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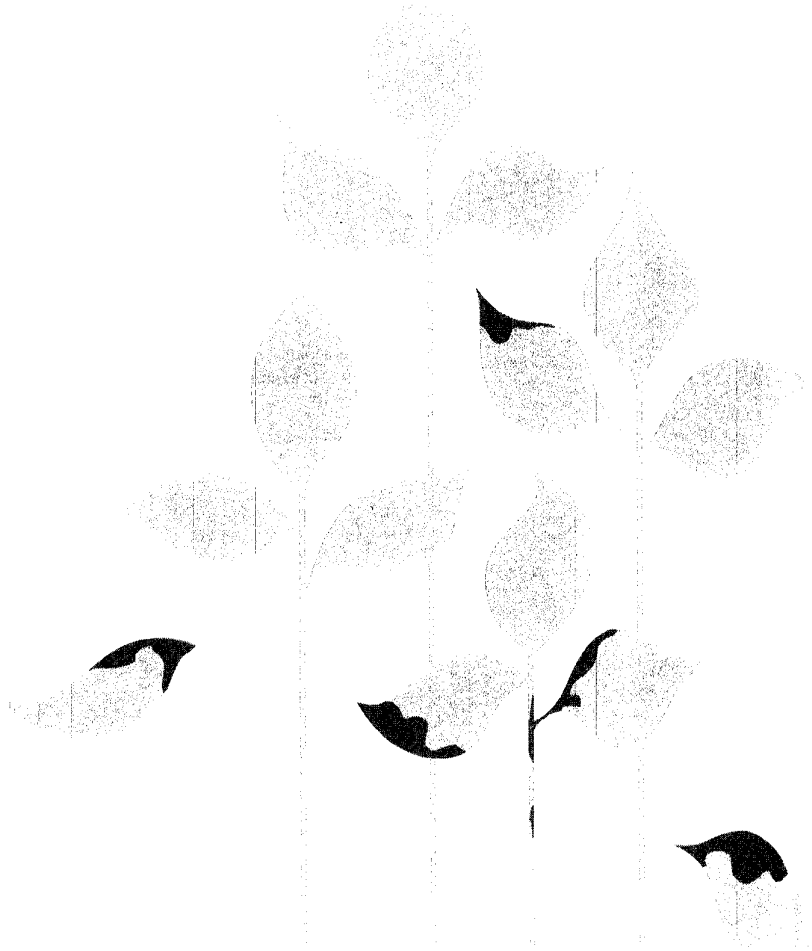

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Contents/Contenu

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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Northern distribution of LTB snow mold in Canada[1]

J. A. Traquair[2], J. B. Lebeau[3], J. E. Moffat[3], and M. Kokko

The occurrence of low-temperature basidiomycete (LTB) snow mold [*Coprinus psychromorbidus*] in the Yukon Territory was confirmed in 1964 by the isolation of the LTB from winter rye from Whitehorse. Although the LTB has been reported to occur in Alaska, we are recording its northern distribution in Canada. Growth characteristics of the Yukon isolate closely resemble those of isolates from Alaska and Alberta.

Can. Plant Dis. Surv. 63:1, 1-2, 1983.

L'occurrence du piétin hibernale [*Coprinus psychromorbidus*] dans le Territoire de Yukon a été confirmé en 1964 par l'isolation du basidiomycète frigophile (BF) sur du seigle d'hiver de Whitehorse. Bien que la présence du BF ait été rapporté en Alaska, ce travail est un registre de sa distribution nordique au Canada. Cet isolat du Yukon présente des caractéristiques de croissance similaires aux isolats obtenus de l'Alaska et de l'Alberta.

Introduction

The basis for claims (1, 6) that the low-temperature basidiomycete (LTB), now identified as *Coprinus psychromorbidus* (3, 5), occurs in the Yukon is not clear. In their survey of snow mold damage in Alaska and the Yukon, Lebeau and Logsdon (2) found LTB snow mold on *Poa pratensis* at College and on *Calamagrostis canadensis* at Summit Lake in Alaska. They were unable to find it in the Yukon. Sprague (4) subsequently showed that this snow mold occurred on grasses at Skagway in Alaska.

In May of 1964, Mr. J. Y. Tsukamoto, an agronomist at Whitehorse in the Yukon, submitted snow mold damaged winter rye (cv. Sitnikoff) to J. B. Lebeau for diagnosis. LTB was isolated and stored in the culture collection at the Lethbridge Research Station. This paper describes the Whitehorse isolate in relation to other *C. psychromorbidus* cultures and documents the occurrence of LTB snow mold in the Yukon.

Materials and methods

Five isolates from the culture collection at the Lethbridge Research Station (LRS) were examined: LRS 006 (= 64.14.1) from winter rye, Whitehorse, Yukon, May 1964: LRS 010 (= 69.1.1) from winter wheat inoculated with W1, Lethbridge, Alberta, March 1969: LRS 011 (= 69.2.1) from alfalfa inoculated with W1, Lethbridge, Alberta, March 1969; LRS 027 (= W18) from Kentucky bluegrass, College, Alaska, May 1956; LRS 028 (= W19) from reedgrass, Summit Lake, Alaska, May 1956. Each isolate was obtained originally by plating segments of diseased crown tissue washed in water, on potato dextrose agar (PDA) and incubating at 1-2°C. Hyphal tip cultures were stored on PDA in tubes at 4°C.

For culture studies, eight replicates of each isolate were grown on PDA in 9 cm petri plates at 10°C in the dark for 6 weeks. After 6 weeks the cultures were removed to the laboratory bench and incubated at 22°C for two weeks under alternating light and dark conditions.

Results and discussion

On PDA growth of the Yukon isolates is very similar to that of isolates from Alaska and Alberta (Fig. 1). Growth is moderate to slow at 10°C. Colonies spread across 9 cm plates in 7 to 8 weeks. Aerial mycelium is white, while the reverse of colonies is slightly yellowish. The colony margin is even, appressed when 1-2 weeks old and later somewhat densely woolly. The surface mycelium is woolly to somewhat cottony. In this respect, the Yukon isolate resembles A-type (e.g. W1) isolates of LTB but is not as cottony as B-type isolates (e.g. W2) previously described (5, 9). Hyphal knots or sclerotial (stromatic) patches (8) are not produced. The Alaska and Alberta isolates, on the other hand, regularly produce hyphal knots bearing honey-colored exudates in 4-5 weeks. Failure to produce hyphal knots and sclerotia has been reported for other isolates of *C. psychromorbidus* from spores or diseased host tissue (5, 7).

Anatomically, the mycelium of the Yukon isolate is very similar to that of Alberta and Alaska isolates. Hyphae are hyaline, thin-walled, 1.8-4.5 μm wide (average 2.5 μm) with clamp connections at cross-walls. After 6 weeks, gnarled and contorted hyphae are observed in the submerged mycelium. Occasionally, these cells are somewhat refractile and thick-walled. Terminal hyphal swellings and antleroid (5) branches on the surface hyphae are rare.

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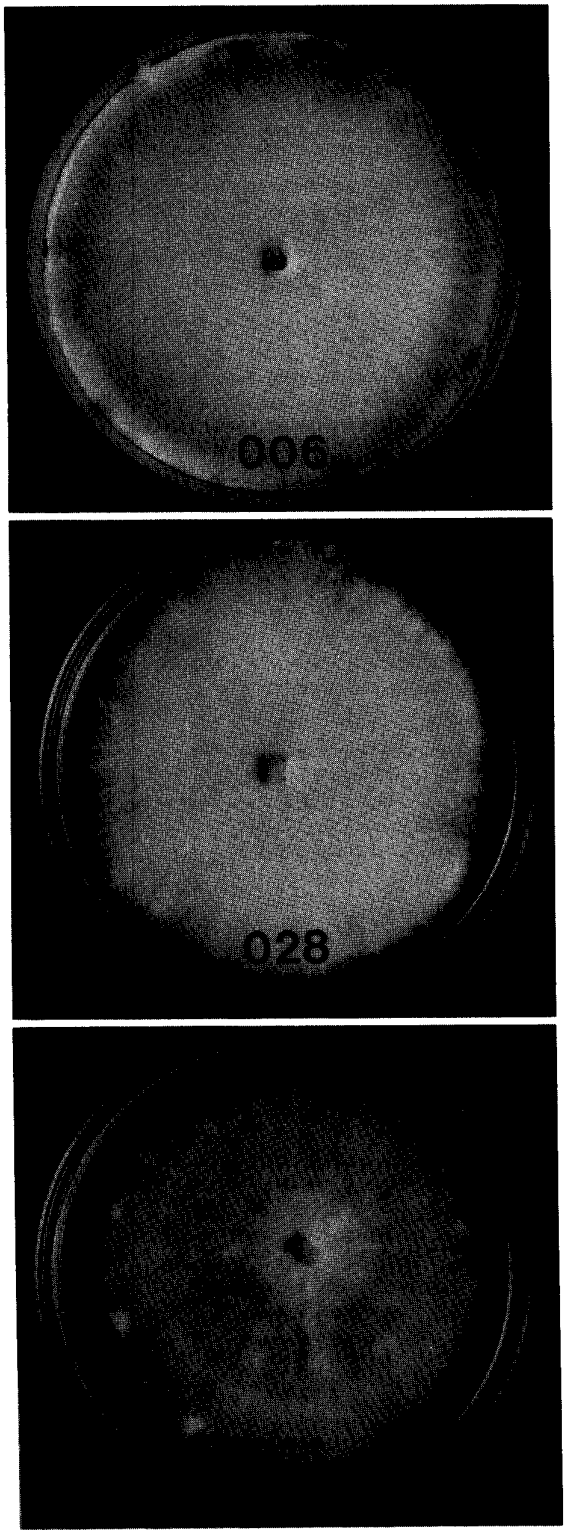

Figure 1. The LTB snow mold, *Coprinus psychromorbidus* for the Yukon (LRS 006), Alaska (LRS 028) and Alberta (LRS 011): 8 weeks old cultures on PDA.

Literature cited

1. Jamalainen, E.A. 1974. Resistance in winter cereals and grasses to low-temperature parasitic fungi. Ann. Rev. Phytopathol. 12: 281-301.
2. Lebeau, J.B., and C.E. Logsdon. 1958. Snow mold of forage crops in Alaska and Yukon. Phytopathol. 48: 148-150.
3. Redhead, S.A., and J.A. Traquair. 1981. *Coprinus* sect. *Herbicolae* from Canada, notes on extralimital taxa, and the taxonomic position of a low-temperature basidiomycete forage crop pathogen from Western Canada. Mycotaxon 13: 373-404.
4. Sprague, R. 1962. Some leafspot fungi on western Gramineae. XVI. Mycologia 54: 593-610.
5. Traquair, J.A. 1980. Conspecificity of an unidentified snow mold basidiomycete and a *Coprinus* species in the section *Herbicolae*. Can. J. Plant Pathol. 2: 105-115.
6. Traquair, J.A. 1981. Discovery of a sporulating state for the LTB snow mold. Canada Agriculture 26: 18-20.
7. Traquair, J.A., and J. Drew Smith. 1982. Sclerotial strains of *Coprinus psychromorbidus*, a snow mold basidiomycete. Can. J. Plant Pathol. 4: 27-36.
8. Ward, E.W.B. 1964. The formation of stroma-like structures in cultures of a sterile low-temperature basidiomycete. Can. J. Bot. 42: 1025-1030.
9. Ward, E.W.B., J.B. Lebeau, and M.W. Cormack. 1961. Grouping of isolates of a low-temperature basidiomycete on the basis of cultural behavior and pathogenicity. Can. J. Bot. 39: 297-306.

Armillaria root rot on urban trees: another perspective to the root rot problem in Newfoundland

by Pritam Singh and G.C. Carew[1]

This article is the first record of Armillaria root rot on ornamental and shade trees in Newfoundland. It discusses the distribution, severity and impact of the disease in urban areas, and implication of these findings on the root rot problem in this Region.

Can. Plant Dis. Surv. 63:1, 3-6, 1983.

Cet article est le premier à mentionner la présence du pourridié-agaric sur les arbres ornementaux et à ombrage de Terre-Neuve. Il discute de la distribution, de la sévérité et de l'impact de cette maladie en zone urbaine, et de l'implication de ces résultats sur le problème de la pourriture des racines dans cette région.

Introduction

Armillaria or shoe-string root rot, caused by *Armillaria mellea* (Vahl ex Fr.) Kummer [= *Armillariella mellea* (Vahl ex Fr.) Karst], is an important root disease affecting a variety of forest, shade, ornamental and orchard trees and shrubs in Canada and the United States. In Newfoundland, *A. mellea* is island-wide in distribution and has been observed both as a parasite and saprophyte on 66 tree provenances belonging to 22 softwood and hardwood, indigenous and introduced forest tree species growing in a variety of site, stand and plantation conditions (Singh, 1981b). Armillaria root rot is now regarded as the most important disease of living forest trees in this Region but it has been recorded only from natural stands and plantations (Fig. 1). This article records the occurrence of the root rot fungus on ornamental trees in Newfoundland and discusses the implications of this finding on the root rot problem in the Region.

Results

In the summer of 1981, two 6 to 7 year old dead ornamental trees of Scots pine, *Pinus sylvestris* L., in a home garden in St. John's showed the presence of characteristic symptoms and signs of Armillaria root rot (Fig. 2). The fungus was well established on all the primary and secondary roots of the infected trees; a few tertiary roots were also infected. The mycelium under the bark reached as far as 18 cm above ground.

Later in the fall, twenty one more chlorotic and dead trees, showing similar symptoms and signs, were observed in this and eleven more home gardens and landscapes in five recently developed urban areas scattered across the Island (Fig. 1). The infected trees belonged to seven species: Sitka spruce, *Picea sitchensis* (Bong.) Carr.; Canada yew, *Taxus canadensis* Marsh.; white birch, *Betula papyrifera* Marsh.; pin cherry, *Prunus pensylvanica* L.F.; American mountain-ash, *Sorbus americana* Marsh.; white spruce, *Picea glauca* (Moench) Voss; and black spruce, *Picea mariana* (Mill.) B.S.P. Examination of these trees and isolation of the pathogen confirmed the presence of *A. mellea.*

There was no apparent above ground source of infection at any of these locations. The houses were located in the newer housing subdivisions developed on recently harvested softwood forests[2]. The gardens were underlain by a 1-2 metre of fill containing top soil (TS), soil (S), gravel and stones (St), and the buried plant materials (P), including decaying logs and twigs, stumps and roots from the previous forest (Fig.3). Sample pits dug in the gardens showed that the buried plant material occurred near the surface as well as 1.5 metres deep; it was decayed and infected with *A. mellea*, which produced abundant rhizomorphs. Many of these rhizomorphs were traced to the roots of infected ornamental trees. Singh (1977 & 1981a) reported that subterranean rhizomorphs are one of the principal means of the spread of *A. mellea* in Newfoundland forests; they being negatively geotropic, grow upwards towards the soil surface and infect roots on the way up. The root systems of the infected trees grew within 0.3 to 1 metre of soil depth.

