Canadian Plant Disease Survey

Inventaire des maladies des plantes au Canada

Vol. 66, No. 2, 1986

Vol. 66, N° 2, 1986

Agriculture Canada

Canada

Canadian Plant Disease Survey

Inventaire des maladies des plantes au Canada

Volume 66, Number 2, 1986

CPDSAS 66 (2) 31-61 (1986) ISSN 0008-476X

Volume 66, Numéro 2, 1986

Contents/Contenu

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

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

Research Branch, Agriculture Canada

Compilers: H.S. Krehm, PhD.
P. Beauchamp, M.Sc.,
Research Program Service,
Agriculture Canada, Ottawa, Ontario K1A 0C6

Direction de la recherche, Agriculture Canada

Compilateurs: H.S. Krehm, PhD.
P. Beauchamp, M.Sc.
Service des programmes de recherche,
Agriculture Canada, Ottawa (Ontario) K1A 0C6

ERRATUM

Volume 65:1, 1985

Page 16

The last paragraph should read:

The existence of a significant negative correlation between numbers of root lesion nematodes and the presence of common root rot in the cultivar Bruce did not indicate that the two groups of organisms were antagonistic to each other. Furthermore, the positive correlation between numbers of stunt nematodes and the incidence of common root rot did not confirm a synergistic interaction. More information is necessary, especially in the spring when host plants are at the seedling stage to further characterize the relationship between such disease inciting organisms. In addition, acurate identifications of fungal and nematode species must be completed.

Relationship between root lesion nematodes and potato yields

J. Kimpinski[1] and K.B. McRae[2]

Numbers of *Pratylenchus penetrans* in experimental potato plots were recorded at planting over a seven-year period. Subsequent tuber yield increases (cv. Superior) after treatment with aldicarb in comparison to untreated plots were recorded also. A straight-line relationship gave an estimate of expected yield increases after treatment. Further studies at other locations and with other cultivars should make numerical relationships of this type useful for a nematode advisory service.

Can. Plant Dis. Surv. 66:2, 33-34, 1986.

Sur une période de sept ans, on a noté au moment de la plantation le nombre de *Pratylenchus penetrans* présents dans des parcelles expérimentales de pommes de terre. On a aussi consigné les augmentations de rendement en tubercules (cv. Superior) après un traitement à l'aldicarbe en comparaison avec les parcelles non traitées. Une relation linéaire permet d'évaluer l'augmentation de rendement après traitement. Des études plus poussées à d'autres endroits et avec des cultivars différents devraient permettre à un service de consultation sur les nématodes d'utiliser ces relations numériques.

Introduction

The deleterious effect of the root lesion nematode, *Pratylenchus penetrans* (Cobb) Filipjev and Sch. Stek. 1941, on potatoes has been demonstrated in Michigan (Bernard and Laughlin 1976, Vitosh *et al.* 1980) and Ontario (Olthof and Potter 1973, Olthof *et al.* 1985). A previous investigation on Prince Edward Island showed that tuber yields improved when fields harboring large populations of *P. penetrans* were treated with a nematicide (Kimpinski 1982). The extent of the yield increases in the above studies appeared to be related to the size of the root lesion nematode populations at planting. This paper examines the relationship between numbers of *P. penetrans* at planting and the resulting percentage tuber yield increases of potatoes (cv. Superior) in experimental plots treated with aldicarb as compared to untreated plots.

Materials and methods

Aldicarb (Temik 15G) was applied at planting in the furrow at 2.24 kg/ha a.i. in each of the seven years. Small whole tubers of Elite III seed were planted in late May or early June at 30 cm intervals in rows 6 m long and 0.9 m apart. There were 4 rows in each plot and the 2 centre rows were harvested in the middle of October. Yield measurements were limited to tubers with no obvious defects in the weight range of 113-340 g (4-12 oz). Recommended cultural and fertilizer practices were followed. Endosulfan and mancozeb were applied as needed for insect and late blight control, respectively. Diquat was applied as a top killer. The soil was a fine sandy loam with an approximate particle size distribution of 70% sand, 20% silt, and 10% clay, and a pH range of 5.1-6.0.

The number of nematodes in soil just prior to treatment was estimated by removing 10 cores, 20 cm in depth from each plot. Each sample was thoroughly mixed and a 50-g subsample was placed in a Baermann pan (Townshend 1963). After 7 days at room temperature, root lesion nematodes which had emerged from soil were counted at 60X with a stereomicroscope. Nematode data were expressed as numbers per kg of dry soil and transformed to logarithms for statistical computations. Yield increases in aldicarb-treated plots as compared to untreated plots were expressed as percentages and transformed to angles (arcsines). Means for the correlation and regression computations were based on at least 4 replicates in each year.

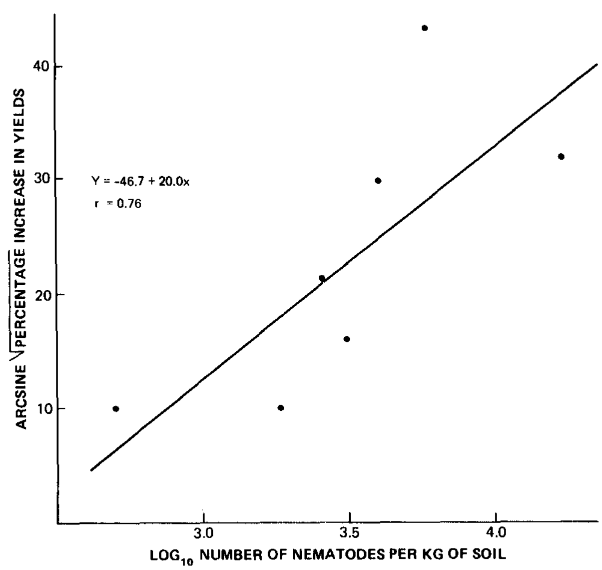

Figure 1. Relationship over seven years between percentage increase of yields in potato cv. Superior after treatment with aldicarb at 2.24 kilograms active ingredient per hectare and numbers of *Pratylenchus penetrans* in soil at planting.

[1] *Nematologist, Agriculture Canada Research Station, Charlottetown, P.E.I. C1A 7M8.*

[2] *Regional Statistician, Agriculture Canada Research Station, Kentville, N.S. B4N 1J5.*

Accepted for publication February 11, 1986

Table 1. Numbers of *Pratylenchus penetrans* at planting and corresponding yield increases of potato cv. Superior after treatment with aldicarb.

Year	Numbers of Nematodes per kg of dry soil at planting	Yield increase* (%)
1980	500	3
1981	1900	3
1982	2500	13
1979	3100	8
1983	3900	25
1978	6000	47
1984	17000	28

* Difference between untreated plots and plots treated at planting with aldicarb at 2.24 kg active ingredient per hectare.

Results and discussion

Table 1 shows the numbers of *P. penetrans* at planting and the corresponding yield increases in Superior potatoes when aldicarb was applied in experimental plots over a seven-year period at the Charlottetown Research Station. Large yield increases were realized when aldicarb was applied to plots which contained 3900 or more nematodes per kg of soil at planting. Figure 1 describes the same relationship when percentage yield increases after treatment and given levels of nematodes at planting were transformed to angles (arcsines) and logarithms, respectively.

Most field samples from the Maritime provinces which are processed at the Charlottetown Research Station contain between 1,000 and 10,000 root lesion nematodes per kg of soil. Therefore, the expected yield gain after treatment could be estimated from this straight-line relationship, if information were available on the size of nematode populations at planting. It is likely that a curvilinear relationship would be more appropriate when nematode populations are very large. However, it should be stressed that the sampling scheme in this study provided estimates only to within about 50% of the true population mean (Proctor and Marks 1974). Further studies at other locations with varying soil and weather conditions, and with other cultivars should alleviate some of the variability. In the meantime, estimates of potential yield gains or losses, and advice on nematicide treatments should be dispensed with a cautionary note.

Literature cited

1. Bernard, E.C. and C.W. Laughlin. 1976. Relative susceptibility of selected cultivars to *Pratylenchus penetrans*. J. Nematol. 8:239-242.
2. Kimpinski, J. 1982. The effect of nematicides on *Pratylenchus penetrans* and potato yields. Am. Potato J. 59:327-335.
3. Olthof, T.H.A. and J.W. Potter. 1973. The relationship between population densities of *Pratylenchus penetrans* and crop losses in summer-maturing vegetables in Ontario. Phytopathology 63:577-582.
4. Olthof, T.H.A., B.D. McGarvey and M. Chiba. 1985. Oxamyl in control of *Pratylenchus penetrans* on potatoes. Can J. Plant Pathol. 7:155-160.
5. Proctor, J.R. and C.F. Marks. 1974. The determination of normalizing transformations for nematode count data from soil samples and of efficient sampling schemes. Nematologica 20:395-406.
6. Townshend, J.L. 1963. A modification and evaluation of the apparatus for the Oostenbrink direct cottonwool filter extraction method. Nematologica 9:106-110.
7. Vitosh, M.L., J.W. Noling, G.W. Bird and R.W. Chase. 1980. The joint action of nitrogen and nematicides on *Pratylenchus penetrans* and potato yield. Am. Potato J. 57:101-111.

Incidence and etiology of pea rots in southwestern Ontario

J.C. Tu[1]

A survey was conducted in Essex and Kent countries in southwestern Ontario in the summers of 1983 and 1984 to determine the incidence and severity of root rots of pea. Approximately 50 and 75% of the commercial fields were surveyed in 1983 and 1984. Cultivars included in the survey were Green Giant (GG) 313, 451, 521, EJ 235 and Mini 381. The incidence of root rot for 1983 and 1984 was not significantly different between the 2 years and averaged 26.5 and 25.9%, respectively. However, the disease severity differed significantly among some of the cultivars. Generally, there was a positive correlation between yield and the incidence X severity. A total of 782 isolations were made. The frequencies of isolating *Fusarium solani, F. oxysporum, Aphanomyces euteiches* and *Pythium* spp. were 7:4:1:1.

Can. Plant Dis. Surv. 66:2, 35-36, 1986.

Durant l'été de 1983 et de 1984, on a effectué une enquête dans les comtés d'Essex-Kent afin de déterminer l'incidence et la gravité du pourridié du pois. L'enquête a couvert environ 50 à 75 % des champs commerciaux en 1983 et 1984 et porte sur les cultivars Green Giant (GG) 313, 451, 521, EJ 235 et Mini 381. L'incidence moyenne du pourridié en 1983 et 1984 a été respectivement de 26,5 et 25,9 % et n'a pas montré de différence significative. Toutefois, la sévérité de la maladie diffère significativement entre certains cultivars. Généralement, on retrouve une corrélation positive entre le rendement et l'incidence X sévérité. Au total, 782 isolements ont été effectués avec une fréquence respective d'isolement de 7:4:1:1 pour *Fusarium solani, F. oxysporum, Aphanomyces euteiches* et *Pythium* spp.

Introduction

Southwestern Ontario is a prime agricultural region in Canada. A variety of vegetable crops are grown here, including processing green peas. In 1981, Ontario growers farmed 8600 ha of peas with a total value of 9.3 million dollars (2) of which 60% was grown in southwestern Ontario and 30% in Essex and Kent counties.

Pea root rot is a world-wide disease problem that prevails in pea growing areas, and has become the major problem confronting pea growers and processing companies in southwestern Ontario. Root rot threatens the continuation of the pea industry in this area. Unfortunately, very little research has been done on pea root rots in Ontario (4). There are more than 20 different fungi that can cause pea root rots (6). The disease no doubt is caused by different fungi in different regions of the world. Thus, a detailed survey is necessary before a research program can be developed.

The incidence and severity of pea root rots were surveyed and a large number of isolations were made on diseased plants to assess the relative importance of various fungi in causing root rots in southwestern Ontario.

Materials and methods

A survey was conducted in Essex and Kent counties in southwestern Ontario. Totals of 48 and 58 fields (or 600 and 800 ha) were surveyed in 1983 and 1984, respectively. The acreages surveyed represented 50 and 75% of the total acreage of the biggest pea processing company in this area. Since peas were grown exclusively for the company on contracts, the growers' fields which were to be surveyed were selected randomly from lists provided by the company.

Surveys were conducted between late May and mid-June each year. For each field, four replications of 10 plants were dug up and the incidence and severity of root rots were assessed. The severity was determined using a 0 to 4 scale where 0 = healthy, 1 = 0-10%, 2 = 11-25%, 3 = 26-50% and 4 = 51-100% root discolored.

A large scale isolation was conducted in 1983 in conjunction with the disease survey. A total of 465 samples was collected randomly from 33 pea fields. Isolations were made by cutting small pieces (ca., 1 mm^3) of discolored root tisue. The tissue pieces were surface sterilized in 1.65% sodium hypochloride solution for 1 min, rinsed in distilled water and placed on an agar plate containing potato dextrose agar (PDA) and 40 μg/ml of novobiocin. For each petri plate, 4 pieces of tissue were plated. The plates were incubated at 22°C. After 4 days of incubation, the fungi that grew out of the tissue pieces were isolated and later identified.

