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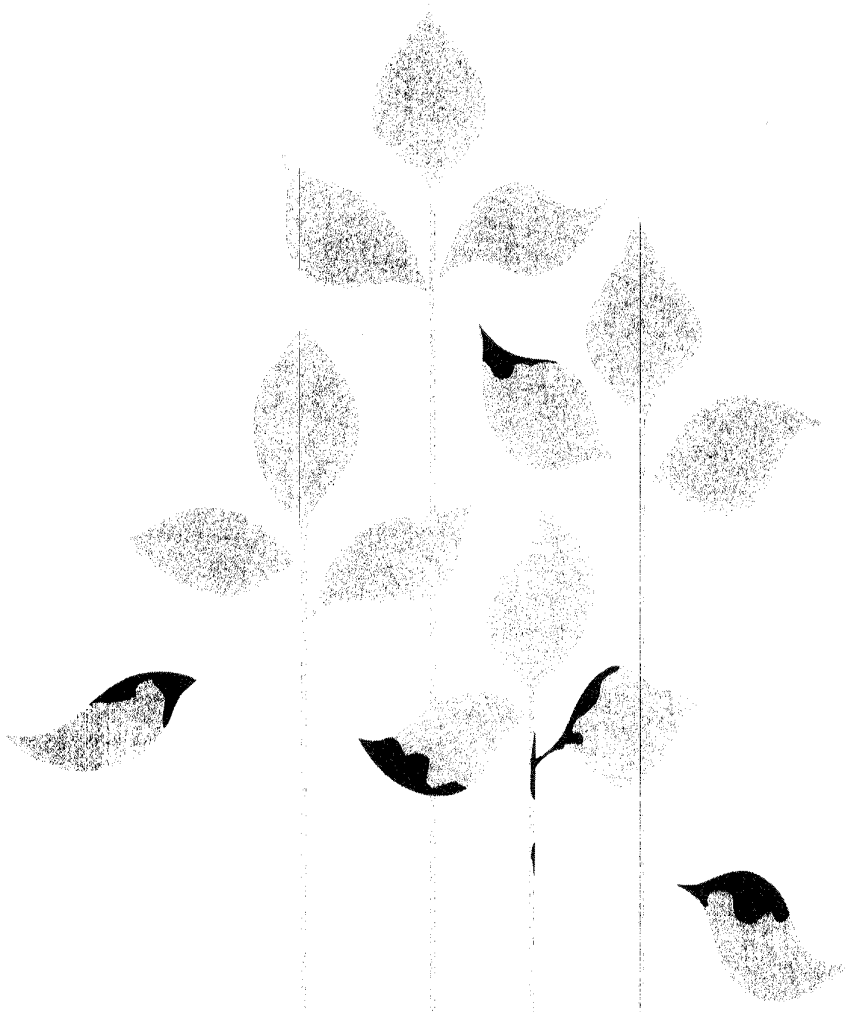

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Contents

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

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

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Olpidium brassicae, tobacco necrosis virus, and Pythium spp. in relation to rusty root of carrots in Ontario and Quebec

D.J.S. Barr[1] *and W.G. Kemp*[2]

Olpidum brassicae, Pythium sulcatum, and *Pythium irregulare* were frequently isolated from roots of carrots grown in soils from Ontario and Quebec carrot fields regardless of the presence of carrot rusty root. Although there were physiological strains of these fungi, there were no apparent differences in morphology or pathogenicity of isolates collected from the Holland-Bradford and Keswick marshes in Ontario, where rusty root disease is a problem, and isolates from non-problem soils. Tobacco necrosis virus was frequently found in problem soils in association with diseased carrots but it was only detected once in a non-problem soil. It is suggested that in addition to *Pythium* spp., tobacco necrosis virus and its vector *Olpidium brassicae* are involved in rusty root etiology.

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Olpidium brassicae, Pythium sulcatum et *Pythium irregulare* ont été fréquemment isolés de carottes cultivées dans des échantillons de sol provenant de champs de carottes de l'Ontario et du Québec frappés ou non par la rousselure. Bien que l'on ait identifié les souches des champignons on n'a relevé aucune différence apparente entre la morphologie et la pathogénicité des micro-organismes isolés des échantillons de terre noire de Holland-Bradford et de Keswick (Ontario), où la rousselure pose un problème, et celles des micro-organismes isolés de sol libres de la maladie. Le virus de la nécrose du tabac a été fréquemment isolé des carottes malades provenant de sols problèmes alors qu'on ne l'a trouvé qu'une seule fois dans les carottes cultivées ailleurs. Nous pensons que le virus de la nécrose du tabac et son vecteur, *Olpidium brassicae*, avec *Pythium* sp. jouent un rôle dans l'étiologie de la rousselure.

Rusty root, an economically important disease of carrots (*Daucus carota* L. var. *sativa* DC) in Ontario, was first reported in the Holland-Bradford Marsh in 1962 (Fushtey and Filman 1968). Although the disease is widespread there and in the nearby Keswick Marsh, it has not been reported in other carrot-growing areas in Ontario and Quebec.

Sutton (1973, 1975) concluded from his work on this disease that several species of *Pythium* cause rusty-root in Ontario carrots and stated that the disease is similar to the lateral-root diseases caused by *Pythium* spp. in muck-grown carrots in British Columbia (McElroy et al. 1971), Wisconsin (Mildenhall et al. 1971), and Florida (Pratt and Mitchell 1973).

The possible involvement of other biotic agents, particularly non-filamentous fungi and soil-borne viruses, in soils associated with the rusty-root disease syndrome, appears to have been overlooked or ignored. One of us (W.G.K.) has frequently found tobacco necrosis virus (TNV) and its fungus vector *Olpidium brassicae* (Wor.) Dand. associated with carrot roots with typical rusty root symptoms in the Holland-Bradford Marsh. Subsequently, in 1973 a survey of randomly selected carrot fields was carried out in the major muck soil areas in southern and southwestern Ontario and in the Napierville-Sherrington-Ste. Clotilde area of Quebec to determine possible relationships between the presence of *Olpidium brassicae*, TNV, *Pythium* spp., and the rusty root disease. In addition, because of the possibility that a specific carrot strain of *O. brassicae* was introduced into the marsh areas, a survey of wild carrots was made at the same time; wild carrots (*Daucus carota* L.) are abundant in eastern, southern, and southwestern Ontario as well as in parts of Quebec, but in marshes they occur only along roadsides.

Materials and methods

Composite samples of soil from carrot fields were assayed for *Olpidium brassicae*, TNV, and *Pythium* spp. by a bait-plant technique. Carrot (Nantes type) seed was sown in about 2-3 cm of each test soil over 4-5 cm of sterilized white sand (24 mesh) in 7.5 cm (3 inch) diam clay pots. After 3-4 weeks growth at 20°C, roots that penetrated the sand were examined for necrosis and cultured for fungi. Sap from small fragments of carrot root grown in each soil was also indexed for TNV by mechanical inoculation to leaves of *Gomphrena globosum* L. and/or *Chenopodium amaranticolor* Coste & Reyn.

O. brassicae was isolated from the pot-grown carrots by sprinkling fragments of washed, air-dried roots of the

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carrot bait plants around roots of carrot seedlings. The inoculated seedlings were then potted in sterile sand, watered daily with Hoaglands solution (1/3 concn), and after 3-4 weeks at 20°C the roots were examined for the fungus. Dried roots from the second or third carrot generation were used as inoculum in *O. brassicae* host range tests. Several isolates of *O. brassicae* from lettuce (*Lactuca sativa* L. cv. Great Lakes) and one from dandelion (*Taraxacum officinale* Weber) maintained in dried roots of these plants were also tested.

Pythium was isolated by placing washed roots from test carrots grown in pots onto Emerson's YpSs agar (Difco) containing neomycin sulfate (200 ppm). *Pythium* mycelium growing from the roots was transferred to 2.4% V-8 juice agar (2% agar), and later to autoclaved hemp seed in water for critical morphological examination.

The origin of soil samples from Ontario is shown in Fig. 1. The soil type, whether organic (muck) or sand, is also recorded. In addition, the location of four collections of

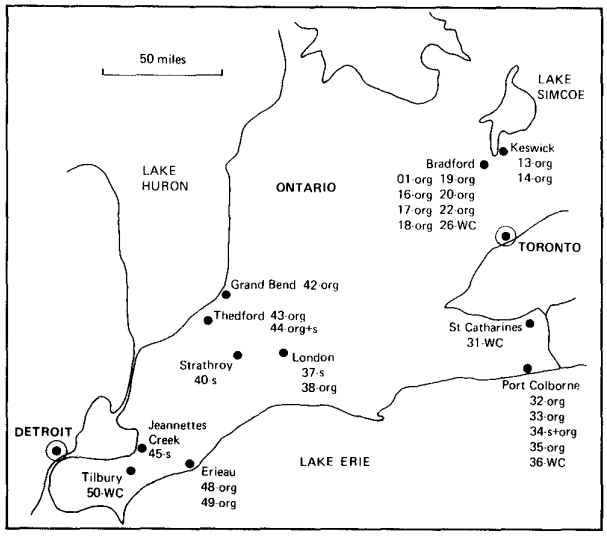

Figure 1. Collections of carrot made in southern and southwestern Ontario in 1973; org - organic (muck) soil, s - sandy soil from carrot producing areas, WC - wild carrot.

wild carrot is shown. Soil collections from Quebec (Nos. 52-62) originated from the carrot-growing region approximately 25 miles south of Montreal; all Quebec soils examined were organic.

Results

Presence of Olpidium brassicae, TNV and Pythium spp. in soils

Both root necrosis and severe infection by *O. brassicae* occurred in carrots grown in most soil samples from fields in which carrots had been grown frequently in recent years or less frequently over many years. These soils were collected from both the Holland-Bradford and Keswick marshes where rusty root is a problem, and from elsewhere in Ontario and Quebec where the disease has not been reported (Table 1 and Fig. 1).

In 1973, TNV was detected in soils only from farms in the Holland-Bradford Marsh where rusty root occurred. However, in 1974 additional checks were made of some of the 1973 locations and the virus was detected in a soil from the London Marsh.

Pythium spp. were found in all soils in which carrot root necrosis occurred (Table 1). Both *P. sulcatum* Pratt & Mitchell and *P. irregulare* Buisman were recovered from plants with severe root necrosis; the former species was isolated more frequently. *P. sulcatum* was also isolated from apparently healthy roots of test plants grown in some soil samples; these isolates failed to induce necrosis in carrot. Other *Pythium* spp. were associated with mild or moderate necrosis.

O. brassicae was abundant in test plants grown in some of the soil samples and often infected about 90% of the epidermal cells (Fig. 2). In spite of the abundance of this fungus in roots there was often no necrosis; when necrosis occurrred *Pythium* spp. were always present. Repotting of these *Olpidium/Pythium* infected plants usually facilitated the spread of *Pythium* and root necrosis to new roots while resulting in a marked decline of detectable *O. brassicae* in the roots of these plants.

Survey of wild carrot for Olpidium brassicae

O. brassicae was found on wild carrots collected from widely distributed areas including some in eastern Ontario remote from commercial carrot production. It was also present on wild carrots by a roadside next to a newly opened marsh area near Port Colborne, Ontario, (No. 36) but not in cultivated carrots grown in the laboratory in muck soil from an adjacent carrot field (No. 35). In four samples (Fig. 1) wild carrots were severely infected and the fungus was easily maintained on wild or cultivated carrots in the laboratory; however, in other cases, the chytrid died-out after carrots were grown in the laboratory under conditions favorable for fungus multiplication.

Morphology of Olpidium brassicae isolates

O. brassicae from wild and cultivated carrots grown in a number of Ontario and Quebec soil samples were compared morphologically for varietal differences; none were observed. Sporangium size varied with host cell size and the number of infective units per cell but was not characteristic of a specific isolate; the smallest sporangia were spherical and 10 μm diam whereas the largest filled the host cell and measured 35 x 80 to 12 x 110 μm. Discharge tube length varied among isolates; they were usually short, up to 5-6 μm in length but in some isolates they were longer, ranging to 45 μm. Resting spores (Fig. 3) were more uniform than sporangia in size; they were always stellate, generally spherical, 12-18 μm diam, but occasionally as small as 9 μm or as large as 18 x 28 μm. Some isolates, however, produced few resting spores in carrot. Zoospores were spherical to subspherical and varied in size from 3.5 to 4.5 (-5.5) μm, with flagellum lengths, including the fine whiplash end, of 16 to 22 μm. Critical examination of flagellum length showed that within many isolates the

Table 1. Presence of root necrosis, *Olpidium brassicae*, and *Pythium* spp. in carrots grown in samples of soil from carrot fields in Ontario and Quebec

Location* and No.	Root necrosis	*Olpidium brassicae*	*Pythium* species isolated
Holland-Bradford & Keswick marshes, Ontario			
13†	severe	abundant	*P. sulcatum* & *P. irregulare*
14	none	trace	none
16†	severe	abundant	*P. sulcatum*
17†	severe	abundant	*P. sulcatum* & *P. irregulare*
18†	severe	abundant	*P. sulcatum*
19	moderate	abundant	*P.* sp.
20†	mild	abundant	*P. sulcatum*
22†	moderate	abundant	*P. sulcatum*
Other Ontario locations			
32	severe	light	*P. sulcatum* & *P. sylvaticum*
33	moderate	trace	*P.* sp.
34	severe	abundant	*P. irregulare*
35	none	none	none§
37	none	none	none
38	severe	abundant	*P. sulcatum*
40	moderate	trace	*P.* sp.
42	trace	none	*P.* sp.§
43	severe	abundant	*P. sulcatum*
44	moderate	trace	*P. sulcatum*
45	trace	none	*P. sulcatum* & *P.* sp.
48	moderate	abundant	*P. sulcatum*
49	severe	trace	*P. sulcatum*
Quebec locations			
52	severe	abundant	*P. sulcatum* & *P. irregulare*
53	none	trace	*P. sulcatum*
54	trace	none	*P. sulcatum*
56	trace	abundant	*P. irregulare*§
57	moderate	abundant	*P. sulcatum*
60	trace	abundant	*P. irregulare*
61	severe	abundant	*P. irregulare* & *P. ultimum*
62	severe	abundant	*P. sulcatum* & *P. irregulare*

* See Figure 1 for location of field samples.

† Samples from fields with history of rusty root disease.

§ *P. sulcatum* was subsequently isolated from other hosts grown in the same soil sample: 35-celery, 42-celery, and 56-dill.

length varied by only 1.5 to 2.0 μm among individual zoospores and the length remained constant when the isolate was grown on different hosts. In conclusion, the morphological differences among isolates were slight, being confined to discharge tube length and minute differences in flagellum length. More important, there were no discernible morphological differences between isolates from the Holland-Bradford and Keswick marsh soils and those from other organic or mineral soils in Ontario and Quebec. Nor could differences be found in isolates from wild or cultivated carrots.

Host range of Olpidium brassicae

O. brassicae isolates were grouped into four types based on their growth habit on carrot and lettuce roots (Table 2). Type 1 isolates all grew well on carrot and wild carrot and some, in addition, on dill, but none grew on lettuce or other nonumbelliferous hosts. Type 2 isolates all grew well on carrot and lettuce. Type 3 isolates grew well on lettuce but poorly on carrot and usually died out after a few weeks on this host. Type 4 isolates grew on lettuce but not on carrot.

When 18 hosts were tested it was soon apparent that variation within types was considerable (Table 2). This variation was likely due, at least in part, to mixtures of *O. brassicae* strains within the original isolates. Repeated transfers through lettuce or carrot indicated that many

Table 2. Type and host range of isolates of *Olpidium brassicae* from plants grown in soils from different locations in Ontario and Quebec

Location* and No.	Original host	Isolate type†	Notes on host range
Holland-Bradford & Keswick area, Ontario			
01§	carrot	1	Grew only on carrot and wild carrot
01§	lettuce	4	Grew on parsley & celery but not carrot
13§	carrot	2	Wide host range
14	carrot	3	Grew on lettuce
16§	carrot	2	Grew only on carrot, dill, lettuce, and spinach among 15 hosts tested
17§	carrot	2	Wide host range
18§	carrot	2	Wide host range
19	carrot	2	**
20§	carrot	2	**
22§	carrot	2	**
26	wild carrot	1	Grew only on carrot and wild carrot
Other Ontario locations			
31	wild carrot	1	Grew only on carrot and wild carrot
32	carrot	3	Grew on lettuce
33	dandelion	4	Grew on dill, parsley, celery, and lettuce
34	carrot	2	**
35	lettuce	4	Not found on carrot in this soil
36	wild carrot	1	**
37	lettuce	4	Not found on carrot in this soil
38	lettuce	2	Wide host range including carrot
40	carrot	2	Grew only on carrot, lettuce, *Plantago*, and *Setaria* among 15 hosts tested
42	—	—	Not found on carrot or lettuce in this soil
43	lettuce	2	Wide host range including carrot
44	carrot	3	Grew on lettuce
45	—	—	Not found on carrot or lettuce in this soil
48	carrot	2	**
49	carrot	3	Grew on lettuce
50	wild carrot	1	Only grew on carrot and dill
Quebec locations			
52	carrot	1	No apparent growth on lettuce**
52	lettuce	3	Poor growth on carrot
53	carrot	3	Grew on lettuce
56	carrot	2	Wide host range
57	carrot	2	Wide host range
60	carrot	2	**
61	carrot	1	**
62	carrot	1	**

* See Figure 1 for location of fields samples.

