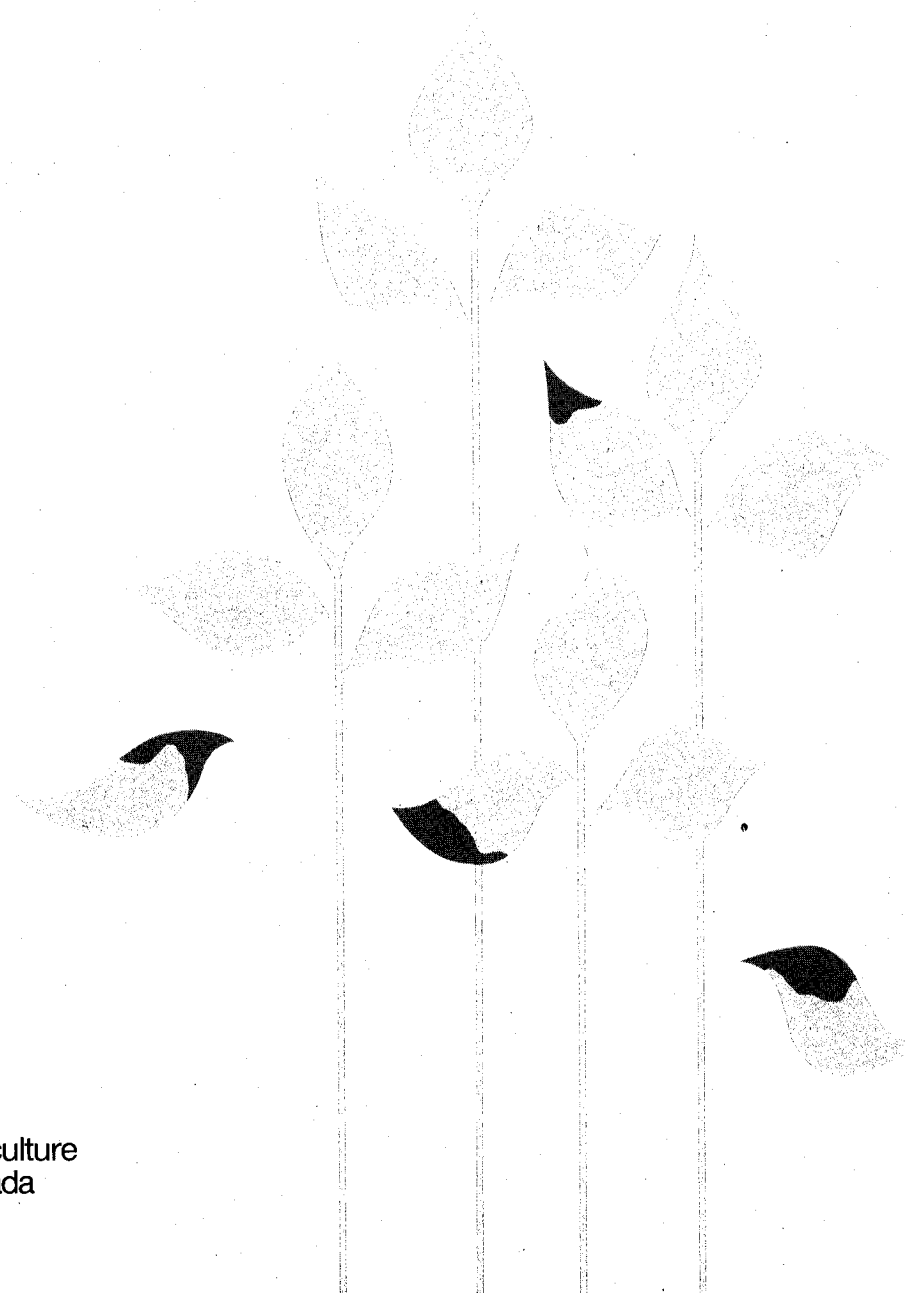


# Canadian Plant Disease Survey

# Inventaire des maladies des plantes au Canada

Vol. 72, No. 2, 1992

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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.

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*L'inventaire des maladies des plantes au Canada* est un périodique d'information sur la fréquence des maladies des plantes au Canada, leur gravité, et les pertes qu'elles occasionnent. La rédaction accepte d'autres communications originales notamment sur la mise au point de nouvelles méthodes d'enquête 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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## Corrigendum

First report of halo spot of barley caused by *Pseudoseptoria stomaticola* in Alberta

S.W. Slopek and T.J. Labun

Canadian Plant Disease Survey Volume 72:1, 5-8, 1992

The reference numbers in the body of the text are incorrect. The following is a list of the reference numbers in the text with the correct reference numbers listed in parentheses: 1(24), 2(31), 3(2), 4(8), 5(9), 6(21), 7(20), 8(16), 9(15), 10(14), 11(6), 12(5), 13(4), 14(22), 15(11), 16(30), 17(26), 18(27), 19(28), 20(29), 21(33), 22(31), 23(12), 24(7), 25(32), 26(25), 27(10), 28(13), 29(23), 30(17), 31(19), 32(1), 33(18), 34(3).

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# A survey of penicillium stem rot on greenhouse cucumbers in southwestern Ontario

W.R. Jarvis<sup>1</sup> and G.M. Ferguson<sup>2</sup>

In 1991, a field survey was conducted in 57 commercial greenhouses of southwestern Ontario as a preliminary action for the rational design of control measures for penicillium stem rot (*Penicillium oxalicum*) of greenhouse cucumbers. The disease was more prevalent in the Niagara area compared to the Leamington area (100% of crops affected versus 46%); it was more prevalent in medium-sized houses than large or small greenhouses; crops grown in rockwool (46%) were more affected than crops grown in soil or soil mix (21%); and the incidence of the disease was associated significantly with gummy stem blight (*Didymella bryoniae*) in spring crops, but not fall crops. No cultivar differences in susceptibility to penicillium stem rot were found. Isolates of *Penicillium oxalicum* (86% of those tested) were resistant to benomyl and two isolates were resistant to iprodione and cross-resistant to benomyl.

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En 1991, une évaluation préliminaire a été menée dans cinquante-sept serres commerciales au sud ouest de l'Ontario pour le modèle des mesures de lutte contre la pourriture de la tige du concombre de serre (*Penicillium oxalicum*). La maladie a été plus prédominante dans la région du Niagara que dans la région de Leamington (100 % des cultures ont été infectées versus 46 %); elle a été plus prédominante dans les serres de dimension moyenne que dans les serres de dimension plus grande ou plus petite; les cultures produites sur laine de roche ont été plus affectées que les cultures produites dans le sol ou un mélange de sol (21 %); et, l'incidence de la maladie a été associée de façon significative avec *Didymella bryoniae* dans les cultures de printemps mais pas dans les cultures d'automne. Aucune différence entre les cultivars n'a été trouvée concernant la susceptibilité à *Penicillium oxalicum*. Quatre-vingt-six pourcent des isolats de *Penicillium oxalicum* évalués ont été résistants au bénomyl et deux isolats ont été résistants à l'iprodione. Ces deux isolats ont eu, aussi, une résistance croisée pour le bénomyl.

## Introduction

The first report of penicillium stem rot of greenhouse cucumbers was from crops in the Leamington area of southwestern Ontario in 1988 (4). The causal organism was first designated as *Penicillium crustosum* Thom but subsequent work showed it to be *P. oxalicum* Currie & Thom (5). The disease has also been reported in England (7), the Netherlands and Scandinavia (8). Little is known of the factors determining the rise of *P. oxalicum* as a widespread and severe pathogen of both stems and fruit of greenhouse cucumbers (4,5,7,8). The survey reported here was undertaken to identify which, if any, of the current greenhouse cropping practices could be associated with incidence and severity of the disease.

## Materials and methods

Fifty-seven growers' properties in the Leamington area of southwestern Ontario were surveyed by scouts in May and June, 1991, to monitor the spring crop and to obtain information from the grower on cropping and disease history. In addition, eight growers (about 28% in the Niagara area) were visited, and information was obtained on 23 crops in all there. The growers were asked a number of questions designed to identify possible predisposing factors, as well as to obtain their estimates of the incidence and severity of the

disease. The scouts inspected and collected samples of the crop to confirm whether or not the grower had identified the disease correctly. The early symptoms of penicillium stem rot (*Penicillium oxalicum*), grey mould (*Botrytis cinerea* Pers.: Fr.), gummy stem blight (*Didymella bryoniae* (Auersw.) Rehm), and white mould (*Sclerotinia sclerotiorum* (Lib.) de Bary) can be superficially similar, but most experienced growers seemed to have identified penicillium stem rot correctly. Powdery mildew (*Sphaerotheca fuliginea* (Schlechtend.: Fr.) Pollacci) and pythium root rot (various *Pythium* spp.) were also observed at the sites.

