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Gene Effects for Parameters of *Peanut bud necrosis virus* (PBNV) Resistance in Peanut

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Abstract: The objective of this study was to determine relative importance of gene effects for PBNV incidence and PBNV severity evaluated at 30, 40, 50 and 60 days after planting. Eight generations of three crosses involved three parental lines were evaluated for disease incidence (percentage of infected plants) and disease severity under natural occurrence of PBNV infection in a randomized complete block design with six replications. Evaluations were carried out at 30, 40, 50 and 60 Days After Planting (DAP). The analysis followed Hayman's model and Gamble's notations were used to describe parameters of gene effects. Joint scaling test was used to determine adequacy of the model. Additive gene effect was the most important contribution to genetic variation in generation means for both disease incidence and disease severity in the cross ICGV 86388 x IC 10. Selection for lower disease incidence and disease severity in this cross is promising. Additive gene effect and additive x additive epistatic gene effect were also important but in lower magnitude in the cross ICGV 86388 x KK 60-1 for disease incidence at 60 DAP. The presence of significant dominance gene effect in this cross for disease incidence might hinder the progress from selection. The consistent and significant additive gene effect for disease severity might provide a better selection strategy. Additive gene effect was significant for disease incidence only in the cross IC 10 x KK 60-1 at 60 DAP. Additive x dominance epistatic gene effect was also significant at 40 DAP, but no genetic parameter was significant for disease severity. This cross is considered less promising.

Key words: Generation means, additive-dominance model, disease incidence, disease severity, genetic variation

INTRODUCTION

Since the first occurrence of peanut bud necrosis disease in peanut (*Arachis hypogaea* L.) caused by thrips-vectored *Peanut bud necrosis virus* (PBNV) in 1985 when few infected plants were observed in a hectare, the disease poses a major threat to peanut production in Thailand especially in the dry season (Wongkaew and Chaupong, 1996). It is the most important virus disease in South (Satyanarayana *et al*., 1996) and Southeast Asia (Reddy *et al*., 1995). In India, yield loss caused by the virus of over 50% in peanut has been reported, estimating USD 89 million per annum (Dwivedi *et al*., 1995). PBNV is closely related to *Tomato Spotted Wilt Virus* (TSWV), a devastating virus of peanut in the United States and other tospoviruses (Culbreath *et al*., 2003).

As the disease is of economic importance, research attempts have been focused on formulating affective disease management programs including host plant resistance. Several useful germplasm sources has been identified and utilized as parental materials to develop new resistant cultivars (Reddy *et al*., 1995). Resistance sources from wild *Arachis* species have also reported (Reddy *et al*., 2000).

So far, information on genetic resistance to PBND has been limited to few studies and the cumulative knowledge is still not conclusive. Buiel (1996) reported that quantitative resistance to PBNV are governed by at least three resistant factors and additively inherited. Dominance and epistatic gene effects are absent in the materials including five resistant and two susceptible genotypes as parents. In a diallel study of combining ability, Pensuk *et al*. (2002) found that gene effects governing PBNV incidence were mainly additive, but non-additive gene effects, though present, were much less than those of additive effects. In a factorial study of combining ability, Kesmala *et al*. (2003) found that gene effects governing PBNV incidence were additive, and non-additive gene effects were not significant. Pensuk *et al*. (2004) reported variations in types of gene actions between three crosses involved two resistant and one susceptible parents and non-additive gene effects were also important in all three crosses. The results were based on evaluation at 60 days after planning only.

Inconsistence of the previous results might be largely due to sensitivity in reactions of peanut lines to different environments or difference in germplasm used. Gene effects might be different in different years or even

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different evaluation times in the same year due to differences in disease pressure and stages of development and intensity of infection during the course of evaluations. As genotype×environment interactions play an important role in quantitative traits, results based on data of single year might be bias and further studies are required.

In this study, we report relative importance of gene effects for PBNV incidence and PBNV severity evaluated at 30, 40, 50 and 60 days after planting. The information may provide a clearer understanding to genetic basis of PBNV resistance and have implication in breeding.