All the infected trees had been transplanted within the past 5 years. They were growing under normal urban environment and were ranging from 1 to 4 metres in height. Some of these trees were apparently healthy, others were growing under stress and showed symptoms of decline, still others were lightly to moderately defoliated by various insect pests. Wargo and Houston (1973), Wargo (1978 & 1981), Schoeneweiss (1975 & 1981) and Tattar (1978) reported that ornamental and shade trees are generally not growing under optimum natural conditions. Often they are under

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[2] *Site conditions of these areas were generally poor and their moisture regime varied from moist to wet [Moisture Regime Scale 4 to 6 (Damman, 1964)]. Prior to housing developments, these sites had supported scrubby black spruce forests.*

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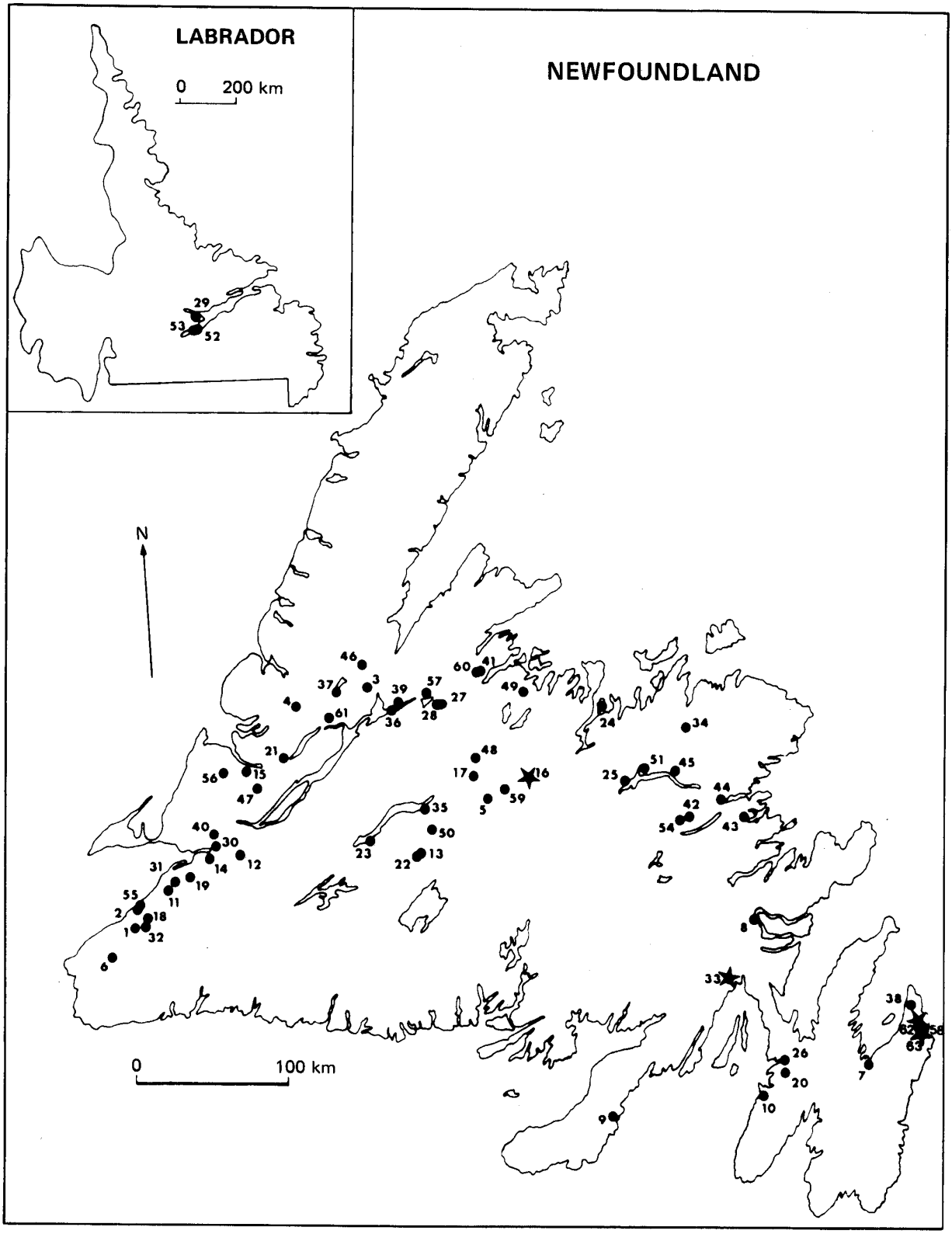

Fig. 1. Distribution of *Armillaria mellea* in Newfoundland and Labrador. Nos. 16, 33, 58, 62 and 63 are the urban areas where the fungus was observed on ornamental trees.

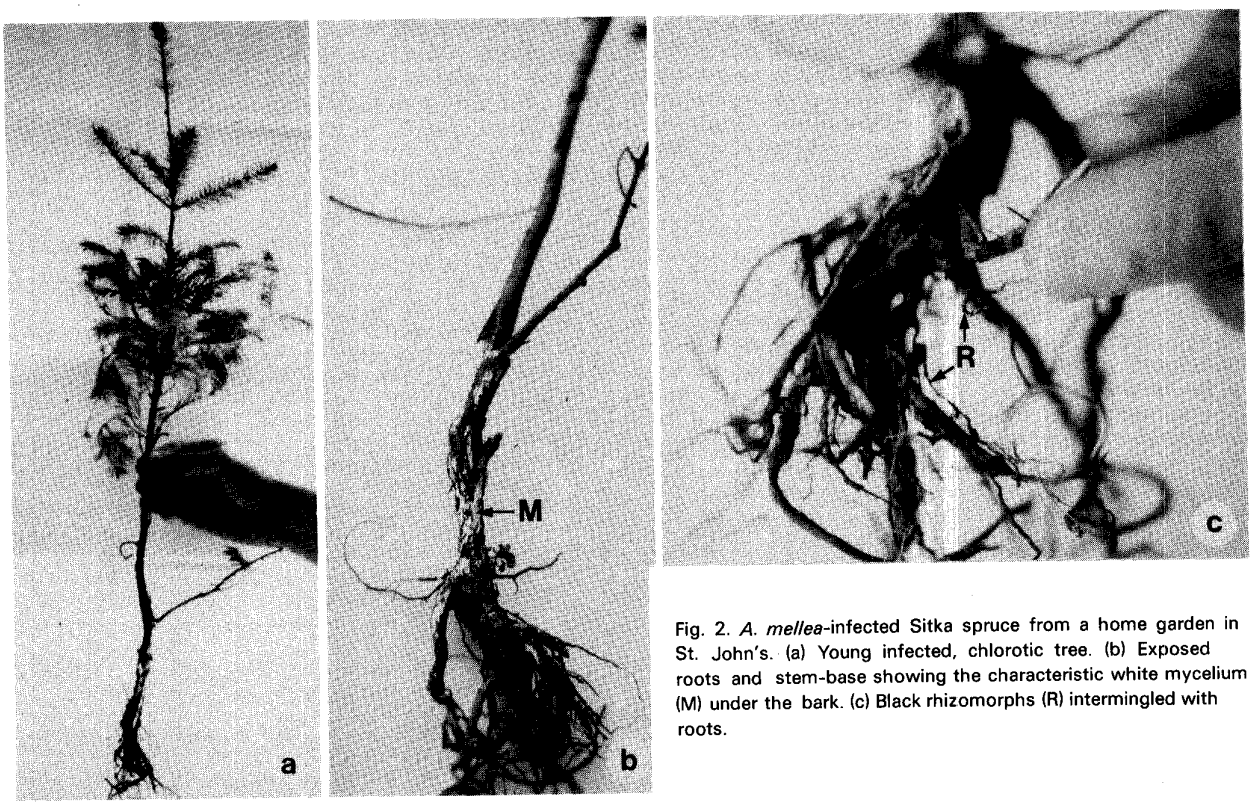

Fig. 2. *A. mellea*-infected Sitka spruce from a home garden in St. John's. (a) Young infected, chlorotic tree. (b) Exposed roots and stem-base showing the characteristic white mycelium (M) under the bark. (c) Black rhizomorphs (R) intermingled with roots.

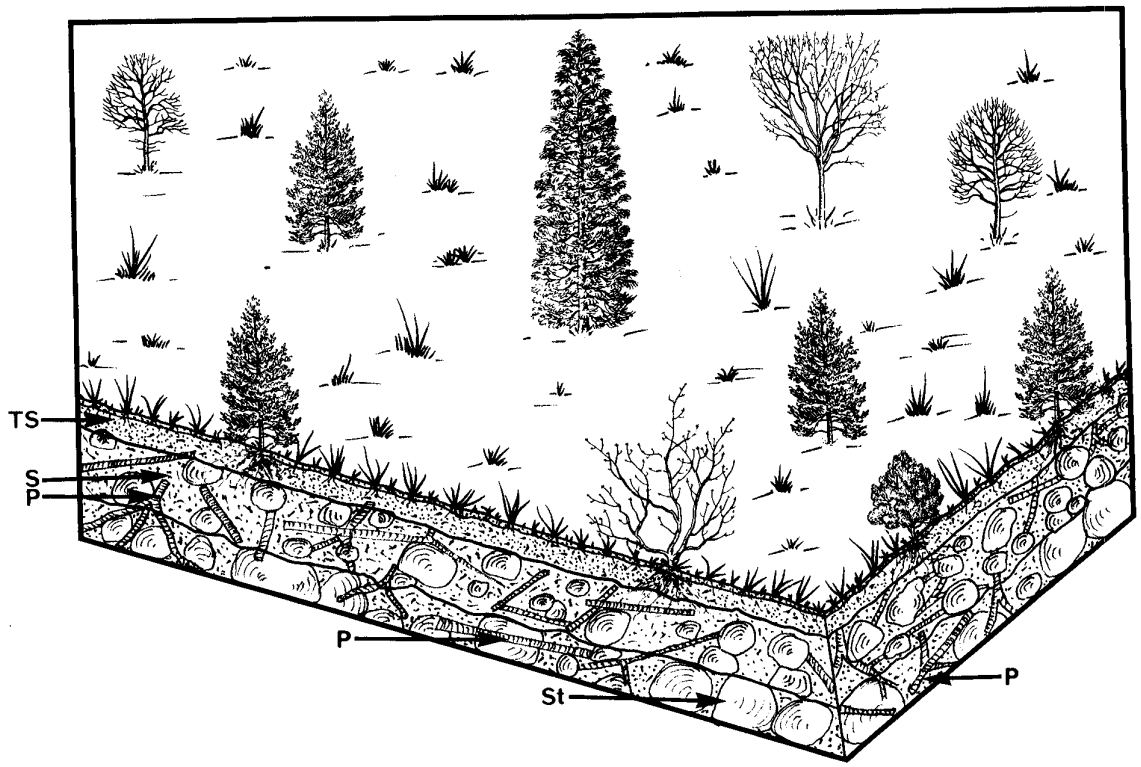

Fig. 3. Schematic diagram of a portion of a home garden showing trees and underground constituents of the fill (Top soil — TS, soil — S, gravel and stones (St), and buried plant materials (P).

various types of stress, such as improper site; drought, frost, poor drainage; driveway, sidewalk and road salting; site changes as a result of construction or fill; urban environment pollutants; severe or repeated defoliation by insects or leaf diseases. These stress factors predispose trees, reduce their vigour and make them more susceptible to the lethal attack of *A. mellea*.

Prospects and control strategies

The record of *A. mellea* on urban trees in Newfoundland is rare and scattered, but has added another perspective to the root rot problem. The pathogen may also threaten trees in home gardens and landscapes developed on cutovers and where *A. mellea*-infected stumps, roots and other plant remains have been buried in the fill; the fungus can survive for several years in such roots and stumps (Singh-unpublished data). Interest in urban trees and shrubs and their management have increased with expanding urbanization, but with increasing variety of stress factors, this disease may become more important in the future.

The following recommendations are offered to developers, homeowners and landscape horticulturists to prevent or minimize damage: (i) Avoid the use of infected stumps, roots and other plant remains in the fill. (ii) Avoid the use of tree species which are susceptible to urban stresses. Tattar (1978) recommended a selection of smaller and slower-growing trees which adapt more effectively to stress and demand less of their site. (iii) Keep the newly planted trees in good health and vigorous growth through proper planting and adequate fertilizer and water supply. (iv) Avoid or alleviate the effects of stress, disturbance or injury during planting or later stages, which may reduce the vigour of the tree. (v) Prevent defoliation by spray with chemical or biological pesticides (Wargo, 1978 & 1981).

Literature cited

1. Damman, A.W.H. 1964. Some forest types of central Newfoundland and their relation to environmental factors. For. Sci. Monogr. 8:1-62.
2. Schoeneweiss, D.F. 1975. Predisposition, stress and plant disease. Ann. Rev. Phytopathology 13:193-211.
3. Schoeneweiss, D.F. 1981. The role of environmental stress in diseases of woody plants. Plant Disease 65:308-314.
4. Singh, Pritam. 1975. Armillaria root rot: Distribution and severity in softwood plantations in Newfoundland. Acta Phytopathologica Acad. Sci. Hung. 10:389-406.
5. Singh, Pritam. 1977. Growth and development of rhizomorphs of *Armillaria mellea* in Newfoundland forest soils. Proc. Amer. Phytopath. Soc. 4:84.
6. Singh, Pritam. 1981a. *Armillaria mellea*: Growth and distribution of rhizomorphs in the forest soils of Newfoundland. European J. For. Path. 11:108-220.
7. Singh, Pritam. 1981b. *Armillaria mellea*: Distribution and hosts in Newfoundland and Labrador. Can. Plant Dis. Surv. 61:31-36.
8. Tattar, T.A. 1978. Diseases of shade trees. Academic Press, New York. 361 p.
9. Wargo, P.M. and D.R. Houston. 1973. Infection of defoliated sugar maple trees by *Armillaria mellea* (Abstr.). Phytopathology 63:209.
10. Wargo, P.M. 1978. Insects have defoliated my tree — Now what's going to happen. J. Arboriculture 4:169-175.
11. Wargo, P.M. 1981. Defoliation and secondary-action organism attack: with emphasis on *Armillaria mellea*. J. Arboriculture 7:64-69.

Low-temperature fungi associated with Alfalfa root and crown rot in central Alberta

D. Stelfox[1] and M. Bertsch[2]

Samples of 2, 3, and 4-year old alfalfa were examined in early spring and late autumn in 1978 to determine the relative prevalence of low-temperature fungi associated with the root and crown rot disease complex. The most frequently isolated fungi were *Fusarium* sp. and *Cylindrocarpon* sp. *Plenodomus* sp. was isolated more frequently in spring than in autumn.