Plant material with symptoms of *P. oxalicum*, *B. cinerea*, *D. bryoniae* or *S. sclerotiorum* was brought back to the laboratory, and spores, when present, were streaked directly on potato dextrose agar containing 0.1 µg/ml benomyl as Benlate 50 WP® or 0.2 µg/ml iprodione as Rovral 50 WP®. Growth of the pathogens at those concentrations of fungicides was taken as indicative of resistance (6). Fungi not sporulating when collected were induced to do so by incu-

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bating the material in a moist chamber for one or two days, and *S. sclerotiorum* was isolated directly from the copious mycelium.

All data were analyzed using chi-square tests. The factors tested for a significant association with penicillium stem rot were:

Region (Leamington vs. Niagara)  
Size of greenhouse range  
Growing medium (soil vs. rockwool)  
Association with other commonly occurring diseases  
Cultivar  
Type of cover (glass vs. plastic)  
Season (spring crop vs. fall crop)  
Sanitation measures  
Type of pesticide application equipment  
Number of fungicide applications  
Greenhouse environment control (manual vs. computerized)

In the case of soil groundbeds, differences in soil type were not taken into account since greenhouse soils have been considerably modified over many years by heavy amendments of organic materials. Significant associations were found in the first four factors; none of the other factors were significantly associated with the incidence of penicillium stem rot at  $P = 0.05$ .

## Results

### Factors significantly associated with penicillium stem rot

**Region.** Each of 23 crops surveyed in the Niagara area was affected by penicillium stem rot, whereas 38 of 83 crops (46%) in the Leamington area (Essex County) were affected, a difference significant at  $P = 0.01$ . All the growers in the Leamington area were surveyed, and although only 28% of the Niagara area growers were visited, the sampled sites appeared highly representative based on reports of other commercial growers in the area and of extension personnel familiar with the growers and their disease problems.

**Size of greenhouse range.** Greenhouse ranges were grouped into three size categories: (1) 0.25 - 0.50 ha (small), (2) 0.60 - 1.0 ha (medium) and (3) >1.0 ha (large). There was significantly more ( $P = 0.05$ ) penicillium stem rot in ranges of medium size than in smaller or larger ranges, but only for the spring crop. The disease was present in 18 (70%) medium sized greenhouses, in five (38%) large, and in five (33%) small greenhouses.

**Growing medium.** Of the two principal growing media, soil and rockwool, a highly significant association ( $P = 0.001$ ) was found for rockwool (Tables 1 and 2). In the spring crop, a mean of 44% of crops grown in rockwool had stem rot, in contrast to 17.4% of crops grown in soil. Similarly, in the fall, 48.2% of the crops grown in rockwool were diseased, against 14.3% grown in soil.

**Association with other commonly occurring diseases.** Penicillium stem rot was significantly associated ( $P = 0.001$ ) with gummy stem blight in all of the spring crops but in only 35% of the fall crops. There was no association of penicillium stem rot with incidence of pythium root rot, grey mould and powdery mildew in either the spring or fall crop.

### Factors not significantly associated with penicillium stem rot

**Season.** The incidence of penicillium stem rot in spring crops was compared with that in fall crops. Thirty-three percent and 45% of spring and fall crops, respectively, had the disease (Tables 1 and 2).

**Type of cover.** Only glass and plastic (double polyethylene) houses were encountered; there were no glasshouses with a plastic liner. Penicillium stem rot was present in 43% and 49% of the glass and plastic houses respectively.

**Cultivar.** Only the two most commonly grown cultivars, Corona and Jessica, were considered for analysis (Tables 1 and 2). Thirty-six percent and 37% of Corona and Jessica crops, respectively, had penicillium stem rot.

Table 1. Incidence of penicillium stem rot in spring greenhouse cucumber crops by substrate and cultivar.

Substrate	Cultivar					
	Corona	Jessica	Bronco	Sandra	Dugan	Ventura
Rockwool	15/33 <sup>1</sup>	8/19	3/6	4/8	0/0	0/2
Oasis	0/1	1/4	0/0	1/3	0/0	0/1
Soil	5/25	2/12	1/5	0/3	0/0	0/1
Soil/Peat	0/1	1/1	0/0	1/2	0/0	0/0
Peat	0/1	0/0	0/0	0/0	0/0	0/0

<sup>1</sup> Affected/total crops.

**Sanitation measures.** Sanitation procedures such as sterilization of rockwool bags, changing of plastic ground cover, steaming vs. fumigation of soil, site location (upwind, downwind) and distance of trash dumping, from the production greenhouse, were factors considered. The disease was present in 30% of those houses where growers used the municipal dump site, and 50% of those where dumping of crop debris was done in the environs of the greenhouse. No contributing factor could be identified.

Of all the growers surveyed, 65% sterilized their rockwool bags, 70% sterilized irrigation drippers, and 66% changed their plastic ground cover; all equally had penicillium stem rot in their crops. Steaming is usually practised for rockwool and fumigation with methyl bromide for soil. Thirteen percent of the crops grown in a steamed soil, and 20% of those grown in fumigated soil had penicillium stem rot.

A straw mulch, steam-sterilized or not, did not affect the disease incidence when it was applied to crops in soil.

**Type of pesticide application equipment.** Low volume fogging machines were compared with high volume sprayers. Penicillium stem rot was equally present in houses using either one of these two types of equipment, the disease was in 59% of houses with low-volume and 52% of those using high-volume equipment.

**Number of fungicide applications.** Growers were separated into four categories according to the number of fungicide

applications made: (1) <10, (2) 11-20, (3) 21-30, and (4) >30. Penicillium stem rot was present in 17 of 25 crops (68%) of growers who had applied less than ten fungicide sprays, seven of eight crops (88%) receiving between 10-20 sprays, two of two (100%) receiving 21-30 sprays, and in seven of ten crops (70%) with >30 sprays.

**Environmental control.** Automation of environmental control appeared to have had no effect on penicillium stem rot incidence, the disease being present in 50% of the houses that had computer-controlled environments, and 48% that had manual control.

#### Resistance of pathogens to fungicides

Results of *in vitro* assays of isolates of *P. oxalicum*, *Botrytis cinerea* and *Didymella bryoniae* are summarized in Table 3.

Cross resistance to both benomyl and iprodione was found in two isolates of *P. oxalicum* and one of *B. cinerea*.

#### Discussion

Four main factors affecting the incidence and severity of penicillium stem rot of cucumbers were identified. There were significantly more crops affected, and by the growers' estimates, more severely affected, in the Niagara area than in the Leamington area; more crops in medium-sized houses (0.6 - 1.0 ha) were affected than in smaller or larger houses; and crops in rockwool were more likely to be affected and more severely so, than crops in soil; and penicillium

Table 2. Incidence of penicillium stem rot in fall greenhouse cucumber crops by substrate and cultivar.

Substrate	Cultivar					
	Corona	Jessica	Bronco	Sandra	Dugan	Ventura
Rockwool	8/16 <sup>1</sup>	10/21	4/8	1/2	4/8	0/1
Oasis	0/0	0/1	0/0	0/0	0/0	0/0
Soil	1/3	0/2	0/0	0/1	0/0	0/1
Soil/Peat	0/0	1/2	0/0	1/2	0/0	0/0
Peat	0/0	0/0	0/0	0/0	0/0	0/0

1 Affected/total crops.

Table 3. *In vitro* resistance of fungal isolates to benomyl (1 µg/ml) and iprodione (2 µg/ml).

	Benomyl		Iprodione	
	Resistant	Susceptible	Resistant	Susceptible
<i>Penicillium oxalicum</i>	19 (86%)	3 (14%)	2 (9%)	20 (91%)
<i>Didymella bryoniae</i>	32 (73%)	12 (27%)	0	43 (100%)
<i>Botrytis cinerea</i>	10 (42%)	13 (58%)	1 (4%)	22 (96%)

stem rot was significantly associated with gummy stem blight in the spring crop, but not in the fall crop.

**Region.** Growers in the Niagara and Leamington areas broadly follow Provincial recommendations (1,2). We are unable to suggest why the disease was more prevalent and severe in the Niagara area, with its rather more diffuse distribution of greenhouses, than in the dense concentration of greenhouses in the Leamington area. There may have been climatic differences between the two areas, which are about 400 km apart, but we have not yet been able to identify them.

**Size of greenhouse range.** The size of the greenhouse may well affect its microclimate, but this factor is likely to be over-ridden by variations in temperature control, humidity control, ventilation, air movement, irrigation systems, ground cover and perhaps several other imponderable factors. Although there may be slight variations in plant density and pruning systems, they are not likely to be affected by house size.

