Analysis of the spatiotemporal structure of rice sheath blight epidemics in a farmer’s field

S. Savary, N. P. Castilla and L. Willocquet

*International Rice Research Institute, Entomology and Plant Pathology Division, MOPO Box 3127, Makati City 1271, Philippines; and IRD, Institut de Recherche pour le Développement (formerly ORSTOM), Centre de Biologie et de Gestion des Populations, 911, Avenue Agropolis, BP 5045, 34 032 Montpellier cedex 1, France

The spatiotemporal structures of two rice sheath blight epidemics were studied during two consecutive (rainy and dry) rice cropping seasons in a farmer’s field in the Philippines. The amount of primary inoculum in the soil measured at transplanting was higher during the dry than during the rainy season, and showed nonaggregated spatial patterns in both seasons. Disease incidence (percentage tillers infected per hill) was monitored at weekly intervals on a (14 x 17) grid of rice hills 1 m apart, at the centre of adjacent, nonoverlapping elementary quadrats of 5 x 5 hills. The rate of disease increase at the onset of the epidemics was higher in the dry than in the rainy season, but was afterwards higher in the rainy than in the dry season. A C(a) test was used to test for overdispersion, and the beta-binomial parameter \( \theta \) was used to assess disease aggregation in quadrats consisting of one, two, four, eight and 16 elementary quadrats. Aggregation was detectable at all quadrat sizes throughout the epidemic, according to the C(a) test. \( \theta \) was highest at epidemic onset and at the smallest (elementary) quadrat size, indicating a very strong aggregation of disease at the individual hill scale. \( \theta \) increased until disease incidence reached 40\%, then declined over time. \( \theta \) values were generally smaller in the dry season. Semivariograms for both epidemics showed strong initial discontinuities, that is, large variance of disease incidence at the elementary quadrat scale. Some aggregation in the spatial distribution of disease incidence was indicated at higher scales in the rainy season, coinciding with the highest rate of disease increase. Semivariograms did not suggest any structure in the dry season, except for one date in the early epidemic stage. Spatiotemporal autocorrelation of disease incidence at 45 days after transplanting yielded very few variables that contributed significantly to describing the disease incidence and its increase in a given hill. In the rainy season these variables were disease incidence (or increase) in the nearest sampled neighbours, and disease incidence (or increase) observed 1 week earlier in the same hill. In the dry season only disease incidence (or increase) observed 1 week earlier in the same hill was significantly correlated with the current incidence (or its increase). The development of a spatial structure in distribution of disease is hypothesized to coincide with the spread of the pathogen from one hill to another, and with the shift from monocyclic to predominantly polycyclic processes in sheath blight epidemics. These results concur with previous reports in suggesting that management of rice sheath blight should first be directed against the leafborne, polycyclic phase of epidemics, rather than their initial phase.

Keywords: disease incidence, geostatistics, monol/polycyclic process, Rhizoctonia solani, rice sheath blight, soilborne inoculum, spatiotemporal analysis

Introduction

Most diseases caused by soilborne pathogens have the underground tissues of host plants as pathozones (Gilligan, 1985; Benson, 1994). However, several diseases caused by Rhizoctonia solani primarily affect the above-ground parts of their host plants (e.g. Yang et al., 1990; Sneh et al., 1996). In the case of rice sheath blight two pathozones can be distinguished (Savary et al., 1997): (i) the base of the plants, and (ii) the sheaths and leaves of the upper part of the plant canopy. The main phases of a rice sheath blight epidemic can be summarized as follows. In the first phase the primary, soilborne inoculum usually generates primary lesions at the base of the plants. This phase is a reflection of the soilborne phase of the pathogen. There is a debate regarding the nature of primary inoculum of rice sheath blight in the tropics: sclerotia (Palo, 1926; Hori & Anraku, 1971; Hashiba & Mogi, 1975; Kozaka, 1975;
Hashiba, 1982; Roy, 1986) have long been regarded as the main sources of inoculum; however mycelium surviving in crop residues (Hori & Anraku, 1971; Mew et al., 1980; Kobayashi et al., 1995), and infected weeds (Kozaka, 1970; Damodar Naidu et al., 1983) appear to play an important role as well. This first phase may be considered monocyclic (Savary et al., 1997). When primary infections are established at the base of the crop canopy, a 'leafborne' (Yang et al., 1990; Savary et al., 1995), polycyclic phase can then develop and superimpose itself on the first (Savary et al., 1997) in the second pathozone, that is, the upper part of the canopy. The primary lesions expand and often coalesce, and give rise to strands of mycelium that run on the surface of healthy sheaths and leaves where they establish new lesions. New infections are also established by running strands of mycelium that progress across the canopy, taking advantage of contacts between healthy and diseased tillers belonging to the same, or different, rice hills (or rice plants in the case of a direct-seeded rice crop; Ou, 1985). The number of contacts between host tissues, as well as leaf wetness duration, strongly enhance the leafborne phase of sheath blight epidemics (Savary et al., 1995; Castilla et al., 1996).

