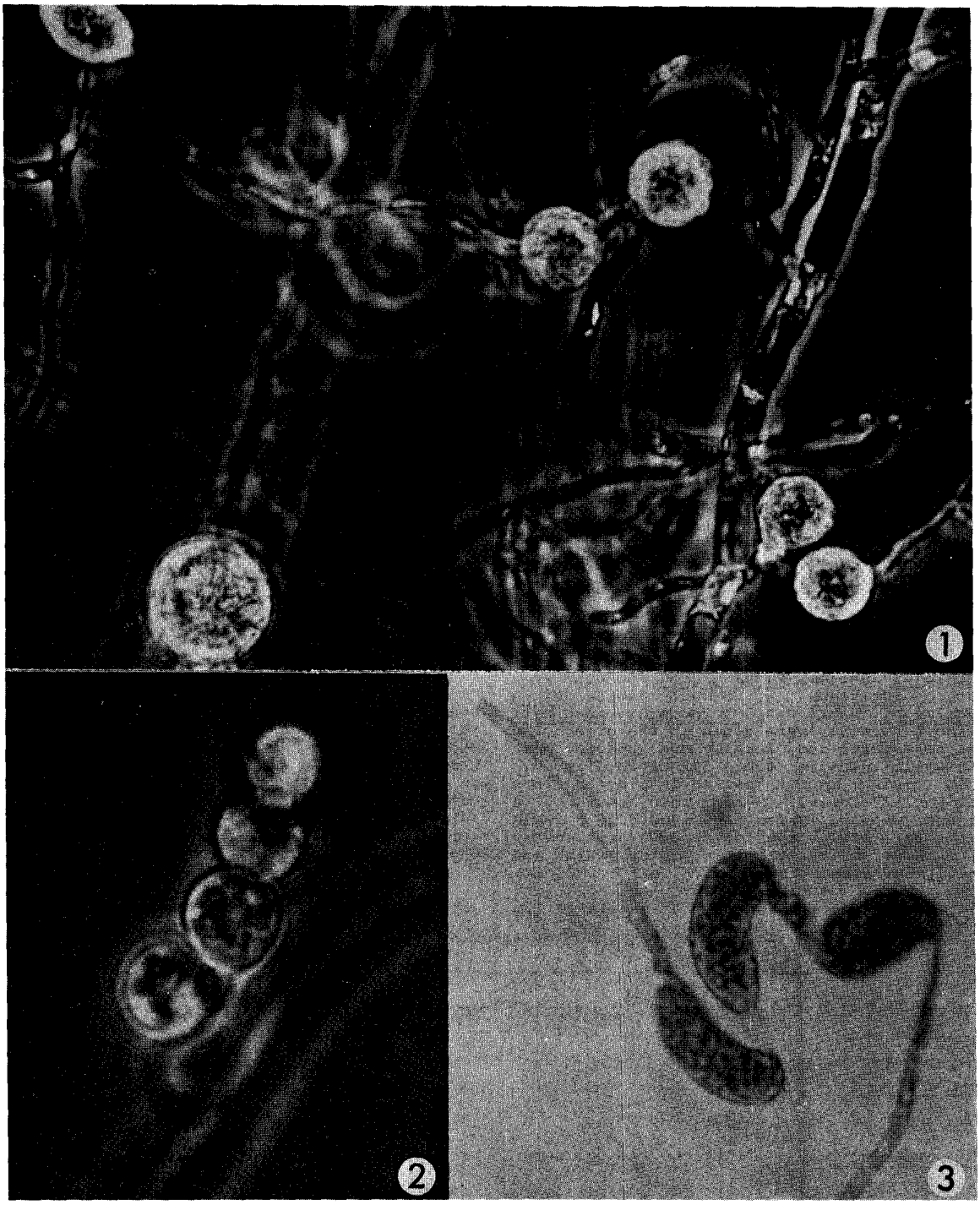


Figure 1–3. *Pythium intermedium*. 1) Single sporangia, X ca. 450; 2) Chain of sporangia, diagnostic of *P. intermedium*, X ca. 900; 3) Repetitive appressoria formed by *P. intermedium* from a sporangium germinating on a glass surface, X ca. 450.

in the later stages. During the first 3 weeks Pythium intermedium consistently grew out of root and collar fragments of seedlings.

Most cultures also yielded moderate to large numbers of nematodes. These were subsequently identified as belonging to the genera Rhabditis Dujardin (1845) and Cephalobus Bastian (1865) by Dr. J.R. Sutherland, Canadian Forestry Service, Victoria, B.C., and Dr. E.J. Hawn, Canada Department of Agriculture, Lethbridge, Alta. These genera are non-stylet-bearing and hence are not considered to be pathogenic to plants.

Isolates of Pythium intermedium were identified by Dr. D.J. Stamps, Commonwealth Mycological Institute, Kew, England; and Dr. O. Vaartaja, Canadian Forestry Service, Ottawa. Isolates did not form oogonia, perhaps because complementary sexual strains were lacking. Other workers have found isolates from Holland to be heterothallic (9). Single sporangia were frequently formed (Fig. 1). Most isolates, however, formed deciduous chains of sporangia (Fig. 2), a diagnostic characteristic unique to P. intermedium (1,5). In cultures on glass slides, germinating sporangia commonly formed repetitive appressoria (Fig. 3).

Inoculations. - To test the pathogenicity of Pythium intermedium, I inoculated, in vitro, aseptic tree seedlings. They were grown for 3 weeks from pre-germinated, surface-sterilized seeds on 2% tap water agar in 18 X 150 mm test tubes, under fluorescent light. Additional test-seedlings were grown aseptically in the greenhouse in 250 ml conical flasks containing vermiculite and a mineral nutrient solution.

Ten seedlings of each species grown in test tubes and 10 grown in flasks were inoculated by dropping a 5 mm diam by 3 mm thick agar plug from 3-day-old cultures of Pythium intermedium into each tube or flask. The seedlings were then incubated in the light at 22C. All the inoculated seedlings were killed by the 3rd day. Reisolations on water agar yielded pure cultures of Pythium intermedium. Uninoculated controls remained healthy and yielded no microorganisms when plated out.

Pythium intermedium de Bary is a rarely isolated member of the genus. Middleton (5) records 24 reports dating from the original description by de Bary in 1881 (1). This species is missing from recent accounts of damping-off of coniferous tree seedlings in the southern United States (2), Britain (3), and Canada (8). It is recorded from nursery soil in Britain (10) and the United States (4) and from Pinus halepensis Mill. seedlings in Australia (6). In Canada, there is only one published report of its occurrence, and that is from nursery soil (7).

Dr. A.W. Henry, in a personal communication, reports that in a study of root disease of ornamental elders (Sambucus spp.) during 1968 and 1969, he isolated Pythium intermedium from soil beneath diseased bushes from six widely separated areas in Alberta: Beaverlodge, Bowden, Ellerslie, Edmonton, Vulcan, and Devon.

Such widespread incidence suggests that earlier investigators may have overlooked the species, or simply relegated it to Pythium sp. The present report clearly establishes Pythium intermedium as a potentially dangerous pathogen to coniferous seedlings, especially when grown in containers in the greenhouse.

Literature cited

1. Bary, A. de. 1881. Zur Kenntniss der Peronosporeen. Bot. Z. 39:553-558.
2. Campbell, W.A., and F.F. Hendrix, Jr. 1967. Pythium and Phytophthora species in forest soils in the southeastern United States. Plant Dis. Rep. 51:929-932.
3. Griffin, D.M. 1965. A study of damping-off, root damage and related phenomena in coniferous seedlings in British forest nurseries, p. 212-227. In Blanche Benzian, Experiments on nutrition problems in forest nurseries, Vol. I. Gt. Brit. For. Comm. Bull. 37:1-251.
4. Hendrix, Floyd F., Jr., and W.A. Campbell. 1969. Distribution of Phytophthora and Pythium species in soils of the continental United States. Can. J. Bot. 48:337-384.
5. Middleton, J.T. 1943. The taxonomy, host range and geographic distribution of the genus Pythium. Mem. Torrey Bot. Club 20:1-171.
6. Vaartaja, O. 1967. Damping-off pathogens in South Australia nurseries. Phytopathology 57:765-768.
7. Vaartaja, O. 1967. Pythium and Mortierella in soils of Ontario forest nurseries. Can. J. Microbiol. 14:265-269.
8. Vaartaja, O., and W.H. Cram. 1956. Damping-off pathogens of conifers and of Caragana in Saskatchewan. Phytopathology 46:391-397.
9. Van Der Plaats-Niterink. 1968. The occurrence of Pythium in the Netherlands 1. Heterothallic species. Acta Bot. Neer. 17:320.
10. Warcup, J.H. 1952. Effect of partial sterilization by steam or formalin on damping-off of Sitka spruce. Trans. Brit. Mycol. Soc. 35:248-262.

BOTRYOSPHAERIA SPP. ON ROSA SP. AND JUNIPERUS SABINA

Robert Hall[1]

This report describes, apparently for the first time in Canada (2,4), the occurrence of Dothiorella states of Botryosphaeria on Rosa and Juniperus.

