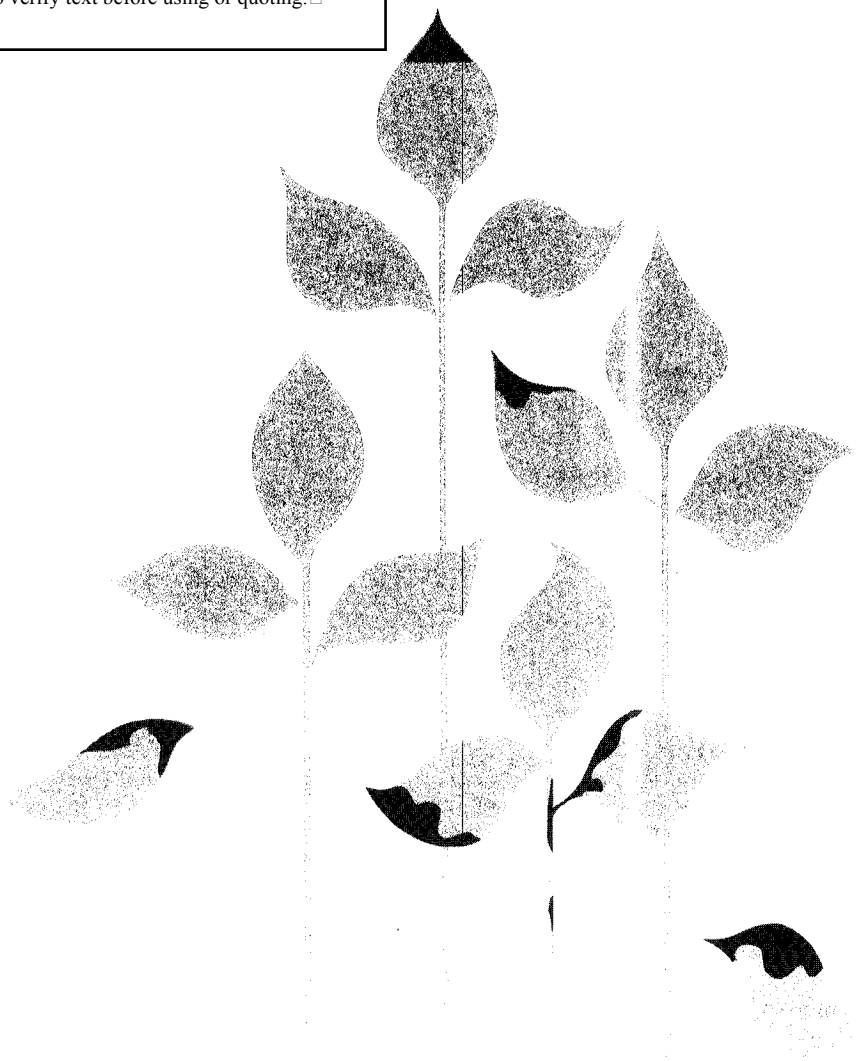
Canadian Plant Disease Survey

Inventaire des maladies des plantes au Canada

Vol. 64, No. 1, 1984

Vol. 64, Nº1, 1984



Agriculture Canada

Canada

Canadian Plant Disease Survey

Inventaire des maladies des plantes au Canada

Volume 64, Number 1, 1984

Volume 64, Numéro 1, 1984

CPDSAS 64(1) 1-23 (1984) ISSN 008-476X

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

The first paragraph of the Results and Discussion of the article entitled "A suggestion for the survey and reporting of native plant pathogens" by R.S. Hunt published in the Can. Plant Dis. Survey 63:2, 57-58, 1983 contains an error. The corrected paragraph follows:

The only native soil-borne root pathogens, for which satisfactory distribution records are available, are *Phellinus weirii* (Murr.) Gilbertson and *Armillaria mellea* (Vahl.:Fr.) Kumm. These are widely distributed (Baranyay & Bauman 1972) and, without further species subdivision and geographic reporting no inferences can be drawn.

Erratum

Le premier paragraphe de la section sur les Résultats et discussion de l'article intitulé: "A suggestion for the survey and reporting of native plant pathogens" par R.S. Hunt publié dans l'Inventaire des maladies des plantes au Canada 63:2, 57-58, 1983 contient une erreur. Le paragraphe corrigé suit ci-dessous:

The only native soil-borne root pathogens, for which satisfactory distribution records are available, are *Phellinus weirii* (Murr.) Gilbertson and *Armillaria mellea* (Vahl.:Fr.) Kumm. These are widely distributed (Baranyay & Bauman 1972) and, without further species subdivision and geographic reporting no inferences can be drawn.

Letter to the editor -

Potato spindle tuber viroid

Sir — Table 1 and the associated text in Dr. Singh's paper "Viroids and their potential danger to potatoes in hot climates" (Canadian Plant Disease Survey, 63(1), 13-18) provides misleading information with regard to the status of PSTV in Scotland, since the reader is given to believe from the title of the table that PSTV occurs in Scotland.

PSTV does not occur in Scotland, or the rest of the UK. It has been intercepted in breeding material passing through quarantine; it has been detected in, and eliminated from, advanced breeding lines; but it has never been found in commercial cultivars in the field or elsewhere in the UK.

M.J. Richardson
Head, Potato and Plant Health Division
Department of Agriculture and Fisheries for Scotland
East Craigs, Edinburgh EH12 8NJ

In reply to statements in Dr. Richardson's letter that "Table 1 and the associated text provides misleading information with regard to the status of PSTV in Scotland", Scotland is mentioned only in Table 1. It contained a listing of viroids reported from various countries and PSTV in Scotland was included on the following evidence: In a discussion-review paper (Harris *et al.* (1979) Plant Health, 231-237) the following comments were made about PSTV in Scotland: (1)"With reference to the Commonwealth Potato Collection infection was confirmed in five out of the 13 lines originally suspected to be infected (Commack & Harris, 1973)". (2)"One mild strain was found in seedlings of *Solanum gourlayi* Hawkes held by the Commonwealth Potato Collection and another strain was intercepted from certain imported clones of *S. tuberosum*". (3)"Seed-transmitted viroid has been detected in wild-tuber bearing *Solanum* species kept in the Commonwealth Potato Collection. In two lines, approximately 10% of true seed was infected, while in other, nearly 100% infection was found. Infection was recovered in one instance, from 7-year-old true seed".

It is now further reinforced from Dr. Richardson's letter that PSTV had indeed "been detected in, and eliminated from, advanced breeding lines". This is similar to the present situation in Canada.

Although PSTV has been known in various countries for a long time, there has been no report of serious economic losses anywhere in the world. PSTV should be treated as a minor disease which can be kept under control by proper certification schemes.

R.P. Singh
Agriculture Canada
P.O. Box 20280
Fredericton, N.B.
E3B 4Z7

Occurrence of tomato black ring virus on grapevine in southern Ontario

L.W. Stobbs and J.G. Van Schagen [1]

This article reports on the first occurrence in Canada of tomato black ring virus, in the Niagara Peninsula, southern Ontario.

Can. Plant Dis. Surv. 64:1, 3-5, 1984.

Cet article mentionne pour la première fois au Canada la présence du virus de la tache annulaire noire de la tomate dans la péninsule du Niagara, à l'extrémité sud de l'Ontario.

Occurrences of tomato black ring virus in grapevine have been reported only in Western Germany. Recently, however, the virus was identified in a commercial planting of Pinot Chardonnay in the Niagara peninsula, Ontario. In a shipment of 2240 finished vines of Pinot Chardonnay, clone 95, 13 vines were found to be infected with tomato black ring virus. These vines were imported into Canada in 1978 as virus-free stock from a nursery in south-west France. An additional 420 vines, received from the same source in 1979, were found to be free of the tomato black ring virus, as were adjacent Pinot Chardonnay clones 96 and 128. This is the first reported introduction of this virus into North America.

In clone 95, tomato black ring virus infections were found only in single vines within rows, with no evidence of virus transmission to adjacent vines. Soil samples taken at the base of infected vines failed to demonstrate any Longidorid species but small populations of *Xiphenema americanum* were present. Cucumber and tomato, grown in soil samples for six weeks, failed to acquire virus as determined by bioassay on *Chenopodium quinoa* or when tested serologically using Enzyme-linked immunosorbent assay.

Vines infected with tomato black ring virus were generally stunted, with older leaves showing mottling, yellowing of leaf margins, vein bunching, and leaf deformation (Fig. 1). Berries were small and poorly set (Fig. 2), a characteristic of nepovirus infection. Serological tests were positive against the G strain of tomato black ring virus obtained from Dr. G. Stellmach (Biologische Bundesanstalt, West Germany) and the Beckett Strain obtained from Dr. B. Harrison (Scottish Horticultural Research Institute, Scotland). A weak reaction was present against the S strain antiserum, suggesting that the isolate was more closely related to the G strain. No serological reaction was apparent with the other viruses tested: tomato ringspot virus, tobacco ringspot virus, raspberry ringspot virus, peach rosette mosaic virus, strawberry latent ringspot virus, tomato bushy stunt virus, cherry leafroll virus, arabis mosaic virus, grapevine fanleaf virus, and grapevine Bulgarian latent virus. Ringspot symptoms which later developed into local lesions were produced in *C. quinoa* and *C. amaranticolor*. Local lesions were also produced on cowpea and tobacco with vein necrosis occurring in bean. Large chlorotic lesions were formed on *Gomphrena* sp. and systemic mottle in cucumber. These symptoms are consistent with those reported by Murant (1970).

