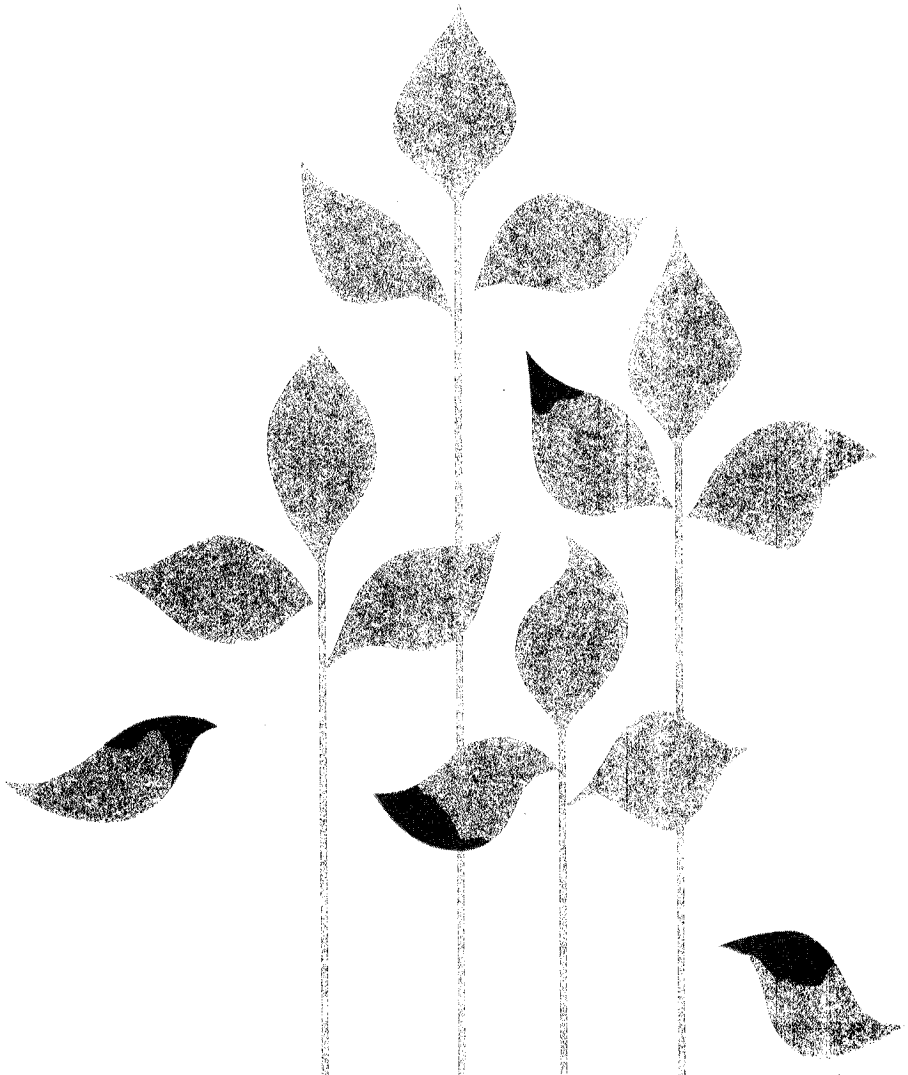
Canadian Plant Disease Survey

Inventaire des maladies des plantes au Canada

Vol. 70, No. 2, 1990

Vol. 70, Nº 2, 1990



Agriculture Canada

Canada

Canadian Plant Disease Survey

Volume 70, Number 2, 1990

CPDS 70(2) 111-140 (1990) ISSN 0008-476X

Inventaire des maladies des plantes au Canada

Volume 70, Numéro 2, 1990

CONTENTS

The *Canadian Plant Disease Survey* is a periodical of information and record on the occurrence and severity of plant diseases in Canada and on the assessment of losses from disease. Other original information such as the development of methods of investigation and control, including the evaluation of new materials, will also be accepted. Review papers and compilations of practical value to plant pathologists will be included from time to time.

Research Branch, Agriculture Canada

Compilers: H.S. Krehm, PhD.
P. Beauchamp, M.Sc.,
Research Program Service,
Agriculture Canada, Ottawa, Ontario K1A 0C6

L'inventaire des maladies des plantes au Canada est un périodique d'information sur la fréquence des maladies des plantes au Canada, leur gravité, et les pertes qu'elles occasionnent. La rédaction accepte d'autres communications originales notamment sur la mise au point de nouvelles méthodes d'enquête et de lutte ainsi que sur l'évaluation des nouveaux produits. De temps à autre, il inclut des revues et des synthèses de rapports d'intérêt immédiat pour les phytopathologistes.

Direction de la recherche, Agriculture Canada

Compilateurs: H.S. Krehm, PhD.
P. Beauchamp, M.Sc.
Services aux programmes de recherche,
Agriculture Canada, Ottawa, (Ontario) K1A 0C6

ERRATUM

Volume 70:(1):85, 1990

The third paragraph should read as follow:
Le troisième paragraphe devrait lire comme suit:

Verticillium wilt (*Verticillium dahliae*) was found in 49% of the fields surveyed with a range from trace to 20% DI. Traces of downy mildew (*Plasmopara halstedii*) were observed in three fields and up to 20% DI in one field in Altona. Low levels of rhizopus head rot (*Rhizopus* spp.), with a range of trace to 5% DI, were observed in 23% of the fields, especially those surveyed towards the end of the season. Traces of septoria leaf spots (*Septoria helianthi*) and stem lesions (*Phoma* spp. and *Phomopsis* spp.) were observed in various sunflower fields towards the end of the season. No signs of sclerotinia head rot (*S. sclerotium*) or botrytis head rot (*Botrytis* spp.) were encountered during the 1989 field survey.

Occurrence of blueberry leaf mottle, blueberry shoestring, tomato ringspot and tobacco ringspot viruses in eleven halfhigh blueberry clones grown in New Brunswick, Canada

Amrik S. Jaswal [1]

In 1980, a field trial was started to evaluate the horticultural value of 11 halfhigh blueberry clones for New Brunswick conditions. Nine of these certified clonal selections were obtained from the University of Minnesota (Mn 61, 84, 135, 327, 330, 332, 350, 354, 360); one from University of Maine (Me-1) and the last one was a N.B. selection (NBBCL) from a seedling line of Mn3. Recently, clones, Mn 332, Mn 350 and Mn 360 were released as cultivars in Minnesota under names of Northsky, Northcountry and Northblue respectively. All halfhigh clones originated from interspecific crosses of highbush *Vaccinium corymbosum* L. and lowbush *Vaccinium angustifolium* plants. All plants of these clonal selections were tested for the occurrence of tomato ringspot (TmRSV), tobacco ringspot (TbRSV), blueberry leaf mottle (BBLMV) and blueberry shoestring (BBSSV) viruses using the enzyme-linked immunosorbent-assay (ELISA) procedure.

All plants of 4 clonal selections namely Mn 84, 135, 354 and NBBCL gave negative ELISA results suggesting that these clones might be less susceptible to these four viruses. Clonal selections which tested positive were: (TmRSV – Mn 61, 330, 332, 350, 360, Me-1; BBLMV – Mn 327, 330, 332, 350, 360, Me-1; BBSSV – Mn 61, 327, 330, 360; TbRSV – Mn 350, 360). From a total of 243 plants tested TmRSV was found to be the most prevalent followed by BBLMV, BBSSV and TbRSV, each infecting 13, 11, 6 and 2 plants respectively.

Can. Plant Dis. Surv. 70:2, 113-117, 1990.

En 1980, on a commencé un essai en plein champ pour évaluer la valeur horticole de 11 clones de bleuet demi-nains dans les conditions météorologiques du Nouveau-Brunswick. Neuf de ces sélections clonales certifiées provenaient de l'Université du Minnesota (Mn 61, 84, 135, 327, 330, 332, 350, 354, 360), une de l'Université du Maine (Me-1) et la dernière du Nouveau-Brunswick (NBBCL) d'une lignée de semis de Mn3. Récemment, on a mis au marché les clones Mn 332, Mn 350 et Mn 360 comme des cultivars au Minnesota sous les noms de Northsky, Northcountry et Northblue respectivement. Tous les clones demi-nains provenaient de croisements interspécifiques du bleuet en corymbe *Vaccinium corymbosum* L. et du bleuet nain *Vaccinium angustifolium* Ait. Tous les plants de ces sélections clonales ont été analysées pour la présence des virus de la tache annulaire de la tomate (TmRSV), de la tache annulaire du tabac (TbRSV), de la marbrure du bleuet (BBLMV) et de la mosaïque en lacet du bleuet (BBSSV) à l'aide du test immuno-enzymatique ELISA.

Tous les plants de quatre sélections clonales soit Mn 84, 135, 354 et NBBCL ont eu des résultats négatifs avec le test ELISA ce qui suggère que ces clones peuvent être moins susceptibles envers ces quatre virus. Les sélections clonales suivantes ont eu des résultats positifs: TmRSV – Mn 61, 330, 332, 350, 360, Me–1; BBLMV – Mn 327, 330, 332, 350, 360, Me–1; BBSSV – Mn 61, 327, 330, 360; TbRSV – Mn 350, 360. Sur un total de 243 plants on a identifié le TmRSV sur 13 plants, le BBLMV sur 11 plants, le BBSSV sur 6 plants et le TbRSV sur 2 plants.

Introduction

Blueberry production in the Province of New Brunswick in Eastern Canada has continued to increase over the past 8-10 years. Various blueberry improvement programs involving interspecific hybridization of lowbush with highbush berries have resulted in the selection of a number of halfhigh clones which have a medium plant stature producing light blue, high quality, high yielding crops. An experimental field trial was started in New Brunswick in 1980 to select a number of halfhigh clones, with desirable characteristics such as high yield, improved fruit quality and high disease resistance.

A number of major viruses and virus-like diseases threaten blueberries in North America (11). Highbush cultivars have been shown to suffer from virus related diseases causing multi-million dollar losses in various parts of the U.S.A. and Canada (4, 5). Since blueberries are woody perennials and represent long-term investments which can be profitable for many years, the occurrence of even a minor virus disease cannot be ignored.

[1] *New Brunswick Department of Agriculture, P.O. Box 6000, Fredericton, New Brunswick, Canada E3B 5H1.*

Accepted for publication December 18, 1989.

Control measures for various *Vaccinium* viruses have been reviewed (11) and disease symptoms well characterized (8, 9, 10, 11). One of the effective ways to guard against virus diseases is to grow cultivars which are resistant or less susceptible to viruses. The main objective of this study was to determine whether or not the 11 halfhigh clones grown in N. B., on trial basis are susceptible to blueberry viruses. All plants of these clones were ELISA tested for viruses for which the antisera was commercially available and included blueberry shoestring (BBSSV), blueberry leaf mottle (BBLMV), tomato ringspot (TmRSV) and tobacco ringspot (TbRVS). BBSSV is aphid vectored (8); transmission of BBLMV is by honey-bees (2) and TmRSV and TbRSV are vectored by the dagger nematodes *Xiphinema americanum* Cobb or *X. rivesi* (1, 4, 6, 7).

