Analysis of Disease Progress Curves, Gradients, and Incidence-Severity Relationships for Field and Phytotron Bean Rust Epidemics

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ABSTRACT

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Six bean rust field epidemics in 1979 and 1980 were monitored for disease severity, disease incidence, hourly temperature, hourly leaf wetness, leaf area per plant, and number of leaves per plant. These epidemics were initiated by artificially inoculating 1.83 × 1.83-m areas at the centers of 44 × 44-m plots of *Phaseolus vulgaris* 'Bountiful' with race 34 of *Uromyces phaseoli* var. *typica*. Disease gradient patterns over time were unique to each epidemic and reflected the pattern of the disease progress curves for the central inoculated area. Disease progress curves appeared to depend more on the frequency and length of wetting period than on temperature. Incidence vs severity relationships differed for each epidemic and were too variable within an individual epidemic to be of practical use for estimating disease severity from disease incidence data. Under controlled phytotron conditions, five epidemics under different environmental regimes, initial inoculum levels, and inoculation dates produced similar-shaped disease progress curves that varied in time displacement. Incidence vs severity relationships reflected the rate of the epidemic, the time of disease onset, and the rate of host growth. These field and phytotron epidemics can serve as verification tools for future disease simulator development.

Additional key words: epidemiology.

In the 6 yr since Shrum (12) developed his flexible plant disease simulator, it has been applied only to wheat stripe rust. One reason for this is that considerable data are required to develop specific components for the simulator to fit a new disease. Another reason is that data on progress of epidemics of the new disease must be combined with detailed weather data collected during the epidemics so that the validity of the simulator adapted to the new disease can be tested. One purpose of our research was to provide these data on which future tests of a bean rust simulator may be based.

Another purpose of the research was to investigate the relationship between disease incidence and severity in bean rust. James and Shih (7) first investigated the relationship between disease incidence and severity for two diseases of wheat; they found those relationships to be of limited usefulness for the estimation of final severity from incidence data representing low severity values. Such an investigation has not been performed with bean rust or with any other disease over the course of an epidemic.

We collected data from both field and phytotron epidemics so that more confidence might be placed in the use of phytotron epidemiological research for the purpose of studying field-related phenomena. Disease gradients in field epidemics were calculated to further characterize spatial and temporal epidemic progression for future simulation. Incidence and severity relationships were examined to investigate their usefulness, not only for estimating disease severity, but also for characterizing epidemics.

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MATERIALS AND METHODS

Both field and phytotron epidemics were induced on *Phaseolus vulgaris* L. 'Bountiful' plants derived from a single seed lot. Urediospores of *Uromyces phaseoli* (Pers.) Wint. var. *typica* Arth., which were derived from a single-pustule isolate through three generations were used in all inoculations. Race 34 of the pathogen (3) was chosen because of its local availability and severity reaction rating of 5 (most severe) (2) on the universal suscept cultivar Bountiful.

Epidemics were induced in phytotron chambers regulated for a 13-hr daylength and the following day/night temperatures and leaf wetness conditions: 18/18 C, wet three nights per week; 21/21 C, wet five nights per week; 24/18 C, wet five nights per week; 24/21 C, wet five nights per week; and 24/24 C, wet three nights per week. Details of leaf inoculation with dry spores, the method of leaf wetting, and the proper host plant growth conditions were previously described in detail (5). The actual numbers of spores used for inoculation varied among the epidemics. Temperature, moisture, and initial inoculum conditions were chosen with the expectation of producing different disease progress curves, thereby producing data more useful for eventual disease simulator testing.

Two field plots (~44 × 44 m, with 50 rows spaced at 91 cm) were located approximately 160 km apart, one in the Piedmont (Raleigh) and the other in the Coastal Plain (Lewiston) regions of North Carolina. Forty-one areas 1.83 × 1.83 m (6 × 6 ft), each spanning two rows, were sampled weekly for disease incidence and severity. The sampled areas were located at the midpoints of the parallel sides and at the corners of a series of squares concentric around the initially inoculated center area (Fig. 1). For the calculation of disease gradients, distances were measured as the distance from the center of the initially inoculated central sampling area to the center of any other given sampling area. Thus, in each of

eight directions, plants sampled at the corners and sides of five concentric squares around the inoculated center, yielding a total of 10 distances from the center (ie, five distances in four directions to the corners of each concentric square, and five distances in four directions to the midpoints of each side of each concentric square). Because isopleth graphs of the epidemics indicated that wind direction had been variable enough to cause spread in all directions, the calculations of disease gradients were based upon averages of the four disease severity values at a given distance.

Plots were planted in early May and early August of 1979 and in early May of 1980. Plant densities were measured 3–4 wk after emergence during each of the six epidemics. After disease was initially detected in the inoculated area, no farm machinery or irrigation equipment entered the fields. Disease ratings were made proceeding from the least to most severely affected areas, and care was taken to avoid brushing against the plants to minimize inoculum spread by physical contact. Air temperature was measured and recorded hourly approximately 800 m from the plots in a standard vented meteorological housing. Leaf wetness was recorded within the bean canopy with a hygrothermograph modified with a third arm for recording the output of a leaf wetness sensor (13). When the leaf wetness sensor was inoperative, estimates of wetness periods were determined from hourly rainfall and relative humidity records after humidity patterns during nights of known dew periods were studied.

Between 6 and 13 days after emergence, all plants in the central sampling areas were inoculated in late evening with a suspension of bean rust spores in 0.1% water agar applied with a hand sprayer. Moistened plastic bags were placed over the plants and removed by 0900 hours the next morning. Because latent periods ranged from 8 to 10 days, the initial weekly disease rating did not begin until 14 days after inoculation, and primary gradients were always observed 21 days after inoculation.