Infected vines were removed from the vineyard and destroyed. Clone 95 will be monitored over the next two years to identify any additional infections. Since the vector is not present, we feel that the virus will be eradicated by these measures.

Literature cited

1 Murant, A.F. 1970. Tomato Black Ring Virus. *In* Descriptions of Plant Viruses, No. 38 Commonwealth Agricultural Bureaux, Farnham Royal, Slough, U.K.

[1] *Agriculture Canada, Research Station, Vineland Station, Ontario LOR 2EO*

Accepted for publication July 20, 1983

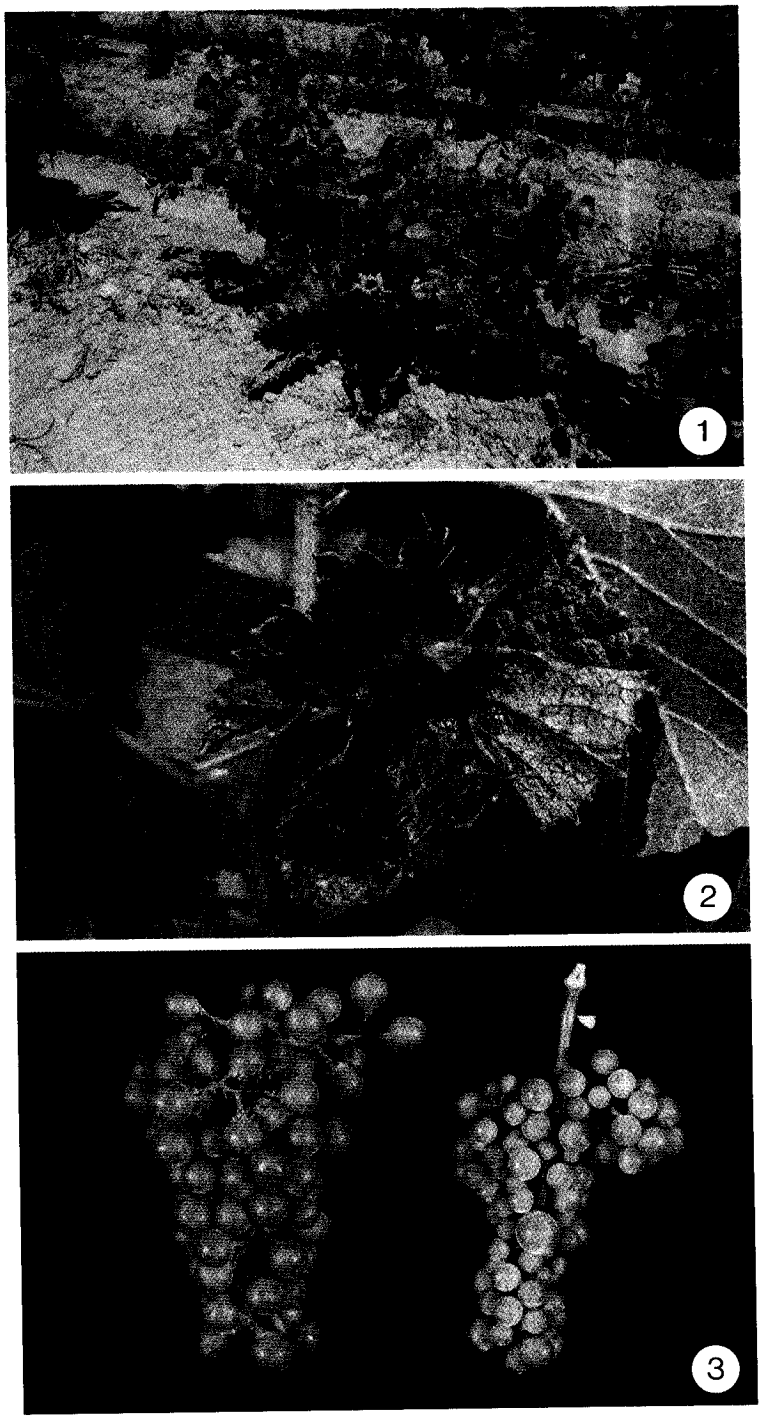


Figure 1 DeChaunac infected with tomato blackring virus, showing general stunting. **Figure 2** Vein banding and mottle on leaves of infected vines. **Figure 3** Comparison of berry set on healthy (left) and infected vines (right).

A possible new downy mildew syndrome on buckwheat seedlings

R.C. Zimmer [1]

Stunting of buckwheat seedlings was observed for the first time in 1982. Other symptom expressions observed were small stem diameter, leaf mottling (interspersion of light and dark green areas), and rugosity of some leaves. These symptoms may be the result of seed-borne systemic infection by the downy mildew pathogen *Peronospora ducometi*.

Can. Plant Dis. Surv. 64:1, 7-9, 1984.

En 1982, le rabougrissement des plantules de sarrasin accompagné de symptômes tels que des tiges de petit diamètre, des feuilles mouchetées (mélange de région vert foncé et vert pâle) et des feuilles rugeuses fut observé pour la première fois. Cet ensemble de symptômes peut être causé par une infection systémique de *Peronospora ducometi*, qui serait transmis par la semence.

Since its reporting in 1978 (8), symptoms of downy mildew of buckwheat in Manitoba, caused by *Peronospora ducometi* Siemaszko & Jankowska, have been observed almost exclusively on the leaves. The symptoms observed included chlorotic local lesions averaging 20 mm in diameter, large irregular chlorotic areas (probably the result of coalescing local lesions), and mottling usually extending throughout most of the leaf blade (Fig. 1a). The color of the infected areas of the leaves generally was a light green. Necrosis occurred in some of the circular local lesions. Reports from other countries of downy mildew on buckwheat also referred only to a foliage phase of the disease (1, 2, 3, 6, 7).

Local lesion symptoms have occurred most often on those leaves located between the middle and upper parts of the affected plants, whereas, mottling or mosaic-like symptoms appeared on those leaves nearer to the top of the plant. Occasional stunting of some upper branches was observed during periods of severe foliage infection (8).

Stunting of buckwheat plants in the seedling stage was observed for the first time in 1982 in field plots at the Morden Research Station (Figs. 1b and 1c). The degree of stunting varied substantially, with severely stunted plants being overgrown by adjacent healthy plants. Other seedlings appeared healthy until a more mature growth stage when mosaic or mottling and rugose symptoms appeared on the upper leaves (Fig. 1d).

Observations to determine the incidence of stunted buckwheat plants were made on several tests on the station. Seed of the cultivar Mancan, produced at the Morden Research Station in 1981, was used in these tests. Two tests planted on May 28, and examined 35-49 days after seeding, contained levels of 3.4% and 15.7% stunted plants. There was little evidence of local-lesion infection at this time. The average ambient daily temperature during the 7-day period following the May 28 seeding dropped to a low of 9°C by May 31 and then rose slowly to 17°C by June 4. In other tests seeded June 1 and June 11, stunted plants were either not evident or were present at a level of less than 1%. The average ambient daily temperature during the 7-day periods following the June 1 and June 11 seedings rose to highs of 18° and 20°C, respectively.

From the results of these observations, it appears that the higher ambient temperatures of 18° and 20°C following seeding may have resulted in fewer stunted plants. This was similar to the findings of Lehman (5) for downy mildew of soybean. He observed that the rapidity of germination of oospore encrusted soybean seed affected the percentage of seedlings infected systemically with *Peronospora manshurica* (Naoum.) Syd. Encrusted seeds planted in cold soil (13°C) gave rise to 40% seedling infection, whereas no systemic infection occurred at 18°C and above.

To determine if this was just a local problem or one of more widespread importance, a survey of several commercial buckwheat fields in southern Manitoba was conducted in early July. Stunting, at levels of 1-10%, was observed in 4 of 7 fields examined.

The etiology of stunting of buckwheat plants was not determined. It seems probable that the symptoms observed were the result of systemic infection from seedborne inoculum. Seed transmission of buckwheat downy mildew has been reported from Russia (6). From the results of surveys in 1979 and 1980, which covered the commercial buckwheat acreage in Manitoba, it would appear that downy mildew also is seedborne in Manitoba. It was found in all fields examined, even in fields where buckwheat had not been grown before.

The effect of systemic-like infection on yield was not determined. It appeared, however, that the yield of such plants would correlate negatively with the degree of stunting.

[1] *Agriculture Canada, Research Station, P.O. Box 3001, Morden, Manitoba ROG 1JO.*

Accepted for publication November 22, 1983

Literature cited

1 Dubinevich, B.N. 1961. Diseases of buckwheat. Zashch. Rast. Moskova 6:25-26. (Rev. Appl. Mycol. 40:690, 1961).

2 Dudka, I.A. and L.I. Burdjukova. 1978. On distribution of

Peronospora ducometi Siem et Jank. an agent of buckwheat peronosporosis in the Ukranian SSR. Ukrainskii Botanichnii Zhurnal 35:411-412.

