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DISEASE PROGRESS CURVES IN THE RICE BLAST PATHOSYSTEM COMPARED WITH THE LOGISTIC AND GOMPERTZ MODELS

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ABSTRACT

Comparison of the linear coefficients from the regression analysis of 307 blast (Pyricularia grisea) disease progress curves of 42 rice genotypes, tested over a period of nine seasons, were made by fitting into the logistic and Gompertz models. The regression parameters *Y*- intercept (a), regression coefficient (b), its standard error (SE_b), coefficient of determination (R^2) and the time required for the disease to reach specific level of severity (T_{50}) were estimated. The estimates of the linear coefficients following Gompertz model, revealed lower values of *b*, SE_b and negative *'a'*; higher values of T_{50} as well as R^2 compared with those by logistic model. In general, the logistic apparent infection rates were higher than the Gompertz rates, the differences being wider for the slow-blasting genotypes compared with the fast-blasting genotypes. The lower Gompertz infection rates accompanied with low negative *'a'* values, resulted in higher T_{50} values, compared with the respective lower estimates through logistic model. Both *b* and T_{50} values estimated by Gompertz model were more consistent among the slow-blasting genotypes over seasons. Out of the 307 disease progress curves, 91.2 % fitted well in to the logistic model and 90.8% in to the Gompertz model, thus suggesting that both the models fit well in to the rice blast pathosystem. The estimates of T_{50} were proportional to the corresponding apparent infection rates in both the models. The benefit from sanitation in terms of the epidemic to reach 0.5 level, was estimated to be 21 and 47 days following logistic and Gompertz models, respectively.

Keywords: rice, blast, disease, Infection, pyricularia grisea, pathosystem, model.

INTRODUCTION

Blast disease of rice (*Oryza sativa* L.) caused by Pyricularia grisea still remains as the most serious disease causing severe yield losses, especially in endemic areas of tropics and sub-tropics (Ou, 1985). Although successful chemical control measures have been evolved, these are too expensive and hence host resistance is given priority in disease control strategy. It is considered as a no-cost technology, especially for the poor and marginal farmers and also an important component of the eco-friendly technique of integrated disease management programme. Genetic analysis has resulted in identification of several major genes governing resistance in the host plant, which function in a race specific manner. Widespread cultivation of such cultivars possessing vertical resistance lead to the development of matching genes for virulence in the pathogen strains with strong ability to overcome the resistance in the host, thus leading to breakdown of resistance. Such instances of breakdown of resistance has induced plant pathologists and breeders to develop genotypes possessing rate reducing resistance, which is believed to be long lasting and more stable (van der Plank, 1963).

Assessment of rate reducing resistance to rice blast disease, otherwise known as slow-blasting resistance, is accomplished by recording sequential observations right from the first day of disease initiation till end of the epidemic and characteristion of the disease progress curves through estimation and comparison of different parameters for evaluation. The proportion of disease increase in a host plant, when plotted against time, gives a sygmoid curve though other types of curves are encountered. It is necessary to linearise such curves in order to evaluate rate reducing resistance for comparison of the epidemics in terms of the rate of disease increase. This could be achieved by applying either the logistic model (van der Plank, 1963) or Gompertz model (Berger, 1981) or Richards model (Richards, 1959) or Weibull model (Weibull, 1951) or Gussian model (Large, 1952). It is not necessary that all the models will fit well to a specific plant-pathosystem nor a specific model will fit in to all the plant-pathosystems or even diseases. Depending upon the nature of the disease progress curves, one model may fit better than the other to a particular disease in any plant-pathosystem. Although the logistic model has ben widely used in analysis of epidemics for evaluation of rate reducing resistance in several plant-pathosystems in the past, serious draw-back in the use of this model as a statistic for studying such resistance has been pointed out by different plant pathologists (Berger1981, Shaner and Finney 1977, Wilcoxson *et al.*, 1975, Luke and Berger, 1982). Kranz (1974) cautioned plant pathologists and breeders not to use a particular transformation model in any pathosystem without verification of the underlying distribution which might lead to erroneous interpretation of the epidemic parameters. The present investigation was therefore aimed at testing the goodness of fit of the logistic and Gompertz transformations to the rice blast pathosystem. The data presented by the senior author in his Ph.D. thesis (Mohapatra, 2002) was further analyzed and interpreted for this purpose.

MATERIALS AND METHODS

Plant material and cultural conditions

The seeds of the rice genotypes were collected from the national germplasm being maintained at the Central Rice Research Institute, Cuttack, India and the International germplasm being maintained at the International Rice Research Institute, Manila, Philippines through the National Bureau of Plant Genetic Resources, New Delhi, India. The origin of these genotypes were different rice growing countries like Bangladesh, Brazil, China, India, Japan, Malaysia, Philippines, Srilanka, USA and West Africa. These genotypes were earlier tested under multi-locational trials all over the world through International Rice Testing Program (IRTP) for various traits, conducted by the International Rice Research Institute, Philippines. The blast disease scores for these genotypes were 1 to 5 in the standard evaluation system (IRRI, 1988).

