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Importance and management of fusarium wilt (*Fusarium udum* Butler) of pigeonpea

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Abstract

Fusarium wilt (*Fusarium udum* Butler) is an important soil borne disease of pigeonpea [*Cajanus cajan* (L.) Millsp], which causes significant yield losses in susceptible cultivars throughout the pigeonpea growing areas. The soil borne fungus enters the host vascular system at root tips through wounds leading to progressive chlorosis of leaves, branches, wilting and collapse of the root system. Temperature, soil type, water retentive nature of the soil and nutrient availability has been shown to affect fusarium population. Disease management strategies have emphasized on integrated disease management practices. Despite extensive pathological and molecular studies, the nature and extent of pathogenic variability in *F. udum* has not been clearly established. Information on characterization of *F. udum* is needed to help identify race differentials. In addition, there is limited knowledge on the inheritance of fusarium wilt and other important traits in pigeonpea thus limiting specific cultivar improvement. This paper reviews the literature on the distribution, symptomalogy, factors that affect its development and control strategies of the disease.

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Introduction

Fusarium wilt (*Fusarium udum* Butler) is an important soil borne disease of pigeonpea [*Cajanus cajan* (L.) Millsp], which causes significant yield losses in susceptible cultivars throughout the pigeonpea growing areas. In India, the annual loss due to this disease is estimated at US $71 million (Reddy *et al.* 1993). *Fusarium* wilt causes economic loss in pigeonpea of about 470, 000 t of grain in India and 30,000 t of grain in Africa (Joshi *et al.* 2001). Kannaiyan *et al.* (1984) reported wilt incidence (and range) in Kenya, Malawi and Tanzania of 15.9% (0-90%), 36.6 (0-90) and 20.4% (0-60%) respectively with annual loss estimated at US $ 5 million in each of the countries. In Tanzania, an incidence of *Fusarium* wilts as high as 96% has been observed (Mbwaga 1995). Losses due to wilt in Kenya vary from negligible amount to 100% depending on the stage of the crop and environmental factors (Kannaiyan and Nene 1981; Sheldrake *et al.* 1984).

Although numerous control measures have been suggested to alleviate the problem of wilt and increase productivity of pigeonpea, their success still remains low due to prohibitive cost of practices and labour-constrained smallholder producers. These practices are both resource and knowledge intensive and small farmers often find it difficult to control the disease especially when the rate of field infestation is high. Research on disease management strategies for *F. udum* may be relevant to areas where the disease is important. This paper reviews the literature on the distribution, symptomalogy, pathogenecity, factors affecting its spread and control strategies of the disease. The paper also suggests priority areas for future research.

Geographical Distribution of Fusarium wilt

The disease was first recorded by Butler (1906) in India. Although the disease is more prevalent in India, east Africa and Malawi where field losses of over 50% are common, it also occurs in Bangladesh, Grenada, Indonesia, Mauritius, Mynmar, Nepal, Nevis, Venezuela, Trinidad, and Tobago (Kannaiyan *et al.*, 1984; Reddy *et al.*, 1993; Marley and Hillocks, 1996). Recently, this pathogen was reported to be spreading in Southern Africa reaching areas in Mozambique (Southern Zambezia province) (Gwata *et al.*, 2006). Although the incidence and distribution information is not available, the disease has also been reported in Zambia (Reddy *et al.*, 1993). Ghana is also included in the distribution list but presence of the disease in the country has not been confirmed (Reddy *et al.*, 1993). In Kenya, the disease was first reported in 1983 when the first released variety (*Munaa*) broke down with *Fusarium* wilt and was withdrawn from the farmers (Kimani, 1991). The disease is found in all pigeonpea growing areas but incidences are high in the eastern areas (Kannaiyan *et al.* 1984; Hillock and Songa, 1993). In Tanzania the distribution occurs around babati in the north in the southern zone around Mtwara and along the coast near Dar es Salaam (Hillocks, unpublished). Although the Fusarium wilt has been observed in Uganda, the present distribution and incidence of the disease is not known.