Results and discussion

Root rot survey. The incidence of pea root rot varied widely from field to field, and differed largely form one cultivar to the other. Variation among fields ranged from 0 to 98.5% and cultivars from 15.8 to 53.5% (Table 1). Similarly, the disease severity also varied widely among cultivars. For example, GG 451 had an average root rot severity rating of 1.6 versus a 3.3 rating for GG 313 on a 0 to 4 scale (Table 1). The differences in incidence and severity among pea cultivars indicate that some cultivars may be more tolerant to one or more root rot pathogens present in the fields.

There is no doubt that root rot is severe, and incidence of this disease is high in the fields in Essex and Kent counties.

Isolation of root rot organisms. Isolations were made on 465 samples collected from 33 pea fields, from which 782 fungal cultures were isolated. Of the 782 fungal cultures 356 were *Fusarium solani*, 201 were *F. oxysporum*, 58 were *Apha-*

[1] *Research Station, Agriculture Canada, Harrow, Ontario NOR 1GO.*
Accepted for publication February 11, 1986

Table 1. Incidence and severity of pea root rot in 1983 and 1984 in Essex and Kent counties.

	Area (ha)						Root rot			
	Total		Surveyed		% Surveyed		Incidence (%)		Severity[a]	
Cultivar	1983	1984	1983	1984	1983	1984	1983	1984	1983	1984
E.J. 235	92.7	90.9	45.9	67.7	50.0	73.6	53.5	34.1	2.1	2.7
Sparkle	131.8	66.4	80.5	48.2	60.6	72.7	15.8	21.1	3.2	2.8
Mini 381	207.7	135.0	111.4	99.5	53.6	74.1	21.3	22.8	2.0	2.1
G.G. 313	188.2	-[b]	74.5	-	39.9	-	43.3	-	3.3	-
G.G. 451	10.0	422.0	0.0	320.0	0.0	75.8	NA	20.2	NA	1.6
G.G. 512	387.3	307.0	222.3	248.2	57.3	80.8	20.7	21.1	2.2	1.8

[a] Root rot rating based on a scale of 0-4 where 0 = healthy, 1 = 0-10%, 2 = 11-25%, 3 = 26-50% and 4 = 51-100% of the root discolored.
[b] Cultivar discontinued in 1984.

nomyces euteiches, 48 were *Pythium* spp. (mostly *P. ultimum*), 6 *Rhizoctonia solani*, 5 *Thielaviopsis basicola*, and the remainder (108) were non-pathogenic fungi, mostly common soil inhabitants.

F. solani was isolated from all pea fields with root rot. *F. oxysporum*, *Pythium* spp. and *A. euteiches* were isolated from 23, 18 and 13 of the 33 fields, respectively. *A. euteiches* was isolated for the first time in southwestern Ontario (5) and it was not as widely distributed as *F. solani* and *F. oxysporum*. It must be noted, however, that *A. euteiches* is a very serious pea root rot pathogen in Wisconsin and Minnesota (3).

In addition, the near wilt (caused by *F. oxysporum* f. sp. *pisi* race 2) which was reportedly only of sporadic occurrence in Ontario by McNeill in 1959, (1) has now been found to be widely distributed in the area surveyed.

There is no doubt that most of the cultivars in use appear to be susceptible to the root rot complex. This is due to our lack of understanding of the etiology of this disease complex. This investigation has identified many severely infested fields, and has also established the relative incidence of each fungal component in the complex. These findings enable the growers to avoid planting peas in heavily infested fields and to select cultivars with specific tolerance or resistance to one or more important pathogens in their fields. This survey will help in establishing research priorities for controlling pea root rot diseases.

Literature cited

1. McNeil, B.H. and H. Howard. 1959. Near-wilt of peas in Ontario. Can. J. Plant Sci. 39:483-490.
2. Ontario Ministry of Agriculture and Food. 1981. Agriculture Statistics for Ontario. Publication 20. Toronto, Ontario. pp. 132.
3. Pfender, W.F. and D. J. Hagedon. 1983. Disease progress and yield loss in Aphanomyces root rot of peas. Phytopathology 73:1109-1112.
4. Reyes, A.A. 1980. Pea root rot development and associated fungal pathogens in Ontario fields. Plant disease 64:392-393.
5. Tu, J.C. 1985. *Aphanomyces euteiches* on pea in Ontario, Canada. Plant Disease 69:268.
6. U.S. Department of Agriculture. 1960. Index of Plant Diseases in the United States. Agr. Handbook No. 165. U.S. Government Printing Office, Washington, D.C. 531 pp.

Interactive effects of foliar diseases and fungicide sprays in cultivars of winter wheat in Ontario

J.C. Sutton and G. Roke[1]

Foliar diseases were monitored and yields were estimated in five cultivars of winter wheat grown in field plots and sprayed or not sprayed with propiconazole (Tilt). Septoria tritici blotch and septoria nodorum blotch progressed collectively at similar rates among the tested cultivars. Ranking of the cultivars in descending order of powdery mildew intensity was Favor, Augusta, Fredrick and Frankenmuth, and Houser. The virulence formula of the race *Erysiphe graminis* var. *tritici* isolated from the plots was 1,2,3a,3b,4,5/Ma,3c. Progress of leaf rust was most rapid in Favor, moderately rapid in Fredrick but slow in Frankenmuth, Houser and Augusta. The septoria blotches became moderately severe on the upper leaves and spikes only when leaf rust or powdery mildew were mild or absent. Tan spot progressed in all cultivars, mainly before anthesis. Propiconazole managed all foliar diseases effectively except for chlorosis and necrosis of unknown etiology. The fungicide significantly increased grain yields of all cultivars except Fredrick.

Can. Plant Dis. Surv. 66:2, 37-41, 1986.

On a surveillé les maladies foliaires et évalué les rendements de cinq cultivars de blé d'hiver dans des parcelles traitées et non traitées avec du propiconazole (Tilt). La tache septorienne (*Septoria tritici*) et la tache des glumes (*S. nodorum*) se sont développées au même rythme parmi les cultivars traités. Le classement des cultivars par ordre décroissant d'intensité du blanc est le suivant: Favor, Augusta, Fredrick et Frankenmuth et Houser. La formule de virulence de la race d'*Erysiphe graminis* var. *tritici* isolée des parcelles était 1,2,3a,3b,4,5/Ma,3c. La progression de la rouille des feuilles s'est avérée rapide chez Favor, modérément rapide chez Fredrick et lente chez Frankenmuth, Houser et Augusta. Les taches septoriennes sont devenues plus ou moins importantes sur les feuilles supérieures et les glumes, seulement lorsque la rouille des feuilles et le blanc étaient faibles ou absents. *Pyrenophora trichostoma* s'est développé chez tous les cultivars principalement avant l'anthèse. Le propiconazole a assuré une gestion efficace de toutes les maladies foliaires, exception faite des chloroses et nécroses d'origine inconnue. Le fongicide a permis une augmentation significative des rendements en grain de tous les cultivars sauf Fredrick.

Introduction

New cultivars of soft-white winter wheat (Favor, Frankenmuth, Augusta and Houser) were introduced into Ontario agriculture in 1980-83. The introduction coincided with a surge of interest in the province in intensive management of winter wheat, particularly the use of fungicides for managing foliar diseases. Foliar fungicides have improved productivity of winter wheat in some growing seasons in Ontario (Sutton 1985) but the extent of yield promotion was a function of the cultivar (Sutton and Roke 1984). Overall disease reactions of the new cultivars have been recorded in routine variety trials but patterns and rates of disease progress and comparative responses of the cultivars to foliar fungicides have not been reported. Because of the potential importance of these variables in disease management, we monitored diseases and examined responses to fungicides of the new cultivars and of Fredrick, the principal cultivar grown in recent years.

Materials and methods

Winter wheat cultivars were grown in plots (1.3 × 3.0 m) arranged in a randomized complete block design at the Elora Research Station near Guelph, Ontario in 1982-83 and 1983-84). Two plots of each cultivar, one sprayed with fungicide and the other unsprayed, were included in each block. Cultivars Augusta, Favor, Fredrick and Houser were grown in both years and Frankenmuth also was grown in 1983-84. The seed was treated with Vitaflo 250 (carbathiin, Uniroyal Inc.) and sown on 17 September 1982 and 15 September in 1983. In both growing seasons the wheat followed red clover in the crop sequence and received 80 kg N (ammonium nitrate) in mid-April. The fungicide product Tilt 250 EC (propiconazole, Ciba-Geigy Canada Ltd) was applied to the wheat at a dose of 125 g a.i. in 300 L water/ha using an air-pressurized backpack sprayer equipped with four hollow-cone D2-13 nozzles and operated at 200 kPa. In 1983, Tilt was applied on 13 and 27 May and 27 June when the wheat was at growth stages (GS) 25-29, 31-32 and 55-60 (Zadoks *et al.* 1974). In 1984, Tilt was applied once on 21 June (GS 69).

Disease on the leaves and spikes was assessed using the scale of Horsfall and Barratt (discussed in Horsfall and Cowling 1978). Assessments were conducted at about weekly intervals beginning 24 May in 1983 and 13 June in 1984. Total disease and senescence was assessed by estimating the percent area discolored and the approximate severity of individual diseases on the assessed leaves was noted. Septoria tritici blotch and septoria nodorum blotch were assessed collectively by estimating the percent leaf area bearing pycnidia of the respective causal pathogens, *Mycosphaerella graminicola* (Fuckel) Schroeter and *Leptosphaeria nodorum* Miller. In 1984, powdery mildew, caused by *Erysiphe graminis* DC. ex Merat f. sp. *tritici* Em. Marchal was assessed on 13 June. One isolate of the pathogen was obtained from each cultivar and characterized with respect to race using the methods of Bailey and MacNeill (1983).

[1] *Department of Environmental Biology, University of Guelph, Guelph, Ontario N1G 2W1.*

Accepted for publication February 11, 1986

To assess yield and yield components in 1983, the number of spikes/m^2 were counted, total grain yield was obtained using a small-plot combine harvester and 1000-kernel weights were estimated. In 1984, 1000-kernel weights were determined for hand-harvested grain. Grain yields were expressed based on 14.8% moisture content.

After each time of sampling, the four plot means for each treatment were analysed by standard analysis of variance and overall treatment means were compared using Duncan's new multiple range test (Steel and Torrie 1980). In 1984 the treatment means were transformed for analysis, using the arcsin transformation. Apparent infection rates (r) were computed from plot means according to the formula (Zadoks and Schein 1979).

$$r = (\text{logit } x_2 - \text{logit } x_1)/(t_2 - t_1)$$

Results

1983. The principal diseases on the wheat foliage were septoria tritici blotch, septoria nodorum blotch and tan spot. Tan spot, caused by *Pyrenophora trichostoma* (Fr.) Fckl., developed mainly at GS 25-28 (mid-May to mid-June) while the septoria blotches (especially septoria tritici blotch) progressed chiefly at GS 25-85 (mid-May to mid-July).

With certain exceptions, disease progressed at similar rates in all cultivars tested. Discoloration of the foliage, associated mainly with disease and senescence, was similar among the various cultivars (unsprayed) in June, but was significantly more extensive in Fredrick than the other cultivars in July (Table 1). Intensities of pycnidia of *M. graminicola* and *L. nodorum* were higher in Houser than in other cultivars when estimated on 7, 21 and 28 June but not on the flag leaf later in the growing season (Table 2). However, r values calculated for progressive increases in pycnidial intensities on the third or fourth leaves in the periods of 14-28 June, 14 June-5 July and 21 June-5 July did not differ significantly among cultivars and ranged from 0.17 to 0.26. Few pycnidia developed on the flag leaves.

Foliar diseases usually were much less intense when Tilt was applied than in the untreated wheat. Tilt reduced foliar discoloration assessed in cultivar Augusta on 21 June, in all cultivars on 28 June, and in Augusta, Favor (leaf 1 only) and Houser, but not Fredrick, on 12 July (Table 1). Tilt applied on 13 or 27 May failed to reduce estimated discoloration of leaves 6, 5 and 4 variously assessed on 24 and 31 May and 7 June. However, the fungicide markedly restricted areas bearing pycnidia of the septoria blotch pathogens when assessed on leaves 6 to 1 at various times in the period of 24 May to 19 July (Table 2). In most instances few or no pycnidia were evident in any cultivar.

Table 1. Disease progress, and effects of Tilt on disease progress, in four cultivars of winter wheat at Elora in 1983.

Cultivar	Tilt applied[+]	Estimated % leaf area discolored: 21 June Leaf 3	28 June Leaf 2	12 July Leaf 2	12 July Leaf 1
Augusta	–	41 b[++]	23 bc	88 de	30 cd
	+	23 a	15 a	74 bc	15 ab
Favor	–	40 b	49 e	80 cd	49 e
	+	45 b	40 de	89 def	31 d
Fredrick	–	45 b	32 cd	98 f	68 f
	+	44 b	18 ab	97 ef	67 f
Houser	–	44 b	25 bc	84 d	24 bcd
	+	22 a	12 a	60 a	10 a

[+] Tilt was applied on 13 and 27 May, and 22 June.
[++] Numbers in a column followed by the same letter are not significantly different (P = 0.05, Duncan's new multiple range test).

Table 2. Progress of septoria tritici blotch and septoria nodorum blotch, and effects of Tilt on disease progress, in four cultivars of winter wheat at Elora in 1983.

