A simulation model of rice sheath blight epidemics (I) Structure and model development

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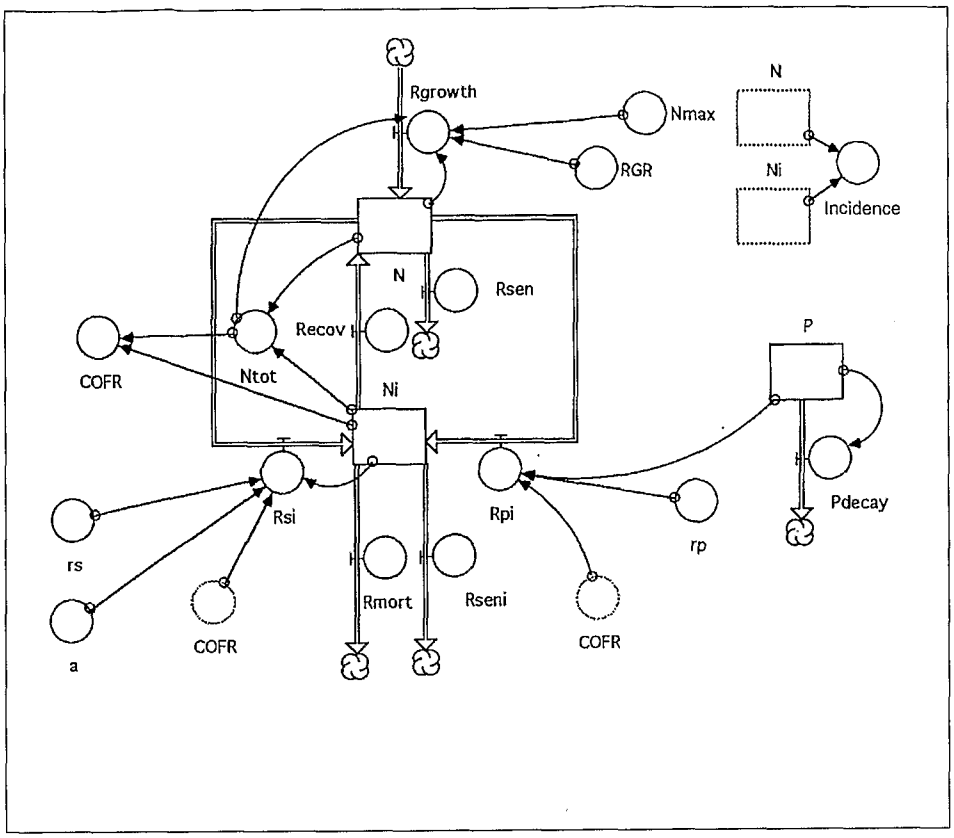
A systems model was developed to simulate rice sheath blight (ShB) epidemics over time. The model considers the two phases of ShB epidemics: mobilization of soilborne primary inoculum and secondary spread. The mechanism involved in secondary spread is a key feature of ShB epidemiology. Once infected, a tiller may in turn infect its neighbors via direct contact between leaves, which enable mycelial strands of the fungus to spread in the canopy. While the primary phase of epidemics is dependent on soilborne inoculum, the secondary phase involves leafborne inoculum and depends on crowding of canopy and climatic factors such as leaf wetness, light, and relative humidity for its multiplication.

The system under consideration in the model consists of a 1-m^2 rice crop, represented by a growing population of tillers. Tillers may belong to two categories: healthy or diseased. Two independent infection processes may lead to the accumulation of diseased tillers—primary infections generated from primary inoculum and secondary infections resulting from the progress of the pathogen in the crop canopy. The rate of change of infected tillers is thus represented by the sum of a rate of primary infection and of a rate of secondary infection: $dNi/dt=(dNi/dt)_p + (dNi/dt)_s$.

The rates of primary and secondary infections are described by a monomolecular model and a modified logistic model: $dNi/dt = r_p P(1-(N/(N+Ni)) + r_s Ni(1-N/(N+Ni))^a$ where N is the number of healthy tillers (per m^2); Ni is the number of infected tillers (per m^2); r_p and r_s are respectively intrinsic rates of primary and secondary infection; P is the current amount of primary inoculum (per m^2); and a is a parameter for disease aggregation. The structure of the model is described in the flow chart of Figure 1.



1. A systems model for rice ShB epidemics. State variables are indicated by rectangles, flows of individuals by double arrows, parameters (estimated or computed) by circles, and numerical relationships by simple arrows. The intrinsic rate of primary inoculum decay under flooded conditions was derived from Roy (1986).

