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A Search for Candidate Gene for Cowpea Powdery Mildew Resistance in the Southern Guinea Ecology of Nigeria

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ABSTRACT

The need to identify candidate gene for resistance to powdery mildew (PM), a major fungal foliar disease of cowpea in Southern guinea savannah of Nigeria has necessitated this research. An experiment involving 13 cowpea genotypes was laid out in a randomized complete block design (RCBD) of two replications at the Botanical Garden, University of Nigeria, Nsukka, Nigeria. Each genotype was scored for susceptibility to the disease. Four parents were afterward selected for progeny generation through a 2 x 2 factorial mating design. The F_1 hybrids were evaluated in RCBD of two replications on the field for powdery mildew resistance. The scored data was transformed by arcsine method before subjection to analysis in SAS (version, 9.3). Analysis of variance (ANOVA) revealed significant ($P<0.01$) differences among the 13 genotypes in their susceptibility to PM. The male, female and the interaction of both significantly ($P<0.001$) differed. Additive genetic variance (510.07) was higher than the dominance genetic variance (387.67). Additive gene action was prominent in this study. The broad and narrow sense heritability estimates were: 99.9% and 56.8% respectively. The average degree of dominance was 1.23 and the genetic advance was 62.09. Heterosis which signifies resistance to PM was observed in the crosses between Nsukka-BA x IT89KD-374-57, IT90K-59 x IT89KD-374-57 and IT90K-59 x Nsukka-1B. The identified resistant genotypes (IT90K-59, Nsukka-BA and IT89KD-374-57) would be resources for further breeding programme. Powdery mildew cowpea resistant cultivar development could be achieved through hybridization programme since the major contribution to the inheritance of the trait was additive.

Keywords: Powdery mildew, inheritance, genetic advance, gene action, F1 hybrids, heterosis.

Introduction

Powdery mildew (PM) is an important fungal disease in several legumes. It is caused by *Erysiphe polygoni* (Braun, 1987). It is an obligate pathogen that establishes lasting interactions with their host tissues. There are about 700 PM species capable of colonizing approximately 10,000 plant species (Braun and Cook 2012). In cowpea, *Podosphaera phaseoli* (syn. *Sphaerotheca phaseoli*) has been indicted as the causal organism (Soylu *et al.*, 2004).

Powdery mildew, a biotrophic fungus has a wide distribution. It is particularly important in climates with warm, dry days and cool nights (Smith *et al*., 1996; Sillero *et al* 2006). Gritton and Ebert (1975) summarized the economic importance of powdery mildew as causing yield and quality losses. Severe infection may cause 25-50% yield losses (Munjal *et al*., 1963; Warkentin *et al*., 1996). Reddy *et al*., (1994) and Shambarkar *et al*. (1997) reported up to 40% loss in Mung bean (*Vigna radiata*) and 45% in sesame (*Sesamum indicum*) respectively. Emechebe and Florini (1997) reported that the damages due to powdery mildew on cowpea in the Sudan savanna (a drier agro-ecology) of Nigeria were moderate. Efficiency of powdery mildew is highly dependent on the weather (Wongpiyasatid *et al*., 1999).

Control of powdery mildew has been by foliar application of chemicals (Ransom *et al*. 1991; Lewellen

and Schrandt 2001; Utkhede *et al*. 2001). Environmental risks and additional production cost of chemical purchase is associated with chemical control method. Hence, the most effective measure to control such a disease would be to breed for resistant varieties.

The genetics of resistance to powdery mildew has been studied in some legumes. Lohnes and Bernard (1992) identified *Rmd* locus to confer resistance to PM in Soybean (*Glycine max*). However, in peas (*Pisum sativum* L.), resistance to PM was reported to be controlled by several separate recessive genes (Heringa *et al*. 1969; Timmerman *et al*. 1994). The report of Tiwari *et al*. (1997) indicted polygenic inheritance for the resistance of the disease in pea but resistance to the same disease in common bean (*Phaseolus vulgaris*) was controlled by two major dominant genes which interact via double recessive epistasis (Rezende *et al.*,1999).

