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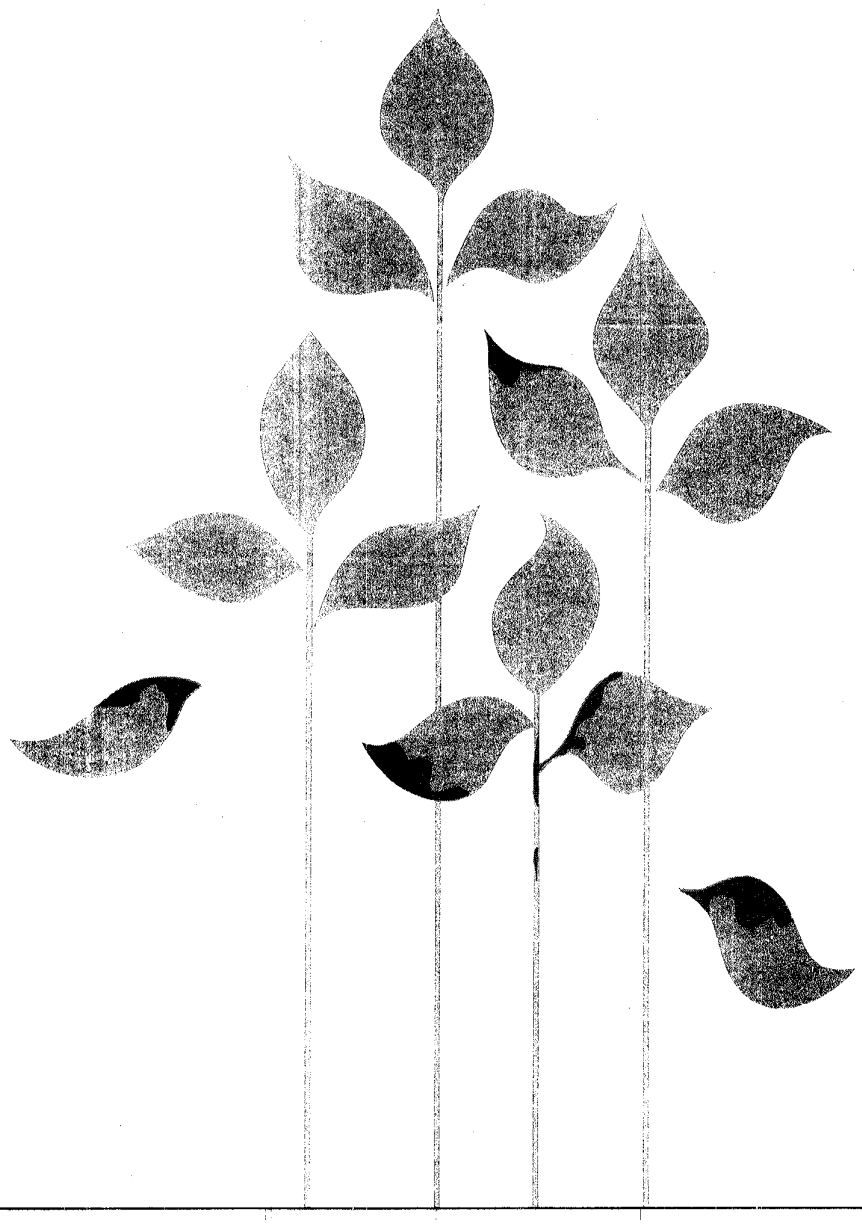

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Contents

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.

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

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Myrothecium roridum, a potential pathogen of rapeseed and mustard in Alberta

J.P. Tewari and W.P. Skoropad[1]

Myrothecium roridum was isolated from the seed of yellow marsh cress (*Rorippa islandica*) collected from near Legal, Alberta. It was inoculated on some cultivars of rapeseed and mustard commercially grown in Alberta and found to be pathogenic. Differences in susceptibility between various cultivars were noted. Many cruciferous weeds found in central Alberta are also susceptible. Although, the disease has so far not been found occurring naturally in the fields, it is a potential pathogen of rapeseed and mustard in Alberta. Some hitherto unreported features of *M. roridum*, as revealed by light and scanning electron microscopy, are reported.

Can. Plant Dis. Surv. 57: 37-41. 1977

On a isolé *Myrothecium roridum* de la graine du cresson des marais (*Rorippa islandica*) récolté près de Legal (Alberta). Le champignon a été inoculé à certains cultivars de colza et de moutarde cultivés commercialement dans la province et s'est révélé pathogène. On a observé une différence de sensibilité entre les divers cultivars. Beaucoup de mauvaises herbes de la famille des Crucifères répandues dans le centre de la province sont également sensibles. Bien que la maladie n'ait pas encore été observée à l'état naturel dans les champs, c'est un agent pathogène potentiel du colza et de la moutarde en Alberta. L'auteur mentionne certaines caractéristiques jusqu'ici non signalées de *M. roridum*, révélées par la microscopie optique et électronique.

Myrothecium roridum Tode ex Fr. is plurivorous and is widespread in temperate and tropical regions of the world (9). This fungus is strongly cellulolytic and produces mycotoxins called trichothecenes, which are capable of causing disease and death in animals (7,9).

In 1975, we isolated *M. roridum* from the seeds of marsh yellow cress, *Rorippa islandica* (Oeder) Borbas. This fungus has a wide host range including some crucifers (2,4,8,9). Since *M. roridum* is present in the environment in Alberta, a study was undertaken to determine if it could parasitize cultivars of rapeseed and mustard grown in this area, and also some cruciferous weeds common in central Alberta.

Materials and methods

Myrothecium roridum was isolated from the seeds of yellow marsh cress collected on 20, August 1975 from near Legal, Alberta. It was grown on potato dextrose agar (Difco) at 25°C. Subcultures have been deposited at the Commonwealth Mycological Institute, Kew, England (I.M.I.No. 204824) and at the National Mycological Herbarium, Agriculture Canada, Ottawa (DAOM 164769). Yellow marsh cress is a cruciferous weed native to Canada and occurs in all provinces usually in damp sites (3).

Plants were inoculated with *M. roridum* in two different ways, a) Detached leaves of Torch (*Brassica campestris* L., Polish rapeseed), Midas (*B. napus* L., Argentine rape), and Accession No. 311726 (*B. juncea* (L.) Coss, oriental mustard, Regional Plant Introduction Station, Ames, Iowa, from Poland, henceforth called the Iowa line) were spot inoculated with a conidial suspension (approx. 100,000 conidia/ml) and kept in moist chambers in petri dishes at room temperature; b) The spore suspension was sprayed on the plants in the greenhouse and in the field at the University of Alberta Farm. The cultivars screened were Midas, Tower (*B. napus*), Lethbridge (LB) 22A (*B. juncea*), Torch, R-500 (*B. campestris*), and Yellow 2 (*B. hirta* Moench). The cruciferous weeds were inoculated only in the greenhouse. At first, difficulty was encountered in obtaining good germination of the seeds of some of the weeds. Consequently, based on the work of Corns (5) on dormancy of the seeds in wild mustard and stinkweed, seeds of the weeds collected from different areas in central Alberta were routinely soaked for 24 h in 1000 p.p.m. aqueous solution of the potassium salt of gibberellic acid before sowing. The cruciferous weeds screened were common peppergrass (*Lepidium densiflorum* Schrad.), flixweed (*Descurainia sophia* (L.) Webb), Indian mustard (*B. juncea*), shepherds' purse (*Capsella bursa - pastoris* (L.) Medic.), stinkweed (*Thlaspi arvense* L.), wormseed mustard (*Erysimum cheiranthoides* L.) and yellow marsh cress (*Rorippa islandica*).

Morphology of the fungus on the leaves of Torch was investigated by light microscopy using a Leitz Wetzlar microscope equipped with an Ultropak incident light illuminator, and by scanning electron microscopy (SEM). The sporulating material was fixed for SEM in three different ways: (a) The material was fixed overnight with osmium tetroxide vapour without any disturbance to the

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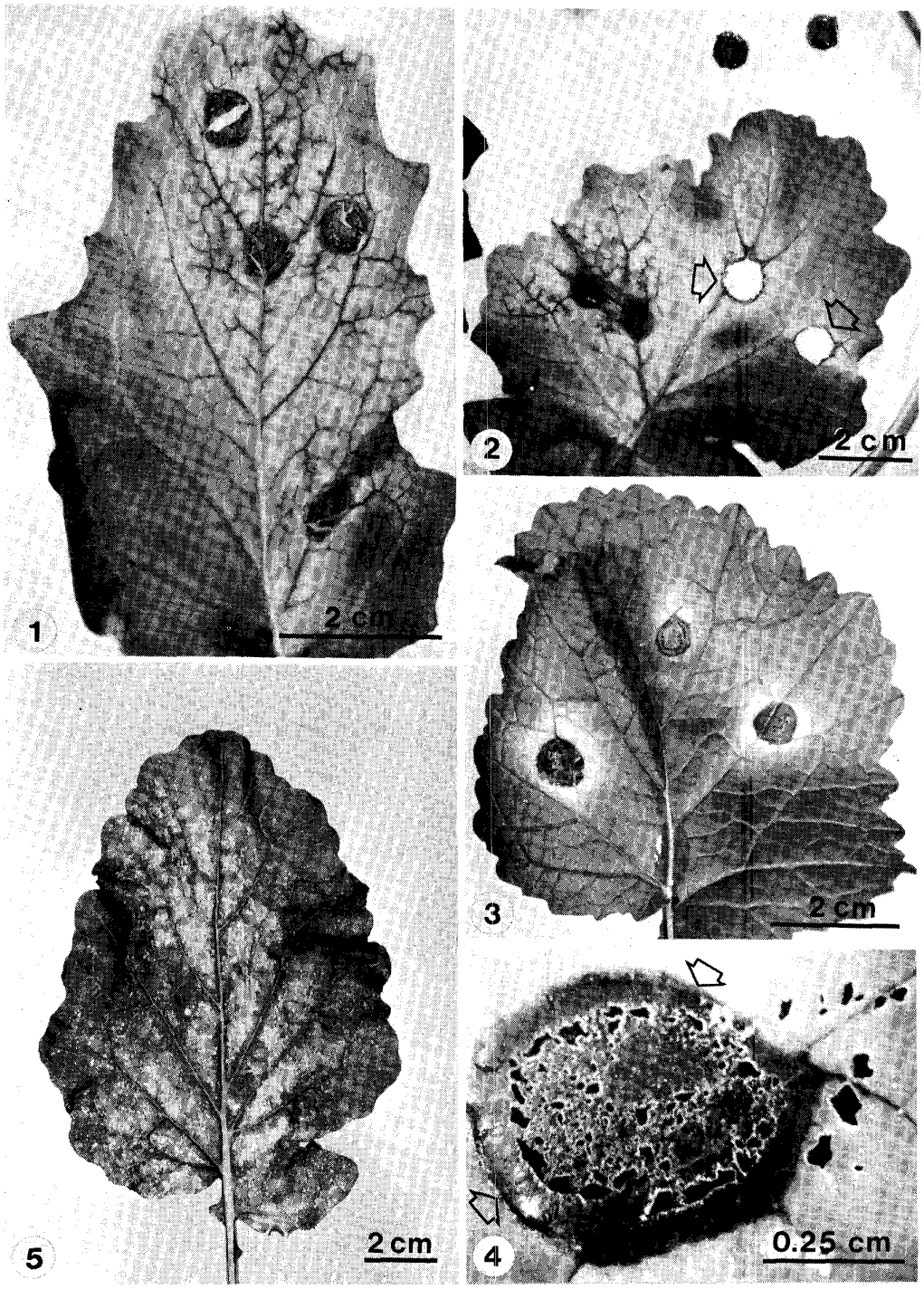

Figures 1-5. Symptoms of *Myrothecium roridum* infection on leaves of rapeseed and mustard. 1,2) Detached leaves of Torch rapeseed, 4 days after inoculation. Note the development of spots, often resulting in shot-holes (arrows) and extensive chlorosis. 3) Detached leaf of Iowa line mustard, 4 days after inoculation. Note limited chlorosis around the spot. 4) Close-up of a spot on a detached leaf of Iowa line mustard, 6 days after inoculation. Note the discrete margin around the spot (arrows) and formation of sporodochia inside and outside the spot. 5) Leaf of Midas rapeseed, 2 weeks after inoculation in the greenhouse. Note the bleached areas.

fruiting bodies; (b) The material was gently flushed once with a stream of distilled water and then vapour-fixed with osmium tetroxide; (c) The conidia were fixed overnight in 3% glutaraldehyde in 0.1 M phosphate buffer (pH 7.0), post-fixed with osmium tetroxide in the same buffer for 4 h, deposited on a Millipore filter (0.45 μ m), and washed five times with distilled water. Pieces of Millipore filter with the conidia adhering to them were used in further preparations. All the materials for SEM were frozen in liquid Freon 12, briefly stored in liquid nitrogen and dried at -70°C in an Edwards-Pearse Tissue Dryer (Model EPD2). After drying, the material was mounted on stubs with conductive glue, coated with gold and examined in a Cambridge Stereoscan S4 scanning electron microscope at the Department of Entomology, University of Alberta.

Results

Symptoms on detached leaves

On Torch a brownish-green, water-soaked spot with a chlorotic halo is evident 24 h after inoculation. Sporodochia of the fungus develop in the spot in 3 to 4 days (Figure 1), and in 4 to 6 days shot holes form (Figure 2) in many leaves. Subsequently the sporodochia form in the chlorotic part of the leaf (specimens deposited DAOM 164768). Leaves of Torch show extensive chlorosis as a result of infection. In the Iowa line progression of the disease is slower (Figure 3), followed by that in Midas. In both these cases the spot has a discrete brownish margin (Figure 4), which is absent in Torch.

Symptoms in the greenhouse and field

The leaves develop bleached areas 4 to 5 days after inoculation (Figure 5). Younger leaves are less susceptible than older ones. Sporodochia develop in 2 to 3 weeks.

Based on a visual rating scale of 0 to 5, where 5 is highly susceptible, R-500 and Torch were rated as 5, LB 22A as 3, Midas and Tower as 2, and Yellow 2 as 1.

All cruciferous weeds except common pepper grass showed infection.

Light microscopy and scanning electron microscopy

Light microscopy revealed that the sporodochia are formed at sites where a clear exudate droplet was initially located. Also, in reflected light a sac-like envelope is seen covering the sporodochium.

Material that was vapour-fixed without washing shows the sporodochia which are irregular in shape and surrounded by a fringe of marginal hyphae (Figures 6,7). The sac-like envelope covering the sporodochium appears wrinkled in some SEM preparations (Figure 8). Faint outlines of the conidia are seen through this covering due to the shallow transmission effect in the SEM.

Phialides and conidia in various stages of development are seen in material that was vapour fixed after brief washing (Figure 9). Some phialides show cracking of the cell wall at the apex. These were interpreted as stages in formation of the first conidium from the phialide. Later stages show conidia in different stages of extrusion from the phialides. A prominent collar is present (Figure 10). During later stages of development, usually five or six knob-like appendages are seen on the distal end of the conidium (Figure 9). These appendages are readily washed off since they are not seen in conidia processed through a series of liquid solutions (Figure 11).

Discussion

Myrothecium roridum is a serious pathogen of some economically important plants (9). Its host spectrum also includes some wild, ornamental, and oleiferous crucifers (2,4,8,9). In Canada, serious outbreaks of *M. roridum* have occurred on cultivated pansy (*Viola tricolor* var. *hortensis*), resulting in considerable damage to the seed crops in B.C. (2). Our results indicate that *M. roridum* is capable of parasitizing cultivars of rapeseed and mustard commercially grown in Alberta. However, natural infection of these crops by the pathogen has so far not been found in Alberta and conditions that could lead to this situation are not known at present.

Light and scanning electron microscopy have revealed some hitherto unreported features of *M. roridum*. The sporodochia develop in places where a clear exudate droplet was initially present. Macroconidia in *Fusarium culmorum* also develop in a similar way (6). The sporodochia in *M. roridum* are covered with sac-like envelopes. This covering morphologically resembles the envelopes on the exudate droplets on the sclerotia of *Sclerotinia sclerotiorum* (1). The phialides have a prominent collar. The developing conidium has 5-6 knob-like appendages on the distal end. These are washed out on passage through liquids. It is of interest that a fantailed appendage is also present on the conidium in *M. verrucaria* (9).

Acknowledgments

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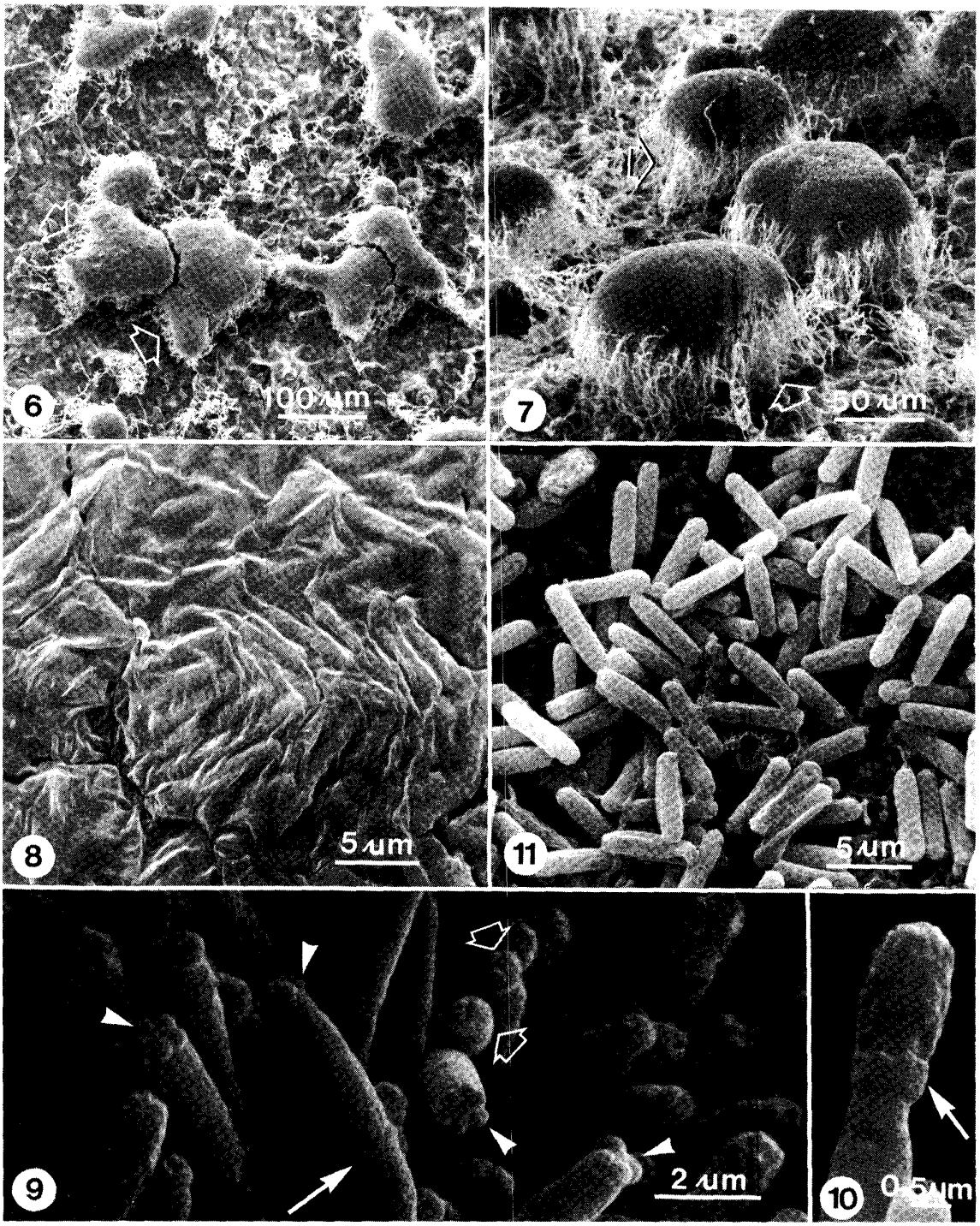

Figures 6-11. Scanning electron microscopy of *Myrothecium roridum* on the leaves of Torch. 6,7) Vapour fixed material as seen from the top (Figure 6) and side (Figure 7). Note the fringes of marginal hyphae (arrows) around the irregularly shaped sporodochia. 8) Surface view of a sporodochium showing the slightly wrinkled sac-like envelope and profiles of conidia. 9,10) Material flushed with distilled water and then vapour fixed showing phialides. Note the collar (small arrows) and knob-like appendages (arrowheads) on distal end of the developing conidium. Phialides with cracked apices represent stages in formation of first conidia (large arrows). 11) Conidia fixed in liquid solutions. Note that the knob-like appendages are not present. The cobweb-like material in the background is the Millipore filter.

