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Heritability, Phenotypic and Genotypic Correlation of *Peanut bud necrosis virus* Resistance and Agronomic Traits in Peanut

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Abstract: The objectives of this study were to estimate heritability for PBNV resistance parameters and to determine phenotypic and genotypic correlations among PBNV resistance parameters and agronomic traits. One hundred and ninety two progenies in the F_5 and F_6 generations of peanut as well as their parents were evaluated under natural infection of PBNV in a randomized complete block design with six replications for two years. Additional experiment of the duplicated materials was also conducted for agronomic evaluation for one year using similar experimental procedures under conditions that favored optimum agronomic performance. Heritability estimates were low to relatively high for both disease incidence and disease severity, depending on crosses, ranging form 0.10 to 0.90 and 0.00 to 0.79, respectively. Phenotypic and genotypic correlation coefficients for PBND incidence and PBND severity were high, ranging from 0.94** to 0.98** and 0.99** and 1.00**, respectively. Phenotypic and genotypic correlation coefficients between PBNV resistance parameters and agronomic characters were generally low for most pairs of characters, except between PBNV resistance parameters and 100-seed weight for which it was moderate (0.42** to 0.60**) PBNV susceptibility is somewhat associated with larger seed and might hinder the progress of breeding for large-seeded peanut with resistance to PBNV.

Key words: Agronomic characters, disease resistance, disease incidence, disease severity, resistance parameters

INTRODUCTION

Peanut bud necrosis disease (PBND) caused by thrips-vectored *Peanut bud necrosis virus* (PBNV) is a recurring problem of peanut production in Asia (Dwivedi *et al*., 1995). Yield loss of 89 million US dollars per annum has been estimated (Reddy *et al*., 1995). It is a distinct species from *Tomato spotted wilt virus* (TSWV), a major pathogen of viral disease of peanut and other crops in the United States (Jones and Baker, 1991) which was identified as the causal agent of PBND (Reddy *et al*., 1995). PBNV is classified as a virus in serogroup IV of *Tospoviruses* (Bunyaviridae) (Gowda *et al*., 1998; Chu *et al*., 2001; Akram *et al*., 2004). Several *Tospoviruses* including *Peanut yellow spot virus* (PYSV), *Groundnut ringspot virus* (GRSV), *Impatiens necrotic spot virus* (INSV) and *Peanut chlorotic fan-spot virus* (PCFV) occur on peanut.

In spite of high variation in symptom expressions of other viral diseases, the typical symptoms of PBNV are quite different. This facilitates disease evaluation under field condition with high degree of precision and further confirmation through ELISA test (Reddy *et al*., 2000).

The typical symptoms of PBNV that are distinct from other viruses are described as follows. Infected leaves become chlorotic spots and then the chlorotic spots become necrotic spots within few days of symptom development. If systemic infection does not occur, infected leaves will be defoliated and the plants are healthy. If systemic infections do occur, there have been variegated on terminal leaves and stunting symptoms occur on terminal buds. Bended and drooped petioles will be observed at this stage because the virus accumulates in the root crowns and can also limit the uptake of water from roots. As the symptoms develop, terminal buds become necrosis (Reddy *et al*., 1995).

Depending on severity of the symptoms and the growth stages of infections, infected plants at early growth stages may die and infected plants at late growth stages may die on the terminal buds or infected branches

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only. If they are alive, they will develop secondary symptoms. The secondary symptoms are described as bushy statutes, smaller leaves and more branches. The plants have healthy appearance and no primary symptom was observed at this stage. The plants may yield few pods, but the pods have smaller and misshaped kernels (Sharma, 1996).

As PBNV and related *Tospoviruses* are damaging pathogens of peanut, extensive studies have been caries out in Asia and the United States, where TSWV is a recurring problem. Germplasm has been evaluated and identified resistant germplasm sources. The cultivars with intermediate resistance to TSWV or PBNV have been releases in the United States and India (Dwivedi *et al*., 1993, 1996).

