Progression Dynamics of Hypocotyl Rot of Snapbean

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ABSTRACT

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Severity of snapbean hypocotyl rot, induced primarily by *Rhizoctonia solani* and in part by *Fusarium solani* f. sp. *phaseoli* was evaluated in a total of 22 fields in Clinton County, Pennsylvania, in which snapbeans were grown for commercial use in 1977 and 1978. The 11 fields selected each year were planted to six cultivars in 1977 and five cultivars in 1978. Disease severity was estimated at weekly intervals for each field as the proportion of hypocotyl surface covered by lesions; the assessments were made on 50 arbitrarily selected plants from each field. The "simple interest" disease model was not appropriate for describing the epidemics as indicated by the shape parameter of the Weibull distribution function. Disease progress was, however, adequately described in all cases with a quadratic first-difference regression model and in some cases with only a linear term in the regression model. A hierarchical cluster analysis performed by using six disease progress curve elements identified the presence of at least two epidemic types for each year. The two types could be differentiated largely on the basis of rate of disease progression.

Additional key word: comparative epidemiology.

The quantitative epidemiology of diseases induced by soilborne plant pathogens remains largely unreported in the phytopathological literature due to the complexity of the host-pathogen-environment interactions and the lack of available quantitative biological data for such diseases. The analysis of disease progression dynamics is, however, important in the characterization of disease cycles and eventual disease management systems (6,18).

Vanderplank (18) proposed the concepts of "simple interest disease" (SID) in which, during a single season, an increase of

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disease occurs without a concurrent increase of inoculum which is effective during that season, and the concept of "compound interest disease" (CID) in which an increase in inoculum occurs concurrently with an increase in disease and more than one disease cycle occurs during a season. Since the proposal of these concepts, Bald (1) stated: "During one experiment, one season or the growth of an annual crop, most soilborne pathogens cause 'simple interest' disease." Although the concept of SID is well-rooted in phytopathological thought, few data are available that confirm or deny the appropriateness of the assumption of SID progression in diseases induced by soilborne pathogens.

Bean hypocotyl rot (root rot), which is induced by *Rhizoctonia solani* Kühn and *Fusarium solani* (Mart.) Appel & Wr. emend. Snyd. & Hans. f. sp. *phaseoli* (Burk.) Snyd. & Hans., is potentially destructive wherever beans are grown. Effective genetic, cultural, or chemical controls for this disease have not been developed (14) and the dynamics of disease progression have not previously been investigated. This study was initiated: to describe the progression dynamics of epidemics of bean hypocotyl rot which occur in fields used for commercial production; to examine the appropriateness of the SID model for describing epidemics of bean hypocotyl rot; and to classify the epidemics of bean hypocotyl rot.

MATERIALS AND METHODS

Cultural conditions. A total of 22 fields in which snapbeans (*Phaseolus vulgaris* L.) were grown for commercial use were sampled during the 1977 and 1978 growing seasons. All 11 fields selected for study each year were located in Clinton County, Pennsylvania, and were planted to six cultivars in 1977 and five cultivars in 1978. Row spacing in each field was approximately 0.92 m with a plant density of ~36/m row. The soil in sampled fields was either Ashton sandy loam or Allenwood sandy clay loam. Field characteristics and cultural information are presented in Tables 1 and 2. One field in 1978, BA_1, had not been previously planted to beans. Sample area 'C' was first planted to beans in 1976, and all other fields had been planted intermittently to beans since 1935.

Disease assessment. Fifty plant specimens were removed at weekly intervals from each field, beginning 7–12 days after planting, until a minimum of five weekly samples were collected for each field. Plant specimens were selected at arbitrary intervals along a diagonal through each field at an approximate 45-degree angle to the bean rows. Plants obviously infected by *Pythium* sp., as indicated by the visual symptoms of wilted foliage and gray, water-soaked stem tissue, were not included in any sample.

Plant specimens were brought into the laboratory, washed in running tap water for 8–10 min, and inspected to determine the severity of hypocotyl rot on each specimen. During the 1977 growing season, the first three weekly assessments of disease severity were obtained by direct measurement. A clear plastic ruler, which was imprinted with a millimeter-scale grid, was placed over the infected area and the total area covered by the lesion or lesions was estimated. The vertical length and the diameter of each hypocotyl section also was recorded and the surface area of the hypocotyl was calculated. Disease severity was expressed as the proportion of hypocotyl surface area covered by lesions. During the 1978 growing season, a similar disease assessment procedure was used for the first two weekly assessments of disease severity for each field.

For all other disease assessments, infected bean hypocotyls were placed along the edge of the clear millimeter-scale plastic ruler. The area of the lesions was visually estimated taking into account the linear length and circumference of the area covered by the lesions. Estimates of lesion area were recorded as 1, 2, 5, 10, 20, 30, 40, 50, 60,70,80,90, or 100 percent of the hypocotyl surface area covered.

Isolation from lesions. The presence of *R. solani* and *F. solani* f. sp. *phaseoli* in hypocotyl lesions was verified weekly during the growing season. At least five plants with lesions were selected from the sample of 50 plants from each field and hypocotyl sections were surface sterilized in 0.5% sodium hypochlorite for 2–3 min. A small

TABLE 1. Field characteristics and cultural information for snapbean (*Phaseolus vulgaris*) fields in Clinton County, Pennsylvania, in 1977

Field[a]	Soil[b] type	pH	Area (ha)	Cultivar	Crop history (1975–1976)	Planting date
BC_1	SL	6.5	5.0	Cascade	hay–potato	May 7
BC_2	SL	6.5	4.6	Cascade	hay–potato	May 9
BE	SL	5.9	2.7	Ramano	hay–wheat	May 30
CA_1	SCL	6.8	2.4	G.V.50	corn–bean	June 15
CA_2	SCL	6.8	2.4	G.V.50	corn–bean	June 15
CA_3	SCL	6.8	2.9	G.V.50	corn–bean	June 12
CA_4	SCL	6.8	2.9	G.V.50	corn–bean	June 12
FF	SL	5.7	5.1	Midas	corn–potato	May 18
FG	SL	6.1	5.6	Bonanza	corn–potato	May 27
GB_1	SL	6.3	3.0	Blue Lake	wheat–corn	June 22
GB_2	SL	6.3	4.3	Blue Lake	hay–wheat	June 28

[a] The first letter = sample area identification, the second letter = cultivar, and the number, when present, = specific planting of a cultivar within a field.
[b] SL = Ashton sandy loam; SCL = Allenwood sandy clay loam.