Materials and methods

Experimental plots

In 1980, a field trial was started to evaluate the cultivar potentials of 11 halfhigh blueberry clones for New Brunswick soil and climatic conditions. Nine of these clonal selections were obtained from the Minnesota State University (Mn series), one from the University of Maine (Me-1), and one was a N. B. Selection (NBBCL) from a seedling line of Mn3. All clones originated from interspecific crosses of highbush *V. corymbosum* L. and lowbush *V. angustifolium* plants. The trial was established at the New Brunswick Horticultural Centre, Hoyt, on a sandy soil of low fertility and low organic matter. The clones were planted in a randomized complete block design with 4 replications. Each plot consisted of 6 plants spaced 1.0 m apart in rows spaced 2.0 m apart. The test plot was surrounded by approximately 100 metres of bushes and tall trees on one side and large blueberry fields on the other three sides.

Sampling procedure

In preliminary investigations blueberry buds gave considerably less background absorbance than mature leaves; buds, therefore, were chosen as a suitable tissue for ELISA tests. Approximately 100-120 buds were collected from different locations on each plant in late October, stored at – 20°C and assayed during early November. Plants of each clonal selection were ELISA tested in separate batches. Antisera for all viruses was purchased from AGDIA Inc. (30380 Country Road 6, Elkhart, IN, U.S.A.).

Preliminary Tests

Preliminary studies were undertaken to determine optimum experimental conditions for ELISA tests. Five healthy blueberry bud samples (70-80 buds), each weighing 0.5 g were homogenized separately, in 5 mL of extraction buffer. Extracts from 4 samples were spiked separately with 1.0 mL each of BBLMV, BBSSV, TbRSV and TmRSV standards; stored at 5°C and ELISA tested at 1, 6, 17 (overnight), 41 and 65 hr time interval. Extract from the fifth sample was used as a negative standard.

In another experiment, spiked extracts prepared as before were incubated in the antibody coated plates under 3 different conditions: (a) 17 hr (overnight) at 5°C; (b) 2 hr at room temperature; (c) 2 hr at 37°C. Test extracts for (b) and (c) had earlier been stored overnight at 5°C.

When infected plants were identified, identical experiments with respect to (a) storage of bud extract and (b) antibody incubation periods, were conducted on their unspiked bud extracts.

ELISA procedure

The double antibody sandwich ELISA, as described by Clark and Adams (3) was followed except that gamma-globulins were labelled with peroxidase and o-phenylene diamine was used as substrate. For analysis 0.2 g of bud tissue (25-40 buds) was homogenized with pestle and mortar in 2 mL of phosphate buffered saline containing 2% polyvinyl pyrrolidone (PVP, mol. wt. 40,000, Sigma) and 0.5% Tween 20. The extracts were placed in plastic covered vials and stored overnight at approximately 5°C and assayed the following morning. One hundred microliters of extract from test samples or negative or positive standards were placed in wells of antibody-coated ELISA plates (AGDIA Inc.). All samples were tested in duplicate wells except negative standards that were used in 4 wells per plate. The plates were placed on a wet paper towel, sealed in plastic bags and kept at room temperature (approximately 25°C) for 2 hours. The plates were emptied, washed thrice with phosphate buffered saline (PBS) – Tween solution, refilled with 100 microliters of enzyme-conjugate and incubated for 2 hr at room temperature. Plates were once again washed with PBS-Tween and refilled with 100 microliters of freshly prepared substrate. After 30 minutes, the reaction was stopped by adding 50 microlitres of 3 M sulphuric acid to each well. Acid addition was essential not only to stop the reaction but also to obtain proper colour development in the assay. Plates were read visually and the absorbance was measured at A490 in an ELISA reader (Titertek Mulutiskan, Type 310 C, Flow Laboratories, Helsinki, Finland).

Results and discussion

Evaluation of experimental conditions

Absorbance (A490 nm) termed "ELISA values" was used to express the virus concentration. These values obtained when spiked bud extracts were ELISA tested after their storage at 5°C for 1-65 hr are presented in Fig. 1. The results suggested that bud homogenates when tested after 17 hr of storage at 5°C did not adversely effect the ELISA values. These values, however, decreased when the storage time was prolonged to 41 hr or beyond.

ELISA values of samples where spiked bud extracts were subjected to 3 different antibody incubation periods are presented in Fig. 2. Absorbance values of these treatment periods, for any particular virus, are almost similar indicating that either of these incubation periods are equally satisfactory for ELISA procedure.

ELISA values of identical experiments conducted on buds of infected plants are presented in Table 1 and 2. The results showed similar trends as were observed with the spiked samples. The bud homogenate stored for up to 17 hr at 5°C did not adversely affect the virus concentration as was evidenced by the ELISA values. The concentration, however, decreased when the extract was stored for 41 hr or more (Table 1). ELISA values of all 3 antibody incubation treatments (Table 2), for each virus, varied in a small range, reinforcing the earlier conclusions that either of these treatments are of equal value for ELISA procedure.

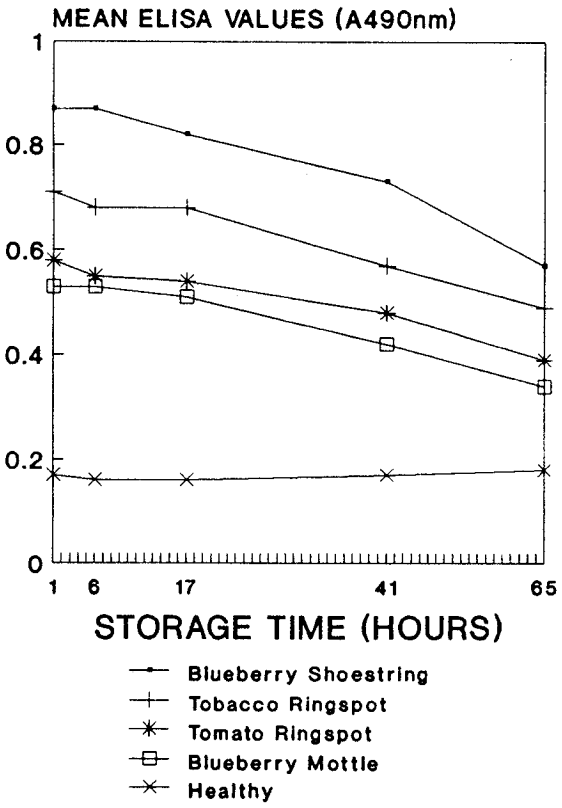


Fig. 1. Effect of storing sample extracts, for 1-65 hr, at 5°C on ELISA values (A490nm). One half gram of healthy blueberry buds (70-80) were homogenized in 5 ml of extraction buffer, spiked with 1.0 ml of virus positive standards and used for ELISA tests. The data are an average of 8 determinations, i.e., 4 each of 2 replicated experiments.

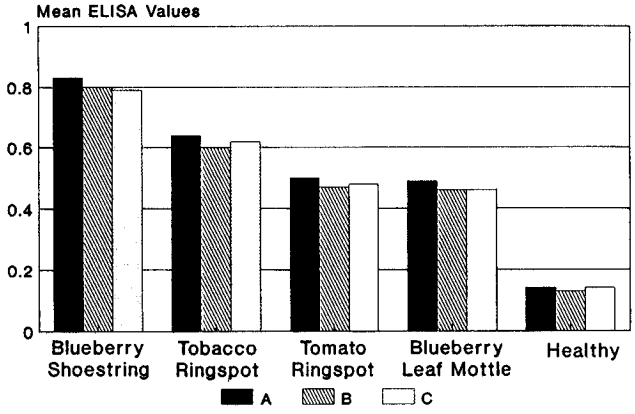


Fig. 2. ELISA values of 3 antibody incubation treatments : (A) Extract placed directly in the antibody coated plate and incubated overnight at 5°C; (B) Extract stored overnight at 5°C then incubated for 2 hr at room temperature; (C) Extract stored as in (B) then incubated for 2 hr at 37°C. Bud samples were extracted and spiked as in Fig. 1.

ELISA results

ELISA test results are presented in Table 3. A few plants in the trial field had died since the 1980 planting; therefore, final plant numbers varied from the initial numbers. The ELISA values of healthy controls of all analyses sets of 11 clonal selections were in the range of 0.10 to 0.21 ($\bar{X} = 0.14$). Test samples showing absorbance value of 0.42 or greater were considered to be ELISA positive.

Mn 332 appeared to be particularly susceptible to TmRSV and BBLMV as 5 of the 24 plants tested were infected individually or with combinations of these viruses. This clone appeared to be less susceptible to BBSSV and TbRSV as all plants tested negative for these viruses.

Mn 360 had the highest rate of infection (24%) and was the most susceptible clone in the lot. This was the only clone which tested positive for all 4 viruses. From a total of 21 plants, 5 were infected: 2 with TmRSV, 1 with BBSSV, 1 with TmRSV and BBLMV and one with TbRSV and BBSSV.

Recently Mn clone 332, 350 and 360 were released as cultivars in Minnesota under the names of Northsky, Northcountry and Northblue respectively. However, all of these clones, especially Mn 360 and 332, although reasonably high yielding under New Brunswick climatic conditions (unpublished information), have been found to be susceptible to the aforementioned viruses. Mn 350 was only moderately

Table 1. Absorbance values* (A490nm) of infected blueberry bud extracts ELISA tested after 1-65 hr of storage at 5°C.

	Time (hours)				
Infecting virus	1	6	17	41	65
Blueberry Leaf Mottle	0.73	0.74	0.70	0.67	0.58
	(0.13)	(0.11)	(0.12)	(0.10)	(0.09)
Blueberry Shoestring	1.10	0.94	0.97	0.83	0.68
	(0.15)	(0.16)	(0.13)	(0.10)	(0.11)
Tobacco Ringspot	0.84	0.86	0.82	0.73	0.54
	(0.13)	(0.15)	(0.16)	(0.13)	(0.12)
Tomato Ringspot	1.50	1.50	1.43	0.89	0.73
	(0.17)	(0.15)	(0.17)	(0.14)	(0.11)

Figures in parentheses represent absorbance values of healthy standards.

* Mean value of 8 determinations, i.e., 4 each of two replicated experiments.

Table 2. Absorbance values[1] of infected blueberry extracts subjected to varying antibody incubation periods.

Infecting virus	Incubation periods: 17 hr* at 5°C (overnight)	2 hr** at room temperature	2 hr** at 37°C
Blueberry Leaf Mottle	0.74	0.70	0.73
	(0.12)	(0.11)	(0.11)
Blueberry Shoestring	1.03	0.96	0.93
	(0.17)	(0.15)	(0.17)
Tobacco Ringspot	0.80	0.76	0.73
	(0.15)	(0.15)	(0.13)
Tomato Ringspot	1.38	1.26	1.22
	(0.18)	(0.14)	(0.16

Figures in parentheses represent absorbance values of healthy standards.

[1] Mean value of 8 determinations, i.e., 4 each of two replicated experiments.

* Bud extract placed directly in the antibody coated ELISA plate.

** Bud extract stored overnight at 5°C before incubation.

susceptible as only 3 of its plants from a total of 21 tested positive: 1 to TmRSV, 1 to BBLMV and TmRSV and one to TbRSV.

Two clonal selections Mn 327 and 330 were ELISA positive 13 and 15% respectively. The results for Mn 327 were noteworthy because it was the only clone found to be free from TmRSV. Mn 61 and Me-1 selections showed 8 and 9% infection respectively, mainly from TmRSV.

