Review Article

Oryza Vol. 55 No. 3, 2018 (363-382)
DOI 10.5958/2249-5266.2018.00046.2

Assessment of partial resistance to rice blast disease

AK Mukherjee, NK Mohapatra and P Nayak*

ICAR-National Rice Research Institute, Cuttack, Odisha, India

****Corresponding author e-mail: nparsuramata@yahoo.co.in***

Received : 12 August 2018 Accepted : 14 September 2018 Published : 20 September 2018

ABSTRACT

Assessment of resistance in any plant pathosystem involves the measurement, growth and development of the pathogen in the host under the influence of the environment. Partial resistance is generally a quantitative measure of disease progress over time and thus can be assessed quantitatively by means of (i) analysis of components of resistance, (ii) estimation of different parameters for evaluation of resistance, (iii) analysis of disease progress curves and (iv) analysis of stability of resistance. In this review, we discuss the analytical approaches on each of these four aspects, beginning with the components of resistance through the parameters to the multivariate analysis of disease progress curves involving principal component analysis, cluster analysis, factor analysis, pattern analysis and also the parametric and non-parametric stability statistics to arrive at the goal of selection of partial resistance. The merits and demerits of each method of assessment are discussed and the most effective method of assessment of partial resistance in rice blast pathosystem is suggested. The analysis of disease progress curves by multivariate analysis involving principal component analysis, cluster analysis, factor analysis, pattern analysis, AMMI analysis and estimation of stability values are recommended for determination of stable partial resistant genotypes.

Key words: *Rice, blast disease, Pyricularia grisea, partial resistance, multivariate analysis, stable resistance*

INTRODUCTION

Consequent upon the past bitter experiences on several instances of breakdowns of vertical resistance in different plant-pathosystems including the rice blast pathosystem (Kiyosawa, 1981) and the total breakdown of blast resistance in Korea in 1977 typhoon (Crill et al., 1982; Ryu et al. 1987); concerted efforts are being made by plant pathologists and breeders to develop genotypes possessing rate-reducing type of horizontal resistance or polygenic resistance or race non-specific resistance (Vander Plank, 1963), general resistance (Caldwell,1968), field resistance (Ezuka, 1972), dilatory resistance (Browning et al., 1977) and partial resistance (Parlevliet, 1979) for control of blast disease.

Resistance in any plant-pathosystem is defined as the ability of the host to hinder the growth and/or development of the pathogen (Robinson, 1969). There are two types of resistance. A total prevention of multiplication of the pathogen in the host is termed as the complete resistance. On the contrary, partial resistance is a form of incomplete resistance in which the pathogen gets the scope to have reduced rate of multiplication and slow rate of expression of disease symptoms. The day to day progress of the disease from the day of disease initiation till the end of the epidemics, when plotted against the time in days, results in a disease progress curve, which is usually sigmoid, although other types of curves are encountered. Such disease progress curve encompasses within it various elements of the host, the pathogen, and the environment active at different stages during the course of the epidemic development and thus can be considered as a complete expression of the anatomy of the disease. One can dissect out these elements, analyze, compare and classify the disease progress curves.

The assessment of disease resistance in any plant-pathosystem can be made in various ways. One may measure the disease incidence, defined as the

number of plant units or tillers or heads or grains or leaves infected and express as the percentage of the total number of units assessed or the disease severity, defined as the area of plant tissue affected by the disease, and expressed as a percentage of the total area assessed. The measures of disease incidence is applicable for monocyclic simple interest diseases like those for false smut, udbatta, kernel smut of rice, smut of sugarcane etc., while, the measures of disease severity is applicable for polycyclic compound interest diseases like those for blast, brown spot, bacterial blight, tungro virus disease of rice and the major rust disease of wheat.

The assessment of partial resistance in different plant-pathosystems have been accomplished by (i) analysis of different components of resistance (Bonman et al., 1991, Crill et al., 1982), (ii) estimation of different parameters for evaluation of resistance (Crill et al., 1982, Fry 1978, Fried et al. 1979, Jeger and Rollinson 2001) and (iii) analysis of the disease progress curves (Jeger et al., 1983; Kendal and Stuart, 1968). The second method has so far been commonly adopted worldwide by plant pathologists as well as breeders in different plant-pathosystems due to the ease in estimation of the parameters. The disease severity can be assessed either once at the peak of the epidemic or several times during the course of epidemics at certain intervals. The former method of assessment measures the cumulative effect of the host-pathogen interactions operating during the course of an epidemic, while the later measures the path of epidemic progress by way of measuring the area under disease progress curves, the apparent infection rates and the time required for the disease to reach a specific level of severity.

Analysis of components of resistance

In compound interest diseases, the disease severity is the cumulative result of several factors or components like (i) Incubation period (*ICP*) - the number of days from inoculation to the first appearance of the visible disease symptoms, (ii) Latent period (*LP*) - the number of days from inoculation till the beginning of spore production, (iii) Infectious period (*IP*) - the number of days from initial sporulation till the lesion ceased to produce spores, (iv) Infection frequency (*IF*) - the number of penetration points observed per unit leaf area from a given amount of inoculum load, (v) Infection efficiency (*IE*) - the ratio of the number of sporulating lesions developed per unit leaf area after 10 days of inoculation to the number of penetration points per unit leaf area expressed as percentage, (vi) Lesion number (*LN*) - the total number of sporulating lesions per leaf, (vii) Lesion size (*LS*), (viii) Lesion area (*LA*) - mean lesion area of 50 lesions selected randomly, (ix) Necrotic zone area (*NZA*) - the estimated necrotic zone area of the lesion, (x) Chlorotic zone area (*CZA*) - the estimated chlorotic zone area around the *NZA*, (xi) The lesion cover (*LC*) - sum of *NZA* and *CZA*, (xii) Sporulation capacity (*SC*) - the total number of conidia produced by a lesion during the entire infectious period (*SP/L*), (xiii) Spores produced per lesion per day (*SP/L/D*), and (xiv) Spores produced per leaf per day ($SP/L_f/D$) by multiplying *SP/L* with the LN/L_f.

Several such components of resistance have been identified in different plant-pathosystems (Parlevliet, 1979). In rice-blast pathosystem, some such components of resistance like *LN*, *LS* and *SC* (Chou et al., 1979); *DE*, *LS* and *SC* (Villareal et al., 1981); *LP*, *LN* and *SC* (Brondy et al., 1988); *DE*, *LP* and *SC* (Castano et al., 1989); *ICP, LC, LN* and *SC* (Nomura and Ishi, 1989); hypal growth, dwarfing index, *LN* and *SC* (Ryu et al., 1990); relative infection efficiency (RIE), *DE, SC, LS* and *ICP* (Sun et al., 1990); diseased leaf area (*DLA*), area under disease progress curve (*AUDPC*), *LS* and *SC* (Wang and Wang, 1991) have been identified. Mukherjee et al. (2013a) identified *ICP*, *LP, IP, IF, IE, LN, NZA, CZA* and *SP* as important components of slow-blasting resistance. These components are genetic and heritable in nature and maximum correlated response and relative selection efficiency could be expected through selection for *LN*, followed by *LN+NZA* (Mukherjee and Nayak, 1997a). Partial resistance to *M. grisea* was recognized in two tall fescue genotypes by longer *ICP* and *LP*, reduced rate of disease progress and lesion expansion, lower final disease incidence (*FDI*), final foliar blight incidence, final mean lesion length (*FLL*) and *AUDPC* (Tredway et al., 2003). According to them, measurement of *ICP*, *LP, FDI* and *FLL* were the most effective and efficient methods for detecting *M. grisea* resistance in tall fescue.

Mukherjee et al. (2013a) recognized three factors each explaining distinct phases of the pathogen

analysed through principal component analysis and factor analysis (Fig. 1). These are (i) the establishment phase involving IF, (ii) the growth phase involving *ICP, IP, LP, LA, LC, NZA* and *CZA*, and (iii) the growth and reproduction phase involving *IE, LN, SP/L, SP/L/D, SP/L$_f$/D* and finally the area under disease progress curve (*AUDPC*).

These components interact among themselves and their effects are cumulative during the course of an epidemic development. Hence, it becomes very difficult to recognize host genotypes possessing such type of resistance, especially from among a large number of test materials, each with several components of resistance. The application of multivariate analysis has special advantage of simplifying such complexities, since the host genotypes under study could be distinctly separated into groups of specific levels of resistance through clustering and ordination. Similar successful numerical classification of the host genotypes on the basis of their attributes of disease assessments at different dates have been made for slow stem rusting in wheat (Rees et al., 1979 a, b; Thompson and Rees, 1979), slow-mildewing in lettuce (Lebeda and Jendrulek, 1988), and early blight resistance in tomato (Madden and Pennypacker, 1979). Mukherjee et al. (2013a) made an attempt to simplify the complexity of genotype x components interactions and classify rice genotypes on the basis of different components of slow-blasting resistance.

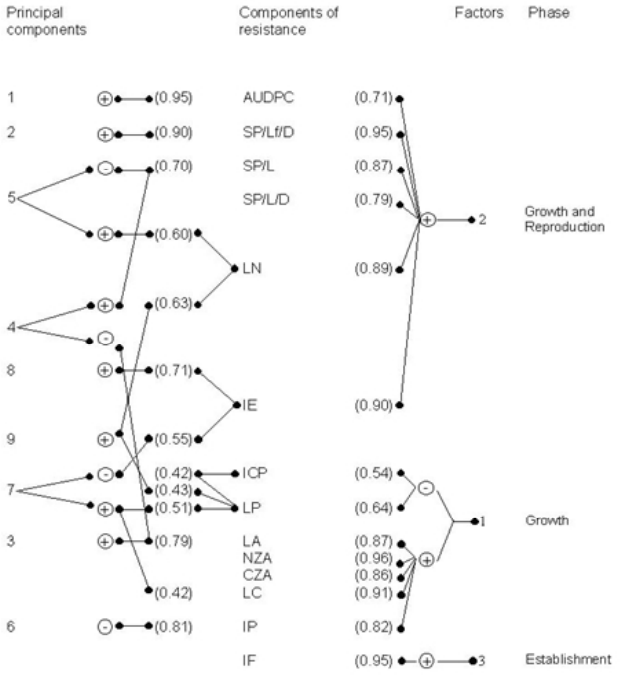

Fig. 1. Diagram depicting the relationship among the components of resistance and the principal components as well as the factors. The signs of the coefficients dominating the latent vectors are shown as positive (+) or negative (-) and the corresponding vectors in brackets.

