# Canadian Plant Disease Survey

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# Inventaire des maladies des plantes au Canada

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

*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.

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# Incidence of wheat spindle streak mosaic in Essex, Kent and Lambton counties, Ontario, 1973-81

*L.F. Gates*[1]

In the 10 seasons 1973-1982 inclusive, symptoms of wheat spindle streak mosaic (WSSM) were moderate to prominent in severity and extent in five years, very widespread and severe in 1973 and 1974, and were slight or confined to lower leaves in 1977, 1979 and 1982. Year by year the average incidence of visible infections for individual counties ranged usually between 4 and 33% infected shoots. Wheat in all three counties had an average of 60% of visibly-infected shoots in 1974 or in 1975. Estimated overall yield losses by counties from visible infections in 1973-81 were: Essex 3.5%, Kent 3.4%, and Lambton 2.4%, currently (1983) representing 3400, 2800 and 3200 tonnes per year respectively, with a total value for the three counties of $1,300,000 annually.

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De 1973 à 1982 inclusivement, on a observé les variations suivantes des symptômes du virus de la filosité panachée du blé, modérés à importants en sévérité et en distribution durant 5 ans, très répandus et sévères en 1973 et 1974, et légers ou limités aux feuilles inférieures en 1977, 1979 et 1982. D'année en année, l'incidence moyenne des infections visibles dans chaque comté varie habituellement entre 4 et 33% de tiges infectées. Dans les trois comtés, le blé en 1974 ou en 1975 avait en moyenne 60% des tiges visiblement infectées. On estime les pertes de rendement par comté dues aux infections visibles à 3.5% pour l'Essex, 3.4% pour le Kent et 2.4% pour le Lambton, ce qui représente pour l'année 1983 des pertes de 3400, 2800 et 3200 tonnes par année respectivement, avec une valeur totale annuelle de $1,300,000 pour les trois comtés.

## Introduction

Wheat spindle streak mosaic (WSSM) occurs most extensively in s. Ontario, Michigan and New York State, and has become particularly prominent in sw. Ontario, where much of Ontario's winter wheat (*Triticum aestivum* L. em. Thell.) is grown and where fields are often planted with wheat every third year. The virus vector is the soilborne fungus *Polymyxa graminis* Led. (5,11), which enters wheat roots in the fall and introduces the virus. Symptoms develop in the spring, when the wheat starts growing again, and are especially prominent when long periods at about 10°C occur (7, 9, 10). Direct controls have not been practical (3, 8), but genotypes differ in incidence and intensity of symptoms. Surveys of the disease in 1973-81 in Essex, Kent, and Lambton Counties of sw. Ontario, and comparisons of its incidence in several commercial cultivars are reported here.

## Methods

Surveys were made during May, when symptoms show the most clearly on recently-grown leaves. Fields were selected at random and enough counts made on 0.3-1 m lengths of row to give a consistent estimate of the proportion of infected shoots. WSSM was also counted or scored in plots of the Ontario Winter Wheat Cooperative Tests at Malden, Ontario, on soil infested with the viruliferous vector.

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## Results and discussion

**Surveys.** Surveys (Table 1) continue those of earlier seasons (1,2). As found earlier by Slykhuis (7,9,10), springs in which very warm periods occurred in April and May (e.g. 1977), or when the weather became and stayed very warm when the wheat started to grow (e.g. 1982), resulted in low infection counts, whereas years with cold May weather (e.g. 1973) or cold nights in May (e.g 1974) were years with high infection counts. As the virus persists for five or more years in resting spores of *P. graminis* (7), and as symptoms are so strongly influenced by weather conditions, variations in disease assessments over the survey period may show variations in symptom expression more than variations in disease incidence.

In the 10 seasons 1973-1982 inclusive, symptoms were moderate to prominent in severity and extent in five years, very widespread and severe in 1973 and 1974, and were slight or confined to lower leaves in 1977, 1979 and 1982. Year by year the average incidence of visible infections for individual counties ranged usually between 4 and 33% infected shoots. Wheat in all three counties had an average of about 60% of visibly-infected shoots in 1974 or in 1975.

Survey counts are generally lower from 1976 onwards when Fredrick was planted over most of the area surveyed. In comparative trials (see below), infection counts were lower on Fredrick than on the previously-grown Yorkstar.

From 1979 onwards, wheat was often planted after soybeans, and therefore late. In 1979, and to some extent later, infection was lower than previously. Late planting is known to reduce symptom level in the following season (3,12), presumably because many of the tillers that then develop in the spring

Table 1. Incidence of wheat spindle streak mosaic in Essex, Kent and Lambton counties in 1973*-81.

| County | Year | Number of Fields with: No disease | Trace of disease (none in counts) | Up to 10% visibly-infected shoots | 11-50% visibly-infected shoots | 51-90% visibly-infected shoots | 91-100% visibly-infected shoots | Average visible infection for all fields (%) |
|---|---|---|---|---|---|---|---|---|
| Essex | 1973 | 5 | 6 | 11 | 9 | 8 | 11 | 39 |
| | 1974 | 7 | 3 | 11 | 21 | 12 | 4 | 32 |
| | 1975 | 2 | 3 | 1 | 11 | 18 | 11 | 60 |
| | 1976† | 6 | 5 | 3 | 3 | 3 | 5 | 31 |
| | 1977 | 11 | 5 | 9 | 11 | 1 | 0 | 10 |
| | 1978 | 5 | 5 | 13 | 25 | 3 | 5 | 26 |
| | 1979 | 3 | 0 | 26 | 38 | 2 | 1 | 19 |
| | 1980 | 13 | 0 | 11 | 9 | 6 | 0 | 16 |
| | 1981 | 10 | 19 | 8 | 8 | 12 | 8 | 30 |
| Kent | 1973 | 2 | 7 | 10 | 11 | 3 | 18 | 46 |
| | 1974 | 0 | 2 | 6 | 10 | 10 | 11 | 56 |
| | 1975 | 6 | 0 | 11 | 9 | 9 | 2 | 33 |
| | 1976† | 1 | 1 | 4 | 13 | 2 | 1 | 27 |
| | 1977 | 5 | 0 | 11 | 5 | 0 | 0 | 9 |
| | 1978 | 5 | 0 | 5 | 10 | 1 | 0 | 21 |
| | 1979 | 6 | 0 | 12 | 8 | 0 | 0 | 8 |
| | 1980 | 3 | 0 | 6 | 7 | 1 | 3 | 30 |
| | 1981 | 11 | 4 | 10 | 11 | 4 | 5 | 24 |
| Lambton | 1973 | 3 | 3 | 3 | 2 | 3 | 2 | 30 |
| | 1974 | 1 | 1 | 2 | 2 | 5 | 7 | 63 |
| | 1975 | 1 | 0 | 8 | 2 | 3 | 0 | 21 |
| | 1976† | 3 | 1 | 5 | 3 | 1 | 0 | 15 |
| | 1977 | 6 | 1 | 6 | 3 | 0 | 0 | 8 |
| | 1978 | 7 | 0 | 4 | 3 | 0 | 0 | 14 |
| | 1979 | 8 | 0 | 9 | 1 | 0 | 0 | 4 |
| | 1980 | 6 | 0 | 3 | 4 | 1 | 0 | 15 |
| | 1981 | 10 | 3 | 8 | 4 | 0 | 2 | 12 |

* Overall average infection levels for 1969-72 were: Essex 37, Kent 35, Lambton 21%. (Gates 1973).
† From 1976 onwards, Fredrick replaced Yorkstar over most of the area surveyed.

mature before symptoms appear. However, planting late enough to reduce WSSM risks winterkill (3), as occurred in 1979 in many fields in Kent County along the shore of Lake Erie.

**Yield losses.** The average percentages of visibly infected shoots in Essex, Kent and Lambton Counties for 1973-81 were 29,28 and 20 respectively. Infected stands or plants compared with healthy ones are reduced in yield by 10% (1; infected v. symptom-free and presumably uninfected areas in fields of Yorkstar), 15% (4; average for 5 cultivars of field comparisons of rows treated with infectious or sterilized inoculum) and 28% (13; average loss in seed weight per plant for 9 susceptible cultivars). The third estimate comes from plants spaced 22 cm apart, and reflects largely the effects of reduced tillering of infected plants, which may be less important in normally-sown rows except in areas thinned by winter damage. From the average of the first two estimates, a fully infected field would lose about 12% in yield. If in the surveys symptomless, though possibly infected, shoots are assumed to give full yields, the overall yield losses by counties from visible infections in 1973-1981 were Essex 3.5%, Kent 3.4% and Lambton 2.4% currently (1983) representing 3400, 2800 and 3200 tonnes per year, respectively, with a total value for the three counties of $1,300,000 annually. In seasons in which wheat in a county averaged 60% infection, its yield loss would be about 7%. Losses in the less susceptible Fredrick in 1976-1981 totalled 6300 tonnes per year for the three counties, with an annual value of $880,000, with the additional risk that infection of Fredrick with WSSM also reduces its winterhardiness (6).

Table 2. Reactions of winter wheat cultivars grown in Ontario to exposure to natural infection with wheat spindle streak mosaic virus, 1975-84, Malden, Ontario.

| | Visibly-infected shoots % | | | Infection score 1 (low) – 9 (high)* | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Cultivar | 1975 | 1976 | 1977 | 1978 | 1979 | 1980 | 1981 | 1983 | 1984 |
| Yorkstar | 86 | 30 | 20 | 8 | 8 | 9 | 6 | 9 | 6 |
| Fredrick | 71 | 18 | 14 | 6 | 5 | 9 | 5 | 7 | 1 |
| Gordon | | | 9 | 5 | 9 | 6 | 6 | 9 | 4 |
| Favor | | | | 9 | 9 | 8 | 9 | 9 | 7 |
| Houser | | | | | | | 6 | 9 | 6 |
| Augusta | | | | | | | 8 | 9 | 6 |
| Frankenmuth | | | | | | | 7 | 9 | 8 |

* 9 = essentially all shoots showing infection.

Cultivar reactions to wheat spindle streak mosaic virus. Fredrick and Gordon (Table 2) showed fewer shoots with symptoms than Yorkstar. The recently-licenced cultivars Favor, Houser, Augusta and Frankenmuth had more infection. Although they yielded as well as or better than Fredrick on soil infested with viruliferous vector, increased use of them will make WSSM more prominent.

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# Emergence failure and top decay in white spruce germinants due to three fungi

*R.K. Mittal[1] and B.S.P. Wang[2]*

A study of seed-borne fungi and their impact on germination quality and quantity of white spruce seeds revealed that *Alternaria alternata* (Fr.) Keissler, *Fusarium oxysporum* Schlecht., and *Penicillium variabile* Sopp caused emergence failure and top decay in the germinants. The symptoms produced are described and discussed by species.
Key words: fungi, white spruce, germinants, disease.

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Une étude sur les champignons transmis par la semence et leur impact sur la qualité de la germination et la quantité de graines d'épinette blanche a révélé qu'*Alternaria alternata* (Fr.) Keissler, *Fusarium oxysporum* Schlecht. et *Penicillium variabile* Sopp causent le manque à la levée et la pourriture du sommet du germe. Les symptômes produits sont décrits et discutés par espèces.
Mots-clés: champignons, épinette blanche, germes, maladie.

