REVIEW

SABRAO Journal
of Breeding and Genetics
45 (3) 341-370, 2013

NARROWING YIELD GAPS THROUGH GENETIC IMPROVEMENT FOR FUSARIUM WILT RESISTANCE IN THREE PULSE CROPS OF THE SEMI-ARID TROPICS

A. K. CHOUDHARY[1,*], S. KUMAR[2], B. S. PATIL[3], J. S. BHAT[3], M. SHARMA[4], S. KEMAL[2], T. P. ONTAGODI[5], S. DATTA[6], P. PATIL[6], S. K. CHATURVEDI[6], R. SULTANA[7], V. S. HEGDE[8], S. CHOUDHARY[9], P. Y. KAMANNAVAR[5] and A. G. VIJAYAKUMAR[5]

[1]IIPR Regional Research Centre cum Off-Season Nursery, Dharwad 580 005, Karnataka, India
[2]International Centre for Agricultural Research in Dry Areas (ICARDA), Rabat, Morocco
[3]IARI Regional Research Centre, Dharwad 580 005, Karnataka, India
[4]International Crops Research Institute for Semi-Arid Tropics (ICRISAT), Patancheru 502 324, India
[5]University of Agricultural Sciences (UAS), Dharwad 580 005, Karnataka, India
[6]Indian Institute of Pulses Research (IIPR), Kanpur 208 024, Uttar Pradesh, India
[7]Bihar Agricultural University (BAU), Sabour 813 210, Bihar, India
[8]Indian Agricultural Research Institute (IARI), New Delhi 110 012, India
[9]Deen Dayal Upadhyay Gorakhpur University, Gorakhpur 200 001, Uttar Pradesh, India
*Corresponding author's email: akiipr23@yahoo.com

SUMMARY

Chickpea, pigeonpea and lentil constitute the major component of low-input agriculture in the semi-arid tropics (SAT). These pulses play an important role in alleviating malnutrition by way of forming an inseparable component to the dietary proteins for millions of peoples living below poverty line in SAT regions. The production and productivity of these food legumes is severely constrained by Fusarium wilt (FW). It has been reported to cause 100% yield losses, if affects them in the seedling stage. However, recent advances in the identification of races of the pathogen, screening techniques to identify FW resistant genotypes, genetic dissection and mapping and tagging of wilt resistance (WR) genes through molecular markers have resulted in the release of several wilt resistant varieties the world over. This has not only narrowed the gaps between potential and realized yields, but also minimized yearly fluctuations in production and productivity of these SAT pulses. However, the pace of advancements for WR in pigeonpea and lentil has not been comparable to those of chickpea. Even in chickpea, multiple gene pyramiding in a single variety is needed to make it resistant to multiple races of Fusarium. In this review paper, we have attempted to document the progress made along with future outlook towards FW resistance in these three pulses of SAT regions.

Keywords: Fusarium wilt, screening techniques, gene tagging, wilt resistance, molecular markers, gene pyramiding

Manuscript received: September 1, 2013; Decision on manuscript: November 21, 2013; Manuscript accepted: November 22, 2013.

Communicating Editor: Bertrand Collard

INTRODUCTION

Pulses are legumes harvested almost exclusively for dry grains. They are important and balanced source of dietary proteins when complemented with cereals for millions of rural and urban poor people the world over where animal products are not consumed due to religious doctrine and/ or

due to unaffordable prices. Pulses are cultivated in the semi-arid tropics (SAT) of majority of the developed and developing countries. In some parts of the world including India, which is the largest producer and consumer of pulses, these are also used as vegetables. Green as well as dry plant parts of pulses are used as an important source of feed and fodder in many crop-livestock production systems of SAT regions. Furthermore, they have been sustaining cereal based cropping systems through biological nitrogen fixation and carbon sequestration since time immemorial.

Pulses form an important component of typically low input agriculture in SAT regions. Chickpea (*Cicer arietinum* L.), pigeonpea [*Cajanus cajan* (L.) Millspaugh] and lentil (*Lens culinaris* Medikus subsp. *culinaris*) are the major pulse crops in these areas. Resource poor small and marginal farmers hardly provide better-than-average-management practices to these crops. These crops are also exposed to harsh, erratic and unpredictable climatic conditions of these regions in addition to biotic stresses imposed by several pathogens and insect pests. This has not only slowed down their productivity growth through genetic means, but also caused yield instability leading to shifts into other crops mainly cereals.

Among the biotic stresses, Fusarium wilt (FW) disease is considered as the most severe yield limiting factor in chickpea, pigeonpea and lentil. In each of these pulse crops, this disease is caused by a distinct species of the pathogen *Fusarium*. The FW affects these pulses from seedling to the stage of maturity, causing heavy losses both in quantity and quality. Chemical and cultural control measures are not very effective in FW management; however, resistance breeding has been very efficient, and many wilt resistant varieties in these pulses have been released and adopted by farmers in SAT regions. Besides, further advancements have been made towards identification and tagging and pyramiding of wilt resistance (WR) genes. Although some old review on individual pulse crop is available, systematic review for wilt resistance at a single platform in these three pulse crops is lacking. The present review is an attempt to document all the progress made and constraints experienced so far along with future outlook for improving wilt resistance in chickpea, pigeonpea and lentil.

SAT PULSES AND IMPORTANCE OF WILT DISEASE

Chickpea

Chickpea is one of the important food legumes in SAT regions. Globally, it is cultivated in more than 57 countries and ranks second in acreage after dry bean. However, it stands third in production following dry bean and peas with the productivity of about 913 kg ha^{-1} (Figure 1a) (FAO, 2012). South and Southeast Asian countries account for more than two-thirds of the total chickpea production. Chickpea is the only cultivated species under the genus 'Cicer', and has $2n = 2x = 16$ chromosomes with a relatively small genome size of 738.09 Mbp (Varshney *et al.,* 2013). *Macrosperma* (Kabuli) and *Microsperma* (Desi) are the two distinct types of chickpea with the production share of 25% and 75%, respectively (Soregaon, 2011). Plant types vary from spreading to erect with non-determinate (NDT) growth habit. However, recently a plant type with determinate growth habit has also been reported (Hegde, 2011). The chickpea is a good source of protein (24.6%), carbohydrate (64.6%) and vitamins (Abu-Salem and Abou, 2011). It also provides calcium, magnesium, potassium, phosphorus, iron, zinc and manganese (Ibrikci *et al.,* 2003). This food legume has diversified uses, and presently as many as 140 countries are importing chickpea (Gaur *et al.,* 2012). Besides this, it can also fix up to 140 kg N ha^{-1}, leaving considerable residual nitrogen for the succeeding crop.

The biotic and abiotic constraints have significantly widened the gap between potential (5.0 t/ha) and realized (0.8 t/ha) seed yield of chickpea, and this gap has been persisting over the years (Pande *et al.,* 2006). Besides, yearly fluctuations in the average yield of chickpea have also been observed.

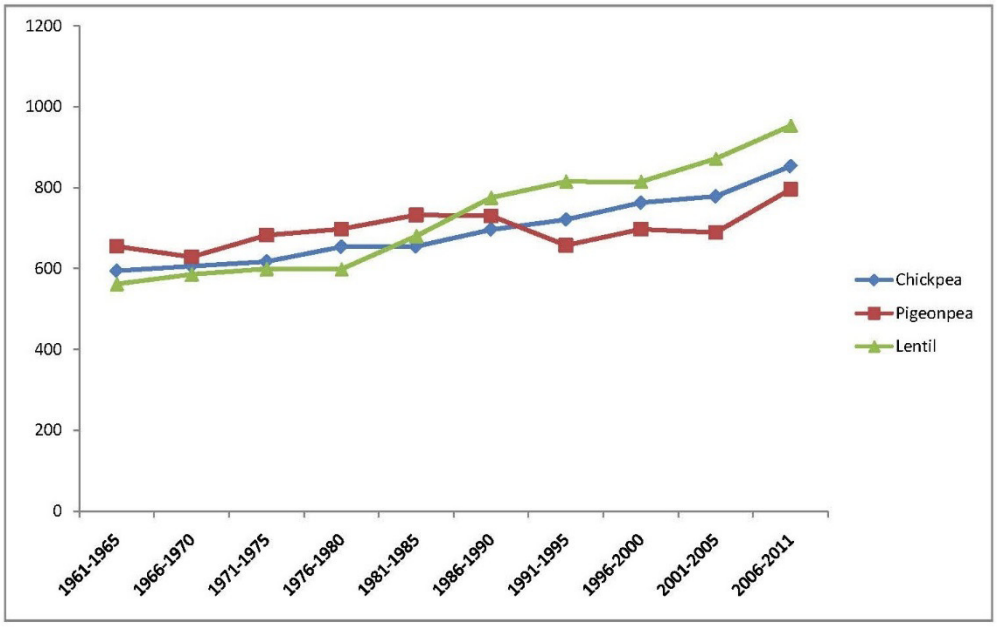

Figure 1a. The global trends in the productivity of three SAT pulses over the last 50 years.

The key biotic constraints contributing to wide yield gap are Fusarium wilt (FW), root rot complex, Ascochyta blight (AB) and Botrytis gray mould (BGM), of which FW and AB are the most devastating diseases, causing heavy loss in chickpea production (Gaur *et al.,* 2012). In central and southern India, FW ranks first among the fungal diseases of chickpea (Ghosh *et al.,* 2013). The average annual yield losses in chickpea due to FW ranged from 10% to 100% (Table 1) depending on the stage of infection, the environmental conditions and susceptibility of the cultivar (Halila *et al.,* 2010; Soregaon and Ravikumar, 2012).

Pigeonpea

Pigeonpea is an often cross-pollinated food legume with 2n = 2x = 22 chromosomes and genome size of 833.07 Mbp (Varshney *et al.,* 2012a). Its wide adaptation to a range of environments and cropping systems is attributed to its large variation (90-300 days) for maturity period (Choudhary *et al.,* 2011) and plant types. It is endowed with protogyny and weak self-incompatibity that have direct significance on the mating system of this crop (Choudhary *et al.,* 2012). Globally, pigeonpea is cultivated on 4.75 million ha area with the production and productivity of 3.68 million t and 774 kg ha^{-1}, respectively during 2010 (FAO, 2012). Pigeonpea, which is primarily an important food crop in India and East Africa, is an important source of dietary protein to the vegetarians. Besides it also ensures high supply of vitamin B, carotene, and ascorbic acid (Miller *et al.,* 1956), which are otherwise deficient in cereals. The use of immature seeds as the fresh vegetable is very common in Gujarat, Maharashtra and Karnataka and many tribal areas of India (Saxena *et al.,* 2010). It is known to improve the soil fertility by fixing nitrogen @ 40 kg ha^{-1} and solubilizing the soil-bound phosphorus. In addition to this, it also improves soil health by adding organic material to the soil (Whiteman and Norton, 1980). The deep root system and perhaps presence of waxy substances on the leaves (Choudhary and Nadarajan, 2011) confer adaptive value to pigeonpea, making it a drought tolerant crop for low input agriculture in drought prone environments of SAT regions.

Table 1. Economic losses due to Fusarium wilt in three SAT pulses.

Crop	Stage of infection	Economic loss (yield/value)	Country	Reference
Chickpea	Seedling stage to pre- pod stage	61%	India	Nema and Khare (1973)
	Flowering stage	43%	India	Nema and Khare (1973)
	Early onset of wilting	77-94%	India	Haware and Nene (1980)
	Medium onset of wilting	58-83%	India	Haware and Nene (1980)
	Late onset of wilting	24-65%	India	Haware and Nene (1980)
	Seedling to pod-filling	10 - 90%	India	Srivastava *et al.* (1984)
	Stage not specified	10-15%	India	Haware *et al.* (1990)
	Stage not specified	48.29%	India	Kumar and Bourai (2012)
	Stage not specified	Up to 95%	Spain	Carranza (1965)
	Stage not specified	10-90%	Spain	Jimenez-Diaz *et al.* (1989)
	Stage not specified	30-99.7%	Spain	Navas-Cortes *et al.* (2000)
	Flowering stage	17%	Iran	Manucheri and Mesri (1966)
	Stage not specified	10-50%	Pakistan	Khan *et al.* (2002)
	Seedling stage	100%	Australia	Bretag (1982)
	Stage not specified	40%	Tunisia	Bouslama (1980)
	Seedling stage	100%	Tunisia	Halila and Strange (1996)
Pigeonpea	Pre-pod stage	100%	India	Kannaiyan and Nene (1981)
	before pod production	99.9%	India	Kannaiyan and Nene (1981)
	Pod maturity	67.1%	India	Kannaiyan and Nene (1981)
	Pre-harvest stage	29.5%	India	Kannaiyan and Nene (1981)
	Stage not specified	US $36 million	India	Kannaiyan *et al.* (1984)
	Seedling to pod filling	10-100%	India	Reddy *et al.* (1990)
	Stage not specified	US $71 million	India	Reddy *et al.* (1993)
	Stage not specified	20–25%	India	Dhar and Reddy (1999)
	Stage not specified	29.34%	India	Kumar and Bourai (2012)
	Stage not specified	36.6% (0-90%)	Malawi	Kannaiyan *et al.* (1984)
	Stage not specified	50-100%	Malawi	Soko (1992)
	Stage not specified	15.9% (0-90%),	Kenya	Kannaiyan *et al*. (1984)
	Stage not specified	US $ 5 million	Eastern Africa	Kannaiyan *et al*. (1984)
	Stage not specified	20.4% (0-60%)	Tanzania	Kannaiyan *et al.* (1984)
	Stage not specified	96%	Tanzania	Mbwaga (1995)
Lentil	Seedling to pod filling stage	50-78%	India	Khare et al. (1979), Agrawal *et al.* (1993)
	Seedling stage	100%	India	Khare (1981)
	Stage not specified	29.98%	India	Kumar and Bourai (2012)
	Stage not specified	5-72%	Syria	Bayaa *et al.* (1986)
	Stage not specified	8.8% (for 10% wilted plants)	Syria	Erskine and Bayaa (1996)

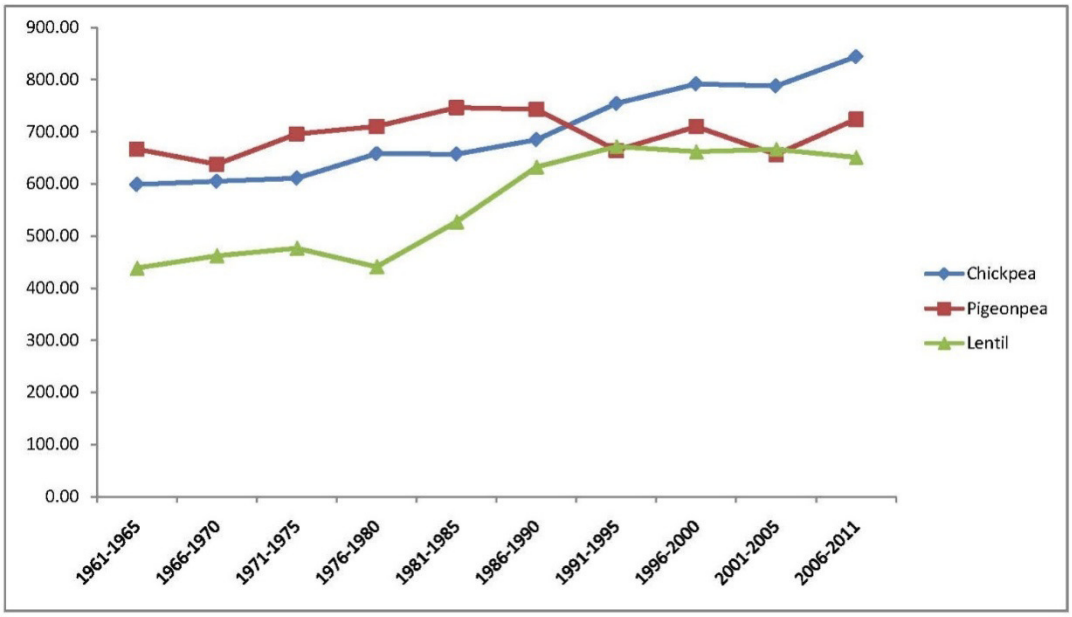

Figure 1b. The productivity trends of three SAT pulses in India over the last 50 years.

