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Identification of dominant and recessive genes for resistance to *Fusarium* wilt in pigeonpea and their implication in breeding hybrids

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Abstract *Fusarium* wilt is an important disease of pigeonpea [*Cajanus cajan* (L.) Millsp.] and it can cause severe yield losses. Chemical control of this disease is difficult and expensive; therefore, cultivation of resistant varieties/hybrids is the most efficient strategy for enhancing the production. In the present study, by using a wilt susceptible cytoplasmic-nuclear male-sterile line and four wilt resistant fertility restorers, one dominant and one recessive gene with dominant suppressive epistatic effects were found responsible for controlling resistance to *Fusarium* wilt. Considering the annual losses and wide spread nature of wilt diseases in pigeonpea, it is imperative that all the inbred and hybrid cultivars have high level of resistance to this disease. The presence of dominant gene for resistance will increase the efficiency of breeding wilt resistant cultivars because it will yield greater proportion of resistant genotypes in segregating generations. In hybrid breeding also, the presence of dominant gene for wilt resistance will be an advantage. The transfer of this gene in female hybrid parents will ease the breeding of wilt resistant hybrids because this will allow the use of both wilt resistant as well as susceptible restorers in generating wilt resistant hybrid combinations.

Keywords Hybrid breeding · Inheritance · Pigeonpea · *Fusarium* wilt

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Introduction

Fusarium wilt is an important disease of pigeonpea [*Cajanus cajan* (L.) Millspaugh]. It is caused by a soil-borne fungus *Fusarium udum* Butler (Butler 1908) and it can survive up to 5 years on infected plant debris. According to Reddy et al. (1990) the germ tubes of the pathogen generally penetrate through the delicate root tips of pigeonpea seedlings. This is followed by a rapid mycelia growth through xylem tissues that block the vascular system in the plants resulting in partial or complete wilting of branches and main stem. Although wilt is reported from over a dozen countries but it is more prevalent in India, Nepal, and Myanmar in Asia; and Kenya, Malawi, and Tanzania in Africa. Wilt disease is known to cause severe yield losses in most regions. Grover and Pental (2003) while studying major production constraints in field crops reported that the losses from wilt disease in farmers' fields is the second largest yield reducer after *Helicoverpa* pod borers. The losses by wilt in Asia were estimated to be about US$35 million, while in the African countries such losses were around US$5 million (Kannaiyan et al. 1984). Although no recent survey has been

conducted, pigeonpea pathologists believe that the wilt incidence has increased significantly over the time (Gwata et al. 2005; Mamta Sharma, personal communication). Although there are a few chemicals and cultural practices which can reduce the disease incidence (Dhar and Reddy 1999), but these are expensive and commercially not viable. Pigeonpea is a crop of small-scale rainfed farmers and predominantly cultivated under subsistence agriculture, where it receives minimum inputs primarily due to cost factors. The best option to overcome this problem is to breed varieties/hybrids with stable genetic resistance. To achieve this, it is imperative to have quality resources such as wilt screening nursery and a set of stable resistant donor parents. A knowledge regarding inheritance of this disease will help in enhancing the efficiency of breeding wilt resistant pure line and hybrid cultivars. So far only limited studies have been conducted to understand the genetic systems that control wilt disease in pigeonpea; and the overall picture about its inheritance is still unclear (Agrawal 2003; Saxena 2008). The present study, therefore, was undertaken to know more about the genetic systems controlling resistance to *Fusarium* wilt disease. Also, it was planned that the materials derived from this study will be useful to breed genetic stocks, embedded with known wilt resistance genes for use in conventional and molecular-assisted breeding programs. The presence of pathogenic variability may also influence the results of genetic studies and breeding efforts hence specific information on the presence of different variants and their relative virulence is essential. At present such information about *Fusarium* wilt in pigeonpea is limited and inconclusive (Chattopadhyay and Sen Gupta 1997; Booth 1978; Reddy and Chaudhary 1985; Pawar and Mayee 1986; Gupta et al. 1988; Okiror and Kimani 1997; Tiwari and Dhar 2011). Therefore, in this study no attempt was made to identify the pathogenic variants present in the wilt sick nursery at Patancheru where the experiments were conducted. However, Dhar (personal communication) claimed that at Patancheru 'Variant 1' of *F. udum* (Tiwari and Dhar 2011) is prevalent.

