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REVIEW

Fusarium wilt of chickpea: physiological specialization, genetics of resistance and resistance gene tagging

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Abstract Chickpea wilt caused by *Fusarium oxysporum* f. sp. *ciceris* is one of the major yield limiting factors in chickpea. The disease causes 10–90% yield losses annually in chickpea. Eight physiological races of the pathogen (0, 1A, 1B/C, 2, 3, 4, 5 and 6) are reported so far whereas additional races are suspected from India. The distribution pattern of these races in different parts of the world indicates regional specificity for their occurrence leading to the perception that *F. oxysporum* f. sp. *ciceris* evolved independently in different regions. Pathogen isolates also exhibit differences in disease symptoms. Races 0 and 1B/C cause yellowing syndrome whereas 1A, 2, 3, 4, 5 and 6 lead to wilting syndrome. Genetics of resistance to two races (1B/C and 6) is yet to be determined, however, for other races resistance is governed either by monogenes or oligogenes. The individual genes of oligogenic resistance mechanism delay onset of disease symptoms, a phenomenon called as late wilting. Slow wilting, i.e., slow development of disease after onset of disease symptoms also occurs in reaction to pathogen; however, its genetics are not known. Mapping of wilt resistance genes in chickpea is difficult because of minimal polymorphism; however, it has been facilitated to great extent by the development of sequence tagged microsatellite site (STMS) markers that have revealed significant interspecific and intraspecific polymorphism. Markers linked to six genes governing resistance to six races (0, 1A, 2, 3, 4 and 5) of the pathogen have been identified and their position on chickpea linkage maps elucidated. These genes lie in two separate clusters on two different chickpea linkage groups. While the gene for resistance to race 0 is situated on LG 5 of Winter et al. (Theoretical and Applied Genetics 101:1155–1163, 2000) those governing resistance to races 1A, 2, 3, 4 and 5 spanned a region of 8.2 cM on LG 2. The cluster of five resistance genes was further subdivided into two sub clusters of 2.8 cM and 2.0 cM, respectively. Map-based cloning can be used to isolate the six genes mapped so far; however, the region containing these genes needs additional markers to facilitate their isolation. Cloning of wilt resistance genes is desirable to study their evolution, mechanisms of resistance and their exploitation in wilt resistance breeding and wilt management.

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Introduction

Chickpea (*Cicer arietinum* L.) is an ancient pulse crop that was first grown in Turkey about 7,000 B.C. It is believed to have been domesticated in Turkey from *C. reticulatum* Ladizinsky, a closely related wild species. After domestication in the Middle East, the crop spread throughout the Middle East, the Mediterranean region, India, and Ethiopia (Ladizinsky 1975; van der Maesen 1987). Its introduction in Mexico, Argentina, Chile, Peru, Australia and the US is a recent event (Duke 1981). Chickpea is most widely grown in South Asia and the Mediterranean region (Saxena 1990; Singh and Ocampo 1997; FAO 2003). It ranks third in the world among pulse crops after peas and beans with an area of 10,374 thousand ha with total production of 7,123 thousand MT and an average yield of 687 kg ha^{-1} (FAO 2003).

Despite economic importance and strong national and international breeding programmes, average yields of chickpea have not improved considerably over the years. Annual growth rate of chickpea production has been low (0.007%) during the last decade (1993–2003) and average yields have been almost static (FAO 1993, 2003). Average chickpea grain yield during 1993 was 671 kg ha^{-1} whereas it was 687 kg ha^{-1} during 2003. One of the major constraints in realization of full yield potential of chickpea is wilt caused by a Deuteromycetes fungal pathogen *Fusarium oxysporum* Schlechtend.: *Fr.* f. sp. *ciceris* (Padwick) Matuo & K. Sato. The pathogen penetrates the vascular bundles of roots of chickpea plants and stops or reduces water uptake to the foliage. The infected plants ultimately wilt and die. The disease is highly destructive and worldwide in occurrence (Kraft et al. 1994). It has been reported from almost all chickpea growing areas of the world including the Indian subcontinent, Iran, Peru, Syria, Ethiopia, Mexico, Spain, Tunisia, Turkey and US (Westerlund et al. 1974; Nene et al. 1989; Halila and Strange 1996). The disease is capable of causing 100% yield loss. Annual yield losses to wilt have been estimated at 10%–90% (Jimenez-Diaz et al. 1989; Singh and Reddy 1991). Persistence of the pathogen in soil and its capacity to survive there for years even in the absence of host (Haware et al. 1996) renders its control difficult. Soil applications of fungicides are costly and lead to indiscriminate killing of beneficial soil microflora. The disease to some extent can be managed by use of biocontrol agents which provide eco-friendly control of the disease (Hervas et al. 1997, 1998; Landa et al. 2001). Non-pathogenic *Fusarium oxysporum*, *Bacillus* species and *Pseudomonas flourescens* were identified suitable for biocontrol of wilt (Hervas et al. 1997; Landa et al. 2001, 2004). Efficacy of wilt management was improved when biocontrol agents were combined with cultural practices such as sowing dates (Landa et al. 2004). More economic, effective and eco-friendly method of disease management is, however, by race-specific vertical resistance genes of the host which are available in the cultigen *C. arietinum* (Jimenez-Diaz et al. 1993; Jalali and Chand 1992; Sharma et al. 2005).