In both field and phytotron epidemics, individual leaflets were rated for disease severity by comparing the infected leaflet to a series of 48 photographs of infected leaves depicting severities ranging from 0.5 to 90%, as determined by a leaf-area meter. The visual rating scale incorporated different-sized leaves and pustules and various pustule densities. Half of all the photographs depicted severities $\leq$ 10%, and 37.5% depicted severities $\leq$ 5%. In this study, disease severity refers to the percentage of leaf area with obviously diseased tissue, including any disease-associated chlorosis and necrosis. Disease incidence was measured as the percentage of leaflets sampled with detectable disease. Nearby bean fields were observed weekly for rust incidence after disease ratings had been made.

In the phytotron, 90–150 leaflet samples were rated on each sampling date (two to three times weekly) among a total of 60 plants per environmental chamber. In the field, 30 leaflets were nondestructively rated from each sampling area each week, 15 from each row. In both field and phytotron, sample leaflets were chosen randomly within three canopy levels: bottom, middle, and top. Disease ratings were discontinued when average disease severity approached 10% (phytotron) or when the oldest bean pods began to form fully developed green seeds (field) (growth stage R-7 to R-8) (8).

At each disease sampling date, 10 randomly selected plants were chosen from the field and three from phytotron chambers for leaf counting and leaf area measurements, again with a leaf area meter. The two primary leaves were counted as a single leaf for these determinations. In the phytotron, because only 60 plants were involved in the experiment, data on leaf number and area samples were drawn from previous experiments at similar temperatures. We were not able to determine leaf area in the field in 1980.

RESULTS

In the phytotron epidemic at 24/24 C, disease severity ratings were initially at a very low level and decreased with time. Disease progress curves for each of the other four phytotron epidemics were similarly shaped (Fig. 2A). The time from initial inoculation to rapid disease increase varied with temperature, but different wetting periods and plant growth stages at time of inoculation also may have been influential factors. Rates of both leaf area increase and number of leaves per plant were greater at 24 C up to 20 days after emergence (Fig. 2B). At 27 C, beginning at ~30 days after emergence, many small leaves accounted for the increase in leaf number despite a drop in leaf area per plant. The coefficient of variation for leaf number and area at any given sampling time was 20–40%.

Disease incidence vs severity curves varied for each phytotron epidemic (Fig. 2C). The curves shown are hand-drawn best fits to the data. The 18/18 C epidemic reached 100% incidence at a lower average severity than all the other epidemics. The curve for the 21/21 C epidemic appeared to reach 100% incidence at a lower severity than the 24/18 C or 24/21 C epidemics, although lack of high severity values makes this result tentative.

Disease progress curves for the inoculated centers of all field epidemics differed markedly for each epidemic (Figs. 3A, 4A, 5A, 6A, and 7A). The epidemic for Raleigh in the fall of 1979 is not shown graphically; the initial inoculation, followed by 12 days of 31 C or higher afternoon temperatures, failed to produce any disease. A second field-wide inoculation, followed by milder temperatures, resulted in very little disease.

Average numbers of leaves per plant and average leaf area per plant were also variable in each epidemic (Figs. 3B, 4B, 5B, 6B, and 7B), reflecting to some degree the vigor of the plants under each set of field conditions. Plants in the field produced fewer and smaller leaves than those in the phytotron. The coefficient of variation for the 10-plant sample used for leaf area determinations was greater at the Raleigh site (15–90%, avg 66%) than at the Lewiston site (12–55%, avg 34%). For determinations of number of leaves per plant, coefficients of variation were 20–45% (avg 34%) at Raleigh at 9–35% (avg 21%) at Lewiston. Thus, plant growth throughout the field was more uniform at Lewiston than at Raleigh.

Double logarithmic plots of disease gradients did not depart significantly ($P = 0.001$) from linearity in all cases (Figs. 3C, 4C, 5C, 6C, and 7C) as determined by regression analysis. Gradients also tended to flatten with time, as shown by statistically significant increasing slopes (less negative) of the double logarithmic plots ($P = 0.01$). Only in the spring of 1980 at Raleigh (Fig. 6C) did the disease gradient not vary with time. In all cases, however, the

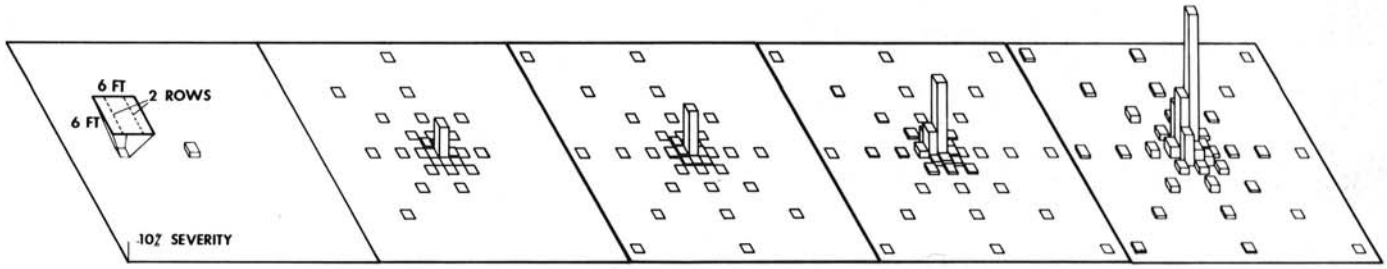

Fig. 1. Bean rust disease severity increase over weekly intervals in the fall of 1979 at Lewiston, NC, showing the 41 disease sampling areas and their arrangement about the initially inoculated center area. Time range is from 20 (first frame) to 48 (last frame) days after emergence. Sampling areas were 1.83 × 1.83 m (6 × 6 ft).