Isolates from rose

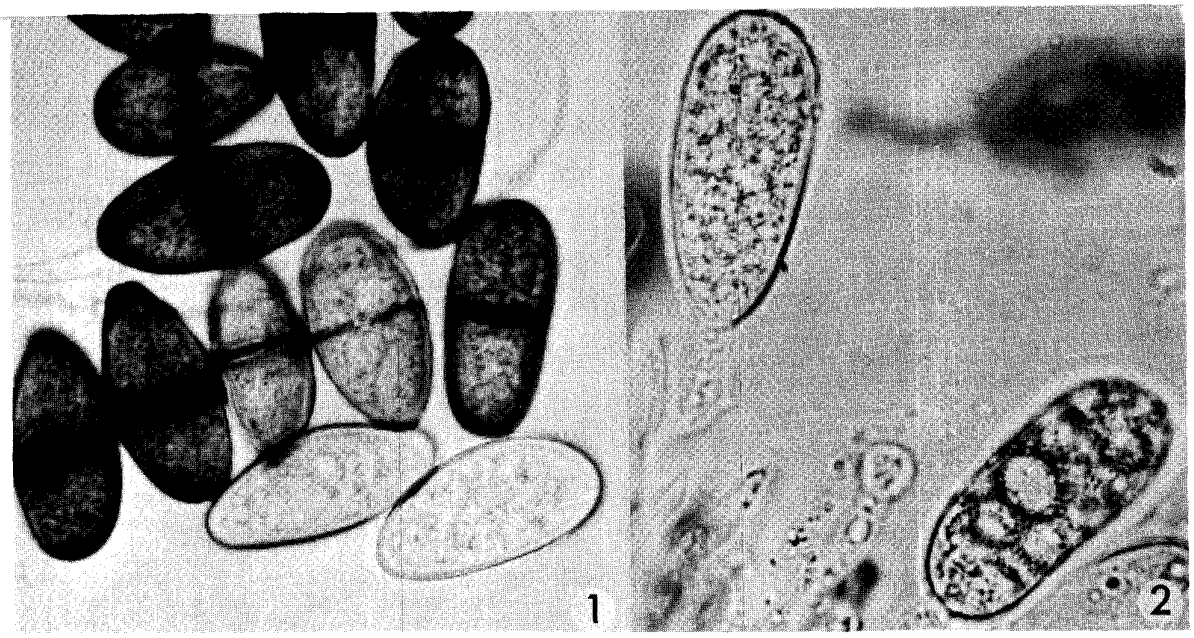
A Dothiorella species matching the description of the Dothiorella state of Botryosphaeria stevensii Shoemaker (4) was isolated from cankers on Rosa sp. cv. 'Pink Sensation' from Burlington, Ontario. The fungus produced Dothiorella pycnidia readily on potato dextrose agar (PDA). As cultures aged the conidia developed a septum and became darkly pigmented (Fig. 1).

Apple fruits wound-inoculated with this fungus were completely rotted within 1 week, and Dothiorella pycnidia were produced within 2 weeks under room conditions. Pathogenicity tests were not conducted on rose. A similar fungus, B. dothidea (Moug. & Fr.) Ces & de Not. (B. ribis Gross. & Dug. var. chromogena Shear, Stev. & Wilcox) causes canker and die-back of roses in Europe (K.A. Pirozynski, personal communication, 1969) and in Alabama, Maryland, Texas, and Virginia (1).

Isolates from juniper

Dothiorella pycnidia were observed on dying 3-year-old bushes of Juniperus sabina L. cv. 'Blue Danube' from St. Catherines, Ontario. The pycnidia were produced separately and abundantly under the bark, especially towards the bases of dying and dead branches. The conidia were usually colorless and aseptate (Fig. 2). Occasionally dark conidia with a single septum were observed. Single spore cultures on PDA incubated under room conditons produced pycnidia containing numerous microconidia and a few much larger conidia of the Dothiorella type. The species of this Botryosphaeria has not been determined. The conidia resemble those of B. stevensii but the rose isolate (B. stevensii?) and juniper isolate are physiologically (growth rate, protein composition, reproduction on PDA) and pathologically distinct.

Stems of 1-year-old and 2-year-old J. sabina cv. 'Blue Danube' plants were wound-inoculated with pycnidia from single-spore cultures. Extensive discoloration of the tissue beneath the bark occurred within 2 weeks. Discoloration and desiccation of the entire plant was apparent 2 months after



Figures 1 and 2. Conidia of *Dothiorella*, X 1250. 1) From rose. 2) From juniper.

[1] Assistant Professor, Botany Department, University of Guelph, Guelph, Ontario.

inoculation. These symptoms matched those observed in the field. Newly-developed Dothiorella pycnidia were present up to several cm from the site of inoculation. This fungus also slowly rotted apple fruits but Dothiorella pycnidia were not produced after 4 weeks.

Juniperus virginiana L. has been reported as a host for Botryosphaeria ribis (Tode ex Fr.) Gross. & Dug. in Alabama, New Jersey, and Virginia (1) and for Sphaeropsis sp. in Lithuania (5). An attempt was therefore made to identify the isolate from J. sabina. Acrylamide gel-electrophoretic protein patterns were prepared from extracts of the rose isolate, the juniper isolate, Botryosphaeria ribis (A.T.C.C. 11232), and B. ribis var. chromogena (A.T.C.C. 11233). B. ribis and B. ribis var. chromogena produced essentially identical patterns. The rose isolate and the juniper isolate produced patterns clearly different from each other and from the B. ribis pattern. These results suggest (3) that the juniper isolate is not B. ribis.

Greenhouse tests indicate the juniper isolate is highly virulent to J. sabina cv. 'Blue Danube'. Since this is the first report of Botryosphaeria on Juniperus in Canada it would be of interest to determine the distribution and pathogenicity of this fungus on junipers and other plant genera, especially those in the family Rosaceae.

Acknowledgment

This work was supported by the Ontario Department of Agriculture and Food. I wish to acknowledge the help of Dr. K.A. Pirozynski, Plant Research Institute, Ottawa, for help in identifying the fungi as Dothiorella states of Botryosphaeria.

Literature cited

1. Anonymous. 1960. Index of plant diseases in the United States. U.S.Dep. Agr. Agr. Handbook 165. 531p.

2. Conners, I.L. 1967. An annotated index of plant diseases in Canada. Can. Dep. Agr. Pub. 1251. 381p.

3. Hall, R. 1969. Molecular approaches to taxonomy of fungi. Bot. Rev. 35:285-304.

4. Shoemaker, R.A. 1964. Conidial states of some Botryosphaeria species on Vitis and Quercus. Can. J. Bot. 4:1297-1301.

5. Zuklys, L.P. 1960. Diseases of ornamental trees newly found in Lithuania. Rev. Appl. Mycol. 39:711.

FOLIAGE DISEASES OF ALFALFA IN NORTHERN SASKATCHEWAN IN 1970[1]

Howard Harding[2]

Abstract

The two primary foliage diseases were black stem (Phoma medicaginis Malbr. & Roum.) and yellow leaf blotch (Leptotrochila medicaginis (Fuckl.) Schüepp). Black stem was the more widespread but its incidence decreased between the June and August surveys while that of yellow leaf blotch increased. Defoliation caused by yellow leaf blotch appeared to be the main source of crop loss. Over 100,000 leaflets and 1600 stems from field samples were examined in the laboratory. There was at least one lesion on 31% of the leaflets and 36% of the stems. There appeared to be little consistent relationship between disease severity, stem height, and leaflet/stem ratio.

Introduction

Although foliage diseases of alfalfa are very common in Saskatchewan, there has been little work done to estimate accurately the losses caused by such diseases. The present survey records disease incidence in a larger number of fields and over a greater area than was previously attempted (1). In addition, the actual leaf area lost to disease has been estimated in field samples by careful examination of individual leaflets in the laboratory. This kind of estimate should provide a base for assessing the meaning of subjective disease ratings done in the field.

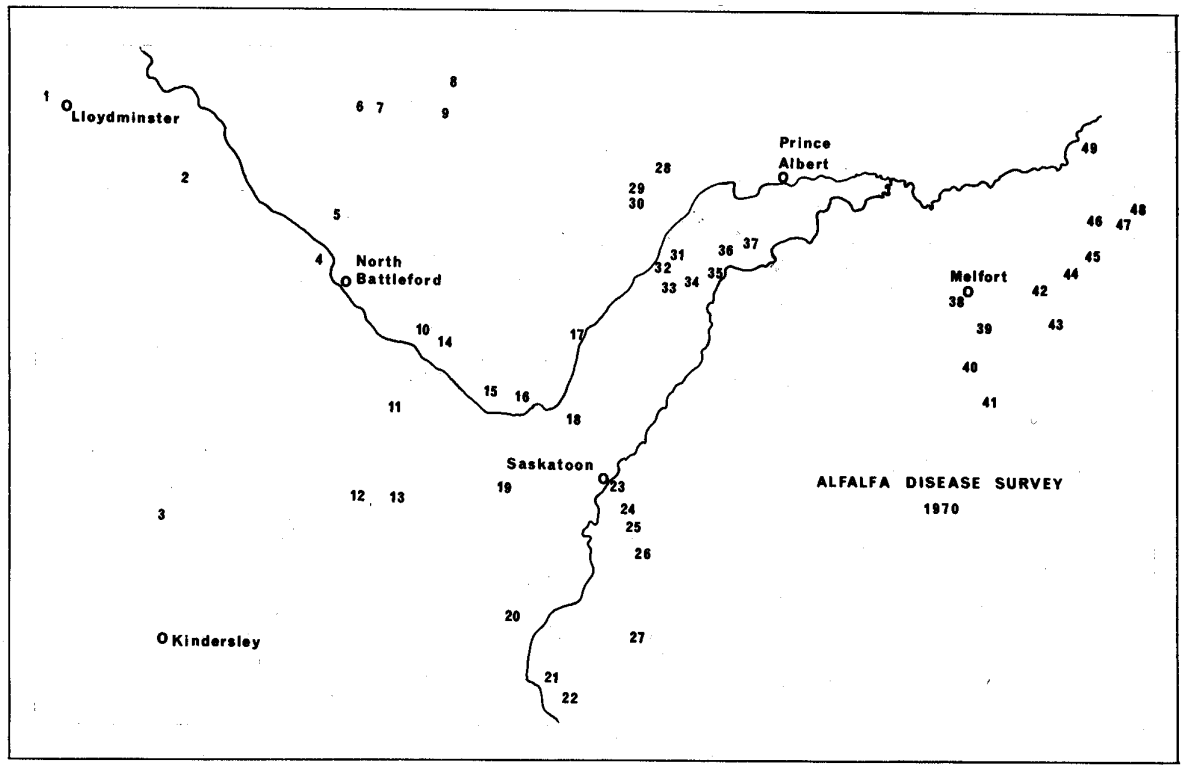


Figure 1. Location of sites visited in 1970 alfalfa disease survey.

[1] Contribution No. 424, Research Station, Canada Department of Agriculture, Saskatoon, Saskatchewan.

[2] Plant Pathologist.