MATERIALS AND METHODS

Plant materials and experimental procedures: Three parental lines (ICGV 86388, IC 10 and KK 60-1) were selected for this study. ICGV 86388 is a PBNV resistant line under sap-inoculated greenhouse conditions (Dwivedi *et al*., 1995). IC 10 is a thrips resistant line and also be resistant to PBNV (Chuapong, 1997). KK 60-1 is a small-seeded high yielding cultivar in Thailand. The three parental lines were crossed in a diallel fashion without reciprocals. The resulting F_1 hybrids were then back-crossed to both parents and also allowed to self-pollinate to generate F_2 generation, respectively. Backcross generations were allowed to self-pollinate to produce backcross self-pollinated generations. Generations other than F_1, BC_{11} and BC_{12} were kindly provided by Dr. V. Pensuk (Pensuk *et al*., 2004) and generations F_1, BC_{11}, BC_{12} were reproduced anew. Eight generations (P_1, P_2, F_1, BC_{11}, BC_{12}, $BC_{11}S$, $BC_{12}S$ and F_2) were then available for generation means analysis study. Wangnamyen, a standard susceptible control, was also included.

Three parental lines, 18 progenies (six progenies for each cross) and a standard control were tested for reactions to PBNV incidence (Percentage of infected plants) and PBNV severity (five rating scores) under natural occurrence of PBND in the dry season after rice harvest. The entries were laid out in a randomized complete block design with 6 replications. Seeds, after fungicide treatment, were sowed on raised beds with 0.80 and 7.50 m in width and length, respectively. Each of which could accommodate two rows with 0.50 cm between rows and 0.30 cm between plants within rows. A single row plot was used for each entry in a replication. Neither pesticide nor fungicide was sprayed during crop cycle. Necessary agronomic practices were carried out including fertilization, gypsum application and weed control. Furrow irrigation was available as crop need.

Reactions to PBNV infection were evaluated at 30, 40, 50 and 60 days after planting (DAP). Plants showing visual PBND symptoms at least on one leaflet were considered diseased and diseased plants for each plot were recorded as disease incidence (percentage of infected plants) (Pensuk *et al*., 2002b). Disease severity was followed the method originally recommended by Boiteux *et al*. (1993) and later modified for peanut (Pensuk *et al*., 2002b). The rating scores were described as follows:

1 = No disease symptom
2 = Local lesion on one or some leaves without systemic infection
3 = Systemic infection without stunting
4 = Systemic infection with stunting
5 = Systemic infection with stunting, bud necrosis, bud die or whole plant dies.

Individual plants in each plot were recorded for disease scores and then the scores were averaged for whole plot.

Statistical analysis: Arcsine transformation was carried out for percentage of infected plants, where x was observed value and the calculations were run by Microsoft Excel. Percentages with value of zero were substituted by (¼)/n and those with value of 100 were substituted by 100-(¼)/n, where n was observed number in each plot. Original data were used for disease severity.

Data were subjected to analysis of variance to determine whether differences among generation means exited and Duncan's multiple range test was used to compared means (Gomez and Gomez, 1984). The calculations were accomplished by MSTAT-C package (Bricker, 1989). Means for parental lines and susceptible control were reported only.

Generation means analysis was separately carried out for each cross to determine relative importance of genetic parameters contributing to differences in generation means for disease incidence and disease severity according to the model described by Hayman (1958). Gamble notations (Gamble, 1962) were used to describe gene effects namely m (mean based on F_2 generation), a (pooled additive effects), d (pooled dominance effects), aa (pooled additive x additive effects), ad (pooled additive x dominance effects) and dd (pooled dominance x dominance effects). Various generation means were weighted by their corresponding invert variance to correct variance heterogeneity of generation means (differences between variances of non-segregating and segregating generations) (Nigam *et al*., 2001). Eight equations of generation means were constructed for each cross and genetic parameters were solved by means of algebra

matrices (Mather and Jinks, 1977; Rowe and Alexander, 1980). The adequacy of the models (additive-dominance and six parameter models) to explain variation in the generation means was determined by joint scaling test with 8-3 and 8-6 degree of freedom for additive-dominance and expanded models, respectively (Mather and Jinks, 1977; Kearsey and Pooni, 1996).

