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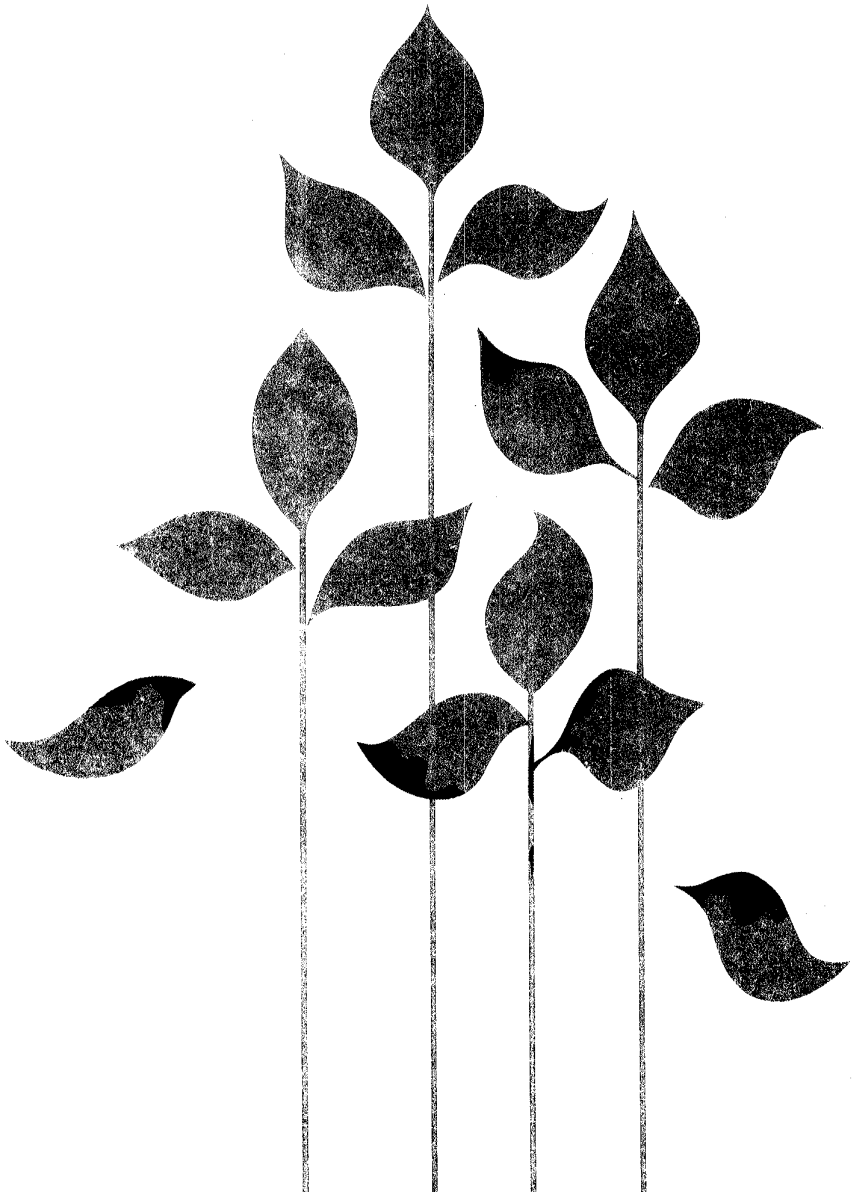

Agriculture Canada

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

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

First occurrence of a severe white leafspot on chinese mustard in Canada

Andres A. Reyes[1]

Pseudocercosporella capsellae was observed to be associated with severe white leafspot on chinese mustard.

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Pseudocercosporella capsellae a été observée en rapport avec l'apparition d'une grave tache blanche dans une culture de chou de Chine. (*Brassica campestris* L. Chinensis Group).

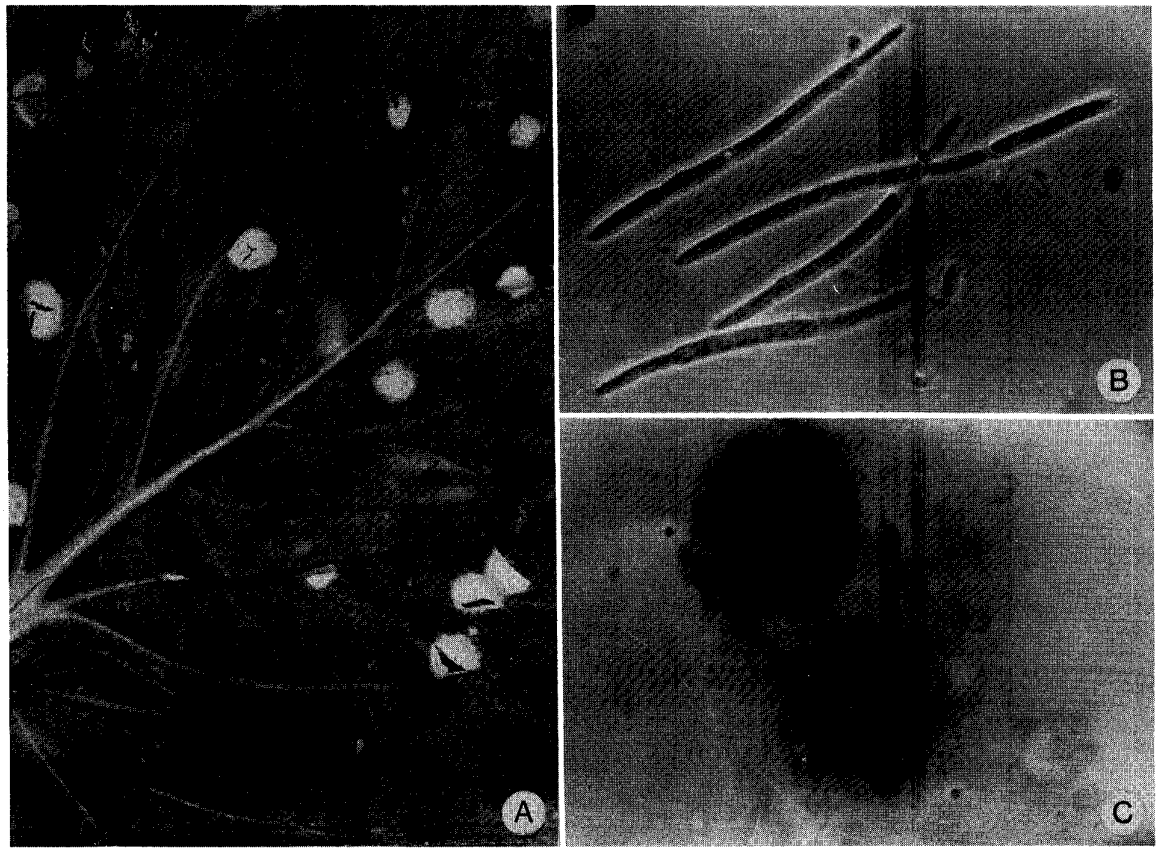

Figure 1. White leafspot associated with ***Pseudocercosporella capsellae.*** (A) Papery white spots on chinese mustard leaf (X 0.6). (B) Conidia (X 1,300) and (C) stromata (X 1,200) of ***P. capsellae.***

A severe leafspot disease was observed on a crop of 8 wk old chinese mustard (sometimes known as celery mustard, bok (pak) choy, and chongee) (*Brassica campestris* L. Chinensis Group) in a 5 ha farm near Toronto, Ontario on June 5, 1978. The spots were circular and averaged 10 mm in diameter (Fig. 1). The centers of the spots were bleached (papery) white and the slightly zonate margins were light brown. Numerous spots were "slit" at the centers. Some spots were overlapped to form large necrotic areas but all were confined to the bottom leaves. Trimming of the infected bottom leaves significantly reduced the marketable product.

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Microscopic examination of slides prepared from field specimens revealed that *Pseudocercosporella capsellae* (Ell. & Ev.) Deighton = *Cercosporella brassicae* (Fautr. & Roum.) Höhn., = *C. albomaculans* (Ell. & Ev.) Sacc., = *C. nesliae* Dearness & Bisby was associated with the spots (Fig. 1). Structures of the fungus observed included mycelia, filiform conidia and conidiophores all of which were septate and hyaline. Subglobular stromata were dark brown. Conidia were tapered at the ends. The average measurements of 31 conidia and 30 stromata were 3 × 74μ and 47 × 57μ, respectively.

P. capsellae did not grow on 2% water agar, potato dextrose agar or V-8 juice agar. Conidia were still ungerminated on these media after 2 weeks incubation at room temperature but it was not determined whether the ungerminated conidia were viable.

To establish the association of *P. capsellae* with white spot, an inoculum of the fungus (2.3×10^3 conidia/ml) was prepared by vigorously shaking 28 g of naturally infected chinese mustard leaves for 60 seconds in 250 ml of water in a 500-ml flask. This inoculum was applied to the leaves of plants with a Mastercraft vaporizer; seedlings were 6 weeks old when treated. Two plants each of 10 different crucifers were treated and the plants were maintained for 2 weeks in a growth-room (22°C, 75% relative humidity) supplied with 14 hr of artificial light (32,000 lux). White spots (2 to 6 mm diameter) developed on eight of the crops: mustard (*B. campestris* L.); turnip (*B. napobrassicae* (L.) Mill.) 'Laurentian'; rape (*B. napus* L.); brussels sprouts (*B. oleracea* L. var. *gemminifera* Zenker) 'Jade Cross'; broccoli (*B. oleracea* L. var. *italica* Plenck) 'Cleopatra'; cauliflower (*B. oleracea* L. var. *botrytis* L.) 'Snowball Y'; cabbage (*B. oleracea* L. var. *capitata* L.) 'Eastern Ballhead'; and chinese cabbage (*B. campestris* L. Pekinensis Group) 'Springtime'. No leafspot developed on either radish (*Raphanus sativus* L.) 'Scarlet Globe' or horseradish (*Armoracia lapathifolia* Gilib.). Inoculated chinese mustard leaves which served as controls developed severe white spots. Conidia of *P. capsellae* were observed microscopically only from infected leaves, thus, confirming association of the fungus with the disease.

There is no previous record that white leafspot has occurred on chinese mustard in Canada. It was reported on turnip in the maritime provinces and in Ontario, and on chinese cabbage in Quebec (2). The disease has been found, however, on several cruciferous crops in the United States and Ceylon (1,3,4,5).