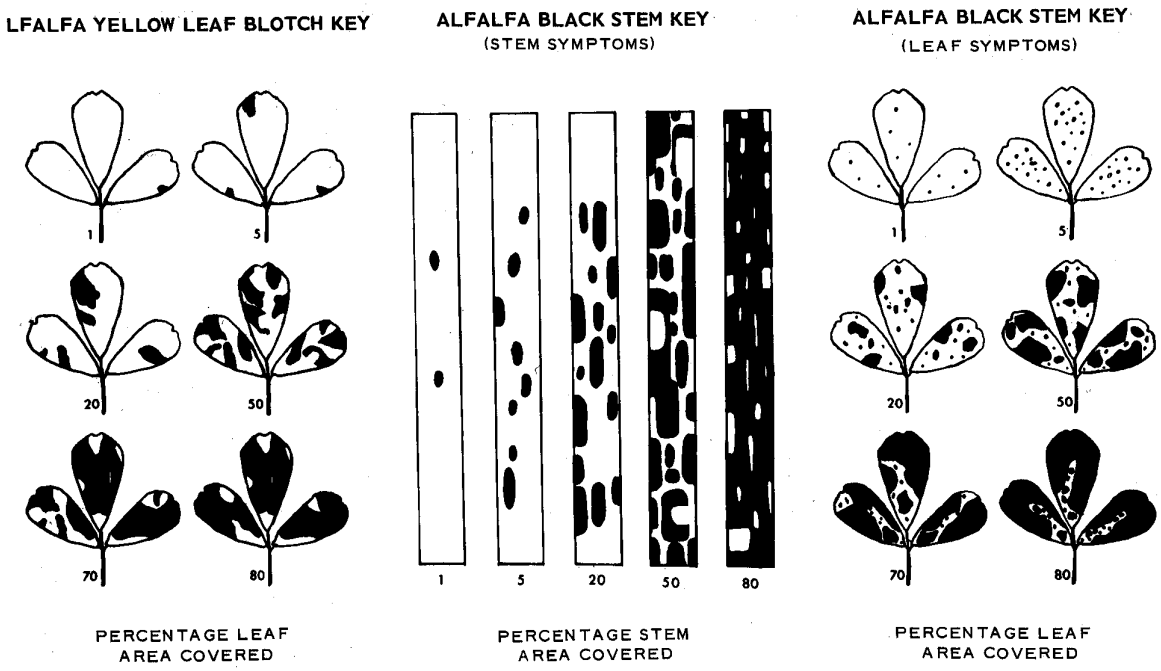


Figure 2. Alfalfa disease rating keys. Shaded areas represent "diseased" tissue whether chlorotic or necrotic.

Materials and methods

Surveys were conducted from June 9 to 18 and August 19 to 24. A total of 64 fields in northern Saskatchewan (Fig. 1) were scored for frequency of plants affected and disease severity. Rating was done as previously described (1) on a scale of 1-10. For laboratory examination, 167 10-stem samples were taken from 4-10 sites in each of 20 fields during the June survey. Each stem and leaflet was examined individually and the area occupied by diseased tissue was estimated using previously prepared disease assessment keys (Fig. 2). Stem ratings were done on the length of stem between the uppermost lesion and the stem base. Also, the < 1% category was included in the 1% category and consequently the amount of stem tissue affected by disease was probably exaggerated. Stem lengths and leaflet/stem ratios were also recorded.

Results

Black stem, caused by Phoma medicaginis Malbr. & Roum. and yellow leaf blotch, caused by Leptotrochila medicaginis (Fuckl.) Schuepp, appeared to be the two primary foliage diseases. Common leaf spot, caused by Pseudopeziza trifolii f. sp. medicaginis-sativae Schmiedeknecht, was found in most fields but in these, with one exception, only a few plants were affected.

Table 1. Alfalfa disease survey June 9-18, 1970

Location no.	Black stem		Yellow leaf blotch	
	% plants affected	Disease rating*	% plants affected	Disease rating*
1	80	1	10	1
2	100	1	20	1
3	40	1	20	1
12	100	2	5	1
14	20	1	0	
16a	30	1	80	3
17	5	1	50	1
21a	100	2	0	
21b	100	2	10	1
21c	100	1	5	1
23	100	1	20	1
24	100	1	20	1
28	20	1	100	1
30	70	1	40	1
34a	70	1	50	2
35	50	1	50	1
37	50	1	50	1
39	100	2	10	1
42	80	1	0	
42	20	3		
46	80	1	20	1
46	20	2		
47	50	1	0	
48a	75	1	0	
48b	100	2	5	1
49	80	2	5	1
49	20	4		

* Where 0 = no disease and 10 = severe disease.

In the June survey (Table 1) black stem was widespread though usually in slight amounts. Yellow leaf blotch was present in most fields but appeared to be causing damage only at location 16a. At location 30, 70% of the plants were slightly affected by downy mildew, caused by Peronospora trifoliorum de Bary.

Table 2. Alfalfa disease survey August 19-24, 1970

Location no.	Black stem		Yellow leaf blotch	
	% plants affected	Disease rating*	% plants affected	Disease rating*
5	10	1	0	
6	70	1	70	2
7	5	1	5	1
8	80	1	100	4
9	50	1	50	1
10	40	1	70	1
10			30	4
11	50	1	70	2
13	20	1	0	
15	10	1	5	1
16a	30	1	60	2
16a			40	6
16b	30	1	0	
18	5	1	40	1
19	70	1	5	1
20	80	1	80	2
20			20	6
21b	100	1	70	2
21b			10	4
22	0		0	
24	80	1	0	
25	80	1	5	1
26	5	1	70	2
27	5	1	90	3
28	70	1	60	4
28			40	4
29	5	1	50	1
29			40	3
30	10	1	60	1
30			40	5
31	5	1	0	
32	70	1	80	2
32			5	5
33	70	1	70	1
34b	5	1	5	1
34c	5	1	0	
36	80	1	40	1
37	80	2	80	4
38a	100	8	80	3
38b	70	1	100	2
40	5	1	5	1
41	5	1	30	2
43	70	1	100	4
44	75	1	80	2
44			5	5
45	0		0	
46	70	1	100	4
47	100	2	70	2
48	50	2	5	1

* Where 0 = no disease and 10 = severe disease.

At the time of the August survey (Table 2) the incidence of black stem had decreased and only in a seed field at location 38 did it appear to be causing real losses. The incidence of yellow leaf blotch was greater and at several locations the disease was causing obvious defoliation. In a newly-established field adjacent to the seed crop at location 38 about 20% of the plants were moderately affected by common leaf spot.

A total of 103,581 leaflets were examined individually in the laboratory and the averages for the 20 fields are shown in Table 3. On an overall average 68.7% of the leaflets were clean, 15.2% had less than 1% of the leaf area occupied by diseased tissue, 11.8% had 1%, 3.4% had 5%, and 0.6% had 20%. Of the 1670 stems 64.5% were clean, 25.7% had an estimated 1% diseased tissue, 7.3% had 5%, and 3.2% had 20%. There appeared to be little consistent relationship between disease severity, stem height, and leaflet/stem ratio (Table 3).

Discussion

Of the two primary diseases, black stem appeared the more widespread; losses caused by it, however, were less than anticipated on the basis of past observations. In particular, the crops grown under contract for alfalfa dehydration plants were remarkably free from black stem. Certainly, traces of the disease were found but probably good crop management reduced black stem losses to a minimum. On the other hand, yellow leaf blotch appeared to be causing some defoliation even in these well-managed fields. In obviously less well-managed stands, the incidence of both diseases was higher.

The high incidence of black stem in seed crops is reflected in the amount of seed-borne inoculum of P. medicaginis. In a survey of seed produced in Saskatchewan in 1969, 85% of the samples carried the organism with levels ranging from less than 1% to 34.4% of the seeds infested (2). It seems reasonable to suggest that with the use of cleaner seed and good crop management the incidence of black stem could be reduced considerably.

On the other hand, most of the diseased tissue on the leaflets examined in the laboratory represented black stem lesions and the fact should not be overlooked that almost a third of the leaflets carried at least one lesion. This estimate is also somewhat low in that a large proportion of the "clean" leaflets were the youngest and, therefore, smallest. Certainly, any more sophisticated estimate of loss should be based on actual leaf area rather than leaflet number.

Although the actual area covered by diseased tissue is relatively small, the presence of just one lesion may decrease the photosynthetic efficiency of a leaflet to a

Table 3. Distribution of individual leaflets into classes based on percentage leaf area occupied by diseased tissue

Location no.	% leaflets in each class							Leaf-stem ratio	Average stem length (cm)
	0	<1%	1%	5%	20%	50%	70%		
3	73	8	10	7	0.8			65	26
12	78	11	11	1				55	24
17	78	6	8	7	0.5			64	24
21a	57	22	16	4	0.6			70	37
21b	46	22	25	6	1.2			58	30
21c	63	18	13	5	1.0			65	39
23	74	18	7	1				62	38
24	58	21	16	5	0.3			62	30
28	76	12	10	2	0.1			90	29
30	79	5	6	3	2.3	2.4	1.7	85	31
34a	62	11	19	8	0.6			68	26
35	80	10	8	1	0.3			76	26
37	65	15	16	3	0.6			62	25
39	67	14	13	4	1.6			37	
42	74	19	5	2				37	
46	79	16	5	1	0.3			47	
47	64	21	13	2				34	
48a	68	22	9	1	0.1			43	
48b	61	20	16	3	0.5			35	
49	71	15	11	2	0.6	0.4		58	

disproportionately large degree. Also, even low levels of infection may substantially affect the nutritional value of the forage. It has recently been shown (4) that the control of P. medicaginis and other leaf-infecting fungi by fairly heavy fungicide applications resulted in an increased carotene content as well as an increase in dry matter. In a different vein, work at North Carolina (3) has shown that coumestrol levels increase in alfalfa leaves following infection by P. medicaginis. It seems, therefore, that further work should be directed towards determining at what level of infection individual leaflets cease to function at maximum efficiency and at what stage the nutritional value of the forage is affected.

Acknowledgment

The assistance of Dr. W.C. James, in preparing the disease assessment keys, is gratefully acknowledged.

Literature cited

1. Harding, H. 1969. Diseases of alfalfa and other forage legumes in Saskatchewan in 1968 and 1969. Can. Plant Dis. Surv. 49:126-127.

2. Harding, H. 1971. Incidence of Phoma medicaginis in alfalfa seed produced in Canada and U.S.A. Can. Plant Dis. Surv. (In press).

