Eur J Plant Pathol (2012) 133:707–714
DOI 10.1007/s10658-012-9949-9

New sources of resistance to Fusarium wilt and sterility mosaic disease in a mini-core collection of pigeonpea germplasm

Mamta Sharma · Abhishek Rathore · U. Naga Mangala · Raju Ghosh · Shivali Sharma · HD Upadhyay · Suresh Pande

Accepted: 23 January 2012 / Published online: 3 February 2012

Abstract Fusarium wilt (FW) and Sterility mosaic disease (SMD) are important biotic constraints to pigeonpea production worldwide. Host plant resistance is the most durable and economical way to manage these diseases. A pigeonpea mini-core collection consisting of 146 germplasm accessions developed from a core collection of 1290 accessions from 53 countries was evaluated to identify sources of resistance to FW and SMD under artificial field epiphytotic conditions during 2007–08 and 2008–09 crop seasons. Resistant sources identified in the field were confirmed in the greenhouse using a root dip screening technique for FW and a leaf stapling technique for SMD. Six accessions (originated from India and Italy were found resistant to FW (<10% mean disease incidence). High level of resistance to SMD was found in 24 accessions (mean incidence <10%). These SMD resistant accessions originated from India, Italy, Kenya, Nepal, Nigeria, Philippines and United Kingdom. Combined resistance to FW and SMD was found in five accessions (ICPs 6739, 8860, 11015, 13304 and 14819). These diverse accessions that are resistant to FW or SMD will be useful to the pigeonpea resistance breeding program.

M. Sharma (✉) · A. Rathore · U. N. Mangala · R. Ghosh · S. Sharma · H. Upadhyay · S. Pande
International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru 502 324, Greater Hyderabad, Andhra, Pradesh, India
e-mail: mamta.sharma@cgiar.org

Keywords Host plant resistance · Mini-core · Pigeonpea · Fusarium wilt and Sterility mosaic disease

Introduction

Pigeonpea [*Cajanus cajan* (L.) Millsp.] is the most versatile grain legume crop grown in the semi-arid tropical and subtropical regions between 25º N and 30º S in Asia, Africa, and America (Van der Maesen 1990). Globally, pigeonpea is cultivated in about 4.5 million ha, adding 3.48 million tonnes of grain to global food production (FAOSTAT 2009). India is a major pigeonpea producer in the world with a contribution of 75–80 per cent. Pigeonpea is a major source of protein; enriches soil; provides fodder and fuel wood; and it is beneficial for arresting soil erosion (Ae et al. 1990; Saxena et al. 2002). Pigeonpea crop has a direct bearing on the economic and financial well-being and on the nutritional status of the subsistence farmers in the subcontinent as it is a low-input, rainfed crop and provides economic returns from each and every part of the plant. However, average yields of pigeonpea are low (450–670 kg per ha) and diseases are the major constraints to the high yield potential of pigeonpea cultivars. Among diseases, Fusarium wilt and sterility mosaic diseases are the major constraints to pigeonpea production worldwide. This is a matter of concern since the domestic demand of pigeonpea is rapidly increasing.

Fusarium wilt (FW), caused by *Fusarium udum* Butler, is the major constraint for limiting pigeonpea

production in all pigeonpea growing regions (Jain and Reddy 1995; Gwata et al. 2006). The disease symptoms usually appear when plants are at the pre- flowering and podding stage (100% loss), at maturity (67%), and at pre-harvest stage (30% loss) but sometimes symptoms also appear in 1–2 month-old plants. The FW incidence increases in the ratoon and perennial crops (Reddy et al.1993) and causes serious yield losses in susceptible cultivars. In India alone, the annual yield loss due to this disease has been estimated at US $ 71 million (Kannaiyan et al. 1984; Reddy et al. 1993). Sterility mosaic disease (SMD), often referred to as "Green Plague", as the affected plants are green with excessive vegetative growth but with no flowers or pods, under congenial conditions spreads rapidly leading to severe epidemics (Singh et al. 1999). SMD infection at an early stage (<45-day-old plants) results in a 95–100% loss in yield (Reddy et al. 1990). Precise data on the impact of SMD and its socio-economic importance are limited, but in assessing the economic importance of various biotic problems of pigeonpea, SMD causes greater yield losses than any other disease affecting pigeonpea in Indian subcontinent. Yield losses due to SMD were estimated at 205, 000 t of grain valued at US $ 76 million annually (Kannaiyan et al. 1984) in India and Nepal in 1993, losses were US$280 million (Reddy et al. 1993). The disease is confined to Asia and apart from India it has been reported from Nepal, Bangladesh and Myanmar, Thailand and Sri Lanka (Nene and Sheila 1990). SMD is caused by Pigeonpea Sterility mosaic virus and is transmitted by an eriophyid mite (*Aceria cajani*) (Kumar et al. 2000).