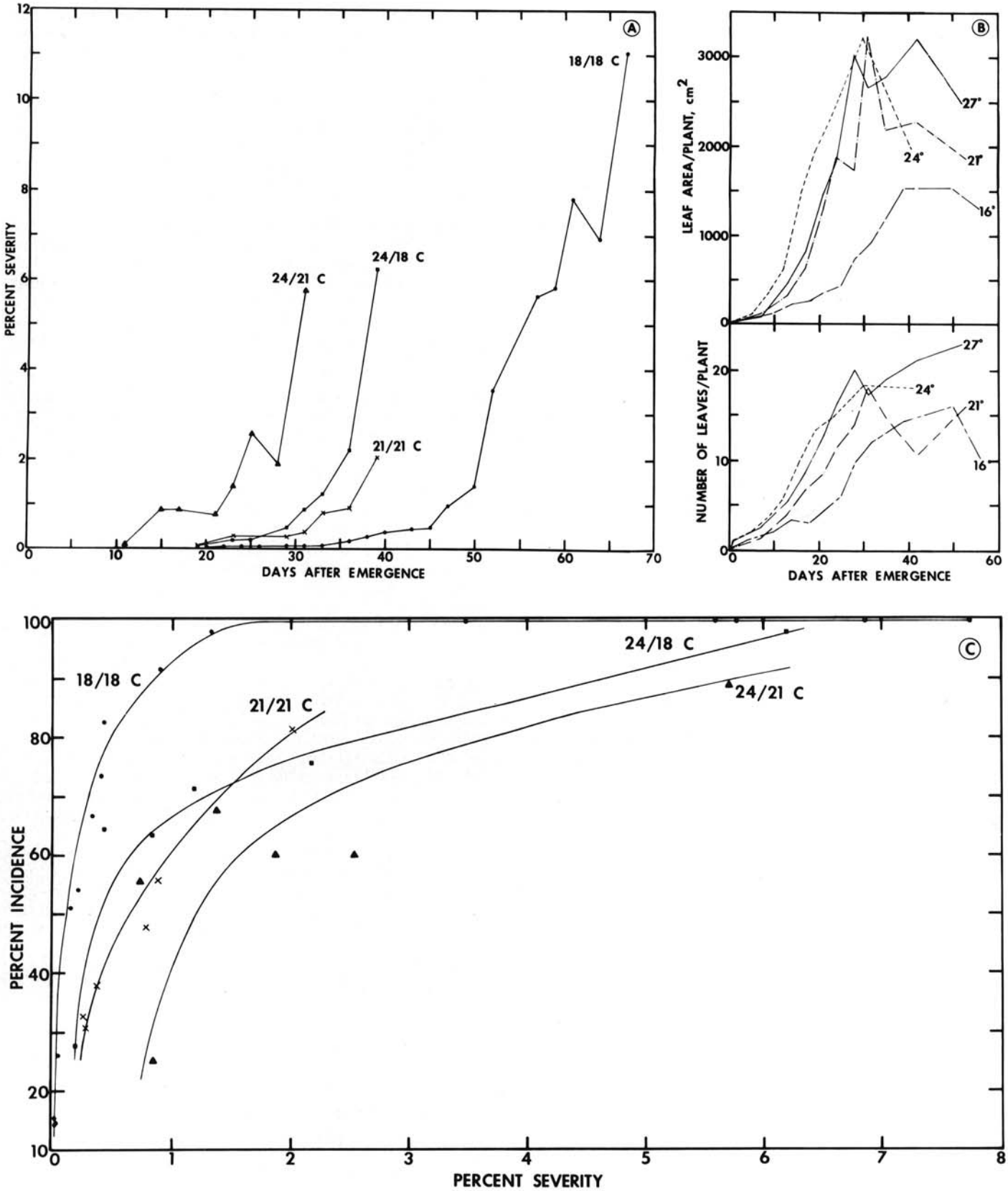

Fig. 2. Characterization of bean rust epidemics carried out in phytotron controlled environment rooms. **A,** Disease severity vs time. Bean plants grown at 24/24 C and inoculated 11 days after emergence produced an antiepidemic. **B,** Average bean leaf area and leaf number per plant vs time for four constant-temperature regimes. Each data point represents the mean of three plants. **C,** Disease incidence vs severity for four epidemics. Each data point represents 90–150 individual leaflet ratings.

gradient remained relatively steep. The inoculated center area and distances yielding average disease ratings of zero were excluded from these graphs. No rust was observed in nearby fields for the duration of all disease ratings for all the epidemics.

The relationship between incidence and severity also varied among epidemics (Figs. 3D, 4D, 5D, 6D, and 7D). In fact, only in spring of 1979 at Raleigh (Fig. 3D) did incidence reach and remain at 100%. The curves presented, which are hand-estimated best fits, may not truly portray the actual best fit, given the great data variability. At least three different sampling dates are included in each graph, and the inoculated center area was excluded as a data point. Data points derived from disease ratings late in the season typically had higher incidence and severity values than those derived from earlier ratings, but this did not appear to change the shapes of these relationships. No consistent correlation was found between the shape of a given incidence vs severity relationship and its respective epidemiological characteristics.

DISCUSSION

The progress of an epidemic under constant temperature conditions may be quite different from that of an epidemic in a regime with fluctuating temperatures, even if the mean temperatures for the two epidemics are similar. For instance, the mean temperature of the 24/18 C epidemic was 21.5 C, just slightly higher than 21 C for the 21/21 C epidemic, but the 24/18 C epidemic progressed much more rapidly. This was in spite of the fact that the two epidemics started with nearly identical initial disease severities at the same host growth stage and each had five wetting periods per week. The effects of fluctuating temperatures on disease progress can be incorporated into a disease simulator such as Schrum's (12), in which changes in rates of disease progress are calculated from hourly mean temperatures.

In the phytotron, distinct temporary reductions in average disease severity occurred in both the 24/21 C and 18/18 C epidemics (Fig. 2A). The reduction at 60–65 days after emergence (18/18 C) was due to senescence of many highly infected leaflets.