† Type 1 - good growth on carrot but none on lettuce; type 2 - good growth on carrot and lettuce; type 3 - poor growth on carrot but good growth on lettuce; type 4 - non carrot isolates which did not grow on carrot.

§ Samples from fields with history of rusty root disease.

** Presence of *Pythium* spp. with inoculum prevented thorough testing.

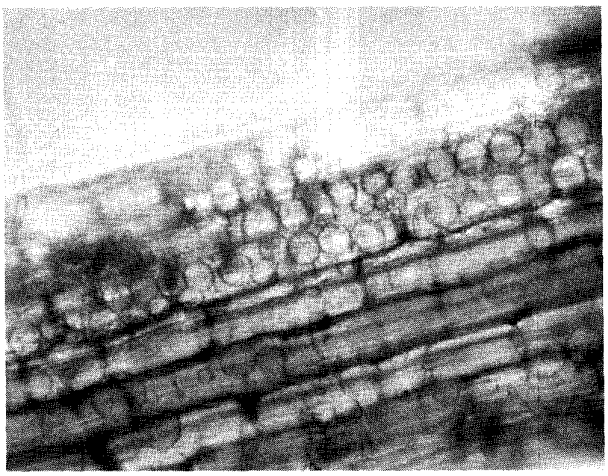

Figure 2. Carrot root containing numerous globular sporangia of *Olpidium brassicae*. ,X200.

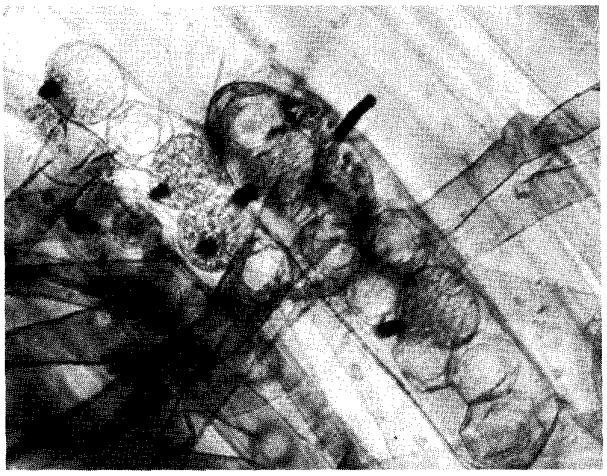

Figure 3. *Olpidium brassicae* in carrot root cells: stellate resting spores (arrows) and globular sporangia with darkly stained discharge tubes., X470.

isolates classed initially as type 2 were mixtures of types 2 and 3; ability to infect carrot being irretrievably lost after successive transfers through lettuce. Furthermore, at least one soil (No. 01) had types 1 and 4. Unfortunately, largely owing to the presence of virulent strains of *Pythium* in the inoculum, it was not possible to test all isolates on all 18 hosts; these results are, therefore, summarized as the number of isolates which grew on a host over the number of isolates tested on a host. Only isolates from carrot are included. Trace infections were considered negative. Results were: cabbage (*Brassicae oleracea* L. cv. Viking extra early strain) 0/21; celery (*Apium graveolens* L. var. *dulca* D.C. cv. Utah Green) 7/16; coriander (*Coriandrum sativum* L.) 6/11; dill (*Anethum graveolens* L. cv. Long Island Mammoth) 10/13; lettuce (*Lactuca sativa* L. cv. Great Lakes) 17/21; oats (*Avena sativa* cv. Clintland 60) 0/12; onion (*Allium cepa* L. cv. Early Yellow Globe) 2/13; parsley (*Petroselinum crispum* Nym. cv. Moss Curled) 5/13; spinach (*Spinacia oleracea* L. cv. America) 8/16; tomato (*Lycopersicum esculentum* Mill. cv. Gardener) 5/15; wheat (*Triticum aestivum* L. cv. Kent) 4/12. Some common weeds were also tested: lamb's quarters (*Chenopodium album* L.) 7/14; broadleaf plantain (*Plantago major* L.) 9/17; purslane (*Portulaca oleracea* L.) 3/12; groundsel (*Senecio vulgaris* L.) 1/8; yellow foxtail grass (*Setaria glauca* (L.) Beauv.) 10/17; dandelion (*Taraxacum officinale* Weber) 1/11.

Tests on commercial and wild carrots indicated there were no differences in susceptibility. Additional tests were done on cv. Spartan Fancy and a new carrot cultivar under trial, both of which have shown some resistance to rusty root in field tests; however, these cultivars were not resistant to *O. brassicae*.

Circumstantial evidence for strains of Pythium sulcatum

During tests with *O. brassicae* it was observed that differences occurred in host range and virulence of *P. sulcatum* frequently associated with carrot root necrosis in some carrot soils. For example *P. sulcatum* on carrot roots originating from a number of different soils generally did not cause necrosis of celery roots. However *P. sulcatum* in a Grandbend soil (No. 42) in which carrots were grown in 1973 and previously in 1964 did not infect carrot roots but was associated with severe root necrosis and stunting of celery plants in laboratory tests.

Pythium irregulare was also isolated from necrotic as well as from healthy carrot roots on a number of occasions and is probably an important cause of root necrosis in pot-grown carrots. However *P. sulcatum* may also have been present in these necrotic roots and not isolated because of its slower growth rate. *P. sulcatum* often occurs in roots without producing oospores and, therefore, can be easily overlooked in direct microscopic examination of roots.

Discussion

The presence of *O. brassicae* in carrot roots and muck soils is not correlated with the rusty root disease syndrome. The fungus occurs in both rusty-root problem and non-problem soils throughout Ontario and Quebec. *O. brassicae* is certainly a primary invader of carrot roots; it is an obligate parasite but does not induce necrosis or multiply in necrotic roots. There appears to be nothing unique about *O. brassicae* isolates from the various problem soils as compared with those from non-problem areas. Some isolates grow abundantly on carrot and other plants, and some are confined to carrot; however both types occur in commercial carrot growing areas where the disease has not been found and also on wild carrot. Whether the populations with affinity for carrot originate in the marsh areas or from a natural reservoir of "carrot strains" from wild carrot, has not been resolved in this study.

With one exception, TNV was detected in carrot roots only in rusty root problem soils. If the virus does occur in non-problem soils it was present in the samples examined at concentrations below the levels detectable by the bioassay method used. Whenever TNV was detected in carrot roots, *Olpidium* sporangia and/or resting spores were easily found.

There is no doubt from this study and others (McElroy et al. 1971, Mildenhall et al. 1971, and Sutton 1975) that *Pythium* is associated with root necrosis. However *Pythium* root necrosis in our pot experiments should not necessarily be equated with rusty root symptoms in the field. One significant question needs answering: if *Pythium* spp. are solely responsible for rusty root then why doesn't the disease occur in other carrot growing areas outside of the Holland-Bradford and Keswick marshes? Virulent strains of *Pythium* spp., particularly *P. sulcatum* implicated by others with rusty-root-like diseases of carrots, occur in many carrot soils from fields in which the disease has not been found. The theory that *Pythium* spp. alone are responsible for rusty root disease does not seem to be conclusive from our observations; the possibility cannot be overlooked that *O. brassicae*, TNV, or *O. brassicae* plus TNV predisposes carrots to rusty root and are implicated with *Pythium*. Nor, however, can certain unique conditions, such as soil type or irrigation system, that might contribute to the disease in the Holland-Bradford and Keswick marshes, be ignored.

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Observations on silvertop of grasses in Alberta

B. Berkenkamp and J. Meeres[1]

Silvertop, a blasting of the heads of grasses, was examined in Alberta. Mites, thrips, and *Fusarium poae* were regularly but not consistently associated with the disease, and a causal agent could not be specified. *Poa pratensis* and *Festuca rubra* were very susceptible. *Bromus inermus, Elymus junceus, E. angustus* and *E. sibiricus* are less susceptible. Varietal differences were found in *Agropyron cristatum* and *A. desertorum*.

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La coulure des graminées a fait l'objet d'une étude en Alberta. Bien que les tétranyques, les thrips et *Fusarium poae* aient été fréquemment, mais pas toujours, associés à cette maladie on n'a pas pu déterminer l'agent causal spécifique. *Poa pratensis* et *Festuca rubra* sont très sensibles à la maladie. *Bromus inermis, Elymus junceus, E. angustus,* et *E. sibiricus* l'y sont moins et on a observé des différences variétales de sensibilité chez *Agropyron cristatum* et *A. desertorum*.

Silvertop, a disease of grasses, is characterized by a bleached, dead seed head and stem above the last node and a healthy flag leaf, sheath, and lower stem. These symptoms appear soon after head emergence, usually before the seed head has fully expanded. The panicle can be pulled out of the flag leaf sheath and the lower end is usually shrunken, darkened and necrotic. This disease has been known since 1875 in the United States and was first attributed to injury by mites. Hardison (3) reviewed the early literature and discussed mites, thrips, and *Fusarium poae* (Peck) Wollenweber as causal agents singly and in combinations. He could not find evidence, in an examination of the disease in Oregon, that mites or *F. poae* were the primary cause of the disease and suggested that other insects may be responsible. Surveys in Alberta have shown (1) that the average incidence of whitehead on brome was 0.44% over the years 1970-1973.

Materials and methods

In 1973 at Lacombe, a 3-year-old test of crested wheatgrass (*Agropyron cristatum* (L.) Gaertn. and *A. desertorum* (Fisch.) Schutt.) cultivars was examined for silvertop in late June and the number of affected heads was counted in a total of 100 heads selected at random from each plot. The four-variety, six-replicate test was analyzed as a randomized block and the shortest significant range calculated. Necrotic basal portions of affected panicles were plated on potato dextrose agar by pulling the panicles out of the flag leaf sheath and cutting off the basal portions aseptically or after surface sterilizing them with a 10% aqueous solution of commercial bleach. The plates were incubated and examined for *Fusarium* spp.

In 1974, culms of various grass species showing silvertop were collected from experimental plots along with comparable culms of healthy plants. These were dissected in the laboratory with the aid of a low-power microscope. The number of plants containing mites and thrips was recorded.

Results and discussion

In the varietal test, highly significant differences in number of silvertop heads were found among varieties of crested wheatgrass. Fairway with 2.3% silvertop and Parkway with 2.5% were not significantly different at the 1% level; however Nordan with 13.7% and Summit with 20.0% were significantly different from each other and from Fairway and Parkway. Differences in silvertop and in insect infestation (Table 1) could not be accounted for by differences in the morphology of the flag leaf sheath or panicle. This demonstration of varietal differences does not assist in defining the cause of the disease, nor do the differences reported between species (5,6). Our observations in Alberta indicate that Kentucky bluegrass (*Poa pratensis* L.) and creeping red fescue (*Festuca rubra* L.) are very susceptible; brome (*Bromus inermis* Leyss.), crested wheatgrass, Russian wild rye (*Elymus junceus* Fisch.), Altai wild rye (*E. angustus* Trin.) and Siberian wild rye (*E. sibiricus* L.) are less susceptible. Timothy (*Phleum pratense* L.) has been reported to be affected by silvertop (6), but the disease has not been observed on this host here.

The frequency with which mites and thrips were found in the flag leaf sheaths of grasses collected from experimental plots at Lacombe is shown in Table 1. A slightly greater percentage of affected than healthy plants were free of infestation. It is possible that on the death of the heads, mites and thrips would leave the sheath; thrips, however, are much more mobile than mites. In several cases, small holes that appeared to

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Table 1. Occurrence of mites and thrips in grasses in 1974

Host	Silvertop affected				Healthy (symptomless)			
	Number of heads examined	% with thrips	% with mites	% not infested	Number of heads examined	% with thrips	% with mites	% not infested
Crested wheatgrass	18	16.7	5.5	77.8	9	33.3	0	66.6
Brome	30	3.3	6.6	90.0	21	4.8	0	95.2
Russian wild rye	33	3.0	0	97.0	3	0	33.3	66.6
Siberian wild rye	6	16.7	0	83.3	3	0	0	100.0
Kentucky bluegrass	85	38.8	45.9	32.9	18	83.3	72.2	5.6
Total or average	172	22.5	24.3	67.6	54	35.2	25.9	59.2

have been made by insects were found in affected sheaths.

Fusarium poae has been generally accepted as the cause of silvertop in Canada and the United States (5); however our isolations, made by placing the lower end of affected panicles on potato dextrose agar, did not support this conclusion. Less than half of the stems yielded *Fusarium* spp. and some were sterile. This is in close agreement with the findings in Oregon (3).

Losses to forage yield from silvertop are probably negligible, but seed production has been reduced up to 95% in the United States (3) and 12-14% in Canada (2). Insecticides, as well as spring and fall burning of dormant grasses (4), have been shown to reduce losses to silvertop.

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The reaction of Thatcher wheat to Canadian races of stem rust[1]

G. J. Green and P. L. Dyck

Triticum aestivum 'Thatcher' was susceptible in both adult and seedling stages to six races of *Puccinia graminis* f. sp. *tritici*, intermediate or moderately susceptible in both stages to seven races, and resistant in both stages to two older races. The genetics of Thatcher's stem rust resistance is poorly understood and its resistance to the two races is not due to genes it is known to carry. Thatcher does not appear to have "adult plant resistance" to the races studied.

Can. Plant Dis. Surv. 55:85–86. 1975

Les plants adultes et les plantules de *Triticum aestivum* Thatcher se sont révélés sensibles à six races de *Puccinia graminis* f. sp. *tritici*. Les deux stades ont montré une sensibilité intermédiaire ou moyenne à sept autres races et ont résisté à deux races plus anciennes. On ne comprend pas encore très bien la génétique de la résistance de Thatcher à la rouille de la tige, sa résistance aux deux races n'étant pas imputable aux gènes qu'on lui connaît. A l'état adulte Thatcher ne semble pas montrer de résistance aux races étudiées.

Triticum aestivum L. 'Thatcher' was the most commonly grown cultivar in western Canada from about 1935, when it was introduced, to 1967. It has been replaced by other varieties because it is susceptible to race 15B of stem rust and to most races of leaf rust. Although its commercial importance has declined it continues to play an important part in wheat production. Stem rust resistant backcross derivatives of Thatcher now predominate in western Canada and Thatcher derivatives are important parents in breeding programs in many parts of the world.

Stem rust resistance inherited from Thatcher has been an important factor in the long-lived stem rust resistance of western Canadian varieties such as Manitou and Neepawa. The stem rust resistance of Thatcher has been assessed periodically but these assessments are not normally published, and we are poorly informed on the reaction of this variety to the races that have occurred in its long history of commercial use. The results recorded here are from an assessment performed with stem rust races found in Canada up to 1972 to obtain data for decisions on how the resistance of Thatcher could be best used in breeding programs.

Materials and Methods

Seedlings were inoculated by dusting urediospores of *Puccinia graminis* f. sp. *tritici* onto them. Adult plants were inoculated by applying urediospores to the culms with the fingers. All plants were incubated in polyethylene chambers where they were sprayed periodically with water. After incubation they were placed on greenhouse benches. The purity of the rust cultures used was confirmed by tests with the differential hosts.

Seedling infection types were recorded according to the method of Stakman et al. (8). Adult plant reactions were rated by recording the infection types on each leaf sheath and calculating an average "reaction index" for each variety-race combination by using an index of 0 to 16 for increasing levels of susceptibility (3). In this index infection type 0 has a value of 1, infection type 1 is 4, infection type 2 is 7, infection type 3 is 13, and infection type 4 is 16.

The cultures selected for the investigation have been described (2). Some are worthy of comment. Race C10(15B-1) represents the race 15B that suddenly became prevalent in 1950 and seriously damaged the varieties of that time, including Thatcher. Race C17(56) predominated from 1932 to 1949 and from 1957 to 1963 inclusive. It has been one of the most important races in North America although it has occurred rarely since 1967. Race C18(15B-1LX) predominated from 1964 to 1969 and race C33(15B-1L) has predominated since 1970. Race C35(32-113) seriously damaged the variety Pitic 62 in 1971. Most races were selected because they appeared to threaten Thatcher and its derivatives. Others were selected for comparison with possibly more virulent races.

Results

The seedling infection types and the adult plant reactions (Fig. 1) agreed reasonably well with the exception of those for races C33 and C51. Thatcher was most susceptible to race C10(15B-1). It was also susceptible or moderately susceptible at both growth stages to races C18(15B-1LX), C34(32), C37(15), C40(32-113), and C42(15). Of these races, only C18(15B-1LX) is sufficiently prevalent to be important. The other races have originated since 1968 and it is clear that many of the races found in Canada in recent years are virulent on Thatcher.

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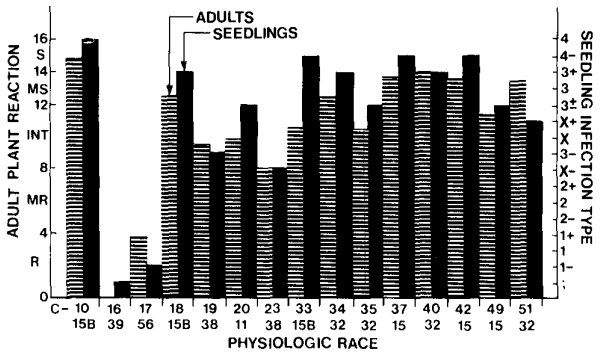

Figure 1. Seedling infection types and adult plant reaction of Thatcher wheat to 15 races of stem rust. Formula numbers are in the line starting C—. The "standard" race numbers are below them.