F. oxysporum f. sp. *ciceris* has eight distinct physiological races (Haware and Nene 1982; Jimenez-Diaz et al. 1993; Kelly et al. 1994). Evaluation of host lines for resistance to different races is laborious, time consuming and costly as the lines must be inoculated with individual pathogen races. The infection process is influenced by environment especially the temperature and inoculum load (Bhatti and Kraft 1992; Navas-Cortés et al. 1998; Navas-Cortés et al. 2000; Landa et al. 2001). A temperature around 25°C and inoculum load of 10^4–10^5 micro- or macro-conidia is optimum for disease development. Though, several race-specific sources of resistance have been identified (Sharma et al. 2005), and some of them exploited to develop wilt resistant chickpea lines (Singh and Jimenez-Diaz 1996), the chances of breakdown of resistance and the appearance of new pathogenic races remain. Combining resistance to more than one race in a commercial cultivar i.e., pyramiding of resistance genes is expected to provide durable resistance against the disease, as the pathogen has to mutate several avirulence genes to overcome the resistance governed by several major genes. The advent of molecular markers linked to resistance genes in diseases such as rice bacterial blight and rice blast has facilitated their pyramiding and, hence, led to development of multirace resistant lines/cultivars.

Physiological specialization in *F. oxysporum* f. sp. *ciceris*

Haware and Nene (1982) reported existence of four physiological races (1, 2, 3 and 4) of *F. oxysporum* f. sp. *ciceris* in India using 10 chickpea lines as differentials. Two additional races (0 and 5) were later identified from Spain (Cabrera de la Colina et al. 1985) and Tunisia (Halila and Strange 1996) whereas another (race 6) was reported from California, USA (Phillips 1988). Race 1 was subsequently divided into two races named as race 1A (from India) and race 1B/C (from Spain) based on variation in reaction on differential host lines (Trapero-Casas and Jimenez-Diaz 1985; Jimenez Diaz et al. 1993). Race 1B/C was also found in USA (California), Syria, Turkey and Tunisia. Thus, a total of eight physiological races of the pathogen have been reported worldwide (Table 1). More recently, races 0 and 6 have been reported in India (Rahman et al. 1998). The same study suggested the occurrence of two additional races in India and was supported by another study by the same author (Rahman et al. 2000). Since plant age, inoculum load and environmental conditions influence the final disease incidence (Bhatti and Kraft 1992; Navas-Cortes et al. 1998; Landa et al. 2001), the occurrence of the two additional races needs to be validated preferably using the refined wilt screening technique (Sharma et al. 2004b). If the existence of the new races is confirmed, the total number of races will increase to ten from the present eight.

The geographical distribution of races shows regional specificity for their occurrence in different regions of the world. Among eight races, 0, 1B/C, 5 and 6 are primarily found in the Mediterranean region and the USA (Phillips 1988; Jimenez Diaz et al. 1989, 1993; Halila and Strange 1996; Jimenez-Gasco et al. 2001), whereas races 1A, 2, 3 and 4 are restricted to the Indian subcontinent (Haware and Nene 1982). Apart from region-specificities, the eight races can also be divided into two groups based on symptomatology of infected plants i.e., yellowing syndrome and wilting syndrome (Trapero-Casas and Jimenez-Diaz 1985). Of eight races, six (1A, 2, 3, 4, 5 and 6) cause wilting syndrome and are economically more important than races 0 and 1B/C that cause yellowing syndrome (Haware and Nene 1982; Jimenez-Diaz et al. 1993; Kelly et al. 1994). Plants infected with races causing

Table 1 Genetics of resistance to different races of the chickpea wilt pathogen *Fusarium oxysproum* f. sp. *ciceris*

Fusarium race	Name of the resistance gene	Effect of resistance gene on wilting	Reference
0	$foc\text{-}0_1/Foc\text{-}0_1$, $foc\text{-}0_2/Foc\text{-}0_2$[a]	Complete resistance[b]	Rubio et al. (2003)
1A	h_1 (syn *foc-1*),	Late wilting	Singh et al. (1987a, b)
	h_2	Late wilting	
	H_3	Late wilting	
1B/C	–		
2	*foc-2*[c]	Complete resistance	Sharma et al. (2005)
3	*foc-3*/*Foc-3*[a]	Complete resistance	Sharma et al. (2004b, 2005)
4	*foc-4*	Complete resistance	Tullu et al. (1998), Sharma et al. (2005)
	Two recessive genes	Complete resistance[b]	Tullu et al. (1999)
5	(*foc-5*/*Foc-5*)[a]	Complete resistance	Tekeoglu et al. (2000), Sharma et al. (2005)
6	–		

