Canadian Plant Disease Survey

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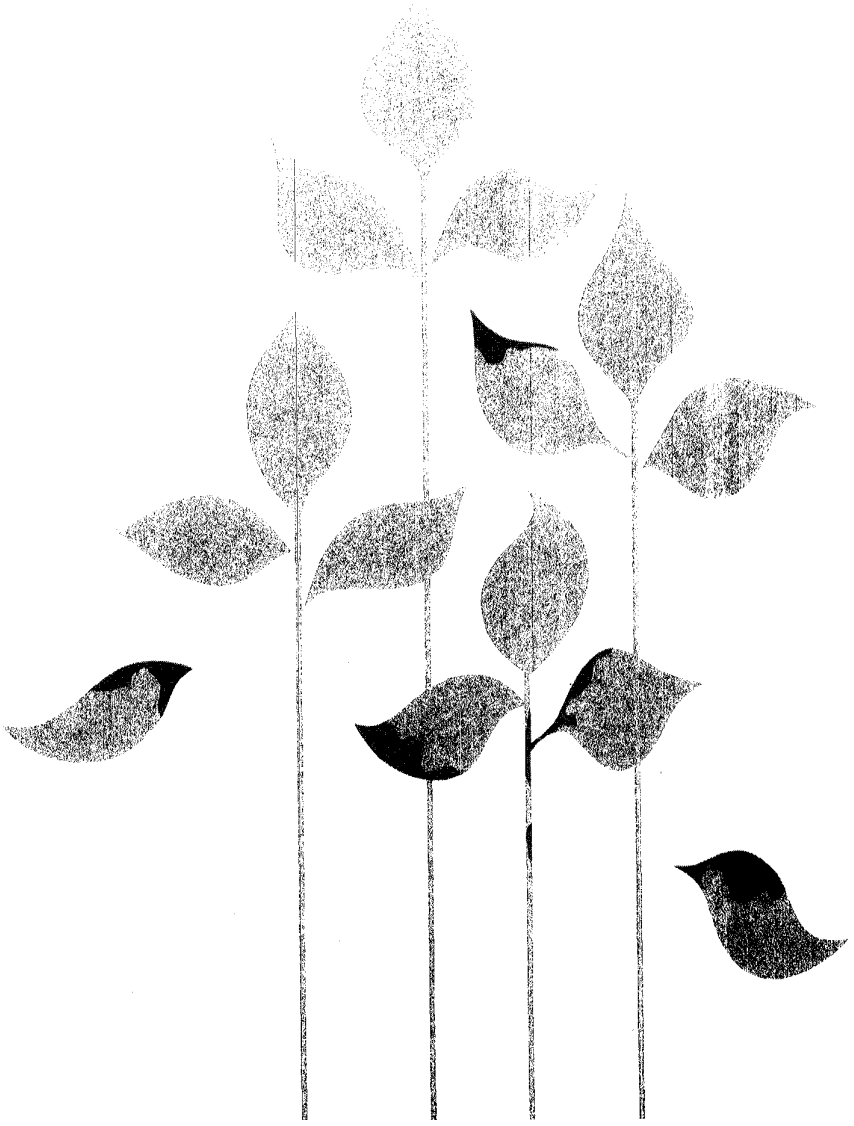

Agriculture

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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 will also be accepted. Review papers and compilations of practical value to plant pathologists will be included from time to time.

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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. De temps à autre, l'inventaire inclut des revues et des synthèses de rapports d'intérêt immediat pour les phytopathologistes.

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Pythium species in alfalfa fields in central Alberta

D. Stelfox[1] and J. R. Williams[2]

Five species of *Pythium* were isolated from seedling alfalfa roots collected during a survey of 4 alfalfa fields in central Alberta in 1976. *P. sylvaticum* occurred on 3 fields, *P. paroecandrum* on 2 fields, and *P. ultimum*, *P. hypogynum*, and *P. torulosum* were each found on 1 field.

Can. Plant Dis. Surv. 60:4, 35-36, 1980.

Cinq espèces de *Pythium* ont été isolées des racines de plantules de luzerne recueillies en 1979 au cours d'une enquête portant sur quatre luzernières du centre de l'Alberta. *P. sylvaticum* a été identifié dans trois champs, *P. paroecandrum*, dans deux et enfin *P. ultimum*, *P. hypogynum* et *P. torulosum* dans un champ chacun.

Introduction

Numerous alfalfa growers in central Alberta report a rapid decline in stand density of recent crops in comparison with stands established following initial sod breaking. A biological toxic agent has been postulated as one of the factors operating in "alfalfa sick soil"(6). Information has been lacking concerning the role of pythiaceous fungi in alfalfa seedling establishment and stand decline in Alberta. *Pythium* spp. have been shown to directly or indirectly affect germinating seed or seedlings of alfalfa and to retard development of plants through root girdling or root tip necrosis, especially on finer textured acid soils (1, 2).

The present study involved a survey for the presence of pythiaceous fungi on alfalfa seedlings in four central Alberta alfalfa fields. Each of the four fields was sown to a companion crop of barley, and had a history of recent poor alfalfa growth. Examination of, and isolations from, the seedlings sampled were carried out at the Plant Industry Laboratory, Edmonton. Isolates obtained in the study were not tested for ability to infect germinating alfalfa seeds and seedlings.

Materials and methods

Alfalfa seeds enclosed in nylon gauze strips to facilitate preemergence sampling were sown in the four fields listed below which were seeded to alfalfa during late May, 1976. Seedlings were randomly collected from the four fields on the following dates:

Field 1 (Bluffton)	27 May, 8 and 30 June, 19 July.
Field 2 (Millet)	8 and 30 June, 12 and 27 July.
Field 3 (Namao)	16 June, 7 and 20 July.
Field 4 (Spruce Grove)	16 June, 8 and 23 July.

Field 1 was located in the grey-wooded soil zone, and fields 2, 3 and 4 were in the black soil zone.

Specimens were placed in polyethylene bags, tied securely, and transported in a cooler to the laboratory. They were then washed under flowing tap water, surface-sterilized in 70% ethyl alcohol for 15 seconds, rinsed twice in sterile distilled water, dried between sterile filter paper, and plated on a selective antibiotic medium (PP) (5) containing *pimaricin* and Pentachloronitrobenzene (*PCNB*). The plates were incubated in darkness at 20°C for 14 days during which they were examined frequently for fungal growth. Aseptate isolates were subcultured on PP agar and retained for future identification. Any seedlings not plated immediately upon reaching the laboratory were stored overnight at 5°C. The upper green portions of larger seedlings were removed prior to surface-sterilizing. Notes were made of the number of seedlings with healthy-appearing roots, as well as those bearing brown lesions, necrotic or "pinched" areas.

Table 1. Occurrence of *Pythium* isolates in four central Alberta alfalfa fields.

Location	Number of seedlings plated	Number of *Pythium* isolates
Field 1 (Bluffton)	338	14
Field 2 (Millet)	461	28
Field 3 (Namao)	402	9
Field 4 (Spruce Grove)	343	18

Results

A total of 69 *Pythium* isolates were obtained from 1594 seedlings plated. Isolates were not obtained consistently from either sick or healthy-looking seedlings and they originated from both hypocotyl and root regions. Several seedlings yielded *Pythium* spp. although their roots showed no rotting or necrosis. No *Phytophthora* sp. was isolated from any of the seedling samples collected.

The greatest number of isolates were obtained from two of the fields located in the black soil zone, and a lesser number were obtained from the other two fields (Table 1). The percentage of isolates recovered from seedlings was highest

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during a two week period in early July on two of the black soil fields (Fig. 1), the lowest percentage recovery occurred in seedlings lifted from the remaining two fields. The percentage of isolates recovered from seedlings was low for all four fields by late July when, presumably, all field soils had warmed and/or dried considerably.

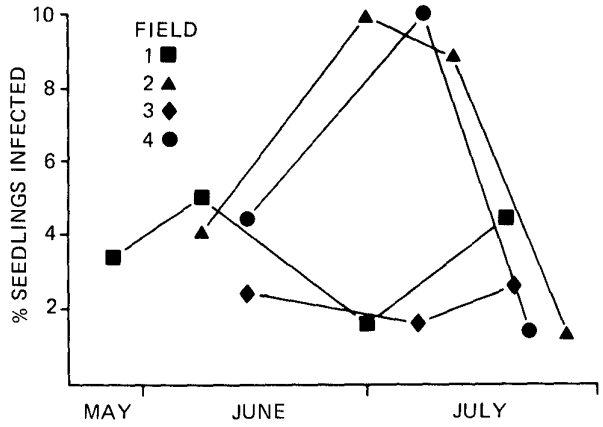

Figure 1. Occurence of *Pythium* isolates from alfalfa seedlings at four locations - 1976.

Several of the 69 *Pythium* isolates obtained were selected on the basis of reproductive structures and on growth patterns at different incubating temperatures. These were identified by Dr. D. J. S. Barr of the Biosystematics Research Unit, Ottawa, Ontario. Four of these isolates were *P. sylvaticum* Campbell and Hendrix and they occurred on three of the test fields (Table 2). Four of the isolates in Field 2 were *P. hypogynum* Middleton. *P. paroecandrum* Drechsler came from Fields 3 and 4. *P. torulosum* Trow originated in Field 2. Two non-fruiting isolates came from Fields 1 and 4. Since not all isolates were submitted for identification it is probable that *Pythium* spp. other than those listed were isolated.

Table 2. *Pythium* isolates obtained from test fields.

Isolate	Field 1	Field 2	Field 3	Field 4
Pythium sylvaticum	X	X		X
P. hypogynum		X		
P. paroecandrum			X	X
P. torulosum		X		
P. ultimum			X	
P. spp.	X			X

Discussion

Pythium spp. were frequently isolated from roots of alfalfa seedlings with and without damping-off symptoms or root necrosis. Isolation from seedlings showing no evidence of root rot or necrosis may have been due to infections having occurred recently and disease development not being sufficiently advanced for symptoms to appear. Spores of the fungi may have survived on healthy roots during surface sterilizing. The higher percentage of seedlings in Fields 2 and 4 yielding isolates may reflect a higher inoculum level in those fields than in Fields 1 and 3. The total rainfall on the four fields surveyed during the sampling period was not appreciably different, thus soil moisture alone does not account for differing inoculum levels.

The number of species isolated and their range suggests a wide distribution of these potential alfalfa pathogens (4, 3) in central Alberta soils. *P. sylvaticum (debaryanum)* has been shown to cause rapid necrosis of germinating seed and seedlings of alfalfa in Iowa and to retard seedling development through root infection (1). The ultimate effects of the pathogen were partial or complete loss of seedling stands and dwarfed plants with root systems inadequate to carry the plants through periods of stress. Dwarfing and yellowing of many surviving mature plants are characteristic symptoms of the crop on "sick" central Alberta soils. In Ohio *P. ultimum* resulted in severe damping-off and poor emergence of alfalfa because of its high virulence, prevalence in seedlings and abundance in the soil (4). The same study indicated that *P. sylvaticum* and *P. paroecandrum* were moderately virulent to alfalfa in the cotyledon stage.

Prolonged susceptibility to fungal infection or a recurrence of susceptibility in older seedlings may be caused by stress factors such as excessive clipping and the presence of companion crops (4) which can result in low light intensity and competition for soil moisture and nutrients. The combined effect of a companion crop, harsh winter conditions, and fungal infection may weaken seedling stands and be a significant contributing factor in the current "sick" alfalfa problem on central Alberta farms.

Literature cited

1. Buchholtz, W. F. 1942. Influence of cultural factors on alfalfa seedling infection by *Pythium debaryanum* Hesse. Res. Bull. 296. Iowa Agric. Expt. Sta.
2. Chi, C. C., and E. W. Hanson, 1962. Interrelated effects of environment and age of alfalfa and red clover seedlings on susceptibility to *Pythium debaryanum*. Phytopathology 52:985-89.
3. Hendrix, F. F., Jr., and W. A. Campbell. 1973. Pythiums as plant pathogens. Ann. Rev. Phytopath 11:77-98.
4. Schmitthenner, A. F. 1964. Prevalence and virulence of *Phythophthora, Aphanomyces, Pythium, Rhizoctonia,* and *Fusarium* isolated from diseased alfalfa seedlings. Phytopathology 54:1012-1018.
5. Tsao, P., and G. Ocana. 1969. Selective isolation of species of *Phytophthora* from natural soils on an improved antibiotic medium. Nature 223:636-638.
6. Webster, G. R., S. U. Kahn, and A. W. Moore, 1967. Poor growth of alfalfa (*Medicago sativa*) on some Alberta soils. Agronomy Journal 59:37-41.

Current status of little cherry disease in British Columbia[1]

J.T. Slykhuis[2], J. Yorston[3], J. Raine[4], R.D. McMullen[2] and Thomas S.C. Li[2]

Little cherry disease was present in most sweet cherry trees (*Prunus avium*) tested from all districts in the Kootenay region where cherry trees were found from 1976 to 1979. In the Creston district where commercial cherry growing continues, most trees of 10 years or older were infected but several trees 25 to 30 years old in isolated sites were disease-free. The virus was graft-transmitted to Sam trees from two oriental flowering cherry trees (*Prunus serrulata*) found in Creston, and from 15 of 30 native bitter cherry trees (*P. emarginata*) growing near abandoned orchards in several Kootenay districts. The apple mealy bug (*Phenacoccus aceris*) a vector suspect, was found in some locations where natural spread occurred recently.