Symptomatology

Being a soil-borne pathogen, *Fusarium udum,* the fungus enters the host vascular system at root tips through wounds leading to progressive chlorosis of leaves, branches, wilting and collapse of the root system (Jain and Reddy 1995). Although the infection occurs in the early seedling stage, symptoms are not visible until later in crop developmental stages (Reddy *et al.*, 1990; Hillocks *et al.*, 2000). The initial visible symptoms are loss of turgidity in leaves and interveinal clearing. The leaves shows slight chlorosis and sometimes becomes bright yellow before wilting (Reddy *et al.*, 1990). Partial wilting of the plant as if there is water shortage even though the soil may have adequate moisture distinguishes this disease from termite damage, drought, and phytophthora blight that all kill the whole plant. Leaves are also retained on wilted plants. Partial wilting is associated with lateral root infection, while total wilt is due to tap

root infection (Nene, 1980; Reddy *et al*., 1993). The most initial characteristic internal symptom is a purple band extending upwards from the base of the main stem. The xylem develops black streaks, and this results in brown band or dark purple bands on the stem surface of partially wilted plants extending upwards from the base visible when the main stem or primary branches are split open (Reddy *et al*., 1990; Reddy *et al*., 1993). This band is more easily seen in pigeonpeas with green stems than in those with coloured stems. The intensity of browning or blackening decreases from the base to the tip of the plant. Sometimes, branches (especially lower ones) dry, even if there is no band on the main stem. These branches have die-back symptoms with a purple band extending from tip downwards, and intensive internal xylem blackening (Reddy *et al*., 1993). When young plants (1-2 months old) die from wilt, they may not show the purple band symptom, but have obvious internal browning and blackening.

Pathogenic races

The existence of variants/races of *F. udum* has been reported and has been cited as a major drawback in the development of pigeonpea varieties resistant to Fusarium wilt (Okiror and Kimani, 1997). *F. udum* isolates from the same site or diverse geographical origins have been shown to exhibit high variability in cultural characteristics (Reddy and Chaudhary, 1985; Gaur and Sharma, 1989) and virulence or pathogenicity on pigeonpea genotypes (Soko et al., 1995 ; Baldev and Amin, 1974; Shit and Gupta, 1978; Nene *et al*., 1981; Okiror, 1986; Gaur and Sharma, 1989; Okiror and Kimani, 1997; Mayer *et al*., 1998; Kiprop *et al*., 2002; Parmita *et al*,. 2005). Baldev and Amin (1974) tested 10 isolates of *F. udum* from various sources on 10 pigeonpea lines. Only three genotypes showed resistance to all the isolates. They also characterized these isolates as the races of this fungus. In Malawi when 60 isolates were inoculated onto the highly susceptible pigeonpea line ICP 2376, all but seven isolates were pathogenic (Soko *et al*., 1995). In a study to verify diversity in *F. udum* on pigeonpea in Kenya using several isolates of the fungus, Okiror and Kimani, (1997) reported strong differences in growth habit, morphology and high variability in terms of their attack on various test cultivars used; and concluded that the isolates are true variants of the pathogen. Similar observations were made by Gaur and Sharma (1989) using 18 pigeonpea varieties against seven isolates of *F. udum* from India and Okiror and Kimani (1997) using six pigeonpea genotypes against 12 isolates of *F. udum* from Kenya. Kiprop *et al.* (2002) observed differential reactions of seven pigeonpea varieties to seventeen different isolates of *F. udum* and concluded that five virulent groups exist among Kenyan isolates. This variability was confirmed by Songa *et al.* (1995) through field trials. Songa *et al.* (1995) found that pigeonpea line ICP 9145, which was wilt resistant at Katumani (Kenya), ICRISAT Asia Centre (India), and Malawi was highly susceptible (71% wilt) at Kiboko (Kenya). Variability of fusarium wilt reactions between countries and even sites within the same country is due to existence of different virulent isolate and environmental influence (Songa *et al*., 1995; Hillock *et al*., 2000). The high variation in cultural and morphology characteristics of this pathogen could be due to environmental conditions, age of the isolates, subculturing, method of storage and culturing conditions Kiprop *et al.* (2002). However, according to Okiror and Kimani (1997) and Kiprop *et al.* (2002) the wide variations in virulence (pathogenecity) to different genotypes of pigeonpea among *F. udum* isolates could be due to environmental conditions and inoculation techniques used. More work need to be done to confirm these reports.

