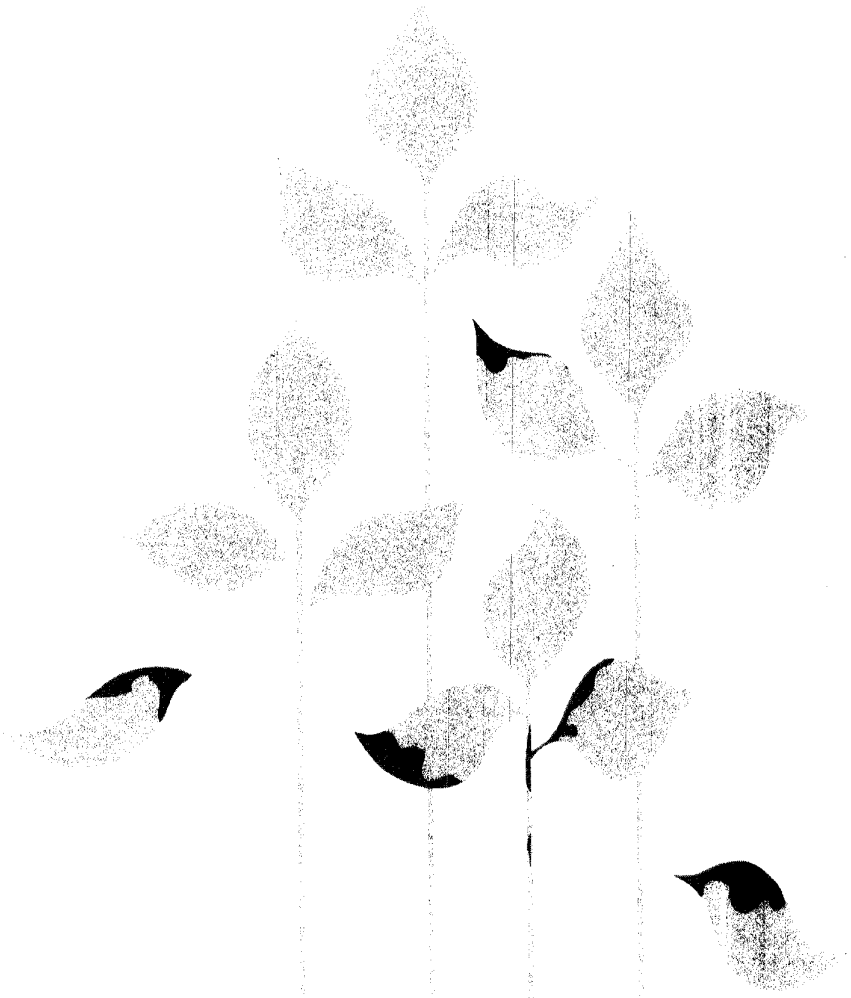
Canadian Plant Disease Survey

Inventaire des maladies des plantes au Canada

Vol. 61, No. 1, 1981

Vol. 61, N° 1, 1981



Agriculture Canada

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Volume 61, Number 1, 1981
CPDSAS 61(1) 1-21 (1981) ISSN 008-476X

Volume 61, Numéro 1, 1981

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 will also be accepted. Review papers and compilations of practical value to plant pathologists will be included from time to time.

Research Branch, Agriculture Canada
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Editorial Board: R. Crête, J.W. Martens, J.T. Slykhuis

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. De temps à autre, l'inventaire inclut des revues et des synthèses de rapports d'intérêt immediat pour les phytopathologistes.

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ERRATA

Volume 60:4, 1980

- P. 43 *G.S.V. Raghaven* should be *G.S.V. Raghavan*

- P. 44 equation (1) should be

$$RRDI = \frac{[\Sigma \text{(no. plants per category) X (no. of category)}]X\ 100}{\text{(number of categories - 1) X (total no. of plants)}}$$

Phomopsis canker on weeping fig in Newfoundland[1]

Michael C. Hampson[2]

Phomopsis cinerescens and a *Botryodiplodia* sp. were isolated in pure cultures from cankers on *Ficus benjamina*, weeping fig tree, grown in a shopping mall, St. John's, Newfoundland. Analysis of the growing conditions suggested the trees were being subject to stress induced by light, nutrient and medium factors, *inter alia*. This report is the first of *P. cinerescens* on *F. benjamina* in Canada. It is hypothesised that the fungus was introduced with the trees, penetrated through bark wounds, and incubated under conditions stressful to the well-being of the tree.

Can. Plant Dis. Surv. 61:1, 3-5, 1981.

On a isolé *Phomopsis cenerescens* et une espèce de *Botryodiplodia* en cultures pures de chancres infestant *Ficus benjamina*, figuier pleureur cultivé dans un mail commercial de Saint-Jean (Terre-Neuve). L'analyse des conditions de croissance donnet à penser que les arbres ont fait l'objet d'un stress provoqué par certains facteurs comme, entre autres, l'éclairement, les apports nutritifs et le substrat. Il s'agit de la première mention de la présence de *P. cinerescens* sur *F. benjamina* au Canada. Il semblerait que le champignon a été introduit avec les arbres, a pénétré par les lésions de l'écorce et a profité de conditions stressantes (pour les arbres) pour entrer en incubation. On recommande de la bouillie bourguignonne comme moyen de lutte.

Introduction

Weeping fig or banyan tree (*Ficus benjamina L.*) displays an epiphytic habit (17) in its native environment (Malaysia) and Bailey (2) described it as a rather unimportant tree horticulturally. In the last decade, the weeping fig has become somewhat ubiquitous as a specimen tree in offices and shopping malls (9), and the object of several studies related to its acclimatization and culture in an artificial environment (5, 6, 10). In this novel environment it is said to be trouble-free but "meticulous in its demands" for light, water, nutrients and constancy of temperature (9).

Ten specimens of weeping fig were shipped to St. John's, Newfoundland, in a constant temperature refrigerated van (temperature unknown) from Florida in August/September 1978 for display in a local shopping mall. Transhipment time was 10 days. Specimens were planted in above-ground (1 m^3, Fig. 1A, a) or below-ground (2 m^3) concrete containers. The planting medium was soil amended with poultry manure and lime, and topped with a 10 cm layer of bark chips. The large containers were protected by grill-work (Fig. 1A, b). Tree height ranged from ca. 3 m to 9 m; the span of the tallest specimen was ca. 5 m. The trees were watered, misted and supplied with soluble 20:20:20 weekly. During the following months the trees produced abundant, healthy-looking foliage.

The purpose of this paper is to describe an outbreak of phomopsis canker on these interior-grown weeping fig trees, and to draw attention to this first report of the causal agent in Newfoundland.

Materials and methods

In April 1980 (20 m after planting in the shopping centre), a 4-m long branch exhibited wilt (Fig. 1A): 24 hr. later the foliage was dead. At the base of the branch a 40 cm × 10 cm lozenge-shaped diffuse, darkened canker, slightly sunken, was found (Fig. 1B). After the branch was removed, extensive discoloration was found to extend for more than 2 m above the canker, basipetally into the trunk (Fig. 1C), and radially more than 3/4 cross-sectional area.

On examining the remaining trees, other cankered areas were identified which were mainly in the lower parts of the trees. Small areas were found on some mid-tree branches. Discolored patches were also found that did not fit the generalized shape of a diffuse canker (Fig. 1D), and browning was evident under the bark of these areas. After two months observation, more of these anomalous discolorations became apparent, their shapes and directions of slant being very suggestive of pressure by rope or some similar binding material. Likewise, after two months, an excised canker (25 cm) was found which displayed further growth of 2-3 cm into the surrounding bark.

The most severe cankers were excised with a sterilized pruning knife. Exposed areas were sterilized with shellac paint and, although not recommended by Tattar (16), a number of treated areas were covered with asphalt paint. Fungicidal treatments are also available (12, 13).

Stem and bark tissue pieces were surface-sterilized (10% javex, 1 min.) and plated out on freshly poured PDA. Dark/black colonies were obtained: two fungi were identified, *Botryodiplodia* sp. and *Phomopsis cinerescens* (Sacc.) Trav. (P. LeClair, personal communication).

Discussion

Although both genera contain parasitic members, *P. cinerescens* is the primary cause of fig canker (1). The association of *Botryodiplodia* and *Phomopsis* spp. is an

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Accepted for publication December 1, 1980

interesting one since a similar association was demonstrated in the etiology of rose stem dessication in Brazil (14). Circumstances curtailed the application of Koch's postulates to the fig complex.

Phomopsis is the imperfect stage of *Diaporthe*, a cosmopolitan fungus with a broad host range. *Phomopsis* spp. have been associated with leaf spots, twig blights, dieback, stem galls and cankers (3, 7, 13). Of the wide group of plants which display these symptoms, *Phomopsis* has been reported in Canada only on Juniper, Siberian Crabapple and cultivated apple, *Cupressus macrocarpa*, gardenia, pine and Douglas-fir (4). The only previous report of *Phomopsis* from Newfoundland was of *P. junipervora* on imported ornamental junipers (15); the other occasions were reported from Nova Scotia, New Brunswick, Quebec, Ontario, Manitoba and British Columbia (4). *Phomopsis* spp. have recently gained recognition as producers of hepatotoxins injurious to farm animals (8).

Phomopsis cinerescens has not been reported on *Ficus benjamina*, and not at all from Canada; of 201 references to *Phomopsis* spp. between 1970 and 1978, none referred to *P. cinerescens*. *P. cinerescens* has been isolated from *Ficus carica* in Europe where it is responsible for fig branch and twig canker; in North America the fig is chiefly cultivated in California, Louisiana and Texas (1). It is concluded, therefore, that *P. cinerescens* has a limited distribution, and was probably imported into Newfoundland with the host trees, as was surmised for *P. juniperova* (15).