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Observations on the 1976 barley yellow dwarf epidemic in eastern Canada[1]

A. Comeau and J.P. Dubuc

The 1976 barley yellow dwarf [barley yellow dwarf virus] epidemic was one of the most important in eastern Canada in the last 20 years. Primary infection was severe in oats near Fredericton, N.B., and at Sherbrooke, St-Hyacinthe, and Disraeli in Quebec. Secondary infection covered most of eastern Canada. Yield losses of 50% to 70% were reported in many areas. Oats, barley, and spring-sown wheat were affected. Ripening was accelerated about 1 week by the disease. Grain quality was affected in many ways: the thousand kernel weight and the hectoliter weight decreased, the percent hull increased, and fungal diseases to which virus-infected plants seemed especially predisposed darkened the grain color and may have contributed to lower germination.

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L'épidémie de BYDV (virus du nanisme jaune de l'orge) de 1976 a été l'une des plus importantes à survenir au cours des 20 dernières années. Les sites majeurs d'infection primaire étaient Frédéricton, N.B., et Sherbrooke, St-Hyacinthe, Disraeli au Québec. L'infection secondaire s'étendait au-delà des limites de l'Est du Canada. Des pertes de rendement de 50 à 70% ont été observées dans plusieurs régions. L'avoine, l'orge et le blé étaient affectés. La maturation des plantes a été accélérée d'environ une semaine par la maladie. La qualité du grain a été affectée de plusieurs façons: le poids de 1000 grains et le poids à l'hectolitre ont été diminués, le pourcentage d'écales augmenté, et les maladies fongiques auxquelles les plants atteints de virus semblaient particulièrement prédisposés ont noirci le grain et ont probablement contribué à une baisse du taux de germination.

Barley yellow dwarf virus (BYDV) affects cereal crops in eastern Canada annually but the level of damage has not been assessed on a routine basis. In Quebec barley yellow dwarf was widespread in 1968 and 1971, and in 1976 it developed in most fields and caused serious losses.

Observations in farmers' fields

In late June and early July we observed a remarkably low level of predators (Coccinnellidae, Syrphidae) and parasites in cereal fields, a fact which may indirectly be related to the BYD epidemic. During July, reports of high aphid populations came from many areas of Quebec, and aerial sprays were applied in the St-Hyacinthe area. On July 22, a trip through the Maritimes revealed an important area of primary infection extending about 100 miles east and west of Fredericton, N.B. Primary infection is easily recognized as it creates circular spots (0.5 to 3 m diameter) of dwarfed, discolored plants in oat fields.

In Quebec extensive areas of primary infection were observed on Aug. 4 in oats near the flowering stage in the St-Hyacinthe, Sherbrooke, and Disraeli areas (Fig. 1). In late-seeded oat fields visual estimates of loss averaged about 70% and were somewhat less in early-seeded fields. Secondary infection is not as striking because it does not create spots where yield is reduced to nothing, and because symptom expression is not always distinctive. However noteworthy levels of secondary infection were found in nearly all fields inspected in the province of Quebec and in most fields inspected in the Maritime provinces except for areas near Charlottetown, P.E.I., and Truro, N.S. The problem with assessing secondary infection is that damage is difficult to quantify by available methods. However we feel that losses in the areas visited must have been very impor-

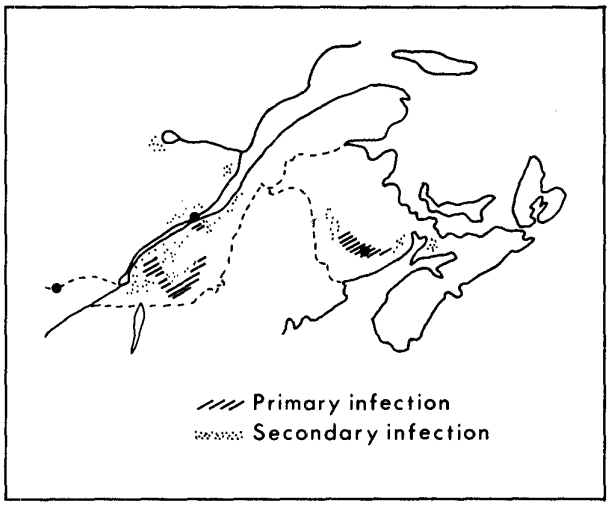

Figure 1. Observed distribution of primary infection (dark shaded) and secondary infection (light shaded) by BYDV in eastern Canada in 1976.

[1] *Contribution No. 103, Station de Recherches, Agriculture Canada, Sainte-Foy, Québec, G1V 2J3*

tant because losses in experimental fields were well above visual estimates, as will be discussed below. Late-sown fields were more visibly damaged by primary and secondary infection.

Aphid vectors appeared to be mostly *Rhopalosiphum padi*, although *R. maidis* and *Macrosiphum avenae* were also easy to find in the Quebec City area. Wheat had the highest observed aphid populations, oats being intermediate, and barley the lowest at the time we made our observations. The highest aphid numbers were found in wheat fields that had received more nitrogen fertilizer than average, supporting Coon's (5) idea that nitrogen may promote aphid reproduction. Aphid numbers appeared to peak during the elongation growth phase, as noted in New Brunswick (1).

After harvest, grain color, germination, and bushel weight were regarded as worse than average by farmers. However, in many fields in Quebec and New Brunswick overall yields seemed near normal, indicating that other factors, such as timely rainfall, may have compensated for losses in yield from BYD.

Observations in cereal trials

Various yield trials in breeding and evaluation projects were examined for the effects of the natural epidemic of BYD on yield, disease symptoms, and grain quality. Notes were taken to establish the relationship between yield losses and disease symptoms. At La Pocatière, primary infection was rare, but 60% of single plants seeded on May 25 had more than 50% yield loss from secondary infection. In other trials some secondary infection was present but we could not evaluate the yield loss. It is commonly found that thin stands or single-plant nurseries suffer heavier damage than plots seeded at the regular seeding rate (10).

At Fredericton, wheat, barley, and oats were all heavily infected in evaluation trials. Oats had losses reaching as high as 98% in certain lines. In the Fredericton Co-op Oat Test the average yield of the check varieties Dorval, Garry, Scott, and Stormont for the 3-year period 1973-1975 was 2845 kg/ha. In 1976, under BYDV infection, these four varieties produced on average 776 kg/ha, that is, 73% less than in the previous years. From 1976 data, we could also pinpoint BYDV-tolerant lines in the Fredericton tests that yielded 2387 kg/ha (Q.O. 158.37 in the Oat Screening Test) and 2007 kg/ha (O.A.338 in the Co-op Oat Test) indicating that BYD and not weather conditions was largely responsible for the 73% loss in the four check varieties. If we consider the fact that Q.O.158.37 and O.A.338 are not fully resistant and did suffer damage, we can assume that losses from BYD damage in the Fredericton trials were indeed at least 70%.

Symptoms in oats were quite obvious at Fredericton as they resulted from primary infection. However, in barley and wheat the symptoms were not as distinct, although yield losses must have occurred. Barley and wheat plants often go through a shock phase after acquiring BYDV and develop visible symptoms, but then further growth produces near-normal leaves with no symptoms, hiding the older diseased leaves. This pattern is occasionally noticed in oats also, especially in certain *Avena sterilis* lines (Comeau, unpublished). The barley trials were not harvested due to bird damage. In the wheat trials (Maritime-Quebec Co-op) the check varieties Opal and Glenlea yielded 1926 and 1789 kg/ha, or 44% and 30% less respectively than the average of the previous 3 years at Fredericton. Unfortunately there were no varieties with known tolerance to BYDV in these tests to confirm whether or not BYDV alone was responsible for this yield difference. It was noted that Fielder wheat, generally a low yielder in eastern Canada, came out with top yield (2490 kg/ha) under virus infection, probably due to some BYDV tolerance.

In the Laval University trials at St-Louis-de-Pintendre, near Quebec City, there was a slight amount of primary infection, mixed with a lot of secondary infection. Symptoms were nearly invisible in barley and wheat, and were more or less visible in oats depending on seeding date. The fact that there was a virus epidemic in that field could easily have gone unnoticed; in fact, observers unfamiliar with BYD noted only that the crop ripened too fast to allow for proper grain filling, and they attributed the damage to excessive rainfall. However, the extent of BYD damage was revealed in a test including 5 check varieties, 15 susceptible entries, and 20 BYDV-tolerant entries. The checks averaged 155 grams per plot, the susceptible lines 139 grams per plot, and the BYDV-tolerant lines 216 grams per plot. The best check was Alma (181 g/plot) and the top tolerant line Q.O.158.43 (267 g/plot). Once again we must be reminded that Q.O.158.43 did suffer damage which is difficult to quantify. Also, the yield potential of Q.O.158.43 has been rather poor in other areas or in years when BYD was not prevalent. In fact in 1975 for three stations it was 9% below the checks, and in 1976 at La Pocatière where BYD damage was not heavy, it yielded 13% below the checks. So the 38% difference between tolerant lines and susceptible checks (216 g vs. 155 g) is believed to underestimate the damage.

Germination tests showed that on average susceptible seed germinated 89% and BYDV-tolerant 92%. Gill (6) previously reported a similar difference in germination for BYDV-affected and healthy Herta barley (84% vs. 87%). This slight loss in germination is accompanied by a more important loss in seedling vigor (6) which can reduce subsequent yield. Martens and McDonald (8) divided plants according to their infection level and demonstrated clearly the damaging effect of BYDV on oat germination (84.5% for moderately diseased, 93.8% for healthy, average of two cultivars).

In pre-observation trials at St-Louis de Pintendre some barley lines possessing the Yd_2 gene for tolerance to BYDV yielded 30% above Laurier, which was the top-yielding susceptible variety.

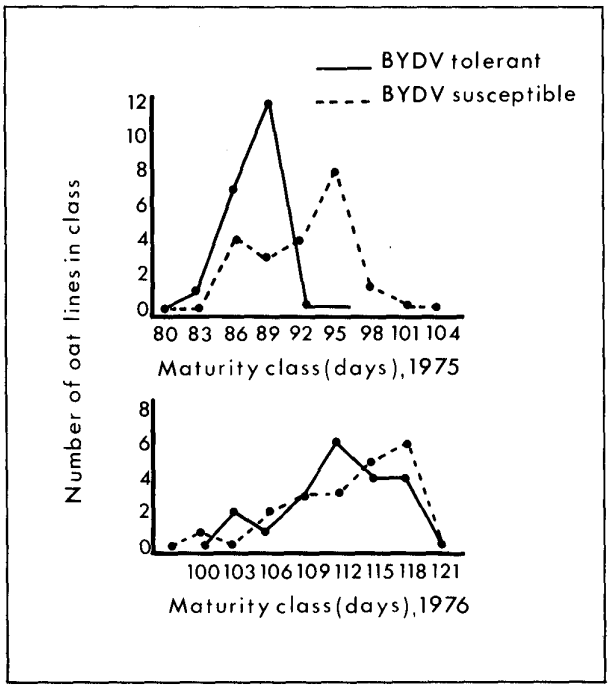

Figure 2. Maturity range of susceptible and tolerant oat lines in 1975 and 1976, showing that the maturity of susceptible lines was accelerated by the disease in 1976.

The widely noted fact that many cereal crops senesced before filling the grain in 1976 may be due mostly to the prevalence of BYDV. This accelerated senescence has been noted before (3,7) and is illustrated in Figure 2; in 1975, the susceptible lines in the Laval University oat trial tended to mature about a week later than the tolerant, but in 1976 both groups were in about the same maturity range because BYD accelerated the senescence of the susceptible group. However this virus effect was not enough to overcome the effect of rainy weather, and the 1976 crop matured late. Premature senescence is damaging because it occurs during grain filling. BYD generally does not alter the heading and flowering dates significantly; only ripening is affected.

Oat plants attacked by BYDV late in the season (secondary infection) may present few or none of the typical symptoms (9), such as reddening of the leaves. In some cultivars, all that will be noted is a whitish tint of the glumes before ripening occurs. Also noted is the predisposition of BYD-affected plants to fungal diseases such as septoria leaf blotch (4). In Fredericton at harvest time the diseased oat plots became black and sooty, apparently from attack by fungi. Grain quality in nearby farmers' fields was below normal, and the grain was dark-colored in many instances.

In the 1976 eastern cooperative oat trials all across eastern Canada there were a few oat lines known for their tolerance to BYDV, especially O.A.236, O.A.240-7, and O.A.338. They ranked respectively 1st, 6th, and 16th (out of 30) for yield (including the Fredericton data, and 1st, 2nd, and 3rd, for hectoliter weight (excluding the hulless oats); we believe that BYDV tolerance has contributed to this performance. It is unfortunate that hectoliter weights were not noted in previous years.

The extent to which the reserve of BYDV in susceptible perennials (timothy, brome, etc.) has increased due to the 1976 epidemic is unknown and deserves investigation. It is logical to expect an increase, but this in itself is not enough to forecast a BYDV epidemic in 1977, as it will depend on the number of aphids flying to disseminate the disease; the level of aphid population within a field also would be expected to influence the amount of damage occurring (2).

Acknowledgments

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Field tests of cereal seed treated with nonmercurial fungicides[1]

R. V. Clark

Several seed treatment chemicals were assessed in the field at Ottawa for their effects on seedling emergence, disease development, and yield of oats, barley, and wheat. None of the chemicals consistently increased seedling emergence or yield of the three cereals over the controls, although one chemical was effective one year. Yield response of individual cultivars varied considerably to treatment of the seed. Seedling blight [*Pyrenophora avenae*] of oats was present in trace amounts in in these tests and its significance could not be determined. Seedling blight [*Cochliobolus sativus*] of barley was prevalent in certain years and was reduced by treatment. More uniform data were obtained with the use of large plots and mechanical equipment than with small plots, and hand labor.

Can. Plant Dis. Surv. 57: 45-48. 1977

On a évalué l'incidence de plusieurs produits chimiques (traitement des semences) sur la levée des plantules, l'évolution des maladies et le rendement de l'avoine, de l'orge et du blé en plein champ à Ottawa. Aucun des produits testés n'a régulièrement accéléré la levée des plantules, ni accru le rendement de ces trois céréales en regard des parcelles témoins, bien que l'un des produits se soit révélé efficace au cours d'une année. Le rendement des cultivars a varié considérablement avec le traitement de la semence. On a constaté la présence de la rayure des feuilles [*Pyrenophora avenae*] de l'avoine au cours des tests, mais on n'a pu évaluer son importance. La brûlure des semis [*Cochliobolus sativus*] s'est manifestée certaines années, mais le traitement a réussi à en atténuer la gravité. L'utilisation de grandes parcelles et de machines a permis d'uniformiser davantage les données que celle des petites parcelles et du travail manuel.

The withdrawal of mercury compounds for use in treating cereal seed for the control of seed and soil-borne diseases has turned attention to other, less toxic chemicals. The withdrawal has resulted in considerable concern over the control of seedling blight [*Pyrenophora avenae* Ito & Kurib.] of oats because previous work (1), as well as growth room and laboratory studies (unpublished data), have shown that this disease is readily controlled by several mercury compounds; the effectiveness of nonmercurial compounds for controlling seedling blight is not known. Seedling blight has occurred occasionally in Ontario especially on the cultivar Gemini. Seed samples of this cultivar collected from a number of locations in eastern Canada in 1970 were found to contain trace to light amounts of *P. avenae*. For this reason field trials were run in 1971 and 1972 comparing several registered and experimental nonmercurial seed treatments on three cultivars of oats. In 1973-75 the investigation was expanded to include oats, wheat, and barley in large plots. This paper reports on the methods used and the effects of the chemicals on emergence, disease development, and seed yields.

Materials and methods

The source, composition, and application rate of the seed treatment chemicals assessed are listed in Table 1. They were applied at recommended concentrations to 225-g samples of seed in 1-liter widemouth erlenmeyer flasks which were rotated for 5 min on a custom-made treater that provided a tumbling action.

Garry, Gemini, and Scott oats (*Avena sativa* L.); Bonanza, Brock, Fergus, Galt, Herta, Paragon, Trent, and Vanier barley (*Hordeum vulgare* L.); and Opal, Selkirk, and Glenlea wheat (*Triticum aestivum* L.) were used in different years. Whenever possible, local high quality seed of a uniform size and condition was used.

In 1971 and 1972, 4-row plots 3.0 m long were planted at the recommended rates for Ontario with a 10-row mechanical plot seeder with the rows 17.8 cm apart. Treatments were replicated four times and a randomized block design was employed. From 1973 to 1975 large plots (7.6 x 2.5 m) were employed using three replicates in a split plot design with cultivars as main plots and chemical treatments as subplots. In 1973 and 1974 seeding was done with a commercial farm drill in plots of 13 rows 17.8 cm apart. Because of planting difficulties in 1974 with the commercial drill, seeding was done in 1975 with a 4-row plot seeder having a bulk hopper attachment. Each plot consisted of 12 rows 22.8 cm apart. In 1974 and 1975 two seeding rates, the recommended rate (oats 76.2, wheat 100.9, barley 107.6 kg/ha) and one-half the recommended rate, were used.

