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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.

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 immediat pour les phytopathologistes.

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ERRATUM
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To dispel the impression that a new type of downy mildew symptom per se is being reported, the title of the article should be changed to:

Pour dissiper l'impression qu'un nouveau type de symptôme du mildiou est rapporté, le titre de l'article devrait lire:

"A possible systemic downy mildew syndrome on buckwheat seedlings"

Root rot and wilt of mung bean in Ontario

T.R. Anderson[1]

Root rot and wilt of mung bean caused severed losses in a seed increase field and a nursery in 1979 and 1980. Root rot was prevalent during the early growing season on clay soil. Wilt occurred during the flowering and late pod filling stages on clay and sandy soil. *Rhizoctonia solani, Thielaviopsis basicola, Fusarium oxysporum* and a *Fusarium* sp. isolated from diseased plants were evaluated for pathogenicity in the greenhouse. *R. solani* and *T. basicola* caused distinct lesions on roots and lower stems similar to those observed in the field. *F. oxysporum* and *Fusarium* sp. were non-pathogenic in greenhouse experiments.

Can. Plant Dis. Surv. 65:1, 3-5, 1985.

La pourriture des racines et la flétrissure du haricot mungo ont causé des pertes importantes dans un champ de multiplication de semence et une pépinière en 1979 et 1980. La pourriture des racines est répandue au début de la saison de croissance en sol argileux tandis que la flétrissure apparait lors de la floraison et du remplissage des gousses sur les sols argileux et sableux. La pathogénicité de *Rhizoctonia solani, Thielaviopsis basicola, Fusarium oxysporum* et *Fusarium sp.* isolés à partir de plants malades, a été évaluée en serre. *R. solani* et *T. basicola* causent des lésions distinctes sur les racines et dans le bas des tiges, semblables à celles observées au champ. *F. oxysporum* et *Fusarium sp.* sont non-pathogènes dans des expériences en serre.

During August 1979, two fields of diseased mung bean [*Vigna radiata* (L.) Wilczek] were observed at King Grain Limited, Chatham, Ontario. One field consisted of clay loam and had been planted previously with soybean [*Glycine max* (L.) Merr.] and the other field consisted of sandy loam used as a breeding nursery. In the clay loam field, approximately 80% of the plants in a seed increase block of mung beans (cv. VC1089) were dead or had symptoms of wilt. Soybeans (cv. Premier) in adjacent border rows were healthy. In the sandy loam field, 90-100% of plants of certain lines were killed while others showed symptoms of wilt but no dead plants. White beans (*Phaseolus vulgaris* L.) and soybeans in border rows had no foliar symptoms of disease but roots had black or red lesions.

The root rot and wilt disease of mung bean had caused extensive plant loss and appeared to be a limiting factor in the commercial production of mung beans. This note reports the etiology of these diseases.

Materials and Methods

Wilted plants were collected in the field and transported to the laboratory in plastic bags. Leaf, stem and root segments were surface sterilized in a 1.25% sodium hypochlorite solution for 1-2 min and plated on potato dextrose agar (PDA). Clay soil collected from the vicinity of diseased plants was placed in 25 cm pots in a greenhouse at the Harrow Research Station. Three replicate pots were each planted with 25 seeds of one of the soybean cvs. Harcor or Evans or one of the mung bean cvs. VC1089, Kawa or M333. Plant stand and severity of root damage were determined 8 weeks from planting. The percentage or root surface that was discoloured or necrotic was assessed on a scale of 1-4, as follows: 1 = 0-10%, 2 = 11-25%, 3 = 26-50%, 4 = 51-100%. Sections of the lower stem and upper roots of 5 plants per replicate were surface sterilized in 1.25% sodium hypochlorite and plated on PDA. Five sections of root per plant were placed on carrot discs in a moist chamber to determine the incidence of *Thielaviopsis basicola*.

In 1980, isolations were made from field plots of VC1089 at three dates during the growing season. Ten plants from each of 3 replicates were selected randomly at 7, 9 and 12 weeks after planting. Three sections 5 mm in length from each stem were surface sterilized and plated on PDA.

The pathogenicity of isolates of *Fusarium* spp., *Rhizoctonia solani* and *T. basicola* were tested on soybean and mung bean in the greenhouse in 10 cm pots. Inoculum of *F. oxysporum* and an unidentified *Fusarium* sp. isolated from diseased mung bean roots was increased in sand-corn meal medium (SCM), 196 g sand 4 g corn meal 30 ml H_2O) for 4 weeks at 20-25°C. The inoculum and medium was mixed with steamed greenhouse potting mix at 0, 2.5, 5.0 and 10% (v/v). Ten seed of each cultivar were planted per pot. Treatments were replicated five times. Soybeans were harvested 8 weeks after planting and mung beans were harvested 15 weeks from planting.

Isolates of *R. solani* from soybean and mungbean were cultured in CMS medium as described previously and mixed with greenhouse soil to obtain concentrations of 5 and 10% (v/v). Ten seeds were planted per pot and treatments were replicated five times. Observations were made 10 weeks from planting.

Inoculum of *T. basicola* from mung bean was prepared by washing the chlamydospores from culture plates of 20% V_8 juice agar and collecting them on a 22 μm mesh screen. Chlamydospores were mixed with soil to obtain inoculum densities of 1×10^2, 1×10^3 and 1×10^4 chlamydospore chains/g dry soil. Pots were planted with 10 seeds of VC1089, Kawa or M333. Treatments were replicated five times. Plants were harvested and examined for symptoms of root rot 8 weeks after planting.

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Table 1. Stand, disease rating and incidence of fungi isolated from soybean (cvs. Harcor and Evans) and mung bean (cvs. VC1089, M333 and Kawa) growing in field soil in the greenhouse.

Cultivar	Stand %	Disease rating*	Incidence of isolation (%)		
			F. oxysporum	*R. solani*	*T. basicola*
Harcor	69a**	1.9 ± 1.0	80	13	67
Evans	67a	1.9 ± 1.0	47	0	60
VC1089	41b	2.8 ± 1.0	67	7	33
M333	40b	2.6 ± 1.2	53	0	47
Kawa	24b	3.3 ± 0.6	40	20	60

*Root rot rating based on a scale of 1-4 where 1 = 0-10%, 2 = 11-25%, 3 = 26-50% and 4 = 51-100% of the root surface discoloured.
**Means followed by the same letter do not differ significantly according to Duncan's Multiple Range Test (P = 0.05).
Note: Other fungi isolated included species of *Alternaria, Chaetomium, Fusarium, Phomopsis* and unidentified fungi.

Results and Discussion

Field observations

Three weeks after emergence, a high percentage of mung bean plants growing in clay soil showed symptoms of root rot. Leaves of infected plants wilted and stems became brown. Infected plants remained upright and red lesions in the cortex were evident on roots and stems near the soil line. Lower roots were completely rotted.

External discoloration or deep cankers were not as evident on plants observed later in the growing season. Diseased plants at a mid to late pod filling stage of growth showed symptoms of water stress. Lower petioles and leaves were collapsed. As the disease progressed, lower and upper leaves developed interveinal necrosis and eventually entire leaves became necrotic. Stems of affected plants appeared healthy although a salmon pink discolouration of the xylem was evident after stems were split longitudinally. The discoloured tissue extended 10-25 cm from the crown and lateral roots into the third or fourth internode and occasionally into the leaf petioles. A white or pink mycelium was frequently observed in the central cortex at the base of dead plants. Mycelium within xylem vessels was observed under the microscope.

Fungi isolated from diseased plants collected in the field consisted of *F. oxysporum* and a brown unidentified *Fusarium* sp., *R. solani* and species of *Phomopsis, Alternaria* and *Chaetomium*. Chlamydospores of *T. basicola* in root lesions were observed under the microscope.

