An updated review of biology, pathogenicity, epidemiology and management of wilt disease of pigeonpea (*Cajanus cajan* (L.) Millsp.)

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**ABSTRACT**

Fusarium wilt caused by *Fusarium udum* Butler is the most widespread and destructive disease of pigeonpea (*Cajanus cajan* (L.) Millsp.). It can cause up to 100% yield losses in the susceptible pigeonpea cultivars. Despite earlier investigations on pathological and physiological characteristics of *F. udum*, the nature of infection process and genetic basis of pathogen variability have not been clearly established. The frequent recurrence of Fusarium wilt and changing scenario of the pathogen in the major pigeonpea growing areas prioritized the research for developing broad spectrum wilt resistant cultivars. The need to study biology of the pathogen, epidemiology of the disease is essential to understand the changing scenario of wilt disease in the context of climate change. This will facilitate to develop and, or refine host resistance screening techniques, identify disease resistance pigeonpea genotypes and the integrated disease management technology. In this review attempts have been made to update the current state of art and science of the wilt including sign and symptoms of the disease, biology of pathogen, epidemiology of the disease, variability of the pathogen, host resistance, and other management options. Available information on biochemical and genetic basis of disease resistance have been updated and discussed with the identification of future research priorities.

**Keywords:** Biology, Epidemiology, *Fusarium udum*, Pigeonpea, Resistance, Variability

Pigeonpea (*Cajanus cajan* (L) Millsp.) is an important food legume grown in semi-arid tropical and sub-tropical farming systems under varied agro-ecological environments. It provides high quality vegetable protein to human beings and is one of the sources of animal feed and fire wood. Its cultivation is confined to developing countries, mostly in Asia and Africa. Globally the area and production of pigeonpea has increased from 2.86 million ha (mha) and 1.96 million tons (mt) in 1980 to 4.36 mha and 3.46 mt in 2006 respectively (FAOSTat 2008) (Fig.1). Pigeonpea represents about 5% of world legume production (Hillocks et al. 2000) and more than 70% is being produced in India. In India, pigeonpea is grown in an area of about 3.73 mha with annual production of 2.31 mt and productivity of 678 kg/ha (Anonymous 2010). However, despite its immense importance in sustainable agriculture its global production per hectare remained static over last three decades. The yield gap observed between the potential yield

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and on-farm yield is mainly due to biotic and abiotic stresses and the lack of efficient management practices.

Among biotic stresses diseases such as Fusarium wilt, sterility mosaic, Phytophthora blight, Cercospora leaf spot, collar rot, dry root rot, Alternaria leaf spot, powdery mildew and phylloidity are well known diseases of pigeonpea. Among them, Fusarium wilt caused by *Fusarium udum* is the most important soil borne disease of pigeonpea capable of causing 30-100% loss in grain yield (Nene et al. 1980, Upadhyay and Rai 1989, 1992, Kannaiyan and Nene 1981, Reddy et al. 1990). The disease was first reported from Bihar state in India (Butler 1906). Pigeonpea wilt is widely prevalent throughout the world and more important in India (Kannaiyan and Nene 1981) and in eastern Africa (Okiror 2002). The annual pigeonpea crop losses due to wilt alone have been estimated about US dollars 36 million in India (Kannaiyan et al. 1984). The disease is emerging as an important constraint to pigeonpea production in Africa (Reddy et al. 1993). It causes up to 60% losses in Kenya, 36% in Malawi, 20% in Tanzania, 16% in Kenya (Songa et al. 1991, Hillocks and Khonga 1996). Recently, wilt has been reported from southern Zambelia province (Mozambique) (Gwata et al. 2006) confirming the further spread of *F. udum* in southern Africa.

### DISEASE SYMPTOMS

Although the infection occurs in the early seedling stage (Fig. 2), characteristic symptoms are not visible until crop development stages (Reddy et al. 1990, Hillocks et al. 2000). The infected plants show symptoms of gradual chlorosis and wilting starting from 4 to 6 weeks after planting. However, wilt symptoms are most conspicuous during the flowering and podding stage. Black streaks in the vascular region (Fig. 3a) as well as under the bark (Fig. 3b) are characteristic signs of the disease. Partial wilting in affected plants is common. Many such plants show a dark purple band extending from the base to several feet above ground towards wilted branches (Fig. 4). In some genotypes purple band extends to one of the two lateral roots, stem/branches. Infection of the tap root most commonly produced complete wilting (Nene 1980, Reddy et al. 1993) (Fig. 5), whereas infection starting and extending from one of the two lateral roots more often caused partial wilting. Exceptions, however, were observed. The dried leaves on wilted plants do not shed for a long time.

Fusarium wilt symptoms can be easily mistaken with Phytophthora blight because the general symptoms of these two diseases are similar. The key distinguishing symptom to differentiate between the two diseases are the browning/blackening of xylem vessels in wilt disease, however, in Phytophthora xylem remains clear and phloem is smoky gray. Also the plants infected with *F. udum* can be easily uprooted whereas reverse is in case of Phytophthora blight. Detailed distinguishing features between these two diseases have been discussed by Pande et al. (2011).

