Direct and Indirect Effects of Nitrogen Supply and Disease Source Structure on Rice Sheath Blight Spread

S. Savary, N. P. Castilla, F. A. Elazegui, C. G. McLaren, M. A. Ynalvez, and P. S. Teng

First author: IRRI-ORSTOM Project on Characterization of Rice Pest Constraints, International Rice Research Institute, P.O. Box 933, 1099 Manila, Philippines, and ORSTOM, 911 avenue Agropolis, B.P. 5045, 34032 Montpellier cedex France; second, third, and sixth authors: Entomology and Plant Pathology Department; fourth and fifth authors: Biometrics Unit, International Rice Research Institute, P.O. Box 933, 1099 Manila, Philippines.

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ABSTRACT

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Effects of nitrogen and type of disease source on focal expansion of rice sheath blight (caused by *Rhizoctonia solani*) were studied in two field experiments. Four disease source types, represented by inoculated individual hills at the center of experimental plots, were obtained by combining two inoculum doses with two heights of inoculation in the canopy. Compared with the low inoculum dose, there was a small but significant increase of disease severity on source hills with the higher inoculum dose, and no disease developed on noninoculated controls. Severity on the sheaths was higher on hills inoculated at the sheath level than on hills inoculated at the leaf level. Conversely, leaf severity was higher on hills inoculated at the leaf level than on hills inoculated at the sheath level. Increased nitrogen supply increased host plant tissue contacts (leaf-to-leaf and leaf-to-sheath), increased the capacity of the canopy to retain moisture, and increased the leaf nitrogen content in both experiments. The size of sheath blight foci was much larger in the rainy (3,000 to 30,000 cm^2) than in the dry (250 to 1,100 cm^2) season. In both experiments, foci expanded most rapidly in the high nitrogen supply (120 kg per ha) level compared with medium (80 kg per ha) and no nitrogen supply. Positioning of inoculum in the upper layer of the canopy (leaf level) on source hills resulted in faster spread of foci than positioning in the lower layer (sheath level). Multiple regression and path coefficient analyses suggested that nitrogen drives focal expansion in sheath blight essentially via indirect effects: increased tissue contacts in the canopy and higher leaf wetness.

Rice sheath blight, caused by *Rhizoctonia solani* Kühn, anastomosis group 1 (7,22) is an increasing concern for rice production in South and Southeast Asia, especially in intensified production systems. These intensified systems rely heavily on the use of high-yielding cultivars, high crop densities, and high rate of fertilizer, especially nitrogen. All these factors have been considered in more or less detail as contributing to increased disease problems (7,21,22).

There are relatively few reports that specifically indicate that sheath blight can be reduced with resistant cultivars (22). The pathogen has an extremely wide host range, and no source of complete resistance to the pathogen has been found so far (7,22). To a large extent, the linkage between increased disease intensity and the use of high-yielding varieties seems to be attributable to corresponding changes in cropping practices and habitat for the pathogen.

High crop density, either through increased sowing rate in direct-seeded rice (20) or higher planting density in transplanted rice (16) has long been advocated as a major factor favoring increase of sheath blight. In Japan, the increased prevalence of the disease over the period 1955 to 1975 is largely attributed to increased planting density (21).

Reports of increased disease intensity associated with increased nitrogen fertilizer supply are also numerous (e.g., 13,15). Like stand density, increased growth caused by higher nitrogen supply results in denser and more humid canopies, and more intercepted light early in the season. Direct changes in the host's susceptibility with higher nitrogen supply have also been postulated, but this is still controversial (13,36,37).

Separating direct and indirect effects of nitrogen supply is difficult. Their influence on the dynamics of the pathogen via crop growth, crop physiology, and crop microclimate are confounded. While these difficulties are common in epidemiological field studies, they are compounded in the case of rice sheath blight by the influence of canopy growth on disease spread and the difficulty of accurately measuring the disease.

The objective of this study was to develop techniques to describe quantitatively the spread and increase of sheath blight epidemics, to evaluate how sheath blight is affected by nitrogen supply, and to assess hypotheses to explain the epidemiological response of sheath blight to nitrogen.

MATERIALS AND METHODS

Crop establishment. Experiments were established at the International Rice Research Institute, Philippines, during a rainy (experiment 1) and during a dry (experiment 2) season. Nurseries of IR72, a short-cycle, high-yielding rice cultivar, were established on 15 July 1992 and 29 January 1993 for experiments 1 and 2, respectively. Seedlings were transplanted at a rate of 6 to 8 per hill, with a spacing of 20 cm between hills within rows and 20 cm between rows.

Inoculum. A *Rhizoctonia solani* anastomosis group AG1-1a isolate obtained from infected rice was maintained in culture tubes containing potato-dextrose agar medium. Prior to inoculation, the fungus was grown in petri dishes containing the same medium and incubated for 10 days at room temperature (20 to 27°C). Plugs of growing hyphae were then transferred to bottles containing an autoclaved substrate consisting of rice grain/rice

hull mixture (1:5, wt/wt) (26) and incubated at room temperature for 2 weeks.

Experimental design. Both experiments had a split-plot design with eight replications, in which nitrogen levels represented the whole plots and inoculation treatments represented the subplots. The individual subplot size was 3.4 × 3.4 m (17 × 17 hills). Within a main plot, subplots were separated from each other by a three-hill-wide border, and main plots by a 0.8-m path including a levee. Three nitrogen treatments were used: N_0 (no nitrogen supply), N_1 (40 kg per ha nitrogen as urea applied at seedling stage and 40 kg per ha nitrogen applied at maximum tillering), and N_2 (as N_1, with an additional 40 kg per ha nitrogen applied when plants were in the boot stage).

Five inoculation treatments were introduced by varying the canopy position where inoculum was placed and the amount of colonized substrate. Treatment S_5 was 5 g of colonized substrate applied at sheath level, L_5 was 5 g at leaf level, S_{15} was 15 g at sheath level, L_{15} was 15 g at leaf level, and C was a noninoculated control. Inoculations were done at 50 days after transplanting (i.e., at maximum tillering/panicle initiation). Prior to inoculation, the central hill of each plot was tagged. Inoculation (26) consisted in inserting in a tagged rice hill a specified amount of the rice grain/rice hull mixture colonized by the sheath blight pathogen.

