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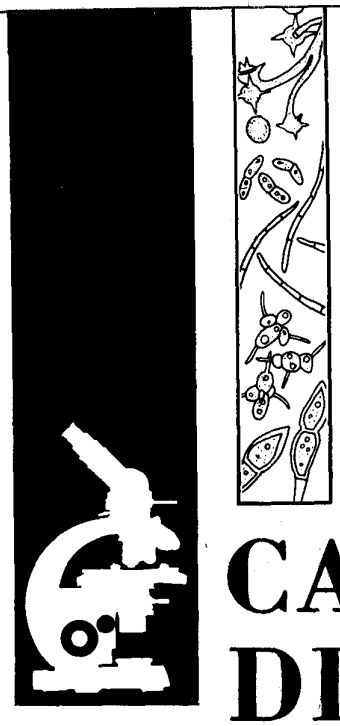

CANADIAN PLANT DISEASE SURVEY

EDITOR W.L. SEAMAN

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

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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. It will also accept other original information such as the development of methods of investigation and control, including the evaluation of new materials. Review papers and compilations of practical value to phytopathologists will be included from time to time."

DETECTION OF TOMATO RINGSPOT VIRUS IN PELARGONIUM IN ONTARIO[1]

W.G. Kemp

Abstract

A virus transmitted by mechanical inoculation from Pelargonium with ringspot symptoms to cucumber was identified as tomato ringspot virus. Its identity was established primarily by serological comparisons with a known culture of tomato ringspot virus and secondarily by simultaneous-infection tests, symptomatology, host reaction, and properties in vitro. It was transferred by means of Xiphinema americanum from infected geranium roots to cucumber bait plants.

Introduction

In March 1967, a sap-transmissible virus was isolated from geranium (Pelargonium hortorum Bailey cv. Appleblossom) that was distinct from any virus previously described from Pelargonium in Canada. It was recovered from one stock plant that succumbed to bacterial stem rot later in the summer. Besides symptoms suggestive of this bacterial disease, a few large, chlorotic ringspots were observed on the lower, older leaves but not on the youngest ones. The virus failed to react with antiserum against tobacco ringspot virus previously isolated from geranium by the author (4). Mechanical transmission of the virus from geranium leaves to other herbaceous plants was erratic and occurred on only two occasions, both in March, when cucumber seedlings developed symptoms 10 days after inoculation. Results of studies on symptomatology, transmission, host range, and serological identity of the virus are presented in this paper.

Materials and methods

Stock cultures of the unknown geranium virus under investigation, as well as an isolate of tobacco ringspot virus and a tomato ringspot virus culture provided by Dr. R. Stace-Smith, Research Station, Canada Department of Agriculture, Vancouver, B.C., were maintained in cucumber for use in comparative tests.

Infected cucumber leaf ground in 0.25% Na_2SO_3 or 2.5% nicotine solutions was used as inoculum in various tests. Five-hundred mesh carborundum was added to the inoculum.

Virus-indexed geranium seedlings (cv. Nittany Lion) at the 4 leaf stage were preconditioned for 48 hr in complete darkness before mechanical inoculation with the virus and were then reinoculated 10 days later after a second 24-hr dark exposure.

The thermal inactivation point, dilution end point, and longevity of the virus in crude sap were determined by conventional methods.

Nematode transmission tests were done by the sequential planting of virus-infected geranium and cucumber bait plants in nematode-infested soil. Three soil samples collected from a cherry orchard at Fonthill, Ontario, with varying populations of Xiphinema americanum Cobb, 1913 were used. Control tests with cucumber indicated that the nematodes were initially virus-free.

An antiserum prepared by the author (4) against tobacco ringspot virus from geranium and another against tomato ringspot virus supplied by Dr. R. Stace-Smith were used in the identification of the virus in question. Agar-diffusion precipitin tests were performed in plastic plates prepared as described previously (3).

Results

TRANSMISSION TO PELARGONIUM

Of 22 inoculated 'Nittany Lion' seedlings, 21 showed visible symptoms within 3 to 4 weeks. The virus was recovered from each plant within 3 months. Later, when the virus was identified serologically, these isolates were checked and found to be identical to the one used in the original inoculation. Eight control plants consisting of seedlings of the same age were planted and prepared for inoculation in the same way but were not actually inoculated. None developed symptoms.

Most of the infected seedlings were stunted; leaves were small and many of them showed yellow veins and chlorotic spots and rings (Figure 1). Leaves on one plant showed whitish rings 1/8- to 1/4 inch in diameter. Two months after inoculation the newest leaves were symptomless, but the plants remained somewhat smaller than comparable healthy controls. No stem lesions were noted.

[1] Contribution No. 176, Research Station, Canada Department of Agriculture, Vineland Station, Ontario

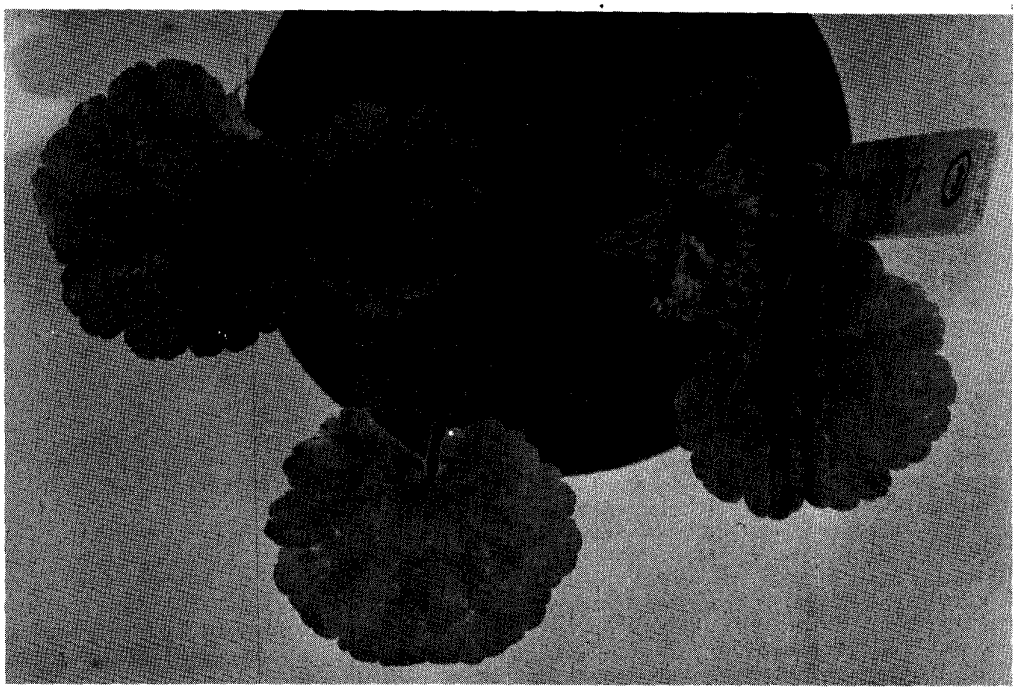

Figure 1. *Pelargonium hortorum* ('Nittany Lion') seedling showing systemic symptoms 5 weeks after mechanical inoculation with a geranium isolate of tomato ringspot virus.

HERBACEOUS HOST REACTIONS

The host range study was restricted to species considered to be of value in identifying viruses of the ringspot type. Symptoms on the various hosts are summarized in Table 1.

Only bean (Phaseolus vulgaris) failed to react to the virus; each of the other plants developed chlorotic or necrotic lesions on the inoculated leaves. Systemic symptoms appeared as chlorotic or necrotic spots or rings, veinal chlorosis, and/or mild mottle. Apical stem necrosis occurred on Chenopodium amaranticolor, C. quinoa, and Vigna sinensis. The reactions of these herbaceous plants suggested that the geranium virus possibly was related to tomato ringspot virus. Subsequently, the virus and an isolate of tomato ringspot virus were inoculated into the same range of test plants under the same conditions. With the exception of certain variations in tobacco and cucumber, the responses of the test plants to the two isolates were similar.

BIOLOGICAL AND PHYSICO-CHEMICAL PROPERTIES

Interference - The geranium virus and the tomato ringspot virus acted as closely related viruses in the presence of each other in two simultaneous-infection tests. No double-virus effect, indicating unrelatedness, was observed (2). Single and double infections with these viruses did not induce any marked differences; all plants were stunted to approximately the same degree and showed no symptoms other than those caused by either of the viruses alone.

Serology - In agar diffusion tests, crude sap from infected cucumber leaves containing the geranium virus reacted specifically with antiserum to tomato ringspot virus but not with antiserum to tobacco ringspot virus. Sap from healthy cucumber failed to react with either antiserum. Furthermore, the precipitin pattern produced when both of these virus isolates were compared in adjacent wells against tomato ringspot virus antiserum was a single, coalescing zone. The fusion of precipitation zones (Figure 2) suggests a close serological relationship between these viruses (1).

Physical Properties - Virus-containing sap maintained at room temperature (ca. 20C) was infective after 24 hr but not after 48 hr. The dilution end point was between 10^{-4} and 10^{-5}, and the virus was thermally inactivated after 10 minutes at 55C. These properties were similar to those of the tomato ringspot virus isolate when both were tested in a comparative study.

Table 1. Symptoms induced in test plants by a ringspot virus from Pelargonium

Host plant	Inoculated leaves	Uninoculated leaves and stems
Chenopodium amaranticolor Coste & Reyn.	chlorotic lesions	chlorotic spots, mottle, leaf distortion, stem necrosis
Chenopodium quinoa Willd.	chlorotic lesions	veinal chlorosis, stem necrosis
Cucumis sativus L.	chlorotic lesions	veinal chlorosis, mottle
Gomphrena globosa L.	buff-centered lesions	mottle, irregular buff-centered lesions
Lycopersicon esculentum Mill.	necrotic lesions	veinal necrosis
Nicotiana glutinosa L.	chlorotic, necrotic spots	chlorotic mottle
Nicotiana tabacum L.	necrotic spots, broken concentric rings	few broken buff rings
Petunia hybrida Vilm.	necrotic spots	veinal chlorosis, mild mottle
Phaseolus vulgaris L.	none	none
Vigna sinensis Endl.	red spots, rings	reddish-brown veins, stem necrosis

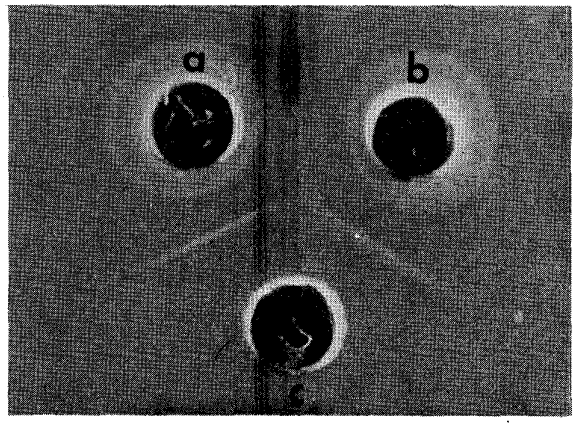

Figure 2. Photograph of precipitation pattern in agar. The meeting of the two precipitin lines indicates that the geranium ringspot virus (a) and the tomato ringspot virus (b) are identical or closely related; well (c) contained antiserum of tomato ringspot virus.

NEMATODE TRANSMISSION

Two of three populations of Xiphinema americanum transmitted the geranium virus. Populations containing 64 and 42 virus-free nematodes per 100 gm of soil acquired the virus from infected geranium roots within 3 weeks and transmitted it to cucumber bait plants within 1 month of their planting in these soils. No transmission to cucumber occurred in soil originally planted with infected geraniums and containing 8 nematodes per 100 gm of soil.

Discussion

The results substantiate an earlier report (5) of the isolation of a probable strain of tomato ringspot virus from geranium. The identification of tomato ringspot virus in geranium in Ontario has been made primarily on a serological comparison with an isolate of tomato ringspot virus. Cursory serological tests indicated that the isolates tested here are

closely related, but the reactions induced by them in tobacco and cucumber under the same conditions suggest that they may be distinct strains. Tomato ringspot virus is the second NEPO-virus detected in geraniums in Ontario; the first, tobacco ringspot virus, was found by the author in the cultivar Mme. Salleron in 1964 (4). Obviously field soils in Ontario contain nematodes that are vectors of this virus. At present, the prevalence and economic importance of tomato ringspot virus in geraniums under greenhouse conditions is uncertain.

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OCCURRENCE OF BACTERIAL CANKER OF SWEET CHERRY AND PLUM IN ONTARIO[1]

B.N. Dhanvantari

Abstract

Bacterial canker caused by Pseudomonas spp. is reported on sweet cherry and plum in important stone fruit growing areas of Ontario. Symptoms of this disease are similar to those described for bacterial canker on the west coast of the United States and in England. This is the first report of bacterial canker on sweet cherry and plum in Ontario.

