ELSEVIER

Agricultural Systems, Vol. 55, No. 3, pp. 359–384, 1997

PII: S0308-521X(97)00014-0 0308-521X/97 $17.00+0.00

Modelling Sheath Blight Epidemics on Rice Tillers

S. Savary, L. Willocquet & P. S. Teng

Entomology and Plant Pathology Department, International Rice Research Institute, P.O. Box 933, 1099 Manila, Philippines

(Received 6 May 1996; accepted 4 February 1997)

ABSTRACT

The structure of a model for rice sheath blight which integrates processes at the tiller level and their contribution to epidemics is reported. The model considers two processes, primary and secondary infection, leading to disease increase. It incorporates disease aggregation in terms of accessibility of healthy tillers to infection by the pathogen from diseased tillers. Parameters from the model were derived either empirically or by numerical optimisation from published data, and from a field experiment. A separate field experiment was conducted for model evaluation. The model has the potential to adequately account for actual epidemics. Sensitivity analysis showed that the intrinsic rate of secondary infection had a large effect on simulated epidemics, while effect of the intrinsic rate of primary infection was comparatively small. The aggregation parameter had a strong effect on simulated epidemics in their later stages. The model adequately simulated the pattern of actual epidemiological data, except in the later stage of epidemics, when it failed to account for decrease in disease incidence at the tiller level. The model was considered to comply with the requirements of a preliminary simulation model, and approaches to improve its performances are discussed. © *1997 Published by Elsevier Science Ltd*

INTRODUCTION

Sheath blight, caused by *Rhizoctonia solani* Kühn, anastomosis group 1 (Gangopadyay & Chakrabarti, 1982; Ou, 1987) has become an important disease of rice, especially in intensive production systems (Otomo, 1989; Savary *et al.*, 1994). From the epidemiological viewpoint, rice sheath blight

shares characteristics with other diseases caused by *Rhizoctonia* spp. in that the primary inoculum is mainly soil-borne (Ou, 1987) while secondary inoculum does not consist of spores, but is predominantly (Gangopadyay & Chakrabarti, 1982; Ou, 1987) in the form of mycelial strands produced by primary lesions that run on the surface of leaves and sheaths to establish new lesions. As a result, epidemics usually exhibit a very strong spatial aggregation (Savary *et al.*, 1995). Two pathozones (Gilligan, 1985; Benson, 1994) might be considered: the base of the rice crop canopy, i.e. the base of tillers, where primary infections predominantly occur, and the upper part of the canopy, i.e. the upper part of the sheaths and the surface of leaves, where secondary infections, and spread, take place.

Kozaka (1961) used the terms 'vertical spread' and 'horizontal spread' to describe sheath blight epidemics. The first refers to the progress of infection along a tiller, from its base to its upper leaves by means of expanding lesions or by means of short-range progress of, and infection by, mycelial structures of the fungus. The second refers to disease spread in the crop, i.e. across tillers and rice plants (Hashiba, 1984). This is second phase is particularly influenced by contacts between host tissues (Savary *et al.*, 1995) that provide a physical bridge for the running hyphal strands to progress. Yang *et al.* (1990) coined the term 'leaf-borne disease' to describe this phase of epidemics caused by pathogens such as *Rhizoctonia* spp. This conceptualisation of the disease thus refers to a first phase where disease is increased in terms of area colonised by the pathogen, and a second one which emphasises the increase in number of host units infected. Quantitative measurement of diseases caused by *Rhizoctonia* spp., however, is difficult (Yang *et al.*, 1990; Savary *et al.*, 1995) and in the case of sheath blight several assessment methods have been developed (Sharma *et al.*, 1990*a*). The difficulty is due to the appearance of the symptoms, their rapid change over time, and the rapid decay of heavily infected tissues, especially under tropical, humid conditions.

A preliminary simulation model is an efficient way to conceptualise the structure of a pathosystem, and a useful step to both synthesise information and identify knowledge gaps (Zadoks, 1971; Teng, 1985). This paper describes a model of sheath blight epidemics that focuses on the tiller as a basic unit of observation, and integrates processes at this level towards the epidemic level. Two infection pathways are considered in order to account for the role of both the primary and secondary inoculum (*sensu* Butt & Royle, 1980), the latter consisting of lesions that are established in the canopy and are physically linked to their progeny by mycelial strands. Because of the distinctive mechanism by which the disease spreads in the canopy, not all healthy tissues available for infection are accessible to the progress of the pathogen. The concept of accessibility is introduced in the model to account for the aggregation of the disease.

MATERIALS AND METHODS

Model structure

Overall structure of the model

The system under consideration consists of a 1 m^2 rice crop, represented by a growing population of tillers. Tillers may belong to two categories: healthy (N) or diseased (Ni). Healthy tillers may become diseased through primary or secondary infection, seen as two independent processes. Primary infection results from the mobilisation of primary soilborne inoculum and occurs in the lower part of the canopy (i.e., at the base of tillers) while secondary infection takes place in the upper part of the canopy, and results from contacts between healthy and diseased tillers (especially through contacts between leaves).

Following Gilligan (1990), the rate of change of infected tillers can thus be represented by the sum of the rates of primary and secondary infections:

$$dNi/dt = (dNi/dt)_p + (dNi/dt)_s. \quad (1)$$

The rates of primary and secondary infections may be described by a monomolecular model and a logistic model, respectively (Gilligan, 1990):

$$dNi/dt = r_p P(1 - (Ni/(N + Ni)) + r_s Ni(1 - (Ni/(N + Ni)) \quad (2)$$

where r_p and r_s are intrinsic rates of primary and secondary infection, respectively (Table 1), and P is the current amount of primary inoculum. Equation (2) represents the core of the model (Fig. 1), which was complemented by additional details, and was integrated numerically using a 1-day time step.

Primary inoculum

In addition to tillers, either healthy or diseased, the system under consideration also includes soilborne propagules, i.e. any infectious units (e.g. sclerotia, mycelium, or colonised crop residues) that may initiate the establishment of (primary) infections (Butt & Royle, 1980). Disease increase is considered from the viewpoint of appearance of newly infected tillers in the system, and therefore a propagule is considered in practice as any pathogen unit that may infect an individual tiller. As a result, the dimension of the intrinsic rate of primary infection (r_p) is expressed as tiller per propagule per day (Table 1). The amount of primary inoculum (P, Table 1) is assumed

TABLE 1
List of state variables, rates, and parameters

Variable	*Meaning*	*Dimension*	*Unit*
State variables			
N	Total number of healthy tillers (per square meter of rice crop)	[Ntiller]	tiller
Ni	Total number of diseased tillers (per square meter of a rice crop)	[Ntiller]	tiller
P	Amount of inoculum (per square meter of a rice crop)	[Npropagule]	propagule
Rates			
Rpi	Rate of primary infection	[Ntiller.T^{-1}]	tiller.day^{-1}
Rsi	Rate of secondary infection	[Ntiller.T^{-1}]	tiller.day^{-1}
Rgrowth	Rate of growth of the tiller population	[Ntiller.T^{-1}]	tiller.day^{-1}
Rsen	Rate of senescence (healthy tillers)	[Ntiller.T^{-1}]	tiller.day^{-1}
Rseni	Rate of senescence (diseased tillers)	[Ntiller.T^{-1}]	tiller.day^{-1}
Recov	Rate of recovery of diseased tillers	[Ntiller.T^{-1}]	tiller.day^{-1}
Rmort	Rate of mortality of diseased tillers	[Ntiller.T^{-1}]	tiller.day^{-1}
Pdecay	Rate of decay of the primary inoculum	[Ntiller.T^{-1}]	tiller.day^{-1}
Parameters (computed by the model)			
COFR	Correction factor	[—]	healthy tiller.total $tiller^{-1}$
I	Sheath blight incidence	[—]	diseased tiller.total $tiller^{-1}$
Ntot	Total number of (living) tillers	[Ntiller]	tiller
Parameters (to estimate)			
r_p	Intrinsic rate of primary infection	[T^{-1}]	tiller.$propagule^{-1}$.day^{-1}
r_s	Intrinsic rate of primary infection	[T^{-1}]	tiller.$tiller^{-1}$.day^{-1}
a	Aggregation parameter.	[—]	—
RGR	Relative growth rate of the tiller population	[T^{-1}]	tiller.$tiller^{-1}$.day^{-1}
Nmax	Maximum tiller number (per square meter)	[Ntiller]	tiller
RRsen	Relative rate of senescence of the tiller population	[T^{-1}]	tiller.$tiller^{-1}day^{-1}$
RRecov	Relative rate of recovery from disease	[T^{-1}]	tiller.$tiller^{-1}$.day^{-1}
RRmort	Relative rate of tiller mortality due to infection	[T^{-1}]	tiller.$tiller^{-1}$.day^{-1}
RRdecay	Relative rate of primary inoculum	[T^{-1}]	propagule.$propagule^{-1}$.day^{-1}