Seed health testing is primarily concerned with evaluating the presence or absence of disease-causing organisms such as fungi, bacteria, viruses, and animal pests (ISTA 1985). In these evaluations, the main difficulty has been to find out whether seedling decay was caused by seed-borne fungi or by outside contamination. Before prescribing seed treatments for field sowings it is essential to determine the source and nature of the fungi causing pre- and post-emergence losses, and also to distinguish pathogenic from non-pathogenic fungi. A study of seed-borne fungi of white spruce (*Picea glauca* (Moench) Voss) was initiated in early 1985 as part of the National Tree Seed Centre's research program. In this paper we report preliminary observations on the impact of three fungi infecting white spruce seeds from Ontario sources.

*Alternaria alternata* (Fr.) Keissler, *Fusarium oxysporum* Schlecht., and *Penicillium variabile* Sopp have frequently been identified on white spruce seeds. They have caused radicle and cotyledon emergence failures of seed tested on moist blotter at 22°C with 12 h light. Top decay of the germinants after 15-18 days was also commonly observed. Based on 200 seeds tested, the initial percentage of infected germinants by all three species was low (4% to 8%) but increased significantly (16% to 25%) with lapse of time (after about 25 days), possibly due to spread of the pathogen. The symptoms produced are described and discussed by species below:

*Alternaria alternata* (Fr.) Keissler

This fungus attacked developing germinants, producing a greyish-green, loose, mycelial growth that turned blackish with age. The growth occurred at the point of the cotyledons' emergence from the seedcoat and also on the tip of cotyledons after seedcoat shedding (Fig.1.). Infected cotyledons frequently failed to shed their seedcoat and turned from green to brown, gradually darkening as the tissue decayed. The infection proceeded along the hypocotyl to include the whole germinant.

*Fusarium oxysporum* Schlecht.

White, cottony mycelia of this fungus developed on germinants as they emerged from seedcoats and also at cotyledon tips after seedcoat shedding (Fig.2). When located on the point of emergence, the fungus checked further growth of the germinant. When the fungus developed after hypocotyl emergence, it gradually spread on the hypocotyl, turning tissue whitish, soft, and watery, and resulted in the death of the germinant.

*Penicillium variabile* Sopp

This fungus produced leaf green, velvet-like, mycelial growth on cotyledons as they emerged from the seedcoat (Fig.3). No immediate disease symptoms were observed but, with advancing age, the cotyledons failed to come out of seedcoats. Eventually, the germinants died.

Frequent isolations of these fungi from seeds using moist blotter and potato-dextrose agar plate tests helped trace the seed-borne nature of the disease. Species of *Fusarium* and *Penicillium*, amongst others, have been reported to cause top blight in douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) and some pines, and moulding of stored seedlings of many forest species in British Columbia forest nurseries (Sutherland and Eerden 1980). This supports present observations on the pathogenicity of these fungi to white spruce germinants. Sutherland and Eerden (1980) have also reported *Alternaria* spp. caused needle-tip dieback in Engelmann spruce (*Picea engelmanni* Parry) and white spruce as a disease of minor importance. However, we found that 16%-25% of white spruce germinants were infected by *Alternaria alternata*. Moreover, as about 40% of seeds were found infected by this fungus, the chances of severe deterioration of seedling stands cannot be overlooked. It is interesting to note here that all three fungi initiate invasion only on cotyledons. Further studies, on the time and place of attack and location of fungi in seeds and germinants, are in progress.

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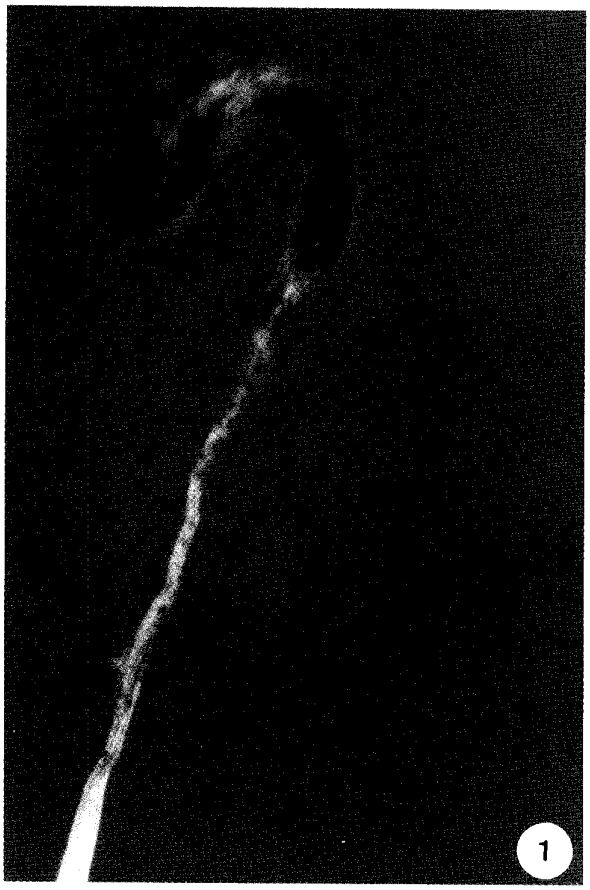



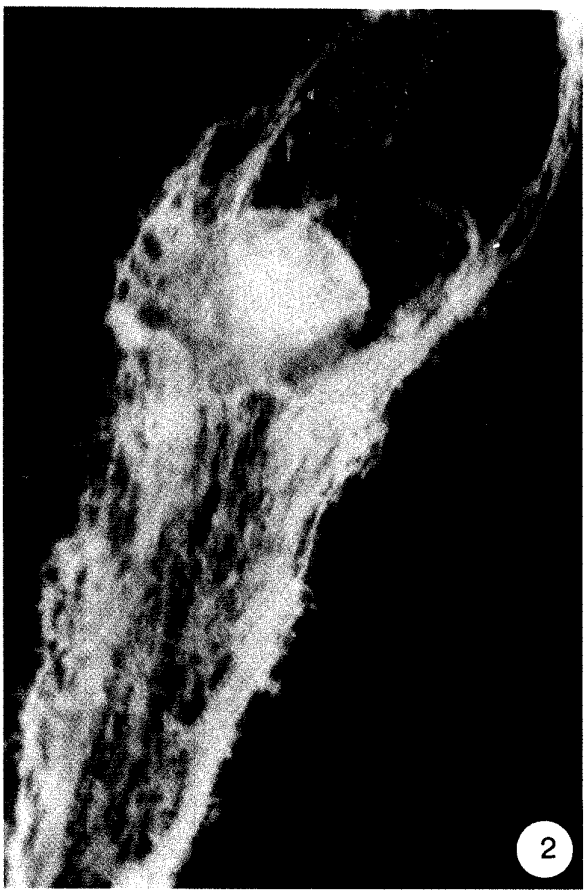


Figure 1 *Alternaria alternata* (Fr.) Keissler preventing growth and causing top decay in white spruce germinants.

Figure 2 *Fusarium oxysporum* Schlecht. preventing growth and causing top decay in white spruce germinants.

Figure 3 *Penicillium variabile* Sopp at cotyledons' emergence from seed-coat in white spruce.

# Occurrence of fusarium head blight and deoxynivalenol (vomitoxin) in two samples of Manitoba wheat in 1984

*R.M. Clear[1] and D. Abramson[2]*

*Fusarium graminearum* Schwabe was identified as the causal agent of fusarium head blight on Sinton hard red spring wheat and Coulter amber durum wheat grown on a farm south of Winnipeg in 1984. Deoxynivalenol (vomitoxin) was found in the grain at levels of 12.6 ppm in the hard red spring and 9.6 ppm in the amber durum. Other *Fusarium* mycotoxins (zearalenone, diacetoxyscirpenol, T-2 toxin and HT-2 were not present. It appears that a corn/wheat rotation and rains at anthesis favored the development of the disease.

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On a identifié *Fusarium graminearum* Schwabe comme la cause de la fusariose du blé rouge dur de printemps, Sinton et du blé dur ambré Coulter cultivés sur une ferme au sud de Winnipeg en 1984. On a trouvé 12.6 ppm de vomitoxine dans le blé dur rouge de printemps et 9.6 ppm dans le blé durum ambré. Zéaralénone, diacétoxycirpénol, T-2 et HT-2, d'autres mycotoxines fusariennes, étaient absentes. Il semble que la rotation maïs/blé et de la pluie lors de l'anthèse ont favorisé le développement de la maladie.

## Introduction

*Fusarium graminearum* Schwabe has previously been reported to occur on cereal grains in Manitoba at low levels (Gordon, 1952), being much more frequent in Eastern Canada. This species of *Fusarium* has been associated with seedling blight, stalk and cob rot of corn, and head blight of wheat, barely and oats. It is also a known producer of deoxynivalenol (DON vomitoxin) in the field. In 1980 this mycotoxin caused much concern when found in Eastern wheats (Trenholm *et al.*, 1981).

This is the first documented occurrence of *F. graminearum*-caused fusarium head blight and DON production in the prairie provinces. A field of Sinton hard red spring wheat (*Triticum aestivum* L.) and one of Coulter amber durum wheat (*T. durum* Desf.) in southeastern Manitoba were affected. Some of the conditions leading to this occurrence are examined.

## Methods

Subsamples of the harvested grains were obtained, surface sterilized in 0.3% sodium hypochlorite solution for one minute, air-dried under a laminar flow hood and plated onto potato dextrose agar (PDA) to isolate the pathogen(s). Incubation was for seven days at 22°C under a 12 hr. on/off cycle of fluorescent and long-wave UV lights. *Fusarium* species identification was done by single spore isolation onto PDA and carnation leaf agar to observe macro- and micro-morphology. Cultures were also sent to the Agriculture Canada Biosystematics Research Institutes, Ottawa, for confirmation of identity.

Subsamples of 50g were prepared for preliminary screening by thin-layer chromatography (TLC) for zearalenone and for several trichothecenes, viz., DON, diacetoxyscirpenol (DAS), T-2 toxin (T-2) and HT-2 toxin (HT-2), by the procedures of Scott *et al.* (1978) and Takitani *et al.* (1979). Further subsamples were prepared for gas chromatography/mass spectometry (CG/MS) by the procedures of Romer *et al.* (1978) and Scott *et al.* (1981). The final extracts were treated with heptafluorobutyryl-imidazole, and the heptafluorobutyrate (HFB) derivative mixture injected in *n*-hexane: benzene 9:1 containing 100 Mg/M1 methoxychlor as an internal standard.

Aliquots of 2 $\mu$L were analyzed using a Hewlett-Packard 5985 B GC/MS system equipped for splitless capillary injection and negative-ion chemical ionization using methane (Rothberg *et al.* 1983). A 12-m silica capillary column coated with OV-101 was run with helium at 230°C. Mass spectra were obtained with ion source temperature of 100°C, and with 1.00cm 3/Min methane giving an ion source pressure of $10^{-4}$ Torr.

Data on field history was obtained from the grower's records. Weather data was obtained from the records at Environment Canada of five locations closest to the outbreak area.

## Results and discussion

Subsamples of Sinton wheat had 68% of seeds infected with four species of *Fusarium*. *F. graminearum* comprised 90% of the isolates with *F. poae* (Peck) Wollenw., *F. sporotrichioides* Sherb, and *F. oxysporum* Schlecht.emend. Snyder and Hansen accounting for the remaining 10%. The Coulter amber durum had 53% of seeds infected by five species of *Fusarium*. *F. graminearum* accounted for 92% of the Fusaria, *F. sporotrichioides* for 4%, *F. poae* 2%, *F. oxysporum* 1% and *F. avenaceum* (Fr.) Sacc. 1%.

Initial TLC screening indicated the presence of high (1 ppm) levels of DON in both wheat samples. Although no other mycotoxins were found at this time, samples were re-assayed for the trichothecenes using GC/MS because of the high toxicity of some of these toxins and because of the moderate sensitivity of the spray reagents used.