Materials and methods

Genetic materials

To study the inheritance of *Fusarium* wilt, four fertility restoring (R-lines) lines which exhibited high levels of wilt resistance for four consecutive years (Table 1) were selected as male parents. The R-lines were derived through pedigree selection from single crosses. These lines were crossed with a highly susceptible cytoplasmic nuclear male-sterile (CMS) line (A-line) ICPA 2051 as a female parent. This mating design ensured quality hybridization with no chance of any self-seed amongst the F_1s. The F_1 plants were selfed using muslin cloth bags to avoid cross pollination and to advance the generation. All the wilt resistant F_2 segregants of cross ICPA 2051 × ICPL 20116 were also selfed to study the segregation in F_3 generation; but sufficient seed for evaluation could be harvested only from 18 plants. To generate additional information on the dominance relationships of the genes controlling wilt incidence, 15 new F_1 experimental hybrids involving wilt susceptible/resistant male parents and ICPA 2051 as female parent were also made to evaluate their disease reaction.

Table 1 Parents used in genetic studies and their wilt reaction in sick nursery at Patancheru, 2006–2009

Line	Parentage	Wilt disease[a] (%)				
		2006	2007	2008	2009	Mean
ICPA 2051	Indian germplasm	–	100	98	100	99.3
ICP 2376 (sus. C)	Indian germplasm	91	94	93	90	92.2
ICPL 20106	MS 3783 × ICPL 87119	8	3	9	0	5.0
ICPL 20116	MS 3783 × ICPL 87119	2	1	1	0	1.0
ICPL 20136	MS 3783 × GAUT 85	0	4	0	0	1.0
ICPL 87119	C 11 × ICP 1-6	2	1	1	0	1.0
ICP 8863 (res. C)	Indian germplasm	0	0	0	0	0.0

[a] Disease score = susceptibility %, calculated by counting susceptible plants from total number of plants

Screening

A wilt screening field plot technique was conceptualized and used by Butler (1908). Subsequently, the technology was improved by McRae and Shaw (1933), Vaheeduddin and Nanjundiah (1956), and Nene et al. (1981). In this technique, the inoculum load of *F. udum* in the sick plot is artificially enhanced and maintained uniformly across the field by incorporating chopped wilted plants into the soil every year. The screening nursery at Patancheru was established in 1975 and since then its mean inoculum load is being maintained above 5×10^6 spores/m^2.The parents and test materials were sown at the onset of rainy season. A basal dose of 100 kg ha^{-1} of di-ammonium phosphate was applied and the crop was grown with two irrigations and three weeding. To monitor the disease build up in the sick nursery one row each of a susceptible (ICP 2376) and a resistant (ICP 8863) control was sown after every 10 test rows. Since the plant mortality within the susceptible rows was high (>90 %), the test materials were sown in non-replicated plots using four meter long ridges, spaced 75 cm apart. The plant to plant spacing was maintained at 25 cm. Since variable numbers of seeds (Table 2) were available for the parents, F_1, F_2, and F_3 generations, the plots size were also different. Counts for the susceptible (completely or partially dead) and resistant (disease free) plants were made between 180 and 190 day after sowing when most of the resistant plants reached maturity. The data from each set of materials were subjected to χ^2 test to assess their goodness of fit to different phenotypic ratios.

Results

Four F_1 hybrids involving a wilt susceptible and four resistant restorers were evaluated along with susceptible and resistant controls in a wilt-sick nursery. The susceptible check recorded >90 % plant mortality across the field, reflecting the presence of high levels of inoculum load of *F. udum* in the entire field and, thereby, allowing remote chance for escape. Most of the F_1 plants in different crosses were resistant suggesting dominance of wilt resistance. The χ^2 tests of F_1 data (Table 2) showed a good fit to 1 resistant: 0 susceptible ($p = 0.48$–1.0 in different crosses). In F_2 generation, although the population size was limited (72–87 plants), the segregation for resistance and susceptibility produced encouraging results. In each population the estimates of χ^2 value showed a good fit to 13 (resistant):3 (susceptible) ratio ($p = 0.27$–0.85); suggesting that one dominant (e.g. Wr_1Wr_1) and one recessive (e.g. wr_2wr_2) gene conferred resistance to wilt disease in pigeonpea, with Wr_1 over-riding the expression of Wr_2 gene. The double recessive ($wr_1wr_1wr_2wr_2$) genotypes were also resistant due to the presence of homozygous recessive wr_2wr_2 alleles. The expected genotypic constitution of the susceptible genotypes was either $wr_1wr_1Wr_2Wr_2$ or wr_1wr_1 Wr_2wr_2. In F_1 and F_2 progenies of cross ICPA