3. Sherwood, R.T., A.F. Olah, W.H. Oleson, and E.E. Jones. 1970. Effect of disease and injury on accumulation of a flavonoid estrogen, coumestrol, in alfalfa. Phytopathology 60:684-688.

4. Willis, W.G., D.L. Stuteville, and E.L. Sorensen. 1969. Effects of leaf and stem diseases on yield and quality of alfalfa forage. Crop Sci. 9:637-640.

CRONARTIUM COMANDRAE IN CANADA, ITS DISTRIBUTION AND HOSTS

J.M. Powell[1]

Abstract

Distribution maps and reports of Cronartium comandrae Pk. on the coniferous hosts Pinus banksiana, P. contorta, P. ponderosa, P. sylvestris, and the alternate hosts Comandra umbellata ssp. umbellata, C. u. ssp. pallida, and Geocaulon lividum are presented for Canada. C. comandrae occurs in all provinces and territories except Newfoundland, Nova Scotia, and Prince Edward Island, although native or introduced hosts do occur in these areas. New records of C. comandrae on P. contorta in Saskatchewan, on C. umbellata ssp. umbellata in Saskatchewan and Manitoba, and on G. lividum in Ontario and Saskatchewan, are reported. There is doubt about an earlier record of the rust on C. umbellata from the Northwest Territories.

Introduction

Comandra blister rust caused by Cronartium comandrae Pk. has been reported on one introduced and eleven native hard pine species in North America (8,15), ranging from New Brunswick to the Yukon and southward to Tennessee, Alabama, Mississippi, New Mexico, and California. It has not been found in Alaska or Mexico although susceptible pines occur there. The uredial and telial states of the rust have been recorded over a similar range on the alternate hosts, Comandra and Geocaulon of the family Santalaceae. A generalized distribution map for C. comandrae on Pinus spp. and Comandra spp. in North America was recently published by Krebill (13). Information on the hosts and distribution of C. comandrae in Canada is scattered. To date, the rust has been reported on three native and one introduced hard pine species, and on both alternate host genera in Canada. This paper attempts to bring together the information and to report recent collections which have extended the known distribution of the rust in Canada.

Materials and methods

Arthur's Manual of the rusts in the United States and Canada (2) has been used as the base reference for information on distribution and hosts in Canada. More recent references on distribution have been gathered from the literature.

All the Cronartium comandrae materials have been seen or information on them obtained from the following herbaria (Herbarium codes follow Lanjouw and Stafleu [14]): ALTA, BPI, CFB, DAOM, DAVFP, FFB, MFB, MONT, NY, PUR, QFB, QMP, SASK, UBC, WIN, WINF(M), WSP, MacDonald College, (Ste. Anne de Bellevue, Que.,) and many of the regional laboratories of the United States Forest Service, some of which contain Canadian material. Information from the Forest Insect and Disease Surveys, Canadian Forestry Service, have also been used extensively for compilation of the distribution maps, especially in the Prairie and British Columbia Regions, where such surveys have been quite thorough. Intensive surveys were also carried out by the author in Alberta and southwestern Saskatchewan. The alternate host genus Comandra has recently been modified by Piehl (17) to include four subspecies of one species (C. umbellata) instead of five species, while he confirms Fernald's (11) study of separating Geocaulon lividum from the genus Comandra. I attempted to follow Piehl's arrangement of the sub-species by using his distribution map, but this was not totally satisfactory as he indicates large intermediate zones between some of the subspecies, e.g. a wide band across the Prairies between ssp. pallida and ssp. umbellata.

Hosts and distribution

REPORTS OF THE RUST ON ITS VARIOUS HOSTS IN CANADA

Pinus banksiana Lamb.

Arthur (2) reported that C. comandrae occurred on this host in Alberta and Saskatchewan. It has also been reported from New Brunswick (10), Quebec (18), Ontario (7), Manitoba (22), and the Northwest Territories (4).

Pinus contorta Dougl.

Reported in Alberta (2), British Columbia (25) and the Yukon (16).

Pinus ponderosa Laws.

Arthur (2) reported it from British Columbia.

[1] Research Scientist, Canadian Forestry Service, Department of Fisheries and Forestry, Edmonton, Alberta.

Pinus sylvestris L.

The rust has been reported on this introduced pine from Manitoba (24), Saskatchewan (12) and Alberta (21).

Comandra umbellata (L.) Nutt. ssp. umbellata Piehl (C. umbellata; C. richardsiana).

Arthur (2) reported that the rust occurred on this host in Ontario and Quebec.

Comandra umbellata (L.) Nutt. ssp. pallida (A.DC.) Piehl (C. pallida).

Arthur (2) reported it from Alberta, British Columbia, Saskatchewan, and the Northwest Territories. Bisby (5) reported its occurrence in Manitoba, and Conners (7) in Ontario.

Geocaulon lividum (Richardson) Fernald.

Arthur (2) gave the distribution on this host (cited as Comandra livida) as Manitoba and Quebec. Ziller and Molnar (26) reported its occurrence in British Columbia, Molnar (16) in the Yukon, and Baranyay et al. (4) in Alberta. Baranyay and Bourchier (3) collected it in 1962 in the Northwest Territories, although the host was reported as Comandra.

DISTRIBUTION OF THE RUST IN CANADA, INCLUDING NEW RECORDS

Figures 1 and 3 show the distribution of C. comandrae on Pinus spp., and Figures 2 and 4 its distribution on Comandra and Geocaulon in western and eastern Canada. C. comandrae would appear to be more prevalent in western Canada, where the distribution of the hosts is more continuous. Surveys have also been more extensive in western Canada, especially in southern Alberta where the author has been studying various aspects of the aerobiology of the rust since 1964 (20). Generally, the northern distribution line for Canada on Krebill's map (13) does not extend far enough north, especially in the Yukon and Northwest Territories, and in Manitoba and Quebec. The collections from Great Whale River on G. lividum have been incorrectly placed in Ontario instead of Quebec. Sutton (23) gives a generalized distribution for Saskatchewan from reports for the years 1965-67. Figure 1 of this paper indicates that the distribution on pine is more continuous across Saskatchewan, and that the rust has been recorded farther north on this host.

The rust can be found in most young and mature stands of Pinus contorta and P. banksiana in western Canada, often reaching local epidemic proportions when the pine is close to stands of Comandra umbellata ssp. pallida in southern areas or Geocaulon lividum in northern areas. In western Canada, its distribution extends to the pine limits in the Yukon and Northwest Territories, but should be found farther north in Saskatchewan and Manitoba than present collections indicate (Fig. 1). The rust extends across the Prairie grassland areas on C. umbellata ssp. pallida through infections of the repetitive uredial state. Infections were often found more than one hundred miles from the nearest pine source (Figs. 1 and 2). In eastern Canada, intensive surveys seem to be lacking and this probably accounts for much of the non-continuous distribution. Local epidemics have been reported and in one case a plantation suffered considerable damage (9). Only one collection of the rust has been made in the Maritimes, despite considerable examination of pines and searching for the alternate host (G.A. Van Sickle, 1969, personal communication). There are no records from Newfoundland or along the north shore of the Gulf of St. Lawrence, east of latitude 65° . Natural stands of the pine host are absent in this area (6), but Comandra does occur (17) and susceptible pines have been introduced.

New provincial distribution records for the rust include two specimens (CFB 8406,8407) collected in 1968 on P. contorta in the Cypress Hills, Saskatchewan. Two collections on C. richardsiana from Manitoba and Saskatchewan are in UBC (82625 and 86877), and two others in WINF(M) (1746,3712) on C. richardsiana or C. umbellata are probably on true C. umbellata ssp. umbellata or an intermediate between ssp. umbellata and ssp. pallida A collection made in 1937 by D.V. Baxter (DAOM 5558 and PUR 48514) from Great Slave Lake, Northwest Territories, and labelled Comandra sp. proved to be G. lividum, which is the earliest collection on this host from that area. An unreported specimen on G. lividum from Ontario is in DAOM (76368), and a specimen was collected from Deception Lake, Saskatchewan, in 1969 (WINF(M) 12039). This host has a northern distribution and only just enters the United States where infected specimens have been collected only in Wisconsin (2), Idaho (BPI, NY, and Pacific Northwest Forest & Ra.Exp.Sta., Portland, Oregon), and Washinton (BPI, DAOM).

There is doubt about location of the collection of Comandra upon which Arthur (2) based his Northwest Territories record. J.A. Parmelee (personal communication, 1966) believed this record was based on the DAOM specimen, No. 1861, from near Martin Cabin, Slave Lake, Alberta, collected in 1929, and that Arthur interpreted 'Slave Lake' as Great Slave Lake, Northwest Territories, instead of Lesser Slave Lake, Alberta. However, Arthur (1) included the Northwest Territories in his distribution list prior to the collection of the above DAOM specimen. There is no record of Comandra from tne Northwest Territories or the Yukon in the DAO or CAN herbaria, or more regional herbaria (ALTA, CAFB, UAC, UBC), and it has not been collected north of about 59° N latitude in Alberta. Porsild and Cody (19), in their checklist of vascular plants in continental Northwest Territories,

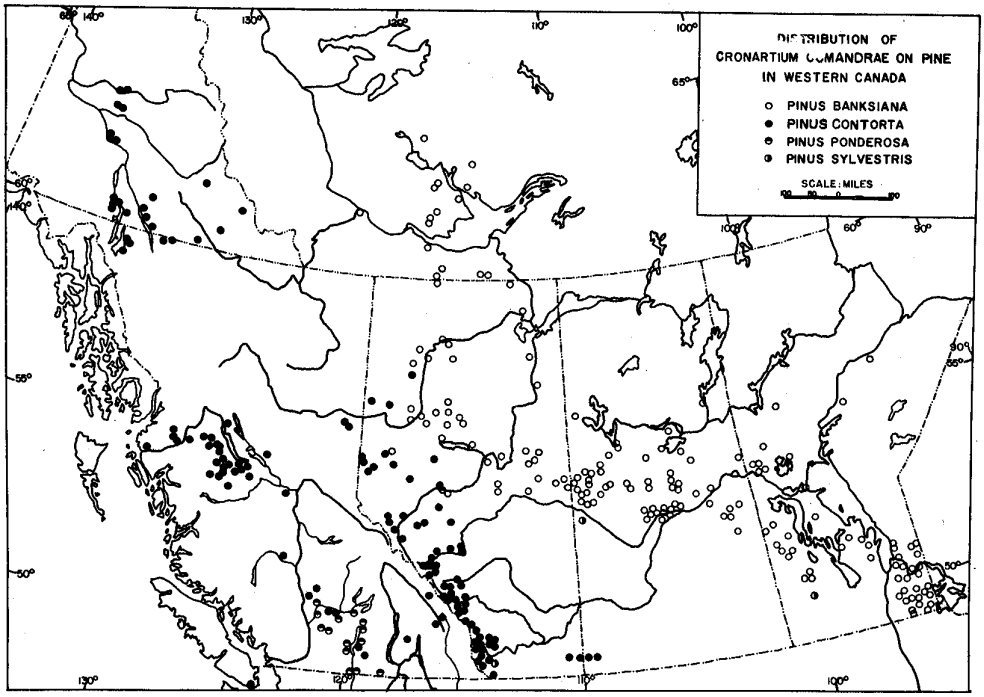


Figure 1. Distribution of *Cronartium comandrae* on *Pinus* spp. in Western Canada.