The different components of resistance contribute independently or jointly towards development of the disease. The contribution of each component towards the final disease severity is difficult to estimate since these components interact among themselves and their effects are cumulative during the course of the disease epidemic. Hence, it is essential to ascertain the genetic and heritable nature of each component, the environmental influence in expression of each component, the association among these components and finally the extent of contribution of each component towards the final disease severity. Mukherjee and Nayak (1997a) reported high genotypic coefficient of variation (GCV) for all components of slow-blasting resistance in rice, except *ICP, LP* and *IF*, thus suggesting that these are least influenced by the environment. In addition to high GCV, high values of heritability along with high genetic advance, genetic gain, correlated response and relative selection efficiency, obtained for all the components, except the previously mentioned three components, suggest that these components are genetic and heritable in nature and are very important in an effective selection programme. Similar results were also reported by Nayak et al. (1987) for bacterial blight resistance in rice.

The strong association among 10 components of slow-blasting resistance in rice both at genotypic and phenotypic levels, higher genotypic correlation than the corresponding phenotypic correlation indicated the modifying effects of environment on the association of components at genotypic level (Mukherjee and Nayak, 1997a). Path coefficient analysis revealed highest direct effect of *LN* followed by *NZA* on the area under disease progress curve both at genotypic and phenotypic levels. Indirect effects of fairly high magnitude were also exerted by other components through *LN* and *NZA* towards *AUDPC*. The *LN* and *NZA* were identified as the most important genetic and heritable components for indirect selection of slow-blasting resistance in rice.

Slow-blasting resistance in rice, characterized by longer *ICP* and *LP*, shorter *IP*, lower *IF, LN/Lf, NZA, CZA*, mean *LA, LC, SC* and finally lower *AUDPC*; was recognized in 13 rice genotypes (Mukherjee et al. 2013a).

Index score

Evaluation of slow-blasting resistance, taking all the component characters into consideration, is difficult since the entire process is labour intensive and time consuming. Indirect selection based on a few important components those have significant contribution towards the final disease, may result in substantial gain over all the component characters. Mukherjee and Nayak (1997a) observed that *LN* and *NZA* are the two most important components of SBR in order of their magnitude of direct contribution towards the total disease, estimated by path coefficient analysis. Adoption of any selection index with a minimum number of component characters and maximum possible selection efficiency in terms of maximum expected genetic advance would be the most efficient and would have wider application value. Construction of selection indices from the estimates of the genotypic and phenotypic variance and covariances of 10 components of slow-blasting resistance in rice, the economic weight of each component and comparison of the expected genetic advance and relative selection efficiency, revealed the superiority of the single-component index 6.42 *LN* and the two-component index 5.61 *LN* + 14.67 *NZA* over rest of the multiple-component indices (Mukherjee et al., 1996). Sequential addition of the components to the index one by one, did not result in any appreciable gain in terms of predicted genetic advance and relative selection efficiency due to the component characters as well as ranking order of the tested 15 rice genotypes from those by direct selection through the parameter *AUDPC*. Hence, they emphasized the importance of the single component-index 6.42 *LN* and the two-component index 5.61 *LN* + 14.67 *NZA* for quick and easy identification of slow-blasting rice genotypes. The detailed methods for estimation of *NZA* (Mukherjee et al., 1997a), and *CZA* (Mukherjee and Nayak, 1997b), *IF* and *IE* (Mukherjee et al., 1997b) for rice blast disease have been developed. The difficulty in estimation of several components of resistance to rice blast disease could be overcome by restricting the critical observations on the components of resistance to the third leaf at seedling stage and fourth leaf at tillering stage (Mohapatra et al., 2001).

Analysis of parameters for evaluation of resistance

The proportion of disease in the host plant, when plotted against time, gives a disease progress curve, which is usually sigmoid; although other types of curves are often encountered. The rate-reducing resistance can be assessed by quantification and comparison of such disease progress curves. Although it is difficult to classify disease resistance into discrete classes, the rate-reducing resistance can be recognized among several genotypes based on a sound knowledge on the host-pathogen system with the help of an efficient evaluation system. The disease resistance in different plant-pathosystems has so far been assessed through estimation of the parameters like the final disease severity (*FDS*), the mean disease severity (*MDS*), the scoring by standard evaluation system (*SES*) (IRRI, 2008), the area under disease progress curves (*AUDPC*) (Shaner and Finney, 1977), the relative area under disease progress curve (*RAUDPC*) (Fry, 1978), the logistic apparent infection rates (r) (Van der Plank, 1963), the Gompertz apparent infection rates (k) (Berger, 1981), the time required for the disease to reach a specific level of severity in logistic (T_{50r}) or Gompertz (T_{50k}) models (Shaner and Finney, 1977), the logit ($logit_a$) or gompit ($gompit_a$) line intercepts, the index-score values (*IS*) (Mukherjee et al., 1996), the infection gradient (g) (Gregory, 1968), the velocity of the disease progress (v) (Minogue and Fry, 1983) or the genotype-scores on first (*PC-1*) and second (*PC-2*) principal components obtained through the principal component analysis (PCA) (Zobel et al., 1988). Each of the parameters has its own advantages and disadvantages as well. It cannot be taken for granted that a single parameter will fit into all the plant-pathosystems and *vice versa*.

Relative importance of parameters

The disease progress curve encompasses within it various elements of the host, the pathogen and the environment active at different stages during the course of the epidemic development and thus can be considered

as a complete expression of the anatomy of the disease. One can dissect out these elements, analyse, compare and classify the disease progress curves. Kranz (1974a) noted that comparative epidemiology is primarily, a quantitative empirical science that includes plotting of the disease progress curve, its transformation and elaboration by statistical, mathematical and computer models. The disease progress curves are of limited use as a method of comparison unless subjected to (i) smoothening the curves (ii) transforming the curves into linearity or curvelinearity and (iii) verifying significant differences among them; which could be achieved by estimation and comparison among different parameters.

The relative importance of 12 parameters in characterisation of rice blast disease progress curves, determined by the degree of variability and inter-correlations among them estimated through factor analysis, revealed maximum inter-correlations among *FDS, MDS, AUDPC, RAUDPC*, *r, k* and *PC -1* consistently for all the nine seasons by way of their inclusion in to factor-1 which accounted for 44.98 to 62.16% of variation during nine seasons of testing with a mean of 54.54%. Besides, the inter-correlation among themselves, each of them was significantly associated with rest of the parameters. Among them the logistic apparent infection rate has been widely used for analysis of epidemics as a very useful parameter in several plant-pathosystems including rice-blast pathosystem. However, serious drawback in the logistic apparent infection rate as a statistic for studying the rate-reducing resistance has been pointed out (Wilcoxson et al., 1975; Shaner and Finney, 1977; Berger, 1981; Luke and Berger, 1982). Berger (1981) reported considerable variation in the logistic apparent infection rate and was of the opinion that some of the information in the disease progress curve are lost in the calculation of *r* due to the errors introduced by lack of linearity, since it is strongly influenced by minor differences in low disease severities early in the season, which becomes much larger when transformed to *logit x/(1-x)*. According to him, the Gompertz model avoids the curvelinearity associated with the logistically transformed values resulting in accurate estimation of the epidemic rate, projection of future disease severity and determination of initial disease in nine plant-pathosystems. The Gompertz transformation was also 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 beans (Waggoner, 1986) and several other plant-pathosystems. On the other hand, a better fit of the logistic model was claimed with wheat powdery mildew-pathosystem (Fried et al., 1979). Analytis (1973) and Berger & Mishoe (1976) obtained a good statistical fit with Gompertz, Bertalanffy and Mitscherlich transformations in several plant-pathosystems. Mohapatra et al. (2008) observed both logistic as well as Gompertz models fitting well into the rice blast-pathosystem.

The two parameter, logit-line intercept ($logit_a$) as well as gompit-line intercept ($gompit_a$) were found to be of some value for comparing the disease progress curves, as an indicator of initial start of the epidemics, but were highly inconsistent in expression of the true nature of the disease progress curve, as evidenced by their poor association with other parameters over nine seasons of testing. The lower '*a*' values obtained for more resistant genotypes could be interpreted as an indicator of initial date of start of epidemic and greater delay in onset of epidemic. On the other hand, the reverse should have happened for the fast-blasting genotypes, which was not always true. This is probably the reason why these parameters were inconsistently included in to either factor-1 or 2 over different seasons of testing. The two parameters namely; T_{50r} and T_{50k} showed maximum inter-correlations and were consistently included in factor-2, accounting for 24.72 to 36.17% of the variation with mean of 31.03% for all the nine seasons of testing and thus were considered as the second ranking parameters, even though they embodied both the position and the slope of the transformed disease progress curves.

The estimates of the parameter *AUDPC* resulted in a better visual comparison among the genotypes, correctly reflected the disease development using all the data available, did not obscure the variation in rate of disease development, exhibited distinct differences among the genotypes, proved most convenient for summation without involving complicated data transformations and was least influenced by minor differences in disease severity early in the season and hence was considered superior to other parameters (Mohapatra et al. 2014). Kranz (1974a,b) analysed different elements of disease progress curves in various pathosystems through factor analysis and reported

AUDPC as one of the important elements in addition to the logistic apparent infection rate. Similar conclusions were also drawn for stem rust resistance in wheat (Wilcoxson et al., 1975), slow-mildewing in Knox wheat (Shaner and Finney, 1977), late blight resistance in potato (Fry, 1978) and slow-blasting resistance in rice (Mukherjee et al., 2005; Mohapatra et al., 2014). The only disadvantage that, it has to be calculated from a common time base, since it is a product of time and severity, could be avoided by estimating the relative area under disease progress curve (*RAUDPC*) for easy comparison between genotypes over different seasons of study.