The term (1-N/(N+Ni)) is commonly called correlation factor. It represents the fraction of healthy tissues (tillers)—i.e., tissues available for infection. One major underlying assumption of the model is materialized by a difference between correction factors for rates of primary and secondary infection, and the introduction of a new term, a, that reflects the limited accessibility of available (healthy) tissues for infection in the secondary phase.

The distribution of primary, soilborne inoculum is considered random due to field plowing, harrowing, and flooding, which results in a randomly distributed probability of each healthy tiller being infected by soilborne inoculum. This assumption is translated in the model by the use of a correction factor (COFR) in this simplest form for the rate of primary infection: (1 - (N/N+Ni)).

When disease becomes established in the crop and progresses within the canopy, however, the typical aggregated structure of the disease develops as a result of the short range of dispersal of the pathogen.

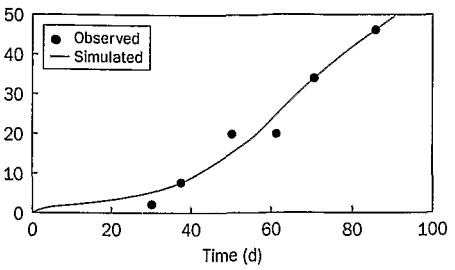
In the course of an epidemic, an additional constraint to disease multiplication therefore develops. In addition to the progressively declining proportion of tillers available to infection, there is a limited number of tillers that actually are accessible to infection from the diseased tillers. The parameter a of the correction factor for secondary infection, $(1 - (N/N+Ni))^a$, represents disease aggregation, or alternately, the limited accessibility of tillers available for infection.

The amount of primary inoculum, P, also varies over time, as result of the flooding of the soil on which the crop is grown. Variation of P is assumed to follow an exponential decay: $dP/dt=k P$.

Some additional features were added to the model and are summarized in Figure 1: 1) the size of the tiller population is made variable over time, reflecting crop growth; a logistic increase (with relative growth rate

[RGR]) was assumed to adequately represent total tiller growth; 2) tillers may recover from infection, and a rate of disease recovery (Recov) was included; 3) severe infection on individual tillers may lead to their death, and a rate of tiller death (Rmort) was included; 4) tiller senescence (Tsen) was also included, and represented as an exponential decay function. This series of equations was linked together and integrated using a 1-d time-step.

Empirical values were derived for the rates of senescence, mortality, and recovery from field experiments at IRRI. An intrinsic rate of primary inoculum decay was derived from published data (k = -0.11; Roy [1986]). Data from field experiments (Gou et al 1983) were then used as a basis for model verification, and to estimate the three parameters of the equation for the rate of infection: r_p, r_s, and a. This was achieved



2. Comparison of observed (dots) data and simulated (line) ShB incidence. Observed data were derived from Gou et al (1983).

using disease incidence (i.e., Ni/(N+Ni) as a synthetic variable representing the processes underlying a ShB epidemic, and the DUD interactive technique of the NLIN procedure of the statistical package SAS. Using this technique, parameter estimates were $r_p = 0.04$, $r_s = 0.09$, and $a = 1.77$.

The results of the simulation indicate that this system model has the potential of adequately describing ShB epidemics, and may serve as a basis for further improvement (Figure 2). Experiments are currently under way at IRRI to further assess the performance of the model and to better document r_s in terms of climatic factors and contact frequency among tillers in a rice crop.

Cited references

Gou FS, Li XQ, Xu CL. 1983. Study on the spatial distribution patterns of the rice sheath blight plant in rice field and its practical amplications. Acta Phytopathol. Sin. 13:27-34.

Roy AK. 1986. Survival of sclerotia of *Rhizoctonia solani* F. sp. *sasakii* in relation to moisture regime of soil. Indian Phytopathol. 39:259-263. ■

A simulation model of rice sheath blight epidemics (II) Model performance derived from sensitivity analysis