Little cherry disease has been found in the Okanagan Valley each year since 1969, except in 1972. The maximum was 314 diseased trees in 1977. The total including 1979 is 1,481 affected trees in the Okanagan, and three in the Similkameen Valley. Intensive annual surveys followed by prompt removal of diseased trees, and since 1977, a spray program to eliminate the apple mealy bug in orchards where the disease has been found, appears to have reduced the rate of spread.

Little cherry disease was not found in sweet cherries in the Fraser Valley, despite the presence of infected oriental flowering cherry trees. This may be related to absence of the apple mealy bug to spread the virus.

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La plupart des cerisiers doux (*Prunus avium*) testés dans tous les districts de culture de la région de Kootenay de 1976 à 1979 étaient atteints de la maladie de la petite cerise. Dans le district de culture commerciale Creston, la plupart des cerisiers de 10 ans ou plus étaient infectés, mais plusieurs sujets isolés âgés de 25 à 30 ans étaient indemnes. On a transmis le virus par greffe à des cerisiers Sam provenant de deux arbres à fleurs orientaux (*Prunus serrulata*) trouvés à Creston et de 15 à 30 cerisiers amers indigènes (*P. emarginata*) rencontrés près de vergers abandonnés dans plusieurs districts de Kootenay. On a observé la présence de la cochenille du pommier (*Phenacoccus aceris*), vecteur possible, dans certains endroits atteints récemment par la propagation naturelle de la maladie.

Cette virose se rencontre dans la vallée de l'Okanagan chaque année depuis 1969, sauf en 1972. Le sommet de l'infestation a eu lieu en 1977 avec 314 arbres atteints. Le total atteint 1481 cerisiers infectés dans l'Okanagan (1979 comprise), et 3 dans la vallée Similkameen. Des relevés annuels intensifs, suivis par l'enlèvement rapide des arbres virosés et, depuis 1977, l'application d'un programme de pulvérisation pour lutter contre la cochenille dans les vergers atteints, semblent avoir réduit le taux de propagation.

Les cerisiers doux de la vallée du Fraser sont exempts de la maladie malgré la présence de cerisiers à fleurs orientaux infectés, ce qui pourrait être attribuable à l'absence de la cochenille vectrice à cet endroit.

Introduction

Little cherry disease (LCD) was noticed first affecting sweet cherry (*Prunus avium* L.) in an orchard near Willow Point, on the west arm of Kootenay Lake in 1933. It soon was recognized to be serious because of its drastic effects on size and quality of fruit and its rapid spread from one orchard to another and to every tree in most orchards. By 1946, when it was reported to be caused by a graft-transmissible virus (LCV), it had spread about 32 km north, at least 24 km west and had crossed the lake and reached Creston 72 km southeast of the site of discovery (2).

The rate of spread of LCD appeared to diminish after 1949 (6). By 1949 most cherry trees in the Kootenays were diseased as far west as New Denver but the disease was not found around Nakusp, 50 km to the west on Upper Arrow Lake. It was present at Makinson and Burton 29 and 37 km southwest of Nakusp but was not found at Fauquier-Needles or at Edgewood 22 and 32 km southwest of Burton. A survey in 1960 disclosed no spread of the disease in the intervening 11 years into the Nakusp, Fauquier-Needles or Edgewood plantings. Similarly, by 1949 the disease had affected all trees in the Robson district at the southeastern tip of Lower Arrow Lake, but no diseased trees were found at Syringa Creek, Deer Park, Broadwater or Renata 15 to 35 km westward from Robson either in 1949 or in 1962.

Up to 1964 the disease had not appeared in the Okanagan Valley, and there was no evidence of spread from the Kootenays to sweet cherries in other fruit growing regions of British Columbia or Northwestern U.S.A. (6).

Because of the serious effects of the disease on the cherry industry in the Kootenays there was concern that it might become a serious threat to British Columbia's major cherry producing region, the Okanagan and Similkameen Valleys.

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Under the British Columbia Plant Protection Act (1954), a Little Cherry Control Area was established including the principal little cherry-free cherry growing districts. Movement of all fruit trees and fresh fruits from the Kootenays and other areas into this region was prohibited. The Act provided for inspection of any grower's crop at any time. Surveys supervised by the British Columbia Ministry of Agriculture were conducted annually and all affected cherry trees found were removed. In 1958 the Little Cherry Control Regulations were amended to include eradication and exclusion from the control area of all oriental flowering cherry trees which might be carriers of LCV.

This report includes an up-dating of the status of little cherry disease in the Kootenay, Okanagan, Similkameen and Fraser Valley regions of British Columbia.

Materials and methods

Surveys for LCD were conducted just prior to fruit picking time which ranged from late June through July varying with location and weather. At this time the fruit on normal trees of the dark fruited cultivars, principally Lambert, Bing, Van and Sam, is dark red to black in color. The affected trees can be detected by the smaller size, dull color, sometimes triangular pointed shape and reduced sweetness and flavor of the fruit. The occurrence of affected fruit only on portions of some branches indicates that the trees is showing symptoms for the first time. The occurrence of affected fruit throughout a tree indicates that the tree has been infected for several years (2).

Indexing to confirm the presence of LCV was done by graft transmission to Sam indicator trees (5). The indicator trees were prepared by bench grafting or shield budding from virus-free Sam trees maintained in the Virus-free Budwood Orchard at Summerland, onto virus-free Mazzard Fl2/1 rootstock 9 mm and up in diameter. These trees were planted 60 cm apart in rows 1.5 m apart in an isolation orchard at Summerland. Graft transmission was done by shield or T budding from source to test trees in August. Two buds from each source were grafted into each of two or sometimes into four test trees. The plots of trees were maintained with care to avoid stress from lack of water or nutrients and from fungal diseases or insect pests. The Sam test trees that received buds carrying LCV developed specific

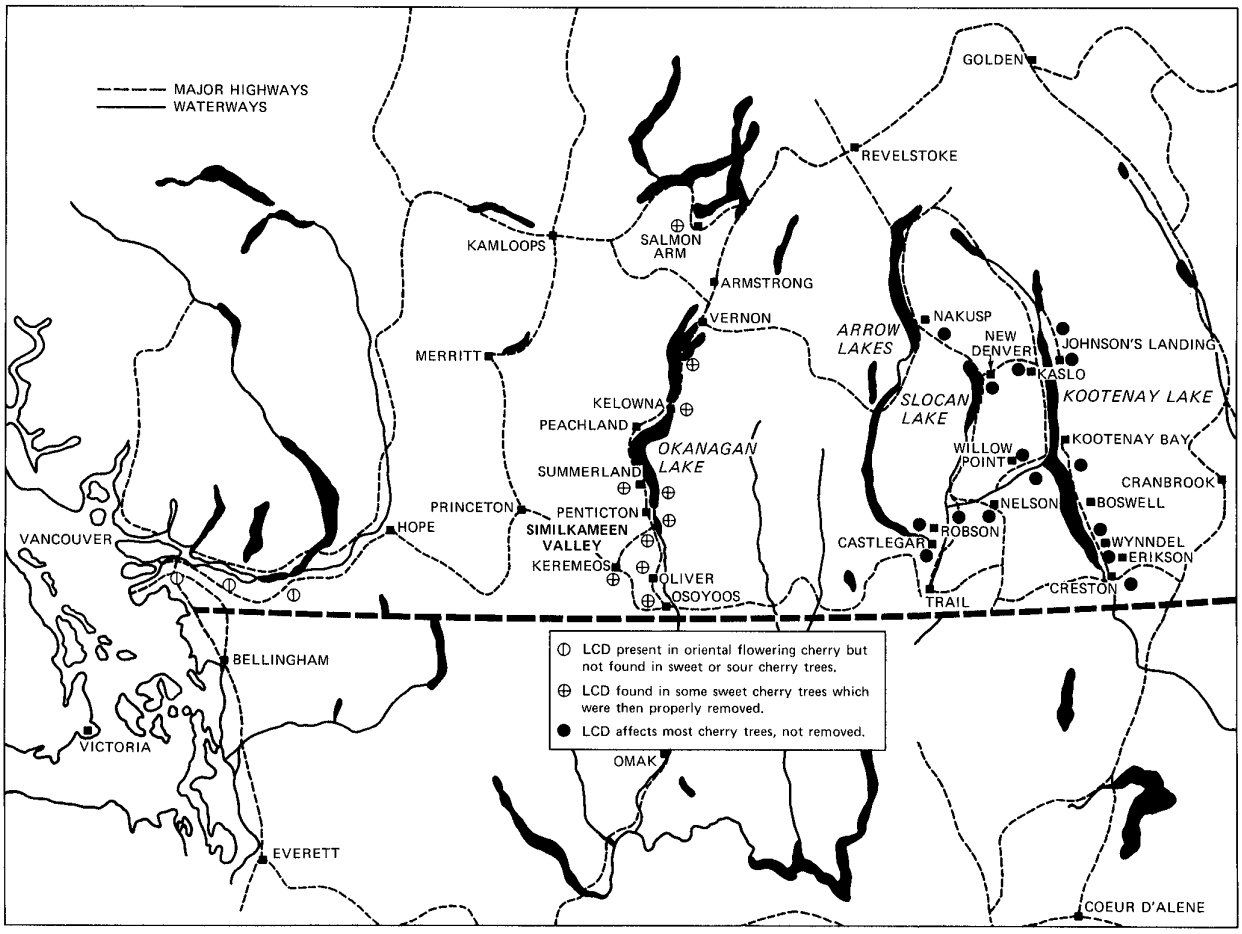

Fig. 1. Distribution of little cherry disease in British Columbia 1969 to 1979.

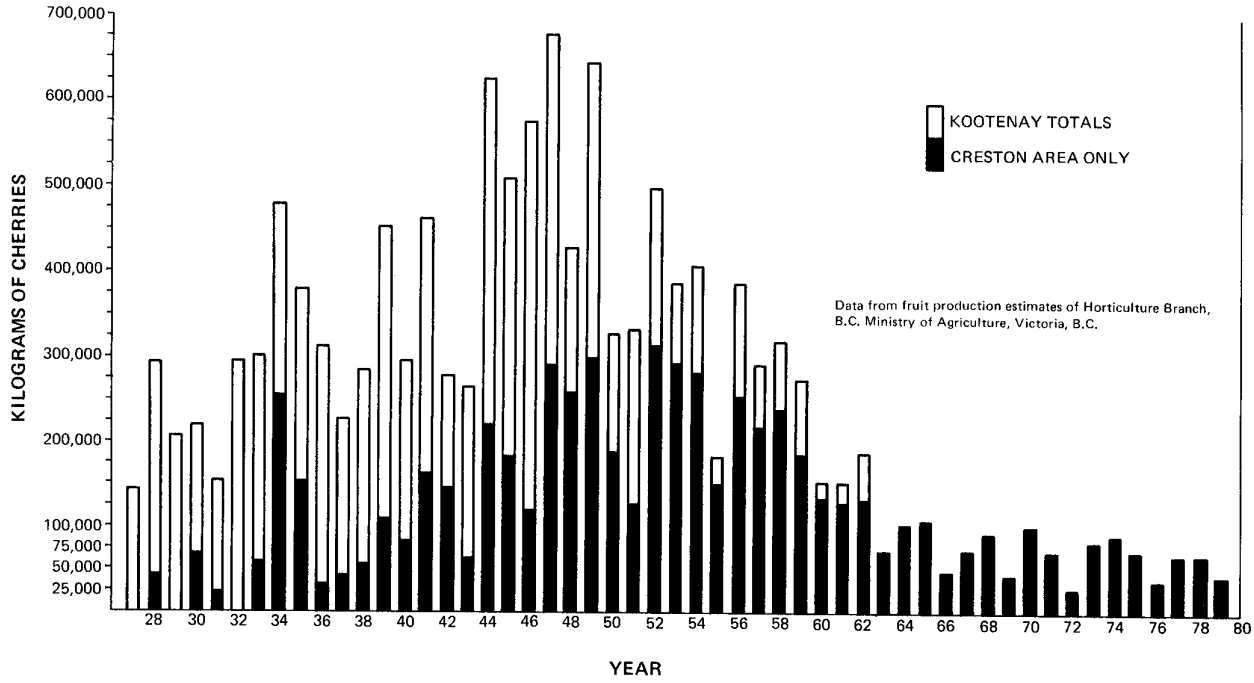

Fig. 2. Sweet cherry production in the Kootenay districts of British Columbia, 1927 to 1979.

interveinal reddening of leaves in August or early September of the following year. If the Sam grafts or buds failed, buds on the Mazzard F 12/1 rootstock were encouraged to develop. Reddening of the leaves of Mazzard F12/1, although not as distinct as on Sam, was useable as an indicator of LCV (4).

Surveys in the Kootenays 1976 to 1979

Among the many small districts scattered through the Kootenays, sites were found where cherries were grown in earlier years in small plantings in gardens or in small orchards seldom as large as two hectares. The plantings were in forest clearings on the gentler slopes or on lower bench lands and usually were within one or two km from the Kootenay, Slocan or Arrow Lakes, or on a valley bottom near a river or creek. Except in the Creston area, few of the cherry trees found in the Kootenays in 1976 to 1979 were receiving cultural attention (Fig. 1). The annual production in the Kootenays from 1976 to 1979, almost all of which was in the Creston area, was 39,000 to 68,000 kg in contrast to 430,000 to 680,000 kg in the years 1946-1949 (Fig. 2). Few of the sweet cherries produced now, even at Creston, are large enough for packinghouse trade so are trucked out privately, sold at roadside stands or sold on a U-pick basis principally to tourists. Little cherry disease appears to be the major factor contributing to the deterioration in size and quality and therefore of quantity of cherries produced in the Kootenay district.