The primary management options to minimize yield losses due to these diseases are by developing cultivars resistant to FW and SMD. There are only few sources of resistance reported to FW and SMD (Nene et al. 1989; Khare et al. 1994). As a result, the search continues for sources of higher levels of resistance for these diseases at ICRISAT, Patancheru. Further, most of the pigeonpea cultivars grown to date are selections from the landraces with a narrow genetic base (Singh et al. 1990). The prime reasons for the low use of diverse germplasm for improvement of quantitative traits in pigeonpea plant breeding program is the extended time and high costs involved in identifying these useful accessions (Goodman 1990). To overcome the need for large-scale evaluation of germplasm collections against various biotic and abiotic stresses, Frankel (1984) proposed the concept of a core collection which is 10% of an entire collection representing most of the diversity of the species. From 12370 pigeonpea accessions available at the ICRISAT Centre, Patancheru, India, a core collection consisting of 1290 accessions from 53 countries was constituted (Reddy et al. 2005), which is still large for the systematic evaluation of traits of economic importance, such as disease resistance. Hence, Upadhyaya et al. (2006) developed a mini-core collection of pigeonpea, comprising of 146 accessions (about 10% of core collection and 1% of entire collection) representing almost the entire spectrum of diversity. The mini-core of 146 accessions of pigeonpea was developed based on evaluation of 18 qualitative traits and 16 quantitative traits of core collection of the 1290 accessions of pigeonpea at the ICRISAT research farm, Patancheru, India, (Upadhyaya et al. 2006). Due to its greatly reduced size, the mini-core subset provides a more economical starting point for proper exploitation of pigeonpea genetic resources for crop improvement. The objective of this study was to evaluate the pigeonpea mini-core set to identify resistance to FW and SMD that could be utilized in pigeonpea disease resistance breeding program.

Materials and methods

Seed source

Seed of the 146 germplasm accessions of the pigeonpea mini-core collection were obtained from the Genetic Resource Division, ICRISAT, Patancheru, India, based on evaluation of the core collection of 1290 accessions of pigeonpea at the ICRISAT research farm, Patancheru, India for 18 qualitative traits and 16 quantitative traits (Upadhyaya et al. 2006). The data for various morphological and agronomic traits indicated that almost entire genetic variation and a majority of co-adapted gene complexes present in the core subset were preserved in the mini-core subset.

The mini-core collection includes 144 accessions from 25 countries and two accessions from unknown places (www.icrisat.org). The composition of the mini-core subset reflected the predominance of accessions from southern India, Sri Lanka, and the Maldives (34.7%), followed by accessions from northwestern India, Pakistan, and Iran (16.7%), Bangladesh, Myanmar, Nepal, China, Taiwan, north eastern India (11.8%), and central India (11.8%). About 8.3% accessions in the mini-core were from

Table 1 Analysis of variance (covariance parameter estimates) for Fusarium wilt incidence in the mini-core accessions of pigeonpea

Sources of variation	Estimate (variance component)	Standard error	Probability (pr>z)
Combined year (pooled data)			
Year	–	–	0.0667 (pr>f)
Accession	1.9802	0.2871	<0.0001
Year x Accession	0.3824	0.1109	0.0006
First year			
Replication	0.005370	0.008027	0.2518
Accession	0.05784	0.009758	<.0001
Second year			
Replication	0.001737	0.002655	0.2565
Entry	0.07166	0.009646	<.0001

southern and eastern Africa, and 3.5% each from western and central Africa, and unknown Indian states (Upadhyaya et al. 2006). Seeds of all the accessions used as susceptible checks for different diseases were obtained from the Department of Legumes Pathology, ICRISAT, Patancheru, India.