[a] Dominant/recessive nature not known

[b] Effect of individual genes in resistance not known

[c] Kumar (1998) found it to be governed by three genes, *a*, *b* and *C*. Each of the three genes led to late wilting whereas the first two genes conferred complete resistance (see text for details)

(–), Genetics of resistance not known

wilting syndrome wilt within three to four weeks of inoculation with no visible yellowing of leaves. On the other hand, infection with races 0 and 1B/C leads to progressive foliar yellowing of plant leaves coupled with vascular discoloration. The wilting of infected plants eventually starts six to seven weeks after inoculation. Wilting and yellowing syndromes have been so far considered race-specific, however, evidence is emerging to indicate that both types of symptoms can be caused by a single race. Race 0 which is considered to cause yellowing syndrome, led to the wilting of plants of *C. reticulatum* (PI489777) within 30 days of inoculation with no evident foliar yellowing (Tekeoglu et al. 2000).

Despite the occurrence of several races, overall genetic make up of the fungus all over the world is narrow. All *F. oxysporum* f. sp. *ciceris* isolates were found to belong to a single vegetative compatibility group (Nogales-Moncada 1997). DNA fingerprinting of races with repetitive sequences also suggested monophyletic lineage (Jimenez-Gasco et al. 2004a, b). Despite monophyletic lineage, geographically isolated populations of the fungus displayed genetic and pathological diversity. The Iranian isolates comprised at least three vegetative compatibility groups (VCGs) (Zamani et al. 2004) whereas the four Indian races were phylogenetically distinct from each other (Sivaramakrishnan et al. 2002; Chakrabarti et al. 2001; Barve et al. 2001). Indian populations of pathogen were also genetically as well as pathologically distinct from those in other countries as is evident from DNA fingerprinting studies (Barve et al. 2001) and confinement of races 1A, 2, 3 and 4 (wilting pathotypes) to the India and 0 and 1B/C (yellowing pathotypes) to the Mediterranean region and California. Thus, atleast two different populations of the pathogen exist worldwide, one native to India and another to other parts of the world. Similar to *F. oxysporum* f. sp. *ciceris*, *F. oxysporum* f. sp. *malvacearum*, the wilt pathogen of cotton, also have two genetically distinct populations, one confined to Australia and the second to remaining parts of the world. In fact, the Australian isolates of *F. oxysporum* f. sp. *malvacearum* have evolved from native non-pathogenic *F. oxysporum* and their evolution was independent from populations in other parts of the world (Davis et al. 1996). Unlike *F. oxysporum* f. sp. *malvacearum* which evolved from two different populations, the populations of *F. oxysporum* f. sp. *ciceris* have evolved from a common ancestor or a single individual (Jimenez-Gasco et al. 2002). The propagules of *F. oxysporum* f. sp. *ciceris* from the founder population then disseminated to different geographical areas possibly through seed where these diverged independently to races by stepwise acquisition of virulences (Jimenez-Gasco et al. 2004a, b). The evolution of geographically distinct virulences appears to be correlated to cultivation of chickpea germplasm lines in these regions. Chickpeas are of two types ‘desi’ and ‘kabuli’. Between these two, ‘desi’ genotypes are grown mainly in the India whereas ‘kabuli’ in the Mediterranean region and California. Resistance to wilt occurs mostly in ‘desi’ genotypes (Haware et al. 1980). Interestingly, races 1A, 2, 3 and 4 which inhabit the India are also the most virulent ones whereas those from the Mediterranean region or the USA are less virulent (Haware and Nene 1982; Jimenez-Diaz et al. 1993; Halila and Strange 1996). Evidently, there exists a correlation between evolution to races and cultivation of chickpea lines.

Identification of pathogen races is based on disease reaction of differential lines. Several different sets of differentials have been used to identify the races. The number of lines in different sets has ranged from as few as eight to as many as 22 (Haware and Nene 1982; Jimenez-Diaz et al. 1989; El-Hadi 1993; Tullu 1996). Many lines in these sets show intermediate reaction to different races and lacked clear cut disease phenotype to differentiate those. Identification of races 2 and 3 is difficult as none of the sets could differentiate precisely between the two (Sharma et al. 2004a). Since, resistance to wilt is vertical in nature, an ideal set should have lines with either no disease or 100% disease. The gap was filled by the development of a set of eight differential lines (Table 2) having vertical resistance genes (Sharma et al. 2005). The set could differentiate six races (0, 1A, 2, 3, 4 and 5) with clear cut disease phenotypes based on no or 100% wilt. Among eight differentials, four were germplasm lines and four recombinant inbred lines (RILs). The set does not have lines for identification of races 1B/C and 6. It would be

