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Additive Main Effects and Multiplicative Interaction Analysis of Host-Pathogen Relationship in Rice-Bacterial Blight Pathosystem

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Host-pathogen interaction in rice bacterial blight pathosystem was analyzed for a better understanding of their relationship and recognition of stable pathogenicity among the populations of *Xanthomonas oryzae* pv. *oryzae*. A total number of 52 bacterial strains isolated from diseased leaf samples collected from 12 rice growing states and one Union Territory of India, were inoculated on 16 rice varieties, each possessing known genes for resistance. Analysis of variance revealed that the host genotypes (G) accounted for largest (78.4%) proportion of the total sum of squares (SS), followed by 16.5% due to the pathogen isolates (I) and 5.1% due to the I x G interactions. Application of the Additive Main effects and Multiplicative Interaction (AMMI) model revealed that the first two interaction principal component axes (IPCA) accounted for 66.8% and 21.5% of the interaction SS, respectively. The biplot generated using the isolate and genotypic scores of the first two IPCAs revealed groups of host genotypes and pathogen isolates falling into four sectors. A group of five isolates with high virulence, high absolute IPCA-1 scores, moderate IPCA-2 scores, low AMMI stability index 'D_i' values and minimal deviations from additive main effects displayed in AMMI biplot as well as response plot, were identified as possessing stable pathogenicity across 16 host genotypes. The largest group of 27 isolates with low virulence, small IPCA-1 as well as IPCA-2 scores, low D_i values and minimal deviations from additive main effect predictions, possessed stable pathogenicity for low virulence. 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 showing stable pathogenicity and most discriminating host genotypes, which could be useful in location specific breeding programs aiming at deployment of resistant host genotypes in bacterial blight disease control strategies.

Keywords : adaptation, AMMI model, biplot display, stable pathogenicity, *Xanthomonas oryzae* pv. *oryzae*

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Rice bacterial blight caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) is one of the serious diseases in most of the rice-growing countries in Asia (Mew, 1987). The use of resistant varieties is most economical, eco-friendly and effective method to control the disease. So far more than 25 *Xa* genes conferring resistance to *Xoo* have been identified, mostly in Japan and at the International Rice Research Institute, Manila, the Philippines (Ezuka and Kaku, 2000; Gu et al., 2004; Ogawa, 1993). These genes function in a race-specific manner. Widespread use of a few resistance genes might accelerate the selection of new pathogen races, at a rate equivalent to 1.64 times increase in specific virulence following resistant host plant selection pressure only after one cropping cycle (Nayak, 1986b). This might lead to a change in pathogen population structure.

The existence of high variability in pathogen population (Ezuka and Horino, 1974; Gupta et al., 1986; Mew and Vera Cruz, 1979; Mew et al., 1992; Nayak et al., 2008a); high levels of genetic diversity (Adhikari et al., 1995, 1999; Ardales et al., 1996; Leach et al., 1992; Nayak et al., 2008c; Nelson et al., 1994; Shanti et al., 2001; Yashitola et al., 1997) and recognition of matching virulence factors in the pathogen strains (Nayak, 1986a; Nayak et al., 2008b) have been reported. This has cautioned the breeders on selection of stable resistant parents for incorporation into their breeding program against specific races of the patho- gen possessing stable virulence.

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 in the host genotypes, the cultivars have to be exposed to repeated testings under different environments, either through multi-locational trials during the same year or repeated testings at the same location during several years. Leonard and Moll (1979) proposed the use of the classical stability analysis model of Eberhart and Russell (1966) for determination of stable resistance in the host genotypes or stable pathogenicity in pathogen strains. They considered each pathogen strain as a separate environment to which the host genotypes were exposed and conversely, each host genotype as a separate

environment for particular pathogen strains. Similar approaches have been made in the evaluation of sorghum lines for resistance to midge (Faris et al., 1979) and corn genotypes for resistance to several downy mildew pathogen strains (Singh, 1975). Hamid et al. (1982) attempted for identification of stable resistance to three fitness attributes among nine corn inbred lines against six isolates of *Cochliobolus carbonum* race-3 by considering each pathogen isolate as a separate environment to which the host genotypes were exposed. Similar approaches were also made to recognize stable pathogenicity in pathogen strains of *Xoo* (Nayak and Chakrabarti, 1985) and stable resistance to rice bacterial blight among the host genotypes (Nayak and Chakrabarti, 1986).

The classical analysis of genotype (G)×environment (E) interaction concentrates on the analysis of stability rather than 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) and 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 G×E interaction such as the Additive Main effects and Multiplicative Interaction (AMMI) model are useful for a better understanding of G×E interaction (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 (ANOVA). 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 AMMI model has been successfully utilized to analyze the G×E interactions and identify stable resistant host genotypes for broom rapes (*Orobanche* sp.) resistance in faba beans (Flores et al., 1996), blast resistance in rice (Abamu et al., 1998), net blotch resistance in barley (Robinson and Jalli, 1999) and late blight resistance in potato (Forbes et al., 2005). 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 and Van den Boogert (1999), and between 10 rice yellow mottle virus isolates and 13 differential host genotypes by Onasanya et al. (2004). Host-pathogen interaction between 8 isolate groups of *Pyrenophora teres* and 13 barley line groups was analyzed with the help of GGE biplot display analysis to arrive at some valuable conclusions on their relationships (Yan and Falk, 2002). Effective breeding programmes on disease resistance depends on a thorough understanding of the complex host genotype-by-pathogen strain 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 analyze and interpret the host-pathogen interaction in rice bacterial blight pathosystem, assess the response of pathogen isolates across the tested host genotypes and identify stable pathogenicity for use in breeding program for development of stable resistant host genotypes.

Materials and Methods

The experimental material comprised of 52 isolates of *Xoo*, isolated from diseased leaf samples collected from 12 rice-growing states and the union territory of Andaman and Nicobar Islands (listed in Table 3). The 16 host genotypes, each possessing known gene/gene combinations for resistance to *Xoo* (listed in Table 7) were used in the present study. Thirty-day-old healthy seedlings were transplanted in a well-puddled field with a spacing of 40×20 cm between rows and plants. High level of nitrogenous fertilizer in the form of urea was applied in three equal splits, first as basal, second at one month after planting and third at pre-emergence stage, in order to provide a total of 120 kg N/ha. The experiment was conducted in a randomized complete block design with four replications.

The plants were clip-inoculated (Kauffman et al., 1973) at boot leaf stage, with a pair of scissors every time dipped into the bacterial cell suspension containing ca. 10^9 cfu. ml^{-1}, prepared from 48 h old actively growing cultures of each isolate grown on modified Wakimoto agar medium. The lesion length developed below the point of inoculation was measured on 21 days after inoculation.

The stable performance of 52 isolates of *Xoo* and their relationship to the response of 16 rice genotypes was analyzed following Eberhart and Russell (1966) model by regressing the mean level of pathogenicity of each isolate on the stability index (equivalent to the environmental index), except that each host genotype was considered as a separate environment to which the pathogen strain was exposed (Leonard and Moll, 1979). An isolate with high

level of virulence, unit regression coefficient (b_i=1.0) and the deviation from regression as small as possible (S^2_d=0) was considered to be highly stable virulent isolate.

In addition to the classical stability analysis, the Additive Main effects and Multiplicative Interaction (AMMI) model was also applied, with additive effects for isolates (I) and host genotypes (G), and multiplicative term for I×G interactions (IGI) for the present set of data. 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

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

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

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

R_{ij} is the residual.

The host genotypic and isolate PCA scores are expressed as unit vector times the square root of λ_k *i.e.* host genotypic PCA score=$\lambda_k^{0.5}\, Y_{ik}$; isolate PCA score=$\lambda_k^{0.5}\, £_{ik}$ (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,

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

The AMMI analysis was 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) were applied to the data.

Results and Discussion

Classical stability analysis. The level of pathogenicity of 52 isolates of *Xoo*, averaged over 16 host genotypes (=environments) ranged between 5.18 cm and 12.33 cm, while the mean response of 16 host genotypes averaged over 52 pathogen isolates ranged between 3.34 cm and 15.86 cm, the general mean being 6.65 cm (Table 1). The environmental index ranged from –3.30 to 9.22. Among the host genotypes, Rantai Emas (*Xa 1+Xa 2*), IR-8 (*Xa 11*) and IR-20 (*Xa 4*) showed high level of susceptibility against all the 52 isolates, while rest of the 13 host genotypes exhibited differential reactions. The pathogen isolates *CRXoo*-26, 28, 31, 38 and 47 were highly aggressive on all the host genotypes, while rest of the 47 isolates showed differential pathogenicity. The pathogenicity of 52 isolates are significantly different from each other and the host genotypes also represented an array of diverse conditions for disease developments as evidenced from highly significant isolate (I) and host genotype (G) mean squares (Table 2). The pooled analysis of variance (ANOVA) showed that the I×G interaction was a linear function of the additive environmental component. The I×G interaction 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 IG interaction. The I×G linear interaction was highly significant when tested against pooled deviation indicated the presence of genetic differences among the pathogen strains for their regression on the environmental index. The pooled deviation was significantly larger than pooled error, indicating the existence of a significant departure from linearity and therefore some of the IG interaction cannot be predicted from the linear regressions.

