Review Article Pandey & Dubey, 9(4): April, 2018:5772-5778]
CODEN (USA): IJPLCP **ISSN: 0976-7126**

INTERNATIONAL JOURNAL OF PHARMACY & LIFE SCIENCES
(Int. J. of Pharm. Life Sci.)

Inheritance and Molecular Mapping of Fusarium Wilt Resistance Gene in Brinjal*(Solanum melongena)*

Akanksha Pandey[1]* and Sanjeev Dubey[2]
1, Centre of Biotechnology studies School of Environmental Biology,
APS University, Rewa, (M.P.) - India
2, Department of Botany, Govt. Model Science College, Rewa, (M.P.) - India

Abstract

The inheritance of resistance to fusarium wilt derived from AB-2 XAB-52 conferring resistance to Fusariumoxysporum has been determined. The F2 individuals of the crossAB-2 (resistance) XAB-52 (susceptible) were screened against Fusarium wilt in combined tests which indicated dominant control by single locus of tightly linked gene(s). Bulked segregant analysis (BSA) was carried out on F2 individuals with a set of 250 DNA markers that resulted in the identification and mapped the *FM1* locus between the two DNA markers bNBS-LRR-45 and bRGA-25 located ~ 2.2 cM apart from each other. These closely linked markers to the wilt resistance gene(s) provide a valuable basis for marker-assisted selection in castor breeding programs.

Keywords: Brinjal, Fusarium Wilt resistance, F2 cross, locus, marker-assisted selection, Genric marker.

Introduction

Brinjal (*Solanum melongena*) which hasprobably originated in the Old World, mainly from India (Daunay 2008)is one of the few cultivated species in the family solanaceae. It has become an economically important vegetable crop in tropical and warm regions(Kashyap et al. 2003) with a global production of 49 million tons in 2013, increasing by 50 % from 2004 to 2013 (FAO 2004, 2013). Fusarium wilt, caused by the soil borne plant pathogen Fusarium oxysporum f. sp. melongenae, is one of the most serious diseases of brinjal. The fungus spreads widely and can persist for many years in the soil. This makes management practices like field idling and cultural control ineffective to eradicate this pathogen (Steekelenburg 1976; Altınok 2005; Daunay 2008; Safikhani et al. 2013). Under such conditions, an approach to develop wilt resistant brinjalcultivars offers a logical way to control this disease. The advancements in the recent past in the area of molecular marker analyses can aidefficient breeding.Susceptibilityof brinjal plants to variety of soil borne diseases is one of the major factors affecting eggplant production. Further, Fusarium wilt is one of the most devastating diseases in eggplant cultivation (Rotino et al. 2004).

*** Corresponding Author**
E.mail: akankshasanadhya88@gmail.com

Fusarium oxysporummelongenae induces vascular wilt disease in eggplant is responsible for heavy yield losses in this vegetable crop particularly in Asian countries (Altinok 2005).

Fusarium wilt is one of the most investigated subject in plant disease management with extensive reports available in several horticultural crops. For instance in important solanaceous vegetable crop tomato, four resistance genes, I, I1, I2, and I3, derived from wild species (Solanum pennellii and Solanum pimpinellifolium) have been identified through molecular mapping studies (Bournival et al. 1990; Hemming et al. 2004; Lim et al. 2008; Ori et al. 1997; Sarfatti et al. 1989; 1991; Segal et al. 1992; Simons et al. 1998). I2 gene from tomato and melon Fom-1 and Fom-2 were subjected to map-based cloning (Brotman et al. 2012; Joobeur et al. 2004; Simons et al. 1998). This revealed that all three genes contain nucleotide-binding site and a leucine-rich repeat (NBS-LRR) domain. Publication of first report on Fusarium wilt in eggplant by Matsuo and Ishigami (1958) was rapidly followed by research efforts to identify resistant eggplant allies. One such prominent study was undertaken by Monma et al. (1996) who identified large number of wilt resistant germplasm in Ghana. Following this, Rizza et al. (2002) developed interspecific hybrids between eggplant and its allies (Solanum aethiopicum) which were found to be completely resistant to Fusarium wilt. Previous studies have reported Rfo-sa1 (a dominant single