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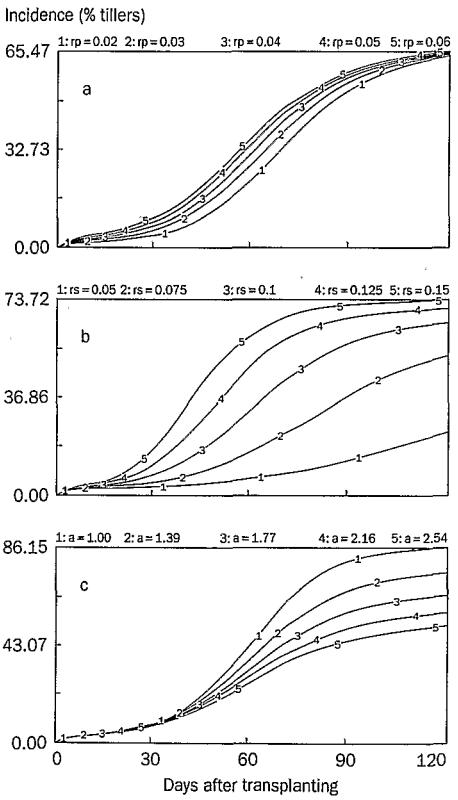
A preliminary simulation model of rice sheath blight (ShB) epidemics was developed, which incorporates a few important characteristics of the disease: a rate of primary infection (which is proportional to the amount of primary, soilborne inoculum, the amount of healthy tissues [tillers] available for new infection, and an intrinsic rate of primary infection, r_p) and a rate of secondary infection (which is proportional to the amount of infected tissues (tillers) and an intrinsic rate of secondary infection, r_s). The rate of secondary infection also depends on the amount of tissues still available for infection and on an aggregation parameter, a.

This model addresses a few research issues, such as 1) the relative effect of variation of parameter values on the behavior of the model; 2) the contribution



A series of simulation runs using a sheath blight simulation model, with varying values of three parameters: r_p (a), r_s (b), and a (c).

of primary inoculum, relative to that of secondary inoculum, resulting in spread of the disease within the rice crop canopy; and 3) the magnitude of the effect of disease aggregation on disease spread.

The figure shows a series of scenarios where the three parameters were independently varied, using optimized parameter values (r_p=0.04, r_s = 0.09, and a = 1.77) as references. A series of ShB incidence progress curves were generated by increasing or decreasing r_p and r_s by 25% or 50% of their initial values. Because a cannot, by definition, be smaller than 1, the following values were tested: 1 (random distribution of disease throughout the epidemic), 1.39, 1.77 (optimized value), 2.16, and 2.54.

Variation in r_p (Fig. a) has a small effect on the shape of disease epidemics, a decrease of r_p resulting primarily in a delay of the epidemics with the same speed. Variation in r_s (Fig. b) has strong effects on both the slopes of the curves and the terminal incidences. As expected, variation in a (Fig. c) affects the shape of curves in a later stage of disease epidemic—the larger the a value, the stronger the disease aggregation, the lower the accessibility of healthy tillers

to diseases, and the slower the epidemic when it enters the polycyclic phase.

This behavior of the model indicates that it is sensitive to variation in r_s. Future development of the model must consider the effect of time-dependent factors on variation in r_s so as to reflect variation of environment under which ShB epidemics develop. This suggests that the polycyclic nature of ShB must be considered a key characteristic of the disease for its management, and that measures taken in the course of a cropping season should prove effective. It also indicates a comparatively high sensitivity of the model to variation in the aggregation parameter, and experiments are necessary for its estimation. ■

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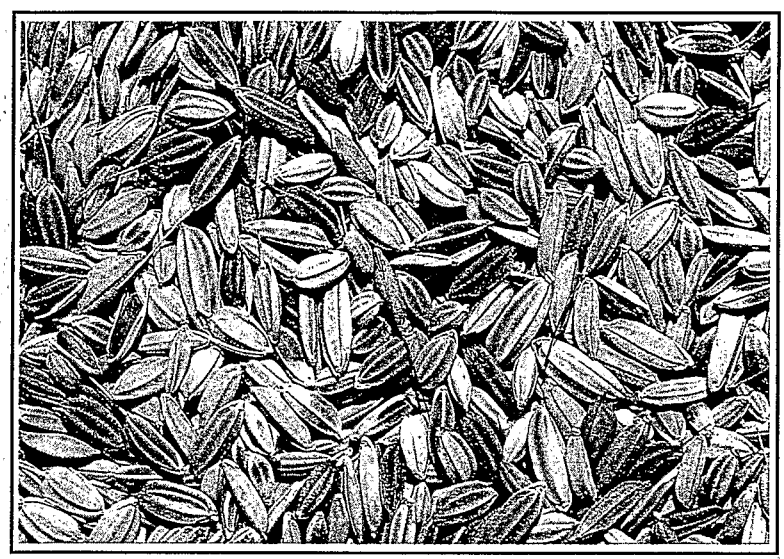

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