Genetic Options for Managing Biotic Stresses in Pulse Crops

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Abstract

In general, the productivity of pulses is low and unstable, primarily due to large number of abiotic and biotic constraints. Thus, major efforts in genetic improvement of pulses have gone on resistance breeding. Excellent progress through traditional breeding methods has been made in development of varieties resistant to some diseases, *e.g.,* fusarium wilt in chickpea and pigeonpea; sterility mosaic in pigeonpea; mungbean yellow mosaic virus (MYMV) in mungbean and urdbean; powdery mildew in urdbean, mungbean and pea; and rust in pea and lentil. Similar level of success could not be achieved in breeding for resistance to other biotic stresses due to unavailability of high level of resistance in the cultivated and cross compatible wild species. Mutation breeding has been rewarding in development of disease resistant varieties in chickpea and mungbean. Sources of high level of resistance have been identified in the wild species for several biotic stresses. Resistance to some stresses is available only in the wild species, for example, resistance to cyst nematodes and bruchids in wild *Cicer* species. However, limited progress has been made in introgression of resistance genes from wild species into the cultivated species, mainly because of barriers to interspecific hybridization in many cases. Concerted efforts are required to understand and to find the ways to overcome the barriers to hybridization between the cultivated species and the wild species of tertiary gene pool. New tools of biotechnology such as transgenic and molecular marker assisted technologies are now available that can be used to accelerate the progress of crop improvement.

1. Introduction

The importance of pulse crops in human, animal and soil health is well known to researchers, policy makers, extension personnel and farmers. However, these crops often receive low priority in cultivation by farmers as compared to rice, wheat and other commercial crops. This is mainly because the yield of pulse crops is generally low and variable. Several factors are responsible for poor and unstable yield of pulse crops, the most important being biotic and abiotic stresses. Sub-optimum growing conditions and poor management provided by farmers to these crops often elevate the effects of these stresses. Estimates of yield losses caused to pulses by individual biotic stresses range from 5 to 30% in India (Table 1). Thus, effective management of biotic stresses in pulse crops can substantially contribute to stability and enhancement of pulses production.

Table 1: Potential disease threats for pulse crops in India

Crop	Disease	Growing states	Extent of damage (%)
Chickpea	Fusarium wilt + root rots	Eastern U.P., Bihar, Jharkhand, Assam, W.B., Rajasthan, Gujarat, Maharashtra, M.P., Chhattisgarh, Orissa, A.P., Karnataka, and T.N.	20-25
	Ascochyta blight	Himachal Pradesh, Jammu & Kashmir, Punjab, Haryana, Rajasthan, Western U.P. and Uttaranchal	5-10
	Botrytis gray mold	Tarai areas of U.P., and Bihar	5-10
Pigeonpea	Fusarium wilt	U.P., Bihar, Jharkhand, W.B., Rajasthan, Gujarat, Maharashtra, M.P., Chhattisgarh, A.P., Karnataka, and T.N.	10-15
	Phytophthora blight	U.P., Bihar, Jharkhand, Punjab, Haryana, and Rajasthan	5-10 (up to 25% in early crop)
	Sterility mosaic	U.P., Bihar, Jharkhand, W.B., Rajasthan, Gujarat, Maharashtra, M.P., Chhattisgarh, A.P., Karnataka, and T.N.	10-15
	Alternaria blight	Eastern U.P., Bihar (Pre-*Rabi* crop)	10-15
Mungbean	MYMV + Cercospora leaf blight	U.P., Haryana, Punjab, Bihar, Jharkhand, Assam, and W.B.	15-20
Urdbean	Powdery mildew and leaf spots	Coastal area of A.P., T.N. and Orissa	15-25
	MYMV	U.P., Haryana, Punjab, Bihar, Jharkhand, Assam, and W.B.	15-20
Pea	Powdery mildew	All crop growing areas	15-20
	Rust	Eastern U.P., Bihar, Jharkhand, Assam, and W.B.	10-15
Lentil	Wilt	Bundelkhand and Eastern U.P., Haryana, and Rajasthan	20-25
	Rust	Eastern U.P., Bihar, Jharkhand, Assam, and W.B.	10-15
Common bean	BCMV	All growing areas	Up to 30

Host-plant resistance offers the most economical, environmentally acceptable and long-term means of controlling biotic stresses. This paper provides an overview of the various options available for enhancing host-plant resistance to biotic stresses in important pulse crops of India.