Overall TmRSV was found to be the most prevalent virus infecting a total of 13 plants. Except Mn 327, all other virus-positive clones, to some degree, were infected by this virus. The soil of the trial plot is sandy and *Xiphinema americanum* Cobb (TmRSV vector) has been reported to thrive well on soils with high sand content (1). BBLMV was the second most common virus and was detected in 11 plants. All infected clones, with the exception of Mn 61, showed BBLMV presence. BBSSV was present to a moderate extent; a total of 5 plants belonging to 4 different clones (Mn 61, Mn 327, Mn 330 and Mn 360) showed positive reactions for this virus. TbRSV was almost non-existent and was detected only in 2 plants, 1 of Mn 350 and the other of Mn 360.

The ELISA positive plants were eye-indexed for virus symptoms during April to September of the following year. TmRSV was found to infect Mn 61, 330, 332, 350, 360 and Me-1. The most conspicuous symptoms appeared on Mn 350; its stems, twigs and branches exhibited circular brownish necrotic spots, chlorotic spots on leaves and some deformed new apical buds. To a lesser extent Mn 332 also showed similar symptoms. The symptoms on other clones were less noticeable, especially Mn 360; two of its infected plants, except for a few deformed apical leaves, were almost symptomless.

BBLMV infected Mn 327, 330, 332, 350, 360 and Me-1. Prominent symptoms were exhibited by 332; two from a total of three infected plants clearly showed curled, deformed, mottled and some chlorotic leaves, stunted growth and some dieback of old stems. Similar symptoms of a lesser severity were also exhibited by Mn 327. A few mottled leaves were noticed on Me-1 and Mn 360, but Mn 330 and 350 were symptomless.

All BBSSV infected plants of Mn 61, 327, 330 and 360 showed some crescent-shaped leaves. One plant of Mn 360 showed typical BBSSV symptoms. It had some curled up leaves and a few reddish streaks on current and one year old growth. At blossom time a few petals also showed reddish streaks. The presence of BBSSV has also been reported in the adjoining province of Nova Scotia (5).

TbRSV was found only in one plant each of Mn 350 and 360. The infected plants showed some circular brownish spots on stems and branches, necrotic and deformed leaves, stem dieback and stunted growth.

From a total of 11 clonal selections only 4 (Mn 84, 135, 354, NBBCL) were found to be completely free from TmRSV, TbRSV, BBLMV and BBSSV. These selections appear to have adjusted well to the N. B. climatic conditions, considering survival rate of their plants. Plants belonging to Mn 135 and Mn 354 over the 9 year period showed no mortality and this loss for Mn 84 and NBBCL was only 1 plant each, suggesting that these clones probably are hardy and not very susceptible to the aforestated viruses. Further work would be needed to determine if these clones are, in fact, resistant to these viruses.

Table 3. Detection by ELISA of BBSSV, BBLMV, TmRSV and TbRSV in eleven halfhigh blueberry clones in New Brunswick.

Clone	No. of plants tested	No. of infected plants	Virus detected	Infected plants Total	Infected plants %
Mn 61	24	1	TmRSV	2	8
		1	TmRSV, BBSSV		
Mn 84	23	-	-	0	0
Mn 135	24	-	-	0	0
Mn 327	24	2	BBLMV	3	13
		1	BBLMV, BBSSV		
Mn 330	13*	1	BBSSV	2	15
		1	TmRSV, BBLMV		
Mn 332	24	1	TmRSV	5	21
		2	BBLMV		
		2	TmRSV, BBLMV		
Mn 350	21	1	TmRSV	3	14
		1	TbRSV		
		1	TmRSV, BBLMV		
Mn 354	24	-	-	0	0
Mn 360	21	2	TmRSV	5	24
		1	BBSSV		
		1	TmRSV, BBLMV		
		1	TmRSV, BBSSV		
Me-1	22	1	TmRSV	2	9
		1	TmRSV, BBLMV		
NBBC1	23	-	-	0	0

* Insufficient number of plants for each replication.

Acknowledgements

The author is thankful to Dr. R.P. Singh for sharing his laboratory facilites and to Mrs. Anne Dick for typing this manuscript.

Literature cited

1. Allen, W.R., L.W. Stobbs, J.G. Van Schagen and B.A. Ebsary. 1988. Association of *Xiphinema* species with soil type and grapevines infected with tomato ringspot virus in Ontario, Canada. Plant Dis. 72:861-863.
2. Childress, A.M. and D.C. Ramsdell. 1987. Bee-mediated transmission of blueberry leaf mottle virus via infected pollen in highbush blueberry. Phytopathology 77:167-172.
3. Clark, M.F. and A.N. Adams. 1977. Characteristics of the microplate method of enzyme linked immunosorbent assay for the detection of plant viruses. J. Gen. Virol. 34:475-483.
4. Converse, R.H. and D.C. Ramsdell. 1982. Occurrence of tomato and tobacco ringspot viruses and of dagger and other nematodes associated with cultivated highbush blueberries in Oregon. Plant Dis. 66:710-712.
5. Lockhart, E.L. and I.V. Hall. 1962. Note on an indication of shoestring virus in the lowbush blueberry *Vaccinium angustifolium* Ait. Can. J. Bot. 40:1561-1562.
6. McGuire, J.M. 1973. Retention of tobacco ringspot virus by *Xiphinema americanum*. Phytopathology 63:324-326.
7. Powell, C.A., L.B. Forer, R.F. Stouffer, J.N. Cummins, D. Gonsalves, D.A. Rosenberger, J. Hoffman and R.M. Lister. 1984. Orchard weeds as hosts of tomato ringspot and tobacco ringspot viruses. Plant Dis. 68:242-244.
8. Ramsdell, D.C. 1979. Physical and chemical properties of blueberry shoestring virus. Phytopathology 69:1087-1091.
9. Ramsdell, D.C. and R. Stace-Smith. 1979. Blueberry leaf mottle, a new disease of highbush blueberry. Acta Hort. 95:37-45.
10. Ramsdell, D.C. and R. Stace-Smith. 1981. Physical and chemical properties of the particles and ribonucleic acid of blueberry leaf mottle virus. Phytopathology 71:468-472.
11. Ramsdell, D.C., J.F. Hancock and A.W. Stretch. 1981. Virus and viruslike diseases of *Vaccinium* (blueberry and cranberry). *In*: Virus diseases of small fruits. Converse, R.H. editor. United States Department of Agriculture, Agriculture Handbook No. 631, p. 101-126.

First report of eyespot [*Pseudocercosporella herpotrichoides*] in wheat in the Prairie Provinces

Stephen W. Slopek [1], Brent Fletcher [2] and Ted J. Labun [3]

This is the first report of *Pseudocercosporella herpotrichoides* ((Fron) Deighton), the causal agent of eyespot, infecting wheat in the Prairie Provinces. The fungus was first found in a field of Katepwa wheat near Smoky Lake, Alberta. In subsequent investigations eyespot was found in both spring (hard red and soft white) and winter wheat crops at several locations in Alberta in 1989. In a survey of ten wheat fields near Olds, Alberta, eyespot was found in all of the fields and on average, 41 percent of stems examined had one or more lesions. In a field of winter wheat near Innisfail, 74 percent of the stems examined had eyespot lesions and of these approximately 25 percent were severe enough to cause lodging.

Can. Plant Dis. Surv. 70:2, 119-121, 1990.

C'est le premier signalement de *Pseudocercosporella herpotrichoides* ((Fron) Deighton), l'agent responsable de la tache ocellée qui infecte le blé dans les Prairies. On a découvert le champignon pour la première fois dans un champ de blé Katepwa près de Smoky Lake en Alberta. Lors d'enquêtes subséquentes, on a signalé la présence du champignon dans les cultures de blé de printemps (roux vitreux et blanc tendre) et d'hiver à plusieurs endroits de la province en 1989. Dans une enquête sur dix champs de blé près de Olds (Alberta), on a trouvé de la tache ocellée dans tous les champs et en moyenne, 41% des tiges examinées affichaient une ou plusieurs lésions. Dans un champ de blé d'hiver près d'Innisfail, 74% des tiges examinées comportaient des lésions dues à la maladie dont environ 25% étaient assez graves pour causer la verse.

Eyespot [*Pseudocercosporella herpotrichoides* ((Fron) Deighton)], also known as foot rot or strawbreaker, is a widespread disease of wheat, barley, oats and rye. It has been reported from Europe, the USSR, South Africa, parts of North America and Australasia (Anon. 1981). In Canada, eyespot has been reported on wheat in Ontario, Quebec, British Columbia (Conners 1967, Ginns 1986) and the Maritime Provinces (Martin 1989) and on barley in Alberta (Slopek 1989). In 1989, eyespot was discovered in a field of Katepwa wheat near Smoky Lake, Alberta. This is the first report of this disease on wheat in the Prairie Provinces.

P. herpotrichoides appears to be widely distributed in Alberta. Slopek & Labun (1990) found eyespot present in ninety percent of the 54 barley fields that they surveyed in Alberta, with an average incidence per field of 24.6 percent. In a survey of ten hard red spring wheat fields near Olds in 1989 eyespot lesions were found in each of the fields. Fifty randomly selected stems were examined per field and an average of 41 percent of these had one or more eyespot lesions present. The highest incidence of eyespot in any one field was 72 percent. Most of the stems examined in the spring wheat that was surveyed had slight to moderate infections, although there were also some severe infections. Maximum yield losses due to eyespot in the spring wheat surveyed were estimated at approximately five percent. Square meter samples were taken in one field of Katepwa spring wheat near Smoky Lake in an attempt to determine the amount of yield loss resulting from eyespot. Samples were taken from a lodged part of the crop and the adjacent standing crop. In the standing crop, the yield was 5.31 t/ha and the thousand kernel weight was 35.8 g. The yield and thousand kernel weight in the lodged area were 4.74 t/ha and 33.3 g, respectively. It is felt that the eyespot was the primary factor responsible for the lodging and yield loss in this crop. Four soft white spring wheat fields in southern Alberta were also surveyed. Eyespot was found in two of these fields but disease incidence averaged only 3 percent.

In addition, an intensive survey of a field of Norstar winter wheat in the Innisfail area was conducted. The crop was severely lodged (Figure 1) and it was suspected that this might be due to eyespot. Plants were collected along two transects through the field and examined for eyespot lesions. A total of 481 stems were examined, of which 72 percent had one or more eyespot lesions; approximately, one quarter of these were severe. The expected yield for this field of winter wheat was 4.0 t/ha but the actual yield obtained was only 2.6 t/ha and was graded as No. 3 Canada Western Red Winter, with high levels of shrivelled grain. It is suspected that eyespot was a major yield loss factor in this winter wheat crop.

Yield reductions due to eyespot infections occur as a result of the direct effects on the movement of water and nutrients in the host and through indirect effects resulting from lodging (Figure 2). In winter wheat, yield losses of up to 50% have been reported (Bruehl et al. 1968). Scott & Hollins (1974) have found that severe infections (stem completely girdled by lesions; tissue softened so that lodging would readily occur (Figure 3)) reduces both grain number per head and the thousand kernel weight. Whereas, slight lesions do not cause

[1] *Regional Crops Laboratory, Alberta Agriculture, Olds, Alberta T0M 1P0.*

[2] *Victoria Trail Farms, General Delivery, Smoky Lake, Alberta T0A 3C0.*

[3] *Ciba-Geigy Canada Ltd., 820-26 St. N.E., Calgary, Alberta T2A 2M4.*

Accepted for publication January 10, 1990.

any yield loss and moderate lesions (one or more lesions occupying at least half the circumference of the stem) only reduce the thousand kernel weight.