RESULTS

No significant difference was observed at 30 days after planting (DAP) for both disease incidence and disease severity (Table 1). At 40 DAP, The resistant parental lines, ICGV 86388 and IC 10, had significantly lower disease incidence and disease severity than did the susceptible parental line, KK 60-1, but they were not statistically different from the standard control, Wangnamyen. At 50 and 60 DAP, the susceptible control and KK 60-1 were similarly susceptible for both disease incidence and disease severity, and they were significantly higher than were IC 10 and ICGV 86388 for both characters. IC 10 was the most resistant lines for disease incidence, having significantly lower disease incidence than did ICGV 86388, another resistant parental line. However, they were similar for disease severity and significantly lower than the susceptible parental line, KK 60-1 and the susceptible control, Wangnamyen.

Additive-dominance model of generation means analysis adequately explained genetic variations in the generations of the crosses ICGV 86388 x IC 10 for both disease incidence and disease severity evaluated at 30, 40 and 50 DAP (Table 2). Therefore, six-parameter model, including epistatic gene effects, was not necessary for the analysis, but the expanded model is required for both disease incidence and disease severity evaluated at 60 DAP. However, the six parameter model was the best fit for disease severity only. As we did not know how to fit the model, the best fit model for disease incidence was unresolved. The results showed that additive gene effects were significant for both disease incidence and disease severity at 30, 40 and 50 DAP. At 60 DAP, both additive gene effect and additive x additive epistatic gene effect were significant for disease severity. No gene effect was statistically important for disease incidence at 60 DAP because standard errors were too large to make genetic parameters significant, and the model was not valid.

For the cross ICGV 86388 x KK 60-1, the expanded model was used to obtain genetic parameters (Table 3). No significant genetic parameter was found for both disease incidence and disease severity at 30 and 40 DAP. The model at 40 DAP was not adequate, and no attempt had been made to correct the model. Significant additive gene effect was present for disease incidence at 60 DAP and disease severity at 50 and 60 DAP. Negative sign for additive gene effect did not show decreasing effect, but it merely indicated that lower parent was assigned as P1. Dominance gene effect was significant for disease incidence at 50 and 60 DAP, but not for disease severity. Positive sign showed increasing effect of susceptibility. Additive x additive epistatic gene effect for disease incidence was also detected at 60 DAP. Intensity of

Table 1: Means of parental lines for peanut bud necrosis disease incidence and disease severity under natural infection of *Peanut bud necrosis virus* (PBNV) in the dry season 2001

	Disease incidence[1/]				Disease severity (1-5)[2/]			
Parental lines	30 DAP	40 DAP	50 DAP	60 DAP	30 DAP	40 DAP	50 DAP	60 DAP
ICGV 86388	8.03	9.44b	53.3b	39.4b	1.1	1.1b	1.4b	1.4b
IC 10	0.88	0.83c	10.3c	3.3c	1.1	1.0b	1.1b	1.0b
KK 60-1	6.14	39.80a	71.7a	73.8a	1.0	1.5a	1.8a	1.9a
Wangnamyen	4.70	19.23b	59.9a	61.6a	1.0	1.2b	1.7a	2.0a
F-ratio	ns	24:1**	44:1**	126:1**	ns	13:1**	18:1**	28:1**
CV (%)	83.1	36.8	17.1	12.6	5.1	11.3	12.6	12.8

[1/] means of original data, [2/]1 = no disease symptom and 5 = severe necrosis, ns, **non-significant and significant at 0.01 probability level, respectively by DMRT

Table 2: Gene effects for peanut bud necrosis disease incidence and disease severity of the cross ICGV 86388 x IC 10 under natural infection of *Peanut bud necrosis virus* (PBNV) in the dry season 2001