Buiel (1996) found additive inheritance of PBNV resistance in populations derived from cross of susceptible cultivars with resistant lines. Pensuk *et al*. (2004) reported additive, dominance and epistatic interaction effects controlling PBNV resistance and additive gene effect was most important. General combining ability variance was more important than specific combining ability variance in the crosses of small-seeded cultivars with PBNV resistant lines (Pensuk *et al*., 2002c). Kesmala *et al*. (2003) also found predominance of general combing ability variance when compared with specific combining ability variance in the crosses of large-seeded peanut cultivars with PBNV resistant lines.

Kesmala *et al*. (2004) reported low broad-sense heritability estimates in F_2 generation for PBND score for the crosses of large-seeded cultivars with PBNV resistant lines for percent infected plants and disease severity based on data of individual plants. Tonsomros *et al*. (2006) found moderate to high broad-sense heritability estimates for PBND and severity in F_4 generation incidence in another population, when evaluations were based on family means. The heritability estimates seemed to be higher when calculations were based on family means in stead of individual plants of segregating population.

Correlations among characters related to PBNV resistance were quite well associated (Tonsomros *et al*., 2006). However, phenotypic correlations between PBNV resistance parameters and agronomic characters were not associated (Kesmala *et al*., 2004). These studies were conducted on different sources of germplasm in early generations of segregating populations.

This study was conducted on the later generations of the crosses of large-seeded cultivars with PBNV resistant lines. We address the questions; 1) what is heritability in later generations of segregating populations for PBNV resistance parameters? and 2) are there any genotypic correlations among PBNV resistance parameters and agronomic characters in advanced generations? The objectives of this study were to estimate heritability for PBNV resistance parameters and to determine phenotypic and genotypic correlations among PBNV resistance parameters and agronomic traits. The information obtained will be important for breeding of peanut for PBNV resistance and acceptable yield.

MATERIALS AND METHODS

One hundred and ninety-two families in the F_5 through F_6 generations derived from 16 crosses of large-seeded high yielding peanut cultivars as female parents with PBNV resistant lines as male parents were used in this study. Female parents included KK 60-3, KKU 72-1, KKU 72-2 and Luhua 11. Male parents consisted of IC 10, IC 34, ICGV 86031 and ICGV 86388. The parents were crossed in M×N mating design (Simmons, 1989) and the resulting crosses were evaluated for combining ability for PBNV resistance in the F_2 generations (Kesmala *et al*., 2003). The details of cross regeneration and parental lines had been reported previously (Kesmala *et al*., 2003). Twelve random families of each cross from the F_2 generations were maintained unselected until F_4 generations. Eight parental lines were also included in the study and thus, there were 200 entries altogether.

The experiments were laid out in a randomized complete block design with 6 replications for two years in the dry season during December 2003 to May 2004 and December 2004 to May 2005 under furrow-irrigated field conditions in Kalasin province in the Northeast, Thailand. After rice harvest in December, the land was ploughed two times. Lime at the rate of 625 kg ha^{-1} was applied during soil preparation. The entries were planted on the raised beds of three meters long, each of which could accommodate each entry of two rows with spacing of 50 cm between rows and 30 cm between plants within rows. Ethephon ($C_2H_6ClO_3P$) at the rate of 0.2% was applied to the seed in order to break possible seed dormancy because the parental lines involved Virginia-type peanut. Captan at the rate of 5 g kg^{-1} seed was also used for seed treatment in order to control soil-born fungal diseases. Seeds were over-planted and the seedlings were thinned to obtain one plant per hill at 14 days after planting. Chemical fertilizer (12-24-12) of N-P_2O_5 and K_2O at the rate of 156 kg ha^{-1} was applied at 20 days after planting. Gypsum ($CaSO_4$) at the rate of 375 kg ha^{-1} was applied at 40 days after planting. Mechanical weeding was also practiced at 40 days after

planting. Irrigations were supplied to the crop at 15 day intervals starting at the first day of planting except for the adequate rainfalls.