3 Jankowska, K. 1929. Onowych dla Polski Chorobach roslin uprawnych. (Notes on diseases of cultivated plants new to Poland). Reprinted from rocznicki Nank Rolniczych i Lesnych (Yearb. Agric. Sylvicult. Sci.). Poznan 21. 10 pp. (Rev. Appl. Mycol. 8:422-423. 1929).

4 Hildebrand, A.A. and L.W. Koch. 1951. A study of systemic infection by downy mildew of soybean with special reference to symptomatology, economic significance and control. Scientific Agriculture 31:505-518.

5 Lehman, S.G. 1953. Systemic infection of soybean by *Peronospora manshurica* as affected by temperature. Elisha Mitchell Sci. Soc. J. 69:83.

6 Savitskiy, K.A. 1970. Grechika (Buckwheat). Moscow: 'kolos'. 312 pp.

7 Tanaka, I. 1934. Eine neue Art des falschen Mehltaupilzes auf dem Buchweizen. (A new species of the downy mildew fungus on buckwheat). Trans. Sapporo Nat. Hist. Soc. 13(3):203-206. (Rev. Appl. Mycol. 13:762. 1934).

8 Zimmer, R.C. 1978. Downy mildew, a new disease of buckwheat (*Fagopyrum esculentum*) in Manitoba, Canada. Plant Dis. Reptr. 62:471-473.

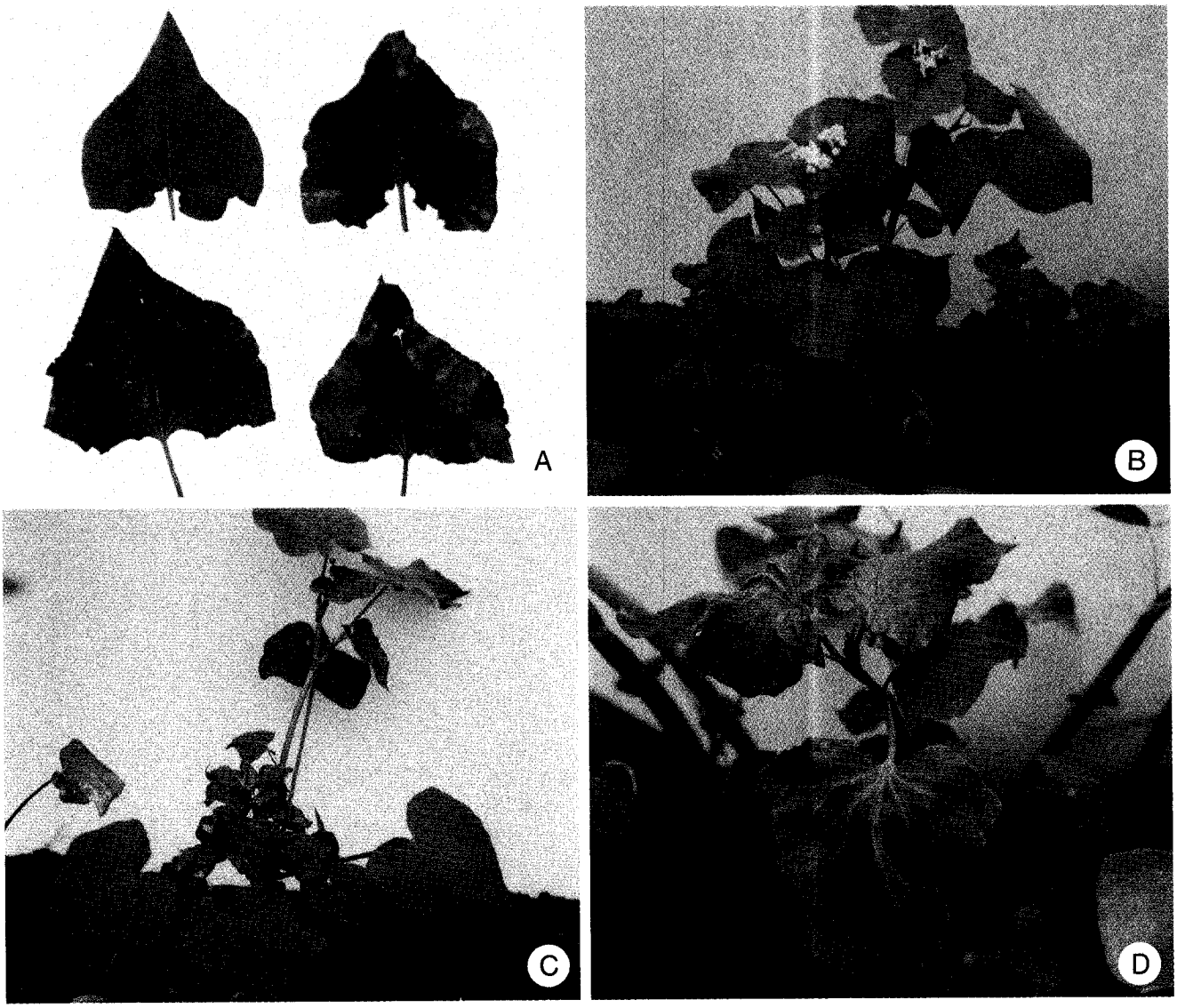


Figure 1 (A to D). (A) Local lesion infection — upper and lower right leaves show circular-like lesions, a number of lesions in the lower right leaf have necrotic outer rings; lower left leaf has coalesced circular chlorotic lesions; and the upper left leaf exhibits a few darker green areas (normal leaf tissue) amongst the lighter green chlorotic infected tissue. **(B)** Two normal plants in the center flanked by infected plants showing, stunting, faint mosaic, rugosity of leaves and small leaves. Flowering of infected plants was suppressed almost entirely. **(C)** Systemic-like infection in which one stem of the plant was stunted and leaves slightly rugose and mosaic, other stem and leaves appeared healthy. **(D)** Systemic-like infection showing a mottling type infection pattern which may fan out from the base of the leaf to all areas of the leaf. Rugosity of the infected leaves was pronounced. The plant was stunted to some degree.

Survey of fusarium head blight and possible effects of cultural practices in wheat fields in Lambton County in 1983

A.H. Teich and K. Nelson [1]

Severity of fusarium head blight was lower where wheat was not planted after maize, where nitrogen and phosphorus fertilization were adequate and where weed density was low.

Can. Plant Dis. Surv. 64:1, 11-13, 1984.

La sévérité de la fusariose du blé s'est avérée moins importante dans les champs où il n'y avait pas eu de maïs l'année précédente, où la fertilisation était adéquate en azote et en phosphore et où la densité de mauvaises herbes était peu élevée.

Introduction

Fusarium head blight of wheat is caused by *Fusarium graminearum* Schwabe and other associated *Fusarium* species. The fungus may attack wheat prior to kernel-filling and cause lower yield, shrivelled kernels and reduction in crop value.

Fusarium graminearum, the anamorph of *Gibberella zeae* (Schw.) Petch is the principal head blight pathogen in Canada (Sutton 1982). Two natural populations exist: Group 1, normally associated with fusarium crown rot, and Group 2, associated with fusarium head blight in cereals and ear rot in maize.

Sutton (1982) in his review of the epidemiology of *F. graminearum* identified the principal inoculum reservoir as host debris: stalks and ears of maize and cereal stubble. The amount of inoculum is reduced as stubble decomposes but even under conditions favorable for decomposition the pathogen can survive for at least a year. Airborne ascospores and macroconidia of *F. graminearum* are probably the major inocula for both fusarium head blight and corn ear rot.

Wheat heads are susceptible to infection by *F. graminearum* from anthesis but receptivity declines after the soft dough stage. Infection by macroconidia is favored by warmth and persistent surface wetness. Symptoms may appear within 2 days of infection (Sutton 1982) or may take a long time to develop.

There are no proven methods of controlling head blight in wheat, but there is abundant conjecture. Seaman (1982) observed that maize and wheat grown in rotation leave abundant debris which is a primary source of inoculum. Burying this debris may reduce primary inoculum, other than chlamydospores which can persist for some years. Several researchers have reported that disease severity may be reduced by avoiding both dense planting and high nitrogen fertilization (Maric *et al.* 1969, Munteanue *et al.* 1972, Wimmer 1978). Martin and Johnston (1982) surveyed wheat fields in the Atlantic provinces to compare occurrence and severity of fusarium head blight in an attempt to correlate these with cultural practices. They suggested that correct timing and appropriate land preparation, incorporating the crop residue as early as possible, rotation with a non-host crop, and controlling host weeds such as quackgrass and barnyard grass may reduce disease frequency. They found that fungicides such as propiconazole applied to the foliage reduced head blight severity and recommended that an integrated program of rotation, tillage, weed control, seed treatment and possibly foliar fungicides may be effective in reducing the severity of head blight in wheat.

