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TECHNICAL MANUSCRIPT 299

SPREAD OF RICE BLAST IN SMALL FIELDS

Thomas H. Barksdale

MAY 1966

UNITED STATES ARMY
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U.S. ARMY BIOLOGICAL CENTER
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TECHNICAL MANUSCRIPT 299

SPREAD OF RICE BLAST IN SMALL FIELDS

Thomas H. Barksdale

Crops Department
BIOLOGICAL SCIENCES LABORATORY

Project 1C522301A061 May 1966

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FOREWORD

The information presented in this manuscript is a detailed study of some of the data derived from experiments previously described in Technical Report 69.

ABSTRACT

Increase and concurrent spread of rice blast from foci in small fields is described under nearly ideal conditions for infection. Lesion counts and severity estimates were made at stations on polar coordinates at regular intervals. Spore loads in the air were measured with rotobars uniformly spaced around the foci and corrected to spore hours per cubic meter. Increase within foci, spread from foci, and relationship between disease and spore load in the air are shown. Lesions were present six days after inoculation. Growth of foci in all fields was similar and logarithmic notwithstanding initial size. Toward the end of the epiphytotic spore load decreased faster than disease attenuated. In early stages of an epiphytotic, when 40 or fewer lesions occur per acre, there is little chance of detecting spores on a rotobar.

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I. INTRODUCTION

This paper describes increase and concurrent spread of rice blast, a "compound interest" type of plant disease, from a single focus in each of three fields. In concept, this study owes much to the work of Schmitt et al.[1] on the spread of black stem rust of wheat from foci. Because fields in this study were small, the establishment of secondary foci was obvious in only one instance and there is no attempt to consider foci as units of spread, as does van der Plank.[2] An exhaustive review of all aspects of rice blast was made recently at a symposium held at the International Rice Research Institute.[3]

II. MATERIALS AND METHODS

A study of spread of the rice blast disease, caused by *Piricularia oryzae* Cav., Race 1, was made in three Gulfrose rice fields; Fields A and B were 1.1 acres and Field C was 1.7 acres in size. Fields were separated by 2.5 miles, and no rice was grown for 100 miles in any direction. This separation gives some assurance that the disease situation in any one field was not appreciably affected by disease in another or by endemic disease. Areas of disease to serve as foci were established in field centers:

	Initial Focus Size		Initial Disease Intensity	
	sq ft	Acre	Lesions/ft	Total Lesions
Field A	4	0.0001	4.25	17
Field B	400	0.01	17.0	12,000 (est.)
Field C	4356	0.1	15.9	118,600 (est.)

Leaf blast was measured periodically as the disease spread and intensified. During early spread entire fields were surveyed; later, measurements were made at sampling stations located at 25-ft intervals along lines radiating from focus centers along polar coordinates (Fig 1). There were 44 such stations in Field A, 47 in B, and 59 in C. When disease intensity was low, these measurements were in terms of lesions/linear row ft. As lesions became more numerous and began to coalesce, we changed to an estimate of per cent tissue affected similar to standards pictured by Chandraratna.[4] Data on both lesions/linear row ft and severity ratings were obtained on 58 occasions when lesions were numerous enough to warrant a severity rating, and yet few enough to count discretely. The relationship seems to be that one lesion/linear row ft equals an average severity of 0.008902% on a crop at about the mid-tillering growth stage; or, in terms of van der Plank's x-proportion of tissue infected—0.000039. Disease data are expressed in terms of x throughout this paper.

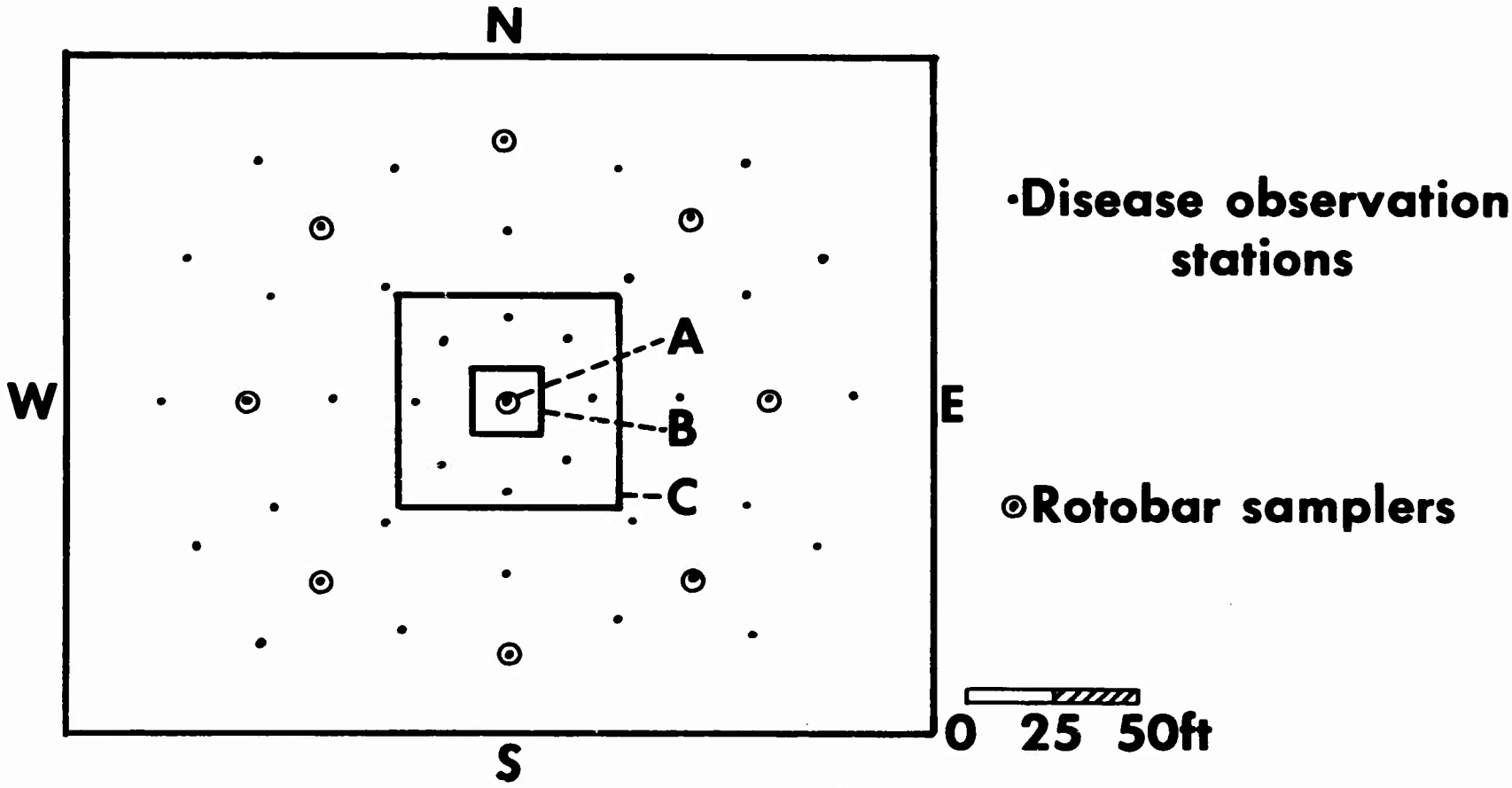


Figure 1. Plot Diagram, Showing Focus Size for Fields A, B, and C, and Location of Spore- and Disease-Sampling Stations.

In addition to disease measurements, the spore load in the air between 3 and 4 AM was measured with rotobar samplers[5-7] placed one meter above ground at the focus center and at 75 ft from center along eight of the coordinates (Fig. 1). The time of sampling was chosen in order to sample a near-peak spore load. There is a diurnal fluctuation in the number of Piricularia spores found in the air, with maximum numbers occurring in the early morning hours between midnight and dawn.[3,8,9] This phenomenon is associated with environmental factors favorable for sporulation and, as some laboratory studies have indicated,[10] for spore release.

The rotobars were chrome-plated to give a reflective viewing surface to facilitate microscopic assessment of the hyaline Piricularia spore. Rotobars were U-shaped; each arm was 6 cm long and 1.59 x 1.59 mm in cross section with a circular trajectory of 4 cm radius. A rubber cement adhesive that remained tacky during exposure in the warm humid environment was used to coat the collecting arms. The flow rate of the sampler was 120 liters/min, or 7.2 m^3/hr.