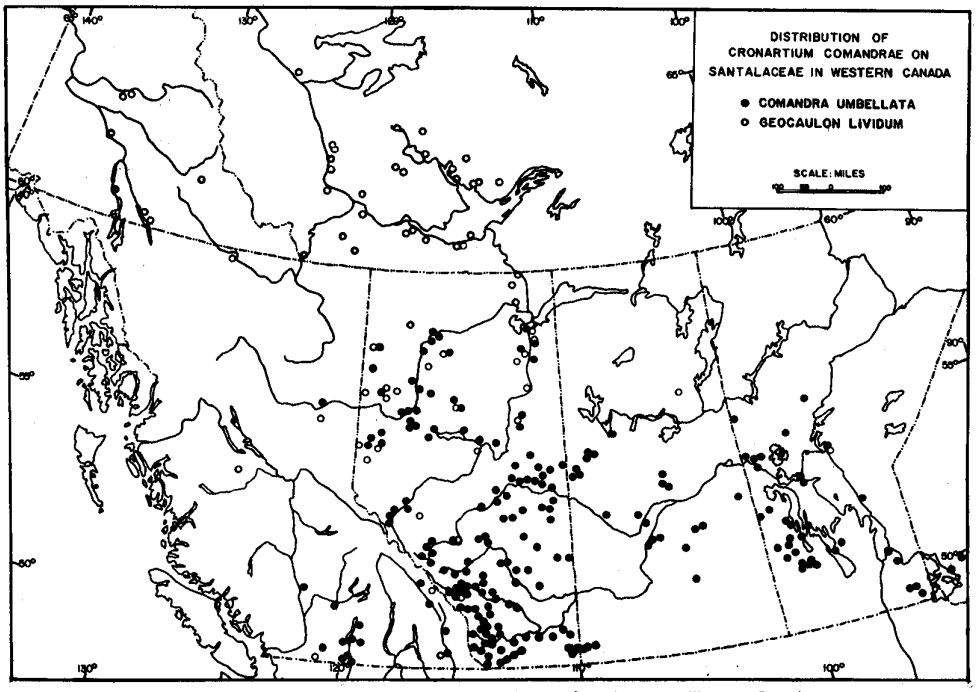


Figure 2. Distribution of *Cronartium comandrae* on Santalaceae in Western Canada.

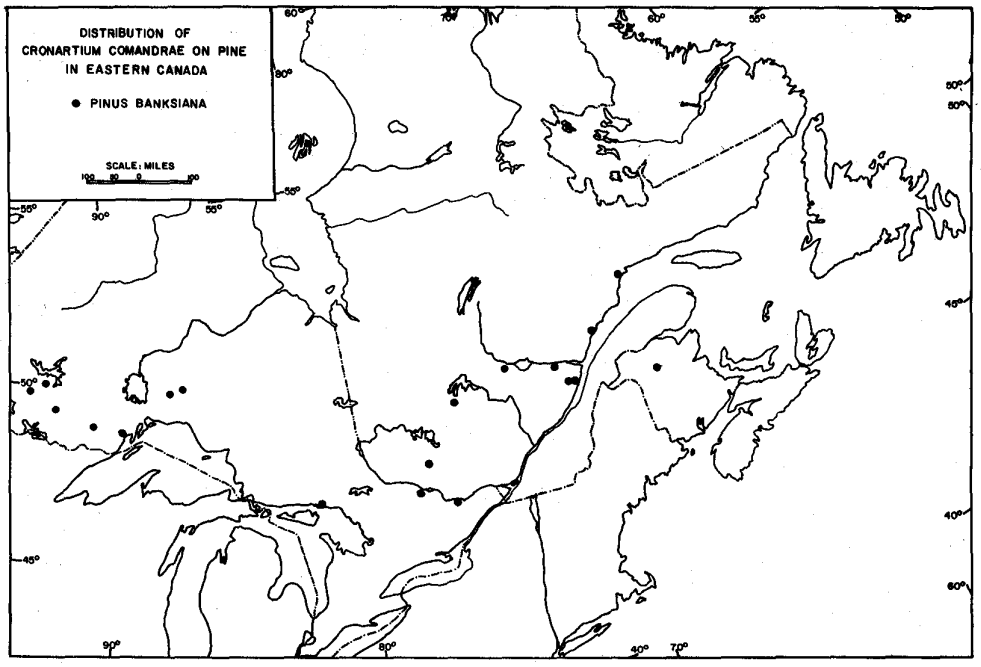


Figure 3. Distribution of *Cronartium comandrae* on pine in Eastern Canada.

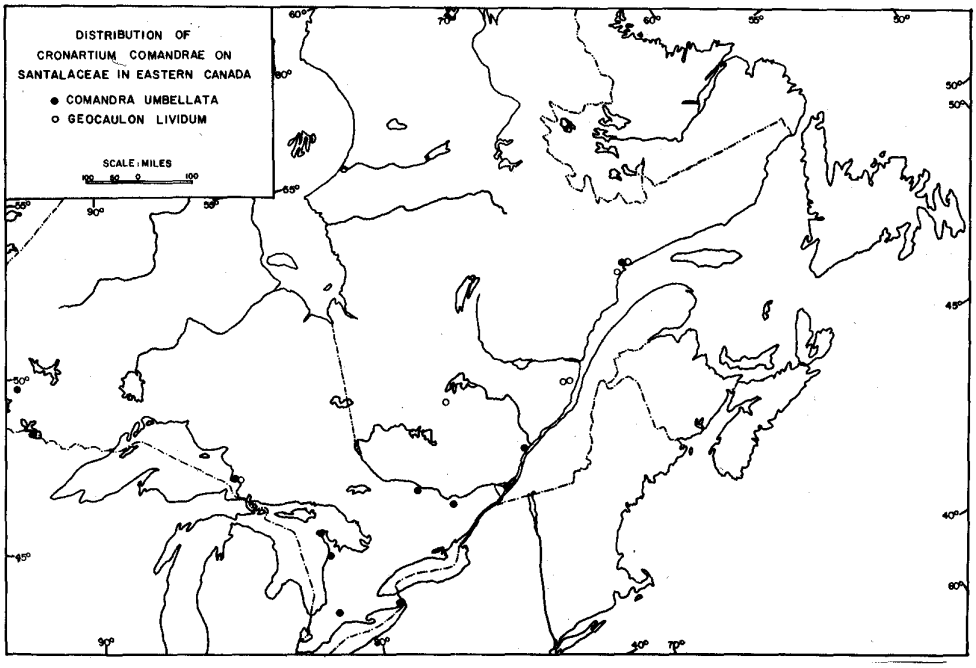


Figure 4. Distribution of *Cronartium comandrae* on Santalaceae in Eastern Canada.

indicated that C. pallida was expected to occur, but at present there is no record from the area. Piehl (17) also does not indicate its presence north of 60°N latitude.

Literature cited

1. Arthur, J.C. 1927. (Uredinales). Additions and corrections (concluded), p. 797-848. In North American Flora 7, Part 12. New York Botanical Garden.

2. Arthur, J.C. 1934. Manual of the rusts in the United States and Canada. Purdue Res. Found., Lafayette, Indiana. 438 p.

3. Baranyay, J.A., and R.J. Bourchier. 1963. Province of Alberta. Forest disease conditions, p. 99-102. In Annual report of the Forest Insect and Disease Survey, 1962. Forest Entomol. and Pathol. Branch, Can. Dep. Forest. 134 p.

4. Baranyay, J.A., R.J. Bourchier, and G.R. Stevenson. 1962. Province of Alberta. Forest disease survey, p. 102-106. In Annual report of the Forest Insect and Disease Survey, 1961. Forest Entomol. and Pathol. Branch, Can. Dep. Forest. 136 p.

5. Bisby, G.R. 1938. The fungi of Manitoba and Saskatchewan. Nat. Res. Counc. Can., Ottawa. 189 p.

6. Cayford, J.H., Z. Chrosciewicz, and H.P. Sims. 1967. A review of silvicultural research in jack pine. Forest. Branch, Can. Dep. Forest. Rural Develop., Publ. 1173. 255 p.

7. Conners, I.L. 1934. Thirteenth annual report of the Canadian Plant Disease Survey, 1933. Can. Dep. Agr., 128 p.

8. Cordell, C.E., H.W. Applegate, and P.J. Huffman. 1969. Pond pine - a new host for comandra blister rust in eastern Tennessee. Plant Dis. Rep. 53:694.

9. Dance, B.W., and D.F. Lynn. 1965. Ontario forest disease conditions, p. 68-77. In Annual report of the Forest Insect and Disease Survey, 1964. Can. Dep. Forest. 141 p.

10. Davidson, A.G., and W.R. Newell. 1957. Atlantic Provinces. Forest disease survey, p. 22-29. In Annual report of the Forest Insect and Disease Survey for 1956. Forest Biol. Div., Can. Dep. Agr. 95 p.