locus), to be responsible for Fusarium resistance in a somatic hybrid line mapped on chromosome 2 (Barchi et al. 2010, 2012; Portis et al. 2014) and SCAR markers linked to a Fusarium resistance locus in an eggplant line, LS2436, based on bulked segregant analysis (Mutlu et al. 2008). However, due to existence of low level of nucleotide polymorphism among eggplant lines,mapping studies on resistance loci in eggplant (S. melongena) have not been reported (Nunome et al. 2001). This creates difficulty in using intraspecific mapping populations for this purpose. Nunome et al. (2003a, b, 2009) and Fukuoka et al.(2012) have developed a large number of informative simple sequence repeat (SSR) and single nucleotide polymorphism (SNP) markers, which have allowed detection of polymorphisms among genetically close lines. These studies resolved to a great extent, the problem of genetic polymorphism and made markers available for genetic mapping for fruit shape and colour (Nunome et al. 2001) and parthenocarpy (Miyatake et al. 2012). In recent years, the eggplant draft genome sequence was published by Hirakawa et al. (2014).

In this study, we report the precise mapping position of a *Fusarium* wilt resistance locus with two alleles derived from cross **AB-2 (resistance) XAB-52 (susceptible)**, based on the mapping populations and linkage maps reported by Fukuoka et al. (2012) and Hirakawa et al. (2014); the positional relationships of resistance loci reported here and in previous studies (Barchi et al. 2010, 2012; Mutlu et al. 2008; Portis et al. 2014); and (3) the availability of resistance-linked markers that is suitable for marker-assisted selection (MAS).

Material and Methods

Plant material and mapping populations

The F2 mapping population used in the study was derived from AB-2 (resistance) XAB-52 (susceptible). The F1 plants were selfed to obtain segregating F2 population. The morphological, Biochemical and pathological characters recorded are as given in table1, 2&3.

Table 1: - Morphological Characterization most prominent resistant and susceptible lines of brinjal

Accession No.1	Plant height (cm)	No. primary branches /plant	Stem diameter (cm)	Days to 1st Flowering	Days to first fruiting	Harvesting period	No. of fruits in inflorescence/plant	Fruit stalk length	Fruit circumference	Fruits/plants	Yield per plant
AB-2	159	16.5	4.8	105	112.5	70	13.91	6.28	23.85	18.92	4588
AB-52	176	15.5	5.87	131.5	144	89.5	3.67	7.6	9.48	6.17	2523

Table 2: - Pathological Characterization most prominent resistant and susceptible lines of brinjal

Accession No.	**Total plant population**	**% Infection after 50 Days**	**% Infection after 65 Days**	**% Infection after 80 Days**	**% Infection after 95 Days**	**% Infection after 50 Days**	**% Infection after 65 Days**
AB-2	**20**	0	0	0	0	0	0
AB-52	25	22.22	33.33	55.56	77.78	22.22	33.33

Table 3: - Biochemical Characterization most prominent resistant and susceptible lines of brijal

Accession No.	**Peroxidase assay (**units/g)	**Phenol content (**µg catechol/g tissue**)**
AB-2	2.01	1520.23
AB-52	0.241	1050.86

Morphological and Pathological characterization of mapping population

The morphological observations of the parents of mapping population were observed in the field. These observations include Plant height (cm), No. Primary branches /plant,Stem diameter (cm), Days to 1st Flowering, Days to first fruiting, Harvesting period, No. of fruits in inflorescence/plant, Fruit stalk length, Fruit circumference, No. of Fruits/plants, and fruit Yield per plant Morphological observations were recorded as per the DUS procedure and other trait specific standard procedures reported. The pathological observations were taken after a fixed interval of days *viz*50 days, 65 days, 80 days and 95 days) for parents and mapping population under natural conditions of field. The observation of each line recorded in two replications for quantitative and qualitative traits along with the contributing traits were used for calculating the mean performance. Data of three randomly selected plants from each replication were averaged. The mean performance, range (minimum and maximum), standard deviation (SD), coefficient of variance (CV %) and number of transgressive segregants for all the traits studied in F2 mapping population were worked out along with mean of parents. The results revealed that the mapping population has segregation between the lines for specific parameters. The mean sum of squares for population was highly significant for the traits related to Fusarium wilt resistance and reported to be highly contributing in Fusarium wilt resistance.