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[1] *Paper No. 587 of the Canadian Grain Commission, Grain Research Laboratory, Winnipeg, Manitoba, R3C 3G8*

[2] *Contribution No. 1251 of the Agriculture Canada Research Station, Winnipeg, Manitoba, R3T 2M9*

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Table 1. Summary of agronomic management data of fields of fusarium head-blighted Sinton hard red spring and Coulter amber durum wheats in southeastern Manitoba in 1984.

| | Sinton | Coulter |
|---|---|---|
| Seeding date | April 20 | May 5 |
| Type of seed | Registered seed | Certified seed |
| Seed source | previous year's crop | previous year's crop |
| Harvest date | August 15 | August 27 |
| Crop Rotation 1983: | Corn (entire field) | 1983: Glenlea wheat 24.3 hectares; corn 16.2 hectares |
| 1982: | Sugar beets (entire field) | 1982: Corn 24.3 hectares; sugar beets 16.2 hectares |
| Seed Treatment | Unspecified fungicide for smut control | none |
| Tillage | Disced in fall, then again in the spring | |
| Fertilizer Autumn: | 43.5 kg actual N/hectare of anhydrous ammonia | |
| Seeding: | 32.6 kg actual N/hectare + 8.2 kg/hectare actual potash and phosphate | |

Only DON tri-HFB and the methoxychlor internal standard were found at the characteristic retention times in the injected samples, examining the selected-ion chromatograms for the following characteristic masses (analyte, m/z): methoxychlor, 381; DAS HFB, 542;T-2 HFB,542; DON tri-HFB, 670 and HT-2 di-HFB 816. The presence of DON as the tri-HFB derivative in both samples was confirmed by selected-ion monitoring and co-chromatography of the ions at *m/z* 884, 670, 630 and 458 characteristic of DON. This mycotoxin was present at 12.6 ppm and 9.6 ppm in the Sinton hard red spring and Coulter amber durum wheats, respectively.

Agronomic practices are given in Table 1. Of potential significance is the growing of corn on the affected fields within the past two years, because the presence of a corn-wheat rotation has been suggested as a main cause of fusarium head blight in Ontario (Teich and Nelson, 1984).

Present in the Sinton and Coulter wheats were shrivelled, chalky white kernels known as "tombstone" kernels. This kernel type constituted 14.7% of the Sinton and 6.4% of the Coulter by weight after combine harvesting. According to Simmonds (1968) this kernel does not develop beyond the early milk stage (1-2 weeks after anthesis) and probably becomes infected prior to this developmental stage. Bechtel *et al* (1985), based on structural studies, concluded that such shrivelled kernels had their development arrested two to three weeks after anthesis. An infection at anthesis could have progressed in two weeks to the level where seed development was seriously affected, producing "tombstone" kernels. Accordingly, both the Sinton and Coulter wheats were likely infected during their respective periods of anthesis, when wheat heads are most susceptible to *Fusarium* sp. (Pugh 1933, Andersen 1948, Sutton 1982) and which coincided with periods of recorded moisture.

Anthesis of the two wheats is estimated to have been about June 20 to 27 for the Sinton, and July 7 to the 14 for the Coulter (E. Czarnecki, personal communication).

During the estimated period of anthesis of the Sinton wheat, five nearby weather stations, Altona, Emerson, Greenridge, Morris 1 and Morris 2, recorded weekly totals of 24.4, 25.6, 27.3, 27.0 and 36.2 mm of precipitation, respectively. The total precipitation in June was 140 mm. During the estimated anthesis time of the Coulter amber durum wheat, the same weather stations recorded weekly totals of 37.0, 36.2, 33.6, 20.0, and 15.4 mm of precipitation. This one week period accounted for over half of the July rainfall, which totalled 44.5 mm. The mean maximum temperature for July of the three stations recording temperature was 26.6°C. High winds and 50 mm of rain were recorded by the grower during August 5-8, after which negligible rainfall occurred. The mean maximum temperature for August of the three stations was 28.4°C. Pugh *et al.* (1933) demonstrated warm temperatures increased the percentage of wheat head infections by *F. graminearum*, therefore the relatively high temperatures experienced in July and August likely aided the disease.

D. Leisle (personal communication) estimated the Sinton would have been at the early dough stage and the Coulter at the milk stage by August 5. The wheat heads would still have been susceptible to infection at this stage of development (Hart *et al.* 1984, Pugh *et al.* 1933) and a series of infections could have occurred up to and during this period as weather conditions permitted (Atanasoff, 1920). Hart *et al.* (1984), reported that production of DON was dependent on the hours of head wetness and not the stage of kernel development. Therefore DON production could also have occurred whenever conditions were suitable. One condition that may have an influence on DON production is temperature. Greenhalgh *et al.* (1983), reported *F. graminearum* grown on corn and rice at 19.5°C mainly produced zearalenone, while at 25°C both DON and zearalenone were formed, and at 28°C mainly DON.

## Conclusion

Rains during anthesis combined with ready inoculum from crop debris of the previous two years resulted in fusarium head blight, and with it the production of "tombstone" kernels and of the mycotoxin DON. There is a possiblity that a series of infections occurred, up to and including the time the kernels were at the early dough (Sinton) or milk stage (Coulter).

Avoiding a corn-wheat rotation appears to be of prime importance, even in a region such as Manitoba where the risk of fusarium head blight is usually very low. Proper crop rotation is currently the most effective control measure known, and should ensure that fusarium head blight caused by *F. graminearum* continues to be generally infrequent in Manitoba.

## Acknowledgements

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# Isolation and characterization of a new necrotic strain (NL-8) of bean common mosaic virus in Southwestern Ontario

*J.C. Tu*

Several isolates of bean common mosaic virus (BCMV) were obtained from 'Sanilac' bean plants showing severe mosaic and occasionally vein necrosis. Based on their pathogenicity to a series of differential host, the isolates were determined to be the NL-8 race of BCMV. This is the first report of this race in Canada.

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L'on a obtenu plusieurs isolats du virus de la mosaïque commune du haricot (BCMV) à partir de plants 'Sanilac' montrant des symptômes de mosaïque sévères et quelquefois une nécrose des nervures. En se basant sur leur pathogénicité envers une série d'hôte différentiel, l'on a déterminé que les isolats font partie de la race NL-8 du BCMV. Ceci est la première mention de la présence de cette race au Canada.

## Introduction

Recommended cultivars of white bean in Ontario can be divided into two groups according to their reaction to bean common mosaic virus (BCMV). One group (i.e., Sanilac and Kentwood) that does not carry 'I' gene develops typical mosaic when infected with NY-15 but, in any case, no vein necrosis and black root are apparent. The other group (i.e. Fleetwood, Seafarer, Harofleet, and Harokent) carries 'I' gene which confers resistance to the two prevalent races (the type and NY-15) as well as all other known races of BCMV. Plants that carry 'I' gene react to BCMV infection in two ways depending on the races of the virus. One develops tip and vein necrosis at moderate temperatures (20-26°C) and the other develops hypersensitive death at high temperature (1,3,4). The latter is referred to as black root because the affected plants exhibit dark brown or chocolate brown coloration of stems and roots (3). Cultivars such as Fleetwood and Seafarer fall into this category. Unfortunately, on many occasions, some infected plants develop tip and vein necrosis at moderate temperatures (20-26°C). This phenomenon indicates possible existence of a different race of BCMV. Moreover in the past 4 years, both black root and mosaic increased substantially in both experimental plots and in commercial fields. Aside from mosaic, death of plant stands due to black root in the resistant cultivars could reach as high as 30% in some fields. Undoubtedly, the disease has reached an epidemic proportion and a new type of resistance may be needed to control this disease, particularly in view of the discovery of new races NL-3 in Michigan and NL-8 in New York (5, 6) and the occurrence of NL-8 in Ontario reported herewith. The rational breeding program for BCMV resistance may have to incorporate 'bc2$^2$' gene together with 'I' gene. Plants carrying these two genes will resist all known races and not develop black root and mosaic.

This note reports the isolation and characterization of a new necrotic race of BCMV not previously found in Canada.

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## Materials and methods

Isolation and inoculation. Leaves of Sanilac plants showing mosaic and vein necrosis were collected. Each sample of leaves was triturated in 0.01 M phosphate buffer, pH 7.0 at the ratio of 1 gm leaf tissue to 3 ml buffer. A small amount of 400 mesh Carborundum was added to the sap. Inoculation was made first to greenhouse grown Sanilac plants by rubbing the sap onto fully expanded primary leaves. Inoculated leaves were rinsed 5-10 min later with tap water.

Assay on differential cultivars. Race characterization followed the differential schemes of Drijfhout *et al.* (1). The differential cultivars used were listed in Tables 1 and 2. The method of inoculation was the same as described previously. All assays were conducted in a 22 ± 2°C greenhouse. For comparative purposes a differential series was similarly inoculated with race NY-15. Disease reactions were observed 10 days after inoculation.

## Results and discussion

Reactions of the series of differential cultivars to the new necrotic isolate are summarized in Tables 1 and 2. Based on the differential host reactions described by Drijfhout *et al.* (1), the new necrotic isolate was determined to be race NL-8 of BCMV. A known race (NY-15) was employed to indicate the adequacy of the testing methods. Results are also included in Tables 1 and 2 for comparison. The results showed clearly that NL-8 was present in the Ontario bean field. This race was first discovered in The Netherlands by Drijfhout and Bos (2) in 1977.

All isolates induced similar reaction in both sets of differentials listed in Table 1 and 2. Thus, they were considered to the same strain (NL-8).

The new strain (NL-8) differs from strain (NL-3) reported in Michigan by Kelly *et al.* (5) by virtue of its inability to infect cultivars such as Redland Green Leaf-C, Puregold Wax, Redland Green Leaf-B and GN-123 (Table 1). It also differs from NL-3 by its inability to induce tip necrosis on Jubila, Topcrop, and Imperial Tendergreen (Table 2). The new strain, like NL-3, causes veinal necrosis on inoculated leaves and severe tip necrosis of Ontario cultivars such as Fleetwood, Seafarer, Harfleet, Harokent, ExRico-23, OAC-Seaforth, and OAC Rico which carry 'I' gene.

Table 1. Disease reaction of a new isolate of bean common mosaic virus on differential bean cultivars compared with known races of the pathogens[a].

| Bean Cultivar | New Isolate | NY-15 | Races I Type | I NL 1 | II NL 7 | III NL 8 | IV NL 6 | V NY 15 | V NL 2 | VI NL 3 | VI NL 5 | VII NL 4 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Dubbele Witte | S | S | S | S | S | S | S | S | S | S | S | S |
| Sutter Pink | S | S | S | S | S | S | S | S | S | S | S | S |
| Redland Green Leaf-C | R | S | R | R | R | R | S | S | S | S | S | S |
| Puregold Wax | R | S | R | R | R | R | S | S | S | S | S | S |
| Redland Green Leaf-B | R | R | R | R | R | R | S | R | R | S | S | S |
| GN-123 | R | R | R | R | R | R | S | R | R | S | S | S |
| Sanilac | S | S | R | R | R | S | R | S | S | S | S | R |
| RM-34 | S | S | R | R | R | S | R | S | S | S | S | R |
| Monroe | R | R | R | R | R | R | R | R | R | R | R | R |
| GN-31 | R | R | R | R | R | R | R | R | R | R | R | S |

[a] Differential host reactions based on Drijfhout *et al.* (1); R = resistant and S = susceptible.