Principal component and factor analysis

It is of interest to note here that the genotype-score on *PC-1* emerged as one of the first ranking parameters due to the fact that the ranking of the genotypes on *PC-1* was continuous during all the nine seasons of testing and *PC-1* alone accounted for more than 90% of the variation in the communality, with a strong association with all the parameters (Mohapatra et al., 2014). The parameter *PC-2* was consistently recognised as the 3rd factor during all the nine seasons of testing, which accounted for 8.60 to 18.89% of the variation with a mean of 11.43% and was not associated with the parameters under factor-2 *i.e.,* $T_{50\,r}$ and $T_{50\,k}$ and also *gompit-a*. One can choose any of the first ranking parameters from among these cafeteria of 12 parameters giving high preference over *AUDPC* and *RAUDPC* and least preference over *FDS* and *MDS* for preliminary screening of large number of test material and at a later stage relying upon the clustering and ordination of genotypes derived through multivariate analyses, for identification of rate-reducing resistance in rice blast-pathosystem depending upon the available resources for computation of the parameters.

Practical application of the parameters

The rate-reducing resistance to rice blast disease has been evaluated mostly through estimation of apparent infection rate (Rodriguez and Galvez, 1975; Villareal et al., 1980; Perez Mangaz, 1981; Ahn, 1981; Ahn and Ou, 1982), *AUDPC* and *r* (Marchetti, 1983; Marchetti and Zianghua, 1986), terminal disease severity and *r* (Sah and Bonman, 1992), diseased leaf area and *AUDPC* (Marchetti and Zianghua, 1986, Bonman et al. 1989,1991), *AUDPC*, lesion number (*LN*) and lesion size (Wang et al., 1989). Index score values using the single component index, 6.4 *LN*, was found to be highly effective in identification of slow-blasting rice genotypes with 93 % of relative selection efficiency (Mukherjee et al., 1996). Slow-blasting resistance was evaluated by adopting different parameters, among which *AUDPC, RAUDPC, r, k*, T_{50r}, T_{50k}, *FDS, SES score, ISLN, ISLN+NZA* and *PC-1*, of which *AUDPC* and *RAUDPC* were found to be superior expression of resistance (Mohapatra et al., 2014). The effect of nitrogen fertilization on the expression of slow-blasting resistance in rice was evaluated based on nine parameters, among which *LN, AUDPC, RAUDPC, r,* and *k* were found to be superior over T_{50r}, T_{50k}, $logit_a$ and $gompit_a$ (Mukherjee et al., 2005).

Methods for estimation of the parameters

It is worth elaborating the methods of estimation of a few parameters for easy understanding and adoption by the young researchers. The proportion of the host tissue damaged due to the disease in a genotype expressed as the percent disease severity on the last day of observation, when the disease reaches a level of 100% severity in the susceptible check can be considered as the final disease severity (*FDS*). The final disease severity can be divided by the number of days from initiation of the disease symptom till the last day of observation in a particular genotype, to arrive at the mean disease severity (*MDS*) level and is expressed as the per cent disease severity per day. The area under disease progress curve (*AUDPC*) can be estimated following the method suggested by Shaner and Finney (1977) which is given by:

$$\text{AUDPC} = \sum_{i-1}^{n}\left[\left(X_{i+1}+X_i\right)/2\right]\left[t_{i+1}-t_1\right];$$

where,

$\mathbf{x_i}$ = the proportion of host tissue damaged at the i^{th} day;

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

n = the total number of observations.

The values of *AUDPC* can be normalized by dividing with the total area of the graph (*i.e.,* the number of days from the first appearance of the disease until

end of the assessment period), following the method suggested by Fry (1978). The normalized *AUDPC* is referred to as the relative area under disease progress curve (*RAUDPC*). The apparent infection rates in logistic (*r*) model (Van der Plank, 1963) as well as Gompertz (*k*) model (Berger, 1981), can be estimated as the regression coefficients of the *logit* or *gompit x* over time (days), presented as per unit per day; *logit x* being $log_e[x/(1-x)]$ and gompit x being $-log_e[-log_e x]$, the regression coefficient b being the apparent infection rate *r* in logistic and *k* in Gompertz models. The *Y*-intercept '*a*' for both logistic ($logit_a$) and Gompertz ($gompit_a$) models can also be considered as two parameters.

The number of days required for the disease to reach 50% severity can be estimated both in logistic (T_{50r}) and Gompertz (T_{50k}) models (Shaner and Finney, 1977) as :

T_{50} = *logit* or *gompit* [{ 0.50/(1.0 - 0.50) } - a] / b, using the values of the point of interception '*a*' and the regression coefficient '*b*' determined from the logit (*r*) or gompit (*k*) analyses for the respective disease progress curves.

The genotype-scores on the first two principal components (*PC-1* and *PC-2*); estimated from the principal component analysis (PCA) by considering the genotypes as the entities and the disease score at intermittent intervals as the variables; can also be considered as two independent parameters.

The univariate and multivariate stability statistics

Additive main effects and multiplicative interaction (AMMI) analysis

The partial resistance is believed to be long lasting, more durable and most stable. It is necessary to analyze stable resistance of host genotypes or stable pathogenicity of the pathogen strains under varied environmental conditions. Several methods have been proposed to analyze GEI or phenotypic stability (Lin et al., 1986; Becker and Leon 1988; Piepho, 1998;). This method can be divided into two major groups, univariate and multivariate stability statistics (Lin et al. 1986). Joint regression is the most popular among univariate methods because of its simplicity of calculation and application (Becker and Leon 1988), where as Additive Main Effect and Multiplicative Interaction (*AMMI*) is gaining popularity and is currently the main alternative multivariate approach to the joint regression analysis in many breeding programs (Annicchiarico, 1997). Joint regression provides a conceptual model for genotypic stability (Becker and Leon, 1988; Romagosa and Fox, 1993). The GEI from analysis of variance is partitioned into heterogeneity of regression coefficients (b_i) and the sum of deviation (ΣS^2d_i) from regressions. Finlay and Wilkinson (1963) defined a genotype with coefficient of regression equal to zero ($b_i = 0$) as stable while Eberhart and Russell (1966) defined a genotype with $b_i = 1$ to be stable. Mukherjee et al. (1998) considered an ideal stable slow-blasting resistant cultivar as possessing (i) low mean disease scores, (ii) least response to environmental changes ($b_i = 0$), and a minimum deviation from regression ($S^2d_i = 0$). Similar considerations were also offered for identification of stable bacterial blight resistant cultivars in rice by Nayak and Chakrabarty (1986). Nayak et al. (2008b) considered the pathogen strains of *Xanthomonas oryzae* pv. *oryzae* possessing mean pathogenicity levels greater than the population mean, unit regression coefficient ($b_i = 1$) and minimum deviation from regression ($S^2d_i = 0$) as stable virulent pathogen strains and those possessing regression coefficient greater than unit ($b_i > 1$), deviation from regression greater than zero ($S^2d_i > 0$) as most unstable.

Most biometricians consider S^2d_i as stability parameter rather than bi (Eberhart and Russell, 1966; Becker and Leon, 1988). According to the joint regression model, a stable variety is one with a high mean yield, $b_i = 1$ and $S^2d_i = 0$ (Eberhart and Russell, 1966). Wricke (1962) suggested using GEI for each genotype as a stability measure, which he termed as ecovalance (W_i^2). Shukla (1972) developed an unbiased estimate using stability variance (σ_i^2) of genotypes and a method to test the significance of (σ_i^2) for determining stability of a genotype. Francis and Kannenberg (1978), used the environmental variance (S^2x_i) and the coefficient of variation (CV_i) to define stable genotype. Comparison among all these stability parameters through Spearman's coefficient of rank correlation revealed to be significantly correlated with W_i^2, b_i, S^2d_i, σ_i^2; and W_i^2, significantly correlated with b_i, S^2d_i, σ_i^2; b_i was significantly correlated with S^2d_i, σ_i^2 and S^2d_i with σ_i^2. They concluded that among the joint

regression stability measures, S^2d_i was largely used to rank the relative stability of cultivars, but bi could be used to describe the general response to the goodness of environmental conditions, whereas, S^2d_i actually measures the yield stability. However, *AMMI*, S^2d_i, W_i^2, σ_i^2, S^2x_i were generally found to be important in determining the comparative stability. Since *AMMI* combines analysis of variance and principal component analysis in one model (Yau, 1995; Purchase, 1997), it was found useful in describing both the GxE interaction and the stability analysis.

Purchase et al. (2000) compared various statistical procedures for assessing the yield stability of the wide range of wheat genotypes grown under dry land conditions to determine the most suitable method. They performed different statistical analyses like: (i) Shukla's procedure of stability variance (Shukla, 1972); (ii) Lin and Binn's cultivar performance measure (PD) (Lin & Binns, 1988); (iii) Finlay and Wilkinson's regression analysis and coefficient (b_i) (Finlay & Wilkinson, 1963); (iv) Eberhart and Russell's deviation from regression (S^2d_i) (Eberhart & Russell, 1966); (v) Wricke's ecovalence (W_i) (Wricke, 1962); and (vi) the *AMMI* model (Gauch, 1988) to determine yield stability. A critical comparison among all these methods done through Spearman's ranking order correlation coefficient test resulted in the superiority of *AMMI* Stability Value (*ASV*), derived from the *AMMI* model, as the most appropriate.

Farshadfar et al. (2011) studied the relationships, similarities and dissimilarities among five yield-stability statistics in wheat genotypes (*Triticum aestivum* L.). These yield stability statistics are:

AMMI stability value (ASV)

AMMI stability value (*ASV*) as described by Purchase et al. (2000), can be calculated

as follows:

$$ASV = \sqrt{\left[\frac{IPCA1_{sumofsquare}}{IPCA2_{sumofsquare}}\left(IPCA1_{score}\right)\right]^2 + \left(IPCA2_{score}\right)^2}$$

Where,

SS_{IPCA1} / SS_{IPCA2} is the weight given to the IPCA1 value by dividing the IPCA1 sum of squares by the IPCA2 sum of squares. The larger the IPCA score, either negative or positive, the more specifically adapted a genotype is to certain environments. Smaller *ASV* scores indicate a more stable genotype across environments.