Evaluation for Fusarium wilt resistance

The pigeonpea mini-core accessions were evaluated in the pigeonpea wilt sick plot under artificial epiphytotic conditions at ICRISAT, Patancheru. A threshold level of the wilt pathogen *Fusarium udum* was maintained by incorporating chopped wilted pigeonpea plants in the sick plot every year. The field trials were conducted for the two different crop seasons in 2007–08 and 2008–09. Each accession was planted in two rows of 2-m length with seed to seed spacing of 15 cm and row to row spacing of 40 cm. A highly wilt susceptible cultivar ICP 2376 was included between every 10 test rows to serve as an indicator/infector rows. The trial was conducted in randomized complete block design (RCBD) with two replications. Periodical wilt incidence was recorded at seedling, flowering and pod formation stages.

Resistance to FW found in the wilt sick plot was confirmed in the greenhouse using the root dip inoculation technique (Nene et al. 1981). Resistant mini-core accessions along with the wilt susceptible ICP 2376 were raised in polythene bags filled with sterilised river sand in a greenhouse maintained at 25±3°C for 8 days. Inoculum was prepared from a single conidial culture of *F. udum* isolated from wilt infected plants collected from the ICRISAT wilt sick plot. For mass inoculum preparation, a 7-mm disc of actively growing *F. udum* culture was put into a 250 ml conical flask containing 100 ml of sterilized potato dextrose broth and incubated for 7 days in an incubator shaker at 25±1°C and 125 rpm. The culture was then

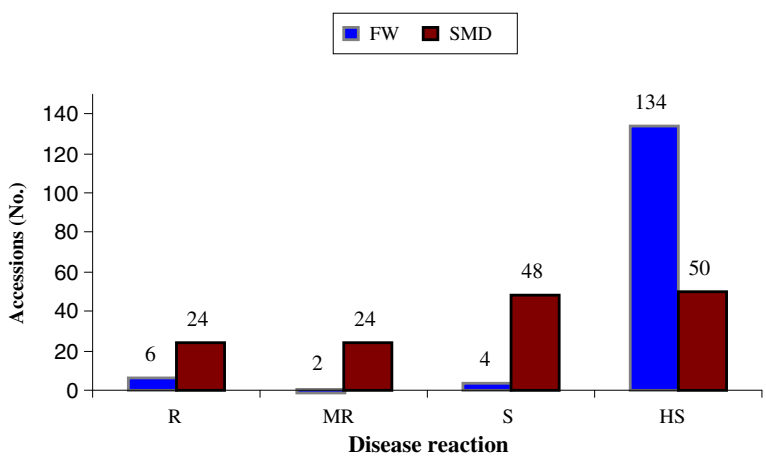

Fig. 1 Frequency distribution of Fusarium wilt and sterility mosaic diseases in pigeonpea mini core accessions (Disease reaction to FW and SMD is based on the mean of 2 years data where, R (resistant) =1% to 10% mortality, MR (moderately resistant) =11% to 20% mortality, S (susceptible) =21% to 50% mortality, and HS (highly susceptible) = >50% mortality