**Growing medium.** Crops in rockwool are more likely to be affected, and more severely so, than crops in soil, which strongly suggests that stress may well be a predisposing factor. In general, crops in rockwool are more precisely controlled by nutrition and yield 10 - 30% more than crops in soil (W.A. Straver, personal communication). Alternatively or additionally there may be nutrients, for example, silicon, available in most soils, but not in rockwool, that might have conferred resistance. We are therefore unable to explain this effect.

#### Association with other commonly occurring diseases.

That penicillium stem rot is associated with gummy stem blight in the spring crop suggests similar predisposing conditions; the temperature and humidity conditions in the spring may be similar for the two diseases. Van Steekelenburg and van de Vooren (9) associated the presence of gummy stem blight in the spring crop with low night temperatures (12 - 16°C), at a constant day temperature of 23°C in a preinoculation period. In the fall crop, there were no significant differences between temperature regimes. Plants grown under drier conditions were less affected by gummy stem blight, but grey mould and powdery mildew were more evident under drier than humid conditions.

**Sanitation.** No major breach in sanitation procedures could be found that might have explained the distribution and severity of the disease. The source of inoculum remains obscure.

The finding of resistance to the fungicides benomyl and iprodione in *B. cinerea* and *D. bryoniae* underlines the necessity

to rely on environmental control of grey mould and gummy stem blight rather than fungicides. The high incidence (86%) of isolates of *P. oxalicum* resistant to benomyl may well mean that penicillium stem rot is an iatrogenic disease (3), exacerbated by benomyl.

It is noteworthy that O'Neill *et al.* (7) also found United Kingdom isolates of *P. oxalicum* to be resistant to 2 µg/ml, as well as to 20 µg/ml benomyl.

#### Acknowledgements

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# Screening of sainfoin cultivars and lines for yield, winter hardiness and resistance to fusarium crown and root rot in east central Alberta

S.F. Hwang<sup>1</sup>, B.P. Berg<sup>2</sup>, R.J. Howard<sup>3</sup> and D.W. McAndrew<sup>4</sup>

A field trial was conducted to evaluate ten cultivars and six breeding lines of sainfoin for dry matter yields, winter survival and resistance to fusarium crown and root rot. Significant differences were observed in dry matter weights of each cut taken in 1989 and 1990. Winter survival of all cultivars and lines was less than 30% three years after seeding, and all suffered from fusarium crown and root rot. With the exception of Nova having the lowest disease severity rating, no significant differences in crown and root rot severity occurred among the cultivars and lines evaluated.

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Un essai au champs a été mené pour évaluer dix cultivars et six lignées généalogiques de sainfoin pour leurs rendements en matière sèche, leur résistance hivernale et leur résistance à la pourriture fusarienne de la couronne et de la racine. Des différences significatives ont été observées dans les poids de la matière sèche de chaque coupe prise en 1989 et en 1990. La survie hivernale de tous les cultivars et lignées a été 30 % moindre trois années après l'ensemencement, et tous ont souffert de pourriture fusarienne de la couronne et de la racine. A part une exception ayant eu le taux de sévérité pathologique le plus bas (ie, Nova), aucune différence significative de sévérité pour la pourriture de la couronne et de la racine ne s'est produite parmi les cultivars et les lignées évaluées.

## Introduction

Sainfoin (*Onobrychis viciifolia* Scop.) is a perennial legume that has been cultivated as a forage crop in Europe and Asia for several centuries (2,4). Despite having considerable merit as a forage crop, sainfoin is not widely grown in Canada (4). Crown and root rot was identified as one of the most important factors affecting the longevity of sainfoin stands in the United States (9). A survey of sainfoin fields in 1983 revealed that this disease also was prevalent and destructive in southern Alberta (6). Crown and root rot is an important disease of other forage legumes including alfalfa (3,5,7), clover (3) and birdsfoot trefoil (1).

Crown and root deterioration in forage legumes can be caused by biotic and abiotic factors. Previous studies have demonstrated that bacterial and fungal species are major components of the disease complex (1,3,5,6,7,10,13,14). *Fusarium solani* (Mart.) Sacc. and species of *Pseudomonas* and *Erwinia* were found to be the most important crown and root rot pathogens of sainfoin in Montana (13). In Alberta, *Pseudomonas fluorescens* Migula, *P. syringae* van Hall, *Erwinia carotovora* subsp. *carotovora* Bergey et al., and *Enterobacter agglomerans* (Beij.) Ewing & Fife were shown to be part of the crown and root rot complex on sainfoin (6). The high frequency of isolation of *F. solani* from diseased roots suggested that sainfoin is a host for this pathogen. Other *Fusarium* spp. also have been implicated in crown and root rot of alfalfa (3,5,7), clover (3) and birdsfoot trefoil (1) in Alberta.

Crown and root rot infection can affect the habit of plant growth and in temperate regions the level of tolerance to low temperatures. Infected buds often become necrotic and, in both alfalfa and sainfoin plants, may become asymmetrical (13,14). Alfalfa plants affected by crown and root rot disease may be severely injured by low temperature (5,8,12). It has been suggested that infected roots accumulate low levels of reserve foods which affects the maximum level of plant tolerance to low temperature (8,11). The development of more resistant, winterhardy cultivars may reduce the adverse economic impact of crown and root rot in alfalfa (8,11) and sainfoin (9). This study was undertaken to evaluate cultivars and lines of sainfoin for their disease resistance and to compare their yield and winter survival under field conditions.

## Materials and methods

A field experiment was established near Bonnyville, Alberta in the spring of 1988. A preemergence herbicide, Eptam 8-E (EPTC 80% EC) at a rate of 4.5 L/ha, and 90 kg/ha of monoammonium phosphate (11-51-0), 20 kg/ha of potash

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(0-0-60) and 19 kg/ha of elemental sulphur (0-0-0-90) were incorporated into the soil prior to planting. Sainfoin seeds of 16 cultivars and lines were surface-sterilized in 70% ethanol for two min, followed by two min in 0.6% sodium hypochlorite, then rinsed three times in sterilized distilled water. The surface-disinfested seeds were sown in vermiculite in root-trainers (Spencer-LaMaire Industries, Edmonton, Alberta) and grown in the greenhouse at  $20 \pm 5^\circ\text{C}$  under 12 to 17 h natural light. After emergence, seedlings were thinned to one per root-trainer cell. One-month-old seedlings of each cultivar and line were transplanted to the field with six plants per meter in a randomized complete block design with four replications. There were four 1-m-rows spaced 20 cm apart in each plot.

Three single-spored isolates of *F. solani* (S-25, S-19, and S-20) were obtained from symptomatic roots of sainfoin seedlings and maintained on potato dextrose agar slants at  $5^\circ\text{C}$ . During the summer of 1989, each isolate was grown on a sterile oat-rye kernel medium (1:1, v/v), mixed, and sprinkled

on the plot at a rate of 25 g/plot. Each plot was cut twice each year at the 20% bloom stage and the sainfoin clippings were oven dried at  $70^\circ\text{C}$  for 24 h to determine dry matter yield per plot. Prior to the first cut during the springs of 1989, 1990 and 1991, winter survival was determined by counting the number of plants with new shoot growth in each plot. The percentage of plants surviving out of the total planted was calculated. In 1991, the surviving plants from each plot were dug up and the roots were bisected longitudinally to visually assess the severity of crown and root rot. Disease severity ratings were determined using the scale: 0 = clean, no disease; 1 = slight, 1-20% of the crown and root discoloured; 2 = moderate, 21-50% of the crown and root discoloured; 3 = severe, 51-100% of the crown and root discoloured; and 4 = dead.

An analysis of variance and Duncan's multiple range test were used to determine whether differences in data on dry matter yield, percent winter survival and disease severity of crown and root rot among cultivars and lines were statistically significant.

Table 1. Average yields from sainfoin plants clipped twice in 1989 and in 1990.