Thatcher was susceptible in the seedling stage but intermediate in the adult plant stage to race C33(15B-1L), which has predominated in Canada since 1970, and moderately susceptible in the adult stage and intermediate in the seedling stage to the rare race C51(32). It was intermediate in both seedling and adult plant stages to races C19(38), C20(11), C23(38), C35(32-113), and C49(15). Most of these races were first found prior to 1964 when the formula method of race identification (1) came into regular use.

Thatcher was resistant only to the old races C16(39) and C17(56).

Discussion

Despite the importance of Thatcher wheat as a commercial variety and as a parent in breeding programs its genotype for stem rust resistance is poorly understood. It carries the identified genes *Sr5, Sr16,* and possibly *Sr12*. *Sr5* is a well-known and well-documented gene. It usually produces an immune reaction (infection type 0) with avirulent races. Gene *Sr16* has been isolated in a susceptible background but it conditions a low level of resistance and has not shown potential practical importance in tests at Winnipeg. The status of gene *Sr12* is less secure. A gene originally thought to be complementary to gene *Sr11* was numbered *Sr12* (4) but later was found to be nonexistent (5). The number was then used for an hypothesized gene on Thatcher chromosome 3B (7). A later investigation of this chromosome failed to reveal a gene for stem rust resistance (6).

None of these genes confer resistance to the wide assortment of races that are avirulent on Thatcher. The variety may have adult plant resistance that is not effective in the seedling stage, but authors usually have been vague on this point when describing the resistance of Thatcher. All of the races used here are virulent on genes *Sr5* and *Sr16*. The effectiveness of gene *Sr12*, if it exists, is unknown. It was identified with avirulent race 111 (7) and may not be effective against the races studied.

In this test the adult plant and seedling reactions were similar except for small differences with races C33 and C51. The results do not demonstrate clearly that Thatcher is susceptible in the seedling stage and resistant in the adult stage to certain races as is implied by the term "adult plant resistance." There is no explanation, in genetic terms, of the high resistance of both seedlings and adult plants of Thatcher to races C16(39) and C17(56).

The frequent isolation in recent years of stem rust races virulent on Thatcher suggests that this variety would be heavily rusted if grown commercially today.

Acknowledgments

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The nature and control of snow mold of fine turfgrass in southern Ontario

S.G. Fushtey[1]

In southern Ontario snow mold in turfgrass is caused by *Fusarium nivale* and *Typhula* spp. both usually occurring together with one or the other predominating. Chemical control trials with bentgrass (*Agrostis palustris*) over the past 5 years showed that fungicides with benomyl or related compounds as active ingredients failed to give satisfactory control where *Typhula* was predominant. Those containing chloroneb or chlorothalonil gave good control. For *Fusarium*, both benomyl and chlorothalonil performed well and a mixture of chloroneb and benomyl at half-rate of each also gave excellent control. The possibility of a synergistic effect between these two materials is indicated. Disease control is dependent on the time of fungicide application and evidence was obtained to show that fungicides should be applied late in the season, probably not before November 1.

Can. Plant Dis. Surv. 55:87–90. 1975

En Ontario, deux moisissures nivéales s'attaquent au gazon. Ce sont *Fusarium nivale* et *Typhula* sp. On les rencontre habituellement ensemble, l'une des deux en prédominance. Des essais de lutte chimique d'une durée de 5 ans ont révélé que les fongicides à base de benomyl ou composés apparentés ne donnent pas de résultats satisfaisants lorsque *Typhula* est l'espèce dominants, à l'inverse des fongicides à base de chloronèbe. Dans les cas de dominance de *Fusarium*, le bénomyl et le chlorothalonil ont donné de bons résultats, de même qu'un mélange de chloronèbe et de benomyl à demi-dose. Il est possible que les deux produits agissent en synergie. L'efficacité du traitement dépend du moment d'application des fongicides. Il semble que celle-ci doit se faire en fin de saison de préférence après le 1er novembre.

Snow mold is among the top two or three most troublesome diseases of turfgrass in Canada. Studies at Guelph have shown that at least two different fungi are involved as primary causal agents but that the distribution of these fungi is unpredictable. For example, on a naturally infested experimental area on the university campus the disease was caused by *Typhula* sp. with only trace evidence that *Fusarium nivale* was also present. In contrast, the disease on a golf course just 0.5 mile distant from the university plots was predominantly *F. nivale* with *Typhula* sp. occurring on most greens but only in small patches and only where deep drifts of snow accumulated. At the Cambridge Research Station where a newly established bentgrass area was artificially infested with heavily diseased material from the campus plots, the snow mold which developed was predominantly *Typhula* in 1972 and 1973 with a noticeably higher incidence of *Fusarium* in 1974. In 1975, the incidence of disease was low and was nearly all *Fusarium nivale*. Apparently local factors influence the development of these fungi to favor one or the other but the nature of these factors is not well understood.

Smith (1974) presented an excellent paper on snow molds in the province of Saskatchewan and reported six different fungi associated with the problem: the low-temperature basidiomycete (LTB) first reported by Broadfoot and Cormack (1941), *Fusarium nivale*, *Sclerotinia borealis*, *Typhula* spp., a sclerotial low-temperature basidiomycete (SLTB), and a fungus with orange rindless sclerotia (ORS). Smith did not state whether these were found in mixtures on the same turf area but this may be assumed from the statement that ORS was found on snow mold patches often associated with SLTB, *S. borealis*, and *Typhula* spp.

Some variability in symptoms of the snow mold disease in Ontario suggests the possibility that more than two fungi may be involved here too but this has not been confirmed. Sclerotia are not commonly found on bentgrass in the Guelph area but those that have been observed are typically those of *Typhula* spp., and an orange sclerotial form of a fungus resembling *Typhula* was found at the Cambridge Research Station in 1975.

Materials and methods

Chemicals tested included commercial products and experimental fungicides. Wettable powders were applied with a hand sprayer in 0.8 qt (0.9 liter) water per plot. Plots measured 50 ft^2 (4.65 m^2) and were replicated four times.

Disease readings were made each year in early April using the Horsfall and Barratt system of rating numbers modified to range from 0 to 11 instead of the original 1 to 12 (Horsfall and Vargas 1945, Redman et al.1971).

Fungicides tested

Tersan 1991, benomyl 50%, WP, DuPont of Canada;
Tersan SP, chloroneb 65%, WP, DuPont of Canada;
Tersan 75, thiram 75%, WP, DuPont of Canada;

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Daconil 2787, chlorothalonil 75%, WP, Diamond Shamrock;
Daconil (Bravo), chlorothalonil 54%, flowable, Diamond Shamrock;
Mertect, thiabendazole 60%, WP, Merck & Co.;
NF-44, thiophanate methyl 70%, WP, Ciba-Geigy;
Proturf F II, chloroneb 6.8%, granular, O.M. Scott & Sons;
Proturf F III, dyrene 8.7%, granular, O.M. Scott & Sons;
Uniroyal 2010, experimental, WP, Uniroyal Chemicals;
Chipman PP395, experimental, slurry, Chipman Chemicals;
Dupont DPX-164, experimental, WP, DuPont of Canada;
Baydam 18654, experimental, WP, Chemagro;
Formaturf, experimental, liquid, Stephenson Chemicals.

Results

Chemical control

For many years, fungicides containing mercury occupied top position in recommendations for control of snow mold. With increasing fears of mercury in the environment as a hazardous pollutant some of these fungicides were taken off the market and those that remained became less widely used as effective nonmercurial fungicides became available. In an effort to keep recommendations for Ontario reasonably well documented a program of testing fungicides for control of snow mold and other diseases of turf was initiated at Guelph in 1967. The early trials showed that materials such as Daconil and Demosan were as effective as the mercurials for snow mold control in the Guelph area. Both commercial and experimental fungicides have been tested each year on bentgrass (*Agrostis palustris* Huds.) plots on the university campus, then at the Cambridge Research Station when facilities were developed there. The results which follow are a summary of data obtained over the years beginning with 1970 when the program was officially recognized.

1970-71 trial

Nine different materials at 2 dosage levels were tested at both the Guelph and Cambridge locations. These included both dry and spray applications of benomyl, thiabendazole, and chloroneb. Of these, only the chloroneb formulations (Tersan SP and Scotts F-5076) gave satisfactory control. Benomyl and thiabendazole gave no indication of control whatsoever. The causal fungus at both locations was *Typhula* sp.

1971-72 trial

This trial was similar to that conducted the previous year except that some of the ineffective materials were replaced by formulations of Daconil (WP and flowable), Dyrene, thiram and thiophanate. Again, excellent control was achieved with the chloroneb formulations (both wet and dry); good control with Daconil (chlorothalonil) but only at the higher dosage (6 oz active); moderate control with Dyrene and only at the high dosage of 12 oz active; poor control with thiram and thiophanate.

1972-73 trial

This trial was conducted at the Cambridge Research Station only, the campus plots having been taken over by buildings. An important objective in this trial was to determine effect of treatment date. To do this, one series of plots was treated on October 20 and provision made for another series to be treated 4 weeks later. A heavy snow fall on November 15 prevented treatment on the scheduled second date. With no sign of a thaw an attempt was made to remove the snow and apply the treatments on December 1.

Although materials containing chloroneb or chlorothalonil gave good control when applied in October none of the treatments gave satisfactory control in the December 1 application. This is attributed to improper placement of the material rather than timing. All of the ice and snow could not be removed from the turf prior to treatment and much of the fungicide was applied onto a thin layer of ice and snow. The trial failed to provide information on the importance of timing of treatments but impressed the importance of getting it done before the snow falls so that the material is all placed in direct contact with the turf.

1973-74 trials

One trial was conducted at the Cambridge Research Station where the causal fungus was predominantly *Typhula* sp. and another on the practice green at the Cutten Golf Club where the fungus was predominantly *Fusarium nivale*. The test materials were all applied at 2 dosage rates and on 2 dates, namely, October 3 and November 4 at Cambridge, October 11, and November 5 at the Cutten Club.

The results can be summarized as follows: At the Cambridge site, good control was obtained with Tersan SP (chloroneb), Daconil 2787 (chlorothalonil), Proturf F II (chloroneb), Proturf F III (dyrene), and Proturf BSF (PMA-thiram) at both dates of application, but control was noticeably better where the materials were applied at the later date. Uniroyal 2010 gave good control in the November treatment only. Tersan 1991 (benomyl) was unsatisfactory at either date.

At the Cutten Club site, Daconil flowable (chlorothalonil) gave excellent control at both dates of application, Tersan 1991 (benomyl) was good at the late date only and Tersan SP was unsatisfactory. These were the only materials used at this site because of limited space.

These results can be interpreted to indicate the following:
1. That fungicides for snow mold control should be applied late in the season, probably not before November 1.
2. That not all fungicides recommended for snow mold control will control both *Typhula* and *Fusarium*; hence it is important to be able to recognize the disease caused

Table 1. Snow mold incidence in turf plots treated with fungicidal chemicals, Cambridge, 1975

Product	Dosage (product) (oz/1000 ft^2)	Dosage (product) (g/m^2)	Percent disease* *Fusarium*	Percent disease* *Typhula*	Percent disease* Total	Percent control
Tersan 1991	2	0.6	0.6	0.0	0.6	91
	4	1.2	1.2	0.6	1.8	74
Tersan SP	6	1.8	1.2	0.0	1.2	83
	9	2.8	0.6	0.0	0.6	91
Daconil (Bravo)	12	3.7	2.3	0.0	2.3	67
	16	4.9	1.6	0.0	1.6	77
Uniroyal 2010	4	1.2	2.3	0.0	2.3	67
	8	2.4	1.2	0.0	1.2	83
Chipman PP 395	5	1.5	4.1	0.0	4.1	27
	10	3.0	1.8	0.0	1.8	74
Dupont DPX-164	4	1.2	1.2	0.0	1.2	83
	8	2.4	1.8	0.0	1.8	74
Baydam 18654	4	1.2	4.2	1.2	5.3	24
	8	2.4	2.3	1.6	3.9	44
Formaturf	6	1.8	2.3	12.3	14.5	0
	12	3.6	2.9	25.8	28.7	0
Check (no treatment)			4.7	2.3	7.0	

* Disease readings according to Barratt-Horsfall rating numbers converted to percent according to Elanco Conversion Tables (see text).

by each of these fungi if fungicides such as chloroneb and benomyl are to be used effectively.

1974-75 trials

These trials were laid out in essentially the same way as in the previous year but the incidence of *Typhula* turned out to be negligible at both sites hence the results are primarily for the control of *Fusarium nivale*. The summary results are given in Tables 1 and 2 for the Cambridge Station and Cutten Club sites respectively.

Disease incidence was low at both sites but particularly so at the Cambridge Station. Furthermore, distribution of disease over the test areas was so uneven that none of the results are statistically significant. Nonetheless, the results do show practical levels of control. In the data for *Fusarium* (Table 1) values of less than 2.3% were derived from individual rating numbers of 0 and 1, the latter representing a trace of disease with negligible damage. The value of 4.7% recorded for the check plots was derived from individual rating numbers ranging from 1 to 3, the latter representing up to 12% disease. Visually this is a substantial amount of disease with appreciable damage. Thus, all materials except Formaturf, Bay Dam 18654, and PP 395 at the lower dosage, provided a practical degree of controlling *Fusarium nivale*. There was insufficient *Typhula* disease generally to give any indication of control. Of some interest is the series of plots treated with Formaturf which developed up to 10 times the disease in the check plots. This may have been due to the phytotoxic nature of this chemical. No injury was evident after application on November 12, at which time the grass was essentially in a dormant state; however when the material was used for dollar spot control in June and August phytotoxic effects were severe.

Concerning data given in Table 2, all three materials (Tersan 1991, Tersan SP, and Daconil) gave satisfactory control at dosages recommended by the manufacturer. Tersan SP performed well against *Fusarium* although it is not recommended for this purpose because past performance has been less effective. Excellent control was obtained when Tersan 1991 and Tersan SP were applied together, each at half the recommended dosage. The performance of this combination appears to be superior to that obtained with the regular dosage of one or the other chemical alone. This would not be expected unless there was some kind of synergistic effect. This possibility is being investigated in laboratory studies.

Discussion and conclusions

The snow mold disease of turfgrass in southern Ontario is caused by *Fusarium nivale* and *Typhula* spp. usually

Table 2. Snow mold incidence in turf plots treated with fungicidal chemicals, Cutten Club, 1975

Product	Dosage (product) (oz/1000 ft^2)	Dosage (product) (g/m^2)	Percent disease* *Fusarium*	Percent disease* *Typhula*	Percent disease* Total	Percent control
Tersan 1991	2	0.6	3.5	0.6	4.1	69
	4	1.2	1.8	0.6	2.4	82
Tersan SP	4	1.2	2.4	1.2	3.6	73
	6	1.8	1.2	0.6	1.8	87
	9	2.8	1.2	0.0	1.2	91
1991 + SP	2 + 3	0.6 + 0.92	0.0	0.6	0.6	95
Daconil (Bravo)	12	3.7	0.6	0.0	0.6	95
	16	4.9	0.6	0.0	0.6	95
Check (no treatment)			11.1	2.3	13.4	

* Disease ratings according to Barratt-Horsfall rating numbers converted to percent according to Elanco Conversion Tables (see text).

occurring together with one or the other predominating. The identity of the *Typhula* spp. have not been determined but two distinctly different sclerotial forms have been observed indicating that two species at least are present. An effort is being made to identify these species.

The control of snow mold is complicated by the fact that it is usually caused by more than one of these fungi occurring at the same time. Apart from proper management to avoid predisposition of turf to disease the only known control is the use of fungicides in the late fall. Until recent years broad spectrum fungicides containing mercury were reasonably effective. As more specific-acting nonmercurials replace the mercuries, chemical control becomes more complicated because not all fungicides recommended for snow mold control are effective against all the fungi that may be involved. For example, benomyl, which is effective against *Fusarium* is unsatisfactory against *Typhula*, while chloroneb, which gives excellent control of *Typhula* is only partially effective against *Fusarium*. Consequently, it is important to know what fungus predominates before one of these fungicides is used.

Concerning timing of treatment, the common recommendation to apply the fungicide as late as possible prior to permanent snowfall is risky as was proven by the 1972-73 trial when a permanent snowfall occurred on November 15. Normally this does not occur until early December in the Guelph area. Vargas and Beard (1971) showed that at Harbor Springs, Michigan, chloroneb and Calo-Gran can be applied as early as mid-October with results equally as good as when applied 1 month later. This would probably apply for the Guelph, Ontario, area as well. The 1973-74 trials showed that treatment in early October was generally inferior to early November for *Typhula* control. For fusarium snow mold, control was achieved only at the later of the two treatment dates October 11 and November 5. These results indicate that treatment for snow mold, should be done no earlier than mid-October.

