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Additive main effects and multiplicative interaction (AMMI) analysis of GxE interactions in rice-blast pathosystem to identify stable resistant genotypes

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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.* The genotypes were raised in uniform blast nursery in a randomized complete block design with three replications. The GEI was analyzed following the regression models as well as additive main effects and multiplicative interaction (AMMI) model. AMMI analysis of variance revealed that the first two interaction principal component axes (IPCA) explained 37.28 and 33.47% of the interaction effects in 14.63 and 14.02% of interaction degrees of freedom, respectively and rest of the five IPCAs were noisy. Integrating biplot display and genotypic stability statistics enabled five groupings of genotypes based on similarities in their performance across environments. The biplot generated using the environment and genotype scores for the first two IPCAs revealed the positioning of the five host genotype groups (HG) into four sectors. HG-1 constituting of 28 genotypes exhibiting low stability index (D_i values), low IPCA-1 as well as IPCA-2 scores and low mean disease scores across seasons of testing, were identified as possessing stable resistance to the disease. 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.

Key words: Additive main effects and multiplicative interaction (AMMI) model, rice blast disease, *Magnaporthe oryzae,* regression model, stable resistance.

INTRODUCTION

Rice blast disease caused by *Magnaporthe oryzae* is one of the serious diseases causing heavy yield losses in most of the rice growing countries. Sincere attempts have been made for identification of several resistant varieties as well as genes governing resistance to different races of the causal pathogen. The presence of wide variability in pathogen population has further complicated the resistance breeding program (Wescott, 1985). Breeders and plant pathologists are often charged with the responsibility of identification and development of stable

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resistant varieties as an economic method of disease control strategy. Selection of stable resistant genotypes across diverse environments represents the ideal for making optimal progress in breeding program. Proper identification and characterization of resistance, its efficient use in breeding program and disease management strategy is facilitated by relating its phenotypic expression to genotype and environment main effects and GEI. During the past few decades, various statistical techniques like joint regression analysis (JRA), AMMI and GGE-biplot have been used to analyze mostly yield parameters (Crossa et al., 1990; Adhikari et al., 1995; Yoshitola et al., 1997; Gauch and Zobel, 1996) and scarcely, disease resistance. The application of regression model has been made for stable blast (Gu et al., 2004; Mukherjee et al., 1998) and bacterial blight (Nayak and Chakrabarti, 1986) resistance in rice. The potentiality of AMMI model was demonstrated for identification of stable blast resistance in rice (Abamu et al., 1998), broom rape resistance in faba beans (Flores et al., 1996) and late blight resistance in potato (Forbes et al., 2005). Stable net blotch resistance in barley was identified by application of both AMMI and JRA models (Robinson and Jalli, 1999).

Stable resistance in the host plant can be evaluated in terms of the number of years it retains original level of resistance and whether it is effective against a large number of different pathogen-genotypes (van der Plank, 1971). In order to identify stable resistance, the host genotypes have to be exposed to repeat testing under different environments, either through multi-location trials during the same year or repeated testing at the same location during several years (Gu et al., 2004). The classical analysis of GEI concentrates on the analysis of stability rather than that of adaptation. The analysis is based on the regression of varietal performance on a site index as proposed and modified by (Finlay and Wilkinson, 1963; Eberhart and Russell, 1966; Perkins and Jinks, 1968; Freeman and Perkins, 1971). Apart from concentrating on stability, these models are also very restrictive in the type of interaction for which they account. These models assume a strong linear relationship between the varietal performance and environmental factors, which requires a dominant physical gradient over environments.

More flexible statistical models for describing GEI such as the AMMI model are useful for a better understanding of GEI. The AMMI model is a hybrid analysis that incorporates both the additive and multiplicative components of the two-way data structure. AMMI biplot analysis is considered to be an effective tool to diagnose GEI patterns graphically. The additive portion is separated from interaction by analysis of variance. The principal component analysis (PCA), which provides a multiplicative model, is applied to analyze the interaction effect from the additive ANOVA model. The biplot display of PCA scores plotted against each other provides visual inspection and interpretation of GEI components. The

integration of biplot display and genotypic stability statistics enables genotypes to be grouped on the basis of similarity in performance across diverse environments.

The great potentiality of AMMI model has been successfully utilized to analyze the GEI and identify stable resistant host genotypes. Following the biplot graphic display of matrices with the application of principal component analysis (Gabriel, 1971), the differential host pathogen interactions between Rhizoctonia solani isolates and tulip cultivars were analyzed by Schneider et al. (1999) and between 10 rice yellow mottle virus isolates and 13 differential host genotypes by (Onasanya et al., 2004). Host-pathogen interaction between 52 isolates of *Xanthomonas oryzae* pv. oryzae and 16 rice genotypes employing AMMI model (Nayak et al., 2008) 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. Effective breeding programs on disease resistance depends on a thorough understanding of the complex host-pathogen interactions, which could be simplified following statistical models like the pattern analysis, principal component analysis, AMMI model or GGE biplot display analysis. The objective of the present study was to (i) analyze and interpret the GEI in rice blast pathosystems, (ii) assess the response of the genotypes across environments and (iii) recognize stable blast resistant genotypes for use in breeding programs.

MATERIALS AND METHODS

Plant material and growing conditions

Seeds of 42 rice genotypes were collected from the International Germplasm collections, International Rice Research Institute (IRRI), Philippines through the National Bureau of Plant Genetic Resources (NBPGR), New Delhi, India and the National Germplasm collections maintained at the Central Rice Research Institute, Cuttack, India. The seeds were sown in a uniform blast nursery with the susceptible check variety Karuna sown in alternate rows as well as all around the nursery, as spreader rows. Thus each test variety was raised within one meter long single-row plot, surrounded by susceptible spreader rows of Karuna. Seeds were sown with a spacing of 10 cm between rows and 5 cm between plants. The experiment was conducted in a randomized complete block design with three replications. High nitrogen fertilizer (100 kg N ha^{-1}) in the form of ammonium sulfate was applied in split doses. High relative humidity was maintained throughout the period of experimentation, by running the sprinkler irrigation system during hotter periods of the day (10 am to 3.30 pm) with an intermittent stoppage of half an hour after each hour of irrigation. The experiment was repeated over a period of nine seasons (5 dry + 4 wet seasons), from dry season 1997 to dry season 2001.

Recording observation and statistical analysis

Critical observations on the percent host tissue damaged by the disease were recorded on the day the spreader rows of susceptible check Karuna succumbed to the disease with 100% severity. The data on average percentage of the terminal disease severity

recorded for five severely infected plants in each test variety were used for analysis of GEI and identification of stable resistant genotypes. The stable performance of 42 rice genotypes tested over a period of nine seasons, was analyzed following regression models of Eberhart and Russell (1966), Perkins and Jinks (1968) and Freeman and Perkins (1971). Genotypes with disease severity scores of ≤10%, regression coefficient b_i=0 and deviation from regression as small as possible ($S^2{}_d$=0) was considered to be highly stable. The AMMI model was applied, with additive effects for the 42 rice genotypes (G) and nine seasons of testing (Environments=E) and multiplicative term for GxE interactions. The AMMI analysis first fits additive effects for host genotypes and environments by the usual additive analysis of variance procedure and then fits multiplicative effects for GxE by principal component analysis (PCA). The AMMI model is

$$Y_{ij} = \mu + g_i + e_j + \sum_{k=1}^{n} \lambda_k \, \pounds_{ik} \, Y_{jk} + R_{ij}$$

where Y_{ij} is the disease score of the ith genotype in the jth environment, g_i is the mean of the ith genotype minus the grand mean, λ_k 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 ith genotype and the jth environment, respectively and R_{ij} is the residual. The environment and host genotypic PCA scores are expressed as unit vector times; the square root of λ_k, that is, environment PCA score = $\lambda_k^{0.5} \, Y_{ik}$; host genotype PCA score = $\lambda_k^{0.5} \, \pounds_{ik}$ (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:

$$D_i = \sqrt{\sum_{s=1}^{c} Y^2_{is}}$$

where, c is the number of significant IPCs, and Y^2_{is} is the scores of the host genotype i in IPCs.

The AMMI analysis was conducted using the computer software INDOSTAT (2004) for windows. To assess fitting AMMI model, predictive and post-dictive approaches offered by Zobel et al. (1988) were applied to the data. The association among the stability parameters was verified by Pearson's correlation analysis.