DNA isolation and quantification

The genomic DNA was extracted from the parents **AB-2 (resistance), AB-52 (susceptible)**, and F2 plants derived from the cross according to the protocol Dellaporta et al. (1983). Leaf samples obtained from 30-35days old plants were freeze dried (-80°C) and lyo-philizedwere ground to a fine powder and used for DNA extraction. The isolated DNA was quantified using NanoDrop and diluted to a final concentration of 50ng/µl for PCR analysis. Phenotypic data recorded for each trait was used to score individual F2 plants. 10 most resistant and 10 most susceptible plants were chosen for bulked segregant analysis *ie*BSA (Michelmore et al. 1991). After quantification,resistant and susceptible pools were prepared withequimolar amounts of DNA from the 10 F2 individuals. Bulked samples were then screened for polymorphism with over 250Oligo-nucleotide primers.

PCR analysis

A set of 250 Oligo-nucleotide primers synthesized from Operon Technologies Inc. (Ala-meda, CA, USA) were used for genotyping study. These markers were initially tested on the parents AB-2 (resistant) and AB-52 (susceptible) for the detection of polymorphism between them. Out of these 250 markers, 68 markers were found to be polymorphic between the contrasting parents. The primers generating reproducible polymorphism among individual plants were selected for genotyping the F2 individuals.

PCR reactions were performed in the Perkin-Elmer 9600 thermal cycler (Barnstead Thermolyne Corp., Iowa, USA). The reactions were performed in a 20 µl reaction mix, containing 25 ng/ul of genomic DNA as template, 10 mMTris-HCl (pH 8.3), 50 mMKCl, 2 mM MgCl2, 2 mM of each dNTP, 5 moles/µl of primer, and 0.3 U of Taq DNA polymerase (BangloreGenei Pvt. Ltd.). The PCR program was set as Initial denaturation at 94°C for 3 min followed by denaturation at 94°C for 1 min, annealing at 60°C for 2 min and extension at 72°C for 2 min and these steps were repeated for 35 cycles with a final extension at 72°C for 5 min and hold at 4°C. The PCR products were resolved on 1.8% agarose gels. Ethidium bromide stained gels were visualized and documented by gel documentation system (BioRad).

Statistical analysis

Segregation ratios of F2 individuals were checked using Chi-square tests. Linkage analysis was performed using a tool, MAP-MAKER (Lander et al. 1987). This tool applies the maximum likeli-hood method for determination of recombination values and the Kosambi's mapping function (Kosambi 1944) for correcting the recombination values to map distances in cM. The logarithm of odds (LOD) score of 3.0 was used as a linkage threshold in this study. MAPMAKER is capable of performing both two-point and multipoint linkage analysis. Two-point linkage analysis is used to work outpair-wise linkage distances between any two segregating loci taken while the multipoint linkage analysis derives the best possible locus order along with a map based on the data on all segregating loci at one time.

Results and Discussion

In field screening, 70 % of the accessions analyzed were observed as resistant while 30 % of them were found to be susceptible to *Fusarium* wilt (Table5.8). As per the disease rating scale, the experimental material was categorized into resistant (R), with wilt incidence (%) less than 30%, resistant (R), with wilt incidence (%) 21.4, tolerant (T) with 30 to 58% wilt incidence (%) and susceptible (SS) group for which wilt incidence (%) was observed to be more than 60 % under field condition after 95 days of

transplanting. The mapping population categorized on the basis of disease response at seedling and reproductive stage for comparison provided a valid basis for an important observation that increase in susceptibility to wilt disease might be contributed by slow wilting resistance of some lines. On the basis of morphological, biochemical and pathological observations mapping population was categorized into three categories *viz* resistant, tolerant and susceptible. Out of a total of 73 lines, 57 lines revealed highly resistant trait and 16 lines were observed to fall in the susceptible category.

For mapping and linkage analysis, a set of 250 DNA markers was used. These markers were initially tested on the parents AB-2 (resistant) and AB-52 (susceptible) for the detection of polymorphism between them. Out of these 250 markers, 68 markers (27.20%) appeared to be polymorphic between the contrasting parents (Fig.1).