Cultivar	Tilt applied[+]	Estimated % leaf area with pycnidia: 24 May Leaf 6	7 June Leaf 5	21 June Leaf 4	28 June Leaf 3	5 July Leaf 2	19 July Leaf 1
Augusta	–	24 bc[++]	22 bc	17 c	20 b	15 c	0.8 ab
	+	4 a	1 a	0 a	0 a	0 a	0.3 a
Favor	–	24 bc	24 c	16 c	23 b	10 b	2.1 c
	+	5 a	4 a	0 a	0 a	1 a	0.9 ab
Fredrick	–	19 b	14 b	12 bc	15 b	8 b	0.3 a
	+	3 a	0 a	0 a	0 a	0 a	0.2 a
Houser	–	28 b	29 c	23 d	34 c	9 b	1.2 b
	+	7 a	1 a	0 a	0 a	0 a	0.8 ab

[+] Tilt was applied on 13 and 27 May, and 22 June.
[++] Numbers in a column followed by the same letter are not significantly different (P = 0.05, Duncan's new multiple range test).

Table 3. Comparative progress of disease, estimated as discoloration of the leaves and spikes, in five cultivars of winter wheat at Elora in 1984.

	Estimated % discoloration+						
	20 June	30 June		7 July		13 July	19 July
Cultivar	Leaf 3++	Leaf 2	Leaf 3	Leaf 1	Leaf 2	Leaf 1	Spike
Augusta	30 b*	8 a	69 ab	6 a	56 c	21 c	14 b
Favor	28 b	25 b	57 bc	60 c	92 b	72 a	30 a
Frankenmuth	15 a	3 a	26 d	9 ab	51 a	31 b	16 b
Fredrick	54 c	10 a	73 a	12 ab	88 b	38 b	16 b
Houser	15 a	4 a	26 d	10 ab	67 c	33 b	9 c

+ Data were detransformed from arcsin-transformed values.
++ Leaves 1 to 3 were the flag, penultimate and third leaves, respectively.
* Values in a column followed by the same letter are not significantly different (P = 0.05, Duncan's new multiple range test).

Tilt significantly promoted yield of Augusta by 13% to 7.9 t/ha, of Favor by 14% to 6.4 t/ha, and of Houser by 17% to 7.7 t/ha. The grain yield of Fredrick was 6.4 t/ha and not increased by Tilt. Yields of sprayed Augusta and Houser were significantly higher than those of sprayed Favor and Fredrick. Thousand kernel weights were not increased significantly in any cultivar. Numbers of spikes/m^2 were 4 to 100% higher in the sprayed than unsprayed wheat.

1984. Powdery mildew, leaf rust caused by *Puccinia recondita* Rob. ex. Desm. f.sp. *tritici*, septoria tritici blotch, septoria nodorum blotch, tan spot and leaf chlorosis and necrosis of unknown etiology were observed in the wheat. Tan spot was confined to the lower half of the canopy and progressed mainly before anthesis. Powdery mildew was first observed at late tillering (GS 26-29) and continued to progress in Augusta and Fredrick until the upper canopy senesced. Rust and the septoria blotches became intense in the upper canopy of some cultivars after anthesis.

Patterns were evident in the ranking of the cultivars with respect to discoloration of the upper three leaves (Table 3). Discoloration usually was relatively severe in Favor and Fredrick, mild in Houser and Frankenmuth, and variously mild to severe in Augusta. Discoloration of Favor was chiefly associated with severe leaf rust, but powdery mildew was severe on leaf 3 and chlorotic flecking affected leaves 2 and 1. Fredrick was affected severely by powdery mildew (leaves 3 and 2), leaf rust (leaf 1) and chlorotic flecking (leaves 3, 2 and 1), but mildly by the septoria blotches. Thrips (Thripidae) completely discolored some flag leaves of Fredrick but not other cultivars. Powdery mildew and the septoria blotches were the main diseases on Augusta, but traces of leaf rust were also observed. Mild leaf rust and septoria blotches developed on Frankenmuth and Houser.

Disease on the spikes differed substantially among the cultivars (Table 3). Powdery mildew was severe on Augusta but not in the other cultivars. Leaf rust was severe on spikes of Favor, moderately severe in Fredrick, but only traces were observed in Augusta, Frankenmuth and Houser. Septoria nodorum blotch was moderately severe on the spikes of Frankenmuth, Fredrick and Houser but only traces of the disease were evident in Augusta and Favor.

Table 4. Intensity of powdery mildew in five cultivars of winter wheat estimated on 13 June 1984 at Elora.

Cultivar	Growth Stage	Powdery Mildew (%)+
Augusta	54-57	41c++
Favor	56-67	66d
Frankenmuth	56-57	19 b
Fredrick	56-57	27 b
Houser	56-57	3 a

+ Estimated area of leaf 4 with powdery mildew. Data were detransformed from arcsin-transformed values.
++ Numbers in a column followed by the same letter are not significantly different (P = 0.05 Duncan's new multiple range test).

Wide differences in susceptibility among the cultivars to powdery mildew were evident in assessments of leaf 4 (Table 4). Favor was the most susceptible but Augusta also was severely diseased. Little powdery mildew developed on Houser. Frankenmuth and Fredrick were of intermediate susceptibility. The same ranking was obtained when powdery mildew was estimated on the sheaths of the lower leaves. Isolates of *E. graminis* var. *tritici* from the cultivars revealed only one race of the pathogen. The virulence formula of this race was 1, 2, 3a, 3b, 4, 5/Ma, 3c. This race overcomes resistance genes Pm Ma, Pm 3c.

Tilt applied on 21 June markedly suppressed all of the diseases, except chlorotic flecking, observed in the upper canopy, as exemplified by data for the flag leaf (Table 5). The suppression was significant as late as 19 July. Moderately severe leaf rust was observed on leaves 1 and 2 of treated Favor on 13 and 19 July.

Severe storms on 5 July caused substantial lodging of the wheat. Lodging was estimated in each plot on a scale of 10 equal increments from 0% to 100%. Lodging of unsprayed and

Table 5. Effects of Tilt+ on disease and senescence in the flag leaf of various cultivars of winter wheat at Elora in 1984.

Date	Tilt	Estimated flag leaf discoloration (%)++ in the following cultivars: Augusta	Favor	Frankenmuth	Fredrick	Houser	Combined**
7 July	–	6 b*	60 b	9 b	12 b	10 b	–
	+	0 a	2 a	0 a	0 a	0 a	–
13 July	–	21	72	31	38	33	76
	+	–	–	–	–	–	5 b
19 July	–	100 b	100 b	100 b	100 b	100 b	–
	+	79 a	98 a	74 a	97 a	98 a	–

+ Tilt was applied on 21 June.
++ Data were detransformed from arcsin-transformed values.
* Values in a column for a given sampling date and followed by the same letter are not significantly different (P = 0.05, Duncan's new multiple range test).
** Combined data for all cultivars.

Table 6. Yield response of five winter wheat cultivars to a single application of Tilt at Elora in 1984.

Fungicide Spray	1000-kernel wt (g)+ of the following cultivars: Augusta	Favor	Frankenmuth	Fredrick	Houser
Check	33.6 a++	34.9 a	34.6 a	38.3 a	34.6 a
Tilt	38.1 b	36.1 a	37.6 b	37.9 a	37.8 b

+ Adjusted to 14.8% moisture content.
++ Numbers in a column followed by the same letter are not significantly different (P = 0.05, Duncan's new multiple range test).

sprayed wheat of individual cultivars did not differ significantly, and averaged as follows: Favor 65%, Augusta 40%, Fredrick 25%, Frankenmuth 22% and Houser 4%.

Tilt significantly promoted yields, estimated as 1000-kernel weights, in Augusta, Frankenmuth and Houser but not in Favor or Fredrick (Table 6). Yield promotion was greater in Augusta (13.4%) than in Houser (9.2%) or Frankenmuth (8.6%).

Discussion

The epidemic patterns of the foliar diseases, especially powdery mildew and leaf rust, varied widely among the winter wheat cultivars. Observations in 1984 of severe powdery mildew on leaf 5 and, later, the spikes of Augusta wheat underscored the potential in this cultivar for mildew epidemics of long duration. Favor showed high susceptibility to powdery mildew in the lower canopy before anthesis and to leaf rust in the upper canopy after anthesis. Rapid development of leaf rust may have precluded progress of powdery mildew in the uppper canopy. Frankenmuth ranked closely to Fredrick in exhibiting moderate resistance to powdery mildew when assessed on 13 June but later showed less mildew than Fredrick. Resistance of Frankenmuth to leaf rust was intermediate to the moderately resistant Fredrick and the highly resistant Augusta. Houser showed remarkably strong resistance to the identified race of *E. graminis* var. *tritici* and high resistance to the prevalent races of leaf rust. The leaf chlorosis and necrosis observed mainly in Fredrick and Favor warrant study to determine the etiology of these symptoms.

Observations in 1983 indicated that tan spot and the septoria blotches progressed at similar rates in the various cultivars. Leaf discoloration associated with all three diseases increased at similar rates before mid-June, while after mid-June, r-values for pycnidial intensities of *M. graminicola* and *L. nodorum* were not significantly different among the cultivars. Significantly higher pycnidial intensities in Houser than in other cultivars during June possibly arose from earlier initiation, or rapid initial progress, of epidemics of the septoria blotches in this cultivar.

Disease patterns on the upper leaves and spikes of the various cultivars in 1984 pointed to possible disease interactions. The septoria blotches progressed to moderately intense levels only when leaf rust and powdery mildew were mild or absent. Intensities of the septoria blotches were low on leaves and spikes of Favor, in which leaf rust was severe, and on the leaves of Fredrick, in which leaf rust and powdery mildew were well-developed. Spikes of Augusta bearing substantial powdery mildew showed only trace amounts of septoria nodorum blotch. On the other hand, septoria nodorum blotch developed substantially on spikes of Frankenmuth and Houser where not more than traces of rust and mildew were observed. Precolonization of wheat tissues by the biotrophic pathogens apparently restricted or precluded colonization by the necrotrophs.

Tilt was highly effective for managing the various foliar diseases except for the chlorosis and necrosis of unknown etiology, as was found in earlier studies (Sutton 1985). In 1983, the sequence of three applications, with the final spray on 22 June, protected the wheat effectively against the septoria blotches until the upper leaves and spikes senesced 3 to 4 weeks later. In 1984, the single spray of 21 June protected all cultivars until senescence except Favor. The development of leaf rust on Favor about 2 to 3 weeks after Tilt was applied probably was related to rapid multiplication of the pathogen (high R_c, Zadoks and Schein 1979), as observed in other susceptible cultivars (Shaner and Hess 1978). Epidemic rates of leaf rust also may accelerate rapidly when residual activity of the fungicide declines (Sutton and Steele 1983).

Tilt significantly increased yields of all cultivars except Fredrick. The fungicide increased yields of Augusta, Frankenmuth and Houser both in 1983 and 1984, and of Favor in 1983. Increase in total grain yield in 1983 was related to numbers of spikes/m^2 but not to increased 1000-kernel weights. Sparsity of rainfall and high temperatures severely stressed the wheat after anthesis in 1983 and probably restricted grain filling. The failure of Tilt to promote yield of Favor in 1984 probably was related to the severe lodging of this cultivar.

The diverse patterns of disease and yield responses to Tilt observed in the various cultivars provide some rationale for fungicide use. The value of fungicide usage clearly was related to the cultivar. The poor yield responses of Fredrick supported earlier conclusions (Sutton and Roke 1984) that fungicide sprays may not be justified in this cultivar. Sprays targeted against powdery mildew are more likely to be justified in Augusta and Favor than in more resistant cultivars. Fungicide timed at anthesis (Sutton 1985) may be warranted in responsive cultivars susceptible to the septoria blotches or leaf rust. The economic benefits of the anthesis spray, however, may depend heavily on weather affecting disease and crop during the grain-filling period.

Acknowledgements

This research was funded by the Natural Sciences and Engineering Research Council (strategic grant no. 0799) and by the Ontario Ministry of Agriculture and Food. It is a pleasure to thank J. Menzies for identifying the race of *E. graminis* var. *tritici*, R. Turek and N.J. Sutton for assistance in disease assessments and L.A. Hunt for providing assistance in planting and harvesting.

Literature cited

1. Bailey, K. and B.H. MacNeill. 1983. Virulence of *Erysiphe graminis* f. sp. *tritici* in southern Ontario in relation to vertical genes for resistance in wheat. Can. J. Plant Pathol. 5:148-153.
2. Horsfall, J.G. and E.B. Cowling. 1978. Phytopathometry: the measurement of plant disease. Pages 119-136 in J.G. Horsfall and E.B. Cowling, eds. Plant disease. An advanced treatise. Vol. II. Academic Press, New York. 436 pp.
3. Shaner, G. and F.D. Hess. 1978. Equations for integrating components of slow leaf-rusting resistance in wheat. Phytopathology 68:1464-1469.
4. Steel, R.G.D. and J.H. Torrie. 1980. Principles and procedures of statistics 2nd ed. McGraw Hill, Inc., New York. 633 pp.
5. Sutton, J.C. 1985. Effectiveness of fungicides for managing foliar diseases and promoting yields of Ontario winter wheat. Phytoprotection 66:141-152.
6. Sutton, J.C. and G. Roke. 1984. Relationships of cultivar, sowing date, and fungicide treatment to progress of septoria tritici blotch and septoria nodorum blotch in Ontario winter wheat. Proc. Br. Crop Prot. Conf. Pests Dis. Vol. 1:121-126.
7. Sutton, J.C. and P. Steele. 1983. Effects of seed and foliar fungicides on progress of net blotch and yield in barley. Can. J. Plant Sci. 63:631-639.
8. Zadoks, J.C. and R.D. Schein. 1979. Epidemiology and plant disease management. Oxford University Press, New York. 427 pp.
9. Zadoks, J.C., T.T. Chang, and C.F. Konzak. 1974. A decimal code for the growth stages of cereals. Weed Res. 14:415-421.