Greenhouse experiments

Organisms isolated from soybean and mung bean growing in field soil transported to the greenhouse were similar to those isolated from plants grown in the field (Table 1). Plant stands of mung bean as a percentage of stands in check pots containing steamed field soil were significantly lower than soybean stands. The root rot rating of mung bean was higher than soybean. Incidence of *F. oxysporum, R. solani* and *T. basicola* isolated from soybean and mung bean varied considerably and the incidence of isolation did not differ significantly (P = 0.05) among hosts. *F. oxysporum* was isolated frequently from mung bean plants growing in clay soil at 7, 9 and 12 weeks from planting (Table 2). Other fungi including *Fusarium* sp, *Phomopsis* sp., *Pythium* sp. and *R. solani* were isolated significantly less frequency (P = 0.05).

Soil infestation with isolates of *F. oxysporum* and *Fusarium* sp. in greenhouse experiments did not result in infection or reduced stands of soybean and mung bean. Additional trials in which roots were dipped in a suspension of spores and transplanted into greenhouse soil failed to demonstrate the pathogenicity of the two species of *Fusarium*. Although the symptoms of mung bean wilt observed in the field and greenhouse resembled wilt induced by *F. oxysporum*, evidence of the involvement of this fungus was inconclusive.

Plant stands of mung beans were significantly less (P = 0.05) than soybeans after 10 weeks in soil infested with the mung bean isolate of *R. solani* (Table 3). Differences among cultivars

Table 2. Fungi isolated from mung bean (cv. VC1089) stems growing in field soil at 7, 9 and 12 weeks from planting.

Organism	Incidence in plant segments (%)		
	7	9	12
Alternaria sp.	51 a*	50 a	32 bc
Fusarium oxysporum	50 a	49 a	68 a
Fusarium sp.	12 b	33 b	34 b
Phomopsis sp.	8 bc	23 bc	17 cd
Pythium sp.	0 c	6 d	0 d
Rhizoctonia solani	0 c	2 d	6 d
Unidentified fungi	11 b	13 cd	1 d

* Means of 3 replicates, 10 plants/replicate, 3 segments/plant. Means followed by the same letter within a column do not differ significantly according to Duncan's Multiple Range Test (P = 0.05).

were less obvious but significantly different in pots containing the soybean isolate. Inoculum concentration did not significantly affect plant stands. Red cankers at or near the soil line were evident on most plants in the experiment and resembled those observed in the field 4 weeks after emergence. Plant losses occurred during emergence in pots in the greenhouse. Symptoms of wilt on older plants were rare. The vascular tissue of infected plants did not have the pink discoloration evident in wilted plants in the field during flowering and pod fill.

Plant stands of mung bean cultivars VC1089, Kawa and M333 were not affected by *T. basicola* infested soil (Table 4). Root rot rating increased significantly (P = 0.05) with increasing inoculum concentration. Significant differences in ratings among cultivars occurred at an inoculum concentration of 1×10^2 chlamydospore chains/g of dry soil but not at other concentrations. *T. basicola* caused brown to black lesions on tap and lateral roots but did not cause discolouration of the vascular tissue in the stem region. Wilt was not observed.

The pathogenicity of *R. solani* and *T. basicola* to mung bean was demonstrated and it is probable that these fungi, especially *R. solani* contribute to early season plant losses. The soybean cultivar Evans was more susceptible than Harcor when inoculated with the soybean isolate of *R. solani* but the mung bean isolate appeared less pathogenic on both soybean cultivars.

Table 3. Plant stand of soybean (cvs. Harcor and Evans) and mung bean (cvs. VC1089, Kawa and M333) after 10 weeks in soil infested with isolates of *Rhizoctonia solani* from soybean and mung bean.

	Isolate and inoculum concentration (v/v)			
	Soybean isolate		Mung bean isolate	
Cultivar	5%	10%	5%	10%
Harcor	81 a*	86 a	88 a	98 a
Evans	42 bc	44 b	84 a	80 a
VC1089	34 c	11 c	30 b	23 b
Kawa	47 bc	18 bc	41 b	41 b
M333	65 ab	20 bc	10 c	33 b

* Plant stand presented as a percentage of control. Means followed by the same letter within a column do not differ significantly according to Duncan's Multiple Range Test (P = 0.05).

The cause of late season wilt of mung bean was not determined but the disease symptoms and presence of *F. oxysporum* within infected tissue suggest this organism was responsible but additional research is required to confirm these observations.

Table 4. Effect of inoculum concentration of *Thielaviopsis basicola* on stand and root rot of 3 mung bean cultivars 8 weeks after planting.

	Inoculum concentration (spores/g soil)							
	0		1×10^2		1×10^3		1×10^4	
Cultivars	S*	R**	S	R	S	R	S	R
VC1089	8.6	(0)	8.4	(3.4)ab***	7.2	(4.1)	8.2	(4.9)
Kawa	7.5	(0)	8.0	(3.9)a	8.0	(3.7)	7.5	(4.6)
M333	8	(0)	8.8	(2.8) b	8.4	(4.2)	8.4	(4.8)

* Plant stand means of 5 replicate pots, 10 seed/pot.

** Root-rot rating (0-6) as follows: 0 = no lesion, 1 = lesion present but not coalescing to girdle the tap root, 2 = a girdling lesion 1-5 mm long, 3 = a girdling lesion 6-20 mm long, 4 = a girdling lesion 21-40 mm long, 5 = a girdling lesion 41-60 mm long, 6 = a girdling lesion 60 mm long.

*** Means followed by the same letter within a column do not differ significantly according to Duncan's Multiple Range Test (P = 0.05).

Sclerotinia minor as the cause of lettuce drop in southwestern Ontario

W.R. Jarvis[1]

Sclerotinia minor is recorded as a cause of lettuce drop in southwestern Ontario, where it occurred together with *S. sclerotiorum* and *Botrytis cinerea*

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Sclerotinia minor est mentionné comme la cause de l'affaissement sclérotique de la laitue dans le sud-ouest de l'Ontario, où on le retrouve accompagné de *S. sclerotiorum* et de *Botrytis cinerea.*

Sclerotinia minor Jagger as such has apparently not been previously recorded as a pathogen of lettuce in Canada but during the summer of 1984, 3 field crops of leaf and 2 of butterhead lettuce in Essex County were found to have up to 50% of heads affected by lettuce drop. In most cases, small aggregated sclerotia on a white mycelium indicated the presence of *S. minor.* Other lettuce heads nearby had the large separate sclerotia typical of *S. sclerotiorum* (Lib.) de Bary, while others had abundant conidiophores of *Botrytis cinerea* Pers.: Nocca & Balbis. In all cases, the gross symptoms were very similar; wilting and death of the outer leaves and a general flaccidity and loss of brightness in colour of the head. Occasionally, *B. cinerea* occurred on the same heads as either *S. minor* or *S. sclerotiorum.* Exiccati of *S. minor* on lettuce were deposited at DAOM.

The three organisms were readily isolated and produced typical sclerotia on potato dextrose agar, and in the case of *B. cinerea,* typical conidia as well. While *S. sclerotiorum* is well-known from British Columbia to Newfoundland, and so reported by Conners (1), *S. minor* is known in the Canadian literature by one of its synonyms, *S. sativa,* (2), and from herbarium material (3). Both species are plurivorous and lettuce is a common host (3).

Acknowledgement

I thank Miss Barbara Jaques for field surveys.

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Successful management of potato spindle tuber viroid in seed potato crop

R.P. Singh[1] and C.F. Crowley[2]

An analysis of field inspection data over a period of 15 years (1969-1983) of New Brunswick seed potato crop showed that the incidence of potato spindle tuber viroid (PSTV) had decreased to the point where it could not be detected by visual observation. This eradication of PSTV in the seed potato crop could be attributed to higher standards or stricter regulations in seed certification programs, use of virus-free seed multiplied at Elite seed farms, enactment of provincial disease eradication acts and strict planting requirements by processing companies in the region.

Can. Plant Dis. Surv. 65:1, 9-10, 1985.