### 2. CAUSAL ORGANISM AND PATHOGEN VARIABILITY

#### Causal organism

The pigeonpea wilt pathogen was first described as *F. udum* from India (Butler 1910) and later from Uganda (Small 1922). Butler (1926) thoroughly investigated the pathogen and found that *F. udum* cannot be distinguished from *F. vasiniaictum* that attacks cotton and sesamum. Based on the distinct cultural characteristics of *F. udum* from *F. vasiniaictum*, Padwick (1940) named the wilt pathogen as *Fusarium udum* Butler var. *cay time*. Later Snyder and Hanson (1940) named the fungus *F. oxysporum f. sp. cajani*, a nomenclature supported by Chattopadhyay and Sen Gupta (1967). However, the name *F. udum* is commonly accepted as the macroconidia of *F. udum* are distinguished by a prominent hook (Booth 1971), Rai and Upadhyaya (1979), discovered the perfect state of *F. udum* on wilted and dead pigeonpea plants near Varanasi in Uttar Pradesh, India, and identified it as a new species of *Gibberella*. Because of the large size of the perithecia, and the 2 celled (and rarely 3 celled) ascospores, it was named as *Gibberella indica*. Singh (1980) also observed *G. udum* near Allahabad in Uttar Pradesh, India and suspected the role of cloudy weather, high humidity, and combinations of high and low temperatures as responsible for its production. The work on the perfect stage of *F. udum* needs confirmation. An interesting Butler’s description (1910) of *F. udum* is as follows:

> “Mycelium may be parasitic or saprophytic. Hyphae are hyaline, slender, much branched, usually with little aerial growth; macroconidia are of the *Cephalosporium* type i.e., produced successively on the ends of short simple or clumped conidiophores and remain bound in a drop of liquid after adjunction, unicellular or with one or more septa, elliptical, hyaline singly, salmon pink in mass, occasionally developing from the surface of minute spherical stroma and then of the *Tuberularia* type, 5.15 x 2.4 μ in diameter; microconidial stage in culture usually moist and bacteria-like, white to salmon-pink, occasionally (on rice) orange red or purple; macroconidia of the *Fusarium* type, formed as the macroconidia but on shorter conidiophores and becoming free as soon as objected, falcate 3 to 5 septate, hyaline, 15-50 x 3-5 μ in diameter (Fig. 6), usually late in appearing; chlamydospores, round or oval, thick walled, hyaline, sometimes in short chains, 5 to 10 μ in diameter”.

Various media are used for culturing and maintenance of the fungus viz., potato dextrose agar (PDA), sucrose-casamino acids agar medium (SCAM), oat-meal agar (OMA) and sand-pigeonpea flour medium (SPFM) (Hukma Ram and Pandey 2011, Ghosh and Sinha 1981, Subramanian 1962, Tiwari Shashi and DharVishwa 2011). The fungus grows well on Rawlin’s and Richard medium but sporulation is more on PDA and Czapek’s media (Prasad and Chaudhary 1977). Production of the Chlamydospores was dependent on nitrogen concentration (Prasad and Chaudhary 1965).
Pathogen variability

Fusarium spp. one of the most diverse groups of fungi have worldwide occurrence under the diverse conditions of soil and climatic factors. Pathogenic variation is a well-known phenomenon among Fusarium spp. Several workers (Baldev and Amin 1974, Shit and Sen Gupta 1978, Pawar and Mayee 1986, Reddy and Chaudhary 1985, Gupta et al. 1988) have reported cultural, morphological and pathogenic variability. The existence of variants/races in F. udum has been reported (Subramanian 1963a, Mukherjee et al. 1971) and is cited as a major drawback in the development of pigeonpea varieties resistant to Fusarium wilt (Green et al. 1981). So far 5 variants (strains) of F. udum have been identified and documented (Reddy et al. 1996, Mishra and Dhar 2003, Mishra 2004). Subsequently, based on the studies with a limited number of F. udum isolates and pigeonpea genotypes, Pawar and Mayee (1986) and Tiwari Shashi and Dhar Vishwa (2011) reported the cultural and the pathogenic variability in the fungus. Songa et al. (1995) has confirmed the variability of F. udum through field trials. Kiprop et al. (2002) observed differential reactions of seven pigeonpea varieties to 17 different isolates of F. udum and concluded that five virulent groups exist among Kenyan isolates. Based on the reaction of four pigeonpea lines, 11 isolates from India were divided into three (Table 1) distinct groups (ICRISAT 1996). Studies conducted at ICRISAT centre, and multilocal testing of resistant genotypes in India also point to the possible presence of physiological races in F. udum and shown differential response of pigeonpea lines to wilt across the locations and seasons (Reddy et al. 1996). However, the existence of races/ strains/ variants in F. udum as still not clear and needs detailed investigations.

| Table 1. Reaction of four differential pigeonpea lines to 11 isolates of Fusarium udum in pot experiments in a greenhouse at ICRISAT Asia Center, 1995-96. |
|-----------------|-----------------|-----------------|-----------------|
| Line           | Strain 1        | Strain 2        | Strain 3        |
| ICP 2376       | S               | S               | S               |
| C 11           | R               | S               | S               |
| ICP 8863       | R               | R               | S               |
| ICP 9174       | R               | R               | R               |