The seeds were shown in Uniform Blast Nursery (UBN) with slight modification of three rows of each test line flanked by two rows of the highly susceptible check Karuna, used as the spreader rows. The experiment was repeated over a period of nine seasons, spread over five years. Fertilizer was applied @ 100 kg N/ha in the form of Ammonium Sulphate in split doses. High humidity (>90% RH) was maintained through out the period of epidemic development by running sprinklers during hottest period of the day (10 AM to 3.30 PM) with intermittent stoppages of half an hour at every one hour of running. Thus 100 % disease pressure was created in the spreader rows of the susceptible check Karuna during all the seasons of testing which facilitated epidemic development in the test genotypes.

Recording observations and analysis of epidemics

Critical-observations on the per cent host tissue damaged, were recorded at every alternate day interval, beginning from the initiation of the disease till completion of the epidemic. The observation was restricted in the middle row, leaving the two side rows as the border effects. Repeated observations facilitated in monitoring and assessment of the disease progress curves.

The rate of disease increase over time was estimated following the logistic model (van der Plank, 1963) as the regression coefficient of the *logit x* on time in days and by Gompertz model (Berger, 1981) as the regression coefficient of *gompit x* on time in days; *x* being the proportion of the host tissue infected by the pathogen, *logit x* being the log_e [$x / (1-x)$] and *gompit x* being - log_e (- log_e *x*). The best fit of a specific model to the data was determined by comparison of the rate parameters (*'r'* for logistic and *'k'* for Gompertz), which is nothing but the regression coefficient *'b'* ; the respective standard errors (SE_b), *y*-intercept (*a*), the coefficient of determination (R^2) and the time required for the disease to reach specific level ($x = 0.50$) of severity in logistic model (T_{50r}) and Gomprtz model (T_{50k}). The estimates of T_{50} values were calculated following the equations suggested by Shaner and Finney (1977) which is given as:

T_{50} - *logit or gompit* [{ 0.50 / (1-0.50)} - *a*] / *b*,

using the values of the point of intersection (*a*) and the regression coefficient (*b*) determined from the *logit (r)* or *gompit (k)* analysis of the respective disease progress curves.

The regression parameters and the T_{50} values were compared with the area under disease progress curve (*AUDPC*) which was estimated as per the equation suggested by Shaner and Finnay (1977) as:

$$AUDPC = \sum_{i=1}^{n} [(X_{i+1} + X_i) / 2][t_{i+1} - t_i]$$

where, X_i = the proportion of the host tissue damaged at i^{th} day

t_i = the time in days after appearance of the disease at i^{th} day

n = the total number of observations

The estimates of AUDPC was normalized by dividing with the total area of the graph (*i.e.* the number of days from first appearance of the disease till end of the observation period x 1), in order to facilitate a better visual comparison among host genotypes over seasons of testing (Fry, 1968). The normalized *AUDPC* was referred to as the relative area under disease progress curve (*RAUDPC*).

Attempts were made to identify the most important parameter for characterization of the disease progress curve by comparison of variability and inter-correlation among them, extracted through factor analysis (Kendall and Stuart, 1968). Each of the parameter was considered as entities and the genotypes as variables. The analysis extracted eigen vectors and rotated factor matrix showing inter-correlation among the variables. The entities included into the factor-1, explaining highest per cent variability present in the cimmunality, were considered as the top ranking parameters. The relation between r and T50r as well as *k* and T50k was analysed by employing multiple regression analyses.

The benefit from sanitation (Δt) was estimated following the method suggested by van der Plank (1963) as:

$\Delta t = (2.3/r_l) \log_{10} (x/x_s)$

where, Δt is the delay in time (days) taken for the proportion of diseased tissue to increase from x_s to x ,

x is the proportion of initially infected tissue without sanitation,

x_s is the proportion of infected tissue with sanitation,

x/x_s is the sanitation ratio of 5.0 and

r_l is the average infection rate.

RESULTS

The disease progress

The maintenance of conducive atmosphere for disease development during the entire period of epidemic build up by way of application of high levels of nitrogenous fertilizers in split doses and creation of high humidity by providing sprinkler irrigation during hottest periods of the days; created conditions for early disease initiation, rapid disease development and highest disease pressure in the susceptible check Karuna and a few other test genotypes. The delayed disease initiation, slower rates of disease development and lower final disease severities were recorded for the slow-blasting genotypes. The disease severities in the susceptible genotypes were significantly highest than those on the slow-blasting genotypes on all assessment dates. Thus a wider range of variability in blast disease development and spread was recorded among the 42 genotypes tested over a period of seasons spread over five years, which facilitated estimation of different regression parameters and a better comparison among the broad spectrum of disease progress curves.