Neither pesticide nor fungicide was applied during crop cycle. The plants were allowed to be infected by the natural occurrence of PBNV, which was most prevalent during the dry season. Data were recorded at 50, 60 and 70 days after planting for percentage of infected plants and disease severity. Percentage of infected plants was calculated for each plot as (infected plants/total plants) × 100. Visual rating of PBNV severity was evaluated for each plot using five level rating scales as suggested by Boiteux *et al*. (1993).

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 die

A bamboo stick was placed near the main-stem of a putative diseased plant at each evaluation time irrespective of where the disease symptom occurred on the plant. The colors of bamboo sticks were changed at different evaluation times for simplicity of the successive observations and for monitoring the disease progress. Data were corrected according to the latter observations if necessary.

In order to confirm whether the field evaluation procedures were valid, terminal leaves of the plants showing diseased symptoms were sampled and determined for the presence of PBNV by direct antigen coating indirect ELISA (DAC-indirect ELISA). This test was conduct for the trial in the dry season in 2004/2005 only. Two samples of each entry showing visual symptoms of PBNV infection were taken randomly. The laboratory protocol followed that reported by ICRISAT (Reddy *et al*., 1991).

The duplicated materials were also tested for agronomic performance using similar experimental procedures under conditions favored optimum growth and yield, but only one year was conducted. At harvest (120 days after planting), pod yield, seed yield, above ground dry weight and pod number were recorded per plant basis for each plot. Seed size was recorded as weight of 100 seeds. Shelling percentage was expressed as percentage of seed weight per pod weight and harvest index was calculated as the ratio of seed weight per above ground dry weight (total weight without roots).

Percentage of infected plants and disease severity were non-continuous data and thus, appropriate transformation method was possibly required. Square root transformation methods (sqrt(X+1)) was selected for both PBNV resistance parameters. The transformation was facilitated by Microsoft Excel program. The data were tested for homogeneity of variances between the data sets of two years and combined analysis of variance was performed separately for eight parents and 192 progenies according to Hoshmand (2006). All calculations at this step were run by MSTAT-C package (Bricker, 1989). Duncan's multiple range test (DMRT) was used to compare means (Hoshmand, 2006).

Variance components for disease incidence and disease severity were obtained by partitioning total phenotypic variance into variance due to environment, variance due to genotype×environment (G×E) interaction and variance due to genotype and then, broad-sense heritability estimates were calculated using relationships as follows (Falconer and Mackay, 1996).

Heritability estimate = Total genetic variance/total phenotypic variance

$$h^2 = \sigma^2_G/\sigma^2_P$$

$$\sigma^2_G = \sigma^2_F$$

$$\sigma^2_P = \sigma^2_F + \sigma^2_{FL}/l + \sigma^2/rl$$

Where:
h^2 = Heritability
σ^2_G = Genotypic variation
σ^2_P = Phenotypic variation
σ^2_F = Variance of families
σ^2_{FL} = Variance of families × environments interaction
σ^2 = Environmental variation
f = No. of families
r = No. of replications
l = No. of environments

Genotypic and phenotypic correlation coefficients for disease data were calculated based on data of individual plots of two environments as described by Kearsey and Pooni (1996). Mean squares for PBNV score (X) and PBNV incidence (Y), Mean Cross Products (MCP), Expected Mean Squares (EMS) and Expected Means of Cross Products (EMCP) are outlined in Table 1. Because agronomic data were recorded at one year only, correlation coefficients related to agronomic characters were based on disease data in one environment. All calculations in this step were run in the Microsoft Excel.

