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Tomato R-Genes against *Fusarium* wilt : Present status and future prospect

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This review deals with an over view of tomato resistance- genes (R-genes) against *Fusarium oxysporum* that cause wilt disease. Polymorphic resistance genes designated as I (Immunity) namely I/I-1, I-2 and I-3 have been described. The I/I-1, I-2 and I-3 confer resistance against *F. oxysporum* race 1, race 2 and race 3 respectively. The identification, location and functional correlation between the I-genes have been described. The evolutionary relationship of the tomato I genes and the *Fusarium* avirulence (avr) genes show adaptive competition between the host and the pathogen. The uses of these R-genes in developing resistant tomato lines and the future scope of achieving durable resistance have also been discussed.

Key words: R-genes, Tomato, *Fusarium oxysporum*

INTRODUCTION

Strains of *F. oxysporum* that cause wilt of tomato plants are grouped in *forma specialis* (f.sp.) *lycopersici.* Several polymorphic resistance (*R*) genes have been identified in the tomato gene pool each of which confer resistance against a subset of *F. oxysporum* f.sp. *lycopersici* (Fol) strains. These are *I* (for *Immunity*), *I-1, I-2* and *I-3*. Races of Fol are named historically according to the *R* gene that is effective against them: the *I* gene and the (unlinked) *I-1* gene are effective against race 1, race 2 overcomes *I* and *I-1,* but is stopped by *I-2,* while race 3 overcomes *I, I-1* and *I-2* but is blocked by *I-3.*

Solanaceous crop plants in general, are prone to fungal wilt diseases. Of the different plants, tomato (*Lycopersicon esculentum*) is severely affected by wilt diseases caused by pathogen *Fusarium oxysporum* f.sp. lycopersici. The wilt organism usually enter the host plant body through young roots and then grow into and up the water conducting vessels of root and stem. They cause plugging and collapsing of the xylem vessel, thereby water supply to the leaves is reduced. This blockage of water supply leads to wilting of leaf petiole and ultimately drooping of the entire aerial parts of the plant. The symptom first appears on outer leaflets. Later the lower leaflets turn yellow and die. It proper measures are not taken, the entire plant will die often before it reaches maturity. Most soil-borne *F. oxysporum* is innocuous and is frequently isolated as an endophyte within the cortex of asymptomatic roots (Gordon and Martyn, 1997). Some pathogenic strains have a narrow host range but can be destructive for a monoculture crop. The host specificity of a particular *F. oxysporum* isolate is described by its forma specialis (f.) (Armstrong and Armstrong. 1975). The pathogenic strain can be designated by its race, which exhibits differential specificity for varieties of the host species. Three host specific races of this pathogen (races-*I, II, III*) have been identified. (Stevens and Rick, 1986).

A dynamic equilibrium between plant resistance and pathogen virulence is maintained by R-gene encoded receptors and avirulence (*avr*) gene product interaction within the plant cell. In most cases that have been studied, plant genes that confer dominant resistance against pathogens encode proteins involved in race-specific pathogen recognition. The best studied of these R-genes are

those that confer strong resistance in a gene-for-gene manner to pathogens that express a corresponding *avr* gene (Nimchuk *et al.* 2003).

Classification of R-genes

R-genes can be broadly classified into four structural classes; **a)** the first family encodes the serine-threonine protein kinase. *Pto* is the only known member that confers resistance to bacterial speck disease in tomato (Martin *et al.*1993). **b)** The second class of R-genes represents the transmembrane receptors with extracellular leucine rich repeats (LRR) domains, which is represented by the *Cf* family of tomato resistance genes (for leaf mold resistance) and *Hs 1* (for nematode resistance) (Jonses *et al.* 1997; Dixon *et al.* 1996, Dixon *et al.* 1998). **c)** The third class of R-gene encodes receptor-like kinase, combining qualities of both classes above e.g. Xa21 imparting resistance to rice bacterial blight (Song *et al.* 1995). **d)** the fourth class includes the majority of functionally described R-genes, the nucleotide binding site leucine rich repeat (NBS-LRR) resistance genes. The proteins encoded by these genes contain three specific domain: i) a variable N terminus, ii) nucleotide-binding site, and iii) leucine rich repeats. Two kinds of N termini are present in NBS-LRR. One type of NBS-LRR contains coiled coils (CC) motif that are supposed to take part in protein-protein interactions. CC motifs appear in the N terminus of both dicotyledons and monocots (Pan *et al.* 2000). The second type of N terminus shows homology to the *Drosophila* Toll or human interleukin receptor-like (TIR) regions that also contain LRR domains (Whithan *et al.* 1994; Hammond-Kosack and Jones 1997). The C-terminal LRR region can participate in protein-protein interaction that is essential for pathogen-speficic gene-for-gene recognition (De Wit *et al.* 1997; Warran *et al.* 1998; Ellis *et al.* 2000).