The counts of spores collected on rotobar samplers were converted to spore hours/cubic meter of air, which is the type of term used in studies of atmospheric diffusion as explained by Pasquill.[11] The term as used here is a measure of the spore load put into the air by a diseased crop, but it is also a measure of exposure or dosage (number of spores that rotobar samplers, plants, and other objects are exposed to in the environment). This measure of dosage is numerically equal to the average concentration during the unit of time chosen. With our data, 100 spore hours/m^3 corresponds to an average concentration of 100 spores/m^3 for the one hour between 3 and 4 AM. Since a constant concentration during the night is not implied, we have used the dosage term.

III. RESULTS

Although disease increase and spread often occur together, the results of this experiment are divided into three groupings: increase within foci, spread from the foci, and the relationship between disease and spore load in the air.

A. INCREASE WITHIN FOCI

Disease within the 4 ft^2 focus of Field A increased very little throughout the season, and a calculation of a rate of increase for this focus is not attempted.

The proportion of tissue affected during the epidemic is tabulated in Table 1 for all three initial foci. A plot of $\log_{10} [x/(1-x)]$ for foci in Fields B and C is shown in Figure 2; although the first observation was made 7 days after inoculation, lesions would have been visible on the 6th day, and the plot begins on the 6th day. The rate of disease increase, r, calculated according to van der Plank's formula 3.6[2] for the Field B focus between days 14 and 35 was 0.222; for the Field C focus between days 10 and 31, r was 0.397. Even if other t values are chosen for the calculation of r, e.g., days 11 and 29 for Field B to give r = 0.325, the r in the larger focus of Field C is greater than r in the smaller one of Field B, and this is especially marked early in the epidemic. A likely explanation suggested by van der Plank's work[2] is that a greater percentage of spores escape from small infected plots than from large ones.

B. SPREAD FROM FOCI

Spread from the 0.0001-acre focus in Field A was first noted 17 days after inoculation (Fig. 3). One lesion was observed 2 ft W and another 2 ft N of center. On day 20, a lesion was spotted 5 ft E and another 15 ft S. On day 23, the focus had enlarged to 40 ft ENE and 12 ft W; in addition a lesion was found 115 ft WSW near the field border and could be considered a secondary focus. After 48 days, lesions could be found throughout the field.

Spread from the 0.01-acre focus in Field B was also first noted 17 days after inoculation (Fig. 4), but disease appeared at greater distances, in all directions, and was more severe than in Field A during the same time interval. After 35 days, disease could be found throughout the field.

Spread from the 0.1-acre focus in Field C was first noted 11 days after inoculation, as one lesion 100 ft SSE. Three days later disease could be found throughout the field. Thereafter, disease severity increased in all parts of the field, but on any given day the severity at a particular location was associated somewhat with its distance from the initial focus. Figure 5 shows the time at which the proportion of tissue affected reached 0.5 or more for various portions of the field. Disease in the initial focus, for some unexplained reason, increased faster in the northern than in the other parts of the focus instead of uniformly.

The increase in area diseased in each field is plotted against time in Figure 6 where dotted lines toward the top of the plots indicate that disease had reached the border of the field in some direction, and one does not know how much farther disease would have extended if the fields had been larger. The plots indicate that the growth of foci in all three fields is similar and logarithmic in spite of initial size, which is surprising because one would expect the relative growth rate of foci to decrease with the decrease in the ratio of their perimeter to area

TABLE 1. PROPORTION OF TISSUE AFFECTED (x) WITHIN INITIAL FOCI

Days After Inoculation	Field A 4 ft^2	Field B 400 ft^2	Field C 4356 ft^2
7 (initial obs.)	0.000378	0.001495	0.001415
10	-	-	0.00202[c]
11	-	0.000833[a]	0.0027
14	0.000734	0.002976[b]	0.0782
17	-	0.005015	-
18	-	-	0.3133
20	0.000312	-	-
22	-	0.04162	-
23	-	-	0.6722
26	0.000401	-	-
29	-	0.226[a]	-
30	0.000512	-	-
31	-	-	0.8988[c]
35	0.000601	0.246[b]	-
38	-	-	0.7444
40	0.000378	-	-
44	0.000312	0.186	-
45	-	-	0.6600
48	0.000067	-	-
51	-	0.178	-
52	-	-	0.6011

a. r between these dates = 0.325.
b. r between these dates = 0.222.
c. r between these dates = 0.397.

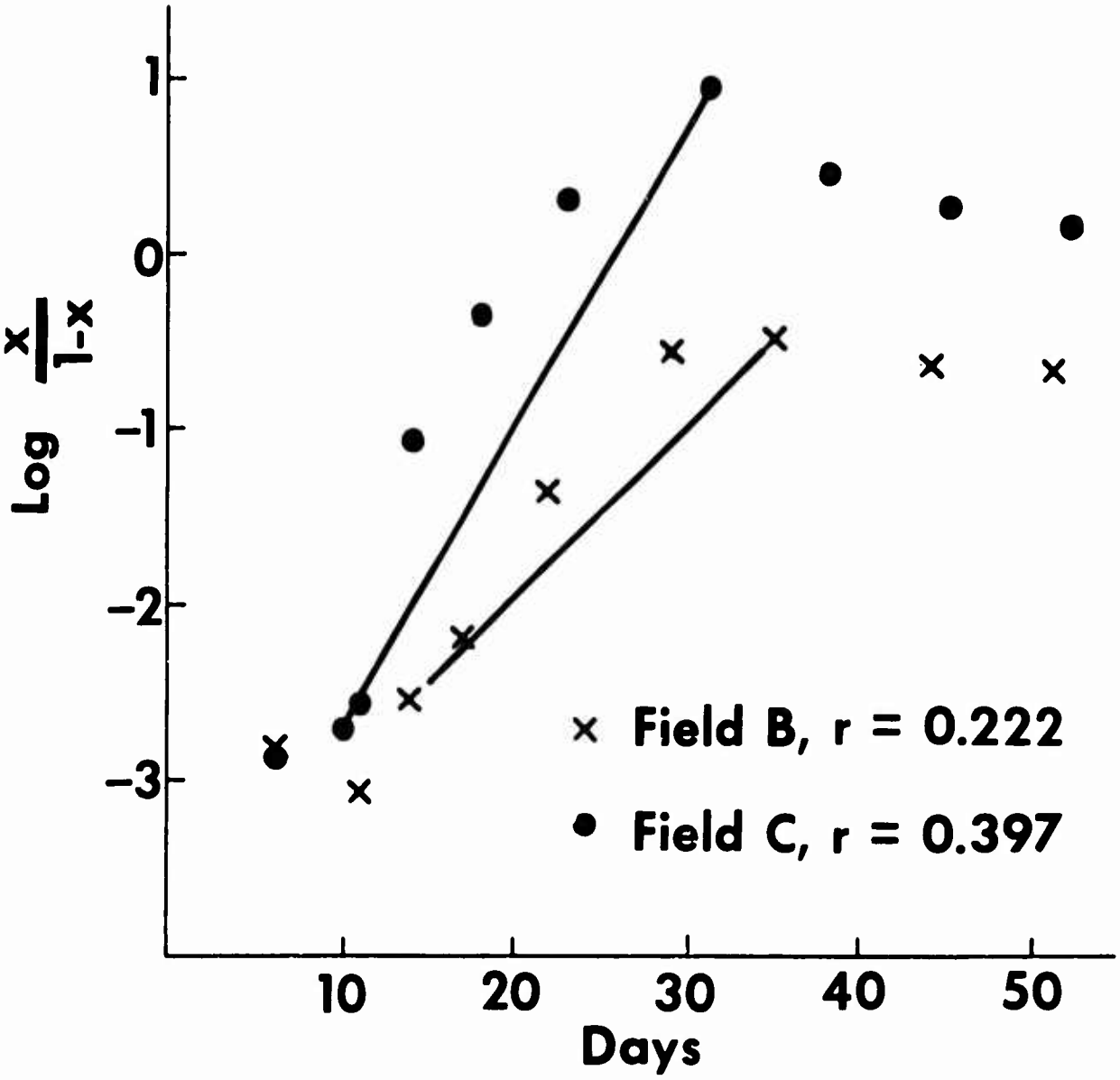


Figure 2. Increase of $\text{Log}_{10} \frac{x}{1-x}$ in Foci.