Comparison between logistic and Gompertz models

The linear coefficients from regression analysis in respect of the disease progress curves for 42 genotypes tested during Rabi 1993 as a representative data set has been presented in Table-1. Similar data sets analysed for 9 consecutive test seasons have been compiled and presented in Table-2. The genotypes expressing zero disease reaction have been eliminated from these compilations. It is evident from the data presented in Table- 1 that the logistic apparent infection rates were 2 to 6 times higher than the Gompertz apparent infection rates or in other words, the regression lines were more steeper in the former than the later model. The *Y*-intercept values in the logistic model were 2 to 4 times lower compared with those in Gompertz model. Both logit-*a* and gompit-*a* showed highly significant correlation with logistic apparent infection rate *r* (-0.567 and -0.898) Gompertz apparent infection rate *k* (-0.451 and -0.859) T_{50r} (0.518 and 0.734) and T_{50k} (0.478 and 0.620), respectively. The Gompertz model provided lower standard errors of the regression coefficient, which is a measure of variability of *Y*-values about the regression line; compaired with those of logistic model. The estimates of coefficient of determinations (R^2) were high for most of the disease progress curves in both the models, with a few exceptions, which did not fit into the model. Based on the estimates of *Y*-intercept (*a*), regression coefficients (*b*) with their standard errors and coefficient of determination (R^2); about 91% of the curves fitted into both the models (Table-2).

The time required for the disease to reach a specific level of severity (x = 0.50) is dependant on both *Y*-intercept (*a*) and the regression coefficient (*b*) of the transformed linearised disease progress curve. The T_{50} estimates were 0.4 to 11 times higher in Gompertz model than those in logistic model. In a few instances however, the T_{50} estimates were too high even to the extent higher than the total duration of the host genotype.

These general observations on different disease progress curves of 42 rice genotypes tested over a period of nine seasons have been well reflected in the figure drawn on average disease progress curves (Figure-1a) as well as the corresponding linearised lines (Figure-1b and c) for four genotype clusters-A to D, recognized through our earlier findings (Mohapatra, 2002). Among the four clusters, A and B were fast-blasting (FB) and C and D were the slow-blasting (SB) genotype clusters. The disease in the FB genotype clusters was initiated early and reached the peak of 100% severity also earlier than those for the SB genotype-clusters, resulting in higher AUDPC as well as RAUDPC values in the former (*AUDPC* = 1153.0, RAUDPC = 51.9 for cluster-A) than the later (AUDPC = 105.2, RAUDPC = 5.8 for cluster-D). The linearised disease progress curves were steeper in the former than the later group of genotypes in both the models. The regression line in logistic model were steeper compared with those in Gompertz model, resulting in higher r values than k values for all the four groups of genotypes. The *logit-a* values were lower than the corresponding *gompit-a* values. The higher *gompit-a* values and lower *k* values resulted in longer T_{50k} estimates compared with T_{50r}. Averaged over the SB and FB genotypes, the T_{50} was delayed by 45 days in Gompertz model over logistic model for the SB genotype cluster-D. There was no difference between the two models, in T_{50} values for FB genotype cluster-A. The delay was 51 and 97 days for SB genotype cluster-D over the FB genotype cluster-A in logistic and Gompertz models, respectively.

Table-1. Linear coefficients from regression analysis of 42 disease progress curves fitted to the logistic and Gompertz models for one representative season of study.

Genotypes		Logistic				Gompertz			
		a	b	SE_b	R^2 (%)	a	b	SE_b	R^2 (%)
15	DZ-192	-4.834	0.076**	0.021	65.69	-1.514	0.014*	0.004	62.54
16	DM-27	-5.965	0.163**	0.021	89.70	-1.842	0.042**	0.005	89.58
21	Tieu-phai	-5.434	0.138**	0.019	88.15	-1.737	0.037**	0.005	89.20
24	E-425	-4.790	0.122**	0.021	89.92	-1.575	0.033**	0.005	83.57
26	Mak-thua	-7.490	0.152	0.054	80.00	-2.097	0.033*	0.012	80.00
27	Sam houang	-4.865	0.118**	0.023	76.17	-1.579	0.032**	0.006	77.96
28	Sakai	-5.897	0.120**	0.020	83.30	-1.839	0.029**	0.004	86.66
29A	Seritus malam-A	-6.230	0.170**	0.019	91.99	-1.897	0.042**	0.005	91.79
29B	Seritus malam-B	-4.764	0.119**	0.021	79.70	-1.570	0.032**	0.005	82.87
30	Jumi-1	-4.484	0.101**	0.028	61.72	-1.482	0.027**	0.007	64.96
32	Laurent-TC	-5.504	0.153**	0.031	74.36	-1.482	0.027**	0.007	64.96
35	Chiang-tsene-tao	-4.834	0.076**	0.021	65.69	-1.514	0.014*	0.004	62.54
36	Chokoto	-5.592	0.133**	0.024	83.15	-1.783	0.034**	0.006	84.21
37	India dular	-7.607	0.593**	0.103	82.52	-3.260	0.302**	0.069	73.30
41	Raj bhawalta	-4.502	0.092**	0.015	80.24	-1.524	0.026**	0.004	84.90
42	Sechi aman	-8.125	0.224**	0.045	86.07	-2.216	0.046*	0.010	82.96
43	Surjamukhi	-4.865	0.118**	0.023	76.17	-1.579	0.032**	0.006	77.96
46	IR-5533-PP-854-1	-7.459	0.228**	0.045	86.48	-2.255	0.060**	0.011	88.42
47	Madhukar	-4.157	0.045*	0.017	42.02	-1.396	0.008*	0.003	40.35
48	Milayeng-51	-5.432	0.095**	0.014	86.79	-1.629	0.018**	0.003	84.53
49	PTB-8	0.000	0.000	0.000	0.00	0.000	0.000	0.000	0.00
54	Dahanala-2014	-5.975	0.112**	0.020	80.81	-1.874	0.029**	0.004	90.20
56A	Lien-tsan-50-A	-4.708	0.070**	0.015	72.19	-1.488	0.012**	0.003	68.50
56B	Lien-tsan-50-B	-4.653	0.107**	0.023	72.24	-1.545	0.030**	0.006	77.08
57	N-22	-4.612	0.152**	0.019	87.54	-1.577	0.049**	0.005	91.59
58	Salum pikit	-6.397	0.141*	0.032	83.05	-1.799	0.026*	0.006	79.82
59	PTB-18	-5.485	0.120*	0.042	57.14	-1.668	0.026*	0.009	57.14
63	DNJ-155	-5.717	0.114**	0.017	87.06	-1.690	0.021**	0.003	85.86
64	DJ-88	-5.406	0.101*	0.036	60.80	-1.623	0.018*	0.007	58.63
66	UCP-188	-5.091	0.082**	0.011	87.27	-1.572	0.015**	0.003	81.85
71	Goda heenati	-4.144	0.086**	0.024	57.35	-1.391	0.023**	0.006	60.55
72	Kalubalawee	-6.342	0.253**	0.018	96.52	-2.229	0.091**	0.009	93.52
75	Bakka-biasa	-6.601	0.246**	0.018	96.05	-2.241	0.083**	0.011	89.17
76	Tiace	-6.885	0.440**	0.071	80.77	-3.016	0.225**	0.049	69.52
77	ARC-7046	-5.463	0.222**	0.019	94.40	-1.927	0.081**	0.006	96.42
80	Prolific	-5.887	0.115**	0.016	87.48	-1.838	0.028**	0.004	90.28
87	Pusa-4-1-11	-6.725	0.304**	0.027	94.68	-2.573	0.127**	0.019	86.64
90	Ratna	-5.689	0.220**	0.014	96.50	-2.008	0.079**	0.008	91.86
91	Jaya	-7.273	0.356**	0.019	98.12	-2.919	0.161**	0.018	91.82
93	CR-289-1045-16	-6.069	0.524**	0.096	80.83	-2.877	0.294**	0.061	76.97
94	CR-570	-4.595	0.112**	0.026	69.95	-1.507	0.030**	0.007	71.78
K	Karuna	-5.849	0.663**	0.119	83.80	-2.558	0.353**	0.079	77.08