Introduction

Bacterial canker is a major disease of sweet cherry and plum in England, New South Wales, California, and Oregon. The history, symptomatology, host range, geographical distribution, etiology and economic importance of this disease have been reviewed recently by Cameron (2). Since then, its known geographical distribution has been extended to Ireland and many parts of Europe (1), southeastern United States (6), Missouri (5), and Nova Scotia, Canada (4).

In England the causal bacteria have been grouped under Pseudomonas syringae van Hall and P. mors-prunorum Wormald, the former being ubiquitous with a wide host range, and the latter being regarded as more specialized; recent work indicates that the pathogens may be considered different ecotypes, if not separate species (3). Bacterial canker of stone fruits in the United States has been attributed to P. syringae, and the only reported occurrence of P. mors-prunorum in North America has been in Nova Scotia, Canada (4).

During the spring and summer of 1968, our attention was called to an unusual dying of terminal twigs and branches accompanied by profusely gumming cankers in the sweet cherry (Prunus avium L.) orchards of Kent Country. Although many bacterial diseases appeared to have been encouraged by unusually heavy rain during the summer of 1968, these symptoms were also present in the preceding years and pathogenic pseudomonads had been isolated from them in 1966 and 1967. There had been cause for confusion, pending extensive isolations and identification of the causal agent, inasmuch as sweet cherry and plum are within the host range of the Cytospora spp. that cause peach canker in Ontario, and because bacterial canker of sweet cherry had not previously been reported to be caused by Pseudomonas sp. in Ontario

Disease survey, symptoms, and isolations

A survey was undertaken to determine the extent of occurrence of canker on sweet cherry, plum and other stone fruits in Essex, Kent, and Lambton Counties of southwestern Ontario and in Lincoln County of the Niagara Peninsula where most of Ontario's stone fruits are grown. Orchards were inspected at Niagara-on-the-Lake, St. Davids, Virgil and Vineland (Lincoln Co.); Cedar Springs and Blenheim (Kent Co.); Ruthven and Harrow (Essex Co.); and Reece's Corners, Forest, and Arkona (Lambton Co.) (Fig. 1).

On sweet cherry, cankers occurred on the trunk and scaffold branches, in the crotches, and at spurs (Fig. 2). In many instances the cankers could be traced to infected spurs. The cankers exuded profuse amber-coloured gum. The cankered tissue was sunken (Fig. 3) and, where it tended to girdle, the parts above were dead or dying (Fig. 4). As a result, the buds failed to develop, or else, after shoot development, the leaves wilted or turned brown and hung down. The latter symptoms, seen in early summer, have been referred to as "shoot withering". Leaf spots were common on affected trees (Fig. 5). The younger spots were purplish pin-points surrounded by a halo of chlorosis, later turning reddish brown and becoming somewhat irregular in outline, 1-3 mm in diameter. Often the leaf spots dehisced, leaving a shot hole. The leaf-spot symptoms were not unlike those caused by Xanthomonas pruni (E. F. Sm.) Dows. but, unlike the latter, did not cause ready defoliation. In a few instances leaf spots due to Pseudomonas and X. pruni occurred on the same leaf. The disease was generally found on older trees, but in Kent and Essex Counties relatively young trees and new plantings were also affected. Sweet cherry varieties affected included 'Bing', 'Early Lyons', 'Schmidt', 'Seneca', 'Vista', and 'Venus'. On plums the symptoms were similar but there was very little exudation of gum from the cankers, which had a tendency to extend more along the longitudinal axis. Plum varieties affected included 'Stanley', 'Damson', 'Italian', and 'Peach'. The leaf spot phase was severe in Lambton Co., and moderate to severe in Essex and Kent Counties and in the Niagara Peninsula. Tree mortality that might

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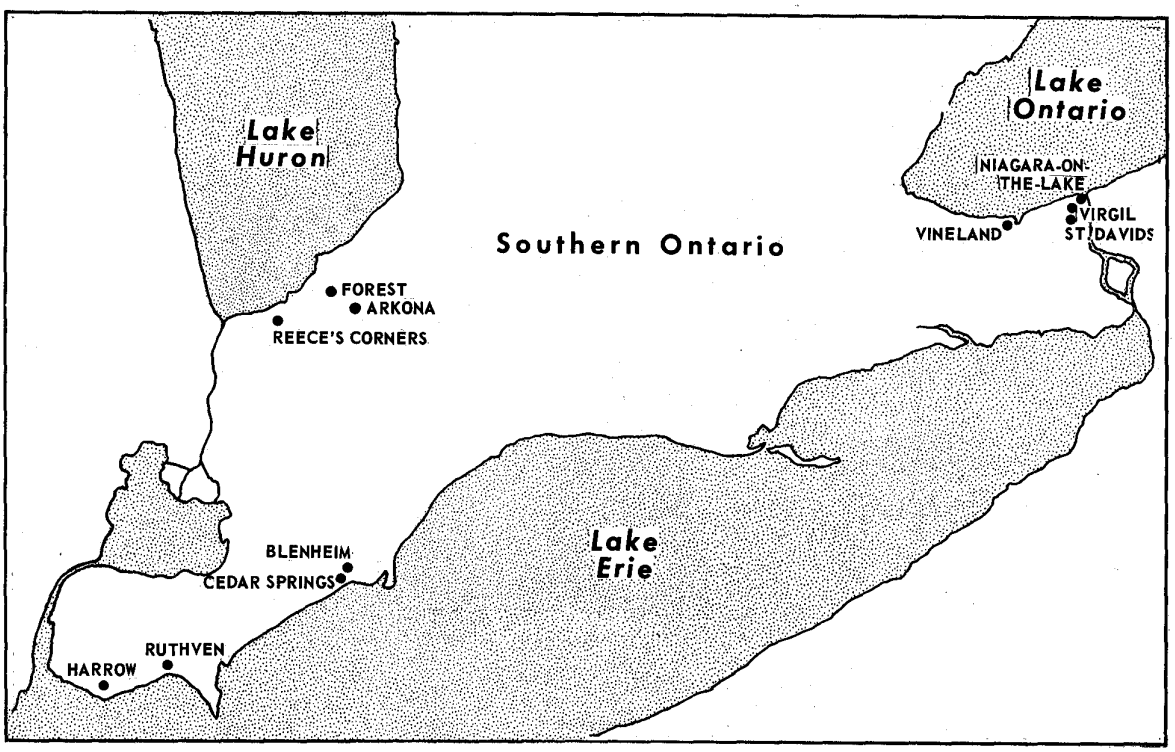
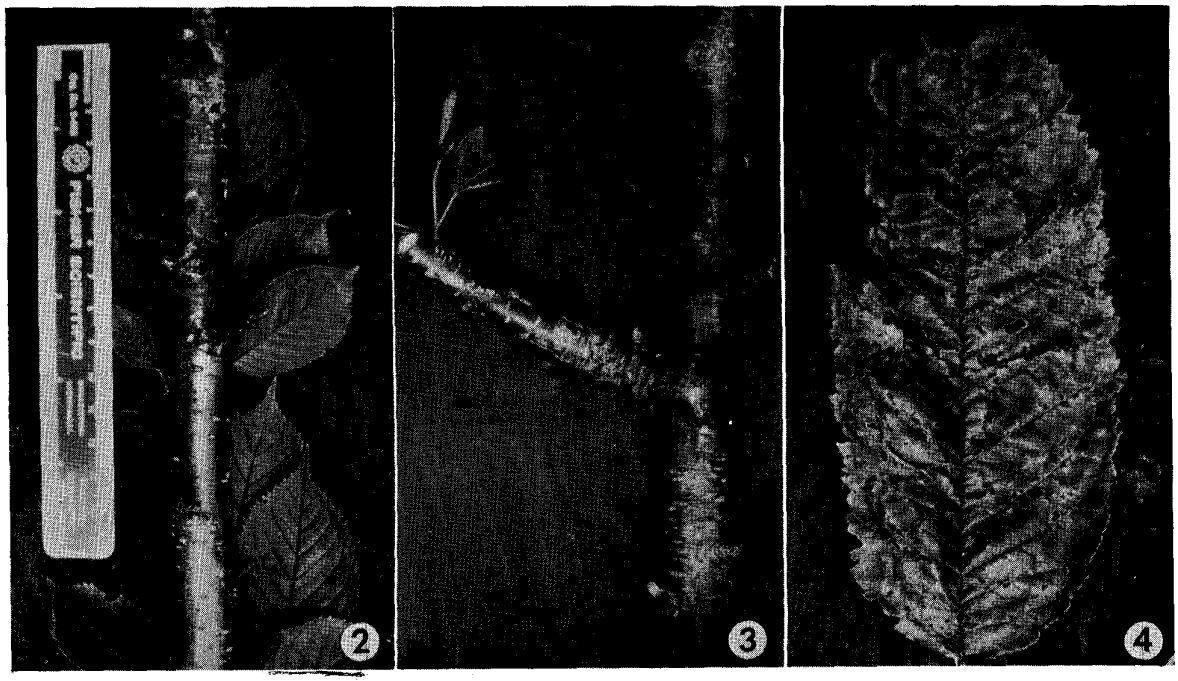


Figure 1. Locations in southern Ontario where bacterial canker of sweet cherry and plum was observed during the spring and summer of 1968.



Figures 2–4. Symptoms of bacterial canker on sweet cherry: 1) An infected spur, 2) Gumming and sunken canker on twigs, 3) Leaf spots and shothole effect.

be attributed to this disease was evident in a few orchards in the Niagara Peninsula and Lambton Co. It has been reported that leaf spotting is particularly severe in England, where shoot and spur withering is also common, but that such leaf spots are rare in California and Oregon (2). In this respect, the symptomatology of the bacterial canker of sweet cherry and plum in Ontario is more like that in England than that on the west coast of the United States.

Isolations carried out extensively from diseased tissue of samples from all the orchards inspected yield pathogenic pseudomonads, and on that basis and on the over-all symptomatology, we call the disease of sweet cherry and plum described here "bacterial canker". Cameron (2) advocates the use of this name in preference to several others, i.e. gummosis, blast of stone fruits, sour-sap, cherry gummosis, and bacteriosis, as recent usage in many parts of the world seems to favor it and also as it is of advantage to use a uniform terminology. In addition to Cytospora spp., which are most frequently isolated from peach cankers, and which are undoubtedly the usual cause of peach canker in Ontario, pathogenic pseudomonads have been isolated occasionally from peach in the last two years from relatively small cankers not unlike those caused by Xanthomonas pruni. The characteristics of the bacteria isolated, and their conformity or lack of it with the published descriptions of P. syringae and P. mors-prunorum will be published elsewhere.

These investigations have added another important stone fruit growing area to the known geographical range of bacterial canker in North America. It is not claimed that it is a new disease in Ontario, because it has probably been present for some time but not reported. The facts that many of the affected trees were usually older and that the growers appear to have been familiar with the symptoms described on sweet cherry and plum support this contention. The implications of these investigations are that the sweet cherry industry in Ontario has now to contend with yet another disease which is difficult to control. The peach industry will also have to take into account the realities of the presence in their close neighbourhood of a pathogen that has been a factor in causing widespread injury and mortality in the peach orchards of California and southeastern United States.

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FIELD RESISTANCE TO PYRENOPHORA BROMI IN BROMUS INERMIS AND BROMUS SPP.[1]

J. Drew Smith[2]

Abstract

A severe epidemic of the leaf spot disease of bromegrass caused by Pyrenophora bromi developed on spaced plants at the Big River field test area in August 1968. The progress of the disease on 20 replicated and unreplicated test entries of Bromus species and strains was followed from July to September. Disease ratings on three dates during the epidemic showed a high positive correlation. Infection was uniform on the test blocks at the height of the epidemic.

Synthetics bred from clones selected from the northern/southern hybrid S-7269 of smooth bromegrass, Bromus inermis, showed higher disease resistance than the resistant standard 'Lincoln'. Synthetics between the clones of S-7269 and field-resistant clones of B. tyttholepis S-1172 also showed high resistance to the pathogen. Parent clones of the synthetics and hybrids were similar to their progeny in reaction to the pathogen. Selections of B. biebersteinii, B. ornans, B. carinatus, and other introduced Bromus spp. also appeared promising as sources of resistance. Northern-type B. inermis cultivars were either very susceptible or not noteworthy for their resistance. Leaf spots on resistant strains of B. inermis often were sharply necrotic; susceptible strains of the same species senesced more rapidly than resistant lines.

