Section 5. Advances in Research

Chapter 2. The epidemiology of rice diseases

S. Savary, I.B. Pangga, L. Willocquet, and P.S. Teng

1. Introduction

The study of plant disease epidemics is a field of research in its own right (Madden et al 2007), which serves a number of purposes. First, plant disease epidemics, as biological phenomena, are of interest from an ecological standpoint. Plant disease epidemics have some unique features; yet, as processes, they share many similarities with human or animal epidemics. This is why modern approaches to the study of plant disease epidemics draw upon concepts used both in general ecology, population biology, and in medical and animal epidemiology (e.g., Madden et al 2007). By contrast with human epidemiology, which addresses both infectious and noninfectious diseases, botanical epidemiology essentially focuses on the former: very little research, so far, has addressed the epidemiology of noninfectious plant diseases. It has been argued that pathogens have been one of the main, and perhaps strongest, determinants of the current diversity of life (Wilson 1992, Levin et al 1999). Thus, plant disease epidemiology involves a diverse body of approaches (e.g., Zadoks and Schein 1979, Leonard and Fry 1989, Kranz 1990, Bergamin and Amorim 1996, Madden et al 2007).

Another reason for studying plant disease epidemics is their importance. Epidemics occur in natural ecosystems, sometimes with dramatic consequences to wild plant populations (e.g., Dinus 1974). However, were it not for the consequences of plant diseases on cultivated plants, plant disease epidemiology would not exist as a field of research. Crop losses in rice caused by epidemics have been known since the beginning of rice cultivation in Asia and in the modern era, have been subject to much study (IRRI 1990). The importance of diseases is discussed elsewhere in this online resource. Rice diseases indeed have been historically and are still, today, a major cause of concern (Zeigler and Savary 2009). Plant disease epidemiology studies the processes leading to disease developing in plant populations (Van der Plank 1963). The understanding of these processes may lead to disease management options. Epidemiological research also provides specific tools, operational definitions, and methods (Zadoks 1972a, Teng 1985), to identify intervention points in disease cycles. At these intervention points, control options may be used with quantifiable, predictable, efficiency.

Lastly, epidemiological research endeavors to prevent epidemics from occurring, through sanitation and prevention techniques, whose efficiencies can, in principle, be quantified and predicted (Heesterbeek and Zadoks 1987). In that respect, botanical epidemiology is very close in its approach to public health research, where it faces similar types of questions: What are the factors that may favor the emergence of new diseases? What is the likelihood of a new disease establishing in a given agroecosystem? What would the likely outcome be?

This chapter comprises a series of sections. First, we briefly introduce some basic methodological concepts. Then, the nature of plant disease epidemics, as a common phenomenon, is illustrated using some well-known rice diseases. We then present a modeling framework that may explain these observed phenomena. This modeling framework is then used to compare epidemics caused by different pathogens and to analyze the variability of epidemics caused by the same pathogen.

This relatively short chapter cannot synthesize the very large amount of research on rice disease epidemiology. Excellent reviews have been published on the general characteristics of epidemics caused by bacterial, fungal, and viral diseases of rice (Mew 1991, Teng 1994a, Hibino 1996). In this chapter, we emphasize methods and approaches to derive perspectives for future research. For this purpose, we introduce EPIRICE, a generic and simple model. EPIRICE is used both as a framework to assemble available information and guide the reader. The generic structure of EPIRICE is used to address five major rice diseases: leaf blast (RBl; **Part II, Section 1, Chapter 2**), brown spot (BSp; **Part II, Section 1, Chapter 2**), bacterial blight (BB; **Part II, Section 2, Chapter 2**), sheath blight (ShB; **Part II, Section 1, Chapter 3**), and rice tungro disease (RT; **Part II, Section 3, Chapter 1**). We chose these diseases because of (1) their global importance, (2) the biological, and especially, the epidemiological, knowledge accumulated on each of them, and (3) the diversity of plant pathogens this selection captures, thus its representativeness of the range of diseases the rice crop is exposed to, globally. These five diseases also account for a good portion of the hierarchy of plant tissues that are affected by rice diseases.

A summary of the modelling work described in this text, with application to global risk analysis for rice health, has been published by Savary et al (2012).

2. Phenomenology: the shape of rice disease epidemics

A common method to describe and provide simple quantification of epidemics is the use of a Disease Progress Curve. **ERD Figure 1** illustrates typical shapes of the disease progress curves of rice disease epidemics observed in rice fields for RBl, BSp, BB, ShB, and RT. Their common feature, as epidemics, includes a phase of increase in disease intensity over time. These epidemics, however, differ in their shapes, time span, speed, maximum intensity, occurrence of a decline phase, and the scale at which disease intensity is measured. The ShB epidemic shows a typical sigmoid shape, whereas the RBl and BB epidemics evolve in two stages, a first one with an increase of disease intensity, followed by a decline. The BSp and RT epidemics have exponential shapes.

The five epidemics strongly differ in their dynamics over time: RBl, ShB, and RT show an early onset and ShB and RBl reach very rapidly (within about 25 days) their maximum levels. The RT epidemic is slower with disease intensity still increasing 40 days after the epidemic onset. By contrast, BB and BSp epidemics start much later, at about 50 days after crop establishment.

The decline in the RBl and BB epidemics is mainly related to removal of infected tissue, associated with an accelerated leaf senescence. The decline in leaf blast epidemic is also related to the progressive decrease in susceptible tissues as plants become older. The scale at which disease intensity is measured differs among diseases. The fraction of leaf area (severity) is used for RBl and BSp, the fraction of diseased leaves (incidence at the leaf level) is used for BB, the fraction of diseased tillers (incidence at the sheath level) is used for ShB, and the fraction of diseased plants (incidence at the plant level) is used for RT. The reader is referred to Nutter et al (1991) for a detailed explanation of disease intensity terminology. These different units for scaling disease intensity correspond to different scales at which epidemiological processes take place, and will be discussed in the next sections.

3. Some main characteristics of five major rice diseases

3.1 Defining epidemiological sites

The five diseases differ in the notion of lesion (Van der Plank 1963), or site, which can be associated to them. In the case of RBl and BSp, a lesion is a fraction of leaf area colonized by the pathogen and where propagules are produced. The trophic relations between leaf pathogens and plant tissues can greatly differ (Ayres 1981) and this is the case for both RBl and BSp. Nevertheless, the epidemiology of both of these diseases can suitably be addressed considering a fraction of leaf area—the potential maximum leaf area a lesion can expand to (Jeger 1986, Bastiaans 1991)—as individual sites in the considered (crop) system.

In the case of BB, an individual leaf may well be considered a single lesion. This is because, in the case of a susceptible plant, the entire leaf blade, or a very large fraction of it, can rapidly be colonized by the pathogen (Mew 1991). The leaf itself then may become a source of inoculum to other, healthy, leaves. In the case of BB, a proper site size is an entire leaf.

By contrast, ShB develops and multiplies in ways that are quite unique, through mycelial strands of the fungal pathogen running onto sheaths, leaves, and stems, where infection cushions and penetration in the plant tissues occur (Matsuura 1986). This process may be quite rapid and, when a tiller becomes infected, it becomes quickly nearly impossible to assign an infection point to a particular infection source. In the case of ShB, it is thus convenient to define a lesion as an entire infected tiller, which may in turn infect other tillers.

RT is caused by two virus species, an RNA spherical virus and a bacilliform DNA virus (Hibino et al 1979). Infection with both virus species needs to occur in order to cause the typical RT symptoms and prior infection by the spherical virus species is required to enable infection by the bacilliform virus (Hibino 1996). Finally, both the pathogens require a vector (the green leafhopper is the most common one) to be transmitted (Rivera and Ou 1965, Hibino 1996). Viruliferous vectors are therefore necessary for disease spread in the host population. As the two viruses are systemically distributed in an infected plant, it is best in this case to consider an entire rice plant as a lesion. For both pathogens, it is at the entire plant level that infection, multiplication, and transmission take place.

Thus, the analysis of disease epidemics caused by these five different pathogens of rice lead us to consider four successive levels of sites in a hierarchy: leaf spot sites for RBl and BSp, leaf sites for BB, tiller sites for ShB, and plant sites for RT.

3.2. Overview of the epidemiological characteristics

This section provides a summary of the biology of the pathogens and the epidemiology of the five diseases.

RBl is a major rice disease on which a very large amount of epidemiological knowledge has been accumulated. RBl has a very rapid rate of increase, particularly on young leaves. It has a short latency period and a fairly long infectious period (Teng 1994b, 1996). Individual lesions on the leaves are relatively large, even though their dimensions will vary depending upon climatic conditions and, even more so, depending on the level of resistance of the host (Schlosser et al 2000); we shall consider here a susceptible host.

