Anthracnose (*Colletotrichum lindemuthianum*) on White Bean (*Phaseolus vulgaris* L.) in Southern Ontario: Spread of the Disease from an Infection Focus

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ABSTRACT

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Spread of anthracnose (*Colletotrichum lindemuthianum*, delta race) from an initial infection focus was studied in 1978 and 1979 in field plots planted with a susceptible white bean cultivar (*Phaseolus vulgaris* 'Fleetwood'). Splashing rain spread the disease for short distances, although some plants adjacent to diseased plants did not become infected. Long-distance spread, 3–4.6 m per rainstorm, was caused by splashing raindrops blown by gusting winds. Disease spread from the infection focus toward the northeastern quarter of plot followed the direction of prevailing winds. Disease severity was highest at or near the axis of the sector nearest the initial infection focus and decreased gradually toward the periphery of the sector. The fungus required about 10 mm of rain to establish infection. In southern Ontario, temperature was not a limiting factor for infection and disease spread, but temperatures above 25 C appeared to restrict disease development.

In Canada, white bean (*Phaseolus vulgaris* L.) production is concentrated in southern Ontario. Approximately 58,000 ha of dry beans are grown yearly. In 1975, an outbreak of anthracnose (13) was caused by the delta race of *Colletotrichum lindemuthianum* (Sacc. & Magn.) Broisi & Cav. This race is new to Canada (10,11), and the common bean cultivars Fleetwood, Kentwood, Sanilac, and Seafarer are susceptible to it. The disease spread rapidly, and in 1977, about 650 of 3,540 ha of select, foundation, and certified seed plots of all cultivars in Ontario were infected with the delta anthracnose race (G. Fuller, Agriculture Canada, *personal communication*).

The interim control measures include treating seeds with benomyl and inspecting selected plots of pedigreed seed growers. Although these measures brought the disease under control, it has not been completely eradicated; some fields have continued to experience anthracnose each year (*unpublished*). Some infected plants may have been overlooked during the inspection, and approximately 1% of seedlings germinated from anthracnose-infected seeds that were treated with benomyl developed anthracnose (V. Wallen, *personal communication*). Furthermore, the fungus easily develops tolerance to benomyl (10–12). Therefore, a few plants infected with anthracnose would be expected to occur in the field until resistant cultivars are available.

Pertinent information on the epidemiology of this disease in southern Ontario is lacking. The sudden occurrence of the latest epidemic awaits explanation.

This article reports how the disease spreads from small infection foci under the weather conditions that prevail in this region.

MATERIALS AND METHODS

A widely accepted commercial white bean cultivar (Fleetwood), which is susceptible to the race delta of *C. lindemuthianum*, was used in these experiments. All seeds used in this experiment were free of anthracnose.

Field plots were established at the Harrow Research Station, and plantings were made on 13 June 1978 and 30 May 1979. The plots were 25 × 30 m, and a total of 26 rows were planted, east to west with a row spacing of 68 cm. Five seeds were sown in a 30-cm row. The plot was surrounded with four guard rows of snap bean (*P. vulgaris* L. 'Improved Tendergreen'), also susceptible to the delta race of *C. lindemuthianum*.

Inoculations were made on 23 June 1978 and 28 June 1979 on plants at the second and third trifoliate stages, respectively. The dates were chosen to precede forecasted rain. A spore suspension was sprayed onto 24 adjacent plants at the center of the plot.

The spore suspension was prepared by adding 10 ml of sterile water to a 3-wk-old colony of the fungus grown on a Mathur's agar plate (2) and scraping the surface of the colony to dislodge the spores. The suspension was adjusted to 10^7 spores per milliliter of water, a concentration that produces maximal infection (9).