Sustainability index (*SI*)

The sustainability index can be estimated by the following formula as described by Babarmanzoor et al. (2009):

$$SI = [(Y-\sigma n)/ YM] \times 100$$

where,

Y= Average performance of a genotype,

σn = Standard deviation and

YM= Best performance of a genotype in any year

Stability index (*I*)

The stability index (I) can be computed following the methods suggested by Rao et al. (2004) which is given by:

$$I = \left(\frac{\bar{y}_{i.}}{\bar{y}_{..}} + \frac{1}{\sigma_i^2}\right) \Big/ \left[\frac{1}{n}\sum_i\left(\frac{1}{\sigma_i^2}\right)\right]$$

where,

$\overline{Y}_i$= Average performance of the i^{th} genotype,

$\bar{Y}$ = the overall mean,

σ_i^2= Sukla's (1972) stability variance of the i^{th} genotype,

n = Number of environments.

The genotypes with highest stability index could be given top ranking and so on.

Yield stability index (*YSI*) and Rank-Sum (*RS*)

The yield stability index (*YSI*) can be calculated by the following formula:

$$YSI = RASV + RY$$

where,

RASV is the rank of *AMMI* stability value, *RY* is the

rank of mean grain yield of genotypes across environments.

Thus, *YSI* incorporates both mean yield and stability in a single criterion. Low values of this parameter shows desirable genotypes with high mean yield and stability.

Rank sum (*RS*) = Rank mean (*R*) + Standard deviation of rank (*SDR*).

RS incorporates both yield and yield stability in a single non-parametric index. Genotypes with the least RS are considered stable with high grain yield.

Standard deviation of rank (*SDR*) can be measured as:

$$S_i^2 = \frac{\sum_{j=1}^{m} (R_{ij} - \overline{R}_{i.})^2}{l - 1}$$

where,

R_{ij} is the rank of X_{ij} within the i^{th} environment,

$\left(\overline{R}_i\right)$ is the mean rank across all environments for the i^{th} genotype and

$$SDR = (S_i^2)^{0.5}$$

The relationships among the yield stability statistics can be derived by conducting principal component analysis (*PCA*) based on the rank correlation matrix using the software *STATISTICA*.

Farshadfar et al. (2011) concluded through principal component analysis of these yield stability statistics that *SI* and *I* are not suitable stability indices for determining stable genotypes with high grain yields. On the other hand, yield stability index (*YSI*), which incorporates *ASV* and mean grain yield in a single non-parametric index, and *RS* (*R* + *SDR*) were the most desirable indices for determining the most stable genotypes with high grain yields. The application of such stability statistics for identification of stable resistant genotypes in any plant pathosystem has not been made so far and hence needs to be explored with necessary modifications for disease resistance.

The most recent development comprises a multiplicative interaction model, which was first introduced in social science (Crossa, 1990), that was later adapted to the agricultural context as *AMMI* (Piepho, 1996). This model was considered appropriate if one is inserted in predicting genotypic yields in specific environments (Annicchiarico, 1997). It combines the analysis for the genotype and environment main effect with several graphically represented interactions for principal component analysis (*IPCAs*) (Crossa, 1990; Abamu and Alluri (1998). Thus, it helps to summarizing the pattern and relationship of genotypes, environment and their interaction (Gauch and Zobel, 1996).

The univariate and multivariate stability statistics (Lin et al., 1986) have been proposed and in wide use to analyse genotype x environment interaction or phenotypic stability. The Joint Regression Analysis (*JRA*) is most popular among the univariate methods because of its simplicity of calculation and application, and it provides a conceptual model for genotype stability. The application of regression model has been made for stable blast resistance in rice (Mukherjee et al. 1998, Gu et al. 2004) and stable bacterial blight resistance in rice (Nayak and Chakrabarty 1986; Nayak et al. 2008b). Mukherjee et al. (2013b) made an attempt to recognize stable blast resistant genotypes through application of the regression models developed by Eberhart and Russell (1966), Perkins and Jinks (1968) and Freeman and Perkins (1971) and the Additive Main effects and Multiplicative Interaction (*AMMI*) models. They concluded that both *JRA* and *AMMI* models were equally potential in partitioning genotype-by-environment interaction in rice blast-pathosystem. However, *AMMI* analysis and GGE biplot display were more informative in differentiating genotype response over environments, describing specific and nonspecific resistance in genotypes, and identifying the most discriminating environments, and thus, could be useful to plant pathologists as well as breeders in supporting breeding programme decisions. In an attempt to analyse and interpret host × pathogen interaction in rice bacterial blight pathosystem, the *AMMI* analysis and biplot display facilitated in a better understanding of the host × pathogen interaction, adaptability of pathogen isolates to specific host genotypes, identification of isolates possessing stable pathogenicity and most discriminating host genotypes, which could be useful in location specific breeding programmes aiming at deployment

of host genotypes in bacterial blight disease control strategies (Nayak et al, 2008ab).

The great potentiality of *AMMI* model was demonstrated for broom rape resistance in faba beans (Flores et al, 1996), identification of stable blast resistance in rice (Abamu et al, 1998), the differential host pathogen interactions between *Rhizoctonia solani* isolates and tulip cultivars (Schneider et al., 1999), between 10 rice yellow mottle virus isolates and 13 differential host genotypes (Onasanya et al., 2004) and late blight resistance in potato (Forbes et al, 2005). Host-pathogen interaction between 52 isolates of *Xanthomonas oryzae* pv. *oryzae* and 16 rice genotypes employing *AMMI* model (Nayak et al., 2008 a,b) and between 8 isolate groups of *Pyrenophora teres* and 13 barley line groups was analyzed with the help of GGE biplot display analysis (Yan and Falk, 2002) to arrive at some valuable conclusions on their relationships. Stable net blotch resistance in barley was identified by application of both *AMMI* and *JRA* models (Robinson and Jalli, 1999).

The multivariate statistical methods are very useful in unravelling patterns in multivariate data from phytopathological studies, especially in epidemiology, ecology, pathology population biology and disease management, and hence these methods should be incorporated into phytopathological research because of their potential for providing a holistic insight into plant disease epidemics (Sanogo and Yang, 2004). These multivariate statistical tools encompass three major tools: (i) Ordination: comprising of principal component analysis, principal coordinate analysis, discriminant analysis, correspondence analysis, multidimensional scaling and factor analysis; (ii) Discrimination: comprising of discriminant analysis, multiple logistic regression, multivariate analysis of variance and cluster analysis; and (iii) Canonical: comprising of canonical correlation, canonical correspondence and redundancy. Ordination aims at describing data by identifying a reduced data dimension of a few variables that account for the greatest amount of variability in the data. Discrimination aims at delineating experimental groups based on a set of variables. Canonical aims at describing and predicting the relationship between two sets of variables.

The *AMMI* model can be applied, with additive effects for isolates (I) and host genotypes (G), and multiplicative term for I×G interactions (*IGI*). The *AMMI* analysis, first fits additive effects for isolates and host genotypes by the usual additive analysis of variance (ANOVA) procedure, and then fits multiplicative effects for IGI by principal component analysis (*PCA*). The *AMMI* model is

$$Y_{ij} = \mu + g_i + e_j + \Sigma\ \lambda_\kappa \pounds_{ik}\ Y_{jk} + R_{ij}$$

where,

Y_{ij} is the lesion length of the i^{th} isolate in the j^{th} host genotype,

g_i is the mean of the i^{th} isolate minus the grand mean,

λ_κ is the square root of the eigen value of the PCA axis k

$\pounds_{ik}$ and Y_{jk} are the principal component scores for PCA axis k of the i^{th} isolate, and the j^{th} host genotype, respectively

R_{ij} is the residual.

The host genotypic and isolate PCA scores are expressed as unit vector times the square root of λ_κ *i.e.,*

host genotypic PCA score= $\lambda_\kappa^{0.5}\ Y_{ik}$;

isolate PCA score=$\lambda_\kappa^{0.5}\ \pounds_{ik}$ (Zobel et al., 1988).

The *AMMI* stability index 'D_i', which is the distance of interaction principal component (*IPC*) point with origin in space, was estimated according to the formula suggested by Zhang et al. (1998) as:

$$D_i = \Sigma\ Y^2_{is}$$

where,

c is the number of significant IPCs,

Y^2_{is} is the scores of the Isolates i in IPCs.

The *AMMI* analysis can be conducted using the computer software *IRRISTAT* for windows, version 5. To assess fitting *AMMI* model, predictive and post-dictive approaches offered by Zobel et al. (1988) can be applied to the data.

AMMI biplot display

The graphical representation of *AMMI-1* biplot reveals the main effect means on the abscissa and the *IPCA-1* scores of the host genotypes as well as the environments simultaneously on the ordinate. The interaction is

described in terms of differential sensitivities of the genotypes or pathogen strains to the most discriminating environmental variable that can be constructed. Displacement along the abscissa reflects differences in main effects, whereas displacement along the ordinate illustrates differences in interaction effects. Host genotypes or environments or pathogen strains appearing almost on a perpendicular line have similar means and those falling almost on horizontal line have similar interaction patterns. Genotypes with *IPCA-1* scores close to zero have small interactions and hence show wider adaptation to the tested environments. A large host genotypic *IPCA-1* score have high interactions and reflects more specific adaptation to the environments with *IPCA-1* values of the same sign (either positive or negative). The scores and main effects can be read from the graph and used to predict the expected level of resistance for any host genotype and environment combination (Schneider et al.1999, Nayak et al. 2008b, Mukherjee et al. 2013b).

AMMI-2 biplot (Fig. 3) is a graphical representation in which genotypes and environments or pathogen strains and host genotypes are displayed simultaneously. The interaction is described in terms of differential sensitivities of the genotypes to the most discriminating environmental variables (*AMMI-axes*). For simple interpretation of the biplot the genotypes with vector end points far from the origin contribute relatively more to the interaction than those with vector end points close to the origin. The biplot displays both host genotypes and environments or pathogen strains and host genotypes in four sectors depending upon the positive or negative signs of the scores on first two principal components. Sector-1 represents host genotypes pathogen strains or environments with positive *IPCA-1* as well as *IPCA-2* scores, while sector-2 represents positive *IPCA-1* and negative *IPCA-2* scores. Sector-3 represents negative IPCA-1 as well as *IPCA-2* scores and sector-4 represents negative *IPCA-1* and positive *IPCA-2* scores.