2. Important Biotic Stresses

2.1 Chickpea

Fusarium wilt, caused by *Fusarium oxysporum* f. sp. *ciceri*, is the most widespread soil borne disease of chickpea. Seven races of the pathogen are known, races 1 to 4 from India (Haware and Nene, 1982), races 0 and 5 from Spain (Jimenez-Diaz *et al.*, 1989) and race 6 from the USA (Phillips, 1988). Inheritance studies indicate that three loci control resistance to race 1 (Singh *et al.*, 1987) as well as to race 2 (Kumar, 1998). Two other loci control resistance to race 4 (Tullu *et al.*, 1999) and one locus each to race 0 and 5 (Tekeoglu *et al.*, 2000b). Several stable sources of resistance have been identified that make breeding for resistance to fusarium wilt an easy task.

Collar rot, caused by *Sclerotium rolfsii* Sacc., and dry root rot, caused by *Macrophomina phaseolina* (Tassi) Goid. (sclerotial state *Rhizoctonia bataticola* (Taub.) Briton-Jones), are the important soil borne diseases in the semi-arid tropics. There has not been much work on characterization of pathogenic variability for these diseases. Inheritance studies suggest that resistance to dry root rot is controlled by a dominant gene (Rao and Haware, 1987). Several genotypes with moderate resistance to dry root rot have been identified at ICRISAT (Pundir *et al.*, 1988) and some of these are also resistant to fusarium wilt (Nene *et al.*, 1989). Sources of moderate level of resistance have also been identified for collar rot (Chitale *et al.*, 1990).

Ascochyta blight, caused by *Ascochyta rabiei* (Pass.) Labr., is the most important foliar disease of chickpea and occurs mainly in areas where cool and humid weather occurs during the crop season. Pathogen is known to be highly variable, but a standard set of chickpea differentials has not been established that can help in identification of races. Several sources of moderate resistance have been identified (Singh and Reddy, 1993). Progress on breeding for resistance to ascochyta blight has been recently reviewed (Malhotra *et al.*, 2003).

Botrytis gray mold (BGM), caused by *Botrytis cinerea* Pres., is another important foliar disease of chickpea in northern India, particularly northeastern region where high humidity and mild temperatures prevail during crop growth, particularly at flowering time. The pathogen of BGM appears to be highly variable. Two dominant genes with epistatic interaction have been identified to confer resistance (Chaturvedi *et al.*, 1995). The genotypes with erect plant type that do not allow buildups of humidity in the plant canopy (*e.g.*, ICCL 87322 and ICCV 88510) are less affected by the disease (Haware and McDonald, 1993).

Pod borer (*Helicoverpa armigera* Hubner) is the most devastating insect-pest of chickpea globally. The breeding for resistance to gram pod borer remains a challenge

in absence of sources of good level of resistance. Several techniques for screening of germplasm for resistance to pod borer are now available and many genotypes that show low to moderate level of resistance have been identified (Sharma *et al.*, 2003)

2.2 Pigeonpea

Wilt, caused by *Fusarium udum*, is the most important disease with annual losses estimated to be around US $ 41 million (Kannaiyan *et al.*, 1984). The genetics of wilt resistance is complex and has not been fully understood. Pathogenic variability and physiologic races have been reported. Based on the reaction of four pigeonpea lines, 11 isolates from India were divided into three distinct groups (ICRISAT, 1996). A number of moderately resistant lines in all the maturity groups are available (Reddy *et al.*, 1993, Amin *et al.*, 1993).

Sterility mosaic disease (SMD) is a serious threat to pigeonpea production in all parts of India. It is characterized by pale green mosaic symptoms on leaves, excessive vegetative growth and lack of flowers (sterility). An early infection to the plants causes up to 100% yield losses (Kannaiyan *et al.*, 1984). The causal organism of SMD remained a mystery for a long time and was tentatively named pigeonpea sterility mosaic virus (PPSMV). Extensive efforts made at ICRISAT on characterization of PPSMV led to identification of a virus with a 32-kDa nucleoprotein and flexuous filamentous particles and segmented ssRNA genome. This is a novel virus with properties unrelated to any characterized virus (Lava Kumar, personal communication). SMD is reported to be controlled by four independent non-allelic genes (Singh *et al.*, 1983) and by four alleles at two loci (Sharma *et al.*, 1984). In most pigeonpea growing areas both wilt and sterility mosaic diseases are problems, therefore, genotypes with combined resistance to both the diseases are needed. Recently, sensitive methods for detection of PPSMV and its biotypes have been developed at ICRISAT. These developments will further facilitate rapid progress in development of SMD resistant pigeonpea varieties.

Phytophthora blight, caused by *Phytophthora drechsleri*, is a problem when pigeonpea crop is subjected to waterlogging at early growth stage. It affects the collar region and all aboveground parts of the plant. Due to highly variable nature of the pathogen, stable sources of resistance to this disease are not available.