Une analyse des données d'inspection au champ, couvrant une période de 15 ans (1969-1983) de culture de la pomme de terre de semence au Nouveau-Brunswick, a démontré une diminution de l'incidence du viroïde de la filosité des tubercules (PSTV) au point ou il n'est plus détectable par observation visuelle. Cette éradication du PSTV dans la culture des pommes de terre de semence peut être attribuée à des standards plus élevés ou à des règlements plus sévères dans les programmes de certification des semences, à l'emploie d'une semence exempte de virus multipliée sur les fermes de semence Élite, à la promulgation de lois provinciales sur l'éradication des maladies et à des exigences de plantation sévères de la part des compagnies de transformation de la région.

Introduction

The potato spindle tuber disease was first reported on the North American continent in New Jersey in 1922 by Martin (7). Martin visited Prince Edward Island, Canada in 1930 and found potato spindle tuber in potato crops growing there (6). Although the disease persisted in Eastern Canada, it was not until 1950 that it reached serious proportions (6). This sudden rise in the incidence of the disease coincided with the planting of large acreages of the new potato variety Sebago (6).

In 1969, leaf and tuber samples from potatoes suspected of having potato spindle tuber viroid (PSTV) were collected from tablestock fields in Eastern Canada and the samples were found to be 92% infected with a mild strain and 8% with a severe strain of PSTV (11). Subsequently, an in depth survey of 80 tablestock fields in New Brunswick revealed an average incidence of 3.8% PSTV among the three major varieties, viz., Kennebec (3.3%), Katahdin (2.5%), and Russet Burbank (4.6%) (12).

Since the last PSTV survey in New Brunswick 15 years ago, many changes have been introduced in the seed certification program and it was of interest to analyze the effect of these changes on the PSTV situation. This paper reports that the PSTV incidence has been reduced to the extent that it has not been observed in the last four years in any seed potato field and discusses the impact of various changes in potato seed production.

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Data collection

Field readings collected over the past fifteen years by potato inspectors of the Food Production and Inspection Branch of Agriculture Canada were used to determine the occurrence of PSTV in seed potato stocks. In this procedure, fields were inspected three times during the growing season and a record of PSTV incidence was made on the basis of visual symptoms. The validity of reading PSTV by symptoms was verified by cross-protection tests using host-indicator plants (11), in which only 38 of 355 samples of suspected PSTV material collected by different inspectors in different provinces could not be confirmed as PSTV infected by indicator tests (11). Both mild and severe strains of PSTV were collected by visual symptoms (11). Thus the field inspection readings were considered accurate for large-scale assessment of PSTV incidence. The PSTV readings made by the inspectors were estimates and ranged from traces to 5%. Irrespective of the amount of PSTV, fields with any PSTV were used to calculate the percentage of fields with PSTV infection. Three types of calculations were made; in one case all cultivars, irrespective of acreage were used, while in the second case only Russet Burbank acreage were used, and in the third case only Kennebec acreage were used to calculate the percentage of fields with PSTV. The selection of Russet Burbank and Kennebec was made to show the differences in symptom expression of the former over the latter, in which PSTV is relatively difficult to visualize (5).

Results

The results (Fig. 1) represent an average of 1988 fields or 10,565.6 hectares per year of seed potato production in New Brunswick. Incidence of PSTV was noted in 5% of the fields containing Russet Burbank in 1969, and decreased steadily until 1975, when it was no longer detectable by visual observation (Fig. 1). However, with the cultivar Kennebec, in which it is relatively difficult to read PSTV, the percentage of infected fields fluctuated from year to year and reached an undetecta-

ble level by 1980. When all cultivars are considered (representing about 30), an intermediate picture between Russet Burbank and Kennebec is visualized (Fig. 1). However, in all cases PSTV detection ceased by 1980 and has not been detected in 1981, 1982, 1983 (Fig. 1) and 1984 (personal communication with potato inspectors).

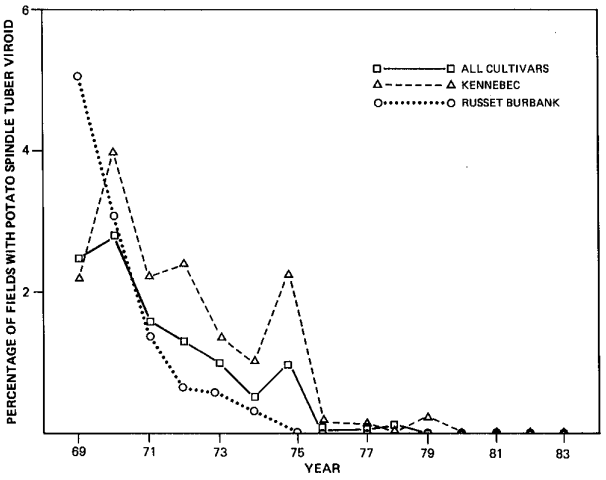

Figure 1. Percentage of fields with potato spindle tuber viroid from 1969 through 1983.

Discussion

From these data it appears that PSTV incidence in New Brunswick seed potatoes has decreased to a level which cannot be detected by visual observation. Because visually-observed samples have been shown to be PSTV infected and represented mild and severe strains (9), this absence of PSTV detection cannot be accounted for by the occurrence of mild strains undetectable by visual observation. Instead, the undetectability of PSTV in seed potatoes in New Brunswick could be attributed to the following changes in seed certification and seed production practices.

Canadian seed improvement programs are constantly striving to upgrade the quality of "elite" seed stocks, which serve as the basis for certified seed production. In an attempt to more effectively control viral, bacterial and viroid diseases, official elite seed farms were established in the mid sixties. These seed farms are staffed with personnel having the expertise to perform technical procedures required for the production of superior disease-free seed.

The second step to improve seed quality was the introduction of seed regulations in the 1970's (1,2), which required that elite seed be grown from virus-free cuttings, plants, or tubers. This regulation further required that all elite I, II, and 10% of the elite III seed, be planted in tuber units and inspected three times during the growing season and that potatoes grown in each category automatically drop a level the following year. These regulations constituting a flush-through system assisted in the reduction of PSTV incidence. In addition to the visual observation, samples of elite I to III were monitored in the laboratory by polyacrylamide gel electrophoresis for PSTV (E.M. Smith - personal communication). Because of the success in the management of PSTV in seed potatoes, the seed regulation was further strengthened in 1980 by allowing "zero tolerance" for PSTV in any seed potato fields at first, second or third inspection (2).

Because of the botanical seed-transmitted nature of PSTV (8), there is a possibility of PSTV introduction to elite seed farms through the introduction of promising and new seedlings or cultivars. This avenue has also been closed to the entry of PSTV by the practice of potato breeders in Canada, who ensure that all cultivars or advanced seedlings are free of PSTV before their multiplication at regional trials or cultivar evaluation at seed farms.

In order to reduce the build-up of virus or viroid diseases, the New Brunswick provincial government passed the Potato Disease Eradication Act (3) which includes PSTV as a prescribed disease. As a prescribed disease, if PSTV is found and confirmed by an inspector, the infected plants must be isolated and disposed of and the farm must undergo thorough disinfection. In addition, this act requires the planting of certified or better grade seed for commercial plantings, which further ensures freedom from PSTV build-up. Besides the provincial government's regulations, the major potato processing company in the region has made it compulsory to plant Foundation class seed for processing purposes (4).

Thus, as stated earlier (9, 10) potato spindle tuber viroid in Canada has become rare compared to the late sixties (12). Although it has been known in various countries (9) for a long time, there has been no report of serious economic losses anywhere in the world in recent years. PSTV should be treated as a minor disease which can be kept under control by proper seed certification and seed production programs.