After inoculation, the field plot was examined weekly for signs of anthracnose infection until the bean plants approached maturity, about mid-August. Attention

Table 1. Spread of anthracnose in 1979 expressed as disease severity at four radial distances from the site of inoculation

Date of observation	Disease severity[a]			
	Arc 1	Arc 2	Arc 3	Arc 4
July 9	5.00 ± 0.10	...	...	...
July 12	5.80 ± 0.04	2.20 ± 0.14	1.00 ± 0.05	...
July 20	5.20 ± 0.90	2.40 ± 0.16	1.60 ± 0.06	1.00 ± 0.00
July 27	4.40 ± 0.06	2.00 ± 0.04	1.80 ± 0.12	1.20 ± 0.04
August 2	4.20 ± 0.14	2.60 ± 0.06	1.80 ± 0.12	1.20 ± 0.04
August 10	5.40 ± 0.11	3.20 ± 0.14	2.00 ± 0.20	1.40 ± 0.06

[a]Expressed on a 0–10 scale (0 = no infection, 1 = 10% or less, 2 = 11–20%, 3 = 21–30% . . . 10 = 91–100% of plants showing disease). Arcs 1, 2, 3, and 4 are 3, 5, 8, and 11 m from the center of the inoculated area, respectively. Each value is the average of five plants.

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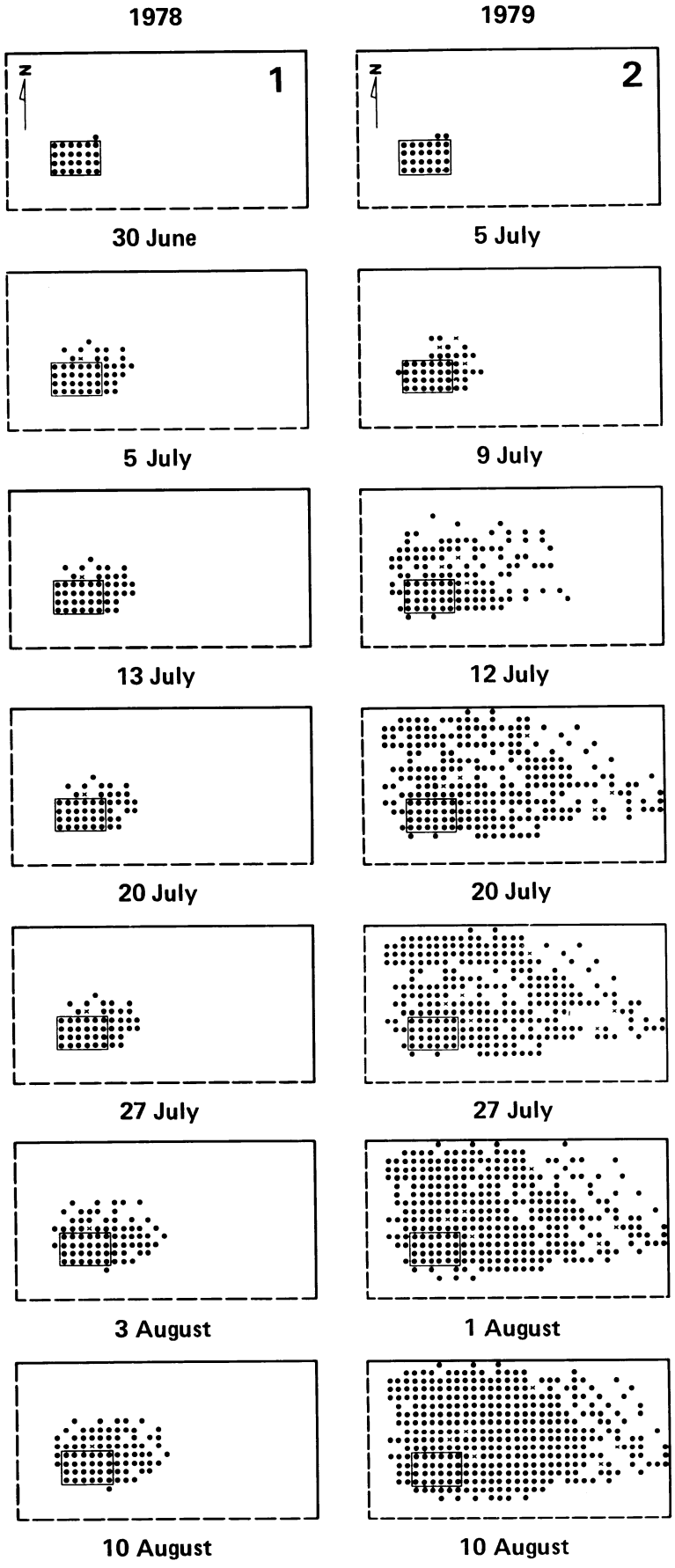