Introduction

The leaf spot caused by Pyrenophora bromi (Died.) Drechs. syn. Pleospora bromi Died. [stat. conid. Drechslera bromi (Died.) Shoem., syn. Helminthosporium bromi (Died.) Died.] is the most important foliar disease of smooth bromegrass, Bromus inermis Leyss., in seed and hay crops on the Black and Gray Soils in Saskatchewan, Alberta, and the Peace River Region of British Columbia (11, 12, 13). Northern-type cultivars of smooth bromegrass that are currently grown in these regions have been found less resistant to the pathogen than southern types (8, 13, 14). Northern/southern hybrids showed intermediate resistance (13). Resistance to P. bromi superior to that in B. inermis has been found in weed bromes (1, 2, 6), in other members of the Bromopsis (Zerna) Section of Bromus to which B. inermis belongs (1, 2, 4, 6), and in hybrids between members of the latter Section (7).

In a test at Big River, Saskatchewan, in 1967 (13), seven clones of S-7269, a northern/southern synthetic of B. inermis, were not infected with P. bromi whereas interplanted 'Carlton 1961' and S-6733 Syn 2 were diseased. Six clones of B. tyttholepis Nevski (10) were also free from infection. These uninfected clones appeared promising as sources of resistance.

This paper reports the field reactions to P. bromi of (a) the resistant clones of B. inermis and B. tyttholepis from the 1967 test, (b) synthetics made from these clones, (c) hybrids between the B. inermis and the B. tyttholepis clones, (d) other Bromus spp. in the Bromopsis Section.

Materials and methods

The origins and characters of the plant material are given in Table 1.

Seeds were germinated in moist chambers and seedlings pricked out into a potting mixture in 5-cm peat pots. Clonal material was divided and grown in similar pots. Subsequently, plants in the pots were set out in the field on 15-17 May when seeded and clonal material had attained about the same stage of growth.

The test area was in a cultivated plot within a hay field of common bromegrass on Gray Woodland Soil at Big River, Saskatchewan. This hayfield bromegrass was an infection reservoir, since crops in previous years were severely infected with P. bromi. Infector rows of very susceptible Bromus sp. OT1927/9833 (13) were planted 5.5 m apart, with 1.5 m between plants. There were five rows of these which divided the test into four blocks, and the plants in them were intended to act as secondary infection foci from primary ones in the hay field. Test entries, in groups of 10 plants at 0.5 m spacing, were planted in the 5.5 m wide blocks at right angles to the five infector rows of susceptible Bromus sp. There were 25 cross rows of test entries,

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[2] Plant Pathologist.

Table 1. Origins and descriptions of test entries

Species	Text - fig. ref. no.	Cultivar, strain or accession no.	Origin	Description
B. inermis Leyss	1	Carlton 1961	Saskatoon	Northern type, susceptible standard cultivar.
	2	Lincoln	Neb., U. S. A.	Southern type, resistant standard cultivar.
	3	OT1561/400-404 and /493	U. S. S. R.	Northern type Russian cultivars.
	4	OT1927/9835	Bulgaria	B. inermis var. aristata. Northern type.
	5	Clones B36-42	Saskatoon	Resistant clones from 1967 test (loc. cit.). From northern/southern hybrid S-7269.
	6	S-7304	Saskatoon	Synthetic of clones B36-42.
	7	S-7306	Saskatoon	Duplicate of S-7304.
B. tyttholepis** Nevski	8	Clones B30-35 from S-1172	Col., U. S. A.	Resistant clones from 1967 test. Selected from S-1172, a synthetic made at Saskatoon from B. erectus received in 1944. **
	9	OT1927/9493	U. S. S. R.	Glabrous, leafy, extreme bunch type. From Central Siberia.
	10	Korean	Uncertain	Leafy, vigorous selection by E. Buglass, Res. Branch, Can. Agr. Exptl. Farm, Indian Head, Sask.
B. inermis × tyttholepis	11	S-7305 BI	Saskatoon	Seed from B. inermis in synthetic of clones B36-42 and B30-35.
	12	S-7305 BT	Saskatoon	Seed from B. tyttholepis in synthetic of clones B36-42 and B30-35.
B. biebersteinii Roem. & Schult.	13	Regar	Idaho, U. S. A.	A high yielding, disease resistant cultivar, released in the U. S. A. in 1966 (5).
	14	Clones B55-61 from OT1927/8572	Hungary	Leafy, winter hardy and disease resistant at Saskatoon.

* Accession no., Forage Crops Section, Research Station, Canada Department of Agriculture, Ottawa.

** According to Nath & Nielsen (10) most of the B. erectus introduced into N. America is B. tyttholepis Nevski.

Table 1 (Continued)

Species	Text - fig. ref. no.	Cultivar, strain or accession no.	Origin	Description
B. ornans Kom.	15	Polycross	Uncertain	From Res. Branch, Can. Agr. Res. Sta., Lethbridge, Alberta.
	16	A52	Uncertain	From Res. Branch, Can. Agr. Res. Sta., Lethbridge, Alberta.
	17	A54	Uncertain	From Res. Branch, Can. Agr. Res. Sta., Lethbridge, Alberta.
B. angrenicus Drob.	18	Clones B23-29 from OT1927/8569	Hungary	Leafy, winter hardy but very disease susceptible at Saskatoon. Morphologically close to B. inermis.
B. carinatus L.	19	Kew collection	Kew, England	Naturalized, from Pacific N. W. of N. America.
Bromus sp.	20	OT1927/9833	Grenoble, France	Taxonomic position uncertain, very susceptible to P. bromi in 1967 test. A small, glaucous, hairy perennial.

each row containing 40 plants. Susceptible and resistant standard cultivars of B. inermis, 'Carlton 61' and 'Lincoln', respectively, were planted across the four blocks of the test alternately as cross rows 4, 8, 12, 16, 20, and 24. Since some of the seed of new introductions germinated poorly and the amount of clonal material was limited, full replication was not possible for all test entries.

Severity of Pyrenophora leaf spot was rated on a 0 to 4 scale, where 0 was no disease and 4, very severe disease. Selenophoma leaf spot, caused by S. bromigena (Sacc.) Sprague & Johnson, was absent and scald, caused by Rhynchosporium secalis (Oud.) J. J. Davis, was present in insignificant amounts on the rating dates of 24 July, 14 August, and 10 September, 1968.

Results and discussion

Pyrenophora leaf spot was noted on common brome in the surrounding hayfield when the test was planted on 17 May 1968. The disease progressed slowly under the dry conditions prevailing in May and June, then rapidly during wet weather in late July, August, and September.

Replicated test entries with the exception of B. ornans 'Polycross' had significantly lower ratings for the disease at all recording dates than the susceptible standard 'Carlton' (Table 1, Fig. 1). On 10 September, when ratings were highest, all replicated test entries had significantly less disease than the resistant standard 'Lincoln'. There were no significant differences in ratings between the duplicate B. inermis synthetics S-7304 and S-7306 on the three dates. In the B. inermis × tyttholepis synthetics, S-7305 BT (B. tyttholepis ♀) showed significantly less severe spotting than S-7305 BI (B. inermis ♀) on 24 July and 10 September. In the replicated test entries, the correlations between disease ratings on the three dates were highly significant. The correlations were probably influenced by the severity and uniformity of infection, being lowest ($r = +0.821$) in the earlier period of infection build-up from 24 July to 14 August and highest ($r = +0.882$) in the later period from 14 August to 10 September when disease became very severe on susceptible strains.

Major differences in disease reaction were apparent even in the unreplicated entries, some of which were represented by six to seven related clones. Clones 36-42, parents of S-7304 and S-7306, B. inermis were highly resistant as were clones B30-35, parents of S-7305 BI and S-7305 BT, B. inermis × tyttholepis. Clones B55-61 of B. biebersteinii OT1927/8572 were all highly resistant to P. bromi. On the other hand, none of the clones of

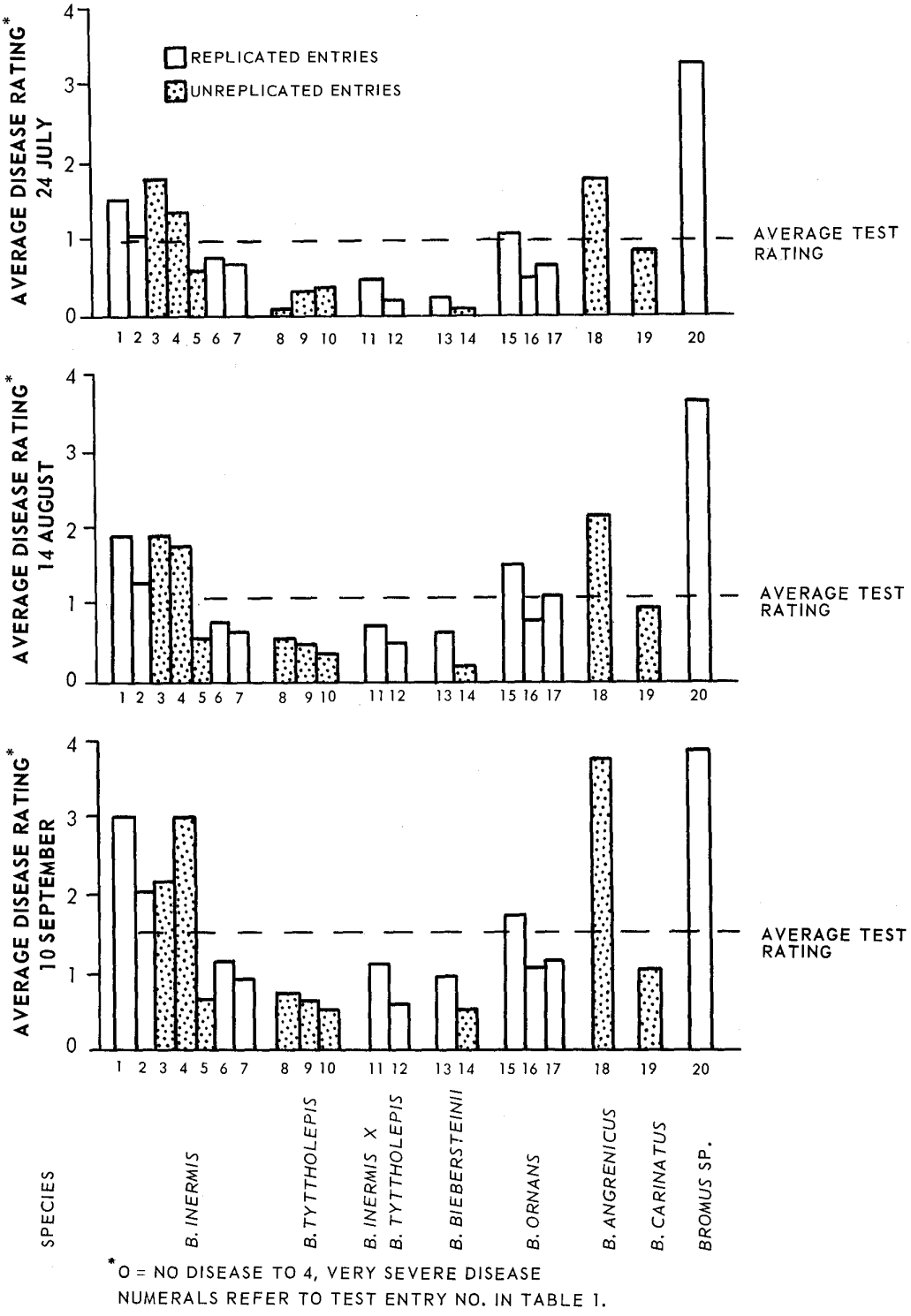

Figure 1. Average disease ratings for *Pyrenophora* leaf spot in *Bromus* spp. and strains on three dates in 1968.

B. angrenicus OT1927/8569 appeared to have much resistance, as ratings were similar to those for Bromus sp. OT1927/9833. None of the other B. inermis introductions showed noteworthy resistance but B. tyttholepis 'Korean' and OT1927/9493 and B. carinatus had low disease ratings. In the unreplicated test entries the correlations between disease ratings on the three dates were highly significant; between 24 July and 14 August, r = +0.931 and between 14 August and 10 September, r = +0.946.

Infection was highly uniform over the four blocks of the test in August and September. This was indicated by coefficients of variability of 11% and 7% in disease ratings on the 125 plants of the infector Bromus sp. OT1927/9833, which served as block markers.

Symptoms of the disease on the resistant B. inermis strains S-7304 and S-7306 and their parent clones B36-42 were very different from those on the susceptible 'Carlton', OT1927/9835 (B. inermis), and B. angrenicus. Foliar lesions on resistant lines were fewer in number, usually smaller in size and with less noticeable halos of yellowed tissues than those on the susceptible sorts. In the resistant lines the leaf lesions frequently were sharply necrotic with charred centres to the spots (Fig. 2). P. bromi was isolated with difficulty from this type of lesion. Occasional plants of 'Lincoln' showed sharp necrotic lesions. Those species and strains which showed rapid vegetative growth, heading, and senescence, e.g. B. angrenicus and B. inermis OT1927/9835 and 'Carlton', had a much higher incidence of Pyrenophora leaf spot than those which developed more slowly e.g. B. inermis S-7304, S-7306, and clones B36-42. Whether the disease contributed to a more rapid senescence or whether senescent tissues were more susceptible and more readily colonized by the pathogen is not known.