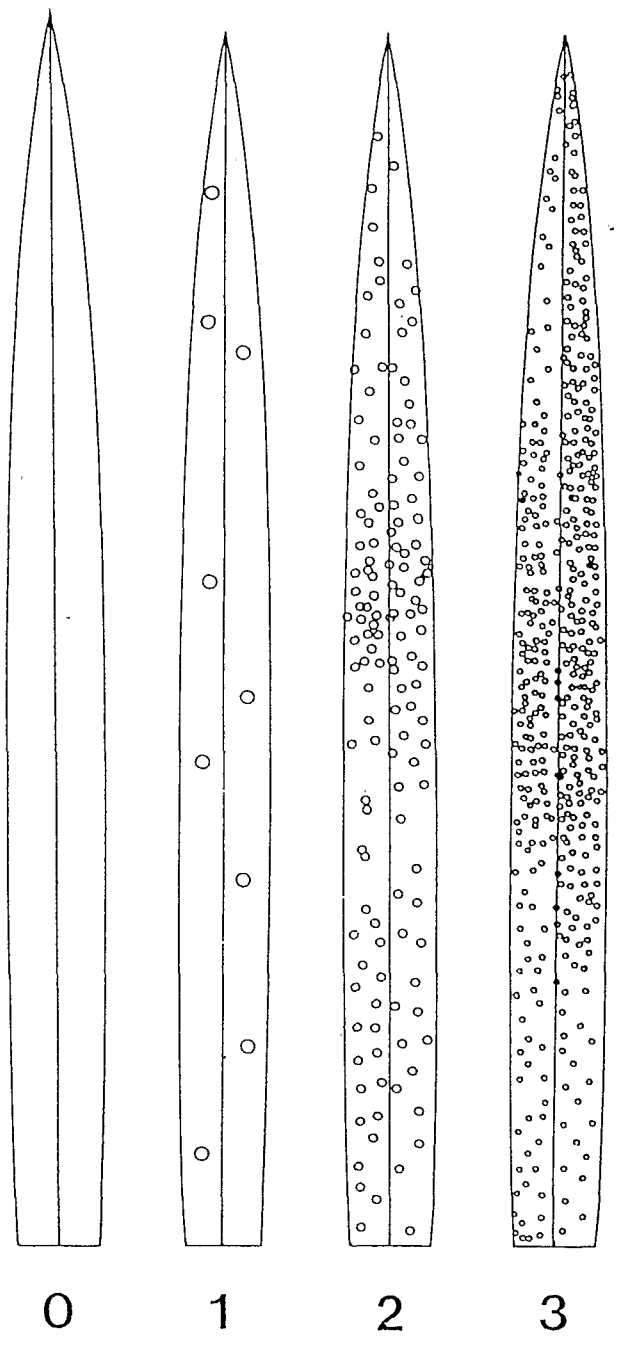

Fig. 1. Diagrammatic scale used to assess leaf wetness of the canopy.

Disease measurement. The establishment of disease in a subplot was assessed as the percentage of sheath and leaf area covered by sheath blight lesions on five tillers chosen at random on the inoculated hill at its center 12 days after inoculation. The shape of a sheath blight focus was assumed to be elliptical, and its area calculated as $a = \pi (x \cdot y) / 4$, where x and y are the length and width of the focus, respectively. The variables x and y were measured as the distance (cm) between the two most distal lesions in a subplot, on both sides of an inoculated hill within and across rows, respectively. Focal area, a, was assessed six times throughout the cropping season, starting 12 days after inoculation. A mean rate of increase of focal area (R) over the six assessments was calculated as

$$R = 1/(n-1) \left[\sum_{i=1}^{n-1} (a_{i+1} - a_i) / (t_{i+1} - t_i) \right]$$

where $n = 6$ is the total number of observations, i the current observation number, and t the time since inoculation.

Host and environmental factors. Leaf wetness was assessed on nine mornings (6 a.m.) during the cropping season with a visual four-point rating scale (Fig. 1). On each morning observations were made for three layers of the crop canopy on each of ten hills chosen at random in each main plot. An average wetness rating w_n was calculated for each of the nine assessment dates as the average score over 10 hills and three layers in each whole plot (nitrogen treatment). The variable w_n was taken as a measure of the potential of a canopy to retain moisture before it evaporates. Canopy wetness was considered to reflect both this potential and rainfall patterns prevailing in the two experiments. The two seasons differed so much in rainfalls that the product $W = w \times r$, where r is the mean daily rainfall during an experiment, and w is the mean of nine average wetness ratings, was used to produce a simplified representation of canopy wetness throughout the cropping season.

Counts of leaf-to-leaf and leaf-to-sheath contacts between neighboring plants were made at three development stages of the crop: panicle initiation, booting, and ripening. One hill was chosen at random in each subplot, and counts were made from visual observation of such contacts between this hill and its eight neighbors. Areas under leaf-to-leaf and leaf-to-sheath contact progress curves were calculated, as well as areas under cumulative (leaf-to-leaf and leaf-to-sheath) contacts (C).

The nitrogen contents of rice leaves were measured at the same development stages as were the tissue contacts. One hill was sampled at random in the noninoculated plot of each main plot (nitrogen level) in each block. The nitrogen concentration of the leaves was determined with the Kjeldahl mineralization method (33). Areas under leaf nitrogen content progress curves (N) were computed.

Data analysis. Sheath blight severities on the leaves (l) and on the sheaths (s) on the source hills at 12 days after inoculation were analyzed with analysis of variance (14,24). The subplot factor, inoculation, was partitioned into effects of inoculum amount and height (8,14). Analyses of variance of the area of foci (a) were done for each observation date in both experiments. The average leaf wetness data (w) per observation date were analyzed across nitrogen levels with a one-way analysis of variance procedure. Log-transformed areas under leaf-to-leaf and leaf-to-sheath contact progress curves were also analyzed using a split-plot design. The leaf nitrogen content was analyzed in both experiments using a one-way analysis of variance across nitrogen levels.