Emergence counts were taken on duplicate 1.5 m portions of row selected at random within each replicate 3 to 4 weeks after seeding. At this time the seedlings were examined for the presence of seedling blight of oats [*P. avenae*] and seedling blight of barley [*Cochliob-*

[1] *Contribution No. 519, Ottawa Research Station, Agriculture Canada, Ottawa, Ontario K1A 0C6*

Table 1. Seed treatment materials assessed at Ottawa, 1971-75

Year	Number	Source*	Product	Chemical Name	Dosage (oz/bu)
1971	1	Du Pont	Ceresan M	ethyl mercury p-toluene sulfonanilide 7.7%	0.5 & 1.0
	2	Uniroyal	Vitaflo	carbathiin 17.3% + thiram 15.4%	2.0 & 4.0
	3	Chipman	Agrox NM	maneb 37.5% + hexachlorobenzene 10%	2.0 & 4.0
	4	Buckman	TCMTB	2-(thiocyanomethylthio)benzothiazole 30%	0.85 & 1.7
1972	5	Uniroyal	Vitaflo	carbathiin 17.3% + thiram 15.4%	2.0
	6	Merk	ME-77	identity not available	1.5
1973	7	Uniroyal	Vitaflo	carbathiin 17.3% + thiram 15.4%	2.0
	8	Chipman	Agrox NM	maneb 50%	2.0
	9	Chipman	TF3219	BAS 3270F(2,5 dimethyl-furane 3-carboxylic acid cyclohexylamide) 15% + maneb 25%	2.0
	10	Chipman	TF3222	pyracarbolid 15% + maneb 25%	2.0
	11	Interprovincial	Busan 30	2-(thiocyanomethylthio)benzothiazole 37.5%	0.85
	12	Interprovincial	Busan DP	2-(thiocyanomethylthio)benzothiazole 18.75% + lindane 26.65%	0.85
	13	Merk	ME-77	identity not available	1.5
1974	14	Chipman	Agrox NM	maneb 50%	2.0
	15	Chipman	TF3222	pyracarbolid 15% + maneb 25%	2.0
	16	Uniroyal	Vitaflo	carbathiin 17.3% + thiram 15.4%	1.5
1975	17	Chipman	Agrox flowable	maneb 25%	2.5
	18	Chipman	TF3262	pyracarbolid 10% + maneb 16.7%	2.5
	19	Uniroyal	Vitaflo	carbathiin 17.3% + thiram 15.4%	1.5
	20	Uniroyal	UBI 2078	identity not available	1.5

* E. I. Du Pont de Nemours & Co., Inc., Wilmington, Delaware; Uniroyal Chemical Division, Elmira, Ontario; Chipman Chemicals Ltd., Hamilton, Ontario; Buckman Laboratories, Inc., Memphis, Tennessee; Merck & Co., Inc., Rahway, New Jersey; Interprovincial Cooperatives Ltd., Winnipeg, Manitoba.

olus sativus (Ito & Kurib.) Drechsl. ex Dastur]. When these diseases were present in more than trace amounts percentage counts of infected seedlings in a given area were made. The prevalence and severity of foliage diseases were determined at growth stages 10.5 to 11.1 on the Feeke's scale (3) by assessing the percentage of leaf area affected by each disease on the top three leaves of 10-15 plants picked at random from rows that were not to be used for determining seed yields. In 1971 and 1972 the two center rows of each plot were harvested by hand, excluding the plants in the 30 cm portion at the ends of each row. From 1973 to 1975, yields were determined by harvesting a 1.25 m wide center portion of each plot with Hegé combine after a 0.8 m border had been removed from each end. The harvest portion of each plot measured 6 x 1.25 m or approximately 0.74 x 10^{-3} ha. The thousand kernel weight of harvested seed was determined on duplicate 250-seed samples.

Results and discussion

In the 1971 and 1972 trials none of the treatments used (Table 1) significantly improved emergence or seed yield of the three cultivars of oats. However, practically no seedling blight was found even in the untreated susceptible check Gemini.

From 1973 to 1975, the seed treatments were assessed on oats, barley, and wheat cultivars and particular emphasis was placed on the use of mechanical equipment and large field plots so that conditions would be more comparable to farming operations. Planting, weed control, and harvesting were done mechanically. In 1973 seven chemicals (Table 1) were compared on Herta and Vanier barley, Garry oats, and Opal wheat, while additional cultivars of barley (Bonanza, Brock, Galt, Paragon, and Trent), oats (Scott and Gemini), and wheat (Selkirk) were treated with Agrox NM and Vitaflo only. On the average, the treatments had no effect on the emergence of the various cultivars (Table 2). The chemical TF3222 consistently improved the yields of the three crops while the other fungicides were variable in this respect (Table 3). However individual cultivars varied considerably in their yield response to treatment, as evidenced by the response of the barley cultivars (Table 4); in this test treated plots of Bonanza and Paragon barley showed no increase in yield, while treated plots of Galt and Trent showed a 20% increase over the check. The variation in the kernel weight of seed of these same cultivars was not as great as the variation in yield.

In 1974 and 1975 fewer chemical treatments were used, with two seeding rates, the recommended rate and one-half the recommended rate. With one or two exceptions emergence counts were not affected by treatment (Table 2). In 1974 there were a significant improvement in emergence of treated compared with untreated barley at the half-rate seeding. The incidence

Table 2. Emergence counts (avg no. seedlings/1,5 m of row) of several cultivars of wheat, barley, and oats grown in the field for 3 years using seed treated with various chemical fungicides

1973					1974								1975							
						Wheat		Barley		Oats				Wheat		Barley		Oats		
						Sdg rate§		Sdg rate		Sdg rate				Sdg rate		Sdg rate		Sdg rate		
Treat. no.†	Wheat	Barley	Oats	Mean	Treat. no.†	1	2	1	2	1	2	Mean	Treat. no.†	1	2	1	2	1	2	Mean
7	48	75	69	64	14	112	63	106	74	77	44	79	17	82	49	91	53	72	50	66
8	52	77	66	65	15	107	62	102	69	75	50	77	18	78	39	87	46	74	53	63
9	50	83	52	62	16	104	64	104	78	84	46	80	19	85	41	94	51	93	55	70
10	52	83	64	66	Control	105	68	102	58	80	52	77	20	72	39	84	54	68	35	59
11	35	78	51	55		NS	NS	NS	**	NS	NS		Control	63	46	85	53	80	52	63
12	46	74	78	66										*	*	NS	NS	**	**	
13	48	77	47	57																
Control	52	77	63	62																
	NS	NS	NS																	

† See Table 1 for chemical identification.
§ Seeding rate 1, recommended rate; rate 2,half recommended rate, see text.
* Significant at the 5% level
** Significant at the 1% level
NS Not significant

Table 3. Yield (g/plot) of several cultivars of wheat, barley, and oats grown in the field for 3 years using seed treated with various chemical fungicides

1973					1974								1975					
						Wheat		Barley		Oats				Barley		Oats		
						Sdg rate§		Sdg rate		Sdg rate				Sdg rate		Sdg rate		
Treat. no.†	Wheat	Barley	Oats	Mean	Treat. no.†	1	2	1	2	1	2	Mean	Treat. no.†	1	2	1	2	Mean
7	1967	1944	1192	1701	14	1662	1150	2367	2496	1536	1394	1767	17	2803	1513	573	552	136C
8	1684	2011	1304	1666	15	1307	1461	2624	2307	1598	1522	1803	18	2865	2025	606	659	1539
9	2214	1446	833	1498	16	1556	1355	2417	2505	1697	1401	1822	19	2742	1547	583	633	1376
10	2219	2073	1206	1833	Control	1599	1186	2448	2385	1703	1393	1786	20	2771	1230	427	665	1273
11	1499	1821	1227	1616									Control	2609	1830	588	469	1374
12	1657	1943	1193	1598														
13	1723	2020	952	1565														
Control	1800	1898	1180	1626														

† See Table 1 for chemical identification.
§ Seeding rate 1, recommended rate; 2, half recommended rate, see text.

of root rot and seedling blight was approximately the same at the two seeding rates (Table 5). Therefore the overall control attained was proportionally greater at the lower seeding rate. Vitaflo was more effective than the other two chemicals in reducing the amount of seedling blight. However at maturity comparable differences in seed yields were not evident, especially between the two seeding rates of barley (Table 3). The yield response to the chemical TF3222 was not as consistent as in the previous year.

In 1975 emergence differences were more evident with oats and wheat than with barley. The differences with oats were significant because the chemical UBI 2078 reduced emergence below the control. In contrast to 1974, the yield of barley at the half-rate seeding was much lower than at the recommended rate. Oat yields on the other hand, were not affected by rate of seeding or by chemical treatment even though emergence counts were different.

The prevalence of the leaf diseases powdery mildew [*Erysiphe graminis* D.C. ex Mérat] of barley and wheat, spot blotch [*Cochliobolus sativus*] of barley and wheat, and crown rust [*Puccinia coronata* Cda. f. sp. *avenae* Erikss. & E. Henn.] of oats was determined near maturity each year, and there was no evidence of any differences, in disease development among the treatments. However, in the larger plots it was observed occasionally that at approximately the time of heading the foliage of plants in treated plots was often somewhat lighter in color than the foliage of plants from untreated seed, particularly among the barley cultivars.

In these tests treatment of seed containing low levels of seed-borne diseases with chemicals generally did not

Table 4. Yield (g/plot) of five barley cultivars in the field in 1973 using seed treated with two chemical fungicides

	Bonanaza		Brock		Galt		Paragon		Trent		Mean	
Treatment	Yield (g)	% of check	Yield (g)	% of check	Yield (g)	% of check	Yield (g)	% of check	Yield (g)	% of check	Yield (g)	% of check
Check	1974		1766		1549		2248		1933		1858	
Agrox NM	1794	97.5	1932	109.4	1853	119.6	2224	98.9	2236	115.7	1999	107.6
Vitaflo	1873	104.4	1977	111.9	1861	120.1	2227	101.0	2281	118.0	2052	110.4

Table 5. Percent root rot and seedling blight infection [*Cochliobolus sativus*] on Herta and Vanier barley grown in the field in 1974 using seed treated with three chemical fungicides

	Herta Seeding rate+		Vanier Seeding rate+		Mean
	1	2	1	2	
Agrox NM	12	22	15	9	14.5
TF3222	15	16	20	19	17.5
Vitaflo	3	7	13	9	8.0
Control	28	19	22	8	19.2

+Seeding rate 1, recommended rate; rate 2, half recommended rate, see text.

improve emergence or seed yield. Even when emergence was increased by treatment improvement in yields did not necessarily follow. In some situations the use of the lower seeding rate resulted in reduced yield but in other cases it did not. It would be difficult to predict when the lower rates could be used safely especially if a seed-borne disease was present. Cultivar response to seed treatment was also quite variable, indicating that specific treatments should be assessed on a considerable number of cultivars before recommendations are formulated. The use of large plots for assessing the merits of seed treatment chemicals is recommended, especially since the problem of interplot interference (2) is probably reduced when plots of this size are employed.

Acknowledgement
The author wishes to thank J. H. Clark for technical assistance.

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Field diseases of onions in coastal British Columbia[1]

D.J. Ormrod, T.A. Swanson, and G.N. Smith[1]

Surveys were carried out in 1975 and 1976 to determine the distribution of white rot [*Sclerotium cepivorum*] and other disorders of onion (*Allium cepa*) in coastal British Columbia. White rot was found in green bunching onions on 7 of 47 farms and in dry bulb onions on 5 of 13 farms. Average losses due to all disorders were estimated at 14% of the green onion crop and 24% of the dry bulb crop.

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On a effectué des relevés en 1975 et 1976 pour déterminer la distribution de la pourriture blanche (*Sclerotium cepivorum*) et d'autres accidents de l'oignon (*Allium cepa*) dans la région côtière de la Colombie-Britannique. On a constaté la présence de pourriture sur des oignons verts à botteler dans 7 fermes sur 47, et sur des oignons secs de bulbilles dans 5 fermes sur 13. Les pertes moyennes attribuables à tous les accidents sont estimées à 14% pour les oignons verts et à 24% pour les oignons de bulbilles.

Green bunching onions have been grown for decades on the small-acreage market gardens in the muck soils of South Vancouver, South Burnaby and adjacent areas. In 1975 approximately 1.46 million pounds (662 tonnes) valued at $309,000 were produced on about 35 acres (74 ha).

Spring-seeded dry bulb onions are a more recent addition to the list of vegetable crops grown commercially in the coastal area. Production has grown steadily over the last 10 years but has now levelled off at approximately 300 acres (741 ha), all grown in muck soils at Cloverdale, 30 miles east of Vancouver. In 1975, 11.8 million pounds (5,352 tonnes) valued at $1,029,000 were produced.

In 1970 two small patches of plants affected by onion white rot [*Sclerotium cepivorum* Berk.] were found in one field of dry bulb onions in the cloverdale area (1). By 1974 the disease was found in every onion field on that farm and caused a loss of about 50% of the crop.

In the spring of 1975, regulations were passed under the British Columbia Plant Protection Act forbidding the growing of onions on land known to contain *S. cepivorum* and restricting the movement of soil and machinery from such land. To determine the usefulness of this regulation as a control measure, surveys of all onion farms in coastal British Columbia were conducted in 1975 and 1976.

In addition to white rot, the survey covered all onion field diseases because certain other pathogens such as smut [*Urocystis magica* Pass. ap. Thum.] were becoming a concern to growers.

The last published estimates of crop losses due to onion diseases in coastal British Columbia are those of Toms in 1965 and 1966 (2, 3). His estimates were based on random observations rather than detailed surveys and the only diseases mentioned were smut and downy mildew in the green bunching onion crop and neck rot in the dry bulb crop.

The estimated losses in green bunching onions were 5% each from smut and downy mildew in 1965 and 1% and 2%, respectively, in 1966; losses to the dry bulb crop from neck rot were 15% each year (2, 3).

Methods

Green bunching onions

All of the known green onion producing farms were surveyed twice in 1975 and twice in 1976. As green onions are transplanted or seeded successively from early spring to late summer it is possible that not all crops were seen during the two surveys. In 1975, 27 acres were surveyed and in 1976, 33 acres. This probably represents more than 75% of the acreage in both years.

Surveyors wore disposable polyethylene boots to prevent the movement of soil from farm to farm. At each farm they walked between beds, visually observing all plants, stopping frequently to pull bunches of approximately 10 plants to determine the incidence of root disorders. Maps were drawn of each farm showing the location of onion fields and disorders within the fields at each survey date.

Dry bulb onions

All of the commercial dry bulb onion farms were surveyed twice each year. In contrast to the green onion survey, this meant that each crop was examined twice. In 1975, most fields were surveyed by walking through in a W pattern pulling 200 plants at each of five locations in a typical 10 acre field. In smaller fields or where white rot was suspected in 1975 and in all fields in 1976, the surveyors walked down every 5th bed to avoid missing any small areas of abnormal growth. Again detailed examination was made of 200 plants pulled at five locations. When white rot was found, the

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Table 1. Incidence* of diseases, physiological disorders, and maggot injury in green bunching onions in 1975 and 1976

Disease or disorder	June 1975		July 1975		July 1976		August 1976	
	No. of farms	Acreage %	No. of farms	Acreage %	No. of farms	Acreage %	No. of farms	Acreage %
Downy mildew	0		0		0		11	0.8
Fusarium rot	0		0		1	0.1	2	0.1
Pink root	1	0.1	1	1.0	0		1	0.1
Smut	3	0.5	4	0.6	4	0.8	3	0.5
White rot	3	0.3	0		5	0.2	0	
Tip dieback and botrytis blast	29	0.7	31	21.7	10	2.6	30	9.6
Other disorders	17	11.0	11	1.8	7	0.4	8	3.1
Maggot injury	18	3.9	7	0.7	20	0.7	21	0.4

* No. of farms affected out of 45 surveyed in 1975, 47 in 1976; and % of total acreage affected.

Table 2. Incidence of diseases, physiological disorders, and maggot injury in dry bulb onions on 13 farms in 1975 and 1976

Disease or disorder	June 1975		July 1975		July 1976		August 1976	
	No. of farms	Acreage %	No. of farms	Acreage %	No. of farms	Acreage %	No. of farms	Acreage %
Downy mildew	0		6(9)*	2.6(18.0)*	2	0.1	13	70.2
Fusarium rot	0		5	1.2	2	0.4	4	0.1
Pink root	1	0.1	4	12.0	0		5	16.0
Purple blotch			0		1	0.1	2	0.1
Smut	10	4.0†	7	1.8**	10	1.2	9	0.4
White rot	0		0		2	0.1	4	0.2
Tip dieback and botrytis blast	4	7.0	9	18.0	8	11.0	8	18.0
Maggot injury	4	0.5	0		11	0.1	9	0.1

* A third survey for downy mildew was conducted in August, 1975; the results of that survey are shown in parentheses.
† Does not include 14 acres turned under prior to the survey due to more than 50% smut infection.
** Does not include 5 acres turned under prior to the survey due to more than 50% smut infection.

area was carefully determined. In the case of isolated infections diseased plants were pulled, placed in plastic garbage bags, removed from the field, and destroyed.

Results and discussion

The survey results are summarized in Tables 1 and 2. The green bunching onion crop was remarkably free of serious diseases (Table 1). By far the most serious problems were the tip dieback - botrytis blast complex and physiological disorders such as unsuitable soil conditions and herbicide injury. Although smut and white rot were present on several farms, infection in the presence of inoculum seems to be the exception rather than the rule. Downy mildew is more serious than indicated by the survey, frequently destroying whole crops in September and October.

In the dry bulb crop also, tip dieback and botrytis blast is of major concern (Table 2). The cause of tip dieback is not known. A soil sampling study carried out in conjunction with the disease survey in 1976 indicated no correlation with soil pH, salinity, or nitrate levels. (R. Kingston, personal communication).

The possible role of ozone injury in the Cloverdale area has not been investigated. Smut losses were high in 1975 but improved disease control methods reduced infection in 1976. Due to the unusually wet August of 1976, downy mildew became epiphytotic and yield reductions as high as 40% were probable in some fields.

Although white rot was not found in the Cloverdale bulb onion area in 1975, it did show up on four farms in 1976. Three of these appeared to be related to the original discovery in 1970 while the other appeared to be a completely separate outbreak.

There are no published reports of pink root [*Pyrenochaeta terrestris* (Hansen) Gorenz, Walker & Larson] or purple blotch [*Alternaria porri* (Ell.) Ciferri] from coastal

British Columbia, but they have been observed here previously. Pink root is particularly common late in the season.

Each disease has a different effect on yield. For example, low levels of smut (<5%) may result in fewer but larger bulbs with little net effect on dollar value. Higher levels of smut reduce yield and dollar value. Downy mildew can completely destroy green onion crops. In dry bulb onions it can reduce yield by up to 40% and delay maturity which may lead to storage problems. Pink root may occasionally reduce yields by up to 30% but helps to mature the crop and prevent botrytis neck rot in storage.