There has been conflicting reports on the diversity of the pathogen using molecular markers. Conflicting reports on the relationship between cultural characteristics and the virulence of the isolate are also available. While studies of genetic diversity using isozyme markers revealed low variation in *F. udum* isolates (Shit and Sen Gupta, 1980; Okiror and Kimani, 1997), Kiprop *et al.* (2002) using AFLP reported high variability. Kiprop

et al. (2002) using 56 isolates from different pigeonpea growing areas in Kenya observed that cultural characteristics of *F. udum* appeared to be independent of aggressiveness and no relationship between aggressiveness and geographical origin of the isolates used. Similar reports using isozyme markers were made by Okiror and Kimani (1997) who found no relationship between protein profiles of 12 isolates of *F. udum* from three districts in Kenya and their virulence. However, Shit and Sen Gupta (1978) reported that isolates of *F. udum* producing luxuriant mycelia growth were weakly to moderately pathogenic (or aggressive). In order to establish the extent of genetic variation of this economically important fungus and relationships with genetic and pathogenic traits, more isolates from other countries and/ or geographical origins should be assayed using other DNA based molecular techniques.

The available reports on the differential reactions of certain pigeonpea genotypes to different isolates raises issues on whether isolates qualify to be called races of *F. udum* or not. In the evaluation of genotypic reaction of fusarium wilt conducted in wilt sick plots in Kiboko (Kenya), Ngabu (Malawi) and Ilonga (Tanzania), Gwata *et al.* (2006) recorded different wilt incidences at Kenya (52.7%) and Malawi (1.7%) for genotype ICEAP 00053 and concluded that there was a differential host response to the disease by the genotype between the two locations. Similar findings in a similar study involving an exotic cultivar (ICP 9145) screened for resistance to the Fusarium wilt disease in Kenya and Malawi were reported (Reddy *et al.*, 1990). According to Gwata *et al.* (2006) these differential host response observed for genotype ICEAP 00053 and ICP 9145 between the two locations (countries) suggests that probably, at least two different pathogenic races of the disease exists in the African region. Considering the mode of spread, more studies need to be done to confirm the existence of these two pathogenic races because the fact that *Fusarium* wilt may carry-over as a contaminant of pigeonpea seed (Nene 1981) suggests that there may also be strains even from other different countries in the Kiboko (Kenya) and Ngabu (Malawi) isolates. However, apart from studies aimed at characterizing *F. udum* isolates (Kiprop *et al.*, 2002), there are few reports attempting to characterize the races of *F. udum*. Most of the available reports relates different isolates to different races (Gwata *et al.*, 2006). This needs further investigation to confirm whether an isolate should be the same as a race.