Five variables—N, C, W, s, and l (Table 1)—were used to describe variation of focal expansion rates (R) in inoculated plots, in

a stepwise, forward-selection, regression analysis. The regressions were performed on subplot means averaged over the eight replications (8), with data combined from the two experiments. The dependent variable was focal expansion rate transformed as natural logarithm (ln $(R + 1)$) prior to regression to reduce variance heterogeneity (6). Several regression models with predetermined combinations of predictors (*W, C, s* and *l; W* and *C; s* and *l; N, W* and *C; N, s* and *l*) were also tested. The resulting equations were assessed by aptness of residual plot, coefficient of determination, and *F* test (25).

Correlation analysis indicated colinearity among predictors. Path coefficient analysis (2,5,9,12,29–31) was used to assess the direct and indirect contributions of multiple, often correlated factors, on focal expansion. Results provided by this technique included (i) a descriptive synthesis of regression equations, and (ii) determination of direct and indirect effects of experimental factors.

RESULTS

Disease intensity on source hills. Mean sheath blight severity on the leaves was higher on source hills inoculated at the leaf level (for L_5 and L_{15}, 34 ± 2 and 47 ± 2%, respectively, in experiment 1, and 24 ± 2 and 31 ± 3%, respectively, in experiment 2) than on source hills inoculated at sheath level (for S_5 and S_{15}, 17 ± 2 and 23 ± 2%, respectively, in experiment 1, and 13 ± 1 and 19 ± 2%, respectively, in experiment 2). Disease severity on the sheaths was higher on the hills inoculated at sheath level (for S_5 and S_{15}, 16 ± 2 and 18 ± 2%, respectively, in experiment 1, and 7 ± 1% and 10 ± 2%, respectively in experiment 2) than on hills inoculated at leaf level (for L_5 and L_{15}, 3 ± 1 and 9 ± 2%, respectively, in experiment 1, and 6 ± 1 and 6 ± 1%, respectively, in experiment 2). Severity of sheath blight increased significantly with increasing amounts of inoculum (Table 2). There was also a small but significant increase of severity on the sheath with increased inoculum amounts. The noninoculated controls had small to negligible disease intensities on the leaves (0% in both experiments) and the sheaths (0.2% in experiment 1, 0% in experiment 2).

Nitrogen had no effect on sheath blight severity on the inoculated source hills in experiment 1, while severity on the sheaths (N_0 = 4 ± 1%, N_1 = 5 ± 1%, N_2 = 8 ±1%) and leaves (N_0 = 14 ± 2%, N_1 = 20 ± 2%, N_2 = 19 ± 2%) was increased with increasing nitrogen in the second experiment.

Plant tissue contacts. The analysis of the (log-transformed) area under leaf-to-leaf contact progress curves indicated a significant effect of nitrogen ($P < 0.01$). Contacts strongly increased with increased nitrogen (N_0 = 605 ± 46, N_1 = 1,625 ± 68, N_2 = 1,816 ± 63 contacts per day in experiment 1; N_0 = 267 ± 23, N_1 = 1,205 ± 49, N_2 = 1,324 ± 44 contacts per day in experiment 2). Compared with the number in inoculated subplots, leaf-to-leaf contacts were slightly more frequent in the noninoculated controls of experiment 2 (C = 1,037 ± 122, L_5 = 841 ± 93, S_5 = 944 ± 113, L_{15} = 889 ± 112, S_{15} = 948 ± 111 contacts per day; $P < 0.01$), probably because of the growth-reducing effect of the disease. Similar results were obtained for leaf-to-sheath contact curves, with a strong nitrogen effect (N_0 = 118 ± 11, N_1 = 271 ± 19, N_2 = 219 ± 14 contacts per day in experiment 1; N_0 = 72 ± 11, N_1 = 189 ± 12, N_2 = 191 ± 12 contacts per day in experiment 2) which was significant in both experiments ($P < 0.01$). Leaf-to-sheath contacts were slightly less frequent in some of the inoculation treatments of experiment 2, probably due to reduction in canopy growth by disease in these treatments (C = 193 ± 21, L_5 = 139 ± 14, S_5 = 149 ± 18, L_{15} = 121 ± 16, S_{15} = 151 ± 18 contacts per day; $P < 0.01$). There was a significant variation ($P < 0.01$) in both leaf-to-leaf and leaf-to-sheath contacts among replications in experiment 1 due to heterogeneity in crop growth over the experimental area.

Leaf wetness. In both experiments, the nitrogen treatments resulted in significant differences in mean leaf wetness ratings at 6 a.m. (w) averaged over 9 observations (N_0 = 1.38 ± 0.12, N_1 = 1.70 ± 0.11, N_2 = 1.78 ± 0.10, $P < 0.05$, in experiment 1; N_0 = 1.61 ± 0.11, N_1 = 2.11 ± 0.14, N_2 = 2.38 ± 0.07, $P < 0.01$, in experiment 2). There were strong differences between the two experimental seasons in accumulated rainfall during the observation periods (424.5 mm in 35 days in experiment 1; 19.9 mm in six days in experiment 2). These rainfall differences, combined with differences in w, resulted in contrasted values of leaf wetness indices (W), ranging from 0.58 (N_0, experiment 2) to 13.27 (N_2, experiment 1).

Leaf nitrogen. Significant differences were observed in areas under leaf nitrogen content curves (N) among the three nitrogen treatments ($P < 0.01$ in both experiments). First experiment means: N_0 = 70.3 ± 1.8, N_1 = 77.2 ± 1.8, N_2 = 85.6 ± 4.0% per day; second experiment means: N_0 = 65.0 ± 1.6, N_1 = 65.0 ± 2.0, N_2 = 75.0 ± 2.5% per day.

Focal expansion. Focal expansion was strongly influenced by nitrogen treatment in the first experiment (Fig. 2). Focal expansion was slowest in N_0, in which focal area only attained a maximum 3,000 cm^2. In contrast, focal area attained 30,000 cm^2 in N_2. In this experiment, L_{15} resulted in the fastest rate of focal expansion in all nitrogen treatments. Generally, a faster rate of spread was observed at the higher inoculum doses (S_{15} and L_{15}) than at lower doses (S_5 and L_5), except in N_2, in which L_5, S_5, and S_{15} resulted in similar curves. Analyses of variance (Table 3) confirmed the significant nitrogen effect throughout the observation period. The contrast of inoculation versus noninoculation also was significant, suggesting that the background level of infection remained low during the course of the experiment. The effect of height of inoculum positioning (L or S) resulted in more variation in rates of focal expansion than did amount of inoculum (5 g or 15 g).