Pigeonpea is fed upon by over 200 species of insects. It, however, being a perennial plant, rapidly recovers the damage caused to its vegetative parts. But recovery of the reproductive parts is low. Podfly *(Melanagromyza obtusa)* and pod borers (*Helicoverpa armigera* and *Maruca vitrata*) are major insect pests. Using field-screening methodology, sources of resistance to all the three major insect pests

have been identified from pigeonpea germplasm including its wild relatives. The resistance level in these sources is low.

2.3 Pea

Powdery mildew, caused by *Erysiphe pisi* Syd., occurs widely in India. It turns the plant white and seriously affects the photosynthetic activity of the plants. It adversely affects quality of green pods and seeds. Several sources of stable resistance are available. Two recessive genes, *er*1 and *er*2, are responsible for resistance. The resistant cultivars have been found to have higher levels of phenolics and phenol oxidizing enzymes than the susceptible cultivars. Among other diseases, root rots caused by *Pythium* spp., *Aphanomyces euteiches* f.sp. *pisi* and *Fusarium solani* are also important (Sharma and Khan, 1997). Their significance in causing yield damage, however, varies with the agro-ecological conditions. Among insect pests, pea and bean weevil, pea moth, pea aphid and pod borer are some of the worldwide pests. Integrated efforts have been made to identify and breed for resistance to *Bruchus pisorum* (Hardie, 1992)

2.4 Lentil

Vascular wilt, caused by *Fusarium oxysporum* Schlecht. Ex Fr. f. sp. *lentis*, inflicts major economic losses in India. The disease appears at seedling stage and also during reproductive growth. Sources of resistance have been identified (Bayaa and Erskine, 1998; Dua *et al.*, 2002) and resistance is under control of five independent genes (Kamboj *et al.*, 1990).

Rust, caused by *Uromyces viciae-fabae* (Pers.) Schroet., is the most important foliar disease of lentil, particularly in areas with high relative humidity. The disease may cause complete crop loss when it occurs at early stage. Several sources of resistance have been identified (Bayaa and Erskine, 1998; Dua *et al.*, 2002). Resistance is known to be under control of a dominant gene (Sinha and Yadav, 1989).

2.5 Mungbean and Urdbean

Mungbean yellow mosaic virus (MYMV) causes serious yield losses in both the rainy and summer season crops. Multilocational evaluation of genotypes for resistance to MYMV revealed differential reaction (Amin and Singh, 1989). Few genes probably control the resistance.

Powdery mildew (*Erysiphe polygoni* D.C.) is a highly devastating disease of winter-sown crop. It is the most important disease in urdbean grown following rainy-season rice in southern India (Satyanarayana, 1989). Limited information is available on pathogenic variability and genetics of resistance.

Cercospora leaf spot (*Cercospora canesceus* Ell. and Martin and *C. cruenta* Sacc.) is an important disease during rainy season. Involvement of different species in causing cercospora leaf spot complicates characterization of species.

2.6 Common Bean

Bean common mosaic virus (BCMV) is the most important disease of common bean and occurs in all regions where common bean is grown. Typical BCMV symptoms include dark green sectors on a lighter green background, usually accompanied by downward curling of the leaf margins. In cases of severe infection, there may be leaf distortion and blistering, stunting of growth and distortion of flowers and pods. Studies indicate that *I* gene confers resistance to most strains of BCMV. The alleles of *bc* gene are responsible for differential reactions of genotypes for resistance. A procedure for screening against BCMV has been suggested by Drijfhourt (1978) in which inoculum was sprayed with a suspension of BCMV particles mixed with carborundum. Recently, BCMV tolerant variety 'Amber' has been released for cultivation in northern India.

3. Genetic Options for Managing Biotic Stresses

3.1 Conventional Breeding

The conventional method of resistance breeding depends on sources of resistance available in the primary gene pool and the ability to identify the resistant genotypes in germplasm and the segregating populations. Thus, the success of this method largely depends on the level of resistance available for a stress in the cross compatible germplasm and the effectiveness of the resistance screening method.

Considerable efforts have been made on screening of germplasm for resistance to stresses and good sources of resistance have been identified for many stresses (Table 2). Varieties resistant/tolerant to diseases/insect-pests have been developed (Table 3). Excellent progress has been made in development of varieties with high level of resistance to diseases where resistance is controlled monogenically or oligogenically, *e.g.*, fusarium wilt in chickpea and pigeonpea; powdery mildew in urdbean, mungbean and pea; sterility mosaic in pigeonpea; mungbean yellow mosaic virus in mungbean and urdbean. In absence of donors with stable and high level of resistance against many diseases breeding efforts could not produce desired results, *e.g.*, resistance to root rots and BGM in chickpea, and *Phytophthora* blight in pigeonpea.