A polygon drawn in the biplot by joining the host genotypes or pathogen strains located farthest from the biplot origin encompassing all the host genotypes or pathogen strains, facilitates identification of the genotypes that are most resistant in specific environments or the pathogen strains that are most virulent to specific host genotypes. The vertex genotypes in a sector are most or least resistant to the environment falling in that sector or the vertex pathogen strains in a sector are most or least virulent to the host genotypes falling in that sector. The discriminating ability of the environments or the host genotypes can be judged by calculating the distance of each environment or host genotype from the biplot origin. The biplot not only displays the interaction patterns of the host genotypes or the pathogen strains under study but also facilitates the visual description of the environments in a 'which win where' pattern as described by Li et al. (2006).

Interaction pattern from response plot

Response plots indicate the nature of GEI with the main effects of genotypes and environments removed. The values plotted for 'each genotype group by environments' / 'groups of pathogen strains by tested host genotypes' are the deviations from additive main effects predictions of each variable. The larger the deviation, the greater is the interaction of the HG with the environment. The response may be positive or negative depending upon whether or not the HG resulted in more or less effects than the main effects expectation. The host genotypes with reasonably stable resistance across environments or pathogen strains with reasonably stable pathogenicity across the tested host genotypes can be recognised from the response plot. Following this procedure, Nayak et al. (2008b) recognised 27 pathogen strains of *Xanthomonas oryzae* pv. *oryzae* possessing stable pathogenicity for low virulence and five pathogen strains possessing stable pathogenicity for high virulence against 16 host genotypes. The identification and use of virulent pathogen strains possessing stable pathogenicity would help in screening for resistant host genotypes and testing of segregating generations in location specific breeding programme for development and deployment of resistant host cultivars in bacterial blight disease control strategy. Mukherjee et al. (2013b) recognised 28 host genotypes with mean disease scores much less than the grand mean, small negative *IPCA 1* scores and smallest interactions, as possessing most stable non-race-specific resistance to blast disease of rice.

Analysis of disease progress curves

Following the theory of Van der Plank (1963, 1968), it

was expected that infection rate (*r*) would be a valuable measure for comparison of disease epidemics in a range of crop genotypes. However, infection rate was the least useful of the various measures examined as reported for different plant pathosystems (Eskes, 1983; Lebeda and Jendrulek, 1988). This may have been a result of the uniform application of the logit transformation and the inclusion of zeros and the small values in the analysis. Since the small values were actual measures of the disease levels at the time of disease assessment, any requirement for their deletion or modification is undesirable. The logit line intercept (*a*) was found to be of value for comparing epidemic in different cultivars. Lower '*a*' values obtained for the more resistant cultivars could be interpreted as an indication of a greater delay in the onset of epidemic in these cultivars. Such delay in epidemic onset was the result of the vertical resistance present in the host genotype (Van der Plank (1963, 1968). According to him this vertical resistance necessitated a preferential increase in the virulence component in the pathogen population before the epidemic developed, and hence the delay. However, the pathogen population (*Puccinia graminis tritici*) in their experiments on the slow-rusting and tolerance to rust in wheat did not change in virulence, and the delay cannot be explained in these terms, even though vertical resistance was undoubtedly with the low intercept values in a number of cases (Rees et al., 1979ab). Thus the application of regression analyses to the data indicated that the separation of resistance into different types (vertical and horizontal) by the slope and nature of disease progress curves as suggested by Van der Plank (1963, 1968) is not as distinct as postulated.

Pattern analysis of disease progress curves

The average assessment over all the dates and the area below the disease progress curve, permit reasonable comparison of the epidemics among the host genotypes and are simple to apply. However, these measures place undue emphasis on high disease levels late in the epidemic. It has been demonstrated that the use of parameters may not necessarily describe the differences in true field resistance with sufficient accuracy (Eskes, 1983). Kranz (1974a) was the first to suggest the possibility of using cluster analysis in comparative epidemiology. Kranz (1974b) referring to classification as achieved by pattern analysis, stated that classification problem may one day attain great prominence in comparative epidemiology. Since epidemiology deals with populations, it can be useful to delimit more objectively groupings of individuals, strains, races, varieties, treatments or reactions which are more similar in epidemiological behaviour amongst themselves than compares with other groupings. The use of pattern analysis in epidemiological evaluation of wheat rust was demonstrated by Thompson and Rees (1979) and also Rees et al. (1979ab). The wide application of the methods of multivariate analysis not only in the study of field resistance in plants but also to plant pathology in general was predicted by Lebeda and Jendrulek (1987 a,b).

The principal criteria for evaluating field resistance are the dynamics of degree of disease spread or in other words, the onset of epidemic and its progress. The curves describing the growth of disease proportion can be clustered together into groups according to similarities in their courses. The comparison of epidemics or disease progress curves has so far been frequently used for testing the host-pathogen interaction. Kranz (1974a,b) suggested the plotting of disease progress curves, transformation of disease progress curves and its elaboration by statistical, mathematical and computer methods. It has however, been expressed by several researchers that the interpretation of epidemiological data only on the basis of the '*r*' or '*k*' may be unsuitable. Both logistic and Gompertz transformations are to a large extent dependent on the approximation of values for which the given transformation function is not defined. When the number of values measured is small, this fact may significantly affect the final calculated values of '*r*' and '*k*' parameters. Processing of epidemiological data by the methods of multivariate analysis involving cluster analysis and principal component analysis and their comparison with other parameters can yield a substantially greater amount of information (Lebeda and Jendrulek, (1988). This fact was partly noted by Thompson and Rees (1979), who suggested and demonstrated the use of pattern analysis in epidemiological evaluation of rust resistance in wheat. It was believed that the application of multivariate analysis not only to field resistance but also to phytopathology in general may lead to important new

discoveries (Lebeda and Jendrulek, 1987a,b, 1988). Pattern analysis, by extracting and displaying the main patterns and trends in multivariate data, often enables one to obtain new perspectives of the problem under consideration. Although it does not supplant other methods for analysing epidemiological data, it does provide a valuable complement to other methods. Pattern analysis as commonly employed consists of the joint numerical classification and ordination of a set of entities on the basis of their attributes (Williams, 1976). The numerical classification produces discrete groups of like entities such that similarities within the groups are greater that between groups. For analysis of any one data set, the tested genotypes can be regarded as entities possessing a number of attribute which are the disease assessment scores at various assessment dates. Ordination does not of itself separate groups of entities but simply displays the relative geometric position of the entities within a multidimensional space defined by the attributes.

The authors and their associates attempted to explore the (i) analysis of HxP interaction in blast and bacterial blight diseases of rice (Nayak et al. 2008a,b, 2009) (ii) analysis of components of resistance (Mukherjee et al. 2013a) (iii) comparison of different parameters for evaluation of resistance (Mukherjee et al. 2005, 2010, Mohapatra et al. 2008, 2014) (iv) identification of stable resistant genotype through application of multivariate analysis involving cluster analysis, principal component analysis and factor analysis (Mukherjee et al. 2013a,b, Mohapatra et al. 2013), and (v) the genetic diversity of virulence among the strains of *Xanthomonas oryzae* pv. *oryzae* (Nayak et al. 2008 a,b,c) .

Mukherjee et al. (2013a) made an attempt to identify the slow-blasting rice genotypes through application of multivariate analysis of components of resistance and reported that (i) the factor analysis recognized three factors, each explaining distinct phases of the pathogen like establishment, growth and reproduction phases (Fig. 1), (ii) the cluster analysis recognized groups of genotypes possessing distinct slow-blasting and fast-blasting characteristics and (iii) Super-imposition of the clustering pattern onto the ordination figure of the genotype-scores on the planes of *PC-1* and *PC-2*, clearly displayed the geometrical positioning of the slow-blasting and fast-blasting genotype-clusters.

Genotype x environment interaction (*GEI*) of 42 rice genotypes tested over nine seasons was analyzed to identify stable resistance to blast disease incited by *Magnaporthe oryzae* (Mukherjee et al. 2013b). The *GEI* was analyzed following the regression models as well as additive main effects and multiplicative interaction (*AMMI*) model. Although, both regression and *AMMI* models were equally potential in partitioning of *GEI*, *AMMI* analysis and the biplot display were more informative in differentiating genotype response over environments, describing specific and non-specific resistance of genotypes, identifying most discriminating environments and thus could be useful to plant pathologists as well as breeders in supporting breeding program decisions.

The basic epidemiological data on per cent blast disease severity scores, recorded at every alternate day intervals were subjected to multivariate analysis (Mohapatra et al., 2013). Cluster analysis classified the rice genotypes into clusters of slow-blasting and fast-blasting groups (Fig. 2). Super-imposition of clustering pattern onto the planes of the ordination figures on the first two principal components (*PC-1* and *PC-2*) clearly revealed the geometrical positioning of the slow-blasting

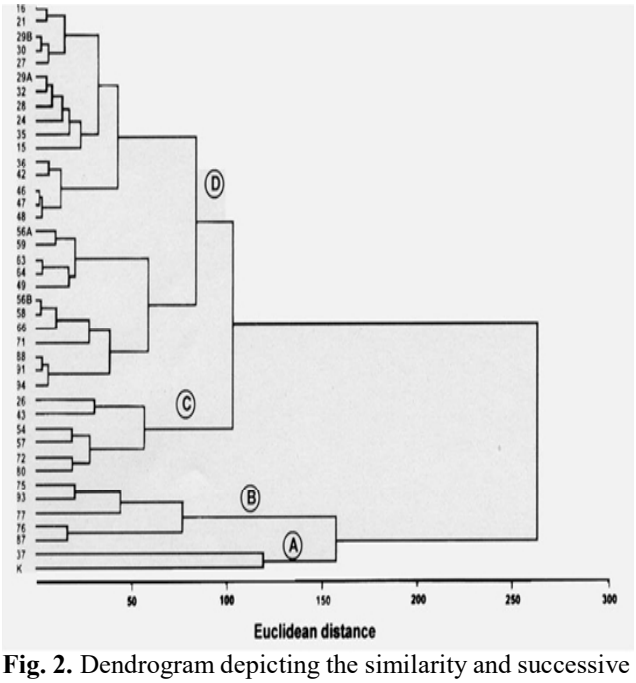

Fig. 2. Dendrogram depicting the similarity and successive clustering of rice genotypes based on the blast disease severity scores at different assessment dates.