TABLE 1. List of variables used to analyze relationships between rate of rice sheath blight spread and environmental, crop growth, and initial disease severity variables

Symbol	Explanation	Units
a	Focus area	cm^2
R	Mean rate of disease spread	cm^2 · day^{-1}
LR	Log-transformed mean rate of disease spread: Log $(R + 1)$	(–)
N	Area under leaf nitrogen content curve	% · day
w	Mean leaf wetness of the canopy at 6 a.m.	(–)
W	Overall wetness of the canopy during the cropping season	(–)
C	Area under the curve of accumulated number of contacts between hills	contact · day
s	Initial sheath blight severity on the sheaths of source plant	%
I	Initial sheath blight severity on the leaves of source plant	%

TABLE 2. Comparison of effects of inoculation treatments on sheath blight severity on the leaves and the sheaths of inoculated rice plants 12 days after inoculation[a]

		Sheath blight severity (%)					
		Inoculum position			Inoculum amount		
Plant tissue	Experiment[b]	Leaf	Sheath	LSD[c]	5 g	15 g	LSD
Leaves	1	40.5	20.0	3.55	25.5	35.0	3.55
Leaves	2	27.5	16.1	3.25	18.8	24.8	3.25
Sheath	1	6.2	17.0	3.35	9.5	13.7	3.35
Sheath	2	5.4	8.8	2.02	6.0	8.3	2.02

[a] Inoculation treatments were combinations of two inoculation heights (at leaf or sheath level) and two doses (5 or 15 g of rice grain/rice hull mixture colonized by *Rhizoctonia solani*.

[b] Experiments 1 and 2 were conducted in the rainy and the dry season, respectively.

[c] Least significant difference ($P < 0.05$).

In the second experiment, sheath blight foci had very low rates of expansion in treatment N_0 (Fig. 3). In N_1 and N_2, inoculation treatments L_5 and L_{15} resulted in greater focal areas than S_5 and S_{15}. In some cases (S_5 and S_{15} in N_2, and L_{15} in N_1), focal area declined in the period just before the last assessment date. In general, however, L_{15} corresponded to the largest maximum focal size in N_1 and N_2. The analyses of variance (Table 3) over successive observation dates showed a significant effect of nitrogen over the entire observation period, and a significant contrast between inoculated versus controls was indicated. The effect of inoculum position on the source hills on focal size was significant on all assessment dates, while the effect of inoculum amount was significant only early in the experiment.

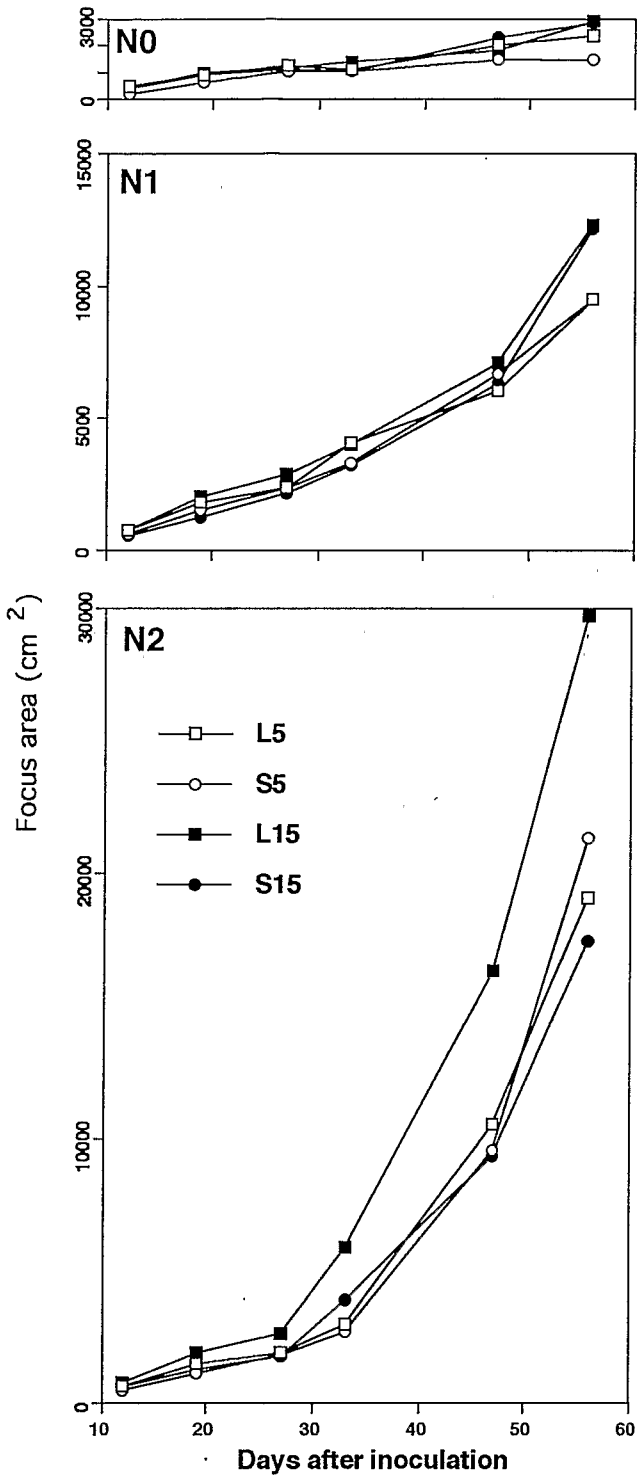

Fig. 2. Focal expansion of rice sheath blight in an experiment conducted in the rainy season using a split-plot design with eight replications. Whole plots were represented by three nitrogen supply treatments: N_0 (no nitrogen supply), N_1 (40 kg per ha nitrogen applied at seedling stage and 40 kg per ha nitrogen applied at maximum tillering), and N_2 (as N_1, with an additional 40 kg per ha nitrogen applied at booting). Inoculations were done at maximum tillering/panicle initiation. Sources were established at the center of sub-plots. S_5 = 5 g of rice grain/hull mixture colonized by *Rhizoctonia solani* placed at sheath level (open circles); L_5 = 5 g of colonized mixture at leaf level (open squares); S_{15} = 15 g of colonized mixture at sheath level (solid circles); L_{15} = 15 g of colonized mixture at leaf level (solid squares). Points represent the mean of eight replications. Noninoculated controls are not shown.