Taking all of these factors into consideration, a dollar loss due to all disorders in the green bunching onion crop would be 14% or $42,000. This figure does not include the cost of control measures.

In the case of bulb onions, botrytis neck rot and other storage rots are acknowledged to be the greatest cause of loss. In 1965 and 1966, Toms estimated the losses due to neck rot at 15% of the crop (2, 3). While the neck rot losses in the 1976 crop were at least 15%, observations over the past several years suggest 12% as a realistic long term average loss. Adding to this figure the losses due to other disorders indicated in the 1975 and 1976 field surveys results in a total loss due to diseases and other disorders of 24% or $240,000. Again, the cost of control measures is not included. By these estimates and based on 1975 prices, it would appear that the total annual loss due to diseases and other disorders of onions in the lower Fraser Valley is in excess of $0.25 million.

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Plant parasitic nematodes from Canada and abroad, 1973-74

Robert Sewell[1]

Nematodes isolated from plants and other material intercepted at ports of entry or detected in surveys are listed.

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L'auteur énumère les types de nématodes isolés de végétaux et d'autres matériels interceptés aux points d'entrée ou décelés à l'occasion d'enquêtes.

Nematodes isolated and identified at the Biosystematics Research Institute from infested plant materials and soil samples include many that are parasitic on native plants and crops. Most samples are from Plant Protection Division (PPD), Agriculture Canada, and comprise material intercepted at ports of entry or detected in surveys of nurseries. Identification services are also provided other government agencies, universities, and individuals. Nematodes identified during 1973-74 and their source are listed below:

Cyst-forming nematodes

Heterodera schachtii Schmidt 1871, sugar beet nematode: leek (*Allium porrum*) 1973 France; plants 1974 California, Netherlands.
Heterodera cruciferae Franklin 1945: leek 1973 France.
Heterodera trifolii Goffart 1932, clover cyst nematode: leek 1973 France; soil (potato) 1973-74 B.C. Que., P.E.I., Alta., N.B.; laurel 1973 Italy; farm machinery 1974 Japan.
Heterodera avenae Wollenweber 1924, oat cyst nematode: soil 1973 Ontario soil survey (PPD); *Bromeliada* sp. 1974 Denmark; nurseries 1974 Ontario survey (PPD).
Heterodera latipons Franklin 1969: soil (potato) 1973-74 P.E.I.
Heterodera goettingiana Liebscher 1892, pea cyst nematode: soil (vehicle) 1973 Japan.
Heterodera glycines Ichinohe 1952, soybean cyst nematode: soil (vehicle) 1973 Japan.
Heterodera punctata Thorne 1928, grass cyst nematode: soil 1973 Toronto, Ont.; Red Bay and Pinmore, Labrador; nurseries, potato field 1974 B.C., Nfld. survey (PPD).
Heterodera weissi Steiner 1949: plants 1974 California , Netherlands.
Heterodera trifolii Goffart 1932, clover cyst nematode: leek 1973 France; soil (potato) 1973-74 B.C. Que., P.E.I., Alta., N.B.; laurel 1973 Italy; farm machinery 1974 Japan; *Lilium* bulbs 1974 Czechoslovakia; heather 1974 England; vehicle 1974 Germany.
Globodera rostochiensis (Wr.) Mulvey & Stone 1976 (*Heterodera rostochiensis* Wollenweber 1923): soil (potato) 1974 Newfoundland.

Root-knot nematodes

Meloidogyne hapla Chitwood 1949, northern root-knot nematode: rose roots 1973 France, Netherlands; celery 1973 Vineland, Ont.; soil (forage) 1973 P.E.I.; rose roots 1974 Tyler, Texas; *Ligustrum* sp. 1974 Tennessee.
Meloidogyne incognita (Kofoid & White 1919) Chitwood 1949: tomato 1973 Tyler, Texas; Clifton Georgia; greenhouse 1973 Calgary, Alta.; hoya roots 1973 Italy; tomato (greenhouse) 1974 Vineland, Ont.; cherry (greenhouse) 1974 Manitoba.
Meloidogyne sp. (description in press): turfgrass 1974 southern Ontario golf courses.

Spiral nematodes

Helicotylenchus pseudorobustus (Steiner 1914) Golden 1956: roses 1973 Netherlands.

Helicotylenchus digonicies Perry, *in* Perry et al. 1959: plants (soil) 1974 USSR.
Helicotylenchus pseudodigonicus Szczygiel 1970: plants (soil) 1974 USSR.
Helicotylenchus canadensis Waseem 1961: carrot (soil) 1974 Bradford, Ont.
Rotylenchus buxophilus Golden 1956: peony 1974 Niagara Falls, Ont.
Rotylenchus fallorobustus Sher 1965: juniper (soil) 1973 East Germany.
Rotylenchus quartus (Andrassy 1958) Sher 1961: plants (soil) 1974 USSR.
Rotylenchus robustus (De Man 1976) Filipjev 1936: juniper (soil) 1973 East Germany.

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Pin nematodes

Paratylenchus hamatus Thorne & Allen 1950: soil 1973 Vineland Station, Ont.

Root lesion nematodes

Pratylenchus penetrans (Cobb 1919), Filipjev & Schwermans-Stekhoven 1941: deciduous shrubs (roots) 1973 East Germany; juniper (roots) 1973 East Germany; rose (roots) 1973 Virgil, Ont.

Foliage nematodes

Aphelenchoides fragariae (Ritzenia Bos 1890) Christie 1932: Rieger begonia (leaves) 1974 Ottawa, Harrow, & Beamsville, Ont.; 1973 Harrow, Ont.

Aphelenchoides saprophilus *Franklin 1957:* Aralia *sp. 1973 Belgium; shallots 1973 France.*

Stunt nematodes

Tylenchorhynchus dubius (Bütschli 1873) Ailipjev 1936: juniper (roots) 1973 East Germany; plant material 1974 USSR.

Macroposthonica ornata (Raski 1958) de Grisse & Loof 1965: *Gardenia* 1973 South Carolina.
Macroposthonia xenoplax (Raski 1952) de Grisse & Loof 1965: soil 1973 Vineland Station, Ont.
Merlinius brevidens (Allen 1955) Siddiqi 1970: soil 1973 Vineland Station, Ont.
Criconemoides sp., (undescribed ring nematode): rose (soil) 1973 Holland.

Other tylenchids

Ditylenchus destructor Thorne 1945, potato rot nematode: Iris bulbs 1973 Mt. Vernon, Wash., USA.
Ditylenchus myceliophagus Goodey 1958: azalea cuttings 1973 Holland.
Tylenchus exiguus de Man 1921: azalea cuttings, laurel, spurge 1973 Yugoslavia, Holland.

Dorylaimids

Trichodorus porosus Allen 1957: gardenia 1973 South Carolina.
Xiphinema americanum Cobb 1913: gardenia 1973 South Carolina.

Other nematodes

All of the following nematodes were isolated from mud samples from the MacKenzie River System, North West Territories; survey by Freshwater Institute, Environment Canada, 1973.
Dorylaimus stagnalis Dujardin 1845
Tripyla - several species
Tobrilus - several species
Enoplia - several species
Gastromermis Micoletsky 1925
Hydromermis Corti 1902
Limnomermis Daday 1911
Lanceimermis Artyukhovskiy 1969
Neomesomernies Nickle 1972.

Plant parasitic nematodes in turfgrass in southern British Columbia

S.G. Fushtey[1] *and F.D. McElroy*[2]

Turfgrass and soil samples from 12 different sites representing golf greens, bowling greens, and playing fields in the Fraser Valley extending from Chilliwack in the east to and including the city of Vancouver were analysed for the presence of plant parasitic nematodes, and 10 genera were recorded. At most of the sites there was no apparent nematode injury to the grass. At two sites unhealthy turf was associated with extremely high numbers of *Helicotylenchus*. Samples from a third site with a disease problem yielded high numbers of *Criconemoides* and *Tylenchorhynchus*. The results show that certain plant parasitic nematodes occur in sufficiently high numbers to cause appreciable damage to turfgrass and that nematodes as agents of disease should not be overlooked in the diagnosis of turfgrass disease problems in this region.

Can. Plant Dis. Surv. 57: 54-56. 1977

Des échantillons de gazon et de sols ont été prélevés en 12 endroits représentatifs des parcours de golf, des boulodromes et des terrains de jeux de la vallée du Fraser, de Chilliwack à l'est, jusque et y compris la ville de Vancouver, puis analysés pour y déceler la présence de nématodes parasitant les plantes. On a ainsi identifié 10 genres de nématodes. Dans la plupart des cas, il ne semble pas que les nématodes aient occasionné de dégâts à l'herbe. En deux endroits, toutefois, on a établi un rapport entre le mauvais état du gazon et les fortes populations de *Helicotylenchus*. L'analyse des échantillons prélevés d'un troisième endroit problème à révélé la présence d'un grand nombre de *Criconemoides* et de *Tylenchorhynchus*. Il ressort de l'étude que les populations de certains nématodes parasites sont assez élevées pour occasionner des dégâts importants au gazon et que ces pathogènes ne doivent pas être négligés lors du diagnostic des maladies du gazon dans cette région.

Plant parasitic nematodes are present in all turfgrass soils, sometimes in large numbers, but their importance as a factor in health of turfgrass is not well defined. In warm climates nematodes are known to cause damage to golf greens and other turfgrass areas, and measures for control are recommended: Heald and Perry (1969), Perry et al (1970). However, the importance of the different nematodes is not well defined. Lucas et al. (1974) found high populations of plant parasitic nematodes in turf throughout North Carolina but states that observations did not reveal a distinct relationship of damage with the nematodes present except with *Belonolaimus longicaudatus* in bermudagrass where the nematodes caused poor growth and quality of the grass. They found large numbers of *Trichodorus, Hoplolaimus, Tylenchorhynchus* and *Helicotylenchus* associated with bentgrass turf but no visible damage. On the other hand, Troll and Tarjan (1954) reported populations of *Tylenchorhynchus* and *Rotylenchus* associated with decline in turf in Rhode Island; Perry (1958) reported decline and root injury in Kentucky bluegrass in Wisconsin caused by *Helicotylenchus* spp. and Feldmesser and Gordon (1974) reported chlorosis, stunting and lack of response to water, fertilizer, and insecticide in bluegrass in New York State related to the presence of *Tylenchorhynchus, Criconemoides*, and *Helicotylenchus*.

The present investigation of the nematode situation in turfgrass in southwestern B.C. was undertaken for two reasons: 1) Unusually large numbers of spiral nematodes (up to 300,000 per kg soil) were found in bentgrass samples from the Fraser Valley some 2 years ago. 2) In September 1976, similarly high populations of plant parasitic nematodes were found in turfgrass samples from the Vancouver area taken from a golf green which would not respond to treatment with a variety of fungicides for the control of fusarium patch disease. Careful examination of the problem green revealed that the fungus (*Fusarium nivale*) had been suppressed but the grass did not recover from the damage caused by the disease. A small trial with nematicides is being conducted on this problem green to determine whether the nematode population could be reduced and if so whether this would have any measurable effect on turfgrass health. The results from this trial are not yet available.

For purposes of the present study, turfgrass samples were collected from 12 different sites in the Fraser Valley representing golf greens, bowling greens, and playing fields within the area ranging from Chilliwack in the east to and including the city of Vancouver. The object of this paper is to report the results of the survey and to assess the importance of plant parasitic nematodes to health of turfgrass in this region.

Materials and methods

Turf samples were obtained by cutting cores with a cup-cutter (10 cm diam) about 15 cm deep. Where possible, samples were taken from areas with a history of good

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Table 1. Nematode populations (no. per kg soil) in turfgrass in southwestern British Columbia

Nematode	Site number*											
	1	2	3†	4	5	6	7	8†	9	10	11	12
Helicotylenchus	366,650	45,650	500 (A) 189,650 (B)	21,300	350	21,300	0	150 (A) 2,200 (B)	600	0	50	34,500
Criconemoides	0	750	0 (A) 0 (B)	0	0	0	41,050	10 (A) 9,400 (B)	0	0	5	0
Paratylenchus	0	0	0 (A) 0 (B)	350	2,550	0	100	0 (A) 0 (B)	13,450	0	950	0
Tylenchus	2,500	1,800	2,800 (A) 2,100 (B)	50	0	2,100	600	2,650 (A) 0 (B)	0	0	50	0
Pratylenchus	0	0	0 (A) 0 (B)	150	1,200	0	0	0 (A) 0 (B)	0	0	300	100
Ditylenchus	0	0	200 (A) 0 (B)	500	200	0	0	0 (A) 0 (B)	0	900	0	0
Heterodera	0	0	0 (A) 0 (B)	0	3,500	0	0	0 (A) 0 (B)	0	0	0	0
Tylenchorhynchus	0	0	0 (A) 0 (B)	0	0	0	0	100 (A) 12,200 (B)	0	0	0	0
Longidorus	0	0	0 (A) 0 (B)	400	0	0	0	0 (A) 100 (B)	0	0	0	0
Aphelenchoides	1,000	0	0 (A) 0 (B)	0	0	0	0	0 (A) 0 (B)	0	0	0	0

* Site key: bentgrass golf green at 1) Vancouver northwest, 2) Vancouver west, 3) Vancouver southwest, 6) Pitt Meadows, 7) Chilliwack west, 8) Chilliwack east, 11) Surrey, 12) Burnaby Mountain; bentgrass bowling green at 4) Surrey central and 9) Burnaby; bluegrass mix playing field at 5) Delta, and 10) Surrey.

† At site numbers 3 and 8, A represents a sample from an apparently healthy area, and B a sample from an area of sparse, chlorotic grass.

and poor growth for comparative purposes. These were collected in polyethylene bags and delivered to the nematology laboratory at the Vancouver Research Station for processing.

Preliminary tests with several extraction methods showed that highest numbers of nematodes were consistently recovered using a combination of the McElroy method (1972) for large species and the Jenkins centrifugal flotation method (1964) for smaller species. A 200 cc subsample was cut from the original core sample, broken up in water, and the suspension passed through 500-, 250-, and 38-um sieves. The residues from the 250- and 38-um sieves were then processed by the McElroy and the Jenkins methods respectively.

Results and discussion

Nematode counts for samples from each of 12 sites are given in Table 1. Probably the most striking feature of these results is the extremely high count of *Helicotylenchus* in some of the golf course sites. Site 1 is the golf green, referred to earlier, which failed to respond to treatment with fungicides for control of fusarium patch disease. The roots of grass plants in the damaged areas were badly discolored and the plants failed to produce new growth above ground. Normally recovery from the disease occurs within 1 to 2 weeks after treatment. In this instance, symptoms of the disease persisted for longer than 6 weeks. The high nematode count is a likely explanation. The presence of sufficiently high numbers of *Helicotylenchus* feeding on the grass roots could very well be the reason why the grass plants failed to regenerate top growth in the damaged areas. Vargas and Laughlin (1972) showed an interaction between the fungus *Fusarium roseum* and the nematode *Tylenchorhynchus dubius* in the development of symptoms associated with fusarium blight. The present observation does not suggest the same kind of interaction but one in which the nematodes contributed to the seriousness of the fungal disease by delaying grass recovery. The pathogenic effect of *Helicotylenchus* is also implicated in site 3. Sample 3A was from a relatively healthy golf course green while 3B was from a green on the same course which exhibited symptoms similar to "summer dormancy" as described by Perry et al. (1959). In mid-

summer the plants cease to grow even though they are well supplied with water and nutrients.

Another result which implicates nematodes with unhealthy turf is that for site 8. Sample 8A was taken from a healthy area of a golf green and 8B from an area of the same green in which the grass was sparse and somewhat chlorotic. Comparison of nematode counts for the two samples shows that sample 8A contained few plant parasitic nematodes whereas 8B yielded high numbers of *Criconemoides* and *Tylenchorhynchus*, both of which have been reported to contribute to turfgrass disease problems. The number of *Criconemoides* found in this sample (9,400 per kg soil) could well be high enough to have caused the damage. Safford and Riedel (1976) reported no symptoms in turf samples with fewer than 1000 per 500 ml soil, but chlorosis and dieback in one sample which yielded 8000 *Criconemoides* per 500 ml soil.

In summary, the results of this survey show that turfgrass in southwestern B.C. supports a variety of plant parasitic nematodes, most of which cause no apparent damage. A few are present in sufficiently high numbers to be suspect when disease problems arise. These are: *Helicotylenchus* (spiral nematode), *Criconemoides* (ring nematode), and *Tylenchorhynchus* (stunt nematode); the same three that Feldmesser and Golden (1974) reported to be causing chlorosis, stunting, and bare spots in bluegrsss turf in New York State. *Helicotylenchus* was found in 10 of the 12 sites and in extremely high numbers at 2 of these sites; up to six times as high as the highest numbers reported by Townshend et al. (1973) for turfgrass in Ontario. *Criconemoides* was found at three sites and was associated with an unthrifty condition of the grass in one of these. *Tylenchorhynchus* was found at one site only but, along with a high population of *Criconemoides*, was associated with unhealthy turf.

More work needs to be done before a reliable statement can be made as to importance of plant parasitic nematodes to the health of turfgrass in this region but the study has shown that at least one nematode, and possibly two others, have contributed to unhealthy turfgrass situations in the sampled areas. This does not mean that nematodes are likely to be a major problem but it does indicate that plant parasitic nematodes should not be overlooked in the diagnosis of turfgrass disease problems in this region.

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Tolerance of Venturia inaequalis to dodine in Nova Scotia[1]

R.G. Ross and R.J. Newbery

Isolates of *Venturia inaequalis* tolerant to dodine were detected following the failure of dodine to control scab in apple orchards in which it had been used successfully for many years; tolerance was confirmed by the response to dodine in artifical media of isolates of *V. inaequalis* from orchards in which dodine gave poor control of scab. The average tolerance of isolates from dodine-sprayed orchards was greater than that of isolates from orchards never exposed to dodine. The level of tolerance of isolates to dodine varied within and among orchards and appeared to be shifting towards increased dodine tolerance.

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On a découvert des isolats de *Venturia inaequalis* tolérants à la dodine dans des vergers de pomme où le produit avait été efficacement utilisé contre la tavelure pendant de nombreuses années. Cette tolérance a été confirmée par la réaction à la dodine de certains isolats de *V. inaequalis* cultivés sur substrats artificiels et provenant des vergers où le produit avait donné de mauvais résultats. La tolérance moyenne des isolats provenant des vergers pulvérisés était plus grande que celle des isolats issus des vergers témoins. Le degré de tolérance des isolats a varié au sein d'un même verger et d'un verger à l'autre, et semblait afficher une tendance à la hausse.