BSp does not quite have the same rate of multiplication as RBl does (Pannu et al 2005). The disease, however, multiplies much faster on older than young leaves (Klomp 1977). Although the quantitative, published, information is fewer than in RBl, the latency and infectious periods of the disease may roughly be considered similar to that of RBl (Sarkar and Sen Gupta 1977). Much has been written on the particular physical environ-

ment that determines the infection efficiency in BSp, including the effect of drought stress and of mineral nutrition (especially the effects of K and N; Ou 1985, Chakrabarti 2001). While these aspects certainly are very important, we do not address these here for lack of quantitative data and for the sake of simplicity. One additional feature of BSp is that the accumulation of lesions on the same leaf accelerates leaf senescence (Klomp 1977).

Compared to RBl and even BSp, much less is quantitatively known on the epidemiology of BB. Scarce field data (Adhikari et al 1999) indicate a fairly rapid rate of disease increase. The latency of the disease is relatively short (Nayak et al 1987), while the duration of the infectious period probably is fairly long. Accelerated leaf senescence is also a feature of BB.

Field-epidemiological data on rice sheath blight indicate an average rate of disease increase (Willocquet et al 2000) and experimental work indicates a short latency period and suggests a short infectious period too. One typical feature of the disease is that it produces (and progresses through) conspicuous disease foci. It thus has a very high level of aggregation (Savary et al 1995). Colonization of sheath and leaf tissues is conducive to accelerated senescence of tillers.

By contrast with the four other diseases, available literature suggests that the rate of increase of RT—at the individual plant scale—is comparatively smaller (Chancellor 1995) in spite of the fact that the latency period of the disease is small (Rivera and Ou 1965) and is related to the fact that that, once infected, a rice plant may be considered indefinitely infectious (Azzam and Chancellor 2002).

4. EPIRICE: a general modeling framework of rice disease epidemics

4.1.Plant disease epidemics as host sites progressing through successive states

A disease epidemic may be seen as the succession of transitions leading individuals belonging to a population of hosts across successive states: healthy, latent, infectious, or removed (Zadoks 1971). Although the terms for these successive states may differ among disciplinary areas (medical epidemiologists, for instance, often use 'exposed' instead of 'latent'), the same series of states has been used to analyze epidemics in human beings, animals, and plants (e.g., Diekmann and Heesterbeek 2001).

While in human and animal epidemiology, the focus is on whole individuals, plant disease epidemiology considers different plant organs, depending on the considered disease. This is because a lesion, in the epidemiological sense, may vary. The size of a lesion may span from a restricted fraction of plant tissues (e.g., of a leaf), to a whole organ (e.g., a shoot or a tiller), to an entire plant. A primary reason for plant disease epidemiologists to consider plant tissues in such a hierarchy is, therefore, the nature of the lesions the host population consists of. Another reason is that the analysis of plant disease epidemics has historically often been associated to the assessment of the damage caused by disease in a crop stand. It is worth noting that this second reason has no methodological link with our ability to model and understand epidemics. Even though the two phenomena are related, the mechanisms underlying epidemics and loss differ.

4.2. A generic epidemiological model

For the purpose of this chapter, we chose a modeling structure that would enable us to address any of five rice diseases: RBl, BSp, BB, ShB, and RT. Specifically, we want to model with the same level of detail (and therefore, of simplification) the epidemics of these five diseases with the following criteria (and thus with the corresponding constraints):

- develop as simple a model structure as possible (and therefore, accept simplifying hypotheses for the sake of generalization);
- incorporate the notions of healthy, latent, infectious, and removed sites (and thus have quantitative measurements of the latent and infectious periods);
- take into account the spatial aggregation of disease (Waggoner and Rich 1981, Jeger 1983, Hughes and Madden 1992, McRoberts et al 1996) and, therefore, parameterize aggregation and/or forward hypotheses on the level of disease aggregation;
- incorporate crop growth as a major determinant of the dynamics of epidemics (Berger 1977) and yet, represent crop growth, i.e., site population growth, in the simplest possible manner;
- incorporate the senescence of plant tissues, whether disease-induced or physiological, as a major determinant of the decline of epidemics (Kranz 1976, Berger et al 1995) and therefore apply general, simplifying hypotheses on the crop growth-epidemic linkage.

4.3. Model structure and hypotheses

The model chosen is based on the concepts developed by Van der Plank (1963) and their translation to botanical epidemiology by Zadoks (1971). The structure is based on the "epidemiological quintuplet" (Zadoks 1971, Zadoks and Schein 1979): the amount of initial inoculum (x_0), the infection efficiency of pathogen propagules (E), the amount of effective propagules produced by a lesion (N), the duration of the latency period (p), and the duration of the infectious period (p). The system considered involves four state variables: H, L, I, and P, respectively, representing the healthy, infected-latent, infected-infectious, infected-removed, and post-infectious sites of the crop stand (**ERD Table 1**). The system also involves a series of rates, including a rate of crop growth (RG), infection (RI), and senescence (RS).

We define C as the proportion of plant sites that are healthy, H, relative to the total number of plant sites, healthy or diseased (D), in the (crop) system: $C = H / (H+D)$.

A central element of the model is the rate of infection, RI, which is written as:

$$dL/dt = RI = Rc\ I\ C^a \quad \text{(Eq 1)}$$

where the rate of infected-latent sites, L, is proportional to (i), the amount of infected-infectious sites I, (ii) to a power function of C, the proportion of plant sites that are healthy relative to the total number of plant sites in the (crop) system, and (iii) to Rc, the basic infection rate corrected for removals (Van der Plank 1963). The value of the exponent parameter a is 1 when there is no aggregation of disease, i.e., when new infections occur at random among the population of healthy sites, and is larger than 1 when the horizon of infection (Van der Plank 1963) of the disease is such that propagules cannot access the entire population of healthy sites, resulting into aggregation (Waggoner and Rich 1981). The amount of tissues that are healthy, and thus available to infection, is at the center of much epidemiological work. When $a = 1$, eq 1 is the model introduced in plant disease epidemiology by Van der Plank (1963), with the strong assumption that all healthy sites (H) are equally exposed to infection – that disease is randomly distributed in the population of host sites – which is a simplification of reality (Campbell and Madden, 1990; Madden et al, 2007). This hypothesis has proven to be acceptable in a number of specific cases. When $a > 1$, equation 1 attempts to account for some heterogeneity in the spatial distribution of disease in a very simplified manner. This has drawbacks, especially when one considers that the aggregation parameter a (i) is a dimensionless parameter, which thus is difficult to

relate to a specific process (Savary et al 1997), and (ii) may not be constant throughout the course of an epidemic (Campbell and Madden 1990). The approach however has proven to be suitable in some cases (Savary et al 1997 Allorent et al 2005).

We describe the growth in a very simple, logistic manner:

$$dH/dt = RG = RRG\ H\ [1 - (TS/Sx)] \quad \text{(Eq 2)}$$

in considering that the rate of plant tissue growth, RG, is proportional to a relative rate of growth, RRG, to the amount of healthy plant tissue (i.e., of sites) that is present, and to a correction factor that depends on the total amount of sites produced at a given time, TS, relative to the maximum amount of sites, Sx, that a crop stand can produce.
The rate of senescence is described as a simple feed-forward process, whereby the amount of senesced sites, S increases over time proportionally to the amount of (healthy) tissue produced, with a constant relative rate of senescence, RRS:

$$dS/dt = RS = RRS\ H \quad \text{(Eq 3)}$$

Many diseases, in rice in particularly, are responsible for accelerated senescence of diseased tissues. This is particularly true for necrotrophic pathogens such as BSp (Klomp 1977) and ShB (Savary et al 1997), but also hemibiotrophic ones such as RBl (Bastiaans 1993). We express the fact that infected host units are encountering a faster senescence process by the equation:

$$dS/dt = RS = RP + RRS\ H \quad \text{(Eq 4)}$$

where RP is the rate of transition from the infectious to the post-infectious state of a diseased site. This equation assumes that infected-latent (L) and infected-infectious (I) sites are not affected by senescence and also that an additive process is involved in senescence, whereby disease-induced and age-induced senescence are simply added. The first hypothesis is acceptable if one considers that (i) latency and infectiousness are transitory stages only, (ii) senescence will take place at a later stage of a disease (and a crop) dynamics, and (iii) it will predominantly affect post-infectious sites that accumulate over time; it also enables a much simpler modeling algorithm. The second hypothesis, while simplifying quite complex physiological interactions, is a way to capture the indirect effect of disease on the senescence of healthy tissues. Underlying this equation is the assumption that disease-induced senescence will equally affect healthy as well as diseased (post-infectious) sites, and this further supposes a random distribution of disease amongst the population of host sites.