Factors affecting infection and spread

Effect of Temperature on the development of Fusarium wilt

Temperature has been cited as one of the weather related factors that favour the development of the wilt disease (Singh and Bhargava, 1981; Agrios, 2005; Ziska and Runion, 2007; Chand and Khirbat, 2009). In pigeonpea, Singh and Bhargava (1981) observed highest fungal population at the soil temperatures between 20-30°C. In a study with chick pea cultivars, Mina and Dubey (2010) reported that the optimum temperature range for growth and sporulation of the pathogen was 28–30°C. This supported the earlier observations of Landa *et al.* (2001) that optimum growth of *F. oxysporum* f. sp. *ciceris* was at 24.5–28.5°C. Observations by Landa *et al.* (2006) showed that chickpea cultivar was moderately resistant to *F. oxysporium* f. sp. *ciceris* when grown in temperature regime of 21–24°C, but highly susceptible at a temperature regime of 25–27°C. Cooke and Baker (1983) in their review of the biological control of plant diseases, noted that the growth of Fusarium wilt pathogens is generally maximimal at 28°C, inhibited above 33°C, and not favoured below 17°C. In a physiological study with *F. oxysporum f.sp. cubense* isolates, the temperature assay revealed that the optimum temperature was 25°C for all the isolates, no growth was observed at temperatures of 5 and 40°C, while very little growth was observed at 10 and 35°C (Groenewald, 2006). Navas-Cortés *et al.,* (2007) through modelling reported positive correlation between wilt severity and soil temperature.

However, according to Ben-Yephet and Shtienberg (1997), the effect of temperatures on wilt occurrence may vary in different pathosystems.

Nutrient status of the soil

The stage of growth, decrease or quiescence of Fusarium population in soil depends on the ecological balance and nutrient availability. Nene, (1981) reported that the persistence of this pathogen is influenced by soil type and nutrient status. High levels of nitrogen fertilization in agricultural soils generally lead to an increase in Fusarium wilt development (Wolts and Engelhard, 1973; Wolts and Jones 1973). Studies have also shown that the form of nitrogen in the soil is important in the development of fusarium wilt disease (Wolts and Jones, 1973; Groenewald, 2006). Wolts and Jones (1973) reported that *F. oxysporum* cultured on ammonium nitrogen was more virulent than the same fresh weight of the organism cultured on nitrate nitrogen. In a study using *F. oxysporum f.sp. cubense* (*Foc)*, Groenewald (2006) observed that *Foc* grew significantly better on the NO_3-N medium invitro than on a NH_4-N medium. Similar results were observed by Walker (1971) who reported that high nitrogen and low potassium favoured the disease, while low nitrogen and high potassium retarded disease development. However, in a different study, Byther (1965), observed reduced disease incidence in the field fertilized with NO3-N compared to fields fertilized with NH_4^+-N. Effects on nitrate and ammonium sources on disease were apparently related to soil PH effects. Nitrate causes an elevation in soil PH while ammonium causes a reduction (Wolts and Jones, 1973). The nitrate form of nitrogen becomes increasing unfavourable for the disease with increasing rate of application, while the ammonium form becomes more favourable as the nitrogen application rate is increased. Relatively low levels of calcium appear more conducive to disease than normal levels.

Soil and soil pH

The amount of wilt incidence is influenced by water retentive nature of the soil and the disease is favoured by slightly acidic and alkaline soils with sand content of more than 50% (Upadhyay and Rai, 1992; Hillock *et al*., 2000). Shukla (1975) observed more *Fusarium* wilt inoculum in sand (94%) than in heavy black cotton soil (18%). However, some soils are suppressive to the pathogen due to their physico-chemical characteristics (Upadhyay and Rai, 1992). Sugha *et al*. (1994), found that a higher soil PH reduced the disease incidences. Lower disease incidence associated with a higher PH was due to decreased availability of the micronutrient that are essential for the growth, sporulation and virulence of the pathogen (Jones *et al*., 1989).

Soil antagonists

The susceptibility pigeonpea cultivars to Fusarium wilt is increased by the presence of certain nematodes in the soil (Hillocks *et al*. 2000). The association between Fusarium wilt and root-knot nematodes is well established (Hillocks and Songa, 1993; Marley and Hillocks 1994; Marley and Hillocks 1996). In India, reports have shown that the cyst nematode, *Heterodera cajani* (Hasan 1984; Sharma and Nene, 1989) and reniform nematode, *Rotylenchulus reniformis* (Sharma and Nene, 1990; Jain and Sharma, 1996) increase susceptibility to the disease. Its growth is also influenced by soil antagonists especially the bacterium *Bacillus subtilis* that produces the antibiotic bulbiformin (Pursey, 1989). The microflora have also been reported to affect the pathogenecity of the fungus (Upadhyay and Rai, 1987).