Table 2. Systemic reactions of new isolate of bean common mosaic virus on bean cultivars compared with known races of the pathogen[a].

| Bean Cultivar[b] | New Isolate | NY-15 | Races I Type | I NL 1 | II NL 7 | III NL 8 | IV NL 6 | V NY 15 | V NL 2 | VI NL 3 | VI NL 5 | VII NL 4 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Jubila | I[c] | I | I | I | I | I | N | I | N | N | N | I |
| Topcrop | I | I | I | I | I | I | V | I | V | N | N | I |
| Improved Tendergreen | I | I | I | I | I | I | V | I | V | N | N | I |
| Widusa | N | I | I | I | I | N | V | I | I | N | N | I |
| Black Turtle Soap | N | I | I | I | I | N | V | I | I | N | N | I |
| Amanda | I | I | I | I | I | I | I | I | I | I | N | I |

[a] Differential host reactions based on Drijhout *et al.* (1).
[b] Cultivars carry dominant alleles of "I" gene.
[c] I = Resistant, no systemic symptoms, virus not recoverable from tips; v = variably sensitive, some plants show systemic necrosis at some temperatures, virus not recoverable from tips; and N = necrotic tip kill of most or all plants, virus not recoverable from tips.

Undoubtedly, a breeding program to incorporate 'bc2$^{2}$' gene into the cultivar with 'I' gene is urgently needed to alleviate the hypersensitive reaction caused by resistant 'I' gene, and to head off the new NL-8 and NL-3 strain in Ontario, New York and in Michigan, respectively.

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# *In vitro* soil temperature tolerance and field overwintering of soybean bacterial blight pathogen, *Pseudomonas syringae* pv. *glycinea*

*P.K. Basu*[1]

A pure culture of *Pseudomonas syringae* pv. *glycinea* adhering to nylon threads buried in sterile and non-sterile soil in petri plates survived and retained its pathogenicity for 11 months at temperatures ranging from -5° to 35°C. The pathogen survived the winter in field plots at Ottawa where infested soybean (*Glycine max*) debris was either left on the soil surface or plowed under at depths of 15-22 cm and 30-37 cm the previous fall and infected soybean plants the following spring.

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Une culture pure de *Pseudomonas syringae* pv. *glycinea*, adhérant à des fils de nylon enterrés dans du sol stérile et non-stérile dans des plats de pétri, a survécu et a conservé sa pathogénicité durant 11 mois à des températures allant de -5° à 35°C. Le pathogène a survécu à l'hiver dans des parcelles au champ à Ottawa sur des débris de soja (*Glycine max*) infestés, laissés à la surface ou enterrés à une profondeur de 15-22 cm et 30-37 cm l'automne précédent, et a infecté les plants de soja le printemps suivant.

## Introduction

Bacterial blight of soybean (*Glycine max* (L.) Merr.) caused by *Pseudomonas syringae* pv. *glycinea* Young, Dye & Wilkie (4,8) is a common disease in most soybean production areas (1,10,14), particularly during cool, wet seasons with frequent rain-storms; hot, dry weather on the other hand may arrest disease development (6,21). The pathogen may survive in infected seed (11), infested host debris (7,13,20) or volunteer soybeans (9). In Minnesota (13) and Nebraska (20) the pathogen survived better when infested soybean debris was kept on the soil surface than when burried. In Brazil (10), however, it was concluded that leaf debris was not a good between-season survival site for the pathogen; the lack of survival was attributed to moderate to high temperatures leading to rapid decay of host tissues and increased activity of antagonistic microorganisms (16). Under laboratory conditions, the pathogen can survive in dried infected leaves for several years (3) and also withstand repeated periods of freezing and thawing (12). Although the thermal death point of fresh cultures of the pathogen can be as high as 49°C, it usually failed to grow at 35°C in liquid or on solid media (4). Pure cultures, used to infest sterilized or non-sterilized soil, lost their viability within a week, whereas the pathogen in leaf tissues buried in similar soil remained viable for 6 weeks, particularly when the soil was relatively dry (20). The effect of temperature on survival of the pathogen in soil is not clearly known. From the evidence available, it is conceivable that if the pathogen does not survive in soil, then crop rotation and/or deep plowing of infested soybean debris should provide some control of the disease if healthy seed is used.

The main objectives of the present work were to determine 1) the influence of a wide range of temperatures on the survival of the pathogen in soil in the absence of host debris in the laboratory and 2) if deep plowing of infested soybean debris in field plots in the fall would reduce disease incidence the following spring.

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## Materials and methods

**The pathogen.** An isolate of *Pseudomonas syringae* pv. *glycinea* Young, Dye & Wilkie (hereafter called pv. *glycinea*), used in this work, was obtained from naturally infected soybean leaves at Ottawa in 1977 and stock cultures were maintained on yeast-dextrose-carbonate agar (19) slants at 4°C. Initially, several routine bacteriological tests (2, 19) were carried out and eventually four differential culture media plus a pathogenicity test (5) were employed to distinguish pv. *glycinea* from other soil bacteria. Color (17) and growth characteristics of the pathogen on these media were as follows: 1) on King's B medium (19), it grew copiously, was buff in color and produced a green fluorescence under ultraviolet radiation; 2) on Kado's D4 medium (19) colonies were glistening bluish-gray (pearly); 3) on Leben's M-71 medium they were reddish to ox-blood with a narrow translucent border (19); and 4) on nutrient agar (19) they were whitish to creamy in color but their growth was poor when compared to other three media.

**Survival under laboratory conditions.** The survival of the pathogen in soil was tested by a thread method (18) and virulence by a method used by Kennedy (13) and Schuster (20). Pieces of 5 cm long nylon thread were soaked in a water-suspension of a 48-h-old culture (on King's B medium) of the pathogen ($10^8$ cells/ml) for 15-20 minutes and then buried in sterilized and non-sterilized soil (3:1:1 mixture of loam, sand and peat by volume) held in 9 cm petri plates with two pieces per plate. The initial soil moisture content was 20% ± 2. Plates were individually wrapped in plastic bags to prevent moisture loss and incubated at 9 temperatures ranging from -5° to 35°C. At each temperature there were 5 sets of 3 plates. After certain periods of incubation (4,41,210,330 and 365 days) threads from one set (3 plates) at each temperature were removed, shaken gently to dislodge loosely adhering soil particles and plated on Leben's M-71 medium. In 3-4 days, the development of reddish to ox-blood bacterial colonies

along or near the pieces of thread indicated recovery of the pathogen. Using the host as a selective medium (13,20) the virulence of representative isolates of the pathogen was tested as follows: for each test, a slightly turbid bacterial suspension was prepared by rinsing 4 pieces of thread in 50 ml water and sprayed on the lower (abaxial) surface of 4 unifoliate soybean leaves with a Paasch air brush until water-soaked (5). Two kinds of controls were included to compare results: a) leaves sprayed with water only and b) leaves sprayed with a suspension ($10^6$ cells/ml) of pv. *glycinea* from stock cultures. Bacterial blight lesions developed on leaves in 5-8 days and the symptoms were rated visually as mild (+), moderate (++) and severe (+++).

**Survival under field conditions.** In a 42 × 66 m field plot at Ottawa, bacterial blight infected soybean plants (previously inoculated with pv. *glycinea*) were plowed under at normal (15-22 cm) and deep (30-37 cm) plowing depths in long strips (4.6 × 66 m, replicated twice). Also included were two non-plowed strips where plant debris was left on the soil surface during the winter of 1979-80. Each of the six strips was separated by a 2.1 m pathway to allow for equipment and vehicle movement. In the spring of 1980, each strip was divided into two equal parts to obtain half-size duplicates, and cultivation (before planting soybeans) was done only in one direction to avoid cross-contamination of soil between the strips. Three soybean cultivars, Maple Presto, Evans and PI 153.293 (Plant Introduction, U.S. Regional Soybean Laboratory, Urbana, Illinois) were planted in the strips in a split plot design (3 plowing types, 3 cvs, 2 replications and 2 duplicates (within replicates). A plot contained three 5.4 m long rows, 15 cm apart. The number of infected plants in the middle row of each plot was counted periodically from the time of emergence until 8-9 leaves were fully expanded. It was assumed that early infections from soil inoculum would begin from the lower leaves. Seed samples (100 per cv.) were tested for seed-borne infection by plating them on Leben's M-71 medium and conducting a pathogenicity test of suspected colonies.

## Results and discussion

**Survival at different temperatures in soil plates.** The pathogen remained viable in soil for 12 months (mo) through a temperature range of -5°C to 35°C but its pathogenicity was affected by prolonged incubation at higher temperatures particularly in non-sterile soil (Table 1). At low temperatures (-5° to 0°C) in both sterile and non-sterile soil it remained virulent (+++) throughout the test period (12 mo). Slight reduction of virulence (++) was noticed after 41 days at or above 30°C. After 7 mo of incubation at 5°-35°C, the pathogen produced moderate symptoms (++), and after 11 mo at 10°-35°C only mild symptoms (+) developed. After 12 mo, the pathogen from sterile soil at 20°-35°C caused mild symptoms as those at 11 mo but suspensions from non-sterile soil produced no symptoms. These results clearly show that pure cultures of the pathogen can survive in soil for a much longer period than previously reported (20). The retention of virulence was greatest at low temperatures indicating that the pathogen should be able to overwinter in temperate regions. The reduction in symptom development was probably related to a loss in the number of virulent cells of the pathogen after prolonged in-

Table 1. Pathogenicity of *Pseudomonas syringae* pv. *glycinea* from sterile (ST) and non-sterile (NS) soil incubated at temperatures from -5° to 35°C during a 12-month period.

| Soil | Temp °C | 4 days | 41 days | 7 mo | 11 mo | 12 mo |
|---|---|---|---|---|---|---|
| ST | -5 | +++* | +++ | +++ | +++ | +++ |
| NS | -5 | +++ | +++ | +++ | +++ | +++ |
| ST | 0 | +++ | +++ | +++ | +++ | +++ |
| NS | 0 | +++ | +++ | +++ | +++ | +++ |
| ST | 5 | +++ | +++ | ++ | ++ | ++ |
| NS | 5 | +++ | +++ | ++ | ++ | ++ |
| ST | 10 | +++ | +++ | ++ | + | + |
| NS | 10 | +++ | +++ | ++ | + | + |
| ST | 15 | +++ | +++ | ++ | + | + |
| NS | 15 | +++ | +++ | ++ | + | + |
| ST | 20 | +++ | +++ | ++ | + | + |
| NS | 20 | +++ | +++ | ++ | + | - |
| ST | 25 | +++ | +++ | ++ | + | + |
| NS | 25 | +++ | +++ | ++ | + | - |
| ST | 30 | +++ | ++ | ++ | + | + |
| NS | 30 | +++ | ++ | ++ | + | - |
| ST | 35 | +++ | ++ | ++ | + | + |
| NS | 35 | +++ | ++ | ++ | + | + |
| Control Pathogen | | +++ | +++ | +++ | +++ | +++ |
| Water spray | | - | - | - | - | - |

*+++ = severe, ++ = moderate, + = mild, and - = no symptoms.

cubation (7 mo or more) at relatively higher temperatures (5°-35°C). However, the pathogen did survive 11 mo at all temperatures tested, indicating its temperature tolerance. The adverse effect of non-sterile soil due to microbial antagonism (10,16) only occurred after 12 mo of incubation at higher temperatures (20°-35°C).