Can. Plant Dis. Surv. 63:1, 7-11, 1983.

Des échantillons de luzerne plantée depuis 2, 3 et 4 ans furent examinées tôt au printemps et tard à l'automne 1983, afin de déterminer l'abondance relative des champignons de basse température associés au complexe de maladies causant la pourriture de la racine et du collet. *Fusarium* sp. et *Cylindrocarpon* sp. furent isolés le plus souvent alors que *Plenodomus* sp. fut isolé plus fréquemment au printemps qu'à l'automne.

Introduction

Winter injury with reduced vigour is one of the main factors involved in lowering yields of alfalfa (*Medicago sativa* L.) in central and north central Alberta. Previous reports associate various low temperature fungal pathogens with alfalfa winter injury and subsequent decline in this region (2, 3, 4, 5, 7, 10, 11). The major fungi are involved with winter crown rot (5), crown rot (7), and various root rots (2, 3, 9). Some, such as *Fusarium* sp. and *Cylindrocarpon* sp. were also found to be prevalent in studies of crown and root rotting fungi of alfalfa in Manitoba and Quebec (1, 8). During a 6-year period over 60% of alfalfa stands in west central Alberta (5) were shown to be affected by winter crown rot. In the Peace River area, a survey (9) revealed a severe level of root rot in 68% of fields inspected.

Increased requests for diagnosis of damage to legumes and perennial grasses in central Alberta led to field and laboratory investigations from the Plant Industry Laboratory, Edmonton in 1977 and 1978. Severe depletion in 3 and 4-year old alfalfa stands was noticeable in the early summer both years. Greatest damage appeared to occur in the Barrhead — Westlock area (Fig. 1) where several thousand acres of uneconomical stands were plowed under in June 1978. Winter of 1977-78 was characterized by a rather light and short-lived snow cover. Affected regions were subjected to extremely low temperatures in early December 1977.

The objective of this study was to determine the identity and prevalence of fungi associated, in spring and late autumn, with crown and root rots of mature alfalfa plants in two regions of central Alberta. Samples were lifted in late May to early June and again in late October to early November 1978. The plant material was refrigerator-stored in the laboratory and cultures obtained were studied over a period of 10 months.

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Materials and Methods

Two, 3 and 4-year old alfalfa plants showing symptoms of foliage yellowing and stunting were selected for the study from 42 fields in the grey-wooded and black soil zones of central Alberta. Tissue from specimens with obvious crown and root lesions, but not in advanced stages of decay, was used for plating purposes. Most samples were transported from the field in portable coolers, then stored in the laboratory at 5°C. A few samples were mailed to the laboratory by extension workers.

Estimates of the top growth of plants were recorded. Top growth was then removed just above the crown area. Surface soil was manually removed, then roots were washed for 15-30 minutes under running tap water to remove soil remnants. After excess water was absorbed by paper towels, outer cortical tissue was peeled away at lesion sites using a sterile scalpel. Small portions of underlying root or crown tissue from lesion margins were selected for plating. Outer cortical tissue was plated, where lesions were shallow or superficial.

Isolations were made on potato sucrose agar (PSA), on acidified potato sucrose agar (PSA-A), and on a selective antibiotic medium (PP) containing pimaricin and pentachloro-nitrobenzene (PCNB). Subculturing utilized PSA, PSA-A, PP and cornmeal agar (CMA). Plates were incubated at 0,5, 10, 15, and 20°C for a minimum of 2 weeks. 0° and 5°C plates were incubated for as long as 20 weeks. At 3-day intervals initial plates were examined and fungal colonies were subcultured. Some cultures remained in a controlled temperature chamber for several months. Duplicates were maintained on PSA or CMA slants at 10°C.

Results

Fusarium spp. were among the most common isolates associated with damaged alfalfa crown and root tissue. They were isolated from 50% or more of rotted tissue, and from nearly 40% of tissue showing punctures, splits, brown flecks, and dark necrotic bands (Table 1). They occurred in 76 and 38% of samples from Regions I and II respectively. One type of

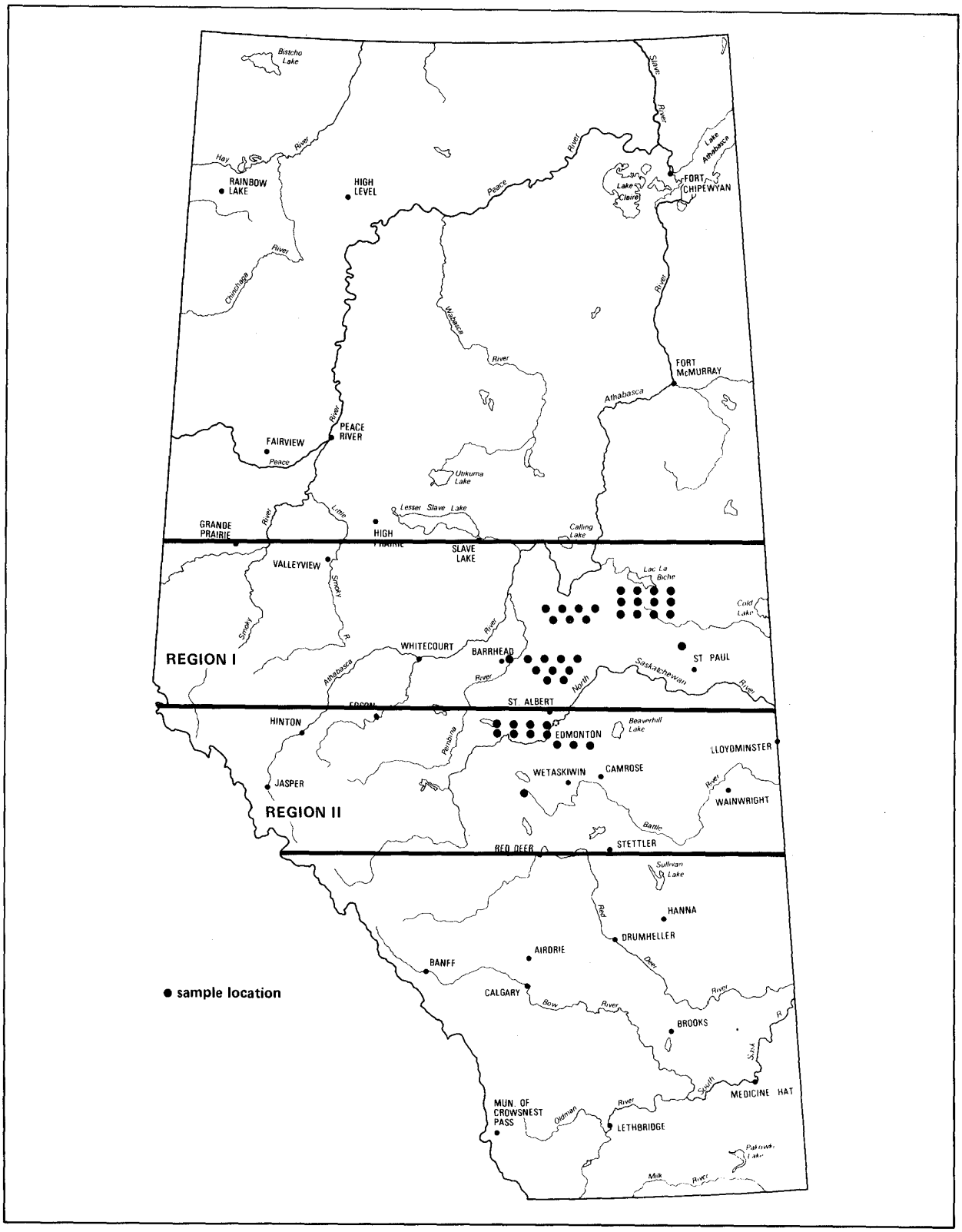

Figure 1. The areas of sampling locations in Regions I and II of central Alberta.

Table 1. Percentage of 42 alfalfa samples from which specific fungi were isolated from crown and root lesions in two regions of central Alberta in 1978

Types of Lesion	Fusarium	Cylindrocarpon	Plenodomus	Pythium	Rhizoctonia	LTB	Botrytis
Crown rot	63	6	10	22	2	2	8
Root rot	50	64	28	0	14	0	0
Root punctures, splits, flecks, bands	37	84	0	5	5	0	0

Fusarium sp. formed tiny sclerotia on host tissue and in culture. In 13 of 34 samples involving rotted crowns, only *Fusarium* sp. was isolated. In 10 instances it was associated in crown tissue with other pathogenic fungi such as *Plenodomus* sp., *Cylindrocapon* sp., *Pythium* sp., and *Rhizoctonia* sp. In damaged root tissue it was isolated alone from one sample. One of the disorders from which *Fusarium* sp. alone was isolated is crown rot of a 2-year old plant (Fig. 2A). All isolates developed well on plates in the laboratory at near-freezing temperatures.

Cylindrocarpon sp. occurred in 64% of the cases involving root rot and 84% involving root banding, splits and punctures (Table 1). This fungus was isolated from 6% of tissues involving crown rot. It occurred in 69 and 85% of samples from Regions I and II respectively. The most commonly isolated species was *C. ehrenbergi* with its reddish-brown sclerotia-like stromata on the surface of advanced root-rot tissue (Fig 2B). *C. destructans*, forming no stromata in culture or on host tissue, also was obtained from rotted root tissue. Both species were associated with more than two-thirds of alfalfa samples from Regions I and II. There was a noticeable effect of season on incidence of *Cylindrocarpon* spp. isolations (Table 2).

Plenodomus meliloti, which usually required 8 to 12 weeks plate incubation to induce pycnidial spore formation, was isolated from 29 and 23% of samples from Regions I and II respectively. It occurred most commonly in root-rotted tissue (Fig. 2D), but sometimes in connection with crown rot (Fig. 2E). The numerous tiny dark brown to black pycnidia formed on or within affected tissue. Beaks frequently developed (Fig. 2C) and extruded spores, following several weeks of low temperature incubation. The fungus was always associated in brown root rot tissue, with *Fusarium* sp. and *Cylindrocarpon* sp. and its prevalence was about the same in Region I (29%) as in Region II (23%) samples. As in the case of *Cylindrocarpon* sp. it was isolated with much greater frequency in spring than in autumn.

Pythium spp. were isolated from damaged crown tissue but not from root rot samples (Table 1). No attempt was made to identify the several different isolates originating from 8 samples most of which came from Region II. Other phycomycetes, generally considered to be soil saprophytes, were isolated.

Rhizoctonia sp. was less prevalent than *Pythium* spp. in spring and autumn samples, and occurred in none from Region II. In the 4 instances where the fungus was present, it was always associated with either *Cylindrocarpon* sp. or *Fusarium* sp. On only one occasion was a low-temperature basidiomycete (LTB) retrieved, and it occurred with other pathogens in crown rot tissue. LTB was recently identified as *Coprinus* sp. (13). A *Botrytis* sp. was isolated once from severely rotted crown tissue and once from reddish-brown flecks on taproot tissue. Several isolates of miscellaneous unidentified fungi were obtained during the study.

Discussion

The results of this study agree with reports (5, 7) of a complex of low-temperature fungi being associated with alfalfa crown and root rot in central Alberta. The virtual absence of LTB isolates from these 1978 samples was likely due to the relatively light and short-lived snow cover during the winter of 1977-78 (5). LTB is reported to be the most destructive fungus attacking alfalfa in spring (2). Very low temperatures in early December 1977, following a prolonged spell of mild weather, may have predisposed alfalfa stands to

Table 2. Percentage of alfalfa samples from which specific fungi were isolated in spring and autumn in two regions of central Alberta in 1978

Season	Fusarium	Cylindrocarpon	Plenodomus	Pythium	Rhizoctonia	LTB	Botrytis	No. Samples
Spring	61	74	39	17	9	4	4	24
Autumn	61	17	11	6	11	0	5	18

late winter-early spring infections by other low temperature pathogens.

Cylindrocarpon sp. is reported (3) as one of the most virulent pathogens attacking alfalfa during early spring in Alberta. Elsewhere in Canada (1, 8) the fungus is considered to be of importance on alfalfa after the first year's growth. Root infections begin at the first sign of soil thawing and proceed through 3 stages from water-soaked tissue to light brown and, finally, to dark brown necrosis (3).

The brown root rot incitant, *Plenodomus meliloti*, has been reported (11) to be one of the most important pathogens associated with "winter-kill". It is native to the Peace River Region as well as to central and north-central Alberta. Infected lesions appear on taproots as soon as surface soil thaws in the spring, or even during mild spells in late winter. Growth and development of pycnidia of the fungus in Alberta have been studied and illustrated (10). Partial recovery of plants severely affected by brown root rot is due to the formation of new roots produced near the crown, a condition frequently observed on samples collected in the Barrhead-Westlock region. This was one of the root abnormalities associated with stunted plants.

At least five pathogenic low-temperature *Fusarium* spp. have been found to cause root injury of alfalfa in Alberta (4). The two which are widespread and cause serious damage in early spring are *F. avenaceum* and *F. arthrosporioides*. In early stages of infection root symptoms can readily be confused with those attributed to *Plenodomus* sp. and *Cylindrocarpon* sp. In the present study, all three of these genera have been isolated from a single root lesion. Where *Fusarium* sp. alone was isolated from the margin of rotted crown tissue, it may have overrun tissue originally invaded by another pathogen. Early spring and late autumn sampling were chosen, since low temperature fungal pathogens are difficult to isolate during summer months. *Plenodomus* spp., in particular, is responsive to isolation attempts in early spring.

Some of the *Pythium* spp. reported (12) to be associated with seedling infection in alfalfa in central Alberta were similar in appearance to isolates of the present survey. The fact that no isolates were obtained from below-ground portions of these mature plants may have been due to loss of rootlets and necrotic root tips when lifting samples.

Rhizoctonia solani Kuhn is known to be part of a complex associated with *Fusarium* spp. in crown bud rot in Quebec, Manitoba, and Alberta (1, 8, 6). Crown rot was evident on all samples from which *Rhizoctonia* sp. was isolated in the present study, and typical taproot symptoms characterized below-ground tissue from which the fungus was retrieved. Damage apparently had progressed over a period of one or two growing seasons, judging by the extent and color of root lesions.