TABLE 2. Field characteristics and cultural information for snapbean (*Phaseolus vulgaris*) fields in Clinton County, Pennsylvania, in 1978

Field[a]	Soil[b] type	pH	Area (ha)	Cultivar	Crop history (1976–1977)	O.M.[c] (%)	Planting date
AA	SL	7.1	3.9	G.V.50	bean–corn	1.2	May 12
BA_1	SL	4.9	2.8	G.V.50	corn–corn	1.9	May 29
BA_2	SL	5.1	3.9	G.V.50	wheat–potato	3.0	June 5
BB	SL	5.3	1.4	Blue Lake	wheat–bean	2.6	June 27
BE	SL	5.5	2.0	Ramano	wheat–bean	2.6	June 24
CA	SCL	6.1	2.4	G.V.50	bean–bean	2.3	June 11
CF_1	SCL	6.1	4.1	Midas	bean–bean	2.3	June 14
CF_2	SCL	6.1	4.1	Midas	bean–bean	2.3	June 16
DB_1	SL	7.2	3.2	Blue Lake	wheat–potato	2.4	June 27
DB_2	SL	7.2	4.0	Blue Lake	wheat–potato	2.4	July 5
EE	SL	6.4	1.0	Ramano	wheat–corn	1.9	June 24

[a] The first letter = sample area identification, the second letter = cultivar, and the number, when present, = specific planting of a cultivar within a field.
[b] SL = Ashton sandy loam; SCL = Allenwood sandy clay loam.
[c] O.M. = organic matter.

tissue sample was aseptically removed from the margin of at least one lesion of the hypocotyl section and placed on acidified 1.5% water agar in a 9-cm diameter petri dish. Cultures were incubated for 7–10 days at room temperature and the fungi isolated were transferred to potato dextrose agar for identification.

Identification of *R. solani* was based on hyphal characteristics including the presence of constrictions of branch hyphae at the point of origin, hyphal branching at nearly right angles, the presence of a septum in the hyphal branch near the point of origin, and the presence of some degree of brown pigmentation in the hyphae (9).

Isolates believed to be *F. solani* were grown under fluorescent light for 2 wk on potato dextrose agar slants and then identified to species level on the basis of macroconidial shape, presence of microconidia, and colony appearance (17). Fifty-six of the 102 isolates of *F. solani* obtained were further tested to possibly indicate formae speciales by a pathogenicity test of these isolates on bean tissue.

Curve elements. From the disease severity values obtained, a disease progress curve (DPC) was developed for each field. Transformation of disease severity values was not made initially since any transformation has within it certain implicit assumptions concerning the nature of disease progression.

DPC's were described using a first difference regression (FDR) model (15,16):

$$y_t - y_{t-1} = B_1(x_t - x_{t-1}) + B_2(x_t^2 - x_{t-1}^2) + (e_t - e_{t-1}) \qquad (1)$$

in which y = disease proportion, B_1 = linear regression coefficient, B_2 = quadratic regression coefficient, x = time of disease assessment, days after planting, e = error term, and t = cardinal numeral of the disease assessment (ie, first, second, third . . .).

Appropriateness of the linear or quadratic model was determined by analysis of residual plots from regression analysis and significance of the regression coefficients (7). If a quadratic term was not needed to describe the DPC, a linear FDR model was used, and thus the B_2 coefficient was zero. The MINITAB Statistical Computing System was used to perform all regression analyses (13).

Area under the disease progress curve (ADPC) was estimated by using the midpoint rule for area estimation (12). Final disease severity (Y_f) was taken as the disease proportion occurring at the last date of disease assessment.

Epidemics were further characterized by fitting curves with the Weibull distribution function (WDF) (10,19). The WDF can be written as:

$$y = 1 - \exp\left\{-[(x-a)/b]^c\right\}, \; x > a \qquad (2)$$

in which y = disease proportion, x = time of disease assessment (days) after planting, a = location parameter, b = scale parameter, and c = shape parameter.

The Weibull probability density function is the first derivative of the WDF and is written as:

$$dy/dx = (c/b)[(x-a)/b]^{c-1} \exp\left\{-[(x-a)/b]^c\right\}. \qquad (3)$$

The quantity dy/dx represents the absolute rate of disease increase and all parameters are defined as indicated previously.

The scale (b) and shape (c) parameters were estimated by using a maximum likelihood technique after the value of the location parameter (a) was set as the time of the first disease assessment less 1 day. Due to the limited size of the epidemic data sets, it was not possible to refine the value of 'a'; therefore, no biological interpretation of the value of 'a' was made in this study. For the WDF, a 'c' value of 1.0 indicates a SID type of progression and a 'c' value of 3.6 indicates a CID type of progression (10). The statistical equality of WDF 'c' values to 1.0 or 3.6 was examined by calculation of a 95% confidence interval about the WDF 'c' value (3) for each epidemic and inspection to determine if the value 1.0 or 3.6 was included in this confidence interval.

Six variables were thus obtained to characterize each disease progress curve. These variables were:

(i) WDF scale parameter (b),
(ii) WDF shape parameter (c),
(iii) area under the DPC (ADPC),
(iv) final disease severity (Y_f),
(v) FDR linear coefficient (B_1), and
(vi) FDR quadratic coefficient (B_2).