	Disease incidence[1/]				Disease severity (1-5)[2/]			
Parameters	30 DAP	40 DAP	50 DAP	60 DAP	30 DAP	40 DAP	50 DAP	60 DAP
m	6.28±0.82**	11.80±1.87**	25.32±1.24**	15.74±4.93**	1.02±0.01**	1.06±0.01**	1.20±0.02**	1.13±0.03**
a	6.95±1.30**	8.18±2.09**	9.51±2.26**	13.26±6.46ns	0.04±0.01*	0.05±0.02*	0.13±0.04*	0.21±0.05*
d	-3.59±2.72ns	3.11±5.13ns	0.86±4.04ns	4.97±21.27ns	-0.03±0.02ns	0.02±0.04ns	-0.03±0.07ns	
aa				22.85±21.54ns				0.20±0.06*
ad				-5.03±10.08ns				
dd				-42.33±38.76ns				-0.31±0.19ns
χ^2	3.32ns[†]	6.05ns[†]	3.91ns[†]	15.42**	6.29ns[†]	7.08ns[†]	6.12ns[†]	7.77ns

[†]Indicates adequacy of additive-dominance model, [1/] means of original data, [2/]1 = no disease symptom and 5 = severe necrosis, ns,*,**non-significant and significant at 0.05 and 0.01 probability levels, respectively

Table 3: Gene effects for peanut bud necrosis disease incidence and disease severity of the cross ICGV 86388 x KK 60-1 under natural infection of *Peanut bud necrosis virus* (PBNV) in the dry season 2001

	Disease incidence[1/]				Disease severity (1-5)[2/]			
Parameters	30 DAP	40 DAP	50 DAP	60 DAP	30 DAP	40 DAP	50 DAP	60 DAP
m	11.18±2.03**	21.73±3.57*	40.08±2.05**	39.36±1.86**	1.04±0.01**	1.14±0.05**	1.49±0.06**	1.56±0.07**
a	-	5.15ns12.90ns	-7.09±3.17ns	-9.20±2.74*	-	0.03±0.22ns	-0.21±0.06*	-0.26±0.07*
d	-24.32±11.73ns	1.18±27.24ns	19.57±4.83*	22.69±4.35**	-	0.00±0.07ns	0.28±0.10ns	0.34±0.16ns
aa	-22.62±12.24ns	2.39±26.88ns	17.25±6.73ns	22.29±5.91*	-	0.08±0.45ns	0.26±0.13ns	0.33±0.15ns
ad	-	15.50±15.08ns	-	-	-	0.24±0.25ns	-	-
dd	56.02±26.99ns	22.25±59.36ns	-	-	0.19±0.10ns	0.41±1.00ns	-	-
χ^2	6.80ns	12.16**	8.75ns	5.68ns	10.99ns	13.06**	6.19ns	7.85ns

†indicates adequacy of additive-dominance model, [1/] means of original data, [2/]2 = no disease symptom and 5 = severe necrosis, ns,*,**non-significant and significant at 0.05 and 0.01 probability levels, respectively

Table 4: Gene effects for peanut bud necrosis disease incidence and disease severity of the cross IC 10 x KK 60-1 under natural infection of *Peanut bud necrosis virus* (PBNV) in the dry season 2001

	Disease incidence[1/]				Disease severity (1-5)[2/]			
Parameters	30 DAP	40 DAP	50 DAP	60 DAP	30 DAP	40 DAP	50 DAP	60 DAP
m	4.27±1.18*	28.76±2.41**	41.50±7.81**	14.02ns±8.55	1.01±0.01**	1.27**±0.09	1.48±0.15**	1.12±0.08**
a	-3.75±1.54ns	-	-	-26.96**±3.36	-0.02±0.01ns	1.10ns±0.88	-	-0.45±0.04**
d	-3.45±0.47ns	18.69±7.62ns	53.32±53.75ns	-73.05ns±55.85	-0.02±0.02ns	-	1.05±0.97ns	-0.60±0.54ns
aa		-	22.31±29.52ns	78.85ns±35.43		-	0.49±0.52ns	1.26±0.44ns
ad		17.23±2.87**	21.06±9.47ns	-		0.22±0.13ns	0.350.19ns	-
dd		-	-	-388.1±209.7ns		1.95±1.78ns	-	-5.17±2.31ns
χ^2	3.31ns[†]	8.71ns	5.43ns	5.39ns	3.62ns[†]	4.76ns	6.64ns	2.11ns

†indicates adequacy of additive-dominance model, [1/]means of original data, [2/]1 = no disease symptom and 5 = severe necrosis, ns,*,**non-significant and significant at 0.05 and 0.01 probability levels, respectively

infection was comparatively higher at late evaluations than at early evaluations especially for susceptible genotypes.