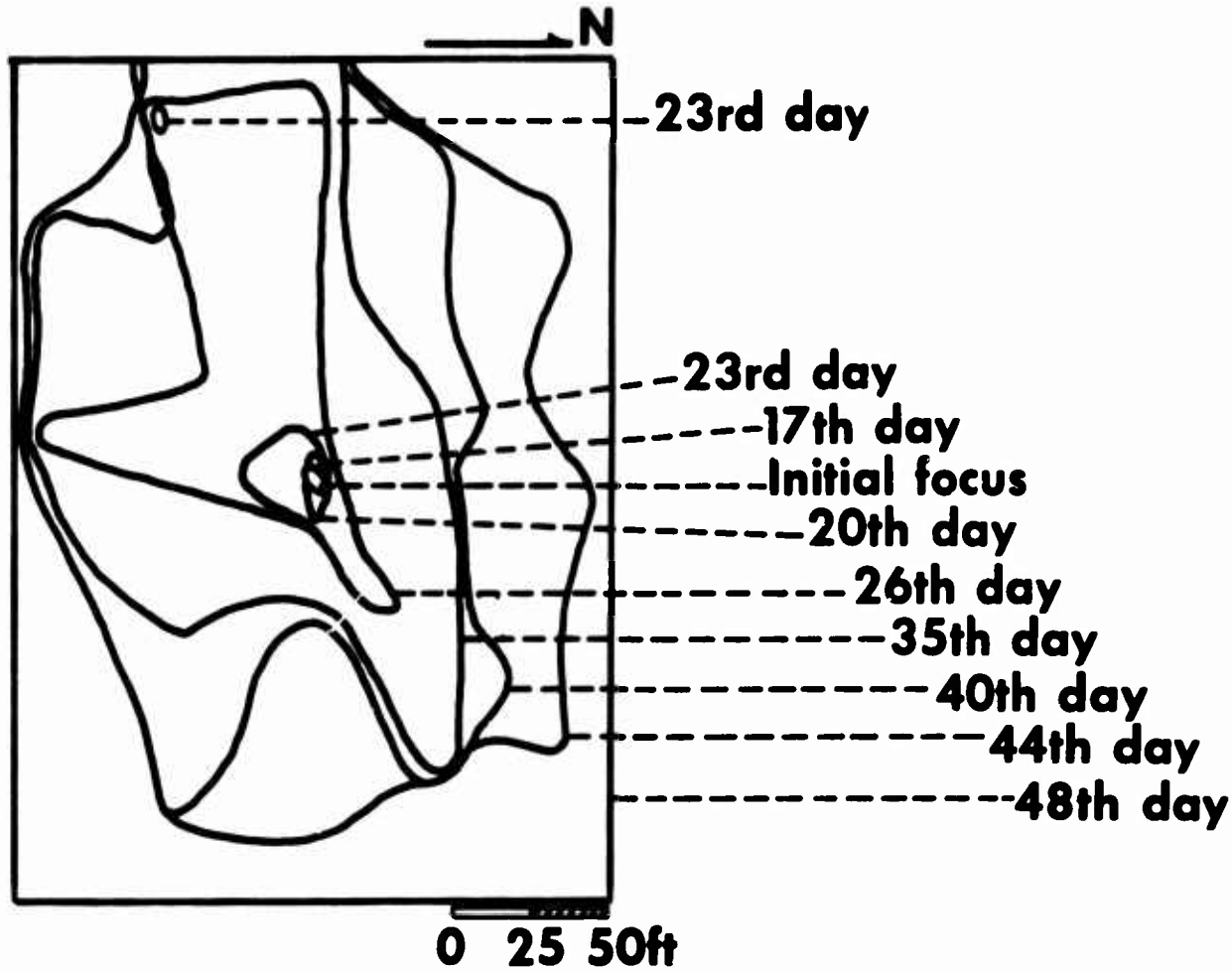


Figure 3. Spread in Field A Showing Limit of Disease Occurrence on Indicated Days after Inoculation of the Initial Focus.

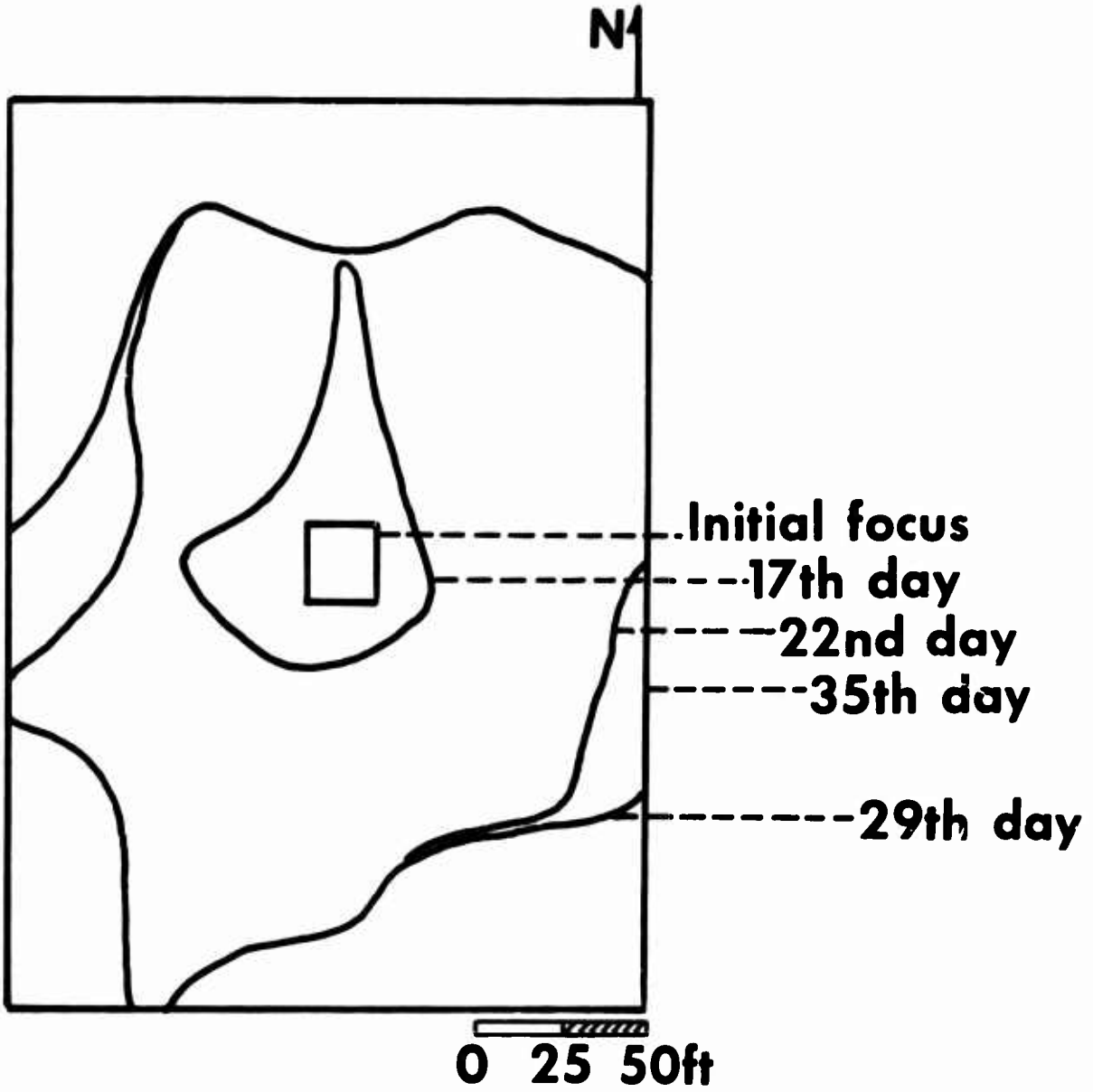


Figure 4. Spread in Field B Showing Limit of Disease Occurrence on Indicated Days after Inoculation of the Initial Focus.