Cluster analysis. Hierarchical cluster analysis (4), which is a multivariate statistical procedure to identify groupings of experimental units; eg, bean hypocotyl rot epidemics, was performed on the DPC's from each year. The specific clustering algorithm used was Johnson's (5) maximum method. The six curve elements mentioned above were used for characterizing each curve, and cluster strength and maximum distances within and between clusters were determined. The Statistical Analysis System (2) was used in performing all calculations for the cluster analyses.

RESULTS

Isolation from lesions. *R. solani* was isolated 519 times from a total of 600 bean hypocotyl lesions selected during 1977 and 1978; *F. solani* was isolated 78 times, and both fungi were isolated from 24 lesions. Of 56 randomly selected isolates of *F. solani* tested for possible pathogenicity on bean tissue, 53 were able to induce an

TABLE 3. Six curve characteristics for bean hypocotyl rot disease progress curves, Clinton County, Pennsylvania, in 1977

	Curve elements[a]					
Field	ADPC	Y_f	B_1	B_2	b	c
BC_1	5.02	0.270	1.143	0.000	33.65	2.79
BC_2	7.67	0.348	0.882	0.000	34.22	1.85
BE	2.33	0.227	−0.680	0.022	53.13	3.57*
CA_1	2.04	0.287	−0.500	0.057	36.18	5.22
CA_2	0.91	0.081	0.282	0.000	82.56	2.38
CA_3	2.60	0.226	0.782	0.000	44.76	3.18*
CA_4	1.89	0.183	−0.390	0.021	47.75	3.39*
FF	6.62	0.394	1.066	0.000	56.55	1.77
FG	10.02	0.714	−0.037	0.036	34.01	3.27*
GB_1	1.63	0.103	0.603	−0.005	27.14	9.18
GB_2	2.86	0.097	0.340	0.000	25.65	7.95

[a] ADPC = area under disease progress curve calculated by using the midpoint rule; Y_f = final disease severity (proportion at time of last assessment); B_1 = first difference regression linear coefficient; B_2 = first difference regression quadratic coefficient; b = Weibull distribution function scale parameter; and c = Weibull distribution function shape parameter; values not significantly different ($P = 0.05$) from 3.6 are indicated with an asterisk. A compound interest type of disease progression is indicated when c = 3.6.

TABLE 4. Six curve characteristics for bean hypocotyl rot disease progress curves, Clinton County, Pennsylvania, in 1978

	Curve elements[a]					
Field	ADPC	Y_f	B_1	B_2	b	c
AA	5.53	0.342	1.075	0.000	49.12	2.29
BA_1	0.38	0.042	0.000	0.002	72.63	4.57
BA_2	3.06	0.272	0.000	0.021	41.51	3.34*
BB	2.27	0.251	−0.725	0.037	38.71	2.34
BE	6.15	0.590	0.000	0.044	44.37	3.54*
CA	7.34	0.602	2.150	0.000	29.42	3.12*
CF_1	6.28	0.335	1.190	0.000	27.21	3.22*
CF_2	4.11	0.301	1.010	0.000	60.79	1.63
DB_1	1.93	0.192	0.000	0.016	30.60	3.56*
DB_2	3.15	0.335	1.580	0.000	28.78	3.36*
EE	5.45	0.408	1.450	0.000	39.70	4.08*

[a] ADPC = area under disease progress curve calculated by using the midpoint rule; Y_f = final disease severity (proportion at time of last assessment); B_1 = first difference regression linear coefficient; B_2 = first difference regression quadratic coefficient; b = Weibull distribution function scale parameter; and c = Weibull distribution function shape parameter; values not significantly different ($P = 0.05$) from 3.6 are indicated with an asterisk. A compound interest type of disease progression is indicated when c = 3.6.

expanding lesion. Very few isolates of other fungi, which may have been secondary invaders, were obtained in the first two samples taken from each field. However, as plant age increased, increasing numbers of isolates of secondary invaders, such as *F. oxysporum, F. roseum*, and *Alternaria* sp., were isolated in addition to the two primary pathogens. *Pythium* sp. were isolated eight times and four of these isolates were obtained in conjunction with *R. solani*. Each year isolates of *F. solani* f.sp. *phaseoli* were obtained more frequently in July and August than in May and June. Of the 53 isolates which induced an expanding lesion on bean stem tissue, 16 were obtained in July and 34 in August. *R. solani* was isolated with about equal frequency each month. Twenty-five randomly selected isolates of *R. solani* from each year's collection were assignable to anastomosis group 2 (8), which contains many isolates of *R. solani* capable of inducing root rots.

Curve elements. The calculated values of the six variables used to characterize the 1977 and 1978 bean hypocotyl rot epidemics are presented in Tables 3 and 4, respectively. These values were estimated from the analysis of disease progress curves developed from disease severity assessments obtained in each field.

In 1977, the ADPC, which indicates disease stress (18), ranged from 0.91 to 10.02 standard units. During the 1978 season, the ADPC ranged from 0.38 to 7.34 units. The final proportions of disease present were also lower during the 1978 season (0.042 to 0.602) than during the 1977 season (0.081 to 0.714).

Analysis of residual plots from regression indicated that all disease progress curves could be adequately described with quadratic regression models, and 12 curves could be described with only a linear term in the model. The linear coefficient, B_1, ranged from −0.680 to 1.143 for 1977 DPC's and from −0.725 to 2.150 for 1978 curves. A quadratic term with a coefficient significantly ($P = 0.05$) different from zero was necessary in the regression model to adequately describe five 1977 DPC's and five 1978 DPC's. Quadratic coefficient values ranged from −0.005 to 0.057 in 1977 and from 0.0 to 0.044 in 1978.

Conditional maximum likelihood point estimates of the Weibull distribution function (WDF) scale parameter (b) and the WDF shape parameter (c) also are given in Tables 3 and 4. Scale (b) parameter values, which are inversely related to rate of disease progress, had a range of 25.65 to 82.56 in 1977 and 27.21 to 72.63 in 1978. In all cases, the WDF 'c' parameter was significantly ($P = 0.05$) greater than 1.0 (3), and the 'c' parameter was not significantly different ($P = 0.05$) from 3.6 for four 1977 DPC's and seven 1978 DPC's.