RESULTS

The disease progress

Conducive conditions created by application of high nitrogenous fertilizer, closer spacing and maintenance of high humidity through sprinkler irrigation system helped in creation of high disease pressure during all the nine seasons of experimentation. The average disease reaction expressed by the 42 rice genotypes over the nine test environments was ranged between 0.93 and 100%, while the mean response of nine environments averaged over 42 host genotypes, was ranged between 15.67 and 26.43%; the general mean being 20.45% (Table 1). The environmental index ranged from -4.78 to 5.97. The genotypes 14 and 42 showed highest level of 100% severity, while the genotypes 1, 2, 3, 6, 7, 9, 11, 13, 15, 16, 18, 19, 20, 21, 23, 24, 26, 27, 28, 29, 30 and 31 showed consistently resistant reactions across

seasons of testing. The rest of the genotypes exhibited variable degrees of resistance to susceptibility reactions. The distribution properties of disease severity (%) among 42 rice genotypes tested across nine seasons presented in a box plot (Figure 1) depicts the degree of dispersion in the population. The minimum and maximum disease scores indicated a broad spectrum of disease reactions ranging from 0 to 100% severity. A significant shift in box position as well as median values towards lower end signified the positive skewness in the distribution. Small inter-quartile range implied high uniformity or low variations among the central 50% observations. There were a number of outliers in the population on the higher side, the origin of which were traced back to the susceptible genotypes.

AMMI analysis of variance

The AMMI analysis of variance of 42 rice genotypes tested over nine environments revealed that 84.04% of the total sum of squares (SS) was attributable to the genotypes (G), 1.05% to the environments (E) and 14.91% to GEI effects (Table 2). A large SS for G indicated that the genotypes were diverse with large differences among the means, causing most of the variations in the level of their disease reactions. The small proportion of SS for E indicated that the difference among the environmental means was not very high. The magnitude of GxE SS was 5.64 times smaller than that for the SS for G, thus indicating that the differences in the response of the genotypes across environments were not that substantial.

The first interaction principal component axis (IPCA-1) accounted for 37.28% of the interaction SS in 14.63% of the interaction degrees of freedom. Similarly, IPCA-2 explained further 33.47% of the interaction SS. The mean square (MS) for both IPCA-1 and IPCA-2 were significant at P = 0.01 level and cumulatively contributed to 70.75% of the total interaction. Therefore, the post-dictive evaluation using F-test at P = 0.01 suggested that these two IPCAs of the interaction were significant for the model with 94 degrees of freedom. IPCAs 3 to 7 captured mostly noise, since the MS were not significant, together explained only 28.75% of the total SS and therefore did not help to predict validation observations. Thus the interaction of the 42 genotypes across nine environments was best predictable by the first two principal components.

AMMI-1 biplot display

The graphical representation of AMMI analysis reveals the main effect means on the abscissa and IPCA-1 scores of both host genotypes as well as the environments simultaneously on the ordinate. The interaction is described in terms of differential sensitivities

Table 1. Mean blast disease scores (severity %) for 42 rice genotypes across nine environments.

Genotype	DS 1997	WS 1997	DS 1998	WS 1998	DS 1999	WS 1999	DS 2000	WS 2000	DS 2001	Mean
DZ-192	3.33	5.83	3.33	1.67	3.33	1.67	3.33	3.33	3.33	3.24
DM-27	0.00	3.33	0.00	1.67	6.67	1.67	3.33	3.33	1.67	2.41
Tieu-phai	0.00	0.00	0.00	1.67	6.67	3.33	3.33	3.33	3.33	2.41
E-425	3.33	15.00	3.33	6.67	8.33	6.67	3.33	6.67	3.33	6.30
Mak-thua	33.33	6.25	33.33	1.67	1.67	3.33	3.33	3.33	1.67	9.77
Sam houang	0.00	2.08	0.00	6.67	6.67	3.33	3.33	3.33	6.63	3.56
Sakai	3.33	0.83	3.33	0.41	3.33	1.67	0.83	1.67	6.67	2.45
Seritus malam-A	3.33	25.00	6.67	3.33	6.67	6.67	3.33	6.67	6.67	7.59
Seritus malam-B	0.00	0.00	0.00	6.67	8.33	6.67	3.33	6.67	6.67	4.26
Jumi-1	0.00	23.33	0.00	6.67	6.67	3.33	1.67	0.83	6.67	5.46
Laurent-TC	3.33	2.08	3.33	1.67	6.67	3.33	3.33	4.16	3.33	3.47
Chiang-tsene-tao	1.67	15.00	1.67	3.33	3.33	1.67	6.67	1.67	3.33	4.26
Chokoto	0.00	0.00	0.00	0.00	6.67	0.00	3.33	3.33	6.67	2.22
India dular	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Raj bhawalta	6.67	0.00	6.67	1.67	6.67	1.67	0.83	1.67	6.67	3.61
Sechi aman	0.00	0.00	0.00	0.00	3.33	3.33	1.67	3.33	3.33	1.67
Surjamukhi	16.67	6.67	16.67	33.33	6.67	6.67	1.67	3.33	3.66	10.59
IR-5533-PP-854	0.00	0.00	0.00	3.33	6.67	6.67	3.33	6.67	6.67	3.70
Madhukar	0.00	0.00	0.00	0.00	3.33	6.67	3.33	6.67	3.33	2.59
Milayeng-51	0.00	0.00	0.00	0.00	3.33	0.00	1.67	1.67	1.67	0.93
PTB-8	3.33	0.00	3.33	2.08	0.00	0.00	0.00	0.41	3.33	1.39
Dahanala-2014	33.33	1.67	3.33	3.33	4.16	3.33	1.67	6.67	4.17	6.85
Lien-tsan-50-A	8.33	1.67	8.33	3.33	3.32	3.33	3.33	1.66	3.66	4.11
Lien-tsan-50-B	0.00	0.00	0.00	3.33	6.67	3.33	3.33	3.33	6.67	2.96
N-22	33.33	1.67	33.33	16.67	16.67	10.83	6.67	10.83	16.67	16.30
Salum pikit	0.00	0.00	0.00	3.33	3.33	1.67	3.33	1.67	1.67	1.67
PTB-18	6.67	0.00	6.67	0.83	4.10	0.00	6.67	4.16	3.33	3.60
DNJ-155	3.33	0.00	3.33	3.33	3.33	6.67	1.67	6.67	3.33	3.52
DJ-88	3.33	4.17	3.33	1.67	3.33	3.20	10.00	3.33	6.67	4.34
UCP-188	0.00	0.00	0.00	3.33	3.33	0.00	6.67	1.04	3.33	1.97
Goda heenati	3.33	0.00	3.33	6.67	6.67	0.00	6.67	2.08	6.67	3.94
Kalubalawee	33.33	100.0	33.33	66.67	33.33	33.33	16.67	66.67	16.67	44.44
Bakka-biasa	100.0	33.33	100.0	100.0	33.33	33.33	16.67	66.67	33.33	57.41
Tiace	100.0	100.0	100.0	100.0	100.0	50.00	66.67	100.0	100.0	90.74
ARC-7046	100.0	66.67	100.0	100.0	33.33	50.00	33.33	100.0	33.33	68.52
Prolific	33.33	0.00	33.33	3.33	3.33	0.83	3.33	3.33	3.33	9.35
Pusa-4-1-11	100.0	25.00	100.0	16.67	66.67	50.00	33.33	66.67	33.33	54.63
Ratna	33.33	100.0	100.0	100.0	33.33	20.00	33.33	20.00	33.33	52.59
Jaya	66.67	100.0	100.0	33.33	66.67	66.67	66.67	66.67	66.67	70.37
CR-289-1045-16	100.0	15.00	100.0	66.67	100.0	50.00	100.0	33.33	100.0	73.89
CR-570	0.00	15.00	0.00	6.67	8.30	3.33	6.67	6.67	8.37	6.11
Karuna	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Mean	24.68	20.70	26.43	21.94	19.96	15.67	16.23	20.08	18.41	20.45
Env. index	4.23	0.25	5.97	1.49	-0.50	-4.78	-4.23	-0.37	-2.05	

DS = dry season; WS = wet season.