From 1976 to 1979 fruit symptoms indicative of LCD were found on almost all sweet cherry trees examined that had been planted 10 years or longer in all districts where cherry trees were found. The districts in which diseased trees were found included Castlegar, Robson, Deer Park, and Renata near the southeastern tip of Lower Arrow Lake, Nakusp on Upper Arrow Lake, New Denver and Silverton on Slocan Lake, Nelson, Willow Point, Queens Bay, Ainsworth, Mirror Lake, Kaslo and Lardeau on the west side of Kootenay Lake, and Argenta, Johnson's Landing, Kootenay Bay, Wynndel, Creston and Erickson along the east side of Kootenay Lake.

The fruit on old Lambert and the few surviving old Deacon trees in the Kootenay Valley were very small, often 14 mm rarely 20 mm in diameter, and irregular in maturity. The cherries on Bing and Van trees wre generally larger than on Lamberts, but irregular in size and color. Sometimes it was difficult to judge whether the irregular fruit size was caused by LCD or some aspects of cultural neglect. A few Bing trees 25 to 30 years old were found in the Creston district with uniformly large fruit (25 to 28 mm diameter). These include three trees on a residential lot on the western edge of Creston, which indexed negative for LCD on Sam. A planting of about 15 trees, some about 25 years old, on the flats of Wynndel, also have had normal fruit each year (mean diam. 23-28 mm). The remaining trees in an experimental orchard of 1,000 Sam trees which was planted in 1975 in a field on the Creston flats about 2 km from other cherry trees, were still free from LCD in 1979.

Thirty-eight of 46 trees with small fruit symptoms that were indexed were found to carry a graft transmissible agent which caused red leaf symptoms on Sam. Also 3 of 22 trees with normal appearing fruit tested positive when indexed on

Sam. The results indicated that most trees of 10 years or older from all districts where samples were collected, including Robson, Nelson, Willow Point, Kaslo, Johnsons Landing, Creston and Erickson, were infected with LCV.

In most districts in the Kootenays, few or no cherry trees are being planted. Exceptions include a planting of 200 trees near Kaslo in 1975 by a new resident who was not aware of the history of the disease in the area. A number of replacement plantings occur in the Creston-Erickson district where cherries are grown despite the disease. Many of these trees have remained unaffected by LCD for 6 to 10 years, but others located near diseased trees have shown LCD fruit symptoms the first year of fruiting.

Kootenay Bay Lambert trees, which were propagated from a Lambert tree which remained free from LCD in an orchard at Kootenay Bay where all other trees were affected, were distributed for planting in different communities in the Kootenays from 1971 to 1974. In all locations where these trees were observed from 1976 to 1979, some of them already were affected by LCD in each community. They appear to be as readily infected and as severely affected as other Lambert trees. In an orchard at Creston where 27 Lambert, 32 Kootenay Bay Lambert and 5 Van trees were planted in a block in 1971, by 1979 only two trees had developed fruit symptoms characteristic for LCD. Both were Kootenay Bay Lambert trees. They were located at a corner of the planting nearest to (25 m) an old diseased cherry tree. The apple mealy bug (*Phenacoccus aceris* Sig.) which is suspected to be a vector of LCV (3), was found on the affected trees.

The virus was detected, by indexing, in two oriental flowering cherry trees (*Prunus serrulata* Lindl.) on residential lots in Creston, and in 15 of 30 old bitter cherry trees (*P. emarginata* (Hook.) Walp.) growing near old orchard sites in several districts including Robson, Willow Point, Kaslo, Kootenay Bay, Wynndel and Creston.

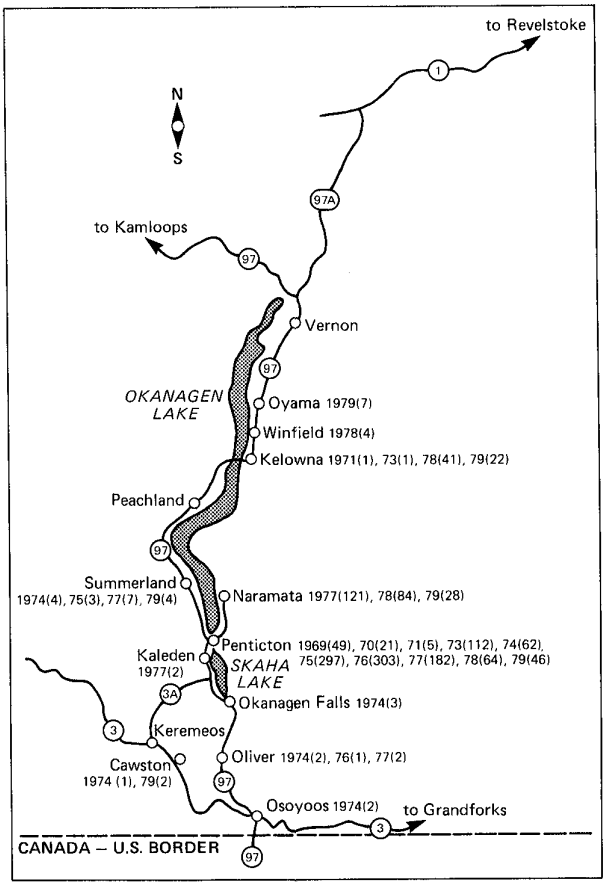

Fig. 3. Numbers of sweet cherry trees found with little cherry disease and removed in the Okanagan and Similkameen (Cawston) Valleys each year including 1969 to 1979.

Surveys in the Okanagan and Similkameen Valleys

Intensive surveys supervised by the British Columbia Ministry of Agriculture did not detect LCD in the Okanagan Valley until 1969 when 49 trees with fruit symptoms characteristic for LCD were found in one orchard near Penticton. All diseased, suspect and adjacent trees were removed in an attempt to eliminate the disease. The presence of LCV in the trees detected by fruit symptoms was confirmed by indexing on Sam, the red-leaf indicator, but the results were not available until a year later. In 1970 only 16 affected trees were found in the same orchard and 5 on scattered residential lots in Penticton. A reduction to only 6 affected trees in 1971, and none in 1972 gave the impression that LCD had been eliminated from the Okanagan Valley, but in 1973 114 affected trees were found. Since then varying numbers of affected trees have been found and removed each year, the maximum being 314 in 1977 (Fig. 1 and 3). The total number of affected trees found in the Okanagan Valley from 1969 to 1979 inclusive is 1,481 out of an estimated total of 155,000 sweet cherry trees (Table 1). In the adjoining Similkameen Valley one affected tree was found in 1974 and two in 1979. The presence of LCV in representative trees from each location has been confirmed by indexing on Sam.

Prior to 1977 the fruit growers had agreed to remove all affected trees before October 1. Commencing in 1977, it was agreed that the affected trees be removed as soon as possible after picking and not later than August 10. In addition it was recommended that before removal of diseased trees the entire orchard should be sprayed with an insecticide (diazinon) to eliminate the nymphs of the apple mealy bug, frequently found on newly affected trees and suspected to be a carrier of LCV. Additional sprays recommended to control mealy bugs in the orchard for the next season included oil with ethion during early spring dormancy, diazinon at petal fall, and diazinon or dimethoate after harvest (1).

Little Cherry Disease in the Lower Fraser Valley

Many thousands of sweet cherry trees occur in the Lower Fraser Valley, most of them in backyard gardens (Fig. 1). They occur in the residential areas of Vancouver and

surrounding municipalities, and near all Valley centers as far east as Hope. Remnants of a few orchards remain near Yarrow, Mission and Chilliwack. Some of the oldest trees occur near old churches in the New Westminster, Fort Langley and Ladner areas. Sour cherries, *P. cerasus* L., also are common in the Lower Fraser Valley, as are numerous volunteer seedlings of both sweet and sour varieties. In addition, some 60,000 ornamental flowering cherries, *P. serrulata*, line the boulevards of Vancouver and many more are found in parks and gardens throughout Vancouver and

Table 1. Cherry trees found with symptoms of little cherry disease in the Okanagan and Similkameen Valleys 1969 - 1979.

Year	Location	Diseased trees found	
		Each location	Total
1969	1 orchard - Penticton	49	49
1970	1 orchard - Penticton	16	
	5 urban lots - Penticton	5	21
1971	1 orchard - Penticton	1	
	4 urban lots - Penticton	4	
	1 urban lot - Kelowna	1	6
1972	none		
1973	7 orchards - Penticton (1 to 22 each)	112	
	1 orchard - Kelowna	2	114
1974	2 orchards - Oliver	2	
	2 orchards - Osoyoos	2	
	1 orchard - Cawston	1	
	1 orchard - Okanagan Falls	3	
	11 orchards - Penticton (1 to 16 each)	48	
	2 orchards - Summerland	4	
	12 urban lots - Penticton	14	74
1975	1 orchard - Penticton	273	
	9 orchards - Penticton (1 to 7 each)	19	
	2 orchards - Summerland	3	
	5 urban lots - Penticton	5	300
1976	1 orchard - Oliver	1	
	1 orchard - Penticton	290	
	3 other orchards - Penticton	4	
	9 urban lots - Penticton	9	304
1977	1 orchard - Kaleden	2	
	2 orchards - Oliver	2	
	4 orchards - Naramata (1 to 110 each)	121	
	30 orchards - Penticton (1 to 56 each)	182	
	2 orchards - Summerland	7	314
1978	4 orchards - Naramata (1 to 65 each)	84	
	15 orchards - Penticton (1 to 14 each)	64	
	10 orchards - Kelowna (2 to 8 each)	41	
	1 orchard - Winfield	4	193
1979	3 orchards - Naramata	28	
	15 orchards - Penticton	46	
	1 orchard - Summerland	4	
	10 orchards - Kelowna	22	
	1 orchard - Cawston	2	
	1 orchard - Oyama	7	109
Total 1969 to 1979			1484

the Lower Fraser Valley. Some flowering cherries planted on boulevards in Vancouver before 1956, when a virus-free program for ornamental cherries was established, induced red leaf symptoms when budded on Sam indicators, and little cherry fruit symptoms when budded on bearing Lambert trees.

A survey in 1977, of 1200 sweet cherry trees in Vancouver and Lower Fraser Valley districts failed to reveal any trees with small fruits characteristic of little cherry disease. In particular, sweet cherry trees near little cherry-infected flowering cherries on Vancouver boulevards did not show symptoms of little cherry disease.

Similarly, in 1979, a limited survey of Lower Fraser Valley districts for apple mealybug suspected to be a vector of little cherry disease in the Okanagan Valley (3) failed to reveal the presence of this insect on any of its common hosts.

Discussion

In the Kootenay region, LCD was present in all districts where cherry trees were found. No concerted efforts have been made to eliminate the disease by removal of all affected trees. Except in the Creston-Erickson district, few trees of 10 years or older are of commercial value because most cherries are very small and poor in flavour and few attempts have been made to control fruit flies, which are prevalent. Although young trees planted near diseased trees in the Creston area usually become affected by LCD before the 8th year, trees grown in several isolated locations have escaped infection for 25 to 30 years. These observations indicate that it may be possible to avoid infection of new cherry plantings in the Kootenays by elimination of diseased trees in the neighborhood before planting and by control of vector insects.

In the Okanagan and Similkameen Valleys where LCD has been detected on a few trees in some orchards in one year and on more trees in the same orchards in subsequent years, there is evidence that such spread can be reduced by sprays to eliminate apple mealybugs, and prompt removal of diseased trees. It appears that the disease could be eliminated from the Okanagan and Similkameen Valleys by vector control and by early detection and prompt removal of all diseased trees. Such a program is handicapped by lack of rapid and reliable methods for detecting infection in trees that do not display fruit symptoms.

Failure to detect LCD in sweet cherries in the Fraser Valley, despite the presence of oriental flowering cherries infected with LCV, may be attributed to absence of apple mealybugs to transmit the virus to sweet cherry.

Literature cited

1. Anonymous. 1980. Tree-fruit production guide for interior districts. Province of British Columbia Ministry of Agriculture. 66 pp.
2. Foster, W.R. and T.B. Lott. 1947. "Little Cherry", a virus disease. Sci. Agr. 27:1-6.
3. McMullen, R.D. 1979. A progress report on little cherry disease vector studies. Unpublished paper presented to Entomol. Soc. Canada, Annual Meeting, Vancouver, B.C., Oct. 2.
4. Posnette, A.F. 1964. Little cherry disease. Plant Pathol. 13:170-172.
5. Wilks, J.M. and M.F. Welsh. 1955. Sweet cherry foliage indicator hosts for the virus that causes little cherry. Can. J. Agr. Sci. 35:595-600.
6. Wilks, J.M. and M.F. Welsh. 1964. Apparent reduction in little cherry disease spread in British Columbia. Can. Plant Dis. Surv. 40:126-130.
7. Yorston, J. 1977. Summary of cherry trees with little cherry virus in the Okanagan Valley. Unpublished report, Plant Pathology Branch, B.C. Ministry of Agriculture, Kelowna, B.C.
8. Yorston, J. 1978. Summary of little cherry survey in 1978. Annual Report, Entomology - Plant Pathology Branch, B.C. Ministry of Agriculture, Summerland, B.C.
9. Yorston, J. 1979. Little cherry survey, 1979. Unpublished report, Entomology - Plant Pathology Branch, B.C. Ministry of Agriculture. Summerland, B.C.