DPC's and density functions (dy/dx) corresponding to a range of Weibull scale and shape parameters are given in Figs. 1–4. Fig. 1 presents DPC's which most nearly represent SID, although for both curves, $77BC_2$ and $78CF_2$, the 'c' parameter was significantly greater than 1.0. The density functions presented in Fig. 1B were generated from estimated Weibull parameters for each specific

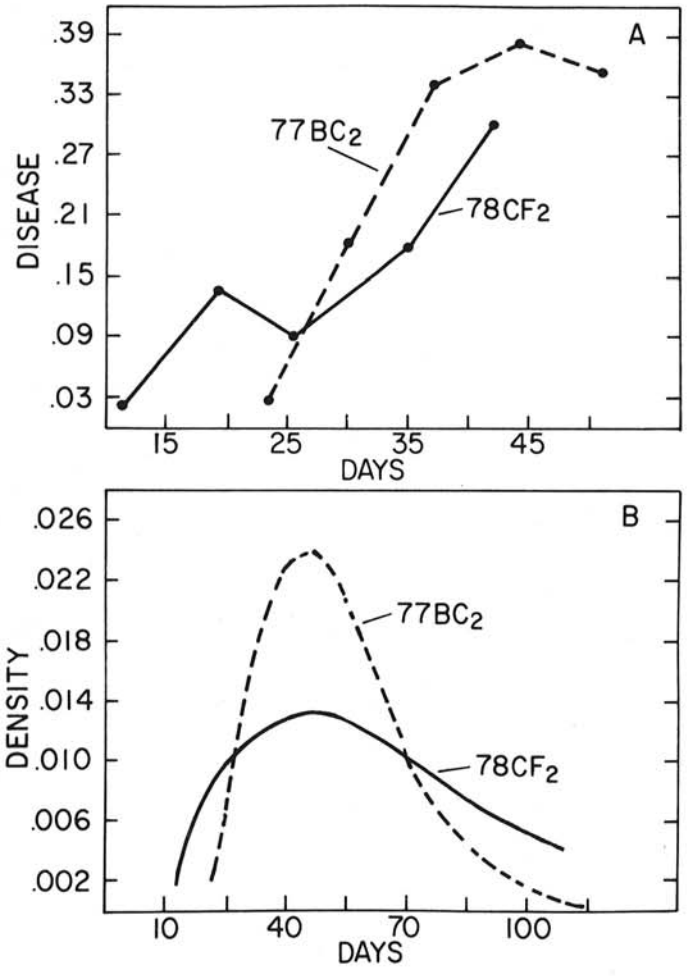

Fig. 1. A, Disease progress curves and **B,** generated probability density function (dy/dx) plots for bean hypocotyl rot epidemics $77BC_2$ and $78CF_2$. Estimated Weibull distribution function scale (b) and shape (c) parameters were 34.22 and 1.85 for $77BC_2$ and 60.79 and 1.63 for $78CF_2$. Disease is proportion of hypocotyl tissue covered by fungal lesions; days are the days after planting. In the alphanumeric field designations, the first number indicates the year, the first letter indicates the sample area identification, the second letter the cultivar (C = Cascade, F = Midas), and the subscript the specific planting of a cultivar within a field.

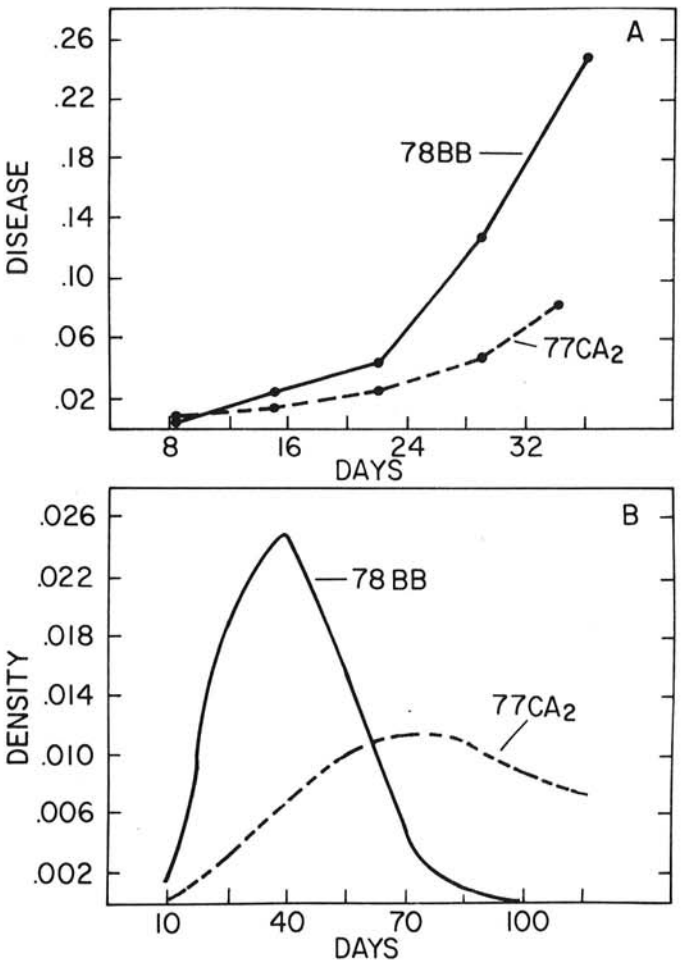

Fig. 2. A, Disease progress curves and **B,** generated probability density function (dy/dx) plots for bean hypocotyl rot epidemics $77CA_2$ and 78BB. Estimated Weibull distribution function scale (b) and shape (c) parameters were 82.56 and 2.38 for $77CA_2$ and 38.71 and 2.34 for 78BB. Disease is proportion of hypocotyl tissue covered by fungal lesions; days are the days after planting. In the alphanumeric field designations, the first number indicates the year, the first letter indicates the sample area indentification, the second letter the cultivar (A = G.V. 50, B = Blue Lake), and the subscript, when present, the specific planting of a cultivar within a field.

curve. Due to the length of the snapbean growing season (approximately 60 days), only a portion of the Weibull density function is realized in a given season. For $77BC_2$ and $78CF_2$ the density functions are strongly skewed to the right, indicating that the greatest absolute rate, dy/dx, of disease progression occurred early in the epidemics. In Fig. 1B, the density plot of $77BC_2$ represents a lower WDF 'b' parameter value than that estimated for $78CF_2$, which also is shown in this figure.