Acknowledgement

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Colletotrichum acutatum, a new pathogen on western hemlock seedlings in British Columbia

J.C. Hopkins, W. Lock, and A. Funk[1]

Colletotrichum acutatum was associated with terminal shoot and branch tip necrosis of container-grown western hemlock seedlings in a greenhouse near Aldergrove British Columbia. This is a new host and distribution record. Infected seedlings were outplanted to forest sites on Vancouver Island before the pathogen identity was established. Surveys of these sites for the next two years failed to detect any new infections. The damage potential of *C. acutatum* in B.C. is discussed.

Can. Plant Dis. Surv. 65:1, 11-13, 1985.

Colletotrichum acutatum est associé avec la nécrose de la pousse terminale et du bout des branches de jeunes plants de pruche occidentale cultivés en contenants dans une serre, située près d'Aldergrove en Colombie-Britannique. Cet article est la première mention de la présence de ce pathogène sur cet hôte et dans cette région. Les jeunes plants infectés furent plantés dans des sites forestiers sur l'île de Vancouver avant que l'identité du pathogène soit connue. Au cours des deux années suivantes, des inventaires de ces sites n'ont pas permis de détecter la présence de nouvelles infections. Une discussion des dommages pouvant être causés par *C. acutatum* en Colombie-Britannique s'ensuit.

Introduction

Colletotrichum acutatum Simmonds ex Simmonds (6,7), previously unreported in Canada, causes a terminal crook disease of Monterey pine (*Pinus radiata* D. Don) in forest nurseries in New Zealand (1) and Chile (5). In New Zealand and Australia, *C. acutatum* has been found on several other species of pine, including *P. contorta* Dougl. *Colletotrichum acutatum* damages other plants such as coffee (3), tomatoes, and strawberries (2) but strains with a restricted host range have been discovered (1).

This report describes damage on western hemlock (*Tsuga heterophylla* (Raf.) Sarg.) associated with *C. acutatum* following its discovery in 1981 within a nursery. It also gives the results of surveys of forest sites where stock containing diseased seedlings had been outplanted.

Nursery Site

A disease affecting the growing tips of western hemlock seedlings was found early in July 1981 at a nursery near Aldergrove, B.C. Seedlings for reforestation, comprising western hemlock, western red cedar (*Thuja plicata* Donn), and Sitka spruce (*Picea sitchensis* (Bong.) Carr.) had been sown that Spring and raised mainly in styroblocks within greenhouses.

Initial symptoms were small lesions on terminal shoots and branches commencing at, or close to, the tip and progressing down the shoot into the needles. Affected tissues were typically light to reddish brown. Very few seedlings had the crooking which characterizes the disease in Monterey pine (1). A few scattered acervuli characteristic of *C. acutatum* (2) were produced on most infected needles.

Isolations from acervuli and from diseased tissues treated with a 10% solution of sodium hypochlorite for 2-3 minutes consistently yielded similar cultures on potato dextrose agar (PDA). Cultures at room temperature were initially white, becoming rose-pink within a few days and turning a deep red.

Conidia, borne on phialides throughout the colony were ellipsoid and pointed at both ends. A few setae occurred scattered through the colony. Colony and spore characteristics are similar to those described for *C. acutatum* isolates from papaw (6).

In November 1981 a typical culture was identified as *C. acutatum* by Dr. Gary Samuels of the Plant Diseases Division, Auckland, New Zealand, then visiting U.S. Department of Agriculture facilities at Beltsville.

In January 1982, random sampling in every seedlot of all coniferous species was carried out in every bay and greenhouse at the nursery. In the greenhouse where infections were known to occur, 10% of all seedlings, except Sitka spruce, were examined. All of the Sitka spruce were examined since they were very close to infected western hemlock. A 5% sample was used in other greenhouses containing no western hemlock.

All seedlings selected by the sampling system were examined for shoot tip or needle damage. Damage was attributed to *C. acutatum* only when typical acervuli and conidia were present or when typical cultures formed on PDA.

The disease was confined to western hemlock and five of the seven seedlots included infected seedlings (Table 1). An estimated 380 infected seedlings were present at the time of examination.

Some seedlings of lodgepole pine, Mugho pine (*P. mugo* Turra) and Colorado spruce (*Picea pungens* Engelm.) were quite close to, but outside of the infested greenhouse; examination failed to show any *C. acutatum*.

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Table 1. Nursery seedlings infected by *Colletotrichum acutatum*[1]

Species	Seedlot Number	Total No. in Seedlot	No. Infected
Western hemlock	3907	226,300	23
	3011	41,100	2
	3909	12,800	5
	2247	2,790	4
	2610	19,800	4
	3066	82,170	0
	3093	19,000	0
		440,950	38

[1] Also examined, but without detecting *C. acutatum* were 9 seedlots of western red cedar and 4 of Sitka spruce, totaling 1.59 and 0.67 million seedlings respectively.

Outplanted Sites

Approximately 70,000 western hemlock seedlings involving seedlots subsequently found to have *C. acutatum* were planted in October 1981 on 4 logged sites near Northwest Bay on Vancouver Island.

Searches for *C. acutatum* were conducted in spring and fall for approximately two years after outplanting. In the most intensive surveys, conducted each September, four or five persons traversed the sites for one day. All western hemlock seedlings with any tip damage, including some from an earlier planting and some that had regenerated naturally, were examined for fruiting bodies of *C. acutatum*.

The mid-June survey, the earliest date possible because of continuous snow cover, yielded 61 suspect seedlings only one of which had an acervulus containing a single conidium typical of *C. acutatum*. Three other seedlings each had acervuli typical of the pathogen but lacked the conidia. Most damage to the suspect seedlings was caused by frost, sun-scald, and grey mould (*Botrytis cinerea* Pers.).

In September 1982, 71 samples with tip damage were examined without finding *C. acutatum*. The damage had been caused mainly by sun-scald or frost although some Sirococcus blight (*Sirococcus strobilinus* Preuss) was also present.

In September 1983, 68 damaged shoots were examined. No evidence of *C. acutatum* was obtained. Most damage was from late spring frosts but a few seedlings had been attacked by *S. strobilinus*, *B. cinerea*, and *Xenomeris abietis* Barr.

Discussion

The discovery of this pathogen on western hemlock constitutes a new host record and the first report of this disease in Canada. British Columbia is not included in the known world distribution of *C. acutatum* (2) and a forest nursery disease extension service that has operated for many years had no record of *C. acutatum* in B.C. Western hemlock is not grown as a forest species in New Zealand. This probably explains the absence of this species from the host list.

The origin of the inoculum at Aldergrove remains unknown. Perhaps it was introduced on some ornamental Monterey pine that the affected nursery had imported from New Zealand. Incipient infections may have been involved. Studies in New Zealand (4) have established that although 50% of conidia introduced into soil become non-viable within four weeks, a high percentage of infected needles yielded the pathogen even after burial for 8.5 months, indicating a capacity to survive in infected tissue for long periods.

A study to fulfill the requirements of Koch's postulates for *C. acutatum* on western hemlock is desirable but the frequent occurrence of acervuli and of conidia typical of *C. acutatum* and the frequent isolation of the fungus provides strong evidence of pathogenicity.

The absence of any infections attributable to *C. acutatum* on the western red cedar and the Sitka spruce, despite the occurrence of these plants throughout their growth in the same greenhouse with the infected western hemlock implies a resistance to *C. acutatum*.

The incidence of the disease discovered in the January survey of the greenhouses was certainly lower than occurred during the preceding summer. Following discovery of the damage, extensive sanitation culling was carried out. Also, sprays of benomyl, captan and chlorothalonil fungicides were applied. Two of those control the disease in New Zealand (8). It seems likely that the higher greenhouse temperatures favored development of the pathogen. Simmonds (6) found the optimum for *C. acutatum* to be 25-26.5°C. The cultural conditions used for rapid seedling growth results in numerous infection courts in the form of succulent growing tips. Free moisture for the infection process would often be present because of the frequent watering of container stock.