Naturally infected leaves of chinese cabbage collected on September 6, 1978 were deposited at the Biosystematics Research Institute, Agriculture Canada, Ottawa K1A 0C6 (Lot No. 78M-128, DAOM 169225).

Acknowledgment

I thank Deborah J. Martin for technical assistance and R.A. Shoemaker (Biosystematics Research Institute) for confirming the identification of *P. capsellae*.

Literature cited

1. Bond, T. E. T. 1941. "White spot" of turnips: A disease new to Ceylon. Trop. Agr. 97:17-18.
2. Conners, I. L. 1967. Annotated index of plant diseases in Canada. Can. Dep. Agr. Publ. 1251. 381 p.
3. Crossan, D. F. 1954. Cercosporella leafspot of crucifers. N. C. Agr. Exp. Sta. Bull. 109. 23 p.
4. Davis, W. H. 1927. Notes on the Cercosporella leafspot of chinese cabbage in Massachusetts. Phytopathology 17:669-670.
5. Miller, P. W., and F. P. McWhorter. 1948. A disease of cabbage and other crucifers due to Cercosporella brassicae. Phytopathology 38:893-898.

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[1]

Michael C. Hampson

Seventy-two cultivars of tomato, six cultivars of tobacco, and species of *Capsicastrum, Datura, Physalis* and *Schizanthus* were inoculated with races 2 and 8, or race 2 only of *Synchytrium endobioticum*. The six cultivars of tobacco tested were resistant, but all tomato cultivars and other species tested were susceptible.

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Soixante-douze cultivars de la tomate, six cultivars du tabac et les espèces des genres *Capsicastrum, Datura, Physalis* et *Schizanthus* ont été inoculés avec les races 2 et 8, ou la race 2 seulement de *Synchytrium endobioticum*. Les six cultivars de tabac qui ont été testés se sont montrés résistants; par ailleurs tous les cultivars de la tomate et des autres espèces n'ont pas manifesté de résistance.

Tomato production is a growing industry in Newfoundland, and is presently of some importance to the local (Newfoundland) economy (1). Much of the tomato production, however, is pursued in areas known to be infested with one or more races of *Synchytrium endobioticum* (Schilb.) Perc. (2). Of fifty-one tomato cultivars previously tested at this Station for their reaction to *S. endobioticum*, none was found to be immune to European race 2 or 8 under our experimental conditions.

Since it is well known (3) that *S. endobioticum* can be transferred experimentally to Solanaceous species other than tomato, it was decided to examine the infection reactions of some of the commoner species (particularly some floriculture specimens) to the Newfoundland wart races, along with other tomato cultivars.

The study reported here was carried out to provide further information on the susceptibility of tomato cultivars and selected Solanaceous species to *S. endobioticum*. This study was also done with the hope that one or more species might prove useful for assay or as an indicator of the presence of the wart disease fungus.

Materials and methods

Seventy-two cultivars of tomato (*Lycopersicum esculentum*), six cultivars of tobacco (*Nicotiana tabacum*), and species of *Capsicastrum* (Jerusalem Cherry), *Datura* (Angel's Trumpet), *Schizanthus* (Butterfly Flower) and *Physalis* were tested.

The seedlings of all cultivars and species were treated the same in regard to cultural practices, and grown under conditions similar to those used in the earlier study (1). The methods of inoculation included: a) transplanting seedlings into sporangia (resting spore)-infested mix (1000 sporangia/g mix); b) dragging root systems through sporangial slurries, and then planting the seedlings in non-infested mix. The potting mix was sterilized peat:perlite:water, 2:1:1/2 (v/v).

Tomato seedlings were inoculated at the four-leaf stage (about 4-wk old); other species were inoculated at 1, 2, 3 or 4 wk after emergence. Seedlings were examined macro- and microscopically (25 X) at 4 wk after inoculation (Figure 1). The seedlings were indexed at harvest as in the earlier study: L 5-20 resting spores; M 20-100; H > 100. European race 8 was used in some of the tomato tests (Table 1), and race 2 was used to inoculate all the tomato, tobacco and other species tested (Tables 1, 2 and 3). Generally, the seedlings were top-irrigated, although a test was made of bottom-irrigation to observe its influence on disease expression.

Results and discussion

It can be seen (Tables 1 and 2) that all the tomato cultivars tested were susceptible to race 2. These cultivars tested with race 8 (Table 1) were also susceptible to that race. These results were predictable since the same pattern appeared in the earlier tests (1). Small differences in virulence between races 2 and 8 were found. It was found in the earlier study that 13% and 7% of the specimens, respectively, were free from infection by race 8 and race 2. In the present study, the percentage escapes were 2 and 5, for races 8 and 2 respectively. Since only five seedlings were used in each test, it is likely that these differences reflected experimental errors. Neither mode of inoculation nor irrigation influenced the incidence of disease among the tomato cultivars.

[1] *Contribution No. 58, Research Station, Research Branch, Agriculture Canada, P. O. Box 7098, St. John's, Newfoundland A1E 3Y3*

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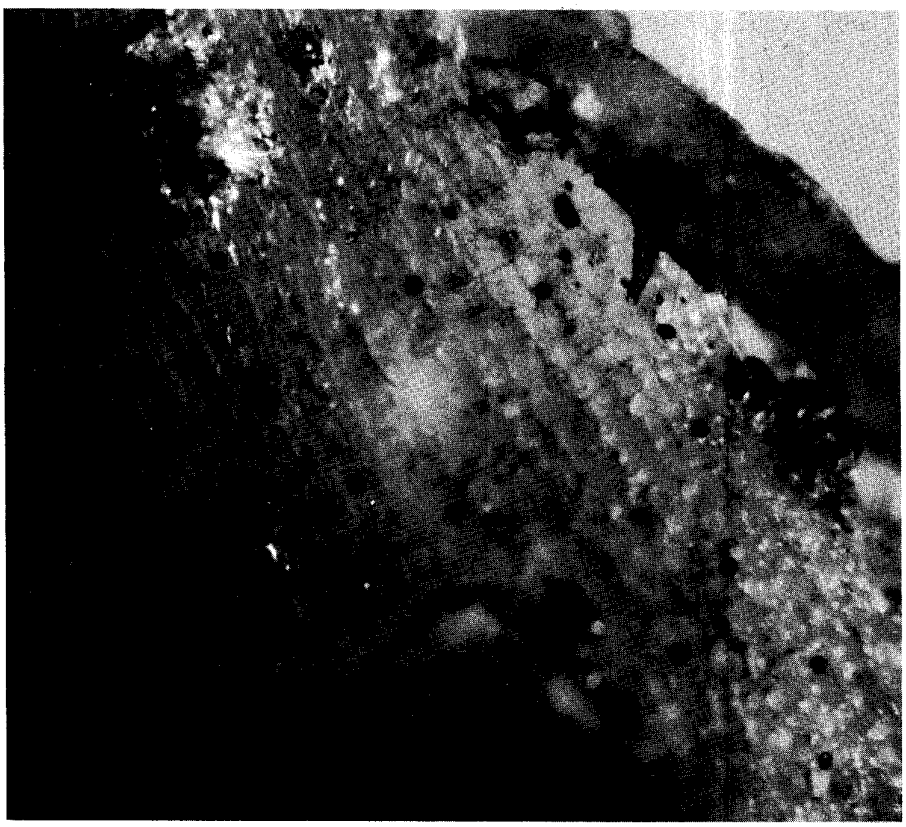

Figure 1. Length of infected tomato root with resting sporangia of *Synchytrium endobioticum.* They appear in the photograph as black dots: magnification 25 X, enlarged 4.7 X.

Table 1. Infection of 36 tomato cultivars inoculated with races 2 and 8 of *Synchytrium endobioticum*.

Tomato cultivar	No. of seedlings[1]/ infection level[2]					
	Race 2			Race 8		
	L	M	H	L	M	H
Bashet Pak	3	2	0	0	5	0
Beef Globe Master Hybrid	2	3	0	2	3	0
Better Boy Hybrid	2	3	0	1	4	0
Burpee Hybrid	3	2	0	0	3	2
Burpee's Pixie Hybrid	2	3	0	0	3	2
Burpee's Sunny Brook Earliana	1	0	4	0	3	2
Cardinal	2	3	0	2	3	0
Delicious	1	4	0	0	4	1
Early Girl Hybrid	5	0	0	1	4	0
Glamour	3	2	0	2	3	0
Golden Queen	4	1	0	3	1	0
Heinz 1370	4	1	0	3	2	0
Heinz 1439	0	4	1	1	1	3
Jet Star	3	2	0	2	3	0
Jubilee	1	3	1	0	4	1
Marglobe	4	1	0	2	3	0
Michigan Ohio	0	3	2	0	4	1
Michigan Ohio Forcing	1	4	0	3	2	0
Monalucie	1	4	0	1	3	1
Moreton Hybrid	4	1	0	3	2	0

Table 1. (cont.)

Tomato cultivar	No. of seedlings[1]/ infection level[2] Race 2 L	M	H	Race 8 L	M	H
Ohio WR	3	2	0	1	3	1
Orange Queen	3	1	0	3	2	0
Ottawa 78	0	4	1	0	5	0
Outdoor Girl	0	4	1	1	3	1
Oxhart	3	0	1	2	3	0
Rapids	2	2	0	3	2	0
Rushmore VF	3	2	0	0	3	2
Setmore	1	4	0	1	4	0
Small Fry	5	0	0	0	4	1
Star Cross	3	2	0	0	5	0
Stokes Early Hybrid	1	1	0	3	2	0
Tiny Tim	4	1	0	4	1	0
Tropic	3	2	0	1	4	0
Tuckcross	0	2	3	0	2	3
Ultra Girl VFN	4	1	0	4	1	0
Vinequeen	1	2	2	0	3	2

[1]Five seedlings used in each test
[2]Infection level based on No. of resting spores/seedling:
L = 5-20; M = 20-100; H = > 100.

Table 2. Infection of 36 tomato cultivars inoculated with race 2 of *Synchytrium endobioticum*.