Contrasts similar to that presented in Fig. 1 are shown in Figs. 2–4. Fig. 2 is representative of DPC's with WDF 'c' parameters of 2.3 to 2.4. Values in this range indicate disease progression which is intermediate between the traditional SID and CID models of Vanderplank (18). In Fig. 3, DPC's representative of a CID progression with WDF 'c' values close to 3.6 are presented. Two DPC's are presented in Fig. 4 for which the WDF 'c' parameters are significantly greater than 3.6, indicating a type of disease progression greater than that expected with CID.

Cluster analysis. A cluster map, graphically representing the results of the cluster analysis, is presented in Fig. 5. Physical proximity of individual clusters; eg, GB_1-GB_2 with BC_1-BC_2, does not indicate the closeness of the relationship among DPC's. The vertical and horizontal lines; eg, between BC_1-BC_2 and FG, indicate proper associations and the appropriate cluster strength for the union of the DPC's. The cluster strength is the standardized maximum distance within a cluster group, and increases numerically as the similarity of units within a group decreases. Large increases in numerical cluster strength were used to delineate clusters of DPC's within years.

Joining of DPC's in the clustering sequence occurred because of the similarities of the DPC's as expressed in the six curve elements chosen for this study. With regard to the 1977 DPC's, GB_1 and GB_2 were joined first, BC_1 and BC_2 were joined second, and then CA_3 and CA_4 were brought together (Fig. 5). FG was then joined to BC_1-BC_2, FF and BE were united, and then CA_1 was joined to BC_1-BC_2-FG. The four vertical cluster branches outlined above were established as two major cluster branches at a cluster strength of approximately 0.60. The two major branches were then joined at a cluster strength of 3.85 and CA_2 was finally added at cluster strength 12.53.

The joining of 1978 DPC's (Fig. 5) occurred in a manner analogous to that described for the 1977 DPC's. The two major cluster branches for the 1978 analysis were joined at cluster strength 11.81.

For the 1977 DPC's, three major cluster groups were delineated that contained six, four, and one DPC, respectively. The first two major cluster groups, containing six and four DPC's, represented the two major branches identified previously which were joined at cluster strength 3.85. These branches were established as major clusters due to the relatively large numerical increase in cluster strength between the union of the components of these cluster

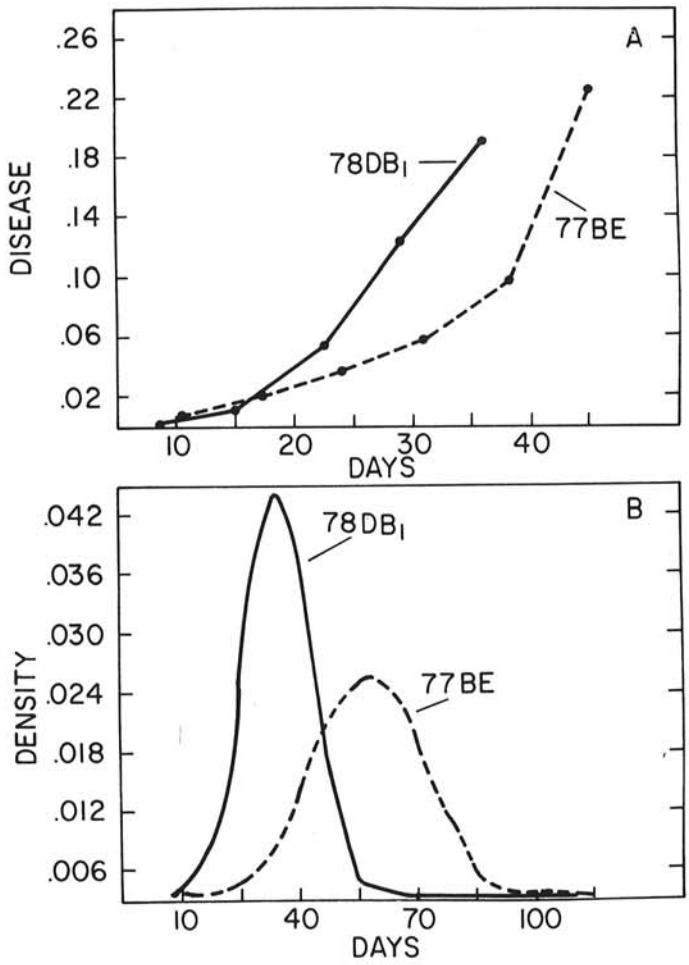

Fig. 3. A, Disease progress curves and **B,** generated probability density function (dy/dx) plots for bean hypocotyl rot epidemics 77BE and $78DB_1$. Estimated Weibull distribution function scale (b) and shape (c) parameters were 53.13 and 3.57 for 77BE and 30.60 and 3.56 for $78DB_1$. Disease is proportion of hypocotyl tissue covered by fungal lesions; days are the days after planting. In the alphanumeric field designations, the first number indicates the year, the first letter indicates the sample area identification, the second letter the cultivar (B = Blue Lake, E = Ramano), and the subscript, when present, the specific planting of a cultivar within a field.