Soil compaction effect in clay soils on common root rot in canning peas[3]

B. Vigier[1] and G.S.V. Raghaven[2]

Root rot problems in canning peas (*Pisum sativum*) were observed during the growing season of 1979 on 12 farms located near the St-Jean and Rougemont regions of southwestern Québec. Root rot is caused mainly by *Fusarium solani* f. sp. *pisi*, a fungus that tends to increase root damage under soil compaction.

A soil dry bulk density increase of 26 percent was observed for farms using higher powered tractors with tire contact pressures ranging from 38 kPa to over 50 kPa.

Can. Plant Dis. Surv. 60:4, 43-45, 1980.

Durant l'été 1979, les dommages causés, sur le pois de conserverie (*Pisum sativum*) par le pourridié fusarien, furent évalués sur 12 fermes situées au sud-ouest du Québec dans la région de St-Jean et de Rougemont. La cause de cette maladie est d'origine fongique, soit principalement le *Fusarium solani* f. sp. *pisi*. Une augmentation de la densité apparente du sol favorise cette maladie.

Un accroissement de la densité apparente du sol de 26% fut observé sur des fermes utilisant des tracteurs à haute puissance dont la répartition de charge par pneu variait de 38 kPa à plus de 50 kPa.

Introduction

The common root rot of peas (*Pisum sativum* L.), is a complex disease, primarily incited by the fungus, *Fusarium solani* (Mart.) Sacc. f. sp. *pisi* (F.R. Jones) Synd. & Hans. (1). Results of experimental and field observations show that severe root rot can cause a 57% loss in pea yield (2). Natural root obstruction in compacted and stoney soils is an important factor in the root rot problem, according to Burke *et al.* (3). Later Burke *et al.* (4) gave field results showing non-existence of root rot in plots where pea roots could easily penetrate the soil; whereas, compacted or hard soils increased pea root rot by delaying root extension out of the pathogen infested plowed layer into the sparsely infested subsoil.

Soil types with high water retaining capacities, such as clay soils, were shown by Temp and Hagedorn (9) to have a slower decrease in root rot potential over years of cropping; and heavy clay soils are more favourable to the development of root rot than lighter soils as shown by Jones *et al.* (5). This study was undertaken to assess the effects of soil compaction on the severity of root rot in peas in southwestern Québec.

Materials and methods

During the growing season of 1979, twelve fields were selected in two regions of southwestern Quebec, namely St-Jean and Rougement, on clayey soil types of Ste. Rosalie soil series with improved drainage (Table 1). Pea varieties were selected according to heat units; 843°C and over were referred to late season varieties. All fields had a minimum of five years rotation with grain crops to reduce residual effect of pea root rot.

Table 1. Pea variety, soil texture, soil series[b] and soil dry bulk density for investigated fields.

Farm[a] No. Pea variety	Surface soil texture (0 to 20 cm)	Mean Soil Dry Bulk Density (15 to 20 cm Zone) (kg/m^3)	C.V.(%)[c]
1. Trojan	Clay†	1203	10.2
2. Medalist	Silty clay loam†	1418	5.2
3. Trojan	Silty clay†	1494	4.9
4. Medalist	Silty clay loam†	1427	4.4
5. Medalist	Silty clay loam†	1527	2.9
6. Trojan	Clay loam†	1289	6.7
7. Champ	Silty clay§	1136	8.6
8. Champ	Clay†	1287	9.6
9. Champ	Silty clay loam†	1203	9.6
10. Champ	Clay§	1266	17.6
11. Perfection 5C	Clay†	1420	5.3
12. Champ	Clay§	1546	5.1

[a] Fields 1-6 and 7-12 were located in Rougemont and St-Jean areas, respectively.

[b] All Ste Rosalie except no. 7, Richelieu. Ref. Quebec Agriculture Department 1942. Soil Map, scale 1:5280.

[c] C.V. = coefficient of variation.

† Subsurface drainage

§ Surface drainage only.

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Severity of pea root rot in field grown plants was judged in four categories ranging from zero to three using a method reported by Basu *et al.* (1) and a disease index was computed from this data to assess damage severity, using the following equation:

$$RRDI = \frac{[\Sigma \text{ (no. plants per category)} \times \text{(no. of category)}] \times 100}{\text{(number of categories - 1)} \times \text{(total no. of plants)}} \quad (1)$$

where, RRDI = root rot disease index. Soil preparation varied slightly from one field to another with a fall plowing followed by two discings and one harrowing in the spring, and rolling after seeding. The differences in tractor sizes used for soil preparation resulted in different levels of soil compaction. Tractor weight, power and rear tire size were recorded to assess soil compaction; each region had three farms having 45kW (60 hp), 63 kW (85 hp) and 75 kW (100 hp) tractors, whereas the remaining three farms had 134 kW (180 hp) tractors, as shown in Table 2.

Table 2. Tractors' model weight† and contact pressures related to engine power and tire size.

Field No.	Engine Power (kW)	Weight per Rear-tire (kg)	Tire Width X Diameter (cm)	Contact Pressure (kPa)
1	45	1474	39.4 X 96.5	38.3
2	134	2310	46.7 X 96.5	50.1
3	134	2310	-	50.1
4	134	2310	-	50.1
5	63	2023	-	44.2
6	75	2051	-	45.1
7	45	1474	39.4 X 96.5	38.3
8	75	2051	46.7 X 96.5	45.1
9	63	2023	-	44.2
10	134	2310	-	50.1
11	134	2310	-	50.1
12	134	2310	-	50.1

†Based on Nebraska Tractor test.
Ref. Agricultural Engineers Year Book, 1975-79.

Data were collected from a sampling plot of 5 m × 5 m located near the centre of each field. Average soil dry bulk density from the surface to depths of 5, 10, 15 and 20 cm was determined using a single probe Troxler density gauge (7). Soil samples were collected for gravimetric soil moisture-content determination (7). There were 4 replications per plot. Data were transformed into soil dry density at four consecutive depths, 0 to 5, 5 to 10, 10 to 15 and 15 to 20 cm according to equations reported by Taylor *et al.* (8).

Root rot was evaluated at the plant flowering stage. In each plot 50 plants were sampled from five different places. Data on plant population and yield of shelled peas over a 1 m × 1 m subplot per field at harvest time were also collected.

Results and discussion

Pea cultivars, soil series, soil texture, surveyed farms identification and soil dry bulk density are listed in Table 1 for both Rougemont and St-Jean regions.

Table 3. Yield results.

Field No.	Fresh yield[a] per m^2 (g)	Fresh yield[b] per ha (kg)	Dry yield[c] per m^2 (g)	Plant population per m^2
1	-	-	-	-
2	305.3	4092	56.5	128
3	363.0	4549	60.6	120
4	449.4	3759	86.9	100
5	349.3	3403	78.3	92
6	191.3	-	38.3	60
7	100.1	-	18.7	100
8	259.5	3005	43.9	100
9	175.3	836	26.6	68
10	361.1	1469	72.6	108
11	101.9	1086	20.0	160
12	133.6	1469	26.1	108

[a]Fresh yield of shelled peas, based on 1 m^2 sample.
[b]Overall fresh yield of shelled peas given by processors.
[c]Dry yield of shelled peas, based on 1 m^2, over-dry basis.

In both regions there is an increase in soil dry bulk density of 26 percent in the depth range of 15 to 20 cm attributed to the use of higher contact pressures of the tractors (Tables 1 and 2). The relation between tractor contact pressures and soil dry bulk density is linear and significant at 0.05 level with a coefficient of determination (R^2) of 48% for the 12 farms.

A correlation between soil dry bulk density and root rot disease index (RRDI) was established. The results are shown in Figure 1 for the St-Jean and Rougemont areas. There is a linear relation between root rot disease index and soil dry bulk density. The equation of the line is given by:

$$RRDI = -70.86 + 0.07\,(\gamma \text{ dry}) \quad (2)$$

where, γ dry = Soil dry bulk density (kg/m^3) in the depth range of 15 to 20 cm whose coefficient of determination (R^2) is 37 percent (significant at 0.05 level) for the 11 farms. The farm no. 9 shown in Figure 1 does not fit the equation (2) shown above, because the field was waterlogged by heavy rainfalls after seeding and this resulted in a poor germination rate and a high level of root rot on remaining plants. The equation of the line strongly indicates that soil compaction could promote root rot severity. The effect of compaction on yield was investigated using an estimate of yield loss caused by root rot. The following equation developed by Basu *et al.* (2) was used:

Estimated yield loss percentage

$$= \text{(Percentage of plants in root rot category no. 3)} \times (0.57) \quad (3)$$

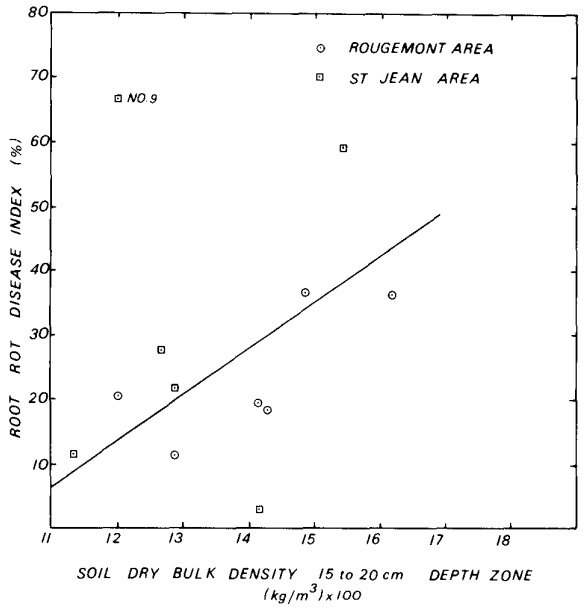

Figure 1. Root rot disease index plotted against soil dry bulk density for both Rougemont and St-Jean areas.

Results gave a maximum yield loss of 4.5 percent and did not correlate with soil compaction. However, these estimated yield loss figures are not in accordance with the 78 percent difference observed in actual fresh yield per m^2. Yield information was also obtained by the processors on an hectare basis, for the 9 farms out of the 12 farms under study and this data correlated well with the yield samples collected ($R^2 = 0.523$ at 0.05 level) indicating that the sampling procedure is in fact a good representation of total field yield (Table 3) and yield variation. Low yield losses obtained with the equation (3) might be biased because of the early root rot severity evaluation which was done at the flowering stage, 10 or 12 days prior to harvest. It is possible that disease might have developed during this period and created more damage than what was estimated. However, soil compaction has been reported to reduce corn silage yields up to 50 percent in Ste. Rosalie clay soil, Raghavan *et al.* (6).

This phenomenon might occur with pea production which is often in rotation with corn on this same soil series. Further studies are required to assess yield loss predictions with soil compaction.

Conclusions

In this study the effects of soil compaction on pea root rot infection were specifically considered. Root rot disease index varied from 3 to 59 percent on both regions where disease index response was found to be dependent on soil dry bulk density. Soil dry bulk density increment was found to be dependent on tractor contact pressures in both regions investigated. Therefore, an increase in dry bulk density resulting from soil compaction can promote root rot damage. Further research in this field is necessary to establish the full impact of soil compaction on canning peas yield loss.

Acknowledgments

The authors wish to thank the various pea processors for their cooperation; MM. G. Samoisette and M. Rémillard for their valuable technical assistance. In addition, critical comments of Dr. Sackston, Plant Science Department of Macdonald Campus are also appreciated.

Literature cited

1. Basu, P. K.; R. Crête; A. G. Donaldson; C. O. Gourley; J. H. Haas; F. R. Harper; C. H. Lawrence; W. L. Seaman; H. N. W. Toms; S. I. Wong and R. C. Zimmer. 1973. Prevalence and severity of diseases of processing peas in Canada, 1970, 71. Can. Plant Dis. Surv. 53:49-57.
2. Basu, P. K.; N. J. Brown; R. Crête; C. O. Gourley; H. W. Johnston; H. S. Pepin and W. L. Seaman. 1976. Yield loss conversion factors for fusarium root rot of pea. Can. Plant Dis. Surv. 56:25-32.
3. Burke, D. W.; D. J. Hagedorn and J. E. Mitchell. 1969. Aphanomyces and fusarium root rot of peas with partial vs total exposure of roots to infested soil. Phytopathology 59:1261-1266.
4. Burke, D. W.; D. J. Hagedorn and J. E. Mitchell. 1970. Soil conditions and distributions of pathogens in relation to pea root rot in Wisconsin soils. Phytopathology 60:403-406.
5. Jones, F. R. and M. B. Linford. 1925. Pea disease survey in Wisconsin. Wis. Agr. Expt. Sta. Res. Bul. 64:31 pp.
6. Raghavan, G. S. V.; E. McKyes; G. Gendron; B. K. Borglum and H. H. Le. 1978. Effects of tire contact pressure on corn yield. Can. Agr. Eng. 20:34-37.
7. Raghavan, G. S. V.; E. McKyes; I. Amir; M. Chasse; R. S. Broughton, 1976. Prediction of soil compaction due to off-road vehicle traffic. Trans. Amer. Soc. Agr. Eng. 19(4):610-613.
8. Taylor, F.; E. Douglas; S. Negi; G. S. V. Raghavan and E. McKyes. 1980. The effects of minimum tillage on corn growth in Eastern Canadian Agricultural soils. Final report. Engineering and Statistical Research Institute Research Branch, Agriculture Canada, Ottawa (Ontario) File no. 07SZ.01843-9-0904:29-30.
9. Temp, M. V. and D. J. Hagedorn. 1967. Influence of cropping practices on Aphanomyces root rot potential of Wisconsin pea fields. Phytopathology 57:667-670.

