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Short Communication

Generation means analysis of resistance to peanut bud necrosis caused by peanut bud necrosis tospovirus in peanut

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With 2 tables

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Abstract

This study was conducted to evaluate the types of gene action governing the inheritance of resistance to peanut bud necrosis disease (PBND) in populations derived from three crosses involving two resistant (ICGV 86388 and IC 10) and one susceptible (KK 60-1) peanut lines. Populations were composed of P_1, P_2, F_1, F_2, BC_{11}, BC_{12}, $BC_{11}S$ and $BC_{12}S$. These populations were evaluated for PBND incidence in a farmer's field in Kalasin province in north-east Thailand, where PBND is a recurring problem. Results showed variations between crosses in the relative contributions of different types of gene effect. The results indicate that multiple genes control the PBND resistance trait, and that the two resistant lines differ in some of these genes. As non-additive gene effects are important in all three crosses, selection for low PBND incidence in these crosses would be more effective in later generations.

Key words: *Arachis hypogaea* — disease resistance — generation mean analysis — peanut bud necrosis tospovirus

Peanut bud necrosis disease (PBND), caused by peanut bud necrosis tospovirus (PBNV) and transmitted by *Thrips palmi* Karny, is currently the most important virus disease of peanut in South Asia (Satyanarayana et al. 1996) and in parts of China, Nepal, Sri Lanka and Thailand (Reddy et al. 1995). It can cause yield losses of over 50% in peanut (Dwivedi et al. 1995) and many other crops including chilli, potato, tomato, tobacco, and early-maturing legumes such as mung bean and urd bean. In India, yearly losses caused by this virus were estimated at more than US$89 million (Reddy et al. 1995).

Genotypic differences in field resistance to PBND have been reported among the 8000 peanut germplasm accessions screened at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) Asia Center in India (Dwivedi et al. 1995). In most cases, field resistance is associated with non-preference regarding the vector. However, in a few genotypes a lower field disease incidence was attributed to slower multiplication of the virus in the plant. Resistance to PBND has also been found in some wild *Arachis* sp. (Dwivedi et al. 1995, Reddy et al. 2000).

Currently, the genetic basis of resistance to PBND is not well understood. Buiel (1996) reported a study on the inheritance of PBND resistance in crosses of five resistant and two susceptible genotypes. He found that resistance to PBND could be explained by at least three resistance factors, which are additively inherited. Dominance and epistasis gene effects were absent. The resistance was also observed to be stable across environments. Pensuk et al. (2002) studied the combining ability for resistance to PBND in a six-parent diallel cross and reported that gene effects governing the trait were mainly additive, but non-additive gene effects were also present.

The objective of the present study was to investigate the types of gene action governing the inheritance of resistance to PBND caused by PBNV in peanut.

Plant materials: Two PBNV-resistant lines (ICGV 86388 and IC 10) of peanut (*Arachis hypogaea*) and one susceptible line [Khon Kaen (KK) 60-1] were selected for use as parents to generate populations in different generations. ICGV 86388 is a line from ICRISAT that is resistant to PBNV and the vector (Dwivedi et al. 1995, Reddy et al. 1996). IC 10 is a line that showed low thrips infestation in tests at Khon Kaen, Thailand and was derived from the cross Robut 33-1 × NC Ac 2214 (Chuapong 1997). NC Ac 2214, a North Carolina State University germplasm line, is resistant to thrips but has a low yield potential and other undesirable traits (Dwivedi et al. 1993). The susceptible line KK 60-1 is an adapted line for Thailand. In 1998, three crosses were made between the three lines; two were resistant × susceptible crosses (ICGV 86388 × KK 60-1 and IC 10 × KK 60-1) and one was a resistant × resistant cross (ICGV 86388 × IC 10). In 1999, the F_1 of each cross was selfed to generate F_2, and also backcrossed to both parents to generate backcrosses to the female parent (BC_{11}) and to the male parent (BC_{12}) using the F_1 as female parents. The BC_{11} and BC_{12} of each cross were selfed to generate $BC_{11}S$ and $BC_{12}S$, respectively. However, seeds obtained for $BC_{12}S$ of the cross ICGV 86388 × KK 60-1 were inadequate for subsequent field evaluation. Thus, eight populations (P_1, P_2, F_1, F_2, BC_{11}, BC_{12}, $BC_{11}S$ and $BC_{12}S$) were available from the crosses IC 10 × KK 60-1 and ICGV 86388 × IC 10, and seven (P_1, P_2, F_1, F_2, BC_{11}, BC_{12} and $BC_{11}S$) from the cross ICGV 86388 × KK 60-1.