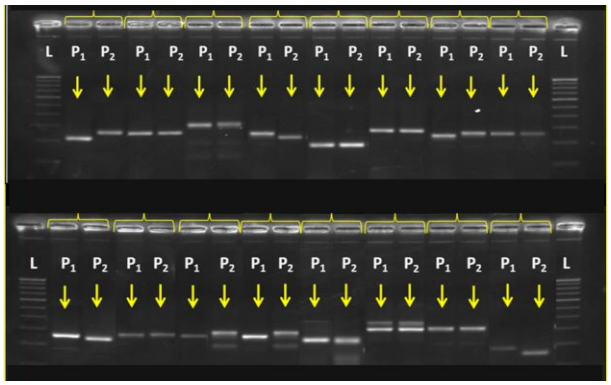

Fig. 1 Polymorphism of markers between the two parents AB-2 (P1) and AB-52 (P2). M: Marker.

For inheritance study, The F_2 (73 in total) were also subjected to testing for fusarium resistance. Out of 73 plants, 57 plants exhibited resistance while 16 were susceptible to the virus. The ratio of resistant and susceptible plants well fitted to the expected Mendelian ratio of 3:1.

The DNA from the resistant AB-2 and susceptible analogue AB-52 cultivars and their F_1 was subjected to generic markers screening. A total of 260 oligo-nucleotide primers were tested, of which 68 primers generated polymorphism between the parents in the cross respectively. The identified polymorphic primers were used to screen the F_2 mapping populations which resulted in the identification of two markers associated with the *Fusarium* wilt resistant phenotype of these, bNBS-LRR45 and bRGA-25The segregation ratio of the two linked markers was in agreement with Mendelian segregation ratios of 3: 1 and 1: for dominant markers (**Table 4 and Fig.2**). Numerous Fusarium resistance resources have previously been reported in wild relatives of brinjal (Monma et al. 1996; Toppino et al. 2008). However, there are challenges in breeding programs using wild species as they require recurrent and long-term backcrossing of cultivated species to overcome deleterious traits contributed by the wild species genomes (Brown 2002; Chittaranjan 2011; Haggard et al. 2013; Legg et al. 1981; Lewis et al. 2007; Zeven et al. 1983). Additionally, interspecific F1 hybrids often turn out to be sterile (Calvo-Asensio et al. 2014; Daunay et al. 1993; Khan et al. 2011; Schaff et al. 1982), hindering further genetic improvement of the lineage. Use of Fusarium wilt-resistant germplasms of cultivated eggplant thus appears to be more useful for breeding. In this study, we genetically characterized two Fusarium wilt-resistant cultivated eggplant material and developed DNA markers closely linked to the resistant locus FM1.The findings of the research efforts made in this study will help in facilitating breeding programs to develop Fusarium wilt resistant eggplant.

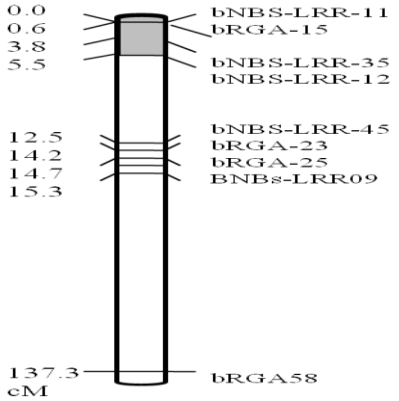

Fig. 2 Fusariumresistance loci on chromosome 2.

Table 4: Segregation for resistance to*Fusarium*wilt in the F_2generations of the cross 'AB-2 and AB-52.

Generation	**Observed frequency**		**Ratio tested**	**X^2**	**P**
	Resistance (RR)	Susceptible(rr)			
AB-2 and AB-52 (field screening)					
F_2	57	16	3:1	0.52	0.4795

The DNA from the resistant AB-2 and susceptible analogue AB-52 cultivars and their F_1 was subjected to generic markers screening. A total of 260 oligo-nucleotide primers were tested, of which 68 primers generated polymorphism between the parents in the cross respectively. The identified polymorphic primers were used to screen the F_2 mapping populations which resulted in the identification of two markers associated with the *Fusarium* wilt resistant phenotype of these, bNBS-LRR45 and bRGA-25The segregation ratio of the two linked markers was in agreement with Mendelian segregation ratios of 3: 1 and 1: for dominant markers.