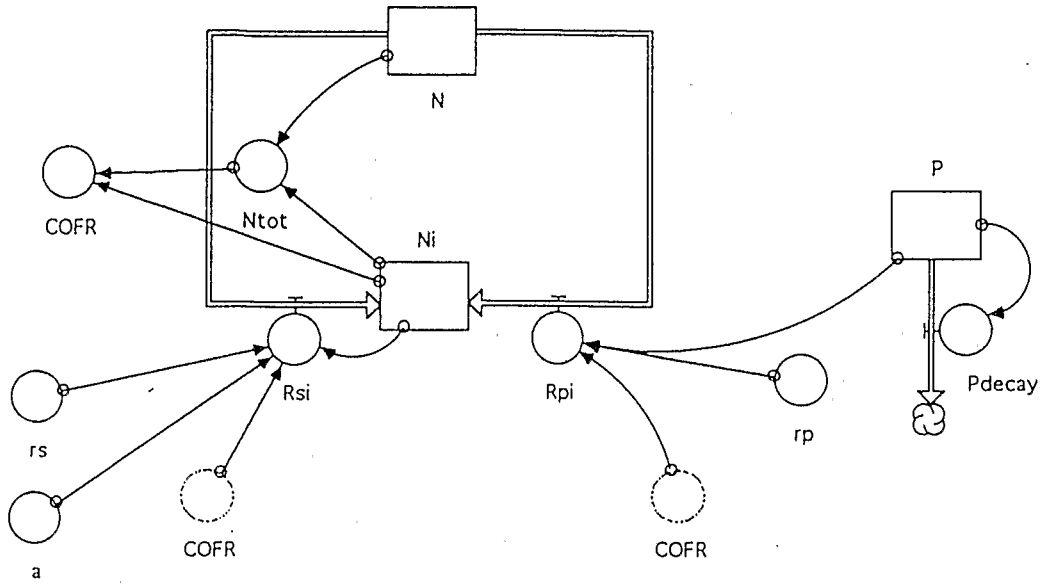

Fig. 1. Overall structure of a preliminary simulation model for rice sheath blight epidemics. Boxes indicate state variables, circles indicate parameters, double arrows indicate flows of individuals (tillers or propagules), and simple arrows indicate numerical relationships. Two flows and their rates indicate two processes of infection, converting healthy tillers (N) into diseased ones (Ni). Symbols for variables are listed in Table 1.

to decay exponentially (Gilligan, 1994) as a result of the flooding of the soil on which the crop is grown (Roy, 1986):

$$dP/dt = -RRdecayP \tag{3}$$

Disease aggregation

Equation (2) does not account for aggregation of diseased tillers. It also does not reflect the restricted accessibility of healthy tillers to infection from infected ones. Aggregation was incorporated in the model using an aggregation coefficient a:

$$dNi/dt = r_pP(1 - (Ni/(N + Ni)) + rsNi(1 - (Ni/(N + Ni))^a. \tag{4}$$

In its original form, the correction factor (Zadoks, 1971) of a logistic equation is:

$$COFR(r) = 1 - (Ni/(N + Ni)). \tag{5}$$

Here, $COFR(r)$ allows to account for the availability of healthy tissues to infection (Van der Plank, 1963; Zadoks, 1971).

The classical expression of logistic increase: $dNi/dt = r_s Ni(1 - (Ni/(N + Ni))$ is based upon the assumption of random distribution of disease, and even dispersal of the pathogen. The Poisson distribution is often appropriate for random counts (McRoberts *et al.*, 1996) and this is reflected in Gregory's (1948) multiple infection transformation. Waggoner and Rich (1981) write:

$$dNi/dn = (Ntot - Ni)/Ntot$$

where $Ntot = N + Ni$ is the total number of (living) host units (tillers), and n is the number of effective propagules, i.e. propagules that initiate new lesions. Under the hypothesis of a Poisson distribution (McRoberts *et al.*, 1996):

$$Ni = (Ntot)[1 - \exp(-n/Ntot)] \tag{6}$$

the correction factor can thus be written as:

$$COFR(r) = 1 - (Ni/(N + Ni)) = 1 - (Ni/Ntot) \tag{7}$$

or:

$$COFR(r) = \exp(-n/Ntot) \tag{8}$$

When the aerial, leaf-borne, polycyclic phase of sheath blight is considered, new infections in the canopy are generated by existing lesions, which produce running strands of mycelium. There is therefore a direct link between newly established infections, and the lesions they originate from, which can be assimilated to effective propagules. In the case of the system under consideration n may be considered as the number of individual sheath blight lesions per square meter that may produce new lesions.

Disease aggregation may result from the uneven dispersal of propagules of the pathogen or environmental factors. In the case of sheath blight, contacts between healthy and infected tissues are necessary for disease spread. The accessibility of available tissues to infection is thus limited, and as a result, sheath blight lesions are not randomly distributed over the population of tillers in an infected rice stand.

Equation (6) represents the variation of infected host units Ni with the number of effective propagules n. The larger the ratio $n/Ntot$, the closer Ni will be to the carrying capacity, $Ntot$. This ratio also reflects equal chance for any host unit to encounter effective propagules. We consider a situation where the chances of any host unit to be reached by an effective propagule are unevenly distributed over the population of host units. Introduction of

parameter a in eqn (2) as an exponent (eqn (4)) amounts to introducing this parameter in eqn (6) as:

$$Ni = (Ntot/a)[1 - \exp(-an/Ntot)] \tag{9}$$

which leads to:

$$dNi/dn = \exp(-an/Ntot). \tag{10}$$

Introducing this dimensionless parameter for aggregation a is equivalent to using a modified correction factor:

$$COFR(a) = [COFR(r)]^a. \tag{11}$$

Aggregation is characterised by $a > 1$, while $a = 1$ corresponds to the logistic correction factor. Consideration of eqn (9) leads to two interpretations for a: when $a > 1$ and with increasing n, (i) Ni approaches much faster a carrying capacity that (ii) has been reduced ($Ntot/a$).