All test entries with ratings less than 1.50 on 10 September merit further study. Since the present-day cultivars of B. inermis suitable for use in

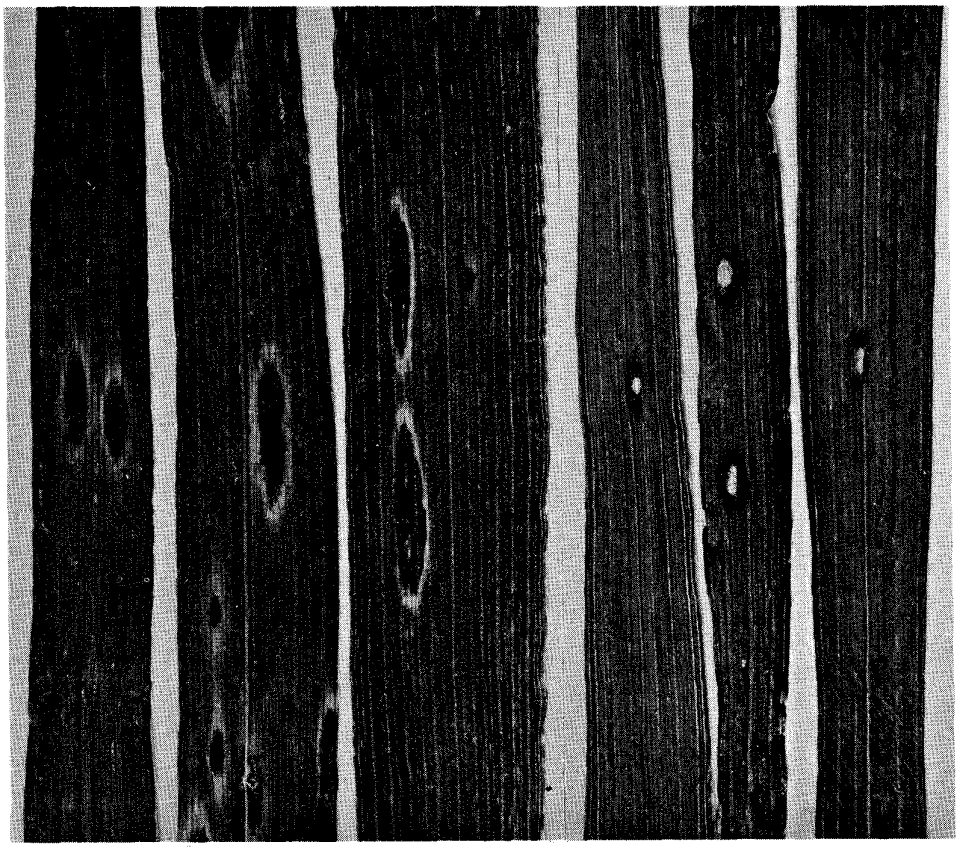

Figure 2. Leaves of a bromegrass susceptible to *Pyrenophora bromi* (left); lessions are large with a pronounced halo. Leaves of resistant bromegrass (right), with small, sharply necrotic lesions.

Western Canada are at best only moderately resistant to P. bromi, the S-7304 and S-7306 synthetics are of the most immediate agronomic interest. Their parent clones, B36-42, were derived by two rounds of mass selection for disease resistance from the high-yielding S-6733 (9), a northern/southern strain bred by R. P. Knowles at Saskatoon. The intermediate line S-7269 in this selection was highly resistant to S. bromigena in tests at Saskatoon in 1968 (unpublished). Since physiologic races of P. bromi may occur, it will be necessary to field test these lines for disease resistance in different localities of North America.

The genetic stability of B. inermis × tyttholepis hybrids is in doubt [(3) and E. L. Nielsen, personal communication] and there may be some difficulties in taking advantage of the resistance to P. bromi derived from B. tyttholepis, as in S-7305 BT. Plants in S-7305 BI and BT were morphologically very variable and some had undesirable agronomic characters.

Clones B55-61 of B. biebersteinii OT1927/8572 that appeared more resistant to P. bromi than those of the 'Regar' cultivar of the same species (5) were also leafier and more vigorous than the latter. Some of the other Bromus spp. which showed high resistance to the pathogen may be of agronomic value in special situations in Western Canada where a bunch bromegrass is desired. B. angrenicus OT1927/8569, which was very susceptible to S. bromigena in test plots at other places in Saskatchewan (unpublished), may be useful in epidemiological studies as in infector plant.

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SCREENING OF POTATO FUNGICIDES 1968[1]

L.C. Callbeck[2]

The fungicides listed below were compared for efficiency in the control of late blight of potato caused by Phytophthora infestans (Mont.) de Bary in the 1968 Screening Test at Charlottetown, P.E.I., Canada. The weight or volume given for a product is the dosage per acre per application.

Copertan. Copper oxychloride. 50% Cu. 2.0 lb. Green Cross Products, Montreal, Quebec

Daconil 2787. Tetrachloroisophthalonitrile. 1.0 lb and 1.5 lb. Diamond Alkali Company, Painesville, Ohio, U.S.A.

Decafentin. Confidential tin compound. 10 oz. Chipman Chemicals Limited, Hamilton, Ontario.

Difolatan 80W. N-(1,1,2, 2-tetrachloroethylsulfenyl) -cis-Δ-cyclohexene-1, 2-dicarboximide. 1.5 lb. Chevron Chemical (Canada) Limited, Oakville, Ontario.

Dithane M-45. Zinc co-ordinated manganese ethylene bis-(dithiocarbamate). 1.5 lb. Rohm and Haas Company of Canada Limited, West Hill, Ontario.

DuTer. Triphenyltin hydroxide (20%). 1.0 lb. Philips-Duphar, Amsterdam, Holland.

Kocide 101. Copper hydroxide (86%). Cu, 50%. 2.0 lb. Kennecott Copper Corporation, New York, N.Y., U.S.A

Nabac 25EC. Hexachlorophene. 8.0 fl. oz. Nationwide Chemical Corporation, Fort Myers, Florida, U.S.A.

Polyram 80W. Zinc activated polyethylene thiuram disulfide. 1.5 lb. Niagara Brand Chemicals, Burlington, Ontario

RH-90. Confidential. 2.5 lb. Rohm and Haas Company of Canada Limited, West Hill, Ontario.

Siaprit. Ethylene thiuram monosulfide. 3.5 lb. S.I.A.P.A., Rome, Italy.

Plots of 'Green Mountain' potatoes, each 4 rows by 50 feet, were planted on June 4, exactly 50 seed pieces being dropped in each row. The treatments were randomized and replicated in 4 ranges. Separating the plots from one another and bordering the design were single rows of the same variety. These rows received no fungicides, their function being to equalize the epidemic, should one develop.

The fungicides were applied with a tractor-mounted sprayer on July 15, 24, August 2, 13, 22, 29, September 9, 17, which schedule gave a mean interval of 9 days.

Insects were controlled by spraying all rows with endosulfan on July 4, July 29, and August 14.

The test was terminated by applying a top killer (sodium arsenite) on September 24, i.e., 112 days after planting. The tubers were dug, graded, examined for rot, and weighed at mid-October.

Table 1. Percentage defoliation*

Treatment	Sept. 13	Sept. 16	Sept. 20	Sept. 24
Copertan	4	9	19	45
Daconil 2787 (1.0 lb.)	3	4	8	18
Daconil 2787 (1.5 lb.)	T**	T	2	6
Decafentin	5	7	14	25
Difolatan 80W	3	4	10	23
Dithane M-45	T	1	3	8
DuTer	7	9	17	36
Kocide 101	9	13	33	58
Nabac 25EC	31	45	89	97
Polyram 80W	3	5	9	17
RH-90	T	T	2	5
Siaprit	3	5	9	21
Check, untreated	65	88	100	100

* Means of five plots.

** T = trace.

1 Contribution No. 191, Research Station, Canada Department of Agriculture, Charlottetown, Prince Edward Island.

2 Plant Pathologist.

Results

The season was the most unusual in the history of this annual fungicide screening program. Adequate rainfall in the early season created a water supply that maintained normal growth of the plants through the driest July on record. The precipitation for the month was only 0.48 inches and the weather continued to be unfavorable to the blight fungus through most of August. Under these conditions, potato growers throughout the province enjoyed a blight-free season, lesions being found in only two small fields of early 'Irish Cobblers'.

It being evident that a natural infection would not occur in the plots, numerous attempts were made to create one artificially by disseminating water suspensions of spores over the plants in the border and buffer rows. The first inoculation was made on July 18 and it was followed by several more, usually just before dark, through the rest of the month and August. By mid-August, only three or four lesions were observed. However, some or all the attempts made on August 23, 24, 25, 26, and 28 were effective, the humidity frequently being high, dews common, and rains of 0.15 and 1.22 inches falling on the 24th and 25th and 0.15 and 0.51 inches on the 29th and 30th.

Under these conditions the disease became well established and the defoliation of the border and buffer rows and of the check plots proceeded at a fast rate in September. In the first week of the month, 0.76 inches fell in five days and in the second there were 0.40 inches in four days.

With the test plots being showered with spores from the source rows, infections developed in them and it became possible to evaluate the relative foliar protection efficiencies of the fungicides. However, the amounts of tuber rot that were found at harvest were lower than might be anticipated; the September rains, with the exception of one of 0.68 inches on the 3rd, were not sufficiently heavy to wash many spores into the hills.

Under the conditions of the test, the best control of disease on the foliage was given by Rh-90, Daconil (at the 1.5 lb. dosage), and Dithane M-45 (Table 1). The two copper fungicides, Copertan and Kocide 101, were relatively poor. Nabac 25EC showed no merit. Difolatan 80W, as in the several previous seasons when epidemics occurred, allowed the least amount of tuber rot (Table 2). No phytotoxicities were observed.

Table 2. Effects of treatments on yield and rot

Treatment	Total (bu/acre)	Smalls (bu/acre)	Rot (bu/acre)	No. 1 (bu/acre)	Rot (%)
Copertan	571.3	62.7	11.0	497.6	1.9
Daconil 2787 (1.0 lb)	637.1	78.5	9.0	549.6	1.4
Daconil 2787 (1.5 lb)	610.9	55.4	7.0	548.5	1.1
Decafentin	593.1	63.8	18.9	510.4	3.2
Difolatan 80W	612.5	58.3	4.2	550.0	0.7
Dithane M-45	622.1	50.6	6.8	564.7	1.1
DuTer	597.0	59.8	13.6	523.6	2.3
Kocide 101	565.6	44.0	9.0	512.6	1.6
Nabac 25EC	554.9	46.2	9.7	499.0	1.7
Polyram 80W	601.3	54.6	18.7	528.0	3.1
RH-90	611.6	44.0	6.6	560.6	1.1
Siaprit	607.4	48.8	15.2	543.4	2.5
Check	496.7	46.6	19.4	430.7	3.9
L. S. D. .05	39.4			40.0	N. S.
L. S. D. .01	52.9			53.7	N. S.

A LEAFHOPPER TRANSMITTED CLOVER DISEASE IN THE OTTAWA AREA

L.N. Chiykowski[1]

Abstract

Ladino clover (Trifolium repens) and alsike clover (Trifolium hybridum) plants exposed to leafhoppers, Aphrodes bicinctus, collected from a field at the Central Experimental Farm, Ottawa, Ontario, developed chlorosis, reddening of leaf margins, adventitious growth at the nodes, witches' broom, and smaller than normal or rudimentary leaves. Flowers were reduced in number and size but appeared to be functional. The percentage of field-collected leafhoppers that induced the disease was increased by feeding the insects on abnormal appearing alsike clover plants obtained from the same field. This together with the time required for symptom appearance indicate that the disease is caused by a transmissible agent. Although most of the individual symptoms have been observed on clover plants infected with various other diseases, the combination of symptoms suggests that this disease may not have been described before.

Introduction

The only causal agent of plant disease known to be transmitted by the leafhopper Aphrodes bicinctus (Schrank) in North America is that causing clover phyllody (2). Since clover phyllody has not been found in the Ottawa area, it was expected that field-collected insects of this species would be free from plant disease inducing agents and could be used in transmission studies on clover phyllody. Accordingly, adult A. bicinctus leafhoppers were collected from a field of grass and volunteer clover at the Central Experimental Farm, Ottawa, and used in an experiment. A group of 25 of these field-collected insects was maintained as a control and was transferred weekly to healthy ladino clover (Trifolium repens L.) or alsike clover (Trifolium hybridum L.) plants. All five plants on which these insects were caged developed symptoms that did not resemble those of clover phyllody.