Albugo candida on *Raphanus sativus* in Saskatchewan[1]

G. A. Petrie

From 1975 to 1983, *Albugo candida* (white rust and staghead) was found on a number of occasions in garden plantings of radish (*Raphanus sativus*) at Saskatoon. Radish has not been reported previously as a host of *A. candida* in Saskatchewan. The symptoms observed are described and illustrated. Eleven cultivars of radish were successfully inoculated. Chinese Rose Winter, Round Black Spanish, and Burpee White exhibited some resistance to white rust. Seed-borne oospores were identified as a possible cause of primary infections. Oospores from hypertrophied radish inflorescences were successfully germinated in the laboratory. The potential significance of radish white rust to rapeseed production in western Canada is discussed.

Can. Plant Dis. Surv. 66:2, 43-47, 1986.

De 1975 à 1983, on a remarqué *Albugo candida* à un certain nombre d'occasion dans des semis de radis (*Raphanus sativus*) dans des jardins de la région de Saskatoon. C'est la première fois qu'on signale en Saskatchewan la présence de *A. candida* sur des plants de radis. Les symptômes observés sont décrits et illustrés dans cet article. On a inoculé avec succès onze cultivars de radis et les cultivars Chinese Rose Winter, Round Black Spanish et Burpee White ont montré une certaine résistance à la rouille blanche. Les oospores transmis par la semence ont été identifiés comme une cause possible d'infection primaire. La germination en laboratoire d'oospores provenant d'inflorescences de radis hypertrophiés a été effectuée avec succès. On discute de l'importance du danger potentiel de la rouille blanche du radis sur la production du colza dans l'ouest du Canada.

Introduction

Conners (3) listed 26 species of Cruciferae as hosts of *Albugo candida* (Pers. ex Lév.) Ktze. (*A. cruciferarum* S.F. Gray) in Canada, including eight commonly cultivated *Brassica* species. Three reports were cited of *A. candida* on radish (*Raphanus sativus* L.) in Manitoba, but there was none from Saskatchewan or Alberta.

By 1971, white rust or staghead was an important disease in commercial crops of turnip rape (*Brassica campestris* L.) in western Canada (1,2,4). In many fields, substantial yield losses resulted from high incidences of sytemic infections of inflorescences ("stagheads") (4). Following the licensing of the resistant cultivar Tobin in 1981 (7), white rust quickly declined in importance on turnip rape. It is still an important disease of Brown and Oriental condiment mustard (*Brassica juncea* (L.) Coss).

White rust can reduce radish seed yields appreciably through destruction of flowers (6). Therefore, although it is of little consequence to gardeners, the disease is of some importance to radish seed producers. In addition, the white rust biotype on *R. sativus* could conceivably infect cultivars or breeding lines of *B. campestris* (10), thus again jeopardizing the canola (rapeseed) industry in western Canada.

From 1975 to 1983, white rust was found on a number of occasions in garden plantings of radish at Saskatoon. This paper describes the symptoms observed, examines the possibility that seed-borne oospores were an important source of primary inoculum, and presents the results of inoculation experiments involving a number of cultivars of *R. sativus*.

[1] *Contribution No. 917, Agriculture Canada Research Station, 107 Science Crescent, Saskatoon, Saskatchewan S7N 0X2.*

Accepted for publication February 11, 1986

Materials and methods

In order to determine whether oospores carried with the seed might be responsible for the white rust outbreaks in home gardens at Saskatoon, 25 commercial seed samples of *R. sativus* were examined for oospores using a seed washing technique (5) (Table 1). Nine samples were purchased and tested in 1976 seven in 1984, and nine in 1985. In addition, attempts were made to germinate oospores from hypertrophied radish inflorescences collected in 1975 and 1976. Within a year of being collected, ripe hypertrophies were ground to powder by rubbing them on sandpaper. Small quantities of the powder, which consisted largely of oospores, were then washed in sterile tap water in Erlenmeyer flasks on a rotary shaker (9). After this treatment, spores were recorded as unchanged, activated, or fully germinated. A spore was considered to have been activated if the central oil droplet had disappeared and the contents of the spore were coarsely granular. Empty spores were considered to have germinated. One hundred and fifty untreated spores from each original sample were examined to determine whether any spontaneous changes had occurred. Adjustments in the totals in the three categories were made accordingly.

Eleven radish cultivars were inoculated with zoospores from zoosporangia, including two lots each of cultivars Round Black Spanish, Chinese Rose Winter, and Burpee White, from seed from different sources (Table 3). The cultivar Raoula, an oilseed radish introduction, was one of the eleven. Plants were grown, 10 per 9.5 cm pot, in Cornell soilless mix (8) at 21°C under 18 h illumination (17220 lux). There were six replications per cultivar. Zoosporangia were scraped from infected leaves, suspended in deionized water in Petri dishes, and incubated at 10°C for 3-4 h to induce germination. A zoospore suspension containing 25,000 or more spores/ml was sprayed on 10- to 14-day-old plants. Inoculated plants were kept under continuous mist for 18 h in darkness at 20°C. Eight to 10 days after inoculation, the number of infected plants per entry was recorded along with infection severity on the most heavily infected leaf on each plant. Severity ratings reflected relative number and size of pustules and were: 0 (no sporulation), 1 (trace), 3 (light), 6 (moderate), and 9 (heavy). The mean ratings for diseased plants alone and diseased plus healthy plants were calculated.

Table 1. Extent of infestation by oospores of *Albugo candida* of commercial radish seed samples purchased and tested in 1976, 1984 and 1985.

Cultivar	Year tested and number oospores per gram of seed 1976	1984**	1985
Cherry Belle			5.4
Comet		+	0.0
Champion	0.0	0	8.6
French Breakfast	0.0	0	0.2
Sparkler	0.3		0.2
White Icicle	0.0		0.4
Chinese White Winter		+	
Burpee White		+	0.4
Chinese Rose Winter			0.0
Round Black Spanish		0	0.8
Crimson Giant	1.7		
Scarlet Globe 1	13.0		
Scarlet Globe 2	0.3		
Scarlet turnip white tip sparkler	3.4		
Scarlet white tip	0.0	+	
Mean spores/g seed	2.1		1.8
Mean % samples infested	55.6	57.1	77.8

** Ten 50X microscope fields examined per slide per sample. + = oospores present at <1 per field, 0 = oospores not seen. Oospore numbers in 1976 and 1985 samples were determined by counting all oospores present on cleared filtration discs (5).

Results

Symptoms observed on radish in Saskatoon collections. Abundant infections usually were observed on radish staring in mid-July. They consisted of sporangial pustules on the leaves, spindle-shaped stem galls, floral hypertrophies, hypertrophy of entire inflorescences or terminal parts of inflorescences, and pod infections (Figs. 1 and 2). Zoosporangial pustules frequently covered hypertrophied floral organs.

Presence of oospores in commercial seed samples of radish. Oospores were recovered from 16 of the 25 samples (64%) (Table 1). They were found in 55.6% of the samples purchased in 1976, 57.1% of those from 1980, and 77.8% of those from 1985. However, the number of oospores per seed sample was small with no more than a few recovered per gram of seed.

Germination of oospores from radish. Changes occurred in over 50% of the spores in both samples (Table 2). Spores in the 1975 sample germinated more slowly than those from 1976. Only 4.6% of the former germinated completely in six days, as opposed to 48% of the latter. However, 50% of the 1975 spores exhibited internal changes in six days, which indicated that the germination process had been initiated. Germination was largely by the sessile vesicular method, one of three different modes of germination previously noted in *A. candida* from rapeseed (9).

Inoculation of radish cultivars. The cultivars Chinese Rose Winter and Round Black Spanish had substantial numbers of "clean" plants, whereas the other cultivars had none or very few (Table 3). The overall white rust severity ratings for these two cultivars were therefore the lowest, followed by the ratings for Burpee White. However, although white rust incidence was high on Burpee White, infected plants of this cultivar were less severely diseased than infected plants of all other cultivars in that the rust pustules were generally smaller.

Discussion

Radish seed sown in western Canada is imported from the United States. The results show that oospores of white rust are continually reintroduced into Canada on seed. Although circumstantial evidence points to this as a source of primary infections in this country, the levels of oospore infestation were low. Also, the inoculum threshold required for infection by oospores is unknown.

Table 2. Germination of oospores of *Albugo candida* from *Raphanus sativus* following washing in sterile tap water.[1]

Year Oospores Collected	Number Counted	Percentage of oospores: Unchanged	Activated[2]	Fully Germinated
1975	150	45.4	50.0	4.6
1976	100	42.0	10.0	48.0

[1] Five days on shaker and one day in still culture.
[2] Central oil droplet gone and contents coarsely granular.

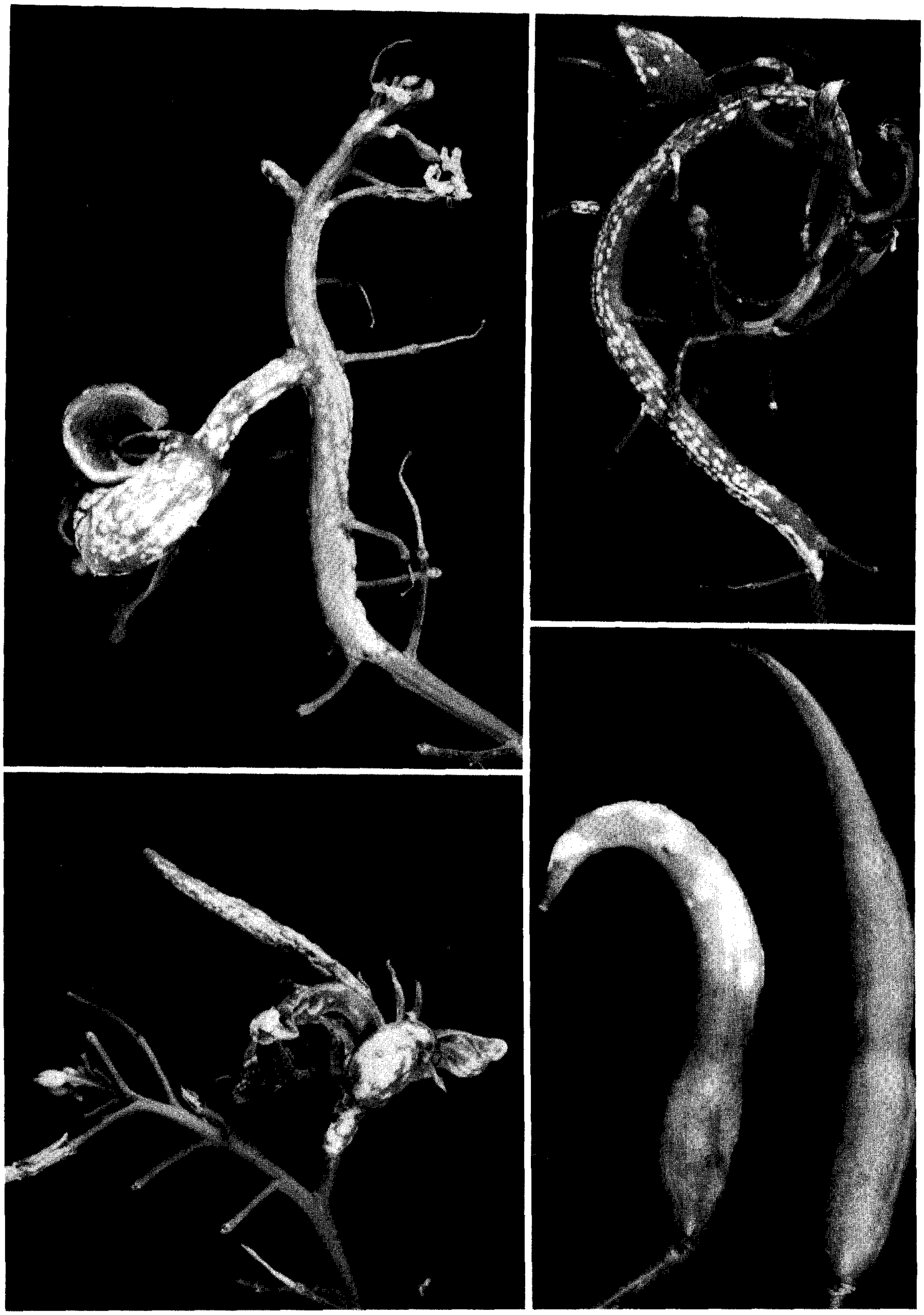

Figure 1. *Albugo candida* on *Raphanus sativus*. Top: two systemically infected peduncles; Bottom right: infected (left) and healthy pods; Bottom left: infection of an individual flower. Note white sporangial pustules on the surfaces of infected parts.