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Snow molds on winter cereals in northern Saskatchewan in 1974[1]

J. Drew Smith

Isolates of a nonsclerotial low temperature basidiomycete (LTB), a *Typhula* sp. (FW), and *Sclerotinia borealis* from grasses were pathogenic on four cultivars of winter wheat (*Triticum aestivum*) and one of rye (*Secale cereale*) in a field test at Saskatoon in the winter of 1973-74 when snow depth and duration was above average. In spring 1974 the same wheat and rye cultivars were killed by *S. borealis* and *Typhula* FW in a cooperative test at Loon Lake in northern Saskatchewan. *S. borealis* also caused damage in a similar test at Scott, while other tests showed light infections of *Fusarium nivale* and/or *S. borealis* or no apparent damage. No clear differential effects of pathogens on cultivars was noted. In winter wheat crops *S. borealis, Typhula incarnata* (a new record for Saskatchewan), *Typhula* FW, and an unidentified sclerotial fungus (ORS) were recovered from dead plants. Of 50 fall rye crops surveyed in northern Saskatchewan in spring 1974, 45 showed damage by *S. borealis*, ranging from a trace infection to over 50% kill. *F. nivale* also caused injury to some rye crops and the LTB was found damaging this crop for the first time in North America. The diversity of pathogens and their ability to cause epidemics or significant damage on winter cereals under suitable conditions indicate the need to define more precisely the components of winter killing. In the breeding of winter cereals for Saskatchewan attention must be paid to snow mold resistance.

Can. Plant Dis. Surv. 55:91–96. 1975

Des isolats d'un basidiomycète psychrophile à sclérote (LTB), d'une espèce du genre *Typhula* (FW) et de *Sclerotinia borealis* prélevés sur des graminées se sont révélés pathogènes à quatre cultivars de blé d'hiver (*Triticum aestivum*) et un de seigle (*Secale cereale*) lors d'une épreuve en plein champ qui s'est déroulée à Saskatoon au cours de l'hiver 1973-1974 dans des conditions d'enneigement plus important et plus long qu'à l'ordinaire. Au printemps 1974, les cultivars de blé et de seigle ont été détruits par *S. borealis* et *Typhula* FW dans un essai coopératif à Loon Lake dans le nord de la Saskatchewan. *S. borealis* a également causé quelques dégâts au cours d'un essai semblable, à Scott, mais les autres essais n'ont révélé que des infections bénignes de *Fusarium nivale* ou *S. borealis* ou aucun dommage visible. On n'a remarqué aucune différence nette dans l'effet des micro-organismes sur les cultivars. Les plants de blé d'hiver détruits renfermaient *S. borealis, Typhula incarnata* (la première manifestation de sa présente en Saskatchewan), *Typhula* FW et un champignon non identifié à sclérote. Sur 50 champs de seigle d'automne examinés au printemps 1974 dans le nord de la Saskatchewan, 45 manifestaient de dégâts commis par *S. borealis*, dégâts variant de l'infection à l'état de trace à une destruction supérieure à 50%. *F. nivale* a également attaqué certaines cultures de seigle où l'on a constaté pour la première fois l'action du basidiomycète psychrophile. La diversité des micro-organismes pathogènes et leur capacité d'entraîner des épidémies ou de causer des dommages sensibles aux céréales d'hiver dans certaines conditions fait ressortir la nécessité d'une étude plus détaillée sur les différents facteurs responsables de la destruction des céréales par l'hiver. La sélection des céréales d'hiver destinées à la Saskatchewan devrait tenir compte de la résistance aux moisissures nivéales.

Snow molds, notably *Fusarium nivale* (Fr.)Ces., *Typhula* spp., and *Sclerotinia borealis* Bub. & Vleug. cause severe losses in winter cereals and grasses in Scandinavia, northern USSR, and Japan (4). In parts of Washington, Idaho, and Montana, *Typhula* spp. and *F. nivale* are important pathogens of winter wheat (12); in southern Alberta *F. nivale* was recorded in epidemic proportions on winter wheat in 1967 (5). *Typhula* spp. have been recorded causing occasional damage to winter wheat in the interior valleys and northern areas in British Columbia (3, 6). *S. borealis* was probably involved in damage to winter rye in Alaska and to wheat and rye in northern British Columbia in 1952 and 1971 respectively (2 and J. S. Horricks, personal communication, 14 June 1974), and it has been found once at high elevation in Washington (13). The host range of the nonsclerotial low-temperature basidiomycete, LTB, a pathogen perhaps unique to certain parts of western North America, includes winter wheat (1). In Saskatchewan, *F. nivale* and *S. borealis* were first noted as widespread pathogens of turf and road verge grasses in 1971 (8). Since then it has become apparent that they and a *Typhula* sp. designated FW are common snow molds on grasses in Manitoba, Saskatchewan, Alberta, and northern British Columbia (8, 9, 10, 11). *Typhula* FW is a different species from the *T. ishikariensis* Imai and *T. incarnata* Lasch ex Fries reported by Vaartnou and Elliott from northwestern Canada (15). Neither *T. idahoensis* Remsberg, which some workers consider synonymous with *T. ishikariensis*, nor *T. incarnata* has yet been recorded on grasses in Saskatchewan, although the latter was found frequently in disease surveys of turfgrasses in British Columbia, Washington, and Idaho in 1973 and 1974 (9 and unpublished).

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This note reports results of 1973-74 field tests to determine whether some of the snow mold fungi from grasses were pathogenic towards winter wheat and rye, and of disease surveys in winter cereals in Saskatchewan made in spring 1974 following a winter when snow depth was much above average.

Methods

Isolates of the LTB, *S. borealis, Typhula* FW, and a *Sclerotium* sp. isolated from grasses were grown on sterile rye grain, at 5°C for the first two species and at 12°C for the latter two, in the dark for periods of 10-14 weeks. In July 1973 inoculum of air-dried cultures of all but the LTB was applied by hand broadcasting to raked and levelled soil of individual plots in a randomized block layout with four replications. Each plot of 11 m^2 received 400 g of inoculum of *S. borealis* or *Sclerotium* sp. or 800 g of *Typhula* FW, which was then raked lightly into the soil surface. LTB inoculum was applied with the seed at 2:1 w/w when plots were sown in September with a V-belt seeder. There were four uninfested plots; 0.3 m wide uninfested pathways reduced the risk of plot contamination. Four rows 3.3 m long and 23 cm apart of each of the winter wheat cultivars Alabaskaja, Kharkov, Ulianovka, and Sundance and of the fall rye cultivar Frontier were sown at the rate of 1 g/m within each plot area. Two snow fences running north and south trapped snow on the test area (Fig. 1). Winter snowfall at Saskatoon for 1973-74 was 1704 mm and a snow cover persisted for 170 days from 31 October to 17 April. This is much above the average snowfall for the previous 33 winters of 1087 mm and 143 days snow cover (personal communication J. Maybank, Physics Department, Saskatchewan Research Council, 19 August 1974).

Results

Field tests

Injury to wheat and rye cultivars from all snow molds except the *Sclerotium* sp. was apparent soon after snow melt in mid-April 1974 (Fig. 1). An estimate of percent plant death per plot was made in mid-May. In plots inoculated with *Typhula* sp. this varied from 40% to 95%. Where kill was 95% only a few plants of Frontier rye survived. In LTB-inoculated plots, kill varied from 25% to 40% and in those with *S. borealis* inoculum plant death was 2-5%. No differential response of cultivars to the LTB or to *S. borealis* was apparent. Occasional plants in both infested and control plots showed typical leaf lesions caused by *F. nivale* (Fig. 3) and some plants died.

Surveys

Disease surveys of winter wheat and rye cooperative cultivar tests established by Agriculture Canada and the Crop Development Centre, University of Saskatchewan, at various centers in the province were made between late April and late June 1974. The locations and Crop Districts (CD) are shown in Figure 1a. The cultivars

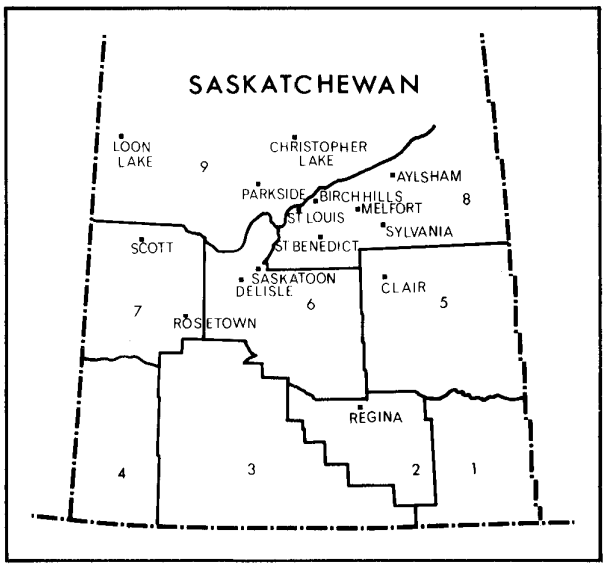
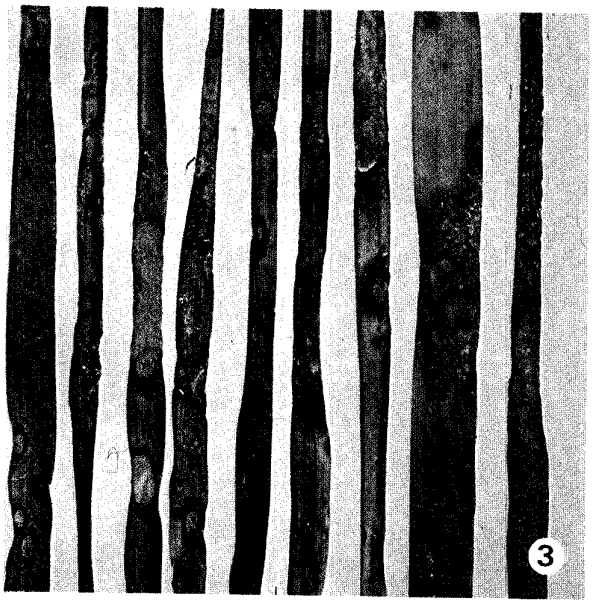


Figure 1a. Saskatchewan crop districts, locations of test plots and survey fields.

included those in the pathogenicity test at Saskatoon. In late April, at Loon Lake (CD 9) all cultivars of wheat and rye showed heavy infection with *S. borealis* (Fig. 4). Sclerotia of *Typhula* FW and of a fungus with orange sclerotia, designated ORS (8, 9, 11) were also found. None of the wheat or rye plants survived. The Rosetown (CD 7) test showed severe winter damage but neither *S. borealis* nor other snow molds were noted. The Scott test (CD 7) showed *S. borealis* infection and winter damage. No clear differential effects on cultivars were evident at Loon Lake, Scott, or Rosetown. At Saskatoon (CD 6) light infections with *S. borealis* and *F. nivale* occurred on winter wheat in tests at two of three locations. Snow mold damage was not found in late June in the tests at Parkside (CD 9), Melfort (CD 8), and Clair (CD 5). In commercial fields of Winalta winter wheat in CD 8, *S. borealis, T. incarnata* (a new record for Saskatchewan), *Typhula* FW, and the ORS fungus were recovered from dead plants at Aylsham, and *S. borealis* and *Typhula* FW from a crop at Sylvania. Three

Figures 1–4. Snow mold damage to fall-planted crops of wheat and rye, Saskatchewan, April–May 1974:
1) Snow mold pathogenicity test, Saskatoon, mid April; on rows of winter wheat and rye, foreground plot *Typhula* FW, next plot LTB.
2) *Fusarium nivale* damage to rye, natural infection, St. Louis.
3) *Fusarium nivale* leaf spots on Yogo winter wheat.
4) Winter wheat plants killed by *Sclerotinia borealis*; note sclerotia (arrows), X 0.5.



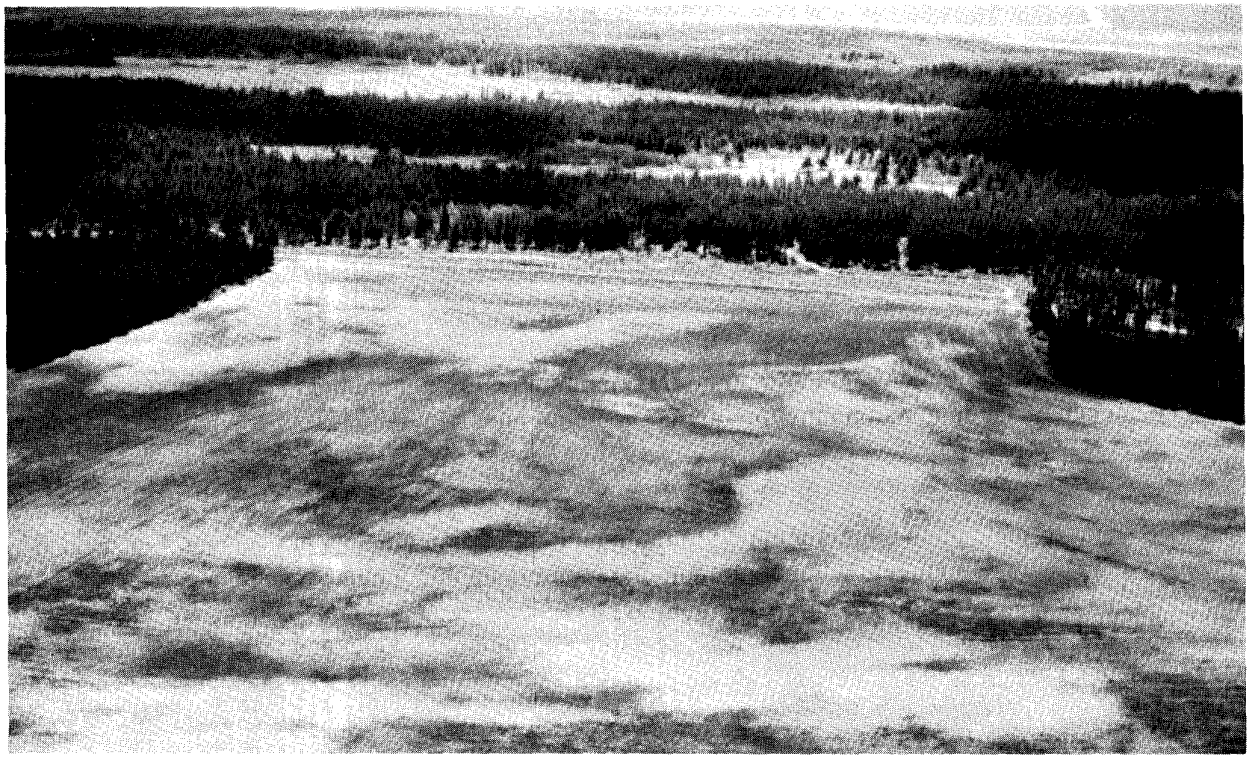


Figure 5. Extensive bleached areas of *Sclerotinia borealis* damage to rye, Prince Albert; aerial photograph June 1974.

crops of Sundance winter wheat in the St. Benedict and Birch Hills areas had no snow mold symptoms in early May.

From 30 April to 16 May surveys of rye fields and other plot tests in Saskatchewan crop districts 6, 8, and 9 (Fig. 1a) were made. Of 50 rye sites examined, 45 showed plants killed by *S. borealis*. Severity ranged from trace to more than 50% kill on 60 acres at Christopher Lake and on 100 acres near Prince Albert (Fig. 5). *F. nivale* was an occasional pathogen in crops except at St. Louis where damage was locally severe (Fig. 2). The LTB fungus was isolated from diseased rye at Delisle where it was apparently causing damage in association with *S. borealis*.

Discussion

Winter wheat production in Saskatchewan has been confined to a few thousand acres in the southwest and scattered fields in the parkland region. Fall rye, a more important crop than winter wheat, occupied about 1.5% or 328,000–502,000 acres of the total cereal acreage in the years 1968-1971 (7). Fall rye is a useful crop on light land for erosion and weed control, spring grazing, and grain production. Winter killing is regarded as a hazard unless the winter-hardy cultivars Frontier, Puma, or Antelope are used and plants are well established by freeze-up (14). Comparatively little attention has been given to the pathology of the rye crop in Canada but the finding for the first time in this province of *S. borealis* and *F. nivale* on heavily damaged stands suggests that an attempt should be made to define more precisely the components of the portmanteau term "winter-killing". The virulence of *Typhula* FW and LTB isolates from Saskatchewan grasses on rye and wheat in the Saskatoon tests and the isolation of the LTB fungus from a field of rye, a new record for western North America, suggest that the hazards of snow mold on this crop require closer examination.

Although the 1974 survey was limited to crop districts 6, 8, and 9, the pathogens obtained, namely *F. nivale, S. borealis, Typhula* FW, and the LTB, are known to be widespread on grasses of the province and generally in western Canada. *S. borealis* was also found in several rye crops in a survey in northern and western Manitoba in July 1974 (Smith, unpublished). In turfgrasses in particular, in western Canada these snow molds cause epidemics only when suitable microclimatic conditions develop. For example, *F. nivale* appears from summer to fall after a rainy, cold period or nonpersistent snow; *S. borealis* and *Typhula* FW seem to need prolonged, deep snow covers. The LTB appears to be able to cause damage even under a shallow, shorter duration snow cover than favors *S. borealis* or *Typhula* FW although it can also reach epidemic proportions under protracted snow covers (9, 10). Probably conditions suitable for snow molds could occur on winter cereals in many areas

of Canada. In the winter of 1973-74, attacks of *S. borealis* on cereals in Saskatchewan appear to have been favored by the prolonged, deep snow cover. Winter wheat is little grown at present in northern Saskatchewan so the survey was limited. However, winter hardy North American and Russian wheat cultivars were eliminated at Loon Lake, largely by *S. borealis*, and were damaged elsewhere by this pathogen and by *F. nivale*. Attempts to extend the range of winter wheat through the use of improved varieties and cultural practices should not overlook snowmold problems intimately related to winter hardiness.