Table 1: Analysis of variance of cross and cross product

		MS character				
Source of variation	df	X	Y	MCP	EMS	EMCP
Environment (l)	l-1					
Reps within l	l(r-1)					
Families (f)	f-1	M_3'	M_3	$M_3'M_3$	$\sigma^2_E+r\sigma^2_{Fl}+rl\sigma^2_F$	$\sigma_{E'E}+r\sigma_{(Fl)'(Fl)}+rl\sigma_{F'F}$
f×l	(f-1)(l-1)	M_2'	M_2	$M_2'M_2$	$\sigma^2_E+r\sigma^2_{Fl}$	$\sigma_{E'E}+r\sigma_{(Fl)'(Fl)}$
Error	l(r-1)(f-1)	M_1'	M_1	$M_1'M_1$	σ^2_E	$\sigma_{E'E}$

Phenotypic correlation (r_P) = $(M_3'M_3)/[(M_3')(M_3)]^{1/2}$, Genotypic correlation (r_G) = $(M_3'M_3-M_2'M_2)/[(M_3'-M_2')(M_3-M_2)]^{1/2}$

RESULTS AND DISCUSSION

The reactions of parental lines for peanut bud necrosis disease incidence (percentage of infected plants) and disease score (disease severity rating) were showed in Table 2. Differences in reactions to PBND incidence and PBND score among parental lines were significant for all evaluation times and three clearly distinct groups of cultivars could be formed on the basis of these observations in both PBND incidence and PBND score. Luhua 11 was most susceptible and had the significantly highest disease incidence and disease score. The cultivars KK 60-3, KKU 72-1 and KKU 72-2 were classified as intermediate group for PBND incidence and PBND score. All male parental lines were classified as resistance group and had the significantly lowest PBND incidence and PBND score.

Although serological analysis is one of the most effective means for evaluation of reaction of peanut lines for PBNV infection, it is not convenient for analysis of large sample size in practical breeding programs and thus, limited number of samples was taken for serological analysis by direct antigen coating indirect ELISA (DAC-indirect ELISA). The results showed that reasonable accuracy of visual evaluation for PBNV reaction was obtained and 26 of 27 putatively infected plants, accounting for 96.3% were confirmed by ELISA (Table 3).

The data of means and estimates of heritability were shown in Table 4. Most heritability estimates were positive, but significances of the values could not be determined. However, the values equal to or less than 0.50 were considered low and the values higher than 0.50 and 0.75 were considered moderate and high, respectively. Therefore, the promising crosses for further selection of the lines within the crosses were determined by not only their lower disease incidence and disease severity but also their high heritability estimates.

Using these criteria, several crosses showed promising for further selection and extensive evaluations. The crosses KK 60-3×IC 10 was selected because of its lower disease incidence and disease severity only. The crosses KK 60-3×ICGV 86031, KKU 72-1×ICGV 86388 and KKU 72-2×ICGV 86388 were selected due to their consistently high heritability estimates for both disease incidence and disease severity. The crosses KK 60-3×ICGV 86388, KKU 72-2×ICGV 86031, Luhua 11×IC 10 and Luhua 11×IC 34 were selected because of their higher heritability estimates for both disease incidence and disease severity, although they were less consistent than were the crosses above mentioned (Table 4).

Phenotypic and genotypic correlation coefficients for PBNV incidence and PBNV score at 60 and 70 days after planting (DAP) were closely relates (r_P = 0.94** to 0.98**; r_G = 0.99** to 1.00**), indicating similarity and consistency of the results (Table 5).

Phenotypic and genotypic correlation coefficients between PBNV resistance parameters and agronomic characters were showed in Table 6. In general, genotypic correlation coefficients were significant in most of the cases in comparison to phenotypic correlation coefficients. No or weak correlations were observed for most pairs of characters. Positive and significant correlations were observed between disease parameters and pod weight, 100 seed weight and harvest index where as significant negative correlation was observed for pod number.