Despite its significant importance in sustainable agriculture and continued breeding efforts towards its genetic improvement, the global productivity of pigeonpea (Figure 1a) has remained static over the last three decades (Varshney *et al.,* 2013).

The gap between potential and on-farm yield is ascribed mainly to the prevalence of various abiotic (Choudhary *et al.,* 2011) and biotic factors together with the cultivation of pigeonpea in marginal lands with low input supply and lack of efficient management practices (Varshney *et al.,* 2012b). Wide year-to-year yield fluctuations have also been observed, which is mainly due to diseases and insect pests (Choudhary *et al.,* 2013). More than 100 pathogens, which include fungi, bacteria, viruses, nematodes and phytoplasma, attack pigeonpea; however, diseases such as FW, sterility mosaic (SM), Phytophthora stem blight (PSB) and Alternaria leaf blight (ALB) have been found economically important in SAT regions. Among the diseases, Fusarium wilt (FW) caused by *Fusarium udum* Butler, is the most important soil borne disease of pigeonpea, causing significant yield losses (Table 1) in India and Eastern and southern African countries (Kannaiyan and Nene, 1981; Okiror, 2002; Gwata *et al.,* 2006). This disease can occur at any stage of plant development, from seedling to the pod-filling stage (Choudhary, 2010). Depending upon the stage of occurrence, FW can cause grain yield losses ranging from 30-100% (Kannaiyan and Nene, 1981; Reddy *et al.,* 1990). The annual loss due to FW alone has been estimated about US $ 36 million in only India (Kannaiyan *et al.,* 1984). Similar losses due to FW have also been recorded in African countries (Hillocks and Khonga, 1996). Although some resistant donors and varieties have been identified and released, respectively, but the magnitude of success has been limited due to poor understanding of racial composition of the causal organism (*Fusarium udum*).

Lentil

The cultivated lentil is an ancient crop originated somewhere in the 'Fertile Crescent'. All *Lens* species are self-pollinated annual diploids with 2n = 2x = 14 chromosomes having haploid genome size of 4063 Mbp (Arumuganathan and Earle, 1991). It is cultivated in as many as 52 countries across the world with an area and

production of 4.08 million ha and 4.36 million t, respectively (FAO, 2012). Its wide-spread cultivation owes to its ability to produce a high-quality protein in drought-prone marginal environments. In India, lentil is cultivated on 1.48 million ha area with production and productivity of 0.98 million t and 660 kg ha^{-1}, respectively (Figure 1b). Among several biotic stresses affecting stability of production, FW is the most important. This vascular wilt, which is caused by *Fusarium oxysporum* f.sp. *lentis*, is a widespread disease of lentil with its report of occurrence from as many as 26 countries in South Asia, Sub-Saharan Africa and West Asia and North Africa (WANA) regions. It was first reported from Hungary (Fleischmann, 1937), and later on from many countries including India (Padwick, 1941), USA (Wilson and Brandsberg, 1965), Czechoslovakia (Ujevic *et al.,* 1965), USSR (Kotava *et al.,* 1965), France (Moreau, 1978), Turkey (Bayya *et al.,* 1998), Syria (Bayya *et al.,* 1986), Myanmar and Pakistan (Bahl *et al.,* 1993), Nepal (Karki, 1993), Ethiopia (Hulluka and Tadesse, 1994) and Egypt (El-Morsy *et al.,* 1997). The disease is known to cause economic yield losses in parts of WANA region, Sub-Saharan Africa and South Asia (Erskine *et al.,* 1994). In India, FW is the major factor limiting lentil production in the states of Uttar Pradesh, Madhya Pradesh, Himachal Pradesh, Bihar, West Bengal, Assam, Rajasthan, Haryana and Punjab (Agrawal *et al.*, 1993; Chaudhary *et al.,* 2009; 2010). Yield losses due to FW in lentil depend on the crop stage at the time of infection (Vasudeva and Srinivasan, 1952; Claudius and Mehrotra, 1973; Khare *et al.,* 1979), environment and crop variety. Wilt incidence at seedling stage can lead to a complete crop failure whereas at adult stage (flowering and podding) infection, the plants are able to produce some grain yield that could be shriveled. Wilt incidence as high as 50-78% has been reported in some fields of Madhya Pradesh (Khare *et al.,* 1979; Agrawal *et al.,* 1993). In west Asian countries like Syria, the yield losses range from 5-72% (Bayaa *et al.,* 1986). There is a strong correlation between wilt incidence and grain yield, estimating 8.8% yield loss for every 10% wilt incidence (Erskine and Bayaa, 1996).

PATHOGENIC VARIABILITY

Chickpea Fusarium wilt

The high genotypic and phenotypic variability in *Fusarium oxysporum* f. sp. *ciceri* (FOC) has been reported by several workers, indicating the possibility that this pathogen might have prophyletic nature. However, despite high variability in FOC isolates regardless of their pathotype, race and pathogenic origin, all isolates belong to the single vegetative compatibility group (Jimnez-Gasco *et al.,* 2004). Recent studies have indicated changes in the race scenario of FOC and existence of multiple races and/or new reactions (Dubey *et al.,* 2012; Sharma, 2013). The existence of FOC races was first reported in India. Four physiological races (races 1, 2, 3 and 4) were identified using 10 differential lines (Haware and Nene, 1982). Presently there are 8 races (Table 2) including 2 sub-classes of race 1 (race 1A from India and race 1B/C from Spain) (Trapero-Casas and Jimenez Diaz 1985; Jimenez Diaz *et al.,* 1993), race 0 and race 5 from Spain (Cabera de la Colina *et al.,* 1985) and Tunisia (Halila and Strange, 1996), and race 6 from California (Phillips, 1988). The 2 races, namely race 0 and race 6, were also reported from India (Rahman *et al.,* 1998; 2000); however, it still needs to be confirmed. Four races, i.e., 1A, 2, 3 and 4 are found in Indian sub-continent (Haware and Nene, 1982), whereas the remaining 4 races (races 0, 1B/C, 5 and 6) are found in the Mediterranean region and the USA (Halila and Strange, 1996; Jimenez-Gasco *et al.,* 2001). However, literature pertaining to the occurrence of race patterns in East Africa and Pakistan is scanty (Khalid Mahmood *et al.,* 2011). The 8 races are divided into 2 groups based on symptomatology of infected plants that is, yellowing and wilting syndromes (Trapero-Casas and Jimenez-Diaz, 1985). Six races (1A, 2, 3, 4, 5 and 6) cause wilting, and are economically more important than the other 2 races (race 0 and 1B/C) that cause only yellowing (Jimenez-Diaz *et al.,* 1993; Kelly *et al.,* 1994).

Pigeonpea Fusarium wilt

Pathogenic variability in *Fusarium udum* (FU) has been noticed under diverse conditions of edaphic and climatic factors (Gupta *et al.,* 1988). So far 5 variants (I, II, III, IV and V) of this pathogen have been identified and documented (Mishra, 2004; Tiwari and Dhar, 2011) using as many as 15 differentials. In India, the presence of pathogenic variability has also been substantiated through field trials performed over several locations and years under All India Coordinated Research Improvement Project (AICRP) on pigeonpea. Variant-I is widely prevalent in India (Choudhary, 2010). In north-east plains, Uttar Pradesh has recorded the presence of all the 5 variants, whereas Bihar has 4 variants. In south and central India, presence of only 3 strains (I, II and III and I, III and V, respectively) has been recorded (IIPR, 2008). In Kenya (Africa) also, pathogenic variability for FU has been observed through field trials (Songa *et al.,* 1995). Differential reactions of seven pigeonpea varieties to 17 different isolates of FU have shown 5 virulent groups among Kenyan isolates (Kiprop *et al.,* 2002) (Table 2).

Pathogenic variability and genetic diversity studies among Indian isolates of FU have been conducted using molecular markers such as randomly amplified polymorphic DNA (RAPD) and amplified fragment length polymorphism (AFLP). These studies have indicated the existence of three distinct pathogenic groups and a high degree of variability in pathogenicity (Sivaramkrishnan *et al.,* 2002; Sukumar *et al.,* 2012). However, further studies are required on the genetics of differential hosts and the variants in order to designate these variants or groups as 'races' (Tiwari and Dhar, 2011).

Lentil Fusarium wilt

Though *F. oxysporum* f. sp. *lentis* (FOL) is host specific, presence of genetic variability has been reported on the basis of reactions in host genotypes and pathogen morphology and cultural characters (Kannaiyan and Nene, 1978; Belabid *et al.,* 2004; Chaudhary *et al.*, 2009; Taheri *et al.,* 2010). Khare *et al.* (1975) reported eight strains of FOL, whereas Kannaiyan and Nene (1978) established seven strains (Table 2). However, no variation in virulence/aggressiveness was detected among these Indian strains that could play a major role in breaking the resistance of existing genotypes of lentil. Belabid *et al.* (2004) studied virulence and vegetative compatibility of 32 Algerian isolates of FOL, and grouped them as a single race. However, these isolates differed in their aggressiveness on susceptible lines. Study on 333 isolates from different states of India revealed 43 cultural and morphological groups (Chaudhary, 2008). On the basis of disease reactions against 7 lentil differentials, these isolates were grouped into three clusters. Similarly, variability analysis of 24 isolates collected from north eastern Indo-Gangetic plains using 40 RAPD and 12 SSR primer pairs revealed two sub-populations with little genetic variations (Datta *et al.,* 2009).

SCREENING TECHNIQUES TO IDENTIFY WILT RESISTANT GENOTYPES

The initial step to utilize host plant resistance (HPR) relates to the development of reliable and reproducible disease screening techniques to evaluate large numbers of germplasm accessions and breeding materials. Effective and efficient screening for resistance to soil borne pathogens such as *Fusarium* spp. calls for simulation of natural soil and environmental conditions and uniform inoculum load across all the plants of test genotypes to discriminate between resistant and susceptible genotypes. In general, screening under field and controlled conditions (green house and laboratory conditions) has been suggested to identify resistant genotypes for FW resistance in chickpea, pigeonpea (Pande *et al.,* 2012a; 2012b) and lentil (Kraft *et al.,* 1994; Alessandro *et al.,* 2006).

Field screening

The most common and widely used method for screening of FW resistant genotypes has been the wilt sick plot (WSP) method. The main advantage of WSP method is that it allows screening of a large number of genetic materials

under field conditions (Infantino *et al.,* 2006). Typical disease symptoms are the main criteria for evaluating breeding lines and establishment of WSPs, while the re-isolation of the causal organism is a confirmatory test. In WSP method, inoculum load needed to get the typical wilt symptoms can vary with race/variant, environmental conditions, crop and its maturity groups and ecotypes (e.g., *Desi* and *Kabuli* types of chickpea) type. For example, about 3,000 propagules per gram (ppg) of soil induced 100% FW mortality in the susceptible line 'ICC 4951' of chickpea (Ali *et al.,* 1994).

Table 2. Races/variants of *F. oxysporum* f. sp. *ciceris*, *F. udum* and *F. oxysporum* f. sp. *Lentis*.

Crop	Race/variant	Reported from	References
Chickpea	Races 1A, 2, 3 and 4	India	Haware and Nene (1982)
	Race 1A	India	Trapero-Casas and Jimenez Diaz (1985)
	Races 0 and 6*	India	Rahman *et al*. (1998)
	Races 1, 2, 3 and 4	India	Gurjar *et al*. (2009)
	Races 0 and 5	Spain	Cabera de la Colina *et al.* (1985)
	Race 1B/C	Spain	Jimenez Diaz *et al*. (1993)
	Races 0, 1A, 5 and 6	Spain	Jimenez-Gasco *et al*. (2003)
	Races 0 and 5	Tunisia	Halila and Strange (1996)
	Races 0, 1B/C, 5 and 6	Mediterranean region and USA	Halila and Strange (1996), Jimenez-Gasco *et al*. (2001)
	Races 0, 1B/C, 5 and 6	Mexico	Arvayo-Ortiz (2011)
	Races 0, 2 and 3	Turkey	Dolar (1997)
	Race 0, 2 and 3	Turkey	Bayraktar and Dolar (2012)
	Race 0	California (USA), Israel, Lebanon, Spain, Syria, Tunisia and Turkey	Jimenez-Diaz *et al*. (1993), Halila and Strange (1996)
	Race 1	California, Israel, Morocco	Jimenez-Diaz *et al*. (1993), Halila and Strange (1996)
	Race 5	California	Jimenez-Diaz *et al*. (1993), Halila and Strange (1996)
	Race 6	California, Israel, Morocco	Phillips (1988), Jimenez-Diaz *et al.* (1993), Halila and Strange (1996)
	Races 0, 2, 3 and 4	Ethiopia	Shehabu *et al*. (2008)
Pigeonpea	Variants I, II, III, IV and V#	India	Mishra (2004), IIPR (2008)
	Variants II, IV and V	India	Tiwari and Dhar (2011)
	Three distinct pathogenic groups	India	Sivaramkrishnan *et al*. (2002), Sukumar *et al.* (2012)
	Variant I	India	Asha *et al*. (2012)
	Three groups	Kenya	Okiror and Kimani (1997)
	Five virulent groups	Kenya	Kiprop *et al.* (2002)
	Two races	Nepal	Joshi (2001)
Lentil	Eight strains	India	Khare *et al.* (1975)
	Seven strains	India	Kannaiyan and Nene (1978)
	Single race	Algeria	Belabid *et al.* (2004)
	Three classes (based on 43 cultural and morphological groups)	India	Chaudhary (2008)
	Two groups	India	Datta *et al.* (2011)

*Needs confirmation, # Further studies required to designate these variants as races

Similarly, 3,283 ppg of FOC race 1 is found effective to discriminate between highly susceptible 'JG-62', late wilting 'K-850' and resistant 'WR 315'varieties of chickpea (Kraft *et al.,* 1994). However, inoculum equivalent to 1795 colony forming units (cfu) per gram of soil is required for screening *Kabuli* chickpea germplasm against FOC race 0 (Halila and Strange, 1997).