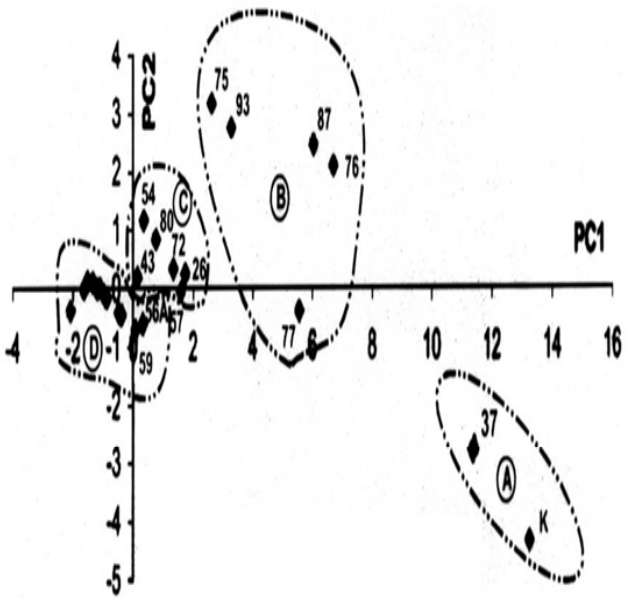

Fig. 3. Ordination of genotype scores obtained from the principal component analysis onto the planes of the first two principal components (*PC-1* & *PC-2*) and superimposition of the clustering pattern obtained from the figure 2. The numerals against each point are the genotypes and the alphabets are the genotype clusters depicted in figure 2.

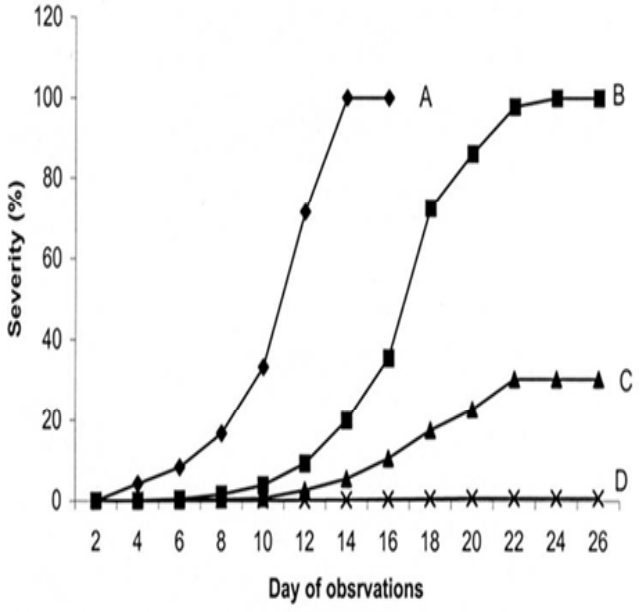

Fig. 4. Average blast disease progress curves for each of the four major groups of rice genotypes (A to D) obtained from the hierarchical classification depicted in the dendrogram (Fig. 2).

genotype-clusters nearer to the intersection between the two ordinates and the fast-blasting genotype-clusters away from it along *PC-1* axis (Fig. 3). Thirty two stable slow-blasting genotypes were recognized by compilation of these data over a period of nine seasons. The average blast disease progress curves for each group of genotypes, obtained from the cluster analysis and displayed in the ordination figure, clearly displays the epidemic progress in these groups of genotypes (Fig. 4).

Thus, *AMMI* analysis could provide (i) a better understanding of the host x pathogen interactions through analysis of variance, (ii) identification of pathogen strains possessing stable pathogenicity or host genotypes possessing stable resistance, and (iii) specificity in pathogenicity pattern and adaptability of the groups of pathogen strains to specific host genotypes groups or host genotype groups to specific environments in a '*which win where*' pattern similar to those reported in rice-bacterial blight pathosystem by Nayak et al. (2008b).

Presentation of data sets

The huge data sets collected by the biological scientists in general and the plant pathologists in particular need to be analysed and presented properly in any scientific forum or even in publications in scientific journals. The usual practice is to present these data sets in frequency distribution curves or in the form of histogram of different frequency classes. The most informative means of displaying a range of numerical data by constructing box and whisker plots was invented by Tukey (1977). A box plot can provide information about the median, the upper and lower quartiles, the highest and lowest values of the data set of which the first two term is the central tendency for the data set and the second and third terms are the measures of central tendency for different parts. In statistical analysis, a box plot is a graph that can be a valuable source of easy-to-interpret information about the sample under study. It can provide information on the sample's range, median, normality of the distribution and skew of the distribution. It can also identify and plot extreme cases within the sample.

It has been in use for medical, ecological, educational and many other branches of science, but

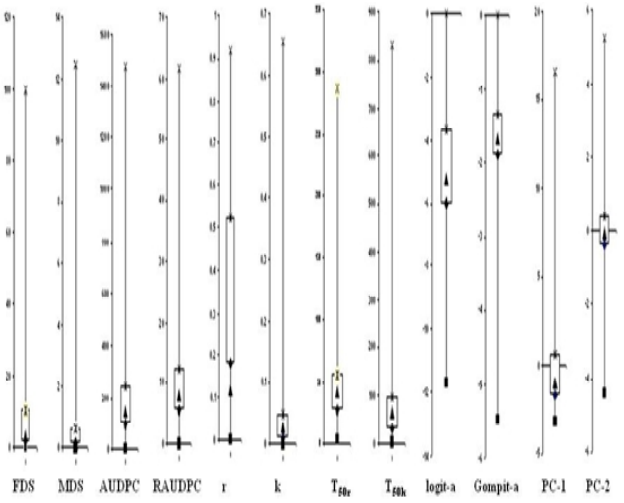

Fig. 5. Box plots of 12 parameters for evaluation of 42 rice genotypes across different seasons of testing for partial resistance to rice blast disease.

its use in plant pathological data presentation is very much limited. Quantification of the effectiveness of recommended fungicides protocol established in 2002 on ray blight disease intensity in Pyrethrum (*Tanacetum cinerariaefoliun* L.) caused by *Phoma legulicula*, to determine the minimum disease intensity thresholds beyond which yield is negatively impacted, was presented in box plots by Pethybridge et al. (2007). The distribution properties of six parameters estimated over nine seasons of testing against rice blast disease caused by *Pyricularia grisea* were presented in box plots depicting the measures of dispersion in the data sets (Mukherjee et al., 2010). The distributions of response ratios for antibiotics, biological controls, and systemic acquired resistance-induced products evaluated for the control of fire blight of apple were also presented in box-whisker plots which showed strong evidence to suggest that among the three products categories, antibiotics were the most effective for fire blight control (Ngugi et al., 2011). These results show strong evidence to suggest that among the three products categories, antibiotics were the most effective for fire blight control. The distribution properties for 12 parameters estimated for evaluation of rice blast disease severity on 42 rice genotypes tested across 9 seasons of study are presented in box plots (Fig. 5) depicting the degree of dispersion in the population estimated through 12 parameters for evaluation of partial resistance. Shifting of the box position for all the parameters towards the lower end signified that the distributions are positively skewed. This was further substantiated by the noticeable shift of the respective medians towards the lower end of the boxes (Mukherjee and Nayak, unpublished). Similar results were also reported by Mukherjee et al. (2010) depicting the measures of dispersion in the data for estimation of area under disease progress curves in rice-blast pathosystem from two data points. The degree of dispersion in rice blast disease severity on 42 rice genotypes tested across nine seasons of study were also presented in box plots by Mukherjee et al. (2013b). Thus the box and whisker plot have been proved to be an informative way to display a wide range of numerical data and the attention of the plant pathologists in general and the rice plant pathologists in particular is drawn to make use of such a valuable tool in presentation of their data sets.

The box plots can be drawn following the methods provided by Noville Hunt on the subject, "Box plots in Excel 2007", either Excel 5/95, or Excel 97/ 2000/2003 or in Excel 2007, available in the Network.

CONCLUSION

Partial resistance is a quantitative analysis of different factors of disease development operating during the progress of epidemic. Scanning of literature revealed that extensive research has been done on analysis of components of resistance, different parameters for evaluation of resistance and analysis of disease progress curves in different plant pathosystems. In this review, we discuss analytical approaches that have been reported so far. We discuss the merits and demerits of each method of assessment. Analysis of components of resistance, although is most accurate and convincing; is time consuming, cost effective, labour intensive and needs special facilities. It has been demonstrated that the use of parameters may not necessarily describe the differences in true field resistance with sufficient accuracy (Eskes, 1983). The available research results discussed herein, lead us to conclude that application of multivariate analysis involving principal component analysis, cluster analysis, factor analysis, the *AMMI* analysis of the daily disease incidence data over the entire period of disease development, the plotting of the ordination figure and geometrical positioning of the genotypes onto the planes of *PC-1* and *PC-2*, will lead

to the success in identification of partial resistant genotypes. The recent review on the application of several statistical tools for data analysis and interpretation in rice plant pathology (Nayak et al., 2018) and the present review on the assessment of partial resistance to rice blast disease in particular, opened up a new direction for the future generations of rice pathologists in achieving the goal of identification of resistant genotypes for adaptation in disease control strategy.