Genetic diversity of parental materials is of significant value for its contribution to genetic variation in the progenies and increases possibility for success of breeding programs. Mean and variance of characters are basic properties of any population and determine the merit of the population in breeding program. Similarly association of characters is vital for selection and improvement in the population. Likewise for PBNV resistance character in a population, lower PBND incidence and PBND score are preferable together with higher genetic variation as indicated by higher heritability estimates. In the present population differences among parental lines for PBNV resistance indicated that high genetic variation would be expected in their progeny populations and the heritability estimates would be high. The results were corresponded with the theoretical expectations because most estimates of heritability were positive although the heritability estimates are relatively low in some crosses. The low values of heritability

Table 2: Means for square root-transformed data for peanut bud necrosis disease incidence (% infected plants) and means for original data for disease severity of parental lines of peanut evaluated across two environments

	Disease incidence			Disease severity		
	50 DAP	60 DAP	70 DAP	50 DAP	60 DAP	70 DAP
Male parents						
IC 10	1.12^{e}	1.21^{c}	1.21^{c}	1.01^{d}	1.03^{c}	1.03^{c}
IC 34	1.52^{de}	1.58^{c}	1.91^{c}	1.05^{d}	1.08^{c}	1.12^{c}
ICGV 86031	1.57^{de}	1.79^{c}	1.88^{c}	1.05^{d}	1.10^{c}	1.14^{c}
ICGV 86388	1.20^{e}	1.69^{c}	1.95^{c}	1.02^{d}	1.08^{c}	1.14^{c}
Female parents						
KK 60-3	2.82^{bc}	3.66^{b}	4.13^{b}	1.24^{bc}	1.53^{b}	1.71^{b}
KKU 72-1	3.03^{ab}	3.58^{b}	4.24^{b}	1.33^{b}	1.67^{b}	1.87^{b}
KKU 72-2	2.24^{cd}	3.55^{b}	3.69^{b}	1.19^{c}	1.59^{b}	1.65^{b}
Luhua 11	3.60^{a}	4.79^{a}	5.46^{a}	1.46^{a}	2.06^{a}	2.30^{a}
F-ratio	23.20**	33.50**	30.00**	24.30**	30.30**	33.00**
CV (%)	31.30	29.10	31.40	9.90	17.00	18.50

Means in the same column followed by the same letter(s) were not statistically significant by DMRT, **Significant at 0.01 probability level, DAP = Days after planting

Table 3: Test of 27 visually infected samples of peanut for direct antigen coating indirect ELISA (DAC-indirect ELISA) at 70 days after planting (DAP) in 2004/2005

No. of samples	Symptom severity	Positive value	Negative value	Percentage of infection
27	5*	26	1	96.3

*26 samples were infected by putative PBNV with level 5 of severity and a sample from IC 10 with level 2 of severity

Table 4: Heritability estimates (h^2) and means with associated standard deviations for square root-transformed data for peanut bud necrosis disease incidence (% infected plants) and heritability estimates and means for original data for disease score of 16 crosses of peanut evaluated across two environments

	Disease incidence				Disease severity			
	60 DAP		70 DAP		60 DAP		70 DAP	
Cross	h^2	Mean	h^2	Mean	h^2	Mean	h^2	Mean
KK 60-3×IC 10	0.10	1.68±1.00	0.72	2.02±1.15	0.00	1.10±0.16	0.54	1.16±0.21
KK 60-3×IC 34	0.54	2.17±1.20	0.75	2.61±1.36	0.63	1.19±0.22	0.55	1.30±0.35
KK 60-3×ICGV 86031	0.57	2.83±1.55	0.80	3.11±1.70	0.41	1.33±0.37	0.69	1.45±0.45
KK 60-3×ICGV 86388	0.46	2.44±1.50	0.73	2.77±1.61	0.26	1.25±0.35	0.51	1.36±0.43
KKU 72-1×IC 10	0.57	2.21±1.60	0.60	2.53±1.71	0.36	1.23±0.46	0.43	1.31±0.53
KKU 72-1×IC 34	0.43	2.47±1.50	0.34	2.92±1.76	0.12	1.27±0.37	0.35	1.40±0.49
KKU 72-1×ICGV 86031	0.21	2.52±1.42	0.28	3.15±1.58	0.08	1.25±0.29	0.10	1.42±0.39
KKU 72-1×ICGV 86388	0.74	2.40±1.63	0.81	2.97±1.80	0.53	1.25±0.37	0.67	1.44±0.57
KKU 72-2×IC 10	0.51	2.19±1.37	0.51	2.49±1.58	0.36	1.18±0.27	0.46	1.27±0.36
KKU 72-2×IC 34	0.49	2.21±1.34	0.44	2.56±1.42	0.52	1.20±0.30	0.39	1.28±0.39
KKU 72-2×ICGV 86031	0.63	3.07±1.98	0.72	3.34±2.14	0.35	1.46±0.62	0.46	1.57±0.75
KKU 72-2×ICGV 86388	0.90	2.92±1.59	0.89	3.19±1.66	0.75	1.37±0.39	0.79	1.46±0.46
Luhua 11×IC 10	0.71	2.31±1.37	0.90	2.80±1.48	0.39	1.21±0.33	0.79	1.33±0.40
Luhua 11×IC 34	0.62	2.57±1.58	0.72	3.16±1.68	0.48	1.29±0.38	0.62	1.45±0.50
Luhua 11×ICGV 86031	0.53	4.64±2.12	0.58	5.24±2.25	0.27	1.83±0.74	0.53	2.12±0.85
Luhua 11×ICGV 86388	0.25	2.35±1.36	0.25	2.89±1.47	0.00	1.22±0.26	0.43	1.36±0.37
Averaged mean		2.56±1.51		2.98±1.65		1.29±0.37		1.42±0.47