Tattar (16) emphasized stress and vigor in the etiology of tree diseases. In acclimatization work (5, 6, 10) with *F. benjamina*, it was demonstrated that shade (duration, intensity), fertilizer, rooting medium, and watering influenced leaf density and drop, root/leaf carbohydrate ratios and tissue mineral levels. Considering the stresses applied to the affected trees through transport, mechanical injury and environmental factors, it is highly likely that the trees were predisposed to infection. For example, at noon on a clear day (July 31), 5% of the foliage was sunstruck and registered 43 klx, 2.4 m away the light registered 1 klx; this must be compared with Conover and Poole's (6) reading of 30 klx for 30% sunlight on experimental trees. *Phomopsis* spp. are wound invaders (7, 11). Since *P. cinerescens* is an introduced species and the hosts appear to have suffered laceration damage, it is suggested the fungus was present at time of original shipment, entered through wounds, and developed under a stress syndrome.

Acknowledgements

I wish to acknowledge the assistance of Dr. J. Bissett, Biosystematic Research Institute, Agriculture Canada, Ottawa, and Mr. D. Thompson, Manager, Village Shopping Mall, St. John's.

Literature cited

1. Anon. 1960. Index of plant diseases in the United States. ARS, USDA Agric. Handbook No. 165. p. 531.
2. Bailey, L.H. 1915. The standard cyclopedia of horticulture. The Macmillan Co., New York. Vol. III - F-K:1201-1760.
3. Butler, E.J., and S.G. Jones. 1961. Plant Pathology. Macmillan and Co. Ltd., London.
4. Conners, I.L. 1967. An annotated index of plant diseases in Canada. Canada Dept. Agri. Publ. 1251. 381 p.
5. Conover, C.A., and R.T. Poole. 1975. Acclimatization of tropical trees for interior use. Hortscience 10:600-601.
6. Conover, C.A., and R.T. Poole. 1977. Effects of cultural practices on acclimatization of *Ficus benjamina* L. J. Amer. Hort. Sci. 102:529-531.
7. Forsberg, J.L. 1975. Diseases of ornamental plants. University of Illinois Press, Urbana, I11. 220 p.
8. Lanigan, G.W., and A.L. Payne. 1979. Phomopsin A production by *Phomopsis leptostromiformis* in liquid media (Fungal parasite of lupine causing lupinosis in sheep). Appl. Env. Microbiol. 37:289-292.
9. Melbourne, R. 1976. Houseplants: *Ficus benjamina*, darling of decorators. Pac. Hort. 37:30-31.
10. Milks, R.R., J.N. Joiner, L.A. Garard, C.A. Conover, and B. Tija. 1979. Influence of acclimatization on carbohydrate production and translocation of *Ficus benjamina* L. J. Amer. Soc. Hort. Sci. 104:410-413.
11. Morehart, A.L., R.B. Carroll and M. Stuart. 1980. *Phomopsis* canker and dieback of *Elaeagnus angustifolia*. Plant Disease 64:66-69.
12. Peterson, G.W., and J.D. Otta. 1979. Controlling phomopsis blight in junipers. Am. Nurse. 149(5):15, 75, 78, 80-82.
13. Pirone, P.P., B.O. Dodge, and H.W. Rickett. 1960. Diseases and pests of ornamental plants. Ronald Press Co., New York. 776 p.
14. Pitta, G.P.B. and J. Teranishi. 1973. *Phomopsis* and *Botryodiplodia* associated with rose stem dessication (In Portuguese, Eng. summary). Biologico 39:21-23.
15. Singh, P. 1978. Phomopsis blight of eastern white cedar in Newfoundland. Bi-monthly Res. Notes, Nfld. For. Res. Cent. 34:3.
16. Tattar, T.A. 1978. Diseases of shade trees. Academic Press, Inc. New York. 361 p.
17. Willis, J.C. 1951. A dictionary of the flowering plants and ferns. Cambridge University Press, Cambridge. 701 p.

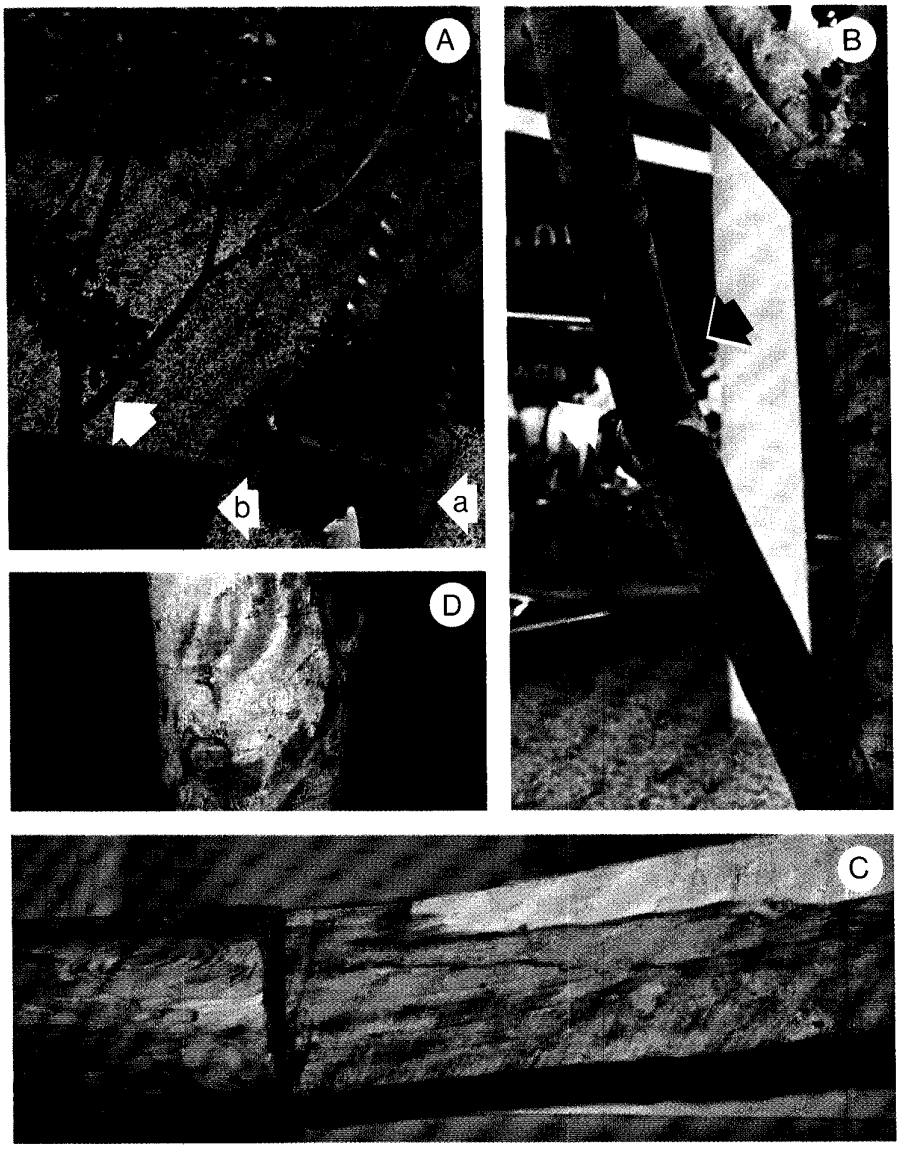

Figure 1. (A), *F. benjamina* branch exhibits wilt and foliar dessication. General view of large potted tree, a - 1 m^3 container, b - grill-work. (B), Large, 40 X 10 cm, canker at base of branch exhibiting wilt. (C), Cross and longitudinal sections of branch displaying extent of internal discoloration. (D), An excised canker displaying further growth, 2-3 cm, two months after excision.

Ascochyta blight of lentils in western Canada: 1978 to 1980

R.A.A. Morrall[1] and J.W. Sheppard[2]

Ascochyta blight of lentils was reported in Canada for the first time in 1978. Surveys of seed samples clearly demonstrated that the disease was already widespread in Saskatchewan and Manitoba. The main symptoms of ascochyta blight consist of purplish-brown shrunken seed and white to tan-colored leaf, stem and pod lesions, which usually become speckled with black pycnidia. The causal organism is similar morphologically to *Ascochyta fabae* Speg. sensu Boerema & Dorenbosch but the authors prefer to retain the name *A. lentis* Bondartzeva-Monteverde & Vassilievsky because of the apparent host specialization of the pathogen. A field survey in Saskatchewan in the summer of 1979 showed very low levels of ascochyta blight in southern and west-central areas. More disease was present in the moister region north of Saskatoon. Reduction in seed quality due to the disease is probably more important than yield losses.

Can. Plant Dis. Surv. 61:1, 7-13, 1981.

La brûlure ascochytique de la lentille a été identifiée pour la première fois au Canada en 1978, alors que des échantillonnages de semences montraient que la maladie était déjà répandue en Saskatchewan et au Manitoba. Les principaux symptômes de cette maladie sont une coloration blanche à brun clair des feuilles, l'apparition sur la tige et les gousses de lésions habituellement parsemées, à maturité, de pycnides noirs et une décoloration brun pourpre et un rabougrissement des graines. La pathogène est morphologiquement semblable à *Ascochyta fabae* Speg. sensu Boerema & Dorenbosch, mais nous préférons lui conserver le nom de *A. lentis* Bondartzeva-Monteverde & Vassilievsky, à cause de sa spécificité apparente en ce qui concerne l'hôte. Une enquête de terrain effectuée en Saskatchewan durant l'été 1979 a indiqué un très faible taux d'infection dans le sud et le centre-ouest de cette province mais les régions plus humides au nord de Saskatoon ont été plus touchées. La baisse de la qualité des semences causée par la brûlure sera probablement plus importante que la réduction du rendement.