Tomato cultivar	No. of seedlings[1]/ infection level[2] L	M	H
Beefmaster	0	1	4
Burpeana Early Hybrid[4]	1	4	0
Burpee's Big Boy Hybrid	3	2	0
Campbell[4]	0	5	0
Early Chatham[4]	2	3	0
Early Red Chief	1	4	0
Early Summer Sunrise[4]	1	3	1
Fireball[4]	2	3	0
Gardener[4]	0	4	1
Heinz 1350	0	3	2
Jet Fire	3	2	0
Moneymaker[4]	3	1	1
New Yorker	5	0	0
Ohio MR 12[4]	2	3	0
Ohio WR 25[4]	4	1	0
Patio Hybrid[4]	4	1	0
Pearson	0	5	0
Presto	3	2	0
Ramapo	2	3	0
Red Top[4]	4	1	0
Rutgers	4[3]	0	0
Scotia[4]	1	3	1
September Dawn	4	1	0
Star fire	4	1	0
Sunray	3	2	0
Supersonic	1	4	0
Swift	2	3	0
Traveller	0	2	3
Tuck Queen[4]	3	2	0
Vantage	1	3	1
Veegan	2	3	0
Veemore[4]	3	1	1

Table 2. (cont.)

Tomato cultivar	No.of seedlings[1]/ infection level[2] L	M	H
Veeroma[4]	0	4	1
Vetomold[4]	0	5	0
Vivid	0	4	1
Wonder Boy[4]	1	4	0

[1]Five seedlings used in each test.
[2]Infection level based on No. of resting spores/seedling: L = 5-20 sporangia/plant; M = 20 = 100; H = > 100.
[3]One seedling failed to grow.
[4]Inoculated by slurrying roots.

Table 3. Number of resting spores found in the root system of seedlings inoculated with *Synchytrium endobioticum* at different times after emergence.

Species or cultivar[1]	Week after emergence 1	2	3	4
Nicotiana tabacum				
White	0	0	0	0
Crimson King	0	0	0	0
Dwarf Crimson	0	0	0	0
Lime Green	0	0	0	0
Daylight Sensation	0	0	0	0
White Bedder	0	0	0	0
Schizanthus sp.	0,1,1,4	0,0,0,4	0,0,1	0,1,12
Physalis franchetii	0,0,1,3	1,2,3,4	0,3,3,9	2,3,7,10
Capsicastrum nanum	2,4,10,15	3,6,11	3,6,7	0,1,49
Datura sp.				
Fastuosa	0,4	7,9	–	–

[1]Five seedlings per week inoculated by slurrying.

The numbers of resting spores in the root systems of the other test seedlings were generally low (Table 3). Although other workers have succeeded in infecting tobacco species (not *tabacum*) under experimental conditions (4), we were unsuccessful. Susceptibility varied with time: increasing (*Physalis franchetii*) or decreasing (*Capsicastrum nanum*) with time after emergence.

It is concluded, therefore, that tomato is generally and equally susceptible to races of *S. endobioticum*. Tomato is seemingly infected with ease irrespective of the modes of inoculation or water supply. None of the other species tested appear to warrant further work on them. The use of tomato as an assay and indicator plant will be pursued, as will its reactions to other wart races present in Newfoundland.

Acknowledgement
I wish to thank Janet Coombes, technician, for her patient work in counting resting spores.

Literature cited

1. Hampson, M. C. 1976. Infection of additional hosts of *Synchytrium endobioticum*, the causal agent of potato wart disease: 1. Tomato. Can. Pl. Dis. Surv. 56:93-94.
2. Hampson, M. C., and Proudfoot, K. G. 1974. Potato wart disease, its introduction to North America, distribution and control problems in Newfoundland. FAO Plant Prot. Bull. 22:53-64.
3. Karling, J. S. 1964. Synchytrium. Academic Press, New York. p. 470.
4. Phadtare, G. G., and Sharma, K. P. 1971. Additional hosts of *Synchytrium endobioticum*. Ind. Phytopathology 24:389-392.

Research on potato wart disease in the U.S.S.R. - a literature review (1955-1977)

Michael C. Hampson[1]

Potato wart disease, recorded in the Soviet Union in 1935, is now found in all six Western Soviet Socialist Republics. It is particularly evident in the Trans-Carpathian mountainous zone of the Ukrainian S.S.R. Since the establishment of a Potato Wart Disease Research Station in Trans-Carpathia in 1944, extensive work has been carried out on the pathology, biology and physiology of the disease and its causal agent. Particular emphasis has been paid to the elucidation of the mechanism of resistance. Since biotypes were discovered in 1962, work has been devoted to immune cultivar production and selection for resistance. A well systematized method is used for crop inspection, quarantine and disease management. Chemical controls have also been developed.

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La galle vérruqueuse de la pomme de terre, signalée en 1935 en Union Soviétique, touche maintenant les six républiques soviétiques socialistes de l'ouest. Elle sévit principalement dans la zone montagneuse transcarpatique de la république ukrainienne. Depuis la fondation en 1944 d'une Station de recherche sur la galle verruqueuse dans cette région, de vastes travaux ont porté sur la pathologie, la biologie et la physiologie de la maladie et de son agent pathogène. On s'est particulièrement intéressé à la compréhension des mécanismes de résistance. Depuis la découverte de biotypes en 1962, les recherches ont été consacrées à la production de cultivars immuns et à la sélection de variétés résistantes. On a élaboré une méthode bien structurée pour l'inspection, la surveillance sanitaire des cultures et l'éradication de la maladie. On a également mis au point des techniques de lutte chimique.

Introduction

Potato wart disease caused by *Synchytrium endobioticum* (Schilb.) Perc. was first described, from Hungary, in 1896. It has been present in Newfoundland, Canada, since the 1900's (16), and is no longer present in the U.S.A. (2,3).

It was extensively reported from 1898 onwards in Western Europe, but not observed in U.S.S.R. until 1935. It is generally conceded (22) that most infection foci in the Ukrainian S.S.R. – the most extensively infested area in Russia - originated from tubers imported from Western Europe during the German territorial occupation of the 1940's.

There is probably no other area in the world where the potato wart disease problem has been tackled with such vigor and vision as in the U.S.S.R. This article reviews work published in the Soviet Union from 1955-1977.

The information for this article was made available from translations of work available to Western observers supplied by the Multilingual Division, Translation Bureau, Secretary of State Department. As far as possible, the phraseology of the translators is used. For example, the uses of the terms "pathogenicity/aggressivity/virulence" are not clearly defined. Therefore, when pathological terms appear in the text and appear to run counter to accepted usage, or are ambiguous, they have been marked with an asterisk, and the phraseology has been transferred verbatim from the translated copy.

Research centers

The main research center was located at Boyany, near Chernovtsy, in 1944, as most foci of potato wart disease were found in the so-called Carpathian arc (Fig. 1). At that time, the station was given a mandate to pursue research into determining immunity, resistance and tolerance of potato varieties* to wart disease with special emphasis on the mechanism of resistance. The station was staffed by approximately twenty research scientists grouped into five sections, and equipped extensively with much modern apparatus (17).

Laboratory, greenhouse and field work is pursued on biological, biochemical and biophysical questions at Boyany. Biochemical work on the mechanism of resistance is also carried out at other institutes, such as the Potato Research Institute, Moscow, the Ukraine Scientific Institute for Plant Physiology, and the Institute of Plant Pathology, Leningrad.

Field testing, for wart resistance, at Chernovtsy is done at high altitudes, where most infested areas are located. Substations at Leningrad, Russian S.F.S.R., Minsk, Byelorussian S.S.R.; and Vilnius, Lithuanian S.S.R. are also concerned with screening varieties for wart reactions.

Hosts affected

Soviet workers have examined the reactions of tomato cultivars and a large number of wild Solanaceous species

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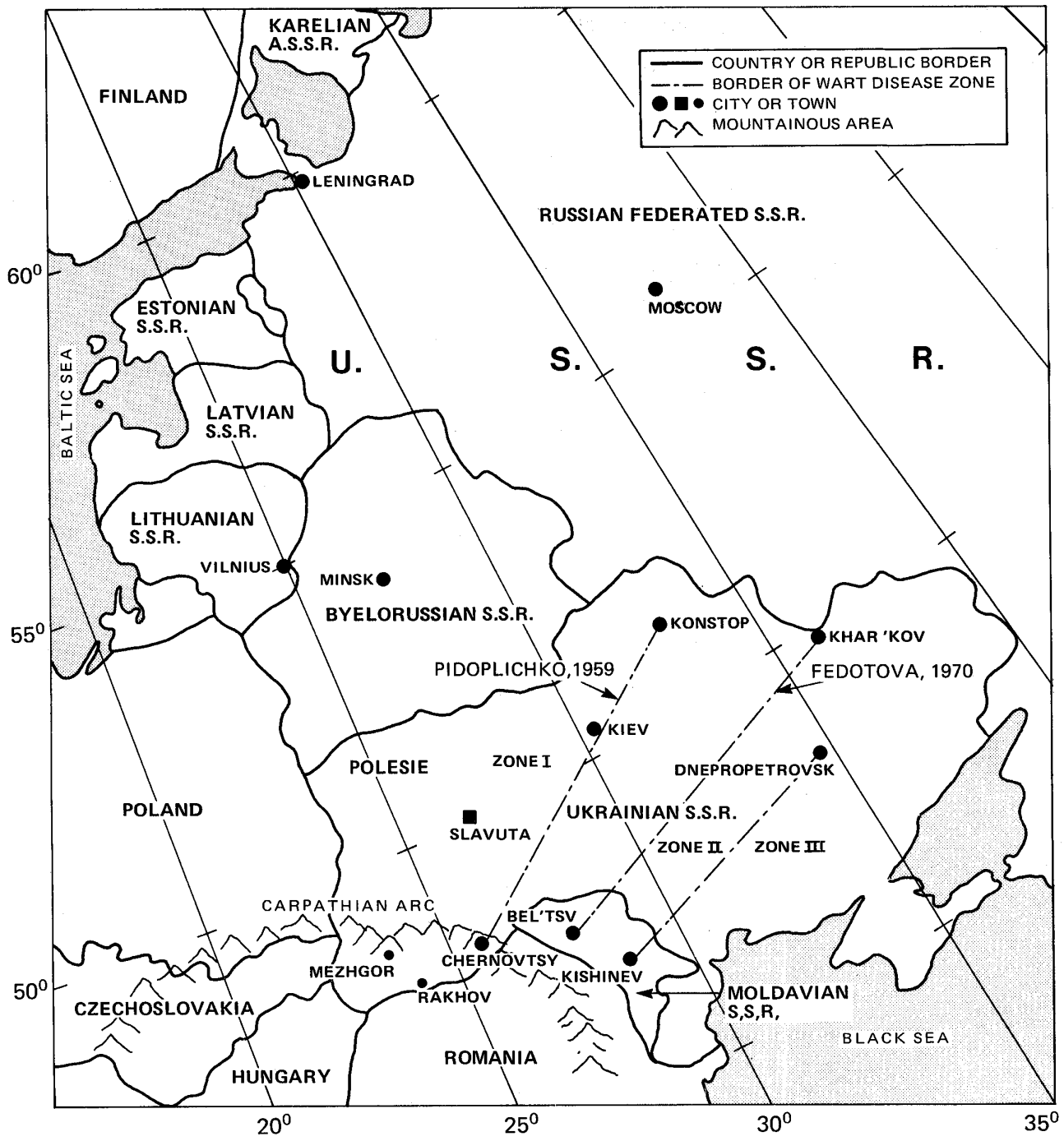

Figure 1. Outline map of Western Russia and adjoining countries, showing locations of major research centers. The sites of original discovery of the common biotype and two other biotypes are shown. Lines connecting cities in Ukrainian and Moldavian S.S.R. delineate zones favourable and unfavourable to establishment of potato wart disease.

to infection by *S. endobioticum* (47). On some species the life cycle of the fungus may not be completed following infection, and visible symptoms may not develop. Potato plants were found to be more affected by sporangia obtained from other Solanaceous plants than by those obtained from potato plants (22). Several South American potato species were found to be susceptible to the Soviet Union biotypes (19).