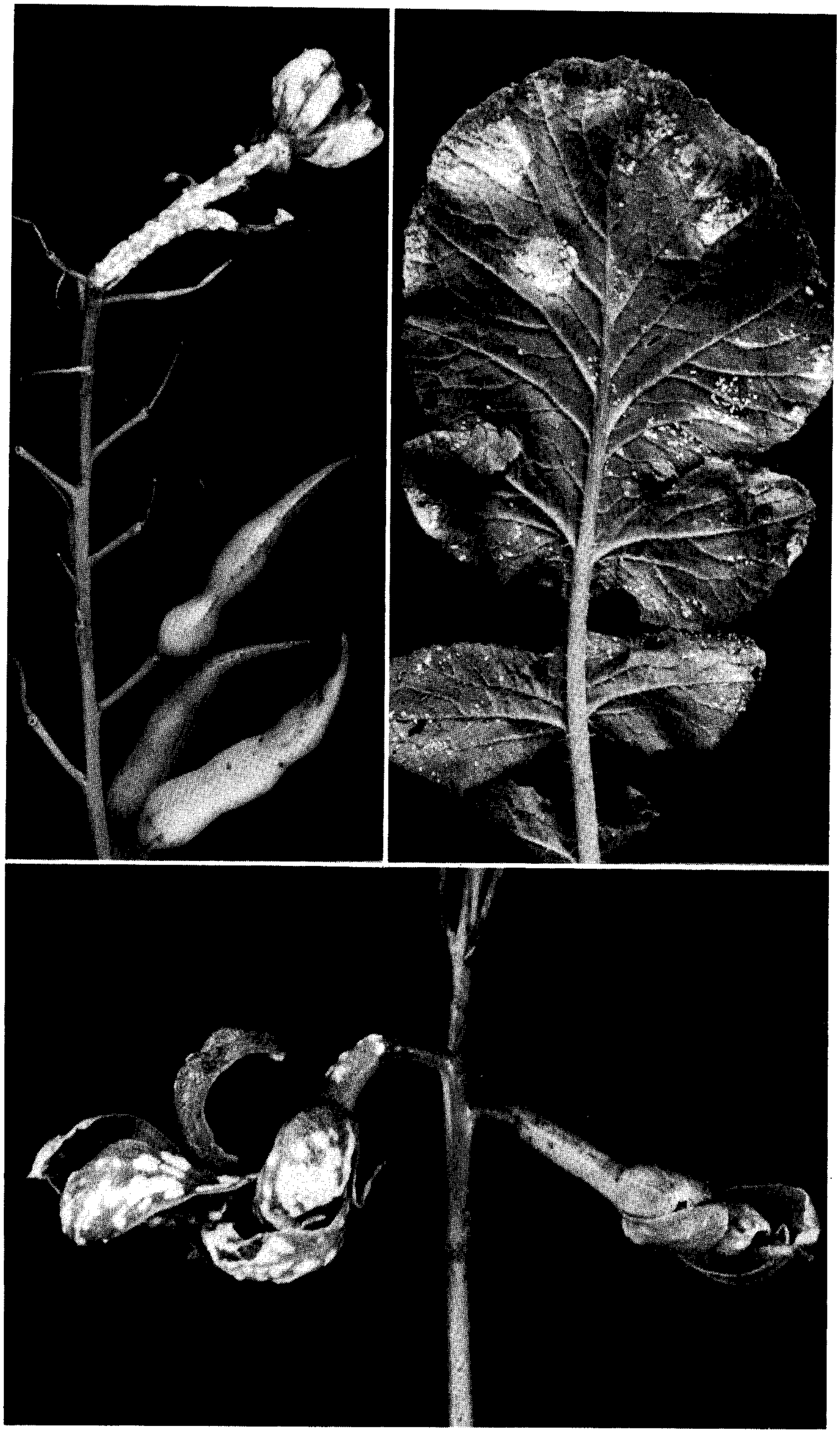

Figure 2. *Albugo candida* on *Raphanus sativus.* Clockwise from upper left: systemic infection of the upper part of a peduncle; sporangial pustules on a leaf; two infected flowers.

Table 3. Incidence and severity of white rust on eleven radish cultivars in an inoculated test.

Cultivar	Mean number[2] plants/10 infected	Mean severity[2] rating (0-9) all plants	Mean severity rating (0-9) infected plants
Cherry Belle	10.0 a	5.95 a	5.95
Comet	10.0 a	5.95 a	5.95
Champion	10.0 a	5.90 a	5.90
French Breakfast	10.0 a	5.75 a	5.75
Sparkler	10.0 a	5.55 a	5.55
Raoula	10.0 a	5.55 a	5.55
White Icicle	10.0 a	5.23 ab	5.23
Chinese White Winter	9.5 a	4.80 abc	4.88
Burpee White (S)[1]	10.0 a	4.17 bc	4.17
Burpee White (E)[1]	9.8 a	3.80 cd	3.87
Chinese Rose Winter (E)	5.7 b	2.75 de	4.92
Chinese Rose Winter (S)	5.8 b	2.68 e	4.68
Round Black Spanish (S)	4.7 bc	2.50 e	5.45
Round Black Spanish (E)	3.8 c	2.08 e	5.07

[1] (S) and (E) denote different seed sources.
[2] Means followed by the same letter within a column do not differ significantly according to Duncan's Multiple Range Test (P = 0.01).

Germination of *A. candida* oospores from radish has not been reported previously. It confirms earlier findings (9) that high percentages of oospores in many collections of *A. candida* germinate in a relatively short time when leached in water. The results of inoculation trials are in general agreement with an earlier study (11) which also reported that the cultivars Round Black Spanish and China Rose Winter contained a proportion of resistant plants.

As severe natural infections were observed only on radish which had been allowed to go to seed, white rust normally would be of no practical significance on this minor garden crop in Saskatchewan. It could, however, become important if susceptible introductions of *B. campestris* (10) were used in canola breeding programs. Care should be taken to guard against this occurring. Host specialization of collections of white rust from Saskatchewan, including that from radish, will be considered in another paper.

Acknowledgements

The author thanks Maurice Bahrey, Patricia Lewis and Marjorie Richardson for their assistance.

Literature cited

1. Berkenkamp, B. 1972. Diseases of rapeseed in central and northern Alberta in 1971. Can. Plant Dis. Surv. 52:62-63.
2. Bernier, C.C. 1972. Diseases of rapeseed in Manitoba in 1971. Can. Plant Dis. Surv. 52:108.
3. Conners, I.L. 1967. An annotated index of plant diseases in Canada. Can. Dept. Agric. Publ. 1251. 381 pp.
4. Petrie, G.A. 1973. Diseases of *Brassica* species in Saskatchewan, 1970-72. I. Staghead and aster yellows. Can. Plant Dis. Surv. 53:19-25.
5. Petrie, G.A. 1975. Prevalence of oospores of *Albugo cruciferarum* in *Brassica* seed samples from western Canada, 1967-73. Can. Plant Dis. Surv. 55:19-24.
6. Pound, G.S. and P.H. Williams. 1963. Biological races of *Albugo candida*. Phytopathology 53:1146-1149.
7. Research Branch Report. 1981. Agriculture Canada Research Station, Saskatoon Saskatchewan. p. 306.
8. Stringam, G.R. 1971. Genetics of four hypocotyl mutants in *Brassica campestris* J. Hered. 62:248-250.
9. Verma, P.R. and G.A. Petrie. 1975. Germination of oospores of *Albugo candida*. Can. J. Bot. 53:836-842.
10. Williams, P.H. 1962. Variability in the host-parasite relationships of *Albugo candida* (Pers.) Kuntze. Ph.D. Thesis, Univ. of Wisconsin, Madison.
11. Williams, P.H. and G.S. Pound. 1963. Nature and inheritance of resistance to *Albugo candida* in radish. Phytopathology 53:1150-1154.

Incidence of soybean mosaic virus and tobacco ringspot virus in southwestern Ontario

J.C. Tu[1]

A survey was conducted in eight southwestern Ontario counties (i.e. Essex, Kent, Elgin, Lambton, Oxford, Middlesex, Perth and Huron) in the summer of 1979 to 1981 to determine the incidence of soybean mosaic diseases. The overall incidence (3-year average) of mosaic diseases for the eight counties was 1.3%, Essex and Kent counties had total mosaic disease incidence of 1.65% compared to the total disease incidence for the other six counties of 0.83%. The average incidence of TRSV-disease was 0.67% and that of SMV-disease was 0.50%. Of the 265 mosaic samples collected, 135 and 102 were TRSV and SMV, respectively. The remaining 28 were other viruses.

Can. Plant Dis. Surv. 66:2, 49-50, 1986.

Huit comtés du sud-ouest de l'Ontario, soit Essex, Kent, Elgin, Lambton, Oxford, Middlesex, Perth et Huron, ont fait l'objet d'enquêtes durant l'été de 1979 à 1981, pour déterminer l'incidence de la mosaïque du soja. L'incidence totale (moyenne sur 3 ans) de la mosaïque pour les huit comtés a été de 1,3 % et de 1,65 % pour les comtés d'Essex et de Kent comparativement à 0,83 % pour les six autres comtés. L'incidence moyenne du virus de la tache annulaire du tabac a été de 0,67 % et celle du virus de la mosaïque du tabac de 0,50 %. Des 265 échantillons de mosaïque prélevés, 135 contiennent le virus de la tache annulaire du tabac et 102 le virus de la mosaïque du tabac. Les 28 échantillons restant contiennent d'autres virus.

Introduction

Ontario is the only province where a large acreage of soybeans is grown. In 1984, the acreage was over 1 million acres of which 85% was in southern Ontario (1). In 1984, Ontario soybeans had a total farm value of 257 million dollars. The soybeans produced in Ontario were insufficient for domestic consumption. Thus, some soybeans were imported to cover the shortfall. In addition, emphasis has been placed on the export market that requires blemish-free seed coats for tofu and other food products. Thus diseases which are detrimental to yield and quality should be controlled. One of these is soybean mosaic.

Soybean mosaic can be caused by several viruses, of those, only soybean mosaic virus (SMV) and tobacco ringspot virus (TRSV) are important in this region (6). At present, none of the registered Ontario cultivars are resistant to SMV and TRSV. The diseased plants produce very few seeds and are often mottled and mostly immature. If a very conservative 1% loss is assumed by these diseases, a yearly loss of 2.5 million dollars can be estimated. Seed coat mottling can reduce the grade and restrict the uses of the crop.

In addition, soybean virus diseases are very prevalent at the research station. Virus infection interferes considerably with soybean experiments and seed production. Considerable labour and funds are expended each year in efforts to control these diseases.

This paper reports the incidence of SMV and TRSV in the eight southwestern Ontario counties.

[1] *Agriculture Canada, Research Station, Harrow, Ontario N0R 1G0.*
Accepted for publication February 27, 1986

Materials and methods

The incidence of soybean mosiac was surveyed in the summers of 1979, 1980 and 1981 in the eight southwestern Ontario counties (Essex, Kent, Elgin, Lambton, Oxford, Middlesex, Perth and Huron). Each county was subdivided arbitrarily into 10 areas, and 2 fields/area were surveyed. For each field, 20 rows were randomly selected and plants in a 6-meter length of a row were examined for signs of bud blight and mosaic. The percentage of plants with the mosaic diseases was calculated from the actual plant counts in the 20, 6-meter rows.

As for collection of mosaic samples, one sample/area was made from all counties except Kent, where 2 samples/area were collected because the disease incidence was approximately doubled that of the other 7 counties. All samples were individually placed in a plastic bag, tagged and transported in an ice chest. A total of 100 samples were collected each year for a period of 3 years. Of the 300 samples, 265 were successfully transferred to Amsoy 71 seedlings in a 21 ± 3°C greenhouse by mechanical inoculation as detailed previously (5).

Identification of TRSV isolates was based on their ability to infect at least 4 out of 5 of the diagnostic hosts: *Chenopodium amaranticolor, Cucumis sativus, Phaseolus vulgaris* cv. Bountiful, *Vigna sinensis* cv. Blackeye Ramshorn and *Nicotiana tabacum* cv. Samsun NN (Gibbs *et al.* 1970); while some isolates of SMV can infect only *P. vulgaris* cv. Bountiful (2).

Results and discussion

The incidence of SMV and TRSV disease in the eight southwestern Ontario counties are summarized in Table 1. There were significantly higher incidences of these virus diseases in Essex and Kent. Essex and Kent had a disease incidence of 1.6 and 1.7, respectively, for the overall 3-year average, while the other six counties had a disease incidence of 0.83%. The average incidence of mosaic diseases in Essex and Kent counties

Table 1. Average incidences (1979-81) of soybean mosaic virus- and tobacco ringspot virus-disease in the eight southwestern Ontario counties.