Finlay and Wilkinson (1963) considered linearity of regression (b_i) as a measure of stability. Eberhart and Russell (1966) emphasized that both linear (b_i) and non-linear (S^2_d) components of genotype x environment interaction should be considered in judging the stability. Samuel et al. (1970) suggested that linear regression could simply be regarded as a measure of response of a particular genotype, whereas the deviation from regression (S^2_d) was considered as a measure of stability i.e. genotypes with lowest or nonsignificant S^2_d being most stable and *vice versa*. In the present study on host-pathogen interaction, a pathogen strain possessing mean pathogenicity level greater than population mean, unit regression coefficient (b_i=1.0) and minimum deviation from regression (S^2_d=0) was considered as stable isolate and b_i>1.0, S^2_d>0 as most unstable isolate.

The pathogen isolate (I) x host genotype (G) interaction for 52 isolates presented in Table 3 revealed that 25 isolates *viz.*, *CRXoo*-2, 11, 12, 13, 14, 15, 16, 22, 23, 25, 26, 28, 29, 31, 36, 37, 38, 39, 40, 44, 45, 46, 47, 48 and 52 showed large interactions with the host genotypes. Rest of the 27 isolates showed very small interaction. Out of these 25 interactive group, *CRXoo*-11, 12, 13, 14, 15, 26, 28, 29, 31, 36, 38, 39, 45, 47 and 48 showed a linear trend with b_i=1.0. These are therefore unresponsive in their pathogenicity to changes in host genotypes but not very stable since deviation from regression (S^2_d) are high. *CRXoo*-9 showed b_i= 1.0, S^2_d=0 but low contribution towards I×G SS as well as

Table 1. Pathogenicity (lesion length in cm) of 52 isolates of *Xanthomonas oryzae* pv. *oryzae* on 16 host genotypes

Pathogen isolates	Host genotypes																Mean
	*1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	
CRXoo-1	4.07	14.02	4.22	2.95	14.00	13.60	2.70	2.90	2.80	3.97	4.22	2.75	3.80	3.97	2.87	2.95	5.36
CRXoo-2	4.02	17.42	3.55	7.07	17.50	17.50	2.97	7.00	2.97	4.30	4.20	2.70	4.35	3.97	2.95	2.97	6.59
CRXoo-3	3.95	14.00	3.15	2.97	14.00	13.80	2.77	3.02	2.47	4.00	4.17	2.65	3.60	4.15	2.72	2.57	5.25
CRXoo-4	4.15	14.10	3.65	3.00	13.90	13.90	2.40	2.90	2.92	4.30	4.07	2.40	3.25	4.15	2.25	2.37	5.23
CRXoo-5	4.07	13.97	4.02	3.10	13.90	13.70	2.32	2.52	2.32	3.97	4.00	2.35	3.92	4.15	2.82	2.45	5.23
CRXoo-6	3.97	13.70	4.25	3.00	13.80	13.80	2.30	2.70	2.67	3.60	4.05	2.80	4.00	4.10	2.75	2.40	5.24
CRXoo-7	3.90	13.97	4.02	2.92	13.90	13.35	2.37	2.90	2.75	4.20	4.00	2.40	4.20	4.10	2.27	2.22	5.22
CRXoo-8	3.82	13.97	4.27	2.80	13.90	13.80	2.62	2.75	2.60	4.17	3.92	2.80	4.32	3.72	2.67	2.32	5.28
CRXoo-9	3.27	14.75	3.70	2.92	14.00	13.90	2.70	3.07	2.30	3.90	3.92	2.40	4.02	3.12	2.77	2.20	5.19
CRXoo-10	3.75	13.80	3.75	3.00	13.90	14.00	2.27	2.65	2.60	4.00	3.97	2.57	3.82	3.62	2.65	2.40	5.18
CRXoo-11	3.82	17.02	4.12	6.70	17.50	16.72	2.77	7..07	2.87	4.07	3.85	2.77	3.92	3.62	2.90	2.77	6.41
CRXoo-12	3.95	16.60	4.15	6.80	17.37	17.00	2.85	7.00	2.95	4.05	3.97	2.70	3.95	3.75	2.72	2.27	6.38
CRXoo-13	3.85	16.70	4.00	7.00	17.20	16.90	2.70	7.10	2.82	4.00	3.95	2.82	3.87	3.87	2.62	2.47	6.37
CRXoo-14	4.05	16.92	4.15	7.10	16.80	17.20	2.75	7.22	2.65	3.90	3.95	2.97	4.00	4.00	2.25	2.77	6.42
CRXoo-15	3.95	16.70	3.50	7.00	16.87	17.20	2.57	6.90	2.70	3.85	4.02	2.77	4.07	3.97	2.72	2.55	6.34
CRXoo-16	8.40	14.87	8.22	2.80	15.00	14.87	7.30	3.40	7.52	8.25	8.30	7.05	8.50	8.65	7.47	7.00	8.60
CRXoo-17	3.72	14.10	4.12	2.92	14.00	13.60	2.30	2.62	2.70	4.00	4.12	2.25	4.30	4.27	2.67	2.40	5.26
CRXoo-18	3.90	14.00	4.05	3.10	13.90	13.95	2.40	2.90	2.60	3.95	4.00	2.30	4.30	4.30	2.55	2.40	5.29
CRXoo-19	3.90	13.80	4.05	3.00	13.90	13.70	2.10	3.00	2.30	3.77	4.00	2.42	4.00	4.45	2.50	2.20	5.19
CRXoo-20	3.72	13.90	4.15	2.92	14.00	13.90	2.62	3.05	2.70	3.92	4.00	2.30	4.00	4.07	2.30	2.15	5.23
CRXoo-21	4.00	14.12	4.27	2.95	13.80	13.70	2.70	2.82	2.75	4.00	3.95	2.57	3.97	3.92	2.40	2.42	5.27
CRXoo-22	4.22	17.80	4.40	7.40	18.50	18.40	2.50	7.40	2.80	4.05	4.60	2.67	4.67	4.30	2.50	2.37	6.79
CRXoo-23	8.40	15.37	8.22	3.40	15.02	14.60	7.50	3.25	7.52	8.05	8.20	7.20	8.25	8.45	7.47	6.97	8.62
CRXoo-24	4.00	14.37	4.00	2.82	13.80	13.00	2.37	2.87	2.95	3.70	3.75	2.60	3.95	3.30	2.60	2.95	5.19
CRXoo-25	4.47	18.40	3.95	7.30	18.50	18.30	2.70	7.35	2.70	4.20	4.40	2.50	4.37	4.15	2.60	2.42	6.77
CRXoo-26	12.27	21.40	12.00	8.40	21.00	21.30	8.60	8.40	8.51	12.00	4.40	8.20	12.05	12.27	8.40	7.97	11.70
CRXoo-27	3.95	13.80	3.70	2.70	14.00	13.70	2.30	2.92	2.82	3.92	4.32	2.30	3.82	4.10	2.40	2.97	5.23
CRXoo-28	12.40	21.77	12.22	8.50	21.50	21.65	8.45	8.22	8.27	12.00	4.40	8.50	12.32	12.00	8.30	8.00	11.78
CRXoo-29	7.72	16.77	7.17	3.87	17.25	16.90	2.57	3.80	2.95	7.50	7.90	2.90	7.35	7.30	2.90	2.60	7.34
CRXoo-30	395	14.00	3.80	2.90	14.00	13.70	2.32	2.80	2.70	3.90	3.87	2.37	4.07	4.15	2.60	2.45	5.23
CRXoo-31	12.10	21.85	12.02	8.07	21.60	21.90	8.35	8.30	8.30	12.00	12.02	8.52	12.00	12.40	8.40	7.90	12.23
CRXoo-32	3.55	13.80	3.95	2.62	13.90	13.70	2.42	3.10	2.60	4..00	4.12	2.85	4.00	4.20	2.40	2.30	5.22
CRXoo-33	3.92	13.97	4.02	2.77	14.10	13.90	2.22	3.10	2.70	3.70	4.00	2.60	3.70	4.20	2.60	2.60	5.26
CRXoo-34	4.15	14.00	4..07	3.12	14.10	14.00	2.40	3.00	2.50	3.97	3.97	3.02	3.90	3.97	2.30	2.60	5.32
CRXoo-35	4.00	13.90	4.05	2.67	14.00	13.80	2.42	2.67	2.55	3.95	4.05	2.50	3.92	3.90	2.55	2.27	5.20
CRXoo-36	7.50	16.60	7..72	4.20	16.80	16.60	3.00	4.20	3.00	7.87	7.95	2.75	7.82	7.52	3.02	2.35	7.43
CRXoo-37	4.30	18.52	4.45	7.50	18.50	18.40	2.77	7.50	2.90	4.02	4.00	2.42	4.22	4.07	2.82	2.25	6.79
CRXoo-38	12.00	22.37	12.15	8.10	21.60	21.80	8.20	8.30	8.30	12.02	12.30	8.50	12.20	12.00	8.40	8.07	12.27
CRXoo-39	7.45	16.90	5.57	3.90	17.37	16.80	2.85	3.80	2.90	7.65	7.17	2.80	7.87	7.47	3.00	2.32	7.37
CRXoo-40	4.30	18.20	4.55	7.50	18.30	17.92	2.82	7.40	2.37	4.45	4.37	2.35	4.30	4.40	2.70	2.15	6.76
CRXoo-41	4.30	13.70	3.82	2.70	14.37	13.80	2.85	3.10	2.45	4.00	4.07	2.70	3.92	4.00	2.50	2.22	5.28
CRXoo-42	4.00	13.80	3.62	3.15	14.05	13.90	2.82	3.00	2.62	3.90	4.00	2.70	3.85	4.07	2.75	2.10	5.27
CRXoo-43	3.95	13.90	4.05	3.00	14.05	13.60	2.27	2.60	2.60	4.07	4.00	2.45	3.80	3.80	2.70	2.27	5.19
CRXoo-44	4.60	18.27	4.10	7.40	18.20	18.40	2.77	7.30	2.37	4.22	4.30	2.55	4.27	4.05	2.70	2.22	6.73
CRXoo-45	7.70	16.92	7.15	4.00	16.90	16.80	3.00	4.00	2.90	7.87	7.80	2.85	7.92	7.60	2.90	2.60	7.43
CRXoo-46	8.30	14.90	7.95	3.40	15.00	14.70	7.52	3.25	7.42	8.07	8.60	7.50	8.17	8.15	7.52	7.07	8.60
CRXoo-47	12.27	22.00	12.00	8.30	21.40	21.60	8.22	8.50	8.30	12.10	12.40	8.47	12.25	12.40	8.55	8.45	12.33
CRXoo-48	7.50	17.05	7.42	4.00	16.90	16.90	2.75	4.00	2.87	7.80	7.70	2.70	7.67	7.60	2.80	2.50	7.39
CRXoo-49	3.85	13.90	3.92	3.10	14.10	13.40	2.67	2.80	2.77	3.80	3.72	2.60	4.20	3.77	2.47	2.60	5.23
CRXoo-50	3.90	14.00	4.05	3.02	13.97	13.70	2.85	2.90	2.80	3.95	3.90	2.65	3.60	3.90	2.37	2.60	5.26
CRXoo-51	4.00	13.60	3.92	2.92	14.10	13.90	2.77	3.00	2.70	3.90	3.92	2.70	3.62	3.95	2.37	2.27	5.23
CRXoo-52	8.15	14.70	8.05	3.40	14.90	15.07	7.52	3.25	7.45	8.00	8.40	7.67	8.07	8.37	7.35	7.00	8.59
Mean	5.45	15.83	5.41	4.47	15.86	15.70	3.53	4.46	3.61	5.44	5.22	3.54	5.47	5.46	3.56	3.34	6.65