Figs. 1 and 2. Weekly records of disease distribution in the field during the 1978 (Fig. 1) and 1979 (Fig. 2) growing seasons. Large rectangles represent the northeastern quarter of the plot. The small inner rectangles indicate the location of the experimentally infected plants. The date of examination of the plot is below each diagram. • = infected plants; x = dead plants.

was given especially to the pattern and rate of the disease spread. In addition, the infected plants in the inoculated area and in several selected points in the field were monitored weekly for changes in disease severity.

The method described by Lim (6) was adopted to select specific plants for determination of the disease severity. Five radiating lines of equal angular spacing were drawn from the center of the inoculated area toward the edge of the northeastern quarter of the field, and four concentric arcs with different radii (3, 5, 8, and 11 m, designated as arc 1, 2, 3, and 4, respectively) were superimposed in the same quarter. Thus, each arc intersected with each of the five lines to produce 20 cross-points. One plant at each cross-point was selected for the disease severity rating.

Data on temperature, wind direction, and precipitation were obtained from a weather station in the research station. Prevailing winds were southerly or southwesterly.

In 1979 after the plants reached full maturity, all diseased plants were pulled, dried, and thrashed. The seeds were then run through a Celectric bean-sorting machine (Devices Instruments Ltd., Welwyn Garden, Hertfordshire, U.K.) four times to sort out seeds with anthracnose lesions. Infected seeds were weighed and their total number estimated by a predetermined conversion factor of 18.3 g/100 seeds for Fleetwood cultivar.

RESULTS

The direction of the spread of anthracnose from the initial infection focus in the bean field during 1978 and 1979 followed the direction of the prevailing wind (Figs. 1 and 2). At Harrow, the wind blows from the south and southwest during the summer months. Consequently, the disease spread into the northeastern quarter of the plot.

Considerably more plants were affected in 1979 than in 1978. The increased amount of precipitation in 1979 probably favored spread of the disease. Total monthly rainfalls of June, July, and August were 72.1, 25.6, and 24.0 mm in 1978 and were 75.7, 44.1, and 77.2 mm in 1979, respectively. Daily temperature and rainfall data for 1978 and 1979 are shown in Figures 3 and 4, respectively.

The pattern of disease spread closely followed the pattern of major rainfall (10 mm or more). Anthracnose appeared on newly infected plants 3–7 days after each major rainfall, depending on temperature (Figs. 1–4). This amount of precipitation rainfall, depending on temperature (Figs. 1–4). This amount of precipitation appeared to provide the 10-hr wet period necessary for the anthracnose fungus to establish its infection (9). Comparison of the profile of rainfall (Fig. 3) and the incidence of the disease (Fig. 1) in 1978 shows that the rains on 25 and 26 June

helped establish the infection but did not cause the disease to spread because the infected plants had not sporulated. The rains on 1 and 2 July and on 28 July contributed to the spread of the disease observed on 5 July and on 3 August, respectively. Lack of precipitation restricted disease spread between 5 and 27 July.