A chlorotic mosaic of fall hawkbit (Leontodon autumnalis)

R.P. Singh[1] *and J.G. McDonald*[2]

A short rigid rod-shaped virus with length classes of 140 nm and 160 nm was found to cause a chlorotic mosaic in 'fall hawkbit' (*Leontodon autumnalis*), a common weed found in potato-growing areas of eastern Canada. The 'fall hawkbit' virus (FHV) is serologically related to Hypochoeris mosaic virus (HMV), a recently described virus found in western Canada. In spite of other similarities between FHV and HMV there were some apparent differences in host range.

Can. Plant Dis. Surv. 60:4, 47-50, 1980.

Un virus court, rigide et à forme de bâtonnet de 140 à 160 mm de longueur est responsable d'une mosaïque chlorotique du liondent d'automne (*Leontodon autumnalis*), mauvaise herbe commune rencontrée dans les régions de culture de pommes de terre de l'est du Canada. Le virus est sérologiquement apparenté au virus de la mosaïque de l'hypochoéris, virus récemment décrit et répandu dans l'ouest du Canada. Malgré certaines autres similarités entre ces deux types de virus, ils affichent certaines différences dans la spécificité de leur hôte.

Introduction

The 'fall hawkbit' (*Leontondon autumnalis* L.) is a common weed in potato-growing areas of eastern Canada. In a survey for possible perennial weed hosts of potato viruses, this species was frequently observed showing a striking yellow mosaic. Since it had been reported from Europe that tobacco rattle virus (TRV) causes a chlorotic spotting in *Hieracium* L. (2), a genus closely related to *Leontodon*, it was initially speculated that TRV might be the causal agent of the disease in 'fall hawkbit'. Studies were therefore commenced to examine this possibility. However, during the course of this investigation a newly described virus named Hypochoeris mosaic virus (HMV) was reported to infect another close relative of *Leontodon, Hypochoeris radicata* L. (1) in western Canada. We here report that the chlorotic mosaic of 'fall hawkbit' is not caused by TRV but by a virus similar to HMV.

Materials and methods

Test plants were grown in a mixture of soil, peatmoss, and sand (2:1:1) in 10 cm clay pots. The virus was propagated in *Nicotiana tabacum* L. cv. Samsun, in a greenhouse maintained at 14-18°C. Infectivity assays were made on *Chenopodium amaranticolor* Costs & Reyn.

Leaf cell extracts were negatively stained with 2% ammonium molybdate, pH 7, using the leaf chopping method (5) and were examined with a Philips 201C electron microscope. The magnification was calibrated as before (5).

Serological testing was done with the SDS-agar diffusion method (4). Antisera to morphologically similar viruses were kindly supplied by Drs. R. Stace-Smith (HMV), H. Huttinga (five isolates of TRV), and L. Bos (Pea early browning virus).

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Results

The perennial weed, 'fall hawkbit', was commonly found on pasture lands and field borders. Naturally infected 'fall hawkbit' plants were observed in the spring (May to early June) when maximum daily temperatures were about 10-15°C. Often 5-20% of the plants in an area were symptomatic. The newly developing leaves showed chlorotic spotting (Fig. 1). This symptom became less apparent and disappeared when temperatures reached about 25°C but reappeared late in October when temperatures were cooler. Recovery of viral infectivity was invariably associated with the presence of this symptom; its disappearance in summer coincided with a loss of infectivity.

The virus from 'fall hawkbit' (FHV) was initially isolated by grinding the fresh leaf tissue in glycine-phosphate buffer (0.05M glycine + 0.03M K_2HPO_4, pH 9.2), and inoculating to Samsun tobacco, but few plants became infected (3/20). The success rate in transmission was not improved by attempting to stabilize the viral genome with a phenol extraction procedure (7). Frozen leaf samples lost infectivity within 3 days, and dilution of sap over 1:50 abolished the infectivity. However, greater efficiency in transmission was obtained when infected Samsun tobacco or *Gomphrena globosa* L. were used as source plants. The following plants were susceptible to FHV.

Chenopodium amaranticolor - Chlorotic local lesions appeared in 7-10 days (Fig. 6); the lesions extended in diameter and sometimes spread to veins, where subsequent necrosis led to premature leaf abscission.

Datura ferox Nees and *D. aegyptica* Vesl. - Oak leaf pattern and veinal necrosis (Fig. 3) developed in 7-14 days.

Gomphrena globosa - Chlorotic to water soaked lesions appeared in 7-10 days. These spread to the veins, causing veinal necrosis and premature leaf wilting, followed by systemic symptoms of necrotic lesions, dark veins and reduction in leaf size (Fig. 7).

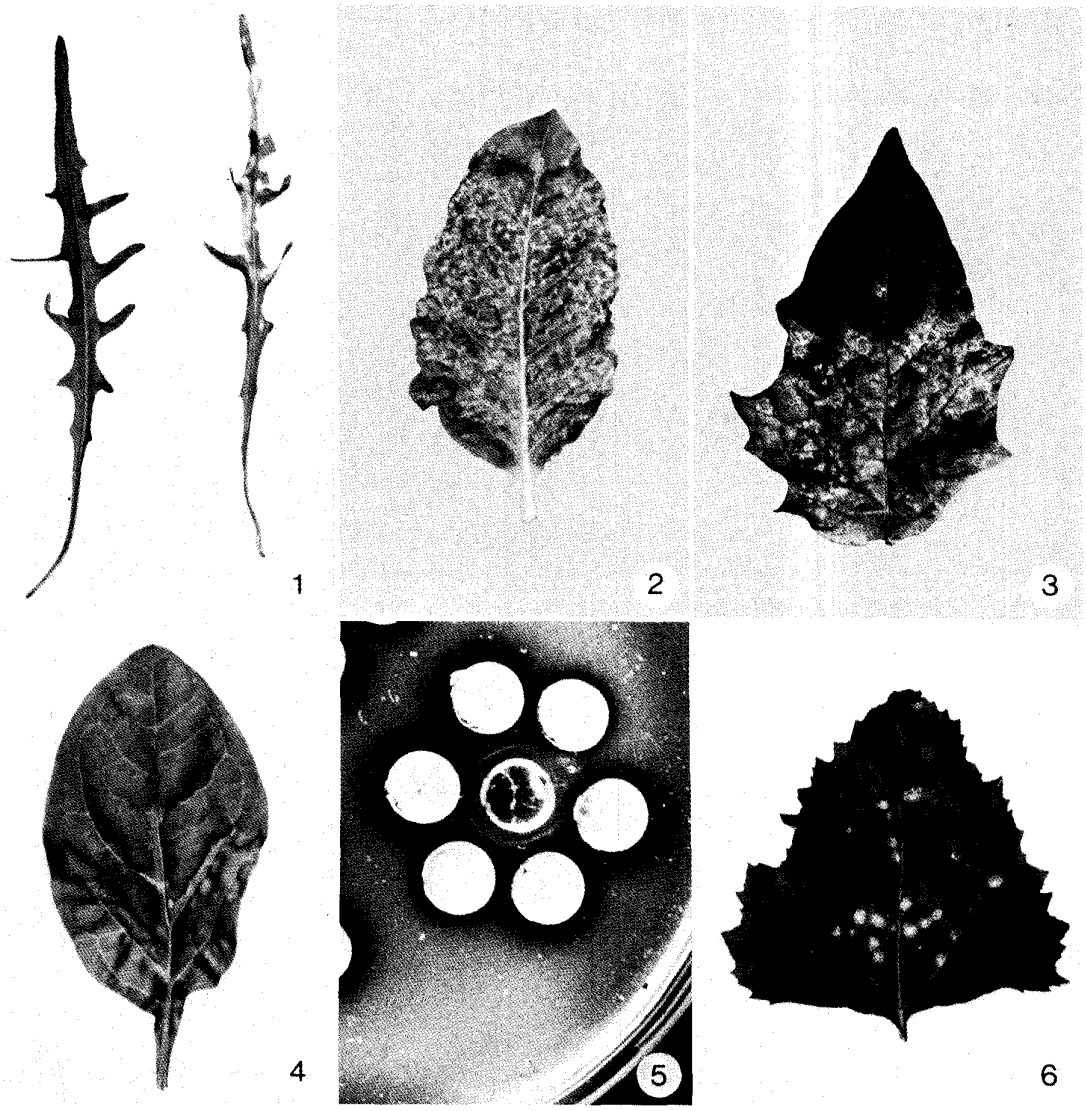

Fig. 1. Left, healthy fall hawkbit leaf; right, naturally infected leaf with chlorotic mosaic.

Fig. 2. Systemically infected Samsun tobacco leaf showing oak leaf pattern.

Fig. 3. Systemically infected *Datura aegyptica* leaf showing necrosis and oak leaf pattern.

Fig. 4. Inoculated *Petunia hybrida* leaf showing veinal necrosis.

Fig. 5. Results of serological reactions in SDS-agar with HMV antisera in the center well: a) sap from FHV-infected Samsun tobacco, b) sap from healthy tobacco, c) sap from healthy fall hawkbit leaves.

Fig. 6. Local lesions on a *Chenopodium amaranticolor* leaf induced by FHV.

Nicandra physaloides Gaertn. Necrotic spots were observed. Not all the inoculated or systemic leaves developed symptoms.

Nicotiana tabacum cvs. Samsun and White Burley - Necrotic lesions along the mid vein, ring spot lesions on the lower leaves or oakleaf patterns (Fig. 2) appeared in 7-10 days. A mild mosaic developed systemically.

Petunia hybrida Vilm. - Necrotic blotches (Fig. 4) along the main veins developed within 2 weeks.

Physalis pruinosa L. - Various degrees of leaf necrosis and leaf wilting were observed after 3-4 weeks.

No viral infectivity was recovered on back-testing of inoculated and uninoculated leaves of the following species: *Nicotiana glutinosa* L., *Phaseolus vulgaris* L. (7 cultivars),

Fig. 7. Systemically infected *Gomphrena globosa* plants showing stunted leaves with necrotic spots and veins.

Pisum sativum L. (5 cultivars), *Scopolia sinensis* Hemsl and *Solanum tuberosum* L. (8 cultivars).

The virus was not transmitted by three species of aphids, i.e., *Myzus persicae* (Sulzer), *Aulacorthum solani* (Kltb.), and *Macrosiphum euphorbiae* (Thomas), from infected Samsun tobacco to healthy tobacco in 3 different tests.

Electron microscopy. Negatively stained extracts from FHV-infected tobacco and fall hawkbit leaves contained very low concentration of short rigid rods, about 23 nm in diameter (Fig. 8). Measurement of the lengths of 328 particles revealed two main size classes of 140 nm and 160 nm (Fig.9).

Serology. Antisera to five isolates of TRV (8) and an antiserum to pea early browning virus, failed to react in SDS-agar with FHV-infected tobacco or 'fall hawkbit' sap. However, a specific reaction was observed (Fig. 5) to an antiserum prepared against HMV.

Discussion

Although FHV showed some resemblance to TRV, particularly in symptomatology, it did not react with several antisera prepared against European strains of TRV. Thus the virus in question is not TRV as had earlier been speculated (6). However, FHV reacted specifically with an antiserum to HMV, a newly described virus from another species of Compositae. In spite of the similarities to HMV, some differences in host range were observed with the 'fall hawkbit' isolate; e.g., it infected *Gomphrena, Datura, Physalis* and *Petunia* species which were not infected by HMV (1).

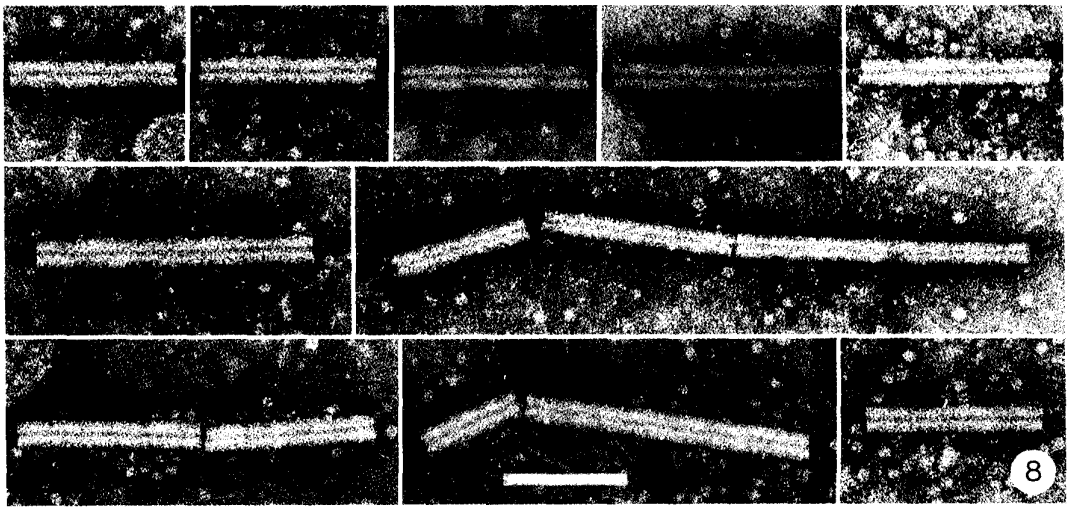

Fig. 8. Particles in leaf extracts of FHV-infected fall hawkbit negatively stained with 2% ammonium molybdate, pH 7. Bar = 100 nm.