REFERENCES

Abamu FJ and Alluri K (1998). *AMMI* analysis of rain fed lowland rice (*Oryza sativa*) trials in Nigeria. Plant Breeding 117: 395-397

Abamu FJ, Akinsola EA and Alluri K (1998). Applying the AMMI models to understand genotype-by-environment (GE) interactions in rice reaction to blast disease in Africa. Int. J. Pest Manage. 44: 239-245

Ahn SW (1981). The slow-blasting resistance. Paper presented at the meeting on Blast and Upland Rice. EMBRPA, CNPAF, Goiania, Brazil, March 8-14: 1981

Ahn SW and Ou SH (1982). Quantitative resistance of rice blast disease. Phytopathology 72:279-282

Analytis S 1973. Zur Methodik der Analyse von Epidemien Dargestellt am Apfelschorf (*Venturia inaequalis* (Cooke) Aderh.). Acta Phytomed 1:1-76

Annicchiarico P (1997). Joint regression vs. *AMMI* analysis of genotypes environment interactions for cereals in Italy. Euphytica 94: 53-62

Babarmanzoor A, Tariq MS, Ghulam A and Muhammad A (2009). Genotype × environment interaction for seed yield in Kabuli Chickpea (*Cicer arietinum* L.) genotypes developed through mutation breeding. Pak. J. Bot. 41(4): 1883-1890

Becker HC and Leon J (1988). Stability analysis in plant breeding. Plant Breeding 101: 1- 23

Berger RD (1981). Comparison of the Gompertz and logistic equations to describe plant disease progress. Phytopathology 71: 716-719

Berger RD and Mishoe JW (1976). GSMP simulation of several growth functions to describe epidemic progress. Proc. Am. Phytopathol Soc. 3: 217

Bonman JM, Bandong JM, Lee YH, Lee EJ and Valent B (1989). Race-specific partial-resistance to blast in temperate japonica rice cultivars. Plant Disease 73: 496-99

Bonman JM, Estrada BR, Kim CK, Ra DS and Lee EJ (1991). Assessment of blast disease and yield loss in susceptible and partially resistant rice cultivars in two irrigated lowland environments. Plant Disease. 75: 462-466

Brondy U, Zapata JC and Nelson RR (1988). Evaluation of rate-limiting resistance of blast disease in rice cultivars using sub-lethal concentration of a systemic fungicide. J.Phytopathol. 122: 174-180

Browning JA, Simons MD and Torres E (1977). Managing host genes, Epidemiology and genetic concepts, Pages, 191-212 *In*, Plant Disease, An Advanced Treatise Vol-I J.G.Horsfall and E.B.Cowlings eds. Academic Press, New York

Caldwell RM (1968). Breeding for general and/or specific plant disease resistance. Pages 263-272, *In*, Proc. 3rd Intl. Wheat Genetic Symp. 1968

Castano J, Mackenzie DR and Nelson RR (1989). Component analysis of race non-specific resistance to blast disease of rice caused by *Pyricularia oryzae*. J. Phytopathol. 127: 89-99

Chou LK, Nuque FL and Crill JP (1979). Varietal resistance and possible components of horizontal resistance to blast. Int. Rice Res. Newsl. 4: 9-10

Crill JP, Ham YS and Beachell HM (1982). The rice blast disease in Korea and its control with race prediction and gene rotation. *In*, Evaluation of gene rotation concept for rice blast control, IRRI, Manila, Philippines, pp. 123-130

Crossa J (1990). Statistical analysis of multilocation trials. Adv. Agro. 44: 55-86

Eberhart SA and Russell WA (1966). Stability parameters for comparing varieties. Crop Sci. 6(1): 36-40

Eskes AB (1983). Expression of incomplete resistance to pathogens. In, Lamberti, F., Waller, J.H., Van der Graaff, N.A. (Eds.), Durable resistance in crops, 169-195, Plenum Press, New York and London

Ezuka A (1972). Field resistance of rice varieties to rice blast. Rev. Plant Prot. Res. 5: 1-21

Farshadfar E, Mahmodi N and Yaghotipoor A (2011). *AMMI* stability value and simultaneous estimation of yield and yield stability in bread wheat (*Triticum aestivum* L.). Austr. J. Crop Sci. 5(13): 1837-1844

Finlay KW and Wilkinson GN (1963). The analysis of adaptation in a plant-breeding Programme. Aust.

J. Agric. Res. 14: 742-754

Flores F, Moreno MT, Martinez A and Cubero JI (1996). Genotype environment interaction in faba bean, comparison of *AMMI* and principal coordinate models. Field Crops Res. 47: 117-127

Forbes GA, Chacon MG, Kirk HG, Huarte MA and Van Damme M (2005). Stability of resistance to *Phytophthora infestans* in potato, An international evaluation. Plant Pathol. 54: 364-372

Francis TR and Kannenberg LW (1978). Yield stability studies in short-season maize.1. A descriptive method for grouping genotypes. Can. J. Plant Sci. 58:1029-1034

Freeman GH and Perkins JM (1971). Environmental and genotype environmental components of variability. VIII. Relations between genotypes grown in different environments and measure of these environments. Heredity 26: 15-23

Fried PM, Mackenzie DR and Nelson RR (1979). Disease progress curves of *Erysiphe graminis* f.sp. *triticii* on chancellor wheat and four multilines. Phytopath. Z. 95: 151-166

Fry WE (1978). Quantification of general resistance of potato cultivars and fungicide effects for integrated control of potato late blight. Phytopathology 68: 1650-1655

Gauch HG (1988). Model selection and validation for yield trials with interaction. Biometrics 44: 705-715

Gauch HG and Zobel RW (1996). *AMMI* analysis of yield trials. *In*, M.S. Kang, and H.G. Zobel Jr (Eds), pp. 85-120. Genotype by Environment Interaction, CRC Press, Boca Raton

Gregory PH (1968). Interpriting plant disease dispersal gradients. Annu. Rev. Phytopathol. 6: 189-212

Gu K, Tian D, Yang F, Wu L, Sreekala C, Wang D, Wang GL and Yin Z (2004). High resolution genetic mapping of Xa27(t), a new bacterial blight resistance gene in rice, *Oryza sativa* L. Theor. Appl. Genet. 108: 800-807

IRRI (International Rice Research Institute) (2008). Standard Evaluation System for Rice. Manila, the Philippines, International Rice Research Institute

Jeger MJ, Jones DG and Griffiths E (1983). Components of partial resistance of wheat seedlings to Septoria nodorum. Euphytica 32: 575-584

Jeger MJ and Viljanen-Rollinson SLH (2001). The use of the area under disease progress curve (*AUDPC*) to assess quantitative disease resistance in crop cultivars. Theor. Appl. Genet. 102(1): 32-40

Kendall MG and Stuart A (1968). The Advanced Theory of Statistics. Vol.3, 2nd Edn. London, Charles Griffin and Co. Ltd.

Kiyosawa S (1981). Gene analysis for blast resistance. Oryza 18: 196-203

Kranz J (1974a). Comparison of epidemics. Anu Rev Phytopathol. 12(1): 355-374

Kranz J (1974b). The role and scope of mathematical analysis and modeling in epidemiology. *In*, Kranz, J. (ed.) Epidemics of Plant Diseases, Mathematical analysis and modelling Springer-Verlag, Berlin pp.7-54

Lebeda A and Jendrulek T (1987a). Application of cluster analysis for establishment of genetic Similarity in gene-for-gene host parasite relationships. J Phytopathol. 119(2): 131-141

Lebeda A and Jendrulek T (1987b). Cluster analysis as a method for evaluation of genetic similarity in specific host-parasite interaction (*Lectuca sativa-Bremia lactucae*). Theor. Appl. Genet. 75(1): 194-199

Lebeda A and Jendrulek T (1988). Application of methods of multivariate analysis in comparative pidemiology and research into field resistance. J. Plant Dis. Prot. 95(5): 495-505

Li W, Yan ZH, Wei YM, Lan XJ and Zheng YL (2006). Evaluation of genotype x environment interaction in Chinese spring wheat by the *AMMI* model, correlation and path analysis. J. Agron. Crop Sci. 192: 221-227

Lin CS, Binns MR and Lefkovitch LP (1986). Stability analysis, Where do we stand Crop Sci. 26(5): 894-900

Lin CS and Binns MR (1988). A superiority measure of cultivar performance for cultivar ×location data. Can. J. Plant Sci. 68: 193-198

Luke HH and Berger RD (1982). Slow-rusting in oats compared with logistic and Gompertz models. Phytopathology 72: 400-402

Madden LV and Pennypacker SP (1979). Principal component analysis of tomato early blight epidemics. J. Phytopathol. 95(4): 364-369

Marchetti MA (1983). Dilatory resistance to rice blast in USA rice. Phytopathology 73: 645-649

Marchetti MA and Xianghua L (1986). Screening techniques to identify slow-blasting rice lines. *In*, Progress in

Upland Rice Research, International. Rice Research. Institute, Manila, Philippines pp. 317-326

Minogue KP and Fry WE (1983). Models for the spread of plant disease, Some experimental results. Phytopathology 73: 1173-1176

Mohapatra NK, Mukherjee AK, Mahana NK and Nayak P (2001). Relative importance of leaf position in critical evaluation of blast disease of rice. Oryza 38(3&4): 140-142

Mohapatra NK, Mukherjee AK, Suriya Rao AV and Nayak P (2008). Disease progress curves in the rice blast pathosystem compared with the logistic and Gompertz models. J. Agric. Biol. Sci. 3 (1): 28-37

Mohapatra NK, Mukherjee AK and Nayak P (2013). Identification of slow-blasting rice genotypes through multivariate analysis of disease progress curves. J. Agric. Biol. Sci. 8(10): 702-710

Mohapatra NK, Mukherjee AK, Suriya Rao AV, Jambhulkar NN and Nayak P (2014). Comparison of different parameters for evaluation of partial resistance to rice blast disease. American Journal of Experimental Agriculture 4(1): 58-79

Mukherjee AK, Mohapatra BK and Nayak P (1996). The use of selection indices for identification of slowblasting rice genotypes. Int. J. Tropical Plant Dis. 14: 179187

Mukherjee AK and Nayak P (1997a). Association among the components of slow-blasting resistance in rice. J Mycol. Plant Pathol. 27: 175-183

Mukherjee AK and Nayak P (1997b). Methods of estimating chlorotic area from the linear measurements of necrotic area of rice blast lesions. Int. J. Trop. Plant Dis. 15(2): 177-181

Mukherjee AK, Mahana NK and Nayak P (1997a). In situ estimation of lesion area of rice blast disease using linear measurements. Indian Phytopath. 50: 431-433

Mukherjee AK, Mohapatra NK and Nayak P (1997b). Technique for determination of infection frequency and infection efficiency as components of slow-blasting resistance in rice. Oryza 35: 184-185

Mukherjee AK, Suriya Rao AV and Nayak P (1998). Stable slow-blasting resistance in rice. Ann. Plant Prot. Sci. 6(1): 11-18

Mukherjee AK, Mohapatra NK, Suriya Rao AV and Nayak P (2005). Effect of nitrogen fertilization on the expression of slow-blasting resistance in rice. J. Agric. Sci. 143: 385-393