*a = Susceptible, b = Resistant

Strain 1: Gwalior and Akola
Strain 2: Dholi, Kanpur, Varanasi and Bangalore
Strain 3: Patancheru, Rahuri, Badnapur and Gulbarga

Cultural and morphological variability

Based on the cultural characters, Gupta et al. (1988) differentiated F. udum isolates of Madhya Pradesh into seven groups. Similarly, Rajendra and Patil (1992) reported the cultural, morphological and physiological variation in the 22 isolates of F. udum collected from Maharashtra state of India. The F. udum isolates have great variation in mycelial color, substrate color, mycelial growth and sporulation (Fig. 7, Kiprop et al. 2002, Sukumar et al. 2012). Some isolates of F. udum also show great variation in conidial length, conidial septation and growth rate (Sinha et al. 2008). Baldev and Amin (1974) tested 10 isolates of F. udum from India on 10 pigeonpea lines. Only three pigeonpea lines were resistant to all the isolates. They also characterized these isolates as races of this fungus. Further existence of races in pigeonpea wilt pathogen was also identified by Rajendra and Patil (1993). Different isolates from India were collected by Sukumar et al. (2012) and they found that these isolates differ in their mycelial color, substrate color, mycelial growth and virulence.

Biochemical variability

Kumar et al. (2007) investigated the pathogenic and biochemical variability among the 11 isolates of F. udum collected from Uttar Pradesh, New Delhi and Hyderabad. This study revealed variability in enzyme production and cell bio-molecular composition viz., total sugar, total protein and amino acids among the isolates. Further it was also noticed that most aggressive isolates were rich in sugar content. Enzymes Polygalacturonase (PG), Pectin methylesterase (PME) and cellulase were more in highly aggressive isolates and less in less aggressive isolates. Studies conducted by Nagabhushana (2006) reported that there is an increased activity of polyphenol oxidase, peroxidase and phenylalanine ammonia lyase and decreased activity of total sugars in resistant genotype WRP-1 of pigeonpea when F. udum and Heterodera cajani were inoculated together. Similar increase in phenolic substance was also observed in maize grown out of seeds treated with P. fluorescens and inoculated with R. solani (Sivakumar and Sharma 2003). Cajanol production in ICP 9145 pigeonpea variety is decreased due to root-knot nematode thereby increasing the susceptibility to Fusarium wilt (Marley and Hillcocks 1994). Lipoygenase activity was significantly higher in the resistant than in the susceptible genotypes of pigeonpea and was enhanced further in response to infection with F. udum (Devi et al. 2000).

Genetic variability

Sukumar et al. (2012) analyzed the genetic variability (RAPD-PCR analysis) of F. udum isolates collected from different geographical locations of India and found a high degree of variability in pathogenicity and genetic diversity among the populations. Similarly Kiprop et al. (2005) analysed 38 isolates of F. udum collected from various districts of Kenya, based on vegetative compatible groups (VCG) and amplified fragment length polymorphism (AFLP) and found that pathogenic isolates of F. udum appear to originate from a single lineage. Sivaramkrishnan et al. (2002) analysed the genetic variability in 36 isolates of F. udum collected from 4 pigeonpea growing states of India using Random Amplified Polymorphic DNA (RAPD) and Amplification Fragment
Length Polymorphism (AFLP) techniques and showed existence of a minimum of 3 specific races of the pathogen prevailing in the pigeonpea growing areas of India.

**Fusarium spp. and Wilt**

Several distinct isolates of *Fusarium* spp. were isolated from wilted pigeonpea plants of which one isolate cause severe wilt but no foot rot (Padwick 1939). Other species of *Fusarium* reported associated with pigeonpea wilt are *F. vasinfectum* (Mitra 1931, 1934, Mundkur 1938, Butler 1926), *F. oxysporum* (Mukiibi 1976) and *F. accuminatum*, *F. equiseti*, *F. merismoides*, *F. semitectum* and *F. solani* (Reddy et al. 1990). There is no doubt that *F. udum* is highly variable, however, there is an urgent need to erect a universal protocol to quantify cultural, morphological and biochemical variation in this pathogen. To determine pathogenic variations, a universally accepted set of host differential genotypes needs to be identified. Molecular characterization of *F. udum* isolates collected from different agro-ecological regions will be a pre-requisite for the development of the durable resistance to wilt disease of pigeonpea.

**HOST RANGE AND MECHANISMS OF RESISTANCE**

The pathogen *F. udum* is host specific to pigeonpea (Padwick 1940, Subramanian 1963a, Booth 1971) with an exception of *Catharanthus roseus*. Kannaiyan et al. (1984) conducted an experiment using 30 weed species, among them only 10 yielded *Fusarium* species but none of these were *F. udum*. Moreover, the weed species tested did not show any symptom of wilt at physiological maturity. The optimum moisture and temperature of soil alter the cessation of rainy season and resistance level of pigeonpea variety was largely responsible for the course of wilt development. This fungus is primarily soil borne facultative parasite and enters the host through fine roots and subsequently colonizes different plant parts (Kaiser and Sen Gupta 1975, Nene et al. 1979). The pathogen sporulates heavily over infected plants and increases its population in vicinity. However, there is no sporulation in resistant varieties (Subramanian 1963a). It is reported to spread more rapidly along the roots than across the soil (Butler 1910).