It is important to use diverse sources of resistance in the breeding programmes to minimize chances of breakdown of resistance in all varieties together by evolution of new pathotypes. Multiple crosses may be used to pyramid resistance genes from

Table 2: Sources of resistance to important biotic stresses in pulses

Crop	Disease/insect-pest	Source of Resistance
Chickpea	Fusarium wilt	Phule G 95007, KWR 108, ICCV 10, GPF 2, IPC 99-13, IPC 99-1, IC 2862, IC 10149, ICC 9023, ICC 9032, IPC 2000-14, IPC 2000-41, ICC 11550, GL 91061, CPS 1, WR 315, JG 74, JG 1265, GL 8834, GL 87079, ICCC 42, H 86-72, IPC 99-10, IPCK 9-3, KPG 259-4, GL 86123, H 86-18, IPC 2000-18, IPC 2000-52
	Ascochyta blight	E 100 Ym, E 100 Y, Phule G 82-1, EC 26446, BRG 8, ICC 7002, GL 84038, GL 84099, BG 276, H 82-5, H 86-18, H 75-35, GLK 88016, BG 257, GL 90169
	Botrytis gray mold	BG 276, ICC 1069, IC 12483, Dhanush, ICCW 92, ICCV 41
	Root rots	H 208, ICC 596, Co G 6, RSG 865
	Pod borer	ICC 506, ICC 5264, ICC 6663, ICC 10619, ICC 10667, ICC 10817, ICC 10817, ICC 12475, ICCL 86102, ICCL 86103, ICCV 7, ICCV 10, C 235, JG 74, PDE 2, Anupam, Pusa 261, Vijay, Vishal
Pigeonpea	Fusarium wilt (FW)	PI 397430, PR 5149, MA 3, ICP 8959, ICP 8863, ICP 9120, ICP 9174, ICP 9177, ICP 10269, ICP 12731 , ICP 12745, ICP 12748, ICP 12758, ICPL 84008, ICPL 89048, ICPL 89049, AWR 74/15, BWR 254, 370, Banda Palera, GPS 26C, Sharan 1-21, Sujata 1-2, IPA 92-1, BWR 23
	Sterility mosaic (SM)	PI 397430, ICP 87119, Narendra 1, BSMR 380, BSMR 736, CP 6997, ICP 7035, ICP 7867, ICP 7998, ICP 8362, ICP 8862, ICP 10976, ICP 10979, ICP 11049, ICP 11204, ICP 11206, ICP 11207, ICP 11231,ICPL 83024, ICPL 90002, ICPL 90011, BSMR 1, BSMR 2, Bahar, DA 11, Purple 1, KA 32-1, Pusa 14
	Phytopthora stem blight (PSB)	KPBR 80-2-1, ICPL 84023
	FW+SM	ICP8860,11298,14271,PR5149, PI 397430, Sel 1, DPPA 85-8,85-11,85-12,85-13,85-14, ICPL 89020, ICPL 880063
	Wilt+SM+PSB	KPL 43, KPL 44
	Pod borer (*H. armigera*)	T21, Bori, BDN 2, ICPL 332, PPE 45-2 (ICP 1964), MA2 Bahar, ICPL 84060, Pant A1, BSMR1
	Pod borer (*M. vitrata*)	ICPL 98001, ICPL 98003, ICPL 98008, ICPL 989014
	Pod fly	ICP 10531-E1, ICP 7941 E1, ICP 7946 E1, ICP 7176-5, KM 7

Mungbean	MYMV	MH 303, ML 5, ML 131, ML 267, ML 353, ML 508, NDM 88-14, PDM 84-143, PDM 84-139, JRUM 1, JRUM 2
Urdbean	MYMV	Uttara, JU 3, DPU 88-1, DPU 88-31, K 66-110, Mash 1-1, NDU 88-8, NP 16, NP 19, UG 135, TAU 5, Pant U 19
Lentil	Rust	Pant L 406, Pant L 639, Precoz 3, Vipasha, Pant L 77-2, DPL 62, Pant L 5
	Wilt	Pant L 77-2, DPL 58, DPL 62, Pant L 5
Pea	Powdery Mildew	DMR 11, Pant P 5, Malviya 15 (HUDP 15)
	Pod borer	EC 33860, Bonville, T 6113, PS 410, 2S 21, 172 M, PS 410