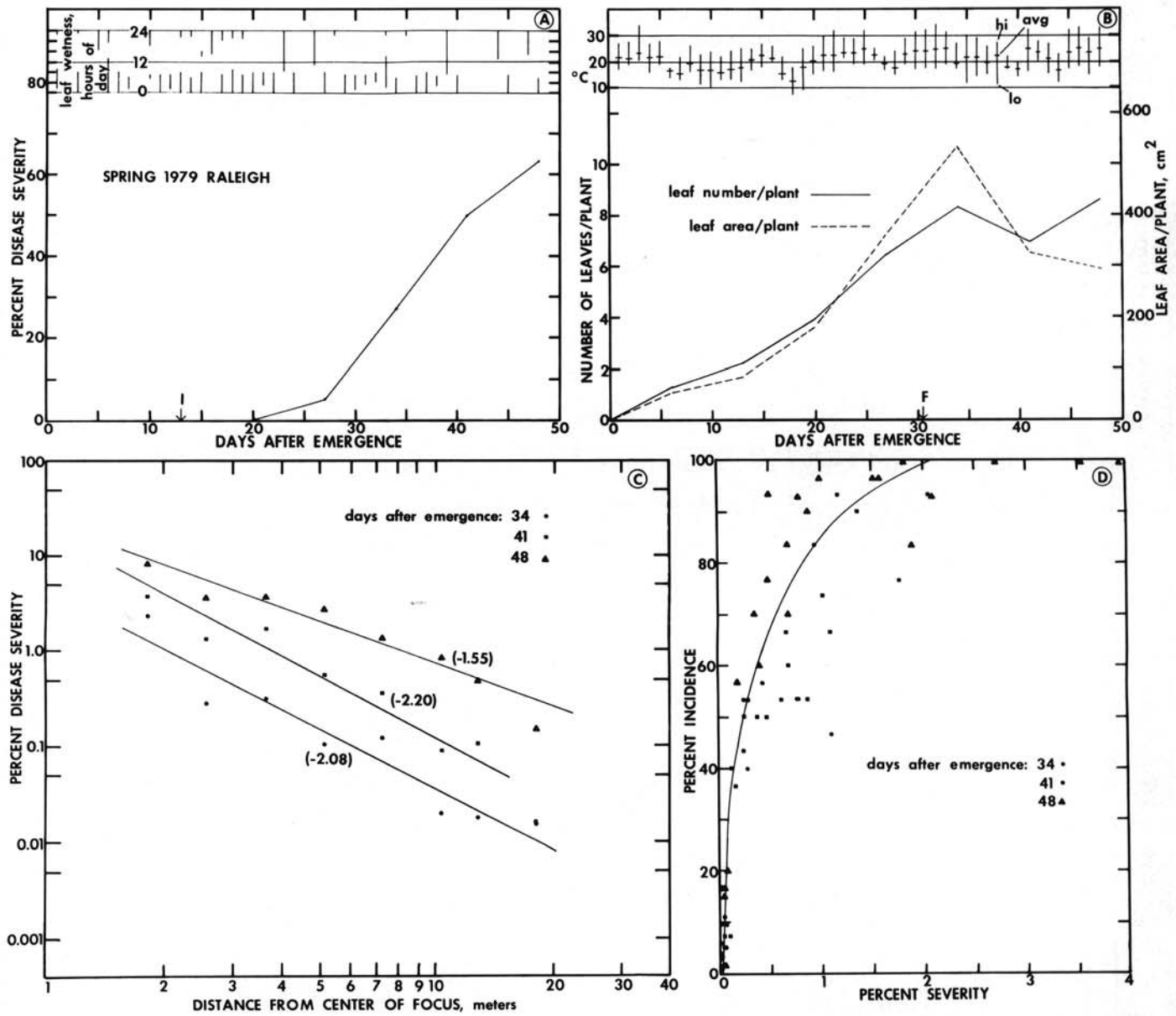

Fig. 3. Characterization of a field epidemic of bean rust in the spring of 1979 at Raleigh, NC. **A,** Disease severity increase in the initially inoculated central area. The scale above represents the hours of the day the leaves were wet. I = inoculation date. **B,** Average bean leaf area (Figs. 3–5) and leaf number per plant. The scale above represents the high, low, and average temperature each day. F = onset of flowering. **C,** Disease severity vs distance from the central initially inoculated area, plotted on a log-log scale for three to four sampling dates. Numbers in parentheses indicate the slopes of the regression of log severity on log distance. **D,** Disease incidence vs severity for all sampling dates. Each point represents at least 30 individual leaflet disease ratings. Plant population density = 16.9 per 1.83 m (6 ft) of row; SE = 4.1; and I = 21 May. In C, the slopes 34 and 41 days are not significantly different ($P = 0.05$).

The sudden removal of these leaves receiving high disease ratings (>70%) resulted in the reduced average severity indicated. Most of these were leaves that had been initially infected 40–50 days after emergence. The reduction at 25–30 days after emergence (24/21 C) was due to rapid host growth; many new leaves received a zero disease rating. Of all the temperature regimes used for the epidemics, only 24/24 C provided more rapid host growth than the 24/21 C regime. In fact, host growth was so great in the 24/24 C epidemic and wetting periods were so sparse, that the plants "outgrew" the fungus; by the time the first latent period was completed, the pustules were already buried within a thick canopy. Dispersal of spores by shaking the plants (5) never enabled the spores to reach the young upper leaves. Thus, a very low initial inoculum level coupled with rapid host growth produced this antiepidemic.

An additional variable included in the phytotron epidemics other than temperature and wetting periods was initial inoculum levels. This factor may best explain the rapid increase of the 24/21 C epidemic. Not only were the plants inoculated at a younger stage, allowing for a higher proportion of young vigorous leaves to become infected than in any of the other epidemics, but the initial inoculum also was much greater. As the first pustules were just beginning to open, disease severity was rated at 0.09% (11 days after emergence). However, by the time all the pustules resulting from the initial inoculation had opened and some had begun to form halos, severity was rated at 0.85% (15 days after emergence), much higher than any other phytotron epidemic. Disease severity then slowly decreased due to increased host growth without a corresponding pustule increase until 23 days after emergence, when infections resulting from the second latent period became visible. This steplike pattern of disease increase has been well described (14), and is also present (but not so noticeably) in the other phytotron epidemics. The weekly rating system of field epidemics in this study masked the expected stepwise form of their respective disease progress curves.