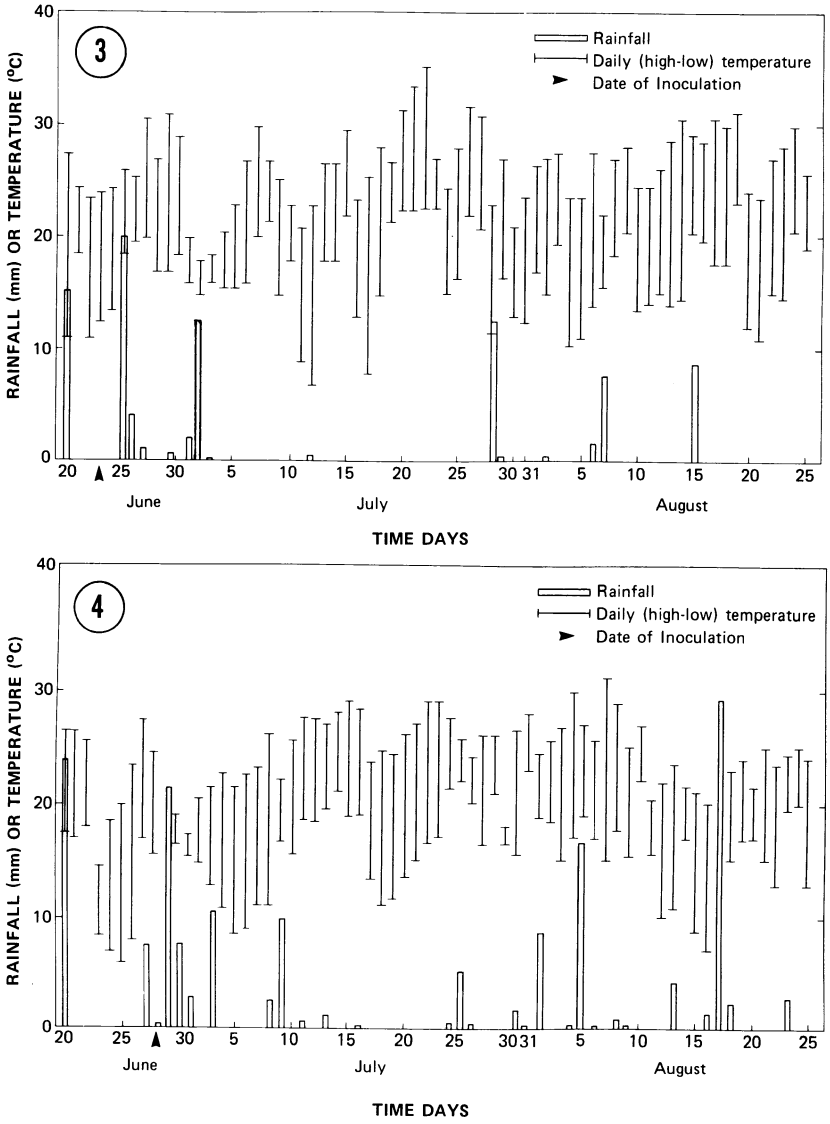
Similarly, in 1979 (Figs. 2 and 4), the rain on 29 and 30 June and 1 July contributed to the infection but not to disease spread because the inoculated plants were at the incubation stage, and the rains on 3,9, and 25 July and 5 August accounted for the spread of the disease recorded on 9 and 12 July and 1 and 10 August, respectively.

The disease spread pattern was observed daily for 10 days starting 3 days after each rainfall. After a rain, plants within a diameter of 1.5 m of diseased plants became infected. Occasionally, a few plants adjacent to diseased ones remained uninfected, suggesting that the splashing raindrops could have missed them (Figs. 1 and 2), but these plants usually became infected after the next rainfall. Long-distance spread was associated with rainstorms in which gusting winds blew splashing raindrops 3–4.6 m away from infected plants. This type of spread was noted between 7, 12, and 20 July 1979 (Fig. 2).

The gradient of the disease in the 1979 field is summarized in Table 1. The disease radiated from the inoculated center toward the northeastern edge of the plot. Disease severity was highest for the plants at arc 1 and decreased toward the edge of the plot.

Field temperature appeared not to be a decisive factor for the infection and spread of bean anthracnose. With sufficient precipitation, the disease spread readily despite high daytime temperatures (25–35 C) between mid-July and mid-August. However, a prolonged period of high daytime temperatures between 25 and 35 C did restrict disease development in the infected plants (Fig. 5) in mid-July to mid-August.

Disease severity increased rapidly in infected plants in the inoculated area beginning at the time of inoculation and worsening steadily for about a week and a half (Fig. 5). Thereafter, disease severity decreased throughout July. The starting date for such a decline varied by approximately a week between 1978 and 1979, probably because of differences in planting dates and temperature profiles. The decrease appears to have resulted from high daytime temperatures that restricted disease progress and stimulated rapid plant growth. Rugosity and epinasty also developed in the leaves with vein infection because the growth of veins was restricted but growth of interveinal tissues continued. Beginning in August, the disease regained its momentum as bean plants approached maturity (Fig. 5). The increase in the disease severity was particularly noticeable in infected pods and leaves.