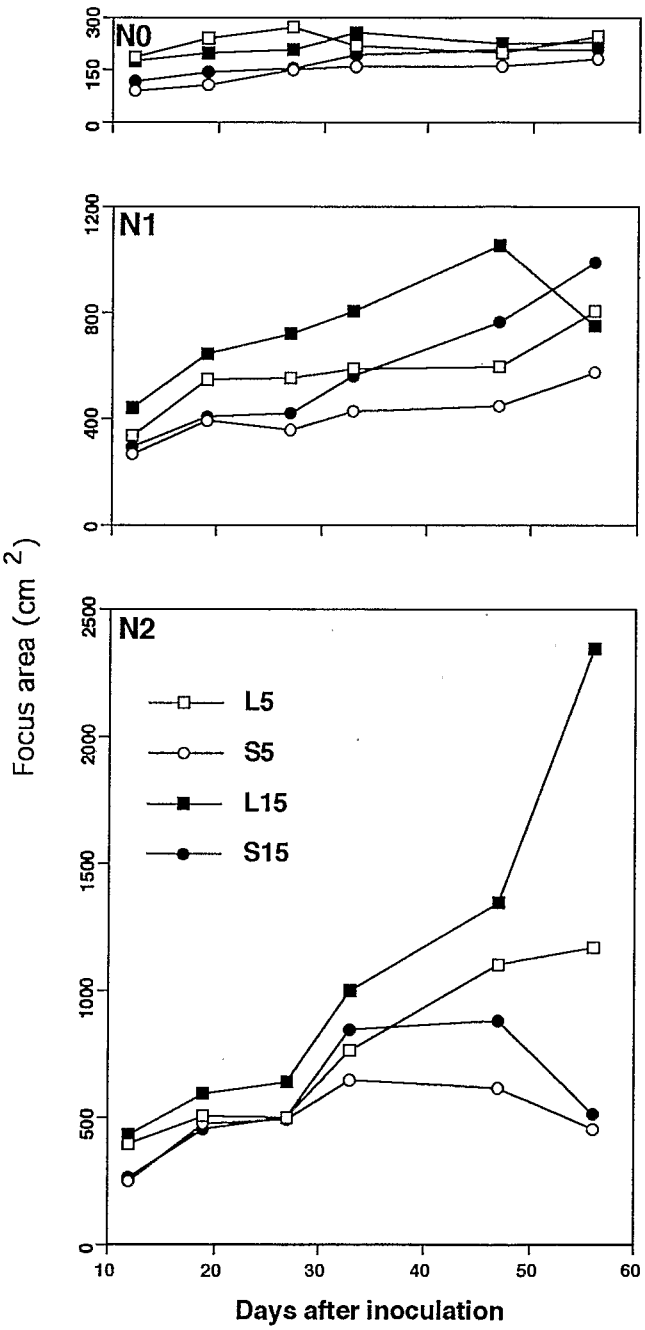

Fig. 3. Focal expansion of rice sheath blight in an experiment conducted in the dry season using a split-plot design with eight replications. Whole plots were represented by three nitrogen supply treatments: N_0 (no nitrogen supply), N_1 (40 kg per ha nitrogen applied at seedling stage and 40 kg per ha nitrogen applied at maximum tillering) and N_2 (as N_1, with an additional 40 kg per ha nitrogen applied at booting). Inoculations were done at maximum tillering/panicle initiation. Sources were established at the center of sub-plots. S_5 = 5 g of rice grain/hull mixture colonized by *Rhizoctonia solani* placed at sheath level (open circles); L_5 = 5 g of colonized mixture at leaf level (open squares); S_{15} = 15 g of colonized mixture at sheath level (solid circles); L_{15} = 15 g of colonized mixture at leaf level (solid squares). Points represent the mean of eight replications. Noninoculated controls are not shown.

Regression and path analysis. Significant colinearity occurred among the variables *l, s, C, W,* and *N* (Table 4). A first regression equation (Table 5) in which all variables were entered into the model accounted for a very large fraction of the variation in *LR* ($R^2 = 0.959$). The contribution of *N* to this first model was, however, not significant ($P = 0.186$). Its removal led to another equation involving *W, C, s,* and *l,* which accounted for a similar variation in *LR* ($R^2 = 0.957$). This second equation was also the outcome of a stepwise, upward-selection procedure involving initially all variables. A regression equation involving *W* and *C* only accounted for a large fraction of variation in *LR* ($R^2 = 0.939$), while a regression equation involving *s* and *l* only accounted for a small fraction ($R^2 = 0.378$). An analysis involving *N, W,* and *C* indicated that consideration of *N* in combination with *W* and *C* was superfluous in describing the data. Conversely, replacing *W* and *C* by *N* in the second equation (and involving *N, l,* and *s* simultaneously, $R^2 = 0.870$) did not improve data description.

Path coefficient analysis partitioned correlation among variables into direct and indirect effects on sheath blight focal expansion (Fig. 4). This analysis identified two groups of variables affecting *LR* in the combined data set: environment-related and crop growth–related variables (*C* and *W*), which are affected to a large degree by nitrogen treatments and initial disease variables (*s* and *l*), reflecting the inoculation treatments. Strong direct effects of *W* and *C* on *LR* were indicated. The direct effect of *N* on *LR* was marginal (as shown in Table 5), but its effects on *W* and *C* were strong and significant ($P < 0.0001$). The effect of *s* on *LR* was weaker than that of *l*, but the two variables showed some degree of colinearity, indicating that *s* affected *LR* partly via *l* (and vice versa). *U* represents all undetermined influences on *LR*.

Path coefficient analyses using the same framework led to similar conclusions when experiments 1 and 2 were considered separately. The analyses yielded significant ($P < 0.05$) path coefficients between *W* and *LR* (0.235 and 0.335 for experiments 1 and 2, respectively) and between *C* and *LR* (0.554 and 0.157), while path coefficients between *N* and *LR* were not significant. The path coefficients between *l* and *LR* (0.106 and 0.211) were larger than those between *s* and *LR* (0.012 and 0.039).