Eyespot is not normally considered to be an important disease in spring cereals (Hollins 1989, Wiese 1977). This disease, however, has the potential to cause significant economic yield losses in winter wheat. At present, very little winter wheat is grown in the Parkland areas of Alberta. With the development of winter wheat cultivars adapted to this area we will undoubtedly see more situations of significant yield losses due to eyespot. At present, there is no active attempt to incorporate eyespot resistance in winter wheat being developed for this area.

Acknowledgements

The assistance of Ron Woolf, District Agriculturist, Alberta Agriculture, Olds, in conducting the disease survey of wheat fields around Olds was very much appreciated.

Literature cited

1. Anon. 1981. Distribution maps of plant diseases. No. 74. Commonwealth Mycological Institute, Kew.
2. Bruehl, G.W., W.L. Nelson, F. Koehler and O.A. Vogel. 1968. Experiments with Cercosporella foot rot (strawbreaker) disease of winter wheat. Bull. of Wash. Agric. Exp. Sta. 694:1-14.
3. Conners, I.L. 1967. An annotated index of plant diseases in Canada. Canadian Dept. Agric. Publ. 1251, Ottawa, Canada. 381 pp.
4. Ginns, J. 1986. Compendium of plant diseases and decay fungi in Canada, 1960-1980. Canadian Government Publishing Centre, Ottawa, Canada. 416 pp.
5. Hollins, T.W. 1989. Plant Breeding International, Cambridge, England. (personal communication).
6. Martin, R.A. 1989. Agriculture Canada, Charlottetown, Prince Edward Island, Canada. (personal communication).
7. Scott, P.R. and T.W. Hollins. 1974. Effects of eyespot on the yield of winter wheat. Ann. Appl. Biol. 78:269-279.
8. Slopek, S.W. 1989. First report of eyespot *Pseudocercosporella herpotrichoides* in spring barley in Alberta. Can. Plant Dis. Surv. 69:125-127.
9. Slopek, S.W. and T.J. Labun. 1990. Incidence of eyespot in barley in Alberta, 1989. Can. Plant Dis. Surv. 70:36.
10. Wiese, M.V. (ed). 1977. Compendium of wheat diseases. American Phytopathological Society, St. Paul, Minnesota. 106 pp.

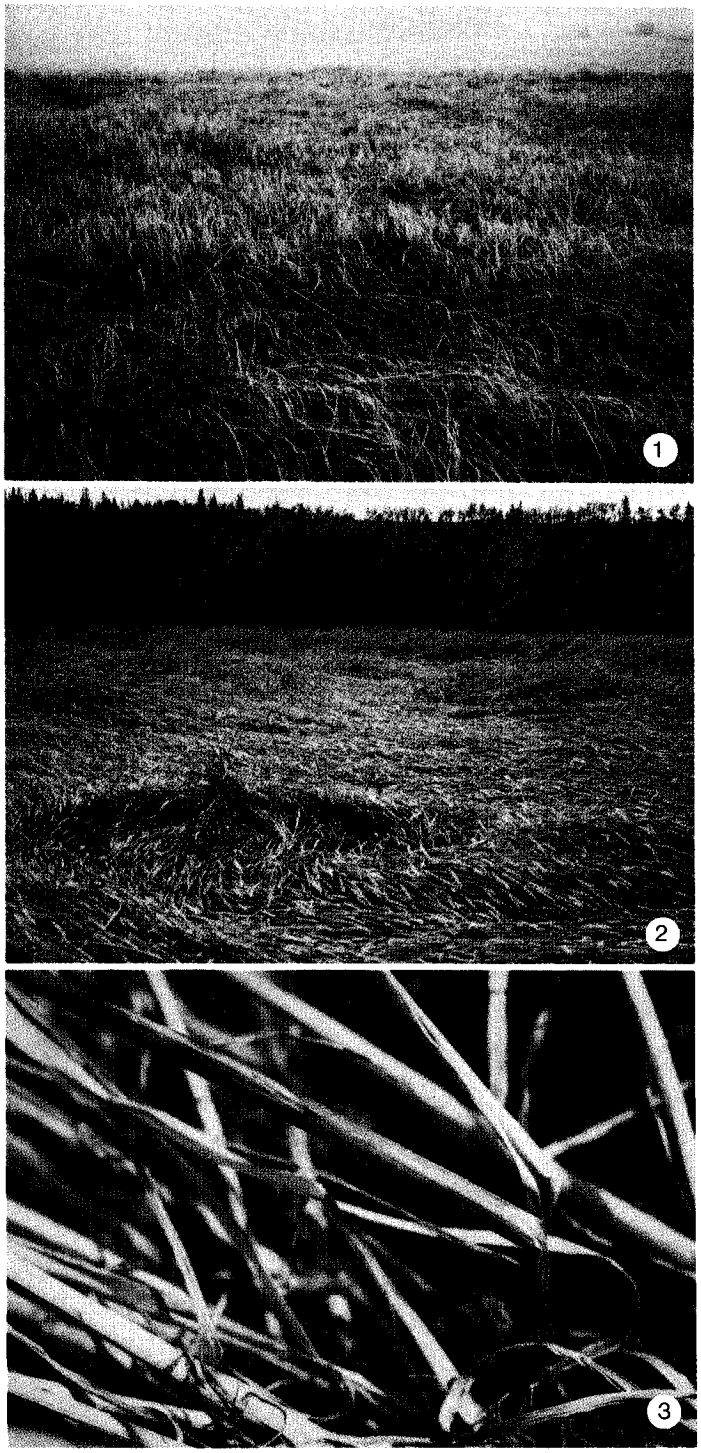


Fig. 1. Lodged crop of winter wheat, cv. Norstar, near Innisfail, Alberta. Seventy-two percent of the stems that were examined had one or more eyespot lesions.

Fig. 2. Lodging in a crop of Katepwa spring wheat near Smoky Lake, Alberta.

Fig. 3. Katepwa spring wheat stem with several eyespot lesions. The lowest lesion causing softening of the stem tissue and lodging.

Ascochyta blight in lentil crops and seed samples in Saskatchewan in 1988

S. Bedi and R.A.A. Morrall [1]

Ascochyta blight caused by *Ascochyta fabae* f. sp. *lentis* was found in 22 of 118 lentil crops surveyed in 1988 in Saskatchewan. Over half of the crops were grown from seed containing at least a trace of the pathogen and three seed samples contained at least 5% infection. Weather conditions were very unfavorable for disease development and crop growth in most areas of the province. Samples of harvested seed were obtained from 77 of the crops and only 31 (40%) showed any infection. However, the highest level was 55%. There was evidence in about 20 crops that the main source of infection was inoculum which had moved into the field from residues of a 1987 lentil crop in an adjacent field. However, movement into the field was limited.

Can. Plant Dis. Surv. 70:2, 123-125, 1990.

Dans une enquête qui a eu lieu en 1988 en Saskatchewan, l'ascochytose (*Ascochyta fabae* f. sp. *lentis*) a été notée dans 22 cultures de lentilles sur 118. Pour plus de la moitié de ces cultures, on a utilisé une semence contenant au moins des traces du champignon pathogène. Cependant, trois échantillons contenaient au moins 5 % de graines infectées. Dans la plupart des régions de la province, les conditions de température et de pluviométrie n'ont pas été très favorables ni pour le développement de l'ascochytose, ni pour la croissance de la culture. Des échantillons de graines provenant de 77 cultures ont été récoltés et analysés. Seulement 31 (40 %) échantillons étaient infectés. Néanmoins, le taux d'infection le plus élevé a été 55 %. Il semble que, pour environ 20 cultures, la principale source d'infection a été l'inoculum provenant des restes de culture de lentilles des champs adjacents, cultivées en 1987. Cependant, l'expansion du champignon pathogène dans le champ était faible.

Introduction

Lentils have been grown in Saskatchewan since 1970 and production peaked in 1987 at over 200,000 ha in the province. The most serious disease of the crop is ascochyta blight, which was first reported in Canada in 1978 (7). Under epidemic conditions the disease has major effects on both seed quality and yield, causing losses of more than 70% of potential income (2). Ascochyta blight is caused by *Ascochyta fabae* f. sp. *lentis* Gossen et al., a pathogen that is specific to lentil and does not affect other pulse crops in western Canada (4).

The pathogen is spread by rain splash and is both seed-borne and stubble-borne. The percentage of infected seeds that give rise to infected seedlings is generally low but infested crop residue is a highly effective source of inoculum and may result in severe epidemics (3). Recommendations for disease control include not seeding lentil crops on lentil stubble and avoiding the use of infected seed (5). The use of these two control measures is effective in controlling ascochyta blight under experimental conditions (R.A.A. Morrall, unpublished data). However, in 1987, there were reports of growers suffering major epidemics after planting disease-free seed in fields that had not been cropped to lentils for four years. Accordingly, a survey was undertaken in 1988 with the principal objective of trying to identify sources of inoculum in crops infested with ascochyta blight.

[1] *Department of Biology, University of Saskatchewan, Saskatoon, Saskatchewan S7N OWO.*

Accepted for publication January 10, 1990.

Methods

One hundred and eighteen commercial crops in nine Saskatchewan crop districts (Fig. 1) were sampled. Attempts were made to obtain information for each field from growers on cropping history for the previous four years, the crops planted in adjacent fields in 1987, agronomic practices and cultivar. In most cases the growers also supplied a sample of the seed that had been planted in the field. These seeds were plated on 20% V8 juice agar to test for ascochyta infection (6). Between 100 and 400 seeds were picked at random from each sample, surface disinfected for 10 min in 0.6% NaOCl, plated and incubated at room temperature for about 10 days before colonies of *Ascochyta* were counted. If no *Ascochyta* was detected in the randomly picked seeds, isolations were made from selected discolored seeds in the sample. If only the selected seeds yielded *Ascochyta* colonies, the infection level was recorded as a trace.

Most of the crops were seeded between late April and early May. Field inspections were carried out from June until harvest time in August. Two visits during the growing season were planned to obtain some idea of changes of disease with time. However, second visits were made only in areas with moderate or good crops; drought-stricken areas were not revisited. During each visit the following data, based on visual estimation while walking through part of the crop, were recorded: crop density and height, growth stage, presence of ascochyta blight and presence of other diseases. In fields where lentils had been planted in an adjacent field in 1987, particular attention was paid to differences between the edge of the crop next to the lentil residues and the rest of the crop. Microscopic examination and isolations were done on specimens whenever there was uncertainty about disease symptoms.

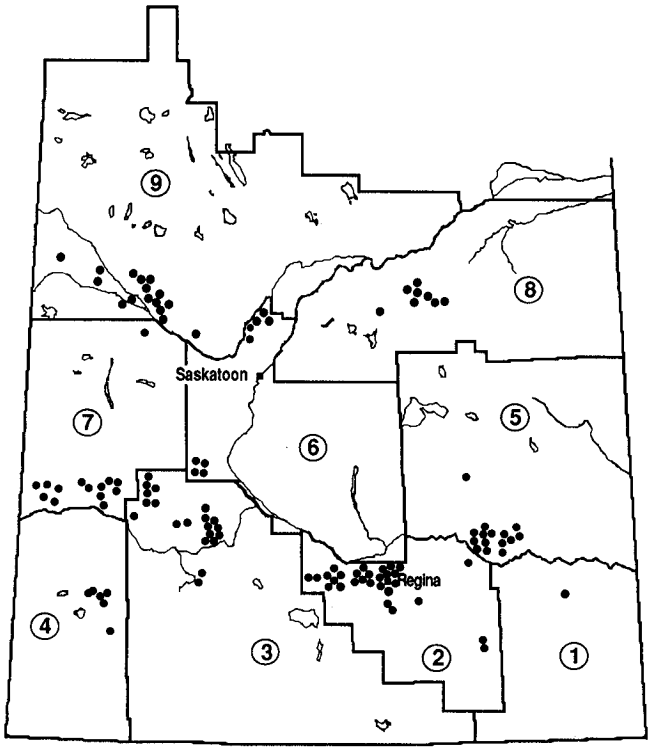


Fig. 1. Map of Saskatchewan crop districts showing approximate locations of lentil crops surveyed.