Table 2 List of chickpea differentials and their reaction to five races of *Fusarium oxysporum* f. sp. *ciceris*

Germplasm accession	Differential line	Race 1A[a]	Race 2	Race 3	Race 4	Race 5
W6-24867	JG-62[b]	S (100.0)	S (94.3)	S (100.0)	S (100.0)	S (100.0)
W6-24868	P-2245[b]	S (100.0)	S (100.0)	S (100.0)	S (100.0)	S (100.0)
W6-24869	SANFORD	R (0)	S (100.0)	S (100.0)	S (100.0)	S (95.0)
W6-24870	CRIL-1-53	S (100.0)	R (0)	R (0)	R (0)	R (0)
W6-24871	CRIL-1-94	R (0)	S (100.0)	R (0)	I (36.4)	I (30.0)
W6-24872	CRIL-1-17	R (0)	R (0)	R (0)	S (100.0)	R (0)
W6-24874	CRIL-1-36	I (33.3)	S (100.0)	S (100.0)	S (100.0)	R (0)
W6-24876	WR-315	R (0)	R (0)	R (0)	R (0)	R (0)

[a] S = Susceptible (90–100% wilt), R = Resistant (0–10% wilt), I = Intermediate (11–89% wilt), disease incidence (%) in parentheses

[b] JG62 and P-2245 are resistant and susceptible, respectively, to race 0 (Tullu 1996; Rubio et al. 2003)

(Courtesy Sharma et al. 2005)

desirable to add lines having vertical resistance to races 1B/C and 6 in this set to facilitate identification of all races of the pathogen. Race identification based on differentials is time consuming and can be erroneous if temperature is not conducive for wilt development. Alternatively, DNA-based diagnostics assays which are fast, do not need screening of differential lines and are not influenced by environment, are being developed for the pathogen and its races (Kelly et al. 1994; Jimenez-Gasco et al. 2001; Jimenez-Gasco and Jimenez-Diaz 2003). Random amplified polymorphic DNA (RAPD) markers have been used successfully to detect the pathogen in soil (Gracia-Pedrajas et al. 1999) and distinguish between yellowing and wilting pathotypes either from isolated cultures (Kelly et al. 1994) or from infected chickpea plants without fungal isolation (Kelly et al. 1998). The technique was further refined to develop RAPD-based detection system for races 0, 1B/C, 5 and 6 (Jimenez-Gasco et al. 2001). RAPD markers are less robust and the results may sometimes be ambiguous. To facilitate precise identification of races 0, 1A, 5 and 6, more robust markers called as sequence characterized amplified regions (SCARs) have also been developed (Jimenez-Gasco and Jimenez-Diaz 2003). The utility of these assays to replace the traditional method based on host reaction for identification of the pathogen and its races is still to be confirmed. However, these assays might need further refinements before these could be used routinely by the pathologists or breeders.

Genetics of chickpea wilt resistance

Early studies on genetics of wilt resistance were restricted to race 1 where it was shown to be inherited by a recessive gene (Ayyar and Iyer 1936; Kumar and Haware 1982; Sindhu et al. 1983). With the identification of phenomenon of late wilting in some genotypes susceptible to race 1 (Upadhyaya et al. 1983a), the focus was shifted to genetics of late wilting. The late wilting was found to be a mongenic trait and was controlled by three independent genes named as h_1, h_2 and H_3, each of which delayed onset of disease symptoms (Upadhyaya et al. 1983b; Singh et al. 1987a, b). Combination of any of the two late wilting genes (h_1h_1 or h_2h_2 or h_1H_3 or h_2H_3) was required for complete resistance to race 1. (Upadhyaya et al. 1983b; Singh et al. 1987a, b). Race 1 used in these studies was from India, hence, the race 1 described here should be considered as race 1A.

Similar to race 1A, resistance to race 2 was initially found to be conferred by a single recessive gene (Pathak et al. 1975), however, later studies revealed involvement of two (Gumber et al. 1995) or three genes (Kumar 1998). Phenomenon of late wilting was also reported after inoculation with race 2 (Gumber et al. 1995). Of the three genes, *a* or *b* in homozygous recessive form or C in dominant form conferred late wilting (Kumar 1998). Complete resistance was expressed when both *aa* and *bb* were present. Interestingly, the third gene whether it is homozygous recessive or homozygous dominant or heterozygous, did not influence the expression of complete resistance by other two genes or imparted any role in complete resistance. The F_3 data of Kumar (1998) and that of F_2 of Gumber et al. (1995) also did not fit well to the three and two gene theories, respectively. This possibly points towards the involvement of fewer/more genes than three in race 2 resistance. Using the F_2s and RILs derived from the same parents that were used by Kumar (1998) to show involvement of three genes, Sharma et al. (2005) demonstrated that resistance to race 2 was governed by a single recessive gene. Differences in results between the two studies can be attributed to the evaluation techniques used. Kumar (1998) and Gumber et al. (1995) evaluated plants in sick plots/pots where time of inoculation of all plants could not be uniform. Moreover, inoculation of all plants cannot be ensured. Since, appearance of wilt symptoms depends upon the time taken from infection of root surface cells to accumulation of sufficient amount of propagules in the vascular bundles, it is very likely that plants infected early will wilt earlier than those infected later. Obviously, differences in the time taken to wilt under such circumstances cannot be the reflection of resistance of the host. On the other hand, Sharma et al. (2005) ensured inoculation of each plant by dipping their roots (injured by cutting the one fifth lower portions to facilitate uniform pathogen inoculation and penetration) in pathogen inoculum containing a constant number of spores (1×10^6 per ml). The vegetative stage at the time of inoculation was also uniform. The same study also questioned the phenomenon of late wilting for race 2 as susceptible parent C-104, F_2s and RILs took almost same time to wilt. The study, however, demonstrated the existence of another kind of race specific resistance which was termed as slow wilting (see chapter below).