Literature cited

1. Aubé, C. and J. Deschênes, 1967. Crown and root rots of alfalfa and red clover and the relative prevalence of associated fungi. Plant Dis. Reptr. 51:573-577.
2. Broadfoot, W.C. and M.W. Cormack. 1941. A low-temperature basidiomycete causing early spring killing of grasses and legumes in Alberta. Phytopathology 31:1058-1059.
3. Cormack, M.W. 1937. *Cylindrocarpon ehrenbergi* Wr., and other species, as root parasites of alfalfa and sweet clover in Alberta. Can. J. Res. C. 15:403-424.
4. Cormack, M.W. 1937. Fusarium spp. as root parasites of alfalfa and sweet clover in Alberta. Can. J. Res. C. 14:493-510.
5. Cormack, M.W. 1948. Winter crown rot or snow mold of alfalfa, clovers and grasses in Alberta. Can. J. Res. C. 26:71-85.
6. Hawn, E.J. 1958. Studies on the epidemiology of crown bud rot of alfalfa in southern Alberta. Can. J. Bot. 36:239-250.
7. Hawn, E.J., W.B. Berkenkamp, and J.B. Lebeau. 1981. Evaluation of losses in alfalfa hay production cause by crown rot. Can. J. Plant Pathol. 3:103-105.
8. McDonald, W.C. 1955. The distribution and pathogenicity of the fungi associated with crown and root rotting of alfalfa in Manitoba. Can. J. Agr. Sci. 35:309-321.
9. McKenzie, J.S. and J.G.N. Davidson. 1975. Prevalence of alfalfa crown and root diseases in the Peace River region of Alberta and British Columbia. Can. Plant Dis. Surv. 55:121-125.
10. Netolitsky, H.J. 1965. Cytology, histology, and pathology of *Plenodomus meliloti*. M. Sc. thesis, University of Alberta, Edmonton, Alberta.
11. Sanford, G.B. 1933. A root rot of sweet clover and related crops caused by *Plenodomus meliloti* Dearness and Sanford. Can. J. Res. 8:337-348.
12. Stelfox, D. and J.R. Williams. 1980. Pythium species in alfalfa fields in central Alberta. Can. Plant Dis. Surv. 60:35-36.
13. Traquair, J.A. 1980. Conspecificity of an unidentified snow mold basidiomycete and a *Coprinus* sp. in the section *Herbicolae*. Can. J. Plant Pathol. 2:105-115.

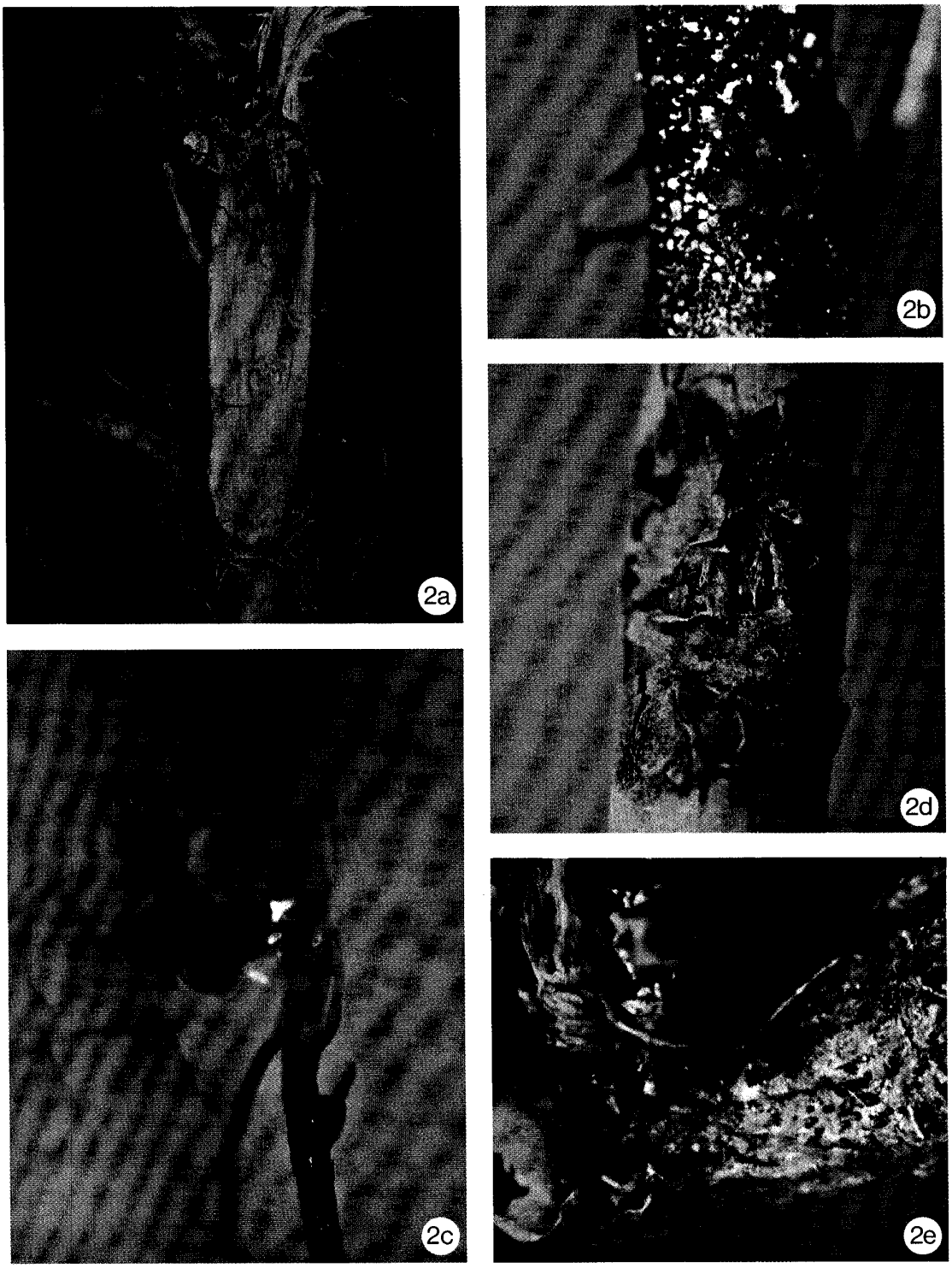

Figure 2 (A to E). Symptoms and signs of crown and root disorders: **(A)** 2-year old plant with crown rot, **(B)** reddish-brown sclerotia-like stromata of *C. ehrenbergi,* **(C)** beaks formed on pycnidia of *P. meliloti,* **(D)** typical advanced brown root rot symptoms, **(E)** Pycnidia formed on rotted crown tissue.

Viroids and their potential danger to potatoes in hot climates[1]

R.P. Singh[2]

Viroids are the smallest agents known to be pathogenic to plants. Unlike viruses, they are devoid of a protective protein coat and are composed entirely of circular ribonucleic acid of low molecular weight (ca. 85,000-130,000 *d*). In spite of their small size, viroids cause serious diseases of avocado, chrysanthemum, citrus, coconut, cucumber, hop, potato and tomato. Although viroid diseases have been reported in both tropical and temperate regions of the world, they induce more severe symptoms at high temperatures. They have wide host ranges and at least six different viroids can induce symptoms in potato similar to those caused by the potato spindle tuber viroid. This creates difficulties in making positive identifications. Viroids spread readily by contaminated knives, sickles, and hilling and cultivating equipment. Viroids are detected by bioassays, by gel electrophoresis on polyacrylamide gels or by nucleic acid hybridization tests. Viroids are not a potential danger to potato crop in hot climates.

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Les viroides sont les plus petits agents phytopathogènes connus. Contrairement au virus, ils sont composés entièrement d'acide ribonucléique ciruclaire à bas poid moléculaire (ca. 85,000-130,00 *d*) et ne possèdent pas d'enveloppe protéinique protectrice. En dépit de leur petite taille, les viroides causent de graves maladies chez l'avocado, le chrysanthème, les citrus, la noix de coco, le concombre, le houblon, la pomme de terre et la tomate. Quoique les maladies causées par les viroides aient été signalées dans les régions tempérées et tropicales du monde, les symptômes induits sont plus sévères en climat chaud. Ils peuvent parasiter de nombreuses plantes et au moins six différents viroides peuvent induire chez la pomme de terre des symptômes semblables à ceux causés par le viroide de la filosité des tubercules, ce qui rend son identification difficile. Les viroides sont propagés facilement par les couteaux, faucilles, buteuse et équipement aratoire contaminés. Ils peuvent être détectés à l'aide de tests biologiques, d'électrophorèse sur gel de polyacrylamide ou de tests d'hybridation de l'acide nucléique. Les viroides ne posent pas une grande menace pour la culture de la pomme de terre en climat chaud.

Introduction

A major new development in plant disease research in recent years has been the discovery of the viroid nature of several serious plant diseases. The first viroid was discovered simultaneously but independently in 1971 by Diener (6) and Singh and Clark (49) who were working on the spindle tuber disease of potatoes (9, 48). Since then, viroid-like agents have been demonstrated for ten additional plant diseases. These diseases and their viroids are: avocado sunblotch, ASBV (3, 55); chrysanthemum chlorotic mottle, ChMV (38); chrysanthemum stunt, CSV (8); citrus exocortis, CEV (40, 45); coconut cadang-cadang, CCCV (35, 36); cucumber palefruit, CPFV (42, 56); hop stunt, HSV (43, 44); tomato bunchy top, TBTV (58); tomato planta macho, TPMV (11); and a viroid carried symptomlessly in *Columnea erythrophae* (30).

There are several reviews available that discuss various aspects of viroid research (5, 7, 16). Therefore, an attempt will be made here to point out the biological similarities of viroids, or to speculate on the impact certain ones may have on potato crops in hot climates.

Occurrence and importance of viroid diseases

Although the first viroid was discovered in the temperate regions of North America, viroid diseases are of worldwide occurrence (Table 1) and have caused serious economic losses in tropical climates. For example, cadang-cadang disease of coconut has caused an estimated loss of 12 million palm trees and is considered to be the major threat to coconut production in the Philippines (35). Hop stunt disease was detected in 17% of the total acreage of hops in Japan's Fukushima Prefecture in 1968 where some garden had up to 60% of the plants infected (59). Potato gothic (= spindle tuber) has been widespread and has researched infection rates of as high as 54% in some provinces of the Ukraine S.S.R. (15). Although spindle tuber is now a minor disease in North America, infection rates of 25-90% were observed in the 1920s (7) and PSTV can cause as much as 65% reduction in the yield of infected plants (7, 52).

Potato spindle tuber viroid in Canada has become rare even in the processing and table-stock field as compared to late sixties (52). The major seed-producing provinces of Canada (New Brunswick and Prince Edward Island) require planting of certified seed for processing and table-stock fields and Canadian potato certification service practices "zero-tolerance" for PSTV in the field. These two measures have reduced the incidence of PSTV in the seed field sharply and in last decade no fields have been rejected because of

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[3] *Unpublished observations of the author in co-operation with Dr. R.A. Owens.*

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Table 1. Occurence of viroid diseases throughout the world.

Disease	Countries
Avocado sunblotch	Australia, Israel, Peru, South Africa, U.S.A., Venezuela
Citrus exocortis	Argentina, Australia, Brazil, Corsica, Israel, Japan, Spain, South Africa, U.S.A.
Chrysanthemum stunt	Australia, Canada, India, Japan, The Netherlands, United Kingdom, U.S.A.
Chrysanthemum chlorotic mottle	U.S.A.
Columnea viroid	U.S.A.
Coconut cadang-cadang	Philippines
Cucumber pale fruit	The Netherlands
Hop stunt	Japan
Potato spindle tuber	Argentina, Brazil, Canada, China, Chile, Peru, Scotland, U.S.A., U.S.S.R., Venezuela
Tomato bunchy top	Ivory Coast, South Africa
Tomato planta macho	Mexico

PSTV. The only possible occurrence of PSTV at present is potato breeding institutions, where germplasms from different countries are maintained and whose diversity of morphological characteristics makes diagnosis of PSTV difficult. Even these institutions in Canada are testing parental material for PSTV on a large scale which has further minimized the risk of PSTV in potato production.

The nature of viroids

Viroids are the smallest agents known to be pathogenic to higher plants. Their molecular weight is 85,000 to 130,000 *d*, and their nucleotide chain is only 243-359 nucleotides long (Table 2). Electron microscopy shows native viroids as rod-like molecules and denatured viroids as covalently closed single-stranded RNA species (18, 42). In a proposed model of their secondary structure, the circles have been shown to elongate and form a defective double-helix, in which double helical segments are separated by short unpaired stretches (18, 37). Two of the sequenced viroids, i.e., PSTV and CSV have 69% sequence homology, but the third viroid ASBV which was sequenced recently shows a much lower (18%) sequence homology with PSTV and CSV (54). This suggests divergence in their evolution and subsequent adaptation.

Table 2. Molecular weights and chain length of viroids.

Viroid	Molecular weight	Chain length
Potato spindle tuber	123,325 (18)*	359 (18)
Chrysanthemum stunt	106-127,000 (17)	356 (54)
Avocado sunblotch	85-100,000 (31)	247 (54)
Coconut cadang-cadang	105,000 (36)	243 (54)
Cucumber pale fruit	110,000 (42)	330 (42)
Hop stunt	99,000 (28)	296 (28)
Citrus exocortis	119,000 (42)	357 (42)

*References

Apparently viroids do not have tertiary structures, because the same binding sites are available to dyes at low and high ionic strength (16, 18). Also, the transfer RNA has free access to anticodon binding loops all over the molecules (16, 37).

Unlike viruses, viroids have no protein coat and they are merely a short strand of RNA. They do not carry enough of the genetic code to accomplish their own replication. Therefore, the question arises as to how the viroids are able to multiply. A complete answer cannot be given, but it is certain that viroid replication occurs without the assistance of helper virus (6) and the viroid itself does not act as messenger RNA (4, 19). This suggests that viroids rely upon enzymes already present in the host plant for their replication. In fact, it has been observed that DNA-directed RNA polymerase II from several plant sources will transcribe full length viroid RNA *in vitro* (34). Combined with earlier evidence that ξ-amanitin inhibits viroid replication, there is a strong possibility of this enzyme alone being involved in replication *in vivo* (34). Another line of indirect evidence supporting a role for RNA polymerase is nutritional studies (53) where manganese stimulated the PSTV symptoms and concentration in infected plants. Since manganese and not magnesium stimulates PSTV concentration, this effect could be similar to that observed in the synthesis of fragmented $Q\upsilon$RNA or foreign RNA in the presence of $Q\upsilon$ replicase (23).

Viroids are hot climate pathogens

Several studies have been done, using potato and tomato as host plants, to determine the effect of various environmental factors on PSTV. It is generally agreed that air temperature is by far the most important factor because of the profound effect it has on foliage symptoms (13, 21, 27, 41).