Table1. Incidence of ascochyta blight in lentil crops in Saskatchewan in 1988.

Sask. crop district**	First inspection* No. of crops	First inspection* No. of crops with ascochyta	Second inspection No. of crops	Second inspection No. of crops with ascochyta
1	1	0		
2	28	0		
3	20	3		
4	6	1		
5	15	0	14	5
6	10	1	5	1
7	14	2	3	1
8	8	0	7	0
9	16	1	15	10
Total	118	8	44	17

* First inspection from June 21 to July 15, second inspection from July 22 to August 4.
** See Fig. 1.

During harvest seed samples were collected by the growers. In crops adjacent to 1987 lentil residues, growers were requested to collect separate samples from the side of the field adjacent to the residues and from elsewhere in the field. The samples were tested for percentage ascochyta infection at Saskatoon, as described above.

Results and discussion

The relative frequency of lentil cultivars and types among the crops surveyed was as follows: Laird – 78%; Eston – 7%; French Green (Du Puy) – 9%; Common Chilean – 6%. In contrast with the other three highly susceptible types, Laird is moderately resistant to ascochyta blight. However, this resistance tends to break down at maturity and a high level of seed infection and discoloration may occur (1).

Drought resulted in poor emergence, short plants, early maturity and very low yields in many areas, especially crop districts 1-4 and 6-7 (Fig. 1). Ascochyta blight was found in only 8 of 118 crops (7%) inspected during initial visits in June or early July and 17 of 44 crops (39%) revisited in August (Table 1). Generally the crops most affected were in central and northern districts, which were least affected by the drought. There was no obvious relationship between disease severity and cultivar, crop density, crop history of the field or agronomic practices. However, by August, the heaviest infection was in a field where the crop had been planted on lentil stubble.

Ascochyta blight was found in 16 of 38 crops (42%) which were adjacent to residues of a 1987 lentil crop. In many of these crops there was more disease on the side of the field adjacent to the residues than elsewhere. In three such crops in the very dry southern and western regions, infected plants were found only very close to the 1987 residues. Thus, movement of ascochyta blight into lentil crops from infested residues in adjacent fields was limited.

The ranges of infection levels in samples of seed planted and harvested are presented in Tables 2 and 3. Over 50% of seed samples planted contained at least a trace of infection and some were as high as 5%. The most heavily infected samples were planted mainly in the northern crop districts, where moister conditions made the potential for disease spread greater.

A substantial number of crops were not harvested because of the drought and 62.5% of all harvested seed samples showed no ascochyta infection (Tables 2 and 3). Samples were collected from two or more parts of 30 crops where either fields with 1987 lentil residues were adjacent or a disease gradient had been observed (Table 3). In 21 of these, levels of seed infection were higher in one part than another, thereby confirming the indication during field inspection that movement of ascochyta into the crop was limited.

Table 2. Percentage ascochyta infection of seed samples planted and harvested by Saskatchewan lentils growers in 1988.*

Percentage ascochyta infection	No. of samples of seed planted**	No. of samples of seeds from crops where only a single sample was harvested
0	50	37
Trace***	14	3
0.25–1.0	22	2
1.25–2.0	7	1
2.25–4.75	8	4
5.00–7.00	3	–
Total	102	47

* See Table 3 for results for harvested seed samples from crops where more than one sample was collected.
** Some samples were planted in more than one field.
*** Trace–ascochyta infection only in selected discolored seeds.

Table 3. Distribution of 1988 lentil crops in which seed samples were harvested at more than one location in relation to extreme values of perecntage seed infection with ascochyta.

Extreme values of % ascochyta infection	No. of crops	Extreme values of % ascochyta infection	No. of crops
0–0	9	3–16	1
0–Trace*	1	5–33	1
0–0.25	1	6–23	1
0–1	7	8–19	1
0–2	2	11–12	1
0–5	1	15–32	1
0.5–8	1	39–55	1
1–46	1		

* Trace–ascochyta infection only in selected discolored seeds.

Root rot and heat canker (5) were also observed in the present survey. Root rot, probably caused by *Rhizoctonia* and *Fusarium*, was present at trace levels in most fields. Heat canker was very common in early summer, due to the extreme heat accompanying the drought. In some fields as many as 50% of the plants were affected.

Conclusions

Low disease severity and seed infection in most crops were a result of very dry conditions in most lentil growing areas. In crops where disease was observed, a frequent source of infection was lentil residues from 1987 in adjacent fields. Gradients of disease severity and percentage seed infection were evident in these crops. Further work on quantitative aspects of such gradients is necessary. However, in about half of the crops surveyed seed-borne inoculum was present, albeit usually at low levels. This emphasizes a potential for disease development in the Saskatchewan lentil crop when very moist conditions prevail, even when stubble-borne inoculum is absent.

Acknowledgements

The authors are grateful for the technical assistance of B. Cavers, J. Melin, C. Harren and J. Downing. Financial assistance was provided by the Saskatchewan Pulse Crop Development Board and the Agriculture Development Fund of Saskatchewan Agriculture.

Literature Cited

1. Gossen, B.D. 1985. Ascochyta blight of lentil in Saskatchewan. Ph.D. thesis, Univ. of Saskatchewan, Saskatoon, Saskatchewan. 164 pp.
2. Gossen, B.D. and R.A.A. Morrall. 1983. Effect of ascochyta blight on seed yield and quality of lentils. Can. J. Plant Pathol. 5:168-173.
3. Gossen, B.D. and R.A.A. Morrall. 1986. Transmission of *Ascochyta lentis* from infected lentil seed and plant residue. Can. J. Plant Pathol. 8:28-32.
4. Gossen, B.D., J.W. Sheard, C.J. Beauchamp and R.A.A. Morrall. 1986. *Ascochyta lentis* renamed *Ascochyta fabae* f. sp. *lentis*. Can. J. Plant Pathol. 8:154-160.
5. Martens, J.W., W.L. Seaman and T.G. Atkinson (eds). 1988. Diseases of field crops in Canada. Can. Phytopathol. Soc. (Revised edition). 160 pp.
6. Morrall, R.A.A. and C.J. Beauchamp. 1988. Detection of *Ascochyta fabae* f. sp. *lentis* in lentil seed. Seed Sci. Technol. 16:383-390.
7. Morrall, R.A.A. and J.W. Sheppard. 1981. Ascochyta blight of lentils in western Canada: 1978-1980. Can. Plant Dis. Survey 61:7-13.

Occurrence of *Peronospora farinosa* f. sp. *chenopodii* on quinoa in Canada

J.P. Tewari and S.M. Boyetchko [1]

Peronospora farinosa f. sp. *chenopodii* is described for the first time on quinoa (*Chenopodium quinoa*) in Canada.

Can. Plant Dis. Surv. 70:2, 127-128, 1990.

Les auteurs décrivent *Peronospora farinosa* f. sp. *chenopodii* pour la première fois sur l'ansérine quinoa (*Chenopodium quinoa*) au Canada.

Quinoa (*Chenopodium quinoa* Willd) is a pseudocereal crop of the Andes where it was first domesticated and cultivated (6). The grain of quinoa contains about 10-17% protein which is higher than in wheat(6). A recent review concluded that quinoa will be a suitable break crop for use in rotation with cereals (6).

A few farmers in Alberta grew quinoa in small fields in 1989. This paper reports on a downy mildew disease of this crop that was observed in one of these fields.

Diseased leaves of quinoa were collected from the Barrhead area on August 30, 1989. Lactophenol cotton blue mounts of the spore-producing structures were prepared and examined by light microscopy. A few diseased leaves have been deposited at the Mycological Herbarium, Biosystematics Research Centre as DAOM 211565.

The diseased leaves showed chlorotic lesions (Fig. 1). The dichotomously branched conidiophores culminated in pointed, slightly curved branches which produced conidia (Figs. 2, 3). The conidiophores ranged from 175-425 µm (average 305 µm) in height and were 8-14 µm (average 11 µm) wide at the bases. The conidia were pyriform and 28.6-36.7 (average 32.2) X 16.3-26.5 (average 20.6) µm in size.

The pathogen was identified as *Peronospora farinosa* (Fr.) Fr. (7). The downy mildew disease of quinoa was first reported from Peru in 1947 (4) and is the most widespread disease of this crop (6). In view of the intergeneric host non-infectivity of *P. farinosa* strains, those on *Chenopodium* spp. were classified as *P. farinosa* f. sp. *chenopodii* (2) to which the present collection belongs. Risi and Galwey (6) indicated that a further subdivision may be necessary as quinoa mildew does not infect *C. pallidicaule* Aellen and that *C. album* L. mildew does not infect some other species of *Chenopodium* L.

This appears to be the first report of *P. farinosa* on quinoa, not only from Canada (3, 5), but also from the whole of North America (Dr. J. Ginns, personal communication). The source of inoculum for this downy mildew disease in Alberta is not certain at this time. *Peronospora* Corda is reported on some *Chenopodium* spp. in the Canadian prairies (3,5). However, due to interspecific host non-infectivity, these are not likely to be the source of inoculum for infection on quinoa. The pathogen is known to be seed-borne (1) and this was probably the source of inoculum of the pathogen in Alberta. A similar conclusion was reached in a study from Britain (6).

[1] *Department of Plant Science, University of Alberta, Edmonton, Alberta, Canada T6G 2P5.*

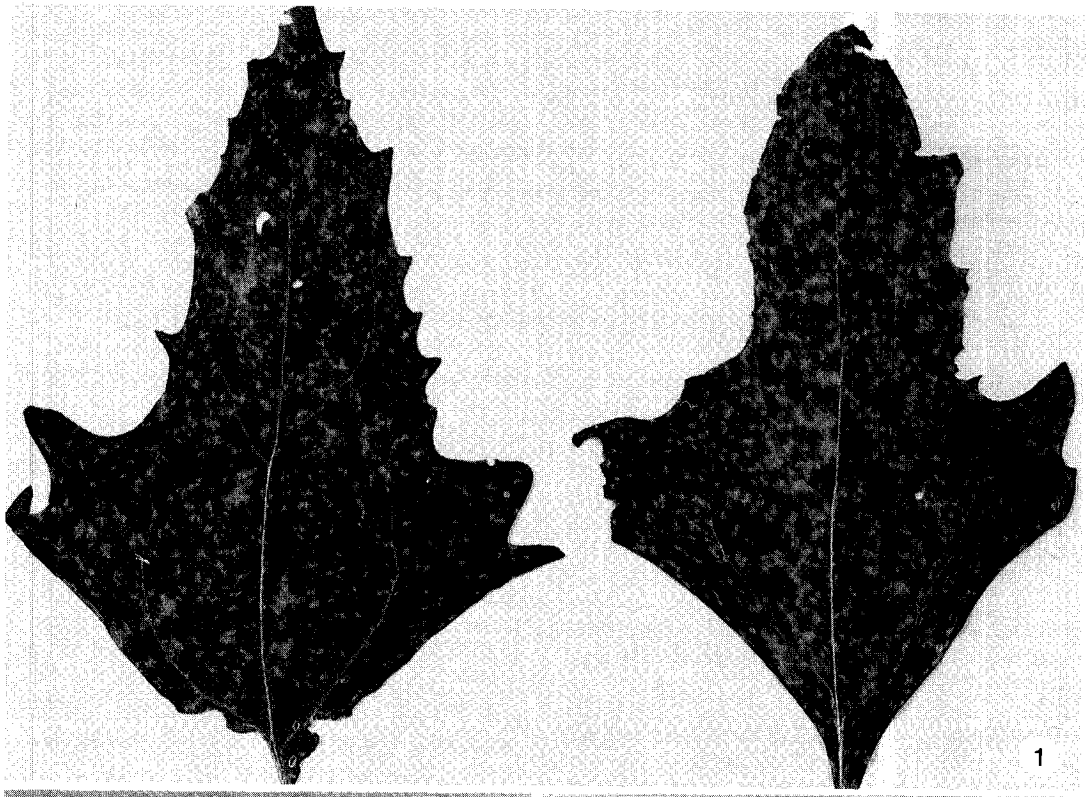
Accepted for publication April 3, 1990.