Table 3: Varieties resistant/tolerant to diseases/insect-pests released in India

Crop	Disease	Resistant Varieties
Chickpea	Fusarium wilt	JG 315, JG 11, Avarodhi, Phule G 5, KWR 108, DCP 92-3, JG 74, Vijay, Alok, ICCV 10, GPF 2, Vishal, H 82-2, ICCV 32
	Ascochyta blight	Gaurav, C 235, GNG 146, BG 261, PBG 1, PBG 5
	Root rot	H 355, H 208
	Pod borer	C 235, Pant G 114, Anupam, JG 74, ICCV 10, Pusa 261, Vijay, Vishal
Pigeonpea	Fusarium wilt	BDN 1, BDN 2, C11, TT6, ICP 8863 (Maruthi), Asha, BSMR 736, DA 11
	Sterility mosaic	Bahar, DA 11, Hy 3C, Pusa 9, ICPL 366
	Alternaria blight	WB 20, Pusa 9, DA 11
	Wilt + sterility mosaic	Asha (ICP 87119)
	Pod borer	ICPL 332
	Pod fly	KM 7
Mungbean	MYMV	Pant Mung1,2,3 & 4, Narendra Mung 1, ML 131, ML 267, ML 337, Samrat
	Powdery mildew	ML 131, Co G 4, Sabarmati, HUM 1, TARM 1, Pusa 9072, Pant Mung 1 & 3, Pusa 105
Urdbean	MYMV	Pant U 30, UG 218, Narendra Urd 1, NP 21, Uttara
	Powdery mildew	LBG 17, LBG 402, Co BG 5, WBU 108
Pea	Powdery mildew	Rachna, Pant P 5, HFP 4, DUP 2, DMR 7, DMR 11, Shikha, JG 885, HFP 8909, IPF 99-25
	Pod borer	Bonville, T 6113
Lentil	Rust	Pant L 406, Pant L 639, DPL 15, DPL 62, Pant L 77-12, Pant L 5
	Wilt	Pant 72-12

diverse sources in cases where resistance is controlled polygenically. ICRISAT used multiple crosses that involved up to eight parents for pyramiding resistance genes for ascochyta blight in chickpea. Some lines derived from these crosses showed enhanced level of resistance to ascochyta blight against all four races tested (PM Gaur, unpublished results).

The progress in breeding for resistance to insect-pests in pulse crops has been limited mainly due to unavailability of sources with high level of resistance and the unavailability of effective field screening techniques. Variations in insect pressure over time and locations and between and within plots in the same experiment affect the reliability of screening. This necessitates screening under controlled conditions. As the level of resistance identified are generally low, high emphasis should be placed on combining different mechanisms of resistance, e.g. antixenosis, antibiosis and tolerance.

Conventional method of resistance breeding will continue to be important in future as ever before. However, there is need to improve effectiveness of this approach by further refining screening methods for resistance to stresses, identifying new sources of resistance, understanding the mechanisms and genetics of resistance, and understanding the variability in the pathogen and its nature.

3.2 Mutation Breeding

The progress of any breeding programme, including resistance breeding, depends on genetic variability available in germplasm collections. This genetic variability has accumulated over the years due to spontaneous mutations, natural outcrossing and recombinations. In the absence of desired traits in the germplasm, the variability is often created through induced mutations. As a complementary method to conventional breeding approach, mutation breeding provides an opportunity to improve a cultivar for a particular trait without disrupting the genotype or to break desirable linkages among existing genes.

Both physical and chemical mutagens have been used to induce mutations in pulse crops. The most effective and widely used mutagen has been γ-rays followed by x-rays (Micke, 1988). The major focus in mutation breeding has been on induction of resistance to biotic and abiotic stresses. Mutants have been induced and some directly used as varieties and others as parents in hybridization programmes.

Mutants have been induced in chickpea for resistance to ascochyta blight (Kharkwal, 1983; Omar and Singh, 1995; Haq *et al.*, 1997, 1999), fusarium wilt (Bhatnagar *et al.*, 1979; Kharkwal 1983; Bravo, 1983), nematodes (Bhatnagar *et al.*, 1985), stunt (Bhatnagar *et al.*, 1979; Kharkwal 1983), leaf miner (Omar and Singh, 1995) and pod borer (Shaikh, 1983). The Indian Agricultural Research Institute (IARI),

New Delhi and Nuclear Institute of Agriculture and Biology (NIAB), Faisalabad, Pakistan had strong programmes on mutation breeding in pulse crops. Three chickpea varieties have been developed by IARI through mutation breeding. Pusa 408 (Ajay) and Pusa 413 (Atul) have moderate resistance to ascochyta blight, while Pusa 417 (Girnar) is also tolerant to wilt and root rots (Kharkwal *et al.*, 1988; Micke, 1988; Dua *et al.*, 2001). The mutation breeding programme of NIAB had a major focus on development of resistance to ascochyta blight, which is the most important disease of chickpea in Pakistan. The programme has led to development of four ascochyta resistant varieties, *i.e.*, CM 72, CM 88, CM 98 and CM 2000 (www.niab.org.pk).