Acknowledgments

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Diseases of specialty crops in Saskatchewan: II. Notes on field pea in 1973-74 and on lentil in 1973

D.L. McKenzie and R.A.A. Morrall[1]

Ascochyta blight [*Ascochyta pinodes*], sclerotinia stem rot [*Sclerotinia sclerotiorum*], and fusarium root rot were the most common diseases of field pea (*Pisum sativum* var. *arvense*) in Saskatchewan in both 1973 and 1974. In 1973 fusarium root rot and heat canker caused by high temperatures near the soil surface were prevalent on lentil (*Lens culinaris*).

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Les maladies les plus communes du pois (*Pisum sativum* var. *arvense*) en 1973 et 1974 ont été, en Saskatchwan, le pourridié ascochytique [*Ascochyta pinodes*], le pourridié fusarien, et la flétrissure sclérotique [*Sclerotinia sclerotiorum*]. En 1973, les maladies dominantes de la lentille (*Lens culinaris*) étaient le pourridié fusarien et un chancre induit par des températures élevées.

Disease surveys were done in Saskatchewan on crops of field pea (*Pisum sativum* var. *arvense* (L.) Poir.) in 1973 and 1974, and on crops of lentil (*Lens culinaris* Medik.) in 1973. These surveys comprised part of a continuing study of specialty crop diseases (3,4,5,6). Both crops are important protein sources and, as such, are being studied intensively at the Crop Development Centre, Saskatoon. However, provincial acreage data suggest that only field pea has considerable commercial prospects in the near future (3,6). Estimated figures for 1973-74 are as follows: field pea: 1973 - 4,000 acres; 1974 - 6,500 acres; lentil: 1973 - 2,500 acres; 1974 - 600 acres. The low acreage in 1974 was the principal reason for not continuing the lentil survey in 1974.

Methods

In 1973 11 fields of lentil and 24 of field pea were examined. Survey trips were made at three different times during the growing season, although all the fields were not surveyed each time. The objective was to avoid missing diseases that might be masked by the end of the growing season, and also to obtain some idea of changes of disease with time. However, time limitations in 1974 required that the 17 fields of pea surveyed be sampled only once, at the end of the season.

The fields of lentil were all in the Eston-Kindersley area (150 miles SW of Saskatoon). In both 1973 and 1974 the majority of crops of field pea were in the traditional growing areas of Bellevue (60 miles NE of Saskatoon) and Nipawin (150 miles NE of Saskatoon). However, one field near Marengo (170 miles WSW of Saskatoon) in 1973 and five fields in the Tisdale area (120 miles NE of Saskatoon) in 1974 were also surveyed; these were in new areas for the cultivation of field pea in Saskatchewan.

Observations prior to the final sampling in 1973 were largely qualitative. The presence, and approximate levels of diseases in each field were noted while walking about 300 meters in a semicircular pattern through the crop. Quantitative estimates of disease intensity at the end of the two growing seasons were made using techniques slightly modified from those described previously (3). Plants were sampled in only two widely separated parts of each field and, in the case of field pea, estimates of percentage area diseased were made on 100 triplets of leaflets and 100 pods from each sample area. Intensity of root and stem diseases of field pea were measured on a m^2 basis, but those of lentil on a percentage of total plants basis. Isolations from diseased plant material were always made, using routine laboratory procedures, when the causal organism was unknown or uncertain.

Results

Field pea (Tables 1, 2)

The principal diseases in both years were ascochyta blight [*Ascochyta pinodes* L.K. Jones], sclerotinia stem rot [*Sclerotinia sclerotiorum* (Lib.) de Bary] and fusarium root rot [*Fusarium* spp.]. Other diseases observed at trace levels were rhizoctonia root rot [*Rhizoctonia* sp.], botrytis stem blight and leaf spot [*Botrytis cinerea* Pers.] and powdery mildew [*Erysiphe polygoni* DC. ex Mérat] in 1973, and bacterial blight [*Pseudomonas pisi* Sackett] in 1974.

In 1973 ascochyta blight had appeared in only one field by mid-June (Table 1). However, by mid-July it was present in all fields in the Nipawin and Bellevue areas, and often at higher than trace levels. By the final sampling date the disease was observed even in the field at Marengo. At the end of the season considerable variation between fields occurred in the mean percent-

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Table 1. Summary of qualitative survey data for field pea, Saskatchewan 1973

District	Date	No. of fields examined	Number of fields where diseases were present			
			Ascochyta blight	Sclerotinia stem rot	Fusarium root rot	Other*
Bellevue	14/6	2	1	0	0	0
	17/7	5	5	1	1	a
	27/8	7	7	5	5	b
Nipawin	19/6	2	0	0	1	0
	11/7	16	16	0	7	0
	4/9	10	10	9	9	c
Marengo	26/6	1	0	0	0	0
	31/7	1	0	0	1	0
	13/8	1	1	0	1	0

* a = leaf spot, (*Botrytis cinerea*) 1 field;
b = root rot, (*Rhizoctonia sp.*) 3 fields, powdery mildew (*Erysiphe polygoni*), 1 field, and stem blight (*Botrytis cinerea*) 1 field;
c = root rot (*Rhizoctonia sp.*) 6 fields and powdery mildew (*Erysiphe polygoni*) 1 field.

Table 2. Intensity of diseases of field pea in late season by district, Saskatchewan 1973-74

Year and District	Percentage area diseased				Number of plants diseased/m^2					
	Ascochyta on leaves		Ascochyta on pods		Sclerotinia stem rot		Fusarium root rot		Rhizoctonia root rot	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
1973										
Bellevue	3.9	1.2– 7.7	2.1	0.9– 3.7	3.0	0–10.5	2.9	0–7.0	0.5	0–1.5
Nipawin	4.7	1.3–10.1	5.9	0.4–13.6	4.1	0–10.0	1.3	0–2.5	0.8	0–1.5
Marengo	2.4	na	0.3	na	0	na	1.0	na	0	na
Mean	4.3	1.2–10.1	4.1	0.9–13.6	3.4	0–10.5	1.9	0–7.0	0.6	0–1.5
1974										
Bellevue	11.3	2.8–19.4	5.7	1.0– 8.0	0.5	0– 1.0	0.2	0–1.0	0	na
Nipawin	2.0	0.2– 6.8	0.4	0 – 0.8	0.3	0– 1.5	0.2	0–1.0	0	na
Tisdale	5.1	3.8– 8.3	2.5	0.8– 2.7	0	na	0.2	0–1.0	0	na
Mean	5.7	0.2–19.4	2.6	0 – 8.0	0.3	0– 1.5	0.2	0–1.0	0	na

na = not applicable

age leaf area infected (Table 2), but there appeared to be generally more disease in the Nipawin area than in the Bellevue area. Pod infections were also common in both areas, with seeds in the more extensively infected pods usually being partially decayed. At the end of the 1974 season ascochyta blight appeared to be more intense in the Bellevue area than in the Nipawin area (Table 2). The presence of the disease at Marengo in 1973 and of relatively high levels of it in the Tisdale area in 1974 were not unexpected in view of the well known seed-

Table 3. Summary of qualitative survey data for lentil, Saskatchewan 1973

Date	Number of fields examined	Number of fields where diseases were present*						
		Fusarium root rot	Rhizoctonia root rot	Unknown† root rot	Botrytis stem rot	Sclerotinia stem rot	Heat canker	Other§
26/6	11	10	2	0	3	0	9	a
31/7	10	5	0	1	0	1	0	b
13/8	9	9	0	5	0	0	5	0

* Diseases indicated were all present at trace levels except for heat canker on 26/6, and fusarium root rot and the "unknown" root rot on 13/8, when the levels ranged from trace to slight.

† The cause of this root rot is unknown; see text.

§ a = Leaf blight (*Alternaria* sp.) 3 fields and leaf blight (*Cladosporium* sp.) 3 fields;
b = Leaf blight (*Alternaria* sp.) 2 fields.

borne nature of *A. pinodes*. Nonetheless, they are disturbing examples of the introduction of unwanted pathogens into new areas of cultivation of the host.

In 1973 and 1974 (Table 2) the mean levels of ascochyta blight, fusarium root rot, and powdery mildew were comparable to those recorded in 1972 (3). Powdery mildew, a common disease on peas in Saskatchewan gardens, was rarely observed in fields in any of the 3 years, probably because of the different microclimate which commonly prevails in large fields. In sharp contrast to other diseases, sclerotinia stem rot was, compared with 1972, 10 times more abundant in 1974 and 50 times more so in 1973. However even in 1973 the disease was only observed late in the season, with the exception of one field in the Bellevue area.

Lentil (Table 3)

One of the major diseases of lentil was fusarium root rot, and most isolates from diseased roots were cultivars of *Fusarium roseum* (sensu Snyder and Hansen). A seedling blight phase of the disease occurred in trace amounts in many fields on June 26. On August 13, shortly before swathing in most fields, the disease affected a mean of 2.7% of the plants (Range: 0.5–6.0%). This was less than in an earlier survey (3), but apparently, as in the earlier survey, damage to infected plants was slight. Root rot or seedling blight caused by *Rhizoctonia* sp. was also recorded in early season in two fields. A third root rot, of unknown etiology, occurred in several fields, and on August 13 affected a mean of 1% of the plants (Range: 0–4.0%). Isolations from diseased tissue yielded only bacteria, and proof of pathogenicity was not established. Most of the bacteria were cream- or yellow-pigmented and gram negative.

Heat canker of seedlings was very common in early summer. The symptoms of basal stem necrosis and leaf discoloration (Fig. 1) were very similar to those on flax described by Vanterpool (7). A period of hot weather with intense insolation had occurred in the area shortly

Figure 1. Heat canker of lentil. Note the constriction of the stem at the soil level and the discoloration of the leaves.

before the survey trip. In midsummer heat canker was not observed on standing plants in the field; however, dead plants with shrunken stem bases were seen lying on the ground, apparently broken off by wind action. In late summer standing plants with heat canker symptoms were again observed, but they appeared to be as vigorous as healthy plants, judging by size and pod formation. A mean of 2.9% of the plants (Range: 0–25.0%) were affected.

Traces of stem rots caused by *Botrytis cinerea* and *Sclerotinia sclerotiorum* were observed at different times during the season. These diseases were also reported in previous surveys (3, 6). Although no attempt was made to assess leaf disease quantitatively, plants with leaf necrosis were occasionally found in early and mid-season. *Alternaria* sp. and *Cladosporium* sp. were isolated from these leaves.

Discussion

In 1973 the weather in the area of Saskatchewan where lentil was cultivated was abnormally dry. This is probably consistent with the low levels of botrytis and sclerotinia stem rots but with the frequent occurrence of heat canker. However, it is worth noting that surveys over 4 years now (3, 6, and present study) have failed to reveal any serious pathological problems on lentil, especially on dryland crops. Thus it is somewhat ironic that lentil acreages have declined in the period 1972-74 in Saskatchewan (3), and that prospects for the near future are not bright. The main problems seem to relate to weed control and the value of the crop relative to cereals.

On the other hand the future for field pea seems bright. This crop is mainly cultivated in the moister northern and northeastern crop districts of Saskatchewan. In this part of the province, especially in the Nipawin area, 1973 was an exceptionally wet year. Such conditions would be expected to have favored two of the major diseases of peas, ascochyta blight and sclerotinia stem rot.

The importance of ascochyta blight on peas throughout Canada is already well documented (1, 2, 3, 6, 8) and considerable work has been done on the disease. However, adequate control appears to be a somewhat distant prospect. Ali-Khan et al. (1) showed that among 1200 plant introductions none showed a high level of resistance to *A. pinodes*. Thus resistant varieties, such as have been used successfully to control *A. pisi* Lib. (9), are likely to be available only after an extensive breeding program.

It is noteworthy that one of the major diseases of pea in this survey, sclerotinia stem rot, was not recorded at all in an extensive survey of peas in eight other Canadian provinces in the years 1970 and 1971 (2). However, our two previous surveys in Saskatchewan (3, 6) recorded this disease. The reasons for this difference are unclear, although two explanations seem possible. Most of the survey by Basu et al. (2) was on processing peas and was done during the main harvest period. Since processing peas are harvested earlier than field (dry) peas, and since in the present survey sclerotinia stem rot was usually observed only in late season, it is possible that the disease had not had time to develop in the fields surveyed by Basu et al. Another possible factor relates to other crops grown in rotation with peas. In the pea growing areas of Saskatchewan, rapeseed, an important alternative host for *S. sclerotiorum* is frequently cultivated, whereas a similar situation may not exist in certain other Canadian provinces.

Acknowledgments

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Pythium sylvaticum in Canadian forest nurseries

O. Vaartaja[1]

Pythium sylvaticum is widespread in Canada and, at least in some soils, very abundant. It was associated with damping-off of *Pinus resinosa* in forest nurseries. In aseptic pathogenicity tests, *P. sylvaticum* was highly virulent.

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Pythium sylvaticum, que l'on trouve partout au Canada, est au moins très abondant dans certains sols. On l'associe à la fonte des semis de *Pinus resinosa* dans les pépinières forestières. *P. sylvaticum* s'est révélé très virulent lors d'essais aseptiques de pathogénicité.

The purpose of this report is to discuss briefly the occurrence of *Pythium sylvaticum* Campbell and Hendrix (1967) in Canada and to describe results of a pathogenicity test with red pine, *Pinus resinosa* Ait.

Incidental to studies on fungitoxicants in nursery soil, to be published elsewhere, observations were made on this *Pythium* species, which deserves special attention. Most isolates of this predominantly heterothallic species do not produce oogonia (unless mated with a compatible strain) and have not been recognized in the past when all *Pythium* species were thought to be homothallic (Middleton 1943).

This species has also been called *Pythium debaryanum* var. *pelargonii* Braun (Pratt and Green 1971), and possibly other names. The synonymy and other confusion surrounding this species is partly a result of the species unexpectedly containing heterothallic and homothallic strains of varying strengths (Pratt and Green 1971). These aspects will be dealt with in another paper.

Methods and results

Occurrence

In 1972, 1973, 1974, and 1975 *Pythium sylvaticum* was frequently found in the sandy soil at the Dolman Ridge nursery at the Canadian Forestry Service's Research Forest at Ramsayville, Ont. The identity of the heterothallic isolates was established through pairings with known female and male isolates. This fungus was also isolated from red pine seedlings growing in the nursery in untreated beds and in beds treated with various fungitoxicants.

In 1973 and 1974 *P. sylvaticum* was similarly found in sandy, litter-amended soil at the nursery of the Petawawa Forest Experiment Station, Chalk River, Ont. An attempt was made to find what proportion of the damping-off disease in this nursery was caused by this fungus. Diseased portions of the seedlings were placed on 1.5% water agar and the pathogens isolated and identified. The incidence of *P. sylvaticum* in 64 seedlings sampled on 23 June 1974 was 10. Similar results were obtained in samplings at other dates.

I have also isolated *P. sylvatium* from *Picea glauca* seedlings from two nurseries in Saskatchewan (Indian Head - loamy soil, and Big River - sandy soil).

The survey of *Pythium* in Ontario forest nurseries, including Petawawa, done in 1966 (Vaartaja 1968) did not report *P. sylvaticum*. Probably this species was present but could not be recognized until Campbell & Hendrix (1967) discovered heterothallism in this species. To check this possibility a few remaining viable cultures from the 1966 survey were reexamined. As a result *P. sylvaticum* was recorded from additional locations in Ontario: Midhurst nursery - sandy soil; Orono nursery - sandy soil; Longlac nursery - sandy soil; Maple - agricultural land (loam); Maple - Southern Forest Experiment Station (sandy soil).

Cultures received from other mycologists in Canada were also identified as *P. sylvaticum*: diseased *Picea engelmannii* seedling from Oliver nursery, Alberta (Dr. D. Hocking); rotting carrot, Lower Fraser Valley, B.C. (Dr. H. S. Pepin); carrot growing in muck soil, Bradford, Ont. (Ms N. Kalu) and Port Colbourne, Ont. (Dr. D. J. S. Barr); *Pinus resinosa*, Fredericton, N.B. (Dr. R. E. Wall).

To determine whether *P. sylvaticum* occurs in Quebec, a compost soil made from sandy turf under a sparse maple forest at Lucerne, Que., was analyzed using methods similar to that in the Ontario survey (Vaartaja 1968). Out of 64 *Pythium* propagules per 1 cm^3 soil, 25 were identified as *P. sylvaticum*.

The above data indicate that (a) *P. sylvaticum* occurs widely in Canada; (b) it is found in different Canadian soils, sometimes in high incidences; and (c) it is associated with conifer damping-off and other plant diseases in Canada.