Because farm machinery was excluded from the field plots 1 wk after inoculation, the bean fields in this study did not mimic commercial fields due to lack of cultivation, irrigation, and fertilizer topdressing. The phytotron-grown plants were different from both our own field plot plants and those found in commercial fields. Therefore, it was essential to include some indication of the plant growth habit in each epidemic if the epidemics were to be compared. For simplicity, number of leaves, leaf area, and flowering onset were monitored in these epidemics.

The variability among disease progress curves of the field epidemics can be explained more by the frequency of wetting

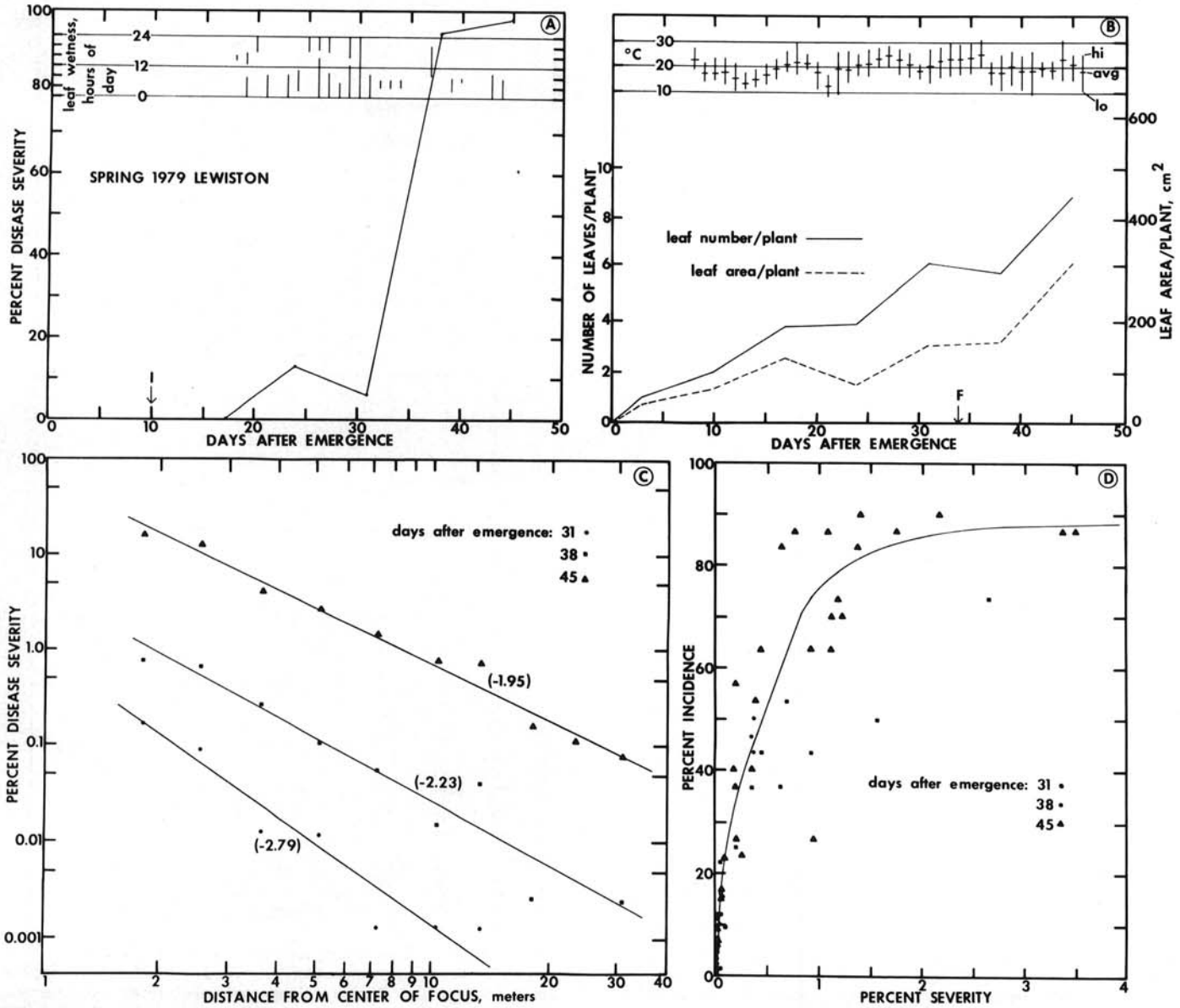

Fig. 4. Characterization of a field epidemic of bean rust in the spring of 1979 at Lewiston, NC. (**A–D** are as described in the caption of Fig. 3). Plant population density = 17.4 per 1.83 m (6 ft) of row; SE = 2.6; and I = 15 May. All slopes differ significantly ($P = 0.01$).

periods than by temperature. In the spring of 1979 at Raleigh (Fig. 3A and B), frequent wetting periods occurred during the first 8 hr of the day, when temperatures were always conducive for spore germination (6,11). The epidemic of spring 1979 at Lewiston (Fig. 4A and B) showed a decrease in disease due to an increase in foliage during several long rain periods (24–31 days after emergence), followed by almost total infection of all leaves. Average disease severity ratings of 95–97% come about only if most leaves are rated at 100% severe. The long heavy rains, coupled with bean beetle damage in this field, contributed to a lack of vigor in these plants. Because the disease rating scale did not distinguish necrosis and yellowing due to disease from that which might be also brought about by other factors on an infected leaf, the very high average severity values of 95–97% observed in this epidemic reflect the total damage to the plants, which was more than the damage due to rust alone.

The epidemic of fall 1979 at Raleigh (not shown) did not develop disease after the initial inoculation. The reason for this was originally thought to be 12 days of 31 C or higher afternoon temperatures immediately following inoculation (9). However, during fall 1979 at Lewiston, plants developed disease despite very similar temperature conditions after inoculation (Fig. 5A and B). The bean plants at Raleigh were heavily infected with *Rhizoctonia,* and this may have adversely affected the rust.