A better understanding of the spatiotemporal structure of rice sheath blight epidemics is necessary to develop efficient management methods for this disease, which has become an important rice disease worldwide (Ou, 1985). The peculiar way in which the pathogen progresses within the host population presumably has a major influence on the spatial progress of the disease and on the dynamics of epidemics. This study was conducted to quantify and analyse the spatial distribution of (i) primary inoculum; (ii) primary lesions and (iii) new, secondary infections, and their relationships over time. The analysis is based on data collected during two spontaneous, successive rice sheath blight epidemics occurring in a naturally infested farmer's field in the Philippines.

Materials and methods

Experimental site

Two rice sheath blight epidemics were monitored during two successive rice cropping seasons. The study was conducted in a farmer’s irrigated rice field at Pila, 15 km from the International Rice Research Institute, in the Laguna Province of the Philippines. The field area was 340 m² (length 20 m, width 17 m), and had a long-standing sheath blight history. Weather data (daily rainfall, radiation and minimum/maximum temperature) were recorded at a standard weather station located about 10 km from the experimental site.

Crop establishment

In the first (rainy) season the field was planted on June 26, 1995 with IR72, a short-cycle, high-yielding rice cultivar. The same variety was planted again in the dry season, on January 3, 1996. Six to nine 12-day-old seedlings were manually transplanted per hill. Spacing between hills was 20 cm within and between rows. This method of crop establishment is typical of the cropping practices used in the Philippines, and prevails in much of the lowland rice production areas of tropical Asia, with local variants (Vergara, 1979; Greenland, 1997). The field was irrigated during both cropping seasons. A sufficient supply of nitrogen was ensured by inflowing water from a fish pond. During the rainy season, however, an additional 25 kg nitrogen ha⁻¹ was applied as urea 50 days after transplanting (DAT). No nitrogen was applied on the dry-season crop.

Sampling and assessment of primary inoculum

Soil samples were collected at points on a 2 × 2 m grid just before transplanting of the 1995 rainy season crop, and of the 1996 dry season crop. The field was carefully marked before transplanting so that each soil sample was taken from a point corresponding to a hill assessed for disease incidence.

The quantification of primary inoculum in the soil was followed Castilla (1998). Each soil sample consisted of 300–350 g soil collected to a depth of ≈5 cm. Each individual soil sample was placed in a pot with drainage holes. Soil samples were air-dried for 10 days at 30–33°C, and the soil was dried uniformly to a soil moisture content of 17–21% (w/w). Each sample was then poured to a particle size of ≈2–5 mm in diameter. Ten seeds of mungbean (Vigna radiata var. Pag-asa) were sown in each pot, covered with soil and watered with 100 mL tap water. The soil moisture content was maintained at ≈30–45% (w/w) until observations were completed.

The number of healthy seedlings, diseased seedlings and ungerminated seeds in each pot was recorded at 2 day intervals. At each observation infected mungbean tissues were cut into small segments, surface sterilized and placed in Petri dishes on potato dextrose agar (PDA) medium with 1 mL L⁻¹ 25% lactic acid. After 2 days the Petri dishes were examined for growth of R. solani. Each R. solani colony growing from an infected tissue was subcultured on PDA. An isolate with the following characteristics (Farmer & Whitney, 1970) was considered as R. solani: (a) branching near the distal septum of hyphal cells, often at or nearly at right angles in older hyphae; (b) constriction of branch hyphae at the point of origin; and (c) formation of a septum in the branch near the point. The pathogenicity of the resulting isolates was tested on rice (cultivar IR72). Five 5 cm leaf segments cut from the third- or fourth leaves of 40- to 50-day-old plants were placed on Petri dishes with water agar (5 g agar L⁻¹). Plugs (8 mm diameter) of 4- to 5-day-old colonies were placed at the centre of each leaf segment, and the Petri dishes were incubated at 25–27°C. After four days the leaf segments were examined for the presence of sheath...
Assessment confirmed when infection was observed on at least remaining practically feasible (Fig. 1). Disease assessment was weekly by counting the tillers, and the number of diseased tillers assessed for sheath blight incidence were at the centre of 5 x 5-hill quadrats (broken lines).

Figure 1 Sampling scheme for the assessment of disease incidence and soil sampling in the field. Rice hills (circles, 0.2 m apart across and along rows) were assessed for disease incidence (closed circles and circles with line) at 1 m (four-hill) intervals. Soil samples (shaded circles) were taken at 2 m intervals. Rice hills assessed for sheath blight incidence were at the centre of 5 x 5-hill quadrats (broken lines).

blight lesions. The presence of the sheath blight pathogen in a soil sample was considered to be confirmed when infection was observed on at least one leaf segment.

Assessment of disease incidence

Disease incidence was assessed weekly by counting the total number of tillers, and the number of diseased tillers of hills located on a 14 x 17 grid that were 1 m (four hills) apart (Fig. 1). This distance was based on previous results indicating that the area of a rice sheath blight focus usually ranges from 0.1 to 3 m² (Savary et al., 1995), covering two to 64 neighbouring hills. This 5 x 5 hill-sampling grid was chosen to enable an analysis at the whole-field scale, while retaining some information pertaining to the focus expansion scale, and remaining practically feasible (Fig. 1). Disease assessments were made from 17 DAT until harvest at weekly intervals. A total of 238 hills were assessed weekly throughout each cropping season.