R genes code for two broad groups of leucine-rich-repeat (LRR) proteins that differ by their protein domain structure and their sites of pathogen specifity (Jone and Tokemoto, 2004). **(a)** The NBS-LRR-containing R proteins mediate recognition of an intracellular pathogen-derived signal. In other words NBS-LRR proteins take part in the resistance signaling in response to penetration of pathogen into a cell. **(b)** The second group of R proteins is found in the cell membrane and consists of an extra cellular LRR domain and a transmembrane (TM) domain (Jones and Tokemoto, 2004). Some of these transmembrane LRR proteins also have an intracellular protein kinases (PK) domain and belong to the larger class of receptor-like protein kinases (RLKs). The extra cellular LRR domain of LRR-TM and LRR-TM-TM-PK proteins acts as the receptor for an extra cellular pathogen-derived signal. The signal can be a viral coat protein, a secreted effector protein, the enzymatic activity of an effector protein, or a fungal polyketide metabolite (Nimchuk *et al.* 2003; Bohnert *et al.* 2004).

The I-genes and resistance :

Both dominant monogenic and oligonenic resistance to *F. oxysporum* are observed in various crop species (Sherbakoff 1949; Beckmann and Roberts 1995). Symptoms have been found to correlate with degree of vascular colonization, and resistance level is quantitatively related to the success in restricting vascular colonization (Gao *et al,* 1995 a, b).

Six *Immunity (I)* loci providing resistance of *F. oxysporum* f. *lycopersici* have been idenfified in *Lycopersicon* genomes (Sela-Buurlage *et al.* 2001). *I, I-2,* and *I-3* genes have been used for developing disease resistant tomatoes (Beckmann and Roberts, 1995).

I-1 gene

Bohn and Tucker (1939) first identified the dominant *I* gene in *L. pimpinellifolium* that confers resistance against *Fusarium oxysporum* f.sp. *lycopersici* race-1. This gene was mapped to chromosome 11 (Paddock, 1950). Later by breeding experiments and RFLP marker mapping it was found that resistance was controlled by single dominant gene I-1, which was not allelic to I, the traditional gene for resistance against *Fusarium,* that was derived from *L. pimpinellifolium.* The immunogenic locus I-1 was found to be positioned on chromosome–7 (Sarfatti *et al.* 1991).

High resolution RFLP mapping for locating *I-1:* utilizing "saturated" RFLP marker mapping, I-1 was located between the markers *TG-20* and *TG-128.* The availability of RFLP markers on both sides of resistance locus helps in *I-1* genotype determination on the basis of disease resistance for backcross progeny and for segregating population in a breeding experiment (Sarfatti *et al.* 1991)

In tomato, thus *I-1* locus is associated with the RFLP markers, which allow cloning of resistance genes. In future, there is high prospect in developing a disease resistant tomato line against *Fusarium* race-1 by incorporating the resistant *I-1* locus to a susceptible variety.

I-2 gene

The dominant *I-2* gene, isolated from wild tomato species (*L. pimpinnelifolium*) (Stall and Walter, 1965), is located on chromosome 11 (Laterrot, 1976). The relationship between f. *lycopersici* races and the resistance loci *I-2* and I are complicated. The *I* locus of *L. pimpinnelifolium* confers strong resistance to race I and no resistance to race 2, but the syntenic *I* locus from *L. pennellii* confers partial resistance to race I while imparting strong resistance to race 2. On the same note the *I-2* locus of *L. pimpinnelifolium* imparts strong resistance to race 2, while the syntenic *I-2* locus of *L. pennellii* confers partial resistance to race 2 (Sarfatti *et al.* 1991, Ori *et al.* 1997).