11. Fernald, M.L. 1928. Geocaulon, a new genus of the Santalaceae. Rhodora 30:21-24.

12. Ives, W.G.H., N.R. Brandt, and B.C. Sutton. 1968. Manitoba-Saskatchewan Region, p. 76-96. In Annual report of the Forest Insect and Disease Survey, 1967. Forest. Branch, Can. Dep. Forest. Rural Develop. 143 p.

13. Krebill, R.G. 1968. Cronartium comandrae Peck. Commonwealth Mycological Inst., Distribution maps of plant diseases, Map 444. 1st ed. 2 p.

14. Lanjouw, J., and F.A. Stafleu. 1964. Index Herbariorum. Part I. The herbaria of the world. 5th ed. Int. Bur. Plant Taxonomy and Nomenclature. Utrecht, Netherlands. 251 p.

15. Mielke, J.L., R.G. Krebill, and H.R. Powers, Jr. 1968. Comandra blister rust of hard pines. U.S. Dep. Agr., Forest Serv., Forest Pest Leaflet 62 (Rev. August 1968). 8 p.

16. Molnar, A.C. 1960. Province of British Columbia. Forest disease survey, p. 105-110. In Annual report of the Forest Insect and Disease Survey 1959. Forest Biol. Div., Can. Dep. Agr. 121 p.

17. Piehl, M.A. 1965. The natural history and taxonomy of Comandra (Santalaceae). Mem. Torrey Bot. Club 22, no. 1. 97 p.

18. Pomerleau, R. 1942. Liste annotee des maladies parasitaires des arbres observees dans le Quebec. Quebec: Serv. Forest. Min. des Terres et Forets. 39 p. (mimeo).

19. Porsild, A.E., and W.J. Cody. 1968. Checklist of the vascular plants of continental Northwest Territories Canada. Plant Res. Inst., Can. Dep. Agr., Ottawa. 102 p.

20. Powell, J.M. 1969. The aerobiology of the aecial state of the comandra blister rust, Cronartium comandrae Peck, in Alberta. Ph.D. Thesis. Univ. British Columbia, 362 p.

21. Powell, J.M., and W. Morf. 1965. The occurrence of Tuberculina maxima Rost. on Cronartium rust infected trees in Alberta. Forest Entomol. Pathol. Branch, Can. Dep. Forest., Bi-monthly Prog. Rep. 21(1):3.

22. Riley, C.G., W.B. Denyer, and R.D. Whitney, 1952. Prairie Provinces, p. 140-146. In Annual report of the Forest Insect and Disease Survey, 1951. Div. Forest Biol., Can. Dep. Agr. 154 p.

23. Sutton, B.C. 1969. Forest pathogen infections 1965-67, p. 103-104. In J.H. Richards, and K.I. Fung [ed.] Atlas of Saskatchewan. Univ. Saskatchewan, Saskatoon. 236 p.

24. Thomas, R.W. 1953. Forest disease survey. Province of Manitoba and

Saskatchewan, p. 92-99. In Annual report of the Forest Insect and Disease Survey, 1952. Div. Forest Biol., Can. Dep. Agr. 154 p.

25. Ziller, W.G. 1953. Annotated check list of the tree rusts in British Columbia. Forest Biol. Div., Can. Dep. Agr., Victoria, B.C. 117 p. (mimeo).

26. Ziller, W.G., and A.C. Molnar. 1953. Forest disease survey, p. 142-154. In Annual report of the Forest Insect and Disease Survey, 1952. Div. Forest Biol., Can. Dep. Agr. 154 p.

DISTRIBUTION OF A NEWLY REPORTED LEAFHOPPER-TRANSMITTED CLOVER DISEASE IN EASTERN ONTARIO

L.N.Chiykowski[1] *and A.T.Bolton*[2]

Introduction

A clover disease was reported recently in the Ottawa, Ontario, area and was shown to be transmitted by the leafhopper Aphrodes bicincta (Schrank) (1). At that time it was reported that clover plants could not be found in the field with symptoms typical of those observed in the greenhouse. Observations in the field in May 1969, during the early growing season, revealed numerous clover plants with symptoms similar to those observed in the greenhouse. The disease was most apparent on red clover and on white clover as a very obvious chlorosis or yellowing of leaf margins of newer leaves accompanied by moderate to severe stunting (Fig. 2).

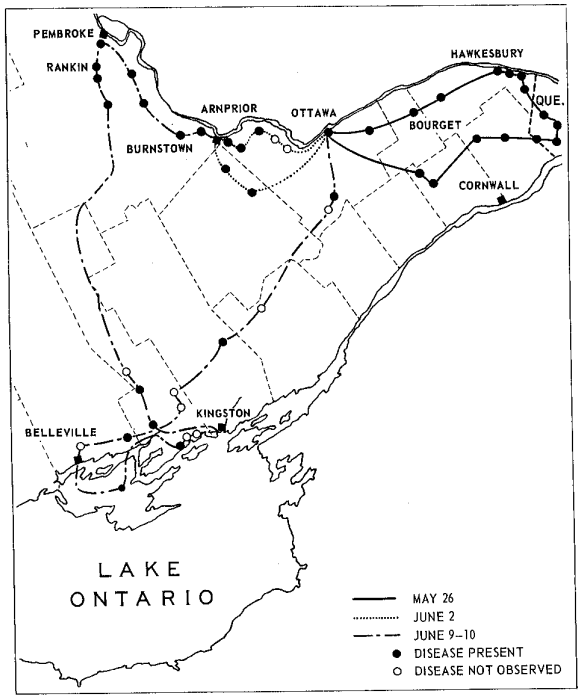


Figure 1. Distribution of a newly reported leafhopper-transmitted disease of clover in eastern Ontario, May – June 1970.

Although symptoms of the disease were readily observed in 1969 in all clover fields at the Central Experimental Farm at Ottawa, no information was available on its distribution elsewhere. Accordingly, surveys were made in May and June 1970 to determine the distribution of this newly reported clover disease in eastern Ontario.

Methods

Surveying was done in fields of red (Trifolium pratense L.), white (Trifolium repens L.), and alsike (Trifolium hybridum L.) clovers, either in pure stands or in mixtures with one another or with alfalfa (Medicago sativa L.) and various grasses. Preference was given to fields containing red clover because the upright growth habit of this species made infected plants easier to see. Observations were made by walking toward the centre of the field as well as along the margin. Volunteer clovers growing along ditches and roadway were examined also.

Results and discussion

The dates on which survey trips were made, the areas covered, and the results obtained are given in Fig. 1.

East of Ottawa, the disease was observed in all 16 fields surveyed. In most fields the incidence of affected plants varied from trace (less than 1%) to light (up to 5%) and the disease was often more prevalent along the margin of the field than toward the centre. Near Bourget, Ont., however, infection reached 50% in some areas of a field in which red clover had been grown for several years. Symptoms were observed a number of times on volunteer clovers growing along ditches and roadways. The disease was found also in red clover in three fields examined in Quebec, near the Ontario border (Fig. 1).

From Ottawa westward to Pembroke, the disease was observed in 18 of 20 fields surveyed. Only trace amounts were found in 16 of the fields, but approximately 20% and 50% of the plants were infected in two fields near Burnstown and Rankin, respectively.

Southwest of Ottawa, infected plants were observed in 9 of 17 fields. The advanced stage of growth in the more southerly areas

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

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

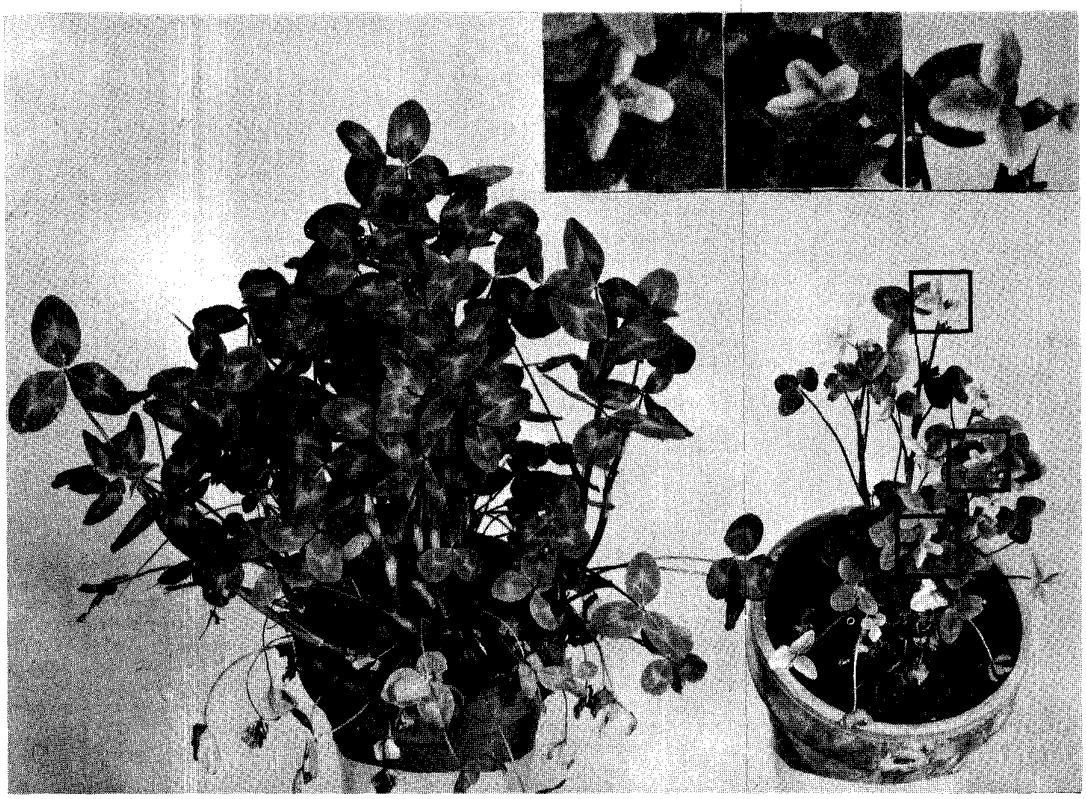


Figure 2. Symptoms of a newly reported leafhopper-transmitted disease on red clover naturally infected in the field. Left, healthy plant, Right, infected plant showing stunting and new leaves with chlorotic margins.

made observations more difficult and may have been responsible for the apparently lower disease incidence.