of the genotypes 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 appearing almost on a
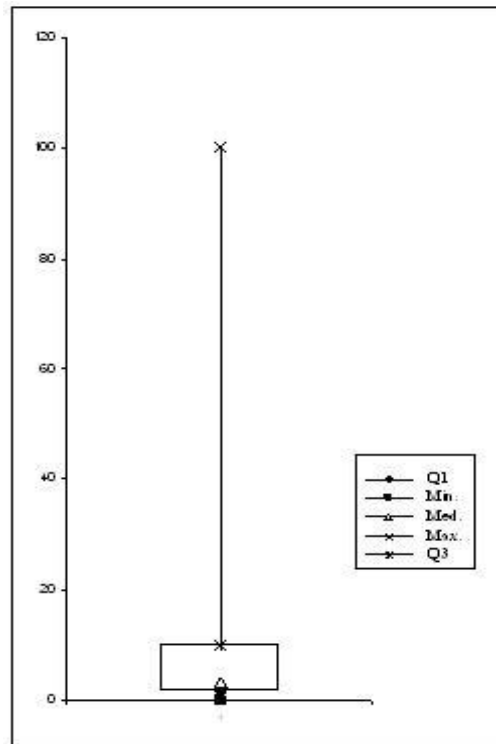
Figure 1. Box plot depicting the distribution properties of disease severity (%) among 42 rice genotypes across nine seasons of testing (♦ q1, ■ min.,▲ med., × max., ＊ q3).

**Table 2.** AMMI analysis of variance for resistance of 42 rice genotypes across nine environments.

Sources of variation	Degrees of freedom	Sum of squares	Mean squares	% Variance explained
Total	377	404609.84	1073.24**	
Genotypes(G)	41	340045.96	8293.80**	84.04
Environments(E)	8	4247.66	530.96**	1.05
GxE Interaction	328	60316.22	183.89**	14.91
AMMI IPCA-1	48	22485.56	468.45**	37.28
AMMI IPCA-2	46	20186.17	438.83**	33.47
AMMI IPCA-3	44	7872.75	178.93	13.05
AMMI IPCA-4	42	6032.20	143.62	10.00
AMMI IPCA-5	40	2183.19	54.58	3.62
AMMI IPCA-6	38	737.61	19.41	1.22
AMMI IPCA-7	36	515.88	14.33	0.86
Residual	34	302.86	8.91	0.50

**Significant at P=0.01 level.

perpendicular line have similar means and those falling almost on a 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 (Figure 2) and used to predict the expected level of resistance for any host genotype and environment combination. For any GE combination in the AMMI-1
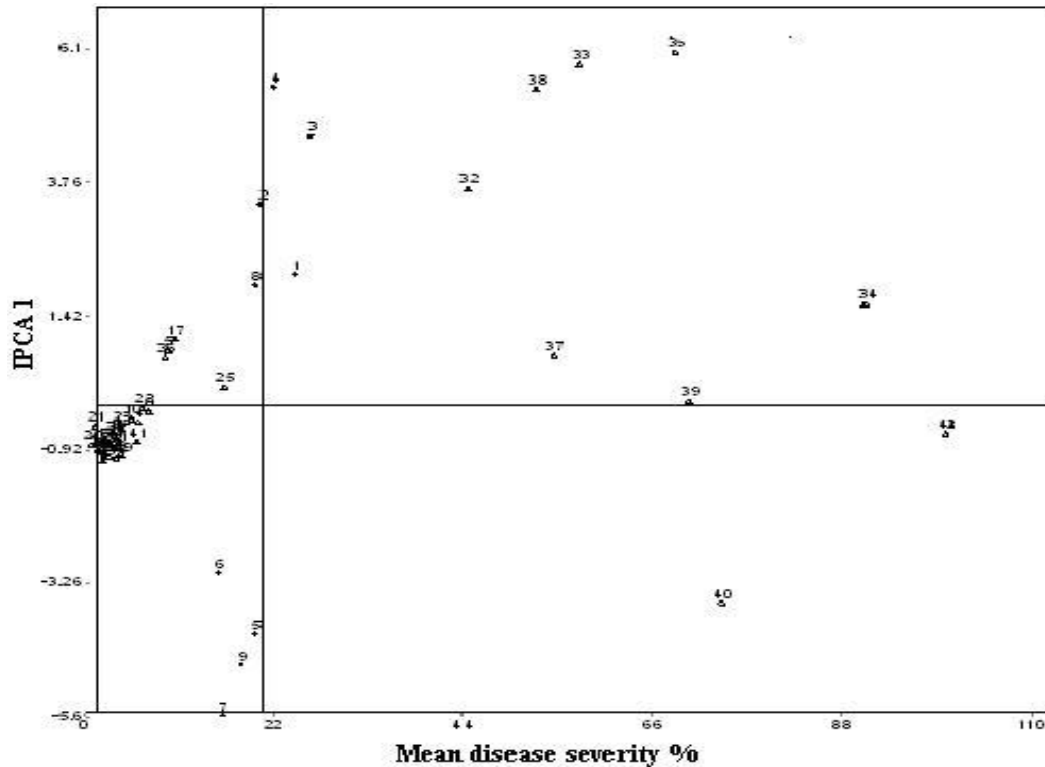
Figure 2. AMMI-1 biplot display of mean blast disease severity 5 and IPCA-1 scores of 42 genotypes across nine environments. Host genotypes (▲), Environments (♦). The numerals for host genotypes and environments are provided in Tables and 3, respectively.

biplot (Figure 2), the additive part (main effects) of the AMMI model equals the G mean plus E mean minus the grand mean. The multiplicative part (interaction effects) is the product of G and E IPCA-1 scores. For example, the highly resistant genotype-20 with environment-9 had a main effect of 0.93%+18.41%-20.45%=-1.11%. The interaction effects would be the products of the respective IPCA-1 scores = -0.82 x -4.68 = 3.84. The AMMI model estimated the resistance of genotype-20 in environment-9 as -1.11 + 3.84 = 2.73%, which fits the observed resistance level of 1.67%. Host genotypes and environments with IPCA-1 scores of the same sign produce positive interactions effects, while the combinations of IPCA-1 scores of opposite signs have negative specific interactions.

Five groupings of the host genotypes (HG) are evident from the biplot generated from the present study (Figure 2). HG-1 includes 28 host genotypes *viz.* 1, 2, 3, 4, 6, 7, 8, 9, 10, 11, 12, 13, 15, 16, 18, 19, 20, 21, 22, 23, 24, 26, 27, 28, 29, 30, 31 and 41 with mean disease score of 3.61% which is much less than the grand mean (20.45%). This group of genotypes has small negative IPCA-1 scores ranging from -1.10 to -0.18. For these genotypes, the AMMI-1 model predicts disease reactions that are close to those of the AMMI-0 model on the environments E-5, 6, 7 and 9 with negative IPCA-1

scores ranging from -0.56 to -0.31 and are well adapted to above respective environments. These genotypes have smallest interactions and hence possess most stable non- race-specific resistance to blast disease.

HG-2 consists of 4 host genotypes viz. 5, 17, 25 and 36 with a mean severity level of 11.49%, which is although less than the grand mean (20.45%) possess moderate resistance to the disease. They have small positive IPCA-1 scores ranging from 0.20 to 1.02 and are well adapted to the environments E-1, 2, 3, 4 and 8 with positive IPCA-1 scores ranging from 1.95 to 5.43. These genotypes have small interactions possessing race-specific resistance to the disease in specific environments.

HG-3 includes 4 host genotypes viz. 32, 33, 35 and 38 with a mean severity level of 55.74% which is much above the grand mean, that is, high susceptible scores. These genotypes have high positive IPCA-1 scores ranging from 3.65 to 6.05. This group of genotypes is well adapted to the environments E-1, 2, 3, 4 and 8 with high positive IPCA-1 scores ranging from 1.95 to 5.43. They have very high interactions and hence are highly unstable across the nine environments.

HG-4 consists of 2 host genotypes namely, 34 and 37 with a mean severity level of 72.69% which is much above the grand mean and possesses high susceptible reactions. They have relatively higher positive IPCA-1

scores ranging from 0.74 to 1.63 and are well adapted to the environments E-1, 2, 3, 4 and 8 with IPCA-1 scores ranging from 1.95 to 5.43. They have high interactions and hence are highly unstable.