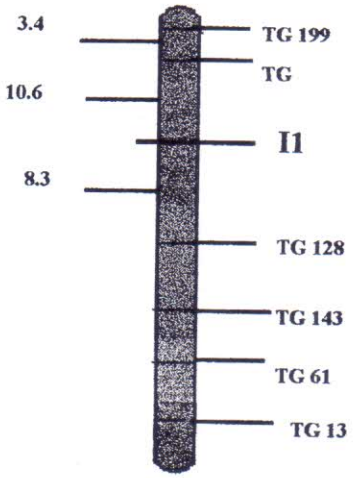

Fig. 1 : Molecular map of tomato chromosome-7 containing I-1 gene : I-1 is found to be positioned between the markers TG-20 and TG-128

Two members of multigene family, designated as *I-2*-C, were isolated from 12 *F.o.lycopersici* race2 resistance loci in tomato (Ori *et al.* 1997). The *I-2* locus is characterized as a cluster of at least seven related NBS-LRR gene sequences (Simons *et al.* 1998). The members of this *I-2-C* family were mapped to five different genomic locations. Two of these are gene clusters located on chromosome 11. Three of these *I-2* homologous-coding sequences confer resistance to race 2. Development of a transgenic plant utilizing *I-2* gene gives complete resistance, while transgenic plants expressing *I-2C-1* and *I-2C-5* exhibit only partial resistance to race 2 (Ori *et al.* 1997; Sela-Buurlage *et al.* 2001). The *I-2* encodes a polypeptide of 1266 amino acids belonging to coiled-coil (CC) NBS-LRR-class of R gene (Simons *et al.* 1998). The *I-2* gene contains a LZ motif lying between the NBS and LRR domain.

The *I-2* gene product is highly homologous to the *I-2-C* gene family characterized by Ori *et al.* (1997). Two members of this gene family *I-2-C-1* and *I-2-C-2,* map at the same locus and are found to be 82% and 88% identical to *I-2*. Expression of partial gene fragments of *I-2-C-1* and *I-2-C-3* in the sense or anti sense orientation imparts immunity to *F.o.lycopersici* race2. Transgenic plants containing *I-2-C-1* did not confer complete resistance. Transgenic veriety developed using cosmids containing *I-2-C-1/I-2-C-2* genes were found to be susceptible. This suggests that *I-2-C/I-2-C-2* is not essential for resistance to *F.o.lycopersici* race-2 and it is the *I-2* that confers full resistance (Mes *et al.,* 1999).

The *I-2* locus on chromosome 11 is composed of seven homologs spanning a region of about 90 kb. The intergenic regions between the homologs are calculated to be 8 to 10 kb. the seven homologs are designated as A to G. The functional *I-2* gene (homolog E), is flanked on one side by *I-2-C-2* (homolog D) and *I-2-C-1* (homolog G) separated by an additional gene coy (homolog F). The homolog A ORF has an oppoite direction to that of D, E, G.

It is widely believed that in the case of the I-2 gene, the number of the 23 amino acid repeats governs the evolution of specificities. The 28 amino acid repeat at the N-terminus of LRR may also influence specificity. Detailed knowledge of this locus will provide us with tool to generate new specificities to keep up with the change in pathogen virulence.

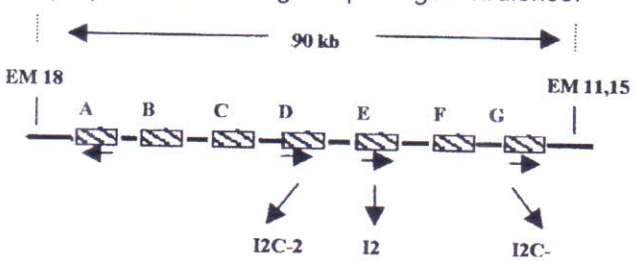

Fig. 2 : I-2 locus on Tomato Chromosome 11. Hatched squares (A-G) are I-2 homologs separated by non-coding regions. The EM 18 and EM 11, 15 are AFLP markers. Arrows indicate direction of ORFs.

I-3 gene

I-3 resistance is confined to a 0.3 cM interval region in the tomato genome which co segregates with a cluster of genes from the S-receptor gene family (Hemming *et al.* 2004). The third race of *F.o.lycoperisici* was first reported in Australia (1978), followed by reports from California and Florida. Resistance to race-3 is controlled by a single dominant gene, described in *L. pennellii* accession PI414773 (McGrath *et al* 1987) and LA716 (Scot and Jones 1989) and the genes responsible in both accessions were named *I-3.*

PCR-based sequence characterized amplified region SCAR, cleaved amplified polymorphic sequence markers (CAPS) and randomly amplified DNA fingerprinting (RAF) markers were used for high resolution mapping around *I-3* locus.