Wilting has been correlated with plant age (Mundkur 1935, Kotasthane and Gupta 1981). In India, pigeonpea being a rainy season crop is usually sown during June-July. Seedling mortality is often seen during August, and adult plants wilt from flowering onwards during November-December (Butler 1906, Kotasthane and Gupta 1981). The ability of the host to withstand invasion by the pathogen increases with age of the host (Subramanian 1962). Wilt incidence had no correlation with erect habit, dwarfness, clustered inflorescence, seed colour, etc. (Pal 1934). Mishra (2004) further added that the

**Table 2: Sources of resistance to Fusarium wilt of pigeonpea**

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Remarks</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICP 6739, ICP 8980, ICP 11015, ICP 13304, ICP 14638, ICP 14819</td>
<td>Resistant both in greenhouse and field except ICP 14638 all are resistant to SMD also</td>
<td>Sharma et al. 2012</td>
</tr>
<tr>
<td>ICP 14976, ICP 15049</td>
<td>Moderately resistant both in greenhouse and field</td>
<td>Sharma and Pande 2011</td>
</tr>
<tr>
<td>ICP 7903, ICP 12031, ICP 12059, ICP 12771, ICP 12775</td>
<td>Highly resistant (asymptomatic) both in greenhouse and field. Originated from India, Tanzania, Philippines, Kenya and Zaire</td>
<td></td>
</tr>
<tr>
<td>ICP 7991, ICP 12841, ICP 13257, ICP 13258, ICP 13618, ICP 14291, ICP 15137</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICEAP 00040</td>
<td>Resistant both in greenhouse and field; originated from India, Tanzania, Philippines, Kenya and Zaire</td>
<td>Gwata et al. 2006 and Tanzania</td>
</tr>
<tr>
<td>ICP 8863, ICP 9145, ICP 9174, ICP 12745, ICPL 333, ICPL 8363, ICPL 88047, BWR 370, DPPA 85-2, DPPA 85-3, DPPA 85-8, DPPA 85-13, DPPA 85-14, Bandapalera, ICP 4769, ICP 9168, ICP 10958, ICP 11299, C 11 (ICP 7118), BDN 1</td>
<td>Showed high levels of resistance in Kenya, Malawi and Tanzania</td>
<td>Reddy et al. 1993</td>
</tr>
</tbody>
</table>

**Fig. 1:** Global pigeonpea area, production and productivity (FAOStat 2008)**
Fusarium udum produces poly methyl esterase, polygalacturonase and cellulase enzymes (Singh and Husain 1962, 1968) and a toxin fusaric acid (Singh and Husain 1964, Prasad and Chaudhary 1974) both in vivo and in vitro. However, toxins produced by F. udum need further investigations. Similar to toxins produced by pathogens, there are reports on the production of antifungal compounds produced by pigeonpea plant. Preston (1977) reported an antifungal compound ‘Cajanone’ from the roots of wilted plants, which was inhibitory to F. udum. Other inhibitory compounds present in the roots extracts of resistant genotypes are chlorogenic acid, caffeic acid and an unknown phenolic acid. Gupta (1994) reported

Fig. 2: Wilt symptoms on seedlings

Width of xylem and vascular bundles and thickness of roots were less in resistant genotypes than in susceptible ones. Thus, these aspects can be determining the resistance level of pigeonpea genotypes.

Fig. 3: (a) Internal xylem blackening, (b) browning of the stem

Fig. 4: Dark purple band extending from the base to upwards

Fig. 5: Complete wilting and drying of the adult plant

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of host in the soil (Butler 1908, Mitra 1925). Kannaivan et al. (1981) found that the fungus can survive in plant stubbles for 2.5 years in Vertisols and 3 years in Alfisols, while, Sharma and Singh (1973) observed that the symptomless carriers for wilt disease are weeds and cultivated plants and could be possible source of primary inoculum of F. udum.

**Transmission of pathogen**

Soil plays a major role in the transmission of the F. udum propagules. Susceptible genotypes of pigeonpea when grown continuously in a field, wilt spreads about 3cm through the soil in one season along the roots (McRae 1924). Air and irrigation water plays an important role in rapid spread of the disease. Termites can spread wilt by carrying the fungus propagules from infested to healthy plants; such infested termites can also cause the disease in pigeonpea plants grown in the sterilized soil (Upadhyay and Rai 1983).