The failure to obtain any evidence of survival and spread of *C. acutatum* at the planting sites may be due either to a low level of inoculum or the long harsh winters or a combination of both factors. The finding of at least one acervulus of *C. acutatum* in June 1982 shows that some inoculum was introduced to the field but the level is unknown. Culling of infected seedlings routinely occurs before shipment. Also, the brittle condition of affected tissues probably caused breakage thus reducing the amount of inoculum taken to the planting site. The initial winter with its continuous snow cover for approximately six months may have been a major factor. These conditions contrast with much warmer conditions in Queensland, Australia and in North Island, New Zealand, where the pathogen is very damaging.

The potential of the strain of *C. acutatum* found in B.C. to have spread to agricultural crops such as tomatoes and strawberries growing in adjacent areas of the Fraser valley is unknown. It has been shown (1) that the New Zealand strain from Monterey pine, designated as f. sp. *pinea*, could also infect lupine, sweetpeas, vetches, and tomatoes. However, isolates originating there from tree lupine and sweetpeas attacked only legumes but not pines. Similarly, isolates from other fruit rots in New Zealand were non-pathogenic on pine.

In New Zealand and Australia, *C. acutatum* infects several species of pine including *P. contorta* (1), suggesting that lodgepole pine in B.C. could be infected under greenhouse conditions.

Except for the collections of *C. acutatum* described in this report, no other collections have been made of this pathogen in B.C. that are known to the authors. The destruction by burning of all western hemlock seedlings within the nursery after the survey had been conducted by staff of the Plant Health Division, Agriculture Canada, probably prevented spread of the pathogen.

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Incidence of root rot and nematodes in barley fields in Prince Edward Island

J. Kimpinski and H.W. Johnston[1]

A survey was conducted in Prince Edward Island during August 1983 in 45 barley fields to observe the incidence of common root rot and population levels of nematodes. Cultivars included in the survey were Birka, Bruce, Perth, and Volla. The dominant plant-parasitic genera were stunt nematodes, *Tylenchorhynchus* spp., and root lesion nematodes, *Pratylenchus* spp. The majority of root lesion nematodes were identified as *P. penetrans*, and the primary fungal pathogen was *Bipolaris sorokiniana*. Population levels of *Tylenchorhynchus* spp. in soil were higher in fields of Birka and Perth than in fields of Bruce and Volla. The numbers of *Pratylenchus* spp. did not differ significantly among any of the cultivars. The general trend was for the incidence of root rot to be positively correlated with numbers of stunt nematodes in soil, and to be negatively correlated with numbers of root lesion nematodes in soils and roots.

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Un inventaire de 45 champs d'orge a été effectué en août 1983 dans l'Île-du-Prince Édouard afin de déterminer l'incidence de la pourriture commune des racines et le niveau des populations de nématodes. Cet inventaire couvre les cultivars d'orge Birka, Bruce, Perth et Volla. Les genres dominants parmi les parasites végétaux s'avèrent être *Tylenchorhynchus spp.*, causant le rabougrissement, et *Pratylenchus* spp., causant les lésions des racines. La majorité des nématodes causant des lésions racinaires fut identifiée comme *P. penetrans* et *Bipolaris sorokiniana* comme le pathogène fongique primaire. Les niveaux de population de *Tylenchorhynchus spp.* s'avèrent plus élevés dans le sol des champs d'orge Birka et Perth que dans celui des champs d'orge Bruce et Volla tandis qu'il n'y a pas de différence significative entre les populations de *Pratylenchus spp.* dans les différents cultivars. Généralement, l'incidence de pourriture commune des racines semble être en corrélation positive avec le nombre de nématodes causant le rabougrissement présent dans le sol et en corrélation négative avec le nombre de nématodes causant des lésions racinaires présent dans le sol et les racines.

Introduction

Diseases periodically cause noticeable yield losses in barley in the Maritime provinces (4). The endemic dysfunction, common root rot, is incited primarily by *Cochliobolus sativus* (Ito and Kurib.) Drechsl. ex. Dastur, conidial state *Helminthosporium sativum* Pamm. King and Bakke., syn. *Bipolaris sorokiniana* (Sacc. in Sorok.), Shoem. (5). The root lesion nematodes, *Pratylenchus penetrans* (Cobb) Filipjev and Sch. Stek. 1941 and *P. crenatus* Loof, 1960, and stunt nematodes (*Tylenchorhynchus* spp.) also occur in cereals in the region (6,7).

Associations between root rot fungi and root lesion or stunt nematodes were observed previously in North America (1,8,11). Powell (10) concluded that these nematodes were important components of the disease in cereals, and it was in this role that they reduced crop yields. The only related study in the Maritime region to date did not detect a relationship between nematodes and fungi in cereals (7). However, the work was conducted at one location only, and the dominant nematode species was *Pratylenchus crenatus*. Previous investigations have indicated that *P. crenatus* is not as pathogenic as *P. penetrans* (2,13).

Therefore, the objectives of this study were to observe the incidence of common root rot and the population levels of root lesion and stunt nematodes in barley fields in Prince Edward Island, and to determine if significant correlations existed between incidences of common root rot and numbers of nematodes.

Materials and methods

A survey was conducted in Prince Edward Island during August 1983 in 45 barley fields of different cropping histories. Three sample sites were selected along the longest diagonal of each field. Approximately 10 plants with roots and soil attached were taken from each site. Close attention was paid to recovering as many of the fine roots as possible. Nematodes were extracted from roots by placing at least 5 g of washed root from each sample in a mist chamber (3). Ten soil cores were taken at random in the plant row at each site with a 25-mm diameter soil probe to a depth of 20 cm. Each sample was mixed thoroughly and a 50-g subsample of soil placed in a modified Baermann pan (12). After 7 days at 20-25°C nematodes that had emerged from soil and roots were identified and counted with a microscope. Nematode data were expressed as number per gram of dry root and per kg of dry soil and transformed to logarithms for calculations of mean populations, analysis of variance, and correlation coefficients.

The plant samples at each site were rated for common root rot severity according to the method of Ledingham et al. (9).

Results and discussion

The dominant plant-parasitic nematode genera recovered in the survey were *Tylenchorhynchus* spp. and *Pratylenchus*

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Table 1. Incidence of common root rot and population levels of root lesion and stunt nematodes in barley during August 1983 in Prince Edward Island.

Cultivar	No. of Samples[a]	Incidence of root rot (%)	Number of Nematodes[b] Per g of root	Per kg of soil	
			Root lesion	Root lesion	Stunt[c]
Volla	87	73	440	1050	770
Bruce	21	63	390	710	870
Perth	18	85	640	1210	2080
Birka	9	70	560	910	2190

[a] Three samples from each field.
[b] Geometric means.
[c] Stunt nematodes were more numerous in Perth and Birka soil than in soil from Volla and Bruce (P = 0.05). Determined by analysis of variance on data where number of samples per treatment varies.

spp., and the majority of the root lesion nematodes were identified as *P. penetrans*. *Bipolaris sorokiniana* was identified as the primary fungal pathogen inciting root rot.

The numbers of stunt nematodes in soil were higher in fields of Birka and Perth than in fields of Bruce and Volla (Table 1). The population levels of root lesion nematodes in roots and soil did not differ significantly among the four cultivars. A significant positive correlation between incidence of common root rot and numbers of stunt nematodes in soil, and a significant negative correlation between the incidence of root rot and the numbers of root lesion nematodes in soil were obtained for the cultivar Bruce (Table 2). The general trend was for the occurrence of common root rot severity to be positively correlated with numbers of stunt nematodes in soil, and to be negatively correlated with numbers of root lesion nematodes in soil and roots.

Table 2. Correlation coefficients between incidences of common root rot (rr) in barley and number of root lesion nematodes in roots (RLR) and soil (RLS), and numbers of stunt nematodes in soil (STS) during August 1983 in Prince Edward Island.

Cultivar	Degrees of Freedom	rrxRLR	rrxRLS	rrxSTS
Volla	85	-0.24**	-0.07	0.01
Bruce	19	-0.27	-0.52**	0.70***
Perth	16	-0.40*	-0.23	0.40*
Birka	7	-0.29	-0.14	0.32

*,**,***Significant at P = 0.1, 0.05 and 0.01, respectively.