Table 2 Segregation for *Fusarium* wilt resistance in F_1 and F_2 generations of four crosses

Cross/generation	Observed plants			χ^2 cal.	Prob.
	Total	Resistant	Susceptible		
F_1 generation (expected ratio 1:0)					
ICPA 2051 × ICPL 20106	18	15	3	0.50	0.48
ICPA 2051 × ICPL 20116	13	13	0	0.00	1.0
ICPA 2051 × ICPL 20136	30	29	1	0.03	0.86
ICPA 2051 × ICPL 87119	13	13	0	0.00	1.0
Pooled data	74	70	4	0.22	0.64
F_2 generation (expected ratio 13:3)					
ICPA 2051 × ICPL 20106	73	63	10	1.22	0.27
ICPA 2051 × ICPL 20116	87	70	17	0.04	0.85
ICPA 2051 × ICPL 20136	72	57	15	0.21	0.65
ICPA 2051 × ICPL 87119	73	60	13	0.04	0.84
Pooled data	305	250	55	0.10	0.89

Table 3 Segregation for wilt resistance observed within wilt resistant F_3 progenies of cross ICPA 2051 × ICPL 20116

Prog. no.	Observed plants		χ^2 cal.	Prob.	Expected F_2 genotype
	No. of resistant plants	No. of susceptible plants			
(A) F_3 progenies segregating in 3:1 ratio					
3	14	6	0.27	0.60	$Wr_1\ wr_1\ Wr_2Wr_2$
10	14	5	0.02	0.89	(2/16)
12	10	4	0.10	0.75	
5	14	5	0.02	0.89	
16	9	5	0.86	0.36	
20	15	6	0.14	0.71	
Pooled ($n = 6$)	76	31	0.90	0.34	
(B) F_3 progenies segregating in 13:3 ratio					
1	16	3	0.11	0.74	$Wr_1\ wr_1\ Wr_2wr_2$
6	18	4	0.01	0.94	(4/16)
8	15	3	0.05	0.82	
14	17	5	0.23	0.64	
18	16	4	0.02	0.89	
Pooled ($n = 5$)	82	19	0.00	0.99	
(C) F_3 non-segregating resistant progenies					
5	18	0	0.00	1.00	$Wr_1Wr_1Wr_2Wr_2$, $Wr_1Wr_1wr_2wr_2$,
7	15	0	0.00	1.00	$Wr_1Wr_1Wr_2wr_2$, $wr_1wr_1wr_2wr_2$,
9	18	0	0.00	1.00	or
2	13	1	0.07	0.78	$Wr_1wr_1wr_2wr_2$
4	15	1	0.06	0.79	
11	17	1	0.06	0.81	(7/16)
19	18	1	0.05	0.82	
Pooled ($n = 7$)	114	4	0.21	0.65	
(D) Number of segregating and non-segregating wilt resistant F_3 progenies in 7:6 ratio					
Segregating	11		0.38	0.54	
Non-segregating	7				

( ) genotypic frequency

2051 × ICPL 20106 although the expected ratios fitted well, but with low probability as compared to other crosses. This could be due to the presence of modifier/minor genes present in the male parent and influence the expression of wilt controlling genes.

18 F_3 progenies derived from randomly selected wilt resistant F_2 plants of cross ICPA 2051 × ICPL 20116 were assessed for their intra-progeny segregation for disease incidence (Table 3). Based on segregation data, the progenies were classified into those (total 11 progenies) segregating for resistance and susceptibility; and those (total seven progenies) where all the segregants were resistant. The expected ratio between these two groups was seven segregating: six non-segregating types; and the χ^2 test showed a good fit ($p = 0.54$) to this ratio.