Cultivar or line	Dry weight (g)/plant <sup>1</sup>					
	1989			1990		
	1st cut	2nd cut	Average	1st cut	2nd cut	Average
Eski	41.5 ab <sup>2</sup>	48.8 ab	90.3 abc	12.4 bc	10.1 b	22.5 de
Fakir	15.7 fg	22.6 b	38.3 f	21.5 ab	10.1 b	31.6 c
Hampshire Common	27.1 bcdef	29.7 b	56.8 def	13.2 bc	10.1 b	23.3 cde
Krasnodar	32.2 abcde	59.8 ab	92.0 ab	16.2 bc	8.9 b	25.1 cde
Melrose	40.5 abc	48.4 ab	88.9 abc	11.1 bc	8.9 b	20.0 e
Nova	42.9 a	46.8 ab	89.7 abc	35.7 a	23.2 a	58.9 a
Octo	24.6 defg	32.2 b	56.8 def	15.9 bc	11.7 b	27.6 cd
Remont	25.8 cdefg	48.0 ab	73.8 bcde	7.9 bc	9.3 b	17.2 e
Sparta	36.1 abcde	58.9 ab	95.0 ab	10.8 bc	11.6 b	22.4 de
Viva	26.6 bcdef	53.3 ab	79.9 bcd	3.1 c	2.3 b	5.4 f
L2082 (Russian selection)	39.0 abcd	48.3 ab	87.3 abc	13.6 bc	5.2 b	18.8 e
L2086 (Polish selection)	24.6 defg	41.8 defg	66.4 cde	21.4 ab	21.8 a	43.2 b
L2092 (Russian selection)	31.3 abcde	43.1 ab	74.4 bcde	11.3 bc	7.4 b	18.7 e
L2110 (American selection)	22.8 efg	31.5 b	54.3 ef	12.6 bc	5.2 b	17.8 e
L2209 (Romanian selection)	36.7 abcde	74.0 a	110.7 a	12.1 bc	11.3 b	23.4 cde
L2334 (Great Britian selection)	11.3 g	28.0 b	39.3 f	9.5 bc	12.1 b	21.6 de

1 Dry weight per plant was estimated by dividing the total dry weight of the clippings harvested from each plot by the number of plants that survived in each plot.

2 Values in a column followed by the same letter are not significantly different using Duncan's multiple range test ( $P = 0.05$ ).

## Results and discussion

Significant differences ( $P = 0.05$ ) were observed in dry matter weights between cultivars and lines for each cut of sainfoin taken in 1989 and in 1990, (Table 1). The average dry matter weight for the first cut of cultivar Fakir was lower in 1989 than the weight for the first cut in 1990. For all of the other cultivars and lines the first and second cut dry weights were higher in 1989 than in 1990. The sum of the average dry matter weights for the two cuts in 1989 for cultivars Eski, Krasnodar, Melrose, Nova and Sparta, and breeding lines L2082 and L2209 varied between 87.3 to 110.7 g/plant and were significantly greater than the yield values for the remaining cultivars Fakir, Hampshire Common and Octo, and breeding lines L2110 and L2334 (38.3 to 56.8 g/plant). In 1990, the sum of the average dry matter weights for Nova (58.9 g/plant) and L2086 (43.2 g/plant) were significantly greater than all of the remaining cultivars and lines (5.4 to 31.6 g/plant).

The 1989 winter survival values among the six lines and ten cultivars tested were not significantly different, except for Fakir and L2334 which had significantly lower survival (69 to 71%) (Fig. 1). In 1990, Eski, Krasnodar, Melrose, Nova, L2082, and L2209 had significantly greater winter survival (71 to 79%) than did Viva, Fakir, Hampshire Common and L2334 (15 to 29%). The remaining six cultivars and lines were intermediate (43 to 68%). The 1991 values for survival among all of the cultivars and lines tested were less than 30%, but Nova had the highest winter survival value (28%), while the values for L2082, L2092, Sparta and Melrose varied between 12 and 16%.

All cultivars suffered from fusarium crown and root rot. With the exception of Nova having the lowest disease severity rating, no significant differences in disease severity ratings occurred among the cultivars and lines evaluated (Fig. 2).

Results of this study have demonstrated that the experimental cultivars and breeding lines evaluated herein, as well as the registered cultivars Melrose and Nova, have commercially unacceptable susceptibilities to crown and root rot. The mean disease severities in all cases were high. Winter survival of all genotypes evaluated was less than 30% three years after seeding. In long-term forage systems, stand persistence is an important attribute in cultivar selection. Winter survival of all sainfoin cultivars and lines evaluated in this study was quite high the first year after seeding, but thereafter the severity of winterkill increased rapidly so that by the third year after seeding all cultivars were almost completely winterkilled. Although Nova had the least disease severity and greatest survival, the yield was below the level of economic benefit. To realize the full potential of sainfoin as a forage crop in western Canada, further research should be directed toward the development of cultivars that are cold hardy and disease resistant.

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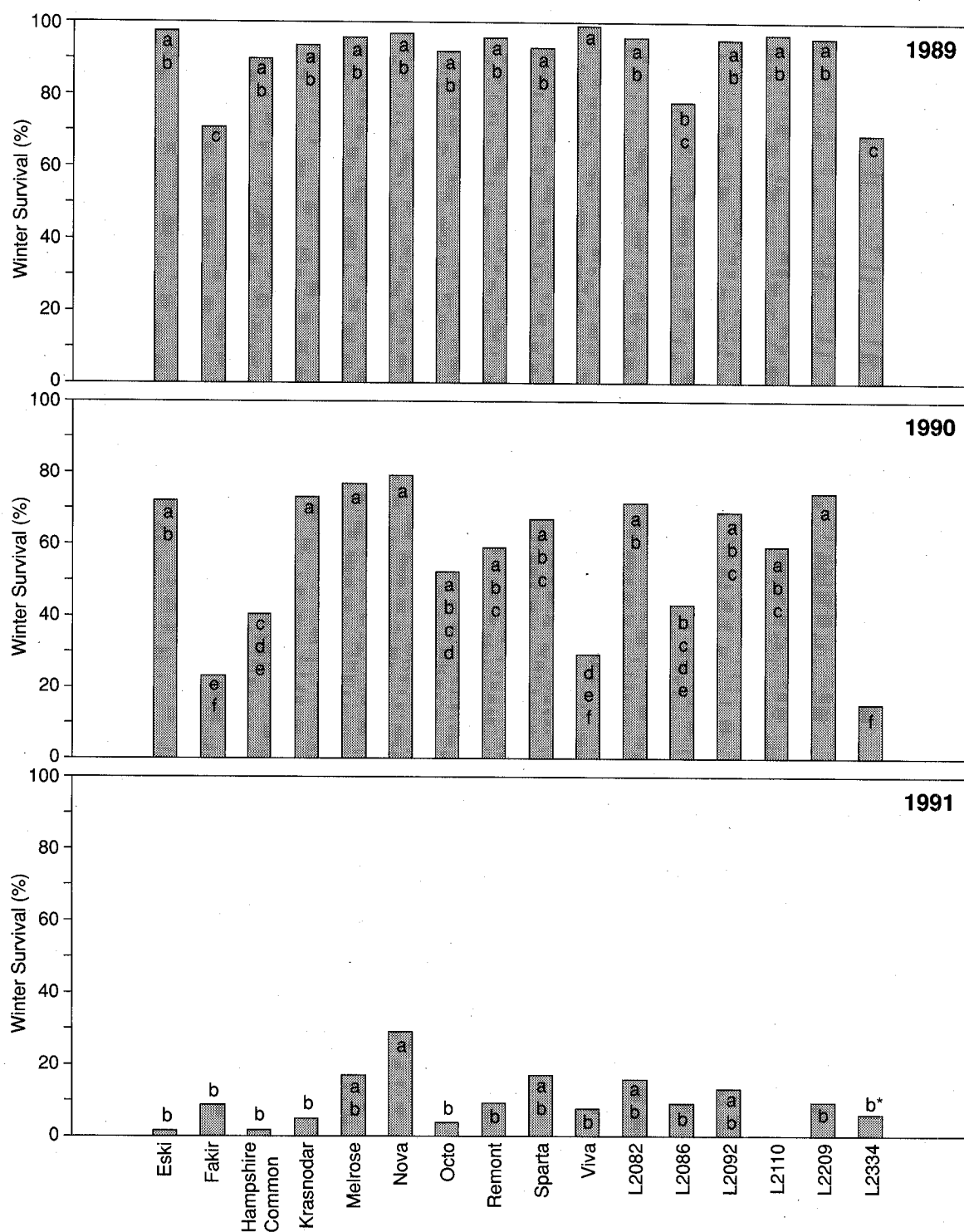


Fig. 1. Percent winter survival of ten cultivars and six breeding lines of sainfoin from 1989 to 1991. \*Means within each year followed by the same letter are not significantly different using Duncan's multiple range test.