Efficacy of Markers Linked to Fusarium Wilt Resistance and Their Validation

The first markers of the two markers identified for fusarium wilt resistance in this study were selected for the validation of linked markers. These markers included bNBS-LRR-45and bRGA-25 and were validated in a set of 8 germplasm lines consisting of 4resistant and 4 susceptible genotypes. The markers could effectively distinguish the resistant and susceptible genotypes. Marker bNBS-LRR-45and bRGA-25 produced resistant type allele in the all resistant genotypes while no alleles were amplified in susceptible genotypes as shown in the Figure 3.

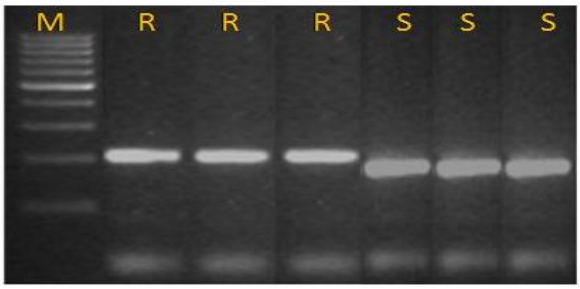

Fig. 3: Validation of the linked markers bNBS-LRR-45 **in resistant genotypes.**

References

1. Altinok H H. 2005. First report of Fusarium wilts of eggplant caused by Fusarium oxysporum f. spmelongenae in Turkey. Plant Pathology 54: 577–9.
2. Barchi L, Lanteri S, Portis E, Stagel A, Vale G, Toppino L, Rotino GL (2010) Segregation distortion and linkage analysis in eggplant (*Solanum melongena* L.). Genome 53:805–815
3. Barchi L, Lanteri S, Portis E, Vale G, Volante A, Pulcini L, Ciriaci T, Acciarri N, Barbierato V, Toppino L, Rotino GL (2012) A RAD tag derived marker based eggplant linkage map and the location of QTLs determining anthocyanin pigmentation. PLoS One 7:e43740
4. Bournival BL, Vallejos CE, Scott JW (1990) Genetic analysis of resistances to races 1 and 2 of *Fusarium oxysporum* f. sp. *lycopersici* from the wild tomato *Lycopersiconpennellii*. TheorAppl Genet 79:641–645
5. Brown JKM (2002) Yield penalties of disease resistance in crops. CurrOpin Plant Biol 5:339–344
6. Calvo-Asensio I, Prohens J, Gisbert C (2014) Vigor for in vitro culture traits in *S. melongena* × *S. aethiopicum* hybrids with potential as rootstocks for eggplant. Sci World. doi: 10.1155/2014/702071
7. Chittaranjan K (2011) Wild crop relatives: genomic and breeding resources - vegetables. Springer, Berlin, pp 1–282
8. D DKosambi, The estimation of map distance from recombination values, Annals of Eugenics, Vol.12, pp.172–175, 1944
9. Daunay MC, Chaput MH, Sihachakr D, Allot M, Vedel F, Ducreux G (1993) Production and characterization of fertile somatic hybrids of eggplant (*Solanum melongena* L) with *Solanum aethiopicum* L. TheorAppl Genet 85:841–850
10. Dellaporta, S.L. Wood, J. and Hicks, J.B. 1983. A plant DNA mini preparation: Version II. Plant Molecular Biology Reporter 1: 19-21
11. FAO (2004) FAOSTAT. Food and Agriculture Organization of the United Nations. http://faostat.fao.org
12. FAO (2013) FAOSTAT. Food and Agriculture Organization of the United Nations. http://faostat.fao.org
13. Fukuoka H, Miyatake K, Nunome T, Negoro S, Shirasawa K, Isobe S, Asamizu E, Yamaguchi H, Ohyama A (2012) Development of gene-based markers and construction of an integrated linkage map in eggplant by using *Solanum orthologous* (SOL) gene sets. TheorAppl Genet 125:47–56
14. Fukuoka S, Saka N, Koga H, Ono K, Shimizu T, Ebana K, Hayashi N, Takahashi A, Hirochika H, Okuno K, Yano M (2009) Loss of function of a proline-containing