Tolerance of the apple scab fungus *Venturia inaequalis* (Cke.) Wint. to the fungicide dodine was first reported in western New York State in 1969 (8). Dodine failed to control apple scab in orchards in which it had previously given excellent control and these failures were most evident where dodine had been used regularly for 5-10 years. Subsequent reports (1, 10) verified that tolerance was related to intensive use of dodine and showed that isolates of the fungus from dodine tolerant orchards were less sensitive to dodine that those from nontolerant sources. Tolerance of *V. inaequalis* to dodine has recently been reported in Michigan (2) and the inheritance of dodine tolerance has been studied (1, 5).

In Nova Scotia dodine has been widely used for apple scab control since it was first recommended for use in 1961. Following the development of tolerance in other areas, apple growers in Nova Scotia were advised to report any failures of dodine to control apple scab that did not appear to be due to poor application or adverse weather. In 1973 and 1974, several growers reported poor scab control with dodine. Tests were done to determine the dodine tolerance level of isolates of *V. inaequalis* from their apple orchards and the results are reported in this paper.

Materials and methods

In 1974, isolates of *V. inaequalis* were obtained from leaves and/or fruit in orchards where dodine failed to control apple scab. Response of the isolates to dodine was determined in potato-dextrose agar which had been autoclaved, cooled to 45°C, and amended by adding a series of concentrations of dodine in ethanol. Each amended medium was poured into 6cm plastic petri dishes each containing 1 ml of the appropriate inoculum prepared by comminuting an agar slant of the isolate of *V. inaequalis* in 100 ml sterile water in a Waring Blendor. After thorough agitation the medium was allowed to solidify and the dishes were incubated at 18°C for 2 weeks. Controls consisted of media containing the same quantity of ethanol as that in which dodine was added. The concentrations of dodine in the media were approximate since in preparing the amended media no allowance was made to compensate for the 1 ml of inoculum and the quantity of amended media added to each dish was estimated by depth to be 9 ml. The dodine used was extracted from commercial grade material and recrystallized to melting point. Its potency was checked periodically by comparing it in an assay with commercial grade dodine. After 2 weeks at 18°C, growth in each dish was rated on a numerical scale of 0 to 10, 0 being no growth, 1 being dishes containing two or three isolated colonies, and 10 being maximum growth like that of the controls.

The isolates were assayed in groups of five and a Race 1 isolate of *V. inaequalis* used in previous work (6) served as a standard and was assayed each time a group was done. Assays were also done on isolates obtained from orchards in 1957 (7), prior to the introduction of dodine for the control of apple scab.

A comparison of the tolerance of isolates to dodine was also done using the agar diffusion technique (3). Filter paper (Whatman No. 1) disks 6 mm in diameter were dipped in ethanol solutions of dodine, drained uniformly, and placed on solidified potato-dextrose agar in large

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Table 1. Growth of isolates of *Venturia inaequalis* obtained from Nova Scotia orchards in 1974 and 1957 in potato–dextrose agar containing different concentrations of dodine

Isolate	Origin: Orchard	Origin: Cultivar	Growth of mycelium at the following concentrations of dodine (ppm): 0.25	0.50	1.0	1.5	2.0
1974 isolates							
1–74	A	King	10.0*	10.0	3.5	0.5	0
2–74	B	McIntosh	10.0	10.0	10.0	7.0	2.5
3–74	C	Red Delicious	9.0	8.5	3.0	2.0	1.0
4–74	C	McIntosh	10.0	10.0	3.0	1.5	1.0
5–74	C	Gravenstein	10.0	9.5	3.0	0	0
6–74	D	Gravenstein	9.0	7.0	2.5	0	0
7–74	D	Cortland	9.5	7.5	3.5	0	0
8–74	E	McIntosh	10.0	10.0	9.0	5.0	2.5
9–74	E	Red Delicious	10.0	10.0	2.5	0	0
10–74	F	McIntosh	10.0	5.0	2.0	0	0
11–74	G	McIntosh	10.0	10.0	8.0	3.0	2.0
12–74	H	Red Delicious	9.0	5.0	2.0	1.0	0
13–74	H	Gravenstein	10.0	9.0	6.5	3.5	2.5
14–74	H	McIntosh	10.0	10.0	10.0	10.0	10.0
15–74	I	Red Delicious	10.0	10.0	7.0	3.0	0.5
16–74	I	Cortland	9.0	6.0	1.5	0.5	0
17–74	I	Cortland	10.0	9.0	2.0	1.0	0.5
18–74	I	McIntosh	6.5	4.0	2.0	1.0	1.0
19–74	I	McIntosh	10.0	10.0	10.0	8.0	3.0
20–74	J	Gravenstein	10.0	10.0	8.0	4.0	1.0
21–74	J	McIntosh	10.0	3.0	1.0	0.5	0.5
22–74	K	McIntosh	10.0	9.0	5.0	0	0
23–74	L	McIntosh	10.0	10.0	6.0	2.0	2.0
24–74	L	Red Delicious	10.0	9.0	2.0	1.0	0.5
Avg 1974 isolates			9.7	8.4	4.7	2.3	1.3
1957 isolates							
1–57			8.0	5.0	1.0	1.0	0.5
3–57			9.0	7.0	2.0	1.0	1.0
4–57			0	0	0	0	0
5–57			5.0	3.5	1.0	0.5	0
6–57			9.0	5.0	1.0	1.0	1.0
7–57			1.0	0.5	0.5	0	0
8–57			10.0	10.0	6.5	3.0	2.5
10–57			10.0	5.5	3.0	1.0	1.0
12–57			2.5	1.5	0.5	0.5	0
16–57			1.0	0	0	0	0
Avg 1957 isolates			5.6	3.8	1.6	0.8	0.6
Race 1**			10.0	8.7	2.6	0.5	0

* 0, no growth; 10, maximum growth

** Average of 18 assays

glass trays. Before pouring, the agar was seeded with inoculum from an agar slant culture of the isolate under test. After 10-12 days at 18°C the diameters of the clear zones around the disks were measured.

In greenhouse tests actively growing apple seedlings (cv. Beautiful Arcade) in pots were sprayed with different concentrations of dodine up to 15 ppm. Soon after the trees had dried, they were sprayed with suspensions of *V. inaequalis* containing about 5 x 10^4 conidia/ml and held in a moist chamber for 48 h.

Results and discussion

The growth ratings (Table 1) for *V. inaequalis* in

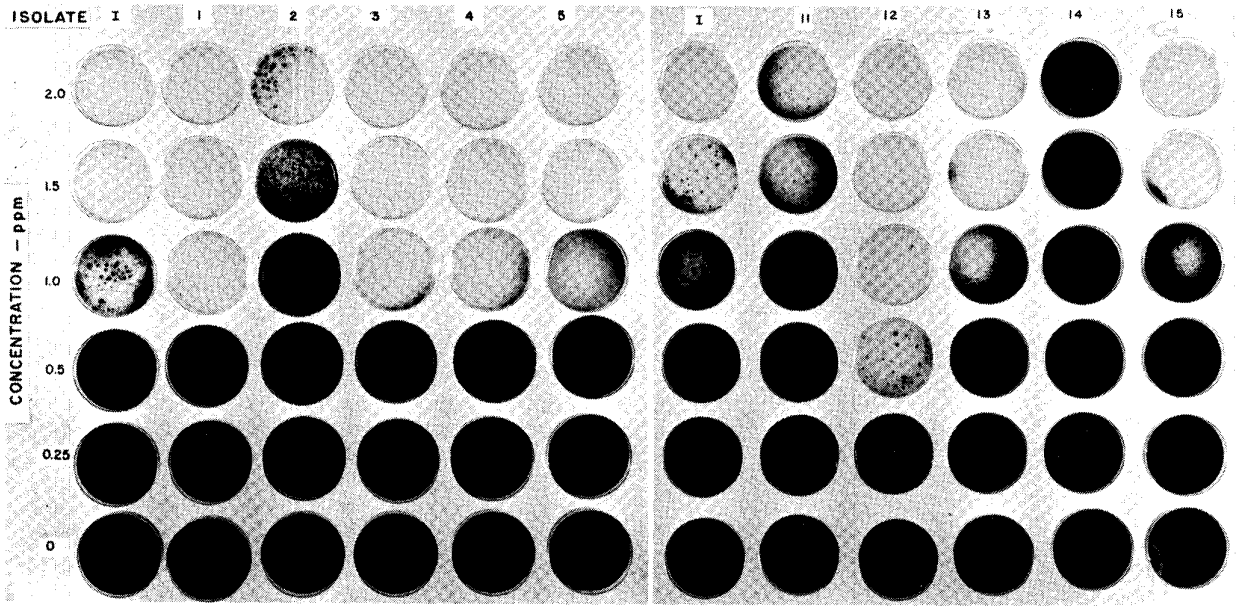

Figure 1. Growth of Race 1(I) and of 10 isolates of *V. inaequalis* obtained from Nova Scotia orchards in 1974 in media containing different concentrations of dodine.

different concentrations of dodine are the average of at least two assays for each isolate. Among both the 1974 group of isolates, from apple orchards in which dodine was not giving satisfactory control of scab, and the 1957 group, which were isolated prior to dodine use, there was considerable variation in sensitivity to dodine. On average the 1957 isolates were more sensitive to dodine than the 1974 isolates and among the latter there were isolates such as 2-74, 14-74, and 19-74 that appeared to be unusually tolerant to dodine. In the 1957 group sensitivity ranged from that of isolate 4-57 where all concentrations of dodine inhibited growth to that of isolate 8-57 which might be suspected of having developed tolerance to dodine if it had been exposed to it.

Orchards H and I were particularly suspect of having lines of *V. inaequalis* tolerant to dodine. This fungicide had been used almost exclusively for the control of apple scab for many years in both orchards and prior to 1974 had always given excellent control. Despite repeated applications of dodine in 1974, scab was abundant on the foliage and fruit and subsequent dodine sprays failed to halt its development. Isolate 14-74 from Orchard H and 19-74 from Orchard I were both tolerant to dodine in culture (Table 1). Isolate 2-74 was also tolerant in culture (Fig. 1) but poor scab control in orchard B was thought to have been due to poor timing of a spray in relation to an infection period. In Michigan, Yoder and Klos (11) obtained the most tolerant isolates from orchards in which dodine was used extensively but not necessarily from orchards with poor scab control.

The growth of isolates 2-74 and 14-74 and of Race 1 of *V. inaequalis* was compared at dodine concentrations up to 10 ppm and the results in Table 2 are the average of the assay repeated four times. A further comparison was done by the agar diffusion technique using disks dipped in dodine, and the results given in Fig. 2 are the average of three assays done at different times. Isolate 2-74 grew in media containing up to 4 ppm dodine and isolate 14-74 up to at least 10 ppm, whereas Race 1 was inhibited by 3 ppm. The disk assay gave the same contrast between the orchard isolates and Race 1 but did not distinguish the same degree of tolerance between isolates 2-74 and 14-74.

The inoculation experiment in the greenhouse did not give conclusive results. Isolate 14-74 did not infect sprayed or unsprayed apple seedlings. In a comparison of Race 1, isolate 2-74, and isolate 19-74 with concentrations of dodine up to 15 ppm, there were no significant differences in control and all lines caused infection at 15 ppm. Szkolnik and Gilpatrick (9) differentiated between sensitive and tolerant isolates at this level of dodine.

It seems unlikely that the sensitivity of field isolates of *V inaequalis* to dodine in culture can be used to determine whether dodine will fail in the orchard because of tolerance. Yoder and Klos (11) obtained tolerant isolates in orchards with good scab control, and in Nova Scotia there was considerable variation in sensitivity among isolates from the same orchard (Table 1). By taking sufficient isolates the 14-74 level of tolerance to dodine

Table 2. Growth of three isolates of *Venturia inaequalis* in media containing different concentrations of dodine

	Concentration of dodine (ppm):									
Isolate	1	2	3	4	5	6	7	8	9	10
2–74	10.0*	8.2	3.7	1.2	0	0	0	0	0	0
14–74	10.0	10.0	10.0	7.2	5.0	3.5	2.5	2.2	1.5	0.5
Race 1	6.0	0.5	0	0	0	0	0	0	0	0

* 0, no growth; 10, maximum growth

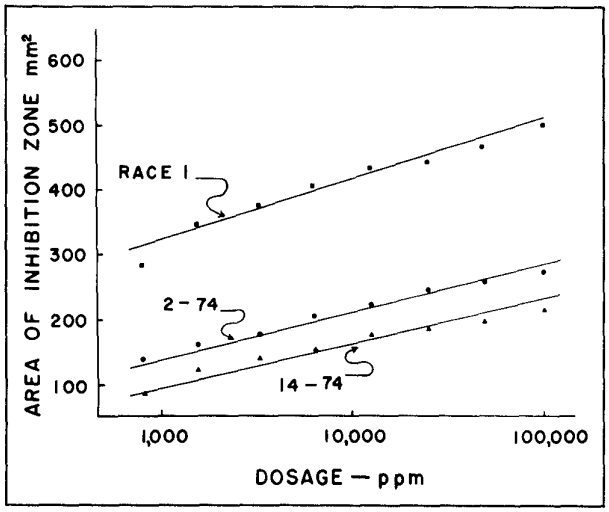

Figure 2. Dosage-response curves for dodine with three isolates of *V. inaequalis*.

might have been obtained in any of the orchards. According to MacNeill and Schooley (4) tolerance in *V. inaequalis* may evolve by adaptation and/or mutation. In culture they obtained a mutation rate of about 1 in 10^6. The results reported here (Table 1) suggest that the average tolerance to dodine has increased with the use of the fungicide in Nova Scotia; this supports the prediction of MacNeill and Schooley that the ecological balance might shift in favor of dodine tolerant strains within populations of *V. inaequalis*. The differences in the two groups of isolates (1957 and 1974) were not as decisive as those Polach (5) reported for isolates from dodine sprayed orchards and isolates from abandoned orchards.

The presence in Nova Scotia of tolerance in *V. inaequalis* to dodine poses a problem for apple growers. In addition to being a good protectant and eradicant or after-rain fungicide for apple scab control, dodine has been used to burn out established scab lesions. The effect of tolerance on its eradicative and burning out properties is not known. The owner of orchard A (Table 1) considered that dodine was no longer effective for the latter purpose. The incidence of tolerance to dodine in Nova Scotia orchards may never be known because growers are changing to other scab fungicides rather than waiting for tolerance to become evident.

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Bacterial blight of field bean: disease progress, yield loss, and crop canopy development in principal cultivars in Ontario[1]

V.R. Wallen and D.A. Galway

Similar disease progress curves were developed from the results of disease assessments for bacterial blight [*Xanthomonas phaseoli*] on Sanilac, Seafarer, and Kentwood field bean [*Phaseolus vulgaris*] cultivars in field plots over a 3-year period 1974-76. Although losses due to bacterial blight were substantial, mean 3-year losses were similar among the varieties, Seafarer 32%, Kentwood 32.2%, and Sanilac 33.1%. Crop canopy development was influenced by the onset and intensity of disease, a hastening of maturity and senescence, and early defoliation of infected plants. In 1975, when yield losses were greatest, differences in canopy development between control plots and infected plots were also the greatest.

Can. Plant Dis. Surv. 57: 61-64. 1977

Les résultats de l'évaluation des effets de la brûlure bactérienne [*Xanthomonas phaseoli*] sur les cultivars de haricot sec (*Phaseolus vulgaris*) Sanilac, Seafarer, et Kentwood, cultivés en parcelles pendant 3 ans (1974 à 1976), ont donné des courbes analogues d'évolution de la maladie. Bien que les pertes de récolte étaient appréciables, les pertes moyennes entre les variétés ont été pratiquement comparables, soit 32, 32.2 et 33.1% pour Seafarer, Kentwood et Sanilac respectivement. Le déclenchement et l'intensité de la maladie, l'accélération de la maturité et de la sénescence, ainsi que la défoliation précoce des plants infectés, ont influé sur la densité du feuillage. C'est en 1975 (année de baisse maximale de rendement) que les différences dans la densité du feuillage des parcelles témoins et infectées ont été les plus marquées.

Sanilac, Seafarer, and Kentwood are now the principal white field bean cultivars grown in southwestern Ontario. Sanilac was released in 1957, Seafarer in 1969, and Kentwood in 1973. The cultivar Seafarer accounts for approximately half of the white bean acreage in southwestern Ontario. All three cultivars are susceptible to common blight and fuscous blight caused by *Xanthomonas phaseoli* (E.F. Sm.) Dows. and *Xanthomonas phaseoli* var. *fuscans* (Burkh.) Starr and Burkh., respectively, but little is known regarding the relative degree of susceptibility of each cultivar to the two blight organisms.

Initially when bacterial blight became a serious problem in Ontario following the introduction of Sanilac, field surveys were conducted (2, 3, 8) that revealed the presence of the heretofore unreported fuscous blight as well as a higher incidence of common blight in Sanilac than in the older cultivar Michelite. The diseases are similar in symptom expression, and in culture the causal bacteria differ only in the production of a pigment (8) by the fuscans organism. Michelite gradually disappeared from the scene because of its susceptibility to anthracnose. At present, bacterial blight is at a tolerable level as determined by ground surveys and as monitored by aerial infrared photography (4, 5, 6). However the quantitative differences in disease incidence and yield loss among the three cultivars are not known.

This study was conducted to compare disease progress among the three cultivars throughout the season, to assess the effects of the disease on yield loss and crop canopy development as shown by sequential aerial infrared photography, and to establish any quantitative differences among the cultivars with respect to susceptibility to blight. Percent ground cover has been utilized to assess growth, and to determine maturity, vigour, and yield potential (1). It was thought that this technique might detect differences in susceptibility not determined by other means.

Methods and materials

Field tests

The experiment was carried out for 3 years, 1974-76. The experimental design consisted of a randomized block 54.9 x 64 m (160 x 190 ft) containing 24 plots each measuring 4.6 x 7.6 m (15 x 25 ft). Each plot contained 7 rows 76 cm (2.5 ft) apart and 7.6 m (25 ft) long. There were eight plots of each cultivar, four inoculated with a mixture of isolates of *Xanthomonas phaseoli* var. *fuscans* and *Xanthomonas phaseoli* and four plots left untreated. There was a 6.1 m (20 ft) roadway between plots.