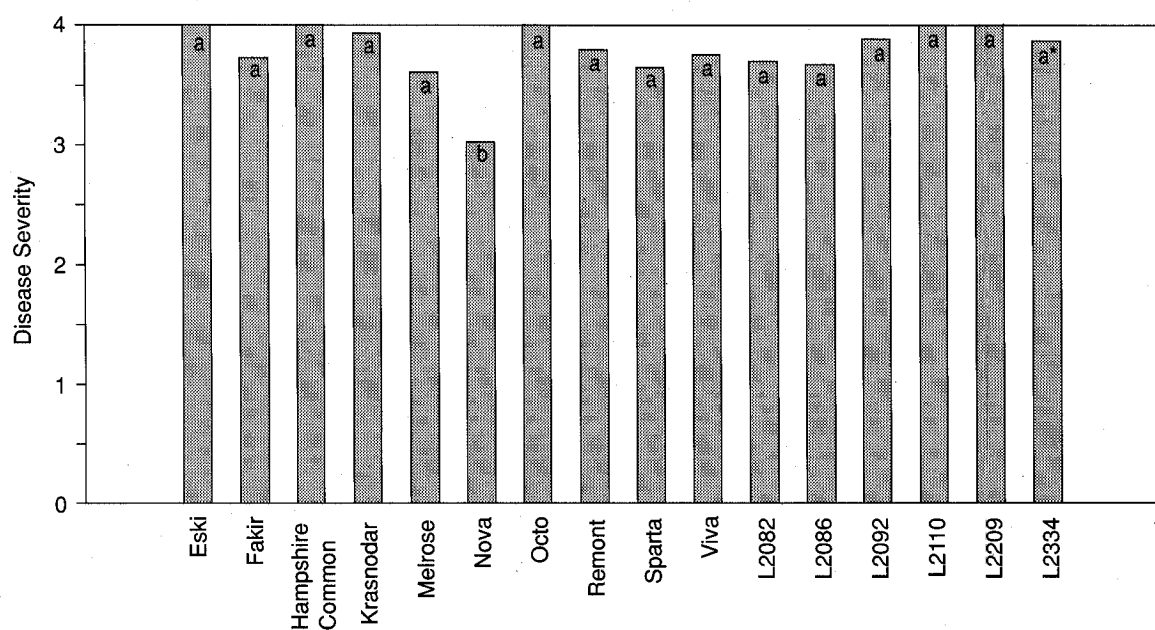


Fig. 2. Disease severity ratings of ten cultivars and six breeding lines of sainfoin to fusarium crown and root rot on a scale of 0-4, where 0 = clean, no disease; 1 = slight, 1-20% of the crown and root discoloured; 2 = moderate, 21-50% of the crown and root discoloured; 3 = severe, 51-100% of the crown and root discoloured; and 4 = dead. \*Means followed by the same letter are not significantly different using Duncan's multiple range test.



# Incidence of the tobacco vein necrotic strain of potato virus Y (PVY<sup>N</sup>) in Canada in 1990 and 1991 and scientific basis for eradication of the disease

R.P. Singh<sup>1</sup>

Eradication is defined as the application of cultural and biological control measures to cause any significant reduction in inoculum and the demonstration of the absence of pathogen over a period of time, from a representative sample, by using the most sensitive detection procedure available to date. The basis of the Canadian eradication initiative lies in the significant difference between occurrence of PVY<sup>N</sup>, the virus spread and aphid flight in European countries in the 1950s, and those of 1990-91 occurring in Canada when the PVY<sup>N</sup> outbreak took place. The prospect of PVY<sup>N</sup> eradication in Canada is based on the very low virus incidence, low aphid pressure, drastic measures taken to reduce inoculum, the massive number of leaf samples tested, and the application of highly sensitive detection procedures. The progress of Canadian eradication measures compares well with the PVY<sup>N</sup> elimination carried out in New Zealand in 1988-89.

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L'éradication est définie comme étant l'application de pratiques culturales et de moyens de lutte biologique qui causent toute réduction significative de l'inoculum et révèlent l'absence de pathogènes durant une période de temps, à partir d'un échantillon représentatif tout en utilisant la méthode de détection la plus sensible disponible présentement. La méthode canadienne en éradication repose sur les différences significatives entre les occurrences du PVY<sup>N</sup>, l'étendu du virus et les vols de pucerons dans les pays européens durant les années 1950, et les vols de pucerons au Canada en 1990-1991 lors des premières manifestations du PVY<sup>N</sup>. Les prévisions d'éradication du PVY<sup>N</sup> au Canada sont basées sur une très faible incidence du virus, un faible nombre de pucerons, des mesures draconiennes pour réduire l'inoculum, un grand nombre d'échantillons de feuilles examinées, et l'application de procédures de détection extrêmement sensibles. Les progrès qu'ont connus les mesures d'éradication au Canada sont comparables aux progrès qui ont été effectués en Nouvelle-Zélande durant les années 1988-89, en ce qui touche l'élimination du PVY<sup>N</sup>.

## Introduction

Eradication as originally defined by Whetzel (1929) means the complete elimination or destruction of a pathogen after it is established in a given area. This type of definition implies absoluteness and is not adequate for a holistic approach to crop protection and crop production (Apple, 1977). Experience has shown that absolute control of disease is economically impractical. On the other hand, the National Academy of Sciences (NAS) United States of America (USA) publication (1968) has used the term eradication to connote any significant reduction in inoculum with the application of cultural and biological control measures. The definition adopted by the 1991 Bi-National PVY<sup>N</sup> meeting based on the North American Plant Protection Organization (NAPPO) recommendation states that eradication of a plant pest has occurred when processes have been applied that lead to the detection of zero levels of the pest in plants; and plant products known to be affected and produced in a defined area (Anon., 1991b). In this paper, the above NAPPO definition is implied for the eradication of PVY<sup>N</sup>.

## Background and history of the eradication proposal. A

strain of potato virus Y which induces systemic vein necrosis of tobacco leaves, known as PVY<sup>N</sup> (de Bokx and Huttinga, 1981) was discovered in seed potatoes of eastern Canada in 1990 (Anon., 1991a, Coffin *et al.*, 1991). A preliminary survey for the PVY<sup>N</sup> strain during the summer of 1990 led to the finding of the virus in three fields each of New Brunswick (NB) and Prince Edward Island (PEI) (Table 1). The seed potatoes planted in NB were traced back to seed sources in PEI, thus indicating only one source of PVY<sup>N</sup>. This survey was followed by an extensive leaf test (Florida-grown potato seedlots) and tuber-sprout test during the winter months (Table 1). As a result, in the 1990 crop a total of four cases of PVY<sup>N</sup> were found in NB and 31 in PEI. This uneven distribution of PVY<sup>N</sup> from a large number of seedlots and their traceback to one seed source indicated that eradication should be possible, if the main seed sources were fully tested and discarded.

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However, in spite of this type of data, a number of scientists and producers were of the opinion that eradication would not be successful. The main reason presented by scientists and producers alike was that seed potatoes from PEI have gone to several provinces of Canada as well as to several seed-producing States of the USA in preceding years. Thus, PVYN should be well established all over North America.

A preliminary review of the survey data (Table 1) suggested otherwise. A preliminary proposal was advanced for the eradication of PVYN from Canada (Singh, 1991). After considerable discussion between scientists from the United States and Canada, along with government and industry personnel, an eradication package was developed mainly for PEI. A progress report on the eradication effort in PEI was made to the annual meeting of the Potato Association of America in Spokane, USA (Coffin *et al.*, 1991).

During the summer of 1991, the opinions of internationally recognized PVYN specialists were sought. Potato and tobacco virologists from Germany, Peru, the United Kingdom and the United States, held two days of discussions with virologists and entomologists from Canada. The objective was to assess the feasibility of eradication of PVYN from Canada. The outcome of this discussion was that eradication was a possibility, however, for the foreseeable future management of seed potato production within affected areas would be required to avoid infection by the virus (Anon., 1991c). Since then, there have been many questions from virologists, potato inspectors and potato producers regarding PVYN eradication. From these discussions it is apparent that there is not a good understanding of the situation in Canada when compared to countries where eradication of PVYN was not successful.

The eradication proposal (Singh, 1991) identified a limited number of infection sources, limited virus spread and most of the spread through seed movement of the potato cultivar Atlantic. In this paper, an attempt is made to demonstrate that a contrast between the situations of Canada and Europe existed at the time of the PVYN outbreak, thus the Canadian eradication proposal is scientifically based, and it compares favorably with an outbreak and control of PVYN in New Zealand (Fletcher, 1989).

**Occurrence of PVYN in European countries.** The diagnosis of PVYN in different countries has been based on the distinctive property of this strain to induce severe veinal necrosis on healthy tobacco plants 7 to 15 days after inoculation. The symptoms in tobacco at first are confined to the smaller veinlets (Fig. 1A), but later the necrosis spreads to petiole (Fig. 1B), and stems (Fig. 1C) with collapse of lower leaves (Fig. 1C,D). However, the necrotic symptoms have to be viewed in absence of other viruses infecting tobacco.