The epidemic in spring 1980 at Raleigh did not develop due to lack of sufficiently lengthy wetting periods (Fig. 6A and B). Wetting periods of less than 3 hr do not support infection, and may actually contribute to the loss of spore viability (6). The wetting periods 22–23 and 25 days after emergence may have led to the disease increase observed at 38 days, which consisted almost entirely of newly opened pustules. By that time, the leaves bearing pustules from the initial inoculation had already senesced. The 8-hr wetting period on day 34 did not appear to result in any increase in disease. Because of the series of short wetting periods from day 26 to day 31, there may not have been any viable urediospores left on the leaves at day 34.

During the spring of 1980 at Lewiston, by contrast, disease developed in spite of few wetting periods (Fig. 7A and B). In fact, uninterrupted dry periods preceding wetting periods at 30 and 33 days after emergence should have allowed spore numbers to increase. Short wetting periods of 1–3 hr can negate the effectiveness of a wetting period, as occurred in spring 1980 at Raleigh (26–30 days after emergence). Lack of such short periods in Lewiston allowed the epidemic to continue. Thus, the disease

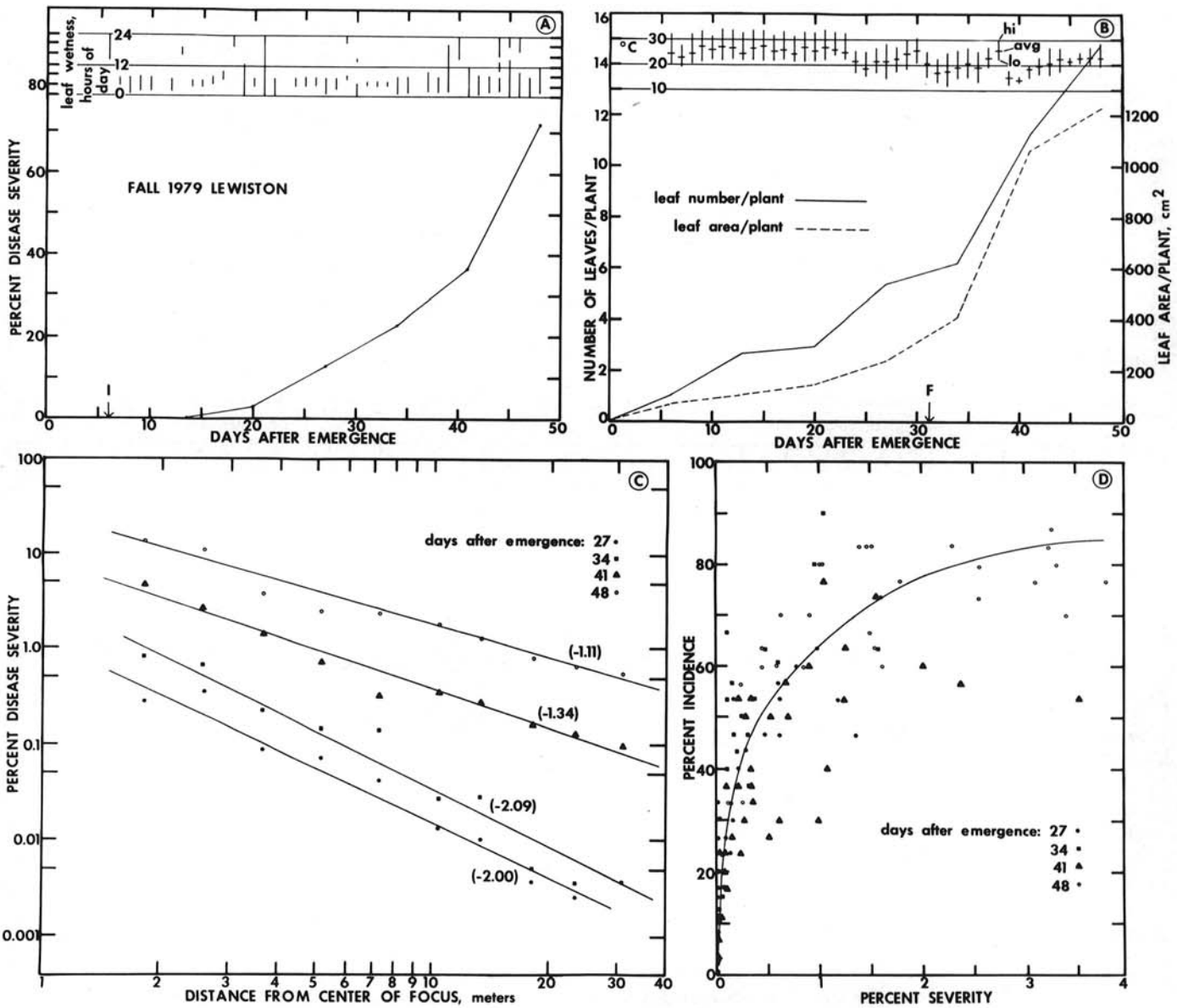

Fig. 5. Characterization of a field epidemic of bean rust in the fall of 1979 at Lewiston, NC. (**A–D** are as described in the caption of Fig. 3). Plant population density = 14.9 per 1.83 m (6 ft) of row; SE = 1.6; and I = 21 August. In B, note the abrupt slope change in leaf area per plant and, in C, that the slopes for 27 and 34 days are not significantly different ($P = 0.05$).

incidence seen after 31 days at Lewiston was due to only three wetting periods: at 25, 30, and 33 days.

As Gregory (4) pointed out, if a disease gradient can be described as $X = a/(d^b)$, then a plot of log (X) (disease severity) against log (d) (distance from focal source) should yield a straight line of slope b. Although this pattern for disease gradients is not universal, it was observed repeatedly in this study (Figs. 3C, 4C, 5C, 6C, and 7C). The flattening of disease gradients over time (increasing values of b) was also observed in four of five epidemics. Berger and Luke (1) plotted logit X vs log d to compare disease gradients. With this transformation, the gradients tend to maintain the same slope over time as long as disease increases at the same apparent infection rate at the center as at the periphery of the plots. While the logit transformation can provide information about apparent infection rates, it tends to obscure the comparisons of actual disease severities at different distances from the focus of infection.