Economic importance

The disease was first reported from Slavuta, in West-Central Ukraine, in 1935 (22). Potato wart disease is now known in the Russian Federated, Estonian, Latvian, Lithuanian, Byelorussian and Ukrainian Soviet Socialist Republics. (Fig. 1).

As of January, 1968, more than 16,000 ha of land in Russia were infested. The numbers of infested sites reported were: farms - 274; household plots - 118,472. In the Ukraine, disease foci are found in the Carpathian arc, the Polesie and Lesostep areas. Koretsky (24) estimates that 70% of the potato crops are concentrated in these areas. Household plots comprise nearly 90% of the infested area which represents about 56% of the area under potato crops.

In the Western Ukraine, wart disease was discovered in 84% of the populated areas, and in 98% of the districts. Only in isolated cases was the disease discovered on collective and state farms (40). In Trans-Carpathia, the disease is concentrated mainly in the highlands. In the valley areas the disease is minimal even when infected tubers imported from the infested highland areas are planted.

Pidoplichko (40) reported that all the concentrations of the disease were found northwest of a boundary connecting Chernovtsy, Kiev and Konstop (Fig. 1). Fedotova (9) challenged this distribution which was based on actual field findings. She used soils from different zones of the country, in bio-climatic chambers. She showed that the southern range could be extended much further south with the boundary passing through Bel'tsv and Khar'kov (Fig. 1).

In the Ukrainian foothills and mountainous areas, the losses are extensive in certain years. In areas east of Carpathia, the losses are minimal (40). Although tuber yield is not markedly reduced, in some cases infected tubers rot quickly and thereby infest the soil. In loss situations, 13-84% yield reduction occurs. In the Leningrad region in 1967, the crop loss was said to be as high as 96%. Yield reduction is at times found to be in tuber size rather than in number.

Regulations require the destruction of all plants within a 3-m radius of an infected plant. This increases the losses suffered by the householder in garden plots. Commercially, crops from an area placed under quarantine must be processed. Hence, an immediate reduction in the value of the crop occurs when the disease is discovered in the seed production areas (24).

Etiology

Sporangial studies: Miloslavova et al. (33) and Golik (14) developed an extraction system for isolating zoosporangia* from plant tissue uncontaminated with organic and mineral residues. The wart tissue was crumbled, dried and screened. Sub-samples were collected on 40-60 μm mesh and dried. Sporangia were floated off from the powder on a mixture of CCl_4 and sulfuric ether. Varying the mixture ratio yielded sporangia of different degrees of physiological maturity.

The sporangia were found to undergo maturation during the resting period. Germination followed if the resting period was accompanied by sufficient warmth and moisture (22). Vladimirskaya (54) reported on observations made over 5 years on the dormant character of the fungus. She found that about 3/4 of a sporangial population germinated in the year of production, 1/5 the following year, and the remainder over a long time base. These groups were designated: annual, biennial and perennial. Under laboratory conditions 60-75% germination occurred in the summer. This coincided with the host's tuberization phase. Under natural conditions, wintering retarded germination of the two dormant groups. Among the "annuals", the complete life-cycle took place in 21 days. Prolonged drought retarded germination, rainy summers accelerated germination.

Sporangial viability was determined by plasmolysis in NaCl, Na_2SO_4, NH_4Cl or NH_4NO_3, and confirmed by deplasmolysis (22). Mirzabekian (36) attempted to culture tumour tissue *in vitro*. He seeded disinfected tumour pieces (3-5 mm) on various nutrient media at pH 5.2-6, in the dark, at 24-25°C. Best gall stimulation occurred on galls harvested in September and cultured on a medium supplemented with coconut milk, next best on a gall extract or potato shoot extract supplemented medium. Growth ceased after 70-80 days. Summer sporangia were seen but no winter sporangia were found. In a later report, Vinkler and Lipsits (53) cultured wart tissue on White's medium and observed sporangia formation.

Biotype studies: The first biotypes of *S. endobioticum* were discovered in Czechoslovakia and Germany in 1941/2. Cultivars in the U.S.S.R. were tested against the new biotypes from 1957 on (23). Tests were also made against the pathogen obtained from four different Russian climatic zones. While none of the resistant cultivars broke down, degrees of susceptibility were found among control cultivars. Further testing of these differences revealed greatest pathogen activity was displayed from zones in the order Chernovtsy/Minsk > Leningrad/Vilnius. The pathogen from zones of greatest occurrence of the disease were said to be most active* (10). Biotypes were later discovered in the Carpathian mountains in 1961-62 (55). They were different from the German biotypes discovered in 1941, '50, '51 and '52. Three biotypes were discovered, named according to the sites of discovery: Mezhgorsky, Rakhovsky and Bukovsky (22, 55). Naturally, the discovery of biotypes, in Trans-Carpathia, stimulated extensive studies on biotypes. In East and West Germany, between 1942 and 1960, 9 biotypes had become established, in East Germany alone 44 such foci had been determined. There was concern that a similar set of events would unfold in the Ukraine.

Yakovleva (56) reported on a study of factors responsible for the origin of new biotypes. Known factors to date were severe soil infestation, continuous (potato) crop production, and the wide distribution of resistant varieties. The hypothesis was developed that one of the responsible factors is an adaptive change following nutrition of the parasite upon cultivars of varying resistance. In support of this hypothesis it was found that necrosis, deformation, proliferation and the presence of sori and sporangia were observable on some resistant cultivars. On others, sporangia were observed but symptom expression did not occur even at 40 days after inoculation. The pathogen's activity was found to be intensified* after passaging through resistant cultivars and transferring to further resistant and susceptible cultivars.

Morphological differences between Mezhgorsky and Rakhovsky biotypes were noted (55). The summer sori, respectively, contained 2-3 sporangia and 5 sporangia. The common biotype displayed sori containing 5-9 zoosporangia.

The biotypes were found to not extend their ranges beyond the Carpathian zone (58). The Bukovsky biotype was found to be the least aggressive* of the three (57). Detailed work reported by Yakovleva, V.I. (58) indicated that the Carpathian biotypes may consist of ''smaller specialized biotypes''. An isolate from Leningrad, however, showed evidence of increased pathogenicity* (57). It was noted from the literature that there were indications of the affection of wart resistant potatoes on the Karelian isthmus (44). Work carried out in Byelorussia failed to detect the presence of aggressive* biotypes there (45).

Fedotova and Yakovleva (12) made the first mention in a Russian article of potato wart disease work in Newfoundland, citing Newfoundland at that time as one of the five geographic areas in the world where racial deviation had occurred (the others were: German Federal Republic, German Democratic Republic, Czechoslovakia and U.S.S.R.). Yakovleva (57, 58) dwelt at some length on the Newfoundland findings and concluded that Newfoundland biotypes differed from the Carpathian biotypes.

Epidemiology

Climate: Soviet researchers maintain that the main environmental factors that influence the establishment of wart disease are soil, temperature and precipitation (22). In the U.S.S.R. the fungus is best adapted to a moderate climate, cool and humid summer, and podzol-type soil.

Soil: Gedz (13) examined the influences of mineral salts on infection, in laboratory and field experiments. Mn, Cu and Mo reduced infection. Tarasova (48) observed that B, Zn and Cu stimulated sporangial germination. Tarasova (48) also noted that soil temperature and soil moisture played a large role in decay of wart tumours, and subsequent release of sporangia and germination. The pH level did not appear to influence germination. The rate of decay in soil types was sandy > sandy-loam > loam, and pH 7.0 > 3.85.

Fedotova (9) noted that disease suppression occurred in certain soil types. The degree of disease development was felt to be related to soil type (22). Thus, dry and warm region soils fostered a slow development, piedmont and montane region soils fostered intensive development. The most serious cases of the disease were observed on light sandy and less frequently on clay and muddy soils. Acidic heavy textured structureless podzol enhanced the disease, calcareous chestnut chernozem did not.

Temperature: Pidoplichko (40) extensively surveyed soil temperatures across the Ukrainian S.S.R. He found that, based on soil temperatures, boundaries could be established between areas where the disease would and would not be a threat. He could not correlate the disease occurrence with the duration of the frost-free period, as Hartman had done in Pennsylvania. Disease-free areas had soil temperatures which reached 24-25°C at 10 cm soil depth. He found that sporangial viability was lost at summer temperatures > 30°C. Pidoplichko found 'disease-free area temperatures' in Trans-Carpathian valleys which had remained disease free. Using growth chambers, Fedotova (9) simulated meteorological combinations for different areas of the country. Thus, three zones of disease establishment were created (Fig. 1). Parallel field tests in all regions yielded the following results: in the North (Leningrad), 60-70% infection occurred annually; in the South (Zone III), small excrescences were noted in the first year, then in subsequent years no further disease nor viable sporangia were found. Temperature was suggested as the limiting factor, but since soil moisture and certain soil properties were also involved, its role was not clear.

Precipitation: Where soil temperatures were borderline, precipitation was found to be a limiting factor. The effect of soil type here was thought to be very important since it would influence the amount of retained moisture.

The southern boundary for infection was found to coincide with a July average of 75 mm at 20°C. As the majority of cultivars which were cultivated in the Ukraine form tubers in July, the ecological conditions during this period appeared to play decisive roles in disease development (22, 40). Thus season is also an important factor in disease development. Vladimirskaya (54) pointed out that massive germination of sporangia was observed in July. Tarasova (48) found that moisture levels above 90% and below 40% suppressed sporangial germination. Fedotova (9) observed that no disease developed at 50% or less of soil moisture content.