For the cross IC 10 x KK 60-1, additive-dominance model was used for both disease incidence and disease severity at 30 DAP, and expanded model was used for both characters at 40, 50 and 60 DAP. The models were adequately best fit for the data. However, additive gene effect was significant for disease incidence and severity at 60 DAP only, and additive x dominance epistatic type of gene actions was found for disease incidence at 40 DAP (Table 4).

DISCUSSION

Genetic diversity of parental lines is important for creating genetic variation in progenies that favors selection for characters under improvement (Fehr, 1987). Our results clearly supported previous studies that differences in PBNV incidence and PBNV severity were caused by genetic differences (Pensuk *et al*., 2002b; Kesmala *et al*., 2006). Differences in parental lines for PBNV incidence and PBNV severity indicate that variation in generation means is caused by both genetic and environmental variations. Thus, genetic parameters conditioning variation in generation means could be determined by generation means analysis.

Previous studies found that the appropriate times for assessing PBNV resistance in peanut under field condition would be as early as 40 days after planting (DAP), but not at 30 DAP (Pensuk *et al*., 2002b) and genetic parameters were determined at 60 DAP only (Pensuk *et al*., 2004). In this study, we showed that gene actions for PBNV incidence and PBNV severity changed during the course of evaluations at 30, 40, 50 and 60 DAP and the changes might be caused mainly by change in intensity of PBNV infections. Our results were quite similar to those reported by Kaneko and Aday (1980) for Philippine downy mildew of maize. They found that dominance gene effect was somewhat associated with higher intensity of downy mildew infection although standard errors were too large to make the effect significant.

In the cross ICGV 86388 x IC 10, the parental lines were not much divergent for both characters (Both parents are resistant.) and additive gene effect was predominant in this cross. Present findings for this cross were in good agreement with those reported by Isleib and Wynne (1983). They found that additive gene effects are likely to play more important role in the inheritance of quantitative traits in peanut, if the selected parents are less divergent. As mentioned above, gene effects were likely to shift to dominance and epistatic, when more disease intensity occurred at 60 DAP. However, the effects were too low and the standard errors were too high to make the effects significant, except for additive x additive gene effect for PBNV severity. Our results for this cross were different from those reported by

Pensuk *et al.* (2004), who found significances of both additive and dominance effects in this cross. Lack of good agreement of the results might be due to difference in disease intensity.

The crosses IC 10 x KK 60-1 and ICGV 86388 x KK 60-1 were similar in that they were crosses of resistant parents with susceptible cultivates. Apart from additive gene effects, dominance and epistatic gene effects were also presented in both crosses. Patterns of gene actions for these crosses were also similar for some extent and the patterns followed general principles for gene effects of quantitative traits in peanut that effects of dominance and epistatic genes are larger in more diverse parents than more closely related parents (Isleib and Wynne (1983). In these crosses, the simple model was adequate for both characters at 30 DAP for the cross IC 10 x KK 60-1 only, when infection was very low. It is most likely that the presence of dominance and epistatic gene effects is dependent on both levels of disease intensity and genetic diversity of parents.

The inadequacy of six parameter model for disease incidence and disease severity in the cross ICGV 86388 x KK 60-1 at 40 DAP might indicate that unimportant parameters should be deleted from the model or the effect of environmental variation was too high.

The similar patterns of gene effects for disease incidence and disease severity indicated that both characters were under the control of the same genetic system. Correlations between these characters were also high (Kesmala *et al.*, 2004; Tonsomros *et al.*, 2006). Disease incidence is much friendly to breeders than disease severity because of its simplicity.

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