This survey has shown that the newly reported leafhopper-transmitted disease is present in most areas of eastern Ontario where clover is grown, having been observed in 43 of 53 fields surveyed. It is too early to assess the economic importance of this disease. The low level of infection observed in most fields suggests that it is of minor importance. However, the wide distribution, high incidence in some older stands of clover, and the stunting of infected plants indicate that the disease may have potential importance.

Literature cited

1. Chiykowski, L.N. 1969. A leafhopper transmitted clover disease in the Ottawa area. Can. Plant Dis. Surv. 49:16-19.

TURF DISEASES IN THE LOWER MAINLAND OF BRITISH COLUMBIA[1]

H. Vaartnou[2]

Abstract

In the Lower Mainland of British Columbia, pink snow mold caused by Fusarium nivale was the most frequently observed turf disease, and fusarium patch caused by Fusarium spp. the most destructive turf disease observed in 1964-66. Brown patch caused by Rhizoctonia solani can also be very serious. Helminthosporium diseases were destructive during warm, moist conditions. Red thread, caused by Corticium fuciforme, occurred on fescue turf in late summer, and rust and powdery mildew were found on bluegrass mainly in late summer and early fall; both diseases reduced the beauty of turf. Dollar spot caused by Sclerotinia homoeocarpa and fairy ring caused by Marasmius spp. occurred occasionally, ruining the appearance of turf.

In this area turf diseases occur mainly between September and April. Bentgrasses are the most susceptible, followed by bluegrasses and fescues. Differences in susceptibility among species and clones are large.

Introduction

Gould (2) stated that in western Washington the most serious turf diseases are caused by Fusarium nivale, Corticium fuciforme, Ophiobolus graminis, and Marasmius oreades. He also suspected that with further research additional pathogens, including Helminthosporium species, would be found. Meiners (3) reported that in the Pacific Northwest, Fusarium nivale first appears on turf in October and November, coinciding with cool, wet weather and continues to develop under snow cover or wet conditions until February or March. Dahl (1) found that Fusarium nivale was destructive on the more vigorous plants.

A survey of turf diseases occurring in the Lower Mainland of British Columbia was conducted during the winter of 1964-65 and the spring of 1966 to determine which diseases were most destructive to established turf, when these diseases were most destructive, and if resistance to them existed among different grass species and clones.

Materials and methods

A survey of turf diseases was conducted at 10 locations in the Lower Mainland, extending from the University of British Columbia campus at Vancouver to Hope, B.C. Visual records and samples for culturing were taken at each location. The following grass species and cultivars were inspected: colonial bent (Agrostis tenuis Sibth.), five clones; colonial bent, mixed; 'Highland' bent (Agrostis sp.); creeping bent (Agrostis sp. cv. 'Congressional'); Kentucky bluegrass (Poa pratensis L.), mixed; Kentucky bluegrass cv. 'Merion'; annual bluegrass (Poa annua L.); creeping red fescue (Festuca rubra L.), mixed; and creeping red fescue cv. 'Pennlawn'.

Turf samples were taken during three seasons, fall (August to December) 1964; winter (January to March) 1965; and spring (April to June) 1966.

Visual observations and photographs were made of each disease on the different species and clones at the various locations. Samples, composed of 5-cm plugs of turf, were taken from the juncture of healthy and diseased turf and were used for isolation of fungi in the laboratory.

Four sections of diseased leaf tissue were taken from each sample. The sections were surface sterilized in a 15% (v/v) Chlorox solution for 5 minutes and placed on Difco Bacto-agar in petri dishes, which were then set aside for 2-3 days in light to allow hyphae of the fungus to develop on the agar. When the hyphae had developed sufficiently, blocks of agar, each containing a hyphal tip, were cut from the culture and placed on potato-dextrose agar (PDA) slants.

To induce sporulation, fungus isolates were grown at room temperature for periods of up to 70 days on Bacto-agar containing approximately six grains of Quaker oats per petri dish. Several cultures on PDA slants were also placed outside for 14 days, where

---

[1] Contribution No. NRG-70-17, Research Station, Canada Department of Agriculture, Beaverlodge, Alberta.

[2] Forage Ecologist, Research Station, Canada Department of Agriculture, Beaverlodge, Alberta. Present address: Plant Industry Division, Alberta Department of Agriculture, Edmonton, Alberta.

they were protected from rain and exposed to fluctuating temperature ranging from 23 to 50 F (-5 to 10C).

Spores produced in culture were examined microscopically after staining with cotton blue-lactophenol or with a combination stain of phloxine and congo red in a 3% solution of KOH. The latter combination was used to observe mycelial cell walls.

Results

It will be noted from Table 1 that during December 1964 and January 1965 the total snowfall was much above average. Snow covered the turf for a period of nearly 2 months in the Vancouver area. This abnormally severe winter followed an unusually wet summer.

From the samples collected, nine major pathogenic fungi were isolated (Table 2): Fusarium nivale (F.) Ces., Fusarium spp., Helminthosporium spp., Rhizoctonia solani Kuhn, Corticium fuciforme (Berk.) Wakef., Puccinia spp., Erysiphe graminis DC. ex Merat, Sclerotinia homoeocarpa F.T. Bennett, and Marasmius spp.

PINK SNOW MOLD (Fusarium nivale) was found in all turf species from September to April. The fungus killed the leaves, resulting in the formation of large unsightly pinkish-grayish-white irregular patches that ruined the appearance of the turf. 'Highland' bentgrass was particularly susceptible to this disease (Fig. 1). However, the plants usually recovered within a month after growth started in spring.

Morphological differences, especially in spore size, occurred between forms attacking the grass in fall and in winter.

Table 1. Climatological data, Vancouver, B.C.

Date	Avg temperature (°F) Mean	Low	High	Precipitation (inches) Total	Snow
1964					
August	60.5	54.2	66.7	1.45	0
September	55.2	49.5	60.9	5.57	0
October	50.7	44.6	56.7	2.34	0
November	41.5	37.4	45.6	5.71	2.0
December	34.4	30.1	38.6	6.19	43.7
1965					
January	36.7	33.1	40.2	7.07	12.8
February	39.7	35.9	43.5	7.42	0.6
March	41.1	34.5	47.6	2.24	2.0
1966					
April	47.2	40.6	53.8	1.10	0
May	52.2	45.8	58.6	2.24	0
June	56.8	51.1	62.4	1.79	0

FUSARIUM PATCH (Fusarium spp.) occurred on bentgrass lawns, mainly in February, and was found most frequently on creeping bentgrass. The fungus killed the plants, leaving dead patches 2 to 30 cm in diameter (Fig. 2) in lawns of creeping bent, 'Highland' bent, and annual bluegrass.

Table 2. Frequency of occurrence of turf disease fungi in the Lower Mainland of British Columbia during three periods, 1964-66

Turf grass	No. of locations	Fusarium nivale F*	W*	S*	Fusarium spp. F	W	S	Helmintho-sporium spp. F	W	S	Rhizocto-nia solani F	W	S	Corticium fuciforme F	W	S	Puccinia spp. F	W	S	Erysiphe graminis F	W	S
Colonial bent																						
Mixed	10	10	10	6	1	4		10	10	9				5			1			2		
Clone 1	4	4	4	2		1		2	4	2		1		2						1		
Clone 2	4	4	4	3		1		1	4	2				3			1					
Clone 3	4	4	4	2				2	4	3				1								
Clone 4	4	4	4	1				1	2	1				1			2					
Clone 5	4	4	4	1		1		2	3	1				3						1		
'Highland' bent	10	10	10	8	1	4	1	8	10	7				8			1			3		
'Congressional' bent†	5	4	4	2	2	5	1	3	5	2				1								
'Merion' Kentucky bluegrass	5	2	3	1				2	5					2			5			5	1	2
Kentucky bluegrass	5	2	4	1				5	5	2				3			5			5	2	2
Annual bluegrass†	10	8	10	5	1	3		6	10	6	1	1		6			7			6	1	1
'Pennlawn' creeping red fescue	5	3	4	1				1	3					5								
Creeping red fescue	8	7	7	5				1	6					8								
Total	78	66	72	38	5	19	2	44	71	35	1	2		48			22			23	4	5

* F = fall 1964, W = winter 1964-65, S = spring 1966.

† Also, Sclerotinia homeocarpa was found in one sample of 'Congressional' bent in fall 1964; and Marasmius spp. in a sample of annual bluegrass in fall 1964, and in winter 1964-65.

Figure 1. Injury to turf caused by *Fusarium nivale*. Left, 'Congressional' bentgrass. Right, 'Highland' bentgrass.

Figure 2. Injury caused by *Fusarium* spp. to a lawn of 'Congressional' bentgrass.

Colonial bent was able to recover partially. The fungus was not isolated from Kentucky bluegrass or creeping red fescue turf.

MELTING OUT (Helminthosporium spp.) At least two Helminthosporium spp. attacked turf in the Vancouver area. In September and October, when the weather was still warm but reasonably moist, helminthosporium diseases caused the lawns to be thinned out by killing some of the tillers. Bentgrasses in shady locations were most affected.

A strain causing leaf spot was isolated from all species of turf grasses but was particularly predominant on some of the bentgrass clones.

BROWN PATCH (Rhizoctonia solani) (Fig. 3) occurred in irregular grayish-brown rings up to 50 cm in diameter. The fungus killed the leaves but not the roots, and the turf recovered within a month. This fungus was isolated from samples of annual bluegrass in October and February and from colonial bentgrass in February in turf samples taken from well fertilized golf greens.

RED THREAD (Corticium fuciforme) was most commonly found in early September in fescue lawns, but it also appeared in lawns consisting mainly of annual and Kentucky bluegrasses and bentgrasses. Cloudy and moist weather in early fall favored the spread of the causal fungus.

RUST (Puccinia spp.) mainly attacked Kentucky bluegrass lawns in early fall and generally those in less fertile locations. It did not kill the grass and nearly disappeared later in the fall.

POWDERY MILDEW (Erysiphe graminis) occurred on bluegrasses in early fall. White mycelial patches formed on the surface of leaf blades and sheaths. The infected leaves finally turned yellowish brown, but the fungus did not kill the plants.