Introduction

Lentils have been grown commercially in western Canada since 1970. Despite fluctuations in the area planted in the first seven years, lentils now represent a well-established cash crop in several regions. The estimated Canadian hectarage for 1980 is 30,000 compared with 19,000 in 1979 and 10,000 in 1978 (Sask. Pulse Crop Growers Assn. Newsletter No. 43, Feb. 1980). The majority of these areas are in Saskatchewan. Surveys of commercial lentil fields in the early and mid-seventies (8, 9, 14, R.A.A. Morrall, unpublished) showed that the crop was relatively disease free, particularly with respect to foliar diseases. Before 1978 the only significant epidemics in western Canada were severe sclerotinia stem rot in isolated fields in Manitoba (A.E. Slinkard, personal communication).

In September 1978 a sample of severely discolored lentil seeds was received from Laird, Saskatchewan, about 70 km north of Saskatoon. Isolations were made from surface disinfected seeds on 20% V8 juice agar (V8). Ninety eight percent of the discolored seeds and 48% of the normal seeds in the sample yielded an *Ascochyta* sp. which caused lesions on roots and shoots developing from seeds in the isolation plates. The same fungus was isolated from leaf and stem lesions on volunteer lentil seedlings and lentil re-growth in the Laird district in early October 1978. Koch's postulates were fulfilled for these *Ascochyta* isolates by inoculating commercial lentil seedlings in pots in the greenhouse. Crude spore suspensions prepared from V8 plate cultures of the fungus were sprayed on 2-week old seedlings and the pots were covered for 24 hours with plastic bags. After 10 - 14 days lesions appeared on the leaves and stems and the same *Ascochyta* was again isolated from them.

Further investigation demonstrated that this was the first record in North America of ascochyta blight of lentils, an important disease known in other parts of the world (2, 4, 6, 7, 11, K. Davatzi-Helena, personal communication). The origin of the disease in Saskatchewan is unclear. Earlier surveys (8, 9, 14) failed to reveal its presence, but subsequent seed tests demonstrated clearly that it had been widespread in 1978. The grower who submitted the original discolored seed sample claimed to have observed a similar problem in 1977. Prior to that, he had grown lentils since 1972, consistently using his own seed.

The purpose of the present paper is (a) to describe the symptoms of the disease, (b) to discuss briefly the morphology and taxonomic position of the pathogen, (c) to report on surveys of seed samples and of commercial fields conducted in 1979 and 1980, and (d) to discuss control of the disease. A preliminary report on parts of the work has already been presented (15).

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Accepted for publication March 18, 1981

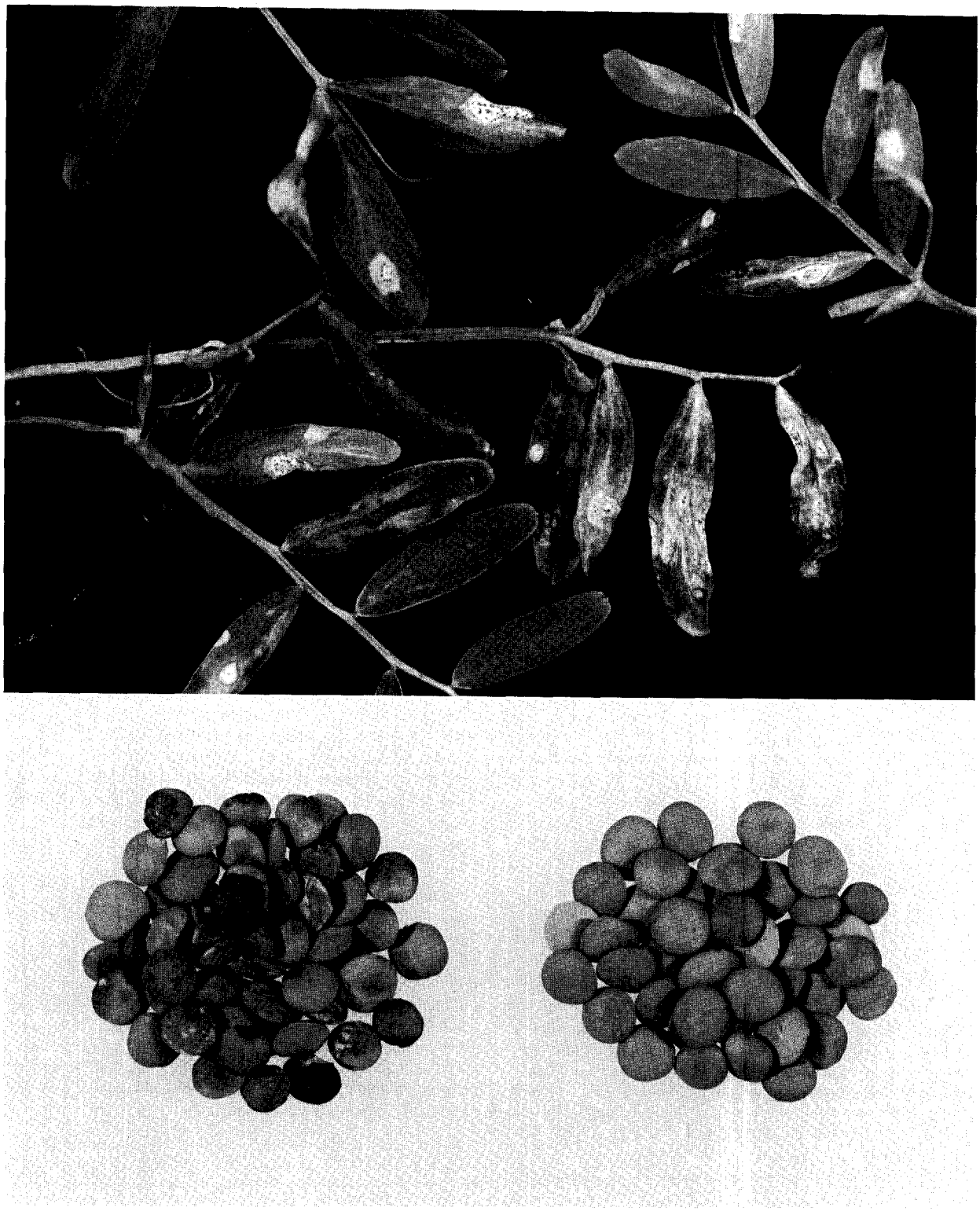

Figure 1. Lesions on lentil leaflets caused by *Ascochyta* infection. Note prominent pycnidia in lesions. (X3)

Figure 2. Healthy lentil seed and seed severely infected by *Ascochyta*. Note discoloration, and shrivelling of infected seed and presence of white mycelial flecks. (X2)

Symptoms of the disease

Lesions on the stems and leaves are initially whitish to greyish becoming light tan colored. Mature lesions usually have darker margins and the centres are speckled with pycnidia (Fig. 1). The pycnidia may be scattered or in concentric circles. Coalescing lesions cause blighting and leaflet abscission. Lesions on pods are generally darker than those on leaves; after the pods have ripened the infected areas tend to have a purplish hue. Severely infected seeds are shrivelled and show a purplish brown discoloration, sometimes in patches and sometimes covering the entire seed (Fig. 2). Occasionally pycnidia and small flecks of mycelium are present on the seed surface. Seeds fail to germinate if they are badly shrivelled and discolored.

Review of the morphology and taxonomic position of the pathogen and names of the disease

The first report of *Ascochyta* on lentils was from Russia in 1938 (4) when the pathogen was described as a new species, *Ascochyta lentis* Bondartzeva-Monteverde & Vassilievsky. The symptoms described are similar to those observed in Saskatchewan, and the description of the pathogen suggests that the differences from Saskatchewan isolates, if any, are only minor. *Ascochyta lentis* from Russia was described as producing 2-celled conidia 11.5-19.5 × 3.5-5.8 μ on lentils, or 13.5-17 × 4-5.7 μ in oat agar culture (4). Saskatchewan isolates generally produce conidia 10-20 × 4-8 μ (Mean: 15.8 × 5.7 μ) on lentil plants and there are a few multiseptate conidia among a majority of 2-celled forms. Unfortunately, we do not know the exact formula of the oat agar used by the Russian workers, but we have tested Saskatchewan isolates on three oat-based agar media (1, 17). On oat agar (1, p. 242) and oatmeal agar (17, p. 51) mycelial growth is dense and sporulation is profuse; however, on oatmeal agar (Shirling and Gottlieb No. 3) (17, p. 51) mycelial growth and sporulation are sparse. There are no significant differences between the three media in conidial dimensions, which are 10-23 × 4-8 μ (Mean 15.6 × 5.9 μ). Multicellular conidia occur occasionally. On V8, Saskatchewan isolates grow well and sporulate profusely; the conidia average 14.7 × 4.1 μ and multicellular forms are slightly more common than on lentil plants or oat-based media. A disease with the same symptoms as those described in Russia and Saskatchewan has been referred to in Argentina either as lentil stain (2) or lentil spot (11) and has been attributed to *A. lentis*. One report from India described a disease with similar symptoms which was called lentil blight; the pathogen was referred to as *A. pisi*, but the figures and description in the text (7) leaves some doubt about the identity. The fungus has larger and more frequently multi-septate conidia than *A. pisi* Lib. fide Jones (5) and is probably identical to the fungus from Saskatchewan. On the other hand, a recent general publication on lentil pathology from India (6) refers to the disease as ascochyta leaf blight caused by *A. lentis*.