Seeding was carried out each year during the last week of May; however in 1974 the plots had to be reseeded on June 12 because of low emergence caused by seed corn maggot injury. Because of the late seeding in 1974 inoculation was delayed until July 16; as a result fewer disease assessments were made and harvest was

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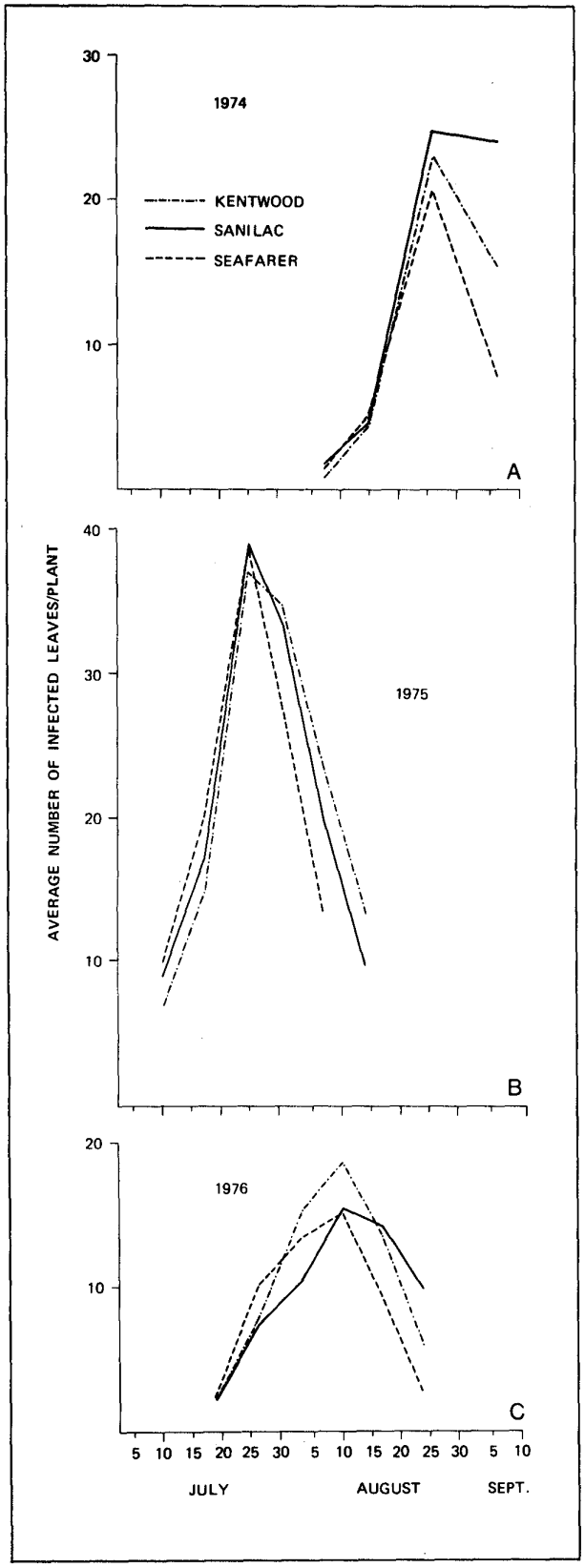

Figure 1. Progress of bacterial blight throughout the growing season, 1974-76, based on the weekly assessment of the number of infected leaves on Sanilac, Seafarer, and Kentwood.

delayed until the last week of September. After emergence, plots were thinned out so that each plot contained an equal number of plants. This was possible in 1975 and 1976 but not in 1974 because of low emergence in the cultivar Sanilac.

Inoculum was prepared by mixing equal numbers of suspended cells of the two organisms of common and fuscous blight. Plants were inoculated at the 3 to 4 leaf stage 3 weeks after planting by applying an aqueous suspension of the two organisms with a knapsack sprayer.

Beginning with the first evidence of disease, the number of infected leaves per plants was determined at weekly intervals throughout the growing season. Ten plants per plot selected at random were tagged so that the same plants were assessed each time.

In 1974 four assessments were made from August 7 to September 6. In 1975 and 1976, six assessments were made from July 10 to August 14 and July 19 to August 24, respectively. The number of infected pods per tagged plant was recorded. For seed weight the outside row on each side of the plots was discarded as well as 76 cm (2.5 ft) of row at each end of the plots. Weights were recorded 1 month after harvest.

Ground cover measurements (canopy development)

Ground cover measurements were made according to the method of Gerbermann et al. (1) utilizing sequential aerial photography. The photography was taken weekly on the day the plants were assessed for disease or as close to that day as possible depending on weather conditions. A Bell-47C helicopter at an altitude of approximately 121.9 m (400 ft) was used for all flights. Exposures were made with a boom-mounted Maurer 70 mm camera positioned for vertical photography. The camera was fitted with a 38-mm Biogon f/4.5 lens, which produced an approximate scale of 1:1,670 (i.e. size of image to distance on ground). Exposing rate was six frames per second and the air speed was approximately 40 knots. Kodak Aerochrome Infrared 2443 film was used throughout the 3 years.

Results and discussion

Disease progress

The three cultivars showed similar disease patterns each year (Fig. 1 A-C). In all 3 years the number of infected leaves reached a maximum during August followed by the early defoliation of heavily infected leaves. This explains the peaking effect in Fig. 1 A-C. Infection levels were consistent for each cultivar, but maximum infection levels differed considerably in the 3 years. Average

Table 1. Comparative Susceptibility of Kentwood, Sanilac, and Seafarer field beans to pod infection by bacterial blight, 1974-76

Year	Cultivar	Avg no.* pods/plant	Avg no. pods infected No./plant	%
1974	Kentwood	28.3	3.4	11.8
	Sanilac	55.2	7.8	14.1
	Seafarer	34.7	4.6	13.3
1975	Kentwood	17.7	14.8	83.5
	Sanilac	23.9	20.9	87.3
	Seafarer	24.1	19.2	79.8
1976	Kentwood	28.8	6.0	20.9
	Sanilac	29.6	8.2	27.8
	Seafarer	23.1	5.4	23.2

* Based on 4 reps of 10 plants/rep.

Table 2. Comparative yield losses of Kentwood, Sanilac, and Seafarer field beans caused by bacterial blight, 1974-76

		1974		1975		1976		Average	
Cultivar	Treatment	Yield†	% Loss	Yield	% Loss	Yield	% Loss	Yield	% Loss
Kentwood	Control	2995.3	6.0	2807.3	62.2	3673.6	30.7	3158.5	32.2
	Infected	2814.0		1061.1		2545.4		2140.4	
Sanilac	Control	2471.5	28.8	2491.6	50.9	3785.1*	19.6	2916.1	33.1
	Infected	1759.6		1222.3		3042.3		2008.1	
Seafarer	Control	3619.9	22.1	2914.7	55.8	2485.6	22.4	3339.9	32.0
	Infected	2820.7		1289.5		2706.5		2272.0	

† Kg/hectare.

* Excludes 2 plots which had extensive groundhog damage.

infection levels in 1976 were the lowest, with 15 to 19 infected leaves per plant, while in 1975 they approached 40 leaves per plant.

The average number of infected pods at harvest (Table 1) varied considerably from year to year but was consistent among cultivars within years. The higher number of pods per plant in Sanilac in 1974 may be explained by the lower emergence of that cultivar, resulting in larger plants and an increased number of pods. In 1974, corresponding to lower yield losses, the average percentage of pods infected varied between 11.8% and 14.1% among the three cultivars; in 1975, corresponding to higher yield losses, infection ranged from 79.8% to 87.3%. In 1976 infection varied from 20.9% to 27.8%.

Yield loss

Over the 3-year period there were very slight differences in mean yield losses among the three cultivars (Table 2). Mean 3-year losses however were substantial, Seafarer 32%, Kentwood 32.2%, and Sanilac 33.1%. In 1975 we reported a yield loss factor of 38 (7), which is slightly higher than the losses reported here. Considerable variability in environmental factors resulted in different year to year losses; 1974, 6.0-22.1%; 1975, 50.9-62.2%; 1976, 22.4-30.7%.

The increase in losses and in number of leaves infected in 1975 may possibly be explained by the higher mean temperature in July (22.4°C) and also the higher total rainfall in July of over 10 cm. The mean temperature in

Table 3. Influence of bacterial blight on growth of field beans determined from ground cover measurements from sequential aerial photographs of field plots

	Percent ground cover					
	Kentwood		Sanilac		Seafarer	
Date	Control	Infected	Control	Infected	Control	Infected
1974						
7 Aug.	75.7	81.2	76.4	55.6	89.6	86.1
16 Aug.	91.6	93.1	84.0	67.4	91.6	84.0
30 Aug.	98.6	97.2	91.6	76.4	95.0	88.0
5 Sept.	100.0	96.5	92.4	70.1	99.3	95.8
16 Sept.	97.9	92.4	87.5	60.0	88.9	72.2
1975						
10 June	28.3	26.3	23.1	25.8	26.6	26.8
20 June	38.4	38.4	38.4	37.0	41.9	43.2
27 June	49.8	52.0	52.2	56.1	54.0	49.4
22 July	88.4	82.8	95.4	91.5	85.2	80.5
31 July	90.8	76.7	96.2	90.9	83.8	75.6
7 Aug.	86.8	68.0	91.2	83.0	73.9	58.7
1976						
24 June	16.2	20.4	17.7	16.8	15.8	17.5
7 July	54.5	55.8	49.0	50.4	52.3	55.4
15 July	81.4	83.4	77.9	82.2	79.7	82.3
21 July	92.3	93.1	94.4	90.9	93.1	90.3
28 July	95.1	95.2	94.4	94.4	95.1	88.2
4 Aug.	95.1	93.0	99.1	95.8	93.8	86.6
11 Aug.	96.5	93.0	100.0	97.2	93.8	86.1
18 Aug.	97.9	89.6	97.2	88.2	91.0	81.9
30 Aug.	97.2	84.0	95.8	84.0	88.2	73.6

July 1974 and 1976 was between 19.4°and 20°C and rainfall was less than 7.6 cm. The August mean temperature was slightly higher in 1975 than in the other years but precipitation was lower. Surprisingly, relative humidity was lower (66%) in 1975 than in 1974 (74%) or 1976 (76%). In all probability, mean temperature and relative humidity have less effect on the development of an epiphytotic than the number and extent of favorable periods for the initiation and spread of the disease.

Ground cover

In all 3 years crop ground cover or canopy development was influenced by disease onset and intensity of disease (Table 3). In 1975, when yield losses and disease intensity were greatest, differences in canopy development between control and infected plots were also the greatest. During the period June 10 to June 27, when infection was at a low level, canopy size was similar in both the control and infected plots. Following the peak in infection level on July 22, control canopies continued to increase in size whereas the canopies of the infected plots of the three varieties decreased. By August 7 the average size of the control canopies was 15% larger than that of the infected plots. In 1974, when losses were smaller, canopy development in control and infected plots continued almost parallel until harvest for Kentwood, which showed only a 6% yield loss. Wide differences occurred in canopy development between the control and infected plots in Sanilac, which showed the greatest loss. Seafarer, in which loss was intermediate, did not show as great a difference in canopy development as Sanilac but had a canopy difference of over 16% on September 16. In 1976, canopy differences were not apparent, except for Seafarer, until August 18; by August 30 canopy differences between control and infected plots ranged from 12% to 15%.

Over the 3 years the consistently smaller canopy development in infected plots in relation to control plots was due to a number of factors: a general slowing down of growth in infected plots during and following the onset of infection; a hastening of maturity and senescence of infected plants, which were harvested 4 to 7 days before those in control plots; and defoliation due to drying out of severely affected leaves.

From these results, it is apparent that Sanilac, Seafarer, and Kentwood are equally susceptible to bacterial blight and that losses of equal magnitude can be expected. The importance of producing disease-free Breeder seed for pedigreed and commercial bean production is therefore emphasized; it is also essential that monitoring of seed through the Select and Foundation grades be continued.

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Blackpatch of forage legumes

B. Berkenkamp[1]

In 1975 blackpatch, a disease of forage legumes caused by *Rhizoctonia leguminicola*, was found at Lacombe, Alberta, for the first time in Canada. Naturally infected red clover (*Trifolium pratense*), sainfoin (*Onobrychis viciifolia*), and cicer milkvetch (*Astragalus cicer*) have been found in the field, and five other legume species have proven susceptible in inoculation tests; *O. viciifolia* and *A. cicer* are new host records. The toxicity of fungus mycelium and culture filtrate to mice was examined but results were inconclusive.

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La "plaque noire", maladie des légumineuses fourragères causée par *Rhizoctonia leguminicola*, a été signalée pour la première fois au Canada dans la région de Lacombe, en Alberta. Le trèfle rouge (*Trifolium pratense*), le sainfoin (*Onobrychis viciifolia*) et l'astragale pois chiche (*Astragalus cicer*) infestés naturellement on été trouvés dans le champ, et cinq autres espèces de légumineuses se sont révélées sensibles lors des essais d'inoculation; *O. viciifolia* et *A. cicer* ont été identifiées comme étant de nouvelles plantes hôtes. On a étudié la toxicité du mycélium fongique et du filtrat de culture sur les souris, et les résultats obtenus n'ont pas été concluants.

The blackpatch fungus, previously unreported in Canada, was isolated at Lacombe, Alberta, from lesions on leaves of red clover and sainfoin in 1975, and of cicer milkvetch in 1976. The disease is suspected to occur in British Columbia on white clover (D. J. Ormrod, personal communication, 1975). Blackpatch was first reported on red and white clovers in 1933 from Kentucky (1). The name refers to the appearance of affected patches in a field, not to symptoms on individual plants. Smith (8) in Wisconsin gave a detailed description of the disease, the host range, and the fungus. Weimer (9) described blackpatch on soybeans, cowpea, kudzu, and blue lupine, while Wells (10) reported the blackpatch fungus causing blight of leaves and stems of big trefoil (*Lotus uliginosis* Schkuhr.) in Georgia. The fungus was described as *Rhizoctonia leguminicola* by Gough and Elliott (5), who reviewed its distribution in the eastern U.S.A. Symptoms of its toxicity to animals have been described as excessive salivation, loss of appetite, frequent urination and defecation, piloerection and lacrimation (3, 6), and the active principle (slaframine) has been characterized (2, 4, 7).

Materials and methods

The host range of the blackpatch fungus was tested with an isolate obtained from sainfoin leaf lesions at Lacombe, Alberta, in 1975. Cultures of the fungus were grown in flasks of malt-yeast broth for 10 days; the mycelium was drained, ground in a Waring blender with distilled water, and sprayed onto potted greenhouse grown plants of the legumes cicer milkvetch (*Astragalus cicer* L.), red clover (*Trifolium pratense* L.), sainfoin (*Onobrychis viciifolia* Scop.), alfalfa (*Medicago sativa* L.), alsike clover (*Trifolium hybridum* L.), white clover (*Trifolium repens* L.), sweet clover (*Melilotus alba* Desr.), and birdsfoot trefoil (*Lotus corniculatus* L.). The inoculated plants were kept in a dew chamber at near saturation for 2.5 days at 22°C. Symptoms were described and severity estimated 7 days after inoculation. Detached leaflet inoculations were made by placing small pieces of fungus from a potato dextrose agar (PDS) culture onto four detached leaflets of each species supported on moist filter paper in petri dishes. Severity was estimated 9 days after inoculation.

Animal toxicity tests were carried out by mixing equal parts by weight of dried fungus mat with regular feed, or by moistening feed with culture filtrate, 0.5 ml/g. Injections were prepared using twenty-one 200 ml flasks containing a total of 750 ml of malt-yeast broth. The flasks were inoculated with the blackpatch fungus and incubated at room temperature for 2 weeks. The fungal mats were then ground wet and extracted in a separatory funnel with chloroform by a method similar to that used by Rainey et al. (7). The residue remaining after evaporation of the chloroform layer was suspended in 2.0 ml of sterile distilled water. Two test mice were given 0.5 ml, and two were given 0.25 ml intraperitoneal injections of fungal extract. Three control mice were injected with 0.5 ml distilled water and one was not injected.

Results and discussion

In PDA culture the colonies of the blackpatch fungus are at first white, becoming dark brown to black, coarse, and forming small sclerotia-like bodies. On leaves incubated under moist conditions in petri dishes, long dark aerial hyphae spread over the leaves and the supporting filter paper (Figure 1). We have not encountered other fungi that form such obvious dark aerial hyphae and consider this diagnostic for blackpatch.

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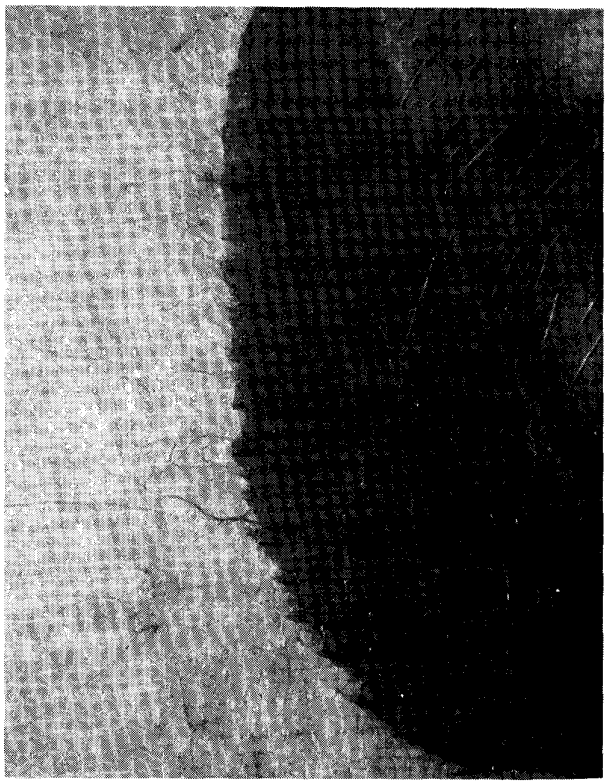

Figure 1. Detached white clover leaf inoculated with the blackpatch fungus and incubated on moist filter paper in a petri dish, showing dark hyphal strands, X 7.

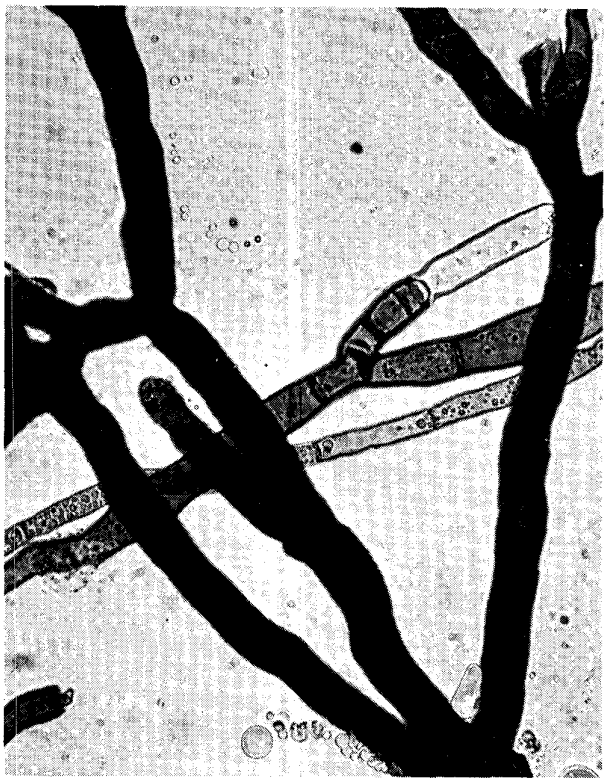

Figure 2. Hyphae of the blackpatch fungus, potato dextrose agar culture, X 330.

The hyphae (Figure 2) vary from 7 to 15 μm in diameter, some being straight walled and some constricted at the septa. The color ranges through hyaline, amber, dark brown, to black, but no green color was noted. Occasionally large, up to 45 μm diameter, swollen chlamydospore-like cells without thickened walls were found in the hyphae.