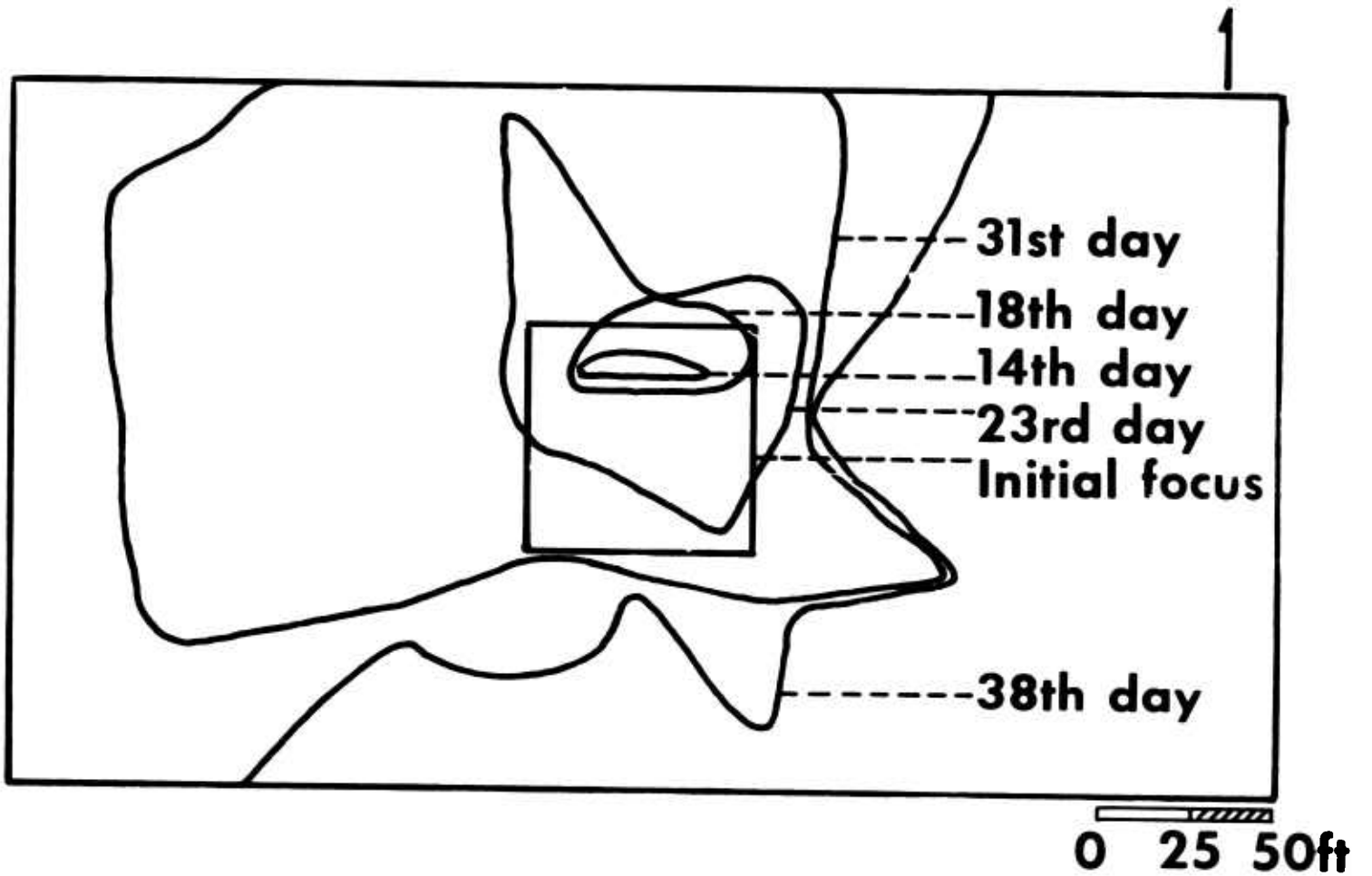


Figure 5. Number of Days after Inoculation of Initial Focus in Field C when x Became 0.5 or Greater in Areas of the Field.

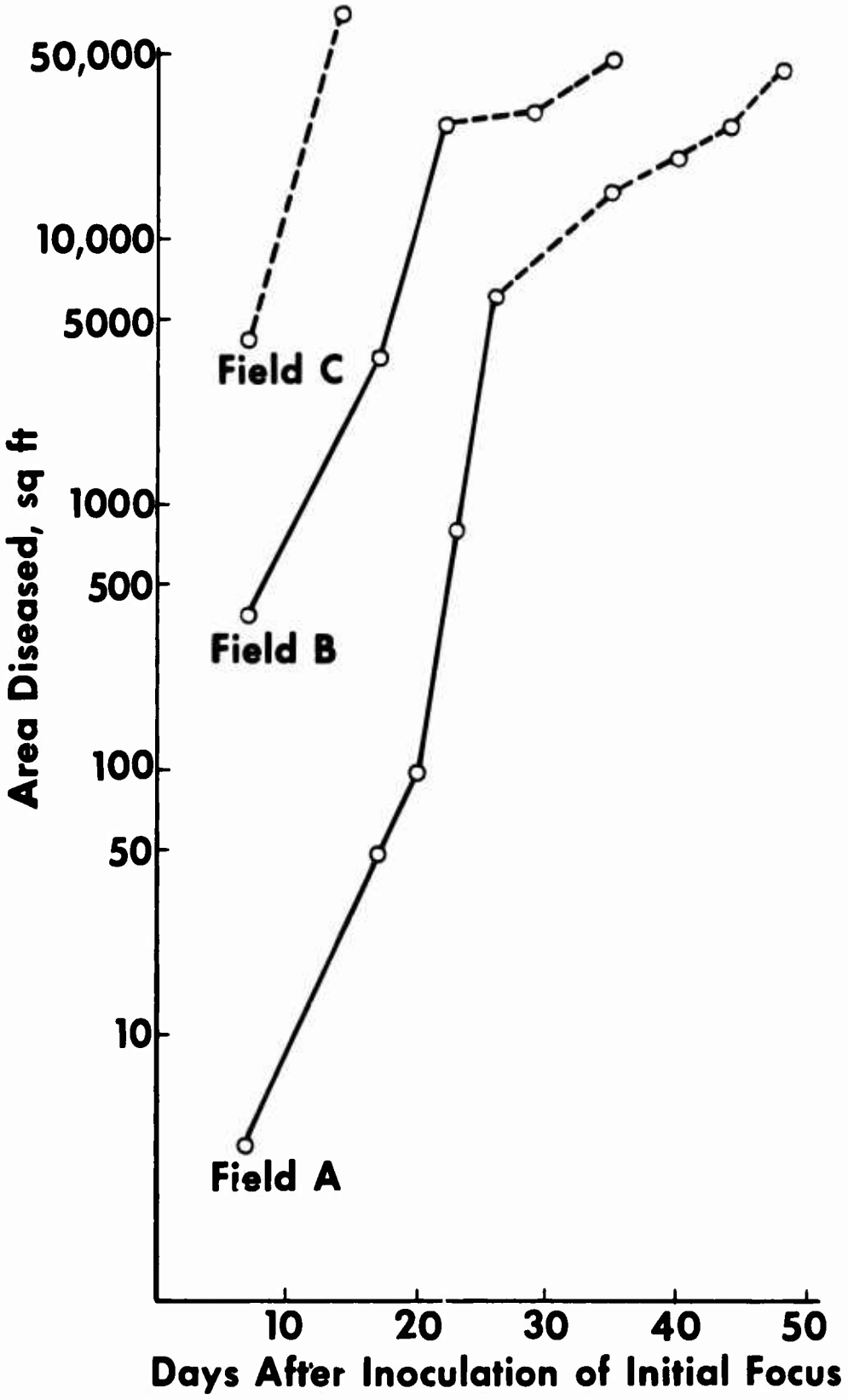


Figure 6. Rate of Disease Spread after Initial Inoculation.

encompassed. The explanation may lie in van der Plank's suggestion,[2] made during a discussion of the distant dispersal phase for potato late blight, that disease may develop "in a large number of small foci close enough together to give the general impression of uniformity." This explanation is enhanced if one accepts his proposition that growth of a population of foci is logarithmic, i.e., foci do not overlap, until x becomes greater than 0.05 in a field as a whole. Field A did not reach the 0.05 level. Field B reached the 0.05 level after 45 days, and Field C after 17 days. The data in Figure 6 therefore, may show increase in many small foci outside the initial foci rather than increased size of these initial foci; that is to say that the recognition of small areas of disease as foci depends on the observation and note-taking procedure.