Cultures of the lentil ascochyta from Saskatchewan have been examined by Drs. J. Bissett (Biosystematics Research Institute, Ottawa) and H.A. van der Aa (Centraalbureau voor Schimmelcultures, Baarn) and compared with related ascochytas from legumes. They both express the opinion that the fungus should be referred to *A. fabae* Speg. sensu Boerema & Dorenbosch (3), which has conidial dimensions of about 15 × 4-5 μ. Dr. Bissett believes that *A. lentis* is possibly a synonym of *A. fabae*. On the other hand, a recent Russian monograph on *Ascochyta* (10) places *A. lentis* in synonymy with *A. boltshauseri* Sacc. fide Sprague (16). In view of these different taxonomic opinions and the fact species delimitation in *Ascochyta* has frequently been based on host specialization, the present authors are reluctant to reject the name *A. lentis* in favor of *A. fabae* or *A. boltshauseri*. Bondartzeva-Monteverde and Vassilievsky (4) mentioned originally that *A. fabae* and *A. lentis* were specific to broad bean and lentil respectively and the lentil *Ascochyta* from Saskatchewan appears to be quite host specific. To date we have been unable to find the same fungus on other native or cultivated legumes in Saskatchewan. In preliminary cross-inoculation tests in the greenhouse using isolates of *A. pinodes* Jones and *A. pisi* from peas, *A. fabae* from fababeans in western Canada and the lentil *Ascochyta* from Saskatchewan, we have induced symptoms only on the original hosts. The results with *Vicia faba* cv. Petite Tic Bean were particularly dramatic. This cultivar is highly susceptible to *A. fabae* and is used at Saskatoon as a "spreader" in disease nurseries. Whereas Petite Tic Bean showed severe leaf and stem spotting and leaf abscission when inoculated with a mixture of isolates of *A. fabae*, it showed no symptoms at all when inoculated with *Ascochyta* from lentils.

The lentil *Ascochyta* and *A. fabae* are somewhat different culturally when grown on V8, but there are microscopic similarities between the two. Thus, despite the apparent host specialization the possibility of *forma speciales* or even physiologic races of one species cannot be ruled out. However, until more isolates of *Ascochyta* from fababeans, lentils and other legumes have been studied and their host ranges tested we are willing to propose erecting *forma speciales* when there is little taxonomic precedent in the genus *Ascochyta*. We intend to retain the name *A. lentis* for the isolates from lentils in western Canada. Further work is in progress in the senior author's laboratory which will lead to a detailed description of the cultural, morphological and pathogenic characteristics of *A. lentis* from western Canada.

Surveys of seed samples

Saskatoon

Once the potential of the disease for serious seed discoloration was realized, it was decided to determine if the Laird outbreak in 1978 was an isolated occurrence. Therefore, from February to early May 1979 a survey of seed samples from the 1978 crop in western Canada was undertaken at Saskatoon.

Commercial seed samples were obtained from various parts of Saskatchewan through Pioneer Grain Ltd., one of the major lentil contracting companies in 1978. Random subsamples of 200 seeds were taken from each sample, surface disinfected for 10 min. in 0.6% NaOCl, plated on V8 and incubated on the laboratory bench for at least one

week. Colonies of *Ascochyta* on the plates were counted to derive percentage infection figures. Where *Ascochyta* was not obtained from a subsample, but discolored seeds had been observed in the original sample, isolations were made selectively from the discolored seeds. If these yielded *Ascochyta* the infection was recorded as a trace. The results (Table 1) showed that the disease had been widespread in Saskatchewan in 1978. At least traces of infection occurred in seed from six geographic regions; several samples from central Saskatchewan showed high levels of infection and severe discoloration.

The second part of the survey conducted at Saskatoon involved lentils destined for seed use in 1979 in western Canada. Samples were solicited from contracting companies and seed suppliers, and, where possible information on the geographic origin of the seed was obtained. Nineteen samples were submitted and subsamples of seeds were plated on V8, as before. Some samples were so severely discolored that it was clearly unnecessary to plate 200 seeds to obtain a reliable estimate of percentage infection with *Ascochyta*. Thus, the estimates (Table 2) were based on subsamples of from 100 to 400 seeds. Again it was evident that the pathogen had been widely distributed in 1978 and it was disturbing to see the extremely poor quality of seed that some contractors were handling. In all, 74% of the samples carried the pathogen; the most heavily infected were from central Saskatchewan.

Table 1. Incidence of Ascochyta in 1978-grown commercial lentil samples from Saskatchewan growers*.

Saskatchewan Crop District	Geographic location in arable part of Saskatchewan	No. samples tested	No. samples with Ascochyta infection	No. of samples in different % infection categories					
				0%	Trace***	0.5%	0.75–2.5%	2.75–5.75%	〉6%
2	S. Central	2	2				1	1	
3	S.W.	2	2				1	1	
5	E. Central	2	2					1	1
6	Central	9	5	4		1		2	2
7	W. Central	1	0	1					
8	N.E.	1	1		1				
9	N.W.	3	1	2	1				
Total	--	20	13	7	2	1	2	5	3

*Samples obtained through Pioneer Grain Ltd.
**Usually 200 seeds per sample plated on 20% V8 agar after surface disinfection for 10 mins. in 10% Javex (0.6% NaOCl).
***Trace - see text for explanation.

Table 2. Incidence of Ascochyta in lentil seed samples tested at Saskatoon in late winter 1979*.

No. of Supplier of seed	Geographic origin of seed	No. of samples	No. with ascochyta infection	No. of samples in different % infection categories				
				0%	0.5%	0.75–2.5%	2.75–5.75%	〉6%
1	? Sask.	1	1				1	
2	? Sask.	1	1					1
3	?	1	1			1		
4	? Manitoba	2	1	1		1		
5	W. Central Sask.	2	0	2				
6	S. Central Sask.	4	3	1		1	1	1
7	Central Sask.	5	5				1	4
8	? W. Central Sask.	1	0	1				
9	Central Sask.	1	1					1
10	? Manitoba	1	1		1			
Total	- -	19	14	5	1	3	3	7

*100-400 seeds per sample plated on 20% V8 agar after surface disinfection for 10 mins. in 10% Javex (0.6% NaOCl).

Table 3. Incidence of Ascochyta in seed samples from the 1978 lentil crop tested in Ottawa.

Origin	No. samples tested	No. samples with *Ascochyta spp.*	No. of samples in different % infection categories				
			0%	0.5%	0.75-2.5%	2.75-5.75%	〉6%
Manitoba	43	18	25	7	4	4	3
Sask.	20	15	5	1	4	2	8
Alberta	1	1			1		
B.C.	6	4	2		1		3
Unknown	2	1	1				1
Total	72	39	33	8	10	6	15

Ottawa

Surveys of seed samples from the 1978 and 1979 crops were also conducted at Ottawa. The samples were obtained from Customs offices, from seed companies and from growers in the four western provinces. However, it is probable that samples from British Columbia were grown elsewhere. More samples from the 1979 crop were obtained than from the 1978 crop, probably because news of the disease had spread among pulse crop growers through newsletters and other media. Producers were invited to submit seed to Ottawa for agar plate testing. Thus, the 1979 crop samples may have been more representative than the 1978 crop samples of all lentil growing areas in western Canada.

Four hundred seeds were drawn from each sample received in Ottawa and surface-disinfected by soaking for 10 min. in NaOCl adjusted to 2% available chlorine. After disinfection the seeds were plated on V8, 10 seeds to a plate, and incubated for 7 days at 22°C with a 12 hr. photo-period and near ultra violet light. After counting colonies of *Ascochyta* on the plates, percentage infection figures were derived.

It is probable that some of the 1978 crop samples overlapped those tested at Saskatoon, but in any case the results (Table 3) confirmed the widespread distribution of the pathogen. Nearly 55% of the samples were infected and 21% showed an infection level above 6%. Most of the heavily infected samples came from Saskatchewan. Samples from the 1979 crop were generally not as heavily infected as those from the 1978 crop (Table 4). About 38% of the 1979 samples were infected and 11% carried more than 6% ascochyta. However, the most severely infected samples came proportionately equally from Manitoba and Saskatchewan.

Field survey, 1979

To obtain further information on the geographic range of the pathogen as a sequal to the initial seed testing, a survey of lentil fields was done in 1979 in Saskatchewan. Emphasis was placed on the major lentil growing districts. Most fields were visited twice, once in mid to late June to check for seedling infection of possible seed-borne origin, and once in early August to check the intensity of disease shortly before harvest. Each field was usually inspected in only one or two places, and mainly qualitative observations of the intensity of ascochyta blight were made. When the symptoms were questionable, plant samples were collected and isolations made to confirm the presence of the pathogen. Where possible, information was obtained from the grower on source of seed, agronomic practices and crop history of the field.

There was an extremely low level of ascochyta blight in most areas of Saskatchewan (Table 5). The disease was found in only 23% of 61 fields visited during the summer, and only 11% of the fields showed more than trace of disease by early August. Generally fields in the central and northeastern crop districts were more heavily infested. The low levels of disease made detailed comparisons with seed source, etc., valueless, but it is noteworthy that ascochyta blight occurred in all three of the fields which had been planted on lentil stubble.