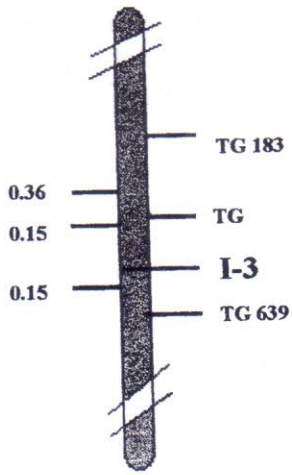

Fig. 3 : The *I-3* gene is localized to a region on tomato chromosome 7 between markers TG 128 and TG 639

The *I-3* gene was placed between the molecular markers CT226 and TG572 (Hemming *et al.* 2004) and is in agreement with other reports (Sela-Buurlage *et al.* 2001). *I-3* is contained within a 0.3 cM interval btween two markers, although greater distances have been reported from other mapping populations (Burbridge *et al.,* 2001).

The cysteine-rich protein, Secreted in Xylem 1 (SIX-1) is required for avirulence of *F.oxysporum* f.sp. *lycopersici* on tomato line, which harbors *I-3* resistance gene specifically (Rep *et al.* 2004, 2005). The protein is secreted by the pathogen during the colonization of xylem. However there are some isolates of *F. oxysporum* f.sp. *lycopersici* that are virulent against tomato line containing I-3 gene, but that does not contain mutation of SIX-I gene (Rep *et al* 2005) Moreover the SIX-1 gene of an I-3–virulent isolate is shown to be fully functional studies show that there is a link between presence of AVR1 and evasion of *I-3* mediated resistance in plants. Perhaps the AVR1 or a gene linked to it encodes a secreted protein or produces a compound that suppresses *I-3* through interfering either directly with *I-3* gene product or disrupts the downstream signal transduction process (Houterman *et al.* 2008).

Application of R-Genes in crop improvement and future prospect

During evolution of the tomato-Fol pathosystem, *I-2* and *I-3* have evolved to recognize, respectively, Avr2 and Avr3 since Avr3 is required for full virulence of Fol, evasion of *I-3* recognition through loss of the *AVR3* gene would be disadvantageous for the survival fitness of the plant. Since 1980's intensive research is being carried on in developing disease resistant crop plants with the help of R-genes. More than 40 genes including single genes and Quantitative Trait Loci, (QTLs) have been identified in tomato that impart resistance against major classes of pathogens (Tanksley *et al,* 1992). Markers tightly linked to these resistant genes are being used to rapidly select resistant crop species in marker assisted selection (MAS) programs. DNA marker technology coupled with commercial plant breeding programs is being used for transferring or

Table 1. : *F.oxysporum.* f.sp. *lycopersici*-tomato pathosystem

Tomato R genes	*F.oxysporum race*		
	Race 1	Race 2	Race 3
I or I-1	Avirulent	Virulent	Virulent
I-2	Virulent of avirulent	Avirulet	Virulent
I-3	Virulent or avirulent	Virulent or avirulent	Avirulent

accumulating ("pyramiding") desirable resistance traits into high yielding crop varieties.

Several of the R-genes have been cloned. Based upon protein domains shared among the R-genes, resistant gene analogs (RGAs) have been traced to nine of the twelve chromosomes. In the 1940s, *I* gene from the wild tomato relative *Solanum (Lycopersicon) pimpinellifolium* was the first *R* gene to be introgressed into tomato cultivars to impart resistance against *Fusarium* wilt. The *I-2* gene, also from *S. pimpinellifolium* against Avr 2, was introduced in commercial cultivars in the 1960s to protect tomato against Fol race 2. The active copy of I-2 gene along with the race 1 resistant gene (I-1 gene) was introgressed from *S. pimpinellifolium* (Sela-Buurlage *et al.,* 2001). The combined effect of *I* and *I-2* successfully provided resistance for about twenty years until race 3 emerged in Australia and N. America. To combat race 3, the *I-3* gene was introgressed from *S. pennellii.* The combination of *I* (or *I-1)* and *I-3* may yield durable resistance of tomato to *Fusarium* wilt disease of tomato, since *I-3* is directed against a virulence factor (Avr3) and *I* (and *I-1*) against the suppressor of *I-3* (Avr1) (Houterman *et al.* 2008).

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