DISCUSSION

Crop growth, as measured by frequency of contacts between plant organs, was greater in the second (dry season) than in the first (rainy season) experiment; this was likely due to differences in the amount of radiation received by each crop. Differences in crop growth among experiments, however, were small relative to within-experiment variation in both leaf-to-leaf (a threefold to fourfold increase) and leaf-to-sheath contacts (a twofold to threefold increase in both experiments) across the range of nitrogen treatments employed. The nitrogen treatments also resulted in small, but significant, differences in the nitrogen content of the leaves (an increase of 17.8 and 13.4% from N_0 to N_2 in experiments 1 and 2, respectively). Additionally, increased nitrogen supply to the crop resulted in an increased capacity of the canopy to retain moisture (*w*).

TABLE 3. Analyses of variance of area of rice sheath blight foci (*a*) measured at several observation dates (*t*) after inoculation of a source hill at the center of rice plots

		Significance of main effect or interaction term[b]											
		Experiment 1[c]						Experiment 2[c]					
Source of variation[a]	df	*t* = 12	*t* = 19	*t* = 27	*t* = 33	*t* = 47	*t* = 56	*t* = 13	*t* = 21	*t* = 27	*t* = 35	*t* = 49	*t* = 56
Replication (R)	7			*	**			**	**	*			
Nitrogen level (N)	2	**	**	*	**	**	**	**	**	**	**	**	*
Error (a)	14												
Inoculation (I)	4	**	**	**	**	**	**	**	**	**	**	**	**
a2: among inoculations	3	**	**	*				**	**	**	**	**	*
Height (H)	1	**	**	*	*			**	**	**	**	**	**
Amount (A)	1	*	*					*		*	**	**	
H × A	1												
Control vs a2	1	**	**	**	**	**	**	**	**	**	**	**	**
N × I	8	*	**		**			**	**	**	**	**	**
N × a2	6		*							**		**	**
N × H	2									**		**	**
N × A	2									*		*	
N × A × H	2												
N × (Control vs a2)	2	**	**	**	**	*	**	**	**	**	**	**	
Error (b)	84												
Total	119												
cv (a) %		7.1	5.4	9.2	7.3	25.1	22.2	4.4	5.5	7.6	7.3	9.8	24.4
cv (b) %		6.2	4.6	6.9	7.8	17.8	16.6	5.3	5.2	5.2	7.4	7.2	21.4

[a] The analysis includes partitioning of effects of height of positioning and amount of inoculum in the source hills.
[b] **: $P < 0.01$; *: $P < 0.05$.
[c] Experiment 1: rainy season; experiment 2: dry season.

TABLE 4. Pearson correlation matrix for variables measured in experiments concerned with rate of focal expansion of rice sheath blight

	I		*s*		*C*		*W*		*N*		*LR*	
Variable[a]	*r*[b]	*P*[c]	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*
LR	0.451	0.027	0.374	0.072	0.767	0.000	0.901	0.000	0.903	0.000	1	0.000
N	0.285	0.176	0.308	0.143	0.773	0.000	0.810	0.000	1	0.000		
W	0.383	0.065	0.404	0.050	0.502	0.012	1	0.000				
C	0.178	0.406	0.198	0.354	1	0.000						
s	–0.209	0.328	1	0.000								
I	1	0.000										

[a] Symbols are explained in Table 1.
[b] Correlation coefficient.
[c] *P* values based on sample size of $n = 24$ treatment means in two experiments.

The inoculation treatments resulted in the expected pattern of disease on the source hills, i.e., higher severity at higher inoculum doses, high severity on the sheaths with inoculation at the sheath level, and high severity on the leaves with inoculation at the leaf level. There was little or no infection caused by natural inoculum. Inoculations were more successful in the first (rainy season) than in the second (dry season) experiment, which may account for the differences in rates of focal expansion between the two experiments. In the second experiment, variation in canopy density among nitrogen treatments may have exerted an early effect on establishment of disease on source hills, as indicated by variation in severity with nitrogen treatments.

To describe the development of rice sheath blight epidemics, Kozaka (17) developed the concept of vertical and horizontal spread. These two phases are represented in these experiments by the establishment of the sources, and the further spread of foci. This concept is equivalent to the more general one of esodemic and exodemic (23), or disease appearance on new host units and its further intensification. In a similar way, two epidemic phases also were distinguished in the development of soybean aerial blight (35): establishment of primary infections, and further focal spread due to interplant mycelial growth. Diseases caused by *Rhizoctonia solani* have been called leafborne (35) to emphasize the importance of tissue contacts on the second phase, their intensification in the canopy.

Vertical spread of sheath blight has been related to temperature, humidity, and physiological status of the plant tissues, while temperature, humidity, and the amount of inoculum affect horizontal spread (10,11). This study puts emphasis on canopy wetness rather than humidity in the air, and on contact frequency between host tissues rather than leaf area index as driving variables of focal expansion. Alternate wet and dry periods have been shown to enhance the rate of sheath blight increase, while permanent wetness is relatively unfavorable, and permanent dryness is inhibitory, even under high relative humidity (18). Rhizoctonia aerial blight of soybean is favored by permanent wetness, and correlations of disease descriptors with accumulated wetness period duration have been found (34).

Inoculation in the upper layer mimics a situation in which the first phase (vertical spread) of the epidemic is bypassed, and provides the newly established lesions with earlier contact with neighboring tillers and hills. This inoculation treatment generally resulted in faster focal spread.

Using the terminology of van den Bosch et al. (32), we found that interesting characteristics of leaf-borne diseases such as rice sheath blight include the fact that the disease is transferred from an infectant to a suscept by infectious units consisting in mycelial strands only, and that the contact distribution reflects physical, and ever-changing, host tissue contacts in the canopy. Direct counts of tissue contacts were used in this study to account for the latter attribute. Future analysis and understanding of epidemics of such diseases are probably strongly dependent on an adequate representation of crop geometry. New rice genotypes are currently being developed for direct seeded, irrigated, cultivation (2). Evaluation of the effect of the canopy architecture of these genotypes on sheath blight epidemics is an important researchable issue. These new genotypes have few but fertile tillers, few, erect, and long-lived leaves, and a very high harvest index; they will require high amounts of fertilizers and will be planted at high density.