* and ** Significant at P = 0.05 and 0.01, respectively.

Table-2. Regression parameters and curve fittings of disease progress curves obtained over nine seasons, compared with logistic and Gompertz models.

	Logistic model					Gompertz model				
Seasons	**Range**		**No. of curves**			**Range**		**No. of curves**		
	a (-ve)	***b***	**Studied**	**Fitted**	**%**	***a*** (-ve)	***b***	**Studied**	**Fitted**	**%**
Rabi, 1991	11.719-1.438	0.839-0.054	28	26	92.86	5.370-1.494	0.498-0.013	28	26	92.86
Kharif,1991	9.139-4.164	0.632-0.076	25	21	84.00	2.584-1.561	0.401-0.016	25	21	84.00
Rabi, 1992	11.713-1.808	0.816-0.028	27	25	92.59	5.499-1.558	0.417-0.012	27	26	96.29
Kharif,1992	9.329-3.361	1.258-0.013	27	22	81.48	4.283-1.198	0.654-0.002	27	20	74.07
Rabi, 1993	8.125-4.144	0.663-0.045	41	40	97.56	3.260-1.391	0.353-0.008	41	41	100.00
Kharif, 1993	8.543-2.289	0.652-0.023	35	30	85.71	2.303-0.797	0.568-0.004	35	29	82.86
Rabi, 1994	7.996-5.349	0.666-0.058	41	40	97.56	5.349-1.357	0.353-0.007	40	39	97.50
Kharif,1994	8.543-0.544	0.981-0.014	41	36	87.80	3.483-0.881	0.517-0.001	41	35	85.36
Rabi, 1995	8.022-4.860	0.800-0.040	42	40	95.24	3.443-1.539	0.416-0.013	41	40	97.56
Total	**11.719-0.544**	**1.258-0.013**	**307**	**280**	**91.21**	**5.499-0.797**	**0.654-0.001**	**305**	**277**	**90.82**