HG-5 constitutes 4 host genotypes viz. 14, 39, 40 and 42 with the highest mean severity level of 86.07% which is much above the grand mean and possesses high susceptibility levels. These genotypes have low negative IPCA-1 scores ranging from -0.07 to -3.59 and are well adapted to the environments E-5, 6, 7 and 9 with high negative IPCA-1 scores ranging from -3.08 to -5.57. They possess stable susceptibility over the environments.

The direction and magnitude of differences among genotypes along the abscissa (disease severity %) and ordinate (IPCA-1 scores) can be read from the graph (Figure 2). The best resistant genotypes should show zero to low level of disease severity and should be stable across the tested environments. Any host genotype showing lower absolute IPCA-1 score would produce a lower absolute GxE interaction effect than that with higher absolute IPCA-1 score and have less variable disease reactions (that is, more stable) across the environments. The host genotype stability ranking based on lower absolute IPCA-1 scores was HG-1 (-0.18 to - 1.10), HG-5 (-0.07 to -3.59), HG-2 (0.20 to 1.02), HG-4 (0.74 to 1.63) and HG-3 (3.65 to 6.05). The groups of host genotypes in HG-1 and HG-2 are depicted on the horizontal axis in AMMI-1 biplot display (Figure 2). HG-1 has very high level of resistance with mean disease severity level of 3.61% and low negative IPCA-1 scores. HG-2 has moderate level of resistance with mean disease severity level of 11.49% and low positive IPCA-1 scores. Hence the 28 genotypes in HG-1 were identified as possessing highly stable resistance, while the four genotypes in HG-2 possessed relatively moderate stable resistance to the disease. The genotypes in HG-3, HG-4 and HG-5 exhibited high levels of disease severity much above the grand mean, small to large absolute IPCA-1 scores which produced higher absolute interaction effects. Thus, these genotypes have more variable disease reactions across the tested environments and hence are considered as unstable.

The nine environments show variability in the main effects and interactions, the IPCA-1 scores showing clear higher negative or positive interactions (Figure 2) due to the two groups of environments (EG). EG-1 constituting of E-1, E-2, E-3, E-4 and E-8 showed highest main effects and large positive IPCA-1 scores, while EG-2 consisting of E-5, E-6, E-7 and E-9 showed high response to the host genotypes with high negative interaction IPCA-1 scores.

Response of five host genotype groups to nine environments

Disease severity of five host genotype groups averaged over nine environments ranged from 3.61% for HG-1 to 86.07% for HG-5 and the response of nine environments ranged between 34.80% for E-7 and 62.93% for E-3 (Table 3). The ranking of the HGs in ascending order of their average severity levels was HG-1<HG-2<HG-3<HG-4<HG-5 and similar ranking of environments on the basis of their average response was E-6<E-7<E-9<E-5<E-2<E-8<E-4<E-1<E-3. The genotypes in HG-1 exhibited highest degree of resistance with the disease severities ranging from 2.14 to 5.11% across nine environments. HG-2 exhibited varied degrees of resistance ranging from 3.65 to 29.16% showing susceptibility under E-1, E-3 and E-4. The host genotypes under HG-3, HG-4 and HG-5 exhibited consistently high degree of susceptibility across all the nine environments.
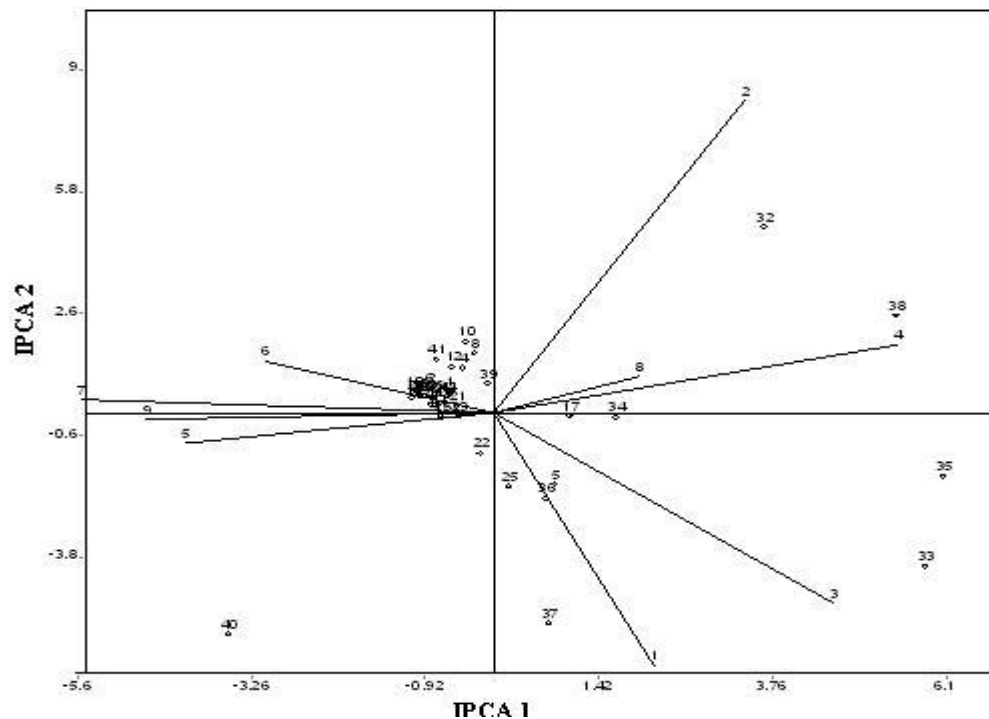
AMMI-2 biplot display

The AMMI is an explorative technique by which the GxE relationship can be expressed in terms of interaction patterns derived in bi-plots. A bi-plot is a graphical representation in which genotypes and environments are displayed simultaneously. The interaction is described in terms of differential sensitivities of the genotypes to the most discriminating environmental variables (AMMI-axes) that can be constructed. These environmental variables and the genotype sensitivities are estimated from the table itself. 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. In the present experiment, the genotypes 32, 33, 35, 38 in HG-3; 37 in HG-4 and 40 in HG-5 have relatively greater contribution to the interaction than the others (Figure 3). The aforementioned genotypes with vector end points far apart, show considerable interactions with rest of the genotypes including the 28 genotypes in HG-1. Genotypes, for which the directions of the vectors almost coincide, have similar pattern of interactions like those in HG-1 and HG-5. On the other hand, when the directions are opposite, the interaction patterns of the corresponding genotypes show negative correlation like those within HG-1, HG-5; and HG-2, HG-3, HG-4. Thus the genotypes and environments showing considerable interactions could be easily identified from the bi-plot.

AMMI analysis extracted values of the scores for IPCA-1 to IPCA-7 in respect of 42 host genotypes as well as nine environments. A biplot is generated using the IPCA-1 and IPCA-2 scores for the 42 host genotypes and 9 environments with the first principal component axis on the abscissa and the second on the ordinate (Figure 3). The biplot displayed both the host genotypes and environments simultaneously in four sectors of a single scattered plot depending upon the positive or negative signs of the scores on the first two principal components. Sector-1 represents host genotypes or environments with

Table 3. Mean response (severity %) of 5 host genotype groups (HG) exposed to nine environments.

Environment	HG-1	HG-2	HG-3	HG-4	HG-5	Mean
DS-1997	3.09	29.16	66.67	100.00	91.67	58.12
WS-1997	4.11	3.65	75.00	62.50	78.75	44.80
DS-1998	2.14	29.16	83.33	100.00	100.00	62.93
WS-1998	2.92	13.75	91.67	58.34	75.00	48.34
DS-1999	5.11	7.08	33.33	83.34	91.67	44.11
WS-1999	2.97	5.41	34.16	50.00	79.17	34.34
DS-2000	3.57	3.75	25.00	50.00	91.67	34.80
WS-2000	3.79	5.21	63.34	83.34	75.00	46.14
DS-2001	4.80	6.25	29.16	66.67	91.67	39.71
Mean	3.61	11.49	55.74	72.69	86.07	45.92

DS = dry season; WS= wet season.