PVYN first appeared in European seed potato growing areas in the 1950s causing a severe epidemic with high losses (Weidemann, 1988). It was supposed to have originated in

South America (Smith and Dennis, 1940; Nobrega and Silberschmidt, 1944; Bawden and Kassanis, 1951) from where it was introduced to Europe. In Europe, it was first detected at the Commonwealth Potato Collection from a naturally infected potato plant collected in South America and was possibly identical with Nobrega and Silberschmidt's isolate (Bawden and Kassanis, 1951). The rapid spread of PVYN in Europe was noted from its reports from several countries within a short period. For example, PVYN was identified from Bulgaria (Kavachevsky, 1950); Switzerland (Bovey, 1955); The Netherlands in 1957 (de Bokx, 1961); England (Richardson, 1958); Germany (Schmelzer and Klinkowski, 1958); Hungary (Szirmai, 1958); Italy (Marceli, 1960); Scotland (Todd, 1961); and Poland (Jankowski and Florczak, 1962). PVYN became economically important first in Germany, where certain commercial cultivars became almost wholly infected (Weidemann, 1988), and in 1958-1960 potato crops suffered seriously due to PVYN in The Netherlands (de Bokx, 1964).

**High incidence of virus in the 1950s.** From some studies published during the 1950s, it appears that the European tobacco crop was severely infected with PVYN. For example, in one study dealing with the economics of tobacco production, it was stated that the tobacco quality started deteriorating when PVYN incidence reached 10-20%, and at 60-70% harvesting of tobacco became unprofitable. In some areas with optimum climate and soil conditions a high incidence of PVYN (100%) was observed in 1957 (Seehofer *et al.*, 1958). During a tour of the tobacco-growing areas of Baden in 1958, a clear correlation was observed between the development of viral symptoms in tobacco and the proximity of diseased potato fields (Steiner, 1959).

High incidence of PVYN in Europe was not limited to the tobacco crop only. In a study dealing with PVYN incidence in potato in Switzerland, the virus incidence was 40-100%, with the seed potatoes received from Northern Germany, The Netherlands, Poland, Austria and Denmark (Keller and Munster, 1961).

Research work from Brunswick, Germany during the 1950s showed that the extent and rapidity of PVYN infection in the tobacco crop depended on the annual situation of aphid vectors. Commencement of aphid activity before June resulted in mass infection by PVYN at an early stage of tobacco growth (Volk, 1960a). In 1954, 1957, and 1959 there were early and high aphid activity resulting in over 50% infection by mid July. In 1955, flights were late and only a few aphids were present, and 50% infection was delayed until early August. *Myzus persicae* was found in the greatest numbers throughout most of the 1950s, except the year 1956, *M. persicae* was the main vector in the fields (Volk, 1960b).

Detection procedures used in the 1950s. PVYN was detected serologically using polyclonal antisera and precipitin tests of eye-sprouts or inoculating extracts to A6 (*S. demissum* x *S. tuberosum* 'Aquila') test plant (Arenz and Hunnis, 1961). In other laboratories, dormant tuber extracts were used for the inoculation of A6 or *Solanum demissum* Y (SdY) test plants (de Bokx, 1961).

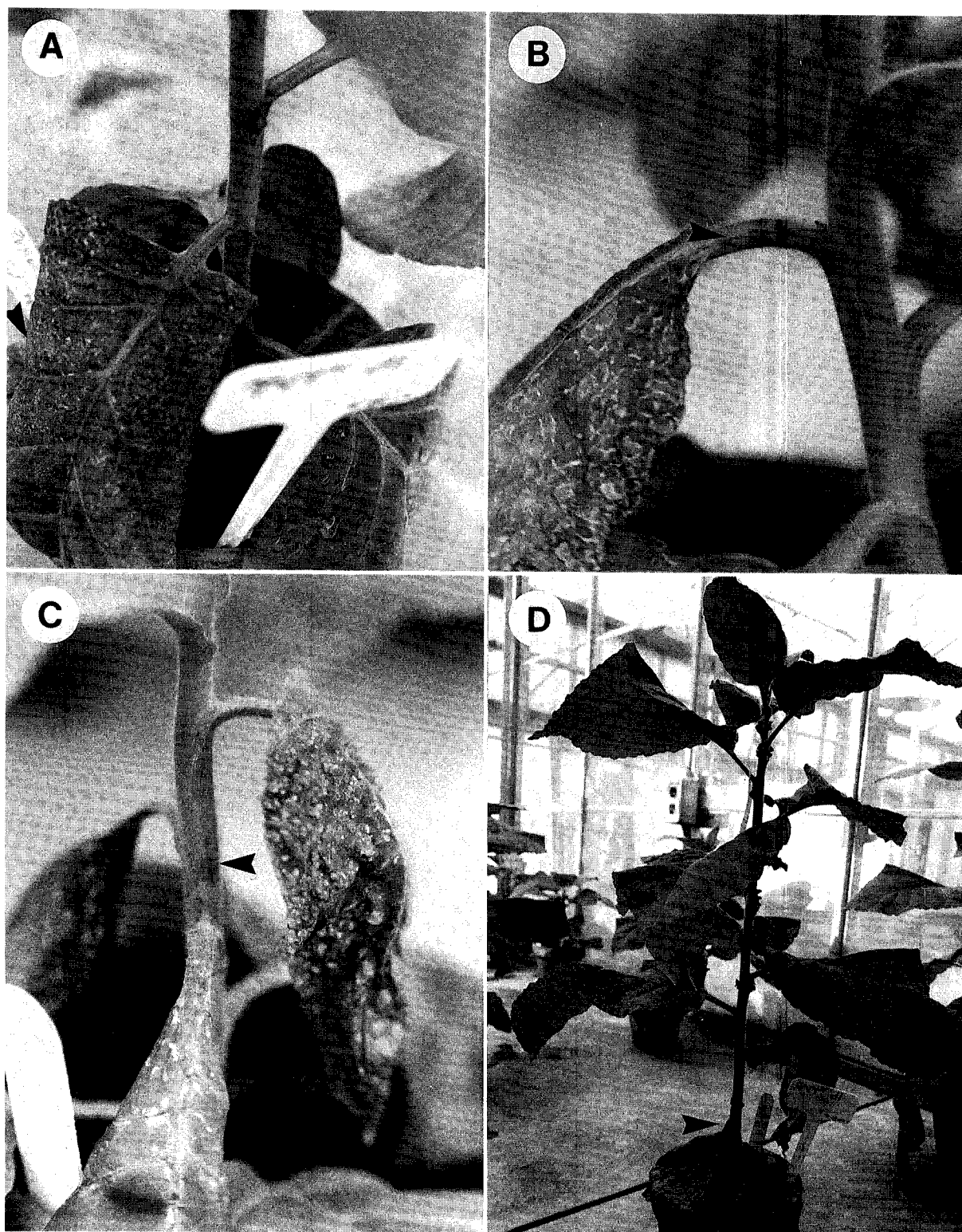


Fig. 1. Samsun tobacco showing necrosis of veinlets (A), petiole (B), stem (C), and collapse of leaves (C, D), infected with PVYN. Arrow heads indicate the location of symptoms.

### Situation in Canada at the time of PVY<sup>N</sup> discovery

**New Brunswick and Prince Edward Island.** The survey results for 1990-91 carried out in Agriculture Canada Seed Certification and Plant Quarantine laboratories are presented in Table 1. It showed that 1.57% of the seedlots from PEI and 0.43% from NB contained PVY<sup>N</sup>. Detailed analysis of postharvest tuber testing from PEI provided further insight into the distribution of PVY<sup>N</sup>. The PVY<sup>N</sup> infection was pre-

dominantly in the cultivar Atlantic (Table 2). Infection of PVY<sup>N</sup> in highly PVY<sup>O</sup> susceptible cultivars Russet Burbank, Shepody and Superior was not detected without infected Atlantic fields being present on the same farm and even then the incidence was limited to one or two fields.

As far as the tobacco crop is concerned, there has not been any record of necrotic symptoms in PEI. In 1991, tobacco plants were planted as bait-plants in several potato fields.

Table 1. Detection of PVY<sup>N</sup> in potatoes during summer and winter of 1990-91 in the Atlantic Provinces, Canada<sup>1</sup>

Type of Survey	Sample Size	New Brunswick	Nova Scotia <sup>2</sup>	Prince Edward Island
Field-Summer	100 leaves	3/163	0/25	3/299
Winter-Florida Test	100 leaves	0/512	NT <sup>3</sup>	0/438
Winter-Tuber Test	100 leaves	1/239	NT	28/1237
	Total	4/914 (0.43%)	0/25 (0%)	31/1974 (1.57%)

1 Data from seed certification laboratories, Food Production and Inspection Branch, Agriculture Canada.

2 Tested at Research Branch, Research Station, Fredericton, New Brunswick.

3 Not tested.

Table 2. Incidence of PVY<sup>N</sup> in Prince Edward Island potato fields in 1990-91 winter test<sup>1</sup>

Field #	Potato Cultivars with PVY <sup>N</sup> Positives			
	Atlantic	Russet Burbank	Shepody	Superior
Farm 1	1/1 <sup>2</sup>	0/6	NT <sup>3</sup>	0/1
2	2/4	NT	NT	NT
3	1/1	NT	NT	NT
4	5/11	3/3	NT	1/2
5	2/3	0/3	1/4	0/2
6	1/1	0/2	0/2	0/1
7	1/2	0/1	0/2	0/1
8	1/1	0/2	NT	0/1
9	1/1	0/4	NT	0/1
10	1/2	0/2	NT	0/1
11	1/1	0/4	0/2	0/5
12	1/1	1/4	NT	NT
13	2/2	0/1	0/1	0/2
14	1/5	NT	NT	NT
Total	21/36	4/32	1/11	1/17

1 Data was obtained from Seed Certification Laboratory, Charlottetown, PEI, Food Production and Inspection Branch, Agriculture Canada. One additional PVY<sup>N</sup> positive was in cultivar Bellisle at a different farm, where no other cultivars were tested.