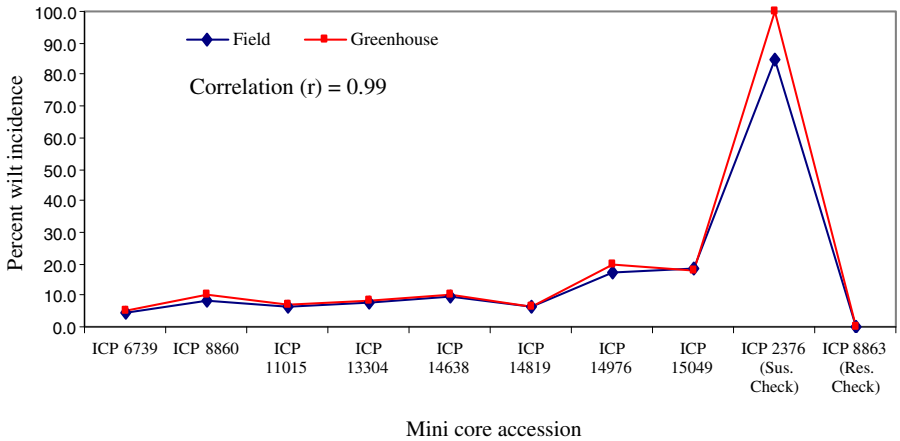

Fig. 2 Comparison and correlation of Fusarium wilt reaction in few mini core accessions in field and greenhouse

homogenized in sterilized distilled water and adjusted to 6×10^5 conidia ml^{-1} with a haemocytometer for use as an inoculum. Eight-day-old seedlings of each test line as well as susceptible cultivar grown in sterilized river sand were uprooted, cleaned with tap water and root inoculated by dipping in inoculum suspension for 1–2 min to enable conidia to adhere to the roots. Inoculated seedlings were transplanted in pre-irrigated sterile vertisol and sand (3:1) in pots and incubated in a greenhouse at 25±3°C. Thirty seedlings of each line were tested in three replications (10 seedlings/pot) in a completely randomized design (CRD). Disease incidence was recorded for 60 days after inoculation.

Evaluation for sterility mosaic disease resistance

Each of the 146 pigeonpea mini-core accessions was evaluated for SMD in the Pigeonpea sterility mosaic disease sick plot during the 2007–08 and 2008–09 crop seasons at ICRISAT, Patancheru. Each and every plant of the test entries was inoculated at the two leaf stage with SMD infested leaves using the leaf staple technique (Nene et al. 1981). The SMD infected leaflet (maintained on the susceptible cultivar ICP 8863 in isolated pigeonpea sterility mosaic disease nursery at ICRISAT) was taken and folded on the primary leaf (at the seedling two-leaf stage) in such a way that its lower surface came in contact with a primary leaf of the test seedling and was then stapled with a small paper stapler for successful SMD infection. Each accession was planted in two rows of 2-m length with seed-to-seed spacing of 15 cm and row-to row-spacing of 40 cm. A SMD susceptible cultivar ICP 8863 was included between every 10 test rows to serve as an indicator/infector row. The trial was conducted in randomized complete block design (RCBD) with two replications. The susceptible cultivar ICP 8863 was planted in the sick plot 1 month in advance of the regular planting time to serve as an infector rows in order to have a good source of virus inoculum. The planting of the test and

Table 2 Analysis of variance (covariance parameter estimates) for sterility mosaic disease incidence in the mini-core accessions of pigeonpea

Sources of variation	Estimate (Variance component)	Standard error	Probability (pr>z)
Combined year			
Year	–	–	0.0036 (pr>f)
Entry	4.0928	0.5366	<0.0001
Year x Accession	0.4641	0.1198	0.0001
First year			
Replication	−0.00007	0.000130	0.6098
Entry	0.04753	0.007035	<0.0001
Second year			
Replication	−9.9E-6	0.000050	0.8420
Entry	0.04694	0.005875	<0.0001

susceptible cultivars was done across the wind direction in the field to facilitate virus transmission through mites. The accessions found resistant to SMD under field conditions were tested in the greenhouse using the leaf staple technique. Resistant accessions were screened in the pots in three replications in a CRD and in each replication five plants were maintained. The procedure for inoculation was similar as explained above.