Methods

Twenty-nine fields were chosen at random. Wheat growers cooperating in this study were interviewed to obtain information on field histories, including: planting and tillage methods, soil type, fertility, nitrogen applications, previous crops, herbicides, and the previous occurrence of head blight in the preceding three years. For sampling purposes, fields larger than 10 ha were sub-divided. Soil cores were taken from fields which had not been tested in the three years preceding this study, and these were sent to the OMAF Soil Analysis Lab in Guelph, Ontario for nutrient analysis. All of the fields were located in Lambton County in Southern Ontario.

In June, prior to anthesis, the fields were surveyed qualitatively for weed population density and predominant species and foliar diseases such as powdery mildew which might conceivably affect susceptibility of the wheat to head blight.

Between July 4 and 14, (anthesis to soft dough stage) each field was visited twice and the incidence of head blight was estimated as follows: the number of blighted heads in each of 36 quadrats, spaced along 3 diagonal transects (approx. 10 m apart) was counted. The quadrats were 10 m (6 rows by 16 paces) and the number of diseased heads per 100,000 heads was later calculated using the average number heads/m row (counted from sections of 6 rows) for each field.

Because of the dry season, some wheat fields were too

[1] *Research Scientist, Agriculture Canada Research Station, Harrow, Ontario, and Graduate Student, University of Guelph, Guelph, Ontario.*

Accepted for publication January 26, 1984

mature by the second set of visits for head blight to be detected, reducing the survey to twenty-two fields.

For analysis, fields were categorized according to their cultural characteristics. The disease incidence of these groups was then compared by analysis of variance (p = 0.05).

Results and discussion

Lack of wet weather during anthesis in 1983 resulted in very low overall incidence of head blight in Lambton county. Averages from the first set of observations (July 4-8) were analyzed, but the second set which had a higher disease level, was considered more reliable, and thus was used to decide on significant differences between factors.

Previous crop. Average incidence of head blight on wheat following maize was 6 to 7 times greater than that of wheat following soybeans or cereals (wheat, barley, oats) (Table 1). While *F. graminearum* can overwinter on both maize and cereal residues, possibly maize debris deteriorates less rapidly or can supply more initial inoculum than cereal debris. None of the growers had noticed 'pink mold' on cereals or maize in the past three years. The abnormally high averages obtained for

Table 1. Frequency of fusarium head blight from July 11-13 as influenced by several factors

Factor	No. of fields	Mean number of heads with symptoms per/ 100,000 plants	Differences significant at P〈0.05
Previous crop			yes
maize	5	36.0	(maize vs other)
small grain	4	7.2	
soybeans	13	5.2	
Nitrogen fertilization			yes
adequate	13	4.6	
inadequate	4	8.8	
Weed density			yes
high	13	6.4	
low	4	2.9	
Phosphorus rating			yes
high	11	4.4	
medium – low	6	7.9	
Fall plowed			no
yes	3	8.5	
no	14	5.0	
Nitrogen source			no
ammonium nitrate	9	6.6	
urea	1	2.9	
both	4	5.1	
other	3	4.3	
Powdery mildew			no
present	4	6.6	
not present	13	5.3	
June rain			no
〉50 mm	6	5.9	
〈50 mm	11	5.4	
1983 Herbicides			no
applied	5	5.4	
not applied	12	5.7	
Underseeded with clover			no
yes	3	5.4	
no	14	5.7	
Soil acidity			no
pH〉7.0	8	5.2	
pH〈7.0	9	8.0	

fields planted after maize tended to obscure any differences due to other factors, therefore the other variables were analyzed without data from fields with preceding maize crops.

Five fields were underseeded with red clover, which had no apparent effect on disease.

Soil fertility. Growers provided information on the season, rate and type of fertilizer applied. Fields with the recommended 90 kg/ha or less of actual N, showed significantly higher blight levels than those with extra nitrogen. Possibly nutrient stress may have increased susceptibility to *Fusarium* infection, or simply produced confusing symptoms such as chlorophyll loss from glumes.

Blight incidence in fields fertilized with nitrates was not significantly higher ($p = 0.05$) than in those fertilized with urea, urea + nitrate, or manure. Nitrogen was applied in both the spring and fall for most fields, an exception will be discussed later.

There were significantly higher blight counts in fields with medium-low phosphorus ratings than in those with high ratings. Potassium and magnesium ratings were high for nearly all fields. Disease levels for fields on acid soils were not significantly higher than for those on soils of pH > 7.0.

Herbicides and weeds. A comparison was made between fields with and without herbicide on the wheat crop and the difference in disease was negligible. Herbicide-treated fields differed in weed population density. Fields with noticeable amounts of weeds (mainly quackgrass, ragweed, buckwheat and mustards) averaged twice as much blight as those without weeds. Weeds could have affected disease by increasing water or nutrient stress on the wheat, or by modifying the crop environment.

Almost all growers used a herbicide on their 1982 crop, however it was difficult to isolate the residual effects of different herbicides since herbicides were confounded with the type of crop grown.

Other factors. Data for fall plowing vs other tillage, mean June rainfall by townships and powdery mildew were analyzed; there were no significant effects.

Fields were prepared by plowing, discing, cultivating, and/or harrowing. A comparison was made between plowed and unplowed fields since plowing is the most effective means of burying crop residues. Only fields previously in cereal were plowed.

From the weather data available, it appears that the time of infection in 1983 was during the last week in June. Townships recording high rainfall for June (> 50 mm) did not have higher blight levels than those with less rainfall (< 50 mm). However, monthly records for townships are too vague a source of weather data, weekly on-farm records would be preferable, and duration of surface wetness is more important than the amount of rainfall.

Early infections of powdery mildew, some of which reached the flag leaf before the onset of dry weather had no significant effect on head blight levels. There was no mildew on the heads.

All but two fields were planted with treated seed (Vitaflo-280). The untreated fields were not more diseased: it is unlikely that the seed treatment would inhibit infection of the spikelets as late as anthesis.

On each of two farms two fields were alike except for the fact that one was seeded with a drill and the other by airplane. The aerial-seeded fields were both on untilled soybean stubble in 1982, were seeded in September rather than October, and nitrogen was applied in the spring only. On both farms, blight counts were lower for seeding by airplane than for drilled fields (0.6 vs 1.6, and 1.9 vs 10.9 heads/100,000), which were also following soybeans. Differences in plant density varied with each farm; apparently this cannot explain the difference in disease levels.

Cultivars. Since the majority of wheat currently grown in Lambton county is Fredrick, no formal comparisons were made between cultivars. Qualitative observation of two Frankenmuth fields showed negligible blight levels, as was the case for adjacent Fredrick areas. Small plots of Houser and Augusta varieties gave blight counts close to that of Fredrick in a field which average 11 diseased heads/100,000.

Conclusion

From these data it appears that of all of the factors studied, the one having the greatest influence on the frequency of fusarium head blight, was a previous crop of maize. Avoiding planting winter wheat on maize stubble appears to be prudent. Controlling weed population density and maintaining adequate soil fertility, in addition to promoting yield, also appear to reduce the frequency of fusarium head blight.

Literature cited

1 Maric, A., Z. Markovic and P. Drezgii. 1969. Epiphytotic appearance of corn blight in 1968 and the influence of some agrotechnical measures on the intensity of infection. Zast. Bilja 20:15-28.

2 Martin, R.A. and H.W. Johnston. 1982. Effects and control of fusarium diseases of cereal grains in the Atlantic Provinces. Can. J. Plant Pathol. 4:210-216.

3 Munteanu, I., T. Muresan, and V. Tataru. 1972. Fusarium wilt and integrated disease control in Rumania. Acta Acad. Sci. Hung. 21:17-29.

4 Seaman, W.L. 1982. Epidemiology and control of mycotoxigenic fusaria on cereal grains. Can. J. Plant. Pathol. 4:187-190.

5 Sutton, J.C. 1982. Epidemiology of wheat head blight and maize ear rot caused by *Fusarium graminearum*. Can. J. Plant Pathol. 4:195-209.

6 Wimmer, J. 1978. Das Auftreten von Kotbenfusariosen bein Mais. Pages 78-93 *in* Veroffentlichungen der Landwirtschaftlich-chemischen Bundesversuchsanstalt. Liny/Dovau, Band Nr. 11.

Bibliography of Viroid Reviews through 1983

R.P. Singh [1]

Forty-eight review articles dealing with viroids were published between 1971 and 1983. About 80% of the articles were contributed by two groups, i.e., T.O. Diener and associates (60%) and H.L. Sanger and associates (20%). As high as 10 review articles were published in the year 1979 and 1983, respectively.

Can. Plant Dis. Surv. 64:1, 15-16, 1984.

Quarante-huit articles de synthèse sur les viroides furent publiés de 1971 à 1983. Environ 80% de ces articles proviennent de deux groupes de chercheurs soit, T.O. Diener et associés (60%) et H.L. Sanger et associés (20%). Jusqu'à dix de ces articles furent publiés en 1979 et 1983 respectivement.