Mutation breeding has been highly successful in development of diseases resistant varieties in mungbean. Mutants have been induced for resistance to MYMV (Shaikh, 1988; Malik, 1991; Gupta *et al.*, 1996), cercospora leaf spot (Shaikh, 1988; Song *et al.*, 1988; Malik, 1991; Wonpiyasatid *et al.*, 1998), and powdery mildew (Wonpiyasatid *et al.*, 1998). In India, the important varieties released include MUM 2 resistant to MYMV (Gupta *et al.*, 1996), TARM 1 and TARM 18 resistant to powdery mildew and MYMV (Pawar and Pandey, 2001), and BM 4 resistant to *Macrophomina* blight (Patil *et al.*, 1993) in India, and NIAB-M51 and NIAB-54 resistant to MYMV and cercospora leaf spot in Pakistan (Malik, 1991). In pigeonpea, the wilt resistant variety (TT 6) was developed through mutation breeding (Pawar and Pandey, 2001).

3.3 Exploitation of Wild Species

The wild species represents potential genetic diversity that may eventually be exploited for improvement of pulse crops for resistance to many biotic and abiotic stresses. However, limited progress has been made in introgression of genes from wild species to the cultigen. The major obstacle is getting hybrids between cultivated and many wild species. Greater efforts are needed in understanding barriers to interspecific hybridization and in identifying ways to overcome these barriers. Efforts are also needed to evaluate the accessions of wild species for sources of resistance in some crops.

Genus *Cicer* consists of 9 annual and 34 perennial species. The cultivated species *C. arietinum* L. can be crossed readily with *C. reticulatum* (the probable progenitor of chickpea) and *C. echinospermum* and thus these species constitute primary gene pool of chickpea. The secondary gene pool consists of *C. bijugum*, *C. pinnatifidum* and *C. judaicum*, all of which have been reported to cross with the cultigen and produce partially fertile hybrids. The remaining three annual wild species, *C. yamashitae*, *C. chorassanicum*, and *C. cuneatum*, constitute the tertiary gene pool as efforts have failed to produce hybrids of them with the cultigen or the hybrids obtained were albino or sterile (Croser *et al.*, 2003).

Sources of high level of resistance have been identified in the wild *Cicer* species for several biotic and abiotic stresses, including ascochyta blight, fusarium wilt, leaf miner, bruchids, cyst nematode, phytophthora root rot and cold. The resistance to cyst nematode and bruchids was identified only in the wild species. Resistance has been successfully transferred for cyst nematode from *C. reticulatum* (Malhotra *et al.*, 2002) and for phytophthora root rot from *C. echinospermum* (Knights *et al.*, 2002) to the cultigen.

Genus *Cajanus,* after merger of genus *Atylosia,* comprises 32 species. Among these, *C. cajan* is the only cultivated species. Sources of resistance have been identified for wilt, sterility mosaic, phytophthora blight (both for P2 and P3 isolates), alternaria blight, pod borer, podfly, and cyst nematode in *C. scarabaeoides*; resistance to sterility mosaic, phytophthora blight and alternaria blight in *C. sericeus;* resistance to sterility mosaic and alternaria blight in *C. albicans* and *C. lineatus*; resistance to phytophthora blight and alternaria blight in *C. platycarpus*; and resistance to podfly in *C. reticulatus*. All these species are in secondary gene pool, except *C. platycarpus, which* is in tertiary gene pool.

Genus *Lens* consists of two species *L. culinaris* and *L. nigricans* (Ladizinsky *et al.*, 1984). There are three subspecies in *L. culinaris* (*culinaris, orientalis* and *odemensis*), which constitute primary gene pool of the cultivated lentil (*Lens culinaris* ssp. *culinaris*). All these species can be intercrossed easily and the hybrids are fertile. There are two subspecies in *L. nigricans* (*nigricans* and *ervoides*), which constitute the secondary gene pool of lentil. Embryo rescue is needed to obtain hybrids between subspecies of *L. culinaris* with the species of *L. nigricans*. Sources of resistance to fusarium wilt have been identified in *L. culinaris* ssp. *orientalis*, *L. nigricans* ssp. *nigricans* and *L. nigricans* ssp. *ervoides*; and to ascochyta blight in *L. culinaris* ssp. *orientalis*, *L. culinaris* ssp *odemensis*, *L. nigricans* ssp. *nigricans*, and *L. nigricans* ssp. *ervoides* (Bayya *et al.*, 1994, 1995). One accession of *L. nigricans* ssp. *ervoides*, ILWC 138, has combined resistance to both the diseases.