The procedures of the "field screening" for WR are almost similar in all the 3 pulses. The details of field screening have been described for pigeonpea (Nene *et al.,* 1981a), chickpea (Nene and Haware, 1980) and lentil (Kumar *et al.,* 2010). It involves planting of test genotypes along with a susceptible cultivar, which serves as an indicator line or the susceptible check after every 2-4 test entries to monitor uniformity of the inoculum in the plot. The widely used susceptible checks are 'JG 62' (a twin podded chickpea variety highly susceptible to all FOC races except race 0, and widely used susceptible check for race 1 to 4 in India), 'ICP 2376' or 'Bahar' (pigeonpea) and 'ILL 4605' (lentil). In addition, resistant genotype(s) (e.g., Maruti, WR 315 and ILL 5588 of pigeonpea, chickpea and lentil, respectively) should also be planted after every 10 rows to monitor if there are other pathogens that can confound the wilt reaction. Two most important sources of resistance in chickpea are the germplasm line 'WR 315' (ICC 11322) and the cultivar 'JG 74'. The former is resistant to all FOC races except race 3, while the latter is resistant to all races except race 2 (Haware, 1998). However, further refinements in field inoculation technique to ensure uniform spread of FW have been suggested for pigeonpea (Reddy *et al.,* 1990) and chickpea (Sharma *et al.,* 2005a). This modified technique produced near 100% wilt incidence in susceptible pigeonpea controls (checks) at ICRISAT, Patancheru. Recently stepwise identification of host plant resistance to diseases of chickpea and pigeponpea has been updated by Pande *et al.* (2012a, b).

For screening a large number of gemplasm lines against FW, WSPs have been developed in ICRISAT, ICARDA, and NARS systems of countries growing these crops. In India, WSPs have been created for all these three crops at IIPR, Kanpur, and at all the major centres of the AICRP on chickpea, pigeonpea and MULLaP (for lentil). Field screening of germplasm and cultivars for wilt resistance has been carried out widely for so many years, which has resulted in the identification of a number of wilt resistant genotypes and cultivars (Figure 2).

Figure 2. Screening of lentil germplasm in wilt sick plot at Tel Hadya, Syria. The picture shows highly susceptible check 'ILL 4605' along with resistant lines.

Screening under controlled conditions

Field screening, albeit widely used, has been criticized for many edaphic and climate factors are not under control, and involvement of other soil borne fungal pathogens and nematodes is also observed. Screening under controlled conditions in glasshouse is suggested to confirm the results of WSP method. This is particularly important for inheritance and molecular mapping and tagging studies using a well characterized race of the pathogen. Moreover, pathogenic diversity studies can be done under the controlled conditions that support genotypic information.

Greenhouse screening

Screening of chickpea germplasm in the greenhouse using pot culture method has been standardized (Nene *et al.,* 1980). Pot screening technique ensures 90% wilt in susceptible lines; however, soil compaction by repeated irrigation may affect the correlation between pot and field performance. Sub-irrigation of pots prevents compaction, keeps surface soil dry and simulates typical field conditions in pots (Phillips, 1988). Ratnaparkhe (1998a) used perlite instead of soil in pots and inoculated all the test plants simultaneously by pruning roots followed by dipping in spore suspension. Another most commonly used greenhouse screening technique is root dip inoculation technique both for chickpea and pigeonpea (Pande *et al.,* 2012a; 2012b). 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 ± 1 ^{0}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. Uninoculated seedlings transplanted in uninoculated sand/soil are used as control (Nene *et al.,* 1981b; Haware and Nene, 1994; Pande *et al.,* 2012b). 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 ^{0}C. After multiplying for 20 days, 200 g of this medium was mixed with 2 kg autoclaved red soil and placed in 15cm plastic pot and were incubated at 25-30 ^{0}C. After 2 days, 7-10 days old seedlings were transplanted in the pathogen infested pots. Wilt incidence was recorded 60 days after transplanting.

Laboratory screening

Laboratory screening technique to identify FW resistant genotypes in pigeonpea has been developed (Nene and Kannaiyan, 1982). It provides better control over the experiments. It consists of multiplication of single conidium cultures of FU on 100 ml PDB in 250 ml flask for 10 days at 25-30 °C. The contents of flasks are further diluted to a final inoculum concentration of 2.5%. Seven to ten days old 15 seedlings are transferred to glass tubes containing 20 ml inoculum. One non-inoculated seedling of each entry is kept in sterile distilled water to be used as the check for each line. The susceptible check (ICP 2671) is also used for comparing the test lines, which usually wilted in 7-10 days. Data recording for wilt incidence is performed after 15 days of inoculation. Non-inoculated seedlings can sustain their healthy state for more than 3 weeks.

Artificial screening techniques as developed by Tullu (1996) and Sharma *et al.* (2005a) in chickpea ensure uniform inoculum load at the same vegetative stage of test plants. In this technique, injury to roots prior to inoculation ensures that all inoculated plants have a nearly equal chance of infection (Sharma and Muehlbuer, 2007). By this technique, 21 asymptomatic and 25 resistant genotypes were identified from a core collection of 211 genotypes representing more than 16000 diverse chickpea germplasm accessions (Pande *et al.,* 2006). Recently, pollen bioassay as a simple and effective screening technique to differentiate resistant, late wilting and susceptible genotypes has been proposed (Ratna Babu and Ravikumar, 2010). Fusaric acid (FA), one of the toxins produced by the *Fusarium*, is used as the selective agent to screen chickpea genotypes.

Concentration of FA to inhibit 50% pollen tube growth differs for resistant, late wilting and susceptible types, which has also been validated by molecular markers.

In lentil, inoculum density of 10^6 conidia ml^{-1} is generally used to inoculate seedlings. Different inoculation methods such as seeding surface of disinfected lentil seeds in the soil infested with the pathogen grown on autoclaved millet or other grains (10% w/w) and inoculating by pouring spores grown on PDA near the roots of 15-day-old seedlings in pots are used (Riccioni *et al.,* 2003). Roots of 10-day-old seedlings grown on sterilized sand can be dipped in a spore suspension with concentration of 10^5 conidia ml^{-1}. The wilt reaction in terms of severity of incidence can be evaluated after 7-10 days of inoculation.

However, despite many limitations, field screening is still a widely used technique to discriminate between resistant and susceptible genotypes of chickpea, pigeonpea and lentil for FW owing to operational simplicity and economy of labour. Nonetheless, it should be used for preliminary screening only. Resistant genotypes must be confirmed for resistant reaction under controlled screening condition. Rapid discrimination between resistant and susceptible genotypes may be performed *in vitro* by using selective agents such as FA, which has already been used in tissue culture studies to select the wilt resistant variants in banana (Matsumoto *et al.,* 1995) and pigeonpea (Pandey *et al.*, 1995). However species/race specificity of FA produced by the pathogen needs to be investigated further. With the availability of diagnostic PCR based molecular markers, resistance to FW can be established without subjecting germplasm and segregating generations for phenotyping in wilt-sick plot. For this purpose, molecular markers that are closely linked with WR genes are required.

GENETICS OF WILT RESISTANCE

Development of wilt-resistant varieties is the major objective in the pulse breeding program to ensure stability in production and productivity (Choudhary, 2010). The accomplishment of the objective more often becomes difficult due to evolution of new races and co-existence of more than one pathotype at any one location. The transfer of FW resistant genes from the donors to an otherwise high-yielding genotype requires knowledge about the inheritance and genetics of wilt resistance. The inheritance studies for FW resistance in chickpea, pigeonpea and lentil are briefly reviewed below:

Inheritance of WR in chickpea

Genetics of resistance to FW in chickpea has been extensively studied, and contradicting results have been reported (Table 3). Resistance to race 1 was first reported as monogenic recessive (Sindhu *et al.,* 1983). Later it was shown that three independent genes (H1/h1, H2/h2, H3/h3) govern the resistance to race 1 (Upadhyaya *et al.,* 1983a; 1983b). However, complete resistance to race 1 is governed by the presence of recessive homozygous alleles at the first 2 loci and of dominant allele(s) at the third locus (Singh *et al.,* 1987a; 1987b). Moreover, there is difference in the time of wilting (Kumar and Haware, 1982) in different cultivars. Although the time of wilting was controlled by three independent genes (named as *h1*, *h2* and *H3*, each of which delayed onset of disease symptoms), WR appeared to be monogenic. Cultivars 'BG 212', 'CPS 1', 'JG 74' and 'WR 315' carrying recessive alleles at both loci are completely resistant to race 1, whereas cultivars 'C 104' and 'K 850', which are homozygous recessive at one of the two loci, show late-wilting. The cultivars 'JG 62', which does not carry any recessive alleles at the two loci, show early-wilting. Recombinant inbred lines (RIL) derived from 'JG 62' and 'WR 315' showed a range of variation for the wilting time, indicating influence of several modifiers or quantitative trait loci (QTLs) with minor effect on major WR genes (Brinda and Ravikumar, 2005).

Resistance to race 2 was earlier reported to be monogenic (Pathak et al., 1975), digenic (Gumber *et al.,* 1995) and trigenic (Kumar, 1998). Interestingly, genetic analyses involving progenies derived from the cross with the same parentage indicated three genes (Kumar, 1998) and single recessive gene (Sharma *et al.,* 2005a) for FW resistance. Sharma and Muehlbauer (2007), however, ascribed this difference to the

evaluation technique and questioned about the existence of late wilting. They called it "slow wilting" by clearly delineating the difference between these two phenomena. Further, they also reported involvement of minor genes which could influence the major genes governing WR to race 2.

Monogenic (Tekeoglu *et al.,* 2000) or digenic (Rubio *et al.,* 2003) mode of inheritance has been reported for resistance to race 0. According to Halila *et al.* (2008), resistance to race 0 is controlled by two different genes; one present in accession 'JG 62' (Foc 01/foc 01) and the second present in accession 'CA 2139' (Foc 02/foc 02). Each of them governs complete resistance to race 0 of the pathogen. They also indicated the presence of minor genes for slow wilting in 'WR 315' along with the major genes for vertical resistance. Similarly, resistance to race 4 has been shown to be either monogenic recessive (Tullu *et al.,* 1998; Sharma *et al.,* 2005a) or digenic recessive (Tullu *et al.,* 1999). Recently, Sharma *et al*. (2004) have reported from their study in 100 RILs (F_7) derived from the cross of 'WR 315' (resistant) × 'C 104' (susceptible) that the resistance to race 3 in chickpea germplasm accession 'WR 315' is inherited as a single gene (designated foc-3).The single gene control for resistance to races 3 and 5 has also been reported in some other similar studies (Tekeoglu *et al.,* 2000; Sharma *et al.,* 2005a; 2005b).

Inheritance of WR in pigeonpea

Pal (1934) was the first to report multiple genes for wilt resistance in pigeonpea. Shaw (1936) and Pathak (1970) documented two complementary genes imparting resistance to FW in pigeonpea. However, Jain and Reddy (1995) established that WR in 'ICP 8863' was a monogenic recessive trait, and designated the gene as *pwr1*. Odeny *et al.* (2009) studied the genetics of WR in the African (ICEAP 0040) and Indian (ICP 8863) genotypes. They found that the WR in 'ICEAP 0040' was controlled by a single recessive gene, while two pairs of recessive genes governed the resistance in 'ICP 8863'. Empirical observations for the wilt reaction in available germplasm resources also substantiate these findings. On the contrary, based on a cross between 'IT 6' × 'ICP 8863' and its F_2 and F_3 segregating generations, Pandey *et al.* (1996) inferred that 'ICP 8863' carried a dominant gene (*FuR1*) for resistance to an FU isolate '8801'. Wilt resistance in some other instances has also been documented as the monogenic dominant trait (Singh *et al.,* 1998; Dharwad *et al.,* 2012). It indicates that the transfer of resistance to the susceptible genotypes may be accomplished through simple backcross breeding. The dominant wilt reaction has also been reported by Okiror (2002). However, resistance was governed by two independent genes with inhibitory and complementary gene interactions for wilt resistance in 'NPP 725' and 'NPP 726', respectively. Saxena *et al.* (2012) have also reported dominant suppressive epistatic effect of a dominant gene over the recessive one for WR in crosses of a FW susceptible cytoplasmic male-sterile (CMS) line with four FW resistant fertility restorers. Such a situation is likely to accelerate hybrid breeding as the transfer of FW resistant gene in the CMS line will allow the utilization of both wilt resistant as well as susceptible restorers in generating wilt resistant F_1 hybrids.

Variant-specific genetic studies have been conducted at the Indian Institute of Pulses Research (IIPR), Kanpur. F_2 mapping populations (> 200 individuals/population) derived from the cross 'ICP 8863' (resistant) × 'Type 7' (susceptible) were screened against 4 FU variants (V1, V2, V3 and V4). Phenotypic data revealed that WR may be governed by 1 or 2 recessive genes. Further evaluation of F_2 mapping population against V2 and V4 variants confirmed that resistance to these variants is governed by 2 recessive genes with duplicate gene action (IIPR, 2012).

Inheritance of WR in lentil

Only limited inheritance studies have been carried out to know the genetics and inheritance pattern of WR in lentil (Table 3). Five independent genes have been reported to confer resistance to FW in lentil (Kamboj *et al.,* 1990). Based on allelism test, 2 duplicate genes and 2 complementary genes have been identified, imparting WR in the variety 'PL 234' and in 'JL

446' and 'PL 286', respectively. However, only a single dominant gene has been reported to control WR in the crosses made at ICARDA (Abbas, 1995). Eujayl *et al.* (1998) also recorded monogenic inheritance for WR in 'ILL 5588' and designated the gene as *Fw*.

TAGGING OF RESISTANCE GENE(S) THROUGH MOLECULAR MARKERS

Though simply inherited, the transfer of WR to locally adapted cultivars has been difficult due to linkage drag and difficulty in accurate phenotyping under field screening because of uneven concentration of inoculum and presence of different races/pathotypes of *Fusarium* spp. affecting these three pulse crops. Therefore, tagging of WR gene(s) through molecular markers is highly desirable. The progress made towards tagging of resistance gene(s) thus far in these three crops are reviewed below:

WR gene tagging in chickpea

The first WR gene tagged in chickpea was H_1 against race 1 (Mayer *et al.,* 1997). Two primers UBC-170_{550} and CS-27_{700} that amplified a DNA fragment linked to FW resistance and susceptibility, respectively, were identified. However, after converting these 2 markers into allele specific associated primers (ASAPs), only CS-27_{700} was found specific to the susceptible allele, and the other one (UBC-170_{550}) appeared to be locus specific. Later, the same RAPD markers were shown to be associated with gene controlling resistance to race 4 at a distance of 9 cM (Tullu *et al.,* 1998). Ratnaparkhe *et al.* (1998a, b) attempted to tag WR gene through ISSR markers in an inter-specific mapping population. They identified two ISSR markers UBC-855_{500} (Ratnaparkhe *et al.,* 1998a) and UBC-825_{1200} (Ratnaparkhe *et al.,* 1998b) linked to the resistance gene for race 4. The co-segregation of UBC-855_{500} with CS-27_{700} suggested that one of the resistance genes to race 4 and race 1 are closely linked. The genetic mapping of the *foc-3* resistance gene and linkage between *foc-1*, *foc-3*, and *foc-4* resistance genes using RAPD, STS, ISSR, and STMS markers were reported by Sharma *et al.* (2004). The STMS marker TA96 was found linked to *foc-3* gene at a distance of 0.6 cM, whereas markers TA27 (STMS) and CS27A (STS) co-segregated with TA96. They further established that *foc-3*, *foc-1* and *foc-4* were linked. *foc-3* appeared to be linked with *foc-1* and *foc-4* at a distance of 9.8 cM and 8.7 cM, respectively, whereas *foc-1* and *foc-4* were mapped closely at 1.1 cM.