a = Y-intercept; b = regression coefficient

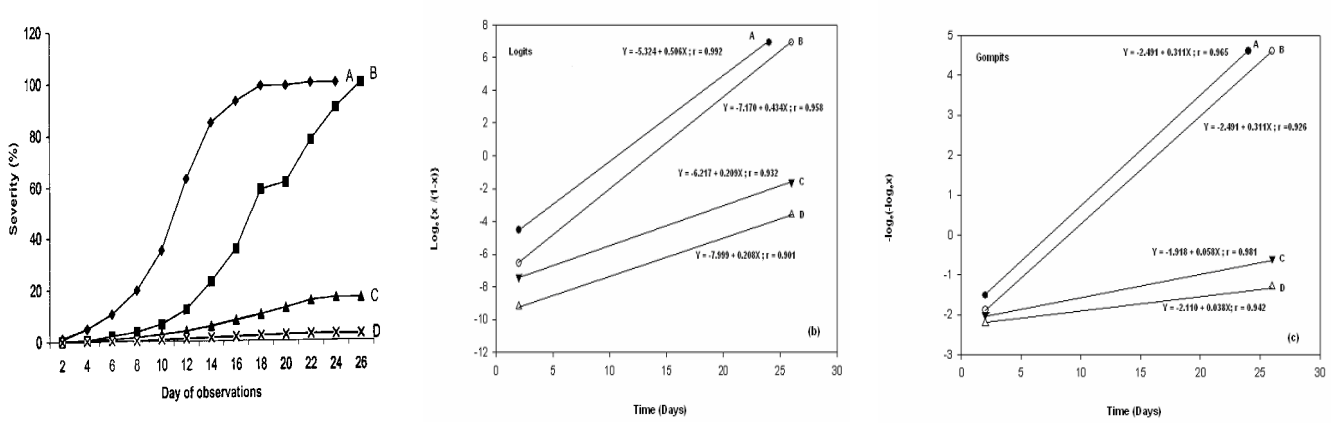

Figure-1. Average disease progress curves for four clusters of rice genotypes tested over nine seasons of study (Figure-1a) the corresponding linearised lines following logistic model (Figure-1b) and Gompertz model (Figure-1c).

Relation between apparent infection rate and the corresponding T_{50} estimates

Attempts were made to find out the relationship between logistic (r) or Gompertz (k) apparent infection rates with their corresponding T_{50} estimates. The 42 rice genotypes, tested over nine seasons, resulted in 378 disease progress curves, out of which 78 cases exhibited either zero reaction in one season or the other, or expressed constant disease score through out the observation period due to which the estimation of r or k was not possible. The relationship exhibited the function of exponential curve, which revealed that the delay in the disease to reach x = 0.5 severity level is inversely proportional to the respective apparent infection rates *r* or *k* (Figures-2a and b). The delay in time required to reach Y=0.5 level in any model can be found by comparing lines across the rate between the range of 0 to 0.98 for logistic and 0 to 0.65 for Gompertz model. The figure clearly demonstrates that if the epidemics were to proceed logistically, the delay in time to reach *Y* = 0.5 level would be shorter than those in Gompertz model.

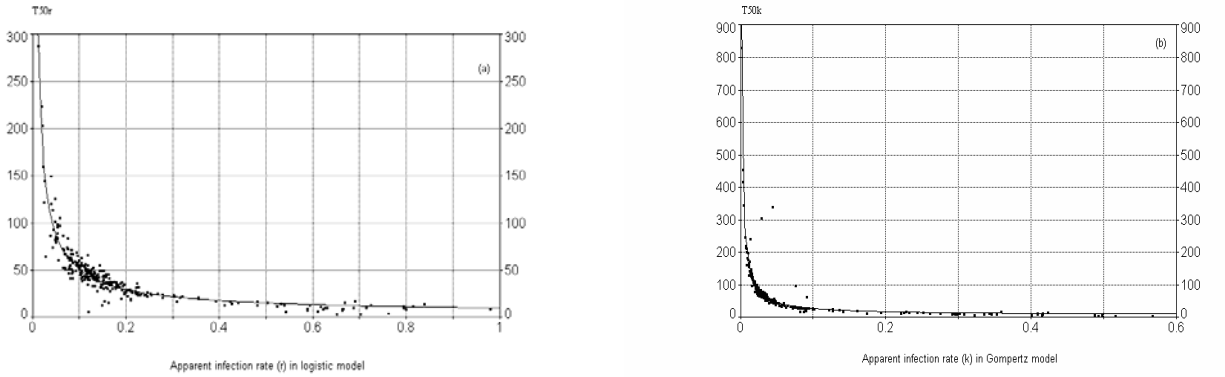

Figure-2. Relation between the logistic apparent infection rate (Figure-2a) and the Gompertz apparent infection rates (Figure-2b), with the corresponding estimates of the time required to reach 50% severity (T_{50}).

Benefit from sanitation

The benefit from sanitation estimated as the delay in reaching $x = 0.5$ level (Δt), with a sanitation ratio of 5 and average apparent infection rate of 0.076 per unit per day in logistic model was 21 days, while the delay with an average Gompertz apparent infection rate of 0.034 was 47 days. The Δt estimates for average apparent infection rate (r_l) of < 0.2, 0.3, 0.4 and 0.5 were 11, 7, 5 and 4 days in logistic and 11, 6, 5 and 4 days in Gompertz model. This suggested the gain in terms of Δt with $r_l > 0.1$ would take drastic sanitation measure and also that sanitation measures are relatively ineffective in delaying fast moving epidemics. The relationship between Δt and different levels of r_l showed the function of exponential curve (Figures-3a and b)**.** The Δt values were inversely proportional to r_l in both the models. Although a 21 days delay in logistic model at sanitation ratio of 5 and $r_l < 0.1$ appears to be satisfactory, a 7 days delay in Gompertz model appears too long a time gain for initiation of the disease. It would take drastic sanitation measures to gain more reduction in initial disease and achieve a corresponding delay in time to reach $x = 0.5$ level.