C. RELATIONSHIP BETWEEN DISEASE AND SPORE LOAD IN THE AIR

To compare the amount of disease present in the three fields at any given time, the proportion of tissue affected, x, was averaged for all sampling stations in each field, converted to logs, and plotted against time (Fig. 7). This procedure seems justified because the location of each station represents more or less the same amount of area in each field (except in Field A where the first observations represent a very small area; therefore lines connecting points on the Field A plot are dotted until after day 30). The field with the largest focus and hence the greatest amount of disease initially had the greatest amount of disease throughout the season; the field with the smallest focus initially had the least. Using the average estimate of x in each field, $r = 0.307$ in Field A between days 30 and 48, $r = 0.312$ in Field B between days 11 and 29, and $r = 0.379$ in Field C between days 11 and 31. The average of these rates estimated during rapid epidemic development is 0.3327.

One can imagine that Field C is comparable to an endemic disease situation and that good sanitation practices have removed some of the initial infection from Field B. Based on size of initial area infected and on the observation that initial intensity was similar in Field B and C, the sanitation ratio, x_o/x_{os}, as defined by van der Plank[2] is $0.1a/0.01a = 10$. In Field A, initial intensity was about 25% that in C; 25% of $0.0001a = 0.000025A$. If the sanitation ratio for Field C vs. A is estimated on the basis of initial area infected one gets $0.1a/0.000025a = 4000$. To calculate the theoretical delay in time (Δt) before onset of epidemic development caused by such sanitation we used van der Plank's formula:[2]

$$\Delta t = \frac{2.3}{r} \log_{10} \frac{x_o}{x_{os}}$$

we find that sanitation should have caused a delay in onset of 7 days in Field B and of 25 days in Field A. Choosing rather arbitrarily to compare these estimates with the observed delay when $x = 0.001$ (or $\log x = -3$) in all three fields (Fig. 7), we find a delay of 7 days in Field B and of 34 days in Field A.

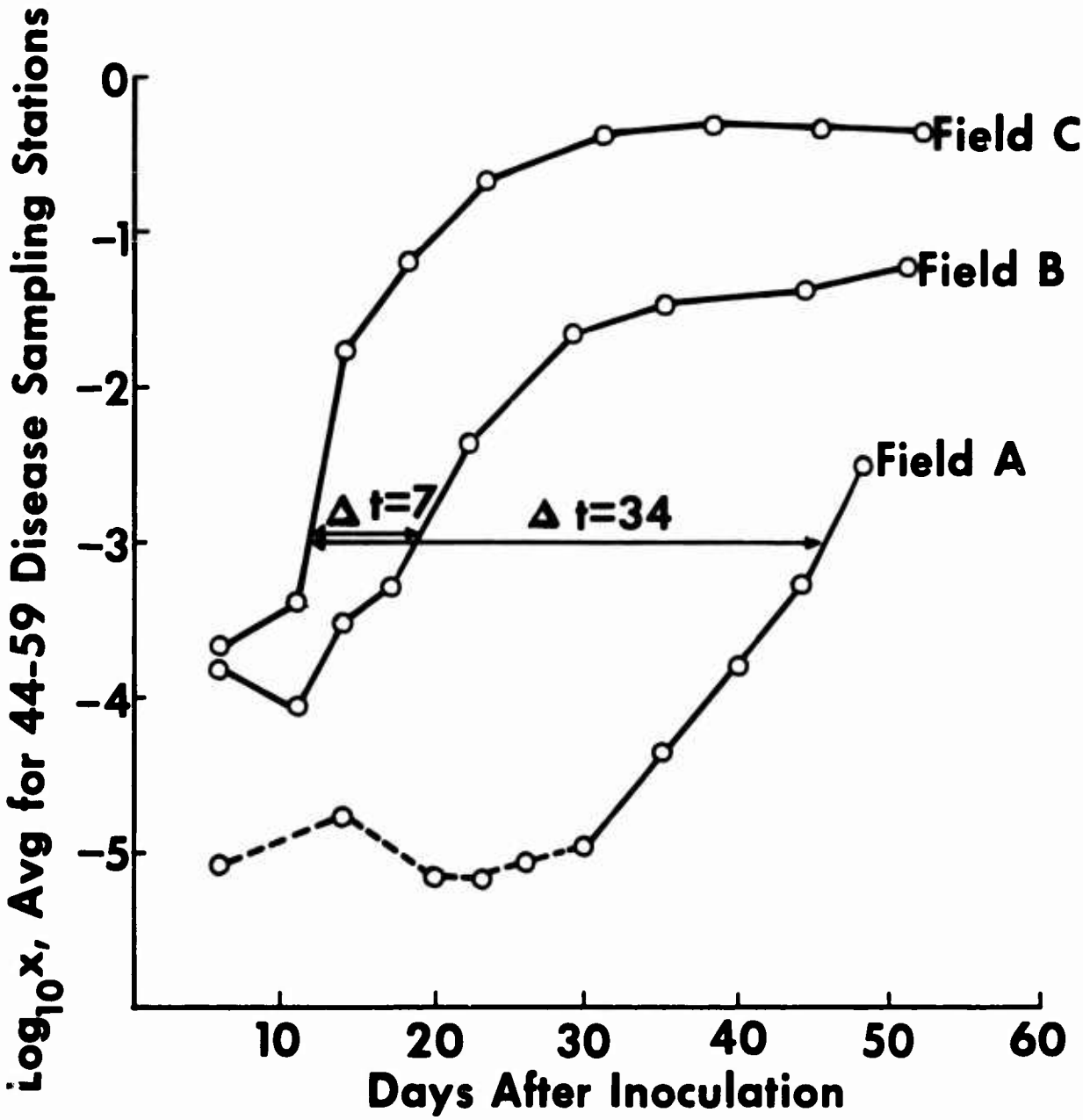


Figure 7. Proportion of Tissue Affected ($\log_{10} x$) in each of the Fields at Various Times.

Daily averages of spore hours/m^3 for nine rotobar stations in each field are plotted on a log scale against time (Fig. 8). As might be expected, the field with the most infected tissue put the greatest spore load into the air. No attempt was made to compute and fit a function to these points. One interesting observation is the rather rapid decrease in spore load associated with older infected tissue after leaf disease stopped increasing rapidly in Fields B and C. This is because young lesions under given environmental conditions sporulate much more heavily than do old lesions.[10]

One fact that has been masked by the previous use of averages to describe what happened in whole fields is the rather wide variation in measured values of x and of spore load at individual sampling stations on a given day in any field. In an attempt to quantitate the relationship between x and spore load, therefore, spore data at a given station were averaged for 3-day periods, the middle day being a day on which x was also measured. Then measurements of x at the given spore sampling station were averaged with three or usually four other measurements of x taken at stations immediately surrounding the spore sampler (Fig. 9). An example of the kind of plots obtained from handling the data in this way is seen in Figure 10. Here, for the rotobar station 75 ft E of center in Field C, a log plot of spore hours/m^3 and of x against time shows that the proportion of diseased tissue and the spore load produced from it increase together. But toward the end of the epidemic, the spore load decreases faster than disease attenuates, as shown by the reverse curve of the plot of spore load against disease on the right side of the figure.

A log plot of all pairs of spore and disease observations while values of both were increasing for all three fields appeared linear. Mrs. Marian W. Jones of our laboratories performed a separate regression analysis on data from each field, and no significant difference among slopes was found. Her analysis of combined data resulted in the following regression:

$$\log Y = 3.57092 + 0.60479 (\log X)$$

where: X = proportion of tissue diseased, and

Y = spore hours/m^3

The standard error of the slope was 0.03852. The correlation coefficient was 0.868; more than 75% of the variation in log (spore hours/m^3) was accounted for by the fitted regression line. Data points, the regression equation, 95% confidence limits for the true regression line, and 95% tolerance limits for individual values are shown in Figure 11. In original units the relationship is expressed as $Y = 3723X^{0.60479}$ (Fig. 12). These equations indicate the sort of observations we are likely to make early and late in an epidemic of rice blast.