Figure 3. AMMI-2 biplot display of 42 rice genotypes and nine environments for their response to blast disease. Scores on the first axis (IPCA-1) account for 37.28% and second axis (IPCA-2) account for 33.47% of GxE SS. Environment points are at the end of the spike. The numerals for genotypes and environments are provided in Tables 1 and 3, respectively.

positive IPCA-1 as well as IPCA-2 scores, while sector-2 represent 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. In the present study, the environments were distributed into four sectors in the following manner: E-2, E-4 and E-8 in Sector-1; E-1 and E-3 in sector-2; E-5 and E-9 in sector-3; and E-6 and E-7 in sector-4. The ranking of the environments in order of their level of response to the disease was E-6<E-7<E-9<E-5<E-2<E-8<E-4<E-1<E-3. Among the host genotype groups, HG-2 and HG-4 fell into sector-2, HG-3 fell into both sector-1 and 2, HG-1 into both sector 3 and 4 and HG-5 into sector-3 and 4. The ranking of these groups of genotypes according to their levels of resistance was HG-1 >HG-2 >HG-3 >HG-4 >HG-5. Thus the biplot not only displayed the GEI but also facilitated in visual description of 'which win where' pattern.

A polygon drawn in the biplot (Figure 3) by joining the host genotypes located farthest from the bi-plot origin, encompassing all other host genotypes, facilitates identification of the genotypes that are most resistant in specific environments. The vertex genotype in a sector is most or least resistant to the environment falling in that sector. In the present study, the vertex genotypes 32, 33, 35, 37, 38 and 40 exhibit susceptibility in all the environments. All the 28 genotypes in HG-1 exhibited highly stable resistance in all the nine environments. However, three of the four genotypes in HG-5 showed high susceptibility in all the nine environments. The two genotypes in HG-4, though not positioned at the vertex, were susceptible in all the nine environments. The response of five genotype in HG-2 were variable, that is, resistant in E-2, E-5, E-6, E-7, E-8 and E-9; and susceptible in E-1, E-3 and E-4 (Table 3).

There was a highly significant correlation between the mean disease severity percent and the IPCA-1 scores (r = 0.398**). Hence, the 'G' main effects can be represented by the IPCA-1 scores for the genotypes. The genotypes with lower IPCA-1 scores would produce a lower absolute GxE interaction effect than those with higher absolute IPCA-1 scores and have less variable degree of resistance (more stable) across environments. The stability ranking of the genotypes based on lower absolute IPCA-1 scores was those in HG-1, HG-5, HG-2, HG-4, HG-3. Thus the genotype group HG-1 (More resistant) and HG-5 (More susceptible) possessed high stability across the tested environments. Among them the genotypes in HG-1 exhibited least mean disease severity and hence possessed stable resistance. The genotypes in HG-5 exhibited highest level of susceptibility for which these were considered as possessing stable susceptibility.

The discriminating ability of the environments can be judged by calculating the distance of each environment from the biplot origin. In this regard, the environments E-1, E-2, E-3, E-4 and E-7 are most discriminating as indicated by long distance from the biplot origin. Host genotypes with IPCA-1 scores >0 responded positively (adaptable) to the environments that had IPCA-1 scores > 0 (that is, their interaction is positive), but responded negatively to the environments that had IPCA-1 scores <0. The biplot revealed that the genotypes in HG-2, HG-3 and HG-4 with IPCA-1 scores >0 responded positively to the environments E-1, E-2, E-3, E-4 and E-8 with IPCA-1 scores >0 and hence their interaction is positive and the genotypes in these three HG groups are adaptable to the five environments.

AMMI stability index 'D$_i$'

The distance of interaction principal component point with the origin in space is the AMMI stability coefficient 'D_i'. The estimate of the stability index 'D_i' incorporates the

IPCA scores of the significant IPCs depending upon their contributions towards the interaction SS. The stability index is useful in evaluation and identification of host genotypes possessing stable resistance. The lower D_i values indicate high stable resistance across the tested environments and *vice versa*. In the present experiment, there was a significant correlation between mean disease severity and the stability index D_i (r = 0.501**).

The ranking of host genotype groups in ascending order of D_i values was those in HG-1 (0.47 to 1.89), HG-2 (1.02 to 2.34), HG-4 (1.64 to 5.56), HG-5 (0.76 to 6.82) and HG-3 (5.99 to 7.08). The 28 genotypes in HG-1 exhibited low D_i values with mean disease score of 3.61%, low negative IPCA-1 scores (-1.10 to -0.18) and consistently resistant reaction across nine environments of testing. Hence these 28 genotypes were identified as possessing stable resistance to the disease. The four genotypes in HG-2 showed low D_i values (1.03-2.34) and small positive IPCA-1 scores (0.20-1.02). These genotypes showed high degree of resistance with occasional susceptibility during one season or the other and hence were considered as possessing average stability. Rest of the genotypes in HG-3, HG-4 and HG-5 showed high levels of disease severity scores, high D_i values, variable IPCA-1 and IPCA-2 scores and hence were not considered as possessing stable resistance to the disease (Table 4). However, the genotypes viz. 14, 39 and 42 showed low D_i values, small negative IPCA-1 scores and consistent high disease scores. Hence these genotypes were considered as possessing stable susceptibility to the disease.

Interaction pattern from response plot

Response plots (Figure 4) indicate the nature of GEI with the main effects of genotypes and environments removed. The values plotted for each genotype group by environments 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. In the present study, 28 genotypes in HG-1 showed positive interactions with E-2, E-5, E-6, E-7, E-8 and E-9 and negative interactions with E-1, E-3 and E-4. These genotypes also showed low mean disease scores and were located near the centre of the biplot in sector-2 (Figure 3). They showed low negative IPCA-1 and positive IPCA-2 scores and hence were recognized as possessing stable resistance to blast disease. The four genotypes in HG-2 showed positive interactions with E-1, E-3, E-4 and negative interactions with E-2, E-5, E-6, E-7, E-8 and E-9; moderately low mean disease scores. These genotypes were located near the centre of the biplot in sector-2, with low positive IPCA-1 scores and

Table 4. Mean blast disease score, estimates of IPCA scores and AMMI stability index (D_i) for 42 rice genotypes across nine environments.

Genotype	Mean	IPCA-1	IPCA-2	IPCA-3	IPCA-4	D_i value
DZ-192	3.24	-0.60	0.53	-0.18	0.02	0.80
DM-27	2.41	-0.85	0.64	-0.20	0.28	1.06
Tieu-phai	2.41	-1.01	0.47	-0.21	0.48	1.11
E-425	6.30	-0.41	1.16	-0.32	-0.01	1.23
Mak-thua	9.77	0.82	-1.86	-0.29	-1.61	2.03
Sam houang	3.56	-0.88	0.64	0.12	0.78	1.09
Sakai	2.45	-0.79	0.21	-0.08	0.17	0.82
Seritus malam-A	7.59	-0.26	1.59	-0.32	-0.95	1.61
Seritus malam-B	4.26	-1.00	0.57	-0.22	0.98	1.15
Jumi-1	5.46	-0.38	1.85	0.24	-0.40	1.89
Laurent-TC	3.47	-0.80	0.32	-0.27	0.22	0.86
Chiang-tsene-tao	4.26	-0.55	1.21	0.09	-0.30	1.35
Chokoto	2.22	-1.10	0.41	-0.08	0.36	1.17
India dular	100.00	-0.63	0.43	-0.16	0.28	0.76
Raj bhawalta	3.61	-0.71	-0.11	0.01	0.10	0.72
Sechi aman	1.67	-0.91	0.46	-0.35	0.39	1.02
Surjamukhi	10.59	1.02	-0.09	1.15	1.65	1.02
IR-5533-PP-854	3.70	-1.08	0.54	-0.39	0.75	1.21
Madhukar	2.59	-1.00	0.52	-0.67	0.50	1.13
Milayeng-51	0.93	-0.82	0.42	-0.16	0.31	0.92
PTB-8	1.39	-0.50	0.18	0.01	0.30	0.53
Dahanala-2014	6.85	-0.18	-1.05	-1.18	0.63	1.06
Lien-tsan-50-A	4.11	-0.48	-0.09	-0.03	0.06	0.47
Lien-tsan-50-B	2.96	-1.05	0.48	-0.04	0.64	1.15
N-22	16.30	0.20	-1.91	0.25	-0.01	1.92
Salum pikit	1.67	-0.79	0.48	-0.03	0.57	0.92
PTB-18	3.60	-0.71	-0.09	-0.13	0.08	0.72
DNJ-155	3.52	-0.67	0.28	-0.55	0.57	0.73
DJ-88	4.34	-1.02	0.46	0.02	0.14	1.12
UCP-188	1.97	-0.94	0.47	0.20	0.56	1.05
Goda heenati	3.94	-0.85	0.22	0.41	0.65	0.88
Kalubalawee	44.44	3.65	4.88	-1.82	-0.01	6.09
Bakka-biasa	57.41	5.83	-4.01	0.30	2.58	7.08
Tiace	90.74	1.63	-0.13	0.86	-0.03	1.64
ARC-7046	68.52	6.05	-1.67	-2.28	1.76	6.28
Prolific	9.35	0.69	-2.24	-0.05	-1.17	2.34
Pusa-4-1-11	54.63	0.74	-5.51	-3.59	-3.46	5.56
Ratna	52.59	5.42	2.54	6.15	-2.61	5.99
Jaya	70.37	-0.07	0.76	-0.88	-5.97	0.76
CR-289-1045-16	73.89	-3.59	-5.80	4.82	0.22	6.82
CR-570	6.11	-0.77	1.41	0.01	0.18	1.61
Karuna	100.00	-0.63	0.43	-0.16	0.28	0.76