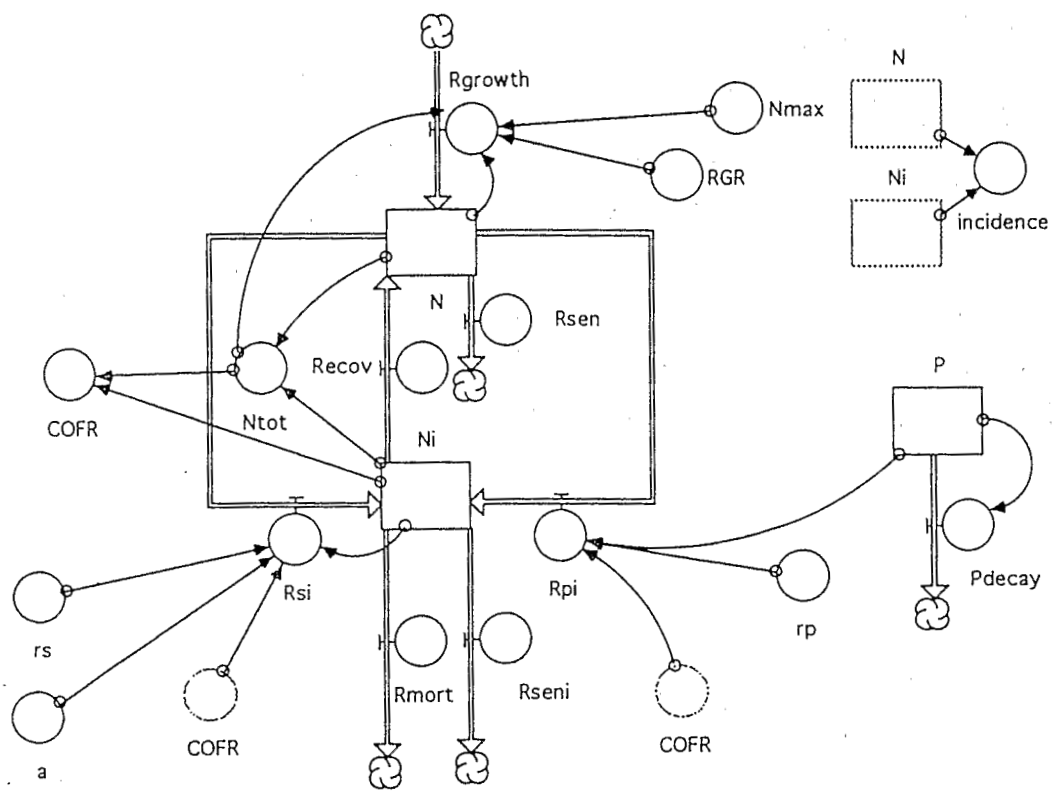

Fig. 2. Structure of a preliminary simulation model for rice sheath blight. Crop growth (tiller increase, *Rgrowth*) has been incorporated, and senescence, death, and recovery from infection are represented. Symbols for variables are listed in Table 1.

Additional components of the model

A few additional components were added to the model to account for basic characteristics of sheath blight epidemics (Fig. 2).

The size of the tiller population was made variable over time, reflecting crop growth. A logistic increase (with relative growth rate (*RGR*)) was assumed to represent adequately total tiller growth:

$$Rgrowth = dN/dt = RGRN(1 - (Ntot/Nmax)) \quad (12)$$

where *Nmax* represents the maximum number of tillers per square meter of the crop.

Tiller senescence (*Rsen*) was represented as an exponential decay function. The rate of senescence *Rsen* was thus assumed to be proportional to an intrinsic rate of senescence and to the size of the populations of healthy (*N*) or infected (*Ni*) tillers. The intrinsic rate of senescence, *RRsen*, was assumed to be the same for both healthy (*N*) and infected (*Ni*) tillers.

Severe infection on individual tillers may lead to their death, and a rate of tiller death (*Rmort*) was included. The rate of mortality was made proportional to an intrinsic rate of mortality for infected tillers (*RRmort*) and to the number of infected tillers (*Ni*):

$$Rmort = RRmort\ Ni \quad (13)$$

When environmental conditions are unfavourable to lesion expansion and multiplication, individual infections may disappear when the tissue where they are established progressively decays and senesces. Tillers may thus recover from infection, and a rate of disease recovery (*Recov*) was included. *Recov* was made proportional to a relative rate of recovery (*RRecov*) and to the number of infected tillers.

Disease incidence was computed as the percentage of diseased over total tillers:

$$I = (Ni/(N + Ni)) \times 100. \quad (14)$$

Steps in model parametrisation and development

The model was developed in four steps. Each step was associated with a new objective (verification, parameter estimation, model evaluation, and sensitivity analysis). The number of parameters determined from the previous step and set for the next one (Table 2) was progressively increased.

First, the structure of the model was encoded using the STELLA II software (Peterson & Richmond, 1994) and verification of the model (Penning de Vries, 1982) was performed, using model outputs (*N*, *Ni*, and *I*) to check the operations performed by the model. Data published by Gou *et al.* (1983) were further used as a first guideline to assess the model's behaviour with respect to incidence. In this first stage (Table 2) seven parameters were estimated empirically, either from data derived from Gou *et al.* (1983) (*Nmax*, *RGR*, and *P*), or from experimental data on tiller dynamics in artificially inoculated rice hills (*RRsen*, *RRmort*, *RRecov*, and *RRdecay*; Savary *et al.*, 1995). In addition, the three parameters of eqn (4), r_s, r_p, and a, were numerically optimised.

The second step was based on an experiment conducted at IRRI's experimental farm, where the initial spatial distribution of disease had artificially been varied. A non-inoculated control was included, allowing parameter estimates for crop growth. The outcome was a set of parameters, empirically estimated or numerically optimised. Among these were three separate, numerically optimised estimates of a, reflecting experimental treatments corresponding to three spatial disease distributions of initial disease.

The third step was based on a second, independent experiment, where the amount of initial disease represented the treatments. This data set was used to numerically compare the outputs of the model to observations, and forward judgement of the performances of the model. In this phase, the empirical estimates of parameters derived from the previous step were retained, except for *Nmax* and r_s, which were considered experiment-specific, and were empirically estimated.

In a fourth step, three parameters, r_s, r_p, and a, were successively considered in a sensitivity analysis of the model.

Field experiments

Two field experiments were used to estimate parameters and assess the model performances. Both experiments involved inoculation treatments which manipulated initial conditions that could influence the behaviour of the pathosystem, and included non-inoculated controls. The latter were used to estimate empirically parameters for crop growth (*RGR* and *Nmax*). Spontaneous epidemics also developed in the controls, and these were used to estimate some variables pertaining to the disease itself (Table 2).

Pattern of initial disease distribution

The effect of pattern of initial disease distribution on sheath blight epidemics was addressed by considering four treatments: aggregated (A), uniform (U),

TABLE 2
Steps in parameter estimation[a]

Parameter[b]	*Step 1: verification (Gou* et al., *1983)*	*Step 2: parameter estimation (first experiment: initial aggregation)*	*Step 3: model evaluation (second experiment: initial incidence)*
Nmax	A	CE	CE
RGR	A	CE	S
RRSen	L	CE	S
RRmort	L	CE	S
RRecov	L	S	S
RRdecay	L	S	S
P	R	CE	CE
r_s	O	CO	CO
r_p	O	CE	S
a	O	O	S

[a]Entries: A: Value arbitrarily chosen based on indications provided in the reference (Gou *et al.*, 1983); R: estimated empirically from the disease progress curve provided in the reference (Gou *et al.*, 1983); L: estimated empirically from the literature (*RRsen*, *RRecov*, and *RRmort*: Savary *et al.*, 1995; *RRdecay*: Roy, 1986); O: numerically optimized using the DUD iterative method of the NLIN procedure of SAS (1988); CE: estimated empirically from data collected in control plots; CO: numerically optimized from data collected in control plots; S: set after completion of the previous step;
[b]The list of parameters is given in Table 1.

and random (R) distribution of artificially inoculated rice hills, and a non-inoculated control (C). The rice variety used was IR72, and the experimental plots were transplanted on 28 June 1995. In A, U, and R, nine hills were inoculated: A consisted of a quadrat with 3×3 hills inoculated at the centre of a plot of 15×15 rice hills (with 0.2×0.2 m spacing between hills); U consisted of a regular spacing (4 hills across and along rows) between the nine inoculated hills, and in R the inoculated hills were selected at random. Inoculation of each hill was done by inserting 5 g of a rice hull/rice grain mixture (Sharma *et al.*, 1990*a*) colonised by *Rhizoctonia solani* Kühn, AGI 1a, at the base of the hill and by placing another 5 g of the mixture at mid height of the hill (Leaño *et al.*, 1993). Inoculations were performed at the maximum tillering stage, i.e. 40 days after transplanting. All plots were surrounded by a one-hill border. Each treatment had four replications arranged in a randomised complete block design. Counts of healthy and diseased tillers were made weekly on 25 hills uniformly distributed in each plot.