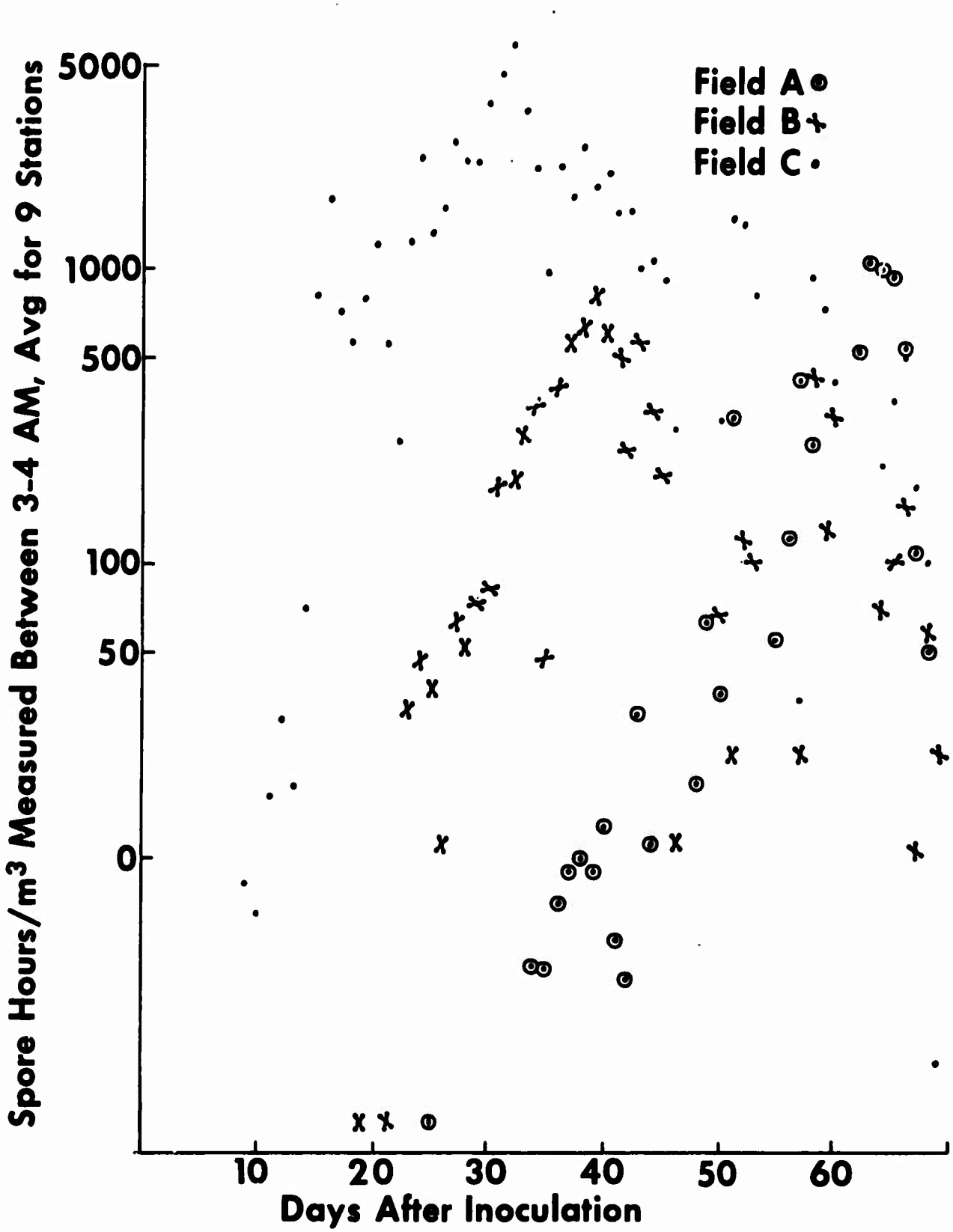


Figure 8. Daily Average Airborne Spore Concentration.

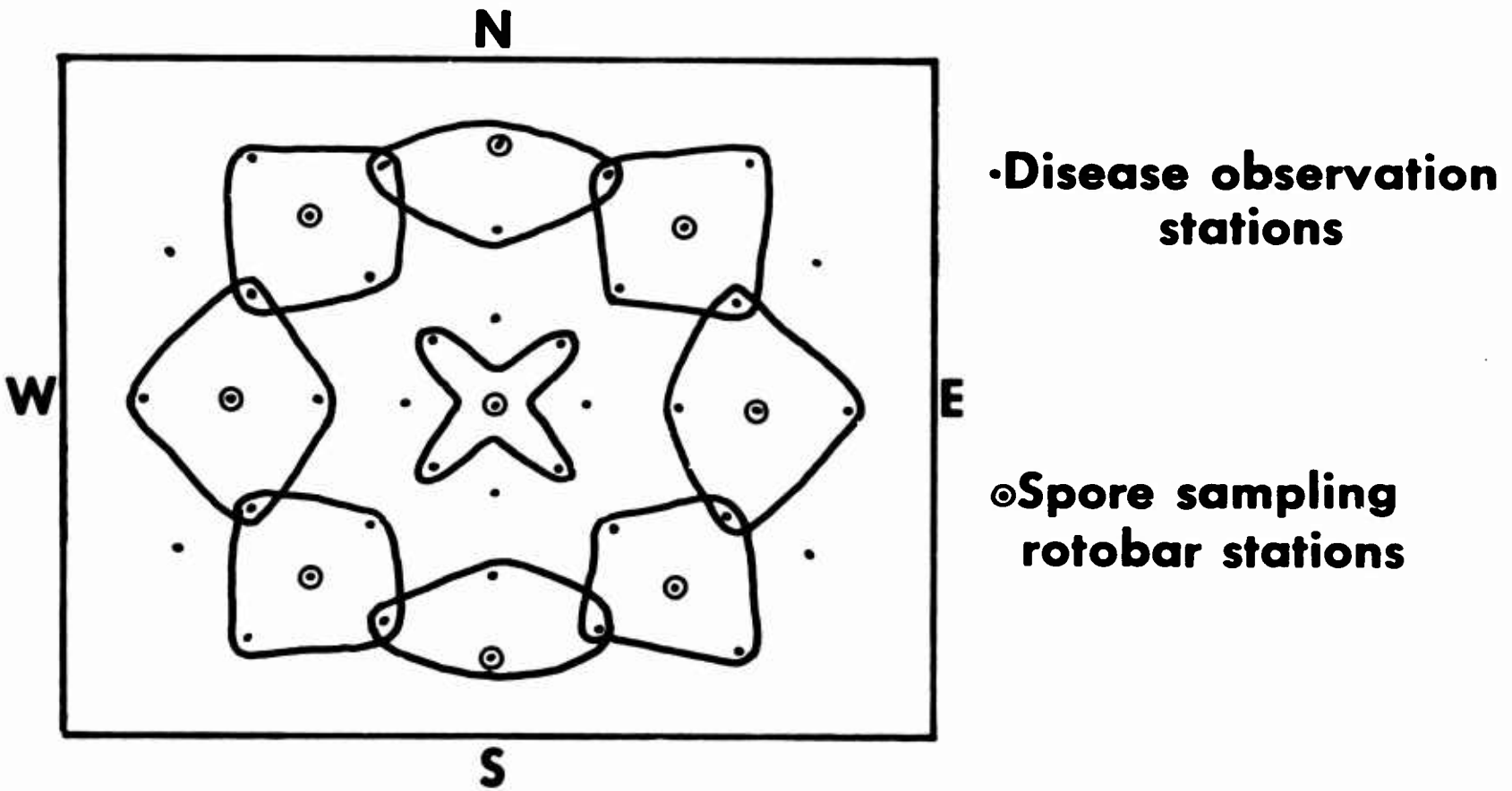
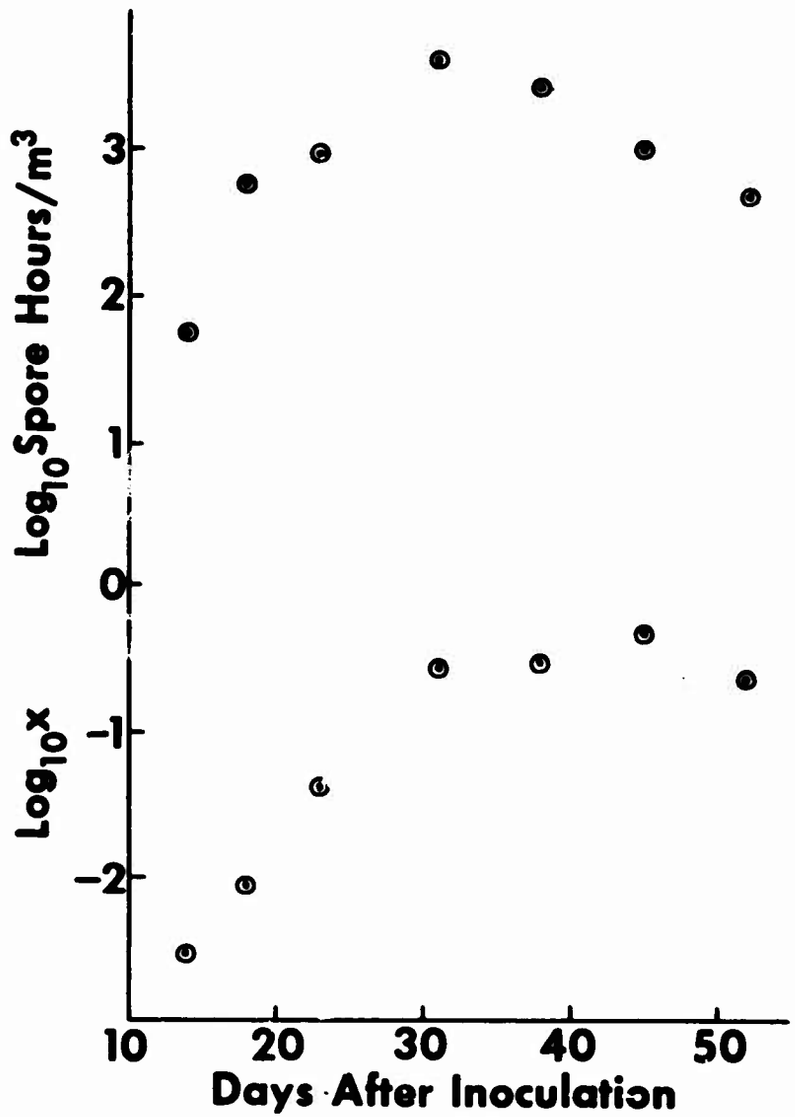