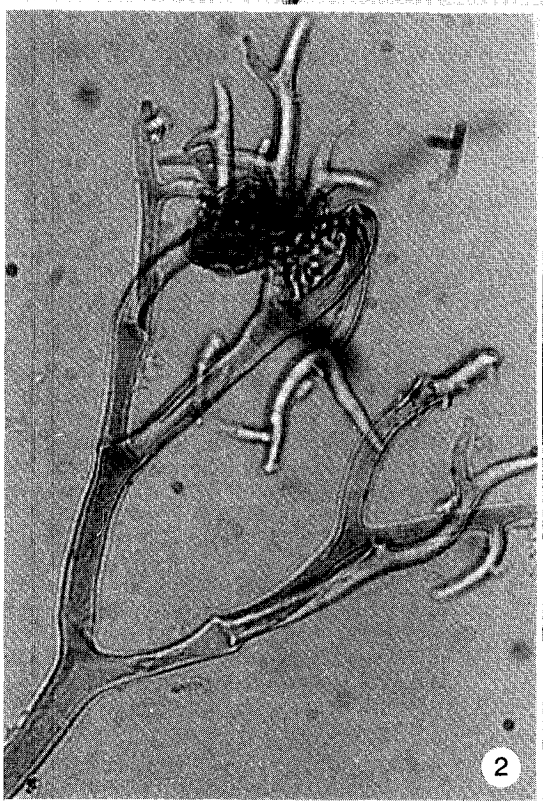
Acknowledgements

We thank Mr. J. Soldan, District Agriculturist, Barrhead, Alberta for showing us a field of quinoa in Alberta and Dr. J. Ginns, Curator, National Mycological Herbarium of Canada, Biosystematics Research Centre, Ottawa for examining the diseased specimens.

Literature cited

1. Alandia, S., V. Otazú and B. Salas. 1979. Enfermedades. *In*: Quinua y Kañiwa. Cultivos Andinos, M.E. Tapia (ed.), Serie Libros y Materiales Educativos No. 49, pp. 137-148. Instituto Interamericano de Ciencias Agrícolas, Bogotá, Colombia.
2. Byford, W.J. 1967. Host specialisation of *Peronospora farinosa* on *Beta*, *Spinacea* and *Chenopodium*. Trans. Br. Mycol. Soc. 59:603-607.
3. Conners, I.L. 1967. An annotated index of plant diseases in Canada. Canada Department of Agriculture. Publication 1251.
4. Garcia, R.G. 1947. "Fitopatología Agrícola del Peru." Estación Agricola de La Molina, Ministerio de Agricultura, Lima, Peru.
5. Ginns, J.H. 1986. Compendium of plant disease and decay fungi in Canada 1960-1980. Research Branch, Agriculture Canada. Publication 1813.
6. Risi, C.J. and N.W. Galwey. 1984. The *Chenopodium* grains of the Andes: Inca crops for modern agriculture. Adv. Appl. Biol. 10:145-216.
7. Yerkes, W.D. and C.D. Shaw. 1959. Taxonomy of the *Peronospora* species on cruciferae and chenopodiaceae. Phytopathology 49:499-507.





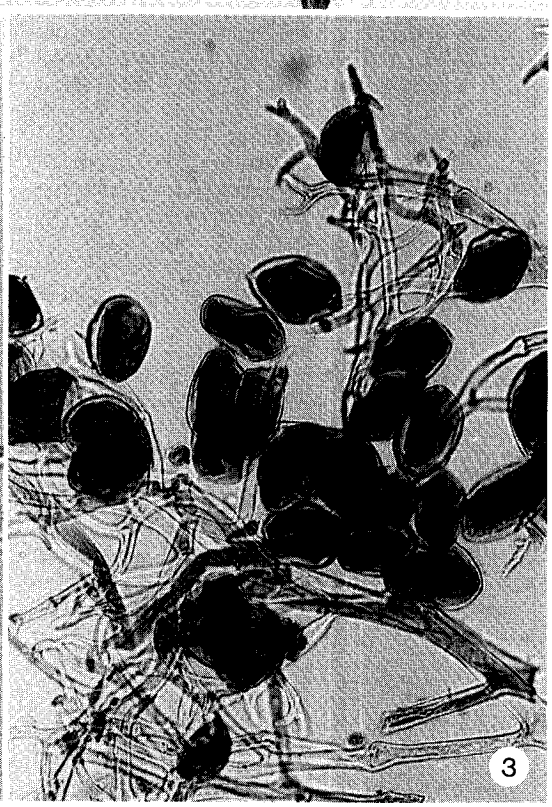


Fig. 1. Chlorotic lesions on the upper leaf surfaces. ×1.4

Fig. 2. Dichotomously branched conidiophore of *P. farinosa* with slightly curved sterigamata (arrowheads). ×770.

Fig. 3. Branches of conidiophore and pyriform conidia of *P. farinosa*. ×520.

Verticillium wilt of irrigated alfalfa in Saskatchewan, 1987-89

B.D. Gossen [1] and G.D. Jesperson [2]

Fields of irrigated alfalfa throughout the southwestern portion of the grainbelt in Saskatchewan were surveyed for verticillium wilt (*Verticillium albo-atrum*) from 1987 to 1989. Plowing down infected crops followed by crop rotation to cereals was associated with the elimination of the disease from irrigation areas at Outlook and Miry Creek, where it had been observed in the early 1980's. However, in the Chesterfield Flats irrigation project, where minimal control efforts had been made, the disease was present in almost every field. Alfalfa crops in two additional irrigation areas near the Alberta border were also infected. Increased frequency of cutting and overhead irrigation were associated with rapid spread of the disease. Spring black stem (*Phoma medicaginis*) was the most prevalent foliar disease in the survey area.

Can. Plant Dis. Surv. 70:2, 129-131, 1990.

Des luzernières irriguées du sud-ouest de la zone de culture de céréales en Saskatchewan ont été examinées à l'égard de la flétrissure verticillienne (*Verticillium albo-atrum*) de 1987 à 1989. L'enfouissement des cultures infectées suivi par le remplacement de la luzerne par des céréales dans la rotation, a permis d'éliminer la maladie des zones d'irrigation de Outlook et de Miry Creek où on l'avait signalée au début des années 1980. Mais dans le cadre du projet d'irrigation de Chesterfield Flats où des efforts de lutte minimums ont été déployés, on pouvait constater la présence de la maladie dans presque tous les champs. Les luzernières de deux zones d'irrigation supplémentaires près de la frontière avec l'Alberta étaient également infectées. L'augmentation de la fréquence de coupe et l'irrigation par aspersion étaient associés avec une propagation rapide de la maladie. La tige noire printanière (*Phoma medicaginis*) était la maladie foliaire la plus fréquente dans la zone d'enquête.

Introduction

Verticillium wilt of alfalfa (*Verticillium albo-atrum* Reinke & Berth.) is a destructive disease in British Columbia, Ontario, Quebec and throughout large areas of the U.S.A. and Europe. In Alberta, it is an important disease in irrigated fields, but is rarely found in dryland fields (Howard and Moskaluk 1988, H.C. Huang, personal communication). The disease was first found in Saskatchewan in 1980 in the irrigation areas near Outlook and along the South Saskatchewan River near the Alberta border (Atkinson 1981, Arny and Grau 1985). In 1981, the disease was still limited to these areas. Growers were advised to plow down infected fields and rotate them out of alfalfa. In 1982, no infected fields were found. Surveys in 1983 and 1984 revealed that the disease was still present in the Chesterfield Flats (west of Leader, near the Alberta border) and Miry Creek irrigation areas (A. Frowd, Saskatchewan Agriculture, unpublished), and at trace levels near Outlook (J.D. Smith, personal communication). Plowing and crop rotation were again recommended for disease control.

The objectives of this study were to determine the extent and severity of verticillium wilt on irrigated alfalfa in the southwestern portion of the province, where it had been observed in the early 1980's, and to assess the effect of eradication procedures on disease incidence, especially in the Chesterfield Flats and Miry Creek areas. Summaries of the survey results from 1987-88 were published previously (Gossen and Jesperson 1988, 1989).

Methods

In 1987, 50 irrigated alfalfa fields were examined from 6 July to 26 August. In 1988, 30 fields were examined from 20 July to 5 August. Drought conditions in many areas in 1988 were so severe that growers were unable to irrigate their fields after the first cut. In these areas, poor growth and severe drought stress precluded assessment of many fields. In 1989, 31 fields were examined from 16 July to 29 September. The survey in 1989 was concentrated on fields along the South Saskatchewan River, in areas where infected fields had previously been identified.

Alfalfa crops were examined by walking through the field in a teardrop pattern, and searching for wilting plants. Most of the fields were in the late vegetative or early bud stage when surveyed. Field diagnosis of verticillium wilt, based on symptoms, was confirmed by isolation of the pathogen: stem pieces were surface-sterilized, split, and incubated on water agar on a laboratory bench for at least 14 days. No pathogenicity tests were made on the isolates. Identification of foliar diseases was based primarily on symptoms in the field, but occasionally isolations were made to confirm the diagnoses.

Results and Discussion

In 1987, verticillium wilt was confirmed from only two fields; one in the Chesterfield Flats irrigation project west of Leader (Figure 1), and the other less than 15 km upriver (west) from the first, in a separate irrigation area near Estuary. In 1988,

[1] *Agriculture Canada Research Station, 107 Science Crescent, Saskatoon, Saskatchewan S7N 0X2.*

[2] *Saskatchewan Agriculture and Food, Soils and Crops Branch, 3085 Albert Street, Regina, Saskatchewan S4S 0B1.*

Accepted for publication February 13, 1990.

