Disease Incidence and Infection Rates of Cercospora apii in Plant Spacing Plots

R. D. Berger

Associate Professor (Associate Plant Pathologist), University of Florida, Institute of Food and Agricultural Sciences, Agricultural Research and Education Center, Belle Glade 33430.

Florida Agricultural Experiment Stations, Journal Series Paper No. 5489. Accepted for publication 22 November 1974.

ABSTRACT

Cercospora apii blight spread faster in celery in closeplant-spacing plots than in wide-plant-spacing plots, resulting in twice as much disease in 8 weeks. Infection rates calculated from weekly disease estimates were useful to interpret treatment differences and effects of weather on disease. The lower incidence of disease in wide-spacing plots was assumed to be from modified microclimatological effects, rather than from diminished numbers of spore arrivals due to possible gradient effects. This was because of the readily wind-disseminated nature of the C. apii spores and the relatively small distance between plants.

Phytopathology 65:485-487

Additional key word: epidemiology.

The plant foliage microclimate exerts a most important influence on infection by leaf spotting fungi (8, 14, 15). The distance between plants within the row and between rows would affect the microclimate (7, 10) and, coincidentally, would alter the rate of disease spread. Vestal (12) and Vestal and Bell (13) found an increase of leaf spot (Cercospora beticola Sacc.) in sugar beet with closer plant spacing due to earlier development of infection foci in plots with close plant spacing. They did not consider differences in rates of disease development. This paper reports on the influence of several plot-spacing configurations of celery (Apium graveolens L. var. dulce (D.C.) on disease increase caused by Cercospora apii Fres., according to the concept described by van der Plank (11).

MATERIALS AND METHODS.—Plants of Florida 2-14 celery were transplanted in the field on 20 September 1973 in Everglades peaty-muck soil. One hundred plants per treatment plot were arranged in seven spacing configurations. The between-row and between-plant spacings, respectively, were 60-15, 30-30, 70-20, 80-25, 45-45, 90-30, and 60-60 cm to achieve the plot areas given in Table 1. Plots were arranged in a randomized complete block design with four replications. There was a minimum of 7 m of cultivated strip as a buffer zone between all plots. Disease estimates, when disease was less than 5\%, were made by calculating lesion numbers times average lesion size divided by plant leaf area (3). A modified Horsfall-Barratt rating scale (6), converted with Elanco tables (9), was used at higher disease incidences. Disease was estimated every 6-7 days. Necessary care not to disturb the foliage was exercised when making disease estimates to avoid mechanical detachment of conidia which would result in uncommonly large increases in disease spread (3). Infection rates (4) were calculated according to van der Plank's formula for compound interest disease (11).

RESULTS.—Disease progress.—No C. apii lesions were observed on plants at time of setting, but new lesions were observed within 10 days, indicating that infections were incubating at transplanting (3). The disease estimates for the initial appearance of disease and selected subsequent rating periods are given in Table 1.

The total number of lesions in the plots increased from 370 on 5 October to 585 on 12 October, but no change in percent disease (0.02%) was calculated. The normal increase in plant growth offset the concurrent advance of disease, as detected by increased numbers of lesions. The disease increased rapidly during the next 12 days and an average of 1.5% blighted foliage was calculated from estimates pooled from the seven treatments for 24 October. Substantial disease increase was observed for the remainder of the season.

Plant-spacing effects.—No specific retardation in disease increase during the early part of the epidemic could be attributed to wide plant spacing. However, slower disease increase in wide-plant-spacing plots was found at higher disease incidences later in the epidemic (Table 1). This effect came largely as the disease increased from less than 2% to over 40% (as observed for the closest plant spacing on 24 October and 26 November, respectively). There was approximately half as much disease (21.1%) in the widest plant spacing when estimates were made on 26 November.

Infection rates.—The infection rates (r) calculated for the first few weeks of the epidemic likewise did not reflect any effect of plant spacing on disease (Table 2). Although plant growth records were concurrently taken along with disease estimates, no correlation of changes in plant canopy on changes in infection rates could be found.

The r values calculated from the disease estimates for 24 October to 26 November (33 days) reflect the significant reduction in disease increase brought about by wide plant spacing (Table 2).

From 26 November to 19 December (23 days), the increase of disease in close-plant-spacing plots approximately equaled new plant growth, giving essentially a 0.00 infection rate. The disease progressed slowly, but substantially in the wide-plant-spacing plots after 26 November. The r values for the period show that increase.