Table 4. Incidence of Ascochyta in seed samples from the 1979 lentil crop tested in Ottawa.

Origin	No. samples tested	No. samples with *Ascochyta spp.*	No. of samples in different % infection categories				
			0%	0.5%	0.75-2.5%	2.75-5.75%	〉6%
Manitoba	33	11	22	4	2	1	4
Sask.	53	23	30	6	7	3	7
B.C.	11	4	7	4			
Total	97	38	59	14	9	4	11

Table 5. Incidence of ascochyta blight in commercial lentil fields in 1979 in Saskatchewan.

Saskatchewan Crop District No.	Geographic location in arable part of Saskatchewan	Total No. of fields inspected	First inspection*		Second inspection		No. fields with more than trace+ of ascochyta
			Total No. of fields	No. fields with ascochyta	Total No. of fields	No. fields with ascochyta	
2	S. Central	29	29	3	27	5	0
3	S.W.	7	7	0	7	0	0
5	E. Central	3	3	0	2	0	0
6	Central	10	2	0	8	5	5
7	W. Central	9	9	1	9	0	0
8	N.E.	3	3	3	1	1	1
Total	- -	61	53	7	54	11	6

*mid-late June
**early August
+Trace-for explanation see text.

Discussion

Ascochyta blight is now established as the major disease of lentils in western Canada. Probably it affects seed quality and marketability more than yield. A report from Argentina (2) refers to the lowered commercial value of infected lentil seeds and many Saskatchewan growers experienced similar losses in 1978. Under Saskatchewan management practices much of the seed discoloration associated with ascochyta infection may be due more to saprophytic growth of the fungus on ripening pods after the crop has been swathed (cut and placed in windrows) than to parasitic infection of immature pods and seeds. Lentil plants are short and are usually cut close to the ground. The swath lies almost flat on the ground rather than on top of a stubble which would assist ventilation. Hence, rain or heavy dew after cutting may cause prolonged wetness of lentil swaths and provide excellent conditions for the saprophytic development of *Ascochyta*. Even when the disease is scarce in a standing crop, the opportunity for severe seed discoloration to develop at harvest time may still exist. Studies of the relative importance of yield and quality losses due to ascochyta blight and of the role of harvest date and weather conditions on seed discoloration are in progress in the senior author's laboratory.

Various ascochyta diseases of legumes are favored by moist weather and ascochyta blight of lentil appears to be no exception (2, 15). The lower incidence of seed infection in 1979 crop samples probably reflects the generally drier harvest conditions in 1979. In August and September 1978 rains in most parts of Saskatchewan resulted in many lentil crops lying in the swath for about four weeks. Similarly, in the field survey in 1979 the low levels of disease in the south and west-central parts of Saskatchewan undoubtedly partly reflected the dry conditions prevailing there for most of the growing season. More disease was present north of Saskatoon where more rainfall occurred. Observations in a smaller number of fields in 1980 (R.A.A. Morrall and B.D. Gossen, unpublished) were similar. However, in the more northern regions the role of infected stubble from previous diseased crops must be considered.

Disease control

Recommendations for disease control to western Canadian lentil growers are at present based only on general pathological principles. Crop rotation, early seeding to escape moister weather at harvest, the use of disease-free seed and obtaining agar plate tests of prospective seed lentils are advised. To date there are no data on ascochyta blight to suggest that pulse crop growers should not include other legumes in a rotation with lentils; *A. lentis* appears to be host-specific and will probably not be transmitted by native legumes, weeds, forage crops or other pulses.

Since ascochyta blight is seed-borne it is possible that seed treatments will help to control the disease. In Argentina the use of several fungicides, including captan and thiram, is recommended (2). Tests in Saskatchewan in 1979 and 1980 (12, 13) with 10 compounds have given equivocal results. However, seedling emergence figures were complicated by factors such as seed of very low germinability (13) and the presence of soil-borne seedling blight organisms (12). Disease control by seed treatment may be feasible only in established lentil growing areas because of the potential interference of fungicides with *Rhizobium* inoculant applied to the lentil seed (13). Moreover, because of the ability of lentil plants to compensate for reduced plant stands by branching, significant increases in seedling emergence from seed treatment will not always be translated into significant yield increases.

In Argentina 509 lentil lines were assessed for disease reaction to ascochyta blight and 115 were assigned to the lowest category, in which only a few spots developed on the leaves (11). However, in India Khatri and Singh (7) tested 947 lines and found that only five had a high level of resistance. Results from Saskatchewan in 1979 and 1980 (R.A.A. Morrall, unpublished) showed that there are significant differences in disease reaction between breeding lines of lentils and that cv. Laird is more resistant than cv. Eston or commercial lentils. Thus, developing greater cultivar resistance should be another objective for disease control in the future.

Acknowledgements

The authors are grateful to the following people for advice and technical assistance: Dr. A. Slinkard, Messrs. B.D. Gossen, D. Kaminski, E. Peters, J. Spruyt, Mrs. S. Reid and Miss N.M. Needham. The work was supported by grants from the Saskatchewan Agricultural Research Foundation and the National Research Council of Canada.

Literature cited

1. Ainsworth, G.C. 1961. Ainsworth and Bisby's dictionary of the fungi. 5th ed. Commonwealth Mycological Institute. 547 pp.
2. Anon. 1979. Lentils Diseases. Proc. 1st Internat. Lentil Res. Semin. Aleppo, Syria, May 1979. 13-16.
3. Boerema, G.H. and M.M.J. Dorenbosch. 1973. The Phoma and Ascochyta species described by Wollenweber and Hochapfel in their study on fruit rotting. Stud. Mycol. 3: 18-19 and 38-39.
4. Bondartzeva-Monteverde, V.N. and N.I. Vassilievsky. 1940. A contribution to the biology and morphology of some species of *Ascochyta* on Leguminosae. Acta. Instit. Bot. Acad. Sci. U.R.S.S. 1938, Ser. II (Pl. Crypt.) 345-376. (In. Rev. Appl. Mycol. 20: 232-234, 1941).
5. Jones, L.K. 1927. Studies of the nature and control of blight, leaf and pod spot, and footrot caused by species of *Ascochyta*. N. Y. Agric. Exp. Stat. Bull. 547: 46 pp.
6. Khare, M.N., S.C. Agrawal and A.C. Jain. 1979. Diseases of lentil and their control. Tech. Bull. Jawaharlal Nehru Univ. 29 pp.
7. Khatri, H. L. and K. Singh. 1975. Lentil blight - a new record for India. Labdev. J. Sci. Tech. 13-B: 73-74.
8. McKenzie, D.L. and R.A.A. Morrall. 1973. Diseases of three specialty legume crops in Saskatchewan in 1972: field pea, lentil, and fababean. Can. Plant Dis. Survey, 53:187-190.
9. McKenzie, D.L. and R.A.A. Morrall. 1975. Diseases of specialty crops in Saskatchewan. II. Notes on field pea in 1973-74 and on lentil in 1973. Can. Plant. Dis. Survey 55:97-100.
10. Mel'nik, V.A. 1977. Opredelitel' gribov roda *Ascochyta* Lib. Leningrad, U.S.S.R. Izdatel'stvo Nauka. 246 pp.
11. Mitidieri, I.Z.M. DE. 1975. La mancha de la lenteja (*Lens culinaris*) Medik causada pro *Ascochyta lentis* Bond. y. Vassil., en la Argentina. Revta Invest. agric., B. Aires. 11:43-55.
12. Morrall, R.A.A. 1980. Evaluation of seed treatments to improve emergence of *Ascochyta* - infected lentil seed. Pesticide Res. Rept., Agric. Canada, Ottawa. 224.
13. Morrall, R.A.A. and B.D. Gossen. 1979. Evaluation of seed treatments for the control of seed-borne ascochyta blight of lentils. Pesticide Res. Rept., Agric. Canada, Ottawa. 406-407.
14. Morrall, R.A.A., D.L. McKenzie, L.J. Duczek and P.R. Verma. 1972. A qualitative survey of diseases of some specialty crops in Saskatchewan in 1970 and 1971; sunflower, safflower, buckwheat, lentil, mustards and field pea. Can. Plant. Dis. Survey. 52: 143-148.
15. Morrall, R.A.A., J.W. Sheppard, and J. Bissett. 1979. Distribution of seed-borne ascochyta on lentils in western Canada in 1978-79. Proc. Can. Phytopath. Soc. 46: 65-66 (Abstr.).
16. Sprague, R. 1935. *Ascochyta boltshauseri* on beans in Oregon. Phytopathology 25: 416-419.
17. Tuite, J. 1969. Plant pathological methods. Fungi and bacteria. Burgess Publ. Co. 239 pp.

Infection of additional hosts of Synchytrium endobioticum, the causal agent of potato wart disease: 3. Tomato as an assay tool in potato wart disease[1]

Michael C. Hampson[2]

Tomato was investigated as an assay tool for *Synchytrium endobioticum* because of a need for a simple, rapid, consistent and dependable assay. To investigate tomato, tomato seeds or seedlings were grown in *Synchytrium endobioticum*-infested soil or potting mix, and signs of infection were indicated by the presence of sporangia in tomato root tissue. Infection was detected 3 d after inoculation, and increased in an exponential fashion with time. The ED_{50} for tomato was ca. 10 sporangia g^{-1}, but infection occurred at propagule densities as low as one sporangium g^{-1}. Infection occurred in seedlings grown in sand to which sporangia were moved by irrigation; downward migration of sporangia was $>$ 10 cm. Tomato infection paralleled potato infection when tomato was grown in soils obtained from field amendment trials. Tomato appears to serve as a useful assay tool for *S. endobioticum*.