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Table 1. Indices of health of red pine seedlings in aseptic inoculation experiments with 19 isolates of *Pythium sylvaticum* and single isolates of 4 other, better known pathogens. 3-month tests in a growth chamber

Pathogen	No. of isolates	Health index*
Pythium sylvaticum	19	4.8 ± 2.6†
Pythium aphanidermatum	1	1.5
Phytophthora cactorum	1	2.0
Pythium ultimum	1	4.0
Rhizoctonia solani	1	7.0
Controls	2	100

* Where 0 = death of seedling before emergence of cotyledons from seeds, and 100 = no symptoms of disease (see text).

† Average ± standard deviation.

Pathogenicity

The potential pathogenicity of 19 representative isolates of *P. sylvaticum* was tested on *Pinus resinosa* in replicated aseptic cultures on autoclaved water agar in large test tubes capped with glass vials. Into these were dropped germinating aseptic seeds that had been treated with 1% sodium hypochlorite to eliminate surface contaminants and incubated on autoclaved water agar until they reached an early stage of germination. The vials were kept for 3 months in growth chambers (night-day temperatures between 15°and 30°C, photoperiod 18 h, light intensity 1500 ft-c). The effect of infection was estimated visually and expressed by an index based on the amount of growth relative to that of seedlings in fungus-free controls. Thus death before cotyledons appeared was rated as 0, death at an early cotyledonal stage as 10, death when first few needles were half grown as 20, slight reduction in root growth as 90. For comparative purposes, the pathogenicity of an isolate of *P. aphanidermatum* (Edson) Fitzpatrick from the Ramsayville forest and one isolate of *Phytophthora cactorum* (Leb. & Cohn) Schroet, *Pythium ultimum* Trow, and *Rhizoctonia solani* Kuehn from the Petawawa Station were similarly tested.

All isolates of *P. sylvaticum* tested killed the seedlings at an early stage, often during later stages of germination (Table 1). These isolates representing male, female, and homothallic strains and widely different geographic origins showed no appreciable differences in pathogenicity. They were from Auckland, New Zealand; Georgia, U.S.A.; British Columbia and Ontario, Canada. The health index (average growth of seedlings) was only 4.8 (control100). The pathogenicity of *P. sylvaticum* was similar to that of the four other fungi, all well known as highly virulent pathogens. The isolate of *P. sylvaticum* from rotting carrot was also virulent on pine thus suggesting lack of pathogenic specialization in this species.

Aseptic tests show only pathogenic potential, which is not always realized in unsterile soil with the abundant competing microflora (Vaartaja 1968). In the tests of Hendrix and Campbell (1968) *P. sylvaticum* was only slightly virulent on three Southern pines in unsterile soil and with a low inoculum density, <20 propagules per gram of soil. With either a higher inoculum density or less competition in soil, one may expect this pathogen to cause considerable seedling losses.

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Occurrence of mottle and redleaf components of carrot motley dwarf disease in British Columbia

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Carrots showing symptoms of motley dwarf disease were found in the lower Fraser Valley in British Columbia in 1974. Incidence of the disease and of the vector aphid *Cavariella aegopodii* was low in several commercial crops examined but was high in a field experiment plot. Tests by manual and aphid inoculation showed that the affected plants contained both components of the disease, carrot mottle virus and carrot redleaf virus. This is the first record of the disease in Canada and the first evidence that both of the component viruses occur in North America.

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Les carottes présentant des symptômes de nanisme bigarré ont été découvertes en 1974 dans la vallée inférieure du Fraser, en Colombie-Britannique. La fréquence de la maladie et de son agent de transmission, le puceron *Cavariella aegopodii*, était faible dans plusieurs des cultures commerciales examinées mais elle était élevée en parcelle expérimentale. L'inoculation à la main et par puceron a révélé que les plantes atteintes hébergent les deux facteurs de la maladie, le virus de la marbrure et celui du roussissement des feuilles de la carotte. Cette maladie n'a aucun antécédent au Canada et il s'agit de la première manifestation simultanée des deux facteurs viraux en Amérique du Nord.

Motley dwarf disease of carrot (*Daucus carota* L. var. *sativa* DC.) was first described by Stubbs (1948; 1952) in Australia, where it caused serious damage to table and seed crops. It has since been reported from New Zealand (Anon. 1959), Japan (Komuro & Yamashita 1956), the U.K. (Watson 1960), Germany (Heinze 1968), and the U.S.A. (Stubbs 1956, Howell & Mink 1974). The disease is characterized by reddening or yellowing of the foliage, accompanied by stunting and loss of yield (Stubbs 1948, 1952; Watson 1960; Watson & Serjeant 1964). In seed crops the disease causes the roots of transplanted stecklings to rot, and the surviving plants yield small amounts of seed with reduced germination (Stubbs 1948).

Watson et al. (1964) showed that affected plants in the U.K. contain two persistent, aphid-borne viruses. One, called carrot redleaf virus (CRLV), is transmissible on its own by the aphid *Cavariella aegopodii* Scop., but the other, carrot mottle virus (CMotV), is transmitted only in the presence of CRLV. However, CMotV is transmissible by inoculation of sap, whereas CRLV is not. It is obvious from the descriptions given by Stubbs (1948, 1952) that both viruses were present in the Australian material, but there is no published evidence to show clearly whether the same two virus components are present in North America. This note reports that both occur in British Columbia.

Methods and results

Carrot crops on 10 farms or smallholdings in the lower Fraser Valley area of British Columbia were inspected in September 1974. Most were late-sown crops planted in late May or June and were substantially healthy, but each contained a few plants (less than 0.1%) showing symptoms typical of motley dwarf disease. These crops had been treated with diazinon or parathion against the carrot rust fly *Psila rosae* Fabr. and were almost free of aphids. In contrast, a small plot of carrots sown in June 1974 in an experimental field of the University of British Columbia (UBC) was heavily infested with *Cavariella aegopodii* and all the plants showed typical symptoms of the disease.

C. aegopodii from symptom-bearing carrots in the UBC plot were placed in groups of five on healthy carrot or coriander (*Coriandrum sativum* L.) seedlings for 1 day and then removed by fumigation with nicotine or methyl bromide. The test plants developed symptoms typical of infection with CRLV after 10 days. The carrots showed slight reddening or yellowing and noticeable retardation of growth; the corianders showed obvious yellowing, stunting, and distortion. To show whether CMotV was also present, leaves from the aphid-inoculated test plants were ground in 0.05 M phosphate buffer, pH 7.0, and the extract was used to inoculate carborundum-dusted leaves of *Chenopodium quinoa* Willd. and *Nicotiana clevelandii* Gray. *C. quinoa* showed minute necrotic local lesions after 4-7 days with no systemic symptoms; *N. clevelandii* developed systemic necrotic vein-etching after 9 days. Both symptoms are characteristic of infection with CMotV (Murant, 1974).

When a group of 40 viruliferous *C. aegopodii* were transferred daily to fresh carrot or coriander seedlings they retained ability to transmit both viruses for at least 4 days (the longest period tested). Four insects that moulted during this period transmitted both viruses

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Table 1. Host range and symptom expression of carrot mottle virus isolates

Plant species	Scottish isolate	B.C. isolate 1	B.C. isolate 2
Apium graveolens L. cv. Utah (celery)	–	–	–
Brassica pekinensis Rupr. (Chinese cabbage)	–	–	–
Brassica rapa L. (turnip)	–	–	–
Capsicum frutescens L. (pepper)	–	–	–
Chenopodium amaranticolor Coste & Reyn.	L	L	L
Chenopodium capitatum (L.) Asch.	L	L	L
Chenopodium quinoa Willd.	L	L	L
Cucumis sativus L. (cucumber)	–	–	–
Datura stramonium L.	–	–	–
Gomphrena globosa L.	–	–	–
Lycopersicon esculentum Mill. cv. Rutgers (tomato)	–	–	–
Nicotiana clevelandii Gray	LS	LS	LS
Nicotiana glutinosa L.	–	–	–
Nicotiana rustica L.	–	–	–
Nicotiana tabacum L. (tobacco)			
cv. Haranova	–	L(S)	–
cv. Havana 425	–	–	–
cv. White Burley	–	–	–
cv. Xanthi-nc	–	L(S)	–
Petunia hybrida Vilm.	–	–	–
Phaseolus vulgaris L. cv. Topcrop (French bean)	–	–	–
Vigna sinensis (Torner) Savi cv. Blackeye (cowpea)	–	–	–

L Necrotic local lesions.

S Systemic necrotic vein-etching.

(S) A few necrotic spots on upper leaves; not fully systemic.

– No symptoms.

afterwards, showing that the viruses resembled other isolates of CMotV and CRLV (Watson et al. 1964) in being persistent (circulative) in the vector. On each day of this test, the larvae that were produced were transferred to celery (*Apium graveolens* L.) to establish a culture of virus-free aphids. Celery appears immune to both viruses and is a good host for the aphid.

Sap-transmission tests and aphid-transmission tests using virus-free *C. aegopodii* showed that similar viruses were present in symptom-bearing plants from each of five of the commercial crops inspected.

Three of the sap-transmissible isolates, after inoculation into coriander plants, ceased to be transmissible by *C.*

aegopodii. This behaviour is typical of CMotV. In host range comparisons (Table 1), two of the B.C. isolates and an isolate of CMotV originally obtained from carrot in Angus, Scotland, (Murant et al. 1969) appeared almost identical. One of the B.C. isolates induced necrotic local lesions in *Nicotiana tabacum* L. cv. Xanthi-nc and *N. tabacum* cv. Haronova; the other did not. This variation is also found with U.K. isolates. In these tests none of the isolates induced symptoms in *Datura stramonium* L., *N. glutinosa* L., *Petunia hybrida* Vilm., or *Phaseolus vulgaris* L., although these species can sometimes be infected by some U.K. isolates in winter.

No virus-like particles were seen when sap from infected carrot or *N. clevelandii* was examined in the electron microscope using potassium phosphotungstate or uranyl acetate negative stains. This too is typical of CMotV and CRLV, although membrane-bound particles, which are possibly virus, have been seen in ultrathin sections of *N. clevelandii* infected with CMotV (Murant et al., 1969, 1973). No attempt was made to see whether such particles were associated with the sap-transmissible isolates from B.C.

Discussion

The symptoms produced by the B.C. carrot viruses in various test plants and their transmission in the persistent (circulative) manner by *C. aegopodii* leave no doubt that they are CMotV and CRLV. In addition, the sap-transmissible component resembled CMotV in losing aphid-transmissibility after being transmitted by inoculation of sap, However, in the absence of reliable antisera the identity of these viruses cannot be confirmed serologically.

The importance of the disease in B.C. may be slight at present because all commercial crops examined seemed substantially free from the viruses and from the aphid vector. However, the high level of infection in the U.B.C. plots suggests that the disease could be a potential problem; the low incidence of the disease in commercial crops is probably due to the general use of insecticides against carrot rust fly.

In Britain the viruses overwinter in wild umbelliferous plants, such as cow parsley, *Anthriscus sylvestris* (L.) Bernh., and wild carrot; carrot motley dwarf disease is therefore especially troublesome following mild winters when *C. aegopodii* can survive as adult aphids on wild umbellifers instead of passing the winter in the egg stage on willow (*Salix* spp.), which is immune to the viruses. In B.C., wild carrot is prevalent (Dale, 1974) and could provide an alternate host for the viruses. It also occurs in Ontario and other parts of eastern Canada and it is possible that the viruses occur there too.

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Effects of drying on the transmissibility of wheat spindle streak mosaic virus in soils from wheat fields in Ontario[1]

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Most soil samples collected in May each year, 1970 to 1973, from areas in winter wheat (*Triticum aestivum*) fields in which 90 to 100% of the plants had distinct symptoms of wheat spindle streak mosaic showed little or no infectivity to wheat test plants grown in them before they were dried. However all were infectious when tested after they had been dried in a greenhouse and kept in polyethylene bags for 4 to 5 months; at that time the percentages of test plants infected ranged from about 5% to 100% for different soils.

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La plupart des échantillons de sol prélevés en mai (de 1970 à 1973) dans les champs de blé d'hiver *Triticum aestivum* manifestant de 90 à 100% de symptômes typiques de mosaique striée n'ont pratiquement pas provoqué d'infection sur des plants de blé cultivés avant le séchage du sol. Cependant, tous les échantillons étaient infectieux après séchage en serre et conservation en sacs en polyéthylène pendant quatre à cinq mois. Au bout de ce temps, le pourcentage de blé infecté variait d'environ 5% à 100% selon l'échantillon de sol.

During annual surveys for wheat spindle streak mosaic (WSSM) in Ontario (1), diseased plants and soil samples were collected from many of the fields for tests to determine the presence of the virus (WSSMV). As noted for such collections in 1969 (4), the numbers of test plants that became infected from the soil samples from different fields varied greatly. Since highly infectious soils were desired for experiments on various aspects of transmission of the virus, selected collections were made each year from areas in which the disease was most prevalent, and these were tested to ascertain their infectivity.

The following is a report of the results of tests to verify the infectivity of soils collected in May each year, 1970 to 1973, from areas of different winter wheat fields in which all plants showed distinct symptoms of WSSM, and the effects of drying on the results achieved.

Materials and methods

The soils were dug, including the roots of diseased plants, to a depth of 8 to 10 cm, placed in polyethylene bags and kept moist until the first tests for soil infectivity were started 1 to 2 weeks later. After the samples were removed for the May test, the remainder of each collection of soil was dried thoroughly on a greenhouse bench, then stored in polyethylene bags in the greenhouse. In October, the dried soils were tested for infectivity by the same procedures and under the same growth conditions as used for the earlier tests that year.

The tests for infectivity of the soils collected in 1970, 1971, and 1972 were done by placing a 4-5 cm layer of the soil to be tested on top of a sterilized potting soil mixture (3 loam soil: 1 sand: 1 peat moss) in one 12 cm pot for each soil. Winter wheat (*Triticum aestivum* L. cv. Kent) was sown in the field soils (20 seeds per pot) and grown in growth chambers at 12°C during a 12 h light period (10,000 lux) alternating with 6°C during a 12 h dark period. Final counts for numbers of plants with symptoms were made 120 days after seeding.

In 1971, two sets of pots of each soil were tested in October, one in the regulated growth cabinets at 6-12°C, the other in a greenhouse in which the temperature fluctuated from about 4°C to 16°C, but was principally around 10°C.

Since it was found that the optimum temperature for infection from soil was 15°C and the optimum for incubation of the virus was about 10°C (2), the tests in 1973 were done by sowing Kent wheat in two 7.5-cm pots of each of the soils, keeping them at 15°C for 4 weeks, then transplanting the plants into 12-cm pots of sterile potting soil and growing them at 6-12°C for an additional 90 days.

Results and discussion

The results of soil infectivity tests showed major variations among the soils, and also between the tests done in May and October each year (Table 1). There was little or no transmission from most of the soils in 1970 and 1971 collections when tested shortly after they were collected in May and before they were dried. Transmission from these and most of the 1972 and 1973 collections was greatly increased after the soils were kept dry until October, but even then, transmission ranged from about 5% for some to 100% for other soils.

The conditions of testing also affected results. In 1971, when an additional replication of test pots was placed in

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Table 1. Transmission of wheat spindle streak mosaic virus from soil samples from fields of diseased wheat in Ontario when tested immediately after collection in May each year, and after dry storage until October

Year	Field no.*	Location	Time tests started May	October	October
1970	17	Palmyra	0/20**	3/21**	
	19	Port Crew	1/19	1/20	
	26	Colchester	0/20	9/18	
	28	Wheatley	0/19	7/21	
	40	Clinton	0/19	8/20	
	43	Bond Head	0/18	10/19	
		non-infectious soil	0/21	0/20	
1971	14	Victoria	0/20**	0/19**	13/20†
	17	St. Williams	0/20	10/20	12/17
	35	Blenheim	1/19	8/11	20/20
	42	Kingsville	1/20	3/16	14/20
	43	Port Alma	0/16	2/20	1/20
	64	Bond Head	0/19	0/17	6/20
	72	Ottawa	0/20	9/19	18/19
	73	Ottawa (mosaic-free)	0/19	0/20	0/20
1972	4	Vineland	3/14**	6/18**	
	10	Dunnville	1/14	2/18	
	14	Wallacetown	2/14	12/21	
	22	Port Alma	0/15	20/20	
	24	Port Alma	4/17	9/20	
	46	Exeter	4/19	1/18	
	61	Bond Head	0/20	14/19	
	63	Bond Head (mosaic-free)	0/20	0/21	
1973	1	Ottawa	6/62††	41/51††	
	41	Amherstburg	2/29	12/35	
	43	Port Alma	0/35	16/35	
	53	Arkona	0/38	15/35	
	59	Exeter	8/36	13/35	
	64	Clinton	0/36	25/35	
	67	Clinton	11/34	15/39	
	82	Bradford	13/32	7/38	
	92	Workworth	0/37	18/34	
		non-infectious potting soil	0/33	0/38	

* 90-100% of the plants were diseased in the areas of each field where all except the non-infectious samples were collected.

** Kent wheat grown in soils in cabinets at 12 ± 1°C during a 12 h light period (10,000 lux) and 6 ± 0.5°C during a 12 h dark period.