Aerial symptoms of Rhizoctonia diseases are difficult to quantify (1,27,35). This is due to the nature of symptoms, the rate at which they evolve, the rapid decay of infected tissues, and the fact that it is difficult to distinguish between infected tissues and healthy tissues on which mycelial spread occurs. Several methods have been developed to assess rice sheath blight intensity (1,3,7, 19,22,27). These methods are primarily aimed at assessing host plant resistance or yield loss. Consideration of focal area and expansion does justice to the spatial nature of the disease, and to mechanisms involved in its spread (28,35). This approach should

TABLE 5. Multiple regression analysis of logarithmic-transformed mean rate (Log [R + 1]) of rice sheath blight focus expansion with data combined across experiments 1 and 2

Model	Variable[a]	Regression coefficient	Standard error	P (2 tail)[b]	adj. R^2[c]	F[d]	P[e]
1[f]	N	1.259	±0.915	0.186	0.959	108	0.000
	W	0.016	±0.027	0.000			
	C	0.001	±0.000	0.000			
	s	0.032	±0.018	0.089			
	I	0.029	±0.008	0.003			
	Constant	−0.920	±1.674	0.589			
2[g]	W	0.186	±0.019	0.000	0.957	129	0.000
	C	0.001	±0.000	0.000			
	s	0.031	±0.018	0.113			
	I	0.028	±0.009	0.004			
	Constant	1.341	±0.324	0.001			
3	W	0.219	±0.019	0.000	0.939	179	0.000
	C	0.001	±0.000	0.000			
	Constant	2.154	±0.217	0.000			
4	s	0.172	±0.059	0.008	0.378	7.98	0.003
	I	0.092	±0.028	0.004			
	Constant	1.199	±1.036	0.260			
5	N	1.015	±1.112	0.372	0.939	119	0.000
	W	0.198	±0.030	0.000			
	C	0.001	±0.000	0.000			
	Constant	0.356	±1.981	0.859			
6	N	6.699	±0.747	0.000	0.870	52.3	0.000
	s	0.069	±0.029	0.030			
	I	0.046	±0.014	0.004			
	Constant	−10.814	±1.421	0.000			

[a] Symbols for variables are given in Table 1.
[b] Significance of coefficient.
[c] Proportion of variation in the dependent variable (R^2) adjusted as: $R^2 - [(p-1)/(n-p)]\,(1-R^2)$, where n is the number of cases (24) and p the number of parameters of the regression.
[d] Computed as: regression mean square I error mean square.
[e] Significance of regression.
[f] All variables forced in equation.
[g] This model was the outcome of stepwise, upward selection involving all variables used in model 1.

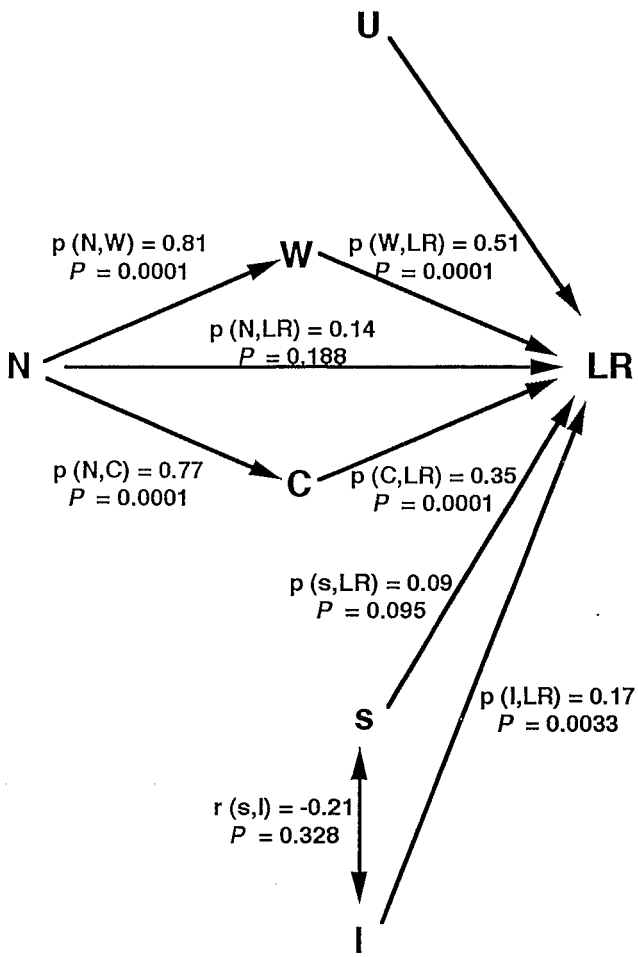

Fig. 4. Path diagram of the effect of nitrogen content in the leaves (*N*), area under accumulated leaf-to-leaf and leaf-to-sheath contact curve (*C*), area under leaf wetness curve (*W*), initial sheath blight severity on the sheaths (*s*) and leaves (*l*) of the source hill on the log-transformed average rate of expansion of sheath blight foci, caused by *Rhizoctonia solani* (*LR*). *U* represents unexplained source of variation. The path diagram is drawn according to the following conventions (29): one-headed arrow = cause-and-effect relationships between two variables, measured by a path coefficient ($p(X,Y)$). The significance level (P) of the path coefficient is indicated; double-headed arrow = correlation between two variables, measured by a correlation coefficient ($r(X,Y)$). The significance level (P) of the correlation coefficient is indicated.

prove useful in further epidemiological and crop loss studies (27).

These experiments suggest that nitrogen supply to the rice crop affects sheath blight by affecting the density of foliage, which results in increased wetness of the canopy and increased contacts among host tissues. The initial distribution and severity of disease on the leaves and/or sheaths of the inoculated source also affect the spread of sheath blight. The path model developed here is a convenient means to summarize and describe field data. It may also serve as a basis for further experiments and the exploration of new hypotheses.