Can. Plant Dis. Surv. 61:1, 15-18, 1981.

On a cultivé des graines ou des plants de tomate dans un mélange d'empotage infesté de *Synchytrium endobioticum* et les signes d'infection se sont manifestés par la présence de sporanges dans le tissu radiculaire. L'infection a été décelée 3 jours après l'inoculation et s'est accrue de façon exponentielle avec le temps. La DE_{50} de la tomate a été de 10 sporanges/g de mélange, mais l'infection est apparue à des densités de propagules aussi faibles que 1 sporange/g. L'infection s'est manifesté chez les plants cultivés dans le sable où l'irrigation a favorisé la migration verticale des sporanges; cette migration vers le bas a dépassé 10 cm. L'infection de la tomate a égalé celle de la pomme de terre dans les cultures de tomate en sols provenant d'essais d'amendement en plein champ. Il semblerait que la tomate puisse servir d'instrument d'essai biologique pour *S. endobioticum*.

Introduction

There are several mechanical methods described in the literature (1, 4, 7, 8, 10) for detecting *Synchytrium endobioticum* (Schilb.) Perc. in soils. For example, Pratt (10) sieved infested soil through wet-sieves, and retrieved sporangia from the finest mesh sieve; Nelson and Olsen (8) centrifuged infested soil with dibromoethane, or mineral oil plus SAE 10 motor oil, and Glynne (1) used chloroform to float sporangia off; Mygind (7) developed a water flotation technique to retrieve sporangia. These techniques provide answers to quantitative questions, but they have definite floors of propagule extractability, hence detectability (4). The biological method using potato is so fraught with inconsistency and unreliability (2, 6) that attention was paid to trials with tomato (*Lycopersicum esculentum* Mill.). Tomato is an additional host of *S. endobioticum* and has been shown to be readily susceptible to infection by this fungus (3). Furthermore, the detection of *S. endobioticum* by tomato involves simple greenhouse culture equipment and standard greenhouse techniques.

This paper is an account of infection experiments with tomato and *S. endobioticum* to determine the first signs of infection and the ED_{50}, and to test tomato as an assay tool.

Materials and methods

Tomato cv. Beefmaster (J. Harris Co. Inc.), an extra large fruited hybrid, was selected for experimental work. It was the most susceptible of all cultivars to *S. endobioticum* that we have tested (3).

Both seedlings and seeds were used for infection. Seedlings, at the four leaf stage, were transplanted into units of sporangia-infested soil or potting mix (perlite:peat moss 1:1, v/v), or slurried in sporangia-suspension prior to transplanting. Seeds were planted directly onto infested mix or sand, covered lightly with mix, and germinated under a polyethylene cover. All seedlings were watered by irrigation to field capacity daily, and fertilized weekly with Van den Elft's tomato nutrient solution. Seedlings were raised in a controlled-environment room at 20°C, 80% RH, and 14-h day.

To infest sand or potting mix, sporangia were separated from tumor tissue by homogenization and screening. The separated sporangia were suspended in water, and the densities adjusted to give, when mixed with measured amounts of potting mix or sand, 1, 2, 5, 10, 15, 20, 30, 40 and 50 sporangia g^{-1} potting medium. After mixing by hand to ensure uniform propagule distribution, aliquots of infested media were distributed among "6-inch" plant pots. Five

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Accepted for publication October 31, 1980

seedlings were planted in each pot. To make sporangial slurries, the separated sporangia were suspended in water to give 95,000 sporangia/ml. To assay sporangial movement in soil, 10,000 separated sporangia were deposited on each sand column and then flushed into the sand with water.

Tomato seedlings were harvested at different intervals, depending on the experiment involved, and root areas were cleansed by brushing with a soft brush under running water.

Table 1. Numbers of sporangia of *S. endobioticum* located in root tissue of tomato at different intervals after inoculation*.

Days after inoculation	Numbers of Sporangia range	$\bar{x}$**	Standard Deviation**
0	0	0	0
3	0-3	⟨1	1
7	0-6	2	2
9	0-13	4	4
13	0-13	5	4
16	1-21	7	7
20	2-15	7	5
23	3-42	16	12
28	1-65	27	20
30	5-105	41	31
34	12-58	37	17
37	5-227	82	72
41	29-292	124	85
43	42-252	136	79

*Ten samples/treatment, inoculated by slurrying roots in 95,000 sporangia/ml.
**Rounded out to nearest whole number.

Potentially adhering sporangia were removed by ultrasonication, the seedlings were examined microscopically (25 ×) (3) and the numbers of sporangia/plant counted.

To assay the downward movement of sporangia, triangular tubes (40 cm L × 3.7 cm W) were constructed from milk cartons and filled with gravel. Another similar tube (20 cm^2) was mounted securely on each gravel tube, and these were $^3/_4$ filled with sieved sand. Resting sporangia were deposited on each sand surface and water was added to the empty $^1/_4$ tube sections in two experiments. The tubes were grouped into six units of four tubes and five units of ten tubes. In the first experiment, the units received 50, 100, 150, 200, 250 or 300 ml water in daily increments of 50 ml; in the second, 5, 10, 20, 40 or 80 ml water in daily increments of 5 ml. The water regimes were based on an ideal of 25 mm water/wk for good potato growth (9). Each sand-filled tube was sectioned into four parts. The sand filled sections, labelled A, B, and C, were embedded separately in potting mix in 10-cm plastic plant pots. Each of the two hundred and forty sections were seeded with at least three Beefmaster seeds. These were culled later to one per section in the first experiment only, and harvested 8 wks after seeding.

To assay the influence of soil amendments on tomato infection, soil samples from amendment plots were planted to tomato transplants. In the field design, microplots each containing nine tubers, set 3 × 3, were amended with either lime, oat/barley straw, dried chopped potato tops, or Ca-, K-, Na- and NH_4- nitrates, singly or in combination. Lime was added to soil at the rate of 1120 kg/ha, straw and potato haulm at the rate of 30t/ha, and the nitrates at the rate of 168 kg N/ha. Some microplots received no treatment. At harvest, an aliquot of soil was removed at the base of each plant, and aliquots from the same microplots

Table 2. Average number of resting sporangia/tomato seedling 5 wks after inoculation at different levels of sporangial density.

	No. resting sporangia in inoculum (g^{-1} potting mix)											
	1*	2*	5*	5#	10†	10*	10#	15#	20†	30†	40†	50†
No. Seedlings	20	20	20	19	20	20	20	20	20	20	20	20
Median No. Sporangia/ Seedling	0.55	0.6	0.6	26	26	1.2	40	53	27.9	41.8	62.7	71.1
Sporangial Range	0-3	0-6	0-3	7-133	6-99	0-3	11-109	10-140	12-127	9-100	16-151	41-123
Sporangial Age (Wk)	20	20	20	1	8	20	1	1	8	8	8	8
% Infection	30	35	30	100	100	45	100	100	100	100	100	100
Ave. % Infection				65		82						

† First experiment
* Second experiment
# Third experiment

pooled. Numbers of sporangia g^{-1} field soil were estimated by the method of Hampson and Thompson (4). Weights of tumor/treatment were recorded.

Results

Time course of infection

Seedlings were inoculated in slurries and planted. Ten seedlings at a time were removed at 2-5 d intervals after inoculation. From Table 1, it can be seen that sporangia were first evident in tomato tissue by 3 d after inoculation. There were wide ranges of sporangial numbers in the samples at each level of examination. Generally, the average number of sporangia/seedling increased in an exponential fashion; at 1, 3 and 5 wk after inoculation the average sporangial numbers were, respectively, 2, 7 and 137/seedling.

ED_{50}

The number of sporangia to bring about 50% infection of tomato seedlings was determined by transplanting 4-wk old seedlings into potting mix previously infested to different levels of sporangial density. In the first experiment, twenty seedlings at each level of 10, 20, 30, 40 or 50 sporangia/g mix displayed 100% infection; in the second experiment, twenty seedlings each at sporangial densities of 1, 2, 5 or 10 sporangia/g mix displayed 30, 35, 40 and 45% infection, respectively; in the third experiment, forty seedlings each at the 5, 10 and 15 sporangia/g level displayed 100% infection, respectively (Table 2).

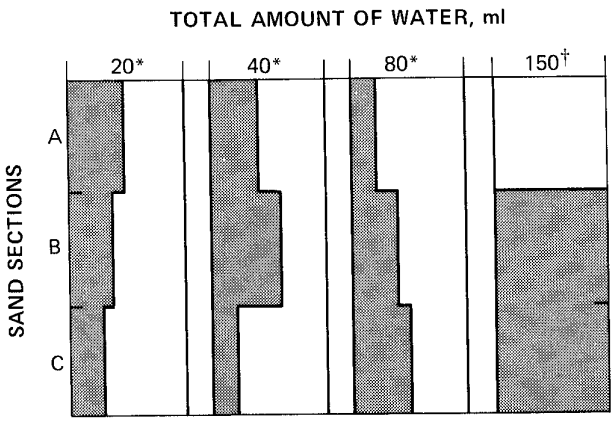

Figure 1. Histogram depicting the depth in sand (particle size, 600-1000 μm) to which sporangia were washed when irrigated. The presence of sporangia was shown by growing tomato seeds or seedlings in a 5-cm top layer of each section. Bar width is proportional to incidence of infection, distance between marks on horizontal axis = 100% infection. A, B and C are top, middle and bottom sand sections, respectively. Irrigation water was applied in multiples of $5^{(*)}$ or $50^{(+)}$ - ml units.