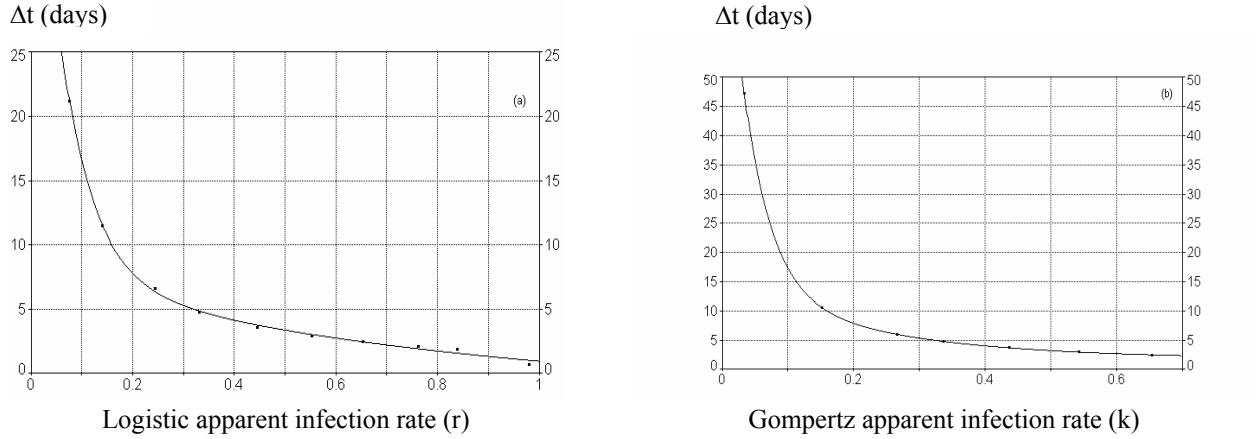

Figure-3. The benefit from sanitation, estimated as the delay in reaching x = 0.5 level, with a sanitation ratio of 5 ($x/x_s = 5$) at different infection rates, following logistic (Figure-3a) and Gompertz (Figure-3b) models.

Relative importance of the parameters

The present set of analyses extracted six parameters viz. *r, k,* T_{50r}, T_{50k}, *logit-a* and *gompit-a*. In order to test their relative importance for characterization of disease progress curves, two additional parameters AUDPC and RAUDPC were also included for comparison. Factor analysis extracted three factors from the rotated correlation matrix explaining 49.5, 26.2 and 24.4% of variability present in the communality (Table-3). The factor matrix printed in bold figures exhibit highest inter-correlation among the variables. The first factor proves high positive inter-correlation for AUDPC, RAUDPC, r and k, indicating that these are the most important parameters for characterization of disease progress curves. The second factor proves high positive inter-correlation for T_{50r} and T_{50k}, which reveals that these are the second ranking parameters. Both logit-*a* and gompit-*a* were recognized as the third ranking parameters.

Table-3. Eigen vectors and factor matrix for eight parameters of slow-blasting resistance in rice.

	Eigen vectors			Factors		
Parameters	**Vector-1**	**Vector-2**	**Vector-3**	**Factor-1**	**Factor-2**	**Factor-3**
AUDPC	0.387	0.355	0.159	**0.961**	-0.195	-0.062
RAUDPC	0.396	0.334	0.156	**0.964**	-0.219	-0.082
r	0.424	0.110	-0.107	**0.830**	-0.268	-0.441
k	0.414	0.244	-0.011	**0.915**	-0.209	-0.287
T_{50r}	-0.304	0.469	-0.404	-0.287	**0.921**	0.141
T_{50k}	-0.276	0.543	-0.391	-0.186	**0.945**	0.161
Logit-a	-0.223	0.407	0.645	-0.051	0.159	**0.961**
Gompit-a	-0.351	0.098	-0.456	-0.516	0.152	**0.774**
Root	5.16	1.30	1.16	3.76	1.99	1.86
% var.expl.	67.76	17.06	15.18	49.45	26.15	24.40

Bold Figures indicate higher inter-correlations.

DISCUSSION

The dynamic process of plant disease increase in time needs critical analyses for comparison of disease progress curves. Although the plotting of the disease increase in a plant population is expected to give a typically sigmoid curve, such assumption becomes inappropriate in any biological system due to the wide interactions among the broad spectrum of host resistance, pathogen virulence and the environmental variations. Hence it is appropriate to linearise the disease progress curves, which helps in determination of the rate parameter for comparison of epidemics, prediction of the future disease intensity and the estimation of the initial disease. This could be achieved by estimating the apparent infection rate by choosing a suitable model from among the cafetaria of several such models available in the literature. Depending upon the nature of the disease progress curves, one model may fit better than the other into a specific plant pathosystem. Although the logistic model (van der Plank, 1963) has been widely used for comparison of the rate of disease progress in several plant pathosystems, of late Gompertz model (Berger, 1981) has been proved more appropriate for analysis of the progress of epidemics. Kranz (1974) cautioned the plant pathologists and breeders not to use a specific model without examining the underlying distribution of the data which might lead to inaccurate estimates of the epidemic parameters.