*These numerals refer to the host genotypes listed in Table 7.

Table 2. Analysis of variance for pathogenicity of 52 isolates (I) of *Xanthomonas oryzae* pv. *oryzae* across 16 host genotypes (G)

Source of variation	d.f.	S.S.	M.S.	F Ratio
Replication within Environment	48	2.96	0.06	0.06
Pathogen isolates (I)	51	3500.54	68.64	66.43[a]
Host genotypes (G)	15	16592.81	1106.19	1070.53[a]
G+(I×G)	780	17663.81	22.65	21.92[a]
(I×G)	765	1071.00	1.40	1.36[a]
G (linear)	1	16592.81	16592.81	16057.98[a]
I×G (linear)	51	318.76	6.25	6.05[a]
Pooled Deviation	728	752.25	1.03	56.38[a]
Pooled Error	2448	44.87	0.02	
Total	831	21164.36	25.47	

[a]Significant at P=0.01 level.

MS-REG. The remaining isolates in the interactive group do not have b_i=1.0 maintained high deviations MS and hence they are not responsive and unstable. The rest of the 27 non-interacting group of isolates showed slopes significantly different from 1.0, maintained small or large deviations MS (MS-DEV) as well as small or high contributions to interaction MS. Thus no useful conclusions about their responsiveness and stability could be made. On the contrary, Nayak and Chakrabarti (1985) identified 6 isolates of *Xoo* possessing average stability and 7 isolates possessing above average stable pathogenicity against 10 host genotypes.

The present model of I×G interactions has involved the estimation of stability parameters related to the regression of the isolate means over 16 host genotypes on an index of tested host genotypes, which is equivalent to the environmental index. The index is the overall mean pathogenicity of all isolates on each host genotype. Both the slope of these regressions and the deviations from regression are interpreted as stability parameters. Westcott (1985) raised the question as to whether such interactions can be summarized by simple regressions on site index. The classical test for constant regression slopes may be significant, but if the regressions account for a very small part of the interaction SS compared to the estimated structural component, then the analysis can at best be a partial description of the situation. Hence, more flexible and informative model like the Additive Main effects and Multiplicative Interaction (AMMI) that can account for greater amount of structural variability, would be more useful for a better understanding of the interactions.

AMMI analysis of variance. The AMMI analysis of variance of 52 isolates of *Xoo* tested for their pathogenicity on 16 host genotypes showed that 78.4% of the total sum of squares (SS) was attributable to the host genotypes (G), 16.5% to the isolates (I) and only 5.06% to I×G interaction effects (Table 4). A large sum of squares for G indicated that the host genotypes were diverse with large differences among the means causing most of the variations in pathogenicity of the isolates of the pathogen population. The magnitude of I×G SS was 3.3 times smaller than that for the isolates, indicating that the differences in the response of the isolates across the host genotypes were not that substantial.

The results from AMMI analysis also revealed that the first interaction principal component axis (IPCA-1) accounted for 66.83% of the interaction SS in 8.5% of the interaction degrees of freedom. Similarly, the second IPCA (IPCA-2) explained a further 21.5% of the interaction SS. The MS for both IPCA-1 and IPCA-2 were significant at P =0.01 level and cumulatively contributed to 88.33% 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 128 degrees of freedom. The prediction assessment indicated that AMMI with only two IPCAs was the best predictive model with 128 d.f. Further IPCAs 3 to 7 captured mostly noise since the MS were not significant, explained only 9.22% to 0.23% of the total SS and therefore did not help to predict validation observations. Thus the interaction of the 52 isolates with 16 host genotypes was best predictable by the first two principal components. The most accurate model for AMMI can be predicted by using the first two PCAs (Gauch and Zobel, 1996; Yan et al., 2000). Schneider and Van der Boogert. (1999) on the contrary reported that four AMMI axes seemed necessary for an adequate description of the interactions between *Rhizoctonia solani* isolates and Tulip cultivars. These results indicate that the number of the terms to be included in an AMMI model cannot be specified a priori without first trying AMMI predictive assessment. The factors like the type of the crop, the diversity in the germplasm i.e. the host as well as pathogen population and the range of environmental conditions will

Table 3. The origin, mean lesion length (cm), the slope of the regression (B_i), deviation from regression (S^2_d) and the contribution of the 52 isolates of *Xanthomonas oryzae* pv. *oryzae* to the interactions