The infection rates calculated from the pooled disease estimates reflected the rapid progress as the disease increased up to 1.5% blighted foliage. The calculated r values for selected periods during the balance of the season depicted the Cercospora blight as progressing with a low to moderate rate of spread (r = .02-.15). The rates adequately reflected weather influence on disease spread.

DISCUSSION.—Cercospora apii blight spread more rapidly in close-plant-spacing plots of celery than in wide-

TABLE 1. Selected disease estimates of Cercospora apii in celery plant-spacing plots in 1973

Plot area (m ²) ^b	Disease ^a (%)							
	5 Oct	24 Oct	19 Nov	26 Nov	19 Dec			
6.8	.02 n.s.	1.5 n.s.	25.8 A°	42.1 A	45.8 n.s.			
7.3	.02	1.7	19.5 AB	45.3 A	46.9			
10.6	.02	0.7	18.0 B	40.1 AB	45.8			
15.2	.01	1.2	14.8 BC	39.1 AB	45.3			
16.4	.02	1.8	21.1 AB	33.6 BC	45.3			
20.5	.02	1.8	14.4 BC	27.3 CD	44.8			
29.2	.03	1.5	10.5 C	21.1 D	42.7			
Mean	.02	1.5	17.7	35.5	45.2			

"Average of four replicates.

Area occupied by 100 plants with various between-row and between-plant spacing.

Means in column followed by same letter are not significantly different by Duncan's multiple range test, P = 0.05.

TABLE 2. Infection rates (r) of Cercospora apii in celery plant spacing plots for selected periods in 1973

Plot area (m²)b	r-value for period ^a								
	12 Oct- 18 Oct	18 Oct- 24 Oct	24 Oct- 1 Nov	24 Oct- 26 Nov	1 Nov- 13 Nov	13 Nov- 26 Nov	26 Nov 19 Dec		
6.8	.30 n.s.	.45 n.s.	.14 B ^c	.12 AB	.09 AB	.12 A	.01 n.s.		
7.3	.29	.45	.14 B	.12 AB	.07 B	.14 A	.00		
10.6	.21	.35	.24 A	.14 A	.07 B	.14 A	.01		
15.2	.33	.41	.16 B	.12 AB	.08 B	.14 A	.01		
16.4	.30	.53	.10 C	.10 BC	.10 A	.10 AB	.02		
20.5	.31	.44	.17 B	.09 C	.02 C	.11 AB	.03		
29.2	.29	.36	.12 BC	.09 C	.06 BC	.09 B	.05		
Mean	.29	.43	.15	.11	.07	.12	.02		

Average of four replicates.

Area occupied by 100 plants with various between-row and between-plant spacing.

Means in column followed by same letter are not significantly different by Duncan's multiple range test, P = 0.05.

spacing plots. This difference in rate of spread resulted in twice as much disease in 8 weeks in the more crowded plants. This influence came largely late in the season when leaves of neighboring plants in close spacing were intertwined which undoubtedly altered microclimatic effects within the plant canopy. Plant spacing had no observable effect on initial epidemiologic development as reported by Vestal (12) for *C. beticola*.

The spores of *C. apii* are readily wind-borne (3) and, in this experiment, no separation of infections by spores from within plot or from interplot transport could be made. The uniformity of disease within particular plots implied very adequate short-distance dispersal of *C. apii* spores. Also the 5-week period required for the unequal initial disease levels to equilibrate suggested moderate interplot movement of spores.

The infection rates calculated from the pooled disease data showed very rapid disease progress when observed disease was less than 2%. Similar rapid infection rates for initial epidemiologic stages have been observed earlier for this disease (2), as well as for *Helminthosporium turcicum* Pass. (1, 4). This may be a characteristic of many epidemics. Although the infection rates were not corrected for plant growth increases as suggested by van der Plank (11) the rates still provided good treatment comparisons, and reflected weather influences on disease spread.

Treatment comparisons were made on observed disease, but the plant-spacing effects on microclimate and

infection would have occurred at least one incubation period (12-14 days) previously. The results suggest general spore movement as being responsible for early disease increase with autoinfection becoming more important when plants became crowded. Since the spores of *C. apii* are readily wind-borne, the effect of wide plant spacing on disease was assumed to be largely the result of modified microclimate on infection rather than the loss of spores among plants from various point source gradients (5).

The commercial Florida celery growers currently use plant and row spacings in which 100 plants occupy 6.8 to 10.6 m² which results in crowded plants to maximize yield. The small benefit of less disease gained by wider plant and row spacing would not offset the yield loss from reduced plant populations per unit land area. Consequently celery growers will likely maintain their current close spacings, and utilize varietal resistance and fungicide applications to reduce the infection rate.

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