Control

Legislative: Potato wart disease control in the Soviet Union was originally based on the understanding that the disease was dangerous to potato crops wherever

such crops were cultivated. Thus, the fungus became the object of quarantine and restrictions on shipping potatoes were introduced. Pidoplichko (40) concluded that this notion was invalid, whereupon, based on his findings, the Ukraine was divided into three zones: Zone I - a wide distribution of the disease, resistant cultivars allowed only; Zone II - a moderate disease distribution, locally produced resistant cultivars only; Zone III - unfavorable to the disease.

Resistant cultivars could be grown on scheduled plots 3 years after prohibition of tomatoes, root and tuber crops.

Quick detection and elimination of disease foci are the current aims of control measures (22). This is bound up with a system of crop inspection. All farms, seed producing stations, research stations and household plots are subject to inspection (24). Inspections are made at time of flowering of potato crops. Fields are sampled and farm implements, etc., are inspected. Suspected samples are sent via an agronomist to the quarantining agents. All inspections, and actions taken are recorded. If infection is suspected the plot in question is quarantined. If confirmed, all produce from the plot is cooked for cattle food or destroyed in a cesspool. Infected areas are fenced off and posted. A lengthy, detailed form is completed for each confirmation thus describing the circumstances surrounding the infection.

Agrotechnical control: A complex of measures is instituted following positive confirmation. These include: planting resistant cultivars, bare-fallowing, organic fertilization of the soil, and summer planting of potato. In Byelorussia, it has been shown that disease suppression takes place over a five year period when resistant potato cultivars, cabbage, corn, lupin and bare-fallow are used on infested plots. Bare-fallowing for 3-5 years brings about 97% germination of zoosporangia. Organic fertilizers such as hog, cow or poultry manure cause 75% sporangia to germinate.

Chemical control: Following work in Czechoslovakia, it was suggested that dinitro-orthocresol be tried as a control agent (40, 46). Chloropicrin was tried but rejected as too expensive. Over 200 organic and inorganic compounds were tried (22). Nitraphen - a complex mixture of sodium salts, nitration products of alkylphenols, water and a wetting agent - was selected (20). Nitraphen testing was begun in the Leningrad oblast in 1957. It induces granularity and coagulation of sporangial contents. The substance is applied as a solution in May-June to fallowed ground (21).

Calcium cyanamide was introduced as a control chemical since it, too, was found to be effective when harrowed in at the rate of 150 g/m^2. It is widely available, inexpensive and useful for both commercial and home-garden plots. Carbamide is also used. At the rate of 1.5 kg/m^2, after 3 years the fungus could no longer be found (49).

Biological control: Small farming allotments and household plots are the most common sources of the disease. Since the dangers and expense of chemical treatment of these sources are high, work had been initiated on finding a biological control (24, 37, 40). Mirzabekian et al. (37) cultured actinomycetes on potato slices. When diluted with sand, the cultures were incorporated into infested pot mixes. It was found that such treatment reduced disease incidence from 97% to 25%. Treatment of soil and tubers produced the best result. A 3-year treatment program, it was suggested might eliminate infection from infested soils.

Selection for resistance: Selection for resistance is complicated by two factors. On the one hand, Fedotova (8) found that host reactions varied from highly resistant, weakly resistant to susceptible. Thus resistant cultivars used to clean soil may perpetuate the disease since the pathogen can penetrate some resistant varieties and complete its developmental cycle. On the other hand, there is more than one biotype (19). The presence of the Carpathian biotypes - though not a threat to the potato industry - complicates selection work. South American potato species are used as sources of resistance. Interspecific hybridization is used in the breeding work at Chernovtsy (43). Numerous promising cultivars have thus been produced. As a result of a wide study of wart-resistance material at the breeding establishments, Bondarenko (1) concluded there were four levels of resistance to potato wart disease: viz 'high resistant', 'resistant', 'low-resistant', and 'susceptible'.

The study of wart resistance was initiated in 1957, and intensified in 1965 following the discovery of the Carpathian biotypes. Under a more closely developed screening process, cultivars that had appeared resistant under field conditions appeared susceptible under laboratory conditions. Saltykova (43) noted that the percentage of Soviet selections susceptible to the Rakhovsky and Mezhgorsky biotypes was much higher than that percentage susceptible to the common biotype.

Biochemical studies

The aim of the studies into the mechanism of resistance was to place the selection of wart-resistant cultivars on a scientific foundation through the biochemical determination of the nature of resistance (25). Through biochemistry, the mandate was to develop a method of rapid determination of resistance, thus shortening the time taken for testing breeders' selections for resistance.

In the forefront of this research stood D.V. Lipsits who authored or co-authored much of the work on resistance mechanisms, until his death in the early seventies. The three areas of study that were developed were: differences in physiology between resistant and susceptible plants; differences in protein between resistant and susceptible plants, and suppression of tumour development.

Lipsits believed that susceptible cultivars must be able to ensure the vigour of the physiological and biochemical

processes necessary to ensure pathogen development and tumour production. Although all classes of cultivars can be attacked, only in susceptible tissue does the fungus proceed to develop secondary infections (28). Therefore, the cause of resistance, it is stated, is not hypersensitivity but an absence of energetic cellular processes. In Lipsits' opinion, the zoospore affects large protein molecules, in their turn bringing about accelerative changes in the entire cellular metabolism. On this hypothesis, potato wart disease could be diagnosed by estimation of SH-groups, dye absorption, methionine-S^{35} levels, protease activity, fermentative susceptibility of protein, protein immuno-chemistry, amino acid and polypeptide composition, protoplasmic and cell membrane permeability (27, 28).

Respiratory levels: Lipsits and Eisinger (30) measured respiratory levels in tissues taken from resistant, susceptible and infected plants. They found that infection was accompanied by an elevated respiration which remained high in susceptible sprouts but fell in leaf and root tissues. Lipsits (28) did not find any differences in the oxidation systems of resistant and susceptible cultivars, and no qualitative changes were found in fermentative respiration. Lipsits also examined differences in polyphenol levels in these plants. He noted that polyphenols accumulated only in susceptible cultivar tissue. He concluded that the polyphenols contributed to disease development.

Auxins: Reingard and Pashkar' (41) examined the differences between resistant and susceptible plants in respect of their auxin content. Increases in auxins were principally noted in the periphery of tumour tissue. More auxin-type substances were found in the free than in the bound fraction. Pashkar' (39) later demonstrated that specific physiologically active substances produced by roots were found essential to tumour development.

Sulfhydryl groups: Lipsits (26) analyzed the differences in number and localization of SH-groups in resistant and susceptible sprout tissue. The quantity of SH-groups in infected sprouts increased before the first visible signs of infection. Also, differences were noted in quantity of SH-groups in leaf and tuber tissue between resistant and susceptible plants. Young tumour tissue was most active in labelled P and S uptake. The proteins of the susceptible cultivars had an enhanced ability to incorporate labelled methionine. This was felt to be an excellent indicator of the degree of immunity.

Peroxidase activity: Kadyrmatov (18) studied the changes brought about in the peroxidase complex of resistant and susceptible sprouts at 2, 3 and 6 days after inoculation with *S. endobioticum*. Negligible changes were noted in the complex from susceptible plants. This finding was held to confirm Lipsits' thesis that an intensive metabolism of the host plant is necessary for attack by *S. endobioticum*.

Free radicals: Oxidation process levels are reflected in the changes of free radical levels (31). Healthy, infected, resistant and susceptible potato sprouts were analyzed for free radicals. Free radical levels were observed in the order: infected susceptible sprouts > suscepticle sprouts > resistant sprouts. Dolyagin et al. (4, 6) reported a much higher level of free radical states in the epidermis of resistant tubers than in that of susceptible ones.

Proteins: Okanenko and Bershtein (38) studied the differences in hydrolyzed alkali-water-soluble and 'residual' protein in tubers and tumours. In susceptible tubers, alkali-soluble protein fraction displayed quantitative increases in amino-acid components, and in peripheral tumour tissue quantitative and qualitative increases in 'residual' protein components were found. The new protein material was regarded as a plant reaction to *S. endobioticum*. Lipsits (26) regarded the new or modified proteins in tumour tissue as active factors influencing the course of infection.

Fedotova et al. (11) extracted the proteins of the fungus, noting characteristic differences between specimens taken from different geographic zones, and between different biotypes. Golik et al, (15) then compared the fungal proteins with host plant proteins. A high level of protein similarity existed with susceptible plants, and as the degree of resistance increased there was a decrease in protein fractions common to incitant and host.

Timchuk and Lipsits (50) demonstrated differences in nucleoproteins between resistant and susceptible shoot tissue. Nucleoproteins from the latter could be enzymatically cleaved more rapidly than those from the former tissue. Timchuk and Lipsits (51) suggested that the stronger* protein reaction in susceptible tissue could be accounted for in terms of both accumulation of proteins as well as changes in molecular structure, and could both explain the nature of resistance and aid in developing an accelerated method for resistance diagnosis.

Dolyagin et al. (5) extracted DNA from normal and tumourous potato tissue. They found a significantly higher DNA content in the tumourous tissue, with an absorbance at 320 $m\mu$ not present in the normal tissue extract. Reshetova et al. (42) also examined nucleoproteins. They established that DNA and RNA were more easily extracted from wart-susceptible cultivar shoots, but a greater amount was extracted from resistant shoots. They suggested that there existed a greater rigidity of the nucleic acid-protein bond in resistant than in susceptible tissues, and that this rigidity could explain the passive background activity of resistant tissues. Thus, activation of metabolism (Lipsits' thesis) after infection of resistant tissue may be made more difficult.

Chemotherapy: Work on disease physiology logically lead to the use of tumour suppressants. Lipsits (26) blocked the balance of SH-groups in susceptible tissue in the laboratory, and prevented tumourigenesis. Field tests, however, failed since the suppressants became phytotoxic. Propylgallate was shown to be an effective tumour suppressant. Lipsits et al. (32) also demonstrated the effectiveness of ionol (2,6-di-ter-butyl-

4-methyl-phenol) dispersed in Tween-60. Later, it was found that Tween-60 alone would suppress tumour formation. Miloslavova et al. (34) examined the effects of gossypol, ionol, Tween-60 and polyethyleneglycols of different molecular weights. These substances did not prevent pathogen penetration nor its development, but manifested anti-tumoural properties only. It was thought that the presence of degenerate tumour cells sensitized the tissue to the anti-tumoural agents.