4.4. A diagrammatic overview of the generic model

ERD Figure 2 shows a diagrammatic overview of the modeling structure. As soon as infected (RI), a healthy site becomes latent, then becomes infectious and later on, post-infectious. The model involves two residence time parameters, the latency period duration, p, and the infectious period, i. Both parameters, p and I, are functions of many factors, including the type of disease and the level of susceptibility of the host to the pathogen. These latter two factors are considered as fixed in the course of a given epidemic. Several other factors, which will vary in the course of an epidemic, can also have strong effects on i and p: the age of the population of host sites (which may become more, or less, susceptible over time); the physiology of the host; and an array of environmental factors, including temperature, humidity, or radiation. For the sake of simplicity and genericity, we consider i and p fixed in the course of a disease epidemic.

4.5. Epidemic threshold

In essence, the above structure has been developed and discussed by several authors under a variety of forms (e.g., Teng 1985, Teng et al 1980, Savary et al 1990, Lannou 1994, Segarra et al 2001). One of the main properties of this model structure is that it enables defining a threshold for an epidemic to occur (Onstad 1992, Jeger and Van den Bosch 1994). This threshold is based on Rc, the basic infection rate corrected for removals (Van der Plank 1963) and i, the infectious period, so that when: i Rc < 1, an epidemic cannot develop.

Rc can be decomposed in two terms (Zadoks 1971): the amount N of efficient propagules produced per lesion per unit time (that is, the amount of propagules that actually are coming into contact with a given host site, diseased or not, per unit time), and the infection efficiency, E, of these propagules (that is, the fraction of effective propagules that generate a new lesion):

$$Rc = N\ E \quad \text{(Eq 5)}$$

The quantity i Rc represents the amount of effective propagules (Rc) produced by an infectious site (I) during the infectious period i. If this quantity is smaller than 1, the population of infected sites (L, I, and R) will decline and the epidemic will stop.
Several approaches to estimate Rc have been offered in the literature (Van der Plank 1963, Campbell and Madden 1990, Sun and Zeng 1993, Segarra et al 2001).
In the early stage of an epidemic:

$$r_1 = [1/ (t_2-t_1)]\ \ln(x_2/x_1) \quad \text{(Eq 6)}$$

where x_1 and x_2 are diseased fractions at two successive dates, t_1 and t_2, and r_1 is the apparent rate of disease increase.
Rc can then be estimated from:

$$Rc = r_1 / \{ [\exp(-r_1\ p) - \exp(-r_1\ [p + i])] \} \quad \text{(Eq 7)}$$

where p and i are the durations of the latency and infectious periods, respectively.

5. Comparing the epidemiology of rice diseases

5.1. Some additional detail to the structure

In the simplified framework used here to analyze and compare epidemics of rice diseases, only three driving variables of the physical and the biological environment are considered: the daily moisture of the canopy, the daily mean temperature, and the age of the crop stand.

Canopy moisture is an important factor in numerous plant diseases and there is a wealth of information on the effects of physical factors on moisture formation and duration in a rice canopy (Luo 1996). The translation of physical variables into the actual moisture of the canopy is an area of its own (Huber and Gillespie 1992) in which we do not wish to enter here. For the sake of simplicity, we applied a simple rule to represent canopy moisture (Luo 1996, Savary et al 1991): when the maximum daily relative humidity is higher than 95%, some moisture is assumed to occur in the canopy.

Temperature is also a well-known and equally important factor of epidemiological processes in many plant diseases. Temperature, in particular, affects the basic infection rate corrected for removals, Rc, as well as the latent (p) and infectious (i) period durations.

Here again, simplicity was sought: p and i were considered fixed in the course of a given epidemic, and thus only Rc was made a function of temperature.

Plant age, too, can have very strong effects on epidemiological processes (including Rc, i, and p). Again, only the effect of age on Rc is considered in the framework used here.

The effects of age, temperature, and wetness are reflected in the model by three modifiers (Loomis and Adams 1983), respectively, RcA, RcT, and RcW. These modifiers are parameters strictly bound between 0 and 1, which are multiplied to a reference, potential, value of the basic infection rate corrected for removals, RcOpt, which is derived from published data (Rc = RcOpt; Eq 7). The running value of Rc is therefore modeled as the product of the four terms:

$$Rc = RcOpt \cdot RcA \cdot RcT \cdot RcW \quad \text{(Eq 8)}$$

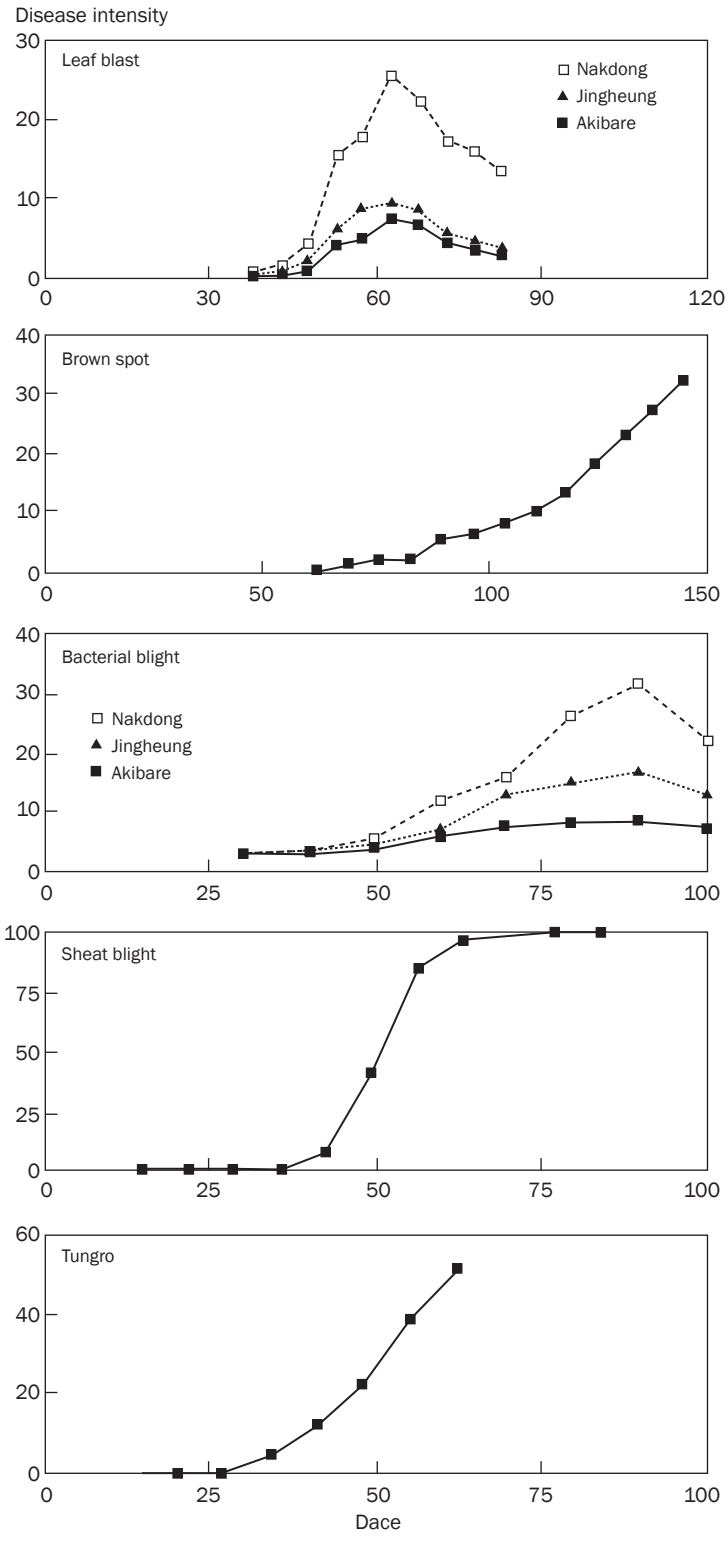
5.2. Parameterization of the model for the five diseases

Parameters and initialization values are listed in **ERD Table 1**, considering a homogeneous system of 1 m^2, and a reference crop growth duration, from crop establishment until harvest, of 120 days. Rice varieties do vary widely in their rate of development, and therefore, in their crop cycle durations (e.g., **ERD Figure 1**). We felt that a duration of 120 days was apt to capture processes that may occur in many, short- to long-duration rice varieties and that this duration was also suitable to address epidemiological processes and the resulting dynamics.