negative IPCA-2 scores; and hence were considered as possessing average stability. The four genotypes in HG-3 showed negative interactions of high magnitude with E-5, E-6, E-7, E-9 and positive interactions of high magnitude with E-1, E-2, E-3, E-4, E-8; positive IPCA-1 scores and both positive and negative IPCA-2 scores. These genotypes were located far away from the centre of biplot at the apex of the polygon and showed mean disease scores much above the grand mean. Hence this group of genotypes was considered to be highly unstable. Two genotypes in HG-4 showed positive interactions with E-1, E-3, E-5, E-8 and negative interactions with E-2, E-4, E-
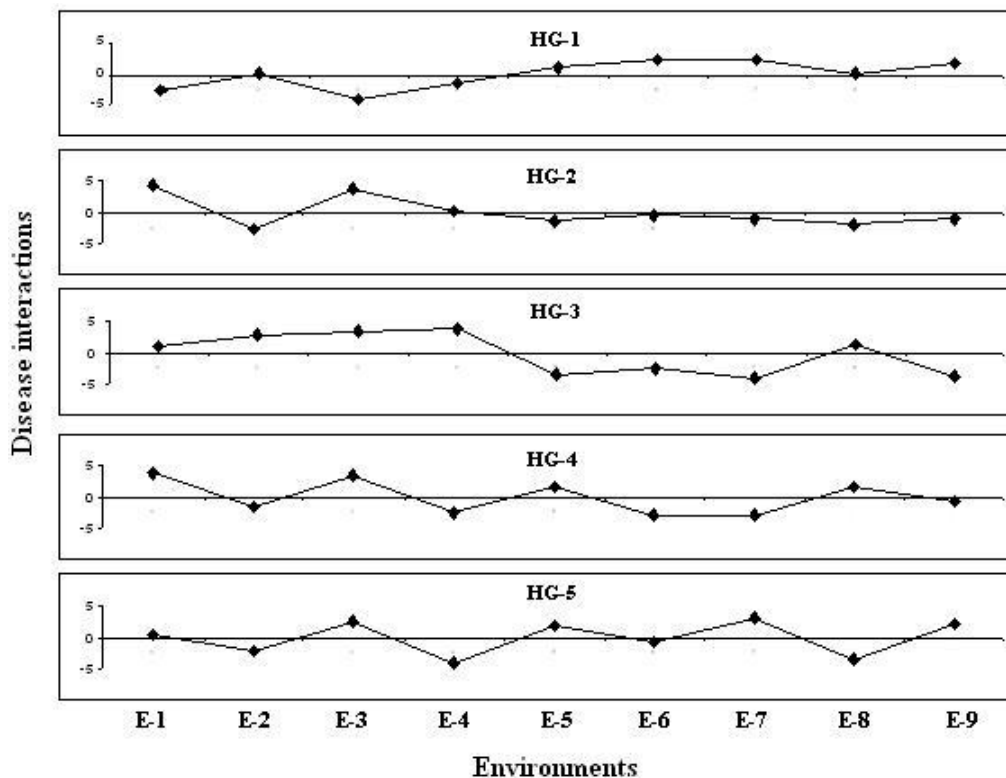
Figure 4. Response plot for five genotype groups and nine environments.

6, E-7, E-9; positive IPCA-1 and negative IPCA-2 scores. These genotypes were located away from the centre of the biplot; showed high mean disease scores and hence were considered as highly unstable. The four genotypes in HG-5 showed negative interactions of low magnitude with E-2, E-4, E-6, E-8 and positive interactions of similar magnitude with E-1, E-3, E-5, E-7, E-9. These genotypes were positioned at the centre of the biplot with negative IPCA-1 and positive IPCA-2 scores. These genotypes were recognized as possessing stable susceptibility due to their highest mean disease scores across nine environments.

Stability analysis by regression models

Attempts were made to compare different stability parameters analysed by AMMI model and regression models. A critical comparison among the three regression models (E and R, P and J, F and P) based on GEI from ANOVA tables and genotype rankings based on b_i and S^2_d, revealed similar trends. Hence, the ANOVA (Table 5) and GEI (Table 6) for Eberhart and Russell model only are presented here. The highly significant G and E mean squares revealed that the disease scores for 42 genotypes are significantly different from each other and the environments also represented an array of diverse

conditions for disease development. The pooled ANOVA showed that the GEI was a linear function of the additive environmental component. The GEI was further partitioned into linear and nonlinear components. The highly significant mean squares (MS) for these components indicated the presence of both predictable and unpredictable components of GEI. Highly significant GxE (linear) interaction indicated the presence of genetic differences among the genotypes for their regression on the environmental index. Significantly larger pooled deviation over pooled error indicated the existence of a significant departure from linearity and therefore some of the GEI cannot be predicted from the linear regressions.

In the present context of stable disease resistance, host genotypes exhibiting low mean disease scores, small regression coefficient ($b_i = 0$) and minimum deviation from regression ($S^2_d = 0$) were considered as possessing above average stability; those exhibiting low mean disease scores, $b_i = 1$ and $S^2_d = 0$ were considered as possessing average stability, while those showing high disease scores, $b_i = 1$ and $S^2_d = 1$ as most unstable for disease resistance. The GEI in the present study revealed that 12 genotypes viz. 5, 17, 25, 32, 33, 34, 35, 36, 37, 38, 39 and 40 showed high mean disease scores, large interactions, $b_i \geq 1$ (Figure 5) and large MS-deviations as well as MS-regressions (Table 6) and hence were considered unstable. The two genotypes viz.

Table 5. ANOVA for blast disease scores of 42 rice genotypes exposed to nine seasons.

Sources of variation	d.f.	Sum of squares	Mean squares	F. Ratio
Replication within Env.	18	9.57	0.53	0.004
Genotypes (G)	41	340045.59	8293.79	59.19**
Environments (E)	8	4247.38	530.92	3.79**
E+(GxE)	336	64563.92	192.15	1.37**
GxE	328	60316.54	183.89	1.31**
Environments (Linear)	1	4247.38	4247.38	30.31**
GxE (Linear)	41	19121.50	466.38	3.33**
Pooled Deviation	294	41195.04	140.12	261.96**
Pooled Error	738	394.75	0.53	
Total	377	404609.58	1073.23	

*Significant at P=0.01 level; d.f. = degrees of freedom.

Table 6. Mean blast disease scores (severity %), the slope of regression (b_i), ms deviation (s^2_d), the contribution of genotypes to interactions for 42 rice genotypes exposed to nine environments.