Introduction

In the early 1970's it was reported that spindle tuber disease of potatoes was caused by a low-molecular weight RNA called a viroid. Since that time review articles on viroids have appeared frequently. Some of the articles have appeared in medical journals or journals not commonly available to most plant pathologists. These early reviews describe the prevalent thinking about the viroids before their true nature was elucidated by biophysical and biochemical methods. Therefore, these reviews are of historical significance. In order to make these titles available to plant pathologists, I have compiled a listing of the review articles published between 1971 and 1983. It appears that viroids are the most reviewed plant pathogen to date!

Result and Discussion

Table 1 shows that a total of 48 review articles were published in a 13- year period, an average of almost four articles a year. Although there is no consistent pattern, certain years appear to have more than others. For example, a total of 10 reviews appeared in 1979. This could account for the increased publicity generated by the complete sequence analysis of potato spindle tuber viroid by German scientists the previous year. Similarly, there were ten reviews in year 1983, which may reflect the use of viroid in recombinant DNA experimentation.

From the bibliographical list it becomes very clear that over 60% of the reviews were written by T.O. Diener and associates, followed by about 20% by H.L. Sanger and associates. In the remaining 20% are the other authors from Canada, United States and U.S.S.R.

Table 1. The number of review articles dealing with viroids, published between 1971 and 1983

Year	Number of articles	References
1971	1	4
1972	1	5
1973	2	6,7
1974	5	8,9,10,11,31
1975	0	
1976	3	12,13,42
1977	6	14,15,16,17,28,29
1978	1	18
1979	10	3,19,20,21,36,37, 43,44,45,47
1980	3	30,38,39
1981	4	22,23,24,32
1982	2	25,40
1983	10	1,2,26,27,32,34, 35,41,46,48
Total	48	
Average	3.7	

Literature cited

1 Cress, D.E., M.C. Kiefer, and R.A. Owens. 1983. Pages 160-164 *in* H.D. Robertson, S.H. Howell, M. Zaitlin, and R.L. Malmberg eds. Plant Infectious Agents — Viruses, Viroids, Virusoids, and Satellites. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York.

2 Cress, D.E., M.C. Kiefer, and R.A.Owens. 1983. cDNAs of potato spindle tuber viroid are infectious. *in* R.B. Golberg ed. Plant Molecular Biology, vol. 12, A.R. Liss Inc., New York.

3 Dickson, E. 1979. Viroids: Infectious RNA in plants. *in* T.C. Hall, J.W. Davies eds. Nucleic acids in plant, vol. 11. CRC Press, Boca Raton, Florida.

4 Diener, T.O. 1971. A plant virus with properties of a free ribonucleic acid: potato spindle tuber virus, pages 433-478. *in* K. Maramorosch and E. Kurstak eds. Comparative Virology, Academic Press, New York.

[1] *Research Scientist, Agriculture Canada Research Station, P.O. Box 20280, Fredericton, New Brunswick, E3B 4Z7, Canada.*

Accepted for publication February 15, 1984

5 Diener, T.O. 1972. Viroids. Adv. Virus Res. 17:295-313.
6 Diener, T.O. 1973. Similarities between the scrapie agent and the agent of potato spindle tuber disease. Ann. Clin. Res. 5:268-278.
7 Diener, T.O. 1973. Potato spindle tuber viroid: A novel type of pathogen. Perspectives in Virology 8:7-30.
8 Diener, T.O. 1974. Viroids: The smallest known agents of infectious disease. Ann. Rev. Microbiol. 28:23-39.
9 Diener, T.O. 1974. The smallest known agents of infectious disease. Reticuloendothelial Soc. J. 322-333 (April issue).
10 Diener, T.O. 1974. Viroids as prototypes or degeneration products of viruses. Pages 757-783. *in* E. Kurstak and K. Maramorosch eds. Viruses, Evolution and Cancer. Academic Press, New York.
11 Diener, T.O. 1974. Viroids. pages 215-227. *in* R. Markham, D.R. Davies, D.A. Hopwood, and R.W. Horne eds. Modification of the information content of plant cells, Elsevier, North Holland.
12 Diener, T.O. 1976. Viroids in Agriculture, pages 273-283. *in* J.A. Romberger, J.D. Anderson, and R.L. Powell eds. Virology in Agriculture. Allanheld Osmun & Co., Publishers, Inc., Montclair, New Jersey.
13 Diener, T.O. 1976. Towards an understanding of viroid nature and replication. Ann. Microbiol (Inst. Pasteur). 127A, 7-17.
14 Diener, T.O. 1977. Viroids: Autoinducing Regulatory RNAs? pages 50-61, *in* C.W. Anderson ed. Genetic Interaction and Gene Transfer, Brookhaven Symposium in Biology, No. 29.
15 Diener, T.O. 1977. Viroids. pages 431-436. *in* K. Maramorosch ed. The Atlas of Insects and Plant Viruses including Mycoplasma viruses and viroids. Academic Press, New York.
16 Diener, T.O. 1977. Viroids. Acta Botanica Indica 5:95-106.
17 Diener, T.O. 1977. Viroids in Agriculture. pages 363-375. *in* L. Bogorad and J.H. Weil eds. Nucleic Acids and Protein Synthesis in Plants. Plenum, New York.
18 Diener, T.O. 1978. Viroids: Persistence in Plants, evolution, and possible animal and human disease agents. pages 113-125. *in* E. Kurstak and K. Maramorosch eds. Viruses and Environment, Academic Press, New York.
19 Diener, T.O. 1979. Viroids: Structure and function. Science 205:859-866.
20 Diener, T.O. 1979. Biology of viroids. pages 281-289. *in* S.B. Prusiner, W.J. Headlow eds., Slow transmissible diseases of the nervous system. Vol. 2. Academic Press, New York.
21 Diener, T.O. 1979. Viroids and viroid diseases. Wiley and Sons, New York.
22 Diener, T.O. 1981. Viroids. pages 914-934. *in* E. Kurstak ed. Handbook of plant virus infections and comparative diagnosis. Elsevier, North Holland.
23 Diener, T.O. 1981. Viroids. Scientific American (January) 66-73.
24 Diener, T.O. 1981. Viroids: Abnormal products of plant metabolism. Ann. Rev. Plant Physiol. 32:313-325.
25 Diener, T.O. 1982. Viroids: Minimal biological systems. Bioscience 32:38-44.
26 Diener, T.O. 1983. The viroid — a subviral pathogen. American Scientist 71:481-489.
27 Diener, T.O. 1983. Viroids. Adv. Virus Res. 28: 241-283.
28 Diener, T.O., and A. Hadidi. 1977. Viroids. pages 285-337. *in* H. Fraenkel-Conrat and R.R. Wagner eds. Comprehensive Virology, Plenum, New York.
29 Diener, T.O., A. Hadidi, and R.A. Owens. 1977. Methods for studying viroids. pages 185-217. *in* K. Maramorosch and H. Koprowski eds. Methods in Virology, vol. 6, Academic Press, New York.
30 Gross, H.J. and D. Riesner. 1980. Viroids: A class of subviral pathogens. Angew Chem. Int. Ed. Eng. 19:231-243.
31 Hrama, D.P. 1974. (Potato spindle tuber viroid and similar causative agents of diseases). Mikrobiolohichnvi Zhurnal. Vol. 36: 659-668.
32 Kleinschmidt, A.K., G. Klotz, and H. Seliger. 1981. Viroid structure. Ann. Rev. Biophys. Bioeng. 10:115-132.
33 Owens, R.A., and M.C. Kiefer. 1983. *in* Y. Becker ed. Recombinant DNA Research and Viruses. Nijhoff, The Hague.
34 Owens, R.A., D.E. Cress, and T.O. Diener. 1983. Viroid-cDNA-uses in viroid detection and molecular biology. pages 185-194. *in* L.D. Owens ed. Genetic Engineering: Applications to Agriculture, Rowman & Allanheld, Totowas, New Jersey.
35 Riesner, D., G. Steger, J. Schumacher, H.J. Gross, J.W. Randles, and H.L. Sanger. 1983. Structure and function of viroids. Biophys. Struct. Mech. 9:145-170.
36 Sanger, H.L. 1979. Structure and function of viroids. pages 291-341. *in* S.B. Prusiner, W.J. Headlow eds. Slow transmissible diseases of the nervous system. Vol 2. Academic Press, New York.
37 Sanger, H.L. 1979. Viroide, eine neue Klasse molekularer Krankheitserreger. Mitt Ber Robert-Koch-Stiftung 1:71-99.
38 Sanger, H.L. 1980. Viroids: Biology, structure and possible functions. Pages 553-601. *in* D.J. Leaver ed. Genome organization and expression in plants. Plenum, New York.
39 Sanger, H.L. 1980. Structure and possible functions of viroids. Ann. N.Y. Acad. Sci. 354:251-278.
40 Sanger, H.L. 1982. Biology, structure, functions and possible origin of viroids. pages 368-454. *in* B. Parther and D. Boulter eds. Encyclopedia of Plant Physiology, New Series Vol. 148. Springer-Verlag, Berlin.
41 Sanger, H.L. 1983. Der weg zuden viroiden und zur Aufklarung ihrer Struktur. pages 307-329. Forschung in der Bundesrepublik Deutschland. Verlag Chemie, Weinheim.
42 Semancik, J.S. 1976. Structure and replication of plant viroids. pages 529-545. *in* D. Baltimore, A.S. Huang and C.F. Fox eds. Animal Virology (ICN-UCLA Symposia on Molecular and Cellular Biology) Vol. 4. Academic Press, New York.
43 Semancik, J.S. 1979. Pathogenesis and replication of plant viroids. pages 343-362. *in* S.B. Prusiner, W.J. Headlow eds. Slow transmissible diseases of the nervous system. Vol. 2. Academic Press, New York.
44 Semancik, J.S. 1979. Small pathogenic RNA in plants — the viroids. Ann. Rev. Phytopathol. 17:461-484.
45 Singh, R.P. 1979. Potato spindle tuber viroid. J. of Indian Potato Association. 6:20-35.
46 Singh, R.P. 1983. Viroids and their potential danger to potatoes in hot climates. Can. Plant Dis. Surv. 63:13-17.
47 Zaitlin, M. 1979. How viruses and viroids induce disease. pages 257-271. *in* J.G. Horsfall and E.B. Cowling eds. Plant Disease Vol. IV. Academic Press, New York.
48 Zaitlin, M. 1983. Viruses and viroids. pages 239-248. *in* T. Kommedahl and P.H. Williams eds. Challenging problems in plant health. Amer. Phytopathological Soc., St. Paul, Minnesota.