Susceptible pigeonpea cultivar

The disease begins in a field in a small patch which enlarges with each successive year that a susceptible cultivar is grown. *Fusarium* wilt is not seed borne but it may be carried as a contaminant of pigeonpea seed (Upadhyay and Rai, 1983). This may explain why wilt is common in areas where the crop is grown year after year making it more devastating in small-scale farmers who retain their seeds.

Others

The main means of spread in a field is along the roots of infected plants, movement of contaminated soil, propagules carried in irrigation water, or rain water run-off. Termites also act as agents of dissemination. According to Okiror (1986), wilting suddenly appears in ratooned crops probably because of the continued use of ratooning knife which acts as a carrier of inoculum from plant to plant.

Inheritance

The knowledge of genetic inheritance is essential for formulation of strategy on how to transfer the genes into adapted susceptible varieties. Conflicting reports have been made on the inheritance of resistance to *Fusarium* wilt in pigeonpea. Resistance to fusarium wilt has been reported to be under the control of two complementary genes (Parmita *et al*., 2005), single dominant gene (Pawar and Mayee 1986; Pandey *et al*. 1996; Singh *et al*. 1998; Karimi *et al*. 2010), 2 genes (Okiror 2002), major genes (Sharma, 1986; Parmita *et al*., 2005), duplicate genes and even multiple factors (Mahotra and Ashok, 2004) and a single recessive gene (Jain and Reddy, 1995). Apart from dominant, recessive and complementary gene action on the control of Fusarium wilt (Kimani, 1991; Kotresh *et al*., 2006) has been reported. Dominant epistatic gene interaction and a single dominant gene play a significant role in controlling resistance to wilt (Parmita *et al*., 2005). Digenic and quantitative genes that are resistant to Fusarium wilt have also been observed (Odeny, 2001).

These differences may be due to several factors. First, the sources and genetic background of the resistant materials used in the above diferent studies were different and this could have contributed to the different findings. According to Odeny (2001), genes for wilt are controlled differently depending on the origin of the resistance source used in a particular cross and the background in which the gene is put. Secondly, the inoculation methods used were different. Some of the studies were carried out in the wilt boxes (Kimani, 1991; Okiror, 2002) or field (Jain and Reddy, 1995). In the field, the environmental and edaphic factors may influence both the disease severity and the expression of the resistance.

Disease control methods

Cultural method

Several cultural control practices are recommended for restricting the severity of the *Fusarium* wilt of pigeonpea. Crop rotation with sorghum [*Sorghum bicolour* (L.) Moench*]*, tobacco (*Nicotiana tabacum* L.), or castor (*Ricinus communis* L.) every three years has been found to free pathogen completely from the field (Verma and Rai, 2008). Pigeonpea intercropped with sorghum had only incidence of 24% wilt against 85% in sole crop treatment (Natarajan *et al*., 1985). One-year break with either sorghum or fallow reduced wilt to below 20% (Verma and Rai, 2008). Application of nitrogen in form of farmyard manure and green manuring with *Crotalaria juncea* also reduce the incidence of wilt disease considerably (Upadhayay and Rai, 1981; Verma and Rai, 2008). Upadhyay and Rai (1981) reported a significant reduction in pigeonpea wilt incidence under mixed cropping with *Crotalaria medicaginea*. Solarisation of the field during summer period reduces the *Fusarium* inoculum (Reddy *et al*., 1993). However, limited studies have been conducted to understand the effect of cultural practices such as intercropping and rotation on the disease, with the aim of developing integrated disease management practices.