2 Number of PVY<sup>N</sup> positives/number of fields tested on each farm.

3 Not tested.

No PVY<sup>N</sup> infection was detected in any of them (unpublished observation, R. Coffin, PEI Department of Agriculture). Although aphids can be detected in PEI prior to June, potato colonizing aphids are not observed until July and even then only in very low numbers. *M. persicae* flights are recorded in late July or early August (unpublished data, J. Diamond, PEI Department of Agriculture).

Surveys of seed potatoes supplied by PEI to several Provinces and States have not yielded PVY<sup>N</sup>-infected plants except a few seedlots in New Brunswick, Nova Scotia, and Ontario. PEI potato seed grown in tobacco-producing areas of the USA has not been observed contributing to an infection due to PVY<sup>N</sup>.

**Ontario.** In 1990, symptoms of PVY<sup>N</sup> in tobacco were observed in some fields planted in close proximity to the infected potatoes. However, in Ontario, PVY<sup>N</sup> has been detected several times on the basis of tobacco symptoms as well as by tobacco bioassay from potato and tobacco crops (Singh, 1969; McDonald *et al.*, 1991). Infection in individual tobacco fields had ranged from 20 to 50% (unpublished observation, OMAF, Delhi). However, most of the infections in tobacco crop have been associated with the plantings of illegally imported foreign potato cultivars and infections in tobacco have dropped drastically in subsequent years following PVY<sup>N</sup> discovery. Volunteer plants and weed hosts may be perpetuating the virus. Compared to the Atlantic Provinces, there are early as well as an abundance of aphid species in Ontario.

**Comparative evaluation of situations in Europe and Canada.** From the foregoing description of European countries in the 1950s and the Canadian situation in 1990-91, the following comparison can be made. PVY<sup>N</sup> in Europe was widespread, with high infection levels in both tobacco and potato crops. Aphids were available early in the season and testing for PVY<sup>N</sup> was carried out on a limited scale with less sensitive procedures. On the other hand, in PEI, the PVY<sup>N</sup> infection of tobacco had not been observed; PVY<sup>N</sup> infection of potato was less than 2%; aphid pressure was negligible; testing had been done on a large scale to determine the distribution of the virus; and the test procedures used were more sensitive than those available in the 1950s in Europe. In the case of Ontario, the incidence of PVY<sup>N</sup> in tobacco fluctuates depending on the source of potato seed planted and the aphid pressure has been higher than PEI. Thus, in comparison with Europe, the amount of PVY<sup>N</sup> infection in Canada is "slight" and factors contributing to the spread of the virus are different.

In Europe, there is no evidence indicating that any attempt was made to eradicate PVY<sup>N</sup>. Most probably, it could have been because infection with PVY<sup>N</sup> reached epidemic proportions before its importance was realized. However, in Canada, PVY<sup>N</sup> was detected in the early stages of virus spread, when the incidence was very low.

In Europe, there was no evidence of an extensive leaf or tuber testing for the eradication of PVY<sup>N</sup>. In Canada, a high proportion of seedlots intended for planting were tested and those found infected were not used for subsequent planting, thus minimizing the amount of inoculum sources in the field.

The seed certification system used in Canada requires that the seedstocks must be derived from virus-free tissue culture material. They are grown for a limited generation in a flush through system. This practice ensures freedom of the nuclear stock from known viruses and viroids. Since there was no emphasis placed on testing for PVY<sup>N</sup> in Canada in earlier years, the present PVY<sup>N</sup> problem could have occurred a few years earlier and remained unnoticed. In the absence of visible symptoms in potato due to PVY<sup>N</sup>, virus spread in the plants for several generations could have taken place. Since PVY<sup>N</sup> testing of planting material would be carried out routinely, PVY<sup>N</sup> has less chance of building up in the potato crop. This was not the situation in Europe, because there certain commercial potato crops were almost wholly infected with PVY<sup>N</sup> in the 1950s (Weidemann, 1988).

**Spread of PVY<sup>N</sup> in eastern Canada.** The spread of the virus in eastern Canada was not as rapid as it was in Europe. From Table 1 it is clear that potato cv. Atlantic had 58% of samples with PVY<sup>N</sup> whereas other cultivars had only 5 to 13% samples with PVY<sup>N</sup>. These other cultivars (Russet Burbank and Shepody) are highly susceptible to PVY<sup>O</sup> by aphid inoculation under field conditions and possess high virus titres (Bagnall and Tai, 1986; Singh and Somerville, 1987). They should have been infected with PVY<sup>N</sup> more than cv. Atlantic if its spread was taking place by aphids (Bagnall and Tai, 1986). The infection of these cultivars is limited to only two farms where PVY<sup>N</sup> infection in cv. Atlantic samples was also high. This could be due to small-scale aphid transmission of PVY<sup>N</sup> to nearby fields from cv. Atlantic. Absence of infection of other cultivars on several farms, where PVY<sup>N</sup> was present in cv. Atlantic, may indicate that other cultivars were farther away from cv. Atlantic fields and aphids did not transmit the virus. Under eastern Canadian conditions a 600 m isolation band has been shown to prevent PVY<sup>O</sup> spread (Bagnall and Tai, 1986). The high incidence of PVY<sup>N</sup> infection in cv. Atlantic could be explained by multiplication and accumulation of infected seed over a few years and its movement to other parts of PEI. Atlantic seed potatoes were distributed in PEI from a limited source of four elite growers in 1989-90; any one of them could have had PVY<sup>N</sup> in their seed for a few years. As the seed potatoes were sold to other farmers, PVY<sup>N</sup> was introduced to other areas of the Island.

**Basis of PVY<sup>N</sup> eradication recommendations.** An eradication strategy must aim to reduce the amount of inoculum from which the disease starts or reduce the rate at which the disease increases in a plant population or both (Van der Planck, 1972). When applying this principle to a tuber-borne disease such as PVY<sup>N</sup>, the basis of eradication was to pre-

vent the multiplication of virus-infected seed potatoes and its spread to other parts of the country. As a result all potato seedlots found positive in any test (winter or summer), their sister-lots and those seedlots in close proximity (buffer lots) were banned from planting. This resulted in drastically reduced inoculum potential. In addition, only PVYN-free laboratory tested material was allowed for planting. In order to reduce the inoculum further, steps were taken to destroy the volunteer potatoes from PVYN positive fields, and the growing of potatoes in private gardens was prohibited in PEI.

The second basic premise for disease eradication is to generate data, which would allow proper timing and application of control measures, particularly for diseases which have sporadic occurrences. In the case of PVYN, infected tubers provide the initial foci. However, in the case of PEI, such foci are in trace amounts; therefore, massive leaf testing steps were recommended with 1,000 to 5,000 leaves per field to detect the seedlots containing PVYN. As shown (Table 3), 4.7 million leaves encompassing about 4,000 fields were tested in the 1991 growing season. Although the survey was large, only 0.58% of the samples were found to be positive for PVYN. This shows a trend for successful reduction of the inoculum source compared to 1990 potato crop (Table 1).

**Comparable situation in a PVYN outbreak in New Zealand.** A situation similar to the one in PEI was encountered in 1985 in New Zealand (Fletcher, 1989). PVYN was detected in 9% of potato samples in 1985. Three years later no PVYN was detected in any sample. The test procedure in New Zealand was similar to that used in Canada; where samples were bulked in groups of 100 leaves and assayed for PVYN using ELISA. *M. persicae* is a common aphid on potato plants in New Zealand, and in laboratory tests it was shown to be an efficient vector of PVYN (Fletcher, 1989).

The occurrence of PVYN does not mean it is going to be the dominant PVY strain in an area. For example, PVYN is an important problem in the Netherlands and Northern Germany, and less a problem in Czechoslovakia, Great Britain and France (Weidemann, 1988), although PVYN occurs in most European countries. Climatic conditions may be responsible for PVYN establishment. Thus, if PVYN in North America has not yet been established, it may be because of some unknown factors or niches, and thus justifying eradication efforts.