Present research needs

The present needs of research into potato wart disease by Soviet researchers are summarized by Ephremenko (7):

1. Collaboration of specialists from other countries having the disease, to solve problems of quarantine and plant protection.
2. Standardization of evaluation of resistance of selection material, since the main method of control is breeding for resistance.
3. Development of effective methods of soil disinfection.
4. Improvement of soil samplings methods, assessment of levels of sporangia in soil, and methods of viability determination.
5. Development of trustworthy, indirect methods of diagnosing wart disease through the study of the nature of immunity and host/pathogen relationships.

Conclusion

The potato wart disease story in U.S.S.R., to date, is a fascinating unfolding of a wide-ranging but concentrated effort to come to terms with the world's number one potato disease. The story obviously is far from finished and we look forward to learning more about the attempts to control the disease, and the discoveries yet to be made in its etiology and epidemiology, and in the biology of the causal agent.

Aside from the concerted and organized effort to manage the disease, two points are particularly striking since they appear to be so disparate from our experience with the (disease and its) causal agent. The first is that Soviet workers can apparently clean their land within five years, whereas we find the organism capable of remaining dormant for more than 30 years. The second is that in the Soviet Union, it is observed that three-quarters of a population of the agent germinate in the first year of production, but we calculate that the half-life of the propagule under Newfoundland conditions cannot be less than 14 months, and 10% of a population is still present after four years. Thus, under Soviet soil conditions, the rate of sporangial loss appears to be about twice that for Newfoundland.

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Literature cited

1. Bondareko, E.E. 1973. Results of a study of the degree of potato resistance to the canker agent *Synchytrium endobioticum* (Schilb.) Perc. (in Russian, English summary). Trudy VN11 Zaschity rastenii 36, 95-97.
2. Brooks, J.L., J.B. Given, J.F. Baniecki and R.J. Young. 1974. Eradication of potato wart in West Virginia. Plant Dis. Rept. 58: 291-292.
3. Craig, F.W. and M.E. Gallegly. 1964. Potato wart persists in West Virginia. Plant Dis. Rept. 48: 468-469.
4. Dolyagin, A.B., M.S. Zschedek, K.W. Krugliakova and D.V. Lipsits. 1970. Free radicals in shoots of potato under normal conditions and potato wart (*Synchytrium endobioticum*) (Schilb.) Perc.) and action of substances suppressing tumour development (in Russian). Mikol. i Fitopatol. 4: 229-234.
5. Dolyagin, A.B., K.E. Krugliakova and D.V. Lipsits. 1974. DNA isolated from healthy potato tissues and from tumours caused by *Synchytrium endobioticum* (Schilb.) Perc. (in Russian). Mikol i Fitopatol. 8(4): 345-347.
6. Dolyagin, A.B., D.V. Lipsits and K.E. Kruglyakova. 1975. Investigation of the level of free radical states in the rind of potato sorts differing as to cancer resistance (in Russian, English summary). Izv. Akad. Nauk S.S.S.R. Ser. Biol. 6, 921-923.
7. Ephremenko, T. 1977. Wart disease (*Synchytrium endobioticum* (Schilb.) Perc.) and its control in the Soviet Union. pp 73-74 *in* 1st. Rep. Working Party on Potato Wart Disease. EPPO Publications, Series Cn. 50, Paris, France.
8. Fedotova, T.I. 1959. On the biology of *Synchytrium endobioticum* (Schilb.) Perc. and the immunity of potato varieties to wart disease (in Russian, English summary). Sbornik CSAZV Rostlinna Vyroba 32(6): 165-174.
9. Fedotova, T.I. 1970. Zones of potential distribution of the potato wart pathogen (in Russian, English summary). Byul. VN11 Zashchity rastenii 15, 15-17.
10. Fedotova, T.I., E.F. Karaseva and M.I. Rakovich. 1957. Differences in the activity of the causative agent of potato canker (in Russian). Doklady Vses. Akad. Sel'skok. Nauk im. V.I. Lenina 22(9): 31-33.
11. Fedotova, T.I., B.B-O. Gromova and I.V. Golik. 1972. The electrophoretic and immunochemical properties of the proteins in *Synchytrium endobioticum* (Schilb.) Perc. (in Russian). Mikol. i Fitopatol. 6(1): 68-70.
12. Fedotova, T.I. and V.I. Yakovleva. 1964. Characteristic of the biotypes of the potato wart fungus in the Carpathian mountainous zone of U.S.S.R. (in Russian). pp. 78-97 *In* T.I. Fedotova, ed. Rak kartofelya i mery bor'by s nim. Kolos Publishers, Leningrad, U.S.S.R.
13. Gedz, S.M. 1957. Effects of Manganese, Boron, Molybdenum and Copper micro-elements on the rise of canker immunity in potatoes (in Russian, English summary). Rept. Acad. Sci. Ukraine 6, 605-608.
14. Golik, I.V. 1973. Method of isolation and fractionation of zoosporangia of the potato wart causative agent, *Synchytrium endobioticum* (Schilb.) Perc. (in Russian. English summary). Trudy VN11 Zaschity rastenii. 36, 112-116.
15. Golik, I.V., B.B-O. Gromova and T.I. Fedotova. 1973. The immuno-chemical similarity of the proteins of *Synchytrium endobioticum* (Schilb.) Perc. and the host plant (in Russian, English summary). Trudy VN11 Zaschity rastenii. 36, 107-111.
16. Hampson, M.C. and K.G. Proudfoot. 1974. Potato wart disease, its introduction to North America, distribution and control problems in Newfoundland. FAO Plant Prot. Bull 22(3): 53-64.
17. Hulea, Ana. 1967. A visit to the station for potato canker in the Ukrainian Soviet Socialist Republic (in Rumanian). Prob. Agr. 19(10): 88-90.
18. Kadyrmatov, I.N. 1973. The change of the peroxidase complex in the sprouts of potato tubers after their inoculation with the

pathogen of *Synchytrium endobioticum* (Schilb.) Perc. (in Russian, English summary). Trudy VN11 Zaschity rastenii. 36, 102-106.

