Multi-environment Testing for Reduced Incidence of Peanut Bud Necrosis Disease in India

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Abstract

Forty groundnut genotypes were tested for field resistance (reduced incidence) to peanut bud necrosis disease during 3 years at four locations in India. The 40 genotypes were grouped into seven clusters using the average linkage cluster analysis. Clusters 1 and 2 contained highly susceptible genotypes (JL 24 and TMV 2). Susceptible to moderately susceptible genotypes formed clusters 3,4, and 5. Cluster 6 represented 29 fairly resistant genotypes, and cluster 7 had the most resistant genotypes [ICGV 86430, 2192- 8(50), and 2169-5(9)]. Genotype x environment interaction variance was significant but small. The field resistance of the genotypes studied was equally effective in all environments. Selection in any of these environments is possible, but is more effective in environments which are favorable for disease development.

Introduction

Groundnut *(Arachis hypogaea* L.) genotypes show a remarkable variation in peanut bud necrosis disease (PBND) incidence. Reduced incidence (field resistance) is the collective result of resistance to peanut bud necrosis virus (PBNV) and of resistance to the vector, *Thrips palmi* Karny. Amin (1985) reported considerable field resistance in cultivar Robut 33-1, and Dwivedi et al. (1993) reported resistance in the ICRISAT germplasm line ICGV 86031. In earlier field studies, in which approximately 900 groundnut genotypes were tested, a wide range of PBND incidence was observed. These differences in disease incidence indicated various degrees of resistance. Therefore, it seemed possible to select among genotypes in a crossing program to improve the level of field resistance. Natural PBND incidence varied between locations. This could result from differences in resistance to the virus and/or the vector, as well as from differences in resistance of the genotypes grown at different locations.

The performance of a genotype depends on both its resistance and the environmental factors. To select efficiently for field resistance, we need to know whether environment and genotype are independent factors or to what extent genotype x environment (G x E) interactions are present. At the initiation of this study, no information was available on the extent of G x E interaction. Similarly, we did not have information on whether selection would yield corresponding results

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ICRISAT Conference Paper no. CP 997.

Buiel, A.A.M., Dwivedi, S.L., Prasad, M.V.R., Singh, A.B., Dharmaraj, P.S., and **Parlevliet, J.E,** 1995. Multi-environment testing for reduced incidence of peanut bud necrosis disease in India. Pages 47-54 *in* Recent studies on peanut bud necrosis disease: proceedings of a Meeting, 20 Mar 1995, ICRISAT Asia Center, India (Buiel, A.A.M., Parlevliet, J.E., and Lenné, J.M., eds.). Patancheru 502 324, Andhra Pradesh, India: International Crops Research Institute for the Semi-Arid Tropics; and P O Box 386, 6700 AJ Wageningen, The Netherlands: Department of Plant Breeding, Agricultural University of Wageningen.

across environments. Substantial G x E interaction or dissimilar results across environments are not only important in determining selection methods in a breeding program, but they may also reveal the occurrence of different virus strains.

The objectives of this multi-environment study were to determine:

- if field resistance operates across environments,
- the optimal location(s) for selection, and
- whether the field resistance is equally effective to the various virus populations to which it is exposed.

The results will lead to the development of effective selection methods for field resistance.

Materials and Methods

Field trials

Forty groundnut genotypes were grown in 12 environments (4 locations x 3 year combinations, Table 1). A large proportion of these 40 genotypes were chosen for their putative field resistance. Seven genotypes, ranging from a low incidence to a high incidence are shown in Table 2. The four locations were spread over three states in India—Uttar Pradesh (Mainpuri), Karnataka (Raichur), and Andhra Pradesh [Rajendranagar and ICRISAT Asia Center (IAC)]—and trials were carried out in the 1991-93 rainy seasons. Each trial comprised four replicates in a randomized complete block design. Plots consisted of two 4-m rows, with 20-cm interplant distance and 50- or 60-cm interrow distance.

Peanut bud necrosis disease occurred in the field as a result of natural infection. The incidence (the percentage of plants showing symptoms) was recorded, and infected plants were labeled every 2 weeks, from approximately 2 weeks after emergence until 3 weeks before harvest. At Mainpuri and Raichur, the PBND incidence was recorded monthly. Scoring and labeling of infected plants was done regularly because often infected plants die, and the PBND symptoms can no longer be identified on these dead plants.