**Overwintering in field strips plowed at different depths.** Seed used for planting gave no evidence of seed-borne infection; therefore, it would be assumed that the primary inoculum for bacterial blight development in the spring of 1980 was from soil. The disease first appeared (June 17) on the lower leaves of three plants in a plot within a deep-plowed strip; 10 days later it was detected in a non-plowed strip, and in the following week, it was found in 13-15 plots within each plowing type. Ten days later (July 14), the number and percentage of infected plants in all 108 plots were recorded (Table 2) and alalysed (AOV). The largest number of infected plants was found in the deeply plowed strips when compared with other treatments but differences were not significant. However, cultivars differed significantly (P = 0.01) as PI 153.293 was more susceptible than Maple Presto or Evans. Thus depth of plowing had little influence on overwintering of the pathogen in this test at Ottawa.

Table 2. Number and percent of infected plants of three soybean cultivars in deep, normal and non-plowed strips on July 14, 1980.

| | Deep | | Normal | | Non-plowed | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| Maple Presto | 76 | 10.7 | 18 | 2.4 | 15 | 2.2 |
| Evans | 70 | 11.6 | 18 | 2.9 | 15 | 2.3 |
| PI 153.293 | 339 | 42.8 | 176 | 21.5 | 265 | 32.3 |
| Mean | 162 | 23.0 | 71 | 9.6 | 98 | 13.6 |

## Conclusions

The soybean bacterial blight pathogen, *P. syringae* pv. *glycinea* withstood a wide range of soil temperatures (-5° to 35°C) for 12 mo, although its virulence was affected by prolonged incubation at higher temperatures, especially in non-sterile soil. Under field conditions at Ottawa, it overwintered in soil plowed at depths of zero to 37 cm. *In vitro* results indicate that the pathogen has the ability to oversummer at higher temperatures. At the present time not enough information is available to propose a rotation procedure to reduce the inoculum potential in soil.

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# Comparative tolerance of oat cultivars to septoria leaf blotch and crown rust

*R.V. Clark and D.A. Galway*

The relative tolerance of 40 oat cultivars, to the septoria and crown rust diseases was determined in field plots in 1980 and 1981. Thousand-kernel weights were obtained for 1) plants artificially inoculated with septoria, 2) plants naturally infected by septoria and crown rust and 3) plants protected from disease by maneb fungicide; from these data tolerance ratios were determined. Based on these ratios, twelve cultivars were significantly more tolerant than the others and six of these were better both years. Most coefficients of correlation for kernel weight ratios for the two years and kernel weight ratios vs yield ratios for one year were significant. In 1980 yield ratios supported conclusions based on kernel weight ratios but the former were generally more variable.

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En 1980 et 1981, on a déterminé au champ la tolérance relative de 40 cultivars d'avoine envers la septoriose et la rouille couronnée. On a obtenu des rapports de tolérance à partir des données suivantes: poids de mille grains pour 1) des plants inoculés avec septoria, 2) des plants naturellement infectés par septoria et la rouille couronnée et 3) des plants protégés de la maladie à l'aide du fongicide maneb. En se basant sur ces données, douze cultivars ont montré une tolérance significativement supérieure aux autres, et six d'entre eux ont montré une performance supérieure les deux années. La plupart des coefficients de corrélation pour les rapports de poids de grains basés sur deux ans et pour les rapports de poids de grains contre les rapports de rendements basés sur un an étaient significatifs. En 1980, les rapports de rendement supportaient les conclusions basées sur les rapports de poids de grain, mais étaient généralement plus variables que ces derniers.

Tolerance has been described by Caldwell *et al* (1) as the ability of certain susceptible cultivars to withstand a severe disease attack without suffering a substantial loss in yield. Simons (5) has shown that tolerance to crown rust (*Puccinia coronata* Cda.) is present in certain oat (*Avena sativa* L.) cultivars and it is a quantitatively heritable but complex trait (6). Clark and Johnston (2) found that tolerance to the septoria disease (*Septoria avenae* Frank f. sp. *avenae*) was present also in certain cultivars. Thus tolerant cultivars are a viable alternative to those with specific gene resistance for controlling crown rust and septoria.

A three year study was begun in 1979 to compare the field tolerance of 45 oat cultivars to the septoria disease. Little disease developed that year even in inoculated plots given several periods of supplemental irrigation. However, septoria and crown rust developed extensively the next two years; septoria developed from natural and artificial inoculation while crown rust was initiated by natural inoculum only. The results reported here summarize the data obtained from the two years of tests when both crown rust and septoria occurred.

## Materials and methods

A randomized complete block design was employed for the treatments each year. Forty-five cultivars were grown in individual rows in 4 replicates within three blocks in 1980 and 1981 in 3-row plots 3 m long with the first two rows of each plot being Opal wheat and the third row oats. An extra 2 rows of wheat were seeded at the end of each replicate. Thus there were always 2 rows of wheat between each individual oat cultivar to reduce interplot interference (3). Rows were spaced 23 cm apart and seeding was done with a 4-row plot seeder. Each year the plants of each cultivar in one block were protected from foliage diseases with the fungicide maneb applied at 10 day intervals beginning in the 4th week of June at a concentration of 4.4 kg/ha in 935 L/ha of water employing three or four applications. The plants in a second block of the same cultivars were inoculated with macrospores of *S. avenae* f. sp. *avenae* at heading time by spraying the top leaves and emerged panicles with a spore suspension of approximately 5 $\times$ $10^3$ macrospores/ml using a Solo knapsack sprayer. The inoculum was applied in the third week in July by which time most of the late cultivars had developed panicles. At time of inoculation and for a number of days following, several periods of 0.5 to 1 hour duration of overhead irrigation were employed as needed to keep the foliage and ground wet utilizing dew type nozzles. A third or check block of cultivars did not receive any artificial inoculum or fungicide spray.

Disease severities were estimated on the top two leaves and panicles 7-10 days after inoculation.

In this study emphasis was placed on the determination of 1000-kernel weights as it had been shown previously that this kind of data, obtained from small plots, was an effective indicator of tolerance to crown rust (5). Kernel weights were determined each year for the various cultivars using two 1000 seed samples from each replicate. Seed yields were determined in 1980 by harvesting 2.4 m of each row. Yields were not determined in 1981 because of bird damage to parts of some plots. The tolerance data are expressed as ratios of artificially inoculated or naturally inoculated plots to the corresponding fungicide sprayed plots; the ratios were obtained by dividing the kernel weight or yield of each diseased plot either artificially or naturally inoculated by that of the corresponding fungicide sprayed plot. Thus the ratios are directly proportion-

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al to the relative tolerance present, with a ratio of 1.0 indicating complete tolerance or no damage from disease (5).

## Results and discussion

In 1980 a moderately severe infection of both crown rust and septoria leaf blotch developed while in 1981 crown rust was so severe that septoria ratings could not be determined on the leaves (Table 1). The artificial inoculation with septoria was done late in the season each year to supplement natural infection and to be sure that most cultivars had headed. Consequently there was little difference between the septoria leaf ratings between the artificially inoculated and the naturally infected plants (Table 1).

Of the forty cultivars included both years 12 showed superior tolerance in one or both of the two years (Table 2) based on significantly higher kernel weight ratios for inoculated plants.

Table 1. Severity of septoria and crown rust (% mean area infected, top 2 leaves) on oat plants grown in field plots at Ottawa in 1980 and 1981.

| | | Treatments | | |
|---|---|---|---|---|
| Year | Disease | Artificial inoculation with septoria | No treatment | Fungicide[1] spray |
| 1980 | Septoria | 37.8 | 31.8 | 2.4 |
| | Crown Rust | 29.9 | 29.1 | 0.9 |
| 1981 | Septoria[2] | — | — | — |
| | Crown Rust | 71.3 | 72.3 | 5.3 |

[1] Three to four foliage applications 10 days apart of maneb fungicide at 4.4 kg a.i./ha in 935 L/ha of water beginning the 4th week of June.
[2] Crown rust so predominant that septoria could not be evaluated.

Table 2. The 1000 kernel weights and tolerance ratios for 40 oat cultivars grown in field plots at Ottawa for 2 years subjected to artificial inoculum of the septoria disease and natural inoculum of crown rust.

| | 1980 | | | | 1981 | | | |
|---|---|---|---|---|---|---|---|---|
| | Kernel Weight | | Ratio[1] | | Kernel Weight | | Ratio | |
| Cultivar | Fungicide sp. | Rank | Inoculated | Natural | Fungicide sp. | Rank | Inoculated | Natural |
| Gemini | 42.0 a[2] | 1 | .838 d-l | .649 ef | 39.3 a | 1 | .660 e-k | .647 f-l |
| Athabasca | 38.2 b | 2 | .869 c-k | .716 c-f | 31.4 d-k | 17 | .890 a | .766 b-h |
| Manic | 38.6 bc | 3 | .793 h-m | .700 def | 35.7 a-e | 5 | .700 c-j | .690 e-l |
| Q075.7 | 36.0 bcd | 4 | .903 a-j | .841 b-e | 34.8 b-g | 7 | .720 c-j | .710 d-k |
| Orbit | 35.8 bcd | 5 | .925 a-g | .880 bcd | 37.9 ab | 2 | .702 c-j | .673 e-l |
| Sentinal | 35.4 b-e | 6 | .826 f-m | .786 b-f | 35.8 a-d | 4 | .655 e-k | .708 d-k |
| OA338 | 35.4 b-e | 7 | .844 d-l | .812 b-f | 34.9 b-f | 6 | .823 bc | .713 d-k |
| Abegweit | 34.6 c-f | 8 | .805 g-m | .757 b-f | 29.6 h-m | 28 | .746 b-g | .755 b-i |
| Scott | 34.4 c-g | 9 | .915 a-h | .766 b-f | 33.1 c-j | 11 | .748 b-g | .776 b-g |
| Roxton | 34.2 c-h | 10 | .874 c-k | .921 bc | 36.4 abc | 3 | .782 a-e | .743 b-k |
| Hudson | 34.2 c-h | 11 | .765 klm | .830 b-f | 30.1 g-m | 25 | .757 a-g | .696 d-l |
| Oxford | 34.0 c-i | 12 | .859 d-k | .936 b | 31.9 c-j | 13 | .750 b-g | .780 b-g |
| Dorval | 33.9 c-i | 13 | .952 a-d | .901 bcd | 31.8 c-j | 15 | .774 a-f | .806 b-e |
| Fidler | 33.7 c-j | 14 | .952 a-d | .822 b-f | 30.8 f-l | 23 | .749 b-g | .869 bc |
| Laurent | 33.5 d-k | 15 | .943 a-e | .801 b-f | 26.6 k-n | 36 | .858 ab | .845 bcd |
| Dula | 32.4 e-l | 16 | .780 j-m | .733 b-f | 30.0 h-m | 26 | .632 g-k | .612 i-l |
| Elgin | 32.1 f-m | 17 | .887 b-k | .847 b-e | 33.7 b-i | 9 | .664 d-j | .685 e-l |
| Foothill | 32.0 f-m | 18 | .779 j-m | .631 f | 33.2 c-j | 10 | .640 f-k | .599 kl |
| Saladin II | 31.7 f-m | 19 | .722 lm | .703 def | 31.0 f-k | 20 | .597 ijk | .552 l |
| Lanark | 31.5 f-n | 20 | .983 abc | 1.183 a | 30.9 f-k | 21 | .741 b-h | .771 b-h |
| Alma | 31.5 f-n | 21 | 1.006 ab | .765 b-h | 29.2 i-m | 30 | .783 a-e | .790 b-f |
| Turbo | 31.3 g-o | 22 | .784 i-m | .760 b-f | 31.8 c-j | 14 | .583 jk | .601 jkl |
| Saladin I | 31.2 g-o | 23 | .706 m | .748 b-f | 31.3 d-k | 18 | .530 k | .551 l |
| Lamar | 31.1 h-o | 24 | .886 b-k | .814 b-f | 30.8 f-l | 22 | .712 c-j | .681 e-l |
| Menomine | 30.9 i-o | 25 | .803 g-m | .746 b-f | 28.8 j-m | 32 | .638 f-k | .640 g-l |
| Garry | 30.6 j-p | 26 | .812 g-m | .785 b-f | 30.7 g-m | 24 | .687 c-j | .648 f-l |
| CI 8175 | 30.5 k-p | 27 | .840 d-l | .828 b-f | 34.2 b-h | 8 | .707 c-j | .675 e-l |
| Random | 30.2 l-p | 28 | .884 b-k | .801 b-f | 31.8 c-j | 16 | .710 c-j | .639 g-l |