19. Kameraz, A.Ya. 1968. Development of potato varieties which are resistant to cancer, p. 20-22. In, The World Potato Collection in the service of socialist agricultural production (in Russian). Trudy Po Prikladnoi Botanike, Genetike 1 selektsii 39(1): 241-260.
20. Kharitonova, Z.M., A.S. Volovik and V.P. Tarasova. 1964. Commercial testing of nitraphen in the fight against the causal agent of potato canker (in Russian). pp. 152-159 *In* T.I. Fedotova, ed. Rak kartofelya i mery bor'by s nim. Kolos Publishers, Leningrad, U.S.S.R.
21. Kharitonova, M., V.P. Tarasova and E.P. Larina. 1969. Nitraphen against potato canker (in Russian). Zaschita rastenii 7: 19.
22. Kharitonova, Z.M. and V.P. Tarasova. 1971. Potato Wart Disease (in Russian). Kolos Publishers, Leningrad, USSR. 46 p.
23. Khizhnyak, P.A. 1957. The biology of the pathogen of potato canker (in Russian). Zaschity rastenii ot vreditelei i boleznei 2(4): 41-42.
24. Koretsky, P.M. 1970. Losses from potato canker on household plots in regions of frequent occurrence of this disease in the Ukraine (in Russian). Mikol. i Fitopatol. 4(4): 366-369.
25. Lipsits, D.V. 1955. Biochemistry and potato canker (in Russian). Priroda (2): 62-63.
26. Lipsits, D.V. 1962. Studies on the biochemistry of the immunity of potatoes to canker (in Russian). Biokhimiya Plodov i ovoshchei. Akad. nauk S.S.S.R., 7: 60-84.
27. Lipsits, D.V. 1963. Incorporation of S^{35}methionine into the proteins of varieties of potatoes susceptible or immune to wart disease. Doklady Biol. Sci. Sec. 146(1): 945-948.
28. Lipsits, D.V. 1965. Die Biochemie der Kartoffel resistenz gegen den Krebs erreger *Synchytrium endobioticum* (Schilb.) Perc. (English summary), pp. 265-281. In Biochemische Probleme der Kranken Pflanze. Symposium Deut. Akad. Landwirt. Berlin. 1964.
29. Lipsits, D.V. 1972. The possibilities of biochemical diagnonis of disease resistance in potato plants. (in Russian, English summary). Sel'skokhozaistvennaya biologiya 7(6): 886-894.
30. Lipsits, D.V. and F.Z. Eisinger. 1958. Respiration of potato plant tissues in connection with resistance of the plant to tumour-producing agents. Soviet Plant Physiol. 5(2): 172-174.
31. Lipsits, D.V., E.K. Kruglyakova, V.M. Chibrikin, and A.B. Dolyagin. 1971. The concentration level of free radicals in the sprouts of various varieties of potatoes differing in their resistance to cancer (in Russian). Akad. Nauk S.S.S.R. Doklady 196(4): 975-976.
32. Lipsits, D.V., K.E. Kruglyakova and A.B. Dolyagin. 1971. Action of ionol and Tween-60 on potato wart. Doklady Biochem. Sec. 198(1-6): 221-223.
33. Miloslavova, T.A., E.K. Reshetova, A.S. Volovik and A.B. Dolyagin. 1973. A method isolating zoosporangia of *Synchytrium endobioticum* (Schilb.) Perc. from potato warts (in Russian). Mikol. i Fitopatol. 7(5): 459-461.
34. Miloslavova, T.A., M.S. Zhedek, A.S. Volovik, K.Ye. Kruglyakova and A.B. Dolyagin. 1975. The effect of wart-inhibiting substances on the susceptibility of potatoes to the fungus (*Synchytrium endobioticum* (Schilb.) Perc. (in Russian). Mikol. i Fitopatol. 9(2): 113-116.
35. Mirzabekian, R.O. 1959. Antibiotic substances of actinomyces origin affecting phytopathogenic micro-organisms (in Russian, English summary). Akad. Nauk S.S.S.R. Izv. Ser. Biol. 24(1): 103-110.
36. Mirzabekian, R.O. 1969. Tissue culture from the wart callus of the potato (in Russian). Mikol. i Fitopatol. 3(4): 337-342.
37. Mirzabekian, R.O., N.V. Sinitsina and O.G. Belyakova. 1961. Elaboration of a biological method for the control of potato wart (in Russian). Agrobiologya 4(130): 566-572.
38. Okanenko, A.S. and B. Bershtein. 1960. Proteins of cancerous growth in the use of potato infected with *Synchytrium endobioticum*. Akad. Nauk S.S.S.R. Doklady 134(3): 180-182.
39. Pashkar', S.I. 1975. On the role of the root system in the growth of wart calluses on potato. Probl. onkol. i teratol. rastenii. Leningrad, U.S.S.R. pp. 403-404.
40. Pidoplichko, N.M. 1959. Control of potato canker in the Ukrainian S.S.R. (in Russian, English summary). Rostlinna Vyroba 32(6): 47-58.
41. Reingard, T.A. and S.I. Pashkar. 1958. Participation of growth substances of the auxin type information of tumours on potato plants. Soviet Plant Physiol. 5: 512-517.
42. Reshetova, E.K., A.S. Volovik and A.B. Dolyagin. 1975. Nucleo-proteinases isolated from the sprouts of both *Synchytrium endobioticum* (Schilb.) Perc. resistant and susceptible potato cultivars (in Russian). Mikol. i Fitopatol. 9(5): 438-440.
43. Saltykova, L.P. 1973. Varietal differences in potato resistance to ordinary and aggressive agents causing potato wart disease (in Russian, English summary). Trudy VN11 Zaschity rastenii. 36, 63-71.
44. Saltykova, L.P. and V.I. Yakovleva. 1976. Soviet and Czechoslovak races of a causal agent of potato cancer, *Synchytrium endobioticum* (Schilb.) Perc. (in Russian). Mikol. i Fitopatol. 10: 503-507.
45. Samersova, V.A., M.I. Danchenko. 1975. Detection of aggressive biotypes of potato wart in Byelorussia. Prob. onkol. i teratol. rastenii. Leningrad, U.S.S.R. pp. 399-401.
46. Sedivy, J. 1975. In memory of Javoslav Zakopal/*Synchytrium endobioticum* Phytopathologist (in Russian). Ochr. Rostl. 11(3): 169-172.
47. Sharikov, K.E. 1975. On canker infection of different representatives of the Solanaceae. Probl. onkol. i teratol. rastenii. Leningrad, U.S.S.R. pp. 398-399.
48. Tarasova, V.P. 1969. Role of the environment in the decay of warts and germination of zoosporangia of *S. endobioticum* on Potato. (in Russian, English summary). Byull. VN11 Zaschity rastenii. 1(13): 42-44.
49. Tarasova, V.P. and V.K. Beskorovainy. 1973. A complex method for controlling potato wart (in Russian). Zaschita rastenii 11: 45.
50. Timchuk, K.S. and D.V. Lipsits. 1970. Histo - and cytochemical studies of inter-relations between the causal organism of potato wart disease *Synchytrium endobioticum* (Schilb.) Perc. and host plant. I. (in Russian). Mikol. i Fitopatol. 4(1): 34-43.
51. Timchuk, K.S. and D.V. Lipsits. 1973. Cyto- and histo-chemical characteristics of the protein-nuclein complex of healthy potatoes and those infected by the cancer-inducing *Synchytrium endobioticum* (Schilb.) Perc. sprouts of varieties of potato contrasting in resistance (in Russian, English summary). Trudy VN11 Zaschity rastenii. 36, 117-122.
52. Trochinskii, N., G. Karytko. Agricultural measures as means for eradication of potato wart. (in Russian). Zasch. rastenii ot Vred. i Boleznei 6: 49-50.
53. Vinkler, G.N., D.V. Lipsits. 1975. Culture of pieces of wart callus detached from potato on nutrient media. Probl. onkol. i teratol. rastenii. Leningrad, U.S.S.R. pp. 401-403.
54. Vladimirskaya, N.N. 1960. Physiological processes in *Synchytrium endobioticum* (Schilb.) Perc. in the dormant zoosporangia state (in Russian). Botan. Zhur. 45(1-6): 97-104.
55. Yakovleva, N.N. 1970. Some morphological peculiarities of aggressive biotypes of potato wart pathogens (in Russian). Mikol. Fitopatol. 4: 267-268.
56. Yakovleva, V.I. 1961. Variability of the potato wart pathogen (in Russian). Vestnik Sel'skokhoziaistvenii Nauki. 12: 72-75.
57. Yakovleva, V.I. 1973. Racial composition of the agent causing potato wart disease in the U.S.S.R. (in Russian, English summary). Trudy VN11 Zaschity rastenii. 36, 78-86.
58. Yakovleva, V.I. 1975. Races of the causal agent of potato cancer and their virulence (in Russian). Mikol. i Fitopatol. 9(5): 421-452.

Further observations on cranberry fungi in Nova Scotia[1]

C. O. Gourley

Sporonema oxycocci, found on the leaves of native and cultivated cranberry and which causes fruit rot, is the principal disease parasite of cranberry in Nova Scotia. Twenty of the fungi identified on cranberry fruit and foliage have not previously been reported on this host in Nova Scotia. The incidence of fungal species from fruit decaying in storage differed from that of diseased berries immediately after harvest from a native bog.

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Sporonema oxycocci qu'on observe sur les feuilles de canneberge sauvage et cultivée dont elle cause la pourriture du fruit, est le principal cryptogame parasite de cette espèce végétale en Nouvelle-Ecosse. Vingt des espèces de champignons identifiées sur les fruits et les feuilles de canneberge n'avaient pas encore été signalées auparavant sur cet hôte en Nouvelle-Ecosse. La mycoflore observée en entrepôt sur des fruits en voie de décomposition diffère de celle qui colonise les fruits malades immédiatement après leur récolte dans une airellière naturelle.

Introduction

Most of the principal decay-producing fungi of cranberry, *Vaccinium macrocarpon* Ait., in North America have been found in Nova Scotia (2, 3, 5, 7). The relative importance of the rot fungi varies with location and with cultural practices (3, 4). Fruit rots are the most important cranberry diseases in Nova Scotia but they seldom occur until after harvest (3). The extent of fungal deterioration of harvested fruit depends on preharvest growing conditions and the inoculum potential in the cranberry plantation. This paper reports on some periodic observations of diseases of cranberry fruit from native and cultivated bogs and the identification of fungi associated with fruit and foliage.

Materials and methods

In November, 1969, cranberry fruit that had been raked from representative areas in a native stand near the sea at Long Point, Inverness County, Nova Scotia, were examined for the amount and cause of fruit decay. The condition of the fruit was determined on 400 berries from the bulked sample by classifying it as either frosted, diseased or healthy. Westerly sea breezes had lessened the chances of freezing injury to the fruit.

Within 2 days the diseased berries were sterilized in 70% (v/v) ethanol and the calyx and 3 sections of the skin and flesh of each berry were placed on potato dextrose agar (PDA) in a Petri plate. The healthy fruit was placed at 9°C for 16 days. At the end of this time the rotted berries were removed and isolations made as before.

One hundred and eight cranberry leaves in the sample with the fruit from the native stand were placed apart on moist filter paper in 15 cm Petri plates for 10 days at room temperature. At the end of this time the incidence of fungal species on the leaves was recorded. The dried buds from 2 blighted inflorescences were also placed on PDA in plates.

In mid December 1975, a sample of stored fruit which had been harvested 2 months previously from a commercial bog at Aylesford, Kings County, was examined for the cause of berry rot. The diseased fruit was removed, surface sterilized in 70% (v/v) ethanol, and sections dissected from the advancing edge of rots were placed on PDA in Petri plates. The species and frequency of fungal isolates were recorded. Pathogenicity tests were conducted with *Penicillium variabile* Sopp. on healthy fruit, surface sterilized as before, by inoculating sound berries and berries that had been artifically wounded with a flamed scalpel.

In early September 1976, cranberry leaves collected from an abandoned bog at Aylesford, Kings Co., that had been out of commercial production for about 5 years, were dipped into 70% (v/v) ethanol, shaken vigorously, flamed and placed on water agar in a Petri plate with the upper surface of the leaf uppermost. The fungi that developed on the leaves and in the agar adjacent to them were identified and recorded.

Results and discussion

Of the 400 berries of late harvested cranberry fruit from the native stand, 34% were classified as frosted, 32% were diseased and 34% were healthy. The amount of decay and the number of fungal species causing rot may have been increased by over-maturity. The infected fruit yielded most of the principal decay producing fungi (Table 1). Three fungi, *Gloeosporium minus* Shear, *Penicillium thomii* Maire and *Phyllosticta putrefaciens* Shear, considered by Shear *et al.* (6) to be of minor importance as initiators of fruit rot, have not heretofore been reported on cranberry in Nova Scotia. Six species

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Table 1. Incidence of fungi isolated from diseased cranberry fruit from a native stand in November, 1969

Organism	Percent of fruit infected	
	At Harvest	After 16 days @ 9°C
Acanthorhynchus vaccinii Shear	0	0.6
**Arthrinium phaeospermum* (Pers.) Grove	0.6	0
**Aureobasidium pullulans* (deBy.) Arn.	8.4	16.2
Ceuthospora lunata Shear	0.6	0
**Chaetomium brevipilium* Ames.	0	1.2
Chaetomium spirale* Zopf. 146885	0	0.6
**Coniothyrium olivaceum* Bon. 142228	0	0.6
**Curvularia clavata* Jain 146884	0	0.6
Diaporthe vaccinii Shear 147609	16.2	1.2
**Gloeosporium minus* Shear 147608	0	1.8
Godronia cassandrae Pk. f. *vaccinii* Groves	1.8	7.5
Guignardia vaccinii Shear	10.8	0
**Myrothecium* sp. 153473	0	0.6
**Penicillium thomii* Maire 145742	12	16.5
Penicillium spp.	18.5	1.8
**Pezizella oenotherae* (Cke. & Ell.) Sacc.	0	1.2
Phyllosticta putrefaciens Shear	0.6	0
Sporonema oxycocci Shear	44.9	23.7
**Thysanophora penicillioides* (Roum.) Kend. 153471	0	0.6
Sterile breakdown	25.2	26.8
Unknown	10.8	10
Number of diseased fruit examined	167	160

* Not previously reported on cranberry in Nova Scotia.
**DAOM accession number, mycological herbarium, Biosystematics Research Institute, Ottawa.

of fungi, *Aureobasidium pullulans* (deBy.) Arn., *Chaetomium brevipilium* Ames, *Chaetomium spirale* Zopf., *Curvularia clavata* Jain, *Myrothecium* sp. and *Thysanophora penicillioides* (Roum.) Kend. are not considered to be pathogenic. The fungi *Cytospora delicatula* Shear, *Discosia artocreas* Tode ex Fr. and *Strasseria oxycocci*

Table 2. Frequency of fungi from diseased fruit in storage from a commercial bog in December, 1975

Organism	Percent of Fruit
Botrytis cinerea Pers.	8
Diaporthe vaccinii Shear	21
Godronia cassandrae Pk. F. *vaccinii* Groves	0.4
Penicillium thomii Maire	0.9
Penicillium variabile* Sopp. 200607; 155527***	24
Penicillium sp.	2
Pestalotia sydowiana Bres.	0.4
Sporonema oxycocci Shear	51
Sterile	0.9
Number of fruit examined	228

*Not previously reported on cranberry in Nova Scotia.
**I.M.I. accession number, Commonwealth Mycological Institute, England.
***DAOM accession number, mycological herbarium, Biosystematics Research Institute, Ottawa.