Aggregation of primary inoculum and sheath blight incidence

A C(α) test statistic (Z) and the beta-binomial parameter θ were calculated to assess aggregation of primary inoculum and disease incidence. Z is the standard normal test statistic, which allows testing of the null hypothesis of random distribution, with the alternative hypothesis that the overdispersion is described by the relative-binomial distribution (Madden & Hughes, 1995). The θ parameter of the beta-binomial distribution is an index of aggregation, which equals zero when there is a random pattern and increases as heterogeneity (corresponding to aggregated spatial pattern) increases (Madden & Hughes, 1995). The parameter of aggregation, θ, was estimated when the C(α) test was significant (P < 0.05). When the number of entities (n) per sampling unit (i) did not exceed 199, Z and θ were computed using the BBD software (Madden & Hughes, 1994). When n was too large to use the BBD software, Z was computed according to Madden et al. (1995a), and θ was computed according to Madden & Hughes, 1995).

In the case of the primary inoculum, the variable analysed (X) was the number of soil samples where the sheath blight pathogen had been detected. Two quadrat sizes (QIP2 and QIP4) were considered, consisting of two or four adjacent, nonoverlapping elementary quadrats (2 x 2 m) where soil samples were taken. In the case of sheath blight incidence the variable analysed (X) was the number of diseased tillers. Five quadrat sizes (QINC1, QINC2, QINC4, QINC8, QINC16) were considered, consisting of one, two, four, eight or 16 adjacent, nonoverlapping, elementary quadrats (1 x 1 m) where rice hills had been assessed for sheath blight incidence. Considering sampling units in spatially contiguous and increasingly larger quadrats allowed us to address aggregation at increasing scales (Campbell & Madden, 1990). Large (eight- and 16-quadrat) sampling units could not be considered in the case of primary inoculum, owing to the larger spacing between soil samplings (2 x 2 m) compared to spacing between disease assessments (1 x 1 m). Individual soil samples (elementary quadrats) were not considered in these analyses as the information generated by each sample resolved into binary information (detection/non-detection).

Power law analysis

The modified power law for disease incidence can be written generally as (Hughes & Madden, 1992):

\[ \ln(V(X)) = a + b \ln(X) \]

where \( V(X) \) is the theoretical variance of a binomial distribution corresponding to a random spatial pattern. When a = 0 and b = 1, X is randomly distributed. When b = 1 and a > 0 there is aggregation, but aggregation does not depend on the mean disease proportion, p. When a > 0 and b > 1 there is aggregation which depends on p (Turechek & Madden, 1999).

The parameters a and b were estimated by linear regression analyses of the incidence data computed at different sampling unit sizes in the two seasons. This analysis allowed the characterization of disease distribution across sampling unit sizes and across years.

Geostatistical analysis

The semivariance, C (h), is defined as half the average
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Figure 2 Spatial distribution of disease incidence over time for the 1995 rainy season epidemic (a) and 1996 dry season epidemic (b). DAT, days after transplanting. Spatial co-ordinates: rice hill number (spacing between hills: 0.2 m). Circles increase in size from smallest (0% < incidence 20%); through 20% < incidence 50%; 50% < incidence 80%; to largest (80% < incidence 100%).

of the squared differences in values between pairs of samples separated by a given lag distance h. Variation in semivariance over lag distances is plotted in a semivariogram (Matheron, 1963; Cressie, 1985, 1991; Tranmgmar et al., 1985; Isaaks & Srivastava, 1989). \( G(h) \) is defined for any distance \( h \) as:

\[
G(h) = \frac{1}{2N_h} \sum \left( F(x_i + h) - F(x_i) \right)^2
\]

where \( N_h \) is the number of pairs \( (x_i, x_i + h) \); \( x_i \) is the
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A sudden increase of $G(h)$, representing a change in spatial pattern at a given scale. A nugget effect at lag zero ($h = 0$) reflects spatial variation occurring below the minimum sampling scale, or measurement error.

Disease incidence was considered a regionalized variable, with $F(x_i)$ as the disease incidence of hill $i$ and $F(x_{i+h})$ as the disease of another hill $h$ units away. Geostatistical analysis was conducted on disease incidence data during both seasons from 17 to 80 DAT using gEOEAS Version 1-1 (Englund & Sparks, 1988).

**Spatialtemporal autocorrelation analysis**

Multiple regression analyses were performed using a forward selection procedure (SYSTAT 5-2, Wilkinson et al., 1992) to determine the spatiotemporal autocorrelation of the disease incidence of a hill with that of its neighbouring hills, and with the disease incidence of this same hill at previous sampling dates. This analysis therefore allowed the simultaneous analysis of the spatial and temporal characteristics of the epidemics. Disease incidence data at 45 DAT were used as the dependent variable. At this date, the disease incidence level in the rainy season was the same as that in the dry season (19%). After 45 DAT the levels and rates of increase in disease incidence started to show very strong differences between seasons.