Table 2. Continued

| Cultivar | 1980 Kernel Weight Fungicide sp. | 1980 Kernel Weight Rank | 1980 Ratio[1] Inoculated | 1980 Ratio[1] Natural | 1981 Kernel Weight Fungicide sp. | 1981 Kernel Weight Rank | 1981 Ratio Inoculated | 1981 Ratio Natural |
|---|---|---|---|---|---|---|---|---|
| Dal | 30.0 l-p | 29 | .900 b-j | .806 b-f | 31.1 e-k | 19 | .682 d-j | .655 f-l |
| Sioux | 29.8 l-q | 30 | .844 d-l | .763 b-f | 28.7 j-m | 33 | .694 c-j | .662 e-l |
| Lang | 29.8 l-q | 31 | .910 a-h | .927 b | 32.5 c-j | 12 | .723 b-i | .748 b-j |
| Leanda | 29.1 l-q | 32 | .706 m | .780 b-f | 29.2 i-m | 31 | .606 h-k | .616 i-l |
| Hinoat | 28.4 m-r | 33 | .910 a-i | .835 b-f | 25.9 mn | 39 | .771 a-f | .883 b |
| Jaycee | 28.2 n-r | 34 | .833 d-l | .800 b-f | 26.7 k-n | 35 | .800 a-d | .793 b-f |
| Kelsey | 27.2 o-s | 35 | .815 g-m | .787 b-f | 29.6 h-m | 27 | .714 c-j | .725 c-k |
| Stout | 26.9 p-s | 36 | .894 b-j | .800 b-f | 29.3 i-m | 29 | .681 d-j | .673 e-l |
| Gopher | 26.6 q-s | 37 | .827 e-m | .763 b-f | 28.7 j-m | 34 | .590 ijk | .623 h-l |
| Clintland | 26.4 r-s | 38 | .909 a-g | .799 b-f | 25.8 mn | 39 | .783 a-e | .765 b-h |
| Clintland 60 | 25.9 s | 39 | 1.020 a | .902 bcd | 24.0 n | 40 | .883 a | 1.030 a |
| Ottee | 25.5 s | 40 | .863 c-k | .921 bc | 26.1 lmn | 37 | .770 a-f | .757 b-i |
| Mean | 31.8 | | .859 | .809 | 31.1 | | .716 | .714 |

[1] Ratios obtained by dividing the kernel weights of inoculated or naturallly infected plants by corresponding weights of plants kept free of disease with maneb fungicide.
[2] Data in columns followed by the same letter are not significantly different (Duncan's Multiple Range Test P = 0.05).

Within this group the cultivars Alma, Clintland, Clintland 60, Dorval, Hinoat, and Laurent had significantly better tolerance both years. These cultivars were quite susceptible to the two diseases with the exception that Clintland 60 has slightly less crown rust both years. Coefficients of correlation between certain kernel weight ratios for the 40 cultivars were significant (Table 3), with the exception of the one between naturally infected plants in 1980 and 1981. Therefore the data for the two years were reasonably consistent. The highly significant correlation between the kernel weight ratios from naturally and artificially inoculated plants in 1980, a year when the septoria disease developed quite well, suggests that tolerance to both septoria and crown rust was similar. The very high correlation between the same data for 1981 was expected since crown rust was very severe on both uninoculated and inoculated plants.

Correlations between kernel weight and yield ratios obtained for the 45 cultivars grown in 1980 were highly significant with coefficients ranging from 0.53 to 0.66. Thus ratios for the kernel weights and yields for that year were in good agreement and both indicated that tolerance was present in certain cultivars. However, when the two types of ratios were analyzed statistically it was found that those for yield had quite high coefficients of variation (C.V.) (Table 4) and would not be

Table 3. Coefficients of correlation between certain kernel weight ratios[1] of 40 cultivars of oats grown for 2-yr in replicated single row plots.

| Categories of ratios correlated | r |
|---|---|
| 1980 inoculated vs. 1980 naturally infected | 0.423** |
| 1980 inoculated vs. 1981 inoculated | 0.351* |
| 1980 naturally infected vs. 1981 naturally infected | 0.232 |
| 1981 inoculated plants vs. 1981 naturally infected | 0.705** |

** and * Significant at P = 0.01 and P = 0.05 respectively.
[1] Ratios obtained by dividing the 1000 kernel weights of inoculated or naturally infected plants by corresponding 1000 kernel weights of plants kept free of disease with maneb fungicide.

Table 4. Mean yield and kernel weight ratios[1] for 45 cultivars of oats grown in 1980 in replicated single row plots comparing plants inoculated with *S. avenae* f. sp. *avenae* with plants naturally infected.

| Variable | Septoria inoculation Ratio | Septoria inoculation S.E. | Septoria inoculation C.V. | Natural inoculation Ratio | Natural inoculation S.E. | Natural inoculation C.V. |
|---|---|---|---|---|---|---|
| Yield | 0.616 | .072 | 23.5 | 0.629 | .088 | 28.1 |
| k. weight | 0.852 | .041 | 9.6 | 0.799 | .046 | 11.6 |

[1] Ratios obtained by dividing yields and 1000 kernel weights of inoculated or naturally infected plants by corresponding yields and 1000 kernel weights of plants kept free of disease with maneb fungicide.

overly reliable due to the large experimental error. The ratios for kernel weights, on the other hand, had reasonable C.V.'s and consequently were more reliable than the yield data. F values for the kernel weight and yield ratios ranged from 1.9 to 4.4 and all were significant at P = 0.01. These conclusions agree with those of Simons and Browning (4) and Simons (5).

These studies have shown that a number of oat cultivars are more tolerant to crown rust and septoria than others and that several consistently showed the trait in successive years. The good tolerance of Clintland corresponds with the results of Simons (4) in his study of tolerance with specific races of crown rust. These studies also show that kernel weights are more useful for measuring tolerance than yields.

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# Distribution and severity of Stewart's bacterial wilt of dent corn in Ontario, 1985

*T.R. Anderson and R.I. Buzzell*

Stewart's bacterial wilt was observed on dent corn in seven counties in 1985. Disease severity ranged from severe to minor in Ontario Corn Performance Tests in Essex and Wellington counties, respectively. Symptoms were limited to late season, foliar infections. The majority of hybrids in the Performance Test at Malden were considered to be susceptible. Isolates of the pathogen differed in virulence following inoculation of seedlings in the greenhouse.

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En 1985, l'on a observé la flétrissure bactérienne de Stewart sur le maïs à grains dentés dans sept comtés. La sévérité de la maladie variait de sévère à secondaire dans les tests ontariens de performance du maïs, dans les comtés de l'Essex et de Wellington, respectivement. Les symptômes ont été limités à des infections foliaires de fin de saison. La majorité des hybrides du test de performance à Malden ont été classés comme susceptibles. Les isolats du pathogène ont démontré une virulence variable au cours d'inoculation de plantules en serre.

## Introduction

Stewart's bacterial wilt of corn (*Zea mays* L.) caused by *Erwinia stewartii* (Smith) Dye was first reported at several locations in Ontario in 1932. The disease was severe on sweet corn and was observed on dent corn in Essex, Kent and Norfolk counties (2). Stewart's bacterial wilt has apparently caused little or no damage to dent corn in Ontario since it was first reported. The disease was reported on sweet corn in Essex and Kent counties in 1953 (4). In 1985, Stewart's bacterial wilt was the most common disease on dent corn in the Ontario Corn Performance Tests (OPT) in Essex County and it was observed in the cooler areas of the province (1).

The disease can be transmitted on infected seed but the most important means of dissemination and overwintering is considered to be the corn flea beetle (*Chaetonema pulicularia* Melch) (6). A forecasting system has been developed in the United States that successfully predicts wilt severity on dent and sweet corn (3). Average monthly temperatures in December, January and February are summed to determine a winter temperature index (WTI). A low WTI indicates reduced winter survival of the beetle vector. This system has not been evaluated under Ontario conditions.

The following report describes the symptoms, distribution and severity of the disease in southwestern Ontario in 1985.

## Material and methods

Isolation and identification of the pathogen: Corn leaves with symptoms of Stewart's bacterial wilt were collected at several locations in southwestern Ontario. Leaves were surface disinfested by swabbing both surfaces with 70% ethyl alcohol. Sections of tissue with water-soaked elongate lesions were placed in sterile distilled water for 15-30 minutes. A loop of the resultant suspension was streaked on nutrient agar (NA) (Difco). Plates were incubated at 28°C for 4 days. Individual colonies were transferred to yeast-dextrose-calcium carbonate medium (YDC) for storage. Two dent cultivars (3707 and 3780A from Pioneer Hi-Bred Ltd.) and a sweet corn cultivar (Golden Cross Bantam) were used in greenhouse pathogenicity trials. The method of inoculation described by Lockwood *et al.* (5) was modified in this study. Inoculum of each isolate tested for pathogenicity was increased in nutrient broth in shake culture for 48 h at 28°C. Culture solutions were diluted by 50% prior to use as inoculum and contained $1 \times 10^7$ to $1 \times 10^8$ viable cells/ml. The shoots of 10 day old plants were excised 5 mm above the epicotyl and inoculum was applied to the cut surface with a sponge or pipette. Five seedlings were inoculated per treatment in each replicate. Treatments were replicated four times. Observations of symptom development were made 7 and 10 days after inoculation. Tests were conducted twice.

Four hundred seeds from each of two entries in the OPT at Malden were surface disinfested in 20% sodium hypochlorite for 5 minutes and plated on NA. Plates were incubated at 28°C for 4-5 days. Colonies of yellow bacteria were transferred to YDC agar and subsequently tested for pathogenicity on greenhouse seedlings.

Field observations: General observations on the incidence of wilt in Ontario were made during late summer and Sept. 16-17, 1985. Entries in the OPT at Malden were rated for disease severity on Sept. 20. Ratings were made on 4 replicates by observing all plants in each single row plot and assigning a value from 0 to 5 based on foliar disease severity where 0 = lesions absent, 1 = several lesions/row, 2 = 1-3 small lesions/plant, 3 = several large lesions/plant, 4 = numerous large lesions/plant, 5 = numerous lesions, leaves shredding and prematurely senescing.

## Results

Stewart's bacterial wilt was first observed at the Research Station in mid-July on sweet corn planted adjacent to the dent corn breeding nursery. Elongate necrotic and chlorotic streaks with irregular margins occurred on the mid and upper leaves. Wilted or dead plants were not observed. Foliar lesions were observed on early dent inbreds approximately one week later.

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Lesions on dent corn were similar to those on sweet corn but frequently eliptical lesions ressembling northern leaf blight were present. Typical water soaked streaks were visible extending from the tips of older eliptical lesions. Lesions on a number of lines were surrounded by a red-brown margin. Foliar disease became more severe throughout the nursery during August. Some early inbreds senesced prematurely and produced small shrivelled cobs with few seeds. Infection on most late inbreds in the nursery was restricted to a few lesions on upper leaves.