Figure 9. Combinations of Spore- and Disease-Sampling Stations from which Data were Taken for Analysis.



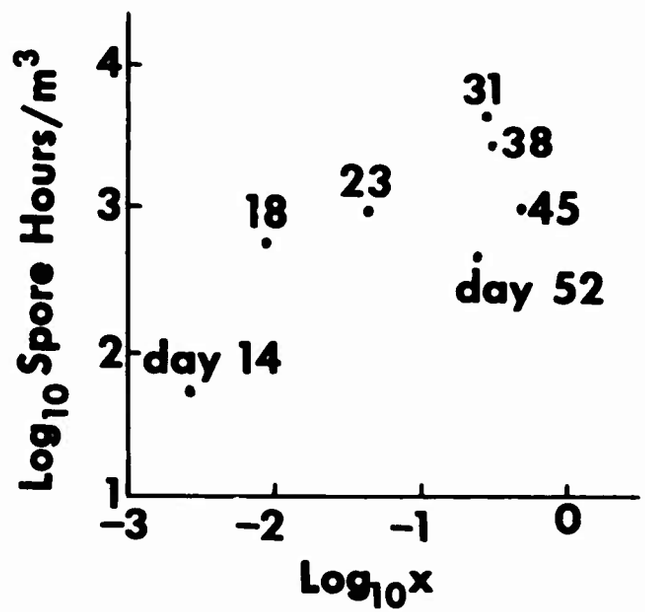


Figure 10. Plots Obtained from the Rotobar Station 75 ft E of Center in Field C.

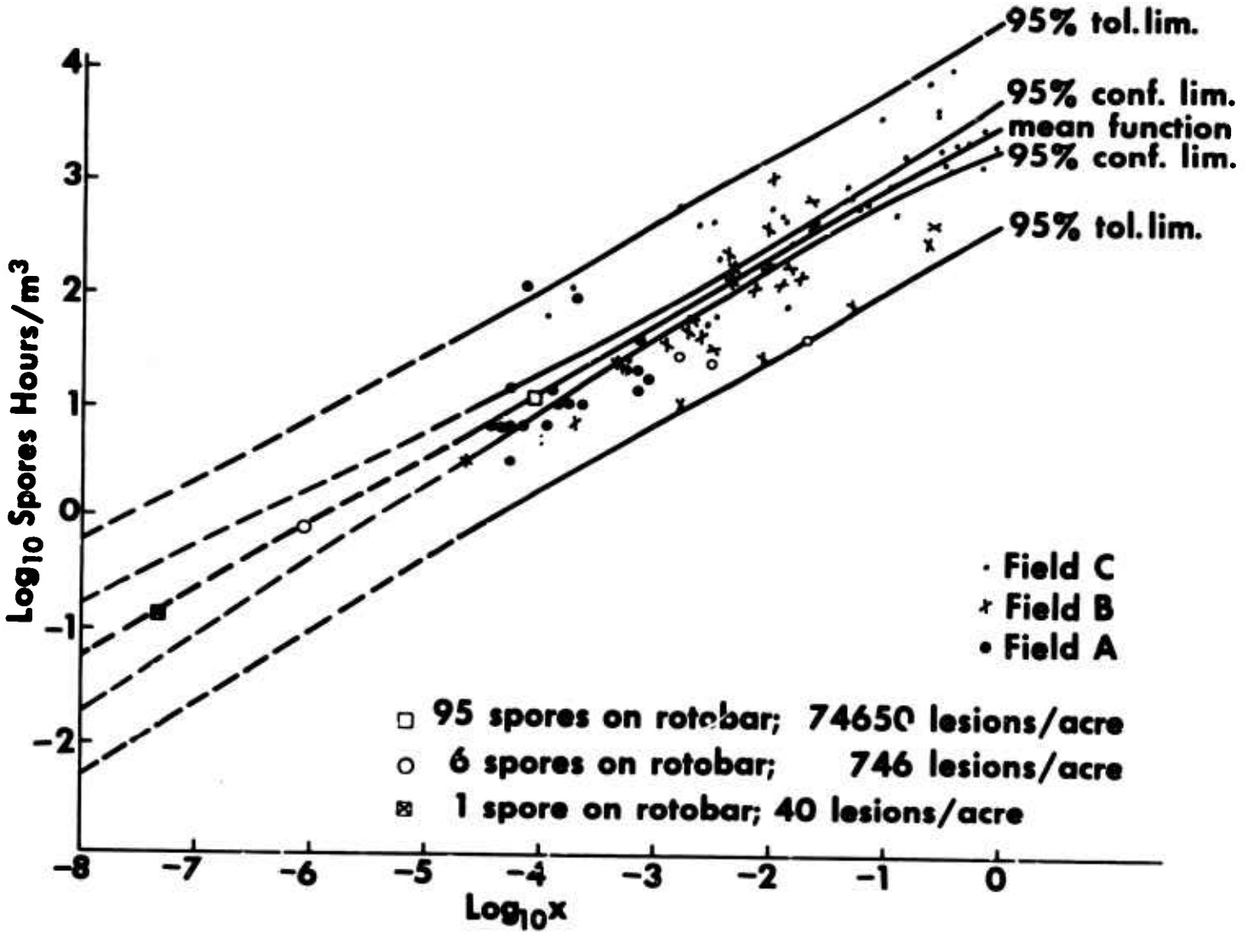


Figure 11. Relationship Between Disease and Spore Load in the Air.