The gradients presented in this study can be used to quickly compare different epidemics over the distance of the whole field and over time. Figure 6C shows that this epidemic progressed little over time or space, and then regressed. The epidemic of spring 1980 at Lewiston (Fig. 7C) clearly progressed at a much greater rate later than earlier in the season. Figures 3C, 4C, and 5C represent data for field-wide epidemics that progressed at steady but respectively unique rates.

Graphs of disease incidence vs severity were originally made with the hope that they might lead to a simpler method of disease rating, especially at low average severities. James and Shih (7) showed that estimation of the severity of two wheat diseases from their incidences in a disease survey could drastically reduce labor requirements over traditional severity assessment methods if incidence was below 65%. A method of exploring and characterizing incidence-severity relationships was presented. Seem and Gilpatrick (10) found that the incidence of powdery mildew on apple was proportional to the square root of severity, and that this relationship was constant among different locations, cultivars, and leaf positions. Results of both studies indicated that the relationship of incidence to severity varied among different seasons, and neither followed the relationship of incidence to severity over the course of an epidemic. Results presented in Figs. 3D, 4D, 5D, 6D, and 7D show that in bean rust epidemics, the relationship of incidence to severity varies between seasons and epidemics. However, within a given epidemic, variability is so high

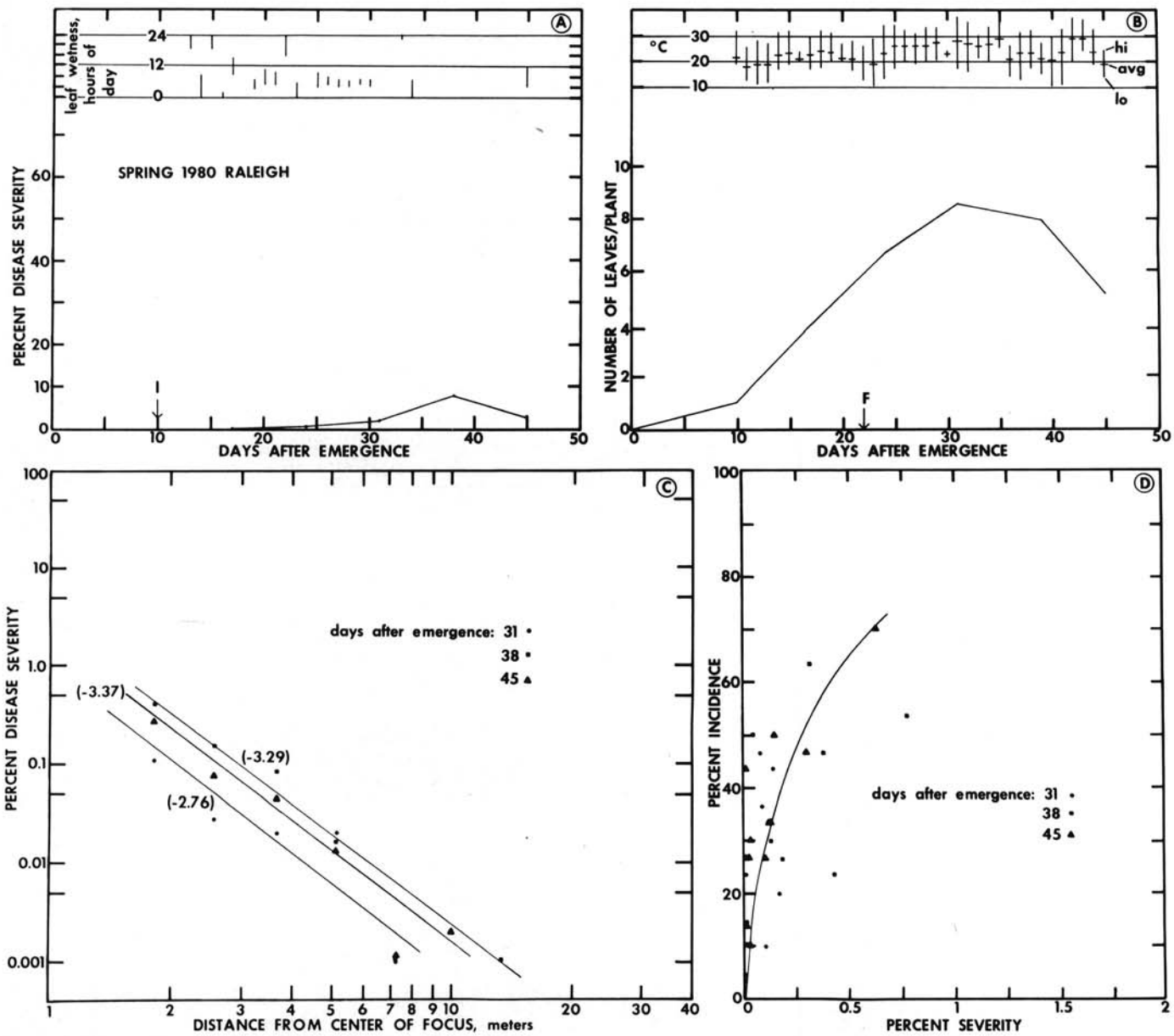

Fig. 6. Characterization of a field epidemic of bean rust in the spring of 1980 at Raleigh, NC. (**A–D** are as described in the caption of Fig. 3). Plant population density = 17.0 per 1.83 m (6 ft) of row; SE = 4.0; and I = 14 May. In C, the slopes do not differ significantly ($P = 0.05$) and, in D, note that the percent severity scale differs from those used in Figs. 3–5, and 7.

that a practical, useful relationship is nonexistent, and only rough estimates are possible. The incidence data presented here might be used with some confidence as long as incidence remained below 20%. At these severity levels, though, it would take very little extra effort to actually count infections rather than infected leaflets. Using incidence data instead of lesion numbers would sacrifice some accuracy, but save very little effort.