Downward migration of sporangia

Figure 1 depicts, in histogram form, the percent infection recorded for tomato at the three levels of sand in the percolation tubes which received 20, 40, 80 and 150 ml water. It may be assumed that the movement (and dispersion) of the propagules within the sand columns is quite complex. The presence of sporangia, however, is shown by tomato. The sporangia were well-moved down the columns. At the 5 and 300-ml levels, respectively, tomato infection in section C was 17 and 100%.

Soil amendment influence on tomato infection

In order to see whether tomato infection was also influenced by soil treatment, tomato seedlings were grown in the soils which had been amended. Fig. 2 shows the soil treatments grouped (clockwise) in increasing order of disease incidence (percent infection) on potato, viz: I, II, III and IV, which order parallels the infection index and total tomato borne sporangial numbers. The total soil-borne sporangial numbers, however, were I > III > IV > II.

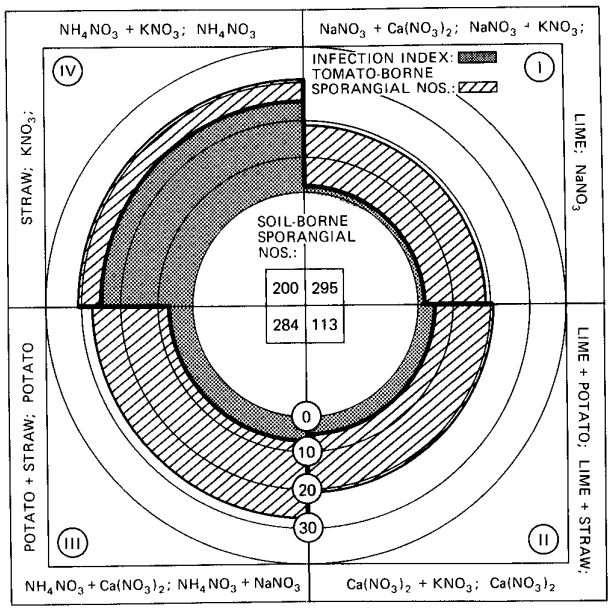

Figure 2. Soil treatments were ranked clockwise in ascending order of disease incidence, and then divided into four groups (I, II, III and IV). The chart shows the total number of soil-borne sporangia (S=85), tomato-borne sporangia (S=6) and infection index value for each quadrant. (Infection Index = weight of tumor mass ÷ number of soil-borne sporangia).

Discussion

The potato has been used as an assay tool by many workers in potato wart disease as the principal host of *S. endobioticum*. It displays, however, erratic and unpredictable infection in field (6), greenhouse and growth room culture (2). Other disadvantages to using potato as an assay plant are that tubers are neither equally available nor in similar physiological condition throughout the year, numbers of potato plants sufficient to be treated statistically consume considerable greenhouse space, under laboratory conditions the potato plant does not appear to be susceptible to low inoculum levels, and laboratory conditions for potato infection have not been clearly defined.

The experiments detailed in this paper demonstrated that tomato (cv. Beefmaster) displayed infection, with *S. endobioticum*, within a few days of inoculation, and 100% infection at 14 d after inoculation, that one sporangium g^{-1} inoculation media was detectable, and that 50% tomato seedlings became infected at inoculum densities of ca. 5 sporangia g^{-1}. It was also observed that disease incidence in tomato increased rapidly with time, that numbers of host-located sporangia varied considerably for all tomato samples at any one inoculum level, and that tomato infection appeared to be strongly influenced by the nature of the inoculation medium in much the same way as potato infection responded.

Nevertheless, since tomato appears extremely susceptible to *S. endobioticum*, the fact that seed is readily available, inexpensive, germinates readily, can be used at all times during the year, that seedlings are readily cultivated and transplanted, and that sufficient numbers of tomato plants can be handled to provide statistical treatment of results, tomato gives us a sensitive tool for exploring facets of wart disease that would otherwise be denied us using potato alone.

Acknowledgements

The author thanks Mrs. Janet W. Coombes for assistance in inoculation and sporangial counting.

Literature cited

1. Glynne, Mary D. 1926. The viability of the winter sporangium of *Synchytrium endobioticum* (Schilb.) Perc., the organism causing wart disease in potato. Ann. Appl. Biol. 13:19-36.
2. Hampson, M.C. 1977. A hypothesis to explain erratic and unpredictable infection in potato wart disease. FAO Plant Prot. Bull. 25(2):68-72.
3. Hampson, M.C. 1979. Infection of additional hosts of *Synchytrium endobioticum*, the causal agent of potato wart disease: 2. Tomato, tobacco and species of *Capsicastrum, Datura, Physalis* and *Schizanthus*. Can. Plant Dis. Surv. 59(1):3-6.
4. Hampson, M.C. and P.R. Thompson. 1977. A quantitative method to examine large numbers of soil samples for *Synchytrium endobioticum*, the cause of potato wart disease. Plant and Soil 46(3):659-664.
5. Hampson, M.C. and N.F. Haard. 1980. Pathogenesis of *Synchytrium endobioticum*. 1. Infection responses in potato and tomato. Can. J. Plant Path. (in press).
6. Hunt, N.R., F.G. O'Donnell, and R.P. Marshall. 1925. Steam and chemical soil disinfection with special reference to potato wart. J. Agr. Res. 31:301-363.
7. Mygind, H. 1961-62. Examination of soil samples for potato wart sporangia. II. Acta Agr. Scand. 11-12, Supplement 10:114-120.
8. Nelson, G.A. and O.A. Olsen. 1964. Methods for estimating numbers of resting sporangia of *Synchytrium endobioticum*. Phytopathology 54:185-186.
9. Parent, R.C., W.N. Black and L.C. Callbeck. 1967. Potato growing in the Atlantic Provinces. Canada Dept. Agric. Pub. 1281. 28 p.
10. Pratt, Margaret A. 1976. A wet-sieving and flotation technique for the detection of resting sporangia of *Synchytrium endobioticum* in soil. Ann. Appl. Biol. 82:21-29.

Incidence of ergot in populations of Ammophila breviligulata[1]

Irene S. Krajnyk and M.A. Maun[2]

This is the first report of the occurrence of ergot (*Claviceps purpurea*) in natural populations of *Ammophila breviligulata* in Ontario. During 1976 the percentage of infected panicles ranged from 6 to 40 at Rondeau Provincial Park (Lake Erie) and 9 to 12 at Pinery Provincial Park (Lake Huron) but the infection decreased significantly during 1977 probably due to low rainfall. Each panicle contained 1 to 4 sclerotia which were located mainly in the lower spikelets of the panicle. The weight, length and diameter of sclerotia ranged from 15.4 to 28 mg, 7.6 to 10.6 mm, and 2.0 to 2.8 mm, respectively.

Can. Plant Dis. Surv. 61:1, 19-21, 1981.

Il s'agit de la première mention de la présence de l'ergot (*Claviceps purpurea*) chez des populations naturelles de *Ammophila breviligulata* en Ontario. En 1976, le taux de panicules infectées variait de 6 à 40% et de 9 à 12% dans les parcs provinciaux Rondeau (lac Érié) et Pinery (lac Huron) respectivement, mais l'infection a régressé significativement en 1977, peut-être à cause du peu de pluie qu'ont reçue ces régions. Chaque panicule examinée comptait de 1 à 4 sclérotes principalement situés sur les épillets inférieurs. Le poids, la longueur et le diamètre des sclérotes variaient de 15,4 à 28 mg, 7,6 à 10,6 mm et 2 à 2,8 mm respectivement.

Introduction

Ammophila breviligulata Fern. is one of the two most common perennial sand binding grasses along the Great Lakes in North America (Olson, 1958). It reproduces both sexually and asexually (Krajnyk and Maun, 1980) but the establishment of plants along the shoreline occurs mainly by fragments of vegetative shoots (Olson, 1958).

The sexual reproduction in *A. breviligulata* is limited only to certain specific habitats such as terraces with sand deposition (Eldred, 1980). Even in flowering habitats, density of flowering culms is low (1 to 23 per m^2) and the percentage of spikelets containing caryopses is only 28 to 48 (Krajnyk, 1979). The poor seed set may be due to infertile pollen (Kubien, 1970), insect damage (Krajnyk, 1979), meiotic abnormalities (Church, 1929), misshapen embryos or endosperm deficiency (Laing, 1958) and soil infertility.

In our studies at Rondeau (Lake Erie) and Pinery (Lake Huron) Provincial Parks, we observed incidence of ergot (causal agent = *Claviceps purpurea* (Fr.) Tul.) in several populations of *A. breviligulata*. Ergot is a major disease of cultivated or naturally occurring cross-pollinated grasses in North America (Hardison, 1976). The open condition of the floret at pollination makes them vulnerable to floral diseases. The development of cross-pollinated crop; *Triticale* species (Briggle, 1969) and male sterile strains of normally self-pollinated crops, wheat (*Triticum aestivum*) and barley (*Hordeum vulgare*), has increased their susceptibility to ergot (Cunfer *et al.*, 1974). Puranik and Mathre (1971) observed that in 1970, 76% of the heads and 36% of the florets of male-sterile barley were infected under natural conditions in Montana. The major source of infection is the carry over of sclerotia in the soil but natural populations of susceptible grasses might also serve as nuclei of infection.