Field experiment: All populations were evaluated for PBNV reactions in a single field experiment during January to May 2000. The experiment was conducted in a farmer's field in Kalasin province in north-east Thailand, where PBND has been a recurring problem. A randomized complete block design with six replications was used. Plots contained single rows, 7.5 m long, with 30 cm spacing between plants and 50 cm spacing between the rows. Benomyl was used as a seed treatment and no other fungicide or insecticide was applied to the crop. All plants of each plot were individually examined for symptoms of

PBND at 30, 40, 50 and 60 days after planting (DAP). Plants showing symptoms on one or more leaflets were regarded as infected and labelled. Different coloured wires were used to label plants that became infected at different evaluation dates. Samples of selected diseased plants were also tested by direct antigen coating enzyme-linked immunosorbent assay (DAC-ELISA) to confirm PBNV infection (Hobbs et al. 1987). Disease incidence was determined as the percentage of infected (symptomatic) plants. Arcsine transformed data were used in statistical analyses to stabilize the error variance for the percentage of infected plants (Gomez and Gomez 1984).

Statistical analysis: A generation mean analysis was separately conducted for each cross to determine additive, dominant and epistatic gene effects following the Hayman (1958) model. The notation of Gamble (1962) was used: m, a, d, aa, ad, dd. As the various generation means did not have equal variances, they were weighted using the inverse of the variance (Nigam et al. 2001). The regression analysis was used to find the best fit model as suggested by Torres et al. (1993) including the variables m, a, d, aa, ad and dd sequentially. Any effect that was not significant at the 5% level of probability was omitted from the model. Finally, only significant parameters were fitted using the weighted least squares method as described by Rowe and Alexander (1980).

Means and their corresponding standard errors for PBND incidence in the different generations of the three crosses are shown in Table 1. Only data from the disease assessment at 60 DAP are presented, as a previous study indicated that disease assessments at this date were most reliable (Pensuk et al. 2002). Differences between crosses were observed for the incidence of PBND in the F_1's relative to their corresponding parental values. For the resistant × susceptible cross IC 10 × KK 60-1, the disease incidence in the F_1 was similar to that in the susceptible parent. For another resistant × susceptible cross (ICGV 86388 × KK 60-1), the F_1 value was equivalent to the mid-parent value. For the resistant × resistant cross (ICGV 86388 × IC 10), the disease incidence in the F_1 was significantly higher than those of the two parents. A reduction in the mean value of the F_2 compared with that of the corresponding F_1 was also observed in the crosses IC 10 × KK 60-1 and ICGV 86388 × IC 10, but not in the cross ICGV 86388 × KK 60-1.

Estimates of different types of gene effect in the individual cross (Table 2) clearly illustrate the variation. Only additive and dominant gene effects were statistically significant in the cross IC 10 × KK 60-1, while all gene effects, except the dominant × dominant epistasis (dd), were significant in the cross ICGV 86388 × KK 60-1. Only dominant and additive × additive epistasis (aa) gene effects were significant in the resistant × resistant cross (ICGV 86388 × IC 10).