Genotypes	Mean score	Slope(b_i)	MS-Dev. (S^2_d)	MS-GxE	MS-REG.
1. DZ-192	3.24	0.060*	1.62	12.58	89.31
2. DM-27	2.41	-0.264*	3.84	23.57	161.70
3. Tieu-phai	2.41	-0.385*	3.50	27.31	193.94
4. E-425	6.30	-0.117*	16.30	30.05	126.25
5. Mak-thua	9.77	3.049*	71.92	116.02	424.72
6. Sam houang	3.56	-0.373*	6.06	29.13	190.65
7. Sakai	2.45	0.072*	4.32	14.66	87.02
8. Seritus malam-A	7.59	-0.004	51.68	57.97	101.98
9. Seritus malam-B	4.26	-0.557*	9.10	38.62	245.31
10. Jumi-1	5.46	-0.243	59.67	71.75	156.30
11. Laurent-TC	3.47	-0.047*	2.25	15.83	110.87
12. Chiang-tsene-tao	4.26	-0.194*	20.97	36.36	144.10
13. Chokoto	2.22	-0.319*	8.06	29.06	176.07
14. India dular	100.00	0.000*	0.00	12.64	101.13
15. Raj bhawalta	3.61	0.409	7.51	10.98	35.39
16. Sechi aman	1.67	-0.324*	1.66	23.59	177.15
17. Surjamukhi	10.59	1.683	75.87	72.28	47.13
18. IR-5533-PP-854	3.70	-0.598*	5.77	37.35	258.38
19. Madhukar	2.59	-0.563*	4.24	34.60	247.14
20. Milayeng-51	0.93	-0.126*	1.45	17.30	128.27
21. PTB-8	1.39	0.297*	1.64	7.68	49.92
22. Dahanala-2014	6.85	1.285	91.35	80.96	8.23
23. Lien-tsan-50-A	4.11	0.500*	3.57	6.29	25.33
24. Lien-tsan-50-B	2.96	-0.427*	5.11	30.22	206.04
25. N-22	16.30	2.359	54.91	71.39	186.71
26. Salum pikit	1.67	-0.226*	1.63	20.43	151.97
27. PTB-18	3.60	0.303*	7.60	12.80	49.14
28. DNJ-155	3.52	-0.109*	4.93	19.87	124.43
29. DJ-88	4.34	-0.363*	5.24	28.06	187.77
30. UCP-188	1.97	-0.317*	4.83	26.17	175.54
31. Goda heenati	3.94	-0.021*	9.29	21.29	105.34
32. Kalubalawee	44.44	1.565	837.67	737.00	32.26
33. Bakka-biasa	57.41	8.276*	368.48	991.72	5354.38

Table 6. Contd.

34. Tiace	90.74	3.759	201.50	272.53	769.72
35. ARC-7046	68.52	6.750*	488.18	845.15	3343.91
36. Prolific	9.35	3.135*	71.06	119.79	460.93
37. Pusa-4-1-11	54.63	5.380	675.29	833.43	1940.36
38. Ratna	52.59	5.761	999.30	1160.89	2291.98
39. Jaya	70.37	1.559	423.45	374.47	31.62
40. CR-289-1045-16	73.89	1.913	1253.52	1107.36	84.26
41. CR-570	6.11	-0.537*	20.45	47.74	238.77
42. Karuna	100.00	0.000*	0.00	12.64	101.13

Slope = slopes of regressions of genotype means on environmental index; ms-dev=deviation from regression component of interaction (s^2_d); ms-gxe=contribution of each genotype to interaction ms; ms-reg = contribution of each genotype to the regression component of the gxe interaction; * = slopes significantly different from 1.00 at p = 0.05 level.
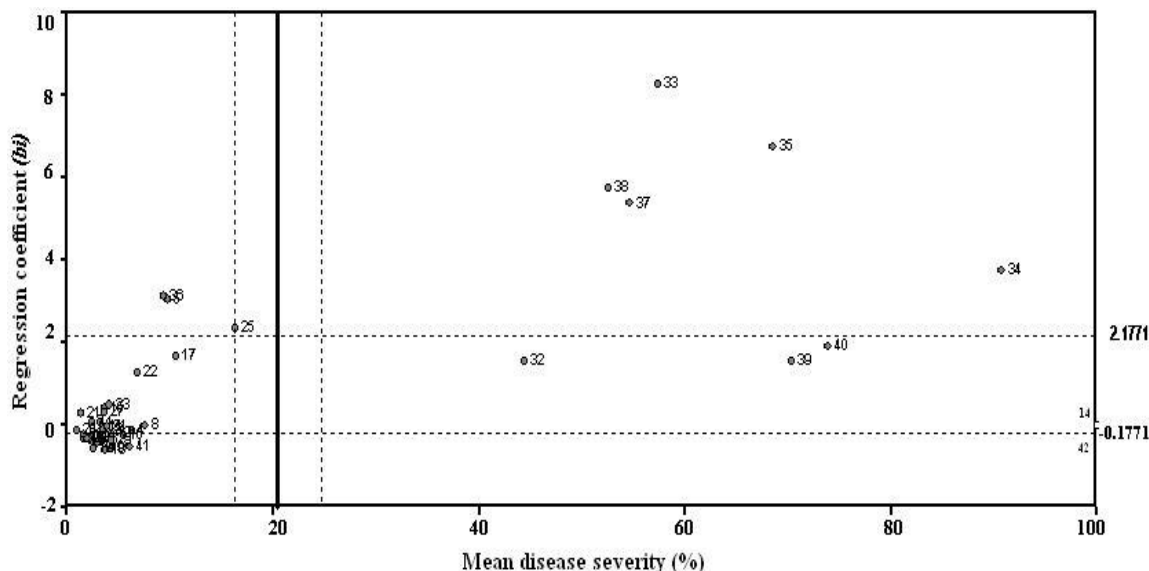

Figure 5. Relationship between mean blast disease scores of 42 rice genotypes and the corresponding stability parameters 'b_i'. The numerals for genotypes are provided in Table 1.

14 and 42, although showed small interactions, zero MS-deviations and b_i = 0; were considered as possessing stable susceptibility due to highest mean disease scores. Four genotypes viz. 8, 10, 15 and 22 showed b_i = 1, small to high deviation from regression as well as interactions and hence were considered as possessing average stability. The 24 genotypes viz. 1, 2, 3, 4, 6, 7, 9, 11, 12, 13, 16, 18, 19, 20, 21, 23, 24, 26, 27, 28, 29, 30, 31 and 41 showed b_i = 0, S^2_d = 0, small interactions and low minimum disease scores across environments, thus exhibiting unresponsiveness in their disease reactions to environmental changes and hence were considered as possessing above average stability in their resistance to rice blast disease.

Association among different stability parameters estimated following E and R model, P and J model, F and

P model, AMMI model was verified by calculating Pearson's correlations (Table 7). Highly significant positive correlation of mean disease score with all the stability parameters implied that stable resistance to the disease across environments was associated with low stability indices. The strong relationship among the parameters further indicated that both regression and AMMI parameters were equally efficient in identification of stable resistant genotypes. A critical comparison of 42 genotypes for their stability across nine environments revealed perfect agreement between the regression and AMMI models in the instability in expression of disease reaction of 12 genotypes viz. 5, 17, 25, 32, 33, 34, 35, 36, 37, 38, 39 and 40 in HG-2, HG-3, HG-4 and HG-5. The two genotypes 14 and 40 possessed stable susceptibility in both the models. Out of rest of the 28 genotypes

Table 7. Correlation among the stability parameters for 42 rice genotypes across　nine seasons of testing.

	D_i	IPCA-1	B_i ER	S^2_d ER	B_i PJ	S^2_d PJ	B_i FP	S^2_d FP
Mean	0.501**	0.398**	0.541**	0.566**	0.541**	0.566**	0.533**	0.565**
D_i		0.662**	0.794**	0.868**	0.794**	0.868**	0.789**	0.858**
IPCA-1			0.833**	0.430**	0.833**	0.430**	0.842**	0.419**
B_i ER				0.609**	0.999**	0.609**	0.998**	0.603**
S^2_d ER					0.609**	0.999**	0.595**	0.997**
B_i PJ						0.609**	0.997**	0.609**
S^2_d PJ							0.595**	0.996**
B_i FP								0.589**

Mean = Mean disease severity %; D_i = AMMI stability index; B_i = slope value; S^2_d = Deviation from regression; ER = Eberhart and Russell model; PJ = Perkins and Jinks model; FP = Freeman and Perkins model; ** = Significant at $P = 0.01$ level.

recognized as possessing stable resistance in AMMI model, 24 genotypes in HG-1 of AMMI model were also recognized as possessing stable resistance in three regression models with $b_i = 0$, $S^2_d = 0$. However, four genotypes viz. 8, 10, 15 and 22 recognized as possessing stable resistance in AMMI model, were considered possessing average stability in regression models with $b_i = 1.0$, high S^2_d values and high interactions, mainly due to high disease scores above grand mean in one environment or the other.