Data analysis

Analysis of the response of 40 genotypes in 10 environments was done by cluster analysis of the genotypes. The final data of incidence were arc sine transformed and standardized (to mean = 0

Table 1. Mean peanut bud necrosis disease incidence (%) across 40 groundnut genotypes at 10 environments in India, 1991-93.

Location	Year	State	Incidence (%)
Raichur	1992	Karnataka	2.5
Raichur	1991	Karnataka	4.4
Raichur	1993	Karnataka	4.5
ICRISAT Asia Center	1991	Andhra Pradesh	9.4
ICRISAT Asia Center	1992	Andhra Pradesh	11.5
Mainpuri	1991	Uttar Pradesh	15.7
Narkoda (Rajendranagar)	1993	Andhra Pradesh	36.5
Mainpuri	1993	Uttar Pradesh	36.7
Rajendranagar	1992	Andhra Pradesh	41.1
Rajendranagar	1991	Andhra Pradesh	51.8

Table 2. Peanut bud necrosis disease incidence (%) at four locations, mean incidence over 10 locations, and the classification in the cluster analysis of seven groundnut genotypes tested in 10 environments in India, 1991-93 rainy seasons.

Entry	Raichur 1993	ICRISAT Asia Center 1991	Mainpuri 1993	Rajendra-nagar 1992	Mean	Cluster
JL 24	22	59	75	99	60	1
TMV 2	11	24	59	89	46	2
89310	13	12	56	75	36	3
86522	1	15	50	64	31	4
89268	0	11	51	48	25	5
86031	3	5	46	23	17	6
2192-8(50)	0	0	13	11	8	7

and SD = 1) per environment for clustering. Standardization of the data set was done because we were interested in the interaction effects. Clustering was performed using the average linkage cluster analysis in SAS (SAS 1988). The average incidence per cluster was used to examine correlations between environments.

The analysis of variance (ANOVA) with environments (E), genotypes (G), and genotype clusters as main effects, and G x E interaction was performed on the arc sine transformed data in GENSTAT (GENSTAT 1994).

Results

Germination was very poor in two environments, Mainpuri in 1992 and IAC in 1993. These environments were therefore omitted from the analysis.

The average nontransformed incidence of the 40 genotypes across 10 environments ranged from 8% [2192-8(50)] to 60% (JL 24) (Table 2). Most of the genotypes had an average incidence between 10% and 25%.

The average incidence of environments ranged from 2.5% at Raichur in 1992 to 51.8% at Rajendranagar in 1991 (Table 1). Raichur had a low level of PBND in all 3 years, with an average incidence below 5%, At IAC, the average incidence was around 10%. At Mainpuri, the average incidence was 16% in 1991, and 37% in 1993. The average incidence at Rajendranagar was 41% in 1991 and 52% in 1992. At Narkoda, which is located near Rajendranagar, the average incidence was 37%.

Results of the cluster analysis of genotypes are shown in Figure 1. Genotype clustering was truncated, resulting in seven clusters, explaining 87% of the genotype sum of squares (SS). Clusters 1 and 2 contained highly susceptible genotypes (JL 24 and TMV 2). Susceptible to moderately susceptible genotypes formed clusters 3, 4, and 5. Cluster 6 represented the largest group of 29 resistant genotypes, whereas the three most resistant genotypes [ICGV 86430, 2192-8(50), and 2169-5(9)] were grouped in cluster 7. The number of genotypes was not equally distributed over the clusters, as cluster 6 contained almost 75% of the genotypes. This was not surprising since we were interested in resistance, and had chosen many promising genotypes for this study. The unequal distribution emphasizes the need for clustering, because a large group of genotypes with a similar incidence will interfere with the comparison of incidence across environments.