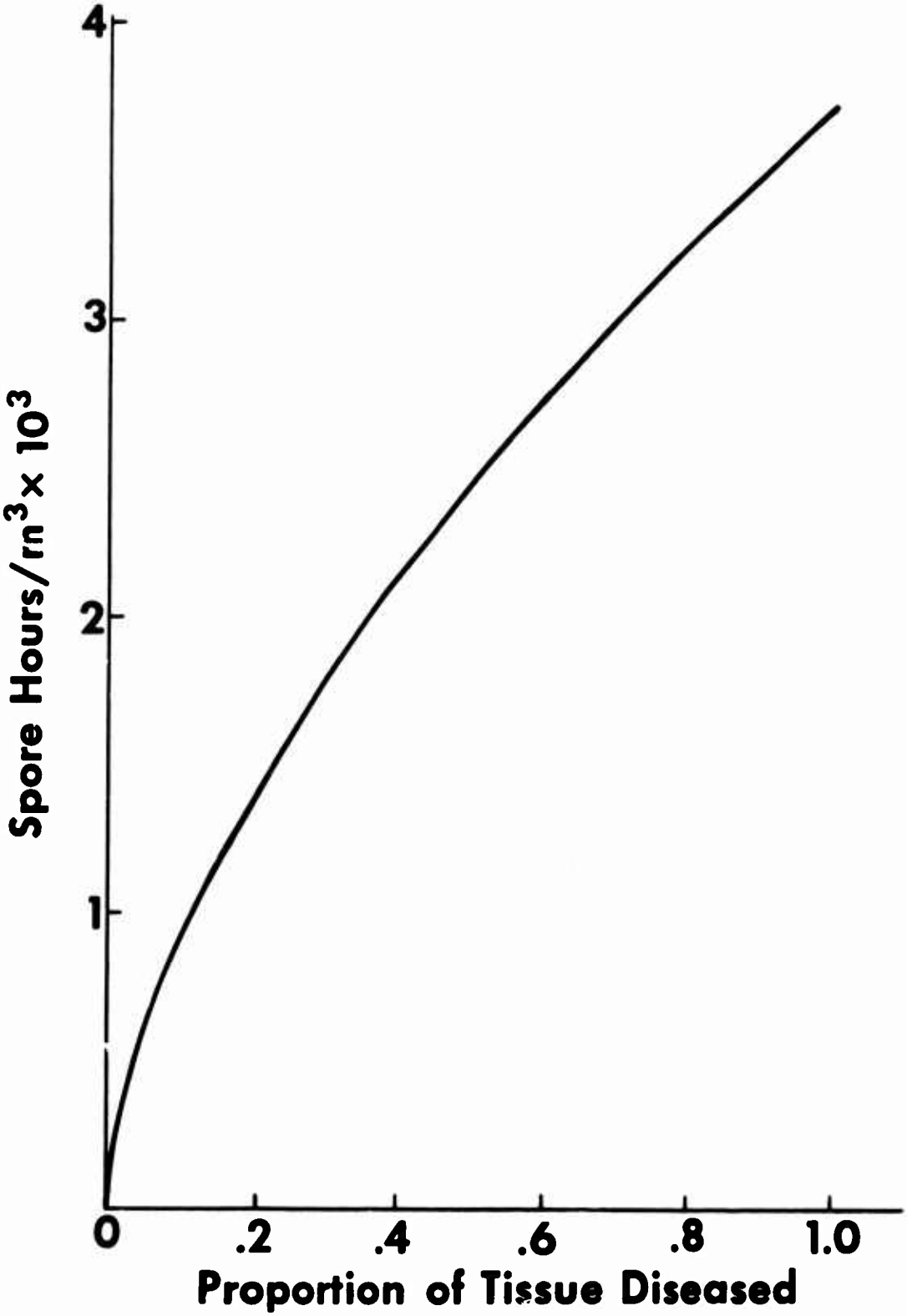


Figure 12. Regression of Spore Hours/m^3 on Proportion of Tissue Diseased, Estimated from Combined Data of Three Fields.

If disease becomes so severe that x = 1, then spore hours/m^3 = 3723. The flow rate of the sampler is 7.2 m^3/hr, and since dosage, D, as defined by Pasquill[11] is known, D = N/S, where D = number of spores and S = sampler flow rate, N can be found. About 26,800 spores would have been observed. Actually, with such high numbers only a fraction of the rotobar need be assessed to obtain quantitative data, but the point is that at such high levels of x no one could fail to detect spores on the sampler.

At the beginning of the epidemic the situation is somewhat different. The estimate that one lesion/linear row ft in the tillering stages gives an x of about 0.000089 would mean that there would be about 74,650 lesions in an acre planted to 7-inch drill spacings. Solving the regression equation at this level of x gives a dosage of 13.23 spore hr/m^3, and then, solving the dosage equation for N, 95 spores would have been observed on the rotobar. A trained plant pathologist should be able to detect both 95 spores on a rotobar and disease in an acre that contains 74,650 lesions. We were able to make observations at somewhat lower values (Fig. 11), but it should be noted that the assessment procedure to detect 95 spores on the entire collecting surface of the bar is much more laborious than that required to determine a number in the thousands. Now, suppose there is only one lesion in every 100 linear ft of row, x = 0.00000089, and there are 746 lesions in an acre; D = 0.8156 and about 6 spores would be observed on the bar. This is an area of supposition, and dotted lines on Figure 11 extend the mean function and confidence limits into the area where data are lacking. It would be difficult to detect 746 lesions in an acre, but probably more difficult to detect 6 spores on the rotobar. Another way of looking at the problem is that one cannot see less than one spore [1 spore/(7.2m^3/hr) = 0.1388 spore hr/m^3]. Such a dosage would be found over a field with an x of about 0.0000000476, or about 40 lesions per acre. In other words, very early in a blast epidemic, when through careful observation one might detect between 1 and 39 lesions in an acre, there would be very little chance of detecting a spore on a rotobar. Of course, one could increase the detection chance by using more samplers and/or by running them for periods longer than an hour, but the assessment problem in terms of man-hours very quickly becomes enormous.

IV. DISCUSSION

The meteorological conditions prevailing during the experiment were thought nearly optimum for disease development, and x did approach 1.0 in parts of Fields B and C. Based on a function we previously derived to describe minimum values of dew period and temperature required for infection[12] conditions on about 93% of the nights would have allowed some infection to occur, or, in other words, conditions were thought to limit infection on only two nights each month. Both

sporulation on lesions and infections are influenced by weather,[3,8] but with many hours of near 100% RH and minimum temperatures seldom less than 70 F on most nights, it is little wonder that so much of the variation in spore load measured in the air can be explained by the amount of diseased tissue in the field.

When disease and spore load are both increasing, measurements of the latter have a certain prediction value, i.e., the spores coming off a diseased crop today are those available for causing new infections that appear about 6 days later. On the other hand, it seems likely from the observations and theoretical calculations reported here that disease in terms of 40 to several hundred lesions per acre will occur in a rice field before the chances become very good of detecting Piricularia spores on a rotobar sampler. Here is a concrete example of Hirst's idea[13] that an acre of susceptible crop during weather favorable to infection is better able to detect spores than any trap or sampler now in use.

Following epidemic blast development, one often observes a period of recovery that occurs between the tillering stage and panicle exsertion (Fig. 2, 7, 10). The complicated effects of plant age and nitrogen fertilization practices on plant susceptibility[3,8,14-16] seem to be the chief reasons for the decline in new infections during the period, while many lesions formed early in the epidemic disappear because they were on leaves that have disintegrated either because of severe disease or because of the eventual death of any rice leaf in whose axil a new tiller forms. The net result is that one often observes a reduction in the proportion of tissue affected.

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13. ABSTRACT

Increase and concurrent spread of rice blast from foci in small fields is described under nearly ideal conditions for infection. Lesion counts and severity estimates were made at stations on polar coordinates at regular intervals. Spore loads in the air were measured with rotobars uniformly spaced around the foci and corrected to spore hours per cubic meter. Increase within foci, spread from foci, and relationship between disease and spore load in the air are shown. Lesions were present six days after inoculation. Growth of foci in all fields was similar and logarithmic notwithstanding initial size. Toward the end of the epiphytotic, spore load decreased faster than disease attenuated. In early stages of an epiphytotic, when 40 or fewer lesions occur per acre, there is little chance of detecting spores on a rotobar.

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