Amount of initial disease

The effect of amount of initial disease on sheath blight dynamics was addressed in four treatments: low, medium, and high initial disease, established by artificially inoculating rice hills, and a non-inoculated control (C). As in the previous experiment, the rice variety used was IR72, and experimental plots of the same size were transplanted at the same density on 24 July 1995. In L, M, and H, three (i.e. 1.3% of the hills); nine (4.0% of the hills); and 27 hills (12.0% of the hills), respectively, were chosen at random and were inoculated using the same technique as described above. Inoculations were performed at the maximum tillering stage, i.e. 40 days after transplanting, and all plots were surrounded by a one-hill border. The treatments were replicated four times, using a randomised complete block design. Data collection and its frequency were the same as in the experiment on initial disease distribution.

Estimation of parameters

Empirical estimation of parameters (step 1)

Roy (1986) reported a reduction in sclerotium survival by 76.5% after one month under flooded conditions. This was used to estimate *RRdecay* using eqn (3).

The article by Gou *et al.* (1983) reported data on the variation of both the proportion of tillers and the proportion of rice hills infected over time. Counts of infected rice hills at the onset of the epidemic, at a time where little within-hill and between-hill disease progress could have taken place, were

considered to reflect primary infections due to soilborne inoculum only. These were used to compute estimates of the initial amount of primary inoculum P, using the relation: $P = P(t)\exp(RRdecay \times t)$, where $P(t)$ is the residual amount of inoculum, as estimated from the observed number of initially infected hills.

Data on inoculated rice hills used as sources of inoculum to study focal expansion of sheath blight (Savary *et al.*, 1995) were used in step 1 to empirically estimate some of the parameters. In this experiment, the inoculated source hills differed in disease severity in response to varying levels of inoculum applied (2.5 or 5 g of a mixture of rice grain and rice hull colonised by the fungus) and to differing height of positioning of the inoculum in the hills (placement at the base or at mid-height of the canopy). The experiment thus involved five treatments represented by the four dose/height combinations (LB, low dose/at the base; HB, high dose/at the base; LC, low dose/at mid-canopy height; HC, high dose/at mid canopy height) and a non-inoculated control (C). It was conducted with eight replications in a randomised complete block design at the maximum tillering stage of the same cultivar, IR72, and resulted in a set of five groups of hills where severity varied from mild to very high, eight days after inoculation. The lowest and highest severities were observed in the LB and HC treatments, respectively.

Numbers of healthy and diseased tillers were monitored weekly on these rice hills for 42 days after inoculation (Δt). These data were used to estimate values for *RRsen*, *RRecov*, and *RRmort*, with the following assumptions:

- no tiller mortality was attributed to sheath blight in the LB treatment during the observation period;
- the rate of senescence was assumed the same among healthy (N) or infected (Ni) tillers;
- since no further increase in tiller number is to be expected at this development stage, variation in the number of infected tillers over this period of time was attributed to the additive combination of three processes: senescence, mortality, and recovery:

$$\Delta Ni/\Delta t = Rsen + Rmort + Recov$$

The three parameters were then estimated in steps:

- data from the uninoculated controls (C) were used to estimate the intrinsic rate of senescence: $RRsen = (\Delta N/\Delta t)(1/N)$;
- data from treatment LB (i.e. with the lowest disease severity) were used to estimate the intrinsic rate of recovery: $RRecov = (\Delta Ni/\Delta t)(1/Ni) - RRsen$; and

- data from treatment HC (i.e. with the highest severity) were used to estimate the intrinsic rate of mortality: $RRmort = (\Delta Ni/\Delta t)\ (1/Ni) - RRecov - RRsen$.

Empirical estimation of parameters (steps 2 and 3)

Control plots of the two field experiments conducted at IRRI were also used to empirically estimate parameters pertaining to crop growth and tiller dynamics (*RGR*, *RRsen*, and *RRmort* in step 2; *Nmax* in steps 2 and 3; Table 2) and to primary inoculum (r_p in step 2; P in steps 2 and 3).

As the two experiments that were conducted at the IRRI experimental farm were established in neighbouring pieces of land, with the same cropping history and in the same year, data collected from their non-inoculated controls were used jointly to estimate *RGR*, *RRsen* and *RRmort*. In both experiments, spontaneous infections from endogenous primary inoculum were observed. Controls in the first experiment (initial disease aggregation) differed from those of the second one (initial disease amount) in having a low terminal disease incidence (11.4% infected tillers) whereas that observed in the second experiment was high (23.3% infected tillers).

The intrinsic rate of tiller growth (*RGR*) was considered a genetic characteristic of the cultivar used, and was estimated from data of the first experiment, assuming a logistic increase until the maximum number of tillers was reached (approximately 45 days after transplanting). No tiller mortality was attributed to sheath blight in the controls in the first experiment, while it was assumed to occur in the controls of the second. Decrease in tiller population after maximum tillering stage was therefore attributed to senescence only in the first experiment, while the stronger decrease in tiller number observed in the controls of the second experiment was attributed to senescence and disease mortality. Using the approach outlined in step 1, *RRsen* was estimated from the controls of the first experiment. *RRmort* was then derived from the value of *RRsen* and the gross variation in tiller number in the controls of the second experiment. As these experiments did not provide data pertaining to tiller dynamics when sheath blight severity is made variable, the previously estimated value for *RRecov* was retained (Table 2). The two experiments also differed in vegetative crop growth and two parameters were used for *Nmax*, as derived from observations at maximum tillering.

While the same rate of decay of primary inoculum was used for both experiments (Table 2) estimates for P and r_p had to be made. The data collected in both experiments included the number of infected rice hills. The approach used with data published by Gou (1983) was thus also applied, and estimates for P pertaining to each experiment were derived from the control plots.

In the early stage of epidemics, i.e. when $(1 - (Ni/(N + Ni)) \sim 1$, when soilborne inoculum is in the process of being mobilised ($P \neq 0$), and when

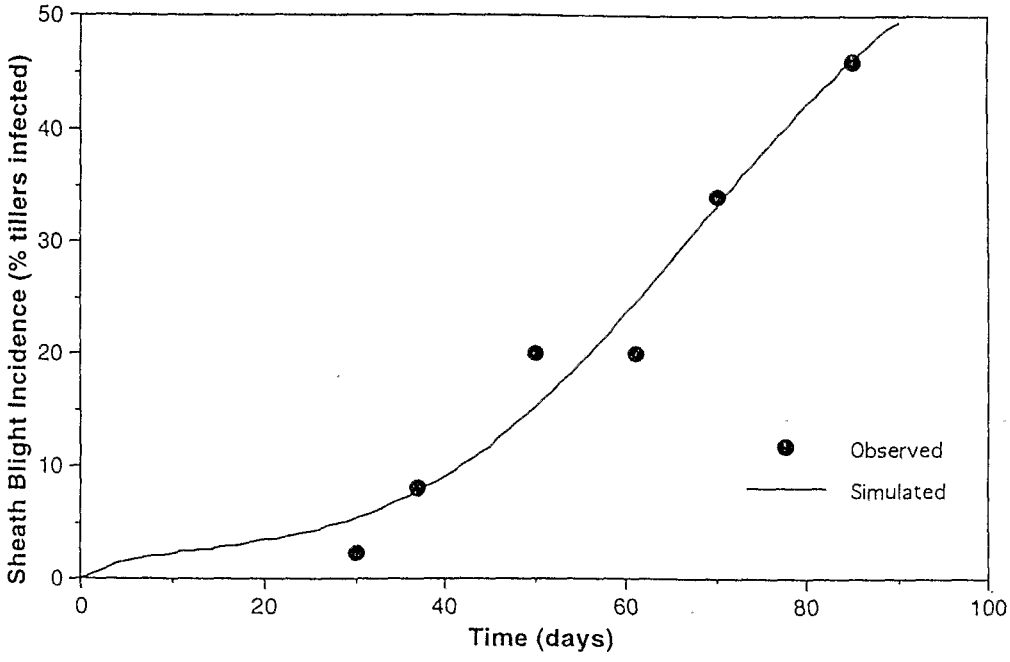

Fig. 3. Comparison of observed (Gou *et al.*, 1983, solid dots) and simulated sheath blight incidence (% diseased tillers, line).