The existence of a significant negative correlation between numbers of root lesion nematodes and the presence of common root rot in the cultivar Bruce did indicate that the two groups of organisms were antagonistic to each other. Furthermore, the positive correlation between numbers of stunt nematodes and the incidence of common root rot did not confirm a synergistic interaction. More information is necessary, especially in the spring when host plants are at the seedling stage to further characterize the relationship between such disease inciting organisms. In addition, accurate identifications of fungal and nematode species must be completed.

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Présence et causes possibles de la coulure des graminées chez la fléole des prés au Québec

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Des travaux ont été entrepris sur la coulure des graminées chez la fléole des prés (*Phleum pratense*) au Québec. Cette maladie provoque le blanchissement et le dessèchement prématuré de l'épi et de la tige à partir du dernier noeud et entraîne la stérilité de l'inflorescence, ce qui affecte la production de semence. La coulure a été trouvée dans les 12 champs de fléole visités dans quatre régions agricoles du Québec en 1983 et le pourcentage d'épis atteints a été en moyenne de 9%. Quoique le *Fusarium poae* ait été isolé fréquemment de tiges malades, l'inoculation de ce champignon sur la fléole par différentes méthodes ne provoque pas de symptôme visible sauf lorsque les tiges sont perforées au moyen d'une épingle entomologique au-dessus du dernier noeud au tout début de l'épiaison. Par contre, les mêmes symptômes ont été reproduits en utilisant une épingle stérilisée. On a récolté des insectes périodiquement au cours des mois de juin et juillet 1983 dans des parcelles de fléole à La Pocatière. Les insectes prédominants appartenaient aux *Cicadellidae*, aux *Cercopidae*, aux *Aphididae*, aux *Thripidae* et aux *Chloropidae*. L'incidence de la coulure a cependant été très faible dans les parcelles. Les *Miridae*, dont l'apparition correspond à celle de la coulure, figurent également parmi les insectes suspects.

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Studies have been initiated on silver top of timothy (*Phleum pratense*) in Québec. This disease is characterized by a whitening and a premature death of head and stalk from the uppermost node, therefore reducing seed yield. Silver top was found in the 12 timothy fields from four agricultural regions surveyed in 1983; 9% of heads were affected. *Fusarium poae* was isolated above the terminal node of diseased stalks but inoculation of this fungus by different methods did not produce any symptom of silver top. Puncturing the stem with an entomological needle above the uppermost node just before emergence of the ears did cause silver top syndrome. Insects, surveyed periodically during June and July 1983, were mainly *Cicadellidae*, *Cercopidae*, *Aphididae*, *Thripidae* and *Chloropidae*. However, silver top incidence was very low in our sampling plots. *Miridae* must also be suspected because their occurrence coincide with silver top appearance.

Introduction

La fléole des prés (*Phleum pratense* L.) est la seule graminée fourragère cultivée au Québec dont la semence est en majorité produite dans la province (Canada Grains Council 1983). Or, la principale «maladie» qui affecte cette culture au Québec est la coulure des graminées («silver top», «white head», «white ear») qui provoque la stérilité des inflorescences. Le rendement en semences peut être considérablement diminué dans les champs fortement affectés. Ainsi, des pourcentages d'infestation de plus de 80% ont été rapportés dans plusieurs régions des États-Unis (Hardison 1959, Keil 1946) et en Allemagne (Wetzel 1977). Au Canada, des pertes de 12 à 14% ont été signalées chez le pâturin des prés en Colombie-Britannique (Creelman 1961) et en Alberta où l'incidence moyenne de la maladie sur le brome a été de 0,44% au cours des années 1970-1973 (Berkenkamp 1974).

Selon une récente revue de la littérature (Gagné *et al.* 1984), les causes de cette maladie peuvent être d'origines diverses. On l'a attribuée entre autres aux thrips (Comstock 1888), aux acariens, principalement le *Siteroptes graminum* (Reuter) (Hodgkiss 1908), à certains hémiptères phytophages (Arnott et Bergis 1967, Peterson et Vea 1971), à un champignon, le *Fusarium poae* (Pk.) Wr. (Keil 1946) et, finalement, à des causes d'origine physiologique (Pohjakallio *et al.* 1960).

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L'ampleur et les causes de la coulure des graminées au Québec étant pratiquement inconnues, nous avons entrepris des travaux en vue d'acquérir des connaissances relatives à l'importance et à l'étiologie de cette maladie de la fléole au Québec.

Matériel et méthodes

Inventaire de la maladie. Nous avons effectué un inventaire de la coulure à l'été 1983 dans 12 champs de fléole destinés à la production de semence et répartis dans les régions agricoles de Nicolet, Richelieu, Sud-Ouest-de-Montréal et Nord-de-Montréal. Les pourcentages d'infestation furent déterminés en comptant le nombre d'épis sains et malades présents à l'intérieur de 10 quadrats de 26 × 45 cm répartis au hasard dans chacun des champs.

Parcelles expérimentales. Nous avons utilisé des parcelles de *Phleum pratense* L. cv. Climax situées à La Pocatière pour des récoltes d'insectes et de plantes. Ces parcelles, établies en 1979, ont servi à des essais de fertilisation pour la production de semence jusqu'en 1982. Elles mesuraient 2,16 × 5,0 m et étaient constituées de quatre rangs de fléole semés dans le sens de la longueur de la parcelle. Huit parcelles ayant reçu les mêmes quantités d'azote (50 kg/ha) et produit les mêmes rendements au cours des années antérieures furent choisies pour y effectuer des récoltes d'insectes. Afin de savoir si un insecticide aurait un effet sur la coulure et sur les insectes susceptibles d'en être la cause, nous avons traité quatre de ces parcelles au diméthoate (Cygon 2-E) et les quatre autres n'ont reçu aucun traitement. L'insecticide fut appliqué le 17 mai et

les 8 et 28 juin, à l'aide d'un pulvérisateur de précision à la dose de 648 g m.a./ha.

Les autres parcelles non utilisées pour les récoltes d'insectes ont servi à des fins d'observation et pour l'isolement des champignons.

Isolement de champignons. Pour vérifier la présence du *F. poae* sur les tiges de fléole, des segments de tiges saines et malades furent prélevés juste au-dessus du dernier noeud et stérilisés en surface en les trempant pendant 15 s dans de l'hypochlorite de sodium 2,5% puis en les rinçant dans trois bains d'eau distillée stérile. Ils furent ensuite déposés dans des boîtes de Pétri contenant de la gélose de pomme de terre glucosée (PDA, Difco) additionnée de 50 ppm de chlortétracycline pour inhiber la croissance des bactéries. Les boîtes furent incubées à 25°C jusqu'à ce que les colonies de champignons soient bien développées (5-7 jours). Les cultures ainsi obtenues ont été repiquées sur PDA pour purification et identification ultérieure.

Récoltes d'insectes. Afin de connaître les insectes présents sur la fléole et déterminer ceux qui sont susceptibles de causer la coulure des graminées, des insectes furent récoltés périodiquement au cours des mois de juin et juillet 1983 à La Pocatière. Les rangs des parcelles décrites précédemment ont été taillés de façon à ne conserver que 15 touffes de fléole par rang. Les insectes furent récoltés en recouvrant rapidement les touffes d'un sac de polythène transparent et en le refermant sur la base de tiges de façon à ne pas laisser échapper les insectes ainsi emprisonnés. Les tiges étaient coupées au niveau du sol et récoltées simultanément dans le sac avec les insectes. A chaque récolte, cinq touffes choisies au hasard furent ainsi récoltées dans chacune des huit parcelles expérimentales et les sacs ont été entreposés au congélateur jusqu'au moment de l'observation en laboratoire. Les tiges étaient alors disséquées et observées à la loupe. Les insectes étaient déposés dans l'alcool puis identifiés. Les nombres d'épis sains et malades ont également été notés.