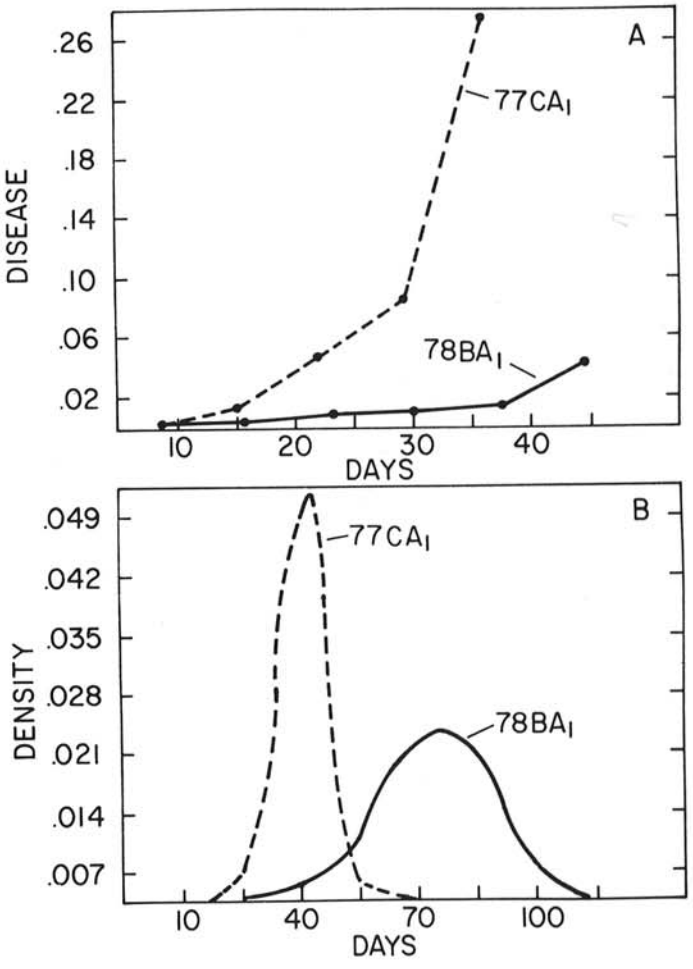

Fig. 4. A, Disease progress curves and **B,** generated probability density function (dy/dx) plots (B) for bean hypocotyl rot epidemics $77CA_1$ and $78BA_1$. Estimated Weibull distribution function scale (b) and shape (c) parameters were 36.18 and 5.22 for $77CA_1$ and 72.63 and 4.57 for $78BA_1$. Disease is proportion of hypocotyl tissue covered by fungal lesions; days are the days after planting. In the alphanumeric field designations, the first number indicates the year, the first letter indicates the sample area identification, the second letter the cultivar (A = G.V. 50) and the subscript the specific planting of a cultivar within a field.

branches and the joining of the cluster branches themselves. The third cluster, which contained only CA_2, was established for similar reasons.

For 1978 DPC's two major cluster groups were obtained which contained six and five DPC's, respectively. These clusters were chosen as a result of the major numerical increase in cluster strength between the establishment of the two major branches and the union of these two branches. A combined cluster analysis for 1977 and 1978 was performed, but no new interpretations could be made.

The maximum standardized Euclidean distance within and between clusters is given for 1977 in Table 5 and for 1978 in Table 6.

TABLE 5. Maximum standardized Euclidean[a] distance within and between clusters obtained through hierarchical cluster analysis for 11 bean hypocotyl rot epidemics in Clinton County, Pennsylvania, in 1977

No. of epidemics	Cluster	1[b]	2[c]	3[d]
6	1	0.587	3.857	12.529
4	2		0.602	5.483
1	3			0.000

[a] Euclidean distance equals the sum of the squared differences among variables.
[b] Cluster 1 includes the following bean fields: GB_1, GB_2, BC_1, BC_2, FG, CA_1.
[c] Cluster 2 includes the following bean fields: CA_3, CA_4, FF, BE.
[d] Cluster 3 includes bean field CA_2.

The diagonal elements of these tables represent the variability (dispersal) within each cluster. In 1977, the two groupings with more than one field; ie, clusters 1 and 2, had similar variabilities. This also was true, but to a lesser extent, in 1978.

Mean DPC characteristics within each major cluster group are presented for 1977 data in Table 7. Cluster 1 of the 1977 analysis had the greatest overall rate of disease progression, represented by the WDF 'b' parameter and FDR B_1 and B_2 coefficients combined. Cluster 1 also had the greatest Y_f, ADPC, and WDF 'c' parameter values. Cluster 3 had the slowest rate of disease progression, which was revealed by the highest WDF 'b' parameter and the presence of only the FDR B_1 coefficient. Cluster 3 also had the lowest mean Y_f, ADPC, and WDF 'c' parameter values. Cluster 2 had values

TABLE 6. Maximum standardized Euclidean[a] distance within and between clusters obtained through hierarchical cluster analysis for 11 bean hypocotyl rot epidemics in Clinton County, Pennsylvania, in 1978

No. of epidemics	Cluster	1[b]	2[c]
6	1	2.499	11.815
5	2		3.561

[a] Euclidean distance equals the sum of the squared differences among variables.
[b] Cluster 1 includes the following bean fields: CA, BE, CF_1, DB_2, AA, EE.
[c] Cluster 2 includes the following bean fields: BA_2, BB, DB_1, CF_2, BA_1.