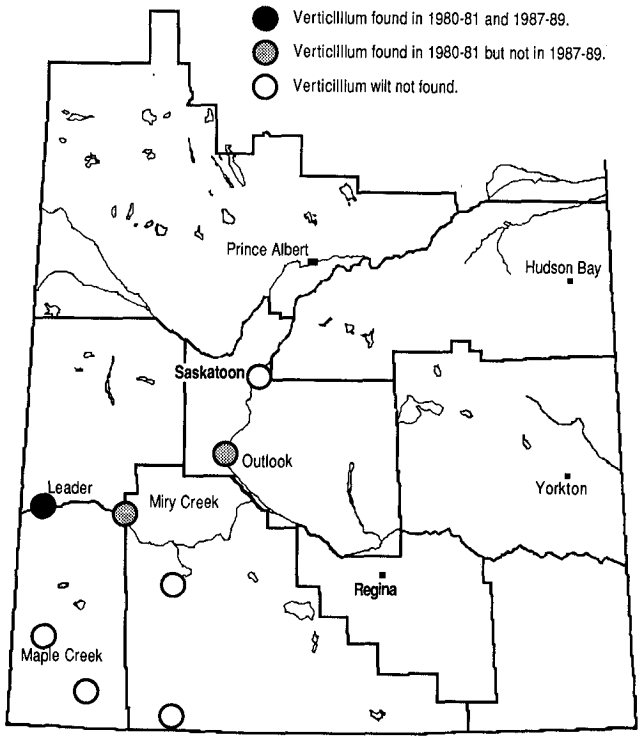


Fig. 1. Distribution of verticillium wilt of alfalfa in Saskatchewan 1980-89.

two additional infected fields were identified; one near Estuary, and another east of Leader. In 1989, five new fields with verticillium wilt were identified; four in the Chesterfield Flats irrigation area, and a fifth at Estuary.

Severity of foliar diseases in the survey area was generally very low over the three years of the study. Spring black stem (*Phoma medicaginis* Malbr. & Roum. var. *medicaginis* Boerema) was the predominant leaf pathogen in southern areas, and was prevalent in central regions as well. However, late in the 1988 season, common leaf spot (*Pseudopeziza medicaginis* (Lib.) Sacc.) developed rapidly to moderate levels (25% of the leaf area affected) in the Outlook area. Downy mildew (*Peronospora trifoliorum* de Bary.) infection was noted at low levels in many locations in 1988-89. A *Colletotrichum* sp. was frequently isolated from the stems of wilted plants from several fields in the Leader and Miry Creek regions in 1988-89.

Although at least some of the fields at Chesterfield Flats had been infected with verticillium wilt for a number of years, disease incidence and severity were low. The stands at Chesterfield Flats were irrigated using gravity-dikes, and were generally old and weedy. They were harvested on a two-cut system. In contrast, in the three infected fields at Estuary and Leader, the disease was observed to spread rapidly, resulting in severe infection. These stands were irrigated with overhead sprinkler systems, and were relatively young, highly productive and aggressively managed (3-4 cuts per year). Both frequency of cutting and irrigation type may have influenced the rate of disease spread, resulting in the differences observed between Chesterfield Flats and the other sites. Each hay cut produces wounds through which the fungus can enter the plant, and spores are inoculated onto the wounds via the cutting blade (Isaac 1957, Christen and Peaden 1982). Therefore, increasing cutting frequency would be expected to increase the rate of disease spread over the course of a season. Also, overhead irrigation results in spores being rain-splashed from infected plants to healthy ones, and produces, temporarily, a moist microclimate within the canopy which is conducive to spore germination and infection. Gravity-dike irrigation does not result in wetting of leaves in the middle and upper portions of the canopy, and so should be less conducive to disease spread.

Discussions with growers revealed that in the Miry Creek irrigation district, wilt-infected fields had been plowed down and rotated to cereals for two years in an effort to eradicate the disease. No verticillium wilt was observed in this area in 1987-89. Similar results were achieved in the Outlook area. In contrast, the age of the stands (some as much as 25 years old) indicated that growers at Chesterfield Flats had not plowed down infected fields. As a result, almost all of the stands in the area were infected in 1989. We conclude that eradication of verticillium wilt in small irrigation areas in Saskatchewan is possible if all the growers in an area are made aware of the problem and are willing to co-operate in an eradication program. Eradication may be more easily accomplished in Saskatchewan than in other areas for two reasons. Firstly, disease spread clearly is restricted by the semi-arid environment, since verticillium wilt occurs almost exclusively on irrigated, and not dryland sites. Secondly, many irrigation areas in the region along the Alberta border are small and relatively isolated, and the potential for introduction or reintroduction of the pathogen from adjacent areas is limited.

All of the owners of affected fields in the Chesterfield Flats irrigation area were contacted in 1989 and advised of the disease problem in their fields. Based on their stated intentions, all of the infected crops will be turned under within the next two years. Growers at the other sites were contacted immediately when infections were diagnosed, and eradication is planned or has already been initiated.

Infected seed is the most probable source of verticillium infection in the fields in southwestern Saskatchewan. Growers with infected crops at Estuary and Leader had all obtained their seed from southern Alberta, where the disease is endemic under irrigated conditions. However, transmission of the disease via contaminated plant material, machinery, insects or irrigation water (summarized in Howard 1985) cannot be ruled out. Growers in this region are being advised to purchase only seed which is certified to be verticillium-free.

Addendum while in press:

Symptoms of *Verticillium albo-atrum* infection were noted in two alfalfa fields in the Miry Creek irrigation area in late June, 1990. The identity of the pathogen was confirmed by isolation.

Acknowledgements

This work was supported in part by a grant from the Agriculture Development Fund of Saskatchewan Agriculture and Food.

Literature cited

1. Atkinson, T.G. 1981. Verticillium wilt of alfalfa: challenge and opportunity. Can. J. Plant Pathol. 3:266-272.
2. Arny, D.C. and C.R. Grau. 1985. Importance of verticillium wilt of alfalfa in North America. Can. J. Plant Pathol. 7:187-190.
3. Christen, A.A. and R.N. Peaden. 1982. Relative importance of sources of verticillium wilt infestation in alfalfa. Phytopathology 72:960 (Abstr.).
4. Gossen, B.D. and G. Jesperson. 1988. Survey of irrigated alfalfa in Saskatchewan. Can. Plant Dis. Surv. 68:61.
5. Gossen, B.D. and G. Jesperson. 1988. Verticillium wilt and foliar diseases of irrigated alfalfa in Saskatchewan in 1988. Can. Plant Dis. Surv. 69:50.
6. Howard, R.J. 1985. Local and long-distance spread of *Verticillium* species causing wilt in alfalfa. Can. J. Plant Pathol. 7:199-202.
7. Howard, R.J. and E.R. Moskaluk. 1988. Survey for verticillium wilt of alfalfa. Can. Plant Dis. Surv. 69:48.
8. Isaac, I. 1957. Wilt of lucerne caused by species of *Verticillium*. Ann. Appl. Biol. 45:550-558.

Diseases of *Rubus parviflorus* in British Columbia

R.E. Wall and S.F. Shamoun [1]

The native shrub, thimbleberry (*Rubus parviflorus* Nutt.) was examined for disease throughout its range in southern British Columbia. Previously unreported fungi were *Discosia* sp., and *Seimatosporium* sp. associated with leaf spots and *Cylindrocarpon destructans* (Zinf.) Scholten, *Naematoloma fasciculare* (Huds. ex Fr.) Kar., *Resinicium bicolor* (Alb. & Schw. ex Fr.) Parm. and *Verticillium* sp. associated with root rots. Pathogenicity tests were conducted with several of the fungi collected.

Can. Plant Dis. Surv. 70:2, 133-135, 1990.

La ronce parviflore (*Rubus parviflorus* Nutt.) a été examinée à l'égard de la présence de maladies dans toute sa zone de distribution du sud de la Colombie-Britannique. Les champignons non déjà signalés sont *Discosia* sp., et *Seimatosporium* sp. associés aux taches des feuilles et *Cylindrocarpon destructans* (Zinf.) Scholten, *Naematoloma fasciculare* (Huds. ex Fr.) Kar., *Resinicium bicolor* (Alb. & Schw. ex Fr.) Parm. et *Verticillium* sp. associés aux pourritures des racines. Les auteurs ont effectué des essais de pathogénicité avec plusieurs des champignons prélevés.

Introduction

Wild shrubs are important reservoirs of pathogens of horticultural crops (Credi et al. 1986). Also, with current concern over the use of chemical herbicides on forest lands, agents that cause diseases of shrubs are potential biological controls. Thimbleberry (*Rubus parviflorus* Nutt.) is an example of such a shrub. It is a noticeable feature of roadsides as well as a frequently dominant weed on cut and burned-over forest land throughout British Columbia (Haeussler and Coates 1986). Consequently, attention has recently been turned to indigenous diseases of this and other non-commercial species which compete with conifers on forest land.

Thimbleberry is included in most plant disease indices (Anonymous 1960; Conners 1967; Farr et al. 1989; Ginns 1986; Lowe 1977; Shaw 1973; Toms 1964), most collections being recorded from the Rocky Mountain and Pacific regions where thimbleberry is most commonly found. In addition, Greene (1957) lists three fungal diseases of thimbleberry in the Great Lakes region, where this shrub occurs in isolated pockets. Virus diseases have been studied by Credi et al. (1986), Stace-Smith (1958), and Stace-Smith and Shier (1988).

Searches to detect and collect diseases of thimbleberry have been conducted since 1987 by the authors and by Forest Insect and Disease Survey staff throughout its range in southern B.C.

Materials and methods

Diseased tissues were examined and cultured within 3 days after collecting. Cultures were made by placing small pieces of tissue from surface sterilized plant material on corn-meal, malt, potato dextrose, and V-8 juice agars or by germinating spores from associated fruiting bodies on agar media. Cultures were incubated in the dark at 20°C and stored at 10°C when fully developed. To induce sporulation, some cultures were exposed to 4 – 5 days of irradiation with near ultraviolet light (365 nm wavelength) during the early stages of growth (Leach 1962).

Inoculations were performed in triplicate in the greenhouse (15-25°C, 16 hr daylength) on potted plants that had been grown from rhizome cuttings. Inoculations with foliar disease fungi were accomplished by spraying to runoff with aqueous suspensions of spores (one million spores/ml) and placing the plants in a dew chamber at 20°C for 24-48 hr. Basidiomycetous root fungi were cultured on sterilized segments of thimbleberry rhizomes and placed in the soil adjacent to the root mass. Root inhabiting fungi imperfecti were applied as spore suspensions to the soil at the base of the plant.

Inoculation results were evaluated at weekly intervals and disease symptoms scored by the following rating system: 0 = healthy, 1 = occasional leaf spots, 2 = noticeable leaf spots or foliar browning, 3 = about 50% of the leaf area damaged, 4 = plant wilted or 60-90% of leaf area lost due to disease, 5 = plants dead.

Results and discussion

Disease collections and inoculation results are summarized in Table 1. Most diseases were collected between July 15 and the time of autumn leaf senescence, but the most common disease, septoria leaf spot, could be found as early as the second week of June. Septoria leaf spot was found in open as well as shaded locations and disease severity appeared to differ little between these two habitats. This disease also was collected on salmonberry (*Rubus spectabilis* Pursh.) and to a lesser extent on raspberry (*Rubus idaeus* L.). With the inoculation method used, *Septoria* was consistently pathogenic. However, inoculation trials with other fungi have not been repeated sufficiently under different conditions to either confirm or refute their pathogenicity (Table 1). *Botrytis cinerea* was associated with fruit rot and blighting of associated pedicels and leaves in mid- to late summer. *Gnomonia* sp. was associated with a circular to oval brown necrotic spot which was prevalent in

[1] *Pacific Forestry Centre, Forestry Canada, 506 West Burnside Rd., Victoria, B.C. V8Z 1M5.*

Accepted for publication April 3, 1990.

Table 1. Collections of thimbleberry diseases in southern British Columbia.