Pouvoir pathogène du *F. poae*. Des graines pré-germées de *P. pratense* cv. Climax furent semées en pots dans du terreau stérile et gardées en chambre de croissance jusqu'à la maturité des plantes. Celles-ci ont alors été coupées au niveau du sol pour favoriser le tallage et l'uniformité de la croissance. On a inoculé le champignon de différentes façons à des tiges de la repousse en utilisant des suspensions aqueuses de *F. poae* (3×10^6 spores/mL) isolé d'une tige de fléole infectée.

Dans un premier test, des tiges furent inoculées à différents stades de croissance en prélevant une gouttelette d'une suspension fongique à l'aide d'une épingle entomologique et en perforant transversalement la tige de façon à introduire les propagules dans la blessure. On a perforé de la même façon des tiges témoins mais avec une épingle stérile. Ces traitements ont été effectués à la base des tiges au stade début-montaison et juste au-dessus du dernier noeud visible aux stades gonflement et début-épiaison.

Dans une autre série de pots, on a contaminé le sol en ajoutant 200 mL d'une suspension de *F. poae* après avoir infligé des blessures aux racines. Des pots témoins ont reçu le même traitement sans champignon.

Une troisième méthode d'inoculation a consisté à déposer, à l'aide d'un compte-gouttes, quelques gouttes d'une suspension du champignon entre la gaine et la tige de la plante au début de l'épiaison, lorsque l'épi émerge de la gaine. Après inoculation du champignon, les plantes ont séjourné pendant 48 h dans une chambre maintenue à 90% d'humidité.

Tableau 1. Présence de la coulure des graminées chez la fléole des prés (*Phleum pratense*) dans certaines régions agricoles du Québec en 1983.

Champ no	Localité	Région agricole	Epis avec coulure (%)†
1	Saint-Philippe	Sud-Ouest-de-Montréal	8,1
2	Saint-Constant	Sud-Ouest-de-Montréal	7,6
3	Sainte-Martine	Sud-Ouest-de-Montréal	9,4
4	Nicolet	Nicolet	6,5
5	Nicolet	Nicolet	0,7
6	Saint-Antoine	Richelieu	9,3
7	Saint-Alphonse	Richelieu	5,0
8	Saint-Roch	Richelieu	7,4
9	Saint-Denis	Richelieu	19,6
10	Saint-Ours	Richelieu	8,0
11	Joliette	Nord-de-Montréal	12,9
12	Joliette	Nord-de-Montréal	12,9
Moyenne			9,0

† Déterminé en comptant les épis sains et malades dans 10 quadrats de 26 x 45 cm répartis au hasard dans chaque champ.

Résultats et discussion

Nous avons trouvé de la coulure dans tous les champs de fléole visités en 1983 et les pourcentages d'épis malades ont varié de 0,7 à 19,6%, la moyenne étant de 9% (tableau 1). Des infestations plus fortes, allant jusqu'à 50%, ont toutefois été signalées dans la région du Lac-Saint-Jean (G. Bossanyi, communication personnelle).

Chez la fléole au Québec, la coulure des graminées se manifeste par le dessèchement et le blanchissement prématuré de l'inflorescence et de la tige à partir du dernier noeud; les autres parties de la plante demeurent vertes et saines. Les tiges malades peuvent facilement être retirées de la gaine et la partie basale située au-dessus du dernier noeud apparaît foncée et nécrosée. Ces symptômes, en général, sont semblables à ceux observés chez d'autres graminées ailleurs en Amérique du Nord (Berkenkamp et Meeres 1975, Keil 1946) et en Europe (Wetzel 1977). D'autres auteurs cependant rapportent un ratatinement de la base de la tige plutôt qu'une nécrose (Peterson et Vea 1971).

Nous avons fréquemment isolé le *F. poae* des tiges de fléole malades, mais aucune des méthodes d'inoculation utilisées n'a permis de démontrer son pouvoir pathogène. Toutefois, la perforation du système vasculaire au-dessus du dernier noeud au début de l'épiaison a permis de reproduire les symptômes de la coulure. Sur 12 épis perforés et inoculés avec le *F. poae* au début de l'épiaison, deux tiges ont produit des épis avec de la coulure et le champignon fut réisolé de 11 de ces tiges. Cependant, trois des quatre tiges témoins ayant été perforées sans champignon ont également montré les symptômes de la coulure et le *F. poae* était absent de ces tiges. L'observation des tiges n'ayant pas produit d'épis malades a révélé que la perforation, dans ces cas, avait eu lieu involontairement au-dessous du dernier noeud. Ces résultats indiquent que la coulure peut être provoquée par une perforation du système vasculaire au-dessus du dernier noeud au moment où l'épi émerge de la gaine. Le même traitement, infligé au stade début-montaison ou au stade gonflement, n'a pas provoqué la coulure des épis. Le *F. poae* inoculé dans le sol n'a provoqué aucun symptôme de maladie apparent chez la fléole de même que l'inoculation du champignon à l'aide d'un compte-gouttes, entre la gaine et la tige lors de l'émergence de l'épi. Le *F. poae* ne semble donc pas pathogène de la fléole et il est possible, comme l'a suggéré Hardison (1959), que ce champignon soit un envahisseur secondaire plutôt que la cause primaire de la maladie. Le fait que les symptômes de la coulure aient pu être reproduits simplement en perforant la tige à un stade de croissance et à un endroit déterminés démontre que des dommages similaires pourraient être causés par certains insectes piqueurs et produire la maladie au champ.

Les nombres d'insectes récoltés dans les parcelles à La Pocatière sont indiqués dans le tableau 2. Ceux retrouvés en plus grand nombre furent les *Cicadellidae*, les *Cercopidae*, les *Aphididae*, les *Chloropidae* et les *Thripidae* (fig. 1). Ces insectes sont tous phytophages sauf les *Chloropidae* qui, cependant, perforent les tiges pour pondre leurs oeufs. Comstock (1888) et Hardison (1959) ont rapporté que la maladie pouvait être causée par les thrips. Ces insectes ont été observés fréquemment entre la gaine et la tige des plants de fléole mais leur présence semble aussi fréquente sur les tige saines que malades. Peterson et Vea (1971) et Arnott et Bergis (1967) ont démontré que certains hémiptères de la famille des *Miridae*