A pure autoregressive process was considered (Reynolds & Madden, 1988). Considering a spatial lag $s$ ranging from 0 to $S$ sampled hills, and a temporal lag $k$ ranging from 0 to $K$ weeks before 45 DAT, the regression model tested on both epidemics can be summarized as:

$$y_{i,t} = \sum_{s=0}^{S} \sum_{k=0}^{K} \beta_{s,k} Y_{i+s,t-k} + \epsilon_{i,t}$$

where $y_{i,t}$ = disease incidence of hill $i$ at time $t$ (45 DAT); $\beta_{s,k}$ = regression coefficient at spatial lag $s$ and temporal lag $k$; $y_{i+s,t-k} = $ mean disease incidence of hills $i$ at spatial lag $s$ (in a rook proximity pattern, Reynolds & Madden, 1988; Campbell & Madden, 1990) and temporal lag $k$; and $\epsilon_{i,t}$ = error term for hill $i$ at time $t$ and represents the difference between the observed and expected $y_{i,t}$.

The same model was also tested after correcting incidences for nonstationarity over time (Reynolds & Madden, 1988), considering $\Delta y_{i,t} = y_{i,t} - y_{i,t-1}$ and $\Delta y_{i+s,t-k} = y_{i+s,t-k} - y_{i+s,t-k-1}$ in the above model.

**Results**

Overall climatic conditions during the two epidemics

The two rice cropping seasons differed markedly, especially in terms of accumulated rainfall (1292 versus 153 mm in the rainy and dry seasons, respectively), but also mean daily radiation (16-8 versus 17-9 MJ m$^{-2}$ day$^{-1}$), mean maximum temperature
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Figure 4 Relationship between the logarithm of observed variance and the logarithm of theoretical variance for a random pattern of incidence of rice sheath blight in the rainy season 1995 and dry season 1996, at different spatial scales. Each dot represents data pertaining to one assessment date. Broken line represents the binomial line (observed variance = binomial variance); solid line represents the line regressed from the data. See Table 1 for corresponding parameters and R².

versus 29-0°C, and mean minimum temperature (24-0 versus 22-8°C).

Primary inoculum

The amount of primary inoculum in the soil at transplanting was significantly lower in the rainy season than in the dry season: the sheath blight pathogen was detected in only 13% of the soil samples taken before the rainy season versus 84% in the dry season ($\chi^2 = 63.3$, $P < 0.001$). In the 1995 rainy season the values of Z found for QIP2 and QIP4 were 0-81 and 0-47, respectively. These values were lower than the critical Z value (1-64 for $P < 0.05$), and were suggestive of a random distribution of primary inoculum. In the 1996 dry season the Z values for QIP2 and QIP4 were 1-30 and 1-28, respectively, and again indicated a random distribution of the primary inoculum.

Spatial distribution of disease incidence over time

The patterns of spatial distribution of disease incidence (percentage tillers infected) were examined in both epidemics. In the rainy season epidemic (Fig. 2a) a small number of individual hills (quadrats) were diseased at 24 DAT, in some cases with high individual incidences. Some clumping of disease appeared to occur until 52 DAT, but no specific spatial pattern could be detected visually afterwards. Near-saturation with disease (100% incidence) occurred after 66 DAT.

In the dry season epidemic (Fig. 2b) no disease was observed at 24 DAT. At 31 DAT sheath blight was observed in many individual hills, and some clumping among infected hills appeared to occur at this date. Visual examination of the distribution of diseased hills did not indicate aggregation of the disease afterwards.

The two sets of maps also indicate that the rate of increase of diseased hills (incidence at the hill scale) was higher in the early stage of the dry (Fig. 2b) than of the rainy (Fig. 2a) season epidemic, even though disease was observed earlier in the rainy season compared with the dry season. Between 31 and 38 DAT the percentage of infected hills increased from 15-5 to 22-3% in the first (rainy season) epidemic, while the increase was from 26-1 to 57-6% over the same period in the second (dry season) epidemic: relative rates of infection at the hill scale of 0-96 versus 4-50% day$^{-1}$ for the first and second epidemics, respectively.

Disease incidence progress curves

At 38 DAT sheath blight incidence at the tiller scale was lower in the first (rainy season, Fig. 3a) than in the second (dry season Fig. 3b) epidemic. The slope of the disease incidence progress curve at the onset of epidemics (24-38 DAT) was higher in the dry than in the rainy season epidemic (Fig. 3). The initially higher rates of increase and levels of disease incidence at the tiller scale in the dry season epidemic are attributable to the corresponding higher level of disease incidence at the hill level. After 45 DAT, however, the rate of increase of disease incidence was much higher (approximately twice as high) in the rainy than in the dry season. The disease progress curve in the dry season epidemic started to taper off after 59 DAT, and showed a decline towards the end of the cropping season. The terminal incidence was approximately three times higher in the rainy compared to the dry season epidemic ($\approx$95% compared to $\approx$30%).

Aggregation of disease incidence

The $Z$ values calculated in the 1995 rainy season were always above the critical $Z$ value, indicating that disease was aggregated at all scales and at all assessment dates. $Z$ values varied with space (quadrat size) and time (Fig. 3a). At 24 DAT, $\theta$ values increased with decreasing sampling unit size, and ranged between 0.007 and 0.24. Disease aggregation increased sharply between 24 and 52 DAT when it nearly reached 3.5 at the elementary quadrat scale (QINC1). $\theta$ decreased progressively until 94 DAT ($\theta$ for QINC1 was 0.62). At each assessment date $\theta$ values decreased with increasing quadrat size from QINC1 to QINC2, and from QINC2 to QINC4; The decline was much less for the subsequent increase in quadrat size.

