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# PRESENT STATE OF THE WILT AND STERILITY MOSAIC DISEASES OF PIGEONPEA

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## ABSTRACT

**Fusarium wilt and sterility mosaic virus (?) are the two most important diseases of pigeonpea, causing considerable yield losses to the crop. Various aspects relating to these diseases, i.e. mode of transmission, pathogenic races, genetics, screening for resistance, breeding methodology and disease control measures using cultural practices and resistant varieties have been reviewed. Latest information of significant use to the geniticists, breeders and plant pathologists, particularly for development of resistant varieties has been compiled.**

**Key words :** Fusarium, sterility mosaic virus, pigeonpea.

Wilt, caused by the fungus *Fusarium udum* [1], and sterility mosaic (SM) (virus ?) [2] are the two economically most important diseases of pigeonpea (*Cajanus cajan* (L.) Millsp.) in India and other pigeonpea growing countries of the world. Wilt and SM are reported to cause an annual monetory loss of 36 and 76 million US dollars, respectively, in India alone [3]. Fusarium wilt is a soil-borne disease, mainly transmitted through plant debris. The plants initially turn pale yellow and gradually die completely. Its expression in plants is seen at pre-flowering and podding stages (100% loss), at maturity (67% loss), and at pre-harvest stage (30% loss) [4]. Its incidence increases in the ratoon and perennial crops [5]. Sterility mosaic, a viral disease, has characteristic symptoms of stunted growth, bushy and pale appearance, mild mottled ring spots on leaflets, and the plants may become partially or wholly sterile. The SM virus is transmitted by eriophyid mite (*Aceria cajani* Channabasavanna) [6], nematodes [7], and through grafting [8].

Different species of *Fusarium* have been isolated by Padwick [9], and the number of strains were reported to be 4 [10], 5 [11], 7 [12] and 8 [13]. Sterility mosaic virus has distinct strains because its pathogenicity differs in different localities [14–16].

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Pigeonpea genotypes show different levels of wilt resistance under field conditions. It has been reported to be controlled by multiple factors without linkage with any distinct morphological characters [17], by two complementary genes with recessive and dominant expression [18], by duplicate genes [19], by a single dominant gene [20], and by a major dominant gene for resistance and minor polygenes for field resistance in the susceptible cultivars [21]. A gene for wilt resistance was transferred from *Atylosia* to *Cajanus* by Kumar [22]. Resistance for SM is governed by four independent nonallelic genes with at least one dominant and one recessive gene for resistance [23]. Multiple allelism with dominance of tolerance over resistance has also been reported [24]. Screening for wilt resistance is done by observing the reaction after inoculation with different races in different cultivars of pigeonpea [25], through inoculation by means of longitudinal cut in the stem 10–15 cm above ground level 45, 75 and 127 days after planting [26], through soil with the inoculum added in pots [27], by incorporation of wilted plant debris in the soil, by cultivation of susceptible cultivars continuously for 2–3 seasons [27], using the green house technique with multiplication of *Fusarium* on sand (90 g) and pigeonpea meal (10 g) media [28], and by electric current infection [29]. Hill sowing is more economical for evaluating large numbers of genotypes for wilt resistance than row sowing as the requirement for area is reduced to 20% [30]. The infectious nature of SM is screened through graft transmission and sap inoculation [31], stapling of diseased leaflets on the primary leaf of the test seedlings [32], planting susceptible cultivars 4–6 months in advance of the test material (spreader row) and inoculation in the spreader rows with the leaf staple technique [33]. The modified infector row technique with four-row hedge of susceptible cultivar infected by the leaf stapling technique on the west side is sufficient for a 2 ha field [34]. The degree of SM infection is directly proportional to the number of infected leaflets used and negatively affected by seedling age; 2–9 leaflets infested with 50–131 SM virus carrying mites are sufficient for staple on the healthy leaflets [35].

Breeding for pigeonpea wilt resistance was started in 1923 at Pusa [36]. The progress in establishing wilt resistant lines from hybridization has been very slow mainly due to lack of purity and uniformity of the resistant parents, complex nature of inheritance based on multiple alleles and high degree of variability for virulence in the wilt pathogen [37]. Healthy plants of local cultivars were screened in a wilt sick plot and selections of healthy and vigorous plants were made at late harvesting stage. The produce of these plants was bulked, again screened, and evaluated for seed yield. Finally resistance was confirmed by changing the direction of rows at right angle [38]. The breeding material developed through hybridization (straight single crosses and back crosses) was screened for both SM and wilt resistance, and the selected populations were further evaluated through bulk breeding method for resistance (Table 1) [39, 40].