**Disease cycle**

Pigeonpea wilt was earlier known to complete its life cycle through the imperfect state. After the discovery of its perfect state (G. indica), Upadhyay and Rai (1992) established that both imperfect and perfect states are important in completing the wilt disease cycle. However, the imperfect state of the pathogen is more important and prevalent in the nature. Longevity of survival of the perfect state is yet to be determined while through the imperfect state, the pathogen can survive in the soil up to five years. Mycoparasitisation on other fungi and host debris plays an important role in the disease cycle (Upadhyay and Rai 1983). After the establishment of seed borne nature of the wilt pathogen (Dwivedi and Tandon 1976), role of seed in the spread of pigeonpea wilt especially in newer areas has become crucial. The extent of seed transmission sometimes reaches as high as 30% (Jeswani and Gemawat 1981). Transmission of pathogen from seed to seedling has been further confirmed by Haware and Kannaiyan (1992). Spread of disease from plant to plant occurs through root contacts, irrigation, rainwater and termites (Upadhyay and Rai 1992).

**Factors influencing disease development**

Fusarium wilt is favoured by low soil temperature and increasing plant maturity (Mundkur 1935). Soil water holding capacity (30%) and soil temperatures between 20 and 30°C favours the disease (Singh and Bhargava, 1981). Addition of soil amendments viz., Zn, Bo and Mn will decrease the activity of F. udum (Sarojini 1951). Presence of host and non-host seeds of legumes and cereals enhance the growth within 96hr, although host seeds do not encourage sporulation (Singh 1974). Slightly acidic alkaline soils with 50% or more sand favour the wilt (Upadhyay and Rai 1989), while heavy black soils not favour the disease (Shukla 1975). Butler (1906) reported that wilt appears in young seedlings but highest

4. **EPIDEMIOLOGY**

**Survival and source of inoculum**

The wilt pathogen F. udum has been reported to survive in soil (McRae 1926) and on pigeonpea seed (Haware and Kannaiyan 1992). However, major infection occurs through the soil. The fungus can remain alive upto 10 years in absence

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**Fig. 6:** A-D: Macroconidia; E-F: Microconidia; G-I: Microconidia in situ on carnation leaf agar. A-F, scale bar =25 μm. G-I, scale bar = 50 μm. (Source: The Fusarium Laboratory Manual, John F. Leslie and Brett A. Summerell 2006).

**Fig. 7:** Colony growth pattern and pigmentation of Fusarium udum isolates on Potato dextrose agar (The first row in each plate is the upper surface and second row the lower surface).
mortality is caused at flowering. Temperatures range of 12-29°C favours the disease development in pigeonpea plants approaching physiological maturity (Mundkur 1935). Wilt incidence can be prolonged by removing the flowers during reproductive stage (Sheldrake et al.1978). Thus the disease aggravates in ratoon crop and there seems close relation between flowering period, wilt intensity and soil temperature that is why the early varieties suffer less due to wilt (Kotasthane and Gupta 1981). This may be due to sufficient and efficient moisture utilization of water and early maturing of the plant before the expression of the symptoms.

Nematodes also play an important role in the spread of the disease by injuring the plants and also affect the growth (Edward and Singh 1979). Pigeonpea infestation by cyst nematode (Heterodera cajani) increases the wilt incidence (Hasan 1984). Some of the nematodes viz., Meloidogyne javanica and M. incognita found associated with the breaking down exponentially the resistance by retarding the accumulation of cajanol an enzyme responsible for resistance in "ICP 9145" (Marley and Hillocks 1994). Combined infection of F. udum with H.cajani caused more wilting than with Meloidogyne spp. alone (Siddiqui and Mahmood 1996).

5. DISEASE MANAGEMENT

Host plant resistance

The preliminary step for exploiting host plant resistance (HPR) is the development of reliable and repeatable techniques for large scale screening of germplasm and breeding lines. Several techniques suitable for Fusarium wilt resistance screening under field, greenhouse and laboratory screenings have been reported (Nene et al. 1982, Kimani et al.1994, Pandey et al. 1996). However, variation in the reaction of the pigeonpea lines between experiments, suggests the need for further refining the screening techniques. Following greenhouse and field screening techniques are commonly used to cull out the wilt susceptible genotypes, and identify the wilt resistant genotypes.

Greenhouse screening

The greenhouse screening technique consisted of multiplication of inoculum, raising of seedlings of pigeonpea in autoclaved soil, root dipping in inoculum and transplanting in pots filled with autoclaved soil and assessing disease incidence. The pathogen is multiplied at 25±10°C for 7 days on potato dextrose broth (PDB) in flasks kept on the shaker incubator. The content was macerated in warring blender for one-two minutes. The seedlings were inoculated by dipping their roots in the inoculum for one minute and then they were transplanted in pot containing autoclaved sand, vertisol or alfisol soil. Un-inoculated seedlings transplanted in un-inoculated sand/soil are used as control (Nene et al. 1981, Haware and Nene 1994). Nene and Kannaiyan (1982) developed a sick pot screening technique. In this technique the fungus was mass multiplied on sand: pigeonpea (9:1) meal medium for 15 days at 28-30°C. After multiplying for 20 days, 200 gm of this medium was mixed with 2 kg autoclaved red soil and placed in 15cm plastic pot and were incubated at 25-30°C. After 2 days, in the pathogen infested pots, 7-10 days old seedlings were transplanted. Wilt incidence was recorded 60 days after transplanting.