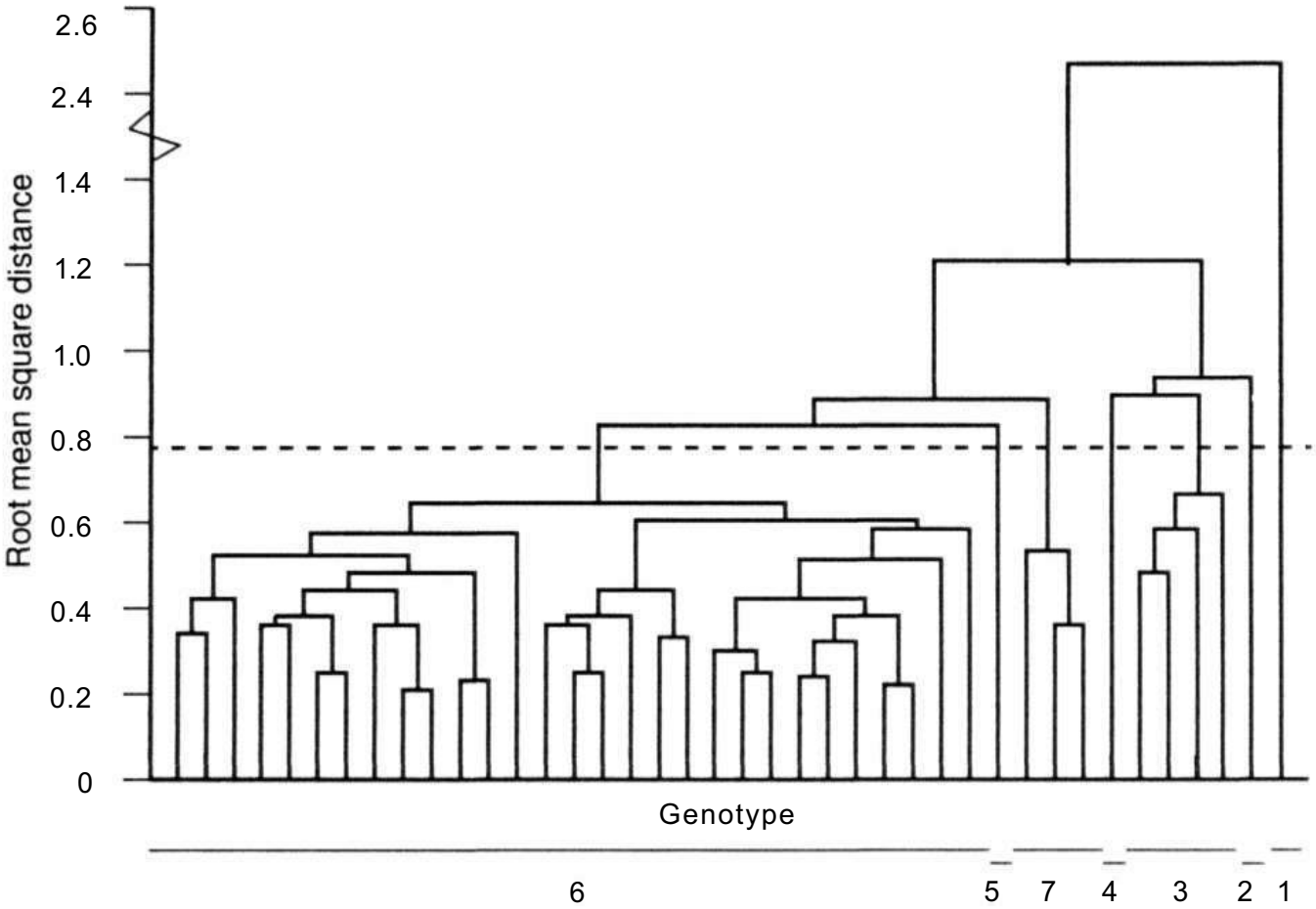


Figure 1. Dendrogram of cluster analysis of 40 groundnut genotypes tested for peanut bud necrosis disease incidence in 10 environments in India.

Table 3. Analysis of variance for arc sine transformed peanut bud necrosis disease incidence of 40 groundnut genotypes across 10 environments in India.