Genus *Pisum* consists of two species *P. sativum* and *P. fulvum*. There are two subspecies in *P. sativum* (*sativum* and *elatius*), which intercross easily and produce fertile hybrids. The subspecies of *P. sativum* can also be crossed with *P. fulvum* with little difficulty but it is necessary to use *P. fulvum* as the pollen parent (Smartt, 1990). *P. fulvum* is a good source of resistance to ascochyta blight and pea weevil (Hardie *et al.*, 1995).

Two wild *Vigna* species, *Vigna radiata* ssp. *sublobata* and *V. glabrescens*, are particularly important in mungbean and urdbean. *Vigna radiata* ssp. *sublobata* is resistant to bruchids (*Callosobruchus maculatus* and *C. chinensis*) and this resistance

has been successfully transferred to *V. radiata* (Tomooka *et al.*, 1991; Kaga and Ishimoto, 1998). The tetraploid (2n=44) species *V. glabrescens* is also a good source of resistance to important biotic stresses. The interspecific crosses of *V. radiata* with *V. glabrescens* have also been possible through *in vitro* germination of immature embryos (Chen *et al.*, 1989). Interspecific hybridization between mungbean and urdbean gives a wide range of useful recombinants. Two varieties, HUM 1 and Pant Mung 4, have been developed from such crosses. A number of advanced breeding lines from such crosses are being evaluated at Indian Institute of Pulses Research, Kanpur (B.B. Singh, personal communication).

3.4 Marker Assisted Selection

The molecular markers offer great opportunity for facilitating and improving precision of selection for resistance in segregating generations. The marker-assisted selection (MAS) can substantially reduce the time and efforts needed to recover high level of resistance from the donor parent and at the same time recovery of the genomes of the adapted cultivar. MAS is particularly important in pyramiding of genes from different sources. The advances in technology on PCR-based markers have led to substantial reduction in time, expenditure and efforts needed for marker analysis. The high-through-put facilities, which include automation in DNA extraction and liquid handling and capillary electrophoresis, make it possible to analyse thousands of samples in few days. The requirement of tissues for DNA extraction is very less and it does not affect the plant growth.

Extensive efforts have been made in the recent past on identification of molecular markers for resistance genes in crop plants, including few important pulse crops. In chickpea, the major emphasis has so far been on identification of markers for resistance to fusarium wilt and ascochyta blight. Three genes for resistance to fusarium wilt (one of the three genes for resistance to race 1, one of the two genes for resistance to race 4 and the gene for resistance to race 5) are in one linkage group. The markers mapped close to these resistance genes include two RAPD, one ISSR (Winter *et al.*, 2000) and one RGA (Huttel *et al.*, 2002) markers. Markers have also been identified for major QTLs controlling resistance to ascochyta blight (Santra *et al.*, 2000; Tekeoglu *et al.*, 2002a; Taylor *et al.*, 2002).

Several studies have been conducted in pea on identification of markers for genes conferring resistance to important diseases. Markers have been identified for *en* gene conferring resistance to pea enation virus (Weeden and Provvidenti, 1988; Yu *et al.*, 1995), gene conferring resistance to bean yellow mosaic virus (Weeden *et al.*, 1984), *sbm-1* gene conferring resistance to P-1 pathotype and *sbm-4* gene conferring resistance to P-4 pathotype of seed-borne mosaic virus (Dhillon *et al.*, 1995), *er-1* gene conferring

resistance to powdery mildew (Timmerman *et al.*, 1994; Tiwari *et al.*, 1998; Rakshit *et al.*, 2001) and *Fw* gene conferring resistance to fusarium wilt race 1 (McClendon *et al.*, 2002). Molecular marker approach is being followed to track pea weevil resistance in crosses between *P. sativum* and *P. fulvum* at the University of Western Australia (Hardie *et al.*, 1995).

In lentil, markers have been identified for the single recessive gene controlling resistance to fusarium wilt (Eujayl *et al.*, 1998) and major genes controlling resistance to ascochyta blight (Ford *et al.*, 1999) and anthracnose (Tullu *et al.*, 2003). A study conducted on marker-assisted pyramiding of the two genes for resistance to anthracnose gave encouraging results (Tar'an *et al.*, 2003).