In potato, the foliage symptoms of PSTV are more severe if the plants are started in the field, and especially if they are planted late in the season when weather is warm. Under such conditions the symptoms persist throughout the growing season. However, if the potatoes are started when the weather is cooler, the symptoms may never appear (13). High air temparature not only exaggerates the symptoms on aerial parts, but it has been shown to double the amount of viroid synthesized in potato tissues at 30°C as compared to 25°C (27). A similar response to high air temperature has also been observed in PSTV-tomato combinations (21, 27, 41).

High temperatures have also been used to develop symptoms and aid in indexing several viroid diseases. Cucumber pale fruit was detected more reliably when night and day temperatures

remained 27 to 32°C (56), and the incubation period was reduced from 76 days at 20°C to 12-21 days at 30°C (56). In chrysanthemum, the highest infectivity with ChMV was obtained when plants were maintained at 21-32°C (22). Hop stunt viroid in cucumber showed marked symptoms at 33°C and only faint symptoms at 21°C. The incubation period was also reduced to 17 days at 33°C, as compared with 38 days at 21°C (44). The remarkable effect of temperature on ASBV in avocado was also noted recently (2). The incubation period of this viroid was reduced to 90-158 days at 28-30°C and all plants developed symptoms; whereas it took 350 days to complete incubation at 18-20°C and only 2 plants developed symptoms. Like PSTV, the avocado plants developed symptoms rapidly when they were subjected to a high temperature soon after inoculation with ASBV and then transferred to cool temperatures. Those subjected to cooler temperature at the time of inoculation and then transferred to high temperature required a much longer time to develop symptoms (2).

Not only do viroids like PSTV multiply very rapidly at high temperatures and cause more severe symptoms in their host, but their rate of multiplication at low temperatures may be so low that the viroid is eliminated from infected plants (24).

Transmission and spread of viroids

Although viroids are naked RNA, they are mechanically transmissible to plants. Some are transmitted readily while others are not transmitted without special conditions. CPFV (56), CEV (12) and ASBV (31) can be transmitted by razor slashing of stems. PSTV (10, 46, 51), and ASBV (57) are transmitted through pollen and seeds, in addition to normal sap transmission.

Viroids are often spread in the field by contaminated tools and cultivating machinery. For example, PSTV was shown to be transmitted by cutting healthy seed with a knife previously used to cut infected tubers (14). In one study 80-100% infection with PSTV was achieved by brushing actively growing healthy plants with diseased foliage (26); in another study, one hundred per cent of the plants were infected when excessive contact of large vines was made with PSTV contaminated cultivating and hilling equipment (25). The citrus exocortis viroid has been difficult to transmit by conventionel sap inoculation of leaves, yet it can readily be transmitted with contaminated budding knives (12). Contaminated knives, tools, and bare hands used during cultural operations are considered to be the chief sources of spread of chrysanthemum stunt (1). Cucumber pale fruit viroid has been spread by pruning operations in the greenhouse (56), and spread of hop stunt in the garden has also been demonstrated when contaminated sickles or bare hands were used to dress or pull shoots (59).

Symptoms and host range

The range of symptoms exhibited by viroid diseases is similar to that of viruses, except that most viroid infections induce stunting of some kind. Stunting of entire plants is common, and there are usually other symptoms such as smaller upper leaves, shortened internodes and an exaggerated upright appearance in plants infected with PSTV, CEV, CSV, CCMV, CPFV, HSV, TBTV, and TPMV. Malformed, dwarfed flowers are often observed on plants infected with CPFV and HSV. There is also a tendency of viroid-infected plants to produce fruits, tubers and cones which are pointed or more elongated than normal. Fruits infected with CCCV and tubers infected with PSTV usually carry longitudinal scarification or growth cracks. ASBV and CEV are known to cause streaking and splitting of bark in avocado and citrus. Discoloration of leaves, tubers, fruits, and stems have also been observed in other viroid-infected plants.

There is a wide host range for some viroids, whereas others are limited to infecting only one family of host plants (Table 3). Since host range can be affected by host adaptation or serial passaging as noted for CPFV (39), it is difficult to generalize on viroid host ranges. Several viroids may induce similar symptoms. For example, PSTV, CEV, CSV, CPFV, TBTV, TPMV, and *Columnea* viroids all induce epinasty of leaves and stunting of tomato plants. Viroids such as PSTV, CEV, CSV, CPFV, TBTV, TPMV and *Columnea* can infect and also cause similar symptoms in potato plants. PSTV, CEV, TBTV and *Columnea*[3] can induce similar local necrotic lesions in *Scopolia sinensis* plants (47, 50, 58).

Table 3. Host range of viroids.

Viroids	Families in which susceptible plants have been found
Avocado sunblotch	Lauraceae
Citrus exocortis	Compositae, Cucurbitaceae, Papilionaceae, Rutaceae, Solanaceae, Umbelliferae
Chrysanthemum stunt	Compositae, Cucurbitaceae, Solanaceae
Chrysanthemum chlorotic mottle	Compositae
Coconut cadang-cadang	Palmaceae
Columnea viroid	Gesneriaceae, Solanaceae
Cucumber pale fruit	Compositae, cucurbitaceae, Solanaceae
Hop stunt	Cucurbitaceae, Solanaceae
Potato spindle tuber	Boraginaceae, Campanulaceae, Caryophyllaceae, Compositae, Convolvulaceae, Dispaceae, Sapindaceae, Scrophulariaceae, Solanaceae, Valerianaceae
Tomato bunchy top	Compositae, Scrophulariaceae, Solanaceae
Tomato Planta Macho	Compositae, Solanaceae

Table 4. Detection of viroids.

Viroids	Bioassay	Page*	C-DNA**
Avocado sunblotch	*Persea americana* cvs. 'Hass', 'Collinson'	+	+
Citrus exocortis	Gynura aurantiaca	+	+
Chrysanthemum stunt	*Chrysanthemum morifolium* cv. 'Mistletoe' *Gynura aurentica*	+	+
Chrysanthemum chlorotic mottle	*Chrysanthemum morifolium* cv. 'Deep Ridge'	+	-
Columnea viroid	-	+	+
Coconut cadang-cadang	-	+	+
Cucumber pale fruit	*Cucumis sativus* cv. 'Sporu' *Lycopersicon esculentum* cv. 'Rentita'	+	-
Hop stunt	*Cumumis sativus* cv. 'Suuyou'	+	-
Potato spindle tuber	*Lycopersicon esculentum* cv. 'Sheyenne', 'Rutgers' *Scopolia sinensis*	+	+
Tomato bunchy top	*Lycopersicon esculentum*	+	-
Tomato planta macho	*Lycopersicon esculentum* cv. 'Rutgers'	+	-

* Polyacrylamide gel electrophoresis.
** Complementary DNA test so far reported.

Some viroids, such as PSTV, CEV, CSV, CPFV, and TPMV can infect *Gynura aurantiaca* plants, but induce different symptoms. While PSTV and CEV induce characteristic symptoms in this species (47, 50) CSV, CPFV and TPMV are carried symptomlessly (11, 39).

Detection of viroids

Various methods for detecting viroids are summarized in Table 4. Except for *Columnea* and coconut viroids, all of them can be tested with indicator plants. All viroids can also be tested by the polyacrylamide gel electrophoresis (PAGE) (27, 33), and several of them can be assayed by complementary DNA techniques (29, 30, 32) (Table 4). Environmental requirements differ with each viroid-host combination, but high temperature is generally a prerequisite for all test plants. *Scopolia sinensis* is an exception to this rule, because it is a better indicator within a temperature range of 18-21°C (47).

Various modifications of the PAGE procedure have been successfully used in viroid testing, and a recent modification (33) enables the entire test to be completed within a day. However, PAGE procedures have not been satisfactory in testing dormant tuber tissues. In one of our experiments only 24 of 36 PSTV-infected tubers were detected by PAGE procedures. On the other hand, we have used the recently developed nucleic acid hybridization test (29), to detect PSTV in individual dormant tubers, and in composite samples of twenty tubers in which only one was infected with PSTV.[3] In addition, we have used this technique to detect PSTV in true seeds obtained from an infected parent. Our studies have thus confirmed that the nucleic acid hybridization tests to be more sensitive than the PAGE tests, as has also been observed for other viroids (32).

Potential danger to potatoes in hot climates

From the foregoing discussion of environmental effects on replication, spread, and host range of viroids, one may conclude that potatoes are susceptible to viroids in hot climates, and that these organisms may pose a significant threat to successful production. However, when one considers the history of some of the more common viroid diseases, a different picture emerges. Several viroids such as avocado sunblotch, citrus exocortis, coconut cadang-cadang and tomato bunchy top have been known to existe for more than 40 years in countries such as Australia, Israel and South Africa and yet there have been no reports of significant losses in their potato crops because of viroid diseases. This suggests that viroids are like viruses (20) in that they can adapt to a wide range of climates and host plants. Certainly, a trend toward host adaptation was observed with CPFV (39).

Interactions between plants, viruses and vectors are greatly affected by environmental conditions (20). The climate imposes restrictions on survival systems by influencing the number of vectors that are present, and a major difference between tropical and temperate regions is the greater range of vectors in the former (20). As shown earlier, viroids are greatly influenced by high temperatures and, therefore, the

tropical climate will favour their synthesis. This, in turn, results in more obvious symptoms and weaker plants. Diseased plants are easier to detect and remove, and thus they are not permitted to remain and perpetuate the disease through successive crops as they do in temperate regions. Thus, there would be less of an effect on potato crop in hot climates.
Unlike viruses, there are no known efficient insect vectors of viroid diseases. Therefore, even with the greater number and variety of vectors in tropical climates (20) they should not be considered a factor in considering the potential for viroid diseases in the potato crop.

Literature cited

1. Brierly, P. and F.F. Smith. 1951. Chrysantheum stunt. Flor. Rev., 107(2778):27-30.
2. DaGraca, J.V. and S.P. van Vuuren. 1981. Use of high temperature to increase the rate of avocado sunblotch symptom development in indicator seedlings. Plant Dis. 65:46-47.
3. Dale, J.L. and R.N. Allen. 1979. Avocado affected by sunblotch disease contains low molecular weight ribonucleic acid. Australasian Plant Pathol. 8:3-4.
4. Davies, J.W., P. Kaesberg and T.O. Diener. 1974. Potato spindle tuber viroid. XII. An investigation of viroid RNA as a messenger for protein synthesis. Virology 61:281-286.
5. Dickson, E. 1979. Viroids: Infectious RNA in plants. *In* Nucleic Acids in Plants. Ed. T.C. Hall and J.W. Davies. CRC Press Inc. Boca Raton, Florida.
6. Diener, T.O. 1971. Potato spindle tuber "virus". IV. A replicating, low molecular weight RNA. Virology 45:411-428.
7. Diener, T.O. 1979. Viroids and Viroid Diseases. Wiley Interscience, New York. 252 pp.
8. Diener, T.O. and R.H. Lawson. 1973. Chrysanthemum stunt: A viroid disease. Virology 51:94-101.
9. Diener, T.O. and W.B. Raymer. 1967. Potato spindle tuber virus: A plant virus with properties of a free nucleic acid. Science 158:378-381.
10. Fernow, K.H., L.C. Peterson and R.L. Plaisted. 1970. Spindle tuber virus in seeds and pollen of infected potato plants. Am. Pot. J. 47:75-100.
11. Galindo, J., D.R. Smith and T.O. Diener. 1982. Etiology of planta macho, a viroid disease of tomato. Phytopathology 72:49-54.
12. Garnsey, S.M. and J.W. Jones. 1967. Mechnical transmission of exocortis virus with contaminated budding tools. Pl. Dis. Reptr. 51:410-413.
13. Goss, R.W. 1924. Effect of environment on potato degeneration diseases. Univ. Nebraska Agr. Exp. Sta. Res. Bull. 26, 40 pp.
14. Goss, R.W. 1926. Transmission of potato spindle tuber disease by cutting knives and seed piece contact. Phytopathology 16:233.
15. Grama, D.P. 1969. Distribution and infectivity of potato gothic in the Ukraine. Microbiol. J. 31:42-45 (translated from Ukrainian).
16. Gross, H.J. and D. Riesner. 1980. Viroids: a class of subviral pathogens. Angewandte Chemie 19:231-243.
17. Gross, H.J., H. Domdey and H.L. Sanger. 1977. Comparative oligonucleotide fingerprints of three plant viroids. Nucleic Acid Res. 4:2021-2028.
18. Gross, H.J., H. Domdey, C. Lossow, P. Jank, M. Raba, H. Alberty and H.L. Sanger. 1978. Nucleotide sequence and secondary structure of potato spindle tuber viroid. Nature 273:203-208.
19. Hall, T.C., R.K. Wepprich, J.W. Davies, L.G. Weathers and J.S. Semancik. 1974. Functional distinctions between the ribonucleic acids from citrus exocortis viroid and plant viruses: cell-free translation and aminoacylation reactions. Virology 61:486-492.
20. Harrison, B.D. 1981. Plant virus ecology: ingredients, interactions and environmental influences. Ann. Appl. Biol. 99:195-209.
21. Harris, P.S. and I.A. Browning. 1980. The effect of temperature and light on the symptom expression and viroid concentration in tomato of a severe strain of potato spindle tuber viroid. Potato Res. 23:85-93.
22. Horst, R.K. 1975. Detection of a latent infectious agent that protects against infection by chrysanthemum chlorotic mottle viroid. Phytopathology 65:1000-1003.
23. Haruna, I. and S. Spiegelman. 1965. Recognition of size and sequence by an RNA replicase. Proc. Nat. Acad. Sci., U.S.A. 54:1189-1193.
24. Lizarraga, R.E., L.F. Salazar, W.M. Roca and L. Schilde-Rentschler. 1980. Elimination of potato spindle tuber viroid by low temperature and meristem culture. Phytopathology 70:754-755.
25. Manzer, F.E. and D. Merriam. 1961. Field transmission of the potato spindle tuber virus and virus X by cultivating and hilling equipment. Am. Pot. J. 38:346-352.
26. Merriam, D. and R. Bonde. 1954. Dissemination of spindle tuber by contaminated tractor wheels and by foliage contact with diseased potato plants. Phytopathology 44:111.
27. Morris, T.J. and E.M. Smith. 1977. Potato spindle tuber disease: procedures for the detection of viroid RNA and certification of disease-free potato tubers. Phytopathology 67:145-150.
28. Ohno, T., J. Akiya, M. Higuchi, Y. Okada, N. Yoshikawa, T. Takahashi and J. Hashimoto. 1982. Purification and characterization of hop stunt viroid. Virology 118:54-63.
29. Owens, R.A. and T.O. Diener. 1981. Sensitive and rapid diagnosis of potato spindle tuber viroid disease by nucleic acid hybridization. Science 213:670-672.
30. Owens, R.A., D.R. Smith and T.O. Diener. 1978. Measurement of viroid sequence homology by hybridization with complementary DNA prepared *in vitro*. Virology 89:388-394.
31. Palukaitis, P., T. Hatta, D. McE Alexander and R.N. Symons. 1979. Characterization of a viroid associated with avocado sunblotch disease. Virology 99:145-151.
32. Palukaitis, P., A.G. Rakowski, D. McE Alexander and R.N. Symons. 1981. Rapid indexing of the sunblotch disease of avocados using a complementary DNA probe to avocado sunblotch viroid. Ann. Appl. Biol. 98:439-449.
33. Pfannenstiel, M.A., S.A. Slack and L.C. Lane. 1980. Detection of potato spindle tuber viroid in field-grown potatoes by improved electrophoretic assay. Phytopathology 70:1015-1018.
34. Rackwitz, H.R., W. Rhode and H.L. Sanger. 1981. DNA-dependent RNA polymerase II of plant origin transcribes viroid RNA into full length copies. Nature 291:297-301.
35. Randles, J.W. 1975. Association of two ribonucleic acid species with cadang-cadang disease of coconut palm. Phytopathology 65:163-167.
36. Randles, J.W. and T. Hatta. 1979. Circularity of the ribonucleic acids associated with cadang-cadang disease. Virology 96:47-53.
37. Riesner, D., K. Henco, U. Rokohl, G. Klotz, A.K. Kleinschonidt, H. Domdey, P. Jank, H.J. Gross and H.L. Sanger. 1979. Structure and structure formations of viroids. J. Mol. Biol. 133:85-115.
38. Romaine, C.P. and R.K. Horst. 1975. Suggested viroid etiology for chrysanthemum chlorotic mottle disease. Virology 64:86-95.
39. Runia, W. Th. and D. Peters. 1980. The response of plant species used in agriculture and horticulture to viroid infections. Neth. J. Pl. Path. 86:135-146.
40. Sanger, H.L. 1972. An infectious and replicating RNA of low molecular weight: the agent of the exocortis disease of citrus. Adv. Bioscience 8:103-116.
41. Sanger, H.L. and K. Ramm. 1975. Radioactive labelling of viroid RNA. Pages 230-253. *In* Modification of the Information Content of Plant Cells. Eds. R. Markham, D.R. Davies, D.A. Hapwood, and R.W. Horne, Amsterdam, North Holland.
42. Sanger, H.L., G. Klotz, D. Riesner, H.J. Gross and A.K. Kleinschmidt. 1976. Viroids are single-stranded covalently closed circular RNA molecules existing as highly base-paired rod-like structures. Proc. Natl. Acad. Sci., U.S.A. 73:3852-3856.

