Ann. appl. Biol. (2002), **141**:143-146
Printed in Great Britain

Combining ability for resistance in peanut (*Arachis hypogaea*) to *Peanut bud necrosis tospovirus* (PBNV)

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(*Accepted 15 July 2002; Received 23 April 2002*)

Summary

The combining abilities of field resistance to peanut bud necrosis disease (PBND) caused by *Peanut bud necrosis tospovirus* (PBNV) were examined to understand the type of gene action governing resistance to the disease, and to identify peanut lines suitable for use as parents in a PBND-resistance breeding programme. The F_1 and F_2 progenies from a six-parent diallel cross and their parents were evaluated under field conditions. They were assessed for disease incidence at 30, 40, 50 and 60 days after planting (DAP), and reactions of the lines to the disease could be best differentiated at 50 and 60 DAP. Results indicated highly significant general combining ability (GCA) effects for PBND incidence in F_1 and F_2 generations. Specific combining ability (SCA) and reciprocal effects were also found to be significant, but their relative contributions to variation among crosses were much less than those of GCA effects. These results suggested that the type of gene action governing resistance to PBND was mainly additive, and selection for PBND resistance in these populations should be effective. Strong correlation coefficients between parental means and GCA effects for disease incidence were seen in both F_1 and F_2 generations, suggesting that *per se* performance of the parental line could be used as a predictor of the capability of the line to transmit its PBND-resistant attribute to progenies. The reciprocal effects were in favour of using resistant lines as female parents. The peanut lines ICGV 86388, IC 10 and IC 34 were found to be suitable for use in a PBND-resistance breeding programme.

Key words: *Arachis hypogaea*, combining ability, diallel analysis, *Peanut bud necrosis tospovirus* (PBNV)

Introduction

Peanut bud necrosis disease (PBND), caused by *Peanut bud necrosis tospovirus* (PBNV), has become a serious yield constraint of peanut (*Arachis hypogaea* L.) in south and southeast Asia. Yield losses up to 90-100% have been reported (Dwivedi *et al.*, 1993; Basu, 1995; Singh & Srivastava, 1995). PBND on peanut was first reported in 1985 from Thailand. The causal agent was assumed to be *Tomato spotted wilt virus* (Wongkaew, 1987). PBNV is now recognised as a distinct species in the genus *Tospovirus* (Reddy *et al.*, 1992; van Regenmortel *et al.*, 2000).

Genotypic differences in field resistance to PBND have been reported from India among 8000 peanut germplasm lines from a world collection (Dwivedi *et al.*, 1995). Genotypes with resistance to PBNV and its thrips vector *Thrips palmi* were developed. Although progress has been made in breeding for resistance to PBND and several high-yielding PBND-resistant peanut lines have been generated (Reddy *et al.*, 1995), information on inheritance of disease resistance was limited. Buiel (1996) studied the inheritance of quantitative resistance in peanut to PBNV in India and reported that the mean disease incidence of the F_1 to F_6 progenies was close to the mid-parent values. Dominance and epistatic factors were absent, and the resistance was inherited additively. The aim of this investigation was to examine the combining abilities of field resistance to PBND, and to identify parental lines which can be used to develop resistance to PBND in Thailand.

Materials and Methods

A six-parent diallel cross of peanut were made at Khon Kaen University, Khon Kaen, Thailand using three PBND-resistant (ICGV 86388, IC 10 and IC 34) and three susceptible cultivars [JL 24, Khon Kaen 60-1 (KK 60-1) and Khon Kaen 4 (KK 4)]. JL 24 is an early maturing cultivar widely adapted to rainfed conditions of India. KK 60-1 and KK 4 are currently grown in Thailand. The line ICGV 86388 is a selection from the cross [(Dh 3-20 × USA 20) × NC Ac 2232] (Dwivedi *et al.*, 1995). IC 10 was derived from the cross Robut 33-1 × NC Ac 2214, and IC 34 from NC Ac 1107 × (NC Ac 2232 × NC Ac 2214) (Chuapong, 1997). These three resistant lines were derived from NC Ac 2214 or NC Ac 2232 or both. NC Ac 2214 is a North Carolina State University germplasm line

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resistant to thrips but has low yield potential and other undesirable traits (Dwivedi *et al.*, 1993). NC Ac 2232 is an accession that has shown consistently low PBND incidence in India (Isleib *et al.*, 1994). These parents were crossed in a full diallel mating design with reciprocals to produce 30 F_1 crosses. Plants were allowed to self-pollinate to produce F_2 seed.

The F_1 and F_2 generations and parental lines were field planted on 21 January 2000 in Kalasin province in Northeast Thailand. A randomised complete block design with six replications was used. Seeds were planted in a single-row plot, 7.5 m-long with 30 cm spacing between plants and 50 cm spacing between rows. All plants in each plot were examined individually for symptoms of PBND at 30, 40, 50 and 60 DAP and for overall PBND incidences. Plants showing symptoms on one or more leaflets were regarded as infected and labeled. Different colours were used to distinguish plants on the basis of age when they became infected. Samples of selected diseased plants were also tested by direct antigen coating enzyme-linked immunosorbent assay (DAC-ELISA) as described by Hobbs *et al.* (1987) to confirm PBNV infection.