Source of variation	df	SS	MS	F
Replicates	3	369.68	123.23	
Environments (E)	9	326497.53	36277.50	214.01***
Residual	27	4576.85	169.51	
Genotypes (G)	39	102415.56	2626.04	43.16***
Among clusters	6	89048.95	14841.49	243.92***
Within clusters	33	13366.61	405.05	6.66***
G x E	351	41575.35	118.45	1.95***
Residual	1162	70701.59	60.84	
Total	1591	546136.56	343.27	

*** $P < 0.001$.

Main effects (environment, genotype, and genotype clusters) were highly significant in the ANOVA of the arc sine transformed incidence (Table 3). The G x E interaction was significant but small (Table 3) because the variance of the interaction (σ_{ge}=14.40) was small compared with the variance of the smallest main effect (genotype, σ_g=62.69).

Figure 2 shows the arc sine transformed incidence for different environments. The differences in incidence among clusters increased with increasing infection level and is shown as the lines of the clusters diverge (Figure 2). It implies that the small G x E interaction was primarily caused by this divergence in incidence between environments. Interactions caused by a reversed order (shown as crossover of lines in Figure 2) did occur but these were of minor importance.

In Figure 3, the interactions are shown in more detail. The clusters were ranked according to the average transformed incidence per environment. Figure 3 shows two main findings. Firstly, most of the interaction resulted from clusters 3, 4, and 5. Clusters 1, 2, 6, and 7 were consistent across environments. Secondly, Figure 3 shows that the results were rather erratic at Raichur in 1992 (with the lowest infection level).

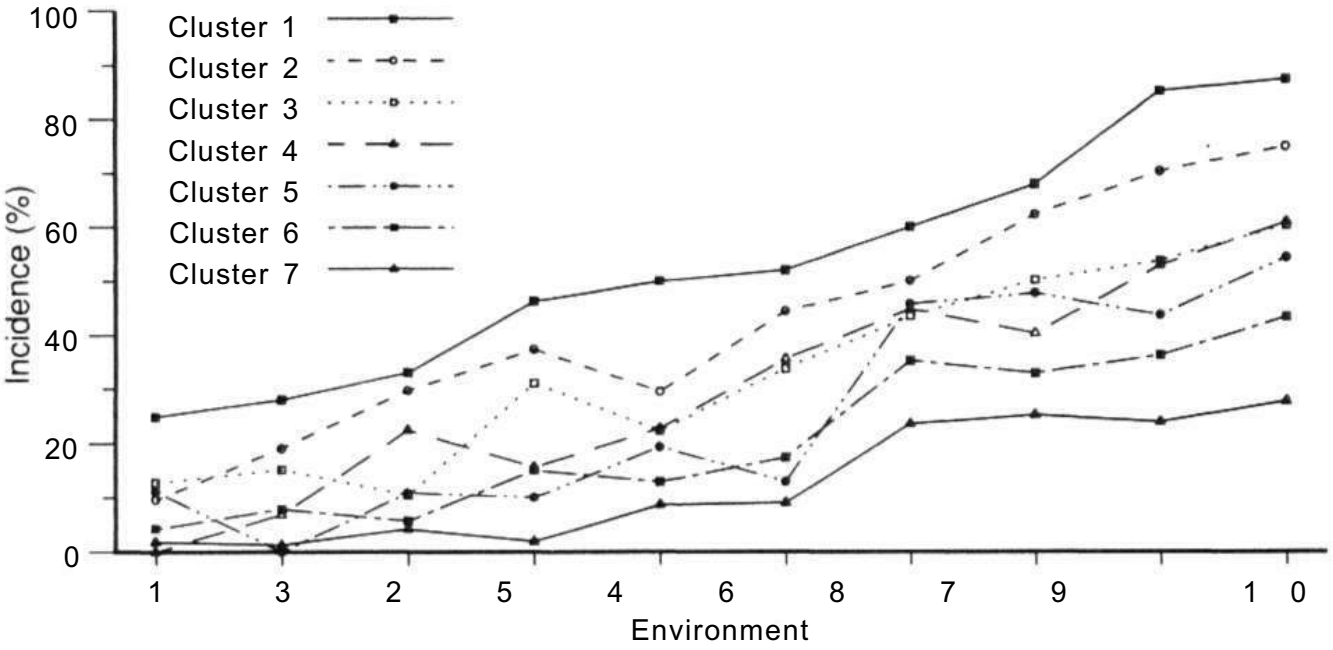


Figure 2. Peanut bud necrosis disease incidence of seven genotypes clusters in 10 environments.