DAP = Days after planting

Table 5: Phenotypic (r_P) and genotypic (r_G) correlation coefficients between peanut bud necrosis disease incidence and disease severity evaluated at 60 and 70 days after planting (DAP) across two years under natural infection of *Peanut bud necrosis virus* (PBNV)

	Disease severity			
	60 DAP		70 DAP	
	r_P	r_G	r_P	r_G
Disease incidence (60 DAP)	0.98**	1.00**	0.96**	0.99**
Disease incidence (70 DAP)	0.94**	1.00**	0.98**	1.00**

**Significant at 0.01 probability level

estimates would also be expected because the genetic variances were in part purified by genotype × environment interactions. However, the estimates were more reliable than those estimated from single location in two years. Although genotype × environment interactions were significant (data not reported), the resistant parental lines and the susceptible parental lines were clearly separated for both disease incidence and disease severity.

Table 6: Phenotypic (r_P) and genotypic (r_G) correlation coefficients between peanut bud necrosis disease parameters and agronomic characters (per plant basis) evaluated at 60 and 70 days after planting across two years under natural infection of *Peanut bud necrosis virus* (PBNV)

	Above ground dry weight (g)		Pod No.		Pod weight (g)		Seed weight (g)		100 seed weight (g)		Shelling percentage		Harvest index	
	r_P	r_G	r_P	r_G	r_P	r_G	r_P	r_G	r_P	r_G	r_P	r_G	r_P	r_G
Disease incidence														
(60 DAP)	0.10	0.12	-0.14	-0.23**	0.21**	0.30**	0.12	0.16*	0.43**	0.56**	-0.11	-0.25**	0.15*	0.32**
(70 DAP)	0.05	-0.01	-0.24**	-0.36**	0.08	0.08	0.00	-0.07	0.46**	0.60**	-0.16*	-0.31**	0.10	0.22**
Disease severity														
(60 DAP)	0.13	0.18*	-0.15*	-0.24**	0.21**	0.30**	0.13	0.15*	0.42**	0.57**	-0.11	-0.27**	0.14	0.29**
(70 DAP)	0.06	0.01	-0.27**	-0.39**	0.11	0.12	0.00	-0.07	0.47**	0.59**	-0.20*	-0.39**	0.00	0.00

*, **Significant at 0.05 and 0.01 probability levels, respectively

This information supported the previous studies of these germplasm lines for reactions to PBNV infections under greenhouse (Pensuk *et al*., 2002a, b) and field conditions (Pensuk *et al*., 2002a; Kesmala *et al*., 2006). More recently, IC 10, IC 34 and ICGV 86388 have been reported to be resistant to *Tomato spotted wilt virus* (TSWV), a closely related species of PBNV, under sap-inoculated greenhouse conditions (Do Nascimento *et al*., 2006). The results confirmed the usefulness of resistant parental lines for peanut breeding programs aiming at developing *Tospovirus* resistance in peanut.