Effects of chemical control on Fusarium Wilt in pigeonpea

Several chemicals have been recommended for the management of *Fusarium* wilt (Singh, 1998). Seed treatment with a mixture of benomyl and thiram at equivalent rates completely eradicate the fungus (ICRISAT, 1987; Reddy *et al*., 1993). Ingole *et al*. (2005) also observed that a combination of carbendazim + thiophanate (0.15 + 0.10%) was effective in reducing the *Fusarium* wilt. Seed treatment with 4g *Trichoderma viride* formulation

+ 3 g thiram kg^{-1} seed and application of 2 kg *T. viride* formulation with 125 kg farm yard manure ha^{-1} has also been reported to control the disease (Verma and Rai, 2008). Addition of boron (Bo), manganese (Mn) or zinc (Zn) and methyl bromide (CH_3Br) to the soil effectively controls *Fusarium* wilt (Perchepied and Pitrat, 2004). This report was supported by Mandhare and Suryawanshi (2005) who recommended the application of *Trichoderma* as a seed treatment and soil application for managing *Fusarium* wilt of pigeonpea. Sinha (1975) observed a significant control of the disease by Bavistic applied as a soil drench at $2gkg^{-1}$ of soil days before inoculation of pigeonpea with *Fusarium udum*. Antibiotics griseofulvin and bulbiformin have been reported to be effective in controlling the disease. Control of the pathogen by organic amendments and by application of trace elements to the soil has also been reported (Reddy *et al*. 1990). However, none of the fungicides have been found to give adequate protection against *Fusarium* wilt disease (Lemanceau and Alabouvette, 1993; Pandey *et al*., 1996; Singh 1998) as the pathogen is primarily a soil inhabitant. Besides killing the non-target beneficial microorganisms in soil, the frequent application of fungicides to the soil causes environmental hazards causing water and soil pollution.

Biological control

In view of the adverse effect of fungicides to the environment and increasing interest in sustainable agriculture, biological control has been reported as an attractive possibility for management of soil-borne plant pathogens. Reports have shown that supplementing the soils with fungal or bacterial antagonists reduces incidences of *Fusarium* wilt (Bapat and Shar, 2000; Singh *et al*., 2002; Anjaiah *et al*., 2003; Mandhare and Suryawanshi, 2005; Maisuria *et al*., 2008). Numerous rhizobacteria have been used as biocontrol agents (Pusey, 1989; Upadhyay, 1992; Bapat and Shar, 2000; Siddiqui *et al*., 2005; Siddiqui, 2006; Siddiqui and Shakeel, 2007). Soil amendment with *Trichoderma harzianum* at all pathogen levels has been reported to give a disease control of 22% -61.5% (Prasad *et al*. 2002). Studies on antagonism, found that *Aspergillus niger*, *Aspergillus flavus*, *Micromonospora globosa* and *Aspergillus terreus* highly suppressed the population of *F. udum* (Upadhyay and Rai, 1981). In a different study on tomatoes (*Lycopersicon esculentum*), Khan and Khan (2002) observed that root-dip application of *Bacillus subtilis, Pseudomonas fluorescens, Aspergillus awamori, Aspergillus niger* and *Penicillium digitatum* resulted in significant decline of *Fusarium* oxysporum f.sp lycopersici population in the rhizosphere. In a bio-control experiment, Anjaiah *et al*. (2003) reported that inoculation of pigeonpea and chick pea seeds with *Pseudomonads aeruginosa* (PNA1) significantly reduced the incidence of *fusarium* wilt in naturally infested soil. Soil antagonists are also known to suppress the development of wilt through induction of resistance (Upadhyay and Rai, 1981; ICRISAT, 1987; Upadhyay, 1992). For large scale management, Mahesh *et al*. (2010) recommended integrated management (systemic fungicide, biocontrol agent and FYM) as the most effective treatment of *Fusarium udum*.