Symptoms

The following symptoms were observed on plants infected in the greenhouse. The first signs of infection appeared about a month after inoculation in the form of a mild chlorosis of new growth in both ladino and alsike clovers. Plants were generally stunted and leaf margins were reddish or chlorotic (Figs. 1 and 2). Old leaves were cupped downward toward the abaxial side, slightly bronze in color and died prematurely. In ladino clover clusters of small short-petioled leaves with downward cupped chlorotic margins developed at the nodes of the creeping stems (Fig. 3). The single long-petioled trifoliolate leaf that normally develops at the node was often missing. In some plants, creeping stems were absent and numerous small leaves with upright petioles developed, giving the plant a witches'-broom appearance (Fig. 4). Some of the new leaves were slow to unfold and were curved at the ends so that they took on a "pin wheel" appearance. Later developing leaves were rudimentary and consisted of 3 short stubs in place of the normal 3 leaflets (Fig. 5). In alsike clover the crown was thickened and the upright stems produced clusters of adventitious growth at the nodes. These clusters consisted of short-petioled leaves that were reduced in size and had chlorotic margins (Fig. 6). Often the upright stems did not develop and the infected plants took on a witches'-broom appearance (Fig. 7). Although flowers in both species of clover were reduced in number and size, they appeared to be functional.

A search of the area from which the leafhoppers were collected did not reveal clover plants with symptoms typical of those observed on plants infected in the greenhouse. However, several abnormal alsike clover plants were found which had dense crowns of small, short-petioled leaves and three or four upright stems. The foliage of these plants was slightly bronze colored.

Transmission of causal agent

Because most groups of leafhoppers collected from the field were already capable of inducing the disease, the possibility existed that the disease was the result of toxins injected by the insects while feeding rather than a transmissible agent. Although no "healthy" insects were available, it was felt that evidence for or against a transmissible agent being involved could be obtained by exposing field-collected insects to plants with symptoms and determining if the percentage of insects inducing symptoms would increase. Two experiments were conducted using abnormal alsike clover from the field as source plants.

In the first experiment, three groups (27 insects per group) of field-collected leafhoppers were caged

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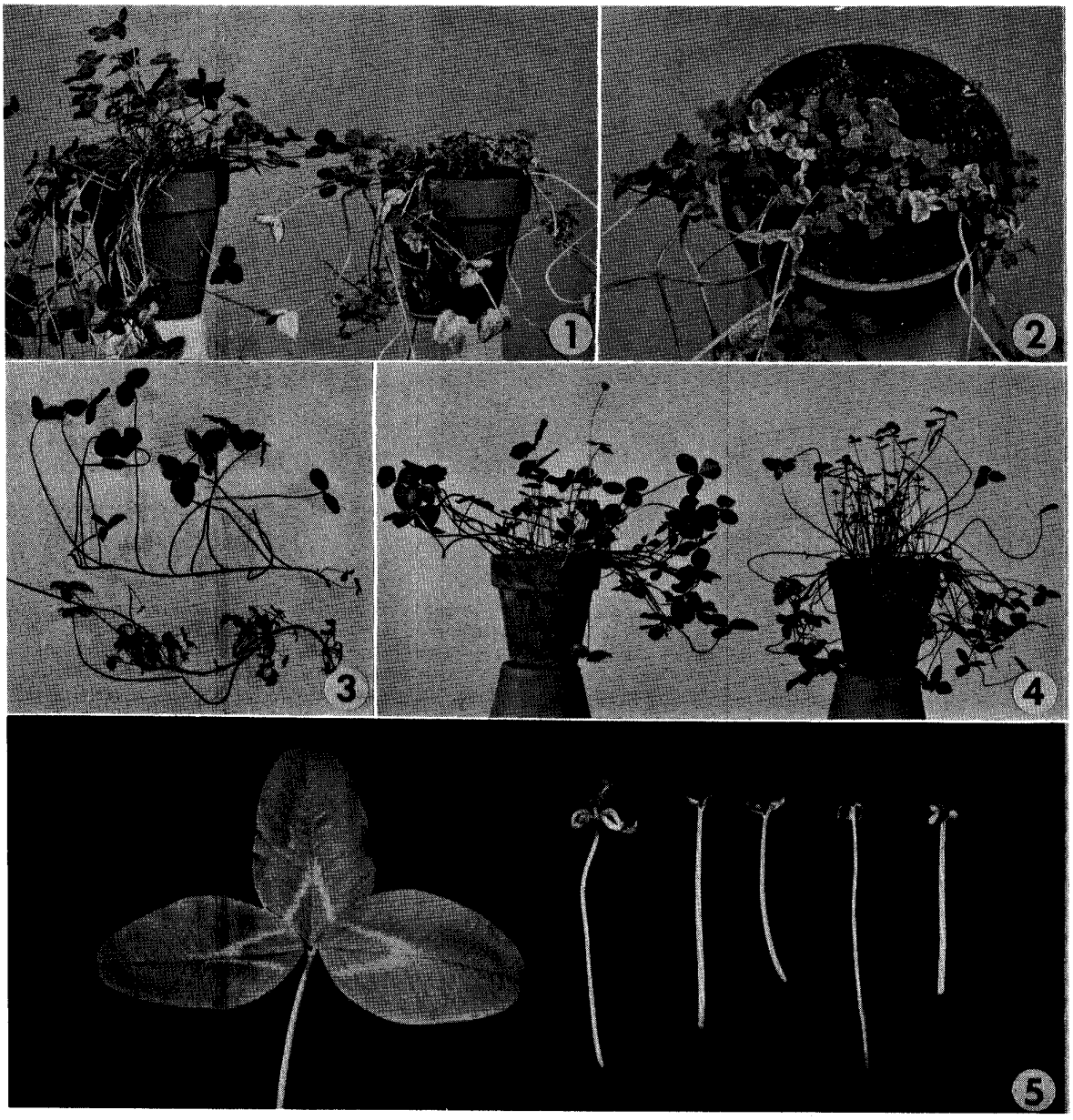
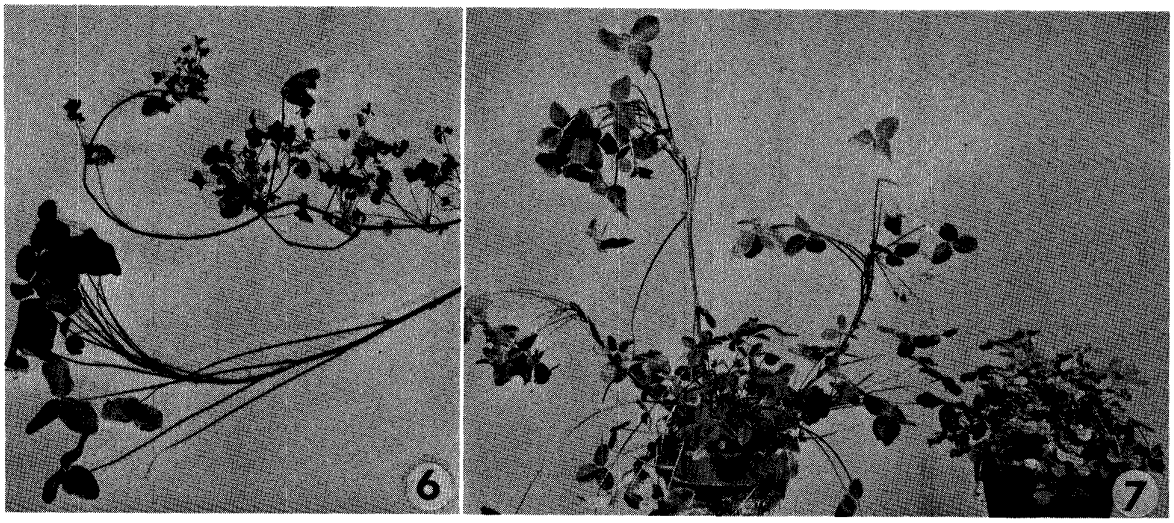


Figures 1–5. Symptoms of an unidentified leafhopper-borne disease on ladino clover (*Trifolium repens*). Figure 1 (right) and Figure 2. Plants stunted and leaves with chlorotic margins; Figure 1 (left) healthy plant. Figure 3. Lower, infected plant showing creeping stem with clusters of small short-petioled leaves at the nodes; upper, healthy. Figure 4. Right, infected plant lacking creeping stems and showing witches' broom. Left, healthy. Figure 5. Right, Infected leaves, showing reduced size, rudimentary leaflets, and pin-wheel symptoms. Left, healthy leaf.

on a healthy ladino clover plant. Each group of exposed and control insects was then transferred weekly to a healthy ladino clover plant. Sixty-two days from the start of the experiment, the 20 survivors from the exposed groups and the 9 survivors from the control group were caged singly for two weeks on alsike clover seedlings.

Two of the exposed groups of leafhoppers induced symptoms on all healthy plants on which they



Figures 6 and 7. Symptoms of unidentified leafhopper-borne disease in alsike clover (*Trifolium hybridum*). Figure 6. Upper, infected stem with clusters of small, short-petioled leaves at nodes. Lower, healthy. Figure 7. Right, infected plant with no upright stems and showing witches'-broom symptom. Left, healthy.

were caged following exposure to the source plants. The third group produced symptoms for the first time on the second healthy plant (18-25 days from the start of the experiment) as well as all subsequent plants on which they were confined. The plant on which the control insects were caged for the first 11 days remained healthy but all subsequent plants developed symptoms. Eleven of the 20 (55%) exposed insects tested singly produced symptoms, while only 1 of the 9 (11%) control insects induced infection.

In the second experiment, 50 field-collected adult leafhoppers were caged for 7 days on each of 3 field-collected alsike clover plants; and as a control, 50 field-collected insects were caged on a healthy ladino clover plant. Both exposed and control insects were transferred weekly to healthy ladino clover plants. Twenty-eight days after the start of the experiment, all surviving insects were caged singly for 2 weeks on healthy ladino clover seedlings.

Each of the three groups of exposed leafhoppers induced symptoms on the first healthy plants on which they were confined (7-14 days from the start of the experiment) and on the two subsequently exposed plants. The control insects also produced symptoms on all plant on which they were caged. When tested singly, 10 of 42 (24%) of the exposed and 1 of 27 (4%) of the control insects caused infection.

Discussion

The results of both experiments showed that the number of field-collected A. bicinctus leafhoppers capable of inducing the disease could be increased by allowing them to feed on diseased clovers. Furthermore, symptoms on plants did not appear until 3 weeks or longer after the insects were removed. Both of these facts point to the conclusion that this clover disease is caused by a transmissible agent. Had a toxin been involved, symptoms should have appeared while the insects were still on the plant or shortly after the insects had been removed.

The symptoms produced and the fact that the causal agent is leafhopper transmitted suggest that this disease may belong to the "yellows" group. Recent findings (3) indicate that this group of diseases may not be caused by viruses as was originally supposed, but rather by Mycoplasma-like organisms. Further studies will be required to characterize the etiological agent involved in this disease.

Attempts to identify this disease on the basis of symptomatology have been unsuccessful. A direct comparison with witches' broom infected clover plants from Vancouver, B. C., revealed differences in leaf shape, size and color. Similarities with Australian legume little leaf were noted by Mr. J. W. Bowyer (personal communication) with the exception that little leaf results in phyllody. Foliar symptoms resembel those described for the parastolbur disease found in Europe (4), but again the flowers of parastolbur infected plants exhibit phyllody. Dr. L. M. Black (personal communication), on first observing the disease, remarked on its general similarity to clover club-leaf, a disease transmitted by Agallian leafhoppers (1). Closer examination, however, disclosed certain differences in leaf shape and coloration. Present indications are that this disease had not been described before.

However, since the present work was done with field-collected insects and source plants, one cannot rule out the possibility that more than one disease or causal agent is involved. It is hoped that the studies on vector-causal agent-host plant relationships now in progress will provide some of the answers.

Acknowledgments

The author wishes to thank Mr. J.E. Lendvay-Zwickl for his technical assistance and Mr. J. Raine, Research Station, Canada Department of Agriculture, Vancouver, for supplying witches' broom infected clover plants.

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SOME RECORDS OF PLANT PARASITIC NEMATODES ENCOUNTERED IN CANADA IN 1968

Robert Sewell[1]

Many plant-parasitic nematodes representing several genera and species were extracted from soil and plant tissues of samples submitted to the Nematology Section, Entomology Research Institute. The majority of samples were submitted by Plant Protection Division, CDA, some by private individuals, florists, horticulturists, etc., and by other Government agencies. Some of these samples came from various parts of Canada, while others were intercepted at port of entry from foreign countries.