LITERATURE CITED

1. Ahn, S. W., de la Peña, R. C., Candole, B. L., and Mew, T. W. 1986. A new scale for rice sheath blight (ShB) assessment. Int. Rice Res. News. 11:17.
2. Analytis, S. 1973. Methodik der Analyse von Epidemien dargestellt am Apfelschorf (*Venturia inaequalis*). Acta Phytomed. 1:1-76.
3. Anonymous. 1988. Standard Evaluation System for Rice. 3rd ed. International Rice Testing Program. IRRI, Los Baños, Philippines.
4. Anonymous. 1993. Rice Research in a Time of Change. IRRI's Medium Term Plan for 1994-1998. International Rice Research Institute, Los Baños, Philippines.
5. Bowers, J. H., Sonoda, R. M., and Mitchell, D. J. 1990. Path coefficient analysis of the effect of rainfall variables on the epidemiology of Phytophthora blight of pepper caused by *Phytophthora capsici*. Phytopathology 80:1439-1446.
6. Draper, N., and Smith, H. 1981. Applied Regression Analysis. 2nd ed. John Wiley & Sons, New York.
7. Gangopadyay, S., and Chakrabarti, N. K. 1982. Sheath blight on rice. Rev. Plant Pathol. 61:451-460.
8. Gomez, K. A., and Gomez, A. A. 1984. Statistical Procedures for Agricultural Research. 2nd ed. John Wiley & Sons, New York.
9. Hampton, R. O. 1975. The nature of bean yield reduction by bean yellow and bean common mosaic viruses. Phytopathology 65:1342-1346.
10. Hashiba, T. 1984. Forecasting model and estimation of yield loss by rice sheath blight disease. JARQ 18:92-98.
11. Hashiba, T., and Ijiri, T. 1988. Estimation of yield loss and computerized forecasting system (BLIGHTAS) for rice sheath blight disease. Trop. Agric. Res. Ser. 22:163-171.
12. Hau, B., and Kranz, J. 1990. Mathematics and statistics for analyses in epidemiology. Pages 12-52 in: Epidemics of Plant Diseases. J. Kranz, ed. Springer-Verlag, New York.
13. Inoue, Y., and Uchino, K. 1963. Studies on sheath blight of rice plants caused by *Pellicularia sasakii* (Shirai) S. Ito. I. Ecology of damage and chemical control. Subsidized Exp. in Yamaguchi Pref. (Plant diseases, insects, and pests), Agric. For. Fish. Res. Council 1:1-153.
14. International Rice Research Institute. 1992. IRRISTAT User's Manual. Version 92-1. IRRI, Los Baños, Philippines.
15. Kannaiyan, S., and Prasad, N. N. 1979. Influence of nitrogen fertilization on development of sheath blight. Int. Rice Res. Newsl. 4:14.
16. Kannaiyan, S., and Prasad, N. N. 1983. Effect of the spacing on the spread of sheath blight disease of rice. Madras Agric. J. 70:135-136.
17. Kozaka, T. 1961. Ecological studies on sheath blight of rice plant caused by *Pellicularia sasakii*, and its chemical control. Chugoku Agric. Res. 20:1-133.
18. Leaño, R. M., Castilla, N. P., Lapis, D. B., and Savary, S. 1993. A simple methodology for analyzing rice sheath blight epidemiologic processes under semi-controlled conditions. Int. Rice Res. Notes 18:42-43.
19. Marchetti, M. A., and Bollich, C. N. 1991. Quantification of the relationship between sheath blight severity and yield loss in rice. Plant Dis. 75:773-775.
20. Mithrasena, Y. J. P. K., and Adikari, W. P. 1986. Effect of density on sheath blight incidence. Int. Rice Res. Newsl. 11:21-22.
21. Otomo, T. 1989. Damage caused by major plant diseases and plant pest forecasting program in Japan. In: Crop Losses Due to Disease Outbreaks in the Tropics and Countermeasures. Trop. Agric. Res. Ser. 22:77-80.
22. Ou, S. H. 1987. Rice Diseases. 2nd ed. C.A.B. International, Farnham House, Farnham Royal, Slough.
23. Robinson, R. A. 1976. Plant Pathosystems. Springer Verlag, Berlin.
24. SAS Institute. 1989. SAS User's Guide: Statistics. Version 6. 4th ed. SAS Institute, Cary, N.C.
25. SAS Institute. 1991. SAS System for Regression. 2nd ed. SAS Institute, Cary, N.C.
26. Sharma, N. R., and Teng, P. S. 1990. Effect of inoculum source on sheath blight development. Int. Rice Res. Newsl. 15:18-19.
27. Sharma, N. R., Teng, P. S., and Olivares, F. M. 1990. Comparison of rice sheath blight assessment methods. Int. Rice Res. Newsl. 15:20-21.
28. Smith, V. L., Campbell, C. L., Jenkins, S. F., and Benson, D. M. 1988. Effects of host density and number of disease foci on epidemics of southern blight of processing carrot. Phytopathology 78:595-600.
29. Sokal, R. R., and Rohlf, F. J. 1981. Biometry. 2nd ed. W. H. Freeman and Co., San Francisco.
30. Torres, C. Q., and Teng, P. S. 1993. Path coefficient and regression analysis of the effects of leaf and panicle blast on tropical rice yield. Crop Prot. 12:296-302.
31. van Bruggen, A. H. C., and Arneson, P. A. 1986. Path coefficient analysis of effects of Rhizoctonia solani on growth and development of dry beans. Phytopathology 76:874-878.
32. van den Bosch, F., Zadoks, J. C., and Metz, J. A. J. 1988. Focus expansion in plant disease. I: The constant rate of focus expansion. Phytopathology 78:54-58.
33. Varley, J. A. 1966. Methods for the determination of nitrogen, phosphorus, and potassium in plant materials. Analyst 91:119-126.
34. Yang, X. B., Berggren, G. T. and Snow, J. P. 1990. Effects of free moisture and soybean growth stage on focus expansion of Rhizoctonia aerial blight. Phytopathology 80:497-503.
35. Yang, X. B., Snow, J. P., and Berggren, G. T. 1990. Analysis of epidemics of Rhizoctonia aerial blight of soybean in Louisiana. Phytopathology 80:386-392.
36. Yu, C. M. 1975. Effect of nutritional and microclimatic conditions on the development of sheath blight disease of rice. Los Baños, Laguna, University of the Philippines at Los Baños.
37. Yu, C. M., Ling, K. C., and Ou, S. H. 1976. Effect of nutritional and microclimatic conditions on the development of sheath blight disease of rice. Plant Prot. Bull. (Taiwan) 18:261-267.