Report of the incidence of Powdery mildew in Nigeria is quite scanty; however, its economic importance could be devastating where the climatic condition favours the development of the organism. The Southern guinea savanna ecology is an important cowpea producing zone in Nigeria (Abayomi *et al*., 2008).

A worthwhile improvement programme for cowpea powdery mildew resistance would need information on the genetic diversity of various genotypes and the gene action controlling the trait. The present study therefore seek to understand the differential susceptibility of different cowpea genotypes to powdery mildew infection, identify candidate genes for resistance in parents and understand the gene action responsible for resistance or susceptibility in the studied genotypes.

Materials and Methods

Susceptibility and/or resistance of thirteen cowpea varieties (Table 1) to powdery mildew were assessed in the field at the Botanical garden of the University of Nigeria, Nsukka. Their seeds were planted in prepared plots at a spacing of 0. 5 x 0. 5m. The experiment was laid out in a Randomized Complete Block Design (RCBD) with three replications. Basic cultural practices were carried out to maintain the plots and the plants.

Symptom of powdery mildew was observed and the extent of attack was estimated following the method of James (1971). Quantity of mycelia growth under and on the surface of the leaves was scored per plant in each plot. Scores were rated as percentage of infection per plant as: 0 (no visible infection), 1 (up to approximately 25% leaf coverage with mycelia), 2 (approximately 26– 50% leaf coverage), 3 (approximately 51–75% leaf coverage), 4 (approximately 76–100% leaf coverage). An average score from the five sampling unit in each plot was taken.

An infection index was calculated following the equation of Wheeler (1969) as:

$$\text{Infection index} = \frac{\text{number of numeric rating}}{\text{total number of plant}} \times \frac{100}{\text{maximum disease category}}$$

Based on the varied performance of the different genotypes in the initial screening, four genotypes: two elites (IT90K-59 and IT89KD-374-57) and two local (Nsukka-BA and Nsukka-1) varieties were selected for cross breeding and progeny generation. A 2 x 2 factorial mating design was employed for the crossing programme and the expected mean square of each component in the 2 x 2 fixed model factorial mating design was presented in Table 2. The generated F_1 hybrids were evaluated on the field in RCBD of two replications. Resistance to powdery mildew was conducted following the earlier screening protocol.

Percentage infection ranged between zero (0%) to 90%. The data was transformed using Arcsine technique to normalize and stabilize the variances as recommended by Gomez and Gomez (1984) and to fit it to parametric format (Fowler *et al.,* 2008). Analysis of variance was conducted using SAS version 9.3 (SAS Institute, 2012) to partition the sources of variations in the two experiments.

Differences among treatments in the two experiments were assessed with the Duncan Multiple Range test at the 0.05 significance level. Analysis of variance of the second experiment was done following the statistical linear model of Comstock and Robinson (1948, 1952):

$$Yijk = \mu + mi\,fj + mf\,(ij) + Rk + eijk$$

Where **μ** is the grand mean, **m** is male, **f** is female and R is replication. Moreover, *i, j* and *k* are the numbers of male, female and the replications respectively.

The components of phenotypic variation includes: Additive variance ($\sigma^2 A$), Dominance variance ($\sigma^2 D$) and environmental variance ($\sigma^2 E$) Variances of the male, female and their interaction were estimated from the equation below:

$$\sigma^2 m = \frac{MSm - MSe}{fr} = \sigma^2 \text{G.C.A} = \frac{1}{2}\sigma^2 \text{A}$$

$$\sigma^2 f = \frac{MSf - MSe}{mr} = \sigma^2 \text{G.C.A} = \frac{1}{2}\sigma^2 \text{A}$$

$$\sigma^2 m \text{x} f = \frac{MSmf - MSe}{r} = \sigma^2 \text{S.C.A} = \sigma^2 \text{D}$$

Where:

$\sigma^2 e = MSe$; $\sigma^2 m$ and $\sigma^2 f$ = Variance for the general combining ability (GCA) for males and females respectively and $\sigma^2 m' f$ = Variance for the specific combining ability (SCA) of the hybrids.