ROOT-KNOT NEMATODES (genus Meloidogyne)

Meloidogyne hapla Chitwood, 1949, the northern root knot nematode, was found on carrot from St. Sauveur des Monts, Quebec, cucumber from Manitoba, Spirea sp. from Tennessee, Chrysanthemum sp. from Ottawa, and Berberis thunbergii from Toronto. Meloidogyne incognita (Kofoid and White, 1919) Chitwood, 1949 was found on tomato from Claxton and Tifton, Georgia, and from Richton, Mississippi. Meloidogyne javanica (Treub, 1885) Chitwood, 1949, the Javanese root knot nematode, was removed from roots of tomato (Lycopersicon esculentum) from Tifton, Georgia.

CYST-FORMING NEMATODES (genus Heterodera)

The cyst-forming nematode, Heterodera trifolii Goffart, 1932, was taken from soil obtained in surveys in Quebec and Ontario, and from soil intercepted on rubber tires, greenhouse plants, and cyclamen bulbs from England, on ornamentals, peony, iris and Dahlia sp. from California and New York, and also from soil on cars and machinery from Hamburg, Germany, herbaceous plants and fern, from Portugal, Sedum sp. from Hungary, ornamentals, Spathiphyllum sp., Rhododendron sp. from Italy, and on passengers' baggage from Lebanon and France. Heterodera humuli Filipjev, 1934, the hop cyst nematode, was detected in soil associated with Cactus sp. and fern from France; carnation from Belgium; Merium oleander, and house plants from Italy; Thymus sp. from Portugal, and in soil on a car from Europe. Heterodera punctata Thorne, 1928, the grass cyst nematode, was removed from soil on a car and trailer from Liverpool, England. Heterodera avenae Wollenweber, 1924 (Filipjev, 1934) was encountered in soil taken from cars and machinery and from rosemary plants from Germany; Begonia sp. and herbs from Portugal; greenhouse plants from Yugoslavia; Sedum sp. from Hungary; vegetables from Spain; and Merium oleander from Italy. Heterodera cacti Filipjev and Schuurmans-Stekhoven, 1941, the cactus cyst nematode, was found on ornamentals; Thuja occidentalis woodwardi and palm tree from Tennessee, North Carolina, and New York; also on Lilium sp. from France. This nematode was also present on material collected during a cyst survey of the following areas in Ontario: London, Windsor and Toronto. Heterodera schachtii Schmidt, 1871 was reported on tulip and herbaceous plants from Holland and Portugal. Heterodera goettingiana Liebscher, 1892 was discovered on herbaceous and greenhouse plants from Portugal. Heterodera fici Kirjanova, 1954 was present on mint from Italy and in soil taken from imported agricultural implements. Heterodera rostochiensis Wollenweber, 1923 was detected on Callum sp. intercepted from Scotland. Material examined after a cyst survey and identified as Heterodera sp. was taken from the following: soil from areas around Toronto and Windsor, Ontario, and Montreal, Quebec; soil from Christchurch, New Zealand; soil from rubber tires from England; soil from Sicily; peony and iris plants from Massachusetts; greenhouse plants and Spathiphyllum sp. from Italy; Sedum sp. from Hungary; Geranium sp. and Cactus sp. from Europe; soil from France.

ROOT-LESION NEMATODES (genus Pratylenchus)

Pratylenchus crenatus Loof, 1960 was found in soil associated with strawberry from Burlington, Washington, and from Christchurch, New Zealand. Pratylenchus penetrans (Cobb, 1917) Filipjev and Schuurmans-Stekhoven, 1941 was found in soil from strawberry from Burlington, Washington, and from Ligustrum vulgare from Boskoop, Holland, and on vegetables from Berwick, Nova Scotia. Pratylenchus pratensis (de Man, 1880) Filipjev, 1936 was screened from soil associated with strawberry from Burlington, Washington. Pratylenchus neglectus (Rensch, 1924) Filipjev and Schuurmans-Stekhoven, 1941 was encountered on strawberry from Burlington, Washington.

SPIRAL NEMATODES (genera Helicotylenchus and Rotylenchus)

Helicotylenchus platyurus Perry in Perry, Darling and Thorne, 1959 was extracted from soil off Tsuga canadensis from McMinn, Tennessee, and Helicotylenchus sp. was found on Ligustrum sp. Helicotylenchus sp. was taken from soil from Christchurch, New Zealand. Rotylenchus robustus (de

1 Nematology Section, Entomology Research Institute, Canada Department of Agriculture, Ottawa.

Man, 1876) Filipjev, 1936 was found on Azalea sp. from Maryland.

STUNT NEMATODES (genus Tylenchorhynchus)

Tylenchorhynchus claytoni Steiner, 1937, the tobacco stunt nematode, was discovered on Rhododendron sp. from Eden, New York, and on Azalea sp. from Maryland. Tylenchorhynchus longus Wu, 1969 was found on balsam and poplar from Bow River forest, Alberta. Tylenchorhynchus brevidens Allen, 1955 was removed from soil off blueberry plants from Geneva, New York.

PIN NEMATODES (genus Paratylenchus)

Paratylenchus projectus Jenkins, 1956 was removed from soil off Rhododendron sp. from Eden, New York.

RING NEMATODES (genus Criconemoides)

Criconemoides demani Micoletzky, 1925 was discovered on vegetables from Berwick, Nova Scotia.

BUD AND LEAF NEMATODES (genus Aphelenchoides)

The Chrysanthemum sp. foliar nematode, Aphelenchoides ritzemabosi (Schwartz, 1911) Steiner and Buhrer, 1932, was found infesting Chrysanthemum sp. from Morden, Manitoba. An Aphelenchoides sp., possibly A. fragariae (Ritzema Bos, 1890) Christie, 1932 was found on strawberry from Washington, Also Aphelenchoides sp. on blueberry plants from Avondale, Newfoundland.

SEED GALL NEMATODES (genus Anguina)

Anguina sp. was extracted from soil with bentgrass from Halsey, Oregon.

DORYLAIMIDS

The American dagger nematode, Xiphinema americanum Cobb, 1913 was found in soil with raspberry plants and Tsuga canadensis from Michigan. Xiphinema sp., possible X. americanum, was recorded on Spartina basiliensis from Guyana.

MISCELLANEOUS (tylenchids)

Tylenchus sp. was discovered on Spartina basiliensis from Guyana and on Acer platanoides from Holland. Ditylenchus dipsaci (Kuhn, 1857) Filipjev, 1936 was found infesting gladiolus bulbs from Washington and Allium sp. intercepted from Germany.

OBSERVATIONS ON CRANBERRY FRUIT ROTS IN NOVA SCOTIA, 1945–55[1]

C.O. Gourley and K.A. Harrison[2]

Abstract

In Nova Scotia, a study of cranberry (Vaccinium macrocarpon) fruit rots between 1945 and 1955 showed that rot seldom occurred until after the fruit was harvested. End rot caused by Godronia cassandrae f. vaccinii was the most important rot of cranberry fruit. Sterile breakdown, which occurred most frequently in refrigerated and immature fruit, was the second most important cause of fruit loss in storage. Ceuthospora lunata and Sporonema oxycocci were isolated more often from infected fruit grown in a well-managed bog than from fruit produced in neglected or wild bogs. Guignardia vaccinii was the most abundant fungus on the skin of healthy fruit, but it seldom caused fruit rot. Latent infections of G. cassandrae f. vaccinii on the calyx may be the main source of inoculum for the initiation of storage rot by this fungus.

Introduction

Decay and breakdown of harvested fruit of cranberry, Vaccinium macrocarpon Ait., caused by pathogenic fungi and physiological factors are well documented in the literature (1, 2, 10, 11, 12, 13). Fruit decay may appear before harvest, during storage, or on the market. The amount of fruit breakdown varies from year to year among different areas of production, and among individual bogs.

Early rot caused by Guignardia vaccinii Shear; blotch rot, by Acanthorhynchus vaccinii Shear; bitter rot, by Glomerella cingulata (Stonem.) Spauld. & Schrenk; and end rot, by Godronia cassandrae Pk. f. vaccinii Groves have been reported to be the most important fungus diseases of cranberry fruit (12,13). Zukerman (13) reported that ripe rot caused by Sporonema oxycocci Shear is currently a more abundant fruit pathogen in Massachusetts than in the past, and that the relative importance of several other rot organisms has changed. He also confirmed earlier reports that it is impossible to distinguish between cranberry rots without resorting to isolation and study of the causal organisms in culture.

Storage breakdown is one of the major problems of harvested cranberry fruit. Doughty et al. (6) reported that losses from physiological sterile breakdown in storage may reach 30% to 35% in some years. They also reported that a small amount of rot can be attributed to pathogenic organisms. Cranberries can normally be held in storage as fresh fruit for 2 to 3 months at 2.2 C to 4.4 C and a relative humidity of 80-85% (9).

In 1966, Chandler and Murray (4) conducted a study of the feasibility of the cranberry industry in western Nova Scotia which resulted in an expansion of this industry. Because of this renewed interest in cranberry production, records taken from 1945 to 1955 on the isolation, identification, and relative importance of the fungi and physiological factors that caused rot and breakdown of cranberry fruit are presented here.

Materials and methods

Potato dextrose agar (PDA) was the culture medium used throughout these studies. Most of the fungi isolated from cranberry fruit grew and sporulated on PDA and they were identified from spore structures and from their characteristic growth habits on this medium. All fruits from which isolations were made were surface sterilized with 70% ethanol. The isolation procedures were those normally employed for the isolation of pathogenic fungi from infected fruit; the skin was removed with a sterile scalpel and a small bit of tissue from the leading edge of the rot was transferred to PDA in petri plates. Skin isolations were made by shaving thin 3-5 mm^2 areas from the surface of the fruit and plating them on PDA. Unless otherwise stated the results were recorded as the percentage occurrence of fungal species or sterile breakdown in the rotted fruit from each sample.

In December 1945 a 5 lb sample of cranberries was obtained from the commercial packing houses at each of the bogs at Arichat in Richmond County and Auburn in Kings County, and isolations were made from the infected fruit. At the same time, a 1-lb sample of cranberries from each of the bogs at Aylesford and Lakeville, Kings County, was purchased from a retail outlet and stored in a refrigerator at approximately 5C for 1 month before isolations were attempted from the infected fruit in each sample.

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[1] Contribution No. 1309, Research Station, Canada Department of Agriculture, Kentville, Nova Scotia. This article was written by the senior author and is based largely on unpublished data from the files of Dr. K. A. Harrison, who retired in 1966.

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No detailed study of cranberry fruit rot was made between 1946 and 1954. However, during this period observations were made on the fungi found on unripe and mummified fruit, on berries found floating on flooded bogs, on berries that had overwintered on the bogs, and on blighted cranberry plants in cultivated bogs. Most of these observations were supported by isolations to determine the causal organisms, and they are included here as part of the cranberry disease syndrome in Nova Scotia.

In January 1955, isolations were made from infected fruit harvested from the following bogs in Kings County:

Palmer - a neglected bog in which renovation had commenced in 1954. Berries were of good size and well colored;

Oyler-Walker - one of the older, well managed bogs with natural frost protection. Fungicides and insecticides were applied for disease and insect control. Berries were large, well colored, and had been stored at approximately 3.3C;

Neglected - a bog without frost protection where the fruit had to be harvested before it was fully mature. Berries were small and poorly colored;

Wild - a natural bog along the seashore. Berries were small, well colored, and badly bruised from rough handling.

To determine if fruit rots could be initiated through the calyx, isolations were made from the dried adhering sepals and decaying flesh beneath the calyx from fruit of the Palmer and Wild bogs. Isolations were also made from the calyx and skin of healthy fruit from the same bogs. The frequency of fungal isolates from infected and healthy fruit was expressed as a percentage of the total fruit examined for each series of isolations.

It is not uncommon for cranberry fruit to have small red spots on the skin that are more prominent when the berries are poorly colored. A sample of light colored berries with prominent red spots was selected from among the healthy fruit from the Palmer and Wild bogs. Isolations were made from the red spots on the skin, from the flesh beneath the red spots and from apparently healthy areas of the skin. Isolations were also made from the skin of well colored, non-spotted berries from the same bogs. The incidence of each fungal species obtained in culture was recorded as a percentage of the total isolations for each test.

Results

In 1945, the end rot fungus G. cassandrae f. vaccinii was isolated from a greater number of infected cranberry fruit than other fungi (Table 1). This soft, watery rot that started at the calyx end and quickly involved the whole berry was the most prominent rot in the samples from the four bogs. About 24% of the rotted fruit were sterile, and sterility was greatest in the fruit that had been refrigerated. A. vaccinii and G. vaccinii were obtained in culture only when a section of a dark, blotchy area on the skin was included with the plated tissue.