Nectria cinnabarina (Tode ex Fr.) Fr. trouvé sur des conifères au Québec

G. Laflamme et R. Cauchon [1]

Cette note rapporte pour la première fois la présence de *N. cinnabarina* sur le mélèze laricin, le mélèze d'Europe et le pin gris. C'est la première mention de la présence de ce champignon sur des conifères en Amérique du Nord.

Can. Plant Dis. Surv. 64:1, 17-18, 1984.

This note is the first record of *N. cinnabarina* on american larch, european larch, and jack pine. This is the first report of this fungus on conifers in North America.

L'ascomycète *Nectria cinnabarina* (Tode ex Fr.) Fr. dont l'anamorphe est *Tubercularia vulgaris* Tode ex Fr. appartient à l'ordre des *Sphaeriales*. Les sporodochies de *T. vulgaris* d'un rose-orangé clair sont plus souvent présents sur le substrat que les périthèces de *N. cinnabarina* d'un rouge assez sombre; les sporodochies apparaissent en premier lieu et sont remplacés le cas échéant par les périthèces sur le même stroma.

N. cinnabarina est depuis longtemps qualifié de saprophyte et aussi de parasite de faiblesse (Boyce 1961), causant alors le dépérissement nectrien, fréquent chez certaines espèces d'érables. Récemment, il a été démontré que *N. cinnabarina* pouvait causer un chancre sur le févier (Bedker *et al.* 1982), de la même façon que le pathogène *Nectria galligena* Bres. sur plusieurs essences feuillues.

T. vulgaris a été rapporté sur de nombreux hôtes. À l'Herbier national d'Ottawa, nous avons dénombré plus de 50 genres de plantes ligneuses servant de substrat à ce champignon. Pour chacun des genres, plusieurs espèces étaient souvent représentées. Par contre, aucun spécimen en collection ne provenait de conifères (Gymnospermes). Dans les principaux manuels de pathologie forestière nord-américains, on ne fait mention de *N. cinnabarina* que sur les feuillus (Boyce 1961; Pirone 1978) ou pas du tout (Tattar 1978; Manion 1981). Il en est de même en Allemagne (Schwerdtfeger 1981) et en France (Lanier *et al.* 1976). À notre connaissance, ce n'est qu'en Finlande (Kujala 1950) que *N. cinnabarina* est rapporté sur les conifères suivants: *Pinus sylvestris* L., *Picea abies* (L.) Karst. (= *P. excelsa* Link), *Abies sibirica* Ladeb. et *Thuya occidentalis* L.

Dans le cadre des relevés annuels des insectes et des maladies des arbres au Québec effectués par le Centre de recherches forestières des Laurentides, de nombreux spécimens de *N. cinnabarina* nous sont parvenus ces dernières années (Tableau 1). Parmi les nombreux hôtes sur lesquels ce champignon fut retrouvé, nous avons relevé les espèces de conifères suivantes: *Larix laricina* (Du Roi) K. Koch, *Larix decidua* Mill., *Pinus banksiana* Lamb, *Pinus sylvestris* et *Thuya occidentalis* L. Sauf pour les deux dernières essences, c'est la première mention de la présence du champignon sur des conifères en Amérique du Nord.

T. vulgaris fructifiait sur des bouts de rameaux de 1 à 2 ans. Le champignon semblait être un parasite de faiblesse attaquant des rameaux d'arbres ayant subi des stress comme la dessication hibernale, un dégel prolongé anormal en hiver tel que rapporté en 1980-81 (Benoit *et al.* 1981), l'embrun de sel de déglaçage, etc. Le champignon aurait alors profité de l'état de dormance de l'hôte pour s'y développer. C'est du moins l'hypothèse que nous retenons compte tenu de nos observations et des travaux de Schoeneweiss (1981) sur le stress comme agent initiateur de certaines maladies.

Nous avons fait des mises en culture de quelques-uns des spécimens et nous procédons présentement à des tests de comparaison avec d'autres isolats de *T. vulgaris* provenant d'espèces feuillues. Bien que Petch (1940) ait éliminé beaucoup d'espèces de *Tubercularia* dans sa revision du genre, il existe des différences entre les colonies de *T. vulgaris* en culture, différences qu'il est important d'étudier.

[1] *Respectivement pathologiste forestier et biologiste, Centre de recherches forestières des Laurentides, Service canadien des forêts, Environnement Canada, 1080, route du Vallon, C.P. 3800, Sainte-Foy (Québec) G1V 4C7.*

Accepté pour publication le 23 février 1984

Tableau 1. Liste des conifères sur lesquels *N. cinnabarina* fut identifié ainsi que les numéros des collections du Centre de recherches forestières des Laurentides

Hôtes	Numéro d'herbier	Numéro de culture	Date	Localité	Récolté* par	Déterminé par
Larix decidua	17232	–	27-09-82	Saint-Isidore** Dorchester (DR)	A.C.	A.C.
Larix decidua	17099	81-875	14-10-81	Scott-Jonction Dorchester (DR)	A.C.	R.C.
Larix laricina	8698	–	04-09-75	Pointe-au-Chêne Argenteuil (DR)	P.T.	R.C.
Larix laricina	17089	81-925	30-10-81	Lac-Sergent Portneuf (DR)	A.C.	A.C.
Pinus banksiana	–	81-402	16-07-81	Pellegrin Gaspé-Est (DR)	G.L.	R.C.
Pinus sylvestris	17694	–	19-05-83	East-Broughton Beauce (DR)	A.C.	A.C.
Thuya occidentalis	16346	–	24-09-76	Les Saules Québec (DR)	A.C.	E.S.

*A.C.: André Carpentier; R.C.: René Cauchon; G.L.: Gaston Laflamme; E.S.: Edgar Smerlis; P.T.: Pierre Therrien.
**DR: Division de Recensement tel que décrite dans Le "Répertoire toponymique du Québec", 1978.

Références bibliographiques

1 Bedker, P.J., R.A. Blanchette et D.W. French. 1982. *Nectria cinnabarina* The cause of a Canker Disease of Honey Locust in Minnesota. Plant Dis. 66: 1067-1070.

2 Benoit, P., G. Laflamme, G. Bonneau et R. Picher 1982. Insectes et Maladies des Arbres: Québec-1981. For. Conserv. Suppl. 48: 19 pp.

3 Boyce, J.S. 1961. Forest Pathology. 3è ed. McGraw-Hill, Book Company. New York, N.Y. 572 pp.

4 Kujala, V., 1950. Über die Kleinpilze der Koniferen in Finnland. Communicationes Instituti Forestalis Fenniae 38.4:56-57.

5 Lanier, L., P. Joly, P. Bondoux et A. Bellemère. 1976. Mycologie et pathologie forestières. Tome II: Pathologie forestière. Masson. Paris. 478 pp.

6 Manion, P.D. 1981. Tree Disease Concepts. Prentice-Hall, Inc. Englewood Cliffs, N.J. 399 pp.

7 Petch, T. 1940. Tuberculariа. Trans. Brit. Mycol. Soc. 24: 33-59.

8 Pirone, P.P. 1978. Diseases and Pests of Ornamental Plants. 5th ed. John Wiley & Sons, Inc. New York, N.Y.

9 Shoeneweiss, D.F. 1981. The Role of Environmental Stress in Diseases of Woody Plants. Plant Dis. 65: 308-314.

10 Schwerdtfeger, F. 1981. Waldkrankheiten. Paul Parey. Hamburg 4è ed. 486 pp.

11 Tattar, T.A. 1978. Diseases of Shade Trees. Academic Press. New York, N.Y. 361 pp.

A new host and distribution record of a larch needle blight, *Meria laricis* Vuill., in Alberta

P.J. Maruyama [1]

Meria laricis Vuill. was identified in four specimens deposited at the Northern Forest Research Centre, Canadian Forestry Service, Edmonton, Alberta. This is the first record of *M. laricis* in Alberta and the first time it has ever been found on alpine larch (*Larix lyallii* Parl.).