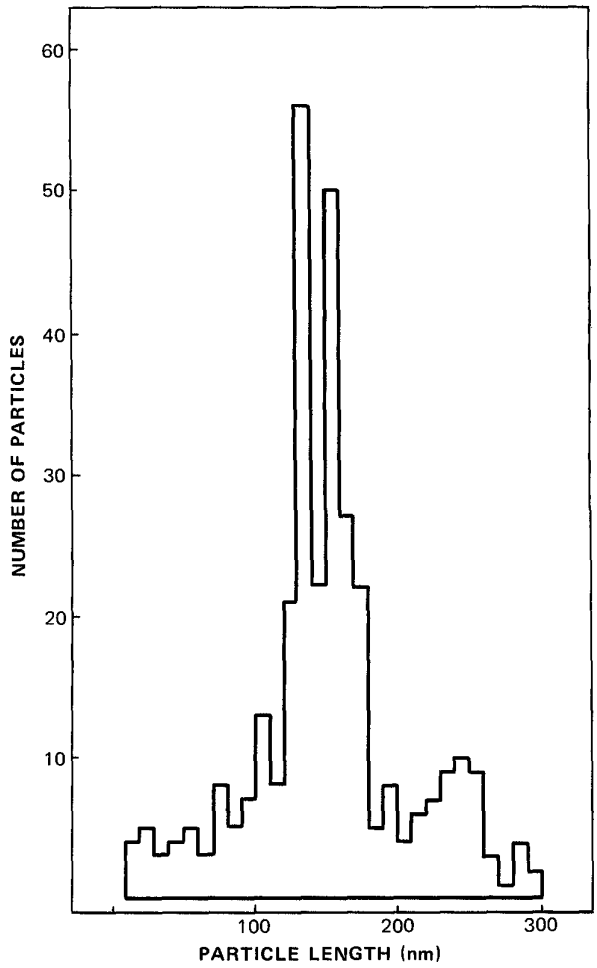

Fig. 9. Length distribution of particles found in negatively stained extracts of FHV-infected fall hawkbit.

Literature cited

1. Brunt, A.A., and R. Stace-Smith. 1978. Some hosts, properties and possible affinities of a labile virus from *Hypochoeris redicata* (Compositae). Ann. Appl. Biol. 90:205-214.
2. Cooper, J.I. and P.R. Thomas. 1970. *Trichodorus nanus*, a vector of tobacco rattle virus in Scotland. Pl. Path. 19:197.
3. Cooper, J.I. and B.D. Harrison. 1973. The role of weed hosts and the distribution and activity of vector nematodes in the ecology of tobacco rattle virus. Ann. Appl. Biol. 73:53-66.
4. Gooding, G.V., Jr., and W.W. Bing. 1970. Serological identification of potato virus Y and tobacco etch virus using immunodiffusion plates containing sodium dodecyl sulfate. (Abstr.) Phytopathology 60:1293.
5. McDonald, J.G., and M.C. Heath. 1978. Rod shaped and spherical virus-like particles in cowpea rust fungus. Can. J. Bot. 56:963-975.
6. Singh, R.P. 1978. Presence of tobacco rattle virus in hawkbit. Fredericton, Res. Sta., Research Summary 1977; pp. 31.
7. Singh, R.P. and R.H. Bangall. 1968. Infectious nucleic acid from host tissues infected with the potato spindle tuber virus. Phytopathology 58:696-699.
8. Van Hoff, H.A., D.Z. Maat, and J.W. Seinhorst. 1966. Viruses of the tobacco rattle virus group in Northern Italy: their vectors and serological relationships. Neth. J. Pl. Path. 72:253-258.

Occurrence of Ascospores of Sclerotinia sclerotiorum in areas of central Alberta[1]

Jill R. Williams[2] and D. Stelfox[3]

Ascospores of *Sclerotinia sclerotiorum* were intercepted more frequently, on agar plates and flowering rapeseed plants, in areas north and west of Edmonton, than in areas to the south and east. There did not appear to be a correlation between total rainfall during the summer and ascospore incidence.

Can. Plant Dis. Surv. 60:4, 51-53, 1980.

Les ascospores de *Sclerotinia sclerotiorum* ont été interceptées plus fréquemment sur gélose d'agar et sur plantes de colza en fleur dans les régions situées au nord et à l'ouest d'Edmonton que dans les parties situées au sud et à l'est de la province. Il ne semble pas y avoir de corrélation entre la pluviométrie totale d'été et la fréquence des prélèvements de spores.

Introduction

Rapeseed produced in south-central and east-central Alberta is rarely as heavily contaminated with sclerotia as rapeseed produced in north- and west-central regions. (Canadian Grain Commission weekly reports on Sclerotinia pieces in oilseed clean-out at Vancouver terminals, for years 1976-1979). The presence of airborne ascospores of *Sclerotinia sclerotiorum* (Lib.) de Bary in central Alberta in July - August 1978 was shown by Williams and Stelfox (1979), and potential for long range dispersal by these spores was suggested. Lower levels of infection by *Sclerotinia*, found in drier areas, may be due to lower ascospore production in these areas, or to reduced survival of incoming ascospores because of dry conditions. The occurrence of ascospores in seven locations, with differing precipitation levels, in central Alberta was monitored in summer 1979.

Materials and methods

Presence of airborne ascospores of *S. sclerotiorum* was determined in two ways: 1) by exposing agar, in petri plates, and incubating after exposure so colonies of *Sclerotinia* could be identified; 2) by placing flowering rapeseed plants in fields for approximately one week, then incubating in humid conditions suitable for disease development.

1) Exposure of agar plates

A set of ten wooden stakes was placed in one field on each of four farms, north, south, east and west of Edmonton, Alberta. Two agar-containing petri plates could be attached vertically on each stake at approximately 60 cm and 125 cm above ground level. Stakes were placed on the east side of the field, at least 30 m apart, with plates facing west, the direction of prevailing winds. Field location and cropping history are given in Table 1. Petri plates (8.5 cm in diameter) contained acidified potato sucrose agar which is suitable for growth of *S. sclerotiorum* and suppression of bacterial contaminants. Each set of plates (20 per field) was exposed for an average of 5.5 hours (4.5 - 7.5 hrs.), from 10 am - 3:30 pm, but on five occasions plates were exposed overnight, for 17 - 21 hrs at field N (twice) and field K (three times). After exposure plates were incubated at room temperature for a minimum of two weeks, with frequent examination for development of Sclerotinia colonies. Plates were exposed during the period June 21 - August 9, with seven exposure dates at D and T, six at N and 3 at K.

2) Exposure of flowering plants

Two varieties of rapeseed, Candle (*Brassica campestris* L.) and Midas (*B. napus* L.) were grown to flowering stage, in 4" pots in growth chambers where they were not exposed to spores of *Sclerotinia*. Each pot containing 4 - 5 plants was well-watered, enclosed in a polythene bag to prevent moisture loss in the field, and plants and pots were transported to the field in a polythene bag to prevent exposure to spores en route. Pots were placed in holes dug at the edge of the crop, so that pot soil was level with the soil surface. Five pots of each variety were placed in each field, on the east side, at least 30 metres apart. After exposure each pot of plants was again transported to the laboratory enclosed in a polythene bag; plants were then misted with tap water to run-off point and kept in high humidity at 20 - 22°C. Plants were periodically examined for infection by *Sclerotinia*, for up to five weeks after exposure; when sclerotia formed on any part of plants in one pot, infection was recorded and plants discarded. As a control, bagged plants were taken to and from the field, but not exposed, then misted and incubated as for exposed plants.

Total precipitation occurring during the summer was recorded at weather stations in Alberta. Data recorded at stations closest to fields where agar plates and plants were exposed are shown in Table 1. Unfortunately some stations

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Table 1. Location and cropping of fields

Field	Direction from Edmonton	Distance (km) approx. from Edmonton	Crop grown in 1978	Crop grown in 1979	Spore trapping method	Total rainfall from May 1 - Aug 29, 1979 (mm)
N	north	12.5	wheat	rapeseed	plates	246.5
T	southeast	60	rapeseed	wheat	plates	180.6
K	south	30	rapeseed	barley	plates	306.1
D	west	7.5	rapeseed	barley	plates	246.5
W	north	80	rapeseed & Alsike clover	clover	plants	259.6*
B	northeast	110	rapeseed	barley	plants	253.1*
V	east	87.5	summerfallow	rapeseed	plants	180.6
H	southeast	80	rapeseed	rapeseed	plants	180.6
G	southeast	140	rapeseed	rapeseed, wheat	plants	252.8
L	south	110	rapeseed	barley	plants	108.9
S	northwest	90	rapeseed	barley	plants	259.6*

*Rainfall from June 11 - August 20, 1979

were 40 - 50 km from field locations and values are not always applicable to the fields.

Results

1) Exposure of agar plates

Average percentages of plates developing colonies of *S. sclerotiorum* (Table 2) indicate the differences in presence of spores in the four fields, with high values obtained in north and west areas (N and D) and low values in south and east (K and T). Spores were intercepted every time plates were exposed at N and D (13 exposure times) and less frequently at T and K (6 out of 9 exposure times). Higher numbers of plates developed colonies at N and D on most exposure dates, and spores were intercepted earlier at N and D than at T. Equal numbers of plates at the two heights on the stakes developed *S. sclerotiorum* so data for the two heights were combined for each exposure.

Table 2. Interception of ascospores of *S. sclerotiorum* by agar plates at four locations

Date	% plates* on which colonies developed after exposure at location: N	D	K	T
June 18 - 24	40	10		
June 25 - July 1	70	50		0
July 2 - 8		50		0
July 9 - 15	95	65	5	
July 16 - 22	100	95	15	10
July 23 - 29		100		50
July 30 - Aug. 5	78	95	40	0
Aug. 6 - 12	100			25
Mean	80.5	66.4	20	14.2

*Values based on total of 20 plates at each location, each time, except at N in last two weeks, when 18 plates were exposed each time.

Table 3. Interception of ascospores of *S. sclerotiorum* by rapeseed plants at seven locations.

Date on which plants put in field	% plants* developing Sclerotinia infection after exposure at location: L	G	H	V	S	B	W
June 18 - 24					30	60	100
June 25 - July 1	20	20	40	20			
July 2 - 8	20	20	10	20	30		
July 9 - 15	0	0	10	30	60	50	20
July 16 - 22			0	0			
July 23 - 29			10	0	0**		
July 30 - Aug. 5	10	10	0	10		70	50
Mean	12.5	12.5	11.7	13.3	40	60	56.7

* Values based on 10 pots of plants exposed each time at each location.
**Severe flea beetle damage to plants, no foliage or flowers remained as infection sites; excluded from average.

Data in Table 2 also show that greater numbers of plates were colonized in mid-late July, when rapeseed crops were flowering, although an appreciable percentage of plates developed colonies of *Sclerotinia* by the end of June in fields N and D.

2) Exposure of flowering plants

Average percentage of plants infected with *Sclerotinia* was higher in the north and west (S, B, W) than in the south and east (L, G, H, V) (Table 3). The number of test plants developing disease was generally higher in late June - early July than later in the season; as the test plants were usually shorter than the field crop by this time, their exposure to airborne spores may have been reduced by the close proximity of the crop. No disease developed on control plants. There was no consistent difference between infection of the cultivars, therefore data are presented as a combined mean for each exposure date.

Total rainfall was low in the V, H and L fields, and high at W, B and S fields, particularly as values given for these areas are for 3 months rather than 4 months. (Table 1)

Discussion

Three possible explanations for the lower spore levels found in south- and east-central Alberta are: 1) number of sclerotia may be intrinsically lower, due to low levels of infection each year; 2) weather conditions may have been less suitable for apothecium production and hence spore production in 1979; 3) spores may not be produced in these areas but are carried south and east by prevailing westerly winds, and a reduction in spore numbers could be expected with increasing distance from inoculum source. Apothecia were only found in field D, but extensive surveys were not carried out in any of the fields.

Infection levels of plants exposed do not seem to be affected by total rainfall at the different locations. Numbers of plants developing infection are identical for G and L, yet weather stations close to field locations show that there was appreciably less rainfall at L than G. However, the highest frequency of disease development on plants in B, W and S fields is consistent with natural infection patterns, as rapeseed with a high level of contamination by sclerotia is produced more frequently in these areas than in the south and east.

Weather data for areas where plates were exposed suggest that field K would have received sufficient moisture for apothecium development, but low percentages of plates exposed developed colonies of *Sclerotinia*. Weather conditions in previous years will have affected production of sclerotia, and inoculum levels may be low due to previous conditions.

It has been reported that heavy rainfall reduces ascospore inoculum as spores are washed down into the soil rather than being released into the air (Krüger, 1974). Duration of conditions suitable for apothecium formation and spore release would be a more critical factor than total rainfall, in determining spore inoculum.

Ascospores of *S. sclerotiorum* were intercepted frequently in the north- and west-central regions, where rapeseed contaminated with high levels of sclerotia is produced in some years. However, sufficient spores were present in the drier regions to cause appreciable disease if climatic conditions suitable for infection had occurred.