Microscopic examination of infected leaf sections revealed non-motile bacteria flowing from cut veins. Yellow, gram negative bacteria were isolated from all leaves with symptoms of Stewart's bacterial wilt. Inoculation of dent and sweet corn seedlings in the greenhouse resulted in a range of symptoms from typical necrotic streaking with some isolates to general chlorosis and wilting of plants with other isolates. Average disease severity rating increased significantly (P = 0.01) from 2.1 to 2.8, 7 and 14 days after inoculation, respectively. In addition, average disease severity ratings of Golden Cross Bantam (2.6), 3707 (2.5) and 3708A (2.2) differed significantly (P = 0.01). The results of the seedling inoculation did not correspond to field ratings for 3707 (1.5) and 3780A (4.5). Isolates of *E. stewartii* from several locations differed significantly in virulence following inoculation of corn seedlings (Table 1). Rating disease severity on inoculated seedlings was difficult because of the range of symptoms produced by different isolates. Some isolates caused chlorosis of leaves emerging from the whorl and limited necrotic flecking on older leaves. In addition, symptoms varied with cultivar. Symptoms on Golden Cross Bantam and 3707 were frequently restricted to chlorosis of new tissue. Necrotic streaks were common symptoms on 3780A. A yellow bacterium was consistently reisolated from plants with symptoms of wilt.

Disease severity in Performance Tests ranged from severe on the majority of entries at Malden to absent on the majority of entries at Elora (Table 2). Severe disease was associated with a higher WTI in Essex County than in Wellington County (Table 3).

Disease severity ratings of the 72 entries in the OPT at Malden ranged from 1.1 to 5.0 (Table 4). The majority of the entries were considered to be susceptible to the disease. Entries with ratings of 4.6 to 5.0 may have sustained yield loss because of premature senescence or reduced leaf area. Entries with only a few lesions apparently possessed considerable resistance.

Entries with resistant ratings included Dekalb DK 496 (1.5), Pioneer 3707 (1.5) and Pioneer 3732 (2.0). Entries with susceptible ratings included Limagrain LG22 (4.8), Pride K4423 (4.8) and Renk RK 24 (4.8).

Incidence of *E. stewartii* in seed was related to foliar disease rating in the field. *E. stewartii* was isolated from 4/400 seeds of Pioneer 3780A that had a field rating of 4.5. The pathogen was not detected in seed of Pioneer 3707 that had a field rating of 1.5.

## Discussion

Although Stewart's bacterial wilt was evident in corn trials and breeding nurseries in Essex and Kent Counties the disease did not appear to cause economic losses in commercial crops. Because most of the hybrids recommended for Essex County were rated as being susceptible in the Malden OPT, disease incidence in the area should be monitored on an annual basis. Since a large proportion on Ontario's seed corn industry is located in Essex and Kent Counties seed transmission is an important aspect of the disease. Although the possibility of disseminating the disease on seed is low (6) the pathogen was isolated from seed in this study. It it also important to eliminate the disease from breeding nurseries to allow use of winter nurseries in countries with restrictions on the importation of seed that might contain Stewart's bacterial wilt.

Table 1. Virulence of *Erwinia stewartii* isolates on corn seedlings[a] 14 days after inoculation.

| Isolate | Origin | Disease rating[b] |
|---|---|---|
| ES85-1 | Woodstock | 4.2[c] |
| ES85-3 | Woodstock | 4.2 |
| ES85-4 | Woodstock | 3.7 |
| ES85-11 | Elora | 2.2 |
| ES85-12 | Fingal | 2.2 |
| ES85-13 | Innerkip | 1.8 |
| ES85-14 | Guelph | 3.6 |
| ES-2 | Cottam | 2.2 |
| Check | | 1.0 |
| | $LSD_{0.05}$ | 0.4 |

[a] Corn cultivars included sweet corn (Golden Cross Bantam) and dent corn (P3707 and P3780A).
[b] Disease rating based on scale of 1-5 where 1 = no disease, 2 = lesions on 1 of 2 inoculated leaves, 3 = lesions on both inoculated leaves, 4 = inoculated leaves wilted or dead, emerging leaves chlorotic, 5 = seedling dead.
[c] Means of 3 cultivars, 2 trials and 4 replicates with 5 seedlings per replicate.

Table 2. Distribution and severity of Stewart's bacterial wilt in 8 Ontario Corn Performance Tests, 1985.

| Test site | County | Heat Unit Rating[a] | Disease Incidence[b] on entries |
|---|---|---|---|
| Malden | Essex | 3500 | slight to severe |
| Woodslee | Essex | 3400 | slight to severe |
| Ridgetown | Kent | 3250 | absent to moderate |
| Wyoming | Lambton | 3050 | absent to slight |
| Fingal | Elgin | 3000 | absent to slight |
| Nairn | Middlesex | 2900 | absent to slight |
| Innerkip | Oxford | 2800 | absent to slight |
| Elora | Wellington | 2550 | absent to trace |

[a] Brown D.M. 1978. Heat units for corn in southern Ontario. OMAF. AGDEX 111/31. 4pp.
[b] absent = no foliar lesions; trace = 1 or 2 lesions per row; slight = 1-2 lesions on several plants within a row; moderate = numerous lesions on all plants in a row; severe = numerous lesions, leaves shredded, premature senescence on all plants in a row.

Table 3. Winter temperature indices 1984-85 and range of disease severity observed at 4 locations in southern Ontario, 1985.

| Location | Mean Monthly Temperature in °F and (°C) | | | Winter Temperature Index[a] | Disease rating[b] |
|---|---|---|---|---|---|
| | Dec. | Jan. | Feb. | | |
| Harrow | 34.5 ( 1.4) | 20.7 (-6.3) | 21.7 (-5.7) | 76.9 | slight-severe |
| Ridgetown | 34.2 ( 1.2) | 19.8 (-6.8) | 23.0 (-5.0) | 77.0 | absent-moderate |
| London (Nairn) | 30.7 (-0.7) | 18.3 (-7.6) | 20.3 (-6.5) | 69.3 | absent-slight |
| Guelph (Elora) | 30.9 (-0.6) | 16.3 (-8.7) | 19.8 (-6.7) | 67.0 | absent-trace |

[a] WTI was derived from degrees Celsius by the formula $\Sigma[(-17.7^{\circ}C - \text{mean monthly temperature } ^{\circ}C) \times -1.8]$
[b] Rating system where; trace = 1 or 2 lesions/row; slight = 1-2 lesions on several plants within a row; moderate = numerous lesions on all plants in a row; severe = numerous lesions, leaves shredding and prematurely senescing on all plants in a row.

Table 4. Frequency distribution of dent corn hybrids in Stewart's bacterial wilt severity classes, Ontario Corn Performance Test, Malden, 1985.

| Disease severity rating[a] | Number of hybrids in class |
|---|---|
| 0 - 0.5 | 0 |
| 0.6 - 1.0 | 0 |
| 1.1 - 1.5 | 2 |
| 1.6 - 2.0 | 1 |
| 2.1 - 2.5 | 3 |
| 2.6 - 3.0 | 9 |
| 3.1 - 3.5 | 17 |
| 3.6 - 4.0 | 23 |
| 4.1 - 4.5 | 14 |
| 4.6 - 5.0 | 3 |

[a] 0 = lesions absent; 1 = 1 or 2 lesions/row; 2 = 1-3 small lesions or most plants; 3 = several large lesions/plant; 4 = numerous, large lesions/plant; 5 = numerous lesions, leaves shredding and prematurely senescing.

Systems based on WTI have been used successfully in the United States to predict wilt. The most recent system predicts severe disease on dent corn at indices greater than 90 and only trace amounts of disease at indices less than 80 (3). Disease severity in Essex was greater than expected based on the WTI of 76.9 for Harrow. Research is needed on factors affecting the survival of the flea beetle vector in Ontario. If Stewart's wilt occurs annually in Ontario studies on the epidemiology of disease will be necessary to control the disease.

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# Susceptibility of apple scab resistant cultivars to *Gymnosporangium juniperi-virginianae, G. clavipes* and *Botryosphaeria obtusa.*

*J. Warner*[1]

Cedar apple rust, quince rust and frogeye leaf spot were evident in 1985 in a fungicide-free second test cultivar evaluation orchard containing scab resistant apple trees. Observations on disease susceptibility were taken for each cultivar and compared to McIntosh and Delicious which were included as standards. The scab resistant cultivars and selections differed in susceptibility to the diseases. There was no evidence that resistance to scab was related to cedar apple rust, quince rust or frogeye leaf spot resistance.

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En 1985, la rouille, la rouille du cognassier et la tache ocellée étaient présentes dans un verger où l'on n'utilisait pas de fongicide afin de faire une deuxième évaluation des cultivars de pommier résistants à la tavelure. L'on a pris des observations sur la susceptibilité aux maladies de chaque cultivar pour le comparer à McIntosh et Delicious, les deux cultivars étalons. Les cultivars et les sélections résistants à la tavelure différaient dans leur susceptibilité envers ces maladies. On n'a pas trouvé d'évidence qu'il y ait une relation entre la résistance à la tavelure et celle à la rouille, à la rouille du cognassier et à la tache ocellée.

## Introduction

Apple diseases are usually controlled by one or more applications of fungicides. Apple scab caused by *Venturia inaequalis* (Cke.) Wint. is the most serious disease affecting apples (*Malus domestica* Borkh.) in northeastern growing areas and may require 12 or more fungicide sprays for control. Growing cultivars resistant to apple scab may allow a major reduction in fungicide use. When fungicide programs are reduced or eliminated, other diseases may become more prevalent on apple. Little information is available on the susceptibility of scab resistant cultivars and selections to other diseases.

This paper reports on the field susceptibility to cedar apple rust (*Gymnosporangium juniperi-virginianae* Schw.), quince rust (*G. clavipes* Cke. and Pk.) and frogeye leaf spot (*Botryosphaeria obtusa* (Schw. Shoemaker) during 1985 of scab resistant cultivars and selections from crosses including *Malus floribunda* Sieb. 821, *M. atrosanquinea* Schneid., and *M. pumila* Mill parentage. Comparisons included McIntosh and Delicious which were used as standards. Of particular interest are the selections from the Ottawa (O) breeding program and the Co-op selections from the Purdue, Rutgers and Illinois Agricultural Experiment Station Cooperative Apple Breeding Program.

## Method

Apple cultivars and selections in a scab resistant second test orchard, planted at the Smithfield Experimental Farm from 1978 to 1983 on M26, 03 and MM106 rootstocks, were used in this study. No fungicides were applied in this orchard during 1985. Insects were controlled using one application each of azinphos-methyl and phosalone, and two applications of phosmet.

A minimum of 100 fruits per cultivar were assessed in late June and early August for cedar apple rust and quince rust. These diseases were identified according to visible host symptoms (13, 17). Only the early August figures were included in this report because they tended to be higher than the late June counts.

The three most severely infected leaves on each of ten terminals per cultivar were rated for rust in late June. The number of rust lesions per leaf was estimated using a scale of 0 to 5 (0 = no lesions; 1 = 1-5; 2 = 6-25; 3 = 26-50; 4 = 51-100; 5 = 101-200 lesions per leaf). The average number of rust lesions per leaf for each cultivar was calculated using the median value for each rating given for each leaf. Size of leaf rust lesions was estimated by comparing the scab resistant cultivars to the standard cultivars, Delicious (small lesion size) and McIntosh (medium lesion size). Rust lesions were checked during August and early September for the presence of pycnia and aecia and the most advanced stage of development was recorded.