Statistical analysis

Data on disease incidence (FW and SMD) was collected from each replication in the randomized experimental block in the field experiments during 2007 and 2008. Data was also collected from greenhouse screening evaluated for all the resistant and moderately resistant accessions. The arcsin transformation (Gomez and Gomez 1984) was applied for percent FW and SMD during the years 2007 and 2008. For combining data across 2 years, Bartlett's test for homogeneity of error variance was done and found significant. Data was transformed to make the error variance homogeneous. Analysis of Variance (ANOVA) was carried out to determine the effect of year (E), accessions (G) and their interaction (G×E) considering year and replication as fixed and accessions as random using the proc *mixed* procedure of SAS software version 9.2 for Windows (SAS Institute Inc. 2008. SAS/STAT® 9.2 User's Guide. Cary, NC: SAS Institute Inc). Since G×E effect was found significant, data on disease incidence were analyzed separately for each year using ANOVA and best linear unbiased predictors (BLUP) for accessions were estimated.

Results

Fusarium wilt resistance

The analysis of variance (ANOVA) showed significant ($P<0.0001$) variation among the 146 mini-core accessions for FW resistance in both the years of evaluation as well as in pooled data of both the years (Table 1). There was no significant effect of year on disease incidence, indicating that differences in the wilt disease incidence were mainly contributed by accessions. Based on the mean disease incidence for 2 years, of the 146 pigeonpea mini-core accessions evaluated for FW resistance, six accessions (ICP's 6739, 8860, 11015, 13304, 14819 and 14638) were found resistant (≤ 10% incidence) and two accessions (ICP 14976 and ICP 15049) moderately resistant (11–20% incidence) to FW (Table 3). Most of the mini-core accessions (91.2%) showed susceptible reactions in both years of evaluation (Fig. 1). Wilt incidence was 100% in the susceptible check ICP 2376. Of the six resistant accessions, five originated from India

Table 3 Origin, identity and disease incidence of resistant accessions to Fusarium wilt and sterility mosaic disease in the pigeonpea mini-core collections during 2007–08 and 2008–09

Accession No.	Origin	Disease incidence (%)		
		2007–08	2008–09	Pooled
Fusarium wilt				
ICP 6739	India	8.05	0.50	4.27
ICP 8860	ICRISAT, India	6.90	9.85	8.37
ICP 11015	ICRISAT, India	6.75	6.45	6.60
ICP 13304	Italy	9.85	5.50	7.67
ICP 14638	ICRISAT, India	9.90	9.50	9.70
ICP 14819	ICRISAT, India	6.85	6.25	6.55
Sterility mosaic disease				
ICP 3576	India	1.10	1.20	1.15
ICP 6739	India	5.00	5.05	5.02
ICP 6845	India	11.60	8.25	9.92
ICP 7869	India	0.00	0.00	0.00
ICP 8152	India	3.35	3.90	3.62
ICP 8860	ICRISAT, India	7.70	7.60	7.65
ICP 9045	India	0.00	0.00	0.00
ICP 11015	ICRISAT, India	0.00	0.00	0.00
ICP 11059	ICRISAT, India	0.00	0.60	0.30
ICP 11230	ICRISAT, India	0.00	0.00	0.00
ICP 11281	ICRISAT, India	0.00	0.00	0.00
ICP 11320	Nepal	9.80	8.75	9.27
ICP 11321	Nepal	3.25	2.30	2.77
ICP 11823	India	4.60	3.85	4.22
ICP 11910	India	0.00	0.00	0.00
ICP 12410	India	3.90	4.80	4.35
ICP 13167	Kenya	2.85	2.35	2.60
ICP 13304	Italy	8.00	6.95	7.47
ICP 13579	Philippines	6.05	5.95	6.00
ICP 13633	Nigeria	6.00	6.25	6.12
ICP 14819	ICRISAT, India	0.00	0.60	0.30
ICP 14976[a]	ICRISAT, India	0.00	0.00	0.00
ICP 15049[a]	ICRISAT, India	1.60	1.90	1.75
ICP 15185	ICRISAT, India	5.55	5.10	5.32

[a] Accessions were moderately resistant to wilt

(ICP's 6739, 8860, 11015, 14819 and 14638) and one from Italy (ICP 13304). Both of the moderately resistant accessions originated from India. All the accessions found resistant in the field showed resistant reactions in the greenhouse indicating very high positive correlation (r=0.99, Fig. 2) between field and greenhouse ratings and the error variance was homogeneous across groups.