Genetics of resistance to other races of the pathogen is comparatively less studied. The resistance to race 3 was found to be monogenic (Sharma et al. 2004b, 2005), however, its dominant or recessive nature is unknown as a RIL population was used. Resistance to race 4 was monogenic recessive in some lines (Tullu et al. 1998; Sharma et al. 2005) whereas it was digenic recessive in Surutato-77 (Tullu et al. 1999). Similar to races 1 and 2, the phenomenon of late wilting was also detected for race 4. There are only a couple of studies on the inheritance of resistance to race 5 which showed it to be governed by a single gene (Tekeoglu et al. 2000; Sharma et al. 2005). However, it is yet to be ascertained whether the resistance gene(s) in two lines are the same or different. Resistance to race 0 was found to be monogenic (Tekeoglu et al. 2000) as well as digenic which may be either dominant or recessive (Rubio et al. 2003). Names of genes conferring resistance to different races and their effect on wilting have been presented in Table 1.

Slow wilting

Apart from vertical form of resistance, slow wilting resistance in chickpea after inoculation with *F. oxysporum* f. sp. *ciceris* has also been observed (Sharma et al. 2005). While studying reaction of chickpea germplasm lines to different races of the wilt pathogen, one line FLIP84-92C(3) showed slow disease progress after appearance of the first symptoms after inoculation with race 2. Comparison of data of this line with that of C-104 (susceptible) indicated that while all plants of C-104 wilted within 3 weeks, only 15.0% of FLIP84-92C(3) wilted. Even after 8 weeks of inoculation, 13.4% plants of FLIP84-92C(3) survived. Slow wilting of FLIP84-92C(3) was race-specific and was observed only for race 2. In addition to FLIP84-92C(3), slow wilting was also observed in some RILs of chickpea obtained from the cross of WR-315 (resistant to all races) and C-104 (susceptible to races 1A, 2, 3, 4 and 5) where two RILs were slow wilting for race 2 and three for race 3. Slow wilting, thus, is a race-specific phenomenon and differs from late wilting in three aspects: latent period, disease progress rate, and final disease severity. In comparison to slow wilting, late wilting refers to susceptible lines showing a prolonged latent period. Late wilting lines eventually show 100% wilt.

The phenomenon of slow wilting in chickpea is similar to that of slow mildewing and slow rusting in crops such as pea and wheat. The genetics of slow wilting resistance in chickpea have not been determined, however, it might involve host genes other than vertical resistance ones. These genes appear to be minor ones which additively slow the development of wilt as is evident from identification of slow wilting lines from cross of resistant and susceptible parents. Slow rusting and slow mildewing resistances in other crops are usually governed by polygenes and quantitative trait loci (QTLs) which have additive effect (Singh et al. 2000, 2005; Xu et al. 2005a, b). Though not very frequently, these two types of resistances are also controlled by single genes (Lewellen and Schrandt 2001; Singh and Huerta-Espino 2003). Among such genes, role of wheat genes, *Lr34* and *Lr46* in slow rusting after infection with *Puccinia triticina* (syn. *P. recondita*) has been extensively studied (Singh et al. 1998; Singh and Huerta-Espino 2003; William et al. 2003).

Molecular markers linked to Fusarium wilt resistance genes

Development of molecular markers in chickpea has been relatively slow due to minimal polymorphism in its genome (Kazan and Muehlbauer 1991; Ahmad and Slinkard 1992; Labdi et al. 1996; Mayer et al. 1997). Isozymes, restriction fragment length polymorphism (RFLP) and RAPD markers used initially to map resistance genes yielded little success (Gaur and Slinkard 1990; Kazan and Muehlbauer 1991; Kazan et al. 1993; Mayer et al. 1997). Inter-simple-sequence-repeat (ISSR), DNA amplification fingerprinting (DAF) and resistance gene analogue (RGA) markers developed later for chickpea revealed more polymorphism compared to isozymes, RAPDs and RFLPs. The major breakthrough, in the development of polymorphic markers was, however, the identification of sequence-tagged microsatellite site (STMS) markers (Huttel et al. 1999; Winter et al. 1999; Niroj et al. 2003; Lichtenzveig et al. 2005). Apart from high robustness and PCR-based nature, these markers were highly polymorphic even for *C. arietinum* where other markers displayed little polymorphism (Udupa et al. 1999). Owing to highly polymorphic nature, these markers were/are being used extensively in mapping studies in chickpea (Cho et al. 2002; Rajesh et al. 2002; Galvez et al. 2003; Sharma et al. 2004b; Sharma and Muehlbauer 2005).