Mukherjee AK, Mohapatra NK and Nayak P (2010). Estimation of area under the disease progress curves in a rice blast pathosystem from two data points. Eur. J. Plant Pathol. 127(1): 33-39

Mukherjee AK, Mohapatra NK and Nayak P (2013a). Identification of slow-blasting rice genotypes through multivariate analysis of components of resistance. J. Agric. Biol. Sci. 8(2): 125-138

Mukherjee AK, Mohapatra NK, Bose LK, Jambhulkar NN and Nayak P (2013b). Additive main effects and multiplicative interaction(*AMMI*) analysis of GxE interactions in rice-blast pathosystem to identify stable resistant genotypes. African J. Agric. Res. 8(44): 5492-5507

Nayak D, Bose LK, Reddy PR and Nayak P (2008a). Host-pathogen interaction in rice-bacterial blight pathosystem. J. Plant Prot. Res. 48(3): 371-384

Nayak D, Bose LK, Singh S and Nayak P (2008b). Additive Main Effects and Multiplicative Interaction analysis of host-pathogen relationship in rice-bacterial blight pathosystem. The Plant Pathol. J. 24 (3): 337-351

Nayak D, Bose LK, Singh UD, Singh S and Nayak P (2008c). Measurement of genetic diversity of virulence in populations of *Xanthomonas oryzae* pv. *oryzae* in India. Commun. Biometry and Crop Sci. 3:16-28

Nayak D, Reddy PR and Nayak P (2009).Variability in *Xanthomonas oryzae* pv. *oryzae*, the incitant of bacterial blight disease of rice. J. Plant Prot. Res. 49(1): 15-26

Nayak P and Chakrabarti NK (1986). Stable resistance to bacterial blight disease in rice. Ann. Appl. Biol. 109(1): 179-186

Nayak P, Suriya Rao AV and Chakrabarti NK (1987). Components of resistance to bacterial leaf blight disease of rice. Phytopath. Z. 119: 312-318

Nayak P, Mukherjee AK, Pandit E and Pradhan SK (2018). Application of statistical tools for data analysis and interpretation in rice plant pathology. Rice Sci. 25(1): 1-18

Ngugi HK, Esker PD and Scherm H (2011). Meta-analysis to determine the effect of plant disease management measures, Review and case studies on soybean and apple. Phytopathology 101(1): 31-41

Nomura K. and Ishi K (1989). Effects of the components of field resistance to *Pyricularia oryzae* in rice on disease development and relationship between the components. Bulletin of the College of Agriculture

and Veterinary Medicine - Nihon University (Japan).Source: http://agris.fao.org/agris-search/search.do?recordID=JP9202311

Onasanya A, Sere Y, Nwilene F, Abo ME and Akator K (2004). Reactions and resistance status of differential rice genotypes to rice yellow mottle virus, Genus Sobemovirus in Cote d' Ivoire. Asian J. Plant Sci. 3: 718-723

Parlevliet JE (1979). Components of resistance that reduce the rate of epidemic development. Anu. Rev. Phytopathol. 17: 203-222

Perkins JM and Jinks JL (1968). Environmental and genotype environmental components of variability. III. Multiple lines and crosses. Heredity 23: 239-256

Perez Mangas JM (1981). Quantitative methods for the evaluation of horizontal resistance on several genotypes to rice blast disease. *In*, Proceedings of the Symposium on Rice Research to Blast, held at Montpellier, France, 18-21 March 1981: 123-40

Pethybridge SJ, Esker P, Dixon P, Hay F, Groom T, Wilson C and Nutter FW Jr. (2007). Quantifying loss caused by ray blight disease in Tasmanian pyrethrum fields. Plant Disease 91: 1116-1121

Piepho HP (1996). Analysis of genotype environment interaction and phenotypic stability. *In*, M.S. Kang, and H.G. Zobel Jr (Eds), pp. 151-174. Genotype by Environment Interaction. CRC Press, Boca Raton

Piepho HP (1998). Methods for comparing the yield stability of cropping system -a review. J. Agro. Crop. Sci. 180, 193-213. *In*, M.S. Kang, and H.G. Zobel Jr (Eds), pp. 151-174

Purchase JL (1997). Parametric stability to describe G-E interactions and yield stability in winter wheat. PhD. Thesis, Department of Agronomy, Faculty of Agri., Univ. of Orange Free State, Bloemfontein, South Africa

Purchase JL, Hatting H and Vandeventer CS (2000). Genotype × environment interaction of winter wheat (*Triticum aestivum* L.) in South Africa, ?. Stability analysis of yield performance. South Afric. J. Plant Soil 17:101-107

Rao M, Lakshmikantha RG, Kulkarni RS, Lalitha Reddy SS and Ramesh S (2004). Stability analysis of sunflower hybrids through non-parametric model. Helia 27: 59-66

Rees RG, Thompson JP and Mayer RJ (1979a). Slow-rusting and tolerance to rust in wheat, I.The progress and effects of epidemics of *Puccinia graminis*, *tritici* in selected wheat cultivars. Aust. J. Agric. Res. 30(3): 403-419

Rees RG, Thompson JP and Goward EA (1979b). Slow-rusting and tolerance to rust in wheat,II. The progress and effects of epidemics of *Puccinia recondita tritici* in selected wheat cultivars. Aust. J. Agric. Res. 30(3): 421-432

Robinson RA (1969). Disease resistance terminology. Rev, Appl. Mycol. 48: 593-606

Robinson J and Jalli M (1999). Sensitivity of resistance to net blotch in barley. J. Phytopath. 147: 235

Rodriguez M and Galvez GE (1975). Indication of partial resistance of rice to the fungus *Pyricularia oryzae* Cav. *In*, Proceedings of the Symposium on Horizontal Resistance to the Blast Disease of Rice. CIAT CE-NO. 9, Cali. Columbia:137-54

Romagosa I and Fox PN (1993). Genotype-Environment Interaction and Adaptation. M.D. Haward, N.O. Bosemark, and I. Romagosa (Eds), pp. 374-390. Plant Breeding, Principles and prospects, Chapman and Hall, London

Ryu JD, Yeh WH, Han SS, Lee YH and Lee EJ (1987). Regional and annual fluctuation of races of *Pyricularia oryza* during 1978-1985 in Korea. Korean J. Plant Pathol. 3: 174-179

Ryu JD, Lee EJ and Oh JH (1990). The factors associated with race nonspecific resistance of Seomjinbyeo to rice blast (*Pyricularia oryzae* CAV.). AGRIS SN - 1013-9389. The Research Reports of the Rural Development Administration - Crop Protection (Korea R.). web link:http://agris.fao.org/agris-search/search.do?recordID=KR9035980

Sah DN and Bonman JM (1992). Effects of seedbed management on blast development in susceptible and partially resistant rice cultivars. J. Phytopathol. 136: 73-81

Sanogo S and Yang XB (2004). Overview of selected multivariate statistical methods and their use in phytopatholgical research. Phytopathology 94: 1004-1006

Schneider JHM, van den Boogert PHJF and Zadoks JC (1999). Exploring differential interactions between *Rhizoctonia solani* AG 2-t isolates and tulip cultivars. Plant Disease 83(5): 474-481

Shaner G and Finney RE (1977). The effect of nitrogen fertilization on the expression of slow-mildewing

resistance in knox wheat. Phytopathology 67: 1051-1056

Shukla GK (1972). Some aspects of partitioning genotype-environmental components of variability. Heredity 28: 237-245

Sun GC, Shi D, Zhuge GZ and Sun SY (1990). Some components of partial resistance to blast in indica rices. Int. Rice Res. Newsl. 15: 11-12

Thompson JP and Rees RG (1979). Pattern analysis in epidemiological evaluation of cultivar resistance. Phytopathology 69(6): 545-549

Tredway LP, Stevenson KL and Burpee LL (2003). Components of resistance to *Magnaporthe grisea* in 'Coyote' and 'Coronado' tall fescue. Plant Disease. 87: 906-912

Tukey JW (1977). Exploratory Data Analysis. Addison-Wesley Pub Co. 26: 163-182

Van der Plank JE (1963). Plant Diseases, Epidemics and Control. New York, Academic Press, pp. 349

Van der Plank JE (1968). Disease Resistance in Plants. - Academic Press, New York

Villareal RL, MacKenzie, DR, Nelson, RR and Coffman WR (1980). Apparent infection rates of Pyricularia oryzae on different rice cultivars. Phytopathology 70: 1224-26

Villareal RL, Nelson RR, MacKenzie DR and Coffman WR (1981). Some components of slow- blasting resistance in rice. Phytopathology 71: 608-611

Waggoner PE (1986). Progress curves of foliar diseases, Their interpretation and use. Pages 3-37, *In*, Plant Disease Epidemiology, Population dynamics and management. Vol. I. Leonard, K.J. and Fry, E.W. (Ed.), McMillan Publishing Co. New York, pp. 372

Wang Z, Mackill DJ and Bonman JM (1989). Inheritance of partial resistance to blast in indica rice cultivars. Crop Sci. 29: 848-853

Wang QF and Wang QM (1991). Study on slow-blasting resistance of rice to *Pyricularia oryzae* Cav. Acta Phytopathologica Sinica 13: 97-102

Wilcoxson RD, Skovmand B and Atif AF (1975). Evaluation of wheat cultivars for ability to retard development of stem rust. Ann. Appl. Biol. 80: 275-281

Williams WT (1976). Pattern Analysis in Agricultural Science. Amsterdam, Oxford and New York, CSIRO, Melbourne and Elsevier, pp. 331

Wricke G (1962). On a method of understanding the biological diversity in field Research. Z.Pfl.-Zücht 47: 92-146

Yan W and Falk DE (2002). Biplot analysis of host-by-pathogen data. Plant Disease. 86: 1396-401

Yau SK (1995). Regression and AMMI analysis of genotype-environment interactions, an Empirical comparison. Agron. J. 87: 121-126

Zhang Z, Lu C and Xiang ZH (1998). Stability analysis for varieties by AMMI model. Acta Agron. Sin. 24: 304-309

Zobel RW, Wright MJ and Gauch HG Jr. (1988). Statistical analysis of a yield trial. Agron. J. 80: 388-393