† Kent wheat grown in soils in a greenhouse with temperature 10 ± 6°C.

†† Kent wheat grown in two 7.5 cm pots of infectious soils at 15°C for 4 weeks then transplanted into sterile soil in 12 cm pots and grown in cabinets at 12°C during 12 h light period and 6°C during 12 h dark period.

a greenhouse in which the temperature was about 10 ± 6°C, a higher proportion of the test plants in most of the soils developed symptoms at these variable temperatures than at the more precisely regulated growth cabinet conditions with the temperature alternating regularly between 6°C and 12°C at 12 h intervals.

Although no direct comparisons were made in the tests reported here, tests done by growing wheat in infectious soil at 15°C for 4 weeks, then transplanting and growing at 6°to 12°C for symptoms to develop, as in 1973 (Table 1), generally result in higher percentages of plants developing symptoms than tests in which the

plants are grown continuously at any one daily temperature regime (2). Even with this procedure, we seldom achieve infection of all test plants even in the most highly infectious soils. Usually most plants become infected in plots of highly infectious soil at Ottawa in October and sometimes in pots of infectious soil kept outdoors through October (23). Wiese and Hooper (5), noting the high infection in pots of infectious soil kept outdoors in the fall in Michigan, reported that symptom severity and percentage of test plants infected were increased by cool and freezing temperatures. A period of such conditions, which was not included in our tests, may be necessary for maximum disease development.

There appear to be several factors affecting the transmissibility of WSSMV from soil. Although certain sequences of temperature are of major importance, moisture content and other factors may also affect the infectivity of the soil at specific times. The low infectivity of most soil samples shortly after collection from the field in May each year indicates a lack of reliability of soil tests done immediately on non-dried soil samples collected at that time. Tests done after the soil samples have been dried for a period appear more reliable. We have not determined the effects of much shorter periods of drying soils collected in May. However, other tests have indicated that keeping soil moist has a temporary suppressive effect on infectivity. At present we have no satisfactory explanation for the great differences in infectivity of soils collected from different fields in which WSSM appeared to be equally prevalent, but the explanation is probably related to the concentration or activity of the vector, and possibly to failure to provide conditions that would activate the vector and virus optimally in all the soils.

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Correction

In the article by G.J. Green entitled "Air-borne rust inoculum over western Canada in 1974", volume 55, no. 2, page 49, the last entry in column 1, Table 1 should read "1974 total".

Proceedings of the 1974 APDW workshop on crown rot of apple trees

Edited by D. L. McIntosh[1]

At a workshop sponsored by the Apple and Pear Disease Workers and held at Summerland, British Columbia, 16-17 August 1974, participants from the USA, UK, Poland, and Canada considered problems associated with collar rot of apple trees incited chiefly by *Phytophthora cactorum*. Topics included pathogens, symptomatology, methods of isolating *P. cactorum*, evaluation of resistance, pathogenic variation in *P. cactorum*, cultural practices and predisposition, control, ecological studies, and areas requiring further research.

Can. Plant Dis. Surv. 55:109–116. 1975

Les 16 et 17 août ont eu lieu à Summerland en Colombie-Britannique des journées d'étude parrainées par les phytopathologistes de la pomme et de la poire. Les représentants des Etats-Unis, de la Grande-Bretagne, de la Pologne et du Canada qui y participaient ont étudié les problèmes relatifs au mildiou du collet chez le pommier dont le principal agent causal est *Phytophthora cactorum*. On a étudié en particulier la nature de l'agent pathogène, la symptomotologie, méthodes d'isolement de *P. cactorum*, l'évaluation du degré de résistance, la variation de la pathogénicité chez *P. cactorum*, les méthodes de culture et la prédisposition des arbres à la maladie, les moyens de lutte, l'écologie. On a également établi les domaines nécessitant des recherches plus poussées.

The Apple and Pear Disease Workers (APDW), with financial support from the Cooperative State Research Service of the United States Department of Agriculture, convened a Workshop for discussion of the crown rot disease of apple trees at the Agriculture Canada Research Station, Summerland, British Columbia, August 16-17, 1974. The following scientists participated: *H. S. Aldwinckle*, New York State Agricultural Experiment Station, Geneva, N.Y. 14456; *Anne M. Alvarez*, University of Hawaii, Honolulu, Hawaii 96822; *S. V. Beer*, Cornell University, Ithaca, N.Y. 14850; *Z. Borecki*, Institute of Pomology, 96-100 Skierniewice, Poland; *R. F. Carlson*, Michigan State University, East Lansing, Michigan 48824; *C. N. Clayton*, North Carolina State University, Raleigh, N.C. 27607; *R. P. Covey, Jr.*, Tree Fruit Research Center, Wenatchee, Wash. 98801; *J. N. Cummins*, New York State Agricultural Experiment Station, Geneva, N.Y. 14456; *H. J. Dubin*, University of Maine, Orono, Maine 04473; *C. O. Gourley*, Research Station, Agriculture Canada, Kentville, N.S. B4N 1J5; *H. W. Guengerich*, Stark Bros. Nurseries, Louisiana, Mo. 63353; *K. D. Hickey*, Winchester Fruit Research Laboratory, Winchester, Va. 22601; *B. F. Janson*, Ohio State University, Columbus, Ohio 43210; *A. L. Jones*, Michigan State University, East Lansing, Michigan 48824; *A. J. Julis*, North Carolina State University, Raleigh, N.C. 27607; *I. C. MacSwan*, Oregon State University, Corvallis, Ore. 97331; *R. C. McCrum*, University of Maine, Orono, Maine 04473; *C. D. McKeen*, Agriculture Canada, Central Experimental Farm, Ottawa, K1A 0C6; *J. L. McIntyre*, Connecticut Agricultural Experiment Station, New Haven, Conn. 06504; *J. Mircetich*, ARS, USDA, University of California, Davis, Calif. 95616; *R. W. Miller*, Clemson University, Clemson, S.C. 29631; *J. E. Mitchell*, University of Wisconsin, Madison, Wisc. 53706; *W. J. Moller*, University of California, Davis, Calif. 95616; *P. C. Pecknold*, Purdue University, W. Lafayette, Ind. 47907; *D. H. Petersen*, Pennsylvania State University, University Park, Pa. 16802; *R. C. Pearson*, Hudson Valley Laboratory, Highland, N.Y. 12528; *R. M. Rosher*, Agriculture Canada Research Station, Summerland, B. C., V0H 1Z0; *G. W. F. Sewell*, E. Malling Research Station, Nr. Maidstone, Kent, England; *E. Shabi*, University of Missouri, Columbia, Mo. 65201.

Topics dealt with are listed below in the order in which they were discussed. The problems encountered by those who have some involvement with crown rot of apple studies or diagnosis were described. Suggestions were offered for overcoming some of them. Many questions were asked about the host-parasite relationship, about the epidemiology of the disease, about its recognition, about the techniques required for manipulation of the pathogen, and about a reliable method of evaluating rootstock resistance. Not all of them could be answered with certainty. The statements which follow were gleaned by the editor from the discussions and from written submissions. Wherever possible the source of information is supplied in brackets following the statement, so readers who wish more detail on the subject can contact the individual directly.

The pathogens

Phytophthora cactorum (Lebert & Cohn) Schroet. has

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Figure 1. Root crown affected by crown rot. Note darker color of infected bark where main roots join the trunk.

been considered the incitant of collar rot and crown rot of apple trees but other pythiaceous fungi also may cause bark rotting. In Poland, collar rot is caused primarily by *Phytophthora cactorum*, less frequently by *Phytophthora syringae* and occasionally by *Pythium* spp., probably *Pythium ultimum*. Symptoms similar to collar rot will also develop after artificial inoculation with *Phytophthora megasperma, P. cryptogea, P. cinnamomi, P. drechsleri*, and *P. citricola*. An epiphytotic of collar rot disease on 6-year old apple trees was attributed to infection by *Pythium ultimum*. It developed as a result of damage to the trunk bark while digging around the trees in the autumn (*Borecki*). In California, several *Phytophthora* spp. have been isolated repeatedly from apple trees affected with crown and root rot from 11 orchards surveyed in Santa Cruz and Sonoma counties. Three unidentified *Phytophthora* spp. as well as *P. megasperma, P. cambivora*, and *P. drechsleri* were recovered from trees 1-18 years old. Three or four different *Phytophthora* species were often recovered from the same orchard and occasionally from the same tree. *P. cactorum* was not isolated from any of the 57 apple trees sampled in the survey, although this species has recently been isolated from crown rot-affected lilac, live oak, and walnut trees elsewhere in California (*Mircetich*). Studies in Wisconsin reveal that *Cytinostroma galactinum* (formerly *Corticium galactinum*) was the cause of a decline of apple stocks in one orchard (*Mitchell*).

Symptomatology

Phytophthora cactorum, causes a *collar rot*, i.e. a rot of the bark of the trunk at or above ground level, as well as a *crown rot*, i.e. rot of the bark around the root crown below ground level. Collar rot may affect the scion variety, or the rootstock if the union is well above ground. Crown rot originates in the rootstock where the main roots join the trunk.

During the 1950s in England, when most orchard trees were low-worked on M2 or M9 rootstocks, *Phytophthora* problems were almost entirely associated with infections of scion variety bark and tissues (i.e. collar rot) at or near soil level. The most susceptible cultivar was Cox's Orange Pippin, and infections of rootstock tissues below soil level (i.e. crown rot) were relatively rare. More recently, extensive orchard plantings have been made using trees worked onto Malling-Merton rootstocks, often with the graft-unions raised above soil level to avoid scion variety infection. With these changes the *Phytophthora* problem has also changed and many disease outbreaks now involve infections of rootstock tissues below soil level (i.e. crown rot). Foliar symptoms of crown rot (general chlorosis often with red coloration of veins and margins) frequently occur long before

Figure 2. Marbled appearance of lesion in which *Phytophthora cactorum* is active. Note sharply defined margin between infected and healthy-appearing tissue.

rootstock stem lesions are apparent at or near soil level. This feature, together with examinations of lifted trees, indicate that most infections initially occur at the bases of the main roots (*Sewell*).

In British Columbia, crown rot disease is characterized by a rot of the bark below ground where the main roots join the trunk (Fig. 1). Frequently the rot progresses upward to the stock-scion union. Often the bark of lateral roots is not affected except close to the trunk. Recently invaded tissue, or an active lesion, is several shades of tan to reddish brown in a marbled pattern. The margin between diseased and healthy-appearing tissue is sharply defined (Fig. 2). Often the appearance of the bark surface over an active lesion does not indicate the condition of the tissue beneath. Usually young vigorous-looking trees which are severely damaged by infection during the growing season, do not show obvious symptoms of distress above ground until mid-August or September when leaves, or initially just midribs, take on a purplish-red color. This coloration intensifies with time until diseased trees can be readily distinguished from others in the planting whose leaves still are green. Premature purpling of the foliage late in the season is not always indicative of crown rot infection. In this region Golden Delicious frequently develops such foliage color without the involvement of crown rot infection. However trees with such coloration are easily spotted in the planting and examination of their bases below ground more often than not reveals a severe crown rot infection. By the time such foliage symptoms appear so much of the trunk circumference is diseased that trees either die or do not grow satisfactorily for several years thereafter.

If an infection affecting an appreciable portion of the trunk below ground is not detected during the season in which it has developed, growth on that side will be delayed the following season. Leaves will be small, fruit will be highly colored, and terminal growth poor. Examination of the bark below ground reveals tissue dead to the cambium but by this time other secondary organisms have been active and the probable cause is not so evident (*McIntosh*).

The symptoms first observed in the spring by South Carolina growers are a lighter green coloration of foliage and a subsequent lack of vigorous growth. Sometimes these trees will set a fruit crop and produce some small fruit but other times they die. When symptoms appear in early or midsummer, they are characterized by a slow rate of growth, a change in color to light green which is often accompanied by yellowing. The keen observer develops an "eye' for affected trees. Occasionally there is an upward folding of the leaves and a discoloration (pink or reddish) of the leaf lamina and midrib from exposure to sunlight. One- to 5-year-old trees suspected of being severely infected often feel loose or poorly anchored when the trunks are grasped and shaken. Trees with a heavy crop may just topple.

Bark discoloration usually extends no more than 25 cm above the soil, mostly in dark brown streaks. Most discoloration and girdling occurs at or below the soil line. Some is found in the primary root system. During dry weather bark on cankered areas dries out and cracks, delineating the cankered area. Callus tissue may or may not encircle the previously infected tissue. Problems which can be mistaken for collar rot if a careful examination is not made are cold injury, mouse damage, southern stem blight [*Sclerotium rolfsii*], occasionally borer damage, and fire blight on susceptible stocks (*Miller*).

In Virginia, the most obvious symptom of crown rot is a partial or complete girdling of the trunk at or near the ground line. Generally the entire underground portion of the stem is watersoaked and brown and the necrotic area usually extends upward to the graft union. Infection may extend into the scion above the union in trees planted below the graft union. On newly infected trees the roots may appear normal except for 5 to 15 cm next to their point of attachment to the trunk. Such trees often show first symptoms near the soil line during late summer or early winter and by the next spring may be completely girdled. Trees that have made normal to above normal growth and have borne one or two crops are affected more than juvenile trees 2 to 3 years old. Affected trees which are completely girdled by spring show poor terminal growth accompanied by a pronounced off-color

in the foliage in early September. These trees usually are completely girdled by December and die the following growing season (*Hickey*).

In Poland, lesions in the lower part of the tree trunk are extensive, reaching a height of 40 cm. Most damaging are lesions girdling the trunk at rootstock-scion junction. Collar rot, in the form of a lesion girdling the stem, is most frequent on the semi-dwarf rootstocks M7, MM106, and others of the MM series. The regenerative ability of the tree has an important influence on the symptoms of collar rot. On artificially inoculated apple trees, the wounds often reach dimensions of more than 12 mm but this necrosis does not progress thereafter and the wound gradually heals. A completely different type of symptom occurs on susceptible varieties of pears, particularly on Russian clones. In their case, the disease involves the entire inoculated shoots, passing in to older branches and the tree trunk. This causes dieback of the entire tree in a single season, similar to that caused by fire blight [*Erwinia amylovora*] (*Borecki*).

There was some discussion of how to distinguish between the symptoms of crown rot, winter injury, and ''wet feet'. Lesions caused by recent activity of *P. cactorum* are not a problem. The characteristic tan to red brown mottling of the affected bark tissue is diagnostic (Fig. 2). Problems arise the year after the pathogen has been active, when above ground symptoms of poor leaf and shoot development are seen early in the season. At this time, after the dead bark has been invaded by secondary organisms, the actual cause of tree death may be difficult to ascertain. The following observations may be helpful in making diagnoses:

1. Winter injury is more likely to affect the trunk at and above ground, while crown rot (as opposed to collar rot) affects tissue below ground.

2. After winter temperatures low enough to cause trunk damage, it is likely that there will be a higher percentage of trees affected in individual plantings than usually are damaged by crown rot in a single season. Five to 10 percent affected in any one season could be attributed to crown rot. Higher percentages would be expected for winter injury.

3. Bark injured by sub-zero temperatures will, in time, separate readily from the underlying wood. This is not characteristic of tissue affected by crown rot.

4. Winter injury often affects only one side of the tree, i.e. that facing the southwest.

5. Much of the wood is discolored in trees damaged by winter. This is not always the case in trees damaged by crown rot when they are examined soon after symptoms of delayed development appear.

Mechanics of isolating Phytophthora cactorum

Sewell and McIntosh stated that the most important factor in the successful isolation of *P. cactorum* is the selection of cortical tissues from the margins of *active-appearing* lesions where infected discolored tissues border healthy-appearing tissues. Strips of bark about 5 mm wide are cut lengthwise through the margin to the depth of the cambium. Chances of recovery are enhanced if the bark pieces are irrigated in tap water for 2-3 days. At the end of this period they can be either cut into 5 mm lengths and transferred to cornmeal agar or examined under a wide-field microscope for development of the characteristic *Phytophthora* mycelium from the edges of the bark. Those bearing *Phytophthora*-like hyphae can be inserted into apple fruits and the wounds sealed with petroleum jelly or adhesive tape. Firm brown lesions develop in the fruit in 4-8 days. Tissue transferred aseptically from the margins of these lesions yields pure cultures of *Phytophthora*.

Isolation from older apparently inactive lesions is much more uncertain, but the same procedure should be followed. However it likely will be advantageous to add to the agar medium compounds which will suppress unwanted fast-growing secondary invaders and bacteria. Good success has been achieved in isolating *P. cactorum* from both active and older lesions by washing infected tissue 30-60 min in running tap water, placing tissue on Difco cornmeal agar containing 100 ppm Nystatin + 200 ppm Vancocin HC1 + 100 ppm quintozene (PCNB). Transferring mycelium growing from this tissue after 3-5 days to plates of the same medium produces pure cultures of *P. cactorum* (*McIntyre*). For a review of the literature on selective media see Tsao, P.H. 1970. Selective media for isolation of pathogenic fungi. *Annu. Rev. Phytopathol.* 8:157-186.