In the present investigation, comparison between the two models in respect of 300 disease progress curves revealed that the apparent infection rates were 0.3 to 8 times higher and *Y*-intercepts 1.2 to 8 fold lower in the logistic than the Gompertz model for the rice blast pathosystem. Mukherjee *et al.* (1995) similarly, reported lower apparent infection rates following Gompertz model than the logistic model, while comparing disease progress in early rice genotypes. While comparing the two models through analysis of 113 disease progress curves in nine plant-pathosystems, Berger (1981) reported that the Gompertz model provided estimates of Y_0 those were 10 to 100 fold lower than the logistic model and suggested that it may be more appropriate to estimate Y_0 with logistic model by extending a curve to *Y* axis that super-inscribes the initial logit values. According to him the estimation of epidemic rate, prediction of future disease severity and determination of initial disease were more accurate with Gompertz model than with logistic model. The much lower difference in the Y_0 estimate between logistic and Gompertz models in the present investigation indicated a comparatively better fit of both these models into the rice blast pathosystem than the other nine pathosystems reported earlier by Berger (1981). Similar findings were also recorded earlier by Mukherjee (1994) and Mohapatra (2002) in rice blast pathosystem.

The two parameters on *logit* (*logit-a*) as well as gompit line intercept (*gompit-a*) could be of value in determination of the initial inoculum. The lower intercept values for resistant genotypes could be interpreted as an indicator of the amount of initial inoculum, the initial date of start of the epidemic and greater delay in onset of epidemic, which are the characteristics of r-reducing resistance (Berger, 1981). Fried *et al.* (1979) used the X_0 with the logistic model by extending the curve to the *Y* axis that super-inscribe the initial logit values and obtained significant reduction in X_0 due to higher proportion of resistant near-isogenic lines in the mechanical mixture (1:2 and 1:3 S:R) over those with susceptible alone or 1:1 mixture of S:R. In the present investigation, the higher a and lower b values obtained for more resistant genotypes could be interpreted as an indicator of greater delay in onset of epidemic as well as longer days for the epidemic to reach specific level of severity, which are the characteristics of rate reducing resistance. This has resulted in significant correlation of both *logit-a* and

gompit-a with *r, k,* T_{50r} and T_{50k}. Rees *et al.* (1979 a and b) similarly, reported strong correlation between low intercept values and high r for stem and leaf rust resistance in wheat. Shaner and Finney (1977) used the parameter time required for the disease to reach 10% severity (T_{10}) as an alternative to *Y*-intercept because it incorporates within itself both the position and slope of the linearised disease progress curve. Two regression lines having the same slope might differ in position if the epidemic in one begins at a later date than the other. In order to characterize the the position of the transformed disease progress curve, we used the time required for the disease to reach $x = 0.5$ severity (T_{50}), which incorporates both *Y*-intercept and regression coefficient of the transformed disease progress curve. The delay of 51 days in logistic and 97 days in Gompertz model for SB genotypes and a 46 days delay for SB genotypes as against no delay in FB genotypes for T_{50k} over T_{50r} was recorded in the present investigation. Similar delay of 17 days required to reach $x = 0.1$ level of severity for the slow-mildewing cultivar Knox over the susceptible cultivar Vermillion was reported by Shaner and Finney (1977).

We used the parameters logit-a, gompit-a, r, k, T_{50r} and T_{50k}, along with two additional parameters *AUDPC* and *RAUDPC* for comparison of epidemics. Among them, *AUDPC, RAUDPC*, *r,* and *k* were recognized as the top ranking parameters due to their inclusion into factor-1, while T_{50r} and T_{50k} were the second ranking and logit-*a* as well as gompit-*a* were the third ranking parameters. While analyzing the relative importance of 12 parameters for evaluation of slow-blasting resistance in rice, T_{50r} and T_{50k} were observed to be second ranking, while logit-*a* and gompit-*a* were inconsistent in their ranking over different seasons of testing, some times second and some times third ranking. Similar to the present findings, AUDPC, RAUDPC, r and k were consistently recognized as the top ranking parameters over seasons of testing (Mohapatra, 2002) and also in studies on the effect of nitrogen fertilization on the expression of slow-blasting resistance in rice (Mukherjee *et al*., 2005).

The logistic infection rates were 1.7 to 6.2 times higher than the Gompertz infection rates for the rice blast pathosystems as per estimation in the present investigation. While comparing the two models through analysis of 113 disease progress curves in 9 plant pathosystems, Berger (1981) reported that the Gompertz model provided estimates of Y_0 those were 10 to 100 fold lower than the logistic model and suggested that it may be more appropriate to estimate Y_0 with logistic model by extending a curve to *Y* axis that super-inscribes the initial logit values. According to him the estimation of epidemic rate, protection of future disease severity and determination of initial disease were more accurate with Gompertz model than with logistic model.

Depending upon the relationship of r or k with the corresponding T_{50r} and T_{50k}, it could be possible to decide whether to grow or not a specific genotype with known degree of resistance. In the present experiment, the r and k were inversely proportional to the corresponding T_{50} estimates, which means that higher the apparent infection rate, shorter the time it takes to reach specific level of severity and vice-versa. Any genotype with 0.1 to 0.2 logistic apparent infection rate could take 35 to 70 days to reach $x = 0.5$ severity level. Sometimes it so happens that by the time the disease reaches a severity level to cause economic loss, the host plant might have crossed the most vulnerable environmental conditions for disease development and might overcome the damage caused by the pathogen. Such genotypes could still be grown safely.