Isolates	Origin	Mean lesion length (cm)	Slope (B_i)[a]	MS-DEV (S^2_d)[b]	MS-I×G[c]	MS-REG[d]
CRXoo-1	Orissa (OR)	5.36	0.921[e]	0.11	0.24	1.98
CRXoo-2	Orissa	6.59	1.169[e]	1.84	2.33	9.15
CRXoo-3	Orissa	5.25	0.939[e]	0.15	0.22	1.20
CRXoo-4	Orissa	5.23	0.949[e]	0.11	0.16	0.84
CRXoo-5	Orissa	5.23	0.940[e]	0.06	0.14	1.16
CRXoo-6	Andhra Pradesh (AP)	5.24	0.926[e]	0.08	0.19	1.76
CRXoo-7	Andhra Pradesh	5.22	0.930[e]	0.04	0.14	1.56
CRXoo-8	Andhra Pradesh	5.28	0.936[e]	0.08	0.17	1.33
CRXoo-9	Tamil Nadu (TN)	5.19	0.975	0.19	0.19	0.19
CRXoo-10	Andaman & Nicobar Islands (AN)	5.18	0.945[e]	0.08	0.14	0.97
CRXoo-11	Punjab (PB)	6.41	1.146	1.82	2.16	6.84
CRXoo-12	Punjab	6.38	1.143	1.77	2.09	6.51
CRXoo-13	Punjab	6.37	1.137	1.92	2.19	6.00
CRXoo-14	Punjab	6.42	1.137	1.98	2.25	6.02
CRXoo-15	Punjab	6.34	1.139	1.88	2.17	6.21
CRXoo-16	Punjab	8.60	0.689[e]	2.73	4.60	30.80
CRXoo-17	Punjab	5.26	0.942[e]	0.07	0.14	1.08
CRXoo-18	Orissa	5.29	0.944[e]	0.03	0.09	1.00
CRXoo-19	Orissa	5.19	0.939[e]	0.03	0.11	1.17
CRXoo-20	Orissa	5.23	0.947[e]	0.04	0.10	0.90
CRXoo-21	Orissa	5.27	0.935[e]	0.06	0.14	1.36
CRXoo-22	Orissa	6.79	1.239[e]	2.01	3.09	18.29
CRXoo-23	Orissa	8.62	0.693[e]	2.46	4.30	30.02
CRXoo-24	Orissa	5.19	0.922[e]	0.21	0.33	1.96
CRXoo-25	Andhra Pradesh	6.77	1.257[e]	2.00	3.27	21.06
CRXoo-26	Andhra Pradesh	11.70	1.055	3.58	3.41	0.98
CRXoo-27	Andhra Pradesh	5.23	0.933[e]	0.12	0.21	1.42
CRXoo-28	West Bengal (WB)	11.78	1.091	3.66	3.59	2.66
CRXoo-29	Madhya Pradesh (MP)	7.34	1.097	1.69	1.78	2.99
CRXoo-30	Madhya Pradesh	5.22	0.943[e]	0.05	0.11	1.04
CRXoo-31	Bihar (BR)	12.23	1.074	1.18	1.22	1.74
CRXoo-32	Uttar Pradesh (UP)	5.22	0.932[e]	0.08	0.17	1.46
CRXoo-33	Gujarat (GT)	5.26	0.947[e]	0.08	0.13	0.89
CRXoo-34	Gujarat	5.32	0.946[e]	0.06	0.12	0.92
CRXoo-35	Gujarat	5.20	0.946[e]	0.06	0.12	0.93
CRXoo-36	Gujarat	7.43	1.058	1.91	1.85	1.08
CRXoo-37	Maharashtra (MH)	6.79	1.261[e]	2.23	3.53	21.68
CRXoo-38	Orissa	12.27	1.085	1.19	1.27	2.31
CRXoo-39	Andhra Pradesh	7.37	1.101	1.71	1.81	3.27
CRXoo-40	Assam (AS)	6.76	1.235[e]	2.03	3.07	17.63
CRXoo-41	Andhra Pradesh	5.28	0.943[e]	0.08	0.15	1.04
CRXoo-42	Andhra Pradesh	5.27	0.937[e]	0.08	0.16	1.27
CRXoo-43	Rajasthan (RJ)	5.20	0.941[e]	0.06	0.13	1.11
CRXoo-44	West Bengal	6.73	1.250[e]	2.04	3.24	19.97
CRXoo-45	Andhra Pradesh	7.43	1.079	1.82	1.83	1.98
CRXoo-46	Orissa	8.60	0.679[e]	2.49	4.52	32.89
CRXoo-47	Orissa	12.33	1.052	1.21	1.19	0.86
CRXoo-48	Andhra Pradesh	7.39	1.093	1.82	1.88	2.74
CRXoo-49	Orissa	5.23	0.928[e]	0.09	0.19	1.65
CRXoo-50	Orissa	5.26	0.934[e]	0.09	0.18	1.39
CRXoo-51	Orissa	5.23	0.936[e]	0.08	0.16	1.30
CRXoo-52	Orissa	8.59	0.682[e]	2.47	4.45	32.20

[a]Slope, Slopes of regressions of isolate means on environmental index (*i.e.* genotypes)
[b]MS-DEV, Deviation from regression component of interaction (S^2_d)
[c]MS-I×G, Contribution of each isolate to interaction MS
[d]MS-REG, Contribution of each isolate to the regression component of the I×G interaction
[e]Indicates slopes significantly different from 1.00 at $P=0.05$ level

Table 4. Additive Main effects and Multiplicative Interaction (AMMI) analysis of variance for pathogenicity of 52 isolates of *Xanthomonas oryzae* pv. *oryzae* across 16 host genotypes

Source of variation	d.f.	S.S.	M.S.	Explained (%)
Total	831	21164.37	25.47[a]	
Pathogen Isolates (I)	51	3500.55	68.64[a]	16.54
Host genotypes (G)	15	16592.81	1106.19[a]	78.40
I x G interaction	765	1071.01	1.40[a]	5.06
AMMI IPCA-1	65	715.74	11.01[a]	66.83
AMMI IPCA-2	63	230.25	3.65[a]	21.50
AMMI IPCA-3	61	98.75	1.62	9.22
AMMI IPCA-4	59	6.94	0.12	0.65
AMMI IPCA-5	57	4.25	0.07	0.40
AMMI IPCA-6	55	3.01	0.05	0.28
AMMI IPCA-7	53	2.48	0.05	0.23
Residual	352	9.59	0.03	0.90
Pooled residual	765	1071.01	1.40	100.00

[a]Significant at P=0.01 level.

affect the degree of complexity of the best predictive model (Crossa et al., 1990).

AMMI-1 biplot display. The AMMI analysis provides a graphical representation wherein it shows the main effect means on the abscissa and IPCA-1 scores of both host genotypes and pathogen isolates simultaneously on the ordinate. The interaction is described in terms of differential sensitivities of the isolates 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. Pathogen isolates (or host genotypes) that appear almost on a perpendicular line have similar means and those fall almost on a horizontal line have similar interaction patterns. Isolates with IPCA-1 scores close to zero have small interactions and hence show wider adaptation to the tested host genotypes. A large pathogen genotypic IPCA-1 score (either positive or negative) have high interactions and reflects more specific adaptation to the host genotypes with IPCA-1 values of the same sign.

The scores and main effects can be read from the graph and used to predict the expected level of pathogenicity for any isolate and host genotype combination. For any I-G combination in the AMMI biplot (Fig. 1), the additive part (main effects) of the AMMI model equals the I mean plus G mean minus the grand mean. The multiplicative part (interaction effects) is the product of I and G IPCA-1 scores (Zobel et al., 1988). For example, *CRXoo*-52 with the host genotype-1 had a main effect of 8.59 cm+5.45 cm−6.65 cm=7.39 cm (Table 1). The interaction effects of *CRXoo* 52 and host genotype-1 would be the products of the respective IPCA-1 scores (Tables 6 and 7)=1.52×0.91=1.37. The AMMI model estimated the pathogenicity of *CRXoo* 52 on the host genotype-1 as 7.39+1.37=8.74 cm, which fits the

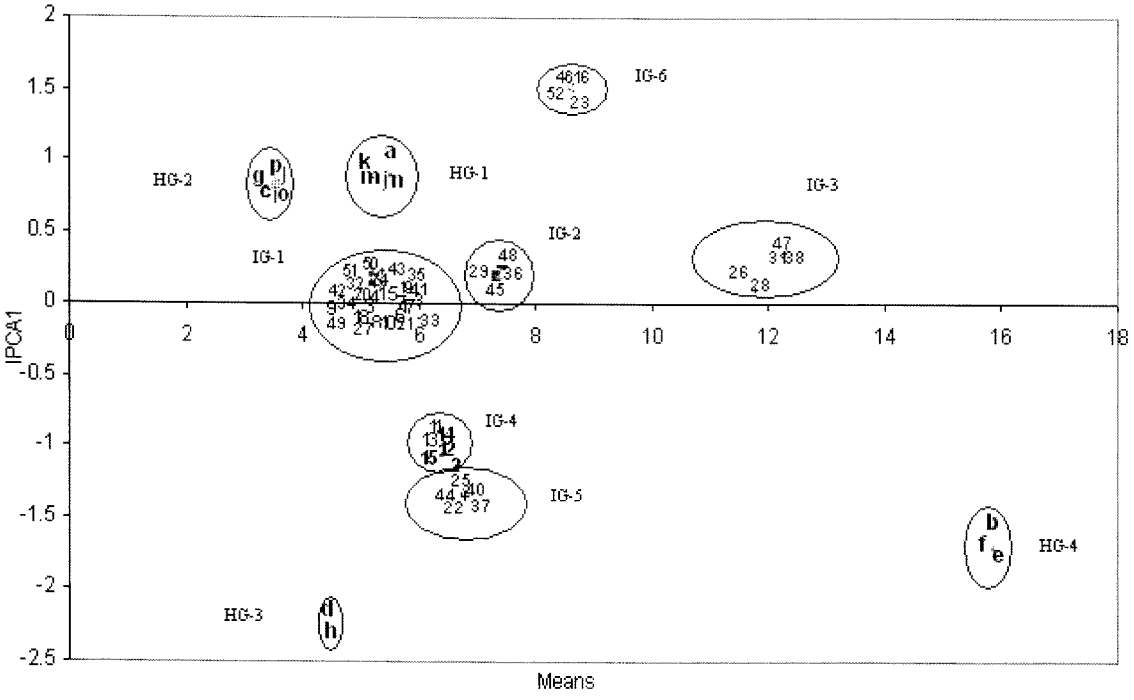

Fig. 1. Biplot of the mean lesion length and the first interaction principal component axis scores of 52 isolates of *Xanthomonas oryzae* pv. *oryzae* and 16 host genotypes. IG, Isolate groups; HG, Host genotype groups.

observed pathogenicity level of 8.15 cm (Table 1). Pathogen isolates and host genotypes 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.