secondary spread has not had the time to translate into visible infections ($(dNi/dt)_s \sim 0$), eqn (4) reduces to: $dNi/dt = r_p P$. Integrating this equation with respect to time allows a numerical estimate of r_p based on the initial number of infected tillers. This approach was followed in the control plots of the first experiment (initial aggregation). Because of the proximity in time and space of the two experiments, and of their similarity in cultivar and cropping practices, the process of primary inoculum mobilisation was not considered different, and the value of r_p which had been estimated for the first experiment was used as a set parameter in step 3 to simulate epidemics of the second experiment.

When simulation of specific inoculation treatments was done, numbers of inoculated tillers were introduced as initial infections superimposed on the number of infected tillers resulting from primary infections. As a rule, the hypothesis was that all tillers in an inoculated hill were infected, and the number of (artificially) infected tillers per square meter was estimated as the product of the average inoculated hills per square meter and the mean number of tillers per hill.

Numerical optimisation of parameters

Numerical optimisation of parameters was achieved with the non-linear regression (NLIN) procedure of SAS (1988). In this procedure, the DUD iterative method was used because it does not require the partial derivatives

of the model with respect to parameters to be known. Incidence was chosen as the dependent variable, as it reflects variation in both diseased tillers and crop growth. The program of the simulation model was included in the NLIN procedure, and the parameters were then optimised so as to provide the best fit between observed and simulated incidences. The convergence criterion used by the NLIN procedure is the residual sum of squares,

$$RSS = \sum_{i=1}^{k} (xo_i - xs_i)^2$$

where $i = 1, \ldots, k$ is the observation number, xs_i is the simulated value for observation i, and xo_i is the observed value for observation i.

The convergence criterion is met when additional iterations would not further reduce RSS. The default option involves a maximum of 50 iterations. In most cases, convergence was reached in 15–25 iterations. If the convergence criterion is met, NLIN produces an ANOVA of the regression, parameter estimates, asymptotically valid standard errors of estimates, and asymptotically valid confidence intervals for estimates.

In the second step of model development (Table 2) control plots of experiments conducted at IRRI were also used to generate optimised estimates of r_s using this method. With values of *RRdecay* and *RRecov* having been set in step 1, values for *Nmax*, *RGR*, *RRsen*, *RRmort*, *P*, and r_p were empirically estimated as outlined above in step 2. Incidence data from the control plots at disease onset (i.e. when $I \leq 7\%$) were then considered, that is, incidences measured in the early stage of epidemics, when disease aggregation was unlikely to have yet had a significant effect on disease spread. The value of parameter a was then arbitrarily set at a value of 1, and these subsets of incidence data were used to numerically optimise r_s.

Model evaluation

Steps 1 (model verification, Table 2) and 2 (parameter estimation) led to a simulation program, and a series of estimated parameters. The second field experiment was used to compare simulated to actual epidemics. This experiment yielded three sets of epidemics where initial disease incidence had artificially been manipulated. A first model evaluation was obtained by visually comparing the patterns of simulated and observed disease progress. Following Teng (1981) the null hypotheses of $a = 0$ and $b = 1$ in regressions of the shape: [observed incidence] = a + b [simulated incidence] were then tested. The regressions were further assessed for aptness of residual plot, coefficient of determination, and F test.

Sensitivity analysis

The response of the model to variation of some of its parameters was studied. Only the parameters that are involved in eqn (4), seen as the basis of the model, were considered, each in turn, to determine their respective influence on the behaviour of the model. Parameters that had been optimised from step 1 in the model verification phase were used for the analysis.

RESULTS

Model verification

The model allows changes in healthy and diseased tillers, and disease incidence over time to be simulated. Estimates for *RRsen* (0.0044), *RRmort* (0.0020), and *RRecov* (0.0130) were computed from data on tiller dynamics in artificially infected hills, and were incorporated in the model. An estimate for *RRdecay* (0.11) was derived from data published by Roy (1986).

An estimate of P (15 propagules m^{-2}) was derived from the data published by Gou *et al.* (1983) which gave an incidence of 3.85% infected hills 25 days after transplanting. This article did not indicate the planting density or the maximum tillering of the (hybrid) rice variety used. Considering that hybrid varieties have a lower tillering capacity than conventional, high yielding cultivars (S. S. Virmani, pers. comm.) a maximum tiller number per square meter (*Nmax*) of 500, based on 25 hills m^{-2} and 20 tillers $hill^{-1}$, was assumed.

Optimisation of r_p, r_s, and a led to a reduction of the residual sum of square to 54, out of a total sum of square of 4141. Estimates for r_p, r_s, and a were 0.038, 0.089, and 1.770, respectively, and these values were used in the model.

The simulated output using this set of parameters and observed data published by Gou *et al.* (1983) are shown in Fig. 3. The simulated curve has the shape of a polycyclic epidemic, and adequately represents the pattern of observed incidence data points.

Experiment on initial disease aggregation: numerical optimisation of a

Empirical and numerical optimisation estimates on control plots (C) yielded the following values for the experiment on initial disease aggregation: $Nmax = 945$; $RGR = 0.14$; $RRsen = 0.0066$; $RRmort = 0.0039$; $P = 24$; $r_s = 0.0575$; and $r_p = 0.0200$. The values of *RRecov* (0.0130) and *RRdecay* (0.11) were retained from the previous step.

Observed incidences in the inoculated treatments with initially aggregated, random, and uniform distribution of inoculated hills were used to produce numerically optimised estimates for a (Table 3). The convergence criterion of the optimisation procedure was met in all three treatments, with a small residual sum of squares (104, 523, 146, for the aggregated, random, and uniform treatments, respectively) compared to sum of squares accounted by the model (1162, 2544, 2556, for the aggregated, random, and uniform treatments, respectively). Table 3 indicates large estimated values for a in general. The estimate for a in the aggregated treatment is significantly larger than the estimates in the random or uniform treatments.

Experiment on amount of initial disease: model evaluation

The parameter estimate for aggregation, in the case of initially random distribution of disease ($a = 2.80$) was retained, and combined with new estimates for *Nmax* (670), P (13), and r_s (0.0466) which were estimated in the control plots.

Figure 4 shows observed incidences in all four replications of the three inoculated treatments, as well as simulated incidences with (continuous line) or without (dashed line) disease aggregation in the model. In general, as in many field experiments on sheath blight (e.g. Savary *et al.*, 1995) a very large variation in incidence at a given observation date is observed across replications. A pattern of distribution of observed incidence data can however be distinguished, with a regular increase in the first weeks following inoculation, followed by a decline. The latter appears earlier and is more pronounced with increasing numbers of inoculated hills (L to M). Omission of disease aggregation (i.e. using a default value of 1 for a) leads to a very strong overestimation of incidence, the more so when the number of inoculated hills increases.