Correlation coefficients (Spearman's r_s) were calculated from the ranking order of clusters among environments (Table 4). Most correlations between environments were significant at $P < 0.05$, except the correlations between Raichur in 1992 (environment 1) and other environments. The average correlation between environment 1 and other environments was 0.54. Furthermore, the average correlation among environments with a low infection (L) was poor (0.52), but a high average correlation was found among environments with an average (A) infection (0.95) and a high (H) infection (0.91).

Discussion

Genotype x Environment interaction was significant but small, and was shown to result largely from a divergent reaction of genotypes across environments and to a much lesser extent from crossover of genotypes. Thus, selection in any of the environments studied here yielded similar results. However, A and H environments discriminated considerably better among genotypes than L environments. Further, the small crossover interactions were relatively more important in L environments than in A and H environments. These interactions caused noise in the data of L environments. The infection level at Raichur (L) was low in three consecutive years; nevertheless, the most resistant genotypes of cluster 7 could be identified as highly resistant on the basis of the combined 3-year data at Raichur.

Table 4. Correlation matrix (Spearman's r_s) of 10 environments with low (L), average (A), and high (H) peanut bud necrosis disease incidence based on ranking of average incidence of seven genotype clusters.

		L 1	L 2	L 3	A 4	A 5	A 6	H 7	H 8	H 9	H 10
L	1	-									
L	2	0.43	-								
L	3	0.54	0.61	-							
A	4	0.46	0.96	0.75	-						
A	5	0.57	0.82	0.93	0.93	-					
A	6	0.39	0.89	0.86	0.96	0.96	-				
H	7	0.79	0.86	0.75	0.89	0.89	0.82	-			
H	8	0.57	0.96	0.54	0.89	0.75	0.79	0.89	-		
II	9	0.64	0.89	0.82	0.96	0.96	0.93	0.96	0.86	-	
H	10	0.46	0.96	0.75	1.00	0.93	0.96	0.89	0.89	0.96	-
Mean		0.54	0.82	0.73	0.87	0.86	0.84	0.86	0.79	0.89	0.87

Mean correlation among:

L environments	**0.52 (n=3)**
A environments	**0.95 (n=3)**
H environments	**0.91 (n=6)**

P <0.05 if r_s 0.750.
P <0.01 if r_s 0.893.