Diallel analyses were performed on PBND incidence in F_1 and F_2 generations using Griffing's method III, model I (F_1s and reciprocals are included but not the parents) (Griffing, 1956; Simmonds, 1979; Falconer & Mackay, 1996). The data were transformed by arcsin in order to stabilise the error variance for the percentage of infected plants (Gomez & Gomez, 1984). The general combining ability (GCA) effect for each parent and the specific combining ability (SCA) of each cross for PBND incidence were estimated for all measurement dates. Tests for significance of GCA and SCA effects were done using the *t*-test. Correlation coefficients between parental means and GCA effects were computed to compare the performance of genotypes *per se* with their performance in crosses.

Results and Discussion

PBND field incidence of the parental lines are shown in Table 1. ELISA of samples from selected symptomatic plants confirmed that the causal agent was PBNV. The disease pressure in this trial was considerably high, with the disease incidence at 60 DAP of the three susceptible lines ranging from 36.5% to 45.7%. The incidence at 60 DAP for crosses between susceptible and resistant parents ranged from 18.0 to 59.1 % in the F_1 generation and from 13.3% to 39.8% in the F_2 generation (data not shown). In general, percentage of PBNV-infected plants increased at later dates as would be expected. The exceptions were that the PBND incidences of IC 34 and IC 10 at 40 DAP were lower than those observed at 30 DAP, and of IC 10 at 60 DAP was lower than at earlier dates. This could be due to the hypersensitivity reaction to the virus of these two resistant lines. Indeed, we observed that on plants with this type of reaction the disease appeared as spots on leaves without systematic symptom. These plants became symptomless at later stages, as those spotted leaves defoliated, thus were considered healthy. Plants with systemic symptoms continued to show moderate or more severe symptoms, but never recovered. Differences among lines in terms of PBND incidence were observed as early as 30 DAP, but clear differentiation between the resistant and susceptible groups only occurred from 40 DAP onward. The coefficient of variation (C.V.) was rather high (51.8%) at 30 DAP, but declined considerably at later dates. It appeared that, based on the level of disease incidence and the value of the C.V., disease assessments at 50 and 60 DAP were more reliable than the assessments at earlier dates. Therefore, only analyses of data from these two dates are presented.

Table 2 summarises the results of combining ability analyses for percentages of PBNV-infected plants in the F_1 and F_2 generations at 50 and 60 DAP. Highly significant GCA and SCA effects were shown in the two generations and at both assessment dates, indicating the importance of both additive and nonadditive gene effects in the inheritance of PBND-resistance. The reciprocal effects were highly significant in the F_1 generation for both assessment dates, suggesting that cytoplasmic factors were also important. These effects, however, were somewhat reduced in the F_2 generation, and were significant only at 50 DAP. Considering the relative magnitudes of GCA, SCA and reciprocal effect sums of squares (Table 2), the contribution of GCA effects to variation among crosses was much higher than the SCA and reciprocal effect contributions, indicating that the gene effects governing resistance to PBND are mainly additive. These results differ somewhat from the study of Buiel (1996) in which only additive gene effect was reported. At 60 DAP, GCA sum of squares accounted for 54.4% and 60.6% of total sum of squares in the F_1 and F_2 generation respectively, indicating that heritability for this character should be considerably high under the conditions of relatively high disease pressure. These results suggest that selection for PBND resistance in these populations should be effective. However, the presence of non-additive gene effects, though at a lower magnitude, suggest that selection for PBND resistance would be more effective at later generations when this type of gene effect is reduced following selfing. In all cases, the F_1 progenies from crosses having resistant lines as female parents showed greater PBND resistance, as indicated by lower disease incidences, than their corresponding reciprocal crosses (data not shown). Although the reciprocal effects were somewhat reduced in the F_2 generation, such results suggest that resistant lines should be used as female parents in crosses to generate PBND-resistance breeding populations for selection.

Estimates of GCA effects could be used to identify superior parents. For PBND-resistance parameters, negative values for GCA effects are desirable. In the F_1 analysis, the genotype ICGV 86388 showed the

Table 1. *Means for PBND incidence (percentage of infected plants) of parental lines at different times of assessment*

	Infected plants (%)[a]			
	30 DAP[b]	40 DAP	50 DAP	60 DAP
ICGV 86388	1.39c	2.75b	4.78b	4.75b
IC 10	4.72bc	4.08b	5.42b	2.06b
IC 34	5.69bc	4.38b	6.59b	6.75b
JL 24	11.51ab	23.91a	33.34a	38.04a
KK 60-1	9.71ab	17.58a	24.51a	36.46a
KK 4	20.54a	32.02a	42.20a	45.77a
C.V. (%)[c]	51.8	39.4	36.6	34.0

[a] The parental means are illustrated as actual infection percentages but statistical analysis was based on transformed data by arcsin. Means in the same column followed by a common letter are not significantly different at 0.01 probability level
[b] DAP = days after planting
[c] C.V. (%) = coefficient of variation (%)