43. Sasaki, M. and E. Shikata. 1977. On some properties of hop stunt disease agent, a viroid. Proc. Japan Acad. 53B:109-112.
44. Sasaki, M. and E. Shikata. 1978. Studies on hop stunt disease I. host range. Rept. Res. Lab. Kirin Brewery Co., Ltd. 21:27-39.
45. Semancik, J.S. and L.G. Weathers. 1972. Exocortis disease: Evidence for a new species of "infectious" low molecular weight RNA in plants. Nature New Biol. 237:242-244.
46. Singh, R.P. 1970. Seed transmission of potato spindle tuber virus in tomato and potato. Am. Pot. J. 47:225-227.
47. Singh, R.P. 1973. Experimental host range of the potato spindle tuber "virus". Am. Potato J. 111-123.
48. Singh, R.P. and R.H. Bagnall. 1968. Infectious nucleic acid from host tissues infected with potato spindle tuber virus. Phytopathology 58:696-699.
49. Singh, R.P. and M.C. Clark. 1971. Infectious low-molecular weight ribonucleic acid. Biochem. Biophys. Res. Commun. 44:1077-1083.
50. Singh, R.P. and M.C. Clark. 1973. Similarity of host response to both potato spindle tuber and citrus exocortis viruses. FAO Plant Prot. Bull. 21:121-125.
51. Singh, R.P. and R.E. Finnie. 1973. Seed transmission of potato spindle tuber metavirus through the ovule of *Scopolia sinensis*. Can. Plant Dis. Surv. 53:153-154.
52. Singh, R.P., R.E. Finnie and R.H. Bagnall. 1971. Losses due to the potato spindle tuber virus. Am. Pot. J. 48:262-267.
53. Singh, R.P., C.R. Lee and M.C. Clark. 1974. Manganese effect on the local lesion symptom of potato spindle tuber 'virus' in *Scopolia sinensis*. Phytopathology 64:1015-1018.
54. Symons, R.H. 1981. Avocado sunblotch viroid: primary sequence and proposed secondary structure. Nucleic Acid Res. 9:6527-6537.
55. Thomas, W. and N.A. Mohamed. 1979. Avocado sunblotch - A viroid disease. Australasian Plant Pathol. 8:1-3.
56. Van Dorst, H.J.M. and D. Peters. 1974. Some biological observations on pale fruit, a viroid-incited disease of cucumber. Neth. J. Pl. Path. 80:85-96.
57. Wallace, J.M. and R.J. Drake. 1962. A high rate of seed transmission of avocado sunblotch virus from symptomless trees and the origin of such trees. Phytopathology 52:237-241.
58. Walter, B. 1981. Un viroide de la tomate en Afrique de l'Ouest: Identité avec le viroide du "Potato Spindle Tuber"? C.R. Acad. Sc., Paris 292:537-542.
59. Yamamoto, H., Y. Kagami, M. Jurokawa, S. Nishimura, S. Ukawa and S. Kubo. 1973. Studies on hop stunt disease in Japan. Rept. Res. Lab. Kirin Brewery Co., Ltd. 16:49-62.

Sclerotinia contamination of Alberta-produced rapeseed, from 1976-1981

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Variation in the shipment of carlots of Alberta rapeseed contaminated with *Sclerotinia* occurred between and within the years 1976-1981. The number of shipping points delivering contaminated seed varied from 6 in 1978 to 28 in 1980. Shipping points in an area north and west of Edmonton generally delivered contaminated seed in each year studied, but the areas around Red Deer and southeast of Edmonton shipped high numbers of carlots with contaminated seed in 1980 and 1981 only.

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Entre les années 1976 à 1981 on retrouve de la variation dans les expéditions de graine de colza contaminée par *Sclérotinia*. Le nombre de point d'expédition ayant des chargement contaminés passe de 6 en 1978 à 28 en 1980. Les points d'expedition situés dans la région au nord et à l'ouest d'Edmonton ont généralement expédié des chargements contaminés chaque année étudiée tandis que ceux situés dans les régions autour de Red Deer et au sud-est d'Edmonton n'en ont expédié un nombre élevé qu'en 1980 et 1981.

Introduction

High levels of contamination of Alberta rapeseed by sclerotia of *Sclerotinia sclerotiorum* (Lib.) de Bary were reported by the Canadian Grain Commission (CGC) in the mid 1970's (2). The increased levels of contamination were presumably due to increases in the severity of Sclerotinia stem rot of rapeseed. The CGC reports indicated that certain areas in Alberta were producing more contaminated rapeseed than others; these reports were used to study variation in production of contaminated seed between locations within each year, and from year to year, from 1976-1981.

Materials and methods

The CGC reports list the carlot origin, number of sclerotia/500 gm sample and date of sampling for all carlots received at the Vancouver terminal. These data were used to calculate the average number of sclerotia/sample for each shipping point in Alberta, on an annual basis. The crop year ran from August 1 to July 31, thus some carry-over of crop from year to year occurred. Also, as rapeseed is frequently stored on the farm for a year or more, it was impossible to guarantee that shipments were made in the harvest year.

All shipping points that delivered rapeseed with an average of 10 or more sclerotia/500 gm sample were considered to produce seed of a high contamination level. The tolerance limit set by Plant Products Division, Agriculture Canada, for all No. 1 rapeseed (Foundation, Certified and Canada) is 1 sclerotium/50 gm seed, thus carlots containing more than 10 sclerotia/500 gm would not qualify as No. 1 seed.

The shipping points delivering contaminated seed were plotted on separate maps of Alberta for each year (Figure 1, A-F). Only shipping points delivering 5 or more contaminated carlots in one year were included, thus each square on the map represents a minimum of 272 tonnes of contaminated rapeseed (assuming an average carlot weight of 54.4 tonnes) delivered to Vancouver. Information on the contamination levels of rapeseed delivered to other terminals, or used in the crushing industry was not readily available, and therefore was not included. Farmers whose rapeseed is badly contaminated with sclerotia may clean the seed before sending it to the elevator, and this factor also was not taken into account.

Results and discussion

The number of shipping points in Alberta delivering rapeseed to Vancouver, and the total number of carlots shipped, declined in 1977 and 1978, increased in 1979 and 1980 and then declined in 1981 (Table 1). The percentage of points shipping contaminated seed and percentage of contaminated carlots declined in 1977 and 1978, and increased in the next three years. The total provincial production figures are not necessarily a direct reflection of production from specific districts and the increase in percentage of contaminated carlots in 1981, when overall rapeseed production declined, could be caused by at least three circumstances: 1) an increase in rapeseed production in areas with a Sclerotinia problem with a decline in production in other areas less favourable for disease 2) a decline in production in areas less favourable for disease 3) an overall increase in disease levels.

The incidence of contaminated seed production varied with location, both within and between years. The six shipping points delivering contaminated seed in 1978, also delivered contaminated seed in almost all years (Fig. 1). These localities seemed to be favourable for disease development every year. The area north of Edmonton appeared more favourable for disease development than the area south of Edmonton in 1976-1979, but in 1980 and 1981 large amounts of contaminated seed were shipped from the Red Deer area and the area southeast of Edmonton. When the annual production and contamination levels are considered for shipping points that delivered contaminated seed in 1980 and/or 1981, it can

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be seen that both total number of carlots shipped and percentage of carlots delivering contaminated rapeseed increased dramatically in those two years (Table 2). In 1976-1979 the average number of sclerotia/sample for all carlots shipped each year from both areas, was less then 10 sclerotia. Four shipping points in the Red Deer area did deliver contaminated seed in 1976-1977, but seed from the majority of the shipping points was not considered contaminated. The

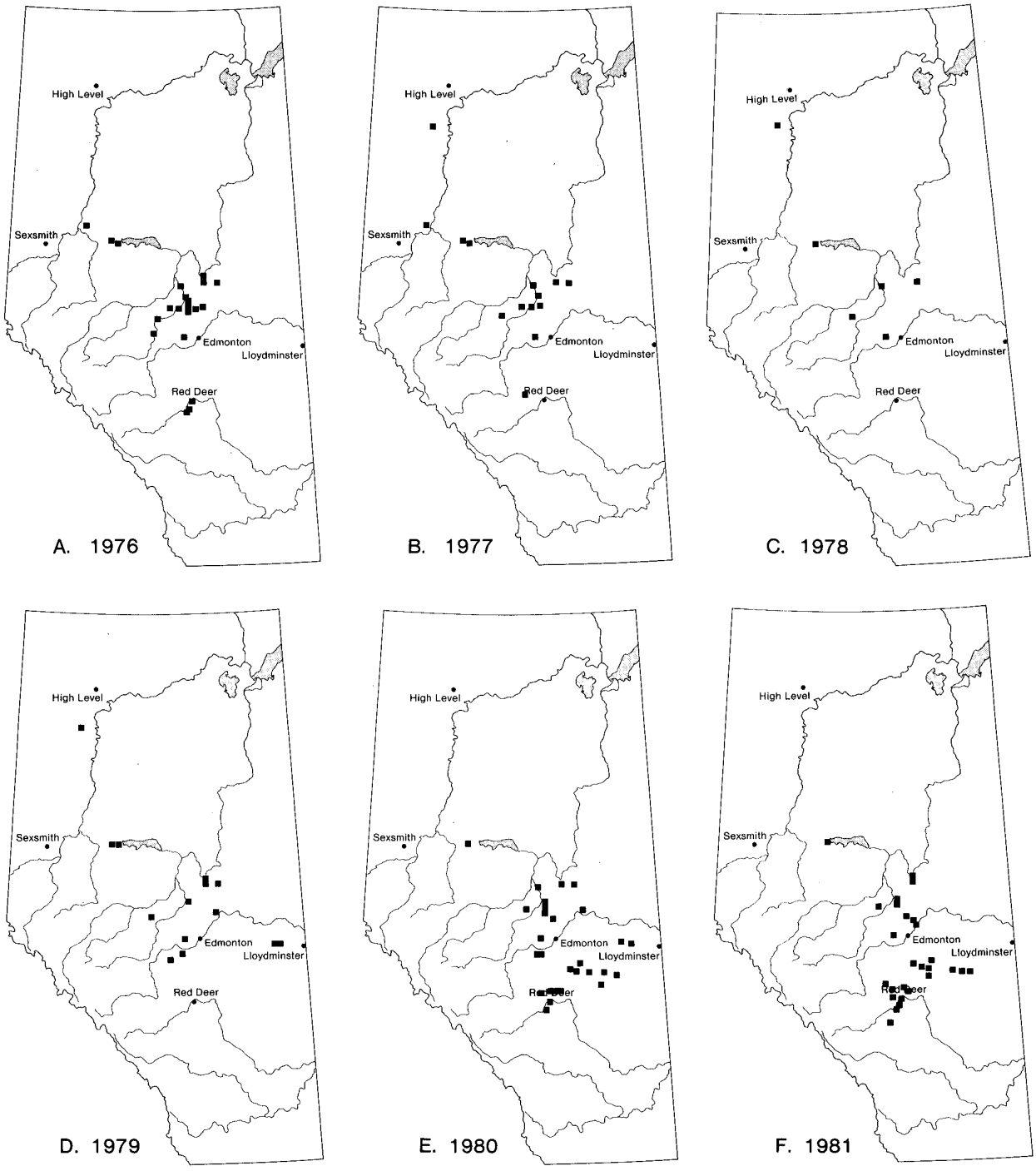

Figure 1. Areas in Alberta shipping contaminated rapeseed; A-F, 1976-1981. Each ■ represents a shipping point that delivered 5 or more carlots, having an average of 10 or more sclerotia/500 gm sample, to Vancouver.

increase in the average contamination level of rapeseed from the Red Deer area in 1981 and the increase in percentage of carlots delivering contaminated seed from both areas in 1981 suggests that contamination levels do not merely reflect changes in production levels.

Table 1. Annual variation in origin and amount of rapeseed production in Alberta, and percentage of shipping points and carlots with contaminated seed.