Six groupings of the isolates (IG) are evident from the biplot generated from the present study (Fig. 1):

IG-1 includes 27 pathogen isolates *viz. CRXoo*-1, 3, 4, 5, 6, 7, 8, 9, 10, 17, 18, 19, 20, 21, 24, 27, 30, 32, 33, 34, 35, 41, 42, 43, 49, 50 and 51 with a mean pathogenicity level (5.24 cm) less than the grand mean (6.65 cm) and small IPCA-1 scores ranging from 0.03 to 0.25. For these isolates, the AMMI-1 model predicts pathogenicity levels that are close to those of the AMMI-0 model on host genotype groups (HG)-1 and 2 with positive IPCA-1 scores ranging from 0.78 to 1.01 and highest pathogencity levels on HG-4 with high negative IPCA-1 scores. They are a heterogeneous group in so far as their origin and distribution is concerned, since these are widespread over nine states of India *viz.* AP, AN, GT, MP, OR, RJ, PB, TN and UP. They possess 4 virulence factors operative against the corresponding *Xa* genes for resistance namely *Xa 1, 2, 4* and *11* (Nayak et al., 2008b) and belong to the least virulent pathotype-16 (Nayak et al., 2008a) and this group of isolates have smallest interactions and hence are most stable isolates possessing low level of pathogenicity.

IG-2 consists of 5 isolates *CRXoo*-29, 36, 39, 45 and 48 with a mean pathogenicity level (7.39 cm) just above the grand mean and small positive IPCA-1 scores ranging from 0.19 to 0.25. They are well adapted to the host genotypes in HG-1 and HG-2 with small positive IPCA-1 scores. They possess 8 virulence factors operative against the corresponding *Xa* genes *viz. Xa 1, 2, 3, 4, 5, 11, 12* and *13* and belong to pathotype-7. They are distributed over 3 states of India namely AP, GT and MP. These isolates have small interactions and hence are relatively stable isolates possessing moderate degree of pathogenicity.

IG-3 includes 5 isolates *viz.*, *CRXoo*-26, 28, 31, 38 and 47 with the highest mean pathogenicity level (12.16 cm) and small positive IPCA-1 scores ranging from 0.11 to 0.37. This group of isolates are well adapted to the host genotypes HG-1 and HG-2 with positive IPCA-1 scores as well as those in IG-3 and IG-4 with high negative IPCA-1 scores. They possess a maximum number of 11 virulence factors operative against the corresponding *Xa* genes *Xa-1, 2, 3, 4, 5, 6, 7, 10, 11, 12* and *13* and belong to the most virulent pathotype-1. They are distributed over the states of AP, BR, OR and WB. These isolates have small interactions and hence are most stable virulent strains among the pathogen population tested.

IG-4 consists of 6 isolates *viz. CRXoo*-2, 11, 12, 13, 14 and 15 with a mean pathogenicity level (6.42 cm) just below the grand mean. They show almost similar large negative IPCA-1 scores ranging from –1.05 to –1.11. These isolates showed specific adaptation to the host genotypes in HG-3 and HG-4 with similar large negative IPCA-1 scores. They possess 7 virulence factors operative against the *Xa* genes *Xa-1, 2, 3, 4, 10, 11* and *12* and belong to the pathotype 14. They are distributed over the states of OR and PB.

IG-5 includes 5 isolates *viz. CRXoo*-22, 25, 37, 40 and 44 with a mean pathogenicity level (6.77 cm) near the grand mean and large negative IPCA-1 scores ranging from –1.30 to –1.40. These isolates show more specific adaptation to the host genotypes with similar large negative IPCA-1 values under the HG-3 and HG-4 and are distributed over the states of AP, AS, MH, OR and WB. These isolates have been reported to possess 7 v-factors similar to those in IG-4 and belong to the pathotype-15.

The mean level of virulence of the isolate groups in ascending order was IG-1, IG-4, IG-5, IG-2, IG-6 and IG-3 possessing 4, 7, 7, 8, 10 and 11 virulence factors (Nayak et al., 2008b) and belonging to pathotypes- 16, 15, 14, 7, 4 and 1, respectively (Nayak et al., 2008a). Although the isolates belonging to IG-4 and IG-5 contained the same v-factors, they were classified into two different pathotypes-14 and 15, respectively based on their virulence patterns on five Indian differential varieties (Nayak et al., 2008a).

IG-6 consists of 4 isolates *CRXoo*-16, 23, 46 and 52 with a mean pathogenicity level (8.60 cm) above the grand mean. They show large positive IPCA-1 scores ranging from 1.50 to 1.57. This group of isolates showing large interactions, show more specific adaptation to the host genotypes in HG-1 and HG-2, which also show similar high positive IPCA-1 scores. These isolates have been reported to possess 10 v-factors operative against the corresponding *Xa* genes *Xa-1, 2, 3, 4, 5, 6, 7, 11, 12* and *13*. They belong to the virulent pathotype-4 and are distributed over the states of Orissa and Punjab.

The differences among isolates in terms of direction and magnitude along the abscissa (lesion length) and ordinate (IPCA-1 scores) are also important. The best virulent/avirulent isolate should show high/low level of pathogenicity and should be stable across the tested host genotypes. An isolate showing lower absolute IPCA-1 score would produce a lower absolute IxG interaction effect than that with higher absolute IPCA-1 score and have less variable pathogenicity (more stable) across the host genotypes. The isolate stability ranking based on lower absolute IPCA-1 scores was IG-1 (0.03 to 0.23), IG-3 (0.11 to 0.37), IG-2 (0.19 to 0.25), IG-4 (1.05 to 1.11), IG-5 (1.11 to 1.39) and IG-6 (1.50 to 1.57). The first three groups of isolates are shown on the horizontal axis in Fig. 1 with low level of pathogenicity (5.82 cm) for IG-1, moderate level of patho-

genicity (8.04 cm) for IG-2 and high level of pathogenicity (12.62 cm) for IG-3. Hence, these isolate groups were identified as possessing stable pathogenicity. The last three groups of isolates showed higher absolute IPCA-1 scores of 1.05 to 1.11 for IG-4, 1.11 to 1.39 for IG-5 and 1.50 to 1.57 for IG-6 and this produced higher absolute interaction effects. Thus these isolates have more variable pathogenicity across the tested host genotypes.

The 16 host genotypes show variability in both the main effects and interactions, the IPCA-1 scores showing clear higher negative or positive interactions (Fig. 1) due to the 4 groups of host genotypes (HG). Among them, HG-4 consisting of Rantai Emas (*Xa 1+Xa 2*), IR-8 (Xa *11*) and IR-20 (*Xa-4*), showed highest main effects and large negative IPCA-1 scores. The HG-3 consisting of two host genotypes namely Java-14 (*Xa 1+Xa 3+Xa 12*) and Cas 209 (*Xa 10*) showed low response to the pathogen isolates and large negative IPCA-1 scores. The HG-1 consisting of 5 host genotypes Kogyoku *Xa 1+Xa 3+Xa 12*), TKM-6 (*Xa 4*), Tetep (*Xa 1+Xa 2*), CB-II (*Xa 3+xa 5+xa 13*) and BJ-1 (*xa 5+xa 13*) as well as HG-2 consisting of 6 host genotypes *viz.* Wase Aikoku-3 (*Xa* 3), IR 1545-339 (xa *5*), *DV-85* (*xa 5+Xa 7*), Semora Mangga (*Xa 4*), Zenith (*Xa 6*) and M. Sung Song *Xa 6*) showed low response to the isolates with positive interaction IPCA-1 scores.

Pathogenicity of six isolate groups on four host genotype groups. The virulence of six pathogen isolates groups (IG) averaged over four host genotype groups (HG) ranged from 5.8 cm for IG-1 to 12.6 cm for IG-3 (Table 5). On the other hand, the mean response of the four groups of HG averaged over all six IG, ranged between 4.8 cm for HG-2 and 17.1 cm for HG-4, which contain highly susceptible host genotypes against all the IGs. HG-1 showed high response to the IG-2, IG-3 and IG-6, while HG-2 was high responsive to IG-3 and IG-6 and low responsive to IG-1, IG-2, IG-4 and IG-5. Similarly, HG-3 showed high response to the IG-3, IG-4 and IG-5 and low response to IG-1, IG-2 and IG-6. Thus IG-3 constituted of most virulent isolates capable of knocking down all the four HGs, followed by IG-6 on HG-1, HG-2 and HG-4 and IG-5 on HG-3 and HG-4. The IG-2 was virulent on HG-1 and HG-4, followed by IG-4 on HG-3 and HG-4. The IG-1 was virulent on HG-4 only. Thus a differential interaction was evident from the response between HG and IG.