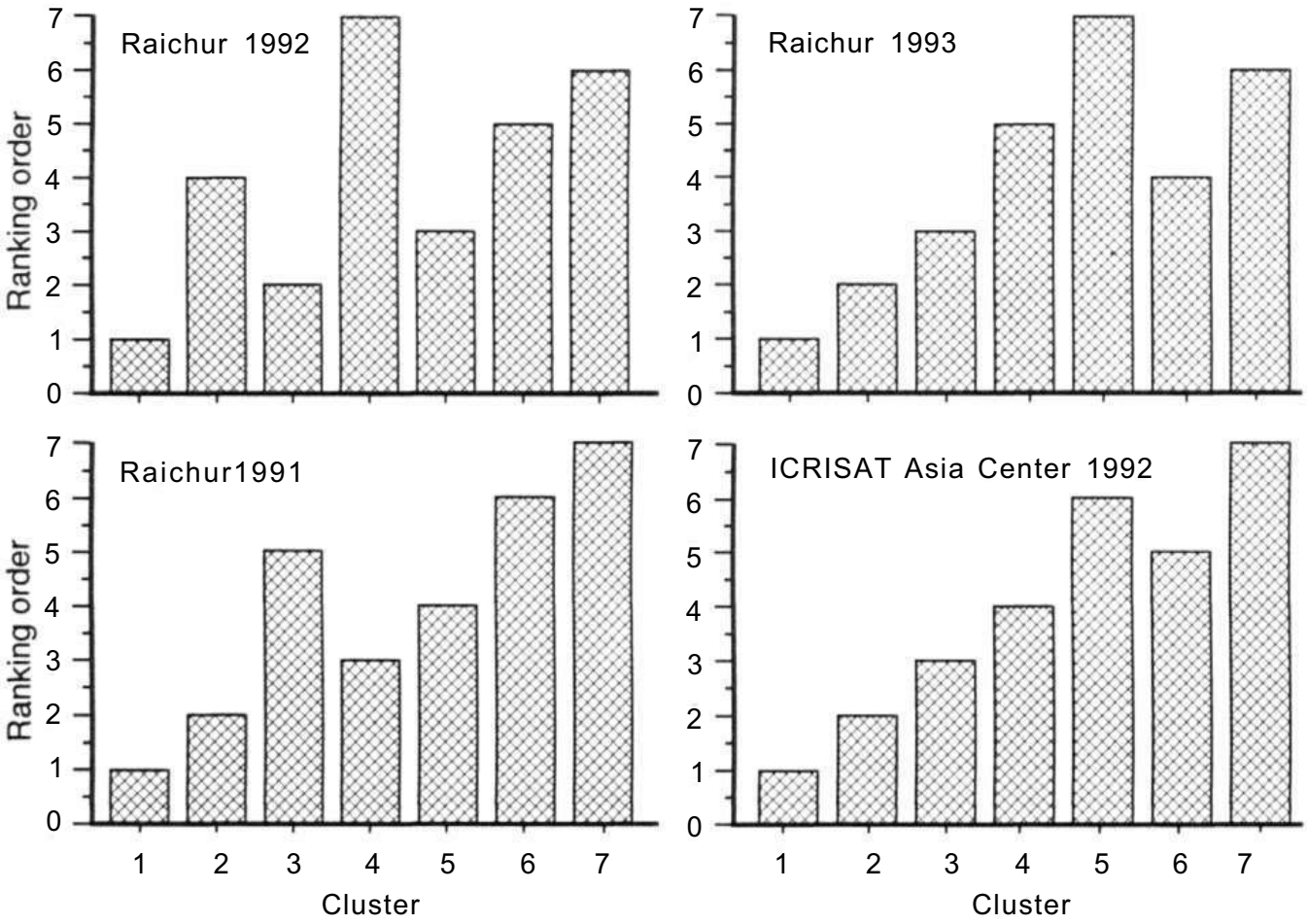
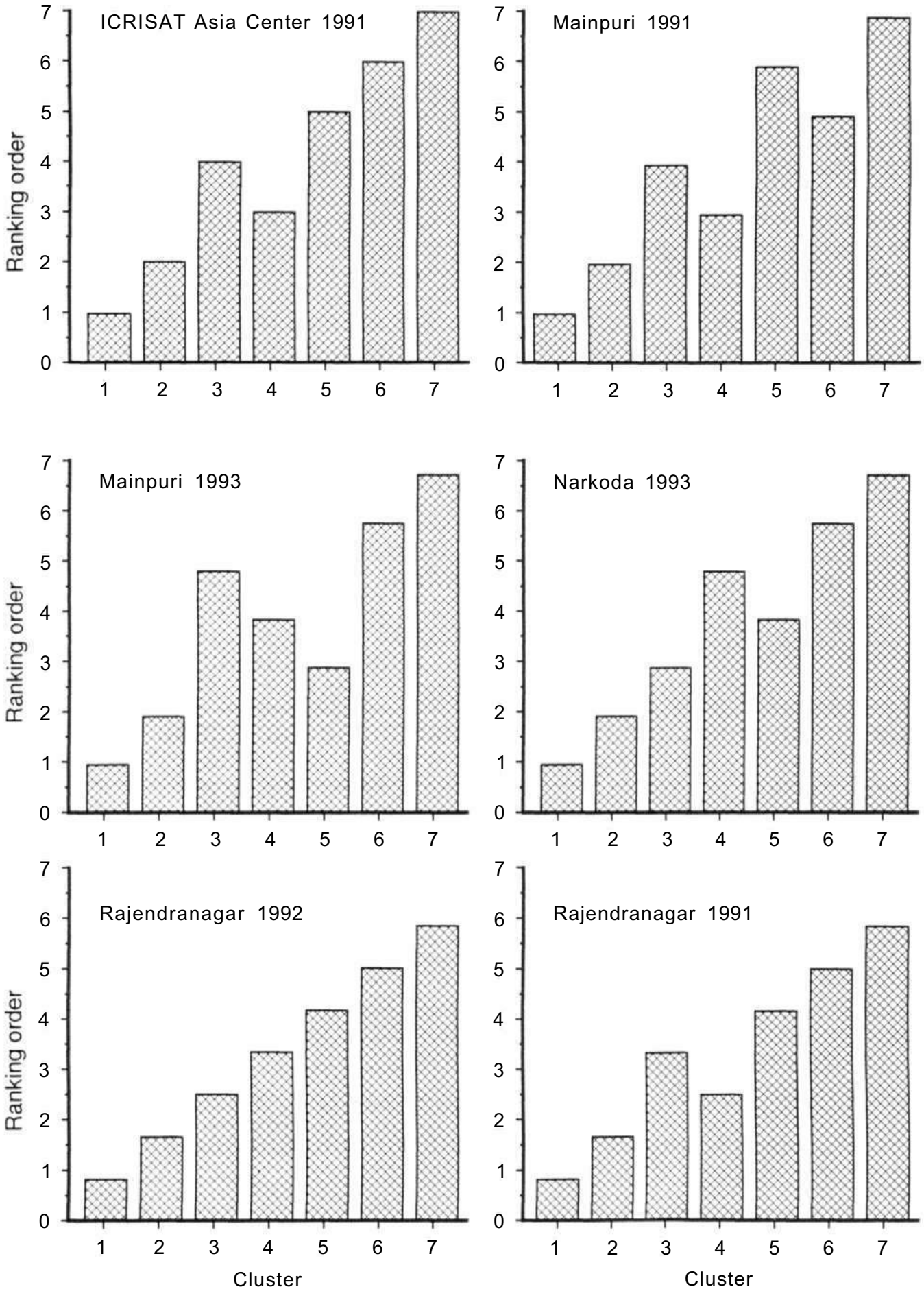