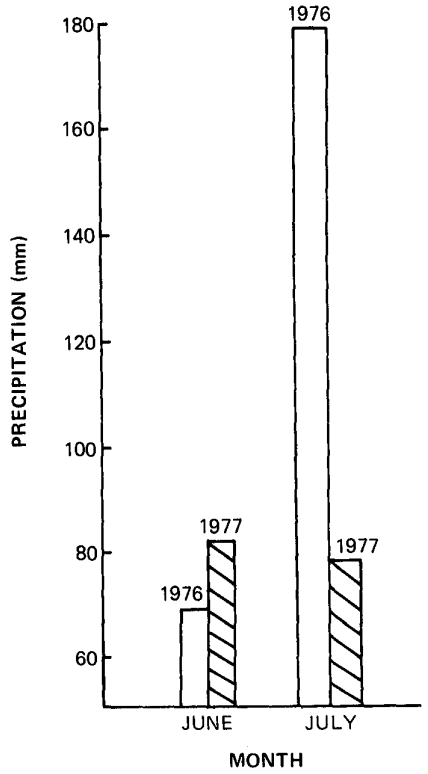

Fig. 1. Monthly mean precipitation during anthesis of *A. breviligulata* in 1976 and 1977 at Rondeau Provincial Park (weather station located 32 km from the park).

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Accepted for publication December 1, 1980.

The purpose of this paper is to report quantitative data on the extent of occurrence of ergot in panicles of *A. breviligulata* along Lake Erie at Rondeau Provincial Park and Lake Huron at Pinery Provincial Park.

Materials and methods

Panicles were harvested at random from 7 populations of *A. breviligulata* at Rondeau Provincial Park and 3 populations at Pinery Provincial Park during 1976 and 1977. The panicles containing ergot sclerotia (horny structures projecting out of spikelets) were separated from the rest of the panicles and the proportion of infected panicles calculated. The location of each sclerotium on a panicle (top, middle or bottom) was recorded. The sclerotium was then carefully removed and its length, diameter and weight were measured.

Table 1. Frequency of infestation of panicles by ergot and number of sclerotia per panicle (± standard error) of *A. breviligulata* at Rondeau and Pinery Provincial Parks.

Name of Park	Population No.	Panicles infected (%) 1976	1977	No. of sclerotia per panicle 1976	1977
Rondeau	1	5.8*	1.1	2.5 ± 0.3	1.4 ± 0.3
	2	11.7*	0.5	3.5 ± 0.6	1.2 ± 0.2
	3	32.2*	4.4	2.5 ± 0.3	1.7 ± 0.2
	4	31.2*	2.2	2.8 ± 0.2	1.4 ± 0.2
	5	40.4*	4.9	2.7 ± 0.2	1.3 ± 0.2
	6	-	4.2	-	1.4 ± 0.2
	7	-	1.8	-	1.3 ± 0.1
Pinery	1	11.7*	0	1.6 ± 0.3	0
	2	9.2*	0	1.8 ± 0.4	0
	3	1.6*	0	-	0

*Significantly different (P = .05) from values in 1977 according to the "t" test.

Results and discussion

Panicles of *A. breviligulata* are susceptible to infection by ergot - a disease specific to species and varieties of the family Poaceae (Brentzel, 1947). Sclerotia were found at the time of panicle maturation (end of July) along Lake Erie and Huron shorelines. The percentage of panicles that contained one or more ergot sclerotia in Rondeau Provincial Park populations during 1976, ranged from 5.8 to 40.4% (Table 1). In contrast only 9 to 12% of the panicles in Pinery Provincial Park populations were infected with ergot. During 1977, however, the disease incidence decreased to only 0.5% to 5.0% of the panicles at Rondeau and 0% at Pinery Provincial Park mainly because of significantly higher precipitation during June and July 1976 (Fig. 1). According to Weniger (1924) ergot epidemics develop in wet seasons.

The number of sclerotia per panicle was also variable during 1976 and 1977. At Rondeau each panicle contained 2.5 to 3.5 sclerotia per panicle during 1976 and 1.2 to 1.7 during 1977 (Table 1). The Pinery populations contained 1.6 to 1.8 sclerotia per panicle in 1976 and none during 1977.

The average weight per sclerotium in Rondeau and Pinery populations during 1976 ranged from 15.4 to 25.8 mg, the length from 8.8 mm to 10.5 mm and the diameter from 2.1 mm to 2.4 mm (Table 2). In 1977, the weight per sclerotium for Rondeau populations ranged from 15.8 to 28 mg, and the length and diameter ranged from 7.6 to 10.6 mm and 2.0 to 2.8 mm, respectively. Such resting sclerotia would provide a source of infection for future generations not only for populations of *A. breviligulata* but also for other susceptible grasses or crops in that region.

The sclerotia on a pancicle were most abundant in the middle and bottom of a panicle. Infected panicles collected from Rondeau populations in 1976 and 1977 showed that only 17% of the sclerotia were found in the top one third of the panicle, 42% in the middle one third and 41% in the bottom one third of a panicle. Examination of panicles from the two populations at Pinery Provincial Park showed that

Table 2. The average weight, length and diameter per sclerotium (± standard error) collected from populations of *A. breviligulata* at Rondeau and Pinery Provincial Parks.

Location	Population No.	Average Size Per Sclerotium: Weight (mg) 1976	1977	Length (mm) 1976	1977	Diameter (mm) 1976	1977
Rondeau	1	15.4 ± 0.8	25.4 ± 7.9	8.8 ± 0.2	10.6 ± 1.5	2.1 ± 0.04	2.4 ± 0.1
	2	16.0 ± 0.9	15.8 ± 9.0	9.1 ± 0.2	7.6 ± 1.0	2.1 ± 0.04	2.0 ± 0.4
	3	25.8 ± 1.2	20.5 ± 2.1	10.4 ± 0.2	10.0 ± 0.5	2.4 ± 0.04	2.2 ± 0.1
	4	20.8 ± 0.8	20.9 ± 6.6	10.4 ± 0.2	10.5 ± 1.1	2.2 ± 0.03	2.1 ± 0.2
	5	23.4 ± 1.0	20.6 ± 2.5	10.4 ± 0.2	10.6 ± 0.7	2.3 ± 0.03	2.3 ± 0.1
	6	-	16.7 ± 2.0	-	9.1 ± 0.5	-	2.1 ± 0.1
	7	-	28.0 ± 3.2	-	10.0 ± 0.7	-	2.8 ± 0.1
Pinery	1	22.0 ± 2.9	-	10.5 ± 0.8	-	2.4 ± 0.1	-
	2	19.6 ± 2.8	-	9.1 ± 0.5	-	2.2 ± 0.1	-
	3	-	-	-	-	-	-

33% of the sclerotia were at the top one third of a panicle, 53% in the middle one third and 14% in the bottom one third (For detailed data please refer to Krajnyk, 1979).

In conclusion it may be stated that shorelines of lakes and oceans provide suitable conditions for the growth and spread of this disease because the germination of sclerotia, ascospores and conidia and the growth of hyphae is the highest at high relative humidity (Brentzel, 1947) and high soil moisture conditions (McCrea, 1931). The ergot sclerotia can replace caryopses in about 3.5 spikelets on a panicle thus contributing to a reduction in the number of fertile spikelets per panicle.

Acknowledgements

The authors express their appreciation to Dr. W.E. McKeen and Dr. D. Fahselt for a critical review of the manuscript. Support for this research came from Natural Sciences and Engineering Research Council of Canada. We thank the Ministry of Natural Resources for granting permission to work at Pinery and Rondeau Provincial Parks.

Literature cited

1. Brentzel, W.E. 1947. Studies on ergot of grains and grasses. North Dakota Exp. Sta. Bull. 348:2-20.
2. Briggle, L.W. 1969. *Triticale* - a review. Crop Sci. 9:197-202.
3. Church, G.L. 1929. Meiotic phenomena in certain Gramineae. I. Festuceae, Aveneae, Agrostideae, Chlorideae and Phalarideae. Bot. Gaz. 87:608-629.
4. Cunfer, B., D.E. Mathre, and E.A. Hockett. 1974. Diversity of reaction to ergot among male-sterile barleys. Plant Dis. Rep. 58:679-682.
5. Eldred, R. 1980. Effects of environmental factors on the flowering of *Ammophila breviligulata* Fernald. M.Sc. Thesis, University of Western Ontario, London, Canada, 114 p.
6. Hardison, J.R. 1976. Fire and flame for plant disease control. Ann. Rev. Phytopath. 14:355-379.
7. Krajnyk, I.S., and M.A. Maun. 1980. Vegetative reproduction in the juvenile phase of *Ammophila breviligulata* Fern. Can. J. Bot. (in press).
8. Krajnyk, I.S. 1979. Reproductive biology of *Ammophila breviligulata*, Fernald. M.Sc. Thesis, University of Western Ontario, London, Ontario, Canada. 164 p.
9. Kubein, E. 1970. Embryological studies in *Ammophila arenaria* (L.) Link. Acta Biol. Cracov. Ser. Bot. 13:1-10.
10. Laing, C.C. 1958. Studies in the ecology of *Ammophila breviligulata*. I. Seedling survival and its relationship to population increase and dispersal. Bot. Gaz. 119:208-216.
11. McCrea, A. 1931. The reactions of *Claviceps purpurea* to variations of environment. Am. J. Bot. 18:50-78.
12. Olson, J.A. 1958. Lake Michigan dune development. 2. Plants as agents and tools in geomorphology. J. Geol. 66:345-351.
13. Puranik, S.B. and D.E. Mathre. 1971. Biology and control of ergot in male-sterile wheat and barley. Phytopath. 61:1075-1080.
14. Weniger, W. 1924. Ergot and its control. North Dakota Agr. Exp. Sta. Bull. 176, 21 p.

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