A close perusal of segregation patterns within the 11 segregating progenies (Table 3) showed the presence of two sub-groups. The first sub-group (five progenies) segregated like F_2 in a di-hybrid (13 resistance:3 susceptible) ratio; while the other sub-group (six progenies) was found segregating for a single gene in the ratio of 3 resistant:1 susceptible. Further, the proportion among 11 segregating progenies fit well to the expected ratio of 2 (segregating for 2 genes):1 (segregating for 1 gene). In the first sub-

Table 4 Wilt incidence in 15 experimental hybrids developed by crossing a susceptible CMS line (ICPA 2051) and wilt resistant fertility restorers in the sick nursery, 2009

Group	Parentage	Wilt[a] % in F_1 hybrids	Expected genotype of		
			Female parent	Male parent	F_1 hybrid
I	ICPA 2051 × MAL 17	100			
	ICPA 2051 × ICP 11440	88			
	ICPA 2051 × ICP 13384	100	$wr_1wr_1Wr_2$ Wr_2	$wr_1wr_1Wr_2$ Wr_2	$wr_1wr_1Wr_2$ Wr_2
	ICPA 2051 × ICP 9158	100	(susceptible)	(susceptible)	(susceptible)
	ICPA 2051 × ICP 12023	100			
II	ICPA 2051 × ICPL 87051	79			
	ICPA 2051 × ICPL 20105	64			
	ICPA 2051 × ICPL 96053	80	$wr_1wr_1Wr_2$ Wr_2	wr_1wr_1 wr_2 wr_2	$wr_1wr_1Wr_2$ wr_2
	ICPA 2051 × ICPL 20138	67	(susceptible)	(resistant)	(susceptible)
	ICPA 2051 × ICPL 20118	62			
III	ICPA 2051 × ICPL 20116	0	$wr_1wr_1Wr_2$ Wr_2	Wr_1Wr_1 wr_2 wr_2	$Wr_1wr_1Wr_2$ wr_2
	ICPA 2051 × ICPL 20110	2		Or	Or
	ICPA 2051 × ICPL 20177	1		Wr_1Wr_1 Wr_2 Wr_2	$Wr_1wr_1Wr_2$ Wr_2
	ICPA 2051 × ICPL 20136	3	(susceptible)	(resistant)	(resistant)
	ICPA 2051 × ICPL 20108	2			

[a] Disease score = susceptibility %, calculated by counting susceptible plants from total number of plants

group, both the resistance genes were present in heterozygous ($Wr_1wr_1Wr_2wr_2$) condition and hence, it segregated in a di-hybrid ratio (13 resistant:3 susceptible) with Wr_1 over-riding the effect of Wr_2 gene. In the second sub-group a single dominant gene was present in the heterozygous (Wr_1wr_1) form. The breeding materials derived from this study could be used to select homozygous lines with one Wr_1Wr_1 Wr_2Wr_2 or both $Wr_1Wr_1wr_2wr_2$ the resistance genes through progeny row testing. For a stable and long lasting genetic resistance, genotypes with multi-genic resistance genes are always preferred over monogenic control of the disease because the former is more broad based and hence more durable. In multi-genic resistance some interactions among major and/or modifying genes are frequent and the lines with dual resistance ($Wr_1Wr_1wr_2wr_2$) genes are expected to perform better and hold promise under diverse growing conditions.

Wilt incidence in the 15 new experimental hybrids developed by crossing a susceptible male-sterile line with resistant/susceptible fertility restorers, showed differential reactions to wilt incidence (Table 4). In group I hybrids, the wilt resistant alleles were absent in the male parents and they produced wilt susceptible hybrid combinations. On the contrary in group II hybrids, the male parents were wilt resistant, but all the resultant hybrids were susceptible. In this group of hybrids the resistance in the male parent was conferred by a pair of recessive alleles at locus 2; and when these were crossed to a susceptible female, they produced susceptible hybrids because the susceptible dominant allele was contributed to the hybrids by the female parent. The high level of wilt resistance recorded in group III hybrids was due to the contribution of a dominant wilt resistance allele from the male parents. Hence, the differences with respect to wilt incidence, observed between group II and III hybrids involving wilt susceptible x resistant crosses were attributed to the differences in the genetic constitution of their male parents.