In the period from 1946 to 1954, G. cassandrae f. vaccinii was the most abundant fungus found in infected unripe, ripe, or mummified cranberry fruit. From 13% to 57% of the decay in stored fruit was end rot, and the amount of this rot varied with the season and the location of the bog. S. oxycocci was the most abundant pathogen in fruit from flooded bogs. It was isolated from berries left on the bog over winter and was easily obtained from the remains of berries along the banks of previously flooded bogs. Pestalotia vaccinii (Shear) Guba and Diaporthe vaccinii Shear were isolated occasionally and were more prevalent on blighted twigs than on fruit. Few isolates of black rot caused by Ceuthospora lunata Shear were obtained from rotted fruit.

In 1955, G. cassandrae f. vaccinii was one of the major causes of breakdown in cranberry fruit from the four bogs (Table 2). Sterile breakdown was more prevalent than end rot in the immature fruit from the neglected bog. More black rot and ripe rot were found in the fruit from the Oyler-Walker bog than in fruit from the other three bogs.

G. cassandrae f. vaccinii was the most frequently isolated fungus from the calyxes and flesh of rotted fruit, while G. vaccinii occurred most often in the calyxes and skin of healthy fruit (Table 3). In general there was a good correlation between the incidence of a fungus species in the flesh with that in the calyx of rotted fruit. With healthy fruit G. vaccinii was more prevalent in the skin than in the calyxes.

When isolations were made from red spots, light-colored, or well-colored skin, G. vaccinii was present on the skin of a greater number of fruit than any other fungus (Table 4). This fungus was isolated more often from the red spots on the skin of light-colored fruit than from well- or light-colored skin. In a few instances, G. vaccinii was isolated from the flesh beneath red skin spots.

Discussion and conclusions

The rot diseases of cranberries caused by the different fungi vary greatly in their prevalence in the different cranberry-growing regions of North America. Early rot caused by G. vaccinii is the most serious one in New Jersey; bitter rot caused by G. cingulata is most important in Massachusetts; while end rot caused by G. cassandrae f. vaccinii is the most serious cranberry rot in Wisconsin and on the

Table 1. Percentage incidence of fungi from rotted cranberry fruit at four locations in 1945

Type of rot	Fungus isolated	Source* of fruit				Mean
		Arichat	Auburn	Aylesford	Lakeville	
Blotch	Acanthorhyncus vaccinii	0	3.2	8.0	0.6	4.1
Black	Ceuthospora lunata	7.0	14.8	0	0	3.1
Bitter	Glomerella cingulata	5.8	3.2	0	0	1.0
End	Godronia cassandrae f. vaccinii	48.8	36.7	57.4	42.9	48.6
Early	Guignardia vaccinii	0	4.5	11.6	3.8	6.9
	Penicillium spp.	0	2.5	1.2	5.8	2.8
	Pestalotia vaccinii	24.4	9.0	2.6	0	4.7
Ripe	Sporonema oxycocci	4.6	11.6	8.7	2.2	6.7
	Unidentified fungi	1.1	11.6	4.1	8.6	6.5
Sterile breakdown		9.3	13.5	19.0	40.0	24.1
Number of diseased fruit examined		86	155	413	312	

* Samples were obtained in December 1945; those from packing houses at Arichat and Auburn were examined immediately, while those from bogs at Aylesford and Lakeville were refrigerated at 5C for 1 month before examination.

Table 2. Percentage incidence of fungi from rotted cranberry fruit produced at four bogs in 1955

Type of rot	Fungus isolated	Source of fruit				Mean
		Palmer	Oyler-Walker	Neglected	Wild	
Blotch	Acanthorhyncus vaccinii	2.8	4.3	0.9	8.6	4.6
	Botrytis cinerea	0	0	0	0.5	0.1
Black	Ceuthospora lunata	0	12.9	1.8	0	2.9
	Diaporthe vaccinii	2.0	2.9	0	15.8	6.0
Bitter	Glomerella cingulata	0	0	0.9	0.5	0.3
End	Godronia cassandrae f. vaccinii	88.8	59.7	41.6	42.1	60.3
Early	Guignardia vaccinii	5.0	0	3.7	14.8	5.2
	Penicillium spp.	0	5.7	4.6	18.1	7.3
	Pestalotia vaccinii	0	1.4	0.9	0.5	0.6
Ripe	Sporonema oxycocci	9.5	17.2	4.6	5.2	9.0
	Unidentified spp.	0.4	3.6	0.9	2.7	1.6
Sterile breakdown		2.8	10.8	53.7	12.4	15.2
Number of diseased fruit examined		241	139	108	209	

Table 3. Percentage incidence of fungi from diseased and healthy tissues of cranberry fruit grown in two bogs in 1955

Fungus isolated	Palmer bog				Wild bog			
	Diseased		Healthy		Diseased		Healthy	
	Flesh	Calyx	Fresh	Calyx	Flesh	Calyx	Skin	Calyx
Acanthorhyncus vaccinii	0	3.4	0	10.0	10.0	5.0	10.0	12.5
Botrytis cinerea	0	0	0	0	1.0	0	0	0
Diaporthe vaccinii	15.9	14.7	0	0	14.0	21.0	0	10.0
Glomerella cingulata	0	0	0	0	0	1.0	0	0
Godronia cassandrae f. vaccinii	86.1	76.1	0	33.0	38.0	42.0	7.5	10.0
Guignardia vaccinii	13.8	12.5	83.7	33.0	4.0	20.0	67.5	35.0
Penicillium spp.	1.0	0	0	0	15.0	22.0	5.0	42.5
Sporonema oxycocci	5.3	3.4	0	6.0	7.0	9.0	0	10.0
Unidentified fungi	4.2	6.8	0	1.0	2.0	13.0	0	0
Sterile	2.1	0	16.0	3.0	17.0	11.0	22.5	10.0
Number of fruit examined	94	88	30	30	100	100	40	40

Table 4. Percentage incidence of fungi from the skin and flesh of cranberry fruit in 1955

Fungus isolated	Appearance of skin			Flesh beneath red spots
	Well-colored	Light-colored	Red spots	
Acanthorhyncus vaccinii	1.1	0	0	0
Diaporthe vaccinii	0.5	0	0	0
Godronia cassandrae f. vaccinii	0.5	0	0	0
Guignardia vaccinii	54.2	21.9	79.7	6.0
Unidentified fungi	0	0.8	0	0
Sterile	43.8	77.3	20.3	94.0
Number of isolations	628	128	128	100

Pacific Coast (5, 7, 13). Hall et al. (8) reported that the incidence of fruit rot is determined largely by the mean temperature during the growing season. In New Jersey, the fruit may rot in the bogs before harvest, but in Nova Scotia, rotting seldom occurs until after the fruit is stored.

In Nova Scotia from 1945 to 1955, G. cassandrae f. vaccinii was isolated from more infected fruit than any other fungus and was the most important cause of breakdown in stored fruit. Botrytis cinerea Pers. and Penicillium spp. are not normally considered to be pathogens of cranberry fruit, but

they may colonize berries through cracks and bruises caused by rough handling during harvest.

Cranberry fruit held in storage for a month or more may show sterile breakdown at temperatures above freezing, especially above 10C, whereas those stored just below freezing may show much low temperature breakdown (5). Sterile breakdown was the second most important cause of loss of harvested fruit in Nova Scotia. Immature fruit may be more susceptible to sterile breakdown than mature fruit. When the fruit was harvested early from the Neglected bog, there was more sterile breakdown than fungus decay. Sterile breakdown ranked fourth after end rot, ripe rot, and black rot, as the cause of breakdown of fruit from the Oyler-Walker bog.

There are few reports in the literature on the colonization of the calyxes of cranberry fruit by fungi and the correlation of these calyx fungi with the causes of fruit rot. Here G. cassandrae f. vaccinii was the most prevalent fungus in the calyx and flesh of infected fruit and it was present on the calyxes of healthy fruit. The calyx may be one of the chief avenues of fruit infection, since end rot, as the name implies, most often occurs in Nova Scotia at the calyx end of the fruit.

Zukerman (13) reported that a great percentage of cranberries contain rot fungi at harvest, but they do not breakdown unless the proper stimulus or "triggering action" occurs. Here G. vaccinii was the most abundant fungus on the skin of healthy fruit, but it was not an important fruit rot pathogen. It was found in only a few rotted fruit, where it was usually associated with other more aggressive rot fungi. Although G. cassandrae f. vaccinii was the dominant fungus isolated from the calyx and flesh of infected fruit, it rarely occurred on the skin of healthy fruit. In Wisconsin, Carlson and Boone (3) found three fungi, A. vaccinii, G. vaccinii, and Gibbera compacta (Pk.) Shear associated with a berry speckle disease of cranberry. In Nova Scotia, G. vaccinii was most frequently associated with red spots on the skin, the flesh beneath the red spots, and the skin of unblemished fruit.

Zukerman (13) found that recent changes in the relative importance of several rot organisms in Massachusetts may have been due to changes in cultural practices. In this study Sporonema oxycocci and Ceuthospora lunata were relatively more important fruit pathogens in the well-managed Oyler-Walker bog than in the other three bogs.

In Nova Scotia end rot is the most important cause of cranberry fruit decay in storage. The fungus found most commonly on the skin of healthy fruit was G. vaccinii, the causal agent of early rot, but it is not an important fruit pathogen in Nova Scotia.

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BRIEF ARTICLES

SUNFLOWER DISEASES IN MANITOBA IN 1968

J.A. Hoes[1]

Sclerotina sclerotiorum (Lib.) de Bary and Verticillium dahliae Kleb. were the most important pathogens of sunflower (Helianthus annuus L.) in Manitoba in 1968. The former caused both head rot and stalk rot but was especially prominent in the head rot phase (Table 1). Most heads affected by S. sclerotiorum were a complete loss and infection must have taken place early and been followed by rapid disease development. Usually the head rot phase of S. sclerotiorum is scarce or absent while the stalk rot phase is more prominent. Presumably the young plants emerging in June escaped infection because this month was relatively dry. Precipitation in July and August, however, was more than twice the long term average amount of 5.3 inches for the two months, while the respective mean temperatures were 2.3F and 5.7F below normal. The prolonged cool and wet weather favored the continued production and discharge of ascospores (3), which infected the young heads, but apparently not the stalks of the plants. Sackston (2) attributed the relative abundance of head rot in 1951, at least in part, to low temperatures and frequent rains during August and September. Head rot was also due to Botrytis cinerea Pers. but damage here was less conspicuous; it was not found in 12 fields, and in the 19 other fields it affected only 1-5% of the plants. Diseased heads were only partially invaded, and environmental conditions had apparently been less favorable to Botrytis than to Sclerotinia.

Table 1. Frequency of head and stalk rot phases of Sclerotina disease in 31 sunflower fields

Phase	No. of fields with affected plants in the following percentage classes: 0	1-5	6-10	11-40
Head rot	2	26	1	2
Stalk rot	16	14	1	0

Verticillium dahliae was as prominent as in other years even though the season was abnormally wet and cool. Of 31 fields, 10 showed not more than 1% infection, 15 fields had 5-20% infected plants, and three fields of 'Commander' had 50-75% diseased plants. The variety 'Peredovik' was generally more resistant than 'Commander', confirming previous observations (1). Downy mildew caused by Plasmopora halstedii (Farl.) Berl. & de Toni was found in only 2/31 fields, causing a trace of infection. Leafspot caused by Septoria helianthi Ell. and Kell. and rust caused by Puccinia helianthi Schw. occurred in all fields but infections were light and no damage was caused. The variety 'Peredovik' showed generally a much lower density of rust pustules than 'Commander' even though in greenhouse studies both are equally susceptible in the seedling stage; 'Peredovik' displays a certain amount of adult plant resistance. Light frost caused serious damage in early planted fields in the Carberry area. Top leaves turned brown and young affected heads showed brown discoloration just below the point of insertion of the florets. Affected heads did not develop.

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FIELD BEAN DISEASE SURVEY IN ONTARIO—1968

V.R. Wallen[1]

In 1968, 13 foundation plots of 'Seaway' and 8 foundation plots of 'Sanilac' field beans were inspected for bacterial blights and other diseases. The various plots ranged between ½ and 2 acres and were located in western Ontario primarily near Hensall, Kippen, and Blenheim. In addition, 23 fields of first-generation and commercial beans were also inspected.

Infected plant tissues, primarily leaves and pods, were collected from infected fields and examined in the laboratory. Common blight caused by Xanthomonas phaseoli (E.F. Sm.) Dowson and fuscous blight caused by Xanthomonas phaseoli var. fuscans (Burkh.) Starr and Burkh. were differentiated by the formation of a brown diffusible pigment by cultures of the fuscous blight organism on nutrient agar.