Sterility mosaic disease resistance

Based on the mean of the disease incidence in both years, 17.12% accessions were found resistant, 16.43% moderately resistant, 33.78% susceptible and 33.78% highly susceptible to SMD (Fig. 1.). Among the 24 resistant accessions, seven accessions (ICP's 7869, 9045, 11015, 11230, 11281, 11910 and 14976) were found free from SMD (0% incidence). The ANOVA showed significant ($P<0.0001$) variation among the 146 mini-core accessions for SMD resistance in both the years of evaluation as well as in pooled data (Table 2). Analysis indicates that differences in the SMD disease incidence were mainly contributed by accessions, since the effect of year was not significant. Of the 24 resistant accessions, 18 accessions originated from India, two from Nepal, and one each from Italy, Kenya, Nigeria, United Kingdom and Philippines (Table 3). Among the 24 moderately resistant accessions, 16 originated from India, two from Tanzania and Kenya and one each from Ghana, Nigeria, Italy and Thailand. All the resistant and moderately resistant accessions were reconfirmed by re-evaluating them in greenhouse screening and showed very high positive correlation (r=0.98, Fig. 3) between field and greenhouse screenings. The error variance was homogeneous across groups.

Combined disease resistance

Combined resistance to FW and SMD (< 10% disease incidence) was found in five accessions, ICP's 6739, 8860, 11015, 13304 and 14819, four of which originated from India and one from Italy (Table 3). Two accessions ICP 14976 and 15049 showed moderately resistant reaction to FW and resistant reaction to SMD (Table 3).

Discussion

Extensive evaluation of an entire germplasm for particular characteristic is difficult and also time consuming. Thus, the concept of core collections that represent a large number of accessions in a germplasm has been put forward to represent the diversity of an entire germplasm (Frankel 1984). A mini-core subset that represents the variation present in the germplasm, with fewer accessions, provides an easy approach for accessing genetic resources. In this study, we evaluated 146 mini-core accessions that comprised 1.2% of the entire germplasm collection and 11.3% of the core collection and represented the total diversity contained in the entire collection of pigeonpea to identify resistance to two economically important diseases of pigeonpea FW and SMD.

We identified six mini-core accessions originating from India and Italy highly resistant to FW both in the wilt-sick plot and greenhouse screening. A very high positive correlation was found between field and greenhouse screening techniques. A considerable effort has been made by ICRISAT in developing wilt-resistant pigeonpeas, adapted to cultivation in the Asia and Africa (Nene and Sheila 1990; Reddy et al. 1998). Gwata et al. (2006) evaluated

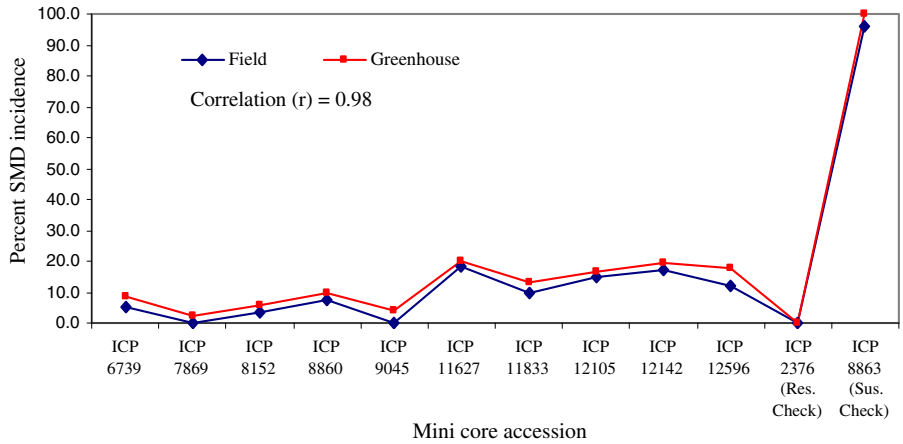

Fig. 3 Comparison and correlation of sterility mosaic disease (SMD) reaction in few mini core accessions in field and greenhouse