Table 2. *Percentages of genotype sum of squares attributable to GCA, SCA and reciprocal effects in the combining ability analyses for PBND incidence in the F_1 and F_2 generations of a six-parent diallel cross*

Effect	F_1		F_2	
	50 DAP[a]	60 DAP	50 DAP	60 DAP
GCA	66.3**	73.4**	73.3**	82.0**
SCA	15.3**	11.5**	13.8**	9.6**
Reciprocal	18.4**	15.1**	12.9*	8.3

*,** Significant at the 0.05 and 0.01 probability levels, respectively, of the respective effects in the diallel analysis of transformed data by arcsin; data represent combination of all crosses
[a] DAP = days after planting

lowest and negative GCA effects for percentages of infected plants at all dates (Table 3). This line was known to be resistant to PBNV (Dwivedi *et al.*, 1995). The lines IC 10 and IC 34 also showed negative GCA values for PBND incidence at all assessment dates, indicating their potential as sources of resistance to PBND. IC 10 was superior to IC 34 as shown by lower values of GCA effects. Both lines have been reported to be resistant to thrips feeding (Chuapong, 1997), but have some undesirable agronomic traits. They both have violet testa colour and small seed size. Similar results to the F_1 analysis were obtained from the F_2 analysis (Table 3). ICGV 86388, IC 10 and IC 34 all showed negative GCA effects for all the dates measured. However, relative performances among these three lines in terms of GCA values for disease incidence were somewhat different from those obtained from the F_1 analysis. While ICGV 86388 had the lowest GCA values for percentage of infected plants at all dates in the F_1 analysis, IC 10 was superior in the F_2 analysis. IC 34 was inferior to ICGV 86388 and IC 10 in both F_1 and F_2 generations.

Correlation coefficients (r) between parental means and GCA effects are shown in Table 3. For all assessment dates, parental means were highly and significantly correlated with GCA effects in both generations. Such a strong correlation suggests that the parental performance could be used as a predictor of the line's capacity to transmit its PBND-resistance attributes to its progenies. Thus, screening of lines in the greenhouse should be a simple but efficient means of identifying parents for inclusion in a PBND-resistance breeding programme.

Although the results of this study indicated that F_1 data were adequate for predicting performance in subsequent generations, large numbers of F_1 seeds are difficult to obtain. As a consequence, studies with F_1 seed generally involve planting in only one environment and estimates of combining ability for certain characters may be biased by environmental interactions. The use of generations later than the F_1 for combining ability analysis would, therefore, be more appropriate.

In this study, the disease pressure was sufficiently high for an effective differentiation among lines for their resistance to PBND. The results, therefore, should be reliable even though lines were tested in only one environment, unless there is significant biological variation in virus isolates. Although the peanut lines used represented only a small sample and were not randomly selected, the results obtained on the types of gene effects suggest that *per se* performance of the line should be a good indicator of its ability to transmit PBND-resistant attributes to its progenies, and superior parents could be identified simply by evaluation of peanut lines for PBND resistance. Also selection for PBND resistance would be more effective at later generations. The three resistant parental lines used in this study, ICGV 86388, IC 10 and IC 34, were found to be good donors of resistance to PBND and potential parents for a PBND-resistance breeding programme.

Table 3. *Estimates of general combining ability (GCA) for PBND incidence and correlation coefficients (r) between GCA effects and parental means in F_1 and F_2 generations of a six-parent diallel cross*

Parent	F_1[a]		F_2[a]	
	50 DAP[b]	60 DAP	50 DAP	60 DAP
ICGV 86388	-10.59**	-10.45**	-7.47**	-7.36**
IC 10	-4.37**	-7.88**	-8.43**	-10.73**
IC 34	-3.13**	-4.49**	-5.42**	-6.93**
JL 24	5.73**	7.79**	5.90**	8.45**
KK 60-1	6.42**	6.76**	8.00**	8.97**
KK 4	5.94**	8.27**	7.42**	7.60**
SE[c]	1.15	1.14	1.10	1.07
r[d] (df = 4)	0.88*	0.97**	0.92**	0.97**

*,** Significantly different from zero at the 0.05 and 0.01 probability levels, respectively;
[a] Values are arcsin transformed of percentages of infected plants; negative values indicate greater resistance
[a] DAP = days after planting
[c] df = 145 for F_1 and 142 for F_2 crosses
[d] Correlation coefficient between GCA effects and parental means for PBND incidence

Acknowledgments

This work was funded by the Royal Golden Jubilee Ph.D. Programme and the Senior Research Scholar Project of Dr Aran Patanothai under the Thailand Research Fund, and also supported by the Peanut Project, Department of Agronomy, Faculty of Agriculture, Khon Kaen University, Khon Kaen, Thailand. The authors wish to thank the Department of Plant Pathology, Faculty of Agriculture, Khon Kaen University for providing the facility for ELISA. The authors are also grateful to Dr C C Holbrook of USDA-ARS, Coastal Plain Experiment Station, Tifton, GA, and Dr T G Isleib of North Carolina State University, NC, USA, for reading the draft and giving useful suggestions for revision.

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