Acknowledgements

The cooperation of the farmers whose fields were used during this study is gratefully acknowledged. The authors also wish to thank Mr. C. Gietz for supplying meteorological data, and Mrs. R. Stevens and Miss J. Ford for technical assistance.

Literature cited

1. Krüger, W. 1974. Untersuchungen über die epidemiologie des rapskrebses, verursacht durch *Sclerotinia sclerotiorum* (Lib.) de Bary. Proceedings of the International Rapeseed Conference, Giessen, West Germany, June 4-8: 595-603.
2. Williams, J. R. & Stelfox, D. 1979. Dispersal of ascospores of *Sclerotinia sclerotiorum* in relation to Sclerotinia stem rot of rapeseed. Plant Dis. Rptr. 63: 395-399.

Cracking of Golden Russet apples

J. T. A. Proctor and E. C. Lougheed[1]

Extensive early season fruit cracking of Golden Russet apple was observed in the 1979 season in central and south-western Ontario. Cracking consisted of deep (up to 10 mm), long (up to 40.3 cm) and wide (up to 20 mm) equatorial furrows containing easily detached cork tissue, and occurred mainly on the stem half of the fruit. Similar cracking was observed on Roxbury Russet and a Minnesota selection (Minn. 1734) but not on Pomograte Russet (also called Swayzie Russet). Cracked fruit were suitable only for processing into juice. Fruit cracking was more severe in trees on the more dwarfing rootstocks, which were also younger and bore fewer fruits per cm of trunk circumference. Cracking appears to be related to crop load and fluctuating water supply in the early part of the growing season.

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Durant la période de croissance de 1979, on a relevé dans le centre et le sud-ouest le l'Ontario de nombreux cas de craquelure précoce du fruit qui consistait en l'apparition, surtout sur la moitié pédonculaire du fruit, de fissures équatoriales profondes (jusqu'à 10mm), longues (jusqu'à 40,3 cm) et larges (jusqu'à 20 mm) contenant du tissu subéreux facilement détachable. Des symptômes semblables ont été observés sur la Roxbury Russet et une sélection du Minnesota (Minn. 1734), mais non sur la Pomograte Russet (Swayzie Russet). Les fruits abimés ne se prêtaient qu'à la fabrication de jus. La craquelure était plus grave chez les arbres greffés sur porte-greffes les plus nanisants qui également étaient plus jeunes et qui portaient moins de fruits par centimètre de circonférence du tronc. Cet accident serait associé à la charge de la récolte et à une fluctuation dans l'apport d'eau au début de la période de croissance.

Introduction

Golden Russet is an apple cultivar which is not grown extensively in Canada (Proctor, 1979) but brings high returns in the market place because of its high quality and easy consumer identification. Production of Golden Russet in 1976 was about 454 tonnes (1,000,000 lb) (Proctor, 1979), and the 1976 Ontario Fruit Tree Census for apples showed only 3,784 trees of this cultivar. For estimates of monetary returns a survey of Guelph supermarkets on January 2, 1980 showed that fancy grade Golden Russet apples cost 30 percent more than fancy grade McIntosh. Because of these high returns Golden Russet is being planted on size controlling rootstocks, although relevant data are not separated from "Other Varieties" in the 1976 Ontario Census (Ontario Ministry of Agriculture and Food, 1976).

From August to September 1979 we received numerous enquiries and fruit samples from growers of Golden Russet requesting an explanation for the relatively high incidence of fruit cracking. Because of the extent of the damage we surveyed a number of orchards in central and south-western Ontario, examined fruit in the laboratory, and analyzed weather records.

Materials and methods

Seven orchards in central and south-western Ontario were visited just prior to harvest and records of Golden Russet trees and fruits made (see Table 2 for details). Fruit from 4 trees of one orchard (at Vineland Station) were harvested and stored at 1°C and 90 percent relative humidity until examined. Subsequently fruit weight, length and diameter, crack location, length, width at the widest point, depth at the deepest point, severity rating on a scale of: 1- no cracking, to 5 - severe cracking (Figure 1), and the number of seeds in each fruit were determined. Water absorption of detached apples and related cracking were studied by weighing each apple, submerging it in water for 2 days at room temperature (22°C), reweighing and recording the number and length of any cracks that developed.

The ranked data for cracking were analyzed using Spearman's coefficient of rank correlation (Steel and Torrie 1960, page 409).

Results and discussion

The most prominent features of the cracked fruit were equatorial furrows which in some cases branched and overlapped (Figure 1). Most cracks contained flakes of cork. The class 2 crack (Figure 1) is uncharacteristic because it was longitudinal rather than equatorial but such cracking did occur sometimes. Some of the fruit received from home gardeners had scab lesions in the furrows indicating poor spray control programs.

The cracking and associated cork made the fruit unacceptable for the fresh market. Removal of the damaged area by automated peelers would mean excessive waste. This means that they could be sold only as apples for processing into juice and therefore would bring low returns to the growers. It is difficult to estimate economic loss because considerable variation in cracking occurred (see below and Table 2). Because of the premium prices paid for this cultivar in the fresh market, but not in the juice market, the loss per kg would be greater than for other cultivars such as McIntosh and Delicious.

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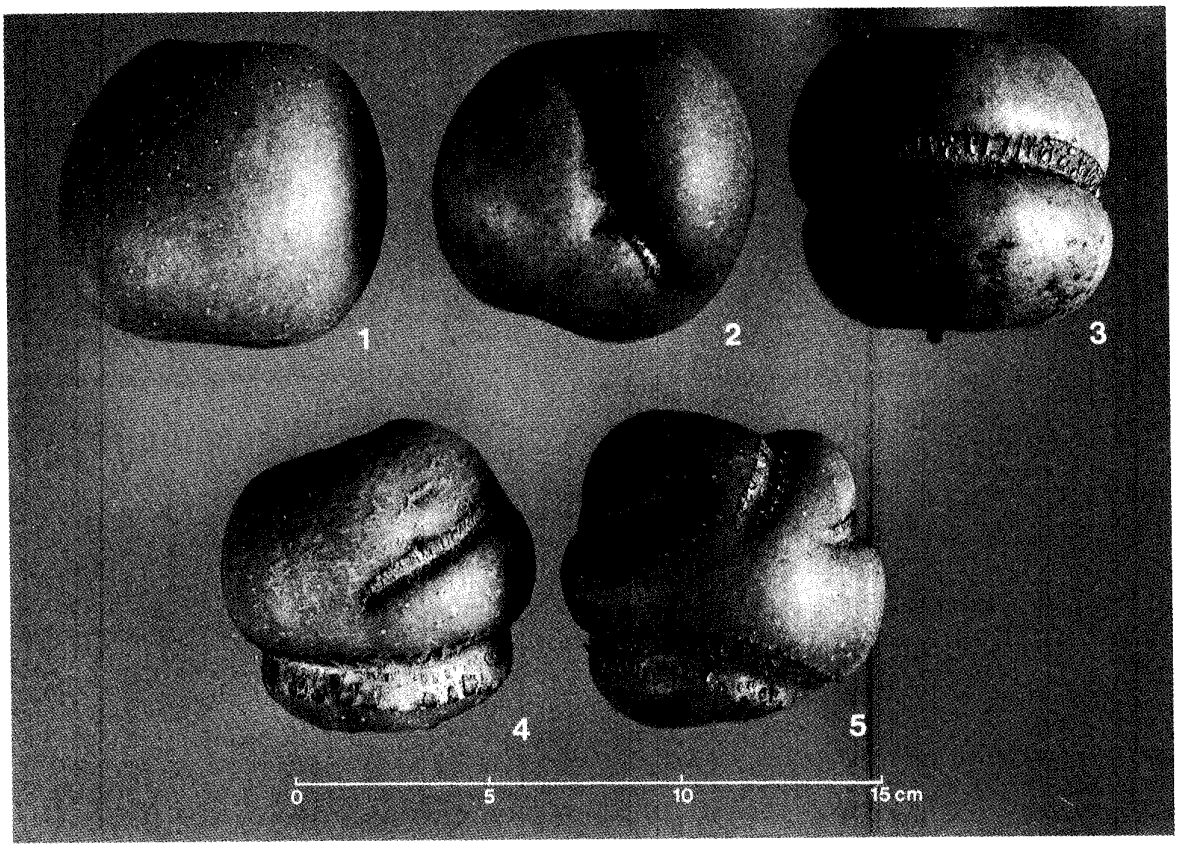

Figure 1. Cracked apples of Golden Russet arranged in a rating scale of 1, no cracking, to 5, extreme cracking.

Sixty percent of the Golden Russet fruit on 4 trees on M.26 rootstocks at Vineland Station had some cracking (Table 1), most falling in class 3 (Figure 1). Spearman's correlation coefficient for ranked data (Steel and Torrie 1960) showed significant relationships between crack rating and all the fruit and crack variables measured (Table 1B). The more damaged the fruit the smaller and lighter it was and the fewer seeds it contained (Table 1). Damage was mainly on the stem half of the fruit although cracks on severely damaged fruit covered the entire apple (see class 5 in Figure 1).

Induction of cracking in harvested apples. Previous work (Verner, 1935) has shown that cracking of detached Stayman Winesap apples could be induced by submerging harvested fruits in water for 2 days. In his work 66 percent of the fruit cracked and the average weight gain and total length of cracks were 2.32 percent and 3.11 cm, respectively. Using a similar technique with Golden Russet revealed that 20 percent of the fruit cracked, and the average length of the crack was 0.50 cm. The cracks were unlike those observed in the orchard, being longitudinal and more like those late season cracks seen on Orenco and illustrated for Stayman Winesap by Halfacre and Barden (1979).

Orchard survey. The survey of orchards (Table 2) showed relatively recent planting of trees on the newer, size controlling rootstock, M.26 (Proctor *et al*, 1974). These trees carried fewer fruit, even when expressed per unit trunk circumference, and had the highest percentage of cracked fruit.

The exact cause of the fruit cracking is not known. It is not thought to be due to a virus (T.R. Davidson, personal communication). The fruit cracking observed on Orenco and described for Stayman Winesap by Halfacre and Barden (1979) and Verner (1935) occurs late in the season just before harvest. In this work cracking of Golden Russet was observed in the orchard in late June and early July, and the wound healing and subsequent cork formation would confirm this. Bell (1938) reported that in early July in Golden Russet a cambium is initiated in the innermost cells of the epidermis and very quickly cells from it differentiate into cork. It could be that at this important stage of fruit growth and development, any stress leading to cracking and death of exterior tissues could result in stimulated activity of this cork cambium and abundant production of cork as seen here.

A possible stress in the 1979 growing season was water supply. There was no precipitation at Vineland Station from June 12 to June 26 (inclusive), an uncommon event. Brown *et al* (1968) predict for St. Catharines, the closest (about 10 km) weather station reported, that such a dry period will

Table 1 (A). Ranking at 274 Golden Russet apples from 4 trees on M. 26 at Vineland Station based on crack rating and the means for several characteristics of the fruits within the various ranks.

Crack Rating*	Percent Of Apples In Rank	Fruit			Crack				
		Weight (g)	Length (mm)	Diameter (mm)	Length (mm)	Width (mm)	Depth (mm)	Position+	Seed No.
1	40	188.90	64.10	74.29	0	0	0	0	5.48
2	8	172.80	62.77	72.23	88.86	8.95	3.41	S	5.23
3	20	153.73	62.38	68.71	176.00	10.78	5.46	S	4.78
4	15	149.37	62.46	68.54	208.88	11.49	6.03	S	4.93
5	17	147.57	60.91	68.36	276.87	12.62	6.24	S&C	4.38

(B). Regression data for crack rating with the variables listed. In all cases the number of apples was 274.

Variable	Spearman's Correlation Coefficient	Level Of Probability Of Obtaining A Greater Correlation Coefficient
Fruit Weight	−0.44	0.0001
Fruit Length	−0.22	0.0002
Fruit Diameter	−0.43	0.0001
Crack Length	0.95	0.0001
Crack Width	0.85	0.0001
Crack Depth	0.87	0.0001
Seed Number	−0.23	0.0001

*See Figure 1 for examples of the crack ratings.
+S = stem half of the fruit. C = calyx half of the fruit.

Table 2. Characteristics of Golden Russet trees and fruits at various locations in central and south-western Ontario at harvest in October 1979*.

Location	Soil Type	Tree			Fruit		
		Age	Root-stock	Trunk Circumference (cm) at 30 cm above soil	Number per tree	Percent cracked fruit	Rating of fruit crack-ing(+) per location
Delhi	Sandy loam	4	M.26	16.1 ± 0.9§	28.5 ± 11.2	11.0	2.0 ± 0.0
Vineland Station	Fine sandy loam	12	M.26	28.2 ± 4.1	68.7 ± 19.6	60.0	3.5 ± 0.3
Burlington	Silt loam	6	M.26	22.5 ± 1.2	172.0 ± 29.2	16.4	2.4 ± 0.2
Rockwood (1 tree)	Gravelly loam	10	M.7	33.0	80.0	20.0	2.5
Belwood 1	Fine sandy loam	15	M.7	50.2 ± 2.7	270.0 ± 14.1	25.2	2.5 ± 0.7
Belwood 2	Fine sandy loam	30	Sdlg	71.4 ± 8.7	1420.0 ± 168.1	3.4	2.0 ± 0.0
Guelph (1 tree)	Sandy loam	40	Sdlg	86.4	152.0	9.2	2.0
Simcoe	Clay	18	Sdlg	98.2 ± 8.7	755.0 ± 161.0	1.4	3.2 ± 0.3

*Data are the means for 4 trees except where noted.
+Rating of cracking - 1, no cracking to 5, extreme cracking. See. Figure 1 for examples of the classes.
§±Standard error.

occur only 5 times in 40 years. While the fine sandy loam at Vineland Station does not vary markedly in its water holding capacity with soils at other locations (Table 2), except possibly the clay at Simcoe, the unusual extended dry period at this time could result in a period of little change in fruit size which followed by the rain reported for 4 consecutive days could have resulted in rapid fruit enlargement and associated cracking. The smaller crop load on trees on M.26 rootstock and the sudden availability of water could also have been a factor inducing cracking.