protein confers durable disease resistance in rice. Science 325(5943):998–1001
15. Haggard JE, Johnson EB, Clair DA (2013) Linkage relationships among multiple QTL for horticultural traits and late blight (*P. infestans*) resistance on chromosome 5 introgressed from wild tomato *Solanum habrochaites*. G3 3:2131–2146
16. Hemming MN, Basuki S, McGrath DJ, Carroll BJ, Jones DA (2004) Fine mapping of the tomato *I-3* gene for *fusarium* wilt resistance and elimination of a co-segregating resistance gene analogue as a candidate for *I-3*. TheorAppl Genet 109:409–418
17. Hirakawa H, Shirasawa K, Miyatake K, Nunome T, Negoro S, Ohyama A, Tamaguchi H, Sato S, Isobe S, Tabata S, Fukuoka H (2014) Draft genome sequence of eggplant (*Solanum melongena* L.): the representative Solanum species indigenous to the Old World. DNA Res .doi: 10.1093/dnares/dsu027
18. Kashyap V, Kumar SV, Collonnier C, Fusari F, Haicour R, Rotino GL, Sihachakr D, Rajam RM (2003). Biotechnology of eggplant. ScientiaHorticulturae 97:1-25.
19. Khan RS, Ntui VO, Chin DP, Nakamura I, Mii M (2011) Production of marker-free disease-resistant potato using isopentenyltransferase gene as a positive selection marker. Plant Cell Rep 30:587–597
20. Lander ES, Green P, Abrahamson J, Barlow A, Daly MJ, Lincoln SE, and Newberg L, 1987. MAPMAKER: an interactive computer package for constructing primary genetic linkage maps of experimental and natural populations. Genomics 1:174–181.
21. Legg PD, Litton CC, Collins GB (1981) Effects of the Nicotianadebneyi black root rot resistance factor on agronomic and chemical traits in burley tobacco. TheorAppl Genet 60:365–368
22. Lewis RS, Linger LR, Wolff MF, Wernsman EA (2007) The negative influence of N-mediated TMV resistance on yield in tobacco: linkage drag versus pleiotropy. TheorAppl Genet 115:169–178
23. Lim GT, Wang GP, Hemming MN, McGrath DJ, Jones DA (2008) High resolution genetic and physical mapping of the *I-3* region of tomato chromosome 7 reveals almost continuous microsynteny with grape chromosome *12* but interspersed microsynteny with duplications on Arabidopsis chromosomes 1, 2 and 3. TheorAppl Genet 118:57–75
24. Michelmore RW, Paran I and Kesseli RV: Identification of markers linked to diseaseresistance genes by bulked segregant analysis: A rapid method to detect markers in specific genomic regions by using segregating populations. Proceedings of National Academic of Sciences, USA 1991, 88:9828-9832.
25. Miyatake K, Saito T, Negoro S, Yamaguchi H, Nunome T, Ohyama A, Fukuoka H (2012) Development of selective markers linked to a major QTL for parthenocarpy in eggplant (*Solanum melongena* L.). TheorAppl Genet 124:1403–1413
26. Mochizuki H, Sakata Y, Yamakawa K, Nishio T, Komochi S, Narikawa T, Monma S (1997) 'Eggplant parental line 1', an eggplant breeding line resistant to *Fusarium* wilt. Bulletin of the National Institute of Vegetable and Tea Science 12:85–90
27. Monma S, Akazawa S, Simosaka K, Sakata Y, Matsunaga H (1997) 'Daitaro', a bacterial wilt- and *Fusarium* wilt-resistant hybrid eggplant for rootstock. Bull NatlInst Veg Ornam Plants Tea 12:73–83 **(in Japanese with English summary)**Google Scholar
28. Monma S, Sato T, Matsunaga H (1996) Evaluation of resistance to bacterial, *fusarium* and *verticillium* wilt in eggplant and eggplant related species collected in Ghana. J JpnSocHorticSci 65:278–279
29. Nunome T, Ishiguro K, Yoshida T, Hirai M (2001) Mapping of fruit shape and color development traits in eggplant (*Solanum melongena* L.) based on RAPD and AFLP markers. Breed Sci 51:19–26
30. Nunome T, Negoro S, Kono I, Kanamori H, Miyatake K, Yamaguchi H, Ohyama A, Fukuoka H (2009) Development of SSR markers derived from SSR-enriched genomic library of eggplant (*Solanum melongena* L.). TheorAppl Genet 119:1143–1153
31. Nunome T, Suwabe K, Iketani H, Hirai M (2003a) Identification and characterization