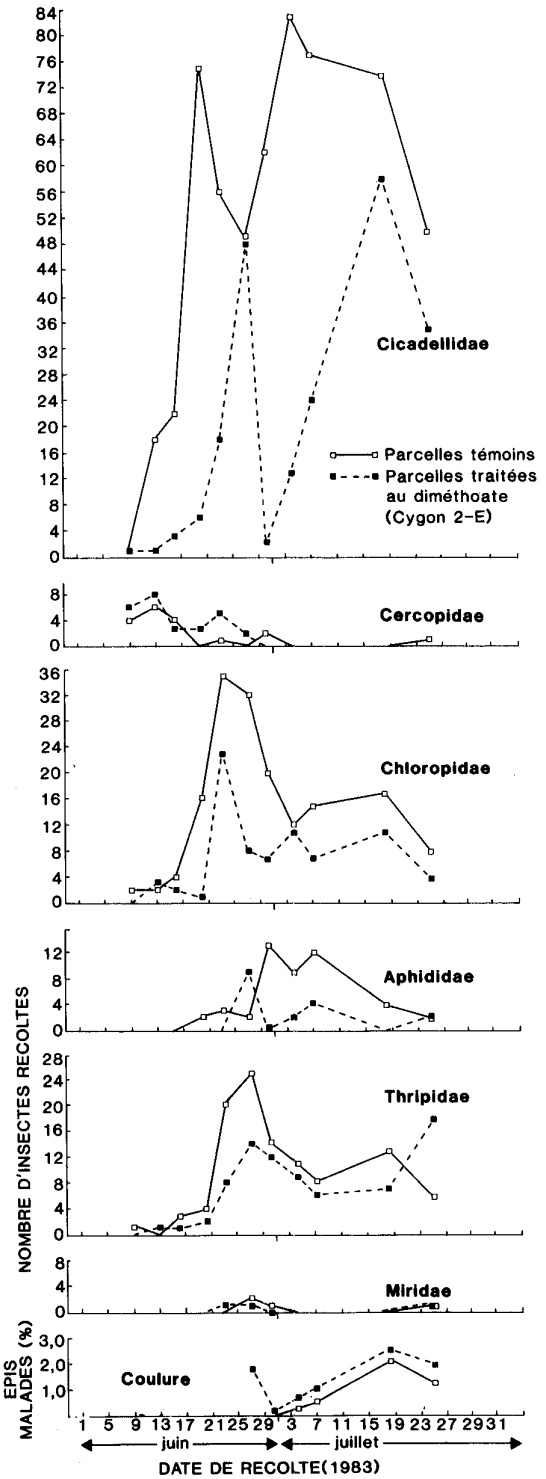

Figure 1. Variation du nombre d'insectes et du pourcentage de coulure au cours de l'été 1983 dans des parcelles de *Phleum pratense* à La Pocatière.

Tableau 2. Nombre d'insectes récoltés pendant l'été 1983 à La Pocatière dans des parcelles de fléole des prés (*Phleum pratense*) traitées au diméthoate (D) et non traitées (T).†

		Nombre d'insectes/récolte‡																					
		09 juin		13 juin		16 juin		20 juin		23 juin		27 juin		30 juin		04 juil.		07 juil.		18 juil.		25 juil.	
Ordre	Famille	D	T	D	T	D	T	D	T	D	T	D	T	D	T	D	T	D	T	D	T	D	T
Homoptères	Aphididae	0	0	0	0	0	0	0	2	0	3	9	2	0	13	2	9	4	12	0	4	2	2
	Cercopidae	6	4	8	6	3	4	3	0	5	1	2	0	0	2	0	0	0	0	0	0	0	1
	Cicadellidae	1	1	1	18	3	22	6	75	18	56	48	49	2	62	13	83	24	77	50	74	35	50
	Coccidae	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	5	0	0	0	0
	Phylloxeridae	0	0	0	0	0	0	0	0	0	1	0	0	2	3	0	0	0	1	0	0	0	1
	Non identifiée	0	0	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Hémiptères	Coreidae	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
	Miridae	0	0	0	0	0	0	0	0	1	0	1	2	0	1	0	0	0	0	0	0	1	1
	Nabidae	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	0	0
	Tingitidae	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
	Non identifiée	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
Thysanoptères	Aeolothripidae	0	0	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Phloeothripidae	0	0	1	4	0	0	0	0	0	0	3	1	3	0	0	1	0	0	0	0	0	0
	Thripidae	0	1	1	0	1	3	2	4	8	20	14	25	12	14	9	11	6	8	7	13	18	6
	Non identifiée	0	4	3	3	0	0	0	0	1	0	0	0	1	4	0	0	2	0	0	0	0	0
Hymenoptères	Braconidae	0	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Diapriidae	0	0	0	0	0	1	0	0	0	0	0	0	0	0	5	1	0	0	0	3	0	1
	Dryinidae	0	0	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	3	0	0	0	0
	Encyrtidae	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
	Eulophidae	0	0	0	0	1	0	1	2	0	1	0	0	0	0	0	0	0	0	0	0	0	0
	Formicidae	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0
	Liopteridae	0	0	0	0	0	0	0	0	0	0	3	1	0	0	0	0	2	0	0	0	0	0
	Scelionidae	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
	Sphecidae	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Non identifiée	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	3	0	0	0	0	1	1
Diptères	Agromyzidae	0	1	0	0	1	0	1	1	0	0	2	1	0	0	1	0	0	0	0	0	0	0
	Anthomyiidae	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0
	Cecidomyiidae	2	1	3	1	1	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Ceratopogonidae	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
	Chamaemyiidae	0	0	1	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0	0	0	0	0
	Chironomidae	0	2	0	0	2	0	3	2	1	0	1	0	0	2	0	0	0	0	0	0	0	0
	Chloropidae	0	2	3	2	2	4	1	16	23	35	8	32	7	20	11	12	7	15	11	17	4	8
	Dalichopodidae	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Empididae	1	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Ephydridae	0	0	0	0	0	0	0	0	1	2	2	0	0	0	0	1	0	0	0	0	0	0
	Helomyzidae	0	0	0	0	0	0	0	0	2	0	2	0	0	0	0	0	0	0	0	0	0	0
	Lonchopteridae	0	3	0	1	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0
	Mycetophagidae	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Neottiophiliidae	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
	Phoridae	0	1	1	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	1	0	1
	Scopeumatidae	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0
	Sepsidae	0	1	1	4	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
	Simuliidae	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
	Sphaeroceridae	0	1	1	14	0	9	0	6	0	1	0	3	0	0	0	3	0	5	3	1	1	0
	Tipulidae	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
	Trixoscelididae	0	0	0	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	2	0	0	0
	Non identifiée	0	1	2	1	1	0	0	0	0	3	0	0	0	0	1	0	0	0	0	0	1	0
Total		10	23	26	65	15	46	23	109	63	123	96	117	30	126	43	124	46	127	85	115	65	72

† D = parcelles traitées au diméthoate (Cygon 2-E) le 17 mai, le 8 et le 28 juin 1983; T = parcelles non traitées (témoin).
‡ Une récolte = les insectes prélevés dans des sacs de polythène placés sur cinq touffes de fléole prises au hasard dans chacune des quatre parcelles/traitement.

causaient la coulure en piquant les tiges juste au-dessus du dernier noeud pour se nourrir. Des insectes appartenant à cette famille furent récoltés dans nos parcelles et la date de leur apparition coαncide avec celle où les premiers symptômes de maladie sont apparus (fig. 1). De plus, leur nombre peu élevé pourrait expliquer les faibles pourcentages d'épis malades dans les parcelles.

Quoique les insectes étaient en général moins nombreux dans les parcelles traitées au diméthoate, des applications plus fréquentes auraient été nécessaires pour obtenir une répression constante des insectes durant toute la saison. Les premiers symptômes de coulure sont apparus aux environs du 27 juin, soit au début de l'épiaison (fig. 1). Les insectes susceptibles de causer la maladie devaient donc être présents durant cette période. Cependant, la faible incidence de la maladie dans les parcelles permet difficilement de faire un rapprochement entre la coulure et une famille d'insectes en particulier. Les pourcentages de coulure sont également trop faibles pour qu'un effet dû au diméthoate puisse être observé.

Conclusion

Le *F. poae* isolé de tiges de fléole atteintes de coulure n'a causé aucun dommage ni symptômes de maladie lorsqu'inoculé de différentes façons. Par contre, les symptômes de la coulure ont été reproduits en transperçant le système vasculaire des tiges juste au-dessus du dernier noeud lors de l'émergence de l'épi dans la gaine. Il est donc plausible que certains insectes puissent occasionner au champ des dommages similaires et ainsi causer la coulure. Parmi les insectes récoltés dans les parcelles de fléole, les plus aptes à provoquer la coulure sont les *Thripidae*, les *Miridae*, les *Aphididae*, les *Chloropidae*, les *Cercopidae* et les *Cicadellidae* à cause des blessures qu'ils sont susceptibles de causer aux plantes avant l'apparition de la maladie.

Les travaux devront être poursuivis au cours des prochaines années afin de recueillir des informations plus précises sur cette maladie et de déterminer la cause exacte de la coulure chez la fléole au Québec.

Des remerciements sont adressés à Hélène Nadeau pour son assistance et à Michel Gagnon (Institut de technologie agricole, La Pocatière) pour l'identification des insectes.

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

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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.

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