the new elite pigeonpea germplasm for FW resistance in three different countries during the 2001–2002 cropping season using wilt-sick plots. The genotype ICEAP 00040 consistently showed a high level of resistance to the disease in the three countries Kenya, Malawi and Tanzania. Resistance to SMD was also identified in mini-core collection of pigeonpea. Seven accessions were found free from SMD (0% incidence) in both years of evaluation and 17 accessions had a very high level of resistance (<10% incidence). These SMD asymptomatic and highly resistant lines originated from different countries representing a high level of genetic diversity in resistance. The G×E interaction was found to be significant in the pooled data for both the diseases; hence the data were analyzed separately for each year. Since the effect of year was not significant, it appears that differences in the disease reaction was due to the genotypic effect in the accessions. Several resistant pigeonpea accessions to SMD have been reported by various workers in previous studies (Khare et al. 1994; Rangaswamy et al. 2005). However, based on the results of pigeonpea multilocation trials, levels of virulence varied and reports of breakdown in resistance have been reported (Nene et al. 1989). Broad based resistance to SMD in wild species *Cajanus scarabaeoides* have been reported to mild and severe strains of PPSMV by Kulkarni et al. (2003).

The information presented in this paper will be of great value to plant breeders in their efforts to develop resistance breeding programs for pigeonpea. There are several reports where mini-core collections have successfully been used to identify resistance sources for diseases in crops like chickpea (Pande et al. 2006) and sorghum (Sharma et al. 2010). Thus, the mini-core collection can be used very effectively as a starting point for research involving screening of the germplasm collection for desirable traits in pigeonpea. Identification of mini-core accessions with resistance against a select combination of two diseases also would permit use of diverse sources for future breeding efforts and ensure a better chance of success in improving the disease resistance of pigeonpea.

References

Ae, N., Arihara, J., Okada, K., Yoshihara, T., & Johansen, C. (1990). Phosphorus uptake by pigeonpea and its role in cropping system of the Indian subcontinent. *Current Science, 248*, 477–480.

FAO STAT (2009). Food and Agriculture organization of the United Nations, Rome. http://faostat.fao.org/

Frankel, O. H. (1984). Genetic perspectives of germplasm conservation. In W. K. Arber (Ed.), *Genetic manipulation: impact on man and society* (pp. 161–170). Cambridge: Cambridge University Press.

Gomez, & Gomez. (1984). *Statistical procedure for agricultural research* (2nd ed., pp. 306–308). New York: Wiley.

Goodman, M. M. (1990). Genetic and germplasm stocks worth conserving. *Journal of Hereditary, 81*, 11–16.

Gwata, E. T., Silim, S. N., & Mgonja, M. (2006). Impact of a new source of resistance to Fusarium Wilt in Pigeonpea. *Journal of Phytopathology, 154*, 62–64.

Jain, K. C., & Reddy, M. V. (1995). Inheritance of resistance to fusarium wilt in pigeonpea *(cajanus cajan* (l.) millsp.). *Indian Journal of Genetics, 55*, 434–437.

Kannaiyan, J., Nene, Y. L., Reddy, M. V., Ryan, J. G., & Raju, T. N. (1984). Prevalence of pigeonpea diseases and associated crop losses in India, Africa, and the Americas. *Tropical Pest Management, 30*, 62–71.

Khare, D., Satpute, R. G., & Tiwari, A. S. (1994). Present state of the wilt and sterility mosaic diseases of pigeonpea. *Indian Journal of Genetics, 54*(4), 331–346.

Kulkarni, N. K., Kumar, P. L., Muniyappa, V., Jones, A. T., & Reddy, D. V. R. (2003). Broad-based resistance to pigeonpea sterility mosaic disease in the accessions of *Cajanus scarabaeoides*. *Indian Journal of Plant Protection, 31*, 6–11.

Kumar, P. L., Jones, A. T., Sreenivasulu, P., & Reddy, D. V. R. (2000). Breakthrough in the identification of the causal agent of pigeonpea sterility mosaic disease. *Journal of Mycology and Plant Pathology, 30*, 249.