Shear which normally occur on foliage or stems (6) appeared in culture plates and may have been associated with the calyxes rather than the skin or flesh of the fruit. These three fungi have not previously been reported on Vaccinium in this province.

The frequency of some fungi isolated from diseased berries immediately after the fruit was gathered and from refrigerated fruit differed (Table 1). The 9°C storage temperature may have enhanced the growth of some fungi more than others.

Zuckerman (8) noted the increasing importance of *Sporonema oxycocci* Shear in Massachusetts. In Nova Scotia it was the most commonly isolated fungus from damaged fruit and it occurred on the calyxes of 30 berries with sterile breakdown. On 5 occasions, *P. thomii* was the only organism isolated from rotted fruit, but usually it was associated with more aggressive pathogens. Other *Penicillium* spp. were not considered to be the primary cause of fruit rot. *Acanthorhynchus vaccinii* Shear, *Guignardia vaccinii* Shear and *Stigmatea conferta* (Fr.) Fr. (= *Gibbera compacta* (Pk.) Shear) have been shown to be associated with speckle or blotch of cranberry fruit (1, 3, 5). The 70% (v/v) ethanol surface sterilization of the fruit may have prevented the isolation of *S. conferta*. Recently it was shown that this fungus can be isolated from fruit following surface sterilization with a weak, 0.5% (v/v), Cl solution (5).

Only five species of fungi were found on the 108 cranberry leaves examined from the native stand. *Sporonema oxycocci* occurred on 91 leaves, *G. vaccinii* on 22, *Strasseria oxycocci* on 1, *D. artocreas* on 1, unknown on 1, and on 21 leaves, no fungus was found.

The dried buds from one inflorescence yielded only *Colletotrichum dematium* (Pers. ex Fr.) Grove, not previously reported here on this host, and from the other, only *Sporonema oxycocci*.

Table 3. Fungi on and from cranberry leaves following 70% (v/v) ethanol treatment in September, 1976

Acanthorhynchus vaccinii Shear
**Alternaria alternata* (Fr.) Keissler
Arthrinium* state of *Apiospora montagnei* Sacc. 161168
Aureobasidium pullulans (deBy.) Arn.
**Cochliobolus sativus* (Ito & Kurib. in Kurib.) Drechsler ex Dastur 160706
Diaporthe vaccinii Shear
Godronia cassandrae Pk. f. *vaccinii* Groves
Mucor sp.
**Papulospora anomala* Hotson
Pestalotia truncata Lev.
Sporonema oxycocci ;Shear
Stigmatea conferta (Fr.) Fr. 165742
An unknown ascomycete

*Not previously reported on cranberry in Nova Scotia.
**DAOM accession number, mycological herbarium, Biosystematics Research Institute, Ottawa.

Stored, decaying fruit from the commercial bog yielded the ripe rot fungus *Sporonema oxycocci* in more than 50% of the isolations (Table 2). *P. variabile*, although obtained from nearly 25% of the diseased fruit, did not produce rot in sound fruit or artifically wounded fruit and it did not colonize autoclaved cranberry fruit in Petri plates. It was often the only fungus isolated from decaying fruit from storage and because of its inability to utilize cranberries as a food source it may have existed as a mycoparasite rather than a symbiont.

Perithecia from natural infections of a *Stigmatea* sp. occurred mostly on the upper surface of more than 50% of the ethanol treated leaves from the abandoned commercial bog. Perithecia also formed on the undersides of some leaves and occasionally on the water agar adjacent to the leaves. The fungus was identified as *S. conferta*. It occurs naturally on the lower surface of cranberry leaves without causing any apparent damage (6). It is not known why perithecia form mostly on the undersides of leaves in nature but it may be a photophobic reaction. Several other fungi not normally found on this host appeared on the leaves or on the agar and none of them were recorded from more than 2 or 3 leaves (Table 3).

Sporonema oxycocci was the principal cause of cranberry fruit rot and appeared most frequently on the leaves of native and cultivated plants. The host range of twenty other fungi is extended to cranberry in Nova Scotia.

Acknowledgement

Some of these observations are a completion of the work begun by Dr. K. A. Harrison, prior to his retirement, and R. W. Delbridge, Nova Scotia Department of Agriculture and Marketing. The assistance of R. A. Murray, Nova Scotia Department of Agriculture and Marketing is greatly appreciated. Dr. Donald Boone, University of Wisconsin, suggested the technique for producing perithecia of *Stigmatea* sp., and Dr. M. P. Corlett, mycologist, Ottawa, identified the fungus as *S. conferta* (Fr.) Fr.

Literature cited

1. Carlson, L. W., and D. M. Boone. 1966. A berry speckle disease of cranberry and its control. Plant Dis. Reptr. 50: 539-543.
2. Conners, I. L. 1967. An annotated index of plant diseases in Canada. Can. Dep. Agr. Pub. 1251.
3. Gourley, C. O., and K. A. Harrison. 1969. Observation on cranberry fruit rots in Nova Scotia, 1945-55. Can. Plant Dis. Surv. 49: 22-26.
4. Hall, I. V., L. R. Townsend, C. L. Lockhart, K. A. Harrison, G. W. Wood, and G. T. Morgan. 1969. Growing cranberries. Can. Dep. Agr. Pub. 1282 Rev.
5. Lockhart, C. L. 1970. Isolation of *Gibbera compacta* from cranberry and the effect of moisture and temperature on ascospore development. Can. Plant Dis. Surv. 50: 108.
6. Shear, C. L., N. E. Stevens, and H. F. Bain. 1931. Fungus diseases of the cultivated cranberry. U. S. Dep. Agr. Tech. Bul. No. 258.
7. Wehmeyer, L. E. 1950. The fungi of New Brunswick, Nova Scotia and Prince Edward Island. National Research Council of Canada Pub. No. 1890.
8. Zukerman, B. M. 1958. Relative importance of cranberry rot fungi during the storage and harvest seasons in Massachusetts, 1956-57. Plant Dis. Reptr. 42: 1214-1221.

Verticillium dahliae from stunted plants of summer savory[1]

C.O. Gourley

Nine fungi including *Verticillium dahliae* were isolated from stunted summer savory (*Satureja hortensis*) plants from Tancook Island.

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Neuf champignons, dont *Verticillium dahliae*, ont été isolés de plants rabougris de sarriette des jardins (*Satureja hortensis*) cultivés à Tancook Island.

About 1 ha of summer savory, *Satureja hortensis* L., is grown commercially on Tancook Island, Lunenburg Co., Nova Scotia. It is usually grown on the same land for several years in plots ca 1000 m^2. Recently growers have complained that in the third year of monoculture, the plants are generally stunted, in most cases less than half the normal plant height of 30 to 45 cm, with few plant losses.

In August, 1977, stunted plants were collected from a plot in which this crop had been grown for the third successive year. Isolations were made from the crown and stem sections of 13 plants, surface sterilized in 2% Cl, and planted onto potato dextrose agar in Petri plates.

The fungi and their frequency from diseased plants were as follows:

Organism	Frequency (%)
Alternaria sp.	31
Cephalosporium sp.	8
Colletotrichum coccodes (Wallr.) Hughes	8
Gibberella cyanogena (Desm.) Sacc. stat conid., *Fusarium sulphureum* Schlecht. (DAOM 166631)[1]	46
Mortierella ramanniana (Moller) Linn. var. *ramanniana* (DAOM 166508)	8
Pyrenochaeta sp.	92
Pythium oligandrum Drechsler (DAOM 166163)	23
Ulocladium atrum Preuss (DAOM 166660)	8
Verticillium dahliae Klebahn (DAOM 166742)	62

[1]*Accession number Mycological Herbarium, Ottawa.*

The fungus *F. sulphureum* is best known as the cause of a storage rot of potatoes (1). It occurs frequently in soil and has been spasmodically isolated from a wide range of herbacious plants, usually being a weak parasite or a saprophyte. *Pyrenochaeta* sp. was most frequently isolated but was always associated with *Fusarium* or *Verticillium* except in one plant where the association was with bacteria. *P. oligandrum* causes damping off, stem and root rot of a wide range of plants under a continuous cultural program (4). In North America it has been reported in the United States only, not in Canada. The reason for this is not known but it may be because its minimum and optimum temperature requirements are 8-9°C and 31°C, respectively. There is no previous report of it on summer savory.

V. dahliae commonly causes a disease after repeated croppings with susceptible plant species (3). Here the frequency of isolation indicated that *V. dahliae* was probably the primary cause of stunted plants of summer savory on Tancook Island. *F. sulphureum* was also frequently isolated but is commonly recognized as a weak parasite or saprophyte.

Fungi on summer savory have been mainly reported from the seed (2). For this reason specimens of the principle and some of the more uncommon fungi have been deposited in DAOM. As far as the author is aware *V. dahliae* has not previously been reported from this host. Subsequent tests are needed to verify the pathogenicity of this fungus on summer savory.

[1] *Contribution No. 1646, Research Station, Agriculture Canada, Kentville, Nova Scotia.*

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Literature cited

1. Booth, C. 1971. The genus fusarium, C.M.I. Kew, Surrey, Eng. pp. 183-185.
2. Conners, I.L. 1967. An annotated index of plant diseases in Canada. Can. Dept. Agr. Pub. 1251, p. 259.
3. Hawksworth, D. L. and P. W. Talboys. 1970. *Verticillium dahliae*. C. M. I. Descriptions of Pathogenic Fungi and Bacteria, No. 256.
4. Waterhouse, G. M. and J. M. Waterson. 1966. *Pythium oligandrum*, C. M. I. Descriptions of Pathogenic Fungi and Bacteria, No. 119.

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