Some of the interactions between the host genotypes and pathogen isolates need special mention. The host genotypes Rantai Emas and Tetep possess the same genes i.e. *Xa 1* and *Xa 2*. The former was completely knocked down by all the 52 pathogen strains, while the later exhibited resistance to 40 strains. This might be due to the presence of some unknown genes in Tetep, imparting resistance to specific

Table 5. Mean response (lesion length in cm) of four rice host-genotype groups (HG) to six *Xanthomonas oryzae* pv. *oryzae* isolate groups (IG)

HG	IG						Mean
	1	2	3	4	5	6	
1 (1, 10, 11, 13, 14)[a]	3.96	7.65	11.55	3.97	4.29	8.29	6.62
2 (3, 7, 9, 12, 15, 16)	2.74	3.62	8.95	2.94	2.85	7.49	4.77
3 (4, 8)	2.85	3.98	8.32	7.00	7.41	3.27	5.47
4 (2, 5, 6)	13.72	16.90	21.65	17.07	18.31	14.92	17.10
Mean	5.82	8.04	12.62	7.75	8.22	8.49	

[a]Numerals in parentheses refer to the host genotype numbers listed in Table 7

isolates of the pathogen, which needs further genetic analysis. Similarly, the bacterial strains in IG-1 possessing four v-factors against *Xa 1, 2, 4, 11* and belonging to pathotype-16, behaved differently. The host genotypes Tetep possessing *Xa 1* and *Xa 2* genes and TKM-6 possessing *Xa 4* gene, exhibited resistance to IG-1 group of strains, indicating the existence of some unknown genes yet to be identified. Similar explanation could be offered for the differential reactions between TKM-6, Simora Mangga, and IR-20 each possessing *Xa 4* gene, the first two exhibiting resistant reaction to most of the isolates while the last one exhibiting highly susceptible reaction to all the isolates. This is possibly the reason why these three host genotypes were distributes in three HG groups *viz.* HG-1, HG-2 and HG-4, respectively.

The AMMI-2 biplot display. The AMMI is an explorative technique by which the host-pathogen relationship can be expressed in terms of interaction patterns derived in biplots. A biplot is a graphical representation in which isolates and host genotypes are displayed simultaneously. The interaction is described in terms of differential sensitivities of the isolates to the most discriminating environmental variables (AMMI-axes) that can be constructed. These hypothetical environmental variables and the isolate sensitivities are estimated from the table itself (Schneider and Van den Boogert, 1999). For simple interpretation of the biplot, the isolates with vector end points far from the origin contribute relatively more to the interaction than those with vector end points close to the origin. Accordingly, the IG-2, 3, 4, 5 and 6 have a greater contribution to the interaction than the IG-1 (Fig. 2). Isolates with vector end points far apart show considerable interactions like the group of isolates under IG-6 and IG-4, 5 as well as IG-2, 3. Isolates for which the directions of the vectors almost coincide, have similar pattern of interactions like the isolates within each group of IG-1 to IG-6. When the directions are opposite, the interaction patterns of the corresponding isolates show negative correlation like those within IG-6 and IG-4,

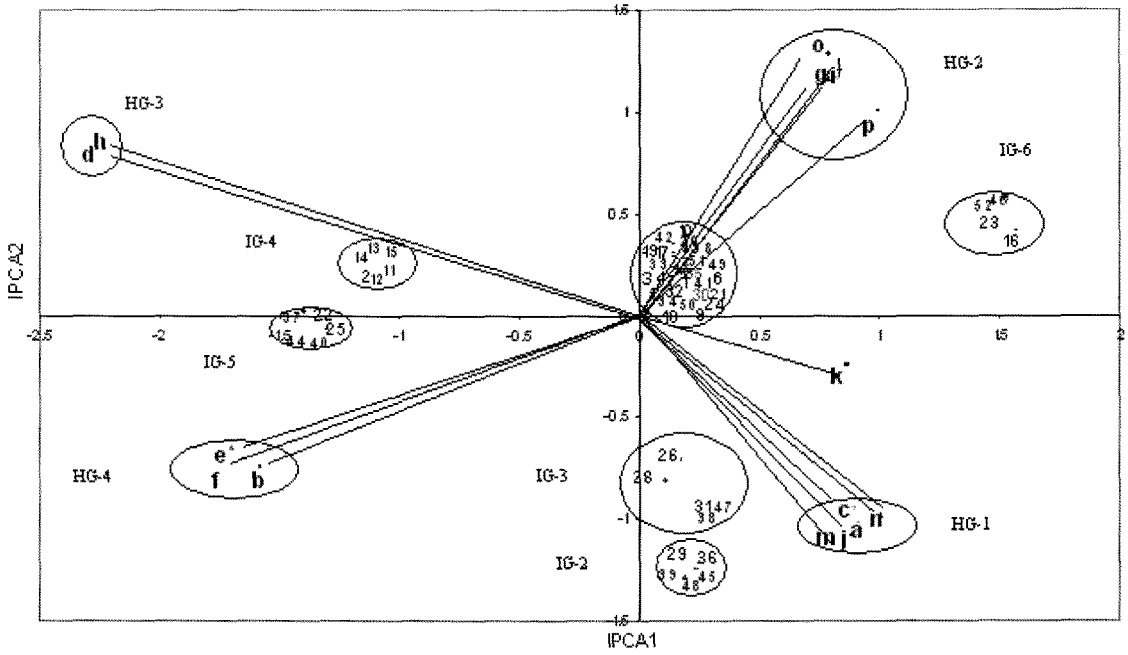

Fig. 2. Biplot of 52 isolates of *Xanthomonas oryzae* pv. *oryzae* and 16 host genotypes for their response to rice bacterial blight using respective IPCA-1 and IPCA-2 scores. IG=Isolate groups, HG=Host genotype groups.

5 as well as IG-1 and IG-2, 3. Isolates and host genotypes showing considerable interactions could be easily identified from the biplot by looking for pairs of isolate vectors and pairs of host genotype vectors for which the length of difference vectors are large and for which the respective vectors by looking at their differences in vector length or for which the difference vectors are almost parallel. The conclusions from the biplot are most reliable for isolates and host genotypes with their vector end points far apart (high non-additivity) like IG-6; IG-2 and IG-3; IG-4 and IG-5; and HG-1, HG-2, HG-3 and HG-4. Isolates or host genotypes close to the origin like IG-1, contribute little to the interaction and can not be discriminated from bias.

In the present study, AMMI analysis extracted values of the interaction principal component axis (IPCA) scores for IPCA-1 to IPCA-4 in respect of 52 pathogen isolates (Table 6) as well as 16 host genotypes (Table 7). A biplot is generated using the IPCA-1 and IPCA-2 scores for the 52 isolates and 16 host genotypes with the first principal component axis on the abscissa and the second on the ordinate (Fig. 2). The biplot displayed both the pathogen isolates and the host genotypes 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 represent isolates or host genotypes with positive IPCA-1 as well as IPCA-2 scores, while sector-2 represent positive IPCA-1 and negative IPCA-2 scores. Sector-3 represent negative IPCA-1 as well as IPCA-2 scores and sector-4 represent negative IPCA-1 and positive IPCA-2 scores. In the present study, each of the 4 host genotype groups fell into one sector *i.e.* HG-1 in sector-2, HG-2 in sector-1, HG-3 in sector-4 and HG-4 in sector-3. The ranking of these groups in order of level of susceptibility was HG-4>HG-1>HG-3>HG-2. Among the isolate groups, IG-1 and IG-6 fell into sector-1, IG-2 and IG-3 fell into sector-2, IG-4 into sector-4 and IG-5 into sector-3. The ranking of these groups of isolates according to their virulence levels was IG-3>IG-6>IG-5>IG-2>IG-4 >IG-1. Thus the biplot not only displayed the interactions of the isolates and host genotypes but also facilitated in visual description of 'which win where' pattern described by Li et al. (2006).