Additive ($\sigma^2 A$) and dominance ($\sigma^2 D$) variances were estimated as:

$$\sigma^2 A = \frac{2\,\sigma^2 m + 2\,\sigma^2 f}{2} \text{ and } \sigma^2 D = \sigma^2 m \text{ x } f$$

The total genetic variation was calculated as:

$$\sigma^2 G = \sigma^2 A + \sigma^2 D$$

And phenotypic variance:

$$\sigma^2 P = \sigma^2 G + \sigma^2 E$$

Broad ($h^2 b.s$) and narrow sense ($h^2 n.s$) *heritabilities* were estimated from the following equations:

$$h^2 b.s = \frac{\sigma^2 G}{\sigma^2 2}, \quad h^2 \text{n.s} = \frac{\sigma^2 A}{\sigma^2 p}$$

$$\text{Average degree of dominance } (\bar{a}) = \sqrt{\frac{2\,\sigma^2 D}{\sigma^2 A}}$$

$$\text{Expected genetic advance } EGA: = \frac{h2n.s.}{(\sigma P) \text{ x } (i)}$$

$$\text{Expected genetic advance as a percent of mean (\%)} = \frac{EGA}{x} \text{ x } 100$$

Where, i = Coefficient of selection which is 2.06 at 5% selection intensity. σP = Phenotypic standard deviation and $\bar{x}$ = Mean

Heterosis (H) = $\overline{F1}$ - ($\overline{Pi}$ +$\overline{Pj}$ /2)

Where: $\overline{F_1}$ = F_1 Mean, $\overline{Pi}$ = Mean of parent one and $\overline{Pj}$ = mean of parent two.

Results

The analysis of variance for the preliminary screening revealed significant ($p<0.01$) variation among the 13 cowpea genotypes for percentage infection to powdery mildew infection (Table 3).Significantly lower mean infection were recorded for IT81D-985 (14.67), Brown Akidi(14.95) and IT89KD-245(15.39). The mean infection for IT95K-56, IT90K-277-2, Nsukka-BA, IT89KD-374-57 and L.25 were moderate ranging from 16.41 to 18.97. However, from Table 3 significantly higher mean infections were recorded for IT90K-59, IT91K-118-20, Nsukka-1and Nsukka-1W genotypes.

Analysis of variance for the factorial mating design revealed highly significant ($p<0.001$) differences among the males, the females and their progeny (Table 4). From Table 4, the mean infection for the parents and the progenies was 31.48%. The coefficient of variation for the experiment was 2.83%.

From Table 5, IT89KD-374-57 (a male parent) had the lowest mean infection of 16.7. Much higher and significant mean infection (46.26 and 37.55) was recorded for Nsukka-1 and Nsukka-BA, respectively. Powdery mildew infection was medium (25.41) for IT90K-59 (Table 5). The performance of the F_1 hybrids with respect to percentage infection with powdery mildew ranged between high susceptibility to high resistance. For example, Nsukka-BA x IT89KD-374-57 and IT90K-59 x IT89KD-374-57, respectively displayed low percentage infection of 12.92 and 20.49 (Table 5). Among the progenies, the highest infection (62.18) was observed in the cross between Nsukka-BA and Nsukka-1.

Various genetic estimates were presented in Table 6. Additive genetic variance (510.06) was much higher than the dominance genetic variance (387.67) in the approximate ratio of 10:8 (Table 6). Broad sense heritability (99.91%) was almost twice higher than the narrow sense heritability (56.77%). From Table 6, the average degree of dominance was 1.23 while the expected genetic advance was 62.1%.