The development of SSR or microsatellite markers has not only expedited the process of tagging of WR genes in chickpea, but also located all the resistance loci in narrow genomic window of < 6 cM (Table 4). Markers tagged closely to *foc-1, foc-2, foc-3* (Gowda *et al.,* 2009), *foc-4* (Benko Iseppon *et al.,* 2003) and *foc-5* (Winter *et al.,* 2000) were identified, and all were mapped on LG 2 of integrated chickpea linkage map (Winter *et al.,* 2000). The two independent genes conferring resistance to race 0 have also been identified (Rubio *et al.,* 2003) and tagged. The first resistance gene ($Foc0_1/foc0_1$) flanked by two markers (OPJ20_{600} and TR59) (Cobos *et al.,* 2005) has been mapped on Linkage Group 3 (LG 3), corresponding to LG 2 of integrated map by Winter *et al.* (2000). Recently, Sabbavarapu *et al.* (2013) mapped two new QTLs (FW-Q-APR-6-1 and FW-Q-APR-6-2) for race 1A in $F_{2:3}$ mapping population of 'C 214' x 'WR 315'. The second gene ($Foc0_2/foc0_2$) is mapped on LG 2 closely flanked by STMS markers, TS 47 and TA 59. Except $foc\text{-}0_1$ and the two QTLs (for race 1A), all other resistance genes to wilt pathogen are sited on linkage group 2 (Sharma and Muehlbuer, 2007). LG 2 corresponding to chromosome F is a hot spot for resistance to wilt pathogens. Reference map of LG 2 needs to be developed using large population size and highly reliable phenotyping method for all the races (0, 1, 2, 3, 4 and 5) to enhance the prospects of marker-assisted selection (MAS) and gene pyramiding for WR in chickpea. Expression of four genes (*GroES*2, 60srp, *CHS* and *IFR*) was essential for resistance to *Foc* race 1, whereas differential upregulation of some of these genes was sufficient for imparting complete resistance to the cultivar 'Digvijay' against FOC races 2 and 4 (Gurjar *et al.,* 2012).

Table 3. Inheritance of FW resistance in three SAT pulses.

Fusarium race/variant	Number and nature of WR gene	Gene symbol	Remarks	Reference
Chickpea				
Race 0	Monogenic or digenic	*foc-0$_1$*/ *Foc-0$_1$*	Complete resistance	Tekeoglu *et al*. (2000)
	Monogenic or digenic	*foc-0$_2$*/ *Foc-0$_2$*	-	Rubio *et al*. (2003)
	Polygenic	-	Act along with vertical resistance genes	Halila *et al*. (2008)
Race 1	Trigenic	*H1/h1, H2/h2, H3/h3*	3 independent genes	Smithson *et al*. (1983)
	QTL	FW-Q-APR-6-1, FW-Q-APR-6-2	-	Sabbavarapu *et al*. (2013)
Race 1A	Trigenic	*h1* (syn *foc-1*), *h2 H3*	Recessive at first two loci and dominant at the third locus	Singh *et al*. (1987a; b)
Race 2	Monogenic	*foc-2*	Complete resistance	Pathak *et al*. (1975); Sharma *et al*. (2005)
	Digenic	-	-	Gumber *et al*. (1995)
	Trigenic	*a*, *b, C*	Individually confers late wilting, first two genes impart complete resistance	Kumar (1998)
	Polygenic	-	Influencing the major WR genes	Sharma and Muehlbauer (2007)
Races 3	Monogenic	*foc-3*/ *Foc-3*	Complete resistance	Tekeoglu *et al*. (2000); Sharma et al. (2005, 2005b)
Race 4	Monogenic recessive	*foc-4*	Complete resistance	Tullu *et al*. (1998); Sharma et al. (2005)
	Digenic recessive	-	Complete resistance	Tullu *et al*. (1999)
Race 5	Monogenic	*foc-5*/ *Foc-5*	Complete resistance	Tekeoglu *et al*. (2000); Sharma *et al*. (2005, 2005b)
Pigeonpea				
Variant not specified	Multiple genes	-	-	Pal (1934)
Variant not specified	Digenic interaction	-	Complementary genes	Shaw (1936); Pathak (1970)
Variant not specified	Monogenic recessive	*pwr1*	WR in ICP 8863	Jain and Reddy (1995)
Variant not specified	Monogenic recessive	-	WR in ICEAP 0040	Odeny *et al*. (2009)
Variant not specified	Digenic recessive genes	-	WR in ICP 8863	Odeny *et al*. (2009)
Isolate 8801	Monogenic dominant	*FuR1*	Dominant gene	Pandey *et al*. (1996)
Variant not specified	Monogenic dominant	-	Dominant gene	Singh *et al*. (1998); Okiror (2002); Dharwad *et al*. (2012)
Variant not specified	Digenic interaction	-	Resistance is inhibitory in NPP 725, and is complementary in NPP	Saxena *et al*. (2012)

			726	
Variants (V1, V2, V3, V4)	Double recessive	-	duplicate gene action	(IIPR, 2012)
Lentil				
Strain not specified	Five genes	-	Independent genes	Kamboj *et al.* (1990)
Strain not specified	duplicate genes	-	Resistance in PL 234	Kamboj *et al.* (1990)
Strain not specified	Two complementary genes	-	Resistance in JL 446 and PL 286	Kamboj *et al.* (1990)
Strain not specified	Monogenic dominant gene	-	ICARDA experiments	Abbas (1995)
Strain not specified	Monogenic dominant gene	*Fw*	Resistance in ILL 5588	Eujayl *et al.* (1998)

Guteirrez *et al.* (2012) demonstrated that resistance gene analogs (RGA05 and RGA07) were upregulated in resistant genotype 'WR 315' within 2 days after infection with FOC 5. These two studies are the foundation for further understanding of host (chickpea)-pathogen (FOC) interaction. However, detailed studies are still required to assign their specific role in defense mechanism and their interaction with other defense related proteins.

WR gene tagging in pigeonpea

Kotresh *et al.* (2006) attempted to identify RAPD markers associated with wilt phenotype by using two F_2 populations derived from two crosses 'GS1' (susceptible) × 'ICPL 87119' (resistant) and 'GS1' × 'ICP 8863' (resistant). PCR testing of bulked DNA from subsets of resistant and susceptible plants revealed the presence of two amplicons of 704 bp and 500 bp (OPM03704 and OPAC11500) with susceptible wilt reaction. Analysis of individual F_2 plants showed a segregation ratio of 3: 1 for the presence: absence of the amplicons in both crosses. Considering the wilt reaction and susceptibility-linked RAPD marker, it was possible to deduce genotype of every F_2 plant and the expected 1RR: 2Rr: 1rr genotypic ratio for wilt reaction.

Screening of 88 pigeonpea lines including 2 resistant checks (ICPL 87119 and ICPL 8863) for WR under field condition was performed by Prasanthi *et al.* (2009). PCR reactions using different primers with genomic DNA of these lines resulted in the identification of 6 resistant sources with specific amplification for WR at 920 bp with OPGO8 primer (Table 4). With the wilt reaction and resistance linked to RAPD marker, it was possible to identify the new resistance sources in a short time which could be released directly or utilized further in the breeding program for FW resistance.

For the first time, parental polymorphism was also studied using sequence related amplification polymorphism (SRAP) and SRAP-RGA techniques in pigeonpea at IIPR, Kanpur (IIPR, 2012). Total 132 (out of 162) markers *viz.,* 68 AFLP, 30 SRAP, 14 SRAP-RGA and 20 AFLP-RGA showed parental polymorphism between 'ICP 8863' (FW resistant variety) and 'Type 7' (FW susceptible variety). Out of these, 125 markers were dominant (76 and 49 were specific to ICP 8863 and Type 7, respectively) and 7 were found co-dominant. Homogeneity of parents involved in 5 crosses was also tested using 3 polymorphic SSR primers (PP-10, PPMC-1 and PPMC-3) and found that parents of only 2 crosses were true. Hybridity of 29 putative F_1 plants derived from 1 of the true crosses was tested using above three SSR markers and 1 RAPD marker (OPP 17).

Table 4. WR genes tagged in three SAT pulses.

Chickpea					
Fusarium race	Gene tagged	Marker identified	Distance (cM)	Linkage group	Reference
Race 0	*Foc0$_1$/foc0$_1$*	TR59 OPJ20$_{600}$	2.0 3.0	LG3	Rubio et al. (2003), Cobos *et al*. (2005)
	(*Foc0$_2$/foc0$_2$*)	TS 47 *Foc-0$_2$* TA 59	<5.0	LG2	Halila *et al*. (2010)
Race 1	*H$_1$*	UBC-170$_{550}$ CS-27$_{700}$ CS27A	7.0 7.2 4.9	-	Mayer *et al*. (1997)
	foc-1	TA59 TA96 TA27	4.4 4.9 4.9	LG2	Iruela *et al*. (2006), Sharma and Muehlbauer (2007)
		TA 110 *foc-1* H3A12	6.0	LG2	Gowda *et al*. (2009)
Race 1A	FW-Q-APR-6-1, FW-Q-APR-6-2	CaM1402–CaM1101 (Flanking) CaM1125–TA22	36.3 41.4	LG6	Sabbavarapu *et al*. (2013)
Race 2	*foc-2*	TA96 TA27 TR19 CS27A	1.5 1.5 4.9 1.5	LG2	Sharma and Muehlbauer (2007), Tekegulu *et al*. (2002)
		H3A12	2.9	LG2	Gowda *et al*. (2009)
Race 3	*Foc-3/foc-3*	TA96 TA27 TR59 CS27A	0.5 0.5 0.5 0.5	LG2	Sharma and Muehlbauer (2007)
		H$_1$B06y *foc-3* TA 194	0.9	LG2	Gowda *et al*. (2009)
Race 4	*foc-4*	TA59 TA96 TA27 TR19 CS27A	3.8 3.3 3.3 3.1 3.3	LG2	Sharma and Muehlbauer (2007), Tekegulu *et al.* (2002)
		CS-27$_{700}$	-	-	Tullu *et al*. (1998)
		UBC-170$_{550}$ UBC-825$_{1200}$ OPU17-1 EAAMCTA12	9.0 5.0 4.1 5.9	-	Ratnaparkhe *et al*. (1998a, 1998b)
		R-2609-1	2.0	LG2	Benko Iseppon *et al.* (2003)
Race 4 and race 1		Co-segregation of UBC-855$_{500}$ with CS-27$_{700}$	--	--	(Ratnaparkhe *et al*. (1998b)

Race 5	*Foc-5/foc-5*	TA27 TA59 TA96 CS27700 UBC-170550	2.9 2.4 2.9 9.2 2.5	LG2	Winter *et al*. (2000), Tekegulu *et al*. (2002), Iruela *et al*. (2006), Sharma and Muehlbauer (2007)
		ECAMCTA07 OP-M20-21045 OP-M20-31103	6.4 12.0 12.0	LG2	Winter *et al*. (2000)
		TA110 TA59	6.5 8.9	LG2	Iruela *et al*. (2007)
Pigeonpea					
Variant not specified	WR gene	OPM03704 and OPAC11500	--	--	Kotresh *et al*. (2006)
Variant not specified	WR gene	OPGO8	--	--	Prasanthi *et al*. (2009)
Lentil					
Strain not specified	*Fw*	RAPD marker OPK-15_{900} OP-BH_{800} and OP-$D15_{500}$	10.8	Coupling phase	Eujayl *et al*. (1998)
		OP-C0465o	-	Repulsion phase	
Strain not specified	WR gene	SSR59-2B	8.0		Hamwieh *et al*. (2005)
Strain not specified	WR gene	AFLP p17m30710	3.5		Hamwieh *et al*. (2005)

Table 5. Important varieties/donors of SAT pulses having FW resistance.

Crop	Resistant variety/donor	Country	Reference
Chickpea	Surutato 77' and 'Sonora 80' (Kabuli)	Mexico	Sharma *et al.* (2005)
	ICCV 2, ICCV 3, ICCV 4 and ICCV 5	India	Kumar *et al.* (1985)
	GL 87078 and GL 87079	India	Singh *et al.* (1994), Gaur *et al.* (2012)
	WR 315, CPS 1, JG 74, Annigeri, Avrodhi, Phule G 5, DCP 92-3, JG 315	India	Ali *et al.* (2001)
	KWR 108, JG 11, JG 315, JG 16, JG 74, Pusa 372	India	Hari Chand and Khirbat (2009)
	ICCV 05527, ICCV 05528, ICCV 96818	India	Sharma *et al.* (2012)
	Sanford, White Spanish, Dwelley, UC 15, UC 27	USA	Sharma *et al.* (2005)
	ICC 7537, DZ 10-4	Ethiopia	Sharma *et al.* (2005), Daba *et al.* (2005)
	CA 2954	Spain	Rubio *et al.* (2004)
	Kabuli CV ICCV2 and UC 15	Sudan	Ali *et al.* (2002)
	Chaffa, Kimiya, ICC 7520	Iran	Sharma *et al.* (2005)
	K 850, Punjab 2000	Pakistan	Ali *et al.* (2004)
	Amdoun 1	Tunisia	Sharma *et al.* (2005)
Pigeonpea	ICP 8863	India	Nene and Kannaiyan (1982)
	BDN 1, BDN 2, C 11, ICPL 87119, BSMR 736, TS 3, WRP 1, DA 11	India	Choudhary and Nadarajan (2011)
	IPA 204	India	Choudhary (2010)
	IPA 16 F, IPA 8 F, IPA 9 F, IPA 12 F	India	Singh *et al.* (2011)
	TK 21/1	Kenya	Kimani *et al.* (1994)
	ICEAP 00040	Kenya, Malawi	Gwata *et al.* (2006)

		and Tanzania	
	ICP 9145	Africa	Karimi *et al.* (2012)
Lentil	Pant L 406	India	Pandya *et al.* (1980)
	Pant L 4	India	Singh *et al.* (1994)
	Pant L 639, Priya, Seri, JL 3, Noori, VL 507 L 4147	India	Rahman *et al.* (2009)
	IPL 306	India	IIPR (2012)
	IPA 98	Iraq	Rahman *et al.* (2009)
	Adaa, Alemaya	Ethiopia	Sarker and Erskine (2002)
	Firat 87, Syran 96	Turkey	Rahman *et al.* (2009)
	Talya 2, Rachayya, Hala	Lebanon	Rahman *et al.* (2009)
	ILL 5883, ILL 5588,ILL 4400, ILL 590	Syria	Erskine *et al.* (1994)
	Idleb 2, Idleb 3, Idleb 4, Ebla 1	Syria	El-Ashkar *et al.* . (2003; 2004a; 2004b)
	ILL 6256	Nepal	Joshi and Maharjan (2003)

WR gene tagging in lentil

Eujayl *et al.* (1998) identified RAPD marker OPK-15_{900} linked with *Fw* gene at a distance of 10.8 cM and established its linkage with the RAPD markers OP-$B17_{800}$ and OP-$D15_{500}$ in coupling and OP-$C04_{650}$ in repulsion phase. These arbitrary markers can be made more useful by converting them into locus-specific sequence characterized amplified region (SCAR) markers for marker-assisted screening and selection. Subsequent study identified one SSR and AFLP markers that were linked with *Fw* gene at 8.0 and 3.5 cM, respectively (Hamwieh *et al.,* 2005). However, WR genes present in the Indian germplasm are yet to be mapped. Efforts are underway to develop mapping populations involving 'Precoz' and 'Sehore 74-3' as the susceptible and 'PL2' and 'IPL406' as the resistant parents. For developing mapping populations without any segregation distortion, molecular markers have been very useful in establishing hybridity of F_1 plants (Solanki *et al.,* 2010). New RILs have been developed at ICARDA involving parents from different geographical regions for mapping race-specific resistance genes.