The first wilt resistance gene to be tagged in chickpea was H_1 (syn. *foc-1*, Mayer et al. 1997). The gene was 7.0 cM from RAPD markers CS-27_{700} and UBC-170_{550} and a Allele Specific Associated Primer (ASAP) marker. Subsequently, markers linked closely to *foc-1* (Sharma et al. 2004b; Sharma and Muehlbauer 2005), *foc-*0_1 (Rubio et al. 2003; Cobos et al. 2005), *foc-2* (Sharma and Muehlbauer 2005), *foc-3* (Sharma et al. 2004b; Sharma and Muehlbauer 2005), *foc-4* (Ratnaparkhe et al. 1998a, b; Tullu et al. 1998; Tullu et al. 1999; Tekeoglu et al. 2000; Winter et al. 2000; Benko-Iseppon et al. 2003; Sharma et al. 2004b; Sharma and Muehlbauer 2005), the second resistance gene for race 4 (Tullu et al. 1999) and *foc*-5 (Ratnaparkhe et al. 1998b; Tekeoglu et al. 2000; Winter et al. 2000; Benko-Iseppon et al. 2003; Sharma and Muehlbauer 2005) were identified. The markers linked to different wilt resistance genes and map distances have been summarized in Table 3.

Comparison of different studies (Tekeoglu et al. 2000; Ratnaparkhe et al. 1998a, b; Winter et al. 2000; Huttel et al. 2002; Benko-Iseppon et al. 2003; Pfaff and Kahl 2003; Sharma et al. 2004b), indicated that four genes (*foc-1*, *foc-3*, *foc-4* and *foc-5*) should be in the same linkage group. Based on marker data of Benko-Iseppon et al. (2003), Huttel et al. (2002) and other studies, Millan et al. (2006) also proposed linkage of *foc-1*, *foc-3*, *foc-4* and *foc-5* (Fig. 1a). Conclusive evidence on clustering of five resistance genes (*foc-1*, *foc-2*, *foc-3*, *foc-4* and *foc-5*) was presented later by Sharma and Muehlbauer (2005, Fig. 1b) who mapped the genes using an intraspecific RIL population derived from the cross of WR-315 (resistant to races 1A, 2, 3, 4 and 5) and C-104 (susceptible). The cluster of five genes spanned 8.2 cM and was situated on LG 2 of chickpea linkage map of Winter et al. (2000). Considering 1 cM is estimated to be 360 kb (Winter et al. 2000), the resistance gene cluster was 2.952 Mb. Among the five genes, *foc-1* and *foc-5* were 2.0 cM apart whereas *foc-5* on the other side was flanked by *foc-3* at a distance of 3.4 cM. The *foc-1* and *foc-3* were estimated to be separated by 5.4 cM. The *foc-3* and *foc-2* were at a distance of 1.0 cM whereas *foc-2* and *foc-4* were at 1.8 cM. Two genes (*foc-1* and *foc-4*) situated on both ends of the cluster were

Table 3 DNA-based markers linked to Fusarium wilt resistance genes in chickpea (Mayer et al. 1997; Ratnaparkhe et al. 1998a, b; Tullu et al. 1998, 1999; Tekeoglu et al. 2000; Winter et al. 2000; Rubio et al. 2003; Benko-Iseppon et al. 2003; Sharma et al. 2004b; Cobos et al. 2005; Sharma and Muehlbauer 2005)

Wilt resistance gene	Markers linked to the resistance gene[a,b]					
	STMS	RAPD	SCAR/STS	ISSR	AFLP	DAF
Foc-0$_1$/foc-0$_1$	TR59 (2.0)	$OPJ20_{600}$ (3.0)	–	–	–	–
foc-1 (syn. *h$_1$*)	TA59 (4.4), TA96 (4.9), TA27 (4.9)	$CS27_{700}$ (7.2), $UBC\text{-}170_{550}$ (7.0)	CS27A (4.9)	–	–	–
foc-2	TA96 (1.5), TA27 (1.5), TR19 (4.9)	–	CS27A (1.5)	–	–	–
Foc-3/foc-3	TA96 (0.5), TA27 (0.5), TA59 (0.5)	–	CS27A (0.5)	–	–	–
foc-4	TA59 (3.8), TA96 (3.3), TA27 (3.3), TR19 (3.1)	$CS27_{700}$ (15.2), $UBC\text{-}170_{550}$ (9.0)	CS27A (3.3)	$UBC\text{-}825_{1200}$ (5.0)	EAAMCTA12 (5.9)	R2609-1 (2.0), OPU17-1 (4.1)
Foc-5/foc-5	TA27 (2.9), TA59 (2.4), TA96 (2.9)	$CS27_{700}$ (9.2), $UBC\text{-}170_{550}$ (2.5)	CS27A (2.9)	–	ECAMCTA07 (6.4)	$OP\text{-}M20\text{-}2_{1045}$ (12.0), $OP\text{-}M20\text{-}3_{1103}$ (12.0)