In the 1996 dry season, $\theta$ values ranged between 0.01 and 0.1 at 31 DAT, when disease incidence was 2.5%. In this second epidemic the initial $\theta$ values were smaller

Table 1 Results of the linear regression analysis of the power law$^a$ for sheath blight incidence data, at different sampling unit sizes, in the rainy season 1995 and the dry season 1996

<table>
<thead>
<tr>
<th>Sampling unit size</th>
<th>Rainy season 1995</th>
<th>Dry season 1996</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$a$</td>
<td>$b$</td>
</tr>
<tr>
<td>One elementary quadrat</td>
<td>2.50(a)</td>
<td>1.40(b)</td>
</tr>
<tr>
<td>Two elementary quadrats</td>
<td>2.26(ab)</td>
<td>1.43(ab)</td>
</tr>
<tr>
<td>Four elementary quadrats</td>
<td>1.91(bc)</td>
<td>1.48(ab)</td>
</tr>
<tr>
<td>Eight elementary quadrats</td>
<td>1.60(cd)</td>
<td>1.60(ab)</td>
</tr>
<tr>
<td>16 elementary quadrats</td>
<td>0.67(de)</td>
<td>1.65(ab)</td>
</tr>
</tbody>
</table>

$^a$The regression tested was $ln(V_X) = a + b ln(V_X)$ where $V_X$ is the observed variance of the number of diseased tillers and $V_X$ is the theoretical variance of the corresponding random distribution.

$^b$Within a column parameters followed by the same letter are not significantly ($P < 0.05$) different according to the t-test (Campbell & Madden, 1990).
than in 1995 at the epidemic onset. The values of $\theta$ computed at the elementary quadrat scale increased sharply until 66 DAT (reaching 2), remained stable to 80 DAT, and finally declined linearly until they reached 94 DAT (Fig. 3b). As in the preceding epidemic, variations of $\theta$ over time were similar at all quadrat sizes, and larger $\theta$ values associated with smaller quadrat sizes were also observed, especially when considering scales between QINC1 and QINCl.

Power law analyses

The modified power law provided a very good description of variation in observed variance at all spatial scales in both seasons, and the regressions were associated with $R^2$ values above 95% (Fig. 4, Table 1). All slope parameters ($b$) were significantly ($P < 0.05$) $>$1, and intercepts ($a$) were significantly $>$0 for sample sizes of one to eight elementary quadrats, and for
sampling units of one and two elementary quadrats, in 1995 and 1996, respectively. In 1995 parameter $a$ decreased with increasing quadrat size, and was significantly larger for one elementary quadrat than for sampling units larger than four elementary quadrats (Table 1). In 1995 parameter $b$ increased with increasing size of sampling unit, and $b$ was significantly larger for one elementary quadrat than for 16 elementary quadrats (Table 1). In 1996 parameter $a$ was largest at a sampling unit of one elementary quadrat. The estimated value of $a$ at the eight-elementary quadrat scale was not significantly ($P < 0.05$) different from zero. In 1996 parameter $b$ decreased with increasing sampling unit size, and $b$ was significantly larger for one than for 16 elementary quadrats. When comparing the values of parameters $a$ and $b$ for one elementary quadrat in 1995 and 1996 using the $t$-test, $a$ was significantly ($P < 0.05$) larger in 1995 than in 1996, and $b$ was significantly ($P < 0.05$) smaller in 1995 than in 1996.
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Table 2. Multiple linear regression analyses of disease incidence of a sampled hill, \( y_i \), at assessment date (45 DAT) with the disease incidence at spatial lag ranging from no to two sampled hills and temporal lag ranging from 0 to 2 weeks before 45 DAT.

<table>
<thead>
<tr>
<th>Equation</th>
<th>Epidemic</th>
<th>Regression equation ( b )</th>
<th>( r^2 )</th>
<th>( F )</th>
<th>( p^a )</th>
<th>( p^b )</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Rainy season</td>
<td>( y_{0,0} = 1.478y_{0,1} + 0.98y_{1,1} )</td>
<td>0.563</td>
<td>82.6</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>2</td>
<td>Dry season</td>
<td>( y_{0,0} = 1.30y_{0,0} )</td>
<td>0.682</td>
<td>260.0</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

*Regression models and variables are described in the text.

1. \( y_{0,0} \) = disease incidence of the same hill (spatial lag = 0) at 1 week before 45 DAT (temporal lag = 1).
2. \( y_{1,1} \) = disease incidence of the nearest sampled hill (spatial lag = 1) at 1 week before 45 DAT (temporal lag = 1).
3. Coefficient of determination.
4. Computed F-value (regression mean square / error mean square).
5. Significance of regression.

Geostatistical analysis

Figure 5 shows semivariograms for both epidemics. At all dates, for both epidemics, very large initial discontinuities in semivariograms ("nugget effect") were observed.

Figure 6 Variation of \( \psi \) values at the elementary quadrat scale with rice sheath blight incidence (a) and with incidence increase (b) in the rainy season 1995 and dry season 1996.