Breeding for SM resistance mainly involves pedigree method after hybridization with resistant parents [37]. On the basis of disease incidence, selection starts $F_3$ onward. All the selected plants are selfed and harvested separately. Single plant progeny rows showing high

**Table 1. Pigeonpea breeding materials resistant to wilt, sterility mosaic, and both diseases**

| Wilt | Sterility mosaic | Combined resistance |
|---|---|---|
| T 16, T 51, T 80 [101] CP 80, C 38-3-1 [102] NP 41, NP 69 [103] Yadgir, No. 3 C 11 [104] C 28, C 36 [105] NP (WR) 15, 16, 18, 42 [36] Borill, Tuljapur 455 Latur 466-1, 476-11, DT 230 [106], Osmanabad, NP (WR) 19, NP 69, S 103, Balapur 10, P 1005, Paras 5, Javud [107, 108], C O2 [103], Kanke 9, 3 [109], C O3, 518 [110], H 76-44, 76-51, 76-65, Prabhat, ICPL 81, ICPL 87, TT 5, TT 6 [111], ICP 8863 (Maruti) ICP 7366 and BDN 1 [41] BDN 2 [112], ICPL 227, ICPl 8357, DA 12 [113], ICP 8863, ICP 10957, 10958, 11290, 11292, 11294 [114], GAUT 82-9, 82-74 [57], BP 1809 [115], JJAL 27 [124]. | ICP 3783, 6986, 6997 7035, HY 3C [116], TT 87, Bahar [117], ICP 4782 [118], DA 11, 13, 15, MA 165, 166, PDA 2, PDA 10 [119], ICPL 4, 8306, 86, MA 97, 128-2, PDA 84-2, ICPL 366, PDA 3, 7, 9 [120] ICPL 176, 288, 8308, 8324, 8074, 84077, 146, MA 166, 167 [121], ICP 10976, 8862 [122], ICPL 269, 8327, Pant A, 8505, 8507, 8508, Bhavanagar 1, NPRR 1, DDPA 84-66-1, 86-61, 84-8-3, ICPL 146, Sehore 367 [113], 134 B, 124 $B_2$ [123] JJAL 13, 24 [124]. | ICPL 335, 8362 7035, 4769 [39], AL 1, ICPL 267 [111, 120]. JJAL 11, 15, 22, 26, 28, 29 [124] |

level of resistance for 2–3 years are evaluated for yield in progeny rows and seeds are maintained by selfing. Pure lines resistant for either of the two diseases were involved in crossing programme [41]. in $F_1$ and $F_2$ these plants were tested for resistance against both diseases. Plants showing resistance for any one or both diseases were harvested separately. The $F_3$ plants susceptible to either disease were discarded, and only those plants which show resistance for both diseases were selected for further screening in $F_4$.

*Fusarium udum* is always present in high quantities in the soils having high proportion of sand [42], 30% water holding capacity, $25 \pm 5^{\circ}C$ temperature, high C : N ratio and sodium

nitrate [43], and with old diseased plants because their root exudates promote germination of the *Fusarium* [44]. Addition of 1% carbohydrate, sucrose, ammonium sulphate, or urea decreases the fungal population in the soil [43]. The *Fusarium* fungi in the root region of the diseased (susceptible) and healthy (resistant) plants differ quantitatively and qualitatively. Some susceptible cultivars had nearly double the *Fusarium* count than the resistant lines at seedling stage [44], which increased from seedling to flowering stage but decreased at pod formation stage in both susceptible and resistant cultivars [45]. In early stage of development the *Fusarium* population decreases in the wilted plants and increased in healthy plants but at later stage it becomes similar in the root of both types [46]. The mycoflora of wilt resistant cultivars consists of soil saprophytes and antagonistic organism [47].

Inoculation of *Fusarium udum* in sterilized soil produces more wilt than in unsterilized soil [48, 49] mainly due to inhibitory activity of the associated mycoflora in the soil, viz., *F. oxysporium* f.sp. *ciceri* and sp. *vasinfectrum* [50] and *Bacillus subtilis* [51]. Sap of SM infected plants inhibits the germination of *Fusarium* conidia [52]. Presence of the vasicular-arbuscular mycorhizae does not affect wilt incidence [53]. *Aspergillus lerrus* [54], and the nematodes *Heterodera cajani* [55, 56] and *Meloidogyne incognita* [57] appear to enhance the virulance of *Fusarium udum*.

*Fusarium udum*, a soil borne pathogen enters the plant through rootlets and wounds in the lateral roots. Its incidence is influenced by the biochemical defence mechanism. Alkaloids accumulate at the site of wound, act as fungitoxic [58]. The chemical cajanone [59], methionine [60], caffeic and chorogenic acids [61] and xylose [62] exudated from resistant pigeonpea cultivars inhibit germination and germtube growth of *Fusarium*. *Fusarium* resistance in pigeonpea has also been reported to be associated with high content of chlorophyll, ascorbic acid, total manganese, free reducing sugar [63] total and reducing sugars, flavanol, phenol, alkaloids, amino nitrogen, cystosine, tryptophan and alanine [61, 64]. Resistance is also associated with the lower quantity of phenylalanine [61, 64], Fe/Mn ratio and carbohydrate accumulation in the roots [63]. Susceptible cultivars depleted sugar rapidly from root and shoots after infection [65]. At all stages of plant growth the resistant cultivars had higher concentration of flavanol and total alkaloids than the susceptible cultivars [66]. More number of xylem vessels are plugged in the seedlings inoculated with the pathogen as compared to the uninoculated plants due to production of pectolytic enzymes in the vessels [67, 68]. Wilt incidence is reduced in the plant from which reproductive sinks are removed [69].