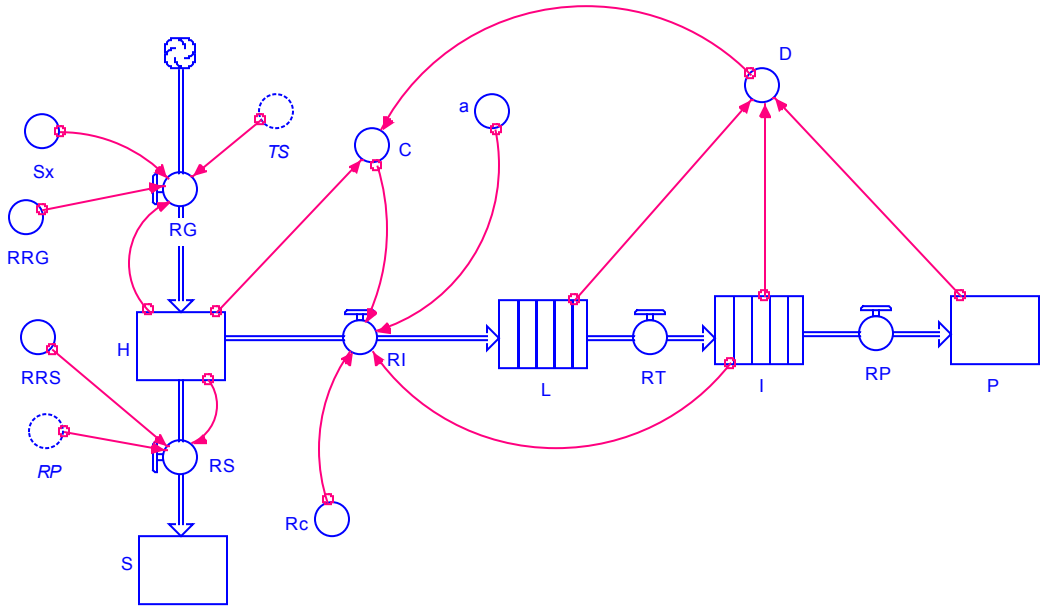
For both RBl and BSp, a maximum leaf area index of 3.5 was assumed. This was used to derive the potential maximum number of sites (fractions of leaf area, in the case of these two diseases) that could be infected in a canopy, based on the respective sizes of lesions reported in the literature. In the case of BB, the site size is one individual leaf. A maximum number of sites for BB was derived assuming a maximum number of four leaves per tiller and 800 tillers per m^2 (Yoshida 1981). The site size for ShB is considered a whole tiller and a maximum population of sites of 800 per m^2 was thus assumed (Yoshida 1981; Willocquet et al 2000, 2004). In the case of RT, where the individual site is a plant, a total population of site of 100 per m^2 was assumed. Note that the modeling structure does not make any hypothesis on the structure of the rice crop stand with respect to its establishment (i.e., whether transplanted or direct seeded). Also, while these maximum population sizes are calculated in the case of RBl and BSp, only reference values are used for diseases where the site is of a higher scale (i.e., leaves, sheaths, or plants, for BB, ShB, or RT, respectively).

The size of the host population sites is governed (**ERD Figure 2**) by two relative rates: a relative rate of growth (RRG) and a relative rate of (physiological) senescence (RRS). Values for these relative rates are indicated in **ERD Table 1**, which are derived from the literature. Again, only reference values are used here, which refer to near-optimum rice growth conditions (Yoshida 1981; Willocquet et al 2000, 2004). Both relative rates of growth and senescence are set to 0 in the case of RT with the assumptions that (1) a crop is being established at a set number of plants per m^2 (i.e., there is no growth in the number of plants after crop establishment), and (2) plants, as a whole, do develop and mature until harvest, but do not die from physiological senescence.

The values used for p and i (**ERD Table 1**) are very different from one disease to another. For RBl and BSp, we use values that are not very different from one disease to another and have been reported in the literature. These values correspond (as for the other diseases) to near-optimal conditions, including maximum host susceptibility. In the case of BB, the value for p is derived from the literature too, whereas the value for i corresponds



ERD Fig 1. Shapes of progress curves derived from the literature for five major rice diseases: leaf blast (Hwang et al 1987), brown spot (Pannu et al 2005), bacterial blight (Adhikari et al 1999), sheath blight (Willocquet et al 2000), and tungro (Chancellor 1995).



ERD Fig 2. Overall structure of EPIRICE (see text for symbols).

to the survival duration of an infected, infectious, site (in the case of this disease, an entire leaf). The i value for BB, therefore, is much longer than for the first two diseases. Similarly, in the case of ShB, the latency period is short, but the infectious period is prolonged to its maximum possible duration. The underlying hypothesis is that an infected tiller remains infectious throughout the epidemic and does not become post-infectious—destruction of tillers by ShB does occur (Savary et al 1997), but is not considered here. A same approach is used for RT, with a short latency period, and a prolonged infectious period. Again, at the plant level, an infected, infectious site remains so throughout the epidemic and plant death from RT is ignored. For all five diseases, the values RcOpt were derived using Eq 7 from published graphs and data showing rapid epidemics and from which rl values were estimated.

The variation of the modifiers for age (RcA), temperature (RcT), and canopy wetness (RcW) is derived from a range of sources (**ERD Table 1**). Each modifier is a response to varying age, temperature, and canopy wetness, and takes values comprised between 0 and 1 accordingly. Each of these functions is not discussed in detail here, but very large differences exist among diseases, of course. For instance there is a sharp contrast between RBl, where RcA decreases strongly as crop ages (Torres 1986) and BSp (Padmanabhan and Ganguli 1954), where it is the opposite. There also is a decrease in RcA with age in the case of RT (Ling and Palomar, 1975).

Note that, in **ERD Table 1**, we kept the modeling as simple as possible and primarily focused on potential epidemics. Therefore, while crop age is accounted for through variation in RcA over time, we kept both the temperature and wetness under optimal conditions for all diseases. Thus, while the framework used here has the potential of addressing daily, detailed, variation of two climatic variables, we do expand discussion on this aspect here.

ERD Table 1. EPIRICE parameter values and references.

Disease	Leaf blast	Brown spot	Bacterial blight	Sheath blight	Tungro
Site size	45 mm^2 of a leaf	10 mm^2 of a leaf	1 leaf	1 tiller	1 plant
Maximum site number	30,000	100,000	3,200	800	100
	Pinnschmidt et al (1995a)	Dasgupta and Chattopadhyay (1977)	Willocquet et al (2000, 2004)	Willocquet et al (2000, 2004)	Azzam and Chancellor (2002)
RRG	0.1	0.1	0.1	0.2	0
RRS	0.01	0.01	0.01	0.005	0
	Yoshida (1981), Willocquet et al (2000, 2004)	Yoshida (1981), Willocquet et al (2000, 2004)	Yoshida (1981), Willocquet et al (2000, 2004)	Willocquet et al (2000, 2004)	Chancellor (1995)
Date of epidemic onset	15 DACE	20 DACE	20 DACE	30 DACE	25 DACE
	Hwang et al (1987)	Pannu et al (2005)	Adhikari et al (1999)	Willocquet et al (2000)	
p	5 days	6 days	5 days	3 days	6 days
i	20 days	19 days	30 days	120 days	120 days
	Hemmi et al (1936), Kato and Kozaka (1974)	Sarkar and Sen Gupta (1977), Levy and Cohen (1980)	Nayak et al (1987), I. Oña, pers. comm.	Castilla et al (1996), N.P. Castilla, pers. comm.	Rivera and Ou (1965)
rl	0.28	0.19	0.25	0.23	0.10
	Hwang et al (1987)	Klomp (1977)	Adhikari (1991)	Savary et al (2001)	Tiongco et al (1993)
Rc	1.14	0.61	0.87	0.46	0.18
RcA	(Strong) decrease with plant age	(Strong) increase with plant age	Decrease with plant age	(Slight) increase over age	(Strong) decrease with plant age
	Torres (1986)	Padmanabhan and Ganguli (1954)	Baw and Mew (1988)	Sharma et al (1990)	Ling and Palomar (1975)
RcT	Varies with temperature	Varies with temperature	Varies with temperature	Varies with temperature	Varies with temperature
	El Refaei (1977)	Sarkar and Sen Gupta (1977)	Horino et al (1992)	Tu et al (1979)	Ling and Tiongco (1977)
RcW	1 if canopy wet, 0 otherwise	1 if canopy wet, 0 otherwise	1 if canopy wet, 0 otherwise	1 if canopy wet, 0 otherwise	Unaffected
	Luo (1996)	Luo (1996)	Mew et al (1992)	Hashiba and Hijiri (1989)	
a	1	1	1	2.8 Savary et al (1997)	1

5.3. Simulated epidemics of the five diseases

The epidemics simulated with EPIRICE for the five rice diseases are shown in **ERD Figure 3**. They indicate that this modeling framework allows capturing the variation observed in actual epidemic shapes (**ERD Figure 1**). The difference in epidemic onset, e.g., between RBl (early onset) and Bsp (late onset) is well simulated. Differences in epidemic speed are also well represented, from slow (BB) to very fast (RBl). The model allows simulating adequately epidemic decline in the case of BB and RBl.