Can. Plant Dis. Surv. 64:1, 19, 1984.

On a identifié *Meria laricis* Vuill. sur quatre spécimens déposés au Centre de recherches forestières du nord, Service canadien des forêts, Edmonton, Alberta. Ceci constitue le premier cas de *M. laricis* rapporté en Alberta, et aussi la première fois que *M. laricis* est trouvé sur le mélèze alpin (*Larix lyallii* Parl.).

Meria laricis Vuill., a needle blight of larch (*Larix* spp.), was first described in France in 1895 (5). This unique fungus is generally classified under Fungi Imperfecti, but some consider it to be a basidiomycete (5). After examining a culture, R.J. Bandoni suggested that this fungus is probably a basidiomycete closely related to Ustilaginales (per. comm. *).

The disease has been reported in Asia, Europe, New Zealand, and North America (1). In North America, Ehrlich (2) reported the disease on western larch (*L. occidentalis* Nutt.) in Idaho and Washington, concluding that it was probably firmly established in the Pacific Northwest. Leaphart (4) lists the disease as also occurring in Canada, and a distribution map prepared by the Commonwealth Mycological Institute (1), which referred to Leaphart's report, recorded the disease in British Columbia. This report of the fungus in British Columbia was not well documented, however, until the disease was positively identified in 1981 from collections on western larch (DAVFP 22583, 22621). The 1981 survey found the disease to be generally present in western larch stands in southeastern British Columbia. The survey suggested that the disease had been present for many years prior to 1981 (3). The disease was also found on several western larch trees planted as ornamentals in Victoria and Duncan on Vancouver Island, and near Harrison Lake, B.C.

Following the confirmation of the fungus in British Columbia, specimens of larch deposited in the Disease Reference Collection of the Northern Forest Research Centre were examined. Four were found to contain *Meria laricis*. One specimen was on western larch collected from Kootenay National Park, B.C. (CFB 1933, 1954), and three specimens were on alpine larch (*L. lyallii* Parl.) collected from Highwood Pass, Alberta (CFB 5481,1962;CFB 21074,1981), and Yoho National Park, B.C. (CFB 21395,1982).

This is the first time *Meria laricis* has been found on alpine larch, and the first record of it in Alberta.

Literature cited

1 Anonymous. 1970. *Meria laricis* Vuill. Map 379, Edition 2 *in* Distribution maps of plant diseases. Commonw. Mycol. Inst., Kew, Surrey, England.

2 Ehrlich, J. 1942. Recently active leaf diseases of woody plants in Idaho. Plant Dis. Rep. 26:391-393.

3 Fiddick, R.L., and G.A. Van Sickle. 1982. Forest insect and disease conditions. British Columbia and Yukon 1981. Environ. Can., *Can. For. Serv., Pac. For. Res. Cent. Victoria, B.C. Inf. Rep.* BC-X-225.

4 Leaphart, C.D. 1964. Diseases of *Larix*. Pages 25-37 *in* Diseases of widely planted forest trees. Proc. FAO/IUFRO Symposium on Internationally Dangerous Forest Diseases and Insects. Section 24: Forest Protection, IUFRO. Oxford, England.

5 Pearce, T.R., and C.H. Holmes. 1933. *Meria laricis*, the leaf cast disease of larch. Oxford For. Mem. 15. 29 pp.

[1] *Northern Forest Research Centre, Canadian Forestry Service, 5320 — 122 Street, Edmonton, Alberta T6H 3S5.*

* *Information contained in a letter from R.J. Bandoni, Department of Botany, University of British Columbia, to Y. Hiratsuka, Northern Forest Research Centre. 1983.*

Accepted for publication February 23, 1984

Epidemiology of barley yellow dwarf virus in Ontario and Quebec in 1982 and 1983 [1]

Y.C. Paliwal [2] and A. Comeau [3]

An epidemic of barley yellow dwarf virus (BYDV) occurred in winter wheat and barley crops in most parts of Ontario and Quebec in 1982-83. Infection of winter wheat averaged 50% but up to 100% BYDV infection was evident in barley fields in south-central Ontario. A vector nonspecific strain of the virus was predominant in winter wheat although levels of a *Rhopalosiphum padi*-specific and a *R. maidis*-specific strains were also significant. The high incidence of BYDV in winter cereals in the autumn appears to have resulted from a large aphid migration in October. In the summer of 1983, there was little evidence of transfer of BYDV from the winter cereals to the spring grains in Ontario. In mid-August BYDV appeared rather suddenly in many areas of Quebec, causing some damage to late-seeded fields.

Can. Plant Dis. Surv. 64:1, 21-23, 1984.

Une épidémie du virus de la jaunisse nanisante de l'orge (VJNO) a atteint le blé d'automne et l'orge d'automne dans la majeure partie du Québec et de l'Ontario en 1982-83. L'infection moyenne du blé d'automne était de 50%, mais une infection de 100% était évidente dans l'orge d'automne de la zone centre-sud de l'Ontario. Une race nonspécifique de virus prédominait chez le blé d'automne, mais avec une présence significative d'isolats spécifiques à *Rhopalosiphum padi* et à *R. maidis*. Le niveau élevé de VJNO chez les céréales d'automne résultait apparemment d'une forte migration de pucerons en octobre. À l'été de 1983, le transfert de VJNO des céréales d'automne aux céréales de printemps a été peu évident en Ontario. Cependant, au milieu d'août, le VJNO apparut assez subitement dans plusieurs régions du Québec, causant des dommages aux champs semés tardivement.

Introduction

After the 1976 epidemic of barley yellow dwarf virus (BYDV) in eastern Canada (4, 7), the disease was present only at low levels in Quebec from 1977 to the summer of 1982. In Ontario, scattered infection reaching 10% occurred in 1979, and local problems developed in the southern part in 1981 and 1982, but no widespread epidemic was observed. However, during the autumn of 1982, the presence of BYDV increased over wide areas of Eastern Canada. The present report deals with observations on an epidemic affecting winter cereals in 1982-83, and an assessment of the transfer of BYDV from winter cereals to spring cereals in 1983.

Observations and Tests

Winter cereals, 1982-83

In Ontario, many warm spells occurred in the fall, and above average temperatures prevailed up to late November. Aphid populations in the maturing spring cereals, wild grasses and the winter wheat were low during the August-October period. A sudden increase in the numbers of aphids on winter wheat due to arrival of wind blown migrant aphids was noted in mid-October to early November. The first BYDV symptoms became visible in winter wheat from November 1 to 15 in different areas. Aphid counts in mid-November ranged from 15 to 150 aphids per meter of row, mostly *Rhopalosiphum padi* L. and some *R. maidis* (Fitch.). Samples of winter cereals were tested for the presence of BYDV and strains of the virus were identified following the procedures reported earlier (6, 7). An average of 50% infection was observed in wheat, and 69% of the field aphids were positive for BYDV. Three vector specific and a vector nonspecific strains of the virus were detected (Table 1).

In Quebec, abundant flights of *R. padi* were encountered between October 5 and 12 in the Quebec city area. The fallout of aphid alates was approximately 50 to 80 aphids per square meter per day for at least 4 consecutive days, according to counts made on five flats of 0.16 m^2 area containing about 60 *Agropyron* plantlets per flat. Eventually the population built up to more than 10 aphids per plant in nearby fields of winter cereals. By November 5, the winter barley plots were showing the severe BYDV symptoms with an estimated 100% infection. BYDV symptoms were common in winter wheat fields also. Triticale cultivar OAC Wintri and all rye lines appeared healthy. One area of 375 m^2 of winter wheat which was sprayed with pirimicarb in September initially looked virus-free but, by November 5, BYDV infected plants were abundant and 1-5 aphids/plant were counted in the field indicating significant BYDV infection despite the use of an aphid-specific insecticide. BYDV symptoms on wheat were also observed at St-Augustin (near Quebec city), La Pocatière, northeast of Quebec city, and at St-Hyacinthe, and all plants were infested with aphids. Three randomly collected wheat plants from La Pocatière were indexed for BYDV and were found to be infected.