Field screening

The most common method used for field screening is the sick plot method. In 1908 Butler tested a number of pigeonpea genotypes in plots severely infected by the wilt fungus. McRae and Shaw (1926) used susceptible variety as an indicator line after every test entry for selecting wilt resistant line in wilt sick area. The diseased debris collected from the previous crop was buried between each row for creating the diseases. Vaheeduddin and Nanjundiah (1956) created wilt sick plot for screening the pigeonpea genotypes by spreading compost made of wilted plants. In each year, care was taken that plot was thoroughly infested with F. udum. The components and procedures of the “field screening” of pigeonpea genotypes for wilt resistance standardized at ICRISAT by Nene et al. (1981) involved, planting of test material with a 30-cm row space and interplanting a susceptible cultivar (e.g. “ICP 2376”), which serves as an indicator line after every 2-4 rows. Reddy et al. (1990) developed a diseased debris field inoculation technique. In this technique, a well leveled Alfisol was selected and wilt susceptible genotype “ICP 2376” were sown as closely as possible (30 ×10 cm) on flat beds preferably before the monsoon rain arrives. When the plants are about 1 month old, approximately 250 kg of diseased plant debris (pigeonpea stems with wilt symptoms were collected during the previous season and stored dry in the field shelter) are scattered over the field. During rain-free days sprinkle irrigation was liberally provided. This technique produced near 100% wilt incidence in susceptible controls at ICRISAT, Patancheru.

Resistance sources

The search for sources of resistance to wilt in pigeonpea began as early as 1905 at Pune in India (Butler 1908, 1910). Subsequently screening has been conducted at many locations and several wilt resistant genotypes identified (Table 2). Singh (2011) after testing several varieties at hot spot locations across India found that the long duration pigeonpea varieties “IPA 16F”, “IPA 8F”, “IPA 9F” and “IPA 12F” were good source of resistance to all the five variants of F. udum prevalent in India and these can be used as resistant donors in pigeonpea wilt resistance breeding programme. Release of long duration wilt resistant pigeonpea variety ICEAP 00040 for commercial production in 2003 by Gwata et al. (2006) confirms that in general long duration pigeonpea genotypes are resistant to wilt. Similarly, Chaudhary (2010) released a
long duration wilt resistant variety IPA 204 in 2009 after testing in 24 pigeonpea growing areas across India.

There is a need to develop high yielding pigeonpea varieties with combined resistance to wilt, sterility mosaic and Phytopthora blight with bold, white seed in the short, medium and long duration groups. Recently Sharma et al. (2012) identified five accessions; “ICPs 6739, 8860, 11015, 13304 and 14819” with combined resistance to wilt and SMD in pigeonpea minicore. Also good sources of multiple disease resistance (wilt, SMD and PB) have been identified in vegetable pigeonpea lines “ICP 7991, 12841, 13257, 13258, 13618, 14291, 15137” (Sharma and Pande 2011). However, there is a need to develop a better understanding of the inheritance of resistance, particularly in view of the fact that genotypes show different levels of resistance under field conditions. The medium duration genotypes “C 11” and “BDN 1” that have large seeds and good yield do not have high level of resistance. Maruti “ICP 8863” is becoming popular in Karnataka where its high yield, good seed size, and high stable wilt resistance are appreciated by farmers (Konda et al. 1986).

Biochemical and histopathological basis of host plant resistance

The biochemical studies conducted by Subramanian (1963b) showed higher contents of chlorophyll, ascorbic acid, free reducing sugars and total Mn in the resistant variety “NP 25” as compared to susceptible “NP 24”. On the other hand, NP 24” had more total carbohydrates in roots as compared to shoot while reverse was true with “NP 15”. The Fe:Mn ratio increased with increasing susceptibility. Resistance to pigeonpea wilt is observed to be associated with higher contents of total sugars, reducing sugars, amino nitrogen, amino acids, phenols, flavanols, alkaloids, xylose, cystine, tryptophan but lower amount of phenylalanine (Murthy and Bagyaraj 1978). Flavanol and chlorogenic acids and an unidentified phenolic compounds present in resistant variety were inhibitory to spore germination. It is considered that cystine counteracts the fungal infections by chelating ferric ions that activate the Fusarium toxin. The cysteine and tryptophan were detected only in shoot of resistant variety but phenylalanine was more in susceptible one (Murthy and Bagyaraj 1978). Flavanol and alkaloid were more in resistant than in susceptible cultivar (Murthy and Bagyaraj 1980). No spores of the pathogen were produced in least susceptible variety NP 15 due to lack of substrate in the root system or the action of some inhibitory substance in the xylem (Subramanian 1963a). Susceptible genotypes will have significantly thicker roots and wider vascular bundles and xylem vessels when compared to resistant genotypes (Chaudhary and Kumar 2000).