Year	No. of points shipping rapeseed	% points shipping contaminated rapeseed	Total no. carlots shipped	% contaminated carlots
1976	145	14.5	1131	34.1
1977	126	11.1	780	31.8
1978	127	4.7	703	17.1
1979	134	10.5	889	27.8
1980	153	18.3	1051	33.5
1981	129	20.9	797	55.0

The variation in production of contaminated seed each year is most likely related to weather conditions during the growing season. Weather conditions, which included soil temperature, rainfall, number of days of rain and sunshine were reported to have a tremendous influence on the severity of stem rot of rapeseed in Germany (3). High humidity and presence of petals and/or pollen are both essential for infection of beans by *S. sclerotiorum* (1) so rainfall during the flowering period is likely to have a critical effect on infection of rapeseed. Unfortunately the lack of detailed rainfall data for shipping point localities in Alberta makes it difficult to study the correlation between rainfall patterns and contaminated seed production.

The mapping of annual production of contaminated seed is useful in predicting areas where Sclerotinia stem rot may be a problem. When large amounts of contaminated seed are produced in one area the number of sclerotia in the soil after harvest will be high. If conditions favourable for ascospore production occur during the flowering period of the crop in the following year then severe disease levels are probable. However it appears that severe outbreaks of disease can occur in areas where previous production of contaminated seed has been neglibible; this is demonstrated by the high level of contaminated seed production in the area southeast of Edmonton in 1980. In 1979 the ascospore levels of *S. sclerotiorum* in the south east area were considered sufficient to cause appreciable levels of disease if climatic conditions suitable for infection occurred (4). The low levels of average sclerotia/sample (Table 2) would probably be sufficient to produce the ascospore inoculum necessary to initiate high levels of disease in 1980.

The maps presented here have been reported, in modified form, at Canola Industry and Growers meetings in Alberta, and at the Oilseeds and Special Crops Sub-committee meeting in Saskatoon, 1982, to indicate where Sclerotinia stem rot has been a problem in past years, and where it may occur in the future.

Table 2. Annual total carlot delivery, percentage of carlots with contaminated seed and average number of sclerotia/carlot sample for areas around Red Deer and southeast of Edmonton.

	Red Deer area[a]			Southeast of Edmonton area[b]		
Year	Total carlot delivery	% carlots with contaminated seed	Av. no. sclerotia/carlot sample	Total carlot delivery	% carlots with contaminated seed	Av. no. sclerotia/carlot sample
1976	66	69.7	5.8	27	0	5.7
1977	62	0	5.8	7	0	2.9
1978	51	0	4.1	35	0	3.4
1979	14	0	4.5	44	0	4.5
1980	115	88.7	11.4	119	85.7	13.3
1981	267	99.2	26.7	94	90.4	13.2

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Literature cited

1. Abawi, G.S. and R.G. Grogan. 1979. Epidemiology of diseases caused by *Sclerotinia* species. Phytopathology 69: 889-904.
2. Canadian Grain Commission. 1976-1981. Weekly reports on Sclerotinia pieces in oilseed clean-out at Vancouver terminal.
3. Krüger, W. 1975. Über die wirkung der witterung auf den befall des rapses durch *Sclerotinia sclerotiorum* (Lib.) de Bary. [Influence of the weather on the attack of rape by *Sclerotinia sclerotiorum* (Lib.) de Bary] Nachrichtenblatt Deut. Pflanzenschutzd. (Braunschweig) 27: 1-6.
4. Williams, Jill R. and D. Stelfox. 1980. Occurrence of ascospores of *Sclerotinia sclerotiorum* in areas of central Alberta. Can. Plant Dis. Surv. 60: 51-53.

Suspected boron deficiency in birdsfoot trefoil in field plots

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Abortion and dropping of flowers, shortened internodes, and discolored and deformed young leaves of birdsfoot trefoil (*Lotus corniculatus*) in field grown plants resembled boron deficiency symptoms in alfalfa. Foliar application of soluble boron corrected the symptoms; seed yield was good. Boron deficiency, previously unreported on field-grown birdsfoot trefoil, apparently was induced by intermittent drought conditions in May, June, and July.

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Des symptômes observés sur des plants de lotier corniculé (*Lotus corniculatus*) en champ, avortement et pertes des fleurs, racourcissement des entre-noeuds, jeunes feuilles décolorées et déformées, ne sont pas sans rappeler ceux de la carence en bore chez la luzerne. Une application foliaire de bore soluble corrige ces symptômes et permet une bonne production de semence. Il semble que cette carence en bore, signalée pour la première fois sur le lotier corniculé en champ, ait été induite par des conditions intermittentes de sécheresse en mai, juin et juillet.

Birdsfoot trefoil (*Lotus corniculata* L.) has been a subject of research at the Macdonald Campus of McGill University for many years. Current work, in addition to the cytogenetic and taxonomic studies of Dr. W.F. Grant, involves production of breeder seed of the cultivars Leo and Mirabel, and control of perennial grass weeds in seed production fields.

Seed increase plots with spaced plants of Mirabel seeded in 1979 and plots solid-seeded in 1981 were located on the E.A. Lods Agronomy Research Station on the campus. Plots of Leo solid-seeded in 1979, and spaced plants started in the greenhouse and transplanted in spring of 1982, were located in isolated fields in the Morgan Arboretum, also on the campus.

Flowering usually starts in established trefoil plots about mid June. Mirabel plots flowered normally, although abortion of bloom and discoloration of leaves was observed on scattered plants in the solid-seeded plots.

Flowering started about mid July in the spaced planting of Leo set out in the spring. The plants developed well, and a very satisfactory yield of about 200 kg seed per ha was harvested.

No bloom had appeared in the solid-seeded Leo by the end of June. On examination it was observed that flower buds had formed, but failed to develop. Anthers and stigmas turned brown, and the buds abscissed. There was a reddish discoloration along the veins of some leaves, and yellowing of entire leaves. Internode growth was restricted, producing a rosette effect. New leaves were deformed. Flower symptoms similar to those present in early July were observed again in mid August in the solid-seeded Leo.

These symptoms were similar to those described for boron deficiency in alfalfa in Quebec (Ouellette and Lachance 1954).

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As it was essential to harvest as much seed as possible from the plot, the plants were sprayed with a soluble boron preparation (SOLUBOR) at the rate of 10 kg borax per ha on July 12. No untreated area was left as a control. Flowering began in the sprayed plot about July 19, and continued until late August. Approximately 100 kg/ha of seed was harvested late in September.

Boron deficiency is likely to occur in alfalfa growing on light soils containing less than 0.3 ppm of water-soluble boron, and on heavy soils with less than 0.5 ppm (Ouellette and Lachance 1954). Soil analyses were made early in July. The pH was 5.7 to 5.6. Boron content was 0.77 ppm from 0 to 15 cm deep, and 0.47 ppm in samples from 15 to 30 cm deep. The soil in the affected plots is classified as a Dalhousie clay.

Flower abortion symptoms were apparent in August in solid-seeded plots of Leo heavily infested with couch grass (*Agropyron repens* (L.) Beauv.) which were being used in a weed-control study. An experiment was made to determine the effects of top dressing with soluble boron, using a split plot design, with applications of 0, 10, and 20 kg/ha of borax on August 31. As no new flowers formed in any plots, including the apparently healthy spaced planting, and no symptoms developed on new growth in any plots after the end of August, no results were obtained.

Availability of boron to plants is affected by water supply as well as by boron content of the soil; repeated cycles of drought may result in apparent boron deficiency even in soils with adequate boron levels (Dionne and Pesant 1978). Five year average rainfall for May, June, July, and August, and the values for 1982 at the Agronomy station were as follows:

	Average monthly rainfall (cms) 1977-1981	Monthly rainfall 1982
May	51	24
June	76	115
July	84	79
August	105	122

The monthly figures indicate drought in May and average or above-average rainfall in June, July, and August of 1982. It was irregular, however. The disease forecasting service of the Quebec Ministry of Agriculture emphasized in its regular releases that the May drought continued for the first half of June. June 1 to 14, precipitation totalled 16 mm at the Agronomy station. From June 26 to July 27, the total was 32.5 mm. Rains were more frequent in late summer, with 59 mm in the last days of July; from 1 to 9 mm on 9 days, and 17, 22, and 67 mm in showers on 3 days in August.

Plots of timothy (*Phleum pratense* L.) near the trefoil showed severe drought symptoms in early June and again in mid July. Although drought symptoms were not conspicious on the trefoil, the plants were certainly subjected to water stress in the solid-seeded plots.

No analyses were made of boron content of tissues of affected and apparently healthy plants. Even without such confirmatory data, it appears most probable that the symptoms observed were caused by a temporary deficiency of available boron, induced by the local drought conditions during May, part of June, and part of July. As far as we can determine, boron deficiency in field grown birdsfoot trefoil has not been reported previously. We plan to study the effect of boron applications on the trefoil plots in the summer of 1983.

Literature cited

1. Dionne, J.L. et A.R. Pesant. 1978. Effets des doses de bore, des régimes hydriques et du pH des sols sur les rendements de la luzerne et du lotier et sur l'assimilabilité du bore. Can. J. Soil Sci. 58:369-379.
2. Ouellette, G.J. and R.O. Lachance. 1954. Soil and plant analysis as means of diagnosing boron deficiency in alfalfa in Quebec. Can. J. Agr. Sci. 34:494-503.

Fusarium nivale (*Gerlachia nivalis*) from cereals and grasses: Is it the same fungus? [1]

J. Drew Smith

Perithecia of the teleomorph of *Fusarium nivale* (*Gerlachia nivalis*) developed in culture on sterilized cereal straws from isolates obtained from cereals. No perithecia developed in isolates from perennial grasses. Nearly all isolates from grasses and cereals were pathogenic on rye seedlings. Is the fungus from cereals and grasses a different species or variety, or is it the same, but the grass isolates have lost the ability to produce perithecia?

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Des isolats de *Fusarium nivale* (*Gerlachia nivalis*) obtenus à partir de graminées vivaces ne produisent pas de périthèces lorsqu'ils sont inoculés sur de la paille de céréale stérilisée contrairement aux isolats obtenus à partir de céréales. Toutefois, presque tous les isolats quelle que soit leur origine sont pathogènes envers les plantules de seigle. Le champignon isolé chez les céréales appartient-il à une espèce ou une variété différente, ou est-il semblable à celui isolé chez les graminées vivaces à la différence près que ce dernier ne serait plus capable de produire des périthèces?

Dobrozrakova (6) and Wollenweber and Reinking (14) reported that the perithecia of the teleomorph of *Fusarium nivale* (Fr.) Ces., *Calonectria graminicola* Wollenw., now named *Monographella nivalis* (Schaffnit) E. Muller (9) with an anamorph *Gerlachia nivalis* (Ces. ex Sacc.) W. Gams and E. Muller (7), were produced freely on cereals in Europe. Bennett (2) failed to find them on cereals in Northern England, but later obtained perithecia and ascospores on sterilized wheat grains in culture (12). The teleomorph is unknown in Canada except in culture (8), but Cook and Bruehl (4) found it on leaves and leaf sheaths of mature wheat plants in Washington and Oregon. Perithecia were found on a grass in the Isle of Rhum, Scotland by Dennis (5), but that is the only record of the teleomorph on grasses that I have found. However, it is common on oats in Scotland and Noble and Montgomerie (10) confirmed the connection between the two states by single ascospore cultures. They also found perithecia in isolates growing in culture on potato dextrose agar.

No perithecia were found during examination and culturing in many cases of fusarium patch disease from turfgrasses in the British Isles and Western Europe from 1951 to 1964 at the Sports Turf Research Institute in England (12). No special techniques were used to induce perithecial production, but a large number of isolates were grown on wheatmeal, oat, potato dextrose, glucose boric, glucose yeast extract agars and on sterile grass clippings and on none of these were perithecial structures seen.

None of the isolates from grasses in Norway examined by Årsvoll (1) produced perithecia in culture (Dr. K. Årsvoll, personal communication).

Between 1972 and 1974, 53 isolations of *Fusarium nivale* were made, mainly from turfgrasses in Saskatchewan, but also from British Columbia and Washington. Isolates were obtained from a *Bromus* sp. and from an unidentified turfgrass in New Zealand (from Dr. G.C. Latch, N.Z.D.S.I.R., Palmerston North). Isolations were also made from fall rye and winter wheat plants at several places in Saskatchewan after snow melt in 1974. Two isolates from seeds of winter wheat from Norway were obtained (from Mr. L.R. Hansen, Plant Protection Institute, As-NLH). Several of the cereal isolates produced mature perithecia in culture on potato dextrose agar on a temperature gradient plate under 12 h near ultraviolet light at 13-17°C. No perithecia formed on any of the grass isolates grown under the same conditions which are those routinely used in this laboratory for the inducement of conidial production. This apparent difference between the isolates from cereals and those from grasses indicated the need for more critical examination of the ability of the isolates to produce perithecia.

Materials and methods

Fifty isolates of the fungus from grasses and 24 from cereals were selected; all produced conidia of the description given by Booth (3) for *F. nivale*. Ten pieces of wheat straw were sterilized for 1 h at 1 atm by autoclaving in 250 ml erlenmyer flasks containing 40 ml distilled water. The straws were inoculated with pieces of *F. nivale* culture cut from the edges of actively growing colonies of the appropriate isolate. The flasks were plugged, capped with foil and incubated at 18°C. The occurrence and abundance of perithecia was recorded after 6 to 8 weeks of incubation (3).

Spore suspensions prepared from 15 grass and 7 cereal isolates were used to inoculate cold-acclimated fall rye seedlings which were then incubated at 0 to 1°C in high humidity chambers for two months (13).

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Results and discussion

None of the 50 grass isolates produced perithecia or perithecial initials. Fourteen of the cereal isolates formed these structures, some more frequently than others. Mature asci with ascospores were found in some of these; most did not mature.

Six of the rye isolates and 14 of the grass isolates were pathogenic on the inoculated rye. Pathogenicity towards grasses was not examined since cereal isolates are known to be pathogenic on grasses (11).

There was great variation in cultural characters of isolates of *F. nivale* from both grasses and cereals as has been previously noted (11) and considerable difference in conidial size. Most turfgrass isolates from Saskatchewan have 0- or 1-septate conidia (Smith, unpublished). We have not examined a sufficient number of cereal isolates to determine septation frequency. Wollenweber and Reinking (14) recognized *F. nivale* and a larger spored var. *majus*. Some of the cereal isolates may have been classified as the latter, and this variety may produce perithecia more freely than the type. An alternative explanation may be that in perennial grasses *F. nivale* has lost the ability to produce perithecia or is unable to do so under the cultural conditions provided. Whether grass isolates may be induced to do so or whether they would regain the ability to form perithecia on cereals is of considerable taxonomic and epidemiological interest.