Does treating the bark tissue with reducing agents or antioxidants increase the chances of successful isolation? Hickey and Mircetich had some success using ascorbic acid but results were not consistent. Does the kind of fruit to be injected with bark or soil have any effect on the results? Borecki stated that some primitive apple types available in Poland were much more useful than dessert varieties. Golden Delicious was not too useful in Argentina (*Alvarez*). Green varieties are preferable to red skinned fruits (*McIntosh*). Sewell compared 15 different varieties in England for this purpose and found no differences.

Evaluation of resistance in rootstocks and scions

Many people have been frustrated and puzzled by their lack of success in producing infection through artificial inoculation. This lack of consistency in response to inoculation undoubtedly has been responsible to some extent for the differing resistance ratings given by individual investigators to the same rootstock.

Aldwinckle has used a technique for evaluating resistance in rootstocks and scions which is similar to one described by Borecki and Millikan (*Phytopathology* 59:247, 1969). Dormant 1-year wood is collected in late winter; sticks are cut into 60 mm internodal lengths; the epidermal layer is removed to expose phloem; four sticks are placed in a petri dish with moistened filter paper to prevent desiccation; a 4 mm plug of V8-juice agar bearing actively growing mycelium is placed in the

center of the upper surface of each piece; after incubation for 4 days at about 24°C the length of brown lesions is measured.

Sewell and Borecki agreed that apple scion cultivars react more uniformly to artificial inoculation than do apple rootstocks, and the resistance of scion tissues can be evaluated readily by inoculating stems using a cork borer to remove bark tissue and allow the placement of an agar disc of mycelium.

Comparative studies of varietal resistance among apple cultivars are valid only if the varieties are worked on similar, preferably very vigorous, rootstocks and if all inoculations are made near the "pink bud' stage of development. Lesions in Cox stems can be up to eight times larger with a vigorous stock (MM104) than with a dwarfing stock (M9). Resistance of scion varieties to artificial inoculation is least during the period from budburst to full blossom; resistance increases substantially with the commencement of extension growth (*Sewell*).

For evaluation of resistance of rootstocks, Borecki compared six different methods and found the most reliable to be inoculation of budded rootstocks, at least 4 years old, under field conditions. Agar discs of mycelium were satisfactory as inoculum. Evaluation of disease development was not made until at least 1 year after inoculation. Inoculation of young unbudded rootstocks under field conditions was the least reliable method. Borecki found considerable variation in the reaction to inoculation during the season. One main peak of susceptibility occurred during flowering, and a second less conspicuous peak occurred in midsummer. He suggested that for rootstock resistance evaluation the stock should be budded with a susceptible scion. If the scion is inoculated the pathogen will progress from the scion into the rootstock, and the size of the rootstock lesion which develops can be measured.

From Sewell's experience it seems that no inoculation method yet tried has given results which reflect reliably the resistance of rootstocks under orchard conditions. Direct inoculation of young rootstock stem tissues of all clones resulted in only small lesions which healed rapidly. This method gave little or no indication of the susceptibility of MM104 or M106 which has been revealed by their field behavior.

Sewell has experimented with zoospore inoculum and has found that young stems of MM104 are resistant to multipoint inoculation (by pin prick wounding under zoospore suspensions); young plants of unworked rootstock clones also responded inconsistently to zoospore soil drench applications. The latter method generally resulted in wilt, due to destruction of the fine root system, followed by gradual but complete recovery. Secondarily thickened *root* tissues of more mature plants may be less resistant than rootstock *stem* tissues. It is possible that environmental factors associated with host predisposition may be of prime importance in determining field behavior of the rootstock clones. Certainly at present, reliable assessments of rootstock resistance can only be derived from field experience. This fact emphasizes the necessity for full confirmation of the causal agent of root and crown disorders, i.e. whether it is winter injury, mechanical damage, or some other pathogen.

At the conclusion of this section there was some speculation about factors which might affect the resistance displayed by artificially inoculated stocks. Cummins was concerned with stress factors, and whether waterlogging, drought, winter injury, or the onset of fruiting might lower resistance or somehow predispose trees to invasion. The question arose as to whether winter injury plays a part in the development of crown rot. McIntosh stated that his records of crown rot incidence did not show an obvious increase in losses to the disease after particularly low winter temperatures.

Rootstock and scion resistance ratings

Rootstocks considered susceptible in some regions are believed resistant in others. What are the reasons for such differences? Are the differences real or the result of differences in methods of assessment, or are they due to factors not yet defined? While there were no simple authoritative answers to these questions, there seemed agreement that MM104 and MM106 are likely to suffer the most damage from crown rot (Table 1). MM111 has been troublesome in British Columbia but not in Michigan or Pennsylvania. M7 has proven susceptible in B.C., South Carolina, and Poland but not in Michigan. Robusta 5 is not being planted in Michigan because of *Phytophthora* problems. Stocks which seem to have suffered least from crown rot include M2, M4, M9, M26, and Antonovka seedlings.

Pathogenic variation in P. cactorum

Pathogenic variation has been proposed as a reason for differences in resistance of individual rootstock clones reported from various regions. Is there any convincing evidence for such pathogenic variation among isolates of *P. cactorum*?

Aldwinckle and his colleagues at Geneva, N.Y., have obtained such evidence by mass inoculating with zoospores apple seedlings grown in flats and also by inoculating excised twigs with agar plugs bearing mycelium. They were able to divide the isolates used into four groups by the way each interacted with six test cultivars in the twig test. Within these groups there were differences in the level of pathogenicity to all cultivars ("aggressiveness" sensu van der Plank). A larger number of test cultivars might allow further subdivision of the four groups. Statistically significant differential interactions were found between 31 cultivars and 3 *P. cactorum* isolates that had not been differentiated by the 6 test cultivars. The four pathogenicity groups do not appear to have been determined by host or area of origin. Representatives of each group had been isolated from apple in Missouri.

Comparison of pathogenicity among *P. cactorum* isolates in Poland and among other species showed that all of

Table 1. Survey of APDW collar/crown rot workshop participants August 16-17, 1974: Regional experience with *Phytophthora* on common apple rootstocks

Respondent	Geographic area covered by response	Rootstocks which are no longer planted or should not be planted because of *Phytophthora* problems	Rootstocks which occasionally have *Phytophthora* problems	Rootstocks which never have *Phytophthora* problems
H.S. Aldwinckle	New York	Maybe MM106	MM106	
A.M. Alvarez	Rio Negro Argentina		MM104	
Z. Borecki	Poland	M7, MM106, MM104	M2, Alnarp 2	M9, Antonovka sdlg.
R.F. Carlson	Michigan	MM109, MM104, Robusta 5	MM106, Alnarp 2, M26	M7, M8, MM111
C.N. Clayton	North Carolina	MM106	M7	
R. Covey	Washington	MM104, MM106	Sdlg, M7, M9	M26
H.J. Dubin	Maine	MM104		
C. Gourley	Nova Scotia		MM106	
H.W. Guengerich	U.S.		All rootstocks including sdlg.	None
K.D. Hickey	Virginia (Shenandoah Valley)	MM104, MM111? , MM106	Std. sdlg., M7, M2	Std. sdlgs., M9
B.F. Janson	Ohio		MM106, M26, M7	M9
A.L. Jones	Michigan	MM104	MM106, sdlg.	
I.C. MacSwan	Oregon	MM104		
D.L. McIntosh	British Columbia	MM104, MM106, M7, MM111	M2, M26, M9, M4	
R.W. Miller	South Carolina	MM106, M7	MM111	
J.E. Mitchell	Wisconsin	MM104?	MM106, M7	
W.J. Moller	California	Not yet clear	MM106, MM104, std. sdlg.	None
P.C. Pecknold	Indiana	MM106, MM104		
D.H. Petersen	Pennsylvania	MM104, MM106	M26, M2, M7	EM9, MM111
G.W.F. Swell	Kent, England	MM104	MM106 locally severe MM109, less common	M2, M9, MM111 M26

them caused necrotic lesions on trunks of apple trees, but the various isolates exhibited distinct differences in their degree of pathogenicity. Among 63 isolates of *P. cactorum*, 6 of Polish origin and 4 of USA origin were the most pathogenic. One isolate of *P. citricola* also was highly pathogenic. Isolates of *P. cactorum* differ widely in the appearance of the culture on exposure to high temperatures, in the intensity of sporulation, in their pathogenicity to different hosts, and in the structure of the sexual and asexual spore forms. It is difficult on the basis of these criteria to identify specific biotypes. After culturing several score *P. cactorum* isolates over the previous 6 years and isolating the pathogen every 6 months from plant tissue, pathogenicity proved a relatively stable character of the fungus. The most pathogenic isolates used in the early period, 1968-69, of the experiments have retained their characteristic pathogenicity (*Borecki*).

McIntosh pointed out the difficulties of obtaining uniform woody host material for studies of pathogenic variation; and the desirability of having a suitable host range for this purpose; he also suggested that initially some use might be made of herbaceous annuals in determining differences in pathogenicity among isolates of *P. cactorum*. In this connection, the recent report from Wisconsin on the susceptibility of safflower was interesting.

Workers interested in studying pathogenic variability should note that isolates of *P. cactorum* are available from the sources listed in Table 2.

Cultural practices and predisposition to infection

Several participants postulated that excess soil moisture favored development of the disease. Incidence of crown and root rot in some apple-growing areas of California reached epidemic proportions following unusually high winter and early spring rainfall in 1973 and 1974 (*Mircetich*). In Virginia the disorder is commonly found in old orchard sites or in low areas of orchards having heavy, poorly drained soils (*Hickey*).

Table 2. Survey of APDW collar/crown rot workshop participants, August 16-17, 1974: Isolates of *Phytophthora* available

Name	Geographical source of isolate	Number of *Phytophthora cactorum* isolates available and source
H.S. Aldwinckle	New York	2
A.M. Alvarez	Rio Negro, Argentina	3-4 (Apple)
Z. Borecki	Poland	63
C.N. Clayton	North Carolina	10
R. Covey	Washington	8 (Apple, pear, irrigation water)
C.O. Gourley	Nova Scotia	2 (Apple)
K.D. Hickey	Virginia (Shenandoah Valley)	2
B.F. Janson	Ohio	10+
A.L. Jones	Michigan	4-8
I.C. MacSwan	Oregon	2 (Apple)
D.L. McIntosh	British Columbia	Several
J.L. McIntyre	Connecticut	2
J. Mircetich	California	Some (From hosts other than apple)
J.E. Mitchell	Wisconsin	50 ±
W.J. Moller	California	Several: *P. cactorum* *P. megasperma* *P. drechsleri* + 3-4 *Pythium* sp.
G.W.F. Sewell	Kent, England	40 ±

Several workers have observed that newly planted trees placed in holes made by an auger frequently settle an inch or two below the original level as the soil is wetted and compacted. They believe that the saucer so created collects water and provides favorable conditions for infection by *P. cactorum*. Mounding newly set trees around the trunk with sand was suggested as a means of correcting this situation and of preventing wind-rocking. As sand trickled down around the trunk in spaces created by wind-rocking, anchorage would be improved.

Planting of trees on mounds was reported to be beneficial in preventing or reducing losses from *Phytophthora* infection in California and Australia.

There was much speculation on whether injury to trunk and root systems from low winter temperatures made trees more vulnerable to crown rot infection. It was postulated that tree losses may be attributed sometimes to crown rot when actually winter injury was the cause.

The only cultural practices correlated with crown rot occurrence in British Columbia were those favoring extreme vigor in young trees. Serious losses to crown rot have occurred on sandy-gravelly loam slopes where drainage appeared to be satisfactory, and more problems have been encountered on light than on heavy soils (*McIntosh*).

It was obvious from the discussion that there is a great deal yet to be learned about the epidemiology of this disease.

Control

Unfortunately there seems to be no single simple satisfactory measure by which the disease can be prevented or cured. Several participants have explored the effects of applying chemicals to soil and to affected trees, but in none of the trials were the results dramatic enough for the treatments to be recommended. McIntosh mentioned the need for a suitable means of evaluating the results of applying chemicals either to the soil or to the tree trunks. Some trees may be infected at the time of treatment, but not display any signs of the infection above ground. Allowance must be made for this situation, either by examing trees before treatment, or by repeating the treatment annually for several years.

Planting trees on mounds above the grade of the orchard has been found beneficial but growers seem reluctant to

change their planting methods unless they have learned from experience that it is worthwhile.

Manipulation of the pathogen in ecological studies

Most of the information discussed under this heading has already been published in articles by Sewell, Sneh, and Meyer in 1973-74.

Borecki described some endogenous factors in bark tissue which affect the growth of *P. cactorum* in vitro. Biologically active compounds are extractable from phloem-cambium tissues with 0.01 M NaOH plus 2% Na_2SO_3. Extracts from the resistant apple tissue either inhibit or have no effect on fungus growth, while those from susceptible tissues stimulate growth. Spectrophotometric studies of the extract obtained from inner bark tissue of apple trees indicate the presence of three biologically active fractions. Phloridzin content was thought initially to influence resistance of apple trees to collar rot but the amount in a resistant variety is not sufficient to inhibit in vitro growth of *P. cactorum*.

The S fraction has a stimulatory effect on the in vitro growth of *P. cactorum*. It has been identified by Missouri workers as a 4-5 sRNA. Concentrations between 0.4 and 2.0 ng sRNA/ml result in a maximum stimulation of *P. cactorum*. This effect is specific for apple sRNA.

Petersen suggested there may be mechanical as well as chemical resistance to *P. cactorum* colonization.

Cummins suggested there may be some value in examining the effect of exogenous growth regulators on resistance of apple to *P. cactorum*.

New candidate rootstocks

Probably the only satisfactory and permanent solution to the crown rot problem will be found in resistant rootstocks which are also horticulturally suitable for the regions where apples are grown. What candidates are there? Where are they being developed?

During the period 1958-1970 in Poland, M4 and M9 were crossed with the hardy apple cultivar Antonovka to develop hardy rootstocks resistant to frost damage and to *Phytophthora*. First selections among seedlings were made at the four-leaf stage by watering plants with zoospore suspensions. Whether or not the survivors are immune to crown rot is not known yet. Stock numbers P-I, P-II, P-XVI, and P-XXII have resistance comparable to or greater than that of M9. P-II and P-XXII are more tolerant of low winter temperatures than M9 (*Borecki*).

Carlson described a series of MAC rootstocks that have been selected in Michigan. They were derived from open-pollinated seed gathered in a block that contained M1 to M16, Robusta 5, and Alnarp 2. The rootstocks display different degrees of dwarfing, precocity, and ease in rooting. Currently they are being evaluated by Cummins at Geneva, N.Y., for resistance to woolly aphids, fire blight, and *P. cactorum*.

Cummins and colleagues at Geneva, N.Y., are also seeking rootstocks to replace specific targets, e.g. M9, M26, MM106. Breeders at East Malling and at Skierniewice have similar objectives. They are quite concerned about *Phytophthora* resistance and are exposing seedlings to zoospore inoculum at an early age. This is effective in eliminating large numbers of candidate seedlings but there is some uncertainty about whether resistance displayed at this age will be present in the trees later in the orchard. Progenies of Dolgo crab have a high percentage of survival in this zoospore test. Similar results occur with progenies of a cultivar from Japan designated 613 in N.Y. Very few progeny of M7 x MM106 survive. Parents which are very hardy seem to have progeny with high resistance to *P. cactorum*.

In response to a question of what could be used to replace MM104 and MM106 lost to crown rot, M9 was suggested as a stock with resistance to *Phytophthora* and adaptability to wet soil situations. For greater vigor than M9, M2 or M4 may be suitable.

Is there any potential for an interstem tree using rootstocks that are resistant to *Phytophthora*? With a multipiece tree the chances of troubles developing are increased because of possible sensitivity to viruses in one of the components.

Does it help to inarch rootstocks that have been severely damaged by crown rot? The consensus seemed to be that trees would not recover their vigor for several years at least, and it would be better to replace trees less than say, 10 years old.

Aspects on which research is warranted

It was obvious from the discussions that we are lacking much knowledge of the disease and the factors which influence its development. Participants offered the following suggestions for future research:

1. Ecology of the pathogen in its environment and its variability. A method is needed by which quantitative data on soil inoculum levels can be obtained.
2. How infection occurs and the histopathology of lesion development.
3. Effect on infection of soil water content, different methods of irrigation, and other cultural practices.
4. Elucidation of the mechanism of resistance, whether it be chemical or physical, so that breeders may be able to make use of the information in their breeding programs for resistance. A prerequisite of course is a reliable method of evaluating resistance.
5. Some means of protecting susceptible rootstocks that have been planted, either biological, eg. by soil amendments, or by recognizing and exploiting natural antagonisms to the pathogen.
6. Elucidation of factors that predispose trees to infection, and the effects of such factors on rootstock susceptibility.

Recommandations aux auteurs

Les articles et les communiqués sont publiés en anglais ou en français. Les manuscrits (l'original et une copie) et toute le correspondance qui s'y rapporte doivent être envoyées au Rédacteur M. W. L. Seaman, à la Station de recherches d'Ottawa, ministère de l'Agriculture du Canada, Ottawa (Ontario) K1A 0C6.

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

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

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

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

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

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

Instructions to authors

Articles and brief notes are published in English or French. Manuscripts (original and one copy) and all correspondence should be addressed to the Editor, Dr. W. L. Seaman, Ottawa Research Station, Agriculture Canada, Ottawa, Ontario K1A 0C6.

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

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

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

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

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

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