County	Disease Incidence (%) TRSV	SMV	Total
Elgin	0.6 ± 0.1 a	0.3 ± 0.2 a	0.9
Essex	1.0 ± 0.2 b	0.6 ± 0.1 b	1.6
Kent	1.2 ± 0.1 c	0.5 ± 0.1 ab	1.7
Huron	0.5 ± 0.1 a	0.4 ± 0.2 ab	0.9
Lambton	0.4 ± 0.1 a	0.3 ± 0.1 a	0.7
Middlesex	0.5 ± 0.1 a	0.4 ± 0.1 ab	0.9
Oxford	0.5 ± 0.1 a	0.3 ± 0.1 a	0.8
Perth	0.5 ± 0.1 a	0.3 ± 0.1 a	0.8

a-c Means in each column with the same letter are not significantly different ($P > 0.05$).

was twice as high as the average of the six other counties. This difference is attributed to the fact that Essex and Kent counties have a longer and warmer growing season and a greater diversity of vegetable crops. The combination of climatic and vegetative factors may flavor the population dynamics of the vectors.

The average incidence of TRSV-disease (0.67%) is higher than that of SMV-disease (0.50%). Both TRSV and SMV are seed transmittible in soybeans. Seeds from SMV diseased plants are generally mottled (4) but those from TRSV diseased plants are not. Thus, SMV diseased seeds can be easily separated from seed lots and consequently reduce its incidence in the field. Of 265 mosaic samples collected, 135 and 102 were categorized as TRSV and SMV, respectively. The remaining 28 had other viruses.

The incidence of TRSV reported here is higher than the 0.15-0.52% reported by Hildebrand and Koch (3) for Ontario. This could be that the incidence of TRSV has increased over time or that Hildebrand and Koch may have underestimated the disease incidence since their detection was based solely on the bud blight whereas strains of TRSV that did not cause bud blight were not accounted for (6).

Literature cited

1. Agriculture Statistics for Ontario. Ontario Ministry of Agriculture and Food 1984. Publication 20. Toronto, Ontario. pp. 80.
2. Harrison, B.C. and A.F. Murant. 1972. Descriptions of plant viruses. Set 5, No. 93. Commonw. Mycol. Inst. and Assoc. Appl. Biol.
3. Hildebrand, A.A. and L.W. Koch. 1947. Observations on bud blight of soybeans in Ontario. Sci. Agr. 27:314-321.
4. Kennedy, B.W. and R.L. Cooper. 1967. Association of virus infection with mottling of soybean seed coats. Phytopathology 57:35-37.
5. Tu, J.C. 1983. Inheritance in *Phaseolus vulgaris* cv. Kentwood of resistance to a necrotic strain of bean yellow mosiac virus and to a severe bean strain of tobacco ringspot virus. Can. J. Plant Pathol. 5:34-35.
6. Tu, J.C. 1986. Strains of tobacco ringspot virus isolated from soybean in southwestern Ontario. Can. Plant Sci. (in press).

Blackleg and other diseases of canola in Saskatchewan in 1984 and 1985[1]

G.A. Petrie

In 1984 and 1985, basal stem canker caused by *Leptosphaeria maculans* was severe on canola (*Brassica napus* and *B. campestris*) in central Saskatchewan (crop districts 6 and 8B), the area in which it traditionally has been most prevalent. In 1984, yield losses were greatly increased by premature ripening of cankered plants by hot dry summer weather. In 1984, 28.5% of 151 fields surveyed in Saskatchewan had plants with basal stem infections, and in 1985, 43.0% of 142 fields had stem-cankered plants. Mean percent of plants per field with infections on leaves or stems was 13.1 in 1984 and 10.9 in 1985. Mean basal stem canker incidence was 9.1% in 1984 and 7.2% in 1985. The average estimated yield loss provincially was 7.2% in 1984 and 5.2% in 1985. Infection levels were very low in northern areas where much of the canola is grown. The mean yield loss in infected fields was 25.2% in 1984 and 12.2% in 1985. In both years diseases other than blackleg were of little importance in these surveys.

Can. Plant Dis. Surv. 66:2, 51-53, 1986.

En 1984 et 1985, la jambe noire causée par *Leptosphaeria maculans* a beaucoup affecté le canola (*Brassica napus* et *B. campestris*) dans le centre de la Saskatchewan (régions agricoles 6 et 8B), région où elle a toujours été plus importante. En 1984, les pertes de rendements ont été beaucoup plus considérables en raison de la maturation prématurée des plants à chancre causée par la température estivale chaude et sèche. En 1984, 28,5 % des 151 champs inventoriés en Saskatchewan contenaient des plants infectés par la jambe noire comparativement à 43 % des 142 champs inventoriés en 1985. En moyenne, il y avait dans les champs infectés 13,1 % des plants avec des infections sur les feuilles ou les tiges en 1984 et 10,9 % en 1985. En 1984, l'incidence moyenne de chancre à la base de la tige était de 9,1 % et, en 1985, de 7,2 %. En 1984, on estimait la perte de rendement moyenne à l'échelle provinciale à 7,2 % et en 1985 à 5,2 %. Dans les régions nordiques où l'on cultive beaucoup de canola, les taux d'infection étaient peu élevés. En 1984, on a estimé la perte de rendement dans les champs infectés à 25,2 % et, en 1985, à 12,2 %. Durant ces deux années, les enquêtes ont montré que les maladies autres que la jambe noire étaient peu importantes.

Introduction

Between 1978 and 1981 the prevalence and incidence of the virulent strain of blackleg (*Leptosphaeria maculans* (Desm.) Ces. & de Not.) increased ten-fold in standing Saskatchewan crops of rapeseed/canola (*Brassica napus* L. and *B. campestris* L.) (6). However, the province-wide average yield loss from basal stem canker was slight. In 1982, basal stem cankers were prevalent in several fields in central crop districts (C.D.) 6 and 8 (4). In 17 heavily infected fields, yield reductions ranged from 0 to 56%. Despite this, the average yield loss for Saskatchewan as a whole was estimated at 6%, due to low infection levels in northern and western areas. Although blackleg was prevalent again in 1983, yield losses of the magnitude seen in 1982 were not observed (5).

During the period 1978 to 1983, the prevalence and incidence of footrot (*Rhizoctonia solani* Kühn and *Fusarium roseum* Lk. emend. Snyder and Hansen) were highest in 1979 and 1982, with respectively, 80.7 and 79.3% of the fields infected and incidences of 16.0 and 8.5% (4, 6). Even in 1978, when the disease was least prevalent, infections were noted in over 40% of the fields and the mean incidence was 6.7%. Other diseases, with the exception of sclerotinia stem rot (*Sclerotinia sclerotiorum* (Lib.) de Bary), were of minor importance from 1978 to 1983 (4, 5, 6). The following is a report of a disease survey of canola conducted in Saskatchewan in 1984 and 1985.

[1] *Contribution No. 921, Agriculture Canada Research Station, 107 Science Crescent, Saskatoon, Saskatchewan S7N 0X2.*

Accepted for publication March 11, 1986

Methods

Fields were sampled as in the 1983 survey while the plants were in flower (5). Basal stem canker severity was assessed (1) and all plants with basal cankers were considered when estimating yield losses (2).

Results

Blackleg. In 1985, basal stem canker was more prevalent but less severe than in 1984 (Tables 1 and 2). Forty-three percent of the 1985 fields and 28.5% of those from 1984 had cankered plants. The estimated yield loss in infected fields in 1984, 25.2%, was twice that in 1985. Mean stem canker incidence and incidence of all stem and leaf infections caused by *L. maculans* were similar in the two years. As in previous years stem canker was most prevalent and severe in central Saskatchewan (C.D. 6 and 8B). There was a relatively small area of heavy infestation around North Battleford (C.D. 9A and 9B). In both years the overall blackleg incidence was low in northern areas (C.D. 8a and 9). Prevalence and incidence of the disease increased considerably in C.D. 5 in 1985 and it has now spread from that area into western Manitoba (Petrie, unpublished data).

Footrot. The prevalence and incidence of footrot were the lowest observed since 1978. Infections were detected in only 36.4% of the 1984 fields and 25.4% of the 1985 fields (Table 3). The mean incidence was 2.2% and 4.3%, respectively.

Other diseases. With a few exceptions, other diseases were not noteworthy in either year. In 1984 pod drop (cause un-

Table 1. Prevalence, incidence and severity of the virulent strain of blackleg in standing crops of canola in 1984.

Crop District	No. of fields	Percentage of fields with plants infected		Percentage of plants per field infected		% fields with over		% loss in yield*	
		at stem base	on any part	at stem base	on any part	10% blackleg	20% blackleg	all fields	infected fields only
1	10	10.0	20.0	0.4	0.8	0.0	0.0	0.4	4.0
5	20	20.0	35.0	1.2	2.8	10.0	0.0	1.0	5.0
6	33	54.6	57.6	24.1	26.5	48.5	33.3	17.0	31.2
8A	19	15.8	52.6	1.3	8.0	21.0	10.5	1.0	6.3
8B	24	50.0	66.7	24.3	27.2	45.8	37.5	17.1	34.2
8	43	34.9	60.5	14.1	18.7	34.9	25.6	10.0	28.6
9A	19	21.1	63.2	5.1	14.1	26.3	21.1	3.5	16.5
9B	26	3.9	34.6	0.2	5.9	23.1	11.5	0.2	4.0
9	45	11.1	46.7	2.2	9.3	24.4	15.6	1.6	14.0
A11	151	28.5	49.7	9.1	13.1	29.1	19.2	7.2	25.2

* Estimated according to McGee (1) and McGee and Emmett (2).

Table 2. Prevalence, incidence and severity of the virulent strain of blackleg in standing crops of canola in 1985.

Crop District	No. of fields	% fields with plants infected		% plants per field infected		% fields with over		% loss in yield*	
		at stem base	on any part	at stem base	on any part	10% blackleg	20% blackleg	all fields	infected fields only
5	27	40.7	51.9	3.9	6.2	22.2	7.4	2.6	6.9
6	27	55.6	59.3	9.3	14.0	37.0	22.2	6.8	12.3
8A	19	26.3	73.7	1.7	5.3	5.3	0.0	1.4	5.2
8B	27	63.0	74.1	16.1	23.4	51.9	33.3	11.5	18.2
8	46	47.8	73.9	10.2	15.9	32.6	19.6	7.3	15.3
9A	21	33.3	33.3	4.0	5.9	9.5	4.8	3.1	9.1
9B	21	28.6	38.1	5.3	6.9	23.8	9.5	3.8	13.3
9	42	31.0	35.7	4.7	6.4	16.7	7.1	3.4	11.1
A11	142	43.0	55.6	7.2	10.9	26.8	14.1	5.2	12.2

* Estimated according to McGee (1) and McGee and Emmett (2).

known) occurred in 42.1% of the fields in C.D. 9A (the northeast). In one field, 60% of the plants were affected, and in another, 52%. Severity generally was light. Grey stem (*Pseudocercosporella capsellae* (Ell. & Ev.) Deighton) became prevalent in northern areas as the season progressed, but again infection was light. Alternaria black spot (*Alternaria brassicae* (Berk.) Sacc. and *A. raphani* Groves & Skolko) occurred at the "trace" to "slight" level of severity on high percentages of plants in many areas except C.D. 6. White rust (*Albugo candida* (Pers. ex Lév.) Ktze) was of very minor importance as was sclerotinia stem rot. However, the survey was conducted too early to adequately assess the latter disease.

In 1985, white leaf spot (*P. capsellae*) occurred in 63% of the fields in C.D. 8A (the northeast). Only occasionally were high infection incidences noted. In a field near Medstead (C.D. 9A), 100% of the plants had leaf spots, but damage was slight. Pod drop occurred at low levels in 44% of the fields in C.D. 5 (east-central area).

Discussion

In three of the last four years, basal stem canker has caused appreciable yield loss in Saskatchewan, principally in C.D. 6 and 8 (4, 5). In 1984 *L. maculans* produced ascospores on canola stubble unusually early in the growing season, resulting in early infection of the crop (Petrie, unpublished). Subsequently, hot dry summer weather contributed significantly to widespread premature ripening of plants with basal stem infections. Although a considerable increase in the prevalence of the virulent strain of *L. maculans* was found in C.D. 5, and

Table 3. Prevalence and incidence of footrot *(Rhizoctonia* and *Fusarium)* in canola crops in 1984 and 1985.

Crop District	% fields with infection		% plants infected per field (all fields)		% plants infected per field (infected fields only)		% fields with over 10% footrot		% fields with over 20% footrot	
	1984	1985	1984	1985	1984	1985	1984	1985	1984	1985
1	30.0	–*	2.0	–*	6.7	–*	0.0	–*	0.0	–*
5	20.0	22.2	0.8	0.9	4.0	4.0	0.0	0.0	0.0	0.0
6	39.4	33.3	3.6	3.7	9.1	11.0	12.1	11.1	0.0	3.7
8	44.2	28.3	4.0	3.0	9.1	10.8	9.3	13.0	4.7	2.2
9	35.6	19.1	7.1	1.1	19.9	6.0	24.4	2.4	13.3	0.0
All C.D.	36.4	25.4	4.3	2.2	11.7	8.6	12.6	7.0	5.3	1.4

* Not surveyed.

although it has spread into Alberta and Manitoba, much of the northern growing districts (8A and 9) continue to have very little blackleg. It remains most severe in central C.D. 6 and 8B, the area in which it was first found on 1975 stubble (3). The increased severity of blackleg is attributed largely to an abundance of undecomposed canola residue bearing ascocarps of *L. maculans* (Petrie, unpublished). Crop rotations commonly used by producers are too short to permit natural destruction of this material. If rotations involving three to four years between canola crops had been widely practiced, there likely would have been appreciably less stem canker.