Literature cited

1. Årsvoll, K. 1975. Fungi causing winter damage on cultivated grasses in Norway. Meld. Norg. LandbrHogsk. 54(9): 49 pp.
2. Bennett, F.T. 1929. *Fusarium* species on British cereals; *Fusarium nivale* (Fr.) Ces. (= *Calonectria graminicola* (B. & Br.) Wollenw.). Ann. Appl. Biol. 20:272-290.
3. Booth, C. 1971. The genus *Fusarium*. Commonw. Mycol. Inst. Kew, 237 pp.
4. Cook, R.J. and G.W. Bruehl. 1966. *Calonectria nivalis* perfect stage of *Fusarium nivale*, occurs in the field in North America. Phytopathology 56:1100-1101.
5. Dennis, R.W.G. 1964. The fungi of the Isle of Rhum. Kew Bull. 19(1):77-131.
6. Dobrozrakova, T.L. 1929. *Fusarium nivale* (*Calonectria graminicola*) on autumn sown cereals near Leningrad. (Abs. in Rev. appl. Mycol. 8:171).
7. Gams, W. and E. Muller. 1980. Conidiogenesis of *Fusarium nivale* and *Rhynchosporium oryzae* and its taxonomic implications. Neth. J. Plant Path. 86:45-53.
8. Gordon, W.L. 1952. The occurrence of *Fusarium* species in Canada. II Prevalence and taxonomy of *Fusarium* species in cereal seed. Can. J. Bot. 30:209-251.
9. Müller, E. 1977. Die systematische Stellung des Schneeschimmels. Rev. Mycol. 41:129-134.
10. Noble, M. and I.G. Montgomerie. 1956. *Griphosphaeria nivalis* (Schaffnit) Muller and von Arx and *Leptosphaeria avenaria* on oats. Trans Brit. Mycol. Soc. 39:449-459.
11. Smith, J.D. 1957. The control of certain diseases of sports turf grasses in the British Isles. Msc. thesis, University of Durham, Vol. 1.
12. Smith, J.D. 1965. Fungal diseases of turf grasses (2nd. ed., revised N. Jackson and J.D. Smith) The Sports Turf Res. Inst. England. 97 pp.
13. Smith, J.D. 1981. Snow molds on winter cereals: guide for diagnosis, culture and pathogenicity. Can. J. Plant Path. 3, 15-25.
14. Wollenweber, H.W. and O.A. Reinking. 1935. Die Fusarien. Paul Parey, Berlin 355 pp.

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Distribution of *Sclerotinia sclerotiorum* in western Canada as indicated by sclerotial levels in rapeseed unloaded in Vancouver, 1973-1981[1]

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The frequency and distribution of stem rot (*Sclerotinia sclerotiorum* (Lib.) de Bary) of rapeseed in western Canada is indicated by the levels of sclerotial infestation in railcar unloads in Vancouver from 1973 to 1981. Sclerotial infested rapeseed has been recorded from all of the major rapeseed growing areas of the four western provinces suggesting that stem rot is a very prevalent disease. Relatively high sclerotial levels were found in rapeseed originating in Alberta crop districts 4, 5 and 6, and low to moderate levels were recorded for Saskatchewan, Manitoba, southern Alberta, and the Peace River area.

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Le niveau d'infestation en sclérote de la graine de colza, mesuré lors de déchargement des wagons de train à Vancouver de 1973 à 1981, permet d'estimer la fréquence et la distribution de la pourriture sclérotique (*Sclérotinia sclérotiorum* (Lib.) de Bary) du colza dans l'ouest canadien. De la graine de colza infestée de sclérote a été signalée en provenance de toutes les régions des quatres provinces de l'ouest où cette culture est importante, ce qui suggère que cette maladie est très répandue. Des niveaux relativement élevés de sclérotes ont été mesurés dans des chargements de graine de colza provenant des régions agricoles 4, 5 et 6 de l'Alberta et des niveaux bas à modérés dans ceux en provenance de la Saskatchewan, du Manitoba, du sud de l'Alberta et de la région de la Peace River.

Introduction

Stem rot is a major disease of rapeseed in western Canada (Duczek & Morall 1971, Morrall et al. 1976, Platford & Bernier 1975, Morrall & Dueck 1982). Ascospore infection of stems usually occurs during petal fall and is dramatically affected by available moisture, temperature and the presence of dead flower parts (McLean 1958, Kruger 1975). Sclerotia are formed inside the hollow stems of infected plants which become brittle and shred easily at maturity. During threshing sclerotia are released from the stem and become mixed with the seed.

A large proportion of the rapeseed grown in western Canada is transported by rail to Vancouver, British Columbia for export. During the unloading of the railcars a 500 g sample is routinely analyzed for dockage; included in the dockage are the sclerotia of *Sclerotinia sclerotiorum* (Lib.) de Bary, which are common contaminants of harvested rapeseed (Dueck et al. 1981). The numbers of sclerotia per 500 g sample have been provided by the Canadian Grain Commission for the period 1973 to 1981. This study is based on records of 9707 samples for the 9-year period. The purpose of this paper is to infer the frequency and distribution of stem rot of rapeseed (*Brassica napus* L. and *B. campertris* L.) in western Canada based on the level of sclerotial infestion and the origin of the sample. Although the sample records are only for rapeseed transported through Vancouver, the data are sufficient to permit identification of areas where stem rot can be expected to be a persistent problem.

Materials and methods

The data consisted of the number of sclerotia per 500 g seed, the arrival date in Vancouver and the grain elevator of origin. Rapeseed carloads arriving in Vancouver from September 1 to August 31 were assumed to have been grown during the previous growing season. From these data the average number of sclerotia per sample for each elevator were calculated on a yearly basis using a computer program. The average level for each elevator was then plotted on a map (Figure 1). The distribution of sclerotial levels was divided into three equal classes designated low, moderate, and high, corresponding to levels of 1.0 to 2.6, 2.7 to 6.2, and more than 6.2 sclerotia per 500 g seed, respectively.

Results and discussion

Disease distribution. The recorded levels of sclerotial infestation are summarized in Figure 1. Crop districts 4, 5 and 6 of Alberta appear to have the highest frequency of stem rot for the period surveyed; elevator reports of sclerotial levels of more than 20 sclerotia per 500 g seed from these areas are common. Saskatchewan and the southern portions of the rapeseed growing areas of Alberta apparently have moderate to low frequencies of stem rot. The samples from Manitoba appear to be equally distributed among the three classes of sclerotial infestation. Carloads of rapeseed infested with sclerotia were recorded from all of the major rapeseed growing areas of western Canada suggesting that stem rot is a very prevalent disease.

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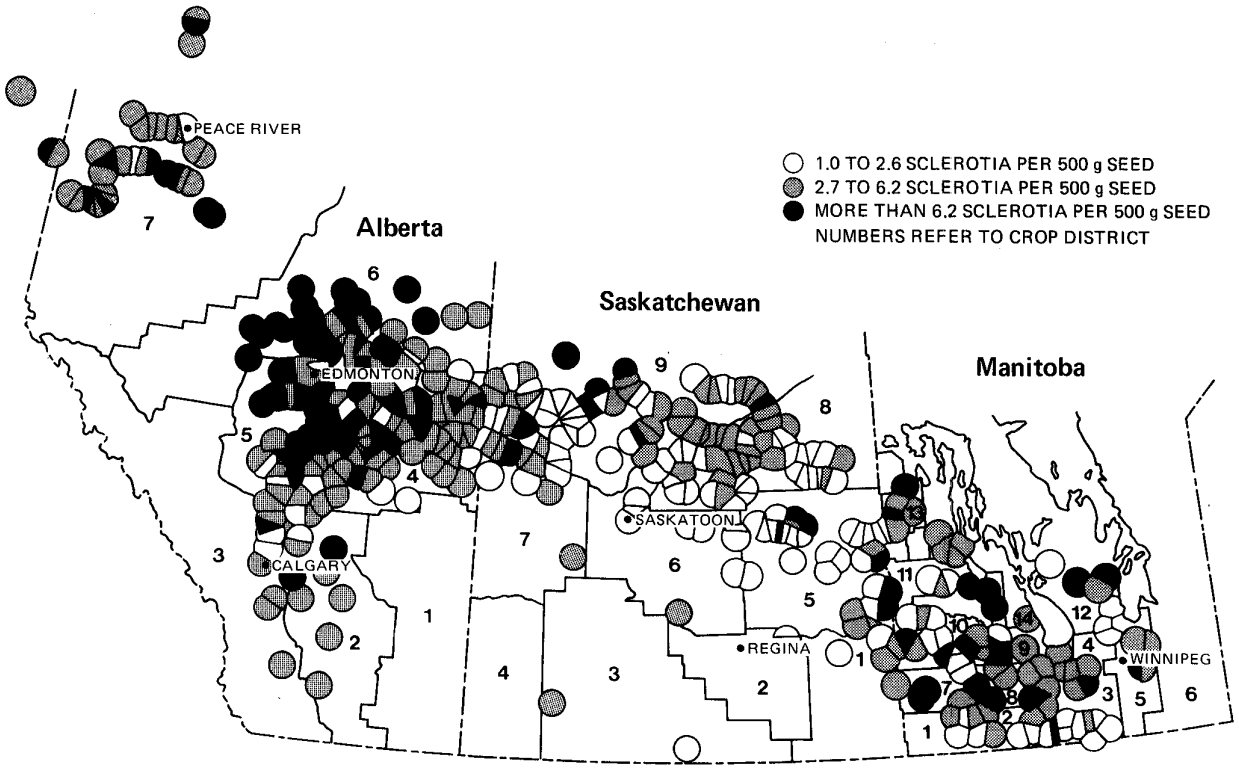

Figure 1. Sclerotial levels in rapeseed unloaded in Vancouver, 1973-1974.

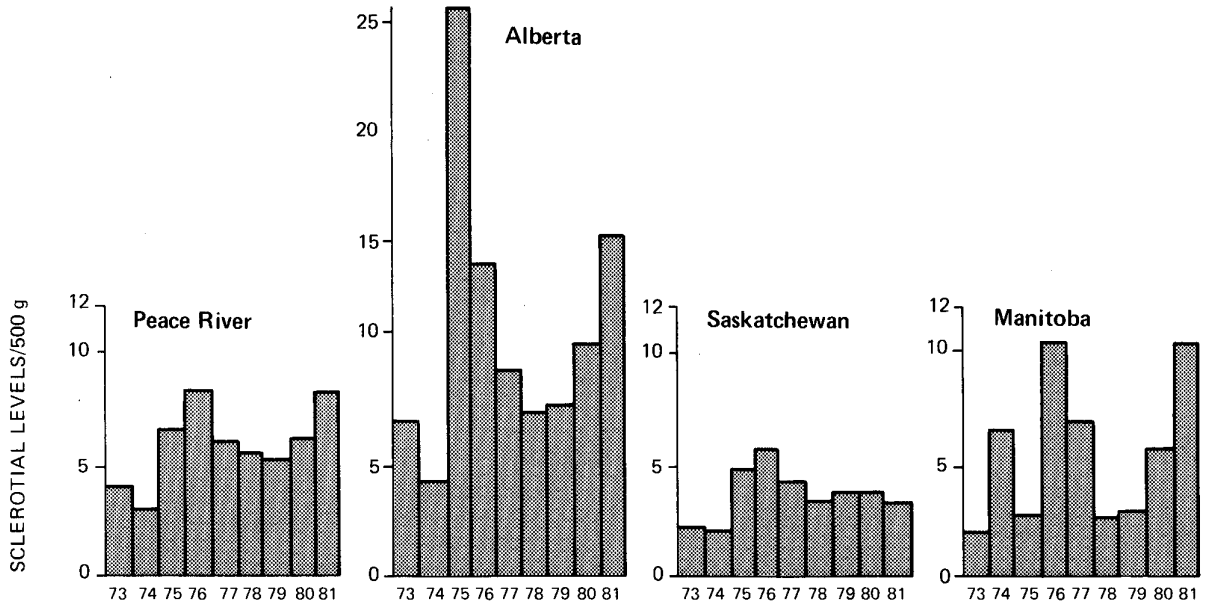

Figure 2. Variation in content of sclerotia of *Sclerotinia sclerotiorum* in samples of rapeseed from railcar unloads at Vancouver by year (1973-81) and area of origin.

Disease variability among years. The greatest contributing factor to the variability among years is probably the amount of available moisture during flowering, the time of ascospore infection. Moisture influences apothecial formation, ascospore dispersal and germination, and the suitability of the infection court (Kruger 1976, McLean 1958, Morrall and Dueck 1982). The absence of increasing trends in the sclerotial levels in seed samples suggest no net increase in the levels of stem rot

infestation in any of the four areas studied over the 9-year period (Figure 2). The frequency of stem rot, as implied by the sclerotial levels in the seed, fluctuates considerably among years in central Alberta and the Peace River area. Conversely, in Saskatchewan the frequency of stem rot appears to have remained relatively constant since 1975. The lower sclerotial levels in samples from Saskatchewan suggest stem rot has a lower frequency than in the other provinces. The lower disease frequency in Saskatchewan could be due to the drier conditions during the growing season.

Attempts to correlate the variability in the apparent disease frequency among years, with the amount of precipitation during the growing seasons, were not successful. The absence of good correlations suggest other parameters also have a major influence on sclerotial infestation levels in seed. In addition, the amounts of precipitation can vary dramatically over relatively small distances and hence the local environmental conditions which influence sclerotial production can also vary considerably (Morrall and Dueck 1982). The data did not identify field location with enough precision for this type of correlation.

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Literature cited

1. Duczek, L.J. and R.A.A. Morrall. 1971. Sclerotinia in Saskatchewan in 1970. Can. Plant Dis. Surv. 51: 116-121.
2. Dueck, J., R.A.A. Morrall, A.J. Klassen and J. Vose. 1981. Heat inactivation of sclerotia of *Sclerotinia sclerotiorum*. Can. J. Plant Pathol. 3: 73-75.
3. Kruger, W. 1975. The influence of some environmental factors on the development of apothecia and ascospores of *Sclerotinia sclerotiorum* (Lib.) de Bary. Die beeinfluessung der apothezien- und ascosporen-entwicklung des rapskrebserregers *Sclerotinia sclerotiorum* (Lib.) de Bary durch umweltfaktoren. Z. Pflanzenkrankh. und Pflanzenschutz 82: 101-108.
4. McLean, D.M. 1958. Role of dead flower parts in infection of certain crucifers by *Sclerotinia sclerotiorum* (Lib.) de Bary. Plant Dis. Rep. 42: 663-66.
5. Morrall, R.A.A., J. Dueck, D.L. McKenzie and D.C. McGee. 1976. Some aspects of *Sclerotinia sclerotiorum* in Saskatchewan, 1970-75. Can. Plant Dis. Surv. 56: 56-62.
6. Morrall, R.A.A. and J. Dueck. 1982. The epidemiology of Sclerotinia stem rot of rapeseed in western Canada. Can. J. Plant Pathol. 4: 161-168.
7. Platford, R.G. and C.C. Bernier. 1975. Diseases of rapeseed in Manitoba, 1973-74. Can. Plant Dis. Surv. 55: 75-76.

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