Host plant resistance

The deployment of cultivars with resistance to Fusarium wilt remains the most effective means of control. The search for sources of resistance to wilt in pigeonpea began as early as 1905 in India. Subsequently screening has been conducted in many locations in India, Malawi and eastern African. Through evaluation of local landraces, extensive world collections, introductions and segregating populations of pigeonpea, resistant/tolerant pigeonpea lines have been identified (Kimani, 1991; Kimani *et al*., 1994; Songa *et al*., 1995; Silim *et al*., 2005; Gwata *et al*., 2006; Karimi *et al*., 2010). The release of many resistant lines indicates the abundance of available resistance to Fusarium wilt within the genus *Cajanus*. The most successfully adopted wilt-resistant cultivar in Africa was ICP 9145 which in the mid 1990s accounted for around 20% of pigeonpea production

in Malawi (Babu *et al*., 1992; Reddy *et al*., 1992). According to Hillocks *et al*. (2000), the resurgence of pigeonpea wilt as a problem in Malawi, has been due to a combination of the lack of a sustainable seed production system to make ICP 9145 widely available to farmers, introgression between local susceptible types and ICP 9145, nematode-induced susceptibility and consumer preference for the cooking qualities of local, wilt susceptible cultivars. In Kenya where ICP 9145 has also been tested, it has not shown the high level of wilt resistance expected. This may be due to a loss of resistance as a result of segregation in ICP 9145 or some other environmental factor in Kenya.

Kenyan elite germplasm ICEAP 00040 is another widely adopted and adapted variety in Africa. Observations from wilt-sick plots (Bayaa *et al*., 1997) at Kiboko (Kenya), Ngabu (Malawi) and Ilonga (Tanzania) research stations where the disease pressure was considered to be high, indicated that disease incidences for ICEAP 00040 were consistently low (<20.0%) at all three locations (Gwata *et al*., 2006). Because of its ability to withstand the high disease pressure, wide adaptability and high yield potential, ICEAP 00040 has been attractive to pigeonpea farmers in the region extending from the semi-arid lowlands of eastern Kenya (5°S) to Mozambique (25°S) and was subsequently released in 1995 for commercial production in Kenya and later in 2003 both in Malawi and Tanzania (Silim *et al*., 2005). ICEAP 00040 has large, cream grains that are preferred by the farmers and markets in the region.

In spite of stupendous success achieved through sustained breeding efforts over the last decade as evidenced from commercially accepted resistant pigeonpea varieties (Reddy *et al*., 1992; Gwata *et al*., 2006), very few resistant cultivars are with the farmers. This is due to lack of effort to breed varieties that are both wilt resistant and farmer acceptable. For instance, in India, wild relatives have been reported to possess many agronomically important traits such as resistance to pests and diseases (Reddy *et al*., 1996; Sharma *et al*., 2003), salinity tolerance (Subbarao *et al*., 1991) and high protein content (Saxena *et al*., 1996), all of which would be useful in cultivated pigeonpea. However, no effort has been made to transfer these useful genes from wild relatives to cultivated pigeonpea. Specific cultivar improvement has been difficult due to the limited knowledge on the inheritance of important traits and lack of understanding on the level of inter- and intra-specific genetic diversity. As different needs and opportunities arise, pigeonpea breeders need to incorporate new genetic sources using various breeding methods aided with modern tools such as biotechnology. Diffusion of the improved varieties has also been limited by lack of good quality seeds. The use of own saved seed by farmers makes production of improved varieties (self pollinated crops like pigeonpea) seeds uneconomical, thus undermining the incentives for private sector investment in commercial production and marketing of such seeds.

Future research needs

Development of well adapted resistant pigeonpea varieties offers the most efficient and economical method for control of Fusarium wilt. Several genotypes within the pigeonpea germplasm have been identified. The resistant genotypes need to be combined with high yield and consumer-preferred agronomic traits so that they can be adopted by the farmers. Knowledge on mode of inheritance for both resistance to fuasium wilt and other agronomic traits need to be well understood. Mapping of the *Fusarium* wilt resistance genes in the already identified resistant lines is recommended. This will help shorten the development of the resistant pigeonpea cultivars and the pyramiding of the wilt resistance with other traits, particularly through the use of marker-assisted selection.

Studies to characterize the races of *F. udum* in pigeonpea in Africa should be done. This will help identify the presence of different races of *Fusarium udum*. It will also confirm whether the isolates are the same as races.

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