Five of the foundation plots were infected by bacterial blight (one of 'Sanilac' and four of 'Seaway,). Within the five plots, considerable variation in the amount of infection was evident. Three plots showed at least 10 areas of infection, and infection in two of the plots was the result of secondary infection from diseased beans inadjacent fields. Two plots showed only a trace of infection with one and two foci of infection, respectively. All infections in foundation plots were of common blight except one plot of 'Seaway' which was infected with fuscous blight resulting from secondary infection

All 23 registered and commercial fields inspected were infected with common blight or fuscous blight or both to varying degrees. Registered fields, the progeny of last year's foundation plots, were generally slightly infected, while infection in commercial fields ranged from 5% to 100%. Twelve commercial fields were infected with fuscous blight. No registered fields were infected with fuscous blight. Twenty-one of twenty-three commercial and registered fields were infected with common blight,

In foundation plots in 1967 and again this year, blight infection originating from infected seed was entirely caused by X. phaseoli. In the 1968 registered crop, arising from 1967 foundation seed, only X. phaseoli was isolated from diseased plant material. However in commercial crops, the seed of which had been propagated for a number of years in Canada, X. phaseoli var. fuscans was prevalent. From 1962 to 1964 fuscous blight was the principal bean pathogen in Ontario (1), and it is still present in the older Ontario seed stocks. There has been no indication that fuscous blight is present in the breeder seed that is imported from Idaho and California to produce the foundation plots.

Sclerotinia rot caused by Sclerotinia sclerotiorum (Lib.) de Bary was found in 10 of the foundation plots and ranged from trace to slight in intensity. It was also present in most of the registered and commercial fields. The disease was much less severe than in 1967 (2) and little damage is expected.

Root rot, caused primarily by Fusarium solani (Mart.) App. and Wr. f. phaseoli (Burkh.) Snyd. & Hansen, was prevalent in eight fields or plots, ranging from a trace to severe in intensity. Most affected fields showed small infected areas of less than 1% of the crop: however in one field more than 50% of the plants were severely infected.

Rust caused by Uromyces phaseoli (Rebent.) Wint. was present in seven fields and ranged from a trace to severe on individual plants. Localized areas of infection in three fields caused early maturity of the crop. Infection was the most severe since field bean surveys were initiated in 1961.

Literature cited

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NEMATODE LEAF BLIGHT OF CHRYSANTHEMUM[1]

J.A. Hoes and H.F. Harp[2]

An epidemic of the leaf nematode Aphelenchoides ritzemabosi (Schwartz) Steiner and Buhrer occurred in 1968 at Morden in observation plots of hardy varieties of Chrysanthemum morifolium (Ramat.) Hemsl., the florists' chrysanthemum. Initial symptoms consisted of brown to black wedge-shaped interveinal leaf areas: later the entire leaf turned brown and necrotic. Severely diseased plants looked withered, all leaves being dead and adhering to the stem. Numerous nematodes were present in partially affected leaves. The nematodes were identified by Dr. K. C. Sanwal, Entomology Research Institute, C. D. A., Ottawa, Ontario. Total precipitation during the growing season was almost twice the long-term average of 12.8 inches. The excessive rainfall no doubt contributed to the severe infection (2).

The varieties were planted in rows or blocks in 1966 and plants touched each other affording easy spread to the pathogen. All plants of a given variety were either severely infected or were completely or nearly symptomless. The apparent uniformity of the infestation suggested that observed differences were real and that classification into resistant and susceptible varieties was justified. Highly susceptible varieties were 'Archibald', 'Beckethau', 'Cameo', 'Candy', 'Cartier', 'MacDonald', 'Pelican', 'Skyline', 'Tilley', and Morden No.'s 6607, 6613 and 6617. Suggested to be resistant are 'Brightness', 'Brown', 'Canary', 'Galt', 'Howe', 'Paige's Gold', 'Sutherland', 'Tupper', 'Whelan', and Morden No.'s 6408, 6608, 6614 and 6618. Hesling and Wallace (1) demonstrated that chrysanthemum varieties differ greatly in their susceptibility to A. ritzemabosi. None of their varieties, however, are among the varieties mentioned in this paper.

1 Contribution No. 78, Research Station, Canada Department of Agriculture, Morden, Manitoba.

2 Plant Pathologist and Horticulturist, respectively.

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CHIVES RUST AT OTTAWA, ONTARIO[1]

D.B.O. Savile

Rust, caused by Puccinia mixta Fckl., has long been known on chives (Allium schoenoprasum L.) from the vicinity of Victoria and Vancouver, B. C., most of the available records having been listed by Savile *(2)*. This rust, which has often been confused with Puccinia porri (Sow.) Wint. and other species (2), is practically confined to chives; but onion (Allium cepa L.) is occasionally attacked when grown close to infected chives. It was recorded by Arthur (1) from Washington, on the Pacific coast, and from Connecticut and New York, on the Atlantic coast. There is also a specimen in DAOM from Woods Hole, Massachusetts. The rust has apparently not previously been recorded in eastern Canada or far inland.

On 14 June 1969 a heavily rusted clump of chives (Savile 5032) was found in a garden at Qualicum on the outskirts of Ottawa. Uredinia were abundant and telia already common. The intensity of infection indicated that the clump must have been infected in 1968. However, there was no infection in a clump in the adjoining garden, from which the rusted clump was derived in 1967. On 10 July 1969 Dr. R.V. Clark discovered a lightly infected clump, with uredinia predominant, in a garden at Meadowlands, about 3 miles from Qualicum.

All North American specimens examined fall within the range of variation seen in European specimens: but different outbreaks seem to stem from separate introductions. In the British Columbia specimens 2-celled teliospores range to 43 µ long, and 1-celled spores are common. In the Ottawa specimens 2-celled teliospores range to 53 µ long, and 1-celled spores are few. In the Massachusetts specimen (W. R. Taylor, July 1947, DAOM ex herb. Wehmeyer) 2-celled spores range to 50 µ long and 1-celled spores are abundant. Thus we have at least three biotypes of this rust in North America, apparently separately introduced with seeds or bulbs, The plant is ordinarily sold as seed, to which detached teliospores may possibly adhere; but. it is probable that bulbs, with sori on the scales, are occasionally brought in clandestinely in settlers' effects.

Because chives is grown predominantly in home gardens the extent of the outbreak is not easily assessed. This preliminary note is presented to encourage readers to check all plantings that they see.

Control of the rust may prove difficult, since the summer use of fungicides would defeat the purpose of growing the plant. However it may be practicable to use a fungicide very early, in the period of teliospore germination. It is probably advisable, where chives is grown in a flower bed that is sprinkled regularly, to cover the plants with plastic during sprinkling, and apply water gently to tho soil, It is proposed to dig the infected plants of Qualicum in the fall, clean them off thoroughly, and replant them in shady sites where they will require less watering. A few plants will be left untouched until spring and watched for development of aecia, which are not often reported.

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[1] Contribution No. 738, Plant Research Institute, Canada Department of Agriculture, Ottawa, Ontario.

POTATO LATE BLIGHT IN CANADA IN 1844–45

P.M. Austin **Bourke**[1]

The following paragraph is quoted from a study (1) of the historic epidemic of potato blight (Phytophthora infestans (Mont.) de Bary) which broke out in Europe in 1845:

> "Although the attack took Europe completely by surprise, blight had already been ravaging the potato crops of North America in the previous two seasons. Stevens (2) has charted the annual progress of the disease from its beginnings in 1843 in the five States closest to the great ports of the east coast of the United States, to an expanded area in 1845 which closely approximates to the limits within which blight is a serious

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