CONVENTIONAL AND MOLECULAR BREEDING FOR WILT RESISTANCE

Both chickpea and lentil are highly self-pollinated crops. As the trait of interest i.e., FW resistance appears to be simply inherited, conventional breeding methods used in autogamous crops such as backcross and recombination breeding should be equally effective for breeding wilt resistant varieties. This holds true for pigeonpea too, which is primarily a self-pollinated crop. However, crossing and handling of segregating generations in pigeonpea need to be carried out in a protected net house to prevent outcrossing by honeybees. Simple field screening in WSPs and selection has resulted in the identification and release of a number of FW resistant donors and varieties, respectively in these 3 pulse crops (Table 5). Recombination breeding, a selection-crossing-selection cycle which consists of controlled crossing between agronomically superior genotype(s) and wilt resistant donor(s) followed by pedigree selection or its various modifications in the segregating generations has been the most utilized breeding approach for incorporating WR in these 3 pulse crops.

The bulk pedigree method has been the preferred method at ICARDA in which targeted crosses are advanced under disease-free conditions as bulks up to F_4 generation, and the selected single plant progenies (F_5) are grown in the wilt-sick plot. Plant progenies with resistant reaction are further evaluated in WSP and in normal field as preliminary screening nursery

(F_6), preliminary yield trial (F_7), and advanced yield trial (F_8). Finally, the elite lines with WR, high yield and other desirable traits in different genetic backgrounds are included in Lentil International Fusarium Wilt Nursery (LIFWN) and other yield nurseries for multi-location testing in the targeted countries. In addition to genetically fixed elite lines and germplasm, segregating populations are also made available to the national programs for selection in the local wilt-sick plot and agro-climatic conditions. Systematic utilization of resistant sources such as 'ILL 5883', 'ILL 5588', 'ILL 4400' and 'ILL 590' at ICARDA has resulted in the development of a wide spectrum of FW resistant varieties for cultivation in different countries. Some of the prominent wilt resistant varieties are 'Idleb 2', 'Idleb 3', 'Idleb 4' and 'Ebla 1' in Syria; 'Talya 2', 'Rachayya' and 'Hala' in Lebanon; 'Firat 87' and 'Syran 96' in Turkey; 'Ada', 'Alemaya', 'Assano', 'Alemtina' and 'Teshale' in Ethiopia; 'Kimiya' in Iran and 'IPA 98' in Iraq. In India, national program has released several wilt resistant varieties, and prominent among them are 'L 4147', 'Pant L 406', 'Pant L 4', 'Pant L 639', 'Priya', 'Seri', 'JL 3', 'Noori', and 'VL 507' (Pandya *et al.,* 1980; Singh *et al.,* 1994; Rahman *et al.,* 2009).

In chickpea, although the source of resistance is mainly present in accessions from Indian continent especially in *Desi* types, wilt resistant varieties such as 'Surutato 77' and 'Sonora 80' were first developed for *Kabuli* types in Mexico. It was followed by the release of FW resistant chickpea varieties in Tunisia (Amdoun 1) and USA (UC 15 and UC 27). In India, two cultivars 'GL 87078' and 'GL 87079' were developed with resistance to all FOC races. Four short duration *Kabuli* varieties (ICCV 2, ICCV 3, ICCV 4 and ICCV 5) with resistance to FOC race 1 were also developed at ICRISAT (Kumar *et al.,* 1985). Sharma *et al.* (2012) reported that genotype and genotype × environment (GGE) biplot analyses allowed the selection of three breeding lines (ICCV 05527, ICCV 05528 and ICCV 96818) with moderate level of disease resistance and stable performance across the environments. In pigeonpea, national and international efforts have led to the release of a number of FW resistant varieties such as 'BDN 1', 'BDN 2', 'C 11', 'ICP 8863', 'ICPL 87119', 'BSMR 736', 'TS 3', 'WRP 1', 'DA 11', 'IPA 203', and the like for cultivation in India (Choudhary and Nadarajan, 2011). 'ICP 8863' appears to be resistant to all FU variants (except variant III) (Tiwari and Dhar, 2011).

With increasing information on host-pathogen interaction, genetic variation in the pathogen and temporal variation in pathogenicity, more efficient screening and breeding methods would be required for improving WR in chickpea, pigeonpea and lentil (Figure 3). For example, early and late wilt reactions are noticeable in genotypes of these 3 food legumes. These host reactions may be under the control of different genetic systems as has been the case with chickpea (Kumar, 1998). Combining them together through marker assisted selection (MAS) may be essential for stable resistance. The near-isogenic lines (NILs) of chickpea with FOC resistance/susceptibility have also been developed based on closely linked markers in RIL population (Castro *et al.,* 2010). Similarly, resistant sources identified in wild species require allelism test to establish their genetic relationship with resistance gene already identified in the cultivated germplasm and, if found alien, these should be introgressed in the cultivated germplasm for durable resistance. However, transfer of desirable alleles is not so simple because of difficulty in efficient tracking for desired and non-desired alleles in breeding lines. This problem can be overcome by advanced-backcross QTL based breeding (AB-breeding) as it is the most suitable for introducing novel alleles from wild relatives to the cultivated species cultivars or varieties in a controlled manner (Tanksley and Nelson, 1996). Furthermore, establishment of pathogenic races in FU and FOL will require search for race-specific resistance genes and their pyramiding in superior genotypes. However, it is difficult through recombination breeding approach by selecting desirable plants on the basis of phenotype. Marker-assisted gene pyramiding can be used to combine in a single genotype the desirable WR genes as well-established tight association between markers and target traits has already been reported in chickpea and lentil (Kumar *et al.,* 2011). Recently, gametophytic selection for WR has been reported to be

effective in chickpea for developing wilt resistant genotypes in a short period (Ravikumar *et al.,* 2013). They have demonstrated the effectiveness of gametophytic selection in two populations segregating for wilt resistance using molecular markers linked to H1 and H2 locus for WR in chickpea. The same may also be tried for pigeonpea and lentil.

Therefore, identification and incorporation of new WR genes in breeding programs, and development of genotypes with multiple combinations of WR genes will remain a continuous activity for sustainable production of chickpea, lentil and pigeonpea. Molecular markers offer a viable option to accelerate breeding progress through indirect selection for WR in segregating generations without actual phenotyping in the wilt-sick plot. Marker-assisted introgression of WR gene(s) is possible only when locus specific co-dominant markers tightly linked with the WR gene(s) are identified. In pigeonpea, such markers are yet to be identified and the information available is very scanty. However, some tightly linked SSR markers for *foc-1* (6 cM), *foc-2* (2.9 cM) and *foc-3* (0.9 cM) have been reported in chickpea (Gowda *et al.,* 2009). Presently, the most tightly linked marker with WR gene '*Fw'* in lentil is AFLP marker p17m30710 (3.5 cM) followed by SSR marker SSR59-2M (8 cM) and RAPD marker OPK-15_{900} (10.8 cM). However, their distance from the gene of interest '*Fw*' does not provide confidence for use in marker-assisted screening and selection. Therefore, there is a need to develop more locus-specific co-dominant markers such as SSR, ESTs, CAPS, and SNPs in the map of these three legumes at the closer proximity (< 1 cM) with WR gene(s). It will make MAS an essential component in resistance breeding to develop FW resistant varieties in chickpea, pigeonpea and lentil.

SUMMARY AND OUTLOOK

Considerable progress has been made during the last three decades in characterizing pathogenic variability of FOC, FU and FOL, identifying resistant sources for FW, establishing genetics of wilt resistance and incorporating WR gene(s) into the improved cultivars of chickpea, pigeonpea and lentil. The rapid adoption of these resistant varieties may prevent yield losses due to FW, reduce the gaps between potential and realized yield and bring about stability in production. However, many milestones have still to be achieved. For example, even in chickpea in which race specificity has been well-established, there are many discrepancies regarding set of differentials used to classify the races. The number of differentials used in different studies ranged from as few as 8 to as many as 22 (Sharma *et al.,* 2005a). Since WR in chickpea is race-specific and governed by major resistance genes, there is a need to develop an improved differential set for chickpea wilt to reduce ambiguity in race determination. The major factor that has led to discrepancy in molecular marker studies relates to the application of different phenotyping methods and disease scoring scales (Tullu, 1996). Therefore, it seems appropriate to standardize uniformly applicable phenotyping method and disease scoring scale along with permanent mapping. Similarly it seems imperative to combine in a single genotype multiple WR genes through MAS to make it resistant to multiple FOC races. The use of FA as a selective agent for scoring wilt reaction needs further investigation as it (fusaric acid) may not be the sole factor resulting in the development of wilt disease in these 3 pulse crops.

In both pigeonpea and lentil, lack of precise knowledge on the existence of pathogen race is the major hindrance to develop durable resistant cultivars for different regions. In spite of good progress in breeding wilt resistant varieties, its impact could not be demonstrated in farmers' fields due to their susceptibility to other soil borne pathogens causing root rot diseases (e.g. collar rot and dry and wet root rots in lentil). Due to lack of efficient screening techniques, stable resistance for these related pathogens could not be identified, and thus remain the major breeding goals in Asia and Africa. Therefore, there is an urgent need to identify resistance genes for these soil-borne pathogens and incorporate them in wilt susceptible cultivars for visible impact in farmers' fields.

Studies are also needed to ascertain that incorporation of WR should not accompany

susceptible reaction for other diseases. For example, 'ICP 8863', which is a highly wilt resistant pigeonpea variety, is used as a susceptible check for SM disease. On the contrary, 'Bahar' that is susceptible to all FU variants is resistant to all the strains of SM in pigeonpea. Such mystery needs to be unravelled. Besides, further studies are required especially in lentil and pigeonpea to establish pathogenic races using an international differential set which is not yet available. Efforts are underway in lentil to develop a common differential set for pathogenicity test which can distinguish different FOL isolates into pathogenic races. This is the pre-requisite for generating information on geographical distribution of races and efficient deployment of race specific resistance genes in lentil cultivars for durable resistance.

However, many gaps still exist in our knowledge on the influence of environmental parameters on disease progression which is very crucial for controlling the disease by cultural practices. Preliminary studies indicate that morphological and anatomical characters as well as biochemical constituents of roots of these pulses do play an important role in disease reactions, and thus influencing the wilt incidence.

However, there is no information on the underlying mechanism of wilt resistance. Marker-assisted breeding for FW resistance in these food legumes is very limited, partly because WR can be easily identified in field and laboratory. However, these markers can be strategically used to avoid combined effect of other soil-borne pathogens and genotype x environment interactions, and in identification of race-specific resistance genes and their pyramiding.

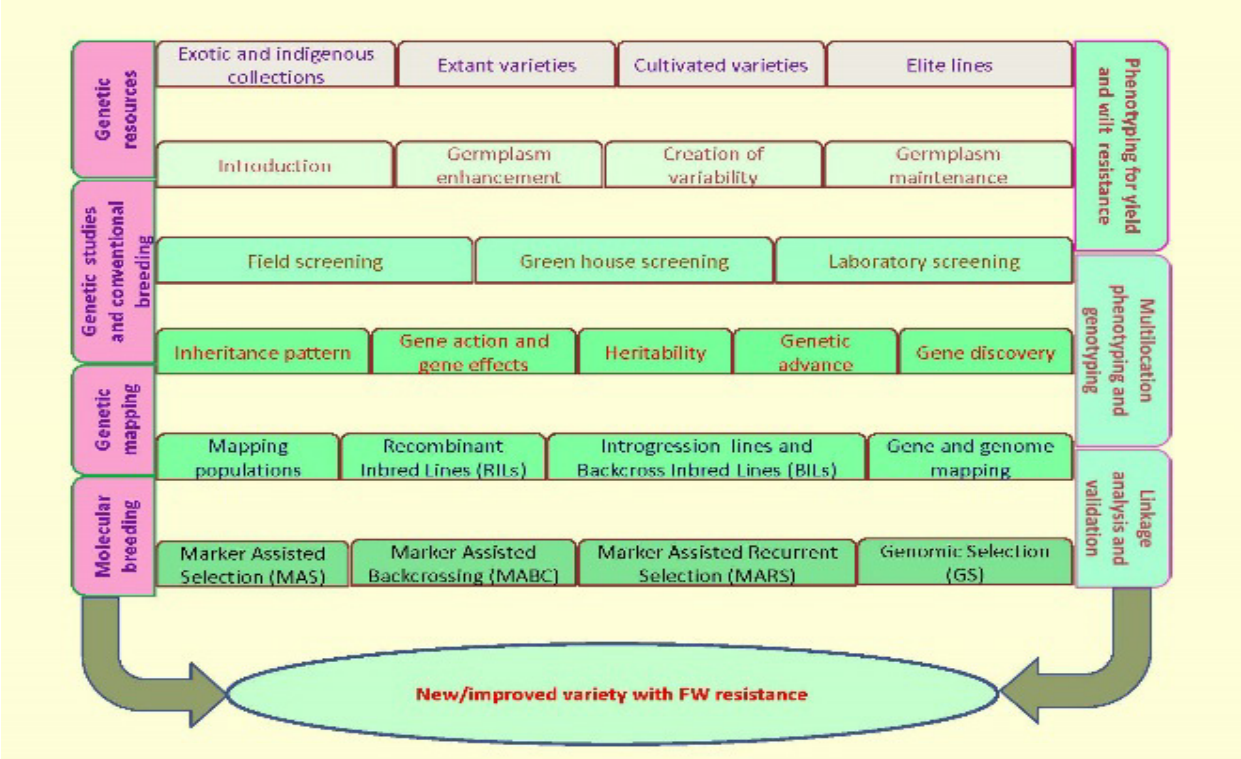

Figure 3. Integration of conventional and molecular breeding for wilt resistance in three SAT pulses.