Although a small sample size was taken to determined percentage of infected plants confirmed by ELISA test, the results revealed the favorable accuracy of visual evaluations. However, the samples of level 5 of disease severity were taken only because of limited resources. In regard to visual evaluations, multiple time evaluations are still necessary in order to monitor the development of disease symptoms because the early symptom appearance (especially level 2) could be confound. The markers of bamboo sticks facilitated the successive evaluations. However, the more symptoms developed to more advanced stages the less confounding effect could occur. Further studies of all levels of disease severity with larger sample size are required to confirm the efficacy of visual evaluation of PBNV resistance in peanut. Furthermore, Murakami *et al*. (2006) reported higher incidence of TSWV, a related species of PBNV, in root crowns compared to leaves, but this has not been reported for PBNV. Culbreath *et al*. (1992) reported incidence of asymptomatic infection based on immunoassays as high as that of disease incidence based on visible disease symptoms. This type of symptoms has not been well understood for PBNV and further investigations are required.

Compared with previous study in early generations, in which some of resistant parents were common to the parents used in this study (Tonsomros *et al*., 2006), the heritability estimates in this study were somewhat lower. This was possibly due to the fact that they evaluated in one location only. Some crosses had high heritability estimates, but some crosses did not. The differences in heritability estimates among crosses might be caused in part by genetic random drift because only small number of families of each generation were used for generation advance.

Environmental variation was also the main cause of lower heritability estimates and accounted for large portion of phenotypic variation. The control of environmental variation under natural epidemic field condition was very difficult because of the clumped infestation behavior of viruliferous thrips population (Hamilton *et al*., 1999). Differences in plant maturity as the result of differences in maturity of their parental lines would affect both thrips population build up and disease symptom development (Buiel and Parlevliet, 1996). Ambient temperatures could affect the differential responses of peanut genotypes to the virus infection (Mandal *et al*., 2002).

Good agreement of genotypic and phenotypic correlation coefficients between disease incidence and disease severity in this study added more information to the results from previous studies (Tonsomros *et al*., 2006). It was conclusive that the same genetic systems of PBNV resistance in peanut controlled both its components of resistance.

No or weak phenotypic and genotypic correlations between disease resistance parameters and agronomic characters indicated that the PBNV resistance in peanut and most agronomic characters were independently inherited. The findings suggested that the pairs of most characters under study were not associated and independent improvement of each character should be practiced. These findings strongly supported previous investigations on the early generation of these materials (Kesmala *et al*., 2003) and other populations for the phenotypic correlation (Tonsomros *et al*., 2006). However, slightly negative associations between disease resistance parameters and pod number and shelling percentage indicated that selection for lower disease incidence or disease severity could yield higher pod number per plant and higher shelling percentage. This was slightly different

from the results in early generation in which the correlation coefficients were very low (Kesmala *et al*., 2003). High propagation of plants with smaller seed and high pod number might alter the correlation between PBNV resistance and agronomic characters in more advanced generations.

Moderate and positive associations between disease parameters and 100 seed weight indicated that improvement of peanut cultivars with resistance to PBNV and large kernels might be difficult in this population because resistant genotypes might yield smaller kernels and vise versa. This might be due to negative genetic linkage of genes controlling PBNV resistance with genes controlling large seed size.

CONCLUSION

In conclusion, these findings supported other studies that the resistant parental lines (IC 10, IC 34 and ICGV 86388) are useful sources of PBNV resistance and these germplasm lines are also resistance to TSWV. Heritability estimates for PBNV incidence and PBNV severity were favorably high enough for further improvement of these characters. Both genotypic and phenotypic correlations between PBNV resistance parameters were high. High disease incidence and disease severity were somewhat associated with large seed size. This information is useful for breeders to formulate appropriate breeding strategies.

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