Heterosis estimates for the four hybrids ranged between -8.88 to 30.21. The four genotypes exhibited significant positive and negative outperformance above their parents. Hybrids with IT89KD-374-57 as pollen parent consistently had low heterosis, however, heterosis estimates of hybrids with Nsukka-1 as paternal parent was not consistent (Table 7).

Discussion

The study shows considerable genetic variability among cowpea genotypes for powdery mildew resistance. The differential response of cowpea genotypes and hybrids further suggested that this character was under genetic control and should therefore be liable to genetic manipulation for improvement. The differential response to the same biotic environment of the screened genotypes seems to agree with the observation of Sultan (2001) that genotypes respond differently to different environment. Disease severity scores of powdery mildew varied from 14.67 for IT81D-985 to 30.323 for Nsukka-1W. IT81D-985, Brown Akidi and IT89KD-245 recorded a significantly superior mean performance for powdery mildew resistance and therefore could be used as donor parents for introgression of the powdery mildew resistance gene through hybridization programme.

IT89KD-374-57 was a product of research at the International Institute of Tropical Agriculture;

Ibadan Nigeria. Adjadi (1996) had earlier identified the genotype among others in a field evaluation trial to have higher capacity for grain production. However in this study, its selection and usage in the 2 x 2 factorial mating design seem to reveal its resistance quality to powdery mildew as a paternal parent. F_1 hybrids (Nsukka-BA x IT89KD-374-57 and IT90K-59 x IT89KD-374-57) in which the genotype was involved as a donor parent were respectively foremost in powdery mildew resistance in this experiment. The Nsukka-BA x IT89KD-374-57 had negative heterosis; corresponding to low powdery mildew infection, this supports the dominant theory of heterosis by (Jones, 1917). However, among the hybrids Nsukka-BA x Nsukka-1 was most susceptibility. Gene assortment after hybridization of the two local varieties could not produce a worthwhile resistant progeny for the disease. Superiority in performance of hybrids may be aided by the presence of elite gene(s) in either or both of the parents.

The present study showed that powdery mildew resistance in the tested cowpea genotypes was largely governed by additive gene effect. Although additive gene effect was predominant, there was an evidence of non-additive component as well. Powdery mildew resistance in Mungbean is governed by more than one gene whose effect are both additive and dominant (Gawande and Patil, 2003). Since genetic improvement by selection relies mainly on additive gene components, significant advances in breeding for powdery mildew could be made. Our finding agrees with that of Waraluk *et al*. (2009) on common bean. In this experiment, the additive genetic variance of male was relatively lower than that of the female; understanding the underlying genetic factor would be necessary. To this end, a complete diallel crossing programme may be proposed to unravel the proportion of the genetic component due to maternal effect.

A narrow and broad sense heritability of 56% and 99% respectively was observed in this study. The closeness of the broad and narrow sense heritability estimates suggests that the environmental influence on this trait is low. The narrow sense heritability estimates in this study is very high according to the recommendation of Robinson *et al*. (1949). The genetic advance of 62% observed in this study was equally very high based on the recommendation of Johnson *et al.,* (1955). The high heritability and genetic advance indicates additive gene action in the control of the trait; thus forestalling that the gene(s) conferring resistance to powdery mildew in cowpea is highly heritable and would respond to selection techniques.

Table 1. Genetic materials for the experiment and the source of collection

S/N	Genotypes	Codes	Source
1	IT90K-59	V1	IITA, Ibadan
2	IT81D-985	V2	IITA, Ibadan
3	Brown Akidi	V3	Nsukka
4	IT88D- 867-11	V4	IITA, Ibadan
5	IT95K-56	V5	IITA, Ibadan
6	IT90K-277-2	V6	IITA, Ibadan
7	IT91K-118-20	V7	IITA, Ibadan
8	Nsukka-1W(Local cultivar (White)	V8	Nsukka
9	Nsukka-BA(Black Akidi)	V9	Nsukka
10	Nsukka-1(Local cultivar Brown)	V10	Nsukka
11	IT89KD-245	V11	IITA, Ibadan
12	IT89KD-374-57	V12	IITA, Ibadan
13	L.25	V13	IAR&T, Ibadan