This indicates that a large fraction of the variance in incidence was attributable to variation occurring at a scale lower than that of the grid of the sampling scheme. Nevertheless, the semivariograms showed a slight structure—a slope indicating a progressive increase in semivariance with increasing distance between samples—in disease incidence at four dates (31, 45, 52 and 59 DAT) in the rainy season epidemic (Fig. 5a). At 31, 45 and 52 DAT, an increase was detectable up to a maximum distance (range) of 13 hills between samples, whereas at 59 DAT this increase was detectable only to an eight-hill distance. The appearance of this structure approximately corresponded to the highest rates of disease increase in this first epidemic (Fig. 3). In the second, dry season epidemic (Fig. 5b), the semivariograms showed a transient, slight (range = 15 hills) structure only at 31 DAT.

Spatiotemporal autocorrelation analysis

Although successive observation dates and increasing distance lags were considered in the analysis, only very few variables, that is, time and space lags, were significantly correlated with incidence and increase in incidence (\( y_{1,1} \) and disease incidence) observed in the two epidemics at 45 DAT. The outputs of a spatiotemporal autocorrelation analysis of disease incidence are summarized in Table 2. Equation 1 shows that at 45 DAT, significant autocorrelation of disease incidence in the rainy season occurred only at the shortest spatial lag (nearest sampled hill) and shortest temporal lag (1 week before 45 DAT). Equation 2 shows that in the dry season significant autocorrelation also occurred at a temporal lag of 1 week, but no significant spatial autocorrelation in disease incidence was detected. When considering the increase in disease incidence in each sampled hill (correcting for nonstationarity of disease incidence), similar equations were obtained:

\[
\begin{align*}
\psi_{1,1} &= 0.478y_{0,1} + 1.378y_{1,1} (\psi_{0,1} : T = 2.71, P = 0.0077, \psi_{1,1} : T = 3.33, P = 0.0011) \\
\end{align*}
\]

for the 1995 rainy season, and:

\[
\begin{align*}
\psi_{1,1} &= 0.299y_{0,1} (\psi_{0,1} : T = 3.44, P = 0.008) \\
\end{align*}
\]
for the 1996 dry season. No significant correlations were found between $Y_{0.2}$ and other lags considered (e.g. $Y_{0.2}$, $Y_{1.2}$, $Y_{2.1}$ or $Y_{2.2}$).

**Discussion**

**Context of the study**

This analysis of two successive epidemics in a farmer's field highlights a series of features that characterize rice sheath blight epidemics under tropical conditions. As stated by Campbell & Madden (1990), "The question of replication is spatial pattern studies is ever persistent... True replication... is usually unfeasible, if not impossible, in natural ecosystems or even in agroecosystems with soilborne or vectorborne organisms." The study reported here, however, is part of a broader context, which helps support its interpretations. A survey of rice diseases, insect pests and weeds conducted in the central plain of Luzon, the Philippines, on a population of 291 fields and covering four cropping seasons (Savary et al., 1994), indicated that sheath blight prevalence (proportion of fields affected) was high in both rainy (69-0%) and dry (62.5%) seasons. Sheath blight prevalence did not directly reflect that primary inoculum; however, the proportion of fields where strong epidemics developed (with a maximum incidence higher than 5% in the course of a crop cycle) was significantly higher in the rainy (75-0%) than in the dry (35-7%) season ($\chi^2 = 12.50$, $P < 0.0001$). In both seasons sheath blight inoculum was present in a large proportion of fields; however, strong epidemics primarily developed in the rainy season. The reasons why stronger sheath blight epidemics develop in the rainy season are well documented, and stem mainly from the presence of moisture in crop canopies for longer periods, making contacts between healthy and diseased plant tissues efficient avenues for the progress of the pathogen, and enabling the spread of the disease (Savary et al., 1995). The spatial pattern of initial infections (which may or may not directly reflect that of the primary inoculum; Belmar et al., 1987) and its effect on the rate of disease increase has also been addressed (Savary et al., 1997), indicating that nonaggregated (uniform or random) patterns lead to faster epidemics than an aggregated pattern does (Savary et al., 1997, F.A. Elasegui & N.P. Castilla, unpublished results).

**Differences between the two epidemics**

The two epidemics considered in the present study differed sharply in their onsets, rates and terminal incidences. While the first (rainy season) epidemic was slow to establish, it later developed at a high rate and led to a very high terminal incidence. The second epidemic, in contrast, established rapidly and had an initially high rate of increase which later declined, and led disease incidence to taper off rather early in the crop cycle, and even to decline towards harvest.

Another important difference between the two epidemics was the amount of primary inoculum detected in the soil at transplanting: small in the first and large in the second. This difference explains why the onset of the second epidemic was so rapid. The spatial distribution of primary inoculum appeared to be nonaggregated in both epidemics, leading to a higher rate of increase in incidence at the hill scale in the second epidemic. Much of the high rate of the early disease increase in the second epidemic can therefore be attributed to a large amount of randomly distributed inoculum, which infected many rice hills and the tillers within the infected hills.

**Interpretation of variations in $\theta$ values**

In order to interpret the meaning of variations of $\theta$ values calculated on sheath blight incidence (percentage of tillers infected) at different scales, one important point needs to be made. $\theta$ for QINC1 refers to only one hill in a quadrat, that is, to disease aggregation at the hill scale, and can be seen as reflecting the association of tillers presenting the same disease status within a quadrat (diseased tillers associated with other diseased tillers within a quadrat, and healthy tillers associated with other healthy tillers within a quadrat; Madden et al., 1995b). A decline of $\theta$ at this scale indicates a decreasing aggregation at the hill scale. On the other hand, a decrease of $\theta$ values pertaining to higher scales (QINC2 to QINC16) reflects the averaging of local variation in incidence, which smooths out spatial variation occurring at a lower scale.