A polygon drawn in the biplot (Fig. 2) by joining the isolate groups located farthest from the biplot origin, encompassing all other IGs, facilitates identification of the IGs that are most virulent to the host genotype groups (Yan et al., 2000). The IGs located at the vertices are either the most or least virulent to some or all of the HGs. The vertex IG for a sector is most virulent to the HG falling in that sector. In the present study, IG-6 is most virulent on HG-1, HG-2 and HG-4; IG-2 is most virulent on HG-1 and HG-4; IG-4 and IG-5 are most virulent on HG-3 and HG-4 (Table 5). The most virulent IG-3, although not located at the vertex, is virulent on all the four HGs. The least virulent isolates in IG-1 are located close to the origin, less specific in virulence and showed virulence only on HG-4. Thus the biplot not only classified the isolates and host genotypes into groups, but also displayed the differential interactions

Table 6. Mean lesion length (cm), estimates of IPCA scores and the AMMI stability index (D_i) for the 52 isolates of *Xanthomonas oryzae* pv. *oryzae* across 16 host genotypes

Isolates	Mean	IPCA-1	IPCA-2	IPCA-3	IPCA-4	D_i value
CRXoo-1	5.36	0.23	0.35	–0.06	–0.02	0.42
CRXoo-2	6.59	–1.11	0.28	–0.02	–0.15	1.15
CRXoo-3	5.25	0.14	0.35	–0.11	–0.09	0.38
CRXoo-4	5.23	0.14	0.24	–0.07	0.05	0.27
CRXoo-5	5.23	0.20	0.19	–0.03	0.04	0.27
CRXoo-6	5.24	0.22	0.27	–0.04	0.09	0.35
CRXoo-7	5.22	0.21	0.18	–0.04	0.27	0.27
CRXoo-8	5.28	0.22	0.22	0.02	–0.01	0.31
CRXoo-9	5.19	0.03	0.30	–0.07	–0.39	0.30
CRXoo-10	5.18	0.14	0.29	–0.06	–0.02	0.32
CRXoo-11	6.41	–1.06	0.34	0.04	0.04	1.11
CRXoo-12	6.38	–1.05	0.28	–0.01	0.23	1.09
CRXoo-13	6.37	–1.08	0.32	–0.02	0.29	1.12
CRXoo-14	6.42	–1.09	0.29	0.01	0.30	1.13
CRXoo-15	6.34	–1.07	0.31	–0.04	0.16	1.11
CRXoo-16	8.60	1.57	0.41	0.04	–0.11	1.62
CRXoo-17	5.26	0.21	0.16	–0.06	0.06	0.27
CRXoo-18	5.29	0.16	0.17	–0.02	0.15	0.23
CRXoo-19	5.19	0.16	0.16	–0.05	0.27	0.22
CRXoo-20	5.23	0.14	0.20	–0.04	0.16	0.24
CRXoo-21	5.27	0.20	0.24	0.01	0.05	0.31
CRXoo-22	6.79	–1.31	–0.03	–0.12	–0.10	1.31
CRXoo-23	8.62	1.50	0.49	0.07	–0.19	1.58
CRXoo-24	5.19	0.20	0.39	0.03	–0.10	0.44
CRXoo-25	6.77	–1.35	–0.02	–0.06	–0.41	1.35
CRXoo-26	11.70	0.17	–0.71	2.07	0.15	0.73
CRXoo-27	5.23	0.21	0.28	–0.14	0.03	0.34
CRXoo-28	11.78	0.11	–0.81	2.09	–0.08	0.82
CRXoo-29	7.34	0.20	–1.22	–0.51	0.08	1.24
CRXoo-30	5.22	0.18	0.22	0.01	0.04	0.28
CRXoo-31	12.23	0.34	–0.95	–0.21	–0.51	1.01
CRXoo-32	5.22	0.20	0.25	–0.08	0.12	0.32
CRXoo-33	5.26	0.15	0.26	–0.04	–0.01	0.30
CRXoo-34	5.32	0.14	0.24	–0.01	0.08	0.28
CRXoo-35	5.20	0.20	0.19	–0.05	–0.05	0.28
CRXoo-36	7.43	0.25	–1.22	–0.49	0.56	1.25
CRXoo-37	6.79	–1.40	0.02	0.07	–0.38	1.40
CRXoo-38	12.27	0.32	–0.96	–0.30	–0.69	1.01
CRXoo-39	7.37	0.19	–1.29	–0.26	0.18	1.30
CRXoo-40	6.76	–1.31	–0.06	–0.05	–0.05	1.31
CRXoo-41	5.28	0.18	0.22	–0.04	0.06	0.28
CRXoo-42	5.27	0.14	0.31	–0.04	0.08	0.34
CRXoo-43	5.20	0.19	0.22	–0.05	0.05	0.29
CRXoo-44	6.73	–1.34	–0.03	–0.03	–0.34	1.34
CRXoo-45	7.43	0.24	–1.25	–0.44	0.26	1.27
CRXoo-46	8.60	1.53	0.58	–0.08	–0.16	1.63
CRXoo-47	12.33	0.37	–0.91	–0.31	–0.39	0.98
CRXoo-48	7.39	0.19	–1.29	–0.43	0.24	1.30
CRXoo-49	5.23	0.18	0.32	0.05	0.11	0.37
CRXoo-50	5.26	0.17	0.33	0.01	0.03	0.37
CRXoo-51	5.23	0.16	0.30	–0.02	0.13	0.34
CRXoo-52	8.59	1.51	0.58	–0.02	–0.11	1.62

with the host genotypes. Similar differential interactions were also reported by Yan and Falk (2002) through the genotype main effect plus genotype by environment interaction (GGE) biplot analysis of virulence of 8 net blotch (*Pyrenophora teres*) isolate groups to 13 barley line groups.

In the present study, there was a highly significant correlation between the IPCA-1 scores and the mean lesion length ($r=0.72^{**}$) for the respective isolates. Hence, the 'I' main effects can be represented by the IPCA-1 scores for the isolates. The pathogen isolates with lower IPCA-1 scores would produce a lower absolute IG interaction effect than those with higher absolute IPCA-1 scores and have less variable virulence (more stable) across host genotypes. The isolate stability ranking based on lower absolute IPCA-1 scores was those in IG-1, IG-2, IG-3 with a few exceptions. Thus the isolates in IG-1 possessed high stability for low virulence across host genotypes, while those in IG-2 and IG-3 showed stability for high virulence. Similarly, the isolate stability ranking based on higher absolute scores of IPCA-1 was IG-6, IG-5 and IG-4, indicating that the isolates in these groups are more variable in virulence (*i.e.* less stable) across host genotypes.

The distance of the host genotypes from the biplot origin is a measure of the discriminating ability for the pathogen isolates. In this regard, the host genotypes Cas-209 (*Xa 10*), Java-14 (*Xa 1+Xa 3+Xa 12*), IR-20 (*Xa 4*), IR-8 (*Xa 11*) and Rantai Emas (*Xa 1+Xa 2*) were most discriminating as indicated by long distance from the biplot origin. As a group, the HG-3 was most discriminating, followed by HG-4, HG-2 and HG-1. A critical insight into the pathogenicity data reveals that the host genotypes in HG-1 and HG-2 showed resistant reaction to most of the isolates, while those in HG-4 showed highly susceptible reaction to all the isolates (Table 1).

Pathogen isolates with IPCA-1 scores >0 responded positively (adaptable) to the host genotypes that had IPCA-1 scores >0 (i.e. their interaction is positive), but responded negatively to the host genotypes that had IPCA scores <0. The reverse applies for the isolates that had IPCA-1 scores <0 (Samonte et al., 2005). The biplot revealed that the isolates in IG-1, IG-2, IG-3 and IG-6 with IPCA-1 scores >0 responded positively to the host genotypes with (HG-1 and HG-2) with IPCA-1 scores >0 and hence their interaction is positive and these isolates are adaptable to the host genotypes in HG-1 and HG-2. On the other hand, the 6 isolates in IG-4 and 5 isolates in IG-5, all with IPCA-1 scores <0 are adapted to the host genotypes in HG-3 and HG-4 with IPCA-1 scores <0.

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

Table 7. Mean lesion length (cm) and estimates of IPCA scores for the 16 host genotypes across 52 isolates of *Xanthomonas oryzae* pv. *oryzae*

	Host genotypes (*Xa* genes)	Mean	IPCA-1	IPCA-2	IPCA-3	IPCA-4
1.	Kogyoku (*Xa 1+ Xa 3+Xa 12*)	5.45	0.91	–1.03	0.41	0.08
2.	Rantai Emas (*Xa 1+Xa 2*)	15.83	–1.58	–0.75	0.17	–0.91
3.	Wase Aikoku-3 (*Xa 3*)	5.41	0.89	–0.95	0.38	0.30
4.	Java-14 (*Xa 1+Xa 3+Xa 12*)	4.47	–2.25	0.79	–0.11	0.74
5.	IR-8 (*Xa 11*)	15.86	–1.70	–0.65	0.02	–0.22
6.	IR-20 (*Xa 4*)	15.70	–1.72	–0.77	0.16	–0.51
7.	IR-1545-339 (*xa 5*)	3.53	0.78	1.16	0.34	–0.20
8.	Cas-209 (*Xa 10*)	4.46	–2.26	0.78	–0.17	0.61
9.	DV-85 (*xa 5+Xa 7*)	3.61	0.83	1.20	0.24	0.01
10.	TKM-6 (*Xa 4*)	5.44	0.89	–1.06	0.24	0.36
11.	Tetep (*Xa 1+Xa 2*)	5.22	0.86	–0.25	–2.99	–0.08
12.	Semora Mangga (*Xa 4*)	3.54	0.83	1.17	0.24	–0.16
13.	CB-II (*Xa 3+xa 5+xa 13*)	5.47	0.88	–1.09	0.32	0.19
14.	BJ-1 (*xa 5+xa13*)	5.46	1.01	–0.99	0.32	0.32
15.	Zenith (*Xa 6*)	3.55	0.83	1.12	0.22	–0.35
16.	M.Sung Song (*Xa 6*)	3.34	0.79	1.29	0.20	–0.17