Table 1: Means and standard errors for peanut bud necrosis incidence (%) at 60 days after planting in different generations of three crosses between resistant and susceptible peanut lines

Generation	IC 10[a] × KK 60-1[b]	ICGV 86388[a] × KK 60-1[b]	ICGV 86388[a] × IC 10[a]
P_1	2.06 ± 3.47	4.75 ± 3.99	4.75 ± 3.99
P_2	36.46 ± 7.13	36.46 ± 7.13	2.06 ± 3.47
F_1	31.19 ± 15.64	21.84 ± 14.25	15.57 ± 9.79
F_2	22.17 ± 12.19	24.01 ± 14.72	9.17 ± 7.50
BC_{11}	16.14 ± 7.68	24.22 ± 7.03	8.03 ± 11.02
BC_{12}	36.67 ± 6.89	38.47 ± 17.58	6.95 ± 6.59
$BC_{11}S$	12.67 ± 7.76	25.61 ± 12.43	14.69 ± 9.31
$BC_{12}S$	31.01 ± 18.04	–	7.51 ± 7.75
MP	19.26	20.61	3.41

BC_{11}, first backcross generation with parental line 1; BC_{12}, first backcross generation with parental line 2; $BC_{11}S$, first backcross generation with parental line 1 selfed; $BC_{12}S$, first backcross generation with parental line 2 selfed; MP, mid-parent value.
[a] Resistant line.
[b] Susceptible line.

Table 2: Estimates of different types of gene effect for peanut bud necrosis incidence at 60 days after planting in three crosses between resistant and susceptible peanut lines

Gene effect	IC 10[a] × KK 60-1[b]	ICGV 86388[a] × KK 60-1[b]	ICGV 86388[a] × IC 10[b]
m	29.09 ± 0.77[c]	34.35 ± 4.25[c]	19.09 ± 2.81[c]
a	−14.65 ± 1.08	−3.78 ± 7.25	NS
d	16.99 ± 2.66	−11.10 ± 17.45	2.33 ± 7.74
aa	NS	−16.99 ± 17.39	−10.63 ± 15.87
ad	NS	7.99 ± 8.01	NS
dd	NS	NS	NS

m, mean; a, sum of additive effects; d, sum of dominance effects; aa, sum of additive × additive epistatic effects; ad, sum of additive × dominance epistatic effects; dd, sum of dominance × dominance epistatic effects.
[a] Resistant line.
[b] Susceptible line.
[c] Statistical analysis was based on transformed data by arcsine; NS indicates non-significance at $P = 0.05$.

The two resistant × susceptible crosses have the susceptible parent (KK 60-1) in common. Yet, the incidence in the F_1's of PBND relative to the incidence of PBND in their corresponding parents indicates that the degree of dominance of the genetic control factor is different between the two crosses. Reduction of the F_2 means to their corresponding F_1 means also differed in the two crosses. These differences indicate that the genetic factors controlling PBND resistance in the two resistant lines are not necessarily the same. This was confirmed by a significantly higher PBND incidence in the F_1 than in the two resistant parents in the cross ICGV 86388 × IC 10. PBND resistance appeared to be controlled by multiple genes. This is in agreement with the finding of Buiel (1996) who reported that resistance to PBNV could be explained by at least three resistance factors.

Additive gene effect accounted for a large portion of the genetic variance in the cross IC 10 × KK 60-1 and a considerable portion in the cross ICGV 83688 × KK 60-1. However, no additive gene effect was observed for the resistant × resistant cross (ICGV 86388 × IC 10). The incidence of PBND was similar in both ICGV 86388 and IC 10 in this study, but the incidence of PBND in their F_1 was significantly higher. However, the level of disease incidence in the F_1 was much lower than that in the susceptible line KK 60-1. It could be that these two lines possess different PBND resistance mechanisms as ICGV 86388 was reported to have field resistance to PBND (Dwivedi et al. 1995, Reddy et al. 1996), but IC 10 has been reported as thrips resistant (Chuapong 1997).

Significant dominant gene effects were obtained in all three crosses, and significant epistasis was also found in two crosses. These results differed from those of Buiel (1996) in which the resistance to PBND was reported to be additively inherited with no dominance and epistasis. An earlier study (Pensuk et al. 2002) showed that the gene effect for PBND resistance was predominantly additive, but non-additive gene effects were also present, although at a lower magnitude. The presence of non-additive gene effects suggested that selection for low PBND incidence in these crosses would be more effective in later generations.

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