The winter of 1982-83 was unusual in having high temperatures in the later half of December, with rain that caused ice accumulation in many fields. The spring was very cold and rainy at first, and these factors combined with BYDV

---

[1] *Contribution No. 1449, Chemistry and Biology Research Institute.*

[2] *Chemistry and Biology Research Institute, Agriculture Canada, Ottawa K1A OC6.*

[3] *Station de recherches, Agriculture Canada, Sainte-Foy, Québec G1V 2J3*

Accepted for publication February 28, 1984

Table 1. Barley yellow dwarf virus in cereals in Eastern Ontario, 1982-1983

Location (County)	Field Sample Type (Plant/Aphid)	No. infected/ No. tested*	Virus strains† identified				
			NS	RPS	RMS	SAS	Total[c]
Winter Wheat (Nov. 1982)							
Ottawa-Carleton	Wheat	9/18	4	3	1	1	9
Ottawa-Carleton	*Rhopalosiphum padi*	3/ 5	2	2	0	0	4
Ottawa-Carleton	*R. maidis*	4/ 5	1	0	4	0	5
Renfrew	Wheat	2/ 4	2	0	1	0	3
Renfrew	*R. padi*	4/ 6	3	2	0	0	5
Total		22/38	12	7	6	1	26
Winter Wheat (June, 1983)							
Ottawa-Carleton	Wheat	15/17	—	—	—	—	—
Spring Cereals (June-July, 1983)							
Ottawa-Carleton	Barley	2/15	2	0	2	0	4
Ottawa-Carleton	Oats	1/16	1	0	0	0	1
Glengarry	Wheat	4/18	1	0	0	4	5
Total		7/49	4	0	2	4	10

* Number of randomly collected field plant samples found infected with the virus/number of samples tested in transmission tests with four species of aphid vectors (Paliwal 1982a,b). In case of aphid samples from the fields, the figures represent number of plants infected/ number of plants infested with two field aphids per plant.

† NS, vector nonspecific; RPS, *R. padi* specific; RMS, *R. maidis* specific; SAS, *Sitobion avenae* specific strains.

[c] Total number of virus strain types recovered from the field samples or from plants infected in tests of field aphids are sometimes greater than the number of infected plants used as sources for strain identification due to recovery of more than one strain from some samples.

infection resulted in high winter kill (60-100%) of winter cereals in most parts of the Quebec province. A limited survey of winter cereals in Quebec and Ontario on June 15 and 16 revealed that at Ste-Rosalie, Que., winter wheat showed heavy winter damage and many surviving plants had BYDV symptoms. Winter rye observed at Ste-Rosalie, Guelph, Ont. and Chatham, Ont. did not show BYDV symptoms. Significant levels of BYDV infection was also noted in many winter wheat fields between Toronto and Chatham. Late-seeded winter wheat fields showed less BYDV damage than the early-seeded fields, similar to that reported in U.S.A. (2). In the Guelph University fields near Guelph, the rate of BYDV incidence in winter barley was nearly 100%, although the severity of symptoms was variable probably due to the presence of several strains of the virus. Overall disease severity was so high that a yield loss of about 90% could be realistically predicted. The winter triticales showed some evidence of BYDV infection, but generally they appeared more tolerant than winter wheat. The interpretation of BYDV incidence in winter wheat was complicated by the presence of some advanced symptoms of wheat spindle streak mosaic virus. The late-seeded wheat seemed to have largely escaped damage in the Guelph area, similar to what was observed in other parts of Ontario. Randomly collected winter wheat plants were indexed for BYDV in the Ottawa-Carleton in June 1983, and the rate of infection observed was very high (Table 1).

Spring cereals, 1983

Many fields of spring cereals were planted at unusually late dates in Ontario. In mid-June, there was no evidence of transfer of BYDV from winter wheat to spring cereals in Ontario. Small numbers of aphids were present in winter wheat near Chatham [*R. padi* and *Sitobion avenae* (Fab.)] and about 5% of nearby oats at the tillering stage showed BYDV symptoms. Infection became somewhat more widespread later in the summer. Incidence of aphids was low in other parts of Ontario also. Apparently, the hot weather prevailing from June 10 to July 15 retarded the development of aphids, so that BYDV was not transferred from winter cereals to spring cereals. This assessment is based on visual observations and on evidence obtained by indexing random samples of Ontario spring cereals for the presence of BYDV which yielded a 14% infection estimate (Table 1). Also, the spectrum of BYDV strains identified from the winter wheat and those detected in spring cereals was different.

In Quebec, the authors did not monitor closely the June aphid migrations, but casual observations indicated that little migration occurred. On July 15, scattered individuals of *R. padi* were observed at Deschambault with less than 1% BYDV infection in the fields, but in the Montreal area there were up to 30 aphids per meter of row of oats, and about 20% BYDV infection of plants at flowering to early milk stages was recorded. In early August, reports of BYDV symptoms came from Témiscamingue, Lac St-Jean and Eastern Townships (Cantons de l'Est) and the Québec-Montmagny area. BYDV infected plants were widespread in the oat fields in Lac St-Jean and Eastern Townships area. Late seeded fields suffered significant damage. Since there was no evidence of primary infection, this BYDV incidence is believed to be due to

a late but substantial migration of viruliferous aphids over a wide area in mid-July. At that time, the spring cereals in Ontario were ripening and winter cereals had largely been harvested.

The summer months in Quebec and Ontario were characterized by above average temperatures and drought which also adversely affected the yield of spring cereals. In fields most severely affected by drought, the BYDV symptoms were masked by the effects of the drought. Aphid populations and migrations in September and October of 1983 were lower than average, and the incidence of BYDV winter wheat fields in Quebec and Ontario was low.

Discussion

Large, unpredictable aphid migrations are increasingly evident as the main cause of BYDV epidemics and short-range transport of BYDV seems to have only minor importance (7 and the present study). Development of heavy aphid populations within a field is generally prevented mainly by biological control agents, but when aphids migrate, they apparently escape to some extent from one part of the biological control complex, namely the Coccinnellids, the Syrphids and the Cecidomyids. The alates may include some individuals parasitized by young larvae of Aphidiid wasps, and it is quite possible that this parasitism results in a reduced ability of the aphid for sustained flight, so that the migrants may end up with reduced levels of parasitism also. The end result is that after migrants alight on a new crop, often hundreds of kilometers away from their starting point (8), they may enjoy a few days of near-optimal reproduction conditions without much interference from their natural enemies.

The geographic area of origin of the aphids affecting Ontario and Quebec winter wheat in the fall of 1982 and the Quebec spring cereals in the summer of 1983 cannot be pinpointed accurately, as there is no systematic aphid trapping system established for this purpose in North America. However, wind directions data contains a dominant south-west or west component from May to September in Quebec and Ontario, according to 30-year averages for most stations in the cereal-growing areas. In the first weeks of October 1982, when the aphid migration was observed, a steady southwesterly wind was encountered in Quebec.

The presence of a heavy epidemic on winter-cereals in 1982-83 did not result in a general BYDV epidemic in Ontario spring cereals despite the fact that there are more winter cereals than spring cereals in Ontario. Aphids migrating out of Ontario winter wheat, if present there in significant numbers, could have been regarded as a serious threat, as in France, aphids coming from small grain cereals contain much more BYDV than aphids coming from maize (1, 5). In Quebec, winter cereals represent very small areas and have no importance as a BYDV reservoir. The fact that BYDV reached rather high levels in many localities of the province of Quebec about mid-July of 1983 indicates that short-range contamination may have much less importance than long-range migrations between localities along the axis of prevailing winds.

Aphid numbers alone are not a good predictor of BYDV epidemic as in the fall of 1979 there was also a heavy aphid migration and a buildup reaching 40 aphids per plant on winter cereals in parts of Quebec but the aphids were essentially virus-free. This may be related to the nature of the crop on which these aphids were previously feeding, as maize for example contains low levels of BYDV and a limited range of virus strains (5, 7).

The routine use of insecticides on winter cereals to control aphids does not seem logical, considering the actual frequency of BYDV epidemics in Eastern Canada and the fact that in many instances the aphids may be predominantly virus-free. However, these observations indicate a need for closer monitoring of aphid migrations and for rapid indexing of aphid populations to determine the percentage of viruliferous aphids so that aphicide applications can be accurately timed and applied only if warranted in order to arrest an aphid population explosion leading to a BYDV epidemic.

Acknowledgements

The authors thank Lloyd Seaman, Luc Couture and J.P. Dubuc for some of the observations and for supplying some field samples.

Literature cited

1 Bayon, F., J.-P. Ayrault and P. Pichon. 1982. La jaunisse nanisante de l'orge. Phytoma — Défense des Cultures, Nov. 1982, p. 17-21.

2 Carrigan, L.L., H.W. Ohm, J.E. Foster and F.L. Patterson. 1981. Response of winter wheat cultivars to BYDV infection. Crop Sci. 21: 377-380.

3 Comeau, A. and C.-A. St-Pierre. 1984. Report no. 5. Trials on the resistance of cereals to barley yellow dwarf virus (BYDV). Agriculture Canada, Sainte-Foy, Québec.

4 Comeau, A. and J.-P. Dubuc. 1977. Observations on the 1976 barley yellow dwarf epidemic in Eastern Canada. Can. Plant Disease Survey 57: 42-44.

5 Lapierre, H. 1980. Les virus des céréales à paille. Phytoma — Défense des Cultures, Sept.-Oct. p. 34-38.

6 Paliwal, Y.C. 1982a. Identification and annual variation of variants of barley yellow dwarf virus in Ontario and Quebec. Can. J. Plant Pathol. 4: 59-64.

7 Paliwal, Y.C. 1982b. Role of perennial grasses, winter wheat, and aphid vectors in the disease cycle and epidemiology of barley yellow dwarf virus. Can. J. Plant Pathol. 4: 367-374.

8 Wallin, J.R. and D.V. Loonan. 1971. Low level jet winds, aphid vectors, local weather, and barley yellow dwarf virus outbreaks. Phytopathology 61: 1068-1070.

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.