The activity of enzymes chlorophyllase, diastatic [70, 71], polyphenolic oxidase and RNase [72] increased substantially after SM infection. Synthesis of sucrose sugar takes place at a lower rate in the diseased plants which is a result of dislocation in total photosynthetic activity [73]. Resistant cultivars are characterized by the presence of specific isoperoxidase [74]. Chlorophyllic proteins [70], carbohydrates, calcium, potash, sodium, manganese [73],

bound amino acids and nitrogen [72] decreases in the sterility mosaic infected plants. The diseased leaves produce higher amount of cytoplasmic protein [71], reducing sugars, nonfermentable reducing substances, total nitrogen [73, 75] and free amino acid [72] than the leaves of healthy plants.

*Fusarium udum* has been shown to spread in the soil up to 275 cm in one season [76] and survives up to 8 years [77] on the tissues invaded by the pathogen [78] and colonized [79]. The fungus grows in the collar region and roots of the infested host in both imperfect (*Fusarium udum*) and perfect phase (*Gibberella indica*). On wilting it survives as saprophyte on dead plant parts for indefinite period or as perfect state [80]. SMV and its vector survive on summer pigeonpea, ratoon growth and perennial crop during off season [81, 82].

Cultural practices have been found to influence the disease incidence. Wilt incidence is reduced by green manuring with *Crotoloria juncia*, heavy application of nitrogen and farm manure [77], crop residue of oat [83], oil cake [84] and neem cake [85], rotation with tobacco over several years [86], mixed or intercropping with sorghum [87], especially grain sorghum [88], and *Crotolaria medicagina* [89]. Mixed cropping with sorghum reduces wilt incidence even in the second season [90]. One year break in crop rotation or fallow also reduces wilt incidence substantially [91]. Post-rainy season sowing [92] and higher plant population [93] also decrease wilt incidence considerably. Irrigation increases the incidence and spread of wilt [94], while oil cakes of margosa and castor stimulate the growth of *Fusarium* [95].

Intercropping with low plant population shows higher incidence of sterility mosaic diseases [96]. Late cultivars have lower incidence of sterility mosaic disease with 50 cm row spacing [97].

Breeding for wilt resistance is usually done by pedigree or mass pedigree selection, although in some cases backcrossing has also been successful [98]. Pedigree method was also reported to be useful in breeding for highly heritable traits, such as sterility mosaic disease resistance [97], however, selection based on single plant yield in early segregating generations has been found to be ineffective [99]. Bulk hybrid, advanced by single pod descent, appears to be a better procedure for breeding high yielding lines which are selected and advanced from the early segregating population tested for resistance in the disease screening nursery [100]. Studies are therefore necessary to determine the usefulness of the phenotypically homogeneous bulk population retaining heterogenesity (e.g. composites) as the final produce rather pure lines, looking to the complex nature of inheritance for multiple disease resistance and pollination behaviour of pigeonpea [98].

Few races of the pathogen and a number of resistant genes against Fusarium wilt are known. Genetics of resistance has also been worked out. Race-specific resistant genes have

not been identified. Number of races, genes for resistance, and nature of inheritance of resistance are not known for the sterility mosaic disease. In both the diseases, the resistance is of quantitative nature, therefore, screening of resistant genotypes is difficult. Similarly, homogeneous response to the pathogen is not guaranteed because of heterogeneous spread of *Fusarium udum* in the soil and sterility mosaic through the vector. Thus, host-pathogen relationship involves the biological nature of pathogen and plant complex pattern leading to slower growth rate for formation of resistant varieties.

The following conclusions emerge from the review of the situation with regard to the two major diseases of pigeonpea:

1. Standard techniques for screening of resistant variety/breeding material should be adopted. For effective screening against SMV, spreader row technique is more effective. Screening for Fusarium wilt resistant lines should be done in the sick plot, and the population of pathogen in the sick plots should be first characterized and then monitored at least once a year to ensure consistency of disease reaction.

2. Resistance of the line selected should be done based on 3–4 years of field screening in the sick plot at different locations and it should be corroborated by laboratory tests, glasshouse, and growth chambers.

3. Identification of different pathotypes of wilt is essential.

4. Systematic genetic studies need to be undertaken to have a better idea about the nature of inheritance of resistance.

Based on the experience gained from the work conducted at JNKVV, Jabalpur and from this review it can be concluded that breeding for wilt resistance may be done by adopting pedigree or mass pedigree method of selection and also through partial backcrossing. Simple bulk selection can also be favoured because of the greater values of both the proportion of homozygotes and genetic variance. Conservation of the genes of the recurrent parent and incorporation of resistant genes for sterility mosaic would be possible by following the pedigree method of selection and backcross breeding or by intermating the $F_2$ plants showing resistance. Resistance for both the diseases can be combined by adopting backcross breeding and advancing the material through bulk population and modified mass selection methods.

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