The 1995 rainy season epidemic was initiated by a small amount of random inoculum. This resulted in infection of a few hills, and often in several tiller infections in the same hill. As a result the epidemic started with a strong aggregation of disease at the hill scale (QINC1). Until 52 DAT, when disease incidence was 40%, the increase in $\theta$ values indicated that hills infected early in the epidemic had many diseased tillers. After 52 DAT, $\theta$ values declined. This may be due to spread of the disease across hills, averaging effect, and disease saturation. This decline in $\theta$ values contained throughout the season as disease increased to a high terminal incidence. Both rapid disease increase and disappearance of patchiness may thus be attributable to favourable conditions for disease spread in this season. In the 1996 dry season, the amount of primary inoculum was large and randomly distributed. Many individual hill infections took place, and this explains why $\theta$ values lower than in 1995 were observed at all scales early in the season. This difference may be attributed to the larger amount of primary inoculum, and of primary infections, occurring in the dry season: in this case $\theta$ values at the elementary quadrat scale reflected a number of low-to-medium incidences (compare Fig. 2a,b at 31 DAT), and fewer null values. As in
the rainy season, \( \theta \) values increased until incidence reached 40%. This corresponded to an increase in the number of infected hills, and to disease intensification within infected hills. The values of \( \theta \) then tapered off and declined until the end of the crop cycle. This decline in disease aggregation over time may be attributed to two different mechanisms. The first corresponds to disease increase (to 73 DAT) and can be attributed, as in the rainy season, to spread of the disease across hills and/or averaging effect. The second corresponds to disease recovery (after 73 DAT), that is, the disappearance of old lesions on tillers where no new lesions appeared. We may assume that tillers that recover from sheath blight infections are randomly distributed, leading to a decrease in disease aggregation. It is worth noting that at the end of the epidemic (94 DAT) in the dry season, the value of \( \theta \) at the elementary quadrat scale was about twice that of \( \theta \) at the same date in the rainy season (1 versus 0.6). At all scales the values of \( \theta \) thus declined less in the dry than in the rainy season, presumably because of unfavourable conditions prevailing in the dry season, hampering hill-to-hill (two- to 16-quadrat scale) and tiller-to-tiller (one-quadrat scale) spread.

The parameter \( \theta \) at the elementary quadrat scale (QINCl) was plotted against disease incidence (Fig. 6a) and increase in disease incidence (Fig. 6b) for both seasons, in order to allow interpretation of the relationships between disease dynamics and disease aggregation. Figure 6(a) shows that in both seasons, disease aggregation first tends to increase with disease incidence (incidence from 0 to 40%), then stabilizes, and finally declines when incidence increases from 80 to 100%. This type of pattern has been observed for other diseases (grape downy mildew, Madden et al., 1995a; strawberry leaf blight, Turechek & Madden, 1999). This bell-shaped pattern also conforms to theoretical relationships between \( \theta \) and disease incidence (Hughes & Madden, 1992; Madden & Hughes, 1995). The values of \( \theta \) also increased when disease incidence increased (Fig. 6b), supporting the hypothesis that disease aggregation and disease spread are related.

**Interpretation of power law analyses**

The modified power law provided a good description of the observed variance of incidence data of rice sheath blight (Table 1, Fig. 4), confirming that this law is adequate for analysing incidence data in many pathosystems (Madden & Hughes, 1995). The synthetic information provided by this analysis is in agreement with the interpretation of variations in \( \theta \): (i) in both seasons parameter \( a \), reflecting overall aggregation, declined with the size of the sampling unit, and \( a \) was larger in the rainy than in the dry season; (ii) the values estimated for parameter \( b \) indicated a linkage between aggregation and disease incidence at all scales in both seasons. This linkage is illustrated by Fig. 6(a) for the individual quadrat scale.

**Interpretation of semivariograms**

A disadvantage of the use of aggregation indices such as \( \theta \) is that they do not take into account the location of samples (Campbell & Madden, 1990). Location is partly addressed by considering \( \theta \) values at increasing quadrat sizes. Examination of semivariograms takes into account proximity among samples, and therefore generates new information. The semivariograms in the first epidemic exhibited some structure from 31 to 59 DAT (Fig. 5a), at approximately the time when the rate of increase of disease incidence was highest. A structure, if any, was detected only once in the second epidemic (31 DAT, Fig. 5b). This suggests that spatial correlations are associated with a second phase of sheath blight epidemics, where contacts among neighbouring diseased and healthy plants (Savary et al., 1995) lead to polycyclic disease increase and disease spread (Savary et al., 1997). It can be hypothesized that in the second epidemic this second, polycyclic phase was hampered. In comparison with the first epidemic, where such a spatial aggregation took place, the second had a very low rate of disease increase. Rice sheath blight develops more quickly under the humid conditions of the rainy season (Gangopadhyay & Chakrabarti, 1982; Webster & Gunnell, 1992). This interpretation is further supported by spatiotemporal autocorrelation analysis which indicates that a spatial correlation was found only in the first epidemic. The only spatial correlation found is a one-lag (four-hill) only, which in turn is a reflection of the very large initial discontinuities found in the semivariograms. Experiments on the spread of rice sheath blight conducted in both rainy and dry seasons (Savary et al., 1995) indicate that the choice of distance between two assessment points for disease incidence was appropriate, as the size of foci obtained in these experiments ranged between 0.1 and 3 m².