[a] STMS: sequence-tagged microsatellite site, RAPD: randomly amplified polymorphic DNA, SCAR: sequence-characterized amplified region, STS: sequence-tagged site, ISSR: inter-simple-sequence-repeat, AFLP: amplified fragment length polymorphism, DAF: DNA amplification fingerprinting

[b] Genetic distance (cM) of the marker from the gene in parentheses

8.2 cM apart. The R gene cluster could further be subdivided into two sub-clusters. Genes *foc-4*, *foc-2* and *foc-3* formed one sub-cluster of 2.8 cM whereas *foc-5* and *foc-1* were in another sub-cluster of 2.0 cM. The two sub-clusters were separated by 3.4 cM. Compared to Winter et al. (2000), Benko-Iseppon et al. (2003) and Millan et al. (2006), where genetic maps were constructed using interspecific mapping populations, the order of genes was different for Sharma and Muehlbauer (2005). Skewed ratios or segregation distortion has frequently been reported when interspecific mapping populations were used (Ratnaparkhe et al. 1998a; Winter et al. 1999, 2000; Benko-Iseppon et al. 2003). Segregation distortion among interspecific crosses might explain the differences in gene order and genetic distances among wilt resistance genes and markers obtained using interspecific and intraspecific mapping populations of chickpea. The gene order as well as map distances might be more accurate for Sharma and Muehlbauer (2005) as a single source of resistance to five genes was used and the mapping population was from an intraspecific cross.

The gene, *Foc-0$_1$/foc-0$_1$* was not linked to cluster of five genes. The gene was located on LG3 (Cobos et al. 2005, Fig. 1c), the linkage group that corresponds to LG 5 of Winter et al. (2000). Thus, there are two distinct clusters of wilt R genes, one situated on LG 2 [chromosome F (or G)] and comprised of genes effective against wilting pathotypes and another on LG 5 [chromosome C (or D)] having gene governing resistance to yellowing pathotype. The genes for resistance to races 1B/C and 6 have not yet been mapped. Between these two, 1B/C causes yellowing syndrome and 6 the wilting syndrome. It would be interesting to find if gene for resistance to 1B/C is linked to *foc-0$_1$* and that effective against race 6 to the *foc-1*, *foc-2*, *foc-3*, *foc-4* and *foc-5* gene cluster. Apart from wilt resistance genes, LG 2 also harbours two QTLs (ar1 and ar2a) for resistance to *Ascochyta rabiei*, some genes with putative function in plant-defense against diseases (Udupa and Baum 2003; Pfaff and Kahl 2003) and a few RGAs. Like resistance genes, defense-response genes also lie in clusters of gene families in other crops (Ruiz et al. 2005). The possible clustering of such genes in

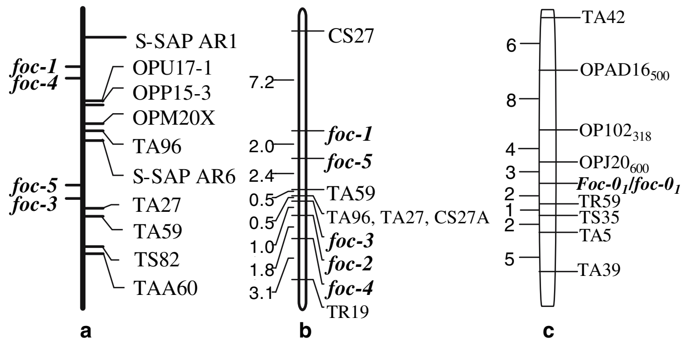

Fig. 1 Linkage map of six wilt resistance genes on chickpea genome. The (**a**) depicts position of four wilt resistance genes (***foc-1, foc-3, foc-4*** and ***foc-5***) on an interspecific linkage map (Millan et al. 2006), (**b**) shows position of five wilt resistance genes (***foc-1, foc-2, foc-3, foc-4*** and ***foc-5***) on an intraspecific linkage map (Figure Sharma and Muehlbauer 2005) and (**c**) shows location of race 0 resistance gene (***Foc-01/foc-01***) on linkage group LG3 (Figure Cobos et al. 2005). The map shown in Fig. 1a is derived from many independent studies. See the differences in the order of resistance genes between inter- (Fig. 1a) and intra-specific (Fig. 1b) maps. Figs. a and b corresponds to LG 2 of Winter et al. (2000) whereas LG3 of Cobos et al. (2005) corresponds to LG 5 of Winter et al. (2000). In Fig. 1a genes are on the left and marker names on the right hand side whereas in Figs. b and c marker names and genes are on the right hand side of the chromosome regions and the distances in centimorgans on the left. Gene names are in bold italics

chickpea can only be revealed after study of more defense-related genes.