REFERENCES

Abbas A (1995). Variation in some cultural and physiological characters and host/pathogen interaction of *Fusarium oxysporum* f. sp. *lentis* and inheritance of resistance to lentil wilt in Syria. PhD Thesis, Faculty of Agriculture, University of Aleppo, Aleppo, Syria.

Abu-Salem FM, Abou EA (2011). Physico-chemical properties of tempeh produced from chickpea seeds. *J. American Sci.* 7: 107–118.

Agrawal SC, Singh K, Lal SS (1993). Plant protection of lentil in India. In: W. Erskine, MC. Saxena, eds., Lentil in South Asia, ICARDA, Aleppo, Syria, pp. 147-165.

Alessandro I, Mohamed K, Luca R, Clarice JC, Kevin EM, Niklaus JG (2006). Screening techniques and sources of resistance to root diseases in cool season food legumes. *Euphytica* 147: 201–221.

Ali A, Ali M, Afzal M (2004). Punjab 2000: A new large-seeded desi chickpea variety for Punjab province of Pakistan. *Intl. Chickpea and Pigeonpea Newslett.* 11: 20-22.

Ali M, Kumar S, Dua RP (2001). Chickpea. In: VL. Chopra, ed., Breeding Field Crops, Oxford & IBH Publishing Co. Pvt. Ltd., New Delhi, pp. 305-331.

Ali MEK, Inanaga S, Sugimoto Y (2002). Sources of resistance to Fusarium wilt of chickpea in Sudan. *Phytopathol. Mediterr.* 41: 163–169.

Ali Y, Alam SS, Haq MA (1994). Evaluation of chickpea lines for wilt resistance. *Pak. J. Phytopathol.* 6: 110-114.

Arumuganathan K, Earle ED (1991). Nuclear DNA content of some important plant species. *Plant Mol. Biol. Rep.* 9: 208-218.

Arvayo-Ortiz RM, Esqueda M, Acedo-Felix E, Sanchez A, Gutierrez A (2011). Morphological variability and races of *Fusarium oxysporum* f.sp. *ciceris* associated with Chickpea (*Cicer arietinum*) Crops. *American J. Agric. Biol. Sci.* 6: 114-121.

Asha SM, Ramappa HK, Gowda MB (2012). Screening of Pigeonpea genotypes for resistance to Fusarium wilt. *Mysore J. Agric. Sci.* 46: 563-566.

Bahl PN, Lal S, Sharma BM (1993). An overview of the production and problems of lentil in South Asia. In: W. Erskine, MC. Saxena, eds., Lentil in South Asia, ICARDA, Aleppo, Syria, pp. 1-10.

Bayaa B, Erskine W, Khoury L (1986). Survey of wilt damage on lentil in Northern Syria. *Arab J. Plant Prot.* 4: 118–119.

Bayraktar H, Dolar FS (2012). Pathogenic variability of *Fusarium oxysporum* f. sp. *ciceris* isolates from chickpea in Turkey. *Pak. J. Bot.* 44: 821-823.

Bayya B, Kumari SG, Akkaya A, Erskine W, Makkouk KM, Turk Z, Ozberk I (1998). Survey of major biotic stresses of lentil in Southeast Anatolia, Turkey. *Phytopathol. Medit.* 37: 88-95.

Belabid L, Baum M, Fortas Z, Bouznad Z, Eujayl I (2004). Pathogenic and genetic characterization of Algerian isolates of *Fusarium oxysporum* f. sp. *lentis* by RAPD and AFLP analysis. *Africa. J. Biotech.* 3: 25-31.

Benko-Iseppon AM, Winter P, Huettel B, Staginnus C, Muehlbauer FJ, Kahl G (2003). Molecular markers closely linked to Fusarium resistance genes in chickpea show significant alignments to pathogenesis-related genes located on Arabidopsis chromosomes 1 and 5. *Theor. Appl. Genet.* 107: 379–386.

Bouslama M (1980). Chickpea improvement in Tunisia. In: Proceedings of International Workshop on Chickpea Improvement. ICRISAT, Patancheru, India, pp. 277-280.

Bretag T (1982). Fungi isolated from chickpea grown in experimental plots in North-West Vectoria. *Intl. Chickpea Newslett.* 7: 12.

Brinda S, Ravikumar RL (2005). Inheritance of wilt resistance in chickpea - a molecular marker analysis. *Curr. Sci.* 88: 701-702.

Cabera de la CJ, Trapero-casas A, Jimenez-Diaz RM (1985). Races of *Fusarium oxysporium* f. Sp. *ciceri.* in Andalucia Southern Spain. *Intl. Chickpea Newslett*. 13: 24-26.

Carranza JM (1965). Wilt of chickpea (*C. arietinum L.*) caused by *B. cinerea* (in Spanish). *Revta. Fac. Agron. Univ. Nae. La Plata* 41: 135-138.

Castro P, Piston P, Madris E, Millan T, Gil J, Rubio J (2010). Development of chickpea near isogenic lines for Fusarium wilt. *Theor. Appl. Genet.* 121: 1519-1526.

Chaudhary RG (2008). Pathogenic variability in *Fusarium oxysporum* f. sp. *lentis. Pulses Newslett.* 19: 3.

Chaudhary RG, Dhar V, Singh RK (2009). Association of fungi with wilt complex of lentil at different crop growth stages and moisture regimes. *Arch. Phytopathol. Plant Prot.* 42: 340-343.

Chaudhary RG, Saxena DR, Dhar V, Singh RK, Namdev JK (2010). Prevalence of wilt root rot and their associated pathogens at reproductive phase in lentil. *Arch. Phytopathol. Plant Prot.*, 43: 996-1000.

Choudhary AK (2010). A wilt resistant line 'IPA 204' of long-duration pigeonpea (*Cajanus cajan*). *Indian J. Agric. Sci.* 80: 907-909.

Choudhary AK, Iquebal MA, Nadarajan N (2012). Protogyny is an attractive option over emasculation for hybridization in pigeonpea. *SABRAO J. Breed. Genet.* 44: 138-148.

Choudhary AK, Nadarajan N (2011). Breeding improved cultivars of pigeonpea in India. IIPR, Kanpur.

Choudhary AK, Raje RS, Datta S, Sultana R, Ontagodi T (2013). Conventional and molecular approaches towards genetic improvement of pigeonpea for insect resistance. *American J. Plant Sci.* 4: 372-385.

Choudhary AK, Sultana R, Pratap A, Nadarajan N, Jha U (2011). Breeding for abiotic stresses in pigeonpea. *J. Food Leg.* 24: 165-174.

Choudhary AK, Vijayakumar AG (2012). Glossary of Plant Breeding. Lap Lambert Academic Publishing, Germany.

Claudius GR, Mehrotra RS (1973). Wilt and root rot disease at lentil (*Lens esculenta*) at Sagar. *Indian Phytopathol.* 26: 268-273.

Cobos M, Fernandez M, Rubio J, Kharrat M, Moreno M, Gill J, Millan T (2005). A linkage map of chickpea based on populations from Kabuli x Desi crosses: location of genes for resistance to Fusarium wilt race 0. *Theor. Appl. Genet.* 110: 1347-1353.

Daba K, Bejiga G, Anbessa Y, Gaur PM, Kumar J, Rao BV (2005). Chefe (ICCV 92318) - a new kabuli chickpea variety for Ethiopia. *Intl. Chickpea and Pigeonpea Newslett.* 12: 15-16.

Datta S, Chaudhary RG, Rai R, Singh RK, Dhar V (2009). Molecular diversity and pathotype analysis of *Fusarium oxysporum* f. sp. *lentis* population from Northeastern Gangetic Plain region of India. *SABRAO J. Breed. Genet.* 41: 1-11.

Datta S, Choudhary RG, Shamim Md, Singh RK, Dhar V (2011). Molecular diversity in Indian isolates of *Fusarium oxysporum* f.sp. *lentis* inciting wilt disease in lentil (Lens culinaris Medik). *African J. Biotech.* 10: 7314-7323.

Dhar V, Reddy MV (1999). Disease management strategies for increasing pulses production. Paper presented in the Brain Storming Meeting on Pulses Production, 26–27 March, 1999 at NBPGR, New Delhi.

Dharwad N, Salimath PM, Mahalinga D (2012). Genetic dissection of Fusarium wilt resistance in pigeonpea. *Plant Arch.* 12: 27-29.

Dolar FS (1997). Determination of the races of *Fusarium oxysporum* f.sp. *ciceris* in Ankara province, Turkey. *J. Turkish Phytopathol.* 26: 11-15.

Dubey SC, Priyanka K, Singh V, Singh B (2012). Race profiling and molecular diversity analysis of *Fusarium oxysporum* f.sp. *ciceris* causing wilt in Chickpea. *J. Phytopathol.* 160: 576–587.

El-Ashkar F, Sarker A, Erskine W, Bayaa B, El-Hassan H, Kadah N, Karim BA (2004a). Registration of 'Idlib-3' Lentil. *Crop Sci.* 44: 2261.

El-Ashkar F, Sarker A, Erskine W, Bayaa B, El-Hassan H, Kadah N, Karim BA (2004b). Registration of 'Idlib-4' Lentil. *Crop Sci.* 44: 2261–2262.

El-Ashkar F, Sarker A, Haddad N, Bayaa B, El-Hassan H, Erskine W (2003). Registration of 'Idlib-2' Lentil. *Crop Sci.* 43: 728–729.

El-Morsy GA, Abou-Zeid NM, Hassanein AM (1997). Identification of Fusarium wilt caused by *Fusarium oxysporum* and pathogen variability in faba bean, lentil and chickpea crops in Egypt. *Egyptian J. Agric. Res.* 75: 551-564.

Erskine W, Bayaa B (1996). Yield loss, incidence and inoculum density associated with vascular wilt of lentil. *Phytopathol. Medit.* 35: 24-32.

Erskine W, Tufail M, Russell A, Tyagi MC, Rahman MM, Saxena MC (1994). Current and future strategies in breeding lentil for resistance to biotic and abiotic stresses. *Euphytica* 73: 127-135.

Eujayl I, Erskine W, Bayaa B, Baum M, Pehu E (1998). Fusarium vascular wilt in lentil: Inheritance and identification of DNA markers for resistance. *Plant Breed.* 117: 497–499.

FAO (2012). Food and Agricultural Organization of the United Nation, FAO Statistical Database, http://faostat.fao.org.

Fleischmann A (1937). Observations on lentil wilt. *Pfianzenbau* 14: 49-56.

Gaur PM, Jukanti AK, Varshney RK (2012). Impact of genomic technologies on chickpea breeding strategies. *Agron.* 2: 199-221.

Ghosh R, Sharma M, Telangre R, Pande S (2013). Occurrence and distribution of chickpea

diseases in central and southern India. *American J. Plant Sci.* 4: 940-944.

Gowda SJM, Radika P, Kadoo NY, Mhase LB, Gupta VS (2009). Molecular mapping of wilt resistance genes in Chickpea. *Mol. Breed.* 24: 177-183.

Gumber RK, Kumar J, Haware MP (1995). Inheritance of resistance to Fusarium wilt in chickpea. *Plant Breed.* 114: 277-279.

Gupta O, Kotasthane SR, Khare MN (1988). Strain variation in *F. udum* in Madhya Pradesh, India. *Intl. Pigeonpea Newslett.* 7: 22-25.

Gurjar G, Barve M, Giri A, Gupta V (2009). Identification of Indian pathogenic races of *Fusarium oxysporum* f. sp. *ciceris* with gene specific ITS and random markers. *Mycologia* 101: 484–495.

Gurjar GS, Giri AP, Gupta VS (2012). Gene expression profiling during wilting in chickpea caused by *Fusarium oxysporum* f. sp. *ciceri. American J. Plant Sci.* 3: 190-201.

Gutierrez N, Gime´nez MJ, Torres AM, Atienza SG, Avila CM, Palomino C (2012). Up-regulation of resistance gene analogs (RGA) in chickpea in the early response to Fusarium wilt. *Euphytica* 186: 793-804.

Gwata ET, Silim SN, Mgonja M (2006). Impact of a new source of resistance to Fusarium wilt in pigeonpea. *J. Phytopathol.* 154: 62-64.

Halila I, Caboo HJ, Rubi J, Millan T, Kharrat M, Gill J (2008). Tagging and mapping a second resistance gene for Fusarium wilt race 0 in chickpea. *European J. Plant Pathol.* 124: 87-92.

Halila I, Rubio J, Millán T, Gill J, Kharrat M, Marrakchi M (2010). Resistance in chickpea (*Cicer arietinum*) to Fusarium wilt race '0'. *Plant Breed.* 129: 563–566.

Halila MH, Strange RN (1996). Identification of the causal agent of wilt of chickpea in Tunisia as *Fusarium oxysporum* f. sp. *ciceri* race 0. *Phytopathol. Mediterr.* 35: 67–74.

Halila MH, Strange RN (1997). Screening of Kabuli chickpea germplasm for resistance to Fusarium wilt. *Euphytica* 96: 273–279.

Hamwieh A, Udapa SM, Choumane W, Sarker A, Dreyer F, Jung C, Baum M (2005). A genetic linkage map of lentil based on microsatellite and AFLP markers and localization of Fusarium vascular wilt resistance. *Theor. Appl. Genet.* 110: 669–677.

Hari Chand, Khirbat SK (2009). Chickpea wilt and its management - A review. *Agric. Rev.* 30: 1–12.

Haware MP (1998). Disease of chickpea. In: DI. Allen, JM. Lenne, eds., The Pathology of Food and Pasture Legumes. CAB International, Wallingford, New York, pp. 473-516.

Haware MP, Jimenez-Diaz RM, Amin KS, Phillips JC, Halila H (1990). Integrated management of wilt and root rots of chickpea, pp. 129-137. In: Chickpea in the Nineties: Proceedings of the second international work shop on chickpea improvement, Patancheru, India.

Haware MP, Nene YL (1980). Influence of wilt at different stages on the yield loss in chickpea. *Trop. Grain Leg. Bull.* 19: 8-40.

Haware MP, Nene YL (1982). Races of *Fusarium oxysporum* f. sp. *ciceri. Plant Dis.* 66: 809–810.

Haware MP, Nene YL (1994). A rapid method for pigeonpea wilt resistance screening. *Indian Phytopathol.* 47: 400-402.

Hegde VS (2011). Morphology and genetics of a new found determinate genotype in chickpea. *Euphytica* 182: 35-42.

Hillocks RJ, Khonga EB (1996). Soil borne diseases in maize-based cropping systems in southern Malawi: incidence and farmer's perception. *African Plant Prot.* 2: 131-138.

Hulluka M, Tadesse N (1994). Chickpea and lentil disease research in Ethiopia. In: A. Telaye, G. Bejiga, MC. Saxena, MB. Solh, eds., Cool season food legumes of Ethiopia, Proceedings of the first national cool-season food legumes review conference, Addis Ababa, Ethiopia, pp. 346-366.

Ibrikci H, Knewtson S, Grusak MA (2003). Chickpea leaves as a vegetable green for humans: evaluation of mineral composition. *J. Sci. Food Agric.* 83: 945–950.

IIPR (2008). Annual report, Indian Institute of Pulses Research (IIPR), Kanpur 208 024, Uttar Pradesh, India.