Nene, Y. L., & Sheila, V. K. (1990). Pigeonpea: geography and importance. In Y. L. Nene, S. D. Hall, & V. K. Sheila (Eds.), *The pigeonpea* (pp. 1–14). Wallingford: CAB International.

Nene YL, Kannaiyan J, Reddy MV (1981) Pigeonpea diseases: Resistance-screening techniques. Information bulletin no. 9. International Crops Research Institute for the Semi Arid Tropics, Patancheru 502 324, AP, India

Nene, Y. L., Reddy, M. V., Beniwal, S. P. S., Mahmood, M., Zote, K. K., Singh, R. N., & Sivaprakasam, K. (1989). Multilocational testing of pigeonpea for broad based resistance. *Indian Phytopathology, 42*, 44–48.

Pande, S., Kishore, G. K., Upadhayaya, H. D., & Rao, J. N. (2006). Identification of sources of multiple disease resistance in mini- core collection of chickpea. *Plant Disease, 90*, 1214–1218.

Rangaswamy, K. T., Muniyappal, V., Kumar, P. L., Saxena, K. B., Byregowda, M., Raghavendra, N., Pandurangaiah, K., Kumar, R. V., Waliyar, F., & Jones, A. T. (2005). ICP 7035 – a sterility mosaic resistant vegetable and grain purpose pigeonpea variety. *SAT e Journal, 1*, 1–3.

Reddy MV, Nene YL, Singh G, Bashir M (1990) Strategies for management of foliar diseases of chickpea. Chickpea in the nineties: proceedings of second international workshop chickpea improvement (pp 117–127). (International Crops Research Institute for the Semi Arid Tropics, Patancheru 502 324, AP, India)

Reddy, M. V., Raju, T. N., Sharma, S. B., Nene, Y. L. & Mc Donald, D. (1993). Handbook of pigeonpea diseases.

Information bulletin no. 42, ICRISAT, Hyderabad, India (ISBN 92-9066-277-8).

Reddy, M. V., Raju, T. N., & Lenne, J. M. (1998). Diseases of pigeonpea. In D. J. Allen & J. M. Lenne (Eds.), *The pathology of food and posture legumes* (pp. 517–558). CAB International: Wallingford.

Reddy, L. J., Upadhyaya, H. D., Gowda, C. L. L., & Singh, S. (2005). Development of core collection in pigeonpea [*Cajanus cajan* (L.) Millspaugh] using geographic and qualitative morphological descriptors. *Genetic Resources and Crop Evolution, 52*, 1049–1056.

Saxena, K. B., Kumar, R. V., & Rao, P. V. (2002). Pigeonpea nutrition and its improvement. *Journal of Crop Production, 5*, 227–260.

Sharma, R., Rao, V. P., Upadhyaya, H. D., Reddy, V. G., & Thakur, R. P. (2010). Resistance to grain mold and downy mildew in a mini- core collection of sorghum germplasm. *Plant Disease, 94*, 439–444.

Singh, L., Gupta, S. C., & Faris, D. G. (1990). Pigeonpea: breeding. In Y. L. Nene et al. (Eds.), *The pigeonpea* (pp. 375–399). Cambridge: CAB International, Univ. Press.

Singh, A. K., Rathi, Y. P. S., & Agarwal, K. C. (1999). Sterility mosaic of pigeonpea: A challenge of the 20th century. *Indian Journal of Virology, 15*, 85–92.

Upadhyaya, H. D., Reddy, L. J., Gowda, C. L. L., Reddy, K. N., & Singh, S. (2006). Development of a mini core subset for enhanced and diversified utilization of pigeonpea germplasm resources. *Crop Science, 46*, 2127–2132.

Van der Maesen, L. J. G. (1990). Pigeonpea: origin, history, evolution and taxonomy. In Y. L. Nene, S. D. Hall, & V. K. Sheila (Eds.), *The pigeonpea* (pp. 15–46). Wallingford: CAB International.