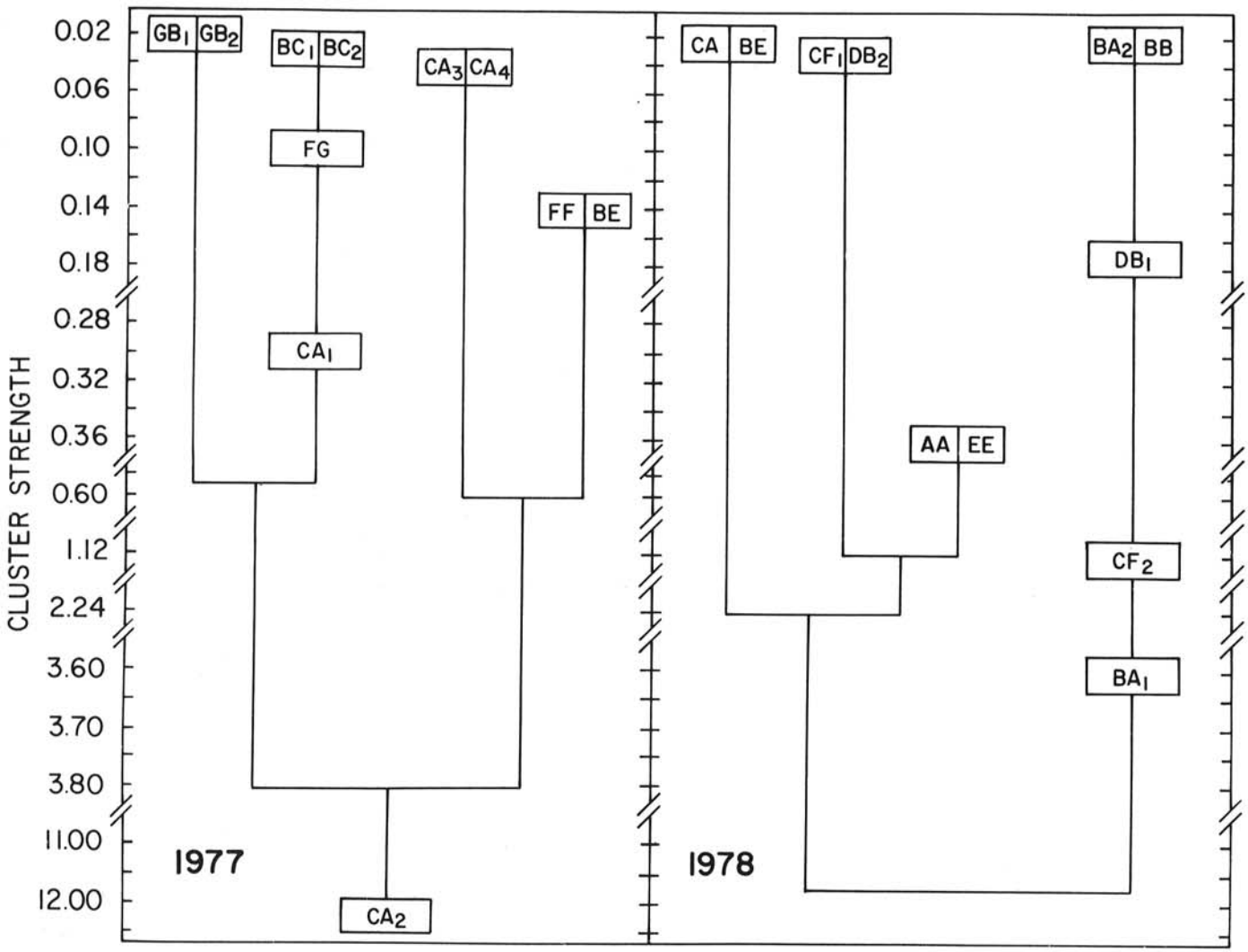

Fig. 5. Cluster maps from hierarchical cluster analysis performed using six curve elements from each of eleven bean hypocotyl rot epidemics in 1977 and in 1978. In the alphanumeric field designations the first letter indicates the sample area identification, the second letter the cultivar (A = G.V. 50, B = Blue Lake, C = Cascade, E = Ramano, F = Midas, G = Bonanza), and the subscript, when present, the specific planting of a cultivar within a field.

intermediate to those of clusters 1 and 3 for all curve elements.

Mean DPC characteristics within the two major cluster groups are given for the 1978 cluster analysis in Table 8. Cluster 1, which contained six DPC's, had the greater rate of disease progression, as represented by the lower WDF 'b' parameter and the combined effects of the FDR B_1 and B_2 coefficients. Cluster 1 also had a greater mean ADPC and Y_f than did cluster 2, but the mean WDF 'c' parameter value for cluster 1 was lower than that for cluster 2.

DISCUSSION

Quantitative epidemiology of plant diseases induced by soilborne pathogens is still in an embryonic stage of development compared with that of plant diseases induced by airborne pathogens. This is due, in part, to the complexity of the soil environment-host-pathogen system and the current lack of information on the interactions which occur below the soil surface.

Bean hypocotyl rot in fields sampled for this study was induced primarily by *R. solani* and in part by *F. solani* f.sp. *phaseoli.* Most disease attributed to *Pythium* sp. was excluded from this study during initial sampling by eliminating plants believed to be infected by that pathogen.

Pieczarka and Abawi (11) studied the effect of *R. solani* and *F. solani* f.sp. *phaseoli* on bean root rot severity in artificially infested, pasteurized soil in a controlled environment and observed no interaction between the two fungi. However, for the organisms involved in this study, if disease progression was analyzed separately for each organism, different types or modes of progression might be encountered. This research was undertaken to study epidemics of hypocotyl rot as they occurred in fields used for commercial production of snapbeans and no attempt was made to differentiate between the effects of the various organisms on disease progression. It was not possible to exclude or differentiate the effects of secondary organisms, which were isolated from some hypocotyl lesions, on disease progression. All disease progression data, therefore, include the effects of *R. solani, F. solani* f.sp. *phaseoli,* and other fungi considered to be secondary invaders.

Hypocotyl rot epidemics involving six snapbean cultivars, in seven locations, and for 2 yr were classified by using cluster analysis. Hierarchical cluster analysis does not provide a specified final number of clusters; the analysis proceeds from a "weak" clustering of individual epidemics to a "strong" cluster in which all the epidemics are included (4). The individual investigator must decide upon a final number of clusters based on large increases in cluster strength (4). It appeared plausible to us that at least two types of hypocotyl rot epidemics occurred each year. These two types of epidemics could be differentiated largely on the basis of rate of disease progression and the types could possibly be labeled on a relative scale as a "fast-rate" and a "slow-rate." These types of epidemics did not correspond to the SID and CID types of disease progression proposed by Vanderplank (18).

