BRIEF ARTICLES

SUNFLOWER DISEASES IN MANITOBA IN 1968

J.A. Hoes'

<u>Sclerotina sclerotiorum</u> (Lib.) de Bary and <u>Verticillium dahlae</u> Kleb. were the most important pathogens of sunflower (<u>Helianthus annuus</u> L.) in Manitoba in 1968. The former caused both head rot and stalk rot but was especially prominent in the head rot phase (Table 1). Most heads affected by <u>Sclerotiorum</u> were a complete loss and infection must have taken place early and been followed by rapid disease development. Usually the head rot

 Table 1. Frequency of head and stalk rot phases of Sclerotina disease in 31 sunflower fields

			th affected	•
Phase	0	1-5	6-10	11-40
Head rot	2	26	1	2
Stalk rot	16	14	1	0

phase of S. sclerotiorum is scarce or absent while the stalk rot phase is more prominent. Presumably the young plants emerging in June escaped infection because this month was relatively dry. Precipitation in July and August, however, was more than twice the long term average amount of 5.3 inches for the two months, while the respective mean temperatures were 2.3F and 5.7F below normal. The prolonged cool and wet weather favored the continued production and discharge of ascospores (3), which infected the young heads, but apparently not the stalks of the plants. Sackston (2) attributed the relative abundance of head rot in 1951, at least in part, to low temperatures and frequent rains during August and September. Head rot was also due to Botrytis cinerea Pers. but damage here was less conspicuous; it was not found in 12 fields, and in the 19 other fields it affected only 1-5% of the plants. Diseased heads were only partially invaded, and environmental conditions had apparently been less favorable to Botrytis than to Sclerotinia.

<u>Verticillium dahliae</u> was as prominent as in other years even though the season was abnormally wet and cool. Of 31 fields, 10 showed not more than 1% infection, 15 fields had 5-20% infected plants, and three fields of 'Commander' had 50-75% diseased

1 Plant Pathologist, Research Station, Canada Department of Agriculture, Morden, Manitoba. plants. The variety 'Peredovik' was generally more resistant than 'Commander', confirming previous observations (1). Downy mildew caused by Plasmopora halstedii (Farl.) Berl. & de Toni was found in only 2/31 fields, causing a trace of infection. Leafspot caused by Septoria helianthi Ell. and Kell. and rust caused by Puccinia helianthi Schw. occurred in all fields but infections were light and no damage was caused. The variety "Peredovik' showed generally a much lower density of rust pustules than 'Commander' even though in greenhouse studies both are equally susceptible in the seedling stage; 'Peredovik' displays a certain amount of adult plant resistance. Light frost caused serious damage in early planted fields in the Carberry area. Top leaves turned brown and young affected heads showed brown discoloration just below the point of insertion of the florets. Affected heads did not develop.

Literature cited

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FIELD BEAN DISEASE SURVEY IN ONTARIO-1968

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In 1968, 13 foundation plots of 'Seaway' and 8 foundation plots of 'Sanilac' field beans were inspected for bacterial blights and other diseases. The various plots ranged between $\frac{1}{2}$ and 2 acres and were located in western Ontario primarily near Hensall, Kippen, and Blenheim. In addition, 23 fields of first-generation and commercial beans were also inspected.

Infected plant tissues, primarily leaves and pods, were collected from infected fields and examined in the laboratory. Common blight caused by <u>Xanthomonas phaseoli</u> (E. F. Sm.) Dowson and fuscous blight caused by <u>Xanthomonas phaseoli</u> var. <u>fuscans</u> (Burkh.) Starr and Burkh. were differentiated by the formation of a brown diffusible pigment by cultures of the fuscous blight organism on nutrient agar.

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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 mate rial. 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 <u>Sclerotinia sclerotio-</u> rum (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 <u>Fusarium solani</u> (Mart.) App. and Wr. <u>f. phaseoli</u>(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 <u>Uromyces phaseoli</u> (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. Infectionwas the most severe since field bean surveys were initiated in 1961.

Literature cited

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NEMATODE LEAF BLIGHT OF CHRYSANTHEMUM'

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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. Initialsymptoms 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.

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