Simulated epidemics show reasonable agreement with observed data points in terms of initial increase of sheath blight incidence. They fail, however, to show any decline. As a result, the simulations appear in fairly good agreement with observations at low level of inoculation (L) for most of the observation period, while this agreement appears acceptable in the eight first observations at medium level (M), and in the five first observations only at high level (H).

In the case of low number of inoculated hills (L), Table 4 indicates that the null hypotheses of slope and intercept being equal to 1 and 0, respectively, in the regression: [observed incidence] = a + b [simulated incidence] is not rejected at a 5% probability level. Similarly, confidence intervals for these parameters indicate that they are not significantly different from 1 and 0, respectively. The regression accounts for 42% of the variation in observed incidence, and is significant at $P < 0.001$.

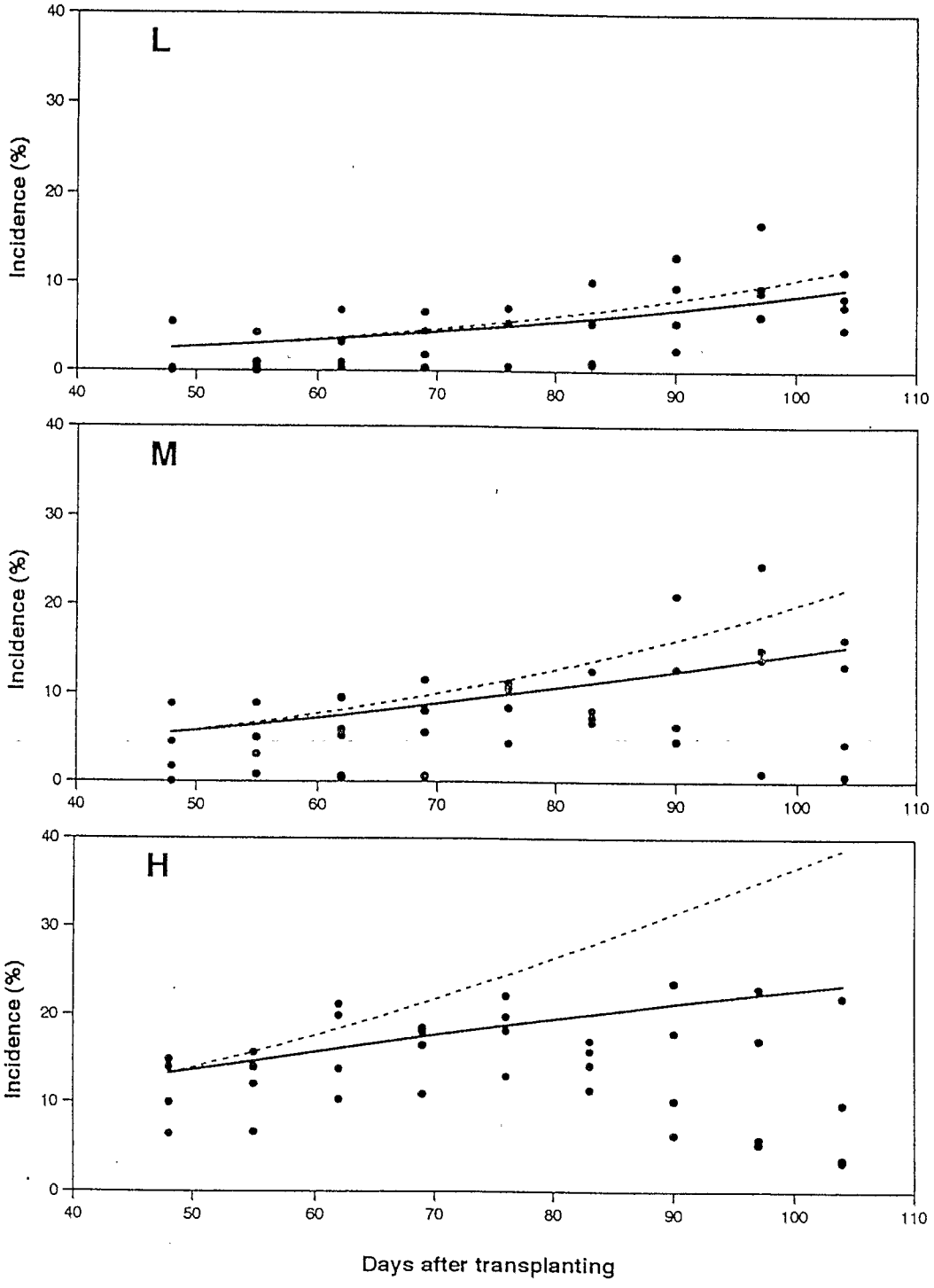

Fig. 4. Comparison of observed (dots) and simulated sheath blight incidence in a field experiment on three levels of inoculation at random with four replications. L: 3 rice hills inoculated in a 225-hill plot; M: 9 rice hills inoculated in a 225-hill plot; H: 27 rice hills inoculated in a 225-hill plot. The continuous line shows the simulated sheath blight incidence. Simulation disregarding aggregation ($a = 1$) is indicated by a dashed line.

TABLE 3

Estimates of the aggregation parameter a in an experiment involving three patterns of spatial distribution of initial disease

Treatment[a]	a[b]	$SE(a)$[c]	F[d]
Aggregated	4·72	0·95	89·0
Random	2·80	1·12	39·1
Uniform	2·18	0·45	142·0

[a]All treatments were based on the inoculation of nine hills within 15 × 15 hills plots. Aggregated treatment: the inoculated hills are grouped in a 3 × 3 quadrat at the center of the plot; Random: the inoculated hills chosen at random; Uniform: the inoculated hills are uniformly distributed over the plot.

[b]Estimated value using the DUD iterative method of the SAS NLIN procedure, using the mean value of four replicates of each treatment.

[c]Asymptotic standard error.

[d]Ratio of regression to residual mean squares.

When the treatment with medium number of inoculated hills (M) is considered, these hypotheses are rejected. The R^2 value is small, in spite of the regression being significant ($P < 0.005$), and the confidence intervals of parameters are very large. Discarding the last observation from the regression analysis however results in non-rejection of the null hypotheses ($a = 0$ and $b = 1$), higher R^2 and F, and in smaller confidence intervals of estimates for slope and intercept.

When the treatment with high number of inoculated hills (H) is considered, a non-significant regression is obtained, indicating a very poor overall description of observations by the model. Reduction of the data set to the five first observation dates, however, leads to a significant ($P = 0.009$) R^2 value, and hypotheses pertaining to slope and intercept not being rejected.

When the regressions were performed using the means of incidence data and 9, 8, and 5 observations for treatments L, M, and H, respectively, the following R^2 values were obtained: 0.84 ($F = 43.9$, $P < 0.001$), 0.96 ($F = 188$, $P < 0.001$), and 0.87 ($F = 28.1$, $P < 0.01$), and estimates for a and b were not significantly different from 0 and 1, respectively.

Sensitivity analysis

Sensitivity analysis was performed on the epidemiological parameters of eqn (4) only: r_p, r_s, and a. The estimates derived from the optimisation procedure performed in step 1 on data published by Gou *et al.* (1983), i.e.: $r_p = 0.038$, $r_s = 0.089$, and $a = 1.77$, were used as references. The values of r_p and r_s were varied by $\pm 25\%$ and $\pm 50\%$ of their estimated values. As a cannot, by definition, be smaller than 1, the following values for a were used:

TABLE 4

Comparison of simulated outputs with sheath blight incidences observed in a field experiment involving three levels of proportions of inoculated rice hills

Treatment	*Proportion of inoculated hills*	*Null hyphotheses*[a]		*Estimates of parameters ± confidence interval (P = 5%)*		*Regression*[b] */obs = a + b/sim*		
		a = 0	*b = 1*	*a*	*b*	R^2	*F*	*P*
Low	(1.3)%							
	9 observation dates	NR	NR	-2.15 ± 2.94	1.26 ± 0.51	0.42	25.8	0.000
Medium	(4.0%)							
	9 observation dates	R	R	-0.36 ± 5.86	0.27 ± 0.55	0.18	8.88	0.005
	8 first observations	NR	NR	-3.05 ± 6.11	1.14 ± 0.62	0.30	13.9	0.001
High	(12.0%)							
	9 observation dates	R	R	—	—	0.00	0.10	0.757
	5 first observations	NR	NR	-6.08 ± 15.10	1.29 ± 0.93	0.29	8.56	0.009

[a]NR: null hypothesis not rejected; R: null hypothesis rejected.