In the 1977 cluster analysis, the three cluster groups with the lowest cluster strength or the greatest similarity between curves were GB_1-GB_2, BC_1-BC_2, and CA_3-CA_4. Each of these clusters was composed of two disease progress curves which represented epidemics occurring on the same variety in the same sample area. Although this pattern did not hold for the 1978 disease progress curves, the association of cultivar and field location may be important in the determination of the epidemic types.

By inspection of the field characteristics presented in Tables 1 and 2, the soil physical characteristics (soil type, pH, and organic matter content) did not have a major influence on the demarcation of cluster groups.

The scope of this study was not adequate and the cultural variables were too numerous to permit conclusions concerning possible differences in cultivar susceptibility to bean hypocotyl rot. Differences in curve characteristics were, however, apparently not due to the differences in snapbean cultivars.

Crop history may be a factor in the determination of epidemic types since inclusion of beans and/or potatoes in the previous 2-year crop sequence generally resulted in more bean hypocotyl rot than in cases where these two crops were not included. The specific influence of crop history can be examined by inspection of final disease severity values for field $78BA_1$ and sample area 'C' for 1977 and 1978. Plants in Field $78BA_1$ which previously had not been planted to beans had the lowest disease severity recorded in this study. Sample area 'C' had not been planted to beans prior to the 1976 growing season. Disease severity in sample area 'C' was somewhat greater for the 1978 growing season than for the 1977 growing season. This may have been the result of the 3 yr of continuous bean culture which could provide for an increase in inoculum of pathogens capable of inducing hypocotyl rot. The effects of cropping history on the progress of epidemics induced by soilborne pathogens should be examined with respect not only to the influence of crop history on final disease severity in a given season, but also on the type and severity of epidemics which occurred during several successive growing seasons.

The mode of disease progression for the 22 bean hypocotyl rot epidemics was neither exclusively of the "simple interest" nor "compound interest" type. The Weibull 'c' parameter was significantly greater than 1.0 for all hypocotyl rot epidemics studied; 'c' was significantly different from 3.6 for 11 of the epidemics. These results were insufficient to conclude that the SID model is not appropriate for describing any disease induced by a soilborne pathogen. However, the question is raised as to the wide applicability of the assumption of "simple interest" disease progress for most diseases induced by soilborne pathogens as proposed by Bald (1).

The assumptions for SID progress sensu Vanderplank (18) are that the fungus in soil is the only source of inoculum, and inoculum from one plant does not infect another plant in the same season. Inocula of soilborne fungi probably do not spread from field to field as rapidly or as easily as those of fungi which induce foliar diseases. This would effectively limit the significance of outside inoculum sources during a single growing season for a disease such as bean hypocotyl rot. The fungus in the soil at the time of planting may, however, not be the only effective inoculum for inducing hypocotyl rot on beans, especially for *R. solani.*

Initial inoculum present at the time of planting may induce the primary lesions on bean hypocotyls, but inoculum produced as a result of energy acquired from the host, subsequently may re-infect

TABLE 7. Mean disease severity progress curve characteristics within each cluster obtained through hierarchical cluster analysis for 11 bean hypocotyl rot epidemics in Clinton County, Pennsylvania, in 1977

	Curve element[a]					
Cluster	ADPC	Y_f	B_1	B_2	b	c
1	4.87	0.303	0.239	0.015	31.81	5.04
2	3.35	0.258	0.195	0.011	50.55	2.98
3	0.91	0.081	0.282	0.000	82.56	2.38

[a]ADPC = area under disease progress curve calculated by using the midpoint rule; Y_f = final disease severity (proportion at time of last assessment); B_1 = first difference regression linear coefficient; B_2 = first difference regression quadratic coefficient; b = Weibull distribution function scale parameter; and c = Weibull distribution function shape parameter.

TABLE 8. Mean disease severity progress curve characteristics within each cluster obtained through hierarchical cluster analysis for 11 bean hypocotyl rot epidemics in Clinton County, Pennsylvania, in 1978

	Curve element[a]					
Cluster	ADPC	Y_f	B_1	B_2	b	c
1	5.645	0.435	1.241	0.007	33.97	2.98
2	2.349	0.212	0.187	0.015	51.80	3.43

[a]ADPC = area under disease progress curve calculated by using the midpoint rule; Y_f = final disease severity (proportion at time of last assessment); B_1 = first difference regression linear coefficient; B_2 = first difference regression quadratic coefficient; b = Weibull distribution function scale parameter; and c = Weibull distribution function shape parameter.

the original host or may infect other adjacent plants. Thus, a secondary source of inoculum could be present. Secondary invaders present in the soil also may gain ingress to established lesions and contribute to disease progression.

The plant spacing in commercial bean fields is such that plant stems may be 1–5 cm apart, and roots from adjacent plants often are intertwined. This physical proximity of plants would allow for the spread of the pathogen(s) between and among plants under proper environmental conditions. Thus, for the system of bean hypocotyl rot and perhaps other similar systems which involve soilborne pathogens, both of the assumptions proposed by Vanderplank (18) for SID progression may be inapplicable.

The inoculum dynamics of populations of soilborne pathogens which induce bean hypocotyl rot should be studied in situ under variable crop sequences and the effects of crop history related to the epidemics of hypocotyl rot. Environmental and host factors which may influence the progress of this disease must also be quantified and examined to determine the effects of these factors on disease progress.

The quantification of disease systems that involve soilborne pathogens should provide valuable information on the similarities and differences which may exist between diseases induced by soilborne and airborne pathogens. This knowledge may result in new strategies for the management of diseases induced by soilborne pathogens.

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