Sclerotia-like bodies are composed of loose branching hyphae with short swollen cells, and are not differentiated into layers. The older, highly vacuolate hyphae usually break smoothly at the septations, but occasionally roughly torn breaks are found between septa. Except for the pseudosclerotia, the hyphae are usually dichotomously branched. The fungus resembles the descriptions of *R. leguminicola* by Smith (8) and Gough and Elliott (5), except that it lacks green pigment and the hyphae are slightly smaller in diameter. According to R. A. Shoemaker and L. K. Weresub (personal communication), this fungus does not belong in the genus *Rhizoctonia*. However the name *R. leguminicola* is used here because a more appropriate name is not available at present. A specimen of the fungus from *Astragalus cicer* has been deposited in the National Mycological Herbarium, Ottawa, Ont., as DAOM 155489.

In the host range test all of the legumes tested were affected to varying degrees. Table 1 shows severity ratings on hosts, as well as symptoms produced by artificial inoculation. Naturally infected plants in the field did not show the light gray areas seen on inoculated plants in the greenhouse. In the field, diseased areas on leaves were dark brown and difficult to distinguish from the symptoms caused by *Stemphylium sarcinaeforme*, as mentioned by Smith (8). Birdsfoot trefoil was infected artificially in the greenhouse, but was not found to be infected in the field. However sainfoin and cicer milkvetch were found to be infected in the field and these are new host records for blackpatch.

The addition of fungal mats and filtrate to feed and the force feeding of ground cultures or filtrate to mice resulted in inconsistent and indistinct symptoms. Intraperitoneal injections of aqueous suspensions of the residue from the chloroform extract of fungal mats resulted in mild symptoms of griping and piloerection about 0.5 h after injection. The treated mice recovered after 4 h and no fatalities occurred. Excessive salivation symptoms were not observed, and mice may be similar to rats, which do not show this symptom (4).

Table 1. Disease severity on inoculated detached leaflets and entire plants, and description of symptoms caused by the blackpatch fungus in greenhouse tests

Host and cultivar	Severity*		Symptoms on entire plants spray-inoculated in greenhouse
	Detached leaflets	Entire plants (avg of 3)	
Alfalfa, Beaver	4	3.0	Pale tan patches with dark margins
Alsike clover, commercial	4	2.7	Small circular brown lesions ("pepperspot") with chlorotic halos, to larger brown spots containing concentric rings
Cicer milkvetch, Oxley	4	4.3	Gray to brown irregular patches, some with dark margins; also dark brown very small spots; occasional young shoot "blasted"
Birdsfoot trefoil, Leo	3	2.0	Very small, brown, circular spots
Red clover, Altaswede	2	2.7	Gray circular patches containing concentric rings ("target" spots); also some very small dark brown spots
Sainfoin, Melrose	5	2.7	Numerous small brown spots
White clover, commercial	2	2.7	Gray to light brown spots varying in size from small to quite large
Sweet clover, Arctic		2.7	Gray to light brown circular patches often with darker brown centers; some with chlorotic halos

* Disease rating: 1 (healthy) to 5 (severely diseased).

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Soybean phytophthora rot in southwestern Ontario

R. I. Buzzell, J. H. Haas, L. G. Crawford, and O. Vaartaja[1]

Phytophthora megasperma var. *sojae* was isolated from dying soybean (*Glycine max*) plants and characterized as to race (1 to 6) using as differentials the cultivars Harosoy, Harosoy 63, Mack, Altona, and Sanga. Most of the 200 isolates obtained during 1973-76 were races 3 or 6 (90%) whereas 9% were races 4 or 5. Race 1 was obtained but race 2 was not found. Twelve of 71 fields contained more than one race. Additional testing of some of the race 6 isolates on the soybean lines PI 103.091 and PI 171.442 showed that races 7 and 8 are also present in southwestern Ontario.

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Phytophthora megasperma var. *sojae* a été isolé de plants moribonds de soja (*Glycine max*) et des races (de 1 à 6) ont été identifiées à l'aide de différents cultivars de soja (Harosoy, Harosoy 63, Mack, Altona et Sanga). Les races 3 et 6 ont été retrouvées dans la plupart (90 %) des 200 isolats recueillis au cours de la période 1974-1976, tandis que les races 4 et 5 n'étaient observées que dans 9 % d'entre eux. On a obtenu la race 1 mais pas la race 2. On a retrouvé au moins 2 races dans quelques-uns des 71 champs. Les analyses supplémentaires portant sur quelques isolats de la race 6 recueillis dans les parcelles de soja PI 103.091 et PI 171.442 ont montré que les races 7 et 8 étaient également présentes dans le sud-ouest de l'Ontario.

Phytophthora megasperma (Drechs.) var. *sojae* A.A. Hildebrand (*Pms*) incites a root and basal stem rot of soybeans (4). The disease was important in southwestern Ontario and in the major soybean growing region of the north central U.S.A. from about 1954, when it was first discovered, until shortly after 1963 when resistant isolines of agronomically acceptable cultivars were released to growers. These new cultivars contained the Rps_1 gene (1) which conferred resistance to what has been subsequently called race 1 of *Pms*.

From about 1965 onward, essentially the entire soybean crop in Ontario was planted with cultivars resistant to race 1. We received occasional reports of plant killing but *Pms* was not isolated from the plants; *Rhizoctonia* sp. was recovered and presumed to be the incitant. In 1973, *Pms* was isolated from Rps_1 cultivars in Ontario. Further work showed that the isolates were different from race 1, race 2 (7), race 3 (8), and race 4 (9); and they have been reported as races 5 and 6 (3). By the latter part of the 1974 growing season about 50 reports had been received of diseased soybeans on the clay soils in southwestern Ontario. We have attempted to obtain an estimate of the prevalence of new pathotypes in this area; a portion of this work has been published (2).

Materials and methods

Soil was collected from around dead soybean plants in 33 fields in Essex, Kent, and Lambton counties (4 in 1973 and 29 in 1974). The soils were placed in 30-cm-diameter fibre pots in a greenhouse. Up to six repetitive plantings of each of four soybean strains (Amsoy, Altona, Harosoy, OX20-8) were made in each soil and isolations made from dying plants. From any strain grown in each soil no more than five *Pms* isolates were saved for testing; in many cases the number recovered was much less than five.

In 1975 a survey was made of the major soybean-producing areas in Essex (including Pelee Island) and Kent counties. Depending upon the frequency of soybean crops in an area, one field per 5 to 10 km of road travelled was checked. Where plant killing was found, dying plants were collected and isolations made from them. We tried to obtain one culture from each of two affected plants in each field. Diseased plants were obtained from eight fields in 1976 in response to grower queries.

All isolations were made from stems of dying soybean plants. They were surface sterilized in 0.5% sodium hypochlorite; seedling hypocotyls were cross-sectioned at the margin of lesions, and woody stems were split and a sliver of stelar tissue removed. The plant pieces were placed on Difco corn meal agar (CMA) amended with 100 ppm pimaricin. Generally *Pms* was the only fungus which grew on the medium but bacterial contaminants were common. Pure cultures were obtained by inverting the agar 4 to 5 days after plating and removing mycelium which had grown through the agar layer.

All cultures were stored on CMA slants under mineral oil at 18-22° C. A set of differential soybean cultivars, Harosoy, Harosoy 63, Mack, Altona, and Sanga, were inoculated by the hypocotyl-puncture method of testing virulence of *Pms* (5). Mycelium grown in agar (3) was used for testing the 1973-74 isolates and mycelium grown in broth was used for the 1975-76 isolates. The pots containing inoculated plants were covered with

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Table 1. Number of isolates (N=200) of *Phytophthora megasperma* var. *sojae* classified into races 1 to 6 in southwestern Ontario during 1973 through 1976

	No. of isolates of races					
Year	1	2	3	4	5	6
1973	0	0	1	0	4	15
1974	0	0	2	10	0	84
1975	2	0	13	3	0	51
1976	0	0	4	1	0	10
Total	2	0	20	14	4	160

polyethylene bags, placed in subdued light for 3 to 4 days and then uncovered. Two to 16 hours after uncovering, plants with rotted hypocotyls and wilted leaves were considered susceptible. A minimum of eight plants per differential strain were tested. If more than 20% of the seedlings gave an anomalous reaction, the testing was repeated.

Results and discussion

Five soybean differential varieties, Harosoy, Harosoy 63, Mack, Altona, and Sanga, are used in distinguishing races 1 to 6 (3); and the additional use of the soybean lines PI 103.091 and PI 171.442 will distinguish races 7, 8, and 9 (6). Inoculation of the full set of differential strains with some of our race 6 isolates indicated that races 7 and 8 are present in southwestern Ontario. Inoculation tests comparing those isolates with type cultures of races 7 and 8 confirmed this. However, Mack is resistant to races 1, 2, 3, 6, 7, 8, and 9, but is susceptible to races 4 and 5. Since the Mack type of resistance is being used in breeding projects, races 4 and 5 are of significance. And since races 7, 8, and 9 behave similarly to race 6 on Mack, they are not of immediate concern and need not be classified separately.

During the period 1973 through 1976, 200 *Pms* isolates were classified into races 1 to 6 (Table 1). Ninety percent of the isolates were races 3 or 6, and 9% were races 4 or 5. Race 1 was found in only one field, and race 2 was not found.

Races 3 and/or 6 were obtained in the majority (86%) of the 71 fields, but races 4 or 5 were obtained from 11% of the fields (Table 2). Only races 1, 3, and 6 were obtained from Kent and Lambton counties; however, more extensive sampling is required in those counties. As a conservative estimate, races 4 or 5 were present in 16% of the fields tested in Essex county; this variability in *Pms* will be of significance when cultivars resistant to races 3 and 6 are brought into production.

Table 2. Distribution of *Phytophthora megasperma* var. *sojae* races by fields (N=71) in counties and townships of southwestern Ontario, 1973–76

	No. of fields with races						
Township	1	3	4	5	6	3 & 6	4 & 6
Essex County							
Anderdon	0	0	1	0	2	0	0
Sandwich W.	0	0	0	0	1	0	0
Sandwich S.	0	0	0	0	1	0	0
Maidstone	0	1	1	0	3	1	2
Colchester N.	0	0	0	0	9	1	0
Colchester S.	0	1	0	1	5	0	1
Gosfield S.	0	0	1	0	1	0	0
Gosfield N.	0	0	0	0	2	1	0
Rochester	0	0	0	0	4	0	0
Tilbury N.	0	1	0	0	1	0	0
Tilbury W.	0	0	0	0	3	0	0
Mersea	0	1	0	0	3	0	1
Pelee Island	0	1	0	0	1	0	0
County total	0	5	3	1	36	3	4
Kent County							
Romney	0	0	0	0	1	0	0
Tilbury E.	1	1	0	0	3	4	0
Raleigh	0	1	0	0	2	0	0
Howard	0	0	0	0	1	0	0
Orford	0	0	0	0	1	0	0
County total	1	2	0	0	8	4	0
Lambton County							
Sombra	0	0	0	0	1	0	0
Enniskillen	0	0	0	0	1	1	0
Brooke	0	0	0	0	1	0	0
County total	0	0	0	0	3	1	0
Total	1	7	3	1	47	8	4

Acknowledgements

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Weeping willow blight in coastal British Columbia[1]

J.T. Hill[2], D.J. Ormrod[2], and R.J. Copeman[3]

Surveys and laboratory studies were undertaken during the summers of 1973 and 1974 to determine the etiology of a blight disease of weeping willow (*Salix babylonica*) in south coastal British Columbia. Of 102 trees surveyed, 70 were infected with *Marssonina salicicola*, 53 were infected with *Pleurophomopsis salicicola*, and 21 with *Cytospora* sp. The validity of earlier host and pathogen reports for the area is questioned.

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Des contrôles sur le terrain et en laboratoire ont été réalisés au cours des étés 1973 et 1974 pour établir l'étiologie de la brûlure du saule pleureur dans la zone côtière du sud de la Colombie-Britannique. Sur les 102 arbres examinés, 70 étaient infectés par *Marssonina salicicola*, 53 par *Pleurophomopsis salicicola* et 21 par *Cytospora* sp. Ces observations remettent en question la validité des rapports antérieurs sur l'identité des organismes pathogènes en cause et celle de leurs hôtes dans cette région particulière.

Willow blight is a major disease of ornamental willows in British Columbia, particularly the weeping willow *Salix babylonica* L. As a result, nurserymen are reluctant to grow weeping willows although a substantial and steady market does exist, particularly for parks and other areas where large, spreading trees are suitable.

Willow blight is a worldwide disease occurring wherever willows are grown. There is no consensus on the primary causal agents, however. Probably the most import components of the disease on a global scale are the scab fungus *Pollacia saliciperda* (All. & Tub.) Arx, and the black canker fungus *Physalospora miyabeana* Fukushi. The relative importance of the two fungi as primary parasites remains unresolved after 50 years of intermittent study (2, 4, 15, 18, 23, 25, 26).

In Canada the diseases caused by these two fungi were first observed in Nova Scotia and Prince Edward Island in the mid 1920's (17). Since then both fungi have been reported numerous times on *Salix* spp. including *S. babylonica* (9, 10, 12, 27). Both fungi were associated with the disease in coastal British Columbia in 1940 and 1941 on a few willows that were not identified to species (5, 6, 7). All of the diseased trees were destroyed, presumably in an attempt to eradicate the disease from the area. Since then the only published reports of the two fungi in coastal British Columbia were from southern Vancouver Island in 1960 and 1961 (11, 12).

Another fungus, *Marssonina kriegeriana* (Bres.) Magn. has frequently been reported as causing anthracnose and twig blight of *S. babylonica* L. in British Columbia (5, 8, 13, 14, 28). *M. kriegeriana* has also been reported on *Salix* spp. elsewhere in the world, although *M. salicicola* (Bres.) Magn. is more commonly associated with disease of *S. babylonica* L. (6, 20, 22, 24, 25).

Numerous other fungi, including *Cytospora* spp., have also been reported on *Salix* spp. but they appear to be of minor importance compared to the above named fungi (6, 24).

A bacterial blight caused by *Pseudomonas saliciperda* is reported to be important in Oregon although documentation of this pathogen is obscure (21).

Materials and methods

In order to clarify the current local situation, surveys were undertaken in the summers of 1973 and 1974. In 1973, specimens were collected only from weeping willows in nurseries in the Fraser Valley. In 1974, the survey was extended to include private homes and public areas. A sample consisted of 2-4 leafed-out branches taken from the perimeter of a tree. All told, samples were collected from 102 trees scattered fairly evenly from Rosedale in the east to West Vancouver in the west end of the Lower Fraser Valley, a distance of about 100 miles.

Diagnosis of potential pathogens was based on disease symptomology and fungus morphology. Rating of pathogenicity was based on the documented reputation of the the fungus and its relationship to healthy and diseased host tissues.

Results

Of the 102 trees sampled only 3 did not exhibit blight symptoms. All of the others exhibited blight with one or more associated fungi. In no case did bacterial infection

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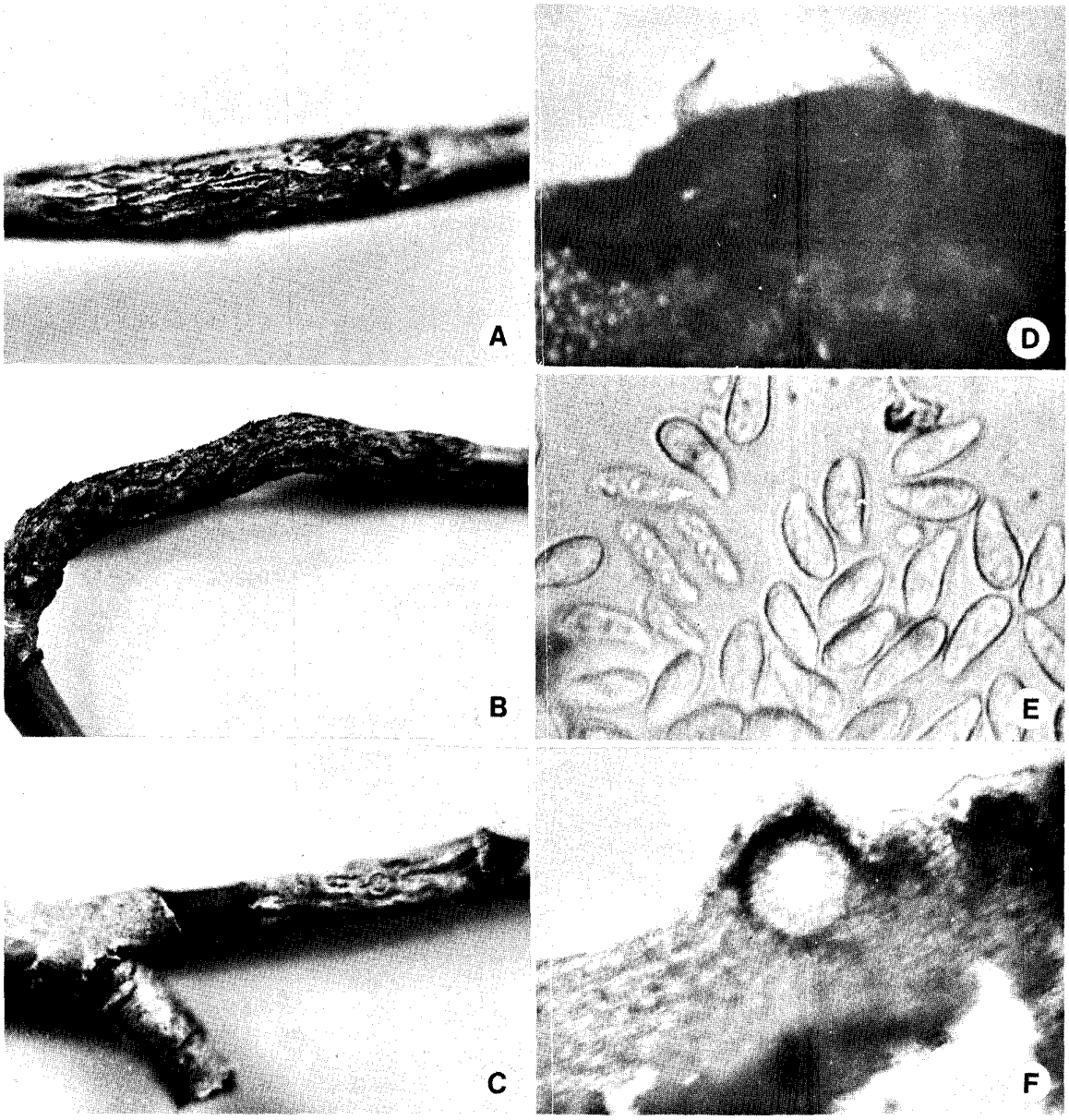

Figure 1. Blight of *Salix babylonica* L. A) A 1-year-old lesion in which pycnidia of *Pleurophomopsis salicicola* are barely visible. B) A 2-year-old canker containing both *Marssonina salicicola* and *P. salicicola*. C) A girdling canker. D) Acervulus of *M. salicicola*, X 80. E) Conidia of *M. salicicola*, X 700. F) Pycnidium of *P. salicicola*, X 100.

appear to be involved. Three distinct fungi were found associated with blight. These occurred either alone or in combination with one another as indicated in Table 1.