[b]/obs: observed incidence; /sim: simulated incidence.

1 (no disease aggregation throughout the epidemic), 1.39, 1.77 (value optimised on this data set), 2.16, and 2.54.

The results of simulations are shown in Fig. 5. Variation in r_p (Fig. 5A) has a relatively small effect on progress of disease incidence. Decreasing r_p from 0.057 to 0.019 does not affect the slopes of incidence progress curves and essentially results in a delay in disease progress. Owing to the duration of epidemics, the terminal incidence however is essentially the same. Increase in r_s (Fig. 5B) has a very strong effect on the slope of epidemic curves, and translates into considerable increase in successive values of disease incidence, including at the end of epidemics. Decrease in a (Fig. 5C), that is increasing the accessibility of healthy tillers to infection from lesions does not influence the shape of disease progress curves in the early stage of epidemics, but strongly affects both slopes and incidence values at a later stage.

DISCUSSION

Model evaluation

Results on model verification (step 1) are indicative of the potential of the model to mimic sheath blight epidemics on the basis of a relatively small set of hypotheses.

The results on estimates of a (step 2) with estimated a values larger than 1 illustrate the aggregated nature of sheath blight. A much higher a value is estimated when initial disease is aggregated as compared to when initial disease is randomly or uniformly distributed. This supports the concept of a reflecting the accessibility of healthy tillers to progress of the pathogen in the canopy.

Statistical comparison of observed and simulated epidemics (step 3) indicated a fair description of epidemics in their early stage by the model, and an adequate representation of initial slopes of disease incidence curves. Actual disease epidemics, however, were not well represented in the later stage of their development, especially at high incidence levels, as no decline in incidence was simulated.

Sensitivity analysis provided an overview of the flexibility of the model, with respect to the intrinsic rates of primary and secondary infection, and disease aggregation. Figures 5A and 5B are very close to simulated epidemics of root disease reported by Gilligan (1990), reflecting the similarity in model design, with two independent processes leading to infection. Figure 5C illustrates the impact of host tissue accessibility, and the shape of simulated epidemics corresponds well with empirical data such as those reported by Gilligan (1990, p. 106).

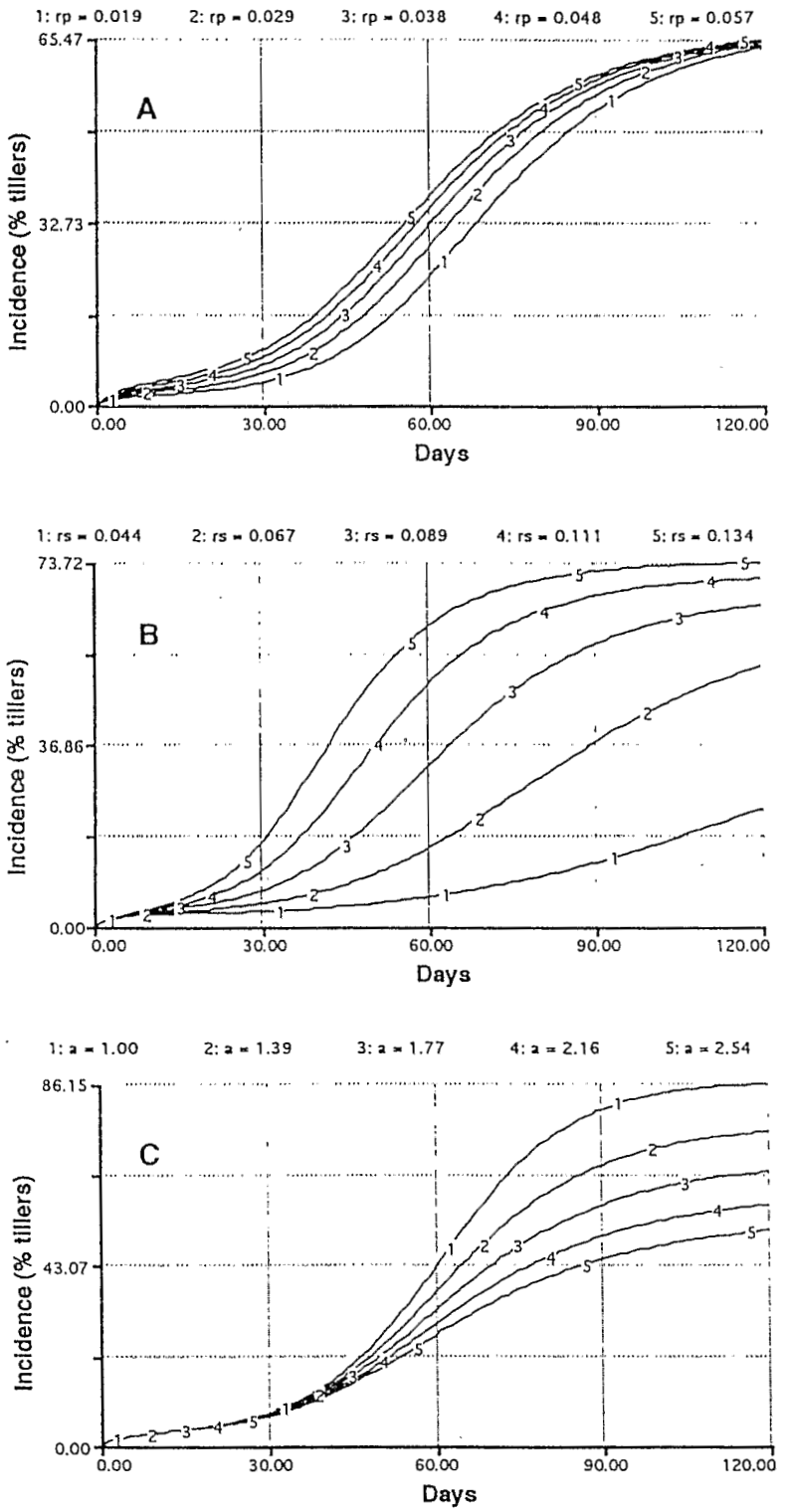

Fig. 5. Sensitivity analysis on three parameters of the model. A: intrinsic rate of primary infection (r_p), B: intrinsic rate of secondary infection (r_s), and C: aggregation coefficient (a). Parameter values (1 to 5) are shown on top of each set of curves, and correspond to figures indicated on the curves.

Key features of the structure of this model are: (i) the consideration of a growing tiller population where disease spreads, (ii) two independent infection processes, and (iii) a distinction between availability and accessibility of tillers, and the introduction of an aggregation parameter to account for the latter. The main objective of this modelling work was to implement this conceptual framework in the case of sheath blight, compare outputs with field data, and assess its value as a basis for further research. In view of its simplicity, the performances of the model may be considered to comply with the requirements of a preliminary simulation model (Penning de Vries, 1982).

Perspectives for improvement

In spite of the encouraging results shown in Fig. 3 with respect to the ability of the model to account for actual epidemics, and in Fig. 5 with respect to its flexibility, Fig. 4 illustrates the improvements that are still needed to increase the model's ability to simulate sheath blight epidemics.