of microsatellites in eggplant. Plant Breed 122:256–262
32. Nunome T, Suwabe K, Ohyama A, Fukuoka H (2003b) Characterization of trinucleotide microsatellites in eggplant. Breed Sci 53:77–83
33. Ori N, Eshed Y, Paran I, Presting G, Aviv D, Tanksley S, Zamir D, Fluhr R (1997) The *I2C*family from the wilt disease resistance locus *I2* belongs to the nucleotide binding, leucine-rich repeat superfamily of plant resistance genes. Plant Cell 9:521–532
34. Rizza F, Mennella G, Collonnier C, Shiachakr D, Kashyap V, Rajam MV, Prestera M, Rotino GL (2002) Androgenic dihaploids from somatic hybrids between *Solanum melongena* and S-aethiopicum group gilo as a source of resistance to *Fusarium*oxysporum f. spmelongenae. Plant Cell Rep 20:1022–1032
35. Safikhani N, Morid B, Zamanizadeh HR (2013) First report of *Fusarium* wilt of eggplant caused by *Fusarium oxysporum* f. sp. *melongenae* in Iran. New Disease Reports 28:16
36. Sarfatti M, Abu-Abied M, Katan J, Zamir D (1991) RFLP mapping of I1, a new locus in tomato conferring resistance against *Fusarium oxysporum* f. sp. *lycopersici* race 1. TheorAppl Genet 82:22–26
37. Sarfatti M, Katan J, Fluhr R, Zamir D (1989) An RFLP marker in tomato linked to the *Fusarium oxysporum* resistance gene I2. TheorAppl Genet 78:755–759
38. Schaff DA, Jelenkovic G, Boyer CD, Pollack BL (1982) Hybridization and fertility of hybrid derivatives of *Solanum melongena* L and *Solanum-macrocarpon* L. TheorAppl Genet 62:149–153
39. Segal G, Sarfatti M, Schaffer MA, Ori N, Zamir D, Fluhr R (1992) Correlation of genetic and physical structure in the region surrounding the *I2 Fusarium oxysporum* resistance locus in tomato. Mol Gen Genet 231:179–185
40. Simons G, Groenendijk J, Wijbrandi J, Reijans M, Groenen J, Diergaarde P, Van der Lee T, Bleeker M, Onstenk J, de Both M, Haring M, Mes J, Cornelissen B, Zabeau M, Vos P (1998) Dissection of the fusarium *I2* gene cluster in tomato reveals six homologs and one active gene copy. Plant Cell 10:1055–1068
41. Steekelenburg N (1976) *Fusarium* wilt of eggplant in the Netherlands. Neth J Plant Pathol 82:191–192
42. Toppino L, Vale G, Rotino GL (2008) Inheritance of *Fusarium* wilt resistance introgressed from *Solanum aethiopicum* Gilo and Aculeatum groups into cultivated eggplant (*S. melongena*) and development of associated PCR-based markers. Mol Breed 22:237–250
43. Yoshida T, Monma S, Matsunaga H, Sakata Y, Sato T, Saito T (2004) Development of a new rootstock eggplant cultivar 'Daizaburou' with high resistance to bacterial wilt and *Fusarium* wilt. Bull NatlInst Veg Tea Sci 3:199–211
44. Zeven AC, Knott DR, Johnson R (1983) Investigation of linkage drag in near isogenic lines of wheat by testing for seedling reaction to races of stem rust, leaf rust and yellow rust. Euphytica 32:319–327

How to cite this article
Pandey A. and Dubey S. (2018). Inheritance and Molecular Mapping of Fusarium Wilt Resistance Gene in Brinjal*(Solanum melongena)*. *Int. J. Pharm. Life Sci.*, 9(3):5772-5778.
Source of Support: Nil; Conflict of Interest: None declared

Received: 01.03.18; Revised: 28.03.18; Accepted: 10.04.18