Simulations allow the visualization of the dynamics of categories of sites which are time-consuming (e.g., healthy sites), very difficult (removed sites), or impossible (e.g. latent sites) to measure in the field. These dynamics provide, in turn, useful insight on the mechanisms underpinning epidemics and their impacts on crops. For example, the dynamics of the number of healthy sites can often be used to estimate yield losses caused by diseases (Teng 1987).

6. Examples of factors that affect plant disease epidemics

6.1. Partial resistance effects on epidemics: leaf blast

Partial resistance to disease may be seen as a series of relative resistance terms acting as breaks in disease progress (Parlevliet and Zadoks 1977, Parlevliet 1979). These relative resistance terms, often commonly called "components of resistance," can act at several stages of the epidemiological process, and thus one may consider:

- a relative resistance for infection efficiency, which reduces E: rrE,
- a relative resistance for the latency period duration p (which increases the duration of the latency period): rrp,
- a relative resistance for the infectious period duration i (which decreases the duration of the infectious period): rri, and
- a relative resistance for effective propagule formation N (which reduces the amount of propagules produced by n infectious lesion): rrN.

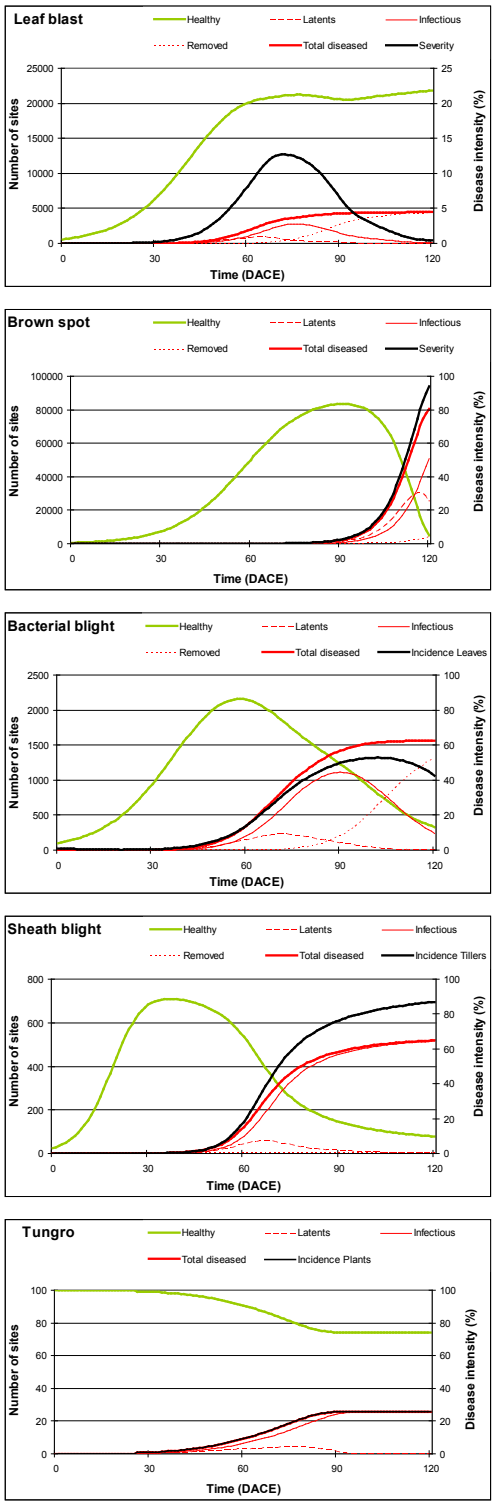
These relative resistances therefore correspond to all four elements of the epidemiological quintuplet, except x_0, the amount of initial inoculum. The relative resistance terms can be operationally defined on the basis of measurements of disease characteristics on a susceptible host check (SC) and of a test variety, which carries some partial resistance (PR) and expressed as (Zadoks 1972b):

$$rrE = 1 - (E_{PR} / E_{SC}); \; rrp = 1 - (p_{SC}/p_{PR}); \; rri = 1 - (i_{PR} / i_{SC}); \text{ and } rrN = 1 - (N_{PR} / N_{SC})$$

The effects of these relative resistances, that is, of partial resistance terms expressed in a formal way, have been analyzed, discussed, and exemplified long ago (Zadoks 1972b) in the case of leaf rust on wheat. The modeling structure enables us to address the same relative resistance in the case of RBl.

Simulated outputs suggest that relative resistance for latent period and number of *effective* propagules represent key components of resistance to target since they have the largest effects in reducing epidemics (**ERD Figure 4**). Relative resistance to the number of effective propagules can be achieved through the reduction of sporulation intensity or through the reduction of infection efficiency. Relative resistance for the infectious period has comparatively a smaller effect. These outputs illustrate how epidemiological knowledge on the monocycle of a plant disease can be up-scaled to epidemics as wholes and provide guidelines for breeding programs aimed at improving quantitative resistance.

Here, we combined sporulation intensity and infection efficiency into one parameter (NE, effective propagules). Note that improvement in the EPIRICE might separate these



ERD Fig. 3. Simulated outputs of five rice disease epidemics under near-optimal conditions. Disease intensities (vertical axis) are expressed with different units:

- **blast and brown spot: fraction of leaf area diseased, i.e., incidence at the fraction of leaf area scale;**
- **bacterial blight: fraction of leaves diseased, i.e., incidence at the leaf scale;**
- **sheath blight: fraction of tillers diseased, i.e., incidence at the tiller scale;**
- **tungro: fraction of plants diseased, i.e., incidence at the plant scale.**

two elements, which may differ in outcomes. For instance, in the case of rusts, E (infection efficiency) usually has a much stronger effect (e.g., Teng et al 1980, Savary et al 1990). In other diseases, such as *Septoria nodorum* in wheat, propagule formation (sporulation, N), which is included in NE, is more important in its effect on disease dynamics (Rapilly and Jolivet 1976).

There appears to have been much debate in the respective contribution of the different components of resistance (Bonman et al 1992, Roumen 1994), perhaps because of lack of shared methodology in defining and comparing relative resistance parameters. The infectious period has been suggested as one important factor to partial resistance (Mukherjee and Nayak 1998). This is also in agreement with the outputs of **ERD Figure 4**. Indeed, propagule formation, and its relationship with the age of a lesion, has been thoroughly analyzed in the case of leaf blast (as well as on the associated neck and collar blast, Pinnschmidt et al., 1995b). The wide variation in i value with host and environmental factors, and the ability of leaf blast to have an extended infectious period, which the latter authors reported in a very detailed study, suggest that the infectious period, and the associated rri, correspond to an important component of partial resistance in blast, in a way similar to that of *Septoria nodorum* blotch of wheat.

6.2. Inoculum aggregation effects on epidemics: sheath blight

In the case of ShB, disease transmission from an infected to a healthy tiller requires a physical contact. This explains the strongly aggregated spatial patterns in ShB epidemics (Savary et al 2001). Outputs from EPIRICE (**ERD Figure 5**) with different levels of aggregation indicate that increased aggregation slows down epidemics and that this effect increases as the epidemic develops. As a result, epidemics taper-off at lower levels when aggregation is higher. A similar effect of aggregation on ShB epidemics has been described when using a specific simulation model with a different structure, but integrating aggregation in the same way (Savary et al 1997).