Genetic basis of host pathogen interaction

Lack of more pigeonpea genotypes resistant to F. udum is due to the difficulty in working with this host-specific Fusarium in breeding programs because of frequent evolution of new races and coexistence of more than one pathotypes at one location (Chaudhary 2010). It appears that the identification of resistance to F. udum is a challenging task because of its cross pollinating ability. Limited reports are available on genetics of wilt resistance in pigeonpea. Pal (1934) was the first to investigate the genetics of wilt resistance in pigeonpea and reported multiple genetic controls. Shaw (1936) and Pathak (1970) reported two complementary genes conferring resistance to Fusarium wilt in pigeonpea. Odeny et al. (2009) studied genetics of resistance in an African “ICEAP 0040” and Indian “ICP 8863” genotypes. They found that the wilt resistance in “ICEAP 0040” was controlled by single recessive gene, while in “ICP 8863”, two pairs of recessive genes governed the resistance. Recently, Dharwad (2012) reported that wilt resistance is governed by single dominant gene. If this is the case then introgression of resistance to the susceptible genotypes will be easy using effective breeding strategy like backcross breeding. Saxena et al. (2012) reported one dominant and one recessive gene with dominant suppressive epistatic effects responsible for controlling resistance to wilt.

Cultural control

Cultural operations play an important role in the control of soil borne diseases mainly deep summer ploughing and soil solarization. Soil solarization is very effective method against F. udum. Various cultural methods like post-rainy sowing and limited application of the urea will help in managing the Fusarium wilt disease (Sharma 1980). Application of Zn will retard the growth of the pathogen (Sarojini 1950). Crop rotation is one of the important cultural practices in controlling the spread of the disease. Crop rotation with sorghum (Natrajan et al. 1985), cereals (Khan and Ashley 1975), tobacco or fallow (Bose 1938, Natrajan et al. 1985) showed the decrease in the pathogen population as well as the disease incidence. When Crotalaria medica ginea is mixed cropped with pigeonpea there is a high reduction in pigeonpea wilt incidence (Upadhyay and Rai 1981). Green manuring with Crotalaria juncea and nitrogen application in the form of farmyard manure will also help in reducing the disease incidence (Upadhyay and Rai 1981, Verma and Rai 2008). Inter or mixed cropping with some of the crops like sorghum is advised as they creates the antifungal effect on the F. udum (Mathur 1954). Flow of water from diseased field to healthy field favours the spread of the disease. Hence it is advised to check the water flow from wilt infested field to healthy pigeonpea fields.

Biological control

For an eco-friendly and sustainable management of Fusarium wilt, biological control with the application of Plant Growth Promoting Rhizobacteria offers a potential nonchemical means for disease management. Several strains
of Pseudomonas and Bacillus have been widely reported as effective biocontrol agents for pigeonpea wilt, though combination of several organisms have been proved more effective in field conditions (Pandey et al. 2011). Among the 3 bioagents viz., T. viride, P. fluorescens and P. aeruginosa tested in vitro and in pots against F. udum, it was found that T. viride completely checked the mycelial growth of F. udum (Hukma Ram and Pandey 2011). The seed dressing by P. dispersa reduced wilt incidence (47%) in field trials, which is greater than Bavistin (41%) and Trichoderma Monitor WP (36%) treatments. P. dispersa is reported to be commercial Fusarium wilt biocontrol agent (Maiasuria et al. 2008).

Combined application of Sinorhizobium fredii KCC5 and P. fluorescens LPK2 (isolated from nodules of Cajanus cajan) and disease suppressive soils of tomato rhizosphere with half dose of chemical fertilizer showed a significant increase in seed germination (94%), per plant number of pods, nodules, shoot length, root length, shoot weight and root weight. Both strains KCC5 and LPK2 led to proto-cooperation as evidenced by synergism, aggressive colonization of the roots, and enhanced growth, suggesting potential biocontrol efficacy against Fusarium wilt in pigeonpea (Kumar et al. 2010). The bacterium B. subtilis has been reported to be antagonistic against F. udum (Vasudeva and Govindaswami 2008). Singh et al. (2002) found that Aspergillus flavus, A. niger, B. licheniformis (strain-2042), Gliocladium virens, Penicillium citrinum and T. harzianum were potent in reducing the Fusarium wilt both in-vivo and in-vitro. Among them G. virens reduced maximum wilt incidence when applied to soil. Pandey and Upadhyay (1999) found T. viride as well as T. harzianum best for checking pigeonpea with seed application.

**Cross protection**

Chadha and Raychaudhuri (1965) reported that the plants infected with the SMD are protected by the Fusarium wilt disease as the sap of the virus infected plant does not contain the glutamic acid or alanine which promotes the germination of Fusarium spores. Pigeonpea plants also show the resistance when they are inoculated with the non-pathogenic Fusarium spp. viz., Fusarum oxysporum f. sp. ciceri and F. oxysporum f. sp. vasinfectum at 3-5 days before inoculation of the pathogen (Kaiser and Sengupta 1969, Maitra and Sinha 1973). Pre Inoculation or simultaneous inoculation of F. oxysporum f. sp. niveum, F. oxysporum f. sp. ciceris, F. solani f. sp. pisi and Cephalosporium sacchari was effective in controlling wilt of pigeonpea to a great extent (Chakraborty and Sen Gupta 1995).