Acknowledgements

The assistance of Maurice Bahrey, Patricia Lewis, Timothy Klaassen, and Edward Knopf is gratefully acknowledged. Valuable support was received from an Alberta Farming for the Future grant.

Literature cited

1. McGee, D.C. 1973. Rapeseed: how to assess the severity of black leg disease and predict yield. J. Agric. (Vict.) 71:241-242.
2. McGee, D.C. and R.W. Emmett. 1977. Black leg (*Leptosphaeria maculans* (Desm.) Ces. et de Not.) of rapeseed in Victoria: crop losses and factors which affect disease severity. Aust. J. Agric. Res. 28:47-51.
3. Petrie, G.A. 1978. Occurrence of a highly virulent strain of blackleg (*Leptosphaeria maculans*) on rape in Saskatchewan (1975-77). Can. Plant Dis. Surv. 58:21-25.
4. Petrie, G.A. 1985. Yield losses in Saskatchewan rapeseed/canola crops from basal stem cankers of blackleg (*Leptosphaeria maculans*) in 1982, with notes on other diseases. Can. Plant Dis. Surv. 65:43-46.
5. Petrie, G.A. 1985. Saskatchewan rapeseed/canola disease survey, 1983. Can. Plant Dis. Surv. 65:47-49.
6. Petrie, G.A., K. Mortensen, and J. Dueck. 1985. Blackleg and other diseases of rapeseed in Saskatchewan, 1978 to 1981. Can. Plant Dis. Surv. 65:35-41.

Détection de rickettsoïdes xylémiques dans la verge d'or au Québec[1]

J.-G. Parent, S. Desjardins et J.D. Brisson

Des rickettsoïdes associés au xylème ont été détectés par microscopie électronique à transmission dans des tissus de verge d'or (*Solidago canadensis* L.). La plante était porteuse de galles apicales déformantes causées par une cécidomyie jamais rapportée au Canada, *Rhopalomyia solidaginis*. Les rickettsoïdes observés, en plus d'avoir les caractéristiques morphologiques typiques de petite taille (0.5-1.0 μm) et de paroi ondulée, contenaient des corps sphériques de nature probablement lipidique. Cet article mentionne pour la première fois la présence de rickettsoïdes xylémiques au Canada. Nous discutons aussi des effets possibles de ces organismes sur les cultures commerciales.

Can. Plant Dis. Surv. 66:2, 55-57, 1986.

Xylem-associated rickettsia-like bacteria, were detected by transmission electron microscopy in tissue from goldenrod (*Solidago canadensis* L.). Plants also bore apical galls caused by a cecidomyiid insect unrecorded in Canada, *Rhopalomyia solidaginis*. In addition to the characteristic small size (0.5-1.0 μm) and rippled cell wall, the observed rickettsia-like bacteria possessed spheric bodies, probably with a lipidic content. This publication is the first report of a xylem-associated rickettsia-like bacterium in Canada. The possible impact of these organisms on commercial crop production is also discussed.

Introduction

Les rickettsoïdes possèdent une ultrastructure de petites bactéries gram-négatives. En nature, ils sont généralement transmis par des insectes vecteurs, surtout des cicadelles. Dans les plantes hôtes, la plupart des rickettsoïdes sont associés aux tissus vasculaires, soit le xylème ou le phloème. Les rickettsoïdes xylémiques ont été observés presqu'exclusivement dans des régions tempérées comme le sud des États-Unis (Hopkins 1977). En Amérique du Nord, la distribution de la maladie de Pierce, la plus importante maladie causée par des rickettsoïdes xylémiques, est limitée au nord par les basses températures hivernales (Purcell 1979). Cette maladie, qui affecte principalement la vigne et la luzerne, n'a pas été détectée dans le nord-est des États-Unis malgré la présence de vecteurs reconnus. La découverte des rickettsoïdes comme procaryotes phytopathogènes est relativement récente (Davis et Whitcomb 1971; Giannotti *et al.* 1970; Hopkins et Mollenhauer 1973), ce qui peut expliquer qu'il n'y ait que deux cas de rapportés au Canada (Benhamou et Sinha 1981; Benhamou *et al.* 1983). Alors que ces deux mentions faisaient état de rickettsoïdes associés au phloème, nous rapportons, pour la première fois au Canada, la présence de rickettsoïdes xylémiques.

Matériel et méthodes

Durant l'été de 1984, nous avons découvert, à Sainte-Foy, Québec, 11 plants de verge d'or porteurs d'une excroissance apicale déformante semblable à des symptômes de phyllodie (Fig. 1). La dissection des spécimens nous a permis de trouver des larves de l'insecte gallicole *Rhopalomyia solidaginis* Lw. (*Diptera, Cecidomyiidae*) (Felt 1918) dans des loges à l'apex des tiges (Fig. 2). Certaines des larves étaient parasitées par *Torymus solidaginis* (Huber) Conn. (*Hymenoptera, Torymidae*). À notre connaissance, ces deux insectes n'ont jamais été rapportés au Canada (Benoit 1985).

Des portions de tiges et de pétioles de 2 plants porteurs de galles ont été prélevées et fixées à la paraformaldéhyde à 2% et à la glutaraldéhyde à 2% dans un tampon cacodylate 0.05 M, pH 7.0 pour 2 heures à 4°C, puis au tétroxyde d'osmium à 1% dans le même tampon pour 1 heure. Les tissus ont ensuite été déshydratés à l'éthanol, enrobés avec le l'«Epon» et sectionnés au couteau à diamant. Des coupes ultra-fines (50 nm) du matériel inclus ont été examinées au microscope électronique à transmission Siemens 101. Des portions d'un plant sain, traitées de façon identique, nous ont servi de matériel témoin.

Les autres plants parasités ont été examinés par microscopie en fluorescence, pour la présence de rickettsoïdes en utilisant le colorant DAPI (diamidino- 4',6 phényl-2 indole), spécifique à l'ADN, sur des coupes épaisses de tiges après une fixation à la glutaraldéhyde à 5% (Schaper et Converse 1985).

Résultats et discussion

Par étude ultramicroscopique de plants porteurs de galles, nous avons décelé des organismes de type rickettsoïde dans les cellules de xylème et du parenchyme associé au xylème (Fig. 3). Seulement trois ou quatre de ces cellules, par coupe observée, contenaient des rickettsoïdes alors qu'aucune cellule du plant témoin n'en possédait. Dans les cellules végétales envahies, les rickettsoïdes étaient nombreux et répartis uniformément, sans tendance à l'agglomération. Un diamètre d'environ 0.5 μm et une longueur variant de 0.5 à 1.0 μm donnaient aux rickettsoïdes une apparence arrondie ou bacilliforme. L'ultrastructure (Fig. 4) était typique des rickettsoïdes avec une paroi ondulée caractéristique (Fig. 5), dite de peptidoglycane (Benhamou *et al.* 1979), limitée par des membranes interne et externe. Les 2 membranes étaient doubles, constituées d'une zone translucide aux électrons entre 2 couches opaques. Des coupes tangentielles (Fig. 6) montraient que la paroi était

[1] *Contribution no. 356. Service de recherche en phytotechnie de Québec, Ministère de l'Agriculture, des Pêcheries et de l'Alimentation du Québec, Complexe scientifique, 2700 rue Einstein, Sainte-Foy, Québec, G1P 3W8.*

Accepté pour publication le 10 avril 1986

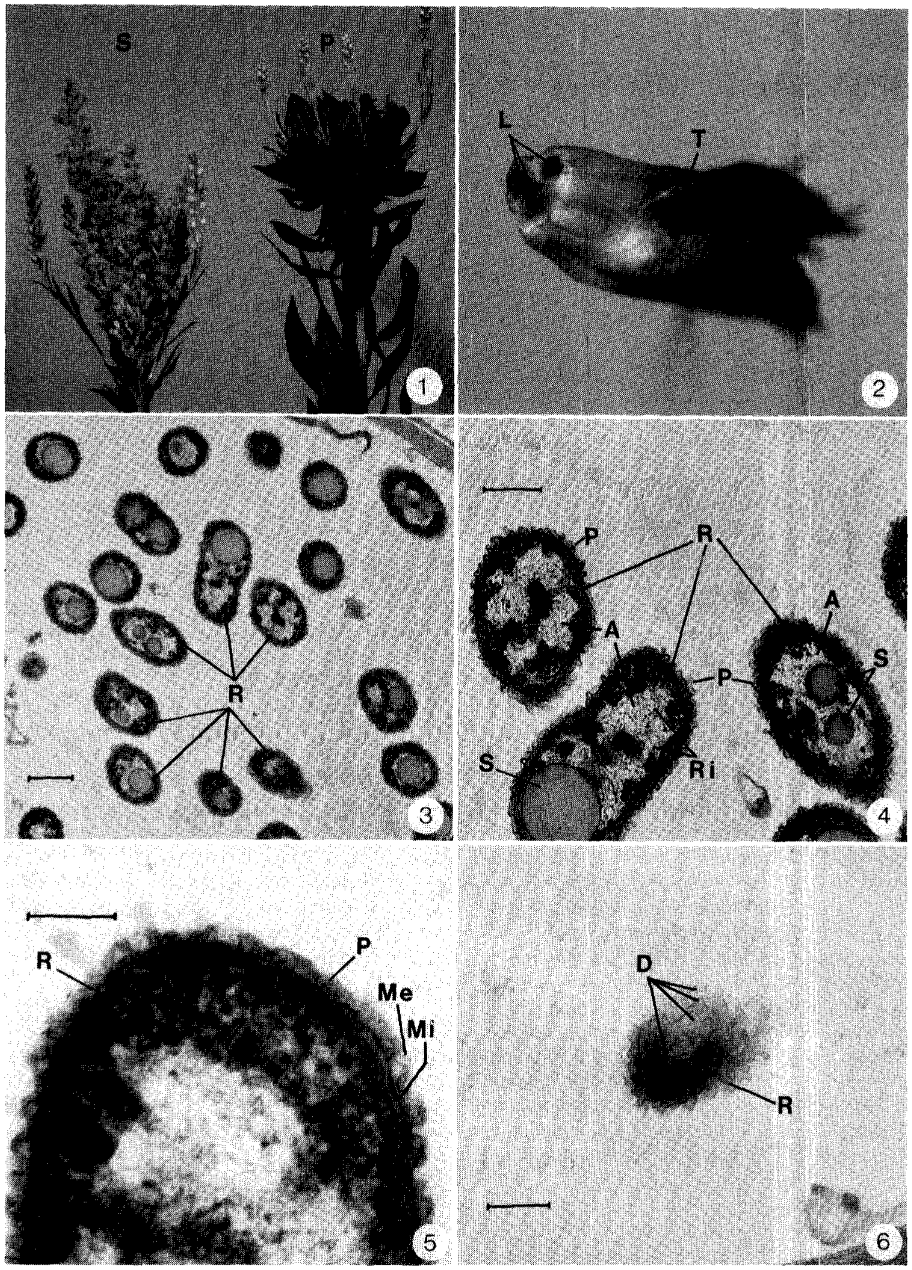

Figure 1. Comparaison des organes floraux de verge d'or saine (gauche) et parasitée par des cécidomyies *Rhopalomyia solidaginis* (droite).

Figure 2. Coupe d'une galle de *Rhopalomyia solidaginis* sur la verge d'or montrant 3 loges (L) et un trou (T) par lequel un des insectes devenu adulte s'est échappé.

Figure 3. Groupe de rickettsoïdes (R) dans une cellule du parenchyme associé au xylème. La barre représente 500 nm.

Figure 4. Ultrastructure des rickettsoïdes xylémiques (R). Les structures visibles sont la paroi ondulée (P), le treillis d'ADN (A), les ribosomes (Ri) et des corps sphériques (S) probablement lipidiques. La barre représente 250 nm.

Figure 5. Ultrastructure détaillée de la paroi d'un rickettsoïde xylémique (R). La paroi (P) est bordée par deux membranes doubles, à l'extérieur (Me) et à l'intérieur (Mi). La barre représente 100 nm.

Figure 6. Coupe tangentielle de la paroi d'un rickettsoïde (R) montrant des dépôts superposés (D) en forme de plaquettes. La barre représente 200 nm.

formée de dépôts superposés en forme de plaquettes. Le cytoplasme était divisé en zones opaques en bordure de la paroi et en zones centrales claires dans lesquelles on distinguait un treillis filamenteux, l'ADN chromosomique, et des granules, les ribosomes. Le cytoplasme pouvait aussi contenir jusqu'à 4 gros corps sphériques (0.1 à 0.4 μm de diamètre) bordés par une membrane simple. Ces corps, de nature probablement lipidique, n'ont jamais été observés dans des rickettsoïdes auparavant.

L'examen de coupes de tissus par microscopie en fluorescence, après coloration au DAPI, nous a permis de localiser des cellules à contenu cytoplasmique fluorescent dans le xylème de plusieurs plants porteurs de galles. Ce résultat suppose la présence de rickettsoïdes dans la plupart des plants infectés par les cécidomyies. Les plants témoins observés ne contenaient pas de cellules fluorescentes au DAPI.