Figs. 3 and 4. Daily record of temperature and precipitation in the growing seasons of 1978 (Fig. 3) and 1979 (Fig. 4).

Vertical spread of the disease from the infected lower leaves to the new growth was achieved by splashing raindrops, because the new growth remained uninfected so long as the dryness prevailed.

Despite the fact that the 1979 growing season was favorable for anthracnose, few diseased bean plants succumbed to the disease. Instead, they grew to maturity and formed pods and seeds with severe anthracnose lesions.

All plants with anthracnose in the 1979 experiment were harvested for seed. The bean-sorting machine sorted out 1,279 g of infected seeds or about 7,000 seeds that resulted from the spread of the disease from the 24 original inoculated plants. On this basis, an average of 300 infected seeds could be recovered from diseased plants that had contracted the disease from each infected seedling in one growing season, given conditions as favorable as those in 1979. If the 24 infected plants had been scattered separately through the plot, more than 7,000 infected seeds probably would have resulted. This demonstrates that seedborne anthracnose is responsible for primary spread of this disease.

DISCUSSION

This study demonstrates that periods of heavy rain are responsible for the spread of *C. lindemuthianum* in the field.

Rain promotes release of many types of fungal spores (4) and is particularly important in the release of anthracnose spores because they are imbedded in a gelatinous substance on the acervuli. Rain has been reported to play a major role in the spread of stem anthracnose (*C. truncatum*) of lima beans (1).

High field temperatures do not seem to affect the spread of the disease even though prolonged high temperatures reduce disease severity (Fig. 5).

C. lindemuthianum is temperature-sensitive. Its growth is checked at 30–31 C

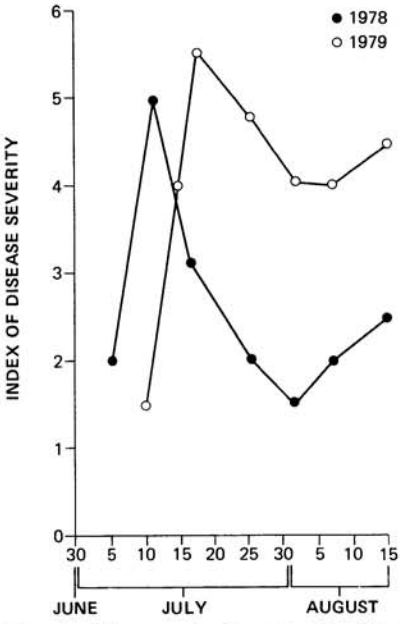


Fig. 5. Changes in disease severity in experimentally infected plants within the infection foci. (Disease severity was measured on a 0–10 scale: 0 = no infection, 1 = 10% or less, 2 = 11–20%, 10 = 91–100% of the plant showing symptoms of disease.)

(3). The temperature limit for infection has been reported to be 26.6 (5), 30 (14), and 32 C (7). Rahe and Kuc (8) showed that the number and size of lesions decreased with increasing incubation temperatures ranging from 28 to 32 C, and no lesions developed on plants kept at temperatures above 32 C.

In southern Ontario in July and August, field temperature frequently reached 25–35 C, yet the disease continued to spread. The low night temperatures may have moderated the effect of high daytime temperatures to provide environmental conditions conducive for infection and disease development; the role of low night temperatures on the epidemiology of bean anthracnose is largely unknown. The discrepancies mentioned for the temperature sensitivity of this fungus may be related to the present observations and warrants further study. To this end, the role of diurnal variations in temperature on the infection and disease development of bean anthracnose is currently under investigation.

The spread of bean anthracnose from an infection focus appeared to be limited to the travel distance of splashing raindrops. In the 1979 growing season, the farthest spread was approximately 10.5 m from the infection focus. For every seedling infected at the beginning of the season, an average of 300 infected seeds were produced at the end of the season. In this context, field spread alone could not account for the sudden outbreak of an epidemic such as that in southern Ontario in 1976-1977. It is reasonable to assume, therefore, that an epidemic of that magnitude might have originated from infected seeds in stock that was distributed to growers.

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