**Autocorrelation analysis**

Spatiotemporal autocorrelation further shows that incidences observed at 45 DAT were correlated only with those observed 1 week earlier in both epidemics. This corresponds with earlier findings on the very transient nature of rice sheath blight symptoms (Savary et al., 1995), and the difficulty of assessing the disease in the field (Savary & Mew, 1996). The same features were found for other Rhizoctonia diseases causing symptoms on the aerial part of plants on soybean (Yang et al., 1990) and white clover (Nelson & Campbell, 1993).

**Synthesizing information on the two epidemics**

Using spatiotemporal information the two disease progress curves of Fig. 3 can be interpreted as follows. The first epidemic was initiated by a limited amount of randomly distributed inoculum; although the initial
rate of increase in incidence was low, this was nevertheless sufficient to establish an epidemic. Favourable environmental conditions of the rainy season and further growth of the canopy led to increased contacts between infected and healthy tissues belonging to the same or different hills, and to a regular, polycyclic, increase in disease incidence. Crop growth was a main contributor of the epidemic build-up, and was reflected by a slight, albeit short-range, spatial structure.

The second epidemic was initiated by a large and randomly distributed amount of soil inoculum. This led many individual hills to be infected in the early stage of the epidemic, and a rapid rate of increase of disease incidence. However, unfavourable environmental conditions prevented this strong disease establishment to further progress into the growing canopy, and much of the polycyclic increase of disease was hampered. The result was a limited (as compared to the rainy season) aggregation of disease, a decline of the rate of disease increase, and a much lower disease incidence.

It can further be hypothesized that the first epidemic largely contributed to the build-up of primary inoculum of the second epidemic. The nonaggregated distribution of primary inoculum observed at transplanting in this study can be attributed to its continual redistribution during land preparation activities (ploughing, harrowing and flooding). The spatial distribution of primary inoculum was found to be aggregated in other studies on rice sheath blight (Yin, 1985; Belmar et al., 1987; Yin & Mew, 1987). However, these studies mainly considered sclerotia as the primary inoculum of the disease. There is controversy as whether sclerotia actually represent the main source of inoculum in rice sheath blight under tropical conditions. Cai et al. (1996), for instance, reported a maximum density of 1-23 sclerotia per litre of soil in the Philippines, compared to 27-87 sclerotia per litre of soil reported by Lee (1980) in the USA. In this study, different sources of primary inoculum, whether sclerotia, mycelium or colonized crop residues, were addressed using a 'mungbean assay'. The scale at which aggregation is addressed also has to be considered, and variation in scales may lead to different results (e.g. Belmar et al., 1987). As in other Rhizoctonia diseases (such as soybean web blight; Yang et al., 1990), the nature of the primary inoculum sources in sheath blight, and their relative importance, need confirmation.

Concluding remarks

This study illustrates how the spatial distribution of disease is related to disease increase in rice sheath blight. It shows that a rapid (rainy season) epidemic is associated to some degree of spatial structure at a 1 m scale, whereas a slow (dry season) epidemic barely shows a spatial structure beyond the scale of an individual hill. Detection of some level of disease aggregation, given the fact that symptoms are so transient, is therefore indicative of disease intensification. This result suggests that some spatial aggregation is a reflection of disease progress provided that environmental conditions are favourable. In the case of sheath blight this may reflect the way the pathogen makes progress into a growing canopy. The large variance left unaccounted for (nugget effect) in the semivariograms is a measure of the importance of processes taking place at a scale smaller than a four-hill (1 m) distance — the few centimetres that separate two tillers of the same or neighbouring hills. The distance step used in this study to assess the disease in the field and to compute the semivariances nevertheless allowed the distinction of differences in spatial patterns during the two epidemics.

The results suggest that, if the primary inoculum is distributed in a nonaggregated pattern, aggregation of disease is an indication of the occurrence of a polycyclic process in rice sheath blight. Similar conclusions were reached by Lannou & Savary (1991) on the groundnut-Rhizoctonia solani pathosystem. They sharply contrast with those pertaining to two other aerially, spore-dispersed pathogens of different pathosystems: groundnut-Puccinia arachidis and groundnut-Cercosporidium personatum, which were addressed in the same analysis (Lannou & Savary, 1991).

This study documents that while the onset of sheath blight epidemics is determined in the plant base pathozone, processes occurring in the leaf pathozone strongly determine the rates of epidemics. Simulation modelling work showed that the second, leafborne processes are more important than the first in determining terminal incidence (Savary et al., 1997). Efficient management strategies should therefore focus primarily on the control of processes occurring during the second, leafborne phase of the disease. These include biological control (Gnamanickam & Mew, 1990) and the manipulation of the crop canopy structure (Willocquet et al., 2000) and its microclimate.

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References