IIPR (2012). Annual report, Indian Institute of Pulses Research (IIPR), Kanpur 208 024, Uttar Pradesh, India.

Infantino A, Kharrat M, Riccioni L, Coyne CJ, McPhee KE, Grunwald NJ (2006). Screening techniques and sources of resistance to root diseases of food legumes. *Euphytica* 147: 201-221.

Iruela M, Castro P, Rubio J, Cubero JI, Jacinto C, Millán T, Gill J (2007). Validation of a QTL for resistance to ascochyta blight linked to resistance to Fusarium wilt race 5 in chickpea (*Cicer arietinum* L.). *European J. Plant Pathol.* 119: 29-37.

Iruela M, Rubio J, Barro F, Cubero JI, Millan T, Gill J (2006). Detection of two QTL for resistance to Ascochyta blight in an intraspecific cross of chickpea (*Cicer arietinum*). *European J. Plant Pathol.* 119: 39-51.

Jain KC, Reddy MV (1995). Inheritance of resistance to fusarium wilt in pigeonpea (*Cajanus cajan* (L.) Millsp.). *Indian J. Genet.* 44: 434-437.

Jiménez-Díaz RM, Alcalá-Jiménez AR, Hervás A, Trapero-Casas JL (1993). Pathogenic variability and hosts resistance in the Fusarium oxysporum f. sp. ciceris / Cicer arietinum pathosystem. In: Proc. 3rd Eur. Semin. Fusarium Mycotoxins, Taxonomy, Pathogenicity and Host Resistance. Hodowsla Ròslin Aklimatyazacja i Nasiennictwo. Plant Breeding and Acclimatization Institute, Radzików, Poland, pp. 87–94.

Jimenez-Diaz RM, Crino P, Halila MH, Mosconi C, Trapero-Casas A (1993). Screening for resistance to Fusarium wilt and Ascochyta blight in chickpea. In: KB. Singh, MC. Saxena, eds., Breeding for Stress Tolerance in Cool Season Food Legumes, ICARDA, Wiley-Sons Pvt. Co. UK, pp. 77-95.

Jimenez-Diaz RM, Trapero-Casas A, Cabrera de la Colina J (1989). Races of *Fusarim oxypsourm* f.sp. *ciceris* infecting chickpeas in Southern Spain. In: EC. Tiamos, C. Beckmann, eds., Vascular wilt Diseases of plants (Vol. H28), NATOASI Series, Springer-Verlag, Berlin, Germany, pp. 515-520.

Jimenez-Gasco MD, Perz-Artes E, Jimenez-Diaz RM (2001). Identification of pathogenic races 0, 1B/C, 5 and 6 of *Fusarium oxysporium* f. sp. *ciceris* with random amplified polymorphic DNA (RAPD). *Europe. J. Plant Pathol.* 107: 237-248.

Jiménez-Gasco MM, Jiménez-Díaz RM (2003). Development of a Specific Polymerase Chain Reaction-Based Assay for the Identification of *Fusarium oxysporum* f. sp. ciceris and Its Pathogenic Races 0, 1A, 5, and 6. *Phytopathol.* 93: 200-209.

Jimenez-Gasco MM, Milgroom MG, Jimenez-Diaz RM (2004). Stepwise evolution of races in *Fusarium oxysporum* f. sp. *ciceri* inferred from fingerprinting with repetitive DNA sequences. *Phytopathol.* 94: 228-235.

Joshi S (2001). Pathogenic variability in Pigeonpea wilt pathogen *Fusarium udum* butler in Nepal. *Nepal Agric. Res. J.* 4&5: 64-65.

Joshi S, Maharjan R (2003). Evaluation of Lentil genotypes to Fusarium wilt. In: Proceedings of 25th National Winter Workshop (Grain legumes), pp. 33-35.

Kamboj RK, Pandey MP, Chaube HS (1990). Inheritance of resistance to Fusarium wilt in Indian lentil germplasm (*Lens culinaris* Medic.). *Euphytica* 50: 113-117.

Kannaiyan J, Nene YL (1981). Influence of wilt at different growth stages on yield loss in Pigeonpea. *Trop. Pest Manag.* 27: 141.

Kannaiyan J, Nene YL, Reddy MV, Ryan TN, Raju TN (1984). Prevalence of pigeonpea diseases and associated crop losses in Asia, Africa and the Americas. *Trop. Pest Manag.*30: 62-712.

Kannaiyan J, Nene YL (1978). Host range of *Fusarium oxysporum* f.sp. *lentis*, *Sclerotinia rolfsii* and *Rhizoctonia solani* isolated from lentil. *LENS* 5: 12.

Kannaiyan J, Nene YL (1978). Strains of *Fusarium oxysporum* f. sp. *lentis* and their pathogenicity on some lentil lines. *LENS* 5: 8-10.

Karimi R, Owuoche JO, Silim SN (2012). Importance and management of Fusarium wilt (*Fusarium udum* Butler) of pigeonpea. *Intl. J. Agron. Agric. Res.* 2: 1-14.

Karki PB (1993). Plant protection of lentil in Nepal. In: Lentil in South Asia (eds., W. Erskine and M.C. Saxena), ICARDA, Aleppo, Syria, pp. 187-191.

Kelly A, Alcala-Jimenez AR, Bainbridge BW, Heale JB, Perez-Artes E, Jimenez-Diaz RM (1994). Use of genetic fingerprinting and random amplified polymorphic DNA to characterise pathotypes of *Fusarium oxysporum* f. sp. *ciceri* infecting chickpea. *Phytopathol.* 84: 1293-1298.

Khalid M, Saleem M, Ahsan M (2011). Inheritance of resistance to Fusarium wilts in chickpea. *Pak. J. Agri. Sci.* 48: 55-58.

Khan IA, Alam SS, Haq A, Jabbar A (2002). Selection for resistant to wilt and its relationship with phenols in chickpea. *Intl. Chickpea and Pigeonpea Newslett.* 9: 19-20.

Khare MN (1981). Diseases of lentil. In: Lentils (eds. Webb, C., Hawtin, G.,). Farnham Royal, England, UK, Commonwealth Agricultural Bureaux, pp. 163-172.

Khare MN, Agrawal SC, Dhingra OD, Kushwaha LS (1975). Variability in the growth of eight strains of *Fusarium oxysporum* f. sp. *lentis* on different solid media. India. *Phytopathol.* 28: 126-128.

Khare MN, Agrawal SC, Jain AC (1979). Lentil diseases and their control. *Tech. Bull.*, JNKVV, Jabalpur, India, pp. 29.

Kimani PM, Nyende S, Silim S (1994). Development of early maturing Fusarium wilt resistant pigeonpea genotypes. *African Crop Sci. J.* 2: 35-41.

Kiprop EK, Mwangombe AW, Baudoin JP, Kimani PM, Mergeai G (2002). Cultural characteristics, pathogenicity and vegetative compatibility of *Fusarium udum* isolates from pigeonpea (*Cajanus cajan*(L) Millsp. in Kenya. *European J. Plant Pathol.* 108: 147-154.

Kotova VV, Khaleeva JN, Shikuhova EG (1965). Fungus diseases of legume. In: Distribution of Pests and Diseases of Crops in the USSR in 1964. *Trudy Vses. Inst. Zasch. Past.* 25: 159-164.

Kotresh H, Fakrudin B, Punnuri BK, Rajkumar SM, Thudi M, Paramesh H, Lohithaswa H, Kuruvinashetti MS (2006). Identification of two RAPD markers genetically linked to a recessive allele of a Fusarium wilt resistance gene in pigeonpea (*Cajanus cajan* L. Millsp.). *Euphytica* 149: 113–120.

Kraft JM, Haware MP, Jimenez-Diaz RM, Bayaa B, Harrabi M (1994). Screening techniques and sources of resistance to root rots and wilts in cool-season food legumes. *Euphytica* 73: 27-39.

Kumar J, Choudhary AK, Solanki RK, Pratap A (2011). Towards marker-assisted selection in pulses: a review. *Plant Breed.* 130: 297-313.

Kumar J, Haware MP (1982). Inheritance of resistance to Fusarium wilt in chickpea. *Phytopathol.* 72: 1035-1036.

Kumar J, Haware MP, Smithson JB (1985). Registration of four short duration Fusarium wilt-resistant kabuli (Garbanzo) chickpea germplasms. *Crop Sci.* 25: 576–577.

Kumar S (1998). Inheritance of resistance of Fusarium wilt (race-2) in chickpea. *Plant Breed.* 117: 139-142.

Kumar S, Bourai VA (2012). Economic analysis of pulses production their benefits and constraints (A case study of sample villages of Assan valley of Uttarakhand, India). *J. Humanities and Social Sci.* 1: 41-53.

Kumar S, Sarker A, Erskine W, Geletu B, Ahmed S, Bayya B, El-Hassan H (2010). Valuable donors for Fusarium wilt resistance in lentil. In: Legumes for Global Health, Proceedings of the V International Food Legumes Research Conference and VII European Conference on Grain Legumes, 26-30 Apr 2010, Antalya, Turkey, pp. 37.

Kumar S, Kumar J, Singh S, Ahmed S, Chaudhary RG, Sarker A (2010). Vascular wilt disease of lentil: a review. *J. Lentil Res.* 4: 1-14.

Manucheri A, Mesri I (1966). Fusarium wilt of chickpea (In Persian). *J. Path.* 3: 1-11.

Matsumoto K, Barbosa ML, Souza LAC, Teixeira JB (1995). Race-1 Fusarium wilt tolerance on banana plants selected by fusaric acid. *Euphytica* 84: 67-71.

Mayer MS, Tullu A, Simon CJ, Kumar J, Kaiser WJ, Kraft JM, Muehlbauer FJ (1997). Development of a DNA marker for Fusarium wilt resistance in chickpea. *Crop Sci.* 37: 1625-1629.

Mbwaga AM (1995). Fusarium wilt screening in Tanzania. In: SN. Silim, SB. King, S. Tuwaje, eds., Improvement of pigeonpea in Eastern and Southern Africa, Annual Research Planning Meeting, 21-23 September 1994, ICRISAT, Patancheru, Andhra Pradesh, India, pp. 101-102.

Miller CD, Branthoover B, Sekiguchi N, Deming H, Bauer A (1956). Vitamin value of foods used in Hawaii. *Hawaii Agric. Exp. Station Tech. Bull.* 30: 303–313.

Mishra S (2004). Studies on variability in *Fusarium udum* Butler, the pathogen of pigeonpea wilt disease and identification of resistant donors. Ph.D. Thesis, CSJM University, Kanpur, India.

Moreau (1978). Diseases and pests of lentil. Institute National de Vulgarisation pour les Fruits, Legumes at Champignons, pp. 95-100.

Navas-Cortes JA, Hau B, Jimenez-Diaz RM (2000). Yield loss in chickpea in relation to development to Fusarium wilt epidemics. *Phytopathol.* 90: 1269-1278.

Nema KG, Khare MN (1973). A concept of wilt of Bengalgram in Madhya Pradesh. Symposium on wilt problem and breeding for wilt resistance in Bengal gram, September 1973, Indian Agricultural Research Institute New Delhi, India, pp. 4.

Nene YL, Haware MP (1980). Screening chickpea for resistacne to wilt. *Plant Dis.* 64: 379-380.

Nene YL, Haware MP, Reddy MV (1981b). Chickpea diseases: resistance-screening techniques, Information Bulletin No. 10, ICRISAT, Patancheru, AP, pp. 10.

Nene YL, Kannaiyan J (1982). Screening pigeonpea for resistance to Fusarium wilt. *Plant Dis.* 66: 306-307.

Nene YL, Kannaiyan J, Haware MP, Reddy MV (1980). Review of work done at ICRISAT on soil borne disease of pigeonpea and chickpea. In: Proceedings of the consultants group discussion on the resistance to soil borne diseases of legumes, 8-11 January 1979, ICRISAT Patancheru, India, pp. 3-39.

Nene YL, Kannaiyan J, Haware MP, Reddy MV (1981a). Pigeonpea Diseases: resistance screening techniques. Inform. Bull. No. 9. pp. 14, ICRISAT, Patancheru, A.P, India.

Odeny DA, Githiri SM, Kimani PM (2009). Inheritance of resistance to Fusarium wilt in pigeonpea (*Cajanus cajan (*L.) Millsp.). *J. Animal Plant Sci.* 2: 89-95.

Okiror MA (2002). Genetics of resistance to *Fusarium udum* in pigeonpea [*Cajanus cajan* (L.) Millsp. *Indian J. Genet.* 62: 218-220.

Okiror MA, Kimani PM (1997). Pathogenic variation of *Fusarium udum* of Pigeonpea. *Indian J. Genet.* 57: 186-192.

Padwick OW (1941). Report of the imperial mycologist. Scientific Report, 1939-40, Indian Agricultural Research Institute, New Delhi, India, pp. 94-101.

Pal BP (1934). Recent progress in plant breeding at pusa, Bihar. *Agric. Livestock India* 4: 511-512.

Pande S, Kishore GK, Upadhyaya HD, Rao JN (2006). Identification of multiple diseases resistance in mini-core collection of chickpea. *Plant Dis.* 90: 1214–1218.

Pande S, Sharma M, Gopika G, Rameshwar T (2012b). High throughput phenotyping of pigeonpea diseases: Stepwise identification of host plant resistance. Information Bulletin No. 93, ICRISAT, Patancheru, Andhra Pradesh, India.

Pande S, Sharma M, Nagavardhini A, Rameshwar T (2012a). High throughput phenotyping of chickpea diseases: Stepwise identification of host plant resistance. Information Bulletin No. 92, ICRISAT, Patancheru, Andhra Pradesh, India.

Pandey RN, Pawar SE, Bhatia CR (1995). Effect of culture filtrate of *Fusairum udum* and fusaric acid on wilt susceptible and resistant pigeonpea cultivars. *Indian Phytopathol.* 48: 444-448.

Pandey RN, Pawar SE, Bhatia CR (1996). Inheritance of wilt resistance in pigeonpea. *Indian J. Genet.* 56: 305–308.

Pandya BP, Pandey MP, Singh JP (1980). Development of Pant L 406 lentil resistant to rust and wilt. *LENS* 7: 34-37.

Pathak GN (1970). Redgram. Pulse crops of India (Kachroo, P., ed.). IARI, New Delhi, India, pp. 14-53.

Pathak MM, Singh KP, Lal SB (1975). Inheritence of resistance to wilt (*F. oxysporum* f. sp. *ciceris*) in gram. *Indian J. Farm Sci.* 3: 10–11.

Phillips JC (1988). A distinct race of chickpea wilt in California. *Intl. Chickpea Newslett.* 18: 19-21.

Prasanthi L, Bhasker Reddy BV, Rekha Rani K, Haranath Naidu P (2009). Molecular marker for screening Fusarium wilt resistance in Pigeonpea [*Cajanus cajan* (L.) Millspaugh]. *Legume Res.*32: 19-24.

Rahman ML, Haware MP, Mian IH, Akananda AM (1998). Races of *Fusarium oxysporum* f. sp. *ciceri* causing chickpea wilt in India. *Bangladesh J. Plant Pathol.* 14: 234-237.