Discussion

In comparison to other economic crops, studies on the inheritance of disease resistance in pigeonpea are limited. Pal (1934) was the first to investigate the genetics of wilt resistance in pigeonpea and reported a multiple genetic control. Shaw (1936) and Pathak (1970) reported two complementary genes conferring resistance to *Fusarium* wilt in pigeonpea. A single

dominant genetic control of wilt resistance was reported by Joshi (1957), Pawar and Mayee (1986), Pandey et al. (1996), and Kotresh et al. (2006). On the contrary, Jain and Reddy (1995) reported a single gene recessive control of *Fusarium* wilt. Odeny et al. (2009) studied genetics of resistance in an African (ICEAP 00040) and an Indian (ICP 8863) genotypes. They found that the wilt resistance in ICEAP 00040 was controlled by a single recessive gene, while in ICP 8863 two pairs of recessive genes governed the resistance. Karimi et al. (2010) observed that wilt resistance in two African cultivars was under the control of a single dominant gene. They also detected the presence of a recessive gene for resistance when a cross involving two susceptible lines KAT 60/8 and ICP 7035 was studied. Tekeoglu et al. (2000) reported that in chickpea the lines resistant to one race of *Fusarium* wilt were found to be susceptible to another race. Similarly in pigeonpea also, a wilt resistant line ICP 7035 that exhibited a high level of wilt resistance in Asia (Reddy et al. 1990) was highly susceptible to wilt in Africa (Karimi et al. 2010). Such events indicate the presence of different *Fusarium* variants in the two continents. To understand further the nature of resistance genes, studies with known races/variants will be required for any long term solution of this disease.

The present study showed that resistance to *Fusarium* wilt was due to the presence of one dominant and one recessive gene with epistatic inhibitor effect. To confirm these results at molecular level, QTL mapping of the populations segregating for *Fusarium* wilt and the corresponding genotypic data is warranted. The random amplified polymorphic DNA (RAPD), simple sequence repeat (SSR), diversity array technology (DArT), and single nucleotide polymorphic (SNP) markers in pigeonpea are now available (Varshney et al. 2010) to support a marker-based hybrid breeding program. Recently, by using DArT markers a reference genetic linkage map has also been developed (Yang et al. 2011) in pigeonpea. These markers could be used to genotype F_2 and F_2-derived populations from the four crosses used in this study to perform marker-trait associations. The markers linked to *Fusarium* wilt QTL (gene/s) could also be used to facilitate the introgression of wilt resistance through marker-assisted back crossing.

In order to break the age old yield barrier in pigeonpea, a commercially viable hybrid breeding technology based on CMS system (Saxena et al. 2005) was developed at ICRISAT (Saxena 2009). Yield advantages of these hybrids have been demonstrated in over 2,000 on-farm trials in five states of India (Saxena and Nadarajan 2010) and Myanmar (Kyu et al. 2011). To take full advantage of this technology it is necessary that the hybrids, besides high yields, have high levels of resistance to major diseases. To achieve this it is imperative to have quality hybrid parents with respect to combining ability, disease resistance, and market-preferred traits.

The nature of wilt resistance genes is expected to have a significant influence on breeding high yielding hybrids. In cases where the resistance to *Fusarium* wilt is controlled by recessive gene(s), the pre-requisite for breeding wilt resistant hybrids will be to introgress the resistance genes in all the three (A, B, R) hybrid parents. Breeding of such hybrid parents is cumbersome and will consume more time and resources. The present inheritance study indicated that a single dominant or a pair of recessive gene governed the resistance to *Fusarium* wilt with dominance epistatic effects. The hybrid breeding programs can be benefitted by incorporating dominant wilt resistance gene in the female parents. The availability of such A-lines will enhance the scope of breeding high yielding wilt resistant hybrids because the crosses made with either resistant or susceptible male parents will always produce resistant hybrids (Table 4). Since hybrid breeding is considered a number game, the availability of wilt resistant male-sterile lines with dominant genes will allow synthesis of a greater number of wilt resistant hybrids each year; thus offering greater probability of success in breeding high yielding wilt resistant pigeonpea hybrids.

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