There is a question of utility of such curves under different situations. The r and k, estimated by two-point method, suggested by van der Plank (1963), is also applicable to find out T_{50} values, since our previous findings have shown a good correspondence between the two methods (correlation coefficient of 0.81 in logistic and 0.91 in Gompertz model). It is also applicable under different situations, since the present disease progress curves encompass a wide spectrum of disease severities ranging from 0 to 0.98 logistic and 0 to 0.65 Gompertz apparent infection rates, which are generally encountered under different environmental conditions. These curves will have wider practical applicability in an integrated disease management program, while taking a decision as to whether to take profilactive measures or not.

Berger (1975) reported considerable variation in the apparent infection rate during the course of epidemic. Logistic transformation has been pointed out to allow misinterpretation of epidemic rates when low disease proportions are encountered (Berger and Luke, 1979; Berger, 1981). They observed significant differences in infection rate caused by large differences in *logit* values (-6.38 and -4.82) for small differences in actual disease at initial reading (0.0017 & 0.008). The logistically transformed values change markedly for slight differences in disease proportions whether those are real differences or errors in disease estimation. Such slight differences in disease proportion at low severities are not amplified when the Gompertz transformation is used. We also observed similar differences as evidenced from those for cluster-C and cluster-D of the linearised disease progress curves (Figures-1b and c).

The best fit of one model over the other has been attained by comparison of the regression parameter *Y*-intercept (Fried *et al.* 1979); R^2, Y_0 & T_{50} (Berger, 1981); R^2 & *RMS* (Waggoner, 1986) and *RMS* (Kranz, 1974). The estimation and comparison of all the parameters in the present investigation resulted in fitting of 91.2% and 90.8%) of the disease progress curves into the logistic and Gempertz models, respectively. Mukherjee *et al.* (2005) reported 78% of the rice blast disease progress curves fitting into both the models. This suggested that both the models fit well into the rice blast pathosystem. The rate reducing resistance to blast disease has been evaluated mostly by estimation of the apparent infection

rate following logistic model (Rodriguez and Galvez, 1975; Villareal *et al*., 1980; Ahn, 1982; Perez Mangas, 1981; Marchetti, 1983; Sah and Bonman, 1992). Mukherjee *et al*. (1995) reported lower apparent infection rates following Gompertz model than the logistic model while comparing the disease progress in early rice genotypes. On the contrary, Berger (1981) reported a better fit of the Gompertz model by analysis of 113 disease progress curves taken from nine plant pathosystems. The Gompertz transformation was reported to be more consistent in detecting the degree of slow-rusting in oats (Luke and Berger 1982), late blight of potato, leaf spot of celeri and rust of bean (Waggoner, 1986) and several other plant pathosystems (Analytis 1973, Berger and Mishoe 1976). On the other hand, a better fit of the logistic model was claimed with wheat powdery mildew patho-system (Fried *et al*., 1979). The best fit of both the models into rice blast pathosystem is further substantiated by the recognition of these two parameters along with AUDPC and RAUDPC as top ranking with high inter-correlations. Similar findings were also recorded by Mohapatra (2002).

Sanitation is the process of reducing the initial inoculum from which the epidemic starts. Rice blast disease control strategy involves some such recommendations of sanitation measures like the use of certified healthy seeds, seed treatment, collection of seed from uninfected healthy plots, summer ploughing and clean cultivation to destroy collateral hosts, destruction of infected plant material and growing resistant cultivars. van der Plank (1963) suggested the method of estimation of benefit from sanitation from several such data points on logistic apparent infection rate and provided detailed estimation of benefit from sanitation for the compound interest diseases potato late blight (Phytophthora infestans) and wheat stem rust (Puccinia graminis). In the present experiment, the relationship between the apparent infection rate and T_{50} is inversely proportional and also the delay in T_{50} estimates beyond 0.1 per unit per day is short. The van der Plank's sanitation ratio of $x/x_s = 5$ at $r_l<0.1$ would result in a likely delay (Δt) of 21 days if the epidemic proceeds in logistic and 47 days in Gompertz model, which means a two times delay in the latter than the former. On the contrary, Berger (1981) reported much longer delay due to logistic model compared with Gompertz model. The sanitation measures are relatively ineffective with further delay beyond apparent infection rates of $r_l>0.1$ at the same sanitation ratio. The delay in initiation of epidemic due to sanitation measures, selection of proper cultivars and adjustment of sowing and planting times would help in overcoming the most vulnerable stage of the host as well as favourable weather conditions to pick up infection leading to epidemic buildup.

The present findings on best fit of both the models to rice-blast pathosystem leaves an option to the rice pathologists as well as breeders to choose any model. These findings have practical implication in screening and breeding for resistance. One can take the decision whether to grow or not any cultivar, by comparing the infection rates with corresponding T_{50} estimates through the standard curve. The delay in initiation of epidemic due to sanitation would help in overcoming the vulnerable stage of the host as well as favourable environmental conditions for epidemic development. These findings can be successfully utilized in integrated blast disease management programme.

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