Rahman ML, Haware MP, Miran IH (2000). Pathogenic variability among chickpea wilt Fusaria. *Bull. Institute Trop. Agric. (Kyushu Univ.)* 23: 7-13.

Rahman MM, Sarker A, Kumar S, Ali A, Yadav NK, Rahman ML (2009). Breeding for short season environments. In: W. Erskine, FJ. Muehlbauer, A. Sarker, B. Sharma, eds., The Lentil: Botany, Production and Uses, CAB International, Wallingford, U.K., pp. 121-136.

Ratna Babu D, Ravikumar RL (2010). Genetic evidence for resistance to Fusarium wilt of pollen grains in chickpea (*Cicer arietinum* L.). *Curr. Sci.* 96: 811-815.

Ratnaparkhe M, Santra DK, Tullu A, Muehlbuer FJ (1998a). Inheritance of inter-simple-sequence-repeat polymorphisms and linkage with a Fusarium wilt resistance gene in chickpea. *Theor. Appl. Genet.* 96: 348-353.

Ratnaparkhe MB, Tekeoglu M, Muehlbauer FJ (1998b). Inter-simple-sequence-repeat (ISSR) polymorphism are useful for finding markers associated with disease resistance gene clusters. *Theor. Appl. Genet.* 97: 515-519.

Ravikumar RL, Chaitra GN, Choukimath AM, Soregaon CD (2013). Gametophytic selection for wilt resistance and its impact on the segregation of wilt resistance alleles in chickpea (*Cicer arietinum* L.). *Euphytica* 189: 173-181.

Reddy MV, Nene YL, Kannaiyan J, Raju TN, Saka VN, Davor AT, Songa WP, Omanga P (1990). Pigeonpea lines resistant to wilt in Kenya and Malawi. *Intl. Pigeonpea Newslett.* 16: 34.

Reddy MV, Raju TN, Sharma SB, Nene YL, McDonald D (1993). Handbook of pigeobpea diseases, (In En. Summaries in En. Fr.). Information Bulletin 14. ICRISAT, Patancheru, A.P. 502 324, India.

Reddy MV, Sharma SB, Nene YL (1990). Pigeonpea: Disease management. In: YL. Nene, SD. Hall, VK. Sheila, eds., The pigeonpea, CAB International, Wallingford, Oxon, UK.

Riccioni L, Giambattista GD, Valvassori M, Malta R, Porta-Puglia A (2003). Fungi associated with root rot of lentil landrace from Sicily. *J. Plant Pathol.* 85: 300.

Rubio J, Hajj-Moussa E, Kharrat M, Moreno MT, Millan T, Gill J (2003). Two genes and linked RAPD markers involved in resistance to *Fusarium oxysporium* f. sp. *ciceris* race 0 in chickpea. *Plant Breed.* 122: 188-191.

Rubio J, Martínez C, Gill J, Moreno MT (2004). Registration of Ascochyta Blight and Fusarium Wilt Resistant CA2954 Kabuli Chickpea Germplasm. *Crop Sci.* 44: 1881-1882.

Sabbavarapu MM, Mamta S, Chamarthi SK, Rathore A, Thudi M, Gaur PM, Pande S, Singh S, Kaur L, Varshney RK (2013). Molecular mapping of QTLs for resistance to Fusarium wilt (race 1) and Ascochyta blight in chickpea (*Cicer arietinum* L.). *Euphytica* (DOI: 10.1007/s10681-013-0959-2).

Sarker A, Erskine W (2002). Lentil production in the traditional lentil world. In: Proceedings of Lentil Focus (ed. Brouwer, J.B.,) Horsham, Victoria, Australia. Pulse Australia, Sydney, pp. 35-40.

Saxena KB, Kumar RV, Gowda CLL (2010). Vegetable pigeonpea – a review. *J. Food Leg.* 23: 91-98.

Saxena KB, Kumar RV, Saxena RK, Sharma M, Srivastava RK, Sultana R, Varshney RK, Vales MI, Pande S (2012). Identification of dominant and recessive genes for resistance to Fusarium wilt in pigeonpea and their implication in breeding hybrids. *Euphytica* 188: 221–227.

Sharma KD, Chen W, Muehlbauer FJ (2005a). Genetics of chickpea resistance to five races of Fusarium wilt and a concise set of race differentials for *Fusarium oxysporum* f. sp. *ciceris*. *Plant Dis.* 89: 385-390.

Sharma KD, Muehlbauer FJ (2005b). Genetic mapping of *Fusarium oxysporum* f. sp. *ciceris* race-specific resistance genes in chickpea (*Cicer arietinum* L.). In: Abstract of the International food legume research conference—IV, Indian Agricultural Research Institute, New Delhi, India, pp. 18–22.

Sharma KD, Muehlbauer FJ (2007). Fusarium wilt of chickpea: physiological specialization, genetics of resistance and resistance gene tagging. *Euphytica* 157: 1– 14.

Sharma KD, Winter P, Kahl G, Muehlbauer FJ (2004). Molecular mapping of *Fusarium oxysporum* f. sp. *ciceris* race 3 resistance gene in chickpea. *Theor. Appl. Genet.* 108: 1243-1248.

Sharma M (2013). Race scenario of *Fusarium oxysporum* f sp. *ciceris,* wilt pathogen of chickpea (*Cicer arietinum* L.)". 12th European Fusarium seminar, palais de la bourse, Bordeaux, France, 12-16 May 2013.

Sharma M, Kiran Babu T, Gaur PM, Ghosh R, Rameshwar T, Chaudhary RG, Upadhyay JP, Gupta O, Saxena DR, Kaur L, Dubey SC, Anandani VP, Harer PN, Rathore A, Pande S (2012). Identification and multi-environment validation of resistance to *Fusarium oxysporum* f. sp. *ciceris* in chickpea. *Field Crops Res.* 135: 82–88.

Sharma M, Rathore A, Mangala NU, Ghosh R, Sharma S, Upadhyaya HD, Pande S (2012). New sources of resistance to Fusarium wilt and sterility mosaic disease in a mini-core collection of pigeonpea germplasm. *European J. Plant Pathol.* : 707-714.

Shaw FJC (1936). Studies in Indian pulses. The inheritance of morphological characters and wilt resistance in arhar (*Cajanus indicus* Spreng.). IARI, New Delhi, India 6, 139-188.

Shehabu M, Ahmed S, Sakhuja PK (2008). Pathogenic variability in Ethiopian isolates of *Fusarium oxysporum* f. sp. *ciceris* and reaction of chickpea improved varieties to the isolates. *Intl. J. Pest Manage.* 54: 143-149.

Sindhu JS, Singh KP, Slinkard AE (1983). Inheritance of resistance to Fusarium wilt in chickpeas. *J. Heredity* 74: 68.

Singh F, Singh IP, Mazumder ND (2011). Identification of Fusarium wilt-resistant sources of long-duration pigeonpea (Cajanus cajan). *Indian J. Agri. Sci.* 81: 1046–1051.

Singh IP, Dhar V, Chaudhary RG, Pandey DK (1998). Genetic of Fusarium wilt resistance in pigeonpea. In: National Symposium on Management of Biotic and Abiotic Stresses in Pulse Crops, 26–28th June 1998, IIPR, Kanpur, p. 15.

Singh IS, Singh JP, Singh AK, Chauhan MR (1994). Pant lentil 4: A high-yielding, rust, wilt and

blight resistant variety for the north-western plains of India. *LENS* 21: 8-9.

Singh KB, Malhotra RS, Halila H, Knights EJ, Verma MM (1994). Current status and future strategy in breeding chickpea for resistance to biotic and abiotic stresses. *Euphytica* 73: 137-149.

Singh LI, Kumar J, Smithson JB, Haware MP (1987b). Genetics of resistance to Fusarium wilt in chickpeas. In: PR. Day, GJ. Jellis, eds., Genetics and Plant Pathogenesis, Blackwell Scientific Publications, Oxford, UK, pp. 339-342.

Singh LI, Kumar J, Smithson JB, Haware MP (1987a). Complementation between genes for resistance to race 1 of *Fusarium oxysporum* f. sp. *ciceri* in chickpea. *Plant Pathol.* 36: 39-43.

Sivaramakrishnan S, Seethakannanand, Singh SD (2002). Detection of genetic variability in *Fusarium udum* using DNA markers. *Indian Phytopathol.* 55: 258-263.

Smithson JB, Kumar JB, Haware MP, Singh HP (1983). Complementation between genes for late wilting of race 1 of *Fusarium oxysporum* f. sp. *ciceri* in chickpea. *Proc. Int. Genet. Cong.*, IARI, New Delhi, India.

Soko HN (1992). Pigeonpea research and development in Malawi. *Intl. Pigeonpea Newslett.* 16: 59-63.

Solanki RK, Singh S, Kumar J (2010). Molecular markers assisted testing of hybridity of F1 plants in lentil. *J. Food Leg.* 23: 21-24.

Songa WA, King SB, Omang PA (1995). Pigeonpea pathology research in Kenya. In: SN. Silim, SB. King, S. Tuwaje, eds., Improvement of pigeonpea in Eastern and Southern Africa. Annual Research Planning Meeting 1994, 21-23 September 1994, Nairobi, Kenya. ICRISAT, Andhra Pradesh, India, pp. 30-37.

Soregaon CD, Ravikumar RL (2012). Segregation of Fusarium wilt resistance in recombinant inbred lines of two diverse crosses of chickpea (*Cicer arietinum* L.). *Karnataka J. Agric. Sci.* 25: 127-128.

Soregaon CD (2011). Mapping molecular marker linked to H2 locus of wilt resistance in chickpea and to locate QTLs linked to other quantitative traits. Ph.D. thesis submitted to UAS, Dharwad, Karnataka, India.

Srivastava SK, Singh SN, Khare MN (1984). Assessment of yield losses in some promising gram cultivars due to Fusarium wilt. *Indian J. Pl. Prot.* 12: 125-126.

Sukumar M, Bakshi A, Bandamaravuri KB, Reddy SS, Saxena S, Arora DK (2012). Genetic diversity and pathogenic variability among Indian isolates of *Fusarium udum* infecting pigeonpea (*Cajanus cajan*(L.) millsp.). *Intl. Res. J. Agric. Sci. Soil Sci.* 2: 51-57.

Taheri N, Fallahati R, Jafarpour B, Bagheri AR, Jahanbaghsh V (2010). Pathogenic and genetic characterization of *Fusarium oxysporum* f. sp. *lentis* by RAPD and IGS analysis in Khorasan province. *World Appl. Sci. J.* 9: 239-244.

Tanksley SD, Nelson JC (1996). Advanced backcross QTL analysis: a method for the simultaneous discovery and transfer of valuable QTLs from unadapted germplasm into elite breeding lines. *Theor. Appl. Genet.* 92: 191–203.

Tekeoglu M, Rajesh PN, Muehlbauer FJ (2002). Integration of sequence tagged microsatellite sites to the chickpea genetic map. *Theor. Appl. Genet.* 105: 847-854.

Tekeoglu M, Tullu A, Kaiser WJ, Muehlbauer FJ (2000). Inheritance and linkage of two genes that confer resistance to Fusarium wilt in chickpea. *Crop Sci.* 40: 1247-1251.

Tiwari S, Dhar V (2011). Prevalence of new variants of *Fusarium udum* in India. *Indian Phytopathol.* 64: 243-246.

Trapero-Casas A, Jiménez-Díaz RM (1985). Fungal wilt and root rot diseases of chickpea in southern Spain. *Phytopathol.* 75: 1146–1151.

Tullu A (1996). Genetics of Fusarium wilt resistance in chickpea. PhD Thesis, Washington State University, Pullman, U.S.A.

Tullu A, Kaiser WJ, Kraft JM, Muehlbauer FJ (1999). A second gene for resistance to race 4 of Fusarium wilt in chickpea and linkage with a RAPD marker. *Euphytica* 109: 43–50.

Tullu A, Muehlbauer FJ, Simon CJ, Mayer MS, Kumar J, Kaiser WJ, Kraft JM (1998). Inheritance and linkage of a gene for resistance to race 4 of Fusarium wilt and RAPD markers in chickpea. *Euphytica* 102: 227-232.

Ujevic I, Kovacikova E, Urosevic B (1965). *Botrytis parasitica* and *Fusarium oxysporum* as dangerous parasites of lentil. *Polnohospodarstve* 11: 584-594.

Upadhyaya HD, Haware MP, Kumar J, Smithson JB (1983a). Resistance to wilt in chickpea. I. Inheritance of late wilting in response to race 1. *Euphytica* 32: 447-452.

Upadhyaya HD, Smithson JB, Haware MP, Kumar J (1983b). Resistance to wilt in chickpea. II.

Further evidence for two genes for resistance to race 1. *Euphytica* 32: 749-755.

Varshney RK, Chen W, Li Y, Bharti AK, Saxena RK, Schlueter JA *et al.* (2012a). Draft genome sequence of pigeonpea (*Cajanus cajan*), an orphan legume crop of resource-poor farmers. *Nature Biotech.* 30: 83–89.

Varshney RK *et al.* (2013). Draft genome sequence of chickpea (*Cicer arietinum*) provides a resource for trait improvement. *Nature Biotech.* 31: 240-248.

Varshney RK, Kudapa H, Roorkiwal M, Thudi M, Pandey MK, Saxena RK *et al.* (2012b). Advances in genomics research and molecular breeding applications in SAT legume crops by using next generation sequencing and high-throughput genotyping technologies. *J. Biosci.* 37: 811–820.

Varshney RK, Mohan SM, Gaur PM, Gangarao NV, Pandey MK, Bohra A, Sawargaonkar SL, Chitikineni A, Kimurto PK, Janila Pasupuleti, Saxena KB, Fikre Asnake , Sharma Mamta, Rathore A, Pratap Aditya, Tripathi Shailesh, Datta Subhojit, Chaturvedi SK, Mallikarjuna Nalini, Anuradha G, Babbar Anita, Choudhary AK et al. (2013). Achievements and prospects of genomics-assisted breeding in three legume crops of the semi-arid tropics. *Biotech. Adv.* 31: 1120-1134.

Vasudeva RS, Srinivasan XV (1952). Studies on the wilt disease of lentil (*Lens esculenta* Moench.). *Indian Phytopathol.* 5: 23-32.

Whiteman PC, Norton BW (1980). Alternative uses of pigeonpea. *Proc. Intl. Workshop Pigeonpeas*, ICRISAT, Patancheru, 502 324, Andhra Pradesh, India, pp. 365–378.

Wilson VE, Brandsberg J (1965). Fungi isolated from diseased lentil seedlings in 1963-64. *Plant Dis. Rep.* 49: 660-662.

Winter P, Benko-Iseppon AM, Huttel B, Ratnaparkhe M, Tullu A, Sonnante G, Pfaff T, Tekeoglu M, Santra D, Sant VJ, Rajesh PN, Kahl G, Muehlbauer FJ (2000). A linkage map of the chickpea (*Cicer arietinum* L.) genome based on recombinant inbred lines from *C. arietinum* x *C. reticulatum* cross: localization of resistance genes for Fusarium wilt races 4 and 5. *Theor. Appl. Genet.* 101: 1155-1163.