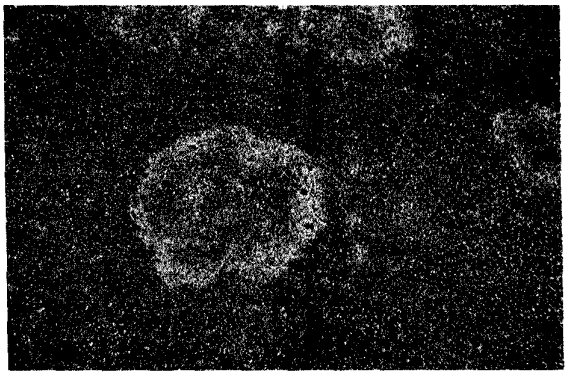


Figure 3. Injury caused by *Rhizoctonia solani* to a golf green of annual bluegrass.

DOLLAR SPOT (Sclerotinia homoeocarpa) occurred on creeping bentgrass in August. Regular dark-brown spots, 3-5 cm in diameter, turning later to a light, bleached color, appeared on the turf. White mycelium, which was very noticeable in early morning, covered the leaves.

FAIRY RING (Marasmius spp.) was identified from samples taken from an annual bluegrass lawn in fall and winter. Dark green rings 20-100 cm in diameter appeared, and grass growing within the rings displayed a lighter color than the rest of the lawn.

Discussion

Pink snow mold was the most frequent and one of the most destructive turf diseases in the Lower Fraser Valley. It was most active under snow cover and after the snow melted during cold, wet weather. It affected all turf grasses examined, but there were large differences in susceptibility among species and clones. Strains of the causal fungus damaging turf in the fall may be different from strains active during winter months.

Fusarium patch was the most destructive disease on bent grass and annual bluegrass lawns in winter. In addition to killing the

leaves, it also killed the roots of the plants and left round dead patches in the middle of the lawn.

Helminthosporium spp. ruined the quality of turf, mainly in early fall, causing the melting-out effect. During longer periods of favorable conditions, the turf was killed and this disease was very serious. Helminthosporium melting out was one of the most prevalent turf diseases, especially on bentgrass.

Brown patch, which previously was regarded as a disease occurring only in hot weather, damaged turf in October and February (Fig. 3). This disease affected turf grasses in winter as well as in late summer and fall in the Vancouver area.

Red thread, one of the most prevalent lawn diseases on fescue on the lower mainland in the warm, humid season, nearly disappeared when the weather became cool. Normally only the leaves were affected, and the turf recovered within 3 weeks.

Rust and powdery mildew were the most prevalent diseases of Kentucky bluegrass. Both occurred in late summer but were not very destructive. They did not kill the plants, but ruined the appearance of the turf.

Dollar spot occurred on creeping bentgrass in the turf nursery at Vancouver but did not spread to other species growing close by. It disappeared within a month and, therefore, was not considered as a major disease in the Lower Mainland.

Fairy ring occurred in only one lawn examined. It was not widespread, but it is considered to be one of the most destructive turf diseases in home lawns in Vancouver.

Acknowledgments

The assistance of Dr. N. MacClean, Division of Plant Science, University of British Columbia, for advice and for identifying pathogens, and of Miss Evelyn Jack, Botanical Garden, University of British Columbia, in laboratory work, is gratefully acknowledged.

Literature cited

1. Dahl, A.S. 1934. Snow mold of turf grasses as caused by Fusarium nivale. Phytopathology 24:197-214.

2. Gould, C.J. 1964. Research on turf grasses diseases in Washington State, U.S.A. J. Sports Turf Res. Inst. No. 40.

3. Meiners, J.P. 1955. Etiology and control of snow mold of turf in the Pacific Northwest. Phytopathology 45:59-62.

INCIDENCE OF PHOMA MEDICAGINIS IN ALFALFA SEED PRODUCED IN CANADA AND THE U.S.A[1]

Howard Harding[2]

Abstract

Phoma medicaginis Malbr. & Roum. was isolated from alfalfa seeds produced in five provinces in Canada and nine states in the U.S.A. The highest incidence was in seed produced in the Prairie Provinces. Eighty-five percent of the samples of Saskatchewan grown seed yielded P. medicaginis, with infestation levels ranging from 0 to 34.2% (average 7.8%).

Introduction

Black stem, caused by Phoma medicaginis Malbr. & Roum., is the most widespread and one of the two most important foliage diseases of alfalfa in western Canada. Several workers have shown that the causal organism is seed-borne (1, 3, 5, 6) and the present study was initiated to determine the incidence of P. medicaginis in alfalfa seed obtained from several locations in Canada and the U.S.A.

Materials and methods

Seed was obtained from five provinces and 10 states in the U.S.A. With two exceptions, all seed was produced in 1969. The New York (Geneva) seed was grown between 1966 and 1969 and two of the Kansas samples were harvested in 1968. Nonsterilized seeds were plated onto malt extract agar (2.0% Difco malt extract, 1.5% Bacto agar) containing 50 ppm vancomycin hydrochloride (Vancocin, Eli Lilly Co.) and 100 ppm streptomycin sulphate (Nutritional Biochemicals Co.) (4). Thirty plates of 12 seeds/plate were prepared from each sample. Plates were incubated in the laboratory at about 24C for 7-10 days and the number of P. medicaginis colonies was recorded. Three reference samples, each of which had a high level of infestation, were plated out at intervals over the time taken to test all the samples.

Results

A total of 198 samples were examined and P. medicaginis was obtained from 75.3% of them. The number of colonies obtained from the three reference samples did not change appreciably over the 6-month period needed to test all the samples. The highest incidence was found in seed produced in the Prairie Provinces (Table 1). Seed produced in Ontario and most of the U.S.A. samples had a low level of infestation. Of seed produced in Saskatchewan, 85% of the samples yielded P. medicaginis. Table 2 shows the incidence of P. medicaginis in seed of named alfalfa varieties produced in several locations in Saskatchewan. Most of the samples from the main seed-growing area around White Fox were quite heavily infested, including three samples of Foundation seed. The lowest incidence was found on the variety 'Roamer', while almost all of the 'Rambler' samples yielded P. medicaginis.

Discussion

The present data indicate that the black stem fungus is present on most of the alfalfa seed produced in western Canada, and this probably contributes to the continued widespread nature of the disease. Disease surveys have shown that black stem is relatively unimportant in well managed forage crops (2) and the use of clean seed would probably further decrease the incidence of the disease. The disease is most prevalent in seed crops, particularly in old stands. It would be enlightening to find out the average age of seed-producing stands in Saskatchewan; and also what measures of crop hygiene are being employed. Certainly, an average of 8% infestation by a plant pathogen in any seed crop is highly undesirable.

Acknowledgments

The assistance of several cooperators, particularly Mr. F.W.S. Dale of the Plant Products Division, Saskatoon, in providing seed samples, is greatly appreciated.

---

[1] Contribution No. 423, Research Station, Canada Department of Agriculture, Saskatoon, Saskatchewan.

[2] Plant Pathologist.

Table 1. Incidence of Phoma medicaginis in alfalfa seed samples produced in Canada and the U.S.A.

Location	No. of samples tested	No. of samples yielding Phoma medicaginis	% seeds yielding P. medicaginis	
			Range	Average
Saskatchewan	64	55	0-34.2	7.8
Alberta	14	13	0-45.3	15.0
Manitoba	13	13	<1.0-27.8	7.5
Ontario	15	13	0-3.3	1.3
British Columbia	3	3	<1.0-8.0	5.4
Idaho	12	3		<0.1
New York	11	9	0-3.6	1.3
Nebraska	5	4	0-6.4	2.2
S. Dakota	18	16	0-2.5	1.0
Minnesota	3	1		<0.1
Kansas	3	2		0.3
Oregon	12	2		<0.1
Montana	12	8	0-2.5	0.9
Iowa	2	0		0
N. Dakota	7	5	0-2.8	0.8

Table 2. Incidence of Phoma medicaginis in alfalfa seed samples produced at several locations in Saskatchewan

Variety	Designation	Location	% seeds yielding P. medicaginis	
			Range	Average
Rambler	Foundation	White Fox	4.7-14.4 (3)*	10.9
Rambler	Certified	White Fox	<1.0-23.3 (10)	13.5
Rambler	Certified	Prince Albert	(1)	13.3
Rambler	Commercial	Moose Jaw	6.1-34.2 (3)	21.6
Rambler	Commercial	Swift Current	4.2-5.6 (2)	4.9
Rambler	Commercial	White Fox	5.6-10.6 (2)	8.1
Rambler	Commercial	Prince Albert	1.9-13.9 (3)	7.1
Rambler	Commercial	Nipawin	9.4-16.7 (2)	13.0
Rambler	Commercial	Kinley	(1)	3.8
Rambler	Commercial	Saskatoon	(1)	21.9
Roamer		Swift Current	(2)	<1.0
Roamer	Commercial	White Fox	(2)	<1.0
Vernal	Certified	White Fox	8.9-15.6 (2)	12.2
Vernal	Commercial	White Fox	(1)	12.5
Beaver	Commercial	Prince Albert	(1)	1.1
Beaver	Commercial	White Fox	(1)	<1.0
Grimm		Saskatoon	(1)	16.4

* Figures in parentheses represent number of samples.

Literature cited

1. Cormack, M.W. 1945. Studies on Ascochyta imperfecta, a seed- and soil-borne parasite of alfalfa. Phytopathology 35:838-855.

2. Harding, H. 1970. Foliage diseases of alfalfa in northern Saskatchewan in 1970. Can. Plant Dis. Surv. (In press).

3. Kernkamp, M.F., and G.A. Hemerick. 1953. The relation of Ascochyta imperfecta to alfalfa seed production in Minnesota. Phytopathology 43:378-383.

4. Latham, A.J., and M.B. Linn. 1961. A non-fungistatic bacteriostatic medium containing streptomycin and vancomycin. Plant Dis. Rep. 45:866-867.

5. Leach, C.M. 1960. Phytopathogenic and saprophytic fungi associated with forage legume seed. Plant Dis. Rep. 44:364-369.

6. Mead, H.W. 1953. Studies on black stem of alfalfa caused by Ascochyta imperfecta Peck. I. Seed and seedling phases of the disease. Can. J. Agr. Sci. 33:500-505.

AUTHOR INDEX TO VOLUME 50