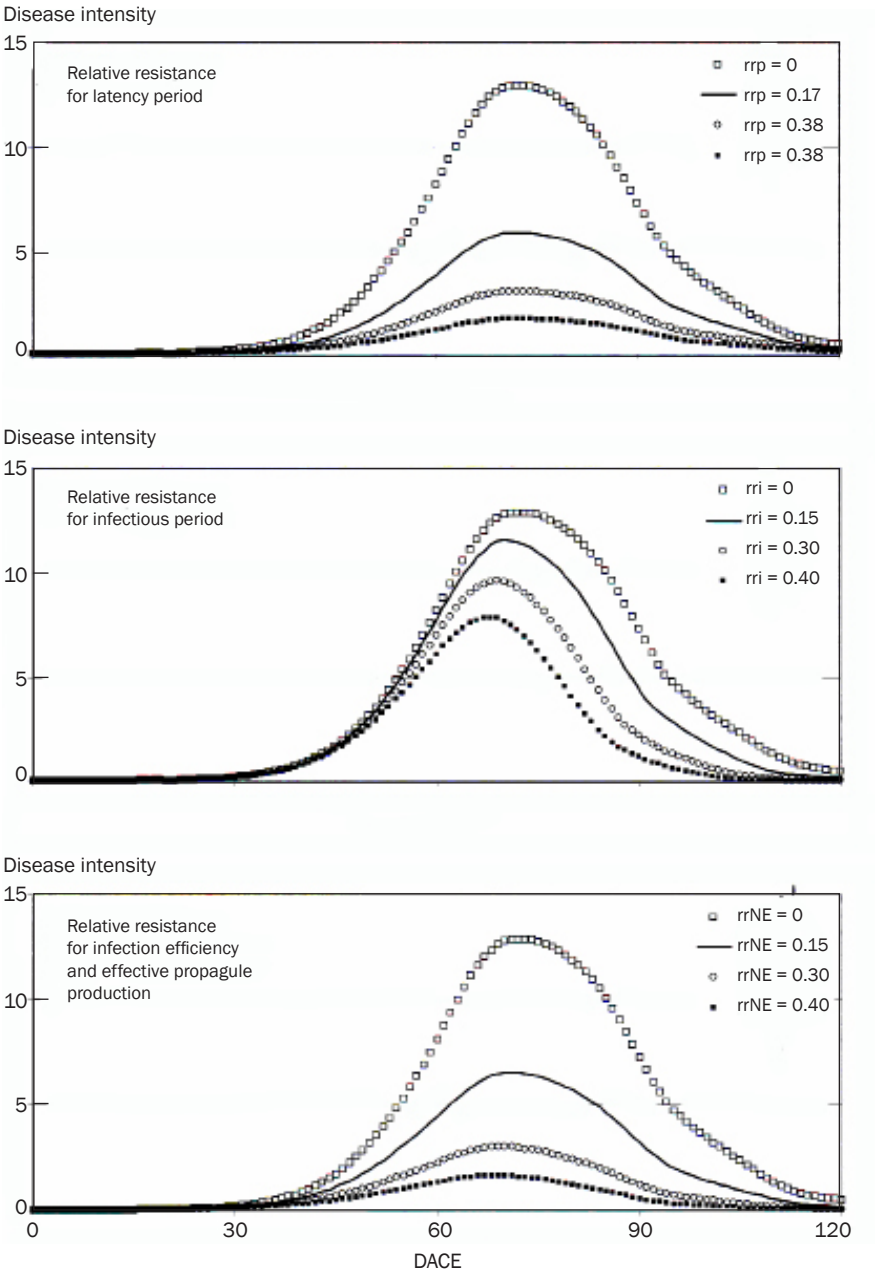
6.3. Epidemic onset time effects on epidemics: tungro

Onset of epidemics can affect greatly their final levels, particularly when the host population becomes resistant over the course of its crop cycle. This phenomenon is reflected in simulated epidemics of RT, with different epidemic onsets (**ERD Figure 6**). Simulated outputs indicate that final disease intensity is decreased by about eight-fold when disease onset is increased from 15 to 45 days after crop establishment. Such effects have been described in the case of tungro (Azzam and Chancellor 2002) and through a general mechanistic simulation of virus epidemics (Madden et al 2000). As a consequence, delay in disease onset can be considered an important entry point for management of RT epidemics, confirming several reports (e.g Azzam and Chancellor, 2002).

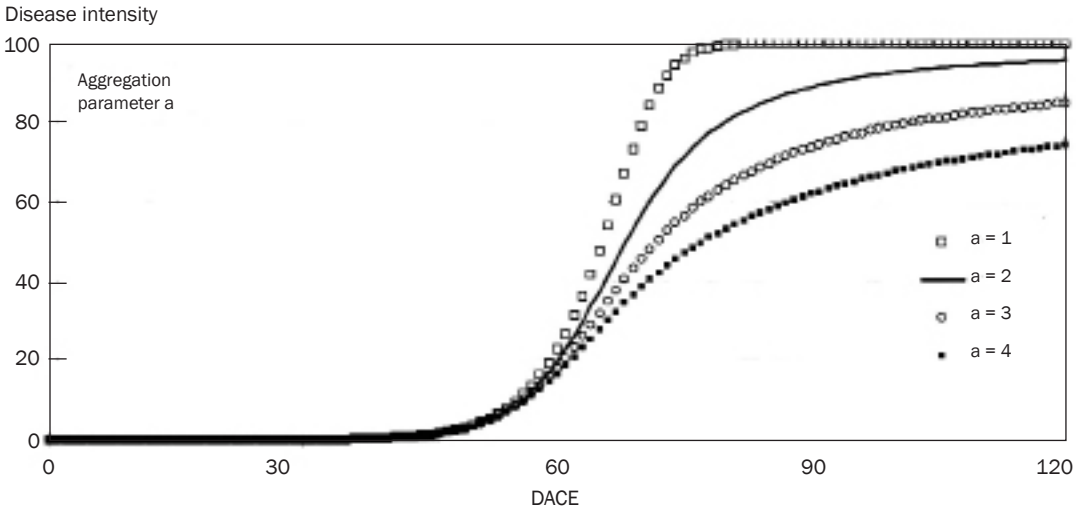
6.4. Challenges for future research

In this chapter, we focused on five rice diseases whose epidemiology have been relatively well-studied. There are still many knowledge gaps in the understanding of rice diseases, especially beyond these five. Some of these gaps represent major bottlenecks for progress in disease management, especially for the screening of host plant resistance. Other gaps hamper our vision of the future of rice diseases. These bottlenecks are summarized in **ERD Table 2**.

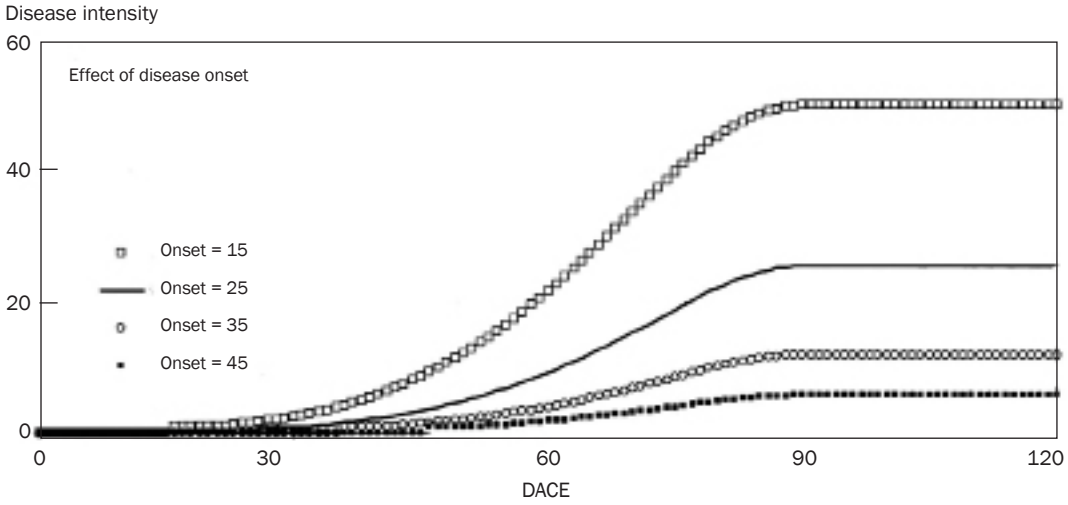
Shifts in cropping systems and cropping practices, partly in response to globalization and its socioeconomic consequences, partly in response to climate change, will have considerable effects on the epidemiology of rice diseases. Climate change alone will



ERD Fig. 4. Simulated effects of components of partial resistance to leaf blast. Disease intensity is expressed as severity, i.e., incidence at the fraction of leaf area scale.



ERD Fig. 5. Simulated effects of aggregation on sheath blight. Disease intensity is expressed as incidence at the tiller scale.



ERD Fig. 6. Simulated effects of date of epidemic onset on tungro Disease intensity is expressed as incidence at the plant scale.

generate a number of such changes. Epidemiological knowledge of rice disease therefore must be strengthened especially for diseases whose dynamics have been documented but poorly understood, as well as for diseases for which our basic biological knowledge is too poor.