DISCUSSION

GEI for resistance to diseases is the differential response of genotypes to changing environmental conditions (Mahalingam et al., 2006). A stable resistant genotype should possess high levels of resistance with low degree of fluctuations, when grown over diverse environments. Analysis of GEI for genotypes reduce errors in the breeding process as the selection in one condition cannot provide advantages in others. Among several methods of univariate and multivariate stability statistics proposed for analysis of GEI, AMMI analysis is widely used because it captures a large portion of GEI SS, clearly separates main and interaction effects and provides meaningful interpretations of the data (Ebdon and Gauch, 2002). The estimation of IPCAs is based on the GE residual matrix. An IPCA reflects some systematic pattern in the matrix indicating environment specific genotype response. Under complex situations, several significant IPCAs may be computed by the AMMI analysis to describe the interactions contained in the residual matrix. But the first few IPCAs capture the largest part of them and thus greatly reduce the dimensionality of the problem.

In the present study, the largest part (85%) of the total variation of blast disease severity was explained by G and E main effects. Of the remaining variation, 70.75% were explained by the significant GEI captured by the first two IPCAs. Further IPCAs captured mostly noise and therefore, did not help to predict validation observation.

The present observation is in agreement with those reported by Nayak et al. (2008), Forbes et al. (2005), Robinson and Jalli (1999) and Flores et al. (1996). On the contrary Abamu et al. (1998) reported significant contribution of first four IPCAs to GE SS. The GEI had a tremendous effect on the performance and ranking of the genotypes in specific environments, indicating that their disease reactions were impaired by interactions with the environment. For example, the genotypes in HG-2 expressed resistance in E-2, 5, 6, 7, 8 and 9 but susceptibility in E-1, 3 and 4 and hence were not stable across environments; while those in HG-1 expressed consistently resistance reactions in all nine environments and thus were highly stable. The genotypes in HG-3, 4 and 5 expressed susceptible reactions across all nine environments.

The AMMI model describes GEI in more than one dimension and offers better opportunities for studying and interpreting GEI than ANOVA and regression of the mean (Vargas et al., 2001). In AMMI, the additive portion is separated from the interaction by ANOVA. Then the IPCA analysis which provides a multiplicative model is applied to analyze the interaction effect from the additive ANOVA model. The biplot display of IPCA scores provides visual inspection and interpretation of the GEI. Integrating biplot display and genotypic stability statistics enables genotypes to be grouped based on similarity in their performance across diverse environments. The five HGs recognized through AMMI analysis of the present data set, exhibited distinct interactions of stable resistance (HG-1) and susceptibility reactions.

The results of AMMI analysis indicated that the model fits the data well which made it possible to construct the biplot and calculate genotype as well as environmental effects. The IPCA scores of a genotype in the AMMI analysis indicate the stability of the genotype across environments. The closer the IPCA scores to zero, the more stable the genotypes are across testing environments. In this study, the 28 genotypes in HG-1 exhibiting consistently low disease reactions across environments and expressing zero to negative IPCA-1

scores were recognized as possessing highly stable resistance compared with the rest four HGs which were unstable. The AMMI model and biplot were useful methods for the analysis of GEI for disease resistance in different plant-pathosystems (Flores et al., 1996; Abamu et al., 1998; Robinson and Jalli, 1999; Forbes et al., 2005 Nayak et al., 2008; Jalata, 2011). In the present study, GE biplot and the stability index D_i values were powerful tools for evaluation of stable blast resistant genotypes. The GE biplot not only displayed the interaction of genotypes and environments, but also facilitated a visual description of 'which win where' patterns. AMMI analysis also helped in explaining the GEIs in specific environments resulting in detection of suitable environment for each genotype group. Thus the present study identified HGs with specific positive adaptation to specific environments, which is a pattern of response that can be used in breeding for resistance to such environments. Overall, the expression of resistance of this group of genotypes to *M. oryzae* was robust, since the resistance levels were consistent in all tested environments. Conversely, the four genotypes in HG-5 showed low negative interactions in four and low positive interactions in five environments, positioned at the center of the biplot with consistent high mean disease scores and thus exhibited well adaptation to respective environments with high levels of stability in their susceptibility. Among them, the genotype42 (Karuna) is being used as a susceptible check in blast screening programs and the genotype-14 (India Dular) as a differential variety in race identification programs.

In the present study, partitioning and interpretation of GEI was based on three linear regression methods and the AMMI analysis. All methods explained similar SS patterns as evidenced from the respective ANOVA tables (Tables 2 and 5), thus indicating that they were equally efficient in partitioning and interpretation of GEI. The two stability measures (b_i and S^2_d) estimated following three regression methods along with the AMMI stability index D_i and IPCA-1 were found to be equally potential in determining the stability of rice genotypes to blast disease. This finding was substantiated by the highly significant correlations among these stability parameters (Table 7) and identification of 24 genotypes viz. 1, 2, 3, 4, 6, 7, 9, 11, 12, 13, 16, 18, 19, 20, 21, 23, 24, 26, 27, 28, 29, 30, 31 and 41 as possessing stable resistance, following all the methods. Since AMMI combines ANOVA and PCA in one model, it was found to be more useful and informative in depicting the stable response of genotypes. However, the regression models also remain a good option. The present findings corroborate with those reported by Pinnschmidt and Hovmoller (2002) that combined JRA and AMMI are very useful for improving the evaluation of varietal resistance to net blotch in barley. On the contrary, AMMI analysis was reported to be a potentially useful method in improving the resistance status of barley to net blotch (Robinson and Jalli, 1999)

and that of rice to blast disease (Abamu et al., 1998). The classical stability ANOVA is not effective for detailed study of underlying patterns of interactions, while the AMMI model has been found to be an effective tool for a more in-depth analysis of interactions (Zobel et al., 1988). AMMI is especially effective where the assumption of linearity of response of genotypes to changes in environment is not fulfilled (Adhikari et al., 1995; Zobel et al., 1988) which is required in stability analysis techniques of linear models. The AMMI model does not require this assumption, because it is a hybrid statistical model which incorporates both additive and multiplicative components of the two-way (G-E) data structure. It separates the additive main effects from the interaction which is analyzed as a series of multiplicative components using PCA and helps to indicate the interaction pattern. The strength of a correlation of the individual principal component with observed effect on the disease component can provide quantitative estimates of their importance as a possible cause of GEI (Ardales et al., 1996).

A promising candidate genotype for stable non-race-specific resistance would exhibit a good mean resistance level, little sensitivity to environmental mean disease levels as reflected by low slope (b_i)values and small specific GEI as reflected by IPCA scores close to zero as well as residuals of the full model close to zero (Pinnschmidt and Hovmoller, 2002, Tiawari et al., 2011). Among the 42 rice genotypes, the 28 genotypes in HG-1 fulfilled these criteria of possessing stable non-race-specific resistance to blast disease, since they showed mean disease scores of 3.6% across environments, small interactions (GEI MS = 6.29-80.96), small specific GEI as reflected by negative IPCA-1 scores (-1.10 to -0.18), IPCA-2 scores (-1.05 to 1.85) and small residuals of the full model close to zero. The genotypes in HG-2 possessing small positive IPCA-1 scores (0.20 to 1.02), negative IPCA-2 scores (-2.24 to -0.09), variable sensitivities to environments (9.35% to 16.30%) and high interactions (71.39 to 119.79), appeared to be possessing race specific resistance in certain environments.

Conclusion

Both regression and AMMI models were equally potential in partitioning the GEI. However, the AMMI model:

(1) Described the GEI in more than one dimension and offered better opportunities for understanding and interpreting GEI;
(2) Described the visual inspection of biplot display and interpretation of GEI facilitated identification of genotypes possessing stable resistance as well as discriminating environments;
(3) Described the specificity in resistance pattern and

adaptability of the genotypes to specific environments in a 'which won where' pattern, and

(4) Integrate the biplot display and genotypic stability statistics enabled grouping of genotypes based on similarity in their performance across environments.

The scientific information obtained could be useful to plant breeders and plant pathologists in supporting breeding program decisions. Thus, AMMI model emerged as a useful tool in partitioning and meaningful interpretation of GEI in rice blast pathosystem.

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