Marssonina sp.

The most abundant fungus found was a *Marssonina* sp. The pathogenicity of *Marssonina* spp. to *Salix* has been well documented and thoroughly discussed (19, 24, 25). Symptoms could be found on young leaves and shoots. Observations made during the survey suggested that only tender immature tissue was susceptible to infection. No new infections were found on mature leaves and shoots.

Symptoms of *Marssonina* infection on young leaves were round to irregular, brown, 0.2-5 mm diameter spots, mostly on larger veins on the lower leaf surfaces and petioles. The lesions tended to coalesce when infection was heavy. A white encrustation appeared at or near the center of the lesion as the conidia in acervuli

Table 1. Occurence of potentially pathogenic fungi from 102 *Salix babylonica* trees in the willow blight survey of south coastal British Columbia, 1973-74

Fungus	Number of trees infected
Marssonina salicicola (M.s.)	35
Pleurophomopsis salicicola (P.s.)	15
*Cytospora sp. (C.*sp.*)*	0
M.s. + P.s.	28
M.s. + C. sp.	11
P.s. + C. sp.	5
M.s. + P.s. + C. sp.	5
Nil	3

approached maturity. If infection was heavy, particularly on petioles, there was considerable defoliation. On shoots the early symptoms were similar to those found on leaves, but the infection followed one of two courses. Where the fungus grew rapidly, girdling of the young shoot took place, resulting in death of the twig beyond the lesion. Where the fungus grew slowly, the bark split longitudinally in the necrotic area forming a canker. In the necrotic area bordering the canker, acervuli appeared as dark brown to black, shiny, raised areas. They split when mature and released conidia. When growth of the fungus was retarded, and particularly where the lesions were coalescent, there was considerable swelling of the twig and it took on a roughened, somewhat, spindle-shaped appearance. On these more mature twigs, cankers appeared to be self-limiting, considerable healing took place, and little wood remained exposed.

The diagnostic criteria of the *Marssonina* agree well with those given by Rimpau (25) for *Marssonina salicicola* (Bres.) Magn. He also described *M. salicicola* as the only *Marssonina* species on *Salix babylonica*. The *Marssonina* species found in this survey has been confirmed as *Drepanopeziza sphaeroides* (Fr.) Nannf., st. conid. *Marssonina salicicola* (Bres.) Magn., by Dr. J. Bisset, Biosystematics Research Institute, Agriculture Canada, Ottawa; herbarium accession DAOM 160914.

Pleurophomopsis sp.

The second most abundant fungus found during the survey was a *Pleurophomopsis* species. Though lesions on leaves and shoots could be found early in the season, direct evidence of the fungus could not be found at this time. Unlike *M. salicicola*, the first fruiting bodies (pycnidia) were not found until late July.

Symptoms of *Pleurophomopsis* infection on twigs were much the same as those given for *M. salicicola*. At first the lesions on young shoots were minute black spots. As the infection progressed, the lesions increased in length up to about 20.0 mm. and were oval shaped or completely girdled the twig. As a lesion matured, the bark split and the edges became slightly raised. At this time pycnidia were readily seen in the necrotic tissue bordering the split and under the edges of the raised bark. On lesions resulting from the previous year's infection, almost completely exposed pycnidia were evident. During wet weather in spring and early summer, these pycnidia discharged masses of conidia, and as the season progressed a great many empty pycnidia were found.

The *Pleurophomopsis* has been identified by Dr. J. Bisset as *Pleurophomopsis salicicola* Petr., DAOM 160915.

Cytospora sp.

The third and least abundant fungus found associated with blighted trees was a *Cytospora* sp. Although the *Cytospora* sp. was found on blighted trees, its pathogenicity has been questioned. Peace (25) considers no species of *Cytospora* to be a parasite of *Salix in* Britain. He notes that when it is found on dead twigs, they have been killed or severely weakened by some other agent.

There is also much confusion regarding the taxonomy of the genus. Dennis (16) considers the genus to be in urgent need of revision, and suggests that separation of the species is extremely difficult. He is also of the opinion that many of the named species are in fact the same and have been named because of their appearance on a particular host. Hepting (19) is of much the same opinion, stating that there are in fact probably fewer species than are found in the literature.

Considering these opinions, it was decided that to spend time on the diagnostic details of the *Cytospora* sp. would not serve any useful purpose. It is sufficient to note that a *Cytospora* sp. was found on dead twigs of *S. babylonica*, but that it was not likely a major blight pathogen.

Discussion

The results of the survey indicate that *Marssonina salicicola* (Bres.) Magn. is the major pathogen in the weeping willow blight disease in coastal British Columbia. Previous reports of *M. kriegeriana* could very well be in error as there are no published records of the criteria used in the original identifications for this area. The identification of a *Marssonina* sp. as a major pathogen has implications for control, as methods developed on other hosts may also be effective on weeping willow (3).

The widespread occurrence of *Pleurophomopsis salicicola* Petr. associated with the disease is an interesting discovery warranting further investigation.

The fact that neither of the classical blight pathogens *Pollacia saliciperda* (All. & Tub.) Arx. and *Physalospora miyabeana* Fukushi were found in this survey suggests that *S. babylonica* L. is highly resistant to these pathogens. This agrees with Hepting (19) but not with Peace (24), Barr (1), or Conners (6). Unfortunately, the few published records rarely combine careful identifica-

tion of both pathogen and host so that the whole question of susceptibility remains in doubt. In south coastal British Columbia, *S. babylonica* is the only willow commonly grown in cultivation; a number of native *Salix* spp. do occur in the area, but they are of no economic importance and their disease status has not been studied.

It is apparent that the causal organisms of willow blight differ geographically and with host species. Identifications based solely on symptomatology and old host records should be treated with reservation.

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Screening systemic fungicides for potato wart disease[1]

Michael C. Hampson

Nine registered or experimental systemic fungicides were evaluated in pot and field tests for control of potato wart disease. Potato tubers were treated by dipping and inoculated by growing them in soil infested with European races 2 or 8 of *Synchytrium endobioticum*. There was a high degree of random infection and in some instances treatments reduced infections considerably but none scored zero infection consistently. It was concluded that the fungicides were of little value in controlling wart disease.

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On a évalué en pot et en plein champ neuf fongicides endothérapiques enregistrés ou expérimentaux dans la lutte contre *Synchytrium endobioticum*. Les tubercules ont été traités par trempage et inoculés par plantation en sol infecté par la tumeur verruqueuse de la pomme de terre. Les races européennes 2 et 8 ont servi d'inoculums. On a constaté un taux d'infection aléatoire élevé et, dans certains cas, les traitements ont considérablement réduit l'infection, mais aucun n'a réussi à l'éliminer complètement et de façon permanente. L'auteur conclut que les fongicides étudiés n'ont pratiquement aucune valeur dans la lutte contre la tumeur verruqueuse de la pomme de terre.

Potato wart disease, caused by *Synchytrium endobioticum* (Schilb.) Perc., is remarkably resistant to fungicides. More than 120 inorganic or organic chemicals, singly or in combination, have been assayed (1, 3, 5-9, 11-15) but the only successful treatments reported were either phytotoxic or acted as soil sterilants.

Early workers were quick to point out a weak link in the disease cycle: the migration of zoospores from resting sporangia to susceptible host tissue. Fungitoxic materials must permeate the soil-zoospore environment to be effective. Fungicides used to coat tuber surfaces could be rendered ineffective by leaching. Systemic fungicides offer the possibility of conferring 'immunity' on plants in wart-infested soil throughout the season.

Materials and methods

Cela W524 (20% triforine), FMC Corporation; NF44 (70% thiophanate-methyl), Ciba-Geigy Canada Ltd.; Bay Dam 18654 (50% cypendazole), Chemagro Ltd.; BAS 3460F (50% carbendazim), and BAS 3270F (50% cyclafuramid), BASF Canada Ltd.; Vitavax 75-W (75% carbathiin) and Uniroyal 1049 (37.5% Vitavax plus 37.5% captan, Uniroyal Chemical, Uniroyal Ltd.; Mertect Flowable (41.8% thiabendazole), Merck and Co., Inc.; and Benlate (50% benomyl), E. I. Dupont de Nemours and Co., Inc. were applied as dips to tubers of a susceptible cultivar. Tubers were dipped either at post harvest and at post sprouting, or after sprouting only. Concentrations as specified by the manufacturer and greater concentrations were tried.

For pot work, wart-free tubers of the cultivars Arran Victory and Pink Pearl were sprouted at 27°C and 80% RH. The lateral sprouts were removed and the tubers dipped for 15 min in test solutions. The dried tubers were potted in 15.3-cm (6-inch) pots with perlite:peat moss (1:2 v/v) and covered with potting mix containing sporangia or rotted wart compost. The inoculated tubers were grown in a controlled environment room (21°C 80% RH, 12 000 lux, 14-h day), watered daily, and fertilized weekly (4) for 4 wk. After a further 4 wk on a greenhouse bench, the plants were harvested. Symptom expression was measured as the weight of the tumor excrescences divided by the weight of the green plant tops per treatment (WTI), and as the percentage of tumor-bearing plants per treatment (PIV). Five tubers comprised an experimental unit for all treatments, except in one experiment where ten tubers were used.

For field work a field was selected known to produce large masses of tumorous galls on Arran Victory. BASF compounds were excluded from this test due to short supply. Four levels of active ingredient (a.i.) were prepared: 250, 1000, 2000 and 4000 ppm. Fifteen Arran Victory tubers were dipped for 15 min in each of the chemical concentrations and planted in duplicate rows, with three control rows in each plot.

Results

Pot experiments

In exploratory work with cv. Arran Victory and race 2 of *S. endobioticum* Benlate at 100, 250, and 500 ppm a.i. resulted in PIV 25, 25, and 0, respectively. Control infection was PIV 14. In the pot experiments proper (Table 1A), all treatments except those with BAS 3270F yielded a PIV 100 value. Wide differences in WTI, i.e. amounts of infection/plant, were experienced. Confirming experiments yielded quite different results, often PIV 0. Control plants yielded wide differences in PIV. Results using Pink Pearl infected with race 8 (Table 1B) were

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Table 1. Wart tumour indices (WTI) and percent infection values (PIV) of Arran Victory and Pink Pearl cultivars 8 weeks after planting tubers treated with different concentrations of systemic fungicides in pots of soil mix infested to different levels with two races of *Synchytrium endobioticum*

Fungicide and rate (ppm active)		No. of treatments*	Sporangia: Age (days)	Sporangia: No./g soil	WTI**	PIV†
A. Arran Victory, S. endobioticum race 2						
Benlate	30	1	60	10^3	1.2	100
	30	2	60	10^3	0.4	100
	30	1	21	10^3	0.9	10
	30	2	21	10^3	0.0	0
	5,000	1	310	10^3	0.0	0
	10,000	1	240	3×10^3	<0.1	20
Mertect	3.15	1	60	10^3	0.1	100
	3.15	2	60	10^3	1.6	100
	3.15	1	21	10^3	0.7	25
	3.15	2	21	10^3	0.0	0
	4,200	1	310	10^3	<0.1	20
	8,400	1	240	3×10^3	<0.0	25
Vitavax	1.2	1	60	10^3	0.8	100
	1.2	2	60	10^3	0.7	100
	1.2	1	21	10^3	1.7	20
	1.2	2	21	10^3	0.0	0
	7,500	1	310	10^3	0.0	0
	15,000	1	240	3×10^3	0.2	75
Uniroyal 1049	0.63	1	60	10^3	0.4	100
	0.63	2	60	10^3	0.6	100
	0.63	1	21	10^3	<0.1	10
	0.63	2	21	10^3	0.0	0
	3,750	1	310	10^3	0.0	0
	7,500	1	240	3×10^3	<0.1	20
BAS 3460F	5	1	60	10^3	<0.1	100
	5	2	60	10^3	<0.1	100
	5	1	21	10^3	0.0	0
	5	2	21	10^3	0.0	0
	5,000	1	310	10^3	0.0	0
	10,000	1	240	3×10^3	0.1	20
BAS 3270F	5	1	60	10^3	0.6	100
	5	2	60	10^3	0.0	0
	5	1	21	10^3	0.2	10
	5	2	21	10^3	0.0	0
	5,000	1	310	10^3	0.7	67
	10,000	1	240	3×10^3	0.0	0
Cela W524						
	10,000	1	310	10^3	0.6	40
	20,000	1	240	3×10^3	0.2	50
NF 44	7,000	1	310	10^3	0.1	25
	14,000	1	240	3×10^3	<0.1	20
Bay Dam 18654	5,000	1	310	10^3	0.0	0
	10,000	1	240	3×10^3	0.0	0
Untreated Controls			60	10^3	1.0	100
			21	10^3	0.1	4
			310	10^3	0.0	0
			240	3×10^3	<0.1	11
B. Pink Pearl, S. endobioticum race 8						
Benlate	5,000	1	240	2×10^3	1.3	100
	10,000	1	200	6×10^4	<0.1	20
Mertect	4,200	1	240	2×10^3	1.3	100
	8,400	1	200	6×10^4	<0.1	25
Vitavax	7,500	1	240	2×10^3	0.9	100
	15,000	1	200	6×10^4	0.0	0

Table 1. (Cont.)

Fungicide and rate (ppm active)		No. of treatments*	Sporangia Age (days)	Sporangia No./g soil	WTI**	PIV†
B. Pink Pearl, S. endobioticum race 8 (cont.)						
Uniroyal 1049	3,750	1	240	2×10^3	<0.1	100
	7,500	1	200	6×10^3	0.0	0
BAS 3460F	5,000	1	240	2×10^3	0.5	100
	10,000	1	200	6×10^4	<0.1	20
BAS 3270F	5,000	1	240	2×10^3	0.0	0
	10,000	1	200	6×10^4	0.0	0
Cela W524						
	10,000	1	240	2×10^3	0.7	100
	20,000	1	200	6×10^4	0.0	0
NF 44	7,000	1	240	2×10^3	0.7	100
	14,000	1	200	6×10^4	0.0	0
Bay Dam 18654	5,000	1	240	2×10^3	1.1	100
	10,000	1	200	6×10^4	0.0	0
Untreated Controls			240	2×10^3	1.0	100
			200	6×10^4	0.0	0

*Based on 5 or 10 tubers per treatment; untreated controls 5-60 tubers/experiment. Single treatments were applied following tuber germination; two treatments were applied following tuber harvest and following tuber germination.

**WTI - wart tumor index.

†PIV - % infection value.

Table 2. Percent infection values (PIV) of Arran Victory plants treated with systemic fungicides at different concentrations and grown in race 2-infested soil in the field for 12 weeks

Fungicide*	PIV for the following fungicide concentrations (ppm a. i.): 250	1000	2000	4000
Benlate	80	20	27	20
Mertect	100	50	27	20
Vitavax	73	53	20	53
Uniroyal-1049	53	40	40	13
Cela W524	73	53	20	13
NF44	93	40	47	7
Bay Dam-18654	93	40	20	47
Untreated check†	42	0	0	0

*Fifteen tubers/treatments.
Forty-five tubers at each fungicide level.

somewhat at variance with those using Arran Victory and race 2. Control plants were all infected in a trial using low a.i. levels, but none was infected in a later trial at higher a.i. levels. Low PIV values (0-25) were recorded for plants treated at the higher a.i. levels.

Field experiment

Higher levels of infection were found in treated than in untreated (check) rows (Table 2). As fungicide concentration rose, there was a general decrease in PIV. When pot and field tests were compared, it was found that in most cases PIV's were higher for treated than for untreated tubers. Although Bay Dam 18654 was the most effective treatment in pot tests (Table 1), it was among the least effective in the field tests. The only consistent result was with Vitavax, which was least effective in both cases.

Discussion

This work was undertaken with the knowledge (2) that systemic fungicides are least effective against Phycomycetes, but was carried out in the hope that a compound of value in respect of potato wart disease might come to light.

It is recognized that a difficulty with chemical control of potato wart disease is in getting the chemical to the infection court. Susceptible tissue - sprouts, stolon buds, eyes - is produced over a long time base. Through the use of systemic fungicides it was hoped that resistance to *S. endobioticum* may have been developed in infection courts.

In the present study, the initial dipping was expected to protect only the sprout infection courts. The pot experiments were terminated after 2 mo growth. Tumorous galls developed at stem bases, which leads us to believe that in these instances infections occurred in sprout tissue. No attempt was made to treat plants later in the

season since it became clear from the pot work that little success was being achieved in effecting control at the infection courts. Some control was developed as a.i. concentrations increased (Table 2). Infections still occurred, however, in pots at 20 000 ppm a.i. and in the field at 4000 ppm a.i., or 8960 kg/ha.

In potato wart disease fungicide control work, it is felt to be imperative that a fungicide consistently yield PIV 0, otherwise sporangia with a longevity exceeding 30 yr (5) will continue to be generated. None of the compounds gave this result consistently. It was concluded that under our experimental conditions the systemic fungicides assayed were not effective in controlling potato wart disease.

Attention is drawn to the observation of wide differences in symptom expression that do not appear to be related to the fungicides or the a.i. levels. Hunt et al. (6) first recorded the capricious nature of *S. endobioticum* in their work in Pennsylvania; wart disease appeared in check pots or 30 infested garden soils, but plants developed infection in only 13 of those plots; on another occasion a pot test of 70 soils showed no wart, while plants in 25 of the plots manifested the disease. Roach et al. (11) found that field testing was more conclusive than was pot work, since no infection occurred in check pots. In the work reported here, 60-day-old inoculum was associated with PIV 100, and 21-day-old inoculum with PIV 0. However in other work we have found young inoculum more infective than older inoculum. Increasing inoculum density did not necessarily increase the chances of succesful infection. Likewise, more tumorous gall tissue developed with PIV 10 than with PIV 100.

The peculiar distribution of levels of infection which developed in these trials merits explanation. Several hypotheses can be advanced for future experimental work. The function of sporangial age is ambivalent since conflicting results arose from using different ages of sporangia in this and other work. Likewise, inoculum densities do not appear to relate directly to infection levels. Very possibly the dormancy characteristic of the sporangium plays a role in infectivity, germinability of sporangia perhaps varying at different phases of the host plant, or at times of the year. The low levels of infection in check plants in the field experiments has recently been paralleled in an experiment involving Sinox, copper sulphate, and Vorlex, inter alia (P. Thompson, personal communication, 1977). An interesting hypothesis that should be followed up is that under natural field conditions in Newfoundland, a soil-inhabiting antagonist limits the activity of *S. endobioticum*, and that the fungicidal materials removed this natural control at the infection courts.

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Author index to volume 57

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