Finally, although we have used EPIRICE as a modeling framework to illustrate the synthesis of epidemiologic knowledge for understanding, analyzing, and predicting behavior, models are by no means always essential to put epidemiologic knowledge to practical use in disease management. Knowledge on specific aspects of the disease cycle (components of epidemics) may often be used on their own to design appropriate management measures, and for as yet minor diseases, this may be the pragmatic approach to use.

References

Adhikari, T.B. 1991. Effects of rice genotype and environment on bacterial blight progression. PhD Thesis, University of the Philippines at Los Baños, 143 p.

Adhikari, T.B., Shrestha, A., Basnyat, R.C., Mew, T.W. 1999. Use of partial host resistance in the management of bacterial blight of rice. Plant Disease 83:896-901.

Allorent, D., Willocquet, L., Sartorato, L., Savary, S. 2005. Quantifying and modelling the mobilisation of inoculum from diseased leaves and defoliated tissues in epidemics of angular leaf spot of bean. European Journal of Plant Pathology. 113:377-394.

Ayres, P.J. 1981. Effects of disease on the physiology of the growing plant. Cambridge University Press.

Azzam, O., Chancellor, T.C.B. 2002. The biology, epidemiology, and management of rice tungro disease in Asia. Plant Disease 86:88-100.

Bastiaans, L. 1991. Ratio between virual and visual lesion size as a measure to describe reduction in leaf photosynthesis of rice due to leaf blast. Phytopathology 81:611-615.

Bastiaans, L. 1993. Effects of leaf blast on growth and production of a rice crop. 1. Determining the mechanism of yield reduction. Netherlands Journal of Plant Pathology 99:323-334.

Baw, A., Mew, T.W. 1988. Scoring systems for evaluating rice varietal resistance to bacterial blight (BB): lesion size by growth stage. IRRN 13:10-11.

Bergamin, F.A., Amorim, L. 1996. Doenças de Plantas Tropicais: Epidemiologia e Controle Econômico. Ceres, São Paulo, Brazil.

Berger, R.D., 1977. Application of epidemiological principles to achieve plant disease control. Annual Review of Phytopathology 15:165-183.

Berger, R.D., Hau, B., Weber, G.E., Bacchi, L.M.A., Bergamin Filho, A., Amorim L. 1995. A simulation model to describe epidemics of rust of Phaseolus beans: I. Development of the model and sensitivity analysis. Phytopathology 85:715-721.

Bonman, J.M., Khush, G.S., Nelson, R.J. 1992. Breeding for resistance to pests. Annual Review of Phytopathology 30:507-528.

Campbell, C.L., Madden, L.V. 1990. Introduction to Plant Disease Epidemiology. John Wiley & Sons, New York. 532 p.

Castilla, N. P., Leano, R. M., Elazegui, F. A., Teng, P. S., Savary, S. 1996. Effects of plant contacts, inoculation pattern, leaf wetness regime, and nitrogen supply on inoculum efficiency in rice sheath blight. Journal of Phytopathology 144:187-192.

Chakrabarti, N.K., 2001. Epidemiology and disease management of brown spot of rice in India. Pp. 293-306 in: S. Sreenivasaprasad and R. Johnson, eds. Major Diseases of Rice: Recent Advances. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Chancellor, T.C.B. 1995. The ecology of the leafhopper vactors of rice tungro viruses in relation to the dynamics of tungro disease. PhD Thesis, University of Reading, 226 p.

Dasgupta, M. K., Chattopadhyay, S. B. 1977. Effect of environmental factors on the development of brown spot of rice caused by ***Helminthosporium oryzae***. Journal of the Society of Experimental Agriculture 2:24-27.

Diekmann, O., Heesterbeek, J.A.P., 2000. Mathematical Epidemiology of Infectious Diseases – Model Building, Analysis and Interpretation. John Wiley and Sons, Chichester, 303 p.

Dinus, R.J. 1974. Knowledge about natural ecosystems as a guide to disease control in managed forests. Proc. Natl. Acad. Sci. USA 1:184-190.

El-Refaei, M.I. 1977. Epidemiology of rice blast disease in the tropics with special reference to the leaf wetness in relation to disease development. PhD Thesis, Indian Agricultural Research Institute, New Delhi, 195 p.

Hashiba, T., Ijiri, T. 1989. Estimation of yield loss and computerized forecasting system (BLIGHTAS) for rice sheath blight disease. Tropical Agricultural Research Series (Japan) 22:163-171.

Heesterbeek, J.A.P., Zadoks, J.C., 1987. Modelling pandemics of quarantine pests and diseases: problems and perspectives. Crop Protection 6:211-221.

Hemmi, T., Abe, T., Ikaya, J., Inoue, Y. 1936. Studies on the rice blast disease. IV. Relation of the environment to the development of blast disease and physiologic specialization in the rice blast fungus. Materials for Rural Improvement, Department of Agriculture and Forestry, Japan No. 105, 145 p.

Hibino, H, Saleh, N., Roechan, M. 1979. Transmission of two kinds of rice tungro-associated viruses by insect vectors. Phytopathology 79:1266-1268.

Hibino, H., 1996. Biology and epidemiology of rice viruses. Annual Review of Phytopathology 34:249-274.

Horino, O., Mew, T.W., Yamada, T. 1982. The effect of temperature on the development of bacterial leaf blight on rice. Annals of the Phytopathological Society of Japan 48:72-75.

Huber, L., Gillespie, T.J. 1992. Modelling leaf wetness in relation to plant disease epidemiology. Annual Review of Phytopathology 30:553-577.

Hughes, G., Madden, L.V. 1992. Analyzing epidemics in time and space. Phytopathology 41:657-660.

Hwang, B.K., Koh, Y.J., Chung, H.S. 1987. Effects of adult-plant resistance on blast severity and yield of rice. Plant Disease 71:1035-1038.

IRRI. 1990. Crop loss assessment in rice. International Rice Research Institute, Los Baños, Philippines.

Jeger, M.J., 1983. Analyzing epidemics in time and space. Plant Pathology 32:5-11.

Jeger, M.J., 1986. Asymptotic behaviour and threshold criteria in model plant disease epidemics. Plant Pathology 35:355-361.

Jeger, M.J., Van den Bosch, F. 1994. Threshold criteria for model plant disease epidemics. I. Asymptotic results. Phytopathology 84:24-27.

Kato, H, Kozaka, T. 1974. Effect of temperature on lesion enlargement and sporulation of *Pyricularia oryzae* in rice leaves. Phytopathology 64:828-830.

Kranz, J. 1976. Effect of some plant diseases on the loss of leaves. Zeitschrift Fur Pflanzenkrankheiten Und Pflanzenschutz 72:1373-1377.

Kranz, J., ed. 1990. Epidemics of plant diseases. Second Ed. Springer Verlag, Berlin

Klomp, A.O. 1977. Early senescence of rice and *Dreschslera oryzae* in the Wageningen Polder, Surinam. PhD thesis, Wageningen University, 97 p.

Lannou, C., de Vallavieille-Pope, C., Goyeau, H. 1994. Host mixture efficacy in disease control: effects of lesion growth analyzed through computer-simulated epidemics. Plant Pathology 43:651-652.

Levin, B.R., Lipsitch, M., Bonhoeffer, S. 1999. Evolution and disease: population biology, evolution, and infectious disease: Convergence and synthesis. Science 283:806-809.

Levy, Y., Cohen, Y. 1980. Sporulation of *Helminthosporium turcicum* on sweet corn: Effects of temperature and dew period. Canadian Journal of Plant Pathology 2:65-69.

Leonard, K.J. Fry, W.E., eds. 1989. Plant Disease Epidemiology. Vol 2. Genetics, Resistance, and Management. MacGraw-Hill, New York.

Ling, K.C., Palomar, M.K. 1975. Studies on rice plants infected with the tungro virus at different ages. Philippines Agriculturist 50:165-177.

Ling, K.C., Tiongco, E.R. 1977. Effect of temperature on the transmission of rice tungro virus by *Nephotettix virescens.* Philippines Phytopathology 11:46-57.

Loomis, R.S., Adams, S.S. 1983. Integrative analysis of host-pathogen relations. Annual Review of Phytopathology 21:341-362.

Luo, W.H. 1996. Simulation and measurement of leaf wetness formation in paddy rice crops. PhD Thesis, Wageningen Agricultural University. 87 p.

Madden, L.V., Jeger, M.J., Van den Bosch, F. 2000. A theoretical assessment of the effects of vector-virus transmission mechanism on plant virus epidemics. Phytopathology 90:576-594.