The estimates for P were 15 and 24 soilborne propagules m^2 in the first and second field experiments, respectively. These figures are two orders of magnitude lower than figures commonly reported in the literature on sclerotia density in the soil (e.g. Chien *et al.*, 1963: 2 10^6 sclerotia ha^{-1}). The difference may be attributable to inoculum decay, and a survival ratio in the order of 0.01 is possible (Roy, 1986) but needs documentation. Survival of sclerotia, and more generally of propagules in the soil is an area important to the understanding of sheath blight epidemics. In addition, estimates for P were derived from initial incidences on hills. These estimates therefore integrate processes leading from soilborne propagules to primary infections.

For simplicity, this preliminary model involves fixed parameters: r_p, r_s, a, and RGR. Its structure provides a framework to address the effects of various factors on these parameters, and to study their impact on sheath blight epidemics. One good example is the intrinsic rate of secondary infection, as influenced by the variation in contacts among tillers over time (Savary *et al.*, 1995), microclimate and especially leaf wetness, nutritional status, and host physiology and development stage (Sharma & Teng, 1990*b*). Similarly, further modelling work could make r_p a function of the nutritional status of the host and development stage. Further analysis of parameter a also is necessary. Accessibility of healthy tillers to infection depends on the dispersal mechanism of the pathogen. The relationships between a and disease incidence (Yang & TeBeest, 1992) at the tiller level must be explored. Further, a can also be seen as a function of crop geometry; a difference in a values in epidemics occurring in direct-seeded and transplanted rice crops should therefore be expected.

The absence of lag period is also a simplification that needs to be assessed. As it stands, the model accumulates infected tillers until they senesce, recover, or die. There are reasons to believe that a finite infectious period exists in sheath blight (Leaño *et al.*, 1993). The effects of finite incubation and infectious periods, and effects of environmental factors such as leaf wetness on the behaviour of the model and its representativeness would be worth considering.

The decline of sheath blight incidence may be explained by several, non-exclusive causes: a change in host plant susceptibility; a decline in contact frequency towards the end of the cropping season; changes in climatic conditions; an increase in the relative rate of recovery as a response to crop development; a higher relative rate of mortality in diseased tillers when becoming older; or a shorter infectious period with increasing development stage. A better description of the later phase of sheath blight epidemics, when they may decline, could represent a good goal for further improvements of this simulation model, as the issue involves many components and relations in the pathosystem.

ACKNOWLEDGEMENTS

This study was conducted under the Joint Project on Characterization of Rice Pest Constraints between IRRI and ORSTOM (Institut Français de Recherche Scientifique pour le Développement en Coopération). We thank G. Hughes for his comments on an early draft of the manuscript.

REFERENCES

Benson, D. M. (1994) Inoculum. In *Epidemiology and Management of Root Diseases*, ed. C. L. Campbell and D. M. Benson, pp. 1–33. Springer Verlag, Berlin.

Butt, D. J. and Royle, D. J. (1980) The importance of terms and definitions for a conceptually unified epidemiology. In *Comparative Epidemiology. A Tool for Better Disease Management*, ed. J. Palti and J. Kranz, pp. 29–45. Pudoc, Wageningen.

Chien, C. C., Jong, S. C. and Chuh, C. L. (1963) Studies on the number of sclerota of rice sheath blight fungus dropped on the paddy field and difference of germinability between natural and cultured ones. *Journal of Taiwan Agricultural Research* **12**, 7–13.

Gangopadyay, S. and Chakrabarti, N. K. (1982) Sheath blight on rice. *Review of Plant Pathology* **61**, 451–460.

Gilligan, C. A. (1985) Probability models for host infection by soilborne fungi. *Phytopathology* **75**, 61–67.

Gilligan, C. A. (1990) Mathematical modelling and analysis of soilborne pathogens. In *Epidemics of Plant Diseases, Second Edition*, ed. J. Kranz, pp. 96–142. Springer Verlag, Berlin.

Gilligan, C. A. (1994) Temporal aspects of the development of root disease epidemics. In *Epidemiology and Management of Root Diseases*, ed. C. L. Campbell and D. M. Benson, pp. 148–194, Springer Verlag, Berlin,

Gou, F. S., Li, X. Q. and Xu, C. L. (1983) Study on the spatial distribution of the rice sheath blight plant in rice field and its practical implications. *Acta Phytopathologica Sinica* **13**, 27–34.

Gregory, P. H. (1948) The multiple infection transformation. *Ann. Appl. Biol.* **35**, 412–417.

Hashiba, T. (1984) Forecasting model and estimation of yield loss by rice sheath blight disease. *JARQ* **18**, 92–98.

Kozaka, T. (1961) Ecological studies on sheath blight of rice plant caused by *Pelliculariasasakii* (Shirai) S. Ito, and its chemical control. *Chugoku Agricultural Research* **20**, 1–133.

Leaño, R. M., Lapis, D. B. and Savary, S. (1993) Analysing the monocyclic process in sheath blight of rice under semi-controlled conditions. *Philippines Journal of Phytopathology* **29**, 1–16.

McRoberts, N., Hughes, G. and Madden, L. V. (1996) Incorporating spatial variability into simple disease progress models for crop pathogens. *Aspects of Applied Biology* **46**, 75–82.

Otomo, T. (1989) Damage caused by major plant diseases and plant pest forecasting program in Japan. In Crop Loss due to Disease Outbreaks in the Tropics and Countermeasures. *Tropical Agriculture Research Series* **22**, 77–80.

Ou, S. H. (1987) *Rice Diseases, Second Edition*. C. A. B. International, Farnham House, Farnham Royal, Slough.

Penning De Vries, F. W. T. (1982) Phases of development of models. In *Simulation of Plant Growth and Crop Production*, ed. F. W. T. Penning de Vries and H. H. Van Laar, pp. 20–25. Pudoc, Wageningen.

Peterson, S. and Richmond, B. (1994) *Stella II*®, Technical Documentation. High Performance Systems, Inc., Loose-leafed.

Roy, A. K. (1986) Survival of sclerotia of *Rhizoctonia solani* f. sp. *sasakii* in relation to moisture regime of soil. *Indian Phytopathology* **39**, 259–263.

SAS Institute Inc. (1988) SAS/STAT User's guide, release 6.03 edition. SAS Institute Inc. Cary, NC, pp. 1028.

Savary, S., Castilla, N. P., Elazegui, F. A., McLaren, C. G., Ynalvez, M. A. and Teng, P. S. (1995) Direct and indirect effects of nitrogen supply and disease source structure on rice sheath blight spread. *Phytopathology* **85**, 959–965.

Savary, S., Elazegui, F. A., Moody, K., Litsinger, J. A. and Teng, P. S. (1994) Characterization of rice cropping practices and multiple pest systems in the Philippines. *Agricultural Systems* **46**, 385–408.

Sharma, N. R., Teng, P. S. and Olivares, F. M. (1990) Comparison of rice sheath blight (ShB) assessment methods. *International Rice Research Newsletter* **15**, 20–21.

Sharma, N. R. and Teng, P. S. (1990) Effect of rice growth stage on sheath blight (ShB) development and yield loss. *International Rice Research Newsletter* **15**, 19–20.

Teng, P. S. (1981) Validation of computer models of plant disease epidemics: a review of philosophy and methodology. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* **88**, 49–63.

Teng, P. S. (1985) A comparison of simulation approaches to epidemic modelling. *Annu. Rev. Phytopathol.* **23**, 351–379.

Van der Plank, J. E. (1963) *Plant Diseases. Epidemics and Control.* Academic Press, New York.

Waggoner, P. E. and Rich, S. (1981) Lesion distribution, multiple infection, and the logistic increase of plant disease. *Proc. Natl. Acad. Sci. USA* **78**, 3292–3295.

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.

Yang, X. B. and TeBeest, D. O. (1992) Dynamic pathogen distribution and logistic increase of plant disease. *Phytopathology* **82**, 380–383.

Zadoks, J. C. (1971) Systems analysis and the dynamics of epidemics. *Phytopathology* **61**, 600–610.