To reach a disease incidence of 100% leaflets infected, a plant must be producing no new leaves either because it is mature or so severely infected that it has ceased growing. The only field epidemic in which 100% incidence was reached in any of the sampling areas occurred in the spring of 1979 at Raleigh (Fig. 3D). In this epidemic the numbers of leaves per plant began to decrease as early as 34 days after emergence of the plants. A similar decrease in numbers of leaves also occurred in the spring of 1980 at Raleigh (Fig. 6), but that epidemic failed to develop, so disease incidence did not reach 100%.

In the phytotron, incidence vs severity curves were distinct for each epidemic and in all but one case contained less variability than curves developed from field data. As in the field, actively growing plants prevented incidence from reaching 100% in all but the 18/18 C epidemic, which lasted 67 days. The incidence vs severity curves presented by James and Shih (7) were derived from disease ratings of single specific leaf positions, whereas in this study ratings were based on random selection of leaflets throughout the canopy. This might account for the greater variability observed in the bean rust incidence vs severity relationships than in James and Shih's (7) wheat rust and mildew curves.

A more rapidly progressing epidemic will reach high severity values sooner and on younger plants, than will a more slowly progressing epidemic that was initiated simultaneously. This is most likely the reason why the phytotron epidemics, especially the most rapidly progressing ones, attained 100% incidence at much higher severities than did the slower epidemics (Fig. 2). Therefore, it appears that the point at which an incidence vs severity curve levels off depends indirectly on the rate of the epidemic, and directly on the rate of new leaf production.

The original motivation behind documenting bean rust epidemics under various environmental conditions was to eventually attempt simulation of these results with a modified version of Shrum's (12) flexible disease simulator. Because disease progress curves and gradient patterns of all the epidemics differ,

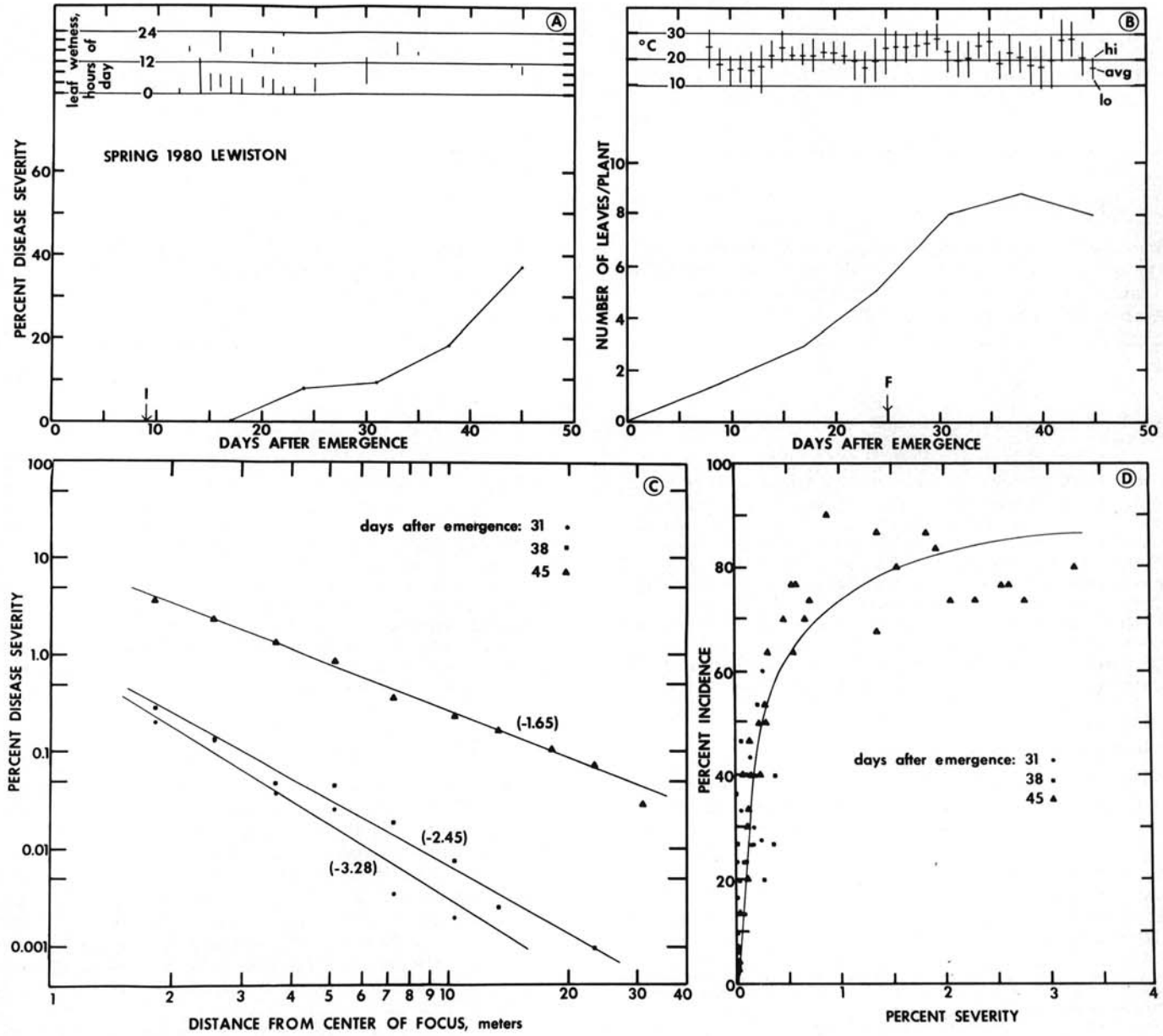

Fig. 7. Characterization of a field epidemic of bean rust in the spring of 1980 at Lewiston, NC. (**A–D** are as described in the caption of Fig. 3). Plant population density = 36.5 per 1.83 m (6 ft) of row; SE = 4.0; and I = 13 May. In C, all slopes differ significantly, $P = 0.03$.

successful simulation of these results should increase confidence in the predictive capability of the simulator and its flexibility over varying environmental regimes.

Complete compilations of data, including hourly temperature values and disease severity values, are available upon request.

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