Disease organism isolated	Location#	Number of inoculation trials	Incubation period (weeks)	Maximum disease rating*
Angular leaf spot				
Septoria rubi West.	1,2,4,9,10	11	4	3
Powdery mildew				
Sphaerotheca macularis (Wallr. ex Fr.) Lind.	2,9,10	-	-	-
Leaf rust				
Phragmidium occidentale Arth.	1,2,9	-	-	-
Other foliage diseases				
Alternaria sp.	1,5,8	1	2	1
Botrytis cinerea Pers.	2,5,8,10	1	2	2
Discosia sp.	2	1	2	1
Gnomonia sp.	10	-	-	-
Seimatosporium sp.	7	2	-	0
Root rots				
Cylindrocarpon destructans (Zinf.) Scholten	6,10	2	2	2
Fusarium oxysporum Schlecht.	6,10	2	2	2
Naematoloma fasciculare (Huds. ex Fr.) Kar.	10	1	-	0
Resinicium bicolor (Alb. & Schw. ex Fr.) Parm.	10	1	-	0
Verticillium sp.	6,10	2	-	0

Collections were made near Campbell River (1), Victoria (2), Vancouver (3), Hope (4), Barriere (5), Revelstoke (6), Meadow Creek (7), Nakusp (8), Crawford Bay (9), and Castlegar (10).

* Diseases rated on a 0 - 5 system, 0 = healthy, 1 = occasional leaf spots or other mild foliar discoloration, 2 = readily visible foliar disease, 3 = about 50% of leaf area lost to disease, 4 = 60 - 90% loss of foliage, 5 = plants dead.

1989. Powdery mildew was quite common, especially on the newer foliage and was particularly damaging to thimbleberry in warm greenhouses or growth chambers (day temperatures of 25°C or higher). Phragmidium leaf rust also occurred widely but was not associated with foliar necrosis during the main part of the growing season. No virus or mycoplasma diseases were confirmed, but symptoms suggesting their presence were observed in several locations.

No conspicuous shoot blights have been found on thimbleberry to date. Dieback was common in mid- to late summer in canes which had flowered and borne fruit. Another frequent cause of dieback was the thimbleberry gallmaker (*Diastrophus kinkaidii* Gillette), a hymenopterous insect which causes large stem galls on current year's shoots (Wangberg 1975). Superficial stem lesions were often observed, but they did not appear to affect the xylem and no particular organisms were consistently cultured. *Dasyscyphus* spp. were often found fruiting on dead stems.

Root diseases were collected in late summer and autumn from shrubs which showed signs of decline. *Naematoloma fasciculare* was cultured both from decaying rhizomes and from spores cast from mushrooms found growing around unhealthy looking plants. *Resinicium bicolor* mycelium, recognized by the abundance of stellate cystidia (Nobles 1953), was observed on the surface of root crowns of shrubs with root rot and was readily cultured from adjacent tissues. *Cylindrocarpon destructans*, *Fusarium oxysporum* and *Verticillium* sp. were cultured from discolored feeder roots. *C. destructans* and *F. oxysporum* were isolated in numerous cultures from roots of plants which had recently been treated with the herbicide glyphosate.

The list of thimbleberry diseases is by no means complete. For instance *Phytophthora* spp., to which this shrub is reported to be moderately susceptible (Bristow et al. 1988) were not isolated by our culture methods. None of the shoot blight or canker diseases commonly reported on commercial *Rubus* cultivars were collected, although a *Gnomonia* sp. was

cultured from leaf spots. Some fungi previously reported on thimbleberry but not collected in this study were *Armillaria* spp. (Toms 1964), *Coleroa chaetomium* (Kunze) Rab. (Barr 1953), *Peronospora rubi* Rahb. and Schroet. (Toms 1964) and *Pucciniastrum arcticum* Tranz. (Lowe 1977). Shaw (1973) lists many other fungi, not all of which are pathogens. It is likely that most of the reported diseases occur sporadically and cause very little damage. Thimbleberry, like other indigenous species has co-evolved with most of the pathogens to which it is normally exposed and is therefore resistant or otherwise adapted to escape infection.

Acknowledgements

We wish to thank Forest Insect and Disease Survey staff of the Pacific Forestry Centre as well as C. Dorworth for several collections and B. Callan, D. Chu, A. Funk and L. Sigler for identification of cultures.

Literature cited

1. Anonymous. 1960. Index of plant diseases of the United States. U.S.D.A. Agric. Handb. No. 165. 531 p.
2. Barr, M.E. 1953. Pyrenomycetes of British Columbia. Can. J. Bot. 31:810-830.
3. Bristow, P.R., H.A. Daubeny, T.M. Sjulin, H.S. Pepin, R. Nestby and G.E. Windom. 1988. Evaluation of *Rubus* germplasm for reaction to root rot caused by *Phytophthora erythroseptica*. J. Am. Soc. Hort. Sci. 113:588-591.
4. Conners, I.L. 1967. An annotated index of plant diseases in Canada. Can. Dept. Agric. Publ. 1251. 381 p.
5. Credi, R., J.L. Shier and R. Stace-Smith. 1986. Occurrence of raspberry bushy dwarf virus in native thimbleberry in British Columbia. Acta Hort. 186:17-21.
6. Farr, D.F., G.F. Bills, G.P. Chamuris and A.Y. Rossman. 1989. Fungi on plants and plant products in the United States. Am. Phytopath. Soc. Press. St. Paul, Minn. 1252 p.
7. Ginns, J.H. 1986. Compendium of plant disease and decay fungi in Canada, 1960 – 1980. Agric. Can. Publ. 1813. 416 p.
8. Greene, H.C. 1957. The fungi parasitic on plants in Wisconsin. Litho Productions, Inc. Madison, WI. 228 p.
9. Haeussler, S. and D. Coates. 1986. Autoecological characteristics of selected species that compete with conifers in British Columbia: a literature review. Canada – B.C. For. Res. Dev. Agreement Rep. # 001. 180 p.
10. Leach, C.M. 1962. The sporulation of diverse species of fungi under near-ultraviolet radiation. Can. J. Bot. 40:151-161.
11. Lowe, D.P. 1977 (rev. 1989). Check list and host index of bacteria, fungi and mistletoes of British Columbia. Can. For. Serv. Inf. Rep. BC-X-32. 315 p.
12. Nobles, M.K. 1953. Studies on wood-inhabiting hymenomycetes. I. *Odontia bicolor*. Can. J. Bot. 31:745-749.
13. Shaw, C.G. 1973. Host fungus index for the Pacific Northwest. I. Hosts. Wash. Agric. Exp. Stn. Bull. 765. 121 p.
14. Stace-Smith, R. 1958. Studies on *Rubus* virus diseases in British Columbia. V. Thimbleberry ring spot. Can. J. Bot. 36:385-388.
15. Stace-Smith, R. and J.L. Shier. 1988. Some properties of apple mosaic virus isolated from thimbleberry in British Columbia. Proc. 5th Int. Symp. Small Fruit Virus Diseases.
16. Toms, H.N.W. 1964. Plant diseases of southern British Columbia. Can. Plant Dis. Surv. 44:143-225.
17. Wangberg, J.K. 1975. Biology of the thimbleberry gallmaker, *Diastrophus kinkaidii*. Pan-Pac. Entomol. 51:39-48.

Author Index to Volume 70

Instructions to authors

Articles and brief notes are published in English or French. Manuscripts (original and one copy) and all correspondence should be addressed to Dr. H.S. Krehm, Research Program Service, Research Branch, Agriculture Canada, Ottawa, Ontario K1A 0C6.

Manuscripts should be concise and consistent in style, spelling, and use of abbreviations. They should be typed, double spaced throughout, on line-numbered paper. All pages should be numbered, including those containing abstract, tables, and legends. For general format and style, refer to recent issues of the *Survey* and to *CBE Style Manual* 3rd ed. 1972. American Institute of Biological Sciences, Washington, D.C. Whenever possible, numerical data should be in metric units (SI) or metric equivalents should be included. Square brackets may be used to enclose the scientific name of a pathogen, following the common name of a disease, to denote cause.

Titles should be concise and informative providing, with the Abstract, the key words most useful for indexing and information retrieval.

Abstracts of no more than 200 words, in both English and French, if possible, should accompany each article.

Figures should be planned to fit, after reduction, one column (maximum 84 × 241 mm) or two columns (maximum 175 × 241 mm), and should be trimmed or marked with crop marks to show only essential features. Figures grouped in a plate should be butt-mounted with no space between them. A duplicate set of unmounted photographs and line drawings is required. Figures should be identified by number, author's name, and abbreviated legend.

Tables should be numbered using arabic numerals and have a concise title; they should not contain vertical rules; footnotes should be identified by reference marks (* † § # ¶ ** ††) particularly when referring to numbers.

Literature cited should be listed alphabetically in the form appearing in current issues; either the number system or the name-and-year system may be used. For the abbreviated form of titles of periodicals refer to the most recent issue of *Biosis List of Serials* published by Biosciences Information Service of Biological Abstracts or to the *NCPTWA Word Abbreviation List*, American National Standards Institute.

Recommandations aux auteurs

Les articles et les communiqués sont publiés en anglais ou en français. Les manuscrits (l'original et une copie) et toute la correspondance qui s'y rapporte doivent être envoyés à D[r] H.S. Krehm, Service des programmes de recherche, Direction de la recherche, ministère de l'Agriculture du Canada, Ottawa, (Ontario) K1A 0C6.

Les manuscrits doivent être concis et faire preuve de suite dans le style, l'orthographe et l'emploi des abréviations. Ils doivent être dactylographiés à double interligne, de préférence sur des feuilles à lignes numérotées. Toutes les pages doivent être numérotées y compris celles portant le résumé, les tableaux et les légendes. Pour plus de renseignements sur le format des feuilles et le style, prière de consulter nos dernières publications et le *CBE Style Manual* (3e ed. 1972) de l'American Institute of Biological Sciences, Washington (DC). Dans la mesure du possible, les données numériques doivent être exprimées en unités métriques, (SI) ou être suivies de leur équivalent métrique. L'emploi de crochets est autorisé pour l'identification du nom scientifique d'un micro-organisme pathogène après le nom commun de la maladie dont il est l'agent causal.

Les titres doivent être courts et révélateurs en contenant, avec le résumé, les mots clés les plus utiles pour le classement et l'extraction de l'information.

Chaque article doit être accompagné d'un *résumé* d'au plus 200 mots en anglais et en français, si possible.

Les figures doivent pouvoir, après réduction, remplir une colonne (maximum 84 × 241 mm) ou deux colonnes (maximum 175 × 241 mm) et devraient être taillées ou montrer les parties essentielles à garder. Les figures groupées sur une même planche doivent être montées côte à côte, sans intervalle. L'article doit être accompagné d'un double des photographies non montées et des graphiques. Les figures doivent être numérotées, porter le nom de l'auteur et une légende abrégée.

Les tableaux doivent être numérotés en chiffres arabes et avoir un titre concis. Ils ne devraient pas avoir de lignes verticales. Les renvois doivent être identifiés par un signe typographique particulier (* † § # ¶ ** ††) surtout lorsqu'il s'agit de nombres.

Les références bibliographiques devraient être citées par ordre alphabétique comme dans les livraisons courantes. On peut utiliser le système de numération ou le système nom-et-année. Pour l'abrégé du titre des périodiques, on suivra l'édition la plus récente de *Biosis List of Serials* publiée par les Biosciences Information Services de Biological Abstracts ou la *NCPTWA Word Abbreviation List* et l'American National Standards Institute, Standards Committee Z39.