Molecular markers have been identified for a single dominant *Br* gene controlling resistance to bruchids in mungbean (Kaga and Ishimoto, 1998). Markers have also been identified for a major QTL accounting for 65% of the variation in resistance to powdery mildew (Chaitieng *et al.*, 2002).

In common bean, a recessive gene bc_3 is known to confer resistance against most strains of BCMV. A dominant inhibitor *I* gene is also needed for conferring resistance to temperature insensitive, necrosis inducing strains of BCMV. Molecular markers have been identified for both bc_3 (Johansen *et al.*, 1997) and *I* (Haley *et al.*, 1994, Melotta *et al.*, 1996) genes. Markers have also been identified for major genes controlling resistance to anthracnose (Young and Kelly, 1996) and rust (Miklas *et al.*, 1993).

3.5 Transgenics

The transgenic technology offers opportunity of introducing genes in a crop from any source including other organisms. It has greatly expanded the scope of plant breeding. It is now possible to develop varieties with enhanced level of resistance to a stress even if sources of resistance for that stress are not available in the cultivated and its cross compatible wild species.

The efforts on development of transgenics in chickpea have mainly focused on introduction of genes for resistance to *Helicoverpa* pod borer. Kar *et al.* (1997) introduced *Cry1Ac* gene of *Bacillus thuringiensis* (Bt) using particle bombardment method. Insect feeding assay indicated inhibition of development of larvae. An efficient *in vitro* regeneration system has been developed at ICRISAT (Jayanand *et al.*, 2003) and transgenics have been developed for *Cry1Ab* and trypsin inhibitor (*SbTI*) genes. The plants are at the T_2 stage and bioassays are in progress. Efforts are also being made to introduce genes that produce antifungal protein such as polygalacturonase inhibitory protein (PGIP), chitinases and glucanases for development of transgenics resistance to fungal diseases (Kiran K. Sharma, Pers. Communication).

A highly efficient *in vitro* regeneration procedure is available for pigeonpea. Both *Agrobacterium* and particle bombardment methods have been successfully used to develop transgenics. Lawrence and Koundal (2000) successfully introduced cowpea protease inhibitor (*CpPI*) gene in pigeonpea variety Pusa 855 through *Agrobacterium*-mediated transformation and confirmed chromosomal integration of the gene in nine-week *in vitro* cultured plants. Pigeonpea transgenics have been developed at ICRISAT for resistance to *Helicoverpa* pod borer by introducing *BtCry1Ab* and *SbTI* genes. The T_6 plants are being evaluated under contained field trial at the ICRISAT campus in Patancheru during 2003.

Excellent progress has been made in development of transgenics in pea. Transgenics expressing α-amylase inhibitor (αAI) gene of common bean showed resistance to bruchids (Schroeder *et al.*, 1995). A gene encoding a multi-domain proteinase inhibitor precursor from *Nicotiana alata* (NaPI) was introduced to obtain resistance to *Helicoverpa* pod borer (Charity *et al.*, 1999). Enhanced resistance to pea enation mosaic virus was obtained by introducing its coat protein gene (PEMV-CP) (Chowrira *et al.*, 1998).

It has been difficult to get efficient *in vitro* regeneration from lentil, mungbean, urdbean and common bean. However, transgenics have been produced in lentil for herbicide resistance (Gulati *et al.*, 2002) and in common bean for herbicide resistance and bean golden mosaic virus resistance (Russell *et al.*, 1993; Aragao *et al.*, 1998) using particle bombardment method. The regeneration system is available for *P. acutifolius*. Thus, this species can be used as bridge species to introgress transgenes into the common bean (Dillen *et al.*, 2000).

4. Prospects

The conventional approaches of resistance breeding have provided several improved varieties of pulse crops with resistance to important biotic and abiotic stresses. There is no substitute for these approaches and these will continue to be mainstay in the future as well. However, efforts are needed on improving effectiveness of these approaches by further refining screening methods for resistance to stresses and identifying new sources of resistance genes in the cultivated and wild species. There is a need to use diverse sources of resistance in breeding programmes and develop cultivars with resistance to multiple stress factors.

The other approaches can supplement the conventional methods. Mutagenesis has potential of creating desired variability including resistance to stresses and thus should find place in resistance breeding. Wild species are valuable sources of resistance genes and concerted efforts are needed on their exploitation. The marker-assisted

selection has greater role to play in resistance breeding particularly when the direct assessment of the phenotype is difficult and a large number of resistance genes are to be combined. The transgenic technology has already proven its worth in many crops including some pulses and its benefit is being extended to other pulse crops. Finally, it can be concluded that a support of biotechnological approaches to conventional breeding methods would lead to rapid progress in development of improved cultivars of pulses with resistance to biotic and abiotic stresses.

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