Table 2. Analysis of variance of factorial mating design (Fixed model)

Sources of variation	DF	SS	*MS*	*EMS*
Replications	*r-1*	$\frac{\Sigma Y..k^2}{mf} - \frac{Y..^2}{mfr}$		
Males (M)	*m-1*	$\frac{\Sigma Yi..^2}{fr} - \frac{Y...^2}{mfr}$	*MSm*	$\sigma^2e + rf\sigma^2m$
Females (F)	*f-1*	$\frac{\Sigma Y.j.^2}{mr} - \frac{Y...^2}{mfr}$	*MSf*	$\sigma^2e + rm\sigma^2m$
M.X F.	*(m-1)(f-1)*	$\frac{\Sigma Yij.^2}{r} - \frac{\Sigma Yi...^2}{r} - \frac{\Sigma .j.^2}{mr} + \frac{Y...^2}{mfr}$	*MSmf*	$\sigma^2e + r\sigma^2mf$
Error	*(mf-1)(r-1)*	$\sum Yijk^2 - \frac{\Sigma Yij.^2}{r} - \frac{\Sigma Y..k^2}{mr} - \frac{Y...^2}{mfr}$	*MSe*	σ^2e
Total	*Mfr-1*	$\sum Yijk^2 - \frac{Y...^2}{mfr}$		

Table 3. ANOVA Summary and means of the thirteen tested genotypes

Source	DF	Mean Square
Genotypes	12	59.5805744**
Error	24	20.085913
		Means
Nsukka-1W		30.326a
Nsukka-1		23.544ab
IT91K-118-20		23.486ab
IT90K-59		20.234bc
Nsukka-BA(Black Akidi)		18.972bc
IT89KD-374-57		18.972bc
IT88D-867-11		17.790bc
IT90K-277-2		16.808bc
IT95K-56		16.408bc
L.25		16.408bc
IT89KD-245		15.397c
Brown Akidi		14.952c
IT81D-985		14.670c
CV (%)		23.496
Mean		19.074

Table 4. Factorial ANOVA for resistance to Powdery mildew in F1 hybrid of Cowpea

Source	DF	Mean Square
Rep	1	0.7953
Female	1	1747.3655***
Male	1	294.4861***
Male*Female	1	776.1307***
Nsukka-1W		30.326a
Error		0.7953
CV (%)		2.8328
Mean		31.4815

Table 5. Mean performances of the parents and the Hybrids

Code	Pedigree	Means
M1	Nsukka-1	46.2606a
M2	IT89KD-374-57	16.7025b
F1	IT90K-59	25.4144b
F2	Nsukka-BA	37.5488a
F1M1	IT90K-59xNsukka-1	30.3437b
F1M2	IT90K-59x IT89KD-374-57	20.4850c
F2M1	Nsukka-BA x Nsukka-1	62.1775a
F2M2	Nsukka-BA x IT89KD-374-57	12.9200d

M1, M2 - Males; F1, F2 – Females; F1M1, F1M2, F2M1 and F2M2 – Crosses between the female and the male parents

Table 6. Estimates of Genetic parameters

S/N	Items	Estimates
1	Additive Variance	510.0652
2	Dominance Variance	387.6677
3	Error Variance	0.79538
4	Broad sense heritability	99.91148
5	Narrow sense heritability	56.76674
6	Average degree of dominance	1.232912
7	Expected genetic advance	62.09446

Table 7. Heterosis Estimates of the four hybrids

Codes	Hybrids	Heterosis
F1M1	IT90K-59 x Nsukka-1	-0.09042
F1M2	IT90K-59 x IT89KD-374-57	0.218125
F2M1	Nsukka-BA x Nsukka-1	30.20458
F2M2	Nsukka-BA x IT89KD-374-57	-8.88563

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