**Future prospects.** From the comparative situation between Europe and Canada, it is clear that eradication of PVYN in Canada is scientifically feasible. The experience in New Zealand supports this optimism. It is possible that seed producing areas of Canada may be free of PVYN within two years, but table-stock or processing potato fields in Ontario may require additional years. In order to declare an area free from a pathogen, regular monitoring of a large number of samples would be needed for four to five years. In the case of the potato in Canada, this period of five years represents the complete cycle of elite material subjected to field growing conditions. Failure to detect PVYN in the samples during this period should be considered as complete eradication. Since PVYN testing should be a part of pre-elite seed production, future crops should remain free of this strain of PVY.

### Acknowledgements

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Table 3. Survey of PVYN in potato in various provinces of Canada during summer 1991<sup>1</sup>.

Province	# Leaves Tested	# Fields		# PVYN Positives	
		Potato	Tobacco	Potato	Tobacco
Alberta	200	2	0	0	0
Manitoba	300	3	0	0	0
New Brunswick	450,000	297	0	1	0
Nova Scotia	34,000	21	0	1	0
Ontario	277,000	140	2	10	2
Prince Edward Island	3,900,000	3,495	4	10	0
Quebec	40,000	8	0	1	0
Total	4,701,500	3,966	6	23	2

<sup>1</sup> Data from Seed Certification Laboratories, Food Production and Inspection Branch, Agriculture Canada.

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## Pseudomonas-like early blight on sweet cherries

Thomas S.C. Li and P.L. Sholberg<sup>1</sup>

Sweet cherry trees in an experimental planting at Summerland, British Columbia, developed late bud opening and other symptoms including chlorotic patches on leaves and blight with gumming and dieback on current year growth. This disease was not caused by any known virus; however, the antibiotic streptomycin reduced disease incidence. The possibility that the causal agent was a bacterium was tested by inoculating tobacco plants with a suspension of bark from an infected tree and with a bacterium isolated from infected bark. Both treatments produced similar lesions on tobacco plants. Furthermore, a tissue suspension made from necrotic lesions on tobacco plants injected, or applied by budding, into 2-year-old cherry trees caused abnormal development. The bacterium has subsequently been identified as an unknown *Pseudomonas* spp. with similarity to *P. cepacia* and *P. gladioli*.

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En Colombie-Britannique à Summerland, dans une plantation expérimentale, des cerisiers sucrés ont développés une ouverture tardive des bourgeons, des régions chlorotiques sur les feuilles et des brûlures avec de la gommose et du dépérissement sur les nouvelles croissances de l'année. Cette maladie n'a pas été causée par un virus connu; néanmoins, la streptomycine a réduit l'incidence de la maladie qui impliquait une bactérie comme agent causal possible de la maladie. On a évalué la possibilité de la présence d'une bactérie en inoculant des plants de tabac avec une suspension d'écorce d'un arbre infecté et d'un isolat de bactérie provenant d'une écorce infectée. Les deux traitements ont produit des lésions similaires sur les plants de tabac. De plus, une solution en suspension composée de tissus nécrotiques de tabac a été injectée, ou appliquée durant une méthode de propagation par bourgeonnement, à des cerisiers vieux de deux ans, et cette inoculation a provoqué un développement anormal de ces derniers. Par la suite, la bactérie a été identifiée comme étant une espèce inconnue de *Pseudomonas* ayant une similarité avec *P. cepacia* et *P. gladioli*.

In the Spring of 1990 and 1991, virus-free sweet cherry trees (*Prunus avium* L., var. Bing) and Japanese flowering cherry trees (*Prunus serrulata* Lindl., var. Kwanzen) at the Agriculture Canada, Research Station, Summerland, British Columbia, developed symptoms which are new to this area. There has been no documented report of this disorder from commercial orchards in the Okanagan-Similkameen Valleys of British Columbia. The symptoms appeared to be somewhat similar to those caused by *Pseudomonas syringae* van Hall on stone fruit (Cameron, 1962; Davidson, 1973). Specifically, the infected branches showed late bud opening, a few chlorotic patches on fully expanded leaves, followed by blight early in the spring (May-June). On current year's growth a gumming and dieback extended in both directions. These symptoms occurred on the same trees every year and slowly spread to adjacent trees. Infected trees developed cankers on the trunk and eventually died if the infected branches were not removed in time. Infected trees were tested for virus by sap transmission to herbaceous hosts and by bud inoculation to woody hosts with negative results. It can be concluded that this disease is not caused by any known virus.

Streptomycin has been used in New York State to control blister spot caused by *Pseudomonas syringae* pv. *papulans* (Rose) Dhanvantari, in commercial orchards (Burr, 1990). Proebsting (1988) reported that frequent sprays of bacteri-

cides such as streptomycin usually kept *P. syringae* populations near undetectable levels. Infected trees at the Experimental Station that were sprayed with streptomycin (0.6g/L and 3000 L/ha) in late September and early March showed reduced symptoms, and there was no new infection on healthy trees nearby.

Three experiments were conducted to attempt to confirm that this disease was caused by *Pseudomonas* spp. Three 6-year-old trees (var. Bing) were chosen in July, 1990 for this study:

Tree 1 had branches with symptoms, some leaves still attached, and had green buds.

Tree 2 had branches with symptoms, without leaves, and had brownish but living buds.

Tree 3 served as a control tree and had leaves and healthy buds.

In the first experiment, infected branches collected from Tree 1 and Tree 2 and control branches from Tree 3 were surface sterilized in 10% bleach for ten min, rinsed with dis-

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tilled water three times and allowed to dry. Their bark was then ground in 0.05M phosphate buffer (pH 7.0) with mortars and pestles. A plastic syringe (Plastipak®) was used to inject 0.5 ml of the buffered bark suspension from each test tree into the underside of the leaves of *Nicotiana tabacum* L. (var. F2C1) via lateral veins as described by Klement (1963). Four leaves on each of four test plants were inoculated and grown in the greenhouse with a 16 h photoperiod and temperature of  $20 \pm 2^\circ\text{C}$ .

Brown patches, 5-10 mm in diameter were observed, near the areas that were injected with the bark suspensions prepared from Tree 1 and 2, four days after inoculation. Only small brown necrotic spots appeared where the needle had pierced the leaf on the control. The development of large brown patches indicated that the inoculum from Tree 1 and 2 were pathogenic.

In the second experiment, small pieces of leaf tissue (approx. 1 cm in diam.) were collected from the edge of the brown necrotic lesions on the inoculated tobacco plants and from control leaves. The tissue was ground in 0.05M phosphate buffer.

Treatment 1: The suspension (0.05 ml) from diseased tobacco tissue was injected with a hypodermic syringe during the growing season into each of six leaves of two, 2-year-old cherry trees var. Bing. Treatment 2: The suspension used

in Treatment 1 was also applied to the trunk of a 2-year-old Bing tree after a "T-cut" was made and wrapped with budding bands. Treatment 3: Healthy tobacco leaf suspensions were injected into six leaves of a Bing tree as control.

The inoculated trees were kept in the greenhouse without heat over the winter and by the beginning of March 1991, the leaves on the control tree had grown to 6 cm long (Fig. 1). The tree with the "T-cut" soaked with infected suspension (Treatment 2) had flowers, but leaf buds remained closed. The trees with leaves injected with infected suspension during the previous growing season were still dormant.

In the middle of June 1991, the control trees had grown normally while trees both from Treatments 1 and 2 lacked new annual growth and had small leaves on 2-year-old wood. Light brown gumming occurred around the dead buds below new leaves.

In the third experiment, sterilized bark sections were inoculated on potato dextrose agar (PDA) and bacteria were isolated aseptically. Sub-cultures of the bacteria growing from the bark were isolated and streaked on PDA. Three loops of bacteria, from a culture grown at  $22^\circ\text{C}$  for two days, were mixed with 5 ml of 0.05M phosphate buffer and injected into four tobacco leaves with 0.5 ml/leaf. Brown patches, similar to those indicated in Experiment 1, started to show at the injection site four days after inoculation.

A bacterial isolate was sent to Dr. G.S. Saddler of the International Mycological Institute, Ferry Lane, Kew, Surrey, TW9 3AF, U.K. for identification, but it could not be positively identified as any known species within the genus *Pseudomonas*. Dr. Saddler pointed out that it was non-fluorescent and showed some similarity to *Pseudomonas cepacia* and *P. gladioli*. Further studies are needed to characterize this causal agent, explain its mode of spreading in the orchard and to develop an effective control for this disease.

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Fig. 1. Effect of tobacco leaf suspension on cherry trees. Right: Control: Leaves inoculated with tobacco leaf suspension from healthy leaves. Middle: T-cut area freshly soaked with tobacco leaf suspension from necrotic lesions and tied with a budding band. Left: Tobacco leaf suspension from necrotic lesions injected into mature leaves.

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