index 'D_i' incorporates the IPCA scores of the significant IPCs depending upon their contributions towards the interaction SS (Zhang et al., 1998). The stability index 'D_i' is useful in evaluation and identification of pathogen isolates possessing stable pathogenicity. The lower D_i values indicate high stable pathogenicity across host genotypes and *vice versa*. In the present study, there was a significant correlation between mean lesion length and the D_i values ($r=0.57^{**}$) and the ranking of isolates in ascending order of D_i values was those in IG-1 (0.22 to 0.44), IG-3 (0.73 to 1.01), IG-4 (1.09 to 1.15), IG-2 (1.24 to 1.30), IG-5 (1.31 to 1.40) and IG-6 (1.58 to 1.63). Thus the 27 isolates in IG-1 and 5 isolates in IG-3 with low D_i values showed stable pathogenicity across 16 host genotypes. Among them, IG-1 showed lowest level of pathogenicity below grand mean, while IG-3 showed highest level of pathogenicity above the grand mean (Table 5), both the groups of isolates possessing low positive IPCA-1 and high negative IPCA-2 scores (Table 6). The D_i values for the isolates in IG-2, IG-4, IG-5 and IG-6 were high, accompanied by high IPCA-1 and low to moderate IPCA-2 scores and hence showed lower level of stability across 16 host genotypes.

Interaction pattern from response plot. The pathogenicity response plots indicate the nature of pathogen isolate-by-host genotype interactions, with the main effects removed. The values plotted for each isolate group by host genotypes are deviations from additive main effects predictions of each variable. The larger the deviation, greater is the interaction between the two. The interaction may be positive or negative, depending upon the response, either higher or lower pathogenicity than the main effects expectation. In the present study, among the 4 host genotype groups, HG-1 constituting of the 5 genotypes 1,10,11,13 and 14, showed positive interactions with IG-2, IG-3 and IG-6, while the HG-2 constituting of 6 host genotypes 3,7,9,12,15 and 16, showed positive interactions with IG-1 and IG-6. The HG-3 consisting of two host genotype 4 and 8 showed large positive interactions with IG-4 and IG-5 while HG-4 constituting of 3 host genotypes 2, 5 and 6 showed small positive interactions with IG-2, IG-3 and large positive interactions with IG-4 and IG-5. Rest of the interactions were negative with a range of small to high magnitude.

The isolate groups with reasonably stable pathogenicity across the host genotypes include IG-1 with low virulence and IG-3 with high virulence (Fig. 3). The 5 isolates in IG-3 showed positive interactions with the host genotypes. 1, 2, 3, 5, 6, 10, 13 and 14, while the 27 isolates in IG-1 showed positive interactions of lower magnitude only with the host genotypes 7, 9, 15 and 16. Both the groups of isolates were located near the centre of biplot (Fig. 2) and showed minimal deviations in response plots (Fig. 3). The isolates in IG-2, IG-4, IG-5 and IG-6 showed specific adaptations to particular host genotypes due to their locations at the vertex of the polygon, which are away from the centre of the biplot (Fig. 2) and also showed higher deviations in response plots (Fig. 3). The isolates in IG-6 showed positive deviations in their response to the host genotypes 1, 3, 7, 9, 10, 11, 12, 13, 14, 15 and 16, while those in IG-4 and IG-5 showed negative deviations in their interactions to the same host genotypes. Among them, highest deviations in interactions

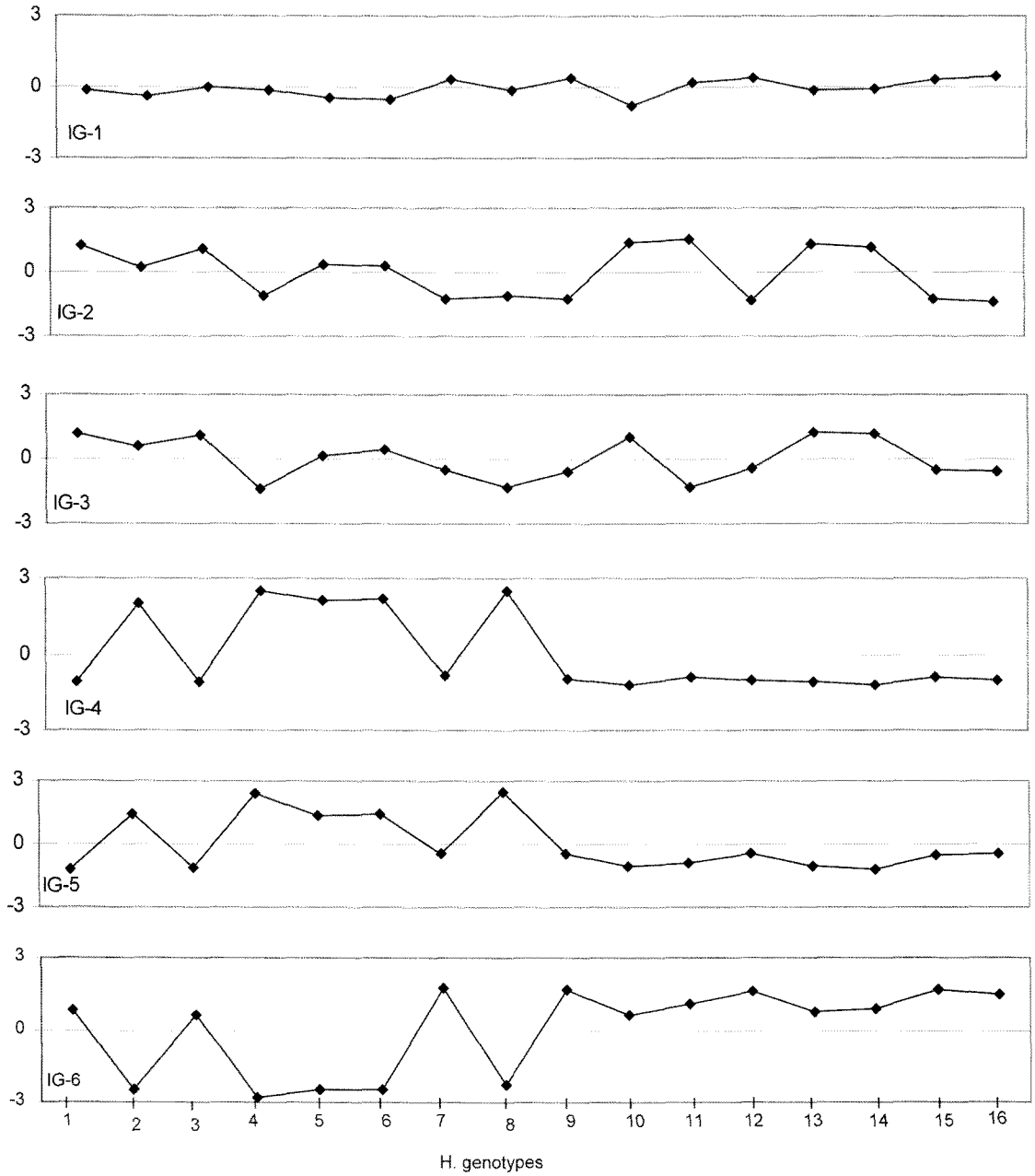

Fig. 3. Pathogenicity response plots for 6 isolate groups (IG) and 16 host genotypes according to the numerals listed in Table 7.

were showed by the isolates in IG-6, followed by IG-5, IG-4, IG-2, IG-3 and IG-1. The AMMI stability index 'D_i' values were also highest for the isolates in IG-6, followed by IG-5, IG-2, IG-4, IG-3 and IG-1 (Table 6). Hence, the 5 isolates in IG-3 were identified as possessing stable pathogenicity for high virulence, while 27 isolates in IG-1 as possessing stable pathogenicity for low virulence, against the 16 host genotypes.

The identification and use of virulent isolates 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 suitable host lines in bacterial blight disease control strategy. The low virulent stable isolates could be utilized in identification of avirulent genes in the pathogen population. The use of isolates possessing highly variable pathogenicity like those in IG-4, IG-5 and IG-6, is no doubt a word of caution to the breeders, for use in a successful breeding programme on bacterial blight resistance in rice.

In conclusion, the AMMI analysis provided (i) a better understanding of the host×pathogen interactions through analysis of variance, (ii) identification of isolates possessing

stable pathogenicity as well as discriminating host genotypes through display of the biplot and (iii) specificity in pathogenicity pattern and adaptability of the pathogen isolate groups to specific host genotype groups in a 'which won where' pattern. The scientific information obtained, could be of considerable use in developing location specific breeding strategies and selecting isolates for utilization in screening of segregating population in a program on breeding for bacterial blight resistance, as an economic method of disease control.

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