Nos données ne nous permettent pas de conclure sur la relation entre les rickettsoïdes et les cécidomyies. L'insecte peut être vecteur ou encore leur présence simultanée peut être fortuite dans les plants observés. L'infection par les cécidomyies pourrait aussi affaiblir la plante et la rendre plus sensible à une infection par des rickettsoïdes transportés par un autre vecteur. Des relevés dans la zone où les plants porteurs de galles ont été trouvés nous ont permis d'identifier des populations d'insectes potentiellement vecteurs: *Philaenus spumarius* (Fall.) (*Hemiptera, Cercopidae*), *Clastoptera obtusa* (Say) (*Hemiptera, Cercopidae*) et *Acutalis semicrema* Say (*Hemiptera, Membracidae*).

Ce travail montre qu'il existe des rickettsoïdes xylémiques au Canada malgré les basses températures hivernales. Les plantes indigènes comme la verge d'or peuvent servir de réservoirs pour ces procaryotes dont le potentiel phytopathogène pourrait s'exprimer dans les cultures commerciales si des hôtes ou des vecteurs convenables devenaient disponibles. Des études sur la gamme d'hôtes de ces rickettsoïdes nous permettraient d'en évaluer le risque. Les rickettsoïdes peuvent être aussi utilisés comme agents de contrôle biologique des mauvaises herbes. Même si l'insecte par la galle produite, constitue un moyen d'empêcher la reproduction sexuée de la verge d'or, comme suggéré pour d'autres galles de fleurs (Shorthouse 1980), le contrôle est incomplet vu l'importance de la reproduction végétative par le rhizome. Toutefois, les rhizomes des plants parasités étaient plus courts et portaient moins de racines adventives que ceux des plants sains. Nos tentatives de culture en serre de plants porteurs de galles, après que les insectes devenus adultes se soient échappés ou après avoir enlevé les galles, ont échoué. Nous soupçonnons l'affaiblissement des organes souterrains comme responsable du manque de vigueur et de la mort des plants parasités au cours de mois suivant la mise en culture. Des plants témoins, présumément sains, ont pu être conservés dans les mêmes conditions. L'hypothèse d'une diminution de la capacité de survie à l'hiver de la verge d'or, suite à l'infection par des rickettsoïdes, reste à vérifier par des expériences dans des conditions naturelles.

Conclusion

Cette première mention, au Canada, de rickettsoïdes xylémiques, doit nous amener à porter attention à leur présence dans les cultures commerciales. Les cas de dépérissements rapides devraient particulièrement être considérés comme abritant des rickettsoïdes jusqu'à présent insoupçonnés.

Remerciements

Nous remercions madame Céline Piché du Service d'entomologie et de pathologie du ministère de l'Énergie et des Ressources du Québec pour l'identification des insectes et madame Line Pelletier pour la dactylographie du manuscrit.

Références bibliographiques

1. Benhamou, N., C. Louis et J. Giannotti. 1979. Étude par cytochimie ultrastructurale des rickettsoïdes et des cellules phloémiques de trèfles dépérissants. Ann. Phytopathol. 11:1-16.
2. Benhamou, N., G.B. Ouellette et F. Pauzé. 1983. Mise en évidence d'infections à procaryotes (rickettsoïdes et mycoplasmes) chez le *Trifolium repens* au Québec. Phytoprotection 64:53-59.
3. Benhamou, N. et R.C. Sinha. 1981. Association of a rickettsia-like organism with a disease of white clover. Can. J. Plant Pathol. 3:191-196.
4. Benoit, P. 1985. Nomenclatura insectorum canadensium. Service canadien des forêts, éd. 299 pp.
5. Davis, R.E. et R.F. Whitcomb. 1971. Mycoplasmas, rickettsiae and chlamydiae: possible relation to yellow diseases and other disorders of plants and insects. Ann. Rev. Phytopathol. 9:119-154.
6. Felt, E.P. 1918. Key to American insect galls. N.Y. State Mus. Bull. 200, 310 pp.
7. Giannotti, J., C. Vago, G. Devauchelle et J.L. Duthoit. 1970. Infections de plantes par un type inhabituel de microorganismes intracellulaires. C.R. Hebd. Séances Acad. Sci. Sér. D 271:2118-2119.
8. Hopkins, D.L. 1977. Diseases caused by leafhopper-borne rickettsia-like bacteria. Ann. Rev. Phytopathol. 17:277-294.
9. Hopkins, D.L. et H.H. Mollenhauer. 1973. Rickettsia-like bacterium associated with Pierce's disease of grapes. Science 179:298-300.
10. Purcell, A.H. 1979. Leafhopper vectors of xylem-borne plant pathogens. Pages 603-625 *dans* K. Maramorosch et K.F. Harris. éd. Leafhopper vectors as plant disease agents. Academic Press, New York, San Francisco et Londres.
11. Schaper, V. et R.H. Converse. 1985. Detection of mycoplasma like organisms in infected blueberry cultivars by the DAPI technique. Plant Dis. 69:193-196.
12. Shorthouse, J.D. 1980. Modification of the flower heads of *Sonchus arvensis* (family Compositae) by the gall former *Tephritis dilacerata* (order Diptera, family Tephritidae). Can. J. Bot. 58:1534-1540.

Dieback of white birch in central British Columbia

J.C. Hopkins and A. Funk[1]

A dieback of twigs and branches of white birch associated with a species of *Sirococcus* is described from the interior wet belt of British Columbia. This is a new host record for a *Sirococcus*.

Can. Plant Dis. Surv. 66:2, 59-60, 1986.

On décrit un dépérissement des rameaux et des branches du bouleau à papier qui serait attribuable à un *Sirococcus*, observé dans la zone pluvieuse de l'intérieur de la Colombie-Britannique. Il s'agit de la première mention d'un *Sirococcus* sur cet hôte.

Introduction

This report describes the symptoms and signs of a dieback of stems and branches of white birch (*Betula papyrifera* Marsh) occurring at several locations in the interior wet belt of British Columbia. The associated fungus has been identified as a species of *Sirococcus* which is characterized by stromatic pycnidia with branched conidiophores and phialides bearing fusoid septate conidia. This coelomycete genus is solely represented in British Columbia by *S. strobilinus* (Preuss) (2) which commonly causes a shoot tip dieback of western hemlock (*Tsuga heterophylla* (Raf.) Sarg.) and sometimes other coniferous genera, but never of a hardwood species. The disease described here bears some resemblance to the dieback of yellow birch found in eastern Canada (1) caused by *Diaporthe alleghaniensis* Arnold.

Material and methods

Following the initial collection of dieback of white birch at Summit Lake near Nakusp in 1984, birches elsewhere in British Columbia were examined for dieback by field staff of the Forest Insect and Disease Survey. Samples of suspect material were forwarded to the Pacific Forestry Centre, Victoria, for examination.

All dieback samples were scrutinized for fungal fruiting bodies which were then sectioned by hand and mounted in lactophenol containing acid fuchsin. Conidia were obtained by wetting fruiting bodies, collecting exuded spores within approximately 1 hour, and mounting them in lactophenol.

Specimens were dried and placed in the herbarium (DAVFP) at the Pacific Forestry Centre.

Results

Collections bearing fruiting bodies were made in 1984 and 1985 from the Summit Lake area where many of the birches were affected. Collections were made in 1985 from Three Forks near New Denver, Grohman Narrows Provincial Park, Nelson and Grampian Park, Nelson.

Fig. 1. Dead terminal bud of white birch with scattered pycnidia of a *Sirococcus*.

Damage consists of dead distal portions of varying length of the affected stems and branches. Specimens with intact bark and buds were presumed to have been killed in the preceding year. Other specimens with much of their bark broken off or decayed and with few if any buds remaining were presumed to have been dead for over a year.

Pycnidia containing conidia typical of the genus *Sirococcus* (4) were found only on specimens believed to be no more than

[1] *Agriculture Canada, Canadian Forestry Service, Pacific Forestry Centre, 506 West Burnside Rd., Victoria, B.C. V8X 1M5.*

Accepted for publication April 28, 1986

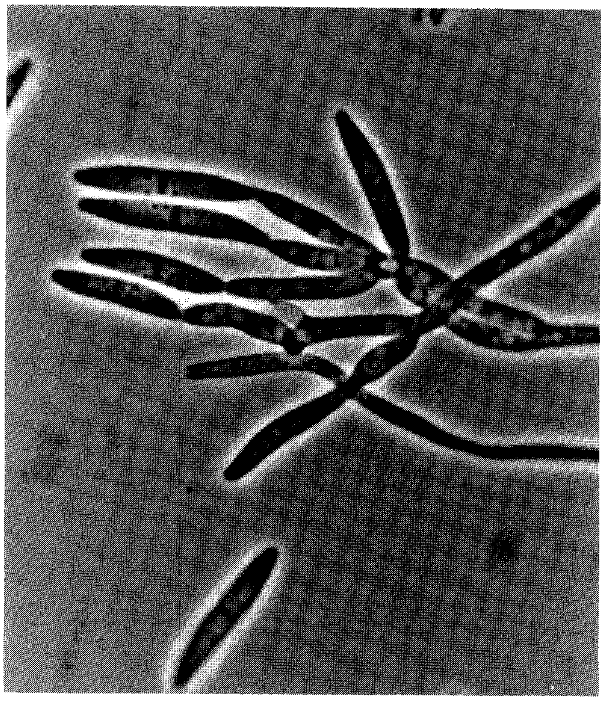

Fig. 2. Conidia of *Sirococcus* on white birch.

one year old. A few structures resembling the pycnidia were observed on specimens with broken bark but none were found containing conidia. The pycnidia were most abundant on buds (Fig. 1) especially at scale margins with a few on affected bark. Pycnidia mostly occurred as clusters but solitary ones occurred too, especially on bark.

Pycnidia are dull black and somewhat irregular in shape though more or less globose. The mean diameter of 100 pycnidia was 0.35 mm with a range from 0.2 to 0.8 mm.

Conidia (Fig. 2) are hyaline fusiform structures. Measurements of 50 spores from each of four collections had a mean length of 17.4 μm with a range of 11.4 to 21.5 μm and a mean width of 3.3 μm with a range of 2.6 to 4.3. Little size variation was observed among collections. Septal frequency (Table 1) varied among collections with some conidia uniseptate, others bi-septate or tri-septate.

Discussion

The occurrence of a *Sirococcus* on dieback of birch constitutes a new host record for *Sirococcus*. The only species of *Sirococcus* known to occur on hardwoods is *S. clavigignenti*, not known from British Columbia, but causing a canker of butternut (*Juglans cinerea* L.) in Wisconsin (3). A comparison of field and culture material involving the fungus on birch, *S. strobilinus*, and *S. clavigignenti* would be helpful in determining the status of *Sirococcus* on birch. Meanwhile, the differences in conidial sizes and conidial septation among the species lends support to the view that the fungus on birch is a new species.

Table 1. Percentage of conidia with septae.

Source	0-Septate	1-Septate	2-Septate	3-Septate
Collection				
1	0	30	22	46
2	0	38	32	32
3	0	28	40	32
4	0	36	32	32
Means	0	33	31.5	35.5

Inoculations of birch are required to determine if the fungus described here is capable of causing a stem and branch dieback. Cross inoculations involving *S. strobilinus* would also be helpful. However, the absence of any obervations of other fungi on the dieback samples and the occurrence of this *Sirococcus* at several locations in 1984 and 1985 lends support to the view that it is pathogenic on white birch.

Acknowledgements

We thank staff of the forest Insect and Disease Survey, and especially Mr. R. Turnquist, for their invaluable assistance.

Literature cited

1. Arnold, R.H. 1967. A canker and foliage disease of yellow birch. Can. J. Bot. 45:783-801.
2. Funk, A. 1972. *Sirococcus* shoot-blight of western hemlock in British Columbia and Alaska. Plant Dis. Reptr. 56:645-647.
3. Nair, F.M.G. and C.J. Kostichka. 1979. *Sirococcus clavigignenti-jugland-acearum*: an undescribed species causing canker on butternut. Mycologia 71:641-646.
4. Sutton, B.C. 1980. The coelomycetes. Commonw. Mycol. Inst. Kew. 696 pp.

Author Index to Volume 66

Instructions to authors

Articles and brief notes are published in English or French. Manuscripts (original and one copy) and all correspondence should be addressed to Dr. H.S. Krehm, Research Program Service, Research Branch, Agriculture Canada, Ottawa, Ontario K1A 0C6.

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

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

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

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

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

Literature cited should be listed alphabetically in the form appearing in current issues; either the number system or the name-and-year system may be used. For the abbreviated form of titles of periodicals, refer to the most recent issue of *Biosis List of Serials* published by Biosciences Information Service of Biological Abstracts or to the *NCPTWA Word Abbreviation List*, American National Standards Institute.

Recommandations aux auteurs

Les articles et les communiqués sont publiés en anglais ou en français. Les manuscrits (l'original et une copie) et toute la correspondance qui s'y rapporte doivent être envoyés à M. H.S. Krehm, Service des programmes de recherche, Direction de la recherches, ministère de l'Agriculture du Canada, Ottawa (Ontario) K1A 0C6.

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

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

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

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

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

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