Figure 3. (Above, and opposite page) Ranking order of the mean peanut bud necrosis disease incidence of seven genotype clusters in 10 environments.


ICRISAT Asia Center 1991
Mainpuri 1991
Mainpuri 1993
Narkoda 1993
Rajendranagar 1992
Rajendranagar 1991
Ranking order
Cluster
0
1
2
3
4
5
6
7

Peanut bud necrosis disease resistance for the genotypes in this study operated in all environments. The ranking of clusters 1, 2, 6, and 7 was consistent. For clusters 3, 4, and 5, the ranking was somewhat irregular. This is probably due to the small differences in mean incidence levels for these clusters (i.e., 25.6%, 30.3%, and 33.4%).

The results showed that the PBND infection levels varied considerably among locations and to a lesser extent, among years within the same location. The interactions observed were very small compared with the main effects, and provided no evidence for virus differences among locations. In earlier studies, Reddy et al. (1992) and Poul et al. (1992) found that PBNV isolates from different locations in India (including those used in this study) reacted with PBNV polyclonal antiserum and with 10 monoclonal antibodies directed against the nucleocapsid protein. This finding, and the results presented here based on genotype reaction under field conditions, indicate that it is unlikely that the prevailing virus populations in these environments were pathogenically different.

The results presented here allow us to draw some general conclusions which will help in establishing a selection program for field resistance to PBND. Highly resistant and highly susceptible genotypes can easily be identified at locations with high or low disease levels. Results obtained at one location are also valuable to predict resistance at other locations. In locations with a low disease pressure, differences between genotypes are relatively small, and as a result, the data are noisier. This makes it more difficult to distinguish between moderately resistant genotypes, but the selection of highly resistant genotypes is not seriously impeded in these environments. We recommend selection at locations with an average or high disease pressure because selection in these discriminating environments yields more reliable results. Nevertheless, when the disease pressure is low (and it may be impossible to predict this beforehand), the combined data of repeated experiments can be used for selection.

Acknowledgements

This study was a result of cooperation between ICRISAT and three national institutes in India. The authors are grateful for the support given by Dr M.S. Basu, Groundnut Coordinator for India, in the execution of these multi-environment trials.

This work was funded by the Directorate General for International Cooperation of the Ministry of Foreign Affairs, The Hague, The Netherlands.

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