Rust infection occurred from naturally occurring sources. Eastern red cedar, *Juniperus virginiana* L., the alternate host for all three rust diseases attacking apple, was within 1/2 to 1 km of the orchard. Wetting periods in early May at the tight cluster stage of bud development (37 hr at 8.5°C), late May at the calyx stage (14 hr at 10°C and 56 hr at 13°C) and early June (22.5 hr at 15°C) served as rust infection periods (5).

Frogeye leaf spot caused by *B. obtusa* was rated on July 22, using a scale of 0 to 3 (0 = no lesions; 1 = 1-5; 2 = 6-25; 3 = 26-50 lesions per leaf). Since leaf spotting was fairly uniform on the oldest shoot and cluster leaves, an average rating for each cultivar was determined by examining 10 shoots per cultivar. Inoculum likely occurred from overwintering cankers on dead bark and twigs in the orchard. Wetting periods in early May and early June would have provided suitable conditions for *B. obtusa* leaf infection (11).

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## Results and discussion

The scab resistant cultivars and selections differed in their susceptibility to the rust fungi and to *B. obtusa* (Table 1). In this study, as well as previous reports (4, 15) there was no evidence that resistance to scab and cedar apple rust were related. Also there does not appear to be an association between scab resistance and either quince rust or frogeye leaf spot resistance.

Apple leaves are susceptible to both cedar apple rust and hawthorn rust, *G. globosum* Farl. (13, 17). Since it is very difficult to distinguish hawthorn rust from cedar apple rust on the basis of leaf symptoms (13), it is possible hawthorn rust was present on the leaves in addition to cedar apple rust. However, Aldwinckle (1) considers hawthorn rust much less frequent in occurrence than cedar apple rust.

Resistance of apple cultivars to cedar apple rust has been characterized as absence of aecia (15) or absence of pycnia and aecia (4, 16). In this study, only four cultivars, Co-op 11, Novamac, O-6414 and O-655 had no pycnia or aecia. Co-op 11 and O-6414 had no cedar apple rust fruit infection. Novamac and O-655 did not bear fruit. Cultivars with pycnia but no aecia had less than 3% fruit infection. Co-op 1, Co-op 14, Macfree, O-546, O-653 and Priscilla had pycnia and also a few aecia, however no fruit infection from cedar apple rust was observed. Trent and HAR13T18 had pycnia and a few aecia but 4 and 8% of the fruit, respectively, was infected with cedar apple rust. Cultivars with numerous aecia had from 12 to 84% fruit infection and were considered very susceptible to cedar apple rust. The cultivars and selections having the most advanced leaf reaction (aecia) tended to have the highest levels of cedar apple rust fruit infection. Cultivar resistance to

Table 1. Susceptibility of scab resistant apple cultivars to cedar apple rust, quince rust and frogeye leaf spot during 1985.

| Cultivar | % Fruit infection: Cedar apple rust | % Fruit infection: Quince rust | Average no. rust lesions per leaf | Lesion size * | Most advanced reaction ** | Frogeye leaf spot rating *** |
|---|---|---|---|---|---|---|
| Britegold | 12 | 1 | 78 | large | A (v.s.) | 1 |
| Co-op 1 | 0 | 2 | 138 | medium | A (v.f.) | 1 |
| Co-op 3 | 1 | 1 | 85 | medium | P | 3 |
| Co-op 6 | 81 | 5 | 132 | large | A (v.s.) | 1 |
| Co-op 7 | 0 | 1 | 57 | medium | P | 2 |
| Co-op 8 | 0 | 0 | 45 | medium | P | 2 |
| Co-op 9 | 35 | 14 | 32 | large | A (v.s.) | 1 |
| Co-op 10 | 0 | 4 | 118 | small | P | 2 |
| Co-op 11 | 0 | 2 | 6 | small | N | 1 |
| Co-op 12 | 62 | 3 | 94 | large | A (v.s.) | 1 |
| Co-op 14 | 0 | 1 | 51 | small | A (v.f.) | 2 |
| Co-op 15 | 38 | 1 | 59 | large | A (v.s.) | 0 |
| Co-op 16 | 51 | 6 | 78 | large | A (v.s.) | 1 |
| Delicious | 0 | 1 | 39 | small | P (v.f.) | 1 |
| HAR4T100 | 0 | 12 | 140 | medium | P | 3 |
| HAR13T18 | 8 | 0 | 63 | small | A (v.f.) | 1 |
| Jonafree | 22 | 0 | 93 | large | A (v.s.) | 0 |
| Macfree | 0 | 9 | 138 | medium | A (v.f.) | 2 |
| McIntosh | 0 | 3 | 39 | medium | P | 2 |
| Moira | 1 | 14 | 131 | medium | P | 3 |
| Murray | 1 | 12 | 100 | small | P | 2 |
| Nova Easygro | 0 | 2 | 81 | medium | P | 2 |
| Novamac | – | – | 55 | small | N | 3 |
| 0-533 | 0 | 6 | 70 | small | P | 2 |
| 0-546 | 0 | 1 | 99 | small | A (v.f.) | 3 |
| 0-625 | 0 | 17 | 86 | very small | P | 1 |
| 0-634 | 0 | 2 | 117 | small | P | 2 |
| 0-637 | 0 | 2 | 67 | medium | P | 3 |
| 0-638 | 0 | 2 | 57 | very small | P (v.f.) | 1 |
| 0-641 | 3 | 4 | 54 | small | P (v.f.) | 1 |
| 0-644 | 0 | 8 | 79 | small | P | 3 |
| 0-645 | 0 | 13 | 77 | small | P | 3 |
| 0-648 | 12 | 8 | 145 | large | A (v.s.) | 1 |
| 0-6410 | 0 | 5 | 124 | small | P | 3 |

Table 1. Continued.

| Cultivar | % Fruit infection: Cedar apple rust | % Fruit infection: Quince rust | Average no. rust lesions per leaf | Lesion size * | Most advanced reaction ** | Frogeye leaf spot rating *** |
|---|---|---|---|---|---|---|
| 0-6413 | 23 | 6 | 104 | medium | A (v.s.) | 2 |
| 0-6414 | 0 | 2 | 44 | small | N | 3 |
| 0-6415 | 2 | 5 | 77 | small | P | 1 |
| 0-6416 | 0 | 0 | 77 | small | P (v.f.) | 1 |
| 0-6417 | 0 | 5 | 73 | small | P | 3 |
| 0-653 | 0 | 10 | 106 | medium | A (v.f.) | 3 |
| 0-654 | 0 | 0 | 124 | small | P | 3 |
| 0-655 | – | – | 58 | small | N | 1 |
| 0-661 | 27 | 6 | 47 | large | A (v.s.) | 0 |
| 0-662 | 0 | 0 | 73 | small | P | 1 |
| 0-663 | 39 | 4 | 48 | medium | A (v.s.) | 1 |
| 0-664 | 0 | 0 | 64 | small | P | 2 |
| 0-669 | – | – | 129 | medium | A (v.f.) | 2 |
| Prima | 72 | 2 | 126 | large | A (v.s.) | 0 |
| Priscilla | 0 | 10 | 141 | small | A (v.f.) | 1 |
| Redfree | 1 | 4 | 49 | small | P | 2 |
| Richelieu | 22 | 5 | 63 | large | A (v.s.) | 0 |
| Sir Prize | 84 | 15 | 108 | large | A (v.s.) | 1 |
| Trent | 4 | 6 | 83 | very small | A (v.f.) | 1 |

*lesion size compared to McIntosh (medium) and Delicious (small).
**A = aecia, P = pycnia, N = nonsporulating lesion, v.s. = very susceptible, v.f. = very few.
***0 = no lesions, 1 = 1-5, 2 = 6-25, 3 = 26-50 lesions per leaf.

*G. juniperi-virginianae* based on the absence of pycnia and aecia would be a more definitive test than based solely on the absence of aecia.

Delicious and McIntosh, when evaluated for cedar apple rust resistance, have been reported to be slightly susceptible (1) or to have a few aecia (9), pycnial lesions, non-sporulating lesions or no macroscopic symptoms (3, 4, 14). In the present study both McIntosh and Delicious had pynical lesions, however, lesion size was larger and pycnia were more plentiful on McIntosh than Delicious. No fruit from either cultivar was infected with cedar apple rust.

Prima was reported susceptible to cedar apple rust (3,6,7) with aecia present (3). The present study agrees with these reports. Sir Prize was very susceptible to cedar apple rust in this study. This agrees with data from New York (6) and Massachusetts (7) but Williams *et al.* (19) report Sir Prize moderately resistant to cedar apple rust. Co-op 16 and Jonafree were also very susceptible to cedar apple rust in this study but Co-op 16 was reported moderately resistant by Williams *et al.* (18). No cedar rust was observed on Jonafree by Dayton *et al.* (10). Priscilla (6,7) and Redfree (20) were reported resistant to cedar apple rust. In this study, 0 and 1%, respectively, of the fruit was infected, although aecial and pycnial leaf lesions did occur. Becker *et al.* (7) reported Macfree leaves were more susceptible to cedar apple rust than were Nova Easygro leaves which agrees with the present study.

Conflicting reports on susceptibility of apple cultivars to cedar apple rust may be due to inoculum concentration and age of apple leaves (2, 5) or confusion with hawthorn rust (13). The present study also reports on the susceptibility of fruit to cedar apple rust. Fruit is not subject to hawthorn rust (13, 17).

Leaf rust lesions occurred on all cultivars. There was a trend for the very susceptible cultivars to have larger leaf lesions. The cultivars with non-sporulating lesions had lesions which were small and fewer in numbers than most other cultivars. However, lesion number, by itself, was not a good criteria to identify leaf susceptibility.

Mowry (15) reported that leaves infected with more than five cedar apple rust lesions tended to abscise during the summer. Leaf abscission was noted on many cultivars in this study, however, no attempt was made to correlate leaf abscission with rust infection.

More of the scab resistant cultivars and selections were susceptible to quince rust than to cedar apple rust. However, percent fruit infection for the most susceptible cultivars was higher for cedar apple rust than for quince rust (84% and 17%, respectively). The quince rust infection reported in this study may be low because fruit drop from quince rust was observed. Coulombe (personal communication) also reported fruit drop on the cultivar Quinte. The Delicious cultivar is usally considered susceptible to quince rust (1) although in this study only 1% fruit infection occurred. Many of the scab resistant cultivars were more susceptible to quince rust than was Delicious.

Coulombe (8) reported no fruit infection from quince rust on Trent and O-546, however, these were susceptible in the pre-

sent study with 6 and 1% infection, respectively. Delicious, Prima and Co-op 1 were susceptible to quince rust in both trials.

Frogeye leaf spot is the foliage symptom of black rot caused by *B. obtusa* (11, 12). Although frogeye leaf spot is not considered an important disease in the northeastern apple growing area (12) it may become more prevalent where fungicide programs are eliminated or reduced. In this study, frogeye leaf spot lesions containing pycnidia were 2 to 4 mm in diameter. Co-op 15, Jonafree, 0-661, Prima and Richelieu appeared resistant to frogeye leaf spot. The other cultivars and selections varied in susceptibility. Where several lesions occurred in close proximity (2 or 3 rating) the spots tended to coalesce forming a larger necrotic area. A rating of 2 or 3 was sufficient to cause leaf abscision.

This report shows the relative susceptibility of the various cultivars and selections to the diseases observed. The cultivars with low disease ratings may have escaped infection or may be susceptible under different conditions or inoculum loads. Cultivars which are resistant to apple scab differ in susceptibility to the apple rust diseases and frogeye leaf spot and may require several fungicide sprays for control of these diseases.

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