Madden, L.V., Hughes, G., Van den Bosch, F. 2007. The study of plant disease epidemics. The American Phytopathology Press. St. Paul, Minnesota, USA. 421 p.

Matsuura, K. 1986. Scanning electron microsocpy of the infection process of *Rhizoctonia solani* in leaf sheaths of rice plants. Phytopathology 76:811-814.

McRoberts, N., Hughes, G., Madden, L.V. 1996. Incorporating spatial variability into simple disease progress models for crop pathogens. Aspects of Applied Biology 46:1-8.

Mew, T.W. 1991. Disease management in rice. Pp. 279-299 in: CRC Handbook of Pest Management in Agriculture 2nd Edition. Vol. III, CRC Press, Inc. Boca Raton, Ann Arbor, Boston.

Mew, T.W., Vera Cruz, C.M., Medalla, E.S. 1992. Changes in race frequency of *Xanthomonas oryzae* pv. *oryzae* in response to rice cultivars planted in the Philippines. Plant Disease 76:1029-1032.

Mukherjee, A.K., Nayak, P. 1998. Sporulation capacity as a component of slow-blasting resistance in rice. Oryza 35:82-85.

Nayak, P., Suriya Rao, A.V., Chakrabarti, N.K. 1987. Components of resistance to bacterial blight disease of rice. Journal of Phytopathology 119:312-318.

Nutter, F.W., Jr., Teng, P.S., Shokes, F.M. 1991. Disease assessment terms and concepts. Plant disease 75:1187-1188.

Onstad, D.W., 1992. Evaluation of epidemiological thresholds and asymptotes with variable plant densities. Phytopathology 82:1028-1032.

Ou, S.H. 1985. Rice diseases, Second Ed. C.A.B. International, Farnham House, Farnham Royal, Slough. 380 p.

Padmanabhan, S.Y., Ganguli, D. 1954. Relation between the age of rice plant and its susceptibility to ***Helminthosporium*** and blast disease. Proceedings of the Indian Academy of Sciences B. 29:44-50.

Pannu, P.P.S., Chahal, S.S., Kaur, M., Sidhu, S.S. 2005. Influence of weather variables on the development of brown leaf spot caused by ***Helminthosporium oryzae*** in rice. Indian Phytopathology 58:489-492.

Parlevliet, J.E., Zadoks, J.C., 1977. The integrated concept of disease resistance: a new view including horizontal and vertical resistance in plants. Euphytica 26:5-21.

Parlevliet, J.E. 1979. Components of resistance that reduce the rate of epidemic development. Annual Review of Phytopathology 17:203-222.

Pinnschmidt, H.O., Batchelor, W.D., Teng, P.S. 1995a. Simulation of multiple species pest damage in rice using CERES-rice. Agricultural Systems 48:193-222.

Pinnschmidt, H.O., Bonman, J.M, Kranz, J. 1995b. Lesion development and sporulation of rice blast. Zeitschrift fur Pflanzenkrankheiten und Pflanzenschutz 102:299-306.

Rapilly, F., Jolivet, E. 1976. Construction d'un modèle (EPISEPT) permettant la simulation d'une épidémie de Septoria nodorum Berk. sur blé. Revue de Statistique Appliquée 24:31-60.

Roumen, E.C. 1994. A strategy for accumulating genes for partial resistance to blast in rice within a conventional breeding program. Pp. 245-265 in: R.S. Zeigler, S.A. Leong, P.S. Teng, eds. Rice Blast Disease. CAB International and International Rice Research Institute, Wallingford, U.K. and Los Baños, Philippines.

Rivera, C.T., Ou, S.H. 1965. Leafhopper transmission of tungro disease of rice. Plant Disease Reporter 49:127-131.

Sarkar, A.K., Sen Gupta, P.K. 1977. Effect of temperature and humidity on disease development and sporulation of *Helminthosporium oryzae* on rice. Indian Phytopathology 30:258-259.

Savary, S., De Jong, P.D., Rabbinge, R., Zadoks, J.C. 1990. Dynamic simulation of groundnut rust, a preliminary model. Agricultural Systems 32:113-141.

Savary, S., Willocquet, L., Teng, P.S. 1997. Modelling sheath blight epidemics on rice tillers. Agricultural Systems 55:359-384.

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., Castilla, N.P., Willocquet, L. 2001. Analysis of the spatio-temporal structure of rice sheath blight epidemics in a farmer's field. Plant Pathology 50:53-68.

Savary, S., Nelson, A., Willocquet. L., Pangga, I., Aunario, J. 2012. Modelling and mapping potential epidemics of rice diseases globally. Crop Protection 34:6-17.

Schlosser, I., Kranz, J., Bonman, J.M. 2000. Characterization of plant type and epidemiological development in the pathosystem "upland rice/rice blast" (***Pyricularia grisea***) by means of multivariate statistical methods. Zeitschrift Fur Pflanzenkrankheiten Und Pflanzenschutz 107:12-32.

Segarra, J., Jeger, M.J., Van den Bosch, F. 2001. Epidemic dynamics and patterns of plant diseases. Phytopathology 91:1001-1010.

Sharma, N.R., Teng, P.S., Olivares, F.M. 1990. Effect of inoculum source on sheath blight (ShB) development. IRRN 15:18-19.

Sun, P., Zeng, S. 1993. On the measurement of the corrected basic infection rate. Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz. 101:297-302.

Teng, P.S., Blackie, M.J., Close, R.C. 1980. Simulation of the barley leaf rust epidemic: structure and evaluation of BARSIM I. Agricultural Systems 5:85-103.

Teng, P.S., 1985. A comparison of simulation approaches to epidemic modeling. Annual Review of Phytopathology 23:351-379.

Teng, P.S., 1987. Quantifying the relationship between disease intensity and yield loss. pp. 105-113 In: Crop Loss Assessment and Pest Management. P.S. Teng (Ed), APS Press, St Paul MN.

Teng, P.S. 1994a. Integrated pest management in rice. Experimental Agriculture 30:115-137.

Teng, P.S. 1994b. The epidemiological basis for blast management. Pp. 409-433 in: Rice Blast Disease. R.S. Zeigler, S.A. Leong, P.S. Teng, eds. CAB International and International Rice Research Institue, Wallingford, U.K. and Philippines.

Tiongco, E.R., Cabunagan, R.C., Flores, Z.M., Hibino, H., and Koganezawa, H. 1993. Serological monitoring of rice tungro disease development in the field: its implication in disease management. Plant Disease 77:877-882.

Torres, C.Q. 1986. Effect of plant age on the expression of resistance to ***Pyricularia oryzae*** Cav. in upland rice varieties. PhD Thesis, University of the Philippines at Los Baños. 82 p.

Tu, C.C., Chang, Y.C., Wang, C.W. 1979. Studies on ecology of ***Rhizoctonia solani***, the causal organism of sheath blight of rice. National Science Council Monthly 7:1208-1219.

Van der Plank, J.E. 1963. Plant diseases. Epidemics and control. Academic Press, New York. 349 p.

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

Wilson, E.O., 1992. The Diversity of Life. Penguin Science, London. 406 p.

Willocquet, L., Fernandez, L., Savary, S. 2000. Effect of various crop establishment methods prac-

tised by Asian farmers on epidemics of rice sheath blight caused by ***Rhizoctonia solani***. Plant Pathology 49:346-354.

Willocquet, L., Elazegui, F.A., Castilla, N.P., Fernandez, L., Fischer, K. S., Peng, S., Teng, P. S., Srivastava, R. K., Singh, H. M., Zhu, D., Savary, S. 2004. Research priorities for rice disease and pest management in tropical Asia: a simulation analysis of yield losses and management efficiencies. Phytopathology 94:672-682.

Yoshida, S. 1981. Fundamentals of rice crop science. International Rice Research Institute, Los Baños, Philippines.

Zadoks, J.C. 1971. Systems analysis and the dynamics of epidemics. Phytopathology 61:600-610.

Zadoks, J.C. 1972a. Methodology in epidemiological research. Annual Review of Phytopathology 10:253-276.

Zadoks, J.C., 1972b. Modern concepts in disease resistance in cereals. In: Lupton, F.A.G.H., Jenkins, G. & Johnson, R., Eds. The Way Ahead in Plant Breeding. Eucarpia, Cambridge

Zadoks, J.C., Schein, R.D. 1979. Epidemiology and Plant Disease Management. Oxford University Press, New York, 427 p.

Zeigler, R.S., Savary, S. 2009. Plant disease and world dependence on rice. In: Chapter 1, R.N. Strange, M.L. Gullino, eds. The Role of Plant Pathology in Food Safety and Food Security, Plant Pathology in the 21st Century, Springer Science+Business.