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Five of the foundation plots were infected by bacterial blight (one of 'Sanilac' and four of 'Seaway'). Within the five plots, considerable variation in the amount of infection was evident. Three plots showed at least 10 areas of infection, and infection in two of the plots was the result of secondary infection from diseased beans in adjacent fields. Two plots showed only a trace of infection with one and two foci of infection, respectively. All infections in foundation plots were of common blight except one plot of 'Seaway' which was infected with fuscous blight resulting from secondary infection

All 23 registered and commercial fields inspected were infected with common blight or fuscous blight or both to varying degrees. Registered fields, the progeny of last year's foundation plots, were generally slightly infected, while infection in commercial fields ranged from 5% to 100%. Twelve commercial fields were infected with fuscous blight. No registered fields were infected with fuscous blight. Twenty-one of twenty-three commercial and registered fields were infected with common blight.

In foundation plots in 1967 and again this year, blight infection originating from infected seed was entirely caused by X. phaseoli. In the 1968 registered crop, arising from 1967 foundation seed, only X. phaseoli was isolated from diseased plant material. However in commercial crops, the seed of which had been propagated for a number of years in Canada, X. phaseoli var. fuscans was prevalent. From 1962 to 1964 fuscous blight was the principal bean pathogen in Ontario (1), and it is still present in the older Ontario seed stocks. There has been no indication that fuscous blight is present in the breeder seed that is imported from Idaho and California to produce the foundation plots.

Sclerotinia rot caused by Sclerotinia sclerotiorum (Lib.) de Bary was found in 10 of the foundation plots and ranged from trace to slight in intensity. It was also present in most of the registered and commercial fields. The disease was much less severe than in 1967 (2) and little damage is expected.

Root rot, caused primarily by Fusarium solani (Mart.) App. and Wr. f. phaseoli (Burkh.) Snyd. & Hansen, was prevalent in eight fields or plots, ranging from a trace to severe in intensity. Most affected fields showed small infected areas of less than 1% of the crop; however in one field more than 50% of the plants were severely infected.

Rust caused by Uromyces phaseoli (Rebent.) Wint. was present in seven fields and ranged from a trace to severe on individual plants. Localized areas of infection in three fields caused early maturity of the crop. Infection was the most severe since field bean surveys were initiated in 1961.

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NEMATODE LEAF BLIGHT OF CHRYSANTHEMUM[1]

J.A. Hoes and H.F. Harp[2]

An epidemic of the leaf nematode Aphelenchoides ritzemabosi (Schwartz) Steiner and Buhrer occurred in 1968 at Morden in observation plots of hardy varieties of Chrysanthemum morifolium (Ramat.) Hemsl., the florists' chrysanthemum. Initial symptoms consisted of brown to black wedge-shaped interveinal leaf areas; later the entire leaf turned brown and necrotic. Severely diseased plants looked withered, all leaves being dead and adhering to the stem. Numerous nematodes were present in partially affected leaves. The nematodes were identified by Dr. K. C. Sanwal, Entomology Research Institute, C. D. A., Ottawa, Ontario. Total precipitation during the growing season was almost twice the long-term average of 12.8 inches. The excessive rainfall no doubt contributed to the severe infection (2).

The varieties were planted in rows or blocks in 1966 and plants touched each other affording easy spread to the pathogen. All plants of a given variety were either severely infected or were completely or nearly symptomless. The apparent uniformity of the infestation suggested that observed differences were real and that classification into resistant and susceptible varieties was justified. Highly susceptible varieties were 'Archibald', 'Beckethau', 'Cameo', 'Candy', 'Cartier', 'MacDonald', 'Pelican', 'Skyline', 'Tilley', and Morden No.'s 6607, 6613 and 6617. Suggested to be resistant are 'Brightness', 'Brown', 'Canary', 'Galt', 'Howe', 'Paige's Gold', 'Sutherland', 'Tupper', 'Whelan', and Morden No.'s 6408, 6608, 6614 and 6618. Hesling and Wallace (1) demonstrated that chrysanthemum varieties differ greatly in their susceptibility to A. ritzemabosi. None of their varieties, however, are among the varieties mentioned in this paper.

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[1] Contribution No. 78, Research Station, Canada Department of Agriculture, Morden, Manitoba.

[2] Plant Pathologist and Horticulturist, respectively.

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CHIVES RUST AT OTTAWA, ONTARIO[1]

D.B.O. Savile

Rust, caused by Puccinia mixta Fckl., has long been known on chives (Allium schoenoprasum L.) from the vicinity of Victoria and Vancouver, B. C., most of the available records having been listed by Savile (2). This rust, which has often been confused with Puccinia porri (Sow.) Wint. and other species (2), is practically confined to chives; but onion (Allium cepa L.) is occasionally attacked when grown close to infected chives. It was recorded by Arthur (1) from Washington, on the Pacific coast, and from Connecticut and New York, on the Atlantic coast. There is also a specimen in DAOM from Woods Hole, Massachusetts. The rust has apparently not previously been recorded in eastern Canada or far inland.

On 14 June 1969 a heavily rusted clump of chives (Savile 5032) was found in a garden at Qualicum on the outskirts of Ottawa. Uredinia were abundant and telia already common. The intensity of infection indicated that the clump must have been infected in 1968. However, there was no infection in a clump in the adjoining garden, from which the rusted clump was derived in 1967. On 10 July 1969 Dr. R. V. Clark discovered a lightly infected clump, with uredinia predominant, in a garden at Meadowlands, about 3 miles from Qualicum.

All North American specimens examined fall within the range of variation seen in European specimens; but different outbreaks seem to stem from separate introductions. In the British Columbia specimens 2-celled teliospores range to 43 μ long, and 1-celled spores are common. In the Ottawa specimens 2-celled teliospores range to 53 μ long, and 1-celled spores are few. In the Massachusetts specimen (W. R. Taylor, July 1947, DAOM ex herb. Wehmeyer) 2-celled spores range to 50 μ long and 1-celled spores are abundant. Thus we have at least three biotypes of this rust in North America, apparently separately introduced with seeds or bulbs. The plant is ordinarily sold as seed, to which detached teliospores may possibly adhere; but it is probable that bulbs, with sori on the scales, are occasionally brought in clandestinely in settlers' effects.

Because chives is grown predominantly in home gardens the extent of the outbreak is not easily assessed. This preliminary note is presented to encourage readers to check all plantings that they see.

Control of the rust may prove difficult, since the summer use of fungicides would defeat the purpose of growing the plant. However it may be practicable to use a fungicide very early, in the period of teliospore germination. It is probably advisable, where chives is grown in a flower bed that is sprinkled regularly, to cover the plants with plastic during sprinkling, and apply water gently to the soil. It is proposed to dig the infected plants of Qualicum in the fall, clean them off thoroughly, and replant them in shady sites where they will require less watering. A few plants will be left untouched until spring and watched for development of aecia, which are not often reported.

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POTATO LATE BLIGHT IN CANADA IN 1844—45

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The following paragraph is quoted from a study (1) of the historic epidemic of potato blight (Phytophthora infestans (Mont.) de Bary) which broke out in Europe in 1845:

> "Although the attack took Europe completely by surprise, blight had already been ravaging the potato crops of North America in the previous two seasons. Stevens (2) has charted the annual progress of the disease from its beginnings in 1843 in the five States closest to the great ports of the east coast of the United States, to an expanded area in 1845 which closely approximates to the limits within which blight is a serious

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problem to-day. Fig. 1 is based primarily on Stevens's work, with the addition of a limited amount of extra information on the impact of the disease in Canada. The diagram bears quite a resemblance to charts of ten-yearly progress of the Japanese beetle from its introduction near Philadelphia in 1916."

The purpose of the present note is to give particulars of the 'limited amount of extra information on the impact of the disease in Canada' which was found in European libraries.

The Dublin heart specialist, Dr. Bellingham (3), received a letter from a Canadian resident who reported the appearance of the disease in 1844 on potatoes grown on a mountain slope about three miles from Montreal; 'the leaves were marked with black spots as if ink had been sprinkled over them'. The disease extended 'over several hundred miles of country including Lower and Upper Canada, and the Northern States'. It recurred in 1845.

The German Dr. Focke (4) quoted from the Quebec Gazette of 1 October 1845 to the effect that indeed it had made an appearance in the province in the previous year.

The eastward extension of the disease in 1844 seems to have stopped short of Nova Scotia which already had a reputation, mentioned by Bosson (5), as a 'famous potatoe growing country'. In the following year the potato crop in New Brunswick and Nova Scotia was unmistakably attacked, according to a letter sent to Professor James Johnston (6) in Scotland by a Mr. Thomas Gilchrist of St. John on 27 September 1845. Although everything had seemed fine two months earlier, now the crop was universally damaged; 'from Halifax to St. John I did not see a single field of potatoes but what was completely destroyed'. It was a strange and novel disease; 'it first attacks the shaw, and so rapid is it that in the course of one or two nights a whole field will be destroyed, and the stench which arises from them is almost unbearable'.

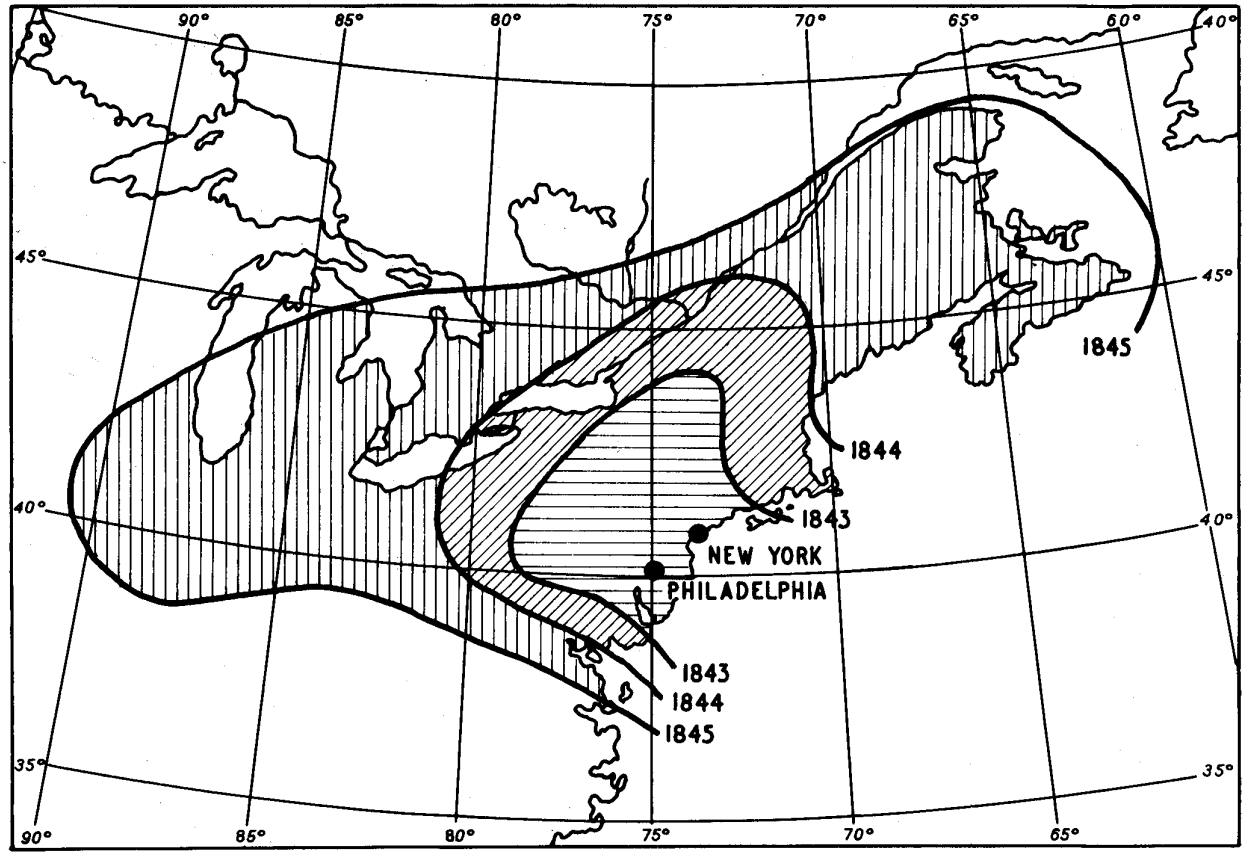

Figure 1. Approximate extent of potato blight attacks in the U.S.A. and Canada in the years 1843–45 (After Stevens).

Some paragraphs in the Gardiners' chronicle (7) confirms the broad Canadian picture without adding further geographical detail. They do mention, in line with some reports from Ireland, that in the valley of the Richelieu river the variety Irish Cup had stood up rather better to the disease than other kinds.

This handful of reports is barely sufficient to outline the approximate extent of the penetration into Canada of the disease in 1844 and 1845. There must exist in North American libraries a mass of relevant information in contemporary local newspapers, periodicals and pamphlets. Perhaps some Canadian student of historical phytopathology may be tempted to make use of these untapped resources, firstly to confirm that the year 1844 saw the first appearance of late blight in the Canadian potato crop, and secondly to define more accurately the geographical limits of the disease in Canada in 1844 and 1845.

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