Cracking of other cultivars. A survey of apple cracking in the Canadian Centennial Museum Orchard at the Horticultural Research Institute of Ontario, Vineland Station, revealed symptoms similar to that on Golden Russet on Roxbury Russet and a Minnesota selection (Minn. 1734) but not on Pomograte Russet (also called Swayzie Russet). For a complete listing of cultivars in this orchard see reference 5. A different type of cracking, illustrated by Halfacre and Barden (1979), and characteristic of Stayman Winesap, was observed on Orenco.

Conclusions

There will be continued production of Golden Russet apples in Canada to meet a continuing and increasing consumer demand resulting in a premium price in the market place. In some years fresh fruit may be suitable only for juice because of cracking presumably due to fluctuating water supply. However, if this is verified as the cause of cracking, more judicious choice of rootstock and soil type, and the use of irrigation may be crucial to, and sound economically, for growers of Golden Russet.

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Literature cited

1. Bell, H.P. 1938. The origin of russeting in the Golden Russet apple. Can. J. Res. 15:560-566.
2. Brown, D.M., G.A. McKay, and L.J. Chapman. 1968. The climate of Southern Ontario. Climatological Studies, Number 5, Meteorological Branch, Department of Transport, Government of Canada, 50 pp.
3. Halfacre, R.G., and J.A. Barden. 1979. Growth pattern, Figures 8-9, p. 241. IN, Horticulture, McGraw-Hill, Toronto.
4. Ontario Ministry of Agriculture and Food. 1976. Fruit tree census, Part III, Apples, 36 pp.
5. Ontario Ministry of Agriculture and Food. 1978. Fruit varieties, a guide for commercial growers. Publication 430, prepared by O.A. Bradt, A. Hutchinson, S.J. Leuty and C.L. Ricketson, 112 pp.
6. Proctor, J.T.A., A. Hutchinson, and W.F. Pierce. 1974. A 10-year trial of seven apple cultivars on Malling 26 rootstock. Can. J. Plant Science 54:661-665.
7. Proctor, J.T.A. 1979. Apple cultivars grown in Canada. Fruit Var. J. 33:12-15.
8. Proctor, J.T.A., and E.C. Lougheed. 1980. A deformity of apples of unknown origin. Can. Plant Dis. Surv. 60:13-16.
9. Steel, R.G.D., and J.H. Torrie. 1960. Principles and procedures of statistics. McGraw-Hill Book Company, Toronto.
10. Verner, L. 1935. A physiological study of cracking in Stayman Winesap apples. J. Agr. Res. 51:191-222.

Effect of dry heat treatments on survival of seed borne Bipolaris sorokiniana and germination of barley seeds[1]

Luc Couture[2] and John C. Sutton[3]

In dry heat treatments of barley seeds naturally infected with *B. sorokiniana*, as the temperature and the time of exposure to treatment increased survival of both fungus and seeds decreased but not at the same rate. A severe reduction in viability of barley was produced under conditions required to eliminate the fungus from the seeds. The best seed survival associated with a treatment giving complete elimination of the fungus was only 42% (60 h at 90°C).

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Dans une expérience portant sur des traitements à la chaleur sèche de semences d'orge contaminées naturellement par le *B. sorokiniana*, la survivance du champignon et celle des graines ont toutes deux diminuées avec l'augmentation de la température et de la durée du traitement, mais à des rythmes différents. Les conditions requises pour l'élimination du champignon des graines ont provoqué une réduction marquée de la viabilité de l'orge. La meilleure survivance de graines reliée à un traitement conduisant à une élimination complète du champignon n'a été que de 42% (60 h à 90°C).

Introduction

Barley (*Hordeum vulgare* L.) seeds harvested from a crop severely affected by spot blotch may contain more than 95% seeds infected by *Bipolaris sorokiniana* (Sacc. in Sorok.) Shoem. (Couture and Sutton, 1978 a). The seed borne fungus serves as primary inoculum causing seedling blight. Eventually this may also provide secondary inoculum produced on above-ground affected parts to cause spot blotch on the foliage (Couture and Sutton, 1978 b).

For many years mercurial fungicides have helped control the seedling blight phase but their use was discontinued. There are reported cases from Great Britain of highly infected seed samples being harvested following the omission of organo-mercurial seed treatment (Hewett, 1975). However, several fungicides currently used for seed treatment of cereals are not as effective as mercury in controlling seedling blight (Wallace and Mills, 1968). Furthermore, because *B. sorokiniana* may persist in barley seeds for 10 years or more (Machacek and Wallace, 1952), ageing is not a practical method for obtaining seeds free from the pathogen.

There is some indication that *B. sorokiniana* may be eliminated from barley by exposure of seeds to a temperature of 100°C for 15 - 30 hours (Atanasoff and Johnson, 1920). For barley grown in the field, Skachkova (1975) reported that heat treatment (not defined) of seeds substantially reduced infection by *B. sorokiniana* in the crop. Heat treatment of seeds has also been used to control other pathogens of barley. The hot water treatment at 54°C for ten minutes has long been recommended for the control of loose smut, *Ustilago nuda* (Jens.) Rostr. (Fushtey, 1969). *Alternaria* sp. disappeared from barley seeds after a seven month period of storage at 41°C (Brown and Robert, 1943).

As an alternative to chemical control, the possibility of using dry heat for controlling seed borne *B. sorokiniana* was examined.

Materials and methods

Seed at 5% moisture content and 89% germination of the 2-row barley cultivar Fergus was used. All the seed was naturally infected by *B. sorokiniana* and the sample was from a field harvested 18 months earlier at Elora, Ontario.

For heat treatments, seed batches contained in 50 ml glass beakers were placed in a thermostatically controlled hot air oven at 70, 80, 90, 100, 110, 120, 130, or 140°C. The samples were removed at time intervals ranging from 15 minutes to 72 hours and allowed to cool.

To determine the percentage of seeds colonized internally by *B. sorokiniana*, 100 seeds from each temperature-time treatment were immersed in 70% ethanol from 20-30 seconds, in 1% sodium hypochlorite for 10 minutes, then rinsed in sterile water and placed in petri dishes on weak carrot agar (Tuite, 1969), supplemented with 50 ppm each of streptomycin-sulphate and chlortetracycline hydrochloride. The seeds were examined at 40X through a stereomicroscope after one week of incubation in the laboratory.

To determine the percentage germination of seeds, 100 seeds from each temperature-time treatment were placed on moist filter paper in petri dishes and incubated for one week in the laboratory. The average of the sample plated on filter paper and of the one previously plated on agar, was used to determine percentage germination.

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Table 1. Effect of dry heat treatments on survival of seed borne *Bipolaris sorokiniana* and germination of barley seeds.

Time of exposure to treatment (h)	Survival (%)* of *Bipolaris sorokiniana* (*Bip.*) and barley seeds (seed)															
	70 C		80 C		90 C		100 C		110 C		120 C		130 C		140 C	
	Bip.	seed	*Bip.*	seed	*Bip.*	seed	*Bip.*	seed	*Bip.*	seed	*Bip.*	seed	*Bip.*	seed	*Bip.*	seed
0.25			100	100	100	99	98	90	96	89	41	59	36	9	0	0
0.50			100	98	95	97	90	84	57	73	17	40	4	2	0	0
0.75											2	5	1	0		
1			100	98	92	96	62	48	17	44	0	1	0	0		
2			100	91	90	90	18	40	4	10	0	0				
3							15	37	2	1	0	0				
4	100	91	100	91	87	90	6	30	0	0						
6							5	14								
8	100	90	99	90	61	90	0	10								
16	99	89	97	88	57	85										
24	98	88	96	86	50	84										
36	98	86	92	83	32	78										
48	98	83	91	82	5	73										
60					0	42										
72					0	34										

*Survival as a percentage of checks: 100% infected seeds and 89% germination.

Results

Survival of both fungus and seeds decreased as the temperature and the time of exposure to treatment increased but not at the same rate (Table 1). The seeds died more rapidly than *B. sorokiniana* at 70, 80, and 130°C and the fungus was differentially more affected at temperatures ranging from 90 to 120°C. Both fungus and seeds were killed in less than 15 minutes at 140°C. The most differential effect beneficial to the barley was at 90°C where, after 8 hours or more of exposure, the viability of the fungus declined much more rapidly than the viability of the seeds.

B. sorokiniana tolerated exposure periods of 48, 4, 0.5, and 0.25 hours respectively at 70 and 80, 90, 100, and 110°C. Exposure periods of 60, 8, and 4 hours were required for complete elimination of the fungus at temperatures of 90, 100, and 110°C. A large difference was observed in the time required, at a given temperature, to kill the fungus in a few seeds as compared to that needed to kill it in all seeds (e.g. at 90°C, a 5% kill was obtained in half an hour but it took 48 hours for a 95% kill). The fungus was killed rapidly (<1 hour) at temperatures of 120°C or more.

It was also observed that other seed borne fungi such as *Alternaria* sp. and *Fusarium* sp. were eliminated at temperatures of 110°C or more while *B. sorokiniana* survived.

Discussion

Exposure periods at various temperatures required to eliminate *B. sorokiniana* from the barley seeds produced a severe reduction in viability of the seeds. The best seed survival associated with a treatment giving complete elimination of the fungus was only 42% (60 h at 90°C). However, this figure might possibly be higher at some temperature-time combinations around 90°C.

Atanasoff and Johnson (1920) reported good control of *B. sorokiniana* at 100°C and an average of about 89 and 73% germinated seeds compared to their check after exposures of 15 and 30 hours respectively at 100°C. Although, this is a much higher survival than the results shown in Table 1, the germination of some of their seed samples was also cut down severely. These differences may be related to differences in moisture content of the seeds, to differences in seed vigor or to the approximate control of temperature indicated by Atanasoff and Johnson (1920).

Grain drying is an increasing practice in Canada. To avoid overheating damage to the grain, the maximum allowable air temperatures to dry barley grain are 45°C for seed or malting, 55°C for commercial use and 80°C for feed (Friesen, 1976). According to results in Table 1, none of these temperatures is likely to reduce the proportion of *B. sorokiniana* infected seeds.

Because the temperature required to kill the pathogen also severely reduces seed viability, it is obvious that dry heat treatment of barley seeds is impracticable for a barley grower. Despite the concomitant high incidence of barley kill, heat treatment may however be useful to obtain *B. sorokiniana* - free barley seeds for experimental purposes. In other respects, on account of our observation that some seed-borne fungi, namely *Alternaria* sp. and *Fusarium* sp., that might interfere with *B. sorokiniana*, can be selectively eliminated by means of heat treatment, the procedure can also be used to facilitate isolation of *B. sorokiniana* from barley seeds as has been done for *Drechslera avenacea* (Curt. ex Cke.) Shoem. in oat seeds (Malone, 1962).

Literature cited

1. Atanasoff, D. and A. G. Johnson. 1920. Treatment of cereal seeds by dry heat. J. Agric. Res. 18(7):379-390.
2. Brown, E. and A. L. Robert. 1943. *Alternaria* sp. on grain kernels killed by high temperature storage. Phytopathology 33(4):333-335.
3. Couture, L. and J. C. Sutton. 1978 a. Efficacies of fungicides in controlling spot blotch of barley. Can. J. Plant Sci. 58(2):311-317.
4. Couture, L. and J. C. Sutton. 1978 b. Relation of weather variables and host factors to incidence of airborne spores of *Bipolaris sorokiniana*. Can. J. Bot. 56(17):2162-2170.
5. Friesen, O. H. 1976. Grain drying. Canada Department of Agriculture, Publ. 1497. 44 pp.
6. Fushtey, S. G. 1969. Smut diseases of grain crops. Ontario Ministry of Agriculture and Food, Publ. 524. 14 pp.
7. Hewett, P. D. 1975. A health survey of barley. Plant Pathol. 24(4):229-232.
8. Machacek, J. E. and H. A. H. Wallace. 1952. Longevity of some common fungi in cereal seed. Can. J. Bot. 30(2):164-169.
9. Malone, J. P. 1962. Studies on seed health. IV. The application of heat to seed oats as an aid in the detection of *Pyrenophora avenae* by the Ulster method. Proc. Int. Seed Test. Ass. 27(3):855-861.
10. Skachkova, L. V. 1975. [The development of root rots of barley in the field in the forest belt system] in Russian. Sb. Nauch. Rabot. NII S. Kh. Tsentr.-Chernozem. Polosy 9(1):162-166.
11. Tuite, J. 1969. Plant pathological methods. Burgess Publishing Company, Minneapolis. 239 pp.
12. Wallace, H. A. H. and J. T. Mills. 1968. Evaluation of seed treatment chemicals for the control of seedling blight of barley. Can. Plant Dis. Surv. 48(4):141-149.

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