Conclusions

Evaluation of chickpea lines for resistance to *Fusarium oxysporum* f. sp. *ciceris* using wilt sick plots can be erroneous as some of the plants might escape penetration by pathogen while in others the penetration is not simultaneous. The technique standardized to reproduce chickpea wilt under artificial conditions (Tullu 1996; Sharma et al. 2004b, 2005) can be useful for evaluation of resistance as well as for studies on inheritance of resistance as it ensures that all test plants are inoculated simultaneously at same vegetative stage with constant inoculum load and are grown under well defined environmental conditions. The injury to roots prior to inoculation ensures that all inoculated plants have a nearly equal chance of infection. The technique can also be useful to confirm results on late wilting and resolve the ambiguity in genetics of resistance to race 2 which is variable among different studies (Pathak et al. 1975; Gumber et al. 1995; Kumar 1998; Sharma et al. 2005). With the elucidation of slow wilting resistance in chickpea, there are now two types of host resistances available for wilt management, (i) the vertical resistance and (ii) the slow wilting resistance. Genetics of slow wilting resistance have not been explored. This resistance can be exploited more effectively in chickpea breeding, if its genetics are studied.

Screening of the progeny plants carrying wilt resistance gene(s) can be facilitated with marker assisted selection (MAS). Chickpea breeders are aiming to exploit MAS for resistance breeding. Efficacy of MAS, however, depends upon closeness of the marker to the gene. Marker density in the LG 2 and LG 5 is still low to facilitate MAS for wilt resistance genes and their positional cloning. There is a need to saturate the chromosomal region harboring wilt resistance genes with more markers to achieve these objectives. Since, sources of resistance to wilt are available within the cultigen, *C. arietinum*, MAS cannot be exploited effectively until polymorphic markers are available for *C. arietinum* populations rather than for interspecific ones. Such markers will also be useful for map based cloning as the differences in genetic and physical distances among markers would be minimum (Winter et al. 2000; Benko-Iseppon et al. 2003). With the advent of STMS markers and availability of sources of resistance to all races in *C. arietinum*, it is possible to map genes using intraspecific populations. STMSs, though highly polymorphic tend to cluster on chickpea linkage maps and their distribution is not uniform (Winter et al. 1999; Lichtenzveig et al. 2005). Amplified fragment length polymorphism (AFLP) and single nucleotide polymorphism (SNP) can be the other category of markers which can be exploited to map those genomic regions where microsatellite density is low. There is only one report on use of AFLP markers in chickpea genetic maps (Winter et al. 2000), whereas SNPs despite their unlimited potential in genomic studies have not yet been exploited. Lack of use of SNPs in chickpea can be attributed to the availability of limited sequence data of chickpea genomic regions. With the addition of more sequences of chickpea DNA segments/genes in the databases, it will be possible to develop SNPs in near future.

Markers linked to wilt resistance genes h_2 and h_3 that confer resistance to race 1A or those governing resistance to races 1B/C and 6 have not yet been mapped. Moreover, inheritance of resistance to races 1B/C and 6 has not yet been studied. Existence of resistance gene clusters as observed in chickpea is a phenomenon already known in crop sciences (review in Hulbert et al. 2001; Chin et al. 2001; Richly et al. 2002; Park et al. 2005). Gene clusters, however, have implications for MAS also. In a situation like chickpea where resistance genes lie a few hundred to a few thousand kb apart, markers flanking the individual genes are needed for effective MAS. An ideal situation can be where the marker is the part of the gene. Such closely linked markers are still not available for wilt resistance genes. On the other hand, it is feasible to ensure the transfer of whole of the resistance gene cluster/subclsuter by using the already available markers. The mechanisms underlying evolution of cluster of wilt resistance genes can be resolved after cloning of individual genes, however, existence of genes governing resistance to wilting pathotypes in a single cluster indicates the possibility of involvement of duplications and recombinations in its evolution. These mechanisms are the most evident ones for evolution of resistance genes clusters in other plant species (Richly et al. 2002; Meyers et al. 2005; Smith and Hulbert 2005). The majority of chickpea wilt resistance genes are recessive in nature. It would be interesting to determine if these have homology to the existing categories of plant resistance genes or these represent a new group of resistance genes. Cloning of chickpea wilt resistance genes, if achieved, is expected to lead to an understanding of their nature, evolution and mechanisms of wilt resistance.

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