**Chemical control**

Being a soil borne disease, chemical control of wilt is not much effective. However, a few reports on this aspect are available regarding in vitro as well as in vivo testing. The mycelial growth of F. udum is inhibited partly or completely by griseofulvin at 0.1 to 2.5 μg/ml (Chakrabarti and Nandi 1969), Benlate (Sinha 1974), Bavistin (Ghosh and Sinha 1981), Topsin M 70 and thiram (Sumitha and Gaikwad 1995), Penchala Raju et al. (2008) conducted an experiment by using five fungicides viz., carbendazim, thiophenate methyl, thiram, captan and dithane Z-78 and found carbendazim inhibited the growth of the fungus completely at 100 ppm followed by thiophenate methyl (96.6%) and thiram (70.0%). Hukma Ram and Pandey (2011) reported that mycelium growth of F. udum was completely inhibited by the fungicides carbendazim (500 μg ml⁻¹), difenconazole (100 μg ml⁻¹), hexaconazole (200 μg ml⁻¹) and combi product of captan + hexaconazole (250 μg ml⁻¹), and carbendazim + mancozeb (500 μg ml⁻¹). Devi and Chhetry (2012) reported that Allium sativum at 20% resulted in 100% inhibition of mycelial growth and spore germination of Fudum.

Seed treatment with fungicides such as Benlate, Bavistin and BAS 38601 F for control of pigeonpea wilt has been found effective (Ghosh and Sinha 1981). The seed borne inoculum of F. udum is eliminated by seed treatment with benomyl and thiram (Haware and Kannaiyan 1992) and Bavistin@ 2g/kg of seed (Pandey and Upadhyay 1999). Benlate being systemic in nature proved to be more effective but its continuous use may develop resistance in the pathogen. Hence, it is suggested to use benomyl mixed with other fungicides specially carbendazim, thiram, difolatan etc. (Kamble and Gangawane 1994). Essential oil from Ageratum houstonianum was found toxic to pathogen but not to pigeonpea plant (Pandey et al. 1983). Devi and Chhetry (2012) reported that aqueous extract of Allium sativum showed highest percentage of disease control.

**Integrated disease management**

For effective management of the pigeonpea wilt, integrated disease management (IDM) is very important not only in controlling the wilt incidence but also to protect soil health. In IDM, there is a need to combine more than one disease management practices. A combination of host plant resistance, cultural practices like deep summer ploughing, mixed cropping, crop rotation, removal of stubbles, seed treatment with bio-control agents etc. was found effective in minimizing wilt incidence (Reddy and Dhar Vishwa 2000). According to Mahesh et al. (2010) a combination of carbendazim seed treatment 2g/kg of seeds + soil application of P. fluorescens, T. viride each @ 2.5 kg/ha in FYM applied @ 50 kg/ha recorded least mean wilt incidence. An integrated treatment of T. viride and T. harzianum with thiram was best with 68% disease control (Pandey and Upadhyay, 1999). Thus seed coating with bio agents proved better and safe for the management of wilt of pigeonpea. According to Hukma Ram and Pandey (2011), combined seed treatment of metiram (0.1%) + T. viride was effective in controlling the wilt disease in pigeonpea.
CONCLUSIONS AND FUTURE OUTLOOK

Resistance to wilt in pigeonpea genotypes has historically been overcome by new pathotypes of F. udum; hence the genotypes intended for release to farmers should be selected based on multi-location, multi-season field trials. Sterility mosaic disease caused by virus is often seen economically reducing the yields of wilt resistant genotypes. Therefore, efforts should be made to develop resistant genotypes with combined resistance to wilt and SMD. Durable resistance may only be possible if array of resistance genes is combined prevailing different mechanisms of resistance against all races/pathotypes in a single cultivar. There is a need to develop a better understanding of the inheritance particularly of the fact that genotypes show different levels of resistance under field conditions. Studies are also needed to determine the genetics and allelic relationships of resistance to wilt in genotypes as an essential precursor of pyramid resistance genes. To understand further the nature of resistance genes, studies with known races/variants will be required for long-term solutions of this disease. At present, such information about Fusarium wilt in pigeonpea is limited and inconclusive. In the anticipated scenario of climate change, studies on the ecology of F. udum and its epidemiology are required to improve the current disease management strategies those are heavily based on host-plant-resistance. Although varieties released from the pigeonpea improvement programs with highest levels of stable resistance to wilt and SMD have served the immediate needs of farmers. However, resurgence of Phytophthora blight and susceptibility of wilt resistant cultivars to this disease (Pande et al. 2011) warned breeding for multiple disease resistance in pigeonpea. There is an urgent call for national programs to focus on more organized breeding schemes that would enable development of genotypes that combine superior agronomic traits. Selection and identification of high yielding genotypes with combined resistance to wilt, SMD and Phytophthora blight with improved agronomic traits should be continue and extend to farmers. Management of wilt is essential to provide increased and stable pigeonpea yields throughout the world. To study the variation in the virulence of the different isolates of